AN ABSTRACT OF THE THESIS OF

Roger Neil Edson for the Master of Science

in Psychology presented on July 11, 1997

Title: Effects of Toxicosis Mediated Potentiation of a Taste/Taste Compound on Drinking and Locomotor Behaviors in Rats

Abstract approved: Stephen F. Davis

The study of taste aversions evolved from watching animals eat something, become ill, and subsequently not eating that substance again. Logically, if the animal is able to associate a certain odor with the substance which caused the illness, it is less likely to begin consuming it. If the odor, which is considered a weak cue compared to taste, becomes a stronger cue because of its association with taste, the odor is said to be potentiated. If odor can be potentiated, perhaps a second, weaker flavor can be potentiated. Next, if taste is a good potentiating stimulus, perhaps stimuli not directly associated with consumption can be potentiated to be aversive. Finally, if various stimuli can be aversive through potentiation, perhaps consummatory behaviors are not the only behaviors that can be affected. The present study explored the potentiating effects of taste aversion conditioning on consummatory and locomotor behaviors. Twenty-seven male Holtzman rats were divided into one of three groups for conditioning and testing. During conditioning, Group WAT was given tap water, whereas Group SAC was given saccharin flavored water (SAC), and Group MIX was given saccharin and denatonium saccharide (DEN) flavored water. All three groups received LiCl to induce malaise. Results indicated the ability of DEN to potentiate the effects of SAC aversion conditioning. More

specifically, Group MIX displayed significant drinking decrements beyond those of Group SAC. Moreover, the presence of DEN on conditioning potentiated the decrement in locomotor behavior. Group MIX displayed significantly longer latencies and significantly more stops and retraces during testing than Group SAC. Results from the present experiment might be helpful in disrupting harmful consummatory behaviors in people with eating disorders or people who suffer medically induced eating problems.

EFFECTS OF TOXICOSIS MEDIATED POTENTIATION OF A TASTE/TASTE COMPOUND ON DRINKING AND LOCOMOTOR BEHAVIORS IN RATS

A Thesis

Presented to

The Division of Psychology and Special Education

EMPORIA STATE UNIVERSITY

In Partial Fulfillment

of the Requirements for the Degree

Master of Science

---

by

Roger Neil Edson

August 1997

Approved for the Division of
Psychology and Special Education

Approved for the Graduate Council

ACKNOWLEDGMENTS

First, I would like to thank Dr. Davis, Dr. Dungan, and Dr. Obiakor whose patience and hard work were necessary for the completion of this project. Understand, you have made my life a little easier.

Next, I would like to thank my friends and co-workers. They always encouraged, supported, and teased me to continue even when I wasn't sure what I wanted to do.

Finally, I would like to thank my family. No words can truly describe their devotion and support. Thank you for helping me during this thesis process. I will always be there for you as you have been there for me.

TABLE OF CONTENTS

LIST OF TABLES

Table Page

LIST OF FIGURES

CHAPTER 1

INTRODUCTION

Taste aversion conditioning has become a popular topic in psychology, particularly during the past three decades. Taste aversions occur naturally when a specific taste becomes associated with illness and that particular taste is later avoided (Garcia, Ervin, & Koelling, 1967). The paradigm for research (Garcia et al.) parallels the natural occurrence of learned taste aversions. An animal is presented with a specified, novel taste, usually a food or liquid. The animal is then subjected to reinforcement (e.g., drug induced illness, radiation). When the animal is again presented with the specific taste, consumption of the taste is very low, if present at all. This paradigm has been used for many taste aversion experiments and has proven to be a very effective tool.

Scientists have observed learned taste aversions since the early 1900s but scientific research did not begin until the 1950s (Chitty, 1954). Chitty studied how to eliminate rat and mouse infestations occurring in large cities. Most research on learned taste aversions has been done using rats as the experimental animals. There are several reasons for using rats. The first reason deals with a behavior called "bait-shyness" (Garcia & Koelling, 1966). Bait-shyness is a trait shared by many mammals after being poisoned. If the animal survives, it is less likely to consume that food later. In rats, bait-shyness is usually referred to as "neophobia," the fear of new things (Garcia et al., 1967). Strains of rats have been specifically bred to be experimental subjects and care for them is easy. Rats are also used because of their inability to regurgitate or "emesis" (Garcia, Ervin, & Koelling, 1966). Lack of emesis means a rat cannot expel foods that upset its stomach. Lacking

emesis, rats are more cautious when eating new foods because a new food that causes illness might cause death. The uniformity of "bait-shyness" responses across species indicates only small numbers of animals are necessary to conduct research (Garcia et al.).

The normal neophobia shown by rats is used as an advantage in taste aversion research. Because of their natural avoidance of novel flavors, rats learn taste aversions more quickly than do other animals. Because rats are cautious, it is important to present them with a taste that is palatable. A novel taste such as saccharin is often introduced in the drinking water (Garcia et al., 1967). The novel taste is used because conditioning a taste aversion with a familiar taste is difficult. Rudy, Rosenberg, and Sandell (1977) and Batsell and Best (1994) reported that a novel taste paired with illness produces a stronger aversion than a familiar taste. Rudy et al. also stated that when stimuli are presented and not followed by aversion conditioning, the stimuli acquire a "learned safety" property.

Limiting research by using rats as the test animals, parameters on stimuli and reinforcements are established. In a learning experiment, the choice of reinforcer depends on the objective of the experiment. In taste aversion, since the object of research is to find how such conditioning occurs, a gustatory reinforcer seems most appropriate (Garcia et al., 1967). Reinforcers such as electric shock seem to have very low effect in learned taste aversions compared to drug-induced illness or radiation treatment. Electric shock has low effect probably because peripheral "pain" is not usually associated with ingestion (Rusiniak et al., 1982). Many experiments use intraperitoneal injections of drugs, such as apomorphine (Garcia et al.) or lithium chloride (LiCl) (Palmerino, Rusiniak, & Garcia, 1980), to induce gastrointestinal illness. The general effect of a drug is to induce a feeling

of discomfort or malaise. For a learned taste aversion to occur, malaise must be somehow associated with ingestion.

Scientists have believed since Pavlov that an unconditioned stimulus (US) and conditioned stimulus (CS) must be closely paired to create associations. A time-delay factor in taste-aversion conditioning was studied by Garcia et al.(1966). Results indicated that in rats the association between consumption of a taste (CS) and feeling ill (US) is very strong and able to perseverate delays that other conditioning preparations cannot bridge. Even after long delays of 75 min, rats made an association between taste and illness. Thus, the association between the novel taste and subsequent illness is quite strong. Perhaps how "ill" the animal feels can also shed light on how associations are created. Three levels of dose and number of injections were used by Garcia et al.. Animals receiving the highest dose of cyclophosphamide, which induced illness after consumption of saccharin water, received only one injection. The lowest dose group received several injections. There was also an intermediate group. The single, high dose group showed the greatest decrement of saccharin water consumption during subsequent presentation. The low dose, multiple-injection group showed the least decrement, whereas the medium dose group showed intermediate results. The most effective method to create a learned taste aversion would appear to be a single high dose of drug to induce illness shortly after consumption of the particular taste. The single, high dose appears to produce a more acute but strong illness, whereas the smaller, multiple-dose procedure produces a chronic but less intense illness. Either way, taste was associated with malaise, and the animals

were less likely to consume the saccharin water. The next step would be to find out if the animal must actually consume something before it associates that event with malaise.

Although taste-aversion conditioning is easily facilitated, taste may not be the only stimulus present. Animals first must find the food and then consume it. To find food, animals also us distal cues such as olfactory, visual, and auditory stimuli (Rusiniak, Hankins, Garcia, & Brett, 1979). Distal cues form weak associations with gustatory responses compared to the associations formed by proximal cues (i.e., taste). However, some association may be formed between weak and strong cues because the weaker distal cues lead to the stronger proximal cues.

Some researchers have indicated that when a weak cue is presented coincident with a strong cue and followed by a US, the associative strength of the weak cue is blocked by the stronger cue (Palmerino et al., 1980). This result is termed "overshadowing" (e.g., Coburn, Garcia, Kiefer, & Rusiniak, 1984; Palmerino et al., 1980; Rusiniak et al., 1979; Rusiniak, Palmerino, Rice, Forthman, & Garcia, 1982). On the other hand, research also indicated that the associative strength of a weak cue can be increased by pairing the weak cue with a strong cue and following this compound CS with a US. This facilitative effect has been called "potentiation" (e.g., Davis, Best, Grover, Bailey, Freeman, & Mayleben, 1990; Coburn et al.; Palmerino et al.).

In order to find out more about the potentiaton of weak cues, Palmerino et al. (1980) conducted experiments that used odor (O), taste (T), or odor/taste (OT) presentations as the CS for three respective groups of rats. Aversions to O and T were tested separately. The overall pattern of results indicated that the aversion to the odor

was potentiated by the taste. Odor alone was not sufficient to produce a strong aversion. These odor potentiation results were supported by evidence from Coburn et al. (1984), Rusiniak, Palmerino, and Garcia (1982), and Rusiniak et al. (1979).

Rusiniak et al. (1979) suggested that perhaps second-order conditioning was involved in potentiation of odor by taste. Second-order conditioning refers to a secondary stimulus, such as odor, becoming progressively more aversive due to continued pairings with a primary stimulus which was previously paired with illness. This two-phase hypothesis means taste initially becomes aversive because of its association with illness; then odor becomes aversive by virtue of its pairing with taste. However, Rusiniak et al. also stated second-order conditioning is too weak to explain fast, one-trial learning. In order to secondarily associate odor with malaise, more time and additional pairings would be necessary. Results from Best, Batson, Meachum, Brown, and Ringer (1985) supported Rusiniak et al. and further stated second-order conditioning contributes little to learned taste aversions. If second-order conditioning is not valid, a faster method of association must take place for one trial learning to occur.

Data from Rusiniak et al. (1979) tended to support the "indexing hypothesis" proposed by Palmerino et al. (1980) to explain potentiation. Although nerves from taste and olfaction lead to different parts of the brain and are probably stored in memory in different ways, by combining odor and taste, odor is somehow remembered in the same manner as taste. However, olfaction has more interference because it is used more often than taste. Association interference is reduced by reducing time between pairings of taste and odor (Palmerino et al.).

Thus, odor apparently depends on the presence of taste in order to increase its aversiveness. Not only is odor a distal cue, it is also a multichannel sense (Palmerino et al., 1980; Davis, Wood, Huss, Hathway, & Roberts, 1995), whereas taste occurs in discrete consummatory bouts. Coburn et al. (1984) conducted research to find the temporal contiguity necessary for taste to potentiate odor. Odor was presented to different animals from 10 min before presentation of taste to 10 min after presentation of taste. After presentation of taste, poisoning was induced. Results showed potentiation of odor by taste from 10 min before taste presentation up to when odor and taste were presented together. Potentiation did not occur when taste was presented before odor. Palmerino et al. also found this asymmetrical relationship between odor and taste. This asymmetrical relationship indicates that odor is only associated with malaise when presented before consumption. Apparently, because odor is used to find food, it only has pre-consumption effects.

If temporal contiguity is a relevant variable in the elicitation of potentiation perhaps spatial contiguity is also necessary. Rusiniak et al. (1982) performed research that compared the effects of an odorant substance placed directly in the water to the effects of the odorant when it was placed on filter paper that surrounded the drinking spout. Both conditions produced odor potentiation; however, odor in the water produced greater potentiation than did odor near the taste (i.e., on the filter paper). Apparently spatial contiguity is also involved in potentiation.

To this point, potentiating effects have been studied via the association of factors involved in consumption. That is to say, odor and taste are associated with each other and

each can be associated with illness. Coburn et al. (1984) suggested the effects of potentiation may hold for activities other than feeding. Rusiniak, Palmerino, and Garcia (1982) experimented with the potentiating effects of LiCl poisoning and footshocks. A compound CS consisting of odor and taste is expected to be dependent upon a delayed visceral effect, such as poisoning, to create a potentiation effect. Conversely, immediate footshock does not alter food palatability so footshock should have no potentiating effects on odor. Results from the footshock experiments differed from the LiCl conditioning. Odor on a disk was a more effective CS than odor in the water for footshock. Taste in the water apparently disrupted odor-shock conditioning. Rusiniak, Palmerino, and Garcia (1982) concluded shock was predicted by location of odor whereas illness tends to be flavor specific. Changing location of the odor had significant results on shock conditioning, whereas location of odor had minimal effects on LiCl conditioning.

Bouton, Dunlap, and Swartzentruber (1987) agreed with the reliability of taste to potentiate odor in aversive learning conditions and further suggested the relative associations between two tastes had not been studied and that taste-nontaste potentiation may not be unique. Varying the concentrations of the target and potentiating tastes, Bouton et al. attempted to discern if one taste could potentiate another taste. In one experiment, three concentrations of sodium chloride (NaCl) were conditioned alone or in compound with a strong saccharin taste. Results indicated that aversion conditioning generally improved with greater NaCl concentrations. Even the weakest NaCl solution was potentiated. To study this relationship further, Bouton et al. conducted another experiment using three concentrations of the strong or potentiating saccharin (SAC)

flavor. The weakest NaCl solution from the first experiment was the target taste. Results showed the NaCl taste was potentiated only with the intermediate concentration of SAC. These results suggest the probability of an optimal combination of concentrations of target and potentiating tastes. These findings are supported by other researchers (e.g., Best, Davis, & Grover, 1989). Bouton et al. mentioned the weak conditionability of the target may be the key prerequisite for potentiation rather than having the status of a distal cue.

Davis, Best, and Grover (1988) also studied stimulus interactions by compounding two tastes and then administering toxicosis conditioning. Davis et al. chose SAC and saline solutions (SAL) as target tastes and denatonium saccharide (DEN) as the potentiating taste. DEN is a colorless, odorless, bitter substance readily consumed by rats in concentrations of 1 part per 10,000 parts water. The single or compound conditioning stimulus (CS) was followed by LiCl injections. Results indicated potentiating effects of DEN on both SAC and SAL. Research by Davis, Freeman, and Nation (1993) and Durlach and Rescorla (1980) also agreed. When two tastes are presented in compound, one taste can potentiate the other.

The research discussed to this point has concerned itself primarily with the potentiation of ingestional factors. Best, Brown, and Sowell (1984) investigated the association between noningestive, diffuse environmental stimuli and potentiation. Best et al. injected LiCl immediately following containment in either an operant chamber or a mouse breeding cage lined with plastic. The rats had either flavored or unflavored water available when placed in the operant chamber or mouse breeding cage. Best et al. found that environmental stimuli interfered with ingestional behavior following conditioning of

the flavored water. This ability of the environment to interfere with ingestional behavior would indicate taste stimuli are capable of potentiating noningestional stimuli.

Klein, Freda, and Mikulka (1985) conducted experiments to examine if environmental stimuli could be conditioned to induce taste aversions. A shuttlebox was divided into equal-sized black or white colored compartments with sucrose solution in one or both compartments or unflavored water in one or both sections. The animals were divided into one of four groups. One group of animals was poisoned in the black compartment, whereas a second group was poisoned in the white compartment. Results showed time spent in the poisoned compartment was less, when sucrose was part of the solution, than in the unpoisoned compartment when sucrose was not part of the solution. These results indicate rats are capable of distinguishing features of the environment and are capable of associating environmental stimuli with malaise. Potentiated aversions of environmental stimuli did not occur when sucrose solution was placed in both black and white compartments.

Best et al. (1985) conducted experiments to establish parameters on taste potentiated environmental stimuli. These researchers showed a novel taste in a distinct environment produces stronger aversions than use of a familiar taste. Rudy et al. (1977) found preexposure to taste during aversion conditioning is similar to preexposure to the environmental stimuli. Preexposure interference of a familiar taste in the potentiation of a novel environment was also noted by Best and Meachum (1986). Just as novel ingestional stimuli are more effective in creating taste aversions; novel environmental stimuli are more likely to be potentiated.

Since novel stimuli are being associated with malaise during potentiation, perhaps animals generalize to associate conditioned stimuli with illness. However, experiments from Best et al. (1985), Davis et al. (1988), Westbrook, Homewood, Horn, and Clarke (1983), and Bowman, Batsell, and Best (1992) showed potentiation was not due to generalized aversions between conditioning stimuli and test stimuli or conditioning stimuli and environment. Instead, the rats distinguished between elements of the test solution and perhaps even elements of the environment.

Evidence so far has shown the ability of tastes to potentiate environmental stimuli. Galef and Osborne (1978) described how predators must distinguish certain distal cues such as odor, visual, auditory, or behavior to avoid poisoning. Telereceptive (distal) cues other than odor tend to require many trials for rats to associate them with illness. Rats can probably distinguish other patterns but under different conditions than are usually present in laboratory studies. In a more natural environment rats would distinguish features of the environment by smell, not only by visual cues. Galef and Osborne subjected rats to toxicosis after ingestion of a visually distinctive object. Results showed rats were able to associate visual cues with toxicosis if illness followed quickly after ingestion.

The parameters of taste potentiation have now been broadened to include not only odor and taste but also environmental stimuli. If it is possible to potentiate odor and other noningestional stimuli, such as features of the environment, perhaps it is also possible to measure a noningestional behavior, such as locomotion, to study the effects of potentiation. Locomotion would be a measure of how long an animal takes to get reinforcement.

Best et al. (1989) studied the potentiation of locomotor behavior. Rats that had learned to run a straight alley for water during training were given water or a SAC solution paired with a peppermint odor during conditioning. All rats were then injected with LiCl. Latencies and number of stops were recorded when the animal was again placed in the straight alley and allowed to run to an empty goal cup with odor present. The results supported the potentiation research by showing the odor/taste group had longer latencies in the straight alley than their control (odor alone) counterparts. The odor/taste group also displayed significantly more stopping than the odor alone group. This research would indicate potentiation of locomotor behavior is possible.

The present study was designed to explore the effects of a potentiated taste aversion and potentiated environmental stimuli on locomotor behavior. Potentiation of taste by another taste was measured using lick data collected during consumption of fluid in the goalbox on each trial. Rats were divided into three groups. The first group (Group WAT) was given tap water during conditioning and served as the control group. Group SAC was given saccharin flavored water during conditioning and was employed to establish a baseline for the effects of a simple saccharin taste aversion. Group MIX was given both saccharin and denatonium saccharide flavored water during conditioning . Group MIX was predicted to show both potentiated taste aversion and locomotor decrements. Group SAC was expected to have drinking decrements lower than Group WAT. Conversely, Group WAT should have had the fastest times in the straight alley whereas Group MIX should have shown the longest times in the straight alley. Group SAC was expected to have latencies somewhere between the other groups.

In addition to the latency recorded on each trial, the number of stops and retraces made by each animal on each trial also were recorded. The results of the stop and retrace measures were expected to parallel those predicted for the latency measure.

CHAPTER 2

METHOD

Participants

Twenty-seven male, albino rats were obtained from the Holtzman Company, Madison, Wisconsin. The animals were approximately 70 days old when they arrived at Emporia State University. Upon arrival the rats were randomly assigned to individual, hanging, wire-mesh cages in the animal vivarium in the psychology laboratory.

Apparatus

The test apparatus consisted of a single straight runway approximately 11.40 cm wide and 12.70 cm high. The runway consisted of a gray start box 38.10 cm long, a black run section 91.44 cm long, and a black goal box 30.48 cm long. Masonite guillotine doors separated the start box and goal box from the run section. Three Lafayette digital timers (Model 54030) recorded start, run, and goal latencies on each trial. The start timer was activated by a microswitch, whereas three photoelectric beams were used to sequentially start and stop the remaining two timers. The photoelectric beams were located 15.20 cm, 92.40 cm, and 116.80 cm beyond the start door. The goal box was fitted with a recessed, plastic receptacle for insertion of a drinking tube. The top of the runway was covered with hinged wire-mesh tops. A Lafayette lickometer (Model 58008) in conjunction with a rotary counter (Lafayette Model 5822) recorded the number of licks made by each subject in the goal box.

Procedure

Upon arrival at the laboratory the animals were divided into one of three groups: water (WAT), saccharin (SAC), or saccharin-denatonium (MIX). The week following arrival constituted a rest period during which the animals were allowed access to food and water on an ad libitum basis. A water-deprivation regime, which allowed each animal 15-minute exposure to water daily, was begun at the conclusion of the week-long rest period. A four-day Pretraining phase was begun two days after the inception of the water-deprivation regimen. During each of the four days of Pretraining each animal was handled one to two minutes each day. On Pretraining Days 3 and 4 all animals were given an individual 5-minute exploration period in the runway with water available in the goal box. During the exploration periods the doors were raised and all electrical equipment was functional.

A Training phase was begun 24 hours following the last day of Pretraining. During the first 11 days of Training all animals received 2 trials per day; 1 trial was administered to each animal on Days 12 and 13 in order to make the procedure more comparable to the conditions employed during the Conditioning and Testing phase. The order for running animals within each group was randomized daily, whereas the order for running groups was cyclic from day to day (e.g., WAT-SAC-MIX; SAC-MIX-WAT, and so forth). On Days 1 through 11 all animals within a designated group received Trial 1 before Trial 2 was administered. To run a trial the designated animal was placed in the start box. Following a three-second confinement, the doors were raised and the animal was allowed to traverse the runway. The start door was lowered when the first

photoelectric beam was broken. The goal door was lowered when the final beam in the goal box was broken. Each animal was confined in the goal box for 30 seconds after breaking the final beam. Water was available in the goal box during Training. The entire runway was swabbed with a damp sponge and air dried at the completion of a trial by all animals in a designated group. Conditioning was conducted 24 hours following the completion of Training and involved the completion of one runway Trial by each animal. At the completion of the runway trial each animal received an intraperitoneal injection of lithium chloride (.30 M, .12% body weight). The groups differed with regard to the fluid that was available in the goal box during Conditioning. Group WAT had access to plain tap water, while Group SAC had access to a saccharin solution (.15% w/v). Group MIX had access to a mixture of .15% saccharin and denatonium saccharide (1 part per 20,000 parts water). A series of daily saccharin tests was begun 24 hours after Conditioning. During this 7-day Test phase a single, daily runway trial was administered in the same manner as during Training and Conditioning. All animals had access to saccharin during these Test trials. Start, run, and goal latencies, as well as stops, retraces, and the number of licks, were recorded on each runway trial for each animal during all phases of the experiment.

CHAPTER 3

RESULTS

Training

Analysis of variance performed on the latency data recorded on the final two days of training failed to yield reliable between-groups effects in the start, $F(2, 24) = .35$, $p > .25$, run, $F(2, 24) = .62$, $p > .25$, and goal, $F(2, 24) = 1.37$, $p > .25$, measures. Likewise, reliable between-groups effects did not exist in the stops, $F(2, 24) = 2.73$, $p = .08$, and licks, $F(2, 24) = 2.84$, $p = .07$, measures. Due to the virtual absence of retraces in all groups on the final two days of Training, these data were not subjected to analysis. Based upon the lack of significant between-groups effects, it can be concluded that the groups were equivalent at the end of training. Figures 1 through 3 display the start, run, and goal latencies, respectively, for the last two days of Training, Conditioning, and the Saccharin Test Phase. Figure 4 displays the mean number of licks made by the three groups on the last two days of Training, Conditioning, and Saccharin Test Phase.

Conditioning

As with the training data, analysis of the start, $F(2, 24) = .63$, $p > .25$, run, $F(2, 24) = 2.94$, $p > .05$, and goal, $F(2, 24) = .96$, $p > .25$, measures failed to yield significant between-groups differences. Similarly, the number of stops made on the conditioning trial did not differ among the groups, $F(2, 24) = .66$, $p > .25$. (As in the case of Training, the low number of retraces displayed by all groups during conditioning made analysis of these data unadvisable.) Thus, the groups were deemed to be equivalent on these measures on the day of conditioning. Unlike the other measures, analysis of the lick data yielded

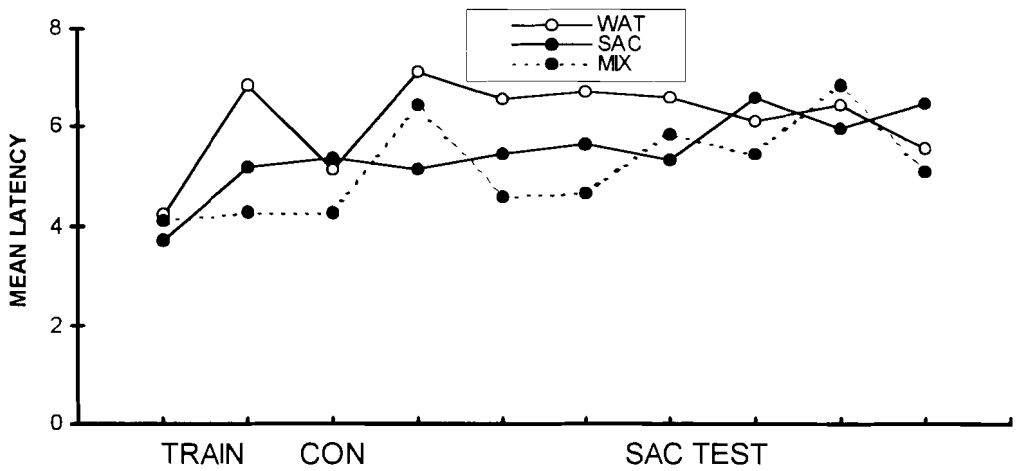

Figure 1. Start Latency (Seconds) for Groups WAT, SAC, and MIX on the Last 2 Days of Training (TRAIN), Conditioning (CON), and During the Saccharin Test Phase (SAC TEST).

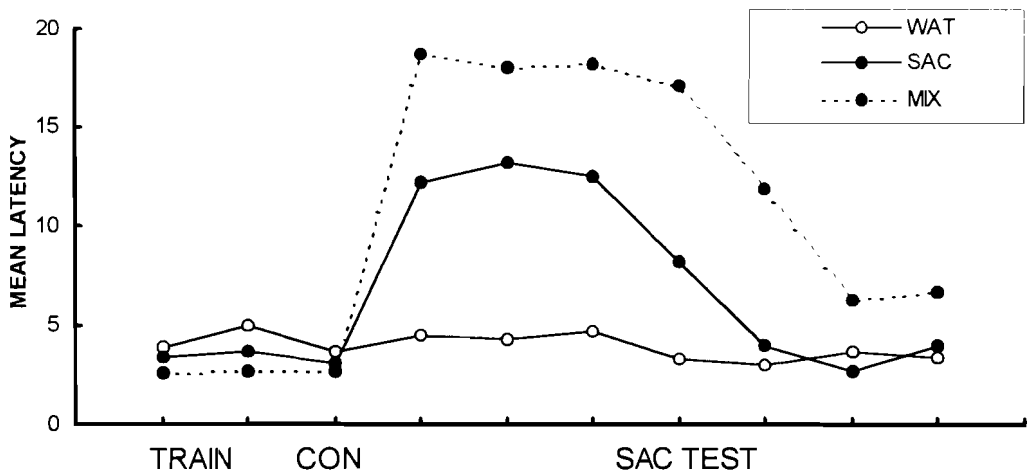

Figure 2. Mean Run Latency (Seconds) for Groups WAT, SAC, and MIX on the Last 2 Days of Training (TRAIN), Conditioning (CON), and During the Saccharin Test Phase (SAC TEST).

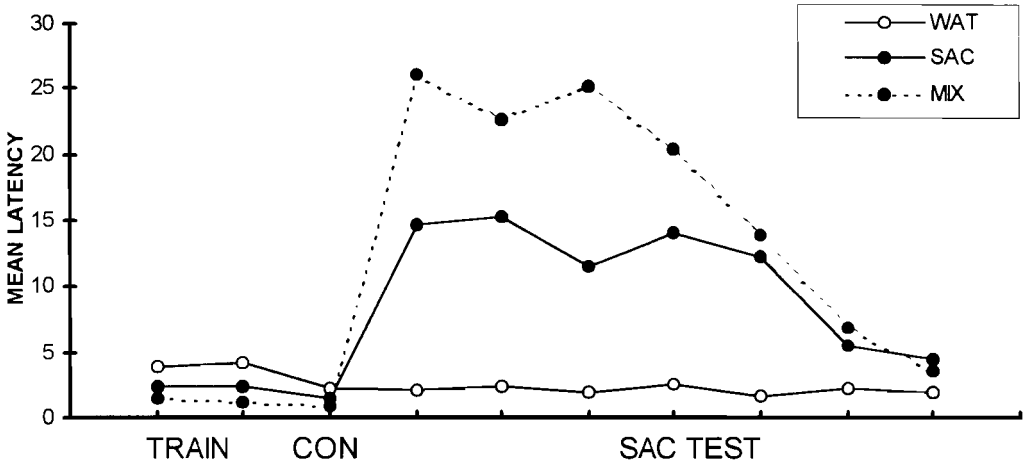

Figure 3. Mean Goal Latency (Seconds) for Groups WAT, SAC, and MIX on the Last 2 Days of Training (TRAIN), Conditioning (CON), and During the Saccharin Test Phase (SAC TEST).

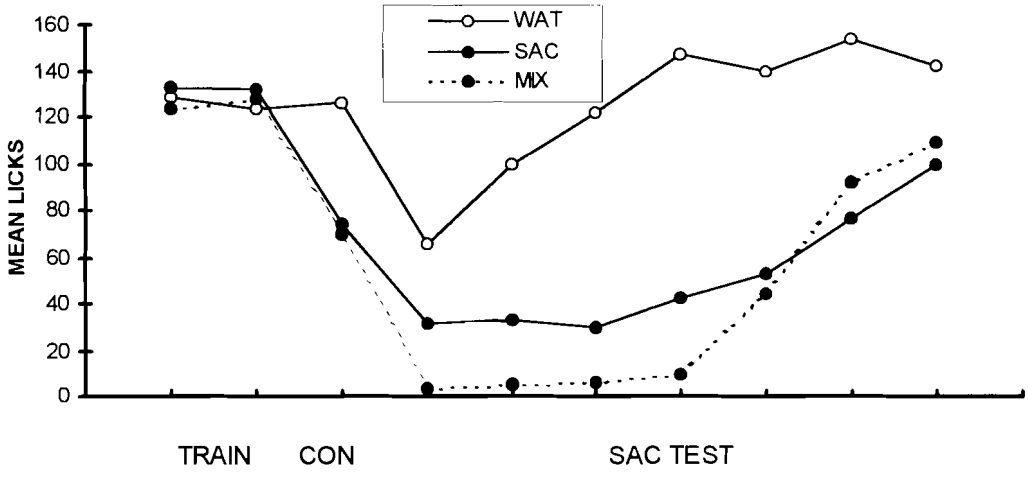

Figure 4. Mean Licks for Groups WAT, SAC, and MIX on the Last 2 Days of Training (TRAIN), Conditioning (CON), and During the Saccharin Test Phase (SAC TEST).

a significant groups effect, $F(2, 24) = 4.87$, $p = .016$. Subsequent Newman-Keuls tests indicated that Group WAT made significantly ($p < .05$) more licks during conditioning than did Groups SAC and MIX, which did not differ reliably from each other. These results indicate that a significant neophobic response was shown by Groups SAC and MIX during Conditioning.

Saccharin Test Phase

Start. Analysis of the start-measure performance failed to yield significance for the groups, $F(2, 24) = 1.26$, $p = .30$, days, $F(6, 144) = 1.66$, $p = .13$, and groups by days, $F(12, 144) = .78$, $p > .25$, effects.

Run. The groups, $F(2, 24) = 4.30$, $p < .05$, days, $F(6, 144) = 3.23$, $p < .01$, and groups x days, $F(12, 144) = 2.79$, $p < .01$, effects achieved significance in the run-measure analysis. The Newman-Keuls procedure, used to probe significant interactions, indicated Group MIX ran significantly ($p < .05$) more slowly than Groups WAT and SAC on Days 1-5. In turn, Group SAC ran significantly ($p < .05$) more slowly than Group WAT on Days 1-3.

Goal. The groups, $F(2, 24) = 4.67$, $p < .05$, days, $F(6, 144) = 3.59$, $p < .01$, and groups x days, $F(12, 144) = 2.83$, $p < .01$, effects achieved significance in the goal-measure analysis. Subsequent Newman-Keuls tests indicated Group MIX approached the goal significantly ($p < .05$) more slowly than: (a) Group WAT on Days 1-5, and (b) Group SAC on Days 1-4. Group SAC approached the goal significantly ($p < .05$) more slowly than Group WAT on Days 1-5.

Licks. Analysis of the lick data yielded significance for the groups, $F(2, 24) = 6.01$, $p < .01$, days, $F(6, 144) = 3.17$, $p < .01$, and groups x days, $F(12, 144) = 1.92$, $p < .05$, effects. Subsequent Newman-Keuls tests indicated Group MIX made significantly ($p < .05$) fewer licks than: (a) Group WAT on all days, and (b) Group SAC on Days 1 through 4. Group SAC also made significantly ($p < .05$) fewer licks than Group WAT on all days of the Saccharin Test phase. A comparison of the number of licks made by Group WAT on Conditioning and the first day of the Saccharin Test phase yielded a reliable difference, $t(8) = 4.31$, $p < .01$. Thus, the introduction of novel saccharin for these subjects during Testing produced a significant neophobic response.

Stops. The groups, $F(2, 24) = 4.55$, $p < .05$, days, $F(6, 144) = 3.11$, $p < .01$, and groups x days, $F(12, 144) = 2.18$, $p < .05$, effects were significant in the stops analysis. Subsequent Newman-Keuls tests indicated Groups SAC and MIX made significantly ($p < .05$) more stops than Group WAT on Days 1 through 4. In turn, Group MIX made significantly ($p < .05$) more stops than Group SAC on Days 1 through 4. Group SAC made significantly ($p < .05$) more stops than the other two groups on Day 5. Table 1 displays the number of stops made by the three groups during the Saccharin Test.

Retraces. Analysis of the retrace data yielded significance for the groups, $F(2, 24) = 3.49$, $p < .05$, days, $F(6, 144) = 2.45$, $p < .05$, and groups x days, $F(12, 144) = 1.87$, $p < .05$, effects. Subsequent Newman-Keuls tests indicated Groups SAC and MIX made significantly ($p < .05$) more retraces than Group WAT on Days 1 through 3. Table 2 displays the mean number of retraces made by the three groups during the Saccharin Test Phase.

Table 1

Means and Standard Deviations for Stops Made by Groups WAT, SAC, and MIX During the Saccharin Test Phase

	Days						
	1	2	3	4	5	6	7
Groups							
WAT	1.00	1.33	1.22	1.44	1.22	1.11	.05
	(1.12)	(1.12)	(.83)	(.88)	(1.09)	(1.05)	(.88)
SAC	2.67	2.89	2.56	2.89	2.56	1.56	1.56
	(1.12)	(.78)	(1.24)	(1.05)	(.53)	(1.13)	(1.24)
MIX	3.67	3.67	3.67	3.78	1.89	1.00	1.11
	(.71)	(.50)	(1.00)	(1.20)	(1.83)	(1.32)	(1.36)

Table 2

Means and Standard Deviations for Retraces Made by Groups WAT, SAC, and MIX During the Saccharin Test Phase

	Days						
	1	2	3	4	5	6	7
Groups							
WAT	.22	.22	.22	.33	.11	.11	.11
	(.44)	(.44)	(.44)	(.71)	(.33)	(.33)	(.33)
SAC	1.44	1.33	1.78	1.33	.33	.22	.22
	(1.01)	(1.00)	(.97)	(.71)	(.71)	(.44)	(.44)
MIX	2.33	2.22	2.33	1.44	.56	.44	.00
	(.71)	(.44)	(.71)	(.73)	(1.33)	(1.01)	(0.00)

CHAPTER 4

DISCUSSION

The present study was designed to explore the effects of potentiated taste and environmental stimuli on consummatory and locomotor behaviors. Following the establishment of group comparability at the end of training, Groups SAC and MIX predictably displayed a significant neophobic response when the novel flavors were introduced during Conditioning. Likewise, Group WAT displayed the neophobic response when novel saccharin was introduced on the first day of Testing. However, unlike groups SAC and MIX, drinking decrements of Group WAT quickly attenuated. Fast attenuation of neophobic respnses concurs with the theory by Rudy et al. (1977) and Batsell and Best (1994) of a "learned safety" association when a US is not followed by aversion conditioning.

The now familiar pattern typically shown for potentiated taste aversions also was displayed in the lick data. Group SAC showed evidence of a simple taste aversion whereas Group MIX indicated evidence of a potentiated taste aversion. Potentiated effects of Group MIX were similar to Davis et al. (1988) and Davis et al. (1993). More specifically, Group MIX made significantly fewer consummatory responses than Group SAC on Test Days 1 through 4. Potentiation was indicated when DEN was not part of the testing solution. Possible confusion of conditioning and testing solutions in Group MIX is refuted by Best et al. (1985), Westbrook et al. (1983), and Bowman et al. (1992). The rats in the present study were apparently able to distinguish elements of the conditioning and testing solutions. In addition, the current results are supported by the

within-compound association theory of Davis et al. (1988). When multiple stimuli are presented together, several associations can be made in a short period of time. Results from the current experiment also support evidence against the second-order conditioning theory proposed by Rusiniak et al. (1979). Within-compound associations would account for the fast learning and significant drinking decrements of Group MIX. Reflecting that a strong taste-aversion was established, groups SAC and MIX made significantly fewer consummatory responses than Group WAT on all Test Days.

Because the disruptive patterns evidenced in the consummatory data were also revealed in locomotor responding, it is arguable that the stimulus attributes of the runway also acquired aversive properties as a result of the conditioning episode. Thus, it can be seen that groups SAC and MIX had longer run- and goal-approach latencies than Group WAT. In turn, the finding that the run- and goal-approach latencies of Group MIX were significantly slower than those of Group SAC on several days during the Test phase indicate the presence of DEN potentiated the environmental aversion shown by Group MIX. Best et al. (1985) and Klein et al. (1985) found environmental stimuli could be conditioned to have a potentiated aversion while Best et al. (1989) found locomotor behavior suppressed when the environment was paired with taste aversion conditioning. Results from the current experiment supports the theory that environmental stimuli can be associated with taste aversion conditioning in addition to effecting a non-consummatory behavior, locomotion. Furthermore, Best et al. (1989) also found generalization from a consummatory behavior to a locomotor behavior was unlikely. Past and present data indicate cross-behavioral potentiation is possible. Moreover, a comparison of Figures 1

through 3 shows the environmental conditioning was strongest in the goal-approach latencies, somewhat attenuated in the run latencies, and not a significant determinant of performance in the start latencies. This pattern of results indicates conditioning was strongest at the point of receipt of the novel flavor and became progressively weaker as the distance from that point increased.

Similar disruptions in locomotor behavior were also displayed in the stop measure. Thus, Table 1 shows groups MIX and SAC made more stops than Group WAT. In turn, potentiation effects were evidenced by the fact that Group MIX stopped more frequently than Group SAC on Days 1 through 4.

The retrace data also reflect the influence of taste-aversion conditioning. Both groups MIX and SAC retraced more often than Group WAT (see Table 2). However, because retracing is a relatively rare behavior this measure apparently was not sufficiently sensitive to detect potentiation effects.

The pattern of results also indicates potentiation of weak, or distal, cues has backward extending effects. Associations made in a retroactive manner would explain why Coburn et al. (1984) found odor had to be presented before or at the same time as taste for odor-potentiation to occur. Another pattern repeated in the current experiment was the attenuation of the drinking decrements and locomotor latencies of groups SAC and MIX.

In summary, the present study corroborates previous research concerning the ability of a taste to potentiate another taste, disrupting consummatory behavior. In addition, the present study also shows the ability of potentiated environmental stimuli to

disrupt locomotor behavior. A limiting factor of the current experiment was the animals were familiar with the environment before conditioning. Pre-exposure interference of the environment was not addressed although Best and Meachum (1986) found such exposure to have little effect concerning taste aversion conditioning.

Although research of the current experiment will most likely not be used to eliminate rat or mouse problems, it does have possible effects in the human world. Using animals, such as rats, to determine how associations between consummatory behaviors and taste aversions occur and how to disrupt such associations might be of help to people with eating disorders. Trying to determine how to disrupt or change environmental associations might make possible the opportunity for individuals with bulimia or anorexia to lead normal lives. Being able to disrupt associations between food and illness would make the lives of chemotherapy patients more bearable. Finding the limits of potentiating effects might help in other areas such as education and maybe even more efficiency in the work place.

REFERENCES

Batsell, W. R., & Best, M. R. (1994). The role of US novelty in retention interval effects in single-element taste-aversion learning. Animal Learning and Behavior, 22, 332-340.

Best, M. R., Brown, E. R., & Sowell, M. K. (1984). Taste-mediated potentiation of noningestional stimuli in rats. Learning and Motivation, 15, 244-258.

Best, M. R., Batson, J. D., Meachum, C. L., Brown, E. R., & Ringer, M. (1985). Characteristics of taste-mediated environmental potentiation in rats. Learning and Motivation, 16, 190-209.

Best, M. R., Davis, S. F., & Grover, C. A. (1989). Straight alley extinction performance is disrupted by taste-aversion conditioning. Learning and Motivation, 20, 358-372.

Best, M. R., & Meachum, C. L. (1986). The effects of stimulus preexposure on taste-mediated environmental conditioning: Potentiation and overshadowing. Animal Learning & Behavior, 14, 1-5.

Bouton, M. E., Dunlap, C. M., & Swartzentruber, D. (1987). Potentiation of taste by another taste during compound aversion learning. Animal Learning & Behavior, 15, 433-438.

Bowman, M. T., Batsell, W.R., & Best, M. R. (1992). Evidence that stimulus generalization does not determine taste-mediated odor potentiation. Bulletin of the Psychonomic Society, 30, 241-243.

Chitty, D. (1954). Control of rats and mice. London: Oxford University Press.

Coburn, K.L., Garcia, J., Kiefer, S. W., & Rusiniak, K. W. (1984). Taste potentiation of poisoned odor by temporal contiguity. Behavioral Neuroscience, 98, 813-819.

Davis, S. F., Best, M. R., & Grover, C. A. (1988). Toxicosis-mediated potentiation in a taste/taste compound: Evidence for within-compound associations. Learning and Motivation, 19, 183-205.

Davis, S. F., Best, M. R., Grover, C. A., Bailey, S. A., Freeman, B. L., & Mayleben, M. A. (1990). The effects of taste extinction on ingestional potentiation in weanling rats. Animal Learning & Behavior, 18, 444-452.

Davis, S. F., Freeman, B. L., & Nation, J. R. (1993). The effects of chronic lead exposure on taste-aversion conditioning. Psychological Record, 43, 205-214.

Davis, S. F., Wood, K. D., Huss, M. T., Hathway, J. E., & Roberts, S. L. (1995). Odor-mediated performance is affected by cadmium injestion. Psychological Record, 45, 389-403.

Durlach, P. J., & Rescorla, R. A. (1980). Potentiation rather than overshadowing in flavor-aversion learning: An analysis in terms of within-compound associations. Journal of Experimental Psychology: Animal Behavior Processes, 6, 175-187.

Galef, B. G., & Osborne, B. (1978). Novel taste facilitation of the association of visual cues with toxicosis in rats. Journal of Comparative and Physiological Psychology, 92, 907-916.

Garcia, J., Ervin, F. R., & Koelling, R. A. (1967). Bait-shyness: A test for toxicity with N=2. Psychonomic Science, 7, 245-246.

Garcia, J., Ervin, F. R., & Koelling, R. A. (1966). Learning with prolonged delay of reinforcement. Psychonomic Science, 5, 121-122.

Garcia, J., & Koelling, R. A. (1966). Relation of cue to consequence in avoidance learning. Psychonomic Science, 4, 123-124.

Klein, S. B., Freda, J. S., & Mikulka, P. J. (1985). The influence of a taste cue on an environmental aversion: Potentiation or overshadowing. The Psychological Record, 35, 101-112.

Palmerino, C. C., Rusiniak, K. W., & Garcia, J. (1980). Flavor illness aversions: The peculiar roles of odor and taste in memory for poison. Science, 208, 753-755.

Rudy, J. W., Rosenberg, L., & Sandell, J. H. (1977). Disruption of a taste familiarity effect by novel exteroception stimulation. Journal of Experimental Psychology, 3, 26-36.

Rusiniak, K. W., Hankins, W. G., Garcia, J., & Brett, L. P. (1979). Flavor-illness aversions: Potentiation of odor by taste in rats. Behavioral and Neural Biology, 25, 1-17.

Rusiniak, K. W., Palmerino, C. C., & Garcia, J. (1982). Potentiation of odor by taste in rats: Tests of some nonassociative factors. Journal of Comparative and Physiological Psychology, 96, 775-780.

Rusiniak, K. W., Palmerino, C. C., Rice, A. G., Forthman, D. L., & Garcia, J. (1982). Flavor-illness aversions: Potentiation of odor by taste with toxin but not shock in rats. Journal of Comparative and Physiological Psychology, 96, 527-539.

Westbrook, R. F., Homewood, J., Horn, K., & Clarke, J. C. (1983). Flavour-odour compound conditioning: Odour-potentiation and flavour attenuation. Quarterly Journal of Experimental Psychology, 35B, 13-33.

I, Roger Neil Edson, hereby submit this thesis/report to Emporia State University as partial fulfillment of the requirements for an advanced degree. I agree that the Library of the University may make it available to use in accordance with its regulations governing materials of this type. I further agree that quoting, photocopying, or other reproduction of this document is allowed for private study, scholarship (including teaching) and research purposes of a nonprofit nature. No copying which involves potential financial gain will be allowed without written permission of the author.

Roger N. Edson
Signature of Author

7/23/97
Date

Effects of Toxicosis Mediated Potentiation of a Taste/Taste Compound on Drinking and Locomotor Behaviors in Rats
Title of Thesis/Research Project

Doug Cooper
Signature of Graduate Office Staff

July 30, 1997
Date Received