Mechanical Response of the Porcine Temporomandibular Joint Disc to an Impact Event and Repeated Tensile Loading

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Aims: *To test for orthotropy in the stress-strain behavior of the temporomandibular joint (TMJ) disc under repeated physiologic loading before and after an impact event.* ***Methods:*** *Two groups, each consisting of 10 discs, were subjected to repeated tensile cycling in the dorsoventral (group 1) and mediolateral (group 3) direction. Two additional groups, each consisting of 10 discs, had preconditioning in the form of a 1.18 N·s impulsive load before tensile cycling in either the dorsoventral (group 2) or mediolateral (group 4) direction. Physiologic loads of 1 to 3 N were cycled at 0.1 Hz, and stress-strain responses were recorded every cycle between 1 to 10 cycles, and then periodically at 50, 100, 500, 750, and 1,000 cycles. The properties of elastic modulus, residual strain upon unloading, and area contained within the hysteresis loop were measured.* ***Results:*** *Dorsoventral loading produced 5-fold higher elastic modulus, 5-fold lower residual strain, and 5-fold lower hysteresis compared to mediolateral tensile loading ($P \leq .001$). Repeated loading effectively reduced the viscous response for all discs, as the elastic modulus increased while residual strain and hysteresis decreased. Impulsive loading caused elastic modulus to increase for dorsoventrally cycled discs, whereas hysteresis decreased for mediolaterally cycled discs ($P \leq .05$).* ***Conclusion:*** *The findings suggest that damage from the impact load may have increased the porosity of the extracellular matrix, which ultimately resulted in additional stress transfer to the collagen fibers during loading. Impulsive loads may be an important preconditioning factor in the fatigue failure of the TMJ disc in vivo.*
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Key words: fatigue, TMJ disc, cartilage, viscoelasticity, stress-strain

Osteoarthritis is a progression of degenerative changes occurring within articular tissues in response to joint loading. Although its etiology appears to be multifactorial, trauma and the subsequent fatigue failure of the cartilage collagen matrix are predominant factors.[1–5] Degeneration of the cartilage is first recognizable as fibrillation and splitting of collagen on the cartilage surface, with an increase in joint friction and wear, and eventual thinning of the cartilage.[6,7] Chondrocyte death and changes in matrix composition are associated with changes in peak mechanical stress, stress rate, and loading duration.[8–10]

The temporomandibular joint (TMJ) is a heavily loaded structure, and the TMJ disc possesses a limited capacity for distributing stress and lubricating the joint.[11–15] Recent evidence suggests that the TMJ disc's mechanical behavior is intricately linked to its highly oriented collagen fiber network, which comprises a large component of the cartilage. The TMJ disc is mechanically orthotropic in nature, with high tensile modulus and strength observed along the dorsoventral axis of the disc. Histologic evidence suggests that within the load-bearing "intermediate zone," major collagen fiber bundles are oriented parallel to the dorsoventral axis of the disc.[16] Depending upon strain rate, stresses on the order of 28 to 38 MPa are required to extend and ultimately fracture the collagen when the load is applied along this axis.[17] In comparison, the density of lateral collagen branches and intermolecular crosslinks, oriented along the mediolateral and superior-inferior axes, is very low.[16] As a result, stresses of only 1 to 2 MPa will cause disc failure to occur when tension is applied along the mediolateral axis.[17] The failure pattern typically shows a splaying apart of dorsoventral fibers, suggesting that initial tissue failure most likely occurs along the weaker mediolateral- and superior-inferior–oriented fibers. Crack orientation is expected to follow the dorsoventral orientation of primary fibers. Although data are limited, cracks induced from rapid impulse loading appear to follow a dorsoventral orientation.[7,17]

It has been shown that critical threshold values exist for the TMJ disc's ability to resist static and impulse loads, above which surface damage occurs.[7,18] Surface cracks appear, thereby increasing the porosity of the extracellular matrix. Excessive hydration of the proteoglycan-water complex ensues, which introduces additional tensile stress into the collagen fibers surrounding a defect. Subsequent joint loading causes the water to flow outward, thereby collapsing the collagen network and reducing the disc's ability to provide weeping lubrication[19] to the joint. In time, friction and wear of articulating surfaces increase, exacerbating the degenerative process.[7]

A goal of this study was to gain an understanding of the TMJ disc's early fatigue behavior following exposure to an impulsive load. Tensile tests were used to measure mechanical property changes since it has been shown that tensile property changes precede surface damage caused by compressive loading.[20] A key feature of this project was that all stresses were maintained at physiologic levels of no more than 0.4 MPa. The first hypothesis tested was that mechanical property values would differ based on the orientation of the tensile stress axis. A second hypothesis was that the structural damage introduced into the TMJ disc following impulsive loading would significantly change its mechanical properties. The properties evaluated in this study were tensile elastic modulus, residual tensile strain, and the amount of hysteresis occurring during a loading-unloading cycle. Due to its functional and anatomic similarity to the human TMJ disc, a porcine model was chosen for testing.[18,21]

Materials and Methods

Specimen Preparation

Specimen preparation procedures followed those described previously.[17] Briefly, fresh porcine TMJ discs were obtained from a local abattoir. The discs were dissected from a posterior auricular approach. Right and left disc pairs were identified for each animal and stored at room temperature (25ºC) in 0.1 M phosphate buffer saline (pH 7.3) for 1 hour. The discs were frozen at –15ºC for no more than 14 days. At the time of mechanical testing, the discs were thawed in 37ºC phosphate buffer saline and trimmed into dumbbell shapes with a metal die cutter. The long axes of the dumbbells were oriented along the mediolateral axes of the right TMJ discs and along the dorsoventral axes of the left TMJ discs. One dumbbell was constructed from each disc. Gauge width was measured with a translating stage measuring microscope, disc thickness was measured with a calibrated linear voltage differential transformer (LVDT), and grip distance served as gauge length. Due to inherent biologic variation in disc thickness occurring within the biconcave region of the intermediate zone, each disc was chosen to fall within the range of 1.7 to 2.2 mm thickness. This produced dumbbell gauge dimensions of 4 mm width × 7.5 mm length × approximately 2 mm thickness. Ten dumbbells were fabricated for each of 4 test groups. Group 1 was subjected to repeated tensile cycling in the dorsoventral direction and group 3 was cycled in the mediolateral direction. Two additional groups were subjected to a 1.18 N·s impulsive load before tensile cycling in either the dorsoventral (group 2) or mediolateral (group 4) direction.

Tensile Testing Procedures

A servo-hydraulic testing machine loaded all specimens in cyclic tension between 1 to 3 N for 1,000 cycles. This loading regimen represented a range of load magnitude that produced stresses approximate to stresses reported for human mastication,[22,23] and it represented a small portion of the stress-strain response typically reported (Fig 1). A sinusoidal waveform was followed during loading, and each specimen was bathed continuously in 37ºC phosphate-buffered saline during cyclic testing.

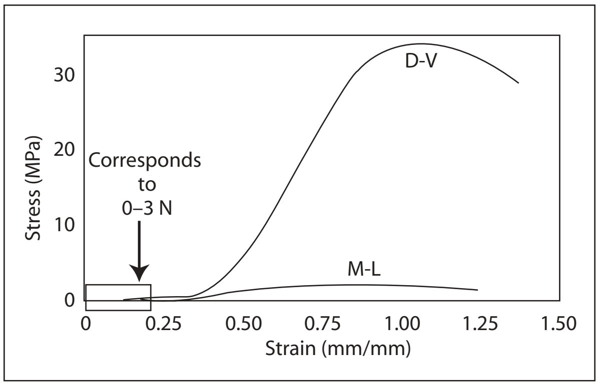

Fig 1 Typical stress-strain curves for 1-time tensile test to failure of porcine TMJ disc. Boxed area represents loading range used in this study. A 500 mm/min displacement rate was used to determine the curves.[17] D-V = dorsoventral loading axis, M-L = mediolateral loading axis.

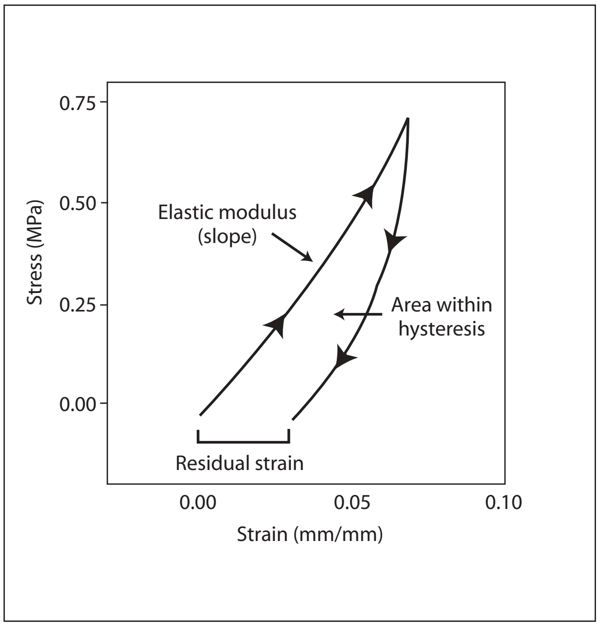

Fig 2 *(Right)* Magnified view of boxed region shown in Fig 1, with measured stress-strain properties identified.

A 0.1 Hz cycling frequency was chosen to provide thorough specimen hydration throughout the test. Stress-strain curves for complete loading-unloading cycles were recorded at cycles 1 to 10, 50, 100, 500, 750, and 1,000. To collect stress-strain data from an unloaded state, loads in the range of 0 to 3 N were applied at these cycles. The properties of tangent elastic modulus, residual (unrecovered) strain, and area contained within the loading-unloading hysteresis loop were measured (Fig 2). These properties were chosen as measures of the elastic, viscous, and combined elastoviscous responses to loading, respectively.

Impulse Loading Procedures

Specimens from test groups 2 and 4 were impulsively loaded prior to being cut into dumbbell shapes. Impulsive loads were delivered using a custom-built apparatus,[7,17] which dropped a mass from a specific height onto an instrumented indenter resting on the disc's surface. India ink was applied to the surface of the indenter prior to indentation, which later facilitated positioning of the metal die cutter during dumbbell fabrication. The magnitude of the impulse was determined from the expression:

$$\text{Impulse magnitude} = \int_{t1}^{t2} F dt = m[2gh]^{1/2}$$

where F was the force associated with the impulse, dt was time, m was the mass, g was the gravitational constant, and h was the height from which the mass was dropped. The height and mass were held constant for all tests, and the discs were exposed to average impulse magnitudes of 1.18 ± 0.182 N·s prior to mechanical testing.

Data Analyses

Means and standard deviations were calculated for each TMJ disc group for the 3 dependent variables of elastic modulus, residual strain, and hysteresis area. The entire data set was subjected to a 2-way analysis of variance (ANOVA)[24] to determine a presence of significant differences ($P \leq .05$) based on loading orientation (dorsoventral, mediolateral) and number of tensile cycles. If significant differences were observed, a Tukey-Kramer post hoc test[24] was used to identify pairwise differences ($P \leq .05$). To study the effects of impulsive loading over time, 2-tailed t tests for each mechanical property were conducted between pristine and impulsively loaded groups for each loading orientation at cycles 1 to 10, 50, 100, 500, 750, and 1,000. Each cycle contained its own unique data subset, therefore only 1 t test was applied to each dorsoventral and mediolateral group pair. An α value of 0.05 with 8 degrees of freedom was chosen.

In the event disc failure occurred before reaching 1,000 load cycles, the number of cycles at failure was recorded and the data were used to construct survival curves. The Kaplan-Meier product-limit method[25] was used to calculate the probability of survival S(t) for each group, and a Log Rank test[25] was applied to compare survival of pristine discs and impulsively loaded discs for cyclical tensile loading along the dorsoventral and mediolateral axes.

Table 1 Mechanical Properties Based on Tensile Axis Orientation (mean ± SEM)

Orientation	Modulus (MPa)	Residual strain (mm/mm)	Hysteresis area (MPa · mm/mm)
D-V	33.8 ± 1.29	0.0046 ± 0.00067	0.0022 ± 0.00015
M-L	7.35 ± 0.23	0.0265 ± 0.00488	0.0094 ± 0.00098

Mean values are pooled for pristine and impulsively loaded specimens, and for number of tensile cycles. For all 3 properties, differences between D-V and M-L are significant at a $P \leq .001$ confidence level. Results from ANOVA and Tukey-Kramer test. D-V = dorsoventral, M-L = mediolateral.

Results

Results obtained from the 2-way ANOVA indicated that tensile axis orientation and the number of loading cycles significantly affected the properties of elastic modulus, residual strain, and area within the loading-unloading hysteresis loop ($P \leq .001$). Significant 2-way interaction terms were noted for residual strain and hysteresis area, indicating that the effect of dorsoventral and mediolateral orientations on these 2 properties were dependent on the number of cycles. Tukey-Kramer comparisons demonstrated that dorsoventrally loaded groups exhibited 5-fold higher values for elastic modulus, and 5-fold lower values for both residual strain and hysteresis area, compared to mediolaterally loaded samples (all $P \leq .001$, Table 1). With pristine and impulsively loaded groups pooled, values for elastic modulus were significantly higher for dorsoventrally loaded discs at each recorded cycle ($P \leq .05$, not shown). However, the effects of loading orientation on residual strain was limited to cycle numbers 1 to 6 and hysteresis area for cycle numbers 1 to 5 ($P \leq .05$, not shown).

Figures 3 to 5 illustrate the effects of repeated tensile cycling on the 3 mechanical properties. Early changes in stress-strain behavior were characterized by an increase in elastic modulus and closing of the hysteresis loop. For all groups, the most dramatic change in mechanical behavior occurred between cycles 1 and 2. Elastic modulus values increased while values for residual strain and hysteresis substantially decreased. With repeated loading this overall trend continued, but changes in residual strain and hysteresis area were smaller after approximately 10 cycles (Figs 4 and 5). By 100 cycles, maximum values of elastic modulus were recorded, which were nearly double those observed for the first cycle. The maximum values were maintained until approximately 500 cycles, at which point the elastic moduli decreased, eventually attaining 68% to 85% of the maximum value at 1,000 cycles (Fig 3).

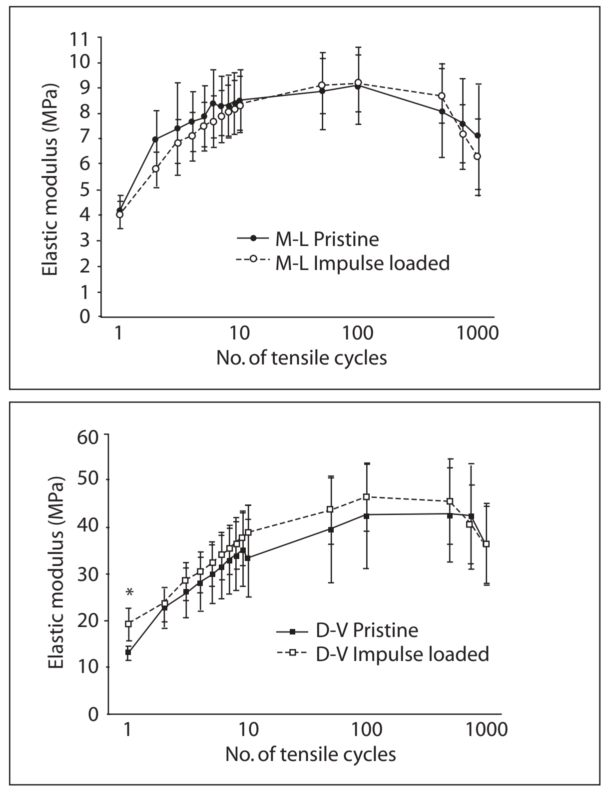

Fig 3 Graphs of elastic modulus vs number of tensile cycles for pristine and impulsively loaded discs. *Denotes significant *t* test comparison ($P \leq .05$) between pristine and impulsively loaded discs at a given cycle number. Abscissa printed in log10 scale. *(Top)* mediolateral (M-L) cycling, *(Bottom)* dorsoventral (D-V) cycling.

Differences in mechanical properties between pristine and impulsively loaded discs at each cycle generally were small and not statistically significant, based on results from *t* tests ($P > .05$). The mechanical property curves presented in Figs 3 to 5 are nearly identical for pristine and impulsively loaded groups, with 2 exceptions. Significantly different values were observed for elastic modulus in dorsoventrally loaded discs at cycle 1 (Fig 3), and for hysteresis area in mediolaterally loaded discs at cycles 50 and 100 (Fig 5, $P \leq .05$).

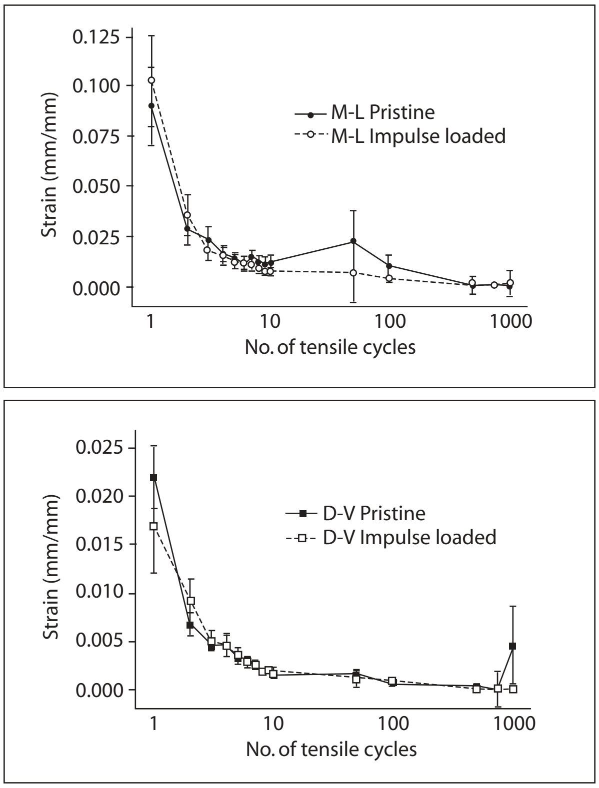

Fig 4 Graphs of residual strain vs number of tensile cycles for pristine and impulsively loaded discs. All *t* test comparisons between pristine and impulsively loaded discs were nonsignificant ($P > .05$). Abscissa printed in log10 scale. *(Top)* mediolateral (M-L) cycling, *(Bottom)* dorsoventral (D-V) cycling.

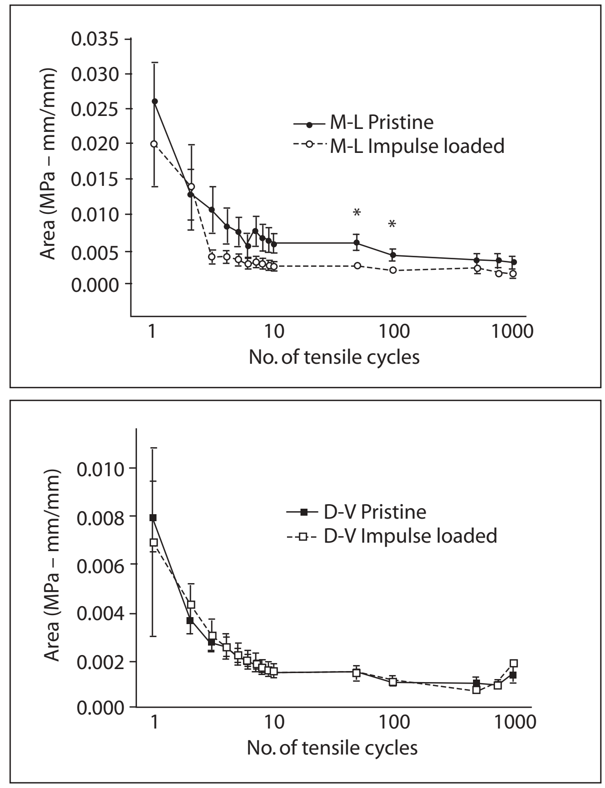

Fig 5 Graphs of hysteresis area vs number of tensile cycles for pristine and impulsively loaded discs. *Denotes significant *t* test comparison ($P \leq .05$) between pristine and impulsively loaded discs at a given cycle number. Abscissa printed in log10 scale. *(Top)* mediolateral (M-L) cycling, *(Bottom)* dorsoventral (D-V) cycling.

Disc failure was uncommon during tensile cycling for the pristine mediolateral disc group and for all dorsoventral disc groups, as Kaplan-Meier estimates demonstrated a 90% to 100% probability of survival after 1,000 cycles (Fig 6). However, discs that were impulsively loaded and subsequently subjected to repeated mediolateral tensile forces demonstrated a 70% survival probability after 1,000 cycles. Results from the Log Rank test indicated a tendency of this group to fail more frequently ($P = .065$).

Discussion

Since primary collagen fibers located on bearing surfaces of the TMJ disc are oriented predominantly in the dorsoventral direction,[16] the tissue's response to mechanical loading is expected to be nonuniform and highly dependent upon the magnitude, direction, and duration of local tensile stress. Results from this study showed that tension applied parallel to the predominant axis of collagen orientation (dorsoventrally) generated an initial stress response that was mostly elastic in nature, as evidenced by a high elastic modulus during loading and low amounts of unrecovered strain during unloading. By comparison, tension applied perpendicular to the collagen fiber alignment (mediolaterally) demonstrated an initial stress-strain response that contained a greater viscous component, typified by lower elastic modulus, greater unrecovered strain during unloading, and greater hysteresis. These results support those reported previously for 1-time tensile tests, in which mechanical properties differed along D-V and M-L orientations.[17]

In this study, repeated loading was accompanied by a stiffening of the extracellular matrix and a loss of viscous response, regardless of loading axis direction. Consistent with the behavior of cartilage under load, early stress-strain changes observed during the first 10 tensile cycles suggested fluid movement out of the porous extracellular matrix. As a result, a greater share of the load was borne by the collagen component of the disc matrix, which in turn underwent changes in its viscoelastic response. Subsequent decreases in elastic modulus observed between 500 and 1,000 cycles, with little accompanying change observed in residual strain and hysteresis, suggested that over time the collagen network structure may have become altered, even when loads low in magnitude and frequency were applied. Under normal circumstances the mechanical changes in the collagen component of the disc's matrix would not occur as quickly as seen in the results shown here. Mastication usually is limited to approximately 10 loading and unloading cycles, punctuated by variable periods of rest, which presumably allows for rehydration of the tissue. Therefore, it is likely that fatigue in these circumstances will take a considerably larger number of cycles, if it occurs at all. It is possible that nocturnal bruxism more closely resembles the experimental protocol used in this study. During bruxism, loads of significant magnitude are maintained on the disc for up to and exceeding 300 seconds. It is possible that under these circumstances, disc matrix rehydration is less likely, and the probability of fatigue is much greater.

Results reported in previous research suggest that mediolaterally oriented collagen fibers undergo fracture under impact, which likely results in a loss of intermolecular crosslinking between adjacent dorsoventrally directed collagen fibers.[7,17] This occurrence further reduces the low collagen fiber density present in the mediolateral direction and increases the mobility of dorsoventral fibers. The net result would be an increase in porosity, followed by excess hydration of the proteoglycan-water complex. As a result, higher tensile stresses would be placed on the collagen network at rest. Any subsequent loading of the tissue would result in more rapid fluid flow out of interfibrillar spaces, thereby increasing the elastic and decreasing the viscous responses at a faster rate, as compared to a healthy disc. Support for such phenomena was provided by the results of this study, where elastic modulus in the dorsoventral direction was consistently higher for damaged discs, and hysteresis area in the mediolateral direction was consistently lower (Figs 3 and 5). In contrast, few differences were observed between pristine and impacted discs for other stress-strain properties. This may be explained by considering that for dorsoventral tensile loading, an inherently low viscous response was observed in healthy discs. Any further reductions produced by impulsive loading caused values for residual strain and hysteresis area to fall into the range of inherent biologic variation occurring among tissue samples, which could not be differentiated by the present measuring system. For tensile loading mediolaterally, the low collagen density in this direction produced elastic modulus values for pristine discs that were 5 times

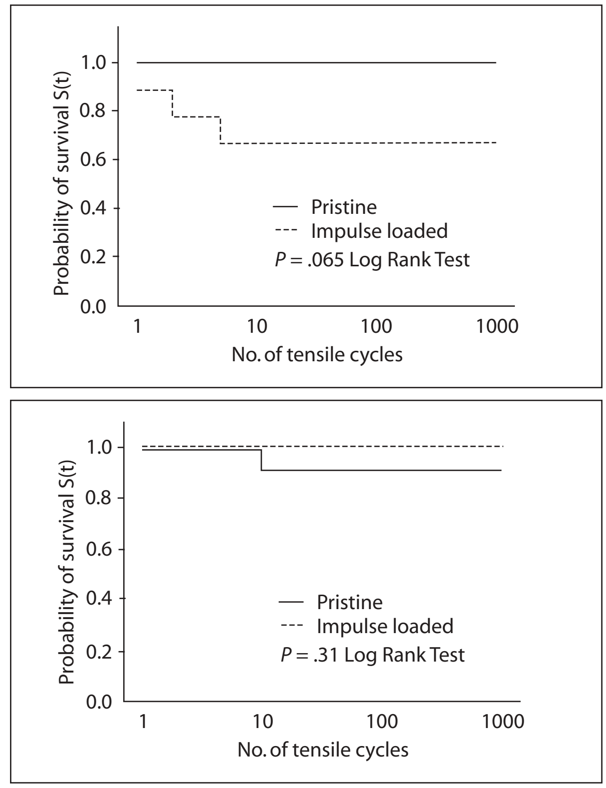

Fig 6 Kaplan-Meier survival curves and results from Log Rank tests for pristine and impulsively loaded discs. Abscissa printed in log10 scale. *(Top)* mediolateral cycling, *(Bottom)* dorsoventral cycling.

lower than those observed dorsoventrally, and damage caused by impulsive loading did not additionally lower the modulus to impart statistical significance. The fact that differences were seen between pristine and impulsively loaded discs for hysteresis area, but not residual strain, suggests that the property of residual strain may not be a sensitive enough measure for detecting tissue damage within the loading regimen used in this study.

Disc failures presented in Fig 6 were observed following the delivery of an impulsive load, and all failures were seen during the first 8 cycles of tensile loading along the mediolateral axis. The data suggest that even when an impulsive blow of moderate magnitude was delivered (1.18 N·s), this exceeded a threshold value for failure. Clinically this implies that following a traumatic event, localized failure may occur during the first few chewing cycles in regions of the disc where mediolateral tensile stresses are imposed. It is conceivable that subcritical threshold damage also may have occurred in discs that did not fail by 1,000 cycles, and flaw propagation to failure would occur only with additional cycling or through delivery of increased tensile load.

The results presented here generally supported the first stated hypothesis, that mechanical property values for the porcine TMJ disc are different along the dorsoventral versus mediolateral axis. However, repeated loading effectively reduced the viscous component for all discs, and property differences observed between the 2 loading directions for residual strain and hysteresis became less conspicuous with increasing numbers of cycles. The second stated hypothesis was only partly supported, namely, that an impulsive blow delivered to the TMJ disc significantly alters the tensile properties of elastic modulus, residual strain, and hysteresis. The inconsistencies in satisfying the second hypothesis are rooted in errors of 2 underlying assumptions of the hypothesis, *(1)* that the viscous and elastic responses to tensile load do not act together and *(2)* that the stress response in the dorsoventral and mediolateral directions are separate entities. In reality, the disc's mechanical response to load is completely congruous, where a significant viscous component present mediolaterally acts in concert with the predominant elastic component present dorsoventrally. When the disc is damaged by impact, its porosity presumably increases, the collagen is altered, and the mechanical response in both directions is affected. An important area for future study will be to measure changes in apparent porosity present in healthy and damaged TMJ discs, and to determine if the degree of porosity increases when both the load magnitude and number of load cycles increase.

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References

1. Peyron J. Review of the main epidemiologic-etiologic evidence that implies mechanical forces as factors in osteoarthritis. Eng Med 1986;15:77–79.
2. Freeman MAR, Meachim G. Aging and degeneration. In: Freeman MAR (ed). Adult Articular Cartilage, ed 2. London: Pitman Medical, 1979.
3. Kreutziger KL, Mahan PE. Temporomandibular degenerative joint disease. Part I. Anatomy, pathophysiology, and clinical description. Oral Surg Oral Med Oral Pathol 1975;40:165–182.
4. Radin EL, Paul IL, Rose RM. Role of mechanical factors in pathogenesis of primary osteoarthritis. Lancet 1972;1: 519–522.
5. Harkins ST, Marteney JL. Extrinsic trauma: A significant precipitating factor in temporomandibular dysfunction. Comparative Radiography 1985;54:271–273.
6. Stegenga B, de Bont LGM, Boering G. Osteoarthrosis as the cause of craniomandibular pain and dysfunction: A unifying concept. J Oral Maxillofac Surg 1989;47:249–256.
7. Nickel JC, Iwasaki LR, Feely DE, Stormberg KD, Beatty MW. The effect of disc thickness and trauma on disc surface friction in the porcine temporomandibular joint. Arch Oral Biol 2001;46:155–162.
8. Chen CT, Burton-Wurster N, Lust G, Bank RA, Takoppele JM. Compositional and metabolic changes in damaged cartilage are peak-stress, stress-rate and loading-duration dependent. J Orthop Res 1999;17:870–879.
9. Clements KM, Bee ZC, Crossingham GV, Adams MA, Sharif M. How severe must repetitive loading be to kill chondrocytes in articular cartilage? Osteoarthritis Cartilage 2001;9:499–507.
10. Ewers BJ, Dvoracek-Driksna D, Orth MW, Haut RC. The extent of matrix damage and chondrocyte death in mechanically traumatized articular cartilage explants depends on rate of loading. J Orthop Res 2001;19:779–784.
11. Hylander WL. Mandibular function and temporomandibular joint loading. In: Carlson DS, McNamara JA, Ribbens KA (eds). Developmental Aspects of Temporomandibular Joint Disorders. Ann Arbor: University of Michigan, 1984:19–35.
12. Smith DM, McLachlan KR, McCall WD. A numerical model of temporomandibular joint loading. J Dent Res 1986;65:1046–1052.
13. Nickel JC, McLachlan KR, Smith DM. A theoretical model of loading and eminence development of the postnatal human temporomandibular joint. J Dent Res 1988;67:903–910.
14. Nickel JC, McLachlan KR. In vitro measurement of the stress distribution properties of the pig temporomandibular joint disc. Arch Oral Biol 1994;39:439–448.
15. Nickel JC, McLachlan KR. In vitro measurement of the frictional properties of the temporomandibular joint disc. Arch Oral Biol 1994;39:323–331.
16. Mills DK, Fiandaca DJ, Scapino RP. Morphologic, microscopic, and immunohistochemical investigations into the function of the primate TMJ disc. J Orofac Pain 1994;8: 136–154.
17. Beatty MW, Bruno MJ, Iwasaki LR, Nickel JC. Strain rate-dependent orthotropic properties of the pristine and impulsively loaded porcine temporomandibular joint disc. J Biomed Mater Res 2001;57:25–34.
18. Tait DC. Impulsive and Static Loading of the Porcine Temporomandibular Joint Disc [thesis]. Winnipeg: University of Manitoba, 1993.
19. McCutchen CW. Joint lubrication. Bull Hosp Jt Dis Orthop Inst 1983;43:118–129.
20. McCormack T, Mansour JM. Reduction in tensile strength of cartilage precedes surface damage under repeated compressive loading in vitro. J Biomech 1997;31:55–61.
21. Kuboki T, Shinoda M, Orsini MG, Yamashita A. Viscoelastic properties of the pig temporomandibular joint articular soft tissues of the condyle and disc. J Dent Res 1997;76:1760–1769.
22. Iwasaki LR, Nickel JC, McLachlan KR. Relationship between growth, function and stress in the temporomandibular joint. In: McNeill C (ed). Science and Practice of Occlusion. Chicago: Quintessence, 1997:125–136.
23. Tanaka E, Tanne K, Sakuda M. A three-dimensional finite element model of the mandible including the TMJ and its application to stress analysis in the TMJ during clenching. Med Eng Phys 1994;16:316–322.
24. Sokal RR, Rohlf FJ. Biometry: The principles and practice of statistics in biological research, ed 2. San Francisco: WH Freeman, 1981:245,251–252,321–344.
25. Lee ET. Statistical methods for survival data analysis. New York: Wiley, 1992.