primary care centre

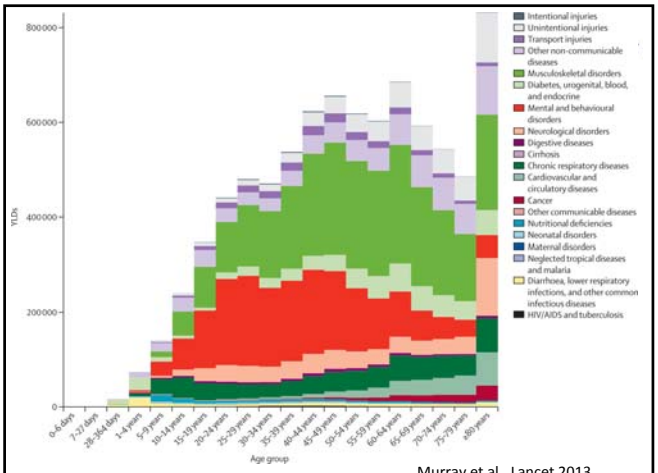
Keele University

The Natural History of Osteoarthritis:

Occurrence, presentation, and course of joint pain

George Peat

Professor of Clinical Epidemiology



Murray et al., Lancet 2013

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Disorder	2010 Mean rank (95% UI)	% change (95% UI)
1 Low back pain	1·0 (1 to 1)	12 (2 to 23)
2 Falls	3·7 (2 to 7)	32 (14 to 50)
3 Major depressive disorder	3·8 (2 to 8)	9 (−11 to 33)
4 Neck pain	3·9 (2 to 7)	13 (2 to 26)
5 Other musculoskeletal disorders	4·7 (2 to 9)	15 (−25 to 70)
6 Anxiety disorders	6·1 (2 to 9)	6 (−14 to 31)
7 COPD	7·1 (3 to 12)	14 (−0 to 31)
8 Drug use disorders	8·5 (5 to 11)	7 (−9 to 25)
9 Asthma	8·7 (4 to 14)	10 (−2 to 22)
10 Migraine	8·9 (5 to 13)	7 (−11 to 27)
11 Osteoarthritis	11·0 (7 to 15)	16 (−4 to 41)
12 Alcohol use disorders	12·2 (9 to 16)	48 (6 to 104)
13 Alzheimer's disease	13·5 (11 to 17)	41 (19 to 64)
14 Road injury	14·5 (11 to 19)	12 (−10 to 36)
15 Schizophrenia	16·9 (11 to 23)	15 (2 to 30)
16 Benign prostatic hyperplasia	17·4 (12 to 24)	26 (−6 to 65)
17 Other hearing loss	18·4 (11 to 26)	−10 (−22 to 2)
18 Diabetes	18·4 (15 to 24)	8 (−9 to 29)
19 Ischaemic heart disease	19·4 (13 to 26)	−1 (−19 to 20)
20 Bipolar disorder	20·2 (13 to 27)	5 (−20 to 37)
21 Dysthymia	20·4 (15 to 28)	10 (−3 to 25)
22 Rheumatoid arthritis	23·0 (18 to 28)	19 (2 to 38)
23 Stroke	23·4 (16 to 32)	50 (−24 to 178)
24 Chronic kidney disease	23·8 (18 to 28)	25 (10 to 45)
25 Edentulism	24·3 (18 to 31)	−32 (−42 to −21)

Murray et al., Lancet 2013

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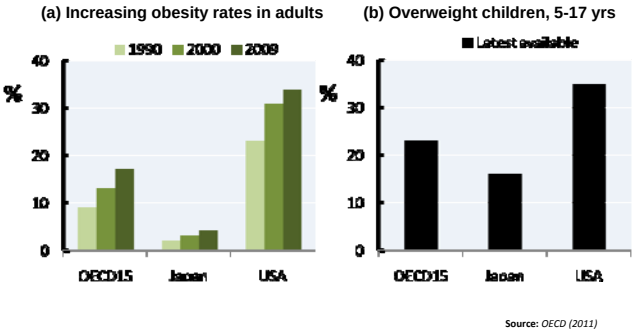

Source: *OECD (2011)*

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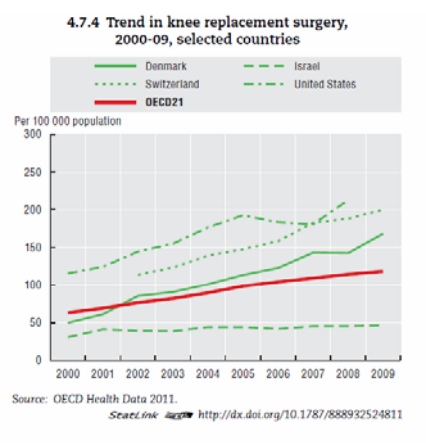

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What is OA?

Defining OA has important implications for prevention, diagnosis, and treatment of this condition. Based on evidence to date, there was consensus that OA is usually a progressive disease of synovial joints that represents failed repair of joint damage that results from stresses that may be initiated by an abnormality in any of the synovial joint tissues, including articular cartilage, subchondral bone[2–4], ligaments, menisci (when present)[5,6], periarticular muscles[7], peripheral nerves, or synovium[8,9]. This ultimately results in the breakdown of cartilage and bone[10], leading to symptoms of pain, stiffness and functional disability[11]. Abnormal intra-articular stress and failure of repair may arise as a result of biomechanical[12], biochemical[13] and/or genetic factors[14]. This process may be localized to a single joint, a few joints, or generalized, and the factors that initiate OA likely vary depending on the joint site. The complexity and variability of OA etiology suggests the need for patient-specific, etiology-based treatment.

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Source: Lane *et al.* OAC 2011

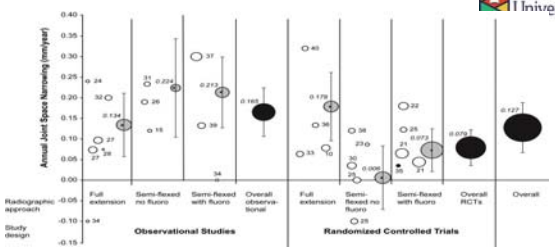


Fig. 2 Annual JSN stratified by study design and radiographic approach. Circles represent individual mean JSN estimates. Circle area is proportional to sample size of corresponding cohort. Means within study design and radiographic approach sub-categories ...

P.S. Emrani , J.N. Katz , C.L. Kessler , W.M. Reichmann , E.A. Wright , T.E. McAlindon , E. Losina

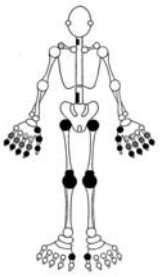
Joint space narrowing and Kellgren–Lawrence progression in knee osteoarthritis: an analytic literature synthesis

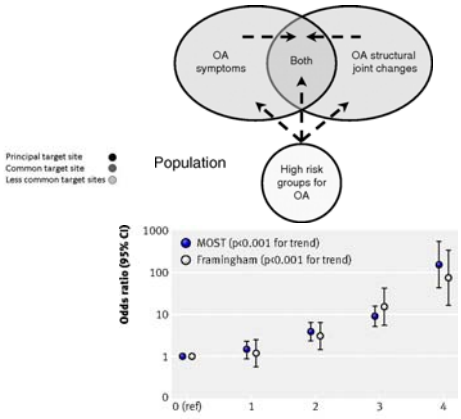
Osteoarthritis and Cartilage Volume 16, Issue 8 2008 873 - 882

http://dx.doi.org/10.1016/j.joca.2007.12.004

While late-stage OA is often characterized by both demonstrable structural damage and patient reports of joint pain, stiffness and disability[17]. There is only a weak correlation between symptoms and pathology, particularly in early stages of the disease[18]. Further, FDA-approved treatments directed at reducing the symptoms of OA have not been shown, to date, to prevent ongoing joint structural damage. For this reason, the Working Group felt that future development of treatments for OA should consider the effects of the treatment on the structural changes at the joint level (the *disease* OA) separately from the effects on patient-reported symptoms (the *illness* OA). Future pharmacotherapy for OA may therefore be considered to be 'structure-modifying' (i.e., designed to prevent the development of joint failure), symptom modifying, or both.

Source: Lane *et al.* OAC 2011





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Vol. 55, No. 5, October 15, 2006, pp 779–785
DOI 10.1002/art.22244
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ORIGINAL ARTICLE

Course of Functional Status and Pain in Osteoarthritis of the Hip or Knee: A Systematic Review of the Literature

GABRIELLA M. VAN DIJK,[1] JOOST DEKKER,[2] CINDY VEENHOF,[1] AND CORNELIA H. M. VAN DEN ENDE,[1] FOR THE CARPA STUDY GROUP

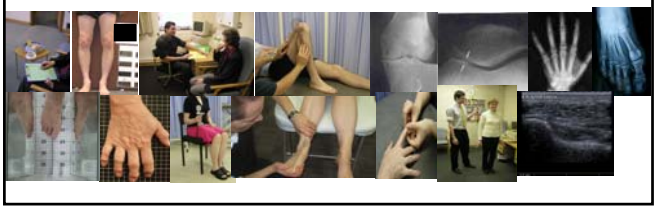
Conclusion. Pain and functional status in hip or knee OA seem to deteriorate slowly, with limited evidence for worsening after 3 years of followup.

Participants

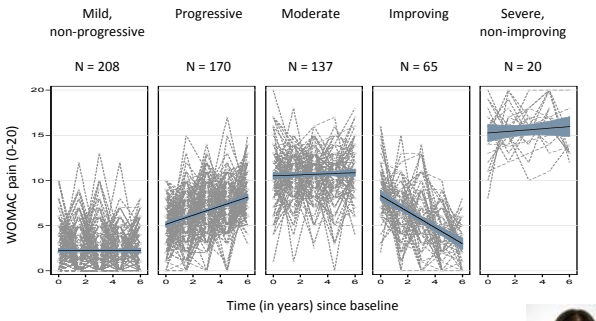
- Registered with participating general practices at time of study recruitment
- Aged 50+ years
- Reported symptoms in past 12 months
- Consented to further contact
- Attended baseline research assessment clinic

Data collection

Started	ACRONYM	Focus	N	Questionnaire	Clinical interview	Physical examination	Functional performance	Anthropometric measurement	Digital photographs	Plain Xrays	Ultrasound	Linkage to medical records	Follow-up (months) 0	18	36	54	72
2002/3	CAS-K	Knee	819	•	•	•	•	•	•	•		•	✓	(✓)	✓	(✓)	✓
2004/5	CAS-HA	Hand	623	•	•	•	•	•	•	•		•	✓	(✓)	(✓)	(✓)	✓
2010/1	CAS-F	Foot	560	•	•	•	•	•	•	•	•	•	✓	(✓)	P		



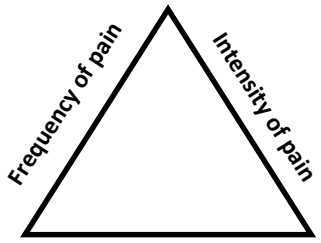
Finding 1. Different pain trajectories



Elaine Nicholls. OARSI 2013

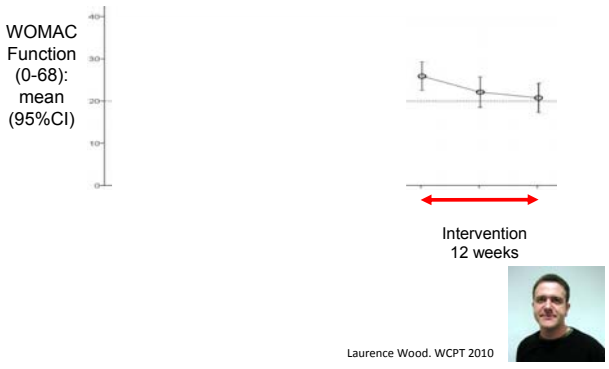
"The median isn't the message."

Stephen Jay Gould



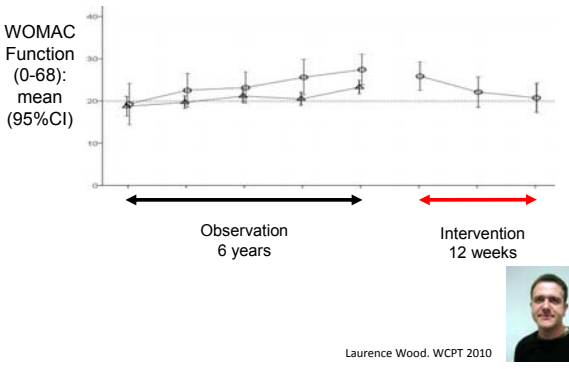
Interference with valued activities and roles

Finding 2. Intervening mid-trajectory



Laurence Wood. WCPT 2010

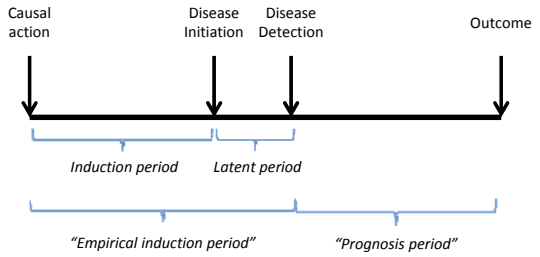
Finding 2. Intervening mid-trajectory



Laurence Wood. WCPT 2010

- Trajectories may be (temporarily) modifiable by episodes of care
- Conversely, might trajectories be adversely modified by acute exacerbations?
- Cumulative effects??

Classical view of natural history



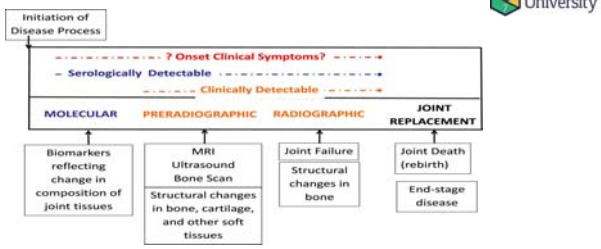


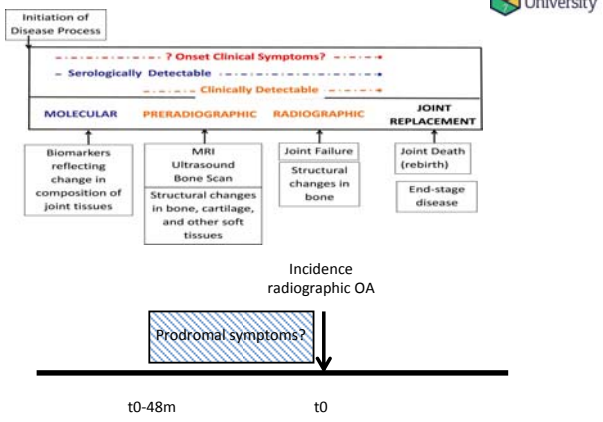
Fig.71 The natural history of OA and the purported roles of biomarkers during the disease process. Original attributed to V Kraus (originally presented at OARSI Congress 2009: Kraus, VB. 2009.

D. Hunter , F. Eckstein , V.B. Kraus , E. Losina , L. Sandell , A. Guermazi

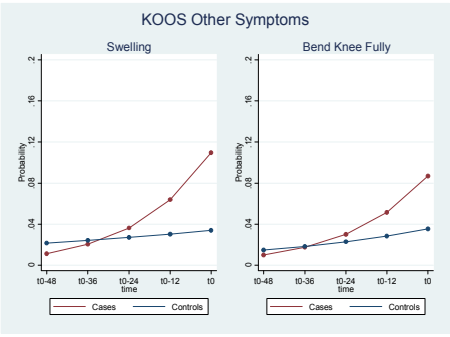
Imaging Biomarker Validation and Qualification Report: 6th OARSI Workshop on Imaging in Osteoarthritis combined with 3rd OA Biomarkers Workshop

Osteoarthritis and Cartilage

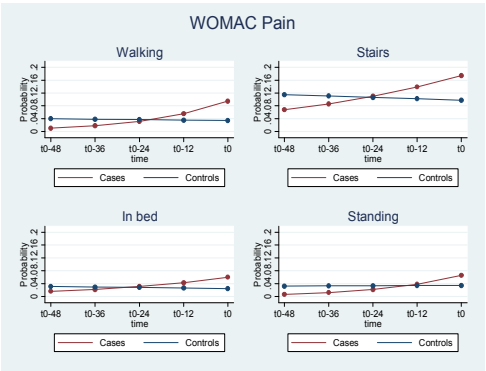
http://dx.doi.org/10.1016/j.joca.2013.04.014



Finding 3. Prodromal symptoms

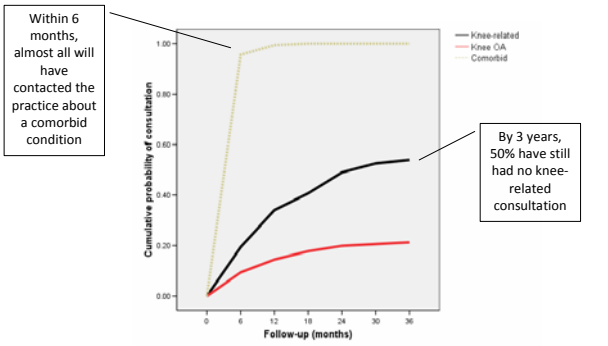


Rebecca Case
(Masters dissertation)



- Pre-radiographic phase is associated with increased symptoms up to 3 years prior to appearance of x-ray changes
- Nociceptive drivers of prodromal symptoms?
- Potential for early clinical recognition and intervention?

Finding 4. The symptom iceberg and the decision to consult

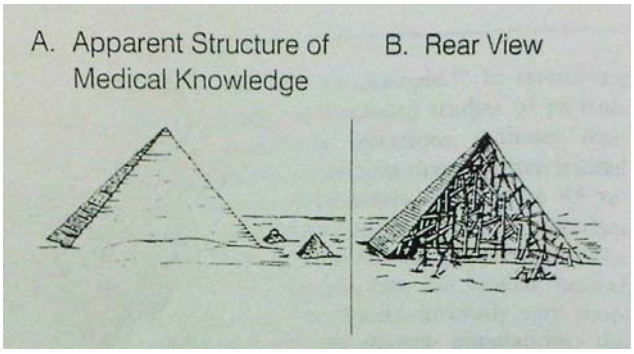


Source: Bedson et al. *Prim Health Care Res & Develop* 2009

Determinant	% contribution
Level of disruption to everyday activities	31
Perceived attitude of GP	24
Anticipated thoroughness of assessment and investigations	14
Competing comorbidity	13
Available management options/treatments	13
Pain characteristics	5

Domenica Coxon
(PhD dissertation)

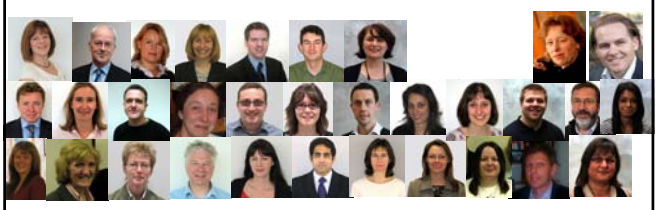
- Not as simple as 'degenerative', 'inevitably progressive'
- Attention is needed to pain and function in their own right
- Estimating the timing and sequence of events and state transitions in the natural history of osteoarthritis is extremely challenging



Source: Rushmer (1980) in Deyo (1993)

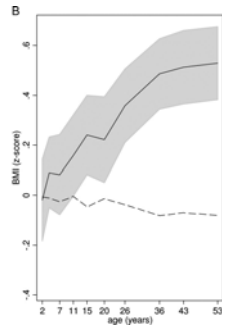
Acknowledgements

Arthritis Research UK | primary care centre



MRC Medical Research Council
Arthritis Research UK
BMA

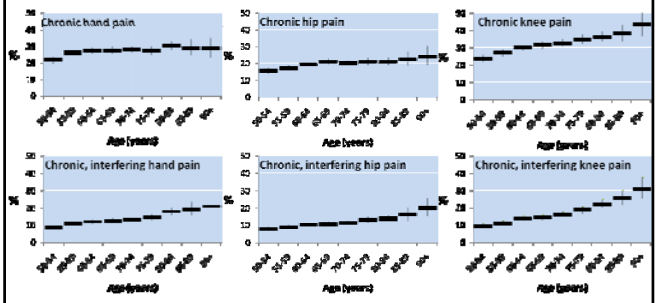
Causal actions that initiate osteoarthritis are complex and cumulative

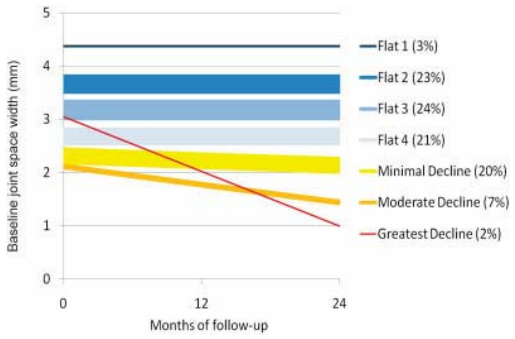


Mean lifetime body mass index (BMI) z-score and 95% CI (shaded area) in women among those with knee osteoarthritis (OA; solid line) at age 53 years.

Wills A K et al. Ann Rheum Dis
doi:10.1136/ard.2011.154021

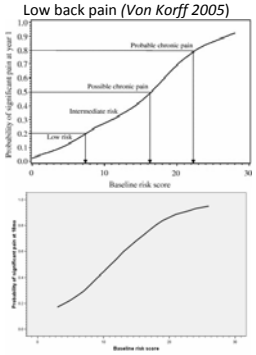
Prevalence of chronic joint pain in the over-50s in North Staffordshire





Generic prognostic indicators of self-reported pain/functional outcome

Low back pain *(Von Korff 2005)*



Prognostic indicators at baseline:

- Current pain intensity
- Interference with activities
- Disability days
- Duration of present episode
- Pain at other sites
- Depression

Clinical interview, physical examination, and severity of ROA add little prognostic information

Sources: Mallen et al. ARD 2007; Thomas et al., ARD 2008; Thomas et al. Pain 2008

Brief, generic prognostic indicators at the point of care: better than clinical judgement (but not good enough)

Table 3. Prognostic Indicators for the Study Models Based on 90 Imputed Data Sets

Prognostic Indicator	Model 1 (Physician Judgment), OR (95% CI)	Adjusted OR (95% CI) Model 2 (Plus Generic Indicators)	Model 3 (Reduced Model)
GP prediction of nonimprovement	2.78 (1.69-4.57)	1.69 (0.96-2.96)	1.66 (0.96-2.90)
Present episode >3 mo	. . .[a]	3.05 (1.86-4.98)	2.99 (1.84-4.86)
Moderate current pain intensity[b]	. . .	0.84 (0.41-1.75)	. . .
Severe current pain intensity[c]	. . .	0.62 (0.28-1.35)	. . .
Significant pain interference with daily activities[d]	. . .	2.01 (1.05-3.84)	1.63 (0.98-2.70)
Multiple site pain	. . .	1.82 (1.03-3.20)	1.72 (1.00-2.96)
Positive depression screen result[e]	. . .	1.00 (0.52-1.93)	. . .
Heuristic shrinkage factor	0.96	0.89	0.93
Uncorrected C statistic	0.62	0.72	0.72

Abbreviations: GP, general practitioner; OR, odd ratio.
[a]Ellipses indicate variable not included in the model.
[b]Current pain intensity (0- to 10-point numerical rating scale) score of 5 to 6.[39]
[c]Current pain intensity (0- to 10-point numerical rating scale) score of 7 to 10.[39]
[d]Pain interference with daily activity (0- to 10-point numerical rating scale) score of 5 to 10.[39]
[e]At least one yes response to Patient Health Questionnaire 2 items.

Sources: Mallen et al. JAMA Intern Med 2013