A HIERARCHICAL MODEL OF A QUIESCENT AND MAXIMALLY ACTIVATED FIBER DURING STRETCH

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Pliometric (lengthening) contractions are more likely to cause injury than either isometric or miometric contractions, but the behavior of muscle fibers during injury and the exact mechanism by which injury occurs were not demonstrated conclusively. For specific protocols, force and strain correlate well with measures of injury. Although techniques exist to study the mechanism of injury, resolving injury at the level of a single sarcomere has not been accomplished. The purpose of this study was to develop a computational model for a single fiber that reflects the changes in the mechanical environment of individual sarcomeres during their response to imposed loads. We hypothesize that the primary mode of injury is a loss of overlap between thick and thin filaments. The hypothesis will be supported if the total strain energy (TSE) is greater in the intrafibrillar regions than the interfibrillar proteins. The homogenization method was used to derive a technique for analyzing an incompressible, linear elastic, two scale model with an active stress (Hollister and Kikuchi, 1994, Biotech. Bioeng, 43:486-586). Both a quiescent fiber and a maximally activated fiber were modeled. The peak strains and total strain energy (TSE) at the sarcomere level were located in the region corresponding to the Z-line. For a constant strain rate of 2 Lf/sec from 0% to 1% strain in the quiescent and activated models, the maximum TSE was found to be 0.236 and 2.87 nN/mm2, respectively. Consequently for an uninjured fiber, the interfibrillar proteins play a negligible role in transferring forces and experience low strains. These observations support our hypothesis that the primary mechanism of injury is the loss of overlap of thick and thin filaments, with secondary damage to the passive structures. Supported by AG-06157.