Testing the Cybernetic Hypothesis

Predictions, refutations and research priorities

Working document for researchers, clinicians, educators, regulators, journalists and public-health decision makers

Testing whether periodontal disease is a local lesion - or a visible sign of disrupted biological regulation.

Purpose. This document indicates how the Cybernetic Hypothesis of Periodontal Disease can be challenged, refined, strengthened or rejected. Its purpose is to make the hypothesis vulnerable to evidence rather than protected by advocacy. The hypothesis began in veterinary clinical observation, was published in 1994, developed in *Raw Meaty Bones: Promote Health* in 2001, and entered wider periodontal medicine debate in 2026. It should now be tested as a cross-species, cross-disciplinary claim about biological regulation.

1. The proposition to be tested

The conventional model treats periodontal disease chiefly as a local sequence: plaque accumulation, bacterial proliferation, gingival inflammation, tissue breakdown and clinical treatment. That model contains truths, but it may be too small. The Cybernetic Hypothesis proposes that periodontal disease is better understood as a failure of biological regulation at an interface where diet, mechanical function, microbial ecology, host immunity, inflammation, systemic health, time and environment interact through feedback loops.

In this model, the mouth is neither an isolated dental site nor merely a contaminated surface. It is a regulatory boundary. Disease may persist when protective feedback becomes amplifying feedback: inflammation selects a dysbiotic ecology, dysbiosis sustains inflammation, and professional or domestic interventions may suppress signs without correcting the generating system.

2. Core predictions

- Explanatory power should increase when studies include diet quality, food texture, chewing function, ultra-processing, oral ecology, host inflammatory response, systemic health and environmental context.
- In carnivores, diets that provide biologically appropriate mechanical challenge should, all else equal, reduce gingivitis, calculus, periodontal pocketing, extractions and recurrent dental procedures compared with soft, processed feeding systems.
- In humans, periodontal progression should be better predicted by models that include dietary pattern, ultra-processed food exposure, metabolic status and inflammatory markers than by plaque indices alone.
- Durable prevention should require system-level change, not repeated local repair alone.
- The same general pattern should be visible across species: altered diet, altered mechanical use and altered ecology should change oral microbial communities and host inflammatory response over time.

3. Behavioural and educational predictions

The current document adds an important public-health test. If oral disease is partly a system failure, then early education and rewarded daily behaviours should have measurable effects. Children should not merely be told to brush. They should be taught that every mouth, food and animal exists in a network of relationships, consequences and feedback.

- Cybernetic teaching from early childhood should improve children's ability to understand diet, microbes, teeth, gums, environment and commercial influence as connected rather than separate topics.
- Rewarded routines for brushing, interdental cleaning, water drinking, lower sugar exposure and healthier food choices should improve plaque control, gingival inflammation, dental attendance and oral-health equity.
- School, childcare, workplace and aged-care programmes that cue, supervise and reward oral-health behaviours should outperform passive advice alone.
- If such programmes produce no measurable improvement in behaviour or oral outcomes when properly implemented, the behavioural component of the cybernetic framework must be revised.

4. What would strengthen the hypothesis?

- Prospective animal feeding studies showing large and durable oral-health differences after restoring mechanical and dietary conditions.
- Human longitudinal studies showing that diet texture, ultra-processing, metabolic status and inflammatory markers predict periodontal progression independently of conventional local measures.
- Mechanistic studies showing bidirectional feedback between oral inflammation and dysbiosis.
- Intervention trials showing durable improvement when diet, hygiene, metabolic health and environmental cues are addressed together.
- Cross-disciplinary replication in dentistry, veterinary science, cardiometabolic medicine, immunology, microbiome research and ecology.

5. What would weaken or refute the hypothesis?

- Large, well-controlled studies showing that diet, food texture, chewing function, ultra-processing, systemic inflammation and environmental context do not materially improve prediction or prevention of periodontal disease.
- Feeding trials in carnivores showing no meaningful periodontal difference between biologically appropriate mechanical diets and soft processed diets when confounders are controlled.
- Evidence that oral dysbiosis reliably precedes and explains tissue destruction without meaningful contribution from host regulatory failure or wider environmental context.
- Demonstration that system-level education and reward structures produce no durable improvement beyond conventional advice.
- Evidence that repeated local procedures provide durable prevention equal or superior to upstream regulation at lower welfare, financial and environmental cost.

6. Minimum methodological safeguards

- Pre-register hypotheses and outcomes wherever possible.
- Use adequate sample sizes, appropriate follow-up and transparent adverse-event reporting.
- Control for age, breed, smoking, medication, socioeconomic status, dental attendance, sugar exposure and baseline disease severity where relevant.
- Measure recurrence, durability and quality of life, not only short-term plaque or pocket indices.
- Publish negative and mixed results. A theory is strengthened by surviving serious tests, not by avoiding them.

7. The decisive challenge

The central question is not whether every detail of the Cybernetic Hypothesis is already settled. It is whether the existing model has sufficient explanatory and preventive power. A serious hypothesis becomes useful when it generates predictions, invites refutation and changes what researchers think to measure. The Cybernetic Hypothesis now deserves that level of testing.

Selected references and source links

1. Lonsdale T. Periodontal Medicine and the Limitations of Linear Causality: Time for a Cybernetic Framework. *Journal of Periodontal Research*. 2026. https://onlinelibrary.wiley.com/doi/full/10.1111/jre.70118

2. Lonsdale T. Cybernetic hypothesis of periodontal disease in mammalian carnivores. *Journal of Veterinary Dentistry*. 1994;11(1):5-8. https://pubmed.ncbi.nlm.nih.gov/7993585/

3. Lonsdale T. *Raw Meaty Bones: Promote Health*. 2001. https://www.thepetfoodcon.com/raw-meaty-bones/

4. World Health Organization. *Oral health fact sheet*. Updated 17 March 2025. https://www.who.int/news-room/fact-sheets/detail/oral-health

5. Centers for Disease Control and Prevention. *Oral Health Tips for Adults*. Updated 15 May 2024. https://www.cdc.gov/oral-health/prevention/oral-health-tips-for-adults.html

6. Centers for Disease Control and Prevention. *About Periodontal (Gum) Disease*. Updated 15 May 2024. https://www.cdc.gov/oral-health/about/gum-periodontal-disease.html

7. Eke PI et al. Periodontitis in US adults: National Health and Nutrition Examination Survey 2009-2014. *Journal of the American Dental Association*. 2018;149(7):576-588.e6. CDC record: https://stacks.cdc.gov/view/cdc/105956

8. Williams AM et al. Ultra-processed Food Consumption in Youth and Adults: United States, August 2021-August 2023. NCHS Data Brief No. 536. August 2025. https://www.cdc.gov/nchs/products/databriefs/db536.htm

9. National Institute of Dental and Craniofacial Research. *Oral Hygiene*. https://www.nidcr.nih.gov/health-info/oral-hygiene