Stress is one of the most alarming health problems in the modern world. This explains a pressing need for explorations into the biological mechanisms and pathways linking stress and health. Physiologically the central role in the stress response play glucocorticoids (GCs). However, the prolonged high level of GCs occurs in several pathologic conditions such as diabetes, starvation, cancer, burn injuries and depression, as well as after long-term medical treatment of synthesized GCs. The catabolic effects of GCs are well known and destructive role of GCs is manifested among others in the brain, bone, liver, heart, and skeletal muscles.

Skeletal muscle accounts for approximately 40% of body mass and is a major GCs target tissue. Under stressful or pathophysiological conditions, such as starvation, coldness or cancer, circulating GCs levels are greatly increased, which in turn decreases the rate of protein synthesis and increases proteolysis to generate amino acids to serve as precursors for hepatic gluconeogenesis. In the skeletal muscles, it leads to the main adverse effects, firstly reactive oxygen species generation (ROS), oxidative stress and secondly the skeletal muscle atrophy and muscle weakness.

The effects induced by GCs may be caused and regulated independently, at multiple levels of control, by the glucocorticoid receptor (GR) and/or activity of 11β-hydroxysteroid dehydrogenase type 1 (11β-HSD-1) both *in vitro* and *in vivo*. The GCs lead their signal mainly through the intracellular GR. However, the GCs bioavailability and action depend not only upon circulating levels or GR content but also on tissue-specific intracellular metabolism by 11β-HSD-1. Key metabolic tissues including liver, adipose tissue, and skeletal muscle express 11β-HSD-1, which a function is to convert inactive cortisone to cortisol or corticosterone. However, increased 11β-HSD-1 activity in skeletal muscle is linked with decreases in muscle mass and elevated gene expression associated with muscle atrophy. The high level of GR content in skeletal muscle may also induce adverse effects of GCs such as atrophy.

Although, the exact effect that GCs have on vitamin D metabolism remains controversial. One of the postulated therapeutics used to reduce the adverse effects of GCs is vitamin D. In general, the numerous studies suggest a positive role of vitamin D in sarcopenia prevention and inhibition of muscle atrophy by suppression of FOXO1 transcriptional activity. Additionally, some researches imply, that vitamin D has an antioxidant potential, both in the central nervous system and skeletal muscles. In work from our laboratory, we showed that the vitamin D deficiency may induce paraspinal muscle atrophy and decreases the concentration of vitamin D receptor (VDR) with simultaneously increasing level of peroxidation markers of lipids and proteins in multifidus muscle in patients with low back pain. There is also indication linking the vitamin D deficiency with GCs treatment. Data showed that the odds of having vitamin D deficiency are twice as likely in patients who reported GCs treatment as compared with those without steroid use.

The mechanism for vitamin D-mediated changes in skeletal muscle is not fully explained, however, it is known that Vitamin D acts mainly via specific binding to an intracellular VDR, interacting with specific nucleotide sequences of over 60 target genes. Moreover, the interaction of vitamin D with other steroid receptor superfamily receptors, including GR possibly occurs, still is insufficiently clarified. Nevertheless, data clearly show cooperative actions of vitamin D and GCs in modulating gene expression, what implies the potential reduction of the adverse effects of GCs excess (during vitamin D supplementation).

Taken together, recently published data indicates, that vitamin D deficiency is associated with lower VDR content, increased oxidative stress and altered the activity of antioxidant enzymes in skeletal muscle. Moreover, it is also reported that vitamin D regulates mitochondrial oxygen consumption and dynamics. The deficiency of vitamin D decreases oxygen consumption rate and induces disruption of mitochondrial function. What is more, dysfunctions of the mitochondrial respiratory chain and dangerous ROS generation are important factors in human pathologies, especially in neurodegenerative diseases where muscle atrophy is observed. We assume that vitamin D deficiency results from the GCs excess may lead to loss of VDR function and it could be partly responsible for the development of muscle atrophy with accompanying muscle weakness.

The main goal of the study is to determine the effect of chronic stress response on serum vitamin D level, oxidative stress, disruption of mitochondrial energy metabolism and atrophy in skeletal muscles. The purpose of the study is also to ascertain the possible mechanism(s) for these changes related to skeletal muscle.