Amyotrophic Lateral Sclerosis (ALS) is a neurodegenerative disease associated with muscle weakness, atrophy and progressive paralysis. With a typical onset between 35 and 50, and a life expectancy after diagnosis of 3–5 years. The disease is affecting about 1-2 per 100 000 each year. In ALS death occurs as a result of respiratory failure.

ALS is still an incurable disease. Riluzole, a drug used in the treatment of ALS, causes many side effects and extends the lifespan of patients only about 2-3 months. There are also no proven manual therapies that can contribute to patients' condition. Until 10 years ago, physicians most often recommended limiting the physical effort of patients with ALS, due to the deteriorating physical fitness of patients, and to the damage that physical exercise may bring. It has even been suggested that certain sports might be associated with increased risk of this disease. However, recent studies conducted on over 650 ALS patients show that physical activity not only does not increase the risk of ALS, but also cause it to slow down. A mechanism explaining how physical activity affects the course and development of the disease is still in doubt.

In order to search for answers to questions related to ALS, the animal model of this disease is used, i.e. a mouse with a mutation discovered by Rosen in 1993 in the gene for superoxide dismutase type 1 (SOD1), whose locus-sites are located on the chromosome 21. Mice with this mutation develop ALS during their lifetime. Research using ALS mice shows that swimming training before the onset of the first symptoms of the disease, like no other activity, delays the time of onset of the first symptoms of the disease, and extends the lifespan of the mouse by 20%. Run training and training in a running wheel did not bring such a spectacular improvement. Therefore, it seems that the search for answers to questions related to ALS treatment in the subject of swim training is the most promising for the current state of knowledge about this disease.

The endurance training, used in this project, is known cause the increase of transcription factors PGC-1α and AMPK (AMP kinase) level which may result in increased mitochondrial biogenesis. Thus their higher content induced in cells may have a beneficial effect on defence against reactive oxygen and nitrogen species.

ALS is a diseases, which affects both nerves and muscles. The vitality of spinal cord cells depends largely on the efficiency of the auto-purification process, or autophagy. It is a catabolic process involving cell digestion of dead or damaged elements of its structure. Incorrect autophagy leads to cell death. On the one hand, the increased activity causes the cell to "digest" too fast, on the other hand, the reduced activity causes the accumulation of non-functional structures, which also leads to cell death. In the proposed research project, we assume that the autophagy process will be improved along with the application of swimming training in mice. The autophagy process will be monitored by examining the levels of beclin-1, LC3I/LC3II and p62 proteins.

The autophagy process can be induced by many factors. One of them is the mTOR kinase (mammalian target of rapamycin), whose function is to regulate cell growth, proliferation and movement. Recent study proves that supplementation with rapamycin of ALS mice, resulting in an increase in autophagy by mTOR regulation. The obtained results showed reduction in life, increase of neurodegeneration and faster development of the disease. In contrast, trehalose supplementation, an inducer of autophagy independent of mTOR, causes a significant delay in the development of the disease, extends lifespan and reduces neurodegeneration.

The aim of the study is to find the answers for the specific questions, whether swimming training will affect energy metabolism and oxidative stress in the spinal cord of ALS mice and control mice, whether will swimming training be a factor regulating the autophagy process; and whether the protective influence of swimming training will take place through the operation of AMPK, an inductor of mTOR-independent autophagy, generated as a result of this exercise.