Periodontal Disease, Olfaction and Carnivore Fitness

A Working Paper on Chemosensory Pathways within the Cybernetic Hypothesis

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Author note: This draft develops ideas first raised in the April 2004 *Raw Meaty Bones Newsletter*, later reproduced in *Spin Doctors: Junk Pet Food Partners in Crime*, in the section "New scientific thought, persistent vet school madness". It is intended as a living document and may be revised as additional evidence, criticism and research pathways emerge.

Abstract

The Cybernetic Hypothesis of Periodontal Disease in Mammalian Carnivores proposes that periodontal disease should not be understood solely as a local oral pathology. In free-living carnivores, periodontal breakdown may form part of a larger regulatory system affecting hunting capacity, social standing, reproduction, survival and ecological balance. In domestic carnivores fed artificial diets, the same biological pathways may be displaced from adaptive regulation into chronic dysregulation, producing local and systemic disease.

This working paper revisits observations first discussed in April 2004 in the *Raw Meaty Bones Newsletter* and later reproduced in *Spin Doctors: Junk Pet Food Partners in Crime*. At that time, work reported by Dr Larry Myers suggested that dental calculus in beagles was associated with depressed olfactory thresholds, with smell returning after dental cleaning and declining again as calculus redeveloped. Dr Johan Joubert and I speculated that the vomeronasal organ may be involved. Two decades later, the available scientific landscape points to a wider set of possible pathways: ordinary olfaction, vomeronasal signalling, trigeminal irritation, oral microbiome-derived volatile and non-volatile chemicals, neuroimmune inflammation, systemic inflammatory mediators, behavioural change and ecological feedback.

The central proposition is simple: periodontal disease may impair the carnivore's sensory interface with the world. In a predator whose survival depends on sense of smell, feeding, mating, defence, hierarchy and prey detection, oral disease may have consequences far beyond teeth and gums. This possibility deserves investigation.

1. Origin of the question

In April 2004, in a *Raw Meaty Bones Newsletter* later reproduced in *Spin Doctors*, I discussed a report concerning dental calculus and olfaction in beagles. The dogs' olfactory thresholds were reportedly depressed when calculus accumulated, restored after dental cleaning, and depressed again as calculus redeveloped. The passage noted that this finding strongly suggested a connection between detector-dog performance and dental health.

> *'the olfactory threshold returned to normal'*
> *Spin Doctors, April 2004, p. 84*

> *'a major factor in the efficacy of detector dogs is good dental health'*
> *Spin Doctors, April 2004, p. 84*

At the time, Dr Johan Joubert, veterinary dentist and cybernetician, and I were intrigued. If periodontal disease could impair sense of smell, then it could affect a carnivore's capacity to detect prey, competitors, mates and enemies. We also speculated that the vomeronasal organ might be involved. The vomeronasal organ, or organ of Jacobson, has anatomical access near the incisive region and is associated in many mammals with non-volatile chemical cues, the accessory olfactory bulb, the amygdala and hypothalamic regulation of reproductive, defensive, ingestive and neuroendocrine functions.

> *'foul fluids from diseased teeth and gums gain immediate access to the vomeronasal organ'*
> *Spin Doctors, April 2004, p. 85*

The 2004 passage asked whether periodontal fluids from diseased teeth and gums could negatively affect smell, behaviour and neuroendocrine function. It then made the cybernetic connection: failing hunters may become the hunted; redundant carnivores may be removed from the system; prey, competitors and enemies may benefit; ecological balance may be maintained.

The original speculation was necessarily tentative. However, it was biologically coherent and testable. Two decades later, it appears even more worthy of investigation.

2. The Cybernetic Hypothesis restated

The Cybernetic Hypothesis of Periodontal Disease in Mammalian Carnivores proposes that periodontal disease in carnivores is not merely a random local infection. It may be part of a larger regulatory system linking diet, oral microbiology, host immunity, systemic inflammation, behaviour, survival and population regulation.

In free-living carnivores, oral health is maintained by the routine ripping, tearing and gnawing of whole carcasses and raw meaty bones. Mechanical cleaning, appropriate oral microbial ecology and normal feeding behaviour are integrated. When food supply, age, injury, immune status or other variables alter the balance, oral disease may progress. In such circumstances the affected animal may suffer pain, infection, systemic inflammatory load, reduced feeding efficiency and reduced hunting capacity. In cybernetic terms, this may form a high-gain feedback loop.

In domestic dogs and cats fed artificial diets, the ecological setting has changed. The carnivore is protected from predation, fed from packets and cans, and treated repeatedly for the consequences of diet-induced disease. The regulatory loop is interrupted. Instead of ecological resolution, chronic disease persists. Periodontal disease becomes a long-term inflammatory engine.

The 2004 olfaction observation suggests an additional dimension. Periodontal disease may not only damage the mouth and body; it may also degrade the sensory apparatus by which the carnivore knows the world.

3. From local pathology to sensory interface

The mouth is usually described in dental terms: teeth, gingiva, plaque, calculus, periodontal pockets, inflammation and bone loss. That description is necessary but incomplete.

For the carnivore, the mouth is also:

1. A feeding apparatus.
2. A mechanical processing system.
3. A microbial habitat.
4. A chemical reactor.
5. A source of volatile and non-volatile compounds.
6. A portal to the respiratory and gastrointestinal systems.

7. A contributor to systemic inflammatory load.
8. A social-signalling organ.
9. A potential influence on olfaction, taste, trigeminal sensation and vomeronasal signalling.

In this wider view, periodontal disease is not merely disease around teeth. It is disturbance at a major biological interface. A carnivore's mouth is close to the nose, intimately connected with feeding and breath, and constantly involved in sensing and signalling. When the oral microbiome becomes dysbiotic and the periodontal tissues inflamed, the consequences may radiate through multiple channels.

4. Candidate mechanisms

4.1 Ordinary olfaction

The most direct pathway is impairment of ordinary sense of smell. Dogs and many other carnivores rely heavily on olfaction. They identify food, prey trails, mates, territories, competitors, danger and social status through scent.

If periodontal disease or dental calculus depresses olfactory thresholds, even temporarily, then the consequences may be profound. The affected animal may detect prey later, follow trails less accurately, miss danger signals, misread social cues or lose competitive advantage. In working dogs, the implications extend to detection, search and rescue, policing, customs, hunting and military applications.

This is the most accessible research pathway. It can be tested by comparing olfactory performance with oral health status before and after periodontal treatment, and by following animals longitudinally as calculus and gingival inflammation recur.

4.2 Vomeronasal signalling

The vomeronasal organ remains a plausible but unproven pathway. In many mammals it detects non-volatile chemical cues and links to neural circuits involved in reproduction, defence, social behaviour and neuroendocrine regulation.

The anatomical proximity of the oral cavity, incisive ducts and nasal/vomeronasal structures raises an important question. Could periodontal exudates, microbial metabolites, altered saliva or inflammatory products influence vomeronasal signalling? Could chronic oral disease distort chemical information relevant to mating, hierarchy, fear, aggression, maternal behaviour or feeding?

This should not be presented as established fact. It should be presented as a biologically plausible mechanism requiring investigation. The hypothesis is not that the vomeronasal organ explains all periodontal-systemic effects. Rather, it may be one component of a broader chemosensory network.

4.3 Oral microbiome-derived chemical signals

The diseased mouth is chemically active. Anaerobic bacteria, tissue breakdown products, blood, inflammatory exudate, saliva, food residues and microbial metabolites contribute to oral malodour and altered breath chemistry.

In ecological terms, bad breath is not trivial. Odour is information. It may signal disease, weakness, immune status, age, diet, social rank or reproductive fitness. A predator with strong oral malodour may become more detectable to prey. A social carnivore with periodontal disease may be perceived differently by pack members. A mate with altered chemical signals may become less attractive. A competitor may detect vulnerability.

This creates a possible link between oral dysbiosis and social/ecological feedback. The oral microbiome may act as a chemical-signalling system, and periodontal disease may distort the signal.

4.4 Trigeminal and irritant pathways

Smell is not the only sensory system involved in nasal and oral chemical detection. The trigeminal system responds to irritants, temperature, pain and chemical stimulation. Periodontal disease produces inflammatory mediators, volatile sulfur compounds and other irritant substances. These may influence nasal comfort, breathing, feeding behaviour, avoidance, grooming, sleep and stress responses.

In domestic animals, such effects may be subtle and chronic. In wild carnivores, even small changes in sensory acuity or behavioural confidence may have large consequences.

4.5 Neuroimmune inflammation

The emerging field of neuroimmune biology shows that sensory tissues and immune pathways are deeply connected. The olfactory epithelium is not a passive smell detector. It is a mucosal surface exposed to the environment and engaged in immune surveillance, epithelial repair and neural signalling.

Periodontal disease may contribute inflammatory mediators, bacterial products and systemic immune activation. These may influence olfactory tissues directly or indirectly. Conversely, altered sensory signalling may influence appetite, stress, endocrine function and behaviour.

The cybernetic point is that these are not one-way chains. They are loops.

4.6 Olfactory receptors outside the nose

Olfactory receptors are no longer regarded only as smell receptors in the nasal cavity. They are reported in other tissues, including immune cells. Their functions remain incompletely understood, but they appear to participate in cellular responses to chemical environments.

This opens a further possibility. Microbial metabolites from periodontal disease may interact with chemosensory receptors beyond classical smell pathways. Such effects might influence macrophage behaviour, inflammation, tissue destruction or resolution. At this stage this remains speculative, but it is consistent with the larger idea that chemical sensing and immune regulation are intertwined.

4.7 Systemic inflammatory mediators

Periodontal disease is associated with local tissue destruction and systemic inflammatory effects. Bacteria, endotoxins, cytokines, matrix metalloproteinases and other mediators may circulate. In the Cybernetic Hypothesis, these systemic effects are central. The mouth is not isolated from the body.

The olfactory and vomeronasal systems may therefore be affected not only by local contamination or odour but also by systemic inflammation. A carnivore with periodontal disease may be impaired through pain, malaise, feverishness, reduced appetite, altered endocrine state, reduced physical performance and impaired sensory processing.

5. Ecological consequences

For humans and domestic animals, periodontal disease is usually framed as a medical problem. For free-living carnivores, it may also be an ecological event.

A carnivore with impaired oral health may:

- hunt less effectively;
- process carcasses less efficiently;
- lose weight;
- develop systemic inflammation;
- emit altered odours;
- misread or fail to detect chemical cues;
- lose social position;

- reproduce less successfully;
- become vulnerable to competitors or prey;
- die earlier.

From the standpoint of the individual, this is disease and suffering. From the standpoint of the ecosystem, it may contribute to regulation. Apex carnivores regulate prey populations. But apex carnivores themselves require regulation. Periodontal disease may be one of nature's ways of placing limits on carnivore success.

This does not mean periodontal disease is 'good' in any sentimental sense. It means that disease processes may have regulatory roles in natural systems. The same phenomenon can be harmful to the individual and functional within the larger system. Cybernetics helps us hold both truths at once.

6. Domestic distortion

The domestic dog and cat live under artificial conditions. They are fed processed diets, denied routine access to whole carcasses and raw meaty bones, protected from predation, provided with veterinary treatments and maintained in chronic disease states.

In this setting, the possible regulatory function of periodontal disease is distorted. The animal does not simply fail as a hunter and exit the system. Instead, the animal remains in the household, often with chronic oral infection, chronic inflammation, repeated treatments and progressive systemic disease.

This is a crucial distinction. In the wild, periodontal disease may contribute to ecological resolution. In domestic conditions, it becomes unresolved pathology. The feedback loop is broken. The disease continues without performing its former regulatory function.

7. Research questions

10. Does periodontal disease measurably impair olfactory thresholds in dogs, cats or other carnivores?
11. Does periodontal treatment improve olfactory performance?
12. Does recurrence of calculus or gingival inflammation correlate with renewed olfactory decline?
13. Are working dogs with better oral health more reliable in scent detection tasks?
14. Do oral microbial profiles correlate with breath volatile compounds and olfactory performance?
15. Do periodontal exudates or oral microbial metabolites influence vomeronasal signalling?
16. Does oral dysbiosis alter social behaviour, mating behaviour, hierarchy or aggression in carnivores?
17. Do systemic inflammatory markers correlate with olfactory impairment in periodontal disease?
18. Are trigeminal irritation pathways involved?
19. Can raw meaty bones and whole-carcass feeding prevent or reverse the sensory and inflammatory consequences of periodontal disease better than artificial diets or dental substitutes?

8. Suggested study designs

8.1 Working-dog observational study

Recruit detection dogs from police, customs, military, search-and-rescue or conservation programmes. Score oral health using standard veterinary dental indices. Measure scent detection accuracy, threshold and endurance. Adjust for age, breed, training, handler and workload. Test whether oral health predicts performance.

8.2 Before-and-after periodontal treatment study

Measure olfactory thresholds in dogs before dental cleaning, shortly after cleaning and during follow-up as plaque/calculus returns. Include inflammatory markers and oral microbiome sampling.

8.3 Diet comparison study

Compare dogs fed raw meaty bones or whole-carcass-derived diets with dogs fed ultra-processed diets. Outcomes should include oral health, olfactory performance, oral microbiome, volatile organic compounds, systemic inflammatory markers and owner/handler behavioural reports.

8.4 Vomeronasal exploratory study

In appropriate animal models or post-mortem material, investigate whether periodontal fluids or oral microbial metabolites can access or influence vomeronasal structures. This work would require careful ethical review and specialist anatomical/neurobiological collaboration.

8.5 Chemical ecology study

Analyse breath and saliva chemistry in carnivores with and without periodontal disease. Investigate whether volatile and non-volatile compounds alter responses in conspecifics, prey species or trained detection systems.

9. Implications

If these pathways are confirmed, the implications are substantial.

For veterinary medicine, oral health would need to be understood not merely as dentistry but as a foundation of sensory, systemic and behavioural health.

For working-dog programmes, dental health could become a core performance variable.

For ecology, periodontal disease could be investigated as a mechanism affecting predator efficiency, prey survival and population regulation.

For human medicine and dentistry, carnivores may offer a powerful model for understanding oral-systemic, chemosensory and neuroimmune feedback.

For the Cybernetic Hypothesis, confirmation of olfactory impairment associated with periodontal disease would provide a tangible, testable high-gain pathway linking oral pathology with survival and ecological regulation.

10. Caution

This working paper does not claim that the vomeronasal organ has been proven to mediate periodontal effects. Nor does it claim that every form of periodontal disease is adaptive. The claim is more modest and more important: periodontal disease in carnivores may influence sensory, inflammatory, behavioural and ecological systems in ways that have not been adequately investigated.

The dominant linear model asks: what causes periodontal disease?

The cybernetic model asks: what does periodontal disease do within the system?

That second question opens new scientific territory.

11. Conclusion

The 2004 observation linking dental calculus and impaired olfaction in beagles deserves renewed attention. At the time, it suggested a possible connection between periodontal disease, smell, vomeronasal function and carnivore fitness. Today, with expanding knowledge of the oral microbiome, systemic inflammation, volatile microbial metabolites, neuroimmune biology and chemosensory receptors, the original question appears even more significant.

Periodontal disease may be more than a local oral disease. In mammalian carnivores it may affect the sensory interface by which the animal feeds, hunts, mates, competes, defends itself and participates in ecological regulation.

The challenge now is to test the pathways.

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