KNOW LOW BACK PAIN
## Development Committee

<table>
<thead>
<tr>
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<th>Role</th>
<th>Location</th>
</tr>
</thead>
<tbody>
<tr>
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</tbody>
</table>

*This program was sponsored by Pfizer Inc.*
Learning Objectives

• After completing this module, participants will be able to:
  – Discuss the prevalence of acute and chronic low back pain
  – Understand the impact of low back pain on patient functioning and quality of life
  – Use appropriate tools for the diagnosis of low back pain
  – Identify red and yellow flags that should trigger referral or further investigation
  – Explain underlying mechanisms of different types of low back pain
  – Select appropriate pharmacological and non-pharmacological strategies for the management of low back pain
# Table of Contents

- What is low back pain?
- How common is low back pain?
- How can the different types of low back pain be differentiated from each other in clinical practice?
- What red and yellow flags should trigger referral or additional investigations?
- How should low back pain be treated based on its pathophysiology?
What is low back pain?

- Pain below the costal margin and above the gluteal folds, with or without radiation to the lower extremity\(^1\)

- **Acute** vs. **chronic** low back is pain classified according to duration:
  - **Acute:** less than 3 months\(^2,3\)
  - **Chronic:** more than 3 months\(^2,3\)

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Discussion Question

How many patients suffering from low back pain do you see during a typical week?
Epidemiology of Low Back Pain

- >80% of adults experience back pain at some point in life
- Incidence is highest in third decade
- Overall prevalence increase with age until the age of 60–65 years
- Men and women are equally affected
- 5th leading reason for medical office visits
- 2nd most common reason (after respiratory illness) for symptom-related physician visits
- Most common cause of work-related disability

KNOW PAIN

A Practical Guide to Understanding, Assessing and Managing Pain
The Low Back Is the Most Common Site of Chronic Non-cancer Pain

*Based on physician survey

Percentage of Patients with Chronic Pain Complaining of Pain at Common Body Sites*

<table>
<thead>
<tr>
<th>Body Site</th>
<th>% of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Back</td>
<td>95%</td>
</tr>
<tr>
<td>Knee</td>
<td>49%</td>
</tr>
<tr>
<td>Neck</td>
<td>37%</td>
</tr>
<tr>
<td>Head</td>
<td>36%</td>
</tr>
<tr>
<td>Hip</td>
<td>34%</td>
</tr>
<tr>
<td>Shoulder</td>
<td>33%</td>
</tr>
</tbody>
</table>
Common Causes of Low Back Pain

Mechanical (80-90%)
(e.g., disc degeneration, fractured vertebrae, instability, unknown cause [most cases])

Neurogenic (5-15%)
(e.g., herniated disc, spinal stenosis, osteophyte damage to nerve root)

Non-mechanical spinal conditions (1-2%)
(e.g., neoplasm, infections, inflammatory arthritis, Paget’s disease)

Referred visceral pain (1-2%)
(e.g., gastrointestinal disease, kidney disease, abdominal aortic aneurism)

Other (2-4%)
(e.g., fibromyalgia, somatoform disorder, “faking” pain)

Pathophysiology of Low Back Pain

Chronic low back pain commonly have multiple potential mechanisms. This is called “mixed pain.”

Nociceptive pain
Most patients with acute non-specific low back pain (85%)

Central sensitization/ dysfunctional pain
May develop over time in some patients with chronic low back pain

Chronic low back pain commonly have multiple potential mechanisms. This is called “mixed pain.”

Neuropathic pain
Radiculopathy (7%)

Nociceptive and Neuropathic Components May Be Present in Low Back Pain

Neuropathic Component of Low Back Pain

• Neuropathic component of low back pain may be caused by:
  – Mechanical compression of nerve root (*mechanical neuropathic nerve root pain*)
  – Damage to sprouting C-fibers within the degenerated disc (*localized neuropathic pain*)
  – Action of inflammatory mediators released from the degenerated disc (*inflammatory neuropathic nerve root pain*), even without mechanical compression

Neuropathic Component of Chronic Low Back Pain

Up to 37% of patients with chronic low back pain may have a neuropathic component to their pain.

Recognizing Neuropathic Pain

Be alert for common verbal descriptors of neuropathic pain.

- Burning
- Tingling
- Shooting
- Electric shock-like
- Numbness

- Various neuropathic pain screening tools exist
- Tools rely largely on common verbal descriptors of pain, though some tools also include physical tests
- Tool selection should be based on ease of use
### Neuropathic Pain Screening Tools

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>LANSS</th>
<th>DN4</th>
<th>NPQ</th>
<th>painDETECT</th>
<th>ID Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pricking, tingling, pins and needles</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Electric shocks of shooting</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hot or burning</td>
<td></td>
<td></td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Numbness</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain evoked by light touching</td>
<td></td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td><strong>Clinical examination</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brush allodynia</td>
<td></td>
<td></td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Raised soft touch threshold</td>
<td></td>
<td></td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Altered pin prick threshold</td>
<td></td>
<td></td>
<td></td>
<td>X</td>
<td></td>
</tr>
</tbody>
</table>

Neuropathic pain screening tools rely largely on common verbal descriptors of pain.

Select tool(s) based on **ease of use** and **validation in the local language**.

Some screening tools also include bedside neurological examination.

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DN4 = Douleur Neuropathique en 4 Questions (DN4) questionnaire; LANSS = Leeds Assessment of Neuropathic Symptoms and Signs; NPQ = Neuropathic Pain Questionnaire

Discussion Question

HOW LONG DOES IT TAKE MOST OF YOUR PATIENTS TO RECOVER FROM LOW BACK PAIN?
Most cases of low back pain are:
- Acute
- Benign
- Self-limiting and tend to resolve over time

Management of Acute Low Back Pain

Clinical presentation: acute low back pain

History and examination

Red flags?

No

Consider differential diagnosis

Advise mobilization and avoidance of bed rest

Provide appropriate pain relief

Provide education and counsel on self-care

Review and assess improvement within 2 weeks

Yes

Investigation and management; consider referral

WHEN DO YOU REFER PATIENTS WITH ACUTE LOW BACK PAIN TO A SPECIALIST?
“Red Flags” Require Immediate Investigation and/or Referral

<table>
<thead>
<tr>
<th>Potential condition</th>
<th>Red flags</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Cancer</td>
<td>• Personal history of cancer</td>
<td>• Age &gt;50 years</td>
</tr>
<tr>
<td></td>
<td>• Weight loss</td>
<td></td>
</tr>
<tr>
<td>Infection</td>
<td>• Fever</td>
<td>• Recent infection</td>
</tr>
<tr>
<td></td>
<td>• Intravenous drug use</td>
<td></td>
</tr>
<tr>
<td>Fracture</td>
<td>• Osteoporosis</td>
<td>• Trauma</td>
</tr>
<tr>
<td></td>
<td>• Steroid use</td>
<td>• Older age</td>
</tr>
<tr>
<td>Focal neurologic deficit</td>
<td>• Progressive or disabling symptoms</td>
<td></td>
</tr>
<tr>
<td>Cauda equina syndrome</td>
<td>• Urinary retention</td>
<td>• Fecal incontinence</td>
</tr>
<tr>
<td></td>
<td>• Multilevel motor deficit</td>
<td>• Saddle anesthesia</td>
</tr>
</tbody>
</table>
Differential Diagnosis of Acute Low Back Pain

<table>
<thead>
<tr>
<th>Intrinsic Spine</th>
<th>Systemic</th>
<th>Referred</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Compression fracture</td>
<td>• Malignancy</td>
<td>• Gastrointestinal conditions</td>
</tr>
<tr>
<td>• Lumbar strain/sprain</td>
<td>• Infection (e.g., vertebral discitis/osteomyelitis)</td>
<td>(e.g., pancreatitis, peptic ulcer disease, cholecystitis)</td>
</tr>
<tr>
<td>• Herniated disc</td>
<td>• Connective tissue disease</td>
<td>• Pelvic conditions (e.g., endometriosis, pelvic inflammatory disease, prostatitis)</td>
</tr>
<tr>
<td>• Spinal stenosis</td>
<td>• Inflammatory spondyloarthropathy</td>
<td>• Retroperitoneal conditions</td>
</tr>
<tr>
<td>• Spondylolisthesis</td>
<td></td>
<td>(e.g., renal colic, pyelonephritis)</td>
</tr>
<tr>
<td>• Spondylolysis</td>
<td></td>
<td>• Herpes zoster</td>
</tr>
<tr>
<td>• Spondylosis (degenerative disc or facet joint)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

It is important to identify and treat the underlying causes of pain whenever possible!
Discussion Question

HOW FREQUENTLY DO YOU FOLLOW-UP WITH PATIENTS WHO PRESENT WITH ACUTE LOW BACK PAIN?
# Recommendations for Follow-Up of Patients with Acute Low Back Pain

<table>
<thead>
<tr>
<th>Patient Population</th>
<th>Frequency of Follow-up</th>
</tr>
</thead>
</table>
| All                                                     | • 2 weeks following initial visit  
• Follow-up options: telephone, e-mail or visit  
• Additional follow-up is indicated               |
| Patients considered at high risk for chronic pain*      | • Earlier and more frequent visits may be appropriate                                 |
| Older patients or patients with:                        |                                                                                      |
| • Progression of symptoms or lack of significant improvement |                                      
• Severe pain or functional deficit                  | • Earlier and more frequent reassessment may be appropriate                           |
| • Signs of nerve root disease or lumbar spinal stenosis |                                                                                      |
| Patients referred for spinal manipulation, acupuncture or massage | • After 4 visits, refer patient to a specialist to determine if functionality has improved |

*See yellow flags; may also want to consider populations at risk if pain persists in the presence of adequate treatment: children and adolescents, women <30 years, men >60 years, patients with specific comorbidities (e.g., diabetes) and immunocompromised or immunosuppressed patients

Follow-Up of Patients with Acute Low Back Pain

Follow-Up Protocol:

1. Review and assess improvement within 2 weeks.
   - No improvement or deterioration:
     - Assess risk of persistent disability:
       - Low risk:
         - Refer to physiotherapist
         - Review within 12 weeks
         - No improvement: consider referral to specialist
       - Medium risk:
         - Consider referral if there is severe, refractory radicular pain/neurological deficit
         - Refer to physiotherapist
         - Improvement: continue supportive management
       - High risk:
         - Refer for biopsychosocial assessment
         - Referral for specialist management
   - Improvement:
     - Continue current management

Discussion Question

IN YOUR PRACTICE, DO YOU REGULARLY ASSESS RISK FOR DEVELOPING CHRONIC PAIN? IF SO, HOW?
Patients at Risk of Developing Chronic Pain

Yellow flags are patient characteristics that can indicate long-term problems requiring greater attention by the physician, particularly in terms of returning to work.

- Pessimistic attitude toward pain, excessive fear of movement and activity and little hope for improvement
- Work-related problems (e.g., dissatisfaction, conflicts)
- Emotional problems (e.g., depression, anxiety, worry)
- Generalized pain (e.g., headache, fatigue, dizziness)
- Desire for passive treatment, little ability to be proactive
- Previous episodes of low back pain that were followed for an extended period of time

Laerum E et al. Tidsskr Nor Laegeforen 2010; 130(22):2248-51.
Management of Persistent Low Back Pain*

Persistent low back pain

Signs and symptoms of nerve root disease or spinal stenosis?

No

Re-evaluate symptoms and risk factors, review diagnosis and consider referral and/or imaging studies

Consider alternative therapy (e.g., interdisciplinary approach incorporating pharmacological and non-pharmacological elements)

Review response

Yes

Consider referral and/or diagnostic MRI

Nerve root compromise or spinal stenosis?

No

Refer for specialist management

Yes

*American College of Physicians and the American Pain Society
Multimodal Treatment of Low Back Pain

- Pharmacotherapy
- Stress management
- Interventional management
- Biofeedback
- Complementary therapies
- Education
- Lifestyle management
- Sleep hygiene
- Physical/occupational therapy

Discussion Question

WHAT NON-PHARMACOLOGICAL APPROACHES TO MANAGING LOW BACK PAIN DO YOU INCORPORATE INTO YOUR PRACTICE?

WHAT NON-PHARMACOLOGICAL MODALITIES YOUR PATIENTS REGULARLY ASK ABOUT?
## Non-pharmacological Treatments for Low Back Pain

<table>
<thead>
<tr>
<th>Moderate Evidence of Effectiveness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Therapy and exercise</td>
</tr>
<tr>
<td>Cognitive-behavioral therapy</td>
</tr>
<tr>
<td>Intensive multidisciplinary biopsychosocial rehabilitation</td>
</tr>
<tr>
<td>Massage</td>
</tr>
<tr>
<td>Yoga</td>
</tr>
<tr>
<td>Heat therapy</td>
</tr>
<tr>
<td>Medium-firm mattress</td>
</tr>
<tr>
<td>Transcutaneous electrical nerve stimulation</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Sufficient Evidence of Effectiveness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Function-centered treatment</td>
</tr>
<tr>
<td>Acupuncture</td>
</tr>
</tbody>
</table>

Evidence suggests bed rest and traction are **NOT** useful.

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Pharmacotherapy for Low Back Pain

• Treatment must balance patient expectations for pain relief and possible analgesic effect of therapy.
• Patients should be educated about the medication, treatment objectives and expected results.
• Psychosocial factors and emotional distress are stronger predictors of treatment outcome than physical examination findings or the duration and severity of pain.

Treatment of Inflammatory Pain

Pain treatment options
- Acetaminophen
- nsNSAIDs/coxibs
- Opioids
- Local anesthetics/channel blockers
- Triptans (for migraine)

Damaged tissue, inflammatory cells or tumor cells

Inflammatory chemical mediators

Changed responsiveness of nociceptors (peripheral sensitization)

Spinal cord

Brain

Changed responsiveness of neurons in central nervous system (central sensitization)

Descending modulation

Ascending input

Coxib = COX-2-specific inhibitor; nsNSAID = non-specific non-steroidal anti-inflammatory drug

Acetaminophen for Management of Low Back Pain

<table>
<thead>
<tr>
<th>Efficacy</th>
<th>Safety</th>
<th>Mechanism of Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Effective</td>
<td>• Favorable safety profile and low cost</td>
<td>• Unclear</td>
</tr>
<tr>
<td>• Efficacy improved by addition of nsNSAIDs or coxibs</td>
<td>• May cause liver damage at doses higher than 4 g/day</td>
<td></td>
</tr>
</tbody>
</table>

**Acetaminophen is the first-line option in acute and chronic low back pain.**

---

Coxib = COX-2-specific inhibitor; nsNSAID = non-selective non-steroidal anti-inflammatory drug
## nsNSAIDs/Coxibs for Management of Low Back Pain

<table>
<thead>
<tr>
<th>Efficacy</th>
<th>Safety</th>
<th>Mechanism of Action</th>
</tr>
</thead>
</table>
| • Effective  
• More effective than acetaminophen alone  
• Improved efficacy in combination with acetaminophen | • Gastrointestinal risk  
• Cardiovascular risk  
• Renal risk | • Block action of COX-2 enzyme, which is induced by inflammatory stimuli and results in increased production of prostaglandins  
• Coxibs specifically inhibit COX-2, while nsNSAIDs block action of COX-2 and COX-1 enzyme, which is involved in gastrointestinal cytoprotection and platelet activity |

First-line option in acute and chronic low back pain

CI = confidence interval; coxib = COX-2-specific inhibitor; nsNSAID = non-selective non-steroidal anti-inflammatory drug; RR = relative risk

Discussion Question

HOW DO YOU EVALUATE GASTROINTESTINAL AND CARDIOVASCULAR RISK IN PATIENTS FOR WHOM YOU ARE CONSIDERING PRESCRIBING A nsNSAID OR A COXIB?

Coxib = COX-2-specific inhibitor; nsNSAID = non-selective non-steroidal anti-inflammatory drug
nsNSAIDs/Coxibs and Cardiovascular Risk

Composite includes non-fatal myocardial infarction, non-fatal stroke, or cardiovascular death compared with placebo; chart based on network meta-analysis involving 30 trials and over 100,000 patients.

Coxib = COX-2 inhibitor; nsNSAID = non-selective non-steroidal anti-inflammatory drug

Risk Factors for Gastrointestinal Complications Associated with nsNSAIDs/Coxibs

ASA = acetylsalicylic acid; coxib = COX-2-specific inhibitor; GI = gastrointestinal; NSAID = non-steroidal anti-inflammatory drug; nsNSAID = non-selective non-steroidal anti-inflammatory drug; SSRI = selective serotonin reuptake inhibitor

Gastrointestinal Effects of nsNSAIDs/Coxibs Beyond the Upper Gastrointestinal Tract

• There is strong evidence to suggest potentially clinically relevant adverse gastrointestinal events are not limited to the upper gastrointestinal tract

• Studies suggest NSAIDs also increase the risk for lower* gastrointestinal clinical events

*Lower gastrointestinal means distal to the ligament of Treitz or fourth segment of the duodenum
Coxib = COX-2-specific inhibitor; GI = gastrointestinal; nsNSAID = non-selective non-steroidal anti-inflammatory drug
# Opioids for the Management of Low Back Pain

**Acute or chronic severe low back pain for short periods of time**

<table>
<thead>
<tr>
<th>Efficacy</th>
<th>Safety</th>
<th>Mechanism of Action</th>
</tr>
</thead>
</table>
| • Effective  
• Evidence insufficient to recommend one opioid over another  
• Efficacy enhanced by addition of acetaminophen and/or nsNSAIDs/coxibs | • Multiple side effects  
• Potential for abuse or addiction | • Alter limbic system activity  
• Modify sensory and affective pain aspects  
• Activate descending pathways that modulate transmission in spinal cord  
• Affect transduction of pain stimuli to nerve impulses |

Coxib = COX-2-specific inhibitor; nsNSAID = non-specific non-steroidal anti-inflammatory drug

Tramadol for the Management of Low Back Pain

• “Atypical” opioid analgesic
• Unique mechanism of action
  – Noradrenergic and serotonergic pathways
  – Opioid effect depends on conversion to active O-demethylated metabolite M1
• Weak binding affinity to mu opioid receptor
• Clinical studies of efficacy in low back pain
• Consider avoiding use in patients with diabetes due to potential for hypoglycemia

WHAT POTENTIAL SIDE EFFECTS DO YOU DISCUSS WITH PATIENTS FOR WHOM YOU ARE CONSIDERING PRESCRIBING AN OPIOID?
### Adverse Effects of Opioids

<table>
<thead>
<tr>
<th>System</th>
<th>Adverse effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrointestinal</td>
<td>Nausea, vomiting, constipation</td>
</tr>
<tr>
<td>CNS</td>
<td>Cognitive impairment, sedation, lightheadedness, dizziness</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Respiratory depression</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>Orthostatic hypotension, fainting</td>
</tr>
<tr>
<td>Other</td>
<td>Urticaria, miosis, sweating, urinary retention</td>
</tr>
</tbody>
</table>

*CNS = central nervous system*

Muscle Relaxants for Management of Low Back Pain

- Diverse group of drugs
- Mechanisms of action not clarified
- Use is controversial, mainly due to side effects and potential for abuse and dependency
- Guidelines do not universally recommend use of muscle relaxants in management of low back pain
- Provide short-term relief of low back pain
  - No differences in efficacy and safety
  - Very few short-term studies
  - No evidence supports long-term use or recommends one over the other

Mechanism-Based Pharmacological Treatment of Neuropathic Pain

Medications affecting peripheral sensitization:
- Capsaicin
- Local anesthetics
- TCAs

Medications affecting descending modulation:
- SNRIs
- TCAs
- Tramadol, opioids

Medications affecting central sensitization:
- $\alpha_2\delta$ ligands
- TCAs
- Tramadol, opioids

Nerve lesion/disease → Brain

SNRI = serotonin-norepinephrine reuptake inhibitor; TCA = tricyclic antidepressant

α2δ Ligands* for Management of Low Back Pain

Useful in combination with other treatments for low back pain with a neuropathic component

<table>
<thead>
<tr>
<th>Efficacy</th>
<th>Safety</th>
<th>Mechanism of Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Pregabalin + coxib combination is more effective than each drug used alone for management of chronic low back pain</td>
<td>• Most common side effects are dizziness and somnolence</td>
<td>• Bind to α₂δ subunit of calcium channel, which is upregulated in neuropathic pain</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Binding reduces neurotransmitter release and pain sensitization</td>
</tr>
</tbody>
</table>

*Gabapentin and pregabalin are α₂δ ligands
Coxib = COX-2-specific inhibitor
Antidepressants for Management of Low Back Pain

Useful in combination with other treatments for low back pain with a neuropathic component

<table>
<thead>
<tr>
<th>Efficacy</th>
<th>Safety</th>
<th>Mechanism of Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Not recommended for non-specific acute low back pain</td>
<td>• TCAs can cause cognitive disorders, confusion, gait disturbance and falls</td>
<td>• Inhibit reuptake of serotonin and norepinephrine, enhancing descending modulation</td>
</tr>
<tr>
<td>• May be considered for low back pain with a neuropathic component</td>
<td>• SNRIs are contraindicated in severe hepatic dysfunction or unstable arterial hypertension</td>
<td></td>
</tr>
</tbody>
</table>

TCA = tricyclic antidepressant; SNRI = serotonin norepinephrine reuptake inhibitor
## Therapies Not Recommended for Low Back Pain

<table>
<thead>
<tr>
<th>ASA</th>
<th>Benzodiazepines</th>
<th>Systemic Corticosteroids</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Insufficient evidence to permit recommendation of its use as an analgesic in patients with low back pain</td>
<td>• Risk of abuse, addiction and tolerance</td>
<td>• Oral or parenteral</td>
</tr>
</tbody>
</table>

ASA = acetylsalicylic acid

### Key Recommendations for Management of Acute Low Back Pain

<table>
<thead>
<tr>
<th>Level A (Consistent Evidence)</th>
<th>Level B (Inconsistent Evidence)</th>
<th>Level C (Consensus)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bed rest is <strong>not recommended</strong></td>
<td>Patient education is beneficial</td>
<td>Red flags are common but do not necessarily indicate serious pathology</td>
</tr>
<tr>
<td>nsNSAIDs/coxibs, acetaminophen and muscle relaxants are effective treatments for <strong>non-specific</strong> acute low back pain</td>
<td>Spine stabilization may reduce recurrence and need for health care services</td>
<td>Imaging is not indicated without findings suggestive of serious pathology</td>
</tr>
<tr>
<td>Spinal manipulation and chiropractic techniques are <strong>not recommended</strong></td>
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Coxib = COX-2 inhibitor; nsNSAID = non-steroidal anti-inflammatory drug
# Therapeutic Recommendations for Management of Low Back Pain

## Non-specific Low Back Pain

### Acute
- Acetaminophen
- nsNSAIDs/coxibs
  - Co-prescribe PPI for patients aged >45 years
- Weak opioids
- Muscle relaxants

## Radicular Pain

If radicular pain is prominent consider addition of:
- $\alpha^2\delta$ ligands
- TCAs

## Chronic

Refer to specialist for:
- Cognitive behavioral therapy
- Complex pharmacological management, including opioids and neuropathic pain medications
- Consider interventional pain therapies
- Consider surgery

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Coxib = COX-2-specific inhibitor; nsNSAID = non-selective non-steroidal anti-inflammatory drug; PPI = proton pump inhibitor; TCA = tricyclic antidepressant

Key Messages

• Most people suffer from low back pain at some point in their life

• 90% of the time low back pain is benign and self-limiting
  – “Yellow flags” may help identify individuals at risk for chronic pain

• “Red flags” requiring immediate action should be assessed in all patients presenting with low back pain

• Pain should be addressed using an interdisciplinary approach including patient education and non-pharmacological therapies
Key Messages (cont’d)

• Pharmacotherapy for acute low back pain may include acetaminophen, nsNSAIDs/coxibs, weak opioids and/or muscle relaxants
  – Addition of α2δ ligands or TCAs should be considered if radicular pain is present

• Patients with low back pain of longer duration should be assessed for neuropathic and central sensitization/ dysfunctional pain
  – These patients may require referral to a specialist

Coxib = COX-2-specific inhibitor; nsNSAID = non-selective non-steroidal anti-inflammatory drug; TCA = tricyclic antidepressant