REVIEW

Low back pain investigations and prognosis: a review

K M Refshauge, C G Maher

Br J Sports Med 2006;40:494-498. doi: 10.1136/bjsm.2004.016659

Low back pain is reviewed in terms of when investigations are useful and its clinical course. Despite the extensive evaluation of the accuracy of investigations such as radiography, magnetic resonance imaging, and myelography, there is a surprising dearth of research to inform their use in primary care. There is no clear evidence on which to base judgments for selection of appropriate tests to confirm or exclude low back pain pathology. It appears that investigations are rarely necessary for low back pain. Specific investigations should be ordered to identify a particular pathology but should not be ordered routinely for general screening. In the absence of pathology, low back pain and its associated disability improve rapidly in the first weeks after onset, but, in contradiction to all guidelines, both commonly persist and the best evidence suggests that recurrences are common.

> ow back pain (LBP) is one of the most common symptoms in the developed world,¹ yet it is one of the most elusive. It can be extremely disabling, and the social and economic burden is enormous.¹ Costs arise from treatment, investigations, compensation for pain and suffering, and lost work time. Ironically, it is likely that part of the burden of LBP arises from unnecessary and ineffective investigations and from incomplete understanding of its clinical course.

CAUSES OF LBP

The cause of the vast majority of LBP is unknown; current tests cannot identify a pathological cause for the pain in at least 85% of cases.² That is, in 85% of cases, even when the most extensive testing is conducted, no apparent cause can be established. For this reason, such LBP is now usually termed non-specific low back pain. Our inability to reliably identify pathology has given rise to numerous hypotheses concerning the cause of LBP, including reduced trunk extensor endurance,3 psychological distress,4 hamstring inflexibility,5 poor muscle control of the trunk,⁶ poor posture,⁷ and low body mass.⁷ However, at best, there is only preliminary evidence to support an association between such impairments and the presence of LBP, and most of the causal hypotheses lack convincing evidence. Given our poor ability to identify the cause of most cases of LBP, invasive and expensive investigations should only be used when findings from the clinical examination indicate the likely existence of serious pathology. It should be noted, however, that the clinical

signs and symptoms of LBP can be treated successfully without a pathological diagnosis.

To identify serious pathology, all guidelines for management of LBP recommend use of a diagnostic triage. Based on the findings from the history and physical examination, LBP is classified into one of three diagnostic categories: non-specific low back pain or simple backache, nerve root/spinal nerve compromise, or potential cases of serious spinal pathology (including infection, cancer, fracture, inflammatory disorders, and cauda equina syndrome).

Because non-specific LBP currently cannot be further classified, it is often referred to according to its duration: acute LBP (duration less than six weeks); sub-acute LBP (duration more than six weeks and less than three months); and chronic LBP (duration more than three months). Although probably simplistic, this classification makes some sense because evidence to date suggests that different treatments are effective in different phases of LBP,⁸ and different factors, such as psychosocial factors, appear to take on greater importance with increasing chronicity.⁹ Non-specific LBP does not benefit from extensive investigations, and in some cases, further examinations have been shown to be harmful.¹⁰

WHEN ARE FURTHER INVESTIGATIONS OF LBP PRUDENT?

Investigations additional to the clinical examination are required when a patient's signs or symptoms (red flags) raise the index of suspicion that there is serious pathology. Relevant investigations include blood tests, nerve conduction tests, imaging such as radiography, magnetic resonance imaging (MRI), computed tomography, and dual energy x ray absorptiometry, and rarely myelography. However, invasive tests should be ordered judiciously, such as when identification of pathology necessitates specific treatment or, more importantly, if the pathology renders a treatment harmful. It is also likely that early detection of certain serious pathologies is vital because delayed treatment leads to poorer outcome.^{11 12} The goal is to identify all, or most, cases of serious pathology with a minimum of unnecessary diagnostic testing.

These invasive investigations should rarely be required for patients with LBP because serious pathology presenting as LBP is rare. The few studies that have investigated the incidence of serious pathology presenting as LBP found that <5% of cases can be classified as serious pathology. For example, in the United States, Deyo and colleagues¹³ found, in a retrospective

Abbreviations: LBP, low pack pain; MRI, magnetic resonance imaging

See end of article for authors' affiliations

Correspondence to: Professor Refshauge, School of Physiotherapy, University of Sydney, PO Box 170, Sydney, NSW 1825, Australia; k.refshauge@fhs.usyd.edu. au

Accepted 7 January 2006

study of acute LBP presenting to primary or secondary healthcare practitioners, that 4% had compression fracture and 1% had cancer, infection, inflammatory disorders, or cauda equina syndrome. An even smaller proportion of serious pathology (1.4% of cases) was found in Australia¹⁴ from a survival cohort of patients with LBP of any duration presenting to primary or tertiary care.

All clinical practice guidelines advise detection of serious spinal pathology from features in the clinical assessment such as age over 50 or under 20 years, fever, widespread neurological symptoms, or unexplained weight loss—for example, National Health and Medical Research Council guidelines.¹⁵ However, the accuracy of these clinical features for diagnosis of serious pathology, particularly in primary care, has rarely been explored. The validity of the information from the few available studies is likely to be poor, because most studies have serious design problems such as patient selection bias and verification bias.

In the few cases of LBP for which further diagnostic work up is required, investigations should be ordered specific to the suspected pathology. Investigations should not be ordered routinely or to screen broadly for a range of pathologies because asymptomatic anomalies exist in the normal population. The practice of routine investigation is in danger of leading to management of benign abnormalities seen on images, such as disc bulge, despite the evidence that these anomalies are rarely related to LBP.²

Taken together, this information suggests that thoughtful referral for further investigation relies on accurate diagnosis from the clinical examination, but accuracy of individual components of the clinical examination is not known. As there is so little evidence, the conservative recommendation should be that clinicians use the clinical practice guidelines for management of acute LBP until better evidence becomes available.

SPECIFIC PATHOLOGIES REQUIRING FURTHER INVESTIGATION Spinal fracture

It is interesting to note that most guidelines reference diagnostic accuracy of the clinical examination for fracture to unpublished data originating from observations reported in a single review.¹³ The methods used in the review were not detailed, precluding assessment of validity of the findings.

We are currently conducting a systematic review of diagnosis of fracture. Three studies^{16–18} investigated diagnosis of fracture in a mix of primary and secondary care settings, and nine studies^{19–27} investigated detection of fracture after blunt trauma in emergency departments. The diagnostic accuracy of features conventionally thought to be associated with fracture, such as age >55 years, use of corticosteroids, and trauma, was low and/or inconsistent across the studies.

Given the lack of evidence on which to base referral. adherence to the guidelines is necessary, in addition to clinical judgment. For young people, fractures are rare unless major trauma is involved. Stress fracture should be suspected among gymnasts²⁸ and fast bowlers in cricket.²⁹ For older people (≥75 years), particularly older women, a compression fracture should be suspected if pain results from minor trauma and osteoporosis is either confirmed or is suspected from hormonal status or steroid use. These clinical judgments should be regularly reviewed as more information becomes available. It is now a public health imperative to identify osteoporosis. When one fracture has occurred, the risk of subsequent fracture is greatly increased,³⁰ and so the person should be placed on an osteoporosis management programme to slow bone loss, prevent falls, and decrease risk of further fractures.

Cancer

The red flags presented in most clinical guidelines for the diagnosis of cancer in patients presenting with LBP in primary care are based on a single study.³¹ In this study, however, Deyo and Diel³¹ identified cases of cancer retrospectively from the hospital's tumour register and not from following patients prospectively. A consequence of this is that those patients who developed cancer but presented elsewhere would not be identified as cancer cases, thereby potentially biasing the findings. Nevertheless, Deyo and Diel³¹ found that four clinical findings strongly predicted cancer in patients with LBP: previous history of cancer, age \geq 50 years, failure to improve with conservative treatment, and unexplained weight loss (>4.5 kg (10 lb) in six months).³¹

Although the diagnostic evidence available for primary care practitioners is scant, the need to identify cancer early is urgent to ensure early and effective intervention and prevent complications. Joines and colleagues $^{\scriptscriptstyle 11}$ evaluated a decision analysis paradigm using data from the original study of Deyo and Diel,³¹ and determined sensitivity and specificity of a range of diagnostic strategies to identify cancer. They also conducted a rudimentary cost-effectiveness analysis on these same data. They recommend that, for patients with any positive finding among the four predictors originally identified, the most appropriate diagnostic work up is to order erythrocyte sedimentation rate (ESR) tests or radiographs. For those with ESR \geq 50 mm/h or positive radiograph, an MRI should be ordered. For any patient with a previous history of cancer, particularly breast or prostate cancer, an MRI should be ordered. If the MRI is positive, the patient should be referred for a vertebral biopsy. However, the study was conducted in a hypothetical setting. Because prevalence of cancer in the population presenting for care can dramatically alter the findings, clinicians should be alert to new knowledge in settings relevant to their patients.

Infection

The red flags for clinical diagnosis of infection are also based on a single study that focused on spinal osteomyelitis.³² The authors retrospectively surveyed patients with spinal infection to determine an association between reported intravenous drug use, urinary tract infection, or skin infection. In the absence of clear evidence, best practice would currently recommend that, when infection is suspected because of fever and sudden onset of severe pain, particularly in the case of intravenous drug use, it should be confirmed by blood tests or aspiration of the relevant tissue.

Inflammatory disorders

Although there are studies of patients with inflammatory disorders presenting with LBP in tertiary care^{33–35} and nonconsulting participants identified in an epidemiological survey,³⁶ there are no studies of diagnostic accuracy of clinical features for inflammatory disorders presenting as LBP in primary care. The design problems in the available studies, such as using pain-free participants as controls,^{33 35} potentially inflate estimates of diagnostic accuracy.³⁷

From the body of literature on inflammatory arthritides,³³⁻³⁶ the cardinal signs include morning stiffness lasting more than 30 minutes, symmetrical peripheral joint involvement, associated signs and symptoms of systemic involvement, and non-mechanical pain—for example, pain and stiffness that decreases with movement and increases with rest. These signs and symptoms have not been evaluated for patients presenting primarily with LBP in primary care. However, it is prudent to use them as raising the index of suspicion and request confirmatory blood tests if they are present, and be alert when patients do not improve rapidly even when these symptoms are absent.

Cauda equina syndrome

There are no studies of diagnostic accuracy for cauda equina syndrome. Clinical observations suggest that cauda equina syndrome should be suspected when leg pain that includes several spinal nerve levels is accompanied by widespread motor and/or sensory weakness and importantly when there is associated urinary and/or faecal incontinence or retention. When a patient presents with this cluster of symptoms, further tests such as radiography, myelography, and, rarely, lumbar puncture should be requested immediately. The earlier the diagnosis is made, the better the outcome.³⁸

Other abnormalities

Many abnormalities can be visualised, particularly as modern imaging techniques have become more powerful. However, most of these abnormalities do not constitute pain provoking or serious pathology. For example, spondylitis and general spinal degeneration are often found on scans of older people, but are generally regarded as benign and are rarely associated with symptoms. Images are therefore not useful in nonspecific LBP, particularly because it is tempting to attribute symptoms to abnormal findings, and this practice can be harmful, prolonging episodes of LBP.¹⁰

As the accuracy of clinical diagnosis of serious pathology is largely unknown, we have little evidence to support clinical judgments made from the history and physical examination, the basis of referral for further assessment. Until such evidence becomes available, the signs and symptoms recommended for clinical diagnosis in all guidelines should be used to alert clinicians to the possible presence of serious pathology. Clinicians should also be alert to atypical progress in their patients with LBP, however, because serious pathology may exist in the absence of signs and symptoms.

PROGNOSIS OF LBP

Non-specific LBP is generally considered as a single entity despite the possibility that there are distinct and separate subgroups within this population that may recover at different rates and with different outcomes and may respond differently to the variety of available treatments. There has been some attempt to identify factors that predict rate of recovery. However, most of these studies have serious methodological problems, such as not using inception cohorts or not providing data to support the conclusions.³⁹⁻⁴² Despite these problems, most guidelines for acute LBP recommend that indicators of poor prognosis be identified and report that factors such as fear avoidance behaviours, presence of leg pain, or low job satisfaction are likely to prolong the episode of LBP.

Prognosis for chronic LBP has rarely been studied and is therefore largely unknown. The few studies that addressed this issue included participants with subacute LBP, had high drop out rates, and those lost to follow up differed significantly from the cohort remaining in the study. Consequently, once a person has persisting LBP, the course of the problem cannot be accurately predicted.

The prognosis for acute LBP has been investigated and has been confidently reported as excellent in all current clinical practice guidelines for the management of acute LBP.⁴³ All guidelines consistently report that acute LBP typically has an excellent prognosis because most cases (up to 90%) recover within six weeks.⁴⁴

Our group found reason to question the guidelines and to hypothesise that the prognosis of acute non-specific LBP might not be so optimistic. In Australia, the prevalence of chronic pain in general was found to be much higher in the population than previously thought,^{45 46} particularly as the majority (>55%) do not seek care for their LBP^{47 48} and recovery from acute LBP was not necessarily complete.⁴⁹ In

addition, there was conflicting evidence about prognosis, varying between recovery in >90% of patients within two weeks⁵⁰ and recovery in only 41% of patients by one month.⁵¹ The longer term prognosis also varied appreciably, from recovery in 98% of patients by three months⁵⁰ to recovery in only 54% of patients by 12 months.⁵¹ These two studies provide very different predictions of risk of developing chronic LBP. These clear contradictions led us to believe that the current view of acute LBP as benign and self-limiting should be reconsidered.

We therefore conducted a rigorous systematic review of studies of the prognosis of acute LBP⁵² and of the factors that increase the risk of poor prognosis. We included prospective studies that used a clearly described inception cohort of participants with acute LBP who were followed for at least three months. Reporting of patient relevant outcomes such as disability, quality of life, and return to work was required, with follow up of at least 80% at each follow up measurement occasion.

Fifteen relevant studies (reported in 20 papers)⁵⁰ ⁵¹ ^{53–70} were located, one of which specifically investigated sciatica.⁷⁰ A consistent finding across these studies was that pain decreased rapidly by 12–84% of initial pain levels (pooled mean 58%) within the first month, and thereafter decreased more slowly for the next two months. Only two studies⁵³ ⁵⁶ investigated longer term outcome, both finding that pain levels remained unchanged at the 12 month follow up. The pooled mean level of pain was 22/100 at one month and 15/100 at 3–12 months.

The findings for disability outcomes were similar to the pain outcomes. Disability decreased by 33–83% of initial levels (pooled mean 58%) within one month. Only one study⁵³ reported longer term (six months) follow up data. The pooled mean level of disability was 24/100 at one month and 14/100 at three to six months.

A particularly interesting finding was that most participants (68–86%) who were off work because of LBP at the start of the study returned to work within one month (pooled estimate 82%; 95% confidence interval (CI) 73% to 91%). By six months, nearly all participants had returned to work (pooled estimate 93%; 95% CI 91% to 96%). Taken together, these findings from different studies suggest that people return to work despite continuing pain and disability. This effect is seen in the trial of Hagen *et al*^{71 72} of advice as a treatment for workers sick listed because of LBP: at follow up 61% of the intervention group had returned to work yet 95% where still in pain.

Recurrence was found to be common. More than a quarter of participants had at least one recurrence within three months, with a cumulative risk of 26% (95% CI 19% to 34%). At 12 months the pooled cumulative risk of at least one recurrence within 12 months varied from 66% to 84% (pooled estimate 73%; 95% CI 59% to 88%), and after three years it was 84%.

In the single study that included patients with sciatica,⁷⁰ both back and leg pain decreased by a mean of 69% of initial scores within one month. Disability decreased by 57% of initial scores within one month. Long term pain and disability data were not available.

Prognostic factors

There is a wealth of information on clinical features that predict poor prognosis from an episode of non-specific LBP. However, few reports are from rigorous prospective studies. Factors commonly thought to predict outcome of an episode of LBP include psychological factors⁹ (such as psychological distress, depression), clinical factors⁵⁹ (such as previous back pain), and work related factors⁵⁹ (such as job satisfaction). In our recent review, ⁵² we located three studies that reported on

What is already known on this topic

- Although low back pain (LBP) is highly prevalent and has been extensively researched, the pathological diagnosis remains a puzzle
- All 11 clinical guidelines consistently recommend that investigations should rarely be used except when serious pathology is suspected. Finally, non-specific LBP was thought to be a benign, self limiting condition

What this study adds

- Conventional investigations are unrewarding for at least 85% of patients with LBP
- There is little evidence to support the signs and symptoms (red flags) used to identify serious pathology, and prognosis has been shown to be poor

prognostic factors for at least 80% of the population studied.^{50 51 59} The only predictor of outcome was score on the Vermont disability prediction questionnaire: a lower score predicted earlier return to work.⁵⁹ However, as most people return to work by three months (pooled estimate of 6% not returned to work), the ability to predict this outcome probably has limited clinical utility.

CONCLUSIONS

LBP has proved to be an enigma for health professionals for decades. It is not possible to identify the cause in most cases, although serious pathology can present as LBP and health practitioners are required to recognise such presentations. However, a review of the diagnostic accuracy of the red flags shows that none have sufficient evidence of accuracy when used in primary care for patients presenting with LBP. This is a major problem, given the imperative to identify serious pathology so that appropriate management can be instituted as early as possible. The most prudent recommendation would be that clinicians treating patients with LBP reevaluate any patients who do not respond rapidly to treatment. It is clear that LBP should markedly improve in the first weeks after onset. If the clinical course appears aberrant, further investigation may be required.

At the end of the last century, it seemed clear that LBP was a benign self limiting condition. Recent work shows that this is not true. Non-specific LBP is often not self limiting; a large proportion of patients experience persistent low intensity pain and disability, but are able to return to work.

Authors' affiliations

K M Refshauge, C G Maher, School of Physiotherapy, University of Sydney, Sydney, NSW, Australia

Competing interests: none declared

REFERENCES

- Atlas S, Deyo R. Evaluating and managing acute low back pain in the primary care setting. J Gen Intern Med 2001;16:120–31.
- 2 Deyo RA, Weinstein JN. Low back pain. N Engl J Med 2001;344:363-70.
- 3 Luoto S, Heliovaara M, Hurri H, *et al.* Static back endurance and the risk of low-back pain. *Clin Biomech* 1995;10:323–4.
- 4 Croft PR, Papageorgiou AC, Ferry S, et al. Psychologic distress and low back pain. Evidence from a prospective study in the general population. Spine 1995;20:2731–7.

- 5 Hultman G, Saraste H, Ohlssen H. Anthropometry, spinal canal width, and flexibility of the spine and hamstring muscles in 45–55 year old men with and without low back pain. J Spinal Disord 1992;5:245–53.
- 6 Hodges PW. The role of the motor system in spinal pain: implications for rehabilitation of the athlete following lower back pain. J Sci Med Sport 2000;3:243–53.
- 7 Milgrom C, Finestone A, Lev B, et al. Overexertional lumbar and thoracic back pain among recruits: a prospective study of risk factors and treatment regimens. J Spinal Disord 1993;6:187–93.
- 8 Maher C, Latimer J, Refshauge K. Prescription of activity for low back pain: what works? Aust J Physiother 1999;45:121–32.
- 9 Linton SJ. A review of psychological risk factors in back and neck pain. Spine 2000;25:1148–56.
- Kendrick D, Fielding K, Bentley E, et al. Radiography of the lumbar spine in primary care patients with low back pain: randomised controlled trial. BMJ 2001;322:400–5.
- 11 Joines JD, McNutt RA, Carey TS, et al. Finding cancer in primary care outpatients with low back pain: a comparison of diagnostic strategies. J Gen Intern Med 2001;16:14–23.
- Borenstein DG. Epidemiology, etiology, diagnostic evaluation, and treatment of low back pain. Curr Opin Rheumatol 2001;13:128–34.
- 13 Deyo R, Rainville J, Kent D. What can the history and physical examination tell us about low back pain? JAMA 1992;268:760–5.
- 14 McGuirk B, King W, Govind J, et al. Safety, efficacy, and cost effectiveness of evidence-based guidelines for the management of acute low back pain in primary care. Spine 2001;26:2615–22.
- 15 Australian Acule Musculoskeletal Pain Guidelines Group. Evidence-based management of acute musculoskeletal pain. A guide for clinicians, 1st ed. Bowen Hills: Australian Academic Press Pty Ltd, 2004.
- 16 Scavone J, Latshaw R, Rohrer G. Use of lumbar spine films. Statistical evaluation at a university teaching hospital. JAMA 1981;246:1105–8.
- 17 Deyo R, Diehl A. Lumbar spine films in primary care: current use and effects of selective ordering criteria. J Gen Intern Med 1986;1:20–5.
- 18 van den Bosch M, Hollingworth W, Kinmonth A, et al. Evidence against the use of lumbar spine radiography for low back pain. Clin Radiol 2004;59:69–76.
- 19 Reinus WR, Strome G, Zwemer FL Jr. Use of lumbosacral spine radiographs in a level II emergency department. AJR Am J Roentgenol 1998;170:443-7.
- 20 Terregino C, Ross S, Lipinski M, et al. Selective indications for thoracic and lumbar radiography in blunt trauma. Ann Emerg Med 1995;26:126–9.
- 21 Gestring ML, Gracias VH, Feliciano MA, et al. Evaluation of the lower spine after blunt trauma using abdominal computed tomographic scanning supplemented with lateral scanograms. J Trauma 2002;53:9–14.
- 22 Holmes JF, Panacek EA, Miller PQ, et al. Prospective evaluation of criteria for obtaining thoracolumbar radiographs in trauma patients [see comment]. J Emerg Med 2003;24:1–7.
- 23 Durham RM, Luchtefeld WB, Wibbenmeyer L, et al. Evaluation of the thoracic and lumbar spine after blunt trauma. Am J Surg 1995;170:681–4; discussion 684–5.
- 24 Frankel HL, Rozycki GS, Ochsner MG, et al. Indications for obtaining surveillance thoracic and lumbar spine radiographs. J Trauma 1994:37:673–6.
- 25 Hsu JM, Joseph T, Ellis AM. Thoracolumbar fracture in blunt trauma patients: guidelines for diagnosis and imaging. *Injury* 2003;34:426–33.
 26 Samuels LE, Kerstein MD. 'Routine' radiologic evaluation of the
- 26 Samuels LE, Kerstein MD. 'Routine' radiologic evaluation of the thoracolumbar spine in blunt trauma patients: a reappraisal. J Trauma 1993;34:85–9.
- Gibson M, Zoltie N. Radiography for back pain presenting to accident and emergency departments. Arch Emerg Med 1992;9:28–31.
 Guillodo Y, Botton E, Saraux A, et al. Contralateral spondylolysis and fracture
- 28 Guillodo Y, Botton E, Saraux A, et al. Contralateral spondylolysis and fracture of the lumbar pedicle in an elite female gymnast: a case report. Spine 2000;25:2541–3.
- 29 Millson HB, Gray J, Stretch RA, et al. Dissociation between back pain and bone stress reaction as measured by CT scan in young cricket fast bowlers. Br J Sports Med 2004;38:586–91.
- 30 Raisz L. Screening for osteoporosis. N Engl J Med 2005;353:164-71.
- 31 Deyo R, Diehl A. Cancer as a cause of back pain. Frequency, clinical
- presentation and diagnostic strategies. J Gen Intern Med 1988;**3**:230–8. 32 **Waldvogel F**, Vasey H. Osteomyelitis: the past decade. N Engl J Med
- 1980;303:360-70.
 33 Calin A, Porta J, Fries J, et al. Clinical history as a screening test for ankylosing spondylitis. JAMA 1977;237:2613-14.
- 34 Sadowska-Wroblewska M, Filipowicz A, Garwolinska H, et al. Clinical symptoms and signs useful in the early diagnosis of ankylosing spondylitis. *Clin Rheumatol* 1983;2:37–43.
- 35 van der Linden S, Valkenburg HA, Cats A. Evaluation of diagnostic criteria for ankylosing spondylitis. A proposal for modification of the New York criteria. Arthritis Rheum 1984;27:361–8.
- 36 Gran JT. An epidemiological survey of the signs and symptoms of ankylosing spondylitis. *Clin Rheumatol* 1985;4:161–9.
- 37 Lijmer JG, Mol BW, Heisterkamp S, et al. Empirical evidence of design-related bias in studies of diagnostic tests. JAMA 1999;282:1061–6.
- 38 Gleave JR, Macfarlane R. Cauda equina syndrome: what is the relationship between timing of surgery and outcome? Br J Neurosurg 2002;16:325-8.
- 39 Gatchel RJ, Gardea MA. Psychosocial issues: their importance in predicting disability, response to treatment, and search for compensation. *Neurol Clin* 1999;17:149–66.
- 40 Linton SJ. Occupational psychological factors increase the risk for back pain: a systematic review. J Occup Rehabil 2001;11:53–66.

- 41 Pincus T, Burton AK, Vogel S, et al. A systematic review of psychological factors as predictors of chronicity/disability in prospective cohorts of low back pain. Spine 2002;27:E109-20.
- Shaw W, Pransky G, Fitzgerald T. Early prognosis for low back disability: intervention strategies for health care providers. Disabil Rehabil 2001;23:815-28
- 43 Koes B, van Tulder M, Ostelo R, et al. Clinical guidelines for the management of low back pain in primary care. An international comparison. Spine 2001:26:2504-14
- 44 Waddell G, Feder G, McIntosh A, et al. Low back pain evidence review, 1st ed. London: Royal College of General Practitioners, 1996.
- 45 Blyth F, March L, Brnabic A, *et al.* Chronic pain in Australia: a prevalence study. *Pain* 2001;89:127–34.
- 46 Blyth F, March L, Cousins M. Chronic pain-related disability and use of analgesia and health services in a Sydney community. Med J Aust 2003:179:84-7
- Walker B, Muller R, Grant W. Low back pain in Australian adults. **1**7 Prevalence and associate disability. J Manipulative Physiol Ther 2004:27:238-44.
- 48 Walker B, Muller R, Grant W. Low back pain in Australian adults. Health provider utilization and care seeking. J Manipulative Physiol Ther 2004;**27**:327–35.
- 49 Croft P, Macfarlane G, Papageorgiou A, et al. Outcome of low back pain in general practice: a prospective study. BMJ 1998;316:1356-9.
- 50 Coste J, Delecoeuillerie G, Cohen de Lara A, et al. Clinical course and prognostic factors in acute low back pain: an inception cohort study in primary care practice. BMJ 1994;308:577-80.
- 51 Schiottz-Christensen B, Nielsen GL, Hansen VK, et al. Long-term prognosis of acute low back pain in patients seen in general practice: a 1-year prospective follow-up study. *Fam Pract* 1999;**16**:223–32.
- 52 Pengel L, Herbert R, Maher C, et al. Acute low back pain: systematic review of its prognosis. BMJ 2003;327:323–7.
- Cooper JE, Tate RB, Yassi A, et al. Effect of an early intervention program on 53 the relationship between subjective pain and disability measures in nurses with low back injury. Spine 1996;21:2329–36. 54 Tate RB, Yassi A, Cooper J. Predictors of time loss after back injury in nurses.
- Spine 1999;24:1930-5; discussion 1936.
- 55 Dettori JR, Bullock SH, Sutlive TG, et al. The effects of spinal flexion and extension exercises and their associated postures in patients with acute low back pain. Spine 1995;20:2303-12.
- 56 Faas A, Chavannes A, van Eijk J, et al. A randomized, placebo-controlled trial of exercise therapy in patients with acute low back pain. Spine 1993;18:1388-95

- 57 Faas A, van Eijk JT, Chavannes AW, et al. A randomized trial of exercise therapy in patients with acute low back pain. Efficacy on sickness absence [see comment]. Spine 1995;20:941-7.
- 58 Fordyce W, Brockway J, Bergman J, et al. Acute back pain: a control-group comparison of behavioral vs traditional management methods. J Behav Med 1986.9.127-41
- Hazard RG, Haugh LD, Reid S, et al. Early prediction of chronic disability after occupational low back injury. Spine 1996;21:945–51.
 Reid S, Haugh LD, Hazard RG, et al. Occupational low back pain:
- recovery curves and factors associated with disability. *J Occup Rehabil* 1997;**7**:1–14.
- 61 Hazard RG, Reid S, Haugh LD, et al. A controlled trial of an educational pamphlet to prevent disability after occupational low back injury. Spine 2000.25.1419-23
- 62 Hides JA, Stokes MJ, Saide M, et al. Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain. Spine 1994;19:165–72.
- 63 Hides JA, Jull GA, Richardson CA. Long-term effects of specific stabilizing exercises for first-episode low back pain. Spine 2001;**26**:E243–8. 64 **Klenerman L**, Slade P, Stanley I, *et al.* The prediction of chronicity in patients
- with an acute attack of low back pain in a general practice setting. Spine 1995-20-478-84
- 65 Malmivaara A, Hakkinen U, Aro T, et al. The treatment of acute low back pain. Bed rest, exercise or normal activity? N Engl J Med 1995;332:351–5. Rozenberg S, Delval C, Rezvani Y, *et al.* Bed rest or normal activity for
- 66 patients with acute low back pain: a randomized controlled trial. [see comment]. Spine 2002;27:1487-93.
- Seferlis T, Nemeth G, Carlsson AM, et al. Conservative treatment in patients 67 sick-listed for acute low-back pain: a prospective randomised study with 12 months' follow-up. *Eur Spine J* 1998;**7**:461–70. **Seferlis T**, Nemeth G, Carlsson A. Prediction of functional disability,
- recurrences, and chronicity after 1 year in 180 patients who required sicke leave for acute low-back pain. J Spinal Disord 2006;13:470-7.
 Sieben J, Vlaeyen J, Tuerlinckx S, et al. Pain-related fear in acute low back
- ain: the first two weeks of a new episode. Eur J Pain 2002;6:229-37.
- 70 Weber H, Holme I, Amlie E. The natural course of acute sciatica with nerve root symptoms in a double-blind placebo-controlled trial evaluating the effect of piroxicam. *Spine* 1993;**18**:1433–8.
- 71 Hagen E, Eriksen H, Ursin H. Does early intervention with a light mobilization program reduce long-term sick leave for low back pain? Spine 2000;25:1973-6.
- 72 Hagen E, Grasdal A, Eriksen H. Does early intervention with a light mobilization program reduce long-term sick leave for low back pain? A 3year follow-up study. Spine 2003;28:2309-16.