

Tooth brushing, tooth wear and dentine hypersensitivity – are they associated?

Martin Addy
Bristol, UK

Evidence suggests that patients suffer the painful symptoms of dentine hypersensitivity when dentine is exposed and the dentinal tubule system is opened to the oral cavity to allow stimuli to trigger a neural response in the pulp via a hydrodynamic mechanism. The processes needed to localise lesions of dentine hypersensitivity include loss of enamel and/or gingival recession. Whilst tooth brushing with or without toothpaste appears to cause minimal wear to enamel (in the absence of acids), circumstantial evidence implicates tooth brushing with gingival recession and exposure of dentine. Other tooth wear processes notably attrition and acid erosion cause loss of enamel and can expose dentine. Therefore sensitivity may result. How lesions of dentine hypersensitivity are initiated is a matter of conjecture and based on extrapolating data from studies, mainly *in vitro*, to affect *in vivo*. Again this circumstantial type of evidence suggests that abrasion by some toothpastes and erosion by dietary acid could open the tubule system. Little is known about the actual effect of desensitising toothpastes on lesions of dentine hypersensitivity even though they are formulated to either occlude dentinal tubules or block the neural response in the pulp. Clinical studies have produced contradictory findings for the efficacy of products and there have been extremely few evidence based reviews. In conclusion, available evidence supports a probable link of tooth brushing, with or without toothpaste and an acidic diet to both tooth wear and dentine hypersensitivity, and suggests also that dentine hypersensitivity is a tooth wear phenomenon. Although there is a need for more direct clinical and scientific evidence for these associations, it is recommended that they be taken into consideration when planning management strategies for the dentine hypersensitivity sufferer.

Key words: Dentine hypersensitivity, abrasion, acid erosion, attrition, toothbrushes, toothpaste, acidic foods, enamel, dentine, tooth wear

Many aspects of dentine hypersensitivity are poorly understood by dental professionals and in particular the aetiology of the condition. This has led to a great deal of conjecture on the subject of how best to treat, or more importantly manage the condition. Much confusion has been caused by conflicting views and opinions. Over recent decades much has been learned through research on dentine hypersensitivity and the former logical approach to the condition has been replaced by a biological strategy. Unfortunately, the next stage of an evidence based understanding of dentine hypersensitivity has not yet been achieved. Thus, many of the conclusions drawn on aspects of dentine hypersensitivity are based on studies *in vitro*, epidemiological

surveys, case reports and even anecdote. Data drawn from such sources provide evidence that may be more circumstantial than factual. One notable and important area of debate, even argument, is the role of tooth brushing and toothpaste in the aetiology, and therefore the treatment of dentine hypersensitivity. Indeed, there was for some time, and may be still, diametrically opposed views on whether dentine hypersensitivity was associated with good oral hygiene or poor oral hygiene. One circular argument was often cited that the pain of dentine hypersensitivity adversely affected oral hygiene practices and resulted in a build-up of plaque and the development of gingivitis, which in turn caused more gingival recession thereby worsening the sensitivity and

so the cycle was repeated. This paper will attempt to refute this supposition by reviewing available evidence, which taken overall appears to link tooth brushing with tooth wear and in turn dentine hypersensitivity. By way of debating this question of associations a brief overview of tooth brushing, tooth wear and dentine hypersensitivity will be given.

Tooth brushing

The oral health and cosmetic benefits of tooth brushing with toothpaste are many and well known to the profession although perhaps not to the general public¹⁻³. Tooth brushing *per se* probably only has the potential to achieve gingival and periodontal health through the mechanical removal of plaque. Even here, the toothbrush has limitations for the control of inter-dental plaque and therefore the prevention of periodontitis in the susceptible individual. Toothpaste has the potential to provide additional or adjunctive oral care benefits through chemical and physical means and the potential value of the most common ingredients are listed in *Table 1*. Thus, over recent decades, toothpaste formulations have been manipulated to deliver chemical and physical mediated benefits ranging from the prevention of caries and supra-gingival plaque and calculus, the removal of extrinsic stains and the treatment of dentine hypersensitivity^{1,3,4,7}. Indeed, there must be other avenues yet to be explored for the use of toothpaste as a delivery vehicle. Are, however, all of the outcomes of tooth brushing with toothpaste beneficial or is there the potential for harm? As with virtually all apparently beneficial therapeutic and preventive regimens in medicine and dentistry a balance often has to be struck between positive effects and side effects. Moreover, possible interactions of treatment or preventive regimens with other ongoing regimens or processes may tip the scales toward side effects. Such considerations apply just as much to tooth brushing with toothpaste as they do to chemotherapy for malignancy except the risk benefit ratio is several orders of magnitude different for the individual recipient. The main issue for the potential for harm from toothpaste relates to the abrasivity of products as a cause for tooth wear and/or hypersensitivity arising from exposed dentine.

Toothwear – dental tribology

Prevalence figures suggest that tooth wear must be the fourth dimension risk factor for the aesthetics, function and longevity of the human dentition behind acute trauma, caries and periodontal disease⁸⁻¹⁰. Tooth wear is a composite term introduced to cover non-carious tooth surface loss by attrition, abrasion and erosion^{11,12}. Perhaps the current terminology should be updated to 'Dental Tribology' to bring the dental profession in line with the scientific body of knowledge of friction, lubrication and wear (tribology). This would seem worthy of consideration since individual wear processes, attrition, abrasion and erosion are peculiar to dentistry and, in tribology, have little or no meaning and in the case of erosion are descriptively incorrect. Thus, in tribology, attrition is two-body wear, abrasion three-body wear and erosion chemico-physical wear^{13,14}. Whatever, it is generally agreed that tooth wear can rarely, if ever, be attributed to a single process but occurs as a result of combinations of the processes even though one may be dominant. A fourth factor may contribute to tooth wear, namely abfraction, by increasing the susceptibility of cervical hard tissues to abrasion and erosion¹⁵. Abfraction is a theoretical concept with evidence drawn from modelling using finite element analysis. Such evidence in turn appears consistent with the wedge shaped wear lesions seen at the buccal cervical area of certain teeth. Essentially, it has been proposed that high tensile stresses due to cuspal flexure on lateral excursions are caused at these sites and lead to microcracks in the enamel and possibly the dentine¹⁵. In everyone some degree of tooth wear occurs within a lifetime but, in a proportion of individuals, the wear reaches pathological levels^{16,17}.

Dentine hypersensitivity

Dentine hypersensitivity is the term used to describe the common, painful condition of the permanent dentition. Largely based on a suggestion in a 1983 publication¹⁸, the term was defined in 1997 after an international conference on the design and conduct of clinical trials for the treatment of the condition¹⁹. The definition states

Table 1 Function of common toothpaste ingredients

Ingredient	Function
Abrasive	Stain removal, polishing
Detergent (Surfactant)*	Foaming, stability, solubiliser, anti-microbial, plaque inhibitory, mouth feel
Binder (Thickener)*	Stability, consistency, appearance
Humectant*	Maintain moisture, Flowability
Flavour*	Taste, Feel, Freshness
Colour*	Appearance
Fluoride	Remineralisation, Caries prevention

*Probably contribute to the consumer expectations for a toothpaste product and indirectly improve compliance with the professional recommendation to tooth brush regularly.

that: “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 defect or pathology¹⁹ (disease²⁰)”. The definition usefully gives a clinical description of the condition and suggests the need to exclude other causes of “dentinal” pain. As with the tooth wear processes, the terminology can be questioned as to accuracy. Common usage over decades and now the agreed definition, however, suggest that the term dentine hypersensitivity be universally adopted and the use of variant terminology be discouraged. In this respect, recently, the suggestion has been made to term the sensitivity associated with periodontal disease and treatment, root sensitivity²¹, since it may have a different aetiology associated with bacterial penetration of the dentinal tubules²², and certainly it does not fit the definition of dentine hypersensitivity. The hydro-dynamic mechanism^{23,24} propounded to explain how appropriate stimuli trigger the painful response in the pulp gives insight into how lesions of dentine hypersensitivity develop. Essentially, dentine has to be exposed and the dentine tubule network opened to permit fluid movement under stimulation⁵. This has led some authors to suggest dentine hypersensitivity is a tooth wear phenomenon whilst acknowledging that much remains unknown or unproven about the aetiology of the condition²⁵.

Thus far, a brief description of the salient features of tooth brushing, tooth wear and dentine hypersensitivity have been given. Already, at a conceptual level, it would not be a giant biological step to consider that the three may be linked. The aim of this paper will be to consider the available evidence as to whether there is indeed a link. From the outset, it is important again to emphasise that much of the evidence of a link is circumstantial and not evidence based. Nevertheless, there are numerous publications relevant to the various topics to be discussed and for the sake of brevity when possible reviews will be cited in support of statements made.

Does tooth brushing cause tooth wear?

Tooth brushing with toothpaste has been stated as the most common oral hygiene habit practised by persons living in developed countries²⁶. Today’s toothpastes and toothbrushes in the 6,000-year history of ‘oral hygiene products’ are relatively recent introductions and date back to the early 20th century²⁷. Previous toothpastes/toothpowders could variably be described as revolting, highly abrasive, erosive and even potentially toxic^{7,27}.

Modern toothbrushes and toothpaste formulations have in place, or in development, national and, more importantly, international standards, which primarily relate to potential safety issues. Most relevant here is the abrasivity of toothpastes²⁸. By definition, toothpastes contain abrasive agents, the role of which is to

remove stains and other superficial deposits from the tooth surface. Different formulations contain different abrasive agents, some being more abrasive than others. Relative Dentine Abrasivity (RDA) is a numeric scale, which indicates the degree of abrasivity, and is useful for comparison between different pastes. A higher RDA value indicates a greater abrasive formula. The allowed pH range for toothpastes (pH 4-10) could be a cause for concern for tooth wear due to acid erosion but virtually all products world-wide are above a pH which might cause demineralisation (pH 5.5 for enamel, pH 6.5 for dentine), or the contained fluoride balances the low pH effect. Several important conclusions from available data concerning the abrasion of hard tissues by toothbrushes alone and with toothpaste can be found in recent reviews and are as follows^{7,29}.

In normal use:

- Toothbrushes alone produce essentially no wear of enamel
- Toothbrushes alone over extended periods of use, measured in years, cause minute amounts of dentine wear, which may be restricted to the smear layer³⁰. The smear layer is an artificial surface structure that is formed when dentine is abraded or cut. The layer is about one micron thick and made up of collagen and hydroxyapatite from the native dentine. The smear layer covers the underlying dentine and occludes the tubules (*Figures 1 and 2*). Tooth brushing with toothpaste in the absence of acid causes little or no wear of enamel because, with the exception of the rarely used non-hydrated alumina, contained abrasives are softer than enamel.
- Tooth brushing with toothpastes does abrade dentine and, from a study *in situ*, the rate of wear shows a reasonable correlation to toothpastes’ RDA values³¹. The cumulative abrasion suggests a loss of 1mm of dentine in 80 – 100 years of tooth brushing²⁹.

In abnormal or abusive use:

- Tooth brushing with toothpaste will still have little or no effect on enamel, but abrasion of dentine can reach pathological proportions²⁹.

It must be emphasised that these conclusions are at best drawn from studies *in vitro*, there are a few *in situ*, and at worst from case or anecdotal reports. Unfortunately, studies *in vivo* that investigate toothpaste abrasion alone would be difficult, perhaps impossible, to design.

The overall conclusion therefore must be that if toothpaste abrasion were the only wear process ongoing in the mouth, in normal use, it would have no clinical significance, except potentially to open dentinal tubules and this will be discussed later. Abrasion by toothpaste, however, is not the only wear process and teeth are exposed to wear by attrition and erosion. It is unlikely

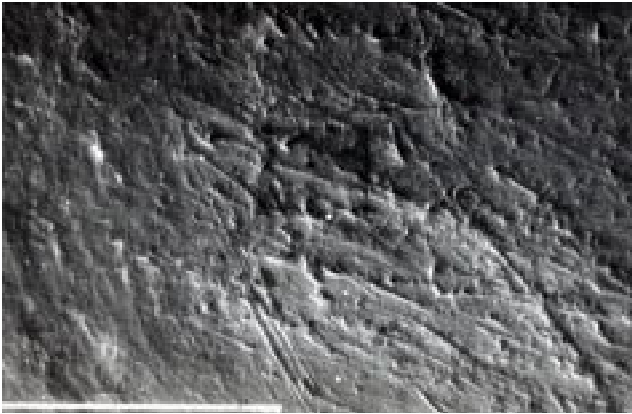


Figure 1. Scanning electron micrograph image of a root surface covered by a smear layer. Note that there are no open tubules apparent.

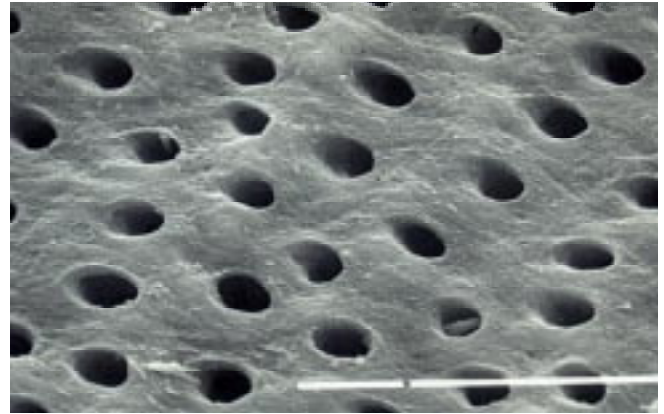


Figure 2. Scanning electron micrograph image of a dentine surface with no smear layer. Note presence of open dentine tubules.

that attrition and tooth brushing with toothpaste would co-operate to cause tooth wear except where attrition has exposed dentine, which is subsequently brushed. Abrasion, on the other hand, does have the potential to enhance tooth wear due to acid erosion.

The potential for erosion in the peoples of developed nations is high because of the levels of acidic food and drink consumption^{9,10,32,33}. Also, in some individuals, frequent contact with the teeth by intrinsic gastric acid is an additional erosive problem^{16,33}. Consumption and sales reports of acidic foods and beverages, epidemiology surveys, studies *in vitro* and *in situ* and review publications all provide strong evidence that acid erosion is a dominant factor in the tooth wear prevalence figures^{10,33}. Moreover, studies *in vitro* and *in situ/ex vivo* indicate that abrasion can co-operate with erosion to increase wear^{34,35}. Indeed, the available data indicate that abrasion and erosion act at least additively, if not synergistically, in the tooth wear process. Relative to the present debate the majority of studies have considered, perhaps rightly so, tooth brushing alone or with toothpaste as the major adjunctive wear process to erosion: there being some studies on attrition and erosion and simulated abrasion and erosion by fibrous acid foods³⁶⁻³⁸. For both enamel and dentine the marked increase in susceptibility to wear by tooth brushing following prior exposure to acids can be explained by the softening process that parallels the actual bulk loss of hard tissue³⁹⁻⁴¹. The softened zone, which can reach depths of several microns in both enamel and dentine, appears highly vulnerable to wear from relatively minor physical insults, including the action of the tongue^{38,42}. This clearly raises the issue of the timing of tooth brushing relative to meals or snacks. The preventive rather than therapeutic action of toothpaste must lead to the logical, if not biological suggestion for brushing before meals. Certainly, after meals brushing should be delayed probably for several hours to allow remineralisation.

In concluding this section, the available evidence implicates tooth brushing with tooth wear but outside abusive tooth cleaning such wear will only reach pathological proportions when combined with the more dominant wear process, acid erosion.

Is tooth brushing involved in dentine hypersensitivity?

For dentine hypersensitivity to occur, dentine has to be exposed (lesion localisation) and the tubule system exposed (lesion initiation)⁵. Evidence from extracted teeth indicates that this is indeed the case^{43,44}. The question relevant to this debate is whether tooth brushing alone or with toothpaste is involved in either or both lesion localisation or initiation. To expose dentine either enamel has to be lost or gingival recession has to occur. Evidence so far discussed suggests that tooth brushing with toothpaste alone would be unlikely to remove sufficient enamel even at the cemento-enamel junction. Reviews have concluded that tooth brushing is implicated in gingival recession, but the available evidence is circumstantial rather than factual^{45,46}. Stronger evidence is available to implicate other causes for gingival recession e.g. periodontal diseases and surgery (as their effects are more measurable). In respect of the present debate it is the possible role of tooth brushing in what might be termed 'healthy recession' that needs to be discussed: accepting, of course, that tooth brushing may be co-destructive to the other individual causes for recession. Such data, drawn from epidemiological and clinical studies and case reports, associate tooth sites of high predilection for recession with the greatest exposure to tooth cleaning and the lowest scores for plaque⁴⁷⁻⁵⁰. If tooth brushing is implicated in recession, the mechanism for such has only been hypothesised upon and therefore the importance of factors relating to the individual, the tooth brushing process, the tooth brush and the tooth-

paste can only be conjecture. Many logical suggestions for the above have been made but so frequently logical and biological thought processes are contradictory. Not surprising, and like dentine hypersensitivity, gingival recession was described as an enigma⁴⁶.

Whether tooth brushing alone or with toothpaste could initiate dentine hypersensitivity can be based on some scientific evidence albeit derived largely from studies *in vitro* and, to a limited degree, *in situ*. A tooth brush alone, as already stated, has little effect on dentine and removal of the smear layer to open tubules takes the equivalent of several years of tooth brushing of any one tooth³⁰.

Toothpaste changes the story significantly but variably dependent on the formulation. Many toothpastes appear to readily remove the smear layer from dentine to expose tubules and over a relatively small number of equivalent tooth brushing days^{51,52}. Studies *in vitro* suggest that this effect is a function of both the abrasive and the detergent systems contained in the product⁵¹. This discussion could be said to indicate that all toothpastes are aetiological factors in dentine hypersensitivity but this is not necessarily the case. Some formulations, although removing the smear layer, then cause narrowing of dentinal tubules presumably by an abrasive smearing process⁵². More interestingly, some toothpastes remove the smear layer and then occlude the tubules with the contained abrasive particles⁵¹⁻⁵³. Most effective in this process appear to be those products that contain artificial silica with a non-ionic detergent: the more common use of sodium lauryl sulphate, an anionic detergent, appears to prevent the attachment of artificial silica to dentine probably by ionic competition. Thus non-ionic detergent systems, such as tego betaine, found in some toothpaste formulations may provide some benefit in conjunction with artificial silicas.

Finally, in discussing tooth brushing and dentine hypersensitivity, one cannot avoid debating the common use of desensitising toothpastes to treat the condition. Although numerous agents have been used in toothpastes to treat dentine hypersensitivity over the years, many of the studies purporting to prove efficacy can be questioned for scientific merit^{53,54}. More recently, studies have tended to conform to the requirements of the classical randomised controlled clinical trial and the recommendations for the design and conduct of clinical trials on dentine hypersensitivity¹⁹. In particular, the studies have focused on products, which might be expected on theoretical or laboratory grounds to occlude tubules or block neural transmission at the pulp. Whether these effects actually occur has still to be proven *in vivo* yet some studies, do show a difference in clinical trials in favour of the active versus the control⁵⁵. In conclusion, there does appear to be an association of tooth brushing with toothpaste both in the localisation, initiation and treatment of lesions of dentine hypersensitivity

Is dentine hypersensitivity a tooth wear phenomenon?

The previous section concluded that both positive and negative associations existed between tooth brushing and dentine hypersensitivity. As tooth brushing was also considered from the first section to be associated with tooth wear this final section considers evidence for attrition and erosion to be associated with dentine hypersensitivity alone or combined with tooth brushing derived abrasion. There is strong evidence, particularly from case reports, that attrition causes tooth wear and, in parafunctional habits, can become pathological as dentine becomes exposed¹⁶.

Interestingly, at least for enamel, one study *in vitro* demonstrated that erosion, combined with attrition, considerably slowed down tooth wear³⁶. An explanation propounded by the authors was that the contacting enamel surface became very rough under neutral pH conditions but very smooth under erosive pH conditions: frictional forces therefore would be markedly reduced. Clearly, if dentine becomes exposed by attrition alone or when combined with erosion, dentine hypersensitivity could occur. Again, however, this would require the wear to open the tubule system. As will be discussed, if erosion were involved, this opening of tubules would almost certainly occur and be consistent with the relatively infrequent presentation of individuals with dentine hypersensitivity at occlusal surfaces who report both grinding habits and high fruit intake^{9,33}. Alone it is not reported how attrition affects exposed dentine: one might suspect that a smear layer would be formed.

At the most common site for dentine hypersensitivity, buccal-cervically, evidence, largely from studies *in vitro* and *in situ*, suggests acid erosion has the potential to both localise and initiate lesions⁵. Thus, studies *in situ* have shown that, in some individuals, imbibing one litre of soft drink per day, not uncommon in many countries, could remove one millimetre of enamel within a period of a few years⁵⁶. At the buccal cervical region of teeth this would account for more than the enamel thickness at this site. Additionally, as already alluded to, the rate of enamel loss would be accelerated by regular tooth brushing^{34,35}. Once exposed, laboratory and *in situ* research demonstrates that acidic beverages can remove the dentine smear layer to expose tubules after the clinical equivalent of a small number of acid beverage doses³⁰. Moreover, some treatments for dentine hypersensitivity which occlude tubules such as oxalates and calcium phosphates appear acid labile⁵² and may easily be removed. Tooth brushing, even without paste, appears to readily remove a dentine smear layer, which has been exposed to even the briefest of acid erosive insults³⁰. Taken together, these data must lead one to conclude that tooth wear processes are at least associ-

ated with dentine hypersensitivity even though such a conclusion is not evidence based in the conventional meaning of the phrase.

Conclusion

The question of 'associations between tooth brushing, tooth wear and dentine hypersensitivity' was posed in the manuscript title. The nature of the majority of the available data used in this review does not allow a more absolute statement concerning association between the three processes to be made other than probably. There does, however, appear to be compelling evidence that tooth brushing and tooth wear are aetiological factors in the localisation and initiation of dentine hypersensitivity. As a result, these processes need to be taken into account when formulating a management strategy for dentine hypersensitivity.

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Correspondence to: Professor M. Addy, Bristol Dental School, Lower Maudlin Street, Bristol, BS1 2LY, UK.
Email: Martin.Addy@Bristol.ac.uk