Reading the Host Response

A New Look at Oral Disease, Microbiome Balance and the Biology of Healing

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Our purpose

When bleeding, dryness, or recurring decay persists, ask what sustains the disease and what would change the plan. This course connects microbial patterns, saliva, host response, and medical history with individualized prevention and measurable outcomes. Local treatment and host assessment work together.

Learning goals

- Explain how biofilm, saliva, immunity, and systemic health interact in oral disease.
- Recognize microscopy patterns and distinguish visual observations from species identification.
- Choose saliva, microbial, and inflammatory tests that answer a clinical question.
- Connect medical history, breathing, sleep, nutrition, and metabolic factors with oral findings.
- Use patient education, indicated treatment, and reassessment to personalize care.

The biological dental health pathway

Step	Question that guides care
Screen	What risks and warning signs are present?
Investigate	What changed, and when did symptoms begin?
Test	Which information would change our clinical decision?
Treat	What local disease and ecological pressures need attention?
Support	What barriers to healing require coaching or collaboration?
Reassess	What improved, what persists, and what should change?

Test–Teach–Treat–Repeat is Barbara K. Tritz's educational framework. "Disease is communication, not disobedience." Use curiosity to build partnership and reduce blame.

1 Read the biofilm and the host response

Phase-contrast microscopy makes living biofilm and cellular patterns visible. Use it to support education and investigation alongside periodontal charting, radiographs, clinical examination, and risk assessment. Morphology and movement alone do not reliably identify a species or establish disease activity. [1, 29]

Observation	Clinical interpretation and next question
Cocci and a relatively calm field	May be compatible with health. A calm sample does not exclude disease at other sites.
Motile rods or spirochete-like forms	Consider a disease-associated pattern. Which sites bleed, and is attachment stable?
Flagellated or amoeboid forms	Consider protozoa. Confirm identity when it would change management; avoid species certainty from appearance alone.
Yeast or hyphae-like structures	Review symptoms, salivary function, medications, dentures, and glucose status. Detection alone does not diagnose candidiasis.
White blood cells	Evidence of a host response. A routine slide observation is not a standardized oral neutrophil count. [17]
Dense or complex biofilm	Review sample site, collection, timing, and clinical context before comparing with a prior sample.

Microbes interact with one another

Laboratory work shows metabolic cooperation between P. gingivalis and T. denticola. Candida albicans and S. mutans can cooperate in experimental cariogenic biofilms. These findings explain possible mechanisms; they do not establish the cause of an individual patient's disease. [10, 25]

T. tenax is more frequently detected in periodontitis, but its causal role and optimal management remain incompletely established. E. gingivalis research combines clinical detection with experimental tissue effects. Assess the whole clinical picture rather than treating a visual finding in isolation. [2, 3]

The host is part of the picture

Periodontitis reflects dysbiosis interacting with an inflammatory host response. Inflammation can persist when the microbial challenge and modifying risks remain. Resolution is an active biological process. Low oxygen in periodontal pockets and hypoxia-related tissue signaling add context; visible redness does not measure effective tissue oxygen delivery. [12, 20, 22, 26]

The course's green, yellow, orange, and red zones are a teaching aid, not a validated diagnostic scale or automatic treatment threshold. Treatment decisions follow the clinical diagnosis.

Notes and clinical connections

2 Assess saliva and the whole patient

Saliva quantity and quality

Saliva provides clearance, lubrication, buffering, antimicrobial components, and calcium and phosphate for remineralization. Xerostomia describes oral dryness; hyposalivation describes measured low flow. A patient's discomfort is real even when a flow result is within the normal range. [7]

- Record resting and stimulated flow when indicated. Standardize collection time, eating and drinking restrictions, and measurement method.
- Measure pH and buffering separately: pH is the current acid–base state; buffering is the ability to resist and recover from an acid challenge.
- Ask about frequent carbohydrate intake, acidic drinks, reflux symptoms, medication changes, and new root-surface exposure.
- Review nighttime dryness, mouth breathing, appliances, and bedtime products. Reduced nighttime flow makes saliva-dependent protection more limited. [6, 7]

Ask for the pH of the current dry-mouth product formula. Laboratory studies show variation in erosive potential; pH alone does not establish clinical tooth damage. Consider mineral content, formulation, and exposure pattern. [8]

Choose the test to match the question

Tool	Contribution and limitation
Microscopy	Shows visual patterns and motion; cannot reliably name or quantify individual species.
Molecular microbial tests	Detect targets included in the panel; results depend on assay, sample site, and collection. Detection does not prove causation.
Inflammatory markers such as aMMP-8	May add information about inflammation and tissue breakdown; interpret the specific assay with clinical findings.
Charting and radiographs	Establish periodontal status, structural changes, and comparison with prior findings.
Medical evaluation and laboratory collaboration	Answer a targeted question about a possible barrier to healing; avoid indiscriminate testing.

Listen for the change behind the symptom

Ask about medications and supplements; diabetes, prediabetes, and the latest HbA1c; pregnancy or hormonal transitions; sleep and nasal obstruction; diet and tobacco; stress and practical barriers. When indicated, coordinate review of CBC, metabolic panel, iron status, B12/folate, vitamin D, or inflammatory markers with the medical team. Ordering and interpretation depend on scope, indication, and clinical context. [11, 16, 21, 23]

One question I will add to my patient history

3 Teach skills and personalize treatment

Make the biology understandable

Show the finding, explain its meaning and limits, and connect it to one achievable next step. Ask the patient to demonstrate the skill and explain the plan back in their own words. “What gets in the way?” often reveals more than repeating instructions.

Daily step	Coaching focus
Reveal	Use disclosing to identify plaque the patient can see and reach.
Brush	Demonstrate gentle placement and systematic coverage with a suitable manual or powered brush.
Clean between	Match interdental brushes, floss, or another tool to anatomy, dexterity, and preference.
Irrigate when helpful	Use as an adjunct for difficult anatomy, implants, or bridges. Choose device-compatible solutions as directed.
Support the environment	Address dryness, frequent sugar or acid exposures, and the remineralization plan.
Review and coach again	Check feasibility, technique, symptoms, and tissue response at follow-up.

Evidence supports daily mechanical plaque control. Powered brushes can improve plaque and gingivitis outcomes compared with manual brushes; this evidence does not establish that every design or brand is equivalent. Interdental-device evidence varies by tool and outcome. [5, 27, 28]

Local care and host support proceed together

Base treatment on the diagnosis and patient needs. Use appropriate biofilm removal and instrumentation, including air polishing, ultrasonics, and hand instruments where indicated. Review tobacco, glucose regulation, nutritional adequacy, hydration, and relevant medication effects. Refer for persistent nasal obstruction or suspected sleep-disordered breathing. [11, 23, 24]

Moisturizing products lubricate tissues; salivary stimulants encourage secretion when gland function remains. They serve different purposes. Select the approach using symptoms, measurements, and the cause of dryness. [7]

Adjuncts need a defined purpose

The presentation discusses topical agents, medicament trays, ozone approaches, and oral probiotics. Evidence, formulation, delivery, and indications differ. Select each adjunct for a specific clinical need within professional scope; document the intended outcome and reassess. An in vitro antimicrobial result does not establish improved patient outcomes.

Probiotic benefits are strain- and condition-specific. A trial of B. animalis subsp. lactis HN019 as an adjunct to periodontal therapy cannot support claims for all oral probiotics. Match the product to the study and monitor clinical response. [15, 24]

A skill or treatment change I will bring to practice

4 Reassess and protect the gains

Compare follow-up findings with the documented baseline. The goal is improved clinical health, comfort, and function. Persistent disease calls for revised care or specialist assessment. A quiet microscope field or one negative test cannot establish stability on its own. [4, 18, 24]

Measure	Baseline	Follow-up
Bleeding on probing and suppuration	_____	_____
Pocket depth and attachment findings	_____	_____
Plaque control and demonstrated technique	_____	_____
Dryness, pain, comfort, and function	_____	_____
Saliva or microbial findings when indicated	_____	_____
Identified host risks and referral progress	_____	_____

When improvement stalls

- Confirm the diagnosis, sampling method, adherence, and whether local irritants remain.
- Revisit saliva, nighttime breathing, medications, glucose control, and relevant nutritional or hormonal clues.
- Review whether the plan is realistic for the patient. Reteach, refine, and arrange appropriate referrals.
- Set the next reassessment and supportive-care interval according to risk and clinical response.

Oral systemic findings need careful interpretation

Periodontitis is associated with systemic inflammatory conditions, with plausible biological links. Association does not establish individual causation. Alzheimer's research involving P. gingivalis and a reported oral F. nucleatum stillbirth case illustrate research directions; neither predicts an individual patient's outcome. [9, 13, 14]

My next patient plan

The finding I need to explain: _____

The information that could change care: _____

One next step agreed with the patient: _____

How and when I will measure response: _____

Thank you

Thank you to Philips Sonicare for sponsoring this presentation, and to each attendee for continuing to ask better questions. Keep investigating, teaching, and measuring the response. Together, we can elevate prevention and help patients participate in their care.

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References

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Alphabetized references and further reading. Bracketed numbers in the handout correspond to this list. Study designs range from laboratory studies and case reports to reviews, trials, and clinical guidance.

1. Armitage GC, Dickinson WR, Jenderseck RS, Levine SM, Chambers DW. Relationship between the percentage of subgingival spirochetes and the severity of periodontal disease. Journal of Periodontology. 1982;53(9):550–556.
https://pubmed.ncbi.nlm.nih.gov/6957592/

2. Bao X, Wiehe R, Dommisch H, Schaefer AS. Entamoeba gingivalis causes oral inflammation and tissue destruction. Journal of Dental Research. 2020;99(5):561–567.
https://doi.org/10.1177/0022034520901738

3. Bisson C, Dridi SM, Machouart M. Assessment of the role of Trichomonas tenax in the etiopathogenesis of human periodontitis: A systematic review. PLoS ONE. 2019;14(12):e0226266.
https://doi.org/10.1371/journal.pone.0226266

4. Caton JG, Armitage G, Berglundh T, et al. A new classification scheme for periodontal and peri-implant diseases and conditions: Introduction and key changes from the 1999 classification. Journal of Clinical Periodontology. 2018;45(Suppl 20):S1–S8.
https://doi.org/10.1111/jcpe.12935

5. Chapple ILC, Van der Weijden F, Doerfer C, et al. Primary prevention of periodontitis: Managing gingivitis. Journal of Clinical Periodontology. 2015;42(Suppl 16):S71–S76.
https://doi.org/10.1111/jcpe.12366

6. Choi JE, Waddell JN, Lyons KM, Kieser JA. Intraoral pH and temperature during sleep with and without mouth breathing. Journal of Oral Rehabilitation. 2016;43(5):356–363.
https://doi.org/10.1111/joor.12372

7. Dawes C, Pedersen AML, Villa A, et al. The functions of human saliva: A review sponsored by the World Workshop on Oral Medicine VI. Archives of Oral Biology. 2015;60(6):863–874.
https://doi.org/10.1016/j.archoralbio.2015.03.004

8. Delgado AJ, Olafsson VG, Donovan TE. pH and erosive potential of commonly used oral moisturizers. Journal of Prosthodontics. 2016;25(1):39–43.
https://doi.org/10.1111/jopr.12324

9. Dominy SS, Lynch C, Ermini F, et al. Porphyromonas gingivalis in Alzheimer's disease brains: Evidence for disease causation and treatment with small-molecule inhibitors. Science Advances. 2019;5(1):eaau3333.
https://doi.org/10.1126/sciadv.aau3333

10. Falsetta ML, Klein MI, Colonne PM, et al. Symbiotic relationship between Streptococcus mutans and Candida albicans synergizes virulence of plaque biofilms in vivo. Infection and Immunity. 2014;82(5):1968–1981.
https://doi.org/10.1128/IAI.00087-14

References continued

11. Guo S, DiPietro LA. Factors affecting wound healing. Journal of Dental Research. 2010;89(3):219–229.
https://doi.org/10.1177/0022034509359125

12. Hajishengallis G. Periodontitis: From microbial immune subversion to systemic inflammation. Nature Reviews Immunology. 2015;15(1):30–44.
https://doi.org/10.1038/nri3785

13. Hajishengallis G, Chavakis T. Local and systemic mechanisms linking periodontal disease and inflammatory comorbidities. Nature Reviews Immunology. 2021;21:426–440.
https://doi.org/10.1038/s41577-020-00488-6

14. Han YW, Fardini Y, Chen C, et al. Term stillbirth caused by oral Fusobacterium nucleatum. Obstetrics and Gynecology. 2010;115(2 Pt 2):442–445.
https://doi.org/10.1097/AOG.0b013e3181cb9955

15. Invernici MM, Salvador SL, Silva PHF, et al. Effects of Bifidobacterium probiotic on the treatment of chronic periodontitis: A randomized clinical trial. Journal of Clinical Periodontology. 2018;45(10):1198–1210.
https://doi.org/10.1111/jcpe.12995

16. Irwin MR, Olmstead R, Carroll JE. Sleep disturbance, sleep duration, and inflammation: A systematic review and meta-analysis of cohort studies and experimental sleep deprivation. Biological Psychiatry. 2016;80(1):40–52.
https://doi.org/10.1016/j.biopsych.2015.05.014

17. Landzberg M, Doering H, Aboodi GM, Tenenbaum HC, Glogauer M. Quantifying oral inflammatory load: Oral neutrophil counts in periodontal health and disease. Journal of Periodontal Research. 2015;50(3):330–336.
https://doi.org/10.1111/jre.12211

18. Lang NP, Adler R, Joss A, Nyman S. Absence of bleeding on probing: An indicator of periodontal stability. Journal of Clinical Periodontology. 1990;17(10):714–721.
https://doi.org/10.1111/j.1600-051X.1990.tb01059.x

19. Marsh PD. Microbial ecology of dental plaque and its significance in health and disease. Advances in Dental Research. 1994;8(2):263–271.
https://doi.org/10.1177/08959374940080022001

20. Mettraux GR, Gusberti FA, Graf H. Oxygen tension (pO2) in untreated human periodontal pockets. Journal of Periodontology. 1984;55(9):516–521.
https://doi.org/10.1902/jop.1984.55.9.516

References continued

21. Murakami S, Mealey BL, Mariotti A, Chapple ILC. Dental plaque-induced gingival conditions. Journal of Clinical Periodontology. 2018;45(Suppl 20):S17–S27.
https://doi.org/10.1111/jcpe.12937

22. Ng KT, Li JP, Ng KM, Tipoe GL, Leung WK, Fung ML. Expression of hypoxia-inducible factor-1α in human periodontal tissue. Journal of Periodontology. 2011;82(1):136–141.
https://doi.org/10.1902/jop.2010.100100

23. Sanz M, Ceriello A, Buysschaert M, et al. Scientific evidence on the links between periodontal diseases and diabetes: Consensus report and guidelines of the joint workshop on periodontal diseases and diabetes by the International Diabetes Federation and the European Federation of Periodontology. Journal of Clinical Periodontology. 2018;45(2):138–149.
https://doi.org/10.1111/jcpe.12808

24. Sanz M, Herrera D, Kebschull M, et al. Treatment of stage I–III periodontitis: The EFP S3 level clinical practice guideline. Journal of Clinical Periodontology. 2020;47(Suppl 22):4–60.
https://doi.org/10.1111/jcpe.13290

25. Tan KH, Seers CA, Dashper SG, et al. Porphyromonas gingivalis and Treponema denticola exhibit metabolic symbioses. PLoS Pathogens. 2014;10(3):e1003955.
https://doi.org/10.1371/journal.ppat.1003955

26. Van Dyke TE, Serhan CN. Resolution of inflammation: A new paradigm for the pathogenesis of periodontal diseases. Journal of Dental Research. 2003;82(2):82–90.
https://doi.org/10.1177/154405910308200202

27. Worthington HV, MacDonald L, Poklepovic Pericic T, et al. Home use of interdental cleaning devices, in addition to toothbrushing, for preventing and controlling periodontal diseases and dental caries. Cochrane Database of Systematic Reviews. 2019;(4):CD012018.
https://doi.org/10.1002/14651858.CD012018.pub2

28. Yaacob M, Worthington HV, Deacon SA, et al. Powered versus manual toothbrushing for oral health. Cochrane Database of Systematic Reviews. 2014;(6):CD002281.
https://doi.org/10.1002/14651858.CD002281.pub3

29. Yousefi L, Leylabadlo HE, Pourlak T, et al. Oral spirochetes: Pathogenic mechanisms in periodontal disease. Microbial Pathogenesis. 2020;144:104193.
https://doi.org/10.1016/j.micpath.2020.104193

Framework attribution: Test–Teach–Treat–Repeat and the six-stage biological dental health pathway are teaching frameworks presented by Barbara K. Tritz. The microscopy color zones are educational descriptors, not a validated diagnostic classification.