

Molecular and Clinical Developments in Tendinopathy

Editorial Comment

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As orthopaedic surgeons, we commonly see patients with complaints of pain in and around tendons and/or tendon insertions. How often do we see runners with a painful Achilles, swimmers or throwers with a painful shoulder, or tennis players with a painful elbow? Despite the frequency of these conditions, our understanding of their etiology and optimal treatment remains limited. Several terms have been used to describe these conditions such as tendonitis, tendinosis, epicondylitis or bursitis. Many of these terms imply that the tendon pain is associated with an inflammatory process. Accordingly, traditional treatment options such as rest, ice, nonsteroidal antiinflammatory medications and corticosteroid injections have focused on reducing this inflammation. Recently the terms tendonitis, epicondylitis and bursitis are falling out of favor, as evidence of inflammation of the tendon and/or bursa is not typically present in these conditions. A more accurate term is tendinopathy. Tendinopathy is an all-encompassing term describing the pathologic, “degenerative” changes that occur within tendons in response to overuse and repetitive trauma. Histopathologic changes associated with tendinopathy include degeneration and disorganization of collagen fibers with increased cellularity, but minimal inflammation. Macroscopic changes include tendon thickening, loss of mechanical properties, and pain.

Our lack of comprehension of the pathologic processes that result in tendinopathy has been a major impediment to

progress in this field. A better understanding of the etiology of tendinopathy is essential in order to develop better treatment options for this condition. We are at an exciting time in this field as research in the last 5 years has improved our understanding of tendinopathy and has set the stage for further breakthroughs in the years to come. Advances in molecular biology, tissue culture, and the development of animal models of tendinopathy and tendon repair have facilitated the recent growth in this field.

The goal of this symposium is to provide the orthopaedic community with a picture of our current understanding of the pathogenesis of tendinopathy and to provide a framework that may guide treatment strategies in the future. The cutting-edge work of many leaders in this field is presented here. Through the use of molecular biology, cell and tissue culture, and sophisticated animal models, the pathologic changes associated with tendinopathy are being elucidated. Work presented in this symposium and elsewhere suggests tendinopathy develops in response to tendon overuse. This overuse results in the induction of molecular events that lead to “degeneration” as well as protective, regenerative changes. As the repetitive trauma of overuse continues, pathologic changes such as the release of matrix metalloproteinases and tendon cell apoptosis begin to outweigh the regenerative ones. The net result is degradation and disorganization of the extracellular matrix and a loss of viable tenocytes. This produces a biomechanically inferior tendon that is painful and prone to tearing. Maintaining adequate tension on the tendon also seems to play an important role in tendon homeostasis as the loss of tension appears to magnify the changes seen in response to overuse. This may explain why the early degenerative changes seen in response to overuse can be reversed by cessation of activity, but chronic tendinopathy does not typically resolve quickly through activity modification alone. The ultimate goal of

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this work is to find ways of preventing tendinopathy and better treatment options once tendinopathy has developed.

As Guest Editors, we are grateful to the staff of Clinical Orthopaedics and Related Research and to all of the authors

and reviewers who have contributed to this symposium. We have been impressed by the quality of the manuscripts submitted and feel that it represents the highest level, cutting edge research in the field of tendinopathy.