5.3. SUMMARY OF FACTORS AFFECTING *STREPTOCOCCUS MUTANS* IN DENTAL PLAQUE

Two factors likely to reduce the presence of mutans streptococci in dental plaque have been considered, the presence of dietary sucrose and the effect of topically applied fluorides.

The relationship between mutans streptococci dental caries prevalence, and available sucrose, continues to be demonstrated. (Weinberger and Wright 1989, Grindefjord *et al.* 1995, Fure 1998, Mattos-Graner et al 1998) The effect of topical fluorides on the presence of mutans streptococci in plaque, was initially demonstrated by the author, (Woods 1971b) and confirmed by Loesche *et al* (1973) and Hamilton (1977)

In addition to a high standard of oral hygiene and a basic systemic exposure to fluorides during tooth development, topical fluorides in their various forms, and the control of dietary sucrose, are likely to be the principal factors that can be employed for individuals to reduce or eliminate established *S. mutans* from dental plaque. In particular, fluoride therapy offers a therapeutic means to control the presence of *S. mutans* in dental plaque.

Recently vertical transmission of mutans streptococci in infants has been demonstrated and it was shown that establishment of mutans streptococci in infants could be prevented by suppression of maternal mutans streptococci with xylotol (Soderling *et al.* 2000). Inhibiting vertical transmission of mutans streptococci from mother to child during a discrete "window of infectivity" has also been reported by Caulfield, Dasanayake, Li, Pan, Hsu, and Hardin (2000).

The development of a "Medical Model" in contrast to the "Surgical Model" for treatment of dental caries is considered in Section 4.6 of this treatise.

A "medical model" for control and treatment of dental caries is a reality, it is a conservative approach for treatment and control of the dental caries and its development has been made possible from understanding the infectious nature of dental caries, knowing the infecting micro-organism or micro-organinms, their natural history, their distribution, and their transmission and establishment in a host (See Section 4.6.).

PART 2

A FIELD STUDY OF *STREPTOCOCCUS MUTANS* AND DENTAL CARIES IN RURAL CHILDREN FOLLOWING REGULAR, SELF APPLIED FLUORIDE.

6. INTRODUCTION TO THE BOOROWA STUDIES

Boorowa is a small, isolated town and community situated on the South West Slopes of New South Wales. It is an inland settlement about 120km north-west of Canberra. The population is approximately 2,000. Boorowa has no dental practice to serve the town and district population, the nearest full time practices are at Yass or Young. The NSW School Dental service provides a limited service to the children of the two Boorowa schools.

In 1970 a community dental programme was established to assist the school children at Boorowa. It consisted of supervised self application of a 10 percent stannous fluoride, zirconium silicate paste. The aim of the programme was to reduce the dental caries experienced by these schoolchildren. Supervision was provided by groups of parents of the children and a dentist. Evaluation of the effectiveness of the scheme was made with the co-operation and assistance of the Department of Preventive Dentistry of the University of Sydney. There was no regular dental practice in Boorowa during the period of the study.

The programme was commenced at a time when there was a high dental caries experience among schoolchildren especially. When the programme was commenced at Boorowa the permanent tooth dental caries experience [D(I)MFT] of children at Boorowa age 8 years was 2.82 (Table 8) and the deciduous tooth caries experience [d(i)mft] was 6.75 (Table 9).

Baseline examinations of the *S. mutans* prevalence in dental plaque and determination of the dental caries status of the school children at Boorowa were conducted at the Boorowa Central School and St. Joseph's Primary School in 1970.

The baseline or initial plaque examination of the children at both Boorowa schools, was made on 1 July 1970. A plaque examination restricted to the children at the Boorowa Central School, was made on 2 December 1970, the results are not included in this report. A complete plaque examination of children at both schools, was made in April 1971 (21 and 24 April). The third plaque examination was conducted on 29 March 1972, the fourth on 2 August 1978, the fifth on 13 June 1984, and the sixth on 8 July 1986.

Following the initial examination of both plaque and dental caries, a regular community programme, called a "Brush-In" and conducted by a dentist, was commenced where school children, supervised by their parents, brushed their teeth with a 10 percent stannous fluoride paste, once each term. At that time there were three terms in each school year. Details of this project and the caries reduction experienced have been reported (Woods,Martin and Barnard 1976, Woods 1976).*

The baseline dental caries examination of children age 5 to 8 years from both Boorowa Primary Schools** were examined for dental caries by two experienced examiners (NDM and PDB) from the Department of Preventive Dentistry University of Sydney. Examinations were conducted at the schools using standardised techniques including standard instruments and light, for determination dental caries indices DMFS.*** Radiographs were not used. The examinations for dental caries were made by the same examiners again in 1972 and 1978. Caries data in 1984 and 1986 were obtained with the co-operation of the School Dental Service which established a school clinic at Boorowa following the 1978 examination.

All the plaque examinations were made by the candidate and plated out for culture within several hours of being obtained.

---

* A copy of this publication is included in Appendix E
** Saint Joseph's Parish School and Boorowa Central School
*** Caries examinations in 1970, 1972 and 1976 were conducted by Professors Noel Martin and Peter Barnard.

7. *STREPTOCOCCUS MUTANS* IN THE DENTAL PLAQUE OF SCHOOL CHILDREN AT BOOROWA, NSW

The role of *S. mutans* in establishing dental caries has been explored in Part 1 of this treatise. It had been reported that a topical application of a fluoride solution to the enamel of teeth relatively free of dental plaque had the potential to reduce the number of *S. mutans* in samples of dental plaque examined (Woods 1971b). It was considered that a regular fluoride application made by children supervised at school could assist these children by reducing the prevalence of *S. mutans* in their dental plaque and lead to a reduction in their dental caries experience. If this was so there could be real benefits for the children at Boorowa, particularly as there was at the time of establishment of the programme no available dental service in the town.

7.1 INTRODUCTION TO THE DENTAL PLAQUE AND *S.MUTANS* STUDIES OF CHILDREN AT BOOROWA NSW.

A programme which could deliver a regular self application of fluoride using a supervised application of 10 percent stannous fluoride in a zirconium silicate toothpaste to the school children at Boorowa was established in late 1970. If the programme of fluoride application were to be successful changes in the prevalence of *S. mutans* in dental plaque should be able to be demonstrated.

The prevalence of *S. mutans* in the dental plaque of children age 5 to 8 years attending the two schools in Boorowa was undertaken as part of the assessment of the dental health programme established at these schools. The children were examined for *S. mutans* in their plaque in 1970 (the baseline study) and subsequently in 1971, 1972, 1976, 1982 and 1986*.

---

* Dental caries experience was also determined during these surveys the plaque and caries examinations being made on consecutive days.

7.2. METHOD - DETERMINATION ON *S. MUTANS* PREVALENCE IN DENTAL PLAQUE

Dental plaque samples were obtained on sterile toothpicks (one for each quadrant of the dentition, from the gingival third of the buccal and labial aspects of the posterior and anterior teeth in the arch), placed in five mL Ringer's Solution and shaken until a homogeneous suspension was obtained*. The samples were taken in mid afternoon and placed on media within three hours. A description of the procedure has been included by Newbrun in his textbook *Cariology* (1989).

A seven millimetre platinum loop was used to place one streak of the undiluted plaque suspension across the surface of the sulphadimidine-fortified Mitis Salivarius agar. Three such streaks could be accommodated on one petrie dish of agar with ease.

After 72 hours aerobic incubation ten random 7mm diameter fields were examined using a low power microscope objective and the total colonies in the ten fields recorded. The results were graded into the following categories:

Grade 1 - no colonies

Grade 2 - from one to seven colonies

Grade 3 - eight colonies or more

On an empirical basis seven colonies per 10, 7mm fields was considered the upper level representative of a transient *S. mutans* being present in the sample of plaque. Values greater than seven indicate a predominance of this type of organism and would suggest that the plaque material had a high concentration of *S. mutans*. These parameters were established earlier and were maintained throughout the study. (Woods 1971a).

---

* The 5 mL bottles contained a porcelain prosthetic molar to assist suspending the sample. Porcelain could withstand the temperatures of sterilization by autoclave.

It is recognized that the field technique method did not conform with procedures employed by other, laboratoyy-based investigators and that the collection, transport and incubation of the samples is likely to have been less sensitive than other procedures, detecting fewer mutans streptococci than may have been present in the plaque examined. It was however possible using this technique to perform tests in the field, to incubate and the score with the resources available. The technique used at the commencement of the study remained unchanged throughout. From clinical experience, the sensitivity and selectivity of the procedure was appropriate for the study and for relating the presence of mutans streptococci to dental caries.

Streptococcus mutans is readily and reliably recognised by its colony form on Mitis Salivarius agar. The appearance of colonies on Mitis Salivarius agar has been described (Krasse 1966):

> *"...the elevation of the colony was highly convex and the edge undulate; the internal structure was finely granular, reminiscent of frosted glass, the colour light bluish-grey in reflected light and grey to dark blue in transmitted light. The size of the colonies after 24 hours incubation was about 0.5mm in diameter. Sometimes polysaccharide formation was observed as a glistening drop on the top of the colony".*

Colonies of *S. mutans* have also been described and illustrated by Shamschula and Charlton (1971) and the sampling of dental plaque and identification of "caries-inducing" streptococci by Jordan, Krasse and Moller (1968).

The technique, developed to permit a relatively large number of plaque samples to be taken and the *S. mutans* status of the plaque determined, was calibrated against a more involved procedure (Woods 1971a).

7.2.1. Calibration of tests

In the calibration technique, the plaque suspension was diluted and equal amounts (0.01mL) were spread on Mitis Salivarius agar (Oxoid), with 1mg per mL sulfadimidine included, and also incorporated within warm (45°C), Tryptone Glucose Yeast agar before the agar solidified (Oxoid Plate Count Agar). The sulfadimidine was added to the Mitis Salivarius Agar to increase selectivity for *S. mutans* (Carlsson 1967).

The inoculated plates were incubated aerobically for 72 hours before they were examined. The colonies grown with the Tryptone Glucose agar were counted and the number per mL of the plaque suspension calculated.

Colonies of *S. mutans* grown on the Mitis Salivarius agar were counted, recognition being on the basis of colony form and biochemical tests for mannitol fermentation, zooglia formation in five per cent sucrose broth, and sulfonamide resistance from representative colonies which were subcultured.

A low power microscope was used for counting and recognising the colonies. The count was expressed per 1,000 colonies grown on the Tryptone Glucose Yeast agar, the latter figures being taken as representative of the total cultivable count. Where no *S. mutans* was detected the minimum number that could have been detected with respect to the dilution was calculated.

7.2.2. Culture and examination of plaque samples taken at Boorowa

Plaque samples collected at Boorowa were transported to Yass in a cool, insulated container where they were plated out immediately and aerobic incubation commenced. The maximum delay in plating out was three hours.

Plaque samples were obtained on sterile pieces of toothpick (one for each quadrant of the dentition, from the gingival third of the buccal and labial aspects of the posterior and anterior teeth in the arch) and placed in 5 mL of Ringer's solution and shaken until a homogeneous suspension was obtained. The samples were all taken in the mid afternoon and placed on media within three hours.

The inoculated plates were incubated aerobically for 72 hours before they were examined. The colonies grown with the Tryptone Glucose Yeast agar were counted and the number per mL of the plaque suspension calculated.

Colonies of *S. mutans* grown on the Mitis Salivarius agar were counted, recognition being on the basis of colony form and biochemical tests* of representative colonies which were subcultured.

A low power microscope was used for counting and recognising the colonies. The count was expressed per 1,000 colonies grown on the Tryptone Glucose Yeast agar, the latter figures being taken as representative of the total cultivable count. Where no *S. mutans* was detected the minimum number that could have been detected with respect to the dilution was calculated. Details of this technique have been reported (Woods 1971a).

7.3. RESULTS OF PLAQUE EXAMINATION

The results of the examinations of the dental plaque of children at Boorowa, age five to eight years, for the period 1970 to 1986, are set out in Tables 6. Table 7 presents a summary of the statistical analysis of the results.

---

* Tests used to identify mutans streptococci were ability to ferment mannitol, zooglia formation in five per cent sucrose broth. Fermentation of mannitol by *S. mutans* was described by Edwardson (1968) Sulfonamide resistance was demonstrated by the isolation media which contained 1mg/mL sulfadimidine.

7.3.1. *Streptococcus mutans* in dental plaque

The results of plaque examinations for *S. mutans* in the dental plaque of children age 5, 6, 7 and 8 years, classified according to the scheme described (Woods 1971a) are set out in Table 6. Data from a limited survey in 1971, of plaque of children age 5, 6, and 7 years, attending schools in Boorowa, is also included in Table 6.

The 1971 survey was made as an interim assessment of *S. mutans* in dental plaque of the infants and primary school children at Boorowa (school grades kindergarten to year 3), following the introduction in 1970 of a dental health programme. There was no caries examination made at this time.

Using the technique and procedure already described (Section 7.2.2.), an initial examination of 125 children at Boorowa was made for *S. mutans* in their dental plaque. In 46 (37 percent) of the children it was not possible to detect *S. mutans* in their dental plaque. At the same time, when examined using the same technique (Woods 1971a), 50 children (40 percent) had *S. mutans* established in their dental plaque and were classified in Grade (Group) 3.

The percentage of children in whom no *S. mutans* was found in their dental plaque (Group 1) increased to 52 percent by the time the 1971 examination was made, and to 57 percent when examined during the years from 1972 to 1986. The lowest percentage (37 percent) of children free of *S. mutans* occurred at the time of the initial, 1970 examination, the lowest percentage (41 percent) of children subsequently was in 1982.

The percentage of children in whom *S. mutans* was established (Group 3) fell following the initial examination in 1970, when it was 40 percent, to 15 percent in 1971 and remained in the range from 11 to 22 percent during the years 1971 to 1986.

Results of plaque examinations for *S. mutans* are set out by age for examinations in 1970, 1971, 1972, 1976, 1982 and 1986 are in Table 6.

Table 6 Boorowa study - Streptococcus mutans in dental plaque of schoolchildren 1970 - 1986; number of children examined and percentages*.

Regular supervised, self applied topical fluoride applications made in each school three times each year 1970 to 1986.

Age and plaque status**	1970 Baseline study	1971	1972	1976	1982	1986
		Regular supervised, self applied topical fluoride applications in each school three times each year 1970 to 1986				
5 years						
Grade 1	17 (40)	28 (50)	22 (48)	12 (57)	8 (40)	11 (44)
Grade 2	10 (23)	19 (34)	16 (35)	8 (38)	8 (40)	7 (28)
Grade 3	16 (37)	9 (16)	8 (17)	1 (5)	4 (20)	7 (28)
6 years						
Grade 1	8 (28)	25 (52)	25 (53)	14 (47)	4 (33)	21 (80)
Grade 2	9 (31)	16 (33)	15 (32)	15 (50)	8 (67)	3 (12)
Grade 3	12 (41)	7 (15)	7 (15)	1 (3)	0 -	2 (8)
7 years						
Grade 1	15 (39)	17 (53)	17 (56)	15 (44)	14 (44)	15 (51)
Grade 2	8 (20)	10 (31)	8 (27)	13 (38)	14 (44)	6 (21)
Grade 3	16 (41)	5 (16)	5 (17)	6 (18)	4 (12)	8 (28)
8 years						
Grade 1	6 (43)	-	1	24 (55)	15 (48)	12 (50)
Grade 2	2 (14)	-	0	15 (34)	10 (32)	6 (25)
Grade 3	6 (43)	-	0	5 (11)	6 (20)	6 (25)
5 to 8 years						
Grade 1	46 (37)	70 (52)	65 (52)	65 (50)	41 (43)	59 (57)
Grade 2	29 (23)	45 (33)	39 (32)	51 (39)	40 (42)	22 (21)
Grade 3	50 (40)	21 (15)	20 (16)	13 (11)	14 (15)	23 (22)
Grades 2+3	79 (63)	66 (48)	59 (48)	64 (50)	54 (59)	45 (43)
TOTAL CHILDREN EXAMINED	125	136	124	129	95	104

* Percentages are in parentheses

** Grade 1 - no S.mutans colonies found in 10, 7mm diam. fields after incubation.

Grade 2 - from 1 to 7 S. mutans colonies found in 10, 7mm diam. fields after incubation.

Grade 3 - 8 or more S. mutans colonies found in 10, 7mm diam. fields after incubation.

7.3.2. Statistical analyses of prevalence of *S. mutans* in dental plaque of Boorowa children aged 5 to 8 years.

A statistical analysis of the changes in the prevalence of *S. mutans* in dental plaque of the children at Boorowa has been made. The analysis tested the effect on the presence of *S. mutans* in dental plaque of a supervised, self applied 10 percent stannous fluoride/zirconium silicate paste, three times annually. Tests were made on the basis of the entire group of children involved in the project and were aimed at assesing the significance of changes in the presence of *S. mutans* in dental plaque following the commencement of the supervised, self applications of stannous fluoride tooth paste.

Data showing the prevalence of *S. mutans* in dental plaque was obtained in 1970, prior to the commencement of the program; this information served as a control against which changes in *S. mutans* were compared. The data classified *S. mutans* in plaque in three "Grades". Grade 1, no *S. mutans* isolated from the sample, Grade 2, the plaque sample indicated a well established and plentiful presence of *S. mutans* and Grade 2 where an occasional*S. mutans* colony was seen.

Control data of the prevalence of *S. mutans* in dental plaque (1970) was used to test data obtained subsequently from the Boorowa survey in 1971, 1972, 1976, 1982 and 1986. The prevalence of *S. mutans* in each of the five examinations from 1971 to 1986 was tested for change. The data was tested for changes in the number of children where *S. mutans* was not isolated (changes in the number of Grade 1 results) and changes in the prevalence of established *S. mutans* in plaque (Grade 3).

The survey data on *S.mutans* in plaque of children at Boorowa which was analysed is summarised in Table 6.

Statistical analysis to determine 'p' values was made using chi squared procedure and results of these comparisons are set out in Table 7.

Analysis of data showing the increased number of children age 5 to 8 years from whom *S. mutans* could not be cultured (Grade 1), supports the conclusion that the introduction of regular, supervised, self applied stannous fluoride tooth paste as a community health program was associated with a significant decrease in the prevalence of *S. mutans*. There was a significant increase in the number of children with no *S. mutans* which could be cultured, with the exception of the 1982 results.

Further analysis of the prevalence of established *S. mutans* (Grade 3) in the dental plaque of the children surveyed, showed a significant reduction in the number of children in all years who had "established" *S. mutans* present (Grade 3).

Examples of the calculations to determine the 'p' values using the chi squared method are set out in Appendix A.

Table 7 Comparison of prevalence of *S. mutans* in dental plaque in children at Boorowa prior to 1970 and participating in regular self-application of zirconium fluoride toothpaste 1971 to 1986.

Comparison of the increase in the number of children where no *S. mutans* was found in dental plaque (Grade 1), 'p' values.

Years compared	'p' value	Comment
1970/1971*	< 0.001	Significant
1970/1972*	< 0.02 > 0.01	Significant
1970/1976	< 0.05 > 0.02	Significant
1970/1982	< 0.50 > 0.30	Not Significant
1970/1986	< 0.001	Significant

Comparison of the decreased prevalence of established *S. mutans* (Grade3) in their dental plaque and 'p' values.

Years compared	'p' value	Comment
1970/1971*	< 0.001	Significant
1970/1972*	< 0.001	Significant
1970/1976	< 0.001	Significant
1970/1982	< 0.001	Significant
1970/1986	< 0.001	Significant

* Comparisons made 1970/1971 and 1970/1972 were restricted to children age 5 to 7 years as there were very few children age 8 included in the surveys made in 1971 and 1972.

Examples of the calculations to determine the 'p' values using the chi squared method are set out in Appendix A.

7.4. DISCUSSION OF *S. MUTANS* PREVALENCE IN DENTAL PLAQUE, 1970 - 1986

The results of examinations of the dental plaque of the children at Boorowa show a reduction in the prevelance of *S. mutans* in the children examined following the commencement of regular supervised use of a 10 percent stannous fluoride toothpaste in each of the three school terms* of the school years 1970 to 1986 (Table 6).

Compared with the baseline study made in 1970 there was a statistically significant reduction in the prevelance of *S. Mutans* in the dental plaque of the children examined. The results of the plaque tests were considered for children age 5 to 8 years as one group.

The results were tested to assess the significance of the increase in children from whom no *S. mutans* could be cultivated (an increase in children with dental plaque in the Grade 1 category). Compared with the baseline survey (1970) there was a significant increase in the numbers of children with no demonstrable plaque *S. mutans* in the years (1971, 1972, 1976 and 1986). The exception was the comparison 1970/1982.

An increase in the number of children from whom no *S. mutans* could be isolated (Grade 1) is complemented by a decrease in the number of children from whom *S. mutans* could be isolated (Grades 2 plus 3) or a decrease in the prevalence of *S. mutans* in dental plaque.

A second statistical analysis made to determine the significance of the decrease in the number of children who had dental plaque with "established" *S. mutans* present (Grade 3).

Compared with the baseline study made in 1970 there was a statistically significant increase in the prevelance of "established" *S. mutans* in the dental plaque of the children examined (Grade 3). Again, the results of the plaque tests were considered for children age 5 to 8 years as one group.

The significance of the decrease in children with "established" *S. mutans* could be cultivated was tested. When compared with the baseline survey (1970) there was a significant decrease in the numbers of children with "established"*S. mutans* in their dental plaque in all the years when surveys were conducted (1971, 1972, 1976, 1982 and 1986).

Both the increase in children free of dental plaque with *S. mutans* present and the decrease in the number of children with "established" *S. mutans* in their dental plaque (Grade 3) are consistent with a decline in dental caries susceptibility. These observations made from the studies of children at Boorowa are supported by the significance of the results of the studies

Caries susceptibility appears from the review presented in Part 1 of this treatise to be associated with the presence of *S. mutans* in dental plaque.

Finally, the studies have illustrated the presence of *S. mutans* in groups or communities of children rather than individual children. The implications of changes in prevalence of *S. mutans* in groups of children on dental caries was tested when the dental caries experience of the children in the surveys was considered.

8. DENTAL CARIES EXPERIENCE OF BOOROWA CHILDREN, 1970 - 1986

The second part of the Boorowa field study is concerned with measuring the change in dental caries experience of the children following the introduction of the regular, supervised self-applied 10 percent stannous fluoride in zirconium silicate toothpaste. Again, the examination in 1970 provided the baseline caries data and the caries experience in the years 1972, 1976, 1982 and 1986 were compared with the baseline data.

The principal index of dental caries used in this evaluation was the percentage of children who had no deciduous tooth caries or the percentage of children with no permanent tooth caries. Dental caries was seen as an infectious disease, either present or absent. The extent or degree of the disease which may ocur may vary in individuals as a result of individual factors which would likely include inhereted morphology of teeth, antibodies present and other immune factors. The index was chosen as a sound indicator of dental caries experience and one which lent itself to ready statistical evaluation. The statistical evaluation was made using the chi squared method, similarly to the method already outlined to test the significance of *S. mutans* in dental plaque.

8.1 INTRODUCTION TO DENTAL CARIES EXPERIENCE OF CHILDREN AT BOOROWA 1970 - 1986

The objective of the dental health programme in Boorowa from 1970 to 1986 was to reduce the dental caries experience of Boorowa children. The means to achieve a reduced dental caries experience was the regular supervised self-application of a 10 per cent stannous fluoride/zirconium silicate toothpaste. It was anticipated that the stannous fluoride paste would reduce the presence of *S. mutans* in the dental plaque of the participating children.

There was sufficient evidence at the time of commencement of the program of a strong association between *S. mutans* and dental caries to encourage the trial. (Woods 1971b)

At the same time as plaque of children age 5 to 8 years at the schools in Boorowa was examined, the children were examined to assess their dental caries experience. While the caries experienced by children aged 5 and 6 years was beyond the control of the programme, the caries potential of their plaque which may not have become evident for 1 to 2 years was of interest.

8.2. DENTAL CARIES EXAMINATIONS 1970 - 1986 MATERIALS AND METHOD

Dental caries examinations of the children made in 1970, 1072 and 1976 by Professors Noel Martin and Peter Barnard from the University of Sydney, Department of Preventive Dentistry, assisted by Ms Barbara Macintosh who recorded the examinations. Examinations made in 1982 and 1986 were made by the NSW School Dental Service.

For examinations made in 1970, 1972 and 1976 equipment and standardised lights were brought from the Department of Preventive Dentistry. Data for 1982 and 1986 was obtained from the records and returns of the School Dental Service in Boorowa under the supervision of and with the cooperation of the Regional Dental Officer, Dr Max Bullus.

8.3. DENTAL CARIES EXAMINATIONS - RESULTS

The results of the dental caries examinations are set out in Tables 8, 9 and 10.

In addition to the tables showing dental caries experience of Boorowa children in the years 1970 to 1986, Australian dental caries experience data from the AIHW Dental Statistics and Research Unit (1992, 1995) and Armfield *et al.* (1999, 2000) has been set out in Tables 15 and 16 in Appendix D.*

---

* A comparison of the dental caries experience of the children at Boorowa with the caries experience of Australian children of similar ages has been made. and a summary is presented in Tables 13 and 14.

Table 8 Boorowa study - permanent tooth dental caries [D(I)MFT] by age 1970-86.

Age	1970	1971*	1972	1976	1982	1986
5 years	0.90	-	0.21	0.00	0.00	0.00
6 years	1.05	-	0.30	0.40	0.00	0.03
7 years	2.17	-	1.32	0.80	0.19	0.25
8 years	2.82	-	1.77	1.30	0.56	0.25

* In 1971 there was no dental caries examination.

Table 9 Boorowa study - deciduous tooth dental caries [d(i)mft] by age 1970-86.

Age	1970	1971*	1972	1976	1982	1986
5 years	4.46	-	3.81	3.40	2.83	3.46
6 years	5.52	-	2.70	4.20	5.06	1.90
7 years	6.50	-	4.28	3.00	3.09	4.04
8 years	6.75	-	5.09	3.30	3.28	1.95

* In 1971 there was no dental caries examination.

Table 10 Boorowa study - Percent caries-free children by age 1970-86 deciduous or permanent dentitions (Total number of children examined in parentheses).

Age	1970	1971*	1972	1976	1982**	1986**
5 years						
Deciduous	30 (17)	-	29 (11)	43 (10)	29 (5/17)	46 (12/26)
Permanent	96 (54)	-	90 (33)	100 (23)	100 (6/6)	100 (6/6)
6 years						
Deciduous	31 (13)	-	41 (21)	28 (9)	18 (3/17)	64 (18/28)
Permanent	57 (24)	-	86 (44)	78 (25)	100 (14/14)	95 (21/22)
7 years						
Deciduous	17 (8)	-	28 (15)	32 (11)	45 (15/33)	38 (10/26)
Permanent	24 (11)	-	49 (26)	74 (25)	87 (27/31)	96 (24/25)
8 years						
Deciduous	7 (3)	-	21 (9)	28 (8)	36 (9/25)	68 (13/19)
Permanent	18 (8)	-	30 (13)	45 (13)	76 (19/25)	81 (13/16)
9 years ***						
Deciduous					11 (1/9)	(1/1)
Permanent					11 (1/9)	(1/1)

* In 1971 there was no dental caries examination.

** Data for 1982 and 1986 caries free children are the numerator and total children examined the denominator of the fraction.

*** Because of few children age 9 years who were examined these results have not been included in the analysis and are recorded only as they were part of the examination conducted.

8.3.1 Statistical analysis of change in dental caries experience in Boorowa children age 6, 7 and 8 years.

The dental caries experience data showing percent dental caries free for children examined in 1972, 1976, 1982, 1986 were tested for a statistically significant difference compared with the 1970 baseline study.

Deciduous teeth and permanent teeth changes in dental caries experience were tested separately for children of ages 6, 7 and 8 years for each of four dental caries surveys 1972 to 1986. The statistical tests were performed using the chi squared method and tests were made on the increase in caries free children in each age group and for each of the four surveys 1972 to 1986 in comparison with the baseline survey of 1970.

The 'p' values for these comparisons and whether or not the changes were significant, are set out in Tables 11 (deciduous teeth) and 12 (permanent teeth)

A second statistical analysis has been made to test the significance of the difference in dental caries experience between children ages 6 plus 7 plus 8 years considered as a group compared with a similar group of Australian children. The years considered were 1976/77, 1982 and 1986. Data for Australian children was obtained from the Commonwealth Department of Health (1986). There was no data available from the Commonwealth Department of Health for the year 1977 which corresponded with the third survey made at Boorowa, the 1977 Boorowa data was compared with the 1976 Australian data.

The grouping of children to include those aged 6, 7 and 8 years in one group was undertaken to provide a similar although not exactly the same, analysis to that made to test the differences in the prevalance of *S. mutans* and reported in section 7.3.2 and summarised in Table 7. In the caries analyses the 5 year age group was not included as there were few permanent teeth present. Results of comparison of dental caries free children in Boorowa and Australia are set out in Table 15.

Among Boorowa children the increase in the percentage of children with caries free deciduous dentitions when compared with the baseline study in 1970, was significant in all ages studied in 1986, that is in children aged 6, 7 or 8 years. In 1982 only children aged 7 or 8 years experienced a significant increase in the percentage with caries free deciduous dentitions. The statistical results are summarised in Table 11.

A significant increase in the percentage of children with caries free permanent dentitions in the survey conducted in 1972, 1976, 1982 and 1986, when compared with the baseline study made in 1970 was established. The group age 8 years in 1972 was the one exception to the otherwise general, significant increase in children with caries free permanent dentitions. The statistical results are summarised in Table 12.

The permanent dentition dental caries experience of the children in the field study at Boorowa, when compared with Australian children was significantly less. Using data from the Commonwealth Department of Health (1987), it was possible to test the difference between Australian and Boorowa childrens' dental caries experience. Again the percentage of caries free children was employed as an index of dental caries experience and the chi squared test was employed. The comparison was restricted to the permanent teeth and the children were considered as a single group age 6 to 8 years. The total number of Australian children was surveyed was obtained from *Dental health of children in Australia 1977-1986* (Commonwealth Department of Health 1987). The data for the Boorowa children was taken from Table 10.

Comparing Boorowa and Australian children as a group aged 6 to 8 years, there was a significantly greater percentage of Boorowa children who had caries free permanent dentitions in the surveys made in 1976 and 1986. (Table 13

Boorowa children compared by age, 6, 7 or 8 years, with Australian children had a significantly greater percentage with caries free deciduous dentitions children age 8 years in the surveys made in 1982 and 1986. (Table 14)

There were no significant differences in the percentage of children with caries free deciduous dentitions in ages 6 and 7 years in the surveys made in 1976, 1982 and 1986 or in those age 8 years in the 1976 survey. The results of the statistical analysis are set out in Table 14.

There was a significant difference in the permanent dentition caries experience of children age 6 to 8 years in the Boorowa study when compared with Australian children for the years 1976/77 and 1986. A similar comparison of the caries free permanent dentitions of the Boorowa and Australian children age 6 to 8 years in 1982 showed no significant difference between the two groups. These statistical comparisons and 'p' values are summarised in Table 13.*,**

---

* The 1976 Boorowa data was compared with the 1977 Australian data.

** It should be noted that in the absence of data from *Dental health of children in Australia 1977-1986* (Commonwealth Department of Health 1987) for Australian children in 1976 and data for 1977 was employed for the comparison with Boorowa children in the 1976 survey. The number of Australian schoolchildren examined in 1982 was not available and a notional figure was used for the calculation. The statistical evaluation was made using the chi squared system as have the other statistical evaluations of dental caries in this treatise.

Table 11. Boorowa study - Increase in percent caries-free deciduous dentitions, aged 6 to 8 years from the baseline 1970 to 1972 - 1986; 'p' values and significance.

Age 6 years Deciduous teeth	1970 (Baseline)	1972	1976	1982	1986
Percent caries free	31%	41%	28%*	18%*	64%
'p' value of change		>0.70	>0.80	>0.30	<0.01
Significance (S - significant NS - not significant)	-	NS	NS	NS	S

* Decrease in percent caries free children 1976 and 1982.

Age 7 years Deciduous teeth	1970 (Baseline)	1972	1976	1982	1986
Percent caries free	17%	28%	32%	45%	38%
'p' value of change		>0.70	>0.05	<0.001	<0.05
Significance (S - significant NS - not significant)	-	NS	NS	S	S

Age 8 years Deciduous teeth	1970 (Baseline)	1972	1976	1982	1986
Percent caries free	7%	21%	28%	36%	68%
'p' value of change		>0.70	>0.05	<0.001	<0.05
Significance (S - significant NS - not significant)	-	NS	NS	S	S

Table 12. Boorowa study - Increase in percent caries-free permanent teeth, aged 6 to 8 years from 1970 to 1972 - 1986, 'p' values and significance.

Age 6 years Permanent teeth	1970 (Baseline)	1972	1976	1982	1986
Percent caries free	57%	86%	78%	100%	95%
'p' value of change		<0.01	<0.05	<0.01	<0.001
Significance (S - significant NS - not significant)	-	S	S	S	S

Age 7 years Permanent teeth	1970 (Baseline)	1972	1976	1982	1986
Percent caries free	24%	49%	74%	87%	96%
'p' value of change		<0.02	<0.001	<0.001	<0.001
Significance (S - significant NS - not significant)	-	S	S	S	S

Age 8 years Permanent teeth	1970 (Baseline)	1972	1976	1982	1986
Percent caries free	18%	30%	45%	76%	81%
'p' value of change		>0.10	<0.02	<0.001	<0.001
Significance (S - significant NS - not significant)	-	NS	S	S	S

TABLE 13 Statistical summary of comparison of Boorowa and Australian children with caries-free permanent dentitions, combined ages 6, 7 and 8 years, 1976/77, 1982, 1986

	1976/77	1982	1986
Australian children			
Total children	103500	311322	122220
Percent caries free	55.6%	75.6%	82.3%
Boorowa children			
Total children	63	60	58
Percent caries free	66%	86%	92%
Comparison of Australian and Boorowa children with caries free permanent dentitions			
'p' value (range)	$p < 0.05$ > 0.02	$p > 0.05$ $p < 0.10$	$p < 0.05$ > 0.02
Significance	YES	NO	YES

TABLE 14 Statistical comparison of percentage of children with caries free deciduous dentitions, Australia and Boorowa, ages 6, 7 and 8 years.

	1976-77	1982	1986
Age 6 years			
Australian children			
Total in group	36504	38500	41917
Percent caries free	34%	43%	50%
Boorowa children			
Total in group	9	17	28
Percent caries free	28%	18%	64%
Significance 'p' value	>0.05	>0.05	>0.05
Significance	NO	NO	NO
Age 7 years			
Australian children			
Total in group	35808	38850	41917
Percent caries free	27%	36%	43%
Boorowa children			
Total in group	11	33	26
Percent caries free	32%	45%	38%
Significance 'p' value	>.0.05	>0.05	>0.05
Significance	NO	NO	NO
Age 8 years			
Australian children			
Total in group	33188	37700	38886
Percent caries free	23%	31%	39%
Boorowa children			
Total in group	8	25	19
Percent caries free	24%	36%	68%
Significance 'p' value	>0.05	<0.001	<0.02
Significance	NO	YES	YES

8.4. DISCUSSION OF REDUCTION IN DENTAL CARIES EXPERIENCE

The index of dental caries employed to assess changes in dental caries experience was the increase in the percentage of caries free children. This index was used as it is a measure of those children who had experienced dental caries; using this index there is no attempt to quantify the degree of dental caries experienced. In the present climate of very low dental caries experience of Australian children it appears more appropriate to consider how many children experience the disease than to consider the degree of dental caries experienced. Like other infectious diseases the degree of dental caries experienced may reflect other parameters of susceptibility, for instance inherited factors related to dental anatomy or acquired factors such as development of immunity.

Comparing the percentage caries free deciduous dentitions of children by age with deciduous teeth in the surveys following the introduction of the dental health programme - that is comparing the data from the surveys of 1972, 1976, 1982 and 1986 with the 1970 baseline data - in 1986 all age groups 6 to 8 years exhibited a significant increase in the percentage who were caries free. Children age 7 and 8 years examined in the 1982 survey also showed a significant increase in the percentage who were caries free (Table 11).

It should be appreciated that children entering the programme when they commenced school at age 5 to 6 years had completely erupted deciduous dentitions and many are likely to have had established dental caries of deciduous teeth.

At age 5 and 6 years in the baseline survey of 1970, 70 and 69 percent of children respectively, had experienced dental caries of their deciduous dentitions (Table 10).

In the surveys from 1972 to 1986, 71 percent to 46 percent of children age 5 and 6 years, experienced deciduous dentition dental caries. The d(i)mft index for these ages in 1970 was 4.46 and 5.52 respectively (Table 9).

Many children aged 5 years entering the programme are likely to have had established dental caries in their deciduous dentition. It could be argued that considering any established dental caries of the deciduous dentition of these children aged 5 years, the deciduous dental caries reduction was delayed but apparent in the later surveys of 1982 and clearly established in the survey of 1986 (Table 11).

The permanent dentition at age 5 years, unlike the deciduous dention, would be just commencing eruption with the appearance of the mandibular and possibly maxillary incisor teeth. The first permanent molars would in most cases, be unerupted. Unlike the deciduosus teeth, almost all permanent teeth would have been exposed to the self applied topical fluorides of the programme very soon after eruption and prior to any caries initiation.

In the baseline study of 1970, dental caries was present in 4 percent of permanent dentitions of children aged 5, at Boorowa (Table 10), the D(I)MFT being 0.90 (Table 8). Dental caries was present in 70 percent of deciduous dentitions of children aged 5 (Table 10) and the d(i)mft was 4.46 (Table 9) in the 1970 baseline study. The dental caries established in the permanent dentition at the time of commencing school and the dental health programme was considerably less than that established in the deciduous dentition.

Unlike the deciduous dentitions the increase in children with caries free permanent dentitions became evident relatively soon after children were introduced to the dental health programme. There were significant increases in the percentage of caries free permanent dentitions for all ages, 6 7 and 8 years, in all surveys 1972 to 1986 with the exception of children age 8 years in 1972 (Table 12). The progressive increase in the percentage caries free permanent dentitions of children age 7 and 8 years in the four surveys conducted 1972 to 1986 should be noted.

With the exception of children age 8 years in the 1972 survey, there were significant increases in the percentage of children with caries free permanent dentitions in all ages 6, 7 and 8 years, and all surveys 1972 to 1986, following the introduction of the dental health programme in 1970

(Table 12). The absence of a significant increase in caries free children age 8 years in the 1972 survey could be consistent with the later eruption of the permanent dentition and further, that this particular group of children would have been among the first to enter the programme two years earlier in 1970, at age 6 years. The usual age of entry for all others in the survey was approximately 5 years, when enrolled at school. There may have been a few exceptions where children moved to Boorowa from elsewhere and were enrolled when they arrived.

A comparison of the dental caries experience of the children examined at Boorowa with the national caries experience is important as the very substantial Australia wide decline in dental caries was established by 1977 (Tables 15 and 16 - Appendix D).

It was considered necessary to test the relationship between caries experience of Boorowa and Australian children to assess whether the decline in dental caries noted at Boorowa was simply a reflection of the national decline in dental caries experience.

Although there was a decline in the deciduous dentition dental caries experience of the Boorowa children age 6 to 8 years it could not be shown that dental caries experience was significantly less than the national caries experience. The reason for this may be similar to that already pointed out, that is the children at Boorowa may have had established but slowly developing deciduous dentition dental caries before commencing school and entering the dental health programme.

If established, dental caries has the potential to continue development. This is consistent with the observation that in the surveys made in 1976 and 1982, where the percentage of children with caries free deciduous dentitions age 6 years decreased (Table 11).

Using the increase in the percentage of children with caries free permanent dentitions as an index of dental caries experience, there was a significant decline in dental caries experienced by Boorowa children which could be attributed to the introduction of the Dental Health Programme at the two schools at Boorowa.

Although there was a statistically significant decline in dental caries experience of the deciduous dentition demonstrated in children age 7 and 8 years in 1982 and aged 6, 7 and 8 years in 1986, when compared with the baseline (1970) data, it should be appreciated that children aged 7 and 8 years had been in the programme since enrolling at school at ages 5 or 6 years. The significant decline in deciduous dentition dental caries experience shown in children could be a reflection of both the national and the community caries decline. This would be consistent with comparisons with Australian data (Table 14) showing at Boorowa only children age 8 years in 1982 and 1986, experienced statistically significantly less deciduous dental caries than Australian children

This pattern of development of deciduous tooth caries appears to be consistent with the initiation of dental caries in the deciduous dentition, prior to children entering the programme during their first (kindergarten) year at school at the age of approximately 5 years.

9. DISCUSSION OF *S. MUTANS* PREVALENCE IN DENTAL PLAQUE AND REDUCTION IN DENTAL CARIES EXPERIENCE

Unlike the majority of studies which are directed to consideration of *S. mutans* in individuals and are directed towards the establishment of a relationship between *S. mutans* in dental plaque and dental caries occurring subsequently in the same individual, this study looks at the prevalence of *S. mutans* in the plaque of a community of school children. The aim of this study was to determine the prevalence of *S. mutans* in the school communities at Boorowa. This was expressed as the percentage of children with no *S. mutans* found in their dental plaque (classified as "Grade 1") and the percentage of children who had *S. mutans* established in their dental plaque (classified as "Grade 3").

There was a third group of results where an occasional *S. mutans* colony was cultured from the dental plaque of the children examined. (Classified as "Grade 2")

The parameters for these assessments of *S. mutans* in dental plaque were those established earlier (Woods 1971a)

Analysis of *S. mutans* in dental plaque of the children examined in 1970, the baseline survey, and subsequently following the introduction of the regular dental health programme at both of the Boorowa schools, was made on the basis of combined groups of children ages 6 to 8 years. Two statistical analyses of the prevalence of *S. mutans* were performed, the first to assess the increase in the percentage of children with no *S. mutans* found in their dental plaque and the second to assess the reduction in the percentage of children with "established" *S. mutans* present in their dental plaque.

The results of the baseline study (1970) and the subsequent surveys are set out in Table 6.

Compared with the baseline survey (1970) there was a significant increase in the numbers of children with no demonstrable plaque *S. mutans* (Grade 1) in the years (1971, 1972, 1976 and 1986). The exception was the comparison 1970/1982. Compared with the baseline study there also was a statistically significant decrease in the prevelance of "established" *S. mutans* (Grade 3) in the dental plaque of the children examined. (Table 7) The prevalence of S. mutans was significantly less in all surveys following the introduction of the programme, with the exception of the survey made in 1982.

As has already been stated it was not possible because children were considered in groups during the study, to relate any changes in the prevalence of *S. mutans* in dental plaque to an individual's dental caries experience. What can be said however, is that the reduction in dental caries of the children in this study following the introduction of regular, supervised self applied topical fluorides was also accompanied by a significant reduction in the presence of *S. mutans* in dental plaque of the school communities in Boorowa.

It has been pointed out that the pattern of significant dental caries reduction demonstrated in the permanent dentition showed one exception, children age 8 years in the survey of 1972, who would as a group have been age 6 years when they were first included in the dental health program commencing two years earlier in 1970 (Table 12). This group of children commenced the programme which started in 1970, a year later than all other children who generally commenced at age 5 years.

By contrast, the significant reduction in dental caries experience in the deciduous dentition (Table 11) was restricted to the surveys of 1982 and 1986. It occurred in all age groups, 6, 7 and 8 years in the 1986 survey*, and in children age 7 and 8 years only in the survey made in 1982*. There was no reduction in dental caries prevalence of the deciduous dentition shown in the surveys of 1972 and 1976.

---

* It is important to note that the dental caries data obtained in 1982 and 1985 were from the School Dental Service examinations. There could have been a change in the dental caries diagnostic standard during these years although the data appears to be consistent with the dental caries data obtained in 1970, 1972 and 1976 by the Department of Preventive Dentistry at The University of Sydney.

The children examined in the surveys of 1982 and 1986 entered the programme when they commenced school when the programme had been conducted for over 10 years. There was greater awareness of the need for and practise of domestic dental hygiene. Additionally the group of children aged 8 years in 1972 had entered tht programme when it commnced in 1970 when they were in Year 1, since then all groups children surveved had entered the programme when they commenced school at age approximately 5 years.

The decline in dental caries at Boorowa occurred at a time when there was a declining national dental caries experience in Australia. However the decline in dental caries which occurred at Boorowa during the period of this study, was shown to be significantly greater than the decline occurring nationally. (Tables 13 and 14)

Although the decline in *S. mutans* in the dental plaque of children in Boorowa during the period 1972 to 1986, the years of the dental health programme, and the decline in dental caries during the same period correspond it is not possible to say on the basis of this study whether the decline in *S. mutans* was the cause of the decline in dental caries.

Certainly there is indirect evidence connecting the events. There is for instance the patterns of decline in dental caries which appear to be consistent with a delay in the reduction in the dental caries experience following the reduction in the prevalence of *S, mutans* in plaque.

While the evidence presented strongly supports the relationship between *S. mutans* in dental plaque and dental caries occurring subsequently and is consistent with this relationship, it in not possible to say on the basis of this evidence that there is an aetiological relationship between caries experience at Boorowa and the prevalance of *S. mutans* in plaque. This has been established elsewhere and reviewed in Part 1 of this treatise.

The aetiological relationship between *S. mutans* and dental caries has been established elsewhere and has been reviewed in Part 1 of this treatise.

What has been described is a parameter for the prevalence of *S. mutans* in the dental plaque of a community of primary school children age 5 to 8 years in a rural school community in New South Wales collectively experiencing a significant decline in dental caries.

The reduction in dental caries in Australian children since 1955 has been remarkable and is set out in Appendix C.

9.1. PREVALENCE OF *S. MUTANS* IN DENTAL PLAQUE OF GROUPS OF SCHOOLCHILDREN

The implications of the incidence of *S. mutans* in dental plaque of a group must be regarded in a different light to the implications of the presence of *S. mutans* in the plaque of an individual. In an individual the absence of *S. mutans* from dental plaque is an indicator that the caries susceptibility of that individual is very low (Woods 1971a, Newbrun 1989).

The multifactorial aetiology of dental caries could easily affect conclusions drawn from the presence of mutans streptococci in the dental plaque of a population. The evidence reviewed earlier supports an essential role for mutans streptococci in the aetiology of dental caries. On the other, hand the presence of mutans streptococci in dental plaque does not always lead to the establishment of dental caries. This could be because mutans streptococci are not highly virulent organisms and other variable factors, such as enamel solubility, maturation and enamel structure, could outweigh the caries potential of mutans streptococci in individuals.

It should be appreciated that all infectious diseases are multifactorial; the multifactorial description is not unique to dental caries. The infectious agent is however essential to the development of infectious disease even though other factors may outweigh the effect of the presence of this agent.

Adopting the multifactorial approach to dental caries, a number of factors were assessed in a Swedish study, for their effectiveness as predictors of dental caries for children age 3.5 years (Grindefjord *et al.* 1995, Grindefjord *et al.* 1996). A statistical analysis of factors likely to contribute to the development of dental caries in young children was made when the children were at age one year.

In the study which involved 692 children, twenty eight factors were considered.

Gender
Mother living in Sweden
Mother's native language
Father living in Sweden
Father's native language
Immigrant background
Non-immigrant background
Social class according to father's occupation
Mother's education
Number of siblings
Place of birth
Time of birth
Chronic illness
Breast-feeing
Unsatisfied child through the day
Unsatisfied child at night-time
Medication
Type of medication
Where the child spends the day
Consumption of sugar-containing beverages
Drinks before going to bed
Drinks during the night
Meal frequency
Candy
Toothbrushing frequency
Toothpaste
Fluoride tablets
Mutans streptococci
Number of teeth
Lactobacilli
pH
Gingivitis
Manifest caries
Initial caries
Occlusion
Proximal contacts between primary molars
Cooperation at the examination

Predictors shown to be significant to the age of 2.5 years were; mutans streptococci ($p<0.01$); immigrant background ($p<0.01$) and candy consumption ($p<0.01$).

For children to the age of 3.5 years, predictors shown to be significant were; mutans streptococci ($p<0.001$); mother's education (p,0.001); immigrant background ($p<0.01$); candy consumption ($p<0.05$) and sugar-containing beverages ($p<0.05$).

Although the studies (Grindefjord *et al.* 1995, Grindefjord *et al.* 1996) is restricted to pre-school Swedish children, it is a very valid assessment of a number of factors which could cause dental caries. It is also noteworthy that mutans streptococci have emerged as significant predictors in this multifactorial analysis.

Reviewing caries prediction, Powell (1998a) recognized that where a number of tests were employed in a caries prediction model, those models where bacterial levels tested were included were the most accurate caries predictors.

The relationship between mutans streptococci and dental caries has been explored by a number of investigators and has been the subject of a number of reviews.

Reviewing caries risk in children and considering means to identify susceptible children, Messer (2000) stated "The microbial tests for detecting mutans streptococci are very good at identifying caries-inactive children, as some who have positive scores develop little caries."

Petti and Hausen (2000) studying a group of 304 children to assess the value of three salivary mutans streptococci tests made at three monthly intervals, stated "The results of this study suggest that in a group of initially caries-free children, the multiple salivary (ms) mutans streptococci, tests had a higher predictive power than a single test." Doubt however, was expressed whether the costs of multiple tests could be justified.

Gabris *et al.* (1999) found statistically significant correlations between dental caries values and salivary microbiological counts.

In a review of caries risk assessment, Reich, Lussi and Newbrun (1999) examined the predictive reliability of a number of factors including fluoride use, diet, plaque bacteria and caries experience. It was reported that the combination of past caries experience and values of mutans streptococci provide a more predictive model than is obtained when either test is used alone. It was concluded "The identification of subjects with a high caries-risk is relatively accurate where children and adolescents are concerned and when sufficient base-line data are available."

Sanchez-Perez and Acosta-Gio (2001) demonstrated the caries-predictive values of mutans streptococci counts from mandibular first-molar fissure plaque finding a strong correlation with caries experience.

In Switzerland it was found (Steiner, Helfenstein and Menghini 1998) that discoloration of of pits and fissures which could be identifies as dental caries, was related to the level of mutans streptococci.

In Italy, Brambilla *et al.* (1999) reported a positive relationship between mutans streptococci and lactobacilli in saliva and dental caries in 9 and 13 year-old children, notwithstanding a relatively low dental caries prevalence.

In Sweden it was found that increasing numbers of *S. mutans* and lactobacilli, alone or in combination were associated with an increased caries frequency (Zickert I, Emilson CG and Krasse B 1982).

In Sweden, Kohler and Bjarnason (1992), examining 155, children age 15 to 16 years found that increased numbers of mutans streptococci and lactobacilli were associated with increasing caries prevalence. *Streptococcus sobrinus* carriers (15 percent of the group) had both higher mean caries prevalence and a higher proportion of subjects with high mutans presence in saliva than was found in the whole group.

Reviewing the relationship between *Streptococcus mutans* and dental caries in children in Mozambique, Carlsson, Olsson and Bratthall (1985) concluded that in individual children, there was an association between high counts of *S. mutans* and dental caries although they noted that *S. mutans* can be widely distributed and can occur in high numbers in a population with low prevalence of dental caries.

Icelandic (Reykjavic) children aged 12 years, were shown to have similar numbers of salivary mutans streptococci and lactobacilli when examined in 1991 to 1984 when similar examinations were made. It was noted in 1991 that as was the case in 1984, significantly more *S. sobrinus* carriers had a high level of salivary lactobacilli as well as higher caries prevalence than children who did not carry this bacterium. A similar conclusion was reached in 1987 when 217 Icelandic children age 11 and 12 years were examined (Kohler and Bjarnason 1987).

In the United States, Thibodeau and O'Sullivan (1999) reported on a study involving 85 children examined over a period of six years from the age of 3.5 years. The study was able to identify, using annual measurements of salivary mutans streptococci, long term caries risk in both the primary and mixed dentitions. The authors concluded that despite limitations in predicting caries risk using microbiological methods, the longitudinal study supported the overall benefits of this type of testing. The study confirmed earlier findings by O'Sullivan and Thibodeau (1996).

A study conducted in Spain (Llena-Puy, Montanna-Llorens and Forner-Navarro 2000) in a study of 167 children age 12 to 13 years, observed statistically significant correlations between caries indices and bacterial counts of mutans streptococci and lactobacilli.

In China, in a study involving 229 preschool children, the presence of *S. mutans* in the oral cavity was examined using the commercial *Dentocult SM* technique. The technique, based on the presence of *S. mutans* was found to be a reliable means to measure the status of dental caries in preschool children.* (Shi, Liang, Hayashi, Yakushiji, Machida 1998, Nishimura *et al.* 1998)

Although dental caries is a multi-factorial disease it is hard to imagine any infectious disease which is not multifactorial even though the number of factors may appear to be fewer when the infectious agent has greater virulence.

The relationship between the presence of mutans streptococci and dental caries susceptibility has been supported widely throughout the world. Studies have been undertaken in North America, South America, India, Scandinavia, Europe, China and Japan, all reporting the relationship between mutans streptococci and caries susceptibility. It is of interest that several of the studies cited found a similar relationship between caries susceptibility and lactobacilli (van Houte 1993).

---

* It is noted that microbial plaque examinations were classified using a similar convention to that used in the Boorowa studies

9.2. MUTANS STREPTOCOCCI IN SCHOOL COMMUNITIES

Where individual cases are considered, the value of bacterial tests in relation to dental caries prediction appears to be established. The implications of the incidence of mutans streptococci in populations or groups of people has not been considered. What is the percentage of a population which harbour mutans streptococci? Does it differ with the age of the group? Does it differ with fluoridation? What is the implication for dental caries experience? Is the prevalence of mutans streptococci diminishing in some communities?

The test described in this work was developed as a screening tool. The test can be used, particularly if repeated at regular intervals, as a guide to an individual's dental caries susceptibility. Essentially it is a simple test which can be performed with basic equipment.

The absence of mutans streptococci in a test result, while indicating a low number of the organisms, is not a conclusive demonstration that no mutans streptococci exist in the plaque being examined. The sensitivity of the test technique is limited.

The presence of many mutans streptococci is however clear evidence of their established presence in the plaque examined. Between these two extremes there is the result that might show an occasional, transient mutans streptococci colony but the result does not necessarily indicate establishment of the organism.

When considering the prevalence of mutans streptococci in dental plaque of groups or communities, its established presence (Grade 3 in the method employed) identifies a subgroup in the population susceptible to dental caries and who may experience dental caries provided other factors do not outweigh the cariogenic potential of mutans streptococci.

Put another way, this subgroup which has established mutans streptococci in their dental plaque is exposed to a cariogenic challenge from mutans streptococci which they may either resist or to which they may yield. Further, a single, isolated examination for mutans streptococci in dental plaque may not always provide representative results for individuals but when similar data from a population is considered variations which might have occurred in individual results are likely to be reduced. As with many pathology or microbiological tests, repeated tests are needed to confirm most test results.

The consistent absence of mutans streptococci in dental plaque determined by a number of tests over a period of time, is a good indicator of low caries activity; it is a clinical observation that in an individual, the persistent absence of mutans streptococci from their plaque almost always indicates they will remain caries free*. This has been observed by a number of investigators including van Houte (1993) and Reich *et al.* (1999).

---

* Clinically, the most usual exception where caries occurs is when there has been developmental enamel damage during enamel development. This is particularly evident as fissure hypoplasia and often occurs where there is a history of a persistent febrile state during the period of tooth development. It is a clinical observation that hypoplastic fissures of the first permanent molars are often associated with a history of a persistent febrile state arising from repeated episodes of tonsillitis or other upper pharyngeal infection. A further clinical observation is that a history of a raised core body temperature during the third trimester of pregnancy appears be related in some cases, to hypoplasia involving fissures of developing deciduous molars.

PART 3
CLOSURE

10. CONCLUSION

Since 1970 dental caries prevalence in Australia has declined very substantially. The decline in dental caries has been attributed mostly to water fluoridation and therapeutic use of fluoride: the fluoridation of urban water supplies; the widespread adoption of fluoridated toothpaste; use where appropriate,of fluoride supplements; and use of clinical preventive dentistry procedures mostly based on fluoride. In 1987, 66 per cent of the Australian population were living in areas served by fluoridated water supplies (Barnard 1988 p8).

The remarkable decline in dental caries in the Australian community, and the rural community of Boorowa (NSW) since 1970, in particularly for children of primary school age, has been reviewed.

Since the essential role of bacteria in the initiation and development of dental caries was established, (Orland *et al.* 1955) research has been directed towards identifying the particular microorganism or microorganisms which cause dental caries. Dental caries has been shown to be an infectious disease. Notwithstanding the multifactorial nature of dental caries, a characteristic shared with other infectious diseases, there is strong evidence that cariogenic streptococci, in particular the mutans group of streptococci, are closely associated with the initiation of dental caries. The particular characteristic of mutans streptococci to utilize sucrose but not to any extent other sugars to manufacture extracellular dextrans has been considered likely to assist in the development of dental plaque and the initiation of dental caries. This characteristic of mutans streptococci can be related to the long established association between sucrose intake and dental caries experience.

11. SUMMARY

Studies described in this treatise set out to demonstrate and record the prevalence of *S. mutans* in the dental plaque of children at Boorowa, a rural community in Southern New South Wales. Children at Boorowa were examined in 1970, the baseline examination, and 1971, 1972, 1976 1982 and 1986. Surveys made in 1971, '72 '76, '82 and 1986 corresponded with the introduction of a Community Dental Health Programme at Boorowa aimed at minimizing the dental caries of the primary school children. The programme was based on regular supervised, self-application of a 10 percent stannous fluoride based toothpaste.

During the period 1970 to 1986, dental caries experienced by children at Boorowa diminished considerably. Initially, in 1970 children 8 years of age had a mean DMFT of 2.82, in 1986 it had fallen to 0.25 (Tables 8 and 9).

In the period 1970 to 1986 the percentage of children whose dental plaque included *S. mutans* at the two Boorowa schools had been reduced from 63 per cent to be within the range from 50 to 43 per cent. Those children in whom no *S. mutans* was found had risen from 37 percent to be within the range 50 to 57 percent (Table 12). When compared with the baseline, the proportion of children with no *S. mutans* in their dental plaque (Grade 1) at examinations made in 1971, 1972, 1976, 1982, and 1986, with the exception of 1982, was significantly greater. (Table 7)

The decline in dental caries recorded in the children at the schools in Boorowa appears to have been in advance of the widespread decline of caries throughout Australia. The greater reduction in the dental caries experience in Boorowa is attributed to the effect of the Community Dental Health Programme established at Boorowa in 1970 and which continued for 18 years until 1986. In addition data presented on the dental caries experience and the prevalence of *S. mutans* in dental plaque of Boorowa children, are an important record of a rural community during the period when dental caries experience throughout Australia was first in decline.

Of considerable importance and arising from studies reviewed on the infectious nature of dental caries, a clear understanding of the natural history of dental caries as an infectious disease has emerged.

In particular, studies confirming the vertical transmission of dental caries from mother to child, has led to the development of a "medical model" as an alternative to the "surgical model", for the treatment of dental caries.

The development of a "medical model" for treatment of dental caries marks the maturity of preventive dental practise.

Evidence supporting the designation of dental caries as an infectious disease has been reviewed and the role of mutans streptococci as the principal infectious agent, has been explored. Evidence that dental caries is an infectious disease and that without the presence of bacteria the dental caries process cannot occur is beyond doubt; Evidence to support the aetiological role of mutans streptococci is very strong. A recent review made by Balakrishnan, Simmonds and Tagg (2000) endorses the established concept of dental caries as an infectious disease.

Studies conducted at Boorowa did not attempt to confirm the aetiological role of *S. mutans* in dental caries. The aetiological role of *S. mutans* in dental caries has been reviewed in *Part 1* of this treatise. The object of the studies was to assess the effect of the Community Dental Health Programme introduced in 1970, on dental caries prevalence and on the presence of *S. mutans* in dental plaque. This objective was realized. The project was commenced at a time when the dental caries experience in the Australian community was very high.

Following the introduction of the supervised, self applications of stannous fluoride there was significant reduction in the presence of *S. mutans* in dental plaque and a substantial and progressive decline in dental caries experience of the children involved with the program. This is consistent with the relationship established between mutans streptococci and dental caries described in Part 1 of this treatise and is also consistent with the concept of dental caries as an infectious disease.

Dental caries experienced by children aged five to eight years attending the schools in Boorowa, a rural community in New South Wales, during the years 1970 to 1986 and the prevalence of *S. mutans* in these children have been documented. This information was obtained from observations made in the field before and during the years when dental caries experience was declining. The opportunity to obtain these data is not likely to occur again.

During the period when Australia was experiencing a remarkable decline in dental caries, Boorowa was also experiencing a marked decline in dental caries which occurred several years ahead of the wider Australian experience. Evidence has been presented to support the conclusion that the early decline in dental caries experienced at Boorowa was the result of the Community Dental Health Programme conducted there from 1970 to 1986.

As has been noted the decline in dental caries experience at Boorowa was significantly greater than the national decline in dental caries.

The decline in dental caries experience in Australia since 1975 has directly improved the quality of life of a large part of the Australian population and relieved strain on the country's health resources. An understanding of the infectious nature of dental caries and mechanisms to control the disease will provide valuable help to extend the benefits of the dental caries reduction to all sections of the community and maintain a low dental caries experience.

Having a record of the prevalence of mutans streptococci in dental plaque leading up to and during the period of decline of dental caries in a rural community, could provide baseline data for future assessments of the microbial challenge leading to the development of dental caries.

Australia still has several population enclaves which have not received the benefit of widespread reduction in dental caries; the information presented in this treatise may assist to bring the benefits of reduced dental caries to these groups in the community. These data may so be of use monitoring change in dental caries prevalence or the prevalence of *S. mutans* in plaque.

In the event that in the future dental caries in Australia were to increase, this information may contribute to developing means for the containment of any such increase in the disease and assist retain the benefits achieved.

Data obtained from this study and information gained from the conduct of the Community Dental Health Programme for 18 years, can provide assistance in reducing dental caries experience in the developing world. The parameters for the prevalence of *S. mutans* in dental plaque in communities of children who are experiencing low dental caries have been defined. This data provides a means to establish a measure of the prevalence of *S. mutans* in the dental plaque of a community of children experiencing a reduction in their dental caries.

The development of a "medical model" for treatment of dental caries has been reviewed and is a realistic alternative to the "surgical model" when considered in the perspective of the long term management of the disease.

Essentially, this treatise commences with a review of the infectious nature of dental caries and the direction that its treatment and control might take leading to the development of the "medical model" for treatment of dental caries. This treatise procedes to report 18 years of field research, recording the prevalence of dental caries and the presence of *S. mutans* in dental plaque of children in a rural community prior to and during the period of decline of dental caries in Australia.

Finally, the Dental Health Programme conducted at the Central and Saint Joseph's schools at Boorowa has been shown to have made a significant contribution to the control of dental caries in the children of the Boorowa community, 1982 to 1986

12. LIST OF REFERENCES

AIHW DENTAL STATISTICS AND RESEARCH UNIT (1992)
The child dental health survey, Australia, 1990. AIHW Dental Statistics & Research Unit Series No. 2. The University of Adelaide, Adelaide.

AIHW DENTAL STATISTICS AND RESEARCH UNIT (1995)
The child dental health survey, Australia, 1993. AIHW Dental Statistics & Research Unit Series No 7. The University of Adelaide, Adelaide.

ARMFIELD JM, ROBERTS-THOMSON KF SPENCER AJ (1999)
The child dental health survey, Australia 1996. AIHW Dental Statistics and Research Ubit Series No. 20, The University of Adelaide, Adelaide.

ARMFIELD JM, ROBERTS-THOMSON KF SPENCER AJ (2000)
The child dental health survey, Australia 1997. AIHW Dental Statistics and Research Ubit Series No. 21, The University of Adelaide, Adelaide.

ALALUUSUA S, MYLLARNIEMI M and KALLIO M (1989)
Streptococcus mutans infection level and caries in a group of 5 year old children. Caries Res 23:190-194.

BALAKRISHNAN M, SIMMONDS RS, TAGG JR (2000)
Dental caries is a preventable infectious disease. Aust Dent J 45(4):235-245.

BARNARD PD, WOODS R, and CUTRESS T (1977)
Western Samoan schoolchildren II. DMFT, gingivitis, enamel fluoride and S. mutans. IADR (Aust. and New Zealand Div.) Perth, 1977, paper 32.

BARNARD PD (1956)
Dental survey of State school children in New South Wales, January 1954 - June 1955, The Institute of Dental Research, The United Dental Hospital of Sydney. Canberra, National Health & Medical Research Council Special Report Series No.8.

BARNARD PD ed.(1988)
Facts and figures Australian dentistry 1986-87. North Sydney The Australian Dental Association.

BARNARD PD and CLEMENTS F (1976)
Oral health care in Australia. Int Dent Jour 26(3):320-326.

BERKOWITZ RJ and JORDAN HV (1975)
The early establishment of *Streptococcus mutans* in the mouths of infants. Arch Oral Biol 20(3):171-174.

BISSET KA and DAVIS GHG (1960)
The microbial flora of the mouth. London: Heywood;55.

BLACK GV (1887).
Dental Caries. In: The American system of dentistry.
Litch WF.(ed). Appendix. Philadelphia, Lea Brothers and Co;729.

BOWDEN GH (1997)
Does microbial composition of plaque/saliva allow for diagnosis of disease activity of individuals? Community Dent Oral Epidemiol 25:76-81.

BOWDEN G, EDWARDSON S. (1994)
In Textbook of clinical cariology, eds Thylstrup A, Fejerskov O. Copenhagen Munksgaard, 65.

BRAMBILLA E, FELLONI A, GAGLIANI M, MALERBA A, GARCIA-GODOY F, STROHMENGER L. (1998)
Caries prevention during pregnancy: Results of a 30-month study.
J Amer Dent Assoc 129:871-77.

BRAMBILLA E, TWETMAN S, FELLONI A, CAGETTI MG, CANEGALLO L, GARCIA-GODOY F and STROHMENGER L (1999)
Salivary mutans streptococci and lactobacilli in 9 and 13-year-old Italian schoolchildren and the relation to oral health. Clin Oral Invest 3(1):7-10.

BRATTHALL D (1970)
Demonstration of five serological groups of streptococcal strains resembling *Streptococcus mutans Odont Revy 21(2):143-152.*

BRATTHALL D (1972a)
Demonstration of Streptococcus mutans strains in some selected areas of the world. Odont Revy 23:401-410.

BRATTHALL D (1972b)
Demonstration of five serological groups of streptococcal strains resembling *Streptococcus mutans. Odont Revy 23(4):141-152.*

BREED RS, MURRAY EGD and SMITH RN eds. (1957)
Bergey's Manual of Determinative Bacteriology, 7th ed. Baltimore: Williams & Wilkins.

BRINNER WW and FRANCIS MD (1962)
The effect of enamel fluoride on acid production by Lactobacillus casei. Arch Oral Biol 7(5):541-550.

BRUDEVOLD F, SAVORY A, GARDNER DE, SPENELLI M and SPEIRS R (1963)
A study of acidulated fluoride solutions - 1. Arch Oral Biol 8(2):167-177.

BURNET M (1962)
Natural history of infectious disease 3rd ed. Cambridge University Press (p171).

BURNETT GW, SCHERP HW, and SCHUSTER (1976)
Oral microbiology and infectious disease. 4th ed. Baltimore, Williams & Wilkins; 201.

BURNETT GW and SCHERP HW (1968).
Oral Microbiology and Infectious Disease, 3rd ed. Baltimore, Williams & Wilkins; 21.

BURT BA, ISMAIL AI, MORRISON EC, BELTRAN ED (1990)
Risk factors for tooth loss over a 28-year period. J Dent Res 69(5):1126-1130.

CAMPUS G, LAMBAU A, LAI S, FALCOLINI G (1997)
Mutans streptococci, lactobacilli and caries experience in 6- to 8- year old Sardinian Urban Children. Caries Res 31:299 (ORCA Congress abstract).

CARLSSON J, GRAHNEN H, JONSSON G and WIKNER S (1970)
Establishment of *Streptococcus sanguis* in the mouths of infants. Arch Oral Biol 15(1):12.

CARLSSON J, SODERHOLM G and ALMFELDT I (1969)
Prevalence of *Streptococcus sanguis* and *Streptococcus mutans* in the mouths of persons wearing full dentures. Arch Oral Biol 14(3):243-249.

CARLSSON J (1965)
Zooglea-forming streptococci, resembling *Streptococcus sanguis*, isolated from dental plaque in man. Odont Revy 16(4):348-358.

CARLSSON J (1967)
A medium for isolation of *Streptococcus mutans*. Arch Oral Biol 12(12):1657-1658.

CARLSSON J (1968a)
A numerical taxonomic study of human oral streptococci. Odont Revy 19(2):137-160.

CARLSSON J (1968b)
Plaque formation and streptococcal colonization on teeth. 1968, Odont. Revy., 19:Supplement 14.

CARLSSON J (1968b)
Plaque formation and streptococcal colonization on teeth. Odont Revy 19:(Supplement)14.

CARLSSON J and EGELBERG J (1965)
Effect of diet on early plaque formation in man. Odont Revy. 16(2):112-125.

CARLSSON J, HAMILTON I (1994)
Metabolic activity of oral bacteria *in* Textbook of clinical cariology, 2nd ed, eds. Thylstrup A and Fejerskov O. Copenhagen, Munksgaard.

CARLSSON P, OLSSON B, BRATTTHALL D (1985)
The relationship between the bacterium Streptococcus mutans in the saliva and dental caries in Mozambique. Arch Oral Biol 30(3):265-268.

CARR LM (1976)
Fluoridation in Canberra II. Aust Dent J 21(5):440-444.

CATALANOTTO FA, SHKLAIR IL, KEENS HJ and LEVINE J (1974)
Prevalence and localization of *Streptococcus mutans* in infants and children. J Dent Res 53:195 (Special issue) (Abst 563).

CAUFIELD PW, DASANAYAKE AP, LI Y, PAN Y, HSU J, HARDIN JM (2000)
Natural history of Streptococcus sanguid in the oral cavity of infands: evidence of a discrete window of infectivity. Infect Immun 68(7):4018-4023.

CLARK JK (1924)
On the bacterial factor in the aetiology of dental caries. Brit J Exp Pat 5:141-147.

COMMONWEALTH DEPARTMENT OF HEALTH (1987)
Dental health of children in Australia 1977-1986. Canberra, Australian Government Publishing Service.

COYKENDAL AL (1973)
Taxonomy of *Streptococcus mutans*. *In Streptococcus mutans* and dental caries. Proceedings of a round table discussion May 10, 1973, 73rd Annual Meeting, American Society for Microbiology, Miami Beach, Florida, National Caries Programme, National Institute of Dental Research: 3-6.

COYKENDALL AL, SPECHT FA and SAMOI H (1974)
Streptococcus mutans in a wild sucrose-eating rat population. J Dent Res 53:243 (Special issue) Abstr 753.

DAVIS GN, KING RM and COLLINS AA (1959)
The relationship between lactobacillus counts, Snyder tests and the subsequent incidence of dental caries. Arch Oral Biol, 1(1):62-73.

DAWES C, JENKINS GN, HARDWICK JL and LEECH SA (1965)
The relation between the fluoride concentrations in the dental plaque and in drinking water. Br Dent J 119:164-167.

DEIBLE RH and SEELEY HW (1974)
In Bergey's Manual of determinative bacteriology (8th ed) eds. Buchanan RE, Gibbons NE. Baltimore: Williams & Wilkins:490-509.

DUANY LF, MENA JC, JABLON JM and ZINNER DD (1971)
Association between microorganisms, plaque, diet and caries in students. J Dent Res 50:233 (Special issue) (Abst).

DVARSKAS RA and COYKENDALL AL (1975)
Streptococcus mutans in wild rats on a low sucrose diet. J Dent Res 54:127 (Special issue A) Abstr 330.

EDWARDSSON E (1968)
Characteristics of caries-inducing human streptococci resembling *Streptococcus mutans*. Arch Oral Biol 13(6):637-46.

ELVIN-LEWIS M, KEUDEL K, KLUGMAN G, McGIVNEY J, BALDWIN R and QUINN D (1975)
Distribution of potentially cariogenic streptococci among zoo animals. J Dent Res 54:127 (Special issue A) Abstr 331.

EMANUELSSON IR, BRATTHALL D (1998)
Genotyping shows different strains of mutans streptococci between father and child and within parental pairs in Swedish families. Oral Microbiol Immunol 13:271-77.

ENGLANDER HR and KEYES PK (1964)
Failure to induce dental caries in hamsters by transfer of plaque and carious debris from humans. J Dent Res 43(4):626. Research annotation)

FISET L, GREMBOWSKI D (1997)
Adoption of innovative caries-control services in dental practice. J Amer Dent Assoc 128:337-45.

FISHER RA, YATES F (1953)
Statistical Tables for Agricultural, Biological and Medical Research. Edinburgh. Oliver and Boyd.

FITZGERALD RJ, JORDAN HV and ARCHARD HO (1966)
Dental caries in gnotobiotic rats infected with a variety of *Lactobacillus acidophilus*. Arch Oral Biol 11(5):473-476.

FITZGERALD RJ, JORDAN HV and STANLEY HR (1960)
Experimental caries and gingival pathologic changes in the gnotobiotic rat. J Dent Res 61(1):9-19.

FITZGERALD RJ and KEYES PH (1960)
Demonstration of the etiologic role of streptococci in experimental caries in the hamster. J Am Dent Assoc 61(1):9-19

FURE S (1998)
Five-year incidence of caries, salivary and microbial conditions in 60-, 70- and 80-year-old Swedish individuals. Caries Res 32:166-74.

GABRIS K, NAGY G, MADLENA M, DENSES Zs, MARTON S, KESZTHELYI G and BANOCZY J (1999)
Associations between microbiological and salivary caries activity tests and caries experience in Hungarian adolescents. Caries Res 33:191-195.

GIBBONS BJ, BERMAN KS, KNOETTWER P and KAPSIMALIS B (1966)
Dental caries and alveolar bone loss in gnobiotic rats infected with capsule forming streptococciof human origin. Arch Oral Biol 11(6):549-559.

GIBBONS RJ and BANGHART S (1968)
Induction of dental caries in gnotobiotic rats with a Levan-forming streptococcus and a streptococcus isolated from substrate bacterial endocarditis. Arch Oral Biol 13(3):297-308.

GIBBONS RJ and LOESCHE WJ (1967)
Isolation of cariogenic streptococci from Guatemalan children. Arch Oral Biol 12(8):1013-1014.

GIBBONS RJ and NYGAARD M (1968)
Synthesis of insoluble dextran and its significance in the formation of gelatinous deposits by plaque-forming streptococci. Arch Oral Biol 13(10):1249-1262.

GOLDSWORTHY NE (1958)
The biology of the children of Hopewood House, Bowral, NSW, II. Observations extending over five years (1952-1956 inclusive). I Introduction. Aust Dent J 3(5):309-310.

GOLDSWORTHY NE and SPIES HC (1958)
The biology of the children of Hopewood House, Bowral, NSW, II. Observations extending over five years (1952-1956 inclusive). 3 The Lactobacillus count and its relation to dental caries. Aust Dent J, 3(10):318-330.

GRINDEFJORD M, DAHLLOF G, NILSSON B AND MODEER (1995)
Prediction of Dental Caries in 1-year-old Children. Caries Res 29:343-348.

GRINDEFJORD M, DAHLLOF G, NILSSON B AND MODEER (1996)
Stepwise Prediction of Dental Caries in Children up to 3.5 Years of age. Caries Res 30:256-266.

GUSTAFSSON BE, BENGT E, QUENSEL CE, LARKE LS, LUNDQVIST C, GRAHNEN H and BONOW BE (1954)
The Vipeholm dental caries study. The effect of different levels of carbohydrate intake on caries activity in 436 individuals observed for five years. Acta Odontol Scand 11:232-388.

HADLEY FP (1933)
A quantitative method for estimating bacillus acidiphilis in saliva. J Dent Res 13:415-428.

HAMILTON IR (1977)
Effects of fluoride on enzymatic regulation of bacterial carbohydrate metabolism. Caries Res 11:(Suppl. 1)262-291.

HANADA N (2000)
Current understanding of the cause of dental caries. Jpn J Infect Dis 53:1-5.

HARDIN G (1960)
The competitive exclusion principle. Science 131:1292-1297.

HARDWICK JL and LEACH SA (1962)
The fluoride content of dental plaque. Arch Oral Biol 7:(Supplement, Proceedings of the 9th Congress of the European Organization for Research on Fluorine and Dental Caries Prevention, Paris) 151-158.

HILL IN and BLAYNEY RJ (1965)
The Evanston dental caries study. XXIII. Oral lactobacilli and decayed tooth surface rates among children in a fluoridated area. J. Dent. Res., 1965, 44(4):718-733.

IKEDA T, SANDHAM HJ, BRADLEY EL (Jr) (1973)
Changes in Streptococcus mutans and lactobacilli in relation to the initiation of dental caries in negro children. Arch Oral Biol 18(4):555-566.

JORDAN HV, ENGLANDER HR, ENGLER WO and KILCZYK S (1972)
Observations on the implantation and transmission of *Streptococcus mutans* in humans. J Dent Res 51(2):515-518.

JORDAN HV, ENGLANDER HR and LIM S (1969)
Potentially cariogenic streptococci in selected population groups in the western hemisphere. J Am Dent Assoc 78(6):1331-1335.

JORDAN HV (1973)
Other organisms and other types of caries. *In* Streptococcus mutans and dental caries. Proceedings of a round table discussion May 10, 1973,

JORDAN HV, KRASSE B, MOLLER A (1968)
A method of sampling human dental plaque for "caries-inducing" streptococci. Arch Oral Biol 13:919-927.

KEYES PH (1960)
The infectious and transmissible nature of experimental dental caries. Arch Oral Biol 1(4):304-320.

KEYES PH (1962)
Recent advances in dental caries research. Bacteriology bacteriological findings and implications. Int Dent Jour; 12(4):443-464.

KEYES PH (1962)
Present and future measures for dental caries control. J Amer Dent Assoc 79(6):1395-1404.

KEYES PH and FITZGERALD RJ (1962)
Dental caries in the Syrian hamster - IX. Arch Oral Biol 7:(3):267-277.

KILIAN M, THEILADE E and SCHIOTT CR (1971)
Isolation of *Streptococcus mutans* from human faeces. Arch Oral Biol 16(5):553-554.

KOHLER B, BRATTHALL D and KRASSE B 1983)
Preventive measures in mothers influence the establishment of the bacteria Streptococcus mutans in their infants. Arch Oral Biol 28:225-231.

KOHLER B, ANDREEN I (1994)
Influence of caries-preventive measures in mothers on cariogenic bacteria and caries experience in their children. Archs Oral Biol 39(10):907-11.

KOHLER B, BJARNASON S (1987)
Mutans streptococci, lactobacilli and caries prevalence in 11- and 12-year-old Icelandic children. Com Dent Oral Epidemiol 15(6):332-335.

KOHLER B, BJARNASON S (1992)
Mutans streptococci, lactobacilli and caries prevalence in 15 to 16-year olds in Goteborg. Part II. Swed Dent J 16(6):253-259.

KOZLOWSKI GG, SHKLAIR IL, KEENE HJ and LEVINE JA (1973)
Prevalence of *Streptococcus mutans* and association with dental caries in children. J Dent Res 52:196 (Special Issue) Abstr.

KRASSE B, JORDAN HV, EDWARDSSON I, SVENSSON I and TRELL L (1968)
The occurrence of certain "caries-inducing" streptococci in human dental plaque material. Arch Oral Biol 13(8):911-918.

KRASSE B (1965a)
The effect of the diet on the implantation of caries-inducing streptococci in hamsters. Arch Oral Biol 10(2):223-226.

KRASSE B (1965b)
The effect of caries-inducing streptococci in hamsters fed diets with sucrose or glucose. Arch Oral Biol 10(2):223-226.

KRASSE B (1966)
Human streptococci and experimental caries in hamsters. Arch Oral Biol 11(4):429-436.

KRASSE B and CARLSSON J (1970)
Various types of streptococci and experimental caries in hamsters. Arch Oral Biol 15(1):25-32.

LARMAS (1992)
Diagnostic tests for normal dental practice. Int Dent Jour 42:199-208.

LEHNER T, CHALLACOMBE SJ and CALDWELL J (1975)
An experimental model for immunological studies of dental caries in the Rhesus monkey. Arch Oral Biol 20(5/6):299-304.

LEHNER T, CALDWELL J and CHALLACOMBE SJ (1977)
Effects of immunization on dental caries in the first permanent molar in Rhesus monkeys. Arch Oral Biol 22(6):393-397.

LILIENTHAL B and REID H. (1959)
A new laboratory test for acid production in saliva - carbohydrate mixtures and its comparison with the Lactobacillus count and the Snyder test. Arch Oral Biol, 1(2):125-132.

LITTLETON NW, KAHEHASHI S and FITZGERALD RJ (1970)
Recovery of specific "caries-inducing" streptococci from carious lesions in the teeth of children. Arch Oral Biol 15(5):461-463.

LLENA-PUY MC, MONTANANNA-LLORENS C, FORNER-NAVARRO L (2000)
Cariogenic oral flora and its relation to dental caries. ASDC J Dent Child 67:42-46.

LOVELOCK DJ (1973)
The loss of topically applied fluoride from the surface of human enamel *in vitro*, using ^{18}F. Arch Oral Biol 18(1):27-29.

LOESCHE WJ, MURRAY RJ and MELLBERG JR (1973)
The effect of topical acidulated fluoride on percentage of *Streptococcus mutans* and *Streptococcus sanguis* in interproximal plaque samples. Caries Res 7:283-96.

MA JK (1999)
The caries vaccine: a growing prospect. Dent Update 26:374-80.

MAJNO, G (1975)
The healing hand. Cambridge MA: Harvard University Press, 16-19.

MARGALIT D and GEDALIS I (1969)
Release of fluoride into saliva after topical fluoride application. J Dent Res 48(1):93-96.

MATTOS-GRANER RO, ZELANTE F, LINE RCSR, MAYER MPA. (1998)
Association between caries prevalence and clinical, microbiological and dietary variables in 1.0 to 2.5-year-old Brazilian children. Caries Res 32:319-33.

MELBERG JR, LAAKSO PV and NICHOLSON CR (1966)
The acquisition and loss of fluoride by topically fluoridated tooth enamel. Arch Oral Biol 11(12):1213-1220.

MESSER LB (2000)
Assessing caries risk in children. Aust Dent Jour 45:(1)10-16.

MICHALEK SM and McGHEE JR (1977)
Virulence of Streptococcus mutans: An antibiotic suppressed rat model for studies of pathogenesis. J Dent Res 56(3):205-211.

MILLER, WD (1887)
Fermentation in the human mouth: its relation to caries of the teeth. In: The American system of dentistry, Vol 1, Litch, WF.(ed) Philadelphia Lea Bros & Co:791-828.

MILLER, WD (1890a)
Micro-organisms of the human mouth. Philadelphia, The S.S. White Dental Mfg Co: 102-105 (Facsimile edition, Karger, 1973).

MILLER, WD (1890b)
Micro-organisms of the human mouth. Philadelphia, The S.S. White Dental Mfg Co: 215. (Facsimile edition, Karger, 1973)

MILLES WT and UNDERWOOD AS (1881)
An Investigation into the Effects of Organisms Upon the Teeth and Areolar Portions of the Jaws. Int Med Congress (7th) vol 3. London Klockmann 523-529. (secondary reference)

NATIONAL HEALTH and MEDICAL RESEARCH COUNCIL (1991)
The effectiveness of water fluoridation. A report from the NH&MRC Working Group. Canberra: Aust Govt Publishing Service. (Table 4 P Barnard);38.

NEWBRUN E (1969)
Sucrose, the arch criminal of dental caries. J Dent Child 36:239-248.

NEWBRUN E (1989)
Cariology, 3rd Ed. London: Quintessence, 285.

Nobre dos Santos M, Melo dos Santos L, Francisco SB, Cury JA (2002) Relationship among dental plaque composition, daily sugar exposure and caries in the primary dentition. Caries Res 36:247-252.

O'SULLIVAN DM and THIBODEAU EA (1996)
Caries experience and mutans streptococci as indicators of caries incidence. Pediatr Dent 18:(1)371-374.

NISHIMURA M, BHUIYAN MM, MATSUMURA S and SHIMONO T (1998)
Assessment of the caries activity test (Cariostat) based on the infection levels of mutans streptococci and lactobacilli in 2- to 13-year-old childrens' dental plaque. ASDC J Dent Child 65(4):248-251.

ORLAND FJ, BLAYNEY JR, HARRISON RW, REYNIERS JA, TREXLER PD, WAGNER M GORDON HA and LUCKEY TD (1954)
Use of germ free animal technic in the study of experimental dental caries. I. Basic observations on rats reared free of all microorganisms. J Dent Res 33(2):147-174.

ORLAND FJ, BLAYNEY JR HARRISON RW, REYNIERS JA, TREXLER PC ERVIN MS GORDON HA AND WAGNER M (1955).
Experimental caries in germfree rats inoculated with enterococci. J Am Dent Assn 50(3)259-272.

ORLAND FJ (1959)
A review of dental research using germfree animals, Annals NY Acad Sci, 78:285.

O'SULLIVAN DM, THIBODEAU EA (1996)
Caries experience and mutans streptococci as indicators of dental caries. Pedaitr Dent, 18(50:371-374.

PETERSON LG (1976)
Fluorine gradients in outermost surface enamel after various forms of topical application of fluorides *in vivo*. Odont Revy 27:25-50.

PETTI S HAUSEN HW (2000)
Caries prediction by multiple salivary mutans streptococci counts in caries-free children with different levels of fluoride exposure, oral hygiene and sucrose intake. Caries Res 34:380-387.

POWELL LV. (1998a)
Caries risk assessment: Relavence to the practitioner. Jour Amer Dent Assoc 129:(Mar)349-53.

POWELL LV (1998b)
Caries prediction: a review of the literature. Com Dent Oral Epidemol 26(6):361-371.

REICH E, LUSSI A and NEWBRUN E (FDI Commission) (1999)
Caries-risk assessment. Int Dent Jour 49:15-26.

RICHARDSON B (1967)
Fixation of topically applied fluoride in enamel. J Dent Res 46(1):87-91.

RODRIGUEZ FE (1930)
Methods of determining quantitatively, the incidence of Lactobacillus acidophilus - odontolyticus in the oral cavity. J Am Dent Assoc 17(9):1771.

ROETERS FJM, van der HOEVEN JS, BURGERSDIJK RCW and SCHAEKEN MJM (1995)
Lactobacilli, Mutans streptococci and Dental Caries: A Longitudinal Study in 2-Year-Old Children up to the Age of 5 Years. Caries Res 29:272-279.

ROGERS AH (1973)
The occurrence of *Streptococcus mutans* in the dental plaque of a group of Central Australian Aborigines. Aust Dent J 18(3):157-159.

ROGERS AH (1975)
Bacteriocin types of *Streptococcus mutans* in human mouths. Arch Oral Biol 20:853-58.

ROGERS AH (1977)
Evidence for the transmissibility of human dental caries. Aust. Dent J 22(1):53-56.

ROGERS AH (1980)
Bacteriocin typing of *Streptococcus mutans* strains isolated from family groups. Aust Dent J 25(5):279-283.

RUSSELL MW, HAJISHRNGALLIN G, CHILDERS NK, MICHALEK SM (1999)
Secretory immunity in defence against cariogenic mutans streptococci. Caries Res;33:4-15.

SANCHEZ-PEREZ L and ACOSTA-GIO AE (2001)
Caries risk assessment from dental plaque and salivary Streptococcus mutans counts on two culture media. Arch Oral Biol 46:49-55.

SCHAMSCHULA RG, ADKINS BL, BARMES DE, CHARLTON G and DAVEY BG (1978)
WHO Study of dental caries etiology in Papua New Guinea. Geneva, World Health Organization, WHO offset publication No. 40.

SCHAMSCHULA RG and BARMES DE (1970)
A study of the streptococcal flora of plaque in caries free and caries active primitive peoples. Aust Dent J 15(5):377-382.

SCHAMSCHULA RG and CHARLTON G (1971)
A study of caries aetiology in New South Wales school children I. The streptococcal flora of plaque and caries prevention. Aust Dent J 16(2):77-82.

SHI S, LIANG Q, HAYASHI Y, YAKUSHIJI M AND MACHIDA Y (1998)
The relationship between caries activity and the status of dental caries - application of the Dentocult SM method. Chin J Dent Res 1(3):52-55.

SHKLAIR IL, KEENE HJ and CULLEN P (1972)
The distribution of *S. mutans* on the teeth of caries free and caries active naval recruits. J Dent Res 51:66 (Special iss) Abstr.

SHKLAIR IL (1973
Streptococcus mutans and the epidemiology of dental caries. *In* Streptococcus mutans and dental caries. Proceedings of a round table discussion May 10, 1973, 73rd Annual Meeting, American Society for Microbiology, Miami Beach, Florida, National Caries Programme, National Institute of Dental Research: 45-48.

SHKLAIR IL and KEENE HJ (1973)
Relationship of *S. mutans* in plaque and faeces to dental caries. J Dent Res 52:207 (Special iss) (Abst).

SIMS W (1966)
The effect of sodium fluoride on the rate of acid production of surface aggregates of streptococci and lactobacilli. J Dent Res 45(3):915-920.

SLADE GD, SPENCER AJ, DAVIES MJ, STEWART JF (2000)
Caries experience among children in fluoridated Townsville and unfluoridated Brisbane. ANZ Jour Pub Health 20(6);623-629.

SLAVKIN HC (1997)
First encounters: Transmission of infectious oral diseases from mother to child. Jour Amer Dent Assoc 128:773-78.

SNYDER ML, PORTER DR, CLAYCOMB CK and SIMS W.(1962)
Evaluation of laboratory tests for estimation of caries activity. J Am Dent Assoc, 65(1):30-45.

SNYDER ML (1940)
Simple colorimetric method for the estimation of relative numbers of lactobacilli in the saliva. J Dent Res, 19(8):349.

SODERLING E, ISOKANGAS P, PIENIHAKKINEN K. TENOVUO J. (2000)
Influence of material xylotol consumption on acquisition of mutans streptococci by infants. J. Dent Res 79:882-87.

STEINER M, HELFENSTEIN U and MENGHINI G (1998)
Association of salivary mutans streptococci with discoloured pits and fissures. Com Dent Oral Epidemiol 26(6):412-417.

STEPHAN RM (1948)
Some local factors in the development of cavities - plaques, acidity, aciduric bacteria, proteolytic bacteria. In: Dental caries mechanism and present control technics. Easlick KA, ed. St. Louis: Mosby, 40-43.

STOOKEY GK (1966)
Fluoride therapy. *In* Improving dental practice through preventive measures. Burnier JL and Muhler JC eds. St Louis Mosby 76-80.

STRAETEMANS MME, van LEVEREN C, de SOET JJ, de GRAFF J and ten CATE JM (1998)
Colonization with Mutans Streptococci and Lactobacilli and the Careies Experience of childrenafter age of five. J Dent Res 77(10)1851-55.

THIBODEAU EA, O'SULLIVAN DM (1995)
Salivary Mutans Streptococci and Incidence of Caries in Preschool Children. Caries Res 29:148-153.

THIBODEAU EA, O'SIULLIVAN DM (1999)
Salivary mutans streptococci and caries development in the primary and mixed dentitions of children. Com Dent Oral Epidemol 27(6):406-412

TRAHAN L (1995)
Xylitol: a review of its action on mutns streptococci and dental plaque - its clinical significance. Int Dent J 45:77-92.

VAN HOUTE J (1993)
Microbiological predictors of caries risk. Adv Dent Res 7(2):87-96.

WAGG BJ (1955)
The determination of lactic acid produced by oral Lactobacilli and streptococci when grown in chemically undefined media. Br Dent J 99:109-114.

WAN AKL, SEOW WK, PURDIE DM, BIRD PS, WALSH LJ, TUDEHOPE DI. (2003)
A longitudinal study of *Streptococcus mutans* in Infants after tooth eruption. J Dent Res 82(7):504-08.

WEINBERGER SJ, WRIGHT GZ (1989)
Correlating *Streptococcus mutans* with dental caries in young children using a clinically applicable micribiological method. Caries Res 23:385-88.

WOOD JM and CRITCHLEY P (1966)
The extracellular polysaccharide produced from sucrose by a cariogenic streptococcus. Arch Oral Biol 11(10):1039-1042.

WOODS R (1967)
Effects of di-sodium phosphate on rat caries and oral flora. J Dent Res 46(6):1307 Abstr.

WOODS R (1971a)*.
A dental caries susceptibility test based on the occurrence of *Streptococcus mutans* in plaque material. Aust Dent J, 16(2):116-121.

WOODS R (1971b)*
The short term effect of topical fluoride applications on the concentration of *Streptococcus mutans* in dental plaque. Aust Dent J. 16(3)152-155.

WOODS R (1973)*
Transmission of Streptococcus mutans among schoolchildren aged 7 to 8 years. J Dent Res 52(3):576, (Abst).

WOODS R (1975)
An assessment of antibiotics for the treatment of pyogenic dental infections. Aust Dent J 20(1):10-13.

WOODS R, MARTIN ND and BARNARD PD (1976)
A community dental health project I. Self applied SnF_2-$ZrSiO_4$ prophylactic paste and dental caries in primary school children. Aust Dent J 21:205-210

WOODS R (1976)*
A community dental health project II. Modification of *Streptococcus mutans* in dental plaque by self applied SnF_2-$ZrSiO_4$, prophylactic paste, and its relationship to dental caries. Aust Dent J 21(4):319-321.

* Copy of paper attached - Appendix E

WOODS R (1988)*
Twenty years of antibiotic sensitivity testing of dental infections. Part 2. A review. Aust Dent J 33(6):505-510.

WOODS R, BARNARD PD and CUTRESS T (1977)
Western Samoan school children I. Streptococcus mutans in dental plaque. Paper 31, I.A.D.R. Meeting (Aust. & NZ Div.) Perth, 1977.

WOODS R, TAYLOR CE and BARNARD PD (1979)
Streptococcus mutans in plaque of city school children. J Dent Res 58:2236 (Special issue D) Abst.

ZICKERT I, EMILSON CG and KRASSE B (1982)
Streptococcus mutans, lactobacilli and dental health in 13-14-year-old Swedish children. Com Dent Oral Epidemiol 10(2):77-81.

ZINNER DD and JABLON JM (1969)
Cariogenic streptococci in infants. Arch Oral Biol 14(12):1429-1431.

---

* Copy of paper attached - Appendix E

13. APPENDICES

ATTACHED APPENDICES

13.1 APPENDIX A.

STATISTICAL ANALYSES - EXAMPLES OF CALCULATIONS

To illustrate the method employed for statistical analysis and comparison of the *Streptococcus mutans* presence in groups of children and to determine the value of *'p'*.

The method used is illustrated by two calculations. The first calculation tests the increase in the proportion of children within the groups tested from whom no *S. mutans* could be isolated from dental plaque samples (Grade 1), the second calculation tests the decrease in the proportion of children with established *S. mutans* present (Grade 3) in their dental plaque.

Both examples test the results for the years 1970 (the baseline survey) and the final survey in 1986.

Basic data to test the significance of the increase in the number of children in the groups examined in 1970 and 1986 was obtained from Table 6. The number of children aged 5 to 8 years in 1970 whose plaque examination was classified Grade 1 (no *S. mutans* isolated) is 46, the balance of the group is (Grades 2 plus 3 from whom *S. mutans* could be isolated) is 79, a total of 125 children. The corresponding numbers of children surveyed in 1986 were 59 (Grade 1) and 45 (Grades 2 plus 3), a total of 104 children.

Testing the significance of the increase of children from whom no *S. mutans* could be isolated 1970/1986, a 2 x 2 table was constructed using these data.

The sum of the data left to right is placed in the fourth column, and the sum of the data verticaly is placed in the bottom line of the table.

	Grade 1 Children	Grades 2+3 Children	Total Children
1970	46	79	125
1986	59	45	104
	105	124	229

The anticipated value (a) for the table is calculated.

$$a = \frac{105 \times 125}{229} = \frac{13125}{229} = 57.31$$

This value (57.31) is inserted in the table next to 1770/Grade 1 children and the other anticipated values are obtained by cross table calculation. (The anticipated values are in brackets)

	Grade 1 Children	Grades 2+3 Children	Total Children
1970	46 (57.31)	79 (67.69)	125 (125)
1986	59 (47.69)	45 (56.31)	104 (104)
	105 (105)	124 (124)	229 (229)

Chi squared is calculated from the sum of four calculations.

$$\frac{(46 - 57.31)^2}{57.31} = 2.232$$

$$\frac{(59 - 47.69)^2}{47.69} = 2.682$$

$$\frac{(79 - 67.69)^2}{67.69} = 1.890$$

$$\frac{(45 - 56.31)^2}{56.31} = 2.272$$

$$\mathbf{Chi^2 = 9.076}$$

The drgree of freedom in this type of 2 x 2 calculation is 1 and the probability 'p' is calculated from statistical tables (Fisher and Yates 1953) and is in this example $< 0.01 > 0.001$.

The increase in the number of children from whom no *S. mutans* could be detected in their dental plaque (Grade 1) in the surveys 1970 and 1986 was found to be significant.

A similar calculatuion was been made to test the significance of the decrease in the number of children who had established *S. mutans* (Grade 3) present in their dental plaque. Similar tables were constructed and calculations performed.

Again, using data from Table 6, the number of children in the survey of 1970 from whom *S. mutans* in dental plaque was established (Grade 3) was 50, the number of children whose dental plaque did not have established *S. mutans* was 75. The comparative data from the survey in 1986 was 23 children who had established *S. mutans* (Grade 3) present in their dental plaque, and 81 children whose dental plaque did not have established *S. mutans* present.

	Grades 1+2 Children	Grade 3 Children	Total Children
1970	75	50	125
1986	81	23	104
	156	73	229

The anticipated value (a) is calculated.

$$a = \frac{156 \times 125}{229} = \frac{19500}{229} = 85.15$$

This value (85.15) is inserted in the table next to 1770/Grade 1 + 2 children and the other anticipated values are obtained by cross table calculation. (The anticipated values are in brackets)

	Grades 1+2 Children	Grade 3 Children	Total Children
1970	75 (85.15)	50 (39.85)	125 (125)
1986	81 (70.85)	23 (33.15)	104 (104)
	156 (156)	73 (73)	229 (229)

Chi squared is calculated from the sum of four calculations

$$\frac{(75 - 85.15)^2}{85.15} = 1.210$$

$$\frac{(81 - 70.85)^2}{70.85} = 1.454$$

$$\frac{(50 - 39.85)^2}{39.85} = 2.585$$

$$\frac{(23 - 33.15)^2}{33.15} = 3.108$$

$$\mathbf{Chi^2 = 8.357}$$

The degree of freedom in this type of 2 x 2 calculation is 1 and the probability 'p' is calculated from statistical tables (Fisher and Yates 1953) and is $< 0.01 > 0.001$.

The decrease in the proportion of children in whom *S. mutans* is established in their dental plaque (Grade 3) in the surveys 1970 and 1986 was found to be statistically significant.

13.2 APPENDIX B.

STREPTOCOCCUS MUTANS IN THE PLAQUE OF CHILDREN IN WESTERN SAMOA AND NEWTOWN*

In 1976, 25 school children aged five to nine years living in Western Samoa, were examined for the presence of *S. mutans* in their dental plaque (Woods *et al.* 1977). The examination for *S. mutans* in their plaque employed the same technique as used at Boorowa (Woods 1971a). In 1978, 143 school children age six to eight years at the inner Sydney suburb of Newtown were also examined to determine the presence of *S. mutans* in their dental plaque (Woods *et al.* 1979). The results of these examinations are set out in Table 15.

When tested using the chi squared method, the prevalence of *S. mutans* in the dental plaque of children at Newtown and Western Samoa did not differ significantly from that found in children at Boorowa in the baseline survey (1970). The value of 'p' was in all cases greater than 0.50. The calculations were made similarly to those illustrated in Appendix A. The data regarding children tested for *S. mutans* in their dental plaque was extracted from Table 15.

Table 15. Prevalence, (percent in parentheses) and number of children with *Streptococcus mutans* in dental plaque, Western Samoa and Newtown*.

S. mutans in dental plaque (Grade)	Newtown* Children	Prevalence (1978)	Western Samoa Children	Prevalence (1976)
Grade 1	46	(32)	5	(20)
Grade 2	64	(45)	8	(32)
Grade 3	33	(23)	12	(48)
Grades 2+3**	97	(68)	20	(80)

* Inner Sydney suburb.

** The sum of Grades 2 and 3 represents the prevalence of *S. mutans* in the dental plaque of the populations studied.

In 1976, school children in Western Samoa appeared from examination and discussion, to practise a low level of oral hygiene and their diet was well endowed with sucrose. There were no fluoridated water supplies, fluoride toothpaste, nor were fluoride supplements used.

In contrast the children examined at Newtown Public School used the fluoridated Sydney water supply and had received some oral hygiene instruction and preventive dentistry treatment from the School Dental Service. It is likely that in 1978 a large number of the children at Newtown used fluoride toothpaste.

The prevalence of *S. mutans* in plaque (Grades 2 plus 3) in the children examined at Newtown was 68 per cent with 23 per cent having *S. mutans* established in their dental plaque (Grade 3). In Western Samoa in 1976, the incidence of *S. mutans* in plaque was 80 per cent and in 48 per cent *S. mutans* was established.

At Newtown the prevalence of *S. mutans* in the dental plaque (Grades 2 plus 3) of children in 1978 was greater than found at any time during the study at Boorowa. However, the 23 per cent incidence of established *S. mutans* in plaque (Grade 3) was slightly higher than that found at Boorowa after the commencement of the community dental health programme but less than that found at Boorowa in 1970 before the scheme was introduced (Table 6).

In Western Samoa the prevalence of *S. mutans* in the plaque (Grades 2 plus 3) of the children examined was greater than found at any time during the studies at Boorowa. The prevalence of *S. mutans* established in dental plaque (Grade 3) was also higher than that found at Boorowa after the start of the programme, being similar to the incidence at Boorowa in the baseline survey, in 1970.

13.3 APPENDIX C.

REDUCTION IN DENTAL CARIES IN AUSTRALIAN CHILDREN

Since 1977 there has been a remarkable reduction in dental caries prevalence in Australian school children. This has been documented from data obtained from the Australian School Dental Service which collected and co-ordinated data from State and Territory School Dental Services (Commonwealth Department of Health 1987). Since 1986 data has been gathered by the Australian Institute of Health and Welfare Dental Statistics and Research Unit, located in Adelaide (AIHW Dental statistics unit 1992, 1995, Armfield *et al.* 1999, 2000).

The dental caries prevalence of the permanent dentition of Australian school children is set out in Tables 17 and 18. The dental caries prevalence has declined substantially since 1977. The DMFT for children age 8 years (for comparison with the oldest group in the Boorowa study), has fallen from 1.94 in 1977 to 0.30 in 1997, a reduction of 84.5 percent. The percent of caries free Australian children age 8 years, set out in Table 18, has risen from 33.91 percent in 1977 to 83.0 per cent in 1993.

The dental caries experience for children aged 8, living in Canberra, 1964 to 1974 was reported (Carr 1976). Canberra had introduced fluoride to the community water supplies in 1964. The DMFT of children 8 years of age was: 1964 - 2.69; 1970 - 1.75; 1971 - 1.59; 1972 - 1.00; 1973 - 1.07; 1974 - 0.89. These changes were attributed to the effects of fluoridation.

Dental caries prevalence of school children in New South Wales in 1954-1955 has been recorded (Barnard 1956). The dental caries experience of the permanent dentition of New South Wales school children age 6, 7 and 8 years in 1954-1955 is set out in Table 16.

In 1954-1955 the DMFT index of New South Wales school children age 8 years was 3.22 and the percent with a caries free permanent dentition at this age was 27.3. Compared with 1993 Australian data the reduction in the DMFT index 1954-1955 to 1993 has been 89.0 percent over 38 years. The increase in caries-free children age 8 years from 1954-55 to 1993 has been 66 per cent.

It is generally agreed that widespread community water fluoridation has been the foundation of the very substantial caries reduction over these years. This conclusion has been reached by the National Health and Medical Research Council who endorsed the findings of its Working Groups on the effectiveness of water fluoridation (National Health and Medical Research Council 1991).

In addition, the almost universal use of fluoride-containing toothpastes throughout Australia is likely to have had a major effect, complementing communal water fluoridation and reducing dental caries. The report of the National Health and Medical Research Council assesses the anti-caries effect of fluoride toothpaste as providing a caries reduction of approximately 25 per cent (National Health and Medical Research Council 1991, p84).

It is likely that fluoride from fluoridation of water supplies has been established in the Australian food chain as a trace element, and a secondary anti-caries effect of fluoridation, supplemented by the anti-caries effects of fluoride toothpaste, has reached beyond fluoridated communities to provide some anti-caries effect.

Continuing dental caries reduction in fluoridated areas

Notwithstanding this comment a differential in total caries experience continues between non-fluoridated and fluoridated communities. This has been shown in the survey of Slade, Spencer, Davies, Stewart (1996). "Caries rates were significantly lower in Townsville (fluoridated) than in Brisbane (non-fluoridated) both in the deciduous dentition (according to age of 32 to 55 percent fewer tooth surfaces affected) and the permanent dentitions (20 to 65 percent fewer tooth surfaces affected)".

Table 16. Dental caries experience of the permanent dentition in New South Wales state schools, (1954-1955) children (boys plus girls) aged 6 to 8 years (Barnard 1956)

All cases (urban plus rural)

Age	DMFT	Percent caries-free
6 years	0.99	58.7
7 years	2.31	23.5
8 years	3.22	10.5

Rural

Age	DMFT	Percent caries-free
6 years	0.80	65.1
7 years	2.04	29.0
8 years	2.99	14.3

Urban

Age	DMFT	Percent caries-free
6 years	1.30	47.4
7 years	2.79	13.4
8 years	3.64	3.7

13.4 APPENDIX D

DENTAL CARIES PREVALENCE OF THE PERMANENT DENTITION OF AUSTRALIAN CHILDREN AGES 6, 7 AND 8 YEARS

Table 17 Dental caries prevalence of the permanent dentition in Australian children* 1977 to 1997. 1977 - 86 (DIMFT) (Commonwealth Department of Health 1987), 1990 (AIHW Dental statistics unit 1992), and 1993, 1997 (DMFT) (AIHW Dental statistics unit 1995)**

Age	1977	1978	1979	1980	1981	1982	1983	1984	1985	1986	1990	1993	1997
6 years	0.51	0.43	0.31	0.25	0.18	0.16	0.12	0.12	0.10	0.09	0.10	0.08	0.07
7 years	1.21	1.09	0.86	0.71	0.58	0.47	0.42	0.37	0.34	0.34	0.24	0.22	0.17
8 years	1.94	1.75	1.46	1.23	1.01	0.92	0.76	0.69	0.61	0.60	0.42	0.36	0.30

* Boys plus girls
**Armfield *et al. (1999, 2000)*

Table 18 Caries free Australian schoolchildren*, percent, permanent dentition 1977 - 97. (Commonwealth Department of Health 1987), 1990 (AIHW Dental statistics unit 1992). and 1993, 1997 (AIHW Dental statistics unit 1995)

Age	1977	1978	1979	1980	1981	1982	1983	1984	1985	1986	1990	1993	1997
6 years	77.84	80.72	85.10	87.13	90.13	90.90	92.63	92.57	93.61	94.70	95.3	95.2	96.5
7 years	53.39	56.58	63.74	68.58	72.48	76.20	78.10	80.33	81.21	81.60	86.1	87.7	89.9
8 years	33.91	38.58	45.38	51.35	57.19	59.45	64.83	66.85	69.84	70.14	77.9	79.7	83.0

* Boys plus girls

13.5 APPENDIX E

ORIGINAL PUBLICATIONS ON *S. MUTANS* AND THE
BOOROWA DENTAL HEALTH PROGRAMME

(Appendix E)

116 Australian Dental Journal, April, 1971

A dental caries susceptibility test based on the occurrence of Streptococcus mutans in plaque material

Robin Woods

Introduction

Although *Streptococcus mutans* has been associated with dental caries since it was described by Clarke in 1924, it was not until the last decade that a suitable animal experimental caries system was devised which could test the caries-producing effect of this organism. Fitzgerald and Keyes[1] induced dental caries in a strain of apparently "caries inactive" hamsters by introducing a strain of streptococci (later classified by Carlsson[2] as *Str. mutans*) isolated from "caries susceptible" hamsters. Larsen and Fitzgerald[3] in 1968 using a strain of *Str. mutans* isolated from a human source induced dental caries in the African White Tailed Rat. Likewise, dental caries has been induced in rats by Gibbons[4] and in hamsters by Krasse.[5]

Keyes,[6] by reviewing the role of plaque-forming streptococci concludes, "The potential of certain strains of streptococci to induce plaque and multisurface cavitation has been conclusively established in animals and strongly implicated in humans." Quite a few of the strains of streptococci referred to resemble *Str. mutans* although other species of streptococci (e.g., *Streptococcus sanguis* and *Streptococcus salivarius*) may also be implicated in the development of dental caries.

It appeared reasonable to see whether any relationship could be established between the occurrence of *Str. mutans* in plaque material and the dental caries experience of patients, in order to establish a means of determining the microbial challenge from this organism in individual cases. As salivary lactobacillus counts and dental caries experience have frequently been related it was decided also to test this relationship.

Materials and methods

Forty-four patients aged 6-15 years receiving regular dental care in a private practice were examined early in 1969. Some of the patients live in an area where the public water supply is fluoridated, some live beyond the water reticulation system and received a fluoride supplement in tablet form (these cases are marked

Received for publication April, 1970.

(1) Fitzgerald, R. J., and Keyes, P. H.—Demonstration of the etiologic role of streptococci in experimental caries in the hamster. J.A.D.A., 61: 1, 9-19 (July) 1960.

(2) Carlsson, J.—A numerical taxonomic study of human oral streptococci. Odont. Revy., 19: 2, 137-160, 1968.

(3) Larson, R. H., and Fitzgerald, R. J.—Caries development in the African white tailed rat (*mystromys albicaudatus*) infected with a streptococcus of human origin. J. D. Res., 47: 5, 746-749 (Sept.-Oct.) 1968.

(4) Gibbons, R. J., Berman, K. S., Knoettner, P., and Kapsimalis, B.—Dental caries and alveolar bone loss in gnotobiotic rats infected with capsule forming streptococci of human origin. Arch. Oral. Biol., 11: 6, 549-560 (June) 1966.

(5) Krasse, B.—Human streptococci and experimental caries in hamsters. Arch. Oral Biol., 11: 4, 429-436 (Apr.) 1966.

(6) Keyes, P. H.—Research in dental caries. J.A.D.A., 76: 6, 1357-1373 (June) 1968.

(Appendix E)

"f" in Tables 1, 2 and 3) others moved to the district recently and had an uncertain fluoride history. All received a topical application of acidulated phosphate-fluoride solution at the outset of the test as part of their treatment. Radiographic examinations with bitewing films were made at the beginning and end of the survey. Samples of plaque material and stimulated saliva were obtained at the beginning of the survey.

Plaque samples

Plaque samples were obtained on sterile pieces of toothpick (one for each quadrant of the dentition, from the gingival third of the buccal and labial aspects of the posterior and anterior teeth in the arch) and placed in 5 ml of Ringer's solution and shaken until a homogeneous suspension was obtained. The samples were all taken in the mid afternoon and placed on media within 3 hours.

Analysis of plaque sample

The plaque suspension was diluted and equal amounts (0·01 ml) were spread in mitis salivarius agar (Oxoid), which had 1 mg per ml sulphadimidine included, and incorporated within tryptone glucose yeast agar (Oxoid, Plate Count Agar). The sulphadimidine was added as suggested by Carlsson[7] who described the medium as selective for *Str. mutans*. The plates were incubated aerobically for 72 hours before they were examined. The colonies grown within the tryptone glucose agar were counted and the number per ml of the plaque suspension calculated. Colonies of *Str. mutans* grown on the mitis salivarius agar were counted, recognition being on the basis of colony form and biochemical tests of representative colonies which were subcultured. A low power microscope was used for counting and recognizing the colonies. The count was expressed per 1,000 colonies grown on the tryptone glucose yeast agar, the latter figure being taken as representative of the total cultivable count. Where no *Str. mutans* was detected the minimum number that could have been detected was calculated.

Screening test

In order to try a simple screening test a second treatment of the plaque samples was devised. A 7 mm platinum loop was used to place one streak of the undiluted plaque suspension across the surface of the sulphadimidine fortified mitis salivarius agar. Three such streaks could be accommodated on one petrie dish of agar with ease. After 72 hours aerobic incubation ten random fields were examined and the total colonies in the ten fields recorded. The results were placed into the following categories:

Group 1—no colonies.
Group 2—less than eight colonies (but more than zero).
Group 3—eight colonies or more.

On an empirical basis seven colonies per 10 fields was considered the upper level representative of a chance, transient *Str. mutans* being present in the sample of plaque. Values greater than 7 indicate a predominance of this type of organism and would suggest that the plaque material had a high concentration of *Str. mutans*.

Lactobacillus count

The stimulated saliva sample was diluted and 0·01 ml was spread on tomato juice agar (Oxoid) and incubated aerobically for 72 hours. Lactobacillus colonies were identified by the morphology of the bacteria when stained and examined microscopically. The lactobacillus counts were expressed per ml of stimulated saliva.

Recognition of Str. mutans

In general, *Str. mutans* is recognized by its colony form. Krasse[5] describes the appearance of colonies on mitis salivarius agar, ". . . the elevation of the colony was highly convex and the edge undulate; the internal structure was finely granular, reminiscent of frosted glass, the colour light bluish-grey in reflected light and grey to dark blue in transmitted light. The size of the colonies after 24 hours incubation was about 0·5 mm in diameter. Sometimes polysaccharide formation was observed as a glistening drop on the top of the colony."

Subcultures of representative colonies were made and tested for their ability to form zooglia in 5 per cent sucrose broth and ferment mannitol. Their hæmolytic effect on horse blood was also recorded. Carlsson,[2] Krasse and Carlsson[8] and Edwardsson[9] enumerated the

[7] Carlsson, J.—A medium for isolation of streptococcus mutans. Arch. Oral Biol., 12: 12, 1657-1658 (Dec.) 1967.

[8] Krasse, B., and Carlsson, J.—Various types of streptococci and experimental caries in hamsters. Arch. Oral Biol., 15: 1, 25-32 (Jan.) 1970.

[9] Edwardsson, S.—Characteristics of caries-inducing human streptococci resembling streptococcus mutans. Arch. Oral Biol., 13: 6, 637-646 (June) 1968.

(Appendix E)

biochemical characteristics of *Str. mutans* and other oral streptococci. Of 12 strains of *Str. mutans* reported one only was unable to ferment mannitol. Ewardsson[9] reported on 49 strains of caries-inducing streptococci isolated from human dental plaque, and reported that they could all ferment mannitol. Mannitol fermentation is a characteristic which with its resistance to sulphonamides helps distinguish *Str. mutans* from other zooglia forming streptococci.

Most of the colonies which were subcultured were hard when touched with a platinum wire although occasional colonies were "granular" and broke easily when touched.

Dental caries experience

Caries experience was evaluated for a period of 11-13 months and was based on the two examinations. Because a number of the patients were losing deciduous teeth the caries experience was corrected and expressed as a rate per 100 teeth for each patient. The rate was corrected for time also. The final expression of the caries experience was new carious lesions per year per 100 teeth.

TABLE 1

Results of bacteriological tests and the dental caries experience of 21 patients

Case number	New carious lesions per year per 100 teeth	*Str. mutans* concentration per 1000*	Salivary lacto-bacillus count*** per ml
1	0·00 (22)	0·040* (24)	600 (13)
2** f	0·00 (22)	0·043* (24)	3,300 (7)
3 f	8·34 (13)	0·191* (24)	99 (23)
4 f	0·00 (22)	0·081* (24)	99 (23)
5	0·00 (22)	0·094* (24)	6,000 (3)
6** f	0·00 (22)	0·071* (24)	99 (23)
7** f	0·00 (22)	0·085* (24)	99 (23)
8** f	0·00 (22)	0·075* (24)	99 (23)
9** f	0·00 (22)	0·019* (24)	99 (23)
10** f	0·00 (22)	0·170* (24)	99 (23)
11	0·00 (22)	0·068* (24)	600 (13)
12** f	0·00 (22)	0·183* (24)	99 (23)
13** f	0·00 (22)	0·072* (24)	99 (23)
14** f	0·00 (22)	0·171* (24)	99 (23)
15 f	0·00 (22)	0·085* (24)	1,000 (12)
16** f	0·00 (22)	0·047* (24)	600 (13)
17** f	0·00 (22)	0·085* (24)	600 (13)
18 f	0·00 (22)	0·080* (24)	99 (23)
19	3·86 (19)	0·095* (24)	99 (23)
20 f	3·86 (19)	0·090* (24)	99 (23)
21	7·79 (15)	0·070* (24)	99 (23)

The rank of each result is bracketed.

Using the screening test, these cases are all classified as Group 1, and the screening test rank is 24 in every case.

** Totally caries-free.

f "Fluoride" patients.

* No organisms isolated in these cases, these values represent the minimum limit of the test.

*** Lactobacillus counts of zero are recorded as 99.

Results

The results of the examinations are presented in Tables 1, 2 and 3. The patients are grouped according to the results of the screening test, Group 1 in Table 1, Group 2 in Table 2, and Group 3 in Table 3. The results of the various tests are listed and the rank or order of magnitude of the particular result is listed with the highest value being allocated the first rank.

TABLE 2

Results of bacteriological tests and the dental caries experience of 11 patients

Case number	New carious lesions per year per 100 teeth	*Str. mutans* concentration per 1000	Salivary lacto-bacillus count*** per ml
22	0·00 (22)	0·872 (13)	99 (23)
23 f	7·79 (15)	0·340 (20)	99 (23)
24 f	0·00 (22)	0·592 (14)	99 (23)
25	0·00 (22)	0·278 (21)	2,500 (9)
26	4·75 (18)	0·536 (17)	600 (13)
27 f	0·00 (22)	0·398 (19)	99 (23)
28 f	7·50 (17)	0·562 (16)	400 (20)
29	3·54 (21)	0·574 (15)	6,000 (3)
30	0·00 (22)	0·128 (23)	600 (13)
31	8·40 (12)	0·253 (22)	99 (23)
32	0·00 (22)	1·065 (12)	400 (20)

The rank of each result is bracketed.

Using the screening test, these cases are all classified as Group 2, and the screening test rank is 13 in every case.

f "Fluoride" patients.

*** Lactobacillus counts of zero are recorded as 99 per ml.

The results of the various tests were compared with the caries experience. The statistical treatment was based on ranking tests and the statistical values are presented in Table 4. The relationship between the screening test and caries experience is summarized in Table 5.

The results of the biochemical tests are summarized in Table 6.

(Appendix E)

TABLE 3

Results of bacteriological tests and the dental caries experience of 12 patients

Case number	New carious lesions per year per 100 teeth	*Str. mutans* concentration per 1000	Salivary lactobacillus count*** per ml
33	19·20 (3)	24·800 (5)	40,000 (2)
34 f	19·40 (2)	118·000 (2)	5,000 (6)
35	12·60 (6)	1·600 (10)	5,200 (5)
36 f	15·40 (4)	1·580 (11)	400 (20)
37	21·40 (1)	2·130 (9)	1,200 (11)
38	10·70 (10)	7·500 (7)	2,700 (8)
39	12·50 (7)	46·000 (3)	400,000 (1)
40	8·34 (13)	0·420 (18)	1,300 (10)
41	12·20 (8)	238·000 (1)	99 (23)
42	11·53 (9)	2·160 (8)	500 (19)
43 f	10·70 (10)	32·000 (4)	99 (23)
44	15·00 (5)	8·960 (6)	99 (23)

The rank of each result is bracketed.

Using the screening test, these cases are classified as Group 3 and are all ranked 1.

f "Fluoride" patients.

*** Lactobacillus counts of zero are recorded as 99.

TABLE 4

Statistical relationships between caries experience and bacteriological tests

Caries experience and . . .		"P" value	
Str. mutans present per 1000 cultivable organisms	From plaque	<0·01	Significant
Str. mutans screening test	From plaque	<0·05	Significant
Salivary Lactobacillus count		<0·10 >0·50	Not significant

Discussion

There is a significant relationship between both the tests performed on *Str. mutans* in plaque material and the dental caries experience. Of the 21 patients listed in Group 1, according to the screening test only 4 developed any dental caries and the degree was equivalent to two cavities or less per year requiring restoration (Table 5). Of these cases X-ray examination was necessary to detect the lesions

TABLE 5

Screening test results related to dental caries experience

Screening test		Number of cases	Dental caries experience		
Grade	Colonies per 10 fields		Number of cases where caries occurred	Percentage of cases where caries occurred	Dental caries experience (New lesions per year per 100 teeth)
1	No colonies	21	4	19	0·00– 8·34
2	Less than 8 colonies	11	5	45	0·00– 8·64
3	8 colonies or more	12	12	100	8·34–21·40

TABLE 6

Biochemical reactions of Str. mutans

Number of cases	Mannitol fermentation		Hæmolysis			Sulphonamide resistance	Zooglia formation in 5% sucrose broth
33 (75)	+		alpha			+	+
4 (9)		—	alpha			+	+
3 (7)	+				gamma	+	+
2 (4·5)	+			beta		+	+
2 (4·5)		—			gamma	+	+
Total: 44 (100)	38 (86)	6 (14)	37 (84)	2 (5)	11 (11)	44 (100)	44 (100)

Percentages in brackets.

(Appendix E)

in two cases (Cases 3 and 21), the other cases were the involvement of the occlusal fissures of the second permanent molar. Of those patients listed as Group 2, five of the eleven developed dental caries, again the degree of the dental caries was of the same order as experienced by the patients listed as Group 1. The twelve patients of Group 3 all developed dental caries, the number of lesions developed each year was higher than patients in the other two groups in all cases except one (Case 40). Patients classified in Group 3 are the patients requiring the greatest degree of dental caries treatment either by restoration or prevention.

Although there was an overall trend showing some relationship between the salivary lactobacillus count and the dental caries experience the relationship was not significant (Table 4). Low lactobacillus counts occurred in quite a few cases where caries was experienced, and relatively high lactobacillus counts in cases where there was no dental caries. These findings are in agreement with the experience of Goldsworthy and Spies[10] who in 1958 reported lactobacillus counts of varying magnitude among a group of caries-free children and high counts in 20 per cent of these cases.

There are 11 caries-free children listed in Group 1, and 3 of these children (27 per cent) had lactobacillus counts up to 3,300 per ml of saliva (Table 1).

The number of cases where *Str. mutans* could not be isolated (Group 1) is 21 of a total of 44 (48 per cent). This is in general agreement with the results of Jordan, Englander and Lim[11] who reported on patients living in an area where the water supply was fluoridated, a range from 47-74 per cent. The results I have reported are higher than the 20 per cent of patients free of *Str. mutans* reported by Krasse and his co-workers[12] in their studies of Swedish students. The dental caries of the patients in the study reported by Krasse and his co-workers[12] was higher than that experienced by the patients reported on by Jordan, Englander and Lim[11] or myself.

The biochemical reactions reported in Table 6 are in agreement with those reported by Carlsson[2] although in a more recent study Krasse and Carlsson[8] include as *Str. mutans* two additional strains which are not resistant to sulphafurazole. As the original isolating media I used contained 1 mg per ml of sulphadimidine it can be concluded that any organism isolated is resistant to this drug.

The experience gained identifying *Str. mutans* has confirmed the observation of Jordan, Englander and Lim[11] who recognized caries-inducing streptococci by their colonial morphology in mitis salivarius agar. Edwardsson[9] was able to grow the species of *Str. mutans* he reported aerobically at 37° C for 48 hours; it was my experience that these organisms could be grown aerobically, although growth is slower than anaerobic cultivation.

The hazards of attempting to predict the likely caries experience for a patient over a period of 12 months from a test which reflects the plaque conditions at a given moment are obvious, yet there is a good correlation in this study between caries experience and the presence or absence of *Str. mutans* in dental plaque material.

The incidence of dental caries in the community will be reduced by the adoption of the multiple fluoride techniques, by approximately 80 per cent; under these circumstances it is more important to separate those patients who have the potential to develop the disease from those who have not. It is to this end that the screening test I am suggesting has been developed. It requires a minimum of laboratory glassware and is extremely economical in its use of media. Using the test large sections of the population can be screened; there is no difficulty screening patients as part of their routine dental examination.

The results of the test may be used to plan more intensive forms of therapy directed towards reduction of dental caries for those patients who need them.

Any test based on identifying bacteria associated with an infectious disease is a measure of the bacterial challenge to the host. The reaction of the host to this challenge will vary with the resistance of the host, however, if there is no bacterial challenge there can be no disease. With this in mind the screening test I have described is based on detecting patients who do not harbour *Str. mutans*, rather than

---

(10) Goldsworthy, N. E., and Spies, H. C.—The biology of children of Hopewood House, Bowral, N.S.W. II. Observations extending over five years (1952-56 (inclusive). Pt. 3. The lactobacillus count and its relation to dental caries. Austral. D. J., 3: 5, 318-330 (Oct.) 1958.

(11) Jordan, H. V., Englander, H. R., and Lim, S.—Potentially cariogenic streptococci in selected population groups in the western hemisphere. J.A.D.A., 78: 6, 1331-1335 (June) 1969.

(12) Krasse, B., Jordan, H. V., Edwardsson, S., Svensson, I., and Trell, L.—The occurrence of certain "caries-inducing" streptococci in human dental plaque material. Arch. Oral Biol., 13: 8, 911-918 (Aug.) 1968.

(Appendix E)

to attempt to grade the results of the tests in order of magnitude. Once a person is liable to the disease it is much more difficult to predict the precise nature of the disease in the individual.

Conclusions

It has been shown that there is a significant relationship between the plaque concentration of *Str. mutans* and the subsequent dental caries experience of a number of patients in a private dental practice.

A screening test has been described, based on the presence of the organism in plaque and a significant relationship has been demonstrated between the results of this test and subsequent dental caries experience.

There was no significant relationship between the salivary lactobacillus counts and subsequent dental caries.

The implication of these findings have been discussed, and the use to which a caries susceptibility test can be put has been pointed out.

Summary

Some of the literature referring to the dental caries inducing effect of *Str. mutans* has been reviewed.

The concentration of the streptococci in dental plaque, a screening test based on the presence of the organism in plaque and salivary lactobacillus counts have been compared to the dental caries experience of the individual patients upon whom the tests were made.

The results of these tests and the implications of these results have been discussed in the light of current research into the infectious nature of dental caries.

P.O. Box 22,
Yass, N.S.W. 2582.

AUSTRALASIAN MEDICAL PUBLISHING CO. LTD.
71-79 ARUNDEL ST., GLEBE, SYDNEY, N.S.W., 2037

(Appendix E)

152 Australian Dental Journal, June, 1971

The short-term effect of topical fluoride applications on the concentration of Streptococcus mutans in dental plaque

Robin Woods

Introduction

Although the anti-caries effect of fluorides is well established, it is yet uncertain by which mechanism this is achieved. Stookey,[1] reviewing fluoride therapy, discussed two possible mechanisms: first, the bacteriostatic influence on the oral flora based on the well-known enzyme inhibition properties of fluoride ions released from enamel; second, the incorporation of fluoride within the hydroxy-apatite of enamel, resulting in a lowered solubility of crystals of enamel apatite in weak organic acids.

Briner and Frances[2] constructed an experimental model closely resembling the tooth-plaque relationship in order to examine the acid production by strains of *Lactobacillus casei* when these systems contained dental enamel treated with sodium and stannous fluorides. They reported an inhibition of lactic acid production when the system included fluoride-treated enamel.

More recently Margalit and Gedalia[3] confirmed that the fluoride content of the enamel of teeth treated *in vitro* with NaF + Na_2HPO_4 or NaF alone was significantly higher than the untreated control. They showed also that the fluoride content of saliva, up to eight days after topical application of NaF + Na_2 HPO_4 or NaF solutions, was substantially higher than before the application. Richardson[4] had demonstrated the removal of fluoride from enamel after its deposition from a topical fluoride by washing. Mellberg, Laakso and Nicholson,[5] who also tested fluoride loss from enamel treated topically with fluorides, concluded that fluoride acquired by tooth enamel from topical solutions is not of a permanent nature.

Fluorides applied topically to the teeth, as an acidulated fluoride solution[6] or contained in a 9 per cent SnF_2 prophylactic paste used in either a non-fluoride area[7] or an area with an optimum amount of fluoride in the water supply,[8] have been shown to be effective means of preventing dental caries. More recently the self-application of a stannous fluoride-zirconium silicate[9] paste has been shown to be an effective means of reducing dental caries in school children.

The relationship of *Streptococcus mutans* to dental caries has been reviewed,[10] and there is evidence that the presence of the micro-organism in the dental plaque is related to human dental caries.

Received for publication July, 1970.

(1) Stookey, G. K.—Fluoride therapy. In improving dental practice through preventive measures. Bernier, J. L., and Muhler, J. C. (ed.). St. Louis, The C. V. Mosby Co., Ch. 5, 76-80.

(2) Briner, W. W., and Francis, M. D.—The effect of enamel fluoride on acid production by lactobacillus casei. Arch. Oral Biol., 7:5, 541-550 (Sept.-Oct.) 1962.

(3) Margalit, D., and Gedalia, I.—Release of fluoride into saliva after topical fluoride application. J. D. Res., 48:1, 93-96 (Jan.-Feb.) 1969.

(4) Richardson, B.—Fixation of topically applied fluoride in enamel. J. D. Res. (suppl.), 46:1, 87-91 (Jan.-Feb.) 1967.

(5) Mellberg, J. R., Laakso, P. V., and Nicholson, C. R.—The aquisition and loss of fluoride by topically fluoridated human tooth enamel. Arch Oral Biol., 11:12, 1213-1220 (Dec.) 1966.

(6) Wellock, W. D., and Brudevold, F.—A study of acidulated fluoride solutions—II. The caries inhibiting effect of single annual topical applications of an acidic fluoride and phosphate solution. A two year experience. Arch. Oral Biol., 8:2, 179-182 (Mar. Apr.) 1963.

(7) Bixler, D., and Muhler, J. C.—Effect on dental caries in children in a non fluoride area of: Combined use of three agents containing stannous fluoride: a prophylactic paste, a solution and a dentifrice. J.A.D.A., 68:6, 792-800 (June) 1964.

(8) Gish, C. W., and Muhler, J. C.—Effect on dental caries in children in a natural fluoride area of combined use of three agents containing stannous fluoride: a prophylacic paste, a solution and a dentifrice. J.A.D.A., 70:4, 914-920 (April) 1965.

(9) Muhler, J. C.—Mass treatment of children with a stannous fluoride-zirconium silicate self-administered prophylactic paste for partial control of dental caries. J. Amer. Coll Dent., 35:1, 45-57 (Jan.) 1968.

(10) Woods, R. G.—A dental caries susceptibility test based on the occurrence of streptococcus mutans in plaque material. Austral. D. J., submitted for publication April, 1970.

(Appendix E)

Australian Dental Journal, June, 1971 153

The use of a prophylactic paste prior to or concurrently with the topical application of fluorides removes the dental plaque and the mucinous cuticle adjacent to the tooth to be treated. After the fluoride treatment there is a loss of the fluoride taken up by the tooth surface, which continues for at least seven days. The plaque and the mucinous cuticle have to be reestablished in an environment containing an amount of fluoride leached from the tooth surface.

In this investigation it was desired to examine the effect of the topical fluoride treatment and the subsequent fluoride loss on the reestablishment of *Str. mutans* after approximately one week.

Materials and Methods

Four groups of patients whose ages ranged from six to fifteen years were included in these studies. All patients had *Str. mutans* present at the tooth surface at the beginning of the study.

The twelve patients in the first group were given a dental prophylaxis with a pumice based paste containing 9 per cent SnF_2; then an acidulated fluoride solution[9] was applied for four minutes. The eight patients of the second group received the prophylaxis only.

The third group of thirteen school children were given a topical fluoride application using only a tooth brush and the stannous fluoride-zirconium silicate self-prophylactic paste described by Muhler.[9]

The fourth group of ten patients received a prophylaxis using a non-fluoride pumice prophylactic paste only.

No patient was given any special instruction concerning oral hygiene or diet.

Bacteriological examination

Plaque samples from the buccal and labial aspects of all teeth present were obtained immediately before the topical fluoride treatment or the prophylaxis and again one week later. These samples were all taken in the mid afternoon. The plaque scrapings were suspended in Ringer's solution and equal amounts of this suspension were spread on Mitis salivarius agar (Oxoid) with 1 mg./ml. sulphadimidine included and incorporated within tryptone glucose yeast agar (Oxoid). The details of this technique have been previously described in full.[10] Colonies of *Str. mutans* were identified on the Mitis salivarius agar and the concentration of these colonies was expressed as the number present per 1,000 colonies cultivated on the tryptone glucose yeast agar.

Results

The results of the tests of the *Str. mutans* concentration at the tooth surface before and one week after the topical fluoride application or the prophylaxis are presented in Tables 1-4. The mean values for concentration of *Str. mutans* in the various groups are presented in Table 5.

The statistical analysis of the results is based on the differences before and after treatment in the individual cases. These differences were tested for a significant variation from zero. The results of these analyses are presented in Table 5.

The concentration of *Str. mutans* at the tooth surface one week after any of the topical fluoride treatments described was lowered significantly. It was lowered in all the cases of Groups 1 and 2; 11 of 14 cases of Group 3; and in 6 of 10 cases only of Group 4.

Discussion

The topical fluorides applied in these studies were preceded by a prophylaxis using pumice paste in Groups 1 and 2; in Group 3 the controlled abrasiveness of the paste is a feature. In these methods of fluoride application the tooth surface is cleaned and polished, the plaque is removed, and new plaque is established after treatment. In this respect the tests reported are a measure of the effect of topical fluoride treatment on the reestablishment of *Str. mutans* at the tooth surface.

Jordan, Englander and Lim[11] reported that repeated application of a fluoride gel had no noticeable effect on the distribution of *Str. mutans* in groups of school children, corroborating the findings of Englander and Keyes,[12] who used hamsters.

Both of these investigations tested fluoride gels applied to the teeth, with no other preparation, and there was no attempt at plaque removal prior to treatment. These tests[11][12] on the distribution of *Str. mutans* were a measure of the effect of fluoride

(11) Jordan, H. V., Englander, H. R., and Lim, S.—Potentially cariogenic streptococci in selected population groups in the western hemisphere. J.A.D.A., 78: 6, 1331-1335 (June) 1969.

(12) Englander, H. R., and Keyes, P. H.—The prevention of dental caries in the Syrian hamster after repeated topical application of sodium fluoride gels. J.A.D.A., 73: 6, 1342-1347 (Dec.) 1966.

(Appendix E)

application on established plaque containing this organism. This is in distinction to testing the reestablishment of the organism on a fluoride-treated tooth surface after cleaning and plaque removal.

TABLE 1

The concentration of Str. mutans *at the tooth surface in individual cases before and one week after the topical application of acidulated fluoride solution to the teeth preceded by a dental prophylaxis using a 9 per cent SnF_2 prophylactic paste. (Group 1)*

Str. mutans concentration per 1,000 plaque organisms before treatment	*Str. mutans* concentration per 1,000 plaque organisms one week after treatment	Reduction of *Str. mutans* concentration after treatment
23·80	0·05	23·75
1·60	<0·27	1·33
179·00	10·90	168·10
1·58	0·05	1·53
76·60	0·54	76·60
3·70	<0·14	3·56
1·17	0·89	0·27
2·67	1·60	1·07
7·51	0·65	6·86
1·73	<1·17	0·56
0·39	<0·08	0·31
34·20	12·10	22·10

TABLE 2

The concentration of Str. mutans *at the tooth surface in individual cases before and one week after a dental prophylaxis using a 9 per cent SnF_2 paste alone. (Group 2)*

Str. mutans concentration per 1,000 plaque organisms before treatment	*Str. mutans* concentration per 1,000 plaque organisms one week after treatment	Reduction of *Str. mutans* concentration after treatment
0·35	0·11	0·24
0·77	<0·57	0·20
1·23	0·07	1·16
46·00	15·09	30·91
7·12	1·24	5·88
1·64	<1·49	0·15
1·25	<0·11	1·14
7·56	1·40	6·16
71·90	4·40	67·50
5·68	4·91	0·77

The protection given teeth against caries by topical fluorides most likely arises from incorporation of fluoride within the enamel structure. It has been reported[12] that *Str. mutans* can colonise and form plaque on the teeth of hamsters protected by topical fluorides, but is not associated with demineralization or cavitation. Notwithstanding these results, it should be clear that after topical fluoride applications there is a considerable loss of fluoride from the teeth and this fluoride is capable of disturbing the bacteria in the plaque. It should be noted that of the tests reported here, a pumice prophylaxis with a non-fluoride paste alone (Group 4) did not

TABLE 3

The concentration of Str. mutans *at the tooth surface in individual cases before and one week after the application of stannous fluoride-zirconium silicate self prophylactic paste. (Group 3)*

Str. mutans concentration per 1,000 plaque organisms before treatment	*Str. mutans* concentration per 1,000 plaque organisms one week after treatment	Reduction of *Str. mutans* concentration after treatment
224·00	29·80	194·20
23·40	25·60	—2·20
78·10	67·00	11·10
1·83	<0·20	1·63
4·00	1·07	2·93
4·26	5·10	—0·84
4·62	0·66	3·96
4·25	5·32	—1·07
2·98	0·20	2·78
28·60	28·10	0·50
4·20	<0·18	4·02
5·66	1·90	3·76
0·98	<0·25	0·73

TABLE 4

The concentration of Str. mutans *at the tooth surface in individual cases before and one week after a dental prophylaxis using a non-fluoride prophylactic paste only. (Group 4)*

Str. mutans concentration per 1,000 plaque organisms before treatment	*Str. mutans* concentration per 1,000 plaque organisms one week after treatment	Reduction of *Str. mutans* concentration after treatment
23·40	85·00	—61·60
12·80	80·00	—67·20
106·50	83·00	23·50
4·06	2·77	1·29
3·55	1·66	1·89
1·06	2·13	—1·07
8·14	2·13	6·04
0·21	0·20	0·01
0·53	2·28	—1·75
85·00	21·28	63·72

(Appendix E)

result in a significant reduction of the organisms at the tooth surface.

Obviously it seems likely that if topical fluorides are applied with sufficient frequency they might prevent the reestablishment of *Str. mutans* altogether at the tooth surface. The frequency of such applications would have to be determined. In these circumstances topical fluorides may reduce dental caries by eliminating the microbial challenge. Less frequent applications most likely depend on changes in the structure of the enamel apatite brought about by the fluoride for the anti-caries effect.

In order to obtain greater insight into the role of *Str. mutans* in dental caries and the effect of topical fluoride applications, long-term longitudinal studies will have to be made.

Summary and Conclusions

Three methods of topical fluoride application, each involving cleaning of the teeth and removal of existing plaque prior to or concurrently with the fluoride application, have been described, and their effect on the reestablishment of *Str. mutans* at the tooth surface has been compared with the effect of a prophylaxis using a non-fluoride pumice paste alone.

It has been shown that after topical fluoride treatment the concentration of organisms in the reestablished dental plaque seven days later is reduced significantly. Simple prophylaxis with a non-fluoride paste alone did not achieve this effect.

The concentration of *Str. mutans* in plaque can be used as an indication of the microbial challenge to the tooth to produce dental caries, only if the test is made before a topical fluoride application by these methods. It is not known how long after the fluoride application that the concentration of the organism in the plaque is affected. The effect is a short-term one recorded seven days after the application of fluoride.

It has been suggested that the reestablishment of *Str. mutans* in the dental plaque after topical fluoride treatment may be affected by the escape of fluoride from the tooth surface and the consequent higher fluoride concentration in the region adjacent to the tooth surface.

TABLE 5

The effect of various methods of topical fluoride application and also a non-fluoride prophylaxis on the concentration of Str. mutans *at the tooth surface*

Type of fluoride application or prophylaxis	Number of Cases	Mean *Str. mutans* concentration per 1,000 plaque organisms before treatment	Mean *Str. mutans* concentration per 1,000 plaque organisms one week after treatment	Significance of reduction ("P" values)
Prophylaxis (9 per cent SnF_2) paste plus acidulated fluoride. (Group 1) ..	12	27·81	3·29	P<0·001
Prophylaxis with 9 per cent SnF_2 paste only. (Group 2)	10	14·35*	2·94	P<0·001
SnF_2-zircate self prophylaxis paste. (Group 3)	13	29·76	12·87	P<0·05 >0·01
Non-fluoride prophylaxis only. (Group 4)	10	24·53	28·05	Not significant

* The concentration of organisms in Group 2 may have been low because these patients were all seen during the May (1969) school holidays, a time of the year when the average count appears to be lower than at other times. Also, these patients as a group are experiencing very little dental caries activity.

P.O. Box 22,
Yass, N.S.W. 2582

AUSTRALASIAN MEDICAL PUBLISHING CO. LTD.
71-79 ARUNDEL ST., GLEBE, SYDNEY, N.S.W., 2037

(Appendix E)

WOODS R (1973)
Transmission of Streptococcus mutans among schoolchildren aged 7 to 8 years. J Dent Res 52(3):576, (Abst).

Abstract of paper (37) presented to the Australia-New Zealand Division of the International Association of Dental Research, Sydney Australia August 9-11 1972.

37. TRANSMISSION OF *STREPTOCOCCUS MUTANS* AMONG SCHOOLCHILDREN AGED 7 TO 8 YEARS - R.G.Woods PO Box 22 Yass NSW 2582

Pencils used communally by schoolchildren were tested to determine if they were a vector in the transmission of *Streptococcus mutans* and other oral flora. In some schools pencils are communal property; they are distributed each morning and collected at the end of each day. *S. mutans*E is distributed widely in the dental plaque of schoolchildren; the incidence seems to increase with age. Dental plaque from 28 children (ages 7 to 8) was examined for *S. mutans, Candida* and *Lactobacillus*. Pencil tops were also taken, left over-night, and examined for these microorganisms. Plaque samples and pencil tops were placed in Ringer's solution to make a suspension, plated on Mitis Salivarius agar (with 1 mg/ml sulfadimidine) and tomato juice agar. Microorganisms were identified by colony form and morphology. *S. mutans* was tested for ability to ferment mannitol and formation of zooglia in 5% sucrose broth. *S. mutans* was present in 15 (61%) of plaque samples; 7 (25%) were heavilyy infected. Lactobacilli occurred in five (18%) of samples and *Candida* in five different samples. *S. mutans* was present on four pencil tops (14%); one had *Candida* and three had lactobacilli. Four contaminated pencils in 28 couldcontact 50% of the class in five distributions, 75% in nine distributions and almost all of the class in 38 distrtibutions. *S. mutans* is widely spread and readily transmitted among schoolchildren. Its presence with other oral flora on pencils suggests that communally used pencils contribute to the transmission of these organisms.

(Appendix E)

Australian Dental Journal, June, 1976 205

JUNE, 1976 AUSTRALIAN Dental Journal Volume 21 :: Number 3

A community dental health project
I. Self applied SnF_2-$ZrSiO_4$ prophylactic paste and dental caries in primary school children

Robin Woods

Noel D. Martin*

AND

P. D. Barnard*

ABSTRACT—A dental health programme which employed group self prophylaxis by school children aged 5-9 years is described. The group used 10 per cent SnF_2-$ZrSiO_4$ paste and was supervised within the school by an auxiliary of parents under the direction of a dentist. There was a 36 per cent reduction in DMFT increment at the end of two years in the children aged 7-9 years.

(Received for publication February, 1975)

Introduction

Self applied concentrated stannous fluoride-zirconium silicate prophylactic pastes have been successfully used in a number of trials to reduce the incidence of dental caries in school children. The procedure is an attractive health measure since it utilizes a minimum of professional man-power and achieves a substantial reduction in dental caries.

In an extensive series of studies Muhler[1] reported significant reductions in the DMFT and DMFS increments following self use of a 9 per cent SnF_2-$ZrSiO_4$ paste. The effectiveness of the programme Muhler devised was improved when the self prophylaxis was made at six-monthly intervals, instead of annually. The percentage caries reduction reported was also greater when a stannous fluoride (0.4 per cent) dentifrice was issued for home use. Significant reductions were reported for children in both non fluoride and natural fluoride areas. In these studies Muhler changed the examiners deliberately to demonstrate that the caries reduction was sufficient to be observed by dentists practising privately. Because of the different examiners it is difficult to make absolute comparisons regarding the degree of caries reduction in the different studies.

* Department of Preventive Dentistry, University of Sydney.

1 Muhler, J. C.—Mass treatment of children with a stannous fluoride-zirconium silicate self administered prophylactic paste for partial control of dental caries. J. Amer. Coll. Dent., 35:1, 45-47 (Jan.) 1968.

Lang and his colleagues[2] reported a marked reduction in dental caries following three applications at six-monthly intervals of a 9 per cent stannous fluoride paste, used in an optimum fluoride areas, he found that home use of a stannous fluoride paste made little difference to the incremental reduction of dental caries.

In 1969 Gish and Mercer[3] reported a reduction in dental caries when school children in both an optimum fluoride community and a non fluoride community used a zirconium silicate-stannous fluoride paste at six-monthly intervals. The children were in groups of 12-20 and were supervised by a hygienist.

Muhler[4] and his co-workers[5] in 1970 reported significant and marked reductions of dental caries increments after one year, when the 9 per cent SnF_2 paste was applied under supervision twice, at six-monthly intervals.

In a trial utilizing self applied 9 per cent SnF_2 paste, Gunz[5] achieved a small reduction in the caries increment one year after a single self application of the paste. She commented on the very large groups of children and suggested that the procedure could be improved with increased and closer supervision.

Reviewing various methods utilized for self application of fluorides, Ripa[6] accepted the effectiveness of several methods including brushing. In particular he reviewed the studies by Lang[2], and Gish[3] and Muhler[4] already mentioned.

It would seem on the bases of these publications that self application of a high concentration stannous fluoride zirconium silicate paste is an effective and acceptable anti-caries measure suitable to be utilized as part of a public health programme directed towards caries control in school children.

The relationship between *Streptococcus mutans* and dental caries has been extensively reported and reviewed by Shklair[7] who has published an extensive bibliography. The relationship between *Str. mutans* and dental caries has been established.

The effect of topical fluoride applications, including a small pilot study, of self applied SnF_2-$ZrSiO_4$ paste, on the concentration of *Str. mutans* in dental plaque has been reported[8] and it has been suggested that one of the mechanisms whereby topical fluorides achieve an anti-caries effect is by reducing the incidence and concentration of *Str. mutans* in dental plaque. From this study it appeared that self applied SnF_2-$ZrSiO_4$ prophylactic paste was similar in effect to other forms of fluoride applications tested and was a promising and convenient method to provide mass topical fluoride treatment.

The trials of self applied SnF_2-$ZrSiO_4$ paste[8,9] as an anti-caries measure, conducted by one of the authors (R.W.) in Yass (1969-1970), were promising and it was decided to establish a programme built around the paste as a means to assist rural communities to overcome the more acute problems of dental caries among their children.

Boorowa, a rural town of approximately 2,000 people on the south west slopes of New South Wales, was selected in 1970. There is no regular dental service in Boorowa and it was thought that if dental caries in the school children was minimized by adoption of what amounted to a public health measure, the treatment needed for repair and rehabilitation of this disease would be reduced to a level the community could provide for themselves.

When the programme was implemented its effectiveness as an anti-caries measure would need to be assessed. In addition the effect reported[8] of the self applied SnF_2 paste on *Str. mutans* in dental plaque could be re-examined, and there would also be an opportunity to assess the relationship between dental caries and *Str. mutans* in plaque to determine whether caries is related to the concentration of *Str. mutans* in plaque before or after the self prophylaxis programme was established.

2 Lang, L. A., Thomas, H. G., Taylor, J. A., and Rothhaar, R. E.—Clinical efficacy of a self applied stannous fluoride prophylactic paste. J. Dent. Child., 37:3, 211-216 (May-June) 1970.

3 Gish, C. W., and Mercer, V. H.—Child self application of a zirconium silicate-stannous fluoride paste — clinical results after one and two years. I.A.D.R. (Abstract) 47th General Meeting, Houston, U.S.A. 1969, Paper 552.

4 Muhler, J. C., Kelley, G. E., Stookey, G. K., Lindo, F. I., and Harris, N. O.—The clinical evaluation of a patient administered SnF_2-$ZrSiO_4$ prophylactic paste in children. I. Results after one year in the Virgin Islands. J.A.D.A., 81:1, 142-145 (July) 1970.

5 Gunz, Gertrude M.—The effect of self applied fluoride paste. J. Public Health Dent., 31:2, 177-181 (Summer) 1971.

6 Ripa, L. W.—Recommendation for the use of topical fluoride self application techniques in a preventive office programme. N.Y. State Dent. J., 39:4, 227-233 (April) 1973.

7 Shklair, I. L.—*Streptococcus mutans* and the epidemiology of dental caries. *In, Streptococcus mutans* and dental caries. Proceedings of a round table discussion, May 10, 1973, 73rd Annual Meeting, American Society of Microbiology (pp. 7-13).

8 Woods, R.—The short term effect of topical fluoride applications on the concentration of *Streptococcus mutans* in dental plaque. Austral. D.J., 16:3, 152-155 (June) 1971.

9 Woods, R.—A dental caries susceptibility test based on the occurrence of *Streptococcus mutans* in plaque material. Austral. D.J., 16:2, 116-121 (April) 1971.

(Appendix E)

An additional aim was to introduce to a community a degree of responsibility for its health care, to help the community help itself. It was hoped that if this pilot scheme was successful it could be implemented in other rural or isolated communities where there is no regular dental service.

Methods and procedures

The programme was conducted within the schools by a private dental practitioner from a town 50 km distant. An auxiliary of school children's parents was established; permission of the education authorities and parents was obtained for all children in kindergarten, first, second, and third classes (age approximately 5-9 years) to be included in the programme.

Children were treated in groups of 15-20 and supervised by seven or eight parents directed by the dentist. The high parent co-operation allows close supervision and instruction and the direction by the dentist prevents the parent taking over from the child; a cardinal principle was that the children must use the toothbrush themselves. As well as being compatible with other skills being learned at this age this procedure allowed the children to spit freely to avoid swallowing the paste, which if swallowed in quantity may have an emetic effect.

The function of the parent auxiliary was to set up the necessary material and supervise the children during the self prophylaxis. In addition the auxiliary acted as a liaison in dental health education with other parents carrying information regarding the need for children to have fluoride supplements in addition to the self prophylaxis, and also to use a fluoride toothpaste regularly at home.

Prior to the commencement of the programme all the children were given a baseline dental examination by two experienced examiners from the Department of Preventive Dentistry, University of Sydney. X-Ray examination was not used. Expected increments of dental caries were calculated from the examination results. In addition plaque samples were taken from all children and were assessed for *Str. mutans* by the screening test described previously[9] and whether or not fluoride supplements had been used, and when, was noted.

The school children applied the self prophylaxis paste under supervision twice late in 1970 and then each term, that is three times each year, throughout the survey period. The following directions were given:

First, wet the brush and squeeze not more than ¼ in on to the brush.

(1) Close teeth and using a circular motion with the brush clean the outside surfaces of all the teeth (left, right, upper and lower), *brush for one minute*, spit out surplus paste.

(2) With teeth apart, clean the inside surfaces of all upper teeth (next to palate) using a circular motion. *Brush for one half-minute*, spit.

(3) With teeth apart, clean the inside surfaces of all lower teeth, *brush for one half-minute*, spit.

(4) Clean biting surfaces of all teeth, upper and lower, *brush for one minute*, spit, rinse and wash brush.

N.B. Allow children to spit out surplus paste as often as they wish.

Avoid getting paste or saliva on clothes.

Apply more paste if required between exercises, usually after second exercise.

Do not rinse mouth or wash brush between exercises.

The exercises were developed in consultation with the children's teachers and were designed to allow exposure of all tooth surfaces to the prophylaxis paste and also to permit the development of a toothbrushing regime which could be demonstrated to the children on the basis of holding the toothbrush in various positions. This is compatible with other skills the children in these age groups were learning, for example the use of such items as writing instruments, knife and fork. Supervising and demonstrating the various exercises by teaching brush positions proved to be a quick and effective method of introducing the children to the self prophylaxis. Each group of children could be treated in approximately 15 minutes, they were placed in protective gowns (made from reversed shirts) and were given their toothbrushes which were kept in the classroom by their teacher.

Each child was provided with two disposable cups, one in which to spit and another with clean water for rinsing. The prophylaxis took place in one school in a wash shed, and another in a weather shed. Although water on tap is a help the main requisite is a cement floor to allow for the inevitable spillage of water and suitable benches to accommodate the cups.

The paste used included 10 per cent stannous fluoride with a zirconium silicate base*. Tests on the paste conducted by the Australian Standards Laboratory showed that the fluoride remained available after the paste had been stored for over two years.

The programme commenced with the baseline clinical examination in June 1970, a plaque examination in July 1970, and the first self prophylaxis in August with a second self prophylaxis in October. Subsequent self prophylaxis occurred each term. The acceptance by the children was very good and the programme was absorbed as part of the school regime.

* Supplied by courtesy of R. M. Creighton.

(Appendix E)

TABLE 1

Mean DMFT, DMFS, and dmft for Baseline and Study Groups Self applied 10 per cent SnF_2-ZrO_4 paste, 3 x years for 2 years (Primary school children, Boorowa, 1970-1972)

Group	Age 5-7 years N	DMFT	DMFS	dmft	Age 7-9 years N	DMFT	DMFS	dmft
Baseline*		1970				1970		
Non-fluoride	94	1.02	1.53	5.98	81	2.79	4.51	6.74
Fluoride supplement	32	0.72	0.91	2.63	15	2.20	3.13	4.93
Total Group	126	0.94	1.37	5.13	96	2.70	4.29	6.46
Study**		1970				1972		
Non-fluoride	33	0.97	1.36	6.21	33	2.36	3.21	6.39
Fluoride supplement	14	0.00	0.00	1.14	14	0.50	0.50	0.71
Total Group	47	0.68	0.95	4.70	47	1.81	2.40	4.70

* The baseline increment was calculated by comparing children aged 5-7 years with children aged 7-9 years at the time of the 1970 examination.

** The study increment was calculated from the 1970 survey and by a second examination of the same children in 1972.

In April 1971, 6-7 weeks after the third self prophylaxis, the children were re-examined for *Str. mutans* in plaque. A preliminary report of the reduced incidence of *Str. mutans* has been presented[10], a full report has been submitted for publication[11].

Self applications of the paste were made each term and after seven such applications the children were re-examined by the same examiners for dental caries. The second clinical examination took place in July 1972.

Results

The control (expected) increment of dental caries was calculated on the basis of the preliminary examination. The children who had received fluoride supplements commencing either prenatally or up to the age of two years were considered as a fluoride group; the balance having commenced fluoride supplements too late to significantly influence those teeth erupted at the time of examination, were considered as a non-fluoride group. The two-year caries increment was calculated for fluoride supplement, non-fluoride and whole groups.

The Study Group consisted of a sample of 47 children who had attended both clinical examinations and at least six of the seven self prophylaxis sessions. Children who attended dentists privately and received other topical fluoride treatment were excluded from the study. The effect of applying these criteria to the Study Group reduced the numbers but precludes objections which might be raised on the grounds of the anti caries effect of topical fluoride treatment obtained privately. Exclusions for home use of fluoride toothpaste were not made, they were used by almost all the Study Group and by approximately half of the students forming the baseline study.

The control increment was calculated on the basis of a total 126 (including 32 who had

TABLE 2

Reduction in two year dental caries increment Self applied 10 per cent Snf_2-$ZrSiO_4$ paste, 3 x year (Primary school children Boorowa, 1970-1972)

Group	Increment Baseline	Study	Reduction Actual	'P' Value
Non-fluoride				
N	94-81	33		
DMFT	1.77*	1.39*	0.38 (21)	<0.05
DMFS	2.98*	1.85*	1.13 (38)	<0.05
dmft	0.76	0.18	0.58 (76)	>0.05
Fluoride supplement				
N	32-15	14		
DMFT	1.48*	0.50*	0.98 (66)	>0.40
DMFS	2.22*	0.50*	1.72 (78)	>0.40
dmft	2.30	–0.43	2.73 (118)	<0.05
Total				
N	126-96	47		
DMFT	1.76	1.13	0.63 (36)	<0.001
DMFS	2.92	1.45	1.47 (50)	<0.001
dmft	1.33	0.00	1.33 (100)	<0.05

Percentages in parentheses.

* Caries increments for the fluoride supplement group significantly lower than for the non-fluoride group.

10 Woods, R. G.—Self applied fluorides and *Streptococcus mutans* in dental plaque. 14th Annual Meeting of I.A.D.R., A.N.Z. Div., Adelaide, S.A., August 1974, Paper 29 (Abstract).

11 Woods, R.—A community dental health project. II. Modification of *Streptococcus mutans* in dental plaque by self applied prophylactic paste, and its relationship to dental caries. Austral. D.J., in press.

received fluoride supplements) children aged 5-7 years compared with 96 (including 15 children who had received fluoride supplements) children age 7-9 years.

The results are presented in Tables 1 and 2. Table 1 sets out the mean DMFT, DMFS and dmft values obtained for the age groups 5-7 and 7-9 years for all children at baseline examination in 1970 and for the Study Groups in 1972. It will be noted that in each category the initial caries experience of the Baseline Group was greater than that of the Study Group except for the dmft in the 5-7 year age group (non-fluoride) where the Baseline Group had 5.98 dmft and the Study Group had 6.21 dmft (Table 1). However the increment dmft for the two year period was 0.76 for the former and 0.18 for the latter; a reduction of 0.58 or 76 per cent, P>0.05 (Table 2). The most significant reduction for permanent teeth and surfaces occurred in the total figures. The increment DMFT for the Study Group of 1.13 indicates a reduction of 0.63 or 36 per cent (P<0.001) and a DMFS of 1.45 indicates a reduction of 1.47 or 50 per cent (P<0.001). Table 2 presents the two year caries increments for both permanent and deciduous dentitions, differences and "P" values derived from statistical assessment.

Discussion

Evaluated on the basis of the number of subjects involved in the study alone the results are modest. A Study Group of 47 subjects is small but the Study Group was reduced by the elimination of as many variables as possible.

The control increments, calculated from the baseline survey are consistent with other Australian surveys. Barnard[12] in 1956 reported a two year DMFT increment of 2.23 for 6-8 year old children and 2.06 for 8-10 year old children, although he found a higher DMFS two year increment of approximately 4.5-4.8. Metcalf[13] reported a two year DMFT increment of 1.93 for 6-8 years and 1.49 for 7-9 year children.

The statistically significant reduction in two year dental caries increments in the permanent dentition (Table 2) for all the study group without fluoride supplement was slightly lower than that reported overseas by Muhler and his colleagues[1,4] where there was no history of fluoride supplements.

It is not strictly possible to compare children in fluoride areas with those who receive fluoride supplements, the voluntary effort involved in the latter in itself indicates an interest in dental health which may not be reflected in all children who receive their fluoride from reticulated water systems.

The values of reduced dental caries increment in fluoride supplement group (DMFT 66 per cent DMFS 78 per cent) appeared higher than that reported by Lang[2] or Muhler[1] for children in in fluoride areas, but because of small numbers these reductions are not statistically significant but are consistent with previous reports of additional benefit brought about by the use of the self prophylaxis paste in optimum fluoride areas[1,2]. Both baseline and study two year dental caries increments were significantly less for the fluoride supplement group when compared to the non-fluoride group.

The reduced caries increment in the deciduous dentition is more difficult to evaluate, partly because of the natural reduction which occurs in dmft following natural tooth loss, particularly when decayed deciduous anteriors are lost.

The reduction in deciduous caries for all cases together was significant, the non-fluoride supplement group considered alone did not show a significant reduction although in this case the fluoride supplement group did when compared to the high baseline dmft increment value.

There were a large number of reversals in the increments of deciduous caries, partly due to the natural reduction in dmft where decayed deciduous teeth are lost but some may have been related to the effect of the trial. Reversals associated with this procedure have already been reported by Muhler[1].

The use of self prophylaxis paste for young school children as an anti-caries measure has some advantages in that it aids their development of dexterity and encourages the use of a toothbrush, an important initial step in plaque control. Further, it has been our experience that a 10 gm tube of the paste is sufficient for 10-12 applications. On the bases of three applications per year this makes a total fluoride availability of less than 80 mg fluoride per year. Even assuming total absorption this amount is not large enough to be of any concern.

The self prophylaxis occurred three times each year.

---

12 Barnard, P. D.—Dental survey of State school children in New South Wales. January 1954-June 1955. Special Report Series, N.H.M.R.C. Australia, No. 8, Canberra 1956.

13 Metcalf, G. W.—Report on caries experience and treatment needs in Western Australian children aged 6-14 years. Prefluoridation dental assessment survey, November 1967. Austral. D.J., 15:1, 50-54 (Feb.) 1970.

(Appendix E)

Satisfactory results have been reported with six-monthly applications[1, 2, 3, 4] although a single application reported by Gunz[5] produced a much smaller decrease in caries. The frequency of this trial, each term, seems very satisfactory and lends itself to easy incorporation within the school programme.

The value of this procedure is that it can achieve an anti caries effect in addition to that achieved by use of fluoridation or fluoride supplements and to take advantage of its potential it should be utilized as part of a multiple fluoride programme. There appears to be a basic advantage in training in plaque removal, less school interruption and need for dental staff supervision for this procedure than for a mouth rinsing programme.

Supervision in the trial was close, two or three children were supervised by each parent under the direction of the dentist. Gunz[5] noted the difficulties of supervising large groups of children. In addition the close supervision in this trial contributed to the smooth handling of the procedure and also established a very effective channel of communication through the supervising parent auxiliary with the community which has proved valuable in conveying dental health information and information regarding the nature of the programme. The help of the parent auxiliary has been valuable in involving the community in its own health programme.

Summary

A programme of self application of a 10 per cent SnF_2-$ZrSiO_4$ preventive paste (3 x per year) by primary school children in schools in a rural community has been demonstrated to be practical and effective in reducing the incidence of dental caries. The programme has been designed specifically for isolated and small rural communities and is directed to involve these communities in their own dental health programme through stimulation and supervision of a dentist visiting the community at regular intervals. The principles of the programme could be applied to other communities, rural or metropolitan. The widespread adoption of such a programme could be an efficient means to reduce the professional manpower needed to control or treat dental disease. The effect of the programme on dental health can be achieved without making unrealistic demands on dental manpower.

P.O. Box 22,
Yass, N.S.W. 2582.

(Appendix E)

Australian Dental Journal, August, 1976

319

Volume 21,
No. 4

A community dental health project

II. Modification of *Streptococcus Mutans* in dental plaque by self applied Snf$_2$-ZrSiO$_4$ prophylactic paste, and its relationship to dental caries

Robin Woods

ABSTRACT—The incidence of *Streptococcus mutans* in the dental plaque of primary school children was reduced significantly following the group self application of SnF_2-$ZrSiO_4$ paste. This confirmed earlier reports of the effect of topical fluoride therapy on *Str. mutans* in plaque.

(*Received for publication February, 1975*)

Introduction

The reduction in dental caries by the group use of self applied concentrated stannous fluoride-zirconium silicate paste by school children and the aims, organization and procedure of this community dental health project has been reported[1]. The effect of plaque removal and topical fluoride application on *Str. mutans* in plaque including a small trial of self applied stannous fluoride prophylactic paste was reported in 1971[2].

The purpose of this article is to report the effect of self applied 10 per cent stannous fluoride zirconium silicate paste* as applied in the previous report[1] on the incidence of *Str. mutans* in the dental plaque of school children and to show the relationship between the modified incidence of *Str. mutans* in plaque and the subsequent dental caries increment.

Methods

The two year dental caries increment for a group of school children aged 7-9 years was determined by clinical examination in mid 1970 and 1972[1]. After the baseline examination the children's dental plaque was examined for *Str. mutans* by the screening test previously described[3]. From the results of these tests the plaque was classified, Grade 1, no *Str. mutans* found; Grade 2, an occasional colony encountered; Grade 3, the plaque was infected. The precise criteria for these classifications have been set out elsewhere[3].

The programme of self prophylaxis commenced soon after the baseline examination and group

* Supplied by courtesy of R. M. Creighton.

1 Woods, R., Martin, N. D., and Barnard, P. D.—A community dental health project. 1. Self applied SnF_2-$ZrSiO_4$ prophylactic paste and dental caries in school children. Austral. D. J., 21:2, 205-210 (Apl.) 1976.

2 Woods, R.—The short term effect of topical fluoride applications on the concentration of *Streptococcus mutans* in dental plaque. Austral. D. J., 16:3, 152-155 (June) 1971.

3 Woods, R.—A dental caries susceptibility test based on the occurrence of *Streptococcus mutans* in plaque material. Austral. D. J., 16:2, 116-121 (Apl.) 1971.

(Appendix E)

TABLE 1

Two year caries increment and plaque status study group 1970-72 children aged 7-9 years

Patient	Two year increment DMFT	Two year increment dmft	Status *Str. mutans* (plaque) Initial	Status *Str. mutans* (plaque) Modified	Patient	Two year increment DMFT	Two year increment dmft	Status *Str. mutans* (plaque) Initial	Status *Str. mutans* (plaque) Modified
155	4	3	3	1	158	1	0	1	1
029	4	3	1	2	073f	1	0	2	1
015	4	−4	3	–	016	0	−9	2	–
039	4	−1	3	–	156	0	1	–	3
043	4	−4	2	2	195	0	0	1	1
018	3	1	3	1	084	0	1	3	1
019	3	8	2	2	087	0	0	1	1
027	3	0	1	2	069f	0	0	3	1
164	3	3	–	2	188f	0	0	1	3
033	2	0	1	1	086f	0	0	1	1
014	2	−4	3	1	038f	0	0	1	2
106	2	−1	3	2	191	0	−1	–	1
003f	2	2	2	1	198f	0	−1	3	2
154	2	1	3	2	184f	0	0	3	1
002f	2	0	1	1	185f	0	−4	1	1
201f	2	−1	3	2	186f	0	−5	1	1
006	2	−2	2	3	187f	0	0	1	1
162	2	−2	–	1	190	0	−6	1	2
149	1	3	–	1	082f	0	−3	2	1
004	1	4	3	–	013	0	−5	2	2
147	1	7	–	2	045	0	1	1	–
148	1	7	3	2	072	−2	−4	1	1
189	1	5	1	–	197	−3	0	2	1
001	1	1	1	2					

Fluoride supplement group: f
Plaque status 3. Plaque infected with *Str. mutans*.
Plaque status 2. Occasional *Str. mutans*.
Plaque status 1. No *Str. mutans*.

self prophylaxes were conducted twice in 1970 and again in first term of 1971 before a second plaque examination was made. This examination was delayed for 6-7 weeks after the third self prophylaxis in order to test the prolonged effect on plaque rather than the short term effect previously described[2].

Self prophylaxis was continued each term for the next four terms, until in mid 1972 those involved in the programme had received a possible seven self prophylaxes. A second clinical examination was conducted in mid 1972, two years after the baseline examination.

In addition to the programme of clinical and plaque examinations for the 47 subjects of the Study Group referred to previously[1] the results of a further two plaque examinations on only 35 subjects are included in this report.

Results

The caries increments for the reduced Study Group of 35 school children are: No fluoride supplements, 29 cases, mean two year DMFT increment 1.03; fluoride supplement, 14 cases, mean two year DMFT increment 0.50 (unchanged); all cases, two year DMFT increment 1.00. Compared with the baseline increments established[1] the non-fluoride supplement group had a 41 per cent reduction ($P<0.01$), all cases 43 per cent reduction ($P<0.001$).

The results of the examinations of the Study Group of children are set out in Table 1.

The second plaque examination for *Str. mutans*, following three self prophylaxes showed a reduced incidence of *Str. mutans* plaque infection (Grade 3). Comparisons are set out in Table 2, (a) and (b).

Whether all of the Study Group cases are considered Table 2 (a), or only the reduced group of 35, a χ^2 test showed the reduction in the incidence of plaque infection is significant, $<P$ 0.01.

(Appendix E)

TABLE 2(a)

Comparison of Str. mutans plaque infection before and after self prophylaxis — Study Group

Plaque status	Number of Cases Initially	After three self prophylaxes
Grade 3	14(34)	3(7)
Grade 2	9(22)	15(37)
Grade 1	18(44)	23(56)
Total:	41(100)	41(100)

Percentages in parentheses

Examination by rank correlation of the *Str. mutans* plaque status and the DMFT increment showed some correlation between the initial plaque status of *Str. mutans* and caries increment in permanent teeth (r = + 0.454).

The correlation between the results of the second plaque examination and the DMFT increment is however, much closer and more significant (r = + 0.734).

The relationship between *Str. mutans* plaque status and dmft increment showed no demonstrable correlation. This could be due in part to the difficulty of interpreting the quantum of caries to develop a hierarchy for rank examination where so many caries reversals occur. This is an inherent problem associated with caries examination of deciduous teeth.

Discussion

The significance of this report is in recognition that self prophylaxis by school children using a 10 per cent SnF_2-$ZrSiO_4$ paste reduces the incidence of *Str. mutans* in dental plaque, and further, that the pattern of the reduced incidence of dental caries subsequent to this observation has a close and significant relationship when considered for individual patients, with the modified pattern of *Str. mutans* in dental plaque.

A parallel effect between *Str. mutans* in plaque and the caries increment has been demonstrated. Both factors were determined by separate methods and personnel. This provides the results of both the investigation of caries and plaque with greater credibility than if either result were considered alone.

In addition, this report has added to the volume of information showing a clear relationship between *Str. mutans* in plaque and caries increment. It is suggested that the reduction in dental caries increment experienced subsequent to the 10 per cent SnF_2-$ZrSiO_4$ self prophylaxis may be mediated in part at least, through reducing the incidence of *Str. mutans* in plaque.

TABLE 2(b)

Comparison of Str. mutans plaque infection before and after self prophylaxis — Study Group, children attending for both plaque examinations, only

Plaque status	Number of Cases Initially	After three self prophylaxes
Grade 3	11(31)	2(6)
Grade 2	8(23)	13(37)
Grade 1	16(46)	20(57)
Total:	35(100)	35(100)

Percentages in parentheses

The reduced incidence of *Str. mutans* in plaque was demonstrated some 6-7 weeks after the third self prophylaxis, sufficient time for plaque re-establishment, in this respect it reflects a truer plaque change than reported earlier[2] when the change was observed over a period of one week only.

Summary

Changes in the incidence of *Str. mutans* in dental plaque following self prophylaxis by school children age 5-7 years using 10 per cent SnF_2ZrSiO_4 paste have been demonstrated confirming an earlier report. A close and significant correlation has been demonstrated between the modified incidence of *Str. mutans* in dental plaque and the subsequent dental caries increment. It is suggested that the anti-caries effect on this self prophylaxis paste may be due in part to its effect on *Str. mutans* in plaque.

P.O. Box 22,
Yass, N.S.W. 2582.