AMELOGENESIS

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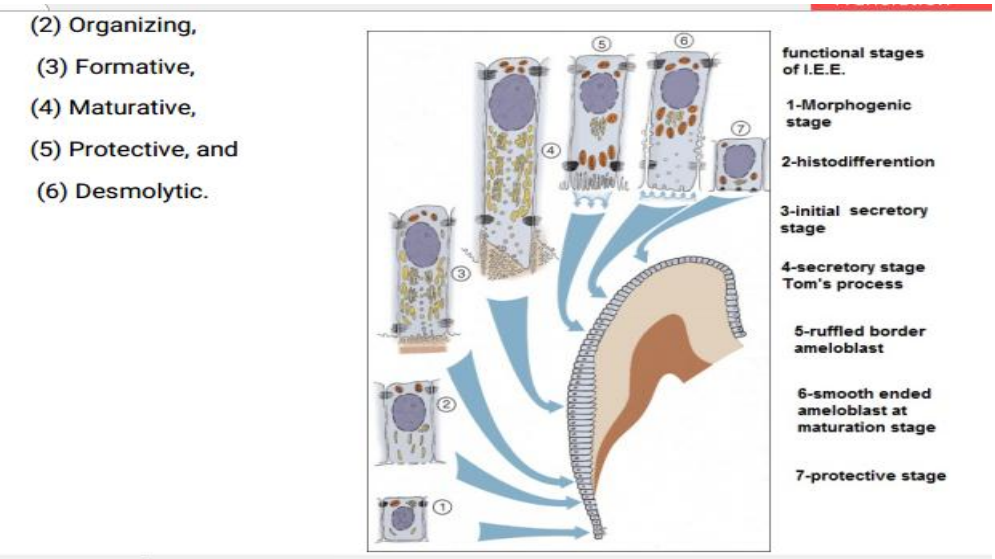
Life cycle of the ameloblasts
According to their function, the life span of the cells of the inner enamel epithelium can be divided into six stages:
(1) Morphogenic ,
(2) Organizing,
(3) Formative,
(4) Maturative,
(5) Protective, and
(6) Desmolytic.



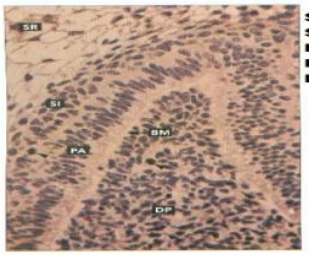
Since the differentiation of ameloblasts is most advanced in the region of the incisal edge or tips of the cusps and least advanced in the region of the cervical loop, all or some stages of the developing ameloblasts can be observed in one tooth germ. Amelogenesis which is the formation of enamel occurs during formative and maturative stages of the ameloblasts.

***1-Morphogenic stage**:*

Before the ameloblasts are fully differentiated and produce enamel, they interact with the adjacent mesenchymal cells (Dental.Papilla) which differentiate into odontoblasts. determining the shape of the DEJ and the crown.

During this stage; the cells are short and columnar, with large oval nuclei that almost fill the cell body.

The Golgi apparatus and the centrioles are located in the proximal end of the cell, whereas the mitochondria are evenly dispersed throughout



SR: stellate reticulum
SI:stratum intermedium
PA:preameoblasts
BM:basement membrane
DP:dental papillae

the cytoplasm. During ameloblast differentiation; the inner enamel epithelium is separated from the connective tissue of the dental papilla by a delicate basal lamina.

2-Organizing stage:

This stage is characterized by a change in the appearance of the cells of the inner enamel epithelium.

They become longer, and the nucleus-free zones at the distal ends of the cells become almost as long as the proximal parts containing the nuclei.

At the same time the clear cell-free zone between the inner enamel epithelium and the dental papilla disappears probably because of elongation of the epithelial cells toward the papilla.

Thus the epithelial cells come into close contact with the connective tissue cells of the pulp, which differentiate into odontoblasts

Preameloblasts secrete proteins similar to those of enamel matrix.

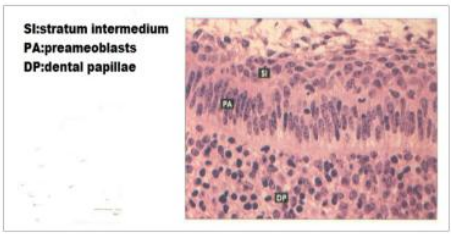
These proteins appear to be phagocytosed by developing odontoblast may play a role in epithelial mesenchymal interaction.

During the terminal phase of the organizing stage the formation of the dentin by the odontoblasts begins.

As long as Preameloblast in contact with the connective tissue of the dental papilla, it receives nutrient material from the blood vessels of this

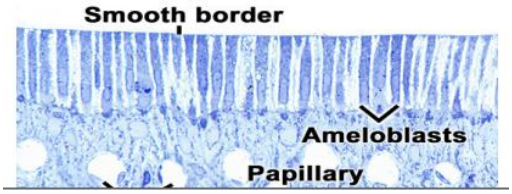
tissue.
When dentin forms, however, it cuts off the ameloblasts from their original source of nourishment, and from then ;they are supplied by the capillaries that surround and may even penetrate the outer enamel epithelium.

This reversal of nutritional source is characterized by proliferation of capillaries of the dental sac and by reduction and gradual disappearance of the stellate reticulum.
Thus the distance between the capillaries and the stratum intermedium and the ameloblast layer is shortened.



3-Formative stage:
The ameloblasts enter their formative stage after the first layer of dentin has been formed.
During formation of the enamel matrix the ameloblasts retain approximately the same length and arrangement.
The earliest apparent change is the development of blunt cell processes on the ameloblast surfaces, which penetrate the basal lamina and enter the predentin called Toms' process.
The junctional complexes which encircle the ameloblast at their distal and proximal ends have fine radiating actin filaments extending into the cytoplasm, forming webs.

These serve to control the substances that pass between ameloblast and enamel.
The juctional complexes which form at the distal end are called distal terminal bars. These terminal bars separate the Tomes' processes from the cell proper.
Secretions from areas close to junctional complexes and from adjacent ameloblasts form the inter-rod enamel.

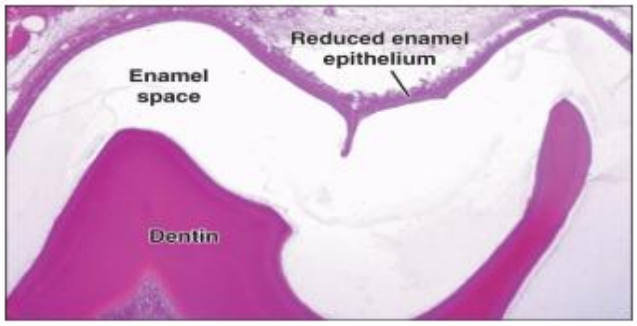


4-Maturative stage:

Enamel maturation (full mineralization) occurs after most of the thickness of the enamel matrix has been formed in the occlusal or incisal area.
In the cervical parts of the crown, enamel matrix formation is still progressing at this time.
During enamel maturation the ameloblasts are slightly reduced in length and are closely attached to enamel matrix.
The cells of the stratum intermedium lose their cuboidal shape and regular arrangement and assume a spindle shape.It is certain that the ameloblasts also play a part in the maturation of the enamel.
During maturation, ameloblasts display microvilli at their distal extremities, and cytoplasmic vacuoles containing material resembling enamel matrix are present. These structures indicate an absorptive function of these cells.

5-Protective stage

When the enamel has completely developed and has fully calcified, the ameloblasts cease to be arranged in a well-defined layer .
These cell layers then form a stratified epithelial covering of the enamel, the so-called reduced enamel epithelium.
The function of the reduced enamel epithelium is that of protecting the mature enamel by separating it from the connective tissue until the tooth erupts.
If connective tissue comes in contact with the enamel, anomalies may develop.
Under such conditions the enamel may be either resorbed or covered by a layer of cementum.
The adjacent mesenchymal cells may then deposit afibrillar cementum on the enamel surface.



6-Desmolytic stage:

The reduced enamel epithelium proliferates and seems to induce atrophy of the connective tissue separating it from the oral epithelium, so that fusion of the two epithelia can occur.

It is probable that the epithelial cells elaborate enzymes that are able to destroy connective tissue fibers by hemolysis.

Premature degeneration of the reduced enamel epithelium may prevent the eruption of a tooth.

1-Formation of the enamel matrix:

The ameloblasts lose the projections that had penetrated the basal lamina separating them from the predentin, and islands of enamel matrix are deposited along the predentin. As enamel deposition proceeds, a thin, continuous layer of enamel is formed along the dentin.

Amelogenin: is the major component of enamel matrix proteins.

It undergoes extracellular degradation by proteolytic enzymes like matrix metalloproteinases into smaller low molecular weight fragments, have specific functions as in regulating crystal growth.

The changes occurring in the ameloblasts after secretory stage and prior to the onset of maturation process are called transition stage.

The ruffle ended ameloblasts show numerous lysosomes and possess endocytic activity. They also promote calcium entry into the forming enamel.

Calcium ions pass actively through the ruffle ended ameloblasts and passively through the sides of the smooth ended ameloblast to the mineralizing front.

Ruffle ended ameloblasts secrete bicarbonate ion to keep the mineralizing

front alkaline, prevent acidification and thereby helps to keep the mineralization process to continue organic components as well as water are lost in mineralization is a striking difference between enamel and other mineralized tissues. Over 90% of the initially secreted protein is lost during enamel maturation, and that which remains in the area of the prism sheath where the abrupt change in crystal orientation occurs.

2-Mineralization and maturation of the enamel matrix:

Mineralization of the enamel matrix takes place in two stages, although the time interval between the two appears to be very small.

In the first stage an immediate partial mineralization occurs in the matrix segments and the interprismatic substance as they are laid down. Nucleation is initiated by the apatite crystallites of dentin on which enamel is laid. The second stage, or maturation, is characterized by the gradual completion of mineralization.

The process of maturation starts from the height of the crown and progresses cervically maturation seems to begin at the dentinal end of the rods.

Thus each rod matures from the depth to the surface, and the sequence of maturing rods is from cusps or incisal edge toward the cervical line.

Maturation begins before the matrix has reached its full thickness.

The advancing front is at first parallel to the DEJ and later to the outer enamel surface. Maturation is characterized by growth of the crystals seen in the primary phase.

Amelogenesis is unique in many ways. مهمه جدا

1-The secretory cell is an epithelial cell whereas all other secretory cells of hard tissues are ectomesenchymal.

2-Noncollagenous proteins are involved in mineralization of enamel whereas in all other hard tissues collagen plays an important role. The matrix of enamel does not contain collagen; in other hard tissues collagen is the major protein.

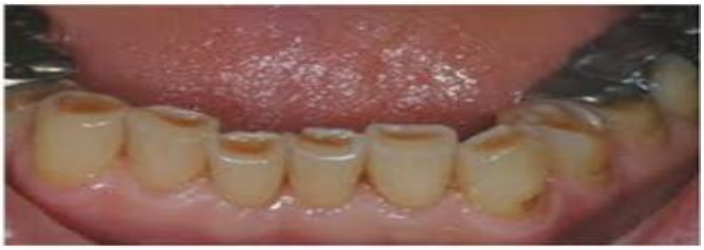
3-The matrix of enamel is partially mineralized; in other hard tissues the matrix is nonmineralized. Enamel therefore lacks a distinct organic phase like osteoid, predentin or cementoid.

4-There is no absorption of secreted matrix in other hard tissues but in enamel formation 90% of secreted matrix is absorbed and this activity is done by ameloblasts itself.

After formation of enamel, ameloblasts undergo apoptosis; hence enamel formation does not occur later on. In other hard tissues formation occurs throughout life.

Age changes:

1-Attrition or wear of the occlusal surfaces and proximal contact points as a result of mastication. This is causing a loss of vertical dimension of the crown and by a flattening of the proximal contour.
Anterior teeth lose their structure more rapidly than do posterior teeth.
2-Localized increases of certain elements such as nitrogen and fluorine, in the superficial enamel layers of older teeth.
This due to continuous uptake, from the oral environment, during aging.
3-The teeth may become darker, and their resistance to decay may be increased.
4-Reduced permeability of older teeth to fluids. The decrease in permeability of enamel due to age is due to increase in the size of the crystal due to ions acquired by it from the oral fluids. The increase in size of the crystal decreases the pores between them causing a reduction in permeability.



FLUORIDTION:

Is incorporation of fluoride ions into the hydroxyapatite crystal which becomes more resistant to acid dissolution (fluroappatite crystals).
This reaction partly affects the reduction of dental caries which is an acid dissolution produced by certain bacteria. systemic hypocalcification of the enamel is the so-called mottled enamel.
A high fluoride content in the water is the cause of the deficiency in calcification. Where the drinking water contains fluoride in excess of 1 .5 parts per million, chronic endemic fluorosis may occur as a result of continuous use throughout the period of amelogenesis for fluorosis
Mottled teeth:are darkly stained teeth due to excessive fluoridation

during tooth formation because of sensitivity of Ameloblasts to fluoride so the amount of fluoride should be controlled during tooth development.

Acid etching:

is an important dental technique used for conditioning the enamel to adhere of fissure sealant, bonding of composite, cementing of orthodontic brackets to the tooth surface. The material used mainly is 37% orthophosphoric acid that acts into 2 steps:

1 -removing dental plaque and other debris from enamel surface.

2-exposing thin layer of enamel with increased porosity through selective dissolution of crystals; which provides a better bonding surface for the restorative and adhesive materials.

Pathologic amelogenesis*:*

If matrix formation is affected by hypoplasia, which is manifested by pitting, furrowing, or even total absence of the enamel, If maturation is lacking or incomplete hypocalcification, in the form of opaque or chalky areas on normally enamel surfaces. The causes of such defective enamel formation can be generally classified as systemic, local, or genetic(hereditary).

1 -The most common systemic influences are nutritional *deficiencies(rickets), endocrinopathies (hypoparathyroidism), febrile diseases, and certain chemical intoxications.*

The discoloration of teeth from administration of tetracyclines during childhood is a very common clinical problem.

2-Local factors affect single teeth, in most cases only one tooth. *The cause of local hypoplasia may be an infection of the pulp with subsequent infection of the periapical tissues of a deciduous tooth if the irritation occurred during the period of enamel formation of its permanent successor. If the injury occurs in the formative stage of enamel development, hypoplasia of the enamel will result.*

An injury during the maturation stage will cause a deficiency in calcification.

The hypocalcified soft enamel matrix is soon discolored, abraded by mastication, or peeled off in layers.

When parts of the soft enamel are lost, the teeth show an irregular, rough

surface.
When the enamel is altogether lost, the teeth are small and brown, and the exposed dentin is extremely sensitive.

3- The hereditary type

of enamel hypoplasia is probably a generalized disturbance of the ameloblasts. Therefore the entire enamel of all the teeth, deciduous as well as permanent is affected. The anomaly is transmitted as a dominant character.
The enamel of such teeth is so thin that it cannot be noticed clinically or in radiographs. The crowns of the teeth of affected family members are yellow-brown, smooth, glossy, and hard.

Functions of IEE cells in tooth development and amelogenesis:

1. Establish the crown morphological pattern during the bell stage.
2. Induce adjacent surface cells of the dental papilla to differentiate into coronal odontoblasts.
3. Differentiate into ameloblasts which secrete enamel.
4. Differentiate into maturation ameloblasts which exhibit cyclic modulation between ruffle-ended and smooth-ended ameloblasts.
5. Form with the other layers of the E. organ the reduced enamel epithelium which protects the enamel surface until the tooth erupts.
6. Contribute to the formation of the *dentogingival junction* of teeth