All unmated females over 8 days old adopted the pheromone-release posture when subjected to the dark-light transition irrespective of the hour of subjective night. All the males responded to the dark-light transition by flying and fluttering. We obtained effective copulations from animals brought into light at all hours of their subjective night.

Mated females do not adopt the pheromone-release posture at any time during the period from first copulation to the production of the first ootheca. After the first ootheca has been produced, a percentage of females start secreting again, and a further number secrete again after the production of the second ootheca (*12*).

Dawn mating may well be the result of a compromise between conflicting selection pressures. Mating mantids are almost certainly particularly vulnerable to predation. They cannot maximize their primary defense by adopting the species-specific cryptic posture nor can they make startle displays for secondary defense. Males fly weakly and must be particularly vulnerable when flying to females. Mating at dawn may reduce the risks from predation because it occurs at a time when visually hunting predators are not active. In Panama, although the birds are stirring at this time they are not foraging. Insectivorous primates start feeding even later than birds (*13*). Nocturnal mating might be even safer than dawn mating, but could be impossible in this case because the final movement of the male onto the female appears to be mediated by visual cues.

We think that the problem experienced by other workers in obtaining mantid matings (*7*) could be due to undetected narrow periodicities of sexual activity such as those described herein. Postures similar to the pheromone-release posture that we have described have been described for other species (*7*). We think that the whole system deserves further research.

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10. The males were separated from the females by three layers of screening: (i) fiberglass insect screening on the cage wall, (ii) a hardware-cloth screen between the rack and the cage, and (iii) the hardware-cloth walls of the individual mantis cages. The rack was placed 1 m from the wall of the experimental cage.
11. In a room approximately 3 m square, illuminated by one 48-inch daylight fluorescent tube and two small shaded windows. Incident light [measured as described in (*9*)] was 12 foot-candles.
12. After mating, all females stopped secreting; but 36.6 percent started again after producing the first ootheca, 16.6 percent after producing the second, and 26.6 percent after producing the third; 20.2 percent were still not secreting after producing the third ootheca ($N = 30$).
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α-Melanocyte-Stimulating Hormone: Reduction in Adult Rat Brain After Monosodium Glutamate Treatment of Neonates

Abstract. *Intraperitoneal injection of monosodium glutamate in neonatal rats resulted in a 90 percent loss of α-melanocyte-stimulating hormone in hypothalamic and extrahypothalamic areas of the brain, whereas the amount of hormone in the pituitary gland did not change. The dramatic reduction of α-melanocyte-stimulating hormone in the brain suggests that its primary source there is the neuronal perikarya of the arcuate nucleus.*

Certain acidic amino acids, such as glutamic, aspartic, cysteic, cysteine sulfinic, and homocysteic acids, are both neuroexcitatory (*1*) and neurotoxic (*2*). Glutamate, the most widely studied of these amino acids, is routinely used in electrophysiological studies to artificially induce neuronal firing, whereas the monosodium salt of glutamate (MSG) is commonly used as a dietary additive. Neuronal degeneration induced by MSG or glutamate has been demonstrated in primates (*3*), hamsters (*4*), guinea pigs (*5*), rats (*6*), and mice (*7*). Neuronal destruction in the brain after systemic administration of MSG is apparent in areas where the blood-brain barrier is leaky—the circumventricular organs (CVO) and contiguous structures (*8*).

The arcuate nucleus of the mediobasal hypothalamus, a region contiguous with the median eminence (a CVO) and one that accumulates subcutaneously administered MSG (*9*), is particularly vulnerable to the toxic effects of MSG. Since the integrity of the mediobasal hypothalamus is essential to normal endocrine function, mature animals treated neonatally with MSG manifest a variety of neuroendocrine deficiencies (*4, 10, 11*).

In the mediobasal hypothalamus, systems containing monoamines (*11*), acetylcholine (*11, 12*), and γ-aminobutyric acid (*12*) have been implicated in the etiology of the MSG-induced endocrine deficits. However, systemic administration of MSG has no effect on hypothalamic regulatory peptides, such as luteinizing hormone-releasing hormone (LHRH), thyrotropin-releasing hormone (TRH), and somatostatin (*11, 13*), in the region of the arcuate nucleus and median eminence. Since MSG is taken up by dendritic and somal membranes but not by axons passing through a region (*14*), the lack of effect of MSG on levels of TRH, LHRH, and somatostatin in the arcuate region suggests that these peptides originate in neuronal perikarya outside of the arcuate-median eminence region. Appropriate doses of MSG destroy 80 to 90 percent of the neuronal cell bodies in the arcuate region. Inasmuch as an important source of α-melanocyte-stimulating hormone (α-MSH), one of several melanotropic peptides in the brain (*15*), is neuronal perikarya of the arcuate nucleus (*16*), we measured by radioimmunoassay the effect of neonatally administered MSG on hypothalamic and extrahypothalamic levels of α-MSH in adult rats.

Neonatal rats of the Zivic-Miller strain received an intraperitoneal injection of MSG (4 mg per gram of body weight) on alternate days throughout the first 10 days of life (*17*). An equal volume of 0.9 percent NaCl was injected in control animals. At 60 days of age, animals were decapitated and their brains were rapidly excised. Brains were sliced with the aid of an ice-chilled Plexiglas holder and

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Table 1. Effect of neonatal administration of monosodium L-glutamate (MSG) on α-MSH levels in adult rat brain. Means and standard errors are given for α-MSH levels in tissue obtained from three animals; *P*, probability that the concentrations of α-MSH from MSG-treated and saline-treated animals are the same; N.S., difference not significant.

Region of brain	α-MSH (pg per mg of wet tissue)		*P*	α-MSH (pg per region)	
	MSG-treated	Saline-treated		MSG-treated	Saline-treated
Arcuate nucleus	30.7 ± 4.3	218.6 ± 24.8	< .001	55.4 ± 12.7	639.4 ± 88.9
Dorsal hypothalamus	8.7 ± 0.9	95.8 ± 3.2	< .001	145.1 ± 17.6	1980.2 ± 80.4
Preoptic–anterior hypothalamus	14.4 ± 5.4	105.5 ± 1.7	< .001	115.3 ± 43.4	2068.1 ± 114.7
Mesencephalon	0.87 ± 0.25	7.1 ± 0.7	< .001	82.7 ± 21.8	962.9 ± 40.0
Amygdala	0.78 ± 0.19	5.7 ± 0.4	< .001	22.1 ± 7.1	169.6 ± 13.2
Nucleus accumbens	3.5 ± 0.1	38.6 ± 7.4	< .01	< 30.0	274.4 ± 74.6
Thalamus	0.41 ± 0.02	6.0 ± 1.1	< .01	< 30.0	529.5 ± 70.9
Septum	9.8 ± 3.8	32.3 ± 7.2	< .05	115.6 ± 25.8	589.0 ± 14.7
Medulla oblongata	1.4 ± 0.1	2.2 ± 0.3	< .05	104.0 ± 10.6	205.0 ± 23.7
Pons	0.47 ± 0.07	1.33 ± 0.21	< .05	< 30.0	120.3 ± 43.8
Cerebral cortex*	0.12 ± 0.003	0.84 ± 0.24	< .05	< 45.0	361.3 ± 72.6
Hippocampus	0.68 ± 0.31	1.01 ± 0.18	N.S.	59.8 ± 26.1	110.2 ± 14.1
Striatum	0.26 ± 0.01	0.46 ± 0.09	N.S.	< 15.0	30.5 ± 14.3
Pituitary gland	$(1.4 \pm 0.3) \times 10^5$	$(0.8 \pm 0.1) \times 10^5$	< .001	$(8.1 \pm 0.8) \times 10^5$	$(9.9 \pm 0.9) \times 10^5$

*Tissue corresponding to frontal cortex was lost in processing; therefore, cerebral cortex represents total cortex minus frontal cortex.

general regions of the brain (*18*) were obtained (Table 1). Each area was frozen, weighed, and placed in a tube containing ten volumes of 2*N* acetic acid at 95°C for 15 minutes. Each sample was homogenized and centrifuged, and the supernatant fluids were decanted and lyophilized. The dry residue was resuspended in assay buffer (containing 0.01*M* Na_2PO_4, 0.14*M* NaCl, and 0.1 percent gelatin, *p*H 7.6), and neutralized with NaOH, if necessary. Content of α-MSH was determined by radioimmunoassay (*19*) with several assay modifications (*20*). The α-MSH antiserum used in this study recognized the final three amino acids at the COOH-terminal end of α-MSH and elongation of α-MSH at the COOH-terminal end greatly reduced the cross-reactivity of the antiserum with the elongated α-MSH molecule. Test samples were assayed in duplicate at several dilutions, and Student's *t*-test was used to determine statistically significant differences between control and experimental groups in tissue levels of α-MSH.

In glutamate-treated animals, endogenous levels of α-MSH were reduced 90 percent throughout the rodent brain (Table 1). Total α-MSH in brains of control animals was 8.0 ng whereas that in MSG-treated animals was 0.8 ng. Of the brain areas examined, only the hippocampus had similar α-MSH levels in MSG-treated and control animals. In contrast to the reduction in α-MSH throughout the brains of MSG-treated animals, the concentration of α-MSH in the pituitary gland (picomoles per milligram of wet tissue) was higher in the MSG-treated animals; however, the total amount of α-MSH in the hypophysis was similar in experimental and control groups. The difference in pituitary concentrations of α-MSH between MSG-treated and control animals reflects simply a 50 percent reduction in wet weight and total protein in the pituitary glands of the MSG-treated animals. The reduction of α-MSH throughout the rodent brain after the administration of MSG provides convincing evidence that the primary source of α-MSH is neuronal perikarya of the arcuate nucleus.

Consistent with the conclusions of this study are the results of previous reports that indicate α-MSH-containing perikarya are localized in the arcuate nucleus of the brain (*16*), and that deafferentation or destruction of the arcuate region (*20*) lowers α-MSH content in extrahypothalamic regions of the brain just as MSG treatment does. Neuronal perikarya containing immunoreactive β-endorphin, β-lipotropin, and adrenocorticotrophic hormone (ACTH) have been localized in the arcuate region, and MSG treatment of neonatal animals resulted in a reduction of ACTH-like and endorphin-like material in hypothalamic and extrahypothalamic areas of the rodent brain (*21*). Furthermore, the localization in the arcuate region of neuronal perikarya containing ACTH, β-endorphin, and α-MSH, and the reduction of these substances after MSG treatment suggest that α-MSH, ACTH, and β-endorphin in the brain may be derived from a common precursor that is synthesized in arcuate-region neurons in a manner similar to that demonstrated in the pituitary gland (*21*).

In an attempt to clarify the mechanism or mechanisms by which the MSG induces the neuroendocrine syndrome, some investigators have found that neonatal MSG treatment alters prolactin, gonadotropin, or thyrotropin release; others have not (*4, 10, 11*). However, in all studies in which growth hormone (GH) was measured, the level of circulating GH fell after MSG treatment. Substances modulating the secretion of GH are incompletely known; however, both stimulatory (unknown) and inhibitory substances (somatostatin) of hypothalamic origin are involved (*22*). Although a cause-effect relationship between the decline in arcuate-region α-MSH and the decrease in release of GH in MSG-treated animals has not been established, several melanotropic peptides in the mediobasal hypothalamus, including α-MSH, are capable of releasing GH in vitro and in vivo (*23*). Furthermore, the arcuate region is implicated as a source of GH-releasing hormone because electrical stimulation of the area of the ventromedial and arcuate nuclei of the hypothalamus enhances GH release, and because lesions in this area reduce GH release (*22*). Finally, reported behavioral deficits in MSG-treated animals (*11, 24*) may be the result of the destruction of the α-MSH-containing cell bodies in the ventral hypothalamus that send multiple projections to numerous extrahypothalamic regions of the brain (*20*). Others have demonstrated that melanotropic peptides, including α-MSH, alter behavioral performance by direct action on the brain and at loci that contain endogenous α-MSH (*25*). Our demonstration that the arcuate nucleus is the primary source of α-MSH in the brain should provide a basis to further understand the physiological importance of α-MSH as a neuroregulatory peptide.

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Mosquitoes: Biting Behavior Inhibited by Ecdysone

Abstract. *Biting in* Anopheles freeborni *is inhibited during ovarian development. Biting inhibition is triggered by ecdysone, a hormone produced by the ovary during oogenesis. Biting inhibition does not occur in females after the removal of ovaries, but is restored by replacing ovaries or injecting ecdysone. Ecdysone also inhibits biting behavior when it is fed to females. This is the first example of ecdysone controlling a nonmolt-related behavior in insects.*

Most mosquitoes must acquire a blood meal in order for their eggs to mature, and in the process of obtaining the blood meal they often transmit disease to man. Between each cycle of egg production the females bite aggressively. After the mosquitoes have become engorged with blood they cease biting until oogenesis is complete (*1*). Therefore, the phase of egg development occurring after the blood meal coincides with a period of nonbiting behavior. The data in this report demonstrate that the association between egg production and biting behavior is mediated by the ovarian hormone ecdysone.

The ovaries begin secreting ecdysone shortly after the female takes a blood meal (*2*), so that the ecdysone titer is increased at the same time that biting behavior disappears. Experiments were conducted with 2-day-old female *Anopheles freeborni*. The ovaries were surgically removed according to a modification of the technique of Spielman (*3*). After a 24-hour recovery period these females were placed in a cage with an excess of males to facilitate mating. Two days later, the same females were allowed to become engorged with blood by feeding from my hand. A group of females with sham operations and a group of normal females were allowed to feed in the same way. After the initial feeding period, nonfeeders were removed so that each group consisted of females which had fed to repletion. On subsequent days each group was given the opportunity to feed for 10 minutes, and the number that fed was recorded. During the experi-

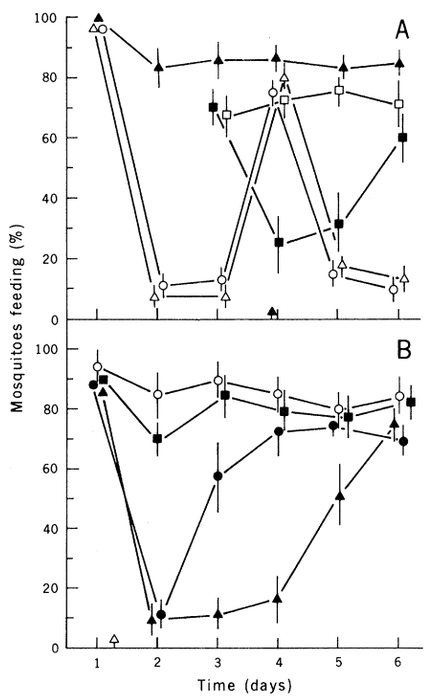

Fig. 1. (A) The blood-feeding behavior of *A. freeborni*. Each point represents the response of 25 females: (▲) ovariectomized, (△) with sham operations, and (○) normal. Early on day 3, one-half of the ovariectomized females were implanted with an undeveloped ovary from a 2-day-old donor and subsequently assayed for feeding behavior (■). Control females were implanted with pieces of midgut (□). Vertical bars show the standard error of the mean for three experiments. The arrow indicates the time at which females with sham operations and normal females oviposited. The mosquitoes were maintained at 26°C, with a photoperiodic cycle (L : D) of 15 hours of light and 9 hours of darkness. (B) Females were injected with β-ecdysone at three different concentrations: (▲) 5 μg/μl, (●) 1 μg/μl, (■) 0.5 μg/μl, or (○) with saline. The open arrow indicates the time of injection. Each point represents the mean response of 25 females; vertical bars show the standard error of the mean for three experiments conducted at 26°C, L : D, 15 : 9.

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