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World Journal of Otorhinolaryngology-Head and Neck Surgery (2018) xx, 1–9

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.keaipublishing.com/WJOHNS; www.wjent.org

Review Article

Conditioned taste aversions

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Received 8 February 2018; accepted 26 February 2018

KEYWORDS
Age differences;
Aversion reactions;
Avoidance;
Hypophagia;
Illness sensations

Abstract When one becomes ill after consuming a meal, there is a propensity to target a particular taste as the cause of the illness. The qualities of the taste most likely targeted include more novel, less preferred, and higher protein content. This association between a particular taste and illness is a form of learning that is termed conditioned taste aversion (CTA). A consequence of the learned association is that the taste will become aversive. When experiencing the taste again, individuals will show aversive reactions such as expressions of loathing, will experience mimicked illness sensations such as nausea, and subsequently, will avoid further exposure to the taste. The ability to acquire CTA occurs across species and across ages within a species. In the rat animal model, however, age differences exist in the capability of acquiring CTAs when increasingly longer intervals are imposed between consumption of a novel sweet solution and onset of illness. Pups have a decreased ability compared to young adults while aged rats have an increased ability. Evidence suggests that the failure of pups to acquire CTA at longer intervals is due to an immature retrieval mechanism and the facilitated ability of aged rats is due to a compromised clock mechanism that tracks the passage of time. Learned taste-illness association serves the critical function of informing individuals of the toxic nature of certain foods, thus preventing further illness and potentially death. Additionally, it contributes to the hypophagia observed during cancer chemotherapy and may contribute to the hypophagia found while suffering from bacterial infection, chronic medical conditions such as cancer, and restrictive food intake disorders such as anorexia nervosa.

Description of conditioned taste aversion

For omnivores such as humans, there is a wide range of substances that potentially can serve as food. Some of these substances provide nutrients and calories necessary for survival, but others are harmful and potentially lethal. There are particular characteristics associated with both beneficial and harmful substances that serve as identifiers

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Peer review under responsibility of Chinese Medical Association.

Production and Hosting by Elsevier on behalf of KeAi

https://doi.org/10.1016/j.wjorl.2018.02.003

Please cite this article in press as: Chambers KC, Conditioned taste aversions, World Journal of Otorhinolaryngology-Head and Neck Surgery (2018), https://doi.org/10.1016/j.wjorl.2018.02.003

and become signals for the consequences of ingestion. In humans, a number of sensory systems are engaged when processing foods, including visual, gustatory, and olfactory systems. For most mammals, however, taste is the primary cue that identifies the post-ingestion consequences of the food.

Mammals enter postnatal life with existing predispositions to exhibit a preference for and an aversion to certain tastes. That is, they exhibit positive ingestion or negative rejection reactions to certain tastes without having had prior experience with the tastes. Premature and full-term neonatal humans and neonatal rats show ingestion orofacial responses to sweet tastes and rejection orofacial responses to bitter tastes.[1–3] Ingestion predispositions also have been found for the tastes of salt,[4,5] starch,[6] and fat.[7] Post hoc explanations for these predispositions point to the caloric and nutritive value of foods with the preferred tastes and the toxic characteristics of many foods that taste bitter.

Although the positive or negative value of these predispositions generally are stable, they can change. Some changes are tied to the internal state of the individual such that preferred tastes can become temporarily less positive and aversive tastes can become temporarily less negative.[8–10] The change in internal state can be either a shift away from homeostasis or a shift back to a homeostatic state. For example, rats deprived of sodium increase their preferences for solutions that contain this substance, even when the solution contains concentrations that are normally aversive.[11] When homeostatic levels of sodium are restored, their aversion to the hypertonic solutions returns. On the other hand, reactions to calorie-rich sweet solutions change from positive to negative as humans and rats go from food depletion to food repletion.[8,12] When a depleted state returns, reactions to calorie-rich sweet solutions become positive again. These temporary shifts in hedonic value are referred to as allesthesia.

Other changes in the valence of predispositions are based on postnatal experience and are relatively permanent. The valence can be strengthened, changed from negative to positive, or changed from positive to negative. For example, preferences for sweet tastes are strengthened simply through repeated exposure. Rats increase their consumption after repeated exposure to sweet sucrose or saccharin solutions. Because saccharin does not have caloric or nutritive value, positive post-ingestion consequences are not critical for the strengthening of preference for sweet tastes.[13] On the other hand, preferences for sweet tastes change to aversions if illness follows the consumption of the sweet taste. The illness can be a consequence of consumption of a food that contains a toxic substance or it can be coincidentally associated with consumption of a nontoxic food such as happens during a bout of flu. Humans and other animals have a strong propensity to blame illness on "something I ate." The subsequent reversal in the preference for a taste (commonly referred to as the conditioned stimulus or CS) that has been associated with illness (commonly referred to as the unconditioned stimulus or US) is called conditioned taste aversion (CTA). It constitutes a learned association between the sensory properties of the taste and the sensory properties of illness and can occur when the onset of illness occurs minutes and even hours after consumption.[14,15]

In the rodent laboratory, CTA typically is induced by intraperitoneal injections of a lithium chloride (LiCl) solution after consumption of a highly palatable novel solution such as sucrose or saccharin flavored water. LiCl evokes nausea and vomiting in humans and a gaping response in nonemetic rats that appears to be an incipient vomiting response.[16–18] The consequences of acquisition of a CTA are threefold. When experiencing that sweet solution again, individuals will: (1) exhibit rejection reactions to this solution; (2) experience and show mimicked illness reactions, that is, reactions that mimic some of the behavioral and physiological reactions that occur during true illness; and (3) reduce or cease consumption of this solution (Fig. 1).

Descriptions of reactions to foods when consumption had been followed by poisoning suggest that rejection reactions are species specific. In rats, rejection responses resemble those exhibited after consumption of a bitter taste such as quinine. These responses include somatic responses (e.g., paw treading and chin rubbing), spillage of the food associated with illness from eating containers, and burying of drinking spouts containing the solution associated with illness.[12,19,20] Coyotes respond by urinating on and rolling over the food.[21] Humans report that the avoided food is distasteful and simply thinking of learned food aversions elicits facial expressions of loathing.[3,22]

Exposure to illness-inducing agents evoke a wide variety of behavioral and physiological responses. In rats, LiCl elicits hypothermia, decreased heart rate, and the behavior lying-on-belly. After a novel taste has been paired with LiCl, re-exposure to the taste will mimic these same responses.[23–25] Responses to illness also include sensations derived from the detection of negative changes in the body. One sensation that commonly is reported during illness in humans is nausea. Simply hearing or thinking about a conditioned taste elicits nausea.[22]

Reductions in consumption of a conditioned taste generally are assessed in two ways. The amount consumed before pairing the taste with LiCl is compared to the amount consumed after pairing. Comparisons also are made between the amount consumed by animals that received pairing of the taste with LiCl and the amount consumed by control animals that received pairing of the taste with normal saline.

When studied in the laboratory, animals typically are given access to only one food/drink before administering an illness-inducing agent. However, under free feeding conditions for humans and many other species, most meals include a number of different food items. Thus, identifying the culprit can be problematic. There are certain types of foods that are more likely to be targets in humans. Frequent targets are major protein sources (red meats, poultry, fish, and eggs) while infrequent targets include sweet (cakes and pies) and non sweet carbohydrates (bread, crackers, rice, and potatoes).[26–28] In addition, blame is directed towards foods that are less preferred and less familiar.

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