wood, which predicts the delivery of food. The form of contact with the predictive stimulus cannot be predicted from the stimulus substitution hypothesis (*12*), but appears to depend upon the nature of both the predictive stimulus and the reward. As an alternative to stimulus substitution, we offer the hypothesis that auto-shaped behavior reflects the conditioning of a system of species-typical behaviors commonly related to the reward. The form of behavior in the presence of the predictive stimulus will depend on which behaviors in the conditioned system are elicited and supported by the predictive stimulus (*13*).

The present approach may have implications for the interpretation of traditional classical conditioning phenomena as well (*2*). Lorenz (*14*) suggested a related hypothesis to explain Liddell's unpublished observations on salivary conditioning in dogs. While working in Pavlov's laboratory, Liddell unharnessed a dog from the conditioning apparatus and allowed it to approach the predictive stimulus. On the basis of Liddell's description, Lorenz identified the dog's behavior toward the predictive stimulus as food begging in the genus *Canis*. Lorenz used this illustration to suggest that an entire behavior system was conditioned by the procedures of classical conditioning, not just an isolated reflex. Our results support this viewpoint.

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References and Notes

1. P. L. Brown and H. M. Jenkins, *J. Exp. Anal. Behav.* **11**, 1 (1968).
2. E. Hearst and H. M. Jenkins, *Sign Tracking: The Stimulus-Reinforcer Relation and Directed Action* (Psychonomic Society, Austin, Tex., 1974).
3. G. B. Peterson, J. Ackil, G. P. Frommer, E. Hearst, *Science* **177**, 1009 (1972).
4. B. R. Moore, in *Constraints on Learning*, R. A. Hinde and J. Stevenson-Hinde, Eds. (Academic Press, New York, 1973); H. M. Jenkins and B. R. Moore, *J. Exp. Anal. Behav.* **20**, 163 (1973).
5. E. A. Wasserman, *Science* **181**, 875 (1973).
6. J. A. Hogan, *ibid.* **186**, 156 (1974).
7. A. G. Bayroff and K. E. Land, *J. Comp. Physiol. Psychol.* **37**, 164 (1944); R. F. Ewer, *Anim. Behav. Monogr.* **4**, 140 (1972); B. G. Galef, Jr., and M. M. Clark, *J. Comp. Physiol. Psychol.* **75**, 341 (1971); F. Steiniger, *Z. Tierpsychol.* **7**, 356 (1950).
8. B. Latane, D. Poor, L. Sloan, *Psychonomic Sci. Sect. Anim. Physiol. Psychol.* **26**, 15 (1969); J. B. Calhoun, *U.S. Public Health Serv. Publ. No. 1008* (1962); E. C. Grant, *Behaviour* **21**, 260 (1963); _____ and J. H. Mackintosh, *ibid.*, p. 256.
9. R. A. Rescorla, *Psychol. Rev.* **74**, 71 (1967).
10. G. B. Peterson, thesis, Indiana University (1972).
11. Orient: point nose at predictive stimulus for at least 1 second; Approach: move nose to within ½ inch (1.3 cm) of predictive stimulus; Sniff: rhythmically move vibrassae and nose within ¼ inch (0.6 cm) of predictive stimulus; Bite: contact predictive stimulus with open mouth and teeth; Social Contact: paw: contact the stimulus with one or both forepaws; groom: lick and comb the fur of the stimulus using the mouth and paws; crawl-over: climb on top of the stimulus with at least three legs off the floor of the cage; anogenital sniff: sniff directed at the anogenital region of the stimulus rat, typically with the head oriented sideways. For a more complete description of the last three categories see Grant and Mackintosh (*8*). Grant and Mackintosh also coded "investigate" and "nose" as social contact behaviors. We lumped these as Sniff and did not count them as Social Contact because we felt the rat looked the same sniffing the platform, the wood, and the stimulus rat. In retrospect, the orientation of "nose" to the head of the stimulus rat potentially made it a distinct category.
12. We do not intend to argue that stimulus substitution effects were absent in the present experiment. Some behaviors shown by the CS^+ animals are readily interpretable as stimulus substitution. Two animals occasionally bit and pulled at the fur of the predictive animal while grooming it [although so did two CS^s animals; see aggressive groom in Grant and Mackintosh (*8*)]; one CS^+ animal bit the platform 12 times, and another animal bit it once (out of 330 acquisition trials). Further, it might be argued that some movements in normal groom and paw resemble components of feeding behavior, thus, some of the increase in these behaviors might be attributable to stimulus substitution. However, even a broadly conceived concept of stimulus substitution does not appear sufficient to explain all behavior in the present experiment. On the basis of stimulus substitution it is difficult to account for the failure of the CS^w animals to bite the wood, the failure of the CS^+ animals to bite the predictive rat, the low incidence of aggressive groom and platform biting in the CS^+ animals and their marked differential increase in crawl-over, a behavior sharing almost no topographical features with feeding. We feel that effects interpretable as stimulus substitution and these apparent exceptions all can be explained within the behavior system approach outlined in the text and the note in (*13*).
13. A system of behavior may be viewed as a collection of species-typical sensorimotor mechanisms potentially sharing common causal factors [see, for example, G. P. Baerends, R. H. Drent, P. Glass, H. Groenwald, in *The Herring Gull and Its Egg*, G. P. Baerends and R. H. Drent, Eds. (Brill, Leiden, 1970) or J. A. Hogan, *Behaviour* **39**, 129 (1971)]. The concept is similar to a species-typical central motive state [C. T. Morgan, in *Psychology: A Study of a Science*, S. Koch, Ed. (McGraw-Hill, New York, 1959), vol. 1, p. 644; D. Bindra, *Psychol. Rev.* **82**, 199 (1974)]. Bindra also discussed the importance of characteristics of the CS in supporting behavior typically elicited by the US, as did Mackintosh [N. J. Mackintosh, *The Psychology of Animal Learning* (Academic Press, New York, 1974), p. 108].
14. K. Lorenz, in *On the Biology of Learning*, K. H. Pribram, Ed. (Harcourt, Brace and World, New York, 1969), p. 47.
15. We thank M. Reitman for inspiration and D. Cassidy for assistance. This research was supported in part by NSF grant GB 36209 and a part of it was included in an undergraduate honors thesis at Indiana University (1974) by D.L.G.

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Effects of Long-Term Corn Consumption on Brain Serotonin and the Response to Electric Shock

Abstract. *Rats fed tryptophan-poor corn diets have reduced levels of brain serotonin and show increased responsiveness to electric shock. This diet-induced hyperalgesia can be reversed by feeding the animals diets with adequate amounts of tryptophan, or by systemic injections of the amino acid.*

Protein-calorie malnutrition experienced early in life may cause long-term deficits in learning and other behavioral capacities of humans and animals. The general behavioral sequels to extreme kwashiorkor or marasmus are well documented; however, the specific alterations in brain function that underlie behavioral changes induced by malnutrition are poorly understood. Our report is one of the first to describe an animal model that relates these behavioral changes induced by malnutrition to the effects of the diet on a brain neurotransmitter.

The concentration of the putative brain neurotransmitter, serotonin, appears to be related directly to the brain and plasma concentrations of the amino acid precursor tryptophan (*1*). Mammals cannot synthesize tryptophan; hence, all of the amino acid must be derived from dietary protein. The quality of dietary protein ingested is important for the maintenance of normal blood tryptophan levels, and ultimately for normal concentrations of brain tryptophan and brain serotonin. For example, plasma and brain tryptophan, and brain serotonin, are greatly diminished in animals fed a diet in which corn, a poor source of tryptophan, is the only protein (*2, 3*).

Changes in brain serotonin after various surgical or pharmacological manipulations

Table 1. Effects of long-term corn consumption and tryptophan injection on brain tryptophan and 5-hydroxyindole levels. Rats were placed on corn (tryptophan deficient) or 18 percent casein control diets for 42 days beginning at weaning. Then, different groups of rats were fasted for 18 hours, injected intraperitoneally with the H_2O vehicle or one dose of L-tryptophan, and killed 1 hour later. All values are means ± the standard error (N = 4 to 7).

Diet	Vehicle		L-Tryptophan at:		
	Free access	Fasted	62.5 mg/kg	125 mg/kg	250 mg/kg
	Brain tryptophan (μg/g)				
Casein	3.8 ± 0.2	5.1 ± 1.1	27.4 ± 2.6	47.8 ± 7.4	102.1 ± 9.9
Corn	1.8 ± 0.1†	5.5 ± 0.7	57.5 ± 0.9†	81.1 ± 12.2†	164.2 ± 8.5†
	Brain serotonin (μg/g)				
Casein	0.32 ± .01	0.32 ± .01	0.44 ± .02	0.50 ± .03	0.50 ± .02
Corn	0.25 ± .02*	0.31 ± .02	0.50 ± .02	0.53 ± .03	0.52 ± .01
	Brain 5-hydroxyindoleacetic acid (μg/g)				
Casein	0.27 ± .01	0.31 ± .01	0.55 ± .03	0.53 ± .04	0.66 ± .02
Corn	0.23 ± .02	0.43 ± .02*	0.79 ± .05†	0.81 ± .05†	0.88 ± .03†

*$P < .05$. †$P < .01$ compared to the appropriate casein-fed group.

are correlated with alterations in physiology and behavior. For instance, Harvey and his co-workers (*4*) have found hyperalgesic responses to painful stimuli following electrolytic lesions that reduce forebrain concentrations of serotonin in rats. Pharmacological reductions in brain serotonin following the administration of the tryptophan hydroxylase inhibitor parachlorophenylalanine are also associated with lower response thresholds to painful stimuli (*5*). The hyperalgesia and decreased levels of brain serotonin can be reversed in these pharmacological and surgical preparations by treating animals with 5-hydroxytryptophan, the immediate precursor of serotonin (*5, 6*).

Changes in electroshock sensitivity are correlated with alterations, induced by lesions or drugs, in the levels of brain serotonin. Since the long-term consumption of a corn diet also reduces brain serotonin, we hypothesized that animals with such a diet might also show changes in electroshock sensitivity. We now report that diet-induced reductions in the concentrations of brain serotonin are correlated with lower response thresholds to electric shock; this increased sensitivity can be partially reversed by nutritional rehabilitation, or by the acute administration of L-tryptophan.

In all experiments, weanling, 21-day-old male rats, born and reared in the Massachusetts Institute of Technology vivarium (from a parent stock of Sprague-Dawley CD-derived rats, Charles River Laboratories), were housed by litter and given free access to water and a corn-based diet poor in tryptophan, or to an 18 percent casein control diet (*7*). Animals were maintained on a 12 : 12 light-dark cycle (Vita-Lite, DuroTest Corp.), throughout the experimental period. Flinch or jump responses to presentations of electric footshock were determined by a modification of the method of Evans (*8*).

In other experiments, animals were placed on the control casein diet or on the corn diet poor in tryptophan for various periods of time. They were then killed to determine the extent to which the levels of brain tryptophan, serotonin, or the deaminated metabolite, 5-hydroxyindoleacetic acid (5-HIAA), were changed as a result of the diet. Brains were removed and bisected midsagittally; half of each brain was homogenized in water and assayed for tryptophan (*9*). The other half of each brain was homogenized in acidified butanol and centrifuged at 28,000*g*; the supernatants were then assayed for serotonin and 5-HIAA (*10*).

The magnitude of shock necessary to elicit a flinch or a jump response in animals that were fed the normal 18 percent casein diet increased slightly over the 14-week testing period—from a mean of 0.11 ± 0.01 ma for the flinch response and 0.57 ± 0.02 ma for the jump response on the first day of testing (at age 3 weeks), to 0.17 ± 0.02 ma and 0.69 ± 0.05 ma, respectively, on the final day of testing with the experimental diets (at 17 weeks of age). These slight increases in the baseline shock intensities were probably due in part to the substantial weight gain in the control group that occurred over the testing interval (*11*). In contrast, animals fed the diet poor in tryptophan showed decreased flinch and jump thresholds to the electric shock beginning as early as the fifth postnatal week—that is, after 2 weeks on the corn diet (Fig. 1). The corn-fed animals remained substantially more sensitive to the shock for the remaining 8 weeks that they consumed the diet. The brains of animals given the corn diet were deficient in tryptophan and serotonin; brain tryptophan was reduced to 45 percent of the control concentration, whereas brain serotonin was reduced to approximately 75 percent of control (Table 1). This finding replicates the earlier work of Fernstrom and Wurtman (*2, 3*). Brain 5-HIAA did not differ significantly between the two groups. The results of this initial experiment show that animals maintained on diets deficient in tryptophan have lower response thresholds to electric shock than do normally fed animals. This hyperalgesia is correlated with reductions in brain tryptophan and serotonin concentrations.

Table 2. Effects of various drugs on diet-induced changes in electroshock sensitivity. Rats were starved overnight (18 hours) and injected with the drug 1 hour prior to testing. All values are mean shock intensities (ma) ± S.E.M. (N = 8).

Treatment	Dose mg/kg	Shock intensity (ma) after diet of	
		Corn	Casein
Tryptophan	0	0.43 ± .02	0.85 ± .05
	62.5	0.54 ± .06	0.95 ± .04
	125	0.69 ± .02*	0.78 ± .06
	250	0.63 ± .04*	0.88 ± .04
L-Dopa	0	0.43 ± .03	0.87 ± .05
	100	0.42 ± .03	0.75 ± .05
	250	0.43 ± .02	0.75 ± .08

*P < .01 compared to appropriate vehicle-injected control group.

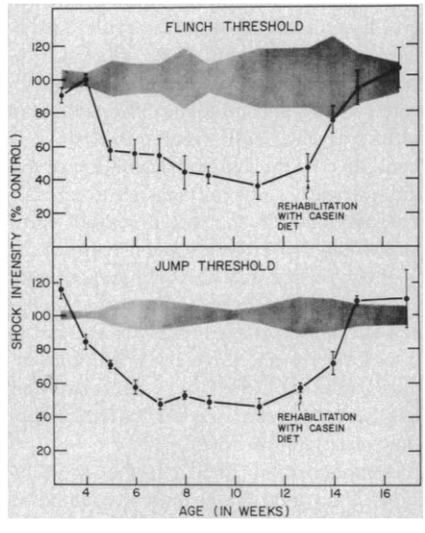

Fig. 1. Animals were placed on tryptophan-deficient corn diets (closed circles, solid lines) or on amino acid balanced casein control diets (shaded areas) beginning at week 3 after birth. After 10 weeks on the corn diet, rats were rehabilitated by feeding them the 18 percent casein diet (beginning at week 13 after birth). All values are the percentages (means ± S.E.) of the shock intensities in the controls for eliciting the flinch or the jump response.

In subsequent experiments, we determined the extent to which the diet-induced changes in electroshock thresholds could be reversed in corn-fed animals by nutritional rehabilitation, or by a single injection of tryptophan. After 10 weeks of free access to the corn diet, the animals deficient in tryptophan were rehabilitated by offering them the 18 percent casein diet (this diet contains 0.16 μg of tryptophan per gram of food, compared with 0.02 μg per gram in the corn diet). The flinch and jump response thresholds of the corn-fed animals returned to normal within 2 weeks after exposure to the diet adequate in tryptophan (Fig. 1). This behavioral rehabilitation is temporally correlated with the return to normal of brain tryptophan and serotonin levels in these animals (*12*).

Other animals given the corn or casein diets for 6 weeks were fasted overnight and then injected with a single dose of L-tryptophan (62.5, 125, or 250 mg/kg, intraperitoneally, in a volume of 3 ml/kg); control animals received the H_2O vehicle brought to *p*H 10 with NaOH, or various doses (125 or 250 mg/kg) of L-dopa, the amino acid precursor for the catecholaminergic neurotransmitters. L-Dopa was administered to determine whether other amino acids might also change the response thresholds to the shock. All response thresholds were determined 1 to 1.5 hours after injection. We again observed that the jump response could be elicited in animals consuming the corn diet by approximately half the shock intensity needed to elicit it in casein-fed rats (Table 2). Injections of L-tryptophan or L-dopa had no effect on the shock thresholds of animals consuming the casein diet; however, the tryptophan injections did significantly increase the shock threshold for the jump response among animals consuming the corn diet. That the tryptophan-induced increase in the electroshock threshold was related specifically to its effects on brain serotonin is suggested by the finding that

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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References and Notes

1. J. D. Fernstrom and R. J. Wurtman, *Science* **174**, 1023 (1971); A. Tagliamonte, G. Biggio, G. L. Gessa, *Riv. Farmacol. Terapia* **11**, 251 (1971); R. J. Wurtman and J. D. Fernstrom, in *Perspectives in Neuropharmacology*, S. H. Snyder, Ed. (Oxford Univ. Press, New York, 1972), pp. 143–193; P. J. Knott and G. Curzon, *Nature (Lond.)* **239**, 452 (1972); J. D. Fernstrom and R. J. Wurtman, *Science* **178**, 414 (1972); B. K. Madras, E. L. Cohen, R. Messing, H. N. Munro, R. J. Wurtman, *Metab. (Clin. Exp.)* **23**, 1107 (1974).
2. J. D. Fernstrom and R. J. Wurtman, *Nat. New Biol.* **234**, 62 (1971).
3. ———, *Sci. Am.* **230**, 84 (1974); R. J. Wurtman and J. D. Fernstrom, *Nutr. Abstr. Rev.* **32**, 193 (1974).
4. J. A. Harvey and C. E. Lints, *Science* **148**, 250 (1965); C. E. Lints and J. A. Harvey, *J. Comp. Physiol. Psychol.* **67**, 23 (1969); S. A. Lorens, J. P. Sorensen, J. A. Harvey, *ibid.* **73**, 284 (1970); J. A. Harvey and C. E. Lints, *ibid.* **74**, 28 (1971); L. M. Yunger and J. A. Harvey, *ibid.* **83**, 173 (1973).
5. S. S. Tenen, *Psychopharmacologia* **10**, 204 (1967); H. C. Fibiger, P. H. Mertz, B. A. Campbell, *Physiol. Behav.* **8**, 259 (1972).
6. C. E. Lints and J. A. Harvey, *Physiol. Behav.* **4**, 29 (1969); J. A. Harvey and L. M. Yunger, in *Serotonin and Behavior*, J. Barchas and E. Usdin, Eds. (Academic Press, New York, 1973), pp. 179–189.
7. The composition of the diets was the same as that in (*2*). The 18 percent casein diet consisted of 180 g of casein, 207 g of dextrose, 167 g of sucrose, 207 g of dextrin, 150 g of Mazola oil, 40 g of Harper's salt [Q. Rogers and A. E. Harper, *J. Nutr.* **87**, 267 (1965)], 10 g of a vitamin mix [R. J. Wurtman, W. J. Shoemaker, F. Larin, *Proc. Natl. Acad. Sci. U.S.A.* **59**, 800 (1968)], 2 ml of 50 percent choline, 35 g of agar, and 1000 ml of H_2O. The corn diet was similar to the casein diet except that it contained 800 g of 80 percent dry enriched white Masa Harina corn (provided by the Quaker Oats Co., Corn Products Division, Chicago, Ill.) in place of the casein, dextrose, sucrose, dextrin, and agar. The corn contained 7 percent protein.
8. W. O. Evans, *Psychopharmacologia* **2**, 318 (1961). Briefly, rats were introduced into the sound-attenuated testing apparatus during the light portion of the day-night cycle. Each animal was given six alternating ascending and descending series of ten shocks of 0.2-second duration. The shocks (0.05, 0.1, 0.2, 0.3, 0.4, 0.6, 0.8, 1.0, 1.3, and 1.6 ma) were delivered from a constant current power source through a scrambler and were presented at 30-second intervals, with an interseries interval of 2 minutes. Rats were observed for the presence of a flinch (any observable crouch or startle response) or a jump response (a response in which the two hind paws left the grid floor simultaneously with the onset of shock). The probability of occurrence of a flinch or a jump response at each shock intensity, for each rat, was converted to a *z*-score, and a straight line was fitted by the method of least squares. The shock thresholds were defined as those shock intensities, interpolated from the fitted curves, that elicited a flinch or a jump response 50 percent of the time.
9. W. D. Denckla and H. K. Dewey, *J. Lab. Clin. Med.* **69**, 160 (1967).
10. G. Curzon and A. R. Green, *Br. J. Pharmacol.* **39**, 653 (1970).
11. The tryptophan-poor corn diet was also a low-protein diet; hence, the growth rates of animals consuming this diet were retarded. Whereas the body and wet brain weights of the corn-fed animals were 66 ± 2 g and 1.53 ± 0.017 g after 6 weeks of eating the diet, the body and brain weights of the animals consuming the 18 percent casein diet were 231 ± 6 g and 1.84 ± 0.15 g, respectively.
12. M. F. Nelson and J. D. Fernstrom, unpublished observations.
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.
14. J. D. Fernstrom and M. J. Hirsch, *Life Sci.* **17**, 455 (1975).
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).
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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-