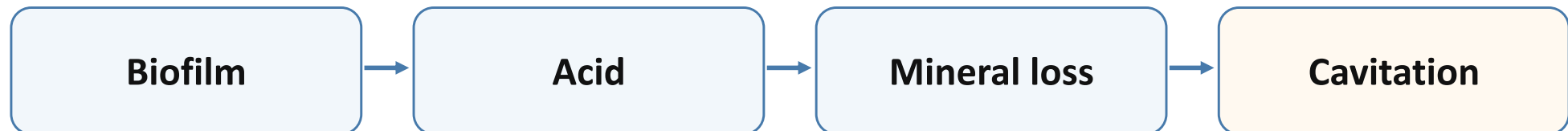


Caries (Tooth Decay)

Disease mechanism, morphology, risk, and clinicopathologic management



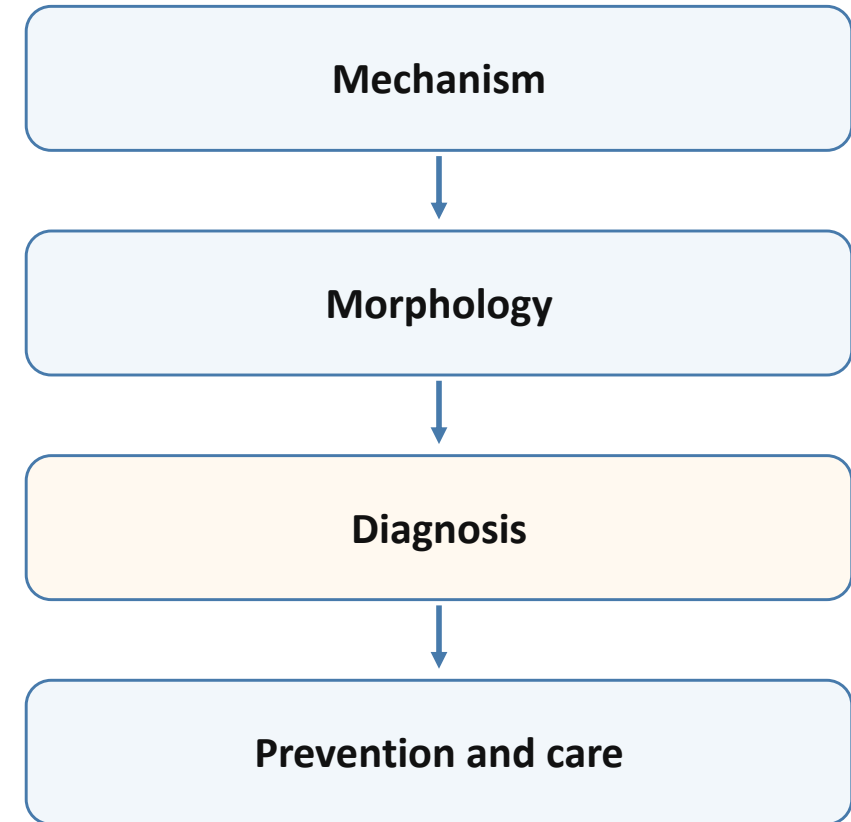
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Learning objectives

- Explain the mechanism linking dental biofilm, dietary sugars and focal tooth demineralization.
- Distinguish early noncavitated lesions from irreversible structural cavitation.
- Connect lesion location and extent to examination findings and complications.
- Compare preventive, nonrestorative and restorative strategies.



Caries is a dynamic hard-tissue disease

- The core lesion is focal loss of mineral from enamel and dentin.
- Acid generated by bacterial fermentation of sugars initiates mineral loss.
- Demineralization and remineralization compete over time.
- A lesion may progress, arrest or regress before structural cavitation.

Demineralization

Sugar exposure

Acidogenic biofilm

Frequent challenges

Protective factors

Saliva minerals

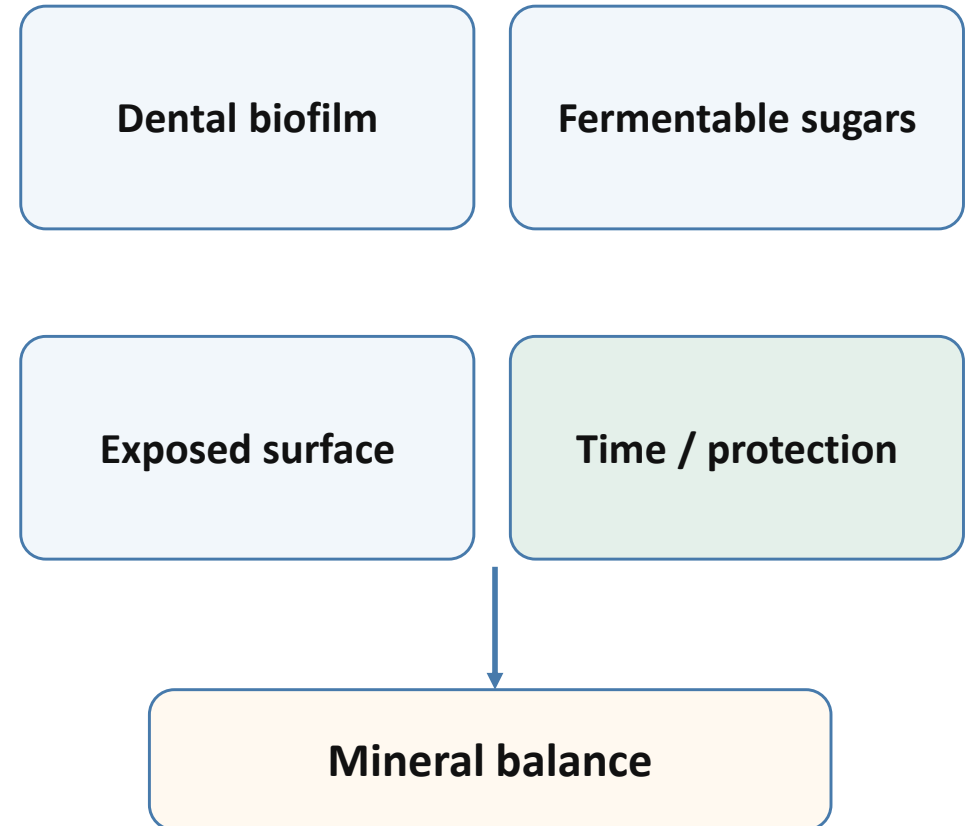
Fluoride

Plaque disruption

Net lesion activity

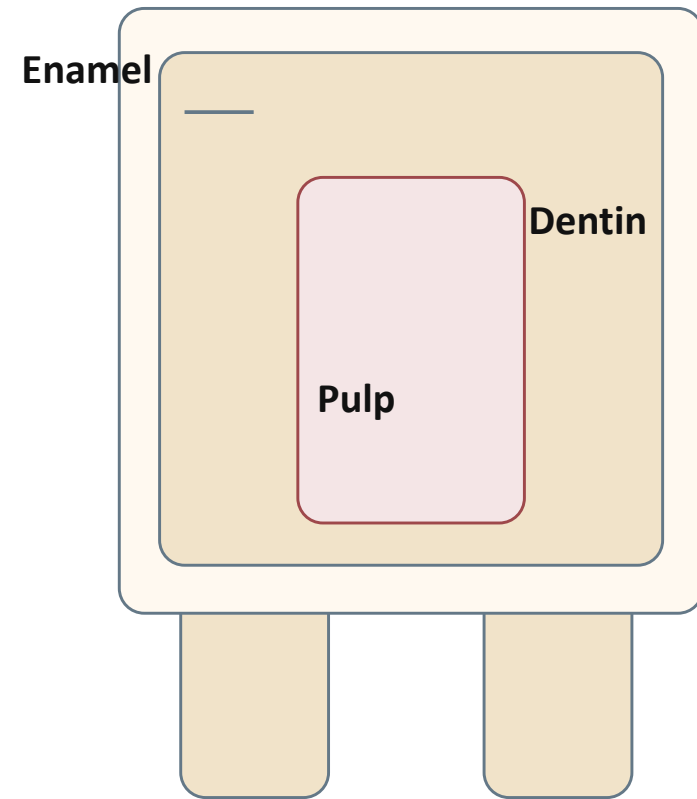
The pathogenic system requires interacting factors

- A dental biofilm provides a local microbial community.
- Fermentable dietary carbohydrates provide substrate for acid production.
- Tooth surfaces experience repeated acid exposure.
- Saliva, fluoride and plaque removal modify the outcome.



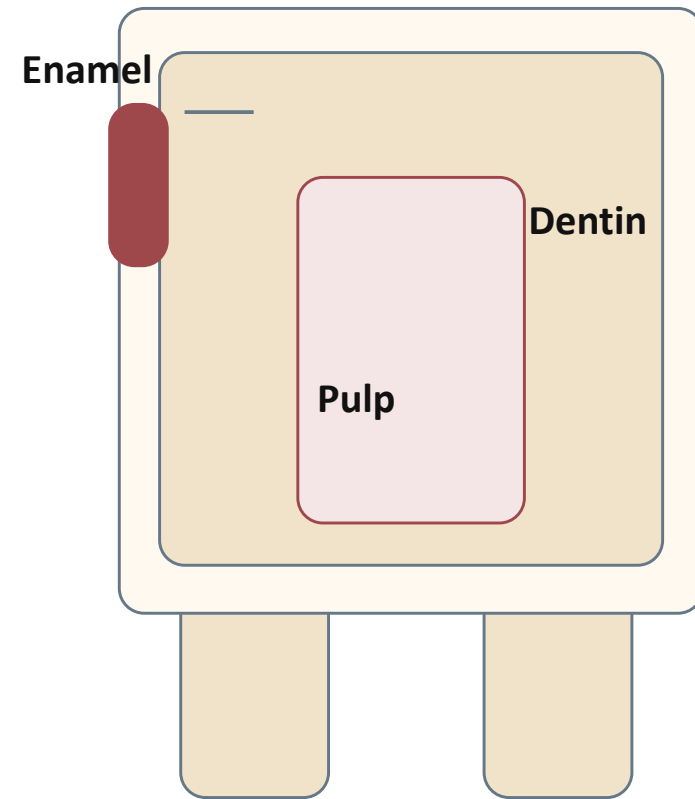
Tooth anatomy determines the consequences of decay

- Enamel covers the crown and is highly mineralized.
- Dentin underlies enamel and forms the bulk of the tooth.
- The pulp is a neurovascular tissue within the tooth.
- Progression toward the pulp explains pain and potential infection.



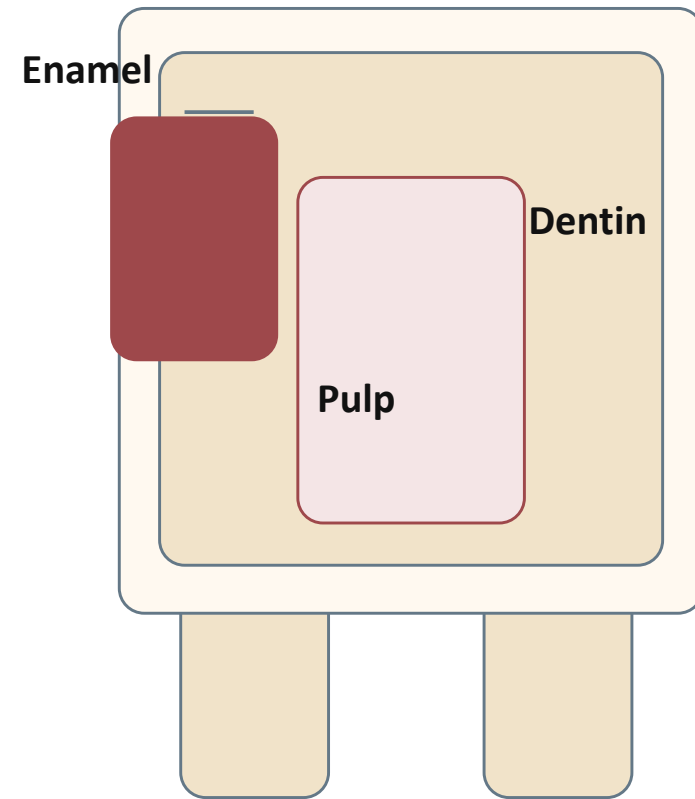
Enamel is the initial mineral barrier

- The outer enamel is exposed directly to biofilm-derived acids.
- Acid removes mineral from the crystalline tooth surface.
- Early demineralization may be visible as a white-spot lesion.
- Surface integrity can be preserved at this early stage.



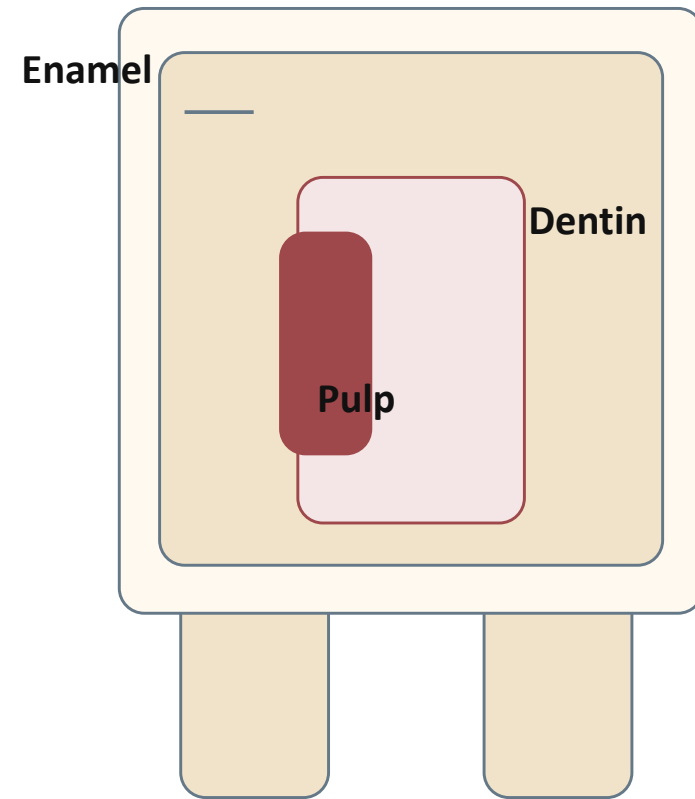
Dentin involvement changes lesion behavior

- Dentin lies beneath enamel and can be affected after further tissue breakdown.
- Dentin involvement marks greater structural extent of the lesion.
- Clinical evaluation distinguishes lesion extent from surface appearance alone.
- Intervention depends on cavitation, lesion activity and patient risk.



Pulpal proximity gives caries clinical importance

- The pulp contains nerves and blood vessels.
- Advancing destruction can produce sensitivity and toothache.
- Untreated advanced lesions can become complicated by infection.
- Facial swelling or fever warrants evaluation for infectious complications.



The oral microbiome is not uniformly pathogenic

- A diverse oral microbial ecosystem.
- Many resident microorganisms are commensal.
- Ecological disruption can favor oral diseases, including caries.
- Disease reflects microbial function in context, not simply the presence of bacteria.

Demineralization

Sugar exposure

Acidogenic biofilm

Frequent challenges

Protective factors

Saliva minerals

Fluoride

Plaque disruption

Net lesion activity

Dental plaque creates a localized acid-producing niche

- Plaque is an adherent biofilm on and between tooth surfaces.
- Its constituents include microorganisms and salivary components.
- Retention near a tooth focuses metabolic acid exposure.
- Consistent cleaning disrupts the biofilm and reduces caries risk.

Adherent dental plaque

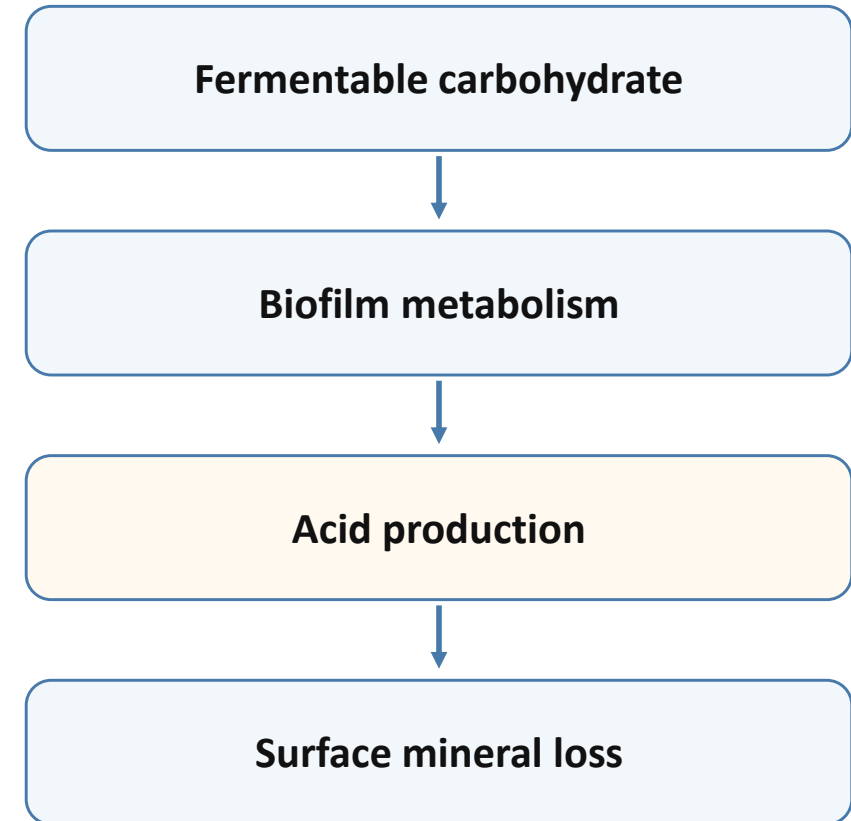
Biofilm layer

Tooth surface

Underlying mineral

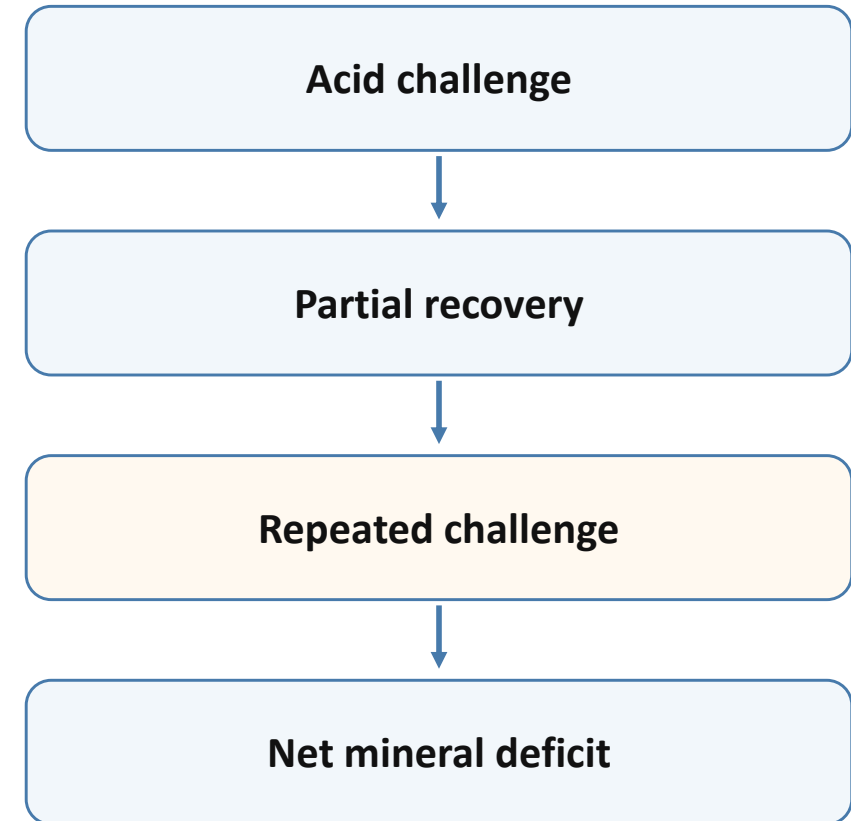
Dietary carbohydrates fuel acid generation

- Oral bacteria ferment sugars and starch-derived carbohydrates.
- Acids produced by fermentation contact the enamel surface.
- Frequent exposure can shift net mineral balance toward loss.
- Dietary counseling emphasizes the frequency of sugar exposure.



Repeated acid attacks are more important than one event

- Meals and sugary beverages initiate transient acid challenges.
- Between exposures, saliva and available fluoride support repair.
- Frequent snacks reduce intervals available for remineralization.
- Caries therefore represents cumulative imbalance over time.



Focal acid production explains focal tissue injury

- Fermentation products are concentrated where biofilm persists.
- Caries need not affect the entire tooth surface uniformly.
- Anatomical grooves and difficult-to-clean contacts retain plaque.
- Site-specific changes are assessed during dental examination.

Pits and fissures

Approximal contact

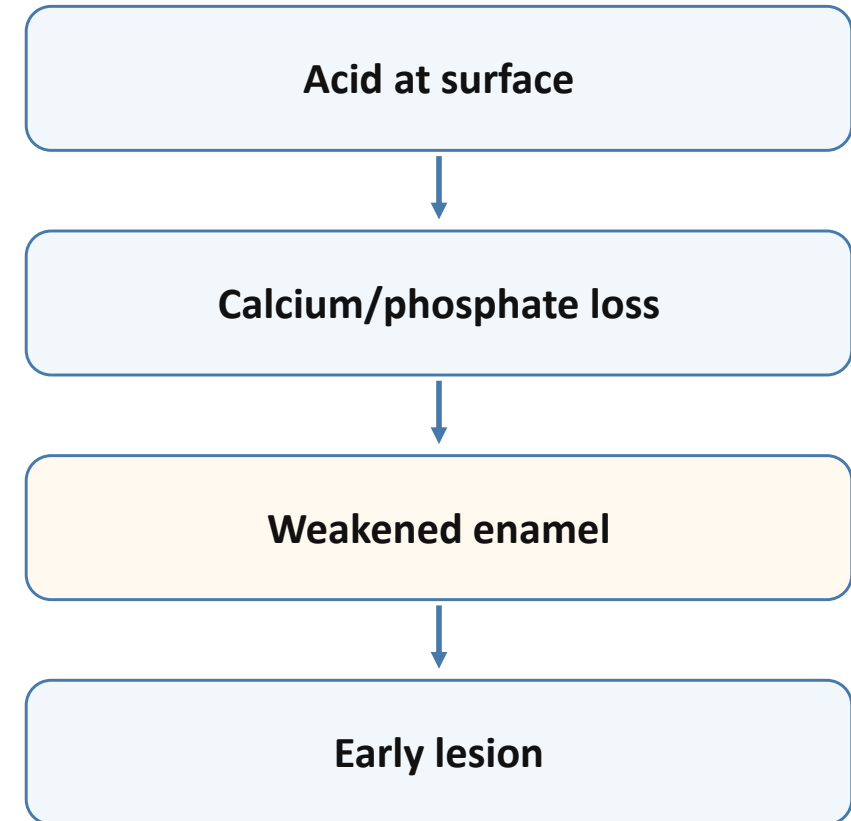
Smooth enamel

Exposed root

Site affects plaque retention

Demineralization removes mineral from enamel

- Microbial acids dissolve mineral from the tooth surface.
- Enamel progressively weakens when mineral loss is sustained.
- Early lesions can exist without a visible hole.
- Clinical appearance and actual mineral loss are not interchangeable.



Remineralization is biologically plausible before cavitation

- Saliva supplies mineral ions, including calcium and phosphate.
- Fluoride helps replace lost mineral and protect enamel.
- Early noncavitated lesions may be arrested or reversed.
- Once a cavity forms, lost tooth architecture does not simply regrow.

Demineralization

Sugar exposure

Acidogenic biofilm

Frequent challenges

Protective factors

Saliva minerals

Fluoride

Plaque disruption

Net lesion activity

The caries balance is a clinical framework

- Pathogenic pressures: Biofilm, sugars and repeated acid exposure.
- Protective factors: Saliva, fluoride and effective cleaning.
- Net balance determines lesion progression, arrest or remineralization.
- Risk-directed care adjusts the modifiable side of this balance.

Risk factors

Sugar exposure

Acidogenic biofilm

Frequent challenges

Protective factors

Saliva minerals

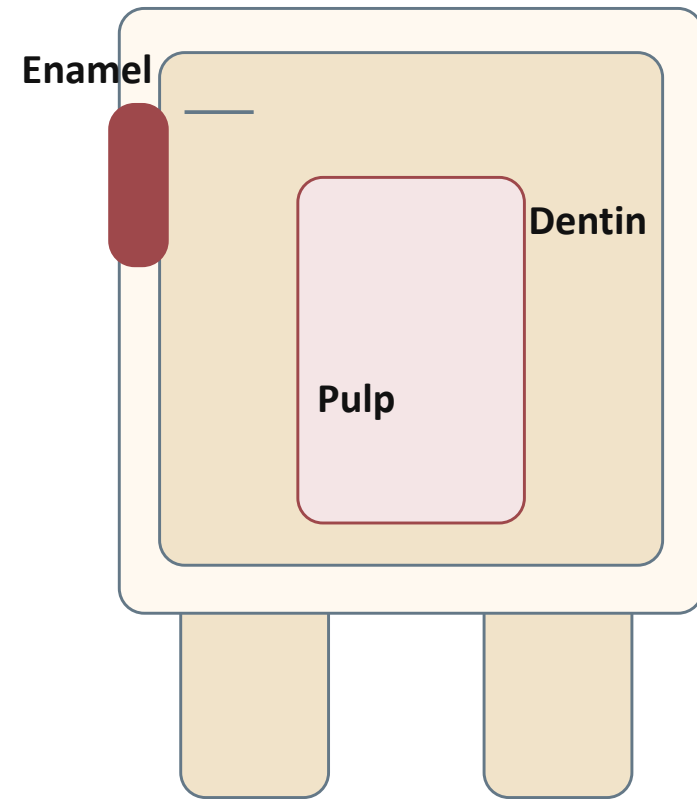
Fluoride

Plaque disruption

Net lesion activity

White-spot lesions signal early enamel injury

- White spots can reflect subsurface mineral loss.
- The tooth may remain noncavitated despite detectable disease.
- Recognition creates a window for nonrestorative measures.
- Activity and risk assessment guide monitoring.



Cavitation is a structural threshold

- Continued mineral loss weakens and breaks down enamel.
- A cavity is a permanent structural defect.
- Biofilm can persist inside a cavity that is hard to cleanse.
- The need for restoration depends on lesion and patient factors.

Intact enamel

White spot

Enamel breakdown

Cavity

Disease progression is a continuum—not two categories

- Subclinical mineral changes may precede visible signs.
- Initial lesions can have an intact outer surface.
- Moderate lesions may show localized enamel breakdown.
- Extensive lesions exhibit overt cavitation and greater depth.

0: Sound

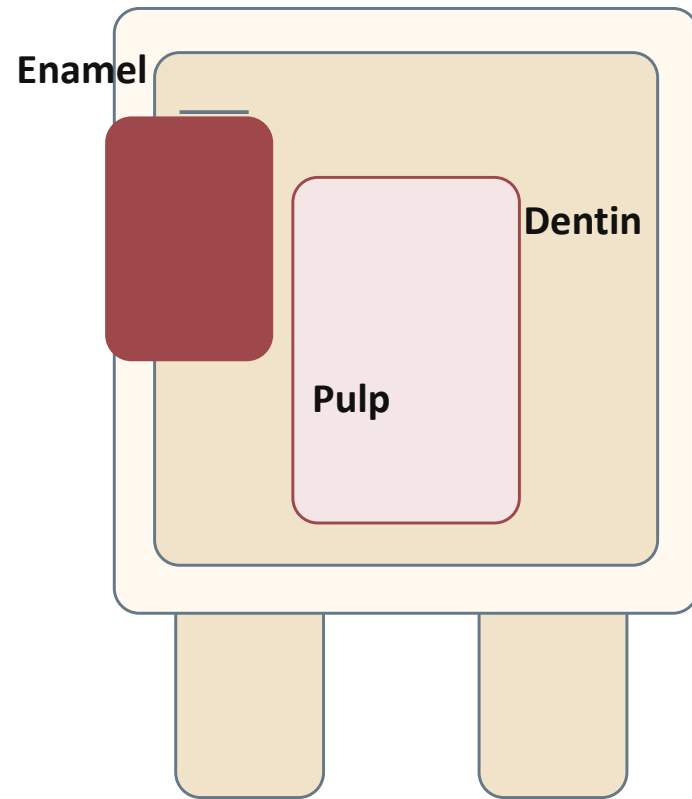
1–2: Initial enamel

3–4: Moderate

5–6: Extensive

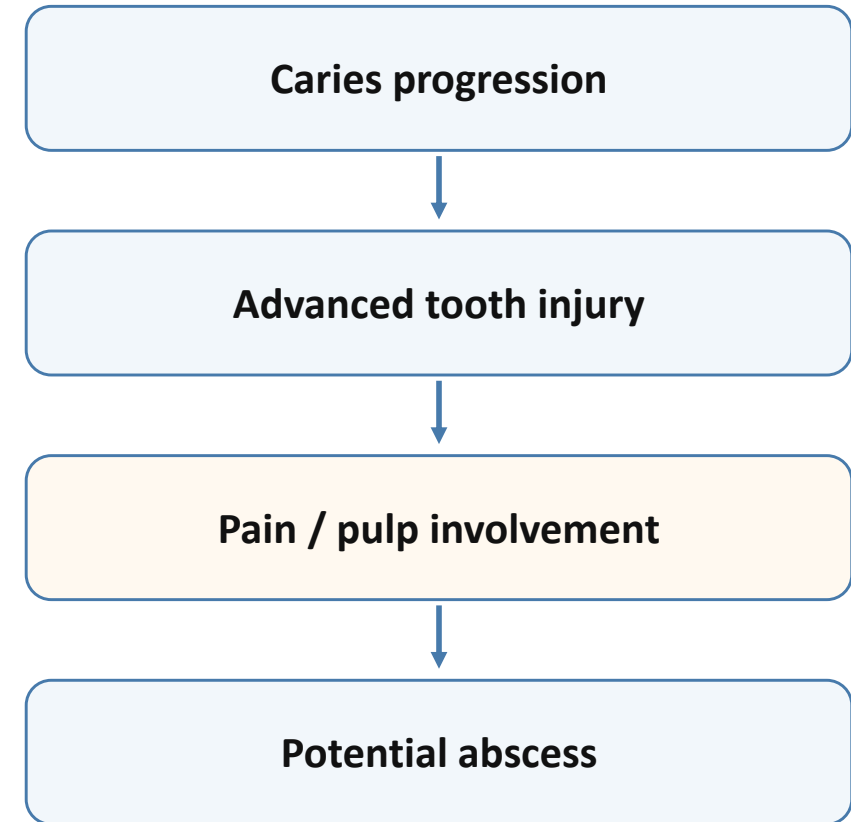
Caries may extend into dentin

- Dentin involvement indicates that disease has spread beyond enamel.
- A lesion can involve dentin even when the surface is not widely cavitated.
- Extent is evaluated using complementary examination methods.
- Clinical decisions should not be based solely on a dark surface color.



Pulpal disease is a possible downstream complication

- Progressive destruction reduces separation from the dental pulp.
- Toothache may develop as decay advances.
- Pulp infection can culminate in a localized dental abscess.
- Early detection helps avoid these later consequences.



Unmanaged caries affects function and quality of life

- Cavities may cause pain or sensitivity to foods and temperature.
- Advanced disease can produce infection and tooth loss.
- Symptoms may be absent in the earliest stages.
- Prevention and detection should not wait for pain.

Clinicopathologic link

Mechanism

Visible or functional result

Clinical response

Pits and fissures create anatomically vulnerable surfaces

- Deep grooves on the chewing surfaces of molars can retain plaque.
- Toothbrush bristles may not adequately reach some fissures.
- Retained fermentable material supports local acid production.
- Sealants provide a physical barrier at selected occlusal sites.

Pits and fissures

Approximal contact

Smooth enamel

Exposed root

Site affects plaque retention

Approximal surfaces present detection challenges

- Contacting surfaces can hide early lesions from direct vision.
- Plaque may remain between teeth without interdental cleaning.
- Bitewing radiographs can help detect approximal disease.
- Diagnostic sensitivity depends on lesion stage.

Pits and fissures

Approximal contact

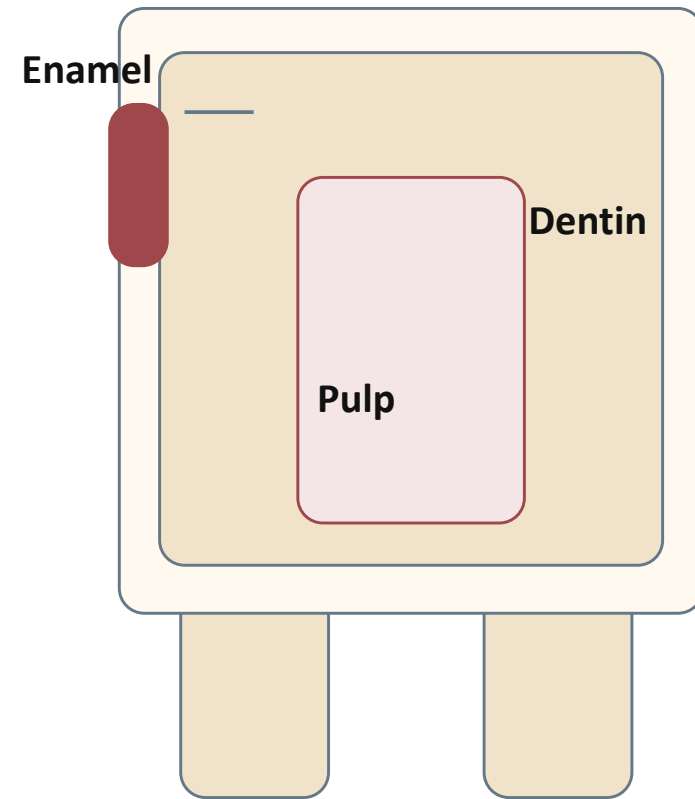
Smooth enamel

Exposed root

Site affects plaque retention

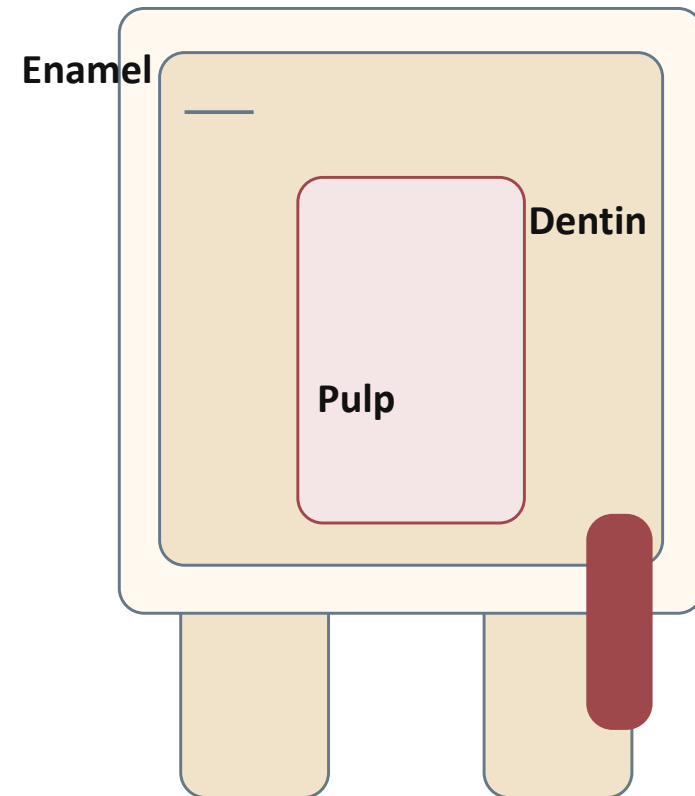
Smooth-surface lesions can indicate sustained risk

- Biofilm-derived acid can damage accessible enamel surfaces.
- Early enamel change may be visible before a cavity appears.
- In young children, anterior smooth-surface decay is a warning sign.
- Effective plaque removal and fluoride target the process.



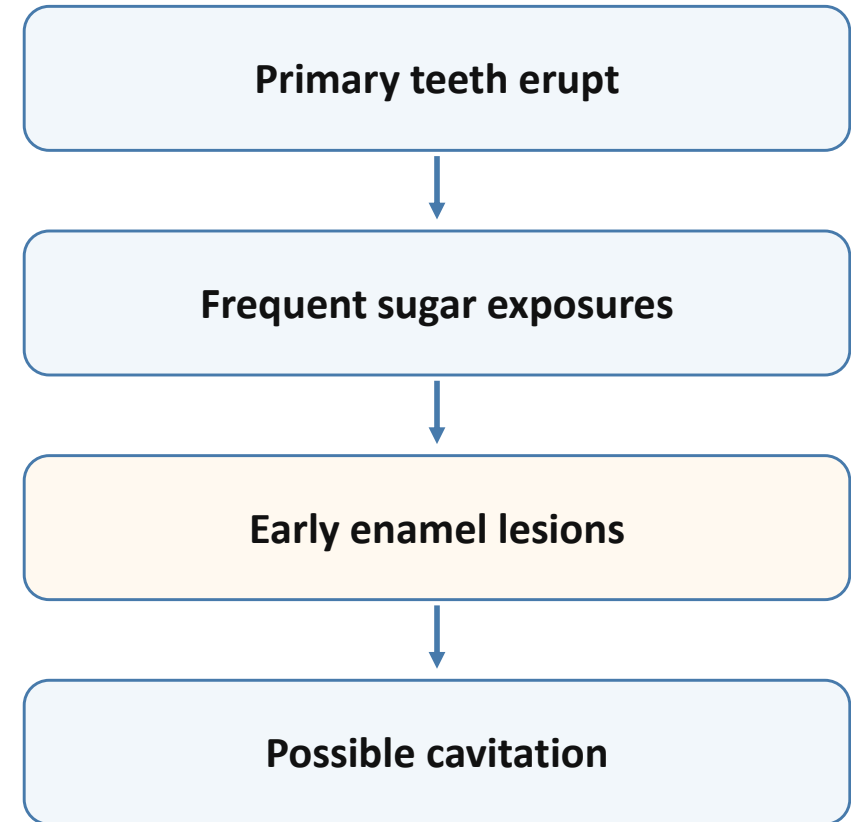
Exposed roots are vulnerable in older adults

- Gingival recession exposes root surfaces to the oral environment.
- Caries may then involve surfaces that were previously protected.
- Older age alone is not the mechanism; Exposure and risk factors matter.
- Prevention includes fluoride and management of dry mouth.



Early childhood caries has a distinct risk context

- Caries can begin as soon as primary teeth erupt.
- Frequent prolonged exposure to sugary drinks raises risk.
- Maxillary anterior teeth may be affected early.
- Caregiver-supported brushing and early dental contact are preventive.



Older patients may have a different surface at risk

- Gum recession permits biofilm to contact root surfaces.
- Reduced saliva can increase the likelihood of progression.
- Caries surveillance remains relevant throughout adulthood.
- Risk assessment should consider dexterity, access and existing lesions.

Risk factors

Sugar exposure

Acidogenic biofilm

Frequent challenges

Protective factors

Saliva minerals

Fluoride

Plaque disruption

Net lesion activity

Saliva is a major protective factor

- Saliva contributes calcium and phosphate for enamel repair.
- Salivary flow and composition affect the caries balance.
- Low salivary function reduces protection against acid challenges.
- Caries risk evaluation should include signs of dry mouth.

Demineralization

Sugar exposure

Acidogenic biofilm

Frequent challenges

Protective factors

Saliva minerals

Fluoride

Plaque disruption

Net lesion activity

Xerostomia changes the risk profile

- Severe dry mouth is listed by ADA among high-risk characteristics.
- Reduced salivary protection can impair remineralization.
- Clinical history should consider contributing therapies and illness.
- Preventive intensity should reflect the patient's individualized risk.

Risk factors

Sugar exposure

Acidogenic biofilm

Frequent challenges

Protective factors

Saliva minerals

Fluoride

Plaque disruption

Net lesion activity

Dietary exposure is modifiable

- Frequent sugary snacks prolong opportunities for acid production.
- Sweetened drinks and carbohydrate-rich foods can contribute.
- Reducing between-meal sugar frequency protects remineralization time.
- Diet advice works with—not instead of—fluoride and hygiene.

Clinicopathologic link

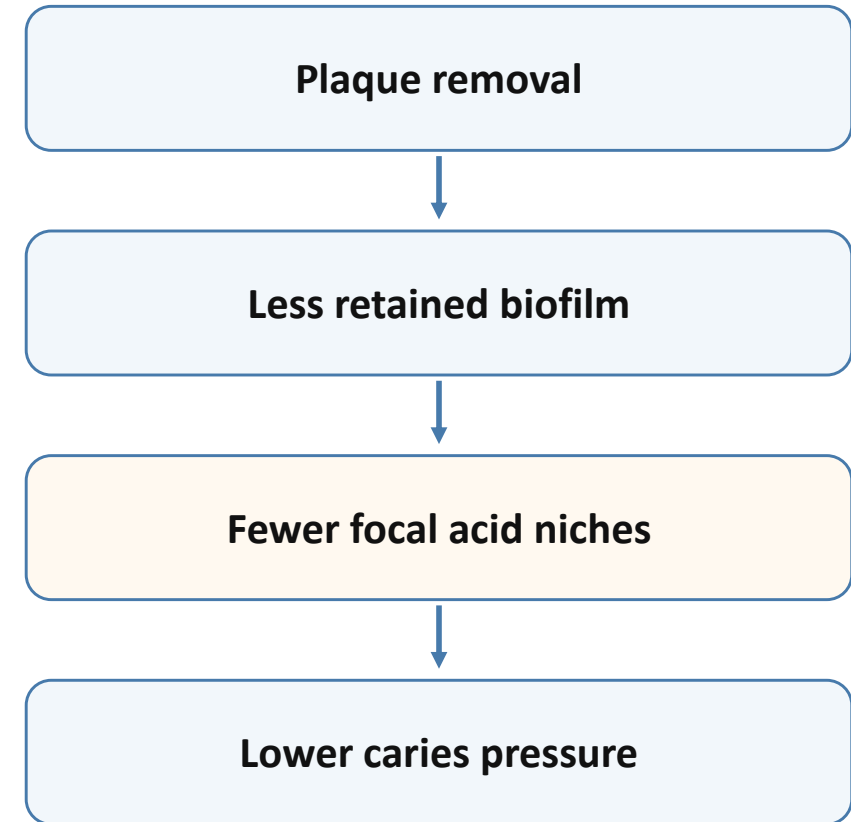
Mechanism

Visible or functional result

Clinical response

Oral hygiene targets the biofilm component

- Twice-daily brushing with fluoride toothpaste is recommended.
- Interdental cleaning addresses plaque between teeth.
- Dental visits can identify persistent plaque and early lesions.
- Plaque control is also relevant to gingival inflammation.



Fluoride increases resistance to acid injury

- Acid-resistant fluoroapatite with hydroxyapatite.
- Topical fluoride supports remineralization of early lesions.
- Fluoride can also reduce bacterial acid production.
- Protection is maintained through consistent appropriate exposure.

Demineralization

Sugar exposure

Acidogenic biofilm

Frequent challenges

Protective factors

Saliva minerals

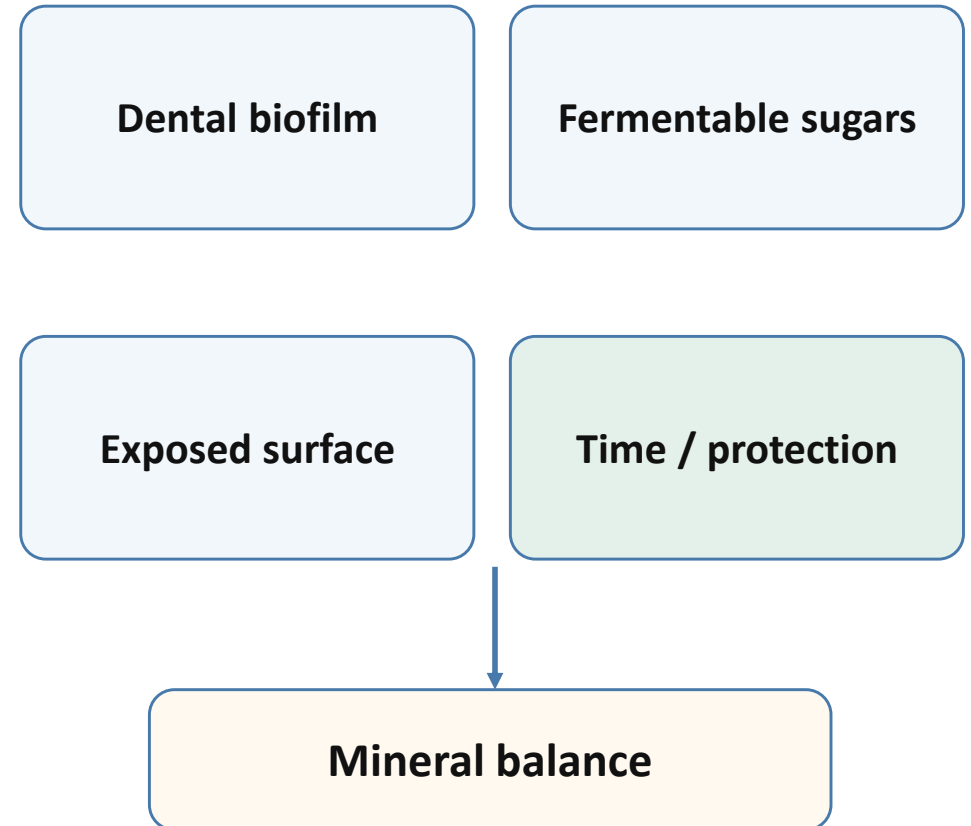
Fluoride

Plaque disruption

Net lesion activity

Fluoride can be delivered by several routes

- Fluoride toothpaste is a routine home-based intervention.
- Fluoridated water provides a population-level preventive measure.
- Professionally applied varnish or gel may be indicated by risk.
- Selection and dose must follow clinical recommendations.



Sealants prevent plaque retention in vulnerable fissures

- A sealant is applied to the chewing surface of posterior teeth.
- Its barrier blocks access of food and microorganisms to deep grooves.
- Sealants can prevent and arrest selected noncavitated occlusal lesions.
- They complement rather than replace other risk control measures.

Pits and fissures

Approximal contact

Smooth enamel

Exposed root

Site affects plaque retention

Community prevention modifies population exposure

- Water fluoridation is associated with reduced caries occurrence.
- School dental sealant programs improve access to preventive care.
- Preventive strategies can reduce treatment disparities.
- Population measures complement individual dental care.

Clinicopathologic link

Mechanism

Visible or functional result

Clinical response

Caries burden is socially patterned

- Shifts in caries burden across resource settings.
- Dietary change can increase risk when preventive access is limited.
- Oral hygiene, fluoride availability and access affect the disease trajectory.
- Risk interpretation must consider environment, not only behavior.

Demineralization

Sugar exposure

Acidogenic biofilm

Frequent challenges

Protective factors

Saliva minerals

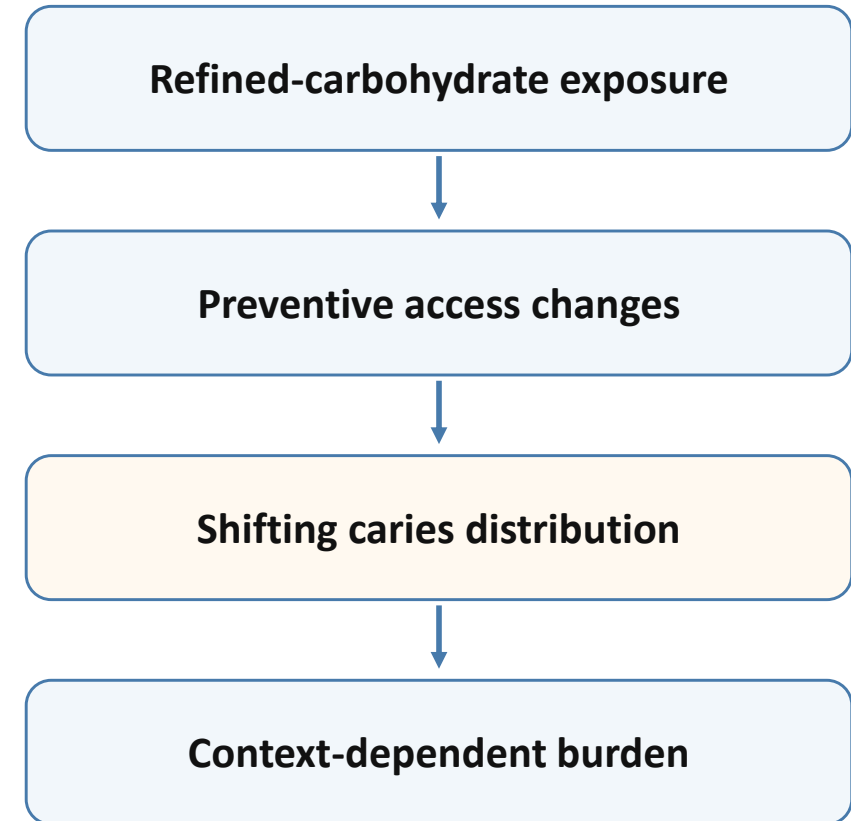
Fluoride

Plaque disruption

Net lesion activity

Historical geography reflects prevention and food environments

- Past higher occurrence in high-resource settings.
- Improved hygiene and water fluoridation contributed to reductions.
- Processed-food consumption is a concern in transitioning food environments.
- These are contextual textbook observations, not current country rankings.



Patient risk differs from lesion severity

Key distinguishing features

Patient-level risk	Tooth-level lesion
Future susceptibility	Current tissue state
Exposure and protection	Depth, integrity, activity
Guides prevention intensity	Guides lesion-specific care

A caries history is itself an informative risk signal

- Previous cavitated or initial lesions contribute to risk assessment.
- Past restorations and tooth loss due to caries may be relevant.
- Current dietary habits and salivary status refine the estimate.
- Formal tools organize information but do not replace judgment.

Clinicopathologic link

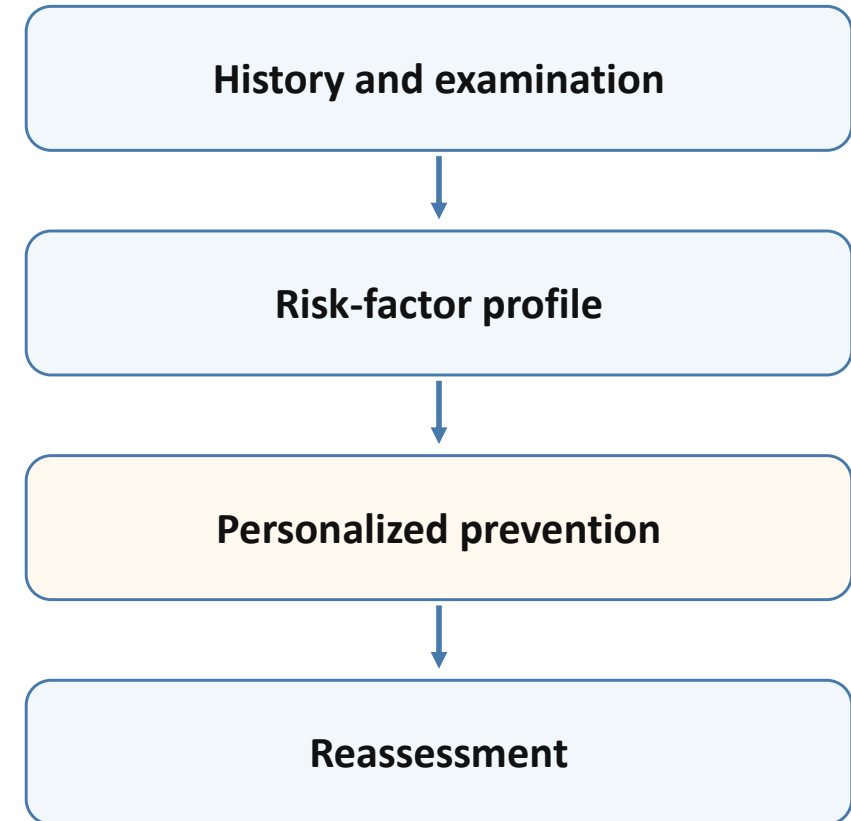
Mechanism

Visible or functional result

Clinical response

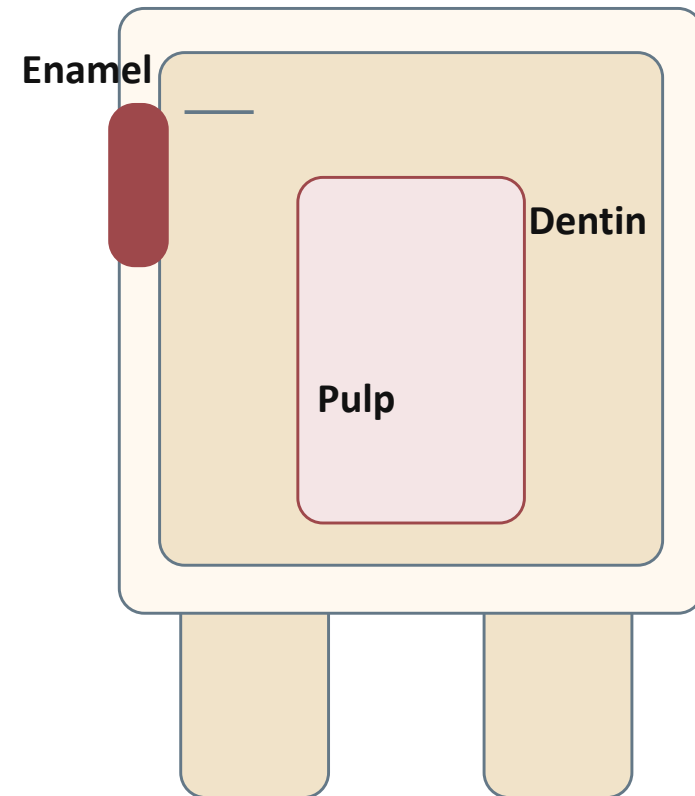
Risk assessment identifies modifiable mechanisms

- Review exposure frequency to sugary foods and drinks.
- Assess fluoride use and oral hygiene practice.
- Review dry mouth, previous caries and existing restorations.
- Link each risk factor to a feasible preventive intervention.



Visual examination detects surface changes

- White, brown or black changes may prompt further evaluation.
- A visible hole strongly suggests a cavitated lesion.
- An apparently intact surface does not exclude early mineral loss.
- Clean, dry examination improves assessment of subtle lesions.



Probing must not damage fragile enamel

- Surface texture and dentin softening can provide adjunctive information.
- Forceful sharp probing of an initial lesion can damage its surface.
- ADA discusses gentle rounded or ball-ended examination approaches.
- Use inspection, clinical context and appropriate imaging together.

Clinicopathologic link

Mechanism

Visible or functional result

Clinical response

Bitewing radiographs complement direct inspection

- Approximal surfaces are difficult to examine visually.
- Bitewings can reveal lesions in contacting tooth surfaces.
- Detection performance varies with lesion extent.
- Interpret imaging alongside clinical findings; No single test is definitive.

Pits and fissures

Approximal contact

Smooth enamel

Exposed root

Site affects plaque retention

ICDAS describes increasing visual lesion severity

- Score 0 represents a clinically sound surface.
- Scores 1–2 represent initial intact-enamel changes.
- Scores 3–4 represent moderate visible breakdown or underlying changes.
- Scores 5–6 represent extensive clinically detectable cavitation.

0: Sound

1–2: Initial enamel

3–4: Moderate

5–6: Extensive

Activity assessment informs whether a lesion is progressing

- Lesion extent and activity are different dimensions.
- Monitoring compares observations over time.
- Biofilm access and the patient's protective factors influence activity.
- The goal is to recognize lesions that may respond to nonrestorative care.

Demineralization

Sugar exposure

Acidogenic biofilm

Frequent challenges

Protective factors

Saliva minerals

Fluoride

Plaque disruption

Net lesion activity

Noncavitated and cavitated lesions are not equivalent

Key distinguishing features

Noncavitated	Cavitated
Surface may remain intact	Structural defect present
Remineralization possible	Lost architecture not regenerated
Prevention and follow-up	May need restoration plus risk control

Not every discolored fissure is a cavity

Key distinguishing features

Color change	Caries diagnosis
Visual observation	Disease interpretation
Can reflect stain or lesion	Uses morphology and risk
Insufficient in isolation	May need imaging or follow-up

Erosion has a different causal mechanism

Key distinguishing features

Dental caries	Dental erosion
Bacterial acid from carbohydrates	Nonbacterial chemical acid
Often plaque-retentive sites	Exposure-related distribution
Biofilm and diet management	Address acid exposures

Abrasion is mechanical—not microbiological—wear

Key distinguishing features

Caries	Abrasion
Biofilm-driven mineral loss	Mechanical tissue wear
May progress to cavitation	Wear pattern linked to friction
Sugar/biofilm/fluoride focus	Identify mechanical cause

Gingivitis principally affects marginal gingiva

Key distinguishing features

Dental caries	Gingivitis
Enamel and dentin	Gingival soft tissue
Acid demineralization	Plaque-associated inflammation
White spot or cavity	Redness, edema, bleeding

Periodontitis destroys tooth-supporting structures

Key distinguishing features

Caries	Periodontitis
Tooth mineralized tissues	Periodontal ligament / alveolar bone
Acid-mediated injury	Chronic destructive inflammation
Cavitation and pulp risk	Attachment loss and mobility

Three common plaque-related disease processes differ

- Caries is tissue demineralization; Gingivitis is gingival inflammation.
- Periodontitis destroys periodontal supporting tissues.
- A shared plaque environment does not make their lesions synonymous.
- Diagnosis identifies the precise tissue involved.

Caries

Enamel / dentin mineral loss

Gingivitis

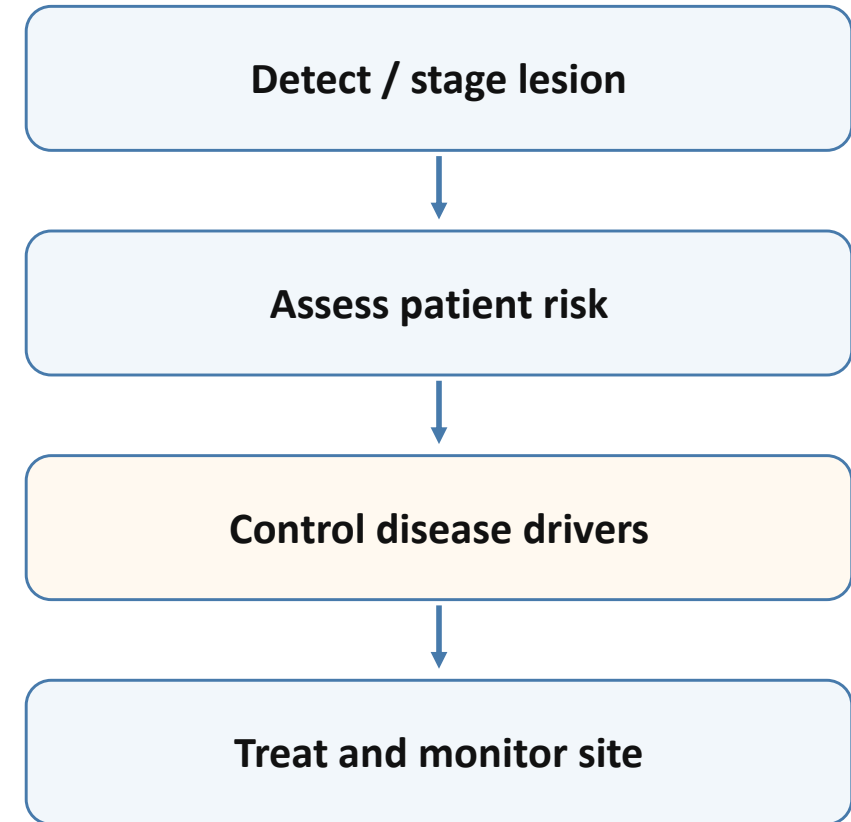
Gingival inflammation

Periodontitis

Supporting-tissue destruction

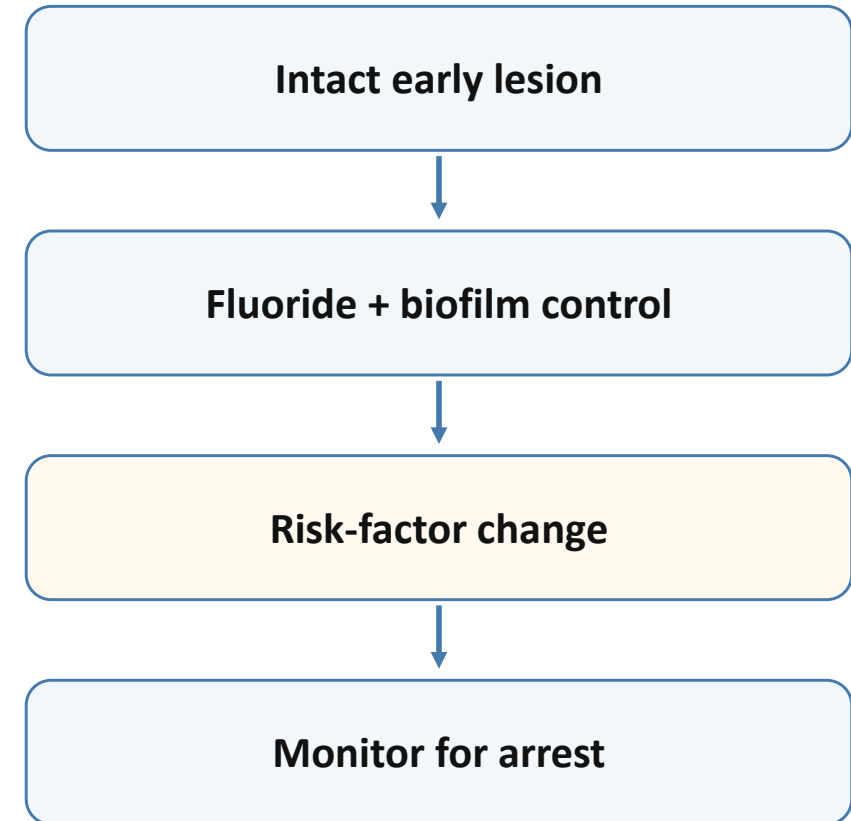
Modern care treats disease activity as well as defects

- Restoration alone does not remove the causal exposures.
- Risk assessment, fluoride and cleaning address the disease process.
- Lesion-specific procedures are added when structurally indicated.
- Follow-up determines whether management has altered progression.



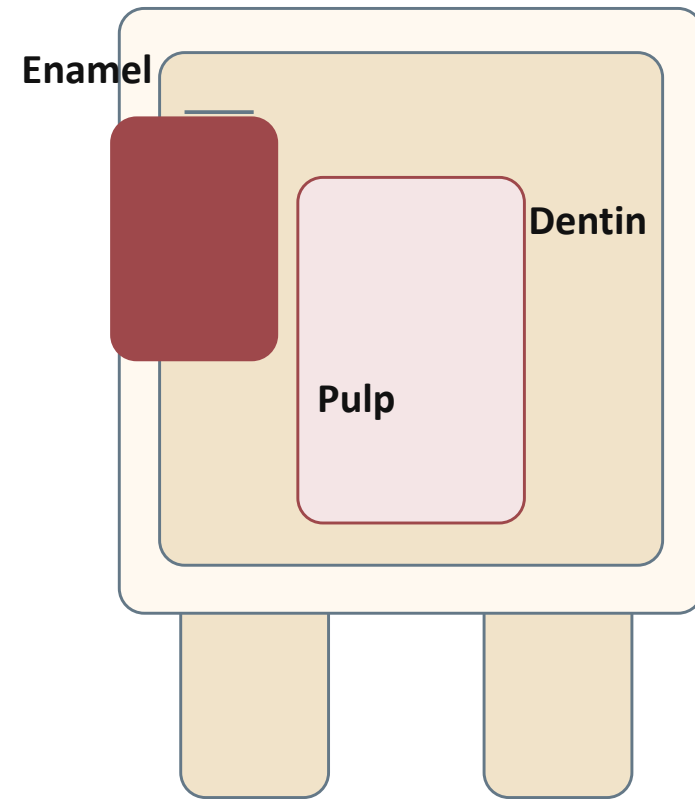
Initial lesions are candidates for nonrestorative care

- Early enamel lesions may respond to fluoride-based treatment.
- Diet and plaque management reduce repeated acid challenges.
- Sealants can arrest selected noncavitated pit-and-fissure lesions.
- Periodic assessment is necessary to establish arrest.



Restoration repairs a structural defect

- Cavitated tissue defects may require operative restoration.
- A filling replaces tissue after removal of affected material.
- Treatment selection depends on depth, site and restorative feasibility.
- Prevention remains essential because a filling does not cure susceptibility.



Silver diamine fluoride has a defined nonrestorative role

- ADA's 2018 guideline includes silver diamine fluoride as an option.
- Use depends on lesion type, dentition and clinical circumstances.
- It is not a universal replacement for definitive dental assessment.
- Recommendations should be interpreted from the original guideline.

Clinicopathologic link

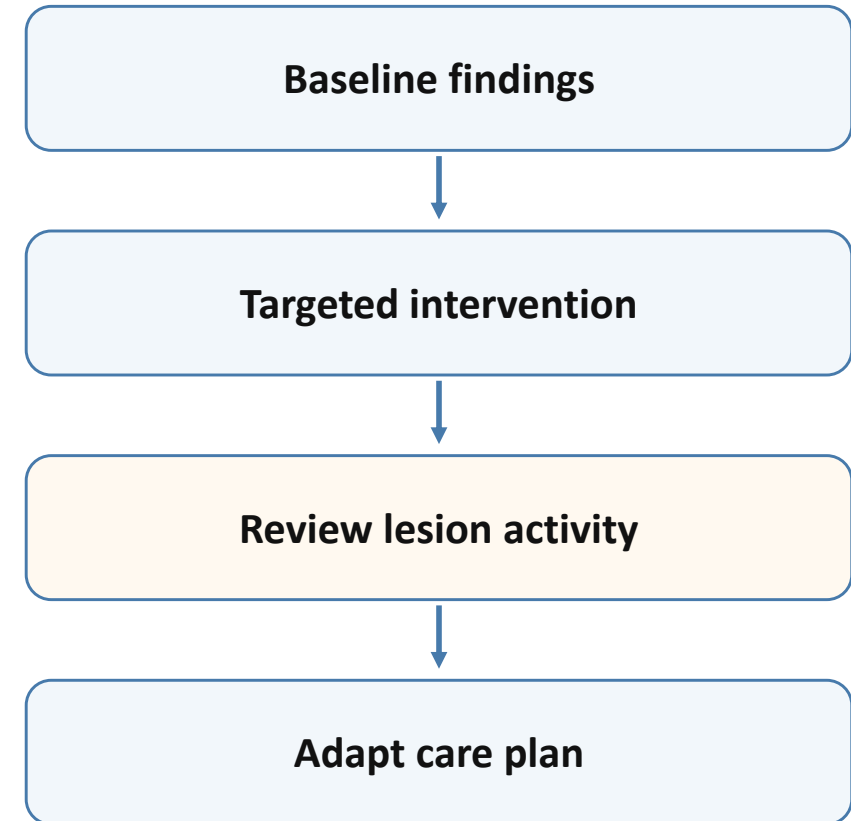
Mechanism

Visible or functional result

Clinical response

Monitoring tests whether prevention is effective

- Document lesion site and presentation at baseline.
- Reassess behavior, fluoride exposure and plaque control.
- Observe whether lesions remain stable or show progression.
- Escalate lesion-specific care when structural risk warrants it.



Case: A child with a new white spot

- Illustrative case: White opaque area on a primary incisor; No visible cavity.
- Likely pathology: Early enamel demineralization from repeated acid exposure.
- Priority: Evaluate diet, fluoride use, plaque and overall caries risk.
- Teaching point: Early caries is not synonymous with a hole.

Clinical reasoning

Identify lesion stage

Assess causal exposures

Select appropriate next step

Case: Dark fissure on an asymptomatic molar

- Illustrative case: Dark occlusal groove found at routine examination.
- Color alone cannot establish a cavitated active lesion.
- Assess integrity, adjacent plaque, risk and indicated imaging.
- Teaching point: Distinguish observation from diagnosis.

Clinical reasoning

Identify lesion stage

Assess causal exposures

Select appropriate next step

Case: Older adult with recurrent root lesions

- Illustrative case: Gingival recession, dry mouth and new cervical defects.
- Exposed root surface and reduced saliva increase caries vulnerability.
- Review saliva-related history and protective measures.
- Teaching point: Risk extends across the lifespan and tooth surfaces.

Clinical reasoning

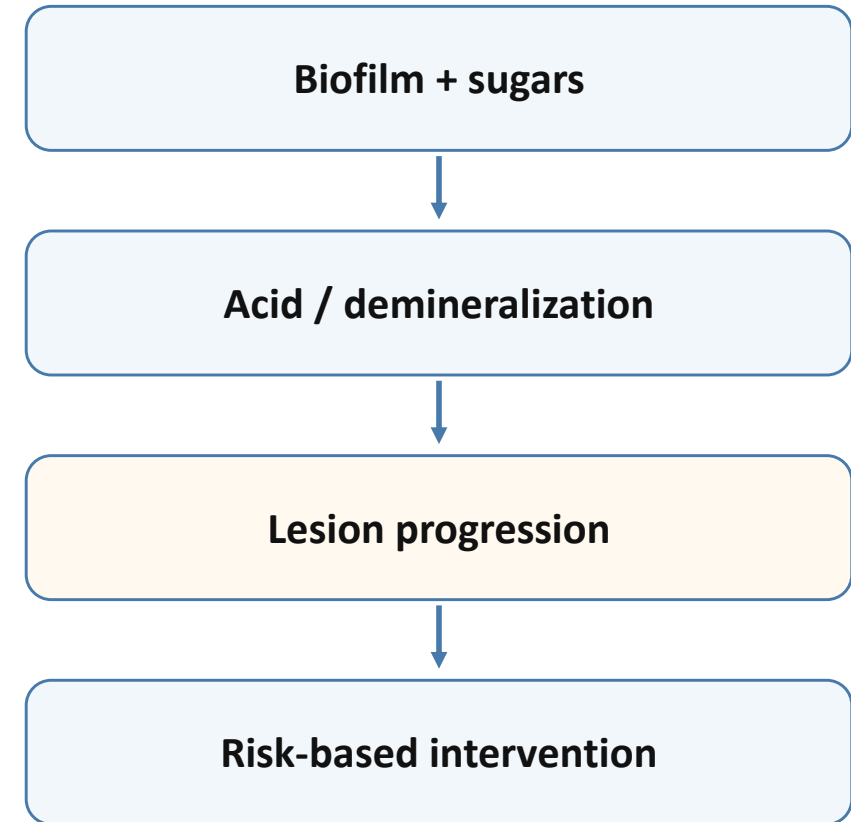
Identify lesion stage

Assess causal exposures

Select appropriate next step

Pathogenesis-to-management synthesis

- Biofilm plus fermentable carbohydrate generates focal acid exposure.
- Sustained net mineral loss proceeds from early lesions to cavitation.
- Anatomy, saliva, fluoride and behavior modify lesion course.
- Preventive and restorative care must target both risk and observed damage.



Reference framework for this lecture

- Primary: Robbins, Cotran & Kumar, Pathologic Basis of Disease, 11th ed. (2026), Ch. 16, pp. 664–665.
- NIH/NIDCR: Tooth Decay and The Tooth Decay Process (reviewed 2025).
- American Dental Association: Caries Risk Assessment and Management; caries management guidelines (2018, 2023).
- CDC: About Cavities, Fluoride, and Dental Sealants (2024 web resources).

Textbook foundation

NIH / NIDCR

ADA guidelines

CDC public health