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IN VITRO FATIGUE OF HUMAN TENDONS

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Abstract—The purpose of this study was to determine the fatigue behaviour of human tendons *in vitro*. The testing was accomplished with the use of specially designed grips and the local measurement of tendon cross-sectional area. Ninety specimens prepared from Extensor digitorum longus (EDL) tendons of the foot were subjected to a cyclic square tension–tension stress waveform at physiological frequencies. The maximum tensile stress was normalised to values corresponding to prescribed levels between 10% and 90% of the calculated ultimate tensile strength (UTS) of 100 MPa. The minimum stress was set at 1% of the UTS. A replication of 10 specimens per stress level allowed the use of statistical models for the distribution of fatigue life.

Results followed a linear model, of form $S = 101.3 - 14.8 \log(N)$, relating the normalised stress to the median number of cycles to failure, therefore suggesting the absence of an endurance limit. The Weibull distribution was found to describe adequately the probability of failure at each stress level. A model which takes into account *in vivo* healing was proposed. This model was able to explain the presence of intact tendons throughout the lifetime of an individual. © 1997 Elsevier Science Ltd

Keywords: Mechanical properties; Human tendons; Fatigue; Probability of failure; Damage-repair model.

INTRODUCTION

Tendons may be considered to be unidirectional fibre-reinforced composites. The building block of the fibrous phase consists of tropocollagen molecules, which reinforce a matrix made up of a hydrated proteoglycan gel. Collagen constitutes, at least, 30% of the wet weight of tendons. Tendons act as linking elements connecting muscles to bones, thus transmitting muscular pulls and external loads.

In slow concentric activities, where the inertial effects may be neglected, the maximal load to which the tendon may be subjected is the muscle isometric tetanic contraction, approximately equal to 0.35 N per square millimetre of muscle belly (Guyton, 1986). As an example, the isometric stress in the patellar tendon can be estimated at 29 MPa, based on the cross-sectional areas of 125 and 10,300 mm^2 for the tendon and the quadriceps femoris muscle, respectively. However, most daily activities consist of quick eccentric movements, where inertial effects play an important role. These dynamic activities have been investigated either *in vitro* by means of kinematic models for the patellar tendon (Wahrenberg *et al.*, 1978) or *in vivo* by surgically implanting buckle strain gauge transducers on the Achilles tendon (Komi *et al.*, 1992). These studies reported tendon stresses in the range 42–110 MPa, the latter exceeding the established values of the ultimate tensile strength.

Normal healthy individuals are estimated to walk approximately 1–1.5 million strides per year (Wallbridge and Dowson, 1982; Weightman *et al.*, 1978). During locomotion, the musculoskeletal system is subjected to a continuous and constant external load, the body weight. Movement is achieved by muscular contraction and the consequent production of torque around the joints of the lower limbs. Evidently, any specific muscle–tendon unit produces a cyclic force with a constant maximal value, which is proportional to the external load. Due to muscle tone, a small amount of tension is always present ensuring that the muscle–tendon units are taut even when the muscle is relaxed. Moreover, any possible slackening due to creep of the tendon would be eliminated by a reduction in the length of the muscle. Therefore, the *in vivo* repetitive loading pattern of tendons in the lower limbs may be broadly classified as a tension–tension square wave, as observed in the *in vivo* tensile load pattern in the Achilles tendon during various form of locomotion (Komi *et al.*, 1992).

Tissue trauma or injury can result from two basic mechanisms. A single impact macro-trauma, such as a blow to a leg or a twisting injury of a joint, will injure bone, muscle, tendon, ligament, and even neurovascular elements. The other mechanism of injury is repetitive microtrauma caused by the repeated exposure of tissue to low magnitude force, which itself will not result in tissue injury (Micheli, 1986). The number of overuse injuries is estimated to be about 30–50% of all sports-related injuries in the U.S.A (Järvinen, 1992). Tendon injuries are common examples of such injuries as, during physical activity, much of the force is focused on the tendon component of the muscle–tendon unit.

The mechanical testing of soft tissues is fraught with practical difficulties involving gripping the specimen, the measurement of cross-sectional area and the degree of hydration during both specimen preparation and testing. These issues are particularly critical in long-term cyclic testing with only a few studies reporting the fatigue behaviour of soft collagenous tissues (Wang *et al.*, 1995; Weightman *et al.*, 1978). The more relevant former study

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investigated the fatigue behaviour of wallaby tail tendon under sinusoidal loading. Such tendons are ideal specimens for investigation as they present a large aspect ratio, with lengths often exceeding 400 mm. Results indicated a series of linear relationships between the logarithm of fatigue life and the peak tensile stress, at loading frequencies over the wide range of 1.1–50 Hz. This dependence on loading frequency may be a direct result of the use of sinusoidal loading, which will introduce a change in the loading rate of the specimen with frequency.

The current study aims to establish the *in vitro* fatigue life of a human tendon of the lower leg. The fatigue characterisation consisted of assessing the number of cycles to failure at prescribed stress levels, normalized by the ultimate tensile strength, as the independent variable.

MATERIALS AND METHODS

Specimen preparation and storage

Human Extensor digitorum longus (EDL) tendons were used in all experiments. These specific tendons, involved in joint movement at the foot and ankle in normal locomotion, exhibited both a large aspect ratio and a relatively uniform cross-sectional area. They had proved suitable specimens in a study establishing their viscoelastic properties under dynamic loading conditions (Schechtman and Bader, 1994). A total of 102 EDL specimens were employed from the lower limbs of 34 human donors, in the age range of 46–90 yr, mean age 71 yr. All tendons were obtained in routine post mortem examinations. Cases of chronic renal, metabolic or endocrine disease, articular or systemic disease were excluded from the study, as such conditions could lead to changes in the mechanical properties.

The specimens excised from the body were immediately cleaned from any surrounding tissues and wrapped in paper towels soaked in Ringer's solution (BDH Chemicals Ltd., U.K., cat. No. 330902Q) buffered with 100 ml l^{-1} of phosphate buffer (Sigma Chemical Co. Ltd., U.K., cat. No. 936-4). The wrapped specimen was then inserted into a self-sealing plastic bag and kept frozen at −20°C until the day of measurement of its physical dimensions followed by the subsequent mechanical test.

Thawing of the specimen was achieved by keeping the wrapped specimen inside the self-sealing plastic bag at room temperature for 1 h.

Measurement of physical dimensions

The total length of each thawed tendon specimen was measured with a vernier gauge to an accuracy of 0.5 mm. The cross-sectional area of the specimen was then measured using an area micrometer, which has been previously described (Schechtman and Bader, 1994). The area micrometer consisted of a slot with known dimensions and a plunger connected to a micrometer head. In the present investigation, the cross-sectional area of the specimens was measured locally, every 5 mm along its length, using the area micrometer with a slot 3 mm wide and 5 mm thick. The specimen was compressed by the plunger with a unique force, determined by the quoted spring stiffness in the ratchet mechanism. Assuming the force was uniformly distributed over the contact area of the plunger, namely 15 mm^2, the resulting pressure was 115 ± 18 MPa. This pressure, which was identical for all specimens, was of a similar magnitude to that previously reported (Butler *et al.*, 1984; Noyes, 1977). The accuracy and reproducibility of this method was of the order of 2% (Schechtman, 1995).

Clamping device

A set of specially designed self-tightening clamps was employed for gripping the specimens. These self-tightening clamps employed pairs of wedges with ridged gripping surfaces, which had been sand-blasted (Schechtman and Bader, 1994). A torque of 10 Nm applied to the locking screws was found adequate to grip the specimens without macroscopic damage during either of the testing procedures.

Quasi-static tensile test

A total of 12 Extensor digitorum longus (EDL) specimens were employed to establish the quasi-static tensile properties. The tendon specimens were tested on a screw-driven testing machine (Instron Ltd., U.K., model 6025). The load cell (2518-204/1 kN) was employed at the full range, with an accuracy of ±1 N. The crosshead displacement was accurate to ±0.01 mm.

The minimum cross-sectional area in the region between the clamps was determined from the cross-sectional area profile. The length of the specimen between the clamps was measured at the first non-zero load registered by the load cell. The crosshead speed was chosen to provide a strain rate of 1% s^{-1}.

The tests were conducted, with the specimens constantly kept moist by spraying with buffered Ringer's solution at room temperature, until gross macroscopic failure. The maximum load and extension to failure were recorded and values of ultimate tensile strength (UTS) and strain to failure were calculated.

Fatigue test

A total of 90 EDL specimens were employed for the characterisation of the fatigue life. The tendon specimens were tested on a hydraulic testing machine (MTS Systems Co., U.S.A., model Elastomer 830). The load cell (661.19 E − 02/10 kN) was employed in the range of 500 N, with a guaranteed accuracy of ±2.5 N. The displacement of the actuator was accurate to a value of ±0.25 mm.

The specimens were subjected to a square cyclic tension–tension load wave in the same physiological frequency range as employed in a previous study, namely 1– 4 Hz (Schechtman and Bader, 1994). This waveform approximates to the *in vivo* loading pattern in tendons (Komi *et al.*, 1992) and ensured that the loading rate was constant regardless of frequency. A typical example of the equivalent stress profile with time is illustrated in Fig. 1. The maximum stress was set at a value corresponding to one of nine prescribed stress levels in the range 10–90% of the UTS in 10% increments. The minimum stress was set at a value corresponding to 1% of the UTS. The frequency of the cyclic loading was varied according to

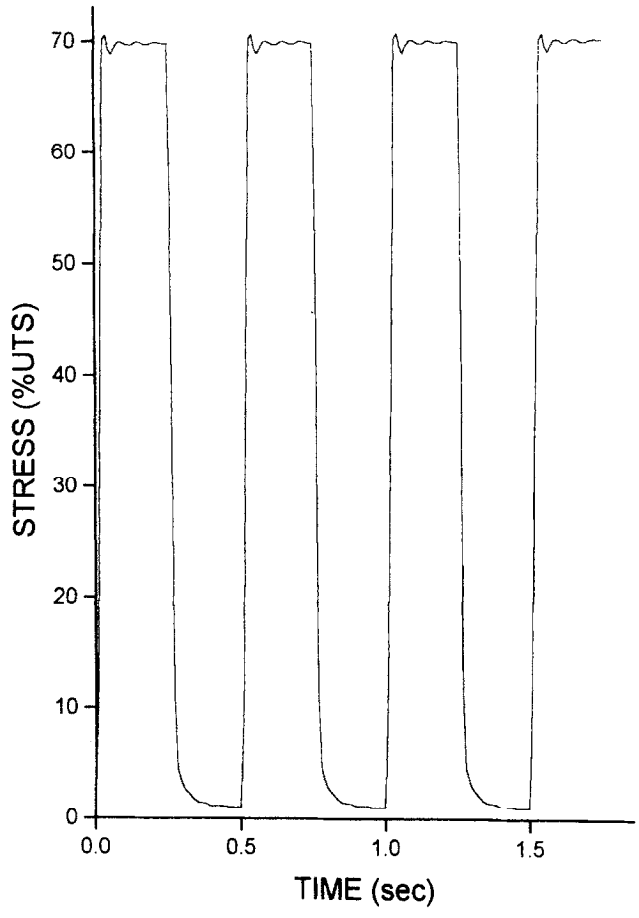

Fig. 1. Typical tensile stress profile applied to a tendon specimen. The specimen was subjected to a maximum tensile stress of 70 MPa at a frequency of 2 Hz.

the stress levels such that:

- at stress levels of 80 and 90% of the UTS the frequency was 1 Hz,
- at stress levels of 60 and 70% of the UTS the frequency was 2 Hz,
- at stress levels of 40 and 50% of the UTS the frequency was 3 Hz,
- at stress levels of 10, 20 and 30% of the UTS the frequency was 4 Hz.

Each specimen was randomly allocated into one of the 9 stress levels with a total of 10 specimens per level. The displacement of the actuator of the testing machine was recorded throughout the test and the corresponding strain to failure calculated, based on the initial clamp-to-clamp distance. The fatigue life for each tendon specimen, expressed as the number of cycles endured until macroscopic failure, was also recorded.

The specimens were kept moist throughout the experiment by the continuous dripping of buffered Ringer's solution, at room temperature, at a flow rate of approximately $0.5\,l\,h^{-1}$.

Data analysis

The statistical distribution of the fatigue life, at each stress level, was estimated by determining the probability of failure with the use of a standard table of median ranks (Johnson, 1964). Three statistical distributions, which are commonly employed to describe the fatigue lifetime in terms of the probability of failure, were evaluated (Hastings and Peacock, 1975; King and Evans, 1967; Krause et al., 1988; Tsai and Ansell, 1990) from the following formulations:

(i) The normal distribution, probability density function

$$f(x) = \frac{1}{\sigma(2\pi)^{1/2}} \exp\left[\frac{-(x-\mu)^2}{2\sigma^2}\right]. \quad (1)$$

(ii) The log-normal distribution, and probability density function

$$f(x) = \frac{1}{x\sigma(2\pi)^{1/2}} \exp\left\{\frac{-[\log(x/m)]^2}{2\sigma^2}\right\}, \quad (2)$$

where μ and σ are the mean and standard deviation of the population and m is the median value.

(iii) The Weibull distribution, probability density function

$$f(x) = \left\{\frac{cx^{c-1}}{b^c}\right\} \exp[-(x/b)^c], \quad (3)$$

where b and c are population parameters. This distribution uses a modified parameter, W, defined by

$$W = \log\left\{\frac{1}{1-P}\right\}, \quad (4)$$

where P is the probability of failure.

The strains to failure were compared at all stress levels using a one-way ANOVA. Also, the correlation coefficients of these models of the statistical distributions were examined for differences using a one-way ANOVA. $P < 0.05$ were considered to be statistically significant.

RESULTS

Quasi-static tensile test

A typical stress–strain curve for one such specimen is presented in Fig. 2. The resulting stress–strain relationship is non-linear in form, as previously reported for both animal and human tendons *in vitro* (Abrahams, 1967; Rigby *et al.*, 1959; Stouffer *et al.*, 1985; Viidik, 1972). The initial region of the curve, the characteristic toe-in region, is characterised by a large increase in strain with increasing stress. A linear region, with an approximately constant modulus of elasticity immediately follows the toe-in region. A third region, the failure region, is characterised by a clear maximum followed by a sharp decrease in the stress values. The ultimate tensile strength (UTS) for the 12 EDL tendon specimens ranged from 80.6 to 118.5 MPa, with a mean value of 99.9 ± 12.2 MPa. These UTS values were similar to those presented in previous studies (Benedict *et al.*, 1968; Butler *et al.*, 1984). The corresponding strain to failure, measured from the crosshead displacement, was found to be $15.3 \pm 2.6\%$.

Fatigue test

The 90 specimens presented a mean cross-sectional area of 2.91 ± 0.92 mm^2 and a mean grip-to-grip length of 72 ± 19 mm.

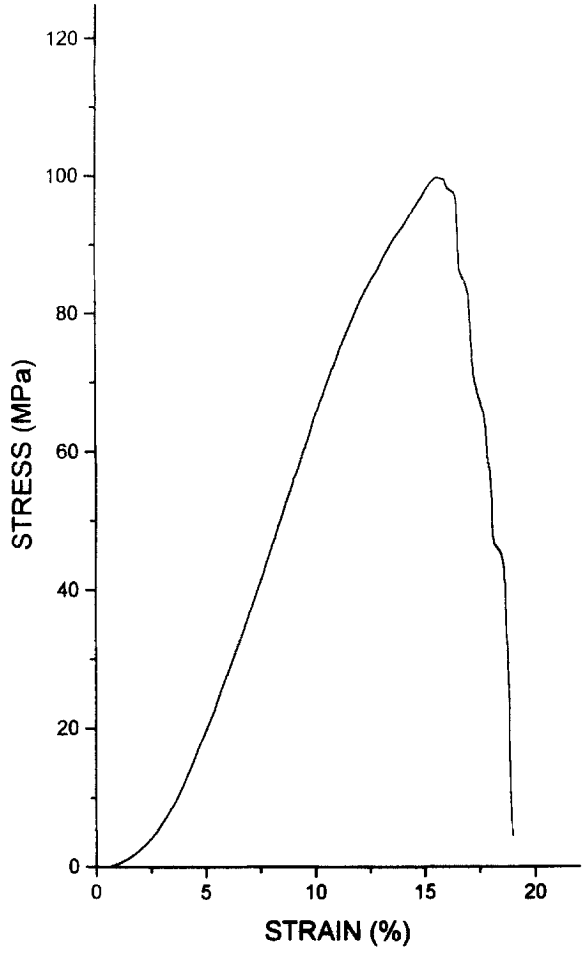

Fig. 2. Typical stress–strain behaviour of an extensor digitorum longus tendon in the quasi-static tensile test.

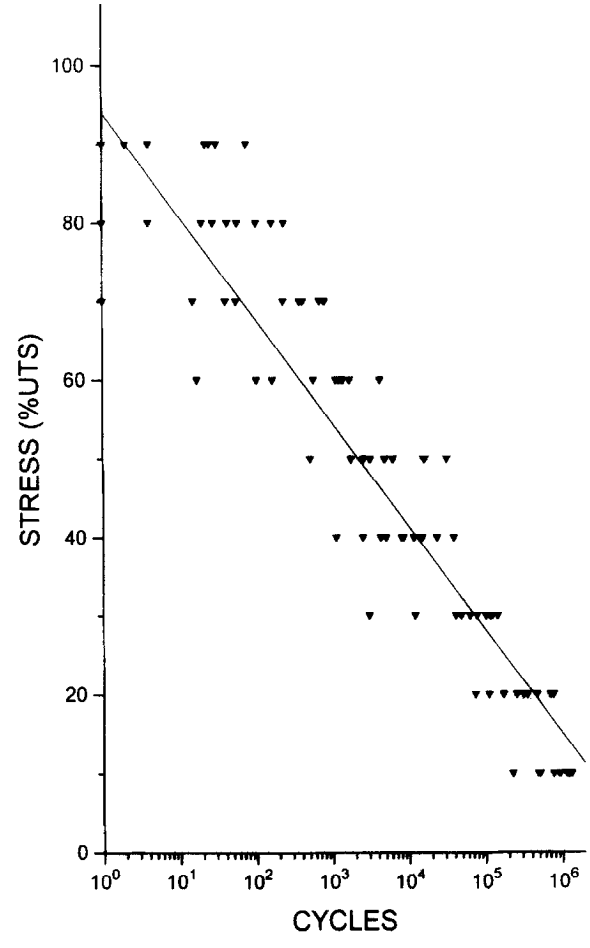

Fig. 3. Normalised stress values versus logarithm of number of cycles to macroscopic failure for 90 tendon specimens.

The mean value of the strain to failure at each stress level ranged from 12.3 to 16.6%. Statistical analysis indicated that the strain values were not significantly different from each other ($p = 0.50$). Thus, these values were pooled, to produce a mean strain value of $14.2 \pm 5.6\%$ for all 90 specimens subjected to fatigue failure.

The results for the fatigue life of all specimens are presented in graphical form in Fig. 3. The graph of normalised stress level against the logarithm of fatigue life, produced a highly significant linear model of the following form:

$$S = 93.98 - 13.13 \log(N), \quad r = -0.94, \quad P < 0.01, \tag{5}$$

where S is the normalised stress expressed as a % of the UTS and N is the number of cycles to failure.

Figure 3 also reveals considerable variation in fatigue life at each stress level. For example, at 40% of the UTS the fatigue life ranged from 1137 to 39,547 cycles. Linear models, relating the probability function for each of the three statistical distributions and the number of cycles to failure, at each stress level, produced highly significant correlation coefficients. Although statistical analysis indicated that these coefficients were not significantly different ($p = 0.07$), detailed analysis of the experimental data suggested that the log-normal and Weibull distributions provided a more accurate representation of the data than the normal distribution of the fatigue life. This is clearly evident in Fig. 4, which presents the three statistical distributions of the probability of failure at a stress level of 40% of the UTS.

Consequently, a linear model based on the median value, equivalent to a 50% probability of failure, or survival, was employed. The relationship of stress level against the median fatigue life produced a linear model of the following form:

$$S = 101.25 - 14.83 \log(N), \quad r = -0.99, \quad P < 0.01 \tag{6}$$

where S is the normalised stress expressed as a % of the UTS and N is the number of cycles to failure. This model predicted a static strength of 101.3 MPa, which was clearly within one standard deviation of the experimental data obtained in the quasi-static tensile tests (99.9 ± 12.2 MPa).

DISCUSSION

The present study is the first to report the *in vitro* fatigue behaviour of human tendons. The test protocol, which included the use of self-tightening clamps, enabled

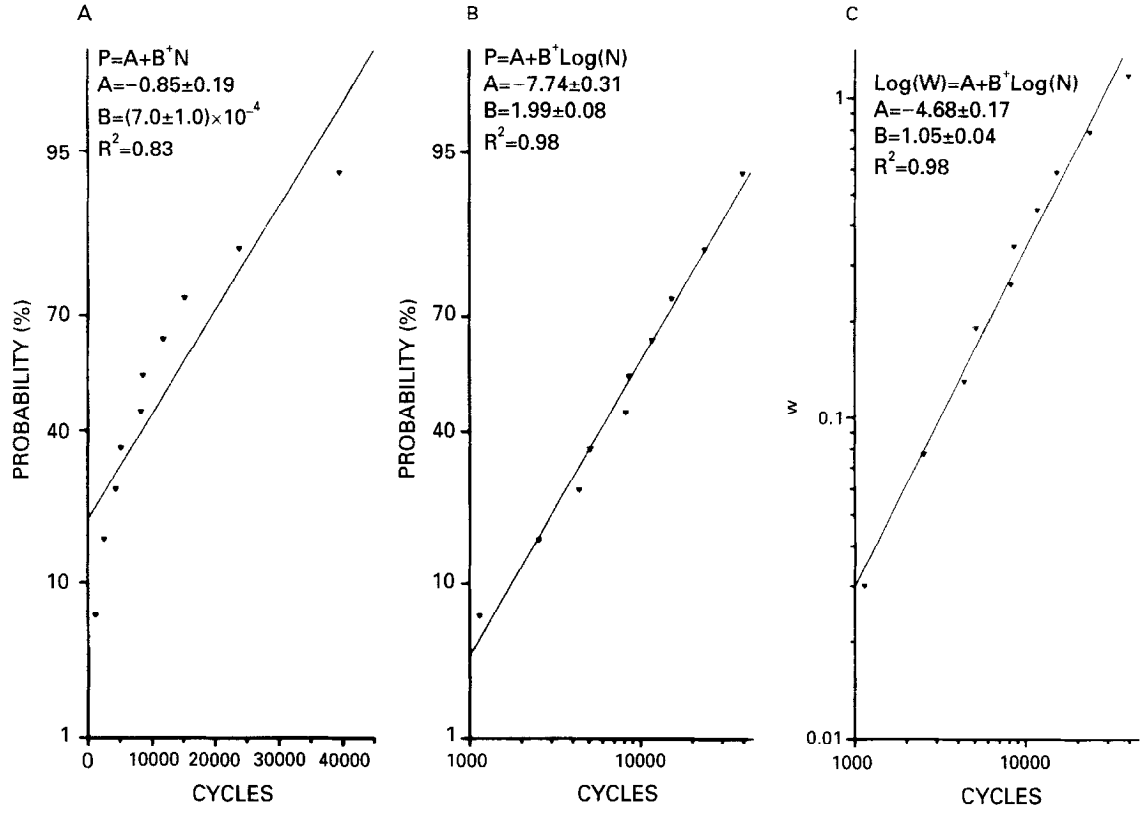

Fig. 4. Statistical distribution of the fatigue life at a stress level of 40% of the UTS. (A) Normal distribution; (B) log-normal distribution; and (C) Weibull distribution, where $W = \log(1/(1 - P))$ for a probability of failure P.

the long-term cyclic testing of tendons. The specially designed clamps gripped the specimens without macroscopic damage and slippage, as evidenced by the clear imprint of the ridged features of the wedges on the surface of the specimen. This justified the choice of the EDL tendons with a large aspect ratio. The fatigue experiment employed a test protocol which ensured that errors, associated with biological variability, were randomised and that significant replication allowed the use of statistical models of fatigue life.

The loading conditions were designed to simulate those reported *in vivo* for human tendons. The applied loading pattern consisted of a square cyclic tensile stress wave in the physiological range of 1–4 Hz. Use of a square wave enabled a variation in the frequency of the loading wave, which reduced the duration of the test, with no subsequent change in the loading rate on the tendon specimens. This was most convenient as tendons are known to exhibit viscoelastic behaviour, which is independent of frequency in the physiological range but sensitive to the rate of strain (Bennett *et al.*, 1986; Hubbard and Soutas-Little, 1984; Schechtman and Bader, 1994; Schwerdt *et al.*, 1980).

All specimens ruptured in the region between the clamps. The failure region was not well defined. However, the general appearance of this region resembled a frayed fabric, in which the failed end presents a gradual tapering to individual fibres. The similarity in failure strains obtained from both the fatigue and static tensile experiments suggested that the failure mechanism of tendons was determined by a limiting value of strain of approximately 15%. This process would involve a gradual recruitment of the tendon fibres followed by their extension until a significant number of the recruited fibres reached this limiting strain.

The present study can be compared with data for wallaby tail tendon (Wang *et al.*, 1995), where at a frequency of 1.1 Hz, the following linear model was estimated:

$$S = 96.5 - 15.8 \log(N), \tag{7}$$

where N is the number of cycles to failure and S is the stress normalised to a % of the UTS, which corresponded to 144 MPa for wallaby tail tendon. It is clear that this model is in good agreement with both linear models proposed in the present study. However, it is worthy of note that the model for animal tendon predicted a shorter fatigue life, as evidenced by the increased value of the gradient. The difference could be related to the distinct function of this wallaby tendon.

The probability of failure at each stress level was adequately described by both the log-normal and the Weibull statistical distributions. However, these distributions differ substantially in their hazard functions, i.e. the probability of failure as a function of time (Lawless, 1982). The log-normal predicts zero probability of failure at extremes of time, with the maximum probability occurring at some intermediate value of time. However, the Weibull distribution generally presents a hazard function which increases monotonically with time. Therefore, the

log-normal would predict an endurance limit, whilst the Weibull distribution implies the absence of such a limit. The present models do not predict an endurance limit, although it is appreciated that the models extrapolated results below the lowest stress level employed in the fatigue experiment, i.e. 10% of the UTS.

The fatigue life of tendons *in vivo* will depend upon the stresses to which they are subjected. Cyclic loading at the level of 40% of the UTS, corresponding to 40 MPa for the extensor digitorum longus tendon, would lead to failure after approximately 8500 cycles. At a stress level of 20% of the UTS, the fatigue life is of the order of 300,000 cycles, which is equivalent to a period of about four months of normal walking activity. However, these predictions based on the *in vitro* linear model for the 50% probability of failure clearly did not take into account the *in vivo* processes of healing and remodelling. Consequently, a modified approach is required to predict these combined processes *in vivo* to explain the presence of intact tendons throughout the lifetime of an individual.

A cumulative damage model for metallic structures was first proposed by Palmgren (1924) and validated experimentally by Miner (1945). Nash (1966) extended the cumulative damage model to self-healing living structures and presented it in the following form:

$$D(t) = D_S(t) + D_A(t) + D_D(t) - H(t) \tag{8}$$

where $D_S(t)$ is the damage associated with mechanical stressing which in fatigue is described by the Miner-Palmgren model. $D_A(t)$ is the damage associated with ageing, $D_D(t)$ is the damage associated with disease and $H(t)$ is the damage repaired by healing. The effects of these inter-related factors on the cumulative damage may be discussed qualitatively. Damage associated with ageing would depend upon the prior *in vivo* loading history; this was not accounted for in the present experiment which, for practical reasons, largely employed tendons specimens derived from relatively aged donors. In addition, both ageing and disease will inevitably produce structural changes which will affect the mechanical behaviour of tendon specimens and their repair potential.

The terms associated with ageing and disease can be neglected if the mechanical stressing upon a healthy structure spans over a short period of time, as was simulated in the present study. The self-healing model is then simplified to

$$D(t) = \sum_{i=1}^{n} \left\{ \frac{n_i}{N_i} \right\} - H(t). \tag{9}$$

$D(t)$ is the cumulative damage index, which ranges from 0 to 1, where 0 represents the undamaged state prior to mechanical fatigue and 1 represents the state of failure due to fatigue. The term $\sum_{i=1}^{n} \{n_i/N_i\}$ is the stress-related damage, where n_i and N_i are the number of cycles at a stress level of S_i and the number of cycles to failure at that same stress level, respectively.

A graphical depiction of this model involving stress-related damage and healing, as proposed in equation (9), is presentedin Fig. 5. This was based on the following assumptions:

(1) The number of load cycles was equivalent to the number of steps that a healthy individual walks per day,

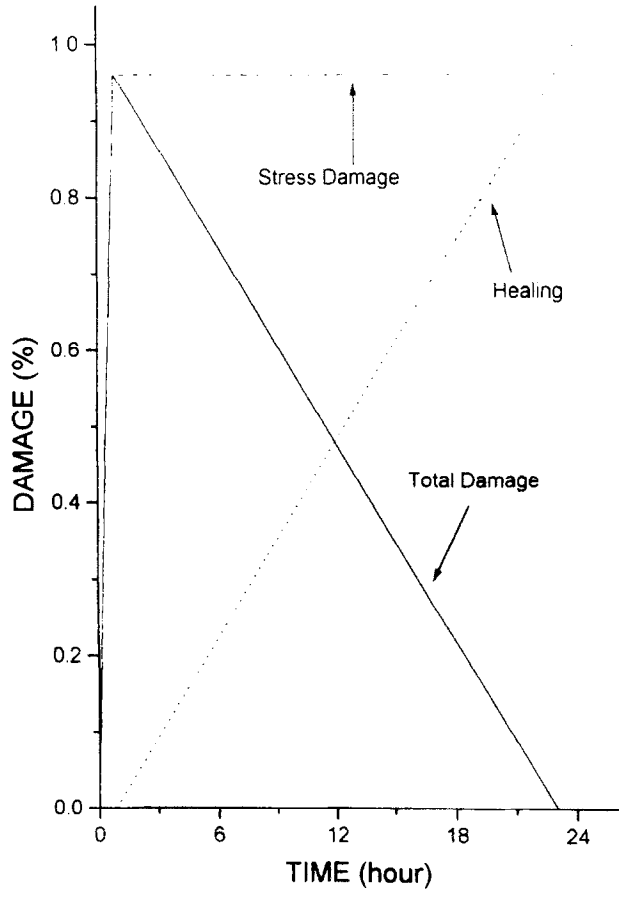

Fig. 5. *In vivo* model of stress related damage and associated healing for human tendons.

i.e. 1,000,000 steps per year divided by 365 days. This value of 3000 steps is approximately equivalent to a daily distance of 2 miles. These load cycles were assumed to be concentrated into a single period of activity during the day.

(2) It has been suggested that during normal activity a tendon *in vivo* is subjected to less than one-quarter of its ultimate stress (Carlstedt and Nordin, 1980), equivalent to 25 MPa. In addition, Ker and colleagues (1988) estimated from measured area ratios that human lower leg tendons would be subjected to stresses of between 11 and 25 MPa. Thus, in the present model a stress level of 20 MPa, equivalent to 20% of the UTS, was selected. At this level the fatigue life was 314,051 cycles. Therefore, assuming uniform damage per cycle and a cyclic activity of 1 Hz, then the rate of stress related damage would be 3.2×10^{-6} per second.

(3) A term associated with healing was based on the few studies (Hayashi, 1993; Noyes, 1977; Woo *et al.*, 1987), which have examined the rate of increase in strength for tendons/ligaments during remobilisation following a period of stress shielding. An increase in mechanical strength compared to control values suggested a healing rate equivalent to approximately 1% per day.

Thus, as inferred from Fig. 5, a healing rate of 1% per day would be sufficient to eliminate, in approximately

20 h, any fatigue damage on a tendon subjected to a stress of 20 MPa during daily normal locomotion. Therefore, the self-healing, or *in vivo*, model would predict that accumulated damage might occur if either the number of load cycles were increased at the same stress level or the stress level was increased. It is clear that the healing mechanism could be responsible for reducing the damage rate at high stress levels and establishing an endurance limit at low stress levels.

As an illustration, an individual who starts training for a sporting event might double the estimated daily mileage. In this case, after a month the accumulated damage would amount to 30% and overuse injuries might occur. Alternatively, this accumulated damage can be considered to be equal to 30% of the fatigue life, estimated at 94,215 cycles at 20% of the UTS. This value lies only above the lowest individual value recorded for the fatigue life at this stress level (Fig. 3). Therefore, a conservative estimate of probability of failure (Johnson, 1964) would lead to 6.7% of the population presenting with fatigue failure of tendons after this number of cycles.

The *in vitro* linear model for fatigue life of tendons, which has been established in the present study, may be utilised in two practical circumstances. It might be employed in the estimation of the stress level at physiological frequencies of repetitive load-bearing activities that an athlete could perform without developing overuse injuries. The model could also provide a design specification for synthetic materials, with no inherent repair potential, used for tendon replacement.

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