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by [Riihimäki H](#)

Affiliation: Department of Epidemiology and Biostatistics, Institute of Occupational Health, Helsinki, Finland.

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Low-back pain, its origin and risk indicators

by Hilikka Riihimäki, MD, MSc¹

RIIHIMÄKI H. Low-back pain, its origin and risk indicators. *Scand J Work Environ Health* 1991; 17:81-90. Low-back pain is a common ailment. Much is known of the pain-sensitive structures of the low back, but for a patient with acute low-back pain the cause can rarely be defined. Although the biochemical properties of the intervertebral discs and their degenerative changes have been unraveled, it is difficult to distinguish between pathological degeneration and normal changes due to aging. As a consequence of the problems in diagnosing back diseases, the most common measure of low-back morbidity in epidemiologic studies has been the occurrence of symptoms. A wide spectrum of work- and individual-related factors have been found to be associated with low-back pain, but little is known about the extent to which these factors are etiologic and the extent to which they are symptom-precipitating or symptom-aggravating.

Key terms: degeneration, lumbar spine, psychosocial factors, review, work load.

Low-back pain has been called the nemesis of medicine and the albatross of industry (1). This saying reflects the prevailing lack of understanding of the nature and etiology, and, consequently, the treatment of back diseases, as well as the great impact this group of diseases has in the industrial setting.

Epidemiologic investigations have shown that 60-80 % of the population experiences back pain at some time during life (2-4)). Low-back pain can develop suddenly or gradually, with or without connection to an initiating event. In a Swedish study (5), two-thirds of the acute low-back patients reported that their back pain developed during recreational activities, often when the person bent forwards, and the onset of symptoms was usually sudden. For two-thirds of the patients with subacute symptoms, the initiation of pain had been gradual, and it usually started during the work-day, but without connection to any special event. The back pain of patients with chronic symptoms had developed insidiously in more than half of the cases.

The exact cause of acute low-back pain cannot usually be defined. In patients who recover within two to three weeks, it seems likely that either a muscle sprain, a minor ligamentous injury, or synovitis of a facet joint could have given the nociceptive impulses, whereas, in patients exhibiting pain for four to eight weeks, a greater ligament or tendon injury is likely to have occurred. The healing time for a disc injury is considerably longer (6).

Origin of low-back pain

All structures sensitive to pain are provided with particular nerve endings, nociceptors. Nociceptors can be activated by mechanical stress or exposure to irritating chemical substances in the surrounding tissue, such as lactic acid, potassium ions, bradykinins, prostaglandins, and histamine, that are released from traumatized, inflamed, or metabolically abnormal, especially ischemic, tissues (7).

In the lumbosacral region nociceptive nerve endings occur throughout the skin and subcutaneous and adipose tissue; the fasciae, aponeuroses, and ligaments; the vertebral periosteum and marrow; the adventitial sheaths of blood vessels; and the fibrous capsules of the lumbar facet and sacroiliac joints (7). In a normal adult intervertebral disc there are no nerve endings in the nucleus pulposus, and nociceptive endings have been detected only in the fibroelastic tissue that binds the posterior surface of the annulus fibrosus to the posterior ligament (7-9). Vernon-Roberts & Pirie (10) detected vascular in-growth, with accompanying nerve endings, into the discs through breaches in the cartilage end plates (Schmorl's nodes) or clefts in the annulus fibrosus. Thus low-back pain can originate from nearly all tissues in the lumbosacral area.

The exact location of the origin of pain in the back is difficult to establish owing to, among other things, complex, anastomotic innervation of the spinal tissues (7). Vernon-Roberts (11) proposed the following sites from which low-back pain could arise: (i) the interior of a disc following in-growth of nerves accompanying vascularization of clefts and prolapses, (ii) pain-sensitive structures (ligaments, dura matter, nerve roots), on which pressure is applied by spondylophytes, (iii) posterolateral disc prolapses or stabilizing osteophytes impinging on nerve roots in intervertebral foramina, (iv) apophyseal joints with osteoarthritis, (v) pseu-

¹ Department of Epidemiology and Biostatistics, Institute of Occupational Health, Helsinki, Finland.

Reprint requests to: Dr H Riihimäki, Department of Epidemiology and Biostatistics, Institute of Occupational Health, Töpeliuskatu 41 A, SF-00250 Helsinki, Finland.

doarthroses (osteophytes impinging upon the dorsal surfaces of the laminae of the neural arches) formed on the vertebrae following disc degeneration, (vi) fractures of facets or facet end plates of the apophyseal joints, (vii) an area of the spinal canal that has been narrowed by posterior disc prolapses and apophyseal osteophytes, particularly when there is already a narrow spinal canal, and (viii) trabecular microfractures in vertebral bodies in association with osteoporosis and Schmorl's nodes. He pointed out that it is possible that there is some pain-inducing pathology in clinically and radiologically "normal" spines.

Many studies have shown that degenerative changes detected in the lumbar spine in plain radiographs are associated with an increased risk for low-back pain (12—19), but there are also several studies in which such an association has not been detected (20—22). My co-workers and I (23) found that degeneration of the lumbar spine is associated with sciatic pain but not with lumbago or nonspecific back pain. Comparison of these studies is difficult because the classification of radiographic findings varies from one study to another.

Low-back pain may be associated with symptoms in a leg. A common cause for back pain radiating to a leg (ie, sciatic pain) is a herniated lumbar intervertebral disc. However, there is experimental evidence that referred leg pain may also arise from irritation of other deep tissues. Mechanical irritation of the posterior annulus fibrosus, the dorsal longitudinal ligament, and the nerve root have been shown to elicit sciatic pain (24), and chemical (hypertonic saline) irritation of the facet joint has caused similar pain (25).

The herniated lumbar intervertebral disc was first discovered by Mixter & Barr in 1934 (26). After this discovery it was hoped that back problems could be solved surgically, but today it is known that this is not the case. With modern imaging techniques, such as computerized tomography (CT), magnetic resonance imaging (MRI), and CT discography, the status of the intervertebral discs can be determined better than before. In clinical practice, when the patients' symptoms and clinical signs concur with the finding of a herniated disc, there are good grounds to presume that the disc disease causes the pain.

However, there is accumulating evidence that disc herniations are common in people with no back pain (27—28) and that in discography positive findings are frequent in symptomless people (29—31). Using MRI, Boden and his co-workers (28) found at least one herniated intervertebral disc in 24 % and spinal canal stenosis in 4 % of symptomless volunteers aged 20 to 80 years. In 20- to 59-year-olds the prevalence of herniated disc was 22 %, and in 60- to 80-year-olds it was 36 %. Spinal stenosis was detected only in the older age group, for which the prevalence was 21 %. At least one level of disc degeneration was noted in 36 % of the younger and in 98 % of the older group of asymptomatic volunteers.

MRI has made it possible, for the first time, to examine the disc status of symptomless people without any known adverse health effects; all other radiologic techniques expose subjects to radiation. However, before MRI is applied in larger population studies, the correspondence between MRI findings and the patho-anatomic status of the disc must be evaluated. MRI offers the possibility to determine why some people with disc herniations have symptoms and others do not. One possible predisposing factor seems to be the relative narrowness of the spinal canal (32—33). Changes in the chemical environment of the nerve and ischemia due to diminished blood supply have been suggested as intermediary steps beyond simple compression of the nerve root and the dorsal sensory ganglion in cases of a symptomatic herniated disc (34).

Difficulties in determining where the low-back pain originates, even with the most sophisticated clinical methods, means difficulties in defining the underlying disease. Thus it is not surprising that in epidemiologic studies the outcome has been measured by symptoms and signs (most frequently radiologic findings). In etiologic research, low-back pain may not be a good indicator of back disease. In addition to low-back pain being nonspecific, it is also possible that the occurrence and/or perception of symptoms is different between the "exposed" and "unexposed" (eg, heavy versus light physical work) subjects with the same underlying disease.

Only a few epidemiologic studies have been done in which the outcome has been more specific, namely, herniated disc (35—39). In these studies also the index series has consisted of people with symptomatic herniated discs, and thus the detected associations between herniated disc and the determinants may be biased. The generalization of the results to disc herniations per se should be done with caution.

Degeneration of the intervertebral disc

The intervertebral disc forms the articulation between the vertebral bodies. The disc is composed of two morphologically different parts, the annulus fibrosus and the semigelatinous nucleus pulposus. In children the discs are vascularized, but an adult's discs are avascular (40). Nutrition of a disc takes place by diffusion through cartilaginous end plates opposing the vertebral bodies and the annulus (41—42).

The nucleus pulposus contains 80—90 % water in early life. The water content decreases with increasing age and is 70 % in the sixth decade. The water content of the annulus is lower, 60 to 70 %, and it does not change markedly with age.

The major components of the disc matrix are collagen and proteoglycans. The nucleus pulposus contains 10—20 % and the annulus 50 to 60 % collagen per dry weight. This pattern changes little with aging, but the proteoglycan content per dry weight decreases

with increasing age. The proteoglycan content of the annulus is 20 % and that of the nucleus 65 % in early life, whereas in the sixth decade the figures are 10 and 30 %, respectively (43). Collagen is responsible for the tensile strength of the annulus, and proteoglycans for the hydrodynamic properties of the nucleus. There is progressive loss of resilience and turgescence of the disc as age advances.

The term disc degeneration is an arbitrary one. It does not denote any specific pathological change. An intervertebral disc presenting features commonly found in the sixth decade could be described as a "degenerated disc" when found in a man 30 years of age (44). One important goal for future research is to differentiate between pathological degeneration and normal age changes in the disc (45).

According to Macnab (44) the three main histological changes in aging discs are the destruction of the hyaline cartilage end plate, the increasing conversion of the gelatinous ground substance of the nucleus into formed elements, and the metaplasia of the annulus into fibrocartilage. Defects in the end plates interfere with the nutrition of the disc. Such defects are frequently followed by the growth of vessels in the disc derived from the vertebral spongiosa, and occasionally granulation tissue can be seen.

In premature degeneration the annulus may show radial tears which are the most common posterolaterally where the annulus is thinner than elsewhere. Sometimes these tears are associated with an extrusion of nuclear material. In addition there may be circumferential tears between the layers of the fibrous annulus. The fact that these clefts may contain fibrin and sometimes exhibit vascular ingrowth suggests that they are formed as the result of traumatic tearing rather than purely the result of a degenerative process (11).

End-plate fractures are compression failures which promote disc degeneration (46—48). Laboratory experiments have shown that in vitro torsional loading of spinal segments produces fissures in the annulus posterolaterally. Acute ruptures of the annulus can only be produced by combined flexion and lateral bending motions, but not by torsion (49). A previously damaged disc is more vulnerable than a healthy one.

Disc degeneration is associated with undue mobility in the motion segment, and this mobility promotes the growth of spondylophytes, especially of the traction spur type (44). However, spondylophytes are not always associated with disc degeneration (50). When the disc degenerates, its height is reduced. This phenomenon leads to a change in stress distribution in the motion segment and to secondary degeneration in the facet joints (51).

Disc degeneration is fairly advanced before it can be visualized in a radiograph as narrowing of the intervertebral space. Though plain radiographs have been in extensive use for decades, data on the correspondence between the pathoanatomic status of the

disc and radiographic signs of disc degeneration are scant (52).

Classification and natural course of back diseases

The difficulty of making precise diagnoses is the single greatest problem of clinical work with patients with back symptoms, and in the research on back diseases as well.

Grabias & Mankin (53) presented a classification of low-back pain which was based on etiologic factors (ie, mechanical, infectious, inflammatory, metabolic neoplastic, and visceral). According to this classification, the majority of people with low-back pain have mechanical causes, which include lumbosacral sprain/strain, degenerative disc disease, spondylolisthesis, spinal stenosis, and fracture. This class is nonspecific, whereas the other five classes consist of specific diseases with exact diagnoses.

Nachemson & Andersson (54) proposed a classification based on symptoms and clinical findings. They defined *insufficiencia dorsi*, lumbago, sciatica, rhizopathy as a special form of sciatica, and lumbago-sciatica. The following prefixes were also defined according to the onset and duration of symptoms: acute (0—3 months in duration, immediate onset), subacute (0—3 months in duration, slow onset), chronic (>3 months in duration), and recurring (symptoms recurred after an interval of no symptoms). A review of the literature indicated that this classification has not attained acceptance outside of Scandinavia.

In cases where the exact cause of low-back pain cannot be determined, it has been recommended to use such terms as "nonspecific" (55) or "idiopathic" back pain (45). Estimates of the proportion of all low-back pain for which no particular etiology can be specified range from 20 % (56) to 85 % (57). In part the variation depends on whether cases in which degenerative changes are seen radiographically in the intervertebral spaces are included in the category of idiopathic low-back pain or not. Back pain initially classified as idiopathic may be shown subsequently to have a definite pathologic etiology in patients followed for sufficiently long periods of time (45).

In a Swedish study (5), careful clinical examination did not reveal objective signs for about one-third of the back patients with acute or subacute symptoms (sick leave for one month) and two-thirds of those with chronic symptoms (sick leave for three months). In the groups without objective findings among the patients with acute or subacute symptoms, the rate of recovery was faster than in the groups with objective clinical signs.

According to a review by Nachemson (6), attacks of acute low-back pain tend to start at the age of 25 years. Symptoms subside in half of the patients within four weeks and in 90 % within two months,

and little can be done in the way of treatment to alter this course. The risk of recurrence over the following couple of years is about 60 %, but decreases during the third year. After six months 2–3 % and after 12 months 1 % of the low-back patients with acute symptoms are continually symptomatic. The mean age of the patients with chronic symptoms is about 50 years. In a recent Swedish study of people who were on sick leave due to back disease, 57 % returned to work within a week, almost 90 % within six weeks, and 95 % within 12 weeks (58).

Risk indicators of low-back disorders

Hildebrandt (59) made a review of epidemiologic studies on risk factors of low-back pain using five comprehensive publications on low-back pain from the 1980s, three books (60–62), and two review articles (63, 64). He found 24 *work-related factors* which were regarded as risk indicators of low-back pain by at least one of the following sources: general: heavy physical work, work postures in general; static work load: static work postures in general, prolonged sitting, standing or stooping, reaching, no variation in work posture; dynamic work load: heavy manual handling, lifting (heavy or frequent, unexpected heavy, infrequent, torque), carrying, forward flexion of trunk, rotation of trunk, pushing/pulling; work environment: vibration, jolt, slipping/falling; and work content: monotony, repetitive work, work dissatisfaction. The list of “generally accepted” risk factors (ie, factors mentioned by at least three sources) was reduced to the following eight: general: heavy physical work; static work load: prolonged sitting; dynamic work load: heavy manual handling, heavy or frequent lifting, trunk rotating, pushing/pulling; work environment: vibration.

There were 55 *individual factors* mentioned by at least one of the sources as a risk indicator of low-back pain. They were as follows: constitutional: age, gender, weight, back muscle strength (absolute and relative), fitness, back mobility, genetic factors; postural-structural: severe scoliosis, difference in leg length; radiographic: severe multilevel degeneration, disc resorption, disc herniation, severe arthrosis of facet joints, spondylarthropathies, spondylolysis, spondylolisthesis, sacralization/transitional vertebra, skeletal defects, fractures, neoplasms, severe kyphosis, lumbar kyphosis; medical: back complaints in the past, infectious diseases, pregnancies, number of pregnancies and childbirths; psychosocial: depression, anxiety, “life events,” family problems, divorce, personality, hypochondriasis, somatization, dissatisfaction with work or social status of work, tense and fatigued after work, high degree of responsibility and mental concentration, poor intellectual capacity, poor ability to establish emotional contacts, less “philosophic” attitude; demographic: socioeconomic situation, edu-

cational level, location of home; other: sports, degree of physical activity, gardening, caring of grandchildren, smoking, alcohol, coughing, work experience. Only the following six risk factors were “generally accepted”: constitutional: age, relative muscle strength, physical fitness; medical: back complaints in the past; psychosocial factors (not specified); other: work experience.

The following 18 factors were mentioned as *nonrisk factors*: constitutional: body build, height/weight indices; postural-structural: kyphosis, lordosis; radiographic: nonspecific radiographic abnormalities, Schmorl’s nodes, osteophytes, disc narrowing, facet asymmetry, spina bifida occulta, osteoporosis, lordosis, scoliosis and increased lumbosacral angle; medical: severe mental problems like psychosis and neurosis; demographic: ethnic factors, marital status. Of the nonrisk factors, nonspecific radiographic abnormalities were considered of no importance by almost all the sources.

Most (74 %) of the risk and nonrisk factors were only mentioned in one or two sources, and for 22 % no reference and for 48 % only one reference was given. Hildebrandt concluded that the interpretation of the vast amount of epidemiologic data on the correlates of low-back pain is difficult.

The review depicts clearly the confusion that prevails today in the field of epidemiology of low-back pain. Most epidemiologic studies have been made on population cross-sections with retrospective experience of exposure and outcome. The most commonly used measure of outcome has been the occurrence of low-back pain based on questionnaire or interview data.

It is not surprising that low-back pain has been found to be related to such a variety of factors. The prevalence of nonspecific low-back pain is high in all populations, and thus there is a good chance of obtaining a relationship between any infrequent health-related characteristic of a population and the prevalence of low-back pain, especially since health problems tend to cluster in populations (65).

Problems may arise in etiologic considerations when symptoms are used as the indicator of a disease. Some factors which aggravate symptoms or modify the perception of symptoms may appear as risk indicators of symptoms even though they are not risk indicators of the underlying disease.

Work-related factors

Heavy physical work. Many cross-sectional studies have indicated that low-back pain is related to heavy physical work (4, 13, 14, 66–73), but there are also studies in which such a relationship has not been detected (74, 75). In most of these studies work was classified as heavy on the basis of general impression without any attempt to analyze the back-loading factors in more detail. In some studies (4) the data on the heaviness of work was also based on subjective per-

ception, which may be influenced by the occurrence of back trouble.

An 11-year follow-up of a Finnish population sample revealed that blue-collar workers had a higher incidence of hospitalization due to herniated lumbar disc or sciatica than white-collar workers (77). A sample of Finnish employees in the metal industry has been followed for 10 years. Both in the cross-section and follow-up, low-back pain was more common among the blue-collar workers than among the white-collar workers. For the blue-collar workers, however, the associations between the indices of physical work load and musculoskeletal morbidity were weak or non-existent (78), possibly because of small variation within the sample. In a one-year follow-up of a Danish sample of the general population, occupational factors did not predict the occurrence of low-back pain (79).

The results of many studies indicate that radiographically detectable degenerative changes of the lumbar spine are related to heavy work (12–14, 19, 66, 75, 80, 81).

Lifting, carrying, pulling and pushing. Loading of the spinal tissues is not harmful unless the endurance of the tissues is exceeded. This excess may be caused by sudden overload or fatigue in repeated loading (82). Gradual increase in loading has a training effect, and muscles, tendons, and also vertebrae adapt to endure heavier loads. As an example, weight lifters have been shown to have less degenerative changes in the lumbar spine than men in heavy physical work (13). A high level of physical activity strengthens both the vertebrae and the discs (83).

Lifting and other sudden motions are often claimed to be the immediate precipitating event in low-back pain. At the Boeing company, improper lifting was the most common self-reported cause of back injury (84), and also among Scottish miners and office workers recent low-back pain was most commonly attributed to lifting (73).

With regard to the long-term effects, several studies indicate that the occurrence of low-back pain is related to lifting, carrying, pulling, pushing, or other sudden maximal efforts (4, 66, 85–91). Chaffin & Park (87) reported that a mismatch between the lifting strength requirements of a job and workers' lifting capacity increase the risk for low-back injuries. They also found that the risk is greater when the frequency of lifts is high or low in comparison with the intermediary frequency.

In a case-referent study (36) herniated lumbar intervertebral disc was not related to lifting. However, in a later similar study an increased risk of herniated disc was found in jobs involving heavy lifting combined with twisting and bending (37).

Twisting, bending and other nonneutral trunk postures. Often nonneutral trunk postures are associated with lifting, and thus it may be difficult to evalu-

ate the isolated effect of such postural load on the back. A relationship has been shown between non-neutral postures and back pain in several studies (66, 73, 88, 92). Keyserling and his co-workers (93) observed the occurrence of nonneutral trunk postures for each of the study subjects in a case-referent study in an automobile assembly plant and found low-back pain to be related to nonneutral trunk postures. My co-workers and I (94) found a relationship between sciatic pain and working in twisted or bent postures (self-report) also among office workers whose work rarely involved the lifting of heavy loads.

Motor vehicle driving. There is mounting evidence that motor vehicle driving is positively associated with risk for low-back pain (68, 88–90, 95, 96), for sciatic pain (91, 94), and for herniated intervertebral disc (38, 77, 97–99). An increase in the prevalence of radiographically detectable degenerative changes of the lumbar spine has also been found among motor vehicle drivers (100, 101).

It is suggested that whole-body vibration combined with prolonged sitting has a deleterious effect on the back. Occupational exposure to whole-body vibration is frequently associated with other back loading factors at work, such as manual materials handling, and therefore the interpretation of the results from epidemiologic studies is ambiguous (102). In addition usually no differentiation between shocks and vibration exposure in terms of frequency, or a distinction between the directional components of vibration, has been made (103). However, there is accumulating experimental evidence supporting the view that whole-body vibration in a seated posture is harmful to the back (82).

Sitting. Some studies have indicated that low-back disorders are related to prolonged sitting or sedentary work (36, 66, 85), but there are also several studies in which no such relationship has been detected (4, 37, 77, 94, 104–106). Thus, evidence for a relationship is scarce, even though it can be considered biologically plausible. It has been shown that the intradiscal pressure is increased in the sitting posture, especially when one sits erect without support (107, 108). It is also possible that prolonged static sitting postures may have a deleterious effect on the nutrition of the intervertebral disc (109).

Other work-related factors. Various psychological and psychosocial factors related to work, such as monotonous work and job dissatisfaction (4, 90, 105, 110) have been reported to be associated with the occurrence of low-back pain. Most of the studies have been retrospective, and it is thus difficult to determine whether psychological features are antecedents or consequences of back pain (111). It is also difficult to de-

termine whether these factors play a role in the etiology of low-back diseases or only affect the perception of symptoms and sickness behavior. In a prospective study among about 3000 aircraft employees psychosocial factors at the workplace were better predictors of future back injuries than physical findings (112).

Trauma

It is plausible that trauma is of importance in the development of back disorders. In true accidental injuries this connection is more obvious than in nonaccidental injuries in which there is no disruption of the normal pattern of work and the first unexpected event is pain in the back (63). In a prospective study by Lloyd & Troup (113), in about one-third of the attacks of low-back pain there was a truly accidental event, in one-fifth a nonaccidental injury, and for the rest there was no indication of any kind of injury. Truly accidental injuries, especially falls, had a worse prognosis than the others. In an analysis of back injuries Manning et al (114) found that two-thirds of true accidental injuries were "underfoot accidents" (slipping of foot, etc), and "handling" was the cause in nearly one-third. In nonaccidental injuries handling was recorded for 63 % of the cases.

The risk of sciatic symptoms has been reported to be increased among workers who have earlier had back accidents (76, 115). Repeated compressive stress can cause microfractures in the end plates and subchondral bone of the vertebrae (10, 47, 116), but it has been suggested that torsional load contributes more to the development of disc degeneration than compressive loads (117).

Repeated minor injuries are presumed to cause irreversible damage and hasten the degenerative process in the spine (118). Degenerated vertebral units are more liable to fail under mechanical stress than healthy ones (119).

Individual characteristics

According to earlier retrospective or cross-sectional studies, the occurrence of low-back pain increases with increasing age up to about 50–60 years of age, after which there is a decline (13, 14, 71, 120, 121). Some prospective studies have, however, revealed that there is no distinct age dependency (3, 122). Men and women tend to be affected with equal frequency (71, 120, 123).

The results from studies on the relationship between anthropometric measures and back disorders are conflicting. In some studies (69, 97, 115, 124) taller people have been found to be at greater risk for low-back pain, but not in others (38, 73, 84, 125). Likewise, a positive relation between excess body weight and risk for low-back disorders was shown in some studies (97, 122, 124, 126) but not in others (38, 69, 125, 127).

General physical fitness, measured as aerobic capacity, has not been shown to predict low-back pain (69, 122). Cady and his co-workers (128) found that fire-

fighters who were unfit had the most injuries, but that the fittest of the firefighters suffered the most severe injuries.

Many cross-sectional studies have shown that people with a history of low-back disorders have weaker trunk muscles than those without such a history (129–131). Imbalance between trunk flexor and extensor muscle strength has been detected in individuals with low-back disorders (132–134). In two prospective studies, the weakness of trunk muscles was not associated with the incidence of low-back pain among previously healthy people (132, 135). In the prospective Boeing study a positive relationship was detected between isometric strength and the occurrence of back injuries (136). This relationship did not however, remain after adjustment for age and gender.

Smoking has been found in many studies to be related to the occurrence of back disorders (38, 69, 79, 89, 91, 122, 126, 137, 138), but there are also studies which have failed to show such a relationship (115). There is experimental evidence that smoking impairs the nutrition of the intervertebral discs however (139).

Individuals with low-back disorders have been shown to have a higher frequency of psychological symptoms than those without disorders (88, 140–143). Most of the reports on the relationship between low-back disorders and psychological factors are from cross-sectional studies. Thus it is possible that the detected associations reflect the secondary effect of low-back disorders on psychological function. Psychological symptoms occur commonly among individuals with chronic diseases, and in this respect back disorders are no exception (65). However, a few prospective studies have shown that psychological symptoms may also have a role in predicting the future incidence of low-back disorders (79, 112).

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