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Cervical facet capsular ligament yield defines the threshold for injury and persistent joint-mediated cervical pain

Kyle P. Quinn
University of Pennsylvania

Beth A. Winkelstein
University of Pennsylvania, winkelst@seas.upenn.edu

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Cervical facet capsular ligament yield defines the threshold for injury and persistent joint-mediated neck pain

Kyle P. Quinn and Beth A. Winkelstein

Department of Bioengineering

University of Pennsylvania

Philadelphia, PA 19104

Original Article

Correspondence: Beth A. Winkelstein, PhD
                     Department of Bioengineering
                     University of Pennsylvania
                     240 Skirkanich Hall
                     210 S. 33rd St
                     Philadelphia, PA 19104-6321
                     (215) 573-4589 (phone)
                     (215) 573-2071 (fax)
                     winkelst@seas.upenn.edu

Abstract

The cervical facet joint has been identified as a source of neck pain, and its capsular ligament is a likely candidate for injury during whiplash. Many studies have shown that the mechanical properties of ligaments can be altered by subfailure injury. However, the subfailure mechanical response of the facet capsular ligament has not been well defined, particularly in the context of physiology and pain. Therefore, the goal of this study was to quantify the structural mechanics of the cervical facet capsule and define the threshold for altered structural responses in this ligament during distraction. Tensile failure tests were preformed using isolated C6/C7 rat facet capsular ligaments (n=8); gross ligament failure, the occurrence of minor ruptures and ligament yield were measured. Gross failure occurred at 2.45±0.60 N and 0.92±0.17 mm. However, the yield point occurred at 1.68±0.56 N and 0.57±0.08 mm, which was significantly less than gross failure (p<0.001 for both measurements). Maximum principal strain in the capsule at yield was 80±24%. Energy to yield was 14.3±3.4% of the total energy for a complete tear of the ligament. Ligament yield point occurred at a distraction magnitude in which pain symptoms begin to appear *in vivo* in the rat. These mechanical findings provide insight into the relationship between gross structural failure and painful loading for the facet capsular ligament, which has not been previously defined for such neck injuries. Findings also present a framework for more in-depth methods to define the threshold for persistent pain and could enable extrapolation to the human response.

1. Introduction

Whiplash injury is a frequent mechanism for chronic neck pain and accounts for half of all patient-care expenses from motor vehicle accidents (Quinlan et al., 2004). Despite the frequency and costs of whiplash, little is known about the injury mechanism in these painful neck injuries. The cervical facet joint has been identified as a source of neck pain (Barnsley et al., 1993, 1994; Cavanaugh, 2000; Chen et al., 2005; Kallakuri et al., 1999; Lord et al., 1996; Lu et al., 2005a,b), and is a likely candidate for mechanical injury due to the bony motions of the spine during neck loading (Bogduk and Yoganandan, 2001; Cusick et al., 2001; Deng et al., 2000; Kaneoka et al., 1999; Ono et al., 1997; Panjabi et al., 1998; Sundararajan et al., 2004; Yoganandan and Pintar, 1997). The capsular ligament in the lower cervical facet joints can, in some cases, exceed its physiologic limit due to an altered spinal kinematic during whiplash (Grauer et al., 1997; Ito et al. 2004; Luan et al., 2000; Pearson et al., 2004; Stemper et al., 2005). However, the consequence of this joint's motion and its relationship to the tensile mechanical response of the facet capsular ligament remains largely undefined and speculative. While the tensile failure properties of the human cervical facet capsule have been previously defined (Mykelbust et al., 1988; Winkelstein et al., 1999, 2000; Yoganandan et al., 2000), these studies do not provide context for the consequences of subfailure loading or possible injury to the ligament.

Many studies have demonstrated the potential for facet capsule injury prior to gross failure. Subfailure capsule injury has been previously observed in isolated ligament and full cervical spine cadaveric specimens (Panjabi et al., 1998; Siegmund et al., 2001; Winkelstein et al., 2000; Yoganandan et al., 2001). Specifically, minor ruptures in the facet capsule were noted before gross ligament failure; these minor failures ("sub-catastrophic" failures) were observed at strains ranging from 35.0-64.8% in shear and tension, implying structural damage of the tissue may be possible before its

failure (Siegmund et al., 2001; Winkelstein et al., 2000). In studies of isolated human cervical spines undergoing inertial loading comparable to whiplash, Pearson et al. (2004) estimated C6/C7 ligament strains of 39.9%; these exceeded strains during physiological motion and led those authors to propose excessive capsular strain as a cause of ligament injury during whiplash. Lee et al. (2004a,b) developed an *in vivo* model of neck pain in the rat to define relationships between tensile loading across the facet joint and behavioral hypersensitivity. In that model, bilateral C6/C7 vertebral distractions between 0.6-0.9 mm produced persistent pain symptoms (Lee et al. 2004a,b). While those *in vivo* studies suggest injury below ligament failure, they have not defined the specific structural response of the capsular ligament. Thus, studies of the isolated cervical facet capsular ligament are needed to define its mechanical response for subfailure loading conditions and to identify the onset of altered structural responses.

Therefore, the primary goal of this study is to define the ligament yield point during tensile distraction of the rat C6/C7 facet capsule. Yield has been suggested as a threshold for injury (Yoganandan et al., 1989). We hypothesize that the displacement and force at which ligament yield occurs will precede gross failure of the ligament. In this study, the loading direction, rate, and spinal level were chosen to match previous *in vivo* studies (Lee et al., 2004a,b), enabling comparisons between the mechanical response at yield and the outcomes from *in vivo* studies. Findings will provide a mechanical determination of an injury response and provide context for ligament mechanics and painful loading conditions.

2. Methods

2.1. Specimen preparation and loading procedure

Male Holtzman rats (n=8) weighing 372-422 g, were used in this study. Studies were approved by the University of Pennsylvania Institutional Animal Care and Use

Committee. The C6/C7 spinal motion segment was harvested and frozen at -20°C for mechanical testing later. Prior to testing, motion segments were thawed, cleared of all remaining musculature and soaked in saline. The right facet joint was removed *en bloc* at the pedicles and spinous processes. The left joints were carefully refrozen for future studies. The ligamentum flavum, interspinous ligament, supraspinous ligament, and dura mater were transected, and the carotid tubercle was removed.

A customized mechanical interface with an Instron 5865 (Instron Corporation, Norwood, MA), was used to apply pure tension (0.02 mm accuracy) to the C6/C7 facet joint by gripping each of the laminae and the transverse processes of the C6 and C7 vertebrae with micro-forceps (Fig. 1a). The superior grips attached to a 10 N load cell (Instron Corporation, Norwood, MA; accuracy of 0.25% measured value). The vertical position of the superior grips and the lateral position of the inferior grips were adjusted to minimize the intervertebral distance (between the midpoints of the C6 and C7 laminae), while maintaining zero measured load (Fig. 1). Fiduciary marks were made on the specimen using a felt tip pen with a 0.20 mm tip to track ligament deformation. The ligament was re-hydrated with saline and preconditioned with 30 cycles to 0.2 mm (approximately 5% of load at gross failure). After 2 minutes of rest, the ligament was distracted at 0.08 mm/s to 5 mm, which was a distraction sufficiently past gross failure. The loading rate was selected to match mean loading rates used *in vivo* (Lee et al., 2004a,b). Force and displacement data were collected at 1 kHz. Two CCD cameras (Vision Reasearch, Inc., Wayne, NJ) were triggered by Instron crosshead motion and simultaneously captured the ligament deformation (38 pixels/mm) at 50 Hz. One camera visualized the dorsal aspect of the ligament (Fig. 1b), while the other camera imaged the lateral aspect (Fig. 1c) so that the entire ligament was observed during testing. Images were used to verify that the specimen did not slip from the forceps' grip and to track ligament displacements during loading.

2.2 Data & statistical analysis

To obtain a consistent reference position for the facet joints in this study and future *in vivo* studies, the gage length was defined at an intervertebral distance of 2.53 mm. This distance was selected based on a pilot study (n=3), and corresponded to a preload of 0.010±0.001 N.

Ligament yield was defined by a decrease in stiffness (Yoganandan et al., 1989). To calculate stiffness throughout loading, the force-displacement data (Fig. 2a) were differentiated using a centered finite difference approximation: $k_i = \dfrac{F_{i+1} - F_{i-1}}{\delta_{i+1} - \delta_{i-1}}$ (Fig. 2b), where the instantaneous stiffness (k_i) at a given time point i, was calculated from the difference in force (F) and displacement (δ) between the previous ($i{-}1$) and following ($i{+}1$) time points. To identify changes in stiffness, the second derivative of the force-displacement data was estimated using the same approach (Fig. 2c). Gross failure was defined at the displacement with maximum force. A minor rupture was defined to occur at any displacement for which stiffness dropped below zero prior to gross failure. Yield occurred when stiffness decreased at a rate of more than 100 N/mm^2 over 1.7 μm (±10 data points). These parameters were chosen to ensure a robust prevention of false positives using this technique.

Maximum linear stiffness and energy were also calculated. To determine the maximum linear stiffness, the instantaneous stiffness data points were sorted starting with the greatest stiffness value until the standard deviation was greater than 5% of the mean – taken as a significant deviation from linearity. A mean value was defined as the maximum linear stiffness. This value is analogous to the slope of a fit to linear portion of the force-displacement curve. Through numerical integration, the energy required for ligament yield, gross failure, and complete ligament rupture were also computed (Fig.

2d). Energy for ligament yield was expressed as a percentage of the total energy for complete rupture.

Maximum principal strain in the capsular ligament was calculated from the deformation of the fiduciary marks during loading. Marks were digitized (2.2% strain precision) into grids containing 14 to 34 nodes for each of the aspects of the ligament at the undeformed, yield, minor rupture, and gross failure points. Four-node planar elements were created, and Lagrangian strain was computed for each element using a customized isoparametric mapping program in Matlab 7.0 (Mathworks Inc., Natick, MA). The maximum principal strain in each specimen for each time point was recorded. The video data were also used to identify the anatomic location of the first ligament tear. Tensile force and displacement were compared between gross failure and the ligament yield point using a paired, one-tailed Student's t-test, and significance was defined as $p < 0.05$.

3. Results

The mean tensile force at gross failure was 2.45±0.60 N, corresponding to a distraction of 0.92±0.17 mm (Tables 1 and 2). The first minor rupture of each ligament occurred at a mean load of 2.20±0.78 N with 0.69±0.13 mm of distraction (Tables 1 and 2). One specimen (AU) did not rupture until gross failure. Decreases in stiffness immediately preceded all minor ruptures (Fig. 2). Yield occurred at 1.68±0.56 N and 0.57±0.08 mm (Tables 1 and 2). Maximum stiffness was 5.45±1.07 N/mm, starting at 0.69±0.13 mm. Yield occurred at significantly lower force and displacement from that of gross ligament failure ($p < 0.001$ for both measures). The energy required for ligament yield was 14.3±3.4% of the total energy required for complete ligament rupture (Table 3); energy to failure was 1.05±0.44 mJ.

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Ligament damage was visible at the yield point for any specimen (Fig. 3). The maximum principal strain in the capsule at yield was 80±24% (Table 4). Small tears first became visible in the lateral aspect of the ligament during the first minor rupture event (Fig. 3c). For each specimen, these lateral tears began over the joint line or the C6 articular process, and progressed dorsally at gross failure (Fig. 3d), which typically resulted in a large tear along the dorsal-lateral ridge of the C6 articular process or the joint line (Fig. 3e). Maximum principal strain in the capsule at minor rupture and gross failure were 92±27% and 151±111%, respectively (Table 4).

4. Discussion

This is the first study to define the facet capsular ligament yield and identify it as distinct from gross ligament failure. While tensile cervical facet capsule properties have been previously reported for the rat *in situ* (Lee et al., 2006), the present study applies pure tensile loading, allowing detection of a yield point and minor ruptures. Mean ligament failure force at gross failure of the present study (Table 2) was similar to that previously reported (Lee et al., 2006). However, the displacement at gross failure (0.92 mm) was lower than that determined *in situ*; this difference can be attributed to experimental conditions. In the present study, the ligamentum flavum was transected; this was not possible *in situ*. The present study applied tension across the joint space, whereas the *in situ* study likely also imposed a component of lateral rotation that would result in larger displacement measurements prior to gross failure. Of note, the displacements at yield measured here (Table 1) are consistent with those joint distractions producing altered physiologic responses *in vivo* (Lee et al., 2004a,b). This suggests that, in addition to its mechanical relevance, capsular ligament yield may also correspond with benchmarks for pain.

The presence of minor ruptures is consistent with observations in failure studies of the human facet capsule (Siegmund et al., 2001; Winkelstein et al., 2000). In those studies minor ruptures occurred in over half of the specimens tested, and the site of gross failure was in the mid-substance of the lateral side of the ligament for the majority of specimens. Likewise, the site of failure and maximum principal strain in our study was in the mid-substance of the lateral aspect for the majority of specimens. Cadaveric human and *in vivo* caprine models of facet loading have suggested these minor ruptures may be the injury event responsible for pain (Lu et al., 2005a; Siegmund et al., 2001; Winkelstein et al., 1999, 2000). While minor rupture of the ligament may correspond to a joint distraction capable of producing pain, our results suggest that changes in the ligament response occur before its rupture. In fact, yield may be more directly related to the conditions of ligament loading adequate to *initiate* pain. Minor rupture did not occur in every specimen, and immediately follows ligament yield in only two specimens (Table 1). In contrast, ligament yield (0.57±0.08 mm) is a more consistent event than rupture (0.69±0.13 mm), having less variation in displacement. This is because yield is a more sensitive measure as it does not require stiffness to decrease to zero (Fig. 2) (Yoganandan et al., 1989). As such, yield can provide a more precise and conservative measure for defining an injury threshold. While the presence of minor ruptures indicates that both the rat and human ligaments undergo similar patterns of damage during tensile loading, further studies are needed to quantify yield for tensile and combined loading of the human facet. Use of the fraction of total energy required to yield (Table 3), and its relationship to gross failure and minor ruptures (Tables 1 and 2), could provide scaling methods if direct detection is not feasible in the human.

In this study, small tears were observed on the lateral aspect of the ligament starting at the first minor rupture. These observations are consistent with the lack of noticeable ruptures during *in vivo* studies that only imaged the dorsal aspect of the

ligament during loading (Lee et al. 2004a,b). While no tears were observed in any of the ligaments at yield in our study (Fig. 3b), it has been hypothesized that displacing soft tissue beyond yield results in permanent deformation (McMahon et al., 1999; Yoganandan et al., 1989). Previous studies have identified soft tissue yield prior to gross failure, defined by a decrease in linear stiffness (Haraldsson et al., 2005; McMahon et al., 1999; Neumann et al., 1992, 1993; Yoganandan et al., 1989); this same concept has been implemented in our study. These and other studies suggest that subfailure loading may produce microscopic damage that changes the mechanical properties of the tissue including: increased laxity, decreased stiffness, and altered viscoelastic properties (Iatridis et al., 2005; Panjabi et al., 1996; Pollack et al., 2000; Provenzano et al. 2002). Continued efforts with this type of experiment and others using increased video resolution, polarized light microscopy, and histological examination could be helpful for detecting micro-tears and other physiological consequences for the magnitudes of distraction associated with ligament yield. Moreover, while studies have reported differential effects of freezing, much work has shown that the frozen storage of ligament has little-to-no effect on its mechanical properties (Callaghan & McGill, 1995; McElhaney et al., 1983; Moon et al., 2006; Turner et al., 1988; Viidik & Lewin, 1966; Woo et al., 1986). The data reported here reflect a response following a single freeze-thaw-preconditioning cycle and should be interpreted accordingly.

The current findings from this study provide biomechanical perspective for previous studies supporting capsule stretch as a mechanism of facet-mediated pain and whiplash injury. Distractions of the rat capsular ligament greater than 0.7 mm have elicited persistent behavioral hypersensitivity and other pain symptoms (Lee et al. 2004a,b). Also, specific aspects of the cellular nociceptive response of the central nervous system are significantly altered after subfailure distraction but not for lower distraction magnitudes (Lee et al., 2004a). Because ligament yield (0.57 mm; Table 1)

occurred below the distraction known to produce persistent pain symptoms and above distraction levels (0.2-0.3 mm) known not to produce any behavioral sensitivity (Lee et al., 2004a,b), the findings from the current study suggest that the mechanical threshold for persistent pain from ligament loading may be near yield. Electrophysiological studies reported discharge saturation thresholds and activation thresholds for nociception at strains of 44.2±16.7% and 47.2±9.6%, respectively in a caprine model (Lu et al., 2005a); those local strains corresponded to joint distraction magnitudes beginning between 12 and 14 mm for the goat facet joint. In our study, yield occurred at 62% of the distraction for gross failure (Table 1). Applying that relationship to the goat, a corresponding yield point would occur between 12 and 14 mm. Interestingly, partial ligament tears were observed beginning at 14 mm of distraction (Lu et al., 2005a). The pure tensile mode of loading used in our study is not the primary joint kinematic in whiplash. Yet, ligament distraction has been reported as a significant local motion in the facet capsular ligament for whiplash in a number of studies and the findings presented here will enable future investigations using the more complicated whiplash kinematics of compression-extension and shear (Pearson et al., 2004; Siegmund et al., 2001; Sundararajan et al., 2004; Winkelstein et al., 2000; Yoganandan et al., 2002).

Maximum principal strains in the capsular ligament are reported (Table 4) to provide context with other studies in the literature. In general, the strains at minor rupture (92±27%) are higher than those reported in other studies of human capsular ligaments for tensile (64.8±73.8%) and shear (35±21%) failure (Siegmund et al., 2001; Winkelstein et al., 2000). However, the mean maximum principal strain at ligament rupture in this study (151±111%; Table 4) is consistent with those strains in the human ligament during tensile failure (Winkelstein et al., 1999, 2000; Yoganandan et al., 2000). Strains at yield (80±24%) were lower than those at minor rupture (Table 4). Based on imaging and estimated C6/C7 ligament thickness (unpublished data), the corresponding

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mean failure stress in this study ranged between 2-4 MPa, which is consistent with failure stress reported for upper and lower human cervical joint capsules (5.67-7.36 MPa; Yoganandan et al., 2000). Continued efforts are ongoing and focused both on defining the regional thickness of the rat cervical facet capsule ligament and examining the stress-strain response of this ligament at yield.

Findings presented here suggest a relationship between structural damage of the facet capsular ligament and potential mechanisms of pain for subfailure distraction. Our data show ligament yield at a significantly lower distraction than gross failure. While these subfailure distractions may not produce visible ligament tears, detection of the ligament's altered structural response may provide an indication of an injury sufficient to elicit sustained nociceptor firing, pain symptoms, and persistent activity in the nervous system. Given the evidence that painful joint distractions begin near ligament yield, this study may suggest that the physiologic range of the facet joint is actually limited to prior to yield. This mechanical study provides a framework for future *in vivo* studies in determining a mechanical threshold for persistent pain, and also provides data for quantitative scaling to other animal models and to the human. These findings provide mechanical definition of altered ligament behavior corresponding with a loading condition known to produce pain, linking mechanical damage and persistent pain for the first time.

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Figure Legends

Figure 1. Fixture for gripping specimens and camera views. (a) The position of each micro-forceps is adjustable in 3 degrees of freedom. Both the C6 and C7 vertebral levels of the specimen (AW) are gripped by the laminae and transverse processes. C6 is connected to the load cell and moving platen, while C7 remains fixed. Dorsal **(b)** and lateral **(c)** camera views of the specimen are shown here at the time of yield. The calibration bar in **(c)** represents 1 mm and also applies to **(b)**.

Figure 2. Analysis of mechanical data from a representative specimen (AF). (a) Force-displacement plot indicating the yield point, first minor rupture, and gross failure. **(b)** The stiffness plot shows the threshold for minor ruptures. **(c)** This plot demonstrates the change in stiffness and the threshold for yield. **(d)** The area under the force-displacement plot shows the corresponding energies to yield (dark shading), gross failure (medium shading), and complete rupture (light shading). Plots **(a-c)** are shown up to gross ligament failure.

Figure 3. Lateral view of representative specimen (AT) during ligament distraction and gross rupture. (a) Unloaded ligament. **(b)** At the ligament yield point no ruptures are visible. **(c)** At the first occurrence of minor ruptures a small tear begins. **(d)** At gross failure the small tear widens (tear is located between the two arrowhead tips). **(e)** At an additional 0.2 mm of distraction beyond gross failure in (d), an appreciable hole (within circle) is apparent on the ventral side of the lateral aspect of the ligament, and is spreading dorsally. The calibration bar in (a) represents 1 mm and applies to all images.

Table 1

Table 1. Summary of displacements measured during events.

Specimen	Gross Failure (mm)	Minor Rupture (mm)	Yield Point (mm)
AF	0.82	0.80	0.59
AG	0.82	0.53	0.51
AM	1.05	0.69	0.58
AN	0.67	0.63	0.53
AO	0.92	0.53	0.46
AT	0.98	0.85	0.70
AU	0.87	0.87*	0.57
AW	1.21	0.65	0.65
Mean (SD)	0.92 (0.17)	0.69 (0.13)	0.57 (0.08)

* did not rupture prior to gross failure

Table 2

<table>
<tr><td colspan="5" align="center">**Table 2. Summary of forces and maximum stiffness.**</td></tr>
<tr><td rowspan="2">Specimen</td><td>Gross Failure</td><td>Minor Rupture</td><td>Yield Point</td><td>Maximum Stiffness</td></tr>
<tr><td>(N)</td><td>(N)</td><td>(N)</td><td>(N/mm)</td></tr>
<tr><td>AF</td><td>3.17</td><td>3.16</td><td>1.91</td><td>6.98</td></tr>
<tr><td>AG</td><td>2.07</td><td>1.69</td><td>1.65</td><td>4.57</td></tr>
<tr><td>AM</td><td>3.49</td><td>3.22</td><td>2.69</td><td>6.41</td></tr>
<tr><td>AN</td><td>2.36</td><td>2.31</td><td>1.94</td><td>5.46</td></tr>
<tr><td>AO</td><td>2.06</td><td>1.69</td><td>1.50</td><td>4.97</td></tr>
<tr><td>AT</td><td>2.62</td><td>2.57</td><td>1.81</td><td>6.54</td></tr>
<tr><td>AU</td><td>2.00</td><td>2.00*</td><td>1.02</td><td>4.41</td></tr>
<tr><td>AW</td><td>1.81</td><td>0.95</td><td>0.93</td><td>4.24</td></tr>
<tr><td>Mean (SD)</td><td>2.45 (0.60)</td><td>2.20 (0.78)</td><td>1.68 (0.56)</td><td>5.45 (1.07)</td></tr>
</table>

* did not rupture prior to gross failure

Table 3

Specimen	Total Energy (mJ)	Energy to Gross Failure (mJ)	Energy to Yield (mJ)	Energy to Yield (% of Total E)
AF	3.04	0.98	0.38	12.3
AG	3.47	0.91	0.38	11.0
AM	4.11	2.08	0.74	17.9
AN	2.00	0.70	0.40	20.0
AO	2.28	0.98	0.30	13.3
AT	2.32	1.06	0.39	16.6
AU	1.66	0.67	0.20	11.9
AW	1.79	1.05	0.20	11.3
Mean (SD)	2.58 (0.87)	1.05 (0.44)	0.37 (0.17)	14.3 (3.4)

Table 3. Summary of energies.

Table 4

Table 4. <u>**Summary of maximum principal strains measured during events.**</u>

Specimen	Gross Failure (%)	Minor Rupture (%)	Yield Point (%)
AF	115	118	86
AG	61	43	44
AM	122	72	65
AN	108	96	82
AO	192	86	73
AT	117	126	115
AU	86	86*	62
AW	411	108	110
Mean (SD)	151 (111)	92 (27)	80 (24)

* did not rupture prior to gross failure

Figure 1

(a) Load Cell — Micro-forceps — Dorsal Camera — Specimen — Lateral Camera

(b) Dorsal View — Intervertebral distance — C6 Transverse Process — C7 Lamina — 16:19:25.611 823

(c) Lateral View — C6 Transverse Process — 16:21:36.135 922

Figure 2

Specimen AF

(a) (b) (c) Minor Rupture (d) Gross Failure (e)