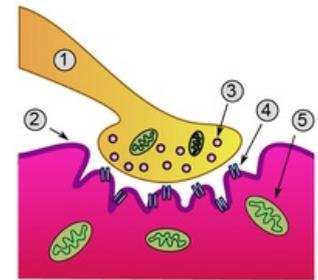


# Neuromuscular disc

**Neuromuscular disc** is a special type of chemical synapse]. Its function is to transmit the impulse from the neuron to the fiber skeletal muscle.

## Structure

- The presynaptic formation is represented by the **axonal termination of the motoneuron** and is deposited in shallow grooves formed by invagination of sarcolemma.
- The postsynaptic formation is represented by the **sarcolemma** (plasma membrane skeletal muscle fibers).
- **Primary synaptic cleft** is the space between the presynaptic ending and the muscle fiber.
- **The secondary synaptic cleft** is the space formed by the secondary invagination of the sarcolemma; The purpose of these invaginations is to increase the reception area of the synapse.
- The sarcolem is flanked by the basement membrane, so the synaptic cleft at the neuromuscular disc is wider (50–70 nm) than at the interneuronal synapses.

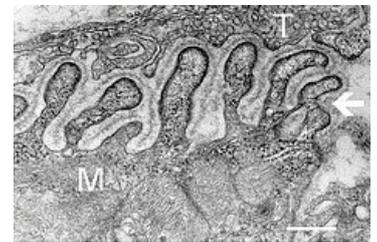


Neuromuscular disc: 1 – presynaptic formation, 2 – sarcolemma, 3 – synaptic vesicles, 4 – nicotinic receptors for ACh, 5 – mitochondrion

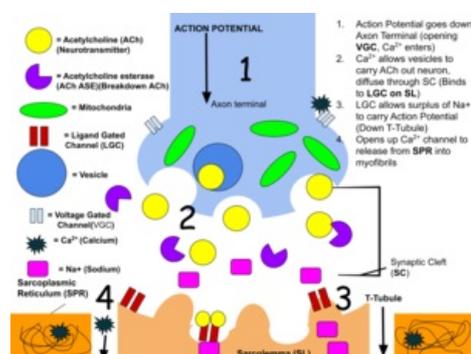
The proper connection creates the **finite branches of axon (telodendria);, which loses its myelin sheath, with sarcolemas of muscle fibers. These endings contain abundantly small, bright vesicles with [acetylcholine]], which is the mediator of these connections.**

## Transmission of impulse on neuromuscular disc

The impulse entering the nerve endings causes exocytosis of the synaptic vesicles and the release of mediator into the synaptic cleft. The mediator in the neuromuscular disc is **acetylcholine** (ACh - synthesized in nerve endings from choline and acetyl coenzyme A). The impulse that reaches the motor neuron termination (telodendria) **depolarization** opens the **calcium channel** and releases about 7000 acetylcholine molecules from vesicles located in the terminal part of the nerve. Acetylcholine exocytosis is transmitted through the **nicotine receptors signal for the formation of AP on sarcolemma. Activation of these receptors causes chemically controlled Na<sup>+</sup> channels to open and influx of Na<sup>+</sup> into the cell (based on the concentration gradient) causes local depolarization (disc potential),** which spreads to both sides of the disc. A muscle cell can respond to every impulse that comes to the nerve endings with an **action potential** (which is determined by the size of the disc, the number of activated receptors, and the density of voltage-gated Na<sup>+</sup> channels near the disc). Spontaneous emptying of one acetylcholine vesicle activates thousands of N-choline receptors, and to equip the action postsynaptic potential, it is necessary to empty about 100 vesicles followed by the opening of about 200,000 channels: a nerve-induced disc current of about 400 nA is formed. In order for neuromuscular transmission to function normally, acetylcholine must be inactivated, i.e. split into 2 inactive components (acetyl and choline) – the disc membrane can repolarize and respond to further release of acetylcholine. This is done by an enzyme, acetylcholinesterase.



Neuromuscular disc in electron microscope; T – terminal axon terminations, M – muscle fibre



Neuromuscular disc, [https://www.vfu.cz/files/fyziologie-prenosu-nervoveho-vzruchu\\_tp.pdf](https://www.vfu.cz/files/fyziologie-prenosu-nervoveho-vzruchu_tp.pdf)

Exocytosis of synaptic vesicles from nerve endings occurs not only at the action potential, but also individually when the vesicle comes into contact with the active part of the presynaptic membrane. In these cases, however, only a small amount of acetylcholine enters the synaptic cleft, so few nicotine receptors are activated. The resulting depolarization is less than 1 mV (the so-called *miniature disc potential*) and therefore does not cause the formation of an action potential on the muscle fiber.

## Links

## Related Articles

- Synapse
- Connection of excitation and contraction
- Ultrastructure of myofibrils, mechanism of contraction

## Bibliography

- TROJAN, Stanislav. *Lékařská fyziologie*. 4. edition. Grada, 2003. 771 pp. ISBN 80-247-0512-5.

