PREVALENCE OF NURSING BOTTLE CARIES (EARLY CHILDHOOD CARIES) IN YOUNG CHILDREN IN POLOKWANE.

Mohammed Khan-Patel

A research report submitted to the School of Oral Health Sciences, University of the Witwatersrand, Johannesburg in partial fulfillment for the degree Master of Science in Dentistry.

Polokwane 2012

DECLARATION

I, Mohammed Khan-Patel, declare this research as my own work and that it has not been submitted in any other research report or thesis for any other degree.

_____	_____
Mohammed Khan-Patel	Date

DEDICATION

To my children Ismaeel and Safiya, may you learn to reach your full potential in all spheres of your lives.

To my wife Yasmine and my parents whose support has made this research report possible.

ABSTRACT

The aim of this study was to determine the prevalence of Nursing Bottle Caries (Early Childhood Caries) among preschoolers in Polokwane and to determine the relationship between breast and bottle feeding practices to dental caries.

The selection of the preschools was based on a geographical radius to the researcher. A consent rate of 65% was achieved, but only 48% of parents completed the dietary questionnaire.

The study showed that dental caries prevalence among preschoolers was 40% and the prevalence of early childhood caries was 21%. Feeding practices showed 73,7% were predominantly breast fed and 30,5% were predominantly bottle fed. Children in the breast fed group had a caries prevalence of 42,9%, compared to the bottle fed group with a caries prevalence of 31%. Despite this difference, the chi-squared test proved that there was no statistical significance in dental caries between breast fed and bottle fed children. The mean DMFT for the entire sample was 2.24.

In the present study caries prevalence rates were found to be similar to other urban areas of South Africa and other developing countries.

Infant feeding practices, whether breast or bottle in isolation could not be conclusively linked to the aetiology of early childhood caries.

TABLE OF CONTENTS

List of Tables

Table

LIST OF FIGURES

ACKNOWLEDGEMENTS

I would – like to extend my sincere gratitude to:

Professor Sid Setzer, Professor of Paedodontic Dentistry, Department of Restorative Dentistry, University of Witwatersrand, whose patience, and guidance has made this research report possible.

Professor Peter Cleaton-Jones, Professor of Experimental Odontology, Director, MRC/University of Witwatersrand, Dental Research Institute, who assisted with calibration and necessary literature and was always willing to advise.

Prof Joy Shakleton, Head of Department of Paediatric and Restorative Dentistry, University of Witwatersrand, for her encouragement and support.

My dental assistants Idah Setati and Olive Mogale who helped with the field work.

The principals, teachers, parents who gave consent and the children who participated in this research.

Mr Dion van Zyl for his help with the statistical analysis.

Ms Anna-marie Maree for sacrificing many precious hours, typing this research report.

CHAPTER 1

INTRODUCTION AND LITERATURE REVIEW

1.1 DEFINITION AND CLASSIFICATION

The first comprehensive description of dental caries in infants was published by Dr Elias Fass in 1962 and he termed it as “nursing bottle mouth”[1] . He described in his paper that nursing bottle mouth is a caries pattern affecting all the primary upper anterior teeth, and upper and lower primary first molars and lower primary canine teeth.

More recently the term “nursing bottle mouth” has been replaced by the the term Early Childhood Caries (ECC)[2]. Most of the diagnotstic criteria relate to the use of a feeding bottle or prolonged breast feeding (feeding bottle tooth decay, feeding bottle syndrome, nursing caries, nursing bottle mouth). However, all the above definitions of descriptions have not established the actual relationship between dental caries and its full range of aetiological factors.

A conference held in 1994 at the Centers for Disease Control and Prevention in Atlanta, United States of America recommended the use of a less specific term, such as “Early Childhood Caries” (ECC), because it was the consensus of the attendees that the link between bottle habits and caries was not absolute[3].
A workshop, convened by the National Institutes of Health (NIH) proposed that the term early childhood caries (ECC) should be used to describe the presence

of one or more decayed (non-cavitated or cavitated lesions), missing (due to caries), or filled surfaces on any primary tooth in children up to 71 months of age[4,5]. This definition was adopted by the American Association of Paediatric Dentistry[6] and subsequently, by several other researchers[7,8,9].

The aims of the ECC conference, held October 18-19 1997, was to ask scientific leaders in this field to re-examine the current knowledge of ECC through critical reviews of the biological and psychosocial mechanisms, the public health implications and the prevention and research needs[10]. The outcomes of the conference were be used to educate health professionals and the general population on the mechanisms and prevention of ECC, as well as to assist in developing and prioritizing a research agenda for the study of the issues whose answers currently are not known.

Early Childhood Caries can be divided into three types based on clinical appearance[11]:

Type I (mild to moderate) ECC

The existence of isolated carious lesions involving molars and/or incisors. The cause is usually a combination of cariogenic semi-solid food and lack of oral hygiene. The number of affected teeth usually increases as the cariogenic challenge persists. This type of ECC is usually found in children who are 2 to 5 years old.

Type II (moderate to severe) ECC

Labiolingual carious lesions affecting maxillary incisors, with or without molar caries depending on the age of the child and stage of the disease and unaffected mandibular incisors. The cause is associated with inappropriate use of a feeding bottle, and with breast feeding or a combination of both, with or without poor oral hygiene. Poor oral hygiene most probably compounds the cariogenic challenge. This type of ECC could be found soon after the first teeth erupt. Unless controlled, it may proceed to become type III ECC.

Type III (severe) ECC

Carious lesions affecting almost all teeth including lower incisors. This condition is found between the age of 3 to 5 years. The condition is rampant and generally involves tooth surfaces that are usually unaffected by caries e.g. mandibular incisors.

1.2 AETIOLOGY

The aetiology of Early Childhood Caries may be divided into biological and associated risk factors.

1.2.1 The biological risk factors are:

- 1.2.1.1 Dietary Substrate
- 1.2.1.2 Susceptible tooth or host
- 1.2.1.3 Cariogenic bacteria
- 1.2.1.4 Dental plaque

1.2.1.1 Dietary Substrate

Children's eating habits have dramatically changed when compared to previous generations.

The major cariogenic food in the human diet is sugar (sucrose)[12]. A frequent supply of substrate (sucrose) to the plaque, usually given as sugary drinks (juices, from feeding bottle) or in older children, snacks in the form solid-cariogenic foods such as sweets, chocolates, cakes, and biscuits contribute significantly to the development of early childhood caries.

If this loading of the plaque with sugars occurs at bedtime and there is no tooth-brushing, caries can progress rapidly. During bottle-feeding with a sugar- containing drink, the upper incisors bathe in these sugar-containing drinks but the saliva from minor salivary glands in the area of these teeth have only limited remineralization properties, whereas the lower incisors remain largely protected by the tongue during bottle-feeding. Feeding on demand with cariogenic food and liquids is regarded as a co-factor for early childhood caries[13].

The acid production from sucrose metabolism disrupts the balance of the microbial community, favoring the growth of mutans streptococci and lactobacilli[14]; and sucrose is a unique cariogenic carbohydrate because it also serves as a substrate for extracellular glucan synthesis[15]. Glucan polymers are believed to enable mutan streptococci to both adhere firmly to teeth and to inhibit diffusion properties of plaque, or increase plaque porosity resulting in greater acid production adjacent to the tooth

surface[16]. Yet, a systematic literature review of caries risk due to sugar consumption has concluded that the relationship between sugar consumption and caries is weaker in an era of fluoride exposure[17].

1.2.1.1 Susceptible tooth or host

Host risk factors for the development of caries are reduced saliva, immunological factors, presence of enamel defects, characterized mainly by hypoplasia, immature enamel, tooth morphology and genetic characteristics of the tooth (size, surface, depth of fossae and fissures) and crowded / malaligned teeth[18,19]. Enamel defects enhance plaque retention, increase mutan streptococci colonization, and in severe cases, the loss of enamel enables greater susceptibility to tooth demineralization. A strong correlation is found between presence of enamel hypoplasia and high counts of mutan streptococci[20]. Enamel defects in the primary dentition are mostly associated with pre-, peri- or post-natal conditions such as low birth weight, and child's or mother's malnutrition or illness[21]. In the primary dentition, the prevalence of enamel defects is common, ranging from 13-39% in normal full-term infants, to over 63% in those born with very low birth weight [22,23,24].

Enamel hypoplasia of primary incisors in poor, urban populations in the United States of America has been reported to be over 50% leaving these children vulnerable to the caries process[25].

In China children aged 3-5years with enamel hypoplasia had 2.5 times greater risk of developing dental caries than children who did not have such defects[26].

Saliva is the major defense system of the host against caries. It removes foods and bacteria, and provides a buffering action against the acids produced. It also functions as a mineral reservoir for the calcium and phosphate necessary for enamel remineralization. During sleep, the decrease in salivary flow rate reduces its buffering capacity, consequently making the teeth susceptible to caries[18,19].

Enamel is immunologically inactive, therefore the main immune defense against streptococcus mutans is provided largely by salivary secretory immunoglobulin A (IgA) or serum and gingival crevicular fluid. As children become contaminated with oral microorganisms they develop salivary IgA antibodies[18].

After eruption, the newly exposed enamel surfaces undergo the final stages of post-eruptive maturation and hardening by incorporating orally available ions, including fluoride. The tooth is most susceptible to caries in the period immediately after eruption and prior to final maturation[27].

The critical pH value for demineralization varies among individuals, but is in the approximate range of 5.2 to 5.5[28].

Tooth remineralization can occur if the pH of the environment adjacent to the tooth is high due to:

- lack of substrate for bacterial metabolism
- low percentage of cariogenic bacteria in the plaque
- elevated secretion rate of saliva
- strong buffering capacity of saliva
- presence of inorganic ions in saliva
- fluoride
- rapid food clearance times[29]

High frequency sugar consumption enables copious acid production by cariogenic bacteria that are adherent to teeth[30]. The acid can demineralize tooth structure depending on the absolute pH decrease, as well as the length of time that the pH is below the critical pH level[30].

1.2.1.3 Cariogenic microorganisms

The main cariogenic microorganisms are Streptococci (mutans, sobrinus) and Lactobacillus. These pathogens can colonize the tooth surface. When combined with products that contain fermentable carbohydrates, the process of metabolism is initiated by the bacteria, producing acidic end products that ultimately lead to the demineralization of tooth enamel, thus contributing to dental caries[5,30].

The primary caregiver of the infant, usually the mother, has been shown to harbour the reservoir of mutans streptococci. Saliva is the vehicle by

which the transfer occurs. The exact method of transmission is unknown, but it is suspected to be due to close contact of mother (or caregiver) and child and the sharing of food and eating utensils[18,31].

Maternal factors, such as high levels of mutans streptococci, poor oral hygiene, low socioeconomic status, and frequent snacking, contribute to this maternal transfer of mutans streptococci to the child[32].

Preschool children with high colonization levels of mutans streptococci have been shown to have greater caries prevalence, as well as a much greater risk for new lesions than those children with low levels of mutans streptococci[33].

Infants may acquire mutans streptococci via vertical and horizontal transmission.

Vertical Transmission

The major reservoir from which infants acquire mutans streptococci is their mothers[33].

The evidence for this concept comes from several clinical studies that demonstrate that mutans streptococci strains isolated from mothers and their babies exhibit similar or identical bacteriocin profiles[33-35] and identical plasmid or chomosomal DNA patterns[36-40]. Suppression of maternal reservoirs of mutans streptococci clearly showed that infection of the baby could be prevented or delayed[41].

Horizontal Transmission

A study conducted among nursery school children (age 12 –30 months) isolated and genotyped mutans streptococci utilizing primed polymerase chain reaction and restriction fragment – length polymorphism analysis[42]. They reported that many children contained identical genotypes of mutans streptococci strains, which indicates that horizontal transmission may be another vector for acquisition of these organisms[43].

A longitudinal investigation evaluated 786 children at age 1 for caries risk factors (mutans streptococci infections, fluoride exposure, dietary habits, oral hygiene) and re-examined them at 3.5 years of age for the presence of dental caries[44]. The presence of mutans streptococci at age 1 was the most effective predictor for caries at age 3.5[44]. These observations and other published results[45,46] clearly illustrate that early infection with mutans streptococci is a significant risk factor for future development of dental caries lesions.

1.2.1.4 Dental Plaque

Demineralization of the tooth's enamel and dentin is caused by the acids that bacteria, mutans streptococci and Lactobacilli produce. Specifically, the bacteria, acid, food debris, and saliva combine to form a sticky substance called plaque that adheres to teeth. The bacteria and plaque feed from the sugars, producing waste products like lactic acids that cause demineralization or tooth decay. If plaque is not removed

thoroughly and regularly, tooth decay will not only present itself, but will continue to flourish.

The presence of visible plaque and its early accumulation have been related to caries occurrence among children[29,47]. A study found that 91% of the children studied were correctly classified into caries risk groups, based solely on the presence or absence of visible plaque[48].

1.2.2 ASSOCIATED RISK FACTORS

1.2.2.1 BREAST FEEDING

The American Academy of Paediatrics identifies human milk as the ideal nutrient for infants on the basis of the extensive scientific evidence demonstrating that breast feeding and the use of human milk provide multiple health related advantages to infants, mothers and society[49,50].

Breast feeding has many advantages: it provides optimal infant nutrition, immunological protection and minimizes the economic impact to the family.

Prolonged and unrestricted breast feeding has been reported to be a potential risk factor for ECC[51-54].

A study of Tanzanian children, found that nocturnal breast feeding and linear hypoplasia were significant factors for the development of rampant caries[55].

Breast feeding and dental caries among children aged 2 through 5 years was studied previously using data from 1988 to 1994 and no association was found[56].

Although breast feeding was not found to be associated with either an increased or decreased risk of ECC, decreased family income and prenatal maternal smoking, both strongly associated with decreased rates of breast feeding as demonstrated in previous studies, were both found to be independently associated with an increased risk of ECC[57].

Maternal smoking during pregnancy is well recognized to be associated with myriad negative perinatal health outcomes in children[58-61], in addition to early termination of breast feeding[62,63]. In a study among these children in which mothers reported smoking during pregnancy were less likely to be breast fed, but maternal smoking during pregnancy also was independently associated with increased rates of ECC[64].

The World Health Organization (WHO) has recommended that children be breastfed until 24 months of age[65].

A study conducted among 1 – to – 4 year old South African children, reported that children who were breastfed for longer than12 months had a lower level of caries than those bottle fed or bottle and breastfed for less than 12 months[66].

Some researchers have suggested that there is no right time to wean the baby from breast feeding, as long as preventive measures, such as tooth brushing with fluoride toothpaste and reducing the frequency of feedings, are incorporated into the child's care[67].

1.2.2.2 BOTTLE FEEDING

Baby bottles predispose to ECC because their nipple blocks the access of saliva to upper incisors, whereas lower incisors are close to the main saliva glands and are protected from liquid contents by the bottle nipple and the tongue[23,68].

Bottle-feeding, especially nocturnal feeding or, particularly, when children are allowed to sleep with a bottle in their mouth, has been considered cariogenic[51,68,69]. It has been found that children who had been bottle fed had a five times greater risk of having ECC compared to children who were breast fed[70].

Surveys from China, Thailand and Tanzania, where feeding with baby bottles is rare, show high caries rates in primary maxillary incisors, a pattern that is generally assumed to be due to bottle-feeding practices[55,71,72].

Several studies have reported that the majority of United States of America preschool population take, or have taken, a bottle to bed[73,74]. Yet only a small portion of these children actually develop dental caries.

A recent study found that children who consumed beverages containing sucrose in their baby bottle had levels of mutans streptococci (the bacteria most associated with ECC) four times the level those who consumed milk in the bottle[75].

A 1993 survey was conducted to determine the relationship between infant bottle drinking patterns and ECC[76]. The sleeping habits of the child and the contents of the bottle were evaluated. The results indicate that children who fell asleep while feeding from the bottle had significantly more cases of ECC than did children who discarded the bottle before falling asleep. Children who discarded the bottle before falling asleep, however had more cases of ECC than did children who were not given the bottle at all at bedtime.

1.2.2.3 SOCIOECONOMIC FACTORS

Children with ECC tend to come from low-income or lower socioeconomic background[77].

Early Childhood Caries is more common in children from single parent families and those with parents of low educational level, especially of illiterate mothers[78].

A survey found that, in Taiwan mothers who had full time jobs were more likely to have children with ECC than those who had part-time jobs or were housewives[79].

A study related to income group and dental caries confirmed that children with parents in the lowest income group had mean Decayed, Missing, and Filled Teeth (dmft) scores four times as high as children with parents in the highest income group[80].

Socioeconomic status can have a considerable impact on environment, availability of foods, access to proper health care, and education of children[81]. These are all factors that can potentially and negatively affect whether a child develops ECC or not.

The cost of ECC treatment in which a restoration or extraction is required, is extremely high for low income families, often these families are unable to make dental care a priority. Thus, children in families with lower socioeconomic status are more likely to have their dental caries left untreated or are likely to have incomplete dental care.

1.2.2.4 ORAL HYGIENE

The effectiveness of oral hygiene measures in young children depends on the attention and awareness of caregivers.

As young children lack the ability to clean their own teeth effectively, parents are recommended to clean their children's teeth at least until they reach school age[82].

It is generally accepted that the presence of dental plaque is a high risk factor for developing caries in young children [47,48].

Some studies have reported that a child's brushing habit, frequency of brushing and/or the use of fluoride toothpaste are associated with the occurrence and development of dental caries[47,48]. It was found that children who did not have their teeth cleaned at bedtime had a higher risk of developing ECC[83].

1.2.2.5 EDUCATION OF THE PARENTS

The education level of the parents has been shown to be correlated with the occurrence and severity of ECC in their children [52,84]. Lower prevalence of dental caries and lower mean dmft scores have been associated with higher levels of parental education[84].

A Canadian study showed children of parents with university education had less than half the average number of mean Decayed Missing, and Filled Teeth (dmft) scores of children who had parents with only elementary school education [81].

A study on maternal factors of ECC showed that the mother is not only the reservoir of cariogenic bacteria, but that her dental knowledge, behavior, as well as the general care of her child, are also some of the factors that contribute to caries risk [27].

1.2.2.6 GENETIC FACTORS

Higher rates of caries have been reported in families, being passed from generation to generation, and children whose parents or/and siblings have carious lesions are at higher risk of developing caries[69,85].

1.2.2.7 FLUORIDE

The constant maintenance of fluoride in the oral cavity is important for enamel resistance, reducing the amount of minerals lost during demineralization and accelerating remineralization [68].

The effect of fluoride in reducing caries is well established. Fluoride may be delivered in two ways: topically and systematically. The topical modalities mainly include fluoride tooth paste, fluoride varnishes, gels and mouth rinses.

There is convincing evidence for the decay-preventing benefit of tooth-brushing when used with a fluoride-containing tooth-paste[86]. Recent publications have shown that daily tooth-brushing with fluoride toothpaste in 3 to 6 year olds significantly reduces caries incidence[87-89].

The most common method for systematically applied fluoride is fluoridated drinking water shown to be effective in reducing the severity of dental decay in the entire populations. A number of studies have shown that five year old children living in a fluoridated area have approximately 50% less caries than those in a non-flouridated area [90,91]. Water fluoridation has not yet been implemented in South African water supplies.

1.3 PREVALENCE

During the past decades the common consensus from many reports worldwide was that dental caries had declined significantly and was continuing to decline in populations [92,93].

Recent studies globally have reported alarming increases in dental caries in children and adults, on primary as well as permanent teeth[94]. Possible reasons for this scenario globally:

1. Increase of bottle versus fluoridated tap water as well as dietary changes.
2. Change in demographics of the world, influx of immigrants to Europe, the U.S.A and Asia.
3. The large movement of rural people to urban centres who have left farming life for urban labour with the result having negative changes in diet, lifestyle and health [94].

The prevalence rate ranged from 1 -2% in developed countries, in developing countries and within disadvantaged populations of developed countries (immigrants, ethnic minorities) the prevalence is as high as 70%[95].

Table 1.3.1

Dental caries prevalence of some developing countries. Adapted from a review by Bagramian[94]

Age	Prevalence %	Sample Size	Country	Year
2 to 6	59 - 92	993	Phillipines	2003
3 to 5	55	2014	China	2007
6	89.4	178	Taiwan	2006
1 to 6	52.9	981	Taiwan	2006
0 to 5	40	1487	Brazil	2007

Table 1.3.2

The prevalence of dental caries in 4-5 year olds in the provinces of South Africa (van Wyk et al; 2004)[96]	
Age group 4 -5*	
	% Caries
Weighted national mean	**50.6**
Western Cape	77.1
Northern Cape	-
Eastern Cape	58.9
Free State	60.1
KwaZulu Natal	52.4
Gauteng	49.1
North West	41.0
Mpumalanga	40.2
Limpopo	31.1
*Primary dentition	

The caries prevalence differed widely in the different provinces. The Limpopo Province had the lowest, while the Western Cape had the highest prevalance[96].

1.4 PREVENTION

Epidemiological data show that the most effective approach for the control of ECC is based on the prevention and not on restorative treatment[97].

The ideal scenario would be to provide expectant mothers with counseling and guidance, since one of the greatest difficulties related to the control of ECC is

the fact that few parents take their children to the dentist before the age of three years[98].

Despite this, medical appointments are very frequent at this age, which makes sense the presence of a dental professional in the medical office, so that children can also have a dental follow-up from birth.

The prevention of ECC depends on multidisciplinary efforts, involving different health professionals and government sectors responsible for investments in health.

ECC, while not life-threatening, its impact on individuals and communities is considerable, resulting in pain, impairment of function, deleterious influence on the child's growth rate, body weight and ability to thrive, thus reducing quality of life[99].

Preventative measures may be implemented on three levels:

1.4.1 Community based measures

1.4.2 Professional measures

1.4.3 Home-care measures

1.4.1 <u>Community based measures</u>

Community based measures are mainly organized and provided by public health authorities and need be funded and carried out nationwide. Examples of such measures are national educational

programs and water fluoridation, which are shown to be effective in reducing the severity of dental caries in entire populations[100]. In the absence of water fluoridation other modalities such as the administration of salt fluoridation or fluoride supplements (beverages, tablets, drops) should be utilized[68]. Supervised regular use of a fluoride mouth rinse and rinsing at certain intervals resulted in the reduction in caries increment in children[101].

The customary preventive dentistry program for preschool children often involves education of parent regarding ways to prevent dental caries. However, outcomes suggest that educational programs improve knowledge, but only have a temporary effect on plaque levels, and have no discernible effect on caries experience[86]. Counseling is needed to help change parenting practices. This should include listening to the parents and understanding their life circumstances, discussing options to reduce risk and suggesting behavior change strategies[77].

Prevention of ECC, with its focus on educational and counseling programs to alter childrens feeding practices, has not changed greatly from the approach that Dr Fass described. Yet there has been surprisingly little evidence that education reduces the prevalence of this disease[102]. Additional measures to enhance the education so that it translates into preventive behaviors need to be explored. However, in some populations, the lack of preventive behaviors and deeply entrenched feeding practices may be difficult to alter. Perhaps more

studies are needed to explore the effect of frequent professional interventions with agents such as fluoride or antimicrobials that do not rely on patient compliance to reduce caries in preschool children[10]. The focus of such programs is to place the responsibility for caries prevention on the health professional, rather than on the child or parent.

One reason that behavioral interventions have not produced convincing results is that mothers or caregivers are unlikely to administer procedures or practice behaviors which are not shared by the community in which they belong[103]. This is a very important principle that well-intentioned dentists frequently overlook.

1.4.2 PROFESSIONAL MEASURES

It is important for health service providers such as paediatricians, family physicians and nurses who treat very young children to be aware of ECC and it causes, since these health professionals have more frequent contact with very young children than oral health care providers such as oral hygienists and dentists.

Prevention of ECC should begin in the pre and perinatal period. Attitudes and awareness of pregnant women may be deficient and unfavorable toward preventive dental practices[104]. It is critical to provide dental care to pregnant women and women of childbearing age[105].

Early Childhood Caries prevention should be focused on educational programs to improve childrens feeding practices and to reduce levels of mutans streptococci infection[106].

In a study in which mothers with high mutans streptococci were given chlorhexidine gel by mouth guard for 5 minutes a day over 2 weeks[107]. Their children showed delayed colonization of mutans streptococci and less caries than children in a control group[107].

There is sufficient need to seal primary molars, as demonstrated in a Head Start report that found that for those children who had dental caries, 86% had caries of the pits and fissures of the molars[108].

1.4.3 HOME CARE MEASURES

The home-care measures include the development and support of self care habits and emphasize the patient's own responsibility in managing the disease, such as with oral hygiene routines and dietary habits. The effectiveness of oral hygiene measures in young children depends on the attention and awareness of caregivers. Nutritional counseling for the purpose of reducing caries incidence in children is aimed primarily at teaching parents the importance of reducing frequent sugar exposures. Two Swedish studies have tested the effect of preventive education for new mothers on the subsequent caries experience of their children[109,110] . One study provided diet and oral hygiene counseling to the test group at 6, 12 and 24 months of age, as well as

fluoride supplements. This study observed a 65% lower caries experience in the 4-year-old children of mothers who received counseling as compared to the control group[109]. Another study with a similar program found a 42% decrease in caries prevalence after 4 years[110]. Although the results of these studies are encouraging, it is not clear why there have not been more studies to explore the potential of dietary counseling in reducing dental caries in preschool children.

1.5 TREATMENT

A survey conducted by van Wyk and van Wyk[96] found that an average 0f 45.6% of children in South African children (4-5 year olds) require dental treatment.

Table 1.5

Percentage distribution of care needed and the mean number of teeth needing care for dental caries per age group and province in South Africa[96].		
Age group 4-5*		
	% children needing care	**Mean number of teeth**
Weighted national mean	**45.6**	**2.1**
Western Cape	73.2	3.9
Northern Cape		-
Eastern Cape	54.4	2.5
Free State	59.7	2.7
KwaZulu Natal	43.7	2.07
Gauteng	43.0	1.4
North West	33.6	2.0
Mpumalanga	36.9	2.2
Limpopo	30.1	0.8
*Primary Dentition		

The percentage of 4-5 year old children in South Africa who need treatment for dental caries ranges from 30-73 per cent. The greatest need was recorded in the Western Cape (73,2%) and the lowest need was recorded in the Limpopo Province (30,1%).

Treatment of ECC can be accomplished through different types of intervention, depending on the progression of the disease.

Areas of decalcification (early of “white spot lesions”) and hypoplasia can rapidly develop cavitation. If lesions are identified early, the use of anti cariogenic agents may reduce the risk of development and progression of caries.

Parents should be taught how to clean their child's teeth with fluoride toothpaste. The surface of the teeth should be carefully scrubbed after each feeding[11].

Fluoride varnish at one-month intervals, may be a practical option, especially when targeted at children with carious maxillary incisors.

Minimal intervention restorative procedures, such as atraumatic restorative treatment (ART), which do not require the use of local anesthesia or dental hand piece are useful to decrease the trauma to both child and parent. The placement of fluoride-releasing glass ionomer is efficacious in both prevention and therapeutic approaches[111].

When cavitation has occurred, more definitive treatment is required.
Early stages of cavitation can be treated restoratively, while advanced stages will require more complicated measures such as strip crowns for the anterior teeth and stainless steel crowns for the posterior teeth.
Depending on the extent of the lesions pulpectomies or extractions may be indicated. Such management of caries becomes extremely expensive and difficult to treat because such young children lack the ability to cope with extensive restorative treatment.
General anesthesia or sedation procedures would be the suitable treatment modalities for these extensive restorative treatments.

Estimates of the cost of treating ECC in young children are very high. No country in the world can afford to provide through public funds all the restorative treatment needed.

Justification for this research

The impact and prevalence of Early Childhood Caries has been discussed, together with the implications for the costs to the State and family for its treatment. It is a preventable disease, but it is still widespread. Although some studies have been undertaken in South Africa, there have been no previous studies done among preschoolers in Polokwane concerning the prevalence of dental caries.

In addition the link between feeding practices and dental caries has never been confirmed. This study will attempt to establish if any relationship does exist.

Aims and Objectives

The aim of the study was to determine the prevalence of Nursing Bottle Caries (ECC) among 2-5 year old children in Polokwane.

The study objectives were:

1. to evaluate the caries experience of preschool children in Polokwane.
2. to investigate breast and bottle feeding patterns and their relationship to Nursing Bottle Caries (ECC).

CHAPTER 2

MATERIALS AND METHODS

2.1 ETHICAL CONSIDERATIONS

Before progressing with the research ethical clearance was obtained from the University of the Witwatersrand Committee for Research on Human/Medical subjects. (Ethics clearance number M050402 – see appendix 1)

Written informed consent was obtained from the principals / educators of pre-schools which participated in the study, as well as from a parent of each child before any child was examined. (see appendixces 2;3 and 4)

2.2 STUDY SAMPLE

Polokwane is a city located in Limpopo Province of South Africa (see Fig 2.1)
The Polokwane Municipality is located within the Capricorn District, and is the capital of Limpopo Province. The Polokwane Municipality accounts for 3% of the total surface area of Limpopo; however over 10% of the population of Limpopo resides within its boundaries[112]. The Polokwane Municipality is 3775km^2 in size and incorporates about 130 000 homes. 94% of the population is Black/African, 5% White and 1% Coloured and Indian.
The municipality is 23% urbanized and 71% rural[112]. The fluoride concentration in the Polokwane Municipal water supply is 0,1 ppm.

Fig 2.1 Map of Limpopo (Polokwane)

Seven preschools in the city were selected, being within a 10 kilometer radius of the dental practice. The dental practice is located in the central business district of the city. Therefore the sample may not be a truly representative for the whole of the Polokwane urban area.

All seven of the preschools are privately owned and located in the urban/ residential areas of the city.

The fees between the preschools varied between a minimum of R500-00 to a maximum of a R1000-00 per month, per child.

The Republic of South Africa has, for many years, had racial groups separated geographically, politically and socially. The end of these segregatory laws has resulted in an influx of people into urban areas.
It is for these reasons that we now have more inclusive representative urban communities of all ethnic groups i.e. African, Caucasion, Coloured and Asian.

For the purpose of this study the location of the preschool would mean which of these ethnic groups predominates in a particular school.

Of these pre-schools one pre-school refused to participate in the study. Each of the remaining six pre-schools were visited to obtain consent from the headmaster of each school as well as to distribute the dietary questionnaires and consent forms for the parents of children to complete. A suitable date and time was arranged with each pre-school for the examinations.

Table 2.1 Preschools visited

	Name	Ethnicity	Number of children examined
1.	Teddy Bear Academy	Predominantly Caucasian	8
2.	Maria Montesorri Preschool	Mixed	10
3.	Kids Connected	Predominantly Asian	56
4.	Dino Park Preschool	Predominantly African	49
5.	Polokwane Preschool	Predominantly African	60
6.	Puzzles Preschool	Predominantly Caucasian	15
Total			198

2.3 CALIBRATION

In all epidemiological studies of dental caries and controlled clinical trials of caries, preventive agents and procedures depend on the use of standardized diagnostic criteria and the diagnostic reproducibility for their credibility[113]. There was only one examiner who conducted examinations on the preschool children. Therefore there was no need inter examiner reproducibility scores. To train and test for diagnostic reproducibility, 200

extracted permanent teeth were mounted in groups of five in forty plaster blocks with each block and tooth numbered. Caries was diagnosed on each tooth according to World Health Organisation criteria[114]. The test was done in natural light using a mirror only, on two separate occasions. A kappa score of 0.76 was achieved.

2.4 INCLUSION AND EXCLUSION CRITERIA

Inclusion Criteria

1. All children aged between 2-5 years were included in the study.
2. Only children of parents or guardians, who had completed the consent form, thus giving informed consent.
3. All children present on a specific day were examined.

Exclusion Criteria

1. Children younger than 2 years and older than 5 years of age.
2. Those parents/guardians who had not given informed consent.
3. Children who were uncooperative and afraid.

2.1 QUESTIONNAIRE

A structured questionnaire was designed to collect the data. The questionnaire was developed to enquire about breast and bottle-feeding patterns among the preschool children and their relationship to dental caries. The questionnaire that was distributed was in the English language.

Items included in the questionnaire were:

1. If the child was breast or bottle feed and for what period?
2. What were the contents of the feeding bottle?
3. If the child was given a night-time bottle or not? (see appendix 5)

2.6 PILOT STUDY

A pilot study was done at one of the seven preschools. The consent forms and dietary questionnaire were handed out to 8 parents. A trial examinations were conducted. The information from the sample questionnaires was adequate to start the study.

2.7 SAMPLE SIZE

Unfortunately statistical advice was not obtained at the beginning of the study therefore no sample size calculations were done. A small sample size could affect the statistical significance of the results.

2.8 DATA COLLECTION

On the day of examination parental consent forms together with the dietary questionnaires were collected from the educators of preschools. A code number was allocated to each child and the gender and age of the child at the last birthday was written on to the examination sheet.

2.9 EXAMINATION OF THE CHILDREN

The World Health Organisation (WHO) criteria for diagnosing dental caries was used[114]. To ensure optimal infection control disposable mirrors and gloves were used to examine each child.

Most children that were examined were seated on a chair with a high backrest, with the examiner standing behind the chair. Examinations were conducted either outdoors or in classrooms with large windows to ensure good natural light. Some of the younger children were anxious and seemed to be more comfortable being held by the educator during the examination. Those children that were not eager to be examined were not compelled to take part.

The examiner would dictate the status of each individual tooth i.e. decayed, missing or filled to the dental chair-side assistant, who recorded the data on to the examination sheet. (see appendix 6). After each child had been examined a report sheet indicating if the child required further dental treatment was completed and sent to the parents. (see appendix 7)

2.10 DATA MANAGEMENT:

A statistician was consulted to help analyze the data. Data was captured in SPSS for Windows (version 13.0), a statistical data analysis software package.

The data analysis involved various steps:

1.) Construction of one dimensional frequency tables to calculate the prevalence rates of dental and nursing bottle caries for the total sample.

2.) Construction of two dimensional frequency tables to calculate prevalence rates of dental and nursing bottle caries for various sub-sample groups, namely age, gender, bottle feeding practices and breast feeding practices.

3.) Construction of Chi-square tests of independence to investigate the statistical relationship between (i) prevalence of bottle caries with gender, age, bottle feeding practices and breast feeding practices; (ii) prevalence of nursing bottle caries with age and gender. The null-hypothesis was stated as no statistical independence or relationship. The tests were based on a level of confidence of 95,0%. The critical level of significance was $P<0,05$.

4.) Construction of a Logistic Regression model with dependant variable prevalence of dental caries and independent variables, age, gender, bottle feeding practices and breast feeding practices.

5. Construction of a General Linear Model (GLM) with dependent variable dmft-score and independent variables age, gender, bottle feeding practices and breast feeding practices. Due to the skewness of the dmft data a natural log transformation of dmft + 1

was used to approximate normality prior to the GLM analysis. Due to statistical differences in the variations across age groups and the dependant variable, Tamhane post-hoc tests were used for comparative analysis.

CHAPTER 3

RESULTS

3.1 INTRODUCTION:

This chapter reports on the results obtained from statistical analysis to determine the prevalence of dental and nursing bottle caries amongst 2 to 5 years olds, and to ascertain if breast or bottle feeding patterns influence the dental caries rate.

3.2 RESPONSE RATES AND SAMPLE CHARACTERISTICS:

The six preschools visited had a combined enrolment of approximately 300 children. Consent was obtained from 198 of these children. These children were all examined and report sheets were completed and sent to the parents. A consent rate of 65% was achieved.

However of the 198 parents that gave consent, only 95 had adequately completed the dietary questionnaire. It was unfortunate that the remaining 103 children examined could not be included in the study. Therefore for the purpose of this study, the results of only these 95 children were considered complete for further analysis. This revised sample size could affect the statistical significance of the results.

The sample comprised of 47 female children and 48 male children.

Statistical analysis showed no statistically significant effects of gender or race on caries prevalence or severity, so these results were pooled for presentation.

For the purpose of this study a child would be classified as being breast fed if the child was breast fed for a period of 12 months or longer. Similarly a child would be classified as being bottle fed if bottle fed for 12 months or longer. There were 4 children who were exposed to both breast and bottle feeding patterns and these children were considered in both groups.

Due to the incomplete data from the dietary questionnaire information regarding the content of the bottle and the night time bottle could not be analysed as part of the study.

3.3 Results pertaining to the study objectives:

3.3.1. Prevalence of Dental Caries

Table 3.1

		n	%
Dental Caries	***No***	***57***	***60%***
	Yes	***38***	***40.0%***
Total		***95***	***100%***

From the sample of 95 children examined, 38 presented with dental caries. This was 40.0% of the sample size.

Prevalence of Nursing Bottle Caries (ECC)

Table 3.2

Nursing bottle caries	No	75	78.9%
	Yes	20	21.1%
	Total	95	100.0%

Nursing bottle caries was defined as decay on two or more labial or palatal surfaces of the upper deciduous incisors. Twenty children presented with this form of decay which represented 21.1%.

i. Prevalence of Dental and Nursing Bottle Caries (ECC) and the influence of the following variables:

3.3.2.1 Age of children

3.3.2.2 Breast fed children

3.3.2.3 Bottle fed children

3.3.2. Prevalence of Dental and Nursing Bottle Caries (ECC) and the influence of the following variables:

3.3.2.1 Age of children

3.3.2.2 Breast fed children

3.3.2.3 Bottle fed children

3.3..3 Caries severity

3.3.4 Results from Logistic Regression and General Linear Model

3.3.2.1 Prevalence of dental caries by Age

Table 3.3

			Age				
			2	3	4	5	Total
Dental caries	No	n	11	18	19	9	57
		%	91.7%	60.0%	50.0%	60.0%	60.0%
	Yes	y	1	12	19	6	38
		%	8.3%	40.0%	50.0%	40.0%	40.0%
	Total	n	12	30	38	15	95
		%	100.0%	100%	100.0%	100.0%	100.0%

The caries prevalence rate increased with age, from 8.3% among 2 year olds to 40.0% among 3 year olds and 50.0% among 4 years olds.

Prevalence declined slightly to 40.0% among 5 year olds.

Pearson Chi-Square Tests

Table 3.4

		Age
Dental caries	Chi-square	6.597
	df	3
	Sig	.086

A Pearson's Chi-Test was used to test if a statistical relationship exists between caries prevalence rates and age. Evaluation of the results revealed an increase in dental caries, prevalence rates with age. However the Chi-Square Test indicates no statistically significant relationship between the two variables (p=0.086) on a 95% level of confidence (p<0.05).

Prevalence of nursing bottle caries by Age

Table 3.5

			Age				
			2	3	4	5	Total
Nursing bottle caries	No	n	12	24	27	12	75
		%	100.0%	80.0%	71.1%	80.0%	78.9%
	Yes	n	0	6	11	3	20
		%	.0%	20.0%	28.9%	20.0%	21.1%
	Total	n	12	30	38	15	95
		%	100.0%	100.0%	100.0%	100.0%	100.0%

The above table reveals that there were no 2 year olds children with nursing bottle caries and prevalence rate remained almost constant at 20,0%

between 3 year olds to 5 year olds. There is an increase among the 4 year olds, who had a prevalence rate of 28.9%.

Pearson Chi-Square Tests

Table 3.6

		Age
Nursing bottle caries	Chi	4.655
	df	3
	Sig	.199[a]

No statistical significant relationship was found between nursing bottle caries and age (p=0.99).

3.3.3.2. Breast fed Children

Prevalence of dental caries with Breast feeding

Table 3.7

			Breast fed		
			No	Yes	Total
Dental caries	No	n	17	40	57
		%	68.0%	57.1%	60.0%
	Yes	n	8	30	38
		%	32.0%	42.9%	40.0%
	Total	n	25	70	95
		%	100.0%	100.0%	100.0%

There were 4 children who were both equally breast and bottle fed for the 12 months, they were considered in both groups. From the total sample of 95 children 70 of them were predominately breast fed (73.7%). The prevalence rate of dental caries amongst this group was 42.9%.

Pearson Chi-square Tests

Table 3.8

		Breast fed
Dental caries	Chi-square	.905
	df	1
	Sig	.342

No statistical significant relationship was found between dental caries and breast fed activity (p=0.342).

3.3.2.3 Bottle fed Children

Prevalence of dental caries with Bottle feeding

Table 3.9

			Bottle fed		
			No	Yes	Total
Dental caries	No	n	37	20	57
		%	56.1%	69.0%	60.0%
	Yes	n	29	9	9
		%	43.9%	31.0%	31.0%
	Total	n	66	29	29
		%	100.0%	100.0%	100.0%

Children who were exclusively bottle fed were 30.5% of the sample. The caries prevalence rate in this group was 31.0%.

Pearson Chi-Square Tests

Table 3.10

		Bottle fed
Dental caries	Chi-square	1.398
	df	1
	Sig	.237

No statistical significant relationship was found between dental caries and bottle fed activity (p=0.237).

The caries prevalence rate was higher among breast fed group (42.9%) as compared to the bottle fed (31.0%).

As indicated by the chi-squared test, there was no statistically significant difference in dental caries between breast fed and bottle fed children.

3.3.3. Caries severity

Details of dmft, dt, mt and ft scores

Table 3.11

dmft	Mean	2.24
	Standard Deviation	3.69
	Minimum	0
	Maximum	17
	n	95
dt	Mean	2.03
	Standard Deviation	3.57
	Minimum	0
	Maximum	17
	n	95
mt	Mean	.12
	Standard Deviation	.54
	Minimum	0
	Maximum	4
	n	95
ft	Mean	.09
	Standard Deviation	.49
	Minimum	0
	Maximum	3
	N	95

The mean dmft for the entire sample was 2.24. This comprised of the mean dt (2.03), mean mt (0.12) and mean ft (0.9).

Details of dmft, mt and ft scores among different age groups.

Table 3.12

		Age				
		2	3	4	5	Total
dmft	Mean	.67	1.33	3.24	2.80	2.24
	Standard Deviation	2.31	2.26	4.58	3.80	3.69
	Minimum	0	0	0	0	0
	Maximum	8	9	17	11	17
	n	12	30	38	15	95
dt	Mean	.67	1.17	3.08	2.20	2.03
	Standard Deviation	2.31	2.00	4.40	3.95	3.57
	Minimum	0	0	0	0	0
	Maximum	8	9	17	11	17
	n	12	30	38	15	95
mt	Mean	.00	.00	.05	.60	.12
	Standard Deviation	.00	.00	.32	1.18	.54
	Minimum	0	0	0	0	0
	Maximum	0	0	2	4	4
	n	12	30	38	15	95
ft	Mean	.00	.17	.11	.00	.09
	Standard Deviation	.00	.65	.51	.00	.49
	Minimum	0	0	0	0	0
	Maximum	0	3	3	0	3
	N	12	30	38	15	95

The caries severity results show that mean dmft increased from 2 to 4 year olds. The dmft score decreased among the 5 year olds.
At each age group, untreated caries (dt) predominated. The mt and ft scores were low for all age groups and showed no particular pattern.

3.3.4 Results from Logistic Regression and General Linear Model

A Logistic Regression Model with dependent variable prevalence of dental caries and independent variables age, gender, bottle feeding and breast feeding practices, identified age as the only significant predictor of dental caries severity.

Similarly a General Linear Model with dependent variable dmft score and independent variables, age, gender, bottle and breast feeding practices showed significant differences across all ages (see appendix 8).

Results from both the Logistic Regression and General Linear Model identified age as the only indicator of dental caries. The small sample size may not be representative of the entire preschool population in the area. Therefore extrapolation of this data may not be accurate.

3.4 Summary of Results:

1. The overall dental caries prevalence rate was 40.0%.
2. The nursing bottle caries prevalence rate was 21.1%.
3. The breast fed group comprised 73.7% of the sample.
4. The bottle fed group comprised 30.5% of the samples.
5. The caries prevalence rate amongst the breast fed group was 42.9%.
6. The caries prevalence rate amongst the bottle group was 31.0%.
7. The mean dmft was 2.24.
8. None Pearsons Chi-square tests revealed any statistical significance.
9. Logistic Regression and General Linear Model.

CHAPTER 4

DISCUSSION

The sample taken from this study only represented the children attending the selected preschools in the area. It does not include children of this age of the entire community. Therefore extrapolation of the data may be questionable as it is not a representative sample of the area.

4.1 Caries Experience

4.1.1 Caries prevalence

4.1.2 Caries severity

4.2 Questionnaire

4.3 Breast and Bottle feeding patterns

4.1 Caries Experience

4.1.1 Caries prevalence:

The goal set by the Department of Health with regards to dental caries in South Africa for the year 2000 was to ensure that 50.0% or more of the children under the age of 6 years are free of dental caries[115]. The most recent National Childrens Oral Health Survey conducted in South Africa (1999-2002) show 39.7% of 6 year old children are caries free[116]. This figure is below the goal of 50.0% set by the Department of Health for the year 2000. In children

dental caries is more severe in the primary than in the permanent dentition[117].

A systematic review of caries prevalence rates in South Africa between 1947-2002, showed decreasing mean prevalence rates from 88% to 57%[118]. The caries prevalence rate in this study was 40.0%. This figure correlates with the 2002 survey conducted in the Germiston area (Gauteng) among preschoolers in which caries prevalence rates ranged between 35% - 46%[119]. It is also similar to the findings by van Wyk & Louw[96] which found that the caries prevalence rate in the Limpopo province was 31,3%. These caries prevalence rates are also similar to other developing countries such as Brazil (40%) [120] and India (51, 9%) [121].

Caries prevalence did not naturally increase with age. The caries prevalence rate among 2 year olds was 8.3%, 40.0% for 3 year olds, 50.0% for 4 year olds and 40.0% for 5 year olds. The reason for this maybe that the 4 year olds formed the largest part of the sample and the 2 year olds the smallest. Another possible factor could be that at age 4 -5 years all the deciduous teeth have erupted compared to 2 year olds where all the deciduous teeth have not fully erupted.

The results revealed that 21,1% of the preschool children had two or more carious labial or palatal surfaces of the upper deciduous incisors.

4.1.2 Caries Severity

A systematic review of dental caries in 5-6 year old Swazi and South African children from the year 1947 to the year 2002 showed a significant decreases in mean dmft from 6.7 to 3.1[118].

Table 4.2 National Oral Health Survey South Africa 1999/2002:

Table 3: Severity of dental caries across geographical locations 1999/2000[116]

Age	dmft/DMFT	Metro Cape	Port Elizabeth	Durban	Bloemfontein
6	dmft	5.09	3.86	3.42	2.47

In the 1999/2002 National Oral Health Survey among 6 year olds the highest mean DMFT scores for dental caries were recorded in the coastal areas of Metro Cape (5.09); followed by Port Elizabeth (3.86) and Durban (3.42) and the lowest in the interior – Bloemfontein (2.47)[116]. The mean dmft among 5year olds in this study was 2.80. This is similar to other interior regions of the country. This could be attributed to the lower water fluoride levels found in the coastal areas of the country as compared to the interior areas[122].

The fluoride levels in all coastal provinces of South Africa are very low (<0,1ppm), with the Western Cape having the largest number of municipalities with these low fluoride levels[123]. These fluoride levels as similar to the fluoride levels in the Polokwane Municipal water supply which is 0,1ppm.

The untreated caries (dt) component predominated in each age group. This may be due to parents attitudes with regards to:

1. Lack of Oral health knowledge:

   Parents poor dental health history and dental hygiene habits also had associations with the child's poor oral health[124]

2. Complacency/Poor attitude:

   A common and immediate consequence of untreated dental caries on quality of life is dental pain. Childrens dental pain affects regular activities, such as eating, sleeping and playing[125]. Most parents seek only emergency dental treatment.

3. Access to care:

   Dental Clinics may not be conducive to child oral health.

4.2 Questionnaire

Despite a pilot study being conducted to check the feasibility of the questionnaire, of the 198 parents that had given consent for their children to be examined only 95 had completed the questionnaire adequately.

Possible reasons for this:

1) The pilot study was conducted in a preschool with predominantly English speaking parents and children.

2) Parents of preschool children who's native tongue was not English did not understand the dietary questionnaire.

3) Time constraints – parents were unable to complete the dietary questionnaire adequately.

4) Parents unable to remember the feeding patterns of their children.

Consequently information regarding habits such as night-time bottle and bottle contents could not be included in the study.

4.3 Breast and bottle feeding patterns

A 1984 study conducted in South Africa found that almost 92% of both rural and urban Black communities were breast-fed[126]. A more recent study found that 70% of mothers breast fed their children for the first 12 months[127]. This is similar to the figure of 73,7% of the children who were breast fed in this study.

A study conducted in the United States of America found that 20% of the children aged between 6 months and 5 years were put to sleep with a bottle[128]. This figure is similar to the 23% of urban Johannesburg children put to sleep frequently with a bottle[127]. In our study it was found that 30,5% of the preschool children were bottle fed, due to insufficient data from the questionnaire we were unable to determine if the child was put to sleep with a bottle or not.

Comparison to other studies is difficult because very few investigators have based the statistical investigation on groups defined by feeding method. The Camden Study in the United Kingdom reported higher caries prevalence in bottle fed children compared to breast fed children[129]. Two studies among 1 to 4 year old South African children investigated the relationship of breast and bottle feeding patterns to dental caries, indicated that nursing caries (ECC) in children breast fed for a prolonged period, is no less common than in children bottle fed for a similar period of time[130,131].

In this study children who were predominantly breast fed had a caries prevalence rate of 42,9% and those who were predominantly bottle fed had a caries prevalence rate of 31%. This suggests that caries prevalence is higher among the breast fed group compared to the bottle fed group. Considering the small size of the sample and chi-square test this could not account for any statistical significance.

Nursing Caries (ECC) is strongly associated with inappropriate breastfeeding patterns such as prolonged feedings once teeth have erupted[132].. There is also little doubt that a prolonged use of nursing bottle in the susceptible infant will result in rampant caries in the primary dentition[133] Although these authors[132,133] report an association between nocturnal bottle/ breast feeding and ECC, it was not possible in this study to make this association.
Since the aetiology of ECC is multi-factorial, questionnaires should be designed not only to include feeding patterns but factors such

as oral hygiene practices, social class, education and marital status of parents, urban and rural demographics.

Unfortunately all of these factors were not included in the questionnaire of this study.

4.4 Recommendations for future prevention strategies and research

Primary prevention must begin in the pre and perinatal period and should consist of advice on maximizing the nutrition of pregnant women during the last trimester of pregnancy and of infants during the first year of life when the enamel is undergoing maturation[68]. Other primary preventative measures that can be applied during this period include water fluoridation or, in its absence, the administration of fluoride supplements and topical applications of fluoride solutions or varnishes soon after the teeth errupt to increase the resistance of enamel to demineralization[68]. Another possible measure is the application of chemotherapeutic agents to reduce mutans streptococci level in pregnant and nursing mothers to inhibit the transfer of acidogenic micro-organisms from mother to child[68].

Secondary preventative measures should be to educate mothers and other caregivers to recognize the early signs of the condition and to persuade and encourage them to adopt feeding habits and home care procedures to prevent the condition from progressing[68].

Researchers have suggested that health policy should classify ECC as a paediatric, rather than a dental problem[134]. The child welfare and child

health communities may be far more influential in promoting the ECC agenda than the dental communities, which can better offer technical expertise. Programs must be measured at the community level. Policy for ECC must similarly require that programs be community based and able to demonstrate their impact at the community level.

The 1997 conference on ECC[10] caries suggested that strong representations should be made to government agencies and organizations to provide adequate funds for research studies into the following areas of concern:

a. Clarify the nutrition/ hypoplasia/ susceptibility to ECC relationship.
b. Determine the effectiveness and consequences of interfering with the transmission of mutans streptococci from mother to child.
c. Undertake equivalency for superiority trials to determine the effectiveness of antimicrobial and other potential secondary preventive agents.
d. Investigate intervention strategies to control undesirable oral health behaviors of infants and parents or caregivers.
e. Assess behavioral and health factors associated with ECC.
f. Determine the influences of nutrition, including iron-deficiency anemia and exposure to lead on the role of salivary factors in resistance and susceptibility to ECC.

Strong representations should be made to government agencies and organisations to emphasize that ECC is a serious problem with important social, political, educational and economic implications

4.5 Limitations of study

There was only one examiner and this limited the size of study sample. No sample size calculations were done prior to the study. A small sample size could have affected the statistical significance of the results.

1.) Completion of the dietary questionnaire proved to be a difficult task to complete for parents. It may have been that some of the parents did not understand the dietary questionnaire. Only 95 of 198 parents who had given consent had fully completed the dietary questionnaire. Factors such as frequency of breast and bottle feeding, night time bottle feeding and feeding bottle contents were not included in this study due to incomplete data. The validity of the data is limited to the accuracy of the information and the dietary questionnaire.

2.) Seventy one per cent of the Polokwane Municipality consists of rural areas [112]. This study only targeted the urban area. Further research needs to be done to include the large rural areas within the Municipality.

3.) Financial and human resources were limited. The entire cost of the study was borne by the researcher.

CHAPTER 5

CONCLUSION

Early Childhood Caries has a debilitating effect on the development, speech, general health and self-esteem of infants. It is a significant public health issue, the dental manifestation is symptomatic of important underlying maternal and paediatric problems.

The prevalence rates of dental caries found in this study was similar to that of preschools in other developing countries and urban areas of South Africa.

In this study Nursing Caries (ECC) was found to be unrelated to the type of feeding pattern i.e. breast or bottle.

The prevalence of dental caries among preschoolers in Polokwane only represented the urban sector of the population.Surveys of dental caries prevalence and severity conducted among the surrounding rural areas of Polokwane may yield results that are different to this study. This may be due to fact South African communities vary from Third to First World with distinct cultural differences for infant feeding as well as dietary habits, often modified by socioeconomic factors and urbanization further research may be required to highlight these issues and impact on dental caries.

The dimensions of ECC as a public health problem are such that it cannot be solved by oral health workers alone. Steps should be taken to give wide publicity to the disease as a social issue and to publicize how parents as well as health workers can recognize, prevent and control it. Prenatal and postnatal and primary health care clinics would be the place to start sensitizing mothers and begin the prevention of dental caries.

REFERENCES

1) Fass E. Is bottle feeding of milk a factor in dental caries?. J Dent Child 1962; 29: 245-251.

2) Tinanoff N, Kast LM, Corbin SB. Early Childhood Caries: positive beginning. Comm Dent Oral Epidemiol 1998; 26 (Supplement): 117-119.

3) Centres for Disease Control and Prevention (CDCP), conference. Atlanta (GA), September 1994.

4) Drury TF, Horowitz AM, Ismail AI, Maertens MP, Rozier RGS, Selwitz RH. Diagnosing and reporting early childhood caries for research purposes. J Public Health Dent 1999; 59: 192-197.

5) Kaste LM, Drury TF, Horowitz AM, Beltran E. An evaluation of NHANES III estimates of early childhood caries. J Public Health Dent 1999; 59: 198-200.

6) American Association of Pediatric Dentistry (AAPD). Early Childhood Caries: unique challenges and treatment options. Pediatr Dent 2000; 22: 21.

7) Hardison JD, Cecil JC, Mullins MR, White JA, Manz M, Ferretti GA. The 2001 Kentucky childrens oral health survey: Findings for children ages 24 to 59 months and their caregivers. Pediatr Dent 2003; 25: 365-372.

8) Tsai AI, Chen CY, Li LA, Hisang CL, Hsu KH. Risk indicators for early childhood caries in Taiwan. Community Dent Oral Epidemiol 2006; 34: 437-445.

9) Psoter WJ, Zhang H, Pendrys DG, Morse DE, Mayne ST. Classification of dental caries pattern in the primary dentition: a multidimensional scaling analysis. Community Dent Oral Epidermal 2003; 31: 231-238.

10) Tinatoff N. Introduction to the Early Childhood Caries Conference: initial description and current understanding. Community Dent Oral Epidemiol 1998; 26 (supplement 1): 5-7.

11) Wyne AH. Early Childhood caries: nomenclature case definition. Community Dentistry and Oral Epidemiology 1999; 27: 313-315.

12) Ripa LW. Nursing Caries. A comprehensive review. Pediatr Dent 1988; 10: 268-282.

13) Wendt LK, Birkhed D. Dietary habits related to caries development and immigrant status in infants and toddlers living in Sweden. Acta Odontologica Scandinvica 1995; 53: 339-344.

14) Marsh PD. Sugar, fluoride, pH and microbial homeostasis in dental plaque. Proc Fin Dent Soc 1991; 87: 515-525

15) Newbrun E. Sucrose, the arch criminal of dental caries. Odontol Revy 1967; 18: 373-386.

16) Tanzer JM. Microbiology of dental caries. In: Slots J, Taubman MA, editors. Contemporary Oral Microbiology on Immunology. St Louis: Mosby Year Book 1992; 377-424.

17) Burt BA, Pai S. Sugar consumption and caries risk: A systematic review. J Dent Educ 2001; 65: 1017-1023

18) Seow WK. Biological mechanisms of early childhood caries. Community Dent Oral Epidemiol 1998; 26 (1 Supplement): 8-27.

19) Schafer TE, Adair SM. Prevention of dental disease. Pediatric Clin North Am 2000; 47: 1021-42.

20) Li Y, Navia JM, Caufield PW. Colonization by mutans streptococci in the mouths of 3 and 4 yearl old Chinese children with or without enamel hypoplasia. Arch Oral Bio 1994; 39: 1057-1062.

21) Seow WK. Enamel hypoplasia in the primary dentistry: A review. J Dent Child 1991; 58: 441-452.

22) Seow WK, Humphrys C, Tudehope DI. Increased prevalence of developmental defects in low-birth-weight children: A controlled study. Pediatr Dent 1987; 9: 221-225.

23) Grahnen H, Larsson PG. Enamel defects in deciduous dentition of prematurely-born children. Odontol Revy. 1958; 9: 193-204.

24) Nino De Guzman P, Minah GE, Jodrey DB, et al. Recording of enamel hypoplasia and early childhood caries at the D1-D4 levels. J Dent Res 2001; 80: 132.

25) AberanthyJR, Graves RC, Bohannan HM. Development and application of a prediction model for dental caries. Community Dent Oral Epidemiol 1987; 15: 24-28.

26) Li Y, Navia JM, Bian JY. Caries experience in deciduous dentition of rural Chinese children in relation to the presence and absence of enamel hypoplasia. Caries Res 1996; 30: 8-15

27) Milgrom P, Riedy CA, Weinstein P, Tanner ACR, Manibusan L, Bruss J. Dental caries and its relationship to bacteria infection, hypoplasia, diet and oral hygiene in 6 to 36 month old chidren. Community Dent Oral Epidemiol 2000; 28: 295-306.

28) Stephen RM, Intraoral hydrogen-ion concentrations associated with dental caries activity. J Dent Res. 1944; 23: 257.

29) Tinanoff N, Kanellis J, Vargas CM. Current understanding of the epidemiology mechanisms, and prevention of dental caries in preschool children. Pediatr 2002; 24: 543-551.

30) Berkowitz RJ. Cause, treatment and prevention of childhood caries. J Can Dent Assoc 2003; 28: 295-306.

31) Caufield PW, Griffen AL. Dental Caries. An infectious and transmissible disease. Pediatr Clin North Am 2000; 47: 1001-19.

32) Wan AKL, Seow WK, Purdie DM, Bird PS, Walsh LJ, Tudehope DI. Oral colonization of Streptococcus mutans in six-month-old predentate infants. J Dent Res 2001; 80: 2060-2065.

33) Thibodeau EA, O'Sullivan DM. Salivary mutans streptococci and dental caries patterns in pre-school children. Community Dent Oral Epidemiol 1996; 24: 164-168.

34) Berkowitz RJ, Jordan H. Similarity of bacteriocins of Streptococcus mutans from mother and infant. Arch Oral Biol 1975; 20: 725-30.

35) Davey AL, Rogers AH. Multiple types of the bacterium Streptococcus mutans in the human mouth and the intra-family transmission. Arch Oral Biol 1984; 90: 453-60.

36) Berkowitz RJ, Jone P. Mouth-to-mouth transmission the batcterium streptococcus mutans between mother and child. Arch Oral Biol 1985; 30: 377-9.

37) Caufield PW, Wannemuehler Y, Hensen J. Familial clustering of the streptococcus mutans cryptic plasmid strain in a dental clinic population. Infect Immun 1982; 38: 785-7.

38) Caufield PW, Childers NK, et al, Plasmids in Streptococcus mutans; usefulness as epidemiological markers and association with mutacins. In, Hamada S, Michaelek S. Proceedings of an international conference on cellular, molecular clinical aspects of streptococcus mutans. Elsevier Science Publishers, Birmingham, Ala 1985; 217-23

39) Caufield PW, Childers NK. District bacteriocins correlate with different groups of Streptococcus mutans plasmids. Infect Immun 1985; 48: 51-6

40) Caufield PW, Ratanpridakul K. Plasmid-containing strains pf Streptococcus mutans cluster within family and racial cohorts; implication in natural transmission. Infect Immun 1988; 56: 3216-20

41) Kulkasni GV, Chan KH, Sandham HJ. An investigation into the use of restriction endonuclease anylysis int the study of transmission of mutans streptococci. J Dent Res 1989; 68: 1155-61.

42) Kohler B. Preventive measures in mothers influence the establisment of streptococcus mutans in their infants. Arch Oral Biol 1983; 28: 225-31.

43) Mattos-Graner R, Li Y. Genotypic diversity of mutans streptococci in Brazilian nursery children suggests horizontal transmission. J Clin Microbiol 2001; 39(6): 2313-8.

44) Grinderfjord M. Stepwise prediction of dental caries in children up to 3.5 years of age. Caries Res 1995; 30: 356-66.

45) Fujiwara T. Caries prevalence and salivary mutans streptococci in 0-2 years old children of Japan. Commun Dent Oral Epidemiol 1991; 19: 151-4.

46) Roeters RJM. Lactobacilli, mutans streptococci and dental caries: a longitudinal study in 2 year old children up to the age of 5 years. Caries Res 1995; 29: 272-9.

47) Karjalainen S, Söderling E, Sewo'n L, Lapinleimu H, Simell O. A prospective study on sucrose consumption, visible plaque and caries in children from 3 to 6 years of age. Community Dent Oral Epidemiol 2001; 29: 136-42.

48) Alaluusua S, Malmivirta R. Early plaque accumulation, a sign for caries risk in young children. Community Dent Oral Epidemiol 1994; 22: 273-276.

49) Gartner IM, Morton I, Lawrence RA. Breastfeeding and the use of human milk. Pediatrics 2005; 115: 496-506.

50) American Academy of Pediatric Dentistry. Policy on Dietary Recommendations for Infants, Children and Adolescents. Pediatr Dent 2005; 27: 36-37.

51) Azevedo TD, Rezerra AC, de Toledo QA. Feeding habits and severe early childhood caries in Brazilian preschool children. Pediatr Dent 2005; 27: 28-33.

52) Dini EL, Holt RD, Bedi R. Caries and its association with infant feeding and oral health – related behaviors in 3-4 year old Brazilian children. Community Dent Oral Epidemiol 2000; 28: 241-248.

53) Savegh A, Dini EL, Holt RD, Bedi R. Oral Health sociodemographic factors, dietary and oral hygiene practices in Jordanian children. J Dent 2005; 33: 379-388.

54) AL – Dahti AA, Williams SA, Curzon ME. Breast feeding bottle feeding and dental caries in Kuwait a country with low-fluoride levels in the water supply. Community Dent Health 1995; 12: 42-47.

55) Matee MJN, van't Hof M, Maselle SY, Mikx FHM, van Pafenstein Helderman WH. Nursing caries, linear hypoplasia and weaning habits in Tanzanian infants. Community Dent Oral Epidemiol 1994; 22: 289-91.

56) Dve BA, Shenkin JD, Qaden CI, Marshall TA, Levy SM, Kanellis MI. The relationship between healthful eating practices and dental caries in children aged 2-5 years in the United States 1988-1994. J Am Dent Assoc 2004; 135: 55-66.

57) Lawrence RA, Lawrence RM. Breastfeeding: A Guide for the Medical Profession. 6th ed. Philadelphia, PA: Elsevier Mosby; 2005.

58) Windham GC, Hopkins B, Fenster I, Swan SH. Prenatal active or passive tobacco smoke exposure and the risk of preterm delivery or low birth weight. Epidemiology 2000; 11: 427-433.

59) Castles A, Adams EK, Melvin CL, Kelsch C, Boulton ML. Effects of smoking during pregnancy: five meta-analyses. Am J Prev Med 1999; 16: 208-215.

60) Schoendorf KC, Kiely JL. Relationship of sudden infant death syndrome to maternal smoke during and after pregnancy. Pediatrics 1992; 90: 905-908.

61) Anderson ME, Johnson DC, Batal HA. Sudden infant death syndrome and prenatal maternal smoking: rising attributed risk in the back to sleep era. BMC Med. 2005; 3: 4.

62) Letson GW, Rosenberg KD, Wu L. Association between smoking pregnancy and breastfeeding at about 2 weeks of age. J Hum Lact 2002; 18: 368-372.

63) Liu I, Rosenburg KD, Sandoval AP. Breastfeeding duration and perinatal cigarette smoking in a population based cohort. Am. J Public Health 2006; 96: 309-314.

64) Lida H, Auigenger P, Billings RJ, Weitzman M. Association Between Infant Breastfeeding and Early Childhood Caries in the United States. Pediatrics 2007; 120: 944-952

65) World Health Organization (WHO). Global Strategy for infant and young child feeding. Geneva: WHO 2003.

66) Roberts GJ, Cleaton-Jones PE, Fatti LP, Richardson BD, Sinwel RE, Hargreaves JA. et al. Patterns of breast and bottle feeding and their association with dental caries in 1 to 4 year old South African children. Dental caries prevalence and experience. Community Dent Health 1993; 10: 405-413.

67) Weerheijm KL, Uyttendaele – Speybrouck BFM, Euwe HC, Groen HJ. Prolonged demand breastfeeding and nursing caries. Caries Res 1998; 32: 46-50.

68) Davies GN. Early Childhood Caries – a synopsis. Community Dent Oral Epidemiol. 1998; 26. (Supplement 1): 106-116.

69) Twetman S, Garcia-Godoy F, Goepferd SJ. Infant oral health. Dent Clin North Am 2000; 44: 487-505.

70) Du M, Bian Z, Guo L, Holt R, Champion J, Bedi R. Caries patterns and their relationship to infant feeding and socio-economic status in 2-4 year old Chinese children. Int Dent J 2000; 50(6): 385-389.

71) Songpaisan Y, Davies GN. Dental caries experience in the Chiangmai/Lamphun provinces of Thailand. Community Dent Oral Epidemiol 1989; 17: 131-5.

72) Douglass J, Wei Y, Zhang BX, Tinanoff N. Dental caries in preschool Beijing and Connecticut children as described by new caries analysis model. Community Dent Oral Epidemiol 1994; 22: 94-9.

73) Kaste LM, Gift HC. Inappropriate infant bottle feeding. Arch Pediatr Adolesc Med 1995; 149: 786 -91.

74) O'Sullivan DM, Tinanoff N. Social and biological factors contributing to caries of the maxillary anterior teeth. Pediatr Dent. 1993; 15: 41-4.

75) Mohan A, Morse DE, O'Sullivan DM, Tinanoff N. The relationship between bottle usuage/content, age and number of teeth with salivary mutans streptococci colonization in 6- to 24-month – old children. Community Dent Oral Epidemiol 1998; 26: 12-20.

76) Schwartz S, Resivaek R, Micheletti P. A child's sleeping bottle habit as a cause of nursing caries. J Dent Childr 1993; Jan-Feb 22-25.

77) Weinstein P. Public health issues in early childhood caries. Community Dentistry and Oral Epidemiol 1998; 26: 84-90.

78) Maciel SM, Marcenes W, Sheiham A. The relationship between sweetness preference, levels of salivary mutans streptococci and caries experience in Brazilian pre-school children. Int J Paediatr Dent 2001; 11: 123-130.

79) Tsai AI, Chen CY, Li LA, Hsiang CL, Hsu KH. Risk indicators for early childhood caries in Taiwan. Community Dent Oral Epidermiol 2006; 34: 437-445.

80) Tang JM, Altman DC, Robertson D, O'Sullivan DM, Douglas JM, Tinanoff N. Dental caries prevalence and treatment levels in Arizona preschool children. Public Health Reports 1997; 112: 319-329.

81) Ismail AI. Determinants of health in children and the problem of early childhood caries. Pediatric Dentistry 2003; 25: 328-333.

82) Rosenblatt A, Zarzar P. Breast-feeding and early childhood caries: an assessment among Brazilian infants. Int J Pedriatr Dent 2004; 14: 439-445.

83) Harris R, Nicoll AD, Adair PM, Pine CM. Risk factors for dental caries in young children a systematic review of the literature. Community Dent Health 2002; 21(supplement): 57-85.

84) Al-Hosani E, Rugg-Gunn A. Combination of low parental educational attainment and high parental income related to high caries experience in pre-school children in Abu Dhabi. Community, Dent Oral Epidemiol 1998; 26: 31-36.

85) American Association of Pediatric Dentistry (AAPD) Dental care for your baby. Available at http//ww.aapd.org/ publications/ brochures/ babycare.asp (2009)

86) Burt BA, Eklund SA. Dentistry, Dental Practice and the Community. 5th ed. Philadelphia, Pa: WB Saunders Company, 1999.

87) Schwarz E, Lo ECM, Wong MCM. Prevention of early childhood caries – results of a fluoride toothpaste demonstration trial on Chinese preschool children after three years. J Public Health Dent 1998; 58: 12-18.

88) Holtta P, Alaluusua S. Effect of supervised use of a fluoride toothpaste on caries incidence in preschool children. Int J Paediatr Dent 1992; 2: 145-149.

89) Sjögren K, Birkhed D, Rangmar B. Effect of a modified toothpaste technique on approximal caries in preschool children. Swed Dent J. 1995; 110(supplement): 1-10.

90) Shellis RP, Duckworth RM. Studies on the cariostatic mechanisms of fluoride. Int Dent Journal 1994; 44: 263-273.

91) Marinho VC, Higgins JP, Sheiham A, Logan S. Combinations of topical fluoride (toothpastes, mouthrinses, gels, varnishes) versus single fluoride for preventing dental caries in children and adolescents. Cochrane Database System Rev 2004, Comment in Evid Based Dent 2004; 5(2): 38.

92) First International Conference on Declining Caries. J Dent Res 1982; 61(Sp Iss).

93) Second International Conference on Decling Caries. J. Dent Res 1994; 44 (Sp Iss).

94) Bagramain RA, Garcia-Goday F, Volpe AR. The global increase in dental caries. A pending public health crisis. Am J Dent 2009; 22: 3-8.

95) Maupomè G. An introspective qualitative report on dietary patterns and elevated levels of dental decay in a deprived urban population in Northern Mexico. J Dent Childr 1998, 276-285.

96) van Wyk PJ, Louw AJ, du Plessis JR. Caries status and treatment needs in South Africa: Report of the 1999-2002 National Childrens Oral health Survey. SADJ 2004; 59(6): 238-242.

97) dos Santos APP,Soviero VM. Caries prevalence and risk factors among children aged 0-36 months. Pesqui Odontol Bras 2002; 16:3.

98) Horowitz HS. Research issues in early childhood caries. Community Dent Oral Epidemiol 1998; 26 (Supplement): 67-81.

99) American Academy of Pediatric Dentistry. Symposium on the prevention of oral disease in children and adolescents. Chicago III, November 11-12; 2005: Conference papers. Pediatr Dent 2006; 28(2); 96-198.

100) Featherstone JD. The continuum of dental caries, evidence for a dynamic disease process. J. Dent Res 2004; 83 (spec No C): C39-C42.

101) Lopez-Del-Valle L, Velazquez-Quitana Y, Weinstein P, Domoto P., Le Roux B. Early childhood caries an risk factors in rural Puerto Rican children. J Dent Childr 1998; 65(2): 132-135.

102) Bruerd B, Kinney MB, Bothwell E. Preventing baby bottle tooth decay in American Indian and Alaska Native communities: a model for planning. Public Health Rep 1989; 104: 631-40.

103) Wendt LK, Hallonsten AL, Koch G. Oral health in preschool children living in Sweden. Part II – a longitudinal study. Findings at three years of age. Swed Dent J 1992; 16: 41-9.

104) Low W, Tan S, Schwartz S. The effect of severe caries on the quality of life in young children. Am Acad Ped Dent 1999; 21(6): 325-326.

105) Lee C, Rezaiamira N, Jeffcott E, Oberg D, Domoto P, Weinstein P. Teaching parents at WIC Clinics to examine their high caries risk babies. J Dent Childr 1994; 61(506): 347-349.

106) Tinanoff N, O'Sullivan DM. Early childhood caries: overview and recent findings. Pediatr Dent 1997; 19: 12-16.

107) Kohler B, Andreen I, Jonsson B. The effect of caries – preventive measures in mothers on dental caries and the oral presence of the bacteria streptoccus mutans and lactobacilli in their children. Arch Oral Biol 1984; 29: 879-883.

108) Vargas CM, Monajemy N, Khurana P, Tinatoff N. Oral health status of preschool children attending Head Start in Maryland, 2000. Pediatr Dent 2002; 24: 257 -263.

109) Holst K, Kohler L. Preventing dental caries in children. Report of a Swedish program. Devel Med Child Neur 1975; 17: 602-604.

110) Holm AK, Blomquist HK, Grossner GG, Grahnen H, Samuelson G. A comparative study of oral health as related to general health, food habits and socio-economic condition of 4-year old Swedish children. Community Dent Oral Epidemiol 1975; 3: 34-39.

111) Weinstraub JA. Prevention of early childhood caries: a public health perspective. Comm Dent Oral Epidemiol 1998; 26(Supplement 1): 62-68.

112) http://www.Polokwane.gov.za/index/suburb - Accessed on 15 November 2011.

113) Cleaton-Jones P, Hargreaves JA, Fatti LP, Chandler H.D., Grossman ES. Dental Caries diagnosis calibration for clinical field surveys. Caries Res 1989; 23: 195-199.

114) World Health Organization – Oral Health Surveys, Basic methods. 1997 Fourth Edition, Geneva: World Health Organization 1997.

115) Department of Health. In van Wyk PJ (ed). National Oral Health Survey South Africa 1988/89. Pretoria: Department of Health, 2003.

116) Department of Health. In van Wyk PJ (ed). National Oral Health Survey South Africa 1999/2002. Pretoria: Department of Health, 2003.

117) van Wyk PJ, van Wyk C. Oral health in South Africa. Int Dent J 2004; 54: 373-377.

118) Cleaton-Jones PE, Fatti P. Dental Caries in children in South Africa and Swaziland: a systematic review 1991-2007. Int Dent J 2009; 59: 363-368.

119) Green CE. Dental Survey in some Germiston Nursery Schools Continuation of surveillance. Research Report 2004; 1-60.

120) Ferreira N, Bèria JU, Kramer PF, Feldens EG, Ferldens CA. Dental caries in 0-5 year old children in Brazilian children: Prevalence, severity and associated factors. Int J Paediatr Dent 2007; 17: 289-296.

121) National Oral Health Survey and Fluoride Mapping. An Epidemiological study of oral health problems and estimation of fluoride levels in drinking water. Dental Council of India, New Delhi, 2004.

122) Ockerse T. Dental Caries: Clinical and experimental investigations C DSc Thesis J. Pretoria: University of Pretoria; 1947.

123) Grobler SR, Chickte UME, Louw AJ. Fluoride concentrations of drinking water in the nine provinces of South Africa. SADJ 2006; 61(10) 446.

124) Mattila M, Rautava P, Sillan paa' M, Paunio P. Caries in five year-old children and associations with family-related factors. J Dent Res 2000; 70 (3): 875-881.

125) Edelstein BA, Vargas CM, Candelaria D. Characteristics of children visiting emergency rooms at dental schools. Pediatric Dent 2006; 28: 431-437.

126) Richard BD, Sinwel R, Cleaton-Jones P, Granath L, MacKeown JM, Walker A, et al. Dietary habits. In: Cleaton-Jones P, Garanath L, Richardson BD, eds. Dental Caries, Nutrient Intake, Dietary Habits, Anthropetric Status, Oral Hygiene and Salivary Factors and Microbiota in South African Black, Indian and White 4 – 5 year – old Children. Cape Town: South African Medical Research Council (MRC), 1991; ch 4.

127) Mackeown M, Faber M. Urbanization and cariogenic food habits among 4-24 month old black South African children in rural and urban areas. Public Health Nutrition 2002; 5 (6): 719-726.

128) Kaste LM, Gift HC. Inappropriate infant bottle feeding. Status of the Healthy People 2000. Arch.Pediatr Adolosc Med 1995; 149: 786-91..

129) Holt R, Joels D, Winter G. The Camden Study. Brit Dent J 1982; 153: 107-109.

130) Roberts GJ, Cleaton-Jones PE. Patterns of breast and bottle feeding and their association with dental caries in 1 to 4 – year old South African children. Community Dent Health 1994; 11: 38-41.

131) Roberts GJ, Cleaton-Jones PE, Fatti LP, Ricardson BD, Sinwel RE, Hargreaves JA, et al. Patterns of breast and bottle feeding and their association with dental caries in 1 to 4 year old South African children. Community Dent Health 1993, 10: 405-413

132) Goepferd SJ. Infant oral health: a rationale. J Dent Childr 1986, July /Aug: 257-260.

133) Ayhan H. Influencing factors of nursing caries. J Clin Pediatr Dent 1996; 20 (4): 313-315.

134) Edelstein B. Policy issues in early childhood caries. Community Dent Oral Epidemiol 1998; 26 (supplement 1): 104-5

Appendix 1

UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG

Division of the Deputy Registrar (Research)

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL)
R14/49 Khan-Patel

CLEARANCE CERTIFICATE	PROTOCOL NUMBER M050402
PROJECT	Prevalence of Nursing Bottle Caries in Young Children in Polokwane (title change)
INVESTIGATORS	Dr M Khan-Patel
DEPARTMENT	Dental School
DATE CONSIDERED	05.04.29
DECISION OF THE COMMITTEE*	Approved unconditionally / Approved unconditionally

Unless otherwise specified this ethical clearance is valid for 5 years and may be renewed upon application.

DATE 18/02/2011 18/02/2011 18/02/2011 05 Prevalence of Nursing Bottle Prevalence of Nursing Bottle 04 26 CHAIRPERSON .....
(Professor PS Cleaton-Jones)

*Guidelines for written 'informed consent' attached where applicable

cc: Supervisor : Prof D Seizer

.....

DECLARATION OF INVESTIGATOR(S)

To be completed in duplicate and ONE COPY returned to the Secretary at Room 10005, 10th Floor, Senate House, University.
I/We fully understand the conditions under which I am/we are authorized to carry out the above-mentioned research and I/we guarantee to ensure compliance with these conditions. Should any departure to be contemplated from the research procedure as approved I/we undertake to resubmit the protocol to the Committee. I agree to a completion of a yearly progress report.

PLEASE QUOTE THE PROTOCOL NUMBER IN ALL ENQUIRIES

Appendix 2

CONSENT FORM

Dear Principal,

My name is Dr. M. Khan-Patel and I am a student at the Department of Restorative Dentistry at the University of Witwatersrand, Medical School. I am investigating the prevalence of tooth decay among pre-schoolers in Polokwane. This study will determine the extent of the disease among pre-schoolers and shed light on factors that contribute to tooth decay.

I hereby request permission to include your pre-school in this study. Learners between the ages of 1-4 years will be examined using a dental mirror only. On completion of the examination, a simplified treatment plan will be given to each child's parent or guardian to present to his/her dentist. I look forward to meeting with both you and the teacher to schedule a tentative date and time. The entire procedure is expected to take between 2-3hrs. In addition, a discussion and demonstration on proper oral hygiene will be included.

Consent forms will be distributed to the teachers as well as parents/guardians. I would like to emphasize that this study is completely voluntary and not participating carries no penalty. The identity of the school and its participants will only be used to correlate data; the complete anonymity of respondents will be maintained.

If you have any questions, more information may be obtained from me at (015) 295-5016.

If you would like your school to be part of the study, please complete the consent form below.

Thank you in advance for your co-operation.

Dr. M. Khan-Patel

I, _____ _____ _____, as principal of, _____ _____ _____, hereby grant Dr. M. Khan-Patel permission to conduct the study on school premises.

Signature of Principal _____ Date: _____

Appendix 3

CONSENT FORM

Dear Educator,

My name is Dr. M. Khan-Patel and I am a student at the Department of Restorative Dentistry at the University of Witwatersrand, Medical School. I am investigating the prevalence of tooth decay among pre-schoolers in Polokwane. This study will determine the extent of the disease among pre-schoolers and shed light on factors that contribute to tooth decay.

I would highly appreciate it if permission were granted to conduct my study in your classroom. The procedure would entail examining learners for tooth decay, using a dental mirror only. Your assistance in maintaining co-operation from the students during the examination process will ensure the success of the study. Proper oral hygiene care is a life long skill that is important for optimum health. With this in mind, demonstrations on correct oral hygiene procedures will be demonstrated to the learners.

I look forward to meeting with both you and the principal to schedule a date and time that would be convenient. The entire procedure is expected to take between two to three hours.

Please note that this study is completely voluntary and not taking part in it carries no penalty.

Thank you in advance for your co-operation.

Dr. M. Khan-Patel

I, _____, hereby grant Dr. M. Khan-Patel permission to conduct the study in my classroom.

Appendix 4

CONSENT FORM

Dear Parent,

My name is Dr. M. Khan-Patel and I am a student at the Department of Restorative Dentistry at the University of Witwatersrand, Medical School. I am investigating the prevalence of tooth decay among pre-schoolers in Polokwane.

This study will determine the extent of the disease among pre-schoolers and shed light on factors that contribute to tooth decay. It is also my fervent desire to educate kindergarten teachers to ensure that children continue to practice proper oral hygiene.

Children will be examined using a dental mirror only. In addition, a simplified treatment plan will be given to each parent or guardian after examination.

Please note that this study is completely voluntary and not taking part in it carries no penalty. As well, complete anonymity is guaranteed.

If you have any questions, more information may be obtained from me at (015) 295-5016.

If you would like your child to participate in the study, please complete the consent form below, and the attached feeding pattern questionnaire.

Thank you in advance for your co-operation.

Dr. M. Khan-Patel

I, _____, agree to my child participating in the study outlined above.

Signature of Parent: _____ Date: _____

FEEDING PATTERNS

Appendix 5

	Start age	Stop age	Frequency per day
Breast only			

	Start age	Stop age	Frequency per day	Contents – Tick appropriate blocks
Bottle only				**Cow's milk**
				Formula – specify which ones _____ _____
				Rooibos tea – Has sugar been added?
				Juice – Type? _____
				Milo
				Nesquik
				Water
				Other – Specify _____ _____

		Start age	Stop age	Frequency per day	Contents – Tick appropriate blocks
Mixed	Breast				
	Bottle				Cow's milk
					Formula – specify which ones _____
					Rooibos tea – has sugar been added?
					Juice – Type? _____
					Milo
					Nesquik
					Water
					Other – Specify _____

	Start age	Stop age	Contents
Night-time bottle			

Appendix 6

Nursing bottle caries in young children in Polokwane (2006)

1. Patient's name_____ CARD number ☐

2. Case number ☐☐☐☐☐☐

3. Date of examination ☐☐ ☐☐ ☐☐

4. Date of birth ☐☐ ☐☐ ☐☐

5. Age at last birthday ☐

6. Sex: Female(1) Male (2) ☐

Dental caries

Deciduous dentition

55	54	53	52	51	61	62	63	64	65
85	84	83	82	81	71	72	73	74	75

Decayed(D)
Missing(M)
Filled(F)
Unerupted(U)
Labial caries(L)
Secondary caries(E)
Filled and primary caries(P)
Incisor(I)
Palatal(C)
Palatal and labial caries(A)

Number of teeth requiring treatment ☐☐

Appendix 7

TREATMENT PLAN

Name of school :_____
Name of child:_____
Date of Birth:_____

Dear Parent

Thank you for participating in this study. After examining your child, I recommend that:

	Yes	No
1) He/ she requires urgent dental treatment		
2) He/she requires routine 6-monthly dental consultations to maintain oral health		

Please consult your dentist as advised above.

Yours sincerely

DR M. KHAN-PATEL
BDS (WITS)

Logistic Regression

Case Processing Summary

Unweighted Cases[a]		N	Percent
Selected Cases	Included in Analysis	95	100.0
	Missing Cases	0	.0
	Total	95	100.0
Unselected Cases		0	.0
Total		95	100.0

a. If weight is in effect, see classification table for the total number of cases.

Dependent Variable Encoding

Original Value	Internal Value
No	0
Yes	1

Block 1: Method = Backward Stepwise (Conditional)

Omnibus Tests of Model Coefficients

		Chi-square	df	Sig.
Step 1	Step	4.546	4	.337
	Block	4.546	4	.337
	Model	4.546	4	.337
Step 2[a]	Step	-.086	1	.769
	Block	4.460	3	.216
	Model	4.460	3	.216
Step 3[a]	Step	-.109	1	.741
	Block	4.350	2	.114
	Model	4.350	2	.114
Step 4[a]	Step	-1.172	1	.279
	Block	3.178	1	.075
	Model	3.178	1	.075

a. A negative Chi-squares value indicates that the Chi-squares value has decreased from the previous step.

Model Summary

Step	-2 Log likelihood	Cox & Snell R Square	Nagelkerke R Square
1	123.326[a]	.047	.063
2	123.413[a]	.046	.062
3	123.522[a]	.045	.061
4	124.694[a]	.033	.044

a. Estimation terminated at iteration number 4 because parameter estimates changed by less than .001.

Appendix 8

Classification Table[a]

Observed			Predicted		
			Dental caries		Percentage Correct
			No	Yes	
Step 1	Dental caries	No	50	7	87.7
		Yes	35	3	7.9
	Overall Percentage				55.8
Step 2	Dental caries	No	50	7	87.7
		Yes	35	3	7.9
	Overall Percentage				55.8
Step 3	Dental caries	No	50	7	87.7
		Yes	35	3	7.9
	Overall Percentage				55.8
Step 4	Dental caries	No	48	9	84.2
		Yes	32	6	15.8
	Overall Percentage				56.8

a. The cut value is .500

Variables in the Equation

		B	S.E.	Wald	df	Sig.	Exp(B)
Step 1[a]	breast_fed	-.358	1.243	.083	1	.773	.699
	bottle_fed	-.852	1.197	.506	1	.477	.427
	gender	-.152	.438	.121	1	.728	.859
	age	.401	.246	2.648	1	.104	1.493
	Constant	-1.269	1.616	.617	1	.432	.281
Step 2[a]	bottle_fed	-.540	.487	1.229	1	.268	.583
	gender	-.144	.437	.109	1	.741	.866
	age	.403	.247	2.661	1	.103	1.496
	Constant	-1.638	.994	2.716	1	.099	.194
Step 3[a]	bottle_fed	-.513	.480	1.144	1	.285	.599
	age	.412	.245	2.815	1	.093	1.509
	Constant	-1.749	.937	3.485	1	.062	.174
Step 4[a]	age	.424	.243	3.045	1	.081	1.529
	Constant	-1.944	.915	4.510	1	.034	.143

a. Variable(s) entered on step 1: breast_fed, bottle_fed, gender, age.

Variables not in the Equation

			Score	df	Sig.
Step 2[a]	Variables	breast_fed	.084	1	.772
	Overall Statistics		.084	1	.772
Step 3[b]	Variables	breast_fed	.072	1	.788
		gender	.109	1	.741
	Overall Statistics		.193	2	.908
Step 4[c]	Variables	breast_fed	.731	1	.393
		bottle_fed	1.155	1	.283
		gender	.020	1	.886
	Overall Statistics		1.338	3	.720

a. Variable(s) removed on step 2: breast_fed.

b. Variable(s) removed on step 3: gender.

c. Variable(s) removed on step 4: bottle_fed.

Step Summary[a,b]

	Improvement			Model			Correct	
Step	Chi-square	df	Sig.	Chi-square	df	Sig.	Class %	Variable
2	-.086	1	.769	4.460	3	.216	55.8%	OUT: breast_fed
3	-.109	1	.741	4.350	2	.114	55.8%	OUT: gender
4	-1.172	1	.279	3.178	1	.075	56.8%	OUT: bottle_fed

a. No more variables can be deleted from or added to the current model.

b. End block: 1

Result: Age was included as the only predictor variable of dental caries prevalence.

Univariate Analysis of Variance (GLM)

Between-Subjects Factors

		Value Label	N
Gender	1	Female	47
	2	Male	48
Age	2		12
	3		30
	4		38
	5		15
Breast fed mainly (initially)	0	No	25
	1	Yes	70
Bottle fed mainly (initially)	0	No	66
	1	Yes	29

Levene's Test of Equality of Error Variances[a]

Dependent Variable: dmf1_ln

F	df1	df2	Sig.
4.230	18	76	.000

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.

a. Design: Intercept+gender+age+breast_fed+bottle_fed+gender * age+gender * breast_fed+age * breast_fed+gender * age * breast_fed+gender * bottle_fed+age * bottle_fed+gender * age * bottle_fed+breast_fed * bottle_fed+gender * breast_fed * bottle_fed+age * breast_fed * bottle_fed+gender * age * breast_fed * bottle_fed

Appendix 8

Tests of Between-Subjects Effects

Dependent Variable: dmf1_ln

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	21.852[a]	18	1.214	1.636	.072	.279
Intercept	18.395	1	18.395	24.789	.000	.246
gender	.008	1	.008	.011	.915	.000
age	8.261	3	2.754	3.711	.015	.128
breast_fed	.003	1	.003	.004	.951	.000
bottle_fed	1.364	1	1.364	1.838	.179	.024
gender * age	4.004	3	1.335	1.799	.155	.066
gender * breast_fed	.003	1	.003	.004	.947	.000
age * breast_fed	.333	1	.333	.449	.505	.006
gender * age * breast_fed	.000	0	.	.	.	.000
gender * bottle_fed	.131	1	.131	.176	.676	.002
age * bottle_fed	.021	1	.021	.028	.868	.000
gender * age * bottle_fed	.000	0	.	.	.	.000
breast_fed * bottle_fed	.000	0	.	.	.	.000
gender * breast_fed * bottle_fed	.000	0	.	.	.	.000
age * breast_fed * bottle_ fed	.000	0	.	.	.	.000
gender * age * breast_fed * bottle_fed	.000	0	.	.	.	.000
Error	56.397	76	.742			
Total	124.451	95				
Corrected Total	78.249	94				

a. R Squared = .279 (Adjusted R Squared = .109)

Result: Age was identified as the only significant predictor of dental caries severity.

Post Hoc Tests

Age

Multiple Comparisons

Dependent Variable: dmf1_ln

Tamhane

(I) Age	(J) Age	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
2	3	-.35	.228	.590	-1.00	.30
	4	-.72*	.248	.039	-1.42	-.03
	5	-.73	.303	.137	-1.59	.14
3	2	.35	.228	.590	-.30	1.00
	4	-.37	.215	.422	-.96	.21
	5	-.38	.276	.705	-1.17	.42
4	2	.72*	.248	.039	.03	1.42
	3	.37	.215	.422	-.21	.96
	5	.00	.293	1.000	-.83	.83
5	2	.73	.303	.137	-.14	1.59
	3	.38	.276	.705	-.42	1.17
	4	.00	.293	1.000	-.83	.83

Based on observed means.

*. The mean difference is significant at the .05 level.

Post Hoc Tests

Age

Multiple Comparisons

Dependent Variable: dmf1_ln

Tamhane

(I) Age	(J) Age	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
2	3	-.35	.228	.590	-1.00	.30
	4	-.72*	.248	.039	-1.42	-.03
	5	-.73	.303	.137	-1.59	.14
3	2	.35	.228	.590	-.30	1.00
	4	-.37	.215	.422	-.96	.21
	5	-.38	.276	.705	-1.17	.42
4	2	.72*	.248	.039	.03	1.42
	3	.37	.215	.422	-.21	.96
	5	.00	.293	1.000	-.83	.83
5	2	.73	.303	.137	-.14	1.59
	3	.38	.276	.705	-.42	1.17
	4	.00	.293	1.000	-.83	.83

Based on observed means.

*. The mean difference is significant at the .05 level.