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Review Article

Embryology and Development: Mandible, Maxillary, Deciduous and Permanent Teeth

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Abstract

Endochondral and membranous (intramembranous) ossification control skeletogenesis. In contrast to endochondral ossification, in which cartilage is replaced by bone, membranous mineralisation involves a highly vascular connective tissue, loaded by proliferating osteoprogenitor cells. The face is formed mostly by intramembranous bones (premaxillary, maxillary, zygomatic, petrous portions of the temporal bone), with the contribution of the frontal, parietal, the squamous portions of the temporal and interoccipital bones. Maxillary and mandible formation, tooth crown and root shaping are related to eruption. Dentin structure is also linked to teeth and bone early stages of development. Growth and transcription factors regulate tooth development, eruption, and resorption. Secretion of 4um/daily contribute to the von Ebner and/or Andresen lines, displaying dentin periodicity.

INTRODUCTION

Except the condylar process, the mandibular bone is intramembranous and its development can be divided into a pre-osteogenic phase, involving mesenchymal condensation, and later at post-condensation stages, it includes many differentiating osteogenic cells [2].

Embryology of the Maxillary

The development of maxillary implies that beneath the frontal process, the two maxillary process are grouped around the oral pit. The two processes appear divided and constricted in the midline, underlining the two maxillary masses.

During the 5th week, the nasal placodes appears on the upper part of the lip. They display two open sites, above the oral pit. The tissues lateral to the nostrils are termed lateral nasal whereas together with the tissue medial to the nostrils, they form what is called the medial nasal process, contributing to form the lips.

At the 6th week, the widened pit become a slit that extend laterally, merge with each maxillary process and the mandibular arch. A ridge of tissue surround each nasal pit. The three parts of the upper lip fuse and unify the lip. Later, the orbicularis oris muscle grows and provide lip support.

At 7th weeks, the nostrils appear more centrally located. The fusion and merging of the tissue masses leads to a recognizable facial form.

The Pediatric Mandible

The bony mandible grows laterally to the first arch cartilage. The body of the mandible and the cartilaginous condyle replace Meckel's cartilage. The condylar unit forms the articulation. The body of the mandible is the center of mandibular growth and the angula is formed in response to the lateral pterigoid and masseter muscles. The coronoid respond to the temporalis muscle development and the alveolar processes form in response to teeth formation and eruption.

The mandible undergoes significant change in its bony structure and the composition of its surrounding soft tissues. Diagram of embryological development of the prenatal mandible is presented in **Figure 1-1**. Bone remodeling occurs in the mandible during childhood **[3,4]**. (Figure: 1-1-2)

Figure 1-1-3 : Overall average distribution of tissue features along the condylar articular surface

(a) Superficial zone : includes a dense fibrous connective tissue and parallel fibrous fibrocartilage

(b) Intermediate zone : distinct layer with :

High cell density

Low cell density

Loose fibrous connective tissue

Dense fibrous connective tissue

Fibrocartilage

(c)Deep zone : Hypertrophic cartilage

Grid –fibrous fibrocartilage

Hyaline-like fibro-cartilage

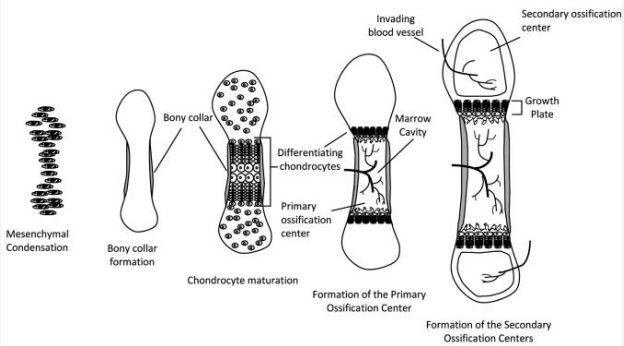

Figure 1 Endochondral ossification of long bone [1].

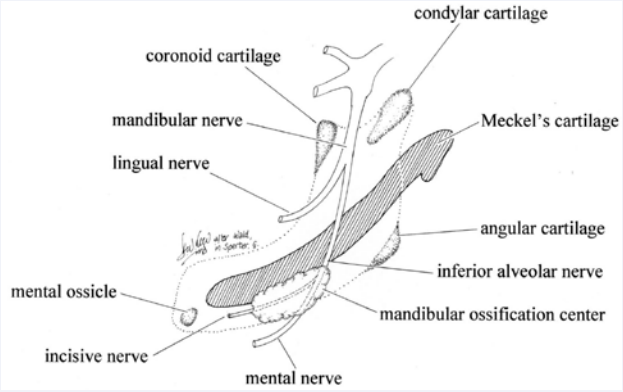

Figure 2 Initial formation of the mandible [5,6].

Calcified cartilage (Mineralized tissues)

(d) Subchondral bone : Compact bone plate

Endochondral ossification (Figure 1-1-3)

The growth of a significant portion of the body and ramus has been attributed to the presence of a growth center implicated in membranous ossification. As the growth plate undergoes cell differentiation and grows in a superior and posterior direction, the mandible is displaced anteriorly and inferiorly **[7]**.

Mandible formation

The mandible, a first pharyngeal arch derivative, originates from neural crest cells that take their position within the mandibular and maxillary prominences during the fourth week after conception. After the formation of the mandibular division of the trigeminal nerve, interactions between the mandibular ectomesenchyme and the mandibular arch epithelium result in the formation of an osteogenic membrane (membranous ossification).

Meckel's cartilage, the initial nonossifying template for early mandibular growth, forms between 41 and 45 days after conception. At the sixth week of life, a single ossification center for each half of the mandible forms lateral to Meckel's cartilage, at the bifurcation of the inferior alveolar nerve and artery into its mental and incisive branches. From this center, ossification proceeds ventrally to the body and dorsally, contributing to the mandibular ramus.

Secondary cartilages form that will eventually give rise to the coronoid process, mental protuberance, and condylar head. The secondary cartilage of the coronoid process gives rise to additional intermembranous bone. The mental protuberance form ossicles in the fibrous tissue of the symphysis The condylar secondary cartilage is the primitive form of the future condyle, providing the cartilaginous material that provide the stimulus for endochondral ossification of the condylar neck. During this time the condylar cartilage takes on a stratified organization with five principal layers:

a) Articular cartilage,

b) Chondroprogenitor cells,

c) Chondroblasts,

d) Nonmineralized hypertrophic chondrocytes,

e) Mineralized hypertrophic chondrocytes.

Dorsally, at the temporomandibular joint, portions of the fibrous perichondrium associated with Meckel's cartilage transform into the sphenomandibular and sphenomalleolar ligaments [7]. The cartilage reveals a strongly positive reactivity for type IX collagen. A strong reactivity for type VI collagen was visible along the bone trabeculae that underneath the carilage portion of the condylar process [8] (Figures 1,2).

Tooth formation

Craniofacial morphogenesis implicates cell adhesion molecules (CAMs), and substrate adhesion molecules (SAMs) that function in cell-cell and cell-substrate interactions (laminin, fibronectin, tenascin, heparin sulfate proteoglycans, and collagens). Structural genes (collagens, dentin phosphorylated proteins, enamel proteins) amplify or reduce (acting as promoters or enhancers) the series of bioactive molecules. These molecules influence cell division, cytological specialization, and/or the shape of the teeth following epithelial-mesenchymal interactions. Several enamel-specific gene products have been identified (amelogenins and enamelins).

From dental placode to erupting tooth, a series of events are observed. At each stage, mutations of growth factors and transcription factors are expressed. They affect tooth matrix deposition and root formation [9,10].

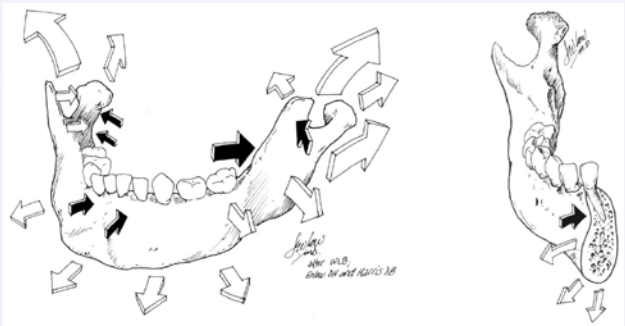

Figure 3 Overall average distribution of tissue features along the condylar articular surface.

The first sign of mammalian tooth development starts by the thickening of the stomodeal oral ectoderm, forming a primary epithelial band. The free margins of this band gives rise to the outer process that demarcate the cheeks and lips, and the inner process (or dental lamina) implicated in the formation of tooth buds. Interactions between the oral epithelium and the mesenchyme. The epithelial cells multiply and form a lamina growing within the subjacent dental mesenchyme (dipping wall).

Dental placodes initiate process of tooth development. It starts by the specification of dental placodes (early thickening or protruding wall). Morphogenesis is followed by a bud formation, becoming a cap and later, a bell. Theses morphogenetic events occurring in the condensed dental mesenchyme are controlled by signaling center. Spatio-temporal induction of the secondary enamel knots determines the cusp patterns of individual teeth and likely involve repeated activation and inhibition of signaling [11]. Matrix secretion contribute to the formation of the crown, followed after eruption by the development of the roots [12].

The epithelial dental lamina includes à proliferating inner enamel-forming layer, where pre-secretory ameloblasts differentiate. This epithelial digit-like protrusions growth and form a lamina in continuity with the oral epithelium. The formation of the plunging wall connected to the dental blade is induced by ecto-mesenchimatous cells. Then, the differentiation of the stellate reticulum and stellate intermedium is occurring. They are involved in the transport from cell-cell and diffusions through the intercellular spaces. Enamel gelly is transfered through the extra (inter)cellular matrix. Together with the outer tooth germ epithelium and the inner epithelial layer, they contribute to the initial pattern of events [13].

Either the formation appears as a continuous gradient or successive well defined areas (placodes) that will be implicated in different teeth formation and determination of tooth region (Figure 1-3). The tooth germ consists of the enamel organ, derived from ectoderm, and the dental papilla, derived from the neural crests. BMP2 and BMP4 inhibit Pax9 expression in the mouse mesenchyme of the branchial arch. This antagonistic signaling determines the sites of tooth buds initial development. In the case of mammalian dentition, four types of teeth are usually considered to form: incisor, canine, premolar, and molar teeth within each dental lamina (Figure 1-5). (Figure:1-1-4)

Signals in the four families, BMP, FGF, Shh and Wnt, have been analyzed. They have distinct molecular actions on the mesenchyme. The signals in each family have been shown to be integrated at different levels in developing teeth. They activate the same transcriptional targets (Figure 1-1-4).

The requirement of different genes in the signaling networks are stage-dependent. Analysis of gene expression and signal responses has elucidated the signaling pathways. Taken together, the requirement of different genes in the mesenchyme changes with the advancing development, emphasizing the sequential nature of the reciprocal interactions and progressive determination and differentiation of the tissues.

Regulation of Tooth shape

Morphogenetically, the FGF signaling combined with areas of non-dividing epithelial cells surrounded by areas of strongly proliferative epithelia, may play a central role in the folding of dental epithelia. No distinct pattern in cell proliferation in the dental mesenchyme has been observed suggesting that intricate temporospatial control of epithelial proliferation and differentiation is required for tooth morphogenesis. (Figure : 1-1-5).

A possible autocrine functional role for TGF-β and its cognate receptor (TGF-β IIR) was due to the temporal and spatial localization patterns during the early inductive stages of tooth morphogenesis [15].

Root formation: Its regulation by biological mechanisms

In coronal predentin, collagen librils are thicker, more densely packed and often arranged parallel to odontoblast processes. Odontoblast processes retreat with the cell bodies move away from the basal lamina. In the crown, the peripheral dentin contains highly branched dentinal tubules whereas in the root dentin is atubular. After a certain amount of root dentin has been deposited, the tubules are formed. A process appears to occur rapidly, resulting firstly in the formation of the granular layé of Tomes. Coronal odontoblasts are columnar, whereas root odontoblasts are cuboidal [16]. Root extension rate in humans was 20 m to 9 m per day with an initial root growth rate in length 5µm per day.

Tooth Eruption

Four theories on the mechanisms of tooth eruption have been elaborated implaying : 1) root elongation, 2) collagen maturation within the alveolar ligament (reduction in length of the tropocollagen becoming mature collagen, 3) the action of metalloproteinase influencing collagen maturation, 3) alveolar bone remodeling (osteogensis in the apical part and osteoclastic destruction of the alveolar bone along the lateral walls of the crypt, 4) blood pressure within the dental pulp. Signals generated by the dental follicle itself may contribute to tooth eruption [17] (Figure 1-3).

Because roots form at the time of eruption. They have long been considered as the force responsible for eruption. However rootless teeth do erupt, and this invalidates this hypothesis. Alveolar bone growth, tooth development, and eruption are interdependent. Formation of bone apical to developing teeth

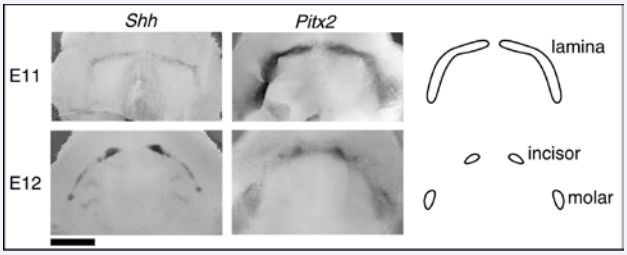

Figure 4 Members of the transforming growth factor (TGF) (BMP4), fibroblast growth factors (Fgf 8 & 9), sonic hedgehog (Shh), Dlx2, Barx1, and Wnt signaling pathways are required during tooth development [14].

has long been proposed as one mechanism for eruption. The fact that active eruption begins only after crown formation suggests a role for the enamel organ and its proteases in the early signaling of eruption.

Formation and renewal of the periodontal ligament is associated with the continuous eruption of permanently growing rodent incisors. However, in case of osteopetrotic mutations a periodontal ligament is present, but teeth do not erupt, and this is also the case of rootless teeth.

Examination of the bony crypt of an erupting tooth by scanning electron microscopy shows that there is resorption in its coronal aspect and formation of alveolar bone in its apical aspect. Light and electron microscopic studies of the follicle and crypt during eruption show that there are osteoclasts and osteoblasts on opposite sides of the erupting tooth, and that the coronal part of the follicle is infiltrated by mononuclear cells a few days before the begining of eruption. These mononuclear cells may be considered to be preosteoclasts. They fuse with osteoclasts on crypt surfaces. The apical parts of the dental follicle and crypt are characterized by cell proliferation.

The most prominent molecule is a sialoprotein. At the onset of eruption, fragmentation of the sialoprotein is a biochemical marker of the beginning of tooth eruption. The follicular content of the metalloproteinases (collagenases and stromelysin) is reduced during eruption. Activation of these proteases at the completion of crown formation initiates the release of metalloproteinases from the dental follicle. Experiments with colony-stimulating factor-1 (CSF-1) showed that this factor accelerates molar eruption by increasing monocytes in the follicle and osteoclasts on crypt surfaces. Its effect on tooth eruption depends upon when CSF-1 administration begins. Studies of molar eruption show that

a) Localized bone formation and resorption are major events in eruption,

b) That root formation is a consequence but not a cause of eruption, and

c) That the availability of an eruption pathway is a requirement.

Single-rooted anterior teeth erupt along the path of the gubernacular canal, the small channel that connects the bony crypt surrounding the tooth to the oral surface of the alveolus. Eruption pathway formation requires bone resorption which is regulated by the dental follicle.

The key to the successful clinical management of tooth eruption consists of understanding that this process consists largely of the local regulation of alveolar bone metabolism to produce bony resorption in the direction of eruption and formation of bone at the opposite apical site. CSF-1, a series of growth factors, root formation, bone formation, osteoclasie in area of the gubernaculum dentis, contribute to the eruption of premanent and deciduous teeth. (Figure: 1-1-6)

DENTIN STRUCTURE

Dentin includes

(a) Peripheral layers (in the crown : the mantle dentin (80-100 µm in thickness), and in the root: the hyaline and granular Tomes layers (30 µm each) constitute the outer limits of the dental pulp. Interglobular spaces, resulting from the lack of fusion of calcospherites, are filled with proteoglycans. They accumulate at this location, in this specific outer layer, and they are not seen elsewhere in circumpulpal dentin, except in the pathologic hypophosphatemic tooth.

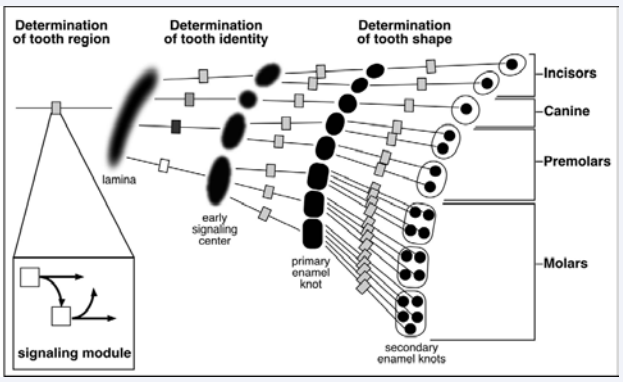

Figure 5 Determination of the tooth shape [14].

(b) Circumpulpal dentins are composed by intertubular and peritubular dentin that are primary and secondary dentins (physiological dentins), formed between the begginig of tooth creation and the eruption of the primary and secondary erupted tooth. Tertiary dentin results from pathological events, namely the carious or abraded teeth. Reparative or reactionary dentins provide answers to the carious diseases. In case of abrasion and/or non-infectious [18]. Expression and function of FGFs-4, -8, and -9 suggest functional redudancy and repetitive use as epithelial signals during tooth morphogenesis.

© With non-mineralized and unfused interglobular spaces.

1) Intertubular dentin is a collagen-rich structure (Type I collagen), whereas peritubular dentin is deprived of collagen fibres. Collagen fibrils are located inside the lumens of dentin tubules. Intertubular dentin display needle-like (600Å in length and 10 Å in thickness) structures, forming hydroxyapatite-like crystals. In the peritubular dentin isodiameter rhombohedric cristals (a=b=25 nm, and c= 9-10 nm) are tightly grouped, forming an homogenous ring in transverse section or a densely packed mineralized tube in longitudinal sections.

2) In dentin, the peritubular dentin thickness varied between 1.0 to 2.5 µm. The number of tubules changed according to the portion of dentin examined (outer or inner zones). The tubule numerical density decreases from the outer dentin toward the inner part of the pulp (11,800mm^2/mm to 4.400 mm^2/mm). The tubule diameters increased from 0.28 µm/mm and 0.39 µm/mm, with a corresponding reduction of the peritubular width that decreased from 0.75 µm near the dento-enamel junction to 0.62 µm toward the pulp. This evaluation contrast with the values found by [20]. Expression and function of FGFs-4, -8, and -9 suggest functional redudancy and repetitive use as epithelial signals during tooth morphogenesis. Expression and function of FGFs-4, -8, and -9 suggest functional redudancy and repetitive use as epithelial signals during tooth morphogenesis. The number of

tubules/mm^2 was about 23,760 (in the deep layer) and displays weak difference with the outer part (18,781mm^2).

Dentin formation cycles of various lengths are representative for biological activities. They form incremental lines, with a variation between very short (ultradian) rhythms (6-8 µm/day) to rhythms with a period of approximately one day (circadian) (16-24 µm/day, reported in the circumpulpal dentin). The average extension rate was 3.8 µm/day. Rhythms with longer cycles of one week, month, season, or even longer, are also supporting the evidence of incremental lines. Von Ebner lines (14 increments, are separated by a regular series of lines measuring a total appoximatively of 18 to 20µm, and sublines, each of 4µm indicative of rythmic changes, or incremental lines. This was also reported as long-period Andresen lines (determined as a 9 days periodicity). They are visible in dentin, and constitute the manifestation of a daily rythm of dentin deposition. Long ago, Owen has reported the lines of contour, which are related with the von Ebner lines detected in enamel and dentin. In the root, periradicular bands are present, at least at the surface of cementum [21] (Figures 6-10).

The composition of dentins extracellular matrix includes type I collagen (90%), among which 11% is of the type I collagen trimer, type III and V collagens), dentin phophoprotein (DPP) cleaved into dentin sialophosphoprotein (DSPP), dentin matrix protein-1 (DMP-1), osteopontin [bone sialoprotein-I (BSP I)], osteocalcin, osteonectine (also named SPARC protein), bone sialoprotein–II (BSP II), proteoglycans (among which CS PGs (biglycan, decorin, lumican, fibromodulin), serum protein (α_2HS-glycoprotein and albumin), growth factors, transcription factors, enamel protein, proteinases (MMPs, Timps, Cathepsin), membrane and extracellular lipids [21] (Figures 4-5).

Dental root resorption

Resorption is based on a classification of internal, cervical, and external resorption associated with periradicular pathosis and resulting from pressure in the periodontal ligament [22]. Osteoclasts or cells erosions called Howship's lacunae, depressed the bone surface. They have ruffled bordé delineated by a clear zone. The dentin-resorbing cells (dentinoclasts) have fewer nuclei and they are smaller than osteoclasts. Dentinoclasts have small or no clear zones in contrast to the well-developed clear zones of actively resorbing osteoclasts. Phagocytosis is carried out by elements of the mononuclear phagocyte system, consisting of neutrophils and mononuclear phagocytes. Osteoclasts are also described as participants arrising as pro-monocytes, entrering in the blood stream as monocytes and becoming macrophages. Macrophages contain cytoplasmic granules containing hydrolases.

Cervical resorption has been designated as invasive cervical resorption, or internal-external resorption because of their pattern of spreading within the root.

Primary (deciduous) vs. Permanent teeth

The **primary enamel** structure showed a lower level of Ca and P, thinner thickness and higher numerical density of rods when compared to permanent teeth. Incremental microstructures are seen **in permanent enamel** as (1) cross-striations and (2) Retzius lines and their surface manifestation, reported as perikymata. Enamel daily secretion rate (DSR) differs with crown position, increasing from the inner to the outer enamel, and from cervical to cuspal regions, and ranging from approximately 2 to 7 µm/day. Enamel daily secretion rate differs with the crown position, increasing from the inner to outer enamel and from cervical to cuspal regions.

The number of tubules/mm^2 (tubule density) of **primary dentin** in deciduous teeth was lower that in permanent teeth. Variations in the tubule diameter appears to be greater in primary dentin than in permanent dentin (1.6µm vs. 0.8µm). Peritubular width ranged from 1.0 µm to 2.0 µm in molar and premolar teeth, and it was also reported that it decreased from 0.75 µm near the DEJ toward the pulp, whereas the thicknest varied between 0.92 µm and 0.62 µm in width. In primary teeth dentin 'S'-shaped curvature was about 26.7% and a straight course about 73,3%, whereas in permanent teeth all specimens showed an 'S'-shaped curvature (Figures 6,7).

In dentin

Short- and long-period features of dentine microstructure are known as von Ebner's lines or Andresen lines, which have been shown to correspond to cross-striations and Retzius lines in enamel, respectively (seprated by approximatively 20µm). Several developmental variables using short- and long- period

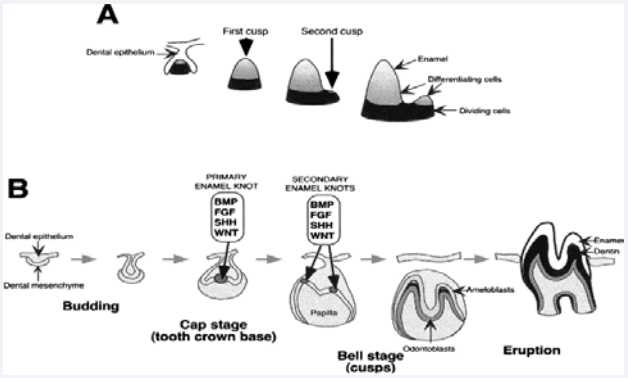

Figure 6 Molecular signals considered to be important for teeth morphogenesis [18, 19].

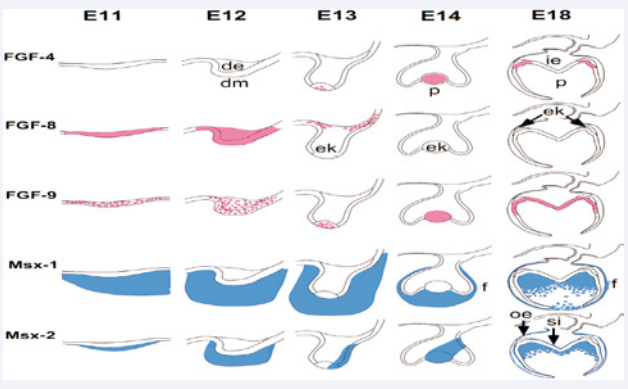

Figure 7 Expression of FGF -4, -8 and -9, Msx-1 and Msx-2 in the developing mouse mandibular first molar[19].

incremental features:

a) Daily secretion rate (4µm /daily rythmic secretion);

b) Periodicity of long-period lines (number of short period increments between successive long-period lines);

c) Number and distribution of long-period lines (or their external manifestation as perikymata/periradicular bands); and

d) Extension rate of crown and/or root growth. Incremental lines in human dentin (von Ebner) have a circadian component. It was suggested that there is a rhythmic and recurrent deposition of dentin matrix by odontoblasts in a 12h-hour rythmic change in the orientation of the matrix components [23] (Figures 7-10).

Dentin growth factors, transcription factors and stem cells

During mammalian tooth development a coordination of growth and differentiation factors are instrumental. Sonic hegehog (Shh) encodes a signaling peptide

(a) Bmps: Prominent signaling molecules are the **Bone Morphogenetic Proteins** (BMPs), **Fibroblast Growth Factors** (FGFs), **Wnt**, and **Hedgehog** (Hh) families. These growth factors function synergistically and/or antagonistically. They contribute to organize and pattern tissues and organs during embryonic development. **BMP4** play a central role during tooth morphogenesis. The type II receptor phosphorylates the type I receptor which in turn phosphorylates the **DNA-binding proteins Smads**. There are eight vertebrate Smad proteins (**Smad 1-8**) divided into three distinct classes:

i. the receptor-activated Smad (Smad1, Smad2, Smad3, Smad5, and Smad8),

ii. the common-mediated Smad (Smad4),

iii. inhibitory Smad (Smad6, Smad7). Among them, Smad1, Smad5 and Smad8 are phosphorylated by BMP type I receptors.

(b)Fgfs and Shh : FGF8 is also primarily responsible for **Lhx6** and **Lhx7** expression in the odontogenic mesenchyme. At the bud and early cap stage, **FGF 9** is up-regulated in the primary enamel knot, where **FGF4** is activated by the Wnt signaling pathway [19]. Expression and function of FGFs-4, -8, and -9 suggest functional redudancy and repetitive use as epithelial signals during tooth morphogenesis.

©Wnts: Wnt gene family represents a large and diverse group of signaling molecules involved in the patterning, proliferation and differentiation of a variety of cell types. They signal through the Frizzled family of receptors. A second type of Wnt receptor is related to the low-density lipoprotein (LDL) receptor and known as **LRP5/6**. **Dickkopf (Dkk)** antagonize Wnt signaling. **Wnt5a, sFrp2,** and **sFrp3** are expressed only in the dental mesenchyme.

(d) Transcription factors : A number of homeobox genes such are expressed with a specific spatial pattern in the first branchial arch. The expression of *Msx1* and *Msx2* is detected in the developing tooth germ in patterns that correlate with morphogenetic steps in tooth development. Pax9 is initially expressed in the presumptive dental mesenchyme. Lef1 is initially expressed in the thickened dental epithelium. At the bud, cap and bell stages, Lef1 transcripts are detected both in the dental mesenchyme and in the immediately adjacent dental epithelium.

CONCLUSIONS

Endochondral and membranous ossification control skeletogenesis. The pediatric mandible implicates the formation of the body of the mandible and the condylar articulation. From the dental placode to erupting tooth growth and transcription factors are expressed. The thickening of the stomodeal oral ectoderm initiate the development of dental placodes. Cap, followed by bell formation contribute to shape of the crown, and the determination of the tooth (incisors, canine, premolars and molars). The teeth includes peripheral layers (mantle dentin, the hyaline and granular layers) and circumpulpal dentin that are parts of primary and secondary dentin. Secretion of 4µm/daily contribute to the von Ebner and/or Andresen lines. BmpS, FGFs, Shh, the hedgehog family, Wnts signaling, Msx-1 and Msx-2 are expressed in the formation of primary and secondary teeth that are influenced by these parameters.

REFERENCES

1. Boskey AL, Coleman R. Aging and bone. J Dent Res. 2010; 89: 1333-1348.

2. Svandova E, Vesela B, Bobek J, Lesot H, Matalova E. Osteogenic and angiogenic profiles of mandibual bone-forming cells. Frontiers in Physiol. 2019.

3. Sicher H. Orban's Oral histology and Embryology Development of the face and oral cavity Mosby Co Saint Louis. 1966. Chapter 1. 1-17.

4. Avery JK, Steele PF. Essential of oral histology and embryology-A clinical approach Mosby –Year book 1992 Chapters 3 & 4. Development of the oral Facial region- Development of the face and Palate 27-50.

5. Luder HU, Leblond C-P, Von der Mark K. Cellular stages in cartilage formation as revealed by morphometry, radio- autography and type II collagen immunostaining of the mandibular condyle from weanling rats. Am J Anat. 1988; 182: 197-214.

6. Marchi F, Luder HU, Leblond C-P. Changes in cells' secretory organelles and extracellular matrix during endochondral ossification in the mandibular condyle of the growing rat. Am J Anat. 1991; 190: 41-73.

7. Cahill DR. Histological changes in the bony crypt and gubernacular canal of erupting permanent premolars during deciduous premolar exfoliation in beagles. J Dent Res. 1974; 53: 786-791.

8. Silbermann M, Von der Mark K. An immunohistochemical study of the distribution of matrical proteins in the mandibular condyle of neonatal mice. I Collagens J Anat. 1990; 170: 11-22.

9. Bei M. Molecular genetics of tooth development. Curr Opin Genet Dev. 2009; 19: 504-510.

10. Thesleff I. Current understanding of the process of tooth formation : transfer from the laboratory to the clinic. Australian Dental J. 2014; 59: 48-64.

11. Jernvall J, Thesleff I. Reiterative signaling and patterning during mammalian tooth morphogenesis. Mech Dev. 2000; 92: 19-29.

12. Tucker A, Sharpe P. The cutting-edge of mammalian development : how the embryo makes teeth . Nature Reviews /Genetics. 2004; 5: 499-508.

13. Lumsden AGS. Spatial organization of the epithelium and the role of neural crest cells in the initiation of the mammalian tooth germ. Development. 1988 ; 155-169.

14. Jernvall J, Thesleff I. Tooth shape formation and tooth renewal : evolcing with the same signals. Development. 2012; 139: 3487-3497.

15. Chai Y, Zhao J, Mogharei A, Xu B, Bringas P Jr, Shuler C, et al. Inhibition of transforming growth factor -β type II receptor signaling accelerates tooth formation in mousee first branchial arch explants. Mech Dev. 1990; 86: 63-74.

16. Thomas HF. Root formation. Int J Dev Biol. 1995; 39: 231-237.

17. Marks SC, Schroeder HE. Tooth eruption: theories and facts. Anat Rec. 1996; 245: 374-393.

18. Jernvall J, Jung H-S. Genotype, phenotype, and developmental biology of molar tooth characters. Yearbook of physical anthropology. 2000; 43: 171-190.

19. Kettunen P, Thesleff I. Expression and function of FGFs-4, -8, and -9 suggest functional redudancy and repetitive use as epithelial signals during tooth morphogenesis. Dev Dyn. 1998; 211: 256-268.

20. Schilke R, Lisson JA, Brauss O, Geurtsen W. Comparison of the number and diameter of dentinal tubules in human and bovine dentine by scanning electron microscopic investigation. Archs Oral Biol. 2000; 45: 355-361.

21. Goldberg M, Dentines. in Piette et Goldberg (eds) La dent normale et pathologique. De Boeck Université, Bruxelles. Chapter 4. 2001.

22. Gunraj MN. Dental root resorption. Oral Surgery, oral medicine, oral pathology. 1999; 88: 647-653.

23. Kawasaki K, Tanaka S, Ishikawa T. On the daily increment lines in human dentine. Archs oral Biol. 1979; 24: 939-943.

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