Mechanical or chemical stress to articular cartilage kills chondrocytes resulting in the development of lesions on the cartilage surface. The typical injury response often causes collateral damage, which results in the spread of these lesions beyond the region of initial injury. This increases the chances of development of post-traumatic osteoarthritis. A balancing act between pro- and anti-inflammatory cytokines determines the amount of collateral damage that occurs and suggests possible therapies for limiting such damage. The goal of this work is to develop a mathematical representation for the interactions of such cytokines and the resulting effects on the chondrocyte population, that may be used to investigate the spread or abatement of post-injury cartilage damage. (Received September 11, 2011)