

# The etiology of low back pain



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# Conflict of interest

I have no consultancy or commercial relationship to any commercial (implant) company

- in any way related to this lecture,
- nor to any of the implants shown in the slides,
- nor to my visit to Brasil.

The travelcosts were paid by Congresso de Cirurgia.

I receive no speakers fee.

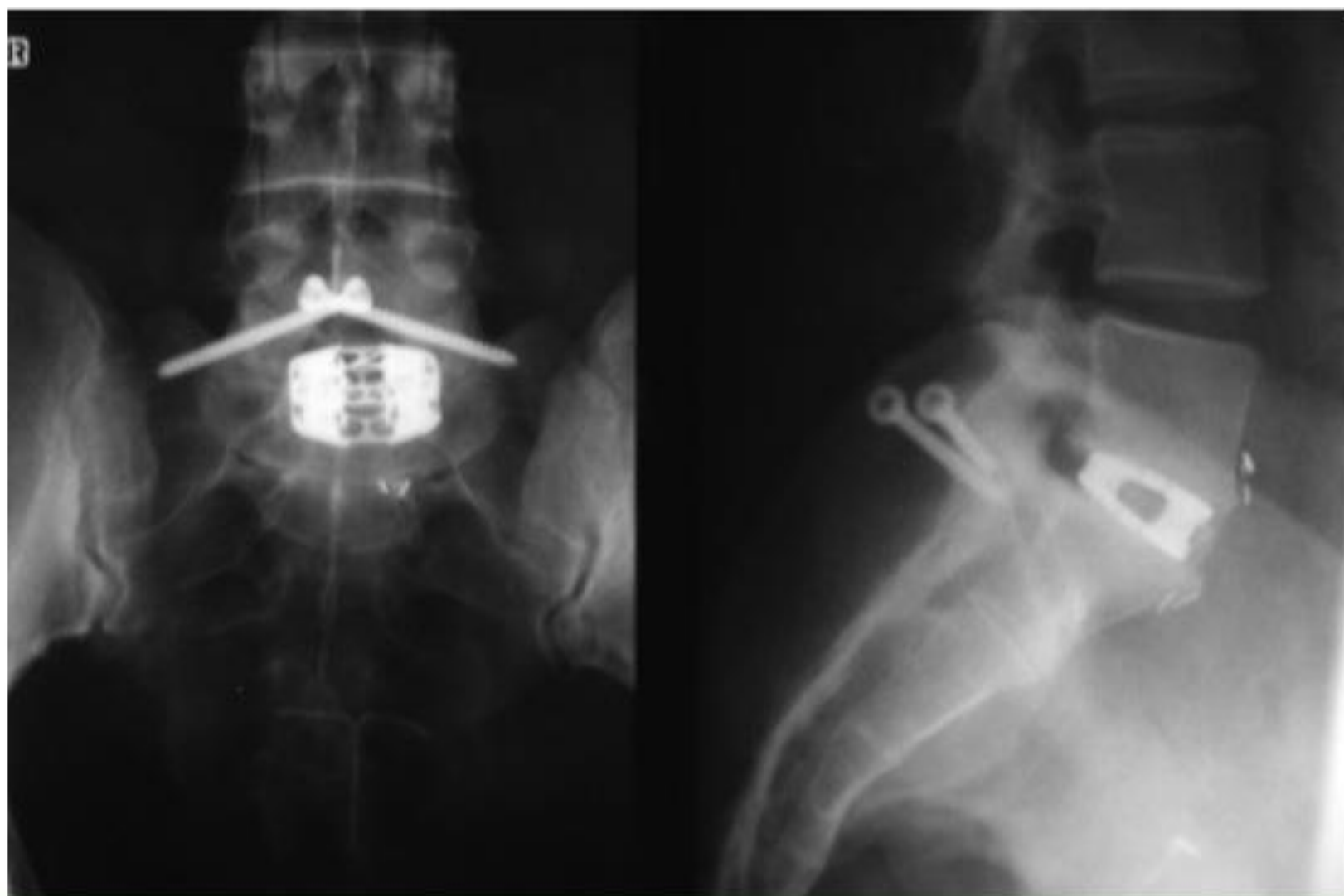
# Presentation outline:

- Case
- Etiology of low back pain
  - Specific
  - Aspecific
- Future developments

# Diagnosis?



Is it painful??





# Traditional classification of low back pain

- 15% specific
- 85% aspecific

## Specific low back pain:

- Instability
- Spondylolisthesis
- Degenerative scoliosis
- Tumors
- Infections
- etc etc.

# A-specific low back pain?

Pain behaviour / conditioning

Non organic signs:

*Non-organic physical* signs in low back pain:  
Waddell et al, Spine 1980

- Tenderness
  - Superficial
  - Non-anatomic
- Simulation
- Distraction
  - Straight leg raising / Flip test
- Regional weakness
- Overreaction

“Whiplash”

Low back pain



The McGraw-Hill Companies

# BusinessWeek

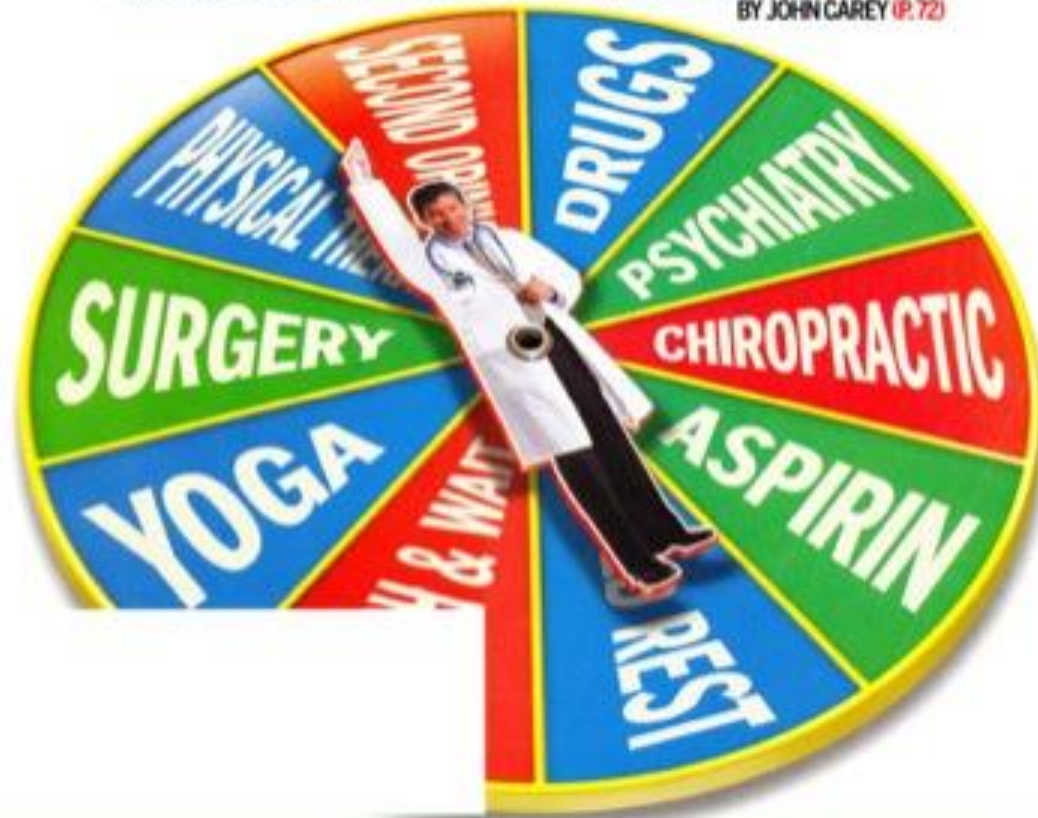
JULY 26, 2004

www.businessweek.com

## Medical Guesswork

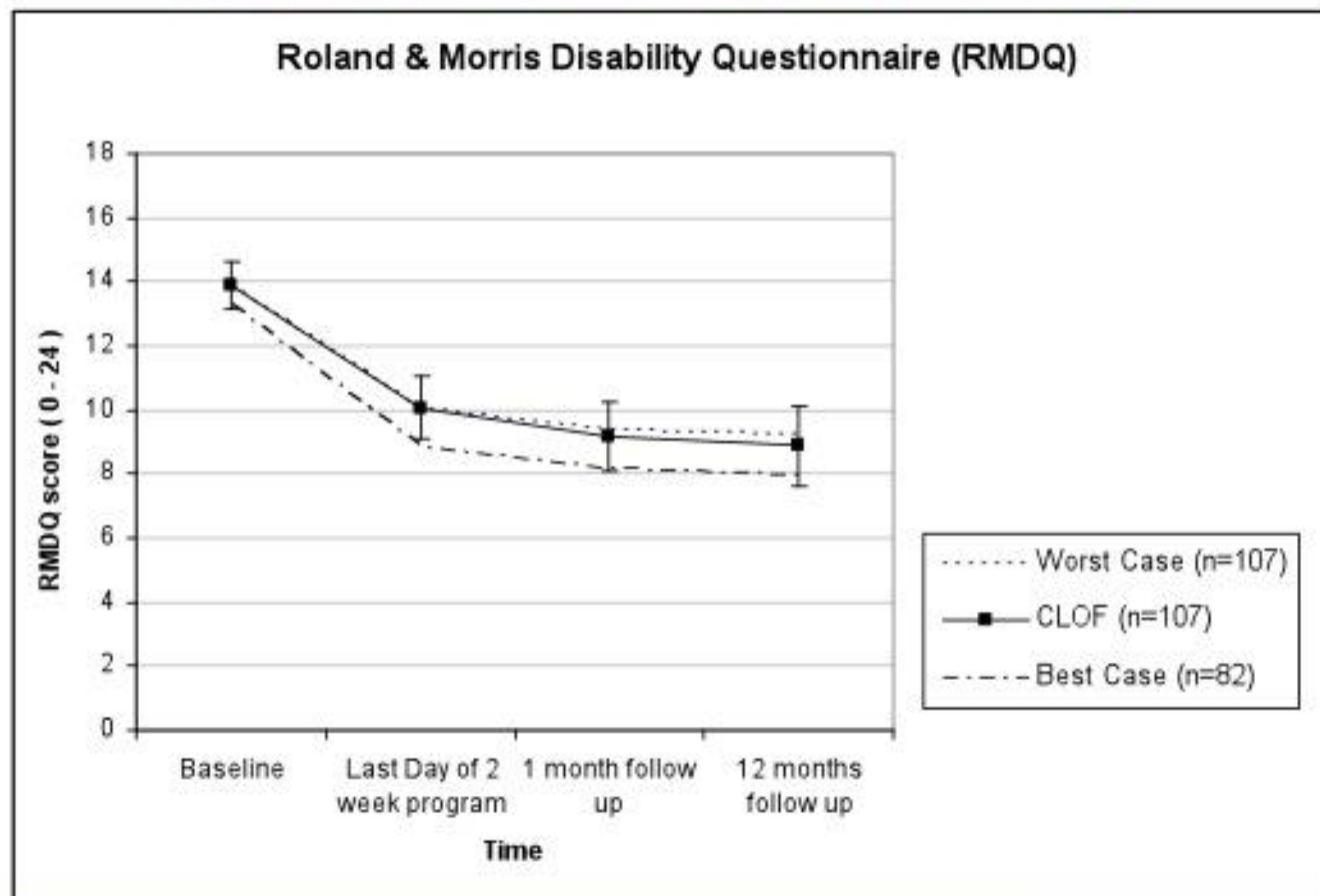
From heart surgery to prostate care, the medical industry knows little about which treatments really work

BY JOHN CAREY (P. 72)

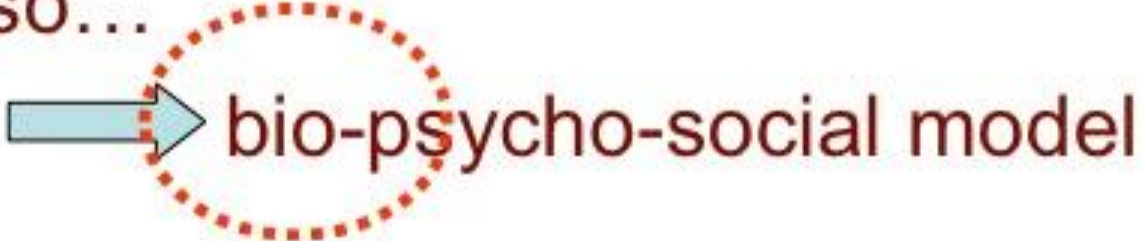


# Real Health

## cognitive behavioral group therapy



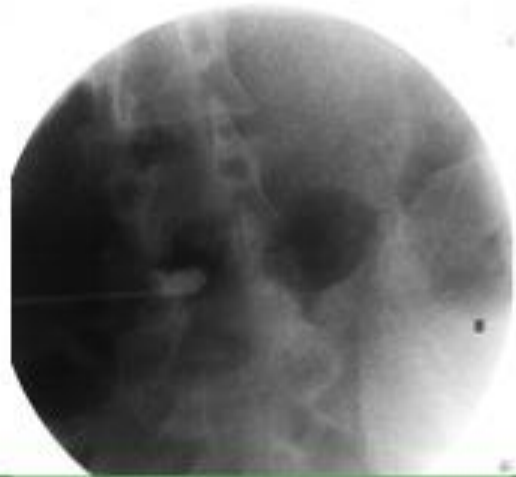
# A-specific low back pain

- 15% specific
- 85% aspecific,
  - Pain behaviour ?
  - No organic cause (psychogenic?)
- But is this so...  


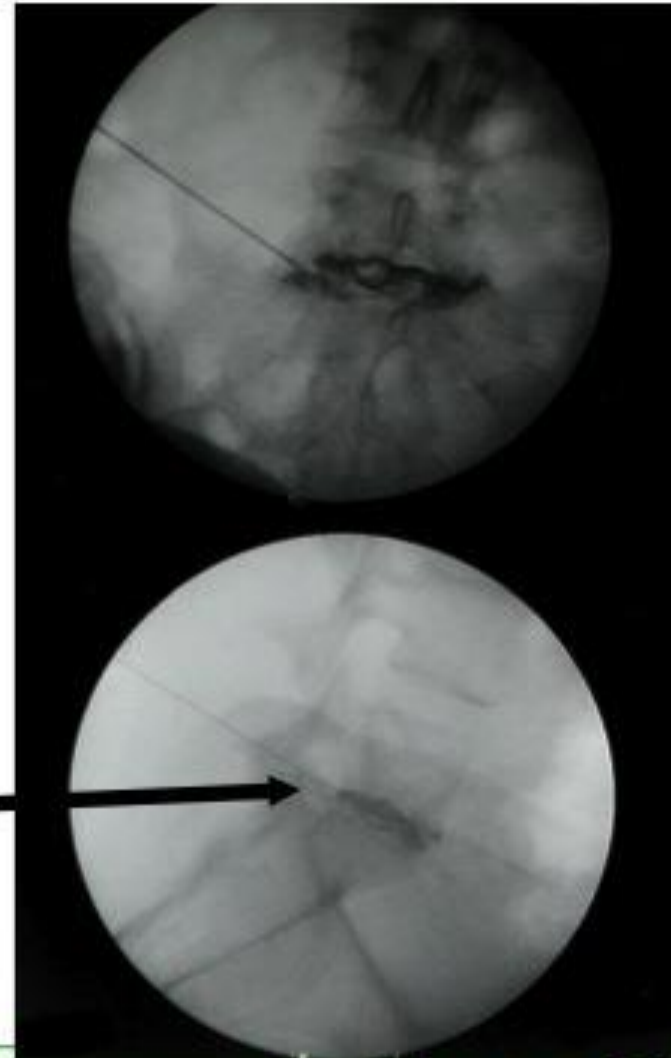
# The pathologic intervertebral disc can be painful: provocative discography



Normal  
morphology  
No pain



degenerative  
morphology  
painful





# Degeneration cascade

1. Normal

2. Instability phase, painful?

- Disc degeneration
- Loss of disc height
- Change in neutral zone
- Facet arthropathy



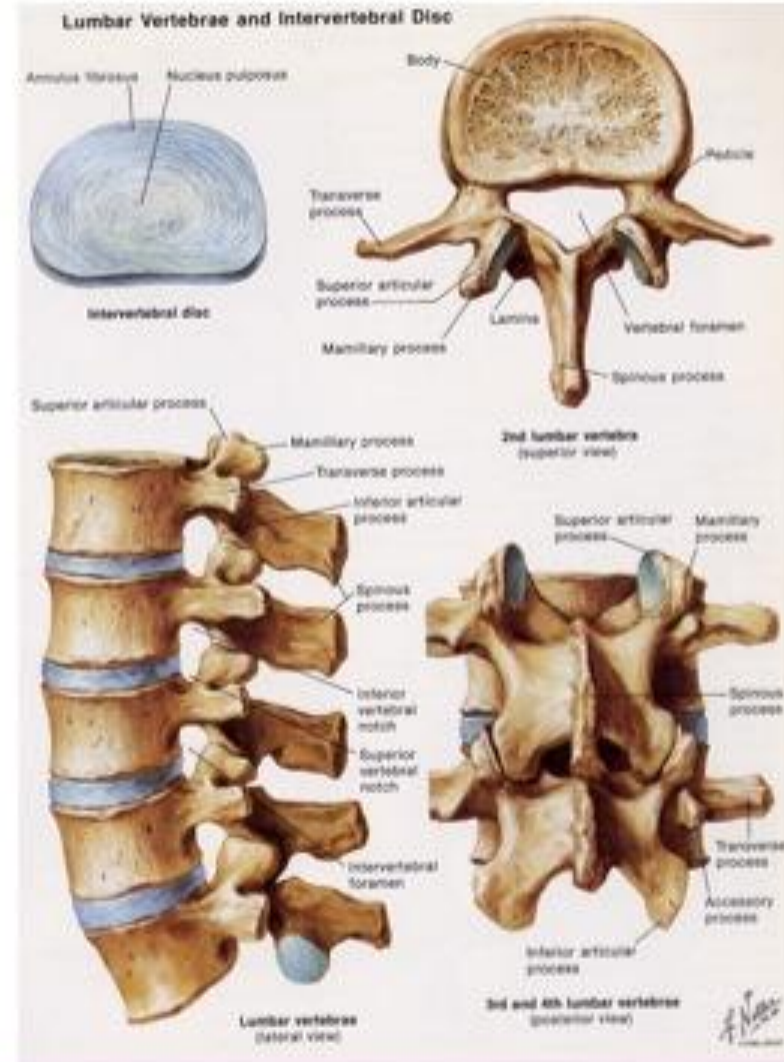
3. Stabilisation phase

- Osteophyte formation

*Kirkaldy-Willis*

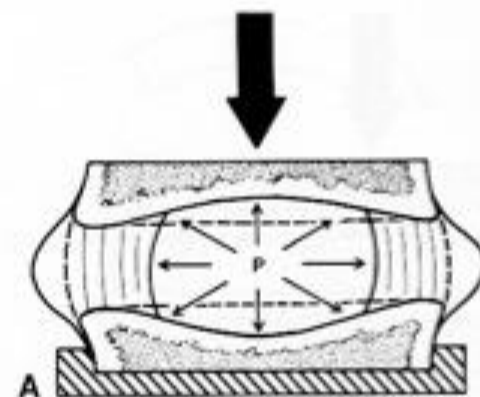


# Disc

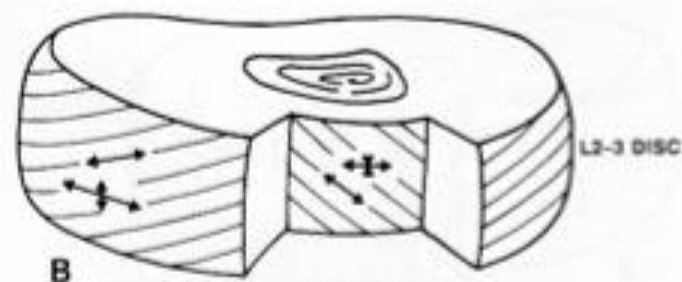


# Disc degeneration: the intervertebral disc

- Largest avascular structure
- Not innervated
- Annulus
  - Collagen type I
  - Mesenchymal cells
- Nucleus
  - Collagen II
  - Collagen IX (very little, provides cross links?)
  - Proteoglycans
  - Cells: Notochordal cells, change to chondrocyte cells, disappear by 40 yrs
  - 70-95% water, low Ph, low O<sub>2</sub>, high lactate, negative charge.



← TENSILE STRESS  
→ COMPRESSIVE STRESS

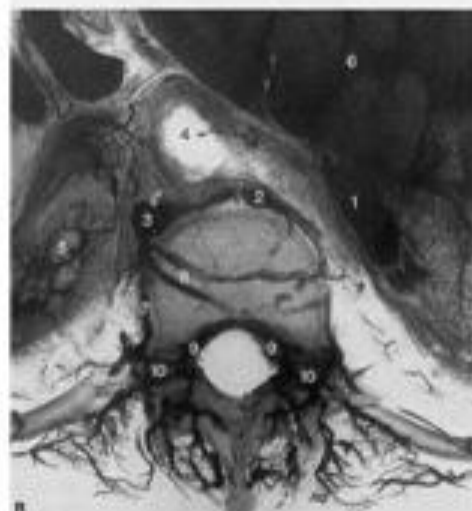




# Vascularisation



Arterial



Venous

# Disc degeneration

## 2 GROUPS??

- I. Age related degeneration, usually painless?
  
- II. Accelerated degeneration
  - a) With genetic predisposition, = painfull group??
  - b) Environmental factors, eg smoking, loading, BMI

# What causes disc degeneration?

## I. Age

- Normal ageing proces



# What causes disc degeneration?

## Ila. Genetics

Ala Kokko, twin studies Finland

- 77% of MRI changes are genetically determined!!
- Collagen type IX allele

Cheung, HK,

804 volunteers, MRI and DNA sample

- Strong relationship MRI DDD - LBP

Collagen IX Trp2 allele



2,4x DDD

Vit D receptor t allele



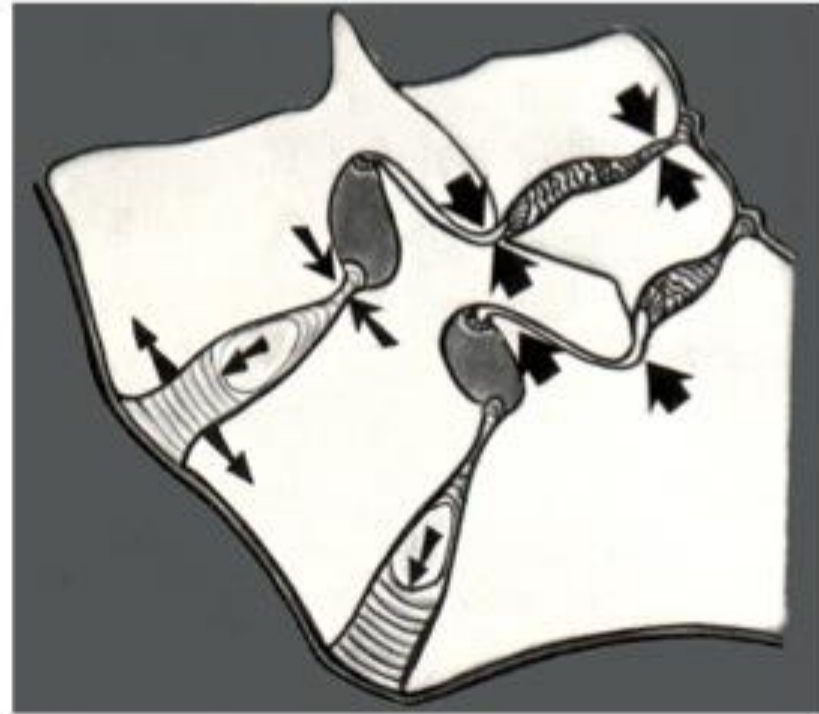
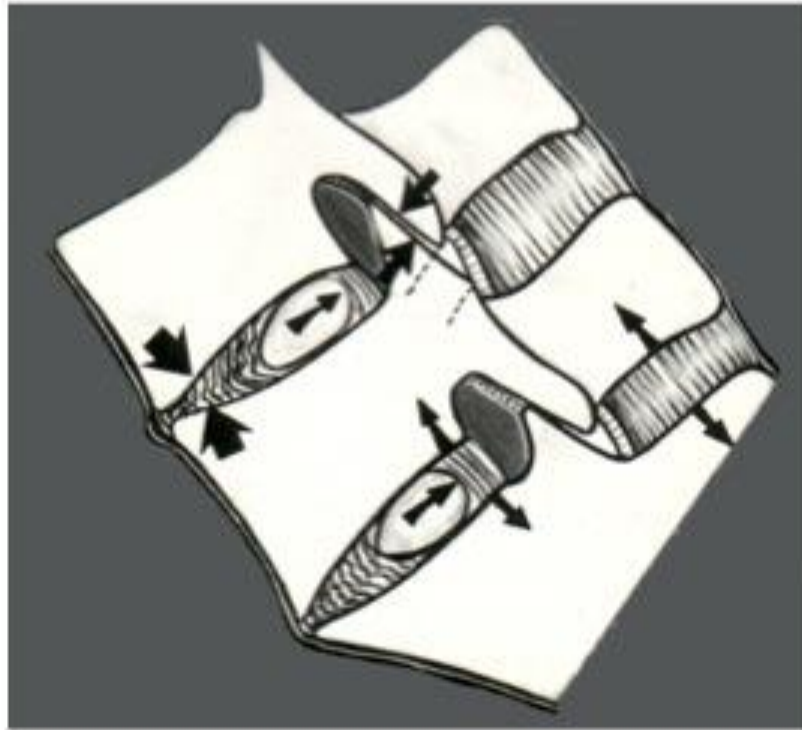
2,4x DDD  
5x HNP

# What causes disc degeneration?

## IIb. Environmental factors

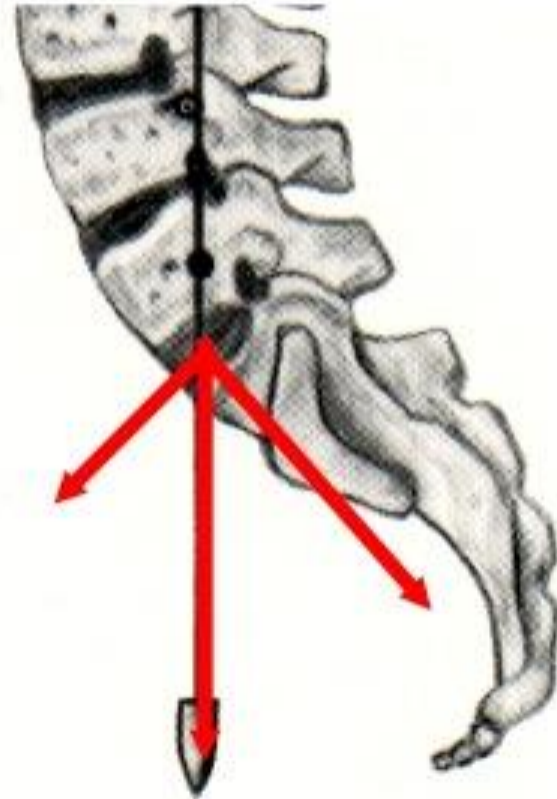
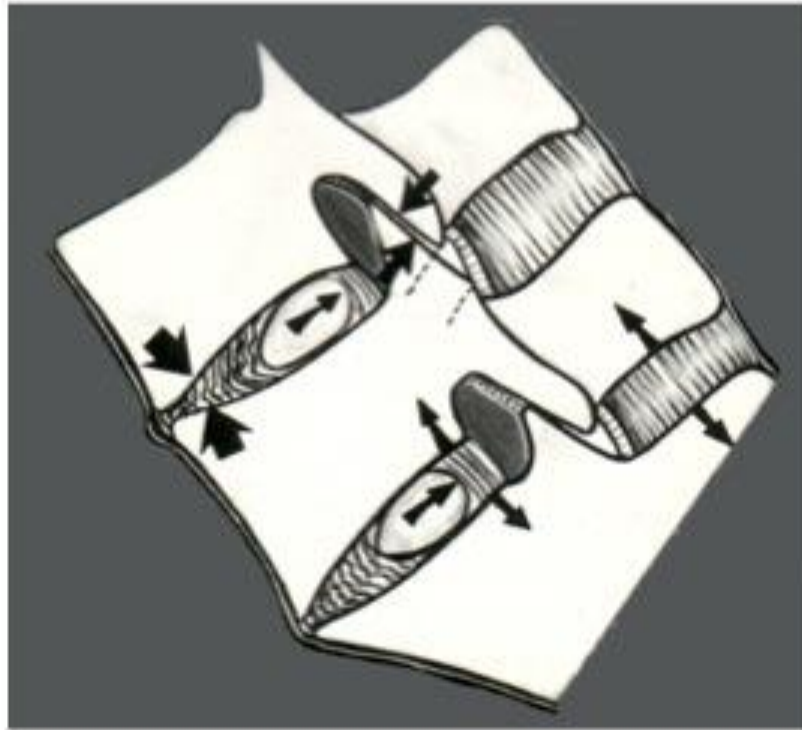
- Mechanical loading, BMI
- Smoking
- Low grade infection
- .....

# What causes disc degeneration? Mechanical loading



# What causes disc degeneration?

## Mechanical loading





What causes disc degeneration?  
**Mechanical loading**

**Facet tropism**

**Coupled motion**





# What causes disc degeneration?

## Biology / disc nutrition

- Endplate
  - Osseous
  - Cartilage
- Permeability
  - Nutrient **diffusion**
  - Fluid **flow**, esp due to cyclic loading



# What causes disc degeneration? Biology / disc nutrition

- Endplate
  - Osseous **sclerosis**
  - Cartilage **calcification**
- Permeability
  - Nutrient **diffusion**
  - Fluid **flow**, esp due to cyclic loading





# What causes disc degeneration? Biology / disc nutrition

- Endplate
  - Osseous **sclerosis**
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- Permeability
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# What causes disc degeneration?

## Biology / disc nutrition

- Endplate
  - Osseous
  - Cartilage
- Permeability
  - Nutrient diffusion
  - Fluid flow, esp due to cyclic loading

sclerosis

calcification

smoking



Cell regulation  $\updownarrow$

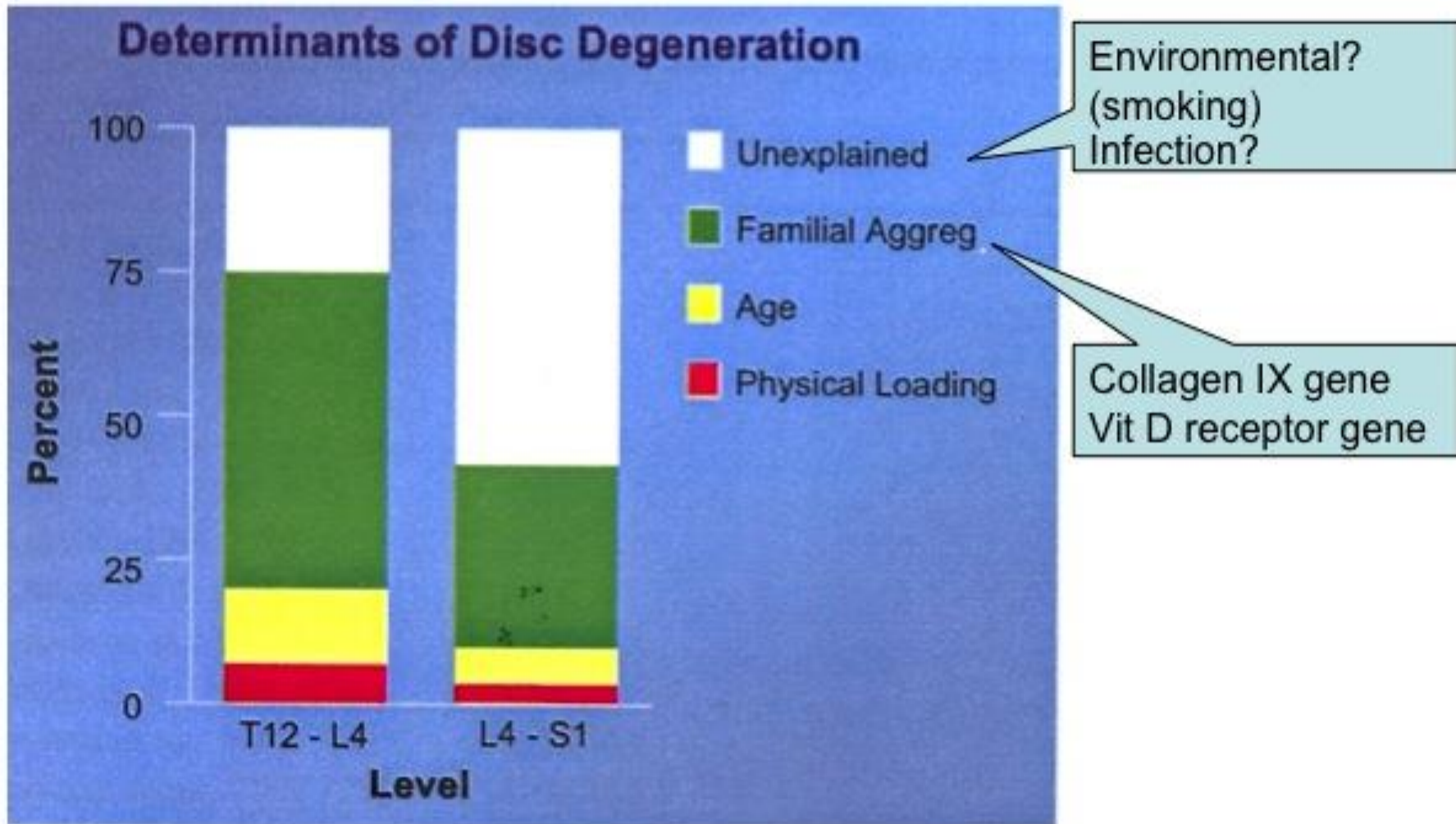
Matrix production  $\downarrow$

Cell death

## What causes disc degeneration? low grade infection

- Fritzell, Eur Spine J. 2004
  - PCR study, bacterial DNA in painfull discs
- Stirling, Lancet 2001
  - Culture and serology: Propionibacteria  
Acne





Battié MC, et al

The Twin Spine Study: contributions to a changing view of disc degeneration.

Spine J. 2009 Jan-Feb;9(1):47-59.

## Biology of disc nucleus degeneration

- Cell regulation  $\updownarrow$ 
  - Cell death
- Matrix degeneration
  - Matrix production  $\downarrow$
  - H<sub>2</sub>O binding  $\downarrow$
- Cytokines  $\uparrow$  IL, TNF

Burke JBJS 2002

Nerlich ESJ 2005

- BMP up/down regulation



## Biology of annulus degeneration

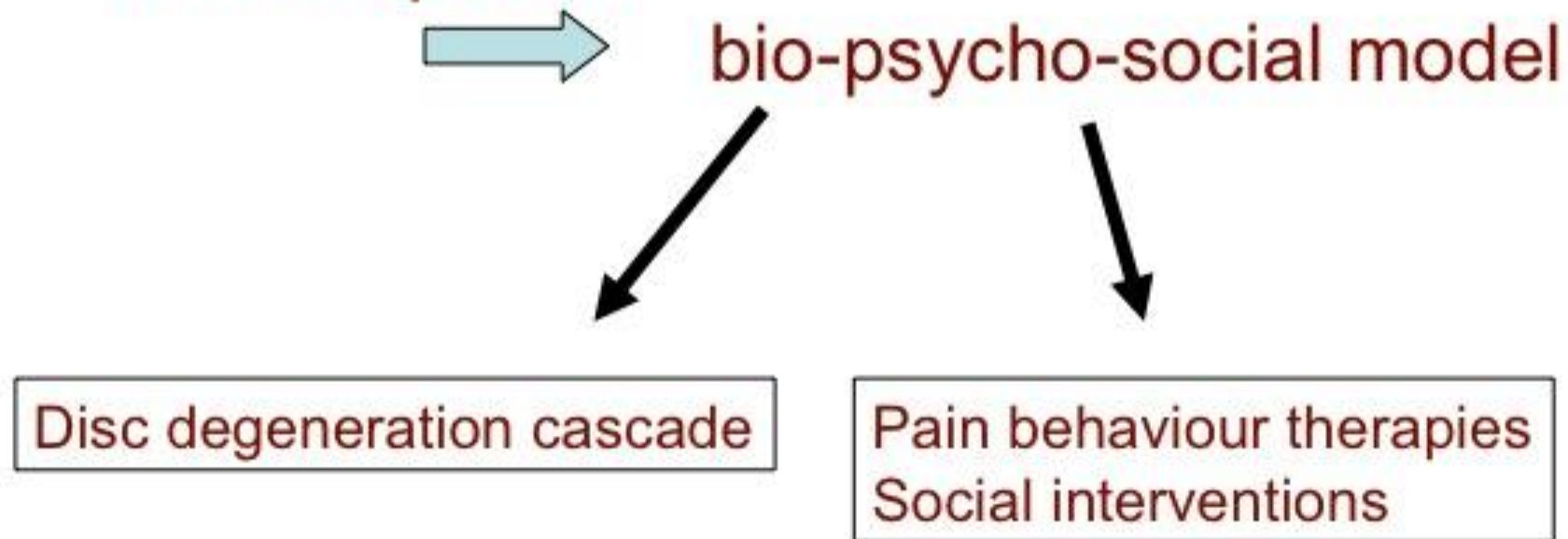
- Annular tears
  - High Intensity Zone (HIZ), Aprill, BJR 1992
- Neo-vascularisation
  - Coppes, Spine 1997



# Low back pain

## Conclusions

- 15% specific
- 85% “aspecific”





# Disc degeneration cascade

- Originates at the disc
- Related to low back pain.

## 2 GROUPS

- I. Age related degeneration, usually painless??
- II. Accelerated degeneration
  - a) With genetic predisposition, = painful group??
  - b) Environmental factors, eg smoking, loading, BMI, infection

Biologic changes in disc

- nucleus
- annulus

# Future treatments of disc degeneration

- Improve end plate permeability
  - Decalcify? But neo-vascularisation
- Gene therapy
  - Collagen IX
  - Vit D receptor t
- Cell injections to make new matrix
  - Mesenchymal stem cells?
    - But how do they survive?
    - And they have genetic defect.
  - Identify individuals at risk **before** endplate changes?  
Before symptoms?
  - Inject cells with carrier matrix
- Growth factor injections to stimulate matrix production,
  - But how to regulate?
  - How to prevent wash-out?
- Treat infection