

THE POSTERIOR LAYER OF LUMBAR FASCIA AS A POTENTIAL SOURCE OF LOW BACK PAIN

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Abstract: The posterior layer of the lumbar fascia is innervated by unmyelinated nerve endings which have a nociceptive capacity. Although indications about tissue injuries in this tissue have been documented, it is not clear to which degree these contribute to the frequent finding of idiopathic low back pain in modern humans. In vivo studies on low back pain patients revealed a reduced shear-motion, which suggests the involvement of an altered fascial architecture in this pathology. In addition, in vivo studies with experimental elicitation of back pain via stimulation of this fascia layer suggest an increased sensitivity of dorsal horn neurons for potential nociceptive stimulation of lumbar fascia. Taken together these findings suggest the posterior layer of the lumbar fascia could possibly be a frequent source of low back pain. However, further studies are needed for a more detailed understanding.

Background: It is generally accepted that disc pathologies account only for a minority of low back cases as a causal factor (Jensen et al. 1994). The majority of low back pain cases are idiopathic. Based on this background Panjabi (2006) proposed that microinjuries in lumbar connective tissues may result in impaired function of embedded proprioceptive mechanoreceptors, which then leads to muscle control dysfunction and subsequent biomechanical impairments. Although this model considered paraspinal connective tissues only, other authors suggested that posterior layer of the lumbar fascia should also be involved as a candidate for similar microinjuries (Schleip et al. 2007; Langevin et al. 2011).

Histological Investigations: Dittrich (1963), as well as Bednar et al. (1995), examined histological pieces of posterior layer of lumbar fascia taken from patients with low back pain during lumbar surgery. They documented frequent signs of injury and inflammation (Dittrich, 1963; Bednar et al. 1995). Several histological examinations documented the presence of clearly nociceptive nerve endings in this tissue layer (Table 1). These studies, taken together, indicate that the lumbar fascia may be able to elicit pain, such as in at least some cases of low back pain. None of these studies included a comparison with healthy age-matched patients. It therefore cannot be excluded, that no difference may exist in fascial properties between low back patients and healthy patients. Moreover, the above studies cannot confirm whether or not the described tissue dynamics are in fact a common source of low back pain.

Reduced Shear-Motion: More recently, Langevin et al. (2011) conducted a comparison of the posterior layer of lumbar fasciae of chronic low back pain patients with those of group of age-matched controls. Using ultrasound cine-recording, this study examined the shear-motion within the posterior layer of the posterior layer of the lumbar fascia during passive lumbar flexion. The low back pain group was found to express a significant reduction in shear-strain compared with their healthy controls. Interestingly, the patients in this study showed increased thickness in this layer of the lumbar fascia. However, this change in thickness was found to be significant in male patients only. The reported reduction in shear-strain could of course be due to tissue adhesions induced by previous injury or inflammation. It would then be consistent with the proposed etiology suggested by Dittrich (1963) and Bednar et al. (1995). However, these findings cannot answer the question whether the observed tissue changes are cause or effect of low back pain. It is indeed possible that the tissue alterations are merely the result of a reduction (immobility) in everyday lumbar movements in low back pain patients.

In Vivo Studies: Several studies have explored the option of stimulating posterior layer of lumbar fascia for the purpose of eliciting nociceptive responses in vivo. Pedersen et al. (1956) pinched the corresponding fascia layer of decerebrated cats and was able to elicit spastic contractions in their back muscles (mostly ipsilateral), and also in their hamstring and gluteal muscles of the same leg.

The observed responses were much stronger in response to pinching the fascia than pinching the underlying muscle tissues. This finding was contrasted to the findings of an extensive investigation done by Kuslich et al. (1991) that used progressive local anesthesia in low back pain patients during disc surgery. While mechanical stimulation of the nerve root induced strong and often radiating back pain symptoms, the same stimulation on the posterior layer of lumbar fascia failed to elicit similar responses in the majority of patients. A more recent examination by Taguchi et al. (2008), on the other hand demonstrated that pinching the posterior layer of lumbar fascia of rats as well as applying hypertonic saline to it with a cotton ball induced clear responses in a significant number of neurons of the dorsal horn of their spinal cord. Since application of hypertonic saline is known to be the most effective stimulus for type VI afferents, the authors interpreted their findings as evidence for a nociceptive functional capacity of the lumbar fascia.

Interestingly, the same study demonstrated that inducing a chronic inflammation in the local musculature lead to a threefold increase in the number of dorsal horn neurons that are responsive to stimulation of the posterior layer of lumbar fascia. Their finding is reminiscent of the study of Gibson et al. (2009), which reported that hypertonic saline strongly increased pain when injected into the investing fascia of a muscle exposed to delayed onset soreness after eccentric exercise, although no comparable response was observed when the substance was injected into the actual muscle itself or into the non-exercised muscle in the contralateral leg.

Conclusion: The innervation of posterior layer of lumbar fascia clearly supports a nociceptive capacity. The nociceptive capacity suggests at least three different mechanisms for fascia-based low back pain sensation: 1) microinjuries and resulting irritation of nociceptive nerve endings in the posterior layer of lumbar fascia may directly induce back pain; 2) tissue deformations due to injury, immobility or excessive may impair proprioceptive signaling, which by itself could induce an augmentation in pain sensitivity via an activity-dependent sensitization of wide dynamic range neurons; and finally, 3) irritation in other tissues innervated by the same spinal segment could elicit an increased sensitivity in the posterior layer of lumbar fascia, which would then respond with nociceptive signaling, even to gentle stimulation. The question whether or not each of these scenarios (or various combinations of them) manifest in low back pain patients, or how often they occur, provides an important but also challenging background for future investigation. Clarification of these questions promises to offer valuable contributions for the treatment and prevention of back pain.

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Study	Tissue source	Method	Nerve endings found:	Remarks
Stilwell 1957	Macaca mulatta (n=17), rabbit (n=4)	Methylene blue.	Rich supply by FNE. Groups of large Pacinian corpuscles at penetration points of dorsal rami through the fascia. Also small Pacinian-like and Golgi-Mazzoni corpuscles.*	Study included human tissues too. However, no nerve type analysis was performed on those.
Hirsch et al 1963	Human (n=?)	Methylene blue	FNE, "complex unencapsulated endings" *	Number of donors not mentioned. Also found: unmyelinated nerve fiber network associated with blood vessels
Yahia et al. 1992	Human (n=7)	IH: Neurofilament protein and S-100 protein	FNE, Ruffini, Pacini.*	
Bednar et al 1995	Human (12),	IH: neuron-specific enolase	No terminal nerves found. *	Study performed with CLBP patients only. Found: small peripheral nerve bundles at the margins and in association with small vessels.
Corey et al. 2011	Rats (5)	3-D reconstructions of thick (30–80µm) tissue sections. IH: PGP9.5, CGRP, fast blue	CGRP positive FNE.	Also found: Some non-terminating CGRP-labeled fibers along blood vessels,

Tesarz et al. 2011	Rat (n=8) Human (n=3)	IH: PGP 9.5, TH, CGRP, SP	Rich innervation with presumable nociceptive nerve endings (PG, CGRP).	Most nerve fibers located in the outer layer of the lumbar fascia and in the subcutaneous connective tissue.
Benetazzo et al. 2011	Human (2)	3D reconstruction of serial sections. IH: S100	Study did not investigate nerve terminations.	Small nerves (mean diameter 15 µm) found, flowing from the superficial sub-layer into the adjacent subcutaneous loose connective tissue. No nerves visible in intermediate and deep sub-layers.
Hoheisel et al. 2015	Rats (10)	IH: PGP 9.5, TH, CGRP, SP	Rich innervation with presumable nociceptive nerve endings (PG, CGRP).	Inflammation of the fascia induced an increase of presumably nociceptive fibers.
Mense et al. 2016	Rats (5)	IH: PGP 9.5, TH, CGRP, SP, TRPV1	Rich innervation with presumable nociceptive nerve endings (PG, CGRP, TRPV1).	Inflammation of the fascia induced an increase of presumably nociceptive fibers

Table 1: Histological studies exploring the superficial layer of the PLF.

IH: immunohistochemical analysis. FNE: free nerve endings. *Method of identification of termination of small nerves not mentioned. Not included in this table are studies on supraspinous, interspinous or iliolumbar ligaments.