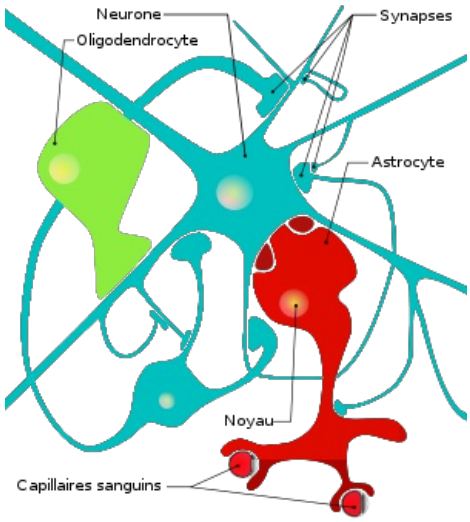
Glial Cell

Glial cells are also known as neuroglia, meaning nerve "glue".

Macroglia

1. **Astrocytes**: they have light cytoplasm, astrocytic filamentous bodies, numerous processes and they can form a glial "scar". Their functions are:
    1. to contribute to the extracellular potassium concentration maintenance
    2. to cover the basal lamina of the capillaries (part of the blood-brain-barrier) and transport some of the nutrients to neurons through their cytoplasm
    3. to cover the surface layer of the CNS (glia limitans)
    4. to cover the surface of neurons, fill in the interneuronal spaces
    5. by finger-like processes surround and isolate synaptic clefts, astrocytes may contribute to the inactivation of neurotransmitters (GABA, glutamate, glycine)
    6. after the brain is damaged, they form astrocytic scar (preventing the regrowth of central axons)
2. **Oligodendrocytes**: they contribute to the surface cover (myelination) of neuronal bodies and processes (glial cell processes surround unmyelinated fibers, layers of myelin). Oligodendrocyte‘s plasma membrane contains voltage-gated ion channels. Myelin-forming cell furnishes channels for the axon → myelin provides a signal to prevent the insertion of sodium channels into the internodal region of a myelinated nerve fibre.



Neuron glial cells diagram fr

Microglia

Microglia are functionally similar to macrophages; is probably of mesodermal origin; microglial cells are activated by some pathologic and reparatory processes in the CNS (gliosis)

Ependymal cells

They line the internal cavities of the CNS - part of the blood-brain barrier and they can absorb and secrete cerebrospinal fluid.

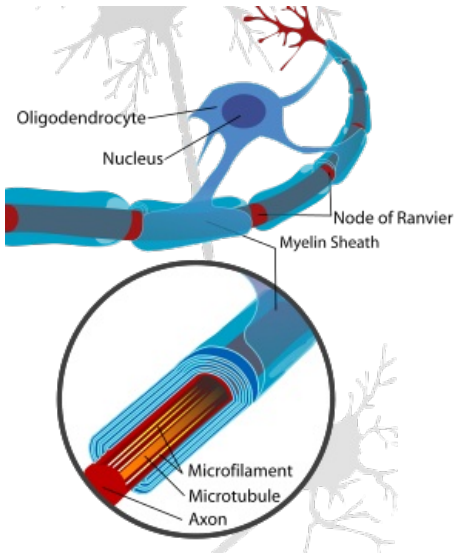
Myelin Sheath

Myelin sheath is formed by oligodendrocytes in the CNS and by Schwann cells in the peripheral nervous system. These cells wrap layer upon layer of their own plasma membrane in a tight spiral around axon. Each myelinating Schwann cells makes myelin layers at a single axon, forming a segment (internodium) that is about 1 mm long and consisting of up to 300 concentric layers of membrane.

Between one segment of sheath and the next, small regions of axon membrane remain bare (node of Ranvier).

Oligodendrocytes form similar segments of sheath at many separate axons simultaneously.

The insulating layer of the myelin sheath reduces the effective capacitance of the axon membrane and prevents almost all current leakage across it. Practically all the Na^+ channels of the axon are concentrated at the nodes, giving the density of several thousand channels per 1 mm^2. The sheathed portion of the axon membrane is not excitable, but has excellent cable properties. The membrane at the node of Ranvier is highly excitable and maintains the amplitude of the action potential → saltatory conduction, leading to acceleration of the conduction and conservation of the metabolic energy.



Neuron with oligodendrocyte and myelin sheath

The following GIF (http://lecannabiculteur.free.fr/SITES/UNIV%20W.AUSTRALIA/mb140/CorePages/Nervous/Images/Myelinani.gif) schematizes the process of myelination by a Schwann cell.

Control of extracellular potassium concentration

Glial cells have a resting potential of about -90 mV (more negative than a typical neuronal membrane), which is nearly identical to E_K (equilibrium potential for K^+). Plasma membrane contains various densities of K^+ channels, which are employed in the control of ECF's $[K^+]$, voltage-gated Na^+, K^+, Ca^{2+} channels, which incorporated into the neuronal membrane and/or may serve to generation of electrical signals with Ca^{2+} probably serving as a second messenger. Gap junctions among glia cells provide a low-resistance pathway for intercellular ionic current and a flow of some substances.

During the repolarization phase and the afterhyperpolarization of each neuronal action potential, a small amount of potassium leaves the neurons into the ECF. Following a sustained neuronal activity, a local increase in $[K^+]$ can be detected. This increase must be cleared to prevent depolarization of neurons and synaptic terminals in the vicinity, through the following ways:

1. Diffusion (a comparatively slow process)
2. Flow of potassium current through glia (spatial buffering) - as the glial resting potential lies close to E_K, increase of extracellular $[K^+]$ depolarizes the glial membrane:
    1. Since the depolarized cells are somewhat negative to the local E_K, potassium enters the cell and serve as charge carriers to the less depolarized regions of the glial syncytium
    2. the electrical circuit is completed by the extracellular flow of sodium and chloride ions →
    3. redistribution of K^+ during the neuronal activity
3. Active transport of extracellular K^+ back into neurons back the glia.

Glial cells at the site of an old brain injury are not as efficient in the spacial buffering of potassium ions → tendency for epileptic seizures in regions of an astrocytic scar, due to rise of ECF $[K^+]$ → depolarization → increased excitability (e.g.: seizures)

Links

Related articles

Sources

- POKORNY, Jaroslav. *Neurophysiology* [lecture for subject Physiology, specialization General Medicine, 1st Faculty of Medicine Charles University in Prague]. Prague. 2010.

Bibliography

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- DESPOPOULOS, Agamnenon – SILBERNAGL, Stefan. *Color Atlas of Physiology.* 5. edition. Thieme, 2003. ISBN 3135450058.