

A Statistical Study on Microorganisms that Cause Tooth Decay and Prevention and Treatment Methods

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Abstract

The current research includes knowledge of the effect of microbes on dental caries, by providing the environment suitable for the growth of bacteria and fungi that cause this decomposition, fermentation and decay and thus erode the texture of the tooth and then full tooth decay. These are the macrophages that lead to tooth decay (*streptococcus species*, *Staphylococcus aureus*, *Candida*, *Lactobacillus*, *Actinomyces*, *Micrococcus*, *Antibacterials*, *Bacillus*, *Actinobacillus*) in different percentages: 33.3%, 21.3%, 14.7%, 12.1%, 8.2, 4.5%, 3.4%, 1.3%, 1.2%), respectively. There are many other factors that help tooth decay occur: the host (tooth and saliva), microorganisms in the form of tooth plaque, substrate (diet).

Keyword: microbes , tooth decay, Prevention and Treatment.

Introduction

Dental caries is an infectious microbiological disease of the teeth that results in localized dissolution and destruction of the calcified tissues. It is the second most common cause of tooth loss and is found universally, irrespective of age, sex, caste, creed or geographic location. Saliva has a cleansing effect on the teeth. Normally, 700–800 ml of saliva is secreted per day. Caries activity increases as the viscosity of the saliva increases. Eating fibrous food and chewing vigorously increases salivation, which helps in digestion as well as improves cleansing of the teeth. The quantity as well as composition, pH, viscosity and buffering capacity of the saliva plays a role in dental caries^(1, 2). Dental caries is one of the most common preventable diseases which is recognized as the primary cause of oral pain and tooth loss. It is a major public health oral disease which hinders the achievement and maintenance of oral health in all age groups⁽³⁾.

Dental caries refers to the localised destruction of susceptible dental hard tissues by acidic by-products from the bacterial fermentation of dietary carbohydrates.

It is a chronic disease that progresses slowly in most of the people⁽⁴⁾, which results from an ecological imbalance in the equilibrium between tooth minerals and oral biofilms (plaque)⁽⁵⁾. Caries is an etiologically complex disease process. It is likely that numerous microbial, genetic, immunological, behavioral, and environmental contributors to risk are at play in determining the occurrence and severity of clinical disease. Assessment tools based on a single risk indicator are therefore unlikely to accurately discriminate between those at high and low risk. Multiple indicators, combined on an appropriate scale and accounting for possible interactions, will certainly be required.

Caries may be characterized by the experience of pain, problem with eating, chewing, smiling and communication due to missing, discolored or damaged teeth. The microbial community of caries is diverse and contains many facultatively and obligately-anaerobic bacteria belonging to the genera *Actinomyces*, *Bifidobacterium*, *Eubacterium*, *Lactobacillus*, *Parvimonas* and *Rothia*⁽⁶⁾. It can also be caused by other bacteria, including members of the mitis, anginosus and salivarius groups of streptococci, *Propionibacterium*, *Enterococcus faecalis*, *Scardovi*, *Prevotella*, *Selenomonas*, *Dialister*, *Fusobacterium*, *Pseudoramibacter*, *Veillonella*, *Atopobium*, *Granulicatella*, *Leptotrichia* and *Thiomonas*⁽⁷⁻¹⁰⁾.

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Bacteroides, *Prevotella*, and *Porphyromonas* species are prevalent on mucosal surfaces and reach very high concentrations in dental plaque, gingival crevices and tonsillar crypts (7).

Traditional approaches to the treatment of dental caries have focused on repairing the consequences of the disease (cavities) rather than the disease itself. From our perspective, person-centered approaches, such as individual risk assessment, active surveillance, oral health literacy, and preventive interventions/therapies, supplemented, when necessary, by surgical care (drilling, filling, extraction) are the essential evidence-based approaches for the effective management of this disease. We see that factors such as fear, total and reimbursed costs, provider availability, transportation, and even parent or caregiver characteristics, including financial distress, depressive symptoms, and limited social networks, can be barriers to care (11). Recently, nanotechnology has emerged in the treatment and protection of teeth, such as nanopowder technology and nanotechnology for metals, which has become a major concern for scientists and researchers.

Materials and methods of work

First\ Materials:-

All diagnostic and general plant media, used reagents and solutions are manufactured by a company (Mast and Oxoid).

Second\ methods of work:-

1. Collection of samples

A total of 40 mouth and teeth swabs were collected for patients that have dental caries in the Dental Unit at Manpuole Hospital in Andrapradesh city, Hyderabad State, India, during (April and May of 2014).

2. Isolation and diagnosis

Samples were growth directly on pre-prepared culture media and laboratory tests were performed to diagnose the bacteria based on the methods of (smith, 2002) and (colle and et al, 1969)⁽¹²⁾, according to the following diagnostics:-

A) Cellular diagnosis:

This was done by examining the pigmented dye of gram to determine the shape and assembly of bacterial cells and the nature of their interaction with the dye.

B) culture diagnosis:

It ensures the diagnosis of the shape and size of bacterial colonies and their effect on the general and specific agro-culture and diagnostic communities.

C) Biochemical tests:

Diagnosis of bacteria is based on their ability to produce enzymes and their biochemical effectiveness.

3. Statistical analysis:

The results of isolation and diagnosis were analyzed statistically using the Kay square test (Daniel ,1978)⁽¹³⁾.

Results and Discussion

The percentages of germs isolated from oral infections showed that *streptococcus species* represented (33.3%) , followed by *Staphylococcus aureus species* (21.3%) , while the *Bacillus* and *Actinobacillus species* represented (1.4% ,and 1.5%) respectively. While the rest of the isolates recorded different ratios of germs as shown in the **table (1)**

Table (1): Proportion of germs isolated from oral infections

No.	Types of germs	Percentages of germs
1-	streptococcus species	33.3%
2-	Staphylococcus aureus species	21.3%
3-	Candida species	14.7%
4-	Lactobacillus species	12.1%
5-	Actinomyces species	8.2%
6-	Micrococcus species	4.5%
7-	Antirobacteriacea species	3.4%
8-	Bacillus species	1.3%
9-	Actinobacillus species	1.2%

In general, the results of this study were consistent with previous , (Saini, 1999) , (Socransky, 1970) such as (Gibbons ,et al. 1946) (Carlsson, 1967) ⁽¹⁴⁻¹⁶⁾. The prevalence and spread of germs of various kinds in the oral cavity, which leads to tooth decay, especially in the

case of negligence and non-cleaning of the teeth and lack of care, which provides a catalyst that leads to loss of age and seek compensation instead of because the presence causes the mouth to disease Including tissue damage and even cancer, which finds the right environment to occur easily. Tooth decay is so widespread that many people do not treat it with the proper seriousness. It is common, for example, lack of interest in the injury of children tooth decay in milk teeth. However, dental caries can lead to complications and complications of serious and far-reaching, even in children whose teeth have not yet developed. Among these complications. Abscesses, Abscess in the teeth, tooth loss, broken teeth, chewing problems, Acute infections, In addition, when dental caries reaches a stage where the aches are very severe, this may interfere with normal daily life, to the extent that it prevents the student from going to school or to work. If the aches are severe and hinder the process of eating or chewing, they may lead to malnutrition and weight loss. If decay leads to tooth decay, this may negatively affect self-confidence. In some very rare cases, abscess caused by dental caries may lead to severe contamination that may endanger the patient's life if not properly treated. The percentage of pathogens that cause oral diseases varies depending on the type of the isolation and method of infection.⁽¹⁷⁾.

Dental plaque is a thin, tenacious microbial film that forms on the tooth surfaces. Microorganisms in the dental plaque ferment carbohydrate foodstuffs, especially the disaccharide sucrose, to produce acids that cause demineralization of inorganic substances and furnish various proteolytic enzymes to cause disintegration of the organic substances of the teeth, the processes involved in the initiation and progression of dental caries. The dental plaque holds the acids produced in close contact with the tooth surfaces and prevents them from contact with the cleansing effect of saliva.^(18, 19).

A Biological Factors of dental caries:-

There is interaction and cooperation between a group of factors that are active and help to prevent tooth caries, and these factors are the conditions that provide suitable and controlled in the incidence of this disease, We can epitomize it up with the following:-

1-Host (teeth and saliva):^(20, 21)

A\ Tooth:

Composition:- Deficiency in fluorine, zinc, lead and

iron content of the enamel is associated with increased risk caries. Morphological **characteristics:-** Deep, narrow occlusal fissures, and lingual and buccal pits tend to trap food rubbish and bacteria, which can cause caries. As teeth get worn (attrition), caries declines.

Position:- The interdental areas are more oversensitive to dental caries. Malalignment of the teeth such as crowding, abnormal spacing, etc. , can increase the susceptibility to caries.

B\ Saliva: ^(22, 23)

Saliva has a cleansing effect on the teeth. Generally, 700– 800 ml of saliva is secreted daily. Caries activity increases as the viscosity of the saliva increases. Eating fibrous food and chewing vigorously all them increases salivation, which helps in digestion as well as lead to cleansing of the teeth. The quantity as well as composition, pH, viscosity and buffering

capacity of the saliva plays an important role in dental caries.

- **Quantity:** Reduction of salivary secretion as found in xerostomia and salivary gland aplasia gives rise to increased caries activity.

- **Composition:** Inorganic fluoride, chloride, sodium, magnesium, potassium, iron, calcium and phosphorus are inversely related to caries. Organic ammonia retards plaque make up and neutralizes the acid.

- **pH:** A neutral or alkaline pH can neutralize acids created by the action of microorganisms on carbohydrate food substances.

- **Antibacterial factors:** Saliva keep enzymes such as lactoperoxidase, lysozyme, lactoferrin and immunoglobulin (Ig)A, which can inhibit plaque bacteria.

2-Microorganisms in the form of dental plaque: ^(24, 25)

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caries. The dental plaque conclude the acids produced in close contact with the tooth surfaces and prevents them from contact with the cleansing action of saliva.

3-Substrate (diet):^(26, 27)

The role of reiterated carbohydrates, especially the disaccharide sucrose, in the aetiology of dental caries is well established. The total amount consumed as well as the physical form, its oral clearance rate and frequency of consumption are important factors in the aetiology. Vitamins A, D, K, B complex (B6), calcium, phosphorus, fluorine, amino acids such as lysine and fats have an inhibitory effect on dental caries. So, caries requires a susceptible host, cariogenic oral flora and a suitable substrate, which must be present for a sufficient length of time.

Prevention and control of dental caries:

Increase the resistance of the teeth.^(20, 21)

Systemic use of fluoride:

- 1- Fluoridation of water, milk and salt.
- 2- fluoride supplementation in the form of tablets and lozenges.
- 3- consuming a fluoride-rich diet such as tea, fish, etc.

Topical:

- 1-Use of fluoridated toothpaste and mouth wash.
- 2-use of fluoride varnishes (in-office application, longer duration of action, high fluoride content).
- 3-use of casein phosphopeptide-amorphous calcium phosphate (CPP-ACP), which is available as tooth mousse, helps to remineralize the soft initial carious, demineralized areas of the teeth.

Treatment of dental caries

Treatment comprises removal of decay by operative procedures and restoration with appropriate materials such as silver fillings, gold inlays, composite resin, glass ionomer cement, full metal or porcelain crowns, etc. In advanced cases, where the pulp of the tooth is involved, endodontic treatment may be required. Where there is extensive destruction of the tooth structure or when endodontic treatment is not feasible, extraction of the

tooth and replacement by an artificial prosthesis may be required.

Ethical Clearance: The Research Ethical Committee at scientific research by ethical approval of both environmental and health and higher education and scientific research ministries in Iraq

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References

1. Daniels TS, Silverman S, Michalski JP, Greenspan JS, Sylvester RA, Talal N. The oral component of Sjogren's syndrome. *Oral Surg* 1975;39:875-85.
2. Kermiol M, Walsh RF. Dental caries after radiotherapy of the oral regions. *J Am Dent Assoc* 1975;91:838-45.
3. Thean H, Wong ML and Koh H. The dental awareness of nursing home staff in Singapur- a pilot study. *Gerodontology*. 2007; 24: 58-63.
4. Selwitz RH, Ismail AI and Pitts NB. Dental caries. *Lancet*. 2005; 369: 51-59.
5. Nyvad B and Takahashi N. Caries ecology revisited: microbial dynamics and the caries process. *Caries Res*. 2008; 42: 409- 418.
6. Marsch PD and Martin MV. *Oral Microbiology*. 5th edition. Elsevier, 2009; pp 220-222.
7. Tanzer JM, Livingston J and Thompson AM. The microbiology of primary dental caries in humans. *J Dent Educ*. 2001; 65: 1028-1037.
8. Becker MR, Paster BJ, Leys EJ, Moeschberger ML, Kenyon SG and Galvin JL. Molecular analysis of bacterial species associated with childhood caries. *J Clin Microbiol*. 2002; 40: 1001-1009.
9. Aas JA, Griffen AL, Dardis SR, Lee AM, Olsen I and Dewhirst FE. Bacteria of dental caries in primary and permanent teeth in children and young adults. *J Clin Microbiol*. 2008; 46: 1407-1417.
10. Ling Z, Kong J, Jia P, Wei C, Wang Y and Pan Z. Analysis of oral microbiota in children with dental caries by PCR-DGGE and barcoded pyrosequencing. *Microb Ecol*. 2010; 60: 677-690.
11. Davis, D. L., and S. Reisine. 2015. Barriers to dental care for older minority adults. *Special Care in Dentistry* 35(4):182-189.
12. Collee, J.G.; Fraser, A.G.; Marmion, B.P. & Simmons, A. (1996). Mackie & McCartney

practical Medical Microbiology. 14th ed., Churchill Living stone, U.S.A.

13. Daniel, W.W. (1978). Biostatistics a foundation for analysis in health science 2nd ed. Johan Wily and Sons Inc, Canada.
14. Gibbons, R. j.; Socransky, S.S.; DeAraujo, W.C. and Van Houte, J. (1964). Studies of the predominant cultivable microbiota of dental plaque J. Arch. Oral Biol.9:365-370.
15. Socansky, S.S. (1970). Relationship of bacteria to the etiology of periodontal disease. J. Dent. Res. 49(supple 2):203-212.
16. Carlson, J.(1967). Presence of various type of non hemolytic *Streptococci* in dental plaque and in other sites of oral cavity in man. Odontol.Rev.18:55-62.
17. Krass, B.O. (1954). The relationship between Lactobacilli, Candida and *Streptococci* and dental caries .Odontol.Rev.5:241-61.
18. Fitzgerald RJ, Keyes PH. Demonstration of the etiologic role of streptococci in experimental caries in the hamster. *J Am Dent Assoc* 1960;**61**:9–19.
19. Rosen S, Kolstad RA. Dental caries in gnotobiotic rats inoculated with a strain of *Peptostreptococcus intermedius*. *J Dent Res* 1977;**56**:187.
20. Babaahmady KG, Marsh PD, Challacombe SJ, Newman HN. Variations in the predominant cultivable microflora of dental plaque at defined subsites on approximal tooth surfaces in children. *Arch Oral Biol* 1997;**42**:101–11.
21. Haldi J, Wynn W, Bentley KD, Law ML. Dental caries in the albino rat in relation to the chemical composition of the teeth and of the diet. IV. Variations in the Ca/P ratio of the diet induced by changing the calcium content. *J Nutr* 1959;**67**:645–53.
22. Daniels TS, Silverman S, Michalski JP, Greenspan JS, Sylvester RA, Talal N. The oral component of Sjogren’s syndrome. *Oral Surg* 1975;**39**:875–85.
23. Kermiol M, Walsh RF. Dental caries after radiotherapy of the oral regions. *J Am Dent Assoc* 1975;**91**:838–45.
24. Fitzgerald RJ, Keyes PH. Demonstration of the etiologic role of streptococci in experimental caries in the hamster. *J Am Dent Assoc* 1960;**61**:9–19.
25. Rosen S, Kolstad RA. Dental caries in gnotobiotic rats inoculated with a strain of *Peptostreptococcus intermedius*. *J Dent Res* 1977;**56**:187.
26. Burt BA, Eklund Sa, Morgan KJ, Larkin FE, Guire KE, Brown LO, *et al*. The effects of sugar intake and frequency of ingestion on dental caries increment in a three-year longitudinal study. *J Dent Res* 1988;**67**:1422–9.
27. Nizel AE. *Nutrition in preventive dentistry: Sciences and practice*. 2nd ed. Philadelphia: WB Saunders; 1981:417–52.
28. Hicks J, Garcia-Godoy F, Flaitz C. Biological factors in dental caries: Role of remineralization and fluoride in the dynamic process of d demineralization and remineralization (Part 3). *J Clin Pediatr Dent* 2 2004;**28**:203–14.
29. Cai F, Shen P, Morgan MV, Reynolds EC. Remineralization of enamel subsurface lesions in situ by sugar-free lozenges containing casein phosphopeptide–amorphous calcium phosphate. *Aust Dent J* 2003;**48**:240–3.