KNOW LOW BACK PAIN

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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 extremity¹
- 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}



1. Airaksinen O *et al. Eur Spine J* 2006; 15(Suppl 2):S192-300; 2. International Association for the Study of Pain. *Unrelieved Pain Is a Major Global Healthcare Problem.* Available at: <u>http://www.iasp-pain.org/AM/Template.cfm?Section=Press_Release&Template=/CM/ContentDisplay.cfm&ContentID=2908</u>. Accessed: July 22, 2013. 3. National Pain summit Initiative. *National Pain Strategy: Pain Management for All Australians*. Available at: <u>http://www.iasp-pain.org/PainSummit/Australia_2010PainStrategy.pdf</u>. Accessed: July 22, 2013.

Discussion Question

HOW MANY PATIENTS SUFFERING FROM LOW BACK PAIN DO YOU SEE DURING A TYPICAL WEEK?

Epidemiology of Low Back Pain

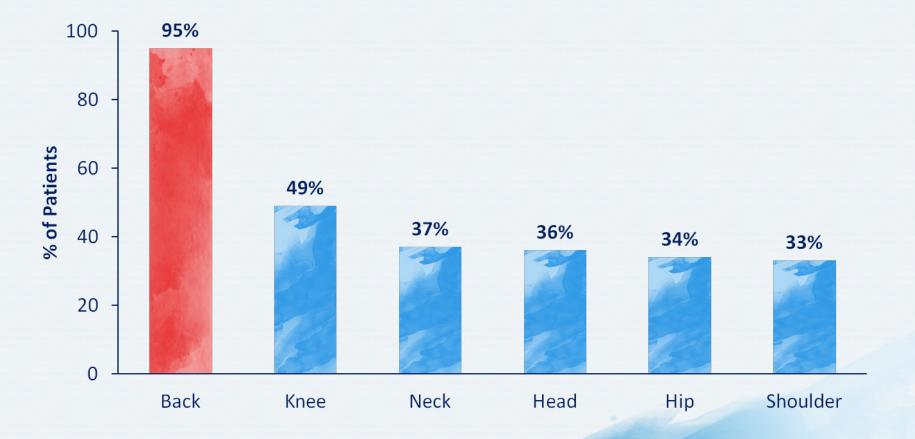
- >80% of adults experience back pain at some point in life¹
- Incidence is highest in third decade²
- Overall prevalence increase with age until the age of 60–65 years²
- Men and women are equally affected³
- **5th** leading reason for medical office visits⁴
- 2nd most common reason (after respiratory illness) for symptom-related physician visits⁴
- Most common cause of work-related disability⁵

 Walker BF. J Spinal Disord 2000; 13(3):205-17; 2. Hoy D et al. Best Pract Res Clin Rheumatol 2010; 24(6):769-813;
 Bassols A et al. Gac Sanit 2003; 17(2):97-107; 4. Hart LG et al. Spine (Phila PA 1976) 1995; 20(1):11-9; 5. National Institutes of Health. Low Back Pain Fact Sheet. Available at: http://www.ninds.nih.gov/disorders/backpain/detail_backpain.htm. Accessed: July 22, 2013.

A Practical Guide to Understanding, Assessing and Managing Pain

The Low Back Is the Most Common Site of Chronic Non-cancer Pain

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



*Based on physician survey

Boulanger A et al. Pain Res Manage 2007; 12(1):39-47.

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)

Cohen S. BMJ 2008; 337:a2718.

Pathophysiology of Low Back Pain

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

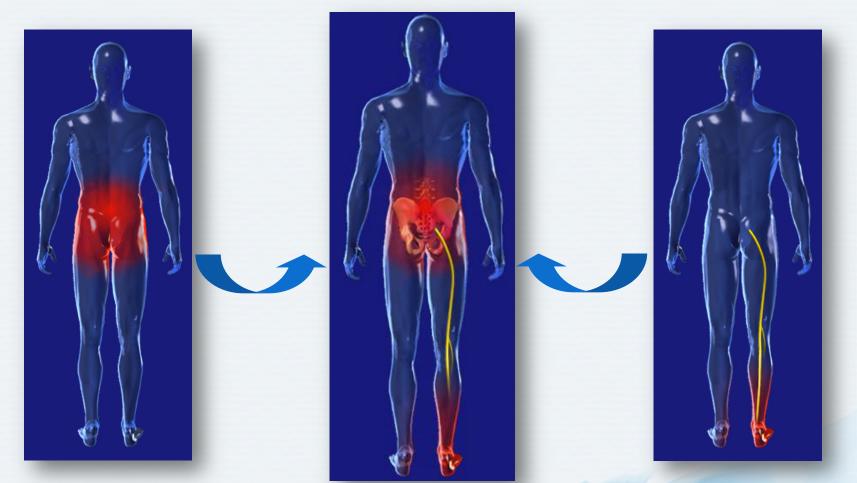
Chronic low back pain commonly have multiple potential mechanisms. This is called "mixed pain."

Neuropathic pain Radiculopathy (7%)

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

Manusov EG. *Prim Care* 2012; 39(3):471-9; Neblett R *et al. Pain* 2013; 14(5):438-45; Vellucci R. *Clin Drug Investig* 2012; 32(Suppl 1):3-10; Woolf CJ, Salter MW. *Science* 2000; 288(5472):1765-9.

Nociceptive and Neuropathic Components May Be Present in Low Back Pain



Nociceptive Component

Neuropathic Component

Freynhagen R, Baron R. Curr Pain Headache Rep 2009; 13(3):185-90.

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.

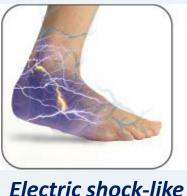




Tingling



Shooting





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

Baron R et al. Lancet Neurol. 2010; 9(8):807-19; Bennett MI et al. Pain 2007; 127(3):199-203; Gilron I et al. CMAJ 2006; 175(3):265-75.

Neuropathic Pain Screening Tools

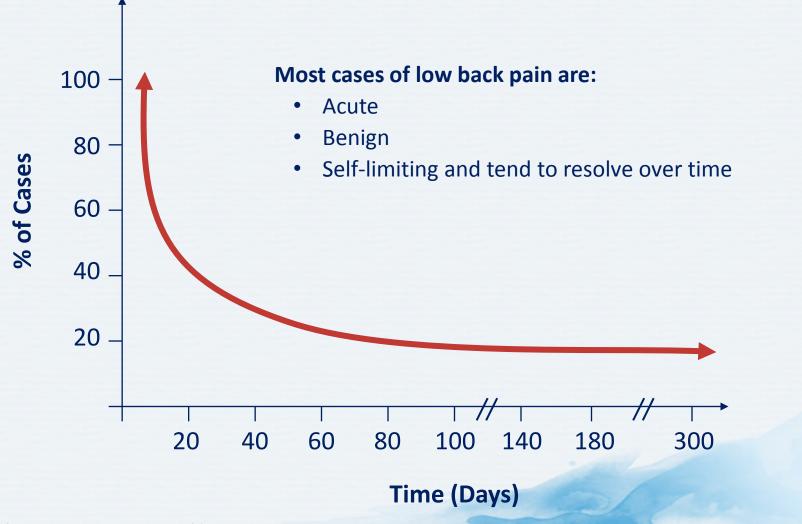
	LANSS	DN4	NPQ	painDETECT	ID Pain
Symptoms					
Pricking, tingling, pins and needles	x	x	x	X	X
Electric shocks of shooting					
Hot or burning	X	descriptors of pain			
Numbness		х	х	X	x
Pain Select tool(s) based on <i>ease of use</i> and validation in the local language					
Clinicar examination					
Brush allodynia		V		oning tools also	
Raised soft touch threshold		Some screening tools also include bedside neurological examination			
Altered pin prick threshold	Jx				
N4 = Douleur Neuropathique en 4 Questions (DN4) questionnaire; ANSS = Leeds Assessment of Neuropathic Symptoms and Signs; NPQ =	- Neuropathic Pa	in Question	naire	h the	

Bennett MI et al. Pain 2007; 127(3):199-203; Haanpää M et al. Pain 2011; 152(1):14-27.

Discussion Question

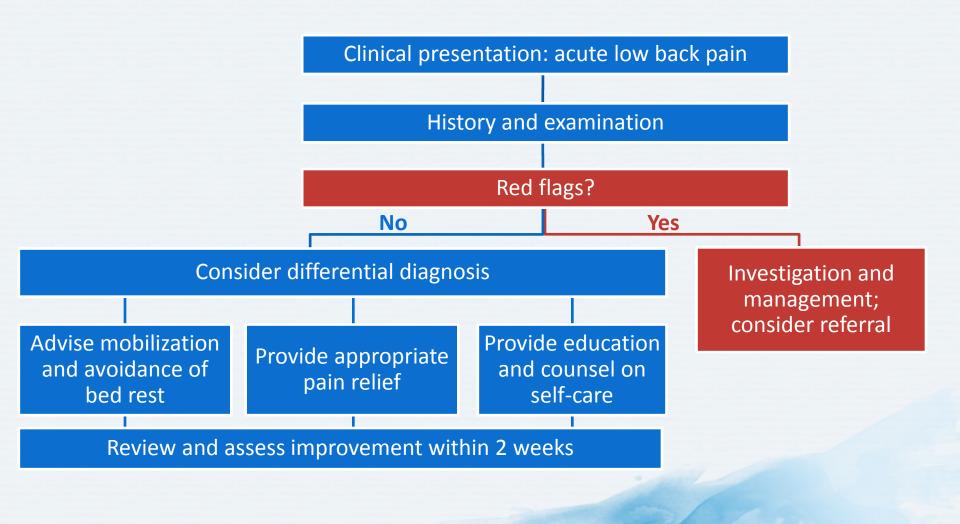
HOW LONG DOES IT TAKE MOST OF YOUR PATIENTS TO RECOVER FROM LOW BACK PAIN?

Natural History of Low Back Pain



Adapted from: Gunn CC et al. Spine 1980; 5(3):279-91.

Management of Acute Low Back Pain



Adapted from: Lee J et al. Br J Anaesth 2013; 111(1):112-20.

Discussion Question

WHEN DO YOU REFER PATIENTS WITH ACUTE LOW BACK PAIN TO A SPECIALIST?

"Red Flags" Require Immediate Investigation and/or Referral

Potential condition	Red flags	
Cancer	Personal history of cancerWeight loss	• Age >50 years
Infection	FeverIntravenous drug use	Recent infection
Fracture	OsteoporosisSteroid use	TraumaOlder age
Focal neurologic deficit	 Progressive or disabling symptoms 	
Cauda equina syndrome	Urinary retentionMultilevel motor deficit	Fecal incontinenceSaddle anesthesia

Differential Diagnosis of Acute Low Back Pain

Intrinsic Spine

- Compression fracture
- Lumbar strain/sprain
- Herniated disc
- Spinal stenosis
- Spondylolisthesis
- Spondylolysis
- Spondylosis (degenerative disc or facet joint

Systemic
Malignanov

- Malignancy
- Infection (e.g., vertebral discitis/osteomyelitis)
- Connective tissue disease
- Inflammatory spondyloarthropathy

Referred

- Gastrointestinal conditions (e.g., pancreatitis, peptic ulcer disease, cholecystitis)
- Pelvic conditions (e.g., endometriosis, pelvic inflammatory disease, prostatitis)
- Retroperitoneal conditions (e.g., renal colic, pyelonephritis)
- Herpes zoster

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

Casazza BA. Am Fam Physician 2012; 85(4):343-50.

Discussion Question

HOW FREQUENTLY DO YOU FOLLOW-UP WITH PATIENTS WHO PRESENT WITH ACUTE LOW BACK PAIN?

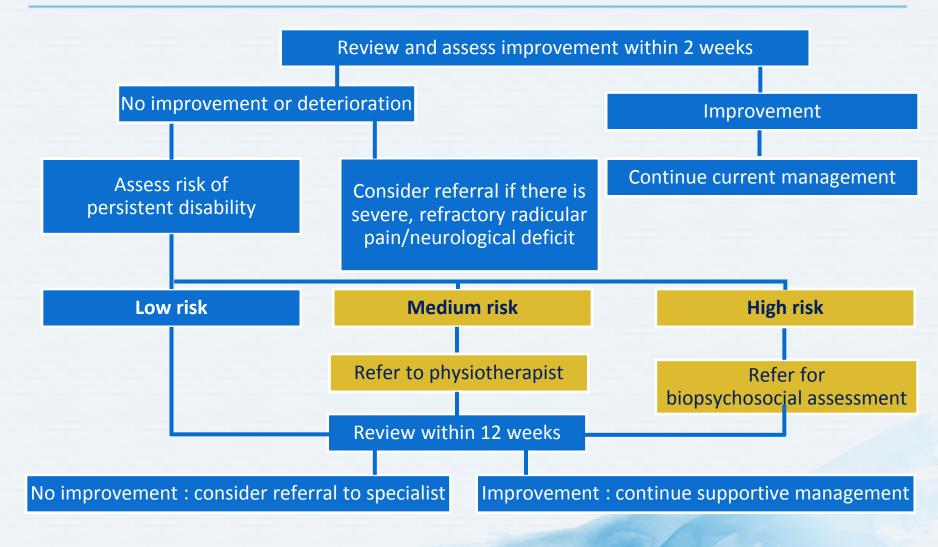
Recommendations for Follow-Up of Patients with Acute Low Back Pain

Patient Population	Frequency of Follow-up
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 Signs of nerve root disease or lumbar spinal stenosis 	 Earlier and more frequent reassessment may be appropriate
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

Ochoa G. In: Díaz Barriga JS, Gamarra AI (eds). *Libro Dolor Musculoesquelético*. Asociacion Colombiana para el Estudio del Dolor, ACED; Bogotá, Colombia: 2010; Savigny P *et al. Low Back Pain: Early Management of Persistent Non-specific Low Back Pain*. National Collaborating Centre for Primary Care and Royal College of General Practitioners; London, UK: 2009.

Follow-Up of Patients with Acute Low Back Pain



Adapted from: Lee J et al. Br J Anaesth 2013; 111(1):112-20.

Discussion Question

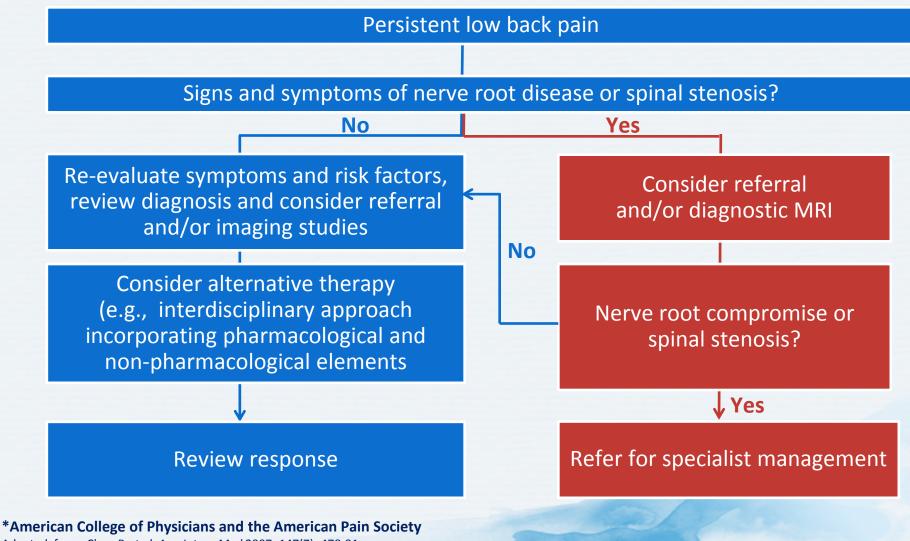
IN YOUR PRACTICE, DO YOU REGULARLY ASSESS RISK FOR DEVELOPING CHRONIC PAIN? IF SO, HOW?

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.

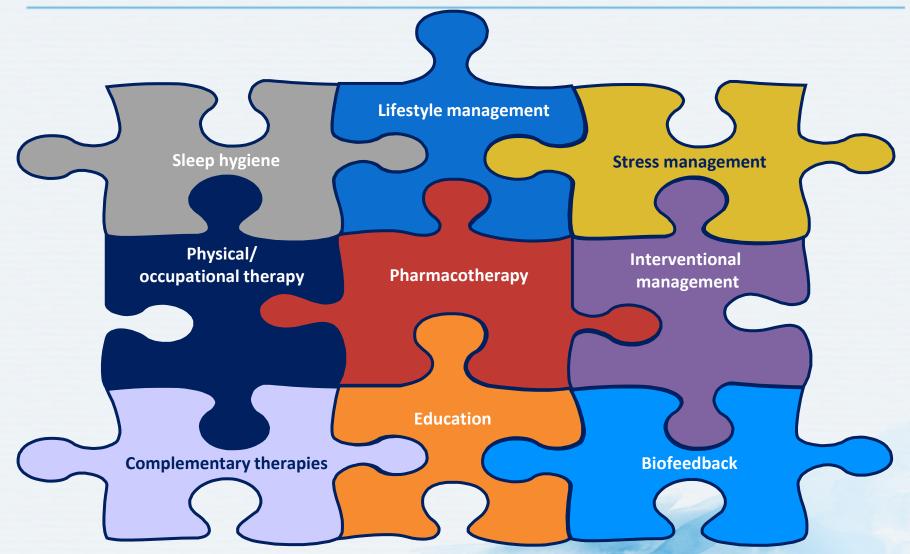
- Pessimistic attitude toward pain, excessive fear of movement and activity and little hope for improvement
- Work-related problems (e.g., dissatisfaction, conflicts)
- Emotional problems (e.g., depression, anxiety, worry)
- Generalized pain (e.g., headache, fatigue, dizziness)
- Desire for passive treatment,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

Management of Persistent Low Back Pain*



Adapted from: Chou R et al. Ann Intern Med 2007; 147(7): 478-91.

Multimodal Treatment of Low Back Pain



Gatchel RJ *et al. Psychol Bull* 2007; 133(4):581-624; Institute of Medicine. *Relieving Pain in America: A Blueprint for Transforming Prevention, Care, Education, and Research;* National Academies Press; Washington, DC: 2011; Mayo Foundation for Medical Education and Research. *Comprehensive Pain Rehabilitation Center Program Guide.* Mayo Clinic; Rochester, MN: 2006.

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

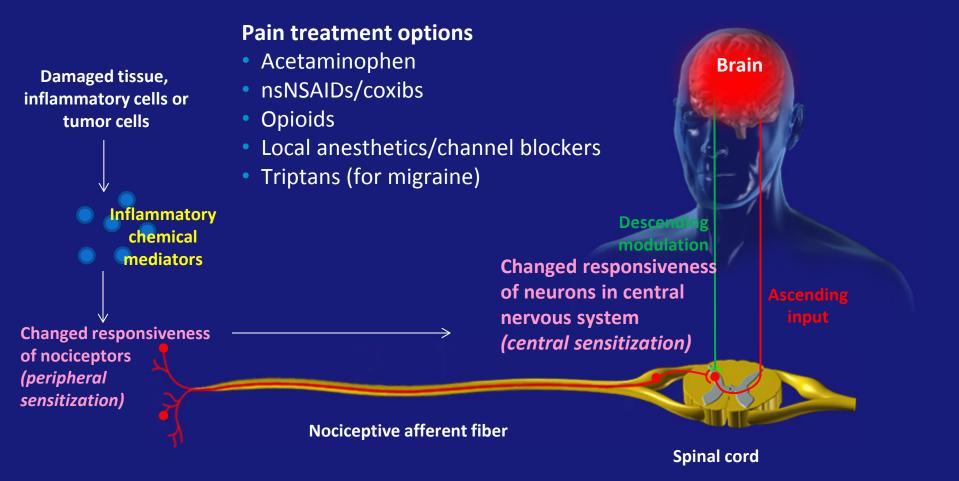
Moderate Evidence of Effect	tiveness		
Therapy and exercise	xercise Moderately effective in pain relief and functional improvement in adults with low back		
Cognitive-behavioral therapy	May reduce pain and disability in patients with chronic and subacute low back pain		
Intensive multidisciplinary biopsychosocial reh			
Massage	vidence suggests bed rest and and		
Yoga			
Heat therapy	traction are NOT useful ck pain		
Medium-firm mattr			
Transcutaneous electrical nerve stimulation	Controversial with evidence both for and against		
Sufficient Evidence of Effectiveness			
Function-centered treatmen	Function-centered treatment More effective than pain-centered treatment for an increase in days able to work in patients with subacute low back pain lasting more than 6 weeks		
Acupuncture	upuncture More effective than conventional therapy but not more effective than sham acupuncture		
Chou R <i>et al. Spine (Phila PA 1976)</i> 2009; 34(10):1066-77; Dagenais S <i>et al. Spine J</i> 2008; 8(1):203-12; Gay RE, Brault JS. Spine J 2008; 8(1):234-42; Hagen KB <i>et al. Spine (Phila PA 1976)</i> 2005; 30(5):542-6; Oleske D <i>et al. Spine 2007; 32</i> (19):2050-7; Pillastrini P <i>et al. Joint Bone Spine</i> 2012; 79(2):176-85; Ramos-Remus CR <i>et al. Curr Med Res Opin</i> 2004; 20(5):691-8; Romano CL <i>et al. Lotthon Traumatol</i> 2009; 10(4):185-91; Sakamoto C. Soen S. Digestion 2011; 83(1-2):108-23; Savigny P <i>et al. Low Back Pain; Early Management</i>			

20(5):691-8; Romanò CL *et al. J Orthop Traumatol* 2009; 10(4):185-91; Sakamoto C, Soen S. *Digestion* 2011; 83(1-2):108-23; Savigny P *et al. Low Back Pain: Early Management of Persistent Non-specific Low Back Pain*. National Collaborating Centre for Primary Care and Royal College of General Practitioners; London, UK: 2009; Toward Optimized Practice. *Guidelines for the Evidence-Informed Primary Care Management of Low Back Pain*. Edmonton, AB: 2009.

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



Acetaminophen for Management of Low Back Pain

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

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 Chou R *et al. Ann Intern Med* 2007; 147(7):505-14; Lee C *et al. Arthritis Rheum* 2004; 51(5):746-54; Lee J *et al. Br J Anaesth* 2013; 111(1):112-20; Mattia A, Coluzzi F. *Minerva Anestesiol* 2009; 75(11):644-53; Watkins PB *et al. JAMA* 2006; 296(1):87-93.

nsNSAIDs/Coxibs for Management of Low Back Pain

Efficacy	Safety	Mechanism of Action
 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

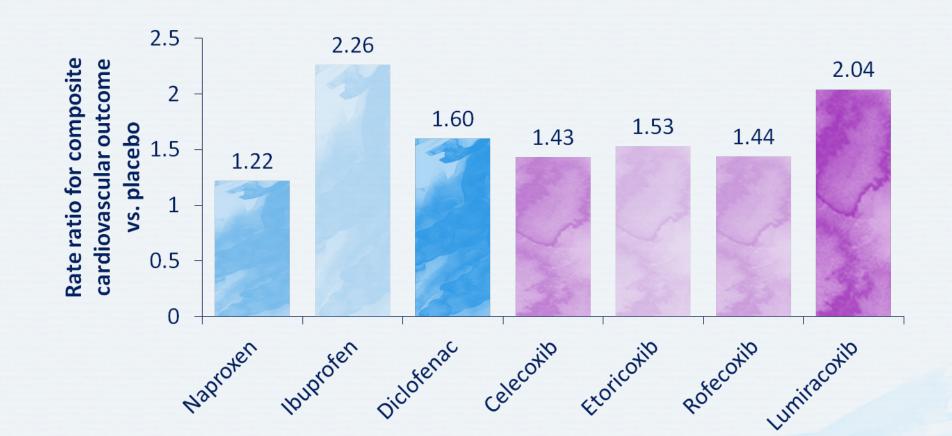
Chou R et al. Ann Intern Med 2007; 147(7):505-14; Lee J et al. Br J Anaesth 2013; 111(1):112-20; Schnitzer TJ et al. J Pain Symptom Manage 2004; 28(1):72-95; van Tulder M et al. Cochrane Database Syst Rev 2000; 2:CD000396; Vane JR, Botting RM. Inflamm Res 1995;44(1):1-10.

Discussion Question

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

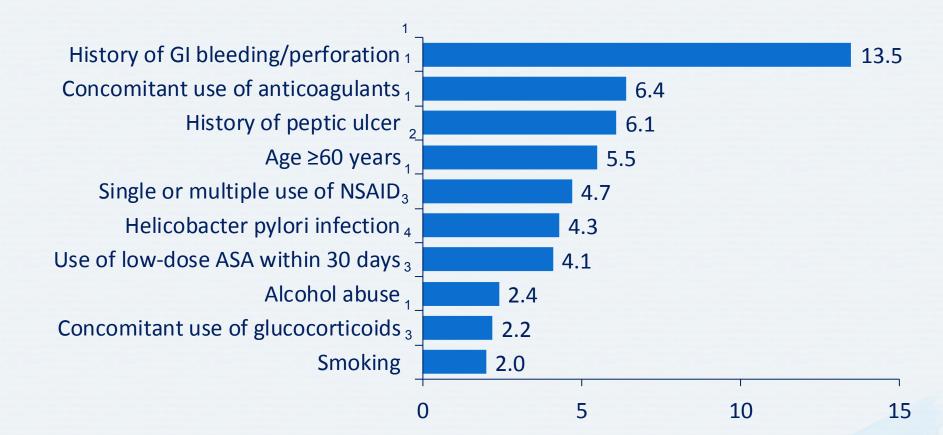
Coxib = COX-2-specific inhibitor; nsNSAID = non-selective non-steroidal anti-inflammatory drug

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 Trelle S *et al. BMJ* 2011; 342:c7086.

Risk Factors for Gastrointestinal Complications Associated with nsNSAIDs/Coxibs



Odds ratio/relative risk for ulcer complications

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

1. Garcia Rodriguez LA, Jick H. Lancet 1994; 343(8900):769-72; 2. Gabriel SE et al. Ann Intern Med 1991; 115(10):787-96;

3. Bardou M. Barkun AN. Joint Bone Spine 2010; 77(1):6-12; 4. Garcia Rodríguez LA, Hernández-Díaz S. Arthritis Res 2001; 3(2):98-101.

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** llison MC *et al. N Engl J Med* 1992; 327(11):749-54; Lanas A, Sopeña F. *Gastroenterol Clin N Am* 2009; 38(2):333-53; Fujimori S *et al. Gastro Endoscopy* 2009; 69(7):1339-46; Laine L *et al. Gastroenterology* 2003; 124(2):288-92; Chan FK *et al. N Engl J Med* 2002; 347(26):2104-10.

Opioids for the Management of Low Back Pain

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

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

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

Chou R *et al. J Pain Symptom Manage* 2003; 26(5):1026-48; Chou R *et al. J Pain* 2009; 10(2):113-30; Furlan AD *et al. CMAJ* 2006; 174(11):1589-94; Kalso E *et al. Pain* 2004; 112(3):372-80; Lee J *et al. Br J Anaesth* 2013; 111(1):112-20; Martell BA *et al. Ann Intern Med* 2007; 146(2):116-27; Rauck RL *et al. J Opioid Manag* 2006; 2(3):155-66; Reisine T, Pasternak G. In: Hardman JG *et al* (eds). *Goodman and Gilman's: The Pharmacological Basics of Therapeutics.* 9th ed. McGraw-Hill; New York, NY: 1996; Scholz J, Woolf CJ. *Nat Neurosci* 2002; 5(Suppl):1062-7; Trescot AM *et al. Opioid Pharmacol Pain Phys* 2008; 11(2 Suppl):S133-53.

Tramadol for the Management of Low Back Pain

- "Atypical" opioid analgesic
- Unique mechanism of action
 - Noradrenergic and serotoninergic 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

Baer P *et al. Can J Diagnosis* 2010; 27(10):43-50; Deshpande A et al. *Cochrane Database Syst Rev* 2007; 3:CD004959; Janssen Pharmaceuticals Inc. *Tramadol Hydrochloride Tablets Full Prescribing Information*. Titusville, NJ: 2013; Jonville-Bera A *et al. Therapie* 2010; 65(5):499-500; Schofferman J, Mazanec D. *Spine J* 2008; 8(1):185-94; Taugourdeau S *et al. Rev Med Interne* 2011; 32(11):703-5; Vorsanger GJ *et al. J Opioid Manag* 2008; 4(2):87-97.

Discussion Question

WHAT POTENTIAL SIDE EFFECTS DO YOU DISCUSS WITH PATIENTS FOR WHOM YOU ARE CONSIDERING PRESCRIBING AN OPIOID?

Adverse Effects of Opioids

System	Adverse effects
Gastrointestinal	Nausea, vomiting, constipation
CNS	Cognitive impairment, sedation, lightheadedness, dizziness
Respiratory	Respiratory depression
Cardiovascular	Orthostatic hypotension, fainting
Other	Urticaria, miosis, sweating, urinary retention

CNS = central nervous system

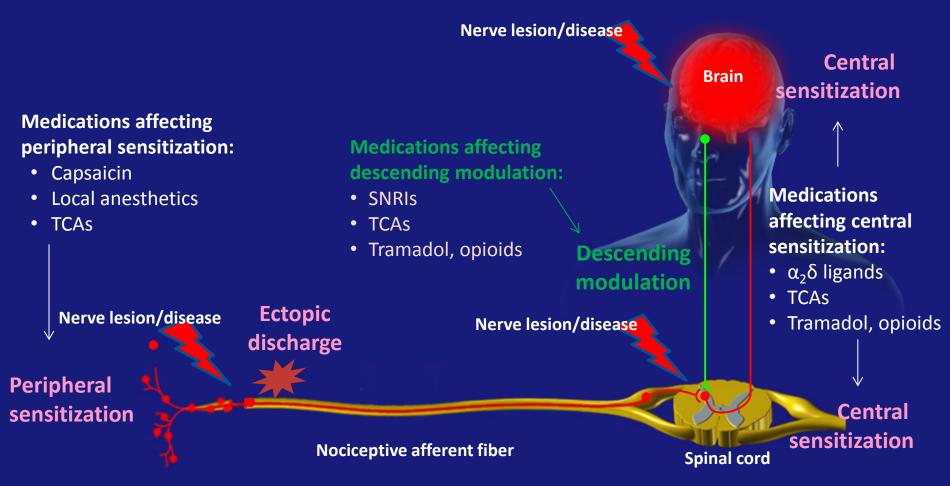
Moreland LW, St Clair EW. *Rheum Dis Clin North Am* 1999; 25(1):153-91; Yaksh TL, Wallace MS. In: Brunton L *et al* (eds). *Goodman and Gilman's The Pharmacological Basis of Therapeutics*. 12th ed. (online version). McGraw-Hill; New York, NY: 2010.

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

Chou R et al. J Pain Symptom Manage 2004; 28(2):140-75; van Tulder MW et al. Spine (Phila PA 1976) 2003; 28(17):1978-92.

Mechanism-Based Pharmacological Treatment of Neuropathic Pain



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

Adapted from: Attal N *et al. Eur J Neurol* 2010; 17(9):1113-e88; Beydoun A, Backonja MM. *J Pain Symptom Manage* 2003; 25(5 Suppl):S18-30; Jarvis MF, Boyce-Rustay JM. *Curr Pharm Des* 2009; 15(15):1711-6; Gilron I *et al. CMAJ* 2006; 175(3):265-75; Moisset X, Bouhassira D. NeuroImage 2007; 37(Suppl 1):S80-8; Morlion B. Curr Med Res Opin 2011; 27(1):11-33; Scholz J, Woolf CJ. Nat Neurosci 2002; 5(Suppl):1062-7.

α2δ Ligands* for Management of Low Back Pain

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

Efficacy	Safety	Mechanism of Action
 Pregabalin + coxib combination is more 	 Most common side effects are dizziness 	• Bind to $\alpha_2 \delta$ subunit of calcium channel, which is upregulated
effective than each drug	and somnolence	in neuropathic pain
used alone for management of chronic		 Binding reduces neurotransmitter release and
low back pain		pain sensitization

*Gabapentin and pregabalin are $\alpha_2 \delta$ ligands Coxib = COX-2-specific inhibitor

Attal N, Finnerup NB. *Pain Clinical Updates* 2010; 18(9):1-8; Bauer CS *et al. J Neurosci* 2009; 29(13):4076-88; Chou R *et al. Ann Intern Med* 2007; 147(7):505-14; Lee J *et al. Br J Anaesth* 2013; 111(1):112-20; Romanó C *et al. J Orthop Traumatol* 2009; 10(4):185.

Antidepressants for Management of Low Back Pain

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

icacy	1

- Not recommended for non-specific acute low back pain
- May be considered for low back pain with a neuropathic component

Safety

- TCAs can cause cognitive disorders, confusion, gait disturbance and falls
- SNRIs are contraindicated in severe hepatic dysfunction or unstable arterial hypertension

Mechanism of Action

 Inhibit reuptake of serotonin and norepinephrine, enhancing descending modulation

TCA = tricyclic antidepressant; SNRI = serotonin norepinephrine reuptake inhibitor Attal N, Finnerup NB. *Pain Clinical Updates* 2010; 18(9):1-8; Lee J *et al. Br J Anaesth* 2013; 111(1):112-2; Skljarevski V *et al. Eur J Neurol* 2009; 16(9):1041-8; Verdu B *et al. Drugs* 2008; 68(18):2611-32.

Therapies Not Recommended for Low Back Pain

ASA	Benzodiazepines	Systemic Corticosteroids
Insufficient evidence to permit recommendation	 Risk of abuse, addiction and tolerance 	Oral or parenteralNo more effective
of its use as an analgesic in patients with low back pain		than placebo

ASA = acetylsalicylic acid

Arbus L *et al. Clin Trials J* 1990; 27:258-67; Chou R *et al. Ann Intern Med* 2007; 147(7):505-14; Derry S *et al. BMJ* 2000; 321(7270):1183-7; Evans DP *et al. Curr Med Res Opin* 1980; 6(8):540-7; Finckh A *et al. Spine (Phila PA 1976).* 2006; 31(4):377-81; Friedman BW *et al. J Emerg Med* 2006; 31(4):365-70; Haimovic IC, Beresford HR. *Neurology* 1986; 36(12):1593-4; Medina Santillán R *et al. Proc West Pharmacol Soc* 2000; 43:69-70.

Key Recommendations for Management of Acute Low Back Pain

Level A	Level B	Level C
(Consistent Evidence)	(Inconsistent Evidence)	(Consensus)
 Bed rest is not recommended nsNSAIDs/coxibs,	 Patient education is beneficial Spine stabilization may reduce	 Red flags are common but do
acetaminophen and	recurrence and need for	not necessarily indicate
muscle relaxants are effective	health care services Spinal manipulation and	serious pathology Imaging is not indicated
treatments for non-specific	chiropractic techniques are	without findings suggestive of
acute low back pain	not recommended	serious pathology

Therapeutic Recommendations for Management of Low Back Pain

	Non-specific Low Back Pain	Radicular Pain
Acute	 Acetaminophen nsNSAIDs/coxibs Co-prescribe PPI for patients aged >45 years Weak opioids Muscle relaxants 	 If radicular pain is prominent consider addition of: α²δ ligands TCAs
Chronic	 Refer to specialist for: Cognitive behavioral therapy Complex pharmacological management, including opioids and neuropathic pain m Consider interventional pain therapies Consider surgery 	edications

Coxib = COX-2-specific inhibitor; nsNSAID = non-selective non-steroidal anti-inflammatory drug; PPI = proton pump inhibitor; TCA = tricyclic antidepressant Adapted from: Lee J *et al. Br J Anaesth* 2013; 111(1):112-20.



- 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 nonpharmacological therapies

Key Messages (cont'd)

- Pharmacotherapy for acute low back pain may include acetaminophen, nsNSAIDs/coxibs, weak opioids and/or muscle relaxants
 - Addition of $\alpha 2\delta$ 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