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Comparative Study of Roles of the Lobus Parolfactorius and Intermediate Medial Hyperstriatum Ventrale in Memory Formation in the Chick Brain

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SERRANO, P. A., S. J. RAMUS, E. L. BENNETT AND M. R. ROSENZWEIG. *Comparative study of roles of the lobus parolfactorius and intermediate medial hyperstriatum ventrale in memory formation in the chick brain.* PHARMACOL BIOCHEM BEHAV **41**(4) 761–766, 1992. – Two discrete areas of the chick brain, the intermediate medial hyperstriatum ventrale (IMHV) and lobus parolfactorius (LPO), were found to have different functions during the formation of memory for a 1-trial peck-avoidance paradigm. Glutamate, ouabain, and emetine, known to disrupt short-, intermediate-, and long-term memory when injected into the IMHV, were injected into the cerebellum and LPO. All amnestic agents investigated produced amnesia when injected into the IMHV; only one of these agents produced amnesia when injected into the LPO, and none of the agents produced amnesia when injected into the cerebellum. The chick brain was also found to exhibit hemispheric asymmetries: The left IMHV and LPO were more sensitive to the amnestic agents than their corresponding right structure. From these data, hypotheses for the roles of these structures during memory are proposed.

Chick IMHV LPO Memory Hemispheric asymmetry Amnestic agents Intermediate-term memory Long-term memory Short-term memory Cerebellum

THE 2-day-old chick provides a useful model to study memory formation because of its predisposition to peck at small objects and remember the consequences (17). Chicks will learn to avoid a small bead dipped in a noxious substance, methylanthranilate (MEA), after pecking it during one 10-s trial. This natural learning activity permits the investigation of biochemical correlates of learning and memory using pharmacological interventions.

From pharmacological interventions, a three-stage model of memory formation has been developed (9). This model identifies short, intermediate, and long-term, serially dependent stages and suggests that each of these stages is dependent upon a specific neurochemical mechanism. Disruption of these neural processes results in amnesia for a learning experience. This evidence has prompted other researchers to investigate neural mechanisms of memory using the 2-day-old chick as a behavioral model. [For reviews by R. J. Andrew, M. Gibbs, G. Horn, S. P. R. Rose, M. R. Rosenzweig, M. G. Stewart, and their collaborators, see (2).]

Studies using biochemical interventions have found discrete areas of the chick brain to be important for memory formation. Kossut and Rose (15) demonstrated that the chick brain utilizes the intermediate medial hyperstriatum ventrale (IMHV) and the lobus parolfactorius (LPO) during learning of a passive avoidance task as shown by increased 2-deoxyglucose (2-DG) uptake. These results are taken as indicators of specific brain regions that are metabolically active during learning. Similarly, the IMHV has also been found to be metabolically active during imprinting as shown by increased incorporation of [^{14}C]leucine into protein (12). Morphological changes in these areas have also been correlated to behavior. These changes again demonstrate correlates of learning (11,14).

Several studies investigating hemispheric asymmetries in the chick have shown the left hemisphere to be more pertinent to memory than the right hemisphere (1,3,8,22) and more specifically the left IMHV (13,20). In contrast, the lateral neostriatum (LNS) has been shown to have a right hemispheric dominance in a one-trial peck avoidance paradigm (19). Hemispheric asymmetries have also been found in the LPO. Using [^{14}C]2-DG higher uptake of 2-DG is reported in the left IMHV and LPO than in the right IMHV or LPO after passive avoidance learning (24).

To further characterize the role of these brain areas during

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learning, pharmacological interventions are employed in the research described in this article. The three brain areas investigated are the IMHV, LPO, and cerebellum. From the results, hypotheses are proposed for the roles of these areas during the three stages of memory formation.

METHOD

Animals and Testing

Male chicks of two different strains were used in these experiments. Experiments 1 and 2 used Dekalb-Warren sex-linked hybrids from the H&N Hatchery (Petaluma, CA). Experiments 3 and 4 used Dekalb XL-white leghorn cockerels from the Dekalb Hatchery (Turlock, CA). The 1-day-old cockerels arrive at the laboratory between 8 and 9 a.m. They are left in their shipping boxes overnight, provided with water, and placed in a dark behavioral testing room at 35°C and 45% relative humidity. The following morning at 8 a.m., lights are turned on and chicks are housed individually in white cylindrical cartons (quart size, 8.3 × 17.8 cm). The cartons, in groups of 10, are covered with a Plexiglas lid to prevent chicks from escaping. The lid has a 5.1-cm air hole centered over each carton. Chicks are allowed to acclimate to their new surroundings for 2 h before training occurs. Initially after chicks are housed, they issue many distress peeps and attempt frequently to jump out of the containers. This behavior decreases once the chick has acclimated.

Chicks are trained on a one-trial passive avoidance task. Each chick is presented for 10 s with a stainless steel 3-mm bead at the end of a stiff wire. The bead is dipped in an aversive substance, MEA. When the chick has pecked at the coated bead, it demonstrates a typical disgust response. This response is characterized by head shaking, beak wiping, and distress peeps. This paradigm is similar to the one developed by Cherkin and Lee-Teng (5). In these experiments, training takes place 5 min after the chick has been injected with an amnestic agent or saline, and the chick is tested 24 h later, except for Experiment 3. All injections are delivered using a 500-μl Hamilton repeating syringe. Each chick receives a 10-μl injection into a specific brain area in each hemisphere. Chicks that receive cerebellar injections receive one 10-μl injection into the medial cerebellum 0.5 mm above the cerebellar commissure. For injection, the chick is placed in a Plexiglas headholder designed by Davis et al. (7). The headholder has a top plate with holes that correspond to different areas of the chick brain. To control the depth of injection, a plastic sleeve is fitted over the upper part of the shaft of a 30-ga needle. The skull of the 2-day-old chick is unossified, and intracranial injections delivered into the unanesthetized chick evoke no signs of discomfort. The standard injection protocol allows 15 chicks to be injected in 5 min. During training, we record the latency to peck, number of pecks, and whether a disgust response is shown. Chicks that do not peck the MEA-coated bead during the 10-s training trial or do not show a disgust response are eliminated. Typically, less than 10% of the subjects are eliminated. Groups of chicks were injected with an amnestic agent or saline into the region of the IMHV, LPO, or cerebellum. Typically, each experiment involved two amnestic agents and saline injected into two areas of the chick brain. This experimental design allows for batch differences that may occur across different areas of the brain and different sensitivities to amnestic agents across batches of chicks. Since every experimental manipulation was not included in every experiment, each experimental group has its own control group. All agents are coded and randomized, and the codes are revealed only at the conclusion of the experiment. A nonparametric χ^2 test is used to test for significance between experimental and control groups.

EXPERIMENT 1: DOSE-RESPONSE FUNCTION FOR AMNESTIC AGENTS INJECTED INTO THE CEREBELLUM

The chick cerebellum, like the mammalian cerebellum, is an area with motor and sensory connections (4). The learning task employed in this experiment is believed to utilize the mechanisms that are characteristic functions of the cerebellum. For this reason, it seemed possible that the memory trace would be susceptible to disruption by injection into the cerebellum of the same agents that cause amnesia in the IMHV. To determine the possible role of the cerebellum during memory formation, glutamate, ouabain, and emetine—agents that when injected into the IMHV disrupt short-term memory (STM), intermediate-term memory (ITM), and long-term memory (LTM), respectively—were injected 3 mm below the surface of the skull into the medial cerebellum. Other sites in the cerebellum were not investigated since the headholder used for injections was designed to investigate forebrain structures. Injections into the cerebellum, therefore, were limited to the medial cerebellum. From verification of injection sites on the skull surface and also within the brain, 85% of injections were within 1.0 mm of the targeted area. The depth of injections ranged between 3.0–3.5 mm. We attempted to determine a dose–response curve for each agent using four doses of glutamate (35–90 mM), four doses of ouabain (0.015–0.05 mM), and five doses of emetine (1.0–8.0 mM). The doses used include the doses that produced amnesia in other areas of the chick brain. The highest dose was slightly below the dose that would disrupt the chick's ability to peck the MEA-coated bead during training. The number of chicks per group ranged between 25–40.

TABLE 1

% RETENTION AT 24-h TEST AFTER AN INJECTION OF GLUTAMATE, OUABAIN, EMETINE, OR SALINE INTO THE CEREBELLUM

Drug	Dose (mM)	% Retention
Saline		81
Glutamate	30	73
	45	68
	60	65
	90	68
Saline		94
Ouabain	0.015	79
	0.025	77
	0.035	89
	0.05	92
Saline		81
Emetine	1.0	71
	2.0	94
	3.0	88
	4.5	82
	8.0	88

Injections (10 μl) were made into the medial cerebellum 5 min before training. Amnesia did not occur with any dose. n = 25–40 per group.

RESULTS

The results of Experiment 1, presented in Table 1, show that over a wide range of concentrations no significant amnesia occurred when glutamate, ouabain, or emetine was injected into the cerebellum. In contrast, bilateral injections of these drugs into the IMHV produced significant amnesia (50% or less retention). The effective doses to produce amnesia with injections of glutamate, ouabain, and emetine bilaterally into the IMHV are 50, 0.027, and 4.0 mM, respectively; neither higher nor lower doses were effective in the cerebellum.

EXPERIMENT 2: DOSE-RESPONSE FUNCTIONS FOR AMNESTIC AGENTS INJECTED INTO THE LPO OR IMHV

Chicks were given a bilateral injection of glutamate, ouabain, emetine, or saline into the LPO (2.4 mm anterior to bregma, 1.3 mm lateral, 5 mm deep). Verification of placement of injection revealed that 80% of injections were in the targeted area. Five doses of glutamate (35–70 mM) or saline were injected into 35–45 chicks per group. Five doses of ouabain (0.02–0.05 mM) or saline were injected into 45–50 chicks per group. Six doses of emetine (0.75–6.0 mM) or saline were injected into 37–80 chicks per group.

For comparison, in the same experiments we made bilateral IMHV injections of the following doses: 50 mM glutamate, 0.027 mM ouabain, 4.0 mM emetine, or saline. Injections were made into a region of the IMHV (1.2 mm anterior of bregma, 1.3 mm lateral, 3 mm deep). There were 30–40 chicks per group. These doses represent the most effective concentrations when injected into the IMHV as previously reported from this laboratory (20).

RESULTS

The results of Experiment 2 show that neither glutamate nor emetine, when injected into the LPO, produces significant amnesia over a range of concentrations (Table 2). This range of doses includes the effective doses for producing amnesia when injected into the IMHV. Ouabain when injected into the LPO produced significant amnesia ($p < 0.01$) at 0.027, 0.035, 0.04, and 0.05 mM (Table 2). These concentrations have also been shown to produce amnesia in the IMHV (20).

EXPERIMENT 3: TIME COURSES FOR THE DEVELOPMENT OF AMNESIA AFTER OUABAIN INJECTIONS INTO EITHER THE LPO OR IMHV

To further characterize the role of the LPO during memory formation, it is critical to determine if ouabain disrupts the same stage of memory formation when injected into the LPO as it does when injected into the IMHV. In this experiment, time courses for the development of amnesia using glutamate, an STM inhibitor, or ouabain, an ITM inhibitor, were determined for these amnestic agents when injected into the IMHV. These time courses provided standard curves for both short and intermediate-term stages of memory formation (9,20). They exemplify the difference between the decline of STM and ITM in the IMHV and can be used to identify the stage of memory formation inhibited when ouabain is injected into the LPO.

Chicks were injected with either saline, ouabain (0.027 mM), or glutamate (50 mM) in the IMHV or with saline or ouabain (0.027 mM) in the LPO. Groups of chicks were tested at various times after training ranging from 1–120 min. There were 30–40 chicks per group.

RESULTS

The time courses for the development of amnesia caused by agents injected into the IMHV were as expected (Fig. 1). The STM inhibitor, glutamate, produced significant amnesia within 5 min after training ($p < 0.01$) and at all subsequent test times. In contrast, ouabain-injected birds did not show significant amnesia at 5 min after training. While they show significant amnesia at 10 min ($p < 0.05$), the amnesia was not fully developed until 15 min, as indicated by more significant amnesia ($p < 0.01$). The differences between the glutamate and ouabain time courses are expected between 1–10 min after training. By 15 min, both time courses are expected to show no differences. A contingency table analysis was run for the 1-, 5-, and 10-min time points for the ouabain and glutamate time courses. The results showed statistical significance ($p < 0.01$). Further analysis of the contingency table for the remaining time points revealed no significant differences, as expected. By 15 min, both time courses showed significant amnesia ($p < 0.01$) as compared to saline-injected controls. The time course for appearance of amnesia when the ITM inhibitor, ouabain, was injected into the IMHV (Fig. 2) was not significantly different at any time point from the course

TABLE 2

% RETENTION FOR A 24-h TEST AFTER INJECTIONS OF EITHER GLUTAMATE, OUABAIN, EMETINE, OR SALINE INTO THE IMHV OR LPO

	% Retention			% Retention			% Retention	
Glutamate Dose (mM)	LPO	IMHV	Ouabain Dose (mM)	LPO	IMHV	Emetine Dose (mM)	LPO	IMHV
Saline	89	84	Saline	87	84	Saline	83	84
35	78		0.02	65		0.75	72	
40	80		0.027	50*	39*	1.5	81	
50	77	41*	0.035	51*		2.25	80	
60	78		0.04	38*		3.0	79	
70	83		0.05	40*		4.0		47*
						4.5	73	
						6.0	61†	

Injections (10 μl) were made bilaterally into the LPO or IMHV 5 min before training. n = 35–75 per group. *$p < 0.01$; †$p < 0.05$.

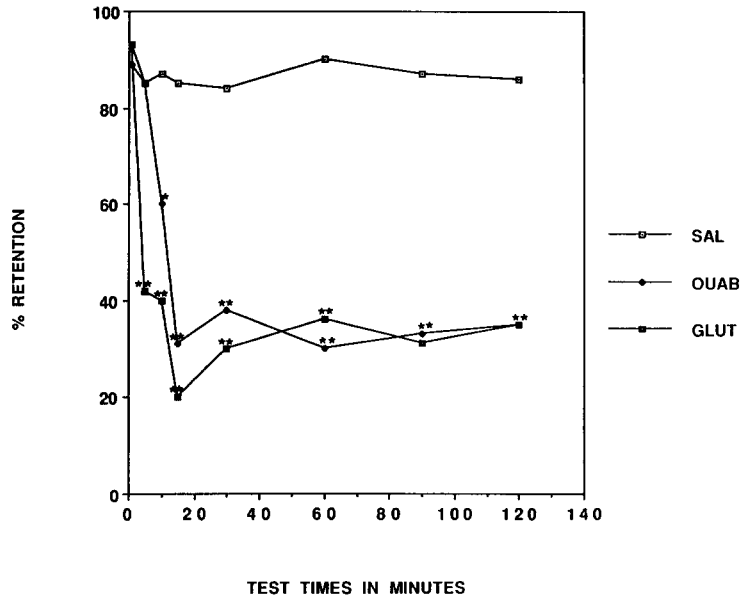

FIG. 1. Time course for appearance of amnesia after injections of glutamate 50 mM, ouabain 0.027 mM, or saline into the IMHV. Injections (10 μl/hemisphere) were made into the region of the IMHV 5 min before training. Groups of chicks were tested at 1, 5, 10, 15, 30, 60, 90, or 120 min after training. n = 30–40 per group. *$p < 0.05$; **$p < 0.01$.

for ouabain when injected into the LPO. These two experimental groups show significant amnesia by 15 min posttraining ($p < 0.01$), as shown in Figs. 1 and 2.

EXPERIMENT 4: HEMISPHERIC ASYMMETRIES IN THE CHICK

Since the chick brain exhibits hemispheric asymmetries during learning, as mentioned in the Introduction section, this experiment investigates the significance of hemispheric asymmetry in the LPO and IMHV during ITM formation. If the memory is primarily maintained in the left or right IMHV, then injecting ouabain into the structure that maintains the memory trace will produce amnesia. Injecting ouabain unilaterally into either the left or right LPO will similarly produce amnesia in the structure that maintains the memory.

All chicks were given bilateral injections into the IMHV or LPO. Unilateral injections of ouabain (0.027 mM) into the left or right brain structure were paired with a unilateral injection of saline into the symmetrical structure. There were also groups of chicks that received a bilateral injection of ouabain (0.027 mM) or saline into the IMHV or LPO. There were 45–56 chicks per group. The eight experimental conditions in this experiment, as well as the results, are shown in Table 3.

RESULTS

The results of the experiment show that ouabain injected into the left IMHV and into both left and right LPO produces amnesia. Ten μl ouabain injected into the left LPO produced an equivalent degree of amnesia as a bilateral injection of ouabain into the LPO, 42% and 43% retention, as shown in Table 3. Both groups of chicks showed significant amnesia ($p < 0.01$) as compared to saline controls. Chicks given saline injections into the LPO demonstrated 89% retention. Chicks receiving a unilateral injection of ouabain into the right LPO also produced significant amnesia as compared to saline controls (63%, $p < 0.05$). This amnesia was significantly less than that in the groups receiving a bilateral injection of ouabain (43%, $p < 0.01$) or unilateral left injection of ouabain in the LPO (42%, $p < 0.01$).

Chicks that received ouabain injections into the left IMHV or both left and right IMHV showed significant amnesia ($p < 0.01$). These two groups showed 45 and 37% retention, respectively. However, ouabain injected unilaterally into the right IMHV did not produce significant amnesia as compared to saline controls: 84 and 93% retention, respectively.

GENERAL DISCUSSION

The data presented in Experiments 1–3 show that there are specific areas of the chick brain involved in memory formation. The cerebellum, however, was not sensitive to the amnestic treatment given to the LPO or IMHV. It is possible that the cerebellum functions utilizing a long-term depression mechanism rather than a long-term potentiation mechanism during passive avoidance learning. At any rate, the memory trace of the learned experience was not affected by amnestic agents injected into the medial cerebellum. Likewise, agents that disrupt STM and LTM do not affect the memory when injected into the LPO. These findings support the conclusions that the chick brain utilizes specific areas during memory formation and that the agents used in these experiments do not diffuse in effective concentrations to other areas of the brain. The data show that the LPO of the chick is an area involved in only ITM formation, whereas the IMHV is involved in all three stages of memory formation: STM, ITM, and LTM. Both morphological and biochemical correlates of learning have been reported in the LPO and IMHV, indicating the importance of these structures during learning (16,18,25).

One hypothesis for the roles of the LPO and IMHV in

memory formation suggests that these two areas maintain the ITM trace. The results show that if either the LPO or IMHV is injected with ouabain amnesia for peck-avoidance learning occurs. This suggests that the memory trace is shared between the IMHV and the LPO. This is supported by the similar time courses for the appearance of amnesia when ouabain is injected into either of these structures. It is hypothesized that the memory trace is dispersed to the LPO from the IMHV since there is no evidence of an STM trace in the LPO.

The hypothesis further suggests that the memory trace, formed in the IMHV, is transferred to additional areas for other stages of memory formation. The LPO, therefore, may be functioning as an area that receives the ITM trace from other areas such as the IMHV or the LNS that are known to maintain the memory trace during all stages of memory formation (20). Other areas may be functioning in conjunction with the IMHV during STM and LTM. Two hypothesis have been proposed by Horn and Rose for the processing of LTM (10,23).

Using an imprinting paradigm, Horn has shown that the memory trace for the imprinted object is transferred from one area, the IMHV, to another area, not yet identified. This unidentified area has been referred to by Horn as the *s'* store. The proposed model utilizes a time-ordered mechanism linking these two areas. Recent findings have also shown evidence of time-ordered processing of a memory trace between the IMHV and another area, speculated to be the LPO (21).

The model proposed by Rose is derived from results of passive avoidance learning in chicks similar to the procedure described here. Rose reports that although there appears to be a systematic processing of the LTM trace between the IMHV and LPO this is not the case. He reports that a posttraining lesion of the left IMHV would produce amnesia if the flow of information is from the left to right IMHV followed by further processing in the LPO. This experimental design did not disrupt the LTM trace. The data revealed that there are other areas involved in LTM in conjunction with the IMHV and LPO.

TABLE 3

% RETENTION FOR A 24-h TEST AFTER UNILATERAL INJECTIONS OF OUABAIN INJECTED INTO THE IMHV OR LPO

	Groups in Experiment 4			
Injection Site	Left Saline, Right Saline	Left Ouab, Right Ouab	Left Saline, Right Ouab	Left Ouab, Right Saline
LPO	89	43*	63†	42*
IMHV	93	37*	84	45*

Injections (10 μl hemisphere) were made 5 min before training into the IMHV or LPO. Values represent the % retention for each group. n = 45–56 per group. *$p < 0.01$; †$p < 0.05$.

In the hypothesized model presented here, the LPO may function as the *s'* store for the ITM trace exclusively, which is different from the function of the LPO and IMHV proposed by Horn and Rose. The data presented here support the hypothesis that the LPO is involved in maintaining the ITM trace along with the IMHV. The data, however, do not show that the LPO can maintain the memory trace without the cooperation of the IMHV. This is not a characteristic function of the *s'* store or the model proposed by Rose (6,10,23). The difference between the role of the LPO proposed here and the role of the LPO proposed by Horn and Rose can be interpreted as differences in the learning paradigms employed and the method of intervention.

Experiment 4 was conducted to determine if the left or right IMHV disperses the memory trace preferentially to the left or right LPO or to both left and right LPO. The data

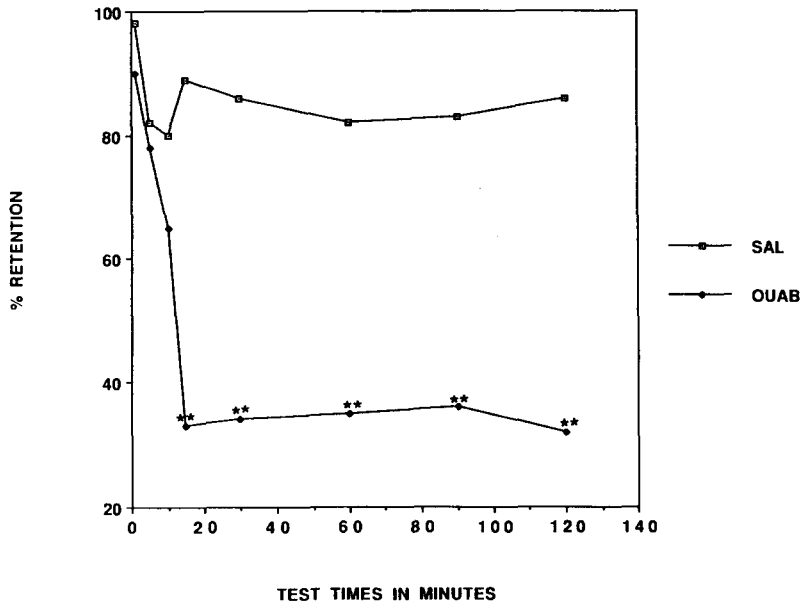

FIG. 2. Time course for appearance of amnesia after injections of ouabain 0.027 mM or saline into the LPO. Injections (10 μl/hemisphere) were made bilaterally into the LPO 5 min before training. Groups of chicks were tested at 1, 5, 10, 15, 30, 60, 90, or 120 min after training. n = 30–40 per group. **$p < 0.01$.

presented show that the memory processing occurring in the left LPO maintains more of the memory trace than in the right LPO. In addition, injections of ouabain into the left LPO produced amnesia to the same degree as a bilateral injection of ouabain into both left and right LPO. This evidence implies that the memory trace maintained in the left LPO is similar to the memory trace in the left IMHV since injections of ouabain into both of these areas produces equivalent degrees of amnesia. The LPO, therefore, presumably receives the memory trace from the left IMHV since an injection of ouabain into either the left or right LPO produces amnesia. These data suggest that it is the left IMHV that is responsible for dispersing the ITM trace to other areas of the brain, such as the LPO. The hemispheric asymmetry between the left and right LPO is not as robust an effect, suggesting that the memory trace is more evenly distributed between these structures.

The findings reported here indicated that the formation of ITM utilizes a neural circuit involving the IMHV and LPO. Further investigation of the LPO and IMHV will lead to a better understanding of the neural mechanisms pertinent to the different stages in which these areas participate.

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