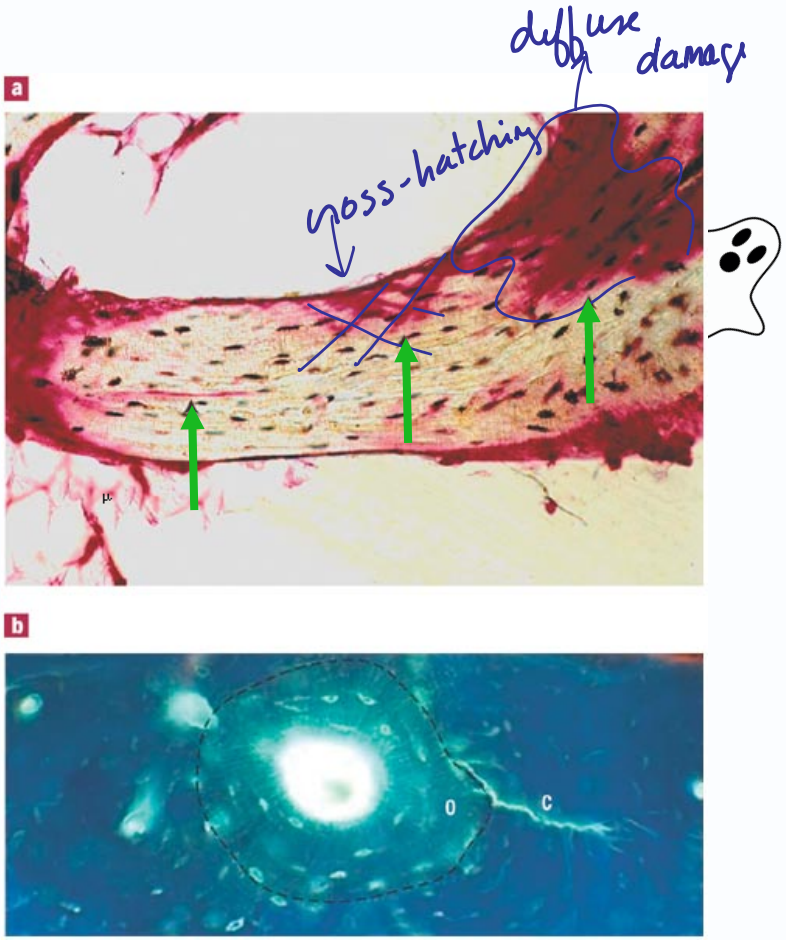
ME 482 Experimental Tissue Mechanics
Professor A. Wagoner Johnson
10/31/16 (start) – Happy Halloween!
Bone: damage & post yield behavior



Damage in bone

- Patients with osteoporosis are prone to compression fractures in the spine bones, or *vertebrae*. The bone cracks under pressure, causing it to collapse in height.

- More than 700,000 such fractures occur every year in the United States.

- The fractures angle the spine forward and produces a hunchbacked appearance, called *kyphosis.* (Kyphosis can also occur with other conditions.)

- **Patients with this condition are subject to <u>debilitating pain, disturbed sleep, decreased lung and intestinal function, and difficulty completing routine activities</u>.**

www.eorthopod.com/public/patient_education/6545/kyphoplasty.html

Microcrack density in healthy individuals

TABLE 17.1 Summary of *in vivo* Microcrack Density Measurements from Normal (Healthy) Individuals

Ref.	Source	Section	Density (No/cm²)	Length (μm)
2	Rib	Transverse	7.5/section	—
39	Rib	Transverse	14.25	88
69	Subchondral; calcified cartilage	Midfrontal	—	56.3 (5.7)
42	Rib	Transverse	14–23	—
46	Female femur	Transverse	0–500	—
	Male femur	Transverse	0–300	—
45	Vertebra	Longitudinal	220–760	53–79
47	Female tibia/femur	Transverse	22.7	2–50
	Male tibia/femur	Transverse	15.2	2–50
48	Femoral trabecular	—	16–36	—
40	Rib	Transverse	7.18	82
49	Femoral trabecular	Longitudinal	50–250	—

Cowin, Bone Mechanics Handbook

Slight aside…repair of bone

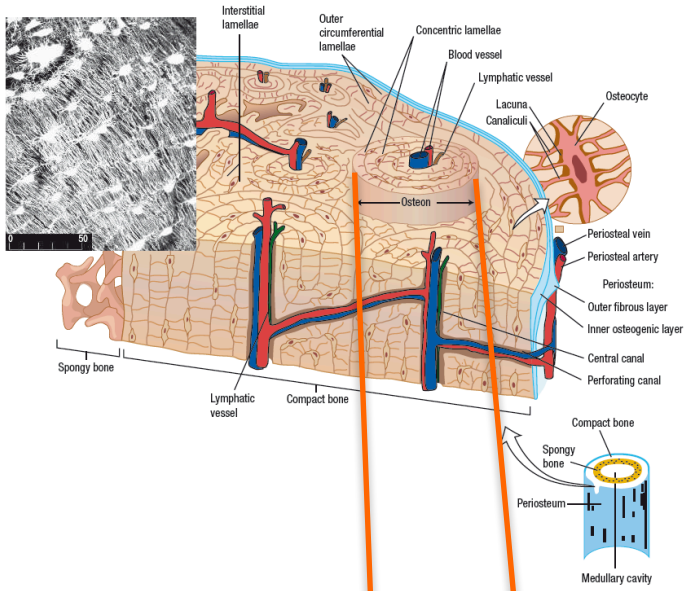


Figure 2 The structure of bone. Colour schematic reprinted from ref. 66. Copyright (2002) Wiley. Black and white microscope image (scale bar in μm) showing osteocytes (white dots) linked by processes (fine white lines) forming the syncytium. Image reprinted from ref. 54. Copyright (2004) with permission from Elsevier.

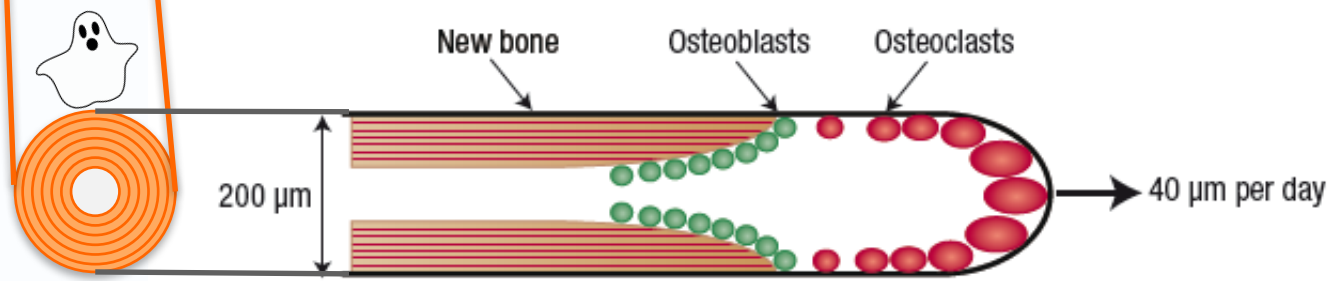


Figure 1 Bone's repair mechanism. A BMU in which osteoclasts and osteoblasts work in sequence to eat away old and generate new bone.

Living with cracks: Damage and repair in human bone, David Taylor, Jan G. Hazenberg & T. Clive Lee Nature Materials 6, 263 - 268 (2007)

Experimental measures of post-yield behavior and damage in bone

- What does damage look like?? What *is* damage?
- How is damage defined?
- How is damage measured experimentally? We will focus on just a couple of measures.
- How is damage different from other inelastic behaviors?
- At what structural level does damage matter? What is the evidence?
- ***What are the characteristics of the load-reload stress-strain curve?***

References on mechanics of damage – Keaveny (v short), Ch16.4, 17 Cowin Bone Mech Handbook

What do we mean by damage and how do we characterize it?

- Need physical evidence and mechanical evidence
  - Decrease in mechanical properties (which ones?)
  - Presence or increase in histological measures/evidence
    - (increase bc we know there we always have some microcracks, but not all result in mechanical evidence of damage)
- Damage can be described as a loss of material continuity (i.e. NOT plasticity in sense of continuum mechanics and dislocations)
- Damage contributes to inelasticity (not plasticity in the engineering material sense)
  - Must distinguish between damage behavior, plastic flow and creep, viscous behavior
- Damage in composites can contribute to toughness
- Damage in bone has biological consequences (injury, repair, adaptive response)

Three complimentary modes of damage characterization (Cowin Ch 17)

- *Mechanical damage characterization*
  - *Property degradation*

- Physical characterization

  - Histology (qualitative)
  - Histomorphometry (quantitative)

- Real-time Acoustic emission
  - (see 17.4 Cowin – v short section if you are interested)

Like in other tissues and materials, we want to correlate damage to mechanical properties and microstructure

- How do we measure permanent changes in mechanical behavior after damage event?
  - Load unload cycle (stress-strain)
  - Creep loading
  - Relaxation loading
  - Fatigue loading

- What properties do we measure to characterize damage state?
  - Stiffness
  - Strength
  - Hysteresis
  - Creep rate
  - Relaxation
  - Yield stress, yield strain
  - Microhardness
  - Residual strain
  - Residual strength

- What characterization techniques do we use and what length scales do we need to consider?
  - Histological and histomorphometric analysis
  - Micro-CT (trabecular, sub-trabecular investigation, deformation/damage mapping within sample)
  - SEM – scanning electron microscopy
    - 3D and topological detail, sometimes collagen fibers visible
    - destructive
  - TEM – transmission electron microscopy
    - Visualize collagen fibrils and mineral crystals relative to damage,
    - only images a v small area, lucky to catch damaged region

State of knowledge in damage processes is summarized as follows

- Lots of experimental evidence that damage accumulation plays a role in inelastic behavior for all loading modes studied thus far

- Histological evidence shows that damage occurs at microstructural level (microcracks) and ultrastructural level (diffuse damage)

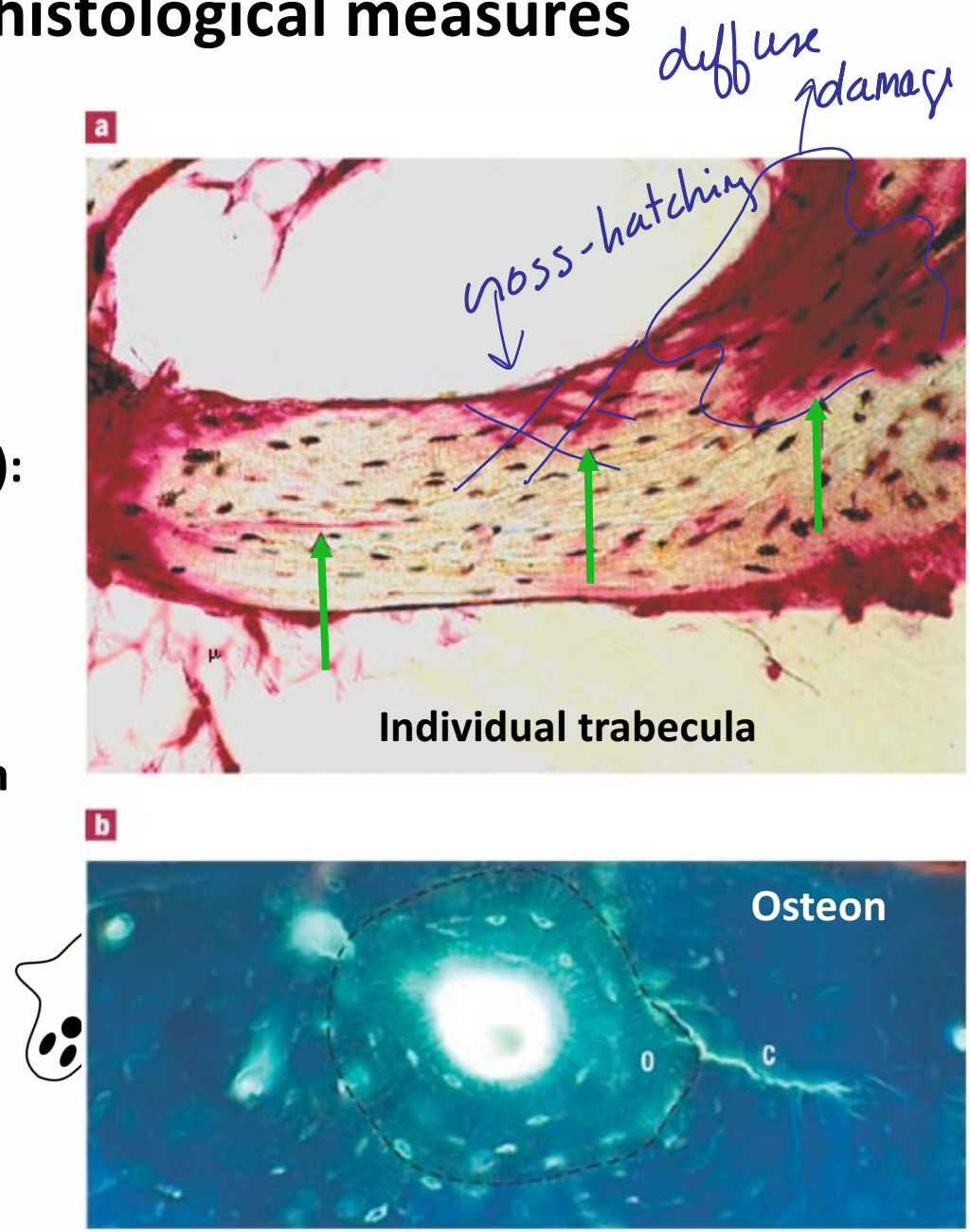
- Correlations have been found between histologically observed damage and mechanical property degradation.
  - Understanding of the relationships is incomplete
  - No consensus on standard method for characterization of mechanical property degradation

Physical Characteristics – histological measures

a, Cancellous bone showing examples of microdamage revealed by staining with basic fuschin (pink).

The arrows indicate (from left to right): a microcrack (~200 μm long), cross-hatching and diffuse damage respectively.

b, microcrack 'C' encounters an osteon 'O', and begins to grow (propagate) around the cement line (dashed line). The microcrack is ~100 μm long.

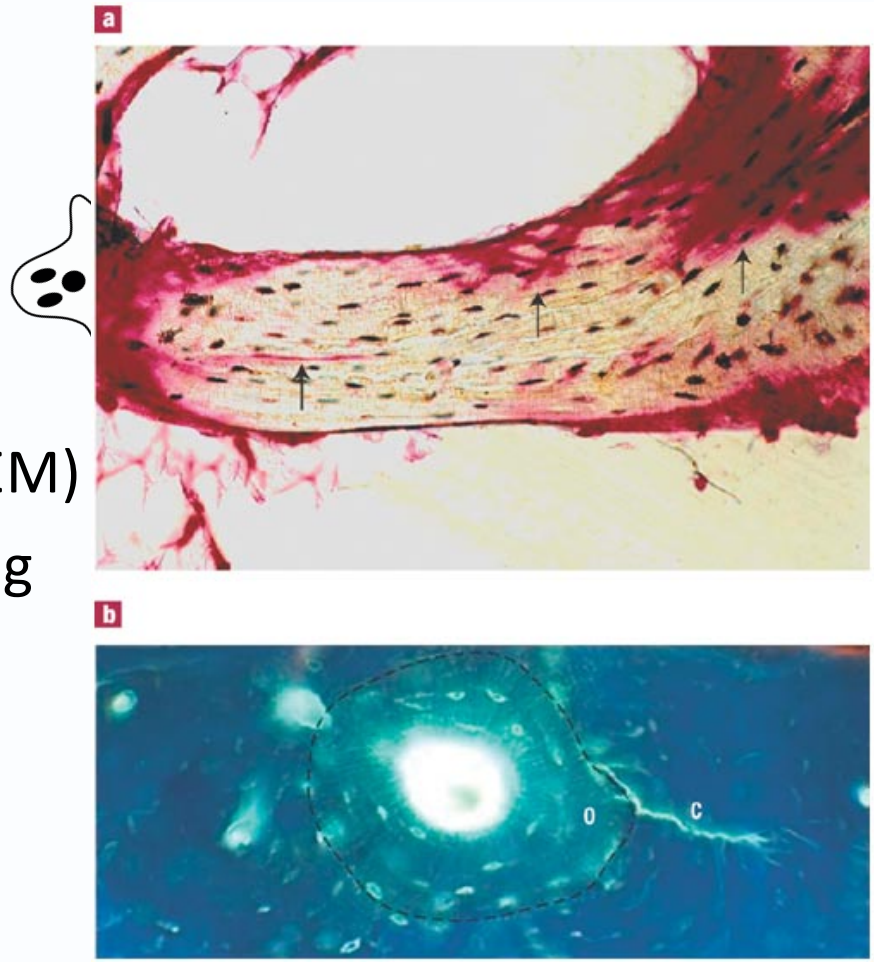


Living with cracks: Damage and repair in human bone
David Taylor, Jan G. Hazenberg & T. Clive Lee
Nature Materials 6, 263 - 268 (2007)

Physical Characteristics – histological measures

- Stains to show microcracks
  - basic fuschin
  - alizarin complexone
  - xylenol orange
  - calcein
  - lead-uranyl acetate (BSE-SEM)
  - New dyes, real-time imaging using CT or MRI?

  - **Stains typically bind to exposed Ca**



Two categories of damage observed histologically

- **Linear cracks**
  - 10-400 microns long
  - Well-defined edges
  - Interfacial cracks
    - Smooth, sharp ends
  - Cross lamellar cracks
    - Jagged because of crossing lamellar interfaces
- **Diffuse**
  - In addition to these individual, **linear cracks**, areas called **'diffuse damage '** are also observed, which contain many small cracks, each of the order of a micrometer in size.
  - Increased uptake of fuchsin or heavy metal stains
  - Both in regions near microcracks and where there are no microcracks
  - Precursor to microcracks?
- These cracks predominate in regions of compression — which constitutes the principal type of loading in our long bones — where they experience shear due to their orientation.
- Multiple cracks of intermediate size also occur, forming cross-hatched patterns
- See figures from previous slides.

Living with cracks: Damage and repair in human bone

David Taylor, Jan G. Hazenberg & T. Clive Lee, Nature Materials 6, 263 - 268 (2007)

- Mechanical loading creates damage; at the microscopic scale one can see small cracks that take the form of planar ellipses, typically having a major axis of length $2c$ = 400microns oriented approximately parallel to the bone's longitudinal axis, and a minor axis of length $2a$ = 100microns.

- The observation of these cracks is greatly aided by the use of coloured dyes that are able to diffuse into the bone and chemically bind to exposed calcium10. These dyes have been improved in recent years and are now available in different colours11, allowing cracks to be labelled at different times during an experiment12. This has yielded a great deal of information about how damage develops.

- Microcracks predominate in regions of compression — which constitutes the principal type of loading in our long bones — where they experience shear due to their orientation.

Living with cracks: Damage and repair in human bone

David Taylor, Jan G. Hazenberg & T. Clive Lee,
Nature Materials 6, 263 - 268 (2007)

- Histological studies have told us a lot about how this damage develops: calcium chelating dyes can be administered *in vitro* and *in vivo*, allowing us to monitor the amount and type of damage
  - in a laboratory specimen of dead bone,
  - or in living animals while repair processes are operating.

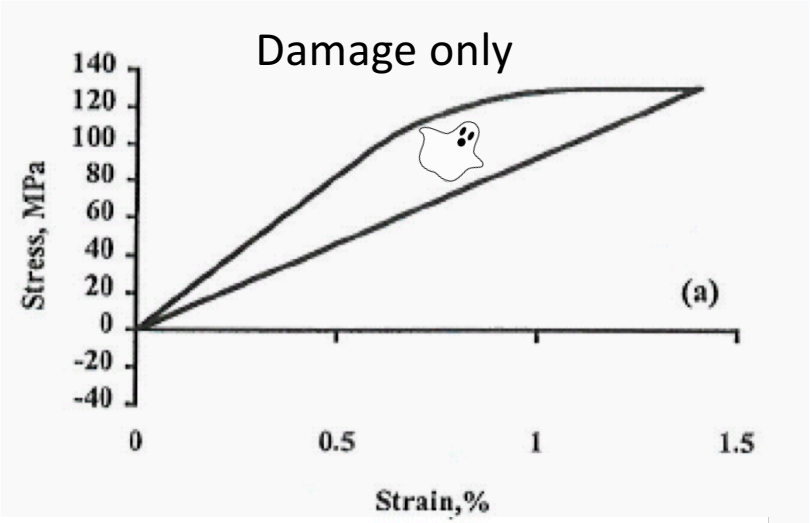
- Other techniques are being developed, such as positron emission tomography[15, 16], and new dyes may allow real-time imaging of microcracks in the body via scanning by computed tomography (CT) or magnetic resonance imaging (MRI)[17].

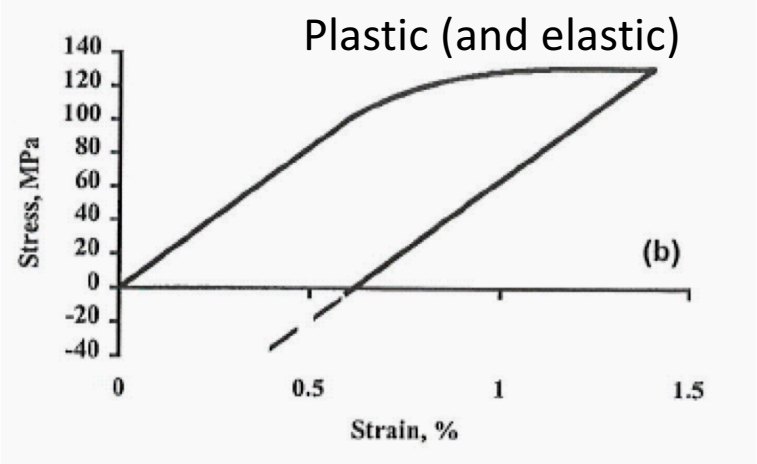
Damage occurs in the post-yield state where linear elastic deformation is exceeded (usually with 0.2% offset method)

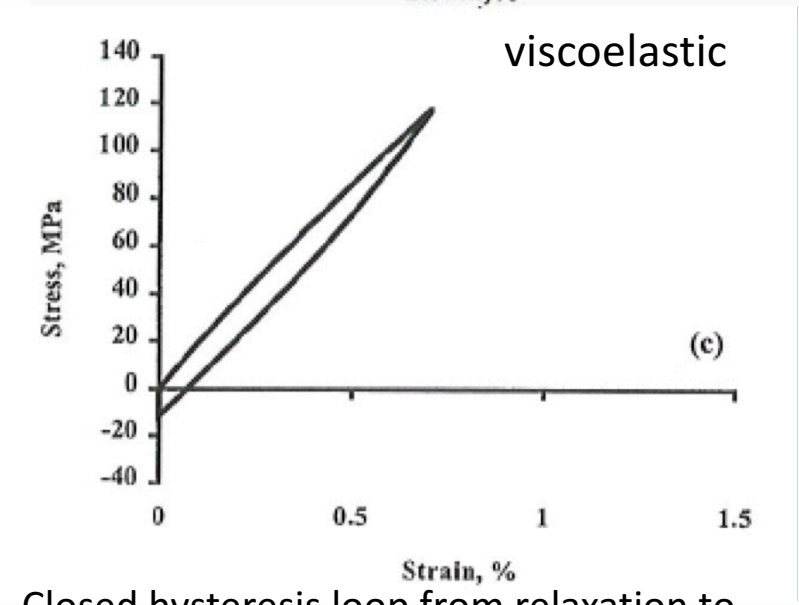
- What are sources of inelasticity in bone?
  - Damage: loss of material continuity via cracks voids, which degrade properties such as stiffness and strength
  - Plasticity as evidence of flow processes that do not change stiffness, but result in irrecoverable (plastic) strain and strain hardening (interfaces moving relative to one another, interface sliding etc.)
  - Delayed elasticity as a dissipative (viscous) process that increases stiffness with loading rate but may not result in irrecoverable strains (creep, relaxation)

- **Cannot distinguish relative roles in test to failure – must do load and reload to monitor changes in properties**

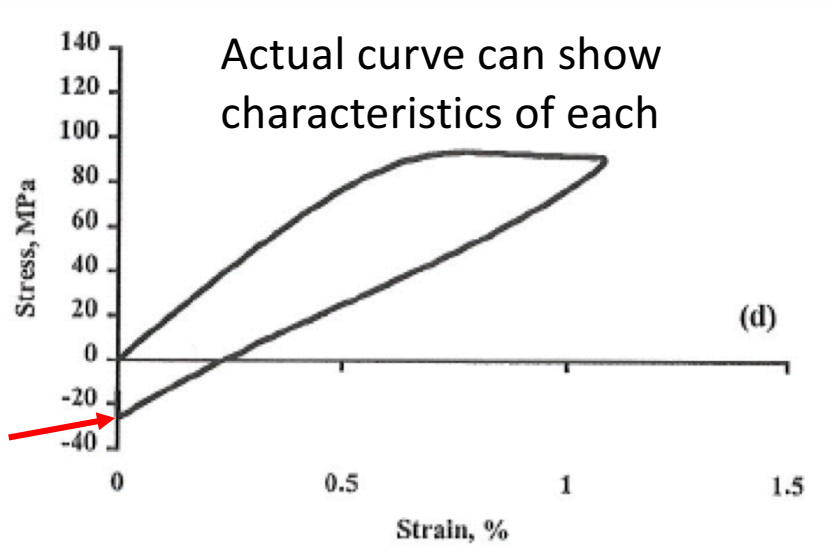
Examine loading and unloading to attempt to separate roles of **inelastic processes** – ch17 Bone Mechanics Handbook, tensile loading, cortical bone shown







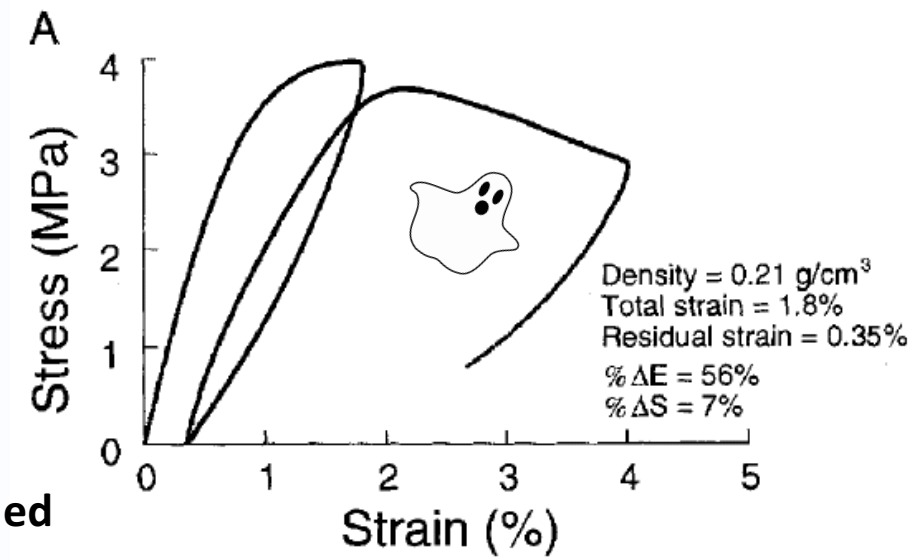
Closed hysteresis loop from relaxation to zero stress at zero strain



Actual curve for bone (cortical). Forced to zero strain, results in 26MPa compressive stress, which relaxes to ~15MPa after 15s.

Damage and postyield behavior in trabecular bone

- Damage and repair part of normal physiologic processes
  - Increases with age
- Damage may
  - Increase risk for osteoporotic fracture
  - Act as stimulus for remodeling
  - Occur during implantation of prostheses
  - Involved in aseptic necrosis of femoral head, degenerative joint disease, other disorders
- **Modulus and strength reduced when reloaded**
  - **does not depend on volume fraction (apparent density)**
  - **Does depend on amount of applied strain**

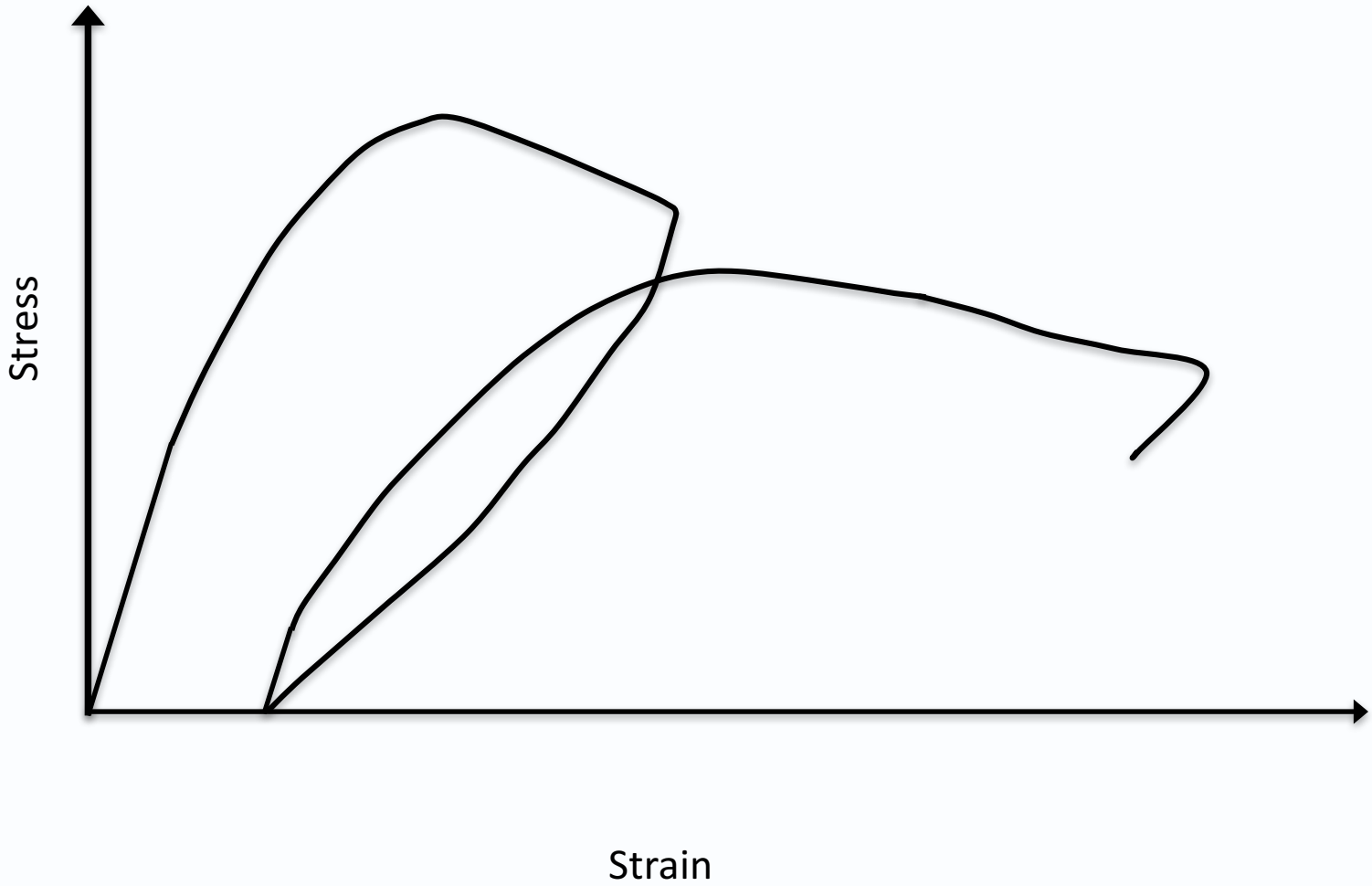


Important points related to damage (incomplete list)

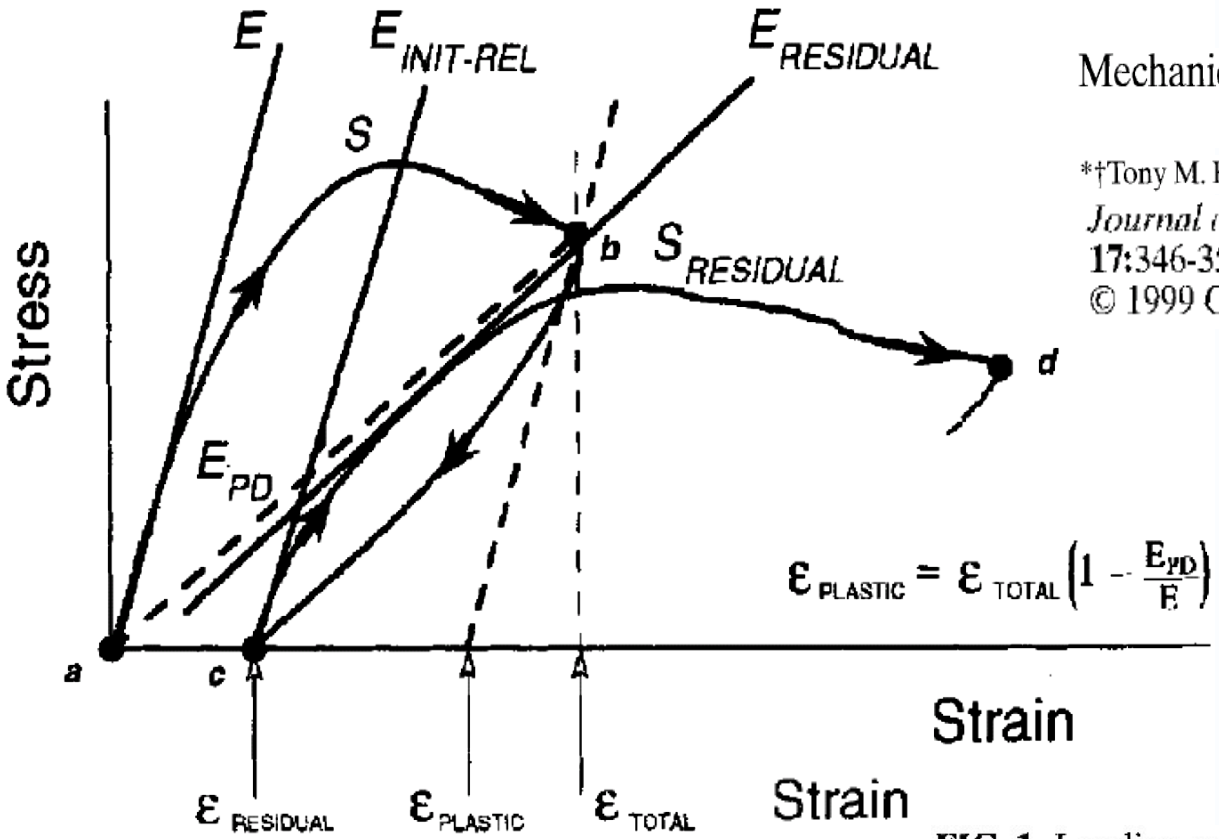
- Characteristics of load-reload curve
  - Draw the curve and identify all the output parameters we talk about
- How do % changes in properties for samples with different architectures or volume fractions compare?
- How do reduction in properties change with increasing total strain? Plastic strain?

Characteristics of the load-reload curve

(can print, label, fill out)



Parameters in the load-reload curve



Mechanical Behavior of Human Trabecular Bone after Overloading

*†Tony M. Keaveny, *Edward F. Wachtel, and *David L. Kopperdahl

Journal of Orthopaedic Research
17:346-353 The Journal of Bone and Joint Surgery, Inc.
© 1999 Orthopaedic Research Society

FIG. 1. Loading protocol and output parameters (see text for details). Loading started at point *a* and proceeded to point *b*, unloading occurred from points *b* to *c*, and reloading occurred from points *c* to *d*. The total strain at the point *b* of unloading is shown as ε_{TOTAL}. The plastic strain, $\varepsilon_{PLASTIC}$, was calculated from ε_{TOTAL}, and the perfect-damage, E_{PD}, and Young's, E, moduli, as shown. This is the permanent strain that would occur if the material behaved in a perfectly plastic manner. The residual strain, $\varepsilon_{RESIDUAL}$, is the measured strain at point *c* just before reloading. $\varepsilon_{INIT\text{-}REL}$ = initial reloading modulus, $E_{RESIDUAL}$ = residual modulus, and $S_{RESIDUAL}$ = residual strength.

What can we note with these two curves?

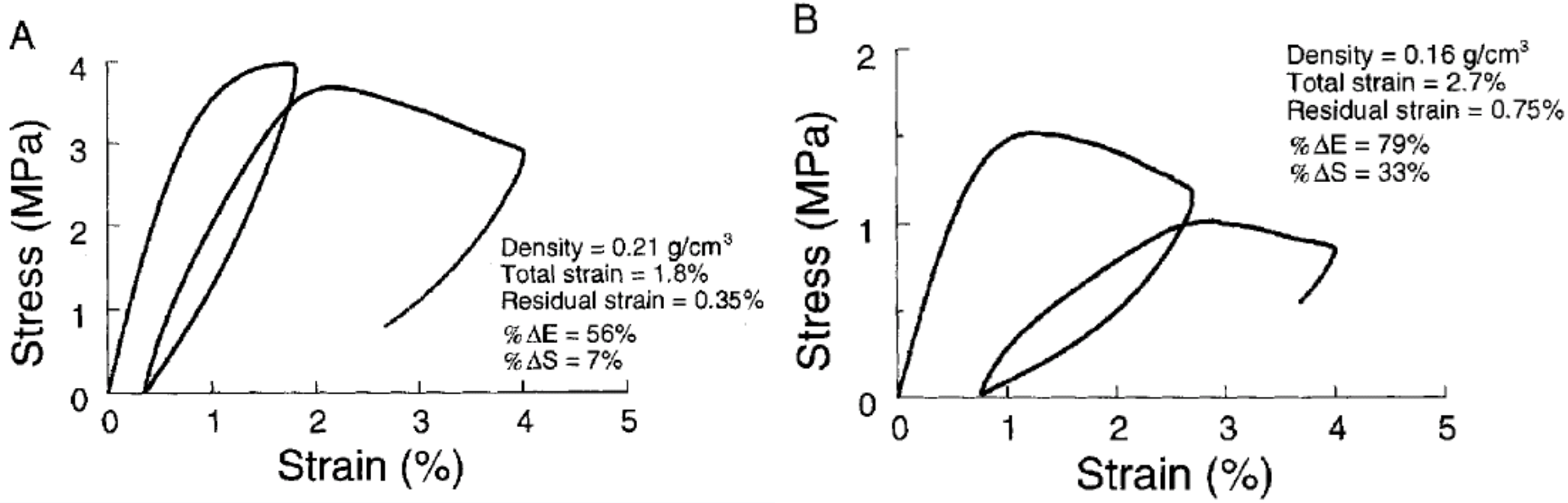


FIG. 2. A and **B:** Examples of load-unload-reload stress-strain curves for two specimens of different apparent densities loaded to different total strains. %ΔE and %ΔS denote the percent modulus and strength reductions, respectively. Note the characteristic secondary linear region in the reloading curve in each case, particularly for the specimen loaded to the higher strain (**B**).

Percent reduction of modulus and strength increase with increasing plastic strain, but have different dependencies

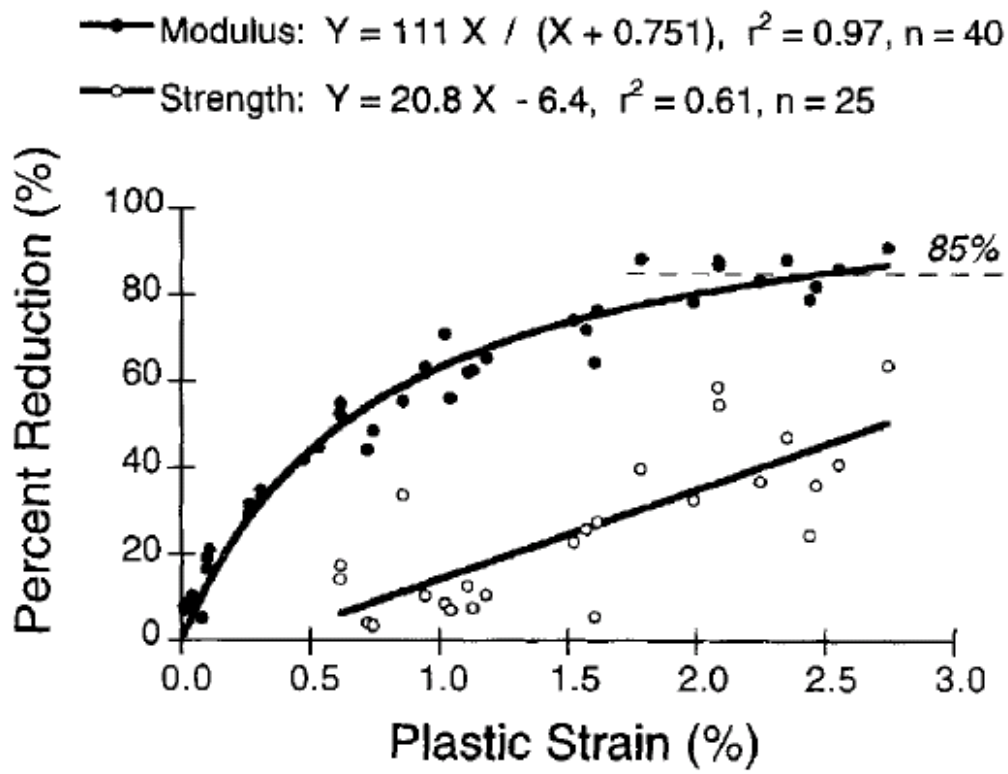


FIG. 4. Dependence of the percent modulus and strength reductions on plastic strain. Strength reduction was calculated for only 25 of the 40 specimens because 15 were not loaded past the ultimate stress on the loading cycle. Values for percent modulus reduction leveled off to an average (±SD) of 85.2 ± 4.1% at plastic strains greater than 1.75%. The reported regressions can be used to predict property degradations from plastic strains. For comparison with other studies (20), the following regressions, using total applied strain as the independent parameter X, are also reported for percent modulus and strength reductions, respectively: $Y = 178 - 496 / (X + 2.58)$, $r^2 = 0.94$; $Y = 20.4X - 16.9$, $r^2 = 0.45$; $X \leq 3.0\%$ in both cases.

IMPORTANT POINTS–How do %ΔS and %ΔE compare for *human vs bovine*? *Trabecular vs whole vertebral body*? **What can we conclude about damage?**

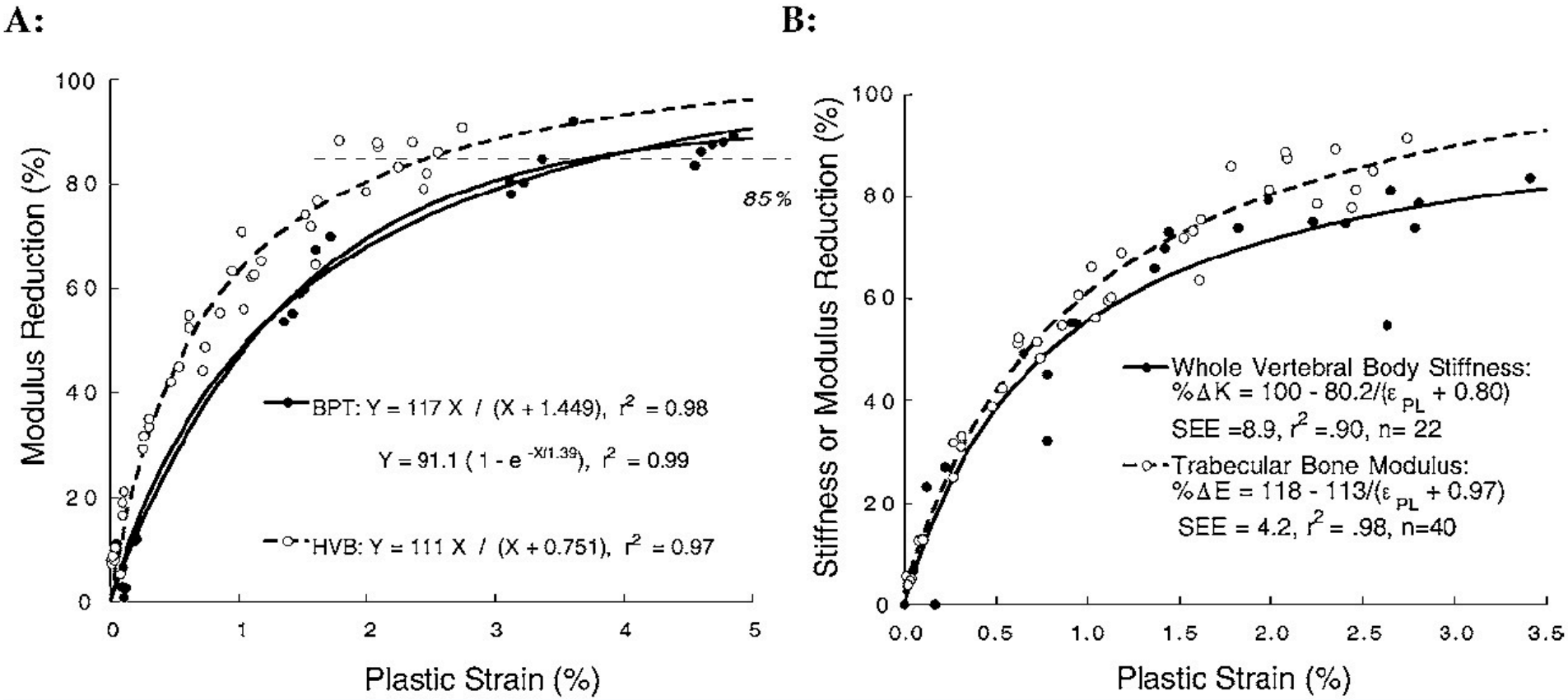


FIGURE 16.19 Dependence of percentage modulus (or stiffness) reduction, for reloading after overloading to various strains, on the applied strain level for cores specimens of vertebral trabecular bone (A) and the whole vertebral body (B). Data for bone from the bovine proximal tibial (BPT) cores are also shown in (A). This post-yield behavior is similar for bovine tibial and human vertebral bone, despite the large differences in denisty and architecture. Further, the behavior of the cores is similar to that of the whole vertebral body (B). Perhaps most surprisingly are the very high correlations between these percent reductions vs. just plastic strain. The same experimental data were used for the vertebral trabecular bone in each graph. The minor differences when plotted are due to the different horizontal scales on each chart, and subtle differences in the definition of the reloading modulus used in the two studies. Different regression models were also used to describe the trends. (Adapted from (A) Keaveny et al., *J. Orthop. Res.*, 17, 346–353, 1999; and (B) Kopperdahl et al., *J. Orthop Res.*, 18, 685–690, 2000. With permission.)