Peer-reviewed | Manuscript received: June 20, 2013 | Revision accepted: July 05, 2013

Taste and Nutrition

3. Development of Taste Preferences and Aversions

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Summary

All our senses help to evaluate the quality of potential food. By sight, we identify spoiled food. The sense of smell distinguishes the aroma of palatable from the malodor of inedible food. The trigeminal system reports about texture, temperature and irritants. Touch and audition determine the ripeness of fruits when we press or knock on them. The sense of taste, however, is particularly critical because it controls innate reflexive appetitive and repulsive behaviors. The brain constructs the flavor of food from the various percepts which, in the context of postprandial experiences, lead to learned preference or conditioned aversion for that food. The flavor of food and the postprandial experiences are stored in the implicit taste recognition memory which enables fast and reliable recognition of food during future encounters. Repeated ingestion reinforces preferences or aversions forming stable dietary patterns that make it difficult for subjects to switch diets.

Keywords: taste, sensory science, taste preferences, food aversions

Citation:
Hübner S, Töle J, Meyerhof W (2013) Taste and nutrition. 3. Development of taste preferences and aversions. Ernaehrungs Umschau international 60(12): 222–227

This article is available online: DOI 10.4455/eu.2013.042

Whereas the previous contributions[1] dealt with the physiological basis of gustation and the influence of environmental factors and genetic disposition on taste perception, in the present article we will discuss the principles that cause taste preferences and aversions and thus impact strongly on our health.

[1] Part 1: Ernährungs Umschau 07/2013, p. 124–131; part 2: Ernährungs Umschau 10/2013, p. 180–186

Innate taste preferences and aversions

In mammals, oral taste buds develop already before birth [1]. This is important because the sense of taste is required for guiding food ingestion directly after birth. Indeed, newborn babies respond to orally administered taste solutions with facial expressions referred to as **gustofacial** reflexes. If presented a sweet stimulus, they show relaxed smiling and enhanced suckling. If, on the opposite, they experience sourness or bitterness, babies pull characteristic faces, do not suck but demonstrate repulsive behaviors. Thus, the taste-evoked facial expressions unequivocally indicate which tastes babies like or dislike [2, 3].

Other mammals including monkeys, rats and mice display similar appetitive and repulsive behaviors. Usually sweets will be eagerly accepted and consumed since sweetness indicates high energy content of food. Presumably, sweet preference, which is especially well expressed in mammals, facilitates the ingestion of the sweet-tasting, sugar-containing mother's milk. On the other hand, bitter tasting food will be rejected preventing effectively the uptake of potential toxins into the susceptible young organism.

The terminus technicus "gustofacial *reflex*" indicates that the described

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sweet preference and bitter aversion are examples for innate unconscious behaviors. This is supported by the behavior of babies with severe forebrain damages or even lacking a functional cerebrum which, like healthy babies, demonstrate the same gustofacial reactions [4]. Moreover, the typical reflexive behaviors are also seen in experimentally decerebrated laboratory animals [5]. In fact, evolutionary very old structures of the brain stem are responsible for the expression of reflexive taste preferences and aversions. Neural circuits in this area which control musculature of jaws, pharynx and tongue integrate gustatory information to evoke suckling and swallowing or spitting and vomiting (◆ Figure 1).

Therefore, it is little astonishing that conscious recognition of taste modalities is unnecessary for the expression of innate preferences and aversions as demonstrated by a recent case report of an adult patient [6]. The subject suffers from extensive forebrain damages, has a restricted memory of only ~45 seconds and is unable to name the tastes of test solutions presented to him which he equally designates as "pop". However, if offered at the same time a salty and a sweet solution, he vehemently asked for the sweet solution.

The innate preferences and aversions encoded in the brain stem are phylogentically very old and enable primitive vertebrates which lack a well-developed cerebrum a quick and vital control of food intake.

Learned taste preferences

Whereas the innate taste preferences and aversions serve a rough and instantaneous assessment of food, the functions of learned preferences and aversions are much more complex. They evaluate the postprandial consequences of the ingested food (◆ Figure 2) and allow a fast recognition of familiar food [7]. Citizens of industrialized countries may consider the latter unimportant but it is (was) a decisive advantage for the rapid acquisition of food as part of the daily struggle for survival in the animal kingdom or during hominid evolution.

Not alone our five basic taste modalities are involved in learning taste preferences, but also the flavor of food. As defined in the first contribution, flavor refers to the entire mouth feel of food also including smell and trigeminal sensations evoked by texture, temperature and irritants (for example capsaicin, piperin and mustard oil of chili, pepper and horse radish, respectively).

Flavor preference and aversion learning begins during the time of weaning from mother's milk. Children and young animals as well are initially neophobic, i. e., they reject unfamiliar food. As they do not have any experience with solid food, the general neophobia protects them against ingestion of unhealthy stuff (◆ Figure 2). Young rats display neophobic behavior around postnatal day 12, just the time when they begin to supplement mother's milk with other foods [8]. Similarly, neophobia also increases in human babies to reach a maximal level at the age of 2 to 6 years. This time span is characterized by increased mobility and associated with the frequent encounter of novel flavors. During adolescence, neophobia attenuates [9].

Acquired positive experiences with palatable food alleviate neophobia. The strategy of animals is to ingest only small amounts of unfamiliar food. If the food satiates the animal and its ingestion does not lead to malaise, animals consider it safe and nutritious and gradually increase the consumed amount. This rule of preference learning, i. e., pairing of flavor and nutritional value, is demonstrated by a common experimental approach (◆ Figure 3). If animals lick a flavored test solution and simulta-

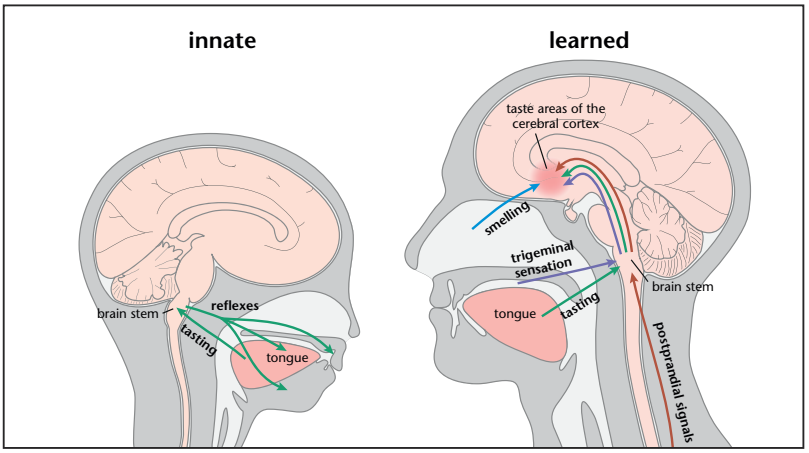

Fig.1: **Innate and learned flavor preferences**
Left: Schematic representation of innate taste preferences. Gustatory information is conveyed from the tongue to the brainstem. Motor neurons in this area evoke reflexes through control over the facial musculature and that of the pharynx.
Right: Learned flavor behavior. In addition to the innate reflexes, learned flavor aversions and preferences are being formed. Gustatory and trigeminal sensations from the mouth, together with smell and postprandial visceral information, are passed on to the flavor processing areas of the cerebral cortex, where they will be evaluated and lead to learned flavor behaviors.

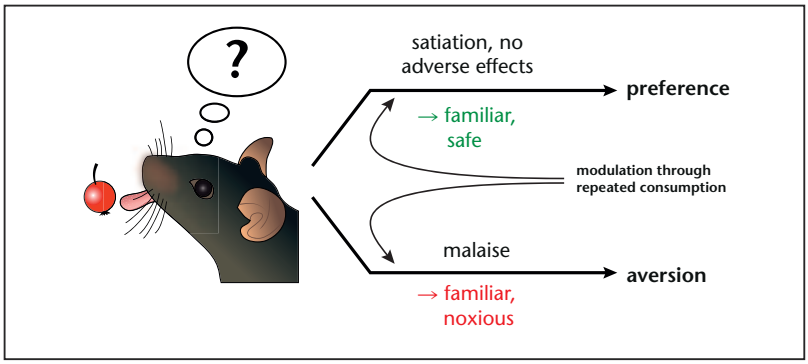

Fig. 2: Formation flavor preferences and aversions
Postprandial consequences determine the behavior of an animal towards an unfamiliar food. If food consumption is followed by satiety and if no malaise is experienced, the animal considers that food as safe and familiar and eats it on future encounters. If on the other hand a food induces malaise, the animal identifies that food as known and dangerous on future encounters and strongly avoids it. Postprandial consequences of future consumptions can enhance or even revert learned preferences and aversions. In this way, animals are able to adapt to changed environmental factors.

neously receive a calorie-rich nutrient solution through a probe into their stomach, they develop a preference for that flavor even if it is bitter and normally repulsive [10].

An experiment by MENNELLA and coworkers demonstrates early establishment of conditioned taste preference in human babies [11]. Infants, adapted to a sour and bitter tasting protein hydrolysate formula fed during their first 7 months of life even ate it after they were fed a cow milk-based formula for two weeks. On the other hand, infants first fed a cow milk-based formula rejected the protein hydrolysate formula under comparable conditions. It has also been shown that babies fed mother's milk accepted the umami taste of glutamate more readily than babies fed cow milk-based formula [12]. This is not surprising considering the fact that the glutamate content of mother's milk is much higher than that of formulas.

Another rule of preference learning involves pairing of familiar with unfamiliar flavors. In this context a subject who likes strawberry yoghurt would tend to prefer a strawberry flavored curd cheese over one flavored with peach. Similar rules apply also to animals. As **coprophagic** animals, rodents simultaneously experience familiar "social" odors and novel odors arising from food consumed by other animals during contact with the feces. By this type of pairing of familiar with novel sensations the animals learn preferences for food that their conspecifics already experienced.

Consumption of a food increases through repeated encounters (◆ Figure 2). Numerous experiences in preference learning therefore also lead to broadened spectra of preferred foods and variable diets.

Flavor of food not only influences what we eat but also how much we eat. If a flavor is paired with concentrated sugar solution administered by gavage, animals will eat less food containing this flavor compared to a pairing of the same flavor with a less concentrated sugar solution. In this phenomenon of conditioned satiation, the organism adjusts the amount of consumed food to the expected supply with calories. Association of flavor with high energy density reduces consumption of food compared with association of flavor with low energy density [10].

Conditioned taste aversions

Unlike the aforementioned formation of taste preferences, aversions are established if malaises occur after the consumption of unfamiliar food (◆ Figure 2). The experience of malaise transforms the initial neophobia into a learned aversion.

The development of conditioned taste aversions have been well examined in rodent models. Yet conditioned taste aversions have also been observed in fish, reptiles, birds, and other mammals including primates [13]. The phenomenon of conditioned taste aversion may serve as an example of classical conditioning. A frequent experimental approach pairs a flavored stimulus with a subsequent intraperitoneal injection of lithium chloride (LiCl) which causes visceral pain. Intriguingly, the animal associates the pain with the experienced flavor and not with the injection. In this conditioning protocol the flavor serves as the conditioned stimulus (CS) whereas the LiCl injection is the unconditioned stimulus (US) [14]. Conditioned taste aversions have some particular properties. Whereas other conditioning approaches such as the well-known PAWLOW experiment require several repetitions to induce robust effects, conditioned taste aversions can be firmly established by a single event [13, 15].

Also the time frame is important for the effect. While instantaneous pain after consumption of food shows little effect, full establishment of conditioned taste aversion requires a time frame of several hours [16]. Moreover, the stronger the malaise is, the longer can be the time between CS and US and the longer the aversion is maintained. A single ex-

posure can induce aversions for lifetime. The strength of the conditioning effect declines, however, with the time interval between CS and US [13].

Still another typical property of classical conditioning is the phenomenon of extinction. If a familiar malaise-inducing food is repeatedly consumed without causing malaise, the aversion attenuates over time (◆ Figure 2) [14].

It is important to note that only negative consequences that relate to the gastrointestinal tract lead to the formation of aversion. Toxins with different targets or induction of externally perceived pain do not cause flavor aversion [14].

Surprisingly, conditioned flavor aversions are also being established if the animal is anaesthetized during the conditioning protocol suggesting that higher cognitive brain functions are not involved. This observation also points to a phylogenetically old age of this phenomenon [13].

Pest exterminators know the phenomenon of conditioned taste aversion as bait timidity in the case of animals which survived ingested poisonous baits [13]. For that reason pest fighting is based on slowly acting toxins which cause their effects after the time period relevant for establishing flavor aversions.

Conditioned flavor aversions cannot only be experimentally induced in laboratory animals but can also be seen in children eating common foods because the same functional principles apply. If consumed food is spoiled or contains toxin, visceral complications are induced which are usually not fatal because of the small amounts ingested. Like pups, children respond to the induced malaise with aversion. Also in this case, the time frame of consumption and experienced malaise as well as the novelty of the food is of relevance for the effect. It is also possible that familiar food becomes repulsive, if subsequent visceral malaise is experienced, induced for instance by gastrointestinal viral infection. The aversive effect generated after ingestion of familiar food is, however, less severe relative to unfamiliar food. This effect of latent inhibition is also a characteristic effect of classical conditioning [17].

Taste recognition memory

The postprandial experiences of a food are stored together with its flavor in the implicit taste recognition memory which is inaccessible to cognition. The formation of the taste recognition memory critically involves the taste areas of the cerebral cortex where the relevant transmission pathways converge (◆ Figure 1) [18]. Other brain areas contribute to the formation of this type of memory. Dopamine producing neurons of the midbrain and their projection areas in the *Nucleus accumbens* are indispensable for the formation of conditioned taste preferences. It is not surprising that this "happiness" hormone and the two brain areas are also involved in reward and addiction. Generation of conditioned flavor aversions involves various brain structures including the amygdala which is important for fear and mental stress as well as the aforementioned *Nucleus accumbens*. Depending on the structures, the neurotransmitters noradrenalin, acetylcholine, and glutamate are important players. In addition, the lateral hypothalamic area which is

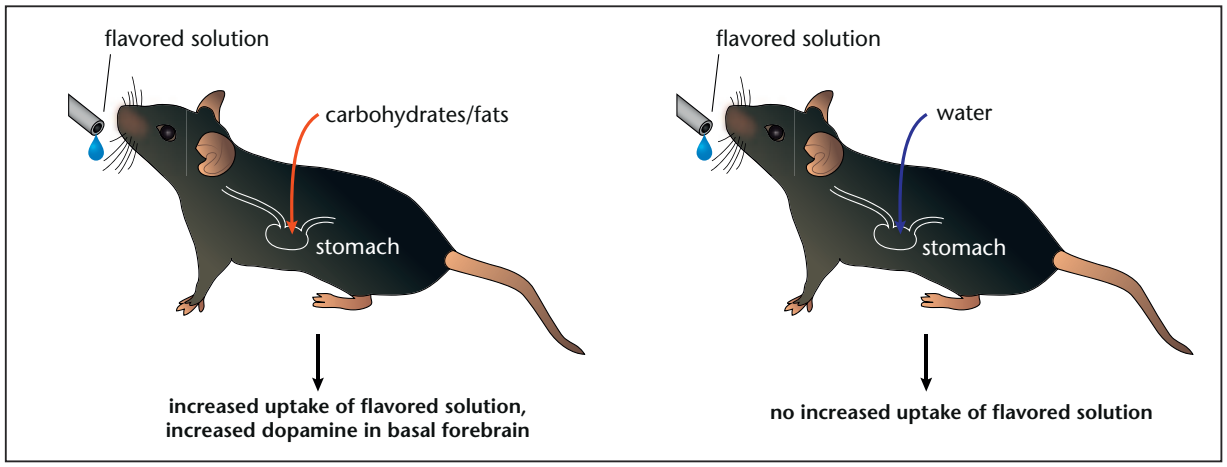

Fig. 3: **Experimental setup to induce conditioned flavor preferences**
A flavored solution is presented to the animal. Every time it is licking the flavored solution, a calorie-rich (left) or a calorie-free (right) test solution is simultaneously administered by gavage directly into the stomach. In the former case the animal develops a preference for the flavor and displays increased release of dopamine in relevant brain areas. In the latter case none of these effects are demonstrated by the animal. Formation of preferences therefore appears to be independent of flavor perception *per se* but rather depends on the availability of associated calories.

crucial for regulating food intake appears to be important for both processes [18].

Postprandial effects on preference learning

We have heard before in detail about the importance of postprandial effects on the formation of preferences and aversions, but we haven't heard about their generation and consequences. However, it is obvious that sensory mechanisms contribute which measure the presence of food in and the chemical content of the alimentary canal (◆ Figure 4) or which register "malaise".

Mechanical sensors located in the gastrointestinal wall record the tension of the stomach and gut induced by the ingested food. Sugar and fat appear to be dominant parameters of the chemical composition of the content of the digestive system. Infusions of sugars and fats into the stomach of mice evoked release of the happiness hormone dopamine in the reward centers of the brain in proportion to the calorie-load (◆ Figure 3) [19, 20]. However, the responsible sensors have not yet been identified. Fatty acids and sugars present in the digestive canal also elicit the release of satiety hormones such as cholecystokinin (CCK), glucagon-like peptide 1 (GLP-1) and peptide tyrosine-tyrosine (PYY) from special cells of the gut mucosa [21]. The information about chemical composition of the luminal content and gut tension can be conveyed to the brain areas which regulate food intake by the vagus nerve or humoral factors [22].

Unlike satiety hormones, ghrelin stimulates food intake. This hormone is released from the gastric mucosa and acts on the brain's reward centers which enhances the desire to eat (◆ Figure 4) [23]. This is illustrated by experiments in rats which received intracranial injections of ghrelin. Relative to control animals, these animals ingested more of a sugar solution and were better motivated in behavioral tests to press a lever for receiving a food reward [24]. Presumably ghrelin plays also a role in the formation of conditioned flavor preferences, yet direct experimental evidence remains to be presented [25].

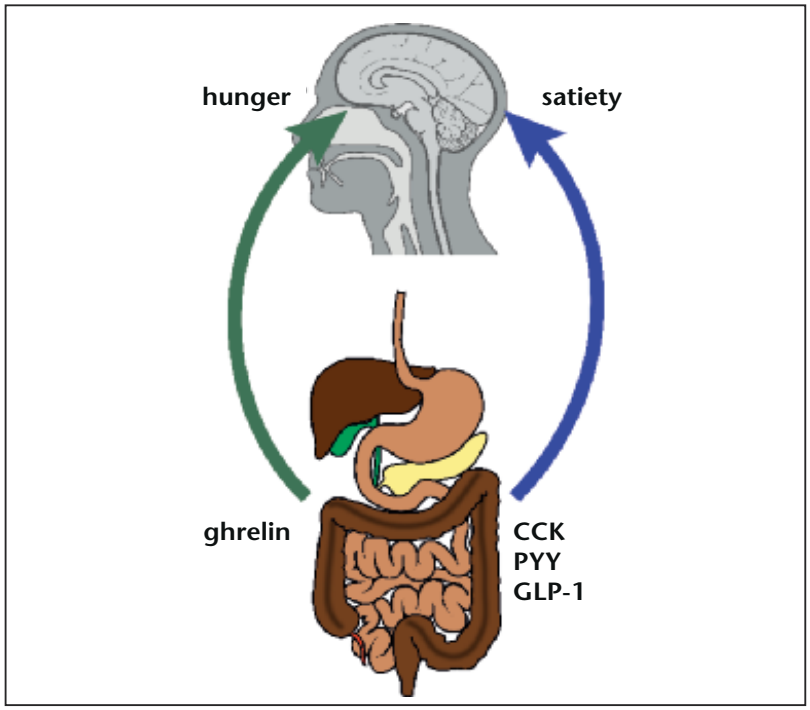

Fig. 4: **Digestive physiology influences hunger and satiety**
Ingested nutrients and presence of food in the alimentary canal result in the release of hormones from the gastrointestinal tract which act on the brain's orexigenic/anorectic control centers to modulate hunger and satiety.
CCK = cholecystokinin; GLP-1 = glucagon-like peptide 1;
PYY = peptide tyrosine-tyrosine

Conclusions

We have seen that our postprandial experiences condition us for our diet. It is obvious that subjects who previously encountered multiple foods live on different diets compared with others which were exposed to monotonous food. The mechanisms underlying preference learning and the fact that taste recognition is part of the implicit memory explain why it is so difficult to switch from an unhealthy nutrition to a healthy one.

Finally, it also becomes apparent which immense influence parents exert on their children. In accordance with the aforementioned principles, experiments with children impressively confirmed that frequent offers facilitate acceptance of originally rejected food. Children initially preferred mashed carrots over spinach. After the tenth offer, however, the children also consumed the spinach. Thus, persistently offering variable foods to children leads to the acceptance of a variety of foods and thus contributes to a healthy diet. Learned and innate behaviors appear not to be independent processes. Rather innate behaviors are subject to modulation by learned and individual experiences. Only through learned behaviors, organisms are able to adapt to their environment and expand their spectrum of foods [9].

Perspectives

At a descriptive level, the principles of eating habits are comparatively well understood. However, the molecular and cellular correlates remain almost completely unknown. Significant research efforts have to be undertaken in order to close the gaps and create the basis for successful nutritional counseling. Experimental work on suitable genetically-engineered mouse models combined with the latest physiological and neurobiological methods and sophisticated behavioral tests is a promising strategy to approach these research objectives. Based on the future advances it should also be possible to expand the knowledge about these problems also in the human system through a combination of genetic and sensory analyses with non-invasive imaging techniques of the brain.

Glossary

gustofacial = derived from lat. *gustus* (= taste), and lat. *facie* (= face)

coprophagia = intake of faeces (and thus also of nutrients produced/released by intestinal symbionts)

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Conflict of Interest
The authors declare no conflict of interest according to the guidelines of the International Committee of Medical Journal Editors.

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DOI: 10.4455/eu.2013.042