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Social Identification of Toxic Diets by Norway Rats (*Rattus norvegicus*)

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In the present experiment, a naive rat (an observer) (a) interacted with two conspecifics (demonstrators) that had recently eaten a diet unfamiliar to the observer, (b) ate two unfamiliar foods in succession, one of which was the food its demonstrators had eaten, (c) suffered toxicosis, and finally, (d) was offered a simultaneous choice between the two diets it had eaten prior to toxicosis induction. During the choice test, observers exhibited an aversion to that diet their respective demonstrators had not eaten. This result indicates that exposure of a rat to conspecifics that have eaten a diet can act, as does actual ingestion of a diet, to reduce that diet's subsequent associability with toxicosis. I discuss this finding as suggesting that interaction with conspecifics may provide an alternative to individual trial and error learning in identification of toxic foods by rats that ingest a number of novel foods in succession before becoming ill.

Free-living Norway rats, like other omnivores, must select a nutritionally adequate diet from among available foods, while avoiding repeated ingestion of any toxic substances they encounter while foraging (Rozin & Kalat, 1971). Identification and subsequent rejection of toxic foods would be relatively simple if rats ate discrete meals of each unfamiliar, potentially hazardous food they encountered and waited between meals of novel foods to independently evaluate the postingestional consequences of each (Rozin, 1969). Indeed, without such discrete sampling of unfamiliar foods, it is difficult to see how an individual rat could discover which of several novel foods it had eaten was toxic and should be rejected in future (Zahorik & Houpt, 1981).

It has been asserted frequently in the literature that rats take discrete meals of each of several novel foods presented to them. Unfortunately, evidence of such adaptive sampling of multiple unfamiliar foods in laboratory situations is not convincing. The data of Rozin's (1969) classic study indicate that rats eat several different novel foods in the first half hour those foods are available. Similarly, Barnett (1956, p. 30) found that when wild Norway rats (*R. norvegicus*) were presented with four novel foods, "it was usual for all four foods to be eaten within the first feeding period." Yet, if one rejects the hypothesis that rats in nature discretely sample those novel foods they encounter, one is left without a plausible explanation of how they might manage to identify toxic substances without repeated ingestion of them.

In natural circumstances, Norway rats are social animals (Calhoun, 1962; Telle, 1966), and each has access to information concerning the foods that others in its social group are eating (Galef, 1983; Galef & Wigmore, 1983; Posadas-Andrews & Roper, 1983). Knowledge of foods eaten by others could be useful to a rat in identifying toxic foods even if it had eaten several unfamiliar, potentially hazardous foods in a single feeding bout.

A new recruit to an established colony, a weanling pup or recent immigrant, could, with some confidence, assume that foods eaten by others were safe. If repeated ingestion of a toxic food present in the environment were truly life threatening, conspecifics living in the same area must be subsisting on safe, available alternatives. If environmental toxins were merely noxious rather than lethal, long-term residents would have had sufficient opportunity to learn to avoid ingestion of toxins, even if their individual strategies for identification of toxic substances were relatively inefficient. Thus, if a naive individual ingested a number of foods it had never before eaten and then became ill, it could benefit by acting as though the toxic agent resided in any food it had eaten that others of its social group had not. Foods being eaten by others are less likely sources of deleterious effects than those foods conspecifics are not eating.

The present experiment is a simplified laboratory analogue of a situation in which a rat, knowledgeable as to the foods others of its social group are eating, ingests several foods it has never before eaten and becomes ill. The experiment was undertaken to determine whether such a rat would form an aversion to an unfamiliar food that conspecifics had not eaten rather than to an unfamiliar food that conspecifics had eaten.

Method

Subjects

Forty-eight experimentally naive, 42-day-old, female Long-Evans rats obtained from Charles River, Canada (St. Constant, Quebec) served as observers. An additional ninety-six 56–63-day-old females from the McMaster colony served as demonstrators.

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Apparatus

Observers were housed individually throughout the experiment in 22 × 24 × 27.5 cm wire-mesh hanging cages. Demonstrators were housed individually in plastic shoe-box cages in a room separate from observers.

Procedure

Treatment of observers and demonstrators during the experiment was as follows:

Step 1. Observers and demonstrators were introduced into their respective cages and placed on a 23-hr food deprivation schedule, receiving powdered Purina Laboratory Rodent Chow for 1 hr/day for 2 days.

Step 2. Following a third 23-hr period of food deprivation, each demonstrator was offered, for 1 hr, a food cup containing either cocoa-flavored diet (Diet COC; 48 demonstrators) or cinnamon-flavored diet (Diet CIN; 48 demonstrators). (Diet COC = powdered Purina Laboratory Rodent Chow adulterated 2% by weight with Hershey's cocoa; Diet CIN = powdered Purina Laboratory Rodent Chow adulterated 1% by weight with McCormick's Pure Ground Cinnamon.)

Step 3. Immediately following feeding of each demonstrator, it was introduced into the cage of an observer and each demonstrator–observer pair was left undisturbed for ½ hr to interact freely. At the end of this first period of interaction, each demonstrator was removed from its observer's cage and replaced for ½ hr with a second demonstrator that had eaten the same diet as the first demonstrator. Thus each observer interacted for ½ hr with each of two demonstrators both of which had eaten either Diet CIN (24 observers) or Diet COC (24 observers).

Step 4. At the end of the 1-hr period of interaction, the second demonstrator was removed from each observer's cage and replaced with a weighed food cup containing either Diet CIN or Diet COC. This food cup was left in the observer's cage for 15 min. At the end of this first 15-min observer feeding period, the first food cup was removed and replaced with a second food cup containing the other diet (either Diet CIN or Diet COC).

Assignment of observers to groups was counterbalanced so that (a) equal numbers of observers interacted during Step 3 with demonstrators previously fed Diet CIN and with demonstrators previously fed Diet COC and (b) equal numbers of observers interacting during Step 3 with Diet-CIN-fed or Diet-COC-fed demonstrators were offered, during Step 4, foods in the order Diet CIN first, Diet COC second (CIN-COC exposure condition), or Diet COC first, Diet CIN second (COC-CIN exposure condition).

Because the experiment required each observer to sample both Diet COC and Diet CIN during Step 4, I discarded any observer (n = 6) failing to eat 0.5 g of each diet during the 15 min that diet was available.

Step 5. Immediately following termination of the second feeding period, each observer was injected ip with 1% of body weight 1% w/v LiCl solution.

Step 6. One hour following injection, pellets of Purina Laboratory Rodent Chow were placed in each observer's cage, and each was given 24 hr to recover from the effects of toxicosis.

Step 7. Following the 24-hr recovery period, each observer was offered, for 22 hr, a simultaneous choice of weighed samples of Diet CIN and Diet COC.

Step 8. At the end of the 22-hr test period, the experimenter determined each observer's intake of Diet CIN and Diet COC and calculated the percentage of Diet COC eaten by each observer.

Results

Figure 1 shows the amount of Diet COC, as a percentage of total amount ingested, eaten by observers in CIN-COC and COC-CIN exposure conditions during the 22-hr test. As can be seen in the figure, and as statistical tests confirmed (Mann-Whitney U tests: COC-CIN exposure group, $U = 1, p < .001$; CIN-COC exposure group, $U = 30$, $p < .01$), observers in both CIN-COC and COC-CIN exposure conditions formed robust aversions to that diet their respective demonstrators had not eaten. Prior interaction with demonstrators that had eaten a diet substantially reduced formation of an aversion to that diet.

As can also be seen in Figure 1, the effect of exposure to demonstrators was not symmetric across diets fed to demonstrators. All observers whose demonstrators had been fed Diet CIN developed an aversion to Diet COC; only half the observers fed Diet COC developed a strong aversion to Diet CIN. Either Diet CIN is less associable with toxicosis than Diet COC (perhaps because the former diet is less discriminable from the subjects' maintenance diet than the latter) or information concerning Diet CIN is communicated between rats more effectively than information concerning Diet COC (perhaps the olfactory cues associated with Diet CIN are stronger to rats (as they are to me) than those associated with Diet COC.

Discussion

It has long been known that previous ingestion of a diet attenuates learning of an aversion to that diet (Revusky & Bedarf, 1967). The present result indicates that exposure of a rat to conspecifics that have eaten a diet, can also attenuate learning of an aversion to the socially experienced diet. Thus diet-identifying cues experienced via social interaction act, as does ingestion of a food itself, to reduce the associability of a food with toxicosis.

Possible mechanisms underlying such familiarity effects on learned aversions have been discussed at length elsewhere (e.g., Kalat & Rozin, 1973; Siegel, 1974), and the present result casts little additional light on the role of the processes of latent inhibition or "learned safety" in diet acceptance. In the absence of a number of requisite control conditions in the present experiment, it is impossible to know precisely how preexposure to a demonstrator influenced subsequent aversion learning: (a) Exposure to demonstrators eating a given diet may have decreased subsequent associability of that diet with toxicosis, (b) Exposure to demonstrators eating a given diet may have increased baseline preference for that diet, or (c) both effects might occur. Although the reasons why interaction with demonstrators has the effect on subsequent aversion learning reported here need to be examined, they are irrelevant to the purposes of the present article, to inquire whether information acquired from conspecifics might aid naive rats in determining which of several novel foods they ate might be responsible for any toxicosis they subsequently experienced.

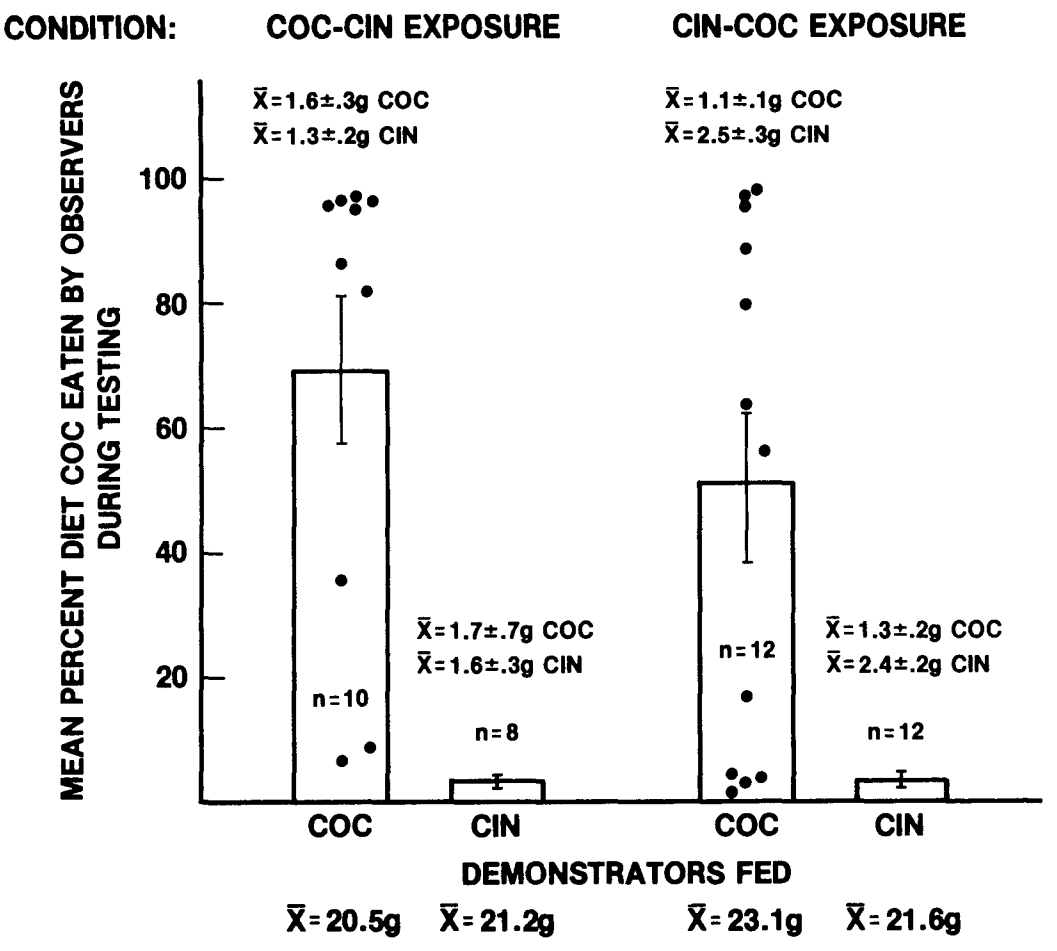

Figure 1. Mean amount of Diet COC eaten, as a percentage of total amount ingested by observers in COC-CIN and CIN-COC exposure conditions during testing, as a function of the diet fed their respective demonstrators. (Means and *SE* above histograms = amounts of Diet COC and Diet CIN eaten by observers in each group during Step 4 of procedure. Means below the abscissa = mean amount eaten by observers during the 22-hr test period [Step 7 of procedure]. COC = Diet COC, CIN = Diet CIN. Flags on histograms = ±1 *SE*.)

The present data are of importance in suggesting a previously unsuspected way in which rats in natural environments might identify toxic foods without engaging in elaborate sampling procedures. A new recruit to an established population may have available a reliable, simple, and previously undescribed rule of thumb for determining which of many new foods it might eat during a single period of foraging could be responsible for any gastrointestinal distress it experiences: If sick, develop an aversion to whatever food you ate that others of your colony have not recently eaten. The need for elaborate sampling strategies to identify toxic novel foods would be obviated, and the problem of successful identification of toxic ingesta largely solved.

The effects on subsequent taste aversion learning of exposure to a conspecific may be observable only within a narrow range of parameters: They might be specific to juvenile rats, to aromatic diets, to relatively mild aversions, and so forth. Obviously, additional studies are needed to address such issues. Regardless of their outcome, the present result suggests that understanding of the learning of aversions to toxic substances by rats living in complex extralaboratory environments will require consideration of the effects of social learning as well as of individual experience in the identification and rejection of toxic foods. Rats are social animals, and life in social groups may provide access to information that is of importance in the acquisition of adaptive feeding repertoires by individual group members.

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