Reversal of Innate Aversions: Attempts to Induce a Preference for Chili Peppers in Rats

Paul Rozin, Leslie Gruss, and Geoffrey Berk
University of Pennsylvania

Although humans frequently develop preferences for innately unpalatable bitter or irritant substances, such preferences are extremely rare in animals. An attempt was made to understand the nature of this difference by systematic experiments with laboratory rats, with chili pepper as the unpalatable substance. In parallel with major aspects of the human experience with chili pepper, rats were exposed to it as a flavoring in all their food for periods up to 11 mo from birth, without significant preference enhancement. Gradual introduction of chili into the diet also had no effect, nor did a series of poisoning and safety experiences designed to teach the rats that only chili-flavored foods were safe to eat. A sequence of seven pairings of chili-flavored diet with prompt recovery from thiamine deficiency did significantly attenuate the innate aversion and may have induced a chili preference in at least one case. Extensive experience with chili did not reliably make rats much less sensitive to its oral effects. The only reliable way to eliminate chili aversion in rats is to destroy their chemical irritant sense, which was accomplished in one group of rats. It is concluded that in contrast to humans, it is extremely difficult to reverse innate aversions in rats.

Almost all animals have receptors that cause them to avoid potentially noxious chemicals (Beidler, 1975; Garcia & Hankins, 1975; Rozin, 1976). Many substances that are innately rejected by mammals produce bitter (taste mediated) or chemical irritant (trigeminally mediated) sensations in humans. Chemicals that stimulate either of these senses tend, in nature, to be harmful. Although there are a few species of vertebrates that may specialize in ingesting some substances with the chemical properties that lead to these sensations (e.g., Hume, 1874), and/or that possess special detoxifying mechanisms for associated toxins, almost all omnivorous vertebrates that have been studied avoid these innately unpalatable tastes.

This research was supported by National Science Foundation Grant BNS 76-80108 to Paul Rozin. We express thanks to H. J. Grill and R. L. Solomon for comments on the manuscript, to the Avoca Division of the R. J. Reynolds Tobacco Co. for providing some of the chili pepper, and to P. H. Todd of the Kalamazoo Spice Extraction Co. for determination of Scoville levels of the chili pepper.

Requests for reprints should be sent to Paul Rozin, Department of Psychology, University of Pennsylvania, Philadelphia, Pennsylvania 19104.

Copyright 1979 by the American Psychological Association, Inc. 0021-9940/79/9306-1001$00.75

1001
mans may refuse foods because of anticipated negative consequences of ingestion and/or because of hedonically negative sensations produced by the food. It has been argued (Garcia, Kovner, & Green, 1970; Rozin & Kalat, 1971) that in taste aversion learning, the poisoned food actually comes to taste bad, a hedonic change. Direct observation of animals suggests that such tastes have become aversive; behavior on exposure (spillage and facial gestures) resembles behavior toward innately unpalatable quinine (Grill, 1975; Rozin, 1967). Similarly, on the positive side, increased preference can be produced by anticipated positive consequences of ingestion (what we call the “medicine” effect) and/or taste enhancement, a positive hedonic effect. Young (1948) emphasized a distinction similar to this, with his concept of palatability tied to hedonic effects in need-free animals. The fact is that nonhuman animals can rarely be induced to show any sort of preference for an innately unpalatable substance, and there is no current evidence that convincingly shows a hedonic shift, from negative to positive, for such substances.

It would be reasonable to appeal to the effects of strong sociocultural forces that are uniquely human as an explanation of the ease with which humans come to prefer innately aversive substances. Social pressure may induce sufficient exposure to unpalatable foods to allow changes of preference to occur in humans. Such pressures would not normally exist in nonhumans and hence would deny them the exposure necessary to produce the preference. This explanation is faulty on two counts. First, it it not an explanation at all. It does not identify what exposure might do to reverse the unpalatability of these substances. Social pressure may cause people to try unpalatable foods, but what makes them come to like these foods? At this time, there are no convincing answers. Second, it is contradicted by an impressive amount of animal data suggesting that forced exposure to unpalatable substances does not lead to preference.

It has proved difficult to turn even a neutral taste stimulus into a highly preferred one, though this can be done (see Rozin & Kalat, 1971; Zahorik, 1977, for general discussions). This contrasts with the ease in establishing acquired aversive tastes. The acquired positive preferences reported in animals have usually been weak. They require a number of conditioning trials, and they extinguish or dissipate easily. What better evidence for this than the well-known fact that rats raised in the laboratory on rat chow, and experiencing many repletions from this chow after deprivation, will abandon it in favor of a new, more palatable diet after but a few days of choice. The weak acquired preferences that have been demonstrated could be interpreted to be mediated by “anticipation” of consequences (medicine effects) rather than hedonic shift.

There have been few experimental attempts to produce preferences for innately negative tastes. Mere exposure of infant rats to bitter tastes did produce a temporary preference (Warren & Pfaffmann, 1959). A more marked preference following mere exposure of rats to a garlic solution has been reported (Capretta & Rawls, 1974). However, this solution was only slightly aversive in comparison with water. Long exposure to some strong (irritant) spices did not significantly enhance the preference of rats in another study (Hilker, Hee, Higashi, Ikehara, & Paulsen, 1967). Two studies explicitly attempted to induce preferences by association of mildly aversive substances with positive events. Pairing of an aversive HCl solution with food in hungry rats led to a transitory reduction in the aversion (Siqueland, 1965). Pairing of a slightly aversive bitter-sweet solution with recovery from morphine withdrawal produced a substantial preference for this solution (Parker, Failor, & Weidman, 1973). This preference was demonstrated under conditions that would suggest a medicine effect rather than a hedonic shift. A few inbred strains of mice (McClearn, 1973) and some individual rats (Richter, 1941) showed a preference for moderate levels of ethanol in water over plain water, which suggests the possibility of a preference for the bitter component of ethanol. In these cases, there is some question whether the preferences are acquired or innate. If acquired, they could represent hedonic shifts, but they might also be medicine effects.

There are a few suggestive instances of
acquired preferences for innately unpalatable substances in nonhuman primates. Weiskrantz and Cowey (1963) reported that rhesus macaques can acquire a preference for initially rejected black currant juice. Kluver (cited in Kalmus, 1969) described voluntary ingestion of quinine powder by rhesus monkeys.

It is possible that the many failures to establish oral preferences for addictive substances in animals, especially alcohol (Mello, 1973; Richter, 1956), have foundered on the fact that most of these substances are innately unpalatable. A recent study demonstrated a rather substantial preference for a neutral tasting solution that was paired with intragastric alcohol repletion in alcohol-withdrawn rats, which suggests that the taste of alcohol is a significant barrier to preference development (Deutsch & Walton, 1977).

This study represents a concerted attempt to establish a preference for an innately unpalatable substance in rats. The substance selected is chili pepper, a chemical irritant. In spite of the fact that chili pepper is one of the most widely consumed spices in the world and is probably consumed on a daily basis by about one fourth of the adults in the world, there have been no studies on the nature or development of this preference (see Rozin, 1978). Chili peppers are rejected by young children and uninitiated adults around the world and, according to anecdotal evidence, by virtually all omnivorous animals. Foods seasoned with chili may be eaten by some omnivorous household animals, but there is no convincing evidence that they like or prefer foods so seasoned. Details on human consumption of chili pepper and the pharmacological effects of capsaicin, the substance that causes the irritation, can be found in a chapter by Rozin (1978; see also Rozin & Schiller, Note 1).

This series of studies is designed either to produce an animal “model” for a pervasive human activity or to indicate some of the reasons for this major difference between humans and animals. We also speculate about possible reasons for chili ingestion and preference in humans and on the long-term effects of chili ingestion. It is hoped that understanding of the development of a preference for this particular innately unpalatable substance will contribute to our understanding of the general process of acquisition of food preferences and, in particular, the reversal of innate rejections.

General Method

All subjects were albino laboratory rats (Charles River CD strain), either raised in the laboratory or obtained at ages 6 mo to 1 yr. They were housed in a temperature-controlled animal room, with a 14:10 hr light/dark cycle. They were occasionally housed in community cages after they were individually marked. All testing was done in standard 25 × 20 × 18 cm wire cages, with water ad lib and food choices as indicated. The basal diet for all animals unless otherwise indicated was powdered Purina Rat Chow. The major additive in these experiments was ground chili pepper. All food-choice measurements were based on daily or hourly weighings of the contents of two glass 2-oz. (57-ml) food cups, attached to the front wall of the cage by wire loops. Paper under the cages was used to collect spillage. Both cups and spillage were weighed to the nearest .5 g. Food-cup positions were alternated daily. Prior to any preference test, rats had at least 2 days to adapt to the two-cup presentation format, with the same (basal) food in both cups. Animals were weighed daily during preference test periods and occasionally in periods between tests.

Experiment 1: Exposure, Learned Safety, and Chili Preference

The simplest interpretation of the genesis of human chili preference is that it results from long-continued exposure to the spice. Although this type of procedure has not worked in prior studies with animals with other unpalatable substances, it is so basic that it was repeated here with longer exposures than in previous studies. The chili level in the diet used produced a piquancy comparable with that of dishes in many chili-eating cuisines (e.g., Mexico, west Africa, Ethiopia, or parts of India and China). In one group of animals, the exposure effects were supplemented by a “safety” procedure. Sickness was induced in rats raised on chili diets whenever they consumed a diet that

1 The chili pepper used in Experiments 1, 2, 4, and 5 was purchased locally, in bulk, under the name of "cayenne pepper." This pepper had a pungency of 40,000 Scoville units. The pepper used in Experiment 3 came from the Avoca Division of the R. J. Reynolds Tobacco Co. and was rated with a pungency of 100,000 Scoville units. One Scoville unit is the human detection threshold under a set of specified conditions.
was not flavored with chili. In this way, chili flavor might have become preferred as a safety signal. On the one hand, this procedure seemed like one that would successfully induce preferences for a neutral diet, on the basis of the learned aversion literature and principles of generalization. On the other hand, it is conceivable that distinctive flavorings that are used repetitively and perversely in particular cultures may serve the function of labeling food as safe (Rozin, 1976, 1978).

Method

All subjects were born in the laboratory within a 10-day period. Control rats (n = 13) were from two litters, and exposure (n = 11) and safety (n = 11) rats were randomly selected from three other litters. Two weeks before mating, exposure and safety mothers were placed on a diet of 15% chili pepper and 85% powdered chow. Pilot studies indicated that rats would maintain themselves and grow normally on this diet as the only source of food, after a brief rejection period lasting, at most, a few days. We wished to have the mothers adjusted to the diet before fertilization. All pups were weaned and marked at 30 days of age and placed in cages with their littermates. They were transferred to individual cages a few days before testing periods. Exposure rats were offered only the 15% chili pepper diet, except in choice tests, through the 11-mo duration of the experiment. Thus, except for mother's milk, they had no other experience of hot chow. They may conceivably have experienced “hot” milk, if any of the capsaicin (the source of piquancy) from the chili appeared in the milk. Safety rats had a similar experience except for the addition of one or two periods of 1-2 wk each during which they received the safety treatments described below. Controls were raised on powdered chow without chili and experienced chili only during choice tests.

At 8 wk of age, somewhat more than half of the rats in each group experienced a 4-day preference test between powdered chow and the same chow with 15% chili pepper. After this test, these rats were returned to group cages with other members of their group. All animals were tested at 7 mo of age (see Table 1 for a plan of the experiment). The 7-mo test was like the 8-wk test except that it included all the rats, some that were experiencing chili (or plain powdered chow) for the first time and others that had had the choice for 4 days at the age of 8 wk. After the 7-mo test, rats were returned to the group cages and maintained on their original diets. Some participated in other preference tests or observational studies, some of which are described below. At 11 mo of age, seven control, four exposure, and four safety rats were retested on a 6-day test of preference between powdered chow and the same with 1% chili pepper. This much lower concentration was clearly different from plain chow to the human palate but was only slightly piquant. The 15 rats retested with the lower concentration experienced, at most, 10 days with diets other than their basic rearing diet in the period between 7 and 11 mo.

The safety groups were treated identically to the exposure groups, except for periods of safety training before the preference tests at 8 wk (if administered) and 7 mo (see Table 1). The first safety period occupied Days 38-46 for the seven animals that were tested at 8 wk of age. The second series was given during Days 185-198 to all 11 animals prior to the 7-mo preference test. The basic idea of the chili-safety experience was to give slightly food-deprived animals experience with a number of distinctively different novel diets. Those containing chili led to normal repletion, whereas those without it were followed by LiCl-induced illness. The second safety-training sequence (Days 185-198) is described below in some detail, and variations from it in the first sequence (Days 38-46) are mentioned.

Three entirely new diets were employed. Each diet came in two forms: 15% chili added or “plain.” The chili diet always contained 1% NaCl (a taste control for LiCl), and the nonchili diet contained 1% LiCl, which reliably produced learned taste aversions at this level. The diets can be briefly described by their most distinctive ingredients, as bouillon, Crisco, and buttermilk.

For each type of diet, the procedure lasted 4 days. Rats were placed on a food-deprivation cycle that allowed them 1-hr access to food twice each day, approximately 6 hr apart. On the first day of the cycle, they were offered a new diet with 15% chili. On the second exposure that day, the alternate nonchili and poisoned diet was offered. On the second day, recovery was permitted, while the standard 15% chili chow was available during both feeding periods. Day 3 was a training session equivalent to Day 1, but with the order reversed: The LiCl version of the same new diet was offered in the first meal of the day, and the nonchili version later in the day. Day 4 was a recovery day, like Day 2, and completed the cycle. Food intakes were measured to the nearest .5 g on all training days. This 4-day cycle was carried out in succession for each of three diets in the order: buttermilk, Crisco, bouillon. The rats were returned to the 15% chili diet for a few days until the subsequent preference test.

The first safety training procedure for the seven rats tested at 8 wk was identical to the procedure described above except that recovery days were not inserted between each of the training days. As a result, in some cases rats did not ingest much of the safe chili diet, or the LiCl diet, on the last of the three new diets, on account of an apparent accumulation of the effects of LiCl poisoning. Thus, a few of the rats in the first safety training cycle may have received four or five effective poisoning-safety experiences instead of six. For this reason, the recovery days were inserted in the second round of safety-poisoning experiences. Overall, then, seven safety rats received two safety sequences, and four received only the second sequence.

The diets were constituted as follows: Bouillon diet—15% chicken bouillon, 35% powdered milk, 35% corn oil, 15% sucrose; Crisco diet—40% Crisco, 50% wheat flour, 10% sucrose; buttermilk diet—100% buttermilk.
Table 1
Plan of Experiment 1

<table>
<thead>
<tr>
<th>Group</th>
<th>Age (in wk)</th>
<th>Control</th>
<th>Exposure</th>
<th>Safety</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>chow</td>
<td>15% chili chow</td>
<td>15% chili chow</td>
</tr>
<tr>
<td></td>
<td></td>
<td>15% chili chow</td>
<td>(12 safety-poison trials*)</td>
<td></td>
</tr>
<tr>
<td>0-6</td>
<td></td>
<td>chow</td>
<td>15% chili chow</td>
<td>15% chili chow</td>
</tr>
<tr>
<td></td>
<td></td>
<td>chow</td>
<td>(Preference tests: chow vs. 15% chili chow [4 days]*)</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td></td>
<td>chow</td>
<td>15% chili chow</td>
<td>15% chili chow</td>
</tr>
<tr>
<td>8</td>
<td></td>
<td>chow</td>
<td>15% chili chow</td>
<td>15% chili chow</td>
</tr>
<tr>
<td>9-26</td>
<td></td>
<td>chow</td>
<td>15% chili chow</td>
<td>15% chili chow</td>
</tr>
<tr>
<td>27-28</td>
<td></td>
<td>chow</td>
<td>15% chili chow</td>
<td>15% chili chow</td>
</tr>
<tr>
<td>29</td>
<td></td>
<td>chow</td>
<td>15% chili chow</td>
<td>15% chili chow</td>
</tr>
<tr>
<td>30-46</td>
<td></td>
<td>chow</td>
<td>15% chili chow</td>
<td>15% chili chow</td>
</tr>
<tr>
<td>47</td>
<td></td>
<td>chow</td>
<td>15% chili chow</td>
<td>15% chili chow</td>
</tr>
</tbody>
</table>

* Animals first tested at 7 mo (Week 29) remained on their basal diets through Weeks 7 and 8.

Results

Exposure rats grew well on the 15% chili diet. Their weights lagged a little behind those of controls from before the time of weaning (see Table 2). By 10 mo of age, 3 of the 13 control rats had died, as against 1 of 11 exposure rats. These results speak against significant toxicity of chili pepper (as does the existence of over 60 million Mexicans). The total of 6 or 12 poisoning experiences did not have a marked effect on the health of the safety animals, even though coupled with virtually exclusive exposure to the 15% chili diet (Table 2). Only 1 of the 11 safety rats died by 10 mo of age.

The LiCl was clearly effective in both poisoning-safety cycles, as judged by depressed food intake on both the first and second day of exposure to each LiCl diet.

Because of the imposed recovery days in the second cycle, the effect was clearer here. The effectiveness of the procedure is most obvious in the increased relative avoidance of the nonchili diet the second time it was presented (Day 3 vs. Day 1 of the cycle for each novel diet). For the first (buttermilk) diet, 33% of the food on the first training day was from the nonchili diet, and only 8% on the second training day. For the Crisco diet, the corresponding numbers are 49% for the first trial and 19% for Day 3, and for the bouillon diet, the figures are 66% and 16%, respectively. Although an aversion is clearly established to the nonchili version of each diet, these data do not provide direct evidence that the rats were gradually learning to avoid any diet not containing chili and/or to treat chili as a safety signal.

The 8-wk preference test (Table 3) revealed a strong avoidance of the chili diet by

Table 2
Growth of Rats in the Three Groups of Experiment 1

<table>
<thead>
<tr>
<th>Group</th>
<th>Male</th>
<th>n</th>
<th>Age: 1 mo</th>
<th>Age: 10 mo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>6</td>
<td>78.7</td>
<td>463</td>
<td></td>
</tr>
<tr>
<td>Exposure</td>
<td>5</td>
<td>71.4</td>
<td>407</td>
<td></td>
</tr>
<tr>
<td>Safety</td>
<td>8</td>
<td>62.2</td>
<td>387</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>7</td>
<td>68.4</td>
<td>303</td>
<td></td>
</tr>
<tr>
<td>Exposure</td>
<td>6</td>
<td>58.6</td>
<td>229</td>
<td></td>
</tr>
<tr>
<td>Safety</td>
<td>3</td>
<td>64.0</td>
<td>262</td>
<td></td>
</tr>
</tbody>
</table>

Table 3
Eight-Week Preference Test: 15% Chili Diet Versus Powdered Chow

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Day 1</th>
<th>Days 3 and 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>6</td>
<td>1.0</td>
<td>.1</td>
</tr>
<tr>
<td>Exposure</td>
<td>7</td>
<td>37.1</td>
<td>* * * .6 * *</td>
</tr>
<tr>
<td>Safety</td>
<td>7</td>
<td>40.4</td>
<td>13.1 * *</td>
</tr>
</tbody>
</table>

Note. All significance estimates are based on a Mann-Whitney U test (one-tailed).

* Mean intake per day for Days 3 and 4.
* p < .05. ** p < .01. *** p < .001.
all groups. The greater acceptance of this diet on Day 1 by exposure and safety groups may be entirely attributable to a neophobic reaction to the plain chow, which these groups had never experienced before. However, in spite of this, on their first day with this new diet, these rats still consumed less than 50% of their food in the form of the chili diet. By Days 3 and 4, there was no significant difference between exposure and control rats (Table 3). However, there is some evidence for attenuation of the aversion of the safety group (Table 3), although they still showed a substantial aversion to chili (13% of the total eaten in the last 2 days was from the chili diet).

The 7-mo test included both these 8-wk animals and those not tested at 8 wk. The data from both of these subgroups were combined to evaluate the results, into three major groups: control, exposure, and safety. As indicated in Table 4 and Figure 1, there is a strong chili aversion in all three groups and a virtually identical avoidance of the chili diet after the first day. On the first day, exposure and safety rats consumed more chili diet than did controls, an effect probably attributable to neophobia.

In the final retest at 11 mo (Table 5), with a much lower concentration of chili, the same pattern of results is seen. There was a higher intake of the slightly piquant diet in exposure and safety rats on Day 1, though not an absolute preference for it. The exposure rats avoided this diet strongly (8%) and at roughly the same level as controls by Days 5 and 6, the last 2 days of testing. As with the 8-wk data, there was a strong but significantly weaker aversion in the safety animals at Days 5 and 6 of testing. In general, exposure to chili for 8 wk to 11 mo had no effect on chili preference after 1 day of choice. Exposure plus 6–12 safety-poison experiences led to a significantly reduced aversion in two of the three tests. However, in all three tests, the safety group showed a substantial aversion to the chili diet at the end of the testing periods.

Experiment 2: Effects of the Gradual Introduction of Chili

The exposure procedures used in Experiment 1 do not parallel the exposure human children normally receive in chili-eating cultures, because these children are exposed to gradually increasing amounts of chili (Rozin,
1978; Rozin & Schiller, Note 1). In this experiment, we incorporated this feature in the rearing of a group of rats. They were reared on very low levels of chili pepper and were subjected to gradually increasing concentrations.

**Method**

Fourteen rats, born in the laboratory, were weaned at 28 days of age. Powdered Purina Rat Chow with 1% chili pepper was the only food available to the mother and to the offspring. The rats were divided at weaning into two groups of seven rats each. The rats of each group were individually marked and housed as separate groups in two community cages. The constant group received 1% chili diet until testing began on Day 60. The gradual group received 1% chili diet for their first 6 days postweaning and then an increased chili concentration every 6 days, covering the values of 2%, 4%, 10%, and 15%. Two days prior to testing, on Day 58, all animals were placed in individual cages with two cups of the appropriate diet (1% chili for the constant group, 15% chili for the gradual group). On Day 60, a 6-day alternating side preference test was begun, with a choice between 1% chili and plain ground chow. Note that the chili concentration used in the test was much lower than the highest level experienced by the gradual group.

**Results**

As can be seen in Table 6, neither group showed evidence of a preference for chili, nor was there a significant difference between them at any time during the 6-day tests. There was, as we have seen before, a much weaker avoidance than in control animals (from Experiment 1, 11-mo test with 1% chili) on the first day. For the last 2 days of the 6-day test, gradual and constant groups showed slightly weaker avoidances than controls. All groups showed a strong aversion to a mildly piquant diet, with a significantly weaker aversion in both groups previously exposed to chili. The absence of any difference between gradual and constant groups is particularly striking when it is realized that the gradual groups came down from a 15% chili diet to the 1% chili choice whereas the constant group had never had a diet with more than 1% chili.

**Experiment 3: Association of Positive Visceral Effects With Chili Ingestion**

The most straightforward method for increasing a preference for a substance would be to follow its ingestion by positive visceral effects. One could claim that this is exactly what occurs each time the rat on an exposure regimen eats a meal of chili diet. However, it might be that the repletion effects are not sufficiently salient and/or that rats eat meals before they experience significant hunger and hence experience few direct positive visceral effects. In this experiment, a concerted attempt was made to induce a positive preference for chili pepper by associating it with recovery from thiamine deficiency, a visceral event that has been successfully used to increase food preferences (Garcia, Ervin, Yorke, & Koelling, 1967; Zahorik, 1977; Zahorik, Maier, & Pies, 1974). Although recovery from deficiency does not correspond to any obvious recurring event that can be assigned to most human chili eaters, the occurrence of significant postingestive effects in these cases cannot be ruled out.

**Method**

Four male and eight female rats, aged 90 days, were randomly assigned to one of two basic groups: recovery and control. The basic design, as initially described by Rozin and Kalat (1971) and first successfully employed by Zahorik et al. (1974), involved the use of three distinctively different diets: safe, deficient, and recovery. The critical test for preference acquisition was between the recovery and the safe diets. The critical test for chili preference (chili being one feature of the distinctive recovery diet) was a choice between two diets that were identical except that one contained chili.
Table 7
Effects of Association of Chili Pepper With Recovery From Deficiency

<table>
<thead>
<tr>
<th>Test No.</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
</tr>
</thead>
<tbody>
<tr>
<td>Choice of diets</td>
<td>Rc-S</td>
<td>Rc-S</td>
<td>Rc-S</td>
<td>Rc-R</td>
<td>Pc-P</td>
<td>Sc-S</td>
<td>Rc-S</td>
<td>Rc-R</td>
<td>Sc-S</td>
<td>Rc-S</td>
<td>Sc-S</td>
<td>Re-R</td>
</tr>
<tr>
<td>Test length</td>
<td>1 d</td>
<td>4 h</td>
<td>4 d</td>
<td>2 d</td>
<td>2 d</td>
<td>2 d</td>
<td>2 d</td>
<td>2 d</td>
<td>2 d</td>
<td>1 h</td>
<td>1 h</td>
<td>1 h</td>
</tr>
<tr>
<td>State</td>
<td>rec</td>
<td>def</td>
<td>rec</td>
<td>rec</td>
<td>rec</td>
<td>rec</td>
<td>rec</td>
<td>rec</td>
<td>rec</td>
<td>def</td>
<td>def</td>
<td>def</td>
</tr>
<tr>
<td>Recovery group</td>
<td>54</td>
<td>75</td>
<td>64</td>
<td>34</td>
<td>24</td>
<td>34</td>
<td>29</td>
<td>14</td>
<td>24</td>
<td>46b</td>
<td>34b</td>
<td>36b</td>
</tr>
<tr>
<td>Control group</td>
<td>19</td>
<td>22</td>
<td>17</td>
<td>13</td>
<td>10</td>
<td>5</td>
<td>8</td>
<td>3</td>
<td>6</td>
<td>36</td>
<td>6</td>
<td>17</td>
</tr>
<tr>
<td>Significance</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>***</td>
<td>*</td>
<td>*</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Best rat</td>
<td>89</td>
<td>89</td>
<td>99</td>
<td>20</td>
<td>52</td>
<td>75</td>
<td>47</td>
<td>18</td>
<td>46</td>
<td>77</td>
<td>95</td>
<td>88</td>
</tr>
</tbody>
</table>

Note. A 2-mo break followed Test 6. R = recovery; S = safe; P = powdered chow; lower case c indicates containing chili; d = days; h = hours; rec = recovered; def = deficient. All significance estimates are based on a Mann-Whitney U test (one-tailed).

The diets used in this experiment were all thiamine deficient, with the exception of the first one. Testing was essential, the latter part of the experiment. For control and recovery rats, the deficient diet was always a powdered, starch-based diet. For half of the rats in each group, the safe diet was an equal palatability, when the recovery diet was without chili pepper. For the other half of the rats in each group, the situation was reversed. The safe diet was the Crisco-based diet, and the recovery diet was the sugar-based diet with 2% chili pepper added (see Footnote 1). For all the rats in each group, the safe diet was a granular, sugary diet and the recovery diet was a Crisco-based bland diet with 2% chili pepper added (see Footnote 1).

In the first stage of the experiment, all rats received 7 days of exposure to the appropriate safe diet. Since this diet was thiamine deficient, there was the possibility that over this 7-day period, some subtle signs of thiamine deficiency might appear (though no overt signs appear for over 2 wk). As assurance against this possibility, each rat was injected with 100 µg of thiamine HC1 on Day 3 of this regimen.

The second stage of the experiment consisted of a series of seven deficiency-recovery cycles. Recovery rats were placed on the deficient diet and continued on this regimen until all rats in the group had lost weight for 2 consecutive days. When this criterion was reached, these rats were all offered the recovery diet in a single cup (alternated in side from one cycle to the next). Ten minutes after this introduction, each rat was injected sc with thiamine HC1 at a dose level of 200 µg/kg. Rats were allowed access to the recovery diet for the following 24 hr. They were then returned to diet D and entered the next deficiency-recovery cycle. On at least one occasion during each deficiency period, the rats were injected sc with approximately .5 ml of water so that they could not learn to associate the injection procedure with recovery. Control rats received the same diet sequence as recovery rats but were not allowed to become thiamine deficient. They were injected with 100 µg of thiamine three times every week. At the same time that recovery rats qualified for a recovery trial, the control rats were offered the same recovery diet and were injected with thiamine in corresponding doses 10 min after this diet was made available. This cycle continued six times.

The third stage involved preference testing. Preference testing might have been ideally carried out when the animals were deficient, since it was in this state that the chili diet produced recovery. However, a preference test in this situation would also be an extinction trial, since thiamine could not be injected during the test (it might follow ingestion of diet S). For this reason, one completely “clean” preference test was carried out after the rats had recovered from the sixth deficiency period. The day following recovery, rats were offered a 24-hr choice between diet Re and diet S. They were then allowed to become deficient again (the seventh standard cycle) on diet D. However, when they reached the deficiency criterion, they were given a 4-hr choice, while deficient, between diet Re and diet S. They were then returned to diet D for the remainder of the following 24-hr period, and they experienced a standard recovery procedure on the next day, which constituted the seventh recovery trial.

Note. A 2-mo break followed Test 6. R = recovery; S = safe; P = powdered chow; lower case c indicates containing chili; d = days; h = hours; rec = recovered; def = deficient. All significance estimates are based on a Mann-Whitney U test (one-tailed).

The diets used in this experiment were all thiamine deficient, with the exception of the first one. Testing was essential, the latter part of the experiment. For control and recovery rats, the deficient diet was always a powdered, starch-based diet. For half of the rats in each group, the safe diet was a granular, sugary diet and the recovery diet was a Crisco-based bland diet with 2% chili pepper added (see Footnote 1). For all the rats in each group, the situation was reversed. The safe diet was the Crisco-based diet, and the recovery diet was the sugar-based diet with 2% chili pepper. The two critical diets were approximately equal in palatability, when the recovery diet was without chili pepper.

The first stage of the experiment, all rats received 7 days of exposure to the appropriate safe diet. Since this diet was thiamine deficient, there was the possibility that over this 7-day period, some subtle signs of thiamine deficiency might appear (though no overt signs appear for over 2 wk). As assurance against this possibility, each rat was injected with 100 µg of thiamine HC1 on Day 3 of this regimen.

The second stage of the experiment consisted of a series of seven deficiency-recovery cycles. Recovery rats were placed on the deficient diet and continued on this regimen until all rats in the group had lost weight for 2 consecutive days. When this criterion was reached, these rats were all offered the recovery diet in a single cup (alternated in side from one cycle to the next). Ten minutes after this introduction, each rat was injected sc with thiamine HC1 at a dose level of 200 µg/kg. Rats were allowed access to the recovery diet for the following 24 hr. They were then returned to diet D and entered the next deficiency-recovery cycle. On at least one occasion during each deficiency period, the rats were injected sc with approximately .5 ml of water so that they could not learn to associate the injection procedure with recovery. Control rats received the same diet sequence as recovery rats but were not allowed to become thiamine deficient. They were injected with 100 µg of thiamine three times every week. At the same time that recovery rats qualified for a recovery trial, the control rats were offered the same recovery diet and were injected with thiamine in corresponding doses 10 min after this diet was made available. This cycle continued six times.

The third stage involved preference testing. Preference testing might have been ideally carried out when the animals were deficient, since it was in this state that the chili diet produced recovery. However, a preference test in this situation would also be an extinction trial, since thiamine could not be injected during the test (it might follow ingestion of diet S). For this reason, one completely “clean” preference test was carried out after the rats had recovered from the sixth deficiency period. The day following recovery, rats were offered a 24-hr choice between diet Re and diet S. They were then allowed to become deficient again (the seventh standard cycle) on diet D. However, when they reached the deficiency criterion, they were given a 4-hr choice, while deficient, between diet Re and diet S. They were then returned to diet D for the remainder of the following 24-hr period, and they experienced a standard recovery procedure on the next day, which constituted the seventh recovery trial.

---

3 The basal deficient diet contained, in grams per kilogram, sucrose, 20; starch, 600; Mazola, 50; casein, 100; egg-white solids, 189; Hegsted salt mix, 40; General Biochemicals thiamine-deficient vitamin mix, 1. Both other diets contained the same levels of salt and vitamin mix. In addition, the sugar-based diet contained sucrose, 659; Mazola, 50; casein, 250. The Crisco-based bland diet contained sucrose, 20; starch, 434; Mazola, 5; Crisco, 250; casein, 250.

4 In the first cycle a lower chili pepper level of .5% was used on the recovery day. This was increased to 1% in the second cycle and to 2% in the third and all subsequent cycles.
Following this seventh cycle, the rats were extensively tested for diet preferences, while recovered. During this period, all rats were injected sc with 100 µg of thiamine three times each week. The sequence of preference tests is indicated in Table 7. All tests involved a choice between a chili-free diet and a diet with 2% chili. The first test was a 4-day choice between diet Rc and diet S. This was followed by a 2-day choice between the recovery diet vehicle (R) and the same diet with chili added (Rc) to determine the extent to which the taste of chili per se, as opposed to other properties of the recovery diet, acquired positive value. The palatability of chili was further tested in 2-day choices between powdered chow (P) and the same with 2% chili pepper (Pc), and then diet S with (Sc) or without (S) chili pepper. Following these tests, the rats were kept on Purina Checkers for 2 mo. They were then retested on three of these tests: Rc versus S, Rc versus R, and Sc versus S. Finally, all rats were placed back on diet D. Recovery rats were allowed to become thiamine deficient, while control rats were injected (as before) with thiamine. When the recovery rats met the deficiency criterion, all rats were given 1-hr preference tests on 3 successive days between the same three basic choices: Rc versus S, Rc versus R, and Sc versus S. The recovery rats were thiamine deficient during all three brief tests.

Results

The deficiency–recovery cycles were effective in every case. Every recovery rat showed a large weight gain on every recovery day. The cycles on diet D varied from 5 to 7 days. Because experiments of this type have shown weak, if any, effects, we intentionally used as a primary test, pairings of safety and recovery diets that differed along many sensory dimensions, only one of which was chili pepper. On this test, whether rats were briefly tested while deficient or for periods of a day or more while recovered, there was a substantial and significant increase in preference for diet Rc (see Table 7 for these and all other results). On all three trials with this pairing of diets, before the 2-mo break in the experiment, average intake of the recovery diet was above 50% of total intake, which suggests an actual preference. This effect was clearest in the deficient situation (75% Rc preference in recovery rats vs. 22% in controls). This is one of the largest effects observed in the acquired preference literature: Its size is enhanced because of the unpalatability of diet Rc, and hence the low intake of this diet in controls.

Subsequent testing in recovered rats isolated the effects of chili per se, pairing identical diets except for the presence or absence of chili. Results were similar in all three tests in the initial round of testing (Tests 4, 5, and 6 in Table 7): There was an attenuation of the aversion to the chili diet but far from an absolute preference. This effect was notably weaker than the Rc–S basic choice and suggests that much of the enhanced preference in the basic tests is related to the nonchili distinctive components of the recovery diet. Retesting after 2 mo (Tests 7–12, Table 7) revealed the same pattern of results but with a further attenuation of chili-diet preferences in all tests. As before, the basic choice (Rc vs. S) produced the greatest attenuation in aversion to chili, although not an absolute preference (29%). Testing during deficiency (Tests 10–12) enhanced the preferences or, more correctly, further attenuated the chili aversion in the recovery group. Although most statistical tests for recovery-control differences (Mann-Whitney U, one-tailed) do not yield acceptable significance in the 2-mo retests, all six tests show a superiority of at least 10 percentage points for the recovered group.

Variability was high in both recovery and control groups. Table 7 shows the data from the recovery animal having the strongest acquired preference for chili. Note that under deficiency conditions some months after training, this rat shows an absolute preference for the chili choice, even when the pairing involved only a difference in chili (Rc vs. R or Sc vs. S).

In summary, (a) seven pairings of chili diet with recovery from thiamine deficiency attenuated the aversion to chili pepper; (b) this attenuation was even stronger when testing occurred during a deficiency state, when the recovery properties of the chili diet would be expected to be manifested; (c) a major portion of the enhanced preference effect is attributable to distinctive aspects of the recovery diet other than chili; and (d) one animal showed an absolute preference for chili when in the deficient state.

We conclude that it is possible to significantly weaken a chili aversion. A positive preference may appear when the animal is in the negative physiological state that chili
diet has, in the past, alleviated. It is quite likely that longer experiments, involving tens or hundreds of recovery cycles, could produce more stable, absolute acquired preferences. The effect might be further enhanced by limiting the difference between recovery and safe diets to merely the presence of chili in the former. The question remains whether these predicted preferences, or the more modest preferences actually demonstrated here, represent hedonic shifts or medicine effects. The considerable enhancement of preference during deficiency suggests a medicine-effect component.

Experiment 4: Effects of Long Exposure to Chili on Sensitivity to Chili

It would be surprising if up to 1 yr of ingestion of food containing significant amounts of a chemical as potent as capsaicin should have no measurable effects (Jancsó-Gábor & Szolcsányi, 1969; Maga, 1975). On the basis of estimates from the literature (Osol & Farrar, 1955), these rats were consuming, based on daily intakes of 20 g (and assuming a capsaicin level of from .15 to 1.5 mg/g of diet), 3–30 mg/day of capsaicin. Since this diet was comparable in hotness to chili-flavored foods in chili-eating countries, it can be assumed that these rats were consuming more chili (per kilogram) than humans do.

Capsaicin has significant effects on chemical irritant sensitivity in rats and humans (Jancsó, 1960; Jancsó-Gábor & Szolcsanyi, 1969). Work from the laboratory of Jancsó and Jancsó-Gábor in Hungary indicates that topical applications of solutions containing capsaicin in the range of .5%–1% to rat or human skin or tongue results in a desensitization that lasts more than a day, as measured by markedly raised thresholds to several different chemical irritants. These concentrations are 5–60 times higher than the 15% chili diet used here, but exposure was over much greater periods in our studies. Furthermore, systemic injections of as little as 50 mg/kg capsaicin into a rat can totally desensitize it to all chemical irritant substances for weeks or months. These rats showed no response to strong irritants, such as capsaicin, mustard oil, or ammonia, placed on their eyes or ears. The rats in this experiment were consuming capsaicin at the rate of about 9–90 mg/kg/day over hundreds of days. Of course, we do not know how much is absorbed, but it is, on the face of it, surprising that so little change in avoidance is seen. One might well expect a loss of sensitivity, as one might well expect to see the same in humans living on chili diets. Indeed, loss of sensitivity to the irritant effects of chili is a possible partial explanation for the development of chili preference in humans.

In this experiment, sensitivity to chili was studied in terms of avoidance threshold: the lowest chili concentration that a rat would avoid, in comparison with the same diet without chili. These data provided suggestive data on desensitization and a very sensitive test for preference changes that chili exposure might produce. Rats from Experiment 1, raised on 15% chili for at least 10 mo, were compared with rats of about the same age that had never experienced chili pepper.

**Method**

Twelve rats from Experiment 1, 11 mo of age, served as experimental subjects. With the exception of participation in a few preference tests, lasting less than a total of 20 days, during which they had access to non-chili-flavored diets, the rats had spent their entire postweaning life on a diet of 15% chili pepper in powdered chow. Twelve rats of approximately the same age (6–12 mo) were taken from the rat colony and used as controls. These rats had been raised on Purina Checkers. One experimental rat died during the experiment. All rats were housed individually and tested in the standard two-cup choice situation.

The basic unit of preference in this study was the 2-day choice between two diets, with intakes measured daily and cup position switched daily. Rats were tested on a staircase method, with a choice between plain powdered chow and the same diet with a measured amount of chili pepper. If a rat avoided the chili diet (defined as less than 40% of total intake from the chili choice), the concentration of chili was halved for the next 2-day cycle, and this procedure was continued until an indifference point was reached. An indifference point was defined as the higher of two consecutive concentrations at which the rat consumed at least 40% of the chili diet. The avoidance threshold was then set as the lowest concentration above the indifference point. Thus if a rat consumed 25% of the chili diet at 4 mg chili/g, 44% at 2 mg/g, and 43% at 1 mg/g, 4 mg/g would be taken as the avoidance threshold. (In a few instances
REVERSAL OF INNATE AVersions

a rat would eat more than 40% at one concentration and
dip below this at the next lowest. In these few cases we
continued to drop the concentration until the criterion
of two successive concentrations above 40% was met.)

All animals began with a choice between pure pow-
dered chow and chow with 4 mg/g chili pepper. In 6 of
23 cases, rats did not avoid this concentration. Under
these circumstances, on the next 2 days the concen-
tration of chili was doubled, instead of halved, and this was
continued until the preference dropped below 40% for
two consecutive 2-day periods. The avoidance
threshold was then defined, as before, as the lowest
concentration avoided above the indifference point.
Avoidance thresholds were determined for 11 experi-
mental and 12 control rats.

Results

The avoidance thresholds for the 23
subjects are graphically displayed in Figure
2. The distributions of chili-naive and
chili-exposed rats overlap considerably.
Four chili-naive rats show by far the lowest
avoidance thresholds, but the other eight are
indistinguishable from the 11-mo-exposed
animals. Overall, the distributions do not
differ significantly (Mann-Whitney
U = 54, p > .05, one-tailed). The geometric mean
avoidance threshold for the exposed groups
was 1.55 mg/g and .27 mg/g for the naive
group. Insofar as avoidance thresholds can
be taken to be a measure of sensitivity, the
evidence for desensitization here is weak.

Experiment 5: Capsaicin Desensitization
and Chili Preference

It is presumed that rats find chili aversive
because of the piquant effects of capsaicin.
These effects are mediated by the chemical
irritant sense. If this is so, then densitiza-
tion to the sensory effects of capsaicin should
eliminate the chili aversion; in some sense,
the chili diet would then taste like chow
flavored with mild paprika. This experi-
ment tested this presupposition.

Method

Six 55-day-old rats served as subjects. They were
housed in individual cages. For the first 8 days of the
experiment, each received sc a daily, increasing dose of
capsaicin. In accord with the procedure of Jancsó
(1960), .1 g of capsaicin was dissolved in 2–3 drops of
ethanol and about 20 drops of Tween 80. Water was
then added to make 10 ml of injection solution. Over
a period of 8 days, rats were injected with the following
sequence of capsaicin doses (in milligrams per kilo-
gram): 24, 31, 31, 48, 65, 80, 100, and 120. Five minutes
before each dose, .7 ml of atropine sulfate solution (.54
mg/ml) was administered to counter some of the acute
effects of capsaicin. On Day 9, the animals received no
no treatments. On Day 10, they were tested for the ef-
effectiveness of desensitization: A drop of 1% ammonia
was placed in one eye. None of the six capsaicin-treated
animals showed any paw-wiping response to this stim-

Figure 2. Chili pepper avoidance threshold for 11
animals raised on 15% chili pepper for 11 mo and for 12
chili-naive controls. (Each point represents the
threshold for one rat.)
Table 8
Preference for Chili in Capsaicin-Desensitized Rats

<table>
<thead>
<tr>
<th>Diet choice</th>
<th>Desensitized rat (n = 6)</th>
<th>Control rat</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Preferencea</td>
<td>n</td>
</tr>
<tr>
<td>Chow vs. 15% chili chow</td>
<td>35.8</td>
<td>13</td>
</tr>
<tr>
<td>Chow vs. 1% chili chow</td>
<td>65.7</td>
<td>7</td>
</tr>
<tr>
<td>Sugar diet vs. 15% chili sugar diet</td>
<td>44.5</td>
<td>6</td>
</tr>
<tr>
<td>Sugar diet vs. 1% chili sugar diet</td>
<td>53.2</td>
<td></td>
</tr>
</tbody>
</table>

Note. Experiment 3 controls faced a choice between 2% chili in the sugar diet and plain sugar diet. The sugar diet contained, in grams per kilogram, sucrose, 659; Mazola, 50; casein, 250; Hegsted salt mix, 40; General Biochemicals thiamine-deficient vitamin mix, 1.

a Mean percentage of chili diet eaten over 2 days.

Discussion

One cannot fail to be impressed by the resistance shown by laboratory rats to the acquisition of a preference for chili pepper. This contrasts to the apparently effortless acquisition of this preference by tens of millions of humans each year by a process that seems little more than “mere exposure.” Mere exposure over 11 mo, or this same exposure coupled with poisoning and safety experiences, did almost nothing to attenuate the innate aversion in rats, let alone establish a preference. Simulation of the human experience of gradual introduction led to equally negative results. Explanation of human preferences as resulting, in large part, from desensitization to the irritant effects of chili pepper was not supported here. Eleven months on a piquant diet produced little change in the rat’s chili-avoidance threshold.

In contrast, two procedures did significantly modify or eliminate chili aversions. One was desensitization with systemic injections of capsaicin. When the sensory irritant response to chili is abolished, the aversion disappears. This result confirms the unpalatability of the irritant properties of chili pepper and establishes them as sufficient to account for chili aversion. However, there is little evidence that such a desensitization actually occurs consequent upon ingestion of chili in concentrations typically consumed by humans. The second effective procedure was a series of seven experiences of recovery from illness following chili ingestion. This might, if extended, lead to an absolute preference. Although the findings reported here are among the most substantial in the learned-preference literature, they can still be taken to support the difficulty in establishing learned preferences in rats, and the major asymmetry between the strong and rapidly acquired learned aversions and learned preferences. The positive findings reported here do not speak directly to the fundamental distinction be-
between hedonic shifts and medicine effects. Given that the major change was produced using recovery effects and that the smaller poison-safety effect was based on manipulation of the consequences of chili ingestion, it is reasonable to guess that the preference shifts shown were motivated by anticipated consequences (medicine effects). This is further supported by the marked enhancement of chili preference during the recovery experiment, when testing was carried out in the deficiency state that chili had in the past relieved.

Both the safety-poison and the recovery procedures were carried out a rather small number of times when viewed in the context of a lifetime of experiences (but a large number of times in comparison with those in other laboratory studies on preferences and aversions). If such experiences did play a role in human preference, it might reasonably be proposed that tens or hundreds of such experiences preceded preference acquisition, so that the results reported here might indicate an adequate mechanism. However, both poison-safety and recovery procedures seem quite artificial with respect to the actual experience of the human chili user (Rozin, 1978; Rozin & Schiller, Note 1). The mere exposure and gradual exposure treatments, which gave no promising results, are much more in line with the human experience.

The data presented do argue that under laboratory conditions with domesticated rats, it would be difficult to establish a model for human chili preference. However, one cannot jump from these data to other species or other circumstances. It might be quite possible to establish such preferences in primates or other domesticated animals, such as dogs. Furthermore, the laboratory experiences provided here are a far cry from the kinds of experiences that humans typically have with chili pepper. It is introduced and eaten in social settings, in conjunction with an elaborated cuisine. Perhaps a more appropriate test of the hypothesis that chili pepper and similar preferences are almost uniquely human would be to study domesticated animals, such as dogs, that are raised around the home in chili-eating cultures and consume chili-flavored food as table left-overs. Such a study is presently underway in Mexico (Rozin).

The resistance to acquisition of a chili pepper preference parallels data on laboratory rats for alcohol and a variety of bitter compounds, and it contrasts with strong human preferences for chili, alcohol, coffee, and other innately unpalatable substances. The problem remains as to why such a "simple" preference acquisition should be so closely limited to humans. Perhaps the correct strategy would be to study humans, because this is where the phenomenon resides. The use of chili pepper to explore this fundamental issue in preference and affect has much to recommend it. Unlike many of the other substances in the innately unpalatable category, chili pepper is safe and nonaddictive.

Reference Note


References


Hume, A. The islands of the Bay of Bengal. Stray Feathers, 1974, 2, 29–324.


Maga, J. A. Capsicum. CRC Critical Reviews of Food Science and Nutrition, 1975, 7, 177-199.


Richter, C. P. Alcohol as food. Quarterly Journal of Studies on Alcohol, 1941, 1, 650-662.


Received January 17, 1979