Spinal Facet Joint Biomechanics and Mechanotransduction in Normal, Injury and Degenerative Conditions

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The facet joint is a crucial anatomic region of the spine owing to its biomechanical role in facilitating articulation of the vertebrae of the spinal column. It is a diarthrodial joint with opposing articular cartilage surfaces that provide a low friction environment and a ligamentous capsule that encloses the joint space. Together with the disc, the bilateral facet joints transfer loads and guide and constrain motions in the spine due to their geometry and mechanical function. Although a great deal of research has focused on defining the biomechanics of the spine and the form and function of the disc, the facet joint has only recently become the focus of experimental, computational and clinical studies. This mechanical behavior ensures the normal health and function of the spine during physiologic loading but can also lead to its dysfunction when the tissues of the facet joint are altered either by injury, degeneration or as a result of surgical modification of the spine. The anatomical, biomechanical and physiological characteristics of the facet joints in the cervical and lumbar spines have become the focus of increased attention recently with the advent of surgical procedures of the spine, such as disc repair and replacement, which may impact facet responses. Accordingly, this review summarizes the relevant anatomy and biomechanics of the facet joint and the individual tissues that comprise it. In order to better understand the physiological implications of tissue loading in all conditions, a review of mechanotransduction pathways in the cartilage, ligament and bone is also presented ranging from the tissue-level scale to cellular modifications. With this context, experimental studies are summarized as they relate to the most common modifications that alter the biomechanics and health of the spine—injury and degeneration. In addition, many computational and finite element models have been developed that enable more-detailed and specific investigations of the facet joint and its tissues than are provided by experimental approaches and also that expand their utility for the field of biomechanics. These are also reviewed to provide a more complete summary of the current knowledge of facet joint mechanics. Overall, the goal of this review is to present a comprehensive review of the breadth and depth of knowledge regarding the mechanical and adaptive responses of the facet joint and its tissues across a variety of relevant size scales. [DOI: 10.1115/1.4004493]

Keywords: spine, facet joint mechanics, mechanotransduction, articular cartilage, capsule, biomechanics

1 Introduction

The zygapophyseal, or facet, joints are complicated biomechanical structures in the spine, with complex anatomy, mechanical performance and effects on overall spine behavior and health. At each spinal level, there is a pair of facet joints located on the postero-lateral aspects of each motion segment, spanning from the cervical to the lumbar spine (Fig. 1). These facet joints are typical diarthrodial joints with cartilage surfaces that provide a low-friction interface to facilitate motion during normal conditions in a healthy spine. Owing to the anatomy of the spine, the mechanical behavior of the facet joint is both dependent on the responses dictated by the overall spine's response and also can directly affect the spine's response, via its relationship to the intervertebral disc, its anatomic orientation, and its own mechanical behavior. The kinematics and mechanical properties of the facet joint and its tissue components have been studied extensively for a variety of different loading conditions [1–11]. Recently, there is growing interest in the facet joint—its biomechanics and physiology—with the advent of disc arthroplasty and there has been increased attention to the relationship between spinal degeneration and its effects on the mechanical environment of the different tissues in the facet joint [12–16]. Therefore, it is the primary goal of this review to present an updated perspective of the anatomy and global mechanics of the spinal facet joint and its individual tissue components in conjunction with their loading during physiologic and nonphysiologic motion. In addition, this review will summarize the mechanotransduction processes by which mechanical loading to the specific tissues of the joint translate into signals that drive physiologic responses in health, injury and trauma, and spinal degeneration. Computational models of the facet joint are also reviewed since there has been quite a bit of work in this area to complement and expand findings from biomechanical experiments and to provide insight about facet joint mechanics otherwise not measureable in typical cadaveric studies. Overall, this review focuses on synthesizing this anatomical, biomechanical and physiological information to give an overview of the facet joint's response to mechanical loading from the macroscopic to the cellular scale, with implications and perspective for future studies of this spinal joint.

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subjected to axial compression [52]. The stress-strain relationship is anisotropic as it depends on the dimensions and organization of the collagen fibers, and the cellular and proteoglycans content that differ across the depth of the cartilaginous layer. In addition, articular cartilage deformation results from a reorganization of its collagen structure and loss of fluid during loading. Fluid loss is a much slower process than the polymer network re-arrangement and so an initial deformation occurs first without any volume change, and a second deformation then results from a change in volume due to fluid loss which produces a nonlinear load-displacement response that is exhibited during unconfined compression [54,55]. This type of behavior highlights the biphasic and time-dependent mechanical properties of articular cartilage in diarthrodial joints [56,57]. Accordingly, creep studies have also demonstrated that the time constant (T) of cartilage to reach equilibrium under maintained compressive loading is a function of the thickness (h), the equilibrium modulus (E), and specific properties of the cartilage, as well as the applied load (π) [51,56]. This equilibrium time constant also depends on porosity (Φ^f), permeability (k), and the drag coefficient (K) as described in Eq. (5):

$$T = \frac{h^2 \cdot K}{\left(\Phi^f\right)^2 \cdot E} = \frac{4 \cdot h^2}{\pi^2 \cdot E \cdot k} \tag{5}$$

In contrast, because of the interactions between the collagen fibers and the proteoglycans during uniaxial confined compression, the relationship between axial and radial stresses (σ) and axial strain (ε) is defined by a linear isotropic constitutive relation (Eqs. (6) and (7)) [58]. In this relationship, the compression axial modulus (H_A) and the chemical stress (β) imposed by the surrounding milieu, as well as the Lamé constants (λ and G), all depend on the concentration (c) of the environment.

$$\begin{Bmatrix} \bar{\sigma}_{11}(c) \\ \bar{\sigma}_{22}(c) \\ \bar{\sigma}_{33}(c) \end{Bmatrix} = \begin{bmatrix} \lambda(c) \\ \lambda(c) \\ H_A(c) \end{bmatrix} \cdot \varepsilon + \beta(c) \tag{6}$$

with

$$H_A(c) \cong 2 \cdot G(c) + \lambda(c) \tag{7}$$

Articular cartilage is a composite material composed of fluid (water) and solid (chondrocytes, collagen, proteoglycans) phases that has anisotropic nonlinear mechanical properties and load-bearing capacity [59]. The difference in response time of the two phases contained in articular cartilage makes its mechanical response dependent on the rate of loading. The dynamic stiffness of the cartilage lining in diarthrodial joints increases with strain rate [60–62]. For example, in a study of cartilage impacts during knee graft implantation, fissures in the cartilage matrix were produced for both single high energy impacts (over 25 MPa) and repetitive impacts (26–35 MPa) across a variety of human, bovine, and porcine species [60,63]. Chondrocyte viability was also reduced by up to 60% for impacts of 1 J compared to a 5% and 20% decrease in cell viability for impacts of 0.25 J and 0.5 J, respectively [52,62,63]. Fissures in the articular surface can allow the enzymes that are contained in the synovial fluid in the joint, such as collagenase and hyaluronidase, to penetrate and break down the cartilage matrix [48]. At the same time, an increase in chondrocyte death can also impair the subsequent synthesis of cartilage proteins that are required for the proper maintenance of the avascular cartilage matrix [63]. A damaged cartilage matrix cannot effectively support compressive loads, distribute pressure, or resist stresses because fissures can penetrate as far as the transition zone and disrupt the matrix structure [62]. In addition, the repair of the cartilaginous matrix and its functionality are compromised by the death of the chondrocytes because the production of molecules imperative for matrix regeneration is reduced, which is then followed by the denaturation of the collagen fibers and the release of proteoglycans, which are needed to retain water and to provide compressibility for the damaged cartilage structure [48,63]. Impact(s) on the articular cartilage can; therefore, cause significant loss of mechanical properties and cellular damage which also may provide the stimulus for the onset of degeneration in that tissue and/or can also accelerate it [52,64,65]. However, the energy transferred to the *facet* joint cartilage during physiologic and/or nonphysiologic loading of the spine remains to be measured.

Explicit experimental studies of facet joint cartilage are limited. Currently, there is only one investigation of canine lumbar facet cartilage, reporting its aggregate modulus to be 554 kPa at equilibrium after an indentation with a 1 mm flat-ended porous-tip [66]. That study also found that the aggregate modulus of cartilage from the facet joint was similar to the modulus of cartilage from other canine diarthrodial joints such as the knee lateral condyle, patellar groove, and shoulder, suggesting that the similarities between human and canine articular cartilage could also extend to facet joint cartilage [66]. It was also reported that human cartilage from the knee and the hip has a compressive stiffness comparable to that of the distal femur in canine models and the proximal femur in baboon models, respectively [67,68], which suggests that the mechanical properties of articular cartilage may be similar among any diarthrodial joints in the body. However, further characterization of human facet joint tissue is needed to verify if the mechanical properties of facet joint cartilage are similar across species as well.

2.3 Synovium, Menisci and Capsular Ligament. Extending from the superior to the inferior articular pillar, two superposed membranes, the synovium and the ligamentous capsule, maintain the articular surfaces in a low-friction environment and provide mechanical resistance to their separation and relative motion. The synovium of the facet joint is a thin and soft periarticular connective tissue [17] with two main layers that secrete synovial fluid components involved in the maintenance of the synovial fluid used to lubricate and nourish the cartilaginous articular surfaces [69,70]. The synovium also regulates the exchanges between the blood and synovial fluid, and contains macrophage cells that phagocytose cell debris and waste contained in the joint cavity [70]. Although the functional role of this structure has been investigated at the cellular level [70,71], it has not been investigated mechanically, most likely because it is difficult to isolate since it is very thin and its outermost layer is intimately merged to the inner surface of the capsular ligament [71]. For the same reasons, and also because the innermost synovial layer is loose, the synovium also likely does not play a substantial role in the mechanical behavior of the joint as a whole. Although, the synovial membrane is very thin, its loose innermost layer bulges into the joint cavity in some areas, forming folds that wedge between the opposing articular surfaces of the facet joint [72–74].

The synovial folds, or meniscoids or menisci, are intra-articular structures that protect the articular cartilage when opposing articulating surface glide on each other during joint motion [75]. This protection is realized since the meniscoids compensate for the incongruence of the joint's articular surfaces, guiding and smoothing their relative motion, and distributing the load over a greater surface area [72,76,77]. Three main types of menisci have been identified in the facet joints across all of the regions of the spine: adipose tissue pads, fibro-adipose meniscoids, and connective tissue rims (Fig. 2). The adipose pads and meniscoids are located mainly at the periphery of the articular surface in the anterior and posterior region of the joint, where they only partially extend circumferentially along the rim of the articular pillar. These tissues are crescent-shaped and have a triangular cross-section in the sagittal plane (Fig. 2), with the base being attached to the capsule and the point extending as much as 9 mm inward toward the interior of the joint [72,75,78]. The connective rims of the synovial tissue are ring-shaped, wraparound the edge of the bony pillar, and are tapered inward towards the center of the joint [72,74,75,77,78]. The meniscoids are composed of fat, fibrous connective tissue

and/or a mix of fat and fibers covered by a cellular synovial lining [72–75,77,79]. Although the meniscoids are known to cover the gap of exposed subchondral bone at the articular surface in order to reduce friction during articular motion, their mechanical role is still unclear [77]. They have been speculated to moderate the load transferred to the cartilage when the articular surfaces engage in compression during any joint motion by distributing the pressure as they move freely in and out of the inter-articular space during motion [72,75–77,80]. This putative function is probably linked to the meniscus entrapment theory that was developed to explain how low back pain symptoms could be caused, and then treated by simple manipulation [73,81]. Although this may be the case under normal loading, these intra-articular folds can become torn at their base under combined substantial compression and shear loading which can also lead to subcapsular hemorrhage and entrapment of the torn pieces in the joint, eventually inducing further physiologic dysfunction and even pain [73,74,82]. Although postmortem and in vivo MRI studies provide evidence of the presence of meniscoids in the spinal facet joints and help to characterize their dimensions and composition, the role of these structures in the biomechanical behavior of the whole facet joint remains unclear.

As in the other joints in the body, such as the knee and the hip, the facet capsular ligament covers the synovium to fully enclose the facet joint, enveloping it in the superior-inferior direction and with nonuniform thickness. For instance, the lumbar facet capsule has been reported to be 2.0 mm thick in the posterior region, and as much as 3.2 mm thick in the anterior region, whereas the superior and inferior regions are approximately 2.4 mm thick [83].The capsular ligament is comprised of dense collagen fiber bundles linked by proteoglycans, with elastin fibers and fibroblasts interspersed [18,84]. The collagen and elastin fibers extend between the laminae of adjacent vertebrae connecting to the ligamentum flavum both in the antero- and postero-medial regions of the facet joint, and completely surrounding the joint's articular surfaces in three dimensions. The collagen fibers are oriented differently along the superior-inferior axis of the capsular ligament [84] and they are crimped [18]. The crimped collagen fibers allow the capsule to undergo substantial excursions without reaching its mechanical limit or inducing local injury. Under load, the fibers can become uncrimped which allows the overall joint to translate and rotate without offering any mechanical resistance.

The capsule, as well as the subchondral bone, synovium and folds, are richly innervated with mechanoreceptive, proprioceptive and nociceptive nerve endings [21,85–88].Therefore, mechanical loading of any of those innervated tissues in the facet joint could activate nerve endings and modulate the signals in the nervous system to initiate the development and maintenance of pain and/or cellular dysfunction. The nervous system is also involved in modulating the overall mechanical response of the facet joint and its tissues since the intensity and frequency of the mechanical stimuli experienced by these nerve endings also provide feedback to the central nervous system which is used to adjust the activity of the surrounding muscles and correct the loading of the joint in real-time [89–91].

3 Facet Joint Macromechanics

3.1 Facet Joint and Spinal Stability. The facet joints guide and constrain the motion of the vertebrae, while also facilitating the transmission of the loads applied to the spine [2,21,66,92]. The facet joints also contribute to and help maintain the stability of the spine. A structural column, like the spine, is considered mechanically stable when the sum of the forces and moments applied to it equals zero. Mechanical stability of the spine is achieved when the paraspinal musculature effectively counteracts the external loads via modification of the shape of the vertebral column. Clinically, the term 'spinal stability' has taken on the definition of the spine's ability to maintain its alignment and to provide protection to the neural structures it encloses during physiologic loading [93]. The clinical assessment of spinal stability/instability is required for a variety of different clinical scenarios, including degeneration with altered kyphosis or lordosis, surgical management or when motions become painful. The assessment is performed using imaging to measure the relative position of the vertebrae, and to detect any malalignment [93,94].

White and Panjabi [24] defined clinical instability of the spine as the spine's loss of ability to maintain its normal motions under physiologic loads which leads to initial or additional neurologic deficit [24]. Although most clinicians agree on the clinical definition of spinal instability, there is still ambiguity in using the term "spinal stability" because its quantitative assessment remains challenging and subjective in the clinical setting [93,95]. Currently, clinicians consider the spine as a three-column system in their assessment of spinal instability with the first column containing the anterior longitudinal ligament and the anterior half of the body and discs, the middle column contains the posterior half of the vertebral body and disc, and the posterior column contains the interspinous ligaments, spinous processes, pedicles, and the facets [93] (Fig. 1). The spine is considered unstable when two of the three columns are not intact. This rule is substantiated by the more complex system implemented by White and Panjabi [24] in which the spine is judged unstable when translations are greater than 3.5 mm and rotations greater than 20 degrees in the sagittal plane during flexion-extension bending [24]. Although injured or damaged facet joints do not a priori dictate that the spine is mechanically unstable, the proprioceptive and nociceptive nerve endings in the facet joint can respond to overload, damage, or injury to alter the musculature feedback and control for providing support to the spinal column. Moreover, injured nerves can also become nonresponsive to loading or motion or exhibit dysfunctional performance, both of which can result in abnormal sensory feedback for the central nervous system's coordination of the various spinal tissues and paraspinal muscles to insure mechanical stability [94].

3.2 Mechanical Contributions of the Facet Joint. The role of the facet joints in the mechanical stability of the spine has been established from biomechanical and mathematical studies. The facet joints prevent two adjacent vertebrae from engaging in relative motions that could overload and damage the surrounding spinal structures, such as the intervertebral disc, the nerve roots that exit the spinal column, and the spinal cord. Consequently, the facet joint tissues are themselves mechanically loaded. For example, Yang and King [2] reported that between 75–97% of the compressive load applied to the lumbar spine is borne by the intervertebral discs, and they estimated that 3–25% is carried by the posterior elements of the vertebral column in what they referred to as "facet force" [2]. In similar experiments using lumbar motion segments, Adams and Hutton [96] measured that under 2 degrees of extension and 560–1030 N of compression, 16% of the load is borne by the facet joints) [96]. Pal and Routal [4] assumed the spine to be mechanically equivalent to three columns; an anterior column composed of the vertebral bodies and discs, and two posterior columns consisting of vertically-connected articular processes. Those authors considered that any compressive load applied to the spine was distributed over the whole vertebral body and areas of the entire facet joints and that the ratio of the articular facet area to vertebral body area could be used as a metric of the load-sharing between the anterior and posterior columns [4]. Using an analysis of detailed facet joint morphology (facet articular area, vertebral body horizontal cross-section area, lordosis angle) Pal and Routal [4] computed that 23% of any axial compressive load is transmitted by the facet joints in the cervical and upper thoracic regions of the spine [4]. They reached the same conclusion in a matched study using the lower thoracic and lumbar regions of the spine, in which their anatomical observations and cross-sectional measurements of the vertebrae showed that the posterior vertebral elements

Straining of the fibers in the facet capsule not only results from vertebral motion but also from the activation of the muscles that can occur during mechanical loading of the spine as the outer surface of the capsular ligament is covered by the surrounding paraspinal muscles [110]. As such, the individual fibers of the capsule can become stretched when the muscles that insert on it contract [83,88,110]. In fact, muscle insertions have been found to cover nearly 23% of the capsule area in the cervical spine with a nonuniform spatial distribution [110], which can give rise to unequal capsular strains and stresses in all the regions (posterior, anterior, lateral) of the capsule when different muscles are activated to stabilize the spine during loading. An inhomogeneous mechanical loading environment (either in direction and/or magnitude) of the capsular fibers can also occur due to vertebral motion alone in the absence of any muscle activation. For instance, during flexion, the fibers of the posterior capsule region are stretched while the fibers in the anterior region of the capsule remain lax [8]; in contrast, during extension this pattern is reversed. Variation in strains in the capsule has also been observed in the facet capsule of the rat, in association with painful and nonpainful mechanical loading conditions imposed to the vertebral bones. Upon facet distractions that are considered to be physiologic, the strains reached $21 \pm 4\%$ in the posterior region, $17 \pm 4\%$ along the postero-lateral ridge, and $18 \pm 4\%$ in the lateral region of the C6-C7 facet capsule. Similar differences in strains of the capsule regions were also reported for painful facet joint distractions in that study [111]. Quantitative polarized light imaging was used to measure fibers kinematics during tensile loading of the capsule [111,112] and it was found that distractions of the joint that correspond to those producing pain also produced a significantly greater fiber dispersion in the posterior ($23.0 \pm 4.9°$) than in the lateral ($16.8 \pm 2.6°$) regions of the capsule [111]. Therefore, stretching of the capsular fibers in each region of the capsular ligament depends on the type of loading and on the extent of muscle insertion in that area.

3.5 Capsular Stretch and Neural Activity. Strains in the capsular ligament and stiffness have also been defined in animal models in which a distraction was imposed across the facet joint in the cervical spine in order to investigate pathomechanisms of facet-based pain [113–123]. In these approaches, an array of 25 to 35 miniature beads was placed on the exposed capsule to calculate strains during cervical distraction. Specifically, Quinn et al. [111] reported a maximum principal strain of 50% for a 700 μm subcatastrophic distraction of the rat C6-C7 facet joint that did not tear the capsule but did produce sustained behavioral hypersensitivity [111]. Kallakuri et al. [121] reported a strain of 73% at failure of the C5-C6 ligament that occurred between 12 and 30 mm tensile stretch in the goat [121]. The capsular strain of 50% measured in the rat model [111] is slightly larger than the 35% strain measured at the first sign of tissue rupture in the human capsule under shear [109] but compares well with the analogous measurement of 65% strain for the human capsule under tension [8]. Similarly, the tensile failure strain of 73% reported for the goat model [121] compares well with the failure strains of $94 \pm 85\%$ and $104 \pm 81\%$ reported by both Siegmund and Winkelstein for the human capsule [8,109] (Table 1). The similarity between the capsular strain values in the human and animal specimens may be a reflection of their similar mechanical function and composition.

When the capsule is stretched, the nerve afferents that innervate it are also stretched, which has been shown to trigger the generation of neuronal signaling to the central nervous system (CNS) in cases of noxious stretch [113,116,117,123]. Lu et al. [116] stretched the C5-C6 facet joint in a goat model and quantified capsule strains as well as the associated activation of afferents from the joint. They found that the capsule contained afferents that responded with firing at both low- and high-thresholds of strain (10% and 47%, respectively) and also that afferents of both types exhibited persistent generation of afterdischarge for up to 5 mins after the release of the applied strain (39–57%) that did not produce tissue rupture [116]. That work strongly implicated afferent injury in the capsule as a possible mechanism of pain because the afterdischarges were hypothesized as potentially having long-term effects in the CNS. Using a rodent model, Lee et al. [113] distracted the C6-C7 facet joint along the long-axis of the spine and measured a three-fold increase in behavioral hypersensitivity, as well as a significant sustained increase in astrocytic activation in the spinal cord in the absence of any ligament failure. Activated astrocytes modulate immune activation, neuronal synapses and play a role in pain signaling [113]. Using the same rodent model, we have found that after a high-rate facet joint distraction, expression of a glutamate receptor is also elevated in the spinal cord and positively correlated with both the degree of strain in the capsule and the amount of behavioral sensitivity [123]. Collectively, the integration of biomechanics with physiological and behavioral outcomes in these in vivo studies indicate that the loading environment of the afferents in the capsular tissue may be responsible for signaling injury and dysfunction (i.e., pain) in that tissue of the facet joint. In fact, from that combined work it has been suggested that the strain threshold for sustained painful capsular distraction may be between 20 and 47% [113,116,117,123,124].

3.6 Facetectomy Alters the Motion Segment Mechanical Response. Cusick et al. [100] reported that both unilateral and bilateral cervical facetectomies produced a loss of strength by as much as 32% and 53%, respectively. In those same cadaveric studies, rotations increased by 18% and joint distraction increased by 19% for application of combined compression-flexion [100]. Zdeblick et al. [102] showed that progressive bilateral facetectomy in multisegment cervical spine specimens subjected to 100 N of compression and 5 Nm of torsion significantly decreased torsional stiffness from 0.37 Nm/degree in an intact specimen to nearly half (0.18 Nm/degree) after a complete C5-C6 facetectomy [102]. When the specimens were subjected to 2 Nm of flexion they measured a 25% increase in the vertical distance between the C4 and C6 spinous processes after a 75% facetectomy, which was not significant but did show an increase in C4-C6 rotation [102]. Nowinski et al. [103] proceeded with a similar graded facetectomy procedure on C2-C7 segments after a C3-C6 laminoplasty had already been performed. Applying moments of up to 1.5 Nm about all three axes, they measured an increase of 7 degrees in sagittal rotation, 9 degrees in axial rotation and 3 degrees in lateral rotation [103]. They also measured an increase in translation but no significant change in coupled motion, after 25% or more facetectomy, which is in disagreement with the results reported by Raynor et al. [99].

In the lumbar spine, partial stepwise and total facetectomies also significantly increase rotation in flexion and axial rotation in motion segments loaded in compression (200 N) and subjected to 8 Nm about the three axes [101]. Tender et al. [104] resected the L5 pars interarticularis followed by a total unilateral facet removal on L5-S1 cadaveric motion segments subjected to 280 N of compression and 7.5 Nm of axial torsion. They found that the unilateral facetectomy significantly increased ipsilateral axial rotation by 1.4 degrees and overall axial ROM by 3 degrees. The increase in rotation, the loss of strength, and the decrease in stiffness in the spinal motion segment following facetectomy demonstrate that the facet joint contributes to spinal mechanical stability in a variety of directions and loading scenarios by limiting the linear and rotational motions during physiological loading [104]. The restriction of motion and the assurance of spinal stability provided by the facet joint stem from the biomechanical properties of the capsular ligament, articular cartilage, and bony pillars that together facilitate the functions of the joint as a whole.

3.7 Cartilage Mechanical Properties. Since the capsule provides support to help keep the facet joint intact during

Table 2 *Continued*

Level	Loading Details: Direction	Loading Details: Magnitude	Property		Technique	Reference
L5-S1	Axial compression	650 N	Force (N)		Pressure film between facet surfaces	[134][b]
	Shear	550 N				
	+			Group 1 Group 2		
	Flexion	6 deg		40 (±13) 45 (±10)		
	Extension			54 (±18) 65 (±18)		
	Lateral rotation – ipsi			50 (±13) 54 (±19)		
	Lateral rotation – contra			9 (±4) 33 (±10)		

Note: C-Cervical, T-Thoracic, L-Lumbar; FEM – Finite Element Model; SL-slideline model, CS-contact surface model, HE-hyperelastic model, FL-incompressibe fluid model of articular cartilage; ispsi – ipsilateral, contra – contralateral.
[a]canine
[b]reported here from non-tabular data of two separate test groups.

during spinal motion, that approach does require that the capsule be cut in order to insert the sensor in the joint. Capsule transection has been shown to contribute to hypermobility of the facet joint [105] and can be hypothesized as also potentially inducing non-physiological joint loads and/or articular contact, and in locations that are not usually loaded in an intact joint. Capsule transection does not likely affect the joint's behavior in extension since the capsule is not stretched and does not bear load during that direction of loading. But, force measurements in flexion, lateral bending, and axial rotation can be biased since the joint's overall mechanical behavior is modified by the capsule transection itself [105,139–141]. This could explain why the facet force values extrapolated from pressure sensor measurements in the study by Niosi et al. [136] were much smaller than those obtained from strain gauge measurements (Table 2). Furthermore, in any loading condition, the pattern and magnitude of contact are modified by the presence of the sensing device [142–144]. A similar, but less-invasive, method was developed by el-Bohy et al. [145] that maintains the integrity of the facet capsule. In that approach, a 1.5 mm-diameter pressure gauge implemented at the tip of a 13-gauge steel tube was positioned below the posterior bony tip of a lumbar inferior facet just above the cartilage covering the lamina of the vertebrae below [145]. Contact pressures of up to 0.3 MPa were measured at the edge of the articular surface of the lowest vertebra when a combined 600 N compression and 15 Nm flexion loading was applied to three-vertebrae lumbar segments. A comparable sparing-capsule technique was recently implemented in cadaveric cervical motion segments to determine average facet pressures of 10.3 ± 9.7 kPa and 67.6 ± 26.9 kPa for 2.7 Nm flexion and 2.4 Nm extension moments, respectively [144] (Fig. 3).

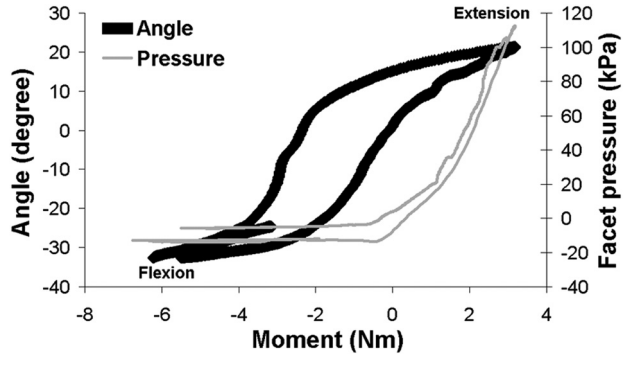

Fig. 3 Representative data quantifying the spinal rotations and pressure responses in the facet of a multisegment (C2-T1) cadaveric cervical spine during a range of bending moments applied in continuous flexion-extension. The pressure response in the C5-C6 facet joint increases with applied extension as contact is developed in the articulating facets, but exhibits a different pattern than the rotation angle. In contrast, during flexion, when the joint opens up there is no pressure detected.

4 Mechanotransduction

Since a portion of the spine's mechanical loading is supported by the facet joint, a variety of mechanical, physical, and chemical cascades are initiated in response to loading of the individual tissue components comprising the facet joint. These physiologic responses occur across several scales, ranging from the macroscopic tissue-level, to cellular and molecular levels via many mechanotransduction mechanisms. Although mechanotransduction can control and contribute to maintenance of the tissues in the joint [57,147–149], this process can also lead to and/or accelerate tissue degeneration and dysfunction [150,151]. The mechanisms of mechanotransduction in articular cartilage, ligaments, and bone have been described in other synovial joints. Broadly, as the first step the external primary spinal input (load or motion) is transformed into a secondary tissue-specific loading profile (Fig. 4). Then, the tissue-specific loads elicit a host of cascading mechanical, electrical, and chemical responses from the various elements that compose the tissue. These responses trigger further chemical changes that affect the intracellular milieu (protein translation, gene transcription, post-translational signaling) and the intercellular signaling (Fig. 4). Both the initial mechanical, electrical, and chemical changes and the modification of the intracellular milieu alter the intercellular signaling as well as the cellular activity (i.e., proliferation, differentiation, apoptosis). Modification of cellular activity can result in the release of chemical agents and electrical signals that influence the maintenance of the extracellular milieu, but can also alter the secondary tissue-specific loading (Fig. 4). Together, these physiological responses can modify the mechanical behavior of the tissue and lead to further changes in its response to mechanical loading and degeneration. Although this cascade has been well defined through a large body of elegant work, very few articles specifically address and detail these processes in the tissues of the facet joint. Therefore, this section reviews the mechanotransduction mechanisms known for the facet joint tissues and also provides a more global review of such mechanisms in similar tissues from other synovial joints.

4.1 Mechanotransduction in the Facet Joint and its Tissues

4.1.1 Capsular Ligament. Since spinal loading and motion are both guided and constrained by the facet joints the primary mechanical loading of the facet joint induces primarily capsular

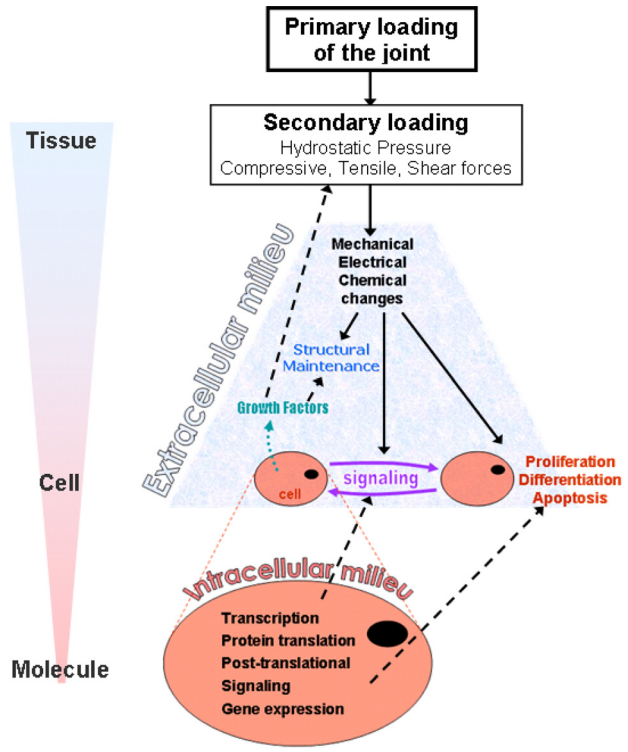

Fig. 4 Schematic representation of the generalized processes of mechanotransduction in synovial joints, across the scales ranging from tissue to molecule

ligament stretch and compression of the cartilaginous articular surfaces and the subchondral bone. In the capsular ligament under stretch, the collagen fiber structure and the nerve endings embedded in that network [152] and cells (fibroblasts, macrophages) are all distorted and activated [153]. Accordingly, capsular deformations of certain magnitudes can trigger a wide range of neuronal and inflammatory responses [124,154,155]. Neurophysiologic studies with a goat model have shown that the nerve endings in the capsule possess different stretch thresholds for activation [116]. Although most of the proprioceptive and nociceptive afferents have a low-strain threshold (~10%) for activation, a few receptors have a high-strain threshold (42%) for signal generation via neural discharge. In addition, capsular strains greater than 47% activate nociceptors with pain signals transmitted directly to the central nervous system [116]. Among both the low- and high-strain threshold neural receptors in the capsular ligament a few sustain their firing even after the stretching of the capsular ligament is released [116,154]. This persistent afterdischarge evident for strains above 45% constitutes a peripheral sensitization that may lead to central sensitization with long-term effects in some cases [154]. Also, in vivo stretch of goat cervical capsule until its rupture (up to ~30 mm) showed that the strains in the capsule averaged 73% and were sufficient to induce changes in axons taken as indicators of dysfunction (i.e., swelling, retraction beads, vacuolations). The effect of the capsule distraction on axonal changes was significant, with the ratio of abnormal to normal axons being greater in the stretched (94/186) than in unstretched capsules (29/108) [121]. Such axonal changes can be a source of hyperexcitability, spontaneous firing, and persistent pain [156] since that axonal dysfunction subsequently disrupts gene transcription of substance P, a neuropeptide protein involved in pain signaling [157]. In addition, inflammation in the facet joint also increases the discharge rate of multiunit nerves, sensitizes the nerves to mechanical stimulation, and activates previously inactive nerves [158].

The neural signals from the capsule travel via the primary afferents to the dorsal root ganglion (DRG) and spinal cord, and can induce several hallmarks of neuroinflammation, including glial activation [155] and cytokine upregulation [159,160]. These inflammatory responses have been reported after failure of the facet capsular ligament and also after its subfailure distraction in a rat model [161]. In response to the injurious stimuli, neuropeptides involved in pain signaling, such as substance P, are also modified. Substance P protein expression in the DRG after painful capsule distractions was twice that of nonpainful distractions of controls [115,124]. Although no gross damage of the capsule was observed after a painful distraction, spinal astrocytic activation was 61% greater and pain symptoms were also increased [113].

Capsular strains causing damage to the ligament structure can also activate fibroblasts directly or indirectly for structural repairs. While strains causing excessive failure of the collagenous ligament structure trigger an inflammatory-driven cellular response, subfailure strains elicit a fibroblast-mediated remodeling response to restore integrity to the damaged collagen structure [153]. Complete tissue tearing elicits an inflammatory response of the tissue that results in macrophage infiltration in order to clear any debris from the damaged collagen fibers and matrix. During the phagocytosis of the debris these cells release molecules that also trigger the recruitment of additional fibroblasts with increased collagen expression and this response can also lead to the formation of a provisional collagenous scar [153]. In the case of a subfailure loading scenario, no inflammatory response is observed and an increase in proteoglycans (decorin, fibromodulin) might actually help to modulate the fibrillogenesis of newly synthesized collagen by the fibroblasts [153].

4.1.2 Cartilage. Compression of the articular cartilage in the joint can occur during any mechanical loading of the facet joint [136]. Although compressive load is transferred via the facets between adjacent spinal levels and contact pressure develops in the facets' articular cartilage, contact is not uniform and the facet surface presents both load-bearing and nonload-bearing regions [162–165]. Given the difference in material properties between the various zones of the same tissue, the mechanisms by which mechanical signals modulate physiologic responses likely also lead to different spatial distributions of the responses in the affected tissues. However, the particular relationship between the mechanical, chemical, and cellular responses to compression in the cartilaginous matrix of the different zones remains largely unreported for the human spinal facet joint. Nevertheless, damage to the cartilage structure elicits an inflammatory response [43,166], which itself can also elicit not just osteoarthritis of the joint but can modulate pain signals from other regions of the joint. For example, one study showed that the inflammatory cytokines IL-6 and IL-1β were present in the facet cartilage retrieved from patients undergoing surgery for lumbar spinal canal stenosis and disc herniation [160]. This result led to the conclusion that pain symptoms might be due not only to mechanical tissue insults but also to chemical irritation of the tissue from the inflammatory agents leaking from the facet joint into the spinal space.

4.2 Mechanotransduction Processes in Articular Cartilage of other Synovial Joints. Mechanical stimuli elicit a cascade of multistep responses including mechanocoupling, mechanotransduction, intracellular conversion, and cellular response from articular cartilage (Fig. 4) [165]. These steps differ between the thick, proteoglycan-rich load-bearing areas and the mechanically weaker nonload-bearing areas of the articular cartilage layer because the extracellular environment (collagen fibers, proteoglycan and water content) varies along the depth of the cartilage layer (Fig. 2). These structural and compositional variations imply that the cellular responses to mechanical loading vary within each zone of the cartilage layer as well [46,167].

Tensile stresses that arise in the more superficial zone of the cartilage layer and hydrostatic pressure increases in the transitional and deep zones are converted at the tissue and cellular levels into electrical, chemical, and biomechanical stimuli [168]. Distortion of the

mechanisms permit the adaptation and maintenance of the bone structure to mechanical loading by acting on bone-regulating genes contained in the nucleus of bone cells leading to their proliferation, differentiation, and survival [148,187].

Mechanical loading of the facet capsule imparts structural changes to the collagen structure that can result in the degradation of its mechanical properties, loss of function, and pain-activating protein generation [88,112,155]. But ligaments also contain fibrogenic cells that are directly and indirectly affected by mechanical loading as chondrocytes in cartilage [188]. Fibroblasts deform when the tissue in which they are embedded deforms. Matyas et al. [189] observed that the nucleus of fibroblasts contained in the rabbit medial collateral ligament were 4 μm longer and 1 μm thinner when the ligament was under 6% tensile strain than at rest. Accordingly, the nuclear roundness decreased from 0.4 to 0.19 [189]. Upon deformation of the cell membrane, stretch-activated ion channels might be activated which permits the penetration and increase of cation concentration in the intracellular milieu and can eventually alter cellular activities as was described above for bone and cartilage cells. Studies of the periodontal ligament showed that mechanical stimuli also indirectly affect fibroblastic activity via trans-membrane and intracellular signaling as for osteoblasts. Disturbance of the ECM homeostasis leads to an intracellular conversion of the mechanical signal into a biochemical one via the transduction of focal adhesion molecules such as FAK and MAP-kinases in the fibroblast [190]. The cells then synthesize and release matrix metalloproteinase in the ECM for the regulation, modification, or degradation of ECM components [169], to modulate the mechanical loading state of the tissue. Similarly, loading of periodontal ligament fibroblasts was shown to activate various kinase proteins (ERK, JNK, p38) that communicate with the inner cellular milieu and activate AP-1 in the cells' nucleus. AP-1 can up-regulate the COL I gene in the nucleus of the fibroblasts, stimulating collagen expression by these cells [191]. Collagen expression is used to either maintain or repair the extracellular matrix.

Fibroblast activity and interaction with the extracellular environment is directly and indirectly affected by mechanical loading and tissue deformation. Such mechanotransduction mechanisms are similar to those described for cartilage and bone. Although the general mechanisms of mechanotransduction in bone, ligaments, and cartilage have been identified, as illustrated in Fig. 3, they remain to be elucidated more specifically for the facet joint tissues.

5 Injury and Trauma of the Facet and Other Synovial Joints

Facet joint injuries result most-often from motor vehicle and sports trauma, such as skiing, snowboarding, cycling, and diving [82,192–195], and include a wide range of bony and ligamentous lesions depending on the extent and type of tissue trauma. Interestingly, unilateral and bilateral facet injuries make up nearly 6% of all cervical injuries, with undisplaced fractures, subluxations, and dislocations being the most commonly reported facet injuries [192,193,195,196]. Facet injuries often directly damage the hard and soft tissues that compose that joint (Fig. 2) [8,197–200]. In addition, facet trauma is also associated with the occurrence of damage to other soft tissues of the spine, such as disc tearing, spinal cord trauma, and/or nerve root compression, all of which can also lead to a transient or even permanent loss of mechanical and neurological function of the facet joint, spinal column and/or physiologic sequelae [193,201–206].

5.1 Facet Joint Injuries. Because the facet joints comprise the integrative biomechanical structure of the spine, any violation of their mechanical integrity as can be caused by injury or trauma directly affects the mechanical behavior of a motion segment or even the overall spinal region [195,206]. For example, a unilateral locked facet at C5-C6 produced by combined lateral bending and flexion of the cervical spine has been reported to significantly reduce the segmental range of motion (ROM) by 2.7 – 3.6 degrees in all modes of loading except in ipsilateral axial rotation [206]. Once unlocked, the ROM of the C5-C6 motion segment was further increased compared to its preinjury intact condition by an additional 3.5 – 8.0 deg. This report, suggests that the capsular ligament had been damaged due to the facet injury. Indeed, Crawford et al. [206] also reported that the laxity of the capsular ligament was significantly increased after that locked facet condition compared to that in the intact condition. Also, the increase in laxity was associated with some ligament tearing, supporting both the hypothesis that the ligament sustained damage and exhibited altered mechanical properties [206]. Taken with reports of instability following facet dislocation [193,201,207], these findings imply that there may be a continuum between the degree of instability and trauma to the facet, with greater instability for more severe facet trauma, including dislocation and the more-extreme fracture.

In a clinical study of patients with cervical unilateral lateral mass facet fractures, Lee and Sung (2009) found that the degree of axial rotation and the segmental kyphosis were significantly greater in those patients whose facet was both fractured and dislocated than in those sustaining only a facet fracture [195]. Despite these differences, both types of injury were associated with instability for the cervical spine in rotation; surgical treatment was required to sufficiently restore stability, again demonstrating the role of the facet joint in limiting spinal motions, in particular rotation. Also, unilateral fracture of the facet joint has been shown to lead to spondylolisthesis, an anterior translation of the superior vertebra, sometimes associated with an axial rotation of the superior vertebra around the intact contralateral mass [195,208]. Such a fracture injury can lead to a variety of neurological disorders since the motion segment is unstable and can compress the spinal cord and/or nerve roots during certain motions. In these cases there is also the potential for capsule injury when the fractured vertebra exhibits abnormal kinematics during physiologic motions that can also impose nonphysiologic compressive stresses on either or both the capsule and cartilage of the contralateral facet joint [208]. In the same way, excision of the capsule and cartilage during a surgical procedure has been shown to increase the sagittal and axial ranges of motion by 38% and 57%, respectively [209]. Most simply, fractures of the articular pillar or lateral mass impose an overt disruption of the facet joint's mechanical properties since they eliminate the joint's ability to support any load and, in so doing, can cause spinal instability and neurological impairment.

5.2 Surgical Treatments of Facet Injuries and Effects on Facet Biomechanics. Fractures of the bones of the facet joint leading to joint separation, comminution, split, and traumatic spondylolysis, require surgical treatment to reduce the anterior translation and axial rotational deformity associated with these injuries [195,210]. However, the type of treatment varies with the type and severity of the fracture [210]. A separation fracture that isolates the entire lateral mass can be treated with a pedicle screw that provides stability and strength while also encouraging bone growth [210]. However, if the separation fracture is also associated with disc and/or ligamentous damage, a one-level reduction and stabilization is recommended to avoid any slippage of the vertebra; fractures which can also result in the development of multiple bone fragmentations and traumatic spondylolisthesis also require only a single-level stabilization to treat the unstable anterior translation of the superior vertebra. Split and severe fractures have been shown to be successfully treated with two-level posterior fixation that resolves both the spinal instability and restores the spinal alignment [210]. In the most severe cases, the articular surface of the facet joint can become completely obliterated and the articulation so disrupted that the constraining and guiding functions of the facet joint cannot be restored; in that case, fusion is necessary.

Surgical fusion can relieve many of the physiologic symptoms caused by facet fracture but the mechanical function of the joint is not fully returned to normal. In fact, the normal range and pattern