until the storage pools of carbon are replenished (Eq. 2).

The uncoupling of μ and λ has implications for models of phytoplankton population dynamics. Usually λ in these models is estimated from measurement of $P(I)$ and integration of the $P(I)$ curve over the photic zone, the photophase variation in light intensities, the chlorophyll a concentrations, and an assumed ratio of carbon to chlorophyll a; it is then assumed that $\lambda = \mu$. Usually homogeneity is assumed and therefore phototaxis, recirculation mechanisms, and transport are neglected. The possible diurnal periodicity of photosynthetic capacity in the phytoplankton can introduce a significant error in the estimation of λ (*16*). Finally, our results imply that even though correction of $P(I)$ for diurnal periodicity may provide better estimates of λ, the assumption that $\lambda = \mu$ is not necessarily valid in the natural system. Rather there would be, in changing photic environments, a selection for individuals or species characterized by a flexible metabolism (*17*), capable of allocating cell carbon to maximize this rate of reproduction (*15, 18*) in changing photic environments and thus optimize survival (*19*).

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References and Notes

1. The specific cell division rate, μ, is defined as $\mu = 1/N(dN/dt)$, where N is the cell concentration. The carbon-specific growth rate, $\lambda = P(I)/C$, where $P(I)$ is the rate of photosynthetic carbon uptake per cell and C, total cell carbon, is the sum of storage carbon, C_s, and structural or constitutive cell carbon, C_c. Under steady-state conditions of growth at any light intensity, I, $P(I) = \mu(I)(C_c + C_s^{max})$.
2. The solution of this linear differential equation is $C_s = C_s^{max}\{1 - \exp[-P(I)t/C_{max}]\}$.
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8. A series of replicate 500-ml Erlenmeyer flasks containing 250 ml of seawater enriched with F/2 [R. R. L. Guillard and J. H. Ryther, *Can. J. Microbiol.* **8**, 229 (1962)] were inoculated with *Gonyaulax polyedra* adapted to high light (210 $\mu E/m^2$-sec) and were incubated at 23° ± 1°C in a photoperiodic cycle of 12 hours of light and 12 hours of darkness with cool white fluorescent illumination of 210 (or 180) $\mu E/m^2$-sec until the specific cell division rates (determined by linear regression of the natural logarithm of the daily cell counts plotted against time) in all the flasks were the same. Replicate pairs of flasks were distributed among eight lower light intensities. Cells were counted in a Palmer-Maloney chamber. As an example of the precision of these division rate measurements, the regression slopes ± S.E. (N = 18) for four replicate flasks at 120 $\mu E/m^2$-sec were 0.22 ± 0.01, 0.22 ± 0.01, 0.22 ± 0.02, and 0.23 ± 0.01 per day. Chlorophyll a, extracted in 90 percent acetone, was measured fluorometrically. Cell carbon was measured in a Perkin-Elmer elemental analyzer (model 240). Photosynthetic carbon uptake was measured by a standard radiocarbon technique [M. E. Loftus, A. R. Place, H. H. Seliger, *Estuaries* **2**, 236 (1979)].
9. The lag appears to be real and may be related to the necessity for replenishment of stored carbon pools.
10. The constancy in the amount of chlorophyll a per cell throughout phase 1 (Fig. 2A) and the parallel shapes of the $\mu(I)$ and $P(I)$ curves (Fig. 2B) contrast with previously published results for *G. polyedra* [B. B. Prezelin and B. M. Sweeney, *Mar. Biol.* **48**, 27 (1978)]. These authors report a μ_{max} of 0.3 per day, chlorophyll a of 36 to 72 pg per cell, and maximum rates of photosynthesis of 150 ng of carbon per cell per hour (O_2 evolution corrected to carbon uptake by the factor 1.2) [B. B. Prezelin and R. S. Alberte, *Proc. Natl. Acad. Sci. U.S.A.* **75**, 1801 (1978)] that are almost identical with those in this report. The differences may be due in part to a longer period of adaptation to steady-state conditions for which our experiments were not designed.
11. This pattern of carbon uptake parallels that observed for the uptake of nitrogen and phosphorus [P. M. Glibert and J. C. Goldman, *Mar. Biol. Lett.* **2**, 25 (1981); R. W. Eppley and E. H. Renger, *J. Phycol.* **10**, 15 (1974); G. W. Fuhs, S. D. Demmerle, E. Canelli, M. Chen, in *Nutrients and Eutrophication*; G. E. Likens, Ed. (American Society of Limnology and Oceanography, Lawrence, Kans., 1972), p. 113; B. H. Ketchum, *J. Cell. Comp. Physiol.* **13**, 373 (1939); E. J. Ketchum and B. H. Ketchum *Biol. Bull.* **123**, 134 (1962); R. B. Rivkin, thesis, University of Rhode Island (1979)]. The condition analogous to $P(I)/\mu(I)C > 1$, the uptake of nutrients at rates higher than that required for cell division, has been reported for N- and P-specific uptake in situations where cells are nutrient-limited and exposed to high nutrient concentrations. [J. C. Goldman, J. J. McCarthy, D. G. Peavey, *Nature (London)* **279**, 210 (1979); J. J. McCarthy and J. C. Goldman, *Science* **203**, 670 (1979); R. B. Rivkin and E. Swift, *J. Phycol.*, in press.]
12. The parallel shapes of $\mu(I)$ and $P(I)$ for steady-state growth require that the total amount of carbon per cell be essentially the same for all light intensities.
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14. This constancy in the amount of carbon per cell may be species-specific; the cellular concentrations of total and storage carbon may be light-dependent in some other algae [B. Shuter (*15*); E. A. Laws and T. T. Bannister, *Limnol. Oceanogr.* **25**, 457 (1980)].
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N-Acetylation Regulates the Behavioral Activity of α-Melanotropin in a Multineurotransmitter Neuron

Abstract. *A multineurotransmitter neuronal system that synthesizes and secretes both acetylated and deacetylated forms of α-melantropin and β-endorphin is present in rat and human brain. The* N*-acetylated form of α-melanotropin had more potent behavioral effects than the deacetylated α-melanotropin. In the case of β-endorphin, however, the deacetylated form has been shown to be more potent than the acetylated form. Enzymatic* N*-acetylation appears to be an important regulatory process for modulating the behavioral activity of peptides secreted from the opiomelanotropinergic multineurotransmitter neuron.*

Neurons have usually been thought to contain and release only one neurotransmitter substance. Recently, however, a number of neurons have been shown to contain more than one putative neurotransmitter. The opiomelanotropinergic neuron is one such multineurotransmitter neuron; this neuron synthesizes and releases at least five neuropeptides—β-endorphin, *N*-acetylated β-endorphin, γ-melanotropin (γ-MSH), and *N*-acetylated and deacetylated α-melanotropin (α-MSH) (*1–3*). All of these peptides are biosynthetically derived from a single precursor protein, pro-opiomelanocortin (*4*). Because α-MSH is formed by endopeptidase cleavage from pro-opiomelanocortin, a deacetylated form of α-MSH is also likely to be present in these neurons and may be one of the α-MSH-related peptides found in brain; deacetylated α-MSH is present in the pituitary glands of a number of species that also synthesize pro-opiomelanocortin (*2, 5*).

To determine if deacetylated α-MSH was present in brain we used high-pressure liquid chromatographic fractionation combined with radioimmunoassays for α-MSH. Fractionation of α-MSH immunoreactive material in rat and human brain demonstrated that deacetylated α-MSH is present in the central nervous

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system of both species and is actually more abundant than α-MSH. Figure 1 shows a high-pressure liquid chromatograph of α-MSH immunoreactivity in rat and human hypothalamus compared to the elution of synthetic α-MSH (Peninsula Laboratories), deacetylated α-MSH (custom synthesis, Peninsula Laboratories), and the methionine sulfoxide forms of these peptides [synthesized by reacting the synthetic peptides with hydrogen peroxide and purifying these peptides by high-performance liquid chromatography fractionation; see (*6*)]. In addition to the immunoreactive peak coeluting with α-MSH, three other immunoreactive peptides that coelute precisely with deacetylated α-MSH and the methionine sulfoxide forms of α-MSH and deacetylated α-MSH were detected by a COOH-terminally specific antiserum (AB 22) which recognized both synthetic acetylated and deacetylated forms of the peptide. An NH_2-terminally directed antiserum (developed by R. Eskay and M. Brownstein), which requires the *N*-acetyl group for recognition, does not detect immunoreactivity coeluting with deacetylated α-MSH or the sulfoxide form of this peptide. The sulfoxide forms of α-MSH and deacetylated α-MSH appear to be formed artifactually during the extraction procedure because methionine sulfoxide peptides are generated during extraction of synthetic α-MSH or deacetylated α-MSH. The fact

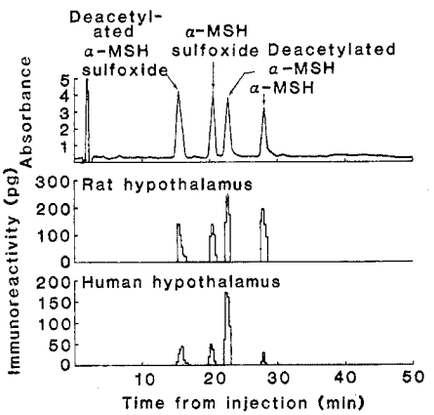

Fig. 1. High-pressure liquid chromatograph of immunoreactive α-MSH in rat and human brain compared to the elution of α-MSH standards. Immunoreactive α-MSH was extracted from three different human brains (8 to 22 hours post-mortem) and from rat brains with 0.1*N* HCl or 2*N* acetic acid and boiled for 10 minutes. Similar results were achieved with each extraction. A portion of the supernatant of the acid extract (obtained by centrifugation at 10,000 *g*) was adjusted to *p*H 3.0, filtered, and subjected to high-pressure liquid chromatography on a μBondapak C18 reverse phase column with triethylammonium formate being used as the buffer and acetonitrile as the mobile phase [see (6)].

that no interconversion of α-MSH to deacetylated α-MSH or deacetylated α-MSH to α-MSH was observed during the extraction procedure of synthetic peptides indicated that these peptides occur naturally in brain.

β-Endorphin has both physiological and behavioral actions: among the former the best known is the induction of analgesia. In contrast to β-endorphin, the *N*-acetylated endorphin has no analgesic potency and does not bind to the opiate receptor (*3*). Therefore, *N*-acetylation appears to regulate the activity of β-endorphin and may also regulate the activity of α-MSH. To examine the influence of *N*-acetylation on the activity of α-MSH, we compared the behavioral activities of deacetylated and acetylated α-MSH in two different tests.

First, we used a Y-maze testing procedure for black-and-white visual discrimination in rats as described previously (*6, 7*). Performance on this task is improved by intraperitoneal administration of α-MSH (*7*), but how this facilitation is brought about is unclear; α-MSH and related peptides have been hypothesized to increase memory, attention, arousal, or the motivational influence of environmental clues (*8*). We found that rats given daily intraperitoneal injections of α-MSH and deacetylated α-MSH (40 μg/kg; 1 mg of peptide per 25 ml of saline) 15 minutes before they were tested showed significantly improved performance on the maze compared to rats treated with saline. The saline-treated rats achieved criterion performance on acquisition and reversal of behavior in the Y-maze in 45 ± 1 and 45 ± 7 trials, respectively. Rats receiving α-MSH achieved acquisition and reversal criteria in 20 ± 4 and 20 ± 6 trials, respectively, approximately twice as fast as the saline-treated rats ($P < .05$; analysis of variance and Duncan's New Multiple Range Test). Treatment of rats with deacetylated α-MSH also produced a slight improvement in acquisition and reversal compared to saline-treated rats, 35 ± 4 ($P < .05$) and 40 ± 6 (not significant), respectively, although the effect was significantly less ($P < .05$ on acquisition, $P < .01$ on reversal) than that induced by α-MSH.

In the second test we evaluated the relative potencies of α-MSH and deacetylated α-MSH in eliciting excessive grooming in rats because α-MSH and the structurally related corticotropin (ACTH) peptides induce an excessive grooming syndrome in a dose-dependent fashion (*8*). The excessive grooming elicited by α-MSH is thought to reflect a state of excessive arousal (*9, 10*) and excessive grooming in rodents is induced by fear, stress, or exposure to a novel situation (*10*). Figure 2 shows the grooming response of rats injected intraventricularly with saline, deacetylated α-MSH (dotted line), or α-MSH (solid line). Rats injected with 0.1 to 10 μl of saline received grooming scores between 25 and 30; these scores were similar to those of rats that received no injections. Both α-MSH and deacetylated α-MSH increased grooming in a dose-dependent fashion although α-MSH was one or two orders of magnitude more potent. The lowest dose of deacetylated α-MSH to significantly increase grooming scores was 600 times higher than that required for α-MSH induction of grooming. The data are consistent with the findings of Gispen *et al.* (*9*), who demonstrated that *N*-acetylated ACTH analogs are more potent in inducing the excessive grooming syndrome.

The qualitative similarities between the behavioral effects of deacetylated and acetylated α-MSH indicate that the α-MSH behavioral information is encoded in the amino acid sequence of the peptide and that the molecular potency is in part regulated by *N*-acetylation. The quantitative differences in potency between α-MSH and deacetylated α-MSH could be due either to differences in

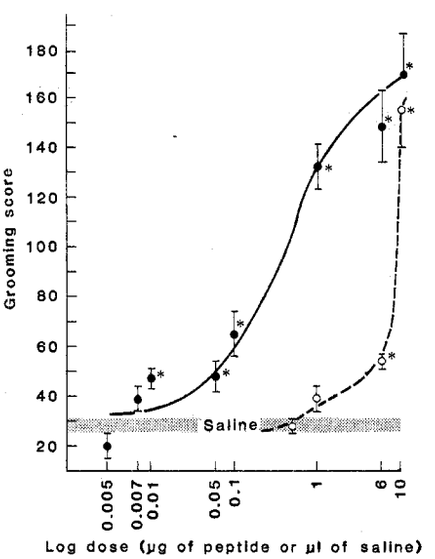

Fig. 2. Effects of intraventricularly administered deacetylated α-MSH (dotted line), α-MSH (solid line), or saline (1 to 10 μl; horizontal gray bar) on induction of grooming behavior. Peptides, 0.005 to 10 μg, dissolved in 10 μl or less of phosphate-buffered saline, *p*H 7.4, were injected into the lateral ventricles of rats prepared with implanted cannulas at least 3 days prior to experimentation. Fifteen minutes after they were injected the rats were evaluated for grooming for a testing period lasting 55 minutes as described (*6, 7, 9*). Every 15 seconds the rat was observed and given a score of 1 if engaged in grooming or 0 if no grooming was observed. The maximum possible score for a single test was 220.

degradation rates by peptidases or to differences in affinity for α-MSH or deacetylated α-MSH receptors. Recent studies demonstrate that brain peptidases degrade deacetylated α-MSH about ten times as fast as they degrade α-MSH (*6*). More rapid degradation of deacetylated α-MSH cannot, however, totally explain the differences in behavioral activity of the two peptides because deacetylated α-MSH is actually more potent than α-MSH in a number of behavioral actions. One behavioral action in which deacetylated α-MSH is more potent than α-MSH is in the ability to block opiate analgesia and opiate receptor binding. Deacetylated forms of α-MSH or structurally related ACTH analogs consistently block opiate-induced analgesia and opiate or β-endorphin receptor binding, whereas the *N*-acetylated α-MSH is devoid of activity (*11*). It is clear that in this case receptor affinity and not peptidase sensitivity is likely to be responsible for the differences in bioactivity and that the *N*-acetyl group may be an important determinant for receptor interaction. The data, therefore, indicate that there are different structural requirements for different behavioral actions of α-MSH, a finding that has been demonstrated previously (*12*), and that interactions with these different postsynaptic receptors could be regulated by acetylation reactions in the presynaptic opiomelanotropinergic neuron. It is interesting that β-endorphin also occurs in both acetylated and deacetylated forms in the opiomelanotropinergic neuron and that acetylation of the β-endorphin eliminates its potential to bind to the opiate receptor and elicit analgesia (*3*). The acetylation of both α-MSH and β-endorphin appears to result from enzymatic mechanisms, and recent results indicate that different enzymes acetylate each of these peptides (*13*). Furthermore, α-MSH acetylating enzymatic activity appears to be induced by physiological manipulations which induce α-MSH synthesis (*13*). Differential regulation of the two acetylating enzymes could alter the ratios of deacetylated and acetylated forms of α-MSH and β-endorphin and by doing so change both the composition of the secretory output of the opiomelanotropinergic neuron and the resulting postsynaptic actions. Such molecular regulatory processes are considerably more complex than the mechanism proposed by the classical model of the neurosecretory cell which releases one neurotransmitter or hormone. It is clear, however, that independent processing of individual neurotransmitters or hormones released from one multiple transmitter secretory cell adds another dimension of complexity and flexibility to intercellular communication. A question of importance is whether cells secreting multiple chemical signals are the exception or rule.

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Excretion of β-Phenethylamine Is Elevated in Humans After Profound Stress

Abstract. *The urinary excretion rate of the endogenous, amphetamine-like substance β-phenethylamine was markedly elevated in human subjects in association with an initial parachuting experience. The increases were delayed in most subjects and were not correlated with changes in urinary pH or creatinine excretion. The data suggest a stress-related role for β-phenethylamine.*

β-Phenethylamine (PEA), an endogenous amine that resembles amphetamine both structurally and pharmacologically (*1*), has been implicated as an etiological factor in paranoid schizophrenia. Patients with this disorder excrete large amounts of PEA in their urine (*2*). Stress may also play a role in paranoid schizophrenia, since in some patients it precipitates psychotic episodes (*3*). Amphetamines can produce a paranoid state (*4*), and stress can reinstate this psychosis in individuals who then are abstinent (*5*). It seems, therefore, that stress may play a role in changes in PEA excretion observed in paranoid schizophrenics. However, there has been no evidence that stress can alter PEA disposition in any species. We now report an elevation in the urinary excretion of PEA in humans following a parachute jump.

Our subjects were male and female college students 18 to 28 years of age. They were admonished to refrain from ingesting alcohol or other drugs during urine sampling periods. Urine was collected from each subject during a 24-hour control period (2100 to 2100 hours) 7 to 21 days before the jump and during the same 24-hour period encompassing the jump. In some subjects urine was collected for an additional 18 hours after the jump. Collected urine was stored at −70°C until being assayed for PEA by gas chromatography–mass spectroscopy (*6*). The urine from each micturition was assayed separately.

Stress during the parachuting experi-

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