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# Effect of Intensive Patient Education vs Placebo Patient Education on Outcomes in Patients With Acute Low Back Pain
## A Randomized Clinical Trial

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**IMPORTANCE** Many patients with acute low back pain do not recover with basic first-line care (advice, reassurance, and simple analgesia, if necessary). It is unclear whether intensive patient education improves clinical outcomes for those patients already receiving first-line care.

**OBJECTIVE** To determine the effectiveness of intensive patient education for patients with acute low back pain.

**DESIGN, SETTING, AND PARTICIPANTS** This randomized, placebo-controlled clinical trial recruited patients from general practices, physiotherapy clinics, and a research center in Sydney, Australia, between September 10, 2013, and December 2, 2015. Trial follow-up was completed in December 17, 2016. Primary care practitioners invited 618 patients presenting with acute low back pain to participate. Researchers excluded 416 potential participants. All of the 202 eligible participants had low back pain of fewer than 6 weeks' duration and a high risk of developing chronic low back pain according to Predicting the Inception of Chronic Pain (PICKUP) Tool, a validated prognostic model. Participants were randomized in a 1:1 ratio to either patient education or placebo patient education.

**INTERVENTIONS** All participants received recommended first-line care for acute low back pain from their usual practitioner. Participants received additional 2 × 1-hour sessions of patient education (information on pain and biopsychosocial contributors plus self-management techniques, such as remaining active and pacing) or placebo patient education (active listening, without information or advice).

**MAIN OUTCOMES AND MEASURES** The primary outcome was pain intensity (11-point numeric rating scale) at 3 months. Secondary outcomes included disability (24-point Roland Morris Disability Questionnaire) at 1 week, and at 3, 6, and 12 months.

**RESULTS** Of 202 participants randomized for the trial, the mean (SD) age of participants was 45 (14.5) years and 103 (51.0%) were female. Retention rates were greater than 90% at all time points. Intensive patient education was not more effective than placebo patient education at reducing pain intensity (3-month mean [SD] pain intensity: 2.1 [2.4] vs 2.4 [2.2]; mean difference at 3 months, −0.3 [95% CI, −1.0 to 0.3]). There was a small effect of intensive patient education on the secondary outcome of disability at 1 week (mean difference, −1.6 points on a 24-point scale [95% CI, −3.1 to −0.1]) and 3 months (mean difference, −1.7 points, [95% CI, −3.2 to −0.2]) but not at 6 or 12 months.

**CONCLUSIONS AND RELEVANCE** Adding 2 hours of patient education to recommended first-line care for patients with acute low back pain did not improve pain outcomes. Clinical guideline recommendations to provide complex and intensive support to high-risk patients with acute low back pain may have been premature.

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Supplemental content

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For the past 5 years, the Global Burden of Disease Study[1] has consistently ranked low back pain as the leading cause of disability worldwide. Low back pain is second only to the common cold as a reason for consulting a general practitioner.[2] A recent international review highlighted a global crisis in the mismanagement of low back pain, with high rates of guideline-discordant care in both high- and low-middle income countries.[3-5] In their call to action, the Lancet Low Back Pain Series Working Group authors recommended that researchers and policy makers: "Develop and implement strategies to ensure early identification and adequate education of patients with low back pain at risk for persistence of pain and disability."[3-5]

To manage uncomplicated acute low back pain (fewer than 6 weeks of pain duration), international guidelines recommend that general practitioners provide advice, education, reassurance, and simple analgesics, if necessary.[6] Although many patients receiving this care improve rapidly, 33% experience a recurrence in the next 12 months[7] and 20% to 30% develop chronic pain (defined as pain duration of 3 months or more).[8]

Patients who are at high risk of pain chronicity may require additional care, including second-line options such as physical (eg, spinal manipulation) and/or psychological therapies (eg, psychologically informed physiotherapy).[6] However, most trials that have evaluated adding second-line treatment options to standard guideline care for patients with acute low back pain have failed to demonstrate effectiveness compared with placebo (eg, addition of spinal manipulation, nonsteroidal antiinflammatory drugs, or both[9]; addition of structured exercises[10]; and addition of acupuncture, massage, or chiropractic care[11]). Patient education, a treatment that authors of a 2008 Cochrane review[12] concluded was effective for acute low back pain when applied in an intensive format and that every major clinical guideline recommends (but with little instruction on intensity),[13] has never been tested in a placebo-controlled trial. Any benefits observed in previous trials of patient education for acute low back pain could be explained by nonspecific effects of the clinical encounter or the characteristics of the usual care comparison.

Pain education, a form of intensive patient education that is often included in pain management programs, requires up to 2 hours during several encounters with a trained health practitioner. It involves detailed discussion of pain, including psychosocial contributors and advice about pacing and activity. Trials have found clinically meaningful effects of pain education on pain and disability in samples of patients with chronic pain.[14]

It is unknown whether intensive patient education, in addition to recommended first-line care, can improve outcomes for patients with acute low back pain. To address this gap in the literature, we conducted, to our knowledge, the first randomized, placebo-controlled trial of patient education for acute low back pain (Preventing Chronic Low Back Pain [PREVENT] Trial).[15]

### Key Points

**Question** Is intensive patient education effective as part of first-line care for patients with acute low back pain?

**Findings** In this randomized clinical trial of 202 adults with acute low back pain from Sydney, Australia, adding intensive patient education to first-line care of patients was no better at improving pain outcomes than a placebo intervention.

**Meaning** Intensive patient education should not be offered to patients with acute low back pain who are receiving first-line care.

## Methods

### Study Design

This was an assessor-blinded, 1:1 parallel group, randomized, placebo-controlled trial. We published a study protocol prior to enrolling participants[15] (the original trial protocol is available in Supplement 1). The trial was prospectively registered. The University of New South Wales Human Research Ethics Committee, Sydney, New South Wales, Australia, approved the study on February 5, 2013 (reference number: HC12664). We obtained written, informed consent from all participants before they enrolled in the trial.

Treatments took place at physiotherapy clinics, general practices, or clinic rooms at a research institute (Neuroscience Research Australia) in Sydney, Australia. One of 2 trial clinicians (A.C.T. and I.W.S.) provided the treatment at participating centers. We recruited participants between September 10, 2013, and December 2, 2015. Trial follow-up was completed on December 17, 2016.

### Participants

We sought to recruit participants aged 18 to 75 years who were seeking care for acute low back pain with or without referred leg pain. Participants with signs of radiculopathy (spinal nerve root compromise) were included. All participants were referred from general practitioners or physiotherapists. We excluded potential participants if they had the following: (1) chronic low back pain (more than 1 on a 11-point pain intensity numeric rating scale for more than 3 months), (2) less than 3 of 10 on the pain intensity numeric rating scale over the past week, (3) low risk of pain chronicity (less than 30% absolute risk of chronic pain according to the Predicting Inception of Chronic Pain (PICKUP) Tool[8] [eMethods 1 in Supplement 2]), (4) clinical features of serious spinal pathology (eg, cauda equina syndrome, infection, fracture, or cancer) assessed by a clinician, (5) poor command of the English language, (6) previous spinal surgery, or (7) a mental health condition that would preclude study participation. Referring clinicians were trained to provide all recruited participants with guideline-based care (advice to stay active, avoid bed rest, option of spinal manipulation, and/or simple analgesics). Staff were reimbursed per participant recruited for time spent on the study.

### Randomization and Masking

We randomized participants in a 1:1 ratio to either intensive patient education or placebo patient education. The allocation schedule was generated by a researcher not involved in any other aspect of the study. That researcher used a computerized random number table to generate the allocation sequence in random block sizes of 4, 6, 8, and 10. The same re-

searcher who generated the allocation sequence placed allocation codes into sequentially numbered, sealed, opaque envelopes.

Before randomization, all participants completed baseline data collection and received a standardized short history and physical examination (approximately 10-minute length) with the trial clinicians (A.C.T. and I.W.S.). The short history and physical examination were standardized using pro forma documents (eMethods 2 in Supplement 2). The trial clinicians opened the envelope containing the group allocation. The allocation was concealed from participants, referring clinicians, other trial staff, and outcome assessors.

All treatment was provided during the acute phase of low back pain within 6 weeks of pain onset. Each participant received 2 × 1-hour individual, face-to-face sessions of either patient education or placebo patient education. The trial clinicians (A.C.T. and I.W.S.) who provided the patient education sessions were the same clinicians who provided the placebo patient education. An expert in pain education (G.L.M.) trained both trial clinicians to deliver the patient education intervention. An expert clinical psychologist in pain management (M.K.N.) trained both trial clinicians in the placebo patient education intervention. Training for the patient education intervention took approximately 16 hours, with 6 to 8 hours allocated for practicing role-play scenarios. Training for the placebo patient education took approximately 4 hours and was supplemented with 4 online 45-minute videos demonstrating techniques for providing a credible consultation that did not include advice or education.

## Interventions

### Intensive Patient Education

We adapted the information and advice provided in the patient education group from the book *Explain Pain*,[16] a text typically used for people with chronic pain. The intervention is described in full and according to the template for intervention description and replication (TIDieR) checklist in eMethods 3 in Supplement 2. In short, participants in the patient education group were provided with a detailed explanation about the biopsychosocial nature of pain in the format of diagrams, metaphors, and stories. The patient education intervention involved 3 main components: (1) reframing unhelpful beliefs about low back pain, (2) presenting information about the biologic basis and protective nature of both acute and chronic low back pain, and (3) evaluating understanding of new concepts and discussing techniques to promote recovery. Content was tailored to the individual according to specific concerns (eg, "I am worried I will have this back problem forever") and misconceptions (eg, "I can't work because my back is permanently damaged") that participants expressed during the consultation. Trial clinicians encouraged all participants to self-manage their low back pain by remaining active and avoiding bed rest. Trial clinicians also instructed participants on behavioral therapy techniques such as pacing.

### Placebo Patient Education

We designed the placebo patient education sessions to control for time with an expert clinician. The sessions mimicked all aspects of the patient education sessions (listening, showing interest, and attention of the clinician) but without the education component. Participants in the placebo patient education group received no information, advice, or education about low back pain from the trial clinician. Participants were encouraged to talk about any topic that they desired. Trial clinician responses were aimed to maintain the discussion for the duration of the session. We included additional detail on the placebo intervention in eMethods 4 in Supplement 2.

## Outcomes and Measurements

We collected self-reported data from participants at baseline (the first intervention session); 1 week after the 2 intervention sessions were complete; and 3, 6, and 12 months after the date of low back pain onset. Participants used online forms to complete outcome assessments. Baseline data included age, sex, duration of episode, number of previous episodes, other painful areas, and work status. An assessor who was masked to treatment allocation arranged the collection of outcome data using online forms. Participants completed the credibility and expectancy questionnaire[17] in paper format immediately after the trial clinician explained the rationale for the study and before randomization. Trial staff monitored adherence to the 2 intervention sessions using a study calendar. The trial clinician audio recorded all intervention sessions, with the participants' verbal consent, to monitor treatment fidelity. Treatment fidelity was evaluated by 2 researchers (G.L.M. and M.K.N.), who listened to the first and second sessions from 10 randomly selected participants and judged whether the sessions were patient education or placebo patient education. We used κ to determine agreement.

The primary outcome was mean pain intensity during the past week (reported on an 11-point pain intensity numeric rating scale), assessed 3 months after the onset of low back pain. Secondary outcomes and process measures are described in eMethods 5 of Supplement 2.

## Statistical Analysis

We published our statistical analysis plan before analyzing our results.[18] A sample of 202 participants was required to ensure 80% power to detect a mean difference of 1 point on an 11-point numeric rating scale for pain intensity. Our power calculation assumed an SD of 2.3 and a 2-sided α of .05 and was adjusted with 15% loss to follow-up. We estimated the effect of the intervention on the primary outcome using a mixed model for repeated measures. We treated time as a categorical variable (1 week and 3, 6, or 12 months) and included group × time interactions to determine treatment effects at each time point. As an exploratory sensitivity analysis, we calculated *P* values from mixed models for repeated measures comparing between-group difference during the full 12-month trial, controlling for baseline and including all time points as categorical. We determined statistical significance to be $P < .05$ for a 2-sided test. We did not include study site (physiotherapy practice, general practice, or research institute) in the model because there was no evidence of site differences between groups ($\chi^2$ test, $P = .14$). Details of the analysis of secondary outcomes is provided in eMethods 5 of Supplement 2 and the complete mediation analysis[19] in eResults 1 of Supplement 2. Two authors (S.L. and H.L.) performed the statistical analyses.

## Results

Between September 10, 2013, and December 2, 2015, we screened 618 potential participants. **Figure 1** shows the flow of participants through the trial. The main reasons for participant exclusion included low risk of pain chronicity (n = 146), chronic pain (n = 79), declined participation (n = 75), or could not be contacted after initial referral from the primary care practitioner (n = 75). Other reasons for exclusion are shown in Figure 1. One potential participant was excluded in error because of pregnancy.

The 2 groups had similar demographic and clinical characteristics at baseline (**Table 1**). Of 202 participants randomized for the trial, 103 (51.0%) were female. Participants were middle-aged (mean [SD] age, 45.1 [14.5] years), had fewer than 2 weeks of low back pain, and had experienced 3 previous episodes of low back pain. Physiotherapists referred most participants (83%). Half of the sample (52%) felt there was a need for further investigation of their symptoms. Psychological characteristics were similar between groups; scores for depression and catastrophizing scales were lower and scores for self-efficacy were higher than those seen in samples from patients with chronic pain who attended tertiary care.[20]

All participants completed both trial sessions. Treatment credibility scores were not different between groups (mean [SD] credibility and expectancy questionnaire score for patient education vs placebo patient education: 36.6 [8.8] vs 35.3 [10.5]; mean difference, -1.3; 95% CI, -4.0 to 1.4). For our treatment fidelity check, raters correctly categorized all recordings as patient education or placebo patient education. There was perfect agreement between raters (κ = 1).

The primary analysis (**Table 2**) showed that patient education was not more effective than placebo patient education at reducing pain intensity at our primary end point (3-month follow-up mean difference, -0.3 points on an 11-point scale; 95% CI, -1.0 to 0.3; *P* = .31). Mean (SD) pain intensity decreased from 6.3 [2.4] at baseline to 2.1 [2.4] at 3 months in the patient education group and from 6.1 [2.2] at baseline to 2.4 [2.2] at 3 months in the placebo patient education group. (**Figure 2**).

There was a small effect of treatment group on disability, with patient education lower than placebo patient education at 1 week (mean difference, -1.6 points on a 24-point scale;

**Figure 1. Flowchart of the Preventing Chronic Low Back Pain (PREVENT) Randomized Placebo-Controlled Trial**

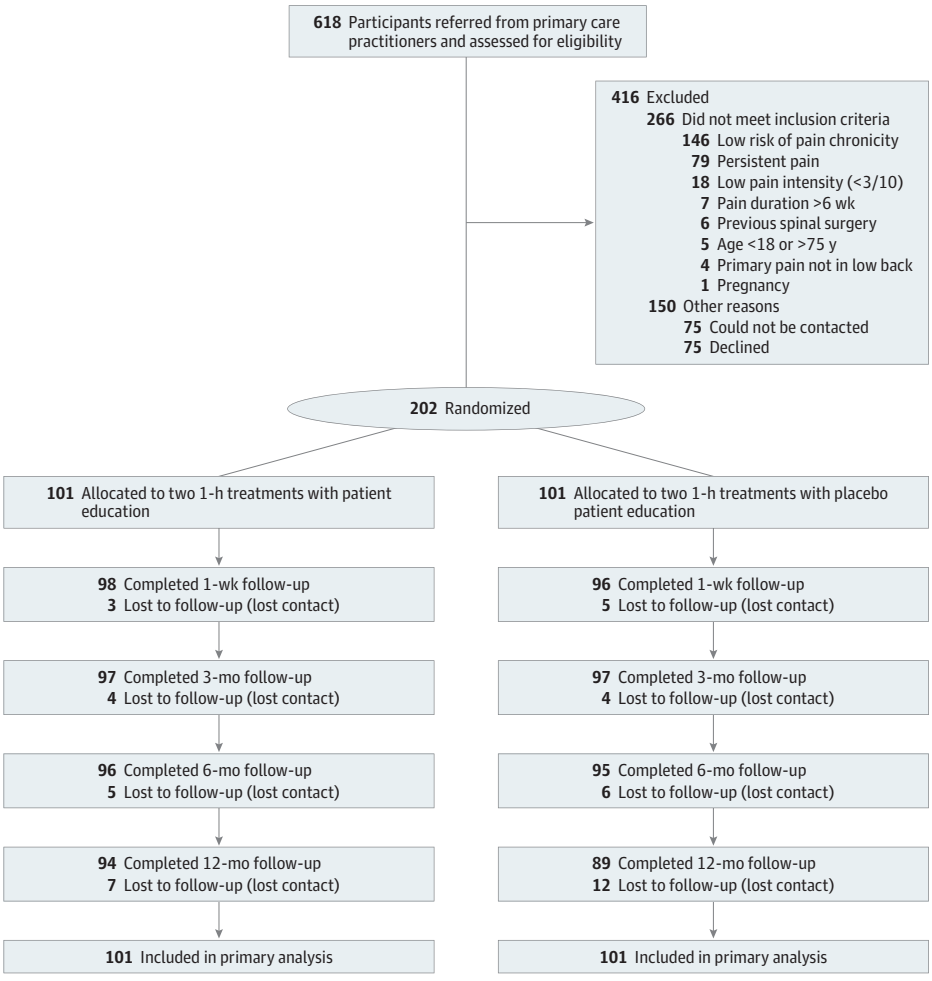


Table 1. Baseline Characteristics[a]

| Characteristic | Patient Education (n = 101) | Placebo Patient Education (n = 101) |
|---|---|---|
| Age, mean (SD), y | 46.5 (14.7) | 43.8 (14.1) |
| Female sex | 53 (52.5) | 50 (49.5) |
| Clinical characteristic | | |
| Pain duration, mean (SD), d | 12.5 (7.7) | 13.5 (8.7) |
| No. of previous episodes, median (IQR) | 3 (5) | 3 (7) |
| No. of other pain sites, mean (SD) | 1.0 (1.2) | 1.1 (1.3) |
| Referred by general practitioner | 19 (18.8) | 16 (15.8) |
| Referred by physiotherapist | 82 (81.2) | 85 (84.2) |
| First episode of back pain | 21 (20.8) | 18 (17.8) |
| Pain referred to leg | 47 (46.5) | 57 (56.4) |
| Pain in areas other than back or leg | 57 (56.4) | 55 (54.5) |
| Work absence or reduced hours | 22 (21.8) | 31 (30.7) |
| Receiving pain medication | 50 (49.5) | 54 (53.5) |
| Outcome scores at baseline | | |
| Pain intensity, mean (SD)[b] | | |
| Week | 6.3 (2.4) | 6.1 (2.2) |
| Current | 4.0 (2.2) | 4.0 (2.3) |
| Pain interference, mean (SD)[c] | 6.0 (2.5) | 6.4 (2.6) |
| Disability, mean (SD)[d] | 11.0 (5.4) | 11.7 (5.8) |
| Depressive symptoms, mean (SD)[e] | 4.1 (3.7) | 5.1 (5.0) |
| Reassurance | | |
| Nothing seriously wrong, mean (SD)[f] | 5.6 (2.7) | 5.4 (2.7) |
| Yes, perceive a need for further tests | 51 (50.5) | 55 (54.5) |
| Process measures at baseline, mean (SD) | | |
| Neuroscience knowledge[g] | 6.0 (1.8) | 5.9 (1.6) |
| Pain attitudes: pain is sign of damage[h] | 2.3 (1.2) | 2.5 (1.1) |
| Pain self-efficacy[i] | 35.5 (13.1) | 33.1 (13.0) |
| Catastrophizing[j] | 18.3 (12.0) | 19.9 (11.2) |
| Back beliefs[k] | 27.7 (6.8) | 28.3 (6.4) |

Abbreviation: IQR, interquartile range.

[a] Data are presented as number (percentage) of patients unless otherwise indicated.

[b] Numeric rating scale with range from 0 (no pain) to 10 (worst pain possible).

[c] Numeric rating scale with range from 0 (no interference) to 10 (highest interference possible).

[d] Roland Morris Disability Questionnaire with range from 0 (no disability) to 24 (high disability).

[e] Depression severity scale of Depression, Anxiety and Stress Scale with range from 0 (no depressive symptoms) to 42 (high depressive symptoms).

[f] "How reassured do you feel that there is no serious condition causing your back pain?" Range from 0 (not reassured at all) to 10 (completely reassured).

[g] Neurophysiology of Pain Questionnaire with range from 0 (no knowledge) to 19 (highest knowledge).

[h] Survey of Pain Attitudes, question 3 from 1-item version: "The pain I feel is a sign that damage is being done." Range from 0 (very untrue for me) to 4 (very true for me).

[i] Pain Self-Efficacy Questionnaire with range from 0 (low pain self-efficacy) to 60 (high pain self-efficacy).

[j] Pain Catastrophizing Scale with range from 0 (low catastrophizing) to 52 (high catastrophizing).

[k] Back Beliefs Questionnaire with range from 9 (maladaptive or pessimistic beliefs) to 45 (helpful or positive beliefs).

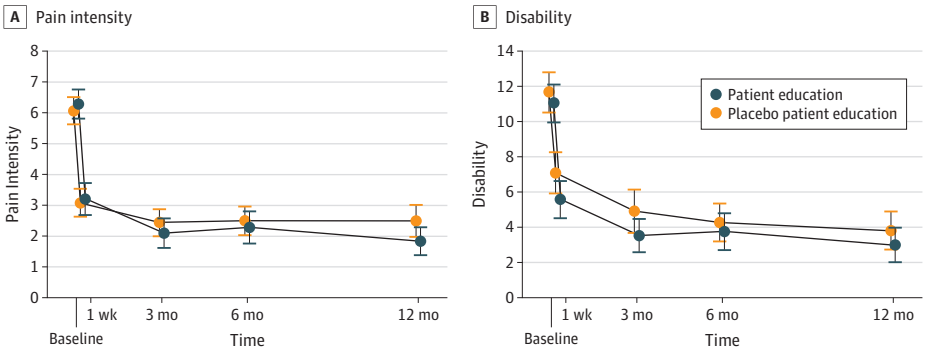
Table 2. Primary Outcomes for the Patient Education and Placebo Patient Education Groups at 1 Week and 3, 6, and 12 Months

| Variable | Point Estimates, Mean (SD) | | Mean Difference (95% CI) | *P* Value |
|---|---|---|---|---|
| | Patient Education | Placebo Patient Education | | |
| Pain intensity during the past week | | | | |
| 1 wk | 3.2 (2.4) | 3.1 (2.2) | 0.1 (−0.5 to 0.8) | .69 |
| 3 mo | 2.1 (2.4) | 2.4 (2.2) | −0.3 (−1.0 to 0.3) | .31 |
| 6 mo | 2.3 (2.6) | 2.5 (2.3) | −0.2 (−0.8 to 0.5) | .59 |
| 12 mo | 1.8 (2.2) | 2.5 (2.4) | −0.6 (−1.3 to 0.1) | .07 |
| Overall intervention effect[a] | NA | NA | NA | .26 |

Abbreviation: NA, not applicable.

[a] *P* value is from mixed models for repeated measures comparing between-group difference during the full 12-month trial, controlling for baseline and including time points as a categorical variable.

Figure 2. Treatment Effects of Intensive Patient Education on Pain and Disability




A, Mean pain intensity score (primary outcome) using a numeric rating scale ranging from 0 (no pain) to 10 (worst pain possible). B, Mean disability outcomes score at 1 week and 3, 6, and 12 months using the Roland Morris Disability Questionnaire ranging from 0 (no disability) to 24 (high disability). Whiskers indicate 95% CIs.

95% CI, -3.1 to -0.1; $P$ = .03) and at 3 months (mean difference, -1.7 points; 95% CI, -3.2 to -0.2; $P$ = .03) (**Table 3**). There were no between-group differences in disability at 6- or 12-month follow-up.

There were some significant between-group differences in secondary outcomes (Table 3). The odds of having a recurrence of low back pain at 12 months were lower in the patient education group than in the placebo patient education group (odds ratio, 0.44; 95% CI, 0.24-0.82). Pain interference and the odds of seeking health care were also lower in the patient education group at 3 months (pain interference: mean difference, -0.8; 95% CI, -1.5 to -0.1; $P$ = .02; health care seeking: odds ratio, 0.43; 95% CI, 0.19-0.93), but results for these variables were not lower at 6 or 12 months. Pain attitudes and reassurance at 1 week were higher in the patient education group (pain attitudes: mean difference, -0.9; 95% CI, -1.2 to -0.5; $P$ < .001; reassurance ["How reassured do you feel that there is no serious condition causing your back pain?"]: mean difference, 1.2; 95% CI, 0.4-2.0; $P$ = .003), and the effect on pain attitudes persisted at 12 months.

Patient education was not more effective than placebo patient education for reducing depressive symptoms, the incidence of chronic low back pain, or global perceived change (Table 3). The causal mediation analysis confirmed that patient education reduced catastrophizing and unhelpful beliefs (primary treatment targets), but these psychologic mechanisms did not reduce pain intensity (full results of mediation analysis reported in eResults 1, eTables 1 and 2, and eFigures 1-3 in Supplement 2). There were no reported adverse events in either treatment group. There was no evidence that out-of-trial therapy confounded treatment effects (eResults 2 and eTable 2 in Supplement 2).

## Discussion

Our study provides evidence that intensive patient education is not effective compared with placebo for patients with acute low back pain. Two 1-hour sessions of patient education were no more effective than a placebo intervention for improving pain at our primary end point of 3 months or at 1 week, 6 months, or 12 months after the onset of acute low back pain. Disability was significantly lower in the intervention group compared with the placebo group at 1 week and 3 months but not at 6 months or 12 months. The short-term effects on disability, although consistent with those from similar trials,[21] were below published guidance on clinically meaningful effects (2 points on a 24-point Roland Morris Disability Questionnaire and 1 point on a 10-point numeric rating scale).[22] Our results suggest that offering more intensive patient education to patients with acute low back pain than that provided as part of standard practice does not reduce pain intensity or lead to meaningful reductions in disability.

Our results challenge a widespread belief that patient education is an effective strategy for treatment of acute low back pain. For example, every clinical guideline recommends patient education to manage acute low back pain.[13] These recommendations are, however, often unaccompanied by an evidence statement (eg, neither US[23] nor UK[22] guidelines cite evidence for patient education) or instruction on how patient education interventions should be conducted.[24] Two systematic reviews have concluded that primary care-based patient education is effective for acute low back pain.[12,25] The available Cochrane review[12] of individual patient education included 6 trials of patient education compared with usual care: 3 trials of brief interventions (<20 minutes) and 3 trials of intensive interventions (>2 hours). The authors concluded that intensive patient education may be more effective at increasing return-to-work rates compared with usual care based on 2 trials (n = 1432). However, those trials did not include pain or disability outcomes. Although a more recent review of 14 trials found that brief patient education could reduce back pain-related distress (n = 4872),[25] it was unclear whether these interventions could improve other clinical outcomes such as pain.[26] Of importance, our mediation analysis (eResults 1 in Supplement 2) suggests that interventions aimed at reducing pain-related distress (eg, catastrophization) are unlikely to influence the pain experience as much as previously thought.

### Strengths and Limitations

This trial[15] had several strengths. It was the first trial, to our knowledge, to test a patient education intervention against a credible placebo (ie, a professional consultation without any information or advice) in patients with acute low back pain.

Table 3. Secondary Outcomes for the Patient Education and Placebo Patient Education Groups at 1 Week and 3, 6, and 12 Months[a]

| Variable | Patient Education | Placebo Patient Education | Effect Measure Mean Difference or OR (95% CI) | *P* Value |
|---|---|---|---|---|
| Chronic low back pain at 3 mo, No./total No. (%)[b] | 33/96 (34.4) | 42/93 (45.1) | 0.63 (0.32 to 1.14) | .13 |
| Disability[c] | | | | |
| 1 wk | 5.6 (5.2) | 7.1 (5.8) | −1.6 (−3.1 to −0.1) | .03 |
| 3 mo | 3.5 (4.6) | 4.9 (6.0) | −1.7 (−3.2 to −0.2) | .03 |
| 6 mo | 3.8 (5.2) | 4.3 (5.2) | −0.8 (−2.4 to 0.7) | .28 |
| 12 mo | 3.0 (4.7) | 3.8 (5.1) | −0.8 (−2.4 to 0.7) | .29 |
| Overall intervention effect[d] | NA | NA | NA | .17 |
| Pain interference[e] | | | | |
| 1 wk | 2.8 (2.7) | 2.9 (2.5) | −0.1 (−0.8 to 0.6) | .71 |
| 3 mo | 1.5 (2.1) | 2.3 (2.4) | −0.8 (−1.5 to −0.1) | .02 |
| 6 mo | 1.8 (2.6) | 1.9 (2.3) | −0.1 (−0.8 to 0.6) | .87 |
| 12 mo | 1.6 (2.4) | 2.0 (2.5) | −0.4 (−1.1 to 0.3) | .30 |
| Overall intervention effect[d] | NA | NA | NA | .16 |
| Depressive symptoms[f] | | | | |
| 1 wk | 2.6 (4.1) | 3.3 (4.3) | −0.7 (−1.8 to 0.5) | .26 |
| 3 mo | 2.1 (3.9) | 2.5 (4.1) | −0.5 (−1.7 to 0.6) | .36 |
| Overall intervention effect[d] | NA | NA | NA | .89 |
| Current pain intensity[g] | | | | |
| 1 wk | 2.3 (2.1) | 2.2 (2.1) | 0.1 (−0.5 to 0.7) | .69 |
| 3 mo | 1.5 (2.0) | 2.1 (2.1) | −0.6 (−1.2 to −0) | .04 |
| 6 mo | 1.8 (2.5) | 1.8 (1.9) | −0.1 (−0.7 to 0.5) | .78 |
| 12 mo | 1.4 (2.1) | 1.7 (2.1) | −0.3 (−0.9 to 0.3) | .33 |
| Overall intervention effect[d] | NA | NA | NA | .13 |
| Seeking health care for low back pain, No./total No. (%) | | | | |
| 3 mo | 73/96 (76.0) | 82/93 (88.2) | 0.43 (0.19 to 0.93) | .03 |
| 6 mo | 44/95 (46.3) | 48/91 (52.7) | 0.77 (0.43 to 1.38) | .38 |
| 12 mo | 32/91 (35.2) | 38/87 (43.7) | 0.70 (0.38 to 1.28) | .25 |
| Global change at 3 mo[h] | 8.1 (1.7) | 7.8 (2.0) | −0.3 (−0.9 to 0.2) | .11 |
| Recurrence at 12 mo, No./total No. (%)[i] | 26/91 (28.6) | 41/87 (47.1) | 0.44 (0.24 to 0.82) | .01 |
| Pain attitudes | | | | |
| 1 wk | 1.3 (1.2) | 2.2 (1.3) | −0.9 (−1.2 to −0.5) | <.001 |
| 12 mo | 1.2 (1.2) | 1.6 (1.3) | −0.4 (−0.7 to 0) | .03 |
| Overall intervention effect[d] | NA | NA | NA | .16 |
| Nothing seriously wrong (0-10) at 1 wk[j] | 7.6 (2.5) | 6.5 (2.9) | 1.2 (0.4 to 2.0) | .003 |
| Yes, perceive a need for further tests at 1 wk, No./total No. (%) | 25/98 (25.5) | 36/96 (37.5) | 0.57 (0.31 to 1.05) | .07 |

Abbreviations: NA, not applicable; OR, odds ratio.

[a] Data are presented as mean (SD) unless otherwise indicated.

[b] Reporting 2 or more on an 11-point pain intensity numeric rating scale during the past week and no periods of recovery at that time.

[c] Roland Morris Disability Questionnaire with range from 0 (no disability) to 24 (high disability).

[d] *P* value is from mixed models for repeated measures comparing between-group difference during the full 12-month trial, controlling for baseline and including time points as a categorical variable.

[e] Numeric rating scale with range from 0 (no interference) to 10 (highest interference possible).

[f] Depression severity scale of Depression, Anxiety, and Stress Scale with range from 0 (no depressive symptoms) to 42 (high depressive symptoms).

[g] Numeric rating scale with range from 0 (no pain) to 10 (worst pain possible).

[h] Global Back Recovery Scale.

[i] Recurrence was defined as answering yes to both of the following questions: (1) "In the last 6 months/12 months, has your lower back pain gone away completely for a period of more than 30 days, only to return later on?" and (2) "If yes, did the return of low back pain last at least 24 hours with a pain intensity of more than 2/10?"

[j] "How reassured do you feel that there is no serious condition causing your back pain?" Range from 0 (not reassured at all) to 10 (completely reassured).

This strategy allowed us to determine the specific effects of patient education and control for effects produced by a clinical encounter, for example, those from the attention of a health professional or from the credibility of an impending treatment. We trained 2 trial clinicians to ensure treatment fidelity. Retention rates were high (>90% at all time points). We followed a published trial protocol[15] and statistical analysis plan.[18] Data were collected and analyzed by researchers who were masked to group allocation.

We used PICKUP, a validated prognosis model,[8] to exclude people with acute low back pain who were at lower than average risk of pain chronicity. Approximately 40% of included participants developed chronic low back pain, a rate twice that of other trials on acute low back pain conducted in the same geographical area of Sydney (approximately 15%-20%).[9,27] We are therefore confident that we included participants who were at high risk of pain chronicity.

This study also has limitations. First, trial clinicians could not be blinded to treatment allocation. However, results of our audit suggested that there were no systematic differences in treatment credibility or treatment fidelity. Second, interventions in the PREVENT trial[15] were provided by trial physiotherapists, and it is unclear whether our results would have been the same if the participant's health practitioner provided the intervention. Third, we performed a number of statistical comparisons, which although planned, increased the

risk of Type I error. Interpretation of the statistically significant effects of intensive patient education on some secondary outcomes, such as pain interference and recurrence and odds of seeking health care (Table 3), must consider this potential limitation. Finally, because both groups received basic patient education as part of recommended first-line care and many recovered despite being classified as being high risk, the potential for between-group differences may have been reduced.

## Conclusions

For patients with acute low back pain who received first-line care, intensive patient education was no more effective than a placebo intervention. Adding complex, time-consuming treatments to primary-care based advice and reassurance is likely to be unnecessary for most patients with acute low back pain.


### ARTICLE INFORMATION

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**OVERVIEW OF THE PROJECT**

Chronic low back pain is a massive health problem in Australia. The evidence is consistent that treatments for chronic back pain have only modest effects. Attempts to prevent chronic low back pain have focused on biomechanics, fear avoidance, work and social-related factors or activity. These approaches are not successful for many people.

We are taking an alternative approach and focusing on two factors that are fundamental determinants of pain, but have hitherto not been considered as potential targets for preventative intervention. The first factor is the *meaning* that an individual attaches to their pain as the meaning of noxious input ultimately determines whether or not it will be painful. Pain does not depend on the true danger to tissues, but on the brain's evaluation of that danger. The second factor is *mood*. Pain, unlike purely sensory perceptions has an affective component. It is this affect that gives pain such a strong survival value and mood cannot be separated from pain. There are very well-established biological pathways by which meaning and mood can upregulate the nociceptive system, leading to increased sensitivity of nociceptive and pain systems and, consequently, chronic pain.

Remarkably, very few attempts have been made to reduce the risk of chronicity by targeting the fundamental determinants of pain, meaning & mood, directly.

This project brings together international experts in several fields and represents the final stage of a decade of clinical and fundamental research. We have identified the factors associated with poor prognosis. We have thoroughly tested and refined a deceptively simple, easily implemented, and inexpensive intervention that targets these factors. We are able to identify the patients who are at high risk for developing chronic low back pain and for whom our novel treatment is ideally suited.

We are now ready to undertake the final stage of this work, the definitive prospective randomised placebo-controlled trial to evaluate if our intervention reduces the proportion of high-risk individuals who develop chronic back low pain.

## 1. PROJECT PRIMARY AIMS AND HYPOTHESIS

The aim of this project is to:

- Establish whether our novel psychoeducative intervention, *Explain Pain*, reduces the development of chronic low back in high-risk individuals.

We hypothesize that:

- The addition of *Explain Pain* to NHMRC guideline-based care for acute low back pain will reduce the proportion of patients who have persistent low back pain at 3 months.

## 2. BACKGROUND

**The problem of chronic low back pain.**

Low back pain is very common[1 2] but not everyone who gets low back pain will develop *chronic* low back pain. In fact, most do not[3]. In the largest ever study of its kind we showed that about 60% of people who have low back pain recover in a few weeks[4], often with minimal intervention[5]. However for the other 40% recovery is slow and the risk of persistent problems is very high (Figure 1). It is this 40% who incur most of the enormous costs associated with low back pain[6 7]. In Australia these patients represent a drain on the economy that is equivalent to building 120 new

general hospitals each year[8]. Any approach that reduces the incidence of chronic low back pain is likely to have a major national impact.

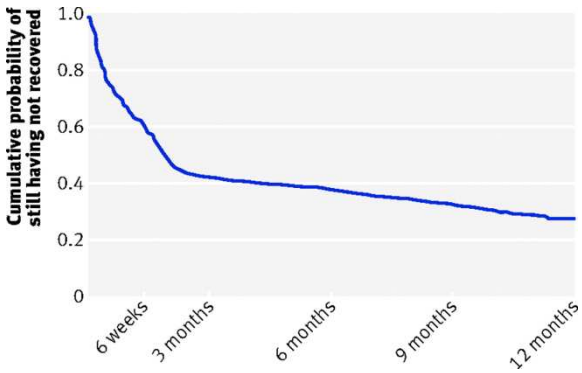


*Figure 1: This graph shows that recovery is rapid in the first few weeks and months after an initial episode of low back pain and that it slows down markedly after 3 months, once chronic low back pain develops. (Henschke et al, 2008 BMJ[4]).*

Our work, and that of others, has consistently shown that treatments for patients with chronic low back pain are seldom effective in returning them to a pain-free or productive life[9 10-12]. These people face a downward spiral of increasingly lengthy periods of severe pain and chronic disability with substantial social and personal disadvantage[2].

**We are proposing that, rather than waiting to treat patients who already have chronic low back pain, much better outcomes are likely to be achieved if we intervene early to reduce the risk of developing chronic low back pain after an acute episode**.

This proposal is both logical and aligned with the NHMRC's Preventative Health Care priority goal of the National Health Priority - Promoting and Maintaining Good Health.

**<u>Biological plausibility of our approach: Changing the meaning of pain and mood of the patient will reduce chronicity</u>**

Pain does not equate to tissue damage, nor does it equate to activity in nociceptors. We have known this for decades – Patrick Wall stated in 1986 that "the mislabelling of nociceptors as pain fibres was not an elegant simplification but an unfortunate trivialization"[13]. That multiple cognitive and contextual factors modulate pain is well established and the mantra that 'nociception is neither sufficient nor necessary for pain' is well accepted in the fundamental pain sciences[14 15]. It is also well established that the *meaning* of one's pain determines descending modulatory control of spinal nociceptors – the stronger one's pain is conceptualised as reflecting tissue damage, the more likely is descending facilitation of spinal nociceptors[16] – and sustained upregulation of spinal nociceptors is a key determinant of central sensitivity and chronic pain[17]. Thus, there is a direct neurological pathway by which the *meaning* of pain to the patient modulates the risk of chronic low back pain.

Evaluating a patient's *mood* is an important part of clinical triage[21] as is the notion that mood affects recovery. Recently, direct biological pathways by which *mood* can modulate chronicity have also been uncovered. Depression is associated with increased expression of pro-inflammatory cytokines, decreased expression of anti-inflammatory cytokines[18], and disruption of the HPA axis (see CIC Moseley – Explain Pain[15] & Blackburn-Munro (2007)[19]. All of these mechanisms upregulate spinal nociceptors and cortical networks implicated in chronic pain[20]. Thus, there is a direct neurological pathway by which *mood* modulates the risk of chronic low back pain.

Indirect pathways by which *meaning & mood* are likely to modulate the risk of chronicity are well recognised clinically – for example the strong belief that pain means damage, and the more one is depressed, the less likely one is to adopt behavioural strategies that promote recovery, for example

return to normal activity and engagement in social and work activities. While we endorse the validity of these indirect pathways, we contend that the direct pathways are more obvious and proximal targets of intervention.

## THIS PROJECT AS THE CULMINATION OF A WIDER RESEARCH PROGRAMME

The four hallmarks of a successful preventative intervention are to (i) identify the factors that are associated with the development chronic low back pain (ii) develop interventions that treat these factors (iii) identify, at an early stage, patients who are at high-risk of developing chronic low back pain (iv) determine whether treating high-risk patients early with the novel intervention decreases the risk of chronicity. We have achieved the first three objectives. This proposal is to fund the final definite stage of our work.

### (i) We have identified the factors that are associated with the development of chronic low back pain

Over the last decade, we have undertaken a series of major prognostic studies that have led to the identification of key variables associated with an increased risk of developing chronic low back pain after an acute episode[3 4 21 22]. Together these variables reflect the *meaning* of one's back pain to that individual and the *mood* of that individual. The major variables are: expectations of persistence, reductions in usual activities and symptoms of depression[4]. Patients at high-risk for chronicity have strong beliefs that they will not recover, that their pain is going to get worse (catastrophising) and that having pain means they should stop what they are doing until the pain goes away[23]. They also score highly on measures of depression. Recent systematic reviews that incorporate data from international cohorts have confirmed our findings[2 24]. International guidelines for the management of low back pain[25] and those working at the coalface, clinicians &injury managers[26], have reached similar conclusions - the influence of variables that reflect *meaning & mood* play a critical role on the development of chronic low back pain.

*KEY POINT:* Variables that reflect *meaning & mood*, are associated with the development of chronic low back pain.

### (ii) We have developed a simple, easy to implement and inexpensive intervention to treat the factors associated with the development of chronic low back pain

The proposed project represents the final stage of over a decade of research into Explaining pain[15]. There is now a large amount of research that shows that carefully explaining to someone the biology that underpins pain changes the *meaning* of their pain. For example, explaining pain changes pain-related attitudes and beliefs, in particular it decreases the conviction that pain is an accurate indication of tissue damage and increases the conviction that pain is modulated by one's thoughts and beliefs. Explaining pain decreases pain-related catastrophising in people with chronic or subacute pain and in pain-free individuals[27-30]. A blinded randomized experiment showed that explaining pain increases pain threshold during a straight leg raise and explaining lumbar spine physiology and anatomy decreases pain threshold during a straight leg raise[31]. Explaining pain has also been shown to decrease pain and disability in people with chronic pain[32]. These findings have now been replicated in other languages and distinct chronic pain groups[29], and are supported by systematic reviews[33].

We have also completed a final pilot study. We predicted that by first shifting the meaning of pain via Explain Pain, the effects of a multidisciplinary programme that targets the indirect effects of meaning and mood on physical, social and work activity, would be enhanced. Chronic pain patients (n=104) were randomly allocated to Explain Pain or to best practice behavioural advice, based on

The Back Book[34], prior to participation in an intensive, cognitive-behavioural therapy based, pain management programme. Six months later, those who had undertaken Explain Pain before their programme, were doing better than those who had not: the odds ratio (OR) for a clinically meaningful reduction in pain was 3 (95% CI = 2 – 9). For disability, the OR was 9.5 (3 – 36). For a positive shift in work status, OR = 6 (2 – 22). That is, our hypothesis was soundly supported.

*KEY POINT:* Explaining pain modifies *meaning & mood*, leading to clinically relevant changes.

**(iii) We can identify the patients who are at high risk of developing chronic low back pain**
Treating all patients with acute low back pain to prevent them developing chronic low back pain is clearly inefficient as 60% will recover within a few weeks with minimal intervention[4]. Additional interventions are better targeted to those at high risk[35]. We aim to treat those patients who are at *high-risk* of developing chronic low back pain[36].

Our systematic review[37] of the Orebro Musculoskeletal Pain Screening Questionnaire (OMPSQ)[38] identified that it is suited for this purpose. A cut-off score of 120 on this questionnaire identifies 92% of those who will recover before three months and 75% who won't (Table 1). These patients were 4 times more likely to have chronic low back pain[39]. We have recently developed a short-form of this questionnaire which our testing indicates has similar properties to the long form[40].

| Cut-off score | Recovered at 3 months (specificity %) | Not recovered at 3 months (sensitivity %) |
|---|---|---|
| 105 | 82 | 46 |
| 110 | 84 | 43 |
| 120 | 92 | 25 |

*Table 1 showing that scores on the OMPSQ under 120 are likely to identify almost all of the patients who recover and 75% of patients who don't recover (Linton and Boersma, 1997[38]).*

Including patients with OMPSQ > 120 in our study will include only a few patients who are likely to recover early (<10%) and we will include almost 75% of those who are likely to develop chronic low back pain.

*KEY POINT:* The OMPSQ allow us to target moderate and high-risk patients and exclude nearly all who would normally go on to recover in a weeks with minimal intervention.

**(iv) We have pilot tested our approach and found promising results.**
The final step before we can definitely test our treatment is to undertake pilot work that demonstrates its feasibility in a clinical setting, and gives some projection of the likelihood that our hypothesis will be supported. We have now completed that step[41]. An initial consecutive cohort of 74 patients with occupational injuries participated and cost-of-injury data show that the OMPSQ successfully predicted poor outcomes. In a second consecutive cohort of 78 patients with occupational injuries, high-risk patients were treated early according to our conceptual model, and the costs of their management were reduced by 25%, principally via an earlier return-to-work. It is notable that savings were achieved despite the additional cost of intervention. This pilot study showed that we can identify patients at high risk of chronicity, intervene early and reduce the risk of chronicity.

**Now it is time to fully interrogate our hypothesis using the gold-standard randomised placebo-controlled clinical trial.**

## 3. RESEARCH PLAN, METHOD AND TECHNIQUES

### Overview of the research design

The study will be a randomised controlled trial evaluating the effectiveness of a brief psychoeducative intervention to prevent the development of chronic low back pain in a group of acute low back pain patients who are at risk of developing chronic low back pain.

Patients with acute low back pain attending primary care (GP, physiotherapist or chiropractor) will be assessed for variables reflecting *meaning & mood*. Patients with high levels of these variables will be randomised to receive NHMRC guideline-based care *plus* sham psychoeducational intervention or guideline-based care *plus* an individualised psychoeducational intervention designed to address the *meaning & mood*. Outcomes will be assessed at 3, 6 and 12 months.

### Patients

We will recruit primary care practitioners using our successful recruitment strategies[4 5 42]. The primary care practitioners will identify consecutive patients with low back pain and provide their contact details to the study researchers. The study researchers will apply the study inclusion/exclusion criteria and consent 250 acute low back pain patients to the study.

**Inclusion criteria:** Patients will be included if they meet all of the following criteria:

- The primary complaint of pain is in the area between the 12$^{th}$ rib and buttock crease. This may, or may not, be accompanied by leg pain.
- A new episode of low back pain, preceded by $\geq$ one month without low back pain[43].
- The duration of current symptoms is less than 4 weeks.
- An OMPSQ score greater than 120.
- Sufficient fluency in English language to understand and respond to English language questionnaires and to engage with the psychoeducative intervention.

**Exclusion criteria:** Patients will be excluded if they have any of the following conditions:

- Known or suspected serious spinal pathology, nerve root compromise, previous spinal surgery[44].
- Currently receiving care for a mental health condition.

### Randomisation

A researcher not involved in patient recruitment or data collection will create a randomisation schedule using randomisation software. The schedule will be in randomly permuted blocks stratified for Work Cover/compensation claim. The schedule will be used to create 250 consecutively numbered, sealed, opaque envelopes containing allocations.

### Procedure

During the consultation the primary care practitioner will contact the study researcher by telephone or email to provide the patient contact details. The study researcher will contact the patient by telephone within 24 hours of the first consultation to conduct the screening, consent and baseline assessments. Once the study researcher has obtained baseline data the patient will be randomised to receive NHMRC guideline care *plus* sham psychoeducative intervention or NHMRC guideline care *plus* the psychoeducative intervention.

All participants will be reminded to continue with the care provided by their primary care clinician for their low back pain. The study researcher will organise an initial appointment with the specially trained clinician to receive either the sham or active psychoeducative intervention.

**NHMRC Guideline care**

All patients will receive NHMRC guideline care. Participating general practitioners, physiotherapists and chiropractors will be trained in the delivery of guideline care based on the NHMRC guideline for recent onset low back pain[45]. The guideline recommends a first-line of care consisting of advice, reassurance and analgesics. Participants will be reassured of the benign nature of low back pain, advised to remain active and avoid bed rest, and instructed in the use of simple analgesics to manage their symptoms. The practitioner may consider second line options such as spinal manipulation if the patient does not respond to first-line care.

**The psychoeducation program – *Explain Pain***

Patients randomised to the psychoeducative intervention will participate in 2 sessions of *Explain Pain* by the specially trained clinician. Our pilot study showed that 2 x 1-hour sessions is sufficient to change the meaning of pain and improve mood. All treatments associated with the intervention will be completed within 2 weeks of randomisation.

*Explain Pain* involves a collaborative clinician-patient interaction. The clinician determines key conceptual frameworks via a recognised questionnaire and targeted interview. The intervention has been refined on the basis of numerous clinical and experimental studies and is informed by current theory in health literacy, conceptual change and educational design. It follows this broad plan: (i) introduction of key concepts identified in assessment and interview, (ii) explanation of key concepts in biological terms, (iii) evaluation and embedding of key concepts. We have recently shown that metaphors and stories provide the best way to introduce key concepts[46]. Metaphors provide visualisation of abstract ideas and their abstraction from the targeted concept reduces cognitive resistance to the same. Thus, metaphors are thought to provoke contemplation and increase the potential for re-organisation of previous meanings.

The most common key concepts are: nociceptive input is modulated at the spinal cord and the brain; the brain evaluates many inputs before selecting a response; pain is the conscious part of the response; the brain modulates the nociceptive signal at the spinal cord. Emphasis is placed on the distinction between pain and nociception, on the biological necessity of multiple influences over pain, on the plasticity of the spinal cord and brain and the importance of neural changes in chronic pain. Explaining pain has strong theoretical support in conceptual change theory, which stipulates that conceptual change requires deep and superficial learning. Deep learning is information that is retained and understood and applied to problems at hand[47] and 'superficial' or 'surface' learning is information which is remembered but not understood or integrated with attitudes and beliefs[48]. Explaining pain takes about two hours. Two sessions will be devoted to explaining pain. Reconceptualisation will be evaluated using established questionnaires.

**The sham psychoeducation intervention**

Patients randomised to the sham psychoeducative intervention will receive 2 x 1 hour sessions of sham psychoeducative education, based on sham advice sessions reported in our previous study[49]. Patients will be given the opportunity to discuss their low back pain and any other problems that they may have. The clinician will respond in an empathetic way, but will not offer any advice or information on pain or their condition. We have previously shown patients find sham advice/education to be credible[49].

**Sample size calculations.**
We calculated sample size using the method of Twisk[50] for mixed models. With 2 repeated observations, an estimated intra-cluster correlation (correlation between the observations) of 0.4, alpha set at 5%, and allowing for 15% loss to follow up, we require 125 patients in each group to have an 80% power to detect a relative reduction in risk (i.e., in incidence proportion) of having low back pain at 3 months of 15%. This implies a number needed to treat (NNT) of 10. We consider these to be the smallest effects that would justify implementation of the intervention. In these calculations we have conservatively ignored the increase in statistical power conferred by baseline covariates and stratification.

**Feasibility**
We have been very successful in recruiting primary care practitioners for several similar trials[4 5 42 49]. We have developed strong links with local clinicians and have a network of practitioners who have expressed interest in participation in future trials. We have designed the trial to minimise the workload on practitioners and interference with normal clinical practice, which is in our experience essential in maintaining practitioners' involvement.

Our previous experience suggests that a primary care practitioner will refer approximately 2 acute low back pain patients for our trial each month. Our pilot study with injured workers suggests that 20% of these patients will be eligible for the trial. We will recruit 50 primary care practitioners who we anticipate will recruit on average 7 acute low back pain patients each over 18-24 months. This will be sufficient to reach our target of 250 patients. In a previous study[4] we recruited 1,600 acute low back pain patients from primary care practitioners over a 24-month period so we believe that our target recruitment of 250 patients can be easily achieved within 24 months.

**Outcomes**
a) The ***primary outcome*** will be the risk (incidence proportion) of having low back pain at 3 months. The 3-month follow up was chosen as the primary outcome as this is the most common definition of chronic low back pain[43 51] and reflects the time when a clear change in prognosis occurs (see figure 1[4])

Low back pain will be determined by numerical pain rating scale (NRS) score of pain intensity > 0, taken from the Chronic Pain Grade[52], a widely used composite measure of pain intensity and disability that provides a method for quantifying the severity of chronic symptoms.

b) The ***secondary outcomes*** will include a condition-specific measure of disability (Roland Morris Disability Questionnaire[53] (RMDQ), 0-24 scale), a patient-generated measure of function (Patient-Specific Functional Scale[54], 0-10 scale) and the OMPSQ[38] (to determine if *meaning & mood* have changed). Each will be assessed at 3, 6 and 12 months. We will also take a measure of recurrence at 12 months[55],

**Data and treatment integrity**
Trial data integrity will be monitored by regularly scrutinising data files for omissions and errors. All data will be double entered and the source of any inconsistencies will be explored and resolved. Treatment adherence will be determined by recording attendance at treatment sessions and by analysing participant activity diaries.

**Statistical Analysis**
The data will be analysed by intention-to-treat and by a statistician blinded to group allocation. We will analyse the effect of treatment separately for each outcome using linear mixed models with

random intercepts for individuals to account for correlation of repeated measures. The model will include terms for important prognostic factors measured prior to randomisation and specified a priori. As we stratified by workers compensation status in the allocation schedule the analysis will be stratified by this variable. We will obtain estimates of the effect of the intervention and 95% confidence intervals by constructing linear contrasts to compare the adjusted difference in proportions (dichotomous variables) or mean change (continuous variables) in outcome from baseline to each time point between the treatment and control groups.

**Justification of study design**

The sham-controlled trial includes key methodological features recognised as minimising bias (e.g. patient/clinician/outcome assessor blinding, concealed allocation, and intention to treat analysis). We will prospectively register the trial and publish the full trial protocol in an open-access journal. The trial report will conform to the extension of the CONSORT statement for non-pharmacological trials.

**Evidence that project will be successfully completed on time**

Our pilot work and a recent Australian study of patients acute low back pain suggests that 20% of patients will score OMSPQ > 120 and be appropriate for our study[56]. That means that we need to screen 1090 to recruit 250 patients to the study (table 1[38 39]). This is well within our capacity as we have screened recently recruited over 3000 patients with low back pain and recruited 1600 with acute low back pain in the same geographical area of Sydney that this study will be based. We have the relationships and systems in place in metropolitan Sydney to ensure recruitment and clinician engagement.

The team has a demonstrated track record of leading and managing large trials such as this to completion. The rigour of our work is reflected in where they have been published – *The Lancet*, *Annals of Internal Medicine, BMJ, Neurology* and *Pain*. Our team has the content expert in Explain Pain (CIC Moseley), a recognised world expert on psychological intervention for pain disorders (CI Nicholas).

**OUTCOMES & SIGNIFICANCE**

Given the cost of low back pain, both financial and personal, any reduction in the proportion of patients developing chronic low back pain is likely to be of major significance to Australian and international communities. This study will provide a definitive evaluation of the efficacy of an extremely promising new treatment designed to prevent chronic low back pain. If found to be favourable, these results will fundamentally change the way acute low back pain is managed in primary care.

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**Summary of changes from original to published study protocol**

- Added Markus Huebscher, Adrian Traeger, Hopin Lee, and Ian Skinner to list of investigators
- Include referring practitioner's rooms as study treatment locations
- Add inclusion criterion of pain intensity ≥3/10 on numeric rating scale (NRS) during the past week
- Use locally developed and validated prognostic model (PICKUP), instead of Orebro Musculoskeletal Pain Questionnaire, with score of >2.3 cutoff for inclusion (equivalent to >30% absolute risk of developing chronic low back pain)
- Add exclusion criterion of chronic spinal pain
- Specify that both study intervention sessions must occur within 2 weeks of initial presentation
- Primary outcome changed from dichotomous pain intensity scale (>=2/10 NRS, y/n) at 3 months to continuous pain intensity scale (0-10) at 3 months; sample size revised down from n=250 to n=202.
- Added all secondary outcomes & process measures listed in published protocol except for Roland Morris Disability Questionnaire.

**Summary of changes from original to published statistical/mediation analysis plans**

- Sample size calculation revised from detecting a relative risk reduction of having >=2/10 pain intensity scale at 3 months, to detecting a 1-point difference on a continuous pain intensity scale at 3 months.
- Prognostic factors not to be included in primary analysis
- Randomisation not to be stratified by worker's compensation status (because this factor was part of the risk screening algorithm which determined inclusion)
- Inclusion of a mechanism analysis (mediation analysis – see file number 6 in this Supplement for full protocol)

# Supplementary Online Content

Traeger AC, Lee H, Hübscher M, et al. Effect of intensive patient education vs placebo patient education on outcomes in patients with acute low back pain: a randomized clinical trial. JAMA Neurol. Published online November 5, 2018. doi:10.1001/jamaneurol.2018.3376



This supplementary material has been provided by the authors to give readers additional information about their work.

## **eMethods 1.** Screening With PICKUP Tool

**Excluding patients at low risk of pain chronicity using PICKUP Tool**

To identify those at low-risk of poor outcome, we screened all potential participants using a validated 5-question prognostic screening tool, PICKUP.(1) The questions included: 1. "How much low back pain have you had during the past week?" 1 = none, 2 = very mild, 3 = mild, 4 = moderate, 5 = severe, 6 = very severe; 2. "Do you have leg pain?" 0 = no, 1 = yes; 3. "Is your back pain compensable, e.g., through worker's compensation or third party insurance?" 0 = no, 1 = yes; 4. "How much have you been bothered by feeling depressed in the past week (0–10 scale)?" 0 = not at all, 10 = extremely; 5. "In your view, how large is the risk that your current pain may become persistent (0–10 scale)?" 0 = none, 10 = extreme. Scores on these 5 questions were converted into an absolute risk for developing chronic low back pain. Risk for developing chronic LBP in acute low back pain trials from a similar geographic area of Sydney was 20%.(2, 3) By using PICKUP and applying a cutoff of <=30% predicted risk in our validation sample we estimated that we would exclude from the PREVENT Trial approximately 60% of the patients with acute low back pain who were less likely to develop chronic LBP. That is, we aimed to include double the number of 'high-risk' participants in our sample compared to an unscreened trial population. Data on PICKUP questions were collected prior to obtaining informed consent.

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**eMethods 2.** Standard History and Physical Examination Form

PREVENT

Patient Name:_____ Recording ☐ 1st Session
Date:_____

PT/ GP_____ 2nd Session
Date:_____

Work_____ Date of injury/ pain onset_____ Days since injury_____

Brief overview:

_____

_____

_____

_____

_____

_____

_____

_____

What the physio/ GP has told you:

_____

_____

Patients Understanding of why painful?

_____

_____

Treatment to date:

_____

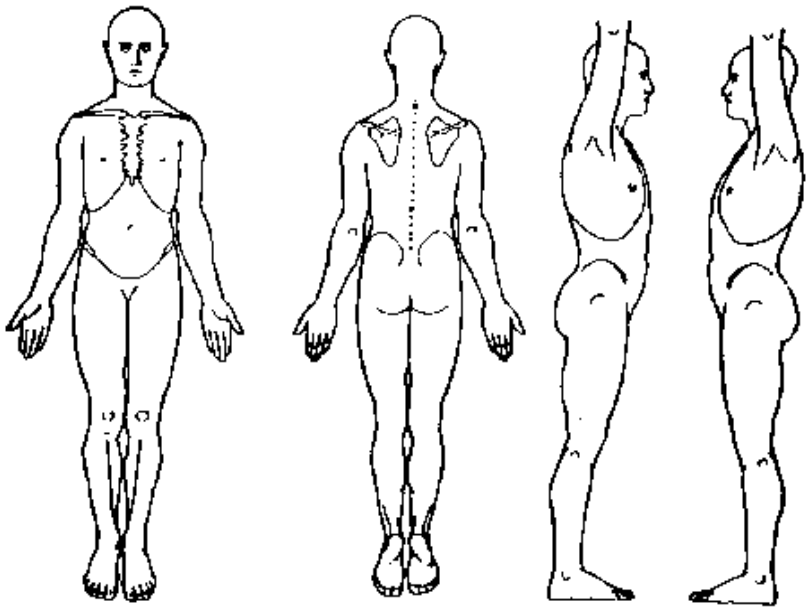
_____

Pain location/ description: Leg Pain Yes ☐ No ☐

_____

_____

_____



Active Movements:

| | Range/ Pain/ Comments |
|---|---|
| Flex | |
| Ext: | |
| R Rot: | |
| L Rot: | |
| R Sflex | |
| L Sflex | |

Neuro Examination

| Level | Movement | R | L | Reflexes | |
|---|---|---|---|---|---|
| L2 | Hip Flex | | | | |
| L3 | Knee Ext | | | | |
| L4 | Ankle DF/ INV | | | Knee Jerk | |
| L5 | 1st Toe Ext | | | | |
| S1 | Ankle PF | | | Ankle Jerk | |

History of Presenting Condition (HPC)
When:/ What doing/ Activity?/ If no incident/ change in activity?/ Progression of Symptoms/ Actions and effect?

Relevant Past History
Previous Episodes?/ Mechanisms?/ Similarities/ Differences with current episode?/ Time to ease?/Achieve full function?/ Treatment and effects?

Pain A:

| | | | | | |
|---|---|---|---|---|---|
| Constant | Yes | No | If yes does it vary? | Yes | No |
| Intensity: | Worst (/10) | | | Best (/10) | |
| Quality | | | | | |
| Depth | | | | | |

Pain B:

| | | | | | |
|---|---|---|---|---|---|
| Constant | Yes | No | If yes does it vary? | Yes | No |
| Intensity: | Worst (/10) | | | Best (/10) | |
| Quality | | | | | |
| Depth | | | | | |

Aggravating Factors
Activity 1

| | |
|---|---|
| Analyse | |
| How long to come on? | |
| What action? | |
| Have to stop? | |
| How long to ease? | |

Activity 2

| | |
|---|---|
| Analyse | |
| How long to come on? | |
| What action? | |
| Have to stop? | |
| How long to ease? | |

Activity 3:

| | |
|---|---|
| Analyse | |
| How long to come on? | |
| What action? | |
| Have to stop? | |
| How long to ease? | |

Other known aggravating factors:
Bending
Lifting
Sitting
Standing
Stairs Up/ Down
Gardening

Easing Factors

Activity 1

| | |
|---|---|
| Analyse | |
| How long to ease? | |
| Relationships? | |
| Therapeutic V Non Provocative? | |

Activity 2

| | |
|---|---|
| Analyse | |
| How long to ease? | |
| Relationships? | |
| Therapeutic V Non Provocative? | |

Activity 3

| | |
|---|---|
| Analyse | |
| How long to ease? | |
| Relationships? | |
| Therapeutic V Non Provocative? | |

Other known easing strategies:
Heat- Hot pack/ shower/ Cold/ Lying/ Sitting

Irritability

| | |
|---|---|
| Severity | |
| Intensity | |
| Time to settle | |

24 Hour Behaviour

| | |
|---|---|
| First thing AM | |
| During the Day | |
| Evening | |
| Sleep | |

Stage of Condition

| | |
|---|---|
| Better | |
| Worse | |
| Same | |

General Health

| | |
|---|---|
| State of General Health | |
| Under doctors care for anything else | |

Medications (ask for steroids/ anticoagulants/ previously used for long period of time)

| Medication Name | For? | Dose |
|---|---|---|
| | | |
| | | |
| | | |
| | | |
| | | |
| | | |
| | | |
| | | |

Tests/ Investigations

**eMethods 3.** PREVENT Trial Patient Education Manual

**Introduction**

- Spiel
- Mechanism of injury
- Neuro examination
- Conversation about worries

**Explaining the diagnosis**

Structural Diagnosis

- Spinal alarm system & non-specific diagnosis
- Disc
- Joint
- SIJ
- Nerve-root
- Muscle

Biomedical diagnosis

- Arthritis/ degeneration
- Spondylolisthesis
- Instability

Other diagnoses

- My back is out
- My pelvis is twisted
- Weak and insecure

**Explaining therapy so far**

Mechanism of physiotherapy treatments

- Manual therapy
- Motor control/stability
- Mackenzie
- Exercise
- Explaining pain

Mechanism of medical treatments

- Tablets
- Injections
- Surgery

Mechanism of alternative treatments

**Explaining pain biology**

What is pain all about?

- What is pain?
- Pain is protective
- Pain is not a measure of tissue damage
- Pain tries to get us out of danger
- Visual metaphors
- Complex output
- Thirst metaphor

How is it processed and what can change it?

- Nociception vs. pain
- Danger to the tissues doesn't = pain
- Pain doesn't always = danger
- Pain processing diagram
- Inflammation
- Tissue healing
- Peripheral modulation – inflammatory soup
- Spinal modulation – gain on the amplifier
- Descending modulation – credible evidence
- The pain neurotag
- Systems to get you out of trouble
- How dangerous is this really?
- Importance of context

Sensitive alarm system

- The spine is hyperprotective
- If the brain perceives vulnerability, protection will increase
- Alarm system metaphor
- Timing of pain – speed of change
- Twin peaks
- Short term and long term sensitization examples

Take home messages

- Pain is protective, not a symptom of damage
- Pain is overestimating what is going on in the tissues
- Understanding this will help you recover

**Explaining what to expect from here**

- Recurrence
- Prognosis
- Pacing
- Tools
- Return to work

**Tricky questions**

- Are you saying it's in my head?
- Does that mean my pain isn't real?
- Could they have missed something?
- So you aren't going to do any massage or anything?
- Shouldn't I get an MRI?

**Introduction**

Spiel

- Firstly I'd like to thank you for being part of our study.
- My background is in clinical physiotherapy and I have specialist training in pain science and acute low back pain.
- We are doing this project because we have learnt so much about low back pain in the last 20 years, that the way we are treating chronic low back pain now is a real focus on you understanding what you need to do and why you need to do it to get better.
- The Commonwealth Government is excited about this and has asked us to try it with people early on, so that is what we are doing.
- So I know you have already been assessed, but we want to double check we have ruled out all the nasty things because we have to protect our own backs here.
- Once we have ruled it out, I want to know about everything that we now know affects pain. And tell you about what we now know about those things in a way you will understand.
- What I have been finding recently with my patients is that it can be really helpful to understand the biology of pain, particularly in low back pain because it can give some explanation for why it is so painful, when often a specific cause cannot be identified. I have also found that the more people know about their pain, and why they need to do certain exercises, the more reassuring this can be. This has also been shown in recent scientific studies. More knowledge about pain tends to help with these problems.
- I'm going to ask you a lot of questions and I'm going to do a lot of talking, but at the end of this, I really hope that you have a clear understanding of what is going on with your back, and a clear direction to plan your recovery.
- I hope you will also have a clear understanding about what to expect from here, and no worries.
- I have been selected as one of the experts because I have been involved in this area for some time, and I am studying a PhD on this topic.
- The aim of this is to give you a level of knowledge and understanding that you need to make the fastest recovery possible.
- So its going to be important that at the end, that I can get an idea of how much you have understood. I also want to know at the end if you still have any things that you don't understand or that you are worried about.
- My job is to teach you this stuff, so its really important that you let me know if you don't understand anything
- Gone are the days when we can give you a pill or an injection – it never works. We now know it doesn't work.
- You have been referred over by ….., can you please tell me in your own words what you think is going on with your back? How do you think its going so far? Ok, I have to do my own set of questions because we are really good at spotting the nasty things, but I just need to double check nothing has been missed (because it'll be on my back). Something can be missed but there no way two people will miss the same thing.
- The best evidence we've got, is that the things that determine recovery are the way you make sense of your pain, and not the things that are in your back. Even now, I can tell that you are really worried about this, and almost convinced that this is never going to get better. One of the big challenges for me is to explain to you why that doesn't have to be the case. Because even expecting that will increase your chances of not recovering quickly. There are no risks at all to thinking about this stuff.

Mechanism of injury

- Clearly you have done something. Otherwise it wouldn't hurt so much… I understand that it started hurting when you lifted that thing….

- Nil MOI: It's very possible that you haven't damaged any tissue; but because there are so many alarm systems…and the brain is always on the look out…that it could be anything! If it hurts that badly then it could be all sorts of stuff. Maybe it was just that things weren't operating as well as they could in there. And this is the way it's letting you know.

- (If someone has an idea of the multifactorial nature of pain): The system is so hyperprotective that you are probably getting close (to an injury). This is where the physio will be great – you just need to be sure to slowly return to function. "what if it just goes back to how it was with a dull ache every now and then??" Maybe that would be a good time for you to see a good physio to help you with a physical upgrading…to help you get a bit fitter and stronger.

Conversation about worries

- Can you tell what concerns you the most about this back pain?
- Have you had any thoughts about what your back might need in terms of medicine or therapy?
- This sort of stuff is important for me to know because it affects your brain evaluation of danger. We know that pain is very much related to your brain's evaluation of danger. There are lots of body systems that can modify this evaluative process.
- (When discussing concepts, remove the observables (emotion, fear, mood) and rather talk about the systems that control these things): It's all about your brain's evaluation of danger. And your immune system can modify that. And your endocrine system can modify that. And your sympathetic nervous system can modify that. And when you worry, that will also change your pain because you are worried about damage.
- Are there any other worries that you have which we haven't covered?
- So how was it the next day…were you worried about that at all? We are going to come back to that because I think I might be able to make sense of that for you.
- My job is to teach you this stuff, and I hope that by the end of our time together, you will have a clear understanding of your back pain, what to expect from here, and no worries.
- Example questions
    - Can you tell me what you think is causing your pain?
    - Have you had any other thoughts about what your back might need in terms of medicine or therapy?
    - What is it that concerns you the most about your back pain?
    - Are you worried at all that will cause damage in your back or slow your recovery?
    - How do you see yourself recovering?
    - How has you family reacted to you having this back pain?
    - What about work? Is work being supportive? How do you feel about going back?
    - What have you been doing to cope with the pain so far?
    - Are there any other worries that you have which we haven□t covered?

**Diagnosis**

Structural Diagnoses

Spinal alarm system

- Lets say you want to protect your most vital thing, like precious jewelry.
- Where would you put it?...in a safe.
- That's exactly how we are constructed!
- Our brain is our most important thing – no brain, no you!
- Our second most precious thing is our spinal cord – that is what keeps the body talking to the brain.
- Picture of how well encased the spinal cord is: thick bone, disc, ligament, muscle
- So let say you wanted to be extra sure no-one went near that precious thing – what would you install?...an alarm system.
- Absolutely. That is exactly the way we are constructed
- Picture of vertebra – that's the bony bit. But you need movement → sideways view: there are these things in here that are just full of ligaments.
- So if you do anything there that's a little bit dangerous, which you have done because it hurts, it rings the alarm bell.
- The alarm bells converge with maybe 150 of them going into one nerve, which goes toward the brain. That message says "danger".
- In fact, we don't know which alarm bell went off and we are never going to know that. And it doesn't really matter because we'll treat it the same way.
- One of the reasons that backs really hurt when you hurt them is because we have so many alarm bells
- We could even do an MRI or CT and we'll see all different shapes and stuff but we have no way of knowing where the alarm bell that rung is. So there is no gain at all in having a scan.
- The reason we know the alarm has rung is that your back really hurts (Your pain is completely legit)
- Clearly when you picked up that thing you did something that rang some alarm bells. But it is so well protected, that you would be ringing alarm bell even with a tiny injury
- In fact, sometimes you don't have any injury at all, you just came a bit close.
- Pain is about protection. It's about stopping you doing things. Which is fantastic if the pain is accurate. One problem we have with the back is that it's overprotective. And if you don't know that and don't realize that that is how we are setup, then you are going to overprotect. When we overprotect and we don't move enough then the problem becomes worse.

Disc

- Disc diagram as strong ligament tissue. A couple of small ligaments in the knee hold the whole thing together. It's just like the ligaments in the ankle – they get injured and heal up. You gradually get back to running, but if you do it too quickly, you could make it worse. Conversely if you don't do enough, you can end up with a really stiff ankle and things take much longer.
- The disc is a really strong ligamentous thing, just like the ligaments in your ankle. Same stuff. Absolutely covered in alarm bells that are looking for anything dangerous. They are all over the bones and joints and ligaments and muscles.
- Picture of vertebra – that's the bony bit. But you need movement → sideways view: there are these things in here that are just full of ligaments.
- Cross hatched ligament diagram
- There is actually no better part of your body to injure, because this part is so solid and well protected. Even when you do injure it, it fixes itself.
- You know how strong ligaments are? A ligament the size of your little finger holds the knee together, every disc has at least as much ligament in it as that or more. As you get older, they don't move as much, but they stay strong.
- Discs are amazing! Cup model: When you bend forward, it put a bit of strain on this ligament (posterior), when you bend back on this ligament, when you twist its over here etc..the ligaments control movement, just like in your ankle. Have you ever sprained your ankle? How is it now?
- Emphasis on similarity between ankle and back ligs
- If we took an image of this with an MRI, we'd see that a few of your discs are curved out a bit. That's completely normal. In fact, there are some people that you can't even see their discs. But they don't have any pain because the alarms haven't rung. Because there is no danger there. 1 in 2 50 year olds will get an MRI that shows changes in the disc. Even if we see a ligament tear here, there are so many alarms that we have no idea whether that is actually what set it off or when that ligament tear occurred. But that isn't an issue anyway because ligaments heal.

Facet Joint

- How do you know its your joint?
- Well that's great. Joints are great!
- Joints love movement and regular compression which are essential for their health.
- Movement distributes the fluid and is really important for the health of joints
- Motion is lotion
- Injuries to these joint are too small to see on xrays or scans but we know that they heal reliably
- We know that it will heal and get working properly again if we slowly upgrade your activity.

Nerve-*root/ "Pinched nerve"*

- When the ligament is torn, the disc is still strong and working well, but the ligament has torn a bit and now the chemicals of inflammation have come the area to heal it.
- And if they get near the nerve they will stimulate the nerve so that your brain gets messages about your leg.
- That will go away, but it can take a while because the blood supply isn't so good.
- We have nerves that are pressing on tissue all over our bodies.
- They are very slippery, and they've got a bit of padding around them.
- Sometimes we get these images and it looks as if we are almost pinching a nerve in the spine. Have you ever heard that term? It feels like that as well!
- Pinched nerves don't really exist. If you are going to pinch a nerve, what you will feel is not pain, but numbness.
- The only way we know if a nerve is truly being compressed is if there is a loss of sensation rather than an increase in sensation – that is a sign of irritation not pinching.
- Don't forget that there are many of these alarm bells, and it's a bit inflamed, and the inflammation makes those alarm bells ready to fire, so all you need to do is move the tissue and those alarm bells will ring as a protective strategy.
- Isn't that great? It's a protective strategy – you aren't even close to injuring.

Muscle strain or spasm

- Muscles are great things to injure because they have an awesome blood supply and they heal really well.
- The other great thing about muscles is you can train them, and they are really adaptable.
- At the moment the muscles in your back are being very protective – there are a few things we can do to modify that like muscle exercises, stretches, pilates.
- The other way we can deal with tight muscles is looking at the nervous system and the brain.
- We are going to talk more about the protectiveness of the system and how we change this in other ways

Biomedical diagnosis

Arthritis/ degeneration /old age

- Timing
    - "So tell me when the pain started." (They probably won't say that they have had slowly building pain for the last 20 years. )
    - "so your pain doesn't match the starting of the changes there…and that makes complete sense because the **danger receptors respond to sudden changes not gradual changes**"
    - e.g. if you put a 42 degree thermode on your finger, you can tolerate it for an hour or more because it doesn't activate danger receptors, but you will get a third degree burn. Whereas if you put a 60 degree thing there, the temp changes so quickly that you take it away so quickly that you don't burn the skin – that is, the protective function works!"
    - If it is really slow, it doesn't work so well. That's why cancers kill you.
    - Its about speed of change. Quick change will set off the danger receptors.
    - If the changes are slow, the brain probably concludes that there is no real danger.
    - If there is no pain, it means that these changes in the tissues are not perceived by your brain to be a threat
    - Most people will notice that their pain started at some stage or another.
    - The degeneration didn't start then…it would have started a long time ago.
    - Xray findings don't necessarily match pain. In fact, your xray would have looked that way for a long time, and you haven't had a big problem until now.
    - Most people with worn joints never know about it.
    - The over 60s have less back pain than the under 60s. This provides a bit more evidence that pain is not necessarily related to the amount of degeneration in the tissues
- Hip replacement success
    - These are your vertebrae all the way up, pelvis and hips. When hips start to wear out, every time you take a step, the entire body weight is on the hip, on one joint surface.
    - So that is quite a sudden increase in danger in there. So I can understand that it would cause some inflammation.
    - There are 3 joint surfaces here – so it is impossible to put the same load through your back every time you walk.
    - When we talk about arthritis in the hip, there is a whole lot of people who have nasty wear and tear and are pain free, and others with no wear and tear and heaps of pain.
    - It can look good on x-ray and still be painful.
    - Why do hip replacements work so well? I don't know. Because when you do a hip replacement you do so much injury: you dislocate the hip, saw through a bone, cut all the muscles and ligaments. And it doesn't hurt!
    - No-one understands why that is. The best explanation that we have is that it is your brain is satisfied that you have done what is required.
- Knee arthroscopy
    - Degenerative knees underwent scope or Placebo. Surgeon went in and fixed it up, or went in and did nothing. And the results were the same. Half of the people that received the Placebo surgery couldn't believe they were in the control group because the results were so good.
    - (However, you need to be very careful not to imply the "its not real" implication)
    - Even when there is severe degeneration, we do the same things.
    - Motion is lotion.

Spondylolisthesis

- Andre Agassi won Wimbledon with one.
- It can look unstable but you can't see all the tough cartilage and ligament in that area – its solid and strong.
- Spondy can be a cause for concern , so can I just check again that you don't have any of these signs?
- OK great – we are clear to go.

Instability

- It really does feel like its unstable doesn't. But it is not unstable.
- Use drawing to show how reinforced the area is.
- If you were to design something to protect the spinal cord, would you design something that was unstable?
- Backs don't collapse.
- There are some signs that will tell us if we need to look further. Do you lose control of your bladder or bowel, stocking numbness etc?
- Anatomy textbook: this is so tightly held together, there is no way it can collapse or slip out or anything.
- But I'd really like to explore that feeling, because that might give us some important information about what your brain is trying to protect you from.
- Sometimes when people don't know when the pain is going to come on, it feels unstable because that is what the pain means to you, that you have damaged something.
- But actually that's not how pain works….spiel. A bit more work required…

Other diagnoses

My back is out

- It's amazing how much it feels like something is out isn't it?
- It's not "out' but that is exactly what is feels like
- The back is really good at giving that feeling
- Anatomy textbook: this is so tightly held together, there is no way it can collapse or slip out or anything.
- But I'd really like to explore that feeling, because that might give us some important information about what your brain is trying to protect you from.
- Sometimes when people don't know when the pain is going to come on, it feels unstable because that is what the pain means to you, that you have damaged something.

My pelvis is twisted

- When muscles in the back go into protective spasm, they can pull you into strange position
- This will resolve itself
- Rather than being a cause of pain it is more likely a symptom of the fact that the back is in protection mode
- We can treat the symptoms and it can give you relief, but it is always important to treat the problem as well as the symptoms
- What I want to do is talk about all the things that have caused your back muscles to respond the way they have

Insecure

- The back is really good at giving that feeling of insecurity
- I can tell you that backs don't collapse.
- There are some signs that will tell us if we need to look further. Do you lose control of your bladder or bowel, stocking numbness etc?
- Anatomy textbook: this is so tightly held together, there is no way it can collapse or slip out or anything.
- But I'd really like to explore that feeling, because that might give us some important information about what your brain is trying to protect you from.
- Sometimes when people don't know when the pain is going to come on, it feels unstable because that is what the pain means to you, that you have damaged something.

**Therapy so far**

Mechanism of physiotherapy treatments

Manual therapy

- When you push on a joint the whole thing moves.
- If you were to push on my back now you would see my whole body move up and down, not just one joint, so it isn't very diagnostic.
- What we do know is that you can't fix tissue by pushing on a joint.
- You haven't solved any problems as such – but the pain goes away.
- Which is so interesting, it tells us quite clearly that we can change your pain even if we aren't fixing the problem.
- Look how modifiable pain is! When we do this (manual therapy), we don't do anything to the problem, but we change the pain. Clearly the problem is not in the tissue, it's in how your brain is interpreting stuff.
- It'll take a bit longer to get the tissues to heal.
- An injection can relieve pain by stopping the alarm bells ringing, but it isn't fixing the problem.
- ….that's really helpful because its given your back all the right signals that indicate – ok, its safe to back on track with things now

*Manipulation/"adjustment"*

- It's amazing how much it feels like something is out isn't it?
- It's not "out' but that is exactly what is feels like
- The back is really good at giving that feeling
- When you go to the chiro, he doesn't put anything back in, he just removes the feeling that it's out by doing things at the joints
- There are some really good chiros, and really bad chiros, just like there are really good physios and bad ones
- There is pretty good evidence that the best of these professions do things that can be helpful
- It has given your back all the right signals that indicate – ok, its safe to back on track with things now
- With chiro, the very best evidence tells us that it has nothing to do with the click, or the joint, and that it is something else. So it might work, but its almost certainly not working how we used to think it worked.
- How might it work? There is a whole bunch of things that physios and chiros can do that will bombard the brain with sensory input. This releases a whole lot of chemicals, and you get a nice, short term pain relief.
- How long does it last?
- This does nothing for the problem, but it can help the pain.
- Your other option there would be to take some serious panadol. It will more than likely do the same thing i.e. give you some relief.
- My job is the help you fix the problem. You need pain relief but that shouldn't be your only treatment.

Motor control/stability

- TA exercises can be really helpful because they get the brain re-connected with the body part.
- We are moving away from calling them "stability" exercises, because the problem with the back is not that it is unstable.
- The spine is incredibly strong, and held together with really strong ligaments and muscles.
- Motor control exercises help get the brain re-connected with that part of your body, and can help reduce un-wanted muscle patterns like guarding.

Mackenzie

- …that's really helpful because its given your back all the right signals that indicate – ok, its safe to back on track with things now
- The exercises can be great because they can reduce pain and gradually get the back moving normally again.
- We used to think they might be pushing the disc "back in" but we know now that isn't the case.
- Discs can bulge and sometimes get injuries around the outside of them, but they never go "out" and need to be pushed back in.
- The exercises probably work because they are gradually getting the back moving again.

Exercise

- Movement not only increases the health of joints, soft tissues, circulatory and respiratory systems, it as another very important function. Educated movement is brain nourishing, because it establishes and re-establishes fine functional sensory and motor representations in the brain, using pathways laid low by fear and ignorance.
- Gradual exercise is a great way of exposing the back to threat in a safe way. This will reduce overprotectiveness which we know is a bit of a problem with back pain.

Explaining pain

- Learning about pain physiology reduces the threat value of pain. Reduced threat will reduce the activation of all our protective systems: sympathetic, endocrine and motor. This in turn helps restore normal immune function.
- Combining pain physiology education with movement approaches reduces pain and improving physical capacity and well-being.
- We want you to understand as much as possible about what is causing pain, not just what you should do about it.
- The best evidence we've got, is that the things that determine recovery are the way you make sense of your pain, and not the things that are in your back.

Mechanism of medical treatments

Tablets

- Tablets can be really useful in the early stages of low back pain
- They can look after some of the chemicals of inflammation, which is great because it helps you get moving
- Just taking the tablets won't be effective though – there is this other stuff we need to consider

Injections

- Injections for the back are really interesting
- You hear stories of people having great success, but a lot of the time it doesn't work.
- The thing is that you anaesthetize the danger messenger nerves as well as sensory nerves, so there is no way of knowing if it was danger messages or just normal sensory messages coming from that area. Remember the diagram….?
- If it can give you relief, then I'm all for it. But it's quite invasive and certainly not a guaranteed outcome.

Surgery

- Surgery should always be the very last resort
- Unfortunately the success rates of surgery for back pain are not good at all
- It also provides some more evidence for the stuff we are going to be talking about. That pain is about much more than just what is going on the tissues…remember the diagram?
- They've been trying the "find it and fix it" approach in back pain for years, and most of the time it doesn't work. This is because back pain is caused by a bunch of complex processes. It's not just about a signal coming from the back…

Mechanism of alternative treatments

- There is a whole bunch of things that these health practitioners can do that will bombard the brain with sensory input. This releases a whole lot of chemicals, and you get a nice, short-term pain relief.
- This does nothing for the problem, but it can help the pain.
- Your other option there would be to take some serious panadol. It will more than likely do the same thing i.e. give you some relief.
- My job is the help you fix the problem. You need pain relief but that shouldn't be your only treatment.

**Pain biology**

What is pain all about?

What is pain?

- Pain is a normal protective response to something the brain has assessed as threatening.
- It designed to get you out of trouble by making you change your behavior
- It involves all of your body systems and all of the responses that occur are aimed at protection and healing
- It's about stopping you doing things. Which is fantastic if the pain is accurate. One problem we have with the back is that its overprotective. And if you don't know that and don't realize that that is how we are setup, then you are going to overprotect. When we overprotect and we don't move enough then the problem becomes worse.
- It's a system that has been perfected throughout the evolutionary process.
- There are many myths, misunderstandings and unnecessary fears about pain
- We've found that understanding how and why we experience pain can be really useful for something like back pain, because it can give some explanation why it is such a painful and disabling thing, even if there has been little or no tissue damage.

Pain is protective

- Pain protects you, it alerts you to danger, often before you are injured or injured badly
- But, the pain system can behave oddly and even fail sometimes
- As a rule, back pain is overprotective. Anything around your spinal cord i.e. spinal pain will be particularly overprotective. That wasn't a problem when we were cavemen and were always forced to "test it out". If we were cavemen, I'm sure if we hurt our back, we would try it the next day, just like you would with an ankle. If you twist your ankle, next morning you get up and you test it out and see how it goes. We should do the same thing with backs. But we tend not to because we get really frightened of it because it means all this stuff. Anything that is unpredictable like back pain can be quite frightening.
- The really interesting thing about pain is that the amount of pain you experience does not necessarily reflect the amount of damage that has taken place.
- Sometimes we can have major injuries and no pain, and other times we can have tiny injuries and a huge amount of pain. Pain is definitely not a good damage meter.
- So we know from the biology of pain that it is not a symptom of damage, it is more of a protective device.
- Even if there is no tissue damage at all, if the brain has assessed a situation as threatening you can experience pain. The more threatening the situation the worse the pain will be.

Pain is not a measure of tissue damage

- Most commonly, pain occurs when your body alarm system alerts the brain to actual or potential tissue damage. But this is only one part of a big story
- Nociception (danger reception/sensation) is not sufficient for pain
- stories: shark attack, hammer in the neck etc etc
- if the brain has decided that the situation is not dangerous or threatening, then pain will not be produced
- If the brain thinks that experiencing pain is not the best thing for survival (imagine a wounded soldier hiding from the enemy) you may not experience pain at the time of a very severe injury
- Many changes in tissues are just a normal part of being alive and don't have to hurt
- you can also have pain with no danger messages coming from the tissues
- stories: phantom limb etc etc
- Scientists did a really sneaky experiment on volunteers who put their head inside a Placebo stimulator and were told that a current would be run through their head. Pain increased in line with the instructed intensity of stimulation even though no stimulation was given. That showed us that there is more to experiencing pain than tissue damage.
- pain is dependent on complex neural processing and adaptation rather than being a robust informer of spinal pathology.

Pain tries to get us out of danger

- As unpleasant as it is, pain serves a very useful purpose. It makes us change our behavior to get us out of danger. That's why ignoring it is not useful. Where it gets tricky is in situations like back pain, where people can often be confused about what is the best approach particularly if our body is telling us one thing, like lie down, and our physio is telling us to get moving.
- Pains from poor posture and sprains are simple 'everyday' pains that can be easily related to changes in tissues. The brain concludes that tissues are under threat and action is required.
- Its about stopping you doing things. Which is fantastic if the pain is accurate. One problem we have with the back is that its overprotective. And if you don't know that and don't realize that that is how we are setup, then you are going to overprotect. When we overprotect and we don't move enough then the problem becomes worse.
- Pain can be so effective that you can't think, feel or focus on anything else.
- This is where it can be useful to think back to pain being an overestimation of what is going in the tissues. You have been checked out for all the real nasties that can cause back pain, and you know now that the pain you are feeling, although its really terrible right now, is not a good indicator of damage.
- In fact, right now, those tissues are better protected than ever!
- Judge whether what you are doing is safe by how vigorous the activity you are doing is, rather than how much pain you are feeling. The brain is probably overestimating things.
- We have really good evidence now that tells us that staying active is very important for recovery from back pain.
- We know biologically why this can be painful to begin with but not damaging.
- If you work with the physio to gradually get back into things, you tissues will be very safe, and you also reassure your brain that it is good to move.

Visual metaphors

- vision is like this as well
- what we see is not simply a reflection of light onto the retina
- that signal goes through very complex, split second processing to give us an image that is biologically useful
- its wrong (the colours are the same), but its biologically useful
- Pain is like this, it's a conscious experience based on complex neural processing, not simply signals coming from the body
- Descartes diagram? More accurate diagram.
- In a split second, and outside of your consciousness, your brain processes a great deal of information and calls on a great deal of previous knowledge. You don't know this is happening. The first thing you are aware of is that you see a sensible and meaningful image/you feel pain. This is conscious representation of what is really there. It is not accurate, but it is meaningful and sensible
- Pain, like vision, is a conscious experience that is based on many complex processes, not just the sensory information coming from your body

Complex output

- It's a hard thing to get your head around but pain is not an incoming thing. I'm going to attempt to explain why.
- Because of this, pain is not always an accurate assessment of danger to the tissues
- Descartes diagram
- Thirst story, vision story
- Anything we experience involves many thoughts and emotional contributions
- We need to talk about the brain in order to really understand pain – especially pain that persists, spreads or seems unpredictable
- The brain evaluates the sensory input from the tissues of the body and draws on complex evaluative processes. Pain then, can be considered conscious experience based on the brain's evaluation of how much danger the tissues are in.
- The evaluation of how much danger the tissues are actually in happens really quickly and happens outside your awareness and control. Pain then, depends on the unconscious evaluation of threat to body tissue.
- Pain is the conscious correlate of perceived threat to tissues that motivates us to get our tissues out of danger.

Thirst

- Thirst is not a great measure of dehydration.
- Similar to pain, thirst is something that makes us change our behavior.
- However there are many times where we can be dehydrated and not be thirsty, because the brain has decided there is no need.
- The same goes for pain – its not a good measure of what is going on in the tissues because it is the product of many complex processes in the nervous system

How is it processed and what can change it?

Nociception vs. pain

- This is the area in your back that we are talking about in which the alarm bells are ringing.
- There are particular nerves that detect danger – and because its your back, and its protecting something important (your spinal cord) there are heaps of them.
- What is interesting here is that these fibers don't transmit "pain" messages, they transmit "danger" messages.
- There are danger receptors in there that respond to chemicals, temperature and mechanical stuff like pressure.
- These are the alarm bells. And there are heaps of them.
- That sends a danger message to the spinal cord which then goes up to the brain.
- But the brain has to think of everything (write things in, what is particular to them??) e.g. worries, beliefs about what has happened (for example, your immediate conclusion is that you have completely ruined something in your back – which is a fair conclusion because it hurts so much, but that causes brain activation)
- It's the sum total of all this that causes your back pain.
- So the danger message itself is not enough to cause pain. In fact, you don't even need a danger receptor to be activated to feel pain.
- If they are coping with this: "If the brains evaluation is different to this (tissue) then the brain changes this (spinal cord). It can turn it up or down.

*Danger to the tissues doesn't = pain* – pain experiments & amazing pain stories

- The ringing of alarm bells in the tissues is not enough for you to feel pain
- E.g. shark attack, impaling of objects, wartime stories, NRL player finishing a game with a broken neck
- In these situations there a heaps of danger messages flooding the system, but no pain is felt
- Many and varied cues may relate to the pain experience, but it is the brain that decides whether something hurts or not. 100% of the time, with no exceptions.
- This tells us that there is much more to the story of pain
- What is happening in the tissues is only one part of the amazing pain experience

*Pain doesn't always = tissue damage* – pain experiments & amazing pain stories

- In fact you don't even need an alarm bell to ring in the tissue to experience pain
- E.g. phantom limb, Courvade syndrome (well documented)
- All you need is the brain to decide a part of your body is in danger
- There are heaps of things that might contribute to the brain deciding this

Pain processing diagram

- this is the area in your back that we are talking about in which the alarm bells are ringing.
- That sends a danger message to the spinal cord which then goes up to the brain.
- But the brain has to think of everything (write things in, what is particular to them??) e.g. worries, beliefs about what has happened (for example, your immediate conclusion is that you have completely ruined something in your back – which is a fair conclusion because it hurts so much, but that causes brain activation)
- It's the sum total of all this that causes your back pain.
- Its up to the brain to construct as sensible a story as possible, based on all the information that is arriving.
- So this shows how pain is not an incoming thing, it's a very complex output, just like something like vision

The Pain Neurotag

- There isn't just one pain centre – there are heaps of areas that pain borrows or hijacks to express itself.
- E.g. the parts that:
    - Organize and prepare movements (pre-motor and motor cortex)
    - Concentration ?introversion (cingulate)
    - Problem solving and memory (prefrontal cortex)
    - Fear and addiction (Amygdala)
    - Sensory discrimination (Sensory cortex)
    - Stress responses and motivation (hypothalamus/thalamus)
    - Movement co-ordination (cerebellum)
    - Memory, special cognitions (hippocampus)
- The brain acts as a "meaning attributor" to the incoming signals
- Lots of different things will change the meaning the brain attaches to the incoming danger message.
- For example:
    - Beliefs
    - Previous events
    - Worries
    - Knowledge
    - Other sensory stuff
    - Social context
    - Anticipated consequences
    - Family
    - Media
    - Culture
    - What the physio said
    - Scan results
- All these things will change the meaning of that incoming danger message. Once the brain takes this into account, it will decide whether what is going on down there is really dangerous or not.
- If its assessed as not really that dangerous → no pain
- If its assessed as really dangerous → pain+++
- All this happens in a split second, and it's outside of your awareness!

Systems to get you out of trouble

Inflammation

- If there is a little bit of inflammation, the whole system is sensitive.
- This makes the danger messenger nerves much more likely to fire.
- The sensitivity will make you pay more attention to the body part and make you protect it and help it heal. Really helpful.
- That sensitivity takes a while to get rid of, so you need to stick at this for a little while.
- Your pain is not to do with damage, its telling you to gradually get moving to flush irritating chemicals out of the area.
- This will be quite painful in the beginning to do this.
- But this is where the physio can be great, they will get you moving in a way that you are completely safe.
- (In acute pain, the inflammation is a nice way of explaining things so that they don't have to face their demons just yet)

Tissue healing

- Even when there is a lot of healing to do, it is a strong and dependable process…unless we don't let it do what it needs to do
- In your situation, the tissues need movement to heal – this gives the area a great blood supply and prevents in stiffening up and getting weak
- Like other injuries, a period of relative rest is appropriate but to heal optimally we need to gradually get that area moving.
- Think about what you would do if you injured your ankle…
- The main thing to know is that whatever you have done in there will definitely heal.
- Tissues with a poorer blood supply like ligaments take a bit longer to heal that blood rich tissues like muscles. This is an even better reason to stay active because it promotes circulation.
- Tissues always heal but they can remain deconditioned and a bit unhealthy
- This is where the physio is great…

Peripheral modulation – inflammatory soup

- Inflammation is designed to hurt, and it does!
- It is essential to the repair process, and is a sign that the repair system is doing a really good job
- It'll go away, but its one of the reasons it's painful to get moving sometimes with a back problem
- The area gets flooded with a bunch of chemicals like immune cells, histamines, clotting factors and enzymes for mopping stuff up. This makes the area swell up sometimes – which is likely to make movement painful.
- The reason it is more painful to move when an area is inflamed is because the danger messenger nerves get sensitized by the chemicals – they get primed to fire.
- That is why even the slightest movement can be really painful.
- The inflammatory soup that is bathing the nerves in the area makes them much more likely to fire → which sends a lot of messages to the spinal cord and up to the brain.
- The brain will be very interested in these signals. But remember that humans are able to draw on a wide variety of cues in order to make the danger message meaningful.
- "issues in the tissues" helps explain a lot of aspects of pain – particularly why only a little bit of damage can result in a lot of pain (if you think about all those sensitive nerves).
- Medications can be useful to clear out a few of these chemicals, and so is movement.
- But the story doesn't end here…

Spinal modulation – gain on the amplifier

- To fully understand pain, we need to head into the spinal cord and up to the brain, which is the command centre of the alarm system
- When the spinal cord gets an influx of danger messages, it adapts really quickly to cope with the demand
- Changes occur in the neurons in the spinal cord as well as the nerves coming down which normally keep the relay quiet
- When this happens its like turning up the gain on an amplifier – the same signal gets amplified as it heads up to the brain
- So now you have another area where the nerves are ready to fire
- This all happens within seconds of getting all the message from the tissues

- The net effect of this is that things that used to hurt, now hurt more and things that didn't hurt before now hurt.
- The danger messenger neurons are much more sensitive and looking out for you.
- Now even just touching the skin or having a slight temperature change might cause a danger message to be sent to the brain
- In a way, your brain is being tricked
- It is operating on faulty information about the condition of your tissues
- All these changes in the spinal cord, which can start happening very early in an injury, give us good evidence why the pain we feel is not a good measure of exactly what is going on in the tissues
- In back pain it is often an overestimation
- This is why we can think of the cord as being a magnifier or amplifier of what is actually going on in the tissues
- The brain gets a distorted image
- When this happens, the brain is being fed info which no longer reflects the true health and abilities of the tissues at the end of the neurons
- This happens to everyone when they are injured
- Metaphors for sensitization
    - Amplifier
    - Super alarm system
    - Computer P malfunction

Descending modulation – credible evidence

- Lets move up to the brain
- The brain is responsible for making the final decision whether something is dangerous for the tissue and needs protecting
- We now know that brain will very likely be getting an overestimate of what's going on in the tissues due to sensitization
- So the brain gets all these danger messages but has to "weigh the world" before it decides if something is actually dangerous enough to need protection from
- The brain acts as a meaning attributor to all these signals
- If the brains evaluation is different to this (tissue) then the brain changes this (spinal cord). It can turn it up or down.
- Many things can affect what these signals mean to a person
- The brain looks for any piece of credible evidence that protection is required
    - This doctor thinks I'm putting it on
    - The CT couldn't find it so it must be really bad and deep
    - Aunt Doris had back pain all her life and now she is in a wheelchair
- Even these thoughts are nerve impulses that are threatening to a brain that is concerned with survival
- If the brain perceives something as threatening, protection (and thus pain) will increase.
- That doesn't mean that you think the pain is worse, the pain is worse.
- So we have a direct pathway by which pain can be changed just by thinking or worrying about something

How dangerous is this really?

- My colleague has a great story that helps understand this idea that the brain ultimately decides if protection via pain is required, by asking "how dangerous is this really?"
- Snake story
- So the point of the story is that pain is a protective response to something the brain has perceived as threatening.
- That story really resonates with me because I see similar things in my patients at the clinic with back pain. Often with recurrent episodes, the pain is severe, similar to the first episode, and results from doing something quite minor like sitting or picking up a pen.
- Think about your situation now and when you first had a back problem. Your body has installed a really sensitive alarm system that may not be giving a great indication of the state of your back.
- All of these things we have talked about can give you a biological explanation as to why the pain comes back so easily. A sensitive alarm system.
- It remembers that you have been here before, and you are in trouble.
- What has happened in you back could be relatively minor, but the nervous system has decided that you need protecting – and now you are hurting. A lot. More sensitivity = more protection = more pain
- So unfortunately it's not as simple as just thinking the pain away. The decision that your back needs protecting was based on a lot of factors outside of your awareness
- Where we think it is important to start is having you learn about this stuff, and hopefully give you a fuller understanding on what your pain means.
- In particular, you now know that pain is always a protective thing and not a symptom of damage

Importance of context

- The context of the pain experience is critical
- Exactly the same minor finger injury will cause more pain is a professional violinist than a professional dancer, because finger damage poses a greater threat to the violinist. The event plays a greater role in the violinist's livelihood and identity
- If you step on a piece of glass down at Bondi, this may or may not hurt immediately. It could really hurt straight away because the danger receptors in the toe have been activated which goes into your spinal cord and up to the brain, and with everything else going on, your brain says: protect the toe, so it makes your toe hurt. Lets say we have that same scenario (walk them through), but some idiot flys around the corner and nearly hits you, this time you get across the road and realize that your toe hurts. In that scenario, you still had all of this happening, but your brain said: actually, that's not the issue, the issue is the auditory input of I'm about to get killed, and the rush of emotion from that. Its not until later that your brain decides to make it hurt.
- Pain is dependent on perceived cause e.g. post mastectomy patients who attribute pain to returning cancer, have more intense an unpleasant pain than those who attribute it to another cause, regardless of what is actually happening in the tissues

Sensitive alarm system

The spine is hyperprotective

- In general, pain is overprotective – it's a survival thing.
- In the lower back, because it is housing our second most important structure, it is particularly overprotective.
- ?? Alarm system metaphor
- If this is not your first episode, it will be even more protective
- Add some inflammation into the mix and the brain is even more interested.
- ??Twin Peaks
- Now we have a system that is so well protected by pain that you can even move!
- So here is how we get the system to calm down and get you back on track
- The pain you are feeling is not likely to be because something is badly damaged

OR

- pain is about protection. Its about stopping you doing things. Which is fantastic if the pain is accurate. One problem we have with the back is that its overprotective. And if you don't know that and don't realize that that is how we are setup, then you are going to overprotect. When we overprotect and we don't move enough then the problem becomes worse.

OR

- As a rule, pain is overprotective. The response of the brain is nearly always to turn down the spinal cord, I'll take care of it. Anything around your spinal cord i.e. spinal pain will be particularly overprotective. That wasn't a problem when we were cavemen and were always forced to "test it out". If we were cavemen, I'm sure if we hurt our back, we would try it the next day, just like you would with an ankle. If you twist your ankle, next morning you get up and you test it out and see how it goes. We should do the same thing with backs. But we tend not to because we get really frightened of it because it means all this stuff.

If the brain perceives vulnerability, protection will increase

- when you are stressed or depressed (using diagram), one thing that changes is your mood – which you feel, but another thing that changes are the chemicals floating around the body.
- And those chemicals we know will activate alarm bells if the body is sensitized.
- So if you are depressed, the pain is going to be worse.
- If you are stressed, your pain is going to be worse.
- They are just the cold hard facts of human biology.
- So its worth us trying to reduce your stress and depression because that will help your pain. (We need avoid implying any illegitimacy. Attributing things to biological mechanisms helps you get away with it)
- Whatever causes your depression/stress/anxiety the same things causes chemical to be released in your body. And we can't help that. We are one machine.
- The chemicals in the body will find receptors anywhere they can. If you have a sensitized nervous system, they are the receptors they will bind to.
- Sticking to the biology can be very useful.

OR

- Someone steals all the power tools out of your shed. You install a fancy alarm system. Cat can walk in front of it any trigger it. The tools are really safe but it give you the shits because its always going off, and you are always having to go and see what the problem is, and often there isn't a problem.

Alarm system & safe metaphor

- you want to protect your most vital thing
- precious jewelry
- where would you put it – in a safe.
- That's exactly how we are constructed!
- Our brain is our most important thing – no brain, no you!
- Our second most precious thing is our spinal cord – that is what keeps the body talking to the brain
- Picture of how well encased the spinal cord is: thick bone, disc, ligament, muscle
- So let say you wanted to be extra sure no-one went near that precious thing – what would you install? An alarm system
- Absolutely. That is exactly the way we are constructed
- Picture of vertebra – that's the bony bit. But you need movement → sideways view: there are these things in here that are just full of ligaments.
- The disc is a really strong ligamentous thing, just like the ligaments in your ankle. Same stuff. Absolutely covered in alarm bells that are looking for anything dangerous. They are all over the bones and joints and ligaments and muscles.
- So if you do anything there that's a little bit dangerous (which you have done because it hurts) it rings the alarm bell.
- The alarm bells converge with maybe 150 of them going into one nerve, which goes toward the brain. That message says "danger".
- In fact, we don't know which alarm bell went off and we are never going to know that. And it doesn't really matter because we'll treat it the same way.
- One of the reasons that back s really hurt when you hurt them is because we have so many alarm bells
- We could even do an MRI or CT and we'll see all different shapes and stuff but we have no way of knowing where the alarm bell that rung is. So there is no gain at all in having a scan.
- The reason we know the alarm has rung is that your back really hurts
- Your pain is completely legit. Clearly when you picked up that thing you did something that rang some alarm bells. But it is so well protected, that you would be ringing alarm bell even with a tiny injury
- In fact, sometimes you don't have any injury at all, you just came a bit close.
- There is actually no better part of your body to injure, because this part is so solid and well protected. Even when you do injure it, it fixes itself. If you go outside, half of those people have injured their back and now its completely functional. Here is Gary Ablett taking a screamer 3 months after he tore the ligament in his disc.

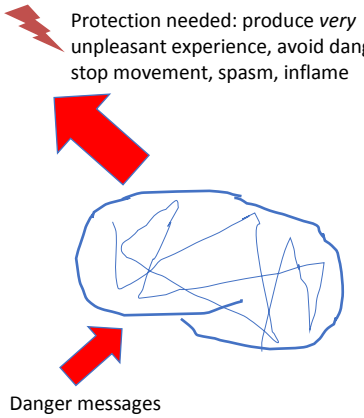
Timing of pain – speed of change

- Part of the healing process is to release inflammatory chemicals.
- They might take a while to get to where you have danger receptors
- When they get there, they will ring alarm bells
- What is really good is that it took two days
- It hurts like anything now, that's a sign of that chemical irritation from a little injury somewhere
- That's good because it tells us it is a little injury
- When you get sudden pain, straight away, it might be more likely that an injury might be ringing the alarm bells
- If you are doing something that is not that sudden, and you get really sudden pain. E.g. if you are walking and talking to your friends, and then BANG you get sudden severe back pain, that's probably a really good sign that you were slowly approaching this zone here (protection line), you might have been distracted by the conversation, the alarm bells are ringing and then there is a lull in conversation and BANG.
- But because you were slowly building up, your alarm bells were going STOP!
- And did you stop? Yeah I couldn't move. There you go! It's a fantastic system.
- When you recover, tissue tolerance probably doesn't change.
- You can increase the height of the mountain by training.
- You return the buffer to normal by gradually increasing what you are doing, and it will creep its way back up to where it was.

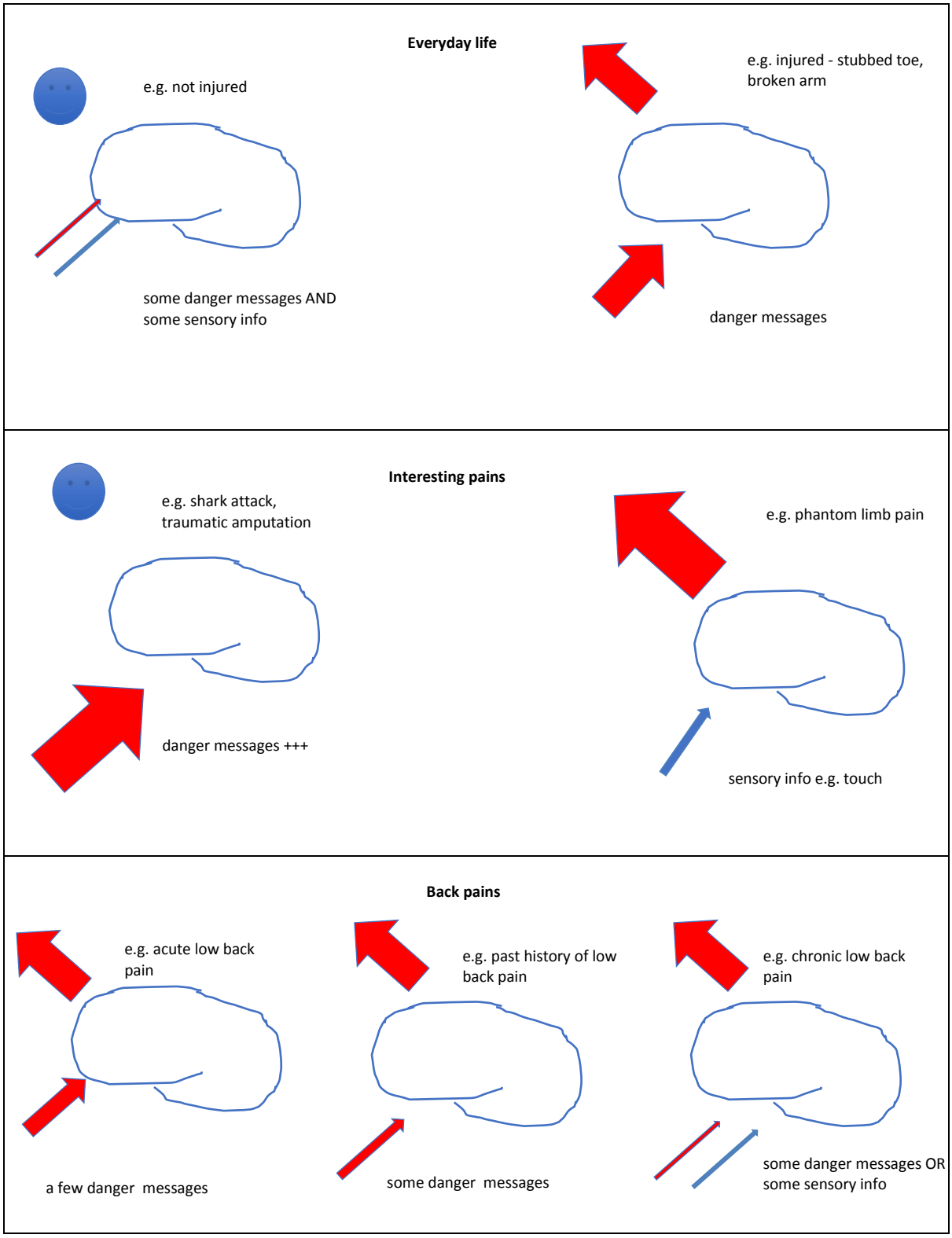
Twin peaks

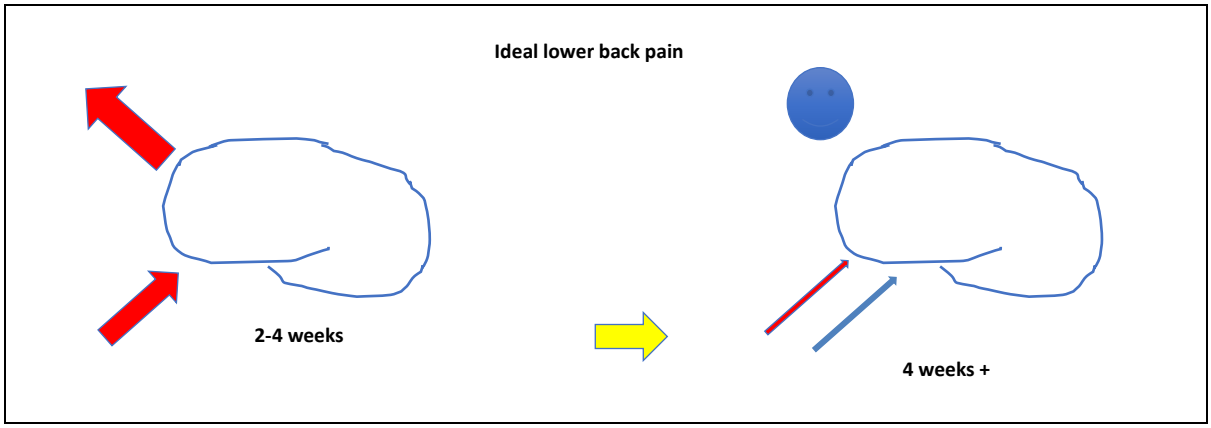
- If there was an incident e.g. bending over, before you bent over on that day, you could probably bend over and lift this much weight.
- But if you had slowly built up the weight you were lifting, you would probably get to here and your back would start hurting, and stop you lifting a heavier load.
- But that's not the way we lift things, we tend to just get in there and lift.
- So you went screaming through this "protect by pain" line.
- And pain is a really effective protector, so it gives us this buffer to protect the tissue.
- And that is because of danger receptors in the tissues. We've talked about that stuff.
- It's a really reliable system that has been perfected throughout the evolutionary process. Its been perfected over generations and generations.
- All animals have this and it's a beautiful system, but it looks like this time it didn't work to protect you in this scenario. Maybe that's just because you did a bit too much a bit quickly.
- And that's nearly always the way that it happens – too much too quickly.
- Insidious onset: this is why we think you haven't actually done anything to the tissues, but you might have come close.
- That's why we think the pain is a sign of other things going on, or you got a bit close. You hit the line. And normally what that makes you do is not pick it up.
- The body will contract muscle and get your immune system going to get you to not pick up the box or push your luck. This response is terrific.
- So you were at this line. And now you have activated this protection thing, particularly if you have injured something.
- Lets say you've torn a little bit of that ligament on the outside of your disc. The disc is really strong, it can cope, but you have exceeded this line(tissue damage line).
- This puts in place a very effective protective mechanism.
- It makes your alarm bells that we talked about a bit more sensitive.
- Then, in a matter of days, this messenger nerve inside your spinal cord becomes more sensitive, so it wants to fire, which means that the danger message gets bigger.
- So this is you now: the disc is only slightly weaker very difficult to tell, but not much. It's a torn ligament that will heal.
- But this: (protect by pain line), because of what's happening in the spinal cord, is probably down here somewhere (lower protect by pain line), because of the sensitivity.
- So now your protect by pain line, when you are a long way short of damaging something, pain will come on because of the sensitivity, not because the tissues are getting damaged.
- The leg pain that is giving you grief at the moment is probably a sign that the nerves are irritated by the chemicals of inflammation, which is going to push the protect line lower again because of that.
- Freaking out that you think something is about to go out and you'll be left with this pain forever – this will take the line down even further.
- Your pain starts at this point – but you are a long way from damaging the tissue. There's no way you are going to get through that.
- Your brain will stop you. You'll hurt, and if that doesn't work it'll make you vomit, faint, fall over, legs wont work.
- The brain will do everything it has to, to stop you getting to that point. So unless you drug yourself up to point of numbness or you are a complete idiot, the tissues are safe.
- Because of the unpleasant experience your brain is producing.

Short term and long term changes in sensitivity – examples

| | Short term | Long term |
|---|---|---|
| Increases sensitivity/pain<br>  | Inflammation<br>Muscle spasm<br>Distress<br>Being very worried/anxious<br>Adrenalin (stress hormone)<br>Fear of damage | Depression<br>Trauma (past or present)<br>Unhappiness<br>Social factors (work/family/friends)<br>General difficulties in life<br>Concern about the future e.g. aging, work ability |
| Decreases sensitivity/pain<br>  | Movement<br>Walking<br>Distraction<br>Medicine<br>Placebo<br>Oxytocin (love hormone)<br>Relaxation | Knowledge about pain<br>Exercise/pacing<br>Happiness<br>Exposure<br>No fear of damage |

How pain might be produced in different contexts. Sharks, nails and peanuts.






Take home messages

- **Pain is protective, not a symptom of damage**
- **Pain is overestimating what is going on in the tissues**
- **Understanding this will help you recover**
- When we first talked, you were concerned about this
- How do you feel about that now?
- **Do you feel like you have an answer to that now?**
- Can you tell me now what you think is causing your pain?
- If you do a little bit more today than you did yesterday, but not much more, you will recover

**What to expect from here**

Recurrence

- Most people that hurt their back will have another episode.
- That is a normal thing that happens because things are hyperprotective.
- You can probably reduce the number of episodes by not just recovering to pain free but then getting fitter and stronger.
- But its also useful that you now know that if you do get a twinge, its not a sign that you have damaged something, it is a sign of protection.
- And you know now that there a many things (in addition to any alarm bells ringing in your back) can could contribute to having this protective response.

Prognosis

- The best evidence we've got, is that the things that determine recovery are the way you make sense of your pain, and not the things that are in your back.
- Even now, I can tell that you are really worried about this, and almost convinced that this is never going to get better.
- One of the big challenges for me, is to explain to you why that doesn't have to be the case.
- Because even expecting that will increase your chances of not recovering quickly. There are no risks at all to thinking about this stuff.

Pacing

- The nature of the system is that if you only progress slowly, and you keep progressing, the system just wont let you damage anything.
- But if you progress suddenly, you might flare-up.
- As long as you apply that principle of gradually increasing what you are doing, like you would with an ankle.
- If you twist your ankle, next morning you get up and you test it out and see how it goes. We should do the same thing with backs. But we tend not to because we get really frightened of it because it means all this stuff.
- If you sprained your ankle, on day 3 it would be feeling a bit better, but you wouldn't run on it yet would you?
- So you don't do that with back either.
- What you would do is check if you could take a few little steps, which could really hurt, so you back and try again tomorrow.
- Use the ankle scenario. It an acute injury of tissue that identical to what is in your back. The commonly targeted culprit of back pain which is the disc. Its identical tissue!

Tools

- Understanding your back pain is crucial
    - Recovery needs understanding of what is going on in your back, as well as these other things we know affect pain
    - Why perform painful activities if you don't understand why they hurt? That just further provokes protective mechanisms.
    - Education, knowledge and understanding reduce the threat associated with pain. Reduced threat has a positive effect on all the input and response systems.
- When I am hurting, it doesn't necessarily mean I am hurting myself
    - Respect pain but don't be afraid of it
- Pacing and graded exposure
    - Every day do a little bit more than you did yesterday. If you stick to these things then this will resolve.
    - If you feel like you are going crazy and you need some temporary pain relief or you need a coach to help you plan these things, then I reckon you should go and see a good physio.

Return to work

- The brain will take into consideration where you are – the baker story.
- Baker would get phantom hand pain whenever he smelt bread because he injured his hand at the bakery.
- Or cyclist who got back and leg pain if we tilted the tv screen to make it look like she was riding up hills.
- This is really sensible!
- Pain is the only system that does this.
- If you get bitten by a snake down in the back shed, you will avoid the back shed – what a clever adaptation!
- This is the same: if you hurt your back lifting an odd shaped box, you are probably not going to want to lift that box again.
- Therefore to be able to recover, we have to train getting back to work, otherwise your brain wont let you do it.
- The way your brain stops you is by making it hurt.
- You might notice that your pain gets worse when you are at work.
- Your back isn't in any more danger at work or more damaged, but it hurts more doesn't it?

**Tricky questions**

*Are you saying it's in my head?*

- This is the question asked most often by people learning the physiology of pain
- We have to honest and say, yes - absolutely all pain bee-sting/paper cut/skiing accident is produced by the brain – no brain no pain!"
- This doesn't mean for a second that it isn't real – much to the contrary – all pain is real.
- In fact, anyone that tells you "it" is all in your head, implying that therefore "it" is not real – does not understand physiology
- Really understanding this is quite empowering.
- Understanding the spinal cord and the brain processes behind the pain experience can provide you with enormous control.

OR

- Every single pain we feel, bee –sting, paper cut its up to your brain whether it hurts or not.
- If we removed your brain the pain would go away but that doesn't mean its not real.
- The pain is an attempt by your brain make you protect your body.
- You don't have any choice when it hurts, its what you do. It works beautifully well.
- ?Give example of the brain making split second decisions about things

*Does that mean my pain isn't real?*

- This doesn't mean for a second that it isn't real – much to the contrary – all pain is real.
- In fact, anyone that tells you "it" is all in your head, implying that therefore "it" is not real – does not understand physiology
- Really understanding this is quite empowering.
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- ?Give example of the brain making split second decisions about things

Could they have missed something?

- If you are the first person who's got some major thing that medicine has missed, then I am really sorry. But if you are like every other human that has ever been investigated, then the risk is minute.

*So you aren't going to do any massage or anything??*

- The thing about massage is that it can relieve the symptoms, but is unlikely to do anything for the cause.
- We know now why it only has a very small effect in scientific research – because what is going on in the actual muscle has almost nothing to do with the problem of back pain.
- All these other things as a whole, cause back pain. And muscle tightness. And if we don't treat these, then we are missing the point.

*Shouldn't I get an MRI?*

- An MRI can show a lot of things because they are really sensitive
- Unfortunately even if the MRI shows something like discs that are curving out or a bit of wear and tear, there is absolutely no way we will know if that is what triggered your back pain
- There are so many alarm bells in there. Any one of them could have rung to give you this pain. And truth is, it doesn't matter where the alarm bell went off, because it will fix itself. The reason we know alarm bells have rung is that you are hurting.
- Sometimes even getting a scan can make people feel worse because it shows a few nasty things, and even though they have nothing to do with the pain, and may have been there for years, people worry that their back has worn out and that's why it's not getting better.
- Many changes in tissues are just a normal part of being alive and don't have to hurt
- We know now that is absolutely not true. I've had patients with perfect scan and in a lot of pain, and others with really nasty scans that haven't got any pain at all.
- We have ruled out all the nasties, everything else that's going on in the back will get better.
- What is really important is that you understand how to best move forward from here, because whatever has happened in your back will heal if you have an MRI or not.

*I'm scared that it's not getting better yet.*

- The best evidence we've got, is that the things that determine recovery are the way you make sense of your pain, and not the things that are in your back. Even now, I can tell that you are really worried about this, and almost convinced that this is never going to get better. One of the big challenges for me, is to explain to you why that doesn't have to be the case. Because even expecting that will increase your chances of not recovering quickly. There are no risks at all to thinking about this stuff.

## **eMethods 4.** PREVENT Trial Placebo Patient Education Manual

**Key principles**

- patient to discuss any topic they wanted to
- no advice provided
- no pain management techniques will be taught
- no reading material provided
- no encouragement or discouragement of ideas presented by the patient
- no information on back pain treatments
- therapist will use techniques such as active listening, along with reflective and reframing statements
- direct questions will be referred to treating practitioner to answer
- a standard response to direct questions will be "As this is a study, I'm not allowed to give you any direct advice. We don't know what the best advice to give people is at the moment anyway. Part of the purpose of this study is to work that out. Who would you normally go to, to get an answer for things like that?"

**Example topics**

1. History of their low back pain
2. Treatments they have received
3. Family
4. Work and lifestyle

**Example responses**

" I can see you are really concerned – the doctor didn't seem too concerned when he sent you over but you still are?"
"I guess there are different ways of looking at things aren't there?"
"So what are the options we've got here?"
"It sounds as though you have a lot of confidence in your doctor. What are the options? What do you think about that?"
"I'm glad we had this chat/ I think you have lots of good ideas/ I can see you have thought a lot about this/ We are keen to follow up and see how things progress"

**Additional prompting questions**

- What have you done during the last week?
- What do you think will help?
- Is there anything else you are expecting to help?
- How is work?
- How is your family?
- How do you feel about your behaviour as a result of the back pain?
- Have you ever had to assist any one else in pain?
- How is your life in general?
- How do you cope with things that stress you?
- What would you say to someone else in your situation?
- What have other people told you about your back pain and back pain in general? Anyone else given you advice? (people at work, pharmacist, yoga/ pilates instructor, friends
- What are your thoughts on medication for back pain?
- What are your thoughts on acupuncture?
- What do you think about surgery for back pain?
- Do you prefer hands on treatment, or exercise treatment?
- Who do you generally turn to for support and help?
- Do you ever use the internet for diagnosing problems or for advice for injuries/ sickness? What websites?
- Who do you consider the best profession to deal with back pain? GP/ Physio/ Chiropractor
- Who would you go to first for a new episode of back pain and why?
- What is the role of imaging (X-ray, MRI, CT scan in the treatment of low back pain?
- What do you think is more important, what the PT/ GP does to you/ prescribes to you, or what they PT/ GP tells you?
- Do you think personality affects recovery of back pain?
- Do you think men and women respond differently to pain in general and back pain?

## **eMethods 5.** Statistical Analysis of Secondary Outcomes

**Secondary outcomes and process measures**

Secondary outcomes were disability (Roland Morris Disability Questionnaire)[1], the proportion of participants who developed chronic low back pain (at 3 months, reporting an average of 2 or more on a 11-point pain intensity NRS over the past week and no periods of recovery during that time), depressive symptomatology (depression severity scale of Depression Anxiety and Stress Scale)[2], healthcare utilisation, global change (Global Back Recovery Scale)[3], recurrence (answering 'yes' to both of the following: i) "In the last 6 months /12 months has your lower back pain gone away completely for a period of more than 30 days, only to return later on?" and ii) "If yes, did the return of LBP last at least 24hrs with a pain intensity of more than 2/10?")[4], pain attitudes and beliefs (Survey of Pain Attitudes[5] and reassurance (assessed using two questions: "How reassured do you feel that there is no serious condition causing your back pain? 0 = not reassured at all, 10 = completely reassured"; "Do you think that your symptoms should be investigated more extensively (laboratory tests, X-rays etc.)"?[6 7].

We collected data on potential mechanisms: catastrophizing (Pain Catastrophizing Scale)[8], back beliefs (Back Beliefs Questionnaire)[9] self-efficacy (Pain Self-Efficacy Questionnaire)[10] and neurophysiology knowledge (Neurophysiology of Pain Questionnaire)[11]. These will be reported separately in a planned mediation analysis.[12]

**Statistical analysis of secondary outcomes**

We also investigated persistence of effects on outcomes at 6 and 12 months by examining the relevant group x time interactions in the mixed models. To compare the incidence of chronic low back pain in both groups, we categorised the status of all participants at the 3 month follow-up time-point as either 'chronic low back pain' or 'recovered'. We defined 'chronic low back pain' as reporting 2 or more on an 11-point NRS for pain over the past week,[13] as well as reporting no periods of recovery (defined as a pain-free period of more than 30 days) during that time.[14] We used a Generalized Mixed Effects Model with a logit link to determine the effect of the intervention on development of chronic low back pain.

We used a similar model as for the primary outcome to estimate intervention effects on continuous secondary outcomes (disability, depression, pain, global change, pain attitudes and healthcare visits). We analysed outcomes at one week. This analysis was not specified in the published statistical analysis plan[15] but was clearly stated in the study protocol.[16] We estimated intervention effects on categorical secondary outcomes (recurrence, further investigations) using logistic regression analyses. For binary outcomes, we used logistic regression models.

We planned a sensitivity analysis to examine the effect of out-of-trial therapy on our primary outcome. That is, we planned a mediation analysis to estimate the direct effect of the intervention on the primary outcome that controls for the effect of out-of-trial therapy.

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## **eResults 1.** Process Evaluation/Mediation Analysis

**Process evaluation – causal mediation analysis**

We followed a published analysis plan for the causal mediation analysis.[1] The main objective of this secondary analysis was to estimate the extent to which catastrophization, beliefs (proximal treatment targets) and self-efficacy (distal target) measured at 1 week post intervention would mediate the effect of pain education on pain at 3 months. Details of the statistical analysis plan and technique are available in the published protocol.[19]

The analysis showed that all indirect effects through the targeted mediators were small and non-significant (eTable 1 and left panel of eFigure 1). These effects were robust to moderate levels of residual confounding (middle and right panels of eFigure 1). Although pain education was superior to placebo pain education in reducing the primary targets (catastrophization and maladaptive beliefs - eFigure 2), these hypothesised psychological mediators were not associated with pain at 3 months (eFigure 3). Pain education did not improve self-efficacy at week 1, a more distal mechanism which was associated with pain at 3 months.

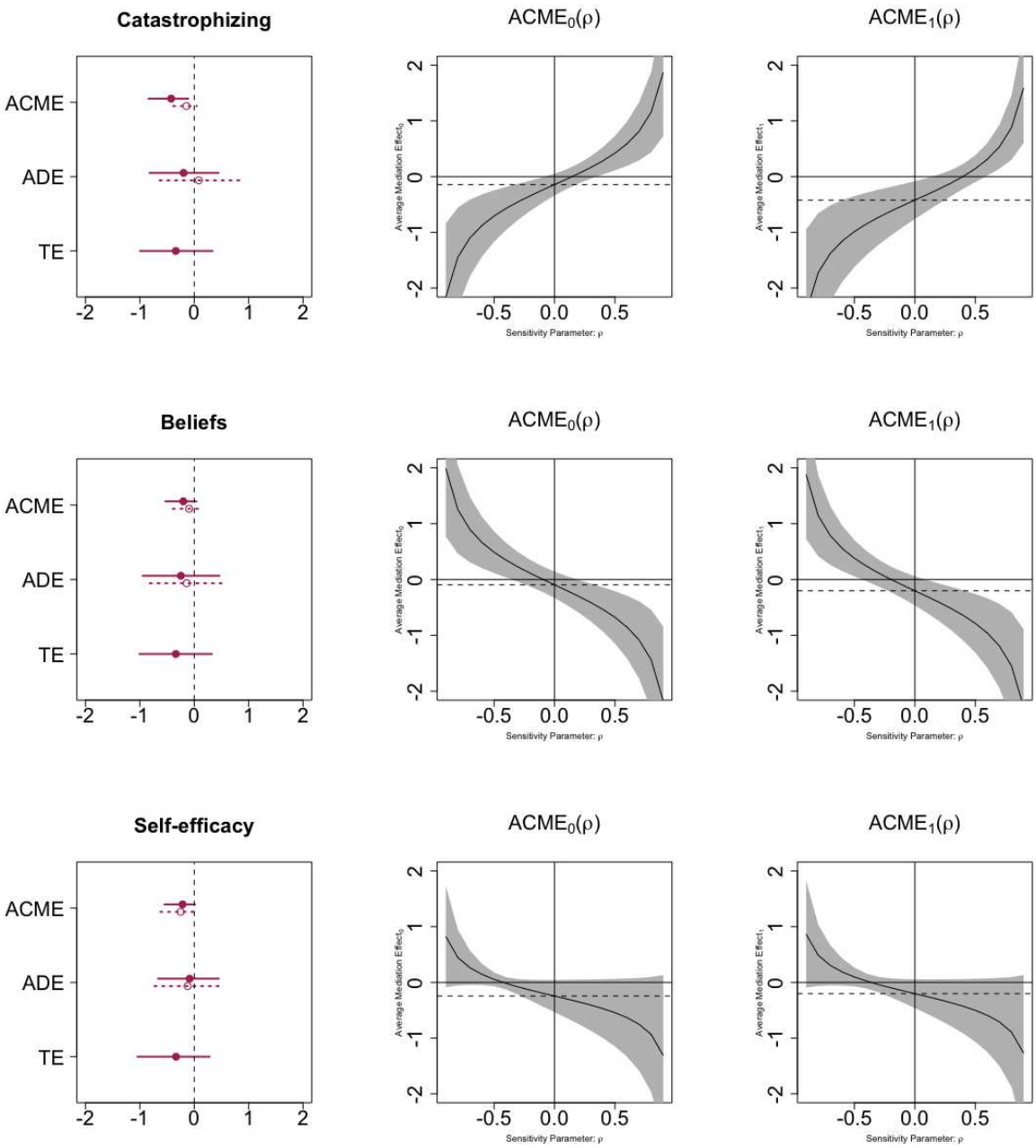
This process evaluation indicates that the intervention produced an effect on the key targeted mediators, but these mediators did not cause changes in the primary outcome. This suggests that psychological constructs (primarily catastrophization and beliefs) may not be worthwhile treatment targets for patients with acute low back pain.

## eTable 1. Results of Causal Mediation Analysis for Primary Outcome (Pain at 3 Months)

| | Intervention-mediator effect | Mediator-outcome effect | Natural indirect effect (ACME) | Natural direct effect (ADE) | Total effect |
|---|---|---|---|---|---|
| **Proximal mechanisms** | | | | | |
| Catastrophization | -4.62 (-7.39 to -1.86) | 0.03 (-0.25 to 3.41) | -0.28 (-0.56 to -0.08) | -0.06 (-0.71 to 0.62) | -0.34 (-0.99 to 0.34) |
| Beliefs | 3.36 (1.35 to 5.36) | -0.03 (-0.09 to 0.03) | -0.15 (-0.38 to 0.02) | -0.19 (-0.85 to 0.48) | -0.34 (-1.01 to 0.33) |
| **Distal mechanism** | | | | | |
| Self-efficacy | 2.97 (-0.28 to 6.21) | -0.08 (-0.12 to -0.05) | -0.23 (-0.55 to 0.02) | -0.10 (-0.68 to 0.44) | -0.34 (-1.05 to 0.28) |

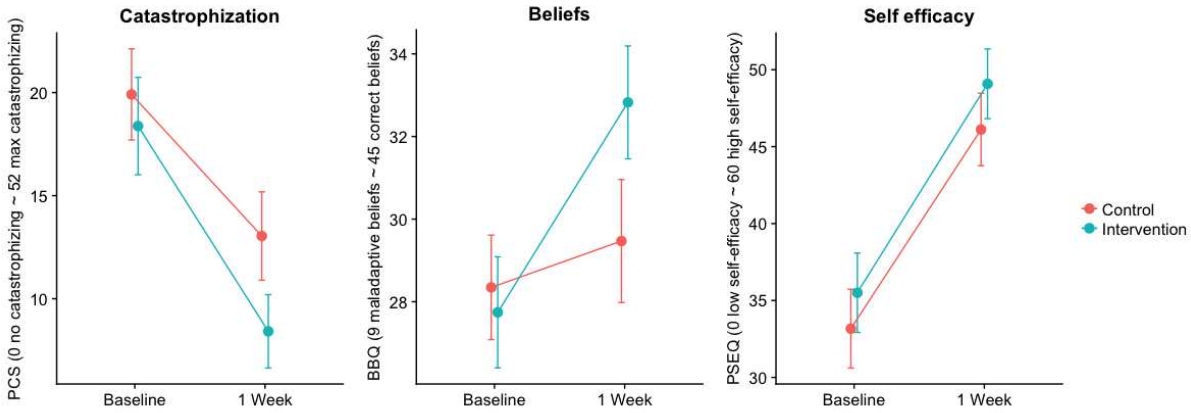
Effects are unadjusted coefficients with their 95% confidence intervals; ACME = average causal mediation effect; ADE = average direct effect

## eFigure 1. Sensitivity Analysis of Mediation Effects in the PREVENT Trial



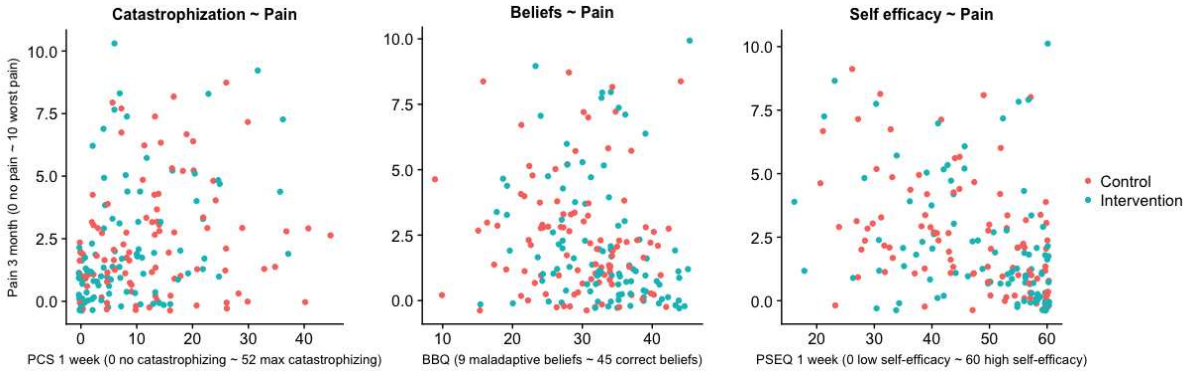

The effect decomposition (left panel) shows how the average effect of the treatment on the outcome - total effect (TE) is decomposed into the average causal mediation effect (ACME), and the average direct effect (ADE). These effects are presented as unstandardized effects with their 95% confidence intervals. The sensitivity plots (middle and right panel) show how much the estimated ACME would change if there was residual confounding of the mediator-outcome effect. The curved solid lines represent the estimated ACME for the control (middle panel) and pain education (right panel) groups at varied levels of residual confounding. The sensitivity parameter (horizontal axis) represents hypothesised levels of residual confounding: 0 indicates no residual confounding, and -1.0 and 1.0 are the maximum levels of residual confounding. The dashed horizontal line represents the estimated ACME when there is no residual confounding (ie. sensitivity parameter = 0).

## eFigure 2. Effects on targeted mediators in the PREVENT Trial




Mean (circles) and 95% confidence intervals (error bars) for primary treatment targets (catastrophization and beliefs) and secondary target (self-efficacy) at week 1 in Pain Education group (blue line) and Placebo Pain Education group (red line).

## eFigure 3. Scatter Plot of Targeted Mediators in the PREVENT Trial




Scatter plot of targeted mediators at week 1 (x-axis) and pain at 3 months (y-axis) stratified by treatment allocation [Pain Education group (blue) and Placebo Pain Education group (red)].

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## eResults 2. Out-of-Trial Therapy–Sensitivity Analysis

**Out-of-trial therapy – sensitivity analysis**
Out-of-trial therapy did not influence the effect of randomisation on primary outcome (pain). Out-of-trial therapy was measured by 'no healthcare visits' vs '1 or more healthcare visits' at 3-month follow-up. The natural direct effect from the mediation analysis (which represents the treatment effect that was not mediated through out-of-trial therapy over 3mo) was equivalent to the total effect of treatment. See eTable 2 below:

**eTable 2. Results of Sensitivity Analysis Evaluating Influence of Out-of-Trial Therapy on Primary Outcome Pain at 3 Months**

| | Mean difference (95% CI) | P Value |
|---|---|---|
| Average Direct Effect on pain (effect not mediated by out-of-trial therapy) | -0.2 (-0.9 to 0.5) | 0.61 |
| Total Effect on pain in sensitivity analysis | -0.3 (-0.9 to 0.4) | 0.48 |
| Total Effect on pain in primary analysis | -0.3 (-1.0 to 0.3) | 0.31 |

# Data Sharing Statement

Traeger. Effect of Intensive Patient Education vs Placebo Patient Education on Pain Outcomes in Patients With Acute Low Back Pain. *JAMA Neurol*. Published November 05, 2018. 10.1001/jamaneurol.2018.3376

**Data**
**Data available:** Yes
**Data types:** Data dictionary
**How to access data:** Data available on request from j.mcauley@neura.edu.au
**When available:** With publication

**Supporting Documents**
**Document types:** Statistical/analytic code
**How to access documents:** Statistical code available on request from j.mcauley@neura.edu.au
**When available:** With publication

**Additional Information**
**Who can access the data:** Available to researchers whose proposed use of the data has been approved.
**Types of analyses:** For pre-specified, approved purpose.
**Mechanisms of data availability:** Available after approval of a proposal