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Physiological effects of flavour perception

Karen L. Teff

The flavour of food, a critical determinant of consumer selection and consumption, also has the potential to regulate how ingested food is absorbed and metabolized. The underlying physiological pathways that facilitate the relationships between flavour and nutrient metabolism are neural connections among the oropharyngeal region, the brain and peripheral tissues. Recent studies have shown that the flavour of food can improve nutrient metabolism in human subjects. Future work should identify how individual flavours can improve nutrient metabolism in both normal and clinical populations.

Food flavour is an essential determinant of what an individual chooses to purchase and consume. Thus, improving and enhancing the flavour of food are of primary concern to the food industry. What may be less apparent is that, in addition to contributing to the hedonic value of a food, flavour has the potential to influence human physiological function.

The relationship between flavour and physiology has been recognized since the turn of the century, when

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Pavlov conducted his ground-breaking experiments demonstrating that the presence of food in the oral cavity enhanced protein digestion in dogs[1]. Similar effects have been demonstrated in humans; Wolf and Wolff[2] reported on an individual who, in 1895, required a gastrostomy (creation of an artificial opening in the stomach) after burning his oesophagus. Through trial and error, the individual learned that in order to gain weight and satisfy his appetite, he had to taste and chew the food before its insertion into his stomach. These early studies imply that food flavour improves nutrient metabolism and influences satiety. The aim of this review article is to discuss the mechanisms that enable flavour to influence nutrient metabolism and to review the evidence establishing a role for flavour in human physiology.

Physiological pathways mediating the effects of flavour on nutrient metabolism

The underlying physiological pathways that facilitate the relationships between flavour and nutrient metabolism are neural connections among the oropharyngeal region, the brain and peripheral tissues (Fig. 1). Stimulation of receptors in the mouth, nasal cavity and throat by food-related sensory stimuli activates neural fibres leading to the central nervous system, where specific brain areas are used as relay stations, integrating sensory information and initiating appropriate responses. The nucleus of the tractus solitarius is the primary site that receives information concerning taste, and when activated it sends a message to the dorsal motor nucleus of the vagus, where the efferent fibres of the vagus nerve originate[3]. The vagus nerve, part of the parasympathetic nervous system, branches extensively and innervates many of the tissues involved in nutrient metabolism including the stomach, intestine, pancreas and liver. Vagal activation releases biologically active substances from the innervated tissues. Saliva[4], gastric acid[5], enzymes from the exocrine pancreas[6] and hormones

from the endocrine pancreas[7-9] are secreted in response to the perception of food flavour. These physiological responses that occur as a consequence of sensory stimulation are termed the cephalic phase reflexes. Vagal activation can also modulate other processes that are associated with food ingestion, such as hepatic enzyme activity[10] and diet-induced thermogenesis (heat production)[11]. Thus, the flavour of food activates the parasympathetic nervous system, which in turn elicits the cephalic phase reflexes and modulates other physiological functions such as thermogenesis and metabolism.
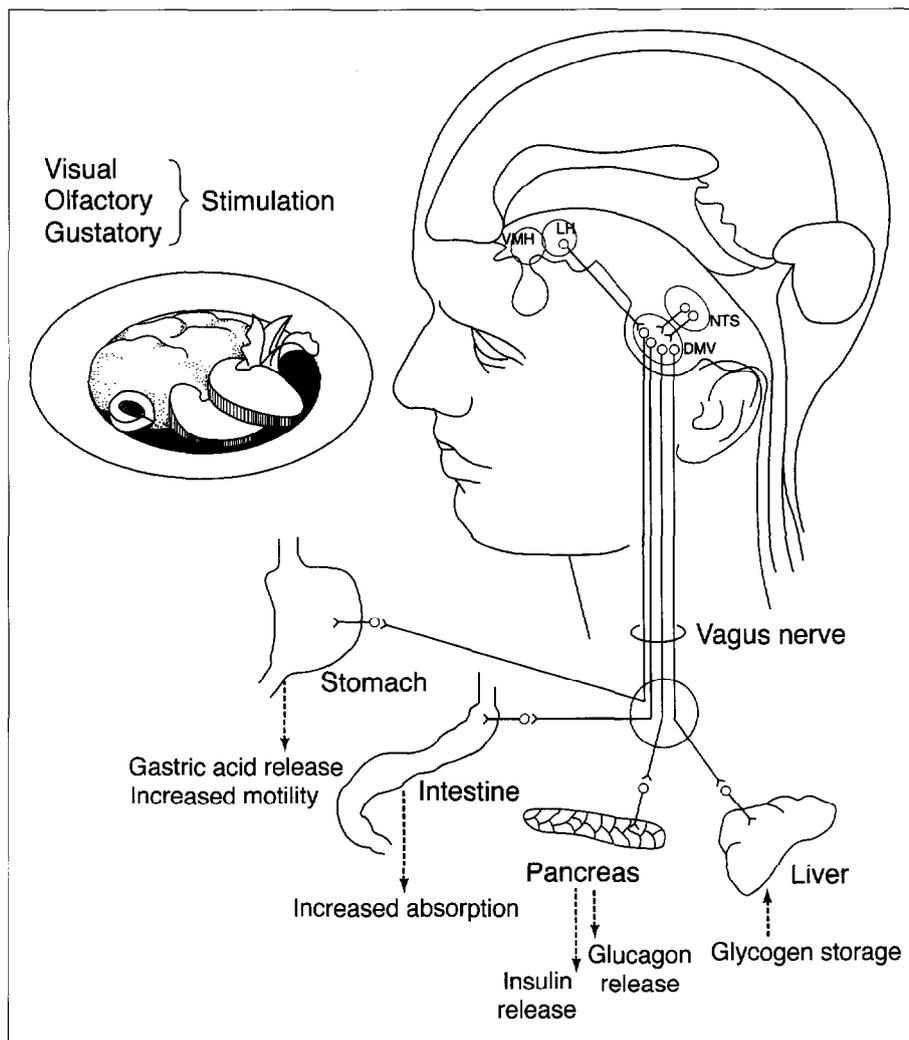
Cephalic phase reflexes: reflexes elicited by the flavour of food

The cephalic phase reflexes can be defined as 'autonomic or endocrine reflexes triggered by sensory contact with foodstuffs rather than by postingestional consequences'[3] (see Glossary). Because the reflexes are neurally mediated, they occur rapidly and can be distinguished from postprandial responses by their temporal patterning. Pre-absorptive release is one of the key characteristics of the cephalic phase reflexes. A good example of this is cephalic phase insulin release, which starts to increase within 2 min following sensory exposure, peaks at 4 min and then returns to a baseline level 8–10 min post-stimulus[8,9]. In contrast, postprandial insulin release is dependent on an increase in the level of glucose (and/or amino acids) in the plasma, does not begin until at least 15 min following food ingestion and peaks after 30–45 min depending on the type of food ingested. To verify that food flavour can elicit the cephalic phase reflexes independently of food ingestion, human subjects have been asked either to smell and look at food or to perform a modified sham-feed in which they taste, chew and then expectorate the food stimuli. Using this approach, the release of saliva[4], gastric acid[12], amylase, gastrin, cholecystokinin[13,14], insulin[9], glucagon[15] and pancreatic polypeptide[14] can all be elicited by the flavour of food. Administration of atropine, a muscarinic antagonist, which blocks the effects of the vagus nerve on the peripheral tissues, inhibits the release of the compounds listed above, thereby confirming neural mediation of the effect[5,16,17].

An additional characteristic of the cephalic phase reflexes is that they are often of smaller magnitude than their postprandial counterparts, such that neural activation by the perception of food flavour results in a mimicking of what occurs during the postprandial phase but on a

Fig. 1

Relationships between the flavour of food and nutrient metabolism. Food-related sensory stimuli activate receptors in the head and neck region, which send a message to the brain. Within the brain, the nucleus of the tractus solitarius (NTS) is the primary site that receives information about taste. Fibres from the NTS relay information to the dorsal motor nucleus of the vagus (DMV), which in turn activates the vagus nerve. Vagal activation elicits the release of gastric acid from the stomach, and of hormones and enzymes from the pancreas, as well as influencing gastric motility and the intestinal absorption of nutrients. (VMH, ventromedial hypothalamus; LH, lateral hypothalamus.)

smaller scale. The magnitude of the cephalic phase response relative to the postprandial response varies depending on the compound being considered. For example, cephalic phase gastric acid accounts for ~33% of the total amount of the gastric acid released during a meal[18], whereas cephalic phase insulin release is equivalent to only 3% of postprandial insulin release[19] (Fig. 2).

The cephalic phase reflexes are generally thought to be preparatory responses that optimize the digestion and absorption of food. However, various hypotheses have been put forward as to the mechanisms mediating these effects and the extent of the involvement of the cephalic phase reflexes in food ingestion. It has been postulated that the cephalic phase reflexes are conditioned responses that provide information on the quantity and quality of the food being ingested, thereby allowing the body to make adaptive changes. Such adaptive changes could be either metabolic (i.e. enhancement of a postprandial response) or behavioural (e.g. initiating or inhibiting food

intake)[20,21]. Some investigators have proposed that the cephalic phase reflexes are directly involved in the regulation of food intake and that eliciting the cephalic phase reflexes by perceiving the flavour of food will increase food consumption. Data supporting this argument originate from animal studies that demonstrated that obese rats have cephalic phase reflexes of greater magnitude than those of normal-weight rats[3], and that rats with larger cephalic phase insulin responses tend to gain more weight than do animals with smaller responses[22]. However, in humans no studies have directly addressed the role of the cephalic phase reflexes in food intake regulation; rather, most experiments have manipulated the palatability of the food and then monitored hunger ratings or food intake without having taken any direct measurements of the cephalic phase reflexes[23,24]. The strongest evidence for the involvement of the cephalic phase reflexes and, hence, flavour in physiological function is in the area of nutrient metabolism.

Effects of flavour on nutrient metabolism

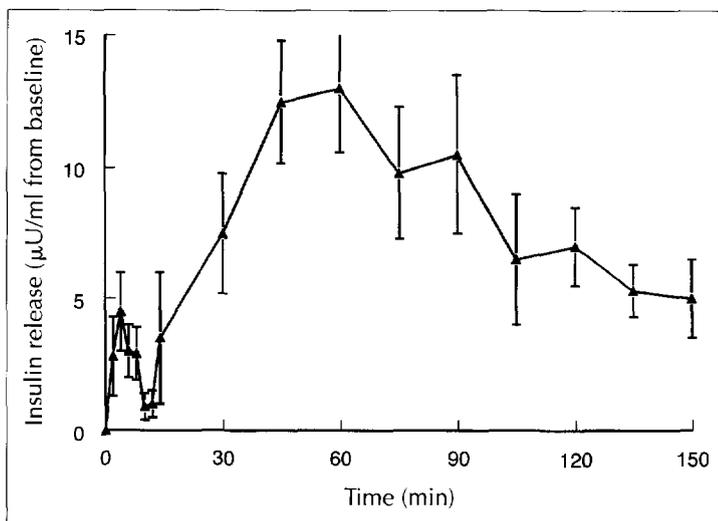The effects of flavour on nutrient metabolism are mediated by activation of the parasympathetic nervous system and elicitation of the cephalic phase reflexes. Because flavour acts on receptors in the head and mouth region, bypassing these receptors by the direct administration of nutrients into the stomach leads to a lack of activation of the parasympathetic nervous system and, hence, no stimulation of the cephalic phase reflexes. The importance of flavour in nutrient metabolism is illustrated by a recent experiment that demonstrated that the mere taste of food improved glucose tolerance in normal-weight subjects[25]. In this experiment, glucose was administered intragastrically through a nasogastric tube on two separate occasions. On one day, the subjects tasted and expectorated a sandwich for 5 min during the intragastric infusion of glucose, whereas on the other day the subjects remained quiet for the same time period. The 5-min exposure to sensory stimuli resulted in a 33% decrease in postprandial glucose levels owing to a significant increase in postprandial insulin release (Fig. 3). Indices of glucose metabolism such as glucose disappearance rate (how fast glucose levels return to normal) and plasma glucagon levels were also influenced by oral sensory stimulation. Studies in which the glucose challenge to elicit insulin release is infused through a vein (intravenous glucose tolerance test) while subjects taste or smell food support these findings. Bryde Andersen et al.[26] reported that the sight and smell of an appetizing meal paired with an intravenous glucose tolerance test resulted in an increased rate of glucose disappearance, whereas Lorentzen et al.[27] reported a decrease in postprandial glucose levels following a modified sham-feed of an appetizing food.

In a study examining the effect of oral sensory stimulation by fat on fat metabolism[28], subjects swallowed 50 g of fat in capsules and then performed a modified sham-feed with crackers alone, crackers plus a non-fat cream cheese or crackers plus a full-fat cream cheese. Only the full-fat cream cheese resulted in significantly higher postprandial triacylglycerol levels than were found when no oral stimulus was provided. The mechanism mediating this effect is not known, although Mattes postulated that some chemical aspect of the dietary fat elicits changes in postprandial lipid metabolism. These studies, the majority of which have been conducted within the past few years, clearly demonstrate that the perception of food flavour activates mechanisms involved in nutrient metabolism. To date, no studies have revealed whether individual flavours can have differential effects on nutrient metabolism. However, several experiments have examined the effects of specific flavours on various cephalic phase reflexes. If a flavour can activate a cephalic phase reflex, then this by definition implies activation of the parasympathetic nervous system, and the potential to influence physiological function.

Fig. 2

Bi-phasic release of insulin illustrating the time (0–10 min post-ingestion) and magnitude of cephalic phase insulin release relative to postprandial insulin release following the ingestion of a sandwich containing 255 kcal (adapted from Ref. 9).

Efficacy of specific flavours in eliciting the cephalic phase reflexes

The majority of studies examining the effects of specific flavours on the cephalic phase reflexes have examined responses in animals. In these studies, individual

taste qualities (i.e. sweet, sour, salty, bitter and umami) have been tested for their effectiveness in eliciting one of the cephalic phase reflexes, with the primary areas of interest being pancreatic exocrine secretions in dogs and cephalic phase insulin secretion in rats. Pancreatic exocrine secretions, monitored by measuring pancreatic volume flow and protein output, have been shown to increase with increasing concentrations of sucrose solutions[6]. Monosodium glutamate is also an effective stimulus, whereas citric acid (sour) and sodium chloride (salty) are not[6,29]. Similar stimuli can elicit cephalic phase insulin release. Sweet solutions composed of either sucrose[30], glucose[31] or saccharin[22] as well as monosodium glutamate[32] have been shown to stimulate cephalic phase insulin release in rats. However, neither quinine (bitter)[33] nor sodium chloride[32,33] elicited the response, suggesting that flavours associated with caloric content, such as sweet (carbohydrate) or umami (protein), may be the most potent stimuli for influencing physiological function.

In humans, the focus has primarily been on larger, more general issues such as the role of palatability in eliciting the cephalic phase reflexes and the magnitude of the responses in clinical populations. Since the early work of Pavlov[1], studies have shown that the palatability of a food stimulus can influence the magnitude of the cephalic phase reflexes. Foods with higher hedonic ratings elicit greater salivary[4,34] and gastric acid release[35] than do foods that are rated as unpalatable. The effect of palatability on cephalic phase insulin release is more controversial; although some studies have demonstrated that food palatability can influence the magnitude of the response[7,36], no such effect was found in another study[37]. Palatable food in the form of a complete meal has also been shown to increase the resting metabolic rate, an indicator of thermogenesis, compared with the same meal rendered unpalatable by blending[11]. In general, these data suggest that there is an increased rate of nutrient metabolism when individuals are exposed to food that they perceive as having a high hedonic value.

Sweet solutions, which are assumed to be highly palatable, have been tested for their efficacy in eliciting cephalic phase insulin release in humans. As discussed above, both nutritive and non-nutritive sweet solutions elicit cephalic phase insulin release in rats. However, in humans, sweet solutions do not appear to be particularly effective stimuli for eliciting the response; in several studies, cephalic phase insulin release was not observed after subjects tasted solutions sweetened with aspartame[38,39], sucrose (nutritive) or saccharin (non-nutritive)[39]. Although two studies did demonstrate significant increases in pre-absorptive insulin release following either ingesting sweetened solutions[40] or tasting a glucose solution[16], the response to this type of stimulus appears to be less robust than to a whole food. One possibility as to why a stimulus that is known to be effective in animals tends to be less effective in human subjects is that psychological factors influence a human's physiological response to the perception of food flavour. For example, restrained eaters (individuals who consciously limit the
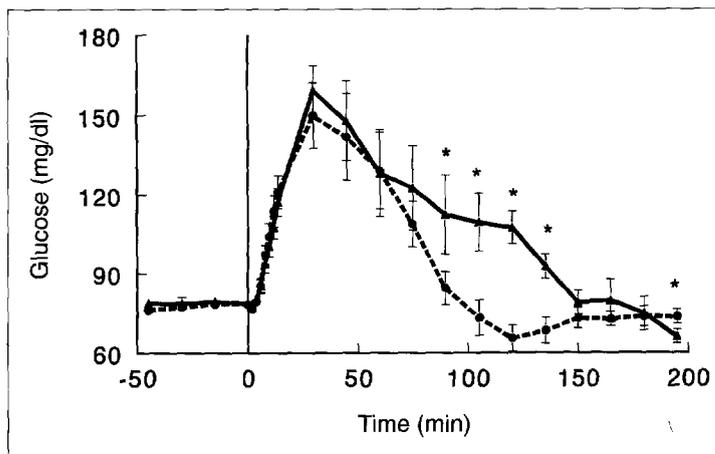
Fig. 3

Effect of a modified sham-feed on plasma glucose levels following intragastric administration of 75 g glucose compared with intragastric glucose alone. Sham-feeding for a 5-min period significantly reduced postprandial glucose levels by ~33% (3433 ± 783 mg/dl/195 min for the sham-feed; 5643 ± 1397 mg/dl/195 min for the control). The vertical line at 0 min represents the onset of the modified sham-feed. The solid line (—▲—) represents plasma glucose levels following intragastric glucose administration alone; the hatched line (--●--) represents intragastric glucose administration paired with a sham-feed. The asterisk (★) indicates differences between curves significant at the $p < 0.05$ level. Reproduced with permission from Ref. 25.

amount that they eat), have greater cephalic phase salivary[41] and insulin responses[37] than non-restrained eaters, suggesting that cognitive attitudes towards food play a role in regulating physiological responses. It is possible that, for humans, liquids are not perceived as 'real foods', or perhaps liquids do not provide adequate sensory stimulation. For example, the magnitude of cephalic phase gastric acid release increases with an increasing combination of sensory components, such that visual stimulation plus olfactory stimulation is more effective than visual stimulation alone, whereas visual stimulation plus olfactory stimulation plus gustatory stimulation results in the response of greatest magnitude[12].

Conclusions and future trends

The role of food flavour in nutrient metabolism was first recognized almost 100 years ago. However, this area of research is still in its infancy. Recent evidence indicates that the flavour of an intact food can influence how food is digested, absorbed and metabolized; however, the efficacy of individual flavours as stimuli has not been examined. In addition, little is known about the relative importance of the individual cephalic phase reflexes and their respective roles in nutrient metabolism. Does a single cephalic phase reflex have the ability to influence physiological function, independent of the other reflexes and activation of the parasympathetic nervous system? In an attempt to address this question, a recent study found that the replacement of cephalic phase insulin release by exogenous insulin infusion in obese subjects improved glucose tolerance even when the activity of the parasympathetic nervous system was inhibited. These data suggest that the presence of insulin during the pre-absorptive period (i.e. cephalic phase

insulin release) is important in glucoregulation, independent of activation of the parasympathetic nervous system[17].

In humans, the issues concerning flavour and nutrient metabolism are particularly complex because of the influences of psychological and social factors on an individual's response to food. An individual's past experience with a particular food, hedonic rating of that food, and general attitude towards eating can all influence the physiological responses to the food stimulus. Furthermore, post-ingestional consequences of food ingestion (e.g. physiological sensations of satiation or fullness), which are at least partially modified by the flavour of food, can be either positively or negatively reinforcing, thereby influencing subsequent physiological and psychological responses.

A final consideration is the potential implication for individuals who may have impaired physiological responses to flavour. Some studies suggest that obese individuals[19,42] and individuals with eating disorders[43,44] have altered or impaired cephalic phase reflexes. The exciting question then raised is whether the impairment of the cephalic phase reflexes is contributing to the disordered metabolism and whether the reflexes can be restored through the manipulation of food flavour.

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