cabbage host plants on the basis of leaf color. The ability of the flies to distinguish colors was improved in intermediate-aged and mature plants as compared with young plants. The degree to which leaf color differences, in concert with host olfactory stimuli, might influence female landings in large homogeneous agricultural patches of these three cultivars remains to be determined.

Our findings together with those of other workers (*5–8*) lead us to suggest that with some exceptions (*3, 7*) leaves of cultivated or naturally growing plants that appear whitish to the human eye, owing to buildup of epicuticular bloom (*16, 18*) or pubescence (*19*), or that are reddish, owing to masking or replacement of chlorophyll by other pigments, may be less detectable by (or less attractive to) certain insect herbivores than are green leaves. Although the presence of bloom produces an increase in overall intensity of leaf reflectance, it acts to desaturate chlorophyll reflectance and thereby to decrease the distinctiveness of peak green leaf reflectance at 500 to 600 nm and to decrease the magnitude of the difference between total insect-detectable energy reflected above 500 nm and that reflected below 500 nm (*2, 5*). In red-colored leaves, the masking or replacement of chlorophyll results in the absence of a reflectance peak at 500 to 600 nm.

In certain cases, buildup of epicuticular bloom could have evolved in response to strong selection pressure from a damaging insect herbivore. It is much more likely, however, that in most cases such buildup evolved in response to abiotic environmental factors or microorganisms (*16, 18*), with insects then obliged to "track" this character (*20*). Alternatively, insect herbivory may have contributed to the evolution of nonsenescing red-colored leaves in various plant species in which flushes of new growth are sometimes red.

Several crop cultivars with bloomed or red leaves are less preferred for landing or oviposition by certain insect species than are their green-leaved counterparts (*5–8, 18, 21*). We believe that, provided plant yield and quality can be maintained, leaf color characteristics merit further investigation by entomologists involved in plant breeding to determine whether it is possible to reduce the attraction of certain insect pests to plants.

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Antipyretic Potency of Centrally Administered α-Melanocyte Stimulating Hormone

Abstract. *Centrally administered α-melanocyte stimulating hormone is much more potent in reducing fever than the widely used antipyretic acetaminophen. This finding supports the hypothesis that the endogenous neuropeptide has a role in the limitation of fever and suggests that it may be clinically useful as an antipyretic.*

There is evidence that the neuropeptides adrenocorticotropic hormone (ACTH)(1-24) and α-melanocyte stimulating hormone (α-MSH)(1-13), which share a 13-amino-acid sequence, can influence centrally mediated processes (*1, 2*), including central control of body temperature (*3–5*). Both peptides lower core temperature of afebrile rabbits when given peripherally or centrally in sufficient doses (*6–8*), and much smaller doses reduce fever without altering normal temperature (*6, 7, 9*). α-MSH is found in brain regions that govern temperature regulation, including the anterior hypothalamus and the septum (*2*). The concentration of α-MSH in the septum rises during fever (*10*), and the concentration in the arcuate nucleus tends to decline at the same time, presumably reflecting axoplasmic transport of α-MSH from its source in cell bodies of the arcuate nucleus to fibers in the septum. Microinjections of α-MSH into the septal region reduce fever (*11*)—possibly mimicking the result of the natural transport. To evaluate the physiological significance of fever reduction by endogenous α-MSH, it is important to compare the antipyretic effect of this peptide with that of a well-known antipyretic drug, such as acetaminophen. Since acetaminophen and α-MSH appear to reduce fever through actions in the central nervous system, and since changes in potency can result from peripheral administration, both substances were given centrally in the experiments described here.

Adult New Zealand White rabbits were implanted with cannulas in a lateral cerebral ventricle (*6*) and restrained in conventional stocks in an environmental chamber at 23°C. A thermistor probe (Yellow Springs No. 701) was inserted 10 cm into the rectum and taped in place. Temperature measurements were made automatically at 10-minute intervals with a MINC 11 computer connected to a Datalogger digital temperature recorder (United Systems). Leukocytic pyrogen (*6*) was injected intravenously after a 1-hour baseline temperature had been determined. Tests of the antipyretic effect of α-MSH and acetaminophen were performed in two separate experiments on the same animals, with the control response to leukocytic pyrogen being determined for each animal before each

experiment. Doses of α-MSH (*12*), acetaminophen (*13*), and sterile saline were assigned according to a table of random numbers. Each dose was injected intracerebroventricularly 30 minutes after the test injection of pyrogen. The 30-minute delay was to ensure that each rabbit was producing the appropriate febrile response to pyrogen. All injections of pyrogen were separated by at least 48 hours to minimize the development of tolerance.

Both substances caused dose-related reductions in fever (Fig. 1), but α-MSH was much more potent. Whereas 0.50 mg of acetaminophen reduced the area under the fever curve 47 percent, only 200 ng of α-MSH was required to cause a 50 percent reduction in the area under the fever curve (Fig. 2). This indicates that α-MSH was more than 2500 times more potent, by weight, than acetaminophen in reducing fever by about one-half. When the molecular weights of α-MSH (1665) and acetaminophen (151) are considered, α-MSH was more than 25,000 times more potent than acetaminophen. No endogenous substance other than ACTH is known to have such potency in reducing fever. This finding supports the idea that α-MSH is important in the limitation of fever (*6–9*).

Previous research with larger doses of α-MSH showed that the hypothermic effect was the result of reductions in heat production and conservation and not of lowering of the thermoregulatory set point (*6*). However, when administered centrally to afebrile adult rabbits in a thermoneutral environment, low doses caused no significant changes in body temperature. One possibility is that, in small amounts, α-MSH competes with leukocytic pyrogen for receptor sites in the brain without altering central temperature control pathways, much as common antipyretics are thought to do (*14*). Alternatively, although α-MSH has no effect on normal body temperature in low doses, it may still reduce fever by inhibiting heat production and conservation pathways, but only when activity in these pathways is at greater than normal levels, as in fever. However, we recently found that central injections of α-MSH in the low doses used in this study did not cause significant changes in body temperature in afebrile adult rabbits exposed to cold (10°C) (*15*). If low doses of α-MSH simply inhibit heat production and conservation pathways in the central nervous system, then body temperature should decrease in the cold when activity in these pathways is increased.

The antipyretic potency of α-MSH and the fact that this peptide reduces fever even when given peripherally (*6–9*) may have clinical significance. ACTH was used to reduce clinical and experimental fever soon after it was first described (*3, 4, 16*), but this peptide also stimulates corticosteroid release, and can, with repeated administration, result in Cushing's syndrome (*17*). On the other hand, the shorter α-MSH molecule, which is derived from ACTH, does not stimulate steroid release, and there appear to be no irreversible deleterious effects when very large doses are given to rabbits and to man (*6–9, 17–19*), nor have side effects been noted after administration of low doses in rabbits (*6–9, 15*). Although α-MSH is relatively expensive, only very small amounts may be required to effectively reduce fever. Since this peptide occurs naturally in man, its use as an antipyretic may amount to simply augmenting an endogenous antipyretic without introducing a foreign substance as with other antipyretics.

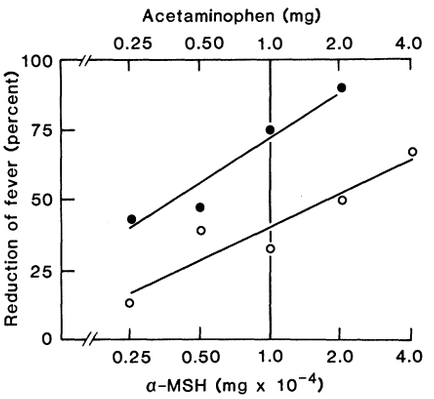

Fig. 1. Antipyretic response to α-MSH and acetaminophen, calculated as the percent reduction from the control fever curve over 4 hours. All doses of α-MSH and acetaminophen produced significant reductions in fever ($P < .01$, Wilcoxon signed-ranks test). Each point represents the mean ($N = 7$) reduction in fever in the same rabbits. The slopes do not differ significantly. Symbols: (○) intravenous leukocytic pyrogen plus intracerebroventricular α-MSH; (●) intravenous leukocytic pyrogen plus intracerebroventricular acetaminophen.

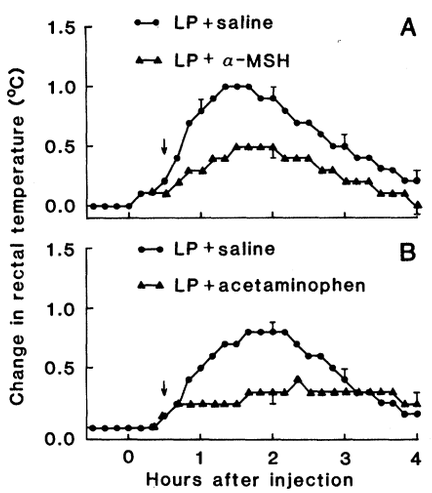

Fig. 2. Reduction of the area under the control fever curve by central administration of α-MSH (200 ng) and acetaminophen (0.50 mg). Each curve represents the mean ($N = 7$) response (± standard error) at each hour. Percent reductions were calculated over a 4-hour period after intravenous administration of leukocytic pyrogen (*LP*). Arrows show when intracerebroventricular injections of the test substances were given.

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