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## Research Article

### Discogenic Low Back Pain: A Pilot Study of Proposed Non-Invasive Diagnostic Parameters

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#### Abstract:

Discogenic low back pain (DLBP) accounts for 39% of chronic lower back pain (CLBP). Unfortunately, accurate diagnosis remains challenging, as clinical examination and magnetic resonance imaging (MRI) may be normal. Provocative Discography (PD) is one method of distinguishing DLBP from other back pain causes. Though technically safe it is considered to be an invasive procedure, and has been linked to latent acceleration of disc degeneration. It is thus reserved for surgical planning, leaving many patients definitely undiagnosed.

This dilemma, has prompted the development of various novel diagnostic approaches, such as intervertebral disc ultrasound, provocative electric vibration and the study of serological biomarkers. Though promising, perhaps the most useful diagnostic marker is the presence of a high intensity zone (HIZ) in the annulus in one of the discs in a CLBP patient. In this case, there is a positive predictive value (PPV) of 88- 90% that the lumbar disc is the pain generator.

It would appear however that the significance of the HIZ remains underappreciated and a poorly understood marker for the non-invasive diagnosis of DLBP.

This paper explores how symptoms, imaging, and examination findings when considered together, might further improve diagnostic accuracy of DLBP in a non-invasive manner.

Combined criteria are already in use for diagnosing of ankylosing spondylitis (AS) and rheumatoid arthritis (RA).

Though this series was small, the back pain symptom patterns and examination findings were consistent with a DLBP patterns anecdotally reported by pain clinicians experienced in discography.

Therefore the development of a more formal DLBP diagnostic system, using not only the presence of HIZ, but symptoms-examination and imaging findings may offer a more accurate diagnosis for CLBP sufferers who are not candidates for provocative discography.

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**Keywords:** Discogenic Low Back Pain, High intensity zone, Lumbar Spine, Diagnosis, Annular Tear, Annular Fissure.

#### INTRODUCTION

It has been claimed that low back pain of discogenic origin (DLBP) accounts for 39% of all chronic low back pain (CLBP) [Schwartz et al 1995] Despite this prevalence non-invasive diagnosis of DLBP remains challenging. It is fairly well accepted however, that CLBP and the presence of a lumbar high intensity zone (HIZ) on T2-weighted magnetic resonance imaging (MRI) is highly suggestive that internal disc disruption (annular tears or fissures) may herald discogenic pain. This concept, proposed by Bogduk, has been verified several times over by corroborative studies, which report that the HIZ has an 87-89% positive predictive value in diagnosing DLBP. [Schellhas et al 1996, Aprill et al 1992, Saifuddine 1998]. However the disputed significance of the HIZ in the context of CLBP has never been formally recognized.

Carragee who found a HIZ in 22% of asymptomatic backs disputed the significance of the HI. [Carragee et al 2000] However, Aprill and Bogduk's original work was never intended to apply to asymptomatic backs. They further concluded that the HIZ had even further value as an accurate predictor of pain- generating intradiscal disruption, heralding

at least a grade III- IV annular fissure. A more recent but less powerful study determined a 97 % correlation between the HIZ and discogram, exhibited by grade 3 or higher annular disruptions. [Hebelka et al 2013]

For non-discography patients, less-invasive diagnostic tools, including intervertebral disc ultrasound, provocative electric vibration (which may be combined with ultrasound), high-sensitivity C-reactive protein, and the development of serological biomarkers have been proposed. Nevertheless, these diagnostic alternatives are not widely in use.

However, the Mackenzie centralization phenomenon on physical examination is reported in the literature to be a viable indicator of DLBP, and has value as a predictor to gauge conservative management and surgical outcomes. [McKenzie et al 2003, Donelson et al 1990, Donelson et al 1997, Long 1995]

Criteria-based systems are already in use for the diagnosis of AS or RA. This paper proposes adopting a symptoms-imaging-examination Cartesian-like diagnostic approach to DLBP, which follows the evidence base and formally recognizes the HIZ's significance in the context of CLBP.

## PROPOSED HISTORICAL SYMPTOM MARKERS OF DLBP

Some authors contend that intervertebral disc degeneration symptoms are too variable and pain drawings therefore cannot be used to accurately diagnose discogenic pain. [Mann et al 1992, Ohnmeiss et al 1999]

The confidence in diagnosing DLBP may be greater in those below the age of 40, as in older patients there is potential overlap with lumbar facet and sacroiliac joint arthropathy. However, one clinician reported observing patients above the age of 65 as also being discography positive. Nevertheless, there do appear to be some unique symptom patterns, which are predictive of positive discography.

Some symptoms may include constant baseline LBP with episodic sharp pain flares of a VAS 6/10 or greater, and an inability to find a 'position of ease.'

The LBP may be described as unilateral or bilateral, deep, dull, achy; worsened by prolonged standing, sitting, twisting, and impact activity. The pain may be temporarily relieved by change in position but with no prolonged position of comfort. Typically, a low-grade LBP is constantly present with intermittent flares of > 6/10 occurring with seemingly trivial activity. LBP usually predominates over limb pain and pain may occur with or without referred lower limb pain.

Other authors have noted similar histories in athletes with DLBP, with histories of flexion/twisting injury and complaints of sitting intolerance. Pain is often confined to the lower back and may be described as deep-seated, diffuse, dull and achy. Coughing and sneezing may also increase pain. DLBP may be particularly challenging in student athletes who may spend the first part of the day sitting in classroom and then further irritate their lumbar discs later in the day playing sport. [Aspegren et al 2007]

Yu et al 2012, reported that the main clinical manifestations of DLBP included axial back pain (100%), pain in the region of the groin (33.3%), pain in the anterior or posterior region of thigh (42.2%), buttock pain (24.4%) and lower extremity pain (11.1%) [Yu] The diagnosis of DLBP is also perhaps more clear in those under the age of forty, where facet joint arthropathy and other potential pain generators can be excluded. Nevertheless positive discography has been observed in patients over the age of sixty-five. A family history of DLBP may also be an important marker. A family history of DLBP, smoking, repetitive high level impact sport, heavy manual labor, or significant LB trauma such as a fall from height may also be significant. [Battie et 1995]

## EXAMINATION FINDINGS

Waddell believed that conventional methods of examination could not differentiate DLBP from other potential pain generators. [Waddell 1996, 1997]

Yrjama et al 1994, reported provoking centralized back pain by ultrasound-guided blunt electrical vibratory shock of the spinous processes, describing it as a 'safe and effective tests' for DLBP. Yrjama also combined period after US imaging

Heller described a manual test, which he called the "digital interspinous pressure"(DIP) test. He believed it to be a test for

discitis, an irritated disc, with digital pressure. He described assessing the L3-S1 disc spaces as a routine part of prone patient exam. The pressure is applied with the edge of the thumb pad at the level of the interspinous space, pressing anterior and superior up toward the superior spinous process. Deep pressure is applied into the interspinous space, while attempting to lift the superior spinous process cephalad, this is provocative in DLBP. He reported that some patients are exquisitely sensitive to even mild pressure; others require substantial pressure to elicit tenderness. He theorized that if the annulus is completely torn, the disc container has lost its internal pressurization, so the test is likely to be negative. When the test is positive for tenderness, there is also a sense of restriction; a sense of vertical compression between the two contiguous vertebrae. [Heller 2016]

Perhaps the most recognized DLBP signs were developed by physiotherapist Robin McKenzie who in 1981, described the phenomenon of 'centralization', which occurs when referred pain moves from a distal to a more proximal location as a marker for DLBP. This phenomenon can be observed when a patient repeatedly bends backward and forward during a clinical examination.

For example, a patient presenting with pain referred into the calf may report that the calf pain reduces after bending backward into full extension a few times. Often, further movements in the same direction will lead to the pain migrating even closer to the spine. When the test movements have been completed, the distal pain remains reduced.

Based on his many years of clinical observation of patients with low back pain, McKenzie claimed that this phenomenon was a reliable indicator of a good clinical outcome.

The McKenzie procedure measures the symptomatic response to repeated end-range movements, with special attention to whether the pain centralizes or peripheralizes. Peripheralization is when midline back pain moves to the side of the back or to the buttocks. Further peripheralisation involves pain radiating down the leg. The further it moves down the leg, the more it is said to peripheralize. Centralization is the opposite. If the pain is no longer down the leg but now is only in the buttocks, it is centralizing. If centralization continues, the pain will recede towards the lumbar midline and eventually, with continued end range movements, may disappear.

With the McKenzie mechanical assessment procedures, the most common direction of testing that centralizes pain is extension, though some patients require lateral side gliding, and a few will centralize with repeated flexion. [McKenzie 2003]

McKenzie theorized the most likely reason for this centralization phenomenon is that the back and leg is caused by displaced nuclear disc material that is mechanically stimulating the pain-sensitive annulus or nerve root. This is referred to as the "dynamic internal disc model". As long as the annulus and the hydrostatic mechanism of the disc are intact, repeated end range loading of the spine (repeated movements) can return the displaced nuclear material, thus centralizing and reducing the pain. If no directed movements

are able to centralize the pain and if multiple movements result in peripheralization of the pain, then it is theorized that the annulus is torn and that the hydrostatic mechanism of the disc is no longer functioning.

Some studies have re-visited the McKenzie procedure for assessing patients with low back pain and have found it to be more accurate than MRI in differentiating discogenic from non-discogenic pain as well as contained from non-contained discs. [Donelson 1990, 1997]

Other studies re-affirmed McKenzie examination findings as correlating with positive provocative discography [Wetzel et al 2003, Laslett et al 2005]

It was also reported that DLBP patients who centralized had better outcome with work hardening programs and rehabilitation than those that did not. [Long et al 1995]

Donelson et al 1990 and 1997, also concluded that a nonoccurrence of centralization accurately predicts poor treatment outcome and was a helpful early predictor of the need for surgical treatment.

It was however noted that surgical patients who presented with centralization pre-operatively, returned to work quicker and had greater surgical pain relief and satisfaction scores. [Karas et al 1997, Werneke et al 2001]

In summary it would appear that provided patients are able to tolerate repetitive lumbar spinal movements on examination, the centralization phenomenon is not only a useful clinical examination marker for DLBP, but can accurately predict both rehabilitation and surgical outcomes.

Some less direct studies have looked at the potential of finding high circulating inflammatory markers such as IL-6, IL-8 and high-sensitivity CRP (HsCRP), as correlating with DLBP. One study concluded a useful diagnostic association between HsCRP and Modic 1 vertebral body changes. [Rannou et al 2007]

Elevated HsCRP was found in acute LBP with sciatica, though it was wasn't found to be elevated in the context of chronic LBP. [Sturmer et al 2005]

Other researchers have looked for plasma protein biomarkers. [Takahashi et al 2001, Videman et 1998, Annunen et al 1999]

Though there are currently no accurate DLBP lab test predictors, the absence of elevated inflammatory markers and positive auto-immune tests, may be an important part of excluding other conditions.

## **IMAGING**

### **X-RAYS**

Weight-bearing lumbar spine x-rays may have utility as an inexpensive and readily available initial screen. By offering a truer picture of the spine's position under load, they may detect indirect signs of degenerative disc disease (DDD) that might contribute to DLBP. They may also exclude other potential pain generators such as scoliosis, fractures, gross segmental instability, facet joint arthrosis, congenital anomalies, pars defects, and degenerative spondylolisthesis. [Richards et al 2007]

Radiographic parameters might even be considered better for staging of disc degeneration than MRI.

[Frobin et al 2001]

In addition to the HIZ, Modic changes have a high value in the diagnosis of lumbar discogenic pain based on the multivariate logistic analysis. Good diagnostic accuracy was obtained from using diagnostic factors including lumbar instability (Angular motion, more than 14.35°) in the radiographic diagnosis of DLBP. [Song et al 2013]

### **MRI**

The role of MRI in diagnosing DLBP is not without controversy. The absence of an HIZ does not the exclude an annular tear. Studies have shown normal or equivocal lumbar MRI have positive discography, with internal disc disruption (IDD) confirmed surgically. [Brightbill et al 1994]

Nevertheless, a finding of lumbar HIZ in the context of a DLBP pain pattern, is considered by several authors to have high diagnostic value. [Schellhas et al 1996, Aprill et al 1992, Saifuddine 1998]

One must also not overlook the importance of absence of other potential pain generators which may by a process of exclusion, guide the clinician to suspect DLBP.

### **SPECT SCAN**

Radio isotope bone scans such as scintigraphy and single Proton Emission Computer Tomography (SPECT) have both been used to evaluate facet joint inflammation and identify targets for guided injections. [Bush et al 2007]

However, hybridization of SPECT-CT has overcome some of the prior issues of specificity.

In patients after post disc-replacement surgery, discography is impossible at the level of previous surgery. In this situation, SPECT-CT may currently be a valuable tool in identifying adjacent level DLBP. [Miller et al 2012]

### **ULTRASOUND**

Naish et al 2003, reported on the diagnostic abilities of ultrasound to identify intradiscal spinal anomalies in canines.

In another study trans-abdominal ultrasound (US) imaging was compared with computed tomography (CT)/discography in order to assess whether the former could be used for screening discs to be examined by CT/discography, or could be used to replace CT/discography. A total of 56 discs in 29 patients was examined by both methods. The US findings were classified as local or generalized disc lesions, and the CT/discography findings according to the Dallas Discogram Description. The sensitivity of US for recognizing a discogram positive disc was 0.95, and its specificity was 0.38. The authors concluded US to be a suitable screening modality for lumbar disc disease prior to CT discography. [Tevonen et al 1991]

### **METHODS**

In our study, patient groups from two different clinics presenting with CLBP and findings of HIZ on lumbar MRI imaging were reviewed to look for any characteristic patterns of history and examination findings.

Retrospective data from the first group was gathered from 2012 to 2015 on a case series of eleven male patients who had

been evaluated in a rehabilitation centre, with chronic lower back pain in whom T2-weighted MRI scan confirmed lumbar disc annular tears. An annular tear was defined as a fissure or focal hyperintensity within the posterior part of the annulus fibrosus without focal extrusion on T2-weighted imaging.

The history, symptoms, clinical examination and imaging findings were reviewed to determine any commonality.. Exclusion criteria included widespread myofascial pain, spinal stenosis, inflammatory spinal arthropathy, prior spinal fracture, and a history of spinal surgery, pelvic pain, serious somatic or psychiatric illness. The assessment were performed by a single musculoskeletal medicine physician with more than 10 years experience, and a senior physiotherapist.

Patients from the other clinic group had been initially reviewed, examined and treated, by another musculoskeletal physician with more than 20 years experience. Retrospective histories were gathered via telephone survey questionnaires, from 14 CLBP patients with T2-weighted MRI-reported lower lumbar disc annular tears, that had attended between 2011-15.

**RESULTS**

One schizophrenic patient being treated with anti-psychotic medications was excluded from the first group. The 10 remaining patients of this group were all males aged 20-42, with a history of CLBP for 3 months to 8 years duration. The mean duration of CLBP was 2.8 years at the time of evaluation. All were able to tolerate complete examination. The mean age of patients was 32.2 years. Their mean height was 178. Cm. with a mean weight of 80.4 kg and a mean body mass index of 25. 2.

All 10 patients reported generalized lower back stiffness with acute flares of episodic sharp and centralized LBP of greater intensity than leg pain, of seemingly random intermittent occurrence, but specifically flared by lifting activities and running.

Nine out of ten of the group reported constant centralized LBP, intolerance to prolonged sitting and standing, which was relieved by lying down and eased by light exercise.

The same percentage reported difficulty in performing activities of daily living.

Eight reported morning lower back stiffness, symptoms relieved by pain medications, and a history of gait and sleep disturbance. Seven recollected an acute onset and six reported onset following acute injury.

Mood changes and depression were reported in 2 patients. Five patients were smokers. Five reported a lower limb paraesthesia; four reported pain flares with coughing or sneezing and three reported referred buttock pain. Two patients reported a history of LBP of such significance that it prompted a visit to casualty for acute pain relief. Two patients reported relief of symptoms with moist heat and prescription of insoles.

On examination, all reported focal centralized segmental tenderness upon either lumbar springing, digital palpation of the interspinous ligaments, and/or lateral flexion-rotational provocation. Examination resulted in increased pain on lumbar

flexion, and LBP increased with passive straight leg-raise testing.

Eight also had a positive seated slump test reproducing lower back and leg symptoms. Only one of the 5 patients with lower limb or buttock radicular pain reported centralization of LBP symptoms on repeat lumbar range of motion testing.

Identical exclusion criteria was applied to patients from the second clinic, so that two patients were exempted due to a history of discectomy. Four others were non-contactable. Of the remaining 9 patients, 5 male and 4 female patients. Their ages ranged between 29-52 years with a mean age of 43 years. Duration of Group 2’s CLBP ranged from circa 9 months to 25 years, with a mean symptoms duration of 8.1 years. Anthropomorphic and exam data was not available for the second group. All however, described characteristic symptoms as outlined in Category ‘History and Symptoms,’ in the table listed below.

On lumbar MRI, all of the patients had reported annular tears at either the L4/5 or L5/S1 levels. Seven of the patients had concomitant degenerative disc disease while six also had same-level disc bulge or protrusion.

Schmorl’s nodes, facet joint arthropathy, and spondylosis were present in two patients. One patient also had ligamentum flavum hypertrophy and pars defects. All patients imaging was negative for inflammatory lower back pain.

Four patients could not recollect a mechanism of injury, while three experienced chronic LBP after a fall. Positive vertebral percussion tenderness was noted in three. Two patients had some signs of hypermobility (mean Beighton score 4.5).

**Table 1. Proposed History-Examination-Imaging Parameters for DLBP**

Category 1	HISTORY & SYMPTOMS
	<p><b>Lower back stiffness with random recurrent acute flares of episodic sharp pain or exacerbated by lifting, twisting impact activity, prolonged sitting or standing &gt; 10 minutes.</b></p> <p><b>Chronic, constant, centralized unilateral or bilateral, deep, dull, achy LBP, with intermittent sharp flares worsened by prolonged sitting, standing, lifting, twisting, and temporarily relieved by change in position but with no prolonged position comfortable and with or without lower limb radiculopathy in a patient less than 40 years of age.*,**</b></p>
	<p><b>Primary symptoms in a patient greater than 40 (Concomitant or other causes of CLBP are more likely e.g.: facet joint arthropathy, paraspinal muscle atrophy and core instability</b></p>
	<p><b>LBP predominates over lower limb pain</b></p>

	<b>Family History of DLBP</b>
	<b>Onset of Primary symptoms at age 20-25 [Ishihara et al 1996, Horner et al 2001]</b>
	<b>LBP causing temporary gait disturbance</b>
	<b>Smoker</b>
	<b>History of repetitive high level torsional or impact sport, heavy manual labor, Significant LB trauma, such as a fall from height or history of repetitive/cyclic work exposure to heavy lifting, and/or repetitive trunk torsional loading. [Norman et al 1998, Kelsey et al 1984] Flexion extension or impact activity [Elliot et al 2002], contact sports, gymnastics. [Goldstein et al 1991, Hangai et al 2009]</b>
<b>Category 2</b>	<b>EXAMINATION FINDINGS</b>
	<b>Centralization phenomenon-[MacKenzie et al 2003]; Digital palpation of the interspinous ligaments, repeat movement testing [Heller 2016]</b>
	<b>Normal exam</b>
	<b>A positive seated slump test reproducing lower back &gt; leg symptoms</b>
	<b>Hypermobility syndrome [Toyone et al 1994]</b>
<b>Category 3</b>	<b>IMAGING FINDINGS AND LABORATORY RESULTS</b>
	<b>Lumbar HIZ on T2-weighted MRI scan</b>
	<b>Lumbar DDD, Modic Changes [Song et al 2017]</b>
	<b>MRI excludes other potential pain generators such as disc protrusion, congenital anomalies, pars defects. Note: Normal scan may not exclude a peripheral annular tear [Osti 1992]</b>
	<b>MRI Normal</b>
	<b>Standing lateral lumbar x-ray findings of decreased disc space correlating to the level of pain. Congenital anomalies (tropism/ lumbalization), , Segmental instability correlating to level of LBP [Song et al 2017]</b>

Psychiatric illness, overt biopsychosocial overlay severe disability, inflammatory spondyloarthropathy excluded.

\*\* These proposed markers further elaborate established Mackenzie Delphi examination criteria. [Chan et al 2003]

**DISCUSSION**

Patients with DLBP may have a completely normal presentation on clinical examination, and may also have unremarkable imaging. However, the Mackenzie centralisation phenomenon when present and exam is tolerated, appears to be a noteworthy predictor of DLBP. [[McKenzie et al 2003, Donelson et al 1990, Donelson et al 1997, Long 1995]. Despite this evidence, controversy remains. Some of our

colleagues still follow the Waddell approach, and continue to regard lower back pain as ‘enigma.’ However, Waddell did however recognize patient dissatisfaction, poor management outcomes, and the need for better CLBP managements. [Waddell 1996, 1997].

Clinicians may well tell patients suffering severe and CLBP for years that there pain is ‘non-specific’, untreatable, and has no determinable causation. This could contribute to reported high patient dissatisfaction and poor prognosis. It may also exacerbate biopsychosocial obstacles to recovery. Providing patients with an accurate evidence-based diagnosis can help improve patient satisfaction, understanding of chronic pain, and will encourage more active management, while diverting from ineffective and expensive treatments.

As also noted from comparative cadaveric studies [Gunzburg et al 1992], a normal MRI does not exclude significant changes in the peripheral structure (so-called ‘rim lesions’ of the intervertebral disc which can produce lower back pain. By examining histological samples, some of these same authors also concluded that peripheral annular tears were due to trauma rather than biochemical degradation, and that they developed independently of nuclear degeneration. [Osti et al 1992]

Though the authors of those papers could not comment on the relationship of these rim lesions to CLBP, traumatic ‘rim lesions’ would probably be more common in active young adults, particularly those participating in sports requiring torsional moments and repetitive flexion-hyperextension activities. Gymnasts frequently sustain low back injuries secondary to hyperextension of the spine during vaulting, walkovers, and dismounts. Goldstein et al, found that 25% of pre-elite gymnasts, 43% of elite gymnasts, and 63% of Olympic gymnasts had MRI abnormalities including spondylolysis, spondylolisthesis and disc disease. [Goldstein et al 1991]

Therefore, an athlete presenting with a DLBP history but normal looking discs on MRI may have a rim lesion annular tear.

Some researchers have also noted a high prevalence of HIZ in asymptomatic backs. [Carragee et al 2000] Bogduk cautioned however that the 88% specificity of the HIZ in predicting discogenic lower back pain could only be applied to patients with chronic lower back pain. [Aprill et al 1992] There are additional MRI and x-ray imaging findings such as Modic change, and spinal instability that also merit attention. [Song et al 2017]

By assigning significance to history-clinical examination-imaging and perhaps other markers, spinal clinicians may devise a criteria based system of diagnosis as for AS and RA. This study reviewed 25 patients with CLBP and confirmed lumbar disc annular tear on MRI imaging. These case histories presented patterns closely paralleling some of the signs and symptoms described in prior PD-confirmed cases. [McKenzie et al 2003] An attempt is also made here to begin the process of formulating workable non-invasive diagnostic criteria for lower back pain of discogenic origin.

Though this series was small, there are threads of

commonality in clinical presentations and histories, which parallel patterns anecdotally reported by experienced spinal clinicians. [McKenzie et al 2003] Markers for examination findings are Mackenzie based, [Long et al 1995] and criteria such as family history are based on genetics. Twin-study research is the subject of larger studies. [Battie et al 1995, Kauppila et al 1994]

Unlike the majority of disc herniations, which have a capacity to resolve with time (average 18 months), [Bush et al 1992], annular tears have a poor capacity for healing and may produce symptoms indefinitely. Therefore, 18 months may be the chosen cut-off point to delineate primary DLBP that had achieved maximum anticipated symptomatic improvement. DLBP may also occur with or without radiculopathy and in the absence of lower limb dural tension signs, and patients also may present with unremarkable clinical examination findings.

One may take a Jacques Derridian perspective, focusing on the significance of the 'presence of absence'. [Derrida 1974] For example, clinical examination and MR imaging in DLBP may be normal, but a negative finding in itself may be important as an aid to the exclusion process. Therefore the absence of any other imaging findings (facet joint degeneration, mechanical nerve root compression) may be relevant in ruling out other potential pain generators.

This research may offer evidence base for consideration of history-exam-imaging diagnostic criteria. Twin studies have identified genetic heritage as being primarily causative, with smoking, lifestyle habits, occupational overload, psychological stress, anthropomorphic variations, hypermobility and prior spinal trauma as ancillary factors. [Battie et al 1995, Toyone et al 1994].

Other studies have defined an association with vitamin D receptor polymorphisms and metalloproteinase mutations, [Videman et al 2001, Takayashi et al 1998], and rare but important associations between ColA8 and ColA9 genetic mutations and pathological disc degeneration. [Annunen et al 1999]

Starvation of amino acids, oxygen, and glucose affects the viability of glycosaminoglycan producing disc cells, which in turn inhibits the production of larger fluid-imbibing aggrecan and aggregate molecules. [Buckwalter 1995, Lyons et al 1981] Deprived of these molecules, the once youthful viscoelastic nucleus pulposus (NP), which at one time held 80% water, [Bogduk 2008, Beard et al 1980] rapidly undergoes osmotic pressure loss and begins to dehydrate. Desiccation alters disc structure, volume, and height, compromising resistance to axial load, with subsequent degeneration and annular defect formation.

As degeneration advances, the once well-defined boundaries between the NP and the annulus fibrosis become blurred, with compressive forces redistributed to the apophyseal ring, facet joints, and the pain sensitive outer one-third of the annulus. Degenerative disc-pain may therefore arise from other structures (some of which are also innervated by the sinuvertebral nerve), as well as the associated paraspinal musculature, ligaments, tendons, facet joints, and vertebrae,

[White et al 2009] complicating the diagnostic picture.

Fremont's work offered yet further pathoanatomical evidence of the origins of DLBP, via pre-surgical disc biopsy. [Frobin et al 2001]. Biopsy findings implied ectopic sinuvertebral nerve growth from the outer one-third of the annulus sometimes even into the NP itself. [Fremont et al 1997]. These findings, which were later confirmed immunohistochemically [Coppes et al 1997], may offer explanations regarding axial spinal load pain intolerance.

Due to the potential acceleration of disc degeneration following provocative discography, [Carragee et al 2009] this procedure is generally reserved for intractable pain patients who are candidates for lumbar interbody fusion or disc replacement procedures. Some authors hold the position that though provocative discography might help surgical decision-making, it is not a gold standard for diagnosing DLBP, and its potential side effects make it an unsatisfactory technique for DLBP diagnosis. [Zhang et al 2009]

For most CLBP patients, lack of a system for weighting clinical and imaging findings renders diagnosis challenging, and negative provocative discography does not necessarily exclude DLBP. [Yu et al 2012]

Some experienced clinicians have reported unique symptom clusters that prompt diagnostic suspicion of DLBP. Furthermore, the absence of findings clinically and on imaging can guide diagnosis by exclusion. Centralization on repeated lumbar movements, (Mackenzie evaluation), is also highly predictive for positive discography, but specificity is reduced in the presence of severe disability or psychosocial overlay. [Laslett et al 2005] Weighted symptom criteria or point-based scoring systems have evolved over time and are in common use for other conditions (AS and RA), granting proven diagnostic utility where laboratory tests and imaging findings may be inconclusive. [Kataria et al 2004]

This paper proposes that the challenges in defining DLBP non-invasively, may be clarified by using a symptoms- based diagnostic approach.

It is hoped however, that formally correlating historical symptoms, imaging findings [Kendrick et al 2001] (or the lack thereof), refined examination methods (Mackenzie/Heller), and laboratory findings (serum biomarkers), may sum up to offer a more accurate non-invasive DLBP diagnostic approach. Likewise, exclusion of gross biopsychosocial factors, and negative laboratory findings may improve the diagnostic certainty, particularly in cases of un-resolved lower back pain beyond 18 months duration. [Bush et al 1992, Inklebarger et al 2015, Anderson et al 2005]. Inflammatory back pain should also be excluded particular in those younger than forty.

There are those who consider that 'provocation discography is still the only available means by which to identify painful discs.' [Peng 2013]

The evidence base tells us however, that this may not necessarily be the case. A criteria based probability system may help to improve DLBP accuracy without discography. Reporting a more accurate diagnosis may improve patient satisfaction, compliance, and guide better management,

potentially steering patients away from costly, time-consuming, unnecessary, and invasive procedures. An accurate diagnosis of DLBP may also guide early and specific rehabilitation, particularly in acute cases. For example, athletes are at a high risk of IDD because of repetitive axial compressive loading. Symptomatic annular tears commonly responds to aggressive conservative care designed in a five-stage rehabilitation program. [Cook et al 2001]

## CONCLUSION

As other criteria-based diagnostic systems, development of a DLBP system would probably be useful, but would require combined international specialist input to gain acceptance. Further audit, Delphi consensus, and research is therefore essential.

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