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ORAL HEALTH AND LIFE SKILLS IN SOCIETY

GUEST EDITORIAL

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The number of adults in NSW with their natural teeth is increasing; but, at the same time, so is the number of people with periodontal disease. Recent findings that pregnancy, diabetes and cardio-vascular disease may be associated with periodontal diseases represent the beginning of a new era in oral health awareness. As the number of elderly people in the community increases, the oral health of the general population becomes more important. Medication usage is expected to rise with this increase in the elderly population, and many medications have a direct or indirect effect on oral health. These topics were introduced in the March issue of the NSW Public Health Bulletin.

This issue—the second of four highlighting both the relationship between oral health and general health and the potential public health consequences of poor oral health—examines the contribution of oral diseases to general health at various stages in life.

While historically oral health has not been prominent within the health system, changes are afoot.[1] The NSW Department of Health has noted promising developments for the collection of oral health data.[1] This year the NSW Health Survey will focus on the health of children and older adults in NSW, and questions on oral health will be included. Such data will assist in the development of robust oral health policies.

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For too long, dentistry has sought to influence health planning by stressing that oral diseases, particularly caries and periodontal diseases, can have a major effect in a few systemic illnesses. It is time to review this approach and stress to those involved in planning for the health of the population that *all* systemic illnesses can have oral health manifestations, and that oral health itself may be directly involved in the causality of prevalent chronic conditions such as cardiovascular disease. Further, a healthy mouth contributes to the day-to-day lifestyle of all residents of NSW; therefore, we need to ensure that oral health becomes a recognised and familiar part of general health and well-being.

This issue of the *Bulletin* seeks to emphasise this by examining oral health at varying stages of our lives and its relationship with general health. For our infant population, the issue of child neglect will be illuminated by its relationship with rapid early-onset dental caries. Inappropriate nutrition and eating disorders are particular problems encountered by adolescents and young adults. The oral health implications of these disorders are potentially significant and contribute to morbidity. The adolescent who is already burdened by low self-esteem can find the damage caused to their teeth by their poor oral health a further handicap in coping with daily life. The importance of primary prevention, particularly that of reducing alcohol intake and smoking cessation—a risk factors for so many illnesses—is stressed.

The next issue in this series will continue to focus on the relationship between oral and general health by considering the oral health needs of the elderly, oral cancer, and the economic and workforce issues that affect poor oral health.

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Submission of articles

Articles, news and comments should be 1000 words or less in length and include a summary of the key points to be made in the first paragraph. References should be set out in the Vancouver style, described in the *New England Journal of Medicine*, 1997; 336: 309–315. Send submitted articles on paper and in electronic form, either on disc (Word for Windows is preferred), or by email. The article must be accompanied by a letter signed by all authors. Full instructions for authors are available on request from the editor.

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Mary Osborn also contributed to the series during her Public Health Officer placement in the Oral Health Branch. In particular, we thank Jane Bell who has played a significant role in coordinating the series.

BOTTLE CARIES AND DENTAL NEGLECT

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Each year large numbers of Australian children undergo dental treatment for bottle caries. This significant form of dental decay affects the first teeth of infants and young children who have, most often, been exposed to bottle feeding for long periods during each day and night.[1,2] A recent study found that 40 per cent of decay in five-year-old Australian children remains untreated.[3] Dental neglect is defined as the failure of a parent or guardian to seek, or follow through with, treatment for caries, oral infections or oral pain. As such, many children with bottle caries can be considered to be suffering from dental neglect. This article describes the prevalence of bottle caries, explores dental neglect, and concludes with simple preventive measures that can be used to protect the teeth of young children.

BOTTLE CARIES

Bottle caries is known by several different terms, including nursing caries, nursing bottle caries, baby bottle caries, and baby bottle tooth decay. Bottle caries is caused by the frequent and prolonged exposure of the teeth to a decay-causing substance, particularly at night when the cleansing effect of salivary flow is low. Such substances include human and cow milk, baby formula, sweetened milk, fruit juices, cordials and other soft drinks. Although bottle feeding is the most common cause, bottle caries can also result from the frequent use of a pacifier dipped in a sweetened substance, such as honey, and from prolonged on-demand breast feeding habits.

The pattern of decay that results from such practices is characteristic and affects, most severely, the upper primary incisor teeth and the primary first molar teeth, followed by the primary second molars and canines. This pattern of decay can be attributed to the eruption sequence of the primary dentition, whereby the first teeth to erupt have the longest exposure to the decay-causing habit. The primary lower incisor teeth are conspicuously spared from the decay process because of the position of the tongue during suckling and salivary secretion from the adjacent submandibular salivary gland ducts.

PREVALENCE

The reported prevalence of bottle caries in western countries of less than five per cent is probably underestimated because of the inaccessibility of the pre-school population for dental examination.[1,2] In addition, data collected may be from biased samples, as children whose parents are aware of an existing dental problem are more likely to present for examination.[1,4] The available Australian data reports prevalence figures ranging from 2.6 to 6.4 percent.[5–7] Studies of such groups of affected children have reported an association with low socioeconomic background, educational status, and age of the mother.[8–10]

Bottle caries is also thought to be a major cause of untreated dental decay in first teeth. A recent study found that 40 per cent of decay in five-year-old Australian children remains untreated.[3] Although not all of this untreated decay can be attributed to bottle caries, it would be expected that a significant proportion of untreated caries in five-year-old children is associated with prolonged bottle or breast feeding habits.[11]

A recent study of patients referred for specialist paediatric dental services at Westmead Hospital Dental Clinical School has found that the most common outcome, accounting for 37 per cent of referred child patients, was treatment of dental decay under general anaesthetic.[12] At the same clinical school, more than 800 children per year receive a general anaesthetic for dental treatment, of which at least 80 per cent is for the management of decay.[13] Of these children, 44 per cent are under three years old, strongly implicating bottle caries as a major cause.[13]

DENTAL NEGLECT

While in the process of developing policy documents of its own, the Australasian Academy of Paediatric Dentistry has adopted policies endorsed by the American Academy of Pediatric Dentistry. These American guidelines define dental neglect as the failure of a parent or guardian to seek treatment for caries, oral infection, or oral pain; or failure of the parent or guardian to follow through with treatment once informed that the aforementioned conditions exist.[14] Figure 1 lists indicators of dental neglect in children.[15] Both the first and third indicators can be associated with a significant number of children with bottle caries.

However, the situation is not as simple and straightforward as it seems. Generally, neglect can be defined as the failure of a parent or guardian to provide an adequate level of care unless the parent or guardian is unable to provide these needs for the child because of poverty.[16] Given that the parents of children with bottle caries are more often of low socioeconomic and educational status, it becomes more difficult to define children as suffering from dental neglect, as opposed to those whose parents or guardians are unable to access dental treatment due to socioeconomic and educational barriers.

Further, in spite of the fact that bottle caries most commonly affect the upper primary incisor teeth and are relatively easy to identify by lay people, parents or guardians often present late with children for consultation and treatment.

FIGURE 1

INDICATORS OF DENTAL NEGLECT IN CHILDREN[15]

- Untreated, rampant caries that is easily detected by a lay person
- Untreated pain, infection, bleeding or trauma affecting the orofacial region
- History of lack of continuity of care in the presence of previously identified dental pathology

Unfortunately, once diagnosed, a small but significant group of children are not brought consistently for follow-up dental care. This group includes those children whose parents or guardians bring them for consultation and are informed of the child's dental condition and treatment needs, but do not follow through with subsequent treatment. It is children who fall into these categories, of presenting late for diagnosis or poor dental attendance, who can be identified as at risk of or suffering from dental neglect.

PREVENTION

Prevention of bottle caries requires appropriate dental education of new and prospective parents, including both dietary and oral hygiene advice. The use of the bottle as a pacifier should be discouraged and infants should not be allowed to fall asleep with a bottle containing decay-causing substances.

Once teeth appear, at around six months of age, they should be cleaned. Initially, this can be achieved by wiping the child's teeth and gums with a damp cloth, then aiming to introduce a small, soft toothbrush by 12 months of age. A smear of junior-strength toothpaste on the brush provides a regular fluoride treatment and should be used routinely from two years of age. Regular dental visits should begin around the child's first birthday to ensure early diagnosis and prevention.

The education and involvement of all maternal and child health professionals in preventing caries, including bottle caries, and in encouraging regular dental visits, is essential. Children's oral health must be regarded as an important component of their general health.

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ADOLESCENTS, NUTRITION AND EATING DISORDERS

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Adolescent girls in Australia have some of the worst eating habits of any group in the community. Some consume too many foods of poor nutritional value, while others try to eat as little as possible. The incidence of anorexia nervosa and bulimia nervosa is highest during the adolescent years for girls. Although adolescent boys suffer much less from eating disorders and most have a higher intake of nutrients because of their legendary appetites, some make poor food choices. Obesity is a growing problem for both boys and girls, with the National Nutrition Survey classifying 25 per cent of boys and 19 per cent of girls as overweight or considered at risk of being overweight.[1]

This article examines some of the eating disorders presenting in adolescents and their impact on oral health. It also describes the patterns of food consumption by adolescents and how this can be influenced to minimise the consumption of unnecessary sugars and saturated fats.

EATING DISORDERS

Surveys show that many adolescent girls are underweight and are dissatisfied with their body weight.[2] Estimates of eating disorders vary, and they depend on the diagnostic criteria used, but studies in South Australia indicate that bulimia nervosa and anorexia nervosa may occur in about 0.5–1.0 per cent of women, while up to five per cent of women have some degree of eating disorder.[3,7] Young women who diet are at most risk. Diagnosis of bulimia and anorexia nervosa is sometimes made by dentists who observe dental erosion due to the acidity of repeated vomiting attacks.

Treatment of eating disorders is difficult. However, the earlier treatment begins, the greater the chances of success. A team approach is necessary, and this should involve a general practitioner, dietitian and psychiatrist or a health professional with expertise in this area.

Anorexia nervosa does not always involve vomiting, although this is a common feature. Some girls simply do not eat, and boys suffering this problem often engage in excessive exercise coupled with low food intake. Bulimia, whether it occurs in conjunction with anorexia nervosa or on its own, is more likely to be noticed by dentists. The diagnostic criteria for bulimia nervosa are:

- recurrent episodes of binge eating
- feeling of lack of control over eating behaviour during binges
- regular inappropriate behaviour to prevent weight gain such as self-induced vomiting, misuse of laxatives or diuretics, strict dieting or fasting, or excessive exercise
- minimum average of two binge and compensatory episodes a week for at least three months
- persistent over-concern with body shape and weight.

GENERAL NUTRITION IN ADOLESCENTS

Adolescents who are growing and playing sport need more food than their parents do. They also have extra requirements for many nutrients, especially calcium and iron. The average adolescent boy meets his needs for these and most other nutrients, although many do not consume enough dietary fibre. The major problem for boys is their consumption of large quantities of saturated fat. Their starch intake is relatively low.

Many girls fail to consume enough iron, zinc, calcium, starch and dietary fibre. Their mean potassium intake is also towards the lower end of the desirable range. Fat intake for both boys and girls is likely to be higher than that reported, since almost everyone in the community now under-reports intake of fat and foods rich in fat. More high-starch foods, such as bread and cereals, in place of fatty foods would be nutritionally desirable.

Both sexes consume a lot of sugars from sucrose added to foods as well as intrinsic sugars present in fruits, and in milk products. In Table 1, the intrinsic sugars in the reported consumption of different types of fruit, vegetables, and natural milk products have been calculated from information in the National Nutrition Survey and compared with consumption of total sugars.[4] Note that sugars in fruit drinks and juices have not been included in intrinsic sugars.

The Australian Dietary Guidelines suggest the consumption of 'only a moderate amount of sugars and foods containing added sugars'.[5] It is difficult to define 'moderate', but the Committee of Medical Aspects of Food Policy (COMA) recommends that no more than 11 per cent of energy should come from non-milk extrinsic sugars,[6] considerably less than Australian adolescents consume.

There is a strong argument that the body does not distinguish between different types of sugars, and any fermentable carbohydrate can contribute to dental decay. However, when discussing nutrition, we need to look beyond such concepts to examine the nutritional company that sugar keeps.

Most breakfast cereals, flavoured milk and yoghurt, and fruits canned in syrup or concentrated juice contain nutrients with their sugar. Sweetened breakfast cereals contributed 4.2 per cent of the sugar in the boys' diets and 2.7 per cent in those of girls.[2] Flavoured milk and yoghurt and canned fruits contributed less than four grams of sugar

TABLE 1

CONSUMPTION OF INTRINSIC AND EXTRINSIC SUGARS

Sex	Boys		Girls	
Age (years)	12–15	16–18	12–15	16–18
Average total consumption of sugars (g)	181	212	128	119
Calculated consumption of intrinsic sugars (g)	39	38	35	27
Average consumption of extrinsic sugars (g)	142	174	93	92
% kilojoules from added sucrose	20	22	17	17

for both boys and girls. Much of adolescents' sugar intake comes from foods with low nutritional value, such as soft drinks, cordials, ice cream, iceblocks, chocolate, spreads, cakes, confectionery, sweet biscuits, pastries and sweet buns. Many of these foods also make substantial contributions to saturated fat intake.

This information is presented not specifically to damn sugar, but to point out that sugar-containing foods form a major part of adolescents' diets. Crisps and similar fatty salty snacks are also popular, and teenage boys and 12- to 15-year-old girls consume more of these foods than any other age group. Such foods also contain a lot of saturated fat.

The combined impact of foods of poor or negative nutritional value leads to a displacement of such nutritionally important foods as fresh fruits. The National Nutrition Survey found that 50 per cent of boys and 42 per cent of girls aged 12 to 15 years and 60 per cent of boys and girls aged 16 to 18 years had consumed no fruit in the 24 hours before the survey. Surveys by the Australian Horticultural Corporation have shown similar figures.

Healthy eating does not mean that any specific food needs to be eliminated. However, better nutrition means that some foods should be restricted to particular occasions. This philosophy complements dental education practices that suggest restricting foods with high caries potential to times when it is appropriate and possible to clean the teeth soon after eating.

Adolescents, like younger children, can accept the idea of menus. What is on the everyday lunch menu or the afternoon snack menu can be quite different from a party or holiday menu, or even what kids eat when out with friends. The everyday menu should feature such foods as fruits, vegetables, legumes, nuts, breads and cereals (with at least some being wholegrain), dairy products (low fat varieties are excellent), plus high-protein foods such as fish, lean meat or poultry. When fats and sugars are added to these basics, they should not dominate the daily diet.

CONCLUSION

Some health professionals who work in the field of eating disorders believe we should not try to change adolescents' eating behaviour in case they develop an avoidance of food, which can lead to disordered eating. However, with the increasing problem of obesity in adolescents, better eating habits are urgently needed. Therefore, we should not shrink from nutrition education, but strive to avoid extremes. Oral health professionals, including dentists, can play an important role in nutrition education and need to understand some of the general health problems associated with nutrition and eating habits among Australian adolescents.

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DEPARTMENTAL INITITATIVES TO ADDRESS EATING DISORDERS

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NSW Health is taking up the challenge to improve pathways to care for children and adolescents who exhibit the earliest symptoms of eating disorders. The aim will be to improve detection and strengthen local networks of professionals who have skills to implement effective early intervention, including those who can offer appropriate specialist child and adolescent mental health support. A sum of $100,000 has been allocated to the Department of Psychological Medicine at the New Children's Hospital at Westmead to develop an effective early intervention model for identifying and treating children and young people with eating disorders.

Expansion of a successful shared-care pilot project involving general practitioners and specialist mental health services in Central Sydney Area has also been funded. This program includes a training component for general practitioners that is expected to equip them with skills to detect eating disorders earlier and to intervene more effectively.

In partnership with the former Ministerial Advisory Committee on Body Image and Disordered Eating, the Centre for Mental Health, NSW Health Department, convened workshops in mid-1998 involving several groups which provide support for people with eating disorders and their families.

NSW Health is collaborating with the NSW Department of Education and Training in the development of the School-Link Program. The current focus of that program is on the prevention and early detection of depression in adolescents; however, the inclusion of components to teach positive coping and life skills has broader implications for mental health promotion.

The Centre for Mental Health and the Health Services Policy Branch are also assisting the Statewide Services Development Branch of the NSW Health Department in a project to develop a plan to provide services for people in NSW who have eating disorders. Consultants are currently working on a comprehensive service model that incorporates the full spectrum of care, from prevention and early intervention through to treatment for people with the most severe and complex problems. The process includes wide consultation with professionals, consumers and carers.

Editor's note:

The NSW Ministerial Advisory Committee on Body Image and Disordered Eating was formed in April 1997 on a recommendation of the Summit on Body Image and Eating Disorders held in Sydney in August 1996. The Summit was held in response to growing concern about health problems associated with disordered eating among young women, adolescents and children. It focused particularly on the effect of the media, fashion trends and other social pressures on women's body image and the relationship between those factors and such eating disorders as anorexia nervosa and bulimia nervosa. Following the release of the report from the Summit, the Minister announced the formation of an intersectoral Ministerial Advisory Committee to further examine the issues identified at the forum and to report back with recommendations for addressing those issues. The primary focus of the committee was on preventing disturbed body image and disordered eating.

DENTAL EROSION: MORE ACID MEANS FEWER TEETH

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The surfaces of the teeth are in a continuing state of flux throughout a lifetime. On contact with the tooth, a piece of grapefruit or a drink of orange juice will cause a demineralisation of the enamel or dentine surface. If no further acid attack occurs, the tooth can be remineralised over time by calcium and phosphate ions from the saliva. The balance of this dynamic ionic exchange can easily be upset resulting, for many patients, in irreversible and extensive tooth tissue loss (dental erosion) that may be difficult, if not impossible, to correct. As our knowledge of this form of tooth structure loss increases, there is a growing awareness of the complexity of the erosion process. This article examines the prevalence of dental erosion, the usual sources of acid responsible for it, other physiological factors that determine the impact of acid on the teeth, and ways of both managing and preventing dental erosion.

Dental erosion is defined as loss of dental hard tissue by a chemical process that does not involve bacteria.[1] There is a perception among some dental clinicians that the occurrence and severity of dental erosion is increasing markedly. Epidemiological evidence now appears to confirm these observations. In a national survey of more than 2000 children in the United Kingdom,[2] as many as 50 per cent of the five- and six-year-old patients studied displayed erosion of their first teeth, and 25 per cent were

found to have had severe erosion involving the dentine or pulp (nerve) of the teeth. Evidence of tooth erosion near the roof of the mouth was detected in more than a quarter of the subjects over 11 years of age, rising to 32 per cent of 14 year olds. Erosion was sufficiently severe in 13 to 15 year olds to have penetrated to dentine in between two and three per cent of the subjects. These alarming statistics appear to indicate an increasing and significant problem for the individual and the community.

SOURCES OF ACID

In simple terms, the acid required for erosion to occur will either be acid going into the mouth (extrinsic) or acid coming out of the stomach (intrinsic). The effect of the acid will be determined by its volume, type and concentration, the length of time it is in contact with the tooth surface, and its ability to overcome the environment of the saliva and the resistance of the tooth.

Extrinsic acids

Dental students are familiar with dramatic textbook photographs, usually of workers in battery acid factories, who suffered from occupational exposure to acids in the air they breathed. No teeth could be seen in these subjects even when they were smiling because all the tooth surfaces exposed to the air had been lost. Before effective occupational health measures were adopted, exposure to acids was common in the chemical and metal industries. Nowadays, occupational exposure is rare, but it is still found routinely in some fields, such as professional wine tasting. Exposure to extrinsic acid can also be associated with such activities as frequent swimming in chlorinated pool water that has not been maintained at the optimal pH.[3]

Dietary considerations

Eating acidic foods and drinking acidic beverages are the most common sources of extrinsic acids that cause dental erosion. Some medicines, such as vitamin C preparations and iron tonics, also contribute acid. Some chewable vitamin C tablets have a pH of about 2.0.

A healthy diet may also contain a substantial quantity of acidic foods. Healthcare workers advocate fresh fruit as a component of a 'balanced' diet as a 'healthy' option, but some fruits have a high acid content. Additionally, in an attempt to control body weight, patients may consume acidic foods such as fruit juices and diet drinks in place of high-calorie alternatives. Diet foods and low-calorie beverages are invariably acidic. The potential for erosive damage by these foodstuffs is not well recognised by the public.

In one study, it was found that the erosion risk for individuals who consumed citrus fruits more than twice a day was 37 times greater than it was for those who consumed citrus fruits less often.[4] Thus, the critical frequency of consumption was more than twice a day. For soft drinks, the critical frequency of consumption was once a day or more. Many soft drinks contain citric, phosphoric, carbonic, and other acids, and their pH value is often less than 4.0. Soft drinks are marketed to the young adult, promoted as being healthy, and are linked with high-profile sportsmen and women. In the United Kingdom, 42 per cent of fruit drinks are consumed by children between the ages of two and nine.[5] Adolescents and children account for 65 per cent of sales of acidic drinks. Sales of soft drinks have increased sevenfold since 1950.

Increasingly popular are the new 'sports drinks'. In a Scandinavian study, sports drinks had the same acidogenicity as fruit juice and carbonated beverages.[3] Most contain citric acid.

The demineralising effect of citric acid is especially great because its chelating action on the enamel calcium continues even after the pH increases at the tooth surface (the citrate ions bind with the calcium in the tooth, thereby forming soluble citrates).[6] It should be noted that the titratable acidity of a solution, that is, the total amount of H+ able to dissociate, is a better indicator of erosive potential than the pH value, which solely measures H+ concentration of the solution. Studies indicate that 5.5 is the critical pH for tooth enamel demineralisation.[7]

Intrinsic acid

There is increasing evidence that regurgitation erosion is a major contributor to tooth tissue loss.[8] Acid moving through the lower oesophageal sphincter into the oesophagus is described as gastro-oesophageal reflux. In some patients, the acid movement becomes chronic and painful and requires treatment. This is termed gastro-oesophageal reflux disease. Regurgitation is the reflux of gastric juice through the upper oesophageal sphincter into the oral cavity. Many factors, including hiatus hernia, pregnancy, obesity, chronic alcoholism, certain types of exercise, and some foods, have been linked to gastro-oesophageal reflux. Reflux symptoms may include heartburn, epigastric pain and regurgitation, although many patients are symptom-free.

It is estimated that more than 60 per cent of the population suffer from gastro-oesophageal reflux at some time,[9] and the abundance of antacid preparations in supermarkets is evidence of the widespread self-medication for this condition. If the reflux becomes longstanding, continual damage to the oesophageal lining can lead to oesophagitis, stricture, ulceration and, in some cases, malignant changes. Further, some medicines have the potential to provoke or increase gastro-oesophageal reflux. These include anti-spasmodic drugs, non-steroidal anti-inflammatory drugs and anti-cholinergics.

Eating disorders such as anorexia and bulimia nervosa have been shown to cause a similar pattern of dental erosion, which is often initially evident on the palatal

surfaces of the maxillary anterior teeth. Rumination (voluntary regurgitation), especially in patients who are mentally handicapped, can also be a possible cause.

The presence of palatal dental erosion and any symptom of reflux is indicative of pathological gastro-oesophageal reflux. In those patients without reflux symptoms but with significant unexplained palatal erosion, gastro-oesophageal reflux should always be suspected, and referral to a gastroenterologist will often be indicated.

CO-FACTORS

Saliva

It is possible that the quantity and quality of saliva may be as important in the development of dental erosion as the concentration or frequency of acid attack. At normal salivary flow rates, acidic drinks are eliminated from the mouth in about 20 minutes and the pH at the tip of the tongue remains low for only about two minutes after the drink has been consumed.[10] In contrast, in patients with low salivary flow rates, the pH remains low for more than 30 minutes. Although the role of salivary calcium and phosphorous in remineralisation following acid attack is not yet fully understood, the buffering capacity of the saliva is of critical importance.

As discussed in the article by Mark Shifter in the March issue of the *NSW Public Health Bulletin*, many prescribed medications lead to xerostomia (dry mouth). This results in less dilution and buffering of the acid as well as less lubrication during mastication. This must exacerbate attritional and abrasive wear of eroded surfaces. Those people who moisten their mouths with acidic drinks will have a reduced buffering capacity and will suffer accelerated erosive damage.

Saliva is known to be important in protecting the oesophagus from gastric acid damage after reflux, and the introduction of acid into the oesophagus is known to stimulate salivary output and buffering capacity.

Teeth

Not all teeth are created equal. It would be expected that those teeth that have had the benefit of fluoride during development will resist acid demineralisation, while already hypocalcified teeth would be more at risk.

RECOGNITION

It is often difficult to differentiate between erosion, attrition and abrasion, as these conditions invariably occur in synergistic combination. Evidence now suggests that erosion is a major contributor in tooth wear. Clinically, lesions indicating an erosive cause are often characterised by:

- smooth enamel surfaces, with developmental ridges absent
- areas of exposed dentine, often with a fine margin of intact enamel at the gingival crevice
- restorations that are standing 'proud' of (higher than) the surrounding tooth surface
- depressions in the cusp tips of posterior teeth, creating a 'rock-pool' effect.

When erosion is suspected, it is essential to take a detailed history that includes an investigation of past and present diet, medical history, medications, slimming habits, occupation, sporting activities, and other factors. It should be remembered that the dentist may be the first to suspect certain psychological diseases, such as bulimia nervosa about which the patient may be secretive. Confrontation in such situations may be counterproductive and, because dentists are not qualified to treat these underlying conditions, a judicious referral to the patient's general medical practitioner is warranted.

SUGGESTIONS FOR PREVENTING DENTAL EROSION

It is advisable to provide patients with printed information giving dietary advice and oral hygiene hints when dental erosion has been detected. This can contain information such as:

- Be aware of the acidity of the foods and beverages you consume.
- Never brush your teeth immediately after eating or drinking anything acidic, otherwise there will be no opportunity for remineralisation.
- Try to always rinse with water after having anything acidic.
- Never have anything acidic last thing before going to bed without rinsing your mouth thoroughly with water because your swallowing frequency and saliva production decreases dramatically during sleep.
- Don't allow a toddler to drift off to sleep with a bottle containing fruit juice. Water is best.
- Don't sip acidic drinks. Belting down a Solo is less damaging to your teeth!
- Remember that fruit juices are generally more erosive than whole fruit, and dried fruits can be very acidic.
- When undertaking physical exertion (sport, gym, etc.), always rinse your mouth with water after drinking a sports drink or soft drink, especially when your mouth has become really dry.
- Never put a mouthguard into an unrinsed mouth.
- Avoid chewable vitamin C tablets.

MANAGEMENT

Following differential diagnosis, it is critical that the source of the acid be identified if at all possible. This

source can then be removed or modified, and preventive measures, such as the prescription of neutral fluoride mouth rinses, can be undertaken. It is important to monitor and record the rate of loss of tooth structure. This is best undertaken using accurate study models that are retained and compared over time. Intraoral photographs may be of some use. Emphasis should be on patient education, behaviour modification and careful monitoring. In most cases, active restorative treatment is not indicated until the preventive measures have been shown to be effective and, even then, only in certain circumstances. These circumstances include:

- significant aesthetic concerns
- loss of vertical dimension that will compromise any possible future treatment
- pain from dentine hypersensitivity that does not respond to conservative treatment.

If required, restorative treatment may vary from the simplest of bonding procedures to extremely complex rehabilitation comprising multiple crowns and onlays.

CONCLUSION

The increasing numbers of older people using medications associated with xerostomia, the increasing use of drugs that can promote gastro-oesophageal reflux and the increasing consumption of acidic soft drinks, fruit drinks and sports drinks all contribute to dental erosion becoming a larger public oral health problem. Just as dental erosion has many factors relating to its aetiology, management and treatment, so there is no single solution. Product modification by the food and drink manufacturers, community education, and individual patient behaviour modification all are required to reduce the damage from this growing problem.

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INFECTIOUS DISEASES, NSW: APRIL 1999

TRENDS

Reports of infectious diseases followed largely expected trends through to early autumn (Figure 1). Statewide, **arbovirus infections** appeared to plateau in March, although late-season increases in **Ross River virus infections** (RRV) were reported in coastal regions. This was notably in the Hunter, Illawarra, Northern Rivers and Mid North Coast areas (Table 1). In inland NSW, increases in RRV were reported earlier, in late spring and early summer. **Barmah Forest infections** also have been reported from the Northern Rivers, Mid North Coast and Illawarra areas, albeit in fewer numbers.

There are encouraging signs that the epidemic of **gonorrhoea** (mainly affecting men in inner Sydney) may have levelled off, although it is probably too early to be sure, given reporting delays. The number of reports of **meningococcal infection** has fluctuated markedly in recent months, with 20 cases in January, seven in February, and 19 in March. This is a reminder that, while this disease tends to peak in winter and spring, it can occur in any season. Reports of pertussis cases are again abating, following a peak last spring.

ASIAN TIGER MOSQUITO FOUND IN BOTANY BAY

A single 'Asian tiger' mosquito (*Aedes albopictus*) was identified in routine trapping carried out by the Australian Quarantine and Inspection Service (AQIS) at Botany Bay in early April. This species is a known vector for **dengue fever** in Asia, and has never before been reported in New South Wales. The *Aedes albopictus* probably arrived on a container ship, possibly as an egg laid in stagnant rainwater. In response to the finding, an intensive mosquito-trapping program was initiated in collaboration with the Medical Entomology Department at Westmead ICPMR and AQIS. No other mosquito of this species has been detected subsequently.

FIGURE 2

REPORTS OF SELECTED INFECTIOUS DISEASES, NSW, JANUARY 1994 TO MARCH 1999, BY MONTH OF ONSET

These are preliminary data: case counts in recent months may increase because of reporting delays

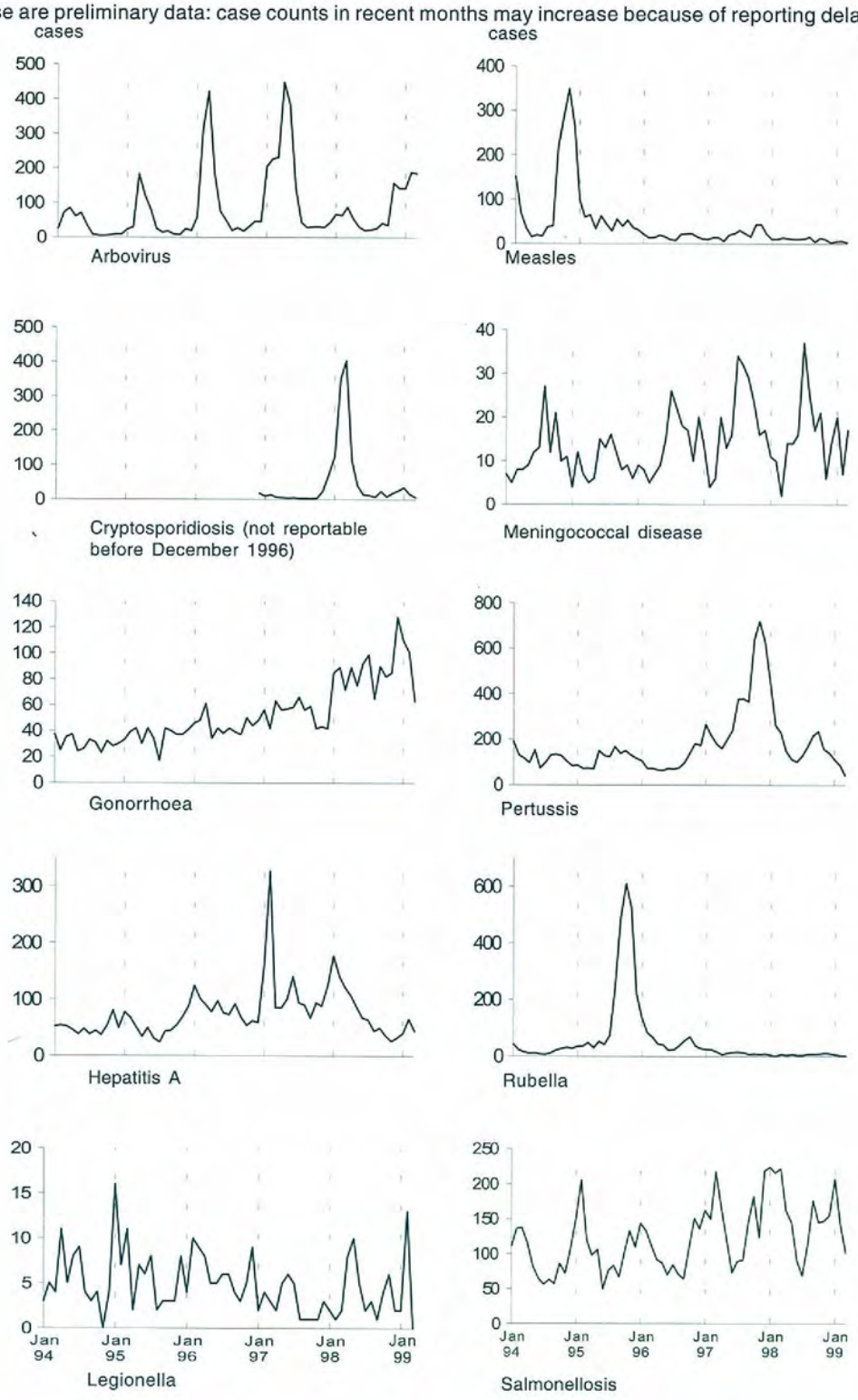

TABLE 2 INFECTIOUS DISEASE NOTIFICATIONS RECEIVED IN MARCH 1999 BY AREA HEALTH SERVICES

Condition	Area Health Service (1999)																	Total	
	CSA	NSA	WSA	WEN	SWS	CCA	HUN	ILL	SES	NRA	MNC	NEA	MAC	MWA	FWA	GMA	SA	for Mar†‡	To date‡
Blood-borne and sexually transmitted																			
AIDS	1	2	1	–	–	–	–	–	–	–	1	–	–	–	–	–	–	5	60
HIV infection*	1	3	–	–	–	–	–	1	5	–	–	–	–	–	1	–	–	33	89
Hepatitis B: acute viral*	–	–	–	–	–	–	–	–	1	1	–	1	–	–	–	–	–	4	14
Hepatitis B: other*	56	43	17	3	–	7	6	12	31	3	6	4	3	2	1	2	2	200	746
Hepatitis C: acute viral*	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	11
Hepatitis C: other*	106	68	49	45	1	29	54	31	113	42	47	18	6	52	11	15	36	728	2,082
Hepatitis D: unspecified*	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	1
Hepatitis: acute viral (not otherwise specified)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Chlamydia (genital)	11	4	6	6	1	9	23	10	16	17	9	8	4	17	11	16	3	175	499
Gonorrhoea*	13	13	3	1	–	–	1	–	54	1	–	–	–	9	3	–	–	102	327
Syphilis	10	3	7	–	–	–	–	1	7	–	3	1	2	1	5	–	1	41	133
Vector-borne																			
Arboviral infection*	2	6	6	5	1	3	54	20	5	28	29	7	8	16	11	24	29	256	570
Malaria*	–	2	–	1	1	2	1	1	1	6	2	2	–	1	–	–	–	20	59
Zoonoses																			
Brucellosis*	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2
Leptospirosis*	–	–	–	–	–	–	–	–	–	3	–	–	–	–	–	–	–	3	11
Q fever*	1	–	–	–	–	–	1	–	–	–	4	4	2	2	1	–	–	15	39
Respiratory and other																			
Blood lead level	2	–	5	3	3	2	5	3	1	1	–	–	–	3	25	1	–	54	226
Legionnaires' disease	–	–	1	–	–	–	1	1	–	–	–	–	–	–	–	–	–	3	17
Leprosy	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Meningococcal infection (invasive)	–	1	5	2	1	1	1	3	2	–	–	1	–	–	–	2	–	19	45
Mycobacterial tuberculosis	12	8	1	2	–	–	–	–	7	–	1	–	–	–	1	–	–	32	83
Mycobacteria other than TB	7	8	–	–	–	2	4	1	21	3	–	–	–	–	–	3	–	49	107
Vaccine-preventable																			
Adverse event after immunisation	3	–	–	–	–	–	–	–	1	–	2	–	–	–	–	–	–	6	19
H. influenzae B infection (invasive)	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	–	1	3
Measles	–	–	–	–	–	1	–	1	1	–	–	–	1	–	–	–	–	4	14
Mumps*	–	1	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	2	5
Pertussis	10	13	11	8	8	8	9	6	13	3	1	2	2	2	1	6	8	111	354
Rubella*	–	–	–	–	–	–	–	–	–	2	1	–	–	–	–	–	–	3	13
Tetanus	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Faecal-oral																			
Botulism	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Cholera*	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Cryptosporidiosis	–	–	1	–	–	–	1	2	–	2	1	–	1	1	1	2	–	12	68
Giardiasis	13	25	14	7	–	2	8	7	11	11	6	5	1	8	–	3	–	122	321
Food-borne illness (not otherwise specified)	2	–	–	–	–	–	–	–	–	3	–	–	–	–	–	–	–	5	6
Gastroenteritis (in an institution)	9	–	–	–	–	31	–	–	–	–	–	–	–	–	–	–	–	40	60
Haemolytic uraemic syndrome	1	1	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	3	6
Hepatitis A	16	5	8	4	6	–	1	3	17	–	2	1	–	–	–	2	1	68	159
Hepatitis E	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Listeriosis*	–	1	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	2	6
Salmonellosis (not otherwise specified)*	10	26	11	5	2	6	10	11	22	21	3	6	2	4	3	8	4	155	534
Typhoid and paratyphoid*	–	–	2	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2	5
Verotoxin-producing *E. coli*	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–

* lab-confirmed cases only † includes cases with unknown postcode ‡ except CCA

CSA = Central Sydney Area
NSA = Northern Sydney Area
WSA = Western Sydney Area
WEN = Wentworth Area
SWS = South Western Sydney Area
CCA = Central Coast Area
HUN = Hunter Area
ILL = Illawarra Area
SES = South Eastern Sydney Area
NRA = Northern Rivers Area
MNC = North Coast Area
NEA = New England Area
MAC = Macquarie Area
MWA = Mid Western Area
FWA = Far West Area
GMA = Greater Murray Area
SA = Southern Area