Review

Theories of tooth eruption: An update

Vanishree H Shivakumar, Anand S Tegginamani

Faculty of Dentistry, SEGi University, Kota Damansara

Abstract

The process for tooth eruption has been a matter of debate. Numerous theories of tooth eruption have been proposed. The understanding of recent advancements regarding the tooth eruption is needed as several types of cells and molecular factors that are believed to be responsible for the tooth eruption. This article reviews previously proposed eruption theories and the recent advances in the eruption mechanism.

Keywords: bone remodeling, dental follicle, tooth eruption

Corresponding Author

Vanishree H Shivakumar

Faculty of Dentistry, SEGi University, Kota Damansara

Email: vanishreeshivakumar@segi.edu.my

Introduction:

Tooth eruption process has never been fully understood. But why is it be only hypothesized? And what forces push the teeth through the soft tissues? Each theory for eruption presents with a problem.[1, 2] The biological processes responsible for tooth eruption have long been a matter of debate. According to Massler and Schour, it can be defined as the movement of a tooth from its site of development within the jaws to its position of function within the oral cavity.[5] The eruption is necessary for the survival

of diverse species and is a continuous process. Which does not stop by reaching the occlusal plane rather continues throughout life. The scientific literature on mechanism of eruption in the field is extremely dispersed. Upon intensive research over the last 30 years, the specific mechanisms responsible for tooth eruption, are not understood. Numerous theories of tooth eruption have been proposed involving almost all tissues in or surrounding an erupting tooth. But none of them alone could review the focuses on human and other mammalian teeth eruption and also analyzes recent observations and experimental data. [3-5]

Movements of tooth are generally divided into three phases as: a) pre-eruptive b) eruptive c) Post-eruptive phases.

Many theories of eruptions were proposed [5, 6]

I] Theories that were not be considered as serious contenders like

- Pulpal pressure
- Pulpal growth, vascular pressure
- Blood-vessel thrust
- Traction by periodontal fibroblasts.

II] Theories that were considered as serious contenders:

- alveolar bone remodeling
- root elongation/periodontal ligament formation
- dental follicle theory

Theories of tooth eruption

A] Cushioned hammock theory:

It was proposed by Harry Sicher in 1942. This theory assumed that cushioned hammock ligament below the tooth is responsible for the eruption of the tooth but was not accepted, according to

Edward HF; it was an artifact in slide preparation, not a ligament.[6]

B] Bone remodeling theory:

Alveolar bone growth, tooth development, and eruption are interdependent. Formation of bone apical to developing teeth has long been proposed as one of the mechanisms for eruption, its programmed bone remodeling & the dental follicle is involved. The mutation of the parathyroid hormone receptor 1 gene is correlated with disturbances in bone remodeling and which leads to primary failure of tooth eruption. This primary failure of eruption is a non-syndromic eruption disturbance and not associated with defective osteoclasts which supports bone remodeling theory. [7]

C] Dental follicle theory:

The theory described as a factor decisive for the eruption process & is capable of inducing, bone resorption above the developing crown and bone apposition & enables the formation of an eruptive pathway. The molecular studies have shown that the eruption is regulated by inductive signals between the dental follicle, stellate reticulum, reduced enamel epithelium, and alveolar bone RANKL gene is a marker gene for bone resorption that shows higher expression in the coronal half of the dental follicle.[6] Bone morphogenetic protein-2 (BMP-2) gene is a marker for bone formation and higher expression in the basal half of the follicle. Eruption is a localized, & a bilaterally symmetrical event in an alveolar bone which is regulated by the dental follicle, a derivative of cranial ectomesenchyme. Recent developments, concerning the paracrine signaling function of the dental follicle in tooth eruption, interactions between the dental follicle, the Reduced enamel epithelium (REE) & stellate reticulum dental follicle recruits of monocytes, followed by bone resorption these apoptotic process have an influence on osteoclastogenesis as stellate reticulum cells releases the interleukin-1α.[6]

This interleukin-1α stimulates the expression of CSF-1 and monocyte chemotactic protein-1. Recently Nel S in 2015 proposed that tooth eruption should be regarded as a stages of tooth development.[4]

D] Periodontal ligament traction theory:

The most frequently cited are the root growth and pulpal pressure, other important causes are cell proliferation, increased vascularity, and increased bone formation around the teeth, and other possible causative agents include endocrine influence, vascular changes, and enzymatic degradation. Probably all these factors have an influencing role.[3] Although all the factors which are associated with tooth eruption are not yet known, elongation of the root and modification of the alveolar bone and periodontal ligament are thought to be the most important factors. These events are coupled with the changes overlying the tooth that produce the eruption pathway. Fibroblasts have traction power and they move incisally along the erupting tooth. The contraction of fibroblast generates significant force for the tooth eruption. Occlusal migration of these periodontal ligament fibroblasts has been said as main factors responsible for tooth eruption, possibly that the periodontal ligament (PDL) could play a role in the supra-osseous phase of the eruptive process in lifting the tooth into the occlusal plane.[4] Collagen synthesis, remodeling and the cells implicated in these processes can however not accepted as the only mediators of tooth eruption as a tooth without a periodontal ligament can still erupt.[1,6,9]

E] Innervation-provoked pressure theory:

According to this theory that tooth eruption depends on the space in the pathway of eruption, Pressure from below & the adaptation of the periodontal membrane. It assumes the existence that the root membrane acts as a glandular membrane. So, the innervation in this membrane causes pressure in the apical part of the tooth which results in tooth eruption.[10-13]

F] The equilibrium theory:

The theory postulates that once the functional plane is reached, the eruption of the tooth is balanced in response to the vertical growth of the mandible is controlled by forces impeding the eruption, as opposed to encouraging forces. These steady forces of masticatory function and the soft tissue pressures from the lips, cheeks, and tongue are the limiting factors of post functional occlusal eruption. The eruptive movement that occurs while the teeth are free of contact, supports the idea that eruptive control is based on the continuous force of the surrounding soft tissues.[6, 7, 13]

G] Neuromuscular theory or unification theory:

This theory of tooth eruption states that the synchronized forces of the orofacial muscles, under the control of the central nervous system (CNS) are responsible for the process. The molecular events prepared a pathway under the control of these forces by the coordinated neuromuscular forces & they are converted into electrical, electrochemical and biomechanical energies for the stimulation of cellular and molecular activities within and around the dental follicle. [5, 6, 14-20]

The probable mechanism for eruption of tooth may be: [22-24]

(i) Due to periodontal ligament or its precursor of the dental follicle, or a combination of fibroblast activity and vascular or tissue hydrostatic pressure.

(ii) It is multifactorial, involving the molecular and enzymatic activities.

Conclusion or confusion!

Tooth eruption is a biological process, which is still not fully understood, there is no theory to explain the generation of eruptive force which supported by sufficient experimental evidence, Many studies & systematic reviews were carried on animal tissues from its development in the bony crypt to its eruption till the occlusal level. Each of the eruption theories has a say to some portion of the eruption process and does not mention the etiology behind the eruption, some only state that it is unknown.

References

1. Avery J. K. Steele P. F, Essentials of Oral Histology and Embryology, Mosby Year Book, 1992.
2. Bath-Balog M. Fehrenbach M. J, Dental Embryology, Histology, and Anatomy, Elsevier Saunders, 2006.
3. Kjær I, Mechanism of Human Tooth Eruption: review article including a new theory for future studies on the eruption process Scientifica 2014, Article ID 341905, http://dx.doi.org/10.1155/2014/341905
4. Nel S, Hendrik HD, Boy SC, Raubenheimer EJ. Recent perspectives vis-à-vis the biological basis of tooth eruption SADJ 2015; 70 (6): 238 – 241.
5. Marks Jr S. C. Schroeder H. E, "Tooth eruption: theories and facts," The Anatomical Record, 1996; 245 (2):374–393.
6. Rabea A A. Recent advances in understanding theories of eruption Future Dental Journal 2018;4: 189–196.
7. Frazier-Bowers SA, Hendricks HM. Failure of tooth eruption: diagnosis and management. In: Wright JT. Craniofacial and dental developmental defects: diagnosis and management. Springer; 2015.
8. Becktor KB, Nolting D, Becktor JP, Kjaer I. Immunohistochemical localization of epithelial rests of Malassez in human periodontal membrane. Eur J Orthod 2007;29(4):350–3.

9. Brin I, Zilberman Y, Galili D, Fuks A. Eruption of rootless teeth in congenital renal disease. Oral Surg Oral Med Oral Pathol 1985; 60:61–4.
10. Kjær I, Kocsis G, Nodal M, Christensen LR. Aetiological aspects of mandibular tooth agenesis-focusing on the role of nerve, oral mucosa, and supporting tissues. Eur J Orthod1994; 16(5):371–5.
11. Becktor KB, Hansen BF, Nolting D, Kjaer I. Spatiotemporal expression of NGFR during pre natal human tooth development. Orthod Craniofac Res 2002; 5(2):85–9.
12. Bille ML, Thomsen B, Kjær I. Apoptosis in the human periodontal membrane evaluated in primary and permanent teeth. Acta Odontol Scand 2011; 69(6):385–8.
13. Proffit WR. Contemporary orthodontics. Fifth ed. Elsevier; 2013.
14. Bille ML, Thomsen B, Kjær I. Apoptosis in the human periodontal membrane evaluated in primary and permanent teeth. Acta Odontol Scand 2011; 69(6):385–8.
15. Loto AO. Tooth eruption: a neuromuscular theory, part one. J. Craniomax. Res. 2017; 4(1):278–83.
16. Wise GE, King GJ. Tooth movement. J Dent Res 2008; 87:414–34.
17. Ruta A, Irena B, Janina T. Factor's influencing permanent teeth eruption. Part one general factors. Stomatologija. Baltic Dental and Maxillofacial Journal 2010; 12:67–72.
18. Ash MM, Nelson SJ. Wheeler's dental anatomy, physiology, and occlusion. Ninth ed. Elsevier; 2003.
19. Kiliaridis S. The importance of masticatory muscle function in dentofacial growth. Semin Orthod 2006; 12 (2):110–9.
20. Wise GE. Cellular and molecular basis of tooth eruption. Orthod Craniofac Res 2009; 12 (2):67–73.
21. Koch G. and Poulsen S., Pediatric Dentistry. A Clinical Approach, Wiley-Blackwell, page 198.2009.

22. Bath-Balog M. and M. J. Fehrenbach, Dental Embryology, Histology, and Anatomy, Elsevier Saunders, 2006
23. Berkowitz B. K. B., Holland G. R., and. Moxham B. J, Oral Anatomy, Histology and Embryology, Mosby, Elsevier, 2009.
24. Kjær I. Mechanism of human tooth eruption: review article including a new theory for future studies on the eruption process. Sci Tech Rep 2014; 2014:1–13.