

**COMPARATIVE CLINICAL TRIAL ON THE EFFECT
OF PROPOLIS EXTRACT AND SODIUM FLUORIDE
MOUTHRINSE ON SALIVARY STREPTOCOCCUS
MUTANS COUNT**

Thesis

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Submitted by

Nihal Safwat Gamal El-Din

Assistant Researcher in the National Research Centre (B.D.S.)

2002

Faculty of Oral and Dental Medicine

Cairo University

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Supervisors

Ass. Prof. Dr. Hala Mohie El-Din Abbas

Professor in Pediatric Dentistry and Dental Public Health
Department
Faculty of Oral and Dental Medicine
Cairo University

Prof. Dr. Ahmed El-Diwany

Professor in Microbial and Natural Chemical Products
Research Department
National Research Centre
(NRC)

Dr. Norhan Abd El-Wahab El-Dokky

Lecturer in Pediatric Dentistry and Dental Public Health
Department
Faculty of Oral and Dental Medicine
Cairo University

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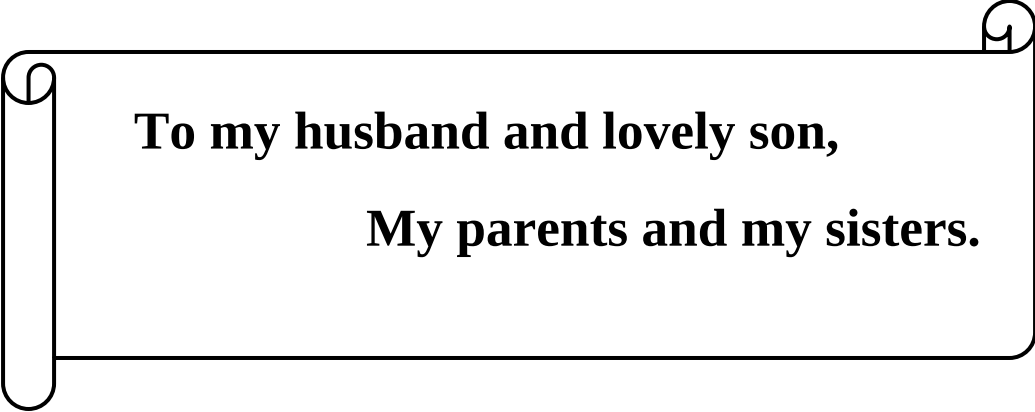
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DEDICATION



**To my husband and lovely son,
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Introduction

Dental caries is the most common oral disease worldwide, it affects the majority of individuals in all age groups during their lifetime. It is regarded as an infectious disease in which changes in the oral environment lead to a pathological shift in the oral biofilm, which then results in localized destruction of the hard tooth tissues **(Petersen et al., 2005 and Selwitz et al., 2007)**.

A group of indispensable factors, the host, bacteria, fermentable carbohydrates and time are necessary for the disease to occur. The inter-relationship between these four key elements may be influenced by a large number of biological and socioeconomic factors thus dental caries is a disease of multifactorial origin **(Selwitz et al., 2007)**.

Dental caries is a transmissible infectious disease in which streptococcus mutans (*S. mutans*) are generally considered to be the main etiological agent. *S. mutans* are the most prevalent caries-associated organisms in humans. As in many infectious diseases, colonization by pathogens is required before the occurrence of the disease **(Berkowitz, 2006)**.

Caries prevention is better than cure, it is regarded as a measure designed to prevent the disease and its clinical symptoms. It also includes treatment for the early signs of initial caries to

prevent further progress and subsequent cavity formation. It is designed to work primarily against the tooth, the bacteria and the fermentable carbohydrates **(Lingström et al., 2003 and Selwitz et al., 2007)**.

Consequently, optimal oral hygiene, reduction in the substrate for bacterial fermentation and frequent exposure to fluoride are all important aspects for caries prevention **(Selwitz et al., 2007)**. The basic methods for preventing dental caries are nearly the same, the certain method may be more suitable for certain population than other population **(Mejàre et al., 2003)**. Thus different preventive strategies will depend on population stratification according to their caries risk assessment **(Seppä, 2001)**.

From these preventive strategies, professional tooth cleaning has been suggested as a method for the prevention of caries and periodontal diseases provided that its time intervals are personalized according to the needs of each patient **(Löe, 2000)**.

Also many types of mouthrinses have been evaluated for their plaque- reducing effectiveness and ability to reduce *S. mutans*, including chlorhexidine, essential oils, triclosan, cetylpyridinium chloride, sanguinarin and various metal ions (tin, zinc, copper).**(Marinho et al., 2003)**.

The most commonly used fluoride-containing mouthrinse is sodium fluoride, the most frequently concentrations used are 0.05% concentration for daily use or sodium fluoride 0.2% concentration for weekly use, the supervised regular use of both types, is associated with a clear reduction in caries increment in both dentitions (**Marinho et al., 2003 and Sköld, 2005**).

Also, Bee Propolis mouthrinse was used to inhibit the growth of *S. mutans* and the activity of bacterium-derived glucosyltransferase (GTF) (**William et al., 2007**).

So the aim of this study is to evaluate and compare the effect of sodium fluoride and Bee Propolis mouthrinses on salivary streptococcus mutans count in a group of Egyptian children.

Review of literature

Dental caries has been the key factor responsible for dental pain and tooth loss in populations all over the world throughout the history of mankind. Despite the preventable nature of dental caries, it still is the main burden of oral diseases in many populations (**Petersen, 2005 and Pitts et al., 2007**). It is not restricted to children and young adults, but also affects almost 100% of the population in the majority of countries and constitutes a particular risk group in the elderly regarding root caries (**Marthaler, 2004 and Petersen et al., 2005**).

In the middle of the last century, a decline in dental caries has been seen among children, adolescents and adults, but there are still a large number of individuals and populations in whom the caries prevalence remains at a high level. Although a reduction in caries prevalence among pre-school children has been found, a tendency towards stagnation in this decline since the end of the 1980s has been reported (**Bader, et al., 2005**).

Dental caries affects 46% of children aged 4 and 80% of 15-years-old. It has been calculated that, every year, around 20-30% of the adult Swedish population develop new carious lesions (**Zickert et al., 2000, Bader, et al., 2005 and Hugoson et al., 2007**).

Dental caries is a process developing as a result of the disturbed balance between the factors comprising the oral environment such as the microorganisms and carbohydrates. Other factors have a protective effect, such as the quantity of the saliva and its buffer capacity (**Axelsson, 2004, Derry and Toumba, 2005, and Featherstone ,2006**).

On the basis of the dynamic equilibrium of these factors **Featherstone (2000)** created the concept of “balanced caries”. According to this concept, the balance between the protective and pathological factors a carious lesion will be protected, stay the same or undergo a reversal (**Nyvad et al.,2004; Pitts and Stamm,2004 and Tabak, 2006**).

Preventive strategies attempt to reduce the risk for disease by influencing its determinants (**Daly et al. 2002**). One of these determinants is bacteria which are organized into a material known as dental plaque which is yellowish coloured film on the surface of the teeth (**McMahon et al.,1993**). Dental plaque is a complex microbial biofilm that is considered as primary etiologic factor in dental caries. Colonization of enamel surfaces by the cariogenic bacterium *S. mutans* is thought to be initiated by attachment to a saliva-derived conditioning film, the acquired enamel pellicle (**Gibbons and Qureshi, 1976**).

The acquired enamel pellicle is formed largely by adsorption of heterogeneous salivary proteins onto dental enamel (hydroxyapatite) and promotes the adhesion of *S. mutans* by specific and nonspecific mechanisms (**Bowden and Hamilton, 1998**). Saliva contains a multitude of proteins that contribute to oral microbial ecology and biofilm formation (**Whittaker et al., 1996**).

Dental biofilm formation after thorough teeth cleaning was positively correlated with both salivary glycoprotein content and *S. mutans* adhesion onto saliva-coated hydroxyapatite surfaces. A higher glycoprotein content forms an “adhesive” conditioning film (acquired pellicle) that promotes a higher rate of bacterial attachment onto hydroxyapatite surfaces in vitro (**Shimotoyodome et al., 2007**).

Studies using phenotyping and/or genotyping methods strongly suggest that the mother is the major primary source of infection for children who carry *S. mutans* and/or *S. sobrinus* strains, and that saliva is the main vehicle by which transfer of *S. mutans* may occur (**Tanzer et al., 2001**). On the other hand other researchers found new genotypes not detected in mothers have also been reported to colonize the oral cavity of children in longitudinal studies, suggesting that additional and extra-familial transmission sometimes occurs, perhaps from other caretakers (**Ersin et al., 2004 and Napimoga et al., 2005**).

The rate and degree of transmission depend on many possible factors, including the degree of infection of the parent, caretaker or playmate, the frequency of contact with the infant and his/her diet and immune status (**Newbrun, 1992**). In addition, early acquisition of *S. mutans* is a major risk factor for early childhood caries and future caries experience (**Grindefford et al., 1996, Roeters, 1997 and Kohler et al., 2003**).

Dental caries has been recognized as the chronic local destruction of teeth by the activity of oral bacteria. Acid produced by the fermentation of dietary carbohydrates causes the initial lesion of dental caries. Since the 19th century, when sucrose became a daily used sweetener by many people worldwide, the increasing prevalence of dental caries had also been noticed (**Wyne et al., 2001**).

Miller (1890) stressed on the importance of acidogenic microbes as caries-inducing organisms, and he and his successors believed that oral lactobacilli was also cariogenic initiator organisms (**Hardie and Whiley , 1997 and Wyne et al., 2001**).

However, it is known that lactobacilli will not induce dental caries, but are considered as secondary invaders in caries lesions (**Hardie and Whiley, 1997**). Instead, some selected species of oral streptococci, *S. mutans* have been demonstrated to be cariogenic in

experimental animals. *S. mutans*, *S. sobrinus* and *S. sanguis* are found in human caries **(Haleem and Khan , 2001).**

Ample evidence indicates an etiological relationship between caries development and *S. mutans* in humans and concluded that bacteria ferment carbohydrates in foods and produce acids, which destroy the hard tissues of teeth, causing dental caries **(Haleem and Khan ,2001).**

A caries lesion is the result of mineral loss due to the activity of the biofilm which accumulates on tooth surfaces. Removing biofilm will halt mineral loss or reverse it in favour of mineral gain, which in turn results in caries arrest . The extent and rate of mineral loss in a caries lesion depend on the composition of bacterial biofilm, the quality and quantity of saliva, the presence of carbohydrate, and concentration of certain minerals as calcium, zinc, potassium and especially fluoride in oral fluids **(Nyvad et al., 1997 and Fejerskov and Kidd, 2004).**

The present understanding about caries initiation and progression indicates a potential for prevention of dental caries by avoiding its risk factors **(Daly et al., 2002 and Fejerskov, 2004).** Caries lesions progress at slower rates than what was previously believed, and caries can be arrested and the affected dental structure undergo remineralization **(Verdonschot et al., 1999 and Lith et al., 2002).**