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Modeling of contractile activity-induced fatigue in human skeletal muscle

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Intensive and/or prolong muscle contractions induce muscle fatigue leading to a decline in exercise performance. This phenomenon arises from central and peripheral changes limiting power output to avoid energy depletion which can cause cell death. The development of muscle fatigue is mainly regulated on the cellular level by both metabolic pathways engaged in ATP synthesis and ATP hydrolysis. However, despite the significant amount of experimental data on metabolic changes contributing to a decrease in power production, a comprehensive mathematical model of the muscle fatigue, which embraces all necessary cellular processes and includes both muscle fiber types, still does not exist. We markedly modified a previous modular model [1] comprising metabolic, signaling and gene expression levels by introducing a new function describing the dependency of power output on the phosphate concentration and pH which affect activation and sensitivity of myosin-actin filaments. To validate the extended version of the model, biochemical data obtained during an intermittent aerobic exercise (rhythmic knee extension for 4 minutes) was used [2]. Numerical analysis of the model was carried out in the BioUML platform [3].

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