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Volar/Dorsal Compressive Mechanical Behavior of the Transverse Carpal Ligament

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Abstract

Mechanical insult to the median nerve caused by contact with the digital flexor tendons and/or carpal tunnel boundaries may contribute to the development of carpal tunnel syndrome. Since the transverse carpal ligament (TCL) comprises the volar boundary of the carpal tunnel, its mechanics in part govern the potential insult to the median nerve. Using unconfined compression testing in combination with a finite element-based optimization process, nominal stiffness measurements and first-order Ogden hyperelastic material coefficients (μ and α) were determined to describe the volar/dorsal compressive behavior of the TCL. Five different locations on the TCL were tested, three of which were deep to the origins of the thenar and hypothenar muscles. The average ($\pm$ standard deviation) low-strain and high-strain TCL stiffness values in compression sites outside of the muscle attachment region were 3.6N/mm ($\pm$ 2.7) and 28.0N/mm ($\pm$ 20.2), respectively. The average stiffness values at compression sites with muscle attachments were notably lower, with low-strain and high-strain stiffness values of 1.2N/mm ($\pm$ 0.5) and 9.7N/mm ($\pm$ 4.8), respectively. The average Ogden coefficients for the muscle attachment region were 51.6kPa ($\pm$ 16.5) for μ and 16.5 ($\pm$ 2.0) for α , while coefficients for the non-muscle attachment region were 117.8kPa ($\pm$ 86.8) for μ and 17.2 ($\pm$ 1.6) for α . These TCL compressive mechanical properties can help inform computational models, which can be used to provide insight into the mechanisms of median nerve injury leading to the onset of carpal tunnel syndrome symptoms.

INTRODUCTION

Carpal tunnel syndrome is a compression neuropathy which may arise from median nerve contact with the digital flexor tendons and/or the carpal tunnel boundary. Anatomically based finite element (FE) models of the carpal tunnel (Ko and Brown, 2007) are useful non-invasive tools for studying median nerve stresses induced by contact with these neighboring tissue structures. However, accurate FE simulations must incorporate physiologically realistic material properties of the tissues involved.

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CONFLICT OF INTEREST

The authors have no conflict of interest to report.

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As the volar bound of the carpal tunnel, the transverse carpal ligament (TCL) serves as an important component of the flexor tendon pulley system in the wrist. The flexor tendons and median nerve wrap around this ligament, pushing compressively against the TCL during finger motion and wrist flexion (Armstrong and Chaffin, 1979). Although transverse compressive mechanical properties have previously been determined for the digital flexor tendons and the median nerve (Main et al., 2011), a suitable constitutive model is lacking for TCL volar/dorsal compression.

Numerous studies have investigated overall compliance of the carpal tunnel complex (Li, 2005; Tung et al., 2010; Zheng et al., 2006) and the TCL's role in maintaining carpal bone stability (Garcia-Elias et al., 1989; Guo et al., 2009; Li et al., 2009; Tengrotenhuysen et al., 2009; Xiu et al., 2010). However, only a few studies have isolated the TCL and assessed its intrinsic properties. In one such study, tensile testing performed on bone-ligament-bone preparations found that the TCL's stiffness was 131.8N/mm (Garcia-Elias et al., 1989). In a similar study, it was found that there was no statistically significant difference in TCL tensile properties between specimens excised from patients undergoing carpal tunnel release surgery versus normal control specimens excised from fresh autopsies (Lin et al., 1983).

Although these studies have specifically addressed intrinsic constitutive mechanical properties of the TCL, they addressed only tensile properties in the radial/ulnar direction. *In vivo*, contact between the TCL and the flexor tendons and/or median nerve is compressive, occurring in a volar/dorsal direction (Armstrong and Chaffin, 1979). Knowledge of the TCL's constitutive behavior under compression is therefore important for accurately assessing median nerve contact stresses that develop from contact with the TCL. In the present work, the intrinsic compressive behavior of the TCL was determined using experimental compression testing in conjunction with FE modeling and optimization techniques.

METHODS

Experimental Testing

Intact carpal tunnels were dissected from eight fresh-frozen cadaveric forearm specimens with a mean age of 72 (± 13.7) years (range: 52–90 years; 4 male, 4 female). Prior to dissection, the specimens were thawed overnight at room temperature. The median nerve and digital flexor tendons were removed from the tunnel, and the carpal bone complex was disarticulated distally from the metacarpals and proximally from the radius and ulna. Excess soft tissues (skin, muscle, fat, etc.) were carefully removed from the volar surface of the TCL. K-wires (0.035 inches in diameter) drilled between the carpal bones stabilized the intact tunnel and preserved anatomic ligament/bone alignment. The center of the TCL was determined using landmarks corresponding to the volar-most aspect of the four carpal bones having TCL attachment sites (Figure 1a). The center location was defined as the midpoint between the hamate and trapezium landmarks in the radial/ulnar direction, and half of the specimen's proximal/distal width at that location. A fiber axis corresponding to the TCL's preferential collagen orientation (Prantil et al., 2010) was defined by a line connecting the midpoint between the hamate and pisiform landmarks with the midpoint between the trapezium and scaphoid landmarks (Figure 1b).

The dorsal aspect of the k-wire-stabilized carpal tunnel complex was potted in polymethyl methacrylate (PMMA) to provide an attachment mechanism for securing the specimen for subsequent mechanical testing. The specimens were potted with the TCL fiber axis aligned with a radial/ulnar cross-hair on the potting form. Care was taken to prevent contact between the PMMA and TCL. Once cured, the PMMA block was manually trimmed to expose the ligament's dorsal surface and to separate the radial and ulnar sides of the tunnel. While

maintaining anatomic alignment, the two separated potted ends of the carpal tunnel were adhered to a tensioning apparatus comprised of two Delrin blocks on cylindrical metal slides (Figure 2). A pair of stainless steel compression springs on the slides applied a constant 5N radial/ulnar tensile load, characteristic of the approximate TCL load borne during a grasp maneuver (Schuind et al., 1995).

Five compression sites per specimen were identified. The first site was the previously described center location. Distal, proximal, radial, and ulnar compression sites were identified 5mm in their respective directions from that central location. Because the thenar muscles originate from the radial/distal side of the TCL and the hypothenar muscles originate from the ulnar/distal side of the ligament (Kung et al., 2005), three of the five sites (radial, ulnar, and distal) were in locations involved with muscle origins. These muscle attachment sites were assessed visually, based on the presence or absence of residual muscle fibers. In three TCL specimens, the small TCL size and the arrangement of the carpal bones prevented access of the platens to the radial and/or ulnar compression sites, so these sites were not tested on those three specimens.

The experimental compression device provided a means to test the compressive mechanical behavior of bulk TCL tissue, independent of the entire carpal tunnel complex and at a level above that of individual collagen fibers (Aifantis et al., 2011). The compression testing device utilized a metallic, 3mm diameter cylindrical upper platen, mounted on an MTS Insight (MTS Systems Corporation, Eden Prairie, MN) actuator in series with a 100N load cell (Figure 3). The lower platen was a stationary, identical 3mm cylinder. Prior to testing at each specific site, the height and angulation of the dorsal surface of the TCL was adjusted so as to lie flat on the horizontal surface of the lower platen. This was done using leveling screws in each corner of the tensioning apparatus and a replica of the lower platen. When the specimen had been correctly positioned within the testing apparatus, compression testing was performed by downward movement of the upper platen.

Zero displacement (datum) was established by bringing the platens into contact without a specimen in between. The upper platen was then retracted and the appropriately leveled tensioning apparatus with mounted specimen was placed on the testing device. The specimen was then immersed in a saline bath that was periodically reheated to maintain a temperature 27°C–37°C. Tissue thickness was assessed by displacing the upper platen towards the lower platen at a rate of 0.05mm/s. Thickness was recorded as the distance between platens at incipient load uptake (0.05N). Next, a total of ten loading/unloading cycles to 10% strain were applied for specimen preconditioning. Finally, displacement-controlled compression testing up to 40% of the measured tissue thickness was performed. Preconditioning and compression testing were performed at a loading rate of 0.5mm/s. A total of three replicate compression tests were performed per testing site, with each test followed by a 20-minute recovery in the saline bath.

Compression force and platen displacement were reported during each test, and the three replicate compression data sets for each TCL site were averaged. This average curve was used to obtain two nominal stiffness values for each compression site. Low-strain stiffness was calculated from the slope of force versus displacement curve between displacements corresponding to 10% and 20% tissue strain. Similarly, a stiffness measurement in the higher-strain region was found between displacements corresponding to 30% and 40% tissue strain. The replicate tissue thickness measurements associated with each test were averaged to obtain a single tissue thickness for each TCL site.

Finite Element Model and Optimization Routine

Site-specific FE models for each TCL compression site (Figure 4) were developed within ABAQUS CAE and run in ABAQUS v6.9-3 (Simulia, Providence, RI, USA). All models were axisymmetric to account for the rotational symmetry of the testing platens, and to decrease the analysis time. The platens were modeled as rigid surfaces, each comprised of a total of 25 rigid, axisymmetric, 2-node elements (ABAQUS type RAX2). TCL testing sites were modeled using a 9mm tissue radius and the average tissue thickness at the site of interest. A 1.5mm span of increased mesh refinement was added to the region that would contact the corners of the platens. The ligament was meshed with approximately 5,000 (depending on tissue thickness) continuum, axisymmetric, 4-node hybrid elements (ABAQUS type CAX4H). The TCL was assigned a first-order Ogden hyperelastic material property definition (Ogden, 1972; Ogden, 1997). For ABAQUS implementation of this first-order constitutive model with an incompressibility assumption, strain energy (W) is expressed as a function of the principal stretches (λ_n), with material coefficients μ and α :

$$W(\lambda_1, \lambda_2, \lambda_3) = \frac{2\mu}{\alpha^2} (\lambda_1^\alpha + \lambda_2^\alpha + \lambda_3^\alpha - 3).$$

In this model, μ is predominately related to material behavior in the low-strain region, while α is related to the high-strain region of the stress/strain curve.

The axisymmetric FE models were used in conjunction with a MATLAB least-squares nonlinear optimization function (The Mathworks, Natick, MA, USA) to obtain Ogden hyperelastic coefficients that best represented the average experimental behavior at each compression site (Main et al., 2011). Briefly, each site-specific FE model was run repeatedly while iterative incremental changes were made to the μ and α coefficients governing material behavior of the model. The optimization function adjusted the Ogden coefficients to minimize error between the average experimental and the FE-computed force/displacement curves. The solution was deemed to be converged either when the iterative changes in the Ogden coefficient values or iterative changes in the relative error decreased below a threshold of 1×10^{-4} . Each FE model iteration required less than one minute to run to completion, and the average convergence time for the entire optimization routine was approximately 30 minutes on an HP xw8400 (3.75TB internal HD) with 2 Quad-Core 3 GHz Intel Xeon processors. Statistically significant differences between Ogden coefficients and stiffness measurements from muscle versus non-muscle compression sites were evaluated using a one-tailed Student's t-test with unequal variance, with p-values less than 0.05 considered statically significant.

RESULTS

Thickness values for the five testing sites are reported in Table 1. The low- and high-strain stiffness values for the center and proximal compression locations were notably different from those at the radial, ulnar, and distal locations (Table 1). As the radial, ulnar, and distal locations corresponded to the locations on the TCL where the thenar and hypothenar muscles originate, compressive data from these three sites were grouped into a “muscle attachment” category. Data from the proximal and center locations were grouped into a “non-muscle attachment” category. The average ($\pm$ standard deviation) low-strain compressive stiffness for non-muscle attachment sites was 3.6N/mm (± 2.7), which was significantly ($p=0.002$) higher than the average low-strain stiffness for muscle attachment sites (1.2N/mm ± 0.5). As expected, the TCL was stiffer in the high-strain region, and again the non-muscle attachment sites were significantly ($p=0.0015$) stiffer (28.0N/mm ± 20.2) than the muscle attachment sites (9.7N/mm ± 4.8).

The optimized first-order Ogden coefficients provided good fits of the TCL compression data (Figure 5). To quantify differences between each average experimental curve and its optimized Ogden fit, a tissue strain-normalized sum of the squared error was found between the experimental and FE model forces. The maximum error was 0.38N^2 , and on average was only 0.02N^2 . Also, the maximum difference in force along the experimental and FE curves was determined. The largest difference was 1.48N (8.4% of the maximum experimental force), and on average was only 0.19N . The optimization routine converged to a single set of coefficient values for each site tested (Table 1). The average μ coefficient for the non-muscle attachment sites was 117.8kPa (± 86.8), which was significantly ($p=0.004$) higher than the average μ coefficient for the muscle attachment sites ($51.6\text{kPa} \pm 19.4$). There was no significant difference ($p=0.14$) in the α coefficient between compression sites. The non-muscle and muscle attachment sites had α values averaging 16.5 (± 2.0) and 17.2 (± 1.6), respectively.

DISCUSSION

The first-order Ogden hyperelastic constitutive model provided a reasonable means to describe the compressive behavior of the TCL under physiologically representative loading. Age of cadaveric donor had no clear effect on mechanical behavior, although compression sites associated with muscle attachments (distal, ulnar, and radial) had noticeably lower μ coefficients and lower stiffness values compared to the non-muscle attachment testing sites (proximal and center). It has been observed that muscle-ligament junctions typically demonstrate a smooth transition from striated muscle fibers to ligamentous connective tissue (Schwarzkopf et al., 2008). In the context of this work, that observation suggests that muscle fibers may remain interspersed within the collagen fibers of the TCL (Rotman and Manske, 1993), potentially decreasing the compressive stiffness of the distal, radial, and ulnar locations relative to the proximal and center locations. In addition to the muscle origins on the TCL surface, a separate fascial tissue layer attaches centrally and proximally on the volar surface of the TCL (Stecco et al., 2010). The TCL and this facial layer are similar histologically (Rotman and Manske, 1993), and this additional layer of collagen fibers may serve to increase the compressive stiffness of the TCL at the central and proximal testing sites.

For the eight TCL specimens evaluated, there was significantly more variation in the μ coefficient than in the α coefficient. The dissections were carefully and meticulously performed, but subjective judgments were nevertheless needed regarding the exact margins of the TCL tissue. The higher variability associated with the μ coefficient may be a result of incomplete isolation of the TCL from the soft tissues (muscle, muscle fascia, fat, etc.) overlying the volar surface of the ligament. Contact of the platen with variable amounts of excess tissue on the surface of the ligament may have caused some of the variability in the measurement of initial load uptake by the ligament. Conversely, removing too much surface tissue may have disrupted the normal ligament structure where it was first contacted by the platen, leading to variable initial load uptake before engaging the intact deep tissue under further compression. Similarly, if the TCL surface was not perfectly level on the lower platen, the initial load uptake would tend to be more variable as compression testing drove the tissue down to touch the lower platen before engaging all of the tissue within the compression region. Since the α coefficient predominately reflects higher-strain tissue behavior, it would be less susceptible to slight variations in the initial load uptake.

It is of somewhat limited value to compare the compressive tissue behavior measured in this work with indentation-type studies that have been performed with the TCL as part of the entire carpal tunnel complex. However, indentation is the most common method of evaluating TCL behavior, so it is an important benchmark. The average TCL stiffness in this

work (28.0N/mm–non-muscle attachment and 9.7N/mm–muscle attachment sites) was in the same general range as the TCL stiffness that has been measured by *in vivo* indentation (9.9N/mm for males and 13.3N/mm for females) (Li, 2005). The slight variation may be a result of the fact that the Li 2005 indentation study included compression of the skin and other presumably softer tissues overlying the TCL (muscle, fat, etc.), whereas in this work, all of the tissue deformation was within the well-organized TCL itself. Another very recent indentation study of exposed cadaveric TCLs *in situ* (Holmes et al., 2011) allowed for simultaneous compression and bowing of the TCL under the indenter (and likely a slight compression of the underlying tendons and nerve). The substantial bowing of the TCL during that experiment makes the results more similar to those from a tensile test, than to the present study. Since the TCL has previously been found to be relatively stiff (131.8N/mm) in tension (Garcia-Elias et al., 1989), the difference in testing methods may explain why the TCL stiffness for the loading configuration studied by Holmes et al. was stiffer than that found for pure compression in this work.

The stiffness of the TCL measured in this work can be more directly compared to the previously measured transverse compressive behavior of the digital flexor tendons and the median nerve (Main et al., 2011). Optimized Ogden coefficients obtained for the TCL specimens were significantly larger than the coefficient values reported for the compressive behavior of the digital flexor tendons and the median nerve (Table 2). Because the TCL is significantly stiffer, contact occurring between the median nerve and the TCL would tend to elicit the development of greater contact stresses on the nerve than would result from contact between the median nerve and the digital flexor tendons.

This work has several limitations. First, the ligament geometry in the FE model was approximated as a uniform thickness along a 9mm length, while in actuality the thickness of the tissue varied slightly at locations 5mm away. To minimize the influence of variations in tissue thickness, the compression platen was small (3mm diameter), and only 1.5mm of the axisymmetric tissue model was loaded, with that loading centered where thickness had been measured. The remaining model length where the tissue thickness was more approximated served mainly as linkage to a remote boundary condition, so that boundary constraints would not unduly influence the portion of the model being compressed. Secondly, although a physiologically based 5N tensile load was applied in the radial/ulnar direction during compression testing, no specific radial/ulnar tensioning step was included in the FE model. Thus, while constitutive properties determined in this work are characteristic of the compressive behavior of the TCL under the applied tensile load of 5N, it remains to be seen what the behavior would be under other tensile loads.

In conclusion, the Ogden coefficients determined in this work provide a reasonable means to describe the volar/dorsal compressive properties of the TCL to 40% strain. This work focused on the behavior of the TCL independent from the other carpal tunnel structures, so that the compressive properties determined would be suitable for implementation into anatomically based FE models of the carpal tunnel. It was found that the non-muscle attachment regions of the TCL were significantly stiffer than the muscle attachment regions. Accurate soft tissue material behavior is needed for accurately quantifying median nerve contact stress, which in turn could help provide a better understanding of potential mechanisms of injury to the median nerve leading to the onset of carpal tunnel syndrome.

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Figure 1. (a) Landmarks (red dots) identified on the TCL's attachment sites on the scaphoid, trapezium, pisiform, and hamate. (b) The ligament's preferential fiber axis is illustrated with the dashed line connecting the scaphoid/trapezium and pisiform/hamate midpoints.

Figure 2. Tensioning apparatus with an attached TCL specimen. The five testing locations (center, proximal, distal, ulnar, and radial) are identified with purple dots. Compression springs applied 5 N of tension to the TCL specimen, and leveling screws in the corners of the Delrin blocks allowed for height adjustment.

Figure 3. TCL compression set-up shown with a TCL specimen attached to the tensioning apparatus and immersed in a saline bath.

Figure 4.

An example site-specific 2D axisymmetric model used for analysis. Each test site had an individualized mesh for the tissue thickness that was measured experimentally. The mesh was highly refined in the area where the upper platen and lower platen corners contacted the tissue, to ensure well-converged contact. The remote end of the tissue specimen (away from the symmetry axis) was constrained against motion in the dorsal/volar- and radial-direction (green fixities). The axisymmetric model revolved around the axis of symmetry is shown in the inset.

Figure 5. Illustrative plot of compressive force versus strain, for a non-muscle region (blue) compression site and a muscle region (red) compression site. Experimental data are shown with the dotted line and the FE data output using the optimized fit of the data are shown with the solid line.

Summary of data from experimental testing and FE optimization, grouped by testing site. The low-strain (LS: 10%–20%) and high-strain (HS: 30%–40%) stiffness values were calculated using the appropriate strain range portions of the average experimental force-versus-displacement curves for each compression site. Average Ogden coefficient values were obtained from the FE optimization routine for each TCL testing site.

Table 1

Location		Thickness		Stiffness				Ogden Coefficients			
		(mm)		LS (N/mm)		HS (N/mm)		μ (kPa)		α	
		avg.	st. dev.	avg.	st. dev.	avg.	st. dev.	avg.	st. dev.	avg.	st. dev.
NM	center (n = 8)	1.9	0.6	2.8	2.0	24.7	18.4	96.7	59.0	17.1	1.7
	proximal (n = 8)	1.7	0.4	4.3	3.3	31.4	22.6	138.9	107.8	17.2	1.5
	distal (n = 8)	2.5	0.5	1.3	0.5	9.6	3.5	60.6	19.4	16.3	1.8
M	ulnar (n = 5)	2.2	0.3	1.3	0.4	13.2	4.1	52.3	12.6	18.0	1.6
	radial (n = 6)	2.3	0.6	0.9	0.5	6.8	5.6	39.0	19.4	15.5	1.9

Table 2

Ratios of the average optimized TCL Ogden coefficients to previously reported (Main et al., 2011) Ogden coefficient values for the median nerve, flexor pollicis longus (FPL) tendon, superficial digital flexor tendons, and deep digital flexor tendons.

Tissue Ratio	Non-Muscle Region		Muscle Region	
	μ (kPa)	α	μ (kPa)	α
TCL to Nerve	9.1	2.6	4.0	2.5
TCL to FPL	4.7	1.6	2.1	1.5
TCL to Superficial	3.3	2.0	1.5	1.9
TCL to Deep	3.0	1.9	1.3	1.8