Oral microbiome dynamics and biological ageing in dentisty

Ageing is not merely a chronological phenomenon; it is, above all, a biological process shaped by cellular, immunological, and microbial changes. Telomeres play a central role in this process, as their shortening is regarded as a marker of diminished regenerative capacity and an increased inflammatory burden. In the oral cavity in particular—where tissue renewal, microbial exposure, and immune responses are closely intertwined—telomere biology offers new insights into the onset and progression of periodontal disease.

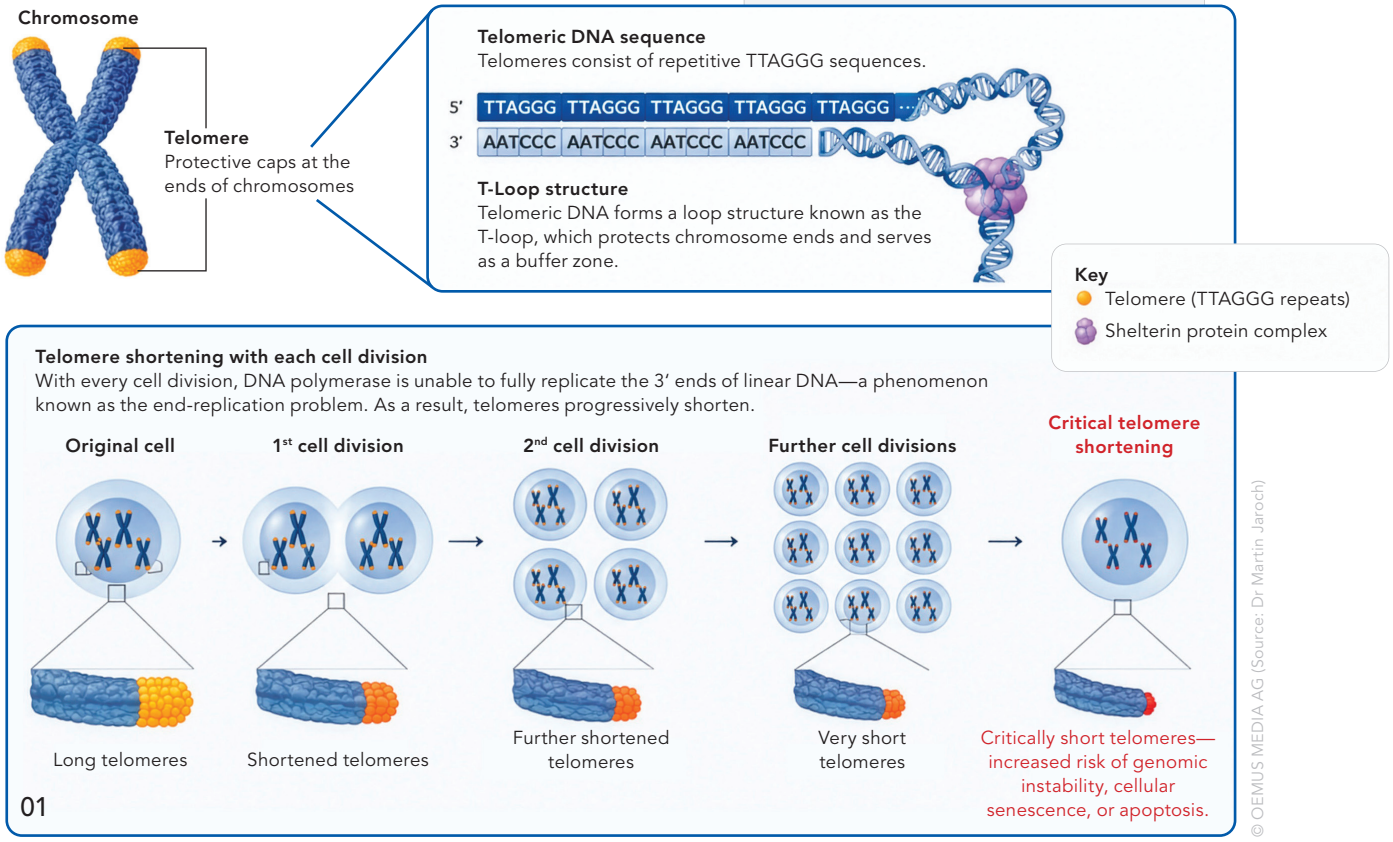
Dr Martin Jaroch, Germany

This article explores the interplay between telomeres, cellular senescence, and the oral microbiome, and illustrates how these factors contribute to a more integrative understanding of biological ageing in dentistry.

The foundations of modern telomere research were laid by the pioneering work of Elizabeth Blackburn, who, together with Jack Szostak and later Carol Greider, elucidated the structure and function of chromosome ends.[1] Telomeres consist of repetitive TTAGGG sequences and protect chromosomes from instability and progressive shortening during each cell division (Fig. 1).[2,3]

Telomeres—the protective caps of chromosomes
Telomeres are composed of repetitive TTAGGG sequences and protect chromosomes from instability and progressive shortening during each cell division.

Functions of telomeres
- Protect chromosome ends from DNA damage and end-to-end fusion
- Prevent genomic instability
- Preserve genetic information during cell division
- Serve as indicators of cellular ageing and replicative capacity



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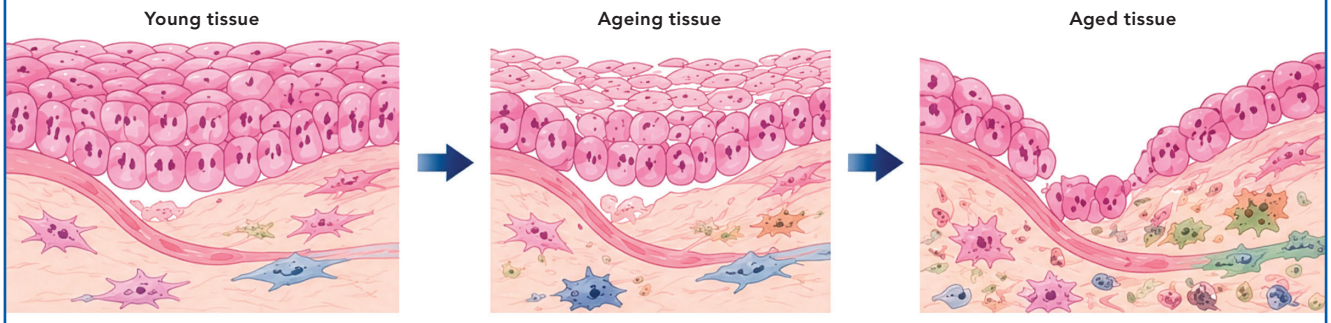
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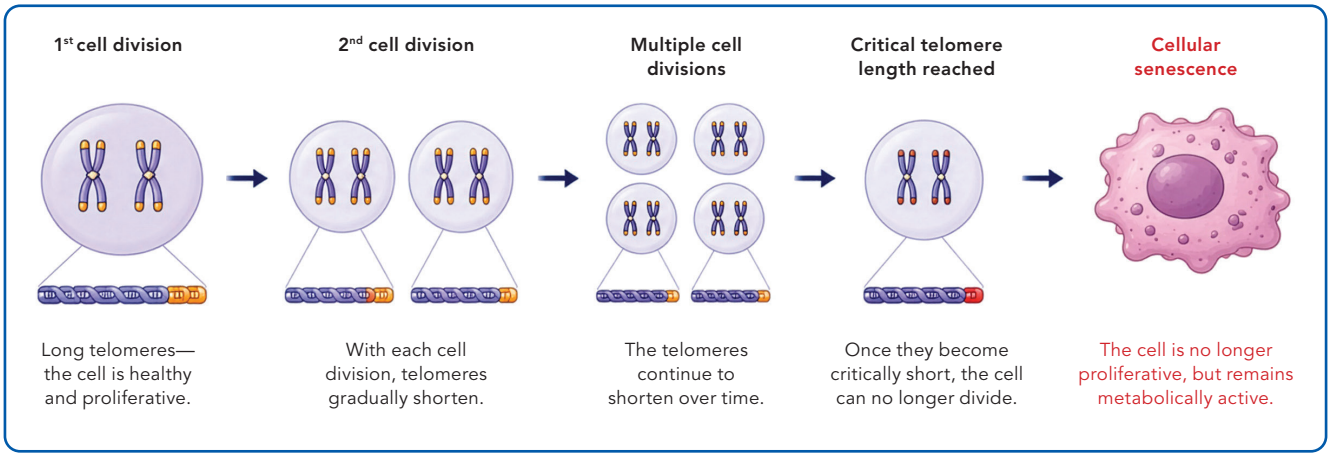


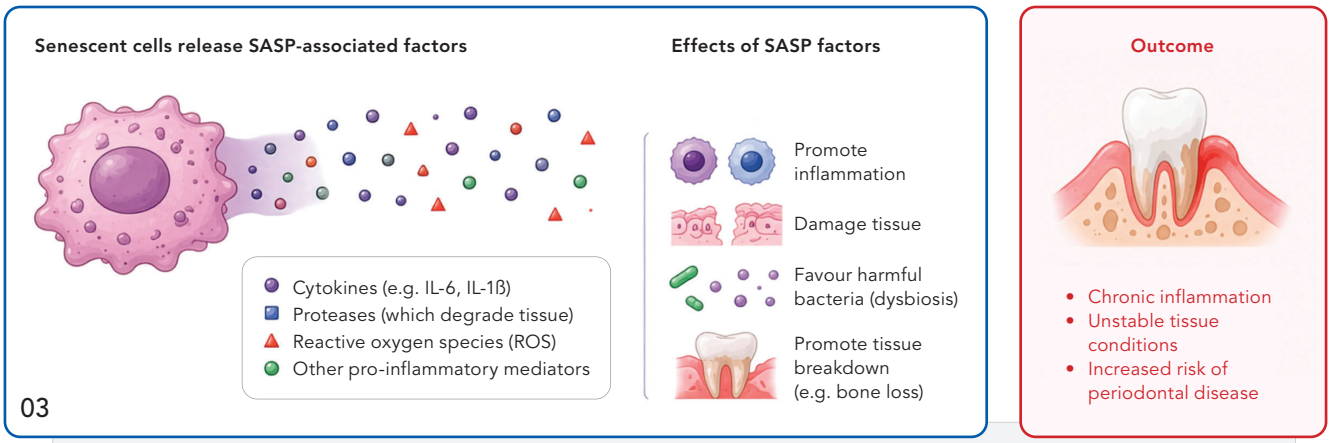
Telomerase activity	high	declining	very low
Telomere length	long	shortened	very short
Cellular regeneration	high	reduced	markedly reduced
Tissue stability	stable	increasingly vulnerable	unstable
Vulnerability	low	increased	very high

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Effects of advancing age on oral tissues
With advancing age, low telomerase activity in somatic oral cells contributes to telomere shortening, diminished regenerative capacity, and greater susceptibility to inflammation, tissue breakdown, and periodontal disease.





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Telomere shortening leads to cellular senescence and inflammation
Key message: Every cell division shortens telomeres. When they become critically short, the cell enters a senescent state. Although it can no longer divide, it remains metabolically active and releases pro-inflammatory factors that damage tissue and amplify inflammation.

This discovery made it clear that cells possess a biological "clock." This is particularly relevant for tissues with a high turnover rate, such as the oral epithelium and the periodontal apparatus, which require continuous renewal due to mechanical stress, bacterial exposure, and ongoing regeneration.

With the discovery of telomerase, Blackburn and Greider identified an enzyme capable of partially compensating for telomere shortening.[1,4] While stem cells and germline cells exhibit high telomerase activity, its expression is markedly reduced in somatic cells. In oral tissues, this contributes to increased vulnerability with advancing age (Fig. 2). Chronic inflammation, which is common in the oral cavity, further accelerates telomere shortening and promotes the functional ageing of periodontal tissues.

Subsequent studies also demonstrated that psychological stress and systemic inflammation are closely associated with shortened telomeres.[3] This is of particular importance in periodontology, as periodontitis is understood as a multifactorial disease in which bacterial dysbiosis, immune responses, and systemic inflammation interact. In this context, telomere length serves as a biological marker of inflammatory burden and regenerative capacity.

Telomeres, senescence and immune ageing

With each cell division, telomeres become shorter. Once they fall below a critical length, the cell enters a state of senescence. Senescent cells lose their ability to divide but remain metabolically active, releasing pro-inflammatory mediators. This so-called SASP (senescence-associated secretory phenotype) includes cytokines, proteases, and reactive oxygen species that destabilise tissues and promote chronic inflammation (Fig. 3).

In the oral epithelium and periodontal ligament, this results in reduced regenerative capacity and increased susceptibility to dysbiotic changes. At the same time, telomere shortening also affects the immune system. T lymphocytes and neutrophils, in particular, are highly sensitive to telomere loss. The resulting immunosenescence impairs the control of microbial biofilms and favours a shift toward anaerobic, inflammation-associated species.

Oxidative stress further amplifies these processes.[5] Bacterial metabolites, smoking, alco-

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hol consumption, and an unbalanced diet generate free radicals that directly damage telomeres. This creates a self-perpetuating cycle of inflammation, oxidative stress, senescence, and dysbiosis.

Lifestyle as a modulator of telomere biology

Telomere length is strongly influenced by lifestyle factors. Chronic psychological stress activates inflammatory signalling pathways and increases cortisol release. At the same time, telomerase activity declines while oxidative stress rises. In oral tissues, this leads to reduced regenerative capacity and greater susceptibility to dysbiosis.

Nutrition also has a substantial impact on telomere integrity. Antioxidant nutrients such as vitamin C, vitamin E, polyphenols, and omega-3 fatty acids reduce oxidative damage and help stabilise inflammation-regulating processes. In doing so, they indirectly support the stability of the oral microbiome.

By contrast, smoking and alcohol exert pronounced telomere-shortening effects. Tobacco smoke contains numerous oxidising substances that cause DNA damage and overwhelm repair mechanisms. Alcohol further increases oxidative stress and alters the oral environment in ways that favour pathogenic microorganisms. Clinically, this is reflected in poorer wound healing, greater bone loss, and more aggressive periodontal disease progression.

Sleep deprivation also adversely affects telomere biology. Chronic sleep disturbances increase inflammatory markers such as IL-6 and CRP and impair immune function. Physical activity, by contrast, exerts a protective effect: it reduces oxidative stress, stabilises immune function, and is associated with longer telomeres and a lower prevalence of severe periodontitis (Fig. 4).

Telomere shortening and periodontitis

Current studies consistently show that patients with periodontitis have shorter telomeres, both in immune cells and in periodontal tissues.[5–7] Telomeres are significantly shortened, particularly in inflamed areas. This finding correlates with attachment loss, periodontal pocket formation, and alveolar bone resorption.

Senescent cells exert a lasting influence on the periodontal microenvironment. Through their SASP profile, they promote inflammatory processes and destabilise microbial balance. As a result, pathogenic species such as *Porphyromonas gingivalis*, *Treponema denticola*, and *Tannerella forsythia* gain a selective advantage.

This dysbiosis, in turn, further increases oxidative stress and DNA damage. Bacterial metabolites such as butyrate impair mitochondrial function and promote senescence-associated processes.[8] At the same time, immune cells produce large amounts of reactive oxygen species which, although intended to exert antimicrobial effects, also cause additional tissue damage and accelerate telomere shortening.

Moreover, telomere shortening affects bone metabolism. Increased RANKL expression and reduced OPG production stimulate osteoclastogenesis and intensify alveolar bone loss. Patients with a high inflammatory burden and short telomeres therefore often show a poorer response to regenerative therapies.

Lifestyle and telomeres—what protects, what harms?

Protective factors		
Healthy diet Plenty of fruit, vegetables, nuts, omega-3 fatty acids, and antioxidants	»	Less inflammation, protects telomeres
Regular exercise Endurance and strength training	»	Reduces stress, strengthens the immune system, associated with longer telomeres
Adequate sleep Seven to eight hours per night	»	Supports regeneration, reduces inflammation, protects telomeres
Stress management Relaxation, meditation, mindfulness	»	Lowers stress hormones, reduces inflammation, protects telomeres

Harmful factors		
Smoking Tobacco smoke damages cells and DNA	»	More inflammation, shortens telomeres, poorer healing
Alcohol Increases oxidative stress and disrupts the oral environment	»	Promotes inflammation, shortens telomeres, favours dysbiosis
Sleep deprivation Too little or poor-quality sleep	»	More inflammation, weakens the immune system, shortens telomeres
Chronic stress Persistent stress increases cortisol levels	»	More inflammation, reduced telomerase activity, shortened telomeres

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Implications for oral health
A healthy lifestyle helps preserve telomere integrity, supports immune function, and promotes a stable oral microbiome. Unhealthy habits, by contrast, drive inflammation, accelerate cellular ageing, and increase the risk of periodontitis.

“Current evidence suggests that shortened telomeres and increased cellular senescence may impair the regenerative capacity of peri-implant tissues and increase susceptibility to peri-implant inflammation.”

Modern research and precision periodontology

Telomere biology is becoming increasingly important for the diagnosis and treatment of periodontitis.[6,7,9] Assessing telomere length in blood, saliva, or gingival tissue can provide valuable insights into inflammatory status and regenerative capacity. Salivary analysis, in particular, is considered a promising noninvasive biomarker approach.

Modern sequencing technologies have also shown that the composition of the oral microbiome is closely linked to processes of biological ageing. Senescent tissues and dysregulated immune responses create conditions under which pathogenic biofilms can develop more readily. Conversely, bacterial metabolites further promote telomere shortening and inflammation.[8,9]

These findings form the basis of precision periodontology. The goal is an individualised diagnostic approach that integrates telomere length, microbiome profiles, and inflammatory markers in order to predict disease risk more accurately and tailor therapies to the patient’s biological profile.

At the same time, therapeutic strategies aimed at modulating senescence are being actively investigated. Compounds such as quercetin, fisetin and metformin may help reduce the burden of senescent cells. Controlled modulation of telomerase activity is also being explored experimentally.[4,9] In addition, probiotic and biofilm-modulating approaches are gaining importance as means of promoting oral microbiome stability and reducing inflammatory processes.

Implants, peri-implantitis and telomeres

The relevance of telomere biology is not limited to periodontitis affecting natural teeth; it is also becoming increasingly important in implant dentistry. Recent studies suggest that shortened telomeres and increased cellular senescence may impair the regenerative capacity of peri-implant tissues and increase susceptibility to peri-implant inflammation. Telomeres may therefore also serve as biomarkers of disease activity, tissue stability, and therapeutic prognosis in peri-implant tissues. The integration of biological ageing markers thus opens new perspectives for the prevention and personalised treatment of peri-implant diseases.[10]

Conclusion

Telomere research has fundamentally reshaped our understanding of periodontitis. Telomeres lie at the intersection of cellular ageing, immune function, inflammation, and microbial ecology. Their shortening reduces the regenerative capacity of tissues, promotes senescence and destabilises the oral microbiome. At the same time, pathogenic biofilms further accelerate telomere loss through oxidative stress and inflammation.

Periodontitis therefore no longer appears primarily as a local bacterial infection, but rather as the manifestation of a biologically ageing and inflammatorily dysregulated system. These relationships are also highly relevant to implant dentistry, as biological ageing processes and inflammatory dysregulation can likewise affect the stability of peri-implant tissues and increase susceptibility to peri-implant diseases. The integration of telomere biology, microbiome analysis, and individualised therapeutic concepts thus opens new perspectives for diagnosis, prevention and regenerative therapy in modern dentistry.

References



Dr Martin Jaroch
Germany
www.drjaroch.de
info@drjaroch.de