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Stress concentrations and bone microdamage: John Currey's contributions to understanding the initiation and arrest of cracks in bone



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ABSTRACT

The microarchitecture of bone tissue presents many features that could act as stress concentrators for the initiation of bone microdamage. This was first identified by John Currey in a seminal paper in 1962 in which he presented the mechanical and biological evidence for stress concentrations at the bone surface, within the bone through the action of stiffness differentials between architectural features including between lamellae, and at the level of the lacunar and canalicular walls. Those early observations set the stage to consider how microscopic damage to bone tissue might affect the properties of bone at a time when most in the scientific community dismissed microcracks in bone as artifact. Evidence collected in the nearly 60 years since those important initial observations suggest that some of these architectural features in bone tissue are more effective as crack arrestors than as crack initiators. Sites of higher mineralization in the bone matrix, particularly interstitial sites in both cortical and trabecular bone, may serve preferentially as locations for crack initiation, whereas those boundaries identified by Currey as both stress concentrators and stress arrestors are more effective at stopping cracks than at initiating them.

1. Introduction

The idea that bone microcracks occur naturally in bone, and that they are targeted for repair, was proposed first by Frost [1]. Except for engineers like Carter and Hayes [2], who embraced in the 1970s the idea that microcracks occurred naturally in bone, it was not until the early 1990s that much of the scientific community accepted this fact. And it was not until the late 1990s or early part of this century that most scientists accepted that cracks are preferentially repaired through signaling mechanisms initiated by osteocyte apoptosis [3–7]. However, John Currey was an “early adopter” in accepting the fact that cracks develop naturally in bone (as they do in other cyclically loaded materials) in his classic paper in 1962 on Stress Concentrations in Bone [8]. In this paper he also describes how size and orientation of both the discontinuities and the cracks can be either more or less dangerous to the full failure of the bone. Thus, besides Frost and in a much more mechanically thoughtful way, John Currey was one of the first to discuss the morphological aspects of bone that allow for crack initiation and arrest.

This review of the role of stress concentrations in bone, therefore, is intended to build on Currey's original observations by describing

research in the last 50 years that addresses his observations specifically as they relate to damage formation and crack arrest. This review will not address stress concentrations as they affect overall mechanical properties, or impact strength, which was part of Currey's presentation. There are many papers, books and book chapters that treat bone microdamage and its repair more broadly, and the reader is referred to these for a more complete treatment of damage morphology, repair, and effects on mechanical properties of bone [9–11].

2. What are microcracks?

Microcracks form in vivo in human bone under conditions of cyclic loading. They cannot be visualized radiographically, and individual microcracks can be difficult even to detect using μ CT in bone removed from the body (ex vivo). There are two types of microcracks that are now termed “diffuse damage” and “linear microcracks” (or just microcracks). Diffuse damage represents a collection of many very small microcracks ($< 10 \mu\text{m}$ in length in a cross-section of bone). Although a collection of these small cracks can be identified on basic fuchsin stained sections, individual cracks within the diffuse damage area can be difficult to identify unless confocal microscopy is used because they

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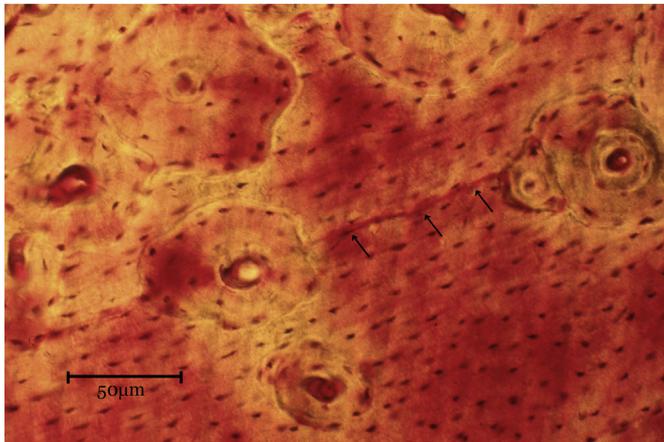


Fig. 1. This shows a microcrack completely contained within the interstitial matrix. Note that the crack is arrested on either end by the cement lines of two different osteons.

are so small [12–14]. It now appears that these cracks are small enough that they can be repaired through processes that do not involve whole bone remodeling [15]. Linear microcracks (Fig. 1) are longer, typically 50–100 μm or more long in cross-section, and may run to nearly 1 mm longitudinally in bone [16,17]. These cracks are repaired by bone remodeling that involves osteoclastic resorption and formation of new bone by osteoblasts in the area where the crack previously existed.

Linear microcracks can grow into “macrocracks” that can become stress fractures or even complete fractures under continued cyclic loading. Unlike microcracks, macrocracks associated with stress fractures often can be seen radiographically or can be imaged in vivo using scintigraphic tracers such as radioactive technesium. Cracks associated with stress fractures may span an entire cortex of a bone so may be many millimeters long in cross-section.

The basic mechanical properties of bone (strength, stiffness, energy to fracture) are often measured using quasi-static mechanical tests, in which a load is applied continuously to a bone until a large crack is formed and the bone breaks quickly. However, microcracks are formed under cyclic loading that over time leads to bone fatigue, or a gradual loss of residual strength and stiffness. Because of bone's microstructure, which can both enable crack initiation but also stop cracks from growing, bone exhibits a 3-phase damage accumulation history (as do other composite materials, and unlike quasi-static loading) during the cyclic loading process prior to failure. These phases are [18]:

- (1) *Initiation*, in which cracks initiate at a structural discontinuity, but are arrested at an adjacent discontinuity. At this stage, cracks are self-limiting, but serve to redistribute stresses within the tissue. Damage initiated at this stage occurs whether the loading is quasi-static or cyclic.
- (2) *Benign accumulation*, in which the accumulation of cracks and the loss of stiffness stabilize. This occurs because cracks become more numerous, and the distance between them becomes smaller so that peak stresses between the cracks are low enough that new cracks cannot form within the material between them. Damage accumulation in this stage leads to intra-lamellar debonding or cement line delamination; therefore, the cracks do not cause catastrophic failure or even change the elastic modulus very much. At this stage, delamination damage is characteristic of cyclic rather than quasi-static loading.
- (3) *Failure* occurs rapidly during the third stage of fatigue as cracks converge into larger cracks and more lamellar debonding occurs. Cracks are no longer self-limiting as in the first stage of damage; as lamellae are damaged, stresses increase in those parts of the matrix that are still intact. Crack growth during this stage may be very



Fig. 2. Crack deflection. A microcrack (arrows) cutting across interstitial cement line boundaries (ICL) and being deflected by one of them. The crack appears to be arrested after being deflected into the ICL. En bloc basic fuchsin stain, orig. mag. 62.5 \times .

similar to matrix damage that occurs just prior to failure during quasi-static loading.

3. Why are cracks important to bone?

From a mechanical standpoint, linear cracks in bone are easy to initiate, but grow less easily [19,20]. This is an elegant mechanism to allow for bone repair and renewal, with several important consequences. One of these is that damage initiation, crack growth, and crack arrest increase the amount of energy required to fracture the bone, at least in the short term, by releasing energy created by stress that would otherwise cause uncontrolled crack propagation and earlier catastrophic fracture [21–23]. This occurs through plastic deformation at the crack tip (an intrinsic mechanism) as well as mechanisms that channel deformation energy away from the formation of new surfaces at the crack tip. For instance extrinsic toughening mechanisms involve crack bridging (by uncracked “ligaments” and collagen fibers that shield the crack tip and slow its propagation) as well as deflection of cracks around microstructural features that act as barriers to unconstrained crack growth [11,23] (Figs. 1, 2). Currey described the latter mechanism (see below) although not the first. Bone tends more toward brittleness than ductility, and extrinsic toughening mechanisms provide a greater influence on the toughness of bone than do mechanisms acting in the material volume at the crack tip (intrinsic toughening mechanisms) [23].

Crack initiation is less important to the inherent toughness of bone but derives its importance physiologically. It causes damage to the osteocytic network that promotes osteocyte apoptosis [7]. The dying osteocytes close to the damage produce less osteoprotegerin (OPG), whereas the healthy osteocytes at some distance from the damage release more RANKL [24,25]. This imbalance of RANKL and OPG promotes the differentiation of osteoclasts and initiates the remodeling process. This relates to the second important consequence of damage formation and arrest in bones. Crack arrest and repair renews the bone matrix and improves the quality of bone through formation of new bone that requires time to become fully mineralized. As about 80% or more of microcracks initiate in the interstitial matrix [26–28] (Figs. 1, 2), this removal of highly mineralized bone and replacement by less mineralized bone increases the overall ductility of bone and maintains its material properties within normal ranges.

To perform these mechanical and physiological functions, it is necessary for bone to have both stress concentrators and stress arrestors, perhaps at different hierarchical levels of bone structure.

4. What did Currey say?

Currey identified three types of stress concentrators in bone:

(1) *Surface discontinuities*

For the most part, there are few discontinuities on the periosteal surface of bone that act as stress concentrators, or stress arresters. In fact, Currey points out, bone appears to be adapted to minimizing such stress concentrators in regions that might be subject to high stresses. So, for instance, the diaphyses of bones are smooth, except in a few locations where muscles attach. This not only reduces stress concentrations but also provides a smoother gliding surface over which muscles can contract without excessive friction to the muscle belly. Where stress concentrations do exist on surfaces of long bones is usually near the ends of the bones in the metaphyseal regions, where for mechanical reasons bending stresses are relatively low. The one stress concentrator that Currey identified on the periosteal surface near the mid-diaphysis of bone is the nutrient artery. As he pointed out, these arteries enter the bone at a slight angle to the long axis of the bone, more in line with the direction of maximum shear stresses, which will reduce their stress-concentrating properties. The highest shear stresses in bone will occur at about a 30° angle to the long axis. Therefore, an angle of the artery between 15–30° will minimize the shear stress concentration on the wall of the cavity housing the artery. This orientation is also consistent with the direction of cracks created when a notched test specimen is loaded in tension [29]. Resistance to crack growth in response to quasi-static shear loading is greater than with tensile loading, suggesting that bone may be adapted to prevent crack growth in shear [30].

It is interesting, therefore, that in a letter to me in 1987, Currey suggests that one problem with the idea that cracks are targeted for repair by remodeling is that cracks (and remodeling) tend not to occur on the “outermost fibers of bone” (the periosteum) where stresses are highest. But, as he pointed out in 1962, one might not expect to find many cracks in this region because of the low density of stress concentrators. Furthermore, mean tissue age of bone close to the periosteal surface is less than that in other areas of the cortex, further reducing the stresses needed for crack formation. As pointed out above, most cracks occur in older interstitial bone, which is more highly mineralized and theoretically more brittle.

One does find more cracks closer to the endocortical surface of bone. This suggests that stress concentrators or mean tissue age (MTA) may hold greater importance than the magnitude of stress (or strain) as strains on the inner surface of bone, for geometric reasons, are lower than those on the periosteal surface. Currey explained that “fracture is less likely to start at the inner surface of the shaft than at the outer surface... [even though the] inner surface ... is in fact much rougher than the outer surface” [p. 116]. He attributed this to the greater level of remodeling on the endocortical surface, which would in fact also “remodel out” cracks that might form in this location. However, the rougher surface might also be expected to have more regions of stress concentration because of its roughness. So, the balance of damage formation, low strains (but potentially high local stresses in some locations), MTA, and remodeling on the endocortical surface make it difficult to evaluate the importance of stress concentrations on this surface.

(2) *Stiffness differentials between morphological components of bone*

Currey states that “if a body has some of its volume occupied by material with a lower modulus of elasticity than the rest, there will be stress concentrations set up in the body on the application of external force, even though the body has no holes or notches in it. The magnitude of the stress concentration will depend upon the orientation and shape of the inclusion, and the difference in elastic modulus between the two materials” [p. 116]. Currey approached this potential source of stress concentrations in bone at a more

molecular level, considering stress concentrations that might occur at the collagen-mineral interface. He dismissed these as meaningless because of the intimate relationship between collagen and mineral, which may act a single material rather than two separate ones. It is now known that there likely is some damage that occurs at this level [31] and that increasing the ductility at this interface can reduce interfacial stresses and make the bone tougher [32].

Possibly because he was considering bone of many different types (including bovine bone which is more laminar and less remodeled), Currey did not identify cement lines in human bone as sites for potential stress concentration. However, the differential between the elastic modulus of cement lines and the bone matrix surrounding the cement lines, is an excellent example of the stress concentrating (but also stress arresting) effects of cement lines in human bone. This is true of cement lines whether one considers them less highly mineralized [33] or more highly mineralized [34] than surrounding bone – it really wouldn't matter. The cement line interface separating Haversian systems from extra-osteonal bone matrix is a region of relatively low shear strength compared to mineralized bone matrix [35,36], and damage can therefore “nucleate” within the cement line and cause debonding of the osteon from the surrounding matrix (Fig. 3). This is one good example of a situation in which stiffness differentials between morphological components of bone can initiate damage as well as keep the crack from growing.

(3) *Internal discontinuities, including lamellae, lacunae and canaliculi*

This category of stress concentration is composed of structures at several different hierarchical levels of organization, from relatively large (~40–50 µm diameter) Haversian canals (Fig. 4), to relatively small (~1–20 µm diameter) canaliculi (Fig. 5) and osteocyte lacunae (Fig. 6). At the larger end of the spectrum, the stress concentrating effects of canals for blood vessels depend on their shape, their length, and their orientation to the long axis of bone (or the direction of load). Currey points out that when the long axis of the channel and the stress direction are parallel, stress concentrations are reduced, and when they are perpendicular, they are at their maximum value. Most Haversian canals run at a slight angle with respect to the bone's long axis, on average about 11–17° with respect to that axis [37], which reduces the shear stress on their walls analogous to what was found for the nutrient artery. Although not all Haversian canals are entirely circular, the canals generally are closer to circularity than to an ellipsoid, which further reduces their stress concentrating effects. On the contrary, Volkmann's canals, which connect the more longitudinally-running Haversian canals, run in directions more perpendicular to the load direction and so may provide a larger stress concentrating effect than Haversian

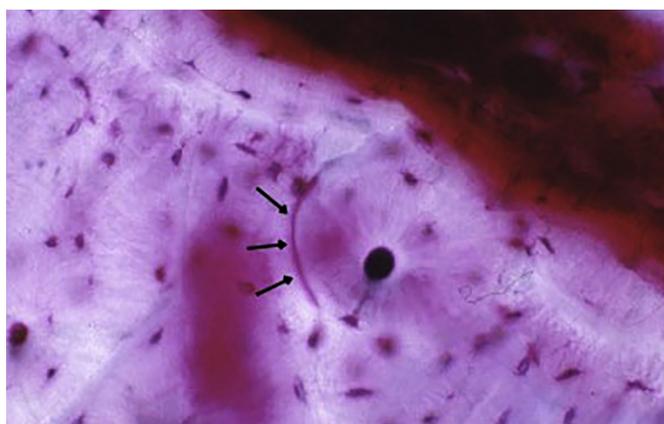


Fig. 3. Partial cement line debonding. A microcrack (arrows) has partially debonded an osteon from its surrounding matrix. En bloc basic fuchsin stain, orig. mag. 62.5×.

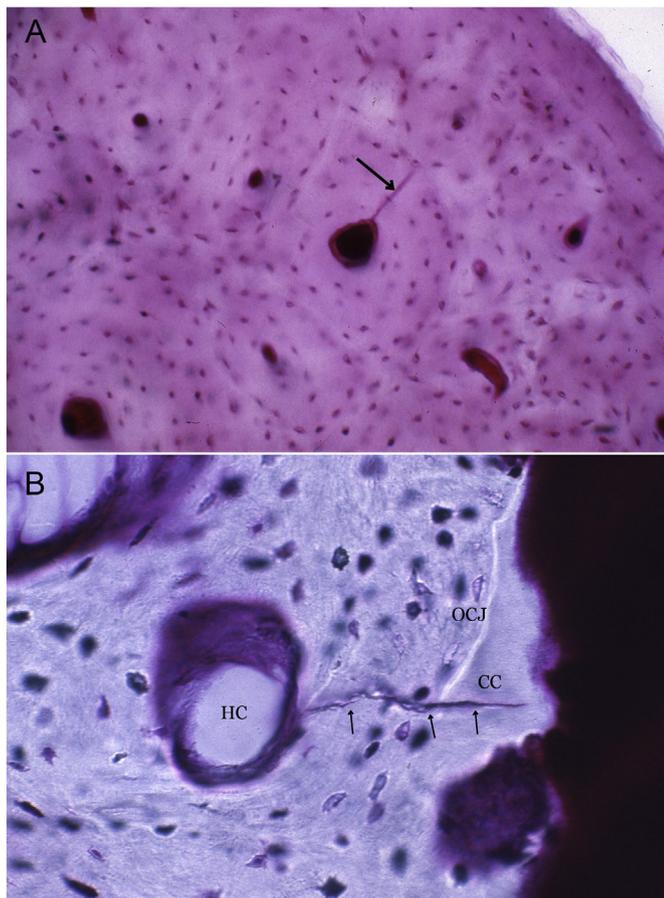


Fig. 4. (a) Microcrack (arrow) running between an Haversian canal and the cement line. It is not clear whether the crack started at the canal and was stopped by the cement line or started within the osteon and was stopped by the canal. Because it would be unusual for a crack to start within an osteon without a clear stress concentration present, the most likely explanation is that the crack started from the canal side. En bloc basic fuchsin stain, orig. mag. 62.5 \times . (b) Another example of a microcrack (arrows) started or stopped by a canal in bone. In this case it is most likely that the crack started within the highly mineralized calcified cartilage (CC), pierced the osteochondral junction (OCJ) and was stopped by the Haversian canal (HC). En bloc basic fuchsin stain, orig. mag. 156 \times .

canals. However, this effect again is reduced by their near circularity, and could be minimized as well by their smaller diameters. Still, in an *ex vivo* study, Reilly and Currey [38] demonstrated tensile cracks initiating (presumably, although they could have been arrested here) around blood vessels and remodeling cavities. Because linear microcracks are now known to initiate a repair response, they will often be found in association with resorption pits (Fig. 7). It also may be possible that pre-existing resorption pits can nucleate cracks at their base because of high stresses there [39–41], although there is some controversy about the relative importance of this to crack formation [42]. McNamara et al. [40] estimate stress concentration factors of about 3 at the base of the resorption cavity, which agrees quite well with Currey's estimate for a cylindrical pore at a right angle to the direction of stress. Many resorption pits tend not to be very circular (Fig. 8) (also see Fig. 9 in Ref. [43]) and stress concentrations at the tip of a cutting front can be several times larger than this. However, in cancellous bone most resorption activity occurs on the surface of the trabeculae, where the bending stresses are highest, yet most microdamage is found 30 μm or more distant from the surface within the interstitial bone of the trabecula [40]. This suggests that in cancellous bone, resorption pits are not actually serving as a primary attractor to initiate bone damage.

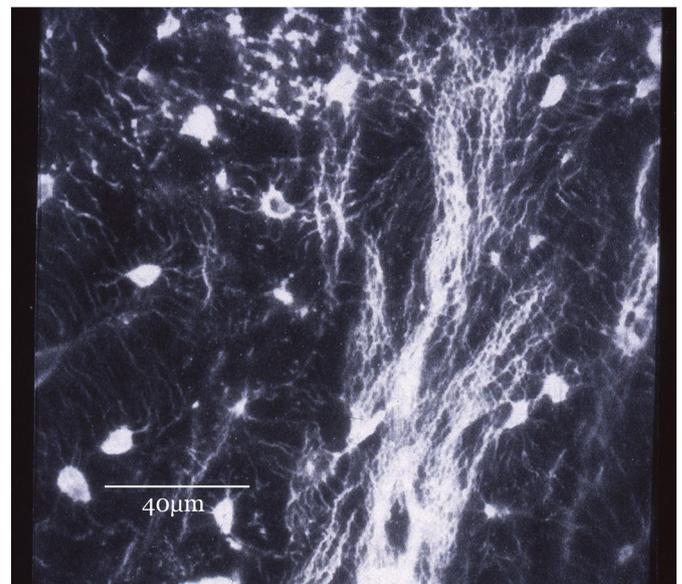


Fig. 5. Microdamage cutting across canaliculi. Confocal image.

Nevertheless, most resorption spaces were associated with microdamage in close proximity, though not intimately touching. This may reflect that two-dimensional views of bone do not accurately reflect the three dimensionality of both cracks and resorption tunnels [although Goff et al. used a 3-dimensional approach]. Furthermore, it is very difficult to tell from cadaveric specimens or from biopsies whether the association of resorption pits and microcracks reflects a stress concentrating effect that initiated the crack, or whether a pre-existing crack initiated the resorption event through targeted remodeling.

Canaliculi are long compared to their diameter of 1 μm or less and are arranged in a more 3-dimensional array than are the canals in bone. Therefore, if their size permits, canaliculi could act as stress concentrators in bone. It is difficult to tell microscopically whether cracks actually do start at canalicular boundaries, but it is clear that they are not arrested by these boundaries as cracks 40–100 μm in length can readily be visualized cutting across many canaliculi (Fig. 4). The extent to which structures of canalicular size actually have any stress concentrating effect is still unknown nearly 60 years after Currey's original observations.

The same may be true of the much larger (compared to canaliculi) osteocyte lacunae that can range from a few microns along their minor axis to 20–25 μm along the major axis. The high local strains around osteocyte lacunae [44] creates bands of stress concentrations between them [45] that provide sites for crack nucleation. If these cracks grow, they can reduce strains around adjacent lacunae, which in turn relieves strain on the Haversian canal wall by a factor of three [46]. The reduction of stress on the Haversian canal wall, essentially putting it into a state of virtual disuse, could contribute to, and complement, the well-known effect of osteocyte apoptosis on signaling for repair of microcracks, as the remodeling process has to begin on the surface of the Haversian canal wall.

As with Haversian systems, shape is important and rounder lacunae (of which there are fewer) are less prone to stress concentrating effects than more spindle shaped lacunae. As the lacunae tend to be oriented parallel to the direction of the lamellae, a crack crossing a lamella would intersect the lacunae in the minor axis, where the stress concentrating effect of the lacuna is highest. However, if the crack was “trapped” by the lamella and began to run circumferentially within the lamella, it would encounter the lacuna along its long axis and the stress concentration would be minimal. Currey's calculation of stress

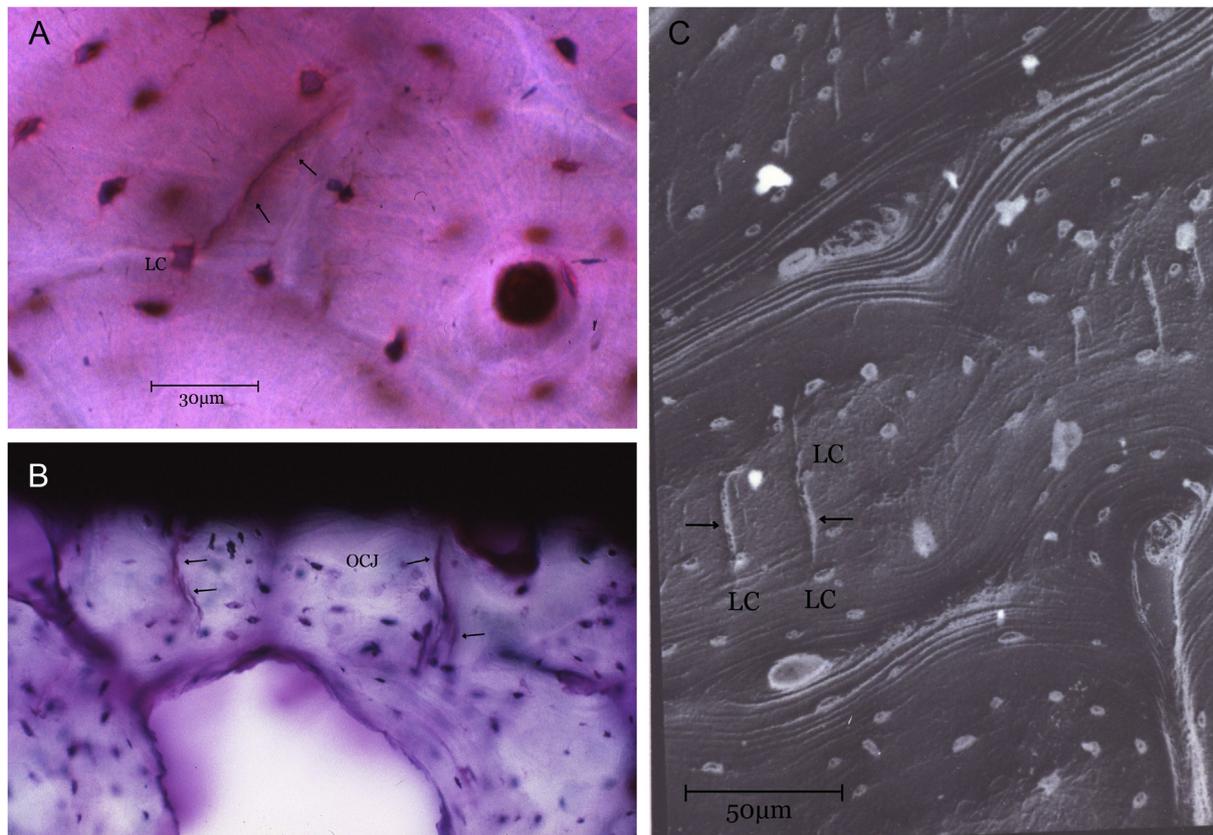


Fig. 6. (a) Microcrack (arrows) that appears to be stopped near a lacunar boundary (Lc). En bloc basic fuchsin stain. (b) Multiple microcracks (arrows) at the osteochondral junction (OCJ) of the metaphysis of the proximal femur. Note on both the right left sides of the image that the cracks appear to be running into the osteocyte lacunae, and being arrested. En bloc basic fuchsin stain, orig. mag. 156 \times . (c) Differential interference contrast (DIC) image of microcracks (arrows) running between osteocyte lacunae (Lc). Note that some appear to be stopped by the lacunae, whereas others re-appear on the opposite side of a lacuna and continue to grow. Bovine plexiform bone.

concentration on lacunar walls from 20 different lacunae indicated that there could be as much as 3–4 times greater stress concentration in the minor direction as in the major.

Linear microcracks can be found stopped by lacunae (Fig. 6), although it seems unlikely that cracks are initiated at this level. However, the stress concentrating effects of lacunae can be demonstrated by the large areas of diffuse damage adjacent to osteocyte lacunae when bone is loaded *ex vivo* [38,47,48]. At high strains, clusters of microcracks form around lacunae, perpendicular to the long axis of the lacuna [48]. This is in accordance with Currey's predictions of the stress concentrating effects of lacunae along the minor axis.

The bottom line to all of this, as Currey recognized, is that discontinuities in bone typically run in the long axis of the bone as an adaptation to minimize otherwise high stress concentrations that would prevail. This is particularly important in the case of hip fractures as the impact energy absorption is reduced by the square of the stress, i.e., if the stress triples, the ability of the bone to absorb energy is 9 times less. Of course, there are other factors at work here as well, such as the overall material properties of the bone tissue. However, the stress concentrating effects would still have particularly negative consequences in the superior cortex of the femoral neck, which is as thin as a single trabecula in many older individuals.

5. Discontinuities in bone as stress arrestors

Stress concentrators can be stress arrestors as well. Similar considerations as those outlined above apply to discontinuities that act as stress arrestors: their effectiveness is based on their size, shape and orientation. Currey relied on indirect evidence from other materials and

from theoretical considerations in proposing that discontinuities in bone could act as stress arrestors. He provided observational evidence that this does occur in various ways at lacunar boundaries (Fig. 5), although lacunae are small and their success at stopping cracks may depend on speed of propagation. This may be the reason that diffuse damage (collections of cracks on the order of $< 10\mu\text{m}$ in length) is more often found associated with lacunar walls than are larger linear microcracks [48]. The rate of growth in turn may depend on the overall heterogeneity of the bone tissue at different length scales, from microns (osteons) to nanometers (mineralization heterogeneity and the collagen-mineral interface). There may still be some uncertainty about size thresholds required to arrest crack growth although these interfaces at submicron levels seem to be good initiators [47].

Work since 1962 has made it clear that osteonal cement lines can definitely act as crack arrestors [2,21,28] (Fig. 9), but their effectiveness may depend on the length of the cracks and the speed at which they propagate [49,50]. Cracks of average length ($\leq 100\mu\text{m}$) are generally stopped by cement lines, but longer cracks are either deflected ($100\text{--}300\mu\text{m}$ in length) or pass through the cement line (cracks $> 300\mu\text{m}$ in length), where they can be stopped by the Haversian canal or continue to grow into adjacent osteons [29,49]. In these cases, length is probably a surrogate for speed of propagation, one reason that the larger cracks are also more often associated with complete fracture of bone [49]. Crack trapping by the cement line not only functions to slow and potentially stop the growing crack – allowing an opportunity for repair – but results in “isolation” of cracks [51] that could potentially delay or prevent the coalescence of cracks that is the final (Stage 3) phase of complete failure of the bone.

It is also clear that crack arrest plays an important physiological

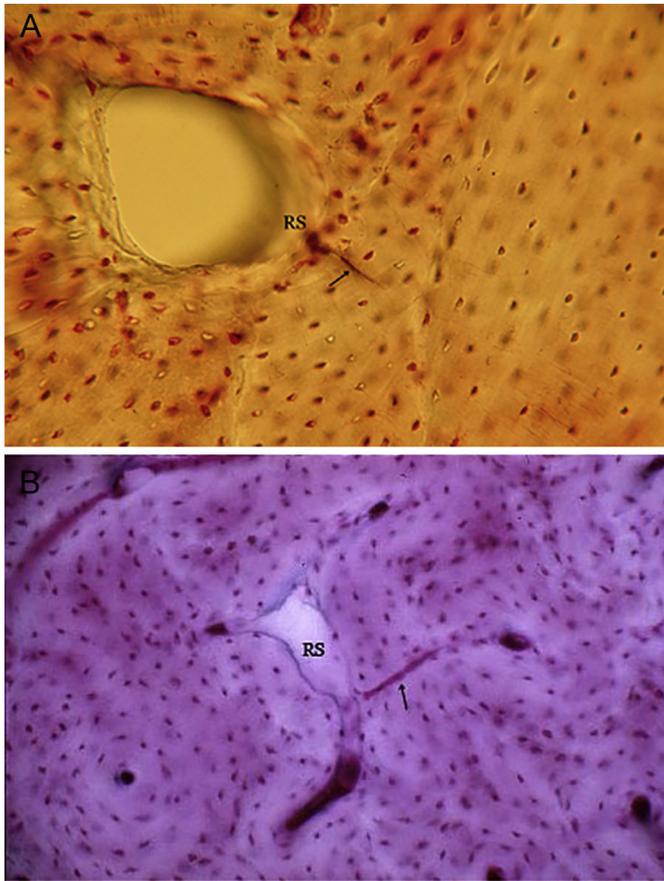


Fig. 7. (a) Microcrack (arrow) at the head of a resorption front (Rs). This resorption front is relatively round, which would reduce its stress concentrating effect. It is possible that the crack pre-existed the resorption pit, rather than the reverse, and that the remodeling front is now in the act of resorbing the crack. En bloc basic fuchsin stain, orig. mag. $32.5\times$.

(b) Another example of a resorption space (Rs, in more longitudinal section) in association with a microcrack (arrow). En bloc basic fuchsin stain, orig. mag. $32.5\times$.

function not only by preventing catastrophic failure of bone, but by allowing the bone time to mount a repair reaction that removes and replaces the damaged tissue [52]. It also may relieve shear stress - bone tends to be weak in shear, but materials with low shear strength often have excellent fatigue properties because the displacement along the shear planes relieves stress and delays failure [36,51]. The effectiveness of these discontinuities is related partly to the stiffness differential that they create in the bone matrix. Although there are differing views about whether the cement line in bone is more mineralized [34] or less mineralized [33,53], the effect of the stiffness differential on crack arrest would still be similar. The risk of fracture in bone often depends less on the magnitude of the stress or strain that initiates a linear microcrack than on the stress required to propagate it through the material [54]. The many discontinuities in bone matrix at all levels of hierarchical structure provide an ideal structure to prevent failure from cyclic fatigue processes.

Lamellar discontinuities may also provide sites for crack arrest, as they do in the hooves of horses [55]. Tubes, analogous to the Haversian canals in bone, run through the hoof and are surrounded by as many as 8 layers of cellular keratin that spiral around the hoof at different angles, in somewhat similar fashion to the lamellae around an Haversian canal. The lamellar structure in human bone, whether osteonal or primary lamellar, is somewhat more complex than this, arranged in a twisted plywood configuration, first observed by Giraud-Guille in 1988 [56]. This is an ideal configuration for stopping cracks as each time the



Fig. 8. Microcrack (arrows) being resorbed by a remodeling front. Note the osteoclasts (Oc) at the resorption front. The front end of this resorption space is cone shaped rather than round, which would increase its stress concentrating effect. Again, however, it is not clear whether the crack initiated at the head of a site of ongoing resorption, or whether the resorption was initiated and targeted to pre-existing damage. En bloc basic fuchsin stain, orig. mag. $312\times$. Reproduced with permission from John Wiley and Sons; Ref. [67].

crack arrives at a new and differently oriented interface, it changes its growth path, loses energy, slowing slightly, and eventually may be stopped. There is evidence that lamellae alone can arrest cracks as lamellar debonding can be found in areas of primary lamellae without osteons [49].

Lamellar boundaries are characterized by either variations in collagen fiber direction, or perhaps size and density of collagen fibers as suggested by Marotti [57]. This raises the question about the role that matrix heterogeneity, apart from stress concentrations, can play in the formation and growth of microdamage. Differences in elastic modulus between the interstitial bone, which is older bone and highly (or “fully”) mineralized, and bone within the boundary of the osteonal cement line, which is younger and often not fully mineralized, are known to be associated with increased longitudinal fracture toughness [58]. Guo et al. [59] modeled these stiffness differentials and showed that the difference between the modulus of interstitial and osteonal bone is a significant factor in nucleation of microcracks.

There is also evidence to suggest that heterogeneity can influence the growth and merging of cracks and can re-direct them to interfaces that can absorb energy [60,61]. Some of these processes may even be active at sub-microscopic levels of organization. Matrix heterogeneity in itself can slow the growth of microcracks in bone sufficiently to facilitate their arrest when they meet a larger architectural feature such as a lamella or cement line [62]. This is one explanation for the observation that cracks can start but also stop in primary lamellar bone that is largely devoid osteonal boundaries. Zioupos et al. [60] suggest that some energy imparted to bone is stored prior to damage formation but can help to drive the damage process once it starts. Presumably,

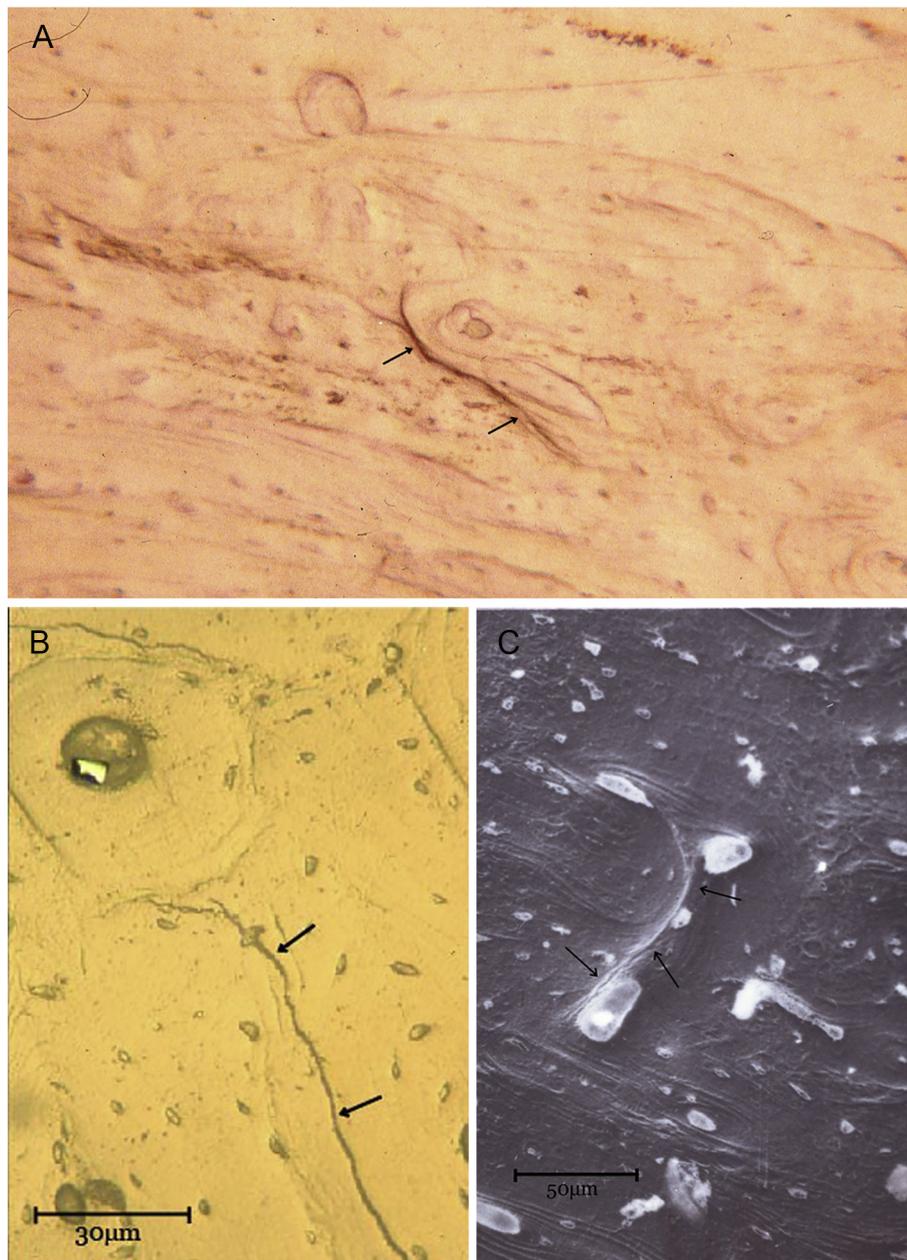


Fig. 9. Images of microcracks (arrows) being arrested at the cement line.
 (a) DIC microscopy, rabbit tibia, orig. mag. $55\times$.
 (b) DIC microscopy.
 (c) DIC microscopy.

some of this energy is stored within the collagen or within the collagen-mineral composite. Others have shown that the nature of the collagen-mineral matrix may be a nidus for, or even help to stimulate the formation of small cracks as a way to delay the formation of larger, more detrimental cracks [63]. This is likely true, although it is important not to confuse this process with a sequential process that defines diffuse damage as a precursor to microscopically visible linear microcracks directly derived from the smaller cracks. This latter view has now been discounted, and diffuse damage is considered to be a different entity, unrelated both the formation of linear microcracks and to their repair [15].

Initially, Currey proposed that Haversian canals can also act as stress concentrators in bone [8,38], as holes do in other materials. And although he seemed to continue at times to hold this opinion [38], he subsequently concluded that vascular cavities (and perhaps also

resorption cavities) did not initiate microcracks but were effective in crack deflection [29]. Because of their size (about $50\mu\text{m}$ in diameter) and shape (nearly round) canals act as very effective stress arrestors in those cases in which a crack may have breached the cement line (Fig. 4). The effect of holes in materials as stress arrestors has been shown many times and is used in nonbiological materials to prevent uncontrolled crack growth. Given this, one might consider that the increased porosity that occurs with age provides an adaptive response to prevent crack growth in a tissue that loses toughness with age. However, Ural and Vashishth [64] showed that a 4% increase in porosity in bone is associated with a 62% decrease in crack growth toughness, suggesting that porosity at least in bone is not a very good crack arrestor. An alternate explanation is that deleterious changes with age in bone material properties [65] more than offset the positive effects of pores as crack arrestors. On the other hand, a similar 4%

increase in porosity is only associated with a 6% decrease (nearly 1:1) in crack initiation toughness. This shows that holes in bone are not particularly good crack initiators but are even less effective as crack arrestors. Interestingly, when both changes in porosity and in bone tissue properties are included in the model simulations, growth toughness decreases by 83% (21% more than with porosity alone), but crack initiation toughness decreased by 51% (45% more than with porosity alone). The implication of this is that variations in crack initiation are driven more by bone material properties, whereas crack growth is affected by porosity.

6. The role of bone matrix properties in crack initiation

The discussion above suggests that there are numerous stress concentrators, and stress arrestors, in bone, some with stress concentration factors that are quite high. However, much data collected over the past 25 years suggest that these stress concentrators in bone are actually not a significant source of damage initiation. In cortical bone, 80% or more of microscopically observable damage is found to initiate within the interstitial bone [26–28], not at cement lines, resorption fronts, or Haversian or Volkmann's canals. One exception to this may be the diffuse damage that is found adjacent to osteocyte lacunae [38], or other sites of stress concentration [29]. In cancellous bone, most of the damage is found within the core of the trabeculae, not on the surface where resorption cavities usually start [42,66]. The observation that cracks form preferentially in interstitial bone is plausible because although interstitial bone contains fewer stress concentrating features than tissue that is younger, it is more highly mineralized and therefore is expected to behave in a less ductile manner. This would precipitate more damage, especially linear microcracks. A second observation is that cracks also develop in non-osteonal bone that has few stress concentrating features above the size of lacunae and canaliculi, and those cracks do not appear to begin preferentially at those interfaces.

Therefore, current data would suggest that most skeletal microdamage is not localized at sites of high stress concentration, but rather initiation is influenced by local material and physical properties of the bone tissue. This is consistent with studies done by Zioupos and Currey [29] about 30 years after Currey's original treatise on stress concentrations [8]. Zioupos and Currey [29] stated that features in bone the size predicted by linear fracture mechanics to serve such a function (from 340 μm to over a millimeter) could not be identified. They concluded that.

“we found no evidence to suggest that vascular or other naturally occurring cavities initiated cracking in laminar bone loaded in tension. The hollow vascular spaces in laminar bone appeared adequately ‘armoured’ (reinforced), cracks did not originate from them, and indeed they were usually able to deflect the microcracking, which circumvented them. Microcracking of the bone tissue was clearly related to the stress or strain experienced by the tissue locally... rather than to the dimensions of holes in the tissue that do not exist.” [p. 985]

Data collected since that time support the idea that, although many architectural features in bone tissue can serve as crack deflectors or even arrestors, material properties and the nature of the stress or strain on the bone are likely to be a greater source of crack initiation.

7. Conclusion

The microarchitecture of bone tissue presents many features that could act as stress concentrators for the initiation of bone microdamage. This was first identified by John Currey in a seminal paper in 1962 in which he presented the mechanical and biological evidence for stress concentrations at the bone surface, within the bone through the action of stiffness differentials between architectural features including between lamellae, and at the level of the lacunar and canalicular walls.

Those early observations set the stage to consider how microscopic damage to bone tissue might affect the properties of bone at a time when most in the scientific community dismissed microcracks in bone as artifact. Evidence collected in the nearly 60 years since those important initial observations suggest that some of these architectural features in bone tissue are more effective as crack arrestors than as crack initiators. Sites of higher mineralization in the bone matrix, particularly interstitial sites in both cortical and trabecular bone, may serve preferentially as locations for crack initiation, whereas those boundaries identified by Currey as both stress concentrators and stress arrestors are more effective at stopping cracks than at initiating them.

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