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**Editorial**

# Antisenescence therapy: The next leap in periodontal medicine

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Aging is an inescapable biological reality—but the tissue damage it brings may not be. Over the past decade, science has begun to uncover one of the most intriguing secrets of aging: the role of cellular senescence in driving chronic inflammation, tissue breakdown, and impaired healing. This understanding is now transforming how we view oral and periodontal diseases. What was once accepted as an inevitable outcome of age is emerging as a potential target for intervention.[1,2]

## 1. Senescence: The Silent Saboteur

Senescent cells are damaged or stressed cells that permanently stop dividing. Though metabolically active, they release a flood of inflammatory and tissue-degrading molecules known collectively as the senescence-associated secretory phenotype (SASP).

In the mouth, these cells accumulate in periodontal tissues, creating a low-grade inflammatory state—sometimes called inflammaging. This persistent inflammatory environment slows healing, accelerates bone resorption, and undermines periodontal stability, even in the absence of overt infection.[3-5]

## 2. Why Traditional Therapies Fall Short

Traditional periodontal treatments—scaling, root planing, surgery, and antimicrobial therapy—are highly effective at removing bacterial plaque and calculus. Yet, they rarely address the underlying biological aging of the tissue. Senescent cells remain behind, continuing to secrete inflammatory mediators and impair regeneration. This may explain why, despite excellent clinical care, some patients still experience chronic inflammation or relapse of disease.

## 3. The Promise of Senotherapy

A new therapeutic vision is taking shape—senotherapy, the targeted removal or modulation of senescent cells to rejuvenate tissue function. Senotherapeutic agents work in two main ways:

1. ***Senolytics*** eliminate senescent cells directly.
2. ***Senomorphics*** suppress their inflammatory secretions without killing the cells.

Among the most promising of these agents is the combination of Dasatinib (a tyrosine kinase inhibitor) and Quercetin (a natural flavonoid), often abbreviated as D+Q. This combination has shown strong synergy in preclinical models, reducing senescent cell numbers, dampening SASP activity, and improving tissue repair responses.[6,7]

## 4. From Bench to the Gingiva

Recent studies have linked Fusobacterium nucleatum, a key periodontal pathogen, to the induction of senescence-like changes in gingival epithelial cells. This bacterium alters lysosomal function and nuclear integrity, leading to impaired cell proliferation and tissue renewal.

Encouragingly, treatment with D+Q has been shown to reverse these senescence markers in vitro and reduce alveolar bone loss in vivo in aged animal models. These findings

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provide a glimpse of what periodontal therapy could look like in the near future—one that not only controls infection but rejuvenates aging tissue at the cellular level.

## 5. The Road Ahead

The promise of senotherapy is compelling, but much remains to be proven. The translation from laboratory to clinic requires careful testing—particularly regarding dosage, safety, delivery mechanisms, and long-term outcomes. However, the conceptual shift it represents is profound: from merely repairing tissue to reprogramming, it for regeneration.

If future clinical trials confirm current findings, antisenescence therapy could redefine periodontal care, offering patients not just symptom control but true biological rejuvenation. Moreover, since oral inflammation has systemic effects, these benefits may extend beyond the mouth—potentially influencing overall health and longevity.[8-10]

## 6. Aging Gracefully, at the Cellular Level

Antisenescence therapy represents more than a new drug class—it's a new philosophy of care. By targeting the molecular roots of aging, we may one day preserve oral vitality well into advanced age. The ultimate goal is not simply to save teeth, but to sustain health, function, and resilience—cell by cell, tissue by tissue, year by year.

## 7. Conflict of Interest

None.

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