The Complexity of Dental Enamel

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Dental enamel constitutes the least quantitative and rarest component of all tissues in the human body but is the most enduring and hardest constituent of ectodermal cellular development. Dental enamel can be a harbinger of history, reflecting the environment during the time it was being formed. Enamel first appeared around 415 million years ago when the suite of genes that encode the proteins required to make enamel appeared in the scales of sarcopterygians.[1]

The vitreous nature of enamel provides its white lucent, iridescent and gleaming appearance as a physically attractive feature of a human smile, but contrarily, it may provide a snarling repulsive warning of a sneer.

The production of enamel by ameloblasts is among the most complex tissues of histogenesis. Amelogenesis is so specialized in its production of enamel that it is not replaceable as enamel in any form of tissue repair. Accordingly, any damage to enamel, be it by acidogenic decay or trauma has become the raison d 'etre for the dental profession in repairing the consequences of enamel loss.

Enamel is an instantaneous fossilized tissue developing in situ in during amelogenesis in living individuals that consequently reflects the environmental and metabolic status of an individual. Any deviation of the genetically determined pathway of enamel formation is permanently imprinted upon the histology of enamel, providing enduring evidence of the dysmorphogenesis. Hence, the genetically determined condition of amelogenesis imperfecta is engrafted upon enamel during its formation and is revealed post-eruption on the teeth exhibiting hypoplasia. Moreover, enamel is unique in both providing information on extant living individuals, and on long-deceased and extinct fossilized species.

Enamel formation

The elaboration of enamel as a complex combination of enamelin, tuftelin, amelotin, ameloblastin, amelogenin, tuftelin interactive protein 11, beta defensin 1, matrix metallopeptidase 20 and enamel structure variables secreted by ameloblasts that take a path from the delicate scalloped amelodentinal junction to the destined cusp tip and down into the crevices of the fissures and along the ultimate enamel margins.[2] The variation of the thickness of enamel at these different locations must presumably be genetically determined by ameloblast viability, diminishing from the peaks of the tooth cusps to the ultimate cessation of amelogenesis at the enamel margins. It would be interesting to investigate the longevity of short-lived marginal ameloblasts if they could be transferred to longer lived cusp tip locations. Is their fate determined by genes or by location?

During amelogenesis, the physical or epigenetic interactions between the inner and outer layers of the enamel organ might determine enamel thickness. The ameloblastin and amelogenin matrix proteins formed during early stages of amelogenesis are removed during maturation by degradation. The accumulated degraded matrix proteins inhibit further ameloblast activity, thereby determining the thickness of deposited enamel. Such interactions may modulate different ameloblast location longevity, and hence, enamel thickness. Ameloblasts located at a molars' highest cusps can produce enamel as thick as 6 mm.

The thickness of dental enamel may act as a proxy for the durability of a tooth and reflect dietary preferences and possibly the lifespan. Enamel thickness has been studied in Plio-Pleistocene hominin mandibular molars, wherein the thick enamel of the robust Australopithecus species decreases in early Homo to that of modern humans.[3] The enamel thickness of Gigantopithecus blacki, over 6 mm in places, was adapted to heavy wear in a way that differed from Pliocene and Pleistocene hominids.[4] G. blacki was adapted to consuming tough fibrous food, and its thick molar enamel allowed for relative longevity.[5] There might be a correlation between molar wear patterns and enamel thickness associated with dietary changes.[6]

Dental Enamel is the hardest tissue in the body that is initiated in a protein gel that is impregnated by nanometer size minerals in a three-dimensional network. The remarkable strength of enamel comes from its ingenious structure that gives it the hardness and toughness to resist the start and spread of cracks. The newly formed enamel matrix is an amorphous calcium phosphate that transforms into apatite crys-

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tals. The unique resilience of enamel to fracturing is due to the misorientations of the nanocrystals within enamel rods to deviate from one another that deflects cracks.[7] The enamel rods or prisms run parallel to one another from the tooth surface to the underlying dentin, but weave and twist as they go by an elegant configuration that confers the significant durability of enamel. The biochemical constitution of enamel is apatite (Ca5(PO4)3(F, Cl, OH) that is constituted of hydroxylapatite, fluorapatite and chlorapatite. Fluorapatite is the least susceptible component to acid disintegration, making it the reason for fluoridation of drinking water to reduce dental decay. The opportunity to regenerate dental enamel has been explored.[8]

Spectroscopic analysis of enamel formed during a period of high radioactivity of 14C isotopes in the atmosphere, as at the time of the Chernobyl explosion, can identify the date of enamel formation. Similarly, the 18C content of dental enamel provides evidence of ingested vegetation during wet or dry periods occurring during amelogenesis. Further, the administration of tetracycline antibiotics during enamel formation is permanently imprinted on teeth, revealed after their eruption.[9]

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