SPORTS INJURIES

Edited by

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Sports Injuries

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Sports Injuries

Foreword

As a middle distance athlete, I have always recognized the challenge of prevention and treatment of sport related injuries. Even a minor 'niggle' can have serious consequences on sporting performance. All athletes—recreational or elite—should have access to expert medical care to allow prompt diagnosis and appropriate intervention to allow a safe and effective return to sport.

Since my athletic career, the delivery of medical care to athletes has progressed beyond recognition. In the past decade alone we in the UK have seen the recognition and development of Sport and Exercise Medicine as a medical specialty, the establishment of the Faculty of Sport & Exercise Medicine and the 2012 Olympic games being awarded to London. These events represent a major leap forward for sport in the UK, and the development of medical expertise should be seen as part of the legacy of the 2012 Olympic Games.

I congratulate Drs Speed and Hutson for their Textbook of Sports Injuries, with contributions from a number of authors who are true experts in their fields. The text is a thorough, highly illustrated and practical guide to expert management of sport injuries across a variety of sports. It takes the reader through basic and applied sciences, to in-depth but accessible sections on injuries and concludes with a section on sport specific complaints.

This textbook will undoubtedly make a significant contribution to the education of a range of professionals working in the field of Sport & Exercise Medicine. Specialty trainees, specialists, and allied practitioners alike will find it an enjoyable and informative read, and an invaluable reference.

Steve Cram
Chairman, English Institute of Sport
Middle distance runner
European, Commonwealth and World Champion and
Olympic Silver medalist

Preface

The evolution of Sports and Exercise Medicine (SEM) in the UK over the last century is well documented in the period after the foundation of BASM (British Association of Sports Medicine) in 1952. Prior to the 1950s, records of services to the injured are sketchy, though there is evidence of a Footballers' Hospital in Manchester at the turn of the nineteenth century, and the creation of the first official post of Medical Officer to the BOA (British Olympic Association) in 1928, the same year as the foundation of FIMS (International Federation of Sports Medicine). BASM (latterly renamed BASEM), was the spur to the formation of the ISM (Institute of Sports Medicine) in 1965, at much the same time as the creation of the English Sports Council. Finally, in February 2005, Sports and Exercise Medicine was established as a standalone specialty in the United Kingdom. The Faculty (FSEM) was created under the auspices of the Royal College of Surgeons of Edinburgh and the Royal College of Physicians of London (RCP) and was officially launched by its patron, the Princess Royal, at the RCP in September 2006.

The Faculty declares that the speciality is 'founded on the disease and wellness models of medicine. It is through the latter, in particular, that SEM physicians in the future can play a leading and vital role in helping determine the Health of the Nation'. The role of the specialist physician in SEM includes the promotion of exercise for health; the prescription of exercise programmes for needy subgroups of the population; management of soft tissue injuries with specific focus on exercise- and sport-related injuries; the provision of highly skilled services to elite athletes; rehabilitation of able and disabled sportsmen and women of all standards 'to expedite return to physical activity, work and increase participation in sport'.

This textbook is intended to provide guidance for all clinicians involved in sports medicine practice, including therapists, the enthusiastic doctor,the sub-specialist SEM doctor, and the specialist consultant in SEM, all of whom require appropriate skills set upon a sound knowledge base of injuries related to sport and exercise, thereby providing a high level of service to all exercising patients from diverse backgrounds who require help.

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Abbreviations

ABC	airway–breathing–circulation
ACL	anterior cruciate ligament
ACPSM	Association of Chartered Physiotherapists in Sports Medicine
AF	annulus fibrosus
ALL	anterior longitudinal ligament
ANA	antinuclear antibodies
AP	anteroposterior
APL	abductor pollicis longus
AS	ankylosing spondylitis
ASIS	anterior superior iliac spine
ATLS	Acute Trauma Life Support
BMC	bone mineral content
BMD	bone mineral density
BMI	body mass index
BMP	bone morphogenic protein
BMSF	bone marrow stromal fibroblast
CI	confidence interval
CK	creatine kinase
CNS	central nervous system
COMP	cartilage oligomeric matrix protein
COX	cyclo-oxygenase
CRP	C-reactive protein
CT	computed tomography
DOMS	delayed-onset muscle soreness
ECM	extracellular matrix
EDL	extensor digitorum longus
EHL	extensor hallucis longus
EIMD	exercise-induced muscle damage
EM	electromagnetic
EMG	electromyography
ESR	erythrocyte sedimentation rate
ESWT	extracorporeal shock wave therapy
FCU	flexor carpi ulnaris
FDA	US Food and Drug Administration
FDB	flexor digitorum brevis
FDL	flexor digitorum longus
FDP	flexor digitorum profundus
FDS	flexor digitorum superficialis
FGF	fibroblast growth factor
FHL	flexor hallucis longus
GDF	growth and development factor
GTN	glyceryl trinitrate
HGF	hepatocyte growth factor
HU	Hounsfield unit
IGF	insulin-like growth factor
IL	interleukin
IP	interphalangeal
ITB	iliotibial band
IU	international unit
IVD	intervertebral disc
LA	local anaesthetic
LCL	lateral collateral ligament
LED	light-emitting diode
LHB	long head of the biceps
LIUS	low-intensity ultrasound
MCL	medial collateral ligament
MCP	metacarpophalangeal
M-CSF	macrophage-colony stimulating factor
MDP	methylene diphosphonate
MRI	magnetic resonance imaging
MSC	mesenchymal stem cell
MTP	metatarsophalangeal
MTPJ	metatarsophalangeal joint
NMES	neuromuscular electrical stimulation
NP	nucleus pulposus
NSAIDs	non-steroidal anti-inflammatory drugs
OA	osteoarthritis
ODI	Oswestry Disability Index
OPG	osteoprotegrin
PBL	problem-based learning
PDGF	platelet-derived growth factor
PG	proteoglycan, prostaglandin
PGE2	prostaglandin E2
PIPJ	proximal interphalangeal joint
PLL	posterior longitudinal ligament
PNF	proprioceptive neuromuscular facilitation
pQCT	peripheral quantitative computed tomography
PRICES	protection, rest, ice, elevation, support
PSIS	posterior superior iliac spine
PTFJ	proximal tibiofibular joint
PTFL	posterior talofibular ligament

PTH	parathyroid hormone
RCT	randomized controlled trial
RF	rheumatoid factor
RICE	rest, ice, compression, elevation
RoM	range of motion
ROS	reactive oxygen species
SAWS	sportsman's abdominal wall syndrome
SCIWORA	spinal cord injuries without obvious radiological abnormalities
SCJ	sternoclavicular joint
SEM	sports and exercise medicine
SIJ	sacroiliac joint
SLAP	superior labrum from anterior to posterior
SPN	superficial peroneal nerve
SPR	superior peroneal retinaculum
STIR	short inversion time inversion recovery
TA	tibialis anterior
TENS	transcutaneous electrical nerve stimulation
TFCC	triangular fibrocartilage complex
TFL	tensor fascia lata
TGF	transforming growth factor
TIMP	tissue inhibitor of metalloproteinase
TLHB	tendon of the long head of the biceps
TNF	tumour necrosis factor
TOS	thoracic outlet syndrome
TP	tibialis posterior
TTS	tarsal tunnel syndrome
US	ultrasonography, ultrasonic
VAS	visual analogue scale
VEGF	vascular endothelial growth factor
Vo2 max	maximum oxygen uptake
WADA	World Anti-Doping Agencycontributors

Contents

SECTION 1

Basic science

1.1

Tendon and ligament biochemistry and pathology

Graham Riley

Introduction

Tendon and ligament pathologies are often seen by general practitioners, rheumatologists, and specialists in musculoskeletal medicine and sports and exercise medicine. Increased participation in recreational exercise and sport, although beneficial for general health and well-being, has led to a substantial rise in their incidence. However, only belatedly are these conditions receiving the attention they deserve from the research community.

Tendons and ligaments are dense fibrous connective tissues, important for joint movement and stabilization, respectively. They have a similar composition and structure and are metabolically active and capable of responding to extrinsic factors such as mechanical load, exercise, and immobilization. Tendon and ligament are grouped together in this chapter so as to avoid repetition of common principles, although it is important to note that there are differences in the range of pathology affecting these tissues. Despite superficial similarities, there are differences in structure, composition, and function between tendon and ligament that make it unwise to extrapolate from one tissue to another.

The purpose of this chapter is to review what is known about the biochemistry and pathology of tendons and ligaments, focusing on conditions that are relevant to the practising clinician in sports and exercise medicine. The chapter begins with an overview of tendon and ligament biochemistry, which is fundamental to an understanding of the disease process.

Extracellular matrix components of tendon and ligament

Like all connective tissues, tendon and ligament are composite materials consisting of collagens, proteoglycans, and a variety of other non-collagenous proteins. Although the extracellular matrix (ECM) is predominantly collagen, many other components contribute to the strength, elasticity, and physiology of the tissue (Fig. 1.1.1). The relatively few cells in the mature tissue are responsible for the synthesis and organization of the ECM. These cells are also responsible for the degradation and replacement of ECM, an activity that is particularly important in tissue development, injury, and pathology. Degradation of the ECM is mediated largely by the resident fibroblasts and macrophages, either by phagocytosis or extracellular proteolysis. The maintenance of the normal tendon and ligament architecture is the result of a delicate balance between the synthesis and degradation of ECM. Disruption of this balance leads to a loss of ECM organization and will ultimately lead to pathology.

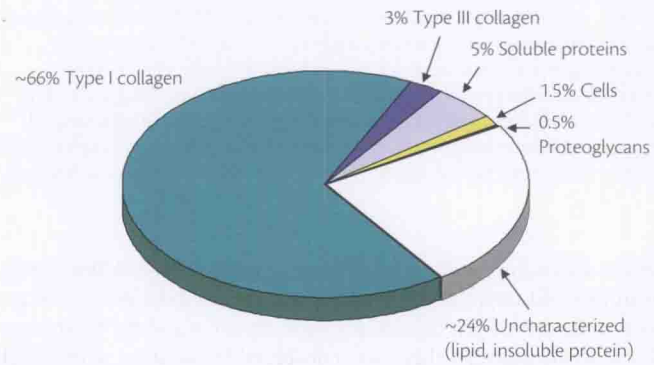

Fig. 1.1.1 Matrix (ECM) composition of tendon. Pie chart showing the approximate proportions of ECM components in a typical flexor tendon as a percentage of the tendon dry weight. The bulk of the tendon is collagen, predominantly type I with a small amount (up to 5 per cent) of type III collagen. The proportion of proteoglycan varies in different tendons and in different sites, representing 0.5–3.5 per cent of the matrix dry weight. A large proportion of the matrix is uncharacterized, thought to be insoluble protein and lipids. The composition of ligament is similar, although the proportion of type III collagen is greater—usually around 10 per cent but up to 40 per cent in some ligaments.

Collagens

The collagen family of glycoproteins has been extensively reviewed elsewhere (Van der Rest and Garrone 1990; Prockop and Kivirikko 1995). Collagen has a unique triple helix structure that, once secreted into the ECM, spontaneously associates with other collagen molecules to form characteristic banded fibrils (Fig. 1.1.2). Since early descriptions of collagen as a single entity, it is now known that there are at least 27 different collagen types, each with a different structure, function, and tissue distribution (Tables 1.1.1 and 1.1.2). However, it is increasingly apparent that collagens, once thought to be restricted to specific tissues, are in fact distributed more widely, albeit as minor constituents of the ECM though no doubt important for the tissue structure and function.

Tendon and ligament are predominantly type I collagen, organized into fibril bundles and orientated with the long axis of the tissue. Collagen comprises between 50 and 85 per cent of the tendon dry

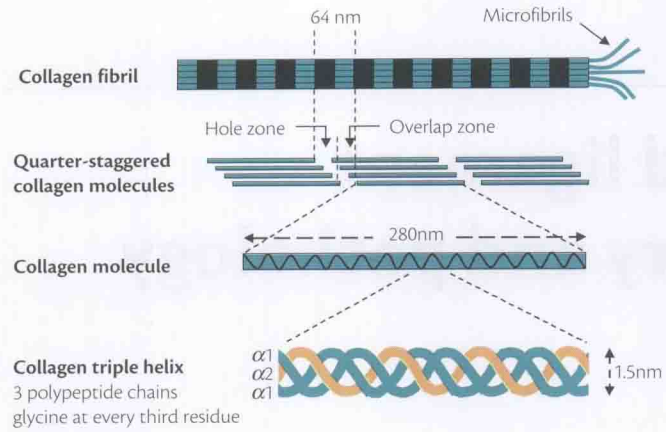

Fig. 1.1.2 Schematic representation of the prototypic collagen molecule. The collagen (type I) molecule consists of three polypeptide chains (designated α1 and α2), wound in a tight triple helix. This is made possible by the unusual amino acid content, with a high proportion of glycine, proline, and hydroxyproline in repeating triplets. Once secreted, each collagen molecule spontaneously associates end-to-end with other molecules, forming a quarter-staggered array. The 'hole zone' takes up electron-dense stains, which accounts for the striated appearance of the fibril under electron microscopy. (Reproduced (modified) from Cawston, T.E. (1998), fig. 1, p. 130, © 1998 with permission from Elsevier Science.)

weight depending on the tendon, species, and location (Elliott 1965). In studies of human tendon, collagen comprised on average 56 per cent of the dry weight in both the supraspinatus and the biceps brachii tendon, although there was considerable variation within each sample group (Riley *et al.* 1994).

In the tendon mid-substance, approximately 95 per cent of the collagen is type I, with the remainder consisting of types III, IV, V, VI, XII, and XIV. Type V collagen is thought to form the core of the collagen fibril and may comprise around 2 per cent of the total collagen (Niyibizi *et al.* 1994). Type III collagen is thought to represent up to 5 per cent of the total collagen (Niyibizi *et al.* 1994), although in studies of normal human supraspinatus tendon we found an average of just over 2 per cent type III collagen (Riley *et al.* 1994). Type III collagen is generally restricted to the endotenon or epitenon, the thin layers of connective tissue that surround the collagen fibre bundles (Duance *et al.* 1977). However many older supraspinatus tendons show distribution of type III collagen throughout the matrix, consistent with the formation of heterotypic fibre bundles (Kumagai *et al.* (1994) and Riley, G.P., unpublished observations). A similar distribution has been found in skin, with type I and type III collagens cross-linked together and found within the same fibrils (Lapiere *et al.* 1977). This interaction appears to have a role in conditioning the collagen fibre organization and ultimate fibre diameter. Type III collagen tends to form smaller diameter fibrils, and changes in the ratio of type III to type I collagen are correlated with the average fibril diameter (Birk and Mayne 1997). The resulting tissue may be more compliant and less resistant to mechanical strain. Although there have been suggestions that changes in the proportion of collagen types may be a consequence of ageing (Kumagai *et al.* 1994), this is not a feature of all tendons, which suggests that other factors such as mechanical strain and injury are implicated. In the supraspinatus tendon the increase in type III collagen is thought likely to represent a history of previous injury and matrix remodelling events in the tissue. Changes in the proportion of collagen types I and III have been reported in other ruptured tendons (Holz 1980), consistent with some underlying process that weakens and predisposes the tendon to rupture. This is discussed in more detail below.

Type IV collagen is present in the basement membranes of tendon blood vessels, and type V collagen is encapsulated within the type I collagen fibrils (von der Mark 1981). Type VI collagen is found distributed throughout the matrix (Bray *et al.* 1993), and collagen types XII and XIV are associated with the collagen (type I) fibril surface (von der Mark 1981; Shaw and Olsen 1991). The precise roles of these so-called 'minor' collagens are uncertain, but they are thought to be important for both cell and matrix interactions.

Ligaments are essentially similar in collagen composition to tendon, although the proportion of type III collagen is generally higher, with reported values from 12 per cent to greater than 40 per cent in some ligaments (Amiel *et al.* 1984; Johnston *et al.* 1995). The amount of type III collagen is thought to account for the elasticity of the tissue, with higher levels in intrinsic ligaments of the wrist (41 per cent) compared to extrinsic ligaments (19 per cent), and these correlated with the strain to failure (Johnston *et al.* 1995). Type III collagen is relatively abundant in the epiligament, the equivalent of the epitenon, that surrounds the ligament fibre bundles (Amiel *et al.*1984). Type VI collagen in ligament is found in microfilaments stretching between the collagen fibrils in a network of electron-dense seams (Bray *et al.* 1993).

Site-specific variations in collagen composition

The collagen composition and organization varies at different sites within tendons and ligaments—they are not homogeneous tissues. At the insertion of bovine knee ligaments and Achilles tendons there are found collagen types II, IX, X, and XI in addition to collagen type I (Fukuta *et al.* 1998; Visconti *et al.* 1996). Type XIV collagen is more abundant at the insertion than elsewhere in the tendon/ligament (Niyibizi *et al.* 1994). Type X collagen is found at the Achilles insertion in the rodent, associated with the region of transition between calcified and non-calcified fibrocartilage (Fujioka *et al.* 1997). In the human Achilles at the bone insertion there are found collagen types I, II, III, V, and VI (Waggett *et al.* 1998). A higher concentration of type III is reported in the rotator cuff tendon at or near the insertion where it might contribute to the high incidence of tear at this site (Riley *et al.* 1994; Kumagai *et al.* 1994; Fan *et al.* 1997). Type II collagen is also found at regions of tendon fibrocartilage where tendon is compressed as it wraps around bone or passes through fibrous pulleys (Ralphs *et al.* 1991; Kumagai *et al.* 1994). In these regions the collagen has a different organization with a meshwork structure reminiscent of that of cartilage (Vogel and Koob 1989). The cells in these regions are rounded and chondrocyte-like, and express proteoglycans once thought to be restricted to cartilage (see below). Type VI collagen, which is normally associated with micro-fibillar networks between adjacent collagen fibres in the tendon mid-substance, is cell-associated in fibrocartilage, similar to the distribution seen in articular cartilage. Levels of matrix gene expression in the fibrocartilaginous regions of bovine tendon are higher than in the tension-bearing regions of the same tendon, demonstrating increased matrix turnover at these sites (Perez-Castro and Vogel 1999).

Collagen fibril organization and structure

The hierarchical structure of a typical tendon was described by Kastelic *et al.* (1978) as consisting of collagen molecules laid down