

Periodontal Disease & The Cybernetic Hypothesis

From local infection to biological regulation, 1992–2026

Why reductionism, germ theory and treatment should be subsumed—not discarded

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A veterinary hypothesis first formulated in 1992 now intersects with modern periodontal medicine, systems biology and cybernetics. The opportunity is not to reject existing science, but to place its parts inside a more explanatory framework of interfaces, feedback, regulation, co-evolution and prevention.

Well, I am surprised. Make sure you cite it in your article in C&T.

_Richard Malik, CVE Valentine Charlton
Consultant, on reading the 2026 *Journal of Periodontal Research* letter

I was surprised too—not because the argument was new to me, but because after 34 years it had crossed an important disciplinary boundary. An idea that began

in veterinary practice in 1992 had entered the human periodontal literature.^{1,3} The event does not prove the Cybernetic Hypothesis of Periodontal Disease. It does, however, create an opportunity to examine it properly.

That opportunity has widened during 2026. The Cybernetic Hypothesis webpage now assembles the historical record, the 1994 *Journal of Veterinary Dentistry* paper, the book-length argument in *Raw Meaty Bones: Promote Health*, the new periodontal medicine letter, diagrams, working papers and proposed tests.^{1-3,16} In July, I also participated in the Cybernetics Society's Conversation at Loughborough University.¹⁵ The value of that gathering lay less in agreement than in the habit of asking a different class of question: not merely 'Which part is defective?' but 'What system is being regulated, by what feedback, across what boundary and over what time?'

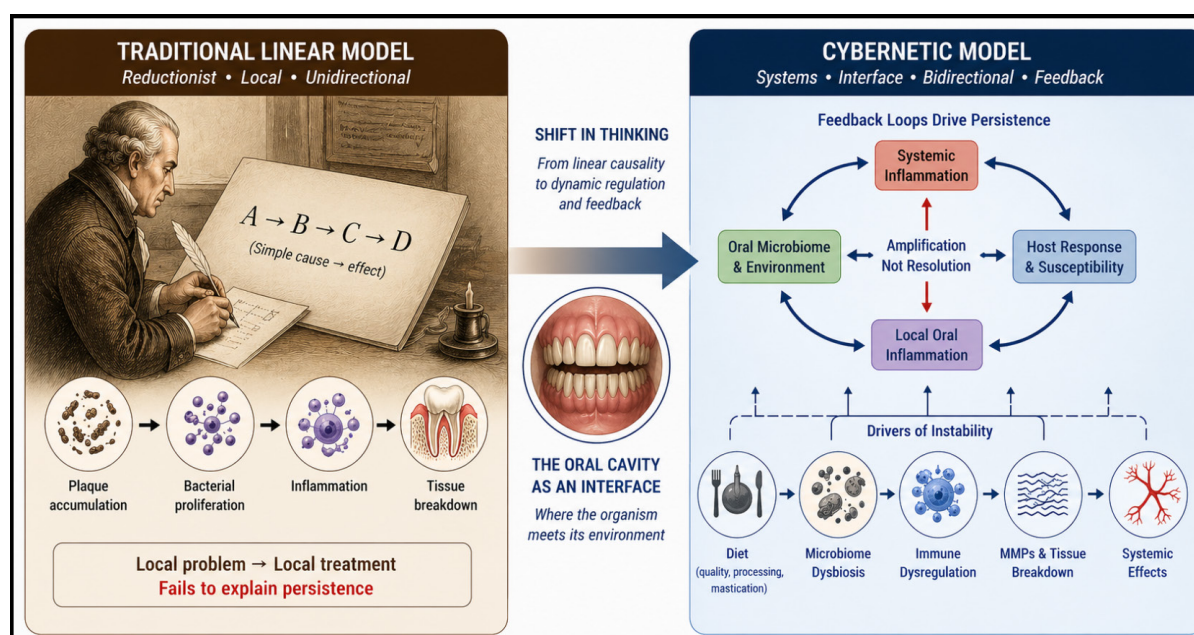


Figure 1. The conventional sequence from plaque to tissue breakdown is useful but incomplete. The cybernetic model places the oral cavity at an interface and asks how diet, microbial ecology, host response, local inflammation and systemic conditions become linked by feedback.

Three Successful Paradigms—And Their Limits

Modern medicine and veterinary medicine have been shaped by three immensely productive habits of thought: reductionism, germ theory and treatment.

Reductionism divides a difficult problem into manageable parts. Germ theory identifies microorganisms and the damage associated with them. The treatment paradigm asks how the damaged part, infectious agent or clinical sign can be controlled. These approaches have delivered anatomy, microbiology, antibiotics, surgery and countless life-saving interventions. The argument for cybernetics is not that these achievements were mistaken.^{2,13,14}

The Problem Arises When Useful Tools Become the Governing Account of Chronic Biological Disease

Reductionism can reveal components while obscuring the relationships that give those components meaning. Germ theory can identify organisms while encouraging us to treat a co-evolved microbial community as an invading army. Treatment can relieve suffering while leaving untouched the conditions that repeatedly regenerate the disease. The result is a familiar clinical sequence: plaque accumulates; bacteria proliferate; inflammation follows; tissues break down; the mouth is cleaned; the process returns.

That Sequence Describes Events. It Does Not Explain Persistence

Research in periodontology has already moved well beyond a simple one-germ/one-disease model. The polymicrobial synergy and dysbiosis model treats disease as the product of a community, host response and ecological shift.⁵ Chronic periodontitis is also increasingly described as a failure of inflammatory resolution rather than merely an excess of inflammation.⁶ Romandini and colleagues have now organised the oral-systemic literature into seven interwoven mechanisms, including microbial translocation, systemic meta-inflammation, trained immunity, immune-cell trafficking, masticatory dysfunction, microbiome dysregulation and shared underlying vulnerabilities.⁷

These are important advances. But they become more coherent when regarded not as a longer list of causes, but as interacting elements of a regulatory system.

The Mouth as a Biological Interface

The mouth is not an isolated compartment. It is an interface at which food, teeth, gums, saliva, microbes, blood flow, immunity, metabolism, behaviour and environment meet. Interfaces are where systems exchange matter, energy and information—and where disturbance can be either corrected or amplified.

In a stable system, inflammatory responses help contain disturbance and then resolve. In an unstable system, outputs return as inputs. Local inflammation contributes to systemic inflammatory signalling; systemic metabolic and immune disturbance alters the susceptibility of periodontal tissues; microbial communities respond to the changed environment; tissue breakdown supplies further nutrients and inflammatory stimuli. The loop sustains itself.

This is Circular Causation, Not a Straight Line

The 2026 *Journal of Periodontal Research* letter argued that the persistent difficulty in isolating the 'independent causal effect' of periodontitis may be partly conceptual.^{3,4} Directed acyclic graphs, Mendelian randomisation and increasingly sophisticated statistical methods can clarify assumptions, but biological systems remain recursive. Diabetes and periodontitis are the obvious example: hyperglycaemia alters immune and tissue responses, while periodontal inflammation adds to systemic inflammatory and metabolic burden. Asking which condition is the single cause may be less useful than asking how the coupled system becomes stable, unstable or capable of recovery.

Cybernetics does not abolish causality. It relocates it. Causation becomes distributed, reciprocal, time-dependent and conditional on the organisation of the whole.

Why Mammalian Carnivores Provide a Revealing Model

Dogs and cats offer an unusually clear view of this organisation because the physical function of the mouth is difficult to separate from nutrition, survival and behaviour.

For a wild carnivore, teeth and gums are working organs. Tearing, shearing and crunching carcass material obtains food while mechanically affecting the oral environment. Food quality, food form, chewing effort, salivary flow, microbial habitat and immune activity are therefore coupled. A carnivore unable to obtain and process food enters a downward spiral of poor intake, weakness, declining immunity and oral disease.^{1,2}

The **Cybernetic Hypothesis** proposes that this coupling has ecological significance. Periodontal decline in a failing wild carnivore may contribute to the attrition of an individual that can no longer perform its regulatory role effectively. In that evolutionary setting, what we call 'disease' may also participate in population and ecosystem regulation.

This is a hypothesis, not an established fact. It invites attempted refutation. Are there balanced populations

of mammalian carnivores in which periodontal disease is absent? How do oral health, hunting success, age, nutritional stress and population dynamics change together? Do the predicted feedback relationships occur?

Domestication creates a revealing disturbance. Modern pets can receive abundant calories in food that requires little tearing or crushing. Protection from starvation and predators allows them to survive for years with a diseased oral interface. The natural coupling between obtaining food and maintaining oral function is weakened. A regulatory process that may have been rapid and self-limiting in the wild becomes chronic amplification in the home.

Two early-life periods deserve particular attention. Deciduous teeth erupt during rapid growth, and permanent teeth later erupt into an already established oral ecosystem. These are likely high-gain periods in which nutrition, chewing, microbial succession and immune development interact intensely. In adulthood, periodontal attrition may play a different role. Lumping these phases together under the singular noun 'periodontal disease' may hide more than it reveals.

The clinical observations that led me to the hypothesis were simple. Many dogs and cats suffered foul mouths and recurrent systemic illness despite conventional treatment. When established dental disease was treated and the feeding system was changed—especially towards appropriately selected raw meaty bones and carcass-derived foods that demanded oral work—owners frequently reported rapid changes in breath, gums, coat, vitality and recurrent illness.² Clinical experience is not a substitute for controlled research. But consistent, reversible observations are not an excuse for avoiding the research either. A later observational comparison of dogs fed raw meat-based or extruded diets also found differences in clinical scores and blood markers, while emphasising the need for better controlled work.¹²

What The Hypothesis Does—And Does Not—Claim

- Bacteria remain important. The hypothesis places microbes within a larger host-microbe-environment system.
- Periodontal disease is not declared the sole cause of chronic illness. Oral and systemic conditions may be coupled expressions of disturbed regulation.
- Treatment remains necessary. Established pathology requires competent scaling, extraction, antimicrobial or other care. The further question is why the pathology arose, persists and recurs.
- Belief is not requested. The hypothesis asks for tests capable of corroborating, modifying or refuting it.

Human Relevance: Scale Without Overstatement

Humans are not dogs or cats, and crude extrapolation would repeat the reductionist error. Yet humans possess the same broad organisational features: an oral interface, co-evolved microbial communities, immune and metabolic feedback, connective-tissue turnover and continuous environmental input.

The scale warrants attention. The World Health Organization estimates that severe periodontal disease affects more than one billion people and that oral diseases overall affect nearly 3.7 billion.⁸ Periodontitis is reported in association with more than 70 communicable and non-communicable conditions.⁷ Association is not proof that periodontal disease independently causes each condition, and many relationships reflect shared determinants. But 'shared determinants' may themselves be evidence of shared regulatory architecture.

The economic setting is equally sobering. Long-term conditions account for about half of the UK's National Health Service (NHS) general-practice appointments,

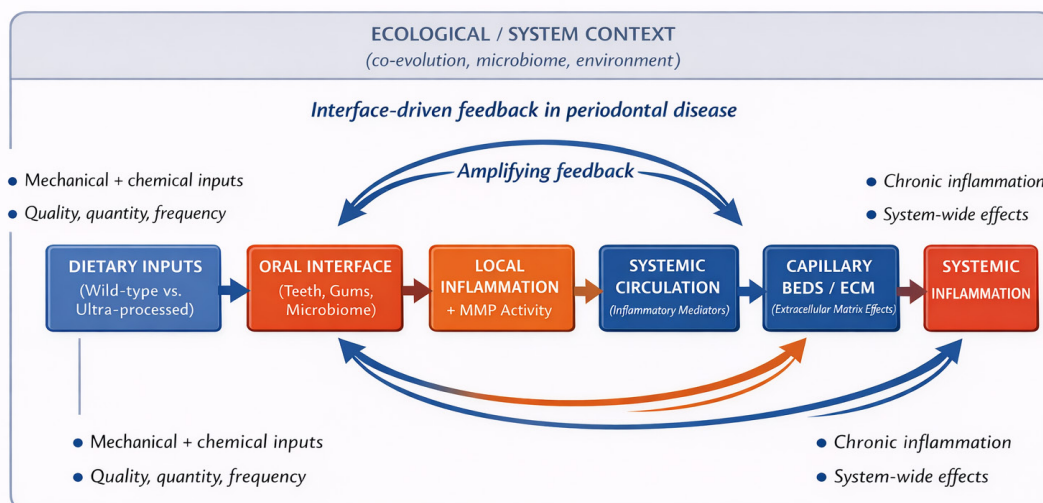


Figure 2. A stylised interface-driven feedback system. Dietary inputs, local inflammation, systemic circulation and tissue effects are connected by amplifying loops within a wider ecological context.

64% of outpatient appointments and more than 70% of inpatient bed days.⁹ An NHS page, citing 2012 Department of Health data, estimated that treatment and care for people with long-term conditions consumed roughly £7 in every £10 of health and social care expenditure.⁹ Total UK healthcare expenditure was provisionally estimated at approximately £345 billion in 2025, although that figure is broader than NHS expenditure.¹⁰

It would be reckless to allocate these costs to periodontal disease alone. It would be equally reckless to assume that an inflammatory interface affecting more than a billion people contributes nothing to the development, amplification or management of chronic disease.

The Appropriate Inference Modest But Important: Even a Small Preventable Contribution From Oral-Systemic Dysregulation Would Matter Greatly at Population Scale

Diet also belongs in this discussion. In people, high exposure to ultra-processed food is associated with adverse cardiometabolic, mental-health and mortality outcomes, although the certainty varies across outcomes.¹¹ In companion animals, 'complete and balanced' nutrient calculations have encouraged attention to chemical composition while marginalising food structure, mastication, behaviour and oral ecology. A cybernetic account asks not only what nutrients enter the bowl or packet, but what the whole feeding process does to the organism over time.

From Treatment to Prevention

The treatment paradigm starts after failure has become visible. Cybernetic prevention begins earlier by identifying the inputs and feedbacks that maintain stability.

For veterinary patients, this means examining the mouth at every consultation, treating pain and established disease, and then changing the conditions that produced it. It means **feeding for function**: considering physical form, chewing behaviour, frequency, microbial consequences and the animal's evolutionary equipment, not merely a nutrient panel. Raw meaty bones are relevant because they restore a class of mechanical and behavioural functions removed by pulverised foods; they should be selected for the individual animal and used with informed supervision, not treated as a commercial recipe or magic ingredient.

For human patients, the intervention cannot simply be copied from carnivores. The principle, however, transfers. Oral hygiene, periodontal treatment, smoking

cessation, metabolic control, diet quality, food texture, mastication, access to care and systemic disease management should be understood as interacting interventions rather than professional silos.

Prevention also changes the research question. Instead of asking only whether periodontal treatment lowers the incidence of a distant disease, we can ask which combinations of oral, dietary, behavioural and metabolic intervention shift the coupled system from amplification towards resolution.

A Research Programme Capable of Failing

A useful hypothesis must take risks. Several tests are now possible.

1. Compare feeding systems, not isolated nutrients

Longitudinal veterinary studies should contrast appropriately designed, functionally natural feeding regimes with conventional ultra-processed regimes, measuring oral status, microbiome dynamics, inflammatory resolution, immune and metabolic markers, behaviour and clinical disease. Existing observational studies and short-term chewing trials are informative, but they do not test the whole cybernetic proposition.

2. Begin before disease is entrenched

Puppies and kittens should be followed through deciduous eruption and permanent dentition, with careful ethical oversight. These predicted high-gain periods may reveal feedback changes that adult cross-sectional studies cannot detect.

3. Measure trajectories and loops

Repeated observations of oral ecology, systemic inflammation, metabolic state, connective-tissue turnover and behaviour can be analysed as a dynamic network. The aim is not to abandon reductionist measurements, but to reconnect them.

4. Seek counter-examples

Wild and captive carnivores, herbivores and omnivores offer natural contrasts. The hypothesis should be revised or rejected if its ecological predictions fail.

5. Test system-level interventions

Studies should combine competent dental treatment with alteration of the feeding environment, then monitor local and systemic recovery. A system-level intervention should produce patterns of timing, sequence, threshold and relapse that a local treatment model does not predict.

Subsumption, Not Rejection

The central proposal is therefore less destructive than it may first appear. Reductionism, germ theory and treatment remain indispensable. Cybernetics places them inside a larger framework of regulation, feedback, interfaces, co-evolution and time.

That is what Chapters 10 and 14 of *Raw Meaty Bones: Promote Health* attempted to do in 2001.² Chapter 10 examined how reductionism, germ theory and the treatment paradigm can misdirect science when mistaken for complete explanations. Chapter 14 proposed a replacement framework with greater explanatory and predictive reach. The 2026 periodontal literature has not proved that proposal, but it has moved markedly towards its language: dysbiosis, bidirectionality, shared vulnerabilities, non-resolving inflammation and interconnected mechanisms.³⁻⁷

The Important Question Is No Longer Whether Germs, Inflammation or Treatment Matter. Of Course They Do. The Question Is Whether They Are Parts Of A System Whose Organisation We Have Failed to See.

Veterinary medicine supplied the original observation and still offers the clearest experimental opportunity. Dentistry and medicine carry the enormous human burden. Evolutionary biology and cybernetics provide the wider frame. It is time for the disciplines to meet at the interface.

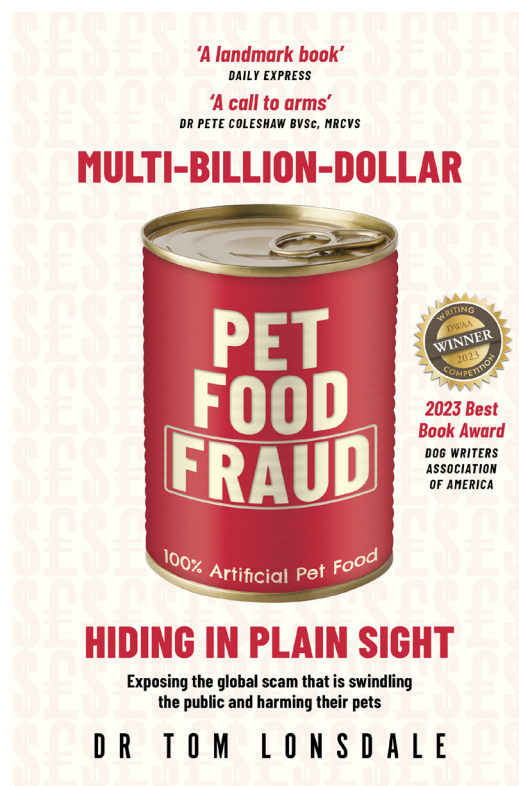
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Further reading

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cve.edu.au/book-review

Tom Lonsdale, June 2018, *Raw meaty bones essentials*, C&T No. 5682, *Control & Therapy* Series.
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