

PATHOPHYSIOLOGICAL ASSESSMENT OF NON-SPECIFIC BACK PAIN





BACK PAIN CAN HAVE A CLEAR UNDERLYING PATHOLOGY

Various clearly defined pathologies can be associated with back pain, including autoimmune disorders, spinal infections, or osteoporotic fractures. These pathologies have relatively clear signs and symptoms and well-defined diagnostic work-ups that ideally lead to a definite diagnosis and targeted treatment.

PATHOLOGY IS UNCLEAR IN ~90% OF BACK PAIN PATIENTS

Nevertheless, back pain in at least 90% of patients cannot be attributed to any specific pathology [6]. As a consequence, diagnostic labels are descriptive in nature, e.g. 'Low back pain, unspecified' (ME.84.2Z in the ICD-11). In many research studies, the description is equally vague (e.g. 'non-specific low back pain').



THIS LARGE PROPORTION IS UNLIKELY A HOMOGENOUS GROUP

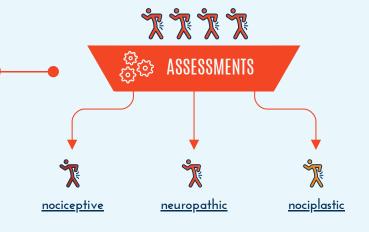


It is highly unlikely that this large category of patients constitutes a similarly unified category as e.g., the autoimmune disorder ankylosing spondylitis. The important question becomes which pathophysiological mechanisms contribute to back pain in this category and how such mechanisms can be identified when assessing individual patients.

PATIENT ASSESSMENT ALLOWS BROAD PATHOPHYSIOLOGICAL CATEGORIZATION

Not all mechanisms are currently known nor can they necessarily be assessed in humans. Nevertheless, broad pathophysiological categories can, at least partly, already be inferred at this point from patient assessment.

Like anywhere in the body, pain in the back can be ascribed to three fundamental pathophysiological categories: nociceptive, neuropathic and nociplastic pain [4], which are not mutually exclusive.



ASSESSMENT SUMMARY

ROUTINE CLINICAL ASSESSMENTS

ADDITIONAL OPTIONS

to better understand pathophysiology (not exhaustive)

History taking/ clinical exam

Pain drawings

Questionnaires

Laboratory tests

Imaging, including
Magnetic Resonance Imaging
(MRI)

Quantitative Sensory Testing (QST)
[8]

























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Let's consider three patients: Alex, Billy, and Sam. They are between 45 and 55 years of age, have had low back pain for six months (i.e. they have chronic pain per definition [10]) and they have recently received MR imaging of the lumbar spine (albeit this might not be in line with recommendations [2,5]). For all three, the MRI shows mild disc degeneration and mild facet joint degeneration at L3/4 and L4/5 without nerve root compression or Modic changes. The GP now sends the three patients to your office for further assessment. How will you find out to which pathophysiological category their pain predominantly belongs?

Alex complains about persistent, burning pain in the lower lumbar region extending paravertebrally and into the buttock in a diffuse pattern. No radiation to the lower limbs. Movement does not aggravate the pain, but sometimes, Alex feels some itching when wearing tight belts or pants.

Clinical examination:

- no sensory or motor deficits
- range of motion (ROM) somewhat reduced in lumbar flexion and extension
- no clear mechanical pattern of pain aggravation by movement
- local tenderness upon palpation in the midline at the level L4/L5

No clear mechanical pattern, but burning pain and itchiness... neuropathic pain?



Neuropathic pain questionnaire, e.g. the Neuropathic Pain Symptom Inventory (NPSI) [1]

• Alex scores 63 points on the weighted NPSI [9]



- **QST** QST @back: increased mechanical detection threshold, reduced pressure pain threshold, mechanical allodynia and hyperalgesia. No alterations in thermal detection or thermal pain thresholds, relative to normative data
 - QST @hand (control area): all tests are normal [8]

Classical signs of central sensitization (mechanical allodynia and hyperalgesia in a

secondary zone, i.e., the skin) and of nerve

damage (increased mechanical detection

threshold). Nevertheless, neuropathic pain

cannot be definitely diagnosed because a

lesion or disease of the somatosensory system

is not confirmed in Alex [3]. Also, the signs

and symptoms cannot be ascribed to a

specific nerve territory or dermatome.

neuropathic component

Evidence for a



Billy suffers from episodic, right-sided paraspinal and right buttock pain, aggravated by movement and standing for a long time and relieved by sitting and walking. Billy denies pain at rest but after intense physical activity s/he feels stiff the next morning for 20-30 minutes.

Clinical examination:

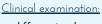
- symptoms can be provoked with extension and rotation of the lumbar
- local tenderness over L4/L5 facet joints and musculature

Symptoms provoked by segmental movements... nociceptive pain?



Detailed | clinical assessment according to Vining and colleagues [11] to confirm the impression and identify the likely nociceptive source

 history taking and clinical exam (3 or more of: > age 50, relief by relief by walking, paraspinal positive onset, extension-rotation test [11]) point to the facet joint as the most likely source of pain



waking him/her up at night.

diffuse tenderness over lumbar spinous processes and paraspinal muscles

Sam reports pain fluctuating in intensity and

location, sometimes extending to the buttocks

and left or right posterior thigh. The pain gets

worse with movement, but Sam also

experiences pain sometimes at rest, sometimes

- lumbar movement is painful in the endrange in all directions and ROM is somewhat restricted
- no sign of sensory or motor impairment

Pain at rest, waking up at night... inflammatory component?



√) Blood test

negative for inflammatory markers

Pain extending in the buttock and legs... neuropathic component?



• NPSI weighted score is 48, i.e., inconclusive [9]

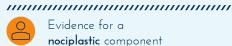


- **QST** QST @painful sites (back and leg): reduced pain thresholds for all modalities, increased pain sensitivity and normal detection thresholds. No dynamic mechanical allodynia [7,8]
 - QST @hand: similar pattern of hypersensitivities, but to a lesser extent



Evidence for a nociceptive component

No further assessments are needed because Billy most likely suffers from nociceptive pain. It is possible that there is an inflammatory component associated with the degenerative changes that result in the movement-associated allodynia. However, there is no indication to suspect systemic inflammation.



Evidence for a nociplastic component

There is no clear evidence for an inflammatory or neuropathic component. A potential nociceptive component remains unclear (because of the movement-related pain), but the exam according to Vining and colleagues [11] does not allow a clear classification. The widespread hypersensitivity (at the hand in addition to the back and leg), the increased spatial pain extent and potentially the fluctuating character point towards nociplastic pain.

OVERALL CONCLUSION

Alex, Billy and Sam represent relatively clear examples of pathophysiological pain categories. In reality, an individual's pain might of course arise from a mix of different pathophysiologies. In addition, each category ought to be comprised of different mechanisms, which themselves are again the product of different sub-mechanisms. It is currently unclear in how much detail pathophysiology has to be understood to be most relevant for treatment; this will also depend at which mechanism treatment is targeted. Nevertheless, contributions of different pathophysiologies to an individual's 'non-specific' back pain can be identified with assessment methods that are already available. The ultimate goal should be to get rid of the unfortunate diagnostic label of 'non-specific' back pain and, by better understanding of pathophysiological mechanisms, to develop and promote more targeted treatment in the future.





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AUTHORS

Petra Schweinhardt, MD, PhD *

Integrative Spinal Research
Department of Chiropractic Medicine
Balgrist University Hospital
University of Zurich
Zurich, Switzerland

University of Zurich Zurich, Switzerland

Luana Nyiroe, DCM

Integrative Spinal Research
Department of Chiropractic Medicine
Balgrist University Hospital
University of Zurich
Zurich, Switzerland

University of Zurich Zurich, Switzerland

Mirjam Baechler, DC, MMEd

Integrative Spinal Research
Department of Chiropractic Medicine
Balgrist University Hospital
University of Zurich
Zurich, Switzerland

University of Zurich Zurich, Switzerland

Laura Sirucek, MSc

Integrative Spinal Research
Department of Chiropractic Medicine
Balgrist University Hospital
University of Zurich
Zurich, Switzerland

University of Zurich Zurich, Switzerland

Susanne Becker, PhD

Integrative Spinal Research
Department of Chiropractic Medicine
Balgrist University Hospital
University of Zurich
Zurich, Switzerland

University of Zurich Zurich, Switzerland

Department of Cognitive and Clinical Neuroscience Medical Faculty Mannheim Central Institute of Mental Health Heidelberg University Mannheim, Germany

 * corresponding author: petra.schweinhardt@balgrist.ch

REVIEWERS

Owen D Williamson, FRCSC Pain Medicine Adjunct Professor School of Interactive Arts and Technology

Simon Fraser University Surrey, BC, Canada Thomas Graven-Nielsen, DMSc, PhD

Center for Neuroplasticity and Pain (CNAP) Aalborg University, Denmark



