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Resistance of "recovery" flavors to later association with illness

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Three groups of rats were given 4 days of exposure to saccharin solution: for the recovery group, exposure to saccharin was paired with recovery from thiamine deficiency; the familiarity group was given sacccharin without experiencing either illness or recovery from illness; the recovery-familiarity group was given saccharin after recovering from thiamine deficiency. When these groups were later given saccharin solution followed by an injection of lithium chloride, only the familiarity and recovery-familiarity groups showed a strong long-lasting aversion to saccharin solution. The results are discussed in relation to the previously observed effects of novelty on food preferences and taste aversions in the rat.

Although there have been several successful demonstrations of rats' preferences for flavors which have been paired with recovery from illness (Garcia, Ervin, Yorke, & Koelling, 1967; Green & Garcia, 1971; Seward & Greathouse, 1973; Zahorik, 1972; Zahorik & Maier, 1969), it has been suggested that none of these studies offers conclusive evidence for any appetitive conditioning involving flavors and positive gastrointestinal consequences and that all these results can be explained using only the concepts of learned aversions, neophobia, and increased neophobia following experience with illness (McFarland, 1973; Rozin & Kalat, 1971). However, more recent evidence suggests that preferences produced by pairing a flavor with recovery from illness are even larger than the "learned safety" effect produced by pairing a flavor with simply being well and that the preferences shown for recovery flavors are probably due in part to associations between the tastes and recovery from illness (Zahorik, Maier, & Pies, 1974).

If rats prefer flavors paired with recovery to other familiar flavors, it seems reasonable to ask whether the inhibitory effects of familiarity on taste aversion learning might be even stronger for recovery flavors than for other familiar flavors. Specifically, familiar flavors are less readily associated with gastrointestinal illness than are novel flavors (Maier, Zahorik, & Albin, 1971; Revusky & Bedarf, 1967), as if familiarity somehow "immunizes" flavors against subsequent association with unpleasant gastrointestinal consequences. If rats develop smaller aversions to flavors which are familiar than to novel flavors when both flavors are paired with gastrointestinal illness, will they develop still smaller aversions to flavors which are first paired with recovery from one g.i. illness and later paired with a second g.i. illness?

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The present experiment is an attempt to assess any differences between the strengths of aversions developed to flavors which have previously been paired with recovery from thiamine deficiency and flavors which have become familiar in other contexts when those flavors are later paired with illness produced by lithium chloride poisoning. The *recovery* group drank saccharin solution on 4 days when they were rapidly recovering from thiamine deficiency; the *familiarity* group received exactly the same sequence of flavors as the recovery group, including the 4 days of saccharin solution, but they were never thiamine deficient. A third group served as a control for the possibility that any differences between the recovery and familiarity groups could be attributed to the fact that only the recovery group had a history of illness prior to the pairing of saccharin and poisoning, rather than to the conditions under which the two groups became familiar with saccharin: this group (*recovery familiarity*) had the same history of thiamine deficiency and recovery as the recovery group, but their 4 days of exposure to saccharin came *after* their last recovery from deficiency rather than being paired with recovery. All three groups were given plain tap water for 4 days following these procedures and then poisoned after 5 min access to saccharin solution.

METHOD

Thirty 60-day-old male rats, CD strain from Charles River Labs, Charles River, New Jersey, served as subjects. The 20 rats which formed the recovery and familiarity groups were run simultaneously; they were randomly assigned to one of those two groups and housed in individual wire mesh cages. The 10 animals assigned to the recovery group were fed thiamine-deficient diet (Nutritional Biochemicals Company) ad lib throughout the experiment, while the 10 animals in the familiarity group were fed a nutritionally complete diet which was identical to the thiamine-deficient diet in every respect except thiamine content. The rats were weighed every 3rd day throughout the experiment, beginning on Day 3.

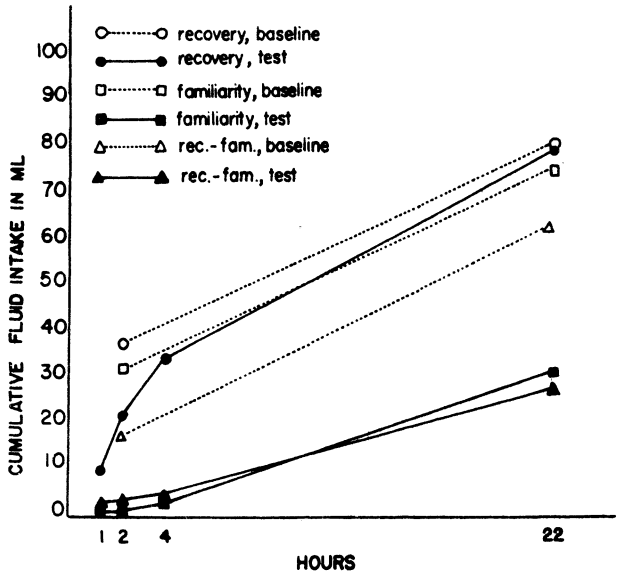

Figure 1. Mean cumulative intake of saccharin solution for the three groups during the baseline period and during the test following the pairing of saccharin with lithium chloride poisoning.

While the recovery and familiarity groups received these diets, it was possible to manipulate thiamine deficiency and recovery from deficiency in the recovery group and to maintain the familiarity group in good health by giving both groups the same series of injections: during the deficiency phase of the experiment (Days 1-23), all subjects were given IP injections of saline every 6th day (Days 6, 12, and 18); and on Days 24, 30, and 36, all subjects were given injections containing thiamine. The saline injections were matched in volume and pH to the thiamine injections, which contained 200 micrograms thiamine HCl/kg body weight on Days 24 and 30, and 1600 micrograms thiamine HCl/kg body weight on Day 36. These manipulations were designed to produce 23 days of increasing thiamine deficiency followed by three brief periods of recovery from thiamine deficiency in the recovery group, while producing neither deficiency nor recovery in the familiarity group.

Both groups were given unlimited access to a distinctively flavored liquid or to plain water on each day of the experiment, and fluid intake was measured daily. During the first 23 days, and on the 5 days following each of the first two thiamine injections, all the rats were given quinine solution (.2 g quinine HCl/liter tap water). On each of the 3 days when thiamine injections were administered, all subjects were given saccharin solution (2 g sodium saccharin/liter tap water). Thus saccharin was paired with recovery from illness for the recovery group and was simply made familiar for the familiarity group.

On the day following the third thiamine injection (Day 37), the water bottles were removed from all cages for 2 h immediately after the fluid intake was measured for Day 36. The bottles of saccharin solution were then returned to the cages for 22 h, and fluid intake was recorded at the end of 2 h and 22 h. These measurements are the baseline data for consumption of saccharin solution which had been paired with recovery or simply made familiar. When the baseline recordings had been completed, all subjects were given tap water for the next 4 days.

The 5th day following the collection of baseline data was the test day (Day 42). The bottles containing tap water were removed from the cages immediately after fluid intake for the preceding day was measured. Then each subject was given 5 min of access to saccharin solution, followed by an IP injection of .3 M lithium chloride (1% of body weight). All subjects drank the familiar and highly palatable saccharin solution eagerly during this 5-min exposure, and there was no significant difference in saccharin intake between the groups. The bottles of saccharin were removed from the cages while the injections were being given and were returned to each cage 2 h after the injection, with fluid intake recorded 1 h, 2 h, 4 h, and 22 h after the bottles were replaced. The 2-h delay before returning the bottles to the cages was imposed to allow the rats to recover from the severe nausea produced by the lithium chloride before measuring fluid intake, thus reducing variability in intake due to differences in the time required to recover from the initial effects of the poison; by the time the bottles were returned to the cages, all the rats had started to eat again, suggesting that nausea was no longer severe. The measurements of fluid intake on Day 42 are the test data for the recovery and familiarity groups.

The 10 rats in the recovery-familiarity group were run 6 months after the first two groups and received exactly the same treatment as the recovery group, with the following exceptions: (1) The flavor paired with recovery from deficiency (Days 24, 30, 36, and 37) was vanilla (50 ml/liter of tap water) instead of saccharin. (2) The 4 days of exposure to saccharin occurred on Days 38-41 when the most rapid phase of recovery from deficiency was complete. Baseline measurements were made on Day 41. (3) The rats in the recovery-familiarity group were given a second 1600-microgram/kg injection of thiamine on Day 42 and were given tap water on Days 42-45. (4) This group received the same 5-min exposure to saccharin, lithium chloride injections, and measurements of fluid intake as the other groups, but their test day was the 46th day of the experiment.

RESULTS

The measurements of fluid intake and body weight throughout this experiment indicated that the rats in the recovery and recovery-familiarity groups became severely thiamine deficient during the first 23 days of the experiment, showed brief recoveries after the thiamine injections on Days 24 and 30, and showed prolonged recoveries from deficiency following the injection of a larger dose of thiamine on Day 36; during the same period, the rats in the familiarity group gained weight continuously and showed no decrease in fluid intake. Because the effects on body weight and fluid intake were virtually identical to those reported in other studies employing similar manipulations to produce thiamine deficiency and recovery (see Zahorik, 1972; Zahorik, Maier, & Pies, 1974), the data for body weight and fluid intake will not be presented here.

When the baseline measurements were made on the 4th day of exposure to saccharin, all animals consumed large quantitites of the solution, with the recovery and familiarity groups consuming more than the recovery-familiarity group after 2 h and after 22 h. Subjects in all three groups continued to drink large quantities of fluids and to gain weight on the 4 days following the baseline measurements, indicating that none of the animals were thiamine deficient during that period.

Figure 1 shows the fluid intake for all groups during the baseline measurements and on the test day, when a 5-min exposure to saccharin solution had been followed by lithium chloride-induced illness. After 1 h of the test period, none of the rats in the familiarity group had consumed a measureable quantity of the saccharin

solution, and only five of the recovery-familiarity rats had started to drink, while each of the animals in the recovery group had consumed at least 1 ml of liquid; after 2 h, none of the rats in the familiarity or the recovery-familiarity groups had consumed more than a few milliliters of saccharin, while the mean fluid consumption in the recovery group was 19.5 ml. All groups drank less during the first 2 h of the test than during the first 2 h of the baseline measurement, suggesting either a learned aversion to the saccharin resulting from its recent pairing with gastrointestinal illness or continuing nausea from the lithium chloride injection. After 22 h, all subjects had consumed appreciable amounts of liquid, but the recovery group had consumed about as much saccharin as on the baseline day, while the other two groups drank less than half as much as they drank on the baseline day. Thus a comparison of the baseline data and test data for these groups suggests that rats poisoned after drinking a familiar flavor develop an aversion to that flavor which lasts for at least 22 h, while rats poisoned after drinking a familiar flavor previously paired with recovery from thiamine deficiency show only a brief avoidance of that taste and no measureable aversion to the taste in a 22-h test.

These effects were confirmed by the results of a 3 x 2 x 2 ANOVA, with the conditions of previous experience with the flavor (recovery vs. familiarity vs. recovery-familiarity) as the between-subjects variable and testing conditions (baseline vs. test after pairing with poison) and time of testing (first 2 h vs. last 20 h) as the within-subjects variables. All the main effects were significant at the .001 level: the animals which had become familiar with saccharin through its pairing with recovery from thiamine deficiency drank more of that flavor than did the subjects in the other two groups ($F = 11.04$, $df = 2,27$); the rats drank more saccharin during the baseline test than they drank after saccharin had been followed by poison ($F = 45.98$, $df = 1,27$); and of course the subjects drank more saccharin during the last 20 h of each test than they drank during the first 2 h ($F = 50.67$, $df = 1,27$).

More important for the central question in this experiment is the significant interaction between treatment groups and testing conditions ($F = 10.62$, $df = 2,27$, $p < .001$); while saccharin intake decreased dramatically following the pairing of saccharin and poison in the familiarity and recovery-familiarity groups, the recovery group consumed almost as much saccharin solution after poisoning as during the baseline period. Post-hoc comparisons revealed that the recovery group consumed significantly more saccharin than either of the other groups after 2 h and after 22 h. All four comparisons were significant at $p < .01$. There were no significant differences between the familiarity group and the recovery-familiarity group. There was a significant interaction between the test conditions and the time of testing ($F = 4.30$, $df = 1,27$, $p < .05$), with all groups drinking a larger proportion of their total fluid intake during the first 2 h of the baseline test than during the first 2 h of the test following poison.

In summary, both groups which had simply been exposed to saccharin solution for 4 days and were then poisoned after drinking saccharin showed strong aversions to that flavor, while rats which had the same solution paired with recovery from thiamine deficiency showed little aversion to saccharin after saccharin had been paired with poison.

DISCUSSION

In 1971, Rozin and Kalat suggested that it was possible to categorize foods as either novel, familiar-safe, or familiar-aversive, based on an animal's previous experience with the food. It was argued that all evidence for the existence of a fourth category of foods, familiar-positive (foods which have been associated with recovery from illness and are thus not only safe but also beneficial), can actually be explained in terms of these three basic categories. Rozin and Kalat also stated that if a food paired with recovery from illness could be shown to be preferred to other familiar foods (familiar-safe) as well as to novel foods, such a demonstration would constitute evidence for a familiar-positive category. The first evidence that flavors paired with recovery from illness are preferred to other familiar flavors was provided by Zahorik, Maier, and Pies (1974); the results of the present study suggest that flavors paired with recovery are also less readily associated with illness than are flavors which have simply been made familiar, even when a flavor is made familiar after repeated experience with illness and recovery as in the recovery-familiarity group. Taken together, these two results offer very strong evidence that rats treat flavors paired with recovery from illness differently than other familiar flavors and that flavors paired with recovery should be assigned to Rozin's familiar-positive category. If learning that a flavor is safe (by experiencing the taste without getting sick) interferes with later associating that taste with illness (Kalat & Rozin, 1973), then learning that a flavor produces beneficial consequences (by experiencing the taste while recovering from illness) seems to produce even stronger interference with later associations between that taste and illness.

While it may seem little more than an academic exercise to make fine distinctions between categories of familiar flavors based on the conditions under which the flavors became familiar, recent evidence suggests that the familiarity variable may play an extremely important role in some of the most intriguing aspects of the taste aversion phenomenon, including learning over long delays and "belongingness." For example, Kalat and Rozin (1973) have proposed that the rat's reduced ability to associate familiar tastes with poisoning and the decreasing ability to associate tastes with illness over long delays may both reflect the same learning mechanism–the association of tastes with the *absence* of illness or "learned safety." More recently, Mitchell, Kirschbaum, and Perry (1975) have reported a series of studies suggesting that differences in familiarity may be critical in producing the apparent specificity with which tastes (rather than exteroceptive cues) become associated with illness. They present evidence that exteroceptive cues are readily associated with illness when a *novel* exteroceptive cue is paired with illness in an otherwise *familiar* situation. These studies suggest that the familiarity of cues, the conditions under which the cues become familiar, and the effects of previous experience with illness on the animal's reaction to familiar cues may be more important factors in taste aversions than most researchers have supposed and that these variables should be given more careful attention.

REFERENCES

GARCIA, J., ERVIN, F. R., YORKE, C. H., & KOELLING, R. A. Conditioning with delayed vitamin injections. *Science*, 1967, **155**, 716-718.

GREEN, K. F., & GARCIA, J. Recuperation from illness: Flavor enhancement for rats. *Science*, 1971, **173**, 749-751.

KALAT, J. W., & ROZIN, P. "Learned safety" as a mechanism in long-delay taste aversion learning in rats. *Journal of Comparative and Physiological Psychology*, 1973, **83**, 198-207.

MCFARLAND, D. J. Stimulus relevance and homeostasis. In R. A. Hinde & J. Stevenson-Hinde (Eds.), *Constraints on learning*. New York: Academic Press, 1973.

MAIER, S. F., ZAHORIK, D. M., & ALBIN, R. W. Relative novelty of solid and liquid diet during thiamine deficiency determines development of thiamine-specific hunger. *Journal of Comparative and Physiological Psychology*, 1971, **74**, 254-262.

MITCHELL, D., KIRSCHBAUM, E. H., & PERRY, R. L. Effects of neophobia and habituation on the poison-induced avoidance of exteroceptive stimuli in the rat. *Journal of Experimental Psychology: Animal Behavior Processes*, 1975, **104**, 47-55.

REVUSKY, S. H., & BEDARF, E. W. Association of illness with prior ingestion of novel foods. *Science*, 1967, **155**, 219-220.

ROZIN, P., & KALAT, J. W. Specific hungers and poison avoidance as adaptive specializations of learning. *Psychological Review*, 1971, **78**, 459-486.

SEWARD, J. P., & GREATHOUSE, S. R. Appetitive and aversive conditioning in thiamine-deficient rats. *Journal of Comparative and Physiological Psychology*, 1973, **83**, 157-167.

ZAHORIK, D. M. Conditioned physiological changes associated with learned aversions to tastes paired with thiamine deficiency in the rat. *Journal of Comparative and Physiological Psychology*, 1972, **79**, 189-200.

ZAHORIK, D. M., MAIER, S. F., & PIES, R. W. RPreferences for tastes paired with recovery from thiamine deficiency in rats: Appetitive conditioning or learned safety? *Journal of Comparative and Physiological Psychology*, 1974, **87**, 1083-1091.

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