Structure of the tendon connective tissue

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Tendons consist of collagen (mostly type I collagen) and elastin embedded in a proteoglycan-water matrix with collagen accounting for 65–80% and elastin approximately 1–2% of the dry mass of the tendon. These elements are produced by tenoblasts and tenocytes, which are the elongated fibroblasts and fibrocytes that lie between the collagen fibers, and are organized in a complex hierarchical scheme to form the tendon proper. Soluble tropocollagen molecules form cross-links to create insoluble collagen molecules which then aggregate progressively into microfibrils and then into electronmicroscopically clearly visible units, the collagen fibrils. A bunch of collagen fibrils forms a collagen fiber, which is the basic unit of a tendon. A fine sheath of connective tissue called endotenon invests each collagen fiber and binds fibers together. A bunch of collagen fibers forms a primary fiber bundle, and a group of primary fiber bundles forms a secondary fiber bundle. A group of secondary fiber bundles, in turn, forms a tertiary bundle, and the tertiary bundles make up the tendon. The entire tendon is surrounded by a fine connective tissue sheath called epitenon. The three-dimensional ultrastructure of tendon fibers and fiber bundles is complex. Within one collagen fiber, the fibrils are oriented not only longitudinally but also transversely and horizontally. The longitudinal fibers do not run only parallel but also cross each other, forming spirals. Some of the individual fibrils and fibril groups form spiral-type plaits. The basic function of the tendon is to transmit the force created by the muscle to the bone, and, in this way, make joint movement possible. The complex macro- and microstructure of tendons and tendon fibers make this possible. During various phases of movements, the tendons are exposed not only to longitudinal but also to transversal and rotational forces. In addition, they must be prepared to withstand direct contusions and pressures. The above-described three-dimensional internal structure of the fibers forms a buffer medium against forces of various directions, thus preventing damage and disconnection of the fibers.

Macroscopic structure of tendons

Tendons are anatomic structures interposed between muscles and bones transmitting the force created in the muscle to bone, and, in this way, making joint movement possible. Basically, each muscle has two tendons, proximal and distal. The point of union with a muscle is called a myotendinous junction (MTJ), and the point of union with a bone an osteotendinous junction (OTJ). The attachment of the proximal tendon of a muscle to bone is called a muscle origin, and that of the distal tendon an insertion.

Healthy tendons are brilliant white in color and fibro-elastic in texture, showing great resistance to mechanical loads. They may vary considerably in shape and in the way they are attached to bone ranging from wide and flat tendons to cylindrical, fan-shaped, and ribbon-shaped tendons. Muscles designed to create powerful, resistive forces, like the quadriceps and triceps brachii muscles, have short and broad tendons, while those that have to carry out subtle and delicate movements, like the finger flexors, have long and thin tendons.

The surrounding structures of the tendons can be divided into five categories. The fibrous sheaths or retinacula (1) are the canals through which the (usually long) tendons glide during their course. Without these bony grooves and notches friction could considerably impair tendon gliding. The grooves and notches are usually lined with a fibro-cartilaginous floor and covered with the fibrous sheath or retinaculum. Characteristic examples are the retinacula of the extensors and flexors of the hand and feet. The reflection pulleys (2) are the anatomic reinforcements of the above-mentioned fibrous sheaths located in places where there are curves along the course of the tendon. Their task is to keep the tendon inside its sliding bed.

The synovial sheaths (3) are access tunnels for tendons at bone surfaces or other anatomic structures that might cause friction. Most frequently, they can be found around the tendons of the hand and feet.
Under a fibrous layer, there are two thin and serous sheets, the parietal and visceral sheets. These sheets form a closed duct including peritendinous fluid for lubrication. In some tendons, i.e., tendons which do not have a true synovial sheath, there can be a peritendinous sheet (4) (paratenon) to reduce friction. It is composed of loose fibrillar tissue and functions as an elastic sleeve permitting free movement of the tendon against surrounding tissues. Characteristically, the Achilles tendon has a well identifiable paratenon with thin gliding membranes (Jozsa & Kannus, 1997).

The tendon bursae (5) are the fifth extratendinous structure playing an important role in the reduction of friction. They are located in those anatomic sites where a bony prominence might otherwise compress and wear the gliding tendon. Typical examples are the subacromial, deep trochanteric, pes anserinus, infrapatellar, and retrocalcaneal bursae.

Paratenon, epitenon and endotenon

Many of the tendons are surrounded by loose areolar connective tissue called paratenon. Only some parts of a few tendons in the hands and feet are actually provided with the above-described two-layer tendon sheaths, i.e., true tendon sheaths can only be found in the areas where a change of direction and increase in friction necessitate very efficient lubrication.

The collagenous fiber system of the paratenon is well defined. The main components of the paratenon are the type I and type III collagen fibrils and the elastic fibrils (Kvist, Jozsa, Järvinen, Kvist, 1985), and the paratenon is lined on its inner surface by synovial cells (Jozsa & Balint, 1978; Williams, 1986).

Paratenon functions as an elastic sleeve (although probably not so effectively as a true tendon sheath), permitting free movement of the tendon against the surrounding tissues (Hess, Cappiello, Poole, Hunter, 1989).

Under the paratenon, the entire tendon is surrounded by a fine connective tissue sheath called epitenon (Fig. 1). On its outer surface, the epitenon is contiguous with paratenon and on its inner surface with the endotenon. Inside the tendon, the endotenon invests each tendon fiber and binds individual fibers, and in larger units fiber bundles, together (Elliott, 1965; Jozsa, Kannus, Balint, Reffy, 1991a) (Fig. 1).

The epitenon is a relatively dense fibrillar network of collagen with strands of 8–10 nm in thickness. This network contains longitudinal, oblique, as well as transversal fibrils. It shows little variation in density or fibril orientation. Occasionally, the fibrils of epitenon are fused with the superficially located tendon fibrils (Jozsa et al., 1991a) (Fig. 2).

The endotenon is a thin reticular network of connective tissue inside the tendon that has a well-developed crisscross pattern of collagen fibrils (Elliott, 1965; Kastelic, Galeski & Baer, 1978; Rowe, 1985). The endotenon fibrils invest tendon fibers and bind fibers together (Fig. 1).

To improve binding, there is a high degree of hydration of proteoglycan components between the endotenon and the surface of the tendon fascicles (Rowe, 1985). Along with its important function of binding, the endotenon network allows the fiber groups to glide on each other and carries blood vessels, nerves, and lymphatics to the deeper portion of the tendon (Elliott, 1965; Hess et al., 1989).

Collagen fiber orientation in tendons

The divergent spiral arrangement of fibers of the flexor digitorum superficialis tendon to encircle the tendon of the flexor digitorum profundus was already illustrated by Leonardo da Vinci (1452–1519) in his earliest studies of the mechanism of the hand in the 15th century. With the current use of transmission and scanning electron microscopes, it is well documented that the collagen fibrils are oriented not only
The types of collagen fiber crossing in tendons: A=parallel running fibers; B=simply crossing fibers; C=crossing of two fibers with one straight-running fiber; D=a plait formation with three fibers; E=up-tying of parallel running fibers.

longitudinally but also transversely and horizontally, with the longitudinal fibrils also crossing each other, thus forming spirals and plaits (Chansky & Iannotti, 1991; Jozsa et al., 1991a) (Fig. 3). This complex ultra-structure of tendons provides good buffer capacity against longitudinal, transversal, horizontal as well as rotational forces during movement and activity.

There is a great tendon-to-tendon variation, and within a tendon site-to-site variation, in collagen content and type distribution (Fan, Sarkar, Franks, Uhthoff, 1997). The frequently observed twists and intertextures of the tendon fibers are likely to be related to optimal transmission of the forces of the muscle contractions by increasing the tensile strength of the tendon in general, as well as in certain anatomical points.

Internal architecture of tendons

Tendons consist of collagen (mostly type I collagen) and elastin embedded in a proteoglycan-water matrix with collagen accounting for 65–80% and elastin approximately 1–2% of the dry mass of the tendon (Curwin, 1997; Hess et al., 1989; Jozsa, Lehto, Kvist, Balint, Reflý, 1989; Kirkendall & Garrett, 1997; O’Brien, 1997; Tipton, Matthes, Maynard, Carey, 1975a). These elements are produced by tenoblasts and tenocytes, which are the elongated fibroblasts and fibrocytes that lie between the collagen fibers, and are organized in a complex hierarchical scheme to form the tendon proper (Hess et al., 1989). Soluble tropocollagen molecules form cross-links to create insoluble collagen molecules, which then aggregate progressively into microfibrils and then into electron-microscopically clearly visible units, the collagen fibrils (Figs. 1 and 4).

Collagen fibers, fiber bundles, and fascicles

A bunch of collagen fibrils forms a collagen fiber, which is the basic unit of a tendon (Fig. 1). A collagen fiber is the smallest tendon unit visible using light microscopy and is aligned from end to end in a tendon (Curwin, 1997). A fiber also represents the smallest collagenous structure that can be tested mechanically, although a larger fiber bundle makes the testing more reliable (O’Brien, 1997).

In this context it should be remembered that there is no standard nomenclature for aggregations of collagen fibrils within the tendon, perhaps because of their great variability (Curwin, 1997; Elliott, 1965). Therefore, the classification presented here is not the only one possible but represents the system the author prefers.

A fine sheath of connective tissue called endotenon invests each collagen fiber and binds fibers together (Fig. 1). A bunch of collagen fibers forms a primary fiber bundle (subfascicle), and a group of primary fiber bundles forms a secondary fiber bundle (fascicle). A group of secondary fascicles, in turn, forms a tertiary bundle, and the tertiary bundles make up the tendon surrounded by the epitenon (Fig. 1).

In human tendons, the diameter of the tertiary bundles varies from 1000 µm to 3000 µm. The diameter of the secondary bundles ranges from 150 µm to 1000 µm. The diameter of both types of bundles is directly related to the macroscopic size of the tendon so that the lowest values are seen in the small tendons, such as the flexors and extensors of the fingers and toes, and the largest diameters in the big tendons, such as the Achilles, tibialis anterior, and extensor hallucis longus tendons (Jozsa & Kannus, 1997). In transverse sections, the profile of the primary fiber
bundles or subfascicles (15–400 μm in diameter) is usually triangular with relatively sharp corners. This shape is influenced, but not completely determined, by compression from the surrounding structures or by the presence of neighbouring fascicles.

One fascicle usually has three or four subfascicles, although Kastelic et al. (1978) reported that tendon fascicles may have up to 10–12 subfascicles. According to our experience, the number of subfascicles varies from tendon to tendon, and occasionally even within the same tendon (Jozsa & Kannus, 1997). The same concerns the diameters of the fascicles and subfascicles. Both the fascicles and tertiary tendon bundles frequently show spiral formation along the course of the tendon.

Either within one fascicle or between different fascicles crimping or wavy formation of the collagen fibers is a characteristic phenomenon. Along the course of individual fibers, or between the superficial and midsubstance fibers of a fascicle, crimping is, however, a rather varying, irregular phenomenon, the crimp angle varying between 0° and 60° (Jozsa & Kannus, 1997; Rowe, 1985). This may be due to varying contribution of the proteoglycan cross-linking to the crimp. The direction of the fiber crimping or the phase of the wave pattern may be completely reversed between the front and back surfaces of the fascicles and subfascicles. Rotation of the subfascicles demonstrates the diversity of the crimp patterns that may occur within just a short length of the tendon (Jozsa & Kannus, 1997).

Collagen fibers. The number of collagen fibers in each primary bundle (subfascicle) may vary considerably from tendon to tendon. The collagen fiber diameter also shows variation, the diameter ranging from 5 μm to 30 μm in the rat tail tendon (Angel & Georghi, 1985). In human tendons, the fiber diameter can be as high as 300 μm (Elliott, 1965). Collagen concentration in each fiber is directly related to the fiber diameter, which in turn depends on the number rather than size of its constituent fibrils (Elliott, 1965).

In the resting state, the collagen fibers and fibrils of a tendon show a wavy configuration which appears already under the light microscope, but especially under the scanning electron microscope, as regular bands across the fiber surface. The fiber orientation is well definable (Rowe, 1985). This configuration disappears if the tendon is stretched slightly corresponding to a straightening of the collagen fibers (Elliott, 1965; Hess et al., 1989). When the tensile force is released, the tendon resumes its normal wavy appearance. Below about 4% elongation, the stress–strain curve of a tendon is reproducible by a sequence of stretches, but as soon as this limit is exceeded the wavy form will not reappear and subsequent deformations will not reproduce the original curve. If an acute stress causes an elongation of 8% or more, the tendon is likely to rupture (Jozsa & Kannus, 1997).

All the above-noted information on tendon elongation has been obtained from in vitro laboratory experiments. Information on tendon elongation during daily or sports activities has been difficult to obtain since in vivo a muscle and its tendon does not act separately but as a unit. However, a current view is that during normal movements a tendon elongation does not exceed the above-noted 4% limit (Jozsa & Kannus, 1997).

The running of collagen fibers along the course of the tendon is not only parallel. Under a polarized light microscope, four types of fiber crossing can be demonstrated (Jozsa, Reffy, Balint, 1984): simply crossing two fibers, crossing of two fibers with one straight-running fiber, a plait formation with three fibers, and up-tying of parallel running fibers with one fiber (Fig. 3). Along the whole length of tendons, the ratio of longitudinally to transversely (or horizontally) running fibers ranges between 10:1 and 26:1 (Jozsa et al., 1991a). In addition, all these fibers form spirals (Jozsa et al., 1991a; Kastelic et al., 1978; Rowe, 1985).

Collagen fibrils. A collagen fiber consists of a variable number of fibrils. The diameter of fibrils varies from 20 nm to 150 nm (Dyer & Enna, 1976; Jozsa et al., 1984). In the human Achilles tendon, the fibrils are between 30 nm and 130 nm in diameter (most of them between 50 nm and 90 nm), while in the flexors and extensors of the fingers and toes, the diameter is 20–60 nm. We observed that the fibril thickness of healthy human Achilles and biceps brachii tendons was between 30 nm and 80 nm (Fig. 1) (Jozsa et al., 1984).

Quantitative morphometric analyses have confirmed that the collagen fibril diameter increases from birth to maturity in animals and that the mean diameter of fibrils is indeed different in different tendons (Moore & De Beaux, 1987). Within one tendon, the proximal and distal parts, or the central and peripheral regions, do not show systematic differences in fibril thickness as evidenced by electronmicroscopy (Dyer & Enna, 1976).

The three-dimensional ultrastructure of a tendon fiber is complex. Within one collagen fiber, the fibrils are oriented not only longitudinally but also transversely and horizontally. The longitudinal fibers do not run only parallel but also cross each other, forming spirals. Some of the individual fibrils and fibril groups form spiral-type plaits as demonstrated by an electron microscope (Jozsa et al., 1991a). On the surface of fibrils, a sequence of elevated and depressed segments with diverse molecular density can be observed (Marchini, Morocutti, Castellani, Leonardi, Ruggeri, 1983).
Importance of the complex three-dimensional structure

The basic function of the tendon is to transmit the force created by the muscle to the bone, and, in this way, make joint movement possible. The complex macro- and microstructure of tendons and tendon fibers makes this possible. During various phases of movements, the tendons are exposed not only to longitudinal but also to transversal and rotational forces. In addition, they must be prepared to withstand direct contusions and pressures. The above-described three-dimensional internal structure of the fibers forms a buffer medium against forces of various directions, thus preventing damage and disconnection of the fibers (Jozsa & Kannus, 1997).

Extracellular tendon matrix

The extracellular tendon matrix is composed of the collagen fibers (see above), elastic fibers, the ground substance, and the anorganic components.

Elastic fibers

Elastic fibers account only for approximately 1–2% of the dry mass of a tendon, whereas in the aorta elastic fibers make up 30–60% of dry weight (Kirke-dall & Garrett, 1997).

The mechanical stability of the tendinous collagen is the most important factor for the mechanical strength of a tendon. The function of elastic fibers is not entirely clear, but they may contribute to the recovery of the wavy configuration of the collagen fibers after tendinous stretch (Butler, Grood, Noyes, Zernicke, 1978).

Elastic fibers are scarcely present in human tendons (Carlstedt, 1987). We found that elastic fibers were actually demonstrable in only 10% of healthy human tendons (Jozsa & Balint, 1978). In contrast, Ippolito et al. (1980) observed elastic fibers in the Achilles tendons of young and old rabbits, and Greenlee & Pike (1971) in the tendons of rats. In some pathological conditions of humans, such as Ehlers-Danlos syndrome and chronic uremia, and Greenlee & Pike (1971) in the tendons of rats. In some pathological conditions of humans, such as Ehlers-Danlos syndrome and chronic uremia, and especially the insertion area include up to 3.5–6.0% GAG, of which 65% is chondroitin sulphate (Karpakka, 1991). The water-binding capacity of these macromolecules (proteoglycans and GAGs) is considerable, improving the biomechanical properties (elasticity) of a tendon against shear and compressive forces. They are also important for stabilization of the whole collagenous system of connective tissue and for maintenance of ionic homeostasis and collagen fibrillogenesis.

Proteoglycans. Proteoglycans (PGs) are composed of a protein core in which one or more GAGs are covalently attached. They are large (molecular weight 10^6 Da), negatively charged hydrophilic molecules that can entrain water 50 times their weight. They are mostly entrapped within and between collagen fibrils and fibers (Karpakka, 1991). By virtue of their high fixed charge density and charge-to-charge repulsion force, PGs are stiffly extended, providing the collagen fibrils with a high capacity to resist high compressive and tensile forces. The mechanism is accentuated by the fact that these molecules are compressed about 20% of their natural solution domain during stress. The proteoglycans also enable rapid diffusion of water soluble molecules and migration of cells. In addition, the presence of the negatively charged groups attracts many positive counterions in this aqueous surrounding and thus creates Donnan’s osmotic pressure (Jozsa, Kvist, Kannus, Vieno, Järvinen, Lehto, 1991b).

The concentration of GAGs is considerably smaller in tendon than in cartilage or other type of connective tissue. The tensional zone of a tendon includes approximately 0.2% GAG (dry mass) of which 60% is dermatan sulphate, while the pressure zone and especially the insertion area include up to 3.5–5.0% GAG, of which 65% is chondroitin sulphate (Merrilees & Flint, 1980). Hyaluronic acid constitutes about 6% of the total GAG. Heparan sulphate can be found at the myotendinous junction (Järvinen, Kannus, Kvist, Isola, Lehto, Jozsa, 1991; Kvist et al., 1991). Of the remaining sulphated GAGs, neither keratan sulphate nor heparin can be demonstrated in tendon tissue (Jozsa et al., 1991b).

Glycoproteins. The glycoproteins are macromolecules that consist of a large protein fraction and a small glycidic component. They have a relatively low molecular weight, 50–100 kDa. Compared with proteoglycans, the glycoproteins have quantitative
differences in the protein-carbohydrate ratio, and qualitative differences in the composition of glycidic radicals.

Tendon tissue contains various non-collagenous proteins whose identities and functions are not yet well established (Kannus et al., 1998). Among them, the so-called adhesive glycoproteins form an interesting subgroup. These macromolecules seem to have a property to bind either other macromolecules or cell surfaces together (Kannus et al., 1998). At least four such molecules have been identified from the tendon belly: fibronectin, thrombospondin, tenascin-C, and undulin (Miller & McDevitt, 1988; Jozsa et al., 1991b; Kannus et al., 1998). Laminin can be found in the vascular walls of the tendons (Jozsa et al., 1991b) and in great amounts at the myotendinous junction (Järvinen et al., 1991; Kvist et al., 1991).

Anorganic components

Anorganic components form less than 0.2% of the tendon dry mass. In tendons, a wide variety of anorganic components have been detected (Lappalainen, Knuottila, Lammi, Alhava, Olkkonen, 1982). Calcium is found in the highest concentrations, the concentration being 0.001–0.01% of tendon dry weight in the tensitional area of a normal tendon, and 0.05–0.1% in the insertion. In pathological conditions such as calcifying tendinopathy, a 10- to 20-fold increase can be detected (Jozsa et al., 1989). Other detected components have been magnesium, manganese, cadmium, cobalt, copper, zinc, nickel, lithium, lead, fluoride, phosphor, and silicon. The trace elements are usually in a concentration of 0.02–120 ppm in tendon tissue (Spadaro, Becker, Bachman, 1970).

In general, the anorganic components are known to be intimately involved in growth, development, and normal metabolism of musculoskeletal structures (Schor et al., 1973). For example, copper has an important role in the formation of collagen cross-linking, manganese is required for several enzymatic reactions during the synthesis of connective tissue molecules, and calcium has a key role in the development of the osteotendinous junction (Minor, 1980).

Concerning tendon tissue specifically, the biochemical and functional role of many of these trace elements is largely unknown.

Tendon cells

The tendon cells, the tenoblasts and tenocytes, comprise about 90–95% of the cellular elements of the tendon. The other 5–10% includes the chondrocytes at the pressure and insertion sites, the synovial cells of the tendon sheath on the tendon surface, and the vascular cells, such as capillary endothelial cells and smooth muscle cells of the arterioles, in the endo- and epitenon. In pathological conditions, many other types of cells, such as inflammatory cells, macrophages, myofibroblasts, can be observed in the tendon tissue (Jozsa & Kannus, 1997).

Tendon cell morphology

The newborn tendon has a very high cell-to-matrix ratio. The cells (tenoblasts) are arranged in long, parallel chains (Ippolito et al., 1980). They have different shapes and sizes. Some are elongated, others rounded, and still others polygonal. In young individuals, a gradual decrease in the cell-to-matrix ratio occurs and the tenoblasts start to resemble each other, being spindle shaped. In adults, the cell-to-matrix ratio further decreases and the cells (now called tenocytes) are very elongated (Ippolito et al., 1980; Jozsa & Kannus, 1997).

The size of tenoblasts varies, the length from 20 μm to 70 μm and the width from 8 μm to 20 μm. The shape of the nuclei varies from ovoid to very long spindle-shaped nuclei. The rough endoplasmic reticulum and the Golgi apparatus are well developed, but few mitochondria are seen in the cytoplasm (Ippolito et al., 1980; Moore & De Beaux, 1987).

In tenoblasts, pinocytotic vesicles and few lysosomes can be observed in the peripheral parts of the cytoplasm. Numerous long and slender cytoplasmic processes extend into the matrix. They contain a remarkable number of cytoplasmic organs and establish the intercellular contacts which can be desmosomal junctions, tight junctions, or gap junction (Jozsa & Kannus, 1997).

The immature elastic fibers with well developed amorphous central cores, when present, seem to be in close contact with the tenoblast plasma membrane. Around the tenoblasts, granular (amorphic) electron-dense material can be also found bearing resemblance to matrix proteoglycans.

All these morphological features of young tenoblasts well support the concept of the high metabolic activity of these cells; i.e., intense synthesis of the matrix components.

As the cell-to-matrix ratio gradually decreases with aging, many other changes occur inside the tendon cells, too. The tenoblasts transform to tenocytes (and occasionally vice versa) and become very elongated, being 80–300 μm in diameter. The nucleus-to-cytoplasm ratio increases, and the cellular processes (2–10 μm in diameter) become longer and thinner, extending far from the main body of the cell. In transverse sections, the tenocytes look, therefore, like spiders. The long cell processes are needed to keep close contact between the cells and the matrix components; that is, to compensate for the decreasing number of cells and increasing amount of tendon matrix.
during aging (Jozsa & Kannus, 1997; Moore & De Beaux, 1987).

The nucleus of an tenocyte is elongated, occupying almost entirely the length of the cell. The nuclear chromatin condensates to the margins of the nuclear membrane. The actin and myosin intracytoplasmic filaments and the pinocytotic vesicles can still be detected in tenocytes, although these elements are more characteristic of tenoblasts (Jozsa et al., 1979; Ippolito et al., 1980).

Ultratructural analysis confirms the impression that tenocytes are metabolically active cells although not at the same level as the tenoblasts. Rough endoplasmic reticulum and Golgi apparatus are still well developed. The cytoplasm has high quantities of free ribosomes. Mitochondria are few in number but have well-defined cristae. Lysosomes can be identified in varying numbers (Jozsa & Kannus, 1997).

Tendon cell metabolism

Today it is well known that tendon cells are active in both energy production and biosynthesis of collagen and other matrix components (Jozsa & Kannus, 1997; O’Brien, 1997).

Animal tendon cells have the enzyme chains for all the three main pathways of energy metabolism: the aerobic Krebs’ cycle, the anaerobic glycolysis, and the pentose phosphate shunt (Jozsa et al., 1979; Tipton et al., 1975a, 1975b). We have shown that the three major pathways of energy metabolism also exist in the tenocytes and peritendinous cells of a human tendon (Jozsa et al., 1979; Kvist, Jozsa, Järvinen, Kvist, 1987).

During the highest growth rate of a young tendon, all the three pathways of energy production are highly active. With increasing age, activity of the Krebs’ cycle and the pentose phosphate shunt decreases while the anaerobic glycolysis remains more or less constant. In other words, the metabolic pathways utilized for production of energy change from aerobic to more anaerobic (Hess et al., 1989; Kannus & Jozsa, 1991).

In matrix metabolism, the tendon cells are capable of synthesizing all components of the tendon matrix; that is, the collagen and elastic fibers, proteoglycans, and structural glycoproteins (Curwin, 1997). In general, the synthetic activity is high during growth and diminishes with age. However, the activity pattern may change drastically in many pathological conditions.

The studies of tendon matrix metabolism have been almost exclusively concerned with collagen metabolism; that is, the literature provides little information about the turnover of the other components of tendon matrix. Concerning collagen metabolism in tendons, current knowledge allows three conclusions (Laitinen, 1967; Jozsa & Kannus, 1997). First, in neonate tendon the collagen synthesis rate is relatively high but reduces drastically (more than in many other connective tissues) with age. Second, collagen turnover, including synthesis as well as catabolism, of an adult tendon is fairly low, comparable to ligamentous tissue. Finally, the metabolically most active collagen is the most newly synthetized, soluble collagen.

The low metabolic rate of tendon tissue is well suited for the main purpose of the tendon: the low metabolic rate with well-developed anaerobic energy production is essential if the tendon is to carry loads and remain in tension for periods of time without the risk of ischemia and necrosis. In other words, the tendon tolerates low oxygen tension well without injury. However, a likely drawback of this low metabolic rate is the slow rate of recovery after activity and of healing after injury (Vailas, Tipton, Caughlin, 1978; Williams, 1986).

Synthesis of the proteoglycans and glycoproteins occurs in two places of a tendon cell. The protein component is synthesized at the rough endoplasmic reticulum but the glycidic part in the Golgi apparatus (Gallop, Blumenfield, Seifter, 1972). After the protein component has been formed in the rough endoplasmic reticulum, a series of several enzymes in the cisternae of the Golgi apparatus conjugate and sulphate the glycidic radicals (O’Brien, 1997). Once the synthesis is complete, the protein-polysaccharide complex is carried through the Golgi apparatus to the plasma membrane to be secreted out of the cell. As described above, the literature provides little information about the turnover rates of tendinous proteoglycans and glycoproteins in health or disease. In healthy connective tissue, the turnover for proteoglycans seems relatively rapid and varies between 2 and 10 days (O’Brien, 1997).

Compared with the knowledge on the biosynthesis of tendon components, even less is known about the anatomic sites and mechanisms of matrix catabolism of a tendon. According to studies from other connective tissue matrices, two ways of degradation seem possible: 1) the tenocytes produce lysosomal or cytoplasmic degradative enzymes and secrete them into extracellular space where degradation of the matrix components occurs, and 2) the degradation of tendon matrix occurs through direct cellular phagocytosis and pinocytosis as is the case with osteoclasts in bone (Jozsa & Kannus, 1997).

Perspectives

Collagenous connective tissue is an essential part of a healthy tendon and in athletic performances its undisturbed function is a prerequisite for smooth operation of the muscle–tendon units. Proper understand-
Tendon connective tissue structure

Key words: connective tissue; endotenon; epitenon; paratenon; tendon; tendon fibers.

References


