

What Psychological Process Mediates Feeding Evoked by Electrical Stimulation of the Lateral Hypothalamus?

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Because electrical stimulation of the lateral hypothalamus (ESLH) can elicit both feeding and reward, most investigators have concluded that stimulation does not evoke the aversive cues associated with hunger. It has been hypothesized, instead, that ESLH primes ingestion by evoking pleasurable taste sensations. A direct taste of this hedonic hypothesis was undertaken in rats that showed stimulus-bound feeding. Contrary to the prediction, it was found that the taste reactions (gapes, tongue protrusions, etc.) during ESLH were more aversive than hedonic. It is suggested that the stimulation influences behavior by potentiating the salience, but not the hedonic value, of external stimuli. The advantages of this incentive salience hypothesis are that it circumvents the need to postulate a hedonic sensory experience during stimulation and that it can explain how evoked feeding may switch to other behaviors when conditions are altered.

Electrical stimulation of the lateral hypothalamus (ESLH) induces eating in rats, guinea pigs, squirrels, cats, dogs, goats, monkeys, and humans. In fact, all mammals that have been studied show ESLH-induced eating (see Glickman & Schiff, 1967; Valenstein, Cox, & Kakolewski, 1970, for reviews). Despite the ease of demonstrating the phenomenon, there is no generally accepted explanation of why ESLH should cause satiated animals to eat. Initially, it was widely accepted that the stimulation must evoke the drive or depletion cues of hunger, that is, cause the animal to experience hunger (Miller 1960, 1973), but subsequent investigations have made this explanation less credible. There are now numerous demonstrations that animals do not behave as though the stimulation had made them hungry. For example, stimulation-elicited feeding appears rigidly focused on particular sensory qualities of a food and, following minor changes in the food or the testing conditions, the stimulated animal may stop eating and switch to a behavior unrelated to feeding (Valenstein, 1975). Further, ESLH does not initially induce rats to seek out food odors, although food deprivation does (Schallert, 1977). Most puzzling for the hypothesis that ESLH evokes hunger was the finding that animals self-stimulate at electrode sites capable of inducing eating even when no food is available (Glickman & Schiff, 1967; Hoebel & Teitelbaum, 1962; Margules & Olds, 1962; Phillips, Cox, Kakolewski, & Valenstein, 1969; Valenstein, 1969). Why would animals find stimulation rewarding if it made them hungry? This was a paradox that could not be resolved. Other explanations have had to be considered.

In an attempt to explain why ESLH might be rewarding and evoke eating, without assuming that it evokes the ex-

perience of hunger, it has been hypothesized that stimulation produces a *hedonic sensory experience* similar to the priming effect that occurs when ingesting a small morsel of palatable food such as a single salty peanut (Hoebel, 1988). The effect of ESLH, according to this hypothesis, would be equivalent to the effect of squirting a small quantity of hedonically pleasant sucrose into the mouth. Hedonic sensation might serve as a reward for instrumental behavior and also facilitate ingestion by "whetting the appetite." Whether appetite that is primed by a small morsel of food should be considered to be hunger is a matter of definition, but it is important to note that such priming effects would not require the direct creation of aversive depletion states. Thus, ESLH could both elicit and reinforce ingestive behavior by activating a neural system of hedonic reward. There are three lines of evidence for the existence of a hypothalamic neural system that could be capable of directly evoking hedonic experience without eliciting aversive depletion cues.

Intrinsic Neurons

One candidate for a substrate of hedonic reward is the set of intrinsic hypothalamic "reward" neurons that have been identified electrophysiologically as responding to food and that appear sensitive to the hedonic quality of stimuli. It has been shown that hypothalamic neurons respond to the taste (Norgren, 1976, 1983) or sight of a palatable food reward and to the sight of conditioned stimuli that have in the past preceded the reward (Rolls, 1981). Particular subregions of the lateral hypothalamus (LH) contain populations of neurons that respond selectively to positive rewards (Schwartzbaum, 1989). By activating neurons in these subregions, electrical stimulation itself might be rewarding.

Dopamine Projections

A second candidate for a hedonic or reward substrate is the ascending system of mesotelencephalic dopamine fibers, which projects from the substantia nigra and ventral teg-

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mentum to the neostriatum and nucleus accumbens. The suggestion that this mesotelencephalic dopamine system might be the neural substrate for the experience of reward has found its strongest formulation in the anhedonia hypothesis of Wise (1978, 1982, 1985), who argued that lesions or blockade of the dopamine system leaves "humans and lower animals incapable of experiencing many, if not all, forms of pleasure" (Wise, 1985, p. 179). A role for dopaminergic systems in some aspect of reward has been supported by studies of the effect of dopamine blockade on instrumental performance for food, ESLH, and pharmacological rewards (Wise & Rompre, 1989), although there is evidence that nigrostriatal dopamine systems are not essential for basic hedonic processes (Berridge, Venier, & Robinson, 1989). Dopamine projections have also been implicated in the elicitation of feeding by ESLH (Mittleman, Castañeda, Robinson, & Valenstein, 1986; Phillips & Fibiger, 1973; Phillips & Nikaido, 1975; Rowland, Marques, & Fisher, 1980) and at least indirectly in ESLH self-stimulation reward (Shizgal, Kiss, & Bielajew, 1982; Wise & Bozarth, 1984).

Gustatory Modulation

Pathways that descend from the LH also provide a candidate for hedonic modulation through their influence on brainstem sensory nuclei (Saper, Swanson, & Cowan, 1979). Taste-sensitive neurons in the hindbrain nucleus of the solitary tract are activated electrophysiologically by sucrose or quinine on the tongue (Norgren, 1976, 1983). Some of these neurons can also be activated by ESLH (Murzi, Hernandez, & Baptista, 1986). The potential of ESLH to activate brainstem gustatory neurons in a manner similar to taste has led to the intriguing suggestion that stimulation-evoked feeding and reward may both be mediated by "artificial taste." According to this hypothesis, ESLH is thought to produce the direct sensation of a palatable taste such as sweetness. Hoebel (1988) has been the strongest proponent of this view, suggesting that rewarding LH stimulation "may reinforce behavior by mimicking taste input from the tongue" (p. 590) and concluding that this same "taste effect helps to explain why LH stimulation induces feeding . . . the stimulation probably whets the appetite as if it were food" (p. 608). Thus, a descending system from the hypothalamus that evokes taste is held by this hypothesis to exert a priming effect that potentiates feeding much as does a palatable morsel of food.

Hedonic Enhancement and Taste Reactivity

Intrinsic neurons of the LH, ascending dopaminergic projections, and descending gustatory pathways all are potential mediators of behavior elicited by ESLH. Each candidate has been argued to be activated during ingestion of palatable food. Artificial activation of these candidates by a stimulating electrode could be expected to evoke hedonics directly, even in the absence of actual food. Stimulation that evoked food hedonics directly would be perceived as food, reacted to as food, and would reinforce and elicit further behavior similarly to food.

If ESLH elicits feeding by activating food hedonics, then this should have a behavioral manifestation similar to the manifestation of natural taste pleasure. Our ability to detect changes in relevant hedonic processes in animals, of course, requires a measure that is sensitive specifically to the palatability of food. This measure should be dissociable from purely sensory (as opposed to hedonic) properties of taste and from reflexive sensorimotor actors. The measure must also be capable of dissociation from measures of actual intake, if it is not to be a redundant assessment simply of the decision to feed rather than of taste affect. The measure that best meets these criteria of sensitivity plus selectivity is the *taste reactivity test* devised by Grill and Norgren (1978b), which uses a pattern of natural reactions elicited by palatable or aversive tastes (Figure 1). These reactions are not tied as fixed reflexes to the mere sensory properties of a taste: Psychological manipulations that alter the palatability of a taste through associative conditioning can change taste reactivity even though the stimulus remains constant (see Grill & Berridge, 1985, for review). Nor does the taste reactivity test merely reflect the decision to ingest (which is influenced by many factors besides taste affect): Taste reactivity can be dissociated from intake measures by physiological or psychological manipulations that alter intake by other means than through food hedonics (Berridge et al., 1989; Pelchat, Grill, Rozin, & Jacobs, 1983).

If LH stimulation does evoke feeding by enhancing taste hedonics, then this enhancement should be reflected by an increase in hedonic taste reactivity during stimulation, just as hedonic enhancement of taste reactivity has been shown to follow food deprivation, sodium depletion, and many other causes of appetite (e.g., Berridge, Flynn, Schulkin, & Grill, 1984; Berridge & Treit, 1986; Grill & Norgren, 1978a). According to the hedonic hypothesis of ESLH-elicited feeding, the administration of ESLH should enhance hedonic reactivity to taste just as would the addition of sucrose to that taste (Berridge & Grill, 1984). In the present experiment, we used taste reactivity measures to determine whether the palatability of taste is enhanced by ESLH in a rat that emits stimulation-bound feeding.

Method

Subjects

Eighty-two male Long-Evans rats (300–450 g) were housed in group cages on a 14:10-hr light–dark cycle. Testing was conducted during the light portion of the cycle. Free access to food and water was provided throughout the experiment.

Surgery

Each rat was pretreated with Bicillin (penicillin G benzathine; 30,000 units im) and anesthetized with ketamine (100 mg/kg im) and xylazine (10 mg/kg im). Bilateral stimulating electrodes were implanted in the LH. Bilateral oral cannulae were implanted lateral to the left and right first maxillary molars to allow taste reactivity testing. Electrodes were formed of bipolar, twisted wire that was insulated except at the tip (Plastic Products, Roanoke, VA). In 38 rats, electrodes were implanted 3.25 mm posterior to bregma, 1.3

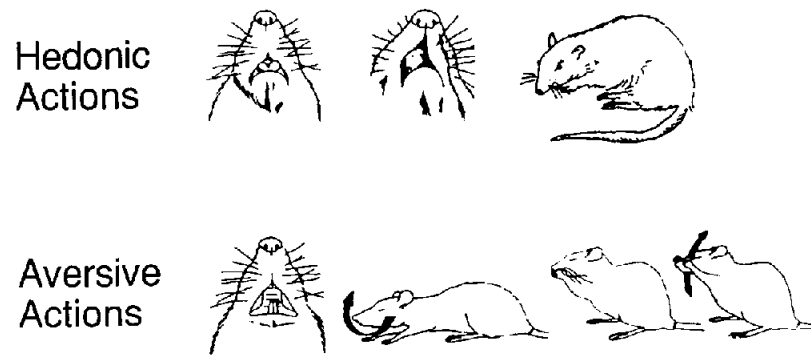


Figure 1. Affective reactions to taste. (Hedonic reactions [top] are elicited by sucrose and other palatable tastes. Hedonic reactions include rhythmic midline tongue protrusion, nonrhythmic lateral tongue protrusion, and paw lick. Aversive reactions [bottom] are elicited by quinine and other non-palatable tastes. Aversive reactions include gape, head shake, face wash, and forelimb flail.)

mm lateral to the midline, and 8.75 mm ventral to a level skull surface. In the remaining 44 rats, the coordinates were $A = -2.0$, $L = \pm 1.8$, and $V = -8.5$ from the same landmarks. Oral cannulae were constructed of polyethylene (PE 100) tubing, flared at one end, and anchored against the edge of the molar with a Teflon washer. Electrodes and oral cannulae were anchored with skull screws and acrylic cement.

Screening for Stimulation-Bound Feeding

One week after surgery, animals were screened for the ability of ESLH to elicit feeding. A rat was placed within the test chamber, and one electrode was connected for stimulation. After 15 min of habituation, stimulation (60 cycle sine wave) was administered beginning at $4 \mu\text{A}$ on a 20-s on–20-s off alternating schedule. Current intensity was increased by $1 \mu\text{A}$ every alternation until the rat (a) ate, (b) displayed either agitation or forced motor responses, or (c) the current reached $30 \mu\text{A}$. If either of the latter occurred, then the trial was stopped. If the rat ate, then that current was maintained for the remainder of the trial. Only if the rat continued to eat or lick at food during stimulation for 20 consecutive stimulation deliveries was it classified as showing a *positive trial*. Rats were required to show positive trials on 3 consecutive days to be classified as *positive stimulation-bound feeders*. Only the 12 rats that reached this criterion for positive stimulation-bound feeding were used in subsequent tests.

Behavioral Testing

Testing was carried out in a Plexiglas food chamber with a transparent floor. A mirror was angled beneath the transparent floor to reflect an image of the rat's head and face into the close-up lens of a video camera. Behavior was videotaped during testing for subsequent slow-motion analysis. Stimulation-bound feeders were tested in three types of condition. First, to confirm that hypothalamic stimulation evoked feeding even when alternative opportunities for action were available, food pellets were placed in the test chamber together with a water bottle while the rat received intermittent periods of ESLH. Second, to ascertain whether the hedonic value of a particular taste was enhanced by ESLH, taste reactivity tests were performed in which solutions of sweet sucrose, sour HCl, or bitter quinine were infused through the oral cannula into the rat's mouth while ESLH was alternately delivered or withheld. Third, to ascertain whether ESLH by itself elicited "vacuum reactions" (movements of

the mouth or tongue even when the mouth was empty) that might affect the scoring or interpretation of taste reactivity data, rats were treated again as in the first condition but without food pellets, water bottle, or any oral infusions.

Confirmation of ESLH-Induced Feeding

Stimulation-bound feeding was videotaped for subsequent detailed analysis in a procedure similar to that used for screening. During an 8-min trial, a stimulation-bound feeder received ESLH in alternating 15-s on–15-s off periods at the current intensity chosen to be effective for that rat during screening. Noyes food pellets were scattered across the floor, and a drinking spout that contained distilled water was available. This test was repeated three times for each rat to ensure the stability of stimulation-bound feeding: once before taste reactivity testing, once after 3 days of taste reactivity testing, and once subsequent to the completion of taste reactivity testing.

Videotaped records were subsequently scored in slow motion for the following actions: *food licking*, *food chewing*, *food grasping* (with mouth or forepaws), *food carrying*, *food dropping*, *object grasping* (biting or reaching for nonfoods such as electrode cord, water spout, wall, etc.), *object licking* (nonfood), *body licking*, *spontaneous tongue protrusion* (without contact with food, object, or body), *spontaneous mouth movement* (rhythmic or irregular opening and closing of the mouth), *rearing*, and any *aversive action* ordinarily emitted to an unpalatable taste (see next section). Grasping, carrying, rearing, and aversive actions were scored each time they occurred. Licking, chewing, mouth movement actions, and locomotion were scored in bouts of up to 5 s duration (e.g., 10 s of continuous licking was scored as two bouts).

Taste Reactivity Palatability Test

Six taste stimuli of different palatability were used to elicit taste reactivity: Sucrose (0.3 M and 1.0 M), sour HCl (0.01 M and 0.1 M), and quinine HCl (3×10^{-5} M and 3×10^{-4} M). A rat was tested for taste reactivity to only one stimulus per day. The order of presentation was random.

For each trial, a rat's oral cannulae were connected to stimulus delivery tubes (PE 50 with PE 10 nozzles), and its LH electrode was connected to the stimulation source (rats that fed to stimulation of both left and right electrodes were tested separately with stimulation

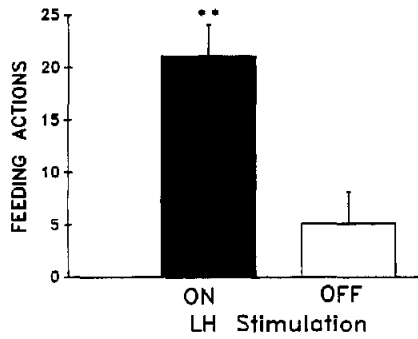


Figure 2. Electrical stimulation of the lateral hypothalamus (LH) elicited feeding by rats classified as positive stimulation-bound feeders. (Bars show the mean $[\pm SE]$ number of actions directed toward food [licking, chewing, grasping, and carrying food] during electrical stimulation of the LH and during its absence. Double asterisk denotes difference significant at $p < .01$ level.)

of each). After 5 min of habituation, a 1-ml volume of the taste solution was infused evenly over 1 min into the mouth via the cannulae. During the minute of infusion, ESLH was delivered in alternating 15-s on–15-s off periods. Each rat thus received a total of 30 s of exposure to the taste in the presence of ESLH stimulation and 30 s of exposure to the taste without ESLH. Behavior was videotaped for subsequent slow-motion analysis.

An observer who was unaware of the content of the taste stimulus scored the videotaped taste reactivity record for the occurrence of positive hedonic acts and aversive acts and for the occurrence of all nonfeeding acts that had been previously scored. *Positive hedonic reactions* were the following: *Lateral tongue protrusions* (nonrhythmic) lasting about 160 ms; *rhythmic tongue protrusions*, along the midline with a cycle length of roughly 160 ms; and *paw licking*. *Aversive reactions* were the following: *Gapes*, large openings of the mouth and jaw lasting about 125 ms; *chin rubbing*, bringing the chin in direct contact with the floor and projecting the body forward; *face washing*, either a single wipe with the paws over the face or a bout of several wipes; *paw treading*, rhythmic movements of the two paws forward and backward at a rate of roughly 3 Hz; *forelimb flails*, shaking of the forelimb back and forth; and *rapid headshaking*. Reactions that were considered to be neither strongly hedonic nor strongly aversive were rhythmic *mouth movements* of the jaw and lips and *passive drip* of the solution out of the mouth. Mouth movements and passive drip were scored in bouts of up to 5 s. Continuous rhythmic tongue protrusions were scored in bouts of up to 2 s. All other actions were scored each time they occurred.

Direct Elicitation of Vacuum Reactions

It was possible that ESLH stimulation may have elicited motor reactions even in the absence of a taste solution or external stimulus. If this happened, then such vacuum reactions could influence the interpretation of taste reactivity changes during ESLH, especially if those vacuum reactions involved tongue, mouth, or paw movement. To ascertain whether ESLH would elicit orofacial motor responses directly, each rat was tested in the food chamber as already described except that food pellets were not present. Behavior was videotaped and subsequently scored for each behavior (except food-related categories): *object grasping*, *object licking*, *body licking*, *tongue protrusion*, *mouth movement*, *rearing*, and *aversive actions* (gape, forelimb flail, etc.).

Results

Behavior Emitted in the Presence of Food

All rats that had been classified as positive feeders during screening demonstrated clear stimulation-bound feeding again when videotaped in the food chamber (Figure 2). For the purpose of statistical analysis, behavior emitted during the food chamber test was combined into the categories: *Feeding actions* (grasping, carrying, or licking food, in addition to actual chewing and ingestion), *nonfood object-related actions* (grasping, licking, biting of spout, wall, floor, or body), *tongue actions* (spontaneous rhythmic or lateral protrusions that were not directed at an object), spontaneous *mouth actions* that were neither aversive nor directed at a food or nonfood object, *aversive actions* (gaping, etc.), and *locomotion* (including rearing).

ESLH significantly altered the emission of behavior across these categories, analysis of variance (ANOVA) $F(1, 11) = 6.56$, $p < .05$, with an interaction between stimulation and behavioral category, $F(5, 55) = 3.46$, $p < .01$ (Figure 3). Feeding actions alone were increased significantly by ESLH (Figure 2; Newman-Keuls, $p < .01$). Feeding actions were increased during ESLH to approximately four times the levels emitted when stimulation was not delivered (Figure 2). A marginal enhancement of locomotion ($p = .09$) was the only other behavioral change produced by ESLH in the presence of food (Figure 3).

Hedonic and Aversive Taste Reactivity

ESLH interacted with taste quality to influence palatability but did not produce an overall enhancement of positive taste hedonics (Figure 4). ESLH amplified affective reactions only for tastes that normally elicited very low levels of either

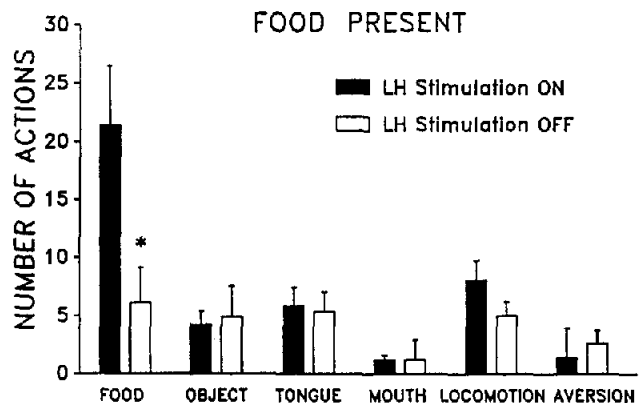


Figure 3. Specificity of behavior elicited by electrical stimulation of the lateral hypothalamus (LH) when food was available. (Only feeding behavior is increased during electrical stimulation of the LH. Behavioral categories are food directed, nonfood object directed [grasping, biting, licking of spout, electrode cord, chamber surface, or body], spontaneous tongue protrusions, spontaneous mouth or jaw movements, locomotion [walking forward or rearing], and aversive reactions [gapes, head shakes, forelimb flails, etc.]. Asterisk denotes difference significant at $p < .01$ level.)

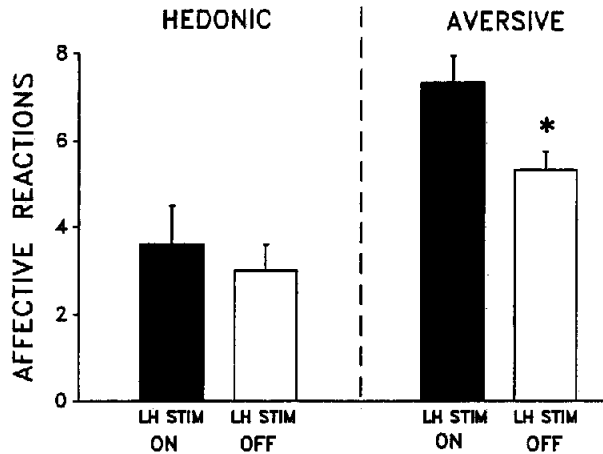


Figure 4. Overall hedonic and aversive reactions to infusions of sweet, sour, or bitter tastes during electrical stimulation of the lateral hypothalamus (LH STIM) and during its absence. (Hedonic reactions are unchanged overall by electrical stimulation. Aversive reactions are enhanced overall by electrical stimulation. Responses to the six solutions of sucrose, HCl, and quinine have been collapsed together to provide a general mean [$\pm SE$].)

positive hedonic or aversive reactivity (Figures 5 and 6). Surprisingly, the enhancement of aversion by ESLH was more pronounced overall than the enhancement of positive hedonics (Figure 4).

ESLH enhanced positive hedonics for one of the six tastes: highly concentrated quinine, a normally unpalatable taste (Figure 5), ANOVA (Taste \times Stimulation interaction) $F(2, 22) = 3.65, p < .05$; Newman-Keuls $p < .05$ for ESLH effect on concentrated quinine. ESLH appeared to decrease hedonic reactions to the taste of dilute HCl ($p < .05$) but did not further enhance tastes that were normally palatable (e.g., sucrose). Overall, ESLH had no effect on hedonic reactivity when taste interaction was not considered, $F(1, 11) = 1.42$ (Figure 4).

On the other hand, aversive reactivity was enhanced by ESLH overall (Figure 4), $F(1, 11) = 10.58, p < .01$, but even aversive enhancement showed an interaction with taste quality (Figure 6), $F(2, 22) = 4.52, p < .05$. The enhancement of aversion by ESLH was most pronounced for tastes that normally are relatively palatable and that elicit little or no aversion naturally: sucrose, in high and low concentrations (Newman-Keuls, $p < .05$ each), and sour HCl (in high concentration, $p < .05$).

Taste controlled both hedonic and aversive reactivity, regardless of the presence of ESLH (Figures 5 and 6). Taste quality (sucrose, HCl, or quinine) was the chief determinant both of hedonic, $F(2, 22) = 4.52, p < .05$, and of aversive reactions, $F(2, 22) = 24.57, p < .01$, whether ESLH was on or off.

Vacuum Reaction Elicitation

In the absence of food, relatively fewer actions of most types were elicited in the vacuum reaction chamber (Figure

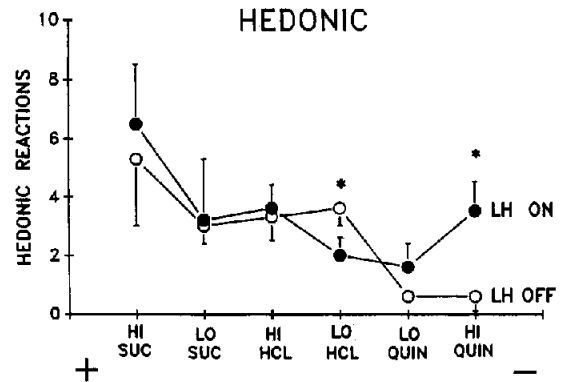


Figure 5. Comparison across tastes of hedonic reactions elicited during electrical stimulation of the lateral hypothalamus (LH) or during its absence. (Hedonic reactions generally were appropriate to a particular taste. Electrical stimulation of the LH increased hedonic reactions only to concentrated quinine and reduced hedonic reactions to dilute HCl. Hedonic responses to other tastes were unchanged by electrical stimulation of the LH. Open circles show mean [$\pm SE$] number of hedonic reactions in the absence of electrical stimulation of the LH. Filled circles show hedonic reactions during LH electrical stimulation. SUC = sucrose; HCL = hydrochloric acid; QUIN = quinine.)

7), but ESLH delivered in 15-s alternating on-off bins still altered ongoing behavior, $F(1, 11) = 10.85, p < .01$, differently across behavioral categories, $F(4, 44) = 4.11, p < .01$. Only locomotion was evoked to a strong degree by ESLH (Newman-Keuls, $p < .05$). Spontaneous tongue protrusions showed a marginal enhancement in the presence of ESLH ($p = .07$). No other increases in oral, object-directed, or aversive actions could be detected in the presence of ESLH.

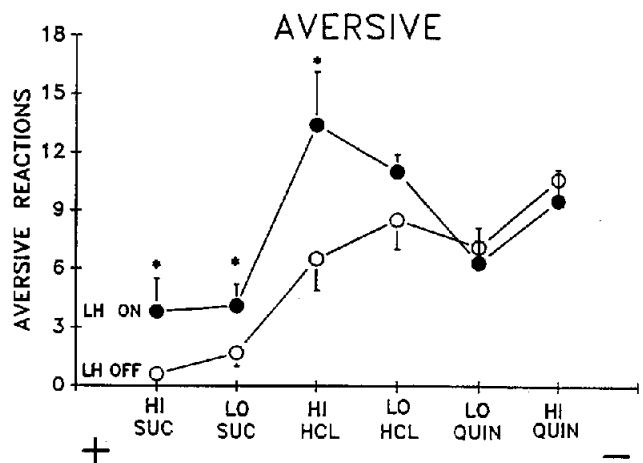


Figure 6. Comparison across tastes of aversive reactions elicited during electrical stimulation of the lateral hypothalamus (LH) and during its absence. (Open circles show mean [$\pm SE$] number of hedonic reactions in the absence of electrical stimulation of the LH. Filled circles show hedonic reactions during LH electrical stimulation. SUC = sucrose; HCL = hydrochloric acid; QUIN = quinine.)

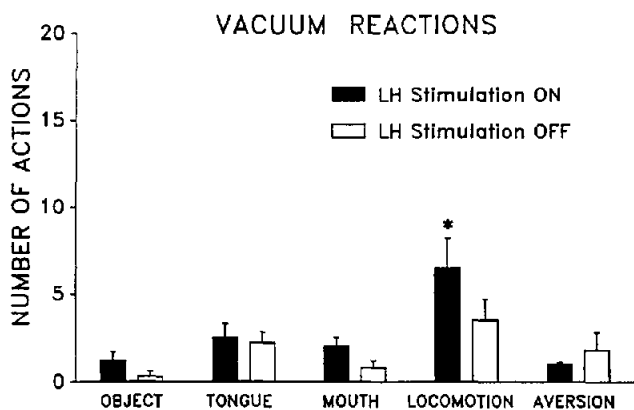


Figure 7. Vacuum reactions elicited by electrical stimulation of the lateral hypothalamus (LH) when food was not available. (Only locomotion was enhanced during electrical stimulation of the LH. Behavioral categories are object directed [grasping, biting, licking of spout, electrode cord, chamber surface, or body], spontaneous tongue protrusions, spontaneous mouth or jaw movements, locomotion [walking or rearing], and aversive reactions [gapes, head shakes, forelimb flails, etc.]

Discussion

These results argue strongly against the hypothesis that animals eat during ESLH because the stimulation activates an hedonic sensation similar to ingesting a small morsel of palatable food. This hypothesis must predict that ESLH would increase the palatability of most foods, whereas the evidence indicates that stimulation does not amplify the hedonic impact of taste. More specifically, it was found that the taste of normally palatable solutions was not enhanced further by ESLH (Figure 4). Instead, aversive reactivity showed an overall enhancement during ESLH (Figure 4). If it is argued that some hedonic process unrelated to food is evoked by ESLH (e.g., a nonspecific hedonic process or a different sensory pleasure), then it would not explain why the elicited behavior is targeted specifically to food. We conclude that stimulation-bound feeding that is directed toward palatable food is unlikely to be mediated by a further hedonic enhancement evoked during ESLH.

Taste-ESLH Interaction

The only exception to the rule that ESLH did not enhance positive hedonics was for a taste that ordinarily is very unpalatable (Figure 5). Concentrated quinine alone elicited higher positive reactivity in the presence of ESLH than in its absence. This hedonic enhancement was not seen for any of the other five sucrose, HCl, or dilute quinine solutions, all of which are preferred over the high concentration of quinine.

The enhancement of aversion by ESLH showed a similar dependence on the natural palatability of the stimulus (Figure 6). Only sucrose and HCl solutions that ordinarily elicit low aversive reactivity became more aversive during ESLH. Solutions that by themselves could elicit higher levels of aversive reactivity, on the other hand, were unchanged in aver-

siveness. In summary, positive and aversive hedonic evaluations of palatability were potentiated by ESLH only when the initial evaluation was very low.

Could Hedonic Potentiation Have Been Masked?

The lack of hedonic enhancement in the case of ESLH is puzzling given the evidence for hedonic substrates within the LH described in the introduction. In light of this puzzle, it is reasonable to ask whether a hedonic potentiation by ESLH might have occurred but been masked by other effects of electrical stimulation, such as motor or sensorimotor activation or stress.

There are at least four lines of evidence, however, against the hypothesis that motor or sensorimotor effects of ESLH masked affective changes in taste reactivity. First, hedonic and aversive reactivity measures remained sensitive to normal palatability differences between sweet and bitter tastes even in the presence of ESLH (Figures 5 and 6). Sucrose elicited relatively high levels of hedonic reactivity and low levels of aversive reactivity, whereas quinine elicited the opposite pattern, regardless of whether or not ESLH was delivered. Second, the activation of motor or sensorimotor processes did not prevent the detection of an ESLH-induced palatability change for stimuli that initially elicited low levels of positive hedonic or aversive reactivity. An enhancement of positive palatability was detected for the concentrated quinine solution, and an enhancement of aversion was detected for solutions of sucrose and of HCl. Third, it is unlikely that a ceiling effect prevented the detection of an hedonic enhancement from the higher sucrose or HCl baselines of positive reactivity. These stimuli were chosen to elicit only moderate baselines of positive hedonic reactivity, which can be elevated above their baselines by manipulations that stimulate ingestion such as benzodiazepine administration (Treit & Berridge, in press; Treit, Berridge, & Schultz, 1987). Fourth, masking of taste reactivity by directly elicited motor or sensorimotor responses seems especially unlikely in this study because no observable orofacial responses were elicited by ESLH in the vacuum reaction chamber where stimulation was delivered without food or taste stimuli (Figure 7). These lines of evidence lead to the conclusion that the best interpretation of the data is the straightforward one: that a hedonic enhancement of taste palatability is not the mediating mechanism of ESLH-induced feeding.

Comparison to Other Appetites

ESLH is nearly unique among appetite inducers in its failure to enhance the hedonic palatability of preferred items. Strong specific appetites for food are produced by 48 hr of food deprivation and for salt after furosemide or deoxycorticosterone administration. Similar depletion states have been shown in human studies to change the affective perception of sweet and salty tastes (e.g., Bertino, Beauchamp, Risky, & Engelman, 1981; Cabanac, 1971). After depletion of body energy or sodium stores in rats, an increase in the hedonic palatability of sucrose (Berridge, in press; Breslin & Grill, 1988; Cabanac & LaFrance, 1989) or NaCl has been dem-

onstrated using the same taste reactivity measures that were used in this study (Berridge et al., 1984; Berridge & Schulkin, 1989; Grill, Schulkin, & Flynn, 1986). Hedonic enhancement is extended even to the targets of conditioned appetites that are created by associative pairings of conditioned stimuli with salt or sucrose (Berridge & Schulkin, 1989; Breslin, Davidson, & Grill, 1987; Delamater, LoLordo, & Berridge, 1986). If hedonic enhancement is a normal component of strong natural appetites, then we can conclude from our current data that ESLH does not induce all of the psychological components of such appetite.

In contrast to more severe food deprivation, the milder caloric depletion induced when animals are deprived of food for only 24 hr does not induce alliesthesia in rats as measured by taste reactivity (Berridge, in press; Breslin & Grill, 1988). This suggests that mild appetitive states can operate through psychological processes that are essentially nonhedonic. It is conceivable that ESLH might also act through these same nonhedonic processes. The voraciousness of eating evoked by ESLH does not suggest that the animals are in a mild food deprivation state, but the lack of hedonic enhancement makes it clear that not all the processes operating during stronger hunger states are evoked by ESLH.

Search for Alternative Explanations

An explanation of ESLH-bound ingestion must possess a number of characteristics. As shown in this study, ESLH can stimulate ingestion, but it does not carry the hedonic enhancement or alliesthesia of strong hunger or salt appetite. The stimulation-bound feeding also lacks homeostasis-appropriate goal specificity (Schallert, 1977; Valenstein, 1970). Animals may develop highly stereotyped preferences for certain foods during stimulation-bound feeding, rejecting other appropriate and palatable foods, and the development of these preferences requires repeated experience with the particular food (Valenstein, 1971). ESLH preferences tend to be targeted toward sensory features of the food that are often irrelevant to their homeostatic value (e.g., food pellets preferred to food powder; greasy texture preferred to moist, etc.). Moreover, it has been shown that the evoked behavior may be switched from feeding to noningestive behaviors by environmental manipulations. Any explanation of these arbitrary and, to a large degree, learned preferences must involve a mechanism that (a) is sufficiently general in direction to embrace a large range of potential targets of behavior and (b) must interact with learning during ESLH experiences to narrow this range into specific stereotyped preferences (Valenstein, 1976). Lastly, the proposed mechanism must be able to reconcile the paradox presented by the well-documented reward properties of ESLH and our present results. In other words, how can a nonhedonic process serve as a reward?

Recent formulations of incentive motivation theory provide the grounds for postulating a psychological component that has all of these features (Bindra, 1978; Dickinson, 1989; Toates, 1986). Although not described explicitly by these authors, this component is implicit in each of their theories of incentive motivation. This component is incentive *salience attribution*: the active assignment of salience and at-

tractiveness to visual, auditory, tactile, or olfactory stimuli that are themselves intrinsically neutral. Salience attribution possesses the qualities of wanting and desiring, but these need to be distinguished from the experience of sensory pleasure.

How might salience attribution operate in incentive motivation? Certain stimuli function as natural rewards or punishers, such as a sweet taste or a cutaneous shock. These stimuli evoke affect the first time they are experienced. They carry motivational significance that is unconditional. The sight of a novel food or a sound that will predict an impending shock, on the other hand, are inherently neutral stimuli which may become positive or negative incentives only by associative pairing with an unconditional stimuli. Once initially neutral stimuli acquire incentive properties, they are no longer affectively neutral: They become attractive and elicit approach, or they become frightening and produce avoidance (see Berridge & Schulkin, 1989; Dickinson, 1989; Mackintosh, 1983; Toates, 1986). In other words, inherently neutral events have acquired salience and value. The hypothesis advanced here is that ESLH selectively activates salience attribution, which is directed by mechanisms of associative learning, but that it does not directly activate the neural substrate of hedonic sensory experience.

Natural Appetite, Stimulation-Bound Behavior, and Salience Attribution

In states of natural appetite, the mechanism of salience attribution is activated together with other psychological components to direct behavior to those stimuli that are relevant to homeostasis. Places and objects associated with food become signals for food and are sought as instrumental goals. Conditioned stimuli for food may become elicitors of feeding and the targets of ingestion themselves (Jenkins & Moore, 1973; Weingarten, 1984). In these cases, salience attribution is directed specifically to stimuli on the basis of their associative history. If salience attribution alone were activated by ESLH, then the resulting incentive facilitation would initially be general. Stimuli in general would become attractive and capable of eliciting behavior of many potential types (Stellar, Brooks, & Mills, 1979). Associative learning mechanisms would require repeated experience with potential targets in order to direct ESLH-elicited salience toward particular stimuli. This feature of salience attribution accounts for why target specificity of stimulation-elicited behavior, when it occurs, often takes time to develop and is influenced by the animal's experience during early trials (Valenstein, 1971).

A second feature that sheds light on stimulation-bound behavior is that salience attribution essentially transforms the perception of visual, auditory, and other senses. It can best be described as a form of perceptual modulation: the attribution of motivational value to the perception of a stimulus that is essentially neutral. This central role of perceptual modulation helps to explain the extraordinary ability of sensory factors to control behavior elicited by ESLH. Unilateral ESLH, for example, may increase responding for food that is signaled to be available through the contralateral eye but not affect responding for food that is signaled to be available through the ipsilateral eye (Beagley & Holley, 1977). Hedonic

or depletion-cue hypotheses of ESLH cannot easily explain such sensory dependence. By the salience attribution hypothesis, a signal seen through the eye contralateral to a stimulating LH electrode is perceived as a salient and attractive food incentive and elicits responding for food. A signal that is relayed to the unstimulated side of the brain is not so perceived and does not elicit feeding.

Reward and Salience Attribution

Incentive hypotheses of motivation also imply a role for salience attribution in instrumental learning and reward. According to incentive theory, when an animal learns that a natural reward is to be found in a particular place or to be gained by manipulating a particular bar, key, or chain, it attributes incentive value to its representation of the place and object as directed by associative learning. Ordinarily, incentive value is attached to an instrumental activity by its association with a hedonic event. The attachment of incentive value ordinarily can be viewed as occurring through a three-stage process of natural reward. First, in encounter with the goal object (e.g., food in the mouth) hedonics are engendered, which triggers the subsequent reward stages. This is the function of neural systems that are true substrates for hedonics. Second, if hedonics are to reward behavior, then a correlation with an associated instrumental act must be registered. This is the role of associative learning. Third, if an associative representation is to alter the course of subsequent behavior, then that action and its eliciting stimulus or representation must actively be attributed with incentive value to elicit approach and to initiate instrumental behavior on future occasions. This final process is the one elicited directly by ESLH. On the first ESLH stimulation, salience attribution alone is elicited. Associative learning mechanisms are closely conjoined to salience attribution, however, and are indirectly recruited when salience attribution is activated. With repeated stimulations this recruitment serves to focus salience attribution on the particular events that were associated with the stimulations.

It is this attribution of incentive salience to the rewarded instrumental act and to its representation that strengthens its subsequent elicitation and leads to repetition of the rewarded act (see Bindra, 1978; Toates, 1986). In "natural" reward, the second and third steps are initiated together by the trigger of a hedonic event. Our results suggest that ESLH circumvents the natural trigger and activates the reward process directly at the level of the third incentive attribution stage (which also recruits the second associative stage) even in the absence of a hedonic experience. In a sense, ESLH would constitute mere "sham reward" (because it would lack the hedonic component of natural reward), but it would mimic hedonic reward in all measures of instrumental behavior. Sham reward and hedonic reward would be indistinguishable in incentive motivation paradigms, but they would appear very different on measures that selectively assessed hedonics, independent of instrumental performance, such as taste reactivity. This interpretation helps make sense of the reports that electrical brain stimulation in humans usually fails to produce an intense hedonic experience even in patients that

self stimulate (Heath, 1972; Sem-Jacobsen, 1976). By our hypothesis, "rewarding" brain stimulation need activate only incentive salience attribution and not the brain substrate of hedonics.

Support From Dopamine Depletion by 6-Hydroxydopamine and Pharmacological Blockade

What might be the neural substrate for salience attribution? At present, the strongest candidate is the mesotelencephalic system of dopamine projections that ascends through the hypothalamus (see Berridge et al., 1989; Blackburn, Phillips, & Fibiger, 1987; Fibiger & Phillips, 1986). The results of a number of studies support the hypothesis that dopamine activation enhances incentives and serves as a reward, yet does not enhance hedonic affect when this is assessed selectively.

Feeding evoked by ESLH requires the participation of dopamine projections (Mittleman et al., 1986; Phillips & Fibiger, 1973; Phillips & Nikaido, 1975; Rowland et al., 1980). ESLH promotes dopamine release, as measured by microdialysis (Hernandez & Hoebel, 1988), and the effectiveness of ESLH at eliciting feeding from rats is correlated directly with individual differences in the rate of sensitization of dopamine forebrain projections (Mittleman et al., 1986). The integrity of dopamine systems has been shown to be crucial also to the reward properties of ESLH (e.g., Wise & Rompre, 1989). Yet in spite of the strong evidence for dopamine mediation of ESLH-elicited behavior, and for a role of dopamine in ESLH reward, our present results indicate that the effects of ESLH on dopamine release are not accompanied by a direct enhancement of hedonics.

Just as the promotion of dopamine release by ESLH potentiates food incentives and induces sham reward (reward without hedonics), so does the disruption of dopamine systems suppress incentive motivation and reward—without decreasing hedonics. Hedonic taste reactivity to sucrose infusions is not impaired by dopamine antagonist administration (Treit & Berridge, in press) at doses that reduce the reward efficacy of food on instrumental responding (e.g., Fouriezos & Wise, 1976). Many studies have documented the ability of dopamine antagonists to alter patterns of food intake, fistulated sham feeding, and ingestive lick bouts in ways that mimic the reduction of sucrose reward produced by dilution or adulteration (e.g., Bailey, Hsiao, & King, 1986; Clifton, Rusk, & Cooper, 1989; Davis, 1989; Geary & Smith, 1985; Smith & Schneider, 1988; Xenakis & Sclafani, 1981). Although these studies have often been interpreted to indicate that dopamine antagonists reduce the hedonic properties of sucrose, it is important to recognize that taste reactivity measures were not used and that the results could be equally well accounted for by a reduction of salience attribution to food incentives. More recently, a demonstrated failure of dopamine agents to alter taste reactivity measures (Treit & Berridge, in press) suggests that the reduction of ingestion by neuroleptics is indeed through changes in salience attribution and not through changes in hedonics. In a more dramatic demonstration, complete aphagia and adipsia follow the

damage of dopamine projections by 6-hydroxydopamine (e.g., Smith, 1976; Stricker & Zigmond, 1976; Ungerstedt, 1971). An analysis of the taste reactivity of such aphagic rats has shown that hedonic taste reactivity is not impaired even in this case (Berridge et al., 1989). This dissociation of hedonics from incentive motivation after dopamine depletion provides the strongest evidence that dopamine systems influence incentive motivation specifically through salience attribution.

Initiation Versus Maintenance

Our discussion of dopamine and salience attribution has focused on the role of this system in initiating behavior. The integrity of dopamine systems has been suggested to be even more important to maintaining motivated behavior once it has been initiated (Wise, 1982). For example, low doses of pimozide increasingly disrupt instrumental performance for ESLH as a session progresses, although early trials of the same session have normal response rates (Fouriez & Wise, 1976). Similarly, a close inspection of the suppression of ingestion of sucrose solutions by D-2 antagonists has shown that these drugs disrupt the maintenance of drinking (i.e., shorten the length of lick clusters during a drinking bout, reduce the number of clusters emitted during a bout, and increase the spacing between clusters of a single drinking bout) without producing a motor impairment of intraclick lick rate (Davis, 1989; Smith & Schneider, 1988). Regarding our hypothesis, the disruption of maintenance suggests that such drugs prevent the continual reboosting of salience attribution to an incentive that normally occurs during a meal or reward. The reboosting appears to be especially vulnerable to neuroleptics and may be suppressed by doses that do not prevent the attribution of preestablished incentive salience. This interpretation is strengthened further by the observations that the disruption of behavioral maintenance by neuroleptics is specific to the environment in which the drug is tested and that switching to a different task may reinstate responding (e.g., Gallistel, Boytim, Gomita, & Klebanoff, 1982). Task specificity can be explained by supposing that neuroleptics have prevented the reboosting of salience attribution to the specific incentive stimuli that are associated with the "extinguished" task.

Final Considerations: Directedness of Stimulation-Bound Behavior

Stimulation-bound behavior is often directed toward natural motivational targets: food, water, prey, sexual partners, and so on. It was this natural directedness that originally gave rise to the hypothesis that ESLH evoked specific natural drives (i.e., homeostatic depletion cues) such as hunger or thirst. This hypothesis still has supporters, despite the evidence described earlier that ESLH-elicited behaviors may differ in significant ways from behavior known to be motivated by a specific drive. The fact that a single animal may show different behavior in response to stimulation by different electrodes has remained the strongest impetus for specific motivation hypotheses (Hallonquist & Mrosovsky, 1986;

Hoebel, 1976). Behavioral specificity of this sort, however rare it might be, poses a challenge to general process hypotheses. How can electrode-specific directedness be explained by the incentive salience attribution hypothesis of ESLH that we propose?

There are a number of sources that might direct ESLH-elicited behavior. Among these are sensory cues arising from ESLH. Peripheral sensations are reported as the single most frequent type of effect during electrical brain stimulation in humans (Sem-Jacobsen, 1976), and facilitation of orofacial sensation is a well-documented effect of ESLH in animals (MacDonnell & Flynn, 1966; Smith, 1972). Orofacial sensation might well bias ESLH-elicited behavior toward targets that would support ingestion, gnawing, licking, and so on. Manipulations of natural motivational variables (e.g., sex hormone removal or supplement, homeostatic depletion or satiety, etc.; Hoebel, 1976) would also be expected to bias behavior elicited by ESLH, but not necessarily through the same mechanism responsible for its evocation (Valenstein, 1973). Instead, motivational variables could bias behavior elicited by ESLH in much the same manner as would sensory or incentive cues. Direct motor effects of ESLH also produce sensory consequences that could bias behavior in other directions (e.g., Sem-Jacobsen's, 1976, report of a woman who experienced "tickling" contractions of pelvic muscles during stimulation). If an electrode activated general incentive salience attribution together with sensory or motor cues, then the evoked behavior might well be biased toward specific forms, but the explanation for this specificity need not lie in a specific motivational state such as hunger or sex. Although it remains possible that specific motivations might be activated by brain stimulation, behavioral specificity by itself does not constitute proof of a specific motive (see von Holst & von St. Paul, 1963).

Most important in directing the specific form of stimulation-bound behavior during activation of incentive salience may be the response tendencies that an individual brings to the situation. "Prepotency" tendencies may exist for a variety of reasons even on the first stimulation trial, but even if they do not, such tendencies will quickly develop during trials with ESLH. Stimulation-bound behavior becomes more stereotyped as the repetition of ESLH strengthens the specificity of salience attribution to the particular target and action. Once established, such stereotypy persists even when the parameters of the eliciting stimulation or when (within limits) the brain targets are changed (e.g., Bachus & Valenstein, 1979; Wise, 1971).

Conclusion

Abundant evidence has demonstrated that ESLH can both reward instrumental behavior and elicit feeding. This linkage between reward and elicited feeding has led to the hypothesis that ESLH evokes an hedonic sensory experience comparable to that which occurs while ingesting a small morsel of palatable food. Despite the plausibility of the hypothesis, our results clearly demonstrate that ESLH does not potentiate hedonic reactions to food.

The paradox that ESLH rewards and elicits behavior with-

out hedonic activation may best be understood within a framework of a salience attribution hypothesis. According to this hypothesis, ESLH elicits behavior by potentiating the attractiveness or incentive salience of external stimuli and their representations, a stage of the process whereby stimuli acquire incentive properties during natural rewards. This hypothesis circumvents the need to postulate that ESLH evokes a specific hedonic taste experience, and it is consistent with the observation that ESLH can be made to evoke different behaviors by environmental manipulations.

Salience attribution and its accompanying eliciting and rewarding qualities appear to be coupled to activity within the mesotelencephalic dopamine system, but dopamine does not appear to mediate hedonic taste experiences. The aphagia and other motivational deficits that result from 6-hydroxydopamine lesions or pharmacological blockade of dopamine projections, occur without accompanying changes in hedonic reactivity.

The hypothesis that dopamine projections mediate incentive salience attribution shares features with a number of other hypotheses that link dopamine projections and ESLH to aspects of incentive motivation (Fibiger & Phillips, 1986) or to "motor approach" functions (Panksepp, 1986; Stellar et al., 1979; Wise, 1987). Salience attribution may be viewed as an extension or as a clarification of such hypotheses. It illuminates the distinction between hedonic and attribution properties of incentive motivation. Salience attribution also highlights the intimate relations between motivation, perceptual processes, and associative learning, and it suggests how these relations might be shared by a variety of phenomena that include, among others, the acquisition of natural food preferences, the elicitation of feeding by ESLH, and the psychological mediation of reward.

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Butcher, Geen, Hulse, and Salthouse Appointed New Editors, 1992–1997

The Publications and Communications Board of the American Psychological Association announces the appointments of James N. Butcher, University of Minnesota; Russell G. Geen, University of Missouri; Stewart H. Hulse, Johns Hopkins University; and Timothy Salthouse, Georgia Institute of Technology as editors of *Psychological Assessment: A Journal of Consulting and Clinical Psychology*, the Personality Processes and Individual Differences section of the *Journal of Personality and Social Psychology*, the *Journal of Experimental Psychology: Animal Behavior Processes*, and *Psychology and Aging*, respectively. As of January 1, 1991, manuscripts should be directed as follows:

- For *Psychological Assessment* send manuscripts to James N. Butcher, Department of Psychology, Elliott Hall, University of Minnesota, 75 East River Road, Minneapolis, Minnesota 55455.
- For *JPSP: Personality* send manuscripts to Russell G. Geen, Department of Psychology, University of Missouri, Columbia, Missouri 65211.
- For *JEP: Animal* send manuscripts to Stewart H. Hulse, Johns Hopkins University, Department of Psychology, Ames Hall, Baltimore, Maryland 21218.
- For *Psychology and Aging* send manuscripts to Timothy Salthouse, Georgia Institute of Technology, School of Psychology, Atlanta, Georgia 30332.