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The Impact of Spine Pathology on Posterior Ligamentous Complex Structure and Function

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Abstract

Purpose of Review Spinal ligament is an important component of the spinal column in mitigating biomechanical stress. Particularly the posterior ligamentous complex, which is composed of the ligamentum flavum, interspinous, and supraspinous ligaments. However, research characterizing the biomechanics and role of ligament health in spinal pathology and clinical context are scarce. This article provides a comprehensive review of the implications of spinal pathology on the structure, function, and biomechanical properties of the posterior ligamentous complex.

Recent Findings Current research characterizing biomechanical properties of the posterior ligamentous complex is primarily composed of cadaveric studies and finite element modeling, and more recently incorporating patient-specific anatomy into finite element models. The ultimate goal of current research is to understand the relative contributions of these ligamentous structures in healthy and pathological spine, and whether preserving ligaments may play an important role in spinal surgical techniques.

Summary At baseline, posterior ligamentous complex structures account for 30–40% of spinal stability, which is highly dependent on the intrinsic biomechanical properties of each ligament. Biomechanics vary widely with pathology and following rigid surgical fixation techniques and are generally maladaptive. Often secondary to morphological changes in the setting of spinal pathology, but morphological changes in ligament may also serve as a primary pathology. Biomechanical maladaptations of the spinal ligament adversely influence overall spinal column integrity and ultimately predispose to increased risk for surgical failure and poor clinical outcomes. Future research is needed, particularly in living subjects, to better characterize adaptations in ligaments that can provide targets for improved treatment of spinal pathology.

Keywords Posterior ligamentous complex · Spinal ligament · Biomechanics · Pathology · Spine surgery · Spine

Introduction

Spinal ligaments represent an important component of the spinal column involved in neural control, dynamic stability, and protection of anatomic structures of the spine [1••]. Spinal ligament dysfunction is hypothesized to play a role in various pathologies, such as segmental instability, adult spinal deformity (ASD), proximal junctional kyphosis (PJK), and failure (PJF) following instrumented fusion, low back pain, and other degenerative conditions. It is even thought that repeated sub-failure injury of the ligament may lead to spinal muscle dysfunction, resulting in a vicious loop of instability and injury, proposed to be a mechanism of nonspecific chronic lower back pain [1••]. Healthy ligaments may also play a role in reducing complications following instrumented spinal fusion, such as vertebral fractures, subluxation, degenerative disc disease, implant failure, facet joint disruption, and PJK/PJF [2–5].

Particularly, the spinal ligament is hypothesized to play an important role in mitigating biomechanical stress especially following fusion where there is an abrupt transition from rigid implants to native soft tissues at the upper instrumented vertebrae (UIV) and the level above (UIV + 1). Ligament augmentation techniques employed in an attempt to reduce risks of complication are becoming more popular, as they are thought to provide a more gradual transition between rigid implant and soft tissue, and additionally are thought to help replace ligamentous structures that may be

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resected during spine surgery [6, 7•, 8•, 9–12, 13••, 14, 15]. Other techniques elect to use more dynamic implants, often termed non-fusion devices, or combinations of fusion with a non-fusion structure to top off the fusion construct, which is likewise thought to provide less rigid stabilization to the spinal column [6, 7•, 8•, 9–12, 13••, 14, 15]. We do know that muscle [16] and bone [17] health are important predictors of fusion outcomes, particularly in ASD [2, 18]. However, it is unclear what role ligament health plays in other pathology, if any [19]. Research characterizing the biomechanics and role of ligament health in spinal pathology and in a clinical context are scarce [19]. Thus, the purpose of this paper is to give an overview of the current literature and future directions for research.

Structure

The posterior ligamentous complex (PLC) is a group of three spinal ligaments thought to be especially pertinent in the thoracolumbar spine and in reducing the risk of PJK/PJF [2, 20••]. These three ligaments, from posterior to anterior, are the supraspinous ligament (SSL), the interspinous ligament (ISL), and the ligamentum flavum (LF), as shown in Fig. 1.

The LF arises from the anterior surface of the superior lamina and extends caudally from C2 to S1 and is classically described as having two layers, superficial and deep [21], while more recent histological studies suggest that the LF is one continuous layer [22]. The LF is composed of 80% elastin surrounded by about 20% loose and disorganized type III collagen with interfibrillar proteoglycan oriented cranio-caudally, which transition to orient more parallel to the spinous processes as they extend dorsally and become confluent with the ISL [23–25].

In contrast, the ISL traverses between each vertebral level from C1 to S1 where ventrally, its fibers are confluent with the LF and insert at the facet joints, and dorsally its fibers attach to the inferior spinous process and are confluent with the SSL [22, 26]. Fibers of the ISL are oriented parallel to the spinous processes, ventrally containing a higher density of elastin due to integration of the LF and centrally and dorsally primarily composed of type III collagen in a crimped pattern with interfibrillar proteoglycan [23–25, 27].

The SSL is the most posterior, beginning at C7 and extending caudally superficial to the spinous processes to L4 [28]. It is primarily composed of loose type III collagen and interfibrillar proteoglycan oriented cranio-caudally perpendicular to the vertebral column, with some studies suggesting a higher ratio of adipose tissue as compared to the other spinal ligaments, which would make it less resistant to biomechanical loads [23–25, 27], although it is also believed to serve as anchorage for the erector spinae tendons [29].

Function

Functions of the PLC are likewise not very well characterized. What we do know is that the thoracolumbar range of motion (ROM) is highly coordinated via the coupling of movement at individual spinal levels between the thoracic and lumbar spine, where each functional spinal unit contributes a small portion to overall ROM [30, 31••]. As such, disruptions of osteoligamentous stabilizers like the PLC at a given functional spinal unit (FSU) will have consequences not only in that spinal segment but in overall spinal function

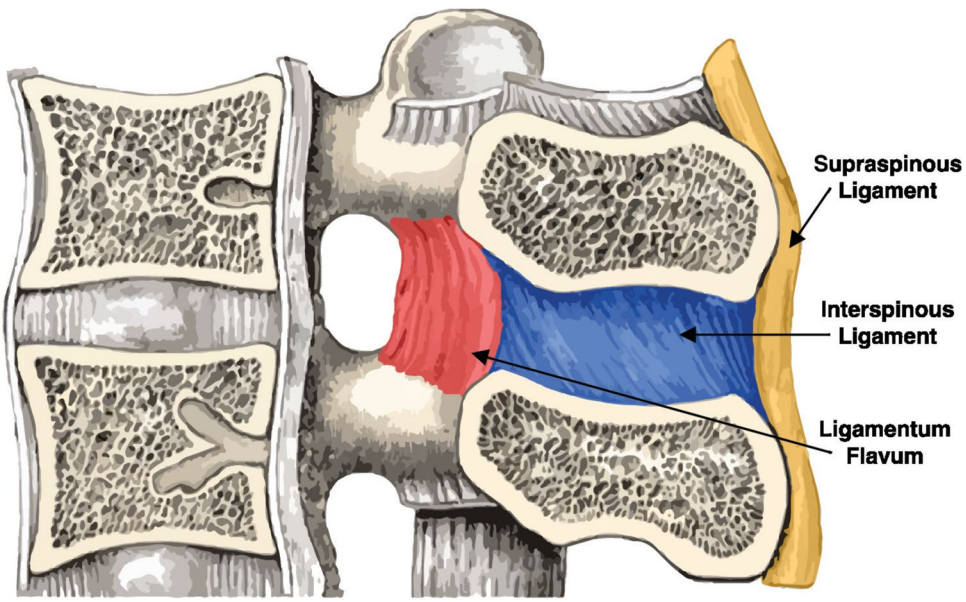

Fig. 1 Diagram depicting a midline sagittal cut of vertebrae with intact ligamentous structures. Ligaments of interest are outlined. The ligamentum flavum (red) forms the posterior wall of the vertebral column with the laminae. Just posterior, the interspinous ligament (blue) spans between adjacent spinous processes, while the supraspinous ligament (yellow) traverses over the most superior aspect of the spinous processes longitudinally

as well [31••]. Anatomically, the PLC sits at the posterior spinal column and is thus important in flexion [32•]. Beyond passive roles, the ligaments of the PLC are highly innervated which suggests a role in the spinal control system, in proprioception and posture, and as a potential pain generator [33].

Biomechanically, the PLC behaves similarly to other human ligaments, where its physical properties are influenced by temperature, time, and loading rate. Higher loading rates result in stiffer-load displacement, and relaxation rates dependent on the initial amount of stretch [34–37]. Uniquely, the PLC follows a non-linear load–displacement curve in both the thoracic and lumbar spine and is stiffer in flexion than in extension [38, 39, 40••, 41]. Collectively, resection of all three ligaments results in a loss of over 25% of passive stability to the human lumbar spine in flexion [42]. Cutting the confluent fibers between ligaments has been shown to reduce the stability and resistance to flexion of the PLC by up to 40% [40••].

Importantly, data in the literature on the tensile properties of ligaments is difficult to interpret due to differences in resection and testing methodology (i.e., strain rate, loading, humidity, FSUs vs isolated ligaments), ligament type (i.e., living subjects vs. cadavers vs. animal models), age, gender, weight, height, whether or not the samples come from a pathological source, and whether or not the authors reported the data in the same units or measures. The parameters reported below are peak force (a measure of ligament strength) which also coincides with force at ligament failure (rupture), tensile stress (a measure of internal force per unit area), tensile strain (a measure of deformation relative to original length), and elastic modulus (a measure of stiffness). Many studies do not report in the same units or parameters, with many describing stiffness in terms of a spring constant (N/mm). So, when possible and if necessary, units were recalculated, or raw data were extracted and used to calculate the aforementioned parameters for comparison (Tables 1, 2, 3 and 4). It should be noted that many discrepancies in ligament testing methodology exist [43], and as such, comparisons between biomechanical parameters from differing studies should be carefully evaluated in context.

The Ligamentum Flavum

In human lumbar FSUs carried through physiologic ROM, the LF accounts for roughly 22% of overall resistance to flexion, while application of smaller loads in flexion places an increasing load on the LF [42]. Additionally, the LF is subject to the highest strain of PLC ligaments in lateral bending [32•]. Resection studies have demonstrated that the LF is the most restrictive ligament of the posterior column in

Table 1 Tensile properties of human ligamentum flavum derived from isolated bone ligament bone complexes or from functional spinal units

Author	Type	*N*	Level	Stress (N/mm^2)	Strain (mm/mm)	Elastic modulus (N/mm^2)	Peak force (*N*)
Nachemson and Evans [44]	Iso; C	10	L3/4	4.3 ± 3.6	0.5 ± 0.2	9.9 ± 8.8	–
Adams et al. [45]	FSU; C	27	L1-S1	1.9 ± 1.5	0.3 ± 0.1	11.7 ± 8.3	216.1 ± 214.2
Panjabi et al. [32•]	FSU; C	–	Lumbar	–	–	–	150–200[a]
Chazal et al. [39]	Iso; C	7	T4-L4	15.3 ± 5.0	0.2 ± 0.04	76.3 ± 30.0	414.3 ± 69.5
Dumas et al. [40••]	FSU; C	25	T11-L5	–	–	–	170[a]
Pintar et al. [46•]	FSU; C	132	T12-S1	3.0 ± 1.0	0.71 ± 0.2	4.3 ± 1.3	–
Mihara et al. [47]	Iso; LS	42	Lumbar	–	–	4.4 ± 1.6	–

Iso, isolated ligament specimens; *FSU*, functional spinal unit specimens; *C*, cadaveric origin; *LS*, living subject origin

[a]Unable to calculate but data provided by authors via text or graphical interpretation

Table 2 Tensile properties of human interspinous ligament derived from isolated bone ligament bone complexes or from functional spinal units

Author	Type	*N*	Level	Stress (N/mm^2)	Strain (mm/mm)	Elastic modulus (N/mm^2)	Peak force (*N*)
Panjabi et al. [32•]	FSU; C	–	Lumbar	–	–	–	50–100[a]
Myklebust et al. [53]	Iso; C	41	T1-S1	–	–	–	83.4 ± 47.3
Pintar et al. [46•]	FSU; C	132	T12-S1	3.5 ± 1.9	0.8 ± 0.3	5.0 ± 3.3	100[†]
Dickey et al. [54]	Iso; C	–	Lumbar	–	–	–	45[†]
Iwanaga et al. [26]	Iso; C	17	L1-L4	–	–	–	109.0 ± 46.3[a]

Iso, isolated ligament specimens; *FSU*, functional spinal unit specimens; *C*, cadaveric origin; *LS*, living subject origin

[a]Unable to calculate but data provided by authors via text or graphical interpretation

Table 3 Tensile properties of human supraspinous ligament derived from isolated bone ligament bone complexes or from functional spinal units

Author	Type	*N*	Level	Stress (N/mm^2)	Strain (mm/mm)	Elastic modulus (N/mm^2)	Peak force (*N*)
Panjabi et al. [32•]	FSU; C	–	Lumbar	–	–	–	50–100[a]
Myklebust et al. [53]	Iso; C	41	T2-S1	–	–	–	309.3 ± 205.1
Pintar et al. [46•]	FSU; C	132	T12-S1	12.3 ± 2.5	0.9 ± 0.2	13.4 ± 2.7	300[a]

Iso, isolated ligament specimens; *FSU*, functional spinal unit specimens; *C*, cadaveric origin; *LS*, living subject origin

[a]Unable to calculate but data provided by authors via text or graphical interpretation

Table 4 Tensile properties of human Interspinous and Supraspinous Ligament together derived from isolated bone ligament bone complexes or from functional spinal units

Author	Type	*N*	Level	Stress (N/mm^2)	Strain (mm/mm)	Elastic modulus (N/mm^2)	Peak force (*N*)
[b]Adams et al. [45]	FSU; C	27	L1-S1	1.0 ± 0.6	0.3 ± 0.1	3.7 ± 2.9	159.8 ± 95.4
[b]Chazal et al. [39]	Iso; C/LS	5/9	T1-S1	8.6 ± 3.0	0.4 ± 0.1	25.7 ± 13.3	183.2 ± 89.9
[b]Dumas et al. [40••]	FSU; C	25	T11-L5	–	–	–	65–82[a]
[b]Hindle et al. [55]	FSU; C	13	L3/4	–	–	–	65.2 ± 24.1
[b]Iida et al. [56•]	Iso; LS	24	Lumbar	1.2 ± 0.6[a]	–	3.3 ± 2.1[a]	203.0 ± 102.9[a]

Iso, isolated ligament specimens; *FSU*, functional spinal unit specimens; *C*, cadaveric origin; *LS*, living subject origin

[a]Unable to calculate but data provided by authors via text or graphical interpretation

[b]ISL and SSL were studied as one ligamentous complex

ROM at the thoracolumbar junction, particularly, resection of the LF results in significant increases in flexion at this level [40••]. Furthermore, the LF has been shown to pre-stress the intervertebral discs (IVDs) ranging from about 15 N in younger patients to about 4 N in elderly patients [44]. This is believed to counteract the internal swelling pressure of the disc and provide some intrinsic stability to the upright spine [44].

Tensile Properties of the Ligamentum Flavum

Tensile properties of human LF are reported in Table 1. There are roughly equal numbers of FSU and isolated ligament studies, while all except one used cadaveric ligament. Maximum stress (tensile strength) ranged from a mean of 1.9 ± 1.5–15.3 ± 5.0 N/mm^2 from Adams et al. and Chazal et al., respectively [39, 45]. Maximum strain varied among all studies, with a maximum mean deformation between 20 and 70% of resting LF length. Elastic modulus ranged from a mean of 4.3 ± 1.3–76.3 ± 30.0 N/mm^2 [39, 46•]. Peak force ranged from 216.1 ± 214.2–414.3 ± 69.5 N. Differences in methodology may help explain the wide range of numbers seen for some measures. Chazal et al. used bone ligament bone (BLB) complexes of the LF and laminae alone, while Adams et al. used FSUs with progressive disruption of ligaments from posterior to anterior [39, 45]. To our knowledge, only one study from Mihara et al. has evaluated LF derived from living subjects undergoing decompression surgery for mainly lumbar spinal stenosis and disc herniations but did not report on all measures [47].

The Interspinous and Supraspinous Ligaments

Together the SSL and ISL are often studied as one entity [39, 45, 48] because it is difficult to anatomically differentiate the two ligaments [28] and they share many of the same functional characteristics. The ISL and SSL together contribute about 6% of overall resistance to flexion in human lumbar FSUs carried through physiologic ROM [42]. In another study of thoracic FSUs, resection of the ISL and SSL results in an approximate loss of 6.6% flexion stiffness [49] suggesting shared roles in both thoracic and lumbar flexion. While moving toward extremes of flexion subjects the ISL and SSL to the highest strains of any spinal ligament [32•, 42, 50]. Changes in ROM following ISL and SSL resection in human studies have shown inconsistent results. Some demonstrate no significant change in ROM [19]; others show increased flexion [51]; and some detail no significant change in ROM with resection of the ISL yet significant increases in flexion with resection of the SSL [52]. Nonetheless, the relationship between the thoracolumbar fascia and the ISL is believed to make it important in lifting motions and in providing anchorage of paraspinal muscles to vertebrae [29].

Tensile Properties of the Interspinous Ligament

Tensile properties of human ISL alone are summarized in Table 2. All reports on biomechanical properties of the ISL are derived from cadaveric samples with a focus on the thoracolumbar spine. Studies evaluating biomechanics of isolated ligament versus FSUs were roughly equivalent in prevalence. Importantly, the only study which reported all biomechanical properties evaluated FSUs and not isolated ligament [46•]. Peak force ranged from 83.4 ± 47.3–109.0 ± 46.3 N. Maximum stress, strain, and elastic modulus were only reported by one group. Maximum stress of the ISL was a mean of 3.5 ± 1.9; maximum strain was a mean of 80%, and elastic modulus was a mean of 5.0 ± 3.3 N/mm^2. From our review, no study has evaluated the full array of tensile properties in the isolated ISL, marking a need for further research.

Tensile Properties of the Supraspinous Ligament

Tensile properties of human SSL alone are summarized in Table 3. To our knowledge, very few have evaluated the SSL alone, and like the ISL, they are majorly derived from cadaveric samples with a focus on the thoracolumbar spine. There were roughly equal numbers of studies evaluating isolated ligament versus FSUs, with one reporting all biomechanical properties based on FSUs [46•]. Based on the data however, the SSL was stiffer, more deformable, and able to resist greater peak forces than the ISL [46•], despite its greater adiposity compared to the ISL [23–25, 27]. Maximum stress of the SSL was a mean of 12.3 ± 2.5 N/mm^2; maximum strain was a mean of 90%; and elastic modulus was a mean of 13.4 ± 2.7 N/mm^2. From our review, none have evaluated the full array of tensile properties exhibited by the isolated SSL, marking a need for further research.

Tensile Properties of the Interspinous-Supraspinous Ligament Complex

Tensile properties derived from the ISL and SSL together as one ligamentous complex are summarized in Table 4. There are roughly equal numbers of isolated ligament versus FSU studies, with a focus on the thoracolumbar spine. Of those that reported on all biomechanical properties, two tested isolated ligaments and one tested FSUs, and of those, two evaluated ligaments from living subjects and two evaluated ligaments derived from cadaveric specimens. Peak force ranged from a mean of 65.2 ± 24.1–203.0 ± 102.9 N. Maximum stress ranged from a mean of 1.0 ± 0.6–8.6 ± 3.0 N/mm^2. Maximum strain ranged from a mean of 30–40%, considerably less than those values reported for the ISL and SSL separately. Finally, elastic modulus ranged from a mean of 3.3 ± 2.1–25.7 ± 13.3 N/mm^2. ISL and SSL have only been evaluated from living subjects as one combined ligamentous complex, as such, there is obviously a need for further research in this area.

Finite element analyses

Finite element analyses (FEAs) are a popular tool for modeling the biomechanics of the spine. These models can be studied without the need for physical samples; however, they do rely on prior animal or human studies to inform biomechanical parameters of various tissues to generate accurate models. Importantly, these parameters often differ across models and research groups and there is no real consensus [57•, 58, 59]. Importantly, as seen above, many of the studies that inform FEA model parameters are derived from cadaveric samples and may or may not have application in living subjects. The contribution of the PLC to spinal stability using FEA has been explored in several studies, with varying assumptions and analytical methodologies. Zander et al. reported that the biomechanical properties of ligament were more important predictors of function when compared to bone, particularly when considering the extremes of ligament strain (i.e., 30° flexion for the PLC structures) or considering higher initial loading (i.e., body weight or applied load). However, disc and facet joint morphology were found to be most important—albeit highly patient-specific [60••].

Because of the influence of inter-individual anatomical variability on FEA outcomes, recent studies have incorporated CT imaging datasets to accommodate patient-specific morphology. For example, Naserkhaki et al. modeled hypo-lordotic, normo-lordotic, and hyper-lordotic spines using CT datasets [61•]. While load sharing remained the same across all spines, the SSL and ISL exerted higher resistance to flexion in the hyper-lordotic spine compared to the normo-lordotic spine, and lower resistance to flexion in the hypo-lordotic spine. Despite the recent use of CT datasets, most FEA studies are historically non-CT derived and generally show adequate concordance with prior animal and cadaveric studies from which they are modeled. Findings from this body of literature suggest that PLC ligaments contribute to overall spinal stability in a level-specific manner, and these contributions vary by ligament and by physiologic movement evaluated. Additionally, a common thread among FEA studies is that higher ligament stiffness predisposes ligament to premature rupture and places increasing biomechanical loads on adjacent bony and fibromuscular structures [62–64, 65•, 66•]. Likewise, decreasing ligament stiffness seems to place preferentially greater loads on adjacent muscular structures [67]. Across the thoracolumbar spine, the PLC is estimated to contribute approximately 30–40% of spinal stability which is suggested as primarily driven by the SSL [62–64, 65•, 66•, 67, 68].

Pathology

Many theories exist to explain the interaction between ligament physiology and various pathological states. Repeated sub-failure stretching of spinal ligaments, increasing age, and concomitant non-ligamentous degenerative pathologies have all been associated with decreased ligament stiffness [56•, 69], while the association with bone mineral density is unclear [70]. Importantly, posterior spinal instrumentation and fusion have been associated with decreased PLC stiffness and tensile strength in a sheep model, which is thought to be due to stress shielding of ligament and adjacent muscle [71•]. Below, we aim to characterize physiological adaptations (or maladaptations) of ligament in the presence of some of the most common spinal pathologies.

Spinal Pathologies of Ligamentous Origin

Ligament hypertrophy is a common cause of spinal stenosis especially in the LF and can result in neural impingement and pain [72]. This process is thought to be driven by the ossification of the ligament in cases of high mechanical or other stressors [72]. Hypertrophic ossification of the LF involves replacement of fibroblasts with chondrocytes, necrosis, and alterations in collagen-proteoglycan content and structure [72]. The overall pathophysiology of these changes in LF is well studied as LF hypertrophy is a common condition which may result in functional and neurologic deficits [72]. It is considered a cytokine-mediated process as key players include transforming growth factor beta (TGF-B), bone morphogenic protein (BMP), and alkaloid phosphatase (ALP) [73]. Mechanical stress is regarded as one of the main etiologies of LF hypertrophy, which has been shown in both animal and human models, ultimately resulting in higher ligament stiffness [74•, 75]. Although there is a paucity of literature regarding primary hypertrophy of the SSL and ISL, morphological and functional similarities between the LF, SSL, and ISL support the idea that all three ligaments may undergo similar pathologic changes in response to mechanical stress [22, 23, 76].

Influence of (Non-ligamentous) Degenerative Pathology on Spinal Ligament

There are strong associations between LF thickening in cadaveric samples of lumbar and thoracic spinal segments with IVD degeneration and facet joint osteoarthritis [77–79]. Thickening of the LF was not independently associated with changes in biomechanical properties but tends to be observed at the L4-5 level and ipsilateral to the side of major pathology [79]. Those same reports and others have found that the ISL and SSL also undergo reductions in ultimate strength and stiffness in response to age-related and non-age-related IVD degeneration [78], facet joint osteoarthritis [56•], and aging [56•]. Other biomechanical studies found that the ISL becomes more functionally important in flexion, taking on higher strain nearly equal to the SSL as compared to non-degenerated lumbar spine; possibly due to anteriorly translated internal axes of rotation (IAR) [32•]. A similar experiment using cadaveric samples under close to physiologic conditions (loading, 100% humidity, body temperature) also found significantly higher spinal segment stiffness at levels with degenerated discs in axial rotation and lateral bending; however, they did not directly measure the ligament elastic modulus [80]. Like the LF, SSL and ISL from degenerative spines show evidence of secondary hypertrophic ossification involving replacement of fibroblasts with chondrocytes, necrosis, and alterations in collagen-proteoglycan content and structure, suggesting that the ISL and SSL are subject to dynamic morphological changes like the LF, in the context of degenerative spine pathology [23, 72, 76].

Scoliotic and lordotic spines also appear to place abnormal stresses on spinal ligaments; however, how this influences ligament tensile properties is not well understood. Prior studies have demonstrated that hyper-lordosis recruits the SSL and ISL to a higher degree particularly in flexion as compared to normo-lordosis, while hypo-lordosis recruits the SSL and ISL to a lesser degree in flexion [61•]. Scoliotic T7-8 FSUs modeled by Little and Adam in 2011 demonstrated abnormal ligament recruitment during a physiologic range of motion. However, it is unclear how this translates into living subjects, as researchers only modeled a single FSU level [81]. Prior biochemical studies have demonstrated no noticeable differences in composition between normal and scoliotic spine ligament [25], yet others have shown biomechanical differences such as increased ligament stiffness of the SSL and ISL in cases of idiopathic scoliotic, when compared to the broader literature [48].

The Impact of Surgical Intervention on Ligament Health

Literature provides a strong hypothesis that the PLC ligaments become stiffer in response to stress. However, surgical intervention, such as incorporation of rigid surgical constructs during spinal fusion, or partial ligamentous resection during a posterior surgical approach may result in significant unloading or non-physiological mechanical loads. In 1998, using a sheep model of bilateral facetectomy and anterior L4-5 discectomy, Kotani et al. found that spinal fixation with transpedicular screws and plates compared to a sham control group resulted in decreased ultimate load and elastic modulus, and histological and morphological changes at the level of operation, which were most pronounced in the posterior

ligaments [71•]. To our knowledge, no other study has evaluated ligament health in this way in the context of spinal fixation. However, animal models of spinal decompression surgery have supported the notion that resection of the ISL during surgery reduces adjacent segment stability and results in increased intervertebral motion [82••, 83••]. In response to these findings, more recent surgical techniques often include the preservation or augmentation of ligament in an effort to prevent degenerative changes and maintain spinal stability post-operatively. One report of posterior pedicle screw fixation from T2-7 in sheep compared three groups, a control with all posterior spinal structures protected, an experimental group with the ISL and SSL completely resected, and an experimental group with the facet joints resected at UIV + 1 [84••]. Their findings suggest that protecting the SSL and ISL during a posterior surgical approach may be the most important factor in reducing PJK risk following instrumentation [84••]. However, this idea is under contention following recent surgical advances incorporating ligament augmentation techniques which show inconsistent results, warranting further study [6, 12, 19, 85•].

Conclusions

Spinal ligaments are essential in the functioning of the spinal column and are involved in stability, neural control, and protection of spinal structures. The PLC is thought to be the most clinically relevant subset of spinal ligaments in the thoracic and lumbar spine, composed of the SSL, ISL, and LF. Together, these ligaments are reported to account for approximately 30–40% of spinal stability and can be impaired in the presence of spinal pathologies. Ligamentous adaptation in the presence of pathologies such as spinal stenosis, osteoarthritis, deformity, and surgical intervention includes hypertrophy and reduction of tensile strength and stiffness, further affecting the capacity for stabilizing the spine. As a result, more recent surgical techniques often include the preservation or augmentation of ligament in an effort to prevent degenerative changes and maintain spinal stability post-operatively. However, the effectiveness of these techniques is still under contention and requires further study. Further research should focus on clarifying the differences in tensile properties and morphology of the PLC, especially from living subjects. Understanding the interaction between spinal pathology and ligamentous properties will highlight targets for potential therapeutic interventions. Likewise, future research may look to understand how ligament properties can be modified surgically or non-surgically to improve patient care and outcomes. Overall, appreciating the role PLC ligaments play in maintaining a healthy and stable spine is crucial in improving our understanding, recognition, and treatment of spinal conditions in this patient population.

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Declarations

Conflict of Interest Bradley Anderson and Bahar Shahidi declare no conflicts of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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