Changes in oral health and condition with age

M G Brading and A Joiner

Bebington, UK

D F Kinane

Philadelphia, USA

In recent years, there has been a dramatic shift in the overall oral health status of popu­lations in the developed world: people are living longer and retaining more natural teeth into old age. While this can be viewed as a sign of improvement, this situation brings its own problems. The prevalence of caries, enamel erosion and chronic periodontal disease increases with age and, for the individual, the effects of these oral diseases also accu­mulate over time. New problems arise in consequence, for example the prevalence of

dentine hypersensitivity increases as roots are exposed due to increasing gingival reces­

sion. Plaque control can become more difficult with increasingly complex restorations and calculus accumulates. There are also issues relevant to social confidence: tooth colour

darkens with ageing, in terms of intrinsic colour and also extrinsic tooth stain, both of

which can affect perception of overall loss of whiteness. In this overall context, the role of personal oral care products and daily oral hygiene routines becomes even more important. This review examines how oral health and condition of the mouth changes with age and describes the potential for modern day toothpastes to reduce unwanted effects that occur during the ageing process.

*Key words: Ageing, caries, erosion, gingivitis, periodontitis, gingival recession, calculus, colour, tooth sensitivity, anti-age toothpaste, dentifrice, zinc citrate, vitamin E, potassium citrate*

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Introduction

The past several decades have seen a dramatic shift in dental healthcare in the developed world’. The improv­ing oral health status of populations, such as people keeping more teeth for longer, has brought impressive benefits but at the same time, has created or raised awareness of other oral and dental health problems2. The focus has changed over time from the restora­tion and extraction of carious teeth, to the prevention of caries and the maintenance of periodontal health throughout life’. As dentition is kept for longer, other problems appear such as secondary caries around ex­isting fillings, root caries due to gingival recession and exposed dentine, and sometimes an increased malo- dour due to reduced salivary flow associated with the increased medication typically taken by elderly people. Existing problems such as calculus, extrinsic stain and dentine hypersensitivity may also increase to being sig­nificant issues for these patients.

This review examines the effect ageing has on the overall health of the mouth and describes how modern day toothpastes can aid in managing these effects during the natural ageing process.

French Insight Study

As well as relevant literature citations, this review also contains data that were obtained from a study con­ducted to determine the dental health status in French Consumers, related to key demographic parameters including age, gender and level of education. The study was carried out in seven locations in France, including one metropolitan, two urban and four rural communi­ties. Research International carried out the recruitment of the subjects for the study on behalf of Unilever Oral Care. The population was proportioned by age, gender and level of education at each location. About 880 subjects (roughly half male and half female), aged between 15-64 years old, were interviewed and then as­

sessed by two dentists. Key parameters from this study are included in this report where relevant.

The effect of ageing on dental caries

A recent review 4 of the available epidemiological data from many countries clearly indicates that there is a marked increase in the prevalence of dental caries, challenging the historical consensus that dental caries prevalence was in decline 5. This global increase is found in children and adults, primary and permanent teeth, and include coronal and root surfaces. While the causes of this increase are unclear and opinions differ, the rem­edy is well known and includes oral health educational programmes, an emphasis on twice daily tooth brushing with a fluoride toothpaste, a reduction of intake in sug­ary foods and regular dental check ups4.

Dental caries can be observed early in life, and is reported to be the most common chronic childhood disease of children aged 5 to 17 years in the USA4. Car­ies in the permanent dentition can be found soon after the eruption of the first permanent molars, where it usually begins in the pits and fissures 6.

Many epidemiological studies around the world have focussed on children and it is clear that caries prevalence increases with age. There are many examples of this *vi%amongst* Brazilian children aged 0-5 years7 and 5 and 12 years old ; Chinese children aged 3-5 years ; Mexican children aged 6-9 and 6-12 years10\*11; United Kingdom children aged between 5 and 8 years and between 12 and 15 years; Children from the USA, aged 5-17 years old4. However, despite the focus here on caries in chil­dren, caries remains a major health issue that increases in magnitude throughout life. For instance, in an exten­sive National Oral Health Survey across India , it was found that the prevalence of dental caries, for both coronal and root surfaces, increased .with the subject’s age. The prevalence was 51.9%, 53.8% and 63.1% in 5, 12 and 15-year-old children respectively. The prevalence increased to 80.2% and 85.0% in adults aged 35-44 and 65-74 years old respectively. The observation that the percentage of adults with experience of caries, restora­tions or missing teeth increased with their age has also been reported in other adult populations. For example, in Australia the mean number of coronal filled surfaces is reported14 as 2.7% for 16-19 year-old subjects and in­creased to 6.3%, 20.1% and 33.8% for 20-34, 35-49 and 50-64 year-old subjects respectively Similar increases are reported14 for a USA adult population, from 4.4% for 16-19 year-old subjects to 27.3% for 50-64 year-old subjects. In a Swedish epidemiological study of 987 subjects in 2003 15, the number of individuals without caries and restorations decreased with age from, for example, 12% of the population at age 20 years to 0% at age 40 years. The corresponding decayed and filled teeth (DFT) increased from 5.9 at 20 years to 113 at 40 years and 17.0 at 60 years. Zhang *eta£A'-*conducted a systematic review of the oral health status of Chinese adults and found that the decayed/missing/ filled teeth (DMFT) index significantly increased with age. Rural subjects presented higher DMFT than urban which increased markedly for rural subjects over 45 years of age. In the UK Adult Dental Health Survey , the mean proportion of teeth which were filled but otherwise sound, increased with subject age from 9% in the 16­24 years age group to 39% by the age of 45-54 years. These studies demonstrate there is an overall increase in caries prevalence with age, however, the pattern and presentation of caries can also change with age. As al­ready mentioned, children first develop coronal caries in pits and fissures, but in those countries with access to professional dental care, these primary lesions are typi­cally already restored. Therefore secondary caries is the problem, particularly as the restorative cycle progresses with increasing complexity of restorations, and the concomitant increase in difficulty of maintaining good plaque control.

One caries problem generally found in older age- groups is that of root caries. Greater life expectancies combined with improvements in tooth retention have resulted in increased numbers of people who have re­tained their teeth into old age. This increase in number of teeth combined with the increase in gingival recession results in older persons with more root surfaces exposed to bacterial plaque accumulation and at greater risk to root caries18. For example, the mean Root Caries Index value in a Swedish population significantly increased with age from 14% to 16% and 22% in the 55, 65 and 7 5 year-olds, respectively19.

In summary, older people are a group prone to ac­tive caries, a conclusion from Thompson20, in a recent review of dental caries experience studies with at least three years of follow-up in older people (> 50 years). Importantly, the widespread perception that root surface caries is their only problem is erroneous: Thompson2 found that while both coronal and root surface caries contributed to the observed increments, there was a consistent pattern whereby coronal caries made the greatest contribution to the overall increment. This finding supports his suggestion that clinical preventative measures for older people should clearly be directed at all caries. It is well established that regular brushing of the teeth with a toothpaste containing fluoride can re­duce the incidence of dental caries21. Indeed, in a clinical study, fluoride toothpaste usage was found to be effec­tive in preventing both coronal and root caries 22

The effect of ageing on tooth wear and erosion

In the current era of improved dental awareness and self-care, many individuals retain their dentition for longer than ever before; and thus the dental tissues must withstand greater exposure to the physicochemical insults imposed by diet and oral hygiene practices which

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can lead to tooth surface loss or tooth wear1. Tooth wear is considered a multifactorial process that may encompass erosion (chemical action), attrition (tooth- to-tooth contact), and abrasion (physical wear, such as from tooth brushing), where erosion is considered the dominant factor23, . Dental erosion is commonly defined as the chemical dissolution of the dental hard tissues without bacterial involvement 25. Erosion is gener­ally caused by acids of either an intrinsic (e.g. stomach acid) or an extrinsic (e.g. dietary acids) nature giving rise to the dissolution of the hydroxyapatite component of dental hard tissues. In enamel, this results in a softened layer of tissue which if not re-hardened, for example by a remineralising agent such as saliva, may be lost due to further dissolution or physical insult25. Once the softened enamel layer is physically lost, it is not replaced and the result is ever decreasing enamel thickness. This can give the affected surface a smooth, glazed appear­ance26 On anterior teeth, enamel may be thinned suf­ficiently for the edges to appear translucent and with further tissue loss there can be chipping of the incisal edges 2 which can ultimately lead to an unacceptable appearance of the teeth27. With the loss of enamel and subsequent exposure of dentine, there is a further risk of hypersensitivity2 .

In a recent review25, the main strategy for minimising tooth wear was described as education. This is because individual’s tooth wear is asymptomatic in the early stages and therefore early detection and intervention by the dental health professional will be of the utmost importance. The review also stated that there is evi­dence that fluoride can provide some protection against erosion, and that the use of fluoride toothpastes and mouthrinses should therefore be encouraged.

The increase in erosion has been demonstrated

in children and young people. For example, in-a UK study29 with 1,308 children aged 12 years at baseline and then re-examined two years later, it was found' that the prevalence of deep enamel lesions increased from 4.9% to 13.1%. Similarly, in a review of data from UK dental surveys of young people it was also concluded that erosion increased with age of children and ado­lescents over time30 In another study31 with groups of subjects aged 10-13 years old and 15-16 years old from the Netherlands, the percentage of subjects with visible smooth wear was 3% in the younger group and 30% in the older group.

In terms of the prevalence of tooth wear in adults, a recent systematic review concluded that the predicted percentage of adults presenting with severe tooth wear increases from 3% at the age of 20 years to 17% at the age of 70 years and that increasing levels of tooth wear are significantly associated with age32. Similarly, Bartlett and Dugmore concluded in their recent review that literature evidence demonstrates that normal levels of erosion and wear are age dependant. Further, in another recent review34 of non-carious cervical tooth surface

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| loss it | was | concluded that the | older the | population | | be- |
| comes | the | greater percentage | of lesions | are | found, | the |
| greater | the | number of lesions | per individual | | occur | and |
| that the lesions become larger. | | |  |  |  |  |

In a study with groups of subjects aged 26-30 years old and 46-50 years old in Switzerland, the prevalence and severity of erosion was greater in the older age group . For example, erosion with involvement of dentine was observed in 7.7% and 13.2% of the younger and older aged groups respectively. A sub-set of the original groups was re-examined six years later and it was found that there was a distinct progression of ero­sion on facial and occlusal surfaces and wedge-shaped defects36. The increase in the defects was more marked in the older group.

The effect of ageing on gingivitis

Chronic marginal gingivitis is defined as the ‘non-spe­cific, reversible inflammatory response to dental plaque involving the gingival margins’ 37. If adequate oral hy­giene is restored and professional cleaning undertaken as necessary, gingivitis usually resolves and the tissues become clinically healthy again. In this way, Lang *et al?\**commented that gingivitis may be perceived as the protective host response against bacterial challenge. However, their review of long-term longitudinal data clearly demonstrated the importance of gingivitis: teeth with consistently non-inflamed gingivae had a 50 year- survival rate of 99.5%, while those with consistently inflamed gingivae had 70% more attachment loss and a 50 year-survival rate of only 63.4%. Based on these data, they concluded that persistent gingival inflamma­tion is a clinically significant risk factor for periodontal attachment loss and tooth loss38.

Factors that enhance plaque retention or impede plaque removal such as crowded or crooked teeth, crowns and other restorations are predisposing factors for gingivitis and the persistence of this inflammation may develop into chronic periodontitis. There is a cor­relation between gingival bleeding and age that could be in part due to the accumulation o f these impediments to plaque control39. Plaque control at the gingival margin can also be complicated by restorations and gingival recession: Robinson40 found that patients over 60 years of age required more frequent oral prophylaxis for the maintenance of gingival health than individuals under 25 years old. This finding was interpreted to be largely due to the increased incidence of exposed root surface in older individuals which allows more rapid plaque accu­mulation and calculus formation than enamel surfaces.

Data from experimental gingivitis studies also indi­cate a change in the inflammatory response of gingivae to plaque with age. Robinson40 showed that elderly individuals develop more rapid and severe gingivitis than young individuals following the abstention of oral hygiene. Likewise, Fransson *etal? x* found that while 65-

80 year-old subjects formed similar amounts of plaque as subjects 20-25 years of age during a 3-week period of abstention from oral hygiene, they developed more gingivitis. This age-related increase in gingivitis was seen in clinical assessments, gingival crevicular fluid volume and also morphometric analysis of histological samples: more inflammatory cells were also found in lesions of older individuals. Similar findings have also been reported by Tsalikis eZö/\*2who compared experimental gingivitis in 20-22 year-olds with that in 61-65 year-olds. In this study, clinical signs of gingivitis and gingival crev- icular volume were more pronounced in the older group. Gingival crevicular fluid levels of the pro-inflammatory cytokine interleukin-1 beta at day 21 of no oral hygiene were also significantly higher in the older group, whereas they remained low for the younger adults. In contrast to Fransson *etal\*\* within this particular study, the plaque accumulation during the experimental period was also significantly higher in the older age group.

Regarding the natural history of gingival inflamma­tion, a review by Anerud *etal\*3* discussed the fact that changes from 'healthy gingiva' to a 'chronic gingivitis' take place early in life and that both the number of in­dividuals with gingivitis as well as the degree of severity within individuals increase through the teens and early twenties. However, the progress of the disease does de­pend on the socio-economic status of the individuals. In the Norwegian population studied, there was no general increase in prevalence and severity of gingivitis up to 40 years of age. In comparison, a similar study in Sri- Lanka showed quite severe forms of gingivitis already by the age of 20 and then a steady increase in mean gingivitis levels through the twenties and early thirties so that at 40 years of age more than 97% of sites bled on probing. Whilst this study appears to highlight two extremes of the condition, it is clear that it is rare for individuals to maintain the gingival health that is seen in early adulthood and the tendency is that gingivitis worsens with age.

The effect of ageing on periodontitis

Gingivitis and periodontitis are considered to bea con­tinuum of the same inflammatory disease44. Chronic adult periodontitis differs from chronic marginal gin­givitis in that there is also loss of attachment between the root surface, the gingivae and the alveolar bone and bone loss itself may occur. The loss of attachment is accompanied by apical migration of the junctional epi­thelium. This is evident clinically by either a pathologi­cally deepened gingival crevice, termed a periodontal pocket, or gingival recession (see below) or indeed a combination of these two, depending largely upon the anatomical situation.

Chronic adult periodontitis is the most common form of periodontitis affecting the general population and is the major cause of tooth loss after the age of

2537 Albandar *etal\*3* found from their studies that the prevalence of periodontitis and the prevalence and extent of attachment loss increase considerably with age. Destructive periodontitis and attachment loss were consistently more prevalent in males than females and more prevalent in blacks and Mexican American than whites 45.

Interestingly however, results from a study by Abdel- latif and Burt showed that oral hygiene was the most important predictor for periodontitis and that the effect of age on the progression of periodontitis could there­fore be considered negkgible when good oral hygiene is maintained. That said, research on the epidemiology of periodontal disease suggests that the disease in older adults is probably not due to greater susceptibility but is instead the result of cumulative disease progression over time 47. In the UK for example, 85% of the population 65 years of age and over had some attachment loss of 4mm or more compared with just 14% in the 16-24 age group '. Likewise in the US, the proportion of adults with at least one site with loss of 2mm or more was 86.4% for the 45-54 age group compared with 37.3% for the 1 8-24 age group48

As mentioned above, periodontitis and gingivitis are considered to be part of the same inflammatory continuum and the consensus of opinion is that preven­tion of gingivitis prevents periodontitis 44. Furthermore, gingivitis is now recognised to be a signification risk factor for periodontal attachment loss and tooth loss38. Therefore controlling gingival inflammation at an ear­lier stage would lead to a reduction in the more serious form of the disease that becomes more prevalent in older adults.

The effect of ageing on gingival recession

Gingival recession is a condition where the gingival margin recedes from its normal attachment at the crown margin with the root to go further apically down the tooth root. The gingival margin then lies against the root surface of the teeth and the root is thus exposed. In affected persons, recession can cause increased sen­sitivity of teeth and may even lead to loss of vitality of the affected teeth. In their studies, Albandar and Kingman 39 reported that there was a significant increase in prevalence, extent and severity of gingival recession with age, a fact that has also been reported by other authors 49,5°.

A number of factors have been shown to be im­portant for gingival recession, including gender, mal- positioned teeth and tobacco consumption .Joshipura *et al\*9* found subjects who brush with excess vigour are likely to have root surface exposure as a result of the trauma due to brushing. However, gingival reces­sion is thought to occur primarily as a consequence of periodontal diseases and whilst over-use or aggressive manipulation of mechanical oral hygiene aids such as toothbrushes or floss are common causative factors, anatomical and other factors may also be involved. Jo- shipura *etal.* also reported that subjects with poor oral hygiene were more likely to have root surface exposure and this was attributed to periodontal disease. Lang *et al3\** stated that it has been convincingly demonstrated that development of periodontitis only occurs in areas of long-standing gingivitis. It can therefore be con­cluded that good control of gingivitis in its early stages will also aid in reducing gingival recession.

The effect of ageing on oral soft tissues

Whilst age-related changes in the oral mucosa remain open to interpretation, it has been reported for many years that clinically the aged oral mucosa is often smoother and dryer. Shklar51 found distinct differences between oral mucosa of young individuals and the oral mucosa of elderly persons, including epithelial and

connective tissue atrophy, van der Velden 52 reported re­search findings suggesting that the degree of periodon­tal breakdown increases with age, that with increasing age inflammation of the periodontium tends to develop more rapidly and that in the process of ageing the peri- dontium shows a slower rate of wound healing. More recently, Karube *etal33* studied gingival microstructure

according to a number of conditions. No difference was found according to underlying disease, medications or the presence or absence of dentures but changes were noted in the microstructure of the gingiva according to age. Likewise, Vandana and Savitha 54 examined the thickness of gingiva in association with a number-of parameters including age in a group of Indians with an age range of 16-3&years. They found that the younger age group had significantly thicker gingiva than that of the older age group.

Despite the findings of Karube *et al,53* reported above, many authors believe that changes in the oral mucosa may be a reflection of associated systemic dis­ease and/ or medication rather than intrinsic age related changes. For instance, saliva is known to play an essential role in the maintenance of oral health. Many older adults take medications many of which are associated with decreased saliva, dry mouth and xerostomia 47, which in turn can have an effect of overall dental health. Nies- sen and Fedele 47 also reported that oral and pharyngeal cancers increase with age which again can impact the overall health of the oral mucosa. This Ending was also reported in a review of the area by Napier and Spei- ght55, stating that older patients, particularly females were more at risk than younger patients at developing potentially malignant oral disorders.

with microorganisms. The pioneer species are the first to colonise such as *Steptococcus. salivarius,S. mitis* and 5\*. *oralis.* After this, the eruption of teeth has a signifi­cant ecological impact on the oral environment and its resident microflora with the acquisition of some genera such as 5. *sanguinis and* X *mutans* occurring optimally at certain ages 56\*58. Puberty causes an increase in spirocha- etes and black-pigmented anaerobes, possibly due to hormones entering the gingival crevice and acting as a novel nutrient source. Eventually, a climax community is reached 59, the resident oral microflora remaining rela­tively stable and coexisting in reasonable harmony with the host37 This stability is termed microbial homeostasis and is due to a dynamic balance among the resident flora through numerous inter-bacterial and host-bacterial interactions. Some life experiences, such as pregnancy may lead to a temporary change in the balance but it is not until later in life that some variations in the oral mi­croflora are seen 37. These variations can be attributed to both direct and indirect effects of ageing. Significantly higher proportions and isolation frequencies of lacto­bacilli and staphylococci (mainly *5\*. aureus) in* saEvawere found in healthy subjects aged 70 or over while yeasts were isolated more often and in higher numbers from saliva in those aged 80 or over. In fact, the incidence of oral candidosis is more common in the elderly due to a number of factors including denture wearing, physi­ological changes to the oral mucosa, malnutrition and to trac element deficiencies 37

The effect of ageing on oral malodour

Malodour in children is relatively rare, even though they may harbour some of the "malodour’ causing organisms. However, oral malodour is relatively common in the adult population. In young adults, it is the metaboEsm of bacteria located on the tongue that accounts for the majority of malodorous compounds found in mouth air. High odour subjects generally have a higher total bacterial load on the tongue, and higher numbers of Gram-negative anaerobes, with higher proteolytic activ­ity, producing volatile sulphur compounds 37 As a person ages, it is beEeved that periodontal disease takes on a more important role in malodour . Indeed, in the older population, oral malodour production may be direcdy related to the patient’s periodontal condition as the production of disagreeable odours occurs more rapidly in patients with periodontal disease. In addition, medica­tions commonly taken in the aged population can induce xerostomia, which can lead to an increase in malodour in this population61, due in part, to the creation of a suit­able environment for associated microorganisms 62

The effect of ageing on oral microbiology

Whist the mouth of a new born baby is usually sterile 37 from the first feeding onwards it is regularly inoculated

The effect of ageing on calculus formation

Calculus or tartar is the term used to describe calcified dental plaque. It consists of intxa-and extracellular de­posits of mineral, as well as protein and carbohydrate. Mineral growth can occur around any bacterium; areas of mineral growth can then coalesce to form calculus, which may become covered by an unmineralised layer of bacteria. Calculus can occur both supragingivally (es­pecially near the salivary ducts) and sub-gingivally, where it may act as an additional retentive area for plaque ac­cumulation, thereby exacerbating periodontal disease.

Over 80% of adults have calculus, either as suprag­ingival and/ or subgingival calculus. In populations with regular oral hygiene and access to professional care, supragingival calculus is first observed in early teen years63. If it is not removed, the levels will obviously in­crease dramatically with time. The French Insight study *(Table shown* that the prevalence of supragingival calculus increases with age. In addition, subgingival cal­culus has also been shown to increase in prevalence and severity with age39. Once formed, professional scaling is required to remove calculus.

The effect of ageing on tooth colour and stain formation

The colour of the teeth is determined by the combined effects of their intrinsic colour, which is influenced by light scattering and absorption within enamel and den­tine, and the presence of extrinsic stains that may form on the tooth surface64'66

It is well documented that as people age their intrin­sic tooth colour has a significant tendency to become darker and more yellow, as determined in a number of study populations 67'73. In the vast array of genetically de­termined tooth colourations, all teeth appear to darken over the course of time 74.

The impact of subject age on tooth colour is be­lieved to be due to a number of factors. As the dental pulp ages it shrinks and leaves secondary dentine in its wake74. At the same time, it is hypothesised that pig­ments and ions of an amorphous organic and inorganic nature permeate and deposit at the enamel-dentine junction, causing the overall dentine chroma to become more saturated74. Due to normal tooth wear processes, the enamel thickness can decrease and the dentine col­our will begin to have a greater influence on the overall tooth colour. The impact on reduced enamel thickness giving rise to increased tooth yellowness has been confirmed with *in vitro* experiments75. The net result of these processes is an overall progressive darkening and yellowing of the teeth with age.

With respect to extrinsic stains, these can also be affected by the general wear, tear and disease of teeth and their supporting tissues that occurs throughout life and that can lead directly or indirectly to tooth discolouration64. Thermal expansion and contraction forces exaggerated by normal tooth flexure can give rise to the creation of cracking and crazing within the enamel surface, which can become more pronounced with time74. Physical trauma can result in the formation of enamel cracks or the bulk loss of enamel, both of which can allow the internalisation of extrinsic stains. In addition, the exposure of dentine through enamel wear or gingival recession can also increase the uptake of staining chromogens into the tooth64.

Extrinsic stain was assessed in the French Insight Study. Staining was scored on the buccal surfaces of the upper and lower incisors. The worst code was recorded for the 8 test teeth. Each code was given a numerical score as shown in *Table 2* and the compound stain index=Intensity\*Area calculated. *Figure 1* shows the mean stain index (I\* A) for each age group and *Table 3* shows the percentage increase in extrinsic stain with each age group.

Control of extrinsic stain is possible with the regular use of a toothpaste76,77. Toothpastes typically contain an abrasive system in order to help reduce or prevent extrinsic stains from forming since a low or non-abrasive paste is unable to prevent extrinsic stains. The toothpaste may contain other ingredients to aug­ment its cleaning performance, such as surfactants and calcium chelators78. The removal of extrinsic stain can be accomplished using whitening toothpastes, which are typically formulated to have an enhanced cleaning performance76,7,9. Interestingly, the removal of existing extrinsic stain had a significant impact on the overall improvement of tooth colour, as clinically measured and perceived by subjects who had used a whitening toothpaste for four weeks77.

The effect of ageing on tooth sensitivity

As discussed by Addy80, the adopted definition for den­tine hypersensitivity states ‘dentine hypersensitivity is characterised by short, sharp pain arising from exposed dentine in response to stimuli typically thermal, evapora­tive, tactile, osmotic or chemical and which cannot be as­cribed to any other form of dental defect or disease’. It is a common oral health problem affecting one or more teeth of many adult individuals. Addy80 commented that hypersensitivity increases with age, with a peak between 30-40 years. In our own studies, hypersensitivity was seen to increase up to and beyond 60 years of age *(Table 4).* The primary cause of exposed dentine is thought to be gingival recession81 but changing diet may also be a factor. Increased consumption of acidic foods and drinks can lead to enamel erosion, a factor which in turn leads to increased dentine exposure. Hypersensitivity is also set to rise in the general population as caries and periodontal disease prevention results in improved oral health status and functionality of the dentition .

Delivery of anti-ageing benefits from a Toothpaste

As seen from all the data reviewed above, increasing age is accompanied by increase in oral problems. Oral

Clinical Parameter

Calculus

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| 15-16 | 17-21 | 22-30 | 31-40 | 41-50 | 51-60 | 61-64 |
| 39 | 69 | 81 | 84 | 88 | 85 | 98 |

Age

with age. Results

from the French Insight Study

**Table 2** Numerical scores for coding used to assess extrinsic stain in the French Insight Study

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Code | Category | Diagnostic Criteria | Score |  |
| N | None | No Stain | 1 |  |
| L | Low intensity | Yellow tan-light stain | 2 | Intensity |
| M | Medium intensity | Medium brown - medium stain | 3 |  |
| H | High intensity | Dark brown or black - heavy stain | 4 |  |
| L | Small area of stain | Stain up to one third of region | 2 | Area |
| M | Medium area of stain | Stain up to two thirds of region | 3 |  |
| H | Large area of stain | More than two thirds of region | 4 |  |

**Table 3** Percentage increase in stain with age. Results from the French Insight Study

Clinical Parameter Age

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
|  | 15-16 | 17-21 | 22-30 | 31-40 | 41-50 | 51-60 | 61-64 |
| Stain | 17 | 17 | 18 | 27 | 26 | 23 | 25 |

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
|  | 15-16 | 17-21 | 22-30 | 31-40 | 41-50 | 51-60 | 61-64 |
| Hypersensitivity | 3 | 1 | 18 | 28 | 33 | 30 | 46 |

**Table 4** Percentage increase in tooth hypersensitivity

Clinical Parameter Age

**y = 0.4154X + 4.6743**

***Figure 1.*** Mean stain index for each age group in subjects with extrinsic stain (n=189) from the French Insight Study

disease and damage is generally progressive and, over time, the effects accumulate and are exacerbated. Also, the changing anatomy due to loss of soft and hard tissue allows new problems to emerge, such as root caries and dentine hypersensitivity. At the same time, throughout developed countries at least, patient expectations of

long-term oral health and aesthetics generally appear to be increasing.

More adults than ever before are retaining more of their natural teeth for longer and the need for effective prevention practices becomes even more important.

As discussed by Albandar and Kingman 39 this will not only improve the level of oral health of the population but could also produce great cost savings for health providers.

Self-performed oral hygiene through tooth brushing has long been the cornerstone of oral prevention. This mechanical approach to plaque removal is a practice now embedded in the developed world, even though it is generally practiced less frequently than the 'twice daily'dental professional recommendation and the levels of dental plaque in populations remain relatively high82. While the commonly achieved efficacy of cleaning by tooth brushing itself may not be ideal, the process has key importance as a means of deEvering topical agents to help protect and maintain the dentition.

The widespread usage of fluoride toothpastes in particular has been attributed as the single most impor­tant factor responsible for reducing caries 83 and there is also evidence that fluoride provides some protection against dental erosion 84,85. The inclusion of stable, avail­able fluoride is therefore generally accepted to be of fundamental importance for toothpaste. However, it is clear that fluoride alone is insufficient to address the problems facing the ageing patient; a multi-functional formulation is needed.

In the mouth, fluoride has complex interactions with calcium that are key for remineraEsation of early caries lesions and for caries inhibition 86,87 Increased levels of calcium in the mouth are associated with a reduction in DMFT (decayed, missing and filled teeth) 88,89. The pres­ence of calcium also affects the overall balance of re- and demmeraksation processes that occur in enamel erosion - addition of calcium to acidic beverages has been shown to reduce their erosive potential significandy 90,9!.

Toothpaste can also be formulated to provide cal­cium' for added benefit and several examples have been reported previously. The addition of calcium glycero­phosphate to a sodium monofluorophosphate (SMFP) toothpaste showed increased dekvery of calcium to plaque 92 with enhanced protection from enamel dem- ineraksation 3 compared to placebo. Calcium benefits are also evident from calcium carbonate-based SMFP formulations, which have been shown to reduce the pH drop of plaque foUowing a sucrose challenge *in vivo\*\** and to reduce enamel demineraksation and enhance remineralisation *in situ\** .More recendy, studies show-

ing that a new toothpaste containing micro-calcium delivered elevated levels of calcium to the mouth and promoted enhanced remineraUsation of demineral­ised enamel lesions 97. Similar findings have also been reported for the use of hydroxyapatite in toothpaste formulations 98. Taken together, these findings support a hypothesis that the provision of an effective source of calcium from a fluoride toothpaste will help to protect against the increasing dental caries and erosion that oc­curs with age.

With respect to gum health, as discussed above, persistent gingivitis is a significant risk factor for loss of attachment and tooth loss, both of which increase with age. Preventing gingival inflammation will therefore help to prevent periodontitis and associated gingival reces­sion. Modulation of the host inflammatory response via redox status is one potential approach99: anti-oxidant deficiency in the oral cavity is associated with poor gum condition 10°. Several toothpastes on the market contain micronutrients such as vitamin E, which has both anti­oxidant and anti-inflammatory effects99, and could potentially be beneficial for gingival health if dekvered to the relevant site of action. While some products have shown delivery of vitamin E to the gingival tissues 102 and penetration into viable gingival tissue layers103, there is limited information on other products.

The more traditional approach in using a toothpaste to prevent gingivitis has been to use specific antimicro­bial agents. Triclosan and zinc are the anti-microbial agents most commonly found in toothpastes and both have clinically proven anti-gingivitis efficacy . The efficacy of a triclosan copolymer system has been re­searched extensively 105 107. Likewise, zinc salts are widely used in toothpastes and mouthrinses and there is a large body of evidence demonstrating it also to be an effica­cious antimicrobial agent *in* vzvo,with both anti-plaque and anti-gingivitis efficacy 105. The chnical efficacy of a 2% zinc citrate formulation in a six month study has previously been demonstrated to reduce plaque and

gingivitis 108 and more recently, the antimicrobial efficacy of a 1% zinc citrate formulation has been reported .

The zinc ion also has additional benefits that are of relevance to the ageing patient. Zinc is well established as an effective anti-malodour agent for the mouth11 ' and together with pyrophosphates and polyphospho­nates zinc has been shown to be effective at restricting the formation of calculus by slowing crystal growth and reducing coalescence 112\*114 This in turn can lead to a reduction in extrinsic stain caused by the formation of calculus and retention of coloured molecules within the calcified material. This is in addition to the stain re­moval effects of toothpaste which are most commonly provided by an abrasive system78.

Regarding abrasivity, it is clear that this is a fun­damental requirement for effective cleaning from a toothpaste, since without abrasives, stain accumulates11 . However, in an ageing population where root dentine is more likely to be exposed by gingival recession, the potential for abrasive formulations to cause unaccept­able dentine wear must be considered. The Relative Dentine Abrasion (RDA) value is commonly used to describe the abrasivity of toothpastes, but this requires appropriate interpretation.

RDA is an *in vitro* measure on dentine specimens and it has been shown that dentine wear *in vitro* increases linearly with increasing RDA 116. However the normal situation in the mouth is different, where dental pellicle, saliva flow and fluoride would be expected to confer a protective effect. This has been seen with pellicle coated specimens *in situ,* where dentine wear rates are significantly reduced compared to water control with no pellicle. The protective effect is such that no signifi­cant difference on *in situ* dentine wear has been found between toothpastes over the RDA range of 90 to 204 1 .This finding remains true in the long-term: with *in situ wear* protocols, there is no significant difference in dentine wear for toothpastes having RDA 90 to 204 after 24 weeks118.

Furthermore, *in situ* wear rates are not linear with time and extrapolation of latter dentine wear rates *in situ* would give approx 450 microns of dentine wear in 80 years, twice daily brushing119 Pickles eZ a//™state: “The data presented here suggest that whilst *in vitro* tests [i.e. RDA] have value in understanding differences in abrasivity between products (for example, for formula­tion optimisation), they cannot be used to predict *in vivo* effects”. Addy and Hunter 2 state: “evidence from *in situ* and *in vivo* suggests that brushing with a toothbrush and toothpaste produces limited dentine wear in a life time of use, and virtually no wear to enamel”. This provides necessary reassurance that the high performance silica cleaning systems generally present in modern tooth­pastes for stain removal79 are suitable and do not pose any undue risk to the mature dentition.

Dentine hypersensitivity can be difficult to resolve and two broad approaches are commonly used in tooth­pastes. First, to interrupt the neural response to pain stimuli and second to occlude open tubules to block the hydrodynamic mechanism. The vast majority of de-sensitising toothpastes contain a potassium salt to ‘numb’ the pain. Potassium nitrate, potassium chloride and potassium citrate are used interchangeably in many countries at a concentration providing 2% potassium ion, which is the densensitising active ingredient81 For tubule blocking strontium chlorid 10% has been present in marketed toothpaste for many years and more recently the efficacy of a formulation contain­ing arginine, calcium carbonate and fluoride has been discussed . However, there is a lack of comparative studies and so far there is no evidence to show superi­ority of newer technologies over older, tried and tested ingredients.

Summary

The challenge for anti-ageing oral care products is to provide relevant multi-efficacy in an appealing format that will be used by individuals to help prevent the de­terioration in oral health that can occur with increasing

age. It is clear that appropriate technologies exist to ad­dress the problems, but most of these seem to appear individually in products focused on single benefits, for example gum health, anti-erosion or anti-sensitivity.

Bringing the relevant ingredients together in a single for­mulation whilst retaining their activity would be greatly beneficial for home care, reinforcing the increasingly important focus on dental prevention.

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References

1. Toumba J. Tooth surface loss —A challenge to oral health. J *CHn Dent 2006* 17(Spec Iss): 85-87.
2. BanoczyJ. Dentine hypersensitivity - general practice considera­tions for successful management. *Int Dent J* 2002 52: 366.
3. Reich E. Trends in caries and periodontal health epidemiology in Europe. *Int Dent J* 2001 51:392-398.
4. Bagramian RA, Garcia-Godoy F, Volpe AR. The global increase in dental caries. A pending public health crisis. *Amer J Dent* 2009 **22:** 3-8.
5. Second International Conference on Declining Caries. *J Dent Res* 1994 44(Sp Iss).
6. Silverstone LM, Johnson NW, Hardie JM *et al.* Dental Caries Aetiology, Pathology and Prevention. 1981 The Macmillan Press Ltd., London.
7. Ferreira N, BeriaJU, Kramer PF *etal* Dental caries in 0-5 year old Brazilian children: Prevalence, severity and associated factors. *Int JPaediatr Dent* 2007 **17:** 289-296.
8. Gomes PR, Costa SC, Cypriano S *etal.* Dental caries in Paulina, Sao Paulo, Brazil and World Health Organization goals for 2000 and 2010. *Cad Sonde Publica200A* **20:** 866-870.
9. Du M, Luo Y, Zeng X *etal.* Caries in preschool children and its risk factor in 2 provinces in China. *Quintessence Int* 2007 **38:** 143-151.
10. Villalobos-Rodelo JJ, Medina-Solis CE, Molina-Frechero N *etal.* Dental caries in school children aged 6-12 in years in Navolato, Mexico: Experience, prevalence, severity and treatment needs. *Biomedica2006* **26:** 224-233.
11. Vallejos-Sanchez AA, Medina-Solis CE, Casanova-Rosado JF *etal.* Caries increment in the permanent dentition of Mexican children in relation to prior caries experience on permanent and primary dentitions. *J Dent* 2006 34: 709-715.
12. Children’s Dental Health in the UK, National Statistics. 2003.

United Kingdom National Technical Reports.

1. National Oral Health Survey and Fluoride Mapping. An Epi­demiological Study of Oral Health Problems and Estimation of Fluoride Levels in Drinking Water. Dental Council of India, New Delhi, 2004.
2. Crocombe LA, Mejia GC, Koster CR *et al.* Comparison of adult oral health in Australia, the USA, Germany and the UK. *Aus Dent J* 2009 **54:** 147-153.
3. Hugoson A, Koch G, Gothberg C *etal.* Oral health of indi­viduals aged 3-80 years in Jonkoping, Sweden during 30 years (1973-2003). *SwedDentJ* 2005 **29:** 139-155.
4. Zhang Q, Kreulen CM, Witter DJ *et aL* Oral health status and prosthodontic conditions of Chinese adults: a systematic review.

*Int J Prosthodont2001* 20: 567-572.

1. Adult Dental Health Survey: Oral Health in the United Kingdom 1998. Office for National Statistics.
2. Jones JA. Root caries: prevention and chemotherapy. *Am J Dent* 1995 8: 352-357.
3. Fure S, Zickertl. Prevalence of root surface caries in 55, 65, and

75-year-old Swedish individuals. *Community Dent Oral Epidemiol* 1990 18: 100-105.

1. Thomson WM. Dental caries experience in older people over time: what can the large cohort studies tell us? *Brit Dent J* 2004 **196:** 89-92.
2. Twetman S, Axelsson S, Dahlgren H *etal.* Caries-preventative effect of fluoride toothpaste: a systematic review. *Acta Odontol ScandlWft* **61:** 347-355.
3. Jensen ME, Kohout E The effect of aTluoridated dentifrice on root and coronal caries in an adult population. / *Am Dent Assoc* 1988 117:829-832.
4. Hunter ML, Addy M, Pickles MJ *etal.* The role of toothpastes and toothbrushes in the aetiology of tooth wear. *Int DenJ*2002 52:399-405.
5. Hooper S, West NX, Pickles MJ *etal.* Investigation of erosion

and abrasion on enamel and dentine: a model *in situ* using toothpastes of different abrasivity. / *Clin Periodontol2003* **30:** 802-808.

1. Barbour ME, Rees GD. The role of erosion, abrasion and at­trition in tooth wear. / C/in *Dent* 2006 17 (Spec Iss): 88-93.
2. Nunn JH. Prevalence and distribution of tooth wear. *In.* Addy M, Embery G, Edgar WAIe?«Z. (eds.) *Tooth Wear and Sensitivity.*

1st ed. pp93-103 Martin Dunitz Ltd: London, 2000.

1. King PA. Restorative management of the worn dentition. *Ini*

Addy M, Embery G, Edgar WM *etal.* (eds.) *Tooth linear and Sen­sitivity.}\*'* ed. pp201-216 Martin Dunitz Ltd: London, 2000.

1. Addy M. Tooth brushing, tooth wear and dentine hypersensitiv­ity - are they associated? *Int Dent J* 2005 **55:** 261-267.
2. Dugmore CR, Rock WP. The progression of tooth erosion in a cohort of adolescents of mixed ethnicity; *Int J Paediatr Dent* 2003 13:295-303.
3. NunnJH, Gordon PH, Morris AJ *etal.* Dental erosion - chang­ing prevalence? A review of British national childrens' surveys. *Int /Paediatr Dent* 2003 13:98-105.
4. #VanRijkom HM, Truin GJ, Frencken JEFM *etal.* Prevalence, distribution and background variables of smooth-bordered

tooth wear in teenagers in The Hague, the Netherlands. *Caries Res2002* **36:** 147-154.

1. Van't Spijker A, Rodriguez JM, Kreulen CM *et al.* Prevalence of tooth wear in adults. *Int J Prosthodont* 2009 22: 35-42.
2. Bartlett D, Dugmore C. Pathological or physiological erosion - is there a relationship to age? *Clin Oral Invest 2008* ***12:*** S27-S31.
3. Wood I, Jawad Z, Paisley C *et al.* Non-carious cervical tooth surface loss: a literature review./ *Dent2008* ***36:*** 759-76&
4. Lussi A, Schaffner M, Hotz P *etal.* Dental erosion in a popu­lation of Swiss adults. *Community Dent Oral Epidemiol* 199$ **19:** 286-290.
5. Lussi A, Schaffner M. Progression of and risk factors for dental erosion and wedge-shaped defects over a 6-year period. *Caries* R 2000 **34:** 182-187.
6. Marsh P, Martin MV. 2009. Oral Microbiology. Fifth edition. Churchill Livingstone Elsevier.
7. Lang NP, Schatzle MA, Loe H. Gingivitis as a risk factor in periodontal disease. *J Clin Periodontal*2009 **36:** 3-8.
8. Albandar JM, Kingman A. Gingival Recession, Gingival Bleed­

ing, and Dental Calculus in Adults 30 Years of Age and Older in the United States, 1988-1994. *J Periodontal* 1999 **70:** 30-43.

1. Robinson PJ. Periodontal therapy for the ageing mouth. *IntDent J* 1979 **29:** 220-225.
2. Fransson C, Berglundh T, Lindhe J. The effect of age on the development of gingivitis. Clinical, microbiological and histo­logical findings. *J Clin Periodontal* 1996 23: 379-385.
3. Tsalikis L, Parapanisiou E, Bata-Kyrkou A *et al.* Crevicular fluid levels of interleukin-1-alpha and interleukin 1-beta dur­ing experimental gingivitis in young and old adults. *J Int Acad Periodontal*2002 4: 5-11.
4. Anerud A, Loe H, Boysen H *etal.* The natural history of peri­odontal disease in man. 7 *Periodont*Rer 1979 14: 526-540.
5. Kinane DF, Attstrom R. Advances in the pathogenesis of peri­odontitis. Consensus report of the fifth European workshop in periodontology. *J Clin Periodontal*2005 32: 130-131
6. Albandar JM, Brunelle J A, Kingman A. destructive periodontal disease in adults 30 years of age and older in the United States, 1988-1994. *J Periodontal 1999 10:* 13-29.
7. Abdellatif HM, Burt BA. An epidemiological investigation into the relative importance of age and oral hygiene status as determinants of periodontitis./ *Dent Res* 1987 **66:** 13-18.
8. Niessen LC, Fedele DJ. Ageing successfully: oral health for the prime of *Xife. CompendContinEduc Dent 2002* **23** (lOSupp): 4-12.
9. Oral Health in America: A report of the Surgeon General. Department of Health and Human Service. 2000.
10. Joshipura KJ, Kent RK, DePaola PF. Gingival recession: intra­oral distribution and associated factors. *J Periodontal\99A* 65: 864-871.
11. Kassab MM, Cohen RE. The etiology and prevalence of gingival recession. *J Am Dent Assoc* 2003 134: 220-225.
12. ShklarG. The effects of aging upon oral mucosa. J *Invest Dermatol* 1966 47: 115-120.
13. van der Velden U. Effect of age on the periodontium. *J Clin Periodontol* 11:281 -294.
14. Karube Y, Oguchi H, Morito, M. Observation of the Micro­structure of the Gingiva in Elderly Persons. Abstract 3356.

International Association for Dental Research, Hawaii, 2004.

1. Vandana KL, Savitha B.Thickness of gingiva in association with

age, gender and dental arch location. *J Clin Periodontal*2005 32: 828-830.

1. Napier SS, Speight PM. Natural history of potentially malignant oral lesions and conditions: an overview of the literature./ *Oral PatholMed2001* **37:** 1-10.
2. Cau field PW, Dasanayake AP, IJ Y *et al.* Natural history of *Streptococcus sanguinis* in the oral cavity of infants: Evidence for a discrete window of infectivity. *InfectJmmun2000* **68:** 4018-4023.
3. Caufield PW, Cutter GR, Dasanayake AP. Initial acquisition of mutans streptococci by infants - evidence for a discrete window of infectivity. *J Dent Res* 1993 **72:** 37-45.
4. Karn Tr\, O'Sullivan DM, Tinanoff N. Colonisation of *Mutans Streptococci in 8-to* 15- month old children./ *Pub Health D ent* 1998 **58:** 248-249.
5. Stevens J Ef Oral ecology. *Technology Review.1991* 100:48-55.
6. Miyazaki H, Sakao S, Katoh Y *etal.* Correlation between volatile sulphur compounds and certain oral health measurements in

the general population./ *Periodontol* 1995 **66:** 679-684.

1. McDowell JD, Kassebaum DK. Treatment of oral and nonoral sources of halitosis in elderly patients. *Drugs and Aging* 1995 **6:** 397-408.
2. Narhi TO, Kurki N, Ainamo A. Saliva, Salivary microorganisms, and oral health in the home-dwelling old elderly - A five year longitudinal study. *J Dent lies* 1999 78: 1640-1646.
3. White DJ. Dental calculus: recent insights into occurrence, formation, prevention, removal and oral health effects of su­pragingival and subgingival deposits. *Eur J Oral Sci* **1997 105:** 508-522.
4. Watts A, Addy M. Tooth discolouration and staining: a rexdew of the literature, *Br Dent J2991* 190:309-316.
5. Joiner A. Tooth colour: a review of the literature. *J Dent* 2004 **32:** 3-12.
6. Joiner A, Hopkinson I, Deng Y *etal.* A rexdew of tooth colour and whiteness. *J Dent* 2008a **36S:** S2-S7.
7. Solheim T. Dental color as an indicator of age. *Gerodontics* 1988 **4:** 114-118.
8. Odioso LL, Gibb RD, Gerlach RW. Impact of demographic, behavioural, and dental care utilization parameters on tooth color and personal satisfaction. *CompendContin Ed Dent* 2000 21(suppl 29): S35-S41.
9. Jahangiri L, Reinhardt S B, Mehra R V, Matheson P B. Relation­ship between tooth shade value and skin color: an observational study. *JProstbet Den12002* 87: 149-152.
10. Goodkind RJ, Keenan K, Schwabacher W B. Use of a fiber­optic colorimeter for an *in* vivo color measurement of 2830 anterior teeth. J *Prostbet Dent* 1987 58: 535-542.
11. Zhao Y, Zhu J. *In vivo* color measurement of 410 maxillary anterior teeth *Chin J Dent* 7?erl998 3: 49-51.
12. Hasegawa A,Motonomi A, Ikeda I, Kawaguchi S. Color of natural tooth crown in Japanese people. Co/orRfj *App* 2000 25: 43-48.
13. Xiao J, Zhou XD, Zhu WC *et al.* The prevalence of tooth discolouration and the self-satisfaction xvith tooth colour in a Chinese urban population. *J Oral Rehab2007* **34:** 351-360.
14. Morley J. The esthetics of anterior tooth aging. *Curr OpirtCosmet Dent 1991* 4: 35-39.
15. Ten Bosch JJ, Coops JC. Tooth color and reflectance as related to light scattering an<jenamel hardness. J *DentP* 1995 74:374-380.
16. Joiner A, Pickles MJ,Matheson JR *etal.* Whitening toothpastes: effects on tooth stain and enamel. *Int Dent J* 2002 **52:** 424-430.
17. Matheson JR, Cox TF, Baylor N *etal* Effect of toothpaste xvith natural calcium carbonate/ perlite on extrinsic stain. *Int Dent J*

2004 **54:** 321-325.

1. Joiner A. The cleaning of teeth. 7»:Johansson I, Somasundaran P (eds.) *Handbook for Cleaning/Decontamination of Surfaces.* Vol. 1 pp371-405 Elsevier: Jxmdon, 2007.
2. Wulknitz, P. Cleaning power and abrasivity of European tooth­pastes. *Adv Dent* 1997 11: 576-579.
3. Addy M. Dentine hypersensitivity: Nexv perspectives on an old problem. *Int Dent j* 2002 52: 367-375.
4. Cummins D. Dentin hypersensitivity: from diagnosis to a break­through therapy for everyday sensitivity relief. *J Clin Dent* 2009 **20** (Spec Iss): 1-9.
5. Eaton KA, Carlile MJ. Too thbrushing behaxdour in Europe: Opportunities for dental public health. *Int Dent]* 2008 **58:** 287­293.
6. Bratthall D, Hansel-Petersson G, Sundberg H. Reasons for the caries decline: What do the experts believe? *Eur ] Oral Sci 1996* **104:** 416-422.
7. Lussi A, Hellwig E. Risk assessment and prex'entive measures. *Monogr Oral Sei* 2006 **20:1**90-199.
8. Fowler C, Wilson R, Rees GD. *In intro* microhardness studies on a new anti-erosion desensitizing toothpaste./ *Clin Dent*2006 17(Spec Iss): 100-105.
9. Rolla G, Saxegaard E. Critical evaluation of the composition and use of topical fluorides, xvith emphasis on the role of calcium fluoride in caries inhibition. *I Dent Res* 1990 **69 Spec No:** 780-5; discussion 820-823.
10. Arends J, ChristoffersenJ. Nature and role of loosely bound fluo­ride in dental caries. / *Dent Res* 1990 69(Spec Iss): 601—605.
11. Shaw L, Murray JJ, Burchell CK, BestJS. Calcium and phos­phorus content of plaque and saliva in relation to dental caries. *Caries Res 1983* 17:543-548.
12. Margolis HC, Moreno EC. Composition of pooled plaque fluid from caries-free and caries-positive individuals following sucrose exposure. *J Dent Res* 1992 **71:**1776-1784.
13. DaxrisRE, Marshall TA, Qian F *etal In vitro* protection against dental erosion afforded by commercially available, calcium- forti­fied 100 percent juices./Mw r *Dent Assoc* 2007 **138:** 1593-1596.
14. Hara AT, Zero DT. Analysis of the erosive potential of calcium- containing acidic beverages. *Eur J Oral Sci* 2008 116:60-65.
15. Duke SA, Rees DA, Forward GC. Increased plaque calcium and phosphorus concentrations using a calcium carbonate toothpaste containing calcium glycerophosphate and sodium monofluorophosphate. *Caries Res* 1979 13:57-59.
16. Lynch RJM, ten Cate JM. Effect of calcium glycerophosphate on demineralization in an *in vitro* biofilm model. *Caries* Rfj-2006 **40:** 142-147.
17. Duke SA. Effect of a chalk-based toothpaste on pH changes in dental plaque *in vivo. Caries R/r* 1986 **20:** 278-283.
18. Cury JA, Francisco SB, Simoes GS *et al.* Effect of calcium carbonate-based dentifrice on enamel demineralization *in situ. Caries Res2993* 37: 194-199.
19. Cury J A, Simoes GS, Del Bel Cury AA *etal.* Effect of a calcium carbonate-based dentifrice on *in situ* enamel remineralization. *Caries Res2995 39:* 255-257.
20. Joiner A, Schafer F, Hornby K *etal.* Enhanced enamel benefits fr6m a novel fluoride toothpaste. *Int Dent J* 2009 59: 244-253.
21. Hornby K, Evans M, Long, M *etal.* Enamel benefits of a new hydroxyapatite containing fluoride toothpaste. *Int Dent J* 2009 **59:** 325-331.
22. Chapple I. Teeth and gums are alixe and need nourishing. *Int Dent]* 2007 57: 117-118.
23. Brock GR, Butterworth CJ, Matthews JB *etal.* Local and sys­temic total antioxidant capacity in periodontitis and health, *j Clin Periodontoi2994* **31:** 515-521.
24. Landvick SV, Diplock AT, Packer L. Efficacy of vitamin E in human health and disease. In Cadenas E and Packer L (editors) *Handbook of antioxidants* Is\* ed Chapter 4 75-97. CRC: Taylor and Francis 2002.
25. Green AK, AlcockJ, Cox TF *etal.* Delivery of xtitaminE acetate and sunflower oil to gums from fluoride toothpaste containing 0.1% vitamin E acetate and 0.5% sunflower oil. *Int Dent J* 2007 57: 124-128.
26. Philpotts CJ, Harding CR, Carlile MJ *et aL Ex viw* delivery and penetration of a-tocopherol acetate and linoleic acid to gingival tis­sue from a toothpaste formulation. *Int Dent J* 2007 57: 129-134.
27. Brading MG, Marsh PD. The oral environment: the challenge for anti-microbials in oral care products. *Int DenJ* 2003 **53:** 353-362.
28. Nabi N, Mukerjee C, Schmid R *etal. In vitro and. in* vzvostudies on Triclosan/ PVM/ MA colpolymer/ NAF combination as an anti-plaque agent. *Am J Dent* 1989 2: 197-206.
29. Volpe AR, Petrone ME, De Vizio W *etal.* A rexdew of plaque, gingivitis, calculus and caries clincial efficacy studies with a fluo­ride dentifrice containing Triclosan and PVM/ MA copolymer.

*/ Clin Dent 1996* 7: S1-S14.

-107. Gaffar A, Afflitto J, Nabi N. Chemical agents for the control of plaque and gingivitis: an overxdew. *Eur J Oral Sci* **1997 105:** 502-507.

1. Williams C, McBride S, Mostler K *etal.* Efficacy of a dentifrice containing zinc citrate for the control of plaque and gingivitis: a

6-month clinical study in adults. *CompendContinEduc Dent* 1998 19: 4-15.

1. Sreenivasan PK, Furgang D, Markowitz K. *et al.* Clinical anti­microbial efficacy of anew zinc citrate dentifrice. *Clin Oral Invest* 2008 13: 195-202.
2. Raven SJ, Matheson JR, Huntington E *et al.* The efficacy of a combined Zinc and Triclosan system in the prevention of Oral Malodour. In: *Bad Breath; A multi-disciplinary approach,* van Steenberghe D and Rosenberg M (Eds). 1996. Leuven University Press, Belgium. Pp 241-254.
3. Navada R, Kumari H, Le S *etal* Oral malodor reduction from a zinc-containing toothpaste. J Clin Dent 2008 19: 69-73
4. Gilbert RJ, Ingram GS. The oral disposition of zinc following the use of an anticalculus toothpaste containing 0.5% zinc citrate. *JPharm Pharmacol\9fâ* 40: 399-402.
5. Segreto VA, Collins EM, DAgostino R *et al.* Anticalculus effect of a dentifrice containing 0.5% zinc citrate trihydrate. *Community Dent Oral Epidemiol* 1991 19:29-31.
6. Mandel ID. Calculus update: Prevalence, pathogenicity and prevention. *JADA* 1995 126:573-580.
7. Forward GC. Role of toothpastes in the cleaning of teeth. *Int Dent JVM*41:164-170.
8. Philpotts CJ, Weader E, Joiner A. The measurement in *vitro of* enamel and dentine wear by toothpastes of different abrasivity. *Int Dent J 2\*5* 55: 183-187.
9. Joiner A, Schwarz A, Philpotts CJ *etal.* The protective nature of pellicle towards toothpaste abrasion on enamel and dentine. JDe«z2008 36: 360-368.
10. Pickles MJ, Joiner A, Weader E *et al.* Abrasion of human enamel and dentine caused by toothpastes of differing abrasivity determined using an *in situ wear* model. *Int Dent J* 2005 55:1988-193.
11. Joiner A. Review of the extrinsic stain removal and enamel/ dentine abrasion by a calcium carbonate and perlite containing whitening toothpaste. *Int Dent J* 2006 56:175-180.
12. Addy M, Hunter ML. Can tooth brushing damage your health? *Int Dent J 2\*3* 53: 177-186.

Correspondence to: M Brading, Unilever Oral Care, Quarry Road East, Bebington, Wirral, CH63 3JW, UK.

Email: Melanie.Brading@Unilever.com