**DEMINERALIZATION AND REMINERALIZATION IN TOOTH DECAY**

**Kavitha.Ml and Shreesha.M2**

l Associate Professor, 2, M.Sc. Biochemistry Student

Department of Biochemistry, Dwaraka Doss Goverdhan Doss Vaishnav College, Arumbakkam, Chennai-106.

**1. INTRODUCTION**

Dental health plays a crucial role in overall well-being, with dental caries (also called as tooth decay) being one of the most common chronic conditions affecting individuals of all ages. It develops when acids produced by bacteria in the mouth gradually break down the tooth structure. This process involves a continuous cycle of mineral loss (demineralization) and repair (remineralization) [1]. If the balance tips toward mineral loss, cavities can form. Understanding these processes and the factors that influence them is essential for preventing and managing tooth decay effectively. This chapter provides an overview of the biological mechanisms, contributing factors, and modern approaches to maintaining tooth integrity through prevention and early intervention.

**2. DENTAL CARIES**

Dental caries is a chronic, multifactorial, and biofilm-mediated disease that results in the destruction of tooth hard tissues due to prolonged acidic challenges in the oral environment. Caries development is not solely a result of poor oral hygiene but rather a complex interplay between microbial communities, host susceptibility, diet, salivary function, and time [2].

** Figure:1 Dental caries**

The etiology of dental caries is primarily microbial. Specific bacteria, including Streptococcus mutans, Streptococcus sobrinus, Lactobacillus spp., and Actinomyces, are highly acidogenic and aciduric. These organisms metabolize fermentable carbohydrates, such as sucrose, glucose, and fructose, producing lactic and acetic acids as metabolic byproducts. These acids lead to a localized decrease in plaque pH, favoring the demineralization of the tooth surface, particularly in stagnation areas like pits, fissures, and proximal surfaces [3]. Over time, repeated demineralization events , if not reversed by remineralization mechanisms, cause structural breakdown of enamel and eventual cavitation.

**3. STRUCTURE AND COMPOSITION OF TEETH**

Both soft and mineralized tissues make up the intricate biological structure that is the human tooth, and each is essential to its integrity and functionality. The four main layers of it are pulp, cementum, dentin, and enamel.

**Enamel** is the hardest substance in the human body and forms its outermost protective layer. Composed of approximately 96% inorganic components mainly hydroxyapatite crystals along with 1–2% organic matter and 2–3% water, enamel provides exceptional resistance to both physical wear and chemical erosion. However, due to its acellular and non-regenerative nature, it cannot repair itself, making early prevention of damage essential [4].

**Dentin** is the primary structural element of a tooth, located just beneath the enamel subcaste. It's made up of around 70 minerals, 20 organic material primarily type I collagen and 10 water. Within dentin are multitudinous bitsy channels called tubules, which serve to carry sensitive signals toward the pulp. Compared to enamel, dentin has a lower degree of mineralization, making it more vulnerable to demineralizing processes [5].

**Cementum** is the thin layer that envelops the tooth root and secures it to the periodontal ligament . With roughly 45–50% inorganic material and the remaining organic matter and water, it is composed similarly to bone. For periodontal attachment and repair, cementum is necessary [3].

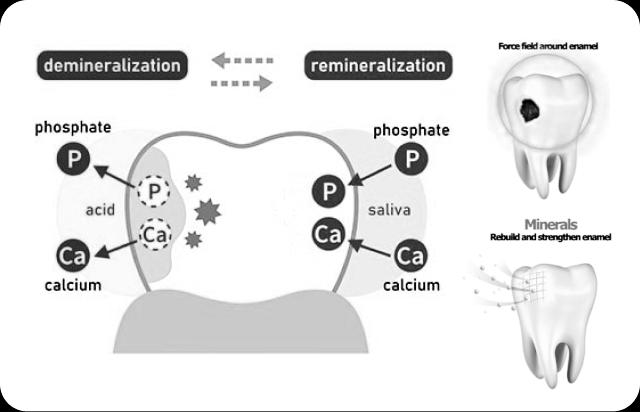
**Pulp** is the soft connective tissue that is the innermost, and it is packed with cells, blood vessels, and nerves. It is essential for dentin formation, immunological response, and tooth vitality. Pulpitis, an infection or inflammation of the pulp, can cause excruciating pain and necessitate endodontic treatment [6]**.**

These tissues work as a cohesive whole to withstand environmental stresses and functional forces [7]. Effective diagnosis and treatment of dental diseases depend on an understanding of the unique characteristics of each component.

**4. DEMINERALIZATION**

Demineralization is the initial stage of the caries process, characterized by the loss of calcium and phosphate ions from the hydroxyapatite crystals of enamel and dentin due to acid exposure in the oral environment. This process is triggered when fermentable carbohydrates, such as sucrose and glucose, are metabolized by acidogenic bacteria most notably Streptococcus mutans and Lactobacillus spp producing organic acids that lower the pH at the tooth surface below the critical threshold of 5.5 [8]. The acidic conditions disrupt the supersaturation of saliva and plaque fluid with respect to hydroxyapatite, leading to subsurface mineral dissolution while the outer enamel may remain intact, forming early non-cavitated white spot lesions [9].

Saliva plays a vital role in buffering acids and supplying minerals; however, reduced salivary flow or altered composition impairs this natural defense, making the tooth more susceptible to progressive demineralization. Repeated acid attacks without sufficient remineralization allow the lesion to advance into the dentin, where the lower mineral content facilitates further decay. If unchecked, the process becomes irreversible, resulting in cavitation that requires restorative intervention.

**Figure:1 Demineralization and Remineralization**

**5. REMINERALIZATION**

Remineralization is the natural or therapeutically assisted process by which partially demineralized tooth structures regain lost mineral content, primarily in the form of calcium and phosphate ions, often facilitated by fluoride. This dynamic repair mechanism plays a vital role in halting or reversing the early stages of dental caries. Under favorable conditions, the dissolved ions in saliva and plaque fluid are redeposited into the enamel lattice, restoring structural integrity and hardness to the tooth surface without the need for invasive intervention [8].

The process of remineralization begins once the pH in the oral environment returns above the critical threshold (approximately 5.5 for enamel and 6.2–6.5 for dentin), often due to the buffering action of saliva. Saliva is rich in bicarbonate, calcium, phosphate, and proteins such as statherin and proline-rich proteins, which stabilize ions in solution and promote their re-incorporation into enamel prisms [10]. These components collectively maintain supersaturation with respect to hydroxyapatite, driving the deposition of minerals into the porous enamel structure previously affected by acid-induced demineralization.

Fluoride, either from systemic sources (e.g., fluoridated water) or topical agents (e.g., toothpaste, varnishes), significantly enhances the remineralization process. It acts by catalyzing the precipitation of fluorapatite a mineral phase more resistant to acid attack than native hydroxyapatite and by inhibiting bacterial metabolism and acid production [11]. Low levels of fluoride in plaque fluid help accelerate the redeposition of minerals and decrease the solubility of enamel, thereby strengthening the tooth surface against further demineralization [12].

Several biomimetic agents have emerged in recent years to enhance remineralization beyond the natural capacity of saliva. One prominent example is casein phosphopeptide-amorphous calcium phosphate (CPP-ACP), which stabilizes high concentrations of bioavailable calcium and phosphate at the tooth surface, promoting site-specific remineralization of early carious lesions [13]. Other agents, such as nano-hydroxyapatite, bioactive glass, and self-assembling peptides (e.g., P11-4), are also being explored for their ability to penetrate subsurface lesions and stimulate crystal regrowth within the enamel matrix.

Remineralization is particularly effective during the early stages of caries development, prior to cavitation. White spot lesions, the initial clinical manifestation of subsurface mineral loss, can often be reversed with consistent use of fluoride toothpaste, dietary control, and application of remineralizing agents [1]. Importantly, remineralization does not replace lost enamel volume in terms of identical microstructure but rather restores functional hardness and resistance to further acid challenge.

The clinical success of remineralization strategies depends on early detection of non-cavitated lesions, patient compliance, and a comprehensive understanding of individual caries risk. Salivary function, oral hygiene, dietary behavior, and fluoride exposure all modulate the efficacy of remineralization in clinical settings [14]. Emerging technologies, such as peptide-guided mineralization and nanomaterial-based delivery systems, hold promise for enhancing both the depth and quality of mineral recovery in demineralized tissues [15].

**6. FACTORS INFLUENCING DEMINERALIZATION AND REMINERALIZATION**

* **pH:**

In a physiological balance of saliva, a normal pH is between 6.5 and 7.4, in which it is supersaturated with calcium and phosphate ions, so demineralization does not take place. Demineralization begins when the pH in the oral environment drops below 5.5, leading to the dissolution of hydroxyapatite (HA) crystals. This acid-induced process damages the enamel, while remineralization primarily driven by saliva helps restore lost minerals. The natural repair of eroded enamel surfaces through remineralization is essential for maintaining tooth integrity and preventing further decay [16].

* **Saliva Flow:**

Reduced saliva flow can impair the buffering capacity and mineral supply for remineralization. Human saliva has a buffering capacity to neutralize the oral cavity’s low pH and acts as a carrier of essential ions, such as fluoride, calcium and phosphate, which have a positive role in enamel’s remineralization. Salivary flow rate, buffering capacity, pH, calcium, phosphate, and fluoride ion concentrations are essential. Acidic pH promotes whereas alkaline pH promotes plaque mineralization [17].

* **Calcium and Phosphate Availability:**

Adequate concentrations of calcium and phosphate ions in saliva are crucial for the remineralization process, which involves the restoration of enamel through the deposition of calcium phosphate onto its surface. Remineralization is characterized by the presence of calcium and phosphate ions in saliva that form hydroxyapatite on the enamel surface [18].

* **Fluoride Concentration:**

Fluoride plays a crucial role in accelerating remineralization and enhancing enamel's resistance to acid attacks. Fluoride promotes the remineralization of teeth by speeding up the formation of fluorapatite on areas where sub-surface enamel crystals have been partially demineralized. Fluoride enhances the precipitation of calcium and phosphate helps to rebuild enamel encourages the formation of fluorapatite, a more acid‑resistant mineral [11].

* **Oral Hygiene:**

Effective oral hygiene practices, like brushing and flossing, help remove plaque and reduce acid production, promoting remineralization [19].

7. ROLE OF SALIVA IN DEMINERALIZATION AND REMINERALIZATION

Saliva is essential in maintaining oral health, particularly through its influence on mineral balance in tooth enamel and dentin. Its functions include buffering acids, providing essential minerals, and supporting overall tooth integrity.

**i) Buffering Acidic Challenges**

Saliva contains significant amounts of bicarbonate, phosphate, and proteins which neutralize acids produced by bacterial metabolism or dietary sources. This buffering ability helps raise plaque pH after acid attacks, slowing or halting demineralization [20]

**ii) Mineral Reservoir and Remineralization**

Supersaturated with calcium and phosphate, saliva serves as a reservoir enabling remineralization when pH normalizes. Salivary proteins (e.g., statherin, proline-rich proteins) help stabilize ions and prevent unwanted precipitation, ensuring availability for enamel repair.

**iii) Formation of the Acquired Enamel Pellicle**

A biofilm-like layer forms on clean enamel within minutes, composed of salivary proteins, lipids, and glycoproteins. This pellicle acts as a semi-permeable barrier against acids and serves as a scaffold for ion deposition .

**iv) Antimicrobial & Enzymatic Actions**

Saliva contains antimicrobial agents (lysozyme, lactoferrin, immunoglobulins) that inhibit harmful bacteria like Streptococcus mutans, reducing acid production. Enzymes such as peroxidases help modulate biofilm ecology, preventing acidogenic shifts.[21]

**v) Salivary Flow and Mechanical Cleansing**

Continuous salivary flow washes away food debris and sugars, minimizing substrate availability for acidogenic bacteria. Reduced flow (xerostomia) is strongly linked to increased caries incidence[22]

**8. CURRENT PREVENTIVE AND THERAPEUTIC APPROACHES**

Demineralization and remineralization are naturally occurring processes in the oral environment. When intervention is timely, non-cavitated lesions can be arrested or even reversed without invasive treatment. Below are the most effective and widely used preventive and therapeutic strategies.

**1. Fluoride-Based Interventions**

* Fluoride remains a gold-standard preventive agent due to its multifactorial role:
* It promotes remineralization by enhancing fluorapatite formation. It inhibits further demineralization by reducing enamel solubility. It exhibits antimicrobial effects on Streptococcus mutans by inhibiting their enzymatic systems [23].

**Common fluoride delivery methods:**

* Toothpastes (1000–1500 ppm fluoride)
* Mouth rinses (0.05% NaF)
* Professional varnishes (5% NaF or 22,600 ppm fluoride)
* Slow-release fluoride devices for high-risk individuals

**2. Calcium and Phosphate Delivery Systems**

* For remineralization to occur, the tooth surface must be in a state of supersaturation with calcium and phosphate ions. Therapeutic products now deliver these ions effectively:

Key systems:

* CPP-ACP (Casein Phosphopeptide-Amorphous Calcium Phosphate): Binds calcium and phosphate, stabilizes them in saliva, and delivers them to early carious lesions.
* CPP-ACFP: CPP-ACP combined with fluoride for triple-action remineralization.
* Bioactive Glass (NovaMin): On contact with saliva, releases calcium and phosphate ions and forms a hydroxycarbonate apatite (HCA) layer on enamel.
* Calcium Glycerophosphate and tricalcium phosphate (TCP): Alternative remineralizing systems often added to toothpaste or varnish [24].

**3. Sealants and Protective Coatings**

* Pit and fissure sealants block bacterial penetration and sugar accumulation in occlusal surfaces. Resin infiltration techniques can arrest non-cavitated lesions, especially interproximal ones.

**4. Xylitol and Sugar Substitutes**

* Xylitol, a non-fermentable sugar alcohol, reduces Streptococcus mutans load and salivary acidity. Chewing xylitol-containing gum increases saliva flow and indirectly promotes remineralization.

**5. Behavioral Interventions and Oral Hygiene**

* Teaching correct brushing techniques along with the consistent use of fluoridated toothpaste plays a key role in promoting dental health. Regular flossing and plaque control [25]. Dietary counseling to reduce intake of fermentable sugars and acidic beverages.

**6. Saliva Stimulation and Substitutes**

* Chewing sugar-free gum stimulates saliva flow. Artificial saliva products are used in patients with xerostomia to mimic natural buffering and mineralizing functions.,[26].

**9.EVALUATION TECHNIQUES FOR DEMINERALIZATION AND REMINERALIZATION**

To assess the effectiveness of preventive and therapeutic strategies in managing early tooth decay, several diagnostic and analytical methods are used. These techniques help in detecting demineralized areas, monitoring remineralization, and evaluating lesion progression.

**1. Visual and Tactile Examination**

* White spot lesions (WSLs) are the earliest clinically visible signs of demineralization.
* Lesions are inspected under good lighting and dry conditions using mirrors and probes.

**2. Radiographic Techniques**

* Bitewing radiographs are commonly used for detecting interproximal caries.Digital radiography enhances image resolution and reduces radiation exposure [27].
* Limitation include it only detects lesions after significant mineral loss (~30–50%).

**3. Quantitative Light-Induced Fluorescence (QLF)**

* Uses fluorescence loss to detect and quantify early enamel demineralization. Useful for longitudinal monitoring of remineralization therapies.
* Can visualize changes invisible to the naked eye .

**4. DIAGNOdent (Laser Fluorescence)**

* Handheld device that emits laser light (655 nm) to detect fluorescence from carious lesions.Provides a numerical value corresponding to lesion severity.
* Non-invasive and ideal for early caries detection in occlusal surfaces [28].

**5. Electrical Conductance Methods**

* Based on the principle that demineralized enamel has higher porosity and therefore greater electrical conductivity.
* Devices measure current flow to assess lesion activity

**6. Transverse Microradiography (TMR)**

* Considered the gold standard for quantifying mineral loss and gain in enamel and dentin.Uses thin tooth sections and X-rays to measure mineral content depth-wise.
* Mainly used in in vitro and in situ studies [29].

**7. Microhardness Testing**

* It assesses the hardness of enamel at the surface or just beneath it to determine the extent of demineralization or remineralization .
* This method is widely applied in laboratory settings for remineralization research.

**8. Scanning Electron Microscopy (SEM)**

* High-resolution imaging technique to observe enamel surface morphology changes due to mineral loss or gain.
* Helpful in understanding crystal structure alterations.

**10. FUTURE PROSPECTS IN THE MANAGEMENT OF TOOTH DEMINERALIZATION AND REMINERALIZATION**

As dental research continues to evolve, the future of managing tooth decay is increasingly shifting toward biomimetic, preventive, and regenerative approaches. New technologies are aiming not only to halt the caries process but to reverse it at the earliest stages.

**1. Biomimetic Materials and Smart Remineralizers**

* Advanced remineralizing agents like peptide-based scaffolds (e.g., P11-4) and bioactive glasses are being developed to mimic the natural mineralization process.
* These materials are engineered to bind to demineralized enamel and provide a framework for apatite crystal regrowth, offering controlled and localized remineralization.

**2. Nanotechnology in Remineralization**

* Nano-hydroxyapatite, due to its enamel-like particle size and surface reactivity, shows exceptional potential for deep lesion penetration and crystal regrowth.
* Nanoparticle-based fluoride, calcium, and phosphate systems offer better surface affinity and slow-release behavior [18].

**3. Gene Therapy and Saliva Engineering**

* Future approaches may include genetic modification of salivary glands to secrete more remineralizing proteins or antimicrobial peptides.
* Engineered saliva could help high-risk patients with hyposalivation or poor mineral buffering.

**4. Targeted Drug Delivery and Nanorobotics**

* Research is exploring nanorobotic delivery systems capable of targeting early lesions with fluoride, calcium, and antibacterial compounds.
* These systems can navigate biofilms and release remineralizing ions only in acidic environments [30].

**5. Artificial Intelligence (AI) in Caries Prediction**

* AI-based diagnostic tools using machine learning and radiomics will help detect incipient lesions earlier and predict caries progression based on patient-specific risk profiles.
* These tools will also help in customizing remineralization therapies [31].

**6. Personalized Remineralization Therapies**

* The future lies in precision oral care, where remineralization strategies are tailored to individual genetic, microbial, and salivary profiles.
* Salivary diagnostics and microbiome analysis may soon determine specific formulations for maximum effectiveness.

**7. Vaccine Development Against Cariogenic Bacteria**

* Development of vaccines targeting Streptococcus mutans and other acid-producing bacteria may provide long-term protection against caries. Research in oral mucosal immunity and peptide-based immunization is ongoing [32].

**11. CONCLUSION**

Demineralization and remineralization are opposing but interconnected processes central to the development and prevention of tooth decay. While acids from bacteria and diet lead to mineral loss, factors like saliva, fluoride, and calcium-based agents help restore tooth structure. Focusing on enhancing natural remineralization through preventive care and modern therapies offers a promising path toward managing dental caries non-invasively [23].

**REFERENCES**

1. Pitts, N. B., Zero, D. T., Marsh, P. D., Ekstrand, K., Weintraub, J. A., Ramos-Gomez, F., ... & Ismail, A. (2017). Dental caries. Nature Reviews Disease Primers, 3(1), 17030. https://doi.org/10.1038/nrdp.2017.30
2. Selwitz, R. H., Ismail, A. I., & Pitts, N. B. (2007). Dental caries. The Lancet, 369(9555), 51–59. https://doi.org/10.1016/S0140-6736(07)60031-2
3. Marsh PD.(1994). Microbial ecology of Dental plaque and its significance in Health and disease. Advances in Dental Research. 8(2):263-271Nyvad, B., & Fejerskov, O. (1997). Assessing the stage of caries lesion activity on the basis of clinical and microbiological examination. Community Dentistry and Oral Epidemiology, 25(1), 69–75. https://doi.org/10.1111/j.1600-0528.1997.tb00897.x
4. Ten Cate, A. R. (2003). Oral Histology: Development, Structure, and Function. 6th ed. Mosby.
5. Roberson, T. M., Heymann, H. O., & Swift, E. J. (2002). Sturdevant’s Art and Science of Operative Dentistry. 4th ed. Mosby.]
6. Kim MJ, Lee MJ, Kim KM, Yang SY, Seo JY, Choi SH, et al. (2021). Enamel Demineralization resistance and Remineralization by various fluoride Releasing dental restorative materials. Materials (Basel).14(16):223-230
7. Lussi A, Schlueter N, Rakhmatullina E, Ganss C.(2011). Dental Erosion. An overview with emphasis on Chemical and histopathological aspects. Caries Research.

45(Suppl 1):2-12

1. Featherstone, J.D.B. (2000). The science and practice of caries prevention. Journal of the American Dental Association, 131(7), 887–899. https://doi.org/10.14219/jada.archive.2000.0307
2. Ten Cate, J. M. (2001).Remineralization of caries lesions extending into dentin. Journal of Dental Research, 80(5), 1407–1411. doi:10.1177/00220345010800050401.
3. Ten Cate, J.M., & Featherstone, J.D.B. (1991). Mechanistic aspects of the interactions between fluoride and dental enamel. Critical Reviews in Oral Biology & Medicine, 2(3), 283–296. https://doi.org/10.1177/10454411910020030401
4. Buzalaf, M.A.R., Pessan, J.P., Honório, H.M., & ten Cate, J.M. (2013). Mechanisms of action of fluoride for caries control. Monographs in Oral Science, 22, 97–114. https://doi.org/10.1159/000325151
5. Featherstone, J. D. (1999). Prevention and reversal of dental caries: Role of low-level fluoride. Community Dentistry and Oral Epidemiology, 27(1), 31–40. https://doi.org/10.1111/j.1600-0528.1999.tb01989.x
6. Reynolds, E.C. (1997). Remineralization of enamel subsurface lesions by casein phosphopeptide-stabilized calcium phosphate solutions. Journal of Dental Research, 76(9), 1587–1595. <https://doi.org/10.1177/00220345970760091001>
7. Amaechi, B. T., Mathews, S. M., Ramirez, J., & Mensinkai, P. K. (2015). Evaluation of nanohydroxyapatite-containing cream for remineralizing early caries lesions. American Journal of Dentistry, 28(6), 321–326.
8. Hannig, M., & Hannig, C. (2010). Nanomaterials in preventive dentistry. Nature Nanotechnology, 5(8), 565–569. <https://doi.org/10.1038/nnano.2010.59>
9. Featherstone JDB. (2004). The continuum of dental caries—evidence for a dynamic disease process. J Dent Res.83 Spec No C:C39-42. Doi:10.1177/154405910408301S08
10. Edgar WM.(1992). Saliva: its secretion, composition and functions.

Br Dent J.172(8):305–312. Doi:10.1038/sj.bdj.4807813

1. Reynolds EC. (2008). Calcium phosphate-based remineralization systems: scientific evidence? Aust Dent J.53(3):268–273. doi:10.1111/j.1834-7819.2008.00064.x
2. Van Houte J. (1994). Role of micro-organisms in caries etiology. J Dent Res. 73(3):672–681. doi:10.1177/00220345940730031201
3. Dawes, C. (2008). Salivary flow patterns and the health of hard and soft oral tissues. Journal of the American Dental Association, 139, 18S–24S. https://doi.org/10.14219/jada.archive.2008.0352
4. Buzalaf, M. A. R., Hannas, A. R., & Kato, M. T. (2012). Saliva and dental erosion. Journal of Applied Oral Science, 20(5), 493–502. https://doi.org/10.1590/S1678-77572012000500001
5. Villa, A., Connell, C. L., & Abati, S. (2015). Diagnosis and management of xerostomia and hyposalivation. Therapeutics and Clinical Risk Management, 11, 45–51. https://doi.org/10.2147/TCRM.S76282
6. Buzalaf, M.A.R., Pessan, J.P., Honório, H.M., & ten Cate, J.M. (2011). Mechanisms of action of fluoride for caries control. Monographs in Oral Science, 22, 97–114. https://doi.org/10.1159/000325151
7. Gao SS, Zhang S, Mei ML, Lo ECM, Chu CH. (2016). Caries remineralisation and arresting effect in children by professionally applied fluoride treatment – a systematic review. BMC Oral Health.;16:12. Doi:10.1186/s12903-016-0171-0
8. Hamba H, Nikaido T, Inoue G, et al. (2011) .Effects of CPP-ACP with sodium fluoride on in vitro remineralization of human enamel. J Oral Sci.53(4):471–477. Doi:10.2334/josnusd.53.471
9. Hicks J, Garcia-Godoy F, Flaitz C. (2004). Biological factors in dental caries: Role of remineralization and fluoride in the dynamic process of demineralization and remineralization.J.Clin.Pediatr.Dent.;28(3):203–214. Doi:10.17796/jcpd.28.3.2256547r16343427
10. Gjorgievska ES, Nicholson JW.(2010) .A preliminary study of enamel remineralization by dentifrices based on Recaldent™ (CPP-ACP) and Novamin™ (calcium-sodium-phosphosilicate). Acta Odontol Latinoam.23(3):234–239.
11. Pretty IA.(2006). Caries detection and diagnosis: novel technologies. J Dent.;34(10):727–739. Doi:10.1016/j.jdent.2006.06.004
12. Gómez J. (2015) .Detection and diagnosis of the early caries lesion. BMC Oral Health.;15(Suppl 1):S3. Doi:10.1186/1472-6831-15-S1-S3
13. Bansal et al. (2021). Nanotechnology in caries prevention: A futuristic approach. Int J Pharm Investig, 11(3), 289–294.
14. Schwendicke et al. (2020). Artificial intelligence in caries detection: A scoping review. J Dent, 100, 103408.
15. Smith, D. J., & Taubman, M. A. (2006). Experimental immunization against dental caries. Immunol Invest, 35(1), 89–103.