Chloride

The chloride ion is the anion (negatively charged ion) Cl^-. It is formed when the element chlorine (a halogen) gains an electron or when a compound such as a hydrogen chloride is dissolved in water or other polar solvents. Chloride salts such as sodium chloride are often very soluble in water.

It is an essential electrolyte located in all body fluids responsible for maintaining acid/base balance, transmitting nerve impulses and regulating fluid in and out of cells. Less frequently, the word chloride may also form part of the “common” name of chemical compounds in which one or more chlorine atoms are covalently bonded. For example, methyl chloride, with the standard name chloromethane (see IUPAC books) is an organic compound with a covalent C−Cl bond in which the chlorine is not an anion.

The implementation of a chloride-restrictive strategy in a tertiary ICU was associated with a significant decrease in the incidence of acute kidney injury (AKI) and the use of renal replacement therapy (RRT) [1].

Developmental, cellular, and subcellular variations in the direction of neuronal Cl- currents elicited by GABAA receptor activation have been frequently reported. We found a corresponding variance in the reversal potential (EGABA) for synapses originating from individual interneurons onto a single pyramidal cell. These findings suggest a corresponding variance in the cytoplasmic concentration of Cl- ([Cl- i]) in individual dendrites. We determined [Cl-]i in the murine hippocampus and cerebral cortex of both sexes by: 1) two-photon imaging of the Cl- sensitive, ratiometric fluorescent protein SuperClomeleon (sCLM); 2) Fluorescence Lifetime IMaging (FLIM) of the Cl- sensitive fluorophore MEQ; and 3) electrophysiological measurements of EGABA by pressure application of GABA and RuBi-GABA uncaging. Fluorometric and electrophysiological estimates of local [Cl-]i were highly correlated. [Cl-]i microdomains persisted after pharmacological inhibition of cation-chloride cotransporters (CCCs), but were progressively modified after inhibiting the polymerization of the anionic macromolecule actin. These methods collectively demonstrated stable [Cl-]i microdomains in individual neurons in vitro and in vivo and the role of immobile anions on its stability. Our results highlight the existence of functionally significant neuronal Cl- microdomains that modify the impact of GABAergic inputs.Significant Statement:Microdomains of varying chloride concentrations in the neuronal cytoplasm are a predictable consequence of the inhomogeneous distribution of anionic polymers such as actin, tubulin, and nucleic acids. Here, we demonstrate the existence and stability of these microdomains, as well as the consequence for GABAergic synaptic signaling: each interneuron produces a postsynaptic GABAA response with a unique reversal potential. In individual hippocampal pyramidal cells, the range of GABAA reversal potentials evoked by stimulating different interneurons was over 20 mV. Some interneurons generated postsynaptic responses in pyramidal cells that reversed at potentials beyond what would be considered purely inhibitory. Cytoplasmic chloride microdomains enable each pyramidal cell to maintain a compendium of unique postsynaptic responses to the activity of individual interneurons [2].

Astrocyte chloride

Astrocyte chloride

1)
Yunos NM, Bellomo R, Hegarty C, Story D, Ho L, Bailey M. Association between a chloride-liberal vs chloride-restrictive intravenous fluid administration strategy and kidney injury in critically ill adults. JAMA. 2012 Oct 17;308(15):1566-72. doi: 10.1001/jama.2012.13356. PubMed PMID: 23073953.
2)
Rahmati N, Normoyle KP, Glykys J, Dzhala VI, Lillis KP, Kahle KT, Raiyyani R, Jacob T, Staley KJ. Unique actions of GABA arising from cytoplasmic chloride microdomains. J Neurosci. 2021 Apr 26:JN-RM-3175-20. doi: 10.1523/JNEUROSCI.3175-20.2021. Epub ahead of print. PMID: 33903223.

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