REVIEW



Can taste be ergogenic?

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Abstract

Taste is a homeostatic function that conveys valuable information, such as energy density, readiness to eat, or toxicity of foodstuffs. Taste is not limited to the oral cavity but affects multiple physiological systems. In this review, we outline the ergogenic potential of substances that impart bitter, sweet, hot and cold tastes administered prior to and during exercise performance and whether the ergogenic benefits of taste are attributable to the placebo effect. Carbohydrate mouth rinsing seemingly improves endurance performance, along with a potentially ergogenic effect of oral exposure to both bitter tastants and caffeine although subsequent ingestion of bitter mouth rinses is likely required to enhance performance. Hot and cold tastes may prove beneficial in circumstances where athletes' thermal state may be challenged. Efficacy is not limited to taste, but extends to the stimulation of targeted receptors in the oral cavity and throughout the digestive tract, relaying signals pertaining to energy availability and temperature to appropriate neural centres. Dose, frequency and timing of tastant application likely require personalisation to be most effective, and can be enhanced or confounded by factors that relate to the placebo effect, highlighting taste as a critical factor in designing and administering applied sports science interventions.

Keywords Taste · Carbohydrate · Caffeine · Menthol · Capsaicin · Bitter

Introduction

Taste is a homeostatic function that aids in deciding what to eat, and acts as a precursor for digestion [1]. Human taste and preferences are evolved due to nutrient availabilities within our ancestral environments [2], where they conveyed information, such as energy density, readiness to eat, or toxicity [1, 3]. Despite being the area most densely populated with taste receptors, taste is not strictly confined to the oral cavity, but frequently incorporates other sensory inputs from the upper digestive tract and auditory, olfactory and visual systems [1, 4–9]. This is most evident in those who suffer

with ageusia (loss of taste), or anosmia (loss of smell), and still respond physiologically to tastes [3, 10], demonstrating taste as a chemical interaction between a chemesthetic agent and receptors, which drives either ingestion or aversion and accompanying hedonic sensations.

Assessment of the physiological responses to taste has not escaped sports scientists, with many 'tastes' now investigated within the literature [11–15] with a view to attenuating fatigue or improving physical or cognitive performance. Depending upon the tastant investigated, impressions of energy availability [16, 17], thermal perceptions [11, 12, 18] and central drive [15, 19] may be altered. Secondary outcomes may also include modifications in autonomic function [20–22], thirst [23, 24] and ventilation [25–27], with further downstream effects depending upon whether tastants are ingested or simply rinsed around the oral cavity and expectorated.

These outcomes are likely to be useful to athletes, but depend heavily upon their exercise modality, prior exposure to and preference for specific tastants, as well as the availability of tastants during an exercise bout. Placebo effects associated with tastants cannot be excluded and indeed may be maximised by including a carefully chosen taste component in personalised sports nutrition interventions

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or matching tastes of interventions to other sensory expectations, such as colour [28, 29]. Previous work has asked whether "the [central] governor has a sweet tooth" [14]; in this review, we explore the ergogenic potential of different tastes administered prior to and during exercise performance. We also raise the question of whether the ergogenic benefits of taste are attributable to the placebo effect. Recommendations for athletes, practitioners, and future research directions are also provided throughout.

Sweet and bitter tastants and athletic performance

Carbohydrate

The efficacy of carbohydrates as a means of supporting endurance performance is well established [30]. However, a clear, over-riding mechanism by which carbohydrate enhances performance is currently unknown; during exercise, only about a quarter of ingested carbohydrate enters peripheral circulation [31], with exogenous carbohydrate demonstrated to contribute only a small proportion of the carbohydrate oxidised during the late stages of prolonged exercise [32]. This lack of a clear metabolic mechanism leads to speculation that the consumption of carbohydrates during exercise may stimulate central pathways associated with sensations of reward or energy availability, which in turn has a performance-enhancing effect [33]. To test this hypothesis, researchers allowed subjects to rinse a carbohydrate solution around the mouth, but not ingest it, removing the metabolic effects of carbohydrate on performance. In the last decade, an exponential increase in research on this topic has been carried out, with a number of reviews [14, 33, 34, 36, 36] demonstrating a clear ergogenic effect of a carbohydrate mouth rinse on endurance performance, particularly in glycogen-depleted participants.

Given that little carbohydrate is absorbed in the oral activity during mouth rinsing, the mechanism(s) by which carbohydrate mouth rinses enhance performance are likely to be central in nature [14]. The tongue contains a number of taste receptors capable of detecting sweet stimuli [37] and these taste receptors when stimulated activate dopaminergic pathways and reward centres within the brain [17, 38]. In turn, this increase in reward may enhance motivation to exercise, allowing the athlete to self-select higher exercise intensities, and reducing the impact of peripheral fatigueassociated signals under both the central governor [39] and psychobiological [40] models of fatigue. There may also be a feedforward effect, whereby the activation of oral carbohydrate receptors suggests that energy is being consumed, allowing for an increase in exercise intensity, although this hypothesis has yet to be experimentally tested.

At present, it appears that the ergogenic effects of a carbohydrate mouth rinse are not taste related per se. This is demonstrated by the fact that tasteless carbohydrates, such as maltodextrin, are ergogenic in a mouth-rinse solution [36], and also activate brain regions similarly to sweettasting carbohydrates, such as sucrose [17]. Similarly, artificial sweeteners provide a sweet taste, but a far smaller activation of key brain regions compared to sucrose [41]. Accordingly, it seems likely that it is the carbohydrate binding to as-of-yet unidentified oral carbohydrate receptors, as opposed to taste itself, that drives the ergogenic effects of a carbohydrate mouth rinse [14].

Bitter tastants

Building on the potential ergogenic effects of a sweet taste as mediated by carbohydrate rinsing (detailed in Sect. 2.1), Gam and colleagues explored the use of bitter tastants on exercise performance (reviewed in Gam et al. [19]). The potential relationship between bitter taste and enhanced exercise performance has a strong molecular underpinning, given that bitter tastants activate similar areas of the brain as sweet tastes [42], with these brain areas being implicated in aspects, such as motor control and the processing of emotions [19].

In their first study exploring the ergogenic effects of a bitter tastant, Gam and colleagues [43] administered 14 competitive male cyclists with a bitter solution containing 2 mM quinine, which was rinsed in the mouth for 10 s and then ingested. The quinine solution enhanced mean power output in a 30-s maximum cycle by 2.4% compared to an aspartame (sweet taste) mouth and by 3.9% compared to water. In a subsequent study [44], a stronger concentration (10 mM) of quinine was utilised, but the solution was only rinsed around the mouth and not ingested. In this scenario, there was no ergogenic effect of the bitter solution on a 30-s cycle sprint, suggesting that the ingestion of the bitter solution is potentially important. The proposed mechanism underpinning the need for ingestion is that there are an increased number of bitter taste receptors beyond the oral cavity in the upper gastrointestinal tract [45] which are not activated following mouth rinse only. Outside the work of Gam and colleagues [43, 44, 46], there is little additional research exploring the ergogenic effects of a bitter tastant, and so further research in this area is warranted. This would be particularly pertinent from a practical approach, with strong bitter tastants-such as those used in the research by Gam and colleagues—able to induce nausea in some subjects upon ingestion [43]; given this information, further research exploring the optimal intensity of the bitter taste would likely be very useful.



Caffeine

Given the demonstrated ergogenic effects of an ingested bitter tastant [43, 46], Pickering [15] recently reviewed whether caffeine—itself a bitter tastant [47] that has been shown to activate bitter taste receptors located in the oral cavity [48]—exerted some of its well-established ergogenic effects [49] via its bitter taste. A small number of studies [50–56] have utilised a caffeine-mouth rinsing protocol as a method to enhance performance. Studies that demonstrated an ergogenic effect employed a repeated 6-s Wingate sprint protocol [50, 53] or a self-paced endurance effort over 30 min [56]; whereas investigations that showed no effect employed either fixed work rate [51], progressive running [55] or repetitions to failure [52] models. Whilst the results are currently equivocal, there is a trend for no-demonstrated performance enhancement when caffeine is rinsed around the mouth for both endurance and high-intensity exercise [15]. The reasons for this are currently unclear; it may be that caffeine's bitter taste is not ergogenic that the caffeine solutions utilised were not sufficiently bitter to evoke an ergogenic effect or that of like quinine [44], ingestion of caffeine is required for its bitter taste to be ergogenic [54]. However, caffeine mouth rinses have been demonstrated to improve cognitive function during exercise [57] and limit mental fatigue [58], suggesting that there might be psychological ergogenic effect of caffeine mouth rinses—and, therefore, potentially caffeine's bitter taste—for future research to uncover.

Sweet and bitter tastes section summary

Based on the research discussed here, there is a clear ergogenic effect of carbohydrate mouth rinsing on endurance performance [14], along with a potentially ergogenic effect of oral exposure to both bitter tastants [19] and caffeine [15] although in the latter two cases, subsequent ingestion of the mouth rinse is likely required to enhance performance. Regarding bitter tastants, it is believed that this subsequent ingestion is required to further stimulate bitter taste receptors in the upper gastrointestinal tract [44]. These bitter taste receptors are not necessarily linked to gustatory neurons [59], meaning that this activation is not associated with "tasting" the bitterness. Additionally, tasteless carbohydrates evoke an identical ergogenic effect as sweet carbohydrates in a mouth rinse [36], whilst sweet-tasting artificial sweeteners do not [33]. As such, it is important to note that the sensation of a particular taste may not be driving these ergogenic effects, but instead it is likely the stimulation of other receptors, which in turn act centrally to enhance performance [14].

Thermal tastants and athletic performance

Chilli and capsaicin

For millennia, humans have included spices, such as chili peppers in their diets, experiencing and often enduring the associated pungent sensation of oral heat [60, 61]. Mechanistically, the sensation of increased temperature derives from the interaction between the compound capsaicin (8-methyl-*N*-vanillyl-6-nonenamide) and transient receptor potential vanilloid-1 proteins (TRPV1) [62]. TRPV1 is also stimulated when temperatures are elevated [63]; hence, foods containing capsaicin are perceived as being hot [62]. This perceptual heat is not limited to taste, with capsaicin also used in topical ointments, patches and sprays as a temporary but targeted analgesic [61]. The application of which is widely used by recreational and elite athletes to reduce joint and muscle pain, whereas the possible ergogenic properties of capsaicin taste and ingestion are an emerging field.

To date, only four studies have investigated the ergogenic properties of capsaicin ingestion [64–66] or mouth swilling [12] in humans, and as such an array of protocols, dosages and performance measures have been assessed. Three studies have investigated the effect of acute supplementation of capsaicin (12 mg) 45 min prior to athletic performance in a 1500-m running time trial [65], four sets of 70% 1RM repeated squats to failure [13], and time to exhaustion during repeated 15-s treadmill running at 120% VO_{2Peak} with 15-s rest intervals [66]. Capsaicin supplementation improved 1500-m time-trial performance (CAP 371.6 ± 40.8 s vs. Pla 376.7 ± 39 s), total mass lifted (CAP 3,919.4 ± 1,227.4 kg vs. Pla $3,179.6 \pm 942.4$ kg) and time to exhaustion (CAP 1530 ± 515 s vs. Pla 1342 ± 446 s) compared to placebo. RPE was also significantly lower, although no differences in blood lactate were shown [13, 65]. Researchers suggested that capsaicin supplementation may have stimulated activation of TRPV1 in skeletal muscle increasing calcium release at the sarcoplasmic reticulum, a phenomenon seen in rodent studies [67]. This increased influx of calcium may have resulted in greater actin and myosin interactions leading to improved performance. Alternatively, capsaicin has been shown to have an analgesic effect [61], which may have lowered RPE values and facilitated performance [13]. Increased endurance capabilities may also be facilitated by spared glycogen and concomitant increase in lipolysis through capsaicin ingestion [68-70].

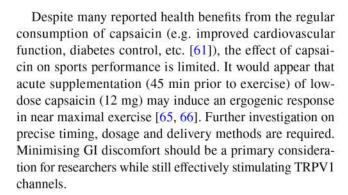
The above literature suggests that ingesting capsaicin as a capsule is effective for improving sport performance. However, when capsaicin is ingested as food, the ergogenic effects are not consistent. A 7-day ingestion of cayenne herbal supplement totalling 25.8 mg day⁻¹ of capsaicin did not result in improved 30-m sprint times nor a reduction in



RPE or muscle soreness scores [64]. Whereas, Lim et al. [71], showed the ingestion of 10 g of hot red peppers 2.5 h prior to exercise (150 w cycling for 60 min) significantly elevated both respiratory quotient and blood lactate levels at rest and during exercise, suggesting increased carbohydrate oxidation. The differences in supplementation type (cayenne vs. red peppers), dose amount (25.8 vs. 12 mg) and protocol (repeated vs. acute) likely contributed to the variation in efficacy; the higher dose in particular may negatively influence GI motility [13]. This is supported by a rodent study that found swimming endurance was optimal when mice were supplemented with 10 mg/kg, 2 h prior to performance [72]. This dose and ingestion timing appear to be a 'sweet spot', with doses or timings that fall below or exceed these values proving ineffective or deleterious to performance, respectively [73]. It should be noted that a similar dosage in a human diet would equate to 100 g of red chilli pepper consumption [74], which would be impractical and likely cause serious gastrointestinal (GI) discomfort [69].

As TRPV1 receptors are found in the oesophagus, stomach, intestine and colon [75], the possibility of GI discomfort is increased following capsaicin consumption. In a study where participants ingested capsaicin capsules, moderate visceral pain was reported following a median dose of 1 mg [76]. Opheim and Rankin's [64] repeated sprint study reported GI distress symptoms increased 6.3 times compared to placebo and resulted in three participants withdrawing from the study [64]; thus, capsaicin-induced GI discomfort may deleteriously affect performance. A possible solution may be the use of a unique variety of chili peppers, CH-19 Sweet, which contain capsiate, a non-pungent capsaicin analogue that has been shown to activate TRPV1 [69, 77] and return similar responses as capsaicin, including improving time to exhaustion in rodent studies [69, 74]. Haramizu et al. [69] also observed no aversion to capsiate ingestion; like carbohydrate, efficacy of capsaicin supplementation may be less about the taste of the intervention and more about the activation of desired receptors.

In each of the aforementioned human studies [64–66], capsaicin was delivered via a capsule. As a result, receptors in the oral cavity were by-passed, eliminating capsaicin's pungent oral sensation. Recently, Gibson et al. [12], employed a 0.2% capsaicin mouth swill every 10 min during repeated 6-s cycle ergometer sprints in the heat (40 °C, 40% relative humidity). This delivery method (mouth swill) directly targets TRPV1 channels in the mouth and reduces possible GI discomfort; yet, results showed no difference in peak power, work performed or RPE across experimental groups (control, placebo, menthol and capsaicin mouth swills). Interestingly, thermal perception (comfort and sensation) was not altered after capsaicin mouth swill compared to control and placebo, but menthol trials reported significant improvements in thermal comfort [12].



Menthol

Menthol imparts its familiar minty flavour via stimulation of transient receptor melastatin 8 (TRP-M8) receptors. These sodium voltage-gated ion channels are especially concentrated in the trigeminal nerve, which innervates the oral cavity, and when stimulated mimics a 'cold' temperature range (8–28 °C; [78]), feeling and tasting 'cool'. The effects of menthol are inversely proportional to the thickness of the stratum corneum [11, 79]; hence, application to the oral cavity often confers a greater stimulatory effect than topical menthol application [11, 80]. Menthol can be experienced by anosmic individuals [81], emphasising its neurological mechanism [82, 83], but the ability to detect menthol has been shown to decline with age [84], suggesting higher menthol concentrations may be required to elicit ergogenic effects in masters athletes.

Menthol application to the oral cavity can be individualised using a preferred menthol concentration and may be enhanced using colour [29]. A relative dose is yet to be administered to athletes, but an experimental dose of 30 mg/kg was prescribed by food scientists investigating the effects of carbonation and menthol upon oral cooling [85]. Partnering menthol's chemosensory cooling effects with physiological coolants, such as ice slurries may further enhance its efficacy [86–88], but there is an increased risk of overstimulation of the trigeminal system potentially resulting in "brain freeze" [89–91].

Performance literature, to date, has assessed the effects of menthol mouth swilling upon cycling in intermittent [12] and time to exhaustion [25, 26, 92] models, as well as running time-trial performance [27, 93]. Intermittent performance was not improved; however, time to exhaustion and time-trial performance demonstrate trivial-moderate improvements (Hedge's g: 0.40; 0.04 – 0.76 [18]). Concomitant improvements in thermal comfort and thermal sensation are noted following menthol exposure [12, 25, 27, 92, 93], with an increase in ventilation also reported [25–27]. These effects are likely mediated by TRP-M8 expression and stimulation of jugular and nodose neurons which provide interoceptive feedback from the alimentary organs and



the cardiorespiratory system [94, 95]. This may explain the increase in ventilation seen with menthol mouth swilling. The rate and volume of airflow passing through the nasal canal also increase TRP-M8 activity and ventilation [96–98]. Whilst this can be contrived in the laboratory, it is likely that this effect is more apparent in ecologically valid settings with faster wind and performance velocities.

Despite participants reporting feeling cooler, no changes in body temperature have been reported to date following the oral application of menthol exclusively [12, 25–27, 92, 93]. An emerging secondary effect of menthol use is an attenuation of thirst [23]; however, the potential ergogenic and contextual relevance of this are unknown as of yet, highlighting that menthol should be applied to sport cautiously. Thirst, more so than taste, conveys a homeostatic message regarding hydration status [99, 100]; however, thirst can also be quenched by carbonated and cool/cold products [85, 100–103], emphasising the role of TRP-M8 receptors in our somatosensory interpretation of cool and refreshing [24, 104–106] and the potential for deception-driven dehydration if water intake is attenuated in an event where hydration status is performance limiting e.g. ultramarathon [107, 108], or in athletes with abnormally high sweat rates [109].

Thermal tastants section summary

Whilst the research pertaining to the TRP channel afferents capsaicin and menthol is in its infancy, in comparison to caffeine and carbohydrate, these thermal tastes may prove ergogenic under certain circumstances and likely serve to disrupt an athlete's perception of their thermal state, which may be ergogenic of itself. Individual sensory thresholds for effective doses likely exist, and timing of administration requires further elucidation, with the potential impact of these strategies on GI discomfort an important consideration. What is clear though is that if capsaicin and menthol are to be supplemented, attaining meaningful doses via wholefoods would either be impractical or ineffective [73, 110]

The sweet taste of placebo

The ergogenic effect of taste could be influenced by the placebo effect. The placebo effect is a desirable outcome resulting from a person's expected and/or learned response to a treatment or situation [28]. Placebo effects have shown to improve sport performance [111–113], with a systematic review reporting small to moderate effects for nutritional (d=0.35) and mechanical (d=0.47) ergogenic aids [114]. Placebo effects are often created within a psychosocial context that influences a person's response to a placebo. These include the interaction between the person receiving the placebo and the person administering it (e.g. participant and researcher), the environment in which it is delivered (e.g.

laboratory) and sensory processes, such as colour, smell and taste [28]. The placebo effect is, therefore, a response to a signal, or set of signals, which conveys information that trigger self-regulatory mechanisms.

While there are many theories to propose the underpinning mechanisms of the placebo effect (e.g. expectancy theory, classical conditioning), in this paper, we adopt a broader and general conception that the placebo effect of taste could be explained through an anticipation on resource allocation. Beedie et al. [115], recently argued that the brain modulates and anticipates the relationship between a signal (e.g. taste) and the body, which regulates subsequent resource allocation. Based on this understanding, the taste of glucose, for example, signals to the brain that resources will soon be available, which in turn, regulates the resources allocated. Theoretically, if a placebo tastes like glucose, the brain would anticipate that glucose has been received and subsequently offloads more resources. In short, the placebo effect may impact the ergogenic effect of taste, through its application of signalling to the brain that more resources are available, which sets in motion a chain of self-regulatory responses that produce an improvement in performance.

Research into taste and the placebo effect on sport performance are limited. However, early research into the placebo effect provides compelling evidence of the significant role the taste can have for inducing placebo effects and influencing physiological responses. Ader and Cohen [118] administered a distinctly flavoured drink followed by a toxic agent capable of suppressing the immune system. After repeated administrations of the drink and toxic agent, the taste of the drink alone resulted in an immunosuppression response. Similarly, Olness and Ader [119] reported a clinical case study of a child with lupus erythematosus (an autoimmune disease) after administering cyclophosphamide paired with taste and smell stimuli similar to Ader and Cohen [118]. After initial pairings of the drug with the sensory stimuli, the taste alone was administered and the patient's symptoms improved after 12 months. The publication of these studies resulted in a proliferation of similar taste aversion research [120], which has demonstrated the influence of taste and anticipatory responses in inducing placebo effects.

It is likely that placebo effects of taste are mediated by neurobiological pathways. While there are many neurobiological pathways associated with the placebo effect, a large amount of research has investigated the role of the endogenous opioid system [121]. This is not surprising given that μ -opioid receptors located throughout the brain are critical

¹ Providing an explanation for why this occurs is outside the scope of the paper, but we refer the reader to the work of Humphrey [117] and Miller, Colloca and Kaptchuk [118], who offer a more thorough explanation.

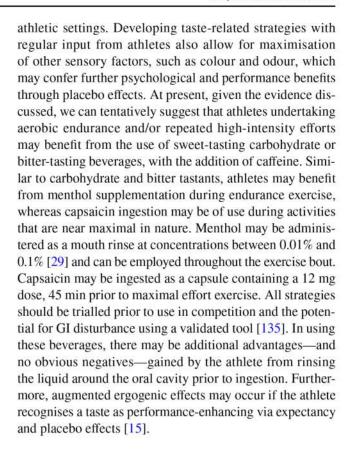


for the reduction of pain [122]. Amanzio and Benedetti [123] exposed participants to a conditioning procedure of the opioid drug buprenorphine and measured pain tolerance and endogenous opioid release in the brain. After repeated trials of the opioid drug, when replaced with saline, pain tolerance significantly increased compared to baseline, which was mediated by increase in activation of the endogenous opioid system. Similar results have been reported elsewhere [124, 125] and highlight the significant mediating role the endogenous opioid system has for inducing placebo effects.

Like placebo effects, taste receptors can also mediate the release of endogenous opioids [126, 127]. Although the magnitude of the effect can depend on age and gender [128], the sweet taste of glucose and sucrose can modulate the production of endogenous opioid release [129], whereas administration of sucrose directly to the stomach has no effect [130]. This suggests that sweet taste can have analgesic effects. However, where the ergogenic effects of taste tend to report pain-relieving effects, placebo effects are often the result of similar mechanisms, e.g. pain, fatigue and perception of effort [112, 113, 131]. While taste could have direct neurobiological mechanisms, there is evidence that placebo effects can mimic the neurobiological pathways of a treatment [132]. It could be suggested that the same pathways activated by taste are also activated by the administration of a placebo. We are by no means implying that the ergogenic effects of taste are the result of a placebo effect, but we, like others [28, 133, 134], are suggesting that the mechanisms in which a nutritional ergogenic aid exerts its effect is likely to be a combination of both. As with most treatments and interventions on sport performance, the ergogenic effect of taste will be influenced via the placebo effect (see Beedie et al. [133]). It is likely that they are both components of a self-regulatory system that act as signals to the brain for resource allocation, which are likely to be mediated by neurobiological pathways, such as the endogenous opioid system. However, there is a lack of research in sport explicitly examining whether the ergogenic effect of taste and the placebo effect activate shared or distinct mechanisms. To help develop knowledge and understanding in this area beyond speculation, empirical research is needed that examines whether the placebo effect of taste is partially or fully responsible for its ergogenic effect.

Practical recommendations

Tastants have the potential to be employed as ergogenic strategies during sport and exercise performance, with tentative evidence supporting the efficacy of sweet [14], bitter [19], spicy [65], and cooling [11] tastants. However, consideration of event demands, nutritional state of the athlete and athletes' performance environment are strongly recommended to successfully employ taste-related strategies in



Future research directions

Future research in taste and athletic performance should consider investigating differences between tasting, swilling and ingesting, and their subsequent effects upon performance; this is especially important given the emerging research that ingestion of bitter tastants, such as quinine and caffeine, is required to maximise their ergogenic effects above those demonstrated through mouth rinse only [15]. Each strategy exposes tastants to different densities and volumes of taste receptors and may be accompanied by other sports nutrition strategies, so the inclusion of tastants need to be weighed against established ergogenic strategies, such as maintaining carbohydrate availability during an event. The optimal dose of each tastant, including their physiological tolerance and associated side effects, also represents an important practical avenue for future research. Similarly, habituation to tastants is also worthy of investigation as we must understand the time course of these strategies to maximise their efficacy. It is acknowledged that there is likely a strong genetic underpinning to preference and responses to tastes [136, 137]. Some work has already begun in caffeine [138, 139], carbohydrate [140, 141] and TRP-M8 [142], but understanding the genetic contributions to liking, or tolerance for, thermal tastes and bitterness may confer further benefits beyond athletic populations.



Conclusion

This review synthesises the evidence from a variety of tastes that have shown ergogenic promise with respect to athletic performance. This efficacy is not limited to taste per se, but extends to the stimulation of targeted receptors in the oral cavity and throughout the digestive tract, which relay signals pertaining to energy availability and temperature to appropriate neural centres. Timing of tastant application, dose and frequency of application likely require personalisation to be most effective and can be enhanced or confounded by factors that relate to the placebo effect.

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