

Research papers

Sensory findings after stimulation of the thoracolumbar fascia with hypertonic saline suggest its contribution to low back pain



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ABSTRACT

Injection of hypertonic saline into deep tissues of the back (subcutis, muscle, or the surrounding fascia) can induce acute low back pain (LBP). So far, no study has analyzed differences in temporal, qualitative, and spatial pain characteristics originating from these tissues. The current study aimed to investigate the role of the thoracolumbar fascia as a potential source of LBP. In separate sessions, 12 healthy subjects received ultrasound-guided bolus injections of isotonic saline (0.9%) or hypertonic saline (5.8%) into the erector spinae muscle, the thoracolumbar fascia (posterior layer), and the overlying subcutis. Subjects were asked to rate pain intensity, duration, quality, and spatial extent. Pressure pain thresholds were determined pre and post injection. Injections of hypertonic saline into the fascia resulted in significantly larger area under the curve of pain intensity over time than injections into subcutis ($P < 0.01$) or muscle ($P < 0.001$), primarily based on longer pain durations and, to a lesser extent, on higher peak pain ratings. Pressure hyperalgesia was only induced by injection of hypertonic saline into muscle, but not fascia or subcutis. Pain radiation and pain affect evoked by fascia injection exceeded those of the muscle ($P < 0.01$) and the subcutis significantly ($P < 0.05$). Pain descriptors after fascia injection (burning, throbbing, and stinging) suggested innervation by both A- and C-fiber nociceptors. These findings show that the thoracolumbar fascia is the deep tissue of the back that is most sensitive to chemical stimulation, making it a prime candidate to contribute to nonspecific LBP but not to localized pressure hyperalgesia.

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1. Introduction

Although spinal structures (vertebrae, intervertebral discs, annulus fibrosus, facet joints, and spinal ligaments) are recognized as common causes of low back pain (LBP) [15], the role of muscles, fasciae, and other soft tissues as a potential source of LBP is often underappreciated [63]. However, there is increasing evidence that muscles and fasciae are involved in the development of LBP [8,47,60,68,69]. Immunohistochemical studies showed that the thoracolumbar fascia is innervated by nociceptive free nerve endings [13,65]. Furthermore, it has been shown that lumbar dorsal horn neurons receive nociceptive input from the fascia [27], suggesting a potential role of the thoracolumbar fascia in LBP.

Injections of hypertonic saline are frequently used to excite nociceptors in deep tissues, resulting in an activation of the nociceptive system by depolarizing small-diameter nociceptive afferent neurons [23,36], while blocking the generation of action potentials in large-diameter nonnociceptive fibers [50]. An injection of hypertonic saline into the abductor digiti minimi muscle and the overlying subcutis elicited similar pain intensities [40], and injection into the infrapatellar fat pad led to substantial pain radiation [5], indicating that many soft tissues display pain sensitivity to chemical stimulation. In the lower limb (tibialis anterior muscle), hypertonic saline injection into the tendon induced higher pain scores and larger referred pain areas than injection into the muscle itself [20], and the overlying crural fascia also showed a higher pain sensitivity to hypertonic saline than the underlying muscle [21], suggesting that connective tissue may generally be more sensitive than muscle. In the lumbar region, injection of hypertonic saline into the paraspinal muscles can evoke acute LBP [1,26,31,41,52], with similar effects on posture as in LBP patients [70], but very few data are available about the thoracolumbar fascia as a potential source of LBP.

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Since there is no comparative study distinguishing the muscle fascia of the low back from other tissue types, this study aimed at investigating the relative contribution of the thoracolumbar fascia, the erector spinae muscle, and the overlying subcutis to pain intensity, pain duration, pain quality, pain distribution, and changes in pressure pain thresholds after isotonic and hypertonic saline injections as human surrogate models of acute LBP. We hypothesized that an injection of hypertonic saline into the thoracolumbar fascia causes the highest pain intensity, the largest pain radiation, and the most pronounced sensitization to blunt pressure.

2. Material and methods

2.1. Participants

Twelve healthy volunteers (6 female, 6 male; mean age: 24.0 ± 1.5 years, mean \pm SD) with no history of back pain participated in this study. All volunteers had sufficient command of the German language. The criteria for exclusion were any medication, or recent surgeries to abdomen, legs, or back. None of the participants withdrew from the study prematurely. The local Ethics Committee of the Medical Faculty Mannheim, University Heidelberg, approved the experimental protocol on human volunteers (2010-274N-MA) according to the current version of the Declaration of Helsinki.

2.2. Experimental protocol

After signing a written consent form, all participants attended 3 study sessions separated by at least 5 days. In each session, pain intensity, pain quality, and pain distribution in response to isotonic/hypertonic saline, as well as pressure pain thresholds (PPT) before and after saline injections were determined (see below). Subjects were advised to lie on a bench face down, minimizing back muscle contraction. The PPT baseline was determined before any saline injection. After hypertonic or isotonic saline injection, the volunteers were asked to rate the magnitude of perceived pain at 10-second intervals for the first 2 minutes, and thereafter at 30-second intervals for the following 23 minutes (ie, total time of pain assessment was 25 minutes) on a numerical rating scale with the end points 0 (=no pain) and 100 (=most intense pain imaginable). The subjects marked the distribution of the experimentally induced pain on a standard human body scheme while they were perceiving it, that is, without depending on episodic pain memory.

After pain sensation had subsided, PPT was determined at 25 minutes after injection and pain qualities were inquired. Within each session, the experimental protocol was performed twice, with hypertonic saline on one side and isotonic saline on the contralateral side. A second determination of PPT was done at 50 minutes after saline injection.

2.3. Saline administration

Bolus injections (400 μ L) of hypertonic saline (5.8%) or isotonic saline (0.9%) as a control were made into the posterior layer of the thoracolumbar fascia, the erector spinae muscle, and the overlying subcutis at lumbar level (L3/L4), about 4 cm lateral to the spinous processes. For all injections, the position of the injection needle was guided by ultrasound (Acuson X150; Siemens, Munich, Germany). Owing to the low echo contrast in subcutis and muscle, the saline distribution could only be assessed in the fascia by ultrasound imaging. Fig. 1 shows a typical example of an ultrasound image before (A) and after (B) hypertonic saline injection into the middle portion of the thoracolumbar fascia. Examples of ultrasound images after hypertonic saline injection for 5 additional

volunteers are shown in Fig. 1C–G. The time course of resolution of hypertonic saline injection into the fascia was measured in 3 healthy volunteers (Fig. 1H). The hypoechoic area marking the injection volume was determined every 10 seconds after bolus injection of hypertonic saline as horizontal and vertical spread.

In contrast to subcutis or fascia injection, saline injection into the muscle was performed vertically about 1 cm beyond the fascia after pulling the skin sideways in order to prevent capillary effects after needle withdrawing, which may lead to fluid reflow. The solution was administered using a 1-mL syringe (Becton Dickinson, Madrid, Spain) and a 27G cannula.

The volunteers were informed that they would receive 2 injections per session into either muscle, fascia, or subcutis. The experimental design of the study was a fully balanced right-left crossover design comprising the order of hypertonic or isotonic saline injection and tissue type selection. All participants were blinded with regard to the injected solution and tissue.

2.4. Pressure pain threshold (PPT)

A pressure algometer (Wagner Instruments, Greenwich, CT, USA) with a round rubber tip (contact area 1 cm²) was pressed on the skin overlying the erector spinae muscle. With the tip size used for stimulation, mainly nociceptors from deep tissues were activated, while the contribution of cutaneous nociceptors to the overall pain was small [35]. The PPT was determined at 4 different locations, including the point of injection (central) and 3 other areas approximately 5 cm cranial, caudal, and lateral to the injection site. The PPTs were determined with 3 series of ascending stimulus intensities, each with a ramp rate of approximately 50 kPa/s (≈ 0.5 kg/cm²).

2.5. Pain distribution

All volunteers were asked to mark the distribution of their acute pain on a standardized 2-dimensional paper form body image while they perceived the experimentally induced LBP. The scheme showed the back, the abdominal, and leg region, of a drawn standardized body and was presented during the entire 25-minute postinjection period. One slim subject developed a slight compression nerve block of the lateral femoral cutaneous nerve due to the long duration of the face-down position, which led to paresthesia that was, however, clearly distinguished from saline-induced pain, and was thus disregarded in the analysis and therefore not plotted in the respective figures.

2.6. Pain quality

The assessment of pain qualities elicited by saline injection consisted of a list of verbal descriptors (Pain Perception Scale, “Schmerzempfindungs-Skala” [SES]) comprising 14 affective and 10 sensory items [19]. Descriptors were rated on a 4-level ordinal scale (0 = no match, 1 = light match, 2 = largely match, 3 = total match).

2.7. Statistics

Statistical analysis was performed using SigmaPlot software, version 12.0 (Systat Software, Inc, Chicago, IL, USA). Significant differences (at P -values < 0.05) were determined by 2-way repeated-measures analysis of variance (ANOVA; factors: tissue and saline concentration) followed by Holm-Sidak post hoc test correcting for multilevel comparison. A comprehensive overview of ANOVA analyses is given in Table 1. All values given in this study are depicted as mean \pm SEM, unless stated otherwise (SD for biological variability of tissue thickness).

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