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Neck and back pain and intervertebral disc degeneration: Role of occupational factors

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Back pain is a near-universal human experience at some time during life, and neck pain is also common. The overwhelming majority of low back and cervical pain is considered to be due to unspecified mechanical factors or disc degeneration, which is a common with ageing and, hence, in people of working age. Back pain and disc disease appear to have significant heritability, based upon twin studies, but environmental factors also contribute – including physical occupational activities in some studies – although the strength of this association remains uncertain. This article examines the contribution of genetic and environmental factors to back pain and disc disease, with a specific focus on occupational exposures.

Back pain is highly prevalent in the Western world with a lifetime prevalence reaching up to 80%, and accounts for considerable work absenteeism. Low back pain (LBP) has been studied more extensively than neck pain, and a number of studies have shown a relationship between LBP and lumbar disc degeneration (LDD), although the strength of this association remains unclear [1–4]. Following a general description of the epidemiology of cervical and lumbar pain and degenerative disc disease, this article focusses on the role of occupational factors in causation of cervical and lumbar pathology and factors that might mitigate risk. Areas of uncertainty and research interest are emphasised, where relevant. Attention is given to risk factors at the workplace and prevention of work-associated cases.

Some degree of LDD is almost universal in adults with ageing [5], and has been repeatedly proposed as one of the main causes of LBP [6–8]. Although an association with LDD was first clearly demonstrated in twin studies [4], and more recently confirmed in a systematic review [9], specific risk factors for LBP remain unclear and universal consensus regarding the strength of the LDD–LBP relationship has

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been variable [3,10–12]. In addition to occupation, other environmental factors, such as obesity and smoking, have been reported to be associated with prevalent LBP, although the quantitative effect of the majority of these has been found to be small [4,13–16]. On the other hand, several twin studies have consistently suggested the presence of a significant genetic component underlying variation in both LBP and LDD [4,17,18].

Clinical features

Classically, the syndrome of LBP related to LDD comprises somatic referred pain with or without radiculopathic features, together with abnormalities on imaging.

However, because neck and back pain are intermittent symptoms, a number of studies have reported only relatively modest relationships between back pain and abnormal radiology whether imaged by plain X-rays, computed tomography (CT) scanning or magnetic resonance imaging (MRI). A recent systematic review [9] suggests that the odds ratio (OR) for an association with LBP is strongest for disc protrusion (OR 3.6), followed by disc degeneration (OR 2,5), annular tears (OR 2.5) and nerve root compression/displacement (OR 2.3).

Case definitions and diagnosis

The lack of standardised clinical criteria and radiological definitions has hampered the undertaking of well-executed epidemiological studies. When LBP is defined by severity of symptoms alone rather than disability as well, there appears to be considerable heterogeneity in the underlying aetiology [9]. More sensitive imaging modalities, especially MRI, have become gradually more widely available and facilitated larger-scale epidemiological studies of LDD. A number of studies have provided estimates of the association between disc degeneration and LBP [4,6,19–25].

Epidemiology

The traditional view of intervertebral disc degeneration (IDD) has been that it was a process related to normal ageing as well as changes related to physical loading over the lifetime. Fundamental to any description of the epidemiology of IDD, however, is the definition of a case and how to measure these. There is no standard definition of back pain or disc degeneration and, for this reason, comparison between studies is difficult. Operationally, disc degeneration is defined largely by the method of evaluation. For example, radiography or CT scan can most usefully assess disc height and osteophytes, whereas MRI can better assess disc signal and structural change, such as prolapse or herniation. For large population studies, the preferred method is MRI, and most systems of evaluation although qualitative, include assessment of disc height, signal intensity, bulging or prolapse and osteophytes. The prevalence rates of these measures differ considerably between studies. For example, the prevalence of lumbar disc bulge ranges from 10% to more than 80% in so-called asymptomatic subjects, and the prevalence of annular tears from 6% to 56% [26]. The prevalence of protrusion ranges from 4% to 76%, and the prevalence of disc degeneration, usually defined by the presence of disc height loss and/or reduced T2 signal intensity, is estimated at 54% [9].

There also appears to be variation in the prevalence of MRI changes at the five different lumbar disc levels with, in general, the lower levels having the highest prevalence of disc changes (Fig. 1), with the exception of Schmorl's nodes, which are more common in the upper lumbar and thoracic region [27]. For these reasons, there is debate regarding the use of summary scores to describe LDD, which have been advocated by some [4] but not others [28].

As noted above, there is no agreement regarding the most appropriate definition of LBP and neck pain for use in population studies. Definitions that do not take into account pain intensity or disability may overestimate pain that is of little or no public health importance. McGregor et al. [4] focussed on pain with a duration of greater than 1 month, accompanied by distal radiation and associated with disability. This definition of pain related to the lifetime prevalence of pain (that is report of pain "ever") rather than to the 1-year period prevalence, which is more commonly used in case-control studies. For LBP using this more conservative definition, the lifetime cumulative prevalence of duration longer than

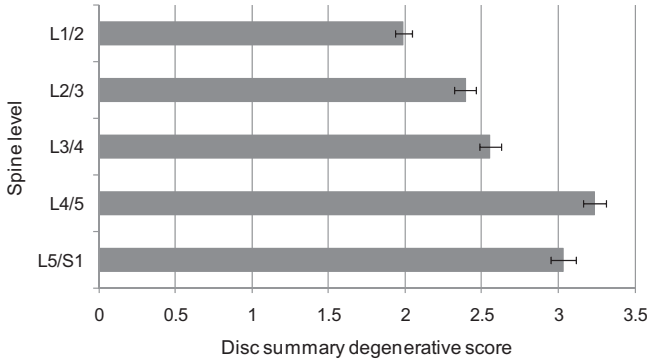

Fig. 1. Mean summary scores for disc degeneration by spinal level based on MRI grade (adapted from Sambrook et al., 1999 [17]).

a month associated with severe disability was 14% [4]. The prevalence of neck pain, using an equivalent definition, was 8%.

By contrast, a systemic review that used more liberal definitions has estimated the 1-month prevalence of LBP to be 30% and the 1-year prevalence to be 50%, and the combined prevalence from three population studies was 67% [6,20,21,29,30].

The progression of LDD over time has been investigated by Videman et al. [31], who investigated 140 male monozygotic (MZ) twins, and studied a variety of MRI traits over 5 years. They observed that there were slow changes in LDD over time with the different subtraits progressing at different rates. Borenstein et al. [2] performed MRI scans on 50 subjects, who had no history of back pain, as baseline (mean age 43.6 years) and repeated the MRI 7 years later in 31 subjects (mean age at follow-up 52 years). Over 7 years, 21 subjects developed back or leg pain, including 12 with a normal scan, five of six subjects with a herniated disc and three of four subjects with spinal stenosis. With regard to the ability of MRI to predict LBP, a positive trend was noted in each disease category, but none was significant; however, with this small sample, the study was underpowered to a large extent. Hassett et al. reported progression rates of 3.9% for anterior osteophytes and 3.2% for disc- space narrowing using plain radiography in 914 female volunteers [32]. The UK Twin Spine Study has performed a 10-year follow-up on 460 twins, and has shown that while all MRI subtraits deteriorated significantly over time, there appears to be little influence on the episodes of severe and disabling back pain reported at the two time points.

Genetic studies

Twin studies have provided considerable information about the role of genetic influences on LDD. Battie et al. [33] studied 115 identical male twins selected for discordance in suspected environmental risk factors, such as smoking. In multivariate analyses, job code explained 7% of the variability in the upper lumbar (T12–L4) degenerative summary score, the addition of age explained a further 9% and when twinship was added, the amount of variability explained rose to 77%. In the lower lumbar spine, heavy leisure-time physical loading explained only 2% of the variability, age 9%, and with twinship, 43% of variability was explained. Although these studies in identical twins suggest a genetic effect, a strong effect of the family environment cannot be discounted. Unfortunately, non-identical twins were not included in the study, making interpretation of the heritability impossible.

To determine heritability of LDD, Sambrook et al. [17] used a cohort of mainly female UK and Australian twins unselected for back pain, and reported that overall heritability was 74% for LDD and 73% for cervical disc degeneration. Examination of individual phenotypic features revealed that disc height and bulge were highly heritable at both sites (59–79%), with osteophytes heritable in the lumbar spine (59%). Osteophytes were not shown to be heritable in the cervical spine, but it must remembered that MRI is not the optimal imaging modality for assessing bony changes, and cervical spine MRI

images are smaller than the lumbar spine images, making accurate assessment of this subtrait a challenge. It must also be recognised that twin studies can sometimes produce an exaggerated estimate of heritability due to certain biases, most commonly selection bias, whereby subjects with symptoms self-select for a study, or due to an imbalance in environmental factors common to a pair, identical twins have greater similarity in environmental factors that are risk factors for a disease. Sambrook et al. [17] showed there was greater concordance in identical twins for certain factors, such as occupational manual work and smoking; however, adjustment for these did not diminish the estimates of heritability, suggesting their effects were small. On the other hand, the number of males in the study was too small to assess definitely any differences between males and females; hence, some caution may be necessary in extrapolating these results, to assess occupational effects in men.

With regard to back pain, two earlier questionnaire-based surveys conducted in the Nordic twin registries, using non-standard case definitions, had produced conflicting results. A relatively small genetic influence on LBP was observed by Heikkila et al. in a Finnish survey for 'sciatica' diagnosed by a physician (where heritability was estimated at 21%) [34]. A larger genetic contribution (heritability 50%) was observed in a survey conducted in Finland for a single question relating to back pain resulting in absence from work [35]. Neither study included objective measures of disc degeneration or covariates.

Using the same, large, unselected, twin population studied as Sambrook et al. [17], McGregor et al. [4] reported a significant genetic effect on LBP and neck pain with estimates of heritability ranging from 52% to 57% for LBP and from 35% to 48% for neck pain. The sources of variation were complex, with major contributions from structural MRI changes for LBP (OR 3.63 for upper quartile of MRI grade), body mass index (OR 2.34 for upper quartile) and smoking (OR 1.60 for "ever smoking"). In this study, MRI change was the strongest single predictor of LBP, whereas for neck pain, psychological distress, smoking and age were more important [4].

Williams et al. [27] have studied Schmorl's nodes in twins and found them in 30% of subjects at any vertebral level, with multiple Schmorl's nodes in 14% (with equal to or more than two nodes). In 9288 vertebral end plates, 374 Schmorl's nodes were found, 153 (41%) in the lumbar spine and 221 (59%) in the thoracic spine. The heritability of Schmorl's nodes was over 70%. There was a positive association between Schmorl's nodes and LDD. Schmorl's nodes were more frequent in subjects with back pain, but this association was explained by the association of Schmorl's nodes with LDD, and Schmorl's nodes were not themselves an independent risk factor for back pain.

A number of subsequent studies have studied candidate genes for LDD, and have identified a number of variants, including Vitamin D receptor (VDR); collagen, type IX, alpha 2 (COL9A2); collagen, type IX, alpha 3 (COL9A3); collagen, type I, alpha 1 (COL1A1); matrilin3 (MATN3); and matrix metalloproteinase (MMP), which have been replicated in some studies, and some inflammatory genes, such as IL-1 and THSD2 [36]. However, little is known of the genetic or environmental factors governing the rate of progression of LDD.

Williams et al. [37] have recently reported linkage studies in 348 women from the Twins UK register, who had undergone spine MRI scanning 10 years earlier. Significant linkage peaks (defined as maximum log of odds (LOD) > 3) were identified for LDD at three chromosomal regions. These included chromosome 1 (position 285 centimorgan (cM)), chromosome 5 (position 175 cM) and chromosome 19 (position 80 cM), close to a peak previously obtained for hand osteoarthritis. The peak on chromosome 19 had an LOD score of 4.06, and the empirical $p = 6.7 \times 10^{-4}$ confirmed reliability of the linkage signal.

Solovieva et al. [38] studied 135 middle-aged men, and reported that the Trp3 allele of the COL9A3 gene plus obesity acted synergistically to increase the risk of a dark nucleus pulposus and posterior disc bulge, and decreased disc height on MRI. They estimated that 45–71% of disc degeneration in persistently overweight individuals with the Trp 3 allele could be attributed to this interaction. These investigators [39] further examined the association between collagen and IL-1β gene polymorphisms and LDD in these same 135 middle-aged occupationally active men. The Trp 3 allele of the COL9A3 gene increased the risk of signal loss on MRI in the absence of the IL-1 βT allele (OR 7) and degenerative changes (OR 8); however, there was no effect of the Trp3 allele in the presence of the IL-1 βT allele. Carriers of the Col11α2 minor allele also had a modest increased risk of disc bulges (OR 2.1) as compared with non-carriers.

In a recent review, Zhang et al. [40] concluded that LDD was not only regulated by multiple genes but also by environmental factors, with gene–gene and gene-environment interactions likely to be present. They concluded that although genome-wide screening had identified a number of genes, each gene appeared to make only a small overall contribution to explaining LDD, and that long-term studies with larger sample sizes were needed.

Occupational associations

In the UK, approximately 20 million working days are lost each year because of LBP, and the latter accounts for 40% of the time lost due to industrial injury [20].

Although the best evidence for assessing the influence of risk factors on disease comes from population-based, prospective, cohort studies, with risk exposures determined at baseline, in practice, a case-control design is more frequently used, as this approach is more feasible. In such a study design, accurate retrospective assessment of occupational exposure is difficult, and usually relies on self-reporting of such exposures rather than objective measures. The other problem in assessing the relationship between occupational exposure and LDD is that there is likely to be a prolonged asymptomatic lead time and, indeed, back pain is usually an intermittent phenomenon – unlike the radiological changes – which are gradually progressive. In these studies, occupational exposure has generally been assessed by categories of activity, for example, heavy lifting, frequent bending or broad occupational categories, such as clerical versus heavy manual labour or truck driving. End points examined have ranged from symptomatic back pain to radiological change or surgical end points for disc prolapse.

One of the most comprehensive studies of the relationship between LBP and occupation was conducted by Lotters et al. [41] The authors performed a systematic review of the literature and calculated the pooled prevalence of LBP in an unexposed population and the pooled OR for each work-related risk factor in a meta-analysis, using a random effects model. An unbiased risk estimate for each risk factor was obtained by correcting the pooled OR for confounding by other risk factors. The pooled prevalence for LBP among unexposed subjects was 22%, 30% and 34% for the <35-year, 35–45-year and >45-year age categories, respectively. The pooled OR was 1.51 (95% confidence interval (CI) 1.31–1.74) for manual materials' handling, 1.68 (95% CI 1.41–2.01) for frequent bending or twisting, 1.39 (95% CI 1.24–1.55) for whole-body vibration and 1.30 (1.17–1.45) for job dissatisfaction. For high exposure to manual materials' handling, frequent bending or twisting and whole-body vibration, the pooled OR was 1.92, 1.93 and 1.63, respectively. They proposed an algorithm for calculating the level of work-relatedness of LBP using the aetiological fraction (Fig. 2). To determine the likelihood of work-relatedness for LBP dichotomously, they proposed a cut-off point of 50%, meaning that, if 50% or more of the calculated probability was due to occupational exposure, the LBP could be regarded as work related, noting that an aetiological fraction of 50% is often used in decision making, for example, in compensating lung cancer patients occupationally exposed to asbestos.

With regard to structural changes on MRI or CT, heavy physical loading, particularly related to occupation, has long been suspected to be a risk factor for LDD, and, generally, most studies have found an association between heavy physical loading and disc degeneration, although the strength of association has varied.

As noted above, Battie et al. [33] studied identical male twins selected for discordance in suspected environmental risk factors. In univariate analyses, heavy lifetime occupational loading in terms of materials handling and positional loading was consistently associated with greater disc degeneration, particularly in the upper lumbar region. Lifetime mean job codes indicating heavy as regards materials' handling and positional loading were associated with greater summary scores of this degeneration ($p = 0.001$). Similarly, mean lifetime occupational lifting was associated with greater degeneration ($p = 0.049$), as well as time spent in bent and twisted postures. Interestingly, decreased signal intensity was significantly related to greater leisure-time physical loading, supporting an environmental influence on disc signal. However, in multivariate analyses, job code explained only 7% of the variability in the upper lumbar (T12–L4) degenerative summary score. In the lower lumbar spine, heavy leisure-time physical loading explained only 2% of the variability. They concluded that genetic factors were more important, as routine heavy physical loading demands at work and leisure explained only a minor

Risk factors	Score if risk factor present		Score
	Exposed	Highly exposed	
• Lifting or manual materials handling	+4	+7	.....
• Frequent bending or twisting of trunk	+5	+7	.....
• Whole-body vibration	+3	+5	.....
• Low job satisfaction	+3	.....	.....
		Total score (0–22)	.....

Total score	Age (years)		
	<35	35–45	>45
	Etiologic fraction		
0 (no exposure)	0	0	0
1	7	7	6
2	14	13	12
3	20	18	17
4	26	23	22
5	31	28	26
6	35	32	30
7	39	35	33
8	43	39	36
9	46	42	39
10	49	44	42
11	52	47	44
12	55	49	46
13	57	51	48
14	59	53	50
15	61	54	51
16	62	56	53
17	64	57	54
18	65	58	55
19	66	60	56
20	68	61	57
21	69	61	58
22	69	62	59

Fig. 2. Flow chart to assess the level of work-relatedness of low back pain. (Cutoff for "highly exposed" under "score if risk factor present": >15 kg for 10% of the worktime for manual materials handling, >10% of the worktime with back bent or twisted 30° for frequent bending and twisting of the trunk, and 5 years' exposure to 1 m/s2 or an equivalent vibration dose for whole-body vibration; horizontal lines under "Etiologic fraction" indicate the 50% level of work-relatedness of low back pain) – from Lotters et al. Scand J Work Environ Health, 2003, 29, 431–440 [41].

proportion of the overall variance in LDD in their twins. Interestingly, in a longitudinal study of these same twins followed up over 5 years, the same authors reported that greater maximal lifting at work, but not occupational load, predicted a greater reduction in lumbar disc height.[41]Occupation was not a significant predictor of back pain in a recent study by Williams et al. [42], but this study surveyed female twins, who had only limited exposure to heavy activity.

Iatridis et al. [43] postulated that although genetics may be the largest factor explaining variance associated with disc degeneration, genetics may have effects on anthropometry and direct mechanical consequences from loading, which could account for the unexplained variance of 25–50%. They note that intervertebral disc cells respond to a mechanical loading in a manner than depends on loading magnitude, frequency and duration, and put forward a comprehensive biomechanical model of the affected healthy and damaging loads and the capacity of the disc to remodel in response to load.

Leino-Arjas et al. [44] examined the relationship between occupational exposures and inpatient care for LDD in Finnish hospitals. In 1996, 3863 subjects were hospitalised in Finland due to LDD. Linking data on occupational title with the Finnish job exposure matrix and the occupational lifestyle matrix, they found in multivariate analyses of women that accident risk, job control and shift work were associated with increased hospitalisation together with age, education, income and body mass index. In men, accident risk and job control were associated with hospital admissions allowing for age, education and income. The authors concluded that accident risk and job control increased the risk of hospitalisation in both sexes. The authors acknowledged that the matrix they used ignored heterogeneity within occupations, but, at the same time, provided an independent perception of occupational status. They concluded that high physical workload, manual material handling, inconvenient work postures and accident risk were all associated with hospital admissions due to LDD in both men and women.

Using a more difficult clinical end point, Saftic et al. [45] investigated risk factors for lumbar disc herniation severe enough to require surgery in a cohort of 1001 Croatians. As the population studied was a rural and relatively isolated one, the authors noted the indication for surgery was likely to be stricter than in urban areas. Subjects, who underwent surgery, were matched with four controls by age, gender and residence. Nearly 67 subjects undergoing surgery were compared with 268 controls. The prevalence of subjects undergoing lower spine surgery in this random sample was 6.7%. Factors such as smoking, socioeconomic status, education and intensity of physical work at home did not contribute to the risk of lumbar intervertebral disc surgery. The highest OR was for a positive family history (OR 4), intensity of physical labour at work defined as hard work (OR 2.94) and high body mass index (OR 2.7). Their occupational analysis included those occupations involving sitting or standing and occupations involving hard physical activity, such as agricultural work, construction work, mechanics, fishing and soldiering. These data were interpreted as suggesting that the heritable risk of intervertebral disc surgery was independent of the risk conferred by heavy physical occupations.

In a Japanese study, Muraki et al. [46] examined the prevalence of knee osteoarthritis and lumbar spondylosis among agricultural forestry and fishery workers in 1471 participants aged greater than 50 years (531 men and 940 women) living in mountainous and seacoast communities. For occupational activities, sitting on a chair had a significant inverse association with LDD risk. Standing, walking, climbing and heavy lifting were associated with osteoarthritis of the knee but not with LDD. The authors concluded that clerical occupations with significant sitting were protective against both radiographic knee osteoarthritis and osteoarthritis in Japanese subjects; however, a relationship between heavy lifting and LDD could not be demonstrated.

In a small occupational study, Luoma et al. [21] examined 164 men aged 40–45 years, including 53 machine drivers, 51 construction carpenters and 60 office workers, who underwent MRI imaging and assessment for back pain. An increased risk of back pain was found in relation to signs of disc degeneration, and LBP was strongly associated with occupational factors. For example, the adjusted OR of LBP for machine drivers was 8.1 (add 95% CI). LBP was more common among machine drivers and carpenters than officer workers.

Roberts et al. [47] examined an algorithm for determining lumbar intervertebral disc shape and height, which they applied to MRI scans in 78 younger men (aged 20–30 years) and 71 older men (aged 31–58 years), all of whom were fit for work. After excluding degenerate lumbar discs at levels L1–L4, they found that intervertebral central disc height was significantly greater in older than in younger

men, supporting a model of the spine with ageing by which the disc adopts a more concave form, which they postulated was a result of changes in the underlying trabecular bone of the vertebral body due to repeated axial loading during normal adult life.

In a Danish study looking at specific occupations or job titles, Jensen et al. [48] conducted a 10-year follow-up study of 2175 long-haul truck drivers, 5060 other truck drivers and 6174 bus drivers. Compared with the general working population in Denmark, the standardised hospitalisation ratios (SHRs) for being hospitalised for intervertebral disc disorders were increased in long-haul drivers and bus drivers (SHRs 133 and 141, respectively) compared with other types of truck drivers (SHR 109). It was concluded that professional driving was a risk factor for intervertebral disc disease.

By contrast, Battie et al. [49] assessed 45 male MZ twin pairs (mean age 50.7 years) from the population-based Finnish Twin Cohort, who were selected to have greatly different patterns of occupational driving during their life (mean difference 27 700 h). Disc degeneration did not differ between highly exposed occupational drivers and their lesser-exposed twin brothers. This is a particularly elegant design of study, controlling as it does for age, sex and genetic factors, as well as unknown confounders but with 45 pairs it may have been underpowered.

Seidler et al. [50] examined 229 male patients with radiographically confirmed spondylosis of the lumbar spine and compared them with 197 control subjects. Of the 229 male patients, 135 had acute lumbar disc herniation. For symptomatic spondylosis with or without disc herniation, the OR for having worked in occupations with high physical workload was 3.2 (95% CI 1.2–8.3) for duration of less than 10 years and 6.2 (95% CI 3.3–11.8) for duration of 10 years or more. Cumulative forces applied to the lumbar spine over the working life were estimated using the Mainz–Dortmund dose model, which records compression force relative to respective duration of lifting process over occupational lifetimes. They observed that for a lumbar spine dose $>9 \times 10^6$ Newton hours, the risk of having radiographically confirmed spondylosis was associated with an OR of 8.5 (95% CI 4.1–17.5). The authors considered that the results indicated that cumulative exposure to lifting or carrying and extreme forward bending increases the risk of developing symptomatic spondylosis. The same authors examined 267 cases of acute lumbar disc herniation and 197 controls, and found a statistically significant positive association between extreme forward bending and lumbar disc herniation and accumulative exposure to weight lifting or carrying. There was also a statistically significant relation between cumulative exposure to weight lifting or carrying and lumbar disc herniation.

In a smaller, negative study, Savage et al. [20] examined 149 working men from five different occupational groups such as car production, ambulance officers, clerical staff, hospital porters and brewery workers, who underwent MRI. Some 34% of the subjects had never experienced LBP. MRI was repeated in 89 subjects after 12 months. Disc degeneration was most common at L5/S1 and, as expected, more prevalent with older age. In 30 subjects, who experienced LBP for the first time over 12 months, no change in MRI appearance was noted that could account for the onset of LBP. No difference in the MRI appearance of the lumbar spine was observed between the five occupational groups, but the sample size in each group was small.

In one of the few studies examining the cervical spine, Jensen et al. [51] examined the incidence of prolapsed cervical intervertebral discs in professional drivers in Denmark over a 10-year period. Almost all men in occupations involving professional driving had a statistically significantly elevated risk of being hospitalised for prolapsed cervical intervertebral discs (SHR 142). However, the risk in drivers doing heavy lifting was lower than in those doing little lifting, and the authors hypothesised that the increased risk may actually relate to vibrations and road shocks, twisting of the neck and acceleration and deceleration or whiplash accidents rather than specifically heavy lifting, given that a lot of heavy lifting off trucks is now mechanised.

Compensation and prevention at the workplace

Despite the ongoing controversy regarding the strength or relationship between occupational factors and disc degeneration, measures to mitigate certain workplace exposures are generally considered appropriate. For example, restrictions on maximal lifting of 25 kg are generally considered appropriate.

At most, 20% of working-age individuals experiencing LBP seek medical help and of those, only 20% report sickness absence and 10% for a workers' compensation claim [52]. Within the 45–65-year-old

age group, LBP is one of the most recently reported reasons for work loss [53]. As attention to return to work (RTW) is proven to be more important than solely focussing on treating injury or pain, physicians need to understand better the work context in relation to the capacity and beliefs of the patient. Factors, such as heavy physical demands, ability to modify work, social support, short job tenure, job satisfaction and fears of re-injury have been shown in systematic reviews to be predictors and determinants of disability after LBP at the work setting [52]. There are a number of workplace interventions with the potential to prevent work disability, including contact by the health-care provider with the workplace, ergonomic worksite visits, temporary modified work activities, including graded activity and RTW coordination. A recent European report on musculoskeletal disorders affecting occupation concluded that for back pain, a combination of appropriate clinical management, rehabilitation and workplace interventions is more effective than single therapy approaches alone [54].

Practice points

- LBP affects between 14% and 80% of working-aged people, depending on case definition.
- Disc protrusion and disc degeneration, as determined by MRI, are strongly associated with LBP.
- Work-related factors, including heavy lifting and frequent bending and twisting, are associated with lumbar disc disease.
- Population studies of cervical pain and cervical disc disease have lagged behind studies of the lumbar spine.

Research agenda

- Large prospective population studies using MRI and accurate baseline assessment of occupational exposures are needed, especially in men.
- There is a need for more studies of cervical disc disease and occupational factors.
- Standardised reporting systems of MRI changes are required.

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