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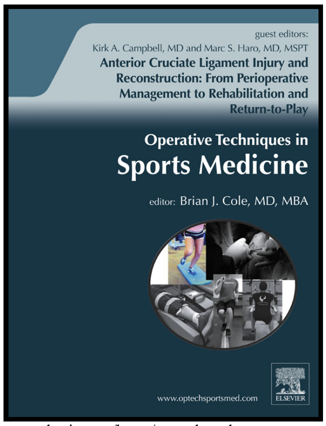

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Rotator Cuff Healing: Improving Biology

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Introduction

Rotator cuff tears are a common source of shoulder pain and disability among older adults and athletes. Full-thickness tears occur in up to 30% of the adult population over the age of 60 and rotator cuff tears are becoming increasingly prevalent as the population ages(1). Rotator cuff tears typically result from acute trauma, chronic degeneration or a combination of these two factors. Surgical treatment with open or arthroscopic repair is plagued by an unpredictable rate of recurrence, ranging from 11 to 94%(2–9). Notably, 85% of failures occur in the first 6 months following repair, implicating poor healing as a likely cause. Some patients have re-tears that are asymptomatic, but literature has demonstrated inferior functional outcomes and satisfaction scores in patients with re-tears following repair(10,11). While modern surgical technique and fixation materials have improved mechanical repair strength, biologic failure following repair remains a challenge. Animal models have shown that after rotator cuff tear and subsequent healing, the tendon-bone interface does not regain its normal histological configuration(12). In addition, the progression of healing and functional recovery is slow and laborious for patients. As a result, recent rotator cuff research has been largely focused on the biologic enhancement of tendon-to-bone healing.

Rotator cuff tears are typically accompanied by a challenging environment for tissue regeneration and tendon-to-bone healing following repair. Following repair, tendons heal in three phases: inflammation, proliferation, and remodeling. The initial inflammatory phase occurs in the first 7 days of healing and involves platelet deposition of fibrin and fibronectin, as well as the