Viscoelastic properties of the human lumbodorsal fascia

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ABSTRACT

The purpose of this study is to provide better understanding of the mechanical response of the lumbodorsal fascia to dynamic and static traction loadings. Since the fascia shows a viscoelastic behaviour, tests in which time is a variable were used, namely hysteresis and stress relaxation. Load–strain and load–time curves obtained from the hysteresis and stress-relaxation tests point out three different phenomena. First, an increase in stiffness is noticed when ligaments are successively stretched, i.e. strains produced by successive and identical loads decrease. Second, if a sufficient resting period is allowed between loadings, stiffening is reversed and strains tend to recover initial values. The third phenomenon, observed in stress-relaxation tests as time progresses, is ligament contraction in stretched and isometrically held samples. This third phenomenon may be explained by the possibility that muscle fibres capable of contracting spontaneously could be present in lumbodorsal fascia ligaments.

Keywords: Lumbodorsal fascia, viscoelasticity, hysteresis, stress relaxation

INTRODUCTION

Lumbodorsal fascia is an extensive sheet of ligamentous tissue which encloses the paraspinal erectores spinae muscles of the lumbar spinae; it has been suggested that it plays an important role in the mechanics of the lumbar spine[1–3]. Recent immunohistochemical studies have supported the hypothesis that the lumbodorsal fascia may play a neurosensory role in the lumbar spine mechanism[4]. Lower back problems having become a major preoccupation for orthopaedists, it is important to understand the mechanics of the back's components, amongst them the lumbodorsal fascia.

Ligaments exhibit viscoelastic behaviour, i.e. their response to mechanical loading varies with time. Tests in which time is a variable are thus needed to evaluate their mechanical properties. Such tests include creep, stress relaxation and stress–strain experiments at constant rate of elongation. Cohen *et al.*[5] studied viscoelastic creep of collagenous tissue and noted that successive creep tests performed without sufficient recovery time for the sample provided different strains. Other studies report that ligament contraction, swelling or increase in viscosity take place as a result of modifications in environmental factors such as temperature[6,7], pH[8,9] and ionic content[10].

The purpose of this study was to evaluate viscoelastic behaviour of lumbodorsal fascia ligaments, particularly when stressed repetitively with intercalated rest periods. Stress-relaxation tests and hysteresis tests, which have been used extensively in our laboratory to study canine anterior cruciate ligaments[11] and human lumbar spine ligaments[12], were chosen for this purpose. We used the computer-controlled mechanical testing system for small biological samples designed and developed in our laboratory[13,14].

MATERIALS AND METHODS

Whole sheets of lumbodorsal fascia were excised from three cadavers of age 59, 69 and 73. The specimens were then stored at −70°C in order to avoid mechanical property deterioration. Lumbodorsal fascia shows obvious lamellations, each comprised of fibres oriented in a specific direction. After thawing, lamellation separation was required to test unidirectional tissue samples. The selected orientation for all test pieces was lengthwise along the spinal cord axis, which represented the main orientation of fibres near the midline.

For the first two sheets, the fascias were thawed, divided into layers of specific fibre orientation, cut into test pieces of dimensions averaging 1.5 mm × 1.0 mm × 30 mm, and stored in a balanced salt solution at 4°C. Because of the lengthy duration of each test, no more than two complete tests were conducted per day, which delayed the utilization of some samples; all tests were conducted less than two weeks after thaw. However, for the third sheet, the dissected samples of similar dimensions were stored

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in Ringer solution at 4°C and all tests were conducted less than six days after thaw.

Each sample was clamped at either end on the testing apparatus and kept submerged in saline solution throughout the series of experiments to avoid drying; all but two tests were carried out at room temperature. The initial length of each sample was defined as being the length of the tissue stretched at an initial load of 0.2 N. This pre-tension, though negligible, ensures that no compression is applied to the sample[14].

Preliminary series of load–strain tests were performed on five test pieces submerged in saline solution, to determine the ultimate non-damaging strain applicable on fascia ligaments. Load–strain curves suggested that some collagen fibres ruptured near 10% strain, while complete tissue rupture occurred, in every case, at less than 16% strain. This result is in agreement with the conclusion of our recent studies[15,16] where, using electron microscopy, we observed that deformations of 8% or more induced rupture in collagen fibres in rabbit medial collateral ligaments. Therefore 6% strain was chosen as an approprite strain level to conduct the following hysteresis and relaxation tests.

Fifteen test pieces were used to study the hysteresis behaviour of lumbodorsal fascia. Among those, 13 samples were tested in the following manner. A 6% deformation was applied on the ligament. The load corresponding to this strain was then identified as the specific amount of load to be applied on all subsequent stretches. The sample was taken back to its initial length, then re-stretched to the maximum load, and so on until five stretch cycles were completed. After that the sample was allowed to rest at its initial length for 30 min after which it was again cycled. Then followed a 1 h resting period and a third cycling. At the end of the three hysteresis experiments, a load–strain curve was produced by elongating the ligament until rupture. Testing procedure for the last two samples was identical except that loading was stopped when it produced a 6% strain on the ligament, instead of a specific load level.

Twenty-eight samples were used to perform relaxation tests. Six of these ligaments were subjected to the following procedure. A 6% strain was imposed on the sample, at which point the mobile clamp automatically stopped. The relaxation of the load as a function of time was then recorded for approximately 800 s. The sample was afterwards returned to its initial length and left to rest for 30 min. A second stretch and relaxation were performed, followed by a load–strain test until rupture. Five other ligaments were tested in the same fashion but with an added hour of rest and a third stretch and relaxation period. DMEM (Dulbecco's Modification of Eagle's Medium, a rich solution mimicking physiological milieu, isotonic with Ringer's solution and pH-buffered at 7.40) having been the solution used so far, five more samples submerged in Ringer solution were used to verify any solution dependency on the relaxation behaviour of fascia ligaments.

The last 12 samples used to study relaxation were subjected to various solution, temperature and humidity conditions. Stretching was performed until a predetermined load was achieved, identical for the three stretches of each ligament. Four ligaments were tested in DMEM solution at room temperature; two ligaments were tested under regular Ringer solution sprayings also at room temperature; two ligaments submerged in DMEM were tested at 4°C and 37°C respectively; and finally four ligaments were tested in Ringer solution at room temperature.

RESULTS

Figures 1 and *2* show typical load–strain curves obtained from the hysteresis tests. It is apparent from these figures that the second and third tests performed on ligaments each require greater loads than previously, i.e. the second test demands greater force from the apparatus to deform the sample to the same level than the first test and so on. This phenomenon was present in both types of hysteresis tests performed: with maximum load cutoff (*Figure 1*) and maximum strain cutoff (*Figure 2*). It is also apparent that the increase in load between tests is greater between the first and second tests than between the second and third.

Figure 3 shows a typical load–strain curve which followed the hysteresis experiment. These curves

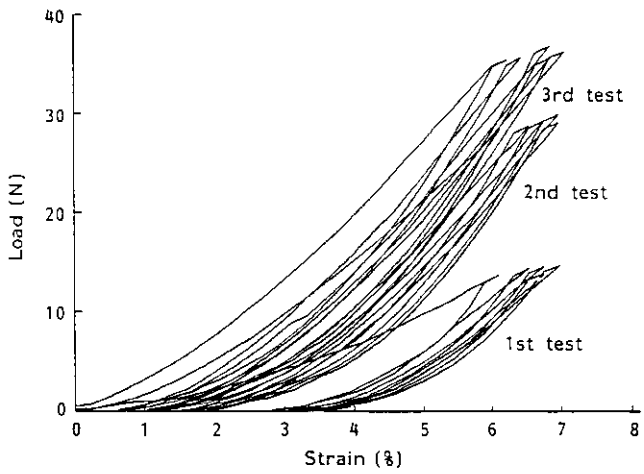

Figure 1 Hysteresis test using controlled load (first test, $F = 14$ N; second test, $F = 29$ N; third test, $F = 36$ N). Resting periods: 30 min between first and second tests; 1 h between second and third tests

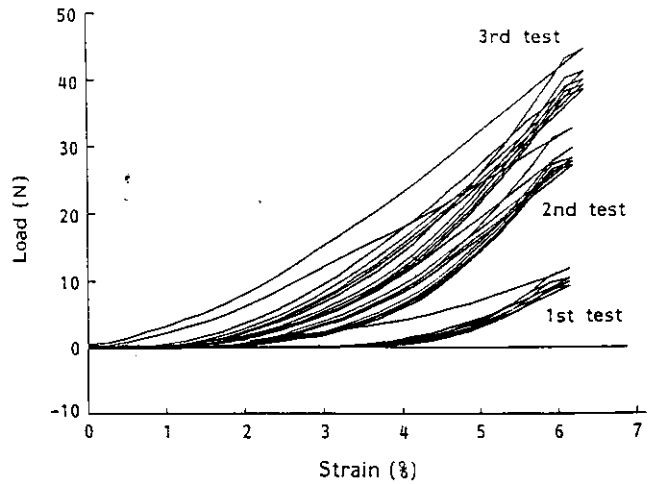

Figure 2 Hysteresis test using controlled strain (all tests, $\epsilon = 6\%$). Resting periods: 30 min between first and second tests; 1 h between second and third tests

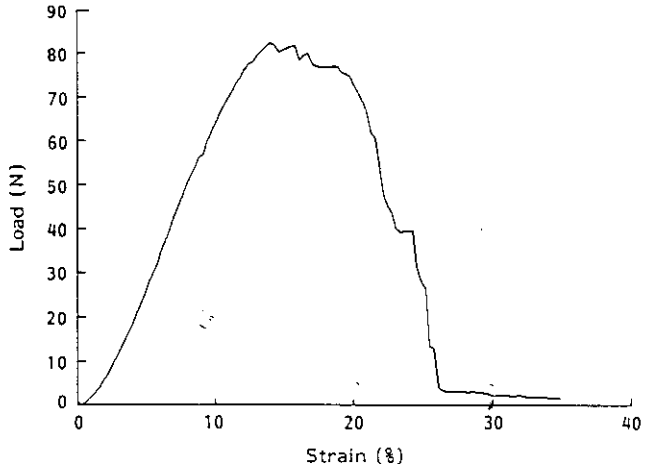

Figure 3 Typical load–strain curve; this particular curve was obtained following a hysteresis test

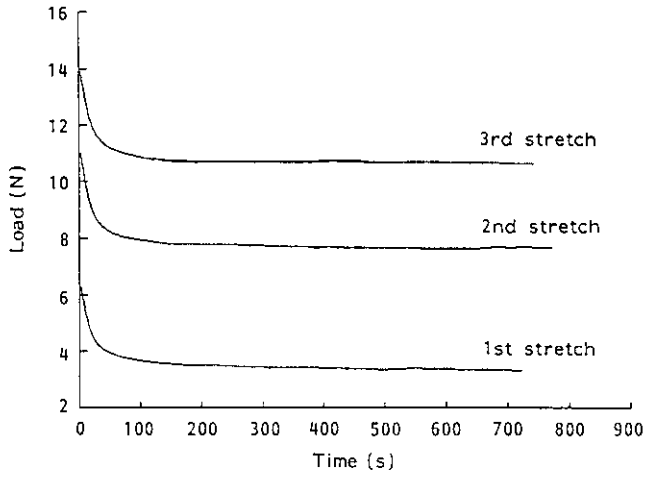

Figure 4 Load–time curves from a relaxation experiment using DMEM solution; the three curves were produced by applying 6% strain on the sample and recording the decreasing load. Resting periods: 30 min between first and second stretches; 1 h between second and third stretches

show strains at rupture varying between 12% and 15%.

Figure 4 shows the load–time curves produced by three stretches at 6% strain of typical relaxation tests using DMEM solution. The curves demonstrate the load's asymptotic approach to a constant residual load in the ligament. Again, we see the phenomenon encountered in the hysteresis tests: the second and third stretches exert increasing loads on the sample to deform it by 6%. *Figure 5* shows the load–time curves of a relaxation test conducted on a sample submerged in Ringer solution. The curve which represents relaxation following the first sample stretch resembles those obtained from the tests using DMEM solution. However the second and third curve show a slight ascension in the load, producing 6% strain as time progresses instead of stabilization to a residual value.

Figures 6–8 show load–time curves obtained from samples submitted to various environmental influences. All the tests were performed by stretches at a fixed load (instead of a fixed strain as with the previous samples). *Figure 6* illustrates the behaviour of relaxation curves for a test carried out at room

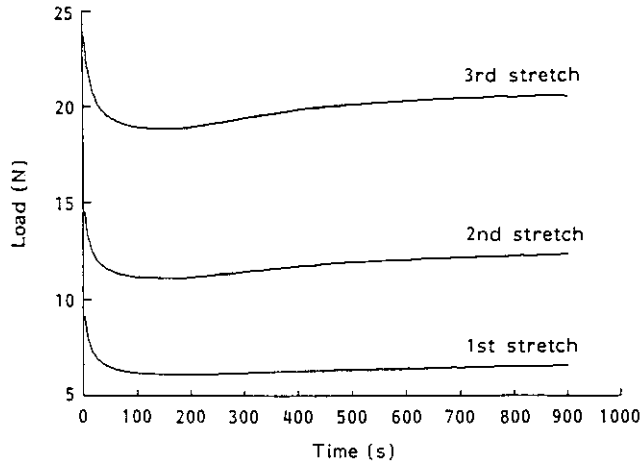

Figure 5 Load–time curves from a relaxation experiment using Ringer solution; the three curves were produced by applying 6% strain on the sample and recording the decreasing load. Resting periods: 30 min between first and second stretches; 1 h between second and third stretches

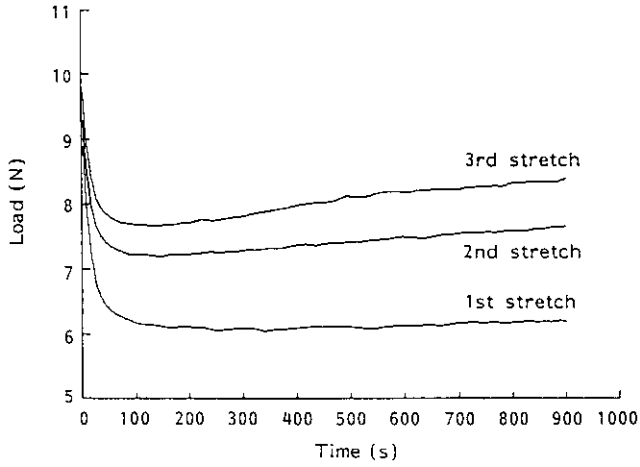

Figure 6 Load–time curves from a relaxation experiment using DMEM solution; the three curves were produced by applying 9 N on the sample and recording the decreasing load. Resting periods: 30 min between first and second stretches; 1 h between second and third stretches

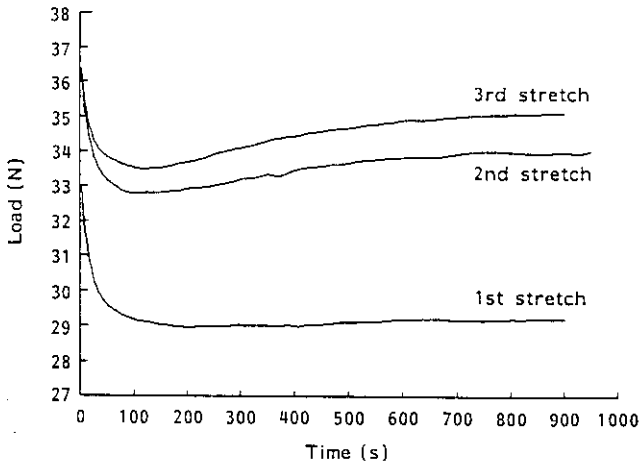

Figure 7 Load–time curves from a relaxation experiment without solution; the three curves were produced by applying 35 N on the sample and recording the decreasing load. Resting periods: 30 min between first and second stretches; 1 h between second and third stretches

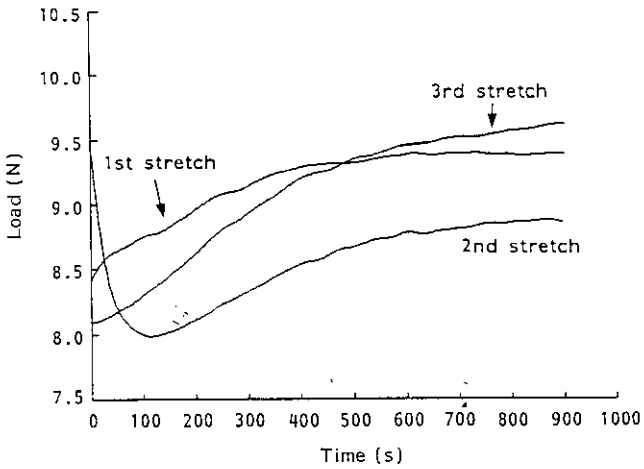

Figure 8 Load–time curves from a relaxation experiment at 4°C using DMEM solution; the three curves were produced by applying 9 N on the sample and recording the decreasing load. Resting periods: 30 min between first and second stretches; 1 h between second and third stretches

temperature with DMEM solution. The slight ascension which was detected in *Figure 5* becomes more apparent in this test where the initial load is 9 N. In fact, the third curve, stopped at 900 s, had a residual load of 8.33 N, and showed no sign of stabilization.

Table 1 gives the various strains produced by stretches 1–3 to an initial load of 9 N in this sample while *Table 2* gives the reduced relaxation function values calculated for the same sample at the end of each relaxation period. The reduced relaxation function ($G(t)$) is defined as:

$$G(t) = \frac{100(\sigma_{init} - \sigma_t)}{\sigma_{init}}$$

If the sample cross-section is taken to be constant, initial and final loads can be substituted for initial and final stresses since

$$\sigma = \frac{F}{A}$$

where σ, F and A represent respectively stress, load and sample cross-section. The reduced relaxation function then becomes:

$$G(t) = \frac{100(F_{init} - F_t)}{F_{init}}$$

The strains listed in *Table 1* show an important drop between the first and second stretch and a slight increase between the second and third stretches. The reduced relaxation function values listed in *Table 2* are strictly decreasing.

Figures 7 and *8*, which illustrate the relaxation tests carried out with no solution and at 4°C respectively also demonstrate a similar ascending slope becoming steeper as the number of stretches increases.

Table 1 Measured strains for the relaxation experiment shown in *Figure 6*

1st stretch	2nd stretch	3rd stretch
11.43%	4.02%	4.53%

Table 2 Reduced relaxation function values calculated for the relaxation experiment shown in *Figure 6*

1st stretch	2nd stretch	3rd stretch
30.03%	17.46%	14.30%

DISCUSSION

The first phenomenon that both the hysteresis and relaxation tests point out is the increase in stiffness of lumbodorsal fascia when successively stretched. This stiffening is illustrated by the greater loads measured at 6% strain in ligaments on the second and third cyclings during hysteresis tests and on the second and third stretches during relaxation tests.

In their study of viscoelastic creep of human digital tendon, Cohen *et al.*[5] observed that consecutive creep tests which were separated by insufficient resting time showed different strains, the strain in the second experiment being smaller than in the first. Cohen explained this by commenting that before application of load in the second experiment, the tendon was in a 'strain-hardened state'. We can suppose that the fascia sample finds itself in this same state when successively stretched: this would explain the increase in load necessary to deform it repetitively by 6% or, conversely, the decrease in its deformation when stressed successively by a constant load.

Relaxation tests using a constant initial load (*Figure 6*) give us information concerning a second phenomenon, the recovery of ligament viscoelastic behaviour. *Table 1*, which lists the strains produced by an initial 9 N load, shows that the second stretching (performed 30 min after the first stretching) induces 65% less strain in the ligament.

However, the 1-h resting period between the second and third loadings at 9 N allowed the strain to increase from 4.02% to 4.53%. This indicates that a sufficient resting period would have restored the strain to its initial value and eliminated in the liagment the strain-hardened state.

The third phenomenon observed in this study of viscoelastic properties of lumbodorsal fascia is ligament contraction. Relaxation curves of *Figures 5–8* all exhibit ligament contraction, but in varying proportions. This is illustrated by the increase in load necessary to maintain the initial strain on the sample (6% in *Figures 4* and *5*, various strains produced by the initial load in *Figures 6–8*) as time progresses. Ligament contraction, swelling or increase in viscosity has many well-documented mechanisms: Elden[8] and Jackson[9] found that pronounced swelling in ligaments develops in the high-acid (pH 2.7) and high-alkaline (pH 8.5) regions; Hayashi[10] reported that solutions with high ionic contents of Ca^{2+}, K^+, and Mg^{2+} increased the normal viscosity of the catch connective tissue in the holothurian body wall between five- and ninefold; the same studies suggested that the contraction of the body wall in low-ionic-strength solutions, i.e. distilled water, could be explained by a rise in osmotic pressure inside the tissue; finally, Elden[6] and Arnold[7] both reported the thermal shrinkage of tendons and collagenous fibre bundles held in isometric conditions at tempera-

tures of 60°C or more. All these studies involved dramatically different experimental conditions, and so served little use in explaining the origin of the ligament contraction observed here.

The unexpected contraction of fascia test samples spurred a series of tests where solution, temperature and humidity were varied. All attempts to eliminate contraction in the test samples failed. It was, however, noticed that samples stretched three times at 6% strain in Ringer solution showed slightly greater contraction than samples tested in DMEM solution (*Figures 4* and *5*). Solution responsibility in the contraction phenomenon does not appear to be convincing, since tests carried out with no solution (only periodical sprayings) also demonstrated an increase in load as time progressed (*Figure 7*). Temperature responsibility was also verified but to no avail, both 4°C (*Figure 8*) and 37°C tests showing similar ligament contraction.

The computer-controlled tensile testing apparatus also came under investigation, to ascertain its correct behaviour in relaxation tests. Experiments using springs and rubber bands showed no contraction and thus proved that the phenomenon was independent of the testing apparatus. Thorough validation tests reported elsewhere[14] confirmed the apparatus reliability.

A possible explanation for the contraction of fascia held under isometric conditions could be the intrusion of muscle fibres in the lumbodorsal fascia. Indeed, many visceral muscles possess the ability to contract spontaneously. Price *et al.*[17] demonstrated that strained and isometrically held intestinal muscles undergo relaxation followed by contraction. In order to test these specimens in a relaxed state (without spontaneous contraction), they used diverse techniques to suppress spontaneous activity, amongst them the use of epinephrine. An histological study of lumbodorsal fascia would therefore be desirable to evaluate whether muscle fibres play a role in the contraction observed here.

The hygroscopic properties of proteoglycans are probably responsible for most of the viscoelastic properties of ligaments. In relaxation tests, the strain may induce internal pressure, forcing the exudation of water out of the proteoglycans, resulting in a general loss of turgidity. It appears unlikely that following an initial perturbation, the affinity of proteoglycans to water starts rising slowly and regains its turgidity, explaining the reported contraction. In future work, we shall verify this possibility by using non-aqueous testing solutions. We shall also assess the effect of digestion of specific components by the ligament matrix by specialized enzymes.

CONCLUSION

The present study suggests that human fascia strained under identical and successive loadings shows three different phenomena. First, stiffening of the sample which decreases strain between first and second loadings. Second, recovery of strain when the sample is allowed to rest sufficiently between loadings. Third, the apparent contraction of fascia samples held under isometric conditions following stretching. This contraction also intensifies as the number of relaxation experiments increases.

Although the literature lists studies where the first two phenomena are reported, it fails to provide insight on the third, the ligament contraction. Our hypothesis that muscle fibres are responsible for the contraction observed in the stress-relaxation tests has yet to be verified by histological studies of lumbodorsal fascia.

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