THE NEUROBIOLOGICAL MECHANISMS UNDERLYING THE SENSITIZATION OF PAIN AND LEARNING

A Senior Honors Thesis

by

BRIANNE COLEMARIE PATTON

Submitted to the Office of Honors Programs
& Academic Scholarships
Texas A&M University
in partial fulfillment of the requirements of the

UNIVERSITY UNDERGRADUATE
RESEARCH FELLOWS

April 2001

Psychology 2

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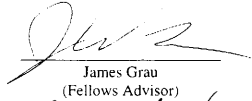
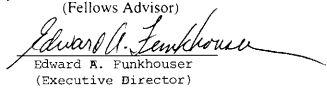


James Grau
(Fellows Advisor)



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April 2001

Psychology 2

ABSTRACT

The Neurobiological Mechanisms Underlying the Sensitization of Pain and Learning. (April 2001)

Brianne Colemarie Patton
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Why animals learn and how they do so has long been a topic of inquiry and research. Recently, King, Joynes, Meagher and Grau (1996) showed that exposure to a mild aversive event (a brief shock) can enhance both learning and pain. My thesis explored the neural mechanisms that underlie this effect. Prior research suggested that the septum may play a role in both pain and learning, but the relationship between these two phenomena had not been defined. My hypothesis was that exposure to an aversive event may impair the activity of the septum and thereby enhance stimulus processing. This in turn could increase the painfulness of subsequent aversive stimuli (hyperalgesia) and facilitate learning. If my hypothesis is correct, then pharmacologically inducing a state of arousal (by injecting scopolamine, an acetylcholine antagonist) should sensitize both pain and learning. As predicted, Experiment 1 showed that scopolamine induces hyperalgesia in a dose-dependant fashion. Experiment 2 examined the drug's effect on learning using a Pavlovian conditioning procedure. If scopolamine enhanced the

painfulness of the aversive unconditioned stimulus, then it should also augment learning. The data show a trend to the non-monotonic function described by Fanselow et al (1994), and can be explained with their explanation of a behavioral systems approach. My data suggest that acetylcholine is indeed playing a role in hyperalgesia and enhanced learning, although more research is needed to further clarify this role.

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The Neurobiological Mechanisms Underlying the Sensitization of Pain and Learning

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Why animals learn and how they do so has long been a topic of inquiry and research. Recently, King, Joynes, Meagher and Grau (1996) showed that exposure to mild aversive event (a brief shock) can enhance both learning and pain. My thesis will explore the neural mechanisms that underlie these effects.

These effects have been linked to the sensitization of pain, a phenomenon known as hyperalgesia. This enhanced sensitivity to pain is observed after exposure to an aversive stimulus, such as shock (Illich, King, and Grau 1995). For example, King, Joynes, Meagher, and Grau (1995) showed that rats exposed to shock vocalized sooner to a noxious thermal stimulus than unshocked controls. Generally, it has been thought that a painful stimulus will induce an opioid anti-nociceptive reaction, and decrease pain perception. However, when vocalization thresholds are used to measure supra-spinally mediated perception, hyperalgesia is uncovered.

The link between enhanced learning and hyperalgesia is evident in a study done by King, Joynes, Meagher, and Grau (1996). They showed that prior exposure to the shock schedule used to induce hyperalgesia (3 0.75s, 1.0mA shocks with a 20s interval) in fact enhanced the reinforcing capacity of an aversive stimulus in both a Pavlovian and instrumental learning task.

This thesis follows the format of Behavioral Neuroscience

These findings suggest that exposure to an aversive event causes an increase in pain, and thereby enhances the affective impact of subsequent aversive stimuli.

Generally, learning is assessed using the behavioral measure, conditioned freezing. Conditioned freezing occurs along a non-monotonic function (Fanselow 1989). At very low, and very high intensities, shock does not support conditioned freezing. Fanselow (1994) provides an explanation of this phenomenon via a behavioral systems approach. He explains, that high, low, and medium shock intesities elicit different reactionary responses from the rat dependant on how much fear the rat is experiencing. Medium shock intensities elicit the freezing response, or a post-encounter response. At low shock intensities, the rat has very little pain, and very little fear, and therefore go into a pre-encounter defense which is described as a number of activities (for a more complete description of defensive modes see Timberlake, 1993; Timberlake & Lucas, 1989). At high shock intensities, the pain experienced mimics that of an actual physical encounter with a predator, and therefore elicits a circa-strike response, which is a burst of activity, i.e. lunging, biting, etc. Fanselow (1994) showed that these rats actually have elevated fear levels as measured by analgesia, and defacation, but exhibit inhibited learning, because the systems engaged in circa-strike prevent the display of freezing.

Relatively little is known about the neurobiological mechanisms that underlie the sensitization of pain and learning. Prior research suggests that the septum may play a role in both pain and learning, but the relationship between these two phenomena has not been defined. My hypothesis is that exposure to an aversive event may impair the

activity of the septum and thereby enhance stimulus processing. This in turn could increase the painfulness of subsequent aversive stimuli (hyperalgesia) and facilitate learning.

As my pilot research progressed, we realized that some additional data were required to strengthen the link to the septum and the induction of arousal. Specifically, if my hypothesis is correct, then pharmacologically inducing a state of arousal (by injecting scopolamine, an acetylcholine antagonist) should sensitize both pain and learning by impairing the acetylcholine based activity of the septum.

Experiment 1

The purpose of experiment one was to assess the role of acetylcholine in sensitization of pain. My hypothesis is that scopolamine will induce hyperalgesia (enhanced pain). As discussed above, the measure of supra-spinally mediated pain is vocalization, specifically vocalization threshold to radiant heat. Because acetylcholine exerts a tonic inhibition over many pathways, it is logical to predict that inhibiting acetylcholine will facilitate pain transmission (possibly by releasing substance P from inhibition). Vocalization as well as tail-flick thresholds were measured to both a noxious thermal stimulus (radiant heat) and gradually incremented shock. A dose response curve was performed using 0.00 (saline), 0.01, 0.1, and 1.0 mg/kg of scopolamine.

Method

Subjects. The subjects were male Sprague-Dawley rats from Harlan in Houston, TX. The rats were between 100-120 days old, and weighed approximately 350-420

grams. All rats will be maintained on a twelve-hour light/dark cycle and procedures will take place during the last half of the light period. There were eight rats per cell, in all of the scopolamine groups, and 12 rats in the saline group for a total of 36 rats.

Apparatus. The apparatuses used during this procedure, were adopted from Meagher et al. (in press) and King et al 1996. The subjects were restrained in opaque, black Plexiglas tubes. These measured 22.0 cm in length, 6.8 cm in diameter, with a 5.5 cm wide Plexiglas floor laying 5.3 cm from the top of the tube. There was also a 1.3 cm wide, 4.8 cm long protrusion of the floor beyond the rear of the tube on which the rat's tail rested. The tubes had ventilation holes in the top of the midsection of the tube. In order to allow the subjects tail to move freely, but keep the rest of the body lightly restrained a band of ortholetic tape was placed across the rear opening of the tube. Chamber fans provided background noise. Tubes were cleaned in between each subject with the disinfecting agent Novalsan. Thermal stimuli were provided by a 375-watt movie light (Sylvania, Type EBR) focused via a condenser lens 8 cm below the light. By placing the rat's tail 4.7 cm below the lens, approximately 2 cm of the rat's tail was illuminated. The tail was placed into a 0.5 cm deep groove cut into an aluminum block, and kept in place under the heat source (still allowing lateral movement) by a 10 cm insulated wire attached to the tip of the tail with porous tape. This wire was attached to an elastic band 11 cm away from the aluminum block. A 0.5 cm lateral movement of the tail triggered a photocell in the aluminum block and terminated the light source. An AC potentiometer (Leviton #6681-W) regulated the light intensity. Tail temperature was

recorded before and after each test (both shock and heat) via a Radio Shack digital thermometer (Model 277 0123) which was taped at the base of the tail.

The electrodes used to deliver gradually incremented tail shock were constructed from a modified fuse clip, covered in electrode paste, and taped to the base of the tail opposite the thermometer. Thresholds to shock were assessed using a manual shocker (BRS/LVE Model SG-903). This allowed continuous variation of shock intensity between 0 and 2 mA. Shock was increased at a constant rate, until a tail flick and a vocalization terminated it. In the absence of the radiant heat device, a small 28-V light, (General Instrumental, 1820) positioned just below the condenser lens, activated the photocell in the event of a tail flick. Vocalizations to stimuli were measured by a microphone (Radio Shack 270-092B) located in a 9.4 mm hole drilled into the front (closed) end of the tubes, 3.5 cm from the bottom. Vocalization levels above 80 dB and 1500 Hz were amplified by a Sanyo amplifier (DCA 611), while frequencies below 1500 Hz were attenuated by approximately 8 dB. The amplified output was passed through a full-wave rectifier that provided a direct-current (DC) voltage that was proportional to the sound intensity recorded by the mocrophone. An analog-to-digital (A-D) converter (Alpha Products, Analog 80) was used monitor this voltage. This system was calibrated by presenting a 4000-Hz sine-wave tone and determining the relation between the digital input and the loudness of the tone. Based on this derived function, the digital inputs were converted to decibels (dB).

Behavioral Measures. All of the responses (latencies and vocalization intensities) were monitored and recorded to the nearest 0.01 second by a Macintosh

computer. The criteria for vocalization responses was 88.6 dB. Each test was terminated after the detection of both a tail flick and a vocalization, or after the maximum time was reached (8 s for heat, 60 s for shock). False alarm trials were conducted to ensure that an overall increase in responding did not influence results (Meagher et al., in press).

Procedure. Rats were given an intra-peritoneal injection of scopolamine (0.01, 0.1, or 1.0 mg/kg) or saline (vehicle),in concentrations that allowed for a volume of 1 ml/kg, and allowed fifteen minutes to acclimate to the laboratory. Next, they were put in the black Plexiglas tubes, an electrode and thermometer were attached to the base of their tail, and they were given fifteen minutes to acclimate to the tubes. They were then exposed to a battery of pain testing during which room temperature was kept constant at approximately 25 degrees Celsius. Their vocalization and motor response thresholds were measured to both radiant heat, and gradually incremented shock. After false alarm levels were obtained, they were tested to either radiant heat, or gradually incremented shock in an ABBA fashion with two minutes in between each test.

Results and Discussion.

As expected, administration of scopolamine lowered pain thresholds in a dose-dependant fashion. For each subject, we recorded four motor responses (two to heat, and two to shock) and four vocalizations. False alarm trials were run for each rat, and tail temperature was recorded before and after each test. The average thresholds in seconds for each group, for each response are provided in Figure 1. ANOVAs and trend analyses were performed, and a $p<0.05$ was considered significant.

Figure1

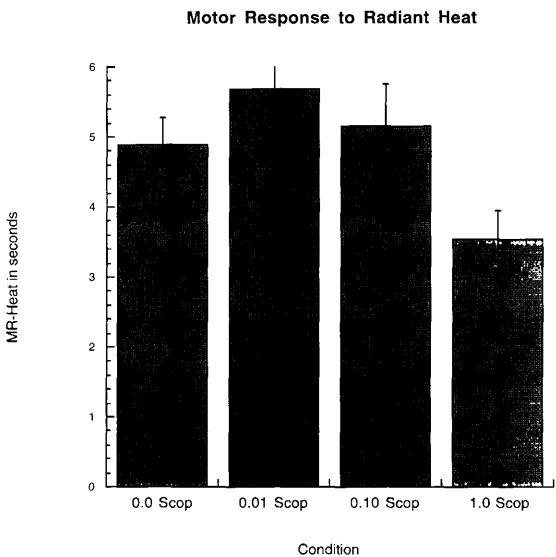

Motor Response to Radiant Heat
MR-Heat in seconds
6
5
4
3
2
1
0
0.0 Scop
0.01 Scop
0.10 Scop
1.0 Scop
Condition

Figure 2

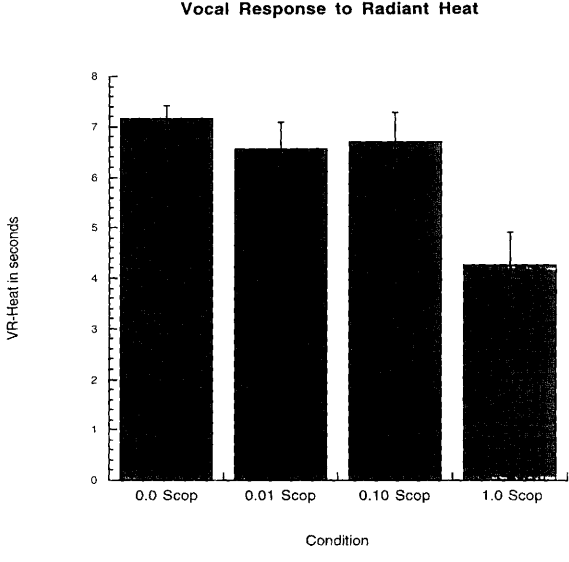

Vocal Response to Radiant Heat
VR-Heat in seconds
0
1
2
3
4
5
6
7
8
0.0 Scop
0.01 Scop
0.10 Scop
1.0 Scop
Condition

Figure 3

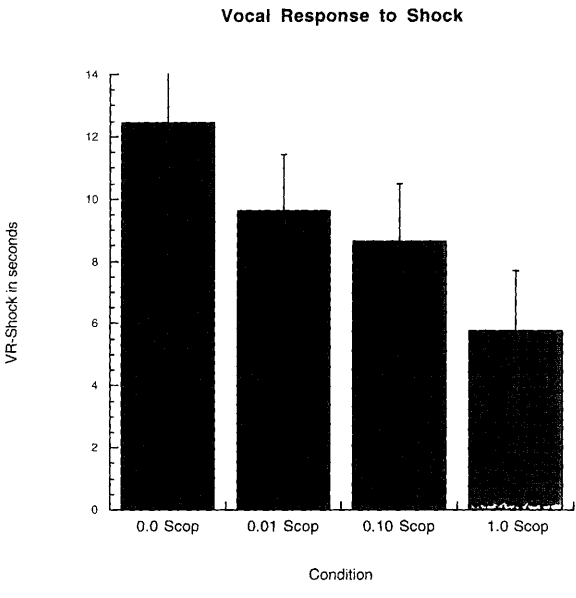
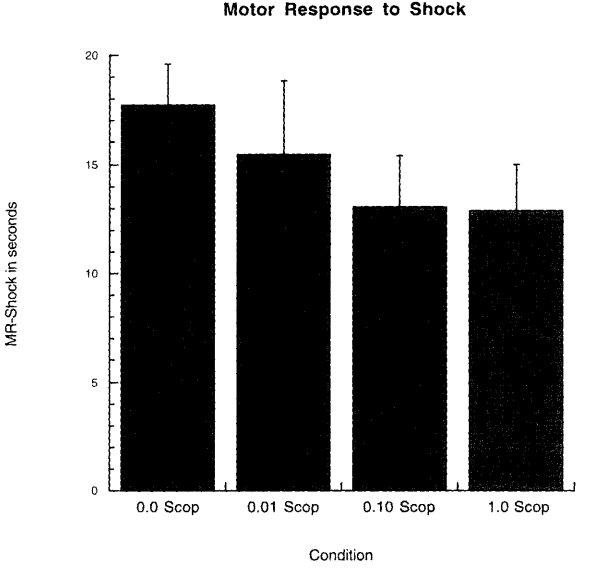

Vocal Response to Shock
VR-Shock in seconds
14
12
10
8
6
4
2
0
0.0 Scop
0.01 Scop
0.10 Scop
1.0 Scop
Condition

Figure 4


Motor Response to Shock
MR-Shock in seconds
20
15
10
5
0
0.0 Scop
0.01 Scop
0.10 Scop
1.0 Scop
Condition

Vocal response to radiant heat produced significant differences, F (3,32)=6.8, p < .05, between groups in a dose-dependant fashion as depicted in Figures 1-4. There also was a significant linear trend (no inflections), F (1,32)=16.0. Neither quadratic (one inflection), or cubic (two inflections), produced significant results.

There was a main effect of motor response to heat F (3,32)=3.6, p < .05, indicating a dose-response curve for this measure as well. For this particular measure, both the linear F (1,32)=5.4, p < .05, and the quadratic, F (1,32)=6.8, p < .05, were significant. The cubic trend analysis revealed no significance.

Although there was no main effect of drug on vocal response to shock, F (3,32)=2.2, p > .05, there was a significant linear trend F (1,32)=6.2, p < .05. Neither the quadratic, or the cubic trends approached significance.

There was no main effect of motor response to shock F (3,32)=1.01, p > .05. Trend anaylses revealed no linear, quadratic, or cubic trend.

These data indicate that scopolamine does indeed adhere to dose response curve. As the dose of scopolamine gets higher, pain reactivity increases. Given that the difference between each dose of scopolamine was significant when two of the four measures were used, and that these two measures (vocalization and motor response to radiant heat) have been observed in known hyperalgesic rats, (King et al. 1996), we can conclude that scopolamine produces the same type of sensitivity to pain as hyperalgesia. This supports my hypothesis that scopolamine induces a hyperalgesic state.

Experiment 2

The next task was to assess the role of acetylcholine in enhanced learning. Given that scopolamine did indeed induce hyperalgesia, then, in keeping with King et al (1996), it should also facilitate learning. To assess this possibility, we used a Pavlovian conditioning paradigm. Scopolamine should increase the affective dimension of pain caused by a weak chamber shock (0.3 mA), and rats should more readily learn an association between the CER chambers and shock. However, we also used a moderate (1.0 mA) shock in the chamber. As discussed above, according to Fanselow et al (1994), conditioned freezing should follow the non-monotonic function, and at 1.0 mA, rats shouldn't freeze as much. Scopolamine could enhance these effects, that is, the scopolamine groups should be much lower relative to the saline group. It is unlikely that scopolamine will disrupt the inhibition of freezing, because, as mentioned above, this inhibition seems to be dependant on the dlPAG, and is mainly glutamate mediated.

Method

Subjects. The subjects were male Sprague-Dawley rats from Harlan in Houston, TX. The rats were between 100-120 days old, and weighed approximately 350-420 grams. All rats will be maintained on a twelve-hour light/dark cycle and procedures will take place during the last half of the light period. There were eight rats per cell, in all groups for a total of 32 rats.

Apparatus. The apparatus used in this experiment was the same as was used in Meagher et al. (in press). Learning was assessed in a conditioned emotional response chamber (CER) measuring 30x26x38 cm (Model RTC-021). The ceiling, front and rear walls were made of clear Plexiglas, while the two side walls were constructed of

stainless steel. The floor consisted of a grid of stainless steel rods 0.4 cm in diameter spaced 1.5 cm apart. This allowed the application of weak (0.5 s, 0.3 mA) or moderate (0.5 s, 1.0 mA) constant current gridshock. Shock was provided by a 660-V transformer through a scrambler. The chambers were housed inside test cubicles, and cubicle fans provided approximately 60 dB of background noise. Chambers were cleaned with a 20% solution of vinegar between each subject, and ambient lighting, as well as house lights in the chambers, was kept constant across conditions. Rats were filmed while in the chambers, with a Hitachi (model 2900A) video camera for subsequent assessment of freezing. Chamber doors were shut, but cubicle doors were left open.

Behavioral Measures. Behavior was scored by a blind rater every three seconds as freezing or moving (activity). Freezing is defined by Fanselow et al (1984) as the absence of all movement except that needed for respiration. Their behavior was observed and scored for three minutes prior to shock and two minutes after shock on day 1, and for eight minutes on day 2. In our lab, using this scoring technique, we have an inter-rater reliability rating of 95% or more.

Procedure. Thirty-two rats were given an injection of either 1.0 mg/kg scopolamine, or saline. They were then allowed to acclimate to the lab for twenty-five minutes. This served to equate the time that the drug was in the system for both experiments. After that, rats were placed in a conditioned emotional response chamber and given a weak or moderate foot shock (0.3 or 1.0 mA respecitively). Their behavior was recorded, and twenty-four hours later, their conditioned fear to the chambers was assessed using conditioned freezing.

Results and Discussion.

I found that scopolamine had an amnesic effect immediately following shock (day 1). There was no significant amnesic effect on day 2. There were four groups: 0.0 mg/kg, and 1.0 mg/kg at 0.03 mA of shock, and 1.0 mA. Zero, and 1.0 mg/kg of scopolamine were chosen because they had the most robust effect, and the effects of lowering or raising the shock intensity is likely to be most apparent here. The results are depicted in Figure 5-7 in terms of percent of time freezing. ANOVAs, again were the analyses performed, and $p < .05$ was considered significant.

Clearly, the day 1 main, effect of drug condition is significant, $F(1,28)=10.08$, $p < .05$. The scopolamine had an amnesic effect during training. There was no crossover interaction between drug condition and shock condition, however, $F(1,28)=2.6$, $p > .05$.

There were no significant effects of drug, $F(1,28)=1.6$, $p > .05$, or shock, condition, $F(1,28)=1.03$, $p > .05$ on day 2. The interaction of shock and drug condition did not prove significant either, $F(1,28)=0.3$, $p > .05$.

When using conditioned freezing as a measure of learning, one needs to take into consideration how accurately this measure portrays fear and learning. In this paradigm, a footshock (US) is paired with a context (CS) and the proportion of conditioned freezing to the chamber represents how well the rat made the association between the context and shock. As mentioned above, Anagnostaras, Maren, Sage, Goodrich, and Fanselow (1999) found that at a dose of 1 mg/kg, impaired learning. Learning, as measured by conditioned freezing, occurs along a non-monotonic function. There is a peak at specific shock intensity, and after that, shock undermines learning. The trend of

the scopolamine group towards a non-monotonic function, is in keeping with my hypothesis. If we were to run more groups, for example, a 0.1 mA shock group, some intermediate scopolamine doses, and boost the number of rats per group, it is very probable that we would see a crossover drug-shock interaction. That is scopolamine, at

Figure 5

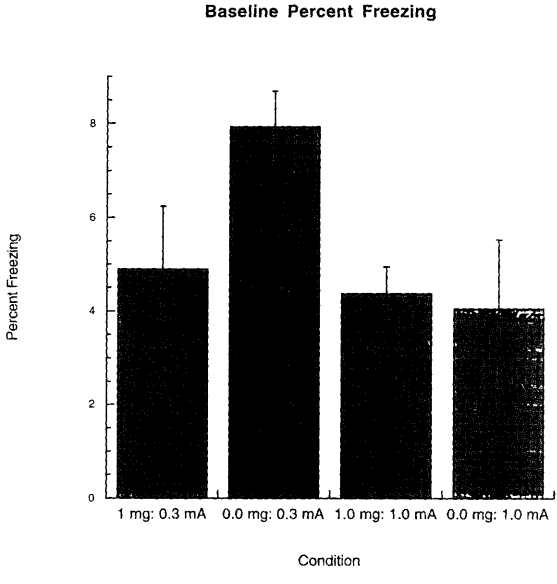


Figure 6

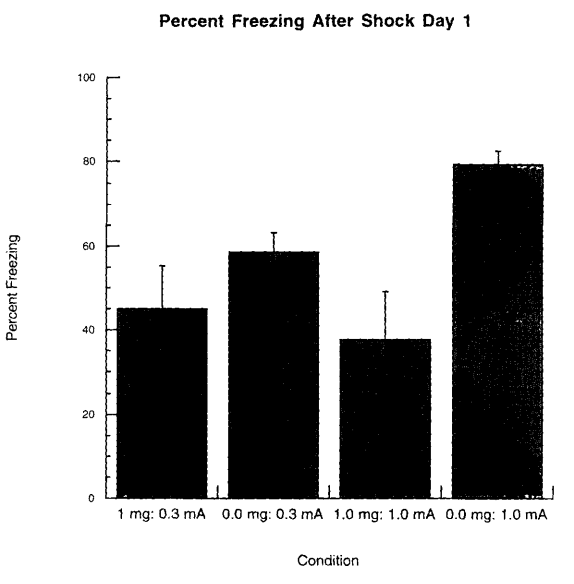
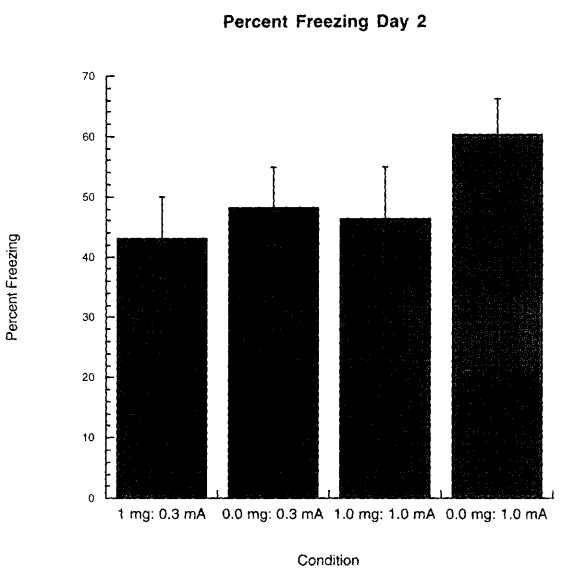

Percent Freezing After Shock Day 1
Percent Freezing
100
80
60
40
20
0
1 mg: 0.3 mA
0.0 mg: 0.3 mA
1.0 mg: 1.0 mA
0.0 mg: 1.0 mA
Condition

Figure 7



high doses, elicits what Fanselow (1994) deem a circa-strike response. This is probably because the increased pain is analogous to that of a physical encounter with a predator. At lower doses, because the pain perception would be lower, subsequently causing less

fear, which Fanselow (1994) claim elicits the post-encounter response of freezing. This will interact with shock intesity, with lower intensities causing a post-encounter defense, and higher a circa-strike defense. Varying levels of shock combined with varying doses of scopolamine should interact. For example, a high dose of scopolamine that usually elicits a circa-strike response, when paired with low shock intensity, would probably elicit a post-encounter response. Measurability also plays a role. In this experiment, saline controls froze at much higher levels than is normally observed. When the control levels of freezing are so high, it is difficult to discern a large difference between groups.

General Discussion

Both of the experiments were informative, and gave support to the idea that acetylcholine is involved in the sensitization of both pain and learning. However, Experiment 1 may be, at this point, the more informative of the two. Response to radiant heat is the more sensitive of the two measures used. During hyperalgesia, vocalization thresholds to radiant heat decrease just as they did with scopolamine. The scopolamine produced various states of hyperalgesia along a dose response curve. The higher the dose of scopolamine, the lower the vocalization thresholds. This suggests that pain reactivity is simply inversely proportional to dose. However, as the doses got higher, tail flick thresholds to radiant heat also decreased. This suggests that scopolamine also has an effect on antinociception. The data suggest that indeed, the higher doses of scopolamine are acting on the spinal cord, and as the doses get lower, the effect is progressively more supra-spinally mediated. This coincides with my hypothesis that low doses of scopolamine will produce hyperalgesia. There is evidence (McCleary, 1961)

that the septum, via acetylcholine exerts a tonic inhibition over various other neural pathways. It is possible that a pathway underlying hyperalgesia is one of these. If an acetylcholine antagonist can inhibit the protective "decreased pain" effect, then it is logical to think that acetylcholine, in the brain, has a protective effect against pain perception, possibly by inhibiting transmission of the painful stimuli. However, in higher doses, scopolamine has an amnesic effect (Anagnostaras, et al., 1999). It does not decrease pain perception at these doses; in fact rats perceive the stimuli as most painful under the highest dose of scopolamine. So why, would rats not learn better about this obviously more painful stimulus? A possible answer comes from Fanselow (1994). These amnesic effects occur only at high shock intensity. At intermediate shock intensities, there is little difference between saline controls, and as the shock intensity goes lower, there is reason to believe that scopolamine may even facilitate learning. It is probable that, at high shock intensities, different behavioral responses are elicited that overshadow the freezing response. Walker et al (1997) showed that in saline rats, fear was still elicited to the CS, by measuring defacation, and fear induced analgesia (decreased pain). We may then postulate that in these rats, learning was still occuring. However in the scopolamine rats, the deficit in freezing, is probably due, not only to a different response system, but also to an amnesic effect. This is supported by the data in experiment one which show that high doses of scopolamine actually increase pain rather than inducing analgesia.

Clearly, we are on the right track to uncovering the underlying pathways of hyperalgesia and enhanced learning. Acetylcholine obviously plays a role. There is

more work to be done to clarify and more thoroughly describe that role. The most pressing issue that needs to be explored is what happens at even lower shock intensity, 0.1 mA for example. It would be in keeping with my hypothesis if at that shock intensity, scopolamine facilitated learning. Also, effects of the intermediate doses of scopolamine at various shock intensities would be informative. At low shock intensities, there may be a crossover effect where a high dose of scopolamine may facilitate learning. After all these data are collected, depending on their results, (however, as of right now, it seems the trend toward my hypothesis will continue) it will become necessary to directly lesion and stimulate the septum. This will allow us to deduce if it is the septum that is responsible for the effects of acetylcholine, or whether or not it is some other structure, or simply acetylcholine in general, non-specific to a neural region.

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Cirriculum Vitae

April 12, 2001

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Education.

1997: Graduation with Honors from Clyde High School in Clyde, TX
1997-present: Four years at Texas A&M University in College Station, TX
Major: Biology and Psychology
Minor: Chemistry

Research Experience.

September 1999-present: Research experience (PSYC 485) in the area of Behavioral Neuroscience with Dr. James Graw, Dept. of Psychology, Texas A&M University. I have run my own experiments, and have a large part in many others currently in progress in the laboratory. I have gained surgical technique, animal care and handling knowledge, and experience with statistics.

March 1998- April 1999: Biological Oceanography research experience with Dr. John Wormuth, Dept. of Oceanography, Texas A&M University. I gained taxonomy information, and research techniques used most frequently in oceanography laboratories.

Spring 1998: Oceanography 491 pertaining to Physical Oceanography, with Dr. Andrew Vastano, Dept. of Oceanography, Texas A&M University. This was in the format of a one on one theoretical discussion on the physical properties and behavior of the ocean.

References.

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