TMS Tutorial on
Biology for Materials Scientists and Engineers
February 25, 2007

Fracture and Fatigue of Biological Materials: Bone and Teeth

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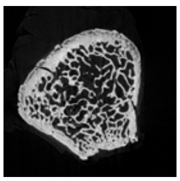

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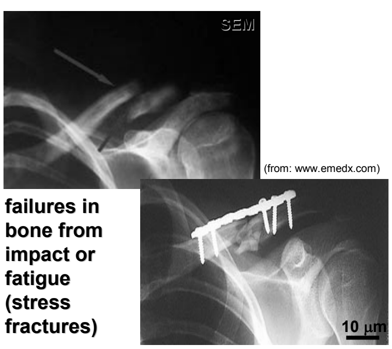
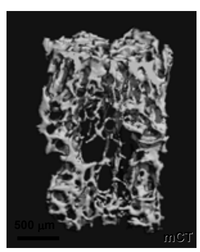
www.LBL.gov/Ritchie

The Problem!

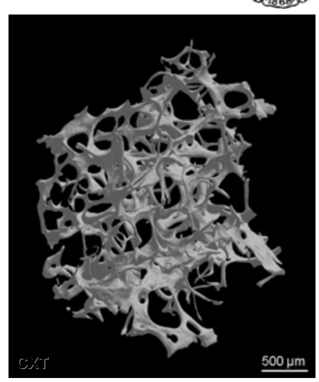


(from: www.emedx.com)

failures in bone from impact or fatigue (stress fractures)



bone damaged by steroids



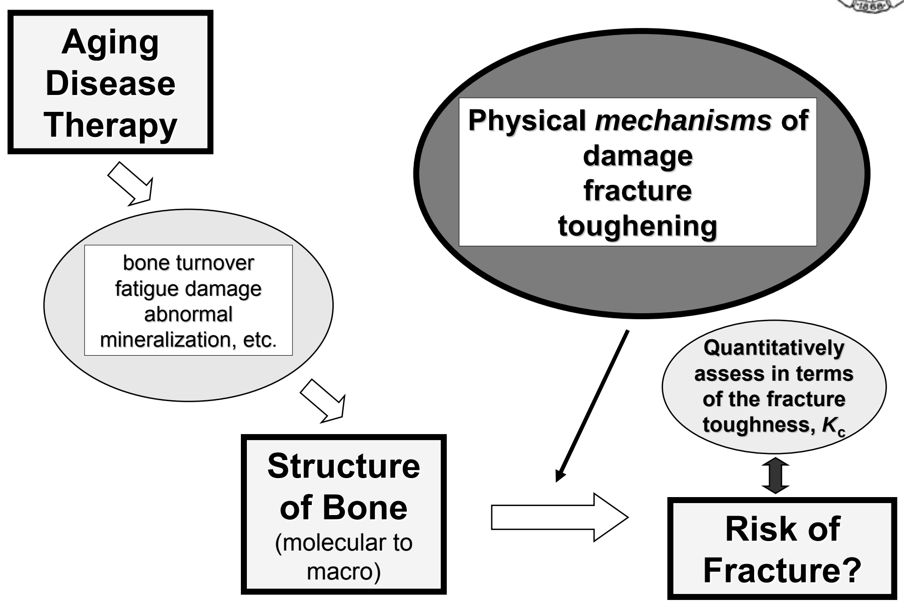
osteoporotic trabecular bone

- 10 million people in the U.S. have osteoporosis

- 1 in 2 women & 1 in 4 men over 50 will have an osteoporosis-related bone fracture over their remaining lifetime
- problem treated in terms of loss of bone mass (*bone quantity*), but this is only a part of the problem – the other issue is *bone quality*

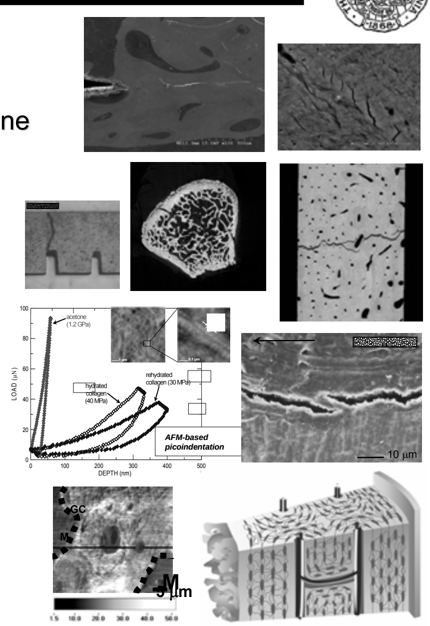
10-fold increase in fracture risk found with aging, independent of bone mineral density

Bone Quality?



Outline

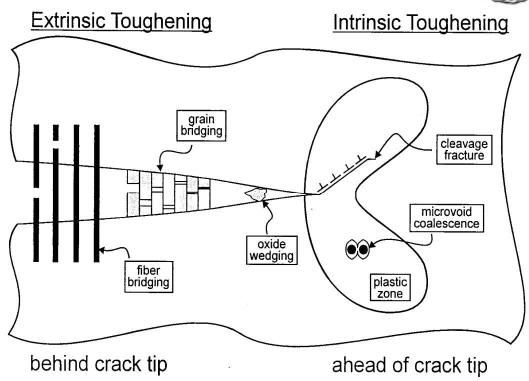
- **Introduction**
  - structural length scales in bone
- **Criteria for fracture**
  - fracture toughness
  - toughening mechanisms
- **Aging, disease and treatment**
  - effect of aging & disease
  - therapeutic treatments
- **Assessment of bone quality**
  - fracture mechanics testing
  - crack path (in bone biopsies)



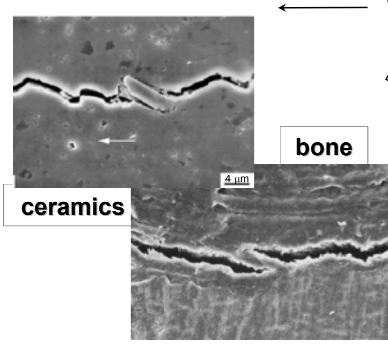
What Controls Fracture in Materials?

Fracture is a mutual competition between:

- ***intrinsic damage mechanisms*** ahead of the crack tip, that promote crack propagation, and
- ***extrinsic toughening (shielding) mechanisms*** behind the crack tip, that inhibit crack propagation



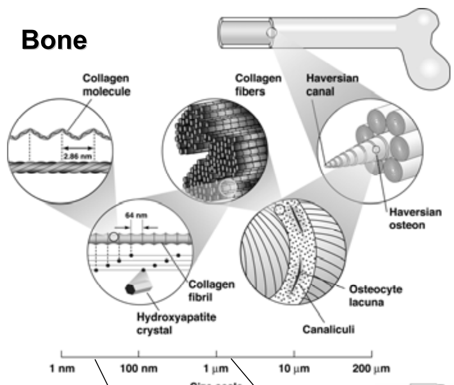
Ritchie, *Mat. Sci. Eng.*, 1988; *Int. J. Fract.*, 1999



- fracture in cortical bone shares many commonalities with structural ceramics, *i.e.,* the importance of extrinsic toughening mechanisms, principally crack bridging, and resultant resistance-curve (R-curve) behavior

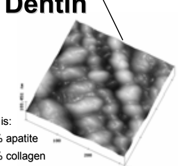
Structural Length Scales in Teeth & Bone

Complex, hierarchical structures

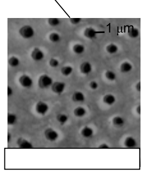


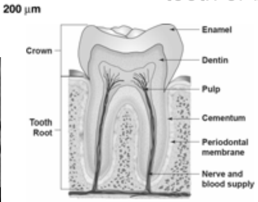
- **building blocks:** collagen & nano-crystalline hydroxyapatite mineral
- **at nanometer scale:** mineralized collagen fibrils
- **at micron scale:** lamellae structure of collagen fibers
- **at micron scale** in dentin: tubules
- **at hundreds of microns** in bone: osteons/ Haversian canals
- **at macro scale:** size and type of the tooth or bone

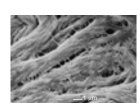
Dentin



Dentin is:
45 vol% apatite
30 vol% collagen
25 vol% fluid



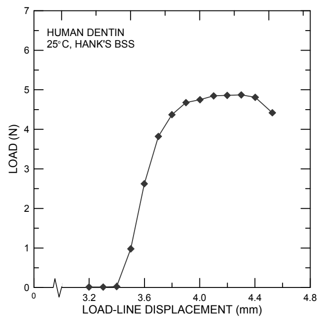


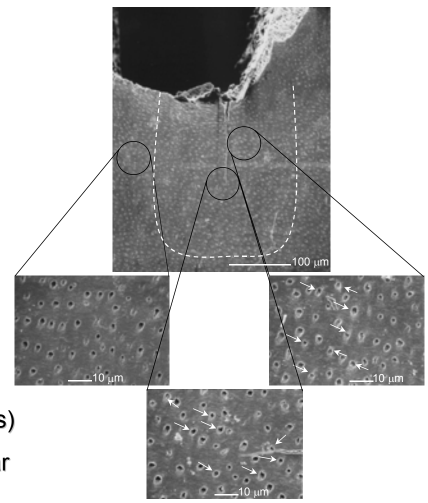


At what length scales do changes occur that lead to fracture risk?

Nature of Inelasticity in Mineralized Tissue

- **uniaxial tensile test in human dentin**



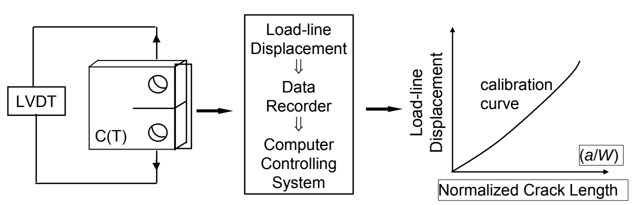


Inelastic deformation results from:

- plastic deformation (in the collagen fibrils)
- microcracking damage (at the peritubular cuffs and in the intertubular matrix)
- poro-elasticity (from fluid in the tubules)?

Nalla, Kinney & Ritchie, *J. Biomed. Mater. Res.*, 2003

Experimental Measurements

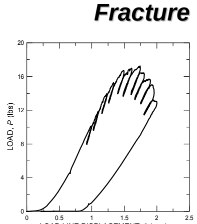


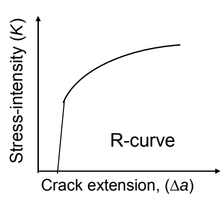
Fracture Mechanics

$$K_c = Q\,\sigma_F\,(\pi a)^{1/2}$$

where
σ_F = fracture stress
a = crack size
Q = geometry factor ~1

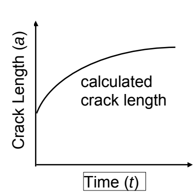
Fracture toughness, K_c

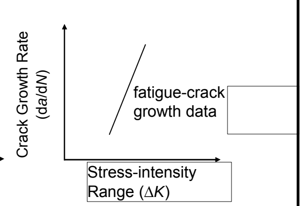




- fracture toughness assessed using crack-resistance curves (R-curves)

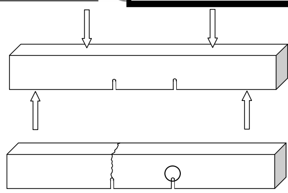
Fatigue crack-growth rates



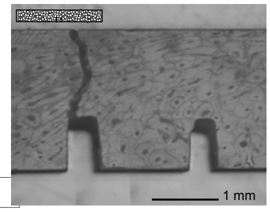


- fatigue-crack growth assessed using *da/dN* vs. ΔK plots (*v-K* curves)

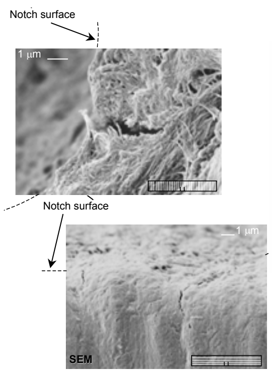
Mechanisms of Fracture Initiation



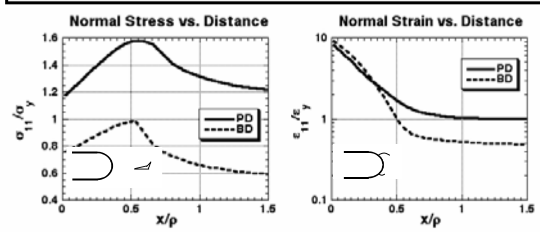
double-notch four-point bend test



- two identical notches in a four-point bend bar
- constant bending moment on both notches
- one notch breaks - the other *freezes* local fracture events just prior to fracture



with inelasticity, stresses peak ahead of notch, strains peak at notch



PD: pressure-insensitive plasticity

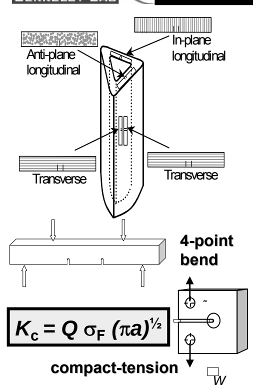
BD: pressure-sensitive microcracking inelasticity

Distance ahead of notch, normalized by the notch-root radius

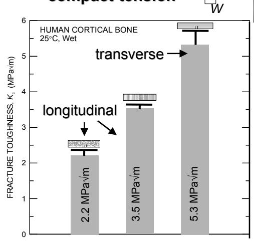
- crack initiation directly *at* the notch root implies that *initiation of fracture* in human cortical bone and dentin is locally ***strain-controlled***

Nalla, Kinney & Ritchie, *Nature Mat*., 2003; *J. Biomed. Mater. Res.*, 2003

Fracture Toughness of Human Bone

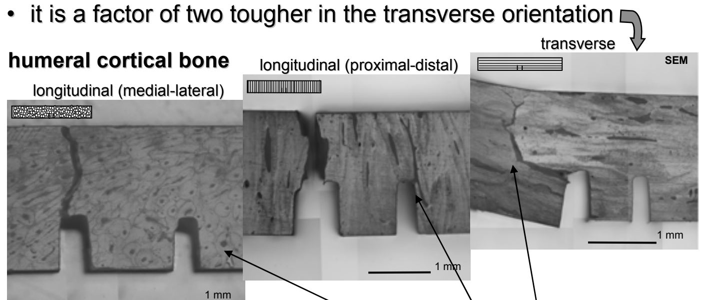


$K_c = Q\,\sigma_F\,(\pi a)^{1/2}$



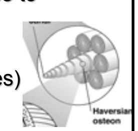
- bone is tougher in certain directions
- it is much more difficult to break than to split
- it is a factor of two tougher in the transverse orientation

humeral cortical bone

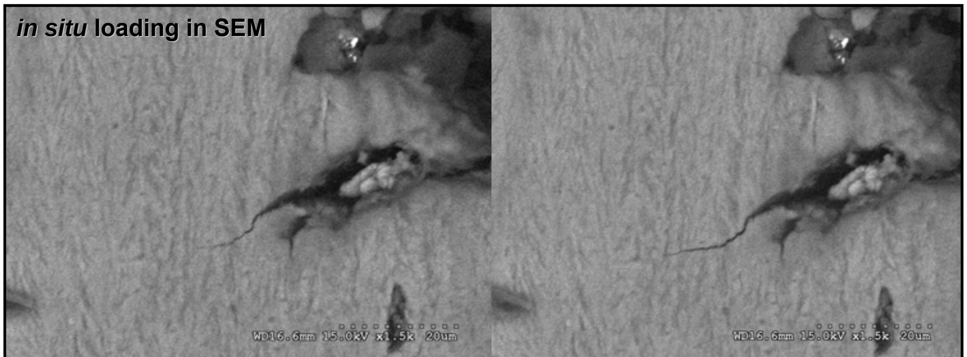


osteons interfaces are sites of preferred cracking

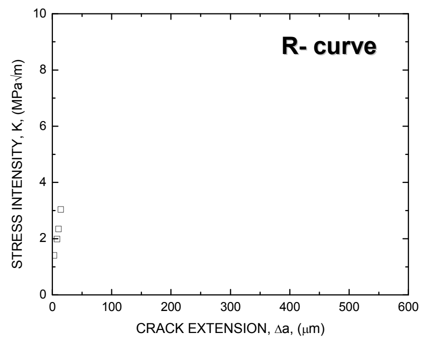
- marked effect of orientation on toughness, *i.e.,* resistance to fracture, due to anisotropic nature of microstructure
- much higher toughness in transverse orientation due to crack deflection along the cement lines (osteon interfaces)
- *"stress-raisers" are not necessarily bad – it's the overall crack path that matters!*



Nalla, Stölken, Kinney & Ritchie, *J. Biomechanics*, 2005 *in situ* loading in SEM

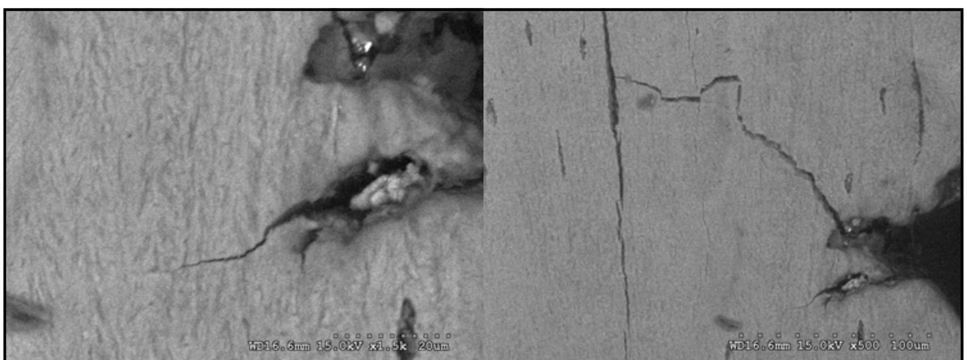


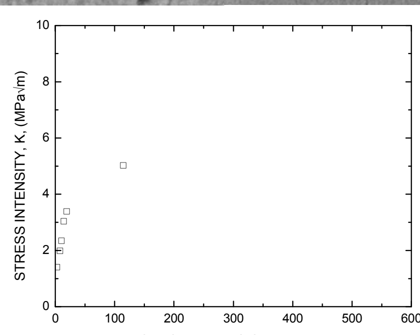
Human cortical bone

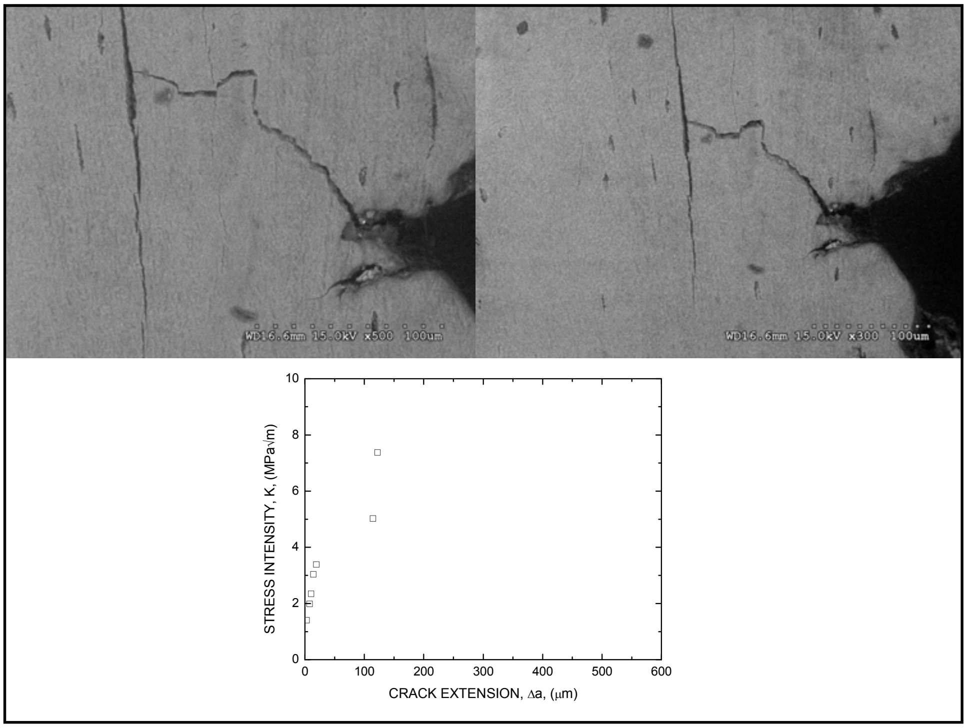


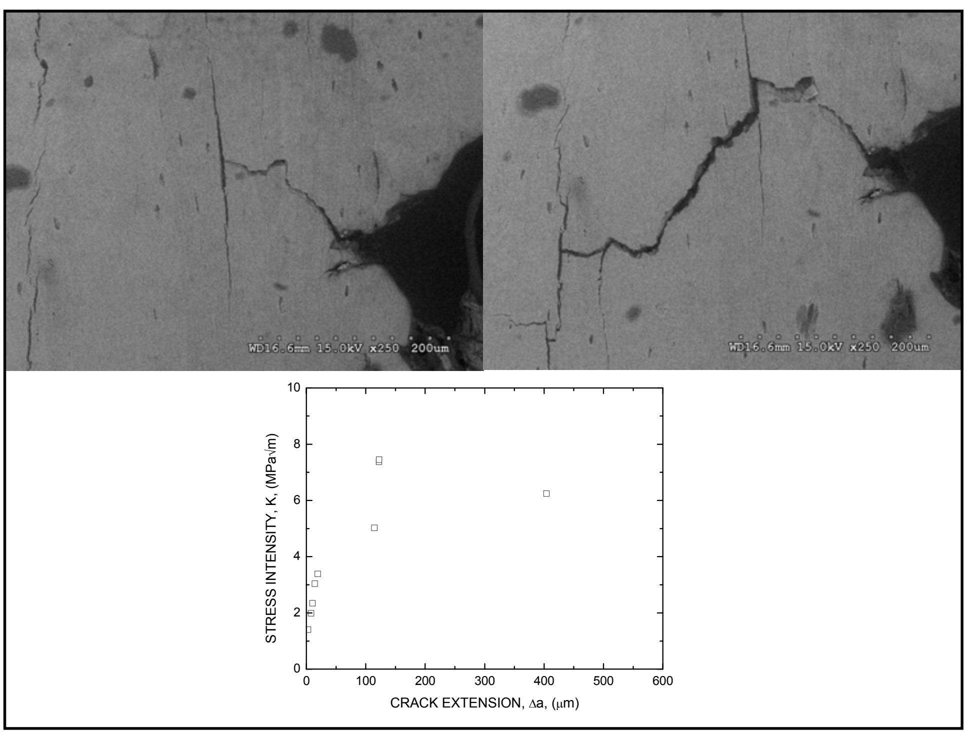
10 μm

Koester, Ager, & Ritchie, 2006






WD16.6mm 15.0kV x500 100um
WD16.6mm 15.0kV x300 100um
STRESS INTENSITY, K, (MPa√m)
CRACK EXTENSION, Δa, (μm)
0
2
4
6
8
10
0
100
200
300
400
500
600


WD16.6mm 15.0kV x250 200um
WD16.6mm 15.0kV x250 200um
STRESS INTENSITY, K, (MPa√m)
CRACK EXTENSION, Δa, (μm)
0
2
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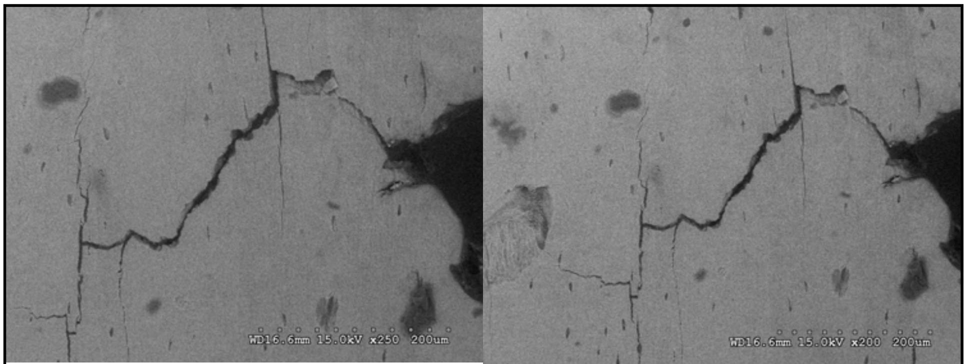
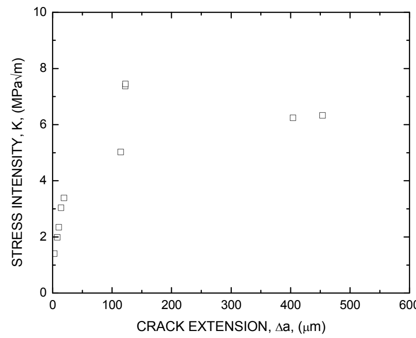
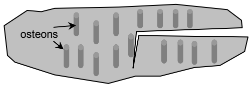




Origins of Toughening in Bone

Contribution to Toughness

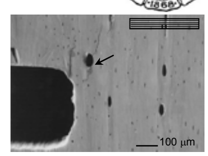
Crack Deflection



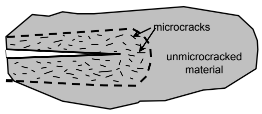
$k_1(\alpha) = c_{11}(\alpha)\, K_I + c_{12}(\alpha)\, K_{II}$
$k_2(\alpha) = c_{21}(\alpha)\, K_I + c_{22}(\alpha)\, K_{II}$
$K_d = (k_{12} + k_{22})^{1/2}$

(Bilby *et al.*, 1978; Cottrell & Rice, 1980)

3.0 MPa√m
(transverse)



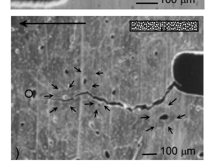
Constrained Microcracking



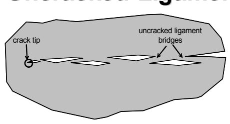
$K_{mic} = 0.22\, \varepsilon_m E' f_m\, l_m^{1/2} + \beta\, f_m K_c$

(Evans & Fu, 1985: Hutchinson, 1987)

~0.05 MPa√m



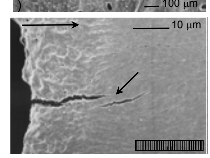
Uncracked-Ligament Bridging



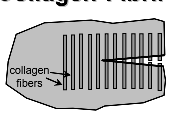
$K_b^{ul} = -f_{ul} K_I [(1+l_u/rb)^{1/2} - 1] \,/\, [1 - f_{ul} + f_{ul}(1+l_{ul}/rb)^{1/2}]$

(Shang & Ritchie, 1989)

1-1.5 MPa√m
(proximal-distal)



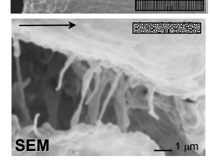
Collagen-Fibril Bridging



$K_b^f = 2\, \sigma_b\, f_f\, (2\, l_f / \pi)^{-1/2}$

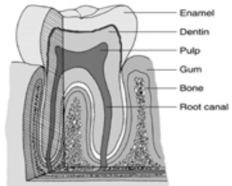
(Evans & McMeeking, 1986)

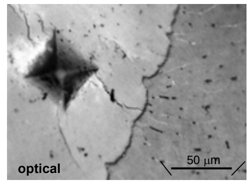
0.1 MPa√m
(medial-lateral)

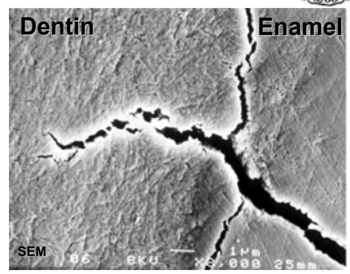


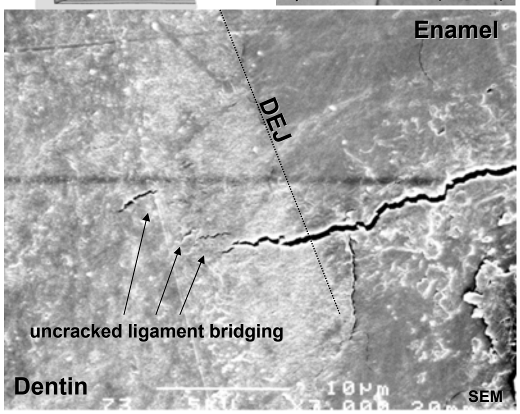
Nalla, Kinney & Ritchie, *Nature Mat.*, 2003; Nalla, Stölken, Kinney & Ritchie, *J Biomechanics*, 2005

Crack Arrest in the DEJ Region in Teeth





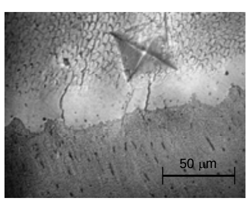




- cracks in harder enamel do not necessarily break the tooth as they arrest "at" the dentin-enamel junction (DEJ)
- cracks arrest when they form elastic bridges in the (mantle) dentin due to the formation of uncracked ligaments in the crack wake

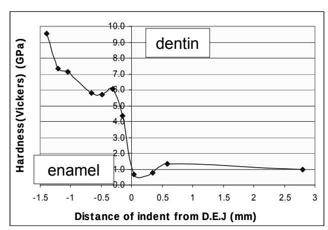
Imbeni, Kruzic, Marshall, Marshall & Ritchie, *Nature Mat.*, 2005

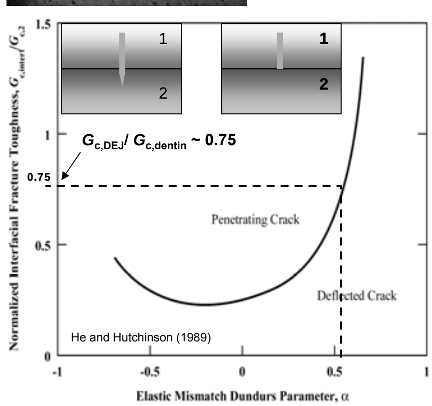
Toughness of the DEJ Region in Teeth



- Toughness of the DEJ assessed from the elastic mismatch between dentin and enamel by whether the crack deflects, arrests or penetrates the interface

after Sun, Becher *et al.*





Elastic mismatch:

$\alpha = (E_1 - E_2)/(E_1 + E_2)$

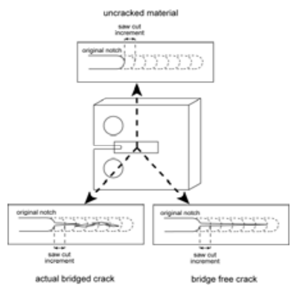
E'_{enamel} (GPa)	E'_{dentin} (GPa)	α	$G_{c,enamel}$ (J/m^2)	$G_{c,dentin}$ (J/m^2	$G_{c,DEJ}$ (J/m^2)
64	20	0.53	~ 25	154	115

- upper-bound toughness of the DEJ estimated to be $G_{c,DEJ}$ ~ 115 J/m^2
- toughness of the DEJ is intermediate between enamel and dentin
- it is ~5-10 times higher than enamel but still 75% of dentin

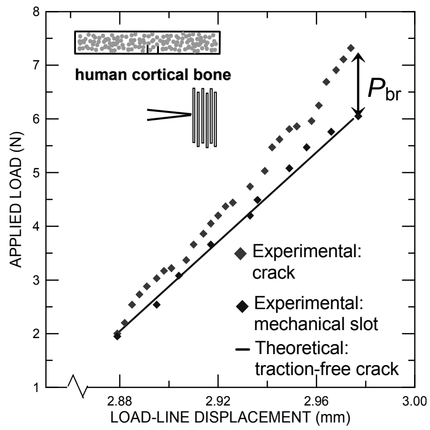
Imbeni, Kruzic, Marshall, Marshall & Ritchie, *Nature Mat.*, 2005

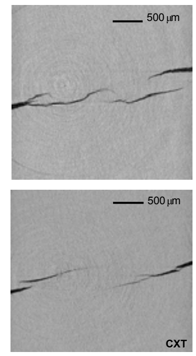
Experimental Proof of Crack Bridging

multi-cutting compliance



crack bridging in human cortical bone verified using a compliance-based technique



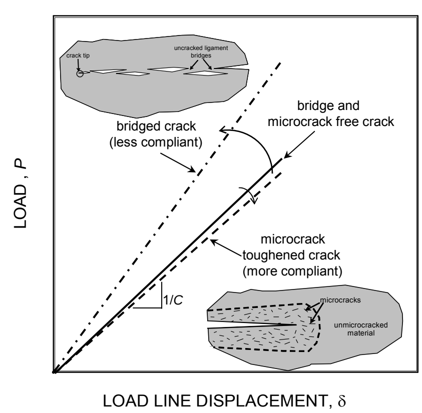


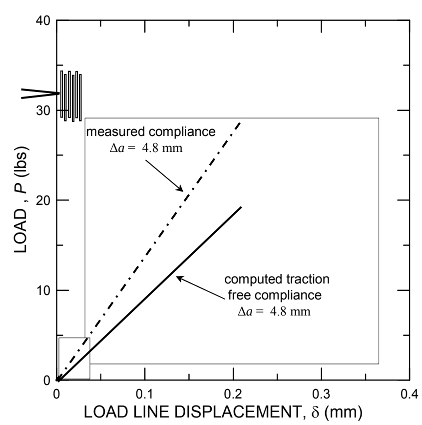
2-D tomographic slices of uncracked-ligament bridged cracks in dentin

- compliance of actual crack is measured before and after machining the wake; results compared to theoretical compliance of traction-free crack (of same length)
- bridging contribution to toughness of bone measured at K_{br} ~ 0.5 – 1 MPa√m, and occurs over large length scales (hundreds of microns)

Kruzic, Nalla, Kinney & Ritchie, *Biomaterials,* 2004

Crack Bridging vs. Constrained Microcracking



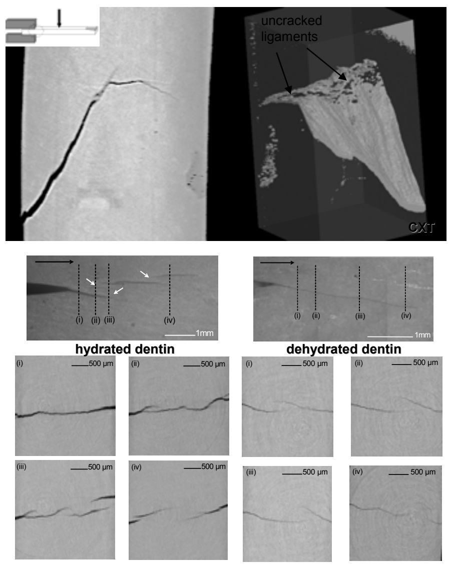


- microcracking based explanation for toughening prevalent in the literature
- crack bridging will reduce compliance, *C*; microcracking will increase compliance
- supports bridging as the main toughening mechanism, rather than microcracking

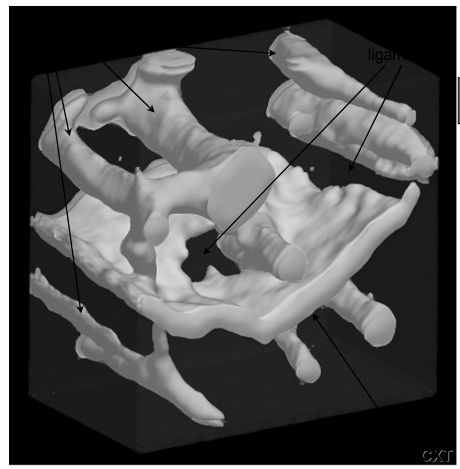
Nalla, Kruzic, Kinney & Ritchie, *Bone,* 2004

Tomographic Evidence of Crack Bridging

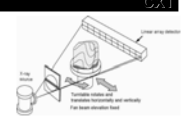
human dentin



human cortical bone



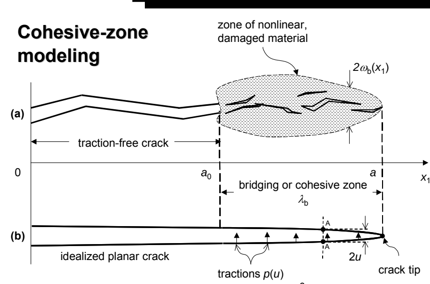
X-Ray Computed Tomography, performed at the Stanford Linear Accelerator Center and Advanced Light Source (LBNL)



Kruzic, Nalla, Kinney & Ritchie, *Biomaterials,* 2003; Nalla, Kruzic, Kinney & Ritchie, *Biomaterials*, 2005

Resistance-Curve Toughness Behavior

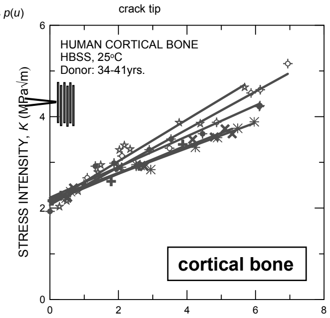
Cohesive-zone modeling

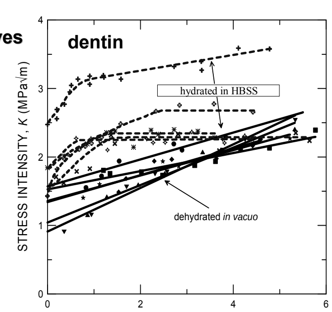


- as bridging zones are ~hundreds of microns in size, they can be comparable with the size of the bone (or tooth) - quoted (single-value) K_{Ic} fracture toughness values are thus likely size- and geometry dependent

Resistance-curves

- presence of crack-bridging does result in crack-size dependent behavior:
  - rising R-curves
  - small-crack effects



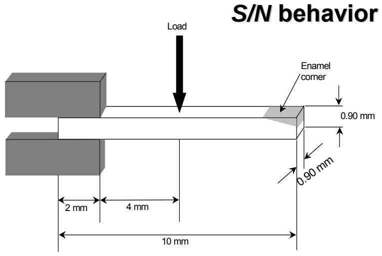


Nalla, Kruzic & Ritchie, *Bone*, 2004
Kruzic, Nalla, Kinney & Ritchie, *Biomaterials*, 2003

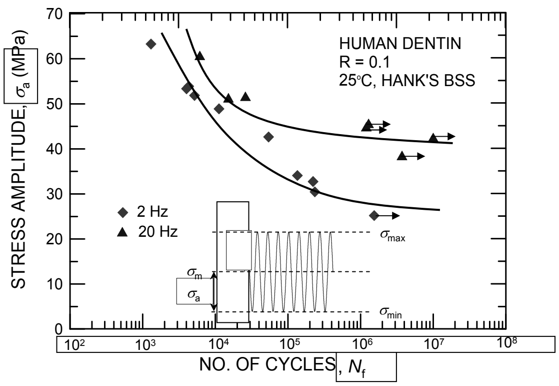
Qang, Cox, Nalla & Ritchie, *Biomaterials, 2006; Bone*, 2006

Fatigue of Mineralized Tissue

S/N behavior



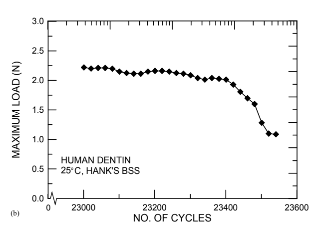
load ratio $R = \sigma_{min}/\sigma_{max}$



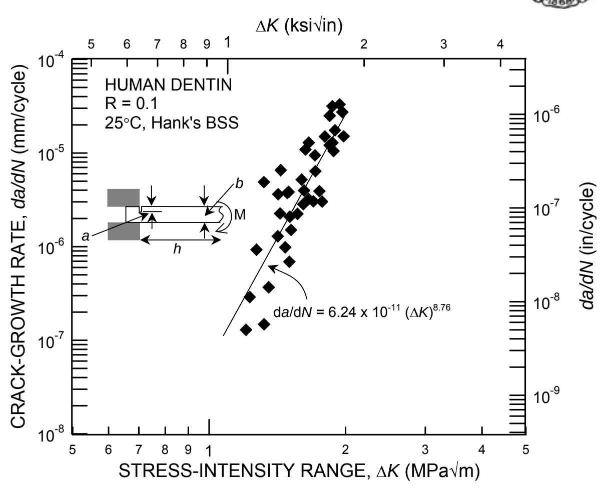
- clear evidence of premature fatigue failure of both human teeth and bone
- not clear whether this is a cycle- or time-dependent phenomenon
- "metal-like" fatigue *S/N* behavior with frequency-dependent fatigue limit at 10^6-10^7 cycles of ~25 and 45 MPa
- comparable at lower frequency to typical masticatory stress levels (~20 MPa)
- fatigue lives, in terms of cycles to failure, are shorter at lower frequency

Nalla, Imbeni, Kinney, Staninec, Marshall & Ritchie, *J. Biomed. Mater. Res.*, 2003

Fatigue-Crack Growth in Human Dentin



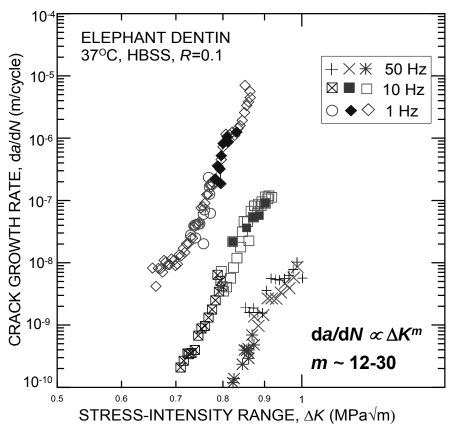
- decay in stiffness used to estimate crack lengths from smooth-bar *S/N* tests

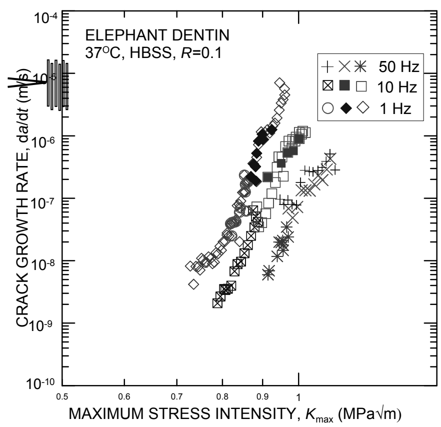


- Paris power-law relationship, $da/dN = C\ \Delta K^m$, where exponent m ~ 8.76
- estimated fatigue threshold, ΔK_{TH} ~ 1.06 MPa√m, ~60% of the fracture toughness

Nalla, Imbeni, Kinney, Staninec, Marshall & Ritchie, *J Biomed Mater Res*, 2003

Fatigue-Crack Growth Data in Dentin



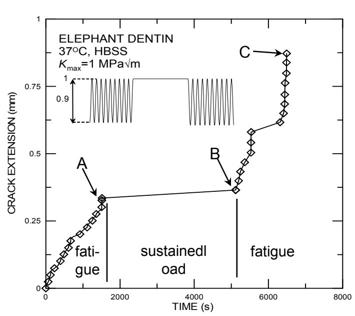


- effect of frequency seen in "per cycle" & "per time" data from 1-50 Hz
- as in many materials, growth rates depend upon both ΔK and K_{max}

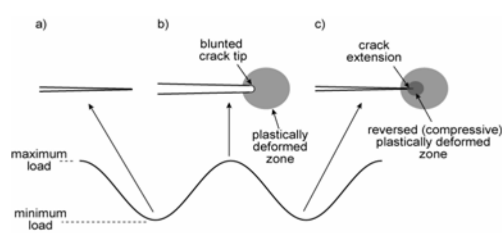
Kruzic, Nalla, Kinney & Ritchie, *Biomaterials,* 2005

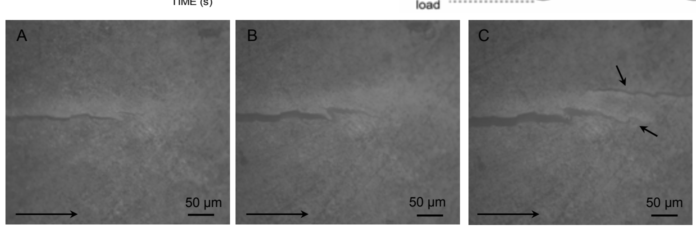
Cyclic/Static Loading Experiments in Dentin

- at constant K_{max}, crack barely propagates when unloading cycle is removed



- crack tends to blunts under static loading
- crack "sharpens" under cyclic loading

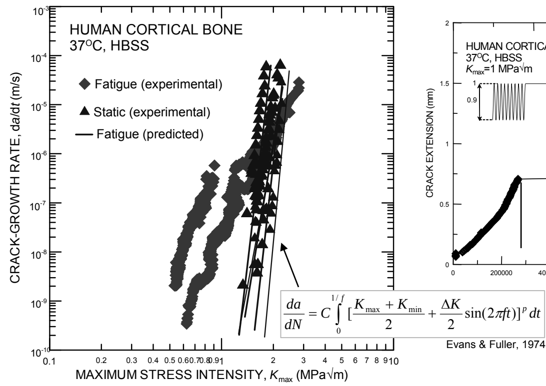


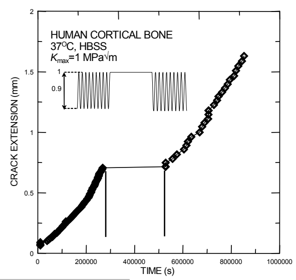


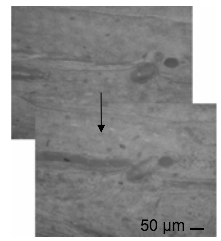
- clear evidence of cycle-dependent fatigue mechanism
- also evidence of a deterioration in the fraction of bridging ligaments

Kruzic, Nalla, Kinney & Ritchie, *Biomaterials,* 2005

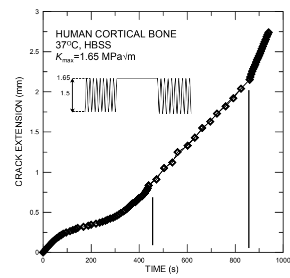
Fatigue vs. Slow Crack Growth in Bone







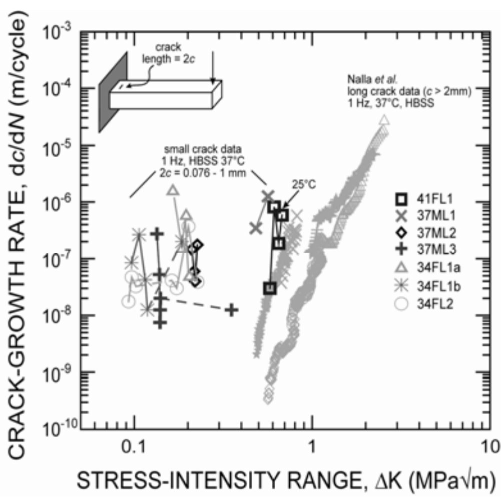
crack-tip blunting



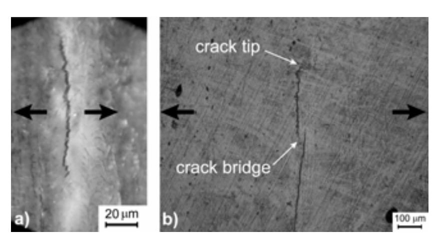
- high growth rates – time-dependent static mechanisms (creep-dominated)
- low growth rates – cycle-dependent fatigue mechanisms (fatigue-dominated)
- crack growth by alternating blunting & resharpening and cycle-induced damage of bridging ligaments

Nalla, Kruzic, Kinney & Ritchie, *Biomaterials,* 2005

Fatigue of Small Surface Cracks



- small surface cracks (~70-1000 μm) grow faster than large (through-thickness) cracks (> 2 mm) at the same ΔK
- such microcracks grow intermittently and locally arrest at microstructural features, e.g., at the osteon structures

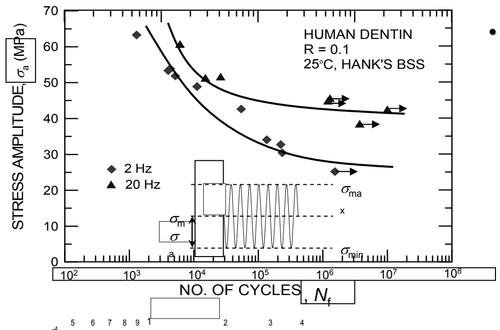


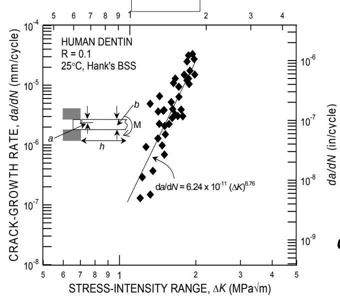
small crack large crack

- their faster growth rates can be attributed to their limited wake, which restricts the development of crack bridging

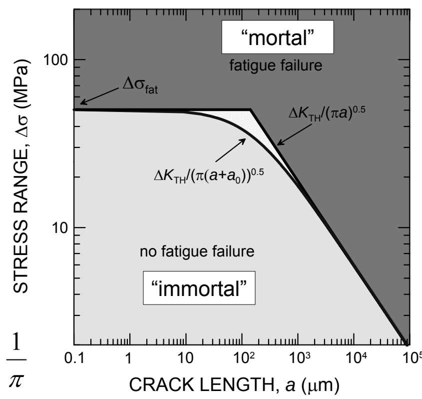
Kruzic, Scott, Nalla, & Ritchie, *J. Biomechanics,* 2006

Kitagawa Diagram for Fatigue of Dentin





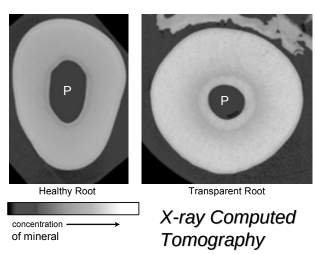
- limiting conditions for fatigue failure can be defined by the fatigue threshold ΔK_{TH} at large crack sizes (> 150 μm) and by the smooth-bar fatigue limit $\Delta\sigma_{fat}$ at small crack sizes



$$a_0 = \left(\frac{\Delta K_{\mathrm{TH}}}{Y\Delta\sigma_{\mathrm{fat}}}\right)^2 \frac{1}{\pi}$$

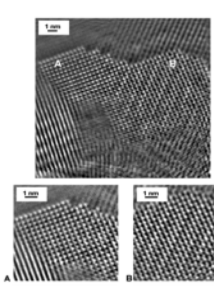
Kruzic & Ritchie, *J. Biomedical Materials Research,* 2006

Effect of Aging on Dentin - Transparency



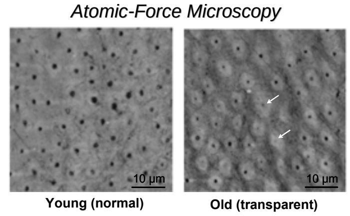
X-ray Computed Tomography

- aging leads to an altered form of dentin – *transparent dentin*
- mineral concentration increases and distribution changes, due to filling up of tubules with mineral
- concentration differences due to crystallite size being slightly smaller in transparent dentin
- collagen environment is changed in terms of intrafibrillar mineral & overall density of fibrils

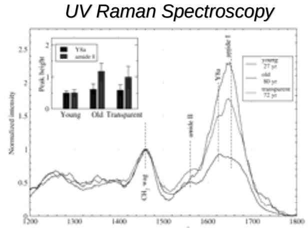


FIB & Transmission Electron Microscopy

Atomic-Force Microscopy



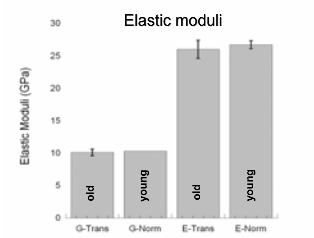
UV Raman Spectroscopy



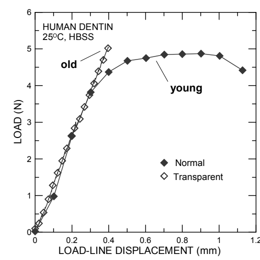
- reconstructed exit-wave lattice images images of intratubular mineral in transparent dentin showing evidence of nanometer-sized single-crystal apatite grains (& Mg-rich β-tricalcium phosphate)

Porter, Nalla, Minor, Jinschek, Kisielowski, Radmilovic, Kinney, Tomsia & Ritchie, *Biomaterials*, 2005

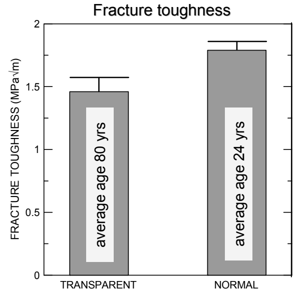
Effect of Aging in Dentin: Property Changes

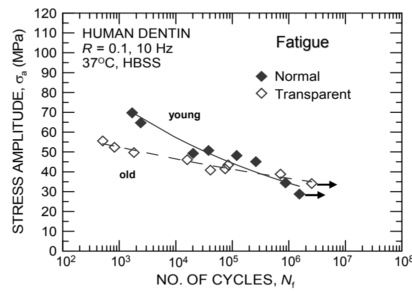


Resonance Ultrasound Spectroscopy

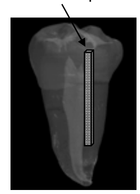


- Young's and shear modulus unchanged with transparency
- normal dentin "yields", with extensive post-yield deformation
- transparent (old) dentin is brittle - no yielding
- fracture toughness is ~20% lower in transparent dentin
- fatigue resistance generally lower in transparent dentin





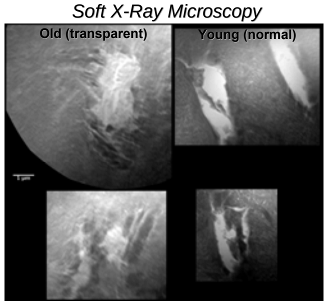
test samples



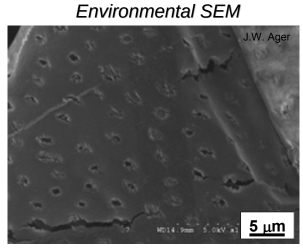
Kinney, Nalla, Pople, Breunig & Ritchie, *Biomaterials*, 2005

Effect of Aging on the Toughness of Dentin

Soft X-Ray Microscopy

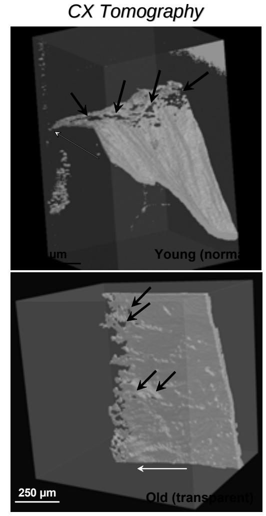


Environmental SEM

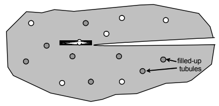


crack path follows tubules

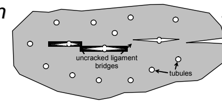
CX Tomography



- with aging, mineral concentration in dentin increases due to filling up of tubules with nanocrystalline apatite (transparent dentin)
- post-yield stress/strain behavior eliminated
- fracture toughness and fatigue resistance is reduced



old dentin

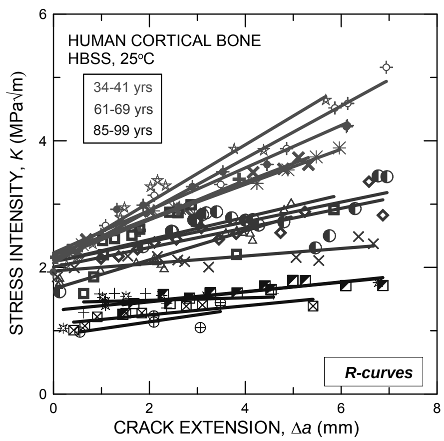


young dentin

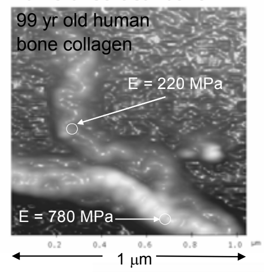
- aging leads to reduced crack bridging, consistent with reduction in fracture toughness
- filled tubules in aged dentin become less effective stress-concentrators

Porter, Nalla, Minor, Radmilovic, Kinney, Tomsia & Ritchie, *Biomaterials*, 2005; Kinney, Nalla, Pople, Breunig & Ritchie, *Biomaterials*, 2005

Effect of Aging on Human Bone

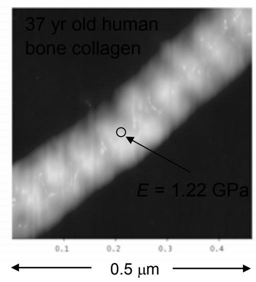


humeral cortical bone



Bone, Dec. 2004

pico-force AFM-based nano-indentation

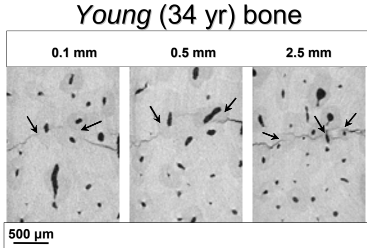


- fracture toughness, and hence risk of fracture, of human bone severely degraded by age
- significant deterioration in the collagen
- effect may be related to excessive remodeling with age, which increases the osteon density

Nalla, Kruzic, Kinney & Ritchie, Bone 2004; Nalla, Kruzic, Ager, Balooch, Kinney & Ritchie, *Mat. Sci. Eng. C*, 2006

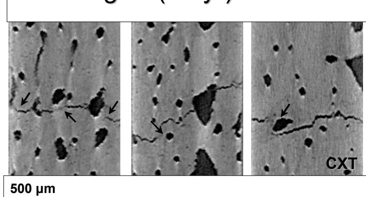
Deterioration in Human Bone from Aging

Young (34 yr) bone

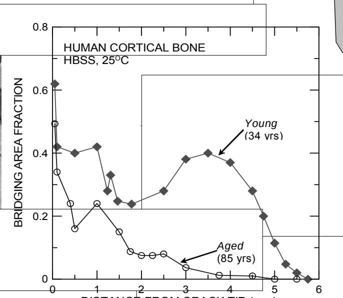


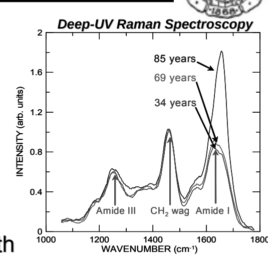
- size and area fraction of crack bridges decrease with age
- possibly from excessive remodeling → higher fraction of osteons
- significant correlation of decrease in toughness with increase in osteon density

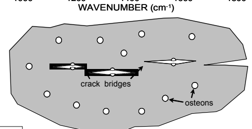
Aged (85 yr) bone

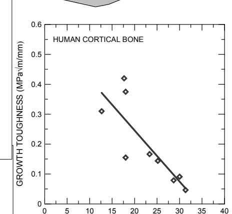


2-D tomographs show progressively fewer and smaller bridges in older bone









Nalla, Kruzic, Ager, Balooch, Kinney & Ritchie, *Mat. Sci. Eng. C*, 2006

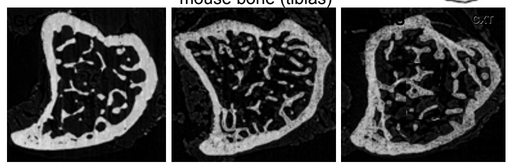
Nalla, Kruzic, Kinney & Ritchie, *Bone*, 2004

Deterioration in Bone from Steroids

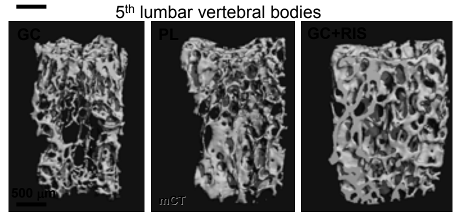
(Nancy Lane, UCDavis)

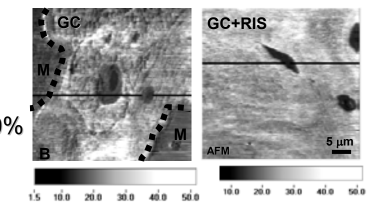
- **Glucocorticoids (GC)** are steroid hormones widely used for inflammatory diseases, such as arthritis & dermatitis
- clinical studies show increased risk of bone fracture (GC-induced osteoporosis)
- GCs induce slower bone turnover by suppressing bone formation
- **Bisphosphonates, e.g., Risedronate (RIS),** are effective therapies, inhibiting bone resorption and reducing fracture risk
- GCs lead to "soft spots" - halos of low stiffness hypo-mineralized bone around larger osteocyte lacunae – toughness (mouse femurs) reduced 20%
- concurrent RIS treatment suppress such "soft spots" - toughness is increased by 25%

mouse bone (tibias)



5[th] lumbar vertebral bodies



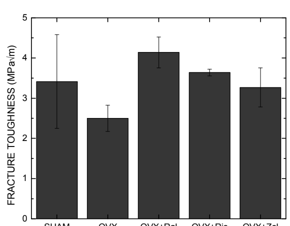


Balooch, Yao, Ager, Balooch, Nalla, Porter, Mastroianni, Ritchie & Lane, *Arthritis & Rheumatism,* 2007

Effect of Raloxifene, Risedronate and Zoledronate on Estrogen-Deficient Bone

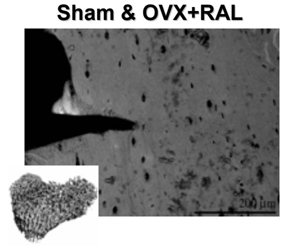
(Nancy Lane, UCDavis)

- study on rat femurs, ovarectomized (OVX) at 18 mts
- given Raloxifene (RAL) or bisphosphonates - Risedronate (RIS) or Zoledronate (ZOL) - immediately afterwards, tested after 60 days
- RAL offsets estrogen-deficiency; RIS & ZOL inhibit bone resorption

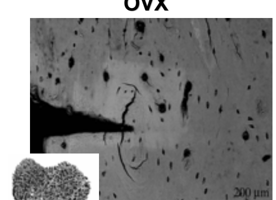


Yao, Balooch, Koester, Ritchie, Lane, *et al.* 2006

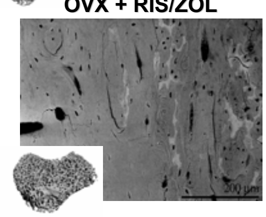
Sham & OVX+RAL



OVX



OVX + RIS/ZOL



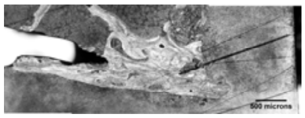
- compared to Sham, crack path, which has a radical effect on toughness, markedly different in RIS & ZOL treated bone
- crack paths are very tortuous in RIS & ZOL-treated cortical bone

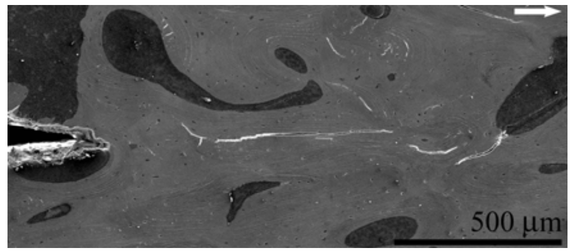
Loss in bone-matrix toughness due to ovarectomy more than compensated by Raloxifene, Risedronate or Zoledronate treatments

Fracture Risk Assessment from Biopsies

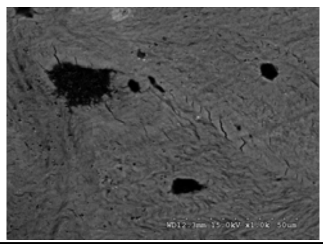
iliac crest biopsies

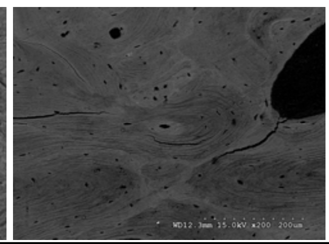
(supplied by Juliet Compston (Camb) via Nancy Lane (UC Davis))





- we can induce stable cracks in cortical bone
- crack path, *c.f.,* micro-structure, used to assess toughening or deterioration
- we believe that we can measure a K_c as a quantitative measure of bone quality for living patients



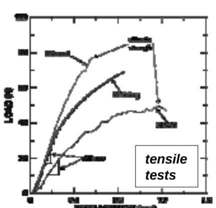


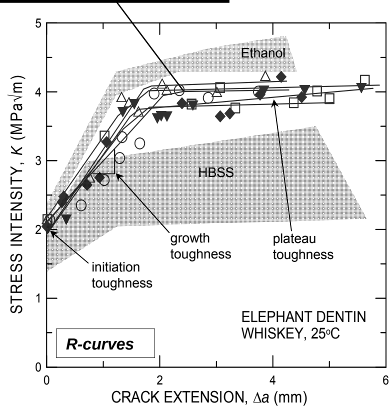
- *microcracking, at cement lines, promotes toughness via bridging*
- *cracks often follow osteocyte lacunae*

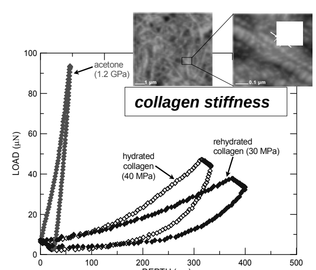
Koester, Ritchie *et al.*, 2006

Alcohol Strengthens Teeth – at least temporarily!

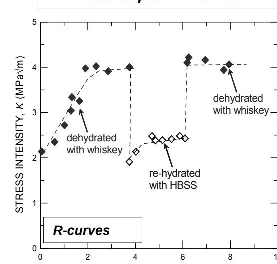
86-proof Black & White scotch whiskey







AFM-based pico-indentation



- compared to water (HBSS), whiskey increases the stiffness, strength & toughness of dentin
- but you do need to keep the alcohol in your mouth, as the effect is reversible!
- effect associated with direct collagen-collagen H-bonding in polar solvants

Nalla, Kinney, Tomsia & Ritchie, *Journal of Dental Research*, 2006 Nalla, Balooch, Ager, Kruzic, Kinney & Ritchie, *Acta Biomaterialia*, 2005

Bone Quality: Transforming Growth Factors

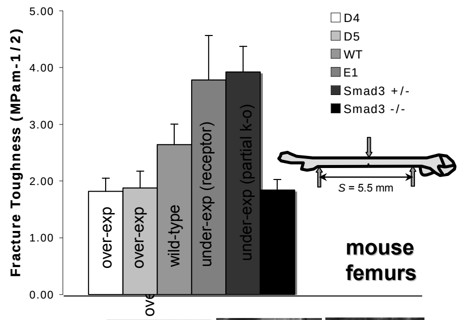
(Rik Derynck, UCSF)

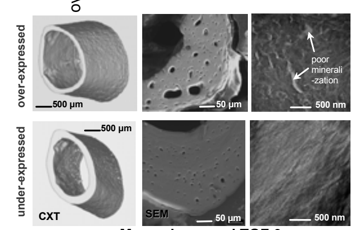
Role of TGF-β on mouse bone

possible new therapy to treat bone disorders?

- TGF-β is a family of proteins (cytokine) that can regulate behavior in bone
- TGF-β can inhibit osteoblast formation - osteoclasts are unaffected
- too much TGF-β (over-expression) leads to (27%) lower bone toughness (and osteoporosis)
- under-expressing TGF-β leads to increased bone deposition, enhanced mineral content, *50% higher bone toughness and more tortuous crack paths*

Fracture Toughness





Mouse bone and TGF-β

Balooch, Balooch, Nalla, Schilling, Filvaroff, Marshall, Marshall, Ritchie, Derynck & Alliston, *PNAS*, Dec. 2005

Conclusions

- One measure of *bone quality* is the fracture toughness. This requires an understanding of fracture mechanisms and how they are affected by microstructure
- Whereas fracture *initiation* in bone is strain-controlled, (crack-growth) toughness is derived from *extrinsic* toughening mechanisms, which promote R-curve behavior
- For crack *propagation*, the salient extrinsic toughening mechanisms are:
  - crack bridging by uncracked "ligaments" (and by individual collagen fibrils)
  - crack deflection along cement lines (transverse orientation)
- Although mechanisms are controlled by the hierarchy of structure, features at coarse length-scales, ~100-200 μm, are most important for fracture toughness
- Aging of dentin and bone identified with a loss in toughness, associated in part with a deterioration in crack bridging (consistent in bone with excessive remodeling)
- Regulation of growth factors (e.g., TGF-β) can have a significant and positive effect on the mechanical properties of bone - at nano to macro length-scales
- Whereas ovarectomies & steroids can prematurely degrade fracture toughness, Raloxifene or bisphosphonate treatments act to restore, or even enhance, fracture resistance - *mechanically due to microstructure-induced changes in crack path*

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- National Center for Electron Microscopy (LLBL)

Web page

- www.LBL.gov/Ritchie