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Impact of aging on oral tissue: navigating the interplay for comprehensive geriatric oral health

Wpływ starzenia się na tkanki jamy ustnej: poruszanie się po wzajemnych zależnościach w celu uzyskania kompleksowego zdrowia jamy ustnej w starszym wieku

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Summary

The term "age changes" refers to all the changes that occur in the body from birth to death. However, it is usual to consider age changes as those which are evident in later life. Aging is a natural process. It has a major impact on the quality of life of an individual.

Age-related wear of tooth surfaces reduces enamel thickness, leading to exposure to deeper layers of the enamel. Age-related changes in dentine involve the formation of tertiary or sclerotic dentine, which reduces the lumen of the dentinal tubules. This leads to a reduction in permeability and a subsequent reduction in the volume of the pulp chamber. Gingival recession is a leading cause of root caries in geriatric individuals. Elderly patients, particularly postmenopausal women report loss of sensation of taste and a burning sensation in the oral mucosa as they age. There are prominent changes in the composition and consistency of saliva, which also affects the individual's perception of taste and leaves them prone to conditions such as xerostomia.

The aging effects on the oral cavity impact an individual's overall well-being; the understanding of its process, as well as differentiating it from pathological changes, can help in providing comprehensive oral healthcare to geriatric patients and improving their quality of life.

Streszczenie

Termin „zmiany wieku" odnosi się do wszelkich zmian zachodzących w organizmie od urodzenia aż do śmierci. Jednak zwykle uważa się, że zmiany związane z wiekiem są widoczne w późniejszym życiu. Starzenie się jest procesem naturalnym. Ma to ogromny wpływ na jakość życia człowieka.

Związane z wiekiem zużycie powierzchni zębów zmniejsza grubość szkliwa, co prowadzi do ekspozycji jego głębszych warstw. Związane z wiekiem zmiany w zębinie polegają na tworzeniu się zębiny trzeciorzędowej lub sklerotycznej, co powoduje zmniejszenie światła kanalików zębinowych. Prowadzi to do zmniejszenia przepuszczalności, a co za tym idzie zmniejszenia objętości komory miazgi. Recesja dziąseł jest główną przyczyną próchnicy korzeni u osób w podeszłym wieku. Pacjenci w podeszłym wieku, zwłaszcza kobiety po menopauzie, zgłaszają z wiekiem utratę czucia smaku i uczucie pieczenia błony śluzowej jamy ustnej. Występują wyraźne zmiany w składzie i konsystencji śliny, co również wpływa na postrzeganie smaku przez daną osobę i podatność na schorzenia, takie jak kserostomia.

Efekty starzenia się jamy ustnej wpływają na ogólne samopoczucie człowieka, a ich zrozumienie i odróżnienie od zmian patologicznych może pomóc w zapewnieniu kompleksowej opieki zdrowotnej jamy ustnej pacjentom geriatrycznym i poprawie ich jakości życia.

Introduction

The quality of life is impacted by oral health. Elderly people take dental health seriously since it affects eating, appearance and comfort. Most vertebrates produce teeth, which are ectodermal organs that come in various sizes, forms and quantities. Enamel, dentine and cementum are the three mineralized tissues that make teeth up, which are intricate structures that surround soft tissues like dental pulp, blood vessels and nerves. The tooth's protective layer, known as nonvital enamel, gradually becomes worn down, discoloured and less permeable as people age.[1]

Aging and age-related issues

Genetics, environment and lifestyle all have an impact on the irreversible biological phenomenon of aging. Aging refers to various functional and anatomical changes in tissues over time, which ultimately reduces the ability to respond to internal and external stressors. It is not the same as the concept of "lifespan".[2]

Humans are most commonly affected by chronic dental diseases. In childhood and adolescence, dental caries is a common occurrence, and gingivitis is a lifetime condition that peaks during puberty. While oral squamous cell cancer is typically associated with advanced age (≥60 years), periodontitis is cumulative and becomes more extensive and severe with age. Aging is a natural process, but people in a population age at different rates. The oral cavity likewise follows this principle, but dentition disorders accumulate over time.[3] A summary of the age-related changes in oral tissue is depicted in Table 1 and Figure 1.

Theories of aging

One way to conceptualize aging is as a series of events from conception to death. An estimated three hundred theories about aging have been put forth by numerous researchers. According to one theory, during aging tissue degeneration is linked to extracellular matrix deposition, which causes fibrosis and fatty infiltration. This causes an increase in stiffness, a decrease in elasticity and anatomical disarray of the organs, which is harmful to their proper functioning.[4] Elderly patients may experience symptoms like burning in the mouth, strange tastes and dry mouth, especially if they are postmenopausal women. Another explanation for aging is that somatic cell mutations or replication errors trigger an autoimmune phenomenon.[5] Broadly, the theories can be classified as seen in Figure 2.

Biological: explains the aging concerning the physiological processes that occur in all living organisms over time.

Sociological: looking at the roles and relationships within which individuals engage in later life.

Psychological: controlled by biology and sociology; addresses how a person responds to the engagements of their age.

Moral/Spiritual: Study how individuals seek to validate his or her existence.

Physiological changes in tooth enamel with age

Enamel is the highly mineralized epithelium-derived outermost covering of the crown of the tooth. The enamel is composed of 96% inorganic material, making it the most highly mineralized tissue in the body. The cells forming the enamel, ameloblasts, are lost as the tooth erupts. Injuries or damage to the enamel layer cannot be regenerated due to a loss of ameloblasts at the onset of tooth eruption.[1] The high mineral content and complex organization allow the enamel to withstand high degrees of masticatory forces. The hydroxyapatite crystals are arranged to form enamel rods, separated by interrod enamel.

Age causes teeth to darken. It is arguable whether this darkening results from a modification in the enamel's structure. While the environment's organic material may contribute

Table 1. Summary of age-related changes in oral tissues

	Changes with age
Dentition	fracture lines, incisal edges are chipped, teeth are darker in colour
Masticatory function	reduced, but adequate efficiency
Temporomandibular joint	no discomfort
Periodontium	limited attachment loss, observed as recession on the buccal surface
Oral mucosa	wound healing slightly delayed, adequate barrier function
Salivary flow	slightly reduced, xerostomia in some individuals with underlying conditions

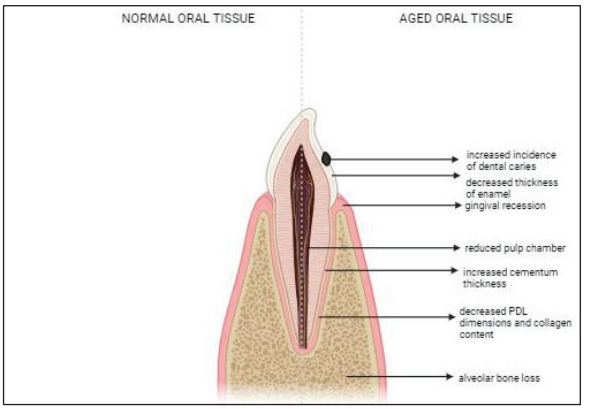

Fig 1. Diagrammatic representation of age changes in tooth and surrounding tissues.

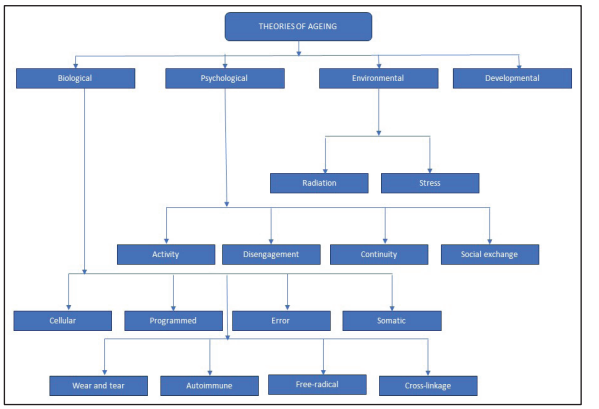

Fig 2. Table depicting theories of aging.

to enamel darkening, another possibility is that the gradually thinning transparent enamel layer may reveal a deeper shade of dentine, which is the layer that thickens with age. There is no denying that as enamel ages, it becomes less permeable. When enamel is still young, it functions as a semipermeable membrane that allows water and other small molecules to slowly pass through the pores between the crystals. As the crystals grow larger and take on more ions, the pores get smaller with age. The changes within this tissue are most prominently reflected in the enamel's surface layer. As aging progresses, ionic exchange with the oral environment alters the composition of the surface layer. Specifically, the surface layer is impacted by a gradual rise in fluoride content, which can be obtained through topical application.[6]

Regarding the effect on the chemical characteristics of the aging enamel, the fluoride-rich layer of "older" teeth will be diminished by the "natural" wear of the enamel surface. Enamel of deciduous teeth contains the highest concentration of fluoride near the incisal edge and the lowest concentration near the cervical edge.[7] But as people age, a significant portion of their surface fluoride is lost. Also, after the age of thirty, the gradient of fluoride concentration shifts and becomes more concentrated near the cervical edge of the teeth.[8]

As enamel ages, its composition and characteristics also alter. Anecdotal evidence suggests that as enamel ages, the density of cracks and craze lines increases. As enamel ages, its hardness and elastic modulus both rise, which increases the brittleness of the indentation. This suggests that elderly patients'

teeth are more susceptible to developing cracks and contact damage.[9]

Biochemical changes

In enamel, increased levels of nitrogen and fluorine cause an increase in the inorganic matrix. Enamel near the surface becomes darker and decay-resistant. There is reduced permeability and enamel becomes brittle. The aspartic acid in human tooth enamel shows an increase in racemization with age. L-amino acids are converted to D-amino acids in racemization. D-aspartic acid accumulates with age.

Age-related changes in dentine

The tooth mesenchyme gives rise to odontoblast cells, which produce the dentine-mineralized matrix. In contrast to enamel, the mineral content of dentine increases with age as a result of ongoing mineral deposition, which can occur as tertiary dentine after injury or as a physiological secondary dentine. The likelihood of tooth fracture is increased by deteriorating enamel and gradual alterations in the mechanical characteristics of dentine.[1] Age-related decreases in sensitivity are correlated with the loss and degeneration of both myelinated and unmyelinated axons. Along with the presence of reparative dentine, there is also an increase in dead tracts and sclerotic dentine, both of which contribute to decreased sensitivity. The tubule diameter gradually decreases as a result of the ongoing intratubular dentine deposition within the dentine. As can be easily observed in a ground section of dentine, this ongoing deposition frequently results in the tubule closing completely because the dentine turns translucent, or sclerotic.[6]

Regarding caries, Jackson and Burch propose that an autoimmune process that disrupts the metabolism of odontoblasts targets these cells; the affected odontoblasts are unable to preserve the integrity of the corresponding protein matrix in the dentine and enamel, leaving the latter open to damage from outside sources. Research indicates that the reason the dentine appears translucent is because the tubules have been filled in with so-called peritubular dentine.[7]

Dentinal tubule diameters from younger and older individuals' teeth have been found to differ significantly from one another. While most of the tubules in older teeth are filled with minerals, the dentine from younger teeth has visibly open tubule lumens. The tubular diameter in coronal dentine varies from 4.9 μm in younger people (between 16 and 30 years old) to 2.9 μm in older people (between 51 and 75 years old). As a result, older people's dentine tubular occlusion reduces the amount of fluid moving within the tubules.[8] The tubule lumens gradually lose diameter as they age because of the progressive mineral filling that occurs inside them. The fatigue and fracture properties of dentine deteriorate with age due to changes in its microstructure. For instance, the fatigue strength has significantly decreased. The coronal dentine's fatigue strength decreases by about 50% for ages loosely defined as young (age ≤ 30) to old (age 55). The mid-coronal third of the root appears to have a less extensive reduction, indicating that aging may vary geographically. A constraint on this understanding is the scarcity of research on the aging process of radicular dentine.[9] Tubule occlusion in older patients resulted in an increase in mineral content in the outer coronal dentine of the dentine.[10]

Physiological changes in cells of dental pulp with age

Dental pulp cells diminish their functions and activities as the pulp cavity narrows with age.[11] The dentine is supported by the pulp, and alterations in pulp age have an impact on the dentine. Similar to all bodily tissues, the dentine-pulp complex varies over time. The most noticeable alteration is the pulp chamber and root canal's declining volume, which is

the result of ongoing dentine deposition. The root canal in older teeth is frequently just a thin channel. Sometimes the root canal can seem to be nearly completely gone. Should pulpal injury arise, the pulp's age dictates its capacity to mend the damage. This continuous restriction in pulp volume likely results in a decrease in the vascular supply to the pulp and starts many of the other age changes observed in this tissue. Young pulps have high rates of cell metabolism, making their cells more vulnerable to damage that can result in altered cell function. However, recovery happens quickly. In young pulps, there is a chance that new odontoblasts will differentiate from the pulp's mesenchymal cells and form repair dentine if the damage is severe enough to destroy the odontoblasts. As one ages, this potential is significantly diminished.[6]

The development of dental pulp calculi, which contribute to the reduction in pulp chamber volume, is another possibility with dental pulp. Decreased pulp sensitivity in the elderly has also been linked to age-related dentinal tubule sclerosis and a decrease in pulp chamber volume. Dental pulp sensibility tests are less useful and produce more false negative results because older patients have fewer nerve branches and more mineralization of the dental pulp nerves. This results in weaker and delayed responses to thermal stimuli. Additionally, as people age, sclerosis of the dentinal tubules will lessen or even completely stop the flow velocity of dentinal fluids, which will further reduce sensitivity.[8]

Age-related changes in periodontium: gingiva, periodontal ligament, cementum and alveolar bone

Gingiva, periodontal ligament, cementum, and alveolar bone make up the complex structure known as the periodontium, which surrounds and supports the teeth. The periodontium's main functions include supporting the tooth's attachment to the jawbone and assisting the tooth in withstanding the force of mastication. Furthermore, the periodontium shields the underlying structure from harmful oral microbiota and prevents damage to the blood vessels and nerves.[12]

Aging has been linked to anatomical and functional changes in the periodontal tissues, such as decreased keratinization and epithelium thinning, while the cementum widens. Alveolar bone and periodontal attachment are lost with aging, although these changes do not significantly affect clinical outcomes. But older people have a more pro-inflammatory state, which makes them more vulnerable to inflammatory, infectious or autoimmune diseases like periodontitis. Aging causes alterations in the oral mucosa's immune and non-immune cells, which compromise tissue homeostasis and hinder wound healing.[13]

In the gingiva, homeostatic regulation appears to be adversely affected by aging. Growing research has shown that the width of the attached gingiva widens with aging, reflecting the periodontium's susceptibility to disease incidence.[14] The loss of attachment may be brought on by gingival recession initiated by cumulative mechanical trauma or gingivitis, and this effect is age-dependent.[15] With age comes a slight decline in periodontal support. The most common way that this becomes evident up is attachment loss, which is typically indicated by gingival recession on the buccal surfaces of ≤3 mm. However, if the tooth is functional and immobile, and the patient is not in pain, attachment loss of more than 3 mm can still be regarded as physiological.[3] Aging is associated with a moderate to severe functional reduction in the periodontal ligament. The previous finding suggested that aging had a deleterious effect on the periodontal ligament's cellular mitotic activity, organic matrix synthesis, and cell and fiber density.[16]

The translucency of the root apex appears to be the most dependable, or the one with the closest straight-line relationship with age, among several tooth changes that are associated with advancing age and are used in determining age for forensic purposes. As age progresses, it appears that the processes causing this translucency gradually extend farther and farther in the direction of the crown until, eventually, the entire root may be impacted.[5] This process appears to be fairly orderly. Alveolar sockets have an uneven, jagged appearance. The size of the alveolar process decreases in edentulous jaws. There is more openness in the internal trabecular arrangement, a sign of bone loss. With age, the distance (roughly 2 points 81 mm) between the crest of the alveolar bone and the CEJ increases. The mental foramen in the upper jaw gets extremely close to the maxillary sinus and the mandibular ridge as a result of tooth loss and alveolar ridge resorption. The alveolar bone gradually and continuously shrinks following tooth extraction.[17] In seven years, the anterior mandibular height decreased overall by 7.87 mm, but interindividual variation increased.[18] The primary clinical outcome of this issue is the inability to fabricate and operate full dentures. Mandibular resorption is more noticeable than maxillary resorption, and many elderly people avoid wearing their lower dentures because they are difficult to manage. Figure 3 shows the pattern of bone loss. Aging cementum is characterized by a steady increase in thickness, especially in the area surrounding the root apex. Furthermore, gingival recession

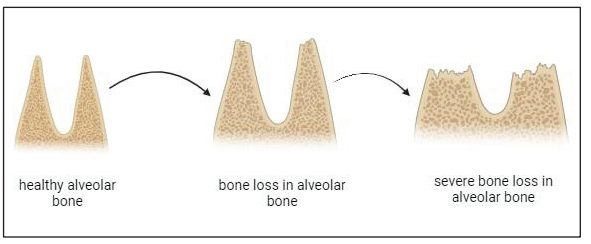

Fig 3. Age-related loss of alveolar bone.

is significantly more common in older people as compared to younger people.[19] Hartmann and Muller additionally indicate that older people exhibit cementum exposure.[20] The incidence of root caries rises with age due to the compromised exposed cementum's low resistance to acidic environments.[21,22] As a result, gingival recession prevention is crucial for maintaining dental health. There is currently no known mechanism to control the aging-related thickening of cementum because the molecular mechanisms of the cementum aging have not been studied in any reports.[11] As people age, their gingival retraction occurs, gradually exposing the cementum that typically lies beneath the gingival lining.[19] This suggests that while aged enamel exhibits decreased susceptibility to caries, the cementum loses its ability to fend off the acid in the oral cavity as we age, leading to an increased incidence of caries at the root.[23]

Age-related changes in flow, consistency and composition of saliva

It is commonly accepted that age-related variations in salivary secretion quantity and quality are ultimately linked to xerostomia in the elderly. The complex biological fluid known as saliva is secreted naturally inside the human mouth and is necessary for the formation of a cohesive, smooth and palatable bolus during eating. By dilution, saliva facilitates the interaction of taste and aroma components in food with the taste buds, thereby playing a significant role in sensory perception. Saliva secretion serves two purposes in addition to those related to eating: it keeps the mouth hydrated and has antimicrobial properties. The physiological effects of aging result in a decrease in the intensity of stimulation and reflex, which alters salivary gland function. Reduced neuronal saliva stimulation (fewer transmitters acting on the receptors), a decrease in blood perfusion at the gland level, and

a reduction in the number of olfactory and taste receptors are all associated with aging. Numerous studies show that as people age, the histological structure of their salivary glands changes due to degenerative processes. This implies that the saliva flow rate may functionally decrease with age. Nevertheless, these studies found no appreciable declines in salivary gland function or salivary flow rate, based on a 3-year longitudinal study on salivary flow rate with healthy candidates who did not use any medications.[24] Conversely, a study involving 540 elderly, healthy individuals revealed a decrease in the salivary flow rate. In this study, the salivary flow rates of the elderly candidates' submandibular and sublingual glands under both resting and stimulated conditions showed a significant decrease. According to another review,[25] older participants' resting salivary flow rate dropped by 44%, whereas stimulated salivary flow rate decreased by 15%. There was an 11% decrease in the flow rate of the submandibular and sublingual glands when they were at rest and a 9% decrease in the flow rate when they were stimulated (25 hours). Figure 4 shows the flowchart of the biochemical alterations in saliva.

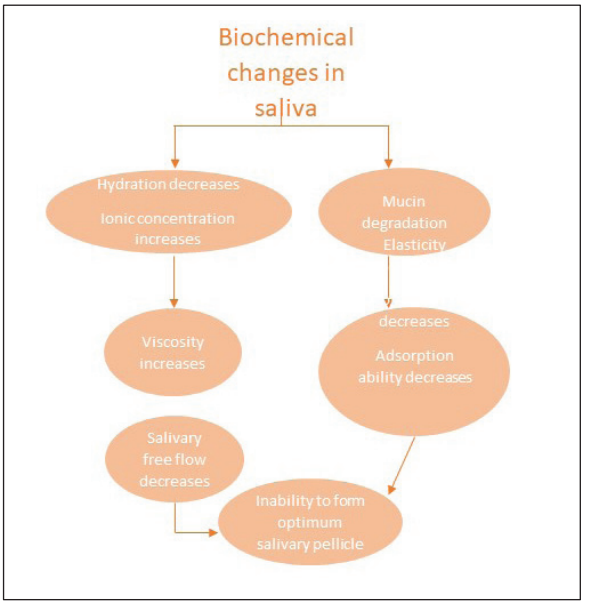

Fig 4. Biochemical changes in saliva.

Histological changes in salivary glands with age

Human salivary glands are divided into three major pairs: parotid, submandibular and sublingual. These pairs secrete 92–95 percent of the saliva secreted, with the remaining saliva secreted by minor salivary glands located in the buccal, labial, palatal, and lingual regions, as well as at the base of the tongue.[26] The largest gland is the parotid, which is behind the lower jaw and in front of the ear. The smallest major glands are the submandibular glands, which are found in the back of the floor of the mouth closure. Sublingual glands are found in the floor of the oral cavity.[27] The three main cell types that make up salivary glands- acinar, ductal and myoepithelial- contribute to salivary secretion into a network of tiny ducts that open beneath the tongue.[28]

According to reports, as people age, the proportions of fatty and fibrous tissue in the major glands increase.[29] Salivary gland histological studies have demonstrated that as people age, the parotid and submandibular glands' proportional volume of fat and fibrovascular tissue increases, despite the fact that the number of ducts in the salivary glands remains the same.[30] However, in older people, the proportional volume of acinar cell secretion was decreased,[31] which is thought to be one of the main causes of dry mouth.[32] Hypofunction of the salivary glands as a whole may arise from all of these histological alterations.[31] There are other known reasons for salivary gland hypofunction in the elderly population besides histological gland atrophy. Moreover, polypharmacy and an increase in age-related illnesses may potentially have an impact on gland function.[33]

Numerous studies have been conducted on the connection between aging and the breakdown of salivary gland structure. According to histological analysis, the mean volume of acini decreased by roughly 25% in the sublingual

salivary glands, 30% in the submandibular glands, and 12% in the parotid glands as people age. Conversely, there was a progressive rise in both fibrotic tissue and lipid droplet infiltration in the salivary glands. Furthermore, ductal dilatation coexists with age-related acinar degeneration in the submandibular glands. The mean proportion of extralobular ducts increased by 80% in the submandibular glands, whereas the mean volume of the striated duct decreased sharply from 60% to 40% of the total duct volume. Additionally, the mean volume of the nonstriated ducts increased significantly. These investigations verified the aging-related degeneration in the salivary gland's parenchyma structures, which may compromise salivary gland function. Apart from changes in histology, aging also results in various physiological alterations that are linked to dysfunction of the salivary gland. For example, a reduction in the quantity of receptors can significantly lower the level of stimulation received by the salivary gland. Salivary gland function can also be compromised by reduced blood flow, impaired neuronal transmission, age-related conditions and medication used by the elderly.[34]

Altered epithelial cells found in the salivary glands that can be identified by their marked granularity, and acidophilia under light microscope are thought to represent an age-related change. The number of oncocytes increases with age.

Physical and histological changes in oral mucosa relative to age

Clinically, the oral mucosa of the elderly is frequently smoother and drier than that of a younger person; it may be characterized as „atrophic" or „friable." However, these changes are probably not due to intrinsic biologic aging of the mucosa; rather, they are likely the result of systemic disease, medication use, or both. Histologically, the epithelium appears thinner, and the flattening of epithelial ridges causes the epithelium-connective tissue interface to become smoother. While research on the rate of tissue turnover and epithelial proliferation in healthy tissue is inconclusive, aging is linked to lower rates of metabolic activity. Age-related reductions in Langerhans cells may be a factor in the deterioration of cell-mediated immunity. Varicosities may appear along with other prominent vascular changes.[6] Many symptoms that affect the oral mucosa, such as burning and strange taste sensations, are typical in older adults, especially in postmenopausal women. These complaints frequently target the area of the foliate papillae, which is the posterior region of the tongue margin. Occasionally, regions of the papilla with a histology that is similar to the faucial tonsils can be found, complete with germinal centres.[5] As we age, our lips and cheeks also develop more sebaceous glands, also known as Fordyce's spots.

A unique mucous layer made up of several papillae covers the tongue's dorsal surface. The tongue is divided into two parts by the V-shaped terminal lingual sulcus: the anterior part, known as the corpus linguae, is located on the floor of the mouth, and the posterior section, known as the radix linguae, is the root of the tongue and is located on the anterior wall of the oropharynx. The lingual tonsils are the lymph follicles located on the tongue's root. The oral floor's muscular structure includes the inferior surface of the tongue. Throughout this area, the mucosa is extremely thin.[35] There are fewer taste buds and taste cells in older people, which is associated with diminished taste function. In addition, the quantity of proliferating taste progenitor/stem cells appears to decrease with age.[36] Any dietary deficiency of iron or B complex vitamins can aggravate changes on the dorsum of the tongue, such as a decrease in the number of filiform papillae and a smooth or glossy appearance. Patients may mistakenly

believe that they have a pathological condition because there are fewer filiform papillae present, which could make the fungiform papillae more noticeable.[6]

Senescence in tissues of the temporomandibular joint

The underlying mechanisms that cause age-related TMJ degeneration are generally unknown. The Geroscience Hypothesis, which is gaining recognition, postulates that slowing down the basic aging process may be able to treat tissue dysfunction and a host of age-related illnesses at the same time. Owing to the TMJ's high occurrence of dysfunctions and special complexity as people age, the geroscience-guided approach may help reduce TMJ degeneration by addressing all aspects of aging. Cellular senescence is one of the fundamental aging processes that has been suggested as a viable target. The essentially stable growth arrest that happens to cells under various stresses is referred to as cellular senescence. Senescent cells, also known as the senescence-associated secretory phenotype (SASP), proliferate in various tissues, including joints, as a result of aging or other pathological conditions. These cells secrete a range of pro-inflammatory cytokines, chemokines, and proteases. It has been suggested that as people age, senescent cells accumulate in the musculoskeletal tissue in a temporal and spatial pattern that compromises the tissue's functionality and exacerbates age-related pathologies. The age-dependent process of chondrocyte senescence may be attributed to a combination of extrinsic and intrinsic factors. The extracellular matrix in the osteochondral tissues of the TMJ is catabolically degraded as a result of the senescent cells' production of pro-inflammatory cytokines.[37]

Histological and dimensional changes in the maxillary sinus

From a clinical perspective, the largest and the most significant paranasal sinus in the orofacial region is the square pyramid-shaped maxillary sinus (antrum of Highmore). Pneumatization is the process by which the growth of the maxillary sinus continues throughout an individual's lifetime. The primary driving force behind pneumatization is the activity of osteoclasts beneath the mucoperiosteal (Schneiderian) membrane of the maxillary sinus.[38,39] Bone quality can be reduced and pneumatization accelerated by age, gender, metabolic disease and endocrine activity. As stated by *Torres* et al., compared to dentate subjects, patients who were completely or partially edentulous had a significantly smaller total maxillary sinus volume. Age had an impact on this result because older patients, irrespective of gender or edentulism status, showed less volume. Age was found to have an indirect relationship with the sinus volume, mediolateral dimensions and meatus distance.[40]

Conclusion

An overview of the various physiological alterations brought about by aging in oral tissues is given in this review. Oral aging is closely related to overall body aging and reflects changes in both areas. Individual variances occur, and various risk factors can alter an individual's rate of aging, including genetics, systemic diseases, smoking, alcoholism, and more. It is possible to propose some basic guidelines for aging to be a healthy process.

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