Introduction

Tooth crowns are exposed lumps of mineralized tissue that represent the only break in the integument of the body. Their function is to break down food particles. However, in the process of doing this, they themselves can easily be damaged. The outermost dental crown tissue is called enamel. The cause of its loss can be either chemical or mechanical. Since enamel is >90% hydroxyapatite by volume, it is vulnerable to chemical attack from acids, dissolving readily when the oral pH falls below 5.5. The erosion of enamel has become a serious problem in modern human populations [1–7], being attributed mainly to the consumption of acidic carbonated soda drinks [8]. However, gastric regurgitation [9] is also of increasing importance as a cause, particularly as the incidence of gastro-oesophageal reflux in the population increases with an increasing trend towards obesity [10].

However, the focus of this review is on mechanical damage to the enamel. This can be extensive and takes several forms (fig. 1), which are classified below in terms of the extent of the damage to enamel thickness.

Key Words
Tooth enamel · Tooth fracture · Mechanical damage

Abstract
A review is presented of the mechanical damage suffered by tooth crowns. This has been the subject of much recent research, resulting in a need to revise some of the thinking about the mechanisms involved. Damage is classified here by scale into macro-, meso- and microfracture. The focus is on the outer enamel coat because this is the contact tissue and where most fractures start. Enamel properties appear to be tailored to maximize hardness, but also to prevent fracture. The latter is achieved by the deployment of developmental flaws called enamel tufts. Macrofractures usually appear to initiate as extensions of tufts on the undersurface of the enamel adjacent to the enamel-dentine junction and extend from there into the enamel. Cracks that pass from the tooth surface tend to be deflected by an enamel region of high toughness; if they find the surface again, a chip (meso-fracture) is produced. The real protection of the enamel-dentine junction here is the layer of decussating inner enamel. Finally, a novel analysis of mechanical wear (microfracture) suggests that the local toughness of the enamel is very important to its ability to resist tissue loss. Enamel and dentine have contrasting behaviours. Seen on a large scale, dentine is isotropic (behaving similarly in all directions) while enamel is anisotropic, but vice versa on a very small scale. These patterns have implications for anyone studying the fracture behaviour of teeth.
Mesofractures start at the tooth surface from an indentation close to an enamel edge. A crack starts from the undersurface of the indented enamel growing downwards into the tissue. However, the crack is then diverted outwards towards a nearby surface. In the process, a chip of enamel, much smaller than a slab, is removed (fig. 1).

Microfractures result from indentations from very small particles on the enamel surface, leading to the loss of small fragments, i.e. to wear (fig. 1).

Does the Loss of Tooth Function Matter?

For virtually all mammals tooth loss does matter because they depend on a functioning dentition in order to eat efficiently. Tooth fractures jeopardize this activity and indications are that accumulating damage is critical to survival. The lifespan of one of the smallest short-tailed shrews, weighing 15–20 g, is limited by wear in the wild. Wild-caught animals generally only live for one breeding season, after which their teeth are heavily worn down to the gums. In contrast, young animals will live for three breeding seasons in captivity with much less wear [12]. At the other end of the scale is the African elephant, weighing 2,700–5,500 kg. Elephants bring their cheek molars into the mouth serially, only having one active molar tooth in each jaw quadrant at a time. Old individuals with very worn last molars either die or seek out vegetation that does not require chewing to swallow [13]. Data for mammals of intermediate size as diverse as primates [14] and marsupials [15–17] also support the importance of intact teeth. As an example of the effect of wear on mammalian populations, some of Pahl’s [16] data on ringtail possums are shown in figure 2, where it is clear that individuals with very worn dentitions, of whatever age, are disproportionately lost from the population.

Evidence for the effect of larger-scale crown fractures, such as wholesale cuspal fractures [18–21], enamel chipping [22–25] and the splitting of tooth crowns in half [25], are less well known, but there is no doubt that ingestion and mastication are impaired. The effect is not the same on modern human populations because of ubiquitous pre-ingestive processing and cooking of foods. These processes are of considerable antiquity [26–28], making foods easy to eat even when teeth are severely worn. However, before any of these types of fracture can be described and analysed, a short description of both the structure and mechanical properties of the tissue is needed.
Enamel Structure

Enamel forms a highly mineralized thin outer coat on the crown surface (fig. 1, 3). It consists mostly of hydroxyapatite crystals embedded in a matrix of protein and water. Each crystal is between 0.1 and 1 mm in length with cross-sectional dimensions of 30–70 nm [29]. The crystals are thus much bigger than those in other mineralized tissues, such as dentine and bone, and they occupy over 90% of the tissue volume. A few thousand crystals are bundled together into elongated structures called prisms, each 3–5 μm in diameter. Prisms extend almost all the way from the enamel-dentine junction to the tooth surface. The gap between crystals within a prism is only 1–2 nm, but between prisms there are much larger 100-nm crystal-free gaps (fig. 3a), occupied by low molecular weight proteins and water [30]. In the inner enamel, prisms follow a sinuous path (fig. 3b). Adjacent prisms in the longitudinal plane are successively slightly out of phase with each other [31]. The successive phase change leads to the appearance of small parcels of prisms crossing each other, a characteristic called decussation (fig. 3c). In humans, the inner 60–80% of enamel thickness consists of decussating prisms. However, the prisms straighten in the outer enamel, passing in parallel to the tooth surface (fig. 3b). This type of enamel is often called ‘radial enamel’ to contrast it with ‘decussating enamel’.

In addition to prisms, there are a number of minor structures in enamel that were previously thought to be trivial, but which are now known to be of major importance. Foremost among these are enamel tufts (fig. 3b). These hypocalcified strands, formed during development, resemble widened prism sheaths and extend from the enamel-dentine junction about one third of the way into the enamel [32, 33]. Previously thought to be a curiosity, they are now implicated as a key protective mechanism of the tooth crown.

A précis of the development of the tissue is important for understanding some of its features. When the tissue is first formed, the crystallites are thin and large proteins that form the scaffolding of the tissue, and which initiate crystallization, occupy the bulk of the tissue. However, after the enamel thickness is completely formed, the cells that secrete the tissue (ameloblasts) convert into cells that somewhat resemble those lining the small intestine. They
6 start to produce digestive enzymes (matrix metalloproteinas) that break the protein scaffolding down into fragments [34]. The ameloblasts then draw most of these protein pieces, and also much of the water, out from the tissue. This removal allows the crystals to expand in breadth into newly created space. In behaving like this, ameloblasts act at a distance of up to 2 mm and their effectiveness must thus be a function of a diffusion gradient. This appears to set up a parallel gradient in the mature tissue in chemical composition [35–39] that is also mirrored in some mechanical properties, as described below.

**Enamel Mechanical Properties**

The elastic modulus $E$ of enamel is its resistance to elastic deformation. This is measured in force per unit area (newtons per metre squared: Nm$^{-2}$). The SI abbreviation for this unit is the pascal (Pa) with 1 Pa = 1 Nm$^{-2}$. However, this is such a small unit that gigapascals (1 GPa = 10$^9$ Nm$^{-2}$) are required to reduce the number of digits in describing most materials. Mature human enamel is very stiff with an $E$ varying between 70 and 110 GPa [40]. The hardness $H$ of enamel is its resistance to plastic (permanent) deformation. It is again measured in force...
per unit area (newtons per metre squared: Nm$^{-2}$). Human enamel is very hard with $H = 3$–6 GPa (fig. 2a) [40]. Both the elastic modulus and hardness are linearly graded, with the lowest values near the enamel-dentine junction and the highest at the tooth surface [40–43]. The properties of the enamel of other mammals vary slightly, but a gradient of hardness and elastic modulus is again usually present [44–47]. There are unlikely to be big surprises in enamels yet to be examined because both modulus and hardness are subject to a ‘rule of mixtures’ wherein the composite tissue is constrained by the stiffest, hardest component of which it is made [48, 49]. The upper limits for human enamel fall close to those reported for pure hydroxyapatite [50].

The major mechanical benefit of composite materials constructed from several components, such as enamel, lies not in its stiffness or hardness, but in its fracture toughness, which is the ability of an object to resist the growth of cracks [51]. The toughness of enamel is not graded in the manner of the elastic modulus and hardness because, unlike these properties, it is not bound by limits set by its components. Instead, it benefits from a synergism between these components that produce high toughness in the composite, compared to components that, in isolation, will break very easily. There are two ways to measure toughness, which are easily interconverted. Here we refer to it as ‘fracture toughness’, which is the effect that a crack of unit length has on the intensity of stress in a loaded object. This intensity depends on the square root of crack length and thus has units of MNm$^{-2}$ m$^{0.5}$ or MPa m$^{0.5}$. It is symbolized here as $K_c$. This quantity varies in enamel not in the form of a gentle gradient, but as a function of enamel structure. For very small cracks of the order of size of a few prism diameters (fig. 4a), structure does not matter and the toughness of enamel is uniformly low [52], resembling that of glass [53]. For larger cracks, everything depends on whether they can track along prism sheaths without deviating or whether they encounter decussation [54], in which case the toughness becomes elevated (fig. 4b, c). Toughness appears to be more elevated when cracks are run from outside in, i.e. the crack starts at the tooth surface and projects towards the dentine (fig. 4b), than when cracks are directed from the inside to outside (fig. 4c) [55]. The ‘inside-out’ crack typically becomes unstable in a test because the crack is being opened directly. However, in life, such cracks would be
loaded not in direct tension but in compression. Crack opening then depends on tension being developed indirectly at right angles to the load. Under these circumstances, ‘inside-out’ cracks in enamel have been shown to be completely stable [56–59].

A comparison of the properties of enamel and dentine is important in understanding how the whole crown behaves. At the scale of a few prism diameters or less, enamel is effectively isotropic. This makes experimentation on the microfractures that lead to wear entirely tractable. However, on this same scale, dentine is very anisotropic, containing large cylindrical holes surrounded by relatively stiff, hard intratubular (or peritubular) dentine. Between tubules, the intertubular dentine is of much lower elastic modulus and hardness (fig. 3d) [60]. Studies of dentinal wear at the level of the actual mechanical events would thus be extremely difficult. However, at any larger scale, the situation is reversed. Enamel is then very anisotropic, showing the gradient of elastic modulus and hardness, coupled with a strong influence of structure on toughness. In contrast, most of the primary dentine – that formed with the tooth and which abuts the enamel – is relatively homogeneous, with the exception of a thin strip of ‘mantle dentine’ directly under the enamel, which is slightly softer and less stiff [61–63]. The toughness of dentine varies little for long cracks, hovering between 1.6 and 3 MPa m$^{0.5}$, but decreasing with dentinal age, presumably because of the gradual accumulation of intratubular dentine [64–67]. As a consequence of its large-scale uniformity, large cracks in dentine have no real preferred orientation dictated by dentinal structure. In contrast, the structure of enamel has a major directing effect [68].

The Importance of Scale

The main ‘scaling factor’ that affects enamel damage is nothing intrinsic to the tissue itself, but is a consequence of the effect of the size of particles that contact its external surface [69]. A small particle, of hardness >2.5 times that of enamel [70–72], has its effect principally on the tooth surface, indenting/sliding against it at very low forces, causing either plastic deformation or fracture of the enamel (fig. 5a). In contrast, contact with a much larger particle of the same hard material causes the enamel to bend. If the enamel cracks, then it will do so on its deep surface (fig. 5b). Lastly, a contact with a large particle of low hardness, <2.5 times that of enamel, can still damage it. Bending of the enamel under the contact is not likely to produce cracking though, because deformation of the particle will produce a large contact area and a large volume under hydrostatic compression that will suppress fracture. There-

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**Fig. 5.** The damage that enamel sustains due to contacts with hard objects (direction and size of force given by the arrows) depends on scale. **a** A small particle that is much harder than enamel tends to indent its surface, producing microfracture (‘wear’) if the particle slides. **b** A large hard particle is more likely to produce a crack on the undersurface of the enamel directly under the contact (a ‘radial’ macrofracture) due to enamel flexure. **c** A softer large particle deforms between teeth. It can still damage the enamel via bending forces, but only away from the contact area, thus forming a ‘margin’ macrofracture.
fore, when sufficient strain energy is available for fractures to be initiated, they are likely to start well away from the zone of contact where tension can be expressed (fig. 5c). We now discuss the type of fractures seen in teeth with respect to the mechanical behaviour of the tissues.

**Macrofracture**

Large-scale damage to a tooth crown is completely incompatible with its continued use. The most common form of such fracture is a through-enamel crack called a lamella (fig. 1). Lamellae are absent from newly erupted teeth, but accumulate during life. Although such lamellae were long thought to start at the enamel surface and progress as ‘outside-in’ cracks [73], experimental evidence now suggests that instead they grow from inside to outside as extensions of enamel tufts [57, 58]. There is a forest of tufts all around the enamel-dentine junction ready to pick this strain energy up [74, 75]. These cracks grow slowly, requiring a sharply increasing force to extend [57, 58]. The relationship between crack extension and force seeking the shortest path to a surface again along a sheath. The lower box shows the typical toughening mechanism of composites. A force drives a primary crack. That crack meets an interface of low stiffness and deflects. The force cannot easily push the primary crack further, but if the force builds, a secondary crack can initiate on the other side of the interface, leaving a ‘bridge’ between the two that will eventually fail, causing fragmentation.

**Fig. 6.** The dependence of different scales of fracture events on enamel properties and tooth crown dimensions. Relationships have been firmly established for radial, margin and chipping fractures. Note that the degree of dependence on crown dimensions varies. These relationships were established by Lee et al. [76] and Lucas et al. [86]. The upper box shows the usual path of a chip (small arrows show its growth path), following a prism sheath in the outer radial enamel, being deflected by decussation and then

is known, with a dependence on enamel toughness and thickness, but additionally on the square root of tooth size. The presence of lamellae can be used to establish a history of force use on a tooth [76].

In themselves, lamellae are relatively innocuous. However, the major issue with these types of crack is a potentially catastrophic fracture, which results in the loss of large slabs of enamel, either part of a cusp (against a hard particle; fig. 5b) or part of the margin of a tooth (against a softer particle; fig. 5c). Margin fractures are often called ‘abfractions’ in the human dental literature [77]. They depend on tooth dimensions in a different manner to that which affects cuspal (radial) cracks (fig. 6). This is because they stem from tensile stresses known as ‘hoop’ stresses [59]. These stresses gain their name from the design of barrels, where metal hoops around the circumference stop internal cracks in the barrels from propagating outwards. Human teeth are very vulnerable to such margin cracks because they lack any protection in the region where the tooth crown meets the root (fig. 1). This marginal region is buttressed in many mammals, but not in humans [69, 78].
Mesofracture

The chipping of enamel (fig. 1) is very common in mammals in general [22–25], being particularly frequent in certain archaic and modern human populations [79–81]. It is also often seen by dentists in a clinical setting, where it is mostly viewed as an aesthetic issue [82]. Yet, a general theory of chipping resistance in materials has only recently started to be developed [83]. Provided the complexities of resistance to fracture seen in human enamel are ignored, and the tissue is treated as responding elastically with little toughness, then a remarkably simple treatment of chipping resistance in ceramic materials can be applied to enamel [84]. Evidence suggests that a crack starts at the surface at a contact close to an edge as what are termed ‘median’ vents [84]. As it extends downwards, energetic considerations favour the crack turning towards the edge, so releasing a chip of tissue. The analysis of chipping resembles those for macrofractures that start from deep in the enamel, but with a differing dependence on tooth dimensions (fig. 6). Again, chipping gives evidence of the force that produced it [24].

A crack that starts on the tooth surface and grows from outside inwards would encounter increasing resistance as it does so because inner decussating enamel would obstruct its progress (fig. 4b). Thus, part of the explanation for the number of chips on human teeth, and the relative rarity of cracks that have grown from outside inwards to reach the enamel-dentine junction, is that the inner decussating enamel is there to protect the integrity of the tooth (fig. 6). A mesofracture is preferable to a split crown that could result if the crack continued its original path and passed into the dentine.

Microfracture

Microfracture refers to the mechanical wear of the tooth surface whereby small amounts of material are lost from the crown surface. These events accumulate over time to be seen by the naked eye as macroscopic wear (fig. 1). Dentists distinguish tissue lost from tooth-tooth contacts, which they call attrition, from abrasion, which is damage produced by small foreign objects that enter the mouth [85]. Here, we use terms that refer to the processes involved. ‘Abrasion’ involves the removal of tissue from a surface either as a ribbon that curls away from the surface (as in the wear of metals) or as a chip. It leaves marks that are sharp V-shaped grooves (fig. 7). Only small particles that are >2.5 times as hard as enamel can do this. Particles that are less hard can only produce plastic deformation of the enamel surface. This is called ‘rubbing’. Material can be moved on the surface, forming a groove surrounded by a mound of tissue, but no material is immediately lost. Such mounds are not seen with abrasion (fig. 7).

Recent evidence suggests that abrasion can take place at forces of 1 mN or less [86], which is negligible considering that maximum bite forces in modern humans can reach 1 kN, six orders of magnitude higher [24]. Clearly, if conditions are right, large amounts of wear can take place by abrasion. Several factors help to prevent this. Firstly, abrasion depends on the presence of ingested particles that are much harder than enamel. It also depends on sliding contacts because this is much more important in producing wear than static contacts. No food tissue eaten by any mammal is as hard as enamel, but grit and dust particles that enter the mouth ingested with food can be much harder [86]. Luckily, unless these particles are sufficiently sharp, this will not result in abrasive wear [83, 87, 88]. The criterion for ‘sufficient sharpness’ is actually set by enamel toughness. Most airborne particles are actually quite smooth-surfaced, and the only way to generate sufficient sharpness is if the particles are broken in the mouth. This produces an audible noise and, luckily, mechanoreceptors in the periodontal ligament of the tooth (fig. 1) are present to pick up the sub-newtonian loads at which dust and grit particles break, so alerting an individual to damage [89]. Very soft particles of whatever size do little mechanical damage to an enamel surface, but there exists an in-between category of particle that is roughly as hard as enamel. Such particles can rub the
Tooth surface, indenting it and doing damage very similar to that which teeth do to each other [86]. Such damage, however, is a plastic indentation without any immediate resulting tissue loss, so strictly it is not wear when considered as a single event.

**Overview of Mechanical Damage to Enamel**

For the first time, several of the patterns of damage to a tooth crown can be expressed quantitatively in terms of the force that produces them (fig. 6). The extent to which properties and crown dimensions affect this force depends on the type of fracture. Margin and radial cracks, despite their similar origin near the enamel-dentine junction (fig. 5b, c), depend on the local thickness of the enamel and on crown dimensions, but with differing exponents (fig. 6). While it is difficult to express resistance to mechanical wear in the same detail, it is clear that enamel hardness (compared to that being contacted) and toughness are the critical properties that resist damage.

The discovery that a standard pattern of cracking in enamel runs ‘inside-out’ has many implications for researchers investigating the protection of a tooth, unknown even a few years ago [90]. Currently, much research is focused on how the enamel-dentine junction is protected from fractures that cross from the enamel into the dentine [63, 91–96]. It might be ironic if most of the protection came from the fact that the general direction of cracks is from close to the enamel-dentine junction, travelling in the opposite direction to that which would fracture into dentine [24]. The role of the mantle dentine may be to encourage the enamel to flex, and thus to strain the tufts (fig. 5b, c). The tufts extend only when forces are high enough [97], thus protecting the dentinal core by directing cracks in the opposite direction. Such cracks that do pass from the exterior inwards may well be deflected by decussating enamel; of course, this may sacrifice a chip of enamel, but it saves the tooth (fig. 6).

The factors that control the splitting of tooth crowns in half are less well known. One theory proposes that the relation for chipping (equation in fig. 6 and top inset) applies also to splitting [25], with the difference that the latter is a consequence of a more central position for crack initiation on the crown [24]. Recent developments have extended some of this theory to variation in crown height [98] and to include the presence of multiple cusps [99].

Judging from experiments [52, 54, 55], the cracking of enamel in macrofractures is likely to follow the typical toughening pattern of composites shown in figure 6 (bottom inset). A primary crack traverses a homogeneous region of the material, e.g. a prism core. It then reaches an interface, which we could identify in enamel as the prism sheath. Here, the crack deflects because the stiffness of the small-protein gel in the sheath is so low that strain energy cannot be built up to advance it; thus, it opens for a short distance along the interface [100]. If the force continues to build, then a secondary crack may initiate on the other side of the sheath in the next prism core. The toughening comes from the fact that the force to start this secondary crack is far higher than that which started the primary one, with the ratio depending on the width of the interface [101]. The gap between the two cracks then remains as a bridging ligament that restricts the opening of the whole fracture as it advances. This pattern is repeated across successive interfaces until, in the end, the ligaments fail with eventual union of the whole crack and the loss of material. This mechanism can amplify toughness in an extraordinary manner, sometimes to be several thousand times higher than the toughness of isolated components. The organization of biological composite tissues is designed to promote the effectiveness of these mechanisms. Teeth do not ‘want’ to be fractured, and virtually no biological tissue ‘wants’ to end up being broken down by the teeth as food. Thus, a composite structure is common to most biological tissues.

**Conclusion**

There is still a lot to learn about tooth crown fractures, and little has yet been done at the same level on root fractures, which are clinically important. Yet the groundwork has been laid for a new era of understanding, particularly of enamel. Some say that the tissue behaves like metal [102], others that it is glassy [58]. We conclude here that, dependent on the influence of structure and the scale at which it acts, both may be correct.

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Lucas PW, Constantino PJ, Chalk J, et al: Indentation as a technique to assess the mechanical properties of fallable foods. Am J Phys Anthropol 2009;140:643–652.

Bajaj D, Arola DD: On the R-curve behavior of human tooth enamel. Biomaterials 2009;30:4037–4046.

Yahyazadehfar M, Bajaj D, Arola DD: Hidden contributions of the enamel rods on the fracture resistance of human teeth. Acta Biomat 2013;9:4806–4814.

Lawn BR, Lee JJ, Constantino PJ, et al: Predicting failure in mammalian enamel. J Mech Behav Biomed Mat 2009;2:33–42.

Lee JJ-W, Kwon J-Y, Chai H, et al: Fracture modes in human teeth. J Dent Res 2009;88:224–228.

Chai H, Lee JJ-W, Constantino P, et al: Remarkable resilience of teeth. Proc Natl Acad Sci USA 2009;106:7289–7293.

Chai H, Lee JJ-W, Kwon J-Y, et al: A simple model for enamel fracture from margin cracks. Acta Biomat 2009;5:1663–1667.

Kinney JH, Balooch M, Marshall SJ, et al: Hardness and Young’s modulus of human peritubular and intertubular dentine. Arch Oral Biol 1996;41.9–13.

Osborn JW: Dentine hardness and incisor wear in the beaver (Castor fiber). Acta Anat 1969;72:123–132.

Renson CE, Braden M: The experimental deformation of human dentine by indenters. Arch Oral Biol 1971;16:563–572.

Zaslansky P, Friesem AA, Weiner S: Structure and mechanical properties of the soft zone separating bulk dentin and enamel in crowns of human teeth: insight into tooth function. J Struct Biol 2006;153:188–199.

El Mowafy OM, Watts DC: Fracture toughness of human dentin. J Dent Res 1986;65:677–681.

Iwamoto N, Ruse ND: Fracture toughness of human dentin. J Biomed Mater Res A 2003;66:507–512.

Imbeni V, Nalla RK, Bosi C, et al: In vitro fracture toughness of human dentin. J Biomech Mater Res A 2003;66:1–9.

Nazari A, Bajaj D, Zhang D, et al: Aging and the reduction in fracture toughness of human dentin. J Mech Behav Biomed Mat 2009;2:550–559.

Waters NE: Some mechanical and physical properties of teeth; in Vincent JFV, Currey JD (eds): The Mechanical Properties of Biological Materials. Cambridge, Cambridge University Press, 1995, pp 99–135.

Lucas PW, Constantino P, Wood BA, et al: Dental enamel as a dietary indicator in mammals. Bioessays 2008;30:374–285.

Tabor D: Hardness of Metals. Oxford, Clarendon, 1951, pp 1–175.

Atkins AG, Felbeck DK: Applying mutual indentation hardness phenomena to service failures. Met Eng Q 1974;2:55–61.

Atkins AG: Topics in indentation hardness. Met Sci 1982;16:127–137.

Sognnaes RF: The organic elements of enamel. IV. The gross morphology and the histological relationship of the lamellae to the organic framework of the enamel. J Dent Res 1950;29:260–269.

Amizuka N, Uchida T, Fukae M, et al: Ultrastructural and immunocytochemical studies of enamel tufts in human permanent teeth. Arch Histol Cytol 1992;55:179–190.

Amizuka N, Uchida T, Nozawa-Inoue K, et al: Ultrastructural images of enamel tufts in human permanent teeth. J Oral Biol 2005;47:3–11.

Lee JJ-W, Constantino P, Lucas PW, et al: Fracture in teeth – a diagnostic for inferring tooth function and diet. Biol Rev 2011;86:959–974.

Grippi JO: Abrasion: a new classification of hard tissue lesions of teeth. J Esthet Dent 1991;3:14–18.

Anderson PSL, Gill PG, Rayfield EF: Modeling the effects of cingula structure on strain patterns and potential fracture in tooth enamel. J Morphol 2011;272:50–65.

Turner CG, Cadien JD: Dental chewing in Aleuts, Eskimos and Indians. Am J Phys Anthropol 1969;31:303–310.

Hylander WL: The adaptive significance of Eskimo craniofacial morphology; in Dahlberg A, Garber TM (eds): Orofacial Growth and Development. Paris, Mouton, 1977, pp 129–169.

Scott GR, Winn JR: Dental chewing: contrasting patterns of microtrauma in Inuit and European populations. Int J Osteoarchaeol 2011;21:723–731.

Andreasen JO, Andreassen FM, Andersson L: Inferring biological evolution from fracture patterns in teeth. J Theoret Biol 2013;338:59–65.

Barani A, Bush MB, Barani A, et al: Tooth enamel fracture and susceptibility to matrix damage in dentin. J Biomed Mater Res 2013;5:55–61.

Lucas PW, van Casteren A, Al-Fadhahal K, et al: The role of dust, grit and photoliths in tooth wear. Ann Zool Fenn 2014;51:143–152.

Lucas PW, van Casteren A, Al-Fadhalah K, et al: The role of dust, grit and photoliths in tooth wear. Ann Zool Fenn 2014;51:143–152.

Lucas PW, van Casteren A, Al-Fadhahal K, et al: The role of dust, grit and photoliths in tooth wear. Ann Zool Fenn 2014;51:143–152.

Lucas PW, van Casteren A, Al-Fadhahal K, et al: The role of dust, grit and photoliths in tooth wear. Ann Zool Fenn 2014;51:143–152.

Lucas PW, van Casteren A, Al-Fadhahal K, et al: The role of dust, grit and photoliths in tooth wear. Ann Zool Fenn 2014;51:143–152.

Lucas PW, van Casteren A, Al-Fadhahal K, et al: The role of dust, grit and photoliths in tooth wear. Ann Zool Fenn 2014;51:143–152.

Lucas PW, van Casteren A, Al-Fadhahal K, et al: The role of dust, grit and photoliths in tooth wear. Ann Zool Fenn 2014;51:143–152.

Lucas PW, van Casteren A, Al-Fadhahal K, et al: The role of dust, grit and photoliths in tooth wear. Ann Zool Fenn 2014;51:143–152.

Lucas PW, van Casteren A, Al-Fadhahal K, et al: The role of dust, grit and photoliths in tooth wear. Ann Zool Fenn 2014;51:143–152.

Lucas PW, van Casteren A, Al-Fadhahal K, et al: The role of dust, grit and photoliths in tooth wear. Ann Zool Fenn 2014;51:143–152.

Lucas PW, van Casteren A, Al-Fadhahal K, et al: The role of dust, grit and photoliths in tooth wear. Ann Zool Fenn 2014;51:143–152.

Lucas PW, van Casteren A, Al-Fadhahal K, et al: The role of dust, grit and photoliths in tooth wear. Ann Zool Fenn 2014;51:143–152.

Lucas PW, van Casteren A, Al-Fadhahal K, et al: The role of dust, grit and photoliths in tooth wear. Ann Zool Fenn 2014;51:143–152.