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Salivary Glands and Saliva: An Overview

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Abstract

Saliva is the least known and the least appreciated of all body fluids. It's hardly something to take seriously until, that is, we lack it. The properties and functions of saliva have been studied extensively for more than sixty years. Saliva is a complex secretion. 93% by volume is secreted by the major salivary glands and the remaining 7% by the minor glands. These glands are located in every region of the mouth except for the gums and the anterior part of the hard palate. Saliva is sterile when it leaves, but ceases to be so, as soon as it mixes with the crevicular fluid, remains of food, microorganisms, desquamated oral mucous cells, etc. This complex biofluid plays an essential role in the maintenance of oral health. Saliva is constituted by water, organic and inorganic components which have biological functions essential for homeostasis of the oral cavity. It contains a wide variety of unique proteins, including proline rich proteins (PRPs) and enzymes such as lysozyme, lactoferrin, peroxydases, and secretory IgAs. Saliva secretion is controlled by the autonomous nervous system with the volume produced varying according to the type and intensity of stimulation. A greater volume is produced before, during and after meals whereas a lower salivary production is observed during sleep. Adequate salivary flow and composition are recognized as important for lubrication and protection of soft and hard oral tissues. Protection of soft tissues is provided against desiccation, penetration, ulceration, and potential carcinogens by mucin and anti-proteases. A major protective function results from the salivary role in stabilizing the ecological balance in the oral cavity via clearance, aggregation and reduced adherence by both immunological and non-immunological means as well as

direct antimicrobial activity. Saliva is effective in maintaining pH in the oral cavity by its buffer capacity and contributes to the regulation of dental biofilm pH and enamel surface integrity. Salivary maintenance of tooth integrity depends on mechanical cleansing and enamel remineralization. For these reasons individuals with impaired salivary synthesis and secretion may have difficulties in eating, swallowing and become prone to oral diseases such as mucosal infections and dental caries.

Keywords: Saliva, Anatomy, Secretion, Constituents.

Introduction

Overview of salivary glands

Salivary gland is any cell or organ discharging a secretion into the oral cavity. These are exocrine glands whose secretions flow into the oral cavity. There are three pairs of large glands located extra-orally known as the major salivary glands and numerous small glands widely distributed in the mucosa and sub mucosa of the oral cavity, known as the minor salivary glands. Both the major and minor glands are composed of parenchymal elements invested in and supported by connective tissue. The parenchymal elements are derived from the oral epithelium and consist of terminal secretory units leading into ducts that eventually open into the oral cavity. The connective tissue forms a capsule around the gland and contains the blood and lymph vessels and nerves that supply the gland. The most important function of these glands is the production of saliva, which provides the primary protection for the teeth and soft tissues of the oral cavity, and assists in the mastication, deglutition and digestion of food.[1] The major salivary glands consist of three bilaterally paired glands namely Parotid glands, Submandibular glands and Sublingual glands. The anterior glands of the tongue, small lingual glands and labial buccal and palatal glands are termed as minor salivary glands.[2] The parotid duct also known as Stensen's duct secretes a serous saliva into the vestibule of the oral cavity.From the anterior border of the gland,it travels parallel to the zygoma, approximately 1cm below it,in an anterior direction across the masseter muscle.It then turns sharply to pierce the buccinator muscle and enters the oral cavity opposite the second upper molar tooth.The wall of the parotid duct is thick with an external fibrous layer containing smooth muscle and mucosa lined by low columnar epithelium. Its calibre is about 3mm, although smaller at its oral orifice.[3]

Histology of salivary glands

These glands are compound racemose in type and have numerous lobes composed of lobules linked by dense connective tissue containing excretory ducts, blood vessels, lymph vessels, nerve fibres and small ganglia. The terminal secretory units of salivary glands are composed of serous, mucous and myoepithelial cells arranged into secretory tubules called acini. The secretions are collected by the intercalated ducts, which are then emptied, into the striated ducts. Serous cells are specialised for the synthesis, storage and secretion of proteins. The typical cell is pyramidal in shape, with its broad base resting on a thin basal lamina and its narrow apex bordering on the lumen. The spherical nucleus is located in the basal region of the cell. At the apical ends of the adjoining cells, the lumen is sealed off from the lateral intercellular spaces by junctional complexes, consisting of a tight junction (zonula occludens), an intermediate junction (zonula adherens), and one or more desmosomes. These junctions serve to hold the cells together as well as prevent leakage of the luminal contents into the intercellular spaces. Finger like branches of the lumen called intercellular canaliculi, extend between adjacent cells almost to their base; they

increase the surface area of the secretory surface and are sealed by junctional complexes along their length.[4]

The physiologic control of secretion

The physiological control is mediated through the activity of the autonomous nervous system. The release of neurotransmitters from the vesicles in the nerve terminals adjacent to the parenchymal cells stimulates them to discharge their secretory granules and secrete water and electrolytes. The neurotransmitters interact with specific receptors located on the plasma membrane of the acinar cell. Norepinephrine, the sympathetic transmitter, interacts with both receptors, while acetylcholine interacts with the cholinergic receptor. Protein secretion is mediated through the adrenergic receptor; stimulation of the adrenergic and cholinergic receptors also causes low levels of protein secretion, but these two receptors appear to be mainly involved in the secretion of water and electrolytes. Receptors for the peptide transmitter substance P are also present on salivary gland cells; substance P stimulates secretion similar to that caused by adrenergic and cholinergic agonists. Vasoactive intestinal polypeptide (VIP) is present in the nerve endings in the salivary glands.[5] Adjacent secretory cells are joined to one another by specialized intercellular junctions called gap junctions. These junctions are permeable to ions and small molecules; thus, changes in the intracellular concentration of these substances in one cell are reflected by parallel changes in the adjacent cells. The discharge of the secretory granules involves the translocation to the luminal cell surface and fusion of the granule membrane with the plasma membrane.[6] Emetic circulation of salivary glands is very important for saliva formation. Actually only in cells where there is blood passage there can be saliva formation. Important studies suggest that pressure generated by salivary secretion is dependent on blood pressure. Parasympathetic stimulation of salivary glands causes a higher blood flow. The current secretion model predicts that, when stimulated, salivary acinar cells lose KCl. The loss of K+ and Cl-, across basolateral and apical membranes, respectively, creates a large transepithelial potential difference. This lumen-negative potential difference drives Na+ flux between acinar cells into the lumen, and H2O follows the resulting NaCl osmotic gradient. Sustained secretion is dependent on the activation of K+ and Cl- reuptake mechanisms located in the basolateral membranes. The net effect of simultaneously activating Cl- efflux and Cl- reuptake mechanisms is transepithelial Cl- movement, the driving force for fluid secretion. It is interesting to note that little desensitization occurs, i.e., in the continuous presence of a muscarinic agonist, salivary glands can secrete for hours. A salivary acinar cell contains four ion transporters, the Na+/K+ adenosine triphosphatase (ATPase), a Na+-K+-2Cl- cotransporter and a Ca2+-activated K+ channel, all located in the basolateral membrane, and a Ca2+-activated Cl- channel located in the apical membrane. Fluid secretion is thought to arise from the concerted actions of these four transporters.[7]

Composition of saliva [8]

Organic components

1.Protein:

Amylase

Lysozyme

Glycoproteins

IgA

Traces of blood proteins

Albumin,IgM,IgG

Transferrin

Lipoproteins

2.Nitrogenous constituents:

Amino acids
Urea
Uric acid
Ammonia
Creatin
Statherian
Sialin
3.Glucose
4.Blood group substances A, B, O.
5.vitamins- water soluble
6.Intrinsic factor- apoerythein
7.Lipids : Cholesterol, fatty acids tri gycerides, phospholipds.
8.Enzymes
Acid phosphatase
Esterases,Lysozyme
Kallikrein
9.Other organic compounds
Citrates
Nitrates
10.Anti –bacterial
Proteins
Lysozyme
Sialoperoxidase
Lactoferrin
Proteins
Salivary enzymes
Mucoproteins & Glycoproteins
Blood group substances
Hormones
Carbohydrates
Lipids
Nitrogen containing substances
Lactoferrin

Inorganic substances [7,9]

Sodium
Potassium
Chlorides
Bicarbonates
Hydrogen ions
Iodine
Fluoride
Thiocyanate
Calcium
Phosphates

Salivary enzymes [9]

Amylase :-1) metabolises starch, converts cooked starch to maltose.
2)produced by acinar cell of the major salivary gland.
Lysozyme: 1) enzymatic protein.2) direct antimicrobial effect. 3) first seen in nasal secretion and tears. 4) its ability to bind to hydroxyapetite suggest antimicrobial role on tooth surface.
Peroxidases: produced by acinar cell of major salivary glands.
Kallikrein
Numerous miscellaneous enzymes
Mucoprotien and glycoprotein:-Contains a large portion of carbohydrates.
Proline, glycine and glutamic acid comprise major amino acids.
Contribute to enamel pellicle and prevent tooth surface bacterial colonization. Eg:- mucin by aggregation promotes bacterial clearance.
Blood group substances:- A, B, O and AB groups
LE antigen family (Lewis antigen family)
Hormones
Parotin : facilitates calicification and helps to maintain serum calcium levels to an optimum. Helps in calcium deposition of teeth.

Carbohydrates: Glucose is present in same concentration as blood. Sub mandibular gland contains hexose and fructose.

Proteins. [9]

Recent developments in proteomics have resulted in the identification of a large number of different proteins, both in whole saliva and in secretions from individual glands. The technique uses an initial separation of proteins by means of electrophoresis or chromatography, isolation of small groups of proteins or their constituent peptides and, after further separation by means of chromatography, identification of the peptides via mass spectrometry. From a database of the peptides in known proteins, researchers can identify the proteins present in saliva. The presence of certain proteins in saliva may be predictive of squamous cell carcinoma. Researchers have identified as many as 309 proteins in whole saliva and 130 in the acquired enamel pellicle. However, proteomics cannot be used to identify the concentrations of the individual proteins, many of which are present in only trace amounts. In fact, more than 95 percent of salivary protein is from the major salivary protein families, which include acidic and basic proline-rich proteins, amylase, high-and low-molecular-weight mucous glycoproteins (MUC5B and MUC7), agglutinins, cystatins, histatins and statherin. After protein synthesis in the salivary glands, many of these proteins undergo posttranslational modifications, which include glycosylation, acylation, deamidization, sulfation, phosphorylation and proteolysis, before they enter the mouth. The main contributor to salivary viscosity is the mucous glycoprotein MUC5B, secreted primarily by MMGs, and it exhibits great heterogeneity in its glycosylation pattern. In a given person, the proportions of different proteins in saliva from a particular gland appear to be independent of the nature of the stimulus. However, differences exist among people, because many of the protein families exhibit genetic polymorphisms.

Amylases [10,11]

Amylase in saliva is present as about 6 isoenzymes [Merritt and Kahn 1978].α-amylase constitutes about 30% of the total protein in parotid saliva but its concentration is considerably lower in submandibular saliva. α-Amylase is a hydrolytic enzyme which cleaves the α (1-4)glycosidic linkages in starch and glycogen. It contains about 1gm atom of calcium per molecule of enzyme and requires chloride ion for its activity. There is always sufficient chloride in saliva for full enzymatic activity and the optimum pH is about neutrality. Amylase is one of the most important salivary digestive enzymes. Two families of isoenzymes, of which one set is glycosylated and the other contains no carbohydrate (Mäkinen, 1989) have been identified. Salivary amylase is a calcium metalloenzyme which hydrolyses the alpha bonds of starches, such as amylose and amylopectin (Hay and Bowen, 1996). Maltose is the major end-product. It has been suggested that amylase accounts for 40 to 50% of the total salivary gland-produced protein, most of the enzyme being synthesized in the parotid gland (Noble, 2000). Human parotid saliva and submandibular saliva contain about 45 mg and 30 mg of amylase, respectively, per 100 mg of protein (Mäkinen, 1989). However, it has also been claimed that amylase makes up about 1/3rd of the total protein content in parotid saliva, and the content in whole saliva would be lower (Pedersen et al., 2002). The concentration of amylase increases with the salivary flow rate, and it is generally considered to be a reliable marker of serous cell function (Almståhl et al., 2001).

Concentrations of inorganic components of whole saliva[9]

	Unstimulated		Stimulated	
Electrolyte	Mean± s.d	Range(µmol/l)	Mean± s.d	Range(µmol/l)
Sodium	7.7 ± 3.0	2 - 26	32 ± 20	13-80
Potassium	21 ± 4	13 - 40	22 ± 20	13 - 38
Magnesium	0.31 ± 0.22	0.15 – 0.6	0.18 ± 0.15	
Copper	0.4	0.2 – 0.8	0.4	0.2 – 0.8
Lead			0.55	0.14 – 1.11
Cobalt			1.2	0 – 2.0
Strontium	0.4 ± 0.01	0.1 - 33	1.0±0.1	0.1 – 1.2
Chloride				10 – 56
Hydrogen carbonate	24 ± 8		25±18	
Phosphate				1.5 – 25
Iodide	5.5 ± 4.2	2 - 22	10 ± 7	
Bromide			14 ± 8	0.01 – 0.1
Thiocynate				
Hypothiocynate			1.2 ± 0.7	
Nitrate			1.1 ± 0.5	79 - 183
Nitrite			178 ± 11	
Fluoride			68 ± 11	
Sulfate	1.45 ± 0.6		5.8 ± 0.25	

Properties of salivary immunoglobulins [9,12,15]

1.Stability of SIgA

SIgA is remarkably stable and therefore suited to function in protease- containing external secretion such as whole saliva. Its resistance may be ascribed both to the dimeric IgA structure per se and to the incorporation of SC. At least 60% of salivary SIgA consists of IgA1 and parotid antibodies to S, mutans occur predominantly in this subclass.

2.Mucosal Defense

SIgA antibodies act in a first line of mucosal defense principally by simple binding to soluble or particulate antigens. This function of immune exclusion is enhanced by cooperation of SIgA with nonspecific defense factors. In addition to effective binding and complexing of antigens. SIgA antibodies show better agglutinating properties than monomeric IgA.In neonates salivary free SC apparently sticks to buccal cells and mediate binding of dimeric IgA from breast milk, thus inhibiting colonization of microorganisms on epithelial cells. The IgA antibodies induce loss of bacterial plasmid that code for adherence associated molecules and resistance to antibiotics. Siga antibodies cooperate with various host cells. Fcα receptors on the polymorphonuclear leukocytes show much higher binding activity for dimeric SIgA. Thus SIgA are able to block receptors for chemotactic signals . SIgA mediate antibody – dependent cyto-toxicity against bacteria.

3.Dental Defense

Salivary IgA is incorporated into dental plaque and probably also into the acquired pellicle. Hum and parotid fluid from young adults regularly contain IgA antibodies to glucosyltransferase (GTF) which inhibit this enzyme. GTF is considered to be important for dental plaque formation. SIgA antibodies cause agglutination of bacteria and enhance elimination of S. mutans from the oral cavity or interfere with bacterial adherence to the enamel.

Influence of Age and Related Variables on salivary Ig [9,13,14]

1. **Infancy and childhood**

IgG is the only readily detectable salivary Ig class at birth The concentration of IgG in whole saliva was found to decrease and only little IgG was detected after 2 months. A considerable portion of the infants have relatively larger amounts of locally produced IgD in their whole saliva the 6 months after birth. Expression of SC in salivary gland epithelium can be detected as early

as the 20th gestational week. At the time of delivery, SC expression has increased and there are more IgA and IgM producing cells in the salivary glands, during first year postpartum a growing number of immunocytes can be noted. Free SC is present in relatively large amounts at birth., and low levels of salivary SIgA and SIgM antibodies to E.coli. and poliovirus were found during the first year of life.

During the same period of time upto about 2 years of life ,there is a striking increase in the salivary IgA level, reaching 50 to 80 mg/ml in unstimulated saliva. After this age there is very little increase in the salivary IgA levels and the adult level is reached during later period of childhood.

Ig levels in human parotid secretion and whole saliva from young adults [9]

Samples	Concentration (mg/l ± S.D)			Secretion rate of IgA (µg/min/gland± SD)
	IgA	IgM	IgG	
Stimulated parotid secretion	39.5± 13.7	0.43± 0.36	0.36±0.30	27.2±8.7
Stimulated whole saliva secretion	35.5±11.6	N.D	N.D	33.8±13.3
Unstimulated parotid secretion	119.6±48.3	N.D	N.D	10.0±6.6
Unstimulated whole saliva	194.0±53.7	2.1±1.9	14.4±9.0	N.D

Vaccination [12]

Experimental vaccination against Dental Caries

Brandtzaeg in 1983 and Krasse in 1987revied the result of vaccination in animals with various S. mutans. Subcutanoeus immunization with killed S. mutans cells shown by Lahner and his colleagues showed a 70% reduction of caries in deciduous teeth of rhesus monkeys. They have also demonstrated that the effect of the subcutaneous vaccine is probably based on the bactericidal and opsonising properties of serum derived IgG antibodies reaching the enamel surface through the crevicular fluid. Lehner et al reported that the dental application of monoclonal antibody to S.mutans afforded reduced caries in monkeys. It was shown that parenteral administration of killed S. mutans vaccines in monkeys has elicited little or no salivary IgA response humans the application of enteric coated capsules with killed indigenous S. mutans showed no increase in salivary antibody level, but there was a marked reduction of oral colonization of implanted S. mutans strains. A second period of immunization resulted in a secretory type of antibody response. Mestecky et al have recently provide support at the cellular level for the induction of a disseminated IgA response after enteric immunization with killed S .mutans whole cells. After 7 days the peripheral blood mononuclear cells showed to secrete a spontaneously specific IgA antibodies. Therefore this indicated that a salivary IgA response to killed S. mutans cells may be elicited in man by the enteric route of immunization.

Two groups of proteins from mutans streptococci represent primary candidates for a human caries vaccine: i) glucosyltransferase enzymes, which synthesize adhesive glycans and allow microbial accumulation, and ii) cell surface fibrillar proteins that mediate adherence to the salivary pellicle. The bacterial adhesin SAI/II , a surface-displayed protein with a molecular mass of 190 kDa, plays an important role in the initial attachment of S. mutans to the tooth surface. Antibodies recognizing this protein prevent colonization of the buccal cavity by the bacterium and could be developed as a vaccine

against dental caries. The most suitable vaccination strategy would be passive immunization, in which monoclonal antibodies or fragments thereof are applied to the tooth surface e.g. using toothpaste, mouthwash or chewing gum. This would make active immunization with the S. mutans adhesin unnecessary.

The murine monoclonal antibody Guy's 13 which specifically recognizes the SAI/II protein of S. mutans and Streptococcus sobrinus has been used successfully to prevent S. mutans colonization and the development of dental caries in non-human primates . The antibody also prevented bacterial colonization in human clinical trials. However, like other murine antibodies, a major limitation in clinical applications may be the human anti-mouse antibody response (HAMA), which can increase the rate of clearance and initiate allergic reactions . The problems associated with murine antibodies can be overcome by replacing murine sequences with their human counterparts, e.g. by chimerization , CDR grafting and guided selection using phage display technology. Furthermore, the use of antibody fragments rather than whole antibodies also removes some of the constant regions that may provoke an immune response.

There has been a growing interest in the use of single-chain fragment variable (scFv) antibodies, in which the variable regions of the heavy and light chains are combined in the same polypeptide chain (Huston, 1988 #2785). The advantages of such derivatives are that they can be expressed as single transgenes in various hosts, they fold spontaneously to adopt the correct tertiary structure, and their small size facilitates tissue penetration. The scFv has the heavy and light chain variable regions joined by a flexible peptide linker allowing the two domains to interact, forming a univalent antibody. Alternatively, diabodies have the same structure but the two domains are joined by a shorter, less-flexible linker, forcing dimerization and the formation of divalent antibodies (Holliger, 1993 #3498).

Streptococcus mutans has been identified as the major etiological agent of human dental caries. The first step in the initiation of infection by this pathogenic bacterium is its attachment (i.e., through bacterial surface proteins such as glucosyltransferases, P1, glucan-binding proteins, and fimbriae) to a suitable receptor. It is hypothesized that a mucosal vaccine against a combination of S. mutans surface proteins would protect against dental caries by inducing specific salivary immunoglobulin A (IgA) antibodies which may reduce bacterial pathogenesis and adhesion to the tooth surface by affecting several adhesins simultaneously. Conventional Sprague-Dawley rats, infected with S. mutans at 18 to 20 days of age, were intranasally immunized with a mixture of S. mutans surface proteins, enriched for fimbriae and conjugated with cholera toxin B subunit (CTB) plus free cholera toxin (CT) at 13, 15, 22, 29, and 36 days of age (group A). Control rats were either not immunized (group B) or immunized with adjuvant alone (CTB and CT [group C]). At the termination of the study (when rats were 46 days of age), immunized animals (group A) had significantly ($P < 0.05$) higher salivary IgA and serum IgG antibody responses to the mixture of surface proteins and to whole bacterial cells than did the other two groups (B and C). No significant differences were found in the average numbers of recovered S. mutans cells among groups. However, statistically fewer smooth-surface enamel lesions (buccal and lingual) were detected in the immunized group than in the two other groups. Therefore, a mixture of S. mutans surface proteins, enriched with fimbria components, appears to be a promising immunogen candidate for a mucosal vaccine against dental caries.

The first step necessary for any pathogenic bacterium to initiate infection is its attachment to a suitable receptor. Several different attachment mechanisms have been identified for oral bacteria (i.e., through surface proteins, such as glucosyltransferases [GTF] and glucan-binding proteins, by sucrose-dependent mechanisms and through surface antigen P1 and/or fimbriae in sucrose-independent functions). Bacterial fimbriae have been defined as small (100 to 300 nm), nonflagellar, filamentous, proteinaceous surface appendages that do not participate in the transfer of bacterial or viral nucleic acids .Streptococcus mutans has been identified as the major etiological agent in human dental caries and comprises a significant percentage of the oral streptococci in carious lesions .Fimbriae have been identified on numerous gram-negative microorganisms as long fibrillar structures but have been reported for only a limited number of gram-positive microorganisms, including some oral streptococci, in which they typically appear as a much shorter fuzzy coat .It is our belief that fimbriae are important virulence factors for S. mutans and are at least partially responsible for S. mutans sucrose-independent adherence to enamel surfaces. We have isolated a mixture of S. mutans surface proteins, which contained fimbria components (fimbria-enriched preparation), as demonstrated by immunostaining and electron microscopy, and have elicited antibodies in rabbits against this preparation.

An essential goal in the development of a vaccine for dental caries is to induce antibodies that block bacterial adhesion and, therefore, prevent bacterial colonization. This should then affect the formation of carious lesions. A number of studies with experimental animals and humans have shown that active and passive immunizations with S. mutans, either with whole cells or with different cellular components, inhibit S. mutans colonization and the subsequent formation of dental caries .An in vitro microbial model was used to demonstrate, for the first time, the efficacy of antibodies against the fimbria-enriched preparation in preventing the formation of carious lesions.

The association of S. mutans soluble cell protein antigens (e.g., P1) or dextran preparations with cholera toxin (CT) and the B subunit of CT (CTB) has been shown to increase the immunogenicity (salivary immunoglobulin A [IgA] antibody responses) of many antigens given perorally, intragastrically, or intranasally without causing toxic effects .However, only two studies have addressed the role of salivary antibodies elicited intranasally by an antigen linked to CTB in protection against dental caries CT is an exceptionally immunogenic antigen. This is attributed to the immunopotentiating (or adjuvant) property of CT, as well as to the ability of nontoxic CTB to bind to cell surface GM_1 ganglioside and act as a carrier protein.

Conclusion

The protective mechanisms in the oral cavity may be divided into two categories, physical and chemical. The physical mechanisms may have two manifestations, the first of which is displacement, by which mucous secretions may displace a microorganism from its resting place on the mucous tissue. The second is removal or flushing, in which the saliva flow washes away organisms and, together with tongue movements, assists in their removal to other parts of the alimentary tract. The chemical mechanisms in saliva may also be said to have two manifestations. One is exogenous, such as bacterial antagonisms that limit some members of the oral microflora, and the other is endogenous which includes the intrinsic constituents of secreted saliva. Saliva not only lubricates the oral tissues, making oral functions such as speaking, eating, and swallowing

possible, but also protects teeth and oral mucosal surfaces in different ways. The lubricating and antimicrobial functions of saliva are maintained mainly by resting saliva. Stimulation of saliva results in a flushing effect and the clearance of oral debris and noxious agents. However, the protective functions of saliva are not limited to the above-mentioned functions. Recent studies have revealed a large number of functions, mediated by both the inorganic and organic components of saliva that should be considered in assessments of the effects of human saliva on dental caries. Some of these studies have introduced a new approach to dental caries from being a bacterially induced multifactorial disease to a disease which may also be influenced by inherited salivary factors. Such genetically regulated salivary components may influence both the colonization and the clearance of micro-organisms from the oral cavity.

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