PERGAMON

Neuroscience and Biobehavioral Reviews 24 (2000) 173–198

NEUROSCIENCE AND BIOBEHAVIORAL REVIEWS

www.elsevier.com/locate/neubiorev

Review

Measuring hedonic impact in animals and infants: microstructure of affective taste reactivity patterns

K.C. Berridge*

Department of Psychology, University of Michigan, Ann Arbor, MI 48109-1109, USA

Abstract

The hedonic impact of taste is reflected in affective facial reactions made by human infants, other primates, and even rats. Originally studied in human infants, affective reactions to taste have also been used by affective neuroscience to identify hedonic brain systems in studies of animals (via application of neural stimulation, pharmacological activation, and neural lesion manipulations). The chief limitation of measuring affective reactions is that it is difficult for experimenters to know how to *interpret* them, and therefore how to interpret changes produced by brain manipulations. This paper notes guidelines to interpretation. It examines the phylogenetic continuity between humans, other primates, and rats in terms of the microstructure of taste-elicited affective reactions. It reviews evidence that affective taste reactivity patterns truly reflect a 'core hedonic process' of palatability or affect, rather than being an ingestion measure, consummatory behavior measure, or a sensory reflex measure. It reviews affective neuroscience studies of taste reactivity that have identified true hedonic brain substrates, and discriminated them from false hedonic brain substrates. It considers the neural bases of incentive 'wanting' versus 'liking'. Finally, it notes the difference between human subjective affective ratings of pleasure and 'core hedonic processes' reflected by behavioral affective reactions.

Keywords: Gustation; Sensation; Feeding behavior; Ingestion; Intake; Affect; Emotion; Facial expression; Neonate; Ontogeny; Development; Primate; Human; Ape; Monkey; Rat; Hedonic; Reward; Pleasure; Aversion; Distaste; Consciousness; Brain; Hypothalamus; Accumbens; Shell; Amygdala; Pallidum; Brainstem; Dopamine; Opioid; GABA; Morphine; Benzodiazepine; Video analysis; Review

Contents

* Tel.: +1-734-763-4365; fax: +1-734-936-8571.
E-mail address: berridge@umich.edu (K.C. Berridge).

0149-7634/00/$ - see front matter
PII: S0149-7634(99)00072-X

1. Introduction

We can learn much about both humans and animals from the microstructure of their behavioral affective reactions, as Darwin pointed out a century ago [1]. This paper concerns the nature and measurement of taste-elicited affective reactions, and their use to study affective brain mechanisms. I will focus on taste reactivity as a measure of *hedonic impact* in humans and animals. For discussion of other aspects of taste reactivity microstructure (related to *intake*, *ingestion pattern*, or *homeostasis*), see studies and reviews by Grill, Spector, Kaplan, Flynn, Norgren, and their colleagues [2–8].

1.1. Background and relation of human and animal taste reactivity patterns

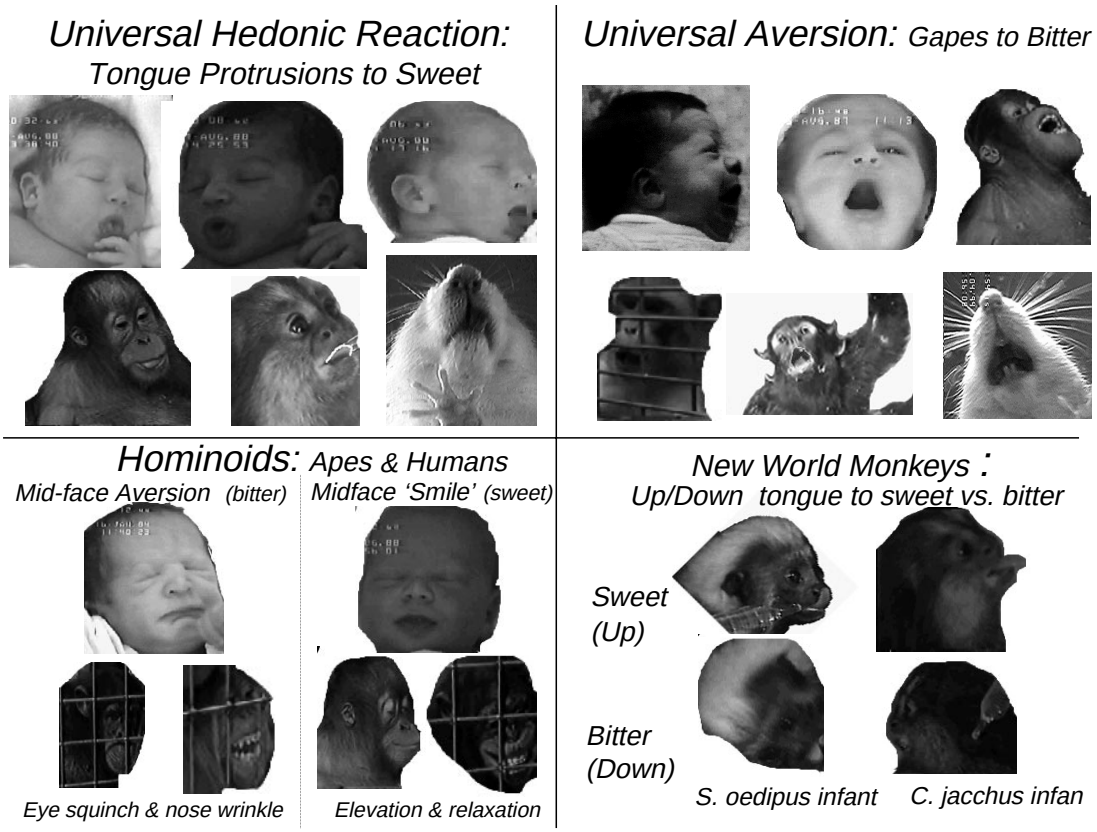
The first of the modern wave of taste reactivity studies appeared around 1970, when Jacob Steiner published photographs of the facial reactions of newborn human infants to sweet, salty, sour, and bitter tastes [9,10]. Taste reactivity measures have subsequently been applied to many topics in the psychology, physiology, and neurobiology of motivation and regulation [11–35]. In the original studies, Steiner strove to test his infant subjects typically within a few hours of birth, before their first postnatal feeding. He was concerned to demonstrate what the human newborn was capable of 'instinctively', in advance of postnatal learning about foods and their nutritional consequences. Thus the infants had absolutely no experience with tastes or their consequences (aside from prenatal swallowing of amniotic fluid) before the first squirt of sucrose, quinine, or another solution into their mouths.

Infant facial reaction patterns were originally considered to be *sensory-typical* by Steiner, who described them in terms of the eliciting sucrose, salt, quinine, and other tastes that triggered particular patterns [9,10,33]. But there were essentially just two types of pattern. Sweet sucrose elicited positive or hedonic[1] patterns of lip smacking and tongue protrusion, accompanied by relaxation of the muscles of the middle face, and an occasional smile. Bitter quinine elicited negative or aversive gapes, and complex grimaces involving retraction of the lips, 'scrinching' of the brows and muscles around the eyes, and 'wrinkling' of the nose (also flailing of the hands and arms, and small shaking or retraction movements of the head away from the taste). Salt, sour, and other tastes evoked various degrees of intermediate reactions between these extremes.[2] Steiner's original observations on human infants have since been replicated several times, both by him and his colleagues and by other groups [19,29,31,35]. Subsequent studies have improved the data resolution through video-recording and fine-grained analyses, but the basic phenomenon has remained the same (Fig. 1). And in the first affective neuroscience application, Steiner went on to show

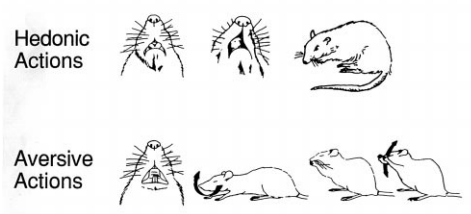
[1] Hedonic can be used with two slightly different meanings. In its narrowest sense, hedonic refers specifically to pleasure or positive effect [224], meaning "Of or relating to pleasure" according to the Oxford English Dictionary [225]. This sense as positive pleasure derives from its etymological roots and usage, ascribed to Cyrenaic philosophers, who believed that pleasure was the proper goal of action (ancient Greek *hedone* = pleasure) [225]. In referring specifically to hedonic patterns of taste reactivity, I will always follow this sense of *positive* affect. In other use, I will append positive to denote the specific pleasure sense of positive affect, since hedonic is often used more broadly in the psychological literature to mean *affect in general*, either positive or negative. The broader sense is less true to the roots of the word, but is too prevalent to be eradicated. In accordance, I will use plain hedonic for the broader sense of affect in general (as in the title of the paper).

[2] Polar opposite organization was noted by Darwin to be a general feature of affective reactions in his treatise on emotion in animals and humans [1].

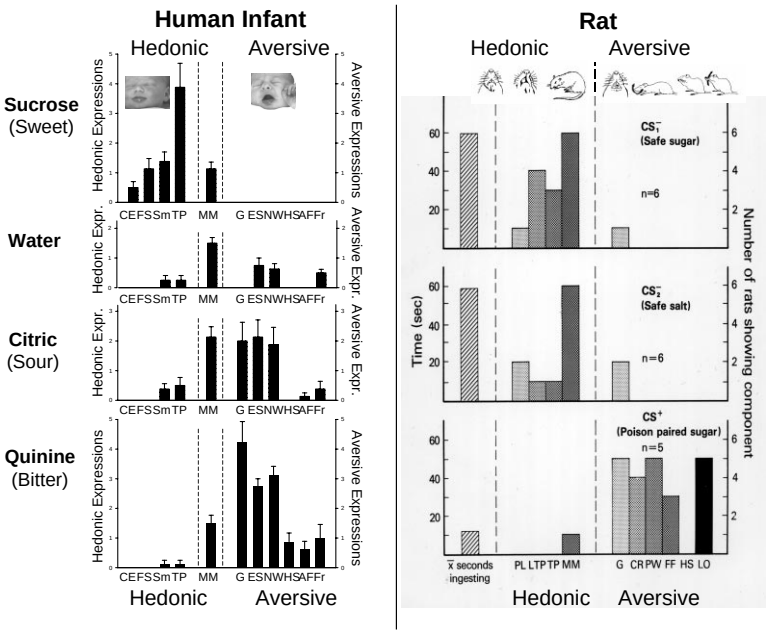
(a)


Universal Hedonic Reaction:
Tongue Protrusions to Sweet
Universal Aversion: Gapes to Bitter
Hominoids: Apes & Humans
Mid-face Aversion (bitter)
Midface 'Smile' (sweet)
Eye squinch & nose wrinkle
Elevation & relaxation
New World Monkeys:
Up/Down tongue to sweet vs. bitter
Sweet (Up)
Bitter (Down)
S. oedipus infant
C. jacchus infan

(b)


Hedonic Actions
Aversive Actions

(c)


Human Infant
Hedonic
Aversive
Sucrose (Sweet)
Water
Citric (Sour)
Quinine (Bitter)
Hedonic Expressions
Aversive Expressions
Hedonic Expr.
Aversive Expr.
CEFSSmTP MM G ESNWHSAFFr
Rat
Hedonic
Aversive
CS- (Safe sugar)
n=6
CS- (Safe salt)
n=6
CS+ (Poison paired sugar)
n=5
Time (sec)
Number of rats showing component
x seconds ingesting
PL LTP TP MM
G CR PW FF HS LO

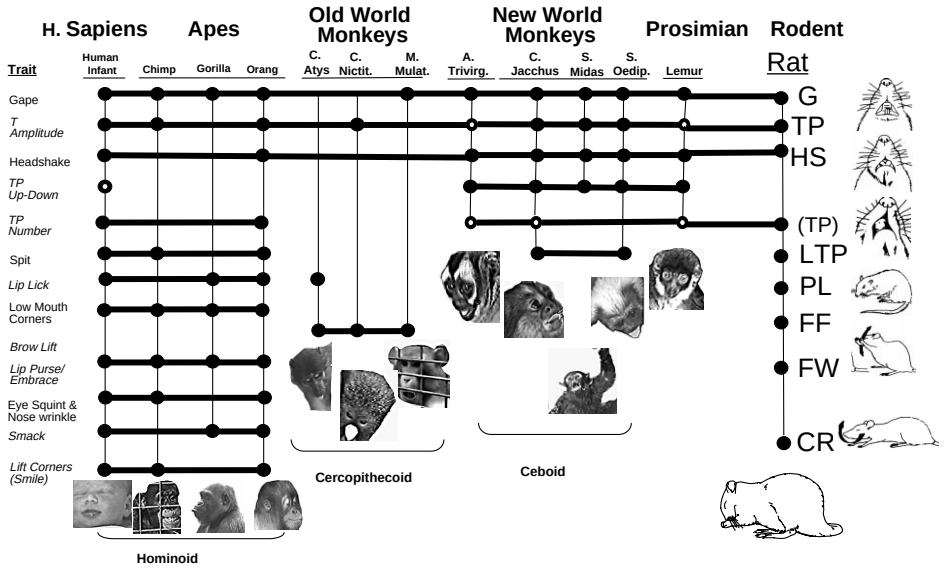


Fig. 2. Behavioral taxonomy of affective taste reactivity components across humans, other primates, and rats (figure modified from Steiner et al. [35]; primate data from Ref. [35], rat data based on Grill and Norgren [22]). Dots and lines show whether particular components are displayed and shared by particular species. Each species is represented by a vertical line (full names in Fig. 1). A solid dot along its line indicates possession of a component (listed at left for primates and at right for rats). Horizontal connections show that the component is shared across species. Open dots denote species-specific variations of a particular trait, such as the reversal of proportion of tongue protrusions to bitter versus sweet in New World monkeys. Superfamily groups are connected horizontally wherever same variant of a component is shared across more than two superfamilies. Component labels are similar to Fig. 1b.

that sweet and bitter tastes elicited the same positive and negative facial reactions from anencephalic infants and hydrocephalic infants, who were born with congenital malformation of the forebrain (or absence, in the case of anencephalics) [9].

Early studies emphasized the *sensory* aspects of taste reactivity patterns in both human infants and animals [9,22]. Only in the following decades did it become clear that facial affective reactions to taste reflected the *hedonic valence* rather than the *sensory identity* of the taste, when physiological, psychological, and pharmacological manipulation studies of animals separated *sensory versus affective* aspects of taste stimuli. Sweet and bitter usually trigger polar opposite patterns of affective reactions. Sour, salt, water, and other tastes typically evoke less intense mixtures of the two polar patterns, rather than distinct reactions corresponding to their own sensory quality. There are no unique identifying reaction patterns for sweet, salt, sour, bitter, umami (a special protein taste linked especially to glutamate that may exist for both humans and other animals [36–39]),

Fig. 1. (a) Affective reactions to taste by human infants, great apes, and monkeys. Top shows hedonic and aversive components that shared across species. Bottom shows components that belong only to one phylogenetic group of species. Hedonic tongue protrusions to sucrose are shown at top left (clockwise: three human infants, young orangutan, young *Callithrix jacchus* New World monkey, adult rat). Aversive gapes to quinine are shown at top right (two human infants, young orangutan, adult rhesus Old World monkey, adult *Saguinus midas niger* New World monkey, adult rat). Expressions made by the middle face musculature, displayed only by hominoid humans and great apes, are shown at bottom left: aversive scrinching of the nose, eyes and/or brow to quinine (human infant, adult chimpanzee, adult gorilla), versus hedonic relaxation of these muscle groups coupled with elevation of the mouth corners to sucrose (human infant, young orangutan, adult chimpanzee). Directional coding of tongue protrusion trajectory upwards to sweet but downwards to bitter, displayed only by New World monkeys, are shown at bottom right: (young *Saguinus oedipus* and *Callithrix jacchus* monkeys). (photographs from Steiner et al. [35], and from personal observations). (b) Original depiction of affective reaction patterns to taste by rats. Hedonic reactions include rhythmic tongue protrusions on the midline, lateral tongue protrusions (which are discrete and nonrhythmic events), and paw licking. Aversive reactions include gapes, head-shakes, face washes (or face wipes), and paw flails (based on Grill and Norgren [22]). (c) Quantitative display of hedonic and aversive taste reactivity components made by human infants (left) and rats (right). Human infant reaction patterns were elicited by sucrose, water, citric acid, and quinine solutions (from Steiner et al. [35]). A shift from hedonic patterns to aversive infant reactions can be observed across those four tastes. Rat reaction patterns show further that the response to a taste is changed when its palatability is altered by taste aversion conditioning (from Berridge et al. [46]; response incidence rather than response quantity is shown, because the study was conducted prior to the development of the time-bin scoring procedure for quantity). Normal hedonic reactions to a sugar (either fructose or maltose) are shown at top; similarly largely hedonic reactions to an isotonic NaCl taste are shown in the middle. Conditioned aversive reactions are shown at the bottom to the taste of the same sugars used for the top (fructose or maltose), after one sugar had been paired associatively with LiCl injections that induced illness. Human hedonic component labels are: CE = corner elevation of the mouths and lips; FS = finger suck; Sm = smacks of mouth and lips; TP = tongue protrusion. Neutral component is MM = mouth movements, irregular and involving the lips. Human aversive components are: G = gape; ES = eye squinch; NW = nose wrinkle; HS = head-shake; AF = arm flail; Fr = frown (corner depression of mouth and lips). Rat hedonic component labels are: PL = paw lick; LTP = lateral tongue protrusion; PT = rhythmic midline tongue protrusion; MM = mouth movements. Rat aversive components are: G = gape; CR = chin rub; PW = paw wipe (equivalent to face wash or face wipe); FF = forelimb flail; HS = head-shake; LO = locomotion.

or other basic tastes. If taste-specific patterns existed, that would imply *sensory coding* of reactions, and would allow an observer to infer the sensory content of the taste reliably from watching the reaction. But with the possible exception of lip pursing (which may be a distinct human reaction to sour [29]), no facial reaction is absolutely specific to a particular taste sensation. "Bitter-typical" expressions can be elicited by concentrated salty, sour, and other tastes. "Sweet-typical" reaction patterns can be elicited by milk or other complex tastes, by dilute NaCl, and by other tastes. An observer therefore cannot tell the precise sensory quality of a taste based on an infant's reaction. By contrast, however, an observer can make an excellent inference concerning whether an human infant '*likes*' a taste based on her or his facial expression—that is, infant facial reactions seem to be an excellent indicator of taste palatability or hedonic impact (Fig. 1) [35].

Human infants are not the only members of our species, of course, to display distinctive facial expressions to positive or negative tastes. Adults do so too. Human infants, however, may be more responsive and 'honest' in their facial expressions than older children or adults. That is perhaps because of the socialization of human emotional expression in early life, and because human voluntary control over facial expression can prevent human facial expression from being an accurate indicator of underlying hedonic impact [30]. As social individuals, we may suppress real emotional reactions or counterfeit false ones. Thus studies of adults or older children have been less successful than infant studies in using facial expressions to measure affective reaction to tastes or odors [20,32], even though adult facial expressions do often express the hedonic impact of food [40]. The facial reactions of some human adults are less constrained by social motives than is ordinarily the case, and Steiner and his colleagues have obtained clear hedonic versus aversive patterns in Alzheimer's patients and in certain other neurological populations [41]. Yet as a general rule, the facial reaction measure of hedonic impact may be most sensitive when applied to human infants or to animals from other species (of any age).

1.2. Evolutionary continuum of primate taste reactivity microstructure

In a series of comparative studies, Steiner and Glaser examined taste reactivity microstructure in great ape, old world monkey, new world monkey and prosimian infants and adults [34,35,42]. Virtually all of those primates emit facial reactions that are appropriate to the human-judged palatability of bitter, sweet, or other tastes, and similar to reactions of human infants. For example, quinine typically evoked gapes and other aversive responses from chimpanzees, gorillas, orangutans, and monkeys [34,42]. By contrast, sucrose evoked tongue protrusions, mouthing movements, and sucking, and other tastes evoked appropriate intermediate responses, from most species of primates [34,42].

The comparative microstructure of human infant and other primate taste reactivity was analyzed explicitly in a recent comparative study by Steiner et al. [35]. In that study, affective reactions to tastes were compared among 12 different primate species (including humans, great apes, Old World monkeys, and New World monkeys). If one believed that the facial expression of hedonic impact is qualitatively unique to human beings then one might have expected that human infant facial reactions would be quite different from all other primates, and that other primate species might be less differentiated from each other. However, that was not the case. Instead, human infant facial reactions to sweet, bitter, and other tastes were found to be closely similar to those of chimpanzee, orangutan, and gorilla. When components were plotted to reveal similarity among species, a single 'hominoid cluster' was formed that contained both human and great apes (humans and great apes together belong to a single phylogenetic group called hominoids, whereas all monkeys belong to different groups). In terms of microcomponent structure, human taste reactions were more similar to great apes, than great apes were to monkeys (Fig. 2). Old World monkeys that evolved in Africa or Asia formed a separate cluster of their own, while New World monkey species that evolved in South America formed a third cluster (this cluster also seemed closest to the single prosimian species that was tested).

The results of Steiner et al.'s [35] comparative analysis indicates that some components of taste-elicited reactions are universal among all primates, such as gapes to bitter or rhythmic tongue protrusions to sweet. Other components belong only to particular species or groups of species. For example, only humans and other hominoids such as great apes responded to sweet tastes with complex lip smacking, or responded to bitter tastes with the complex 'scrinching' movements involving musculature of their brows, nose, and middle face. Conversely, only South American monkeys appear to show affectively coded shapes of tongue protrusion (forming an upward pointing trajectory to sweet but a downward trajectory to bitter) [35].

The upshot of all this is that the particular configuration of components shown by a particular primate is largely a function of genotype. No two primate species are identical in the microstructure of their facial reactions to taste, but the degree of difference between them is continuous rather than categorical, and is proportional to the phylogenetic distance between the species.

1.3. Taste reactivity patterns of nonprimate animals

The evolutionary continuum of taste reactivity microstructure extends to nonprimate animals as well. Grill and Norgren published a landmark study in 1978 of the behavioral taste reactivity components emitted by rats [22]. The affective reactions of rats are distinctly related

Table 1
Match between prediction and data for allometry rule of duration. Species or superfamily body weights (average adult), and predicted and observed cycle durations for rhythmic tongue protrusions. Predicted durations were generated by the allometric equation, sec duration = 0.26 × (kg body weight)$^{0.32}$. Note that the predicted duration specifies a *predicted range for the species* as a whole, and heavier members are not predicted to have longer durations than lighter members of the same species. Observed cycle durations for gorillas, infant humans, chimpanzees, Old World monkeys, and New World monkeys were taken from Steiner et al. [35]. Observed durations for rats taken from Grill and Norgren [22] and from personal observations. Observed lick cycles for mice were provided by K.N. Hewitt and P.G. Clifton (personal communication) based on their own lickometer data

Species or superfamily	Species adult weight (kg)	Predicted duration (s)	Observed duration range (s)
Gorilla	90–160	1.1–1.3	1.2–1.3
Human	70–100	1.0–1.1	0.8–1.2
Chimpanzee	35–45	0.8–0.9	0.8–0.9
Old World monkeys	5–10	0.4–0.5	0.3–0.5
New World monkeys	0.25–1.0	0.16–0.26	0.18–0.25
Rats	0.25–0.5	0.16–0.20	0.12–0.15
Mice	0.02–0.04	0.07–0.09	0.09–0.11

to those of primates (Fig. 2). Infusions directly into the mouth (through an implanted oral cannula) of bitter quinine elicited gapes, head shakes, forelimb flails, and other reactions from rats, which Grill and Norgren termed aversive, following Craig's 1918 definition of aversion as "a state of agitation which continues so long as a certain stimulus, referred to as the disturbing stimulus, is present" [43; p. 91]. These reactions were scored in fine detail by Grill and Norgren in a frame-by-frame video-analysis. Conversely, sucrose elicited a different pattern of reactions Grill and Norgren called 'ingestive', including rhythmic tongue protrusions and mouth movements, and 'lateral tongue protrusions' (somewhat like a lateral licking of the lips or chops) [22,23,44]. Hamsters also show similar positive reactions to sucrose (lateral and midline tongue protrusions) and aversive reactions to quinine (gapes, chin rubs, headshakes, forelimb flailing, etc.) [45].[3] Subsequent studies of taste reactivity patterns have added paw licking to the list of positive or 'ingestive' sucrose-elicited reactions for rodents [21,46], compiled procedures for comparing humans and nonhumans [35], and developed methodological improvements, such as time-bin scoring procedures to balance hedonic and aversive categories of reactions [21,47,48], and methods to avoid the masking of palatability shifts by response demand properties of the taste [2].

[3] Positive hedonic reactions to sweet tastes seem to be shared more widely among species than ecological considerations would suggest. Even species that normally restrict their diet to leaves (gorilla, orangutan, hamster, guinea pig) or seeds show positive taste reactivity to sucrose (and choose sucrose in preference tests [226]), just as do omnivore species that also normally eat sweet fruits and other foods (chimpanzee, rat). The relative ubiquity of hedonic reaction to sweet, and of sweet preference (domestic cats, as carnivores, are among the very few mammals positively known not to express a sweet preference; for an excellent review of comparative taste preference see Beauchamp and Mason [226]) may indicate that hedonic reactions to sweet evolved relatively early in primate and rodent ancestors, prior to the specialization of particular species into monophagous herbivore niches restricted to grass and leaves or to seeds and roots.

1.4. Comparing microstructure: allometric timing rule relates humans to rats

Aversive gapes and positive hedonic tongue protrusions are universal affective expressions, emitted by human infants, other primates, and rats. To say that the 'same' microcomponent (for example, rhythmic tongue protrusions to sucrose) is emitted by two different species is not to say that the movements are *identical*. After all, physical anatomy of the 'same' part varies across different species. For example, all mammals have a head, but its shape and size may be different in different species. It is not surprising, then, that *behavioral morphology* varies systematically too, even for the 'same' (i.e. analogous and homologous) taste reactivity microcomponent. The important point is that the variation must be merely systematic if the microcomponent is to be considered truly the 'same'.

An instance of 'merely systematic variation' is the scaling of component *speed* to body *size*. The duration of the 'same' stereotyped reaction in different species appears to be directly proportional to the average adult body size (mass) for that species. Such size-based timing rules are called *allometric*, and are very common in physiology and behavior (the duration of heartbeat rhythms, walking step cycles, etc. [49]).

Certain taste reactivity components also follow an allometric timing rule, especially those that are highly *rhythmic* and/or *stereotyped* in duration. A good example is rhythmic tongue protrusion to sucrose, which by definition follows a precise rhythm. Another example is gape to quinine, which is quite stereotyped in duration. Allometric timing can give rise to a perceptual illusion of difference between species of different sizes. For example, when one compares rhythmic tongue protrusions made by a human infant with rhythmic tongue protrusions made by a New World Saguinus or Callithrix monkey, the rhythm is so different in speed as to appear almost to be a totally different reaction. Human infant tongue protrusions are slow and almost languid, about 1 per second, whereas the South American monkeys emit

rapid-fire tongue protrusions more than three times faster. Yet both species follow the same generative timing rule in which movement duration is directly proportional to body mass [35]. These rates correspond to an average human adult body mass of around 80 kg, whereas the average adult body mass for these New World monkeys is only about half a kilogram.

The allometric timing rule can be stated in the form of an equation. For primates, and perhaps all mammals larger than about one-half kilogram, the timing of rhythmic tongue protrusions is: duration (sec) $= a \times$ (average body mass in kg)^b, where a and b are both constants ($a = 0.27,\ b = 0.32$) [35]. Steiner et al. found that the actual duration of primate tongue protrusions closely follows this allometric rule ($r = 0.88$; Fig. 2). Therefore gorillas (weighing about 100 kg) have tongue protrusion cycles even slower than humans (approximately 1000 ms), whereas chimpanzees (weighing about 60 kg) are slightly faster (approximately 850 ms) than humans [35]. Old World monkeys are faster still (approximately 480 ms on average), and tiny New World monkeys have the fastest tongues of all primates (approximately 200 ms) [35]. In general, as average adult weight increases across species, the b exponent value of 0.32 means that the duration of cycles grows roughly in proportion to the increase in the cube root of body mass. A similar allometric rule applies to the duration of gapes [35].

Although the duration of the components are predicted on the basis of *adult* body mass (Table 1), the rule also appears to apply equally to infants of each primate species [35]. Infants have components that are similar in duration to adults of the same species, despite the size difference between infants and adults. Thus human infants follow the adult rule in their timing (corresponding to adult body mass, and not to their own small body mass), and so do infant New World monkeys [35]. This suggests that the timing evolved parameters evolved to coordinate adult movements, perhaps because they have the greatest impact on fitness. It means also that the *timing parameters are programmed into the brain* for both infants and adults. The duration cannot result passively from the physics of movement, since if it did small infants would be faster than adults. The only alternative is that timing must be actively imposed by neural central pattern generators. Infant brain timing circuits are genetically coded in advance to conform to the species-typical size they will have if they grow to be adults. Similar infant anticipation of adult timing, and genetic programming of timing parameters, has been found for other types of stereotyped movement [50].

Interestingly, it appears allometric timing rules for hedonic and aversive reactions might also extend beyond primates, connecting humans to rodents. For example, it is interesting to note that Grill and Norgren [22] originally reported that rats have a timing of approximately 120 ms for the duration rhythmic tongue protrusion cycles (in our lab, we generally obtain a range of 120–150 ms for rats that weigh approximately 300–400 g; the equation predicts 180 ms). And mice, weighing only about 30 g, have a tongue protrusion lick cycle of about 90–110 ms duration (mouse lick cycle data provided by K.N. Hewitt and Dr

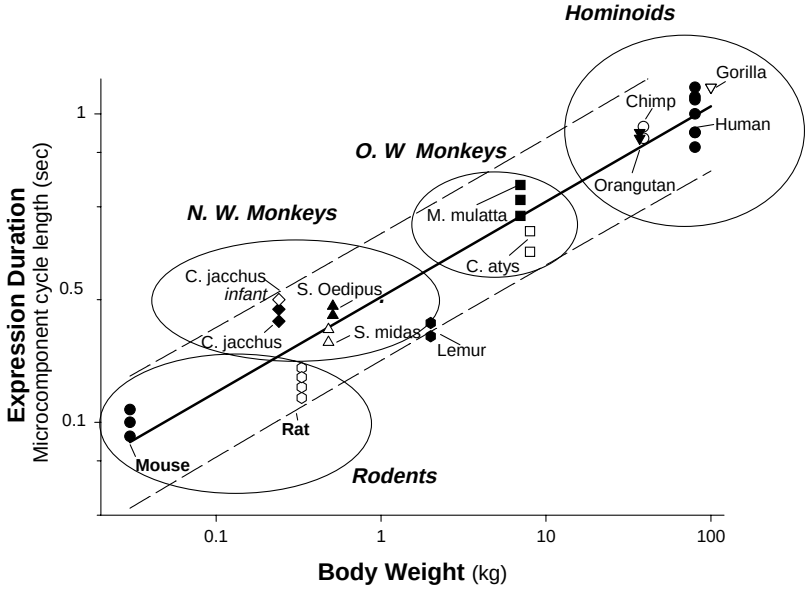


Fig. 3. Allometric timing rule for duration of rhythmic tongue protrusions made by human infants, other primates, and rats and mice (graph modified from Steiner et al. [35]). Identical symbols show individuals of the same primate species and age group (primate data from Ref. [35]). The duration of a rat and mouse tongue protrusion cycle is represented by the rat drawing (data based on Grill and Norgren [22] and personal observation; mouse data courtesy of K.N. Hewitt and Dr Peter G. Clifton). The vertical axis shows component duration (in seconds along logarithmic scale) and the horizontal axis shows average body mass (in kilograms along a logarithmic scale). For more detail, see Steiner et al. [35]. The dark diagonal line shows the allometric function generated by the equation, ms cycle duration $= 0.26 \times$ (kg average adult weight)$^{0.32}$. The dashed lines show the limits of the 95% prediction interval.

Peter Clifton, personal communication, August 1999 and Ref. [51]; the equation predicts 85 ms).

Of course it would be necessary to do an explicit comparative study before drawing a strong conclusion, but these values correspond rather well to the extrapolation from the primate allometric line shown in Fig. 3. I have repeated the nonlinear regression analysis of Steiner et al. after adding rodent data to the primate data, and find little change in the values for the allometric equation ($y = a \times x^b$). The exponential *b* value remains 0.32 in both cases, and the value of the *a* constant changes from 0.27 to 0.26. This is hardly any change at all, and the correlation between duration and body weight (r^2 value) actually improves from 0.88 to 0.91 after adding the rodents to the primates. Thus the underlying relationship between timing and body mass appears to span from gorillas to mice. Even though a gorilla weighs 3000 times more than a mouse, each follows an equivalent allometric timing rule, the parameters of which are likely programmed into the motor circuitry of their brains.

2. Taste reactivity patterns as a measure of palatability

We now return to the important question of how to interpret positive versus negative patterns of taste reactivity as measures of hedonic impact. The central point of this paper is that these patterns reflect *core processes of positive hedonic impact and negative aversive impact*. Admittedly, this was not the initial interpretation of taste reactivity patterns. Originally, taste reactivity patterns were interpreted as either *sensory reflexes* [9] or as a measure of *intake* (that is, the decision either to *ingest* a substance or not) [22,23]. But many subsequent studies have now shown that the *affective pattern* of taste reactivity components reflects *palatability or affect* more closely than it reflects either *ingestion* or *sensation* [52–54]. If this is true, then it makes more sense to use the terms *hedonic pattern* versus *aversive pattern* to describe positive and negative affective taste reactions, rather than *ingestive pattern* versus *rejection pattern* or other alternative terms.

2.1. Taste reactivity patterns reflect palatability

One reason to believe that hedonic/aversive taste reactivity patterns reflect affective processes is that they correspond to human subjective ratings of taste pleasure in many situations. To conclusively test the relationship between affect and affective reaction, it is necessary to separate affective from sensory properties of a taste, so as to show that a hedonic reaction, for example, reflects the pleasure of a sweet taste rather than the intensity of its sweetness sensation. This can be done via physiological, psychological, pharmacological, or neural manipulations that keep the *sensory* properties of a taste unchanged, while altering its *palatability*.

A few studies of human infants have found evidence that the facial affective reaction to a taste is altered by particular experiences or physiological states relevant to palatability. For example, Crystal and Bernstein [55] reported that the taste of NaCl elicited a less aversive reaction from human infants who inadvertently had been exposed to conditions that would promote sodium deficiency as fetuses (owing to severe morning sickness on the part of their mother) than infants who had never been sodium deficient. Similarly, Soussignan and colleagues reported that human newborns who had recently been bottle fed were more likely to respond with aversive facial reactions to the smell of their milk formula than infants who had not recently been fed [56]. These observations suggest that the facial reactions of human infants are not simply to the sensory properties of the taste or odor (discussed further below).

Ethical concerns naturally preclude more dramatic manipulations of human infants that might further distinguish between sensory versus affective control of reactivity. Much more has been done in animal studies to resolve the issue of whether taste reactivity truly reflects hedonic and aversive affect [11,21]. Manipulations capable of changing the human subjective affective perception of a taste virtually always change the taste reactivity patterns of rats in ways that correspond to the change in human affect (at least, if the manipulation can be applied to animal studies). Human subjective reports and rat affective reactions thus both seem to reflect similar ‘likes’ and ‘dislikes’.

Physiological manipulations of affect. For example, regarding salt appetite and palatability, physiological sodium depletion has been shown several times in rats to produce a very dramatic enhancement of hedonic reactions to a concentrated salty taste, and to abolish aversive reactions [57–63]. This corresponds to the milder alliesthesia (enhancement of pleasure by a physiological depletion state) for sweetness that occurs during ordinary hunger [64]. The sensory pleasure of sweetness to humans is enhanced by hunger and suppressed by caloric satiety [64–66]. Similarly, hedonic reaction patterns of rats to sweet tastes are enhanced by hunger and are suppressed by caloric satiety [2,13,67,68].

Likewise, the palatability of saltiness for humans is selectively enhanced by physiological sodium deficiency (salt appetite) [63,69]. As already mentioned, human infants who were exposed to prenatal conditions that may produce a salt appetite, associated with their mothers’ severe morning sickness [70], have been reported to show fewer aversive reactions to a very salty taste than do other infants [55]. Similarly, hedonic reactions of rats to NaCl are dramatically increased, and aversive reactions dramatically decreased, by sodium depletion [59–62].

Psychological manipulations of affect. Taste pleasure for humans can be abolished and replaced with subjective aversion by associative pairing of a palatable food with gastrointestinal illness [71]. Similarly, hedonic reactions of rats to sweetness are abolished and replaced by aversive behavioral reactions by pairings of taste with LiCl or certain

other noxious agents [72–74]. Conversely, aversive reactions are diminished, and hedonic reactions enhanced by associative pairing of a mildly unpalatable taste or other conditioned stimulus with a strongly hedonic taste (unconditioned stimulus) [58,75,76], or by giving rats experience with the pharmacologically rewarding consequences of ingesting a particular taste [77–79]. These experience-induced shifts in rat affective taste reactivity patterns seem reminiscent of the conditioned subjective aversions developed by humans for illness-paired tastes, and of human acquired subjective preferences for originally disliked tastes [71].

3. Affective neuroscience: use of taste reactivity to identify hedonic brain systems

A primary biopsychological use of the affective taste reactivity measure has been to identify *brain mechanisms that mediate the hedonic or aversive impact* of the taste (Fig. 3). Most of these studies involve techniques that can only be used in animals, and thus animal studies have produced insight into hedonic brain organization that could not have been obtained in other ways.

3.1. Hedonic neuroanatomical and neurotransmitter systems

Affective neuroscience studies using taste reactivity patterns have identified a number of *neuroanatomical* brain structures as crucial to the mediation of *hedonic impact* (Fig. 3). These include the far-lateral hypothalamus (actually ventral pallidum) [80–83], the nucleus accumbens shell [27], sites in the brainstem, and possibly the amygdala. Lesions and various forms of neurochemical stimulation directed to these structures can cause dramatic changes in hedonic or aversive reaction patterns elicited by food. In recent years, increasing attention has been given to the relevant neurotransmitter system within these brain structures that mediate hedonic impact.

3.1.1. Opioid systems

Pharmacological techniques have identified several *neurochemical* systems that make crucial contributions to taste affect. Manipulation of these neurochemical systems by drug administration markedly alters the balance of hedonic/aversive reactions. Considerable evidence indicates that opioid neurotransmitter systems mediate *positive hedonic palatability* [84–88]. A taste reactivity study has further pinpointed the shell of the nucleus accumbens as a site of opioid-mediated hedonic enhancement. In a recent dissertation study, Peciña used intracranial microinjections of morphine and a fos-based measure of neuronal activation to map where in the brain morphine elicited feeding and enhanced hedonic reactions. She identified a specific hedonic opioid site within the caudoventral portion of the shell of the nucleus accumbens [27,89,90]. A role for opioid systems in taste hedonics is consistent with suggestions from intake and food preference studies of animals that opioid systems mediate food palatability [91–96], and also with some studies of decrements in the human subjective perception of food pleasantness after administration opioid agonist drugs [97–100].

3.1.2. Benzodiazepine systems

The benzodiazepine/GABA system also has been identified by taste reactivity studies as a mechanism of positive hedonic taste affect [48,101,102]. Activation of benzodiazepine receptors facilitates the activation of GABA receptors and so enhances consequent Cl^- ion influx into neurons that express the receptor. Benzodiazepine agents such as diazepam, chlordiazepoxide, or midazolam, promote feeding in animals and humans [102–105], an effect which was suggested in the 1980s by Cooper to involve a drug-induced increase in the hedonic palatability of food [103,106]. Subsequent taste reactivity studies have clearly confirmed Cooper's hypothesis. When administered to rats peripherally, or into the cerebral ventricles, or directly to the hindbrain, benzodiazepines enhance hedonic reaction patterns just as morphine does [107–114]. Food consumption by humans is increased by benzodiazepine administration [110], which is consistent with a palatability enhancement (although it is not proof). As yet the effect of benzodiazepines on human ratings of palatability still have not been explicitly studied, and remain to be examined. The results of animal taste reactivity studies make a clear prediction about the psychopharmacology of human hedonic impact in this case—and time will tell whether the prediction is true.

3.1.3. Serotonin systems

Finally, a possible role for serotonin in hedonic processing has been suggested by two studies of the effects of D-fenfluramine on taste reactivity. Fenfluramine, which promotes serotonin release and blocks reuptake [115], suppresses voluntary intake. Barnfield, Parker and colleagues reported fenfluramine to increase aversive taste reactivity patterns [116], and Gray and Cooper found it to suppress positive hedonic reaction patterns [117]. This suggests that serotonin causes a specific negative shift in palatability. On the other hand, another study by Treit and Berridge [114] found that the serotonin receptor agonists gepirone and buspirone both seemed to have merely a sensorimotor suppression effect on taste reactivity patterns, and did not shift the affective balance between hedonic and aversive patterns. The role of serotonin systems in mediating hedonic impact thus deserves further study.

3.2. Identification of brain systems that are 'false hedonic candidates'

Equally important as the identification of affective brain substrates have been demonstrations of *failures* to modulate

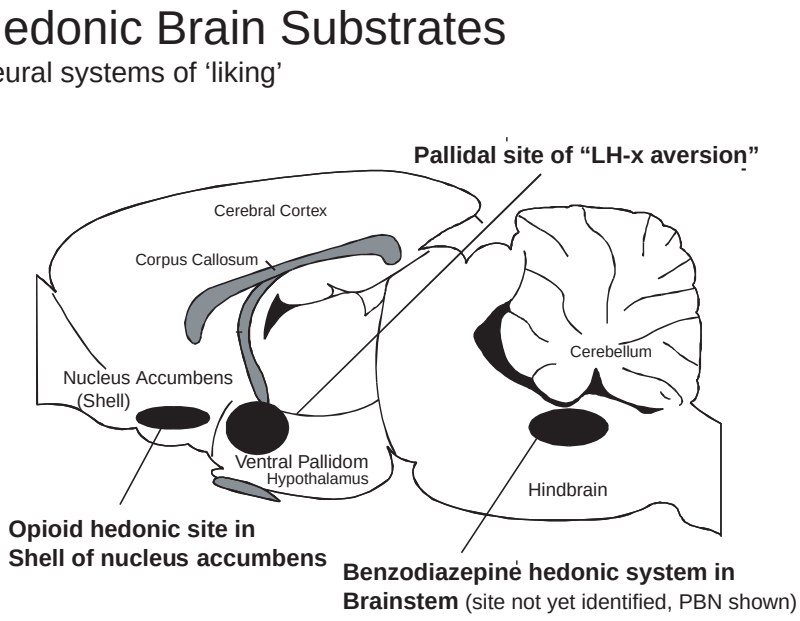


Fig. 4. Brain systems that mediate the hedonic impact of taste. These 'liking' brain systems have been identified by affective neuroscience studies that employed taste reactivity measures. See text for explanation. Based on: for hedonic opioid site in the shell of accumbens [27,86–88,90], for pallidal site of LH-lesion aversion [81,154], and for benzodiazepine brainstem systems [48,111,112,222,223].

taste affect by brain manipulations that change other aspects of food reward or the motivation to eat (Fig. 4). This is true especially when the relevant brain systems have been thought to be candidates for hedonic neural substrates. The importance of distinguishing between substrates of 'wanting' versus 'liking' arises in part from neural dissociations such as those below, in part from the close tie it highlights between 'wanting' systems and associative learning in guiding incentive motivation, and in part from its ability to resolve paradoxes, such as why an antagonist drug that is presumed to reduce the impact of a reward might lead to less rather than more behavior intended to obtain that reward (see Berridge and Robinson for discussion [12]). 'False hedonic candidates' among brain substrates, as identified by taste reactivity studies, include the ascending mesolimbic system, the central nucleus of the amygdala, most or all of the lateral hypothalamus itself, and neurochemical systems such as dopamine and bombesin [11,12,28,52, 61,81,118,119].

3.2.1. Mesolimbic dopamine systems

Perhaps the most significant among these 'false hedonic candidates' is the mesolimbic dopamine system, since many have long believed these dopamine projections to be one of the brain's chief 'pleasure systems' for food and other rewards [120–126]. The results of a series of taste reactivity studies using dopamine antagonist drugs, agonist drugs, electrical stimulation, or dopamine-depleting brain lesions, now point clearly to the conclusion that whatever dopamine systems contribute to the process of reward, they do *not* mediate the hedonic impact of tastes [11,12,28,114,119]. Although several taste reactivity studies by Parker and colleagues in the early 1990s suggested that pimozide might shift the hedonic impact of tastes [127–129], the results of further collaborative studies by Parker and our lab showed that the behavioral changes reflected a sensorimotor general suppression, and not a shift in the balance of hedonic versus aversive reaction patterns [28]. Even massive depletion of mesolimbic dopamine by 6-OHDA lesions has been shown to leave unsuppressed the hedonic impact of tastes, in two studies by Berridge and Robinson [12,119]. A similar 'sensorimotor-not-hedonic' interpretation seems to apply to the effects of dopamine agonist drugs on taste reactivity since both hedonic and aversive reactions can be suppressed by drugs such as amphetamine [11,114,130] (and Peciña and Berridge, personal observations). Even amphetamine microinjections directly to the nucleus accumbens that promote motivation to work for a food reward seem to fail to enhance hedonic impact (Wyvell and Berridge, in preparation).

In general it can be concluded that evidence from taste reactivity studies militates strongly against a hedonic role for dopamine. That of course raises the fascinating question of what dopamine *really does* (my colleague Terry Robinson and I have offered a possible answer in Ref. [12]). Dopamine manipulations change behavioral measures that infer hedonic impact based on an individual's willingness to obtain a reward in ways consistent with changed pleasure. That is, such measures infer how much a reward is 'liked' based on how much it is 'wanted'. The paradox of changed 'wanting' without change in 'liking' points to the hitherto unsuspected existence of a psychological process that can masquerade as hedonic impact in a host of psychological tests, yet not be hedonic impact. Regarding

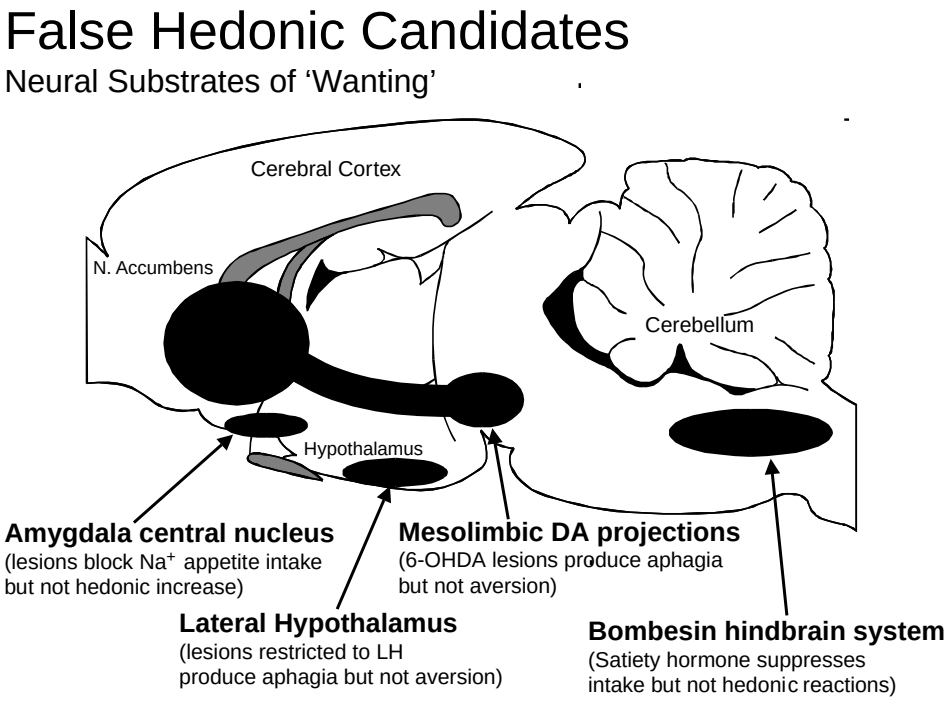


Fig. 5. 'False hedonic candidates' that fail to mediate hedonic impact (even if they mediate some other aspect of reward). These 'wanting' brain systems mediate the motivation to eat, but they do not cause hedonic or aversive shifts in taste reactivity patterns. See text for explanation. Based on: for mesolimbic dopamine system [11,12,28,114,119,177], for amygdala central nucleus [61], for lateral hypothalamus [81,118,154,193], and for bombesin [52,159].

dopamine in particular, my colleagues and I have called this process '*incentive salience*', and have attempted to define some of its properties [11,12,118,119,131,132]. Incentive salience, we suggest, masquerades as hedonic impact only when measured in ways that infer 'liking' based on changes in 'wanting'-related responses [12] (Fig. 5).

3.2.2. Accumbens GABA systems

Robust eating is elicited from rats by microinjection of glutamate antagonists into the nucleus accumbens, which block excitatory glutamate inputs to GABAergic medium spiny neurons [133]. Eating is similarly elicited by accumbens microinjections of GABA agonists such as muscimol, which also might be expected suppress the output of GABAergic medium spiny projections to other regions such as the ventral pallidum [134–136]. Kelley and colleagues showed that accumbens muscimol microinjections increased the intake of sucrose solutions but not of saccharin solutions, and suggested that the increase in consumption therefore was not accompanied by an increase in the palatability or reward properties of the tastes [134]. We have conducted pilot studies of the effects of accumbens muscimol microinjections, and our preliminary results support their conclusion. Our observations suggest that eating induced by the GABA agonist in the nucleus accumbens may be modulated by environmental conditions, and is not accompanied by an enhancement in hedonic reaction patterns to taste (Reynolds and Berridge, personal observations). Although much work remains to be done, the evidence so far indicates that these manipulations of accumbens GABA neurons may cause rats to 'want' food more, but not to 'like' it more.

3.2.3. Amygdala

The *central nucleus of the amygdala* is another brain system that has been suspected to mediate the hedonic impact of foods, based on intake or preference tests, but appears not to do so when examined by affective taste reactivity tests. One reason for having been suspected of mediating taste's hedonic impact is that amygdala lesions have been reported to change the food preferences of monkeys [137,138], and to reduce neophobia and taste aversion in rats [139–141]. Electrolytic lesions of the central amygdala also abolish salt appetite in rats, the preference for NaCl solution that ordinarily accompanies states of physiological sodium depletion, and simultaneously abolish the reward value of salt in instrumental tests [142–145]. But surprisingly, lesions of the central nucleus of the amygdala do not suppress at all salt-appetite alliesthesia, that is, the hedonic enhancement of salt palatability that occurs after sodium depletion [61]. Physiological depletion of sodium still causes a dramatic increase in hedonic reactions to a highly salty taste, and still suppresses the aversive reactions that are ordinarily elicited [61]. Thus the amygdala-damaged rats appear to 'like' the salty taste, and not to 'dislike' it, when sodium depleted, but in spite of that they still do not 'want' the salty taste they 'like' [61,144].

An interesting question is precisely how the neural lesion produces this effect. For example, is it due to loss of central amygdala neurons or instead to loss of fibers from the cortex? Another interesting question is how the abolition of salt 'wanting' (but not 'liking') by central amygdala lesions relates to the incentive salience deficits produced by dopamine lesions. Although it is unlikely that amygdala lesions eliminate the basic attribution of incentive salience to 'wanted' stimuli, unlike dopamine systems, they nonetheless may mediate the *selection* and associative *targeting* of incentive salience on to *particular stimuli* [131]. A final question is how the apparent role of the central amygdala in salt 'wanting but not liking' compares to the role of other amygdala nuclei in mediating hedonic impact. An early taste reactivity study by Simbayi and Boakes implicated the basolateral nucleus of the amygdala as a possible substrate for the change in 'liking' produced by taste aversion learning, because lesions there apparently blocked the shift from hedonic to aversive patterns [146]. That picture is somewhat obscured, however, by Dunn and Everitt's report [147] that damage to the overlying cortex, and not to the amygdala, is actually responsible for the deficits in taste aversion learning, together with Kiefer's report [141] that only taste *avoidance*, and not taste *aversion*, is blocked by lesions of the gustatory cortex. Thus at present it remains unclear as to what are the exact roles of amygdala versus neocortex in mediating 'liking' versus 'wanting' changes after taste aversion learning. The role of amygdala nuclei in hedonic impact clearly deserves further investigation.

The *lateral hypothalamus* proper (LH) is another 'false hedonic substrate', at least in the sense that most of the LH does not causally mediate the hedonic impact of taste. This is surprising, since neurons in the lateral hypothalamus do seem to respond to the hedonic impact of tastes [148–153] (though many of these neurons may be in 'far lateral hypothalamus' or ventral pallidum), and some lesions of the anterior far lateral hypothalamus produce active aversion as well as aphagia [82,83,154]. However, a taste–reactivity mapping study by Cromwell showed that the *aversion* after a lesion actually is caused by damage to the ventral pallidum immediately adjacent to the LH, and not to the lateral hypothalamus itself [81]. Lesions restricted to the LH do not cause aversion, even if they cause aphagia [81,154], and aversion seems to result only if a LH lesion penetrates anterolaterally into the ventral pallidum [11,81,154]. Further, Valenstein and I found that LH *stimulation* never enhances the hedonic reaction to palatable food, even if it causes rats to eat vigorously [118]. Thus, hedonic impact is neither diminished by LH damage, nor enhanced by LH stimulation. This indicates that the activity of LH neurons does not mediate hedonic impact in a causal sense, even if their electrophysiological or neurochemical activation *reflects* hedonic value, as a feedback signal or a relay to other systems.

Other 'false hedonic candidates' include *neurohormonal* systems involved in appetite. For example, Flynn and his colleagues have shown that *bombesin*, a satiety hormone, suppresses intake without suppressing the 'gustatory reinforcing' or hedonic properties of food [52]. Bombesin has been well demonstrated to suppress food and fluid intake in a variety of paradigms [155–162]. However, bombesin fails to suppress hedonic reactions to sweet tastes or to increase or aversive taste reactivity patterns [52,159].

4. Insufficiency of ingestive response, consummatory response, and 'sensory reflex' interpretations of taste reactivity

Now we return once again to the issue of interpreting affective reaction patterns, and specifically to the relative merits of alternative interpretations. Is it necessary to posit that taste reactivity patterns reflect hedonic impact or palatability? Many authors have suggested simpler interpretations of taste reactivity patterns, such as that taste reactivity patterns reflect a *brainstem reflex* or at least a *sensory reflex* of some sort, or that taste reactivity patterns reflect merely the *decision to swallow a taste* (i.e. an *acceptance*, *ingestion* or *intake* measure), or that it is simply a *consummatory behavior* in the sense of the early ethologist Wallace Craig [43]. Reflex, ingestive behavior, and consummatory behavior labels apply to some aspects of taste reactivity, and have certainly been often used in taste reactivity studies [4–6,22,44,46,73,163]. Why not therefore use them now? The answer is straightforward: because they are inadequate.

We would all agree that the *simplest adequate interpretation* is generally the best in science. But 'adequate' is every bit as important in this criterion as 'simplest'. Simple explanations that are inadequate remain forever simply inadequate. To accept them over a less simple but adequate interpretation constitutes a scientific step backward rather than forward. Such an error imposes a permanent barrier to understanding because it guarantees that future data will be misinterpreted. Thus a crucial question regarding simpler alternative interpretations of taste reactivity is whether they are *adequate to account for the known data*. I think that anyone who fully considers the data currently available will be forced to reject as inadequate these simpler alternatives, and to conclude that a hedonic/aversive interpretation of taste reactivity patterns is the only adequate interpretation and therefore the best. The reason, in brief, is that although positive hedonic affect, voluntary ingestion, and sweet sensation ordinarily cluster together, they dissociate from each other under some conditions, especially after certain brain manipulations. Thus it is quite possible for an animal to ingest without showing the positive hedonic taste reactivity pattern, or to show the positive pattern without ingesting, or to reject without showing the aversive pattern. This deserves to be considered in more detail.

4.1. Inadequacy of 'ingestive', 'intake', 'acceptance' or 'swallowing' labels

The taste reactivity paradigm was originally invented by Grill and Norgren as a means of studying the remaining homeostatic intake competence of the brains of animals who would not voluntarily eat [22,23]. The focus on ingestive competence led to the reaction patterns being considered 'ingestive' [163–165]. Wise has suggested that this remains the best interpretation, arguing that "the taste reactivity paradigm simply measures the consummatory responses of ingestion or rejection and adds little to what we can infer from other consummatory measures as to the hedonic impact of rewarding stimuli" [166; p. 252]. But despite its reasonable beginning, it is now clear that taste reactivity patterns cannot be equated with ingestion. The reason is that *intake/ingestion measures and taste reactivity patterns can be pulled completely apart* from each other by a host of neural and pharmacological manipulations. That must mean that acceptance or ingestion of a food is different from the reaction measured by taste reactivity patterns.

4.1.1. Ingestion suppression without aversion

Consider first the empirical and conceptual difference between aversion (active gapes, etc. reflecting unpleasantness or dislike) versus mere rejection of a food (either refusal to eat or passive dropping of a food from the mouth). Although most manipulations that cause aversion also cause food to be rejected, many satiation manipulations that cause rejection of a food do not cause aversion. For examples, Cabanac and LaFrance showed that caloric satiation diminishes hedonic reactions of rats to sucrose [13,68]. They did not specifically examine whether satiation also changed active aversive reactions (gapes, etc.; although it did cause food *rejection* through passive dripping of the solution), but in a similar study I replicated their satiation-induced hedonic suppression, and found satiation was *not* accompanied by increased aversive reactions [67]. Adding special forms of satiation such as *sensory-specific satiety* [167–169] to caloric satiation still did not induce actively aversive reactions such as gapes or head-shakes [67]. Even adding an extreme physiological '*super-satiation*' failed to cause active aversion to the taste of food [67]. Super-satiation was produced by trickling a palatable milk or sucrose solution into a rat's mouth until it had consumed 10% of its body weight in food within a 30 min period (this would be equivalent ounce-for-ounce to a human who ate a 10–20 lb meal at a single sitting!), yet even that degree of satiation merely suppressed hedonic reactions and did not cause active aversion [67]. Other examples of a rejection/aversion difference come from certain forms of associative learning. Pelchat et al. [54] found that rats quickly learned to avoid a particular taste and to freeze their consumption if they encountered it, after experiencing pairings between the taste and the administration of an electric shock to their feet. But shock-paired tastes never elicited aversive reaction patterns, in contrast to rats who had had the taste paired with LiCl, who did show aversion [54]. Similarly, Parker and her colleagues have shown, in an important series of taste reactivity studies on the difference between food avoidance and aversion, that although most drugs produce conditioned taste avoidance when paired associatively with food (even rewarding drugs), actual aversion accompanies the rejection only in the case of drugs that lack rewarding properties [170–172].

4.1.2. Food avoidance without hedonic suppression

Beyond the group of manipulations that fail to produce aversion are those that also fail to selectively *suppress hedonic* reactions when they suppress intake. Several brain manipulations and pharmacological manipulations that suppress intake have this additional dissociation from taste reactivity patterns As noted above, these include dopamine-depleting 6-OHDA lesions, central amygdala lesions, and administration of bombesin. All of these produce taste avoidance, at least in some situations, but none of them produce aversion, nor do they even suppress hedonic reaction patterns to the taste of the food or salt that is refused [12,52,61,119,144].

4.1.3. Intra-oral intake suppression without hedonic suppression

A final type of dissociation between intake and taste reactivity patterns can be seen in the case of manipulations that not only suppress *voluntary food consumption*, but also suppress even '*intra-oral intake*', that is, passive swallowing of a sucrose solution that is infused into the mouth through an intra-oral cannula. This measure of intake was part of the original Grill and Norgren paradigm, and continues to be a useful ingestion measure for many studies [173–175]. But even though intra-oral intake is elicited by the same oral stimulus as taste reactivity, the two responses sometimes dissociate.[4] For example, as already mentioned, central amygdala lesions block salt-appetite induced increases in intra-oral intake, as well as reducing voluntary salt consumption. Yet hedonic reactivity patterns still increase in lesioned animals during salt appetite, even though intra-oral intake does not [61,133]. An opposite dissociation is produced after bombesin administration: intra-oral intake is suppressed by bombesin (just as voluntary ingestion is suppressed), but hedonic reactions remain unsuppressed [52]. Clearly, if intra-oral intake can change without changing hedonic reaction patterns, and if hedonic

[4] A potential caveat regarding the dissociation of taste reactivity patterns from actual ingestion was pointed out to me by Dr Alan Spector (personal communication, 1998). The dissociated affective reaction and intake measures should both be tested under exactly the same conditions (i.e. at the beginning of a meal, at the end of a meal, etc.) in order to show that the dissociation is truly due to a difference between the experimental conditions under which they are employed. That has sometimes been done, but perhaps not always. It would be desirable for future studies of dissociation to be conducted with this point in mind.

reaction patterns can change without changing intra-oral intake, then intake and taste reactivity cannot be the same thing. Only one of these can be 'ingestion'. It makes sense that 'ingestive' as a label be reserved for behavior related to the actual *amount ingested* (i.e. measures of intra-oral intake and voluntary intake) rather than be assigned to taste reactivity patterns that can separate from the amount consumed. Since hedonic reaction patterns can change independently from changes in the amount ingested (measured either by voluntary ingestion or by intra-oral ingestive), and since ingestion can change independently of hedonic reaction patterns, it can therefore be misleading to call taste reactivity 'ingestive'; 'hedonic' is a better label for characterizing the positive taste evaluation it reflects.

4.2. Taste reactivity is not just consummatory behavior

The idea of consummatory behavior (applied to feeding) is often considered to be identical to the idea of ingestion (amount consumed) as in the quote from Wise above [166]. However, they are not necessarily the same. 'Consummatory behavior' is a term originally introduced by the early ethologist, Wallace Craig, in 1918 to describe the terminal phase of motivated behavior [43], and distinguish it from preceding appetitive phases. Is 'consummatory behavior' an adequate label to apply to taste reactivity patterns—even if consumption is not? Taste reactivity patterns have been called 'consummatory' by a number of authors (including me) [44,46,144]. However, there are serious difficulties with the assertion, 'taste reactivity is simply consummatory behavior'. Unlike 'ingestion', 'consummatory' as a label is not so much occasionally wrong when applied to taste reactivity, as it is unacceptably vague.

In Craig's original sense, the terms 'appetitive' and 'consummatory' refer to *temporal* phases of motivated behavior: *appetitive* behavior occurs *prior* to capture of the goal object, whereas *consummatory* behavior occurs *after* it [43]. Taken merely as a temporal label, consummatory can be applied legitimately to taste reactivity patterns, since the reactions occur after food is obtained. But 'consummatory behavior' includes too many different types of behavior to be changed coherently by a single brain manipulation, and thus it is too vague to use as an explanatory category of brain-behavior relations.

Regarding eating, there are at least three types of consummatory behavior, which usually occur at approximately the same time but which are quite distinct. First, consummatory ingestive behavior in Craig's original sense included the actual *licks and bites of voluntary eating* [43]. Second, the *hedonic/aversive taste reactivity patterns* studied here occur during the consummatory phase [4,5,21,22,44,46,164,176,177]. Third, the act of *swallowing a substance already in the mouth*, as opposed to spitting or spilling it out, is a form of consummatory behavior. In many cases, these various senses of 'consummatory' have been taken by their authors explicitly or implicitly to be interchangeable [3–5,21,44,46,164,165,178]. But they often dissociate in different directions and therefore cannot be truly interchangeable or the same. A few examples will suffice:[5]

The case of amygdala lesion-induced impairment of salt appetite discussed above provides one example of a dissociation among different 'consummatory' behaviors. Amygdala lesions disrupted the first and third senses of 'consummatory' (voluntary licking of salt and intra-oral intake) but not the second sense (affective taste reactivity) [61,144]. In the same way, caloric satiety produces rejection of food in the first sense (voluntary intake) and third sense (passive intra-oral intake) of consummatory behavior, as described above, but not in the second sense of aversive affective reaction patterns.

Likewise, the case of bombesin discussed above provides another example. Flynn and colleagues have reported that bombesin suppresses the first sense of consummatory behavior (voluntary licking for sucrose or salt solutions) and also suppresses the third sense (passive intra-oral intake, even in decerebrate rats) [157–162], but fails to suppress the second sense of consummatory behavior (hedonic or aversive taste reactivity patterns) [52,159]. Flynn concludes that these satiety peptides "inhibit intake without affecting the gustatory reinforcing properties of the food" [52; p. 113], an interpretation similar to one of 'liking' versus 'wanting'.

Even intra-oral intake (the third sense of consummatory behavior) can be detached from voluntary intake (the first sense) and from affective taste reactivity (the second sense). The intra-ventricular administration of neuropeptide Y promotes voluntary food intake, but it does not enhance the amount of a nutritive solution that rats swallow when they are fed directly by intra-oral infusion [175]. Neuropeptide Y also fails to produce strong shifts in hedonic/aversive patterns of taste reactivity to caloric solutions ([175]; Peciña and Berridge, personal observations).

Disruption of dopamine neurotransmission also dissociates the different types of consummatory behavior, as described above, indicating that some but not others are mediated by dopamine systems. Extensive 6-OHDA lesions that produce aphagia eliminate ordinary consummatory behavior, even if food is placed immediately in front of a rat [179], and dopamine antagonists suppress voluntary eating [180–183]. Yet the affective pattern of taste reactivity is not shifted by dopamine antagonists (though they produce sensorimotor suppression), nor are hedonic reactions changed by 6-OHDA lesions, even if accumbens and neostriatal dopamine is depleted by 99% [12,28,114,119]. Neither is intra-oral intake suppressed by dopamine antagonists or 6-OHDA lesions [177,184].

Thus some types of consummatory behavior are immune

[5] For other examples of dissociation among types of consummatory behavior (e.g. different effects of dopamine antagonists on consummatory *pattern* versus on *amount* of intake), see Refs. [123,182,227–230].

to these manipulations, but other types are not. This can only mean that there are really different types of consummatory behavior, which have separable mechanisms, and these types of consummatory behavior differ in their susceptibility to a particular manipulations. In summary, the term 'consummatory behavior' actually refers to a number of different types of behavior that follow contact with a goal. It is not a coherent single category in which to classify a response. The term cannot be used, therefore, either as a good *description* of a particular behavior's mechanism, or as an *explanation* for why it is or is not changed by a particular brain manipulation.

4.3. A taste reactivity pattern is not a mere reflex

Is a taste reactivity pattern a mere brainstem reflex? Or is it at least a sensory reflex of some sort, even if mediated by more than the brainstem?

Both for human infants and for rats, the basic triggering mechanism and motor components of taste reactivity are generated by the brainstem, since anencephalic infants and decerebrate rats both show the appropriate reaction to sucrose or quinine [9,23]. But the forebrain exerts hierarchical control over the pattern of taste reactivity under ordinary conditions [11,21]. Neural manipulations restricted to the forebrain produce dramatic changes in affective patterns of taste reactivity.

For example, hedonic reaction patterns of rats are enhanced by microinjection of an opioid agonist into the forebrain ventricles or directly into the shell of the nucleus accumbens [27,87,88]. Conversely, aversive reactions are enhanced by ablation of the telencephalon [23], or by focal destruction of neurons in the ventral pallidum or far lateral hypothalamus [80–83,154]. Many psychological manipulations of palatability (e.g. associative learning of taste-aversions or taste-preferences) are also mediated principally by forebrain neural systems, and therefore also control hedonic taste reactivity patterns via hierarchical control [185–187]. Thus the forebrain clearly controls the pattern of hedonic/aversive reactions.

4.3.1. Evidence against higher-brain reflex

It would still possible for taste-reactivity to be a reflex to sensation, even if mediated partly by the forebrain, if the reaction pattern were triggered in a rigid S–R fashion by sensory stimuli. But the response pattern to the same stimulus can vary so dramatically that the concept of reflex becomes either demonstrably false (if reflex is held to involve a rigid S–R relationship) or else meaningless (if it is not). One can flip the affective response to the taste of concentrated NaCl, for example, back and forth repeatedly between aversive and hedonic simply by bringing the rat in and out of salt appetite state induced by a physiological sodium imbalance [57,60,63]. Similarly, the hedonic response to sweet can be reversed to aversion by associative aversion learning [6,46,54,73,74,188]. Or conversely, aversive responses to a taste can be shifted toward hedonic by various forms of preference learning [75,76,79], or by interactive combinations of associative learning and physiological need states (involving alcohol withdrawal or sodium deficiency [53,58,77,189,190]).

4.3.2. Conditioned sensory reflex?

In an attempt to account for changes in taste reactivity patterns induced by conditioning or physiological state shifts, yet preserve a pure (S–R) sensory reflex interpretation, Nader, Bechara and van der Kooy [191] have suggested that the neural representation of the *sensory stimulus* itself is changed by these manipulations. Nader et al. [191] suggest that the "taste reactivity paradigm is a better measure of the food's subjective sensory, as opposed to hedonic, properties" [191; p. 100]. By their interpretation, taste aversion learning, for example, produces essentially a kind of conditioned *sensory illusion* of aversive stimuli. After pairing with LiCl, they propose, that the taste of a saccharin CS "will elicit conditioned aversive (sensory) effects that will decrease consumption, as well as other conditioned responses such as the oro-facial behaviors that taste reactivity measures" [191; pp. 101–102]. In other words, the sweet conditioned stimulus elicits a bitter or otherwise unpleasant sensory illusion, which in turn elicits the behavioral reaction. Conversely, a *conditioned hedonic enhancement* could be explained by this 'sensory reflex' interpretation as a sensory illusion of sweetness, triggered by the conditioned stimulus that was previously paired with a sweet UCS. Conditioned illusions cannot be invoked to explain direct changes in taste reactivity patterns produced either by physiological state (e.g. hedonic enhancement by hunger or sodium appetite) or by pharmacological or neural manipulations (e.g. hedonic enhancement by intra-cranial morphine or benzodiazepines). But presumably Nader and colleagues might argue that these physiological manipulations themselves directly alter 'discriminative sensory properties' via a purely sensory alliesthesia (change in taste sensation induced by a physiological state), so that hunger, salt appetite, morphine, etc. might thus make a food taste sweeter.

However, there is little reason to believe that such sensory illusions occur powerfully enough to account for the behavioral shifts. Certainly, there is no evidence at all from human subjective reports of the effects of conditioning, hunger, etc. Humans who have developed a conditioned taste aversion for a sweet food or drink do not report that the food subsequently has a bitter taste after conditioning. The food tastes as sweet as it did before—but now they perceive it as unpleasant [71,192]. That is, they report a hedonic shift for the same sensation, and not a different sensation. Similarly, regarding hunger, humans report enhanced hedonic ratings but no increase in sweetness intensity during hunger [66,193]. Unless humans lack a sensory shift during hunger

or aversion learning that rats possess, these manipulations seem more likely to alter hedonic, rather than sensory, properties of food perception.

A faint glimmer of support for the 'sensory illusion' hypothesis comes from electrophysiological studies of rats and monkeys by Scott and colleagues and by Rolls and colleagues, which indicate that there may be changes in the *neural representation of gustatory signals* at various levels of the brain under hunger and satiety states [194–200]. It could be construed from such studies that NaCl tastes sweet after sodium depletion, or that a sugar tastes bitter after associative pairing with LiCl. However, such changes are probably better interpreted as changes in a taste's *palatability* rather than as changes its *sensory* features [11]. In the one explicit account of gustatory recoding that comes close to the sensory illusion hypothesis, McCaughey and Scott have suggested that the neural representation of salty tastes in the rat hindbrain nucleus of the solitary tract becomes more 'sugar-like' during states of sodium depletion [201]. But even McCaughey and Scott caution that "it is not suggested that NaCl assumes all the qualities of sugar in deprived rats—tasting sweet and releasing insulin, for example" [201; p. 673]. Thus a 'sweet illusion' could not be the cause of the deprivation change in behavioral reaction. Changes in NaCl gustatory coding in the nucleus of the solitary tract may well reflect early stages of the enhanced *hedonic* value of a taste that remains salty during sodium appetite, rather than a sensory shift to illusory sweetness.

In any case, the ability of rats to *recognize* particular tastes as the same before and after appetite shifts, preference or aversion conditioning, etc. indicates that the sensory features of the taste must remain relatively constant. For example, in taste aversion conditioning, rats can easily learn to recognize and distinguish between two very similar sugars, maltose and fructose, which they should not be able to do if aversion conditioning disrupted normal sensory coding [46]. Nader and colleagues posit that neural coding of the sugar sensation in this case is sweet during the first moments of encounter with a conditioned taste, to allow recognition, but then changes to a different sensation of bitter or nausea to produce aversion [191]. Yet there is no actual evidence of such temporal sequences of neural coding or of other evidence that would indicate 'delayed illusions' of this type.

The 'sensory illusion' explanation becomes even more strained when applied to salt appetite or to pharmacological enhancements of taste reactivity patterns. Exactly what happens to the taste sensory signal during sodium appetite is unclear, as the electrophysiological evidence has been mixed. Sodium depletion has been reported to decrease gustatory signals evoked by NaCl in the peripheral chorda tympani nerve, but to produce mixed sensory changes in the brain {Shimura, 1997 #2582; Tamura, 1997 #2583; Scalera, 1997 #2026; Contreras, 1979 #2585 [202]}. An electrophysiological study by Jacobs, Mark and Scott reported in 1988 that responsiveness to salt by gustatory neurons in the hindbrain nucleus of the solitary tract did indeed diminish when rats were physiologically depleted of sodium, and that there was a shift in coding from 'salt-best' neurons to 'sweet-best' neurons [203]. However, a more recent 1997 study by Tamura and Norgren reported that the amplitude of NaCl gustatory signals in the nucleus of the solitary tract was *increased* during sodium deficiency [204]. Similarly Beauchamp and colleagues found that humans experience the taste of salt either as unchanged or as *more* intense [69].

If central gustatory neurons become more responsive to the taste of salt, it would be impossible for a purely sensory change to explain why individuals drink more of highly salty solutions that are normally too salty and unpalatable. The too salty taste would become saltier still. Thus a sensory enhancement cannot explain why highly salty tastes elicit enhanced hedonic taste reactivity patterns from sodium depleted rats [57,59,62,63] or why very salty foods receive enhanced hedonic ratings from sodium depleted humans [69]. For an enhancement of the palatability of concentrated salt to be due to sensory changes, the taste signal would have to be massively weakened within the brain, so as to dilute the sensory qualities of the solution that is ordinarily too salty. Finally, even if intake of highly salty foods or solutions were increased via a reduction in sensory intensity, reducing its aversiveness, then the sensory intensity of threshold salt concentrations should also be reduced, and the dilute salt solutions should not be preferred to water. But in fact the intake of all concentrations of NaCl, high and low, has long been known to be increased by sodium deficiency: the intake-concentration curve does not simply shift, it rises altogether [205,206]. It is very difficult indeed to account for salt appetite in terms of a sensory change in gustation, and therefore difficult to account for changes in affective taste reactivity patterns in terms of a sensory change.

Pharmacologically induced changes in taste reactivity, such as by morphine, likewise favor a hedonic explanation rather than a sensory explanation, since the available evidence indicates that opioid manipulations do *not* change the sensory discriminative properties of taste. For example, naloxone does not alter human subjective ratings of sensory sweetness intensity or of other sensory qualities of food [207], nor does it alter the ability of rats to perform a sensory discrimination task that requires them to recognize the taste of sucrose [208]. Yet opioid agonists and antagonists do alter hedonic subjective ratings in humans, and they also alter hedonic/aversive taste reactivity patterns and other measures of palatability in rats [15,84,86,87,91,97–100,207,209]. In conclusion, evidence fails to support the sensory illusion hypothesis that pharmacological states, appetite states or conditioned food preferences/aversions cause massive shifts in a taste's sensory properties.

4.3.3. Summary: hedonic label is more accurate than acceptance/ingestion, consummatory behavior, and reflex labels

I apologize if I have belabored the dissociations among hedonic/ingestive/acceptance/consummatory/reflex categories beyond the patience of the reader. But these dissociations are crucial to the legitimacy of taste reactivity as a measure of hedonic impact, since it is a hedonic measure only if the simpler alternative interpretations can be rejected. The tendency to adopt these simpler yet inadequate alternative labels has been so strong in behavioral neuroscience studies in the past that I feel the evidence against them cannot be too strongly emphasized.

It seems reasonable to conclude that if one seeks an adequate interpretation of the nature of taste reactivity, one must *reject* the suggestion that the reactions are simply a manifestation of acceptance or ingestion of a food, and reject the 'consummatory' behavior label, as well as the 'sensory reflex' label, and even 'conditioned sensory reflex' label for taste reactivity. The first three labels are fine as loose descriptors, but not as definitions of the nature of taste reactivity patterns, and the fourth label is simply mistaken. The demonstrations of dissociation described above show that the relation of hedonic taste reactivity patterns to ingestion, consummatory behavior, and to the sensation of sweetness is merely correlative and is not definitive. The definition that best fits the available evidence is that hedonic and aversive reaction patterns reflect the *affective* properties of the stimulus: its palatability, or whether the individual 'likes' it.

5. Methodological issues

5.1. Voluntary eating versus intra-oral infusion

Can affective neuroscience studies of taste reactivity patterns be done equally well if an animal or infant eats/drinks voluntarily or is it better for the experimenter to control stimulus delivery? The original paradigm of Steiner delivered the taste stimulus directly to a human infant's mouth via dropper. The same mode of passive delivery has been used in most subsequent studies of human infant taste reactivity [19,29,33,35,210]. Similarly, Grill and Norgren originally used intra-oral cannula to directly deliver a taste-containing solution to the mouth of an animal, and most subsequent affective neuroscience studies have continued to use the intra-oral delivery technique. Grill and Norgren's original reason for cannulae delivery was that they wished to study the residual competence of decerebrate animals, which would never voluntarily eat [22,23,73]. That motive applies also to a number of other brain manipulations that abolish or reduce voluntary intake, such as 6-OHDA lesions of the mesolimbic system, electrolytic or excitotoxic lesions of the lateral hypothalamus, or administration of dopamine antagonist drugs, bombesin, etc. [3,12,28,52,81,119,127,129,157,211]. In such cases, one often wants to know whether reduced hedonic impact is the cause of reduced voluntary ingestion. If voluntary ingestion is absent or reduced, then it means that the *stimulus magnitude* that elicits taste reactivity would also be reduced in any test based on voluntary intake. Intra-oral delivery provides a way to guarantee that the animal receives a full stimulus magnitude so as to allow meaningful analysis of whether the response magnitude is normal.

Intra-oral delivery is also useful for examining manipulations that *increase* intake, for essentially the same reason. Namely, it ensures that any observed increase in hedonic reaction patterns is due to increased hedonic impact and not just to the fact that the animal has voluntarily consumed more of the eliciting stimulus. Further, Grill, Roitman, and Kaplan have shown intra-oral delivery can be used to detect very slight changes in taste reactivity (such as hedonic alliesthesia produced by only 24 h of food deprivation), by examining responses immediately *after* the stimulus, even though such tiny shifts are obscured by response demands when the stimulus is present [2]. Thus for many affective neuroscience studies, delivery of the stimulus through oral cannula offers a way to determine whether changes in hedonic impact are the cause of changes in intake.

On the other hand, several taste reactivity studies of animals have successfully used voluntary free-feeding paradigms [54,109,117]. Steiner and colleagues have allowed adult gorillas, chimpanzees, orangutans, and other primates to voluntarily sample as they chose from beakers or delivery tubes containing solutions of sucrose, quinine, or other taste stimuli. Pelchat et al. [54] showed that rats emitted aversive reaction patterns when they voluntarily sampled a taste that had been associatively paired with LiCl, but not when they sampled a taste that had been paired with electric shock. Fuerté et al. [156] showed that declines in intake of a diet deficient in an essential amino acid was accompanied by development of an aversion to its taste, in rats that were allowed to sample it freely. Gray and Cooper [117] showed that D-fenfluramine administration reduced hedonic reactions during voluntary consumption of sucrose, but did not change aversive reactions during the (very brief) period that rats voluntarily sampled quinine. Conversely, Gray and Cooper [109] found that a benzodiazepine enhanced hedonic reactions during voluntary consumption of a sucrose solutions. Of course it is important in such cases to consider separately effects on intake versus effects on affective taste reactivity patterns, but this can perhaps be achieved by calculating the observed magnitude of taste reactivity as a function of the amount consumed (i.e. the stimulus size).

In general, it can probably be concluded that intra-oral delivery is the best technique in cases where the relation between hedonic impact and intake amount is important (i.e. the relation between 'liking' and 'wanting', especially in affective neuroscience studies). But if the goal is simply to know whether or not a manipulation has any effect on

affective reaction patterns, then the use of voluntary intake designs can be a useful first step. Finally, voluntary intake designs may be necessary in order to study affective reactions to *solid* food (perhaps controlling in other ways for stimulus size), since intra-oral cannulae delivery is restricted to liquid taste stimuli.

5.2. Hedonic versus aversive categories of taste reactivity components

The defining feature of affective taste reactivity components is that they are organized into hedonic and aversive categories that reflect positive and negative palatability [21,22]. Individual components are not elicited randomly or as single motor elements. Instead, a stimulus elicits a *hedonic or aversive pattern* of taste reactivity components (or both patterns on rare occasions [212]). There are essentially three criteria that can be used to identify a particular component as belonging to the hedonic or the aversive category. These affective assignment criteria could be called (1) *shared stimulus*, (2) *temporal association*, and (3) *shared outcome*.

5.2.1. Shared stimulus criterion

Sucrose typically elicits rhythmic tongue protrusions from both human infants and rats, also paw licking and lateral tongue protrusions from rats, and facial relaxation and retraction of the lips, together with an occasional eye crinkle and smile, from human infants [9,19,22,35]. Quinine typically elicits gapes, head shakes, and limb flails from both human infants and rats, and also face wipes and occasional paw treads and chin rubs from rats, and nose wrinkling and eye squinching from human infants [9,19,22,35]. All the members of the hedonic category share their eliciting stimulus in common, as do all members of the aversive category. Stronger concentrations of either sucrose or quinine elicit members of the same category as moderate concentrations, just in greater numbers. Thus a defining feature of hedonic and aversive patterns is that all members of a category share their prototypical stimuli.

5.2.2. Temporal association criterion

The members of the hedonic category tend to be elicited in close temporal proximity to one another, and to follow one another in sequence [21,212]. This is necessarily true for prototypical hedonic or aversive stimuli, but it is also true to a large extent even for tastes of mixed palatability (NaCl, water, KCl, saccharin, complex flavors). When both hedonic and aversive reactions are emitted to the same taste, the members of the hedonic category will more often occur in immediate succession to another hedonic member, than to a member from the aversive category, and vice versa [21,212]. It is a relatively rare occurrence to alternate back and forth directly between categories [21,212]. More often, in cases of mixed palatability, there will be a series of hedonic reactions, followed by a series of aversive reactions, and so on. Such temporal clustering among the members of a category suggests that they originate together from activation of a hedonic affective state or aversive affective state.

5.2.3. Shared outcome criterion

All members of a category tend to change together when the palatability of a taste is altered by changing its concentration, or by changing a physiological state relevant to the taste, or by creating a learned preference or aversion based on associative experience, or by introducing a pharmacological manipulation or neural manipulation. The members of the other category typically do not change, or if they do, they change in the opposite direction. As sucrose concentration is increased, for example, the number of hedonic reactions all grow together, and aversive reactions diminish. As quinine concentration is increased, the number of aversive reactions all grow together and hedonic reactions diminish. As caloric hunger or sodium depletion is induced, the members of the hedonic category elicited by sweet or salty tastes grow together. Simultaneously, after sodium depletion, the members of the aversive category elicited by a strongly salty taste all diminish. Administration of morphine or benzodiazepines also increases the frequency of hedonic category members, while decreasing the frequency of the aversive category members. On the other hand, production of a learned aversion by pairing of sucrose with LiCl-induced illness, reduces all members of the hedonic category, while increasing all members of the aversive category [6,26,46,54,72,73,171]. Likewise, a ventral pallidal lesion and telencephalon ablation both eliminate the members of the hedonic category, and increase the members of the aversive category. Thus a defining feature of members of an affective category is that they all tend to change in the same direction after a manipulation that shifts palatability, and differently from the members of the opposite affective category.

5.3. Obtaining hedonic and aversive categories from individual behavioral components

As the first step in any taste reactivity analysis, it is crucial to score each microcomponent separately (e.g. rhythmic tongue protrusions, gapes, etc.). Once this has been done, components can be grouped into affective categories if desired. For scoring rat taste reactivity patterns, the strongly hedonic category includes lateral tongue protrusions, rhythmic tongue protrusions, and paw licking (outside of paw licking during grooming sequences) [21,48]. The strongly aversive category includes gapes, chin rubs, head-shakes, face washing, and forelimb flails (again not including the latter three components when they occur in normal grooming sequences). Human and primate microcomponent clusters are somewhat different. For example, the hedonic components of human infants also include lip smacks, smile-like elevation of the mouth

corners, and finger sucking; and their aversive components include frown-like depression of the mouth corners, nose wrinkling, and scrinching of the eyes and brows [9,16,29,35,42].

The chief reason for grouping components into larger affective categories is to simplify data presentation, and so make it easier for a reader to see hedonic and aversive patterns. These are important benefits for affective neuroscience studies, but it is always important to remain aware of individual components, and of how they change relative to each other. That is because a sensorimotor change in microcomponent emission can sometimes masquerade as an affective shift. For example, if a manipulation produces a sensorimotor decrement of *all* types of responses together (hedonic, aversive, and other behavioral categories), but only the hedonic category of response is counted (or only the aversive category), then one may be misled into believing that a hedonic or aversive shift has occurred [28,127,129]. It is important to compare changes in one affective category against the other category. The signature of a *true affective shift*, in almost all cases, is that a similar change applies to most components within an affective category, but not to components in the other affective category (which remain unchanged or change in the opposite direction), and not to components that are neutral.

5.4. Balancing hedonic and aversive components

When grouping into hedonic and aversive categories, it is useful to adopt a scoring system that allows all components within a particular affective category to make *equivalent contributions* to their total hedonic score or aversive score. If raw counts of different components are simply added together, an imbalance is likely to result. Some individual components occur very frequently (e.g. a rat may emit hundreds of rhythmic tongue protrusions in a minute) while others are rare (e.g. the number of lateral tongue protrusions will rarely exceed several dozen in the same minute). If every occurrence of each microcomponent were counted towards the affective score, then the count for frequent components would swamp those of rare components. But a change in a rare affective component is often at least as informative as a change in a more frequent one. In order to avoid this problem, and to balance the contribution across all components, a 'time bin' scoring system can be applied to frequent components [47]. We have found that a balanced representation of all components can be obtained for rats using a scoring system in which paw licks and rhythmic mouth movements are scored in 5 s bins (up to 5 s of continuous paw licking scored as a single occurrence; if a pause interrupts, the clock is reset), and rhythmic tongue protrusions scored in 2 s bins [47]. For newborn human infants, similarly, we have found that a balance can be achieved by using 2 s bins to score hedonic rhythmic tongue protrusions, lip smacks, and complex mouth movements, and aversive eye squinching and nose wrinkling [35]. All other human and rat components are scored each time they occur.

5.5. Neutral third category of taste reactivity components

Some components are elicited by tastes, yet are emitted in ways that seem not strongly tied to either hedonic or aversive affective categories. For human infants, these include many facial movements that are not linked to affective taste reactivity categories [16]. For rats, these components may include passive dripping of the infused solution, locomotion (walking and rearing), and rhythmic mouth movements. Passive dripping often occurs in an aversive context, but it can also occur simply because the animal fails to respond with any affective reaction. Locomotion similarly appears in aversive contexts, but it also can occur as part of exploration, and hence is not a reliable indicator of aversion specifically. Mouth movements, by contrast, often accompany hedonic reactions, but are almost a necessary correlate of ingestion (unlike the stronger hedonic components), and so are a default response to a taste that is consumed. The score for mouth movements actually *decreases* once the hedonic palatability of a taste grows past a certain point, because the frequent occurrence of other hedonic reactions subtracts from the amount of time available for mouth movements.

The term 'neutral' seems useful to apply to such actions, though it does not mean that these components never carry any affective tone at all. Sometimes, after all, mouth movements may accompany hedonic evaluations or passive drip or locomotion may accompany aversion. Neutral merely means that scores of these components are not reliable indicators of a strong affective response, because they are too often under the control of some factor other than affect. Therefore it is best to consider them separately from hedonic and aversive categories of components.

Even components that are ordinarily strongly hedonic or strongly aversive can sometimes occur in a 'neutral' context—such as ordinary grooming movements by rats. These include rhythmic tongue protrusions and paw licks (otherwise hedonic), on the one hand, and head-shakes, forelimb flails, and face wipes on the other (otherwise aversive). But those components are not hedonic or aversive during self-grooming: they are then simply grooming movements. The key feature in such cases is the *sequential pattern* of the components, which helps identify whether the context is affective or neutral. In a hedonic context, for example, paw licks, rhythmic tongue protrusions, and lateral tongue protrusions tend to occur in close temporal proximity to each other, and not contiguous to most aversive components. In aversive contexts, head-shakes, face wipes, and forelimb flails tend to occur in close contiguity, and are not typically emitted next to hedonic components. But when rats groom themselves spontaneously, by contrast, face wipes by the paws often alternate with paw licks and tongue protrusions. In this case, the sequence is self-grooming, and

therefore neutral at least as regards the taste stimulus, neither strongly hedonic nor strongly aversive. An oral infusion of water will often trigger grooming sequences, as though the rat treats the water as saliva and uses it to wash its body. Dilute taste solutions of other stimuli will sometimes elicit the same grooming response. Sequential organization is the easiest way to classify whether the behavior is affective or is occurring as part of a grooming sequence. In our lab, we classify a face wipe or forelimb flail as belonging to neutral grooming if either occurs within two seconds of a tongue protrusion or paw lick. If more than two seconds elapses between emission of the 'hedonic' and 'aversive' neighbors, then the pattern is scored as affective.

This sequential criterion for distinguishing hedonic/aversive patterns from grooming is useful but it has one weakness. It fails to register true alternations between hedonic and aversive reactions, which also occur under some conditions [21,212]. But true alternation between hedonic and aversive patterns is relatively rare. Losing the registration of those rare events is the price of having an easy scoring criterion. In cases where one desires to distinguish true hedonic/aversive alternation from nonaffective grooming, it is always possible to devise more extensive criteria to identify neutral sequences. Our experience has shown that when our sequential 'neutral' criterion is triggered by an alternation of paw lick and face wipe, for example, that alternation is usually embedded in a larger grooming sequence (in which the rat goes on to groom its body, for example). These larger sequential criteria are more cumbersome to apply, but can be exploited if needed to ensure a higher degree of accuracy in making hedonic/neutral/aversive classifications. In the meantime, the sequential criterion given above is an easy and effective way to separate taste-elicited hedonic or aversive sequences from 'neutral' sequences of ordinary grooming.

5.6. Scoring in real-time versus slow motion

The flow of taste reactivity components is simply too fast for an observer to gain more than a crude sense of whether the pattern is dominated by hedonic or aversive actions. If that is all that is required, and if quantitative precision is not crucial, then a real-time analysis (i.e. not a slow-motion analysis) can suffice.

But for confidence in the precision of quantitative scoring, a slow motion video-analysis is essential. We have used a slow-motion analysis in which the speed can be varied between approximately 1/3 speed and a frame-by-frame (1/30 speed), depending on the momentary density of components. The ability to repeat selected portions of videotape is also important to resolve cases of ambiguity. This allows one to score all components accurately (for example, all instances of lateral tongue protrusions, which are often very brief) and to resolve difficult classifications (for example, to distinguish between low amplitude gapes versus large mouth movements; gapes have a distinctive retraction of the lip corners that creates a triangular mouth opening, and are less rhythmic than mouth movements). Computer-assisted video-scoring systems are now available that increase the ease of slow-motion video-scoring, although it remains possible to do a good job with simply a high quality variable-speed video-player and plenty of patience.

6. Conceptual issue: hedonic core process, but not subjective pleasure

When using taste reactivity as a measure of 'liking' or hedonic impact it is important to be clear about a potential confusion. Use of terms such as 'like' and 'dislike' does *not* necessarily imply that taste reactivity patterns reflect a *subjective experience of pleasure* produced by a food. Instead, behavioral affective reactions reflect *hedonic and aversive core processes*. These core processes are evaluations performed by the brain of the stimulus' affective impact, and reflected in the resulting stream of behavior. Hedonic core processes may ordinarily underlie subjective pleasure (when subjective pleasure exists, through further processing by additional brain systems) but *hedonic/aversive core processes are not identical to subjective pleasure* [131,213].

Even though I stressed earlier that affective reaction patterns (and the core evaluation of hedonic impact that they reflect) are typically correlated with subjective reports of pleasure, at least in cases when it is possible to compare them, the two are still distinguishable. A taste reactivity pattern remains merely a behavioral *affective reaction* [1,155], and not a subjective psychological experience. In order to produce the affective reaction, it is only necessary that the brain have made a core hedonic evaluation (or aversive evaluation) of the stimulus. This is precisely why my colleagues and I place quotation marks around 'liking' when we refer to the core process reflected by an affective reaction—to acknowledge its difference from the ordinary meaning of the word as conscious, subjective, unmodified liking [11,12,28,131,132].

Ordinarily in normal brains these core hedonic evaluations may be elaborated into the conscious experience of pleasure familiar to us all (probably by higher-level neural systems). But there is evidence that sometimes core hedonic evaluations can be made in the *absence* of subjective pleasure experience [213–216], and there is also evidence that conscious subjective experience is sometimes mistaken about underlying hedonic evaluations [217–221]. Finally, in affective neuroscience there are cases in which a normal subjective experience of pleasure almost certainly does not accompany the core hedonic evaluation displayed in behavioral affective reaction. For example, in the case of affective reaction patterns emitted by decerebrate animals or anencephalic human infants, the hedonic or aversive reaction is normal to at least some stimuli (though it may not be

capable of hierarchical modulation), showing that affective brain systems are highly distributed [9,21,23]. Yet it is doubtful that a normal subjective experience of pleasure or displeasure could exist in the absence of the forebrain. Thus 'liking' may sometimes occur without conscious or subjective liking.

The important point for affective neuroscience studies of taste reactivity is that hedonic and aversive patterns of affective reactions reflect a brain's underlying core evaluation of 'liking' or 'disliking' for a taste *whether or not* there is a corresponding subjective experience of pleasure. An understanding of core hedonic processes, and their embodiment in neural systems, will be valuable whether one's goal is to know how the brain responds to affective events or how it constructs subjective pleasure (it is beyond the scope of this paper to say more on the relation between subjective pleasure and the core hedonic processes revealed in affective taste reactivity patterns, but I have discussed this issue in more detail elsewhere [131]).

7. Conclusions

1. Pioneering studies by Steiner [9,10,33] and by Grill and Norgren [22,23,73,185] developed taste reactivity as a valuable measuring tool. Later studies have revealed a great deal about the *affective* nature of taste reactivity patterns, and about their relation to other aspects of ingestive behavior. Subsequent studies have also demonstrated the important value of taste reactivity measures as a tool for affective neuroscience.
2. Human hedonic and aversive reactions are directly related to the affective taste reactivity patterns of other animals. Human and other primates each have their own unique constellation of reactions, but the constellations are similar in proportion to their degree of phylogenetic relatedness. This evolutionary continuum of hedonic and aversive taste reactivity patterns extends even to the rat, which opens the way for many affective neuroscience insights that would otherwise be unattainable.
3. Affective patterns of taste reactivity reflect the hedonic impact or aversive impact of a taste. In other words, they reflect a taste's palatability—whether it is 'liked'. Ordinarily, palatability is correlated both with sensory qualities of the taste (e.g. sweet versus bitter), and with the decision of whether to ingest. But many situations disrupt that correlation. Thus palatability, as reflected by affective taste reactivity measures, is separable from sensory qualities, ingestion or intake, and from other forms of ingestive consummatory behavior. In cases of separation among these processes, as well as in ordinary situations, affective taste reactivity patterns track only the hedonic or aversive response to the taste.
4. Affective neuroscience studies have successfully applied taste reactivity measures as a tool to identify brain systems that mediate the hedonic impact or aversive impact of tastes. The results of such studies are sometimes confirmatory, demonstrating that brain systems hypothesized to mediate hedonic impact ('liking') indeed do so (e.g. accumbens opioid systems). However, the results are in other cases surprising. Taste reactivity results have identified several hedonic substrates that otherwise might not have been considered to be hedonic (e.g. brainstem benzodiazepine systems). Second, taste reactivity results have showed that particular neural systems previously *believed* to mediate food's hedonic impact do *not* do so after all (e.g. the mesolimbic dopamine system, amygdala, brainstem bombesin system). These studies have thus highlighted the existence of reward processes that are separable from hedonic impact, such as incentive salience ('wanting'). Future studies may clarify the nature of 'liking' and 'wanting' processes, reveal how their brain systems are distributed, and how the systems interact to control motivated behavior.
5. Hedonic impact in human infants may be studied using the same taste reactivity techniques. Such studies of infants reveal insights into underlying hedonic and aversive dispositions. It will never be ethical to subject human infants to deliberate manipulations such as are used to reveal hedonic brain mechanisms in animal affective neuroscience studies. But both natural variation in the stimuli and states that human infants encounter in ordinary life, and a number of special medical and neurological conditions that sometimes afflict unfortunate individual infants, could be usefully examined in taste reactivity studies. Such investigations could reveal aspects of emotional lives in infants, and tell us at least something about their underlying physiological and neural mechanisms.

Acknowledgements

I thank Prof. H. Kissileff for suggesting the idea of this paper, and Profs J. Steiner, P. Clifton, S. Thornton, and E. Valenstein for comments on an earlier version of the manuscript. This paper was written while the author was a Fulbright senior scholar at the CNRS Centre Européen des Sciences du Goût, located at the University of Burgundy, Dijon, France. I am grateful to my colleagues there for their generous hospitality during my visit, especially Profs S. Thornton, S. Nicolaidis, and M. Fantino.

References

[1] Darwin C. The expression of the emotions in man and animals. Oxford, UK: Harper Collins/Oxford University Press, 1872–1998 Revised and with commentary by P. Ekman.
[2] Grill HJ, Roitman MF, Kaplan JM. A new taste reactivity analysis of the integration of taste and physiological state information. Am J Physiol 1996;271:R677–87.
[3] Grill HJ, Spector AC, Schwartz GJ, Kaplan JM, Flynn FW.

Evaluating taste effects on ingestive behavior. In: Toates FM, Rowland NR, editors. Feeding and drinking, 1. Amsterdam: Elsevier, 1987. p. 151–88.
[4] Kaplan JM, Roitman MF, Grill HJ. Ingestive taste reactivity as licking behavior. Neurosci Biobehav Rev 1995;19:89–98.
[5] Kaplan JM, Spector AC, Grill HJ. Ingestion rate as an independent variable in the behavioral analysis of satiation. Am J Physiol 1990;258:R662–71.
[6] Spector AC, Breslin P, Grill HJ. Taste reactivity as a dependent measure of the rapid formation of conditioned taste aversion: a tool for the neural analysis of taste-visceral associations. Behav Neurosci 1988;102:942–52.
[7] Spector AC, Klumpp PA, Kaplan JM. Analytical issues in the evaluation of food deprivation and sucrose concentration effects on the microstructure of licking behavior in the rat. Behav Neurosci 1998;112:678–94.
[8] Travers JB, Norgren R. Electromyographic analysis of the ingestion and rejection of sapid stimuli in the rat. Behav Neurosci 1986;100:544–55.
[9] Steiner JE. The gustofacial response: observation on normal and anencephalic newborn infants. Symp Oral Sens Percep 1973;4:254–78.
[10] Steiner JE. Discussion paper: innate, discriminative human facial expressions to taste and smell stimulation. Ann NY Acad Sci 1974;237:229–33.
[11] Berridge KC. Food reward: brain substrates of wanting and liking. Neurosci Biobehav Rev 1996;20:1–25.
[12] Berridge KC, Robinson TE. What is the role of dopamine in reward: hedonic impact, reward learning, or incentive salience? Brain Res Rev 1998;28:309–69.
[13] Cabanac M, Lafrance L. Facial consummatory responses in rats support the ponderostat hypothesis. Physiol Behav 1991;50: 179–83.
[14] Chevialier-Skolnikoff S. Facial expression of emotion in nonhuman primates. In: Ekman P, editor. Emotion in the human face. Cambridge, UK: Cambridge University Press, 1982. p. 11–89.
[15] Clarke SN, Parker LA. Morphine-induced modification of quinine palatability: effects of multiple morphine–quinine trials. Pharmacol Biochem Behav 1995;51:505–8.
[16] Ekman P, Friesen W. Measuring facial movement with the facial action coding system. In: Ekman P, editor. Emotions in the human face. Cambridge, UK: Cambridge University Press, 1982. p. 179–215.
[17] Fox NA, Davidson RJ. Taste-elicited changes in facial signs of emotion and the asymmetry of brain electrical activity in human newborns. Neuropsychologia 1986;24:417–22.
[18] Frijda NH, Tcherkassof A. Facial expressions as modes of action readiness. In: Russell JA, Fernández-Dols JM, editors. The psychology of facial expression. Cambridge, UK: Cambridge University Press, 1997. p. 78–102.
[19] Ganchrow JR, Steiner JE, Daher M. Neonatal facial expressions in response to different qualities and intensities of gustatory stimuli. Infant Behav Dev 1983;6:189–200.
[20] Gilbert AN, Fridlund AJ, Sabini J. Hedonic and social determinants of facial displays to odors. Chem Senses 1987;12:355–63.
[21] Grill HJ, Berridge KC. Taste reactivity as a measure of the neural control of palatability. In: Sprague JM, Epstein AN, editors. Progress in psychobiology and physiological psychology, 11. Orlando, FL: Academic Press, 1985. p. 1–61.
[22] Grill HJ, Norgren R. The taste reactivity test. I. Mimetic responses to gustatory stimuli in neurologically normal rats. Brain Res 1978;143:263–79.
[23] Grill HJ, Norgren R. The taste reactivity test. II. Mimetic responses to gustatory stimuli in chronic thalamic and chronic decerebrate rats. Brain Res 1978;143:281–97.
[24] Izard CE, Fantauzzo CA, Castle JM, Haynes OM. The ontogeny and significance of infants' facial expressions in the first 9 months of life. Dev Psychol 1995;31(6):997–1013.
[25] Messinger DS, Fogel A, Dickson KL. A dynamic systems approach to infant facial action. In: Russell JA, Fernández-Dols JM, editors. The psychology of facial expression, Cambridge, UK: Cambridge University Press, 1997. p. 205–26.
[26] Parker LA. Rewarding drugs produce taste avoidance, but not taste aversion. Neurosci Biobehav Rev 1995;19:143–51.
[27] Peciña S. Contrasting roles of mesostriatal dopamine and opioid systems in food 'wanting' and 'liking'. Doctoral dissertation in Psychology, University of Michigan, Ann Arbor, 1998, 132 pp.
[28] Peciña S, Berridge KC, Parker LA. Pimozide does not shift palatability: Separation of anhedonia from sensorimotor suppression by taste reactivity. Pharmacol Biochem Behav 1997;58:801–11.
[29] Rosenstein D, Oster H. Differential facial responses to four basic tastes in newborns. Child Dev 1988;59:1555–68.
[30] Studies in emotion and social interaction. In: Russell JA, Fernández-Dols JM, editors. The psychology of facial expression. Cambridge, UK: Cambridge University Press, 1997 400 p.
[31] Soussignan R, Schaal B, Marlier L, Jiang T. Facial and autonomic responses to biological and artificial olfactory stimuli in human neonates: re-examining early hedonic discrimination of odors. Physiol Behav 1997;62:745–58.
[32] Soussignan R, Schall B. Children's facial responsiveness to odors: Influences of hedonic valence of odor, gender, age, and social presence. Dev Psychol 1996;32:367–79.
[33] Steiner JE. Human facial expressions in response to taste and smell stimulation. Adv Child Dev Behav 1979;13:257–95.
[34] Steiner JE, Glaser D. Taste-induced facial expressions in apes and humans. Human Evol 1995;10:97–105.
[35] Steiner JE, Glaser D, Hawilo ME, Berridge KC. Hedonic impact: facial expression of human infants, great apes, and monkeys. Submitted for publication.
[36] Beauchamp GK, Bachmanov A, Stein LJ. Development and genetics of glutamate taste preference. Ann NY Acad Sci 1998; 855:412–6.
[37] Bellisle F. Glutamate and the UMAMI taste: sensory, metabolic, nutritional and behavioural considerations. A review of the literature published in the last 10 years. Neurosci Biobehav Rev 1999;23:423–38.
[38] O'Mahony M, Ishii R. A comparison of English and Japanese taste languages: taste descriptive methodology, codability and the umami taste. Br J Psychol 1986;77:161–74.
[39] Rolls ET, Critchley HD, Wakeman EA, Mason R. Responses of neurons in the primate taste cortex to the glutamate ion and to inosine 5′-monophosphate. Physiol Behav 1996;59:991–1000.
[40] Rozin P, Lowery L, Ebert R. Varieties of disgust faces and the structure of disgust. J Pers Social Psychol 1994;66:870–81.
[41] Perl E, Un S, Hamburger R, Steiner JE. Taste- and odor-reactivity in elderly demented patients. Chem Senses 1992;17:779–94.
[42] Steiner JE, Glaser D. Differential behavioral responses to taste stimuli in non-human primates. J Human Evol 1984;13:709–23.
[43] Craig W. Appetites and aversions as constituents of instincts. Biol Bull Woods Hole 1918;34:91–107.
[44] Grill HJ. Production and regulation of ingestive consummatory behavior in the chronic decerebrate rat. Brain Res Bull 1980;5:79–87.
[45] Brining SK, Belecky TL, Smith DV. Taste reactivity in the hamster. Physiol Behav 1991;49:1265–72.
[46] Berridge K, Grill HJ, Norgren R. Relation of consummatory responses and preabsorptive insulin release to palatability and learned taste aversions. J Comp Physiol Psychol 1981;95:363–82.
[47] Berridge KC, Grill HJ. Isohedonic tastes support a two-dimensional hypothesis of palatability. Appetite 1984;5:221–31.
[48] Berridge KC, Peciña S. Benzodiazepines, appetite, and taste palatability. Neurosci Biobehav Rev 1995;19:121–31.
[49] Schmidt-Nielsen K. Scaling, why is animal size so important? New York: Cambridge University Press, 1984.
[50] Colonnese MT, Stallman EL, Berridge KC. Ontogeny of action

syntax in altricial and precocial rodents: grooming sequences by rat and guinea pig pups. Behavior 1996;133:1165–95.
[51] Horowitz GP, Stephan FK, Smith JC, Whitney G. Genetic and environmental variability in lick rates of mice. Physiol Behav 1977;19:493–6.
[52] Flynn FW. Applications of taste reactivity to the study of the neural-hormonal controls of ingestive behavior. Neurosci Biobehav Rev 1995;19:109–20.
[53] Kiefer SW. Alcohol, palatability, and taste reactivity. Neurosci Biobehav Rev 1995;18:133–41.
[54] Pelchat ML, Grill HJ, Rozin P, Jacobs J. Quality of acquired responses to tastes by Rattus norvegicus depends on type of associated discomfort. J Comp Psychol 1983;97:140–53.
[55] Crystal SR, Bernstein IL. Infant salt preference and mother's morning sickness. Appetite 1998;30:297–307.
[56] Soussignan R, Schaal B, Marlier L. Olfactory alliesthesia in human neonates: prandial state and stimulus familiarity modulate facial and autonomic responses to milk odors. Dev Psychobiol 1999;35:3–14.
[57] Berridge KC, Flynn FW, Schulkin J, Grill HJ. Sodium depletion enhances salt palatability in rats. Behav Neurosci 1984;98:652–60.
[58] Berridge KC, Schulkin J. Palatability shift of a salt-associated incentive during sodium depletion. Quart J Exp Psychol (b) 1989;41:121–38.
[59] Flynn FW, Grill HJ, Schulkin J, Norgren R. Central gustatory lesions. II. Effects on sodium appetite, taste aversion learning, and feeding behaviors. Behav Neurosci 1991;105:944–54.
[60] Flynn FW, Schulkin J, Havens M. Sex differences in salt preference and taste reactivity in rats. Brain Res Bull 1993;32:91–95.
[61] Galaverna OG, Seeley RJ, Berridge KC, Grill HJ, Epstein AN, Schulkin J. Lesions of the central nucleus of the amygdala. I. Effects on taste reactivity, taste aversion learning and sodium appetite. Behav Brain Res 1993;59:11–17.
[62] Grill HJ, Schulkin J, Flynn FW. Sodium homeostasis in chronic decerebrate rats. Behav Neurosci 1986;100:536–43.
[63] Schulkin J. Sodium hunger: the search for a salty taste. New York: Cambridge University Press, 1991.
[64] Cabanac M. Physiological role of pleasure. Science 1971;173:1103–7.
[65] Cabanac M. Sensory pleasure. Quart Rev Biol 1979;54:1–29.
[66] Laeng B, Berridge KC, Butter CM. Pleasantness of a sweet taste during hunger and satiety: effects of gender and "sweet tooth". Appetite 1993;21:247–54.
[67] Berridge KC. Modulation of taste affect by hunger, caloric satiety, and sensory-specific satiety in the rat. Appetite 1991;16:103–20.
[68] Cabanac M, Lafrance L. Postingestive alliesthesia: the rat tells the same story. Physiol Behav 1990;47:539–43.
[69] Beauchamp GK, Bertino M, Burke D, Engelman K. Experimental sodium depletion and salt taste in normal human volunteers. Am J Clin Nutr 1990;51:881–9.
[70] Nicolaidis S, Galaverna O, Metzler CH. Extracellular dehydration during pregnancy increases salt appetite of offspring. Am J Physiol 1990;258:R281–3.
[71] Rozin PN, Schulkin J. Food selection. In: Stricker EM, editor. Neurobiology of food and fluid intake, 10. New York: Plenum Press, 1990. p. 297–328.
[72] Breslin PA, Spector AC, Grill HJ. A quantitative comparison of taste reactivity behaviors to sucrose before and after lithium chloride pairings: a unidimensional account of palatability. Behav Neurosci 1992;106:820–36.
[73] Grill HJ, Norgren R. Chronically decerebrate rats demonstrate satiation but not bait shyness. Science 1978;201:267–9.
[74] Parker LA. Behavioral conditioned responses across multiple conditioning/testing trials elicited by lithium- and amphetamine-paired flavors. Behav Neural Biol 1984;41:190–9.
[75] Breslin PA, Davidson TL, Grill HJ. Conditioned reversal of reactions to normally avoided tastes. Physiol Behav 1990;47:535–8.
[76] Delameter AR, LoLordo VM, Berridge KC. Control of fluid palatability by exteroceptive Pavlovian signals. J Exp Psychol (Anim Behav) 1986;12:143–52.
[77] Kiefer SW, Bice PJ, Badia-Elder N. Alterations in taste reactivity to alcohol in rats given continuous alcohol access followed by abstinence. Alcohol Clin Exp Res 1994;18:555–9.
[78] Kiefer SW, Dopp JM. Taste reactivity to alcohol in rats. Behav Neurosci 1989;103:1318–26.
[79] Zellner DA, Berridge KC, Grill HJ, Ternes JW. Rats learn to like the taste of morphine. Behav Neurosci 1985;99:290–300.
[80] Berridge KC, Cromwell HC. Motivational-sensorimotor interaction controls aphagia and exaggerated treading after striatopallidal lesions. Behav Neurosci 1990;104:778–95.
[81] Cromwell HC, Berridge KC. Where does damage lead to enhanced food aversion: the ventral pallidum/substantia innominata or lateral hypothalamus? Brain Res 1993;624:1–10.
[82] Stellar JR, Brooks FH, Mills LE. Approach and withdrawal analysis of the effects of hypothalamic stimulation and lesions in rats. J Comp Physiol Psychol 1979;93:446–66.
[83] Teitelbaum P, Epstein AN. The lateral hypothalamic syndrome: recovery of feeding and drinking after lateral hypothalamic lesions. Psychol Rev 1962;69:74–90.
[84] Doyle TG, Berridge KC, Gosnell BA. Morphine enhances hedonic taste palatability in rats. Pharmacol Biochem Behav 1993;46:745–9.
[85] Hill KG, Kiefer SW. Naltrexone treatment increases the aversiveness of alcohol for outbred rats. Alcohol Clin Exp Res 1997;21:637–41.
[86] Parker LA, Maier S, Rennie M, Crebolder J. Morphine- and naltrexone-induced modification of palatability: analysis by the taste reactivity test. Behav Neurosci 1992;106:999–1010.
[87] Peciña S, Berridge KC. Central enhancement of taste pleasure by intraventricular morphine. Neurobiology 1995;3:269–80.
[88] Peciña S, Berridge KC. Morphine microinjections enhance feeding and palatability in the shell but not core of nucleus accumbens. Neurosci Abstr 1996;22:1408.
[89] Peciña S, Berridge K. Opioid feeding site objectively mapped within shell of nucleus accumbens. Neurosci Abstr 1997;23:529.
[90] Peciña S, Berridge KC. Localization of an opioid eating site in accumbens shell based on a Fos map of microinjections. Submitted for publication.
[91] Cleary J, Weldon DT, O'Hare E, Billington C, Levine AS. Naloxone effects on sucrose-motivated behavior. Psychopharmacology 1996;126:110–4.
[92] Cooper SJ, Jackson A, Morgan R, Carter R. Evidence for opiate receptor involvement in the consumption of a high palatability diet in nondeprived rats. Neuropeptides 1985;5:345–8.
[93] Giraudo SQ, Grace MK, Welch CC, Billington CJ, Levine AS. Naloxone's anorectic effect is dependent upon the relative palatability of food. Pharmacol Biochem Behav 1993;46:917–21.
[94] Higgs S, Cooper SJ. Midazolam induced rapid changes in licking behaviour: evidence for involvement of endogenous opioid peptides. Psychopharmacology 1997;131:278–86.
[95] Levine AS, Weldon DT, Grace M, Cleary JP, Billington CJ. Naloxone blocks that portion of feeding driven by sweet taste in food- restricted rats. Am J Physiol 1995;268:R248–52.
[96] Welch CC, Kim EM, Grace MK, Billington CJ, Levine AS. Palatability-induced hyperphagia increases hypothalamic dynorphin peptide and mRNA levels. Brain Res 1996;721:126–31.
[97] Drewnowski A, Krahn DD, Demitrack MA, Nairn K, Gosnell BA. Naloxone, an opiate blocker, reduces the consumption of sweet high-fat foods in obese and lean female binge eaters. Am J Clin Nutr 1995;61:1206–12.
[98] Yeomans MR, Gray RW. Effects of naltrexone on food intake and changes in subjective appetite during eating: evidence for opioid involvement in the appetizer effect. Physiol Behav 1997;62:15–21.
[99] Yeomans MR, Wright P. Lower pleasantness of palatable foods in nalmefene-treated human volunteers. Appetite 1991;16:249–59.
[100] Yeomans MR, Wright P, Macleod HA, Critchley JA. Effects of

nalmefene on feeding in humans. Dissociation of hunger and palatability. Psychopharmacology 1990;100:426–32.
[101] Cooper SJ, Higgs S. Neuropharmacology of appetite and taste preferences. In: Legg CR, Booth DA, editors. Appetite: neural and behavioural bases. New York: Oxford University Press, 1994. p. 212–42.
[102] Cooper SJ, Higgs S, Clifton PG. Behavioral and neural mechanisms for benzodiazepine-induced hyperphagia. Appetite 1995;24:78–79.
[103] Cooper SJ. Benzodiazepines as appetite-enhancing compounds. Appetite 1980;1:7–19.
[104] Kelly TH, Foltin RW, King L, Fischman MW. Behavioral response to diazepam in a residential laboratory. Biol Psychiatry 1992;31:808–22.
[105] Wise RA, Dawson V. Diazepam-induced eating and lever pressing for food in sated rats. J Comp Physiol Psychol 1974;86: 930–41.
[106] Cooper SJ, Estall LB. Behavioural pharmacology of food, water and salt intake in relation to drug actions at benzodiazepine receptors. Neurosci Biobehav Rev 1985;9:5–19.
[107] Berridge KC, Treit D. Chlordiazepoxide directly enhances positive ingestive reactions in rats. Pharmacol Biochem Behav 1986;24:217–21.
[108] Cooper SJ, Higgs S. The benzodiazepine agonist midazolam microinjected into the rat parabrachial nucleus produces hyperphagia. Soc Neurosci Abstr 1994;20:1285.
[109] Gray RW, Cooper SJ. Benzodiazepines and palatability: taste reactivity in normal ingestion. Physiol Behav 1995;58:853–9.
[110] Haney M, Comer SD, Fischman MW, Foltin RW. Alprazolam increases food intake in humans. Psychopharmacology 1997;132:311–4.
[111] Parker LA. Chlordiazepoxide enhances the palatability of lithium-, amphetamine-, and saline-paired saccharin solution. Pharmacol Biochem Behav 1995;50:345–9.
[112] Peciña S, Berridge KC. Brainstem mediates diazepam enhancement of palatability and feeding: microinjections into fourth ventricle versus lateral ventricle. Brain Res 1996;727:22–30.
[113] Söderpalm AHV, Hansen S. Benzodiazepines enhance the consumption and palatability of alcohol in the rat. Psychopharmacology 1998;137:215–22.
[114] Treit D, Berridge KC. A comparison of benzodiazepine, serotonin, and dopamine agents in the taste-reactivity paradigm. Pharmacol Biochem Behav 1990;37:451–6.
[115] Fuxe K, Farnebo LO, Hamberger B, Ogren SO. On the in vivo and in vitro actions of fenfluramine and its derivatives on central monoamine neurons, especially 5-hydroxytryptamine neurons, and their relation to the anorectic activity of fenfluramine. Postgrad Med J 1975;51(Suppl 1):35–45.
[116] Barnfield A, Parker LA, Davies AM, Miles C. Fenfluramine-induced modification of palatability: analysis by the taste reactivity test. Pharmacol Biochem Behav 1994;48:875–9.
[117] Gray RW, Cooper SJ. d-Fenfluramine's effects on normal ingestion assessed with taste reactivity measures. Physiol Behav 1996;59:1129–35.
[118] Berridge KC, Valenstein ES. What psychological process mediates feeding evoked by electrical stimulation of the lateral hypothalamus? Behav Neurosci 1991;105:3–14.
[119] Berridge KC, Venier IL, Robinson TE. Taste reactivity analysis of 6-hydroxydopamine-induced aphagia: implications for arousal and anhedonia hypotheses of dopamine function. Behav Neurosci 1989;103:36–45.
[120] Di Chiara G, Tanda G. Blunting of reactivity of dopamine transmission to palatable food: a biochemical marker of anhedonia in the CMS model? Psychopharmacology 1997;134:351–3 Discussion: p. 371–7.
[121] Gardner EL. Brain reward mechanisms. In: Lowinson JH, Ruiz P, Millman RB, Langrod JG, editors. Substance abuse: a comprehensive textbook. Baltimore, MD: Williams and Wilkin, 1997. p. 51–85.
[122] Koob GF, Le Moal M. Drug abuse: hedonic homeostatic dysregulation. Science 1997;278:52–58.
[123] Smith GP. Dopamine and food reward. In: Morrison AM, Fluharty SJ, editors. Progress in psychobiology and physiological psychology, 15. New York: Academic Press, 1995. p. 83–144.
[124] Wise RA. Neuroleptics and operant behavior: the anhedonia hypothesis. Behav Brain Sci 1982;5:39–87.
[125] Wise RA. The anhedonia hypothesis: Mark III. Behav Brain Sci 1985;8:178–86.
[126] Wise RA, Spindler J, deWit H, Gerberg GJ. Neuroleptic-induced "anhedonia" in rats: pimozide blocks reward quality of food. Science 1978;201:262–4.
[127] Leeb K, Parker L, Eikelboom R. Effects of pimozide on the hedonic properties of sucrose: analysis by the taste reactivity test. Pharmacol Biochem Behav 1991;39:895–901.
[128] Parker LA. Aversive taste reactivity: reactivity to quinine predicts aversive reactivity to lithium-paired sucrose solution. Pharmacol Biochem Behav 1994;47:73–75.
[129] Parker LA, Lopez Jr N. Pimozide enhances the aversiveness of quinine solution. Pharmacol Biochem Behav 1990;36:653–9.
[130] Parker L, Leeb K. Amphetamine-induced modification of quinine palatability: analysis by the taste reactivity test. Pharmacol Biochem Behav 1994;47:413–20.
[131] Berridge KC. Pleasure, pain, desire, and dread: hidden core processes of emotion. In: Kahneman D, Diener E, Schwartz N, editors. Well-being: the foundations of hedonic psychology. New York: Russell Sage Foundation, 1999. p. 527–59.
[132] Robinson TE, Berridge KC. The neural basis of drug craving: an incentive-sensitization theory of addiction. Brain Res Rev 1993;18:247–91.
[133] Maldonado-Irizarry CS, Swanson CJ, Kelley AE. Glutamate receptors in the nucleus accumbens shell control feeding behavior via the lateral hypothalamus. J Neurosci 1995;15:6779–88.
[134] Basso AM, Kelley AE. Feeding induced by GABA(A) receptor stimulation within the nucleus accumbens shell: regional mapping and characterization of macronutrient and taste preference. Behav Neurosci. 1999;113:324–36.
[135] Stratford TR, Kelley AE. GABA in the nucleus accumbens shell participates in the central regulation of feeding behavior. J Neurosci 1997;17:4434–40.
[136] Stratford TR, Kelley AE, Simansky KJ. Blockade of GABA(A) receptors in the medial ventral pallidum elicits feeding in satiated rats. Brain Res 1999;825:199–203.
[137] Baylis LL, Gaffan D. Amygdalectomy and ventromedial prefrontal ablation produce similar deficits in food choice and in simple object discrimination learning for an unseen reward. Exp Brain Res 1991;86:617–22.
[138] Butter CM, McDonald JA, Snyder DR. Orality, preference behavior, and reinforcement value of nonfood object in monkeys with orbital frontal lesions. Science 1969;164:1306–7.
[139] Burns LH, Annett L, Kelley AE, Everitt BJ, Robbins TW. Effects of lesions to amygdala, ventral subiculum, medial prefrontal cortex, and nucleus accumbens on the reaction to novelty: implication for limbic-striatal interactions. Behav Neurosci 1996;110:60–73.
[140] Fitzgerald RE, Burton MJ. Neophobia and conditioned taste aversion deficits in the rat produced by undercutting temporal cortex. Physiol Behav 1983;30:203–6.
[141] Kiefer SW, Orr MR. Taste avoidance, but not aversion, learning in rats lacking gustatory cortex. Behav Neurosci 1992;106:140–6.
[142] Galaverna O, De Luca Jr LA, Schulkin J, Yao SZ, Epstein AN. Deficits in NaCl ingestion after damage to the central nucleus of the amygdala in the rat. Brain Res Bull 1992;28:89–98.
[143] Schulkin J, Marini J, Epstein AN. A role for the medial region of the amygdala in mineralocorticoid-induced salt hunger. Behav Neurosci 1989;103:179–85.

[144] Seeley RJ, Galaverna O, Schulkin J, Epstein AN, Grill HJ. Lesions of the central nucleus of the amygdala. II. Effects on intraoral NaCl intake. Behav Brain Res 1993;59:19–25.
[145] Zhang DM, Epstein AN, Schulkin J. Medial region of the amygdala: involvement in adrenal-steroid-induced salt appetite. Brain Res 1993;600:20–26.
[146] Simbayi LC, Boakes RA, Burton MJ. Effects of basolateral amygdala lesions on taste aversions produced by lactose and lithium chloride in the rat. Behav Neurosci 1986;100:455–65.
[147] Dunn LT, Everitt BJ. Double dissociations of the effects of amygdala and insular cortex lesions on conditioned taste aversion, passive avoidance, and neophobia in the rat using the excitotoxin ibotenic acid. Behav Neurosci 1988;102:3–23.
[148] Burton MJ, Rolls ET, Mora F. Effects of hunger on the responses of neurons in the lateral hypothalamus to the sight and taste of food. Exp Neurol 1976;51:668–77.
[149] Hoebel BG, Mark GP, West HL. Conditioned release of neurotransmitters as measured by microdialysis. Clin Neuropharmacol, Part A 1992;15(Suppl 1):704A–5A.
[150] Norgren R. Gustatory system. In: Paxinos G, editor. The rat nervous system, New York: Academic Press, 1995. p. 751–71.
[151] Ono T, Sasaki K, Nakamura K, Norgren R. Integrated lateral hypothalamic neural responses to natural and artificial rewards and cue signals in the rat. Brain Res 1985;327:303–6.
[152] Rolls ET. The brain and emotion. Oxford, UK: Oxford University Press, 1999.
[153] Schwartzbaum JS. Electrophysiology of taste, feeding and reward in lateral hypothalamus of rabbit. Physiol Behav 1988;44:507–26.
[154] Schallert T, Whishaw IQ. Two types of aphagia and two types of sensorimotor impairment after lateral hypothalamic lesions: observations in normal weight, dieted, and fattened rats. J Comp Physiol Psychol 1978;92:720–41.
[155] Epstein AN. Instinct and motivation as explanations for complex behavior. In: Pfaff DW, editor. The physiology of motivation, New York: Springer, 1982. p. 25–58.
[156] Feurté S, Nicolaidis S, Berridge KC. Conditioned taste aversion in rats for a threonine-deficient diet: demonstration by the taste reactivity test. Submitted for publication.
[157] Flynn FW. Fourth ventricle bombesin injection suppresses ingestive behaviors in rats. Am J Physiol 1989;256:R590–6.
[158] Flynn FW. Effects of fourth ventricle bombesin injection on meal-related parameters and grooming behavior. Peptides 1991;12:761–5.
[159] Flynn FW. Caudal brain stem systems mediate effects of bombesin-like peptides on intake in rats. Am J Physiol 1992;262:R37–44.
[160] Flynn FW. Fourth ventricular injection of selective bombesin receptor antagonists facilitates feeding in rats. Am J Physiol 1993;264:R218–21.
[161] Flynn FW. Mammalian bombesin-like peptides suppress sham drinking of salt by sodium-deficient rats. Peptides 1996;17:951–6.
[162] Flynn FW, Robillard L. Inhibition of ingestive behavior following fourth ventricle bombesin injection in chronic decerebrate rats. Behav Neurosci 1992;106:1011–4.
[163] Flynn FW, Grill HJ. Intraoral intake and taste reactivity responses elicited by sucrose and sodium chloride in chronic decerebrate rats. Behav Neurosci 1988;102:934–41.
[164] Grill HJ, Kaplan JM. Caudal brainstem participates in the distributed neural control of feeding. In: Stricker EM, editor. Neurobiology of food and fluid intake, 10. New York: Plenum Press, 1990. p. 125–49.
[165] Schwartz GJ, Grill HJ. Relationships between taste reactivity and intake in the neurologically intact rat. Chem Senses 1984;9:249–72.
[166] Wise RA. A brief history of the anhedonia hypothesis. In: Legg CR, Booth DA, editors. Appetite: neural and behavioural bases. New York: Oxford University Press, 1994. p. 243–63.
[167] Le Magnen J. Habits and food intake. In: Code CF, editor. Alimentary canal, 1. Washington, DC: American Physiological Society, 1967.
[168] Rolls BJ. Sensory-specific satiety. Nutr Rev 1986;44:93–101.
[169] Rolls BJ, Rolls ET, Rowe EA, Sweeney K. Sensory specific satiety in man. Physiol Behav 1981;27:137–42.
[170] Parker LA, Rennie M. Naltrexone-induced aversions: assessment by place conditioning, taste reactivity, and taste avoidance paradigms. Pharmacol Biochem Behav 1992;41:559–65.
[171] Smith RJ, Parker LA. Chin rub CRs are elicited by flavors associated with apomorphine, scopolamine, methscopolamine, physostigmine and neostigmine. Pharmacol Biochem Behav 1985;23:583–9.
[172] Turenne SD, Miles C, Parker LA, Siegel S. Individual differences in reactivity to the rewarding/aversive properties of drugs: assessment by taste and place conditioning. Pharmacol Biochem Behav 1996;53:511–6.
[173] Seeley RJ, Kaplan JM, Grill HJ. Effects of interrupting an intraoral meal on meal size and meal duration in rats. Appetite 1993;20:13–20.
[174] Seeley RJ, Kaplan JM, Grill HJ. Effect of occluding the pylorus on intraoral intake: a test of the gastric hypothesis of meal termination. Physiol Behav 1995;58:245–9.
[175] Seeley RJ, Payne CJ, Woods SC. Neuropeptide Y fails to increase intraoral intake in rats. Am J Physiol 1995;268:R423–7.
[176] Kaplan JM, Bednar I, Sodersten P. Simultaneous display of sexual and ingestive behaviour by rats. J Neuroendocrin 1993;4:381–92.
[177] Qian M, Johnson AE, Sodersten P. CCK-8 inhibits ingestive behavior in rats with lateral hypothalamic 6-OHDA lesions. Neuroreport 1998;9:2763–7.
[178] Grigson PS, Kaplan JM, Roitman MF, Norgren R, Grill HJ. Reward comparison in chronic decerebrate rats. Am J Physiol 1997;273:R479–86.
[179] Ungerstedt U. Adipsia and aphagia after 6-hydroxydopamine induced degeneration of the nigro-striatal dopamine system. Acta Physiol Scand Suppl 1971;367:95–122.
[180] Schneider LH, Davis JD, Watson CA, Smith GP. Similar effect of raclopride and reduced sucrose concentration on the microstructure of sucrose sham feeding. Eur J Pharmacol 1990;186:61–70.
[181] Schneider LH, Watson CA, Davis JD, Gibbs J, Smith GP. Microstructural analysis of the inhibition of sucrose sham feeding by SCH 23390. Appetite 1989;12:122–4.
[182] Tyrka A, Gayle C, Smith GP. Raclopride decreases sucrose intake of rat pups in independent ingestion tests. Pharmacol Biochem Behav 1992;43:863–9.
[183] Wise RA, Raptis L. Effects of naloxone and pimozide on initiation and maintenance measures of free feeding. Brain Res 1986;368:62–68.
[184] Tyrka A, Smith GP. Potency of SCH 23390 for decreasing sucrose intake in rat pups depends on mode of ingestion. Pharmacol Biochem Behav 1991;39:955–61.
[185] Pfaffmann C, Norgren R, Grill HJ. Sensory affect and motivation. Ann NY Acad Sci 1977;290:18–34.
[186] Schafe GE, Bernstein IL. Forebrain contribution to the induction of a brainstem correlate of conditioned taste aversion. II. Insular (gustatory) cortex. Brain Res 1998;800:40–47.
[187] Schafe GE, Seeley RJ, Bernstein IL. Forebrain contribution to the induction of a cellular correlate of conditioned taste aversion in the nucleus of the solitary tract. J Neurosci 1995;15:6789–96.
[188] Parker LA, Carvell T. Orofacial and somatic responses elicited by lithium-, nicotine- and amphetamine-paired sucrose solution. Pharmacol Biochem Behav 1986;24:883–7.
[189] Kiefer SW, Badia-Elder N, Bice PJ. Taste reactivity in high alcohol drinking and low alcohol drinking rats. Alcohol Clin Exp Res 1995;19:279–84.
[190] Kiefer SW, Badia-Elder NE. Taste reactivity in high-alcohol-sensitive and low-alcohol-sensitive rats. Alcohol 1997;14:225–9.
[191] Nader K, Bechara A, van der Kooy D. Neurobiological constraints on behavioral models of motivation. Ann Rev Psychol 1997;48:85–114.
[192] Rozin P, Fallon A. The acquisition of likes and dislikes for foods, What is America eating? Proceedings of a Symposium (National

Research Council). Washington, DC: National Academy Press, 1986. p. 58–71.
[193] Berridge KC, Zajonc RB. Hypothalamic cooling elicits eating: differential effects on motivation and emotion. Psychol Sci 1991;2:184–9.
[194] Chang FC, Scott TR. Conditioned taste aversions modify neural responses in the rat nucleus tractus solitarius. J Neurosci 1984;4:1850–62.
[195] Mark GP, Scott TR. Conditioned taste aversions affect gustatory activity in the NTS of chronic decerebrate rats. Neurosci Abstr 1988;14:1185.
[196] Mark GP, Scott TR, Chang FC, Grill HJ. Taste responses in the nucleus tractus solitarius of the chronic decerebrate rat. Brain Res 1988;443:137–48.
[197] Rolls ET, Murzi E, Yaxley S, Thorpe SJ, Simpson SJ. Sensory-specific satiety: food-specific reduction in responsiveness of ventral forebrain neurons after feeding in the monkey. Brain Res 1986;368:79–86.
[198] Rolls ET, Yaxley S, Sienkiewicz ZJ. Gustatory responses of single neurons in the caudolateral orbitofrontal cortex of the macaque monkey. J Neurophysiol 1990;64:1055–66.
[199] Scott TR. The effect of physiological need on taste. In: Capaldi ED, Powley TL, editors. Taste, experience, and feeding. Washington, DC: American Psychological Association, 1990. p. 45–61.
[200] Scott TR, Yaxley S, Sienkiewicz ZJ, Rolls ET. Gustatory responses in the nucleus tractus solitarius of the alert cynomolgus monkey. J Neurophysiol 1986;55:182–200.
[201] McCaughey SA, Scott TR. The taste of sodium. Neurosci Biobehav Rev 1998;22:663–76.
[202] Contreras RJ. Changes in gustatory nerve discharges with sodium deficiency: a single unit analysis. Brain Res 1977;121:373–8.
[203] Jacobs KM, Mark GP, Scott TR. Taste responses in the nucleus tractus solitarius of sodium-deprived rats. J Physiol 1988;406:393–410.
[204] Tamura R, Norgren R. Repeated sodium depletion affects gustatory neural responses in the nucleus of the solitary tract of rats. Am J Physiol 1997;273:R1381–91.
[205] Fregly MJ. Specificity of the sodium chloride appetite of adrenalectomized rats: substitution of lithium chloride for sodium chloride. Am J Physiol 1958;195:645–53.
[206] Richter CP. Salt appetite of mammals: its dependence on instinct and metabolism. L'instinct dans le comportement des animaux et de l'homme, Paris: Masson, 1956. p. 577–632.
[207] Drewnowski A, Krahn DD, Demitrack MA, Nairn K, Gosnell BA. Taste responses and preferences for sweet high-fat foods: evidence for opioid involvement. Physiol Behav 1992;51:371–9.
[208] O'Hare E, Cleary J, Bartz PJ, Weldon DT, Billington CJ, Levine AS. Naloxone administration following operant training of sucrose/water discrimination in the rat. Psychopharmacology 1997;129:289–94.
[209] Rideout HJ, Parker LA. Morphine enhancement of sucrose palatability: Analysis by the taste reactivity test. Pharmacol Biochem Behav 1996;53:731–4.
[210] Crystal SR, Bernstein IL. Morning sickness: impact on offspring salt preference. Appetite 1995;25:231–40.
[211] Berridge KC, Fentress JC. Trigeminal-taste interaction in palatability processing. Science 1985;228:747–50.
[212] Berridge KC, Grill HJ. Alternating ingestive and aversive consummatory responses suggest a two-dimensional analysis of palatability in rats. Behav Neurosci 1983;97:563–73.
[213] LeDoux J. The emotional brain: the mysterious underpinnings of emotional life, New York: Simon and Schuster, 1996.
[214] Murphy ST, Zajonc RB. Affect, cognition, and awareness: affective priming with optimal and suboptimal stimulus exposures. J Pers Social Psychol 1993;64:723–39.
[215] Winkielman P, Zajonc RB, Schwarz N. Subliminal affective priming resists attributional interventions. Cogn Emotion 1997;11:433–65.
[216] Zajonc RB. Feeling and thinking: preferences need no inferences. Am Psychol 1980;35:151–75.
[217] Kahneman D. Assessments of individual well-being: a bottom–up approach. In: Kahneman D, Diener E, Schwartz N, editors. Well-being: the foundations of hedonic psychology. New York: Russell Sage Foundation, 1999.
[218] Kahneman D, Fredrickson BL, Schreiber CA, Redelmeier DA. When more pain is preferred to less: adding a better end. Psychol Sci 1993;4:401–5.
[219] Kahneman D, Wakker PP, Sarin R. Back to Bentham? Explorations of experienced utility. Quart J Econ 1997;112:375–405.
[220] Nisbett RW, Wilson TD. Telling more than we can know: verbal reports on mental processes. Psychol Rev 1978;84:231–59.
[221] Wilson TD, Schooler JW. Thinking too much: introspection can reduce the quality of preferences and decisions. J Pers Social Psychol 1991;60:181–92.
[222] Berridge KC. Brainstem systems mediate the enhancement of palatability by chlordiazepoxide. Brain Res 1988;447:262–8.
[223] Higgs S, Cooper SJ. Hyperphagia induced by direct administration of midazolam into the parabrachial nucleus of the rat. Eur J Pharmacol 1996;313:1–9.
[224] Booth DA. Learned ingestive motivation and the pleasures of the palate. In: Bolles RC, editor. The hedonics of taste. Hillsdale, NJ: Lawrence Erlbaum, 1991. p. 29–58.
[225] Brown L. The new shorter Oxford English Dictionary on historical principles. Oxford/New York: Clarendon Press/Oxford University Press, 1993.
[226] Beauchamp GK, Mason JR. Comparative hedonics of taste. In: Robert CB, editor. The hedonics of taste. Hillsdale, NJ: Lawrence Erlbaum, 1991. p. 159–83.
[227] Clifton PG, Rusk IN, Cooper SJ. Effects of dopamine D1 and dopamine D2 antagonists on the free feeding and drinking patterns of rats. Behav Neurosci 1991;105:272–81.
[228] Clifton PG, Vickers SP, Somerville EM. Little and often: ingestive behavior patterns following hippocampal lesions in rats. Behav Neurosci 1998;112:502–11.
[229] Davis JD. Deterministic and probabilistic control of the behavior of rats ingesting liquid diets. Am J Physiol 1996;270:R793–800.
[230] Tyrka A, Smith GP. SCH23390, but not raclopride, decreases intake of intraorally infused 10% sucrose in adult rats. Pharmacol Biochem Behav 1993;45:243–6.