

What Is the Source of Chronic Low Back Pain and Does Age Play a Role?

Michael J. DePalma, MD,* Jessica M. Ketchum, PhD,[†] and Thomas Saullo, MD[‡]

*Department of Physical Medicine and Rehabilitation, Virginia Commonwealth University/Medical College of Virginia Hospitals, Richmond, Virginia;

[†]Department of Biostatistics, Virginia Commonwealth University, Theater Row, Richmond, Virginia;

[‡]Department of Physical Medicine and Rehabilitation, Virginia Commonwealth University/Medical College of Virginia Hospitals, VCU Spine Center, Richmond, Virginia, USA

Reprint requests to: Michael J. DePalma, MD, VCU Spine Center, 8700 Stony Point Parkway, Suite 260, Richmond, VA 23235, USA. Tel: 804-323-2982; Fax: 804-323-2998; E-mail: depalmamj8@yahoo.com.

Abstract

Objective. The objective of this study was to estimate the prevalence, mean age, and association of prevalence and age of lumbar internal disc disruption (IDD), facet joint pain (FJP), sacroiliac joint pain (SIJP), spinal and pelvic insufficiency fractures, interspinous ligament injury/Baastrup's Disease, and soft tissue irritation by fusion hardware.

Design. The study's design was a retrospective chart review.

Setting. The study was set in an academic spine center.

Patients. A total of 378 cases from 358 patients were reviewed of which 170 cases from 156 patients who underwent diagnostic procedures were included.

Interventions. Discography, dual diagnostic facet joint blocks, intra-articular sacroiliac joint injections, anesthetic injections of painful interspinous ligaments/opposing spinous processes/posterior fusion hardware, or percutaneous augmentation were performed.

Outcome Measures. Prevalence and age were analyzed for each diagnosis group.

Methods. Patients with recalcitrant low back pain underwent diagnostic procedures based on their clinical presentation until the pain source was identified.

Results. The prevalence of internal disc disruption, facet joint pain and sacroiliac joint pain was 42%, 31%, and 18%, respectively. Patients with internal disc disruption were significantly younger than those with facet joint pain or sacroiliac joint pain. Increased age was associated with a decreased probability of internal disc disruption and increased probabilities of facet joint pain and sacroiliac joint pain as the source of low back pain until approximately age 70.

Conclusion. Our data confirm the intervertebral disc as the most common etiology of chronic low back pain in adults. Based on our sample, the younger the patient, the more likely low back pain is discogenic in origin. Facetogenic or sacroiliac joint pain is more likely in older patients.

Key Words. Internal Disc Disruption; Sacroiliac Joint; Facet Joint; Low Back Pain; Adult

Introduction

Low back pain (LBP), a ubiquitous complaint, is believed to be difficult to diagnose [1–3]. Traditionally, the notion that the etiology of 80% to 90% of LBP cases is unknown has been perpetuated across decades [1–4]. Fundamentally, any spinal structure can serve as the source of LBP in affected patients provided the structure: 1) is innervated; 2) is capable of causing pain similar to that encountered clinically; and 3) is susceptible to disease or injury known to be painful. Investigators [5–10] have adduced the above principles to identify the structural causes of LBP in adult patients. Over the better part of the previous two decades, evidence has been compiled verifying the sources of LBP and initial prevalence estimates for painful lumbar intervertebral discs (IDD) [5], symptomatic facet joint pain (FJP) [6–8], and sacroiliac joint pain (SIJP) [9,10].

Independent studies of adult LBP patients have estimated prevalence rates for IDD [5], FJP [6–8], and SIJP [9,10] of 39%, 15–32%, and 13–18.5%, respectively. More recently, emerging reports suggest the presence of LBP emanating from opposing lumbar spinous processes (Baastrup's disease) or degeneration of the intervening interspinous ligament [11–17], and soft tissue irritation by fusion hardware [18]. The most common etiology of LBP in the young to middle age adult is IDD [5] followed by FJP

[6–8] and SIJP [9,10] with the latter two occurring at a seemingly similar prevalence rate. Schwarzer's seminal papers helped establish the etiology of adult LBP although not clearly identifying how age affects prevalence of each diagnostic group (IDD, FJP, SIJP). Soon thereafter, other reports documented similar prevalence rates of FJP [6–8] and SIJP [9,10] suggesting a predilection of these two conditions for older subjects [6,7].

The specific aims of the present study were to 1) estimate the prevalence of lumbar IDD, FJP, SIJP, spinal and pelvic insufficiency fractures, interspinous ligament injury/Baastrup's Disease, and soft tissue irritation by fusion hardware in consecutive LBP patients having completed precision, controlled diagnostic spinal procedures; 2) compare the mean age among IDD, FJP, and SIJP cohorts; and 3) determine if age is significantly associated with the prevalence of IDD, FJP, or SIJP.

Methods

After obtaining Institutional Review Board approval, charts from consecutive LBP patients were reviewed. Enrolled cases were patients suffering from LBP recalcitrant to spine-focused physical therapy, oral analgesics, and oral anti-inflammatory medications, whose LBP was incapacitating and thus interfering with daily activities who underwent diagnostic spinal procedures. These were LBP patients presenting to a community-based, multi-disciplinary, academic spine center. Patients were referred to the spine center from community and university spine surgeons (neurosurgery and orthopedics), physiatrists, non-spine surgeons, primary care physicians, rheumatologists, endocrinologists, neurologists, and occupational health physicians.

Each patient either underwent provocation lumbar discography (PLD), dual diagnostic facet joint blocks (FJB) with local comparative anesthetics, intra-articular diagnostic SIJ injections (sacroiliac joint block [SIJB]), injection of anesthetic into putatively painful interspinous ligaments/opposing spinous processes/posterior fusion hardware, or percutaneous augmentation depending on the clinical presentation. Some subjects underwent multiple diagnostic procedures until the source of their LBP was identified. If the initial diagnostic procedure was negative, the next most likely structure in the diagnostic algorithm was interrogated. However, once a source of the subject's LBP was identified, subsequent diagnostic procedures were not performed. This interventional spine care diagnostic algorithm was consistently applied to all consecutive low back pain patients evaluated by the lead author.

Patients reporting paravertebral LBP without midline LBP [9,19] which was exacerbated by standing and/or walking [20] and who demonstrated ≤ 2 positive SIJ provocative maneuvers [21] and/or a lack of centralization during McKenzie evaluation [22] typically underwent FJB first, followed by SIJB and then PLD if the preceding diagnostic procedure was negative (Figure 1). Strict operational criteria were adhered to for all blocks with 0.5 mL of anes-

thetic injected per International Spine Intervention Society (ISIS) Guidelines. One percent lidocaine is used for the first block with 0.5% bupivacaine for the second. Duration of expected relief should be ≤ 2 hours for lidocaine and ≤ 8 hours for bupivacaine. Facet joint mediated pain was usually approached first with diagnostic intra-articular injections, and if positive, followed by a second diagnostic block of the respective medial branches (or medial branch and dorsal ramus as appropriate). The side and joint level selected by pain referral pattern [23,24] were investigated first moving from most likely to less likely facet joint (FJ) level. Only one side and level was injected at any one time moving on to the next most likely facet joint level only after a negative initial diagnostic block. Patients reporting paravertebral LBP without midline LBP [9,10,25,26] and three positive out of five SIJ provocative maneuvers [21,27] without centralization during McKenzie evaluation [22] underwent SIJB followed by FJBs and then PLD unless the initial diagnostic blocks were positive. Patients reporting paravertebral LBP with a previous history of posterior fusion with pedicle screws and hardware whose LBP was reproducible by single digit palpation over the hardware, underwent diagnostic blockade of the hardware in a triple blockade fashion using 2% lidocaine first, then 0.5% marcaine second, followed by a placebo injection. Patients reporting midline LBP with or without paravertebral LBP, centralization during McKenzie evaluation [22], and/or LBP during sustained hip flexion [28] underwent PLD initially followed by FJB or SIJB if discography was negative (Figure 2). Patients reporting midline LBP without paravertebral LBP which was aggravated by standing and walking and not provoked by sitting or sustained hip flexion (SHF) with evidence of opposing lumbar spinous processes on imaging, first underwent diagnostic interspinous injection [11,29] of the segmental level supported by the cephalad to caudad location of the LBP. Osteopenic patients with LBP reproducible on percussion [30] and magnetic resonance imaging (MRI), or computerized tomography/bone scan (in the case of contraindication to MRI) evidence of an insufficiency fracture of the vertebral body or sacrum underwent percutaneous augmentation (Figure 3) [31–36].

Positive discography was defined as concordant/partial concordant LBP ($>6/10$) at low pressure (<50 psi over opening pressure) due to \geq Grade III annular tears [37–39]. Diagnostic blockade of FJ, SIJ, or other structures was deemed positive if the patient's index pain was relieved by $\geq 75\%$ after injection of each anesthetic [40–42]. In the case of fusion hardware blockade, minimal relief ($<75\%$) after the placebo injection was required to constitute a positive block. Insufficiency fractures were deemed the source of LBP if the patient's clinical symptoms were significantly reduced after percutaneous augmentation [31–36].

Based on the results of discography, diagnostic blockades, or LBP reduction after percutaneous augmentation, subjects were classified as having IDD, FJP, SIJP, fusion hardware-mediated soft tissue pain, Baastrup's Disease, or vertebral or sacral insufficiency fractures. Charts of

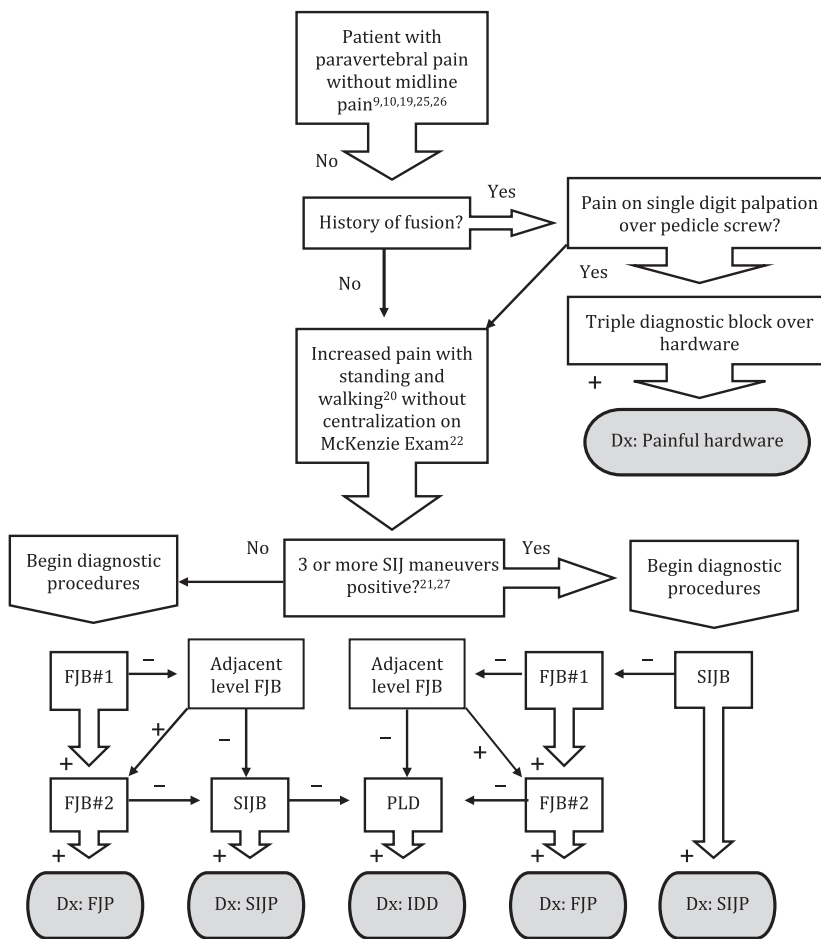


Figure 1 Algorithm for assessing predominant paramidline LBP. SIJ = sacroiliac joint; FJB = facet joint blocks; FJP = facet joint pain; SIJP = sacroiliac joint pain; PLD = provocation lumbar discography; IDD = internal disc disruption.

patients who did not undergo definitive diagnostic procedures due to clinical improvement of LBP were reviewed but not enrolled in this study. Initially, the prevalence of each source was estimated by computing the proportion of patients with each diagnosed source presenting with LBP out of all diagnosed patients. The mean age for each of the diagnostic groups was estimated and compared statistically between the IDD, FJP, and SIJP groups using analysis of variance (ANOVA) and Tukey's honestly significant difference (HSD) to adjust for multiple comparisons. The relationship between age and the probability of each source of LBP (IDD, FJP, SIJP, or other) was modeled with multinomial logistic regression analyses assuming a generalized logit link function. The significance level for all tests was 5%. SAS v.9.2 (SAS Institute, Cary, NC, USA) was used for all data analysis and graphics.

Results

A total of 378 cases from 358 subjects (34.9% male) seen between November 2007 and December 2008 were reviewed. Patients had a mean age of 52.8 years (standard deviation [SD] = 15.0) and median duration of LBP of 12 months (interquartile range [IQR] = 6–24). There were 208 cases from 202 patients not included in subsequent

calculations because these patients did not undergo definitive diagnostic procedures. Of the 170 cases from 156 patients presenting with LBP whose low back disorder was definitively diagnosed, the mean age was 54.4 years (SD = 16.2) and median duration of LBP was 12 months (IQR = 6–32), and 28 had a history lumbar fusion.

Cases were grouped according to the diagnosis of their LBP. The prevalence of each source of LBP and mean age of the cases within each group is summarized in Table 1. Sources of LBP were primarily identified as being IDD, FJP, or SIJP (>90%). There were four patients (eight cases) presenting with two simultaneous source structures, and one patient (three cases) presenting with three simultaneous source structures. The most predominantly affected level for both IDD and FJP was L5-S1 followed by L4-L5 (see Table 2). Of the 71 cases of IDD, 19 (26.8%) involved more than one level with 12 (16.9%) involving two levels and seven (9.9%) involving three levels.

An ANOVA test indicated that the mean age was significantly different among the IDD, FJP, and SIJP groups ($F_{2,151} = 31$, P -value < 0.0001). Using Tukey's HSD to adjust for multiple comparisons, cases of IDD were significantly younger than cases of FJP or SIJP; however,

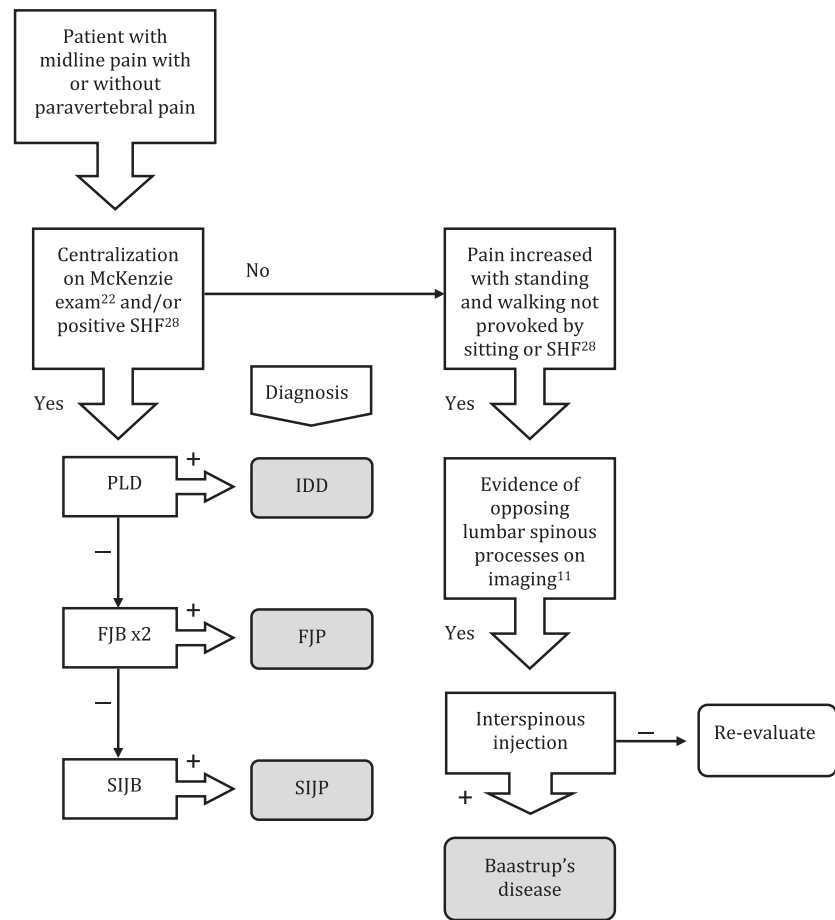


Figure 2 Algorithm for assessing LBP presenting in midline. PLD = provocation lumbar discography; FJB = facet joint blocks; IDD = internal disc disruption; FJP = facet joint pain; SIJP = sacroiliac joint pain; SHF = sustained hip flexion.

the mean age was not significantly different between cases of FJP and SIJP. The multinomial logistic regression analyses indicated that age was significantly associated with source of LBP ($\chi^2_3 = 41.8$, P -value < 0.0001). The predicted probabilities and their associated 95% confidence intervals for each source as a function of age are shown in Figure 4. In general, increased age was associated with a decreased probability of IDD as the source of LBP. In addition, increased age was associated with increased probabilities of FJP and SIJP as the source of LBP until approximately age 70, and then with decreased probabilities of FJP and SIJP as the source.

Discussion

The findings of the present study corroborate earlier reports [5–10] of the prevalence rates of IDD [5], FJP [6–8], and SIJP [9,10] in LBP patients (Table 3). We estimated the prevalence of IDD to be 42% (95% confidence interval [CI] = 35% to 49%), the prevalence of FJP to be 31% (95% CI = 24% to 38%), and the prevalence of SIJP to be 18% (95% CI = 13% to 25%). Our data confirm that the intervertebral disc is the most common etiology of chronic LBP in the adult LBP population. Our prevalence estimate for lumbar IDD mirrors that of Schwarzer’s observation of 39% (95% CI = 29% to 49%). The sample size studied

Figure 3 Clinical assessment of LBP suspicious for insufficiency fracture. LBP = low back pain; MRI = magnetic resonance imaging; CT = computerized tomography.

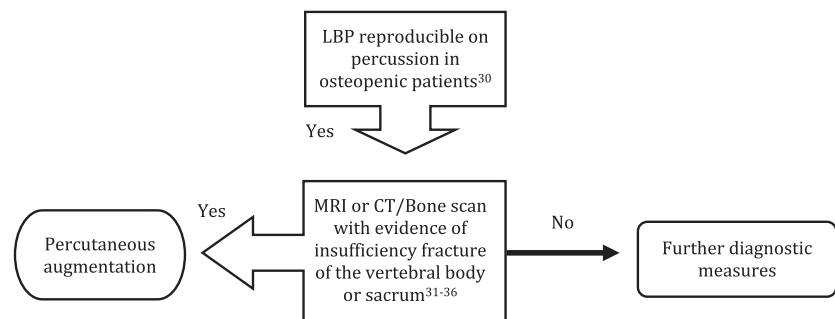


Table 1 Prevalence and mean age by source of LBP

Source of LBP (N = 170)	Count	Prevalence (%)	95% CI prevalence (%)	Mean age (SD)	95% CI age
Intervertebral disc	71	41.8	(34.6, 49.3)	43.7 (10.3)	(41.3, 46.1)
Lumbar facet joint(s)	52	30.6	(24.2, 37.9)	59.6 (13.1)	(56.0, 63.3)
Sacroiliac joint(s)	31	18.2	(13.2, 24.7)	61.4 (17.7)	(54.9, 67.9)
Vertebral insufficiency fracture	5	2.9	(1.3, 6.7)	79.0 (11.8)	(64.3, 93.7)
Pelvic insufficiency fracture	3	1.8	(0.6, 5.1)	71.3 (11.7)	(42.2, 100.4)
Baastrup's disease	3	1.8	(0.6, 5.1)	75.3 (4.7)	(63.6, 87.1)
Fusion hardware	5	2.9	(1.3, 6.7)	59.6 (19.4)	(35.4, 83.8)

LBP = low back pain; CI = confidence interval; SD = standard deviation.

here allowed for a reasonably accurate prevalence estimate with a tighter confidence interval than the previous report by Schwarzer (margin of error 14 vs 20). Our patient cohort is similar and dissimilar to Schwarzer's. The female gender was more commonly affected in our study (66%) and the majority of cases were spontaneous and gradual in onset (66%) and not due to motor vehicle collision, lifting, falls, or other traumatic events. Schwarzer's patient cohort of IDD cases were predominantly male (66%) and primarily work related (56%) which logically involved an episode of poor biomechanics such as improper lifting techniques or repetitive lifting. The fact that our IDD prevalence estimate is in-line with Schwarzer's suggests a similar occurrence of lumbar IDD across demographic categories within the adult population. An injured intervertebral disc more commonly causes chronic LBP than injury of a facet joint or sacroiliac joint and accounts for half of all chronic LBP cases.

Our prevalence estimate for FJP is significantly higher than Schwarzer's 1994 North American finding of 15% (95% CI = 10% to 20%) which was arrived at by employing less

stringent diagnostic criteria in a cohort of LBP subjects with median age 38 years. The incongruity between our FJP prevalence data and Schwarzer's could be explained by the difference in age of the groups studied. In a separate 1995 paper, using stringent diagnostic block criteria, Schwarzer reported an Australian FJP prevalence rate of 32% (95% CI = 20% to 44%) in a patient group with a median age of 59 years [6], which is similar to the mean age of 53 years for our entire LBP sample. Manichikanti reported a FJP prevalence rate of 27% (95% CI = 22% to 33%) among a broad range of age groups with a median age younger (approximately 47 years) [7] than Schwarzer's Australian population and our study cohort. The confidence intervals for the prevalence estimates of the FJP diagnostic category from our work, Manichikanti's paper, and Schwarzer's Australian paper do not overlap Schwarzer's North American report suggesting there may be categorical differences between studied patient samples such as age.

Using reduction of LBP as the diagnostic criterion, Schwarzer found an SIJP prevalence of 30% (95%

Table 2 Side and level of source structure for cases of IDD, FJP, and SIJP

	IDD (N = 71) (%)	FJP (N = 52) (%)	SIJP (N = 31) (%)
Side of source structure			
Left	—	24 (46.2)	17 (54.8)
Right	—	16 (30.8)	12 (38.7)
Bilateral	—	12 (23.1)	2 (6.5)
N/A	71 (100.0)	—	—
Level of source structure			
L1-2	3 (4.2)	1 (2.0)	—
L2-3	12 (16.9)	—	—
L3-4	6 (8.5)	—	—
L4-5	21 (29.6)	15 (29.4)	—
L5-S1	28 (39.4)	35 (68.6)	—
T10-11	1 (1.4)	—	—
Unknown	—	1	—
N/A	—	—	31 (100)

IDD = internal disc disruption; FJP = facet joint pain; SIJP = sacroiliac joint pain.

Table 3 Predicted prevalence estimates based on age compared to estimates from prior studies

Source	Study	Estimates from prior studies			Estimates based on model [†]	
		Age [‡] (years)	Prevalence (%)	95% CI (%)	Prevalence (%)	95% CI (%)
IDD	Schwarzer (a)	36.7	39	(29, 49)	77.2	(67.0, 87.3)
	Manichikanti (b)	~47	26	(18, 34)	55.0	(45.4, 64.5)
FJP	Schwarzer (c)	38.4	15	(10, 20)	16.2	(8.1, 24.3)
	Schwarzer (d)	59	32	(20, 44)	40.7	(31.7, 49.8)
	Manichikanti (e)	~47	27	(22, 33)	27.5	(19.1, 36.0)
SIJP	Schwarzer (f)	32.8	30	(16, 44)	5.3	(0.7, 9.8)
	Schwarzer (f)	32.8	13	(6, 44)	5.3	(0.7, 9.8)
	Maigne (g)	45.3	18.5	—	12.9	(6.4, 19.5)

CI = confidence interval; IDD = internal disc disruption; FJP = facet joint pain; SIJP = sacroiliac joint pain.

[†] Predicted prevalence using age value from corresponding study.

[‡] Mean or median age from study.

Schwarzer (a): The Prevalence and clinical features of internal disc disruption in patients with chronic low back pain. *Spine, Volume 19, Number 17, 1995 pp 1878–1881.*

Manichikanti (b): Evaluation of the Relative Contributions of Various Structures in Chronic Low Back Pain. *Pain Physician, Volume 4, Number 4, pp 308–316 2001, ISSN 1533-3159.*

Schwarzer (c): Clinical Features of Patients with Pain Stemming from the Lumbar Zygapophysial Joints. *Spine, Volume 19, Number 10, pp 1132–1137.*

Schwarzer (d): Prevalence and clinical features of lumbar Zygapophysial joint pain: a study in an Australian population with chronic low back pain. *Annals of the Rheumatic Diseases 1995; 54: 100–106.*

Manichikanti (e): Age-Related Prevalence of Facet-Joint Involvement in Chronic Neck and Low Back Pain. *Pain Physician, 2008; 11:67-75 ISSN 1533-3159.*

Schwarzer (f): The Sacroiliac Joint in Chronic Low Back Pain. *Spine, 1995 Volume 20, Number 1, pp 31–37.*

Maigne (g): Results of Sacroiliac Joint Double Block and Value of Sacroiliac Pain Provocation Test in 54 Patients with Low Back Pain. *Spine: Volume 21(16), 15 August 1996 pp 1889–1892.*

CI = 16% to 44%) utilizing single diagnostic blocks in a sample with a median age of 33 years. The authors more selectively screened appropriate subjects to undergo diagnostic SIJ blocks. When considering the entire LBP cohort prior to screening, the prevalence as detected by single diagnostic blocks may have been as low as 13% (95% CI = 6% to 20%). In a subsequent study, Maigne employed a dual diagnostic block paradigm and reported a prevalence rate of 18.5% of SIJP in a subject sample with a median age of 45 years [10]. Our observed SIJP prevalence appears similar to both of these previous reports. If we accept our data and Maigne's data as more rigorously obtained, the prevalence rate of SIJP appears to be on the order of 18–19% and less common than IDD in the adult LBP population.

Age is associated with the prevalence of IDD, FJP, and SIJP in adult LBP patients. The mean age of our IDD subgroup of patients (44 years) was statistically younger than the mean age of our FJP (60 years) and SIJP (61 years) subgroups, while the latter two groups were similar in age. The positive relationship between age and the prevalence of FJP has been previously suggested [6,7]. However, the positive relationship between age and the prevalence of SIJP has not been previously reported. Based on our sample, we found that increases in age were significantly associated with decreases in the probability of IDD as the source of LBP and increases in age were significantly associated with increases in the prob-

ability of FJP or SIJP as source the LBP until approximately age 70. In simpler terms, the younger the LBP patient, the more likely his or her LBP is discogenic in origin (Figure 1). In contrast, the older the LBP patient, the more likely his or her LBP is facetogenic or SIJ in origin (Figure 1). For the eldest of the population, other sources of LBP (Baastrup's disease or insufficiency fractures) were suggested. Our findings of the relationship between age and the prevalence estimates of IDD, SIJ, or FJP as the source of LBP seem congruous with earlier reports.

For ethical reasons, each patient reviewed in this protocol did not undergo all diagnostic interventions (discography and diagnostic blocks). Rather, a focused, pragmatic algorithmic approach was adopted. Ultimately, one could argue that an erroneous calculation of the prevalence estimate for lumbar IDD, FJP, and SIJP was committed. By not performing discography on every patient, it is plausible that we failed to detect all cases of IDD and have underreported it. A similar comment could be made about diagnostic FJ and SIJ blocks. However, each patient analyzed underwent definitive diagnostic procedures until we reached confirmation of the source of that patient's LBP. If a patient was initially evaluated with diagnostic FJ and/or SIJ blocks which were negative, that patient underwent discography to verify the presence of IDD and vice versa. Only patients whose clinical status improved with proper care did not undergo diagnostic procedures.

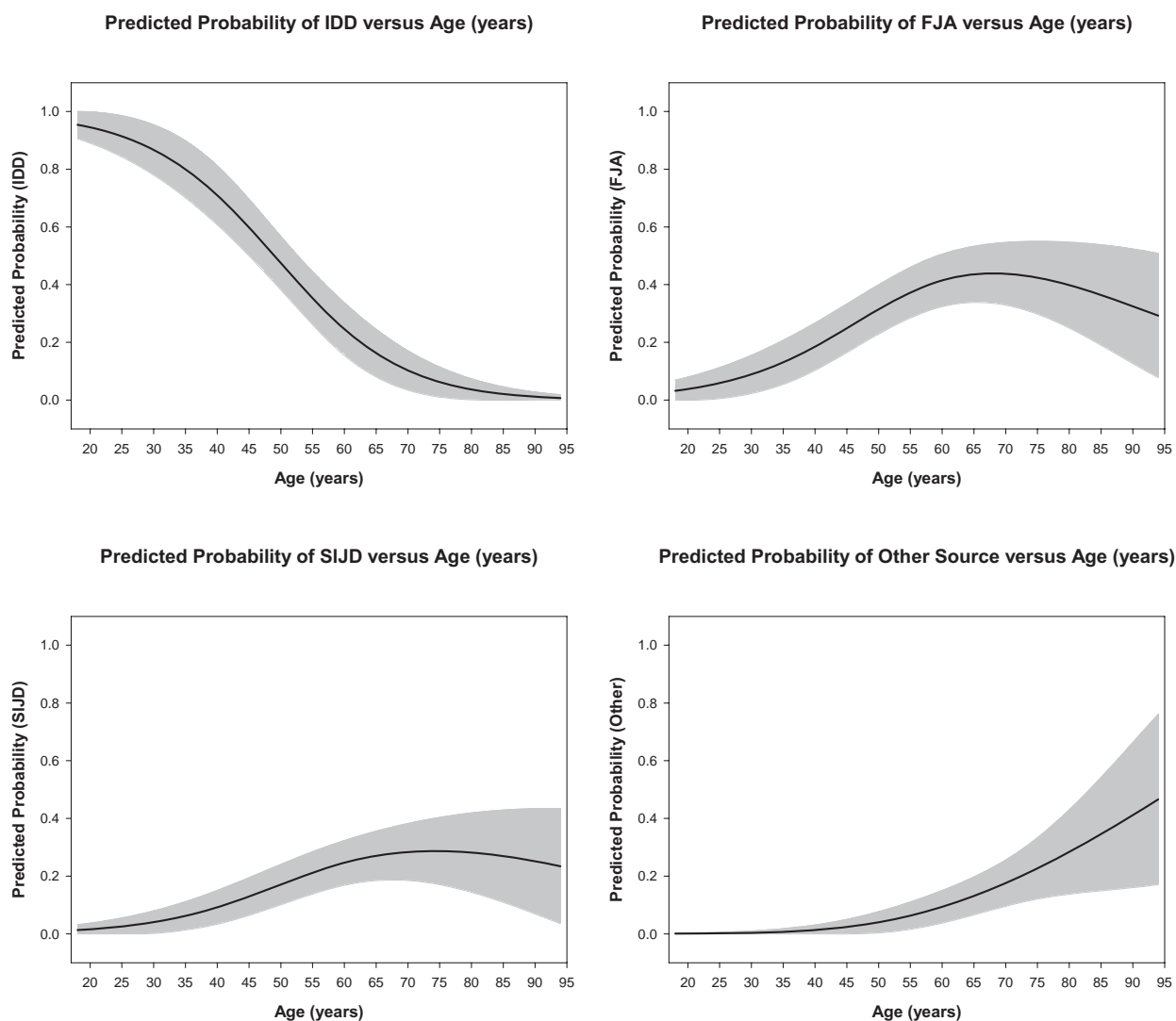


Figure 4 Predicted probabilities and 95% confidence intervals for internal disc disruption (IDD), facet joint pain (FJP), sacroiliac joint pain (SIJP), and other sources of low back pain (LBP) as a function of age.

Opponents of discography and, to a lesser degree, diagnostic procedures in general, would contend that false positive rates have overestimated our prevalence estimates. Application of meticulous technique and strict adherence to supported operational criteria for discography [39] will minimize false positive rates to acceptably low levels [38] allowing accurate detection of symptomatic lumbar internal disc disruption [5,39,43,44]. Similarly, sufficiently performed diagnostic FJ blocks and SIJ injections are associated with acceptable false positive rates. Lastly, if our findings were skewed by false positives, we would have likely observed different prevalence data less congruent with previous reports. By virtue of the fact that most previously reported prevalence estimates for each diagnostic group (IDD, FJP, SIJP) fall within our CIs for each group, our findings are likely accurate.

Our method of determining clinically significant insufficiency fractures could be attacked due to two recent randomized controlled trials calling into question the therapeutic benefit of vertebroplasty when performed based on MRI imaging. [45,46] Langdon et al. [47], found that pain with closed-fist percussion was a reliable indicator of symptomatic vertebral compression fracture with a sensitivity of 87.5% and specificity of 90%. The diagnostic algorithm for our current study required imaging evidence of an insufficiency fracture in conjunction with pain on percussion over the fracture level prior to percutaneous augmentation. It may be that the performance of vertebroplasty on patients selected based on imaging findings is less successful than when performed predicated on combined imaging and physical examination findings. [48] Nonetheless, imaging and exam evidence of active insuf-

fracture coupled with pain reduction after augmentation of the index fracture solidified that fracture's role as the source of the patient's LBP.

Accurate diagnosis of the specific source of chronic LBP will help break the futile cycle into which many patients are directed of ineffective spinal procedures. In essence, the therapeutic utility of the diagnosis itself is that treatments can then be directed, or avoided, toward the source of symptoms. For example, on face it would seem indefensible that a 35-year-old chronic LBP patient would experience LBP relief after undergoing neurotomy of the bilateral L4 medial branches and L5 dorsal rami. Despite the performance of technically sound neurotomy procedures, this procedure is doomed for a poor outcome because the most likely source of this patient's symptoms resides within the anterior column unaffected by a procedure targeting a posterior element. As opposed to dated beliefs that 90% of LBP cases cannot be diagnosed [1–3], numerous investigations have now demonstrated that the converse is in fact true [5–10,49].

References

- 1 Dillane JB, Fry J, Kalton G. Acute back syndrome: A study from general practice. *Br Med J* 1966;2:82–4.
- 2 Spratt KF, Lehmann TR, Weinstein JN, et al. A new approach to low back physical examination. Behavioral assessment of mechanical signs. *Spine* 1990;15(2):96–102.
- 3 Nachemson AL. The natural course of low back pain. Chapter 5. In: White AA, ed. *American Academy of Orthopedic Surgeons Symposium on Idiopathic Low Back Pain*. St Louis: CV Mosby; 1982:46–51.
- 4 Valkenburg HA, Haanen HCM. The epidemiology of low back pain. Chapter 2. In: White AA, ed. *American Academy of Orthopedic Surgeons Symposium on Idiopathic Low Back Pain*. St Louis: CV Mosby; 1982:9–22.
- 5 Schwarzer AC, Aprill CN, Derby R, et al. The prevalence and clinical features of internal disc disruption in patients with chronic low back pain. *Spine* 1995;20(17):1878–81.
- 6 Schwarzer A, Wang SC, Bogduk N, McNaught PF, Lauren R. Prevalence and clinical features of lumbar z joint pain: A study in an Australian population w/chronic low back pain. *Ann Rheum Dis* 1995;54:100–6.
- 7 Manchikanti L, Manchikanti KN, Cash KA, Singh V, Giordano J. Age-related prevalence of facet-joint involvement in chronic neck and low back pain. *Pain Physician* 2008;11:67–75.
- 8 Manchukonda R, Manchikanti KN, Cash KA, Pampati V, Manchikanti L. Facet joint pain in chronic spinal

- 9 Schwarzer AC, Aprill CN, Bogduk N. The sacroiliac joint in chronic low back pain. *Spine* 1995;20:31–7.
- 10 Maigne JY, Aivaliklis A, Fabrice P. Results of sacroiliac joint double block and value of sacroiliac pain provocation tests in 54 patients with low back pain. *Spine* 1996;21(16):1889–92.
- 11 DePalma MJ, Slipman CW, Siegelman E, et al. Interspinous bursitis in an athlete: A case report. *J Bone Joint Surg Br* 2004;86-B(7):1062–4.
- 12 Knievel S, Lamer T. Chapter 17: Midline posterior element disorders. In: DePalma MJ, ed. *iSpine*. New York: Demos Publishing; 2010 (in press).
- 13 Jaffe MJ, Giovanello MT. Successful radiofrequency ablation for Baastrop's disease and intraspinal ligament pain: A case report. *International Spine Intervention Society 17th Annual Scientific Meeting, Toronto CN, July 2009*.
- 14 Maes R, Morrison WB, Parker L, et al. Lumbar interspinous bursitis (Baastrup disease) in a symptomatic population: Prevalence on MRI. *Spine* 2008;33(7):E211–5.
- 15 Bywaters EG, Evans S. The lumbar interspinous bursae and Baastrop's syndrome: An autopsy study. *Rheumatol Int* 1982;2(2):87–96.
- 16 Newman PH. Sprung back. *J Bone Joint Surg* 1952;34:30–7.
- 17 Scapinelli R, Stecco C, Pozzuoli A, et al. The lumbar interspinous ligaments in humans: Anatomical study and review of the literature. *Cells Tissues Organs* 2006;183(1):1–11.
- 18 Bhargava A. Chapter 18: Fusion hardware mediated low back pain. In: DePalma MJ, ed. *iSpine*. New York: Demos Publishing; 2010 (in press).
- 19 Laslett M, McDonald B, Aprill CN, Tropp H, Oberg B. Clinical predictors of screening lumbar zygapophyseal joint blocks: Development of clinical prediction rules. *Spine J* 2006;6:370–9.
- 20 Revel M, Poiraudou S, Auleley GR, et al. Capacity of the clinical picture to characterize low back pain relieved by facet joint anesthesia: Proposed criteria to identify patients with painful facet joints. *Spine* 1998;23(18):1972–6.
- 21 Laslett M, Aprill CN, McDonald B, Young SB. Diagnosis of sacroiliac joint pain: Validity of individual provocation tests and composites of tests. *Man Ther* 2005;10:207–18.

DePalma et al.

- 22 Laslett M, Young SB, Aprill CN, McDonald B. Diagnosing painful sacroiliac joints: A validity study of a McKenzie evaluation and sacroiliac provocation tests. *Aust J Physiother* 2003;49:89–97.
- 23 Fukui S, Ohseto K, Shiotani M, et al. Distribution of referred pain from the lumbar zygapophyseal joints and dorsal rami. *Clin J Pain* 1997;13(4):303–7.
- 24 Lipetz J. Lumbar pain—An algorithmic methodology. In: Slipman CW, Derby R, Simeone FA, Mayer TG, eds. *Interventional Spine: An Algorithmic Approach*. London: Elsevier; 2007:975–90.
- 25 Fortin J, Aprill C, Ponthieux B, Pier J. Sacroiliac joint: Pain referral maps upon applying a new injection/arthrography technique. Part II: Clinical evaluation. *Spine* 1994;19:1483–9.
- 26 Fortin J, Dwyer A, West S, Pier J. Sacroiliac joint: Pain referral maps upon applying a new injection/arthrography technique. Part I: Asymptomatic volunteers. *Spine* 1994;19:1475–82.
- 27 van der Wurff P, Buijs EJ, Groen GJ. A multitest regimen of pain provocation tests as an aid to reduce unnecessary minimally invasive sacroiliac joint procedures. *Arch Phys Med Rehabil* 2006;87(1):10–4.
- 28 Patel RK, Slipman CW. Lumbar spine disorders lumbar degenerative disk disease. *eMedicine Specialties Physical Medicine and Rehabilitation*, January 18, 2007.
- 29 Lamer TJ, Tiede JM, Fenton DS. Fluoroscopically-guided injections to treat “kissing spine” disease. *Pain Physician* 2008;11:549–54.
- 30 Langdon J, Way A, Heaton S, Bernard J, Molloy S. Vertebral compression fractures: New clinical signs to aid diagnosis. *Ann R Coll Surg Engl* 2010;92:163–6.
- 31 Jensen ME, Evans AJ, Mathis JM, et al. Percutaneous polymethylmethacrylate vertebroplasty in the treatment of osteoporotic vertebral compression fractures: Technical aspects. *AJNR Am J Neuroradiol* 1997;18:1897–904.
- 32 Evans AJ, Jensen ME, Kip KE, et al. Vertebral compression fractures: Pain reduction and improvement in functional mobility after percutaneous polymethylmethacrylate vertebroplasty. A retrospective report of 245 cases. *Radiology* 2003;226(2):366–72.
- 33 Grados F, Depriester C, Cayrolle G, et al. Long-term observations of vertebral osteoporotic fractures treated by percutaneous vertebroplasty. *Rheumatology* 2000;39(12):1410–4.
- 34 Barr JD, Barr MS, Lemley TJ, et al. Percutaneous vertebroplasty for pain relief and spinal stabilization. *Spine* 2000;25(8):923–8.
- 35 Frey ME, DePalma MJ, Cifu DX, et al. Percutaneous sacroplasty for osteoporotic sacral insufficiency fractures: A prospective, multicenter, observational pilot study. *Spine J* 2008;8(2):367–73.
- 36 Frey M, DePalma MJ, Cifu DX, Bhagia SM, Daitch J. Efficacy and safety of percutaneous sacroplasty for painful osteoporotic sacral insufficiency fractures. A prospective, multicenter trial. *Spine* 2007;32(15):1635–40.
- 37 Derby R, Kim BJ, Chen Y, Seo KS, Lee SH. The relation between annular disruption on computed tomography scan and pressure-controlled discography. *Arch Phys Med Rehabil* 2005;86(8):1534–8.
- 38 Wolfer LR, Derby R, Lee JE, et al. Systematic review of lumbar provocation discography in asymptomatic subjects with a meta-analysis of false-positive rates. *Pain Physician* 2008;11:513–38.
- 39 Lumbar disc stimulation (provocation discography). In: Bogduk N, ed. *Practice Guidelines. Spinal Diagnostic and Treatment Procedures*. San Francisco, CA: International Spinal Intervention Society; 2004:20–46.
- 40 Bogduk N, Holmes S. Controlled zygapophysial joint blocks: The travesty of cost-effectiveness. *Pain Med* 2000;1:25–34.
- 41 Manchikanti L, Pampati V, Fellows B, Bakhit CE. The diagnostic validity and therapeutic value of lumbar facet joint nerve blocks with or without adjuvant agents. *Curr Rev Pain* 2000;4:337–44.
- 42 Slipman CW, Issac Z. The role of diagnostic selective nerve root blocks in the management of spinal pain. *Pain Physician* 2001;4(3):214–26.
- 43 Moneta GB, Videman T, Kaivanto K, et al. Reported pain during lumbar discography as a function of annular ruptures and disc degeneration. A re-analysis of 833 discograms. *Spine* 1994;19(17):1968–74.
- 44 Walsh T, Weinstein J, et al. The question of discography revisited. A controlled prospective study of normal volunteers to determine the false positive rate. *J Bone Joint Surg Am* 1990;72:1081–8.
- 45 Buchbinder R, Osborne RH, Ebeling PR, Wark JD, et al. A randomized trial of vertebroplasty for painful osteoporotic vertebral fractures. *N Engl J Med* 2009;361:557–68.
- 46 Kallmes DF, Comstock BA, Heagerty PJ, et al. A randomized trial of vertebroplasty for osteoporotic spinal fractures. *N Engl J Med* 2009;361:569–79.

- 47 Langdon J, Way A, Heaton S, Bernard J, Molloy S. Vertebral compression fractures—New clinical signs to aid diagnosis. *Ann R Coll Surg Engl* 2010;92:163–6.
- 48 DePalma MJ, Beall DP, Frey ME, Kallmes DF, Prather H. Point/counterpoint: Vertebroplasty. *PMR* 2010;2: 862–7.
- 49 DePalma M, Ketchum J, Queler E, et al. What is the etiology of low back pain, and does age effect the prevalence of each etiology? An interim analysis of 170 consecutive cases. *Pain Med* 2009;10(5):949.