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Interplay of Mitochondrial Dynamics and Function in Cartilage Tissue[1] ASHLEY LASKO, Rochester Institute of Technology, ARIEL BOHNER, Cornell University, MOUMITA DAS, Rochester Institute of Technology, MICHELLE DELCO, Cornell University — Post-traumatic osteoarthritis (PTOA) results from joint trauma. While there are no current curative treatments for PTOA, research suggests that such treatments must target events shortly after injury. An early response in damaged cartilage tissue is mitochondrial dysfunction. In healthy cells, the mitochondrial population is likely to be dominated by healthy, larger fused mitochondria, while in populations that are largely unhealthy, mitochondria are smaller and unfused to be easily removed by mitophagy. The production of energy, glycosaminoglycan (GAG), and the control of reactive oxygen species (ROS) within cartilage are indications of cell health. To understand the dynamics of mitochondria in cartilage and its interplay with mitochondrial function and cellular health, we developed a mathematical model that describes the processes of mitochondrial biogenesis, fission, fusion, mitophagy, and includes the dynamics of energy production, GAG, and ROS. We study this model with different initial conditions to determine which cases result in a healthy, a senescent, and a dead cell. Our results may help us understand what conditions must be met for a cell to survive mechanical stress, and how clinical treatments will preserve cell vitality after trauma.

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