

Abstract

Cerebral ischemia, also known as stroke, is one of the most common causes of death. It is accompanied by the formation of edema, which can be characterized as an influx of water and osmolytes into the brain, causing volume alterations. We recognize two types of cerebral edema – vasogenic, characterized by the disruption of the blood-brain barrier (BBB) and increase of the extracellular volume, and cytotoxic, caused by the increase of the volume of cells, mainly glia. The major contributors to the formation of cytotoxic edema are the astrocytes, which, in physiological conditions, are responsible for the maintenance of the BBB and keeping the homeostasis of the brain and spinal cord or central nervous system. The mechanism responsible for the process of volume and osmotic changes are the transmembrane channels, mainly aquaporin 4 (AQP4) and transient receptor potential vanilloid 4 (TRPV4). AQP4 is the main pathway for water influx as well as efflux when the edema subsides. TRPV4 is likely responsible for the maintenance of the osmotic balance of the organism, although its precise role in the formation of the edema has not yet been fully elucidated. The main aim of this thesis was to categorize the types of cerebral ischemia and edema, and to describe the process of cerebral edema formation and the role of glial cells in this process and nervous tissue recovery following ischemia. Another aim of this thesis was to review and summarize the available information about AQP4 and TRPV4 involvement in these processes.

Key words: brain, ion channels, water transport, astrocytes, NG2-glia