Cartilage Tribology and its role in Osteoarthritis

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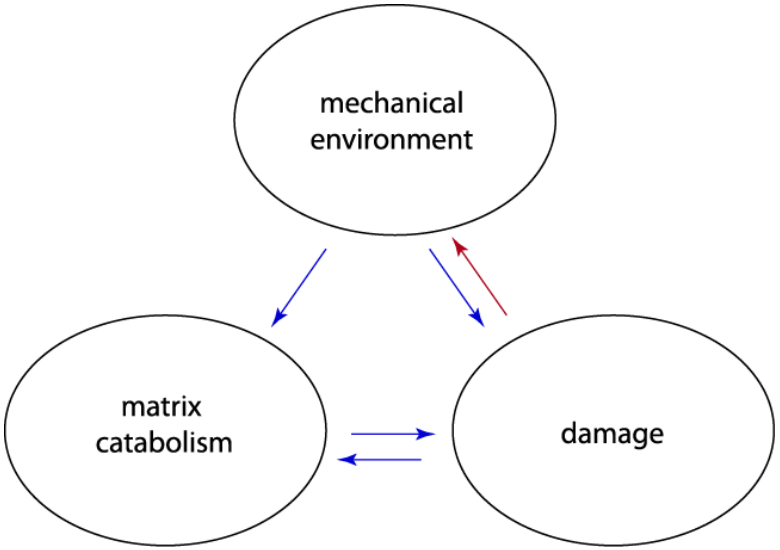
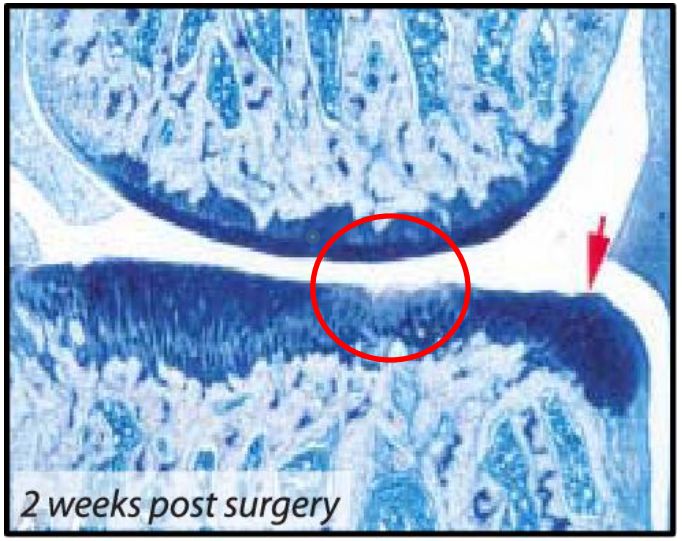
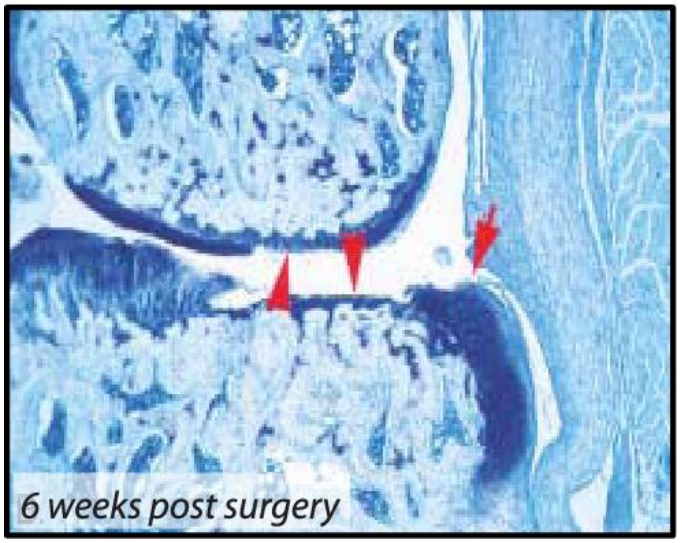
Observations

1) Healthy cartilage is wear-free
2) OA initiated by mechanical factors (obesity, ACL, impact, meniscus, etc.)
3) Initiates locally and propagates to failure



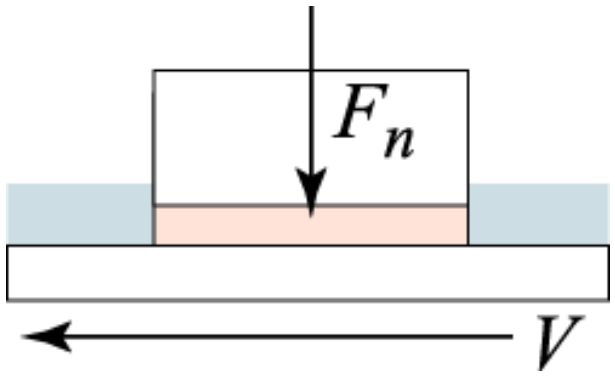
Hypothesis: damage impedes lubrication, lubrication disruptions cause damage





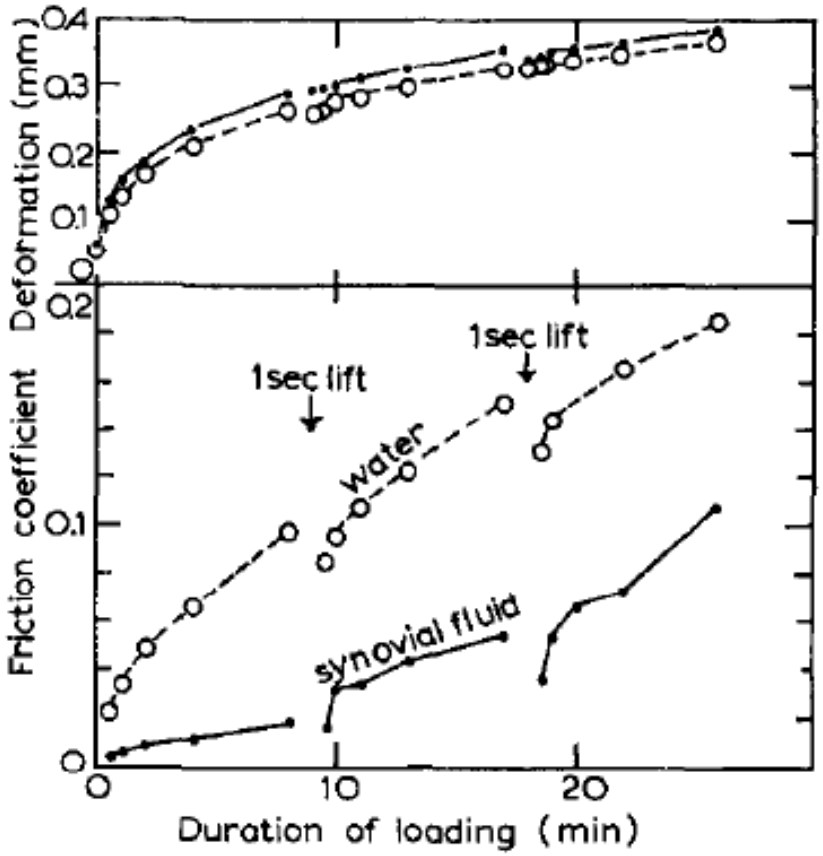
Rat knees-MMT, Bendele 2001

Tissue composition and structure dominate lubrication



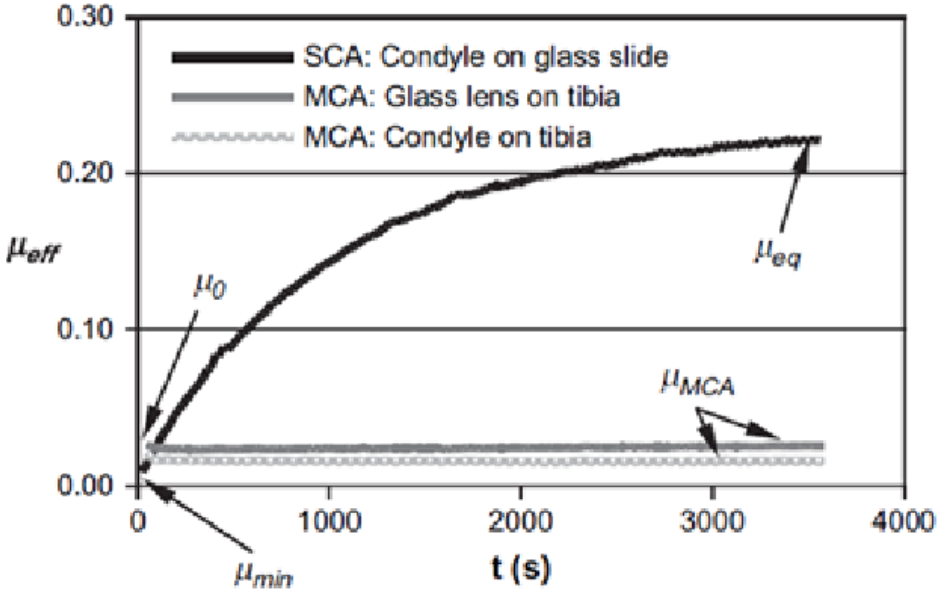
Stationary Contact Area (SCA)

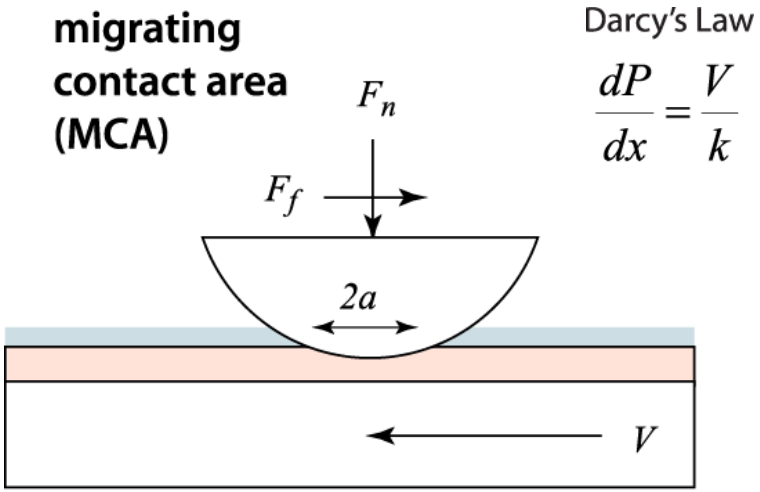
The frictional properties of animal joints C.W. McCutchen, Wear, 1962



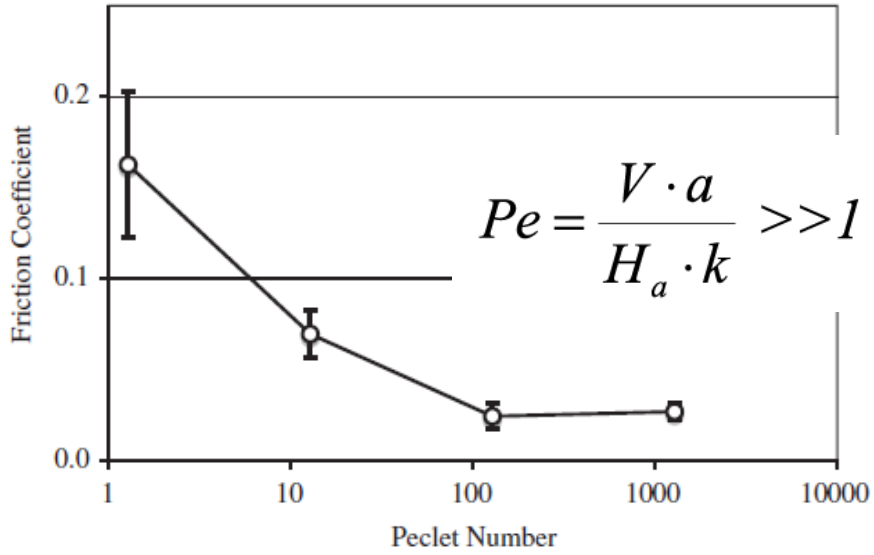
1. Primary self-pressurization (interstitial lubrication)
2. Secondary boundary (~2X), HD, EHD, and squeeze film
3. Hydration restored during times of rest

Discovery of interstitial lubrication sustainability: 2008





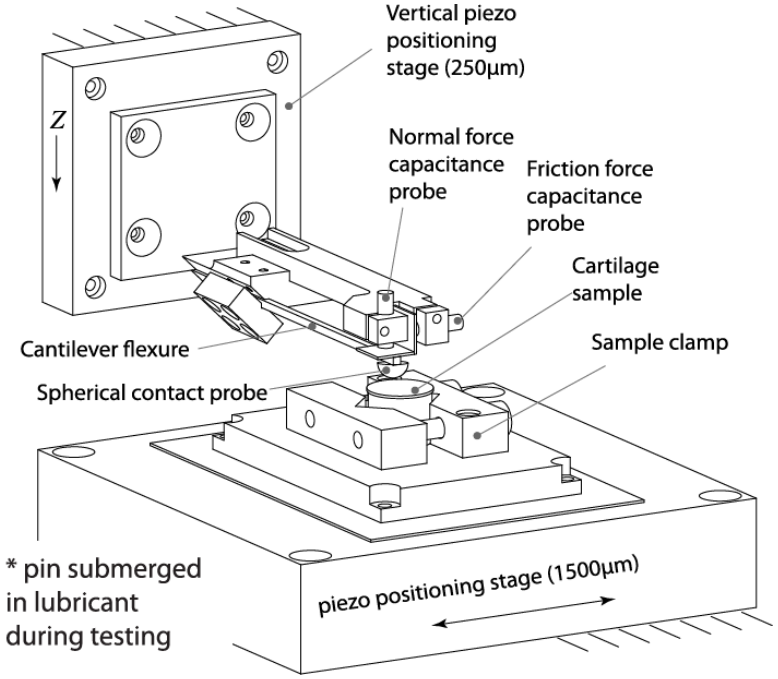
Caligaris et al., 2008

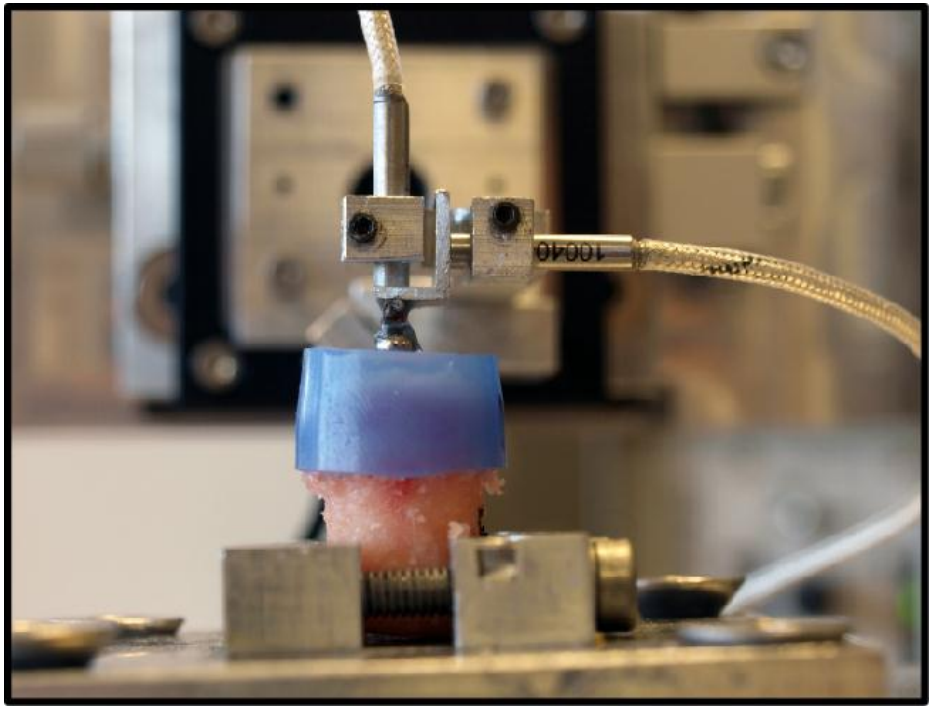


- Cartilage on cartilage is sustainable
- Pin-on cartilage produces a physiologically consistent response
- ***Primary physiological mechanism:*** Interstitial lubrication

Methods:

1) MCA: physiological maintenance
2) Localized
3) In-situ measurements of contact mechanics & fluid load support





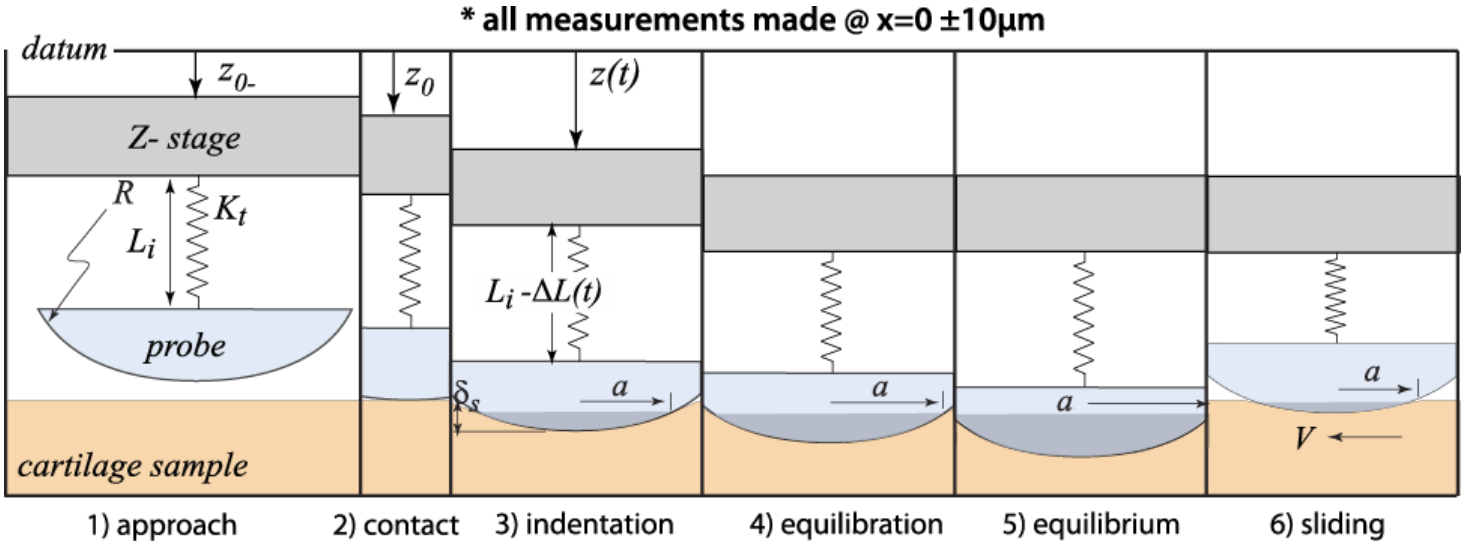
Microtribometer Specs:

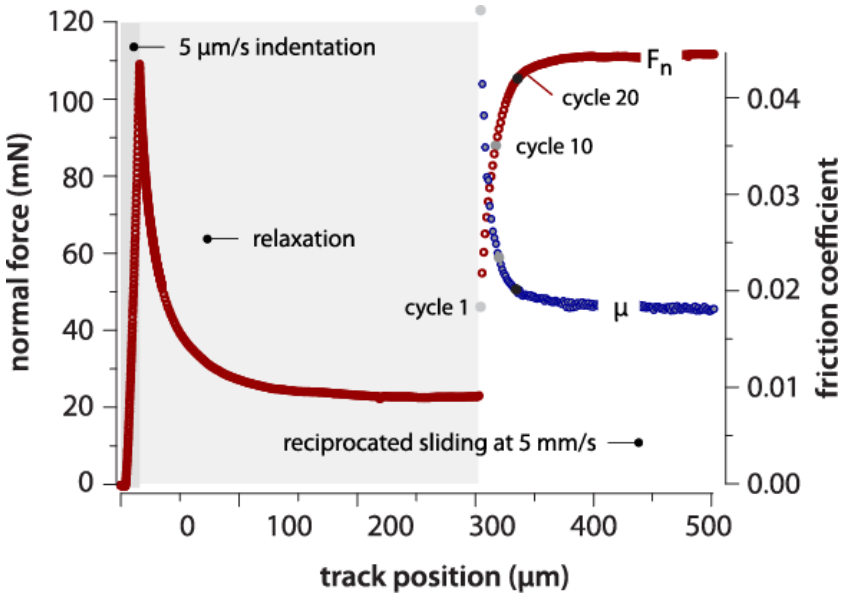
1) Load cell: 1-1000mN ± 1mN

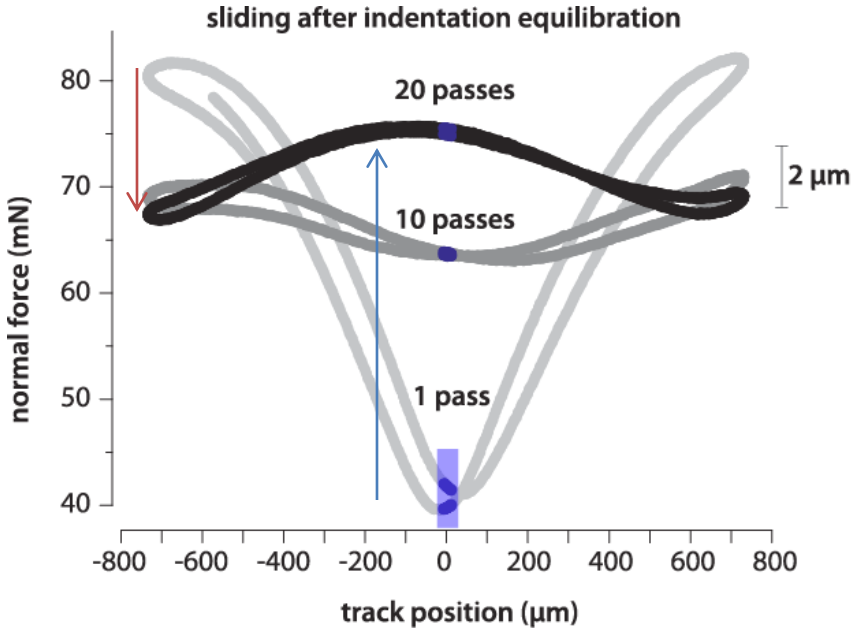
2) Vertical stage: 0-250µm ± 25nm

3) Lateral stage: 0-1.5 mm, 0-5 mm/s

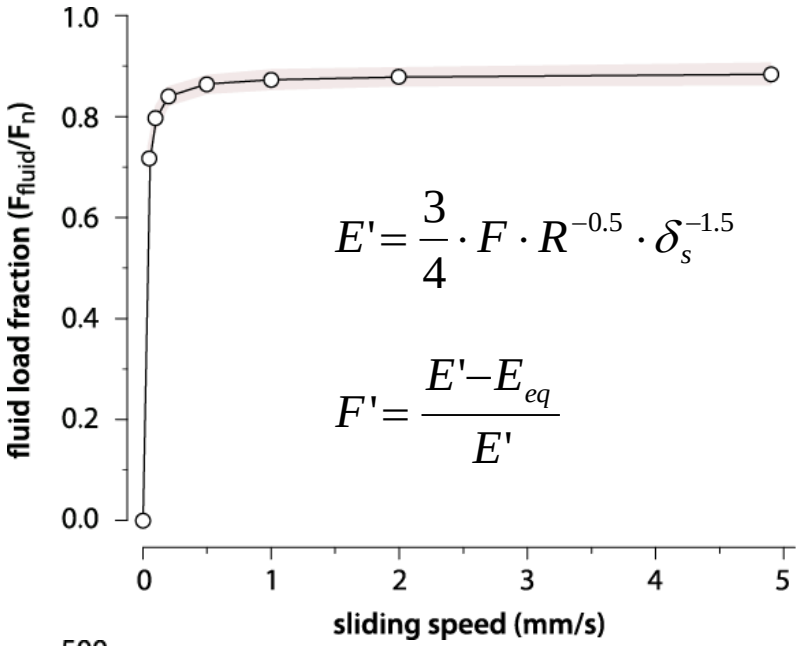
Example measurements: motion induced pressurization





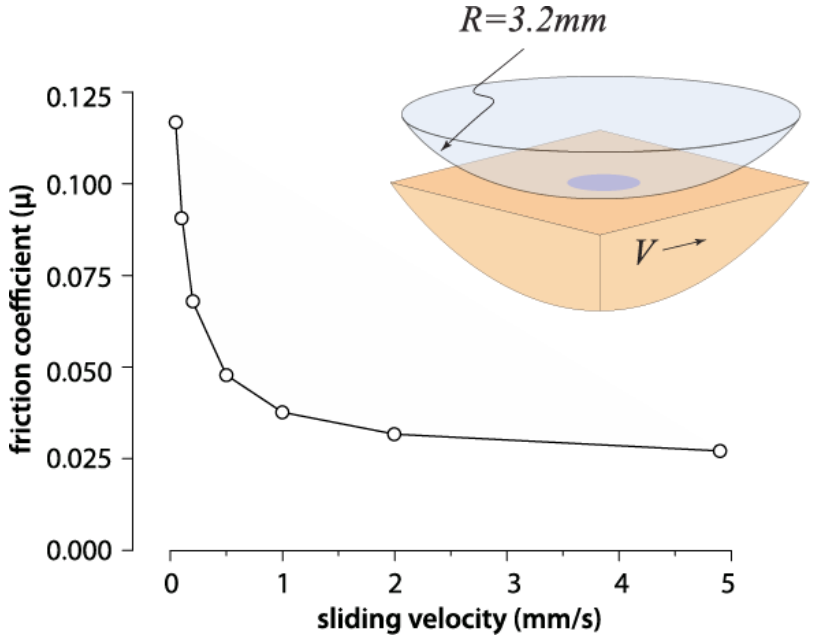


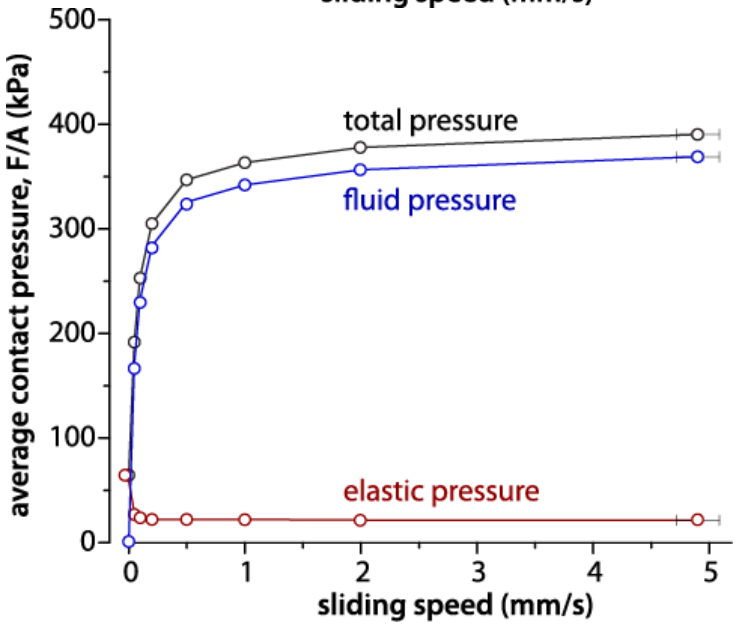
Interstitial pressurization response to sliding



$$E' = \frac{3}{4} \cdot F \cdot R^{-0.5} \cdot \delta_s^{-1.5}$$

$$F' = \frac{E' - E_{eq}}{E'}$$





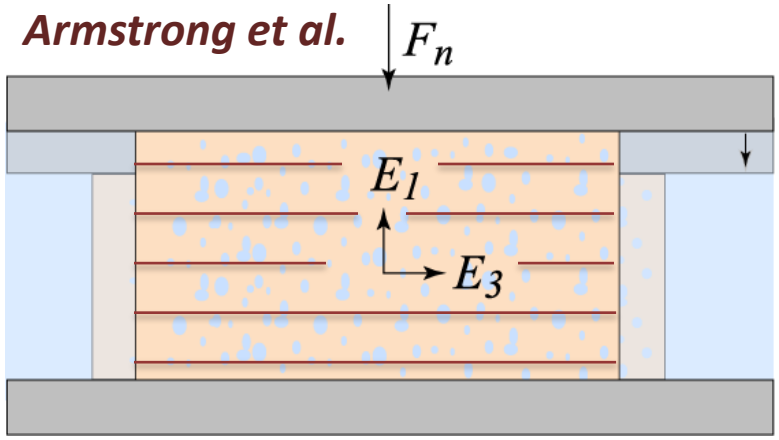
1) Interstitial pressure increases w/ speed
2) Fluid pressure dominates friction and matrix stress
3) M***aximum fluid load fraction*** likely dominates ***wear***

E.D. Bonnevie *et al.*, Tribology Letters, 2011

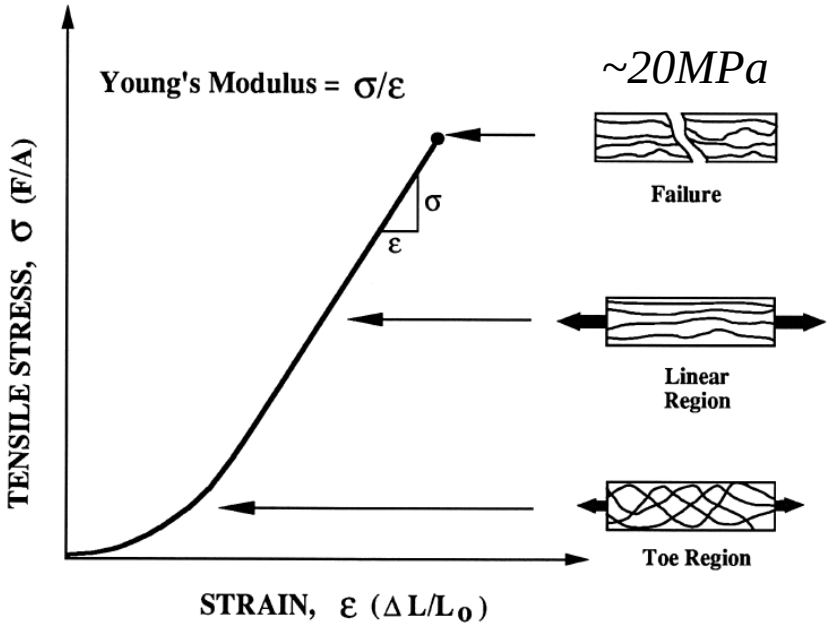
Collagen dominates the maximum fluid load fraction

Poroelastic solutions

1) Confined: 100%
2) Unconfined: 33%
3) Hertz: 50%



Unconfined compression: F'_{max}=33%



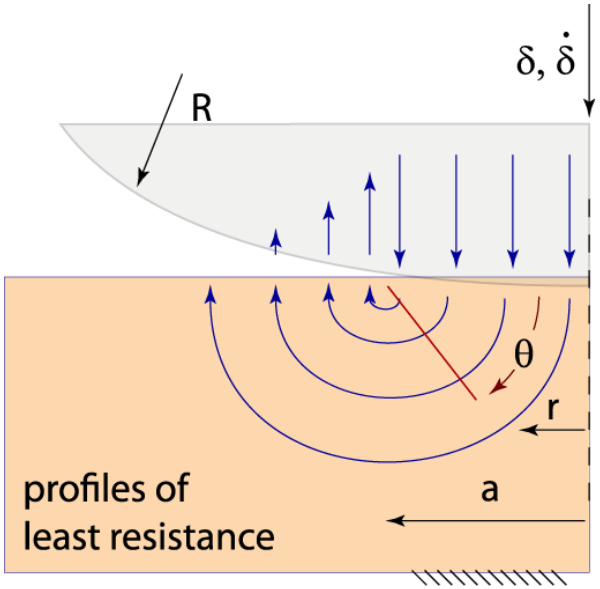
Hertzian contacts

$$F'_{max} = \frac{E^*}{E^*+1} \qquad E^* = E_t/E_c$$

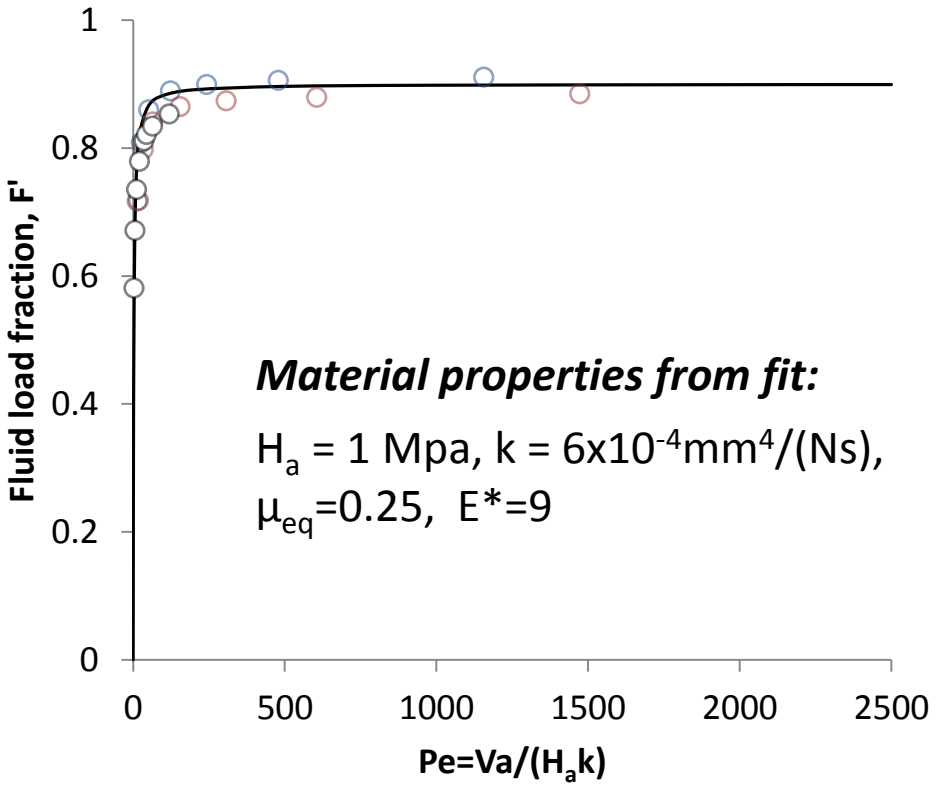
Tensile stiffness is critically important to cartilage function**

*****not widely-recognized***

Modeling, validation, gaining insights into structure/function



E.D. Bonnevie *et al.*, Biomechanics, 2012



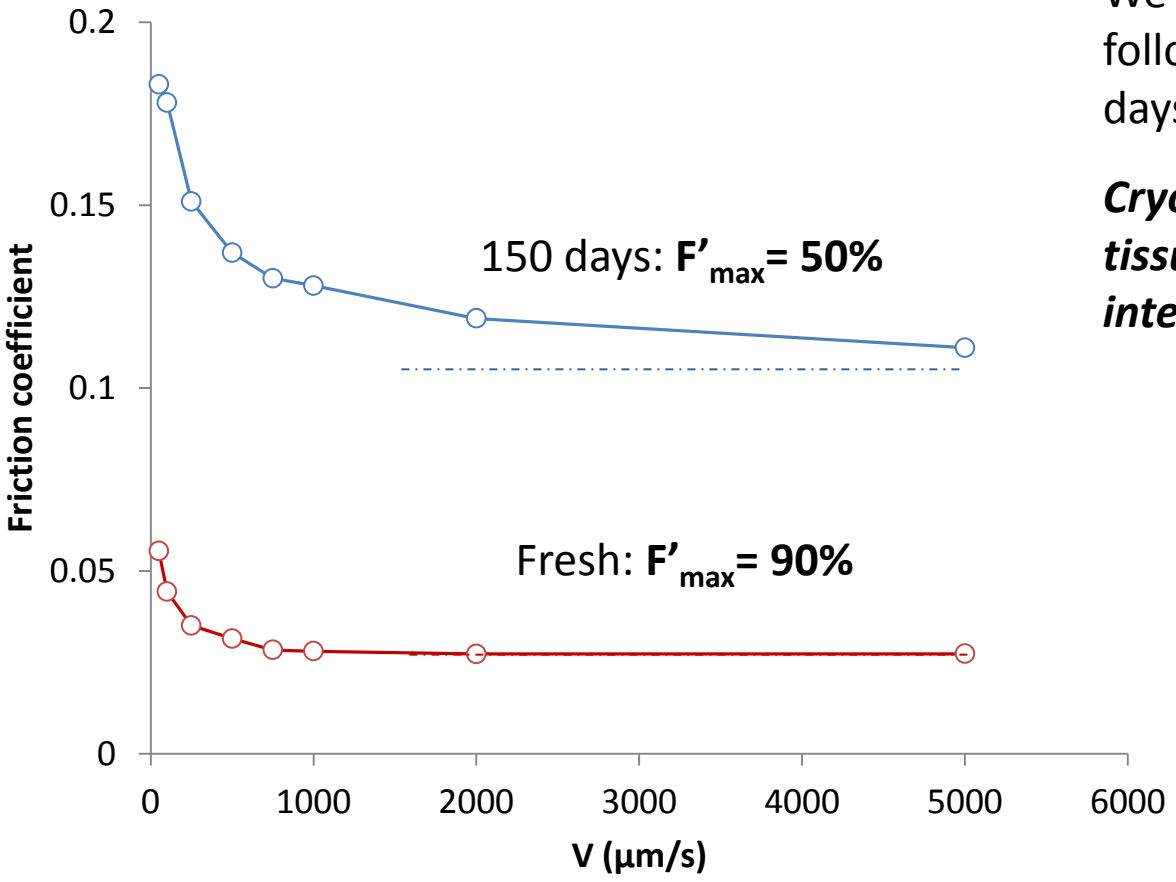
Fluid load fraction:

$$\frac{F_p}{F} = F' = \frac{Pe}{Pe+2} \qquad Pe = \frac{\dot{\delta} \cdot R}{H_a \cdot k}$$

$$F' = F'_{max} \frac{Pe}{Pe+2} \qquad F'_{max} = \frac{E^*}{E^*+1}$$

1. Indention and sliding results agree
2. Fit gives expected material properties
3. Physiological: Pe ~10,000
4. ***Dominated by E* not Ha, k***

Tissue damage disrupts lubrication

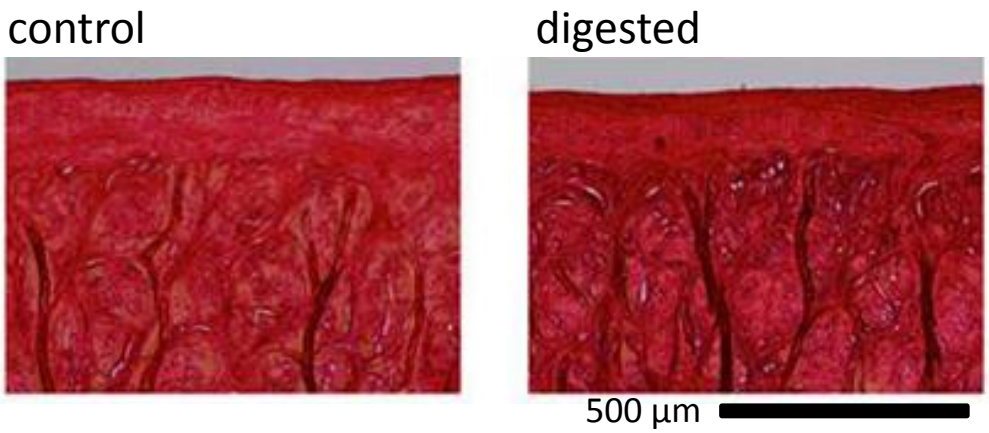


Ateshian, 2010: unclear if tissue degradation results in functional degradation (literature SCA)

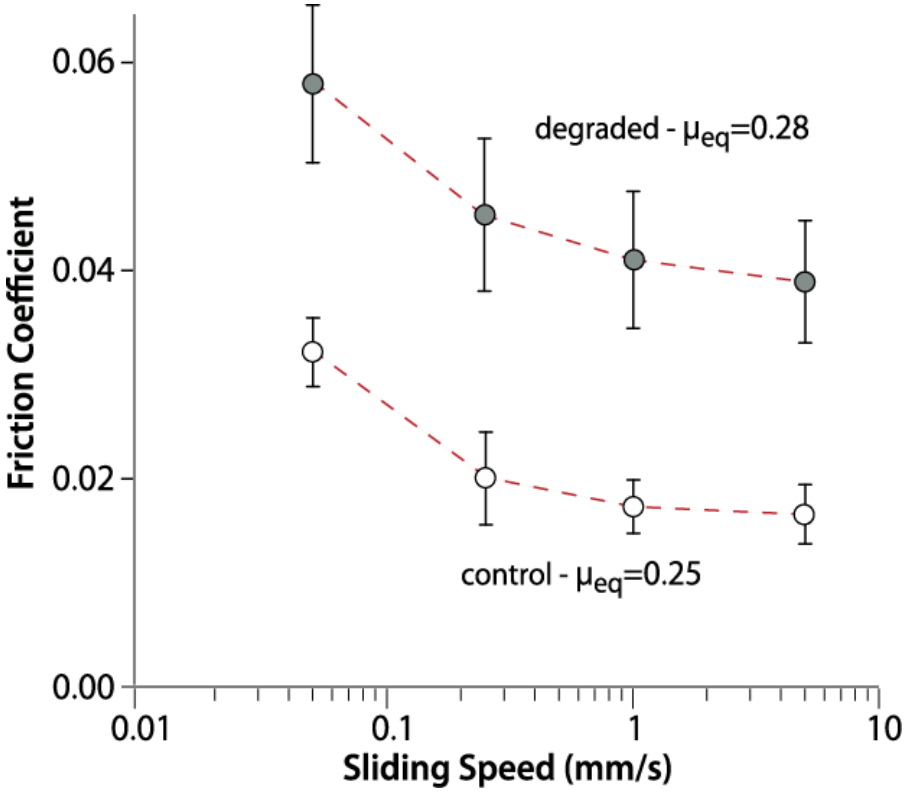
We tested a porcine TMJ condyle following extraction and after 150 days at -80°C

Cryopreservation can damage tissue and severely compromise interstitial lubrication

Functional sensitivity to intentional papain degradation



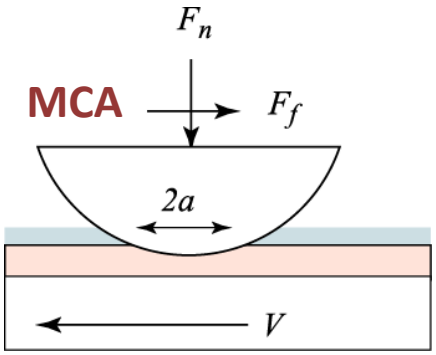
V.J. Baro *et al.*, Bone, 2012



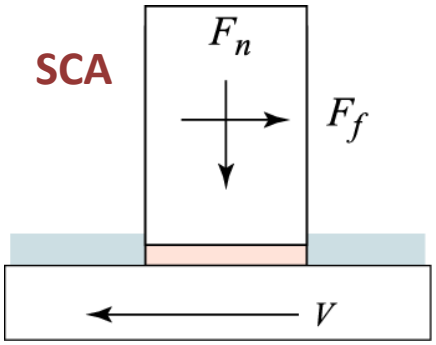
1) No visual damage
2) Aggregate modulus and total load capacity reduced (~60%)
3) Friction increased (~120%)
4) F'_{max}=93% => F'_{max}=86%
5) E*=13 => E*=6

Function is sensitive to tissue degradation

Reducing interstitial lubrication causes damage

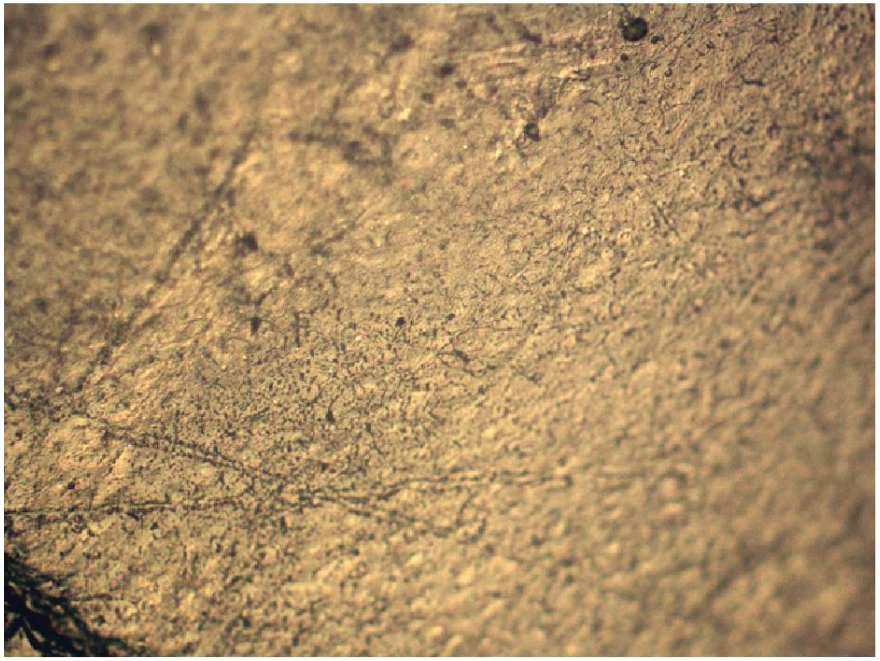


R=1.6mm
R_a=100nm
F_n=50mN
N=1,000 cycles
D=3m
P=680 kPa

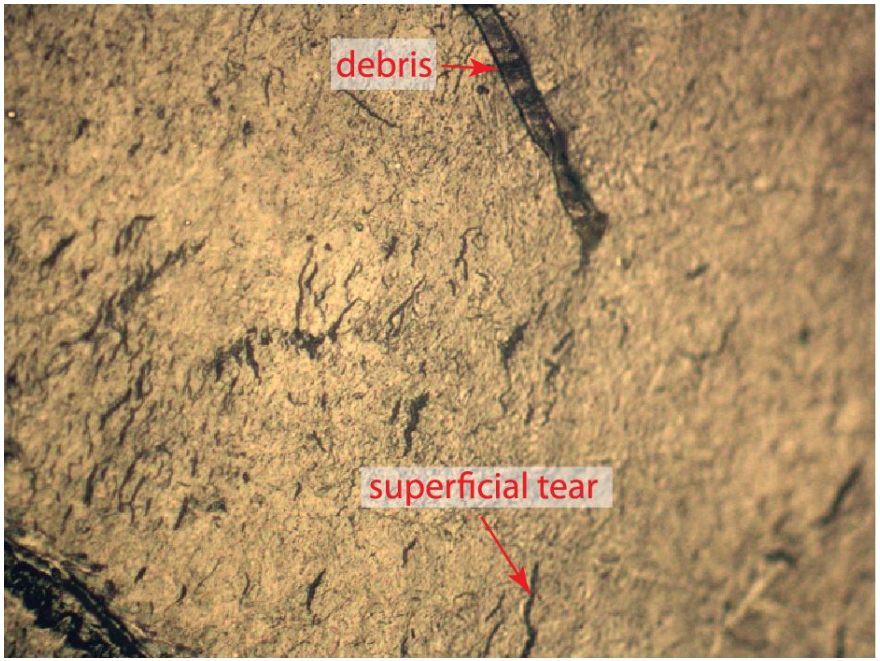


A=77mm^2
R_a=15nm
F_n=10N
N=70 cycles
D=0.7m
P=150 kPa

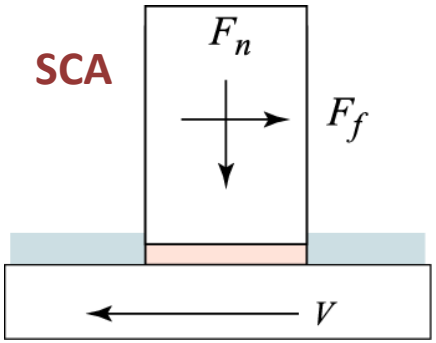
Worn MCA, 2.04MPa-m



Worn SCA, 0.105MPa-m



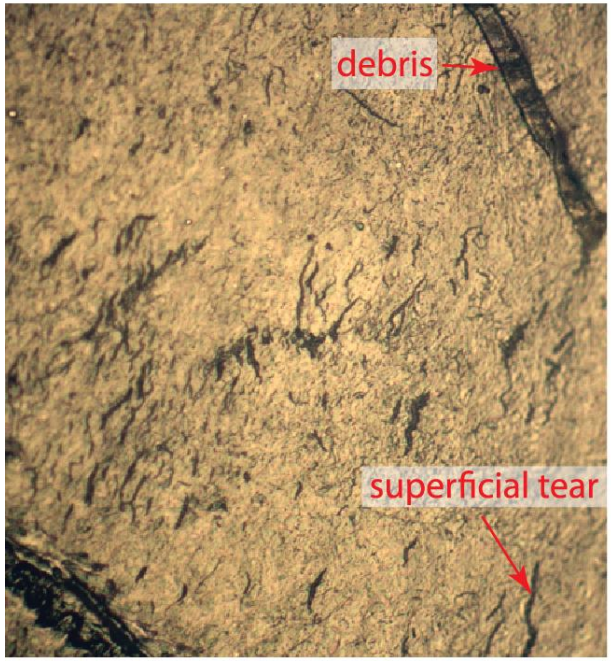
Progression of wear in the absence of interstitial pressure



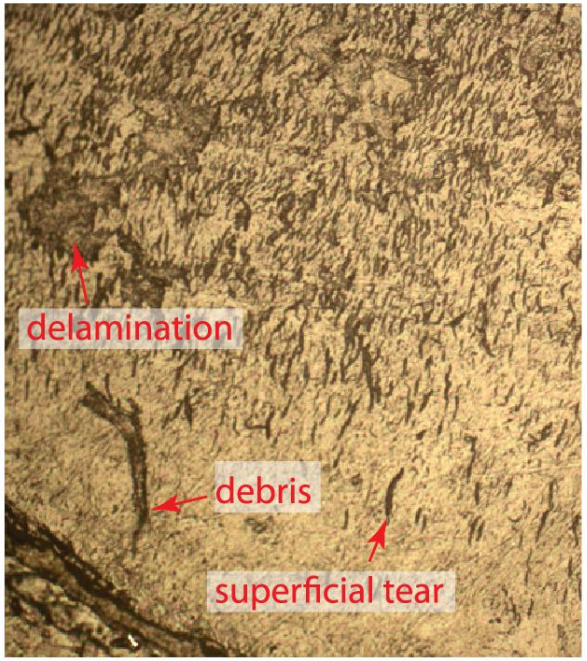
A=77mm^2
R_a=15nm
F_n=20N
N=70 cycles
D=0.7m
P=300 kPa

- Reducing interstitial pressure caused rapid surface damage
- Damage caused 3X increase in friction in subsequent MCA testing
- ***Damage disrupts lubrication which causes damage – positive feedback***

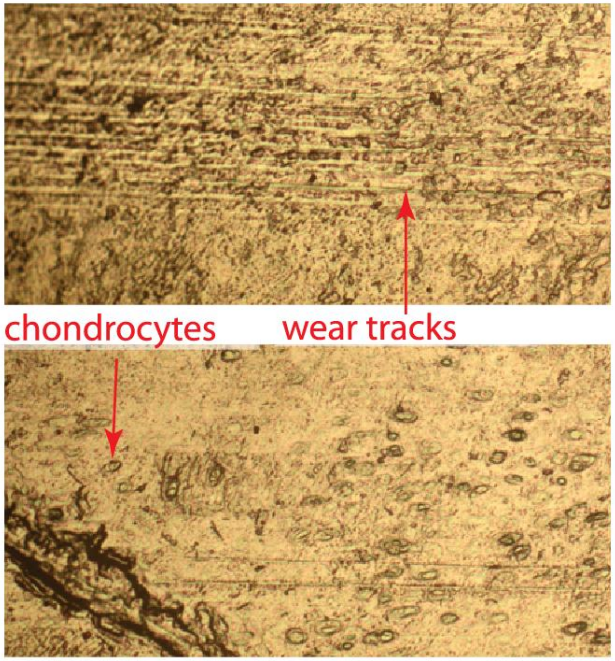
SCA, 0.105 MPa-m



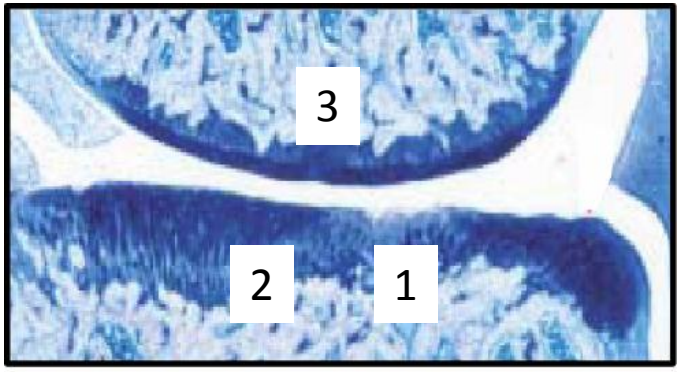
SCA, 0.135MPa-m



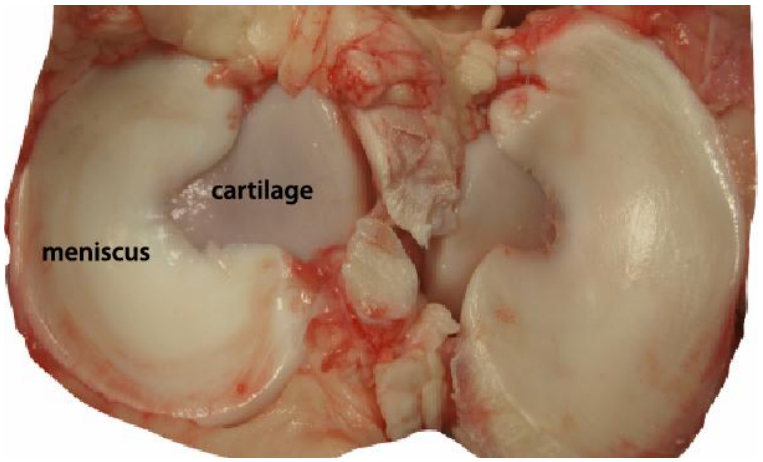
SCA, 0.285MPa-m

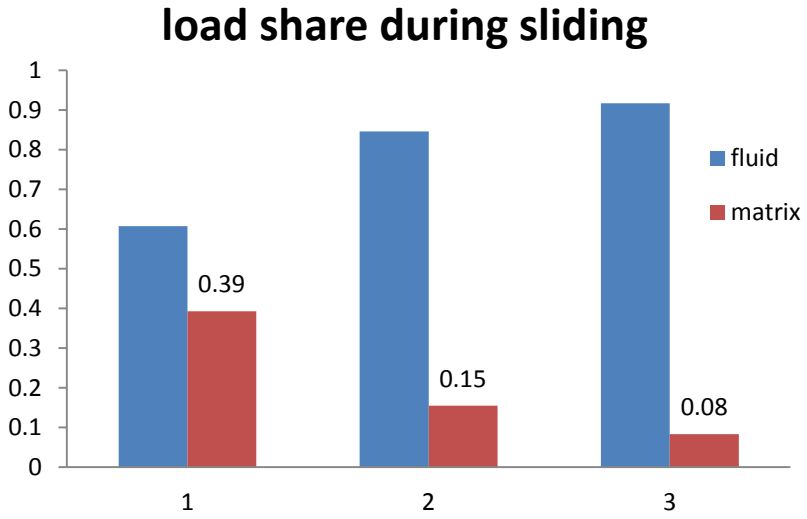


Functionality reflects regional demands



2 weeks after meniscus removed





Site 1 is buried beneath the meniscus and never experiences sliding contact. This is where damage initiates after meniscus tear (Bendele, 2001)

Site 1 has relatively poor interstitial lubrication (E*~1)

May explain altered loading as a risk factor for OA

Closing

1) Joint motion actively maintains and restores interstitial lubrication
   passive motion for therapy, rehabilitation, prevention
2) Tissue damage impedes lubrication
   impact can damage tissue initiating OA
3) Reduced interstitial lubrication increases wear by orders of magnitude
   initiated damage progresses rapidly to failure
4) Underexposed regions may be underprepared for sliding contact
   altered loading can initiate OA
5) Indicate an independent biomechanical mechanism of OA

Acknowledgements

The authors gratefully acknowledge support from COBRE Grant Number P20-RR016458 from NIH