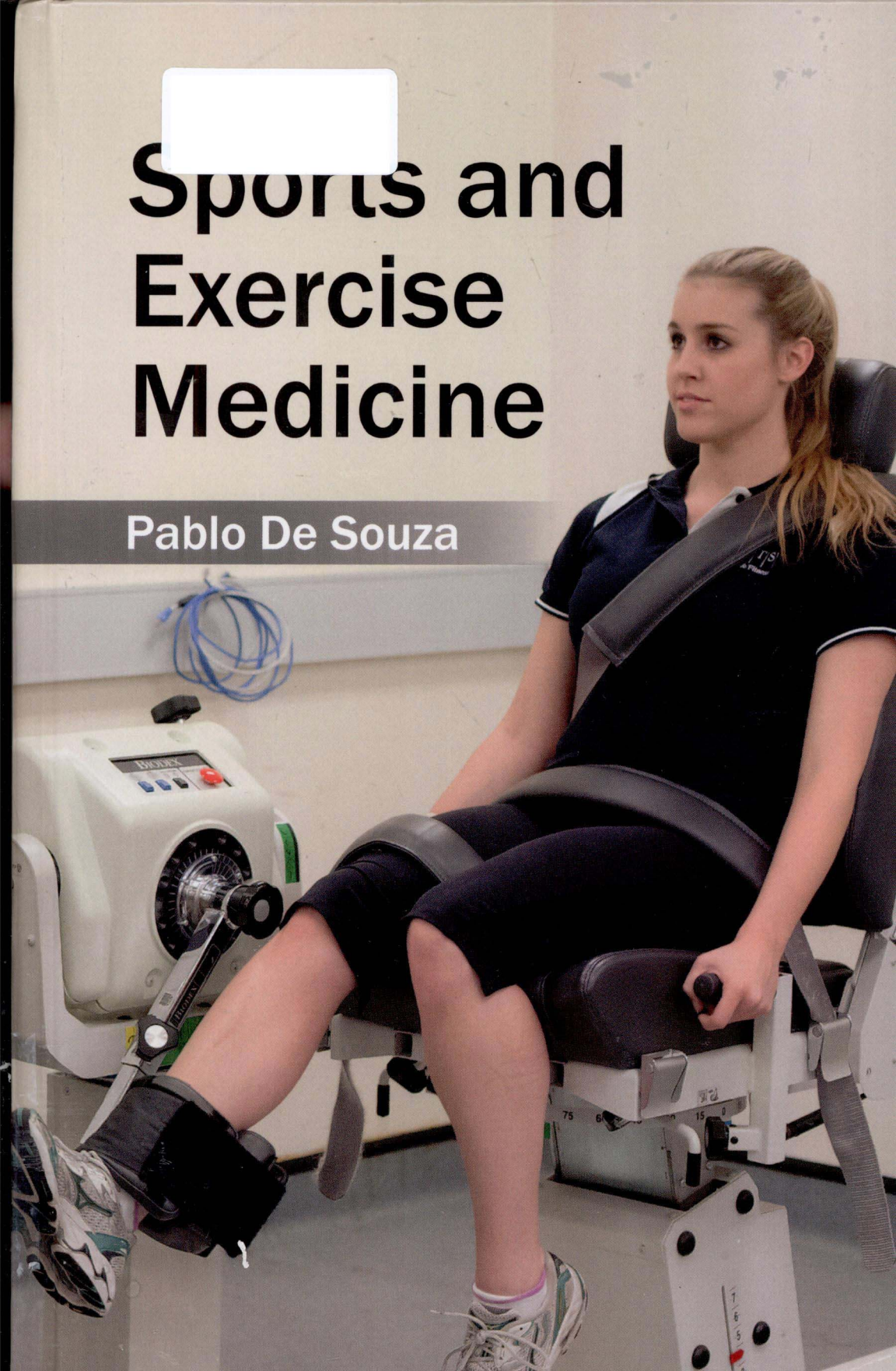
Sports and Exercise Medicine

Pablo De Souza



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Preface

A descriptive account on novel areas of current knowledge in the vast field of sports and exercise medicine has been provided in this insightful book. Experts from across the world have contributed in this book. It discusses the physiology behind sports injuries and describes novel and intriguing approaches to manage such injuries. It also explores the relation between health, performance and exercise by elucidating novel information in areas such as the use of iron supplementation for performance, exercise and immunity, impacts of exercise on reactive oxygen species, and the proposed advantages of authentic and simulated altitude training. It is a well-researched and comprehensive book which will serve as a valuable source of information for physiologists, physical conditioners, sports medicine specialists, coaches, students and physiotherapists.

The researches compiled throughout the book are authentic and of high quality, combining several disciplines and from very diverse regions from around the world. Drawing on the contributions of many researchers from diverse countries, the book's objective is to provide the readers with the latest achievements in the area of research. This book will surely be a source of knowledge to all interested and researching the field.

In the end, I would like to express my deep sense of gratitude to all the authors for meeting the set deadlines in completing and submitting their research chapters. I would also like to thank the publisher for the support offered to us throughout the course of the book. Finally, I extend my sincere thanks to my family for being a constant source of inspiration and encouragement.

Editor

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Sports Medicine

Chapter 1

The Physiology of Sports Injuries and Repair Processes

Kelc Robi, Naranda Jakob, Kuhta Matevz and
Vogrin Matjaz

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1. Introduction

Sports injuries are among the most common injuries and therefore present a significant public health problem. Physiologic processes after injuries are often neglected while much more attention is being paid to the management of symptoms. However, comprehension of these processes is becoming more and more important as therapies are getting increasingly focused on specific molecular and cellular processes. In recent decades, extensive research of tissue regeneration after injury and degeneration, including molecular pathways in healing, helped towards better understanding of this process and led to discoveries of new potential therapeutic targets. In this chapter physiology of sports injuries and the latest advances in understanding pathophysiological processes after injury will be discussed.

2. Physiology of tendon and ligament injury and repair

For skeletal muscles to act properly they must be attached to the bone. Tendons serve as mediators of force transmission that results in joint motion, but they also enable that the muscle belly remains at an optimal distance from the joint on which it acts. Tendons act as springs, which allows them to store and recover energy very effectively. Ligaments on the other hand attach bone to bone and therefore provide mechanical stability of the joint, guide joint motion through their normal range of motion when a tensile load is applied and prevent excessive joint displacement. Although tendons and ligaments differ in function, they share similar physiological features with a similar hierarchical structure and mechanical behavior.

2.1. Histoanatomical features of tendons and ligaments

Tendons are made up predominantly of collagen fibers embedded in proteoglycan matrix that attracts water and elastin molecules with a relatively small number of fibroblasts.

Fibroblasts are the predominant cell type in tendons. They are spindle shaped and arranged in fascicles with surrounding loose areolar tissue called peritenon. Cells are orientated in the direction of muscle loading. In mature tendon tissue they are arranged in parallel rows along the force transmitting axis of the tendon. Long cytoplasmic processes extend between the intratendinous fibroblasts, enabling cell-to-cell contact by gap-junctions.

Fibroblasts are connected to the extra cellular matrix (ECM) via integrins that permit the cells to sense and respond to mechanical stimuli which appears vital for their function because this way the mechanical continuum is established along which forces can be transmitted from the outside to the inside of the cell and vice versa. Integrins are also likely candidates for sensing tensile stress at the cell surface. It is also speculated that integrin-associated proteins are involved in signaling adaptive cellular responses upon mechanical loading of the tissue [1-5].

Type I collagen is the major constituent of tendons, accounting for about 95% of the dry tendon weight. Collagen type III accounts for about 5% of the dry tendon weight, but smaller quantities of other collagens are also present, including types V, VI, XII and type II collagen. The latter is primarily found in regions that are under compression [1-3].

Fibroblasts secrete a precursor of collagen, called procollagen, which is cleaved extracelullarly to form type I collagen. The synthesis of collagen fibrils occurs in two stages: intracellular and extracellular. The pro α-chains are initially synthesized with an additional signal peptide at the aminoterminal end with the function to direct movement of the polypeptides into the rough endoplasmic reticulum where it is cleaved off. Triple helix with three polypeptide chains wound together to form a stiff helical structure is formed intracellularly. Then the procollagen is secreted into the extracellular matrix where it is converted to collagen. Finally, collagen molecules aggregate and the cross-links responsible for its stable structure are formed [1-4].

The parallel arrangement of the collagen fibers in tendons enables them to sustain high tensile loads. Collagen molecules group together to form microfibrils, which are defined as 5 collagen molecules stacked in a quarter-stagger array. Microfibrils combine to form subfibrils, and those combine further to form fibrils (50-200 nm in diameter). Fibrils combine together to form fibers (3-7 μm in diameter) which further combine to form fascicles, and these group together to form a tendon. Fascicles are separated by endotenon and surrounded by epitenon. At the level of fascicles, the characteristic »crimp« pattern can be seen histologically (discussed later in this chapter) (Figure 1) [1-4].

Proteoglycans (PGs) account for 1-5% of the dry weight of the tendon. PGs are highly hydrophilic they attract water molecules. The predominant proteoglycans in the tendon are decorin and lumican. Biglycan and decorin (and collagen type V) regulate collagen fiber diameter in fibrillogenesis. Because decorin molecules form cross-links between collagen fibers they

may increase the stiffness of the fibrils. Proteoglycans are also responsible for lubricating collagen fibers and thus allowing them to glide over each other [2-4]. Aggrecan, a normal structure of articular cartilage, in found in tendons that are under compression [5].

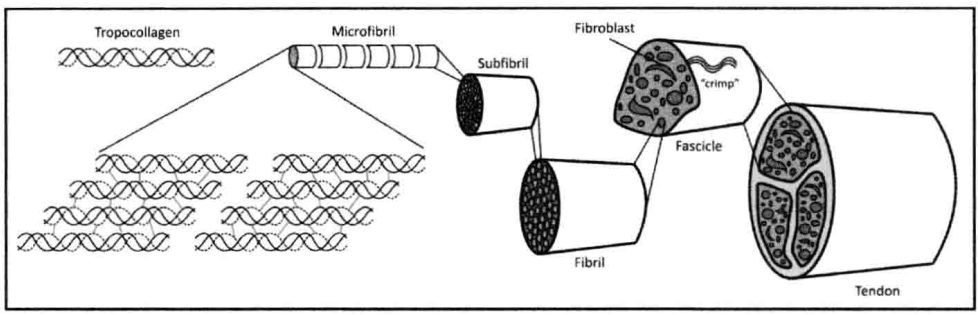

Figure 1. Structure of a tendon. See text for details. Adopted from Kastelic et al. [6]

Although tendons and ligaments are very similar in structure, there are some differences between them. (1) Ligaments consist of lower percentage of collagen molecules, but a higher percentage of the proteoglycans and water. (2) Collagen fibers are more variable and have higher elastin content and (3) fibroblasts appear rounder. (4) Furthermore, ligaments receive blood supply from insertion sites (Table 1) [1, 2].

Content / Feature	Ligaments	Tendons
Fibroblasts	20%	20%
Ground substance	20-30%	lower
Collagen	70-80%	Slightly higher
Collagen type I	90%	95-99%
Collagen type III	10%	1-5%
Elastin	Up to 2x collagen	scarce
Water	60-80%	60-80%
Organisation	More random	Organized
Orientation	Weaving pattern	Long axis orientation

Table 1. Differences between tendon and ligament structure

2.1.1. Vascular supply

There are two types of tendons: (1) tendons covered with paratenon, and (2) sheathed tendons. They mainly differ in vascular supply. In sheathed tendons a mesotenon (vincula) carries a vessel that supplies only one part of the tendon. Therefore, parts of the tendon are relatively avas-

cular and their nutrition depends on diffusion. On the other hand, paratenon-covered tendons receive their blood supply from vessels entering the tendon surface and forming a rich capillary system. Because of the difference in the vasculature, paratenon-covered tendons heal better. As stated above, ligaments receive their blood supply from insertion sites [2, 3].

There is still an ongoing debate about the efficiency of the blood supply to tendons during exercise. Experiments showed that although the increase in tendon blood flow is somehow restricted during exercise, there is no indication of any major ischemia in the tendon region. The question remains how blood flow to the tendon region is regulated. Several candidates as regulators of blood flow in skeletal muscle have been proposed, and it is possible that similar substances and metabolites are vasoactive also in the tendon region suca as bradykinin [2].

2.1.2. Insertion sites

As tendons attach skeletal muscles to bony structures, two types of tendinous junction are to be distinguished – osteotendinous where tendon attaches to the bone and musculotendinous where it attaches to the muscle. Four distinct zones have been observed at the osteotendinous junction, with a gradual change between them (Figure 2). (1) The first zone is structurally similar to the tendon propter, but with smaller amounts of PG decorin. This zone is followed by (2) fibrocartilage, where mostly collagen type II and III are found, but also small amounts of types I, IX and X. Furthermore, there is less PGs aggrecan and decorin. In the third zone, (3) mineralized fibrocartilage is made up of mainly collagen type II, but large quantities of collagen X and aggrecan are also present. The fourth zone is (4) bone, build up mainly of collagen type I and minerals [1-3].

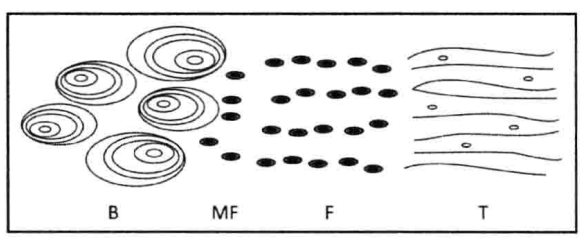

Figure 2. Diagram of a osteotendinous junction; B – bone; MF – minarelized fibrocartilage; F – fibrocartilage; T – tendon.

At musculotendinous junction, muscle cells are involuted and folded to provide maximal surface for attachment where fibrils attach. Sarcomeres of the fast contracting muscles are shortened at the junction, which may reduce the force intensity within the junction [3].

Ligaments insert into bone in two ways: through indirect or direct insertions. In indirect insertions the superficial layer is continued at with the periosteum and the deeper layer anchores to bone via Sharpey's fibers. In direct insertions, fibers attach to bone at 90° angle. Four distinct zones have been observed, with a gradual change between ligament midsubstance, fibrocartilage, mineralized fibrocartilage, and bone [2].

2.1.3. *Biomechanics of tendons and ligaments*

Typical parameters describing the tendon/ligament mechanical properties are *strain*, which describes the elongation/deformation of the tendon (ΔL) relative to the normal length (L0); *stress*, the tendon force (Ft) relative to the tendon cross-sectional area (CSA), *stiffness*, the change in tendon length (ΔL) in relation to the force applied (ΔFt) and *modulus*, which describes the relation between tendon stress and tendon strain and represents the properties independently of the CSA (Figure 3 and 4). High modulus indicates stiffer tissue [7-9].

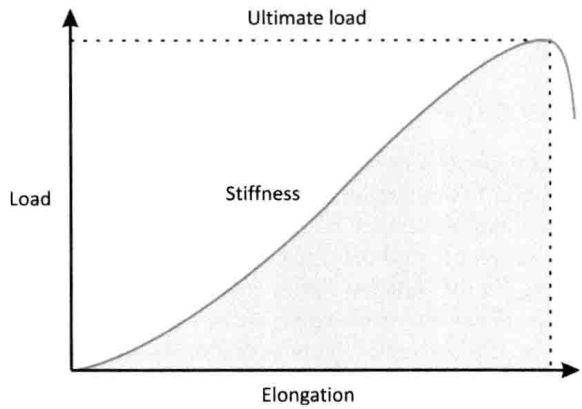

Figure 3. Structural properties of the bone-ligament-bone complex - A load/elongation curve; stiffness is represented by the slope of the curve; ultimate load is the highest load applied to the bone-ligament-bone complex before failure; the dashed area under the curve is the maximum energy stored by the complex [7, 9].

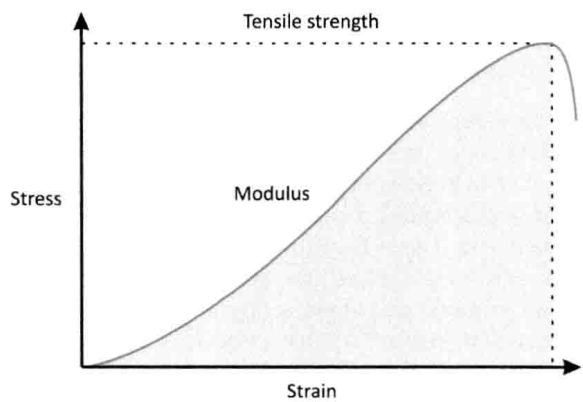

Figure 4. Mechanical properties of the bone-ligament-bone complex – A stress/strain curve; modulus is represented by the slope of the curve; tensile strength is the maximum stress of the bone-ligament-bone complex before failure; the dashed area under the curve represents the strain energy density [7, 9].

The biomechanics of ligaments is similar to tendon biomechanics. The biomechanical properties of ligaments are described as either structural properties of the bone-ligament–bone complex or the material properties of the ligament midsubstance itself. Structural properties of the bone-ligament-bone complex depend on the size and shape of the ligament, therefore they are extrinsic measures. They are obtained by loading a ligament to failure and therefore represented as a load-elongation curve between two defined limits of elongation. Mechanical properties are intrinsic measures of the quality of the tissue substance and are represented by a stress-strain curve [7, 8].

A tendon is the strongest component in the muscle-tendon-bone unit. It is estimated that tensile strength is about one-half of stainless steel (e.g. 1 cm^2 cross-section of a tendon can bear weight of 500-1000 kg) [3, 9].

2.1.4. Non-linear elasticity and viscoelasticity

There are three distinct regions of the stress/strain curve: (1) the toe region, (2) the linear region, and (3) the yield and failure region (Figure 5). In normal activity, most ligaments and tendons exist in the toe and somewhat in the linear region. This region is responsible for nonlinear stress/strain curve, because the slope of the toe region is not linear. The toe region represents "un-crimping" of the collagen fibrils. Since it is easier to stretch out the crimp of the collagen fibrils, this part of the stress strain curve shows a relatively low stiffness compared to linear portion. The toe region ends at about 2% strain when all crimpled fibers straighten. When all collagen fibrils become uncrimped, the collagen fibers stretch. The tendon deforms in a linear fashion due to the inter-molecular sliding of collagen triple helices. If strain is less than 4%, the tendon will return to its original length when unloaded, therefore this portion is elastic and reversible and the slope of the curve represents an elastic modulus. When a tendon/ligament is stretched beyond physiological limits, some fibrils begin to fail. Micro failure accumulates, stiffness is reduced and the ligament/tendon begins to fail. This occurs when intramolecular cross-links between collagen fibers fail. The tendon therefore undergoes irreversible plastic deformation. When the tendon/ligament is stretched to more than 8-10% of its original length, macroscopic failure follows [2, 3, 7].

Viscoelasticity refers to time dependent mechanical behavior. In other words, the relationship between stress and strain is not constant but depends on the time of displacement or load. There are three major characteristics of a viscoelastic material of ligaments and tendons: creep, stress relaxation, and hysteresis or energy dissipation. *Creep* indicates increasing deformation under constant load. This is in contrast with the usual elastic material, which does not elongate, no matter how long the load is applied (Figure 6). *Stress relaxation* is a feature of a ligament or tendon meaning that stress acting upon them will be eventually reduced under a constant deformation (Figure 7). When a viscoelastic material is loaded and unloaded, the unloading curve is different from the loading curve. This is called *hysteresis*. The difference between the two curves represents the amount of energy that is dissipated or lost during loading (Figure 8). If loading and unloading are repeated several times, different curves are obtained. However, after about 10 cycles, the loading and unloading curves do not change anymore, but they are still different. In other words, the amount of hysteresis un-