led to a dramatic increase in urine osmolality (+148 percent). Another humoral factor, aldosterone, is also unlikely to be a mediator of the observed sodium retention because of the rapid onset of this response and the fact that potassium excretion did not increase (*14*) but instead decreased in parallel with sodium excretion (−38 percent from a baseline of 58 μEq/min for the high heart rate reactors in the HR group).

Since heart rate responses to this type of task reflect sympathetic activity (*7*), the relation between heart rate response and decreased sodium excretion obtained for HR subjects suggests a common mediation by the sympathetic nervous system. This interpretation is reinforced by the results of studies in the dog (*4*) and the spontaneously hypertensive rat (*5*) demonstrating that stress-induced sodium retention is abolished by infusion of propranolol or by surgical destruction of the renal sympathetic nerves. However, the lack of sodium retention shown by the LR subjects who were high heart rate reactors indicates that cardiovascular and renal sympathetic responses do not always show parallel changes.

Sodium retention mediated by the renal nerves has been linked to hypertension development in the spontaneously hypertensive rat; renal denervation reduces this sodium retention and delays the pathogenic process (*15*). Similarly, long-term increases in BP have been shown to develop in dogs exposed to daily shock-avoidance combined with saline infusion, although neither the stressful task alone nor the saline alone led to any change in baseline BP (*16*). The data for spontaneously hypertensive rats indicate that the tendency to retain sodium has a genetic basis and contributes to hypertension development. The fact that all but one of the men who showed acute sodium retention in the present study had a hypertensive parent leads to the speculation that the tendency to retain sodium during stress may be familial in man as well, and that it may reflect a predisposition to develop hypertension.

Future investigations of human renal excretory responses with the use of sympathetic antagonists such as propranolol should provide more direct evidence of the role of sympathetic activity in mediating stress-induced sodium retention. Ideally, such studies should also assess possible changes in glomerular filtration rate, renal blood flow, plasma renin activity, and vasopressin. Other investigations are also necessary to evaluate the possible relationship between such retention and development of hypertension. These studies could include additional research with animal models during chronic exposure to stress, and also the lengthier but more definitive long-term follow-up investigations in man.

KATHLEEN C. LIGHT
Departments of Psychiatry and Physiology, School of Medicine, University of North Carolina, Chapel Hill 27514
JOHN P. KOEPKE
PAUL A. OBRIST
Department of Psychiatry, University of North Carolina
PARK W. WILLIS, IV
Department of Medicine, University of North Carolina

References and Notes

1. B. Folkow, *Physiol. Rev.* **62**, 347 (1982).
2. A. C. Guyton, T. G. Coleman, A. W. Cowley, R. D. Manning, R. A. Norman, J. D. Ferguson, *Circ. Res.* **35**, 159 (1974).
3. J. De Champlain, L. Krakoff, J. Axelrod, *Circ. Res. Suppl.* **24**, I-75 (1969); L. Tobian, *Arch. Intern. Med.* **133**, 959 (1974); E. Ambrosioni, F. V. Costa, C. Borghi, L. Montebugnoli, M. F. Giordani, B. Magnani, *Hypertension* **4**, 789 (1982).
4. A. Grignolo, J. P. Koepke, P. A. Obrist, *Am. J. Physiol.* **242**, R482 (1982); J. P. Koepke, A. Grignolo, P. A. Obrist, *Fed. Proc. Fed. Am. Soc. Exp. Biol.* **40**, 553 (1981); J. P. Koepke, K. C. Light, P. A. Obrist, *Am. J. Physiol.*, in press.
5. S. Lundin and P. Thoren, *Acta Physiol. Scand.* **115**, 115 (1982).
6. N. K. Hollenberg, G. H. Williams, D. F. Adams, *Hypertension* **3**, 11 (1981).
7. K. C. Light, *Psychophysiology* **18**, 216 (1981).
8. E. Syvalahti, R. Lammintausta, A. Pekkerinen, *Acta Pharmacol. Toxicol.* **38**, 244 (1976).
9. W. E. Miall, *Practitioner* **207**, 20 (1971).
10. S. Julius, in *Hypertension*, J. Genest, E. Koiw, O. Kuchel, Eds. (McGraw-Hill, New York, 1977), pp. 630–639.
11. Borderline hypertension was defined by a mean SBP of 140 mmHg or greater, based on eight readings intermittently above and below 140 obtained with the subject seated during a rest period lasting at least 1 hour (*10*).
12. Information on parental normotension or hypertension was obtained by contacting the parents of the subjects. Of the 80 parents, 76 (95 percent) reported that they had been medically examined during the previous year, and all those reporting hypertension described appropriate diet control or drug treatment. Only two subjects, including one in the stress condition, had two hypertensive parents.
13. K. M. McDonald, P. D. Miller, R. J. Anderson, T. Berl, R. W. Schrier, *Kidney Int.* **10**, 38 (1976).
14. A. C. Barger, R. D. Berlin, J. F. Tulenko, *Endocrinology* **62**, 804 (1958).
15. S. R. Winternitz, R. E. Katholi, S. Oparil, *J. Clin. Invest.* **66**, 971 (1980).
16. D. E. Anderson, in *Circulation, Neurobiology and Behavior*, O. Smith, R. Galosy, S. Weiss, Eds. (Elsevier, New York, 1982), pp. 247–258.
17. This work was supported in part by PHS grants HL-23718, HL-18976, HL-01096, and RR-46. We thank A. Grignolo, W. J. Arendshorst, J. W. Woods, and W. B. Blythe for advice and cooperation.

16 November 1982; revised 4 February 1983

Conditioned Cues Elicit Feeding in Sated Rats: A Role for Learning in Meal Initiation

Abstract. *Pavlovian conditioning was used to teach rats an association between an arbitrary external cue and food. Presentation of the conditioned cue elicited feeding by sated animals. The meal constituted approximately 20 percent of daily intake, and it was compensated for by a reduction of subsequent intake.*

Several hypotheses regarding the physiological mechanisms underlying the initiation of feeding share the assertion that organisms begin to feed in response to a state of energy depletion. The hypotheses differ in the specific physiological events identified as reflecting the state of energy balance and assumed, therefore, to be instrumental in meal initiation (*1*). Recent analyses of food intake control systems indicate, however, that organisms eat not only as a result of energy demands but also in response to other factors not directly associated with energy depletion. Some recent theoretical treatments of human feeding behavior stress that learned social and cognitive factors are critical in determining when an organism will eat and sometimes dominate regulatory signals linked to the state of energy balance (*2*). Little convincing experimental evidence exists, however.

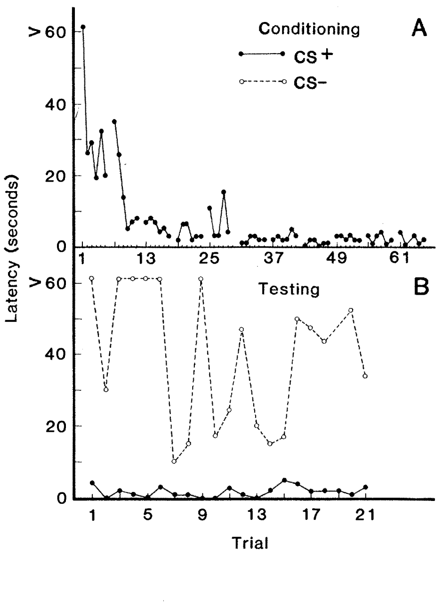

Fig. 1. Group median latency to initiate feeding after the delivery of liquid diet into the food cup (A) during conditioning and (B) during testing.

I have tested the hypothesis that external cues that, through a process of Pavlovian conditioning, have become associated with feeding determine when a meal will take place. Eating elicited by conditioned cues in the absence of any background energy depletion (that is, when animals are sated) would support the hypothesis.

Seven male Long-Evans rats (mean weight, 340 g) were housed individually. The experiment consisted of two phases, conditioning and testing. During conditioning, a 4½-minute conditioned stimulus (CS+), consisting of a buzzer and a light presented together, was presented before every meal. The meal, consisting of 8 ml of a liquid diet based on evaporated milk, was delivered into the home cage during the last 30 seconds of the CS+. Rats received six meals per day with an average intermeal interval of 3.5 hours. A CS− (an intermittent pure tone) was sounded at the midpoint of every intermeal interval and, thus, was not paired with food. The use of a photocell beam located at the front edge of the food cup allowed the time rats spent nosing into the food cup when the CS+ was presented, the latency to feed once the meal was delivered, and the time spent eating to be recorded. This procedure was continued for 11 consecutive days, providing 66 trials of the CS+ paired with food.

Throughout testing, which began the next day, rats fed freely from a continuously available food bottle containing liquid diet. The ability of conditioned cues to affect feeding was assessed by presenting animals once a day with the CS+ followed by delivery of a liquid diet meal. The independent variables mimicked those of conditioning trials.

The effectiveness of Pavlovian conditioning is evident (Fig. 1A). By day 6 of conditioning and on all subsequent conditioning trials, rats initiated eating with a median latency of less than 5 seconds (*3*).

The most critical observations are the feeding responses rats made to the CS+ during testing. The CS+ elicited feeding on all 21 test days even though rats were tested while satiated (Fig. 1B). The median latency, as during conditioning, was less than 5 seconds. The similarity of the feeding response elicited by the CS+ during testing and conditioning was further corroborated by the absence of significant differences between the two phases in the time rats spent nosing into the food dish in anticipation of the meal [$t(6) = 1.29$] and the cumulative feeding time to CS+ presentation [$t(6) = 0.39$].

To examine the stimulus specificity of the CS+-induced feeding response, the CS− was also presented once a day during testing, but was followed by delivery of a meal. Rats responded with delayed and attenuated feeding (Fig. 1B), indicating that the rapid feeding in response to the CS+ did not result from simple arousal.

The same procedures were followed in a second study with seven rats, except that test days were of two types. On nonsignal days, no conditioned meals were elicited. On alternate signal days, a CS+ was presented and followed by 15 ml of liquid diet delivered into the food cup. The diet remaining in the food cup after 15 minutes indicated the size of the meal supported by CS+ presentations. Comparing the 24-hour intake from the free-feeding food bottle on nonsignal and signal days indicated whether rats compensated for signaled meals by reducing their subsequent diet intake from the free-feeding food bottle.

Although rats were sated at the time of testing, presentation of a CS+ resulted in an average intake of 20 ± 2 percent of their 24-hour free-feeding caloric intake (Fig. 2). Further, rats compensated for signaled meals by reducing their food intake from the food bottle: intake on signal days was nearly identical to that on nonsignal days [$t(6) = 1.62$, not significant].

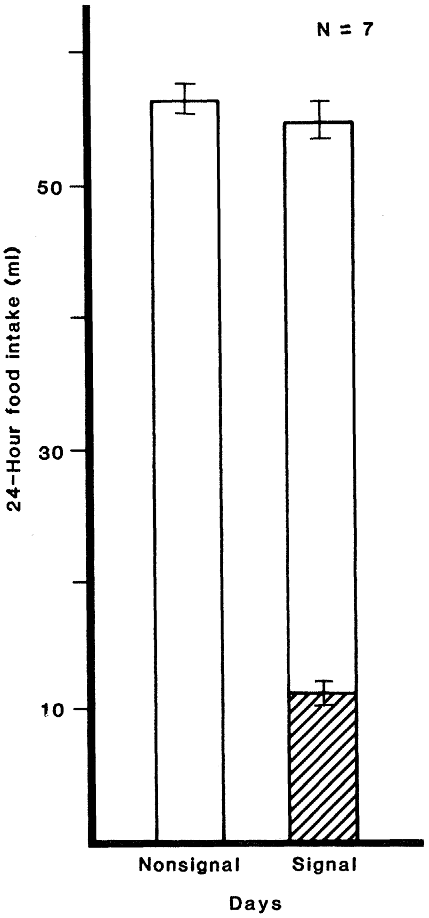

Fig. 2. Daily food intake of rats on nonsignal and signal days. Hatched bar, intake during a signaled meal; open bar, intake from free-feeding food bottle. Vertical lines indicate 1 standard error of the mean.

The results demonstrate that cues that have become signals for food can subsequently initiate a meal. Once such an association has been learned, stimuli retain their ability to influence feeding for protracted periods and even under a state, satiation, that might be expected to minimize the impact of such events. Ingestion supported by conditioned cues can be substantial. In a single conditioned meal, functionally sated rats ingested 20 percent of their total daily caloric intake. Feeding controlled by learned cues also seems to represent part of the natural feeding repertoire of these animals in that they compensated for the calories ingested at signaled meals by reducing later food intake. These findings of a role for learned cognitive factors in the instigation of feeding are consistent with the anecdotal descriptions and introspections concerning meal initiation in humans. The data also provide an empirical demonstration of this phenomenon as well as an animal model for further investigations.

Although the results do not deny a role for regulatory energy depletion signals in the control of eating, they suggest that a complete theory of meal initiation must incorporate the concept of learned controls and not rely totally on energy depletion as the only determinant of ingestion.

Many questions remain. One concerns the physiological mechanism mediating the effects of conditioned cues. Evidence that learned cues elicit vagally mediated release of digestive secretions (*4*) may also indicate that elaboration of neural anticipatory visceral responses underlies feeding in response to conditioned cues. If so, truncal vagotomy should severely disturb the initiation of feeding controlled by learned cues and, more generally, should affect the experience of hunger; attenuation or abolition of hunger sensations have been reported by humans after truncal vagotomy (*5*).

A second issue concerns the potential role of externally controlled feeding in the development of obesity. Normal animals compensate for cue-elicited feedings by reducing their later intake. If obese organisms, or those predisposed to obesity, fail to compensate, as some hypotheses suggest (*6*), persistent responding to conditioned cues would result in positive energy balance and obesity.

HARVEY P. WEINGARTEN
Department of Psychology,
McMaster University,
Hamilton, Ontario, Canada L8S 4K1

References and Notes

1. J. LeMagnen, *Behav. Brain Sci.* **4**, 561 (1981); J. Mayer, *Ann. N.Y. Acad. Sci.* **63**, 15 (1955).
2. J. Bellisle, *Neurosci. Biobehav. Rev.* **3**, 163 (1979); D. A. Booth, *Br. Med. Bull.* **37**, 135 (1981); J. Davis, *Behav. Brain Sci.* **4**, 580 (1980); J. Rodin, *Am. Psychol.* **4**, 361 (1981).
3. When the CS+ was presented, and before food was delivered, rats nosed into the food cup in anticipation of the meal. In contrast, rats learned to ignore the CS− and did not nose into the food cup when it was presented.
4. H. P. Weingarten, *Physiol. Behav.* **27**, 217 (1981); S. C. Woods, J. R. Vasselli, E. Kaestner, G. A. Szakmary, P. Milburn, M. V. Vitiello, *J. Comp. Physiol. Psychol.* **91**, 128 (1977).
5. L. R. Dragstedt, W. L. Palmer, P. W. Schafer, P. C. Hodges, *Gastroenterology* **3**, 450 (1944); J. E. Kral, *Am. J. Clin. Nutr.* **33**, 416 (1980).
6. S. Schachter, in *Neurophysiology and Emotion*, D. C. Glass, Ed. (Rockefeller Univ. Press, New York, 1967), p. 117; S. Schachter and J. Rodin, *Obese Humans and Rats* (Erlbaum, Hillsdale, N.J., 1974).
7. This research was supported by grant funds from the Medical Research Council of Canada and the Natural Sciences and Engineering Research Council of Canada. I thank C. Malinski for assistance with the experiments and Dr. Shepard Siegel for a critical reading of the original manuscript.

12 August 1982; revised 26 November 1982

Behavior of Antarctic Krill, *Euphausia superba*: Chemoreception, Feeding, Schooling, and Molting

Abstract. *Krill do not feed by passive, continuous filtration but use area-intensive searching and various rapid feeding behaviors to exploit local high food concentrations. Chemicals alone at low concentrations, not particles, trigger feeding. Krill form dense schools that move rapidly and migrate primarily horizontally. Abrupt disruption of a school can trigger mass molting, and molts may act as decoys.*

Euphausia superba is the dominant herbivore of the Southern Ocean. Today, in the virtual absence of southern baleen whales, the standing stock of "unutilized" krill is purported to equal the combined world harvest of all other marine species (*1*). Despite extensive investigation of krill (*2*), little is known of their behavior at sea or winter biology. In January and February 1982, we observed individual *E. superba* in aquariums at Palmer Station on Anvers Island off the west coast of the Antarctic Peninsula, and we used scuba to observe behavior of schools at sea (*3*). We describe a series of complex behavior patterns that may alter assumptions about euphausiid biology.

Euphausia superba feeds primarily on phytoplankton. Feeding experiments in laboratory flow chambers (*4*) showed that *E. superba* feeds episodically on phytoplankton and is not a continuous filter feeder. When not feeding, *E. superba* swims rapidly, pressing its six pairs of thoracic endopodites together to form a keel (Fig. 1a). When feeding, the euphausiid throws these legs out to enclose a packet of water and algae in a basket that closes ventrally and anteriorly. With the basket fully expanded, setae of adjacent endopodites overlap, forming narrow suture zones. When feeding begins the thoracic exopodites stop their metachronic beating and pronate 90° to flatten laterally against the suture zones (Fig. 1b). The dactyls fold back and also overlap the long setae of the endopodites. The result is a basket that is watertight, which was determined by adding fluorescein dye and phytoplankton to water presented to the euphausiid. Algae collect inside the basket on the setae while the euphausiid expels water by rapidly compressing the endopodites. The exopodites, like flapper valves, lift together in a horizontal direction as water is forced laterally through the setae in parallel jets. The feeding bout is repeated one to five times per second. After the mouthparts gather a food bolus the feeding bouts cease, the mandibular palps press the bolus against the mandibles, and ingestion ensues.

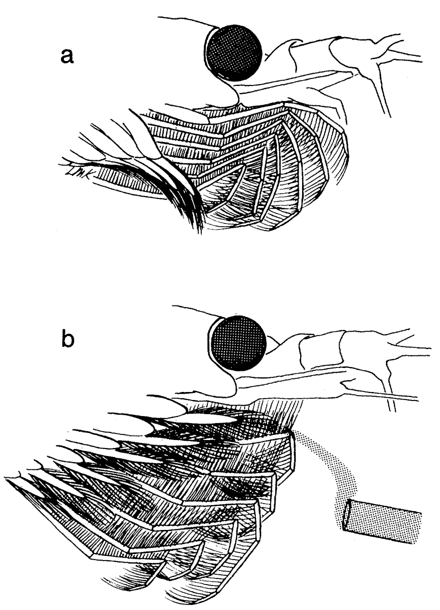

Fig. 1. Deployment of thoracic legs in *Euphausia superba*. Drawings were traced from photograph; intersetal distances are not to scale. (a) Swimming posture: *E. superba* presses endopodites tightly together into a streamlining keel; beating exopodites assist propulsion and steering. (b) Feeding posture: Opening basket draws in water and particles anteriorly; at full extension, ventral and anterior closure, folded dactyls, and exopodites flattened against suture lines between overlapping setae of adjacent endopodites make the expanded basket watertight.

We report that *E. superba* feeds in response to chemical cues. We tested 20 tethered *E. superba* with different concentrations of substances available at Palmer Station and used four or five individuals for each test (*5*). The lack of response to Miocene and Recent diatom skeletons indicates that particles alone do not trigger feeding. In the absence of particles, chemicals alone did elicit the full feeding bout, with a threshold concentration of $10^{-5}M$ for histidine, lactic acid, citric acid, and acetic acid.

In aquariums and in slow-swimming schools at sea individuals occasionally opened the thoracic basket briefly, presumably to test for the presence of food. When they encountered food in the aquarium, the euphausiids swam fast and in tight circles, rapidly filling the feeding basket. When a food bolus accumulated, feeding stopped and the euphausiid swam away to process the food. Area-intensive foraging occurred at the surface also in response to phytoplankton or attractive chemicals floating in local high concentrations. Krill then rolled upside down against the surface, made rapid spinning turns, and quickly swept the surface with the feeding legs. Area-intensive foraging permits rapid food acquisition, indicating that grazing rate calculations made with the assumption of continuous filtration at low food concentration are probably not correct. Feeding behavior of other herbivorous euphausiids should also be reexamined.

In another mode of surface feeding individuals swim forward horizontally, just beneath the surface, and hold one branch of each antennule out of the water. Floating particles are flicked out of the surface film for inspection and sometimes eaten. A school of *E. superba* fed this way at dawn in the Gerlache Strait during a flat calm in February 1982. Thousands of antennules protruded through the surface, leaving tiny wakes on the flat water as the school swam slowly forward.

Another feeding mode, studied only in the laboratory, has implications for winter biology of polar euphausiids. We concentrated and refroze algae that live attached to sea ice into blocks which were floated in the aquarium. As the algae softened, *E. superba* beneath the ice located the food by an area-intensive search. They then fully extended the dactyls of the thoracic endopodites and raked algae into the food basket, working along the submerged surfaces or