Conditioned inhibition of rotation-induced taste aversion

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In three experiments, we investigated the existence of conditioned inhibition of body rotation-induced taste aversion. Rats were given conditioned inhibition training in which the taste of saccharin was always followed by rotations, but the taste of vanilla was not. Flavor-preference tests, retardation-of-acquisition tests, and summation tests of inhibition indicated that the vanilla stimulus had acquired conditioned inhibitory properties. These findings could not be interpreted as functions of either initial solution preferences or simple nonassociative effects of flavor preexposure. They lend support to a theory that views learning mechanisms as being central to the phenomenon of motion sickness, and suggest that inhibitors might be effectively employed to ameliorate its symptoms.

According to the sensory-conflict, or neural mismatch, theory of motion sickness (Reason & Brand, 1975), situations that produce motion sickness are characterized by conditions of "sensory conflict." Sensory conflict is usually taken to mean a compensatory reflex-induced discontinuity or asynchrony among visual, proprioceptive, and somatosensory inputs brought about by conditions of excessive vestibular stimulation (Kohl, 1983). That is, motion information signaled by the eyes and proprioceptors is at variance (i.e., in conflict) with the kinds of inputs that are expected on the basis of past experience. The central component of this condition is vomiting, and the most frequently reported secondary symptoms are pallor, sweating, and nausea.

Treisman (1977) has suggested that these primary and secondary components of motion sickness, involving as they do neural pathways among visual, vestibular, and vomiting centers, would be hard to imagine as having arisen by chance in the absence of some benefit to organisms that possess them. He speculated that all species must have been under continuous evolutionary pressure to develop mechanisms to protect against ingesting and ingested toxic materials. Such protective mechanisms are rejection by taste and rejection through emesis (i.e., vomiting) provoked by the effects of toxins on chemoreceptors in both the stomach lining and the *area postrema* in the lower portion of the brain (Carlson, 1981). This reflexive emetic response is clearly advantageous to animals that possess it if its purpose is to rid the digestive system of a poison before it can permanently damage neural tissue. Treisman further suggested that the subjectively un-

pleasant aspects of nausea and malaise may also function to contribute to a process of aversive conditioning. This conditioned aversion would provide a protective mechanism against ingesting that toxin in the future. A third such mechanism is concerned with the effects of neurotoxins on systems involved in responding to motion. Treisman claimed that ingestion of some toxins could produce perceptual distortions or sensory conflicts very similar to the effects of excessive vestibular stimulation. Emesis in response to provocative vestibular stimulation (which mimics the effects of some toxins) could, according to Treisman, be an accidental by-product (and cost) of possessing a detection system for ingested toxins.

The literature on conditioned taste aversion (CTA) is replete with evidence that gastrointestinal upset (an unconditioned response, or UR) occurring up to several hours after ingestion of a toxin (an unconditioned stimulus, or US) can result in an aversion to a taste (a conditioned stimulus, or CS) associated with that upset. If motion sickness is, as Treisman (1977) suggested, related to toxin-based aversions, then similar outcomes in learning experiments employing toxic USs and those employing vestibular stimulation could be expected. Braun and McIntosh (1973), Green and Rachlin (1973), and Haroutunian and Riccio (1975) have successfully demonstrated that rats, which are incapable of emesis, will learn to avoid a taste associated with the illness produced by whole-body rotation. Ossenkopp and Tu (1984) have argued that this vestibular-induced CTA, even in nonemetic species, provides evidence not only for the generality of the motion-sickness phenomenon across species, but also for the fact that a common mechanism may be involved in its production.

The present effort was a further attempt to study vestibular-induced CTA. In addition, however, we sought to explore the possible effects of another Pavlovian process in CTA, that of conditioned inhibition. A conditioned inhibitor is a stimulus which, through learning,

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comes to signal the absence of a US. Such inhibitors can prevent or reduce the magnitude of a conditioned response (CR). Best (1975) and Taub and Revusky (1975) were the first investigators to demonstrate this phenomenon in toxin-induced CTA. Successful demonstration of conditioned inhibition with vestibular-induced illness would not only indicate the generality of the phenomenon within the CTA framework, but would also provide indirect support for Treisman's notion relating toxin effects to those of provocative vestibular stimulation. In addition, such a demonstration would suggest that a conditioned inhibitor might be employed to actively inhibit the maladaptive reflex mechanisms responsible for motion sickness by preventing or reducing their conditioned aspects.

EXPERIMENT 1

Rescorla (1969) claimed that there are two principal tests necessary for proper demonstration of conditioned inhibition: the retardation-of-acquisition test and the summation test. In the retardation test, the putative inhibitor is paired with the US and the rate of acquisition of an excitatory CR is compared with the rate of acquisition in a group that has previously been given some control treatment. In a summation, or compound stimulus, test of inhibition, the putative inhibitor is presented in compound with a known excitator, and the response to this compound is compared with the response to the excitator alone. In the present study, we employed both tests of conditioned inhibition. Best (1975) demonstrated that a taste that had come to signal the absence of toxin-induced illness would be preferred over a taste that had not acquired such conditioned illness-inhibitory properties. This preference definition of conditioned inhibition was also employed in the present study.

In Experiment 1, we attempted to (1) replicate the basic demonstration of conditioned aversion to a taste associated with rotation (e.g., Haroutunian & Riccio, 1975), (2) demonstrate conditioned inhibition using Best's (1975) preference definition, (3) demonstrate inhibition using summation tests, (4) conduct a retardation test of inhibition, and (5) compare the effects of an inhibitory conditioning procedure to those of a random control procedure, that is, one in which there was a noncontingent, or "random," relationship between a taste stimulus and rotation. Monroe and Barker (1979) found that such a relationship between saccharin and a low dosage of lithium chloride pro-

duced no excitatory conditioning to the saccharin. Little is known about the role in discriminative conditioning of the degree of contextual excitation on the development of inhibition to an exteroceptive stimulus (LoLordo & Fairless, 1985). Even less is known about this issue when interoceptive (e.g., gustatory) CSs are concerned. If it is assumed that in differential conditioning of taste aversions, some generalization of excitation from a CS+ to a CS- is required to make the latter inhibitory, then the group receiving this treatment might be expected to function as a no-conditioning control.

Method

Subjects

The 18 male Sprague-Dawley rats weighed 130–150 g at the start of the experiment. They were divided into three groups of 6 subjects each, housed individually, and allowed unlimited access to food and water for 3 days after arriving in our laboratory. The lighting in the vivarium was automatically controlled to provide a 16-h light (6:00 a.m.–10:00 p.m.)/8-h dark (10:00 p.m.–6:00 a.m.) cycle. Temperature was maintained at about 20°C. Relative humidity was maintained at 45%–55%.

Materials and Apparatus

The rotation device consisted of a Q-Con Control Unit, Speed Control Type C 10-p-4, 115-V, 60-Hz motor produced by the Japan Servo Company. Step-down resistors permitted rotation at 78 rpm. The device was mounted at a 45° angle from the top of a 31.4 cm vertical upright extending from a 53.3 cm criss-cross wooden base. This arrangement permitted rotation at 45° from the horizontal axis. Four tubular stainless steel rods extended 30.7 cm from the center of the motor. Appended to the end of each rod was a 17.8 × 7.6 × 7.6 cm wire mesh (0.64 cm) cage with a wooden floor. Individual polyurethane holding cages (47 × 25 × 19 cm) were used during the two-bottle choice tests. The flavor solutions were a 0.3% sodium saccharin solution (3 g sodium saccharin/liter of water), a vanilla solution consisting of 1 ml McCormick vanilla extract to 30 ml water, a saccharin/vanilla compound solution (one part saccharin solution to one part vanilla solution), and a peppermint solution consisting of 1 ml McCormick peppermint extract to 30 ml water. All events were timed by a Junghans timer.

Procedure

After the 3-day period of habituation, the subjects were placed on a water-deprivation schedule, which permitted them 15 min/day access to water in their home cages once every 23 h. This water-deprivation schedule was in effect for 2 days prior to the start of the study proper.

There were three groups of subjects in this experiment: (1) a random control (RC) group, (2) a differential inhibition (DI) group, and (3) a differential inhibition plus novel stimulus (DINS) group. Table 1 depicts the general design of the experiment. Phase 1, which

Table 1
Summary of Procedures in Experiment 1

Group	Phase 1	Phase 2	Phase 3	Phase 4	Phase 5
Random	Saccharin–water choice	Saccharin + zero contingency Vanilla + no spin	Choice tests		
Differential inhibition	Saccharin–water choice	Saccharin + Spin Vanilla + No Spin	Choice tests		
Differential inhibition plus Novel CS	Saccharin–water choice	Saccharin + Spin Vanilla + no spin + random peppermint	Choice tests	Vanilla + spin Peppermint + spin	Vanilla–water Peppermint–water choice

lasted 1 week, established a stable, 60% preference for saccharin over water in a two-bottle test. This strategy was employed to provide a stable baseline from which a reduction in saccharin consumption could be convincingly demonstrated as a consequence of an aversive conditioning procedure. Phase 2 consisted of six conditioning cycles. Phase 3 was a conditioned-inhibition two-bottle test phase. Phase 4 was a retardation-of-conditioning procedure. Phase 5 was a two-bottle preference test, designed to detect any effects of the preceding treatment.

Phase 1. On the day following the initial water-deprivation period, the subjects were placed in their individual holding cages for 15 min and given a choice between tap water and a 0.3% solution of sodium saccharin. Bottles were placed side by side, 10.2 cm apart, in the grilled mesh tops of the cages. The positions of the bottles were randomized over days. This procedure was continued until a stable saccharin preference of 60% (saccharin/saccharin + water) or greater was reached. All subjects were exposed to this procedure for 7 consecutive days.

Phase 2. Once the 60% saccharin preference criterion was met, the subjects in the two differential inhibition groups received a 15-min single-bottle exposure to saccharin in their home cages on each of 6 saccharin-rotation days. Thirty seconds later, all rats received five 2-min rotations, at 78 rpm, at 45° from the horizontal axis. Between these 2-min periods, the apparatus was permitted to come to a complete stop (about 45 sec of progressively slower rotation) before being immediately restarted. It was expected that saccharin would function as a conditioned excitator for these subjects. On the 3 succeeding days of the first five cycles, the subjects in the two differential inhibition groups were exposed to the vanilla solution for 15 min in their home cages. Thirty seconds later, these subjects were placed in the apparatus for a 15-min period during which they were not rotated. Vanilla, it was assumed, would acquire conditioned inhibitory properties for these subjects.

To obviate the possibility of neophobia during summation testing, the subjects in the DINS group were exposed to a 1-ml solution of McCormick Peppermint to 30 ml tap water (as the novel stimulus) for 2 min, 10 h before the conditioning period on 7 randomly selected days of the study.

Conditioning took place during the light cycle between the hours of 6:00 p.m. and 8:00 p.m. for all subjects.

The following order of CS-US relationships was presented to the subjects in the RC group during this phase. On Day 1 of two of the conditioning cycles (Cycles 3 and 5), the subjects in the RC group were exposed to saccharin followed by rotation; on Day 1 of Cycles 2 and 6, the subjects were exposed to saccharin followed by no rotation; and on Day 1 of Cycles 1 and 4, the subjects were exposed to rotation which was not preceded by saccharin. In other words, the probabilities of saccharin plus rotation, saccharin and no rotation, and rotation and no saccharin, were equal. On the 3 succeeding days of each cycle, the subjects in this group were exposed to the vanilla solution, were placed in the apparatus, and were not rotated. As indicated previously, the assumption was that this "zero-contingency" procedure would result in no excitation in response to the saccharin and, thus, would provide no excitatory context against which inhibition to the vanilla could develop.

Phase 3 began on the day following the sixth saccharin-rotation treatment day for all subjects.

Phase 3. In Phase 3, all subjects were exposed for 15 min to the following two-bottle tests of preference. Day 1—saccharin versus vanilla. This choice was presented in order to assess any differential preference between the putative excitator and the putative inhibitor. It was predicted that vanilla would be the preferred solution for the subjects in both differential inhibition groups but not for those in the RC group. Day 2—saccharin plus vanilla versus water. This was a summation test of inhibition; the choice was presented in order to assess any differential preference between a compound of the excitator and the inhibitor and a neutral solution. The subjects

in the differential inhibition groups should prefer the compound to water. Day 3—saccharin plus vanilla versus saccharin alone. This choice was presented in order to assess any differential preference between a compound of the excitator and the inhibitor and the excitator alone, that is, a summation test of inhibition. Again, the compound should be preferred by the subjects in the differential inhibition groups. In addition, on Days 4 and 5, respectively, the subjects in the DINS group were exposed for 15 min to the following two-bottle preference-tests: saccharin plus peppermint versus water and saccharin plus peppermint versus saccharin alone. These choices were presented in order to assess any differential preference between a compound of the excitator and a novel taste stimulus and a neutral solution (water), as well as between this compound and the excitator alone. This latter manipulation is a control procedure for a summation test of inhibition. Peppermint should not increase the preference for saccharin on either test day.

Phase 4. On Days 1 and 2 of the retardation phase, the subjects in the DINS group were given 15-min initial two-bottle preference tests: on Day 1 for vanilla versus water, and on Day 2 for peppermint versus water. Following this determination of initial preference level for these two flavors, the subjects in this group were given three conditioning trials (with the rotation US) to each taste stimulus.

Phase 5. On the day following each of the above conditioning trials, there was a 15-min, two-bottle flavor-preference test (with water) for each flavor to assess any excitatory effects of the rotation treatment. It was expected that an aversion to vanilla would develop more slowly than one to peppermint.

Results and Discussion

Figure 1 shows the mean volumes of saccharin and water consumed by the three groups on the last day of Phase 1. As may be noted, saccharin was the preferred solution on this day. Figure 2 summarizes solution preferences for all test days of Phase 3. As may be seen, on Test Day 1, there was a diminution in the consumption of saccharin as a consequence of the conditioning procedure for all groups. An analysis of variance (ANOVA) revealed no difference among the groups in saccharin consumption on the last day of Phase 1 [$F(2,15) = 0.18, p > .05$]. However, an ANOVA applied to the saccharin intake data of the Day 1 of Phase 3 yielded a significant between-groups difference [$F(2,15) = 4.85, p < .05$]. A Tukey's HSD test was performed and indicated that the subjects in the RC group consumed significantly more saccharin than did the subjects in the DINS group, indicating that less aversion to saccharin had developed in the RC group.

Figure 2 also shows the vanilla-solution intakes for all groups, on each of the first 3 test days. ANOVAs were conducted on these solution intakes for the individual test days and revealed that on Test Day 1 (saccharin vs. vanilla), there was no difference among the groups in vanilla intake. However, an ANOVA of the percentage of vanilla solution consumed on this day indicated a significant difference among the groups [$F(2,15) = 4.68, p < .05$]. An HSD test showed that the mean percentage of vanilla solution consumed by the RC group was significantly less than that consumed by the DINS group. On Test Day 3 (saccharin-vanilla compound vs. saccharin alone) there was a significant difference among the

Figure 1. Mean saccharin and water intakes in Phase 1 of Experiment 1. Standard deviations are indicated by error bars.

groups in saccharin-vanilla solution consumption [$F(2,15) = 3.92, p < .05$]. An HSD test indicated that the amount of saccharin-vanilla solution consumed by the RC group was significantly less than that consumed by the other two groups. However, on Test Day 2 (saccharin-vanilla compound vs. water), there was no significant difference among the groups in saccharin-vanilla solution intake [$F(2,15) = 0.72, p > .05$].

On the whole, these data indicate that the vanilla solution had acquired inhibitory properties (as defined by Best, 1975) in all but the RC group. This assertion, however, must be tempered by the fact that all groups showed a preference for the saccharin-vanilla compound solution

in the preference test of Day 2 (i.e., against a neutral solution, water).

Figure 2 also shows the results of the compound stimulus tests with saccharin and the peppermint solution in the DINS group. As can be seen, addition of the peppermint did not produce an increase in consumption of the saccharin solution on either test day. Wilcoxon signed-ranks tests were conducted to determine if compound-solution consumption measures differed between Test Days 2 and 4 (saccharin plus vanilla vs. water, and saccharin plus peppermint vs. water) and between Test Days 3 and 5 (saccharin plus vanilla vs. saccharin alone, and saccharin plus peppermint vs. saccharin alone). Both comparisons re-

Figure 2. Mean solution intakes on all Phase 3 test days of Experiment 1. Standard deviations are indicated by error bars.

vealed significantly greater consumption of the compound containing vanilla (both $T_s = 0$, $p < .05$), thus demonstrating the effect of vanilla's conditioning history in these summation tests.

Figure 3 shows data for the DINS group in the retardation phase of the study. As may be seen, there was a marked preference for the vanilla solution when it was pitted against water on the initial preference-test day of this phase. This constitutes additional evidence compatible with the claim that the vanilla taste was functioning as an inhibitor in this group. As may also be seen in Figure 3, there was no evidence of excitation in response to the vanilla solution, which would have been indexed by reduced consumption of the vanilla solution from Days 1 to 3, despite three pairings with the rotation US. A Wilcoxon signed-ranks test conducted on the preference data between Test Day 1 and Test Day 3 confirms this observation ($T = 7.5$, $p > .05$). However, a Wilcoxon test performed on the preference data between Test Day 1 and Test Day 3 revealed a significant reduction in peppermint consumption over the 3 test days ($T = 0$, $p < 0.5$), thus indicating conditioning of excitation to this novel stimulus.

These findings show that taste aversion based on rotation can be established (thus replicating the findings of Braun & McIntosh, 1973; Green & Rachlin, 1973; Haroutunian & Riccio, 1975, and Ossenkopp & Tu, 1984). These data also show that such rotation-induced taste aversion can be inhibited. This demonstration of conditioned inhibition of taste aversion serves to replicate and extend the findings of Best (1975) and Batson and Best (1981) that a taste that signals the absences of illness is preferred in a two-bottle choice test. Using Rescorla's (1971) two principal tests necessary for proper demonstration of conditioned inhibition (summation and retardation), we were able to show evidence for conditioned inhibition of rotation-induced taste aversion (employing Best's preference definition) in all of our groups, including perhaps,

our RC group. That conditioning of saccharin aversion appears to have occurred in this group is not surprising given the one-trial nature of taste-aversion conditioning, which makes difficult the sequencing of CSs and USs required for a truly random group. In addition, Benedict and Ayres (1972), Kremer and Kamin (1971), and Quinsey (1971) have shown that with few conditioning trials, there may be evidence of conditioning in a random control group. This excitation could have provided the context against which vanilla may have acquired some inhibitory properties in our RC group. If this was indeed the case, it would indicate that the control procedure employed in this study did not function in that capacity and thus does not permit unequivocal interpretation of the data. An additional interpretational problem stems from the fact that the approach behavior shown to the vanilla solutions could possibly have resulted from a simple unconditioned preference for them. In the second experiment, we sought to address these issues. Specifically, in Experiment 2 we sought to (1) further study conditioned inhibition of rotation-induced taste aversion by employing more traditional Pavlovian conditioning controls, and (2) determine whether the results demonstrated in Experiment 1 could be attributed to an inherent preference for any solution.

EXPERIMENT 2

According to Domjan and Burkhard (1982), when experiments call for only a few trials—as do the present ones—it is preferable to use control groups that receive only the CS, or only the US. The control groups employed in Experiment 2 therefore, were these more traditional CS-only and US-only groups. In addition, in Experiment 2, we assessed initial preference levels for the solutions in order to determine whether flavor-choice behaviors resulting from the conditioning procedures could be attributed to inherent preferences for any flavor.

Figure 3. Mean intakes of vanilla and peppermint solutions in retardation tests of Experiment 1.

Method

Subjects

The subjects were 18 male Sprague-Dawley rats weighing approximately 130–150 g at the start of the experiment. They were divided into three groups of 6 subjects each, housed individually, and allowed unlimited access to food and water for a 3-day habituation period upon arriving in the laboratory.

Materials and Apparatus

The materials and apparatus were the same as those used in the first experiment.

Procedure

After a 3-day habituation period consisting of ad lib food and water, the subjects were placed on 15-min/day access to water for the duration of the experiment. For the subjects in the DI group, rotation treatments always followed exposure to the saccharin solution whereas exposure to the vanilla solution was always followed by periods of nonrotation. The subjects in Group CS-only received an equal number of exposures to saccharin and vanilla solutions as experienced by the subjects in the DI group, but these were always followed by periods of nonrotation. The subjects in the US-only group experienced the same number of rotations as did the subjects in the DI group. These rotations, however, were not signaled by saccharin. The subjects in this group also received an equal number of exposures to periods of nonrotation as did the subjects in the other groups; however, these periods were not signaled by the vanilla solution.

The experiment consisted of the following four phases: taste-preference assessment (Phase 1), saccharin-preference development (Phase 2), conditioning (Phase 3), and a taste-preference test (Phase 4).

Phase 1. Phase 1 consisted of 3 days of initial flavor-preference assessment in a two-bottle choice situation. On Day 1, all groups received a 15-min exposure to vanilla and water. On Day 2, the two-bottle choice was between the saccharin plus vanilla solution and water. On Day 3, the choice was between saccharin and water.

Phase 2. During Phase 2, which lasted 3 days, a 60% preference for saccharin over water was established in a two-bottle choice situation. All subjects received an equal number of saccharin–water choices during this phase.

Phase 3. During Phase 3, the subjects in the DI group were exposed to five 4-day cycles. On Day 1 of each cycle, the rats received 15-min access to saccharin in their home cages, followed 30 sec later by five 2-min periods of rotation. On the following 3 days, the rats received 15-min access to vanilla in their home cages followed by a 15-min period of nonrotation in the apparatus. For the sixth cycle, the subjects in this group received one session of saccharin followed by rotation. The subjects in the CS-only group received the same treatment schedule as those in DI group, except that the CS-only group subjects never received the rotation treatment. On Day 1 of each cycle, the CS-only subjects received a 15-min exposure to saccharin in their home cages and were placed in the apparatus for a 15-min period of nonrotation. On the following 3 days, the subjects in this group received 15-min access to vanilla in their home cages followed by a 15-min period of nonrotation in the apparatus. The subjects in Group US-only received water 2 h after rotation on Day 1 of each cycle. On the following 3 days, this group received water 2 h after a 15-min period of nonrotation in the apparatus.

Phase 4. Phase 4 consisted of 3 test days. On Day 1, all subjects received a two-bottle choice between the saccharin solution and water. The subjects in the DI group were expected to show a decrease in their intake of saccharin. The subjects in the other two groups were not expected to exhibit such a decrease. On Day 2, the two-bottle choice was between the vanilla solution and water.

We expected to see an increase in vanilla preference in the DI group, but not in the other two groups. On Day 3, in order to assess any differential effects of a compound of the excitator and the inhibitor and the excitator alone, the subjects received a two-bottle choice between the saccharin plus vanilla solution and water. Vanilla should increase saccharin consumption in the DI group, but not in the other two groups.

Results and Discussion

Separate ANOVAs conducted on both raw flavor solution-intake measures as well as on percentages of flavor solutions consumed in Phase 1 failed to reveal any flavor-preference differences within any group at the start of the experiment (all F s, $p > .05$).

Figure 4 depicts intakes in the saccharin–water preference tests in Phases 2 and 4. A repeated measures ANOVA performed on the saccharin-intake data between Phase 2 and Phase 4 yielded a significant groups $\times$ phase interaction [$F(1,15) = 4.99$, $p < .02$]. No other effects were significant. Tukey's test indicated that saccharin consumption for the DI group decreased significantly from Phase 2 to Phase 4, while it increased significantly for the CS-only group. Phase 2 and Phase 4 saccharin consumption did not differ for the US-only group.

Because the groups differed in total fluid intake in the vanilla versus water preference test of Phase 1, the percentage of vanilla consumed in Phases 1 and 4 was analyzed (see Figure 5). A repeated measures ANOVA conducted on the percentage of vanilla solution consumed between Phases 1 and 4 revealed a significant groups $\times$ phase interaction [$F(1,15) = 4.19$, $p < .04$]. Tukey's test showed that the subjects in the DI group significantly increased their vanilla intake from Phase 1 to Phase 4 while subjects in the other two groups did not.

Figure 6 shows the intake data for saccharin when it was pitted against water and for the saccharin–vanilla compound, also pitted against water. A repeated measures ANOVA conducted on Phase 4 saccharin-alone and saccharin plus vanilla consumption measures yielded significant between-groups differences [$F(2,15) = 3.66$, $p < .05$] and a strong interaction effect [$F(1,15) = 8.58$, $p < .004$]. Tukey's test revealed a significant increase in saccharin consumption for the DI group when vanilla was added to the solution, thus indicating the success of this summation test of vanilla as an inhibitor for this group. Vanilla produced no such increase in saccharin consumption for the other groups.

These results indicate that there were no within-groups differential preferences for any flavor solution at the start of the experiment. They also demonstrate that the conditioning procedure produced an aversion to saccharin in the DI group, but did not do so in the US-only group, despite six exposures to the rotation stimulus. Nor was there evidence of saccharin aversion in the CS-only group. In fact, the procedures produced an increase in saccharin consumption for this group. Although the effect was not significant, the CS-only group also appeared to increase its consumption of the vanilla solution from Phase 1 to

Figure 4. Mean saccharin and water intakes in Phases 2 and 4 of Experiment 2. Standard deviations are indicated by error bars.

Phase 4, so much so, in fact, that its Phase 4 vanilla preference was indistinguishable from that of the DI group's. This increase in intake of a solution as a simple function of the number of periods of access to it has been demonstrated by Domjan (1976, 1977) and Siegel (1974). This "preexposure effect," due perhaps to the habituation of neophobia, could have contributed to Phase 4 vanilla intake in both groups. We were, therefore, unable to determine how much of this vanilla-preference intake was due to the associative effects of inhibition and how much to the nonassociative effects of preexposure.

The purpose of the third experiment was to determine whether employing a control group in which *both* CS and

US exposures were equivalent to those of a conditioning group could serve to separate the two sources of preference increase.

EXPERIMENT 3

Monroe and Barker (1979) reported that an aversion to saccharin failed to develop in a group of rats exposed to a backward relationship between lithium chloride and saccharin. Such a backward conditioning procedure could provide a no-conditioning baseline in which CS and US exposures are equated with those of a conditioning group. The presence of the US in this alternative to a CS-only

Figure 5. Mean percentage of vanilla consumed in Phases 1 and 4 of Experiment 2. Standard deviations are indicated by error bars.

Figure 6. Mean intakes of saccharin alone and in compound with vanilla in Experiment 2. Standard deviations are indicated by error bars.

control could serve to "sensitize" ingestional neophobia (Domjan, 1977) and attenuate the effects of CS preexposure. In Experiment 3, we tested this hypotheses.

Method

Subjects

The subjects were 18 male Sprague-Dawley rats weighing approximately 130–150 g at the start of the experiment. They were divided into three groups of 6 subjects each, housed individually, and allowed unlimited access to food and water for a 3-day habituation period upon arriving in the laboratory.

Materials and Apparatus

The materials and apparatus were the same as those used in the first two experiments.

Procedure

After a 3-day habituation period consisting of ad lib food and water, the subjects were placed on a 15-min/day water-deprivation schedule for the duration of the experiment. For the subjects in the DI group, rotation treatments always followed exposure to the saccharin solution, whereas exposure to the vanilla solution was always followed by periods of nonrotation. The subjects in the backward group received an equal number of exposures to saccharin as experienced by the subjects in the DI group, but these exposures always followed periods of rotation. The subjects in the backward group also received an equal number of exposures to the vanilla solution as experienced by subjects in the DI group, but these exposures followed periods of nonrotation. The subjects in the US-only group experienced the same number of rotations as did the subjects in the two other groups. They were not, however, exposed to saccharin during conditioning. The subjects in this group also received an equal number of exposures to periods of nonrotation as did the subjects in the other groups; however, they were not exposed to the vanilla solution during conditioning.

The experiment consisted of the following four phases: taste-preference assessment (Phase 1), saccharin-preference development (Phase 2), conditioning (Phase 3), and a taste-preference test (Phase 4).

Phase 1. Phase 1 consisted of 2 days of initial flavor-preference assessment in a two-bottle choice situation. On Day 1, all groups received a 15-min exposure to vanilla and water. On Day 2, the choice was between saccharin and water.

Phase 2. During Phase 2, lasting 2 days, a 60% preference for saccharin over water was established in a two-bottle choice situation. All subjects received an equal number of saccharin/water choices during this phase.

Phase 3. During Phase 3, the subjects in the DI group were exposed to five 2-day cycles. On Day 1 of each cycle, the rats received 15-min access to saccharin in their home cages, followed 30 sec later by five 2-min periods of rotation. On the following day, the rats received 15-min access to vanilla in their home cages followed by a 15-min period of nonrotation in the apparatus. For the sixth cycle, the subjects in this group received one session of saccharin followed by rotation. The subjects in the backward group received the same rotation-treatment schedule as those in the DI group, except that the backward group subjects received saccharin 30 sec after their rotation treatment. That is, on Day 1 of each cycle, the subjects in the backward group received a 15-min exposure to saccharin in their home cages 30 sec after a rotation treatment. On the following day, the subjects in this group received a 15-min access to vanilla in their home cages following a 15-min period of nonrotation in the apparatus. The US-only group subjects received water 2 h after rotation on Day 1 of each cycle. On the following day, this group received water 2 h after a 15-min period of nonrotation in the apparatus.

Phase 4. Phase 4 consisted of 2 test days. On Day 1, all subjects received a two-bottle choice between saccharin solution and water. Saccharin consumption should decrease for the subjects in the DI group, but not for those in the other two groups. On Day 2, the two-bottle choice was between the vanilla solution and water. Vanilla consumption should increase for the subjects in the DI group, but not for those in the other two groups.

Results and Discussion

A repeated measures ANOVA conducted on Phase 2 and Phase 4 saccharin-intake measures revealed significant treatment [$F(2,15) = 10.73, p < .01$] and interac-

Figure 7. Mean saccharin and water intakes in Phases 2 and 4 of Experiment 3. Standard deviations are indicated by error bars.

tion effects [$F(1,15) = 8.49, p < .01$]. Tukey's test showed that the subjects in the DI group drank significantly less saccharin in Phase 4 than in Phase 2, indicating that an aversion to saccharin had developed in this group. There were no significant differences in saccharin intakes between Phase 2 and Phase 4 in the other two groups, indicating that these subjects had not developed a saccharin aversion. Figure 7 depicts these results.

Figure 8 shows the effects of conditioning on the vanilla-intake data. A repeated measures ANOVA on vanilla intake between Phase 1 and Phase 4 revealed significant treatment [$F(2,15) = 5.44, p < .01$] and interaction effects [$F(1,15) = 10.01, p < .01$]. Tukey's test showed that all groups increased their vanilla consumption from Phase 1 to Phase 4. Separate nonparametric ANOVAs between pairs of groups confirmed the fact that the subjects in the DI group consumed more vanilla in Phase 4 than did the subjects in the backward Group [$H(1) = 7.41,$

$p < .01$] and the US-only group [$H(1) = 8.31, p < .01$]. Vanilla intake did not differ between the backward and the US-only groups [$H(1) = 1.64, p > .05$].

These results demonstrate that, despite the fact that saccharin aversion developed only in the DI group, vanilla intake increased from Phase 1 to Phase 4 in all groups. As in Experiment 2, the effects of flavor preexposure were evident in the data of the present experiment. However, despite these effects, Phase 4 vanilla intake in the DI group was significantly greater than vanilla intake in the backward and US-only groups. That is, the control procedure employed in the present experiment, although failing to reduce the effects of preexposure entirely, did permit us to separate the associative effects of conditioning in the DI group from the nonassociative effects of preexposure in the backward group.

These findings lend support to an interpretation that the vanilla solution was indeed functioning as a conditioned

Figure 8. Mean vanilla and water intakes in Phases 1 and 4 of Experiment 3. Standard deviations are indicated by error bars.

inhibitor in the DI group. The data from all three experiments provide converging evidence for the existence of conditioned inhibition of rotation-induced taste aversion, thus demonstrating the generality of this phenomenon within the CTA framework.

GENERAL DISCUSSION

In Treisman's (1977) hypothesis linking the evolution of motion sickness to mechanisms for protecting organisms from the effects of ingesting and ingested neural toxins, a significant role is attributed to learning. To wit, organisms become motion sick when visual, vestibular, and proprioceptive inputs conflict with learned expectancies, and learning mechanism serve to ensure future avoidance of a toxin when its taste becomes aversive through association with toxin-induced gastrointestinal upset. Data from the present research effort, along with those of researchers like Ossenkopp and Tu (1984), constitute further evidence for the role played by learning mechanisms in the phenomenon of motion sickness.

These experiments also serve to replicate those of Braun and McIntosh (1973), Green and Rachlin (1973), and Haroutunian and Riccio (1975) in demonstrating that rotation stimulation, like a variety of toxins, will support taste-aversion learning. That taste aversion can be conditioned by means of provocative vestibular stimulation lends credence to Treisman's (1977) speculation that such stimulation mimics the effects of some toxins and that susceptibility to motion sickness may be part and parcel of possessing a system for detecting and expelling toxins. Indeed, as Braun and McIntosh (1973) note, rotation stimulation has two features in common with many of the USs that are effective in producing CTA: (1) it induces visceral discomfort and emesis (in species capable of vomiting), and (2) these symptoms are eliminated by small lesions in "chemoreceptor trigger zones" of the *area postrema* (Borison & Wang, 1953).

Evidence for the related and critical role played by the vestibular system in contributing to motion-induced visceral discomfort in taste-aversion conditioning in rats is seen in the findings of Haroutunian and Riccio (1975), and Wilpizeski, Lowry, Green, Smith, and Melnick (1987) have shown that bilateral labyrinthectomy also prevents rotation-induced taste aversion in squirrel monkeys.

All of these findings converge to lend support to Treisman's (1977) notion that rotation- and toxin-induced taste aversions are related. Given this presumed relationship, it is not surprising that our findings of conditioned inhibition in rotation-induced taste aversion are similar to those of Best (1975) and Batson and Best (1981) with toxin-induced CTA (i.e., with apomorphine and lithium chloride, respectively). That is, like these researchers, who employed an A+, AX- conditioning procedure, we were able to show, using a discriminative conditioning procedure, that a taste that signals the absence of illness is more acceptable in a two-bottle preference test than the

same taste in a control condition. This condition inhibition was demonstrated, as in Best's (1975) experiment, in the presence of an excitatory context (e.g., on Test Day 1 of Experiment 1) and, like Batson & Best (1981), in its absence (e.g., during the retardation tests of Experiment 1 and in the vanilla-preference test of Experiment 3). The existence of conditioned inhibition was also indicated in the compound stimulus tests of Experiments 1 and 2.

These findings parallel those of Taukulis and Revusky (1975), who demonstrated the power of an olfactory conditioned inhibitor (developed with an A+, AX- procedure) to elevate consumption of an excitatory taste stimulus.

The present results, however, contrast sharply with those of Delamater, Kruse, Marlin, and LoLordo (1986), who failed to demonstrate conditioned inhibition in taste-aversion conditioning. Like Delamater et al., the present authors are hard-pressed to discover a reason for these discrepant findings.

In addition to the above considerations, the present effort also had an applications-oriented goal, that is, one of determining the feasibility of developing a conditioned inhibitor of motion-induced taste aversion to be employed in ameliorating the symptoms of motion sickness.

Konorski (1967) suggested that conditioned inhibitors elicit an affective state opposite to that elicited by unconditioned stimuli. More specifically, he asserted that an inhibitor, signaling as it does the absence of a reinforcer, becomes associated with two new centers, one sensory, the other affective, which have their own inhibitory connections to the sensory and affective centers activated by presentation of the reinforcer (the US) and/or the excitatory stimulus (the CS). In the present case, hypothetically, a "nonrotation" center and a "motion-sickness anti-drive" center sent inhibitory connections to a "rotation" and a "motion-sickness" center, respectively, thus suppressing their activation. According to this account, when our inhibitor was presented, it produced an affective state opposite to that elicited by the aversive excitatory CS—that is, "nonmotion sickness." The possibility that this occurred in our study is seen in all the demonstrations of preference for the inhibitory taste stimulus. This finding raises the intriguing question of whether conditioned inhibitory stimuli could be used to inhibit maladaptive reflexive responses in general, including those associated with motion sickness. Future research employing a more direct index of motion sickness can answer this question.

REFERENCES

- BATSON, J. D., & BEST, M. R. (1981). Single-element assessment of conditioned inhibition. *Bulletin of the Psychonomic Society*, 18, 328-330.
- BENEDICT, J. D., & AYRES, J. B. (1972). Factors affecting conditioning in the truly random control procedure in the rat. *Journal of Comparative & Physiological Psychology*, 78, 323-330.
- BEST, M. R. (1975). Conditioned and latent inhibition in taste aversion

- learning. *Journal of Experimental Psychology: Animal Behavior Processes*, **1**, 97-113.
- BORISON, H. L., & WANG, S. C. (1953). Physiology and pharmacology of vomiting. *Pharmacological Reviews*, **5**, 193-230.
- BRAUN, J. J., & MCINTOSH, H., JR. (1973). Learned taste aversions induced by rotational stimulation. *Physiological Psychology*, **1**, 301-304.
- CARLSON, N. R. (1981). *Physiology of behavior*. Boston: Allyn & Bacon.
- DELMATER, A. R., KRUSE, J. M., MARLIN, S., & LOLORDO, V. M. (1986). Conditioned inhibition in taste aversion learning: Testing methodology and empirical status. *Animal Learning & Behavior*, **14**, 6-14.
- DOMJAN, M. (1976). Determinants of the enhancement of flavored-water intake by prior exposure. *Journal of Experimental Psychology: Animal Behavior Processes*, **2**, 17-27.
- DOMJAN, M. (1977). Attenuation and enhancement of neophobia for edible substances. In L. M. Barker, M. R. Best, & M. Domjan (Eds.), *Learning mechanisms in food selection* (pp. 151-175). Waco, TX: Baylor University Press.
- DOMJAN, M., & BURKHARD, B. (1982). *The principles of learning and behavior*. Belmont, CA: Brooks/Cole.
- GREEN, L., & RACHLIN, H. (1973). The effect of rotation on the learning of taste aversions. *Bulletin of the Psychonomic Society*, **1**, 137-138.
- HAROUTUNIAN, V., & RICCIO, D. C. (1975). Acquisition of rotation-induced taste aversion as a function of drinking-treatment delay. *Physiological Psychology*, **3**, 273-277.
- KOHL, R. L. (1983). Sensory conflict theory of space motion sickness: An anatomical location for neuroconflict. *Aviation, Space, & Environmental Medicine*, **54**, 464-465.
- KONORSKI, J. (1967). *Integrative activity of the brain: An interdisciplinary approach*. Chicago: University of Chicago Press.
- KREMER, E. F., & KAMIN, L. J. (1971). The truly random control procedure: Associative or nonassociative effects in the rat. *Journal of Comparative & Physiological Psychology*, **74**, 203-210.
- LOLORDO, V. M., & FAIRLESS, J. L. (1985). Pavlovian conditioned inhibition: The literature since 1969. In R. R. Miller & N. E. Spear (Eds.), *Information processing in animals: Conditioned inhibition* (pp. 1-49). Hillsdale, NJ: Erlbaum.
- MONROE, B., & BARKER, L. M. (1979). A contingency analysis of taste aversion conditioning. *Animal Learning & Behavior*, **7**, 141-143.
- OSSENKOPP, K.-P., & TU, G. S. (1984). Motion sickness in quail: Body-rotation-induced conditioned fluid aversions in *C. coturnix japonica*. *Journal of Comparative Psychology*, **98**, 189-193.
- QUINSEY, V. L. (1971). Conditioned suppression with no CS-US contingency in the rat. *Canadian Journal of Psychology*, **25**, 69-82.
- REASON, J. T., & BRAND, J. J. (1975). *Motion sickness*. New York: Academic Press.
- RESCORLA, R. A. (1969). Pavlovian conditioned inhibition. *Psychological Bulletin*, **72**, 77-94.
- RESCORLA, R. A. (1971). Summation and retardation tests of latent inhibition. *Journal of Comparative & Physiological Psychology*, **75**, 77-81.
- SIEGEL, S. (1974). Flavor preexposure and "learned safety." *Journal of Comparative & Physiological Psychology*, **87**, 1073-1082.
- TAUKULIS, H. K., & REVUSKY, S. H. (1975). Odor as a conditioned inhibitor: Applicability of the Rescorla-Wagner model to feeding behavior. *Learning & Motivation*, **6**, 11-27.
- TREISMAN, M. (1977). Motion sickness: An evolutionary hypothesis. *Science*, **197**, 493-495.
- WILPIZESKI, C. R., LOWRY, L. D., GREEN, S. L., SMITH, B. D., & MELNICK, H. (1987). Subjective concomitants of motion sickness: Quantifying motion-induced illness in squirrel monkeys. *Otolaryngology, Head & Neck Surgery*, **97**, 433-440.

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