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REVIEW

Taste disorders in cancer patients and current therapeutic approaches: a narrative review of an understudied topic in supportive care

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Abstract

Background Despite being among the most common cancer treatment-related side effects, taste disorders have received limited study, and their management remains challenging. Recent data have associated dysgeusia with decreased quality of life and increased mortality. We discuss here the state of the science on this topic and address clinical concerns and understudied therapeutics, with the aim of stressing the importance of this side effect and the need for effective treatment.

Methods We selected extant articles covering cancer therapy-associated dysgeusia in English language peer-reviewed literature from PubMed, Medline, Cochrane Library, and Scopus databases. We identified 368 articles, of which 92 were reviewed in detail. The relevant publications have described heterogeneous populations and a multitude of clinical characteristics. The mechanisms of altered taste perception in cancer populations have not been completely elucidated, which in turn has impaired the development of potential interventions, including prevention and management.

Conclusion Dysgeusia in cancer patients may be more relevant to treatment success and quality of life than previously accepted. Unfortunately, this topic has not been adequately addressed and requires further study.

Keywords Cancer therapy · Dysgeusia · Prevention · Treatment

Introduction

Cancer therapy-induced taste disorders have a reported prevalence of 17 to 86%. This wide range is due to differences in assessment (patient-reported vs. objective testing) and the type of cancer and treatments involved [1]. Despite this wide prevalence, the topic has received limited study, and its management remains challenging. Extant articles covering dysgeusia have described heterogeneous oncology populations and a variety of clinical characteristics and associations, thus making any firm conclusions difficult. Additionally, the mechanisms of altered taste perception in oncology populations have not been completely elucidated, which in turn has impaired the development of potential interventions [1]. Recent findings that dysgeusia is associated with reduced quality of life and early death highlight the need for the development of effective interventions for this condition [2].

Many medical conditions and iatrogenic factors have been associated with taste changes. Many of these changes are self-limiting and tend to resolve with discontinuation of the precipitating factor [2–7]. Normal aging has also been associated with the gradual onset of dysgeusia/hypogeusia/ageusia, although recent articles have challenged this relationship [4, 5].

Cancer patients undergoing cytoreductive therapy represent a distinct population, in which taste is potentially affected by numerous factors. Chief among these are regimen-related taste bud damage, therapeutic and supportive medications, salivary qualitative and quantitative changes, and alterations of the oral and gastrointestinal microbiome [8, 9]. Targeted antineoplastic treatments have also affected gustation [10], as have immune checkpoint inhibitors [11, 12]. In combination cancer therapies, multiple mechanisms of taste change may be at work, which further complicates management [13, 14]. Gustatory changes induced by various oncology therapies have not received sufficient study

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to allow a quantitative assessment of their specific role in dysgeusia [1, 2, 13, 14].

A cross-sectional study reported that dysgeusia was present in up to half of the patients on chemotherapy and was associated with diarrhea, anorexia, oral mucositis, nausea, constipation, vomiting, fatigue, and risk of death [15]. A similar study reported that the severity of chemosensory decline in hospice cancer patients is statistically associated with the time of death [2]. These findings suggest that dysgeusia may influence vital factors (nutrition stands to reason) or may be comorbid with dysfunction of other vital processes.

Hormone therapies and immune checkpoint inhibitors have been reported to affect gustation [15]. The impact of dysgeusia upon malnutrition risk has been reported to be 3.36 times (95% CI 2.68–4.02) with chemotherapy, based on patient-reported outcome (PRO) [16]. Taste perception has been shown to impact dietary intake independent of performance status in patients with advanced cancer [17]. In head and neck cancer, taste change is prevalent throughout the cancer trajectory, persists during survivorship, and is associated with energy and nutrient intake and significant weight loss [18]. Specific taste qualities affected in this patient population included decreased umami and fatty tastes and heightened response to salty taste.

A few principles may guide the search for clinical prevention and palliation of taste disorders: First, the ideal solution for detrimental side effects of cancer therapies rests with addressing the underlying cause/mechanism. Second, such solutions must not affect the therapeutic action that the specific cancer treatment has on tumor cells. And third, a preventive approach is always preferable [6, 17, 18]. With these principles in mind, finding effective prevention and/or treatment of taste disturbances becomes quite challenging. In this article, we discuss the current understanding of dysgeusia etiologies, associations, prophylaxis, and therapy, and address the issues that obstruct more rapid progress in this arena. We aim to stimulate interest in the study of dysgeusia mechanisms, with identification of actionable processes that may lead to effective management of this disorder.

Methods

We searched the Medline, PubMed, Cochrane Library, and Scopus databases for English language articles using a combination of subject headings and free words. The period searched was 1990 to 2024. We included the terms Cancer, Chemotherapy, Radiation therapy, Immune therapy, Targeted therapy, and Dysgeusia/hypogeusia/phantogeusia/ageusia/parageusia/taste disorder. We identified 368 articles, of which 92 were retained for full evaluation after reviewing the abstracts. These latter articles informed the content of this narrative review.

Discussion

Physiology of taste

Taste (gustation) is a perception derived from afferent stimuli originating mostly from the mouth and oropharynx. It is typically triggered by exposure of sensors to tastants [19]. Taste consists of five well-defined basic qualities: sweet, bitter, salty, sour, and umami (savory) [20]. Additional taste qualities have been proposed, including fat taste, which may be mediated by receptor transduction and/or nonspecific transport across the cell membrane [21] and spicy taste (e.g., capsaicin) mediated by small nerve fibers. Separate receptors may be activated in *Kokumi* taste, which enhances additional flavor sensations. A water/fluid receptor has also been postulated [22].

Numerous specialized receptors exist on tongue papillae and other gastrointestinal mucosal sites, primarily within the oral cavity. Extra-oral sites include the nose, esophagus, stomach, pancreas, and lungs. The taste system is highly redundant, with bilateral distribution and transmission along multiple cranial nerves. Current evidence showed that the entire human tongue perceives each of the taste qualities. Nevertheless, there are areas such as the tip and the rear of the oral tongue that display higher taste detection [4]. The role of taste buds at other mucosal locations has not been clearly defined.

Recently, the protein sonic hedgehog (Shh) has been associated with the function, growth, and development of taste buds. Salivary levels of Shh correlate with taste perception and recovery after disruption [23]. Loss of Shh can cause receptor differentiation failure, renewal issues, and/or loss of function. Cancer treatment with Hedgehog pathway inhibitors can lead to taste alterations or loss [24].

Taste disorders

The most applied method of classification of taste disorders in clinical practice separates qualitative from quantitative disturbances [1, 3, 25]. The nomenclature of the disturbances is as follows:

- Dysgeusia: the general terminology for any kind of taste disorder
- Parageusia: qualitative impairment, which reflects a triggered inappropriate taste distortion (e.g., bitter or metallic)

- Phantogeusia: qualitative impairment, which delineates a non-triggered, permanent, or intermittent distortion, akin to phantom pain.
- Hypogeusia: a quantitative reduction of taste.
- Ageusia: a quantitative disturbance reflecting absence of taste.

A few mundane factors can impact the severity of taste dysfunction, except for ageusia, which is typically constant. These include liquid intake, and specific foods and medications, as well as hormonal imbalances. These disorders can occur alone or in clusters (e.g., metallic parageusia can occur within otherwise normal taste, or be associated with other altered taste qualities).

Partial etiology of taste disorders

A variety of physiological and/or pathological conditions may produce taste disorders. For many of those, mechanistic processes await elucidation. The complexity of intrinsic and extrinsic factors at work makes undoing this Gordian knot quite daunting. While some dysgeusias may have obvious causes (neurological factors such as cerebrovascular accident or cranial nerve disease), most remain idiopathic and are likely multifactorial.

Sensory disorders may accompany classic neurological diseases (e.g., stroke or peripheral nerve disease). Taste alterations can also be an early warning of significant or life-threatening illness, including amyotrophic lateral sclerosis, multiple sclerosis, lung cancer, myasthenia gravis, or COVID-19 [26–32]. More than 200 medications have been reported to have gustatory effects [33]. Surgical procedures that are close to or affect the afferent neural pathways may also produce taste alterations [34, 35]. One or more of these etiologic factors may be involved in cancer therapy-induced taste changes. Discerning which factor is responsible for what aspect of dysgeusia in a cancer patient is challenging but may help direct the appropriate intervention(s) [33, 34].

Dysgeusia associated with cancer therapy

Prevention of mucositis may be associated with reduced taste damage, suggesting that direct mucosal injury affects the taste organs and associated nerves [2, 6, 9]. Radiation therapy may damage the mucosa, including disruption to taste receptors, regional nerves, and oral microbiome. Chemotherapy may act mostly through topical effects due to oral secretion and systemic exposure of the receptors, conduction, and the central nervous system. Taste change may therefore be either a peripheral signal transmission, a central change, or, in selected cases, both [13–15].

Cytotoxic pharmacological agents may directly damage epithelium, including taste buds and adjacent mucosa, as these tissues have a relatively high turnover rate. While nerve cells are relatively resistant to chemotherapy, selected drug classes such as taxanes and platinum agents do cause neurotoxicity [13, 14]. Chemotherapy-induced microbiological changes in the oral cavity may also contribute to gustatory dysfunction [9]. Higher drug concentrations in saliva may correlate with increased subjective dysgeusia [36, 37].

There are conflicting reports regarding which chemotherapy agents are associated with a higher prevalence of taste disorders. In general, the effect of chemotherapy on taste correlates with the medication used and its dose and duration of treatment [38, 39]. Cisplatin has been shown to inhibit growth and differentiation and promote apoptosis of taste receptor organoids [40].

An evaluation of "metallic taste" in cancer reported a prevalence of 29% and a sizeable impact on nutrition [41]. As a limitation, assessment of oral health, including candida overgrowth, was not reported, particularly as oral candidiasis is associated with metallic taste. Further study of this taste alteration is necessary.

In hematopoietic stem cell transplantation (HSCT), patients treated with conditioning protocols receive high-dose chemotherapy. Specific regimens may also include total body irradiation (TBI). Reportedly, 80% of this group of patients had a decrease in sweet taste, and no patients could identify umami taste [42]. Expectedly, there was significant myelosuppression with resultant neutropenia, anemia, and thrombocytopenia in this population. Supportive therapy consisting of analgesics, anti-inflammatories, sedatives, anti-microbials, and targeted drugs was also commonly used.

In allogenic HSCT, post-transplantation graft-versus-host disease (GVHD) may significantly involve oropharyngeal tissues including mucosa, taste receptors, and salivary glands [36]. Management of GVHD may also impact taste. Thus, it is likely that multiple mechanisms are involved in dysgeusia in HSCT patient populations [36–40]. As most complications after HSCT develop in clusters, it remains difficult to identify the exact associations and causative mechanisms.

Of note is the effect that hedgehog inhibitors like vismodegib and sonidegib have on taste. Shh is vital for the maintenance of homeostasis in epithelial cells and in epithelial-mesenchymal communications. However, Shh is also intimately involved in various tumors' growth, including oral squamous cell carcinoma [23, 24]. Hedgehog pathway inhibition used for tumor control results in the loss of both nerve cell-derived and salivary-derived Shh, thus producing severe dysgeusia. Most taste characteristics are usually recovered after the cessation of treatment, with recovery from Hedgehog/Smoothened inhibition with the cancer drug sonidegib [43].

Dysgeusia associated with radiation therapy (RT)

Ionizing radiation is an integral part of many antineoplastic regimens, used as primary and/or adjuvant therapy. It is a non-specific cytotoxic therapy used in both hematology and oncology. Cell killing is produced mostly by sublethal damage to the cell structure and its nuclear contents, followed by apoptosis. The two main methods of radiation delivery are external beam (EBRT) and internal radiotherapy [44, 45]. The latter includes radioactive implants and brachytherapy, whereas the former encompasses parallel ports, dimensional conformal radiotherapy (3DCRT), intensity-modulated radiation therapy (IMRT), volumetric modulated arc therapy (VMAT), stereotactic body radiation therapy (SBRT), image-guided radiotherapy (IGRT), and particle therapy (PRT) [46]. Systemic administration of radioactive iodine used in the treatment of thyroid cancer can also impact saliva and taste function [35]. Regional radiation therapy, particularly in head and neck cancer, has a direct impact upon taste function. A prospective study found 88% of patients reported moderate to severe taste disturbance while taste testing revealed some change in all patients, with umami taste loss, reduced salt taste, and total general loss identified [42]. A recent review reported taste change in 97% of HNC patients at week 4 [47]. Persisting dysgeusia may be related to taste bud, neurological, and/or salivary change, and alterations in the local microbiome.

Radiation can directly damage taste buds, nerve cells, and salivary glands, especially when it is directly targeted to the head and neck. It may also alter the structure of taste pores, leading to a disrupted delivery of flavor molecules to receptor cells, or a thinning of the papillary epithelium. The hypotheses to explain ionizing radiation-induced taste impairment include inflammation of afferent nerves (neuropathy) that supply taste buds, direct damage to differentiated cells, and ablation of proliferating progenitors, preventing the renewal of taste cells and taste function. The loss of taste progenitor cells may also result in a reduced recovery rate of damaged taste buds [44]. By virtue of its production of reactive oxygen species (ROS), RT can also cause inflammatory cytotoxicity and neurotoxicity [45]. Addition of adjuvant chemotherapy for radiosensitization (chemo-RT) has been associated with increased severity of side effects, including taste dysfunction [48].

Radiation hyperfractionation has been associated with an increased profile of side effects, including taste, while SBRT, IGRT, IMRT, and PRT produced less dysgeusia. Currently, head and neck cancer RT is delivered via IMRT or IGRT, while PRT is increasingly available, but currently used in a small number of centers [49, 50].

In head and neck cancer, dysgeusia is typically reported starting in the fourth and fifth weeks of treatment, when more than 40 Gy has been delivered to the tongue. Recovery of sensation generally begins within 6-month post-therapy, but deficits may persist indefinitely, often for the rest of the patient's life [46, 47]. Changes in taste during radiation may be variable but generally include a decrease in sensation of sweet, salt, and umami [51].

Cancer therapy effect on saliva

Taste is an important stimulus for the production of saliva, and in turn, saliva supports the perception of taste. Saliva is the natural oral solvent for food components to reach the taste pores and taste receptors. Salivary composition may also affect taste, as a specific tastant quality must be above normal saliva concentration to be perceived [6]. Thus, alterations in both saliva quantity and quality can influence gustatory sensation [52–54]. The potential impact upon the oral microbiome has had limited evaluation in relation to taste disorders and may be impacted by altered saliva function [54].

Major salivary gland tumors will indubitably affect secretion. However, more commonly, cancer therapies produce profound alterations in both quantity and quality of saliva through pathologic, psychological, and iatrogenic mechanisms [54]. While most chemotherapy-induced salivary changes are reversible upon treatment discontinuation, many patients have long-term dysfunction [55].

Immune checkpoint inhibitors may trigger autoimmune reactions that directly alter the exocrine glands. This type of impairment typically includes a sicca syndrome associated with severe mouth and eye dryness and appearing 3 to 6 months after treatment initiation. Separately, direct immune-mediated neuropathy may also affect taste function. While these effects usually ameliorate after treatment cessation, they tend to persist for a lifetime [56]. Other current targeted therapies may affect salivary production, but the effect is typically less pronounced. For example, talquetamab, a bispecific antibody binding to GPRC5D receptors, affects taste and saliva production in a majority of treated multiple myeloma patients [57], which may represent a class effect of new antibody drug conjugates.

RT has a direct effect on the acinar cells of the serous salivary glands, which produce about 70% of the salivary volume. This toxicity typically results in permanent damage in patients receiving cumulative RT doses >40 Gy to the parotid region. The less pronounced effect of radiation on the mucous glands accounts for the clinical detection of ropey, thick saliva responsible for dysphagia, nausea, and vomiting [50–52]. Introduction of computer-aided technology in RT delivery has contributed to a significant reduction in the severity of side effects, including hyposalivation. Programs are built to spare the parotid glands to the extent possible [44, 49]. PRT has the lowest profile of deleterious effects on salivation [46, 58] based upon dose distribution radiation.

Oral infection

The oral cavity is home to millions of microorganisms and is a common location for infection in cancer populations. Significant microbial shifts typically occur due to therapy [36]. In patients with mucositis, oral care is compromised, microbial loads increase, and bacteria shift to a more anaerobic flora [36, 59]. Required antibiotics may lead to further shifts in the microbiome. In the setting of immune compromise, systemic spread of the infection is also possible. These changes are likely to influence taste sensation [59]. Overgrowth of anaerobic bacteria may produce an excess of sulfur-containing metabolites, which are responsible for halitosis and altered taste. Viral organisms, particularly those from the Herpesviridae, may produce chronic lingual ulcerative lesions that destroy the existing taste buds. These sensory changes are typically transient and may dissipate with the resolution of the infection [59].

Other factors

Drugs used to treat complications of cancer therapy can also cause dysgeusia. Anticholinergic medications used to prevent or treat nausea, vomiting, and/or diarrhea have antisialagogue effects, as do tricyclic antidepressants, antihistamines, and opioid analgesics. Clinical depression and its medical management have also been identified as possible causes of dysgeusia [5].

Dysgeusia in pediatric oncology

There is a dearth of publications addressing taste changes in children treated for malignant disease. Additionally, results appear to be widely variable. The prevalence of dysgeusia in this group is reported to be high, but heterogeneity in methodology and populations prevents robust conclusions [60]. While some studies reported more severe dysgeusia during treatment, followed by a relatively rapid return to baseline, others [61] showed no dysgeusia score change during active therapy. However, self-reported taste changes reached a prevalence of 80%. The authors did not provide a solid explanation for this large discrepancy. A different study of 502 subjects reported a prevalence of 45% in taste change, with 10% being severe [62].

Most authors agree that older age, nausea, vomiting, and mucositis were associated with worse outcomes, and that dysgeusia was associated with decreased food intake and lower QoL. Food aversions are likely to develop in severely affected children [63]. Symptoms improved in most children with discontinuation of therapy [60–63] .

Prevention and treatment (Table 1)

Dietary supplements

- Zinc is important in taste due to its presence in taste buds and its role in cell division and protein metabolism. However, studies in taste management in cancer patients have shown mixed results [13]. Small clinical trials with objective endpoints suggest a benefit of zinc supplementation in taste management [64, 65]. The variability of results may depend on the zinc supplement used, the dose and duration of treatment (typically 100–150 mg elemental zinc/day), the bioavailability of zinc, the patient populations studied, and the outcome measures used [65]. Further study of potential prophylaxis and dysgeusia treatment with zinc is needed.
- Polaprezinc (zinc L-carnosine), known as a GI protector with potential reduction of TNF-alpha, has also shown mixed results in small trials. Data suggest a potential prophylactic effect on taste dysfunction [66, 67].
- Glutamine is an amino acid that is involved in nucleotide synthesis and has been assessed for potential protective effects in peripheral neuropathy and in taste. Conflicting results have been reported [13, 32]. Stasser et al. [68] reported a controlled trial using PROs. After 25% of patients dropped out of the study, there was no effect of glutamine upon taste. Shono et al. [69] investigated the mono-sodium glutamate supplementation effect on the expression of taste receptors of umami and sweet and found a reduction of chemotherapy-induced dysgeusia. The authors suggested the effect was due to maintaining taste receptor activity (t1R3). While encouraging, more data are needed on this topic.
- Lactoferrin has been studied for its impact on taste and smell due to its potential effect on metallic flavor [13]. This milk protein has immune-modulating and anti-inflammatory effects, which may alter the gut microbiome and may decrease lipid oxidation, thus reducing unpleasant flavors [70, 71]. A study using PROs in 25 patients (with 19 completing the study) on lactoferrin supplementation (750 mg/day) found statistically improved taste scores from baseline [72].
- Fish oil (6 g/day) has no significant effect on taste according to a small study [13].
- Miracle fruit (*Synsepalum dulcificum*) has been reported to change the taste of food from acid to sweet. A small randomized study showed improvement in taste perception in a small number of cancer patients with 2-week use six times a day [72]. Another study found improvement in only 34% of the patients, and a third one noted improved sour perception but no overall taste improvement [73].

Table 1 Potential approaches to taste management

Potential Approaches for Taste Management			
Management	Agent	Comment	References
Supplement			
	Zinc	100-150mg elemental zinc/day	13,64,65
	polaprezinc	Zinc-L-carnosine	13,32,68,69
	glutamine		13,32,68,69
	lactoferrin	750 mg/day	13,70–72
	glutamate		74,75
	MSG	2.7 g/day	76.77
	Salt		78
Medication			
	theophylline	Hedgehog pathway	79
	amifostine	Radio-protection	80
	megestrol	480 mg/day	81
	cannabinoids	THC:CBD	82–84
Taste Change			
Bitter blockers	methylflavone		93
	valsartan, ghrelin, topiramate		95
Sour/sweet	miracle fruit	Synsepalum dulcificum	72,73
Neuropathy			
	clonazepam		88
	gabapentin		89
Photobiomodulation		red/infrared light	90–92
Saliva stimulation		Sialagogues/PBM	90–92, 96–106
Dietary Management			

- Glutamate acts as a gustatory stimulus of several taste receptors as well as a neuromodulator, upregulating taste-1 receptor (T1R3) gene expression. It also impacts neurotransmission via modulation of neurotransmitter release [74]. Salivary glutamate levels have been related to pleasant taste, and low levels have been associated with an increase in unpleasant taste [75].
- Monosodium glutamate (MSG) may interact with kokumi taste receptors, enhancing the intensity of umami, sweet, salt, and fatty taste [76]. The effect of MSG was assessed in 25 HNC patients (2.7 g/day vs. control). The treatment led to increased expression of T1R3, which subserves sweet and umami receptors, and increased taste sensitivity [77].
- Salt (sodium chloride) has been shown to block some bitter flavors, thought to be related to decreased signaling of taste receptor TAS2R16, suggesting the need for continued study of taste blocking with topical intervention [70].

Medications

- Recent research suggests that activating the Hedgehog pathway, potentially through stimulating Shh, could help regenerate taste buds and restore taste function in cases of taste loss. For example, theophylline has been shown to increase saliva's Shh levels and improve taste dysfunction [78]. Further study is warranted.
- Amifostine has been used as a radioprotector related to its function as a free radical scavenger. Improvement in mucositis and taste was reported in patients treated with 500 mg prior to chemo-radiotherapy in HNC, suggesting prevention of mucositis and preservation of salivary function, and potentially maintenance of taste function. However, side effects of amifostine (nausea and vomiting; blood pressure drops) were prevalent and often severe [79].
- Megestrol (480 mg/day) in patients with advanced cancer may enhance appetite and taste, specifically impacting umami taste by acting as an agonist of progesterone receptors [80]. Effects on taste were shown in a controlled trial of patients with head and neck cancer, suggesting significant improvement in taste and smell.
- The endocannabinoid system plays a role in food reward perception. In 10 healthy males, tetrahydrocannabinol/cannabidiol (THC/CBD)-rich or placebo cannabis was delivered by inhalation in doses of $4 + 1$ mg or $25 + 10$ mg or placebo, in three sessions set 2 weeks apart. No difference in taste intensity was seen, but the

desire to eat was increased with THC/CBD treatment [81].
- THC was examined in a small study of 11 subjects and found to have a chemosensory impact upon taste from baseline, with enhanced taste perception and increased appetite in adults with advanced cancer [82]. No control group was studied.
- A meta-analysis assessing appetite-related symptoms in cancer patients treated with dronabinol, nabilone, and cannabis extract included five studies. There was a positive effect upon taste perception in one study and limited evidence of increased appetite [83].
- Twenty-two patients who received CBD (300 mg/day) for 8 days, and 10 control subjects were followed for three cycles of chemotherapy, with the CBD group showing a significant difference in assessing weak and strong sweet taste ($p = 0.03$) and weak and strong saltiness ($p = 0.04$), suggesting an impact on taste changes associated with platinum-based chemotherapy [84].
- A systematic review of controlled trials reported a reduction in chemotherapy-induced nausea and vomiting (CINV) with cannabinoids compared to placebo but found insufficient evidence for an effect upon taste disturbance [85].

Neuropathy management

Taste dysfunction in oncology patients may be associated with neuropathy. This has been suggested by results from studies of oral mucosal burning sensation and recovery of taste function. Clonazepam for the treatment of burning mouth syndrome showed that improvement in oral burning pain was associated with decreased bitter taste [86]. Similarly, the management of oral burning pain was associated with improved tasting of salt and bitter in patients treated with topical clonazepam [87]. Gabapentin was reported to improve taste recovery following chemotherapy in two patients with glioblastoma [88].

Photobiomodulation (PBM) therapy

PBM with red/infrared light has received increasing attention for prevention of loss and management of taste recovery representing a potential effect by prevention of damage and accelerating repair of mucosa and taste buds [42, 89–91]. Due to encouraging but variable results, further study is needed.

Bitter blockers

Masking of taste, specifically bitter taste, has been examined as a strategy for use in oral pharmaceutical therapy and to block the taste of salivary secretion of medications. 6-Methylflavone reportedly reduces bitter sensation [92] while sodium acetate, sodium gluconate, and adenosine 5′-monophosphate have been considered, but clinical trials remain needed [93]. Other proposed but insufficiently tested medications include lidocaine and P2X2/P2X3 purine receptor inhibitor AF-353 (5-(5-iodo-methoxy-2-propan-2-ylphenoxy)pyrimidine-2,4-diamine). Valsartan, ghrelin, and topiramate may affect response to bitter taste by binding to bitter receptors, resulting in a blocking effect [94].

Saliva stimulation

Hyposalivation has been associated with taste change in oncology and should be assessed in patient care [55]. Studies of saliva substitutes have not shown a positive impact upon taste in the oncology setting, and studies of salivary gland stimulation have shown conflicting results [55, 95, 96]. Some studies have shown that dysgeusia/ageusia is related to xerostomia and is more common in cancer patients with dry mouth [97–101], whereas others did not detect any impact [102]. As most studies identified a relationship between saliva and taste, the assessment and management of hyposalivation are indicated in the case of taste change and for symptoms of dry mouth and oral health [103, 104].

Dietary interventions

Nutritional/dietary interventions include diet modification avoiding unpleasant flavors, increasing desired flavors, adding flavor enhancers to food, changing the temperature of food, and small frequent meals. Cooler temperatures may be helpful with volatile foods. Food modification with spices, marinades, seasoning, and sauces has been used. Avoiding unpleasant smells of food preparation and service may add benefit. Modifying the texture of foods may support oral intake as sensory changes, including sensitivity to roughness and temperature, may be present [103]. Using non-metal utensils may reduce metallic taste. Oral hygiene maintenance and avoidance of alcohol-containing mouthwash are recommended [104].

Management summary

There is a relative dearth of robust research examining prophylactic and therapeutic measures related to gustatory deficits. General prevention of injury to normal tissues includes those important to sensorial function, such as mucosa, nerves, salivary glands, and the immune system. Additionally, good oral hygiene and appropriate dietary modifications are commonsense measures required for improved outcomes. Currently, there are no strongly supported approved medications or non-pharmaceutical methods for the prevention or therapy of taste side effects in cancer patients, and more research is needed.

Pearls for practice

As taste alterations associated with cancer therapies have a complex etiology, pinpointing specific causes is a daunting task. Nevertheless, identifying the most likely cause(s) and understanding the applying pathophysiology are of utmost importance when deciding what treatment may be appropriate. Supportive therapies targeting the most likely etiological factors are the most likely to succeed [103].

In the prophylactic arena, maintenance of homeostasis in the oral cavity is a must. Good oral hygiene is essential, and additional control of the microbial flora can be achieved with selective antimicrobial agents, when necessary [104].

Herpes virus reactivation can be largely avoided by the use of preventive antivirals in patients who have pre-existing positive titers and/or a history of recurrent infections. Similarly, prophylactic antifungals can mitigate the overgrowth of opportunistic organisms like *Candida* species. The tongue and major salivary gland shielding from radiation therapy is a simple and effective way of preserving saliva production and impacting taste function in head and neck radiation patients [103, 105].

When hyposalivation is present, sialagogues (e.g., pilocarpine, cevimeline, bethanechol) may provide stimulation of residual salivary function, mitigating changes in microbiome and providing substrate for tastant access to taste receptors. In addition, saliva contains epidermal, fibroblastic, and nerve growth factors that play a role in mucosal and potential neuro-epithelium repair. However, keeping in mind that secretion of cytotoxic medications in saliva is possible during chemotherapy, sialagogues may be best considered after systemic cytotoxic drug elimination [103].

Sour foods or candy should be avoided due to their acidic components that can irritate mucosal surfaces and increase demineralization of teeth. Also, chewing gum may be considered, recognizing that it may lead to soft tissue trauma, particularly when mucositis is present, or trauma occurs with dry mucosal surfaces while chewing [103, 105].

Taste changes in the setting of oral or nasal infection may be alleviated by antimicrobial therapy. Neural damage may respond to neuromodulator medications such as gabapentin, pregabalin, and clonazepam.

Supportive methods that are inexpensive and largely free of side effects, and have shown evidence of efficacy should be used liberally, both as prophylactic and therapeutic modalities. Cryotherapy and PBM (LLLT) have been well tolerated by a vast majority of cancer patients and should be considered for wide use [103, 104]. Zinc and other supplements including monosodium glutamate and glutamine have shown mixed results but, due to their mild side effect profile, can be considered in patient care. Systemic medications including megestrol and cannabinoids have shown potential efficacy in the study of taste and appetite. Dietary modifications should be considered in relation to oral condition, saliva function, and swallowing function, which may require collaborative and multidisciplinary patient care for the best outcomes [103, 105].

Due to multiple mechanisms involved in taste dysfunction in oncology, it is likely that a combination of approaches to management is needed for the best outcomes. For example, salivary flow increase, oral hygiene, interventions directed at taste function, and dietary/nutrition management may need to be concomitantly applied.

Future directions

It is likely that sensory side effects in oncology patients are regimen- and patient-specific. The literature addresses common radiation and cytotoxic therapy, and new agents including targeted therapies and immunotherapies are increasingly recognized as driving taste dysfunction.

Acquiring new data at the cellular and molecular levels could clarify the pathways of therapy-induced sensory deficits and point toward targeted interventions. Clinical studies in higher-risk cancer therapies, including radiation-induced sensory dysfunction and chemotherapy, targeted therapy, and immunotherapy protocols, should examine the effects of combined preventive and therapeutic strategies covering protection and regeneration of all cell types involved in taste. In addition, developing more targeted cancer treatments that spare sensory neural components may avoid this type of complication. As our knowledge of the morbidity of sensory dysfunction develops, renewed exploration of this topic comes into focus and must be pursued.

Endpoints of future clinical studies should be clearly defined. In addition to patient-reported outcomes, future studies should incorporate well-validated objective taste and smell tests in addition to standardized patient-reported outcomes. While many studies are sophisticated in other ways, their use of unreliable tests that fail to control for response biases undermines their effectiveness. Oral assessment, including mucosal health, saliva function, and oral hygiene, should be assessed and considered covariables in all studies.

Author contributions A.B. and J.E. reviewed the literature and wrote the main manuscript text. All authors reviewed and edited the manuscript.

Data availability No datasets were generated or analysed during the current study.

Declarations

Competing interests The authors declare no competing interests.

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