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Topographic Organization of Projections from the Amygdala to the Hypothalamus of the Rat

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SUMMARY

Afferent fibers from the amygdala to subdivisions of lateral, ventromedial and dorsomedial hypothalamic nuclei were investigated in rat by retrograde transport of horseradish peroxidase. Small (intranuclear size) peroxidase deposits were placed in hypothalamic nuclei by iontophoresis of a tracer solution containing poly-L-α-ornithine which greatly limited diffusion. The medial, central and amygdalo-hippocampal nuclei of the amygdala were found to be the major donors of amygdaloid afferent fibers to the hypothalamus, but there was also substantial labeling of somata in cortical, basomedial, basolateral and lateral amygdaloid nuclei and the intra-amygdaloid bed nucleus of the stria terminalis. No fibers projected from the posterior cortical nucleus of the amygdala to the hypothalamus. Most amygdaloid projections to the lateral hypothalamic area originated in the anterior half of the amygdala, while projections to the ventromedial hypothalamic nucleus arose along the entire length of the amygdala except the posterior cortical nucleus. The amygdalo-hippocampal area projects to the medial hypothalamus. Other amygdaloid nuclei project to both the medial and lateral hypothalamic nuclei. These topographic organizations of amygdaloid afferent fibers to various subdivisions of the hypothalamic nuclei are discussed and compared with other anatomical studies on these connections.

INTRODUCTION

The mammalian amygdala and hypothalamus are important interacting structures in the control of behavioral functions, principally feeding[4,14,36,38,39], offensive and defensive behavior[3,14,15,27,36,42] and behavior related to reproduction[4,14,16,36].

Extensive research primarily aimed at the study of hypothalamic control of feeding

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behavior indicates that the hypothalamus is important in regulating feeding. Various studies[11,35,40,41] associate these hypothalamic areas with autonomic mechanisms related to feeding and metabolic control, and since they contribute to limbic output, they must receive information from higher limbic centers such as the amygdala. The complex network of neural connections between the amygdala and the hypothalamus have previously been studied by both neuroanatomical[5,13,28,32] and electrophysiological[8,10] methods.

More modern anatomical autoradiographic, degeneration[6,9,12,16,20,21,33] and retrograde transport techniques[1,2,17,18,23,24] have demonstrated amygdaloid inputs to the hypothalamus. These modern anatomical studies, performed on various species, demonstrated that the ventromedial hypothalamic nucleus (vmh) is a major recipient of afferent fibers from the corticomedial group of the amygdala, whereas the dorsomedial hypothalamic nucleus (dmh) receives only minor inputs from these nuclei. The lateral hypothalamic area (lha) relations with the amygdala are reported to be predominantly with the deep amygdaloid nuclei such as the central (ac), basomedial (abm), basolateral (abl) and lateral (al) amygdaloid nuclei.

Several of the earlier studies[9,12,18,20,33] as well as our preliminary investigation[22] indicated that the projection fields of the various amygdaloid nuclei are not homogeneously distributed over their hypothalamic target nuclei. This suggests that different projection patterns to the various hypothalamic nuclei might correlate with functional differentiation. The question of details of subnuclear organization of projections from the amygdala to the lha, vmh and dmh remains. In the present study this was investigated by amygdaloid afferent fiber retrograde transport of horseradish peroxidase (HRP) after application to subdivisions of the various hypothalamic nuclei. The following three criteria were employed: (1) tracer deposits had to be limited to the subdivisions being studied (200–250 μm diameter); (2) the tracer uptake area had to be clearly defined by limiting diffusion from the deposit area; and (3) there must be no detectable tracer leakage in the injection track.

MATERIALS AND METHODS

Experiments were performed on 102 male albino Wistar rats weighing 240–360 g. Animals were anesthetized with sodium pentobarbital and placed in a stereotaxic apparatus. Stereotaxic procedures were performed using König and Klippel's system and coordinates[19]. Beveled glass micropipettes with inner tip diameters of 14–20 μm were filled with freshly prepared solution containing (w/v) 20% HRP (Sigma, type VI), 0.9% NaCl and 0.3% poly-L-α-ornithine (Sigma, type I-C). The electrophoretic drive was a 15-min train of 7-s positive DC pulses at 7-s intervals delivered by a constant current generator (Nihon Kohden). During the first 5 min the current was slowly increased from 0–3 μA, then maintained at that level for 10 min. After electrophoresis, the pipette was left in situ for 10 min before retraction.

After 24 h the animals were again anesthetized, perfused transcardially with 100 ml saline containing 10 IU/ml heparin, followed by 150 ml of 0.5% paraformaldehyde, 1.5% glutaraldehyde and 4% sucrose in 0.05 M phosphate buffer at pH 7.4. After overnight storage at 4 °C in buffered 30% sucrose, brain sections of 40 μm thickness were cut on a cryostat microtome. The sections were stored in buffered 30% sucrose and 30% ethyleneglycol at − 20 °C until further processing. Every second section was reacted for HRP according to Mesulam's tetramethyl benzidine method and counterstained with neutral red-saffranine O[26]. Several of the remaining sections were stained following the benzidine dihydrochloride procedure of De Olmos and Heimer[7].

RESULTS

The first 30 experiments were limited to developing a reproducible iontophoretic injection procedure to provide a dense, 200–250 μm deposit of HRP with strictly limited diffusion area (Fig. 1). Of the 56 successful cases that met the established criteria, 35

Fig. 1. Photomicrograph of representative horseradish peroxidase (HRP) deposit in the ventromedial hypothalamic nucleus (vmh) stained by tetramethyl benzidine to produce a deep, dark blue stain of the deposit. Iontophoretic delivery results in very limited diffusion. Irregular debris around the injection spot can be interpreted as transported tracer rather than diffusion. Scale bar: 250 μm.

were in the lha, 11 in the vmh and 10 in the dmh. The remaining 16 cases did not meet the criteria and were discarded.

HRP injections into the lateral hypothalamic area (lha) (Fig. 2)

In general, lha injections produced retrograde labeling limited almost exclusively to the anterior half of the amygdala, and much less labeling than vmh injections. The regions labeled by lha injections were the cortical, medial, central, basolateral, basomedial and lateral nuclei of the amygdala and the intra-amygdaloid bed nucleus of the stria terminalis. Among these nuclei the medial and central nuclei are the major sources of amygdaloid projections to the lha.

Injections into the lha and vmh differentiated the medial column of the amygdala, which corresponds to the medial nucleus described by König and Klippel[19], into three subnuclei[21]: the anterior medial amygdaloid nucleus (ama); the intermediate medial amygdaloid nucleus (ami) and posterior or amygdalo-hippocampal area (aha). The ama and ami were separated by a plane immediately anterior to A 5.15. More labeled cells were found in the ama than in the ami. The aha was completely devoid of labeling.

Labeling in the central amygdaloid nucleus (ac) was always in the anterior half, mostly in the center or the medial shell.

Cortical nuclei (aco) projections to the lha were almost exclusively from the anterior (coa) and periamygdaloid cortical nuclei (pac). Labeling in the posterior cortical nucleus (cop) was rarely observed.

The basomedial (abm), basolateral (abl), and lateral (al) nuclei are minor contributors to the lha. Labeling was never strong in the intra-amygdaloid bed nucleus of the stria terminalis (abst). Surrounding the root of the stria terminalis, a few scattered cells were found to be labeled.

The extent of retrograde amygdaloid labeling was highly correlated to the longitudinal and transverse site of injection in the lha. Injections into the anterior-posterior lha were arbitrarily divided into four groups: a far-anterior group between coordinates A 6.36–A 5.86, an anterior group between A 5.86–A 5.08, an intermediate group between A 5.08–A 4.46, and a posterior group between A 4.46–A 3.99. The coordinates refer to the interaural line described by König and Klippel[19]. The locations of one far-anterior (C65), two anterior (C66 and C58), two intermediate (C56 and C57) and one posterior (C53) lha injections and the results are exemplified in Fig. 2.

Injections into the far-anterior lha produced the least amygdaloid labeling. It is worth mentioning, however, that considerable labeling in the ami occurred after dorsal injection in this site (C65). After far-anterior injections, sparse labeling occurred in the cortical and anterior medial amygdaloid nuclei. Central amygdaloid labeling was very limited, especially after injection into the medial part of the far-anterior lha.

Retrograde transport to the amygdala from the anterior lha was more prominent (C66 and C58). The dorsal anterior deposit in C66 resulted in only sparse labeling in the cortical and medial nuclei and in occasional HRP-positive cells in the central nucleus and intra-amygdaloid bed nucleus of the stria terminalis. A more ventral

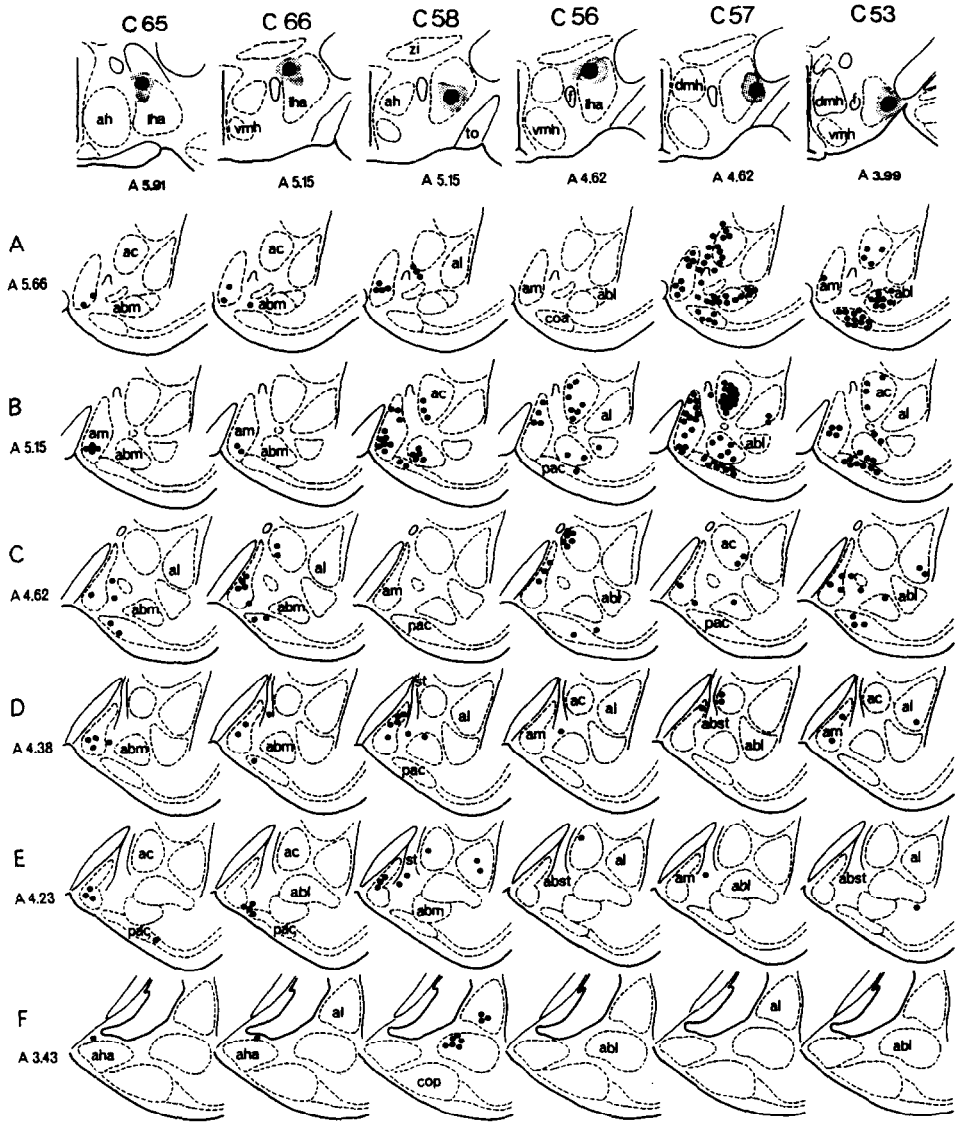

Fig. 2. Six HRP injections into the lateral hypothalamic area (lha) and labeled sites. C65: injected into the far-anterior lha at 5.91 mm anterior to the interaural line[19]; C66 and C58: injected into the anterior lha at 5.15 mm; C56 and C57: injected into the intermediate lha at 4.62 mm; and C53: injected into the posterior lha at 3.99 mm. Retrograde amygdaloid labeling after each injection indicated by black dots in sections below each injection. Transverse sections (A–F) at anterior-posterior levels indicated by numbers at left[19]. Comparison of the labeling in each horizontal row illustrates differences due to different injection sites.

injection at the same anterior level (C58) labeled a greater variety of locations. In C58 several cells were labeled in the medial nucleus at various levels, and a few in the central, basomedial, basolateral and lateral nuclei. Some HRP-positive cells were also found at the base of the stria terminalis.

A similar situation, but with different numbers of labeled cells, can be observed after injections at intermediate levels (C56 and C57). The ventrolateral injection shows that this part of the lha receives far more amygdala projections than the dorsal part. Most cells were labeled in the anterior part of the medial nucleus, the central nucleus, anterior and periamygdaloid cortical nuclei and the basomedial nucleus following injection into the intermediate ventrolateral lha. Few projections to this part of the lha seem to originate in the basolateral and lateral nuclei and the bed nucleus of the stria terminalis.

A similar pattern was evident in the posterior aspects of the lha, but the numbers of the labeled cells were smaller. Hence, caudad to the lha level A 4.46, there was much less labeling in the amygdala. As before, the ventrolateral injections at these levels (C53) labeled more neurons in the anterior and periamygdaloid cortical nuclei, and in the central and basomedial nuclei. Sparse retrograde marking of cells was observed in the lateral, medial and basolateral nuclei.

In conclusion, it appears that amygdaloid afferent fibers to the lha are organized in anterior-posterior and transverse planes. Of the lha injections, injections into the anterior and intermediate regions produced the greatest variety and extent of amygdaloid labeling. Furthermore most amygdaloid nuclei were more strongly labeled by ventrolateral than by dorsal lha injections. This was most obvious for the central, cortical, basolateral and basomedial nuclei and for the anterior part of the medial nucleus. One might also conclude that central, basolateral, basomedial and lateral nuclei maintain stronger projections to more caudad levels in the lha, while the opposite is true for the intermediate medial nucleus. The bed nucleus of the stria terminalis seems to connect with lha levels more anterior than those that receive afferent fibers from the central, basolateral, basomedial and lateral nuclei.

HRP injections in the ventromedial hypothalamic nucleus (vmh) (Fig. 3)

Retrograde labeling from the three hypothalamic nuclei that were studied revealed by far the most numerous projections from the amygdala to the vmh. Numerous vmh afferent fibers from the amygdala can be demonstrated in the anterior (ama) and intermediate (ami) medial amygdaloid nuclei and in the amygdalo-hippocampal area (aha).

Somewhat less labeling was found in the central and basomedial, and in the bed nucleus of the stria terminalis (abst), but this still equaled or exceeded that by lha injections. Least prominent was retrograde transport to the basolateral and lateral nuclei. Most striking were the absence of labeling in the posterior cortical nucleus and the very strong labeling in the amygdalo-hippocampal area, a continuation of the granular layer of the ventral subiculum (Fig. 4). In contrast to the lha afferent fibers, amygdaloid afferent fibers to the vmh were found over the entire anterior-posterior

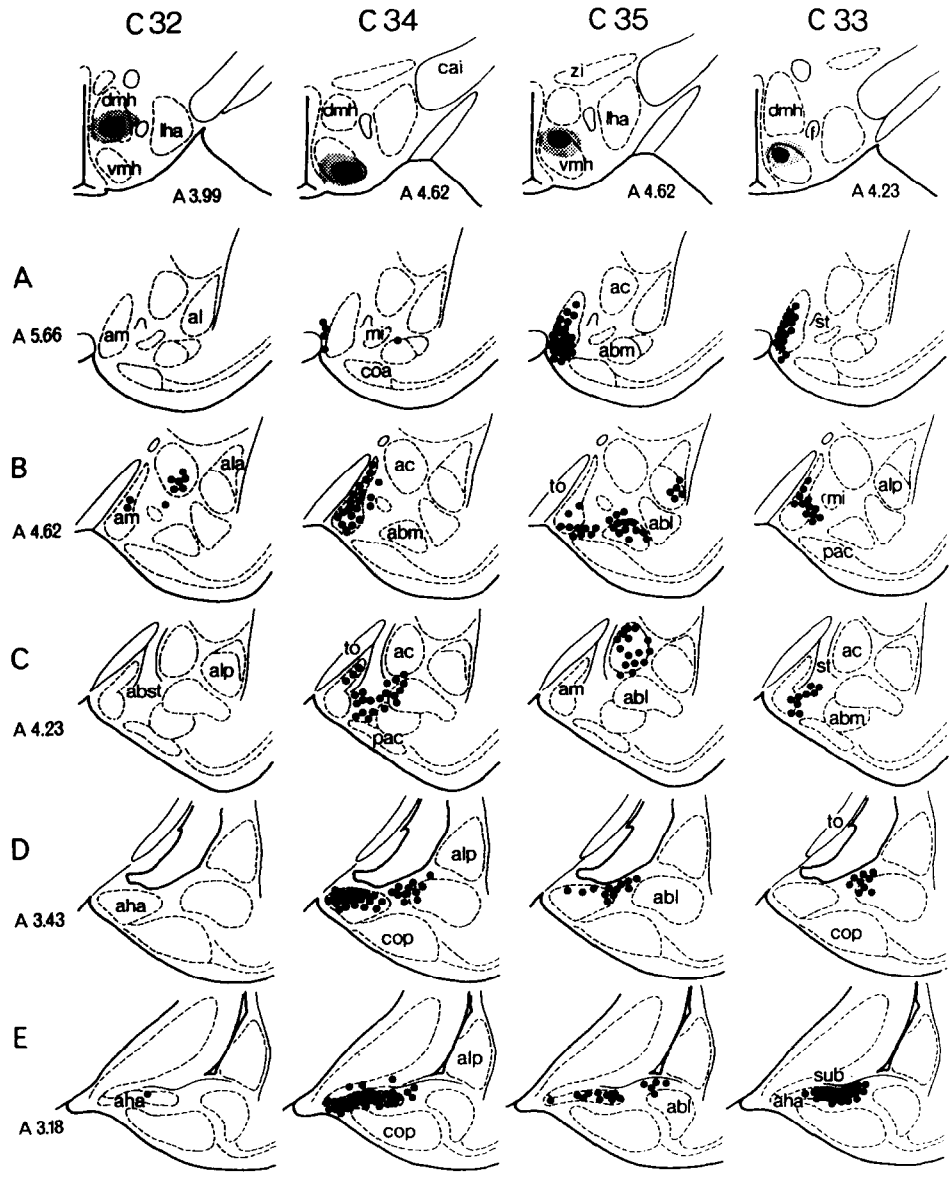

Fig. 3. HRP injections into the dorsomedial nucleus (dmh) and ventromedial nucleus (vmh) at various levels. C32: injected into the dmh at 3.99 mm anterior to the interaural line[19]; C34 and C35: injected into the anterior vmh at 4.62 mm; and C33: injected into the posterior vmh at 4.23 mm. See legend for Fig. 2.

Fig. 4. Photomicrograph of densely labeled somata in the amygdalo-hippocampal area (aha) after HRP injection into the ventromedial hypothalamic nucleus. Note: posterior cortical nucleus at bottom is completely devoid of labeling.

extent of the amygdala. Labeling in the central nucleus, when present, was located in the more posterior parts of this nucleus. This was also true of labeling in the basolateral nucleus.

Another striking observation was the considerable labeling in the poorly-defined central area between the medial and basal nuclei, generally referred to as the bed nucleus of the stria terminalis (Fig. 5). We believe this area may extend more rostral and caudad than is generally indicated in the literature[21,31] since we found labeled cells more rostrally and caudally at the base of the stria terminalis.

The amount of amygdaloid labeling depended on the vmh injection site. This suggests a topographically organized amygdalofugal system. To demonstrate this, three injections and the results are depicted in Fig. 3 (C33, C34 and C35). The injections represented in C34 and C35 were both made into anterior parts of the vmh; more ventrally in C34 and more dorsally in C35. The anterior ventral vmh injection strongly labeled the intermediate medial nucleus, amygdalo-hippocampal area and the bed nucleus of the stria terminalis. Several cells were labeled in the medial aspects of the caudad basolateral nucleus, but none were found in the cortical, central, lateral and basomedial nuclei. Anterior dorsal vmh injections (C35), on the other hand, resulted in considerable labeling in the anterior medial nucleus and some in the amygdalo-

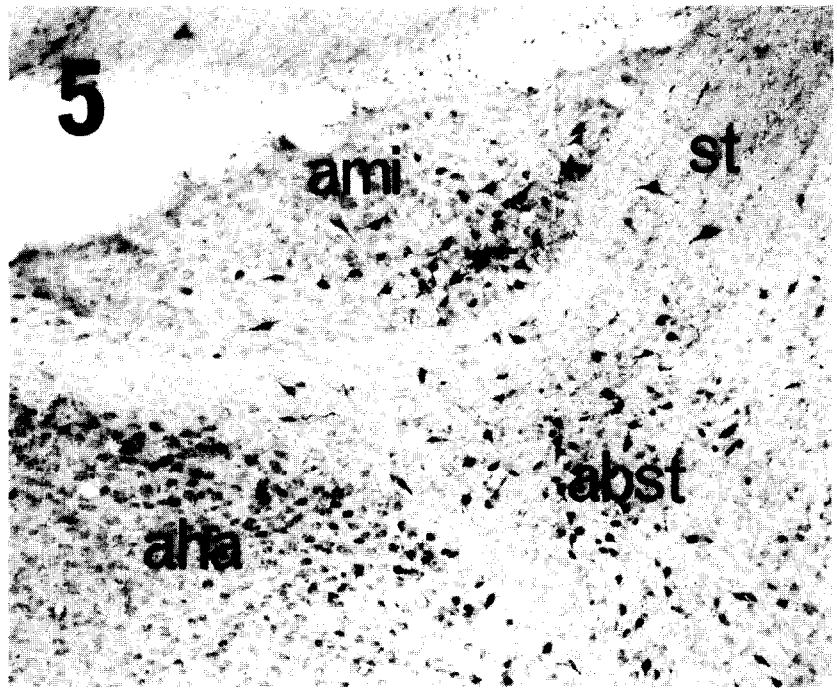

Fig. 5. Photomicrograph of amount and position of labeling in intra-amygdaloid bed nucleus of stria terminalis (abst) after HRP injection into ventromedial hypothalamic nucleus. Intermediate part of the medial amygdaloid nucleus (ami) is dorsal and amygdalo-hippocampal area (aha) is ventromedial to the abst.

hippocampal area, and the central, basomedial, and lateral nuclei. Labeling in the lateral nucleus was always confined to what is called the posterior lateral nucleus (alp) by König and Klippel[19]. More posterior vmh injections (C33) resulted in strong labeling in the medial nucleus, especially in the anterior medial nucleus and the amygdalo-hippocampal area; some in the basolateral and central nuclei (central nucleus observed in sections not shown here); but none in the cortical, basomedial and lateral nuclei. In conclusion, amygdaloid afferent fibers to the vmh also seem to be organized in anterior-posterior and transverse planes. It seems worth mentioning that injections into the ventral parts of the vmh also labeled some cells in the contralateral amygdalo-hippocampal area. This indicates a crossed contribution of the amygdalo-hippocampal area to the vmh.

HRP injection into the dorsomedial hypothalamic nucleus (dmh) (Fig. 3).

Of the three hypothalamic nuclei studied, the dmh (C32) received the fewest amygdaloid projections and only from the central, basomedial, lateral and medial nuclei. The central nucleus appeared to be the major source. The anterior and intermediate medial nuclei, the basomedial and lateral nuclei (basomedial and lateral nuclei observed

in sections not shown here) and the amygdalo-hippocampal area were only sparsely labeled. It was also observed that the more anteriorly and ventrally placed dmh injections resulted in the most labeling in the amygdala.

DISCUSSION

This discussion treats the data under separate headings according to the hypothalamic termination of projections from various amygdaloid nuclei.

Projection fields of the cortico-medial nuclear group of the amygdala

Projections of the anterior medial amygdaloid nucleus (ama) (Fig. 6A)

Based on hypothalamic afferent fibers identified by retrograde transport of HRP, we divided the neural population of the medial nucleus into two subnuclei: anterior (ama) and intermediate (ami) amygdaloid nuclei. These correspond to the anterior and posterodorsal parts of the medial nucleus described by Krettek and Price[21] except that our anterior part is smaller and our intermediate part is larger than theirs. Other than this, the ama projection field to the vmh that we saw agrees with that described by Krettek and Price[20]; i.e. there is a strong amygdaloid projection to the entire core of the ventromedial nucleus. Although Krettek and Price[20] do not describe a dmh projection in detail, their illustrations suggest minor projections to ventral aspects of the dmh, which we also found. The ama projections seen in this study confirm previous retrograde transport studies in which extensive labeling in the ama was observed after vmh injections of HRP, but our method permitted finer resolution of the connected areas. The ama projection to the vmh must, without any doubt, be regarded as quantitatively the most significant amygdala projection to the hypothalamus.

The ama projections that we found to the lha also agree with the findings of Krettek and Price[20]; i.e. this field projects mainly to the tuberal part of the lha[37], which is equivalent to the lha at the level of the vmh. In the present study this tuberal projection appears to be limited to the more ventral zone of the lha. Projection of the ama to the lha have also been described in other reports[1,18,24], but have been omitted from some[2,23].

Projection of the intermediate medial amygdaloid nucleus (ami) (Fig. 6B)

In our study a moderate projection of the ami to the ventral half of the vmh was found. Although Krettek and Price[21] discriminate between an anterior and a posterior medial nucleus, they do not explicitly describe a projection pattern from the latter. Kevetter and Winans[16], working with hamsters, presented data that indicated projections from more posterior parts of the medial nucleus to more ventral aspects of the vmh. A minor ami projection to the caudad half of the dmh has not received much attention in anterograde transport studies, but retrograde transport to the ami after dmh injection of HRP has been observed by other authors[2,23]. The ami projection to the more

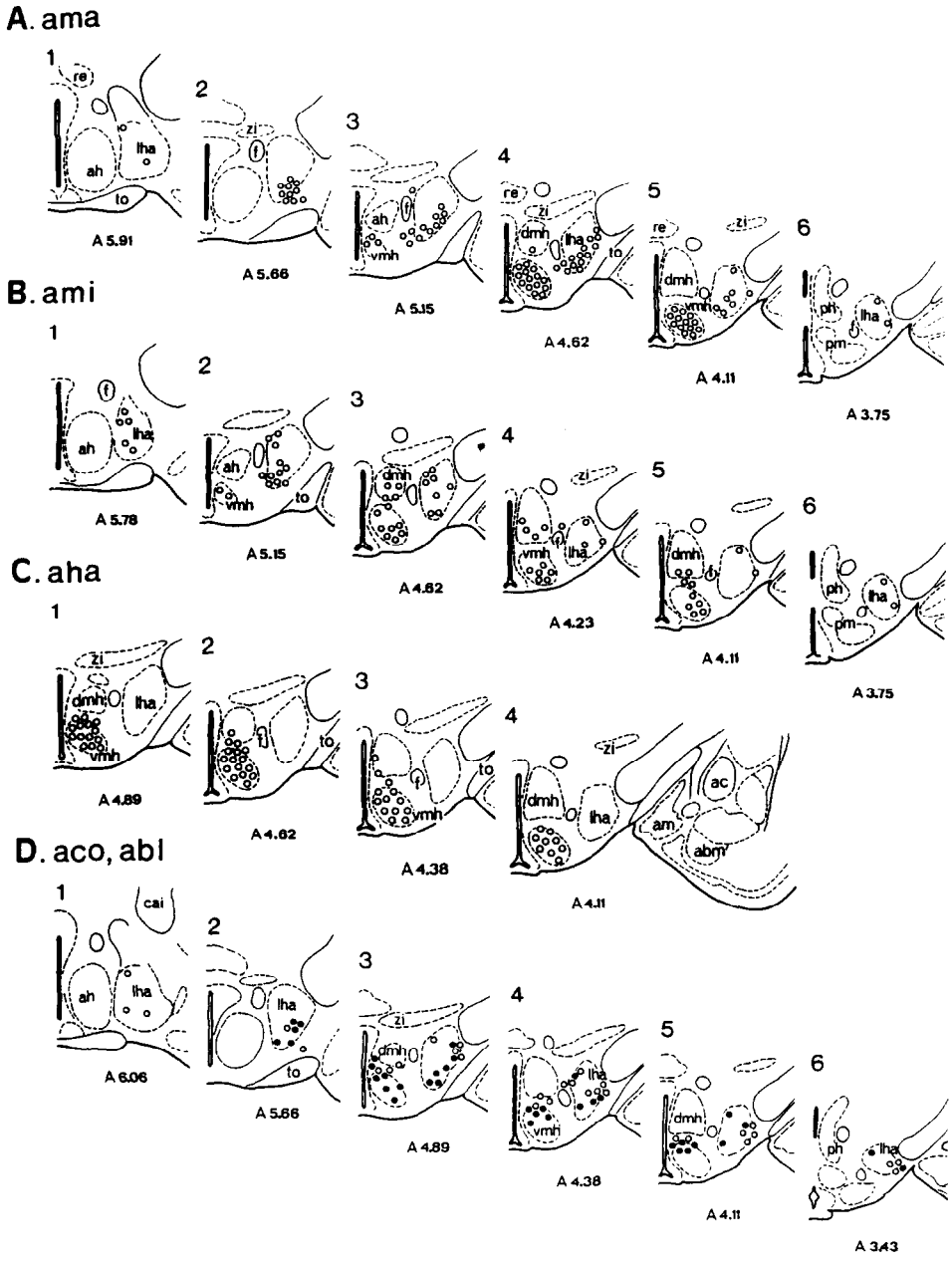

Fig. 6. Schematic representation of projections (open and filled circles) from somata of anterior medial amygdaloid nucleus (ama), intermediate medial amygdaloid nucleus (ami), amygdalo-hippocampal area (aha), and the cortical (aco) and basolateral (abl) amygdaloid nuclei. In D, open and filled circles show the terminations of the cortical and basolateral amygdaloid nuclei, respectively. The distribution of each termination in the hypothalamus is plotted on sections from rostral (1) to caudal (6). The positions of the symbols delineate loci of projections, and density of symbols reflects the relative number of termination.

medial column of the lha has not been described in detail before. Only Barone *et al.*[1] mention the projections from the medial nucleus to the lha, but without reference to the position of the hypothalamic injections or the precise position of the labeled somata within the nucleus.

Projection of the amygdalo-hippocampal area (aha) (Fig. 6C)

The aha has prominent projections to the vmh, and limited projections to the ventral ridge of the dmh, but aha labeling after injection of the lha is totally absent. Since our method does not permit discrimination between projections to core or shell, comparison between our results and those of anterograde transport or degeneration studies remains difficult. It is evident, however, that the aha projections to the medial hypothalamus that we found show a pattern similar to that reported by Krettek and Price[20] but excluding the differentiation between core and shell projections. As in previous studies of HRP transport, the aha can hardly be disputed to be the source of hypothalamic afferent fibers. All reports mention strong labeling in the aha after vmh injections[2,17,23], and some aha projections to the dmh are reported[2], but aha inputs to the lha have not previously been reported.

Projection of the cortical amygdaloid nuclei (aco) (Fig. 6D)

As described, the amygdaloid cortical sources of afferent fibers to the hypothalamus were found exclusively in the anterior and periamygdaloid cortical nuclei. It must be concluded that the posterior cortical nucleus (cop) is not involved in amygdalofugal systems terminating in the hypothalamus. Krettek and Price[20] do not separately describe projections from the various cortical nuclei and only mention minor vmh projections of the cop in combination with aha projections. The cop projection that they found, however, might be explained by a spread of tracer in the aha from the cortical injection site. A more discriminating investigation of cortical amygdaloid efferent fibers, although performed in hamsters, agrees more closely with ours. In that study, minor vmh projections from anterior cortical cell groups and absence of efferent fibers to the hypothalamus from the posterior cortical nucleus of the amygdala were found[16]. In previous HRP studies, sparse cortical projections to the lha were reported with no mention of afferent fibers to the vmh from the cortical nuclei[1,24].

Projection fields of the deep amygdaloid nuclei

Projection of the basolateral amygdaloid nucleus (abl) (Fig. 6D)

The abl appears to send fibers primarily to the lha, but the vmh also receives some input from the abl. These abl projections were not reported in detail by Krettek and Price[20,21]. Projections from the abl to the tuberal part of the lha in the monkey, however, have been reported[33]. These were located in the ventromedial part of the lha at the level of the vmh. This also appears to be the major target in the rat. Basolateral projections to the lha were also reported by Kita and Oomura[18] and McBride and Sutin[24].

Projections to the vmh from the abl were previously observed only once, by Kita and Oomura[17].

Projection of the central amygdaloid nucleus (ac) (Fig. 7A)

Overall, ac projections to the lha are considerable. Most labeling in the ac was observed in the center or the medial shell of this nucleus (Fig. 2). This labeling that we observed approximately corresponds to the medial and intermediate subdivisions of McDonald[25].

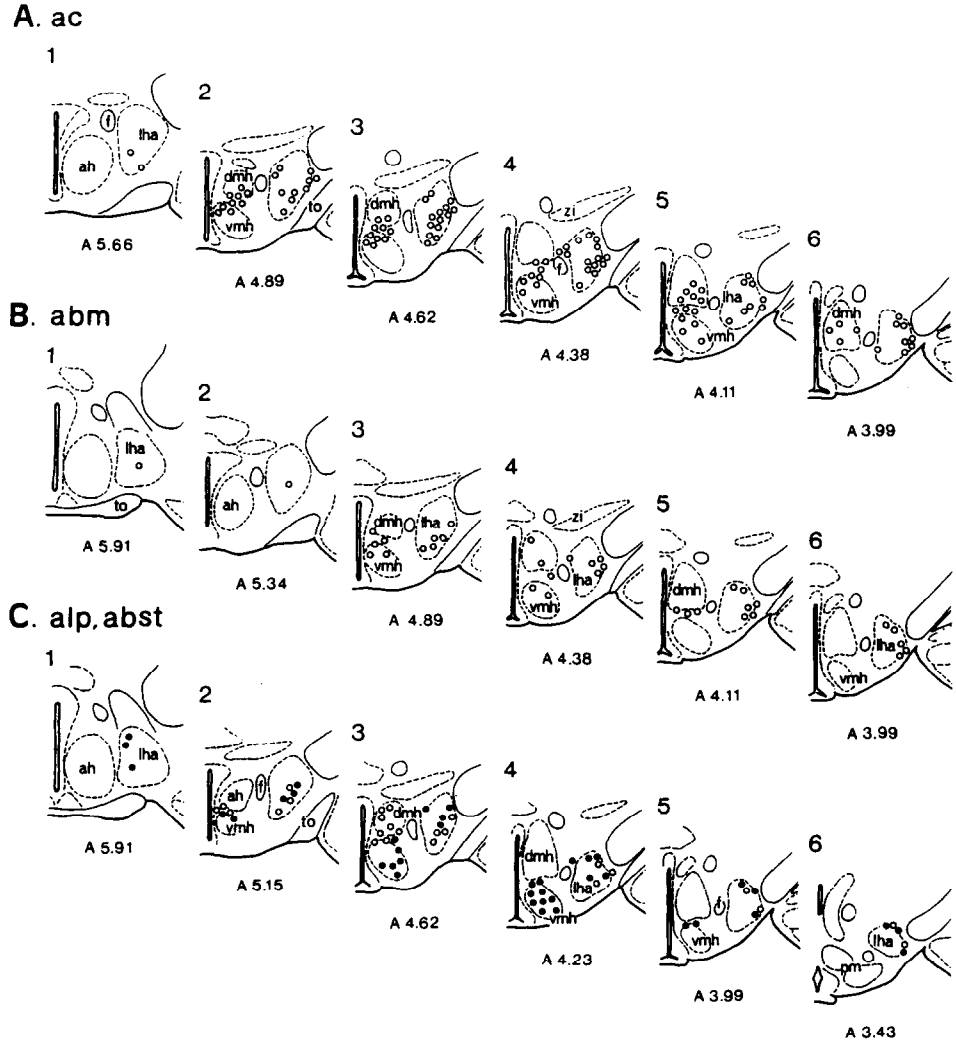

Fig. 7. Projection field of central amygdaloid nucleus (ac), basomedial amygdaloid nucleus (abm), and posterior lateral amygdaloid nucleus (alp; open circles) and intra-amygdaloid bed nucleus of stria terminalis (abst; filled circles). See legend for Fig. 6.

The ac is the most significant amygdaloid area of input to the dmh, but it is a minor source of input to the vmh. It should be emphasized that the major ac target in the lha is probably the lateral column of the lha. The lha-directed projection from the ac was also reported by Krettek and Price[20], but they did not report projections from the ac to the medial hypothalamic nuclei. A more detailed study of ac projections in the monkey, by Price and Amaral[33], presents additional information about projections to the dmh and the dorsal vmh. This agrees with the projection pattern that we described.

The ac projection to the lha is supported by most, if not all, retrograde transport studies[1,2,18,23]. Retrograde transport to the ac after medial hypothalamic injection has been previously reported by Berk and Finkelstein[2], Kita and Oomura[17] and Luiten and Room[23], but none of these studies revealed the precise target areas within the medial hypothalamic nuclei.

Projection of the basomedial amygdaloid nucleus (abm) (Fig. 7B)

We observed abm projections to all three hypothalamic nuclei. From our results, this appears to be a minor input source to the hypothalamus, but it is described as major by Krettek and Price[20,21] who reported a rather strong abm projection to the core of the vmh. Their anterograde tracer injections into the abm, however, also included the lateral and basolateral nuclei. These two nuclei, in our opinion, also maintain vmh projections; an opinion not shared by Krettek and Price[20,21]. Sparse abm labeling after HRP injection in the vmh, however, was also observed by Berk and Finkelstein[2], and McBride and Sutin[24], while other authors do not describe these projections at all[17,23]. The abm projection to the lha is more in agreement with autoradiographic studies[20,21]. We also found projections from abm cells to the tuberal part of the lha[37], which appears to be the main target area in the hypothalamus for projections from the abm.

Projection of the lateral amygdaloid nucleus (al) (Fig. 7C)

The al can be divided into anterior and posterior portions[19]. These overlap considerably on the ventral side of the posterior part (alp) where it fuses with the basolateral nucleus. Labeling in the al was observed only in the alp in our experiments. This agrees with the findings of Barone et al.[1] and Kita and Oomura[18]. The al projection to the hypothalamus, as demonstrated by our experiments, is probably the least important of all amygdala projections to the hypothalamus since it is limited to a few target cells in anterior parts of the dmh, and to some scattered cell groups in the posterior half of the lha and the anterodorsal part of the vmh.

Krettek and Price[20,21] suggested that the basolateral nucleus (abl) and al influence the hypothalamus indirectly via the basomedial amygdala and the ventral subiculum. This is a striking discrepancy between our findings and theirs. Electrophysiological experiments[29,30,34], in which responses of vmh and lha neurons to abl stimulation had short latency, can be explained by direct hypothalamic projections from these amygdaloid nuclei.

Projection of the intra-amygdaloid bed nucleus of the stria terminalis (abst) (Fig. 7C)

The abst can be distinguished from all other amygdaloid cell populations in that it cannot be defined as a clear concentration of somata. The abst can best be regarded as cells that are embedded in the base of the stria terminalis, situated between the central, medial, basomedial and basolateral nuclei. We believe that cells that can be described as abst cells appear in a further anterior and posterior column than indicated by Ottersen[31] or Krettek and Price[21], and we found labeled somata in this cell-sparse area.

The projections from the abst that we found in our study do not seem to be unimportant. There are substantial projections to different parts of the vmh, and it is one of the few amygdaloid cell groups that sends fibers to the dorsal aspects of the lha. Our data, however, do not find much support in the literature. Afferent fibers of the abst have been studied[31], but data on its efferent systems are scarce. A description of the abst is given by Krettek and Price[21], but these authors make no mention of amygdalofugal connections originating in the abst.

When anatomical differentiation of neuronal connections can be demonstrated, it might be tempting to discuss its correlation with functional differentiation. However, the present investigation suggests that functional specificity or functional differentiation is, to a certain degree, based on a subnuclear organization of structural connections. Hence, we believe that detailed knowledge of hypothalamic input sources is a prerequisite for further investigation of hypothalamic contributions to behavior.

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ABBREVIATIONS

abl	basolateral amygdaloid nucleus
abm	basomedial amygdaloid nucleus
abst	intra-amygdaloid bed nucleus of the stria terminalis
ac	central amygdaloid nucleus
aco	cortical amygdaloid nucleus
ah	anterior hypothalamic nucleus
aha	amygdalo-hippocampal area
al	lateral amygdaloid nucleus
ala	anterior lateral amygdaloid nucleus
alp	posterior lateral amygdaloid nucleus

am	medial amygdaloid nucleus
ama	anterior medial amygdaloid nucleus
ami	intermediate medial amygdaloid nucleus
cai	capsula interna
coa	anterior cortical nucleus of the amygdala
cop	posterior cortical nucleus of the amygdala
cp	nucleus caudatus putamen
dmh	dorsomedial hypothalamic nucleus
f	fornix
lha	lateral hypothalamic area
mi	intercalated nucleus of the amygdala
pac	periamygdaloid cortex
ph	posterior hypothalamic nucleus
pm	premammillary nucleus
re	nucleus reuniens of the thalamus
st	stria terminalis
sub	subiculum
to	optic tract
vmh	ventromedial hypothalamic nucleus
zi	zona incerta

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