Layered Caries Prevention Model: A Hypothesis

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> **Working draft, not peer reviewed.** This is a hypothesis paper, not medical or dental advice. Talk to a dentist about your own care. References marked † are still being verified.

Abstract

Dental caries is the most prevalent preventable chronic disease, yet prevention is usually delivered as separate, product-based advice. We hypothesize that caries incidence falls further under a layered, risk-stratified model whose base layer is the frequency of fermentable-carbohydrate exposure, not any single agent. Layer 1 reduces daily acid challenges by consolidating eating occasions and lowering fermentable carbohydrate between meals. Layer 2 strengthens remineralization: twice-daily fluoride toothpaste (higher concentration at high risk), spitting without rinsing, a delay before eating or drinking, and adjunct remineralizing agents. Layer 3 adds clinician-applied fluoride varnish, silver diamine fluoride for active lesions, and pit-and-fissure sealants. A speculative fourth element is biofilm modulation with oral probiotics (including *Bacillus subtilis* natto, whose extracts inhibit *Streptococcus mutans* biofilm in vitro) and plant-derived agents (coconut oil pulling, mastic chewing gum, essential-oil rinses), tested only on plaque, gingivitis or bacterial counts. Because the layers act on different terms of the demineralization–remineralization balance, we propose their effects are partly additive, and that Layer 1 raises the benefit of Layers 2 and 3. Evidence is strongest for fluoride, then sealants, varnish and sugar restriction; moderate to low for silver diamine fluoride and remineralizing agents; and absent for single-meal or low-carbohydrate patterns, for *B. subtilis* in humans, and for caries outcomes with any Layer 4 agent. No trial has tested the combination. We state falsifiable predictions and feasible tests, including factorial and split-mouth designs. If supported, the model would enable risk-based, mechanism-ordered prevention.

Keywords: dental caries; prevention; silver diamine fluoride; remineralization; oral microbiome; dietary frequency

1. Introduction

Untreated caries in permanent teeth is among the most common health conditions worldwide [1]. It is a dynamic, diet-dependent process. Plaque bacteria ferment dietary carbohydrate to acid, plaque pH falls, and mineral leaves the tooth until saliva, fluoride and other factors restore the balance [1,3]. Repeated acid challenges also select for acid-producing, acid-tolerant species, so diet shapes the biofilm as well as feeding it [2,3]. The same ecological logic has been extended to periodontal disease [4].

The evolutionary record fits this picture. In ancient dental calculus, the periodontal pathogen *Porphyromonas gingivalis* was already present in Mesolithic hunter-gatherers and was more frequent in Neolithic and later farmers. *Streptococcus mutans* was not detected in the Mesolithic and Neolithic samples, was rare in Bronze Age and medieval samples, and became dominant only in modern samples, apparently with the Industrial Revolution and its refined sugar and flour [5]. Farming therefore shifted the oral microbiota toward a disease-associated state, but the main caries organism followed much later. The ancient groups were small (4 to 18 samples per period) [5]. Reviews of the modern evidence likewise treat free sugars as a central driver of caries [6].

Current prevention nonetheless arrives as separate components: fluoride products, sealants, professional agents and general dietary advice, each tested in its own trials. We found no trial that tests them together, and no framework that orders them by the biological term each one acts on. Here we propose one. We also ask whether the diet layer should target the number of acid challenges per day, a variable whose independent effect is hard to separate from total intake [7], rather than total sugar alone.

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2. The hypothesis

Statement. Caries is best prevented by four layers ordered by the term of the demineralization–remineralization balance each one acts on, with exposure frequency as the base layer. The intensity of each layer should scale with individual caries risk.

The layers

1. **Exposure (Layer 1).** Fewer, shorter acid challenges per day: consolidate eating into defined occasions, avoid fermentable carbohydrate and acidic drinks between them, and keep sticky or retentive sugar and starch to meals [6,8]. A low-carbohydrate pattern is one route to this, not a requirement.
2. **Mineral defense (Layer 2).** Twice-daily brushing with fluoride toothpaste, with a higher concentration for high-risk adults [9]. After brushing, spit and do not rinse, and leave a defined interval before eating or drinking, so fluoride stays in plaque and saliva [10,11]. Adjuncts (hydroxyapatite, calcium phosphate, arginine, xylitol) belong here.
3. **Clinician-applied (Layer 3).** Fluoride varnish [12], sealants on susceptible pits and fissures [13], and silver diamine fluoride for active lesions [14], where Layers 1 and 2 cannot reach or have failed.
4. **Biofilm modulation (Layer 4, speculative).** Oral probiotics, including *Bacillus subtilis* natto, and plant-derived agents (coconut oil pulling, mastic chewing gum, essential-oil rinses), to bias plaque away from acid producers or reduce plaque.

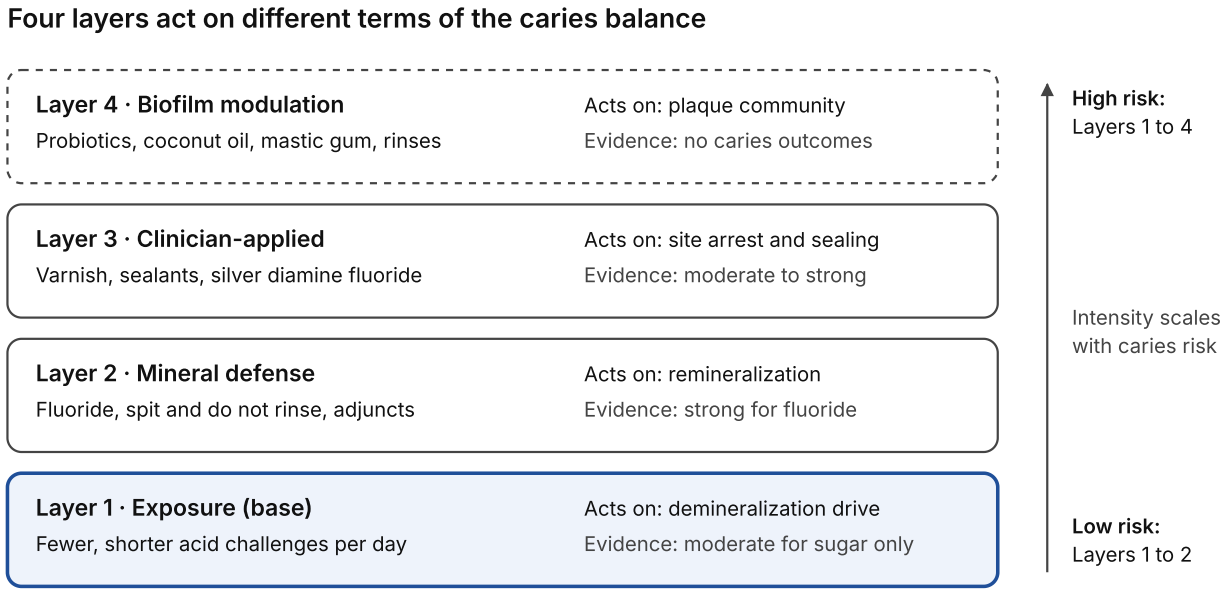


Figure 1. The layered model. The dashed outline marks the speculative layer. Concept diagram prepared with AI assistance.

Causal chain. Layer 1 lowers the number of low-pH episodes and the pressure that selects aciduric bacteria [2,3]. Layer 2 raises fluoride and mineral availability during each recovery phase, so each recovery gains more mineral. Layer 3 protects or arrests the sites where the first two are weakest. Because the layers act on different terms of the balance, their effects should be partly additive.

Three testable claims

- **H1, additivity:** the combination lowers caries increment more than any single layer.
- **H2, dependency:** the benefit of Layers 2 and 3 is larger when exposure frequency is low, because frequent acid challenges overwhelm fluoride's recovery window.
- **H3, stratification:** low-risk people gain little from Layer 3, and high-risk people gain most.

How this differs from current thinking. Each measure here is established or at least studied. We differ in three ways. First, the diet target is challenge frequency and carbohydrate retentiveness, not total sugar alone. Second, we predict a dependency between diet and fluoride benefit (H2) that we found no trial testing. Third, we separate what has trial support (Layers 2 and 3) from what does not (single-meal or low-carbohydrate patterns, and Layer 4) [15], and label each accordingly.

How the idea evolved. It began from examining self-assembled home routines that stack many products without ordering them, and asking which parts have trial support.

3. Evaluation of the hypothesis

Evidence is graded by what each study measured. Strong means randomized trials or systematic reviews with caries outcomes. Table 1 summarizes the grades.

3.1 Layer 1: exposure

Observational and experimental evidence supports limiting sugar. In the Vipeholm study, sugar eaten between meals, especially in sticky form, raised caries activity more than sugar eaten with meals [7]. A systematic review for the World Health Organization found less caries when free sugars supplied under 10% of energy, with possible further benefit below 5%, on moderate-quality, mostly observational evidence [16]. Cooked, retentive starch is cariogenic, especially with sugar, whereas raw starch is much less so [8].

Evidence for the frequency component is weaker, because frequency and total intake are hard to separate in these data [7]. Evidence for low-carbohydrate or single-meal patterns is absent for caries. A 2024 scoping review of ketogenic diets and the periodontium found about eight small studies, mostly of other outcomes, with sparse caries data [15]. A pilot trial of an oral-health-optimized low-carbohydrate diet reported less gingival inflammation, which is not a caries outcome [17]. Plausible harms, such as low saliva, nutrient gaps and erosion from acidic foods, are untested [15].

3.2 Layer 2: mineral defense

Fluoride toothpaste is the best-supported single measure. A Cochrane review found a dose–response with fluoride concentration, mostly in children and adolescents [9]. For adults at high risk, one trial of 135 people found that 5,000 ppm toothpaste hardened root lesions more than 1,350 ppm over six months. The outcome was a surface-hardness surrogate, follow-up was short, and the manufacturer funded it [18].

Spitting without rinsing has indirect support. Within a clinical trial, rinsing after brushing was associated with greater caries increment [10], and fluoride is retained longer in saliva without rinsing [11]. We found no trial of a delay before eating or drinking, so that element rests on mechanism alone.

Adjuncts have weaker evidence. A meta-analysis of nano-hydroxyapatite could not pool in vivo caries data and rated the evidence very low [19]. A two-year trial of about 6,000 children found that 1.5% arginine toothpaste reduced caries increment 16.5% more than 1,450 ppm fluoride, but the manufacturer funded it [20]. A Cochrane review found low-certainty evidence that xylitol-fluoride toothpaste may modestly outperform fluoride alone in children, and insufficient evidence for other xylitol products [21].

3.3 Layer 3: clinician-applied measures

Fluoride varnish reduces caries in children and adolescents [12], and sealants reduce occlusal caries in the permanent molars of children [13]. Silver diamine fluoride (SDF) has weaker, more qualified evidence. The 2024 Cochrane review pooled 29 randomized trials (13,036 participants, mostly children), all at high risk of bias [14].

SDF probably prevents new root caries compared with placebo or no treatment (mean difference −0.79 surfaces, 95% CI −1.40 to −0.17; 3 trials, 439 participants; moderate certainty). Evidence that it arrests caries in primary teeth is of low certainty, and for permanent coronal surfaces it is very uncertain. Against fluoride varnish it showed no difference in new caries in primary teeth. Staining and adverse effects were poorly reported [14]. A US clinical guideline gives conditional support to 38% SDF for cavitated lesions [22].

3.4 Layer 4: biofilm modulation (speculative)

Water extracts of natto, a food fermented with *Bacillus subtilis* natto, inhibited sucrose-dependent biofilm formation by cariogenic streptococci in vitro [23]. Inhibition tracked protease activity, occurred without killing the cells, and was abolished by heat and a protease inhibitor, while soybean extract alone increased biofilm [23]. This is a laboratory result on food extracts. We found no human or animal caries data for *B. subtilis* natto, and it is unknown whether the organism colonizes the mouth or only passes through. For *Streptococcus salivarius* M18, a randomized trial in children reported lower plaque scores, and salivary *S. mutans* was lower in children colonized by M18; both are surrogate outcomes [24].

Coconut oil pulling. A 2024 systematic review of 25 randomized trials (1,184 participants) rated the certainty of evidence very low for every outcome [26]. Chlorhexidine reduced plaque more than oil pulling (SMD 0.33, 95% CI 0.17 to 0.49; 8 trials). Gingival index and salivary *S. mutans* did not differ significantly (*S. mutans* SMD 0.19; 6 trials) [26]. Most trials were at high risk of bias, lasted 30 days or less, and reported no caries outcome [26]. In a 14-day trial of 60 volunteers, *S. mutans* counts fell 22.8% with coconut oil and 25.7% with chlorhexidine, without a significant difference between them [27]. A 30-day trial of 80 people concluded that virgin and regular coconut oil were as efficient as chlorhexidine in reducing gingivitis [28].

Mastic chewing gum. In a double-blind pilot trial (10 per group, 7 days without tooth cleaning), mastic gum gave lower plaque (2.69 vs 3.15, P = 0.001) and gingival index scores (0.44 vs 0.66, P = 0.021) than placebo gum [29]. Small human studies report lower salivary mutans streptococci after chewing mastic gum [30,31]. The one caries-related study, an in situ crossover of 15 people, found less demineralization with mastic and with xylitol, with no difference between them [31].

Essential-oil rinse and eucalyptus. A systematic review of 19 trials found chlorhexidine better than a standardized essential-oil mouthwash for plaque, with no significant difference in long-term gingival inflammation; the authors called it a reliable long-term alternative for gingival inflammation [32]. In a 12-week double-masked trial of 97 adults with gingivitis, eucalyptus-extract chewing gum improved plaque, gingival index and bleeding versus placebo, with no effect on clinical attachment level [33]. In a 2024 crossover trial of 60 people with gingivitis, a eucalyptus-oil toothpaste reduced gingival bleeding more than a miswak toothpaste (P = 0.002) [34]. Neither reported caries.

Gingival recession. Recession is not addressed. We found no trial of a rinse, oil or gum for recession; the Cochrane review of recession treatment covers surgical root coverage, with low to very low quality evidence [35].

3.5 Evidence against the model

- **No combination trial.** Each layer is tested alone, mostly in children, and adult evidence is thin [9,14].
- **Redundancy.** If fluoride and sugar control already prevent most caries, added layers may give diminishing returns. SDF showed no extra benefit over fluoride varnish for new caries in primary teeth [14].
- **Industry funding.** The arginine and 5,000 ppm trials were manufacturer-funded [18,20].
- **Frequency versus amount.** The central diet claim is not cleanly separable in existing data [7].
- **Harm and adherence.** Strict low-carbohydrate or single-meal patterns may carry nutritional risks, and adherence is uncertain [15]. Sugar alcohols are not risk-free: higher blood erythritol was associated with cardiovascular events in cohort studies, though causation is unproven [25].
- **Surrogate outcomes.** Every Layer 4 agent has been tested on plaque, gingivitis or bacterial counts, not caries [26,29,31].
- **Out of scope.** Other agents with gingivitis-only data, such as most supplements, are excluded because they lack caries evidence.

Table 1. Evidence grade for each component of the model.

Component	Layer	Evidence for caries	Main limitation
Free-sugar restriction	1	Moderate	Mostly observational [16]
Eating-frequency consolidation	1	Low	Confounded with intake [7]
Low-carbohydrate or single-meal pattern	1	None found	No caries trials [15]
Fluoride toothpaste	2	Strong	Mostly children [9]
5,000 ppm toothpaste	2	Low	One 6-month surrogate trial [18]
Spit, do not rinse	2	Low	Observational, within a trial [10]
Delay before eating	2	None found	Mechanism only
Nano-hydroxyapatite	2	Very low	No pooled caries data [19]
Arginine toothpaste	2	Low to moderate	Manufacturer-funded [20]
Xylitol	2	Low	Mainly children [21]
Fluoride varnish	3	Moderate to strong	Children and adolescents [12]

Component	Layer	Evidence for caries	Main limitation
Sealants	3	Moderate to strong	Children [13]
Silver diamine fluoride	3	Moderate (root caries), low (arrest)	High risk of bias [14]
B. subtilis natto	4	In vitro only	No human data [23]
S. salivarius M18	4	Surrogate only	Counts, not caries [24]
Coconut oil pulling	4	Very low (GRADE); no caries data	Surrogates only [26]
Mastic chewing gum	4	Surrogate only	Small trials [29,31]
Essential-oil mouthwash	4	Gingivitis only	No caries data [32]
Eucalyptus extract or oil	4	Gingival outcomes only	No caries data [33,34]

4. Hypothesis testing

The hypothesis can be tested at three levels, from cheapest to most definitive. Table 2 lists what would support or weaken each claim.

Laboratory and in situ. pH-cycling models can vary the number of daily acid challenges with and without fluoride, testing the dependency in H2 on enamel and root dentin. Palatal-appliance studies in volunteers add real saliva and plaque. For Layer 4, natto-derived protease and live *B. subtilis* can be tested in multispecies biofilm models, then in animal caries models, before any human use.

Clinical. A factorial randomized trial with the layers as factors tests H1 and H2 directly. The main effects show each layer, and the interaction between Layer 1 and Layers 2–3 tests dependency. Caries-risk strata (H3) can serve as a blocking factor, and split-mouth designs suit Layer 3 agents. Outcomes should be lesion-level (ICDAS scores, radiographic progression), with salivary flow, buffering capacity and *S. mutans* and *Lactobacillus* counts as mechanistic secondary outcomes. A Layer 1 arm needs verified intake data and a credible control, because blinded dietary trials are hard.

Secondary analysis. Existing cohorts and trial datasets that recorded eating frequency and fluoride use could be re-analysed for an interaction between the two, a cheap first test of H2.

Table 2. Predictions and what would count against them.

Claim	Prediction	Would weaken the claim
H1, additivity	Combined layers give the lowest caries increment	Combination no better than fluoride alone
H2, dependency	Fluoride benefit is larger when acid challenges are fewer	No diet–fluoride interaction
H3, stratification	Layer 3 benefit is larger in high-risk participants	Equal benefit across risk strata
Layer 4	Natto protease lowers acid output in multispecies biofilms	No effect in biofilm models
Layer 4 plant agents	A lesion-level caries benefit beyond fluoride	Fewer bacteria or less plaque but no caries difference

5. Consequences and limitations

If confirmed, the model would change prevention in three ways. Clinicians could rank interventions by mechanism and risk rather than by product. Eating-pattern counselling would become a measured part of caries prevention with a defined target. And it would show which clinician-applied agents add value for whom, potentially sparing low-risk patients treatments they do not need.

If H2 is confirmed, the benefit of fluoride depends on diet, and fluoride trials should record dietary exposure and analyse it as an effect modifier. If the hypothesis is refuted, the result still helps: a null interaction would show that layers can be chosen independently, and a null combination effect would show where returns diminish.

Limitations. The model is assembled from separate trials, mostly in children, and extends them to adults and to combinations without direct evidence. Several elements rest on mechanism alone: the post-brushing delay, single-meal or low-carbohydrate patterns, and Layer 4. The scope is caries only; periodontal and systemic claims are excluded.

Safety. SDF stains lesions black, is unsuitable for people with silver allergy, and is applied by a clinician; its regulatory status for caries varies by country. High-concentration fluoride products are prescribed or professionally applied. Strict low-carbohydrate patterns need nutritional monitoring, including fat-soluble vitamins. Oil pulling has an aspiration risk: two case reports describe lipoid pneumonia [36]. This paper presents a hypothesis and is not clinical advice.

Declarations

Use of generative AI. This manuscript was drafted with the assistance of Claude (Anthropic) for literature search, summarization and drafting. The author reviewed and edited the content and takes full responsibility for it. Figure 1 was prepared with AI assistance.

Competing interests. The author operates Realien Labs, which sells merchandise, ebooks and apps. No product discussed in this paper is sold by Realien Labs.

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Ethics. No human or animal data were collected.

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