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Three-Year Incidence of Low Back Pain in an Initially Asymptomatic Cohort

Clinical and Imaging Risk Factors

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Study Design. Prospective cohort study of randomly selected Veterans Affairs out-patients without baseline low back pain (LBP).

Objective. To determine predictors of new LBP as well as the 3-year incidence of magnetic resonance imaging (MRI) findings.

Summary of Background Data. Few prospective studies have examined clinical and anatomic risk factors for the development of LBP, or the incidence of new imaging findings and their relationship to back pain onset.

Methods. We randomly selected 148 Veterans Affairs out-patients (aged 35 to 70) without LBP in the past 4 months. We compared baseline and 3-year lumbar spine MRI. Using data collected every 4 months, we developed a prediction model of back pain-free survival.

Results. After 3 years, 131 subjects were contacted, and 123 had repeat MRI. The 3-year incidence of pain was 67% (88 of 131). Depression had the largest hazard ratio (2.3, 95% CI = 1.2–4.4) of any baseline predictor of incident back pain. Among baseline imaging findings, central spinal stenosis and nerve root contact had the highest, though nonsignificant, hazard ratios. We did not find an association between new LBP and type 1 endplate changes, disc degeneration, annular tears, or facet degeneration. The incidence of new MRI findings was low, with the most common new finding being disc signal loss in 11 (9%) subjects. All five subjects with new disc extrusions and all four subjects with new nerve root impingement had new pain.

Conclusion. Depression is an important predictor of new LBP, with MRI findings likely less important. New imaging findings have a low incidence; disc extrusions and nerve root contact may be the most important of these findings.

Key words: cohort study, epidemiology, low back pain, prognosis, risk factors, magnetic resonance imaging. **Spine 2005;30:1541–1548**

Researchers have questioned the clinical importance of many spine imaging findings for nearly 60 years.[1–13] Findings such as disc height loss and disc bulges are common in individuals without low back pain (LBP). As imaging techniques advance, our ability to accurately depict anatomy improves, yet paradoxically, confusion regarding the clinical importance of some anatomic findings increases.[6,8,9,12–14]

We previously reported a cohort of subjects with little or no back pain and no sciatica at the time of magnetic resonance imaging (MRI). Some reported prior back pain, allowing us to identify imaging findings associated with earlier symptoms.[15] Most imaging findings fell into one of five categories: (1) common findings with little relationship to either aging or previous back pain (e.g., annular tears, disc protrusions); (2) common findings that were associated with increased age, but not with prior symptoms (e.g., disc bulges, facet joint degeneration, endplate changes, mild spondylolisthesis); (3) common findings related both to aging and previous LBP (e.g., decreased disc signal on T2-weighted images, decreased disc height); (4) rare findings unrelated to age, but strongly associated with previous back pain (disc extrusions and nerve root contact); and (5) moderate or severe stenosis, which was related to previous back pain, mild current symptoms and aging.

We sought to determine the 3-year follow-up incidence of LBP in this cohort and identify its risk factors. We also aimed to describe incident imaging findings and their relationship to onset of new symptoms.

■ Materials and Methods

Cohort Assembly. We previously described details of the cohort assembly.[15] Briefly, we used electronic records to randomly sample patients from four clinics at the Veterans Affairs Puget Sound Health Care System, Seattle Division, stratifying by age, with half of the subjects aged 35 to 52 years and the remainder aged 53 to 70.

After excluding patients with ICD-9 diagnostic and procedure codes related to LBP or lumbar surgery, we contacted the remaining patients in random order. We excluded subjects with any history of back surgery, chymopapain injections, discography, acute lumbar trauma, fibromyalgia, peripheral neuropa-

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thy, or serious comorbid disease that could affect 3-year survival. Eligible subjects completed a back pain bothersomeness questionnaire and a modified Roland Disability Scale.[16] We excluded patients with any sciatica or LBP more than "mildly bothersome" in the previous 4 months, or a modified Roland score ≥3.

All imaging was free, and we reimbursed subjects $55. The University of Washington Institutional Review Board approved the study and all subjects gave written informed consent.

Baseline and Outcome Measures. Participants completed a baseline questionnaire that included demographic information, comorbidity, back pain-specific functional status, general functional status, and health-related quality of life.

Our primary measure of back pain and sciatica was the pain frequency index (PFI).[17] Subjects rated the frequency of the following four symptoms on a 1 (none of the time) to 6 (all of the time) point scale: (A) low back or buttock pain; (B) sciatic leg pain; (C) numbness or tingling in the leg, foot, or groin; and (D) weakness in leg or foot. The aggregate index scores ranged from 4 to 24. We defined an incident case as a subject who rated pain frequency for low back or buttock pain as more than "some of the time" (>2), or any of the other three symptoms as more than "none" (>1).

We used the modified Roland Disability Scale to assess the functional impact of LBP. Scores range from 0 (no dysfunction) to 23 (most severe functional impairment).

We assessed health-related quality-of-life with the SF-12,[18,19] a 12-item version of the SF-36 general health status questionnaire. The SF-12 provides physical and mental component scores, which range from 0 (poor health) to 100 (good health) and are normalized so that a score of 50 equals the mean score in the US general population.

Subjects completed a comorbidity questionnaire[17] that relies on self-reported conditions. Subjects classified their previous history of LBP as none previously, 1 to 5 episodes, or >5 episodes.

We contacted subjects every 4 months by telephone, except at 36 months, when they returned for a repeat MRI. We asked all outcome questions at 12, 24, and 36 months. At all other follow-ups, subjects were given the full battery of questions only if they scored back pain >2 on the pain frequency questionnaire.

Imaging. We used a Philips 1.5 Tesla system to perform all MRIs, obtaining sagittal and axial T1- and T2-weighted images through each of the five lumbar intervertebral disc levels. One of two attending neuroradiologists, both senior members of the American Society of Neuroradiology and with clinical and academic expertise in lumbar spine imaging, interpreted all MRI images (J.G.J., D.R.H.). Other than knowing subjects were asymptomatic at baseline, clinical information was concealed from the radiologists.

The radiologists independently interpreted a sample of the examinations, and agreement between readers for each finding was calculated using the unweighted κ statistic for dichotomous variables and the weighted κ statistic for ordinal variables.[20,21] The radiologists discussed disagreements and reached a consensus interpretation. We repeated this process until agreement was substantial, after which the radiologists each interpreted half of the remaining images.

The same radiologist interpreted the baseline and 3-year images to avoid inter-reader variability. The radiologists reviewed both the baseline images and their interpretations when reading the follow-up images, again to minimize variability other than true anatomic changes.

Subjects were not informed of MRI findings, unless a finding required immediate medical attention, because "labeling" patients with MRI diagnoses might have altered their behavior, sensitized them to trivial symptoms, or patients might have amplified symptoms that occurred.

At each lumbar disc level, the radiologists recorded the presence or absence of anatomic findings that are postulated to cause back pain or sciatica. Whenever possible, the radiologists used published diagnostic definitions.[8,22–25] Disc herniations were classified as protrusions or extrusions.[26] Endplate changes were classified as type 1 (edema), type 2 (fat), and type 3 (sclerosis) according to the scheme of Modic *et al*.[27] See Appendix for a complete description of imaging variables.

Data Analysis. We used Microsoft Access and SPSS[28] for data management and analysis, respectively. We applied the χ^2 test for trend (linear by linear) for ordinal variables, Yates continuity correction for dichotomous variables, and Fisher exact test for sparse contingency tables.[29]

We used a proportional hazards model to investigate the relationship between the incident LBP and potential risk factors. Clinical covariates included in our primary model were age, gender, history of prior LBP, smoking history, body mass index (BMI), and depression. Depression was ascertained with a checklist adapted from previous studies, using the following 3 questions: (1) Do you have depression? (2) Are you being treated for depression? (3) Does depression limit your activities? Baseline imaging covariates tested in our model were bulge, protrusion, extrusion, disc height loss, disc signal loss, annular tear, type 1 endplate findings,[27] central stenosis, facet degeneration, and nerve root contact.

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■ Results

Subject Characteristics

Of 148 enrolled subjects, 123 returned for repeat MRI after 3 years. We contacted eight additional subjects by telephone, making the 3-year follow-up rate 88.5% (131 of 148). We contacted 94 subjects (64%) at all 9 follow-up intervals. The follow-up rates at the 4-month time-points varied between 84.5% at 12 months and 98.6% at 4 months, with a mean of 90.7%. Because of occasional missing data, the sample size in each of the subsequent analyses varies slightly.

Compared with subjects available for follow-up, the 17 subjects without follow-up were more likely to be disabled for nonback pain causes [7 of 17 (41%) versus 21 of 131 (16%), $P = 0.02$, χ^2], have been hospitalized for back pain [2 of 17 (12%) versus 1 of 131 (1%), $P =$

Table 1. Subject Characteristics (n = 131)

	Under Age <53 (n = 68)	Aged ≥53 (n = 63)	*P* (Difference Between Age Groups)
Female (%)	13 (19)	4 (6)	0.030 *
Ethnic origin			
White/caucasian (%)	56 (82)	56 (89)	0.289
Black/African American (%)	6 (9)	4 (6)	0.594
Other			
Married (%)	41 (60)	28 (44)	0.069
Highest level of education			
Less than high school (%)	1 (1)	2 (3)	0.137
High school graduate (%)	39 (57)	22 (51)	
College graduate (%)	28 (41)	29 (46)	
Employment status (not mutually exclusive)			
Working full time (%)	37 (54)	9 (14)	<0.001
Disabled, not due to back pain (%)	11 (16)	10 (16)	0.962
Other medical problems			
Hypertension (%)	18 (26)	36 (57)	<0.0001
Arthritis (%)	11 (16)	17 (27)	0.14
Depression (%)	14 (21)	7 (11)	0.16
Diabetes (%)	7 (10)	13 (21)	0.09
Heart disease (%)	2 (3)	11 (18)	0.005
Current smoker (%)	18 (26)	9 (14)	0.15
Mean BMI (min-max)	28.3 (18.3–43.3)	28.4 (18.6–42.9)	0.95 †
Previous back pain			0.005
None (%)	23 (34)	38 (60)	
One to five times (%)	30 (44)	20 (32)	
More than five times (%)	15 (22)	5 (8)	
Location of previous back pain (% of those with prior pain)			0.76
Back only (%)	39 (57)	21 (33)	
Traveled into one or both legs (%)	6 (13)	4 (16)	
Ever hospitalized for LBP or sciatica (%)	0 (0)	1 (2)	0.47 ‡
Surgery or injections for LBP or sciatica (%)	0 (0)	0 (0)	NA

* All *P* values based on Pearson $\chi[\sigma\upsilon\pi]2[\rho]$ asymptotic 2-sided test except where otherwise noted.
† Independent two-sample *t* test.
‡ Fisher exact test.

0.04, Fisher exact test], and have heart disease [5 of 17 (29%) versus 13 of 131 (10%), $P = 0.04$, Fisher exact test]. The mean age at enrollment of subjects available for follow-up was 54 (9.9 SD) years and the majority of subjects were white males, reflecting the population at this Veterans Affairs hospital (Table 1). Forty-seven percent (69 of 148) of the subjects had never experienced LBP and 16% (23 of 148) had had more than five episodes. All 21 subjects with self-identified depression at baseline were either being treated for depression (19 of 21) or said that their activities were limited by depression (15 of 21). Moreover, subjects with self-identified depression had significantly lower mean SF-12 mental health component summary scores than subjects without depression [56.5 (4.2 SD) versus 38.2 (12.9 SD), $P < 0.001$]. Additional baseline findings can be found in a previous publication.[15]

LBP and Back-Related Functional Status

For our primary analysis, we defined incident back pain as a score of >2 for low back pain or >1 for leg pain, numbness or weakness on the 6-point PFI at any time during the 3-year follow-up. Thus, our definition of incident back pain also includes radicular pain. Correlations between the symptom frequency index and other measures of back symptoms and function were strong (Pearson correlation coefficient between the pain frequency index and Roland disability scale at 3 years was 0.78, $P < 0.01$).

Sixty-seven percent of subjects (88 of 131) had incident LBP over 3 years, with 58 subjects (44%) having pain that was at least moderately bothersome. These proportions were not significantly different for the subjects <53 versus ≥53, so we report only data for the entire cohort. The SF-12 was relatively stable over time, but there was deterioration in the back-specific pain and functional status measures.

Incident MRI Findings (n = 123)

New imaging findings were infrequent. The most common new finding after 3 years was disc signal loss in 11 of 123 (9%; Table 2). No subjects developed MRI evidence of cancer or infection.

There were 114 discs in 67 subjects that had decreased height at both baseline and follow-up, and an additional 8 discs in 6 subjects that had lost height after 3 years. At baseline, 3 of these 6 subjects had demonstrated height loss at other levels.

Five subjects had a disc that changed from normal to bulging, 8 had a disc that changed from normal to protrusion, and 1 had a disc that changed from bulging to protrusion. Five subjects had a disc that changed from normal to extrusion, and 4 of these had new nerve root contact with these disc extrusions (Figure 1).

Table 2. Baseline Prevalence and Cumulative 3-Year Incidence of New MRI Findings (n = 123)

Finding	No. (%) Subjects With Finding at Baseline (n = 148) (Prevalence)	No. (%) of Subjects With New Finding (n = 123) (Incidence)	No. (%) of Subjects With Improved Finding (n = 123)
Disc signal loss	124 (84)	11 (9)	0
Endplate change (any type)	39 (26)	10 (8)	0
Disc protrusion	49 (33)	9 (7)	2 (2)
Facet degeneration	27 (18)	9 (7)	0
Bulge	65 (44)	6 (5)	3 (2)
Disc height loss	84 (56)	6 (5)	0
Annular tear	57 (38)	6 (5)	0
Disc extrusion	9 (6)	5 (4)	0
Root contact	5 (3)	4 (3)	0
Lateral recess stenosis (moderate or severe)	3 (2)	4 (3)	0
Central stenosis (moderate or severe)	15 (10)	2 (2)	0
Spondylolisthesis	27 (18)	2 (2)	1 (1)

Some imaging findings became less severe or even normal. Four discs in 3 subjects changed from bulging to normal. Two subjects had protruded discs that transformed to bulges, and one of these subjects had a 10% retrolisthesis that reverted to 0% (Figure 2).

There were 8 levels in 7 subjects that changed from normal endplates at baseline to type 1 at 3 years, and 3 levels in 3 subjects that changed from normal to type 2. There was only 1 level that changed from type 1 to type 2 at 3 years. All 42 discs with type 2 endplates, and the

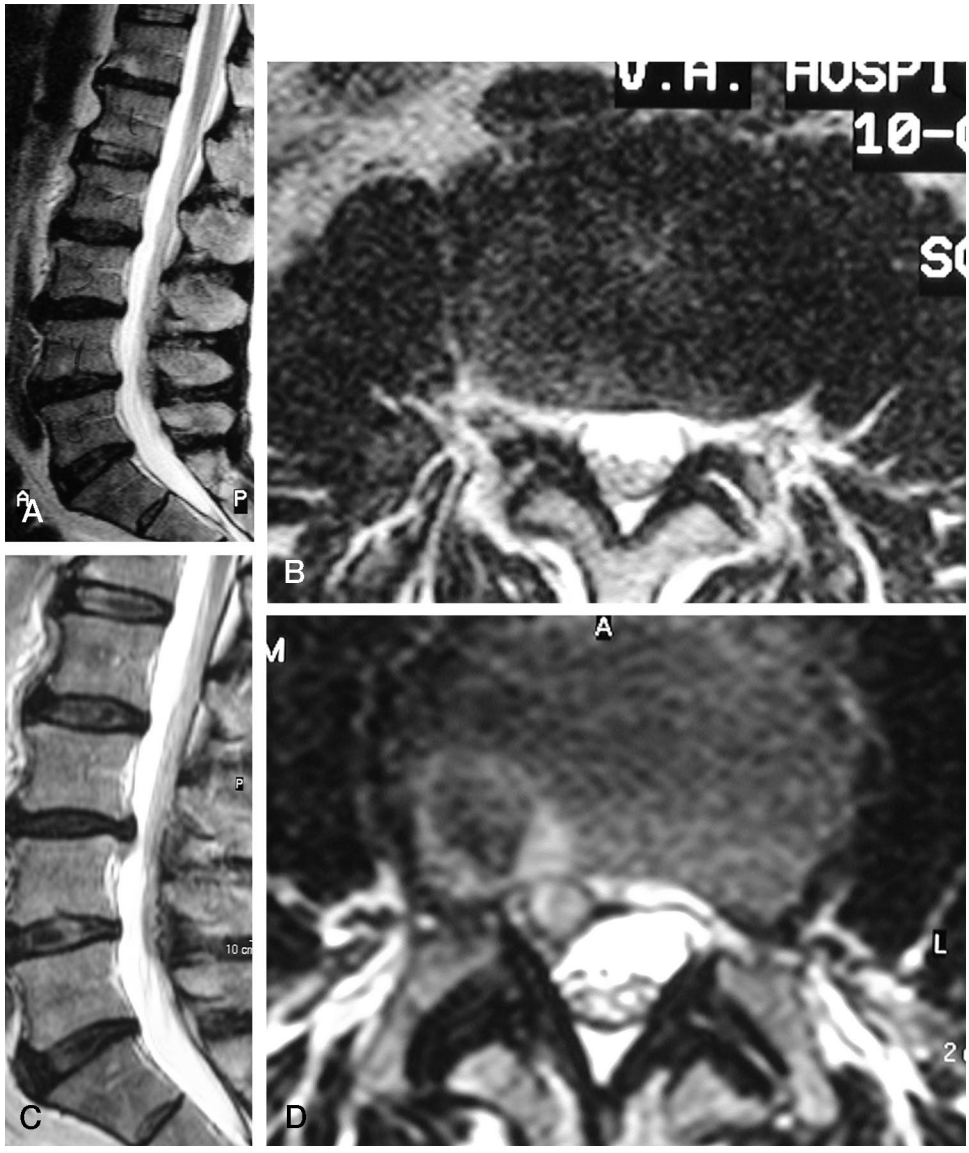

Figure 1. Sagittal (**A**) and axial (**B**) T2-weighted images demonstrate a diffuse bulge at the L3–L4 disc but no evidence of a posterior disc extrusion. At the 3-year follow-up imaging, sagittal T2-weighted image (**C**) demonstrates a new disc extrusion. On axial images this new extrusion is located in the right paracentral and subarticular (**D**) regions and contacts the L4 nerve root in the lateral recess.

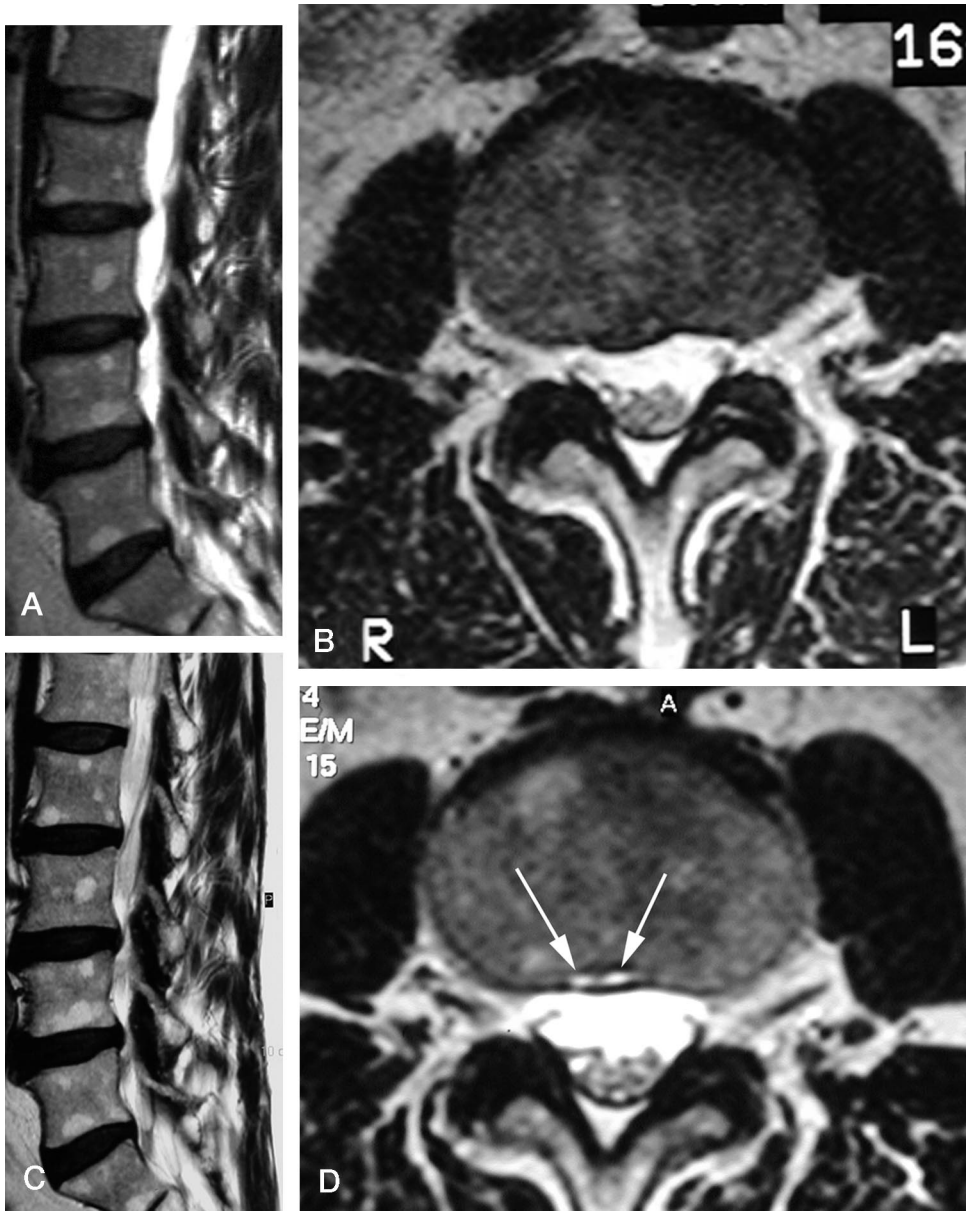

Figure 2. Sagittal (**A**) and axial (**B**) T2-weighted images at baseline demonstrate a small, right paracentral disc protrusion associated with a mild (10%) retrolisthesis of L2 on L3. Repeat imaging at 3-years (**C** and **D**) shows resolution of the protrusion and listhesis, but continued diffuse bulging at this level. High signal in the anulus fibrosus is now present where the protrusion had been (arrows). Incidental note is made of focal fat deposition at multiple levels.

one disc with a type 3 endplate at baseline remained stable over 3 years.

Risk of Incident LBP with Imaging and Clinical Factors

We used Cox regression models to determine which baseline factors predicted new onset of back pain during follow-up. Separate models of individual baseline imaging variables controlling only for age and a multivariate model that combined clinical factors and the three baseline imaging findings with the largest univariate hazard ratios and clinical factors produced similar findings. Therefore we report only the results of the multivariate model (Table 3). Self-identified depression was the strongest predictor of subsequent back pain, with a greater hazard ratio (HR) than any imaging finding (HR = 2.3, 95% CI = 1.2–4.4). Surprisingly, disc protrusions were associated with a lower risk of subsequent back pain (HR = 0.5, 95% CI = 0.3–0.9). Nerve root contact (HR = 1.9, 95% CI = 0.6–5.8) and central stenosis (HR = 1.8, 95% CI = 0.8–4.1) had the largest hazard ratios among baseline imaging findings, and they were associated with incident back pain in the expected directionbut not statistically significant. Figure 3 depicts the survival curve generated by this Cox regression, comparing groups with and without baseline depression.

Pain measures among subjects with (n = 123) and without (n = 25) baseline depression were similar (little or none) at baseline, but pain scores among depressed patients were greater at every follow-up time point. The difference in the proportion of subjects with and without baseline depression who developed back pain was greatest at year 1 (0.71 versus 0.34, $P < 0.01$), then decreased through year 3 (0.86 versus 0.75, $P = 0.24$).

The paucity of new imaging findings during the 3 years limited our power to detect a significant relation-

Table 3. Multivariate Cox Regression Hazard Ratios for Back Pain Prediction (n = 128, 72 events, 56 censored)

	Hazard Ratio	95% CI
Age	1	0.99–1
Prior low back pain (>5 episodes)	1.4	0.6–2.9
Current smoker	0.6	0.3–1.3
Depression	2.3	1.2–4.4
BMI	1	1.0–1.1
Male gender	0.9	0.4–2
Baseline imaging results		
Disc extrusion	1.2	0.4–3.4
Disc protrusion	0.5	0.3–0.9
Nerve root contact	2.2	0.6–8
Central stenosis	1.9	0.8–4.8

Note. All variables included in model.

ship between new imaging findings and incident LBP. However, all five subjects with new extrusions (Fisher exact test, $P = 0.17$), all four subjects with new nerve root contact (Fisher exact test, $P = 0.3$) and both subjects with new central stenosis (Fisher exact test, $P = 1$) developed pain.

■ Discussion

Our results indicate that depression is a stronger predictor of who will subsequently report LBP than baseline imaging findings. Subjects with self-reported depression at baseline were 2.3 times as likely to have back pain compared with those who do not report depression. Over 3 years, progression of anatomic abnormalities was infrequent and only occasionally associated with new onset of back pain. The small numbers of new imaging findings indicate that we would need more subjects or longer follow-up to identify associations between MRI findings and new back pain. However, even with our relatively small number of subjects, we were able to demonstrate that findings of nerve root contact, disc extrusion, or central canal stenosis were associated with both previous back pain and, possibly, new onset of pain.

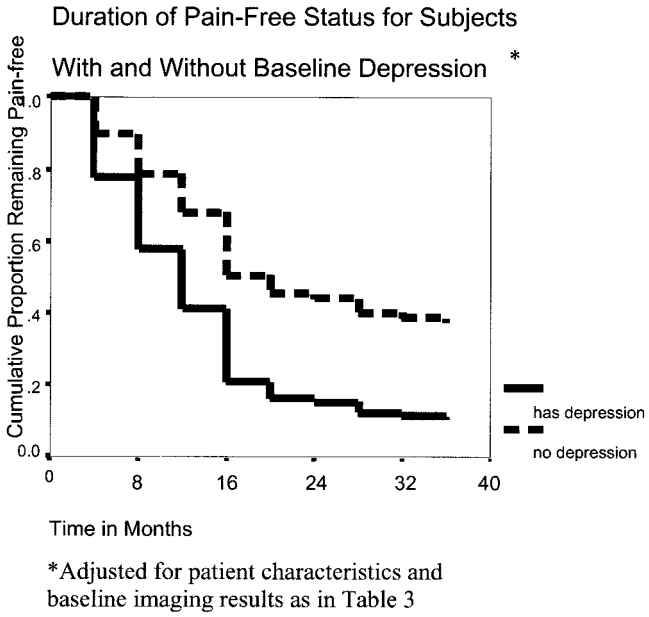

Figure 3. Survival curve for the development of LBP, comparing subjects who had depression at baseline with those that did not, controlling for baseline imaging results and clinical characteristics. Subjects without baseline depression had longer pain-free survival than those with baseline depression. The number of subjects at each time point for the survival analysis were as follows: baseline = 130; 4 months = 111; 8 months = 85; 12 months = 63; 16 months = 43; 20 months = 37; 24 months = 35; 28 months = 32; 32 months = 31.

Other investigators have demonstrated the importance of psychological and psychiatric factors both as predictors for future pain and correlates of current LBP.[30,31] In his review, Linton concluded that there is strong evidence that depression is related to levels of pain and disability. Whereas prior studies have demonstrated the importance of psychological and psychiatric factors, they have not compared the importance of these factors with anatomic factors based on imaging.

We did not use a depression-specific scale, such as the Beck depression score, because we were concerned about questionnaire burden. However, the validity of our screen is supported by the fact that all subjects with depression were either being treated for depression or had their activities limited by depression. The lower SF-12 mental component scores among those with depression further help to validate our assessment of depression. Nevertheless, future studies should probably incorporate depression-specific scales to verify the importance of depression as a predictor of future back pain.

Our study did not demonstrate a statistically significant association of any imaging finding, either baseline or incident, with new back pain, possibly attributable in part to our small sample size and relatively short follow-up. However, among baseline imaging findings, nerve root contact and central canal stenosis had the highest hazard ratios for new pain.

Our data present a consistent picture with prior studies. In our analysis of baseline data,[15] we concluded that central stenosis, nerve root contact, and disc extrusion were the most important imaging findings related to prior LBP. Our current analysis indicates that central stenosis, disc extrusion, and root contact may also be risk factors for future LBP.

The role of disc protrusions is surprising. Not only were pre-existing protrusions not a risk factor for subsequent LBP, but protrusions were associated with a lower risk of developing back pain, despite 3 of the 5 subjects with root compression having had protrusions. Subjects without protrusions were twice as likely to develop back pain as subjects with protrusions. Disc extrusions were associated with past back pain but protrusions were not. We speculate that protrusions could represent a less painful mechanism for discs to degenerate, perhaps by relieving intradiscal pressure without inciting an inflammatory reaction or nerve root compromise. However, firm conclusions should await confirmation by other investigators.

One ambiguity in associating new imaging findings with incident pain is the uncertainty of which came first. Future studies can address this problem to a certain extent by obtaining new imaging studies at the time of incident pain.

Five subjects had imaging "abnormalities" improved after 3 years. This emphasizes a phenomenon already described for disc extrusions but not for disc bulges or protrusions.

Imaging findings that were not associated with new back pain are just as important as those that were. Annular tears, type 1 endplate changes, disc height loss, disc bulges, and facet degeneration were not associated with either past or future LBP. This result challenges treatment decisions based largely on these imaging findings.

Also somewhat surprising is the higher prevalence of prior back pain in the subjects under age 53. This may be because of recall bias, with older subjects finding it more difficult to remember prior episodes of LBP, or possibly stoicism among the elderly.

The generalizability of our results may be limited by the low proportion of women, non-whites, and subjects not working full-time and the relatively high proportion of disabled people compared with the US population at large. These demographics are reflective of the Veterans Affairs population in the Pacific Northwest. Nevertheless, other populations should be studied to validate the importance of psychological factors as predictors of back pain.

Our findings are an additional step toward disentangling the complex relationship between MRI findings and LBP. They reinforce the importance of some imaging findings and suggest that others may have little clinical relevance. Finally, our findings emphasize that anatomic findings may not be the best predictors of who develops LBP. We suggest that when treating patients with LBP, medical providers should pay greater attention to the important role of depression rather than focusing on the findings of imaging studies.

■ Key Points

- Depression is an important predictor of new low back pain.
- MRI findings are likely less important in predicting future back pain than psychological factors.
- New imaging findings have a low incidence.
- Disc extrusions and nerve root contact may be the most clinically important new imaging findings.
- We did not find an association between new low back pain and Type 1 endplate changes, disc degeneration, annular tears, or facet degeneration.

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Note. Appendix available for viewing online only through ArticlePlus.

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