phosphates obtained after alkaline hydrolysis of the product. Almost all the radioactivity (88 percent) was recovered in adenosine monophosphate; whereas terminal nucleosides would be hydrolyzed to adenosine.

The lack of inhibition of the enzyme activity by treatment with ribonuclease or by addition of exogenous RNA (Table 1) indicates that the bulk of the mitochondrial RNA does not act as a primer. Initial experiments, however, suggest that poly(A) itself can act as a primer, as has been reported for the corresponding nuclear enzyme (*6*).

Because tumors contain fewer mitochondria per cell (*8*) and consequently less mitochondrial protein per gram of wet tissue than do control tissues (*9*), they can be expected to show relatively low poly(A)-synthesizing activity. However, the low enzyme activity of hepatomas (only 1 to 2 percent of that in normal liver) cannot be explained on this basis. Furthermore, enzyme activity has been expressed per milligram of protein. The loss of enzyme activity in tumors cannot be due to adenosine triphosphatase that may be present in the enzyme preparation, because (i) Ca^{2+}, which is required for the nucleotidase activity (*10*), was completely removed by exhaustive dialysis, and (ii) additional ATP included in the reaction mixture did not increase the specific activity of the hepatoma enzyme (Table 1).

The tumor enzymes could have been associated with an inhibitor or inhibitors of poly(A) polymerase. To test this possibility, we conducted two sets of experiments. First, the liver enzyme was incubated together with each tumor enzyme under optimal assay conditions. Inhibition of liver enzyme activity was not significant (about 20 percent), which demonstrates the absence of any potent inhibitors in the tumor enzyme. In the second experiment, the liver enzyme was incubated for 30 minutes, and the product formed was further incubated for 20 minutes with tumor enzyme. Again, there was no significant change in the liver enzyme activity (less than 20 percent). In these experiments, 0.3 to 0.6 mg of tumor enzyme protein was used.

The experiments reported here show that transformation of parenchymal cells into hepatoma cells results in a considerable loss of mitochondrial poly(A) polymerase. Further experiments with other normal tissues and with growing tissue such as regenerating liver are required to prove unequivocally whether the relative lack of this enzyme in hepatomas is unique to malignant cells.

Unlike nuclear poly(A), which is involved in the transport of messenger RNA (mRNA) from the nucleus to the cytoplasm (*11*), the function of mitochondrial poly(A) is not known. Hence, the significance of the lowered activity of this enzyme in hepatomas cannot be assessed. Since mRNA containing poly(A) segments can be retained on Millipore filters in the presence of 0.5*M* KCl (*7, 12*), it is possible that liver mitochondrial mRNA, as compared to mRNA of hepatomas, contains larger segments of adenylate residues at the 3′ terminal end.

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12 June 1972; revised 28 August 1972

Hypothalamic Norepinephrine: Circadian Rhythms and the Control of Feeding Behavior

Abstract. *The time of day is a decisive determinant of the effects of* l-*norepinephrine on feeding behavior. During the dark, direct application of* l-*norepinephrine to the hypothalamus of rats suppressed feeding behavior. During the light, treatment with the same dose of* l-*norepinephrine facilitated feeding behavior. Thus,* l-*norepinephrine has dual and opposite effects on feeding behavior. A hypothalamic substrate that fluctuates in a circadian rhythm could account for both actions of* l-*norepinephrine.*

The addition of exogenous *l*-norepinephrine (*l*-NE) to the lateral hypothalamus affects feeding behavior. Both stimulant (*1*) and suppressant (*2*) effects have been reported, but the conditions that determine when each of these opposite actions will occur are unknown. This has led to the development of a controversy between proponents of the noradrenergic-feeding and the noradrenergic-satiety theories. The implications of each theory have been reviewed by Hoebel (*3*). We now report data that appear to resolve the controversy. The effects of the addition of exogenous *l*-NE to the hypothalamus appear to be dependent on differences in the internal state of the hypothalamus associated with the environmental cycle of darkness and light. In the dark, this treatment suppressed feeding behavior. In the light, the same dose of

l-NE applied to the same hypothalamic site facilitated feeding behavior. The type of light-dark cycle and the time of drug administration generally have been omitted in the psychological literature on rodent feeding behavior. Some information, however, is available about these important variables (*1, 2*). Thus, it is possible to evaluate earlier experiments in terms of present results.

Eight male albino rats (Charles River) at a body weight of 300 g were adapted to a reversed light-dark cycle with cool white fluorescent lights turned off automatically at 0740 hours E.S.T. and on at 1940. The rats were housed in automated housing (Environmental Sciences) and given free access to pellets of Purina Laboratory Chow and filtered tap water. Three to four days a week they were placed in Plexiglas chambers in the dark at 1030 hours for a period of 1 hour. The chambers contained a full burette of milk (undiluted Pet condensed milk) and a full burette of filtered tap water. Both liquids were at room temperature. Each burette was covered by a Plexiglas guard and contained a stainless steel wire that was connected to a contact-sensitive relay, designed to record the number of licks made by the rat. We used printout counters and cumulative recorders to monitor the rate of the licks. Spillage of milk and water was collected in petri dishes, which allowed total consumption of milk and water to be calculated at the end of the hour for each rat. In addition, the rats were weighed before and after the feeding test. Stabilization of milk-licking behavior developed within 2 to 3 weeks. Some water-licking behavior also occurred but was negligible in quantity. We then anesthesized the rats with pentobarbital and surgically implanted bilateral cannulas that were aimed for the lateral hypothalmic site, as in (*2*). Several weeks were allowed for recovery from surgery and stabilization again of milk-licking behavior. *l*-Norepinephrine hydrochloride was administered by means of duplicate inner cannulas that were weighed on a Cahn electrobalance (G-2) before and after they were loaded with the powdered drug. Immediately prior to the milk-licking test, empty cannulas were removed from the brain and immediately replaced with cannulas that had been loaded with a 25 μg dose of *l*-norepinephrine hydrochloride (*4*). All drug treatments were preceded by at least one control day, and the drug treatments did not occur more often than once each week. No drug treatment was made unless the rats were fully stabilized from the prior treatment. Insertions of empty inner cannulas were without effect on feeding behavior.

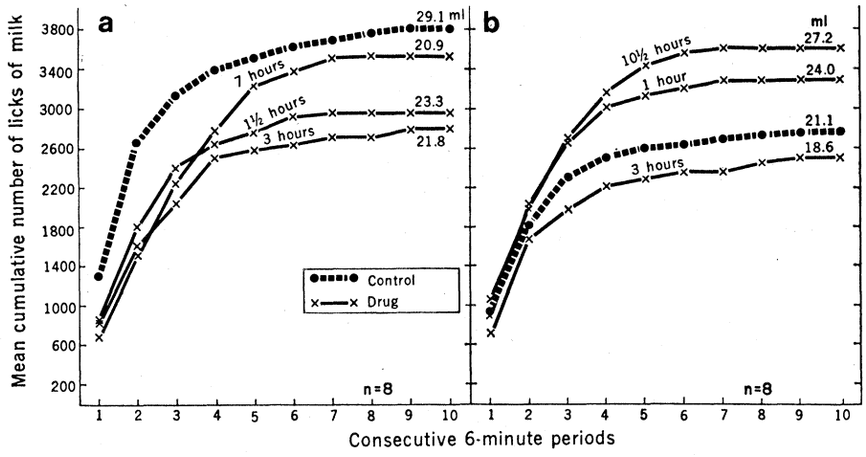

Fig. 1. Effects of direct bilateral application of *l*-norepinephrine (25 μg) to the lateral hypothalamus at various times of the day, on mean cumulative number of licks of milk and mean total intake of milk in milliliters. The number of hours between the beginning of the light (b) or dark (a) period and the start of the drug test is indicated on each drug curve. Control scores were obtained on the day prior to treatment with *l*-norepinephrine.

All tests during week 1 occurred about 3 hours after the onset of darkness at 1030 hours. Treatment with *l*-NE produced a suppression of mean intake of milk and mean number of licks of milk from the control levels prior to treatment with *l*-NE (Fig. 1a). Both suppressions were statistically significant ($t = 2.00$, d.f. $= 7$, $P < .05$ for intake and $t = 3.27$, d.f. $= 7$, $P < .01$ for number of licks). None of the subsequent control tests prior to drug treatment in the dark differed significantly from the first control or from each other. All of these control results were averaged.

During week 2, tests were made approximately 3 hours into the light at 2230 hours. This and subsequent control tests prior to drug treatment in the light did not differ significantly from each other and also were averaged. The control in the light shows less mean intake of milk and fewer mean licks of milk in comparison to the control in the dark (Fig. 1, a and b). Both decreases were statistically significant ($t = 2.56$, d.f. $= 7$, $P < .025$ for intake and $t = 3.28$, d.f. $= 7$, $P < .01$ for number of licks). The suppressant effects of light on rodent feeding behavior are well known (*5*). Treatment of the hypothalamus with *l*-NE at 3 hours into the light had no statistically significant effect on mean intake of milk or mean number of licks of milk ($t = 1.06$, d.f. $= 7$, $P > .05$ for intake and $t = 0.63$, d.f. $= 7$, $P > .05$ for number of licks). In weeks 3 and 4, treatments with *l*-NE were made 1 and 10½ hours into the light. In both cases (Fig. 1b) *l*-NE increased milk intake and milk licking of the rats significantly from the controls in the light ($t = 1.93$, d.f. $= 11$, $P < .05$ and $t = 2.50$, d.f. $= 7$, $P < .025$, respectively for intake; $t = 3.59$, d.f. $= 11$, $P < .005$ and $t = 2.40$, d.f. $= 7$, $P < .025$, respectively for number of licks). Individual controls prior to drug treatment are in Table 1 for each of the three drug treatments administered in the light.

In weeks 5 and 6, the rats were tested during darkness (Fig. 1a), and *l*-NE once again significantly suppressed both mean intake of milk and mean number of licks of milk (at 1500 hours, about 7 hours into the dark, $t = 2.06$, d.f. $= 7$, $P < .05$ for intake and $t = 3.06$, d.f. $= 7$, $P < .01$ for number of licks; and at 0900 hours, approximately 1½ hours into the dark, $t = 3.44$, d.f. $= 7$, $P < .01$ for intake and $t = 1.12$, d.f. $= 7$, $P > .05$ for number of licks. The reinstatement of the suppressant effects of *l*-NE in the dark indicates that the long-term series of treatments with NE did not measurably affect the final behavioral response to *l*-NE. Therefore, it is unlikely that drug-induced irritations or lesions are responsible for the opposite effects of *l*-NE on feeding behavior. Individual mean control scores prior to drug treatment are shown in Table 1 for each of the three drug treatments given in the dark.

The role of circadian rhythms in the neurochemical control of feeding be-

havior can no longer be ignored. Rats are nocturnal animals. Their feeding pattern is circadian. When they are maintained on a 24-hour, light-dark cycle, aproximately 80 percent of their total food intake occurs in the dark. The main feeding period begins at the same time each night, with clocklike regularity. The feeding rhythm remains circadian after the animals are blinded or if they are kept in continuous light, but the onset of the feeding period begins to drift by a fixed number of minutes each day. These experiments reveal an internal clock that runs somewhat faster or slower than 24 hours per day. This indicates that the circadian feeding rhythm is endogenous; that is, it is synchronized but not driven by the environmental light-dark cycle (*5*). Hypothalamic lesions eliminate all manifestations of the endogenous circadian feeding rhythm (*6*). The hypothalamus may contain parts of an oscillation system that drives the feeding rhythm.

Two circadian rhythms of *l*-NE have been identified in the rat hypothalamus. One of these rhythms, present in the anterior hypothalamus, has a peak in the middle of the daily dark period and low points throughout the light period. The second rhythm, present in the posterior hypothalamus, also reaches a peak in the middle of the dark period and reaches its lowest point at the end of the dark period (*7*). It is not known if either of these rhythms is endogenous. In cats, however, it has been established that the circadian rhythm of *l*-NE in the anterior hypothalamus is endogenous. It persists in constant light (*8*). Interestingly, *l*-NE appears to act as a synaptic transmitter in control of circadian rhythms of pineal hydroxyindole-*O*-methyltransferase (*9*) and liver tyrosine aminotransferase (*10*). It has been proposed that the liver aminotransferase rhythm may be generated, in part, by the periodic release of *l*-NE in the periphery (*10*). Perhaps the periodic release of *l*-NE in the anterior hypothalamus serves a similar function and participates in the generation of the feeding rhythm. However, there is no direct evidence for *l*-NE or any other hypothalamic substance as a part of the circadian oscillator for feeding behavior.

How can the same dose of a synaptic transmitter substance that suppresses feeding behavior during the dark, facilitate it in the light? Different internal states of the hypothalamus produced by dark and light may be responsible for the opposite effects. The concentration of endogenous *l*-NE in the anterior hypothalamus reaches its lowest value in the light (*7*). Perhaps in this state, the addition of exogenous *l*-NE would increase endogenous concentrations. However, this would not be a large increase, even with the high doses used in our study, because of the extensive catecholamine uptake mechanisms in the hypothalamus (*11*) and because of the presence of the enzyme monoamine oxidase, which would degrade much of the absorbed *l*-NE (*12*). We predict that a shift in a hypothalamic substance such as *l*-NE from low to medium concentrations may be responsible, in part, for the shift in feeding behavior from the relatively anorexic condition associated with light to the normally hungry condition produced in the dark. We view the phenomenon of noradrenergic elicited feeding as a transient shift from an anorexic state induced in the light to a higher level of feeding that normally occurs in the dark. Our results suggest that low concentrations of hypothalamic *l*-NE may be an essential prerequisite for the demonstration of the facilitatory effects of *l*-NE on feeding behavior.

Table 1. Mean control scores prior to treatment with *l*-norepinephrine. Number of rats is indicated in parentheses.

Time of test (hours from onset)	Mean licks of milk (No.)	Mean intake (ml)
	Dark	
1½ (8)	3812	30.1
3 (8)	3676	30.5
7 (8)	3760	26.7
	Light	
1 (8)	2750	20.6
3 (8)	2309	19.3
10½ (12)	3270	23.6

The concentration of hypothalamic *l*-NE and other biogenic amines also can be lowered by lesions of the lateral hypothalamus (*13*). Exogenous *l*-NE enhances the recovery of feeding behavior when administered intraventricularly to anorexic rats recovering from such lesions (*14*). Thus, both lateral hypothalamic anorexia and light-induced anorexia are reversed by treatment with *l*-NE. Both forms of anorexia may be related to the excessively low concentrations of hypothalamic *l*-NE.

The concentration of *l*-NE in the anterior hypothalamus reaches its highest concentration in the dark. In this state, the addition of exogenous *l*-NE would be expected to raise endogenous concentrations, but again the increase would be small. We propose that a shift of a hypothalamic substance, such as *l*-NE, from medium to high concentrations may be responsible, in part, for the shift from normal hunger to satiety. Results with phentolamine, an agent that prevents endogenous *l*-NE from occupying alpha-adrenergic receptors in the periphery, provide support for this proposal. Direct application of phentolamine to the lateral hypothalamus in darkness blocked the effects of satiety on feeding behavior, as indicated by intense overeating (*2*). This suggests that certain forms of obesity may be related to a failure to generate concentrations of *l*-NE high enough to suppress feeding.

In conclusion, we find that treatment of the lateral hypothalamus with *l*-NE has opposite effects on feeding behavior that depend on the time of day the drug was administered. During the dark, treatment with *l*-NE suppressed feeding behavior. During the light, treatment with the same dose of *l*-NE facilitated feeding behavior. Sufficient evidence exists that the neurochemistry of the hypothalamus is not a steady state, but fluctuates according to a variety of rhythms (*15*). Circadian differences in a hypothalamic substance, such as *l*-NE, may provide the basis for the dual and opposite actions of exogenous *l*-NE on feeding behavior. These findings appear to resolve the controversy between the noradrenergic-feeding and the noradrenergic-satiety theories.

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particularly in comparison to the total amount of *l*-NE found in the hypothalamus (about 0.1 μg). It is possible to suppress or stimulate feeding behavior with doses of *l*-NE as low as 5-μg. Quantities below 5 μg fail to affect feeding behavior. Possibly, these quantities are too small to influence sufficient postsynaptic receptor sites. Uptake mechanisms on the surface of vascular, glial, and neuronal cells remove *l*-NE from the extracellular hypothalamic space. These mechanisms, in conjunction with intracellular monoamine oxidase, may reduce substantially the amount of exogenous *l*-NE that penetrates to the synaptic receptor sites.

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9 May 1972; revised 24 August 1972

Plasma Testosterone Levels in the Male Rhesus: Influences of Sexual and Social Stimuli

Abstract. *Four adult male rhesus monkeys were provided access individually to a group of receptive females. Each male assumed dominance and engaged in frequent copulations. Plasma testosterone levels increased two- to threefold during this period. Next, each male was subjected to sudden and decisive defeat by a large all-male group, and plasma testosterone fell following this experience. Two males were later reintroduced to the females, and plasma testosterone rose rapidly to the previous elevated levels.*

It has been reported that plasma testosterone in male rhesus monkeys living in social groups is correlated with dominance rank and frequency of aggressive behavior (*1*). It was not clear whether the increased levels of testosterone observed in more dominant or aggressive males preceded the differences observed in social behavior, or whether the differences in testosterone were a reflection of the effect of the social environment. Did subordinate animals with lower frequency of aggressive behavior have lower testosterone levels because of their rank and relationship to the other animals, or was testosterone secretion relatively stable and thus functioning in some way to influence the animal's behavior and social rank? The present study was undertaken to see if alterations in the animal's social environment would affect plasma testosterone levels.

By systematically manipulating the social environment for four adult male rhesus monkeys over a 4-month period, we observed both elevations and depressions in plasma testosterone. When males were provided access to sexually receptive females, testosterone levels increased, and when they were briefly exposed to a large group of males, resulting in sudden and profound defeat, testosterone levels fell.

The studies took place in large outdoor compounds, approximately a third of an acre (about 0.13 ha) in area. Plasma testosterone was measured by a modification of the protein-binding technique of Mayes and Nugent (*2*), and social interactions were scored with the use of a standard behavioral inventory. The first study was on the effect of access to sexually receptive females. This occurred in three phases, each lasting 2 weeks: (i) individual cage; (ii) access as the only male to a group of adult females ($N = 13$), some in estrous; and (iii) return to cage.

Access to a group of females provided a mixed stimulus. Males became the alpha, or dominant, animal, along with engaging in frequent sexual behavior (for example, sex present, hip-touch, mount). The social interactions during the first hour after the males were placed with the females are shown in Table 1. Throughout the hour, males displayed frequent noncontact aggression, usually in the form of threat or chase (26 to 29 percent of all behaviors observed). They received frequent submissive behavior (for example, avoidance, grimace, squeal, crouch) from the females (48 to 55 percent), and by the end of the hour received no aggression (contact or noncontact) from the females. Contact aggression (for example, bite, hit, slap, pull) was relatively infrequent and was rare throughout the period with the females. Receiving frequent submissive responses and no threats from the females reflected the males' assumption of dominance status following their introduction to this new group. In the first 20 minutes, sex behavior accounted for 23 percent of all social interactions, and rose to 46 percent by the end of the hour. Grooming and sex behavior continued to be the most frequent forms of social interaction between the males and the females during the following 2 weeks.

Plasma was drawn for testosterone analysis from 0900 to 1000 hours to minimize the effects of diurnal varia-

Table 1. Behavior responses scored during the first and last 20 minutes of the hour after male monkeys were introduced to the female group and the male group. The values represent the various behaviors as a percentage of total behavior.

Response	Introduction to females		Introduction to males	
	First 20 min	Last 20 min	First 20 min	Last 20 min
Does				
Contact aggression	5	2	39	18
Noncontact aggression	26	29	11	3
Submission	9	6	44	76
Sex	23	46	0	0
Other	37	17	6	3
Receives				
Contact aggression	2	0	49	32
Noncontact aggression	11	0	23	15
Submission	48	55	3	0
Sex	14	14	0	3
Other	25	31	25	50