large doses of L-dopa had no effect on shock thresholds (Table 2) (*13*). Tryptophan injections produced dose-related increases in the concentrations of brain tryptophan, serotonin, and 5-HIAA among animals consuming either the corn or the casein diets; however, brain tryptophan and 5-HIAA were increased to a greater extent in corn-fed animals. These differences have been observed previously (*14*) and may reflect increased transport of tryptophan into the brain, perhaps resulting from differences in the concentrations of plasma tryptophan relative to the other neutral amino acids that normally compete with it for transport from the plasma into the brain (*1*).

Our results indicate that animals consuming tryptophan-poor diets that decrease brain serotonin will become hyperalgesic to electric shock. A similar correlation between brain serotonin and pain sensitivity has been noted by others using various pharmacological or surgical techniques for reducing brain serotonin (*4–6*). When viewed together, these findings suggest that brain neurons containing serotonin may suppress the sensitivity or reactivity of rats to painful stimuli. We believe that our results are the first that relate behavioral changes that follow dietary manipulations directly to a change in a putative brain neurotransmitter. These nutrition-induced changes in brain neurochemistry and behavior can be reversed by dietary or pharmacological manipulations; by feeding animals diets adequate in tryptophan or injecting them with replacement doses of the amino acid, their response thresholds to electric shock can be restored almost to normal (*15*). These behavioral changes appear to be related specifically to alterations in brain neurons containing serotonin.

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13. Several lines of evidence indicate that the diet-induced changes in electroshock sensitivity were not due to the growth retardation seen in animals consuming the low-protein corn diet. (i) The injection of tryptophan or other drugs thought to specifically alter brain neurons containing serotonin produce predictable changes in the electroshock thresholds of corn-fed animals (R. B. Messing, L. Fisher, L. Phebus, L. D. Lytle, in preparation). (ii) Although the body weights of the corn-fed animals differed significantly from the body weights of the control group within 1 week of the experiment, the body weights of the corn-fed animals remained approximately constant throughout the course of the experiment, even though their electroshock thresholds were decreased greatly from their own baseline levels. (iii) The electroshock thresholds of the corn-fed animals were restored to normal within 2 weeks after consumption of the casein control diet, even though body weights of these animals were only 42 percent of those of the casein-fed animals.
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15. Our data on other behavioral tests of rats fed the tryptophan-poor corn diet over a long period indicate that these animals are hyperactive in running wheels, hyperreactive to environmental stimuli, and display bizarre social behavior. Some of these behavioral changes appear to be similar to those described in animals with pharmacological or surgical reductions in brain serotonin; however, some of these behavioral changes may also result from the nonspecific effects of malnutrition (R. B. Messing, L. Fisher, L. Phebus, L. D. Lytle, in preparation).
16. Supported by a grant from the W. T. Grant Foundation to L.D.L., a grant from the John A. Hartford Foundation, and a postdoctoral fellowship from the Medical Foundation to R.B.M.

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Trigeminal Lemniscal Lesions and the Lateral Hypothalamic Syndrome

Bilateral lesions in the region of the lateral hypothalamus result in profound behavioral impairments. Rats with such lesions become aphagic and adipsic, show severe sensory and motor impairments, and often starve to death within 6 to 10 days. If kept alive by intragastric intubation of liquid nutrients, some animals are eventually able to maintain themselves on lab chow and water. Nevertheless, persistent deficits in their feeding and drinking responses to acute nutritional needs indicate that recovery of function is not complete. The initial disabilities, progressive recovery, and enduring residual deficits together form what has become known as the "lateral hypothalamic syndrome" (*1*).

Although the critical effect of these lesions has traditionally been attributed to destruction of cell groups within the hypothalamus, increasing attention has recently been paid to the possible role of damage to fiber bundles which traverse this region. Zeigler and Karten (*2*) have now reported that in the rat, lesions of the trigeminal lemniscus, which courses through the diencephalon, produce functional deficits which they find reminiscent of the lateral hypothalamic syndrome. While this is an interesting extension of Zeigler's important work on trigeminal deafferentation in the pigeon (*3*), we believe that they overstate their case when they suggest that "many of the deficits previously subsumed under the rubric of the 'lateral hypothalamic' syndrome are actually the result of incidental damage to the trigeminal lemniscus" (*2*). Although their lesioned rats did become aphagic and adipsic in the immediate postoperative period, (i) it is difficult to attribute these impairments to the bilaterally damaged tissue alone, since the lesions were markedly asymmetrical and included considerable nonspecific damage; (ii) the animals did not show any of the other early symptoms of lateral hypothalamic damage, such as akinesia, catalepsy, and general sensory neglect (*4*); (iii) the median duration of adipsia was only 2 days, whereas it commonly lasts several weeks after lateral hypothalamic lesions (*1*); (iv) aphagia seemed to continue beyond recovery from adipsia, whereas this never is observed after lateral hypothalamic lesions (*1*); and (v) no evidence was presented regarding any of the residu-

al deficits seen after hypothalamic lesions with the exception of increased food wastage and reduced body weight, neither of which necessarily reflect trigeminal damage.

For these reasons, we believe that the syndrome described by Zeigler and Karten (*2*) bears only a superficial resemblance to the lateral hypothalamic syndrome. As such, their findings may be contrasted with accumulating evidence that the critical effects of lateral hypothalamic lesions can be reproduced by extensive damage to the ascending dopaminergic neurons which traverse this area (*5*). For example, lesions placed at extrahypothalamic sites along these projections produce aphagia, adipsia, and akinesia that may last several weeks or longer (*6, 7*), and similar results also have been obtained following the administration, at the same intracerebral sites, of 6-hydroxydopamine (6-HDA) (*7*), a drug which can be used to destroy catecholamine-containing neurons while producing minimal damage to adjacent fibers. In addition, these rats show the same patterns of gradual recovery from aphagia and adipsia, as well as most of the residual deficits, that are seen in rats with lateral hypothalamic lesions (*8*). Interestingly, among the few deficits that 6-HDA–treated rats do not share with "recovered laterals" is an increased food wastage. While this may represent a contribution of lemniscal damage to the lateral hypothalamic syndrome (*9*), it is also possible that lateral hypothalamic lesions interrupt descending fibers to the salivary motor nuclei in the brainstem.

We do not question the possible importance of sensory factors to feeding and drinking behaviors, or the possibility that sensory function is impaired by lateral hypothalamic lesions. On the other hand, we believe that many of the observed sensorimotor deficits may not result from damage to primary afferent or efferent pathways but from destruction of dopaminergic neurons which serve to gate the transmission of sensory and motor signals. Thus, although the absence of consummatory behaviors may be the alterations that are most conspicuous to the investigator and are of gravest consequence to the experimental animals, their impairments are not limited to motivated ingestive behaviors but seem to include any voluntary activity that demands orientation to sensory stimuli and sustained alertness (*5*).

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9. Note that lateral hypothalamic lesions which deplete striatal dopamine by only 60 percent can produce the entire syndrome of behavioral disruptions, whereas intraventricular injections of 6-HDA are not similarly effective until at least 90 percent depletions of dopamine are obtained [M. J. Zigmond and E. M. Stricker, *Science* **182**, 717 (1973)]. These findings suggest that electrolytic lesions may interrupt nondopaminergic neurons which contribute to the observed effects.

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In our report (*1*) we reached three conclusions: (i) central trigeminal structures play an important role in the control of ingestive behavior in two vertebrate classes —birds and mammals; (ii) the characteristic lateral hypothalamic (LH) lesion is likely to produce incidental damage to central trigeminal structures; (iii) components of the LH syndrome are attributable to trigeminal damage. Stricker *et al.* do not challenge the validity of these conclusions, merely their implications (*2*). For reasons which we hope to make clear we consider their criticisms to be irrelevant to the main thrust of our report. Nevertheless, they deserve substantive comment.

Briefly, Stricker *et al.* contend that damage to central dopaminergic neurons *is*, while damage to trigeminal structures is *not*, followed by "critical" features of the LH syndrome. We would comment as follows.

1) Catalepsy, akinesia, and general sensory neglect, although they may be characteristic of both the LH and 6-hydroxydopamine syndromes, can only confound the analysis of deficits *specific* to the control of eating and drinking. Indeed, their last sentence seems to imply that injections of 6-hydroxydopamine do impair a wide variety of integrated behavior patterns. We are aware of no evidence that dopaminergic neurons (as contrasted with cholinergic or adrenergic neurons) "gate the transmission of sensory and motor signals." Even if true, this statement is hardly a recommendation for the use of 6-hydroxydopamine injections as an analytic technique for the study of ingestive behavior mechanisms. The fact that trigeminal lesions produce aphagia, adipsia, and a specific (namely, trigeminal) sensory "neglect" in otherwise alert and active animals suggests that the trigeminal system makes a relatively *specific* contribution to the control of ingestive behavior.

2) Damage to *peripheral* trigeminal structures (nerve and ganglion) also produces a syndrome including aphagia, adipsia, spillage, and increased latency to feed. The extent and persistence of the deficits in feeding are a function of the texture and palatability of the diet (*3*).

3) Conclusions as to residual deficits after trigeminal lesions must await completion of our long-term studies. However, Stricker and Zigmond have themselves reported that rats injected with 6-hydroxydopamine "showed some but not all of the residual deficits of recovered laterals" (*4*). We did not suggest that the LH syndrome should be attributed solely to trigeminal damage. To attribute so disparate a collection of deficits to any single causal factor would be an unwarranted inference from the available data. Zigmond and Stricker have noted elsewhere (*5*) that rats made aphagic by injections of 6-hydroxydopamine may resume feeding despite an almost total depletion of striatal dopamine. Not surprisingly they suggested that "damage to pathways other than the nigrostriatal system can be important in the LH syndrome" (*4*).

That we should be debating the "critical" features of the LH syndrome—surely a semantic rather than an empirical problem—suggests we have lost sight of the substantive issues. As Epstein notes, "The(se) systems for the initiation of ingestive behavior are not centered in the lateral hypothalamus, except in the sense that an unusually large number of their partial components are focussed there. An obvious task now is the identification and analysis of these partial components" (*6*). It is from the perspective of this passage that we view the criticisms of Stricker *et al.* as irrelevant to our findings. Our study was not an attempt to account for the LH syndrome. It was a logical extension of our

previous work on the role of trigeminal mechanisms in the control of vertebrate ingestive behavior (*7*).

For this reason our study involved lesions of a specific sensory pathway rather than damage to a large number of diencephalic structures or massive depletion of regional neurotransmitters. We obviously think it significant that so restricted a lesion should have such disruptive effects. Nevertheless, given the relative specificity of the trigeminal lesion we are not surprised that there should be significant differences between the trigeminal and the LH syndromes. It is precisely such differences that are of greatest interest, because they enable us to identify the unique contributions of the trigeminal system to the control of ingestive behavior.

The fact that both trigeminal and neurochemical lesions produce some of the same deficits suggests that they may operate upon common substrates. We hope future research from both our laboratories will clarify the relation between central dopaminergic and central neurosensory processes in the control of eating and drinking.

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3. Moreover, section of the infraorbital branch of the trigeminal nerve (vibrissae denervation) produces the hunched posture and lowered snout seen after electrolytic or neurochemical lesions in the rat. Because these deficits involve peripheral manipulations of trigeminal sensory structures they reinforce our conclusion that the syndrome we described is of trigeminal rather than nigrostriatal origin (A. Marwine and H. P. Zeigler, paper presented at the meeting of the Eastern Psychological Association, held in New York City, April 1975).
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17 July 1975

Aerosols and Polar Temperature

Reck (*1*) considers that some workers (*2*, *3*) have incorrectly attributed surface cooling at high latitudes to increases in atmospheric dust there. She concluded "that aerosol particles over the polar regions have not been responsible for the ice mass increase in the Arctic, since they always have a heating effect."

Her conclusion was predestined, however, by her decision to consider a dust layer introduced "in the lowest 0.75 km of the atmosphere," an unrealistic way to consider the effect of global dust. In polar regions such dust is introduced near the top of the troposphere and deposited with snow at the surface, not vice versa. I would agree with her result as applied to dust generated at the surface, but this is a relatively rare occurrence at high latitudes.

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20 August 1975

The original choice of a low-lying aerosol layer (*1*) was based on the results of balloon soundings by Hofmann *et al.* (*2*) for 85°N, 3 December 1971 and 6 December 1972. Hofmann *et al.*'s measurements indicated that aerosol abundances at altitudes lower than 4 to 5 km were as much as five times greater than abundances at higher altitudes.

We have performed additional calculations for 85°N in July with a high-altitude aerosol layer. For the present background aerosol abundance (turbidity corresponding to an optical density of 0.065) and also for ten times the amount, heating is obtained when the aerosol layer is located near the tropopause (between 8.4 and 12.1 km).

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