The temporal lobes have been suspected to play a role in feeding behavior ever since Brown and Schafer (9) reported in 1888 that temporal lobectomies in two monkeys resulted in “insatiable” appetites. Later studies confirmed hyperphagia as well as obesity in monkeys and humans with bilateral temporal lobe damage or temporal lobectomies (e.g., 10, 46, 55, 67), and similar results were observed in monkeys given bilateral resections of the amygdaloid complex and adjacent temporal cortex (62).

Subsequent lesion studies in dogs and cats established that the critical site was indeed the amygdala (20, 24, 41, 49, 50, 72). However, there was disagreement regarding the specific nuclei involved. Three studies determined that the damage caused by obesity-inducing lesions lie in the lateral nuclei or the junction between the lateral and basal nuclei (20, 24, 50). Fonberg (19–22) found that lesions of the lateral nuclei, particularly those in the ventral posterior portions, produced hyperphagia and obesity in dogs, whereas dorsomedial lesions (i.e., central and medial nuclei) resulted in aphagia and weight loss. Wood (72) and Koikegami et al. (41), on the other hand, reported marked hyperphagia in cats given lesions of the medial and central nuclei. Anand and Brobeck (4) observed no change in food intake in cats with lesions of either the lateral amygdala or the central and medial amygdala.

Numerous studies have examined the effects of amygdaloid lesions on food intake and body weight in rats, resulting in a similar collection of inconsistent and contradictory results. Initial studies that employed large lesions that destroyed most of the amygdala reported hypophagia and weight loss (4, 12, 41, 56, 61, 64, 65). Subsequent studies of rats with smaller lesions aimed at the basolateral and/or lateral nuclei reported weight gain (8, 23, 49), no change in body weight (18, 28, 40, 45, 58, 60), or even weight loss (14). Studies employing lesions of the corticomedial nuclei were equally mixed, reporting weight gains (26), weight losses (12, 58), or no changes in body weight (59, 60, 63). Central nucleus lesions have similarly been reported to result in either weight/fat gain (7, 42), aphagia and/or weight loss (8, 11, 23), or no change in food intake and body weight (16, 29, 57, 59, 60). In recent years, King and colleagues (30–33, 35–40) have reported hyperphagia and moderate obesity in female rats given bilateral lesions of the most posterdorsal aspects of the amygdala, with weight gains of 50–80 g typical in the first 15 to 20 days after lesions. The critical nuclei were determined to be the posterdorsal medial nucleus and the intra-amygdaloid bed nucleus of the stria terminalis (G. F. Alheid, B. M. King, J. T. Cook, K. N. Rossiter, B. L. Rollins, S. J. Shammah-Lagnado, unpublished observations).

This confusing array of results has deterred some researchers from further exploring the role of the amygdala in food intake and regulation of body weight (personal communications). To better understand the effects of various amygdaloid lesions on body weight, the present study reexamined the effects of electrolytic
lesions of the amygdala in female rats. Common areas of overlap were examined to help identify the area of damage critical for weight gain.

METHODS

Animals. A total of 128 adult (110–130 days old) female Long-Evans hooded rats were used (Harlan Sprague-Dawley, Indianapolis, IN). Females were used because the large majority of studies of the effects of ventromedial hypotalamic lesions on food intake and body weight used female rats. All animals were individually housed in standard rat cages (9.5 in. long 7 in. wide 7 in. high; no activity wheel) in a temperature-controlled colony (21–24°C) with a 12:12-h light-dark cycle throughout the experiment.

Lesions. Bilateral electrolytic lesions were produced under pentobarbital sodium anesthesia (50 mg/kg) by passing anodal current between the uninsulated tip of an insulated stainless steel electrode (Plastics One, Roanoke, VA) and a rectal cathode. Posterodorsal lesions and central nucleus lesions were performed with a 1.5-mA current produced for 20 s with 0.2 (posterodorsal) or 0.4 mm (central) of the electrode tip uninsulated. Basolateral amygdaloid lesions were produced with a 2.0-mA current for 30 s with 0.9 mm of the electrode tip uninsulated. Electrodes were positioned relative to bregma with the use of a Kopf small animal stereotaxic instrument. With the upper incisor bar positioned horizontally with the interaural line, the electrodes for posterodorsal lesions were positioned 1.7 mm posterior to bregma (AP), 4.5 mm lateral to the mid sagittal suture (ML), and 8.4 mm below the surface of the skull (DV). For basolateral lesions, the coordinates were (in mm) AP −0.8, ML 4.7, and DV 9.4. For lesions involving a greater portion of the central nucleus, the coordinates were (in mm) AP −0.4, ML 4.2, and DV 8.4. Control animals had holes drilled in the skull at the same coordinates, and electrodes were lowered to a depth 1.0 mm above the target site.

Procedure and histology. The study compared four groups of animals: sham lesions (n = 29), posterodorsal amygdaloid lesions (n = 47), basolateral amygdaloid lesions (n = 37), and lesions aimed at the central nucleus (n = 15). Rats with sham, posterodorsal, or basolateral lesions were used in a subsequent behavioral study (to be reported separately), which required that 21 of the animals with posterodorsal lesions be food restricted starting on day 4. Except for being weighed daily, all the other animals (including 26 rats with basolateral posterodorsal lesions) were undisturbed for 10 days. All animals were allowed to feed ad libitum (Harlan Teklad pellet diet) during the 3- or 10-day observation period reported here.

At completion of the study, the rats with lesions were killed and perfused with physiological saline and a 10% Formalin solution. The brains were stored in 10% Formalin and later frozen and sliced into 40-μm coronal sections. The sections were stained with cresyl violet, and initial histological analysis was performed in a blinded fashion (i.e., without knowing changes in body weight) by light microscopic examination. The extent of the lesions was determined with use of the stereotaxic atlas by Paxinos and Watson (54).

RESULTS

Sham lesions. Of the 29 rats with sham lesions observed in this study, 21 weighed less than they had preoperatively on day 3 (mean change ± SE = −5.8 ± 1.1 g), and only 16 weighed more than they had preoperatively by the tenth day after surgery. The mean 10-day weight change was +1.7 ± 1.8 g, and the greatest weight gain observed was 18 g (see Table 1).

Posterodorsal lesions. “Posterodorsal” refers to an area, not a specific nucleus (analogous to ventromedial hypothalamus vs. ventromedial hypotalamic nuclei). Four serial sections of six lesions are provided in Fig. 1. In terms of weight gain, the most effective lesions were centered at their maximal point of damage immediately ventral and adjacent to the dorsal tip of the optic tract in the posterior aspects of the amygdala. With small lesions (of which all 6 in Fig. 1 are examples), the posterior extent of the damage fuses into (and eventually becomes indistinguishable from) the lateral ventricle. In the case of larger lesions or lesions that begin more posterior than those in Fig. 1, the damage normally extends into the amygdalohippocampal area and ventral hippocampus. However, these lesions are less effective in producing weight gain (see Ref. 30). Weight gain was also attenuated whenever the damage extended into the ventral aspects of the caudal globus pallidus.

An examination of Fig. 1 reveals that lesions in the most posterodorsal aspects of the amygdala do not have to be large to produce weight gain. For example, rat 1F sustained the smallest lesions yet displayed the largest weight gain (63 g/10 days). The lesion illustrated in Fig. 1A is also remarkable for its small size and the substantial weight gain it produced. Histological analysis of 32 rats sustaining small lesions that produced substantial weight gains revealed that the areas of damage they shared in common were in the posterodorsal medial amygdaloid nucleus and the bed nucleus of the stria terminals. There were varying

<table>
<thead>
<tr>
<th>Group</th>
<th>Initial Weight</th>
<th>Days 1–3</th>
<th>Days 4–10</th>
<th>Day 3</th>
<th>Day 10</th>
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</thead>
<tbody>
<tr>
<td><strong>Sham lesions</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>29</td>
<td>279.2 ± 3.4</td>
<td>18.9 ± 0.6</td>
<td>22.3 ± 0.6</td>
<td>−5.8 ± 1.1</td>
<td>1.7 ± 1.8</td>
</tr>
<tr>
<td>PDA</td>
<td>271.0 ± 3.8</td>
<td>31.0 ± 0.3</td>
<td>34.6 ± 1.2</td>
<td>22.8 ± 1.9</td>
<td>45.4 ± 2.4</td>
</tr>
<tr>
<td><strong>Basolateral nuclei</strong></td>
<td></td>
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<tr>
<td>Basolateral</td>
<td>275.4 ± 3.4</td>
<td>22.3 ± 2.0</td>
<td>25.2 ± 1.3</td>
<td>0.8 ± 1.6</td>
<td>9.8 ± 2.6</td>
</tr>
<tr>
<td>Basolateral + partial PDA</td>
<td>287.2 ± 2.4</td>
<td>26.9 ± 2.3</td>
<td>31.9 ± 2.5</td>
<td>0.0 ± 0.6</td>
<td>21.0 ± 1.4</td>
</tr>
<tr>
<td>Basolateral + PDA</td>
<td>280.9 ± 9.4</td>
<td>30.2 ± 5.5</td>
<td>33.5 ± 4.8</td>
<td>16.7 ± 8.6</td>
<td>32.0 ± 7.1</td>
</tr>
<tr>
<td>Central nuclei + PDA</td>
<td>286.6 ± 7.1</td>
<td>31.5 ± 4.0</td>
<td>30.6 ± 2.0</td>
<td>16.8 ± 3.1</td>
<td>22.7 ± 3.6</td>
</tr>
</tbody>
</table>

Values are mean grams ± SE. Posterodorsal area (PDA) data are only for those rats with small bilateral lesions that were fed ad libitum for 10 days.
degrees of damage to the medial and dorsal portions of the posterior basomedial nucleus and the caudal portions of the central nucleus. The lesions were also in the immediate area where axons come together to form the stria terminalis. Examination of the smallest lesions (e.g., Fig. 1, A and F) revealed that there was greater damage to the bed nucleus than to the posterodorsal medial nucleus. For 11 rats with very small bilateral lesions that were fed ad libitum for 10 days, the mean weight gain was 22.8 ± 1.9 g after 3 days and 45.4 ± 2.4 g after 10 days. Daily food intakes reflected weight gains (see Table 1).

Central and medial lesions. Seven of fifteen animals had extensive bilateral damage to the central nucleus. Four serial sections through the brains of three of these rats are displayed in Fig. 2 (A, B, C). Note that the lesions are larger than those observed in rats with posterodorsal lesions (Fig. 1). The lesions for rats A, B, and C began more anterior than the lesions displayed in Fig. 1 and included much more extensive damage to the central nucleus. But because the lesions were larger, the damage continued posterior to include much of the area destroyed by posterodorsal lesions, i.e., posterodorsal medial nucleus, bed nucleus of the stria terminalis, and eventual fusion with the lateral ventricle. However, all three lesions invaded the central aspects of the globus pallidus to varying degrees. This was especially true of the lesions in rat B. Despite this, the weight gain for the seven animals with bilateral damage to the central nuclei (and varying degrees of damage to the posterodorsal amygdala) still exceeded those observed after sham lesions (mean = 22.7 ± 3.6 g/10 days).

Rat 2D had the greatest bilateral damage to the medial nucleus of any rat observed. The damage included the anterodorsal division of the medial nucleus in anterior sections, the posterodorsal division and bed nucleus of the stria terminalis in posterior sections, and complete fusion with the lateral ventricle. There was no damage to the globus pallidus. However, weight gain (34 g/10 days) was no greater in this rat than in rats with much smaller lesions limited to the posterodorsal amygdaloid nucleus and bed nucleus (Fig. 1).

Basolateral lesions. Because of the amount of electrode tip exposed and duration of current, lesions aimed at the basolateral nuclei were large. However, there was considerable variation in the extent of the
damage. Of 37 animals with lesions, 12 had lesions that, either unilaterally or bilaterally, extended well lateral to (and greatly spared) the basolateral nuclei. Another three had lesions that were too posterior, and in two others the damage spared over half of the nuclei ventrally. The data for these 17 animals were eliminated, leaving 20 animals with extensive bilateral damage to the basolateral nuclei. However, among these 20 animals, there was still considerable variation in the dorsal-ventral extent of the damage, with varying amounts of damage to the lateral, basolateral, and basomedial amygdaloid nuclei. Brain sections of eight representative rats with basolateral lesions are presented in Fig. 3.

The 10-day weight changes in the 20 rats with bilateral basolateral lesions ranged from 24 to 144 g. In the initial examination of the brain sections, it was immediately apparent that weight gain was directly related to the dorsal-most extent of the damage in any one hemisphere. Sections were enlarged with a microprojector, and the distance measured between 1) the most dorsal point of the lesion and 2) the dorsal tip of the optic tract within the same coronal plane in which the most effective posterodorsal lesions were observed. There was a significant negative correlation between this distance and 10-day weight gain ($r = -0.81, df = 19, P < 0.001, r^2 = 0.66$). For three of the rats with basolateral lesions, damage to the posterodorsal amygdala was nearly identical (unilaterally or bilaterally) to that observed in rats with intentional posterodorsal lesions (e.g., Fig. 3C). Their mean 10-day weight gain was 32 g. Five additional rats had damage that extended far enough dorsally (in at least 1 hemisphere) to damage large portions of the posterodorsal medial nucleus and the bed nucleus of the stria terminals (e.g., Fig. 3C). The mean 10-day weight gain for these five rats was 21 g.

For the remaining 12 rats, mean 3- and 10-day weight gains were 0.8 ± 1.6 and 9.8 ± 2.6 g, respectively. The 10-day weight gains (range = −4 to +23 g) were significantly greater than those observed in rats with sham lesions ($t = 2.52, df = 39, P < 0.01, d = 0.86$). However, even among this group, the best weight gains were observed in those animals in which lesions were large enough to extend far enough posterior to infringe on the most posterior aspects of the bed nucleus just ventral to the lateral ventricle.

**DISCUSSION**

The weight changes that were observed in female rats given sham lesions were similar to those that had previously been reported. Adult female rats almost always lose weight in the first 3 to 5 days after sham lesions, and many do not exceed their preoperative weights by day 10 (30, 33, 35–37, 40). Against this backdrop, the initial weight gain of female rats with posterodorsal amygdaloid lesions is impressive. Although female Long-Evans rats generally take 6 to 10 h to become fully ambulatory after pentobarbital sodium anesthesia, rats with posterodorsal lesions often display a weight gain in the first 24 h after surgery. In the present study, the initial mean 3-day gain of 22.8 g computes to a net difference of +28.6 g compared with the mean loss of −5.8 g displayed by rats with sham lesions. The 3- and 10-day weight gains of the
Rats with posterodorsal lesions were comparable to the gains that have often been reported for female rats with lesions of the paraventricular hypothalamic nucleus (5, 68–70). The weight gains were less than those generally observed after lesions of the ventromedial hypothalamus (e.g., 34). Weight gain in rats with posterodorsal amygdaloid lesions generally plateaus 15 to 20 days after surgery, compared with 3 to 6 wk for rats with well-placed lesions of the ventromedial hypothalamus (34).

Analysis of the brains of rats with the smallest posterodorsal lesions indicated that the critical area for weight gain included the posterodorsal medial amygdaloid nucleus and the bed nucleus of the stria terminalis. Damage to these structures can clearly be seen in the serial sections of Fig. 1. In a recent study, Alheid et al. (unpublished observations) used multiple regression analysis to determine that damage to the intra-amygdaloid bed nucleus of the stria terminalis and the posterodorsal medial amygdaloid nucleus accounts for ~35% of the variance in weight gain after (larger) posterodorsal amygdaloid lesions. Examination of the smallest lesions in the present study revealed that there was little damage to other amygdaloid nuclei, and examination of the lesion of rat D in Fig. 2 suggests that more extensive damage to the medial nucleus does not further enhance weight gain. Others have also observed no weight gain in rats with lesions of the (posteroventral and anterodorsal) medial amygdaloid nuclei and/or cortical nuclei (28, 59, 60, 63). Alheid et al. (unpublished observations) found that damage to the caudal globus pallidus was negatively loaded in
predicting the weight gain (i.e., damage reduced weight gain). Together, damage to the posterodorsal medial amygdaloid nucleus, the intra-amygdaloid bed nucleus, and the globus pallidus accounted for 97% of the variability in weight gain.

Although damage produced by the smallest lesions was limited to the posterodorsal medial amygdaloid nuclei and bed nucleus of the stria terminalis, one must also keep in mind that axons are collecting throughout these areas to form the stria terminalis. Three studies reported that sectioning of the stria did not result in excessive weight gain in male rats (6, 8, 51), but these results may have been due to a sex difference in weight gain after amygdaloid lesions (37) and damage to the globus pallidus (6, 8), internal capsule (8), or cortex and dorsal striatum (51). Ehrlich (17) reported excessive weight gains in male rats given electrolytic lesions of the fornix (26% greater than controls in the first 3 days), but an examination of the lesions she made indicates that the damage was large enough to have almost certainly included the stria terminalis. Coronal knife cuts anterior to the ventromedial hypothalamic nuclei, a projection site for a major branch of the stria terminalis, have been found to result in hyperphagia and/or excessive weight gain in female rats and some male rats (25, 53, 66). Food intake was at least 150% of normal, and weight gains were at least double the normal rate. In addition, electrical stimulation of the medial portion of the amygdala suppresses food intake, and transection of the stria terminalis prevents the suppression (71). Examination of the effects of injection of a cellular neurotoxin (e.g., ibotenic acid) into the posterodorsal amygdala should be valuable in clarifying whether the effects of electrolytic lesions are due to damage to local nuclei or to fiber pathways.

The finding that incidental damage to the globus pallidus is negatively correlated with weight gain (Alheid et al., unpublished observations) is particularly pertinent when considering the role of the central nucleus. Box and Mogenson (8) and Ganaraj and Jeganathan (23), for example, reported that central nucleus lesions “drastically” decreased food intake and body weight. However, besides aphagia, their rats displayed sensory-motor deficits, including excessive gnawing and spillage of their food pellets. Others have reported similar results for rats, cats, and dogs (e.g., 4, 11, 19, 21). Aphagia is a well-established consequence of damage to the caudal globus pallidus (16, 44, 48, 52, 60). Previous studies had already established that lesions limited to the central nucleus, without incidental damage to the globus pallidus, do not result in hypophagia, weight loss, or sensory-motor deficits (7, 16, 29, 57, 60, see also Ref. 42). Obesity-inducing posterodorsal amygdaloid lesions often invade the caudal portions of the central nucleus, and the present results demonstrate that excessive weight gains are possible even in rats with much more extensive damage to the central nucleus (Fig. 2, A, B, and C). Box and Mogenson (8) also observed mild hyperphagia and weight gain in male rats with lesions that included damage to the ventral half of the central nuclei.

Ganaraj and Jeganathan (23) also reported that basolateral lesions resulted in small weight gains (mean = 23 g/3 wk) in young male rats, but their data are equally notable for a lack of male-typical weight gain by rats with sham lesions (mean = 2 g/3 wk). In our experience, young male rats with sham lesions begin to display sex-typical weight gains within days after surgery (37). Most studies that have examined the effects of bilateral electrolytic lesions of the basolateral nuclei in male rats did not find excessive weight gains (e.g., 14, 18, 45, 58, 60), although Box and Mogenson (8) reported hyperphagia and obesity in four male rats with lesions of the ventral basolateral nuclei in the most posterior aspects of the amygdala. Lenard et al. (43) observed a small increase in weight gain in male rats given 6-OHDA lesions of the lateral nucleus (~130% of preoperative weight compared with 124–125% for controls after 18 days).

At first glance, the results obtained with basolateral lesions in the present study appeared to support those of Ganaraj and Jeganathan (23). However, the lesions in the present study were quite large, and there was a high correlation between weight gain and the dorsal extent of the lesions. The most substantial weight gains were observed in rats with lesions that clearly invaded the posterodorsal medial amygdaloid nucleus and the bed nucleus of the stria terminalis dorsally in at least one hemisphere. Previous work has established that unilateral posterodorsal amygdaloid lesions result in weight gains equal to those produced by bilateral lesions during the first 2–3 days after surgery (32). [Unilateral ventromedial hypothalamic lesions also result in hyperphagia and obesity (47).] Nevertheless, when the data for these rats (e.g., Fig. 3, A, B, and C) were eliminated, the remaining 12 animals still displayed a small but statistically significant increase in weight gain compared with controls (9.8 vs. 1.7 g, respectively, in 10 days). However, even among these rats, weight gain was clearly related to the dorsal extent of the lesions. Compare, for example, the lesions of rats D and E (Fig. 3) with those of rats G and H. All four have extensive damage to the basolateral nucleus, but the lesions in rats D and E (which resulted in moderate weight gain) have a greater dorsal extension. Rats G and H displayed no excessive weight gains. This suggests that the small weight gains observed in these 12 animals may have been due to the damage infringing on the critical areas within the posterodorsal amygdala (i.e., posterodorsal medial nucleus/bed nucleus or ventral portion of the area where axons are coming together to form the stria terminalis). Two previous studies reported hyperphagia in rats with lesions of the ventral amygdala, but the effects were either very small (26) or transient [and observed only on a high-fat diet (8)]. The lesions in another study that resulted in mild hyperphagia were posterior and extended dorsally into the same region described here (15).

The excessive weight gains observed in the present study after a variety of differently placed amygdaloid
lesions (posterodorsal area, medial, basolateral, and even central nuclei) may be important in reconciling the discrepant results reported for other species. In dogs and cats, dorsomedial lesions (medial and central nuclei) have resulted in both aphagia/weight loss (4, 19, 21) and hyperphagia/obesity (41, 72), whereas lesions of the lateral or basolateral group of nuclei have resulted in no change (4) or hyperphagia and obesity (20, 21, 24, 49, 50). Inspection of published photographs of lesions suggests that the aphagia following lesions of the medial and central nuclei were probably due to additional damage to adjacent nonamygdaloid structures involved in sensory-motor functions [e.g., 19, 21 (Fig. 6, p. 458)].

Regarding hyperphagia and obesity, two possible explanations of the role of the amygdala arise: 1) much of the amygdala is inhibitory for feeding behavior and body weight or 2) the seemingly diverse lesions overlapped in some critical area. The first possibility seems unlikely in view of the vastly different embryonic development of the medial/central nuclei vs. the basolateral complex of nuclei (27). The results of the present study with rats support the second possibility. Substantial weight gains were observed only when lesions infringed on the most posterodorsal aspects of the amygdala, regardless of whether the lesions were centered in the basolateral group of nuclei, the medial nucleus, or the central nucleus. Published photographs of brain sections for other species are limited, and one must also keep in mind the medial rotation of the amygdala as species ascend phylogenetically (27). However, an inspection of the basolateral lesions in dogs [22 (Fig. 5, p. 115)] reveals extension dorsally into a common area near the dorsal tip of the optic tract. The lesions spared the medial nucleus, but the fibers of the stria terminalis still collect in this area. In fact, in the figure legend of their photographs, Green et al. (24) state that in addition to damage to the lateral nucleus, the ventral part of the stria terminalis is injured. Lesions that did not result in weight gain were clearly more ventral to this [24 (Figs. 19 and 20, p. 543)], similar to G and H in Fig. 3 of the present study. Wood (72) did not publish photographs, but if the lesions involved the central nucleus as described, they, too, would have damaged the bed nucleus and stria terminalis. Morgane and Kosman’s [50 (Fig. 1, p. 159)] lesions were large and destroyed nearly all of the amygdala. Koikegami et al. [41 (Fig. 1, p. 215)] observed obesity in only one cat, but this medially placed lesion also infringed on the proposed critical area at the dorsal end of the optic tract.

We cannot rule out the possibility that lesions that spare the posterodorsal amygdala (i.e., posterodorsal medial nucleus, bed nucleus, and stria terminalis) result in small weight gains. In fact, should the critical structure prove to be the stria terminalis, then one would expect to observe some weight gain after lesions of nuclei from which axons of the stria terminalis originate. There is little evidence to support the cortical (posteroventral and anterodorsal), medial, or central nuclei. The best evidence favors the basolateral or lateral nuclei. However, it should be noted that in those few studies that found hyperphagia and/or excessive weight gain in rats with basolateral or lateral lesions, the effects were usually very small and the lesions were either very dorsal or very posterior (8, 15, 23, 26, 43, present study).

In conclusion, the present study identifies the critical site for lesion-induced obesity in rats to be the posterodorsal medial amygdaloid nucleus and the bed nucleus of the stria terminalis, as indicated by both very small lesions and as the point of overlap in variously placed large lesions (when there is no incidental damage to the globus pallidus). This is the same area in which axons come together to form the stria terminalis. Examination of obesity-inducing lesions in cats and dogs also suggests that the critical area is the bed nucleus and/or stria terminalis. With the determination of the critical amygdaloid site for lesion-induced obesity, future research can now be directed at understanding the etiology of this experimentally induced obesity.

**Perspectives**

Lesions of the posterodorsal amygdala in many ways mimic, although to a smaller extent, lesions of the ventromedial hypothalamus. This includes dynamic and static phases of hyperphagia and weight gain (33), hyperinsulinemia (31), and a greater magnitude of weight gain in females (37). Lesions of the posterodorsal amygdala in rats do, in fact, result in extensive anterograde degeneration in the stria terminalis and ventromedial hypothalamic nuclei, with little or no degeneration in the paraventricular hypothalamic nuclei (Alheid et al., unpublished observations; 32). Initial studies of putative neurotransmitter mechanisms suggest a possible role for serotonin (13). Alheid and colleagues (1, 2) include the posterodorsal amygdala as part of the medial corridor of the “extended amygdala.” However, posterodorsal amygdaloid lesions do differ notably from ventromedial hypothalamic lesions in some respects. This includes a strong preference for carbohydrates (39), but otherwise a lack of finickiness (38). Thus there is no exact duplication of feeding systems within the amygdala and medial hypothalamus. It is suggested that the amygdala modulates the hypothalamus with regards to regulation of food intake. More succinctly, the ventromedial hypothalamus, with its numerous glucose receptors, may monitor “hunger,” whereas the amygdala, with its major afferent input from the olfactory bulb, may direct goal-oriented “feeding behavior.”

**REFERENCES**


AMYGDALOID LESIONS AND BODY WEIGHT


