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Faster Acquisition of an Olfactory Discrimination Following Septal Lesions in Male Albino Rats[1,2]

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VOM SAAL, F. S., L. W. HAMILTON AND R. J. GANDELMAN. *Faster acquisition of an olfactory discrimination following septal lesions in male albino rats.* PHYSIOL. BEHAV. 14(6) 697–703, 1975. – Normal rats and rats with septal lesions were maintained on a 23.5-hr water deprivation schedule and trained to bar press for water reinforcement, which was available during the presentation of one odor (S^D) but not another (S^Δ). Vanilla and vinegar were the olfactants. Both groups showed evidence of discrimination within the first 2-hr of training and reached asymptotic discrimination ratios greater than 90 percent, but the rats with septal lesions reached successively higher levels of discrimination faster than the controls. The results suggest a septal inhibitory influence on the olfactory bulbs.

Septal Lesion Olfactory discrimination Olfactometer Inhibition

IN 1934, Swann [31] demonstrated that large areas of the rat's rhinencephalon could be destroyed without disrupting the performance of an olfactory discrimination task. The finding of Brown and Ghiselli [4] that no single subcortical area of the rat's brain could be linked directly to a decrease in performance of an olfactory discrimination task further added to the idea that the term smell brain was perhaps a misnomer. Since the structures previously grouped together in the rhinencephalon have been associated with many behaviors unrelated to olfaction, the term limbic system is used today to describe forebrain structures surrounding the diencephalon. There has been a resurgence of interest in the relationship of many limbic structures to olfaction, however, especially in relation to pheromones and their ability to release specific behaviors. Also, Swann's [31] paradigm has been criticized [21] in that to reach the goal box the rat had to tunnel through scented shavings which could have entered its mouth and stimulated taste receptors.

The septum is the most anterior structure of the limbic lobe and has been implicated in many different types of inhibitory behaviors. In aversive paradigms where the organism is required to inhibit a previously rewarded response, animals with septal lesions appear to be less able to inhibit responding in the face of repeated punishment [24]. In appetitive paradigms it has been found that rats with septal lesions respond at significantly higher rates than controls under continuous reinforcement [17,22], fixed ratio [22], fixed interval [14,17], and differential reinforcement of low rate [6,23] schedules of reinforcement. Increased responding following septal lesions occurs when food or water is used as the reinforcement. Increased resistance to extinction also occurs following septal lesions [19, 27, 30].

The septum also appears to be involved in reactivity to sensory stimuli. Increased reactivity to light [15], sound and heat [5], cold [32], and shock [20] have been reported following septal lesions. Changes in reactivity to both non-nutritive sweet (saccharin) and bitter (quinine) solutions, as well as increased reactivity to sucrose, have also been reported following septal lesions [1,2].

Recent experiments with septal lesions in mice have indicated that lesions of the septum may enhance the acquisition of an olfactory discrimination but not affect the rate of acquisition of a visual or auditory discrimination in a go/no-go head poke paradigm [9]. The present paradigm was designed to allow a comparison between the inhibitory decrements and facilitated reactivity to stimuli which follows lesions of the septum in rats. These two effects were pitted against each other in such a way that any

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decrement in inhibition would work against the faster acquisition of the olfactory discrimination as measured by the rate of bar pressing during the S^D and S^Δ periods.

METHOD

Animals

The animals were 14 male Blue Spruce albino rats, 175 days old at the beginning of testing. All animals were individually housed throughout the experiment.

Apparatus

The apparatus consisted of two standard Lehigh Valley operant chambers enclosed in sound attenuating boxes (Fig. 1). A motor-driven magazine located outside the chamber delivered a drinking tube through a 2 cm dia. hole located 5 cm from the response lever on the back wall of the chamber. Each box had a fan located on the back which evacuated the odors through a 10 cm dia. evacuation tube and, subsequently, outside the building. A 2.5 cm dia. vent in the front of the box with a grid covering allowed air to enter the box. The odors were introduced just beneath the grid floor at the front of the chamber, thus passing through the chamber which was constantly being evacuated. A double glass window in the front of the box allowed visual observation of the rat's behavior. A 7.5 W house light was on in the box at all times. A second 2.5 W light, located above the lever, was used to signal time-out periods between odor presentations.

The odor delivery system consisted of a compressor which pumped air through either of two flasks by means of solenoids. One flask contained vanilla extract and the other contained distilled vinegar. The flasks were connected to the compressor and operant chambers by means of ¼ in. i.d. plastic tubing. Before entering the flasks, the air passed through both charcoal and a dessicant. Two more solenoids located just outside the chamber prevented odors in the tubes from entering the chamber while a trial was not in progress. Gilmont No. 12 flow meters were placed on the plastic tubing outside of the boxes so that the rate of air flow could be measured. The odors were introduced into the chambers at the rate of 1500 ml of air per min. The solenoids were alternated randomly to eliminate the possibility of their sound becoming a discriminative stimulus. Standard mechanical programming equipment was used. The number of bar presses and reinforcements were recorded on digital counters and on a cumulative recorder.

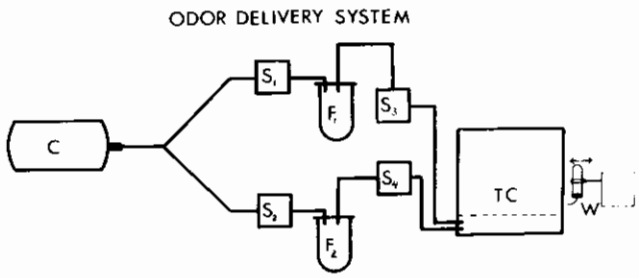

FIG. 1. Odor delivery system: C = compressor, S_1 – S_4 = solenoids to control air flow, F_1 = flask containing vinegar, F_2 = flask containing vanilla, TC = testing chamber, W = water bottle and delivery mechanism. Connections are made via plastic tubing.

Procedure

All animals were given ad lib access to Purina lab pellets in the home cage throughout the experiment and were maintained on a 14/10 light/dark cycle. Five days prior to shaping, all subjects were placed on a 23.5 hr water deprivation schedule in the home cage. All rats were shaped on a CRF schedule until they had obtained 20 reinforcements, each reinforcement consisting of a 5 sec period of access to the drinking tube. During shaping, the odors were not presented, but the compressor was in operation. After all rats had received 20 reinforcements, ad lib access to food and water was allowed overnight, and surgery was performed the next day.

Surgery. Eight rats received large bilateral septal lesions, and 6 rats served as operated controls. Surgery was performed under Equithesin (Jensen-Salsbery) anesthesia (0.25 cc/100g body weight). Lesion electrodes were stereotaxically lowered into the septum bilaterally using the following de Groot [13] coordinates: AP = +7.8 mm; L = ±0.8 mm; H = +1.2 mm. Bilateral lesions were made using 1.5 mA of anodal current delivered through each electrode for 15 sec. Control rats had a hole drilled in the skull, and the dura was punctured, but the electrode was not lowered into the brain.

Postsurgical recovery. All rats were given ad lib access to food and water for 4 days following surgery. On Day 5 all rats were returned to the 23.5 hr water deprivation schedule with ad lib access to food. On postsurgical Day 7 all rats were reshaped on a CRF schedule to a criterion of 20 reinforcements which, for most rats, required only about 5 min. Again, no odors were presented.

Acquisition training. On postsurgical Day 8 discrimination training was begun. The groups were subdivided and for one-half of each group vanilla served as the S^D and vinegar as the S^Δ; for the other half, vinegar served as the S^D and vanilla as the S^Δ. Sessions were 30 min long and consisted of six trials, 3 S^D and 3 S^Δ, each 5 min long and arranged in a pseudorandom series. The only restriction placed on the order of the trials was that no more than 2 S^D or S^Δ trials were presented consecutively. The odor trials were separated by a 30 sec intertrial interval during which the odor from the previous trial was evacuated from the box. During the intertrial interval the light located above the lever was turned on. Each rat received one session per day. Reinforcement was available during the S^D trials on a VI 30 sec schedule. The duration of water availability was 3 sec rather than 5 sec as was used during shaping. All rats were given 16 days of acquisition training.

Stimulus control. On Days 17 and 18 the plastic tubing was disconnected from the flasks, and each rat received two no-odor sessions to determine if the rats were utilizing the odors of some extraneous cue as the discriminative stimulus. On Days 19–21 all rats were given three more days of acquisition training to reestablish the discrimination before reversal training was begun.

Reversal training. On Day 22 reversal training was begun. All rats for which vanilla had served as the S^D were now reinforced only in the presence of vinegar. All rats that had been reinforced during acquisition in the presence of vinegar were now reinforced only in the presence of vanilla. Reversal training was continued for 15 days. Reinforcement was again available during the S^D trials on a VI 30 sec schedule.

Histology. Following termination of reversal training rats

with septal lesions were sacrificed for histological verification of the lesions. They were anesthetized with Equithesin and perfused intracardially with isotonic saline followed by 10 percent formol saline solution. The brains were removed and placed in a glucose-formol saline solution for 5 days. Every fourth 40 μ thick frozen section was mounted and stained with cresyl violet.

RESULTS

Histology

All eight rats in the experimental group were found to have extensive damage to the anterior septal area with some posterior sparing. Reconstructions depicting the extent of the lesions are shown in Fig. 2.

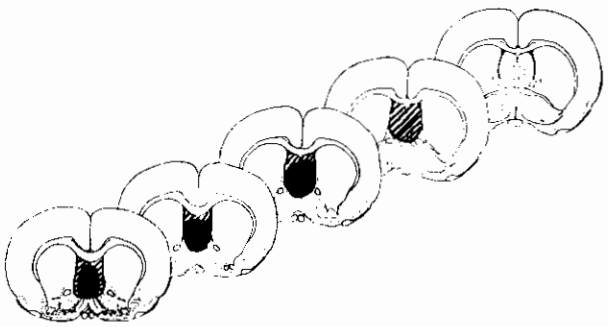

FIG. 2. Reconstructions of the minimum (shaded) and maximum (lined) extent of brain damage.

Acquisition Training

All rats performed at chance levels on Day 1 of acquisition training. By Day 11 the mean discrimination ratios for both groups were about 90 percent. Since no difference was found in the rates of acquisition of the olfactory discrimination based on the odor used as the S^D, the data for all experimental rats were pooled as were the data for all control rats. Analysis of variance performed on the discrimination ratios for the first 11 sessions revealed a significant Groups effect, $F(1,12) = 6.46$, $p<0.05$, the mean discrimination ratio for the rats with septal lesions being 78.7%, and the mean for the control rats being 71% for the first 11 sessions. There was also a significant Sessions effect, $F(10,120) = 18.31$, $p<0.001$.

In an attempt to reduce any intrasession variability which might have resulted from the chance sequence of S^D and $S^\wedge$ trials, a single score was calculated to reflect each rat's level of discrimination for sliding blocks of 3 S^D and 3 S^Δ trials. Each session consisted of 3 S^D and 3 $S^\wedge$ trials so that over the 16 acquisition sessions there were 48 S^D and 48 S^Δ trials. The formulae for calculating the scores, known as sliding averages [33], are presented in Table 1. These discrimination ratios were used to determine the number of S^D trials that each rat needed to reach successive levels of discrimination (see Fig. 4). Analysis of these data revealed a significant Groups X Trials interaction, $F(7,84) = 2.88$, $p<0.01$, indicating that rats with septal lesions reached successively higher levels of discrimination faster than did controls.

In addition to these indices of discrimination, some general observations on the rate and distribution of responses may also be in order. The total number of bar presses per session for rats with septal lesions was significantly higher than for control rats, $F(1,12) = 27.15$, $p<0.001$. The critical value (C.V. = 72) indicated that rats with septal lesions differed from control rats on all sessions, since the difference between the group means was greater than 72 for all sessions. There was also a significant Sessions effect, $F(10,120) = 2.76$, $p<0.005$, indicating that both septal and control rats increased their rates of bar pressing during acquisition (see Table 2). Analysis of variance performed separately for the total number of bar presses per session during only the S^D trials over the first 11 acquisition days revealed that the brain-damaged rats responded significantly more during the S^D trials than did control rats, $F(1,12) = 20.89$, $p<0.001$, C. V. = 66. A significant Sessions effect, $F(10,120) = 8.49$, $p<0.001$, revealed that both control rats and rats with septal lesions increased their rates of bar pressing during the S^D trials over the course of

TABLE 1

FORMULAE USED IN THE CALCULATION OF SLIDING AVERAGES FOR BLOCKS OF 3 S^D AND 3 S^Δ TRIALS

$$\frac{\dfrac{S^{D}_{1} + S^{D}_{2}}{S^{D}_{1} + S^{D}_{2} + S^{\Delta}_{1} + S^{\Delta}_{2}} + \dfrac{S^{D}_{2} + S^{D}_{3}}{S^{D}_{2} + S^{D}_{3} + S^{\Delta}_{2} + S^{\Delta}_{3}}}{2},$$

$$\frac{\dfrac{S^{D}_{2} + S^{D}_{3}}{S^{D}_{2} + S^{D}_{3} + S^{\Delta}_{2} + S^{\Delta}_{3}} + \dfrac{S^{D}_{3} + S^{D}_{4}}{S^{D}_{3} + S^{D}_{4} + S^{\Delta}_{3} + S^{\Delta}_{4}}}{2},$$

$$\frac{\dfrac{S^{D}_{46} + S^{D}_{47}}{S^{D}_{46} + S^{D}_{47} + S^{\Delta}_{46} + S^{\Delta}_{47}} + \dfrac{S^{D}_{47} + S^{D}_{48}}{S^{D}_{47} + S^{D}_{48} + S^{\Delta}_{47} + S^{\Delta}_{48}}}{2}$$

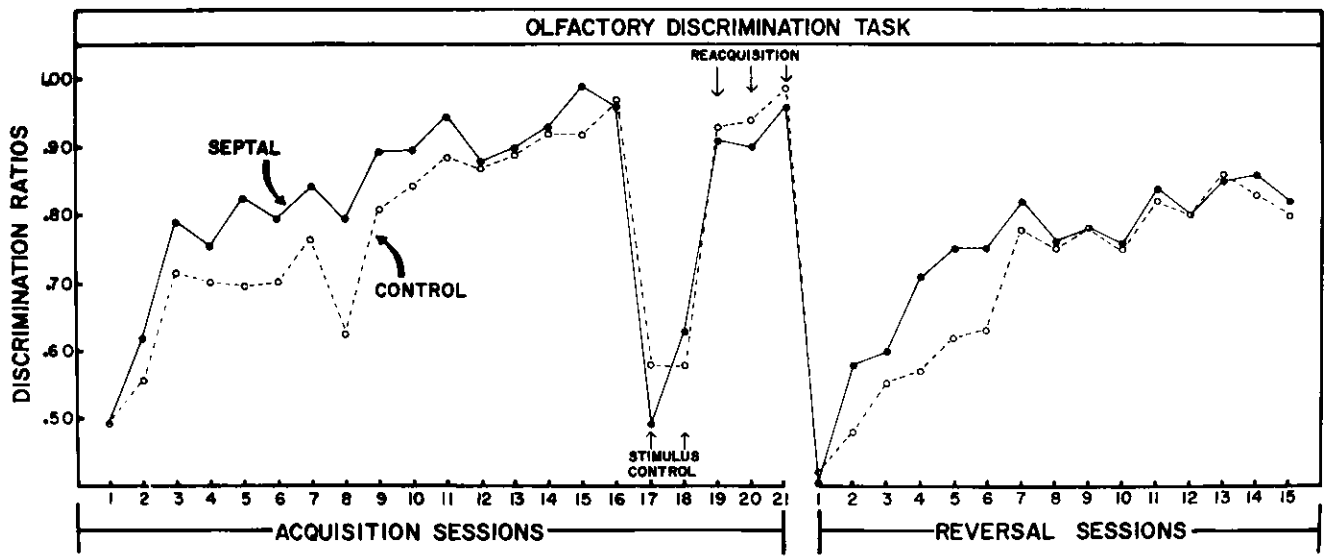

FIG. 3. Mean discrimination ratios $[S^D/(S^D + S^\Delta)]$ per session for acquisition and reversal. Sessions 17 and 18 served as stimulus control days, and no odors were presented.

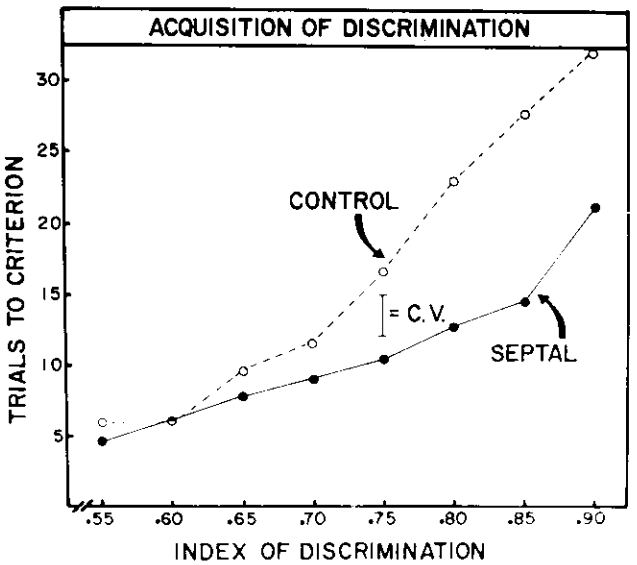

FIG. 4. The mean number of S^D trials needed to reach successive levels of discrimination, based on sliding blocks of 3 S^D and 3 S^Δ trials (see Table 1).

acquisition. However, while the total number of S^Δ responding per session for the experimental rats was initially higher than that of the controls, by the third session there was no significant difference in S^Δ responding per session between the septal lesioned and control rats. Analysis of these data revealed a significant Groups X Sessions interaction, $F(10,120) = 3.92$, $p<0.001$, C.V. = 37, indicating that the rats with septal lesions were able to inhibit responding during S^Δ (extinction) trials as well as controls by the third session.

With removal of the odors on Days 17 and 18 the discrimination ratios for both groups dropped to chance levels. With introduction of the odors on Days 19–21, all rats exhibited discrimination ratios of 90 percent or above by Day 21 (see Fig. 3).

Reversal Training

Once the olfactory discrimination task had been learned, reversing the odors did not result in any significant difference in the rate of reacquisition of the discrimination between the rats with septal lesions and the controls. Analysis of variance performed on the discrimination ratios for the first 7 days of reversal training revealed only a significant Sessions effect, $F(6,72) = 16.79$, $= p<0.001$, indicating that both groups relearned the discrimination (see Fig. 3).

The rates of bar pressing per session during reversal training were similar to those observed during acquisition training in that there was a significant Groups effect, $F(1,12) = 13.77$, $p<0.005$, C.V. = 38, and a significant Sessions effect, $F(14,169) = 2.84$, $p<0.001$. Analysis of the total number of responses during the S^D trials per session revealed that rats with septal lesions responded significantly more than controls, $F(1,12) = 13.74$, $p<0.005$, C.V. = 99. A significant Sessions effect was also found, $F(14,169) = 13.74$, $p<0.001$. Analysis of the total S^Δ bar presses per session revealed a significant Groups X Sessions interaction, $F(14,169) = 6.01$, $p<0.001$, C.V. = 48, but the rats with septal lesions took longer to reduce their S^Δ response level to that of the control rats than was the case during the initial acquisition phase of the experiment (see Table 2). It is possible that due to the impaired ability of septal-damaged rats to reverse a postoperatively acquired response to positive reward [16] that the rats with septal lesions did not acquire the reversal task faster than the control rats. This is supported by the fact that while during the initial acquisition of the olfactory discrimination the S^Δ response rates for septal lesioned and control rats were not significantly different after the second session, it was not until Day 7 of reversal training that the S^Δ response rates of the rats with septal lesions dropped to control levels.

TABLE 2

MEAN NUMBER OF BAR PRESSES PER SESSION

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	C.V.‡
											Acquisition											
Total $S^D + S^\Delta$																						
Septal†	348	365	305	308	291	350	333	409	356	397	332	459	434	439	406	379	479	597	387	487	367	72
Control	137	108	133	154	127	207	176	213	173	259	209	232	251	225	256	217	254	206	225	246	226	
Total S^D																						
Septal†	171	223	239	233	239	275	281	314	313	356	300	406	385	408	400	363	247	373	358	434	383	66
Control	73	62	99	116	93	152	132	156	142	217	181	201	223	203	234	210	150	121	207	232	222	
Total S^Δ																						
Septal	176*	141*	65	73	51	74	52	85	42	40	31	54	50	31	6	16	231*	225*	30	53	19	37
Control	64	45	33	38	33	54	43	56	30	41	27	31	28	22	23	8	104	85	18	14	4	
											Reversal											
Total $S^D + S^\Delta$																						
Septal†	587	475	581	567	533	608	480	518	470	521	589	582	563	581	478							138
Control	218	205	292	273	290	269	307	314	269	342	395	362	357	414	303							
Total S^D																						
Septal†	235	273	348	400	394	445	394	393	359	390	491	464	477	497	388							99
Control	100	127	185	182	209	203	239	232	216	253	323	292	306	324	230							
Total S^Δ																						
Septal	352*	203*	233*	167*	139*	164*	87	125	114*	131	98	117	88	83	90							48
Control	118	78	107	91	80	66	68	82	53	89	73	70	51	92	73							

*Significantly different from controls on sessions indicated ($p<0.05$).
†Significantly different from controls across all sessions ($p<0.05$).
‡The critical value (C.V.) is based on the mean square of the error term and hence reflects the variance of the groups [C.V. = t0.05(2 MS/n)½].

There were no differences in the number of reinforcements received during the initial acquisition or the reversal sessions between the septal lesioned or control rats.

DISCUSSION

Lesions of the posterior septum produce primary hyperdipsia in rats [3]. Carey [7] has reported an increase in water intake with ventral posterior septal lesions in rats not accompanied by an increase in response rate for water reinforcement. Similarly, an increase in operant responding for water reinforcement not accompanied by an elevated ad lib water intake occurred following dorsal anterior septal lesions. The dissociation of increased water intake and response output with discrete septal lesions indicates that neither increases in water intake nor response output for water reinforcement reflect an increase in thirst motivation. Blass and Hanson [3] concluded that septal hyperdipsia was a result of the removal of an area that inhibited the lateral hypothalamus, and that the septum regulates the amount of water consumed in response to intravascular fluid depletion. Wishart and Mogenson [36] found that electrical stimulation of the septum resulted in decreased water intake in ad lib and water deprived rats. They proposed that this was due to the septum receiving information from cells signalling expansion of the intracellular volume and exerting an inhibitory influence on the lateral hypothalamic drinking system. Keesey and Powley's [18] finding that septal lesions resulted in a decrease in the threshold for lateral hypothalamic self stimulation indirectly supports this hypothesis. Carey [7] interprets changes in bar press rates following dorsal anterior septal lesions as resulting from a decrement in response inhibition. It is unlikely, therefore, that the results of the present experiment are due to a heightened responsivity to the water reinforcement or are an artifact of the increased response output. Carlson and Vallante [9] have reported that mice which increase their response rates following septal lesions do not acquire either a visual or auditory discrimination faster than controls.

The anatomical structure of the olfactory system argues against any modulation of detection thresholds at the receptor level following septal lesions. It is possible that the septum may exert an inhibitory influence on the olfactory bulbs via its interconnections with the olfactory tubercle. The olfactory bulbs, to which the axons of the olfactory receptor cells project, are extensions of the cerebral hemispheres and as such are part of the central nervous system. The olfactory epithelium differs from other sensory systems in that it does not appear to contain intrinsic neurons that could influence the transmission of impulses from the receptor cells to the olfactory bulbs. The retina and cochlea are innervated directly by centrifugal fibers, while the olfactory epithelium seems to be lacking this direct input from central olfactory areas. Centrifugal olfactory fibers project only as far as the olfactory bulbs and may serve to inhibit or facilitate activity in these structures [10]. Thus, any inhibitory effect of the septal damage would necessarily be exerted on neurons in the olfactory bulbs. Ottoson and Shepherd [25], in trying to explain the finding that olfactory bulb mitral cells and olfactory receptor cells respond continuously without decline in amplitude for over an hour to repeated stimulation of moderate intensity, have hypothesized that secondary (central) olfactory areas may influence the excitatory state of neurons in the olfactory bulbs. Since receptor and mitral cells respond repeatedly with an apparent absence of adaptation, the commonly observed rapid decrease in olfactory perception that occurs with prolonged exposure to a given odor may depend on inhibition at the level of the olfactory bulbs by secondary olfactory areas.

It has been found that the septum receives fibers from and projects fibers to the olfactory tubercle. Afferent fibers from the olfactory tubercle pass via the medial aspect of the medial forebrain bundle and terminate in the medial septum and vertical limb of the bed nucleas of the diagonal band of Broca. Short efferent fibers pass ipsilaterally from the lateral septum along the medial aspect of the accumbens nucleus to terminate in the olfactory tubercle. There does not appear to be a direct afferent connection to the septum from the olfactory bulbs or anterior olfactory nucleas [11, 12, 28]. Cragg [12] has reported that there are bulbopetal fibers from the ipsilateral olfactory tubercle which project to the deep bulbar layers around the glomeruli. These fibers appear to synapse with granule cells which in turn from synapses with the secondary dendritic processes of the mitral cells. The olfactory tubercle may be the primary afferent relay nucleas for bulbopetal fibers from secondary olfactory areas [26]. Any central inhibitory influence would probably be on the excitability of the mitral cells which are though to be the sole source of efferent fibers from the olfactory bulbs to secondary olfactory nuclei [34]. Rall *et al.* [29] reported that stimulation of the granule cells in the olfactory bulbs resulted in inhibitory postsynaptic potentials (IPSP) in the mitral cells. Inhibition of the granule cells was found to result in disinhibition of the mitral cells (i.e., the IPSP was abolished). Thus, it would be through inhibition or stimulation of the granule cells that the excitability of the mitral cells could be modulated by central olfactory areas.

The present data support the finding of Carlson and Vallante [9] that mice with septal lesions acquired an olfactory (but not an auditory or visual) discrimination task faster than controls in a go/no-go head poke paradigm. Mice with septal lesions responded at higher rates under all stimulus conditions but only acquired the olfactory discrimination faster than the controls. This indicates that the increased rate of acquisition of the olfactory discrimination shown by septal-damaged mice was not an artifact of the heightened response rate observed following septal lesions.

It may be that there is an enhanced responsivity to olfactory stimuli as a result of a loss of inhibitory input into the olfactory bulbs following destruction of the septal area. While the septum has traditionally been regarded as being involved in behavioral inhibition, the present data, as well as that of Carlson and Vallante [9], indicate that the septum may also be involved in physiologic inhibition in the olfactory bulbs as well.

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