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REVIEW

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Personal oral hygiene and dental caries: A systematic review of randomised controlled trials

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Objective: To conduct a systematic review of randomised trials assessing the association between personal oral hygiene and dental caries in the absence of the confounding effects of fluoride.

Background: Dental caries continues to affect close to 100% of the global population. There is a century-old conflict on whether dental caries is caused by poor oral hygiene or poorly formed teeth (ie, teeth with dental defects). Resolving this conflict is of significant public health importance as these two hypotheses on dental caries aetiology can lead to different prevention strategies.

Methods: A systematic search for randomised trials was conducted using predefined criteria in 3 databases. The impact of personal oral hygiene interventions on coronal dental caries incidence was evaluated using random-effects models.

Results: Three randomised studies involving a total of 743 participants were included. Personal oral hygiene interventions failed to influence the incidence of dental caries (Δ Decayed, Missing and Filled Surfaces (DFMS) = −0.11; 95% confidence interval: (−0.91, 0.69; *P*-value < .79)) despite meticulous deplaquing of teeth. There was no significant heterogeneity in the trial results (heterogeneity chi-squared = 1.88, *P* = .39). The findings were robust to sensitivity analyses, including consideration of the results of nonrandomised studies.

Conclusion: Personal oral hygiene in the absence of fluorides has failed to show a benefit in terms of reducing the incidence of dental caries.

KEYWORDS
dental caries, epidemiology, oral hygiene, systematic reviews

1 | INTRODUCTION

Dental caries remains the most prevalent pathological condition worldwide, with minimal change in prevalence and incidence over the last two decades.[1] The evidence is mounting that dental caries is a growing concern in the ageing populations.[2] There are two major hypotheses on how this pandemic can be brought under control: one is through implementing improved personal oral hygiene and the other through addressing the defects in the coronal enamel-structure. These two conflicting hypotheses can be schematically presented within the context of a causal framework and are now briefly described (Figure 1).

The oral hygiene hypothesis postulates that biofilms on tooth surfaces will produce acids in the presence of dietary carbohydrates. These acids demineralise the enamel and eventually allow cariogenic bacteria to invade the dentin and the pulp. Under this hypothesis, mechanically removing the biofilm from tooth surfaces with toothbrushes or interproximal cleaning devices is believed to prevent dental caries. Historically, the motto of the proponents for the oral hygiene hypothesis was that "clean teeth do not decay."[3]

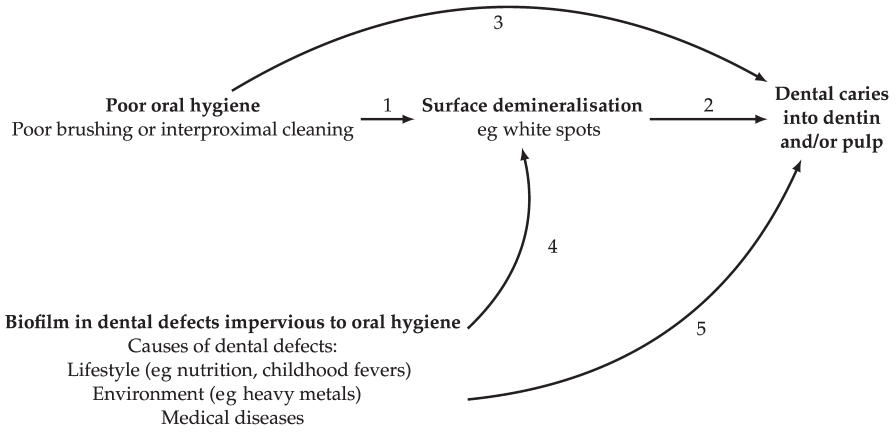

FIGURE 1 Mediation framework for evaluating the aetiology of dental caries. Arrows 1-3 illustrate the "oral hygiene" or the "clean tooth" hypothesis of dental caries. Arrows 4 and 5 illustrate the dental defect or "sound tooth" hypothesis of dental caries. Both hypotheses were widely debated in the early 20th century

The dental defect hypothesis posits that dental caries starts in microscopic cracks or crevices in teeth, and not on defect-free or sound enamel. The biofilm within the dental defects is also thought to become cariogenic in the presence of dietary carbohydrates. However, the biofilm cannot be removed with a toothbrush or interproximal cleaning devices; hence, oral hygiene is believed to be ineffective. Under this alternative hypothesis, prevention of dental caries must focus on preventing the formation of dental defects during odontogenesis, by repairing the defects from the pulpal side, or by sealing or surgically eliminating surface defects in the enamel. Historically, the motto of the proponents for the dental defect hypothesis was that "sound teeth do not decay".[4]

A key step in resolving the conflict between these two hypotheses is to determine the value of personal oral hygiene without the confounding effects of fluoride, the latter having strong evidence in support of its anticariogeneic effect.[5] The aim of this study was to report on a systematic review of randomised trials assessing the association between personal oral hygiene and coronal dental caries.

2 | METHODS

2.1 | Search strategy

The systematic search was performed following the guidelines set out by the Preferred Reporting Items for Systematic Reviews and Meta-analysis (PRISMA).[6] Three databases (PubMed, Web of Science and the Cochrane Central Register of Controlled Trials) were searched for articles published between January 1950 and February 2017. The protocol was neither established a priori nor registered nor were there date restrictions on the manual searches. The search was conducted with the following search strategy: ("oral hygiene"[MeSH Terms] OR ("oral"[All Fields] AND "hygiene"[All Fields]) OR "oral hygiene"[All Fields]) AND ("dental caries"[MeSH Terms] OR ("dental"[All Fields] AND "caries"[All Fields]) OR "dental caries"[All Fields]) AND Clinical Trial[ptyp]." Titles, abstracts, full-text papers and grant reports were screened for additional references. A two-phase strategy was employed for the search that in a first phase included screening of titles and abstracts of the articles identified as potentially relevant during the electronic search, followed in a second phase by an assessment of the full-text of articles deemed eligible for inclusion (eg, a systematic review[7]). Reports in languages other than English were excluded. Personal oral hygiene was defined as brushing of teeth with or without interproximal cleansing devices.

2.2 | Inclusion criteria

Controlled clinical trials (CCTs) of personal oral hygiene interventions were included if the primary aim was to assess the impact of toothbrushes with or without interproximal cleaning devices on dental caries, if the trial design mentioned the word "random" and was prospective, if dental caries scores (as a count) were reported at baseline and at the end of the follow-up, and if there was a concurrent control group. There were no restrictions on method of treatment assignment, participant characteristics or on primary vs permanent teeth. Nonrandomised studies which met all other inclusion criteria were accepted to assess the robustness of the primary analysis.

2.3 | Exclusion criteria

Trials were excluded in which the effect of personal oral hygiene interventions was combined with fluoride products or dietary interventions and the control group had no such interventions. Trials were excluded also if they focused on dental caries surrogates (eg, bacterial counts), professional prophylaxis or where oral hygiene assignment was based on patient behaviour. Trials with a primary aim of assessing chemotherapeutics (e.g., fluoride, chlorhexidine) were excluded. Retrospective studies, cross-sectional studies and letters to the editors were not considered.

2.4 | Data extraction

Sample size, effect sizes and available measures of variability were abstracted independently by two readers (PH, MH). For each CCT, information was collected on the nature of the oral hygiene intervention, on the unit of random assignment (patients, classes, schools), on the blinding of the examiners and on the characteristics of the design and analysis.

2.5 | Quality assessment

The CCT quality was quantified using a modified Jadad scale[8] and risk of bias measures for randomised controlled trials.[9] The Jadad score and our modification of it is a score between 0 and 5 where 0 indicates poor quality and 5 highly rigorous quality. The Jadad modification consisted of changing the word "double-blind" to "single-blind" as it was considered impossible to blind trial participants towards self-performed tasks such as brushing and flossing (Table 1). Baseline caries comparability was abstracted prior to drop-out to the extent possible. A CCT was labelled commercially funded if it reported receiving a grant from an oral hygiene company for the conduct of the study.

2.6 | Data synthesis

Summary DMFS estimates were based on random-effects models. The heterogeneity of the studies was evaluated using the heterogeneity chi-squared statistic. Effective sample sizes for the trials were calculated assuming an intracluster coefficient (ρ) of 0.02.[10] This effective sample size was calculated as the total sample size divided by the design effect ($1+\rho$ (m-1)) where m is the average class or school size when the cluster size was unavailable, otherwise m was calculated as the sum of the cluster sizes squared divided by the number of participants in that group when cluster sizes were reported.[11] For studies which assigned classes to treatments, the number of classes in public schools was calculated assuming each class had on average about 20 pupils. Radiographic caries increment scores combined with clinical scores were selected over clinical caries increments alone when both outcomes were available.[12]

Exploratory analyses were conducted to assess the robustness of the findings. Included in these secondary analyses was an assessment of the impact of nonrandomised studies. These nonrandomised studies included different dental outcome scores (such as Decayed, Missing or Filled Teeth or DMFT) for caries and were standardised using the Glass's Δ method.[13] Highly significant heterogeneity was taken as an indication to analyse the data using a random-effects model, not fixed-effects models.[14,15] All analyses were completed using SAS 9.4 (including the forest and the metaanal macros), STATA 11.1 meta-analysis software and R 3.3.2 (meta).

3 | RESULTS

3.1 | Selection of controlled clinical trials

A total of 984 unique citations from 3 sources were identified (Figure 2). Thirteen references of interest were identified for full-text review, which included 12 published articles and one abstract for which we obtained the NIH grant report.[12,16-21] After full-text review, three randomised trials were included (Figure 2). Four nonrandomised trials were retained for the purpose of sensitivity analyses.

TABLE 1 The design, interventions and outcome quality of included and excluded trials used to measure the effectiveness of personal oral hygiene in controlled dental caries

Trials	Design					Intervention
References	Reporting of commercial funding	Experimental unit of analysis	Random sequence generation	Allocation concealment	Blinding of clinical outcome assessment	Supervised oral hygiene
Silverstein et al[16]	No	Student	?	?	+	Daily[2]
Horowitz et al[18]	No	Student	?	?	+	Daily[2]
Ashley & Sainsbury[12]	No	Class	?	?	+	2 wk[2]
Fosdick[22]	Yes	NR	−	−	−	No
Clark et al[21]	No	School	−	−	+	Varied
Spears et al[19]	No	Class	−	−	−	Daily[2]
McKee et al[17]	No	School	−	−	+	Daily[1]

Cochrane risk of bias was judged to be + low risk of bias, − high risk of bias or ? unclear risk of bias. NR = Not reported 1. supervision only during first year of a 3-year study. 2. On schooldays

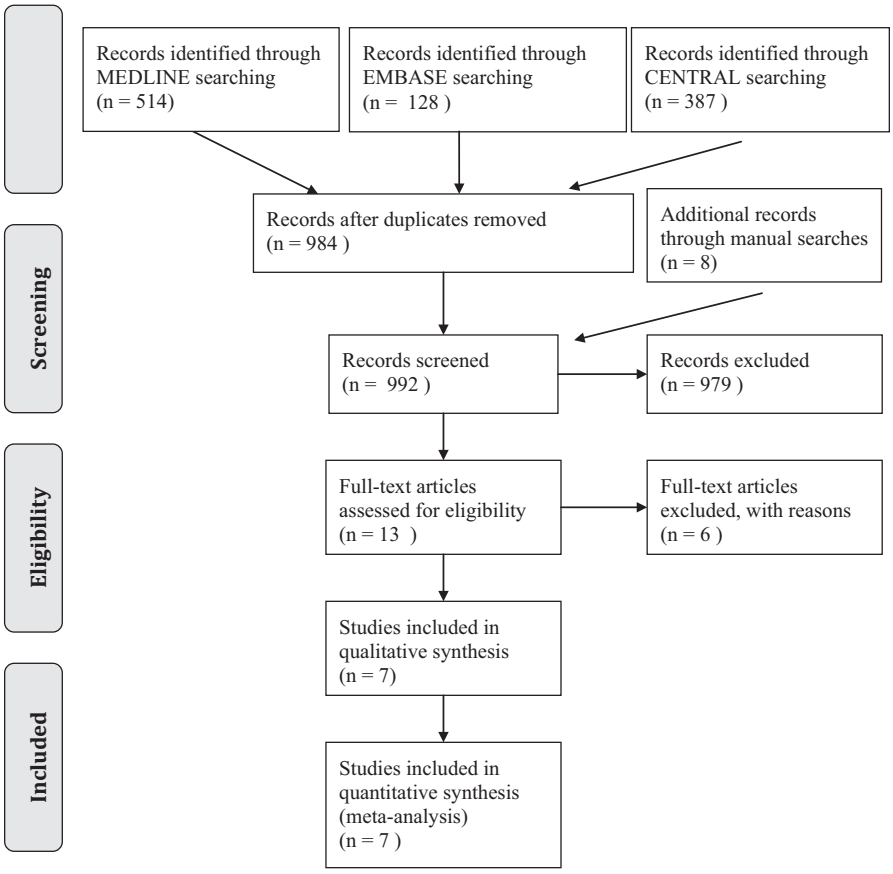

FIGURE 2 Flow chart of literature searches and identified articles

3.2 | Characteristics of randomised controlled trials assessing dental plaque removal

Three randomised trials on 743 participants were identified (Table 1); two trials were conducted in the United States (US), and one in the United Kingdom (UK).[12] The enrolled participants were girls aged 11 and 12,[12] and boys and girls aged 10 and 13,[18] and 12 and 13.[16] All three trials reported DMFS scores; two trials continued for 3 years[12,18] and one trial for 29 months.[16] The units of randomisation were participants[16,18] and classes.[12] The two US trials were

				Outcome			
Supervised Flossing	Disclosing tablets	Water fluoridation	Drop-out rate	Incomplete outcome assessment	*P*-value for Baseline difference	Jadad Score	Selective reporting
Yes	Yes	No	37%	−	.83	4	+
Yes	Yes	No	39%	−	.60	4	+
Yes	Yes	NR	15%	+	.97	4	+
No	No	NR	NR	−	NR	0	+
NR	No	NR	NR	−	.16	2	−
Yes	Yes	Low	48%	−	NR	0	+
Yes	Yes	No	71%	−	.49	3	+

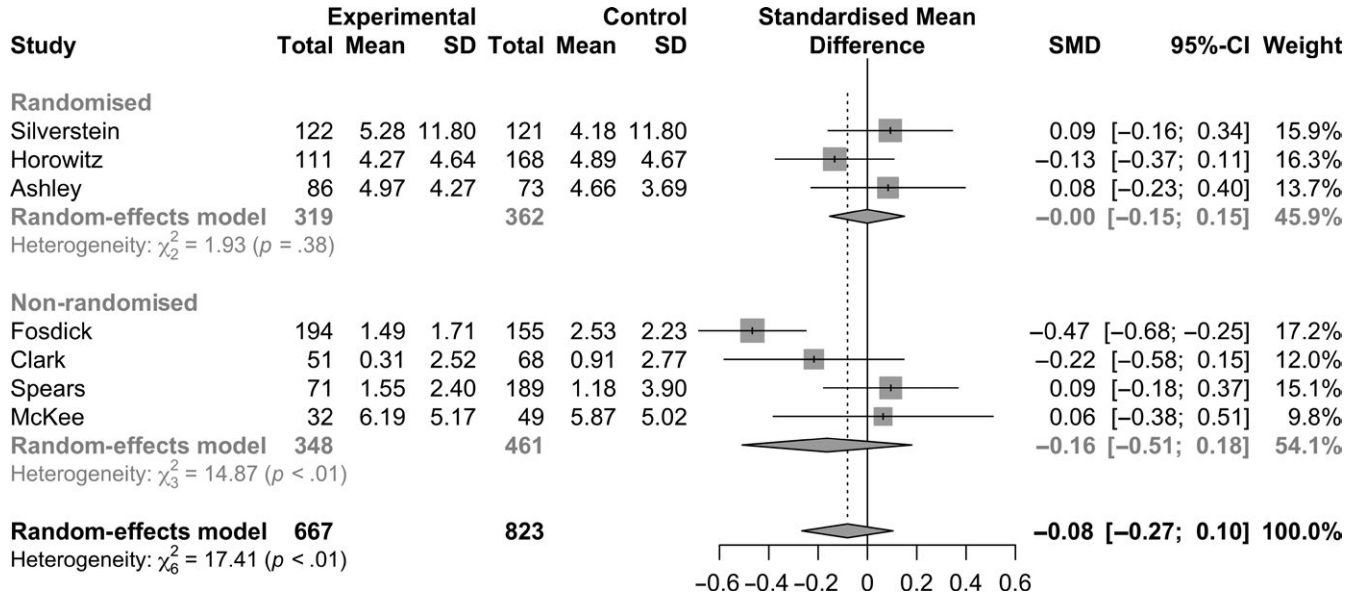

FIGURE 3 Forest plot displaying the impact of personal oral hygiene effect without fluoride on the *standardised* mean difference (SMD) for coronal dental caries scores for both randomised and nonrandomised trials. Sample sizes displayed are effective sample sizes which differs from actual sample sizes in cluster trials. Random-effects models lead to the consistent conclusion that personal oral hygiene did not influence the incidence of dental caries. Inclusion of the only commercially funded study created statistically significant heterogeneity, which, if ignored in the analysis by choosing a fixed-effect model, led to an overall benefit of personal oral hygiene. *Note:* 1. Silverstein: Caries outcomes in 2 schools summarised using random-effects model. 2. Horowitz: No assumptions. 3. Ashley: Assuming 6 classes in the oral hygiene intervention group, five in the control groups, and ρ = 0.02. 4. Fosdick: Assuming each of the 7 schools had one oral hygiene intervention group, one control group, and ρ = 0.02. The standard deviation for the control group was obtained using the reported standard deviation for the treatment group, assuming pooled variance and the relationship between the *F* statistic and the *T* statistic ($F = t^2$). 5. Clark: ρ = 0.02. 6. Spears: Assuming 5 classes in the oral hygiene intervention group, 13 classes in the control group, ρ = 0.02. 7. McKee: Standard deviation in both groups estimated as ln(standard deviation) = 0.64 + 0.55*ln(increment) and ρ = 0.02. Estimate of baseline DMFS score included participants whom dropped out

conducted in nonfluoridated communities. The level of water fluoridation was not reported for the UK trial. There were no significant differences in the baseline score for caries between the intervention and control groups. The Jadad score was "4" for all 3 trials. None were funded by commercial companies. Drop-out rates ranged between 15% and 39% (Table 1). Details on standard deviation calculations are presented as a footnote in Figure 3.

3.3 | Synthesis of results

There was no heterogeneity between trials (Heterogeneity chi-squared = 1.88 (degrees of freedom (*df*) = 2) *P* = .390), and a random-effects model showed that the oral hygiene interventions did not influence the incidence of dental caries [Decayed, Missing or Filled Surfaces (DMFS) = −0.11; 95% confidence interval (CI) (−0.91, 0.69)]. These results remained unchanged when restricting the analyses to two trials (n = 522) with significant reductions in gingivitis [DMFS = −0.34; 95% CI (−1.59, 0.91), *P*-value = .60].

3.4 | Risk of bias across controlled clinical trials

Meta-regression or statistical assessment of publication bias was not performed due to the limited number of trials and minimal variability in terms of duration or quality of the randomised trials.

3.5 | Sensitivity analyses

One of the three randomised trials used clustered randomisation, which we accounted for in our main analysis. Results did not change appreciably when the clustered randomisation of this trial was ignored [DMFS: −0.05; 95% CI (−0.80, 0.70), *P*-value < .90, heterogeneity chi-squared = 2.03 (*df* = 2) *P* = .362].[12,16]

Four nonrandomised trials with a mean Jadad score of 1.5 were identified (Figure 3). Two trials reported an increased risk of caries and two trials a decreased caries risk. One trial funded by commercial sponsors had a Jadad score of 0 and found a highly significant beneficial impact of oral hygiene on dental caries. This trial produced significant heterogeneity both within the nonrandomised trials (heterogeneity chi-square = 14.87, 3 *df*, *P*-value < .01) and within all 7 combined (heterogeneity chi-square = 17.41, 6 *df*, *P*-value < .01). Randomised and nonrandomised trials led to almost identical results when this outlier was removed from the analysis [standardised mean difference (SMD = 0.00; 95% CI (−0.15, 0.15), *P*-value .99, vs SMD = 0.00; 95% CI (−0.20, 0.20), *P*-value .99, respectively].

When the industry-funded study was included,[22] a random-effects model showed no significant benefit of personal oral hygiene in terms of reducing dental caries ([SMD = −0.08; 95% CI (−0.27, 0.10), *P*-value = .39]. This conclusion was robust towards the

assumption that the industry-funded study assigned participants, rather than classes to schools.

Ignoring the highly significant heterogeneity and nonetheless choosing a fixed-effect model leads to a finding of significant personal oral hygiene effects.[14,15] Both the overall significant effect, and the significant heterogeneity was driven by one industry-funded study. When the industry-funded study was included by means of a fixed-effects model, it led a significant benefit of personal oral hygiene in terms of reducing dental caries incidence (([SMD = −0.11; 95% CI (−0.21, −0.00), *P*-value = .042]. The therapeutic effect of personal oral hygiene increased in significance in a fixed-effect model if the assumption was made that the industry-funded study randomly assigned subjects, and not classes, to the oral hygiene intervention ($P < .001$).

4 | DISCUSSION

A meta-analysis of controlled clinical trials does not support the hypothesis that improved personal oral hygiene without fluoride decreases the risk of coronal caries. This lack of effectiveness cannot be attributed to the presence of water fluoridation dwarfing the benefits of oral hygiene because two of the three randomised trials were conducted in nonfluoridated communities. Neither can the lack of effectiveness be attributed to non-supervision of the oral hygiene intervention. Two randomised trials with strict protocols involving daily supervised plaque staining and removal on school days reported significant reduction in gingivitis but not of dental caries.[16,18] These randomised trial findings are robust towards sensitivity analyses. The randomised trial findings are also robust when considering nonrandomised trials with the exception of one industry-funded, nonrandomised study which was the only study to report highly significant benefits of personal oral hygiene.

The primary conclusion of this systematic review, that oral hygiene has no impact on dental caries rates, is strengthened by other independent lines of evidence. A network meta-analysis of six systematic reviews on 130 controlled trials selected under the auspices of the Cochrane Oral Health Group compared the effectiveness of four fluoride products (gels, varnishes, toothpaste and rinses) in over 60 000 participants. The findings of this network meta-analysis showed that fluoride toothpaste, and fluoride rinse were similarly effective in preventing coronal dental caries. So, in effect, personal oral hygiene was shown to be an inactive delivery mechanism for an active pharmaceutical, fluoride. Our findings are furthermore consistent with systematic reviews on the ineffectiveness of self-performed flossing,[23-25] professional dental plaque removal[26,27] and removal of dental plaque prior to fluoride applications for preventing coronal dental caries.[28]

Controlled trials on topical chemotherapeutics provide additional insights on the validity of the oral hygiene hypothesis. Biofilm on smooth non-dental surfaces, but not in cracks and crevices, can be removed by mechanical cleansing and chemotherapeutics.[29] Chemotherapeutics are therefore more likely to be effective if dental caries starts on smooth, defect-free surfaces. A century of controlled trials provides insights into this assumption. A hexylresorcinol rinse was the first major antimicrobial advertised commercially as an effective caries control, but in one of the first controlled trials, it increased the caries rates.[30] This finding was followed by trials involving about one thousand children who were assigned to a placebo- or a penicillin-containing toothpaste. The penicillin-containing toothpastes were found to be unsuccessful at controlling caries.[31,32] Chlorhexidine was described in 1994 as the most potent chemotherapeutic agent against dental caries surrogates,[33] but it too was deemed ineffective following a meta-analyses of controlled trials on the endpoint of frank cavitation.[10] Thus, trials on chemotherapeutics support the dental defect hypothesis on dental caries etiology.

Roughly one century ago, Mellanby predicted the failure of the oral hygiene hypothesis and had proposed instead, based on experimental animal research, that dental caries started in dental defects.[34] This dental defect hypothesis was consistent with the epidemiology of dental caries and widely accepted in the medical and dental professions.[35] The dental defect hypothesis ended up explaining dramatic historical and geographical trends in dental caries prevalence in ways that the oral hygiene hypothesis could not.[36-38] The ubiquity of dental caries in the early 20th century was attributed to sugar, rickets and paediatric fevers, and the latter two identified as causes of dental defects.[39] According to the dental defect hypothesis, the dramatic decline of caries in wealthier countries that occurred in the second half of the 20th century is attributed to the widespread use of dietary vitamin D supplements to overcome epidemics of paediatric malnutrition, and to national vaccination programmes which started in the 1960s and reduced infant and childhood fevers. The emergence of defect-free teeth following these vaccinations and vitamin D supplementation created a larger population of children with defect-free teeth resistant to the carcinogenicity of sugar and bacteria.[36] Dental defects as drivers of caries incidence can explain why caries rates can increase or decrease regardless of sugar intake or water fluoridation.

The dental defect hypothesis is also consistent with the effectiveness of vitamin D[40] and may lead to different perspectives on how fluoride prevents dental caries. Research suggests that fluoride, just like vitamin D, may exert, at least partially, its beneficial effects systemically[41]; topical fluorides therefore possibly reduce dental caries because of their systemic absorption through the oral mucosa and not due to their topical effects. This would explain how fluoride can be effective even though it poorly penetrates dental plaque.[42] This would also explain why common wisdoms on how fluoride strengthens teeth have become questioned. Specifically, incorporation of up to 1000 ppm fluoride into the outer dental enamel (hydroxy-apatite) has been shown to offer no resistance against demineralisation attacks.[43] Even enamel fluorapatite (30 000 ppm fluoride) was shown to offer minimal resistance to demineralisation[44] and to offer a questionable barrier to dental caries.[45] Further

investigations should determine to what extent fluoride and vitamin D prevent dental caries through pulpal side mechanisms.[46]

This review does not question whether clinicians should provide advice on the *potential* benefits of oral hygiene for preventing dental caries. There is still substance to the arguments that small therapeutic effects of personal oral hygiene remained undetected in statistically underpowered trials, that findings in healthy paediatric populations with no exposed cementum do not extrapolate to adults with exposed cementum or decreased saliva flow, or that adults with other systemic diseases or disorders may benefit from personal oral hygiene in terms of dental caries prevention. Indeed, oral hygiene can be a pleasant and cost-effective way to deliver fluoride, reduce gingivitis, remove food impactions, or to help patients in their recovery from oral surgical procedures. The dangers in the unqualified promotion of oral hygiene for dental caries prevention are that it may lead individuals to select fluoride-free toothpastes, to sacrifice effective fluoride exposure for interproximal cleaning without fluor, or to forego effective therapeutics such as fluoride rinses. Such beliefs which reduce fluoride exposure increase dental caries risk and are most dangerous when used as a justification to promote sugar consumption, and to perpetuate the myth that sugar is safe to eat as long as one brushes their teeth.[47]

5 | CONCLUSIONS

A large body of controlled trials has failed to provide convincing evidence in support of the efficacy of personal oral hygiene in preventing or controlling coronal dental caries.

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REFERENCES

1. Kassebaum NJ, Bernabe E, Dahiya M, Bhandari B, Murray CJ, Marcenes W. Global burden of untreated caries: a systematic review and metaregression. *J Dent Res*. 2015;94:650-658.
2. MacEntee M, Bryant S, Keller H, Nguyen C, Yao C. Caries control for frail elders. In: Fejerskov O, Nyvad B, Kidd EAM, eds. *Dental Caries: The Disease and its Clinical Management*, 3rd edn. Chichester, West Sussex, UK; Ames, Iowa: John Wiley & Sons Inc.; 2015:321-332.
3. Gies WJ. The dentifrice problem. *J Am Dent Assoc*. 1921;8:851-859.
4. Mellanby E. The influence of diet on teeth formation. *Lancet*. 1918;192:767-770.
5. Salanti G, Marinho V, Higgins JP. A case study of multiple-treatments meta-analysis demonstrates that covariates should be considered. *J Clin Epidemiol*. 2009;62:857-864.
6. Moher D, Stewart L, Shekelle P. Implementing PRISMA-P: recommendations for prospective authors. *Syst Rev*. 2016;5:15.
7. Stein C, Santos NML, Hilgert JB, Hugo FN. Effectiveness of oral health education on oral hygiene and dental caries in schoolchildren: systematic review and meta-analysis. *Community Dent Oral Epidemiol*. 2018;46:30-37.
8. Jadad AR, Moore RA, Carroll D, et al. Assessing the quality of reports of randomized clinical trials: is blinding necessary? *Control Clin Trials*. 1996;17:1-12.
9. Higgins JP, Altman DG, Gotzsche PC, et al. The Cochrane Collaboration's tool for assessing risk of bias in randomised trials. *BMJ*. 2011;343:d5928.
10. Rethman MP, Beltran-Aguilar ED, Billings RJ, et al. Nonfluoride caries-preventive agents: executive summary of evidence-based clinical recommendations. *J Am Dent Assoc*. 2011;142:1065-1071.
11. Burnside G, Pine CM, Williamson PR. Statistical aspects of design and analysis of clinical trials for the prevention of caries. *Caries Res*. 2006;40:360-365.
12. Ashley FP, Sainsbury RH. The effect of a school-based plaque control programme on caries and gingivitis. A 3-year study in 11 to 14-year-old girls. *Br Dent J*. 1981;150:41-45.
13. Ialongo C. Understanding the effect size and its measures. *Biochem Med*. 2016;26:150-163.
14. Borenstein M, Hedges LV, Higgins JP, Rothstein HR. A basic introduction to fixed-effect and random-effects models for meta-analysis. *Res Synth Methods*. 2010;1:97-111.
15. Kuan Y, Tam K. The selection of fixed- or random-effect models in recent published meta-analyses. 2015. Cochrane Colloquium; Vienna.
16. Silverstein S, Gold S, Heibron D, Nelms D, Wycoff S. Effects of supervised deplaquing on dental caries, gingivitis, and plaque. *J Dent Res*. 1977;56:Abstract 169.
17. McKee DP, Faine RC, Murphy RF. The effectiveness of a dental health education program in a nonfluoridated community. *J Public Health Dent*. 1977;37:290-299.
18. Horowitz AM, Suomi JD, Peterson JK, Matthews BL, Voglesong RH, Lyman BA. Effects of supervised daily dental plaque removal by children after 3 years. *Community Dent Oral Epidemiol*. 1980;8:171-176.
19. Spears ND, Goldstein C, Gordinier N, Littman J. The effect of daily supervised deplaquing on dental caries in school children. A multiphasic study after 21 months. *J Calif Dent Assoc*. 1978;6:36-42.
20. Koch G. Effect of sodium fluoride in dentifrice and mouthwash on the incidence of dental caries in school children. *Odont Revy*. 1967;18(Suppl. 12):37-43.
21. Clark CA, Fintz JB, Taylor R. Effects of the control of plaque on the progression of dental caries: results after 19 months. *J Dent Res*. 1974;53:1468-1474.
22. Fosdick LS. The reduction of the incidence of dental caries; immediate tooth-brushing with a neutral dentifrice. *J Am Dent Assoc*. 1950;40:133-143.
23. Hujoel PP, Cunha-Cruz J, Banting DW, Loesche WJ. Dental flossing and interproximal caries: a systematic review. *J Dent Res*. 2006;85:298-305.
24. Sambunjak D, Nickerson JW, Poklepovic T, et al. Flossing for the management of periodontal diseases and dental caries in adults. *Cochrane Database Syst Rev*. 2011;(12):CD008829.
25. Poklepovic T, Worthington HV, Johnson TM, et al. Interdental brushing for the prevention and control of periodontal diseases and dental caries in adults. *Cochrane Database Syst Rev*. 2013;(12):CD009857.
26. Azarpazhooh A, Main PA. Efficacy of dental prophylaxis (rubber cup) for the prevention of caries and gingivitis: a systematic review of literature. *Br Dent J*. 2009;207:E14; discussion 328-329.
27. Riley P, Worthington HV, Clarkson JE, Beirne PV. Recall intervals for oral health in primary care patients. *Cochrane Database Syst Rev*. 2013;(12):CD004346.
28. Weyant RJ, Tracy SL, Anselmo TT, et al. Topical fluoride for caries prevention: executive summary of the updated clinical

recommendations and supporting systematic review. *J Am Dent Assoc*. 2013;144:1279-1291.
29. Biofilms Stoodley P. Flow disrupts communication. *Nat Microbiol*. 2016;1:15012.
30. Bunting RW, Hadley FP, Jay P, Hard DG. The problem of dental caries. *Am J Dis Child*. 1930;40:536.
31. Hill TJ, Sims J, Newman M. The effect of penicillin dentifrice on the control of dental caries. *J Dent Res*. 1953;32:448-452.
32. Hill TJ, Kniesner AH. Penicillin dentifrice and dental caries experience in children. *J Dent Res*. 1949;28:263-266.
33. Emilson CG. Potential efficacy of chlorhexidine against mutans streptococci and human dental caries. *J Dent Res*. 1994;73:682-691.
34. Nicolson M, Taylor GS. Scientific knowledge and clinical authority in dentistry: James Sim Wallace and dental caries. *J R Coll Physicians Edinb*. 2009;39:64-72.
35. McCollum EV. *The Newer Knowledge of Nutrition*, 5th edn. New York, NY: The Macmillan Company; 1939.
36. Goodson JM, Tavares M, Wang X, et al. Obesity and dental decay: inference on the role of dietary sugar. *PLoS ONE*. 2013;8:e74461.
37. Diesendorf M. The mystery of declining tooth decay. *Nature*. 1986;322:125-129.
38. McLaren L, Singhal S. Does cessation of community water fluoridation lead to an increase in tooth decay? A systematic review of published studies. *J Epidemiol Community Health*. 2016;70:934-940.
39. National Research Council - Committee on Dental Health. A survey of the literature of dental caries. 1952; 277.
40. Hujoel PP. Vitamin D and dental caries in controlled clinical trials: systematic review and meta-analysis. *Nutr Rev*. 2013;71:88-97.
41. Larmas M. Dental caries seen from the pulpal side: a non-traditional approach. *J Dent Res*. 2003;82:253-256.
42. Watson PS, Pontefract HA, Devine DA, et al. Penetration of fluoride into natural plaque biofilms. *J Dent Res*. 2005;84:451-455.
43. Featherstone JD. The science and practice of caries prevention. *J Am Dent Assoc*. 2000;131:887-899.
44. Ogaard B, Rolla G, Ruben J, Dijkman T, Arends J. Microradiographic study of demineralization of shark enamel in a human caries model. *Scand J Dent Res*. 1988;96:209-211.
45. Muller F, Zeitz C, Mantz H, et al. Elemental depth profiling of fluoridated hydroxyapatite: saving your dentition by the skin of your teeth? *Langmuir*. 2010;26:18750-18759.
46. Waddington RJ, Langley MS. Altered expression of matrix metalloproteinases within mineralizing bone cells in vitro in the presence of fluoride. *Connect Tissue Res*. 2003;44:88-95.
47. Smith P. Coca-Cola myths are real thing, says Australian ad watchdog. *Financial Times*. April 13, 2009;Companies & Markets: 13.

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