TASTE AVERSION LEARNING IN PEROMYSCUS

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This is to certify that the

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ABSTRACT

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By

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Taste aversion learning was reviewed, with emphasis on single trial learning, learning with long conditioned stimulus to unconditioned stimulus intervals, and the specific relationship between gustatory cues (as the CS) and induced illness (as the US). An "adaptive" explanation of the phenomenon was discussed, and taste aversion learning was related to toxin avoidance, to the presence of plant secondary compounds, to predation on model/mimic systems, to selection of specific nutrients, and to optimal foraging. Since the existing literature relates poorly to these potential functions (because of limitations imposed by the almost exclusive study of the domestic rat), an in-depth investigation of taste aversion learning in a nondomesticated species was proposed.

Mice of the native, rodent genus Peromyscus were used as subjects for such an investigation. All experiments were carried out using flavored fluids (usually 20% sucrose solution) as the taste cues and intraperitoneally injected lithium chloride solution as the illness-inducing agent. The acceptability/aversiveness of the test flavor was determined by using the quantity of fluid drunk as the

measured variable. The results indicated: (1) that taste aversion learning does occur in Peromyscus, (2) that a systematic variation in the dosage of toxin produces a systematic variation in the degree of aversion, (3) that aversions may be produced toward solutions flavored with sucrose, HCl, NaCl, or quinine, (4) that the aversion is directed specifically toward the flavor associated with illness, (5) that prior, nontoxic experience with a flavor later associated with illness decreases the degree of the aversion but increases the duration of the aversion, (6) that if another, safe fluid is simultaneously available, the aversion to a flavor associated with illness persists indefinitely, (7) that differences in age (weanling vs. adult) or degree of domestication (F_1-from-wild-caught vs. 30-year-lab-reared animals) appear to exert no differential effects upon taste aversion learning, and (8) that subspecies differences (Peromyscus maniculatus bairdi vs. P. m. blandus) lead to pronounced differences in aversion acquisition.

In conclusion, the findings made with Peromyscus were compared with those obtained with other animals, and the apparent similarities and differences were analyzed. Finally, an integrated model of the decision making and learning processes involved in dietary selection was developed.

TASTE AVERSION LEARNING IN PEROMYSCUS

By

Robert John Robbins

A DISSERTATION

Submitted to
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DEDICATION

I dedicate this work to the hundreds of mice whose involuntary participation was necessary for its accomplishment. I sincerely hope that the knowledge gained proves worthy of their sacrifice.

ACKNOWLEDGMENTS

I would like to express my appreciation to my major professor, Dr. John A. King, and to the other members of my guidance committee, Drs. Martin Balaban, M. Ray Denny, Richard W. Hill, and the late Stanley C. Ratner, for their advice and counsel during this undertaking. Financial support for this research was provided by a National Science Foundation Predoctoral Fellowship and by a teaching assistantship in the Department of Biological Science. Computational facilities were provided by the Michigan State University Computer Center.

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INTRODUCTION

Taste aversion learning, characterized briefly as the learned rejection of a food that has been associated with illness, is a robust, powerful phenomenon heretofore studied almost exclusively in terms of learning theory. Although nominally within the form of classical Pavlovian conditioning, taste aversion learning possesses a number of exceptional characteristics which render it both a challenge to and an exceptionally useful study paradigm for classical learning theory: (1) strong single trial learning is readily obtained, (2) the conditioned stimulus to unconditioned stimulus (CS/US) interval may be as long as 24 hours, and (3) a necessary relationship between cue and consequence has been demonstrated--that is, if an animal is presented a variety of cues across many sensory modalities, then made ill, it will almost invariably learn to avoid only the taste cues presented (see Literature Review, following, for specific citations). Furthermore, the phenomenon is general (many different illness-inducing agents, even rotation, have been successfully employed) and phylogenetically widespread (having been demonstrated in cats, codfish, cougars, coyotes, frogs, garter snakes, gerbils, guinea pigs, hamsters, humans, mice, monkeys, quail, rats, red-tailed hawks, and wolves).

These attributes that make the phenomenon particularly relevant to learning theory also make it relevant to other areas. Existing

studies indicate that a single pairing of toxicosis with a distinct flavor can lead to an animal's total rejection of that flavor on subsequent encounters. A phenomenon this powerful simply cannot be ignored if a complete ecological theory of foraging strategy is to be developed. Furthermore, the paradigm can be used to provide valuable experimental tests of optimization models for predators encountering a model/mimic system. And, if the phenomenon proves as profound and as general as the extant literature suggests it is, it may lead to new pest control techniques which act by altering the pest species' food preferences, rather than by extermination. This method would possess the singular advantage of eliminating the economic damage produced by the pest without removing that species from the ecosystem. Thus, learning theory, models of foraging strategy, and pest control methodology can all be united profitably with the numerous studies of taste aversion learning.

However, the bulk of the existing literature is not directly relevant to foraging strategy models or to pest control considerations because: (1) most of the work has been done on domestic species, particularly the white rat; and (2) the comparative work has, for the most part, tested no more than the simple occurrence of the phenomenon under optimal conditions. Furthermore, analysis of the comparative studies has been hampered by the fact that many such studies have used large or exotic species (requiring experiments of small sample size) with wide phyletic distances separating the species tested, thus rendering response comparisons virtually meaningless.

Consequently, if an ecologically oriented, functional study of the phenomenon is to be accomplished, extensive studies on a new species will have to be carried out. The animals studied should be of such a nature that large sample size studies may be economically performed, and the animals should provide a meaningful set of genetic and ecological differences tractable to careful and controlled analysis. Mice of the genus Peromyscus are well suited for this study. They are (1) abundant, (2) easy and inexpensive to obtain and rear, and (3) widespread across North America, occurring as hundreds of species and subspecies in a variety of habitats (Hooper, 1968). There is the drawback that no literature exists on taste aversion learning in Peromyscus, but as this is true for virtually any species it is hardly a fatal drawback.

Although the absence of basic research does preclude the immediate asking of questions directly relating taste aversion learning to foraging strategy, it does not preclude the asking of questions which examine the phenomenon in Peromyscus while simultaneously assessing its potential as a contributing factor to foraging strategy. With these considerations in mind, this dissertation will examine taste aversion learning in Peromyscus. Specifically, it will ask:

1. Does taste aversion learning occur in Peromyscus?
2. Does taste aversion learning in Peromyscus show a dose/response effect, or is it an all or nothing phenomenon?
3. Is taste aversion learning in Peromyscus affected by the quality of the flavor cue?
4. Is taste aversion learning in Peromyscus specific to the flavor paired with illness, or is it a generalized neophobia?

5. Is taste aversion learning in Peromyscus affected by prior, nontoxic experience with the poisoned flavor? Does the amount of prior experience produce differential effects?

6. Is taste aversion learning in Peromyscus a long lasting phenomenon, or is it transitory? That is, for how long will a single flavor/toxicosis pairing alter an animal's feeding preferences?

7. Is it possible to relate differences in taste aversion learning in Peromyscus to independent biological variables? Specifically, is it possible to detect differences associated with age, subspecies, or domestication?

The relationship of these questions to taste aversion learning's potential contribution to foraging strategy are reasonably apparent. For the phenomenon to be generally applicable to foraging strategy it should possess the following characteristics: (1) it should occur readily in the population; (2) it should not be limited by the quality of the flavors involved--if aversions can be formed only against certain specific flavors, the generality of the phenomenon is much reduced; (3) it should be directed with reasonable specificity toward the flavor paired with illness--if an aversion were only a generalized reluctance to eat, it could at best produce only brief and transitory modifications of an animal's foraging strategy; (4) it should not require absolute novelty of the flavor for either aversion acquisition or maintenance--if it did, the generalized applications of the phenomenon would be obviously limited; and (5) it must be long lasting--the greater the duration of an aversion, the greater the potential contribution to overall foraging strategy. Finally, if functional analysis is to proceed, it must be possible to link differences in the phenomenon with other important biological differences in the population. Once this is done, the functional analysis may begin apace.

LITERATURE REVIEW

Introduction

This section will not review the literature dealing with taste aversion learning in Peromyscus, since that literature is nonexistent. Neither will it attempt to exhaustively review the general literature dealing with taste aversion learning, as that has already been done by numerous authors (Garcia and Ervin, 1968; Garcia, McGowan, and Green, 1972a; Garcia, McGowan, and Green, 1972b; Rozin, 1975; Rozin and Kalat, 1971; Rozin and Kalat, 1972; Shettleworth, 1972; for a recent textbook treatment, see the discussion of the "Garcia Effect" in Bolles, 1975); nor will it attempt to provide an overall bibliographic treatment of the taste aversion literature, as the very recent publication of an indexed bibliography performs that function admirably (Riley and Baril, 1976). Rather, this section will briefly discuss the discovery of the taste aversion learning phenomenon and will treat in detail the evidence delineating the particularly intriguing attributes of taste aversion learning--powerful single trial learning, long CS/US intervals, and the specificity of cue to consequence. Then, the need for an adaptive explanation of these attributes will be considered and an attempt will be made to integrate the findings of taste aversion studies with the literature dealing with the general problems of feeding and optimal foraging strategy. Finally, a critique will be

made of the extant attempts to perform this integration, and suggestions for future work will be offered.

The Basic Phenomenon of Taste Aversion Learning

History

Although it has long been common knowledge among exterminators that rats which have survived one poisoning attempt will subsequently avoid the poisoned bait (for example, see Forbush, 1914), the scientific investigation of this phenomenon did not begin until relatively recently. During and following World War II, Richter, working in the United States, and Rzoska, working in Great Britain, set out to study this phenomenon as part of the war-time concern with rat control (inspired by no small fear of rat-based disease warfare). Their published works (Richter, 1953; Rzoska, 1953) show similar findings: (1) Rats will avoid a poisoned food after a single sub-lethal experience with it; (2) The aversion is not generalized to other foods, but restricted to the poisoned flavor; and (3) The type of flavor and the type of poison both affect the strength of the aversion.

Despite the detail with which these authors investigated the phenomenon, very little additional work was generated directly by their findings. Meanwhile, in the early 1950s John Garcia was investigating the effects of ionizing radiation upon food and water consumption. Noting that the suppression of consumption occasioned by the radiation was increasing with each exposure, he suspected that learning might be involved, and in 1955 he published a report (Garcia, Kimeldorf, and Koelling, 1955) that concluded, "Rats tend to avoid a

taste stimulus that has been associated with radiation exposure." Garcia and his co-workers continued to study this phenomenon during the late 1950s and early 1960s (Garcia and Kimeldorf, 1957; 1958; 1960; Garcia, Kimeldorf, and Hunt, 1956, 1961; Garcia, Kimeldorf, Hunt, and Davies, 1956). Although this work was generating very little additional research, Nachman (1962, 1963a, 1963b), while investigating the generalization of sodium preference to lithium preference by adrenalectomized rats, independently discovered that learned taste aversions could be produced using the gastro-intestinal distress induced by LiCl ingestion as the unconditioned stimulus.

Thus, by the mid 1960s, research in four independent laboratories had begun to elucidate the phenomenon of taste aversion learning under controlled conditions, yet very little attention was being paid to this work. Then, in 1966, Garcia and his coworkers published two papers which claimed that taste aversion learning possessed properties that challenged the assumptions of classical learning theory. The first (Garcia and Koelling, 1966) asserted that, "given reinforcers are not equally effective for all classes of discriminable stimuli. The cues, which the animal selects from the welter of stimuli in the learning situation, appear to be related to the consequences of the subsequent reinforcer." The second (Garcia, Ervin, and Koelling, 1966) found that illness produced by apomorphine injections was effective in conditioning a taste aversion, even if the onset of illness followed the termination of drinking by as much as 75 minutes. The shocking nature of these conclusions is evidenced by the fact that both papers were rejected by prestigious journals and were eventually

accepted only by lesser known publications. Also, Seligman and Hager (1972) report that, "One investigator, who had worked for years on delay of reinforcement, remarked publicly, 'Those findings are no more likely than birdshit in a cuckoo clock.'"

The controversial nature of these results prompted many researchers to perform replicates. With a decade of additional work now accomplished, the original conclusions still stand: taste aversion learning is characterized by single trial learning, by long CS/US intervals, and by a specificity of cue to consequence. The following sections will discuss in detail these findings, their challenges, and their replicates.

Single Trial Learning

From the first controlled studies of Richter (1953) and Rzoska (1953), it has been readily apparent that rats can acquire taste aversions after a single toxic experience. Similarly, single trial taste aversion acquisition has been reported in many other species ranging from codfish (MacKay, 1974) to coyotes (Gustavson, Garcia, Hankins, and Rusiniak, 1974) and from guinea pigs (Braveman, 1974, 1975) to quail (Wilcoxon, Dragoin, and Kral, 1971). Thus, the fact of single trial taste aversion learning has never been in question. However, the significance of it has been alternately asserted and challenged.

Seligman (1970, 1972) has made a great deal out of the observation that animals can make some associations with fewer trials than required for other associations. In fact, he has offered a new concept to learning theory, that of "preparedness," and has defined

it as follows: "The relative preparedness of an organism for learning about a situation is defined by the amount of input (e.g., numbers of trials, pairings, bits of information, etc.) which must occur before that output (responses, acts, repertoire, etc.), which is construed as evidence of acquisition, reliably occurs." Further, Seligman would divide the continuum of preparedness into three more or less distinct regions: the prepared region (with innate responses at the most prepared position, followed immediately by single trial learning), the unprepared region (dealing with the learning of arbitrary associations), and the contraprepared region (dealing with associations that cannot be made). He also notes that ethologists have been primarily interested in events of the prepared region, while psychologists have concerned themselves with the unprepared region. This point is nicely substantiated by contrasting Meehl's (1950) assertion that "a reinforcer can be used to increase the probability of any learnable response," with Tinbergen's (1951) observation that "The student of innate behavior . . . is repeatedly confronted with the fact that an animal may learn some things much more readily than others."

Seligman's hypothesis relies upon the assumptions that differences in preparedness derive solely from differences in associability and concomitantly that these differences have been adaptively shaped by natural selection (which in turn implies that all prepared associations are adaptive). Shettleworth (1972) has taken strong exception to these assumptions:

> Seligman . . . has attempted a broader synthesis of the experimental demonstrations of constraints on learning by suggesting that differential ease of learning different things represents different degrees of associative "preparedness." However, the

> "preparedness dimension" is merely an operational classification of learning tasks. It substitutes one oversimplification (that animals are more or less "prepared" to learn things and that "prepared" behaviors are acquired and extinguished differently from "unprepared" or "contraprepared" ones) for another (that the laws of learning are the same for all arbitrarily selected elements). This obscures the fact that apparent differences in learning difficulty may be brought about by a number of different mechanisms, not all of which are specifically associative."

And, she might have added, not all of which are necessarily adaptive. Despite her objection to Seligman's assumption of a purely associative and adaptive origin for differential ease of learning, Shettleworth in no way objects to an adaptive analysis of the problem. Indeed, she suggests that, "A complementary approach to constraints on learning is to ask whether what and how animals learn is related in a nonarbitrary way to what would be adaptive in natural conditions." Thus, although it may be unwise to assert that single trial taste aversion learning is adaptive, the failure to recognize that it might be, and to analyze it accordingly, would be equally imprudent.

Long CS/US Intervals

In the original report of radiation-based taste aversions, Garcia, Kimeldorf, and Koelling (1955) found that rats that had had saccharin solution consumption paired with a 6-hour exposure to radiation (5.0 r/hr) acquired a distinct and long lasting aversion to the flavor of saccharin. Although the simultaneous presentation of conditioned stimulus (saccharin) and unconditioned stimulus (radiation) followed the standard temporal arrangement for classical conditioning, the duration of the presentation was greatly in excess of the few seconds normally employed. In a subsequent paper (Garcia and Kimeldorf, 1957), designed specifically to test for differences among

animals subjected to trace conditioning, simultaneous conditioning, and backward conditioning, it was again noted that results for radiation-based taste aversion learning followed "the paradigm of conditioning despite the fact that . . . the duration of the stimuli is measured in hours, rather than in seconds." Since classical conditioning theory required that the interval between onset of CS and onset of US be no more than a few seconds, the authors were forced to postulate that either learning was occurring despite a long delay or that radiation was producing some immediate, but unknown effect upon the animals. They concluded, "The implication of the present series of studies is that [radiation produces] a prompt effect on the rat, presumably through stimulation of the nervous system."

However, as evidence continued to be accumulated that long exposures to ionizing radiation could produce learned taste aversions (review by Garcia, Kimeldorf, and Hunt, 1961), McLaurin began to suspect that the unorthodox temporal parameters used in such studies indicated that the phenomenon was not truly learning. He tested this (McLaurin, 1964) by offering rats saccharin, then exposing them to radiation following a delay of 3, 60, 120, or 180 minutes. All groups, regardless of the length of delay between saccharin consumption and onset of radiation, showed equal and profound aversions, and this absence of an expected delay of punishment gradient forced him to conclude that radiation-based taste aversion learning could not be classified within the framework of the classical conditioning paradigm.

Meanwhile, Garcia and his coworkers had been considering the difficulties inherent in the use of radiation to produce taste

aversions, particularly the problem of determining precisely when the relevant effect of the radiation was occurring. To circumvent this, they performed a taste aversion experiment using intraperitoneal injections of apomorphine hydrochloride as the illness-inducing agent. Apomorphine produces an obvious illness within minutes of injection. Four groups of rats were subjected to the following treatments: (1) saccharin consumption, followed by shock, (2) saccharin consumption, followed by saline injection, (3) water consumption, followed by apomorphine injection, and (4) saccharin consumption, followed by apomorphine injection. Only the sacc/apo group showed any subsequent aversion to saccharin. The failure of the water/apo group to show an aversion ruled out pseudoconditioning (sensitization) as the cause of the aversion. Next, the authors subjected five groups of rats to sacc/apo pairings with CS/US delays of 30, 45, 75, 120, and 180 minutes. Aversions were formed with delays up to 75 minutes and the degree of aversion was proportional to the length of delay--that is, a delay of punishment gradient was observed. The authors concluded that true learning was occurring, even with a CS/US interval of 75 minutes.

Garcia et al.'s demonstration of a delay of punishment gradient appeared to contradict McLaurin's failure to detect such a gradient. However, Smith and Roll (1967) replicated McLaurin's work using a larger dose of radiation (100 vs. 61.4 roentgens) and CS/US delays of 0.0, 0.5, 1.0, 2.0, 3.0, 6.0, 12.0, and 24.0 hours. With this paradigm a delay of punishment gradient was found, but it was not readily apparent except in delays greater than the 3.0 hour maximum employed

by McLaurin. Other replicates have been performed which demonstrate that McLaurin's failure to find a gradient was an artifact produced by the use of insufficiently long delays. Revusky and Garcia (1970) have provided an excellent review, "Learned associations over long delays," which treats in detail the evidence for and against this phenomenon. In this review they argue convincingly that taste aversion learning truly represents learning over a long CS/US interval.

Despite the great and growing body of evidence that taste aversion learning over a long delay does occur, some authors remain skeptical, even truculently so. The most prominent of these, M. E. Bitterman (1975), has argued strongly (and ad hominem) that all apparent indications of learning over long delays are the result of improperly controlled experimentation ("Problems of control abound in these aversion experiments, perhaps because they are not always uppermost in the minds of the investigators."), and he suggested that the actual explanation is, "smell and taste receptors are stimulated again at the time of illness by food returned to the mouth from the stomach."

This possibility of restimulation has not been ignored by students of taste aversion learning, and a large number of experiments have been performed which test and appear to rule out aftertaste or restimulation: (1) Many workers have produced aversions with delayed illness paradigms using dilute HCl as the taste stimulus (Ader, 1973a, 1973b; Braveman, 1974; Dragoin, 1971; Etscorn, 1973; Etscorn and Stevens, 1973; Garcia, Green, and McGowan, 1969; Wilcoxon, Dragoin, and Kral, 1971). Since the concentration of the HCl used as a taste

stimulus was much less than that found in the stomach, and since an appreciable delay occurred between drinking and illness (24 hours in the Etscorn and Stevens study), it is unlikely that any illness-induced vomiting could produce a distinguishable aftertaste. (2) Kalat and Rozin (1970) found that if rats were given one novel flavor, then another novel flavor, then made ill, the aversion would always be directed to the same flavor, regardless of the order of presentation. This argues against aftertaste, since the most recently consumed flavor should have the strongest aftertaste. (3) Kalat (1974) found that, "When rats are poisoned after drinking two concentrations of the same solute, rats reared on water acquire aversions mainly to the more concentrated solution, but rats reared on a still more concentrated solution acquire aversions mainly to the less concentrated solution, which for them is more novel." This clearly should rule out regurgitated aftertaste, as the balancing of all groups for order of presentation insured a stomach load of equally concentrated saccharin in all groups. (4) Most persuasively, Roll and Smith (1972) found a strong aversion in rats which were allowed to drink saccharin, placed into a deep surgical anesthesia, irradiated with 100 roentgens while anesthetized, and maintained under the anesthesia for 8.5 to 10.0 hours following the irradiation. Control animals, treated similarly except sham irradiated, showed little or no aversions. This procedure seems to rule out illness-induced restimulation of taste receptors.

Despite these findings and others cited by Garcia, Hankins, and Rusiniak (1976) in a rebuttal to his earlier article, Bitterman

remains unconvinced--as is evident in his counter-rebuttal (Bitterman, 1976). Although I am personally unimpressed with Bitterman's counter arguments, particularly as a result of his tendency to attack only the weakest arguments in favor of long-delay learning (e.g., he dismissed the significance of the two-concentrations-of-solute experiment by citing the admitted procedural difficulties in an experiment by Rozin, 1969, while ignoring the same results in the untainted experiment of Kalat, 1974, cited above), the reader is urged to investigate this heated controversy by reading the arguments of Bitterman and Garcia et al., in the original.

Considering the still controversial nature of the claims for taste aversion learning with a long CS/US interval, it is probably best not to assert that the case for this phenomenon has been conclusively made. However, it is possible to assert that pseudoconditioning (see arguments above and in Revusky and Garcia, 1970) and mediation by secondary reinforcers (this simply cannot explain single trial acquisition) cannot account for the data. Thus, only the argument in favor of aftertaste or illness-induced restimulation of taste receptors still commands support. And, as is evident from the arguments above, in Revusky and Garcia (1970), and in Garcia, Hankins, and Rusiniak (1976), this theory is, if not refuted, under severe challenge. But, whatever the outcome of the debate on learning mechanism, it is impossible to deny that taste aversion learning does occur when the ingestion of the flavor is separated from the onset of illness by a substantial time interval.

The Specificity of Cue to Consequence

As early as 1957, Garcia, Kimeldorf, and Hunt had observed that, "A single [radiation] dose of 30 r at 5 r/hour is sufficient to demonstrate conditioning in the saccharin preference test which utilizes a stimulus-response sequence closely associated with the gastro-intestinal functions, namely taste and consumption. [But,] there is a considerable loss in sensitivity when situational stimuli are substituted for the taste cue . . . ." However, it was not until 1966 that a direct assertion was made claiming a necessary relationship between cue and consequence: "[G]iven reinforcers are not equally effective for all classes of discriminable stimuli. The cues which the animal selects from the welter of stimuli in the learning situation, appear to be related to the consequences of the subsequent reinforcer" (Garcia and Koelling, 1966).

This claim has sparked a lively debate and has led to numerous experiments designed to test the relative efficacy, in a poison-based aversion learning paradigm, of cues presented in various sensory modalities. Because the concept of specificity of cue to consequence is controversial and important, and because so much work has been done regarding it, a review of the major work will now be presented, organized by cue category.

Visual. Much of the work on the association of visual cues with toxicosis derives almost directly from the 1966 paper of Garcia and Koelling, "Relation of cue to consequence in avoidance learning." In that experiment, rats were presented with a compound stimulus: flavored water was offered in a drinking spout which was modified so

that each time the rat's tongue contacted the spout a clicking relay fired, providing power to a 5 watt bulb. Thus, with each lick the rat experienced a gustatory, a visual, and an auditory cue. One group of rats was given shock as the unconditioned stimulus following each trial with the "bright-noisy-tasty" water, another group was irradiated (i.e., was made ill) following each trial. After a number of acquisition trials, each group was subdivided with one subgroup tested on the audio-visual component of the compound stimulus and the other on the taste component. Figure 1 gives the results. The carefully

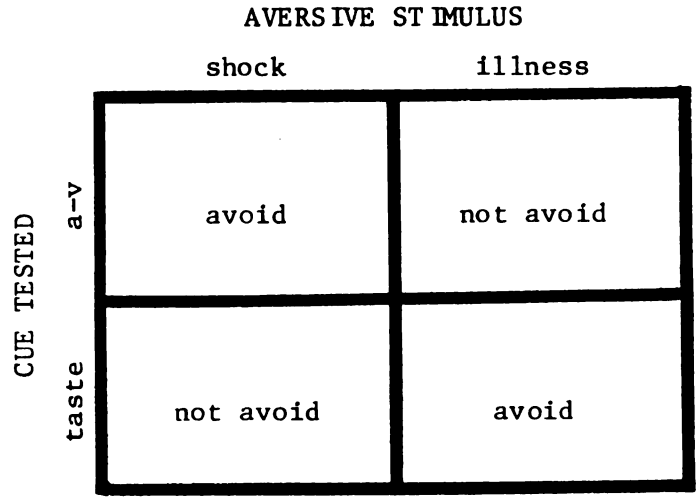


Fig. 1.--Results of Garcia and Koelling, 1966.

balanced design of their experiment lends credence to the authors' conclusion that a necessary relationship exists between cue and consequence in avoidance learning. Furthermore, in a similar experiment Garcia, McGowan, Ervin, and Koelling (1968) used solid food pellets that differed in size and in flavor and attempted to condition an

aversion using shock and x-ray-induced illness as the consequences. Figure 2 gives their results. Again, a well balanced design provides evidence for a necessary cue/consequence relationship.

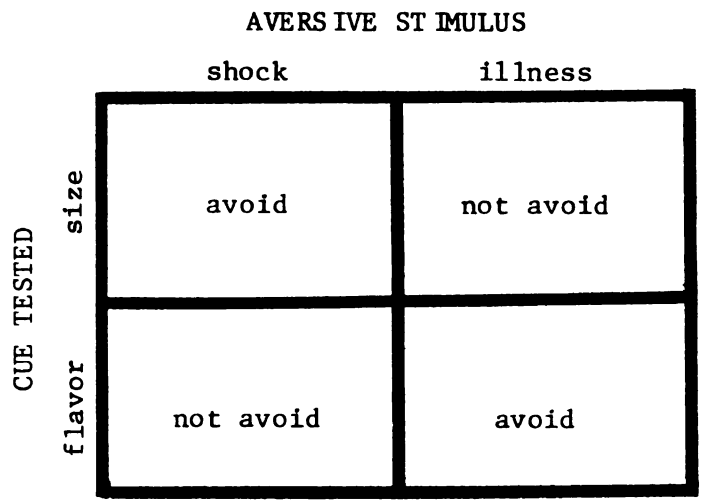


Fig. 2.--Results of Garcia, McGowan, Ervin, and Koelling, 1968.

However, Domjan and Wilson (1972) suggested that "the effect may result from differences in the way in which rats ordinarily receive gustatory and audiovisual stimulation. Novel gustatory cues are normally experienced only in conjunction with ingestion, whereas the reception of audiovisual stimulation often does not depend on a specific response by the organism." In order to test this theory, "both taste and nongustatory CS's were presented in the absence of approach and ingestive behaviors to minimize involvement of ingestion in the associative process." This was accomplished by sounding a buzzer and simultaneously rinsing the oral cavity of a non-fluid-deprived rat with a saccharin solution via an implanted cannula. This was followed by either shock or LiCl-induced illness. The

results were similar to the earlier work: illness led to an aversion to the flavor but not the auditory cue, while shock led to an aversion to the auditory cue but not the flavor. Domjan and Wilson then tested for the effect of the compound presentation of the stimuli by repeating the experiment with the auditory or taste cues presented individually, then followed by the different aversive stimuli. Again it was found that illness occasioned an aversion to flavor, but not sound, while shock produced an aversion to sound, but not flavor.

In 1972, Green, Bouzas, and Rachlin noted that the difference might not lie in the modality of the unconditioned cue, but rather in the duration of the unconditioned cue. That is, in all of the previous work the shocks had been delivered as a brief pulse while the illness had an obvious duration of a half an hour or more. To test for this effect they offered three groups of rats saccharin, then gave one group lithium chloride, one group discrete shock, and one group continuous shock for 52 minutes. Again they found that only illness led to an aversion of the flavor cue.

Considering that Garcia and Koelling's 1966 work has withstood these two challenges (and that it was repeated using shock and apomorphine-induced illness by Green, Holmstrom, and Wollman in 1974) it appears that strong evidence does exist for a cue/consequence relationship in aversion learning. However, comparative studies have indicated that taste is not always the dominant cue in aversions developed due to illness.

Wilcoxon, Dragoin, and Kral (1971) compared the performance of rats and bobwhite quail when given taste and visual cues followed by

cyclophosphamide-induced illness. With the stimuli presented individually they found that "Bobwhite quail, like the rat, learn in one trial to avoid flavored water when illness is induced by a drug one half hour after drinking. In contrast to the rat, quail also learn to avoid water that is merely darkened by vegetable dye." Most surprisingly, when the flavor and visual stimuli were presented together, followed by illness, followed by a test on the cues individually, they found the results given in Figure 3. That is, the rats performed

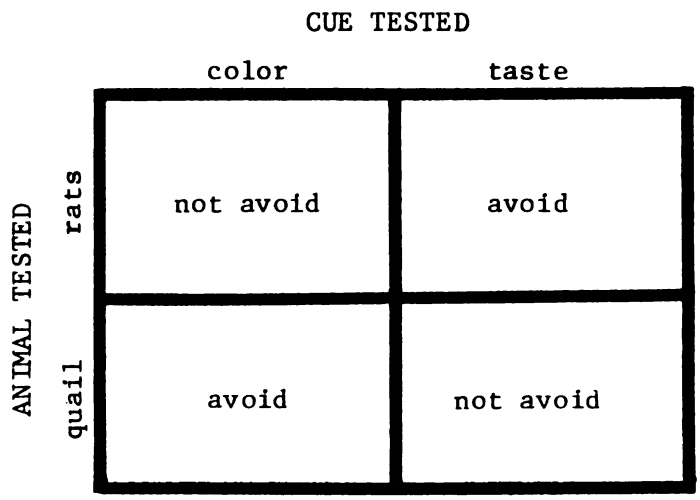


Fig. 3.--Results of Wilcoxon, Dragoin, and Kral, 1971.

according to the expectations generated by the earlier work of Garcia and Koelling, while the quail performed exactly conversely. A further control experiment eliminated the possibility that the quail were using a slight flavor cue from the dye. A final control experiment ruled out the possibility that the trauma of handling was of greater significance to the quail than to the rats. The authors concluded by

suggesting that the well known visual acuity of birds might provide the key to this difference in behavior.

However, Braveman (1974) noted the evidence of similar visual acuity in rats and guinea pigs, then demonstrated that guinea pigs could learn to avoid visual (color) cues when these were individually paired with LiCl-induced illness. In 1975, Braveman pointed out that the salience of a cue modality could be a function of the intensity of stimulation in that modality and suggested that the findings of Wilcoxon et al. might be the result of the specific stimulus intensities employed in their study. In order to guard against this possibility when he tested the relative salience of visual and taste cues in guinea pigs, he preceded toxicosis with pairings of varying concentrations of flavors and varying intensities of color, then tested for an aversion to the color cue or to the taste cue. At all stimulus intensity levels tested he found strong aversions to the taste cue, but no aversion to the color cue. These results indicated that in guinea pigs (as in rats) taste cues far overshadow visual cues in poison-based avoidance learning.

In addition to the vision-versus-taste studies cited above, a number of papers have studied the individual pairing of visual cues as the conditioned stimulus with illness as the unconditioned stimulus. Green monkeys (Johnson, Beaton, and Hall, 1975) have been shown to form poison-based aversions to purely visual stimuli. Capretta (1961) demonstrated that chickens also will form aversions to strictly visual stimuli. However, the work on rats has yielded less clear cut results. Wilcoxon et al. (cited above) had found no aversion to visual cues

if these were paired with illness. But Garcia, Kimeldorf, and Hunt (1957) found that rats irradiated for 5 hours in a black compartment showed a subsequent decrease in preference for that compartment. However, Martin and Ellinwood (1974) found that although methamphetamine was an effective US in producing an aversion to a flavor, it was ineffective in producing an aversion to a black compartment (but it did produce a slight aversion to a gray compartment). Hargrave and Bolles (1971) found that an IP injection of 3 ml/kg of body weight of a 15% saline solution was effective in producing taste aversions, but was not effective in producing an aversion to a distinct visual environment (a striped compartment or a gray compartment). Garcia and Koelling (1967) compared aversions in rats induced by x-rays, ingested toxin (LiCl), and injected toxin (apomorphine) and found that all were effective in producing taste aversions, but not visual aversions.

These difficulties in training rats to avoid a visually distinct compartment may be explained by the results of Best, Best, and Mickley (1975), who found that "Rats subjected to apomorphine-induced malaise following a 2-min placement in a black compartment avoided this black compartment significantly more than controls in a choice situation. The degree of aversion, however, was substantially reduced when animals were provided water (or saccharin) in the black compartment during conditioning and testing." Water or some other fluid was available during conditioning and testing in all of the studies which found it impossible to condition an aversion to a visually distinct environment. This difficulty in producing an aversion to a visual cue

in the presence of a taste cue is not only consistent with the work of Best et al., but is predicted by the original 1966 Garcia and Koelling paper.

In summary, the literature indicates that poison-based avoidance learning *can* be accomplished using visual cues, but the ease with which this may be done is determined by the species tested. Attempts have been made to attribute the inter-taxic differences in dominant modality to specific differences in the species' niches: Wilcoxon, Dragoin, and Kral (1971) cite the bobwhite quail's diurnal nature and visual acuity as an *a priori* reason for suspecting the dominance of vision in this species. Braveman (1974, 1975) discounts the importance of visual acuity, while Johnson, Beaton, and Hall (1975) stress the importance of visual acuity and the relative significance of vision in the animal's niche. However, since no studies have been done on species which are phylogenetically closely related, yet ecologically diverse, and since in actuality very little comparative work has been done, any claims regarding ecological predictors of the dominant modality are severely premature.

Positional. Only a few studies exist in which position has been used as a cue in poison-based avoidance learning, and their results seem to indicate very little salience for position *per se*. Some studies on wild populations have touched upon the learning of position aversions: Barnett (1963) in his discussion of "bait-shyness" notes that feral rats which have learned to avoid poisoned baits show no aversion to the location of the bait. Tevis (1956), studying the reaction of feral *Peromyscus maniculatus* and *P. truei* to poisoned

Douglas-fir seeds, found that mice which had survived the toxin rapidly learned to avoid ingesting the toxic seeds, but showed no aversion to the position of the seeds: "Bait-shy mice . . . avoided Douglas-fir seed yet regularly explored the [bait stations], leaving within tracks, droppings, and the remains of insects captured outside. Even when the [stations] offered nothing but Douglas-fir seed, mice visited them frequently, as they did natural nooks and crannies . . . ."

In laboratory studies, Galef and Clark (1971) found that brown rats which had been trained to distinguish between a safe and a toxic food which differed in flavor and in position always followed the flavor cue when the positions were reversed. Garcia, Kovner, and Green (1970) found that rats which were given saccharin in one arm of a T maze, then made ill, rapidly learned to avoid ingesting saccharin, but continued to enter the arm of the maze associated with poisoning. In their study on green monkeys, Johnson, Beaton, and Hall (1975) found that switching the positions of the containers had no effect upon the aversion directed to the color of the containers. Rozin (1969) gave rats two drinking stations which differed in position and in visual appearance, but which contained the same saccharin solution. He found that if drinking from one of the containers was followed <u>immediately</u> with illness, aversions could be produced. However, with a 30-minute delay between CS and US, no aversions were formed. (Strong flavor aversions are easily produced with a 30-minute CS/US interval.) In one of my own pilot studies I found that control group mice which were simultaneously offered two drinking tubes which differed in position and in contents (tap water vs. saccharin) showed

a strong position preference, while experimental animals that had had saccharin paired with a LiCl injection followed the flavor cues exclusively and gave no evidence of a position preference.

In summary, it seems that position per se has a very low salience as a cue for poison-based avoidance learning. Although aversions have been produced when illness was contingent upon cues that differed in position and in visual appearance (Rozin, 1969, above; also several papers discussed in the section on vision: Martin and Ellinwood, 1974; Garcia and Koelling, 1967; Best, Best, and Mickley, 1973; Garcia, Kimeldorf, and Hunt, 1957), no experiments have been performed in which position alone was used as the discriminatory cue. Furthermore, in studies where position and other cues were played against one another the position cue has been consistently overshadowed.

Olfactory. Because of the close relationship between olfaction and taste, one might expect that studies of poison-based avoidance learning using odor as the primary cue would produce results similar to those obtained using taste. In fact, one might expect that olfaction could be strongly implicated in those experiments which purport to study only taste aversions. This has not been the case.

In an early study Garcia and Koelling (1967) reported that a comparison of aversions induced by x-rays to the sight of a distinct visual environment, to the odor of "lilies of the valley" perfume, and to the flavor of dilute vinegar produced the following results: a strong aversion to the flavor, an intermediate aversion to the odor (as recorded by fluid intake in its presence), and no aversion to the visual cues (also recorded by intake). Hargrave and Bolles (1971)

reported that rats which had learned to avoid distinctly flavored fluids associated with delayed (30 minutes) illness showed no aversion to an arm of a Y-maze which was marked with the odor of that fluid. On the other hand, Supak, Macrides, and Chorover (1971) were able to produce an aversion to the odor of amyl acetate if the odor was present while the rats were drinking a 0.1 molar LiCl solution, and Pain and Booth (1968) produced an aversion to odorized glucose solution paired with illness. Unfortunately, however, both works suffer from methodological difficulties: in Supak et al. the animals ingested the toxin. This produces a rapid onset of illness, and Rozin (1969) has shown that the range of stimuli associable with toxicosis is inversely proportional to the length of the CS/US interval. In Pain and Booth's study the odorant was placed in the fluid consumed and no control was run to test for the flavor of the chemical added as an odorant. Lorden, Kenfield, and Braun (1970) checked for the effect of flavor on an apparent odor aversion and found that an illness-induced aversion to the smell of isopropanol was much increased if the rats could also taste the isopropanol.

Thus, it seems that aversions can be formed toward odors, but that these aversions are weaker than those formed toward flavor. However, Taukulis (1974) suggested that all of the studies claiming low associability for odor suffered from a methodological problem: the odor was not localized at the ingested fluid, except in those studies in which the odor was in the fluid (in which case flavor was not controlled). To test the effect of localizing the odor, Taukulis devised a drinking spout within a larger spout through which odorized

(with amyl acetate vapor) air was dispensed. With this apparatus he was able to produce significant aversions with CS/US intervals of up to 4 hours. But, it is possible to object to Taukulis' results on the grounds that the physical proximity of the vapor source and the fluid source could easily lead to the amyl acetate going into solution on the rat's tongue, thereby invoking taste. Taukulis acknowledges this possibility, then discounts it, although he did not specifically control for it.

In view of the contradictory evidence that exists regarding the formation of poison-based olfactory aversions, an alternate question might be asked: How important is olfaction in the formation of poison-based aversions toward flavors? Richter (1953) offered rats food laced with a lethal dose of alpha-naphthyl thiourea (ANTU), a commercially employed rat poison. The survivors of this undertaking developed strong aversions to the poisoned food. Then Richter removed the olfactory bulbs of all the survivors and found that over half ate lethal doses of the toxin. Finally, when he severed the lingual and glossopharyngeal nerves of the remaining survivors, all rats ingested lethal doses and died. The fact that better than 50% of the animals lost their aversions upon the removal of the olfactory bulbs might be taken as strong evidence for a highly significant olfactory component in the learning. This interpretation is disputed, however, by the 1973 work of Hankins, Garcia, and Rusiniak who reported that <u>peripherally</u> anosmic rats were equal or superior to normal controls in developing and maintaining aversions, while bulbectomized animals were inferior to controls in both the acquisition

of taste aversions and the learning of conditioned suppression of licking in the presence of white noise with shock as the aversive cue. Since these animals were deficient in a task thoroughly unrelated to olfaction, it seems reasonable to conclude that the operation produces a more severe deficit than mere anosmia. Following this reasoning, only the data from the peripherally anosmic animals are unconfounded, and they indicate that olfaction is not important, and certainly not necessary, for the acquisition and retention of poison-based taste aversions.

Auditory. Garcia and Koelling's 1966 experiment involved presenting a rat with a visual, an auditory, and a gustatory cue ("bright-noisy-tasty" water; see section on vision above for a detailed discussion of this paper) followed by illness. No aversion was found to the combined audio-visual component of the stimulus. Garcia and Koelling (1967) tried pairing a "distinct environment," which included auditory cues, with illness and found no aversion. In the only direct study of audition, Domjan and Wilson (1972) paired a buzzer with LiCl-induced illness and found no aversion. Thus, the small amount of evidence that has been obtained indicates no association of auditory cues with illness.

Textural. Only two papers have dealt directly with textural attributes of the food as a potential cue, and their results precisely contradict one another. Rozin (1969) offered rats dry powdered food and the same food mixed with water. Neither the animals that had dry food associated with poisoning, nor those that had wet food associated

with poisoning showed any aversion. On the other hand, Garcia, Hankins, Robinson, and Vogt (1972) using a very similar design were able to produce aversions. It is difficult to attempt to explain such contradictory results, except to note that the experiments differed in some procedural details: neither the food cue nor the toxin was the same in both studies. Further work is obviously needed if the possibility of poison-based textural aversions is to move beyond the realm of speculation.

Thermal. For temperature also, only two papers exist, and although their results are not directly contradictory, neither are they wholly in accord. Nachman (1970) was successful in producing an aversion to hot (43°C) distilled water which had been paired with LiCl-induced illness, while Rozin (1969) was unsuccessful in producing a discrimination between two foods with similar flavors, but which differed in texture (liquid vs. solid), position (right vs. left), container (cup vs. tube), and temperature (30°C vs. 10°C). Since evidence cited above on overshadowing (particularly Best, Best, and Mickley, 1973) indicate that additional cues may in some cases inhibit learning that might otherwise occur, it is possible that the wealth of nongustatory cues offered by Rozin actually made learning more difficult for the animals than the simple temperature differential offered by Nachman. In any event, with only two inconsistent studies extant on the subject, it is difficult to put forward strong claims regarding the associability of temperature cues with toxicosis.

Summary. With the exception of audition, some evidence exists for poison-based aversion toward a number of cues besides taste. However, in every case (with the exception of visual cues in the bobwhite quail) if the alternate cues were offered in direct competition with taste, they were overshadowed. Thus, the evidence indicates that animals do use a cue modality hierarchy when making associations with poisoning--that is, there is a necessary relationship between cue and consequence in avoidance learning. Furthermore, in that species which has been most thoroughly studied, the rat, the evidence is overwhelming that the dominant modality is taste.

The Need for an "Adaptive" Explanation

Taste aversion learning has been shown to occur with single trials, with long CS/US intervals, and with some specificity of cue to consequence. Since all three of these attributes are advantageous to animals attempting to maximize their dietary options while minimizing their contacts with toxins, many authors have been quick to suggest, some even to assume, that these attributes are specific adaptations resulting from selection pressure in favor of animals that were most skillful at avoiding poisons. For example:

> A cursory inspection reveals that, like many other species, the rat has been admirably designed by natural selection to associate gustatory and olfactory stimuli with internal states.
>
> Garcia and Ervin, 1968

> Such experiments demonstrate quite clearly that the rat's learning ability is adapted to the fact that illness or well-being normally is caused by something ingested, that it normally occurs some time after ingesting the thing that causes it, and that foods can reliably be distinguished by their tastes.
>
> Shettleworth, 1972

> Natural selection has designed the rat with another distal-proximal system to cope with the internal environment. Foodstuffs are chemically analyzed by gustatory and olfactory receptors when sniffed and eaten. Later, as the food is absorbed, internal receptors report on the ultimate effects with the internal environment. These two afferent categories converge upon a visceral center which is relatively insulated from stimuli arising in the external environment. Since food absorption takes time, this system has become specialized to handle long interstimulus intervals.
>
> Garcia, McGowan, and Green, 1972

> . . . the tendency to associate tastes with aversive internal consequences as opposed to associating either element with anything else . . . seems eminently sensible from an adaptive point of view.
>
> Rozin and Kalat, 1972

> The demonstration of rapid one-trial conditioning in snakes over a long delay and with an unambiguous effect, illustrates both the potency of what seems to be a very adaptive process and its widespread occurrence in vertebrates.
>
> Burghardt, Wilcoxon, and Czaplicki, 1973

> Rats are prepared, by virtue of their evolutionary history, to associate tastes with malaise.
>
> Seligman, 1970

Granted that the attributes of taste aversion learning are useful for avoiding toxins, but all of these authors seem to forget that "useful for" and "adapted to" are not synonymous in the strict biological sense. Iain Douglas-Hamilton, in his interesting account, Among the Elephants (1975), has pointed out that an elephant's tusks are quite useful for puncturing Land-Rovers. But to suggest that they are adapted to such a task would be ludicrous. Yet that is precisely the logical fallacy committed by those who have assumed a particular adaptive explanation for taste aversion learning, relying upon the "obvious" nature of this assumption. The danger in "obvious"

evolutionary explanations has been pointed out by Hulse, Deese, and Egeth (1975) who have noted that if it is obviously advantageous for rats to evolve taste aversion learning to avoid ingesting toxins, it is also obviously advantageous for them to evolve some mechanism for dealing with the occasional errors made by this learning system--yet rats are incapable of vomiting.

All of this is not to suggest that an adaptive analysis is to be avoided. Quite the contrary. The challenge of any distinct biological feature, behavioral or morphological, is to explain its function, its adaptiveness. But this is not a simple task to be accomplished by inspecting the results of a single experiment, or even a series of experiments, looking for the most obvious explanation. Rather it is to be accomplished by a painstaking and exhaustive analysis postulating every conceivable explanation, followed by the formation of hypotheses and discriminatory tests to sort among these hypotheses. That this type of analysis has not been undertaken by those who see the obvious adaptive value of taste aversion learning is demonstrated in their diverse opinions regarding precisely what taste aversion learning is adapted to. Some authors are convinced that the phenomenon is pronounced in rats in response to years of poisoning by man (Shettleworth, 1972; Galef and Clark, 1971b). Others feel that it is an adaptation to omnivory (Hargrave and Bolles, 1971; Alcock, 1975). And still others feel that it is an adaptation to the risks of feeding in general and should be found in many animals, even those that show little or no other learning abilities (Kalat and Rozin, 1972; Rozin and Kalat, 1971). The development of discriminatory

tests to assess the relative validity of these hypotheses would be comparatively easy. Yet no one has performed any, no one has suggested any, and no one has even examined the literature to see if any have already been performed.

However, before the criticism of this conjecture on adaptation becomes too harsh, it is important to recall that functional analysis has not been the primary intent of these authors. Instead they have been concerned with the study of learning, and, as Shettleworth (1974) has pointed out, "Perhaps because the study of learned behavior does not share ethology's tradition of concern with the function of behavior in the natural environment, discussions like those just referred to sometimes seem to ignore the fact that speculations about the function of certain types of learning (or nonlearning) are not accounts of mechanism."

Perhaps so, and perhaps this suggests that the comparative method of ethology should be employed in the functional analysis. Although the ethological approach has, until now, been applied only to the study of differences in innate behavior patterns, there is no inherent reason why it could not be applied to learning. Indeed, Hinde (1970), in a discussion of the ethological approach to functional analysis, has observed, "There is, of course, no reason why we should not ask similar questions about the selective significance of differences between individuals in learning ability, or powers of perception, though such problems have not yet been tackled experimentally." Although the comparative approach does have the difficulty of requiring vast amounts of descriptive data, some authors believe it to

be the only method capable of providing the background information necessary for a truly functional analysis: "All the important new facts brought to light by ethology . . . are due to the application of the comparative method and could not have been discovered by any other technique (emphasis in the original)" (Konrad Lorenz, 1970).

However interesting and controversial Lorenz's assertion, a full blown methodological discussion will not be entered into here. (A detailed consideration of methodology is presented below beginning on page 54.) Rather, this section will conclude by asserting that taste aversion learning is a distinct biological character requiring an adaptive explanation and that this explanation can be begun only after a detailed consideration has been given to all of the possible contributions of taste aversion learning to feeding strategy--not just the contribution of toxin avoidance. This consideration will be made in the next section of the Literature Review.

Functional Implications of Taste Aversion Learning

Introduction

Nearly everyone who has considered the adaptive value of taste aversion learning has assumed that its primary function is toxin avoidance. Other aspects of feeding in which taste aversion learning might play a role have, for the most part, not been considered. An effort to remedy that omission will be presented here. As toxin avoidance has been the area most closely examined, it will be treated first, followed by plant secondary compounds, predation on model/mimic systems, and the selection of specific nutrients.

Toxin Avoidance

Although toxin avoidance has been the assumed primary function of taste aversion learning, only two pieces of evidence exist to support this assertion: (1) in laboratory experiments animals have rapidly learned to avoid ingesting foods containing toxins, and (2) efforts to exterminate feral rodent populations using poisoned baits have nearly always met with failure, apparently due to learned avoidance of the bait.

Despite the difficulties inherent in basing an evolutionary argument upon such scanty evidence, a number of authors have done just that. As discussed above (p. 30), these arguments tend to fall into three categories: (1) Man has long been directing an extensive poisoning campaign at rats. The resulting intense selection pressure has led to the highly developed taste aversion learning skills found in rats. (2) Omnivorous animals, occupying a variety of habitats and faced with a wide array of possible foodstuffs, are constantly encountering the risk of ingesting toxic materials. Consequently, natural selection has produced highly refined taste aversion learning capabilities in omnivores. (3) All animals must eat to live and all animals incur a risk of toxicosis whenever foreign substances are ingested. Therefore, natural selection should produce taste aversion learning in almost all animals.

A direct effort to decide among these three theories has not been made, but a review of the existing data can suggest which of the three seems most valid: (1) The first theory, relating the phenomenon to human poisoning efforts, is severely weakened if the

phenomenon is demonstrated in species other than rats--particularly species never subjected to extermination campaigns. Single trial taste aversion learning has been demonstrated in cats (Kimeldorf, Garcia, and Rubadeau, 1966), codfish (MacKay, 1974), garter snakes (Burghardt, Wilcoxon, and Czaplicki, 1973), guinea pigs (Braveman, 1974, 1975), hamsters (Zahorik and Johnston, 1976), and quail (Wilcoxon, Dragoin, and Kral, 1971). With this evidence it appears unlikely that human poisoning efforts have played a fundamental role in the evolution of taste aversion learning. (2) The second theory, relating taste aversion learning to omnivory, is greatly weakened by any evidence of the phenomenon in animals with restricted diets. Although none of the species on the list above is exceptionally stenophagic, neither is any of them exceptionally omnivorous. Thus, there is some weak evidence against the omnivory theory. However, Freeland and Janzen (1974) have reported some observations which suggest that koalas, which feed on a highly restricted diet of Eucalyptus leaves, may form learned taste aversions. "[T]he koala cannot subsist on one species of Eucalyptus alone. If given a single species of Eucalyptus, it eats the leaves for 1 day and then rejects them." Since Eucalyptus leaves all contain toxic essential oils and phenols, but the specific toxins vary from species to species, Freeland and Janzen hypothesize that the switching from one species to another may be necessary for the koala to avoid toxicosis due to the swamping of an individual detoxifying enzyme system. If this were true, and if the eventual rejection of leaves from a single species were a learned rejection, then the existence of taste aversion learning in the almost

definitively stenophagic koala would destroy the notion that the phenomenon is tightly bound to omnivory. Clearly, a specific test for taste aversion learning in koalas is an experiment worth doing. (3) The third theory, relating taste aversion learning to feeding in general, would be most severely damaged by a failure to demonstrate the phenomenon in a species that has otherwise been shown to possess moderate learning capacities. Despite a very extensive literature, such a failure has never been reported. Furthermore, the demonstration of the phenomenon in species as diverse as codfish, garter snakes, and hamsters lends credence to the general feeding theory. And, were it possible to show taste aversion learning in koalas, the general feeding theory would be most enhanced.

If the three theories are considered in terms of the data examined above, it appears that human extermination campaigns have not been crucial to the evolution of this phenomenon and that taste aversion learning is more likely related to eating in general than restricted to omnivory.

It is still possible, however, to object to any of these theories on the grounds that innate rejection of toxic foods is by far the most optimal strategy and that therefore selection in favor of the ability to develop learned aversions must be fairly limited. But, innate aversions can only develop toward foods in the species' environment that are regularly present, readily identifiable, and reliably toxic over a long enough time scale to allow selection to work. Furthermore, if we grant the slightest possibility that animals may encounter toxic foods toward which they have no innate

aversions, it is easy to demonstrate that the selection pressure in favor of any mechanism that could minimize the number of ingestions of that food would be intense: Any time an animal ingests a toxic food it experiences some probability, p, that it will survive, and some probability, q, that it will die ($p + q = 1$). The probabilities of surviving or dying over a set of n separate ingestions can be calculated from the binomial expansion of $(p + q)^n$, assuming a constant p over all n trials. The probability of surviving all n ingestions is p^n, while the probability of not surviving is given by the sum of the remaining terms in the expansion, which must equal $1 - p^n$. As can be seen from Figure 4, as n increases, the probability of survival rapidly approaches zero, even if the probability of surviving after one trial is fairly high. For example, even if an animal has a 50% chance of surviving a single ingestion of a particular toxin, it has only a 3% chance of surviving five separate ingestions. Therefore, any specialized learning abilities which could reduce the number of trials required before an animal learned to avoid a toxic food would be exceptionally favorable traits, subject to intense selection pressure. Thus, despite the potential infrequency of encounters with unpredictably toxic substances, natural selection would still highly favor taste aversion learning.

Considering the greatly reduced risk of toxicosis-caused mortality accruing to an animal with highly developed taste aversion learning skills, this potential function for taste aversion learning seems well established--indeed, this is the basis of the "obvious adaptive value" assumed by the many authors cited above. However,

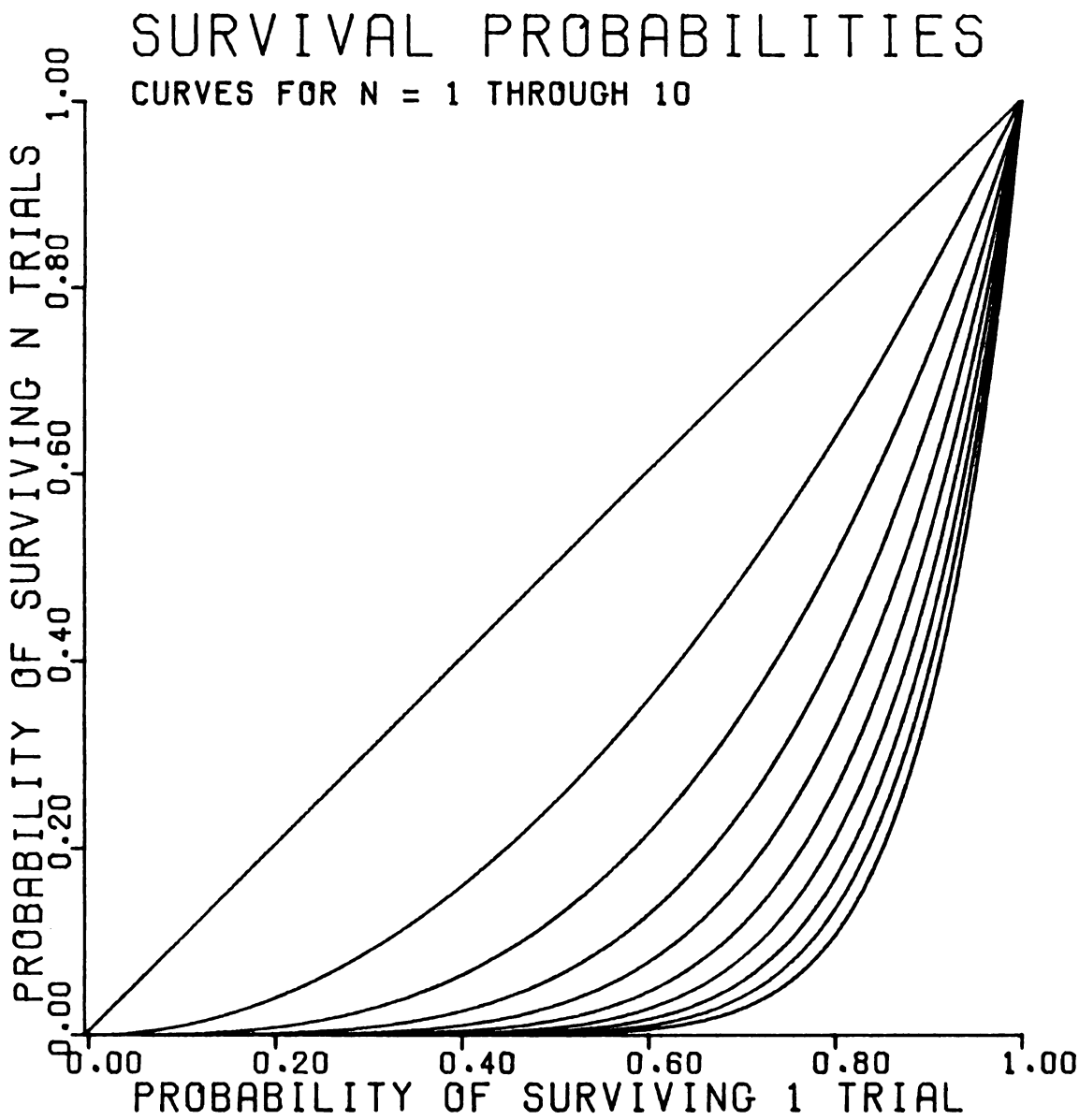


Fig. 4.--The probability of surviving n trials as a function of the probability of surviving one trial.

demonstrating that this is an important function of the phenomenon would involve showing that differential taste aversion learning abilities led to differential toxicosis-caused mortality rates under natural conditions. This has not yet been attempted. Therefore, we are forced to conclude that toxin avoidance may be an intuitively and theoretically appealing function of taste aversion learning, but it has not yet been demonstrated to be so.

Plant Secondary Compounds

In a major review, Freeland and Janzen (1974) have pointed out that, "Each plant and plant population produces a relatively distinct set of defensive chemicals (so-called secondary compounds), . . . [and that] these compounds can exist in nature in concentrations far exceeding those required to cause death or severe physiological damage to almost any mammal that might eat them." They further note that herbivores have two possible strategies for dealing with the presence of these compounds: (1) avoidance and (2) detoxification.

Taste aversion learning may not only play an important role in the avoidance strategy, but in the detoxification strategy as well. Freeland and Janzen cite evidence to show that the herbivore's detoxification system usually must be induced by low level exposure to the toxic compound before full scale detoxification can occur, and that this is true with the herbivore's own enzymes and with those of detoxifying symbiotic gut flora. In view of this, the authors postulate some behavioral attributes for an optimal herbivore:

> When an herbivore encounters a new food, we expect the animal to treat the food with extreme caution and only consume small quantities until it has determined the capacity of its detoxification

> system for that particular set of compounds. To avoid physiological damage, mammalian herbivores should be capable of learning to eat, or not to eat, a specific food with the ingestion of extremely small quantities, and preferably need only one trial. They should have a very good memory system for the flavors and tastes associated with plants and plant parts.

This is virtually an exact description of the attributes already demonstrated in taste aversion learning studies.

Of course, this correlation between the observed attributes of taste aversion learning and the predicted attributes of an optimizing herbivore does not prove that taste aversion learning is an adaptation to herbivory, but it is certainly suggestive. Furthermore, the fact that taste aversion learning can be advantageous to animals eating dangerous compounds which they can detoxify greatly increases the potential generality of the phenomenon and the likelihood of selection in its favor.

Predation on Model/Mimic Systems

Taste aversion learning and predation on model/mimic systems are related both theoretically and practically. The theoretical relationship is readily apparent in a statement of the necessary characteristics of mimicry:

> To maintain that a given relationship between a predator and two or more prey species represents an example of Batesian mimicry, a minimum of four conditions must be met:
>
> (1) The predator's reaction to the model species must be known, i.e., it must be demonstrated that the predator will show an aversion to the model as a function of prior experience with it . . . .
>
> (2) The predator must be deceived by the signal emitted by the mimic in that he responds to the mimic as if the mimic were in fact the unpalatable model; furthermore, the predator's avoidance of the mimic must be a function of his experience with the model . . . .

> (3) The signal that the predator responds to in his rejection of the mimic must be of a similar nature to the signal that he uses to reject the model . . . .
>
> (4) The mimicry complex must be explainable in evolutionary terms . . . .
>
> Czaplicki, Porter, and Wilcoxon, 1975

The first three of these statements could just as accurately describe the conditions required for the demonstration of a learned taste aversion. However, the striking congruency of these two paradigms is not offered as proof that taste aversion learning evolved as an adaptation to predation on model/mimic systems, nor even that the evolution of taste aversion learning was significantly affected by selection resulting from predation on model/mimic systems. Indeed, this would be disputed by the known occurrence of taste aversion learning in herbivorous species such as the guinea pig (Braveman, 1974, 1975) which have little likelihood of encountering such systems. On the other hand, the usefulness of taste aversion learning to predators encountering mimicry is apparent. Theoretical considerations (Estabrook and Jesperson, 1974; Bobisud and Potratz, 1976) have suggested that optimizing predators encountering a model/mimic system should learn to avoid the entire system after a single encounter with a toxic model, and single trial learning has been one of the most consistent findings in taste aversion studies. Thus, taste aversion learning and optimal predation upon model/mimic systems are similar in their specific parameters as well as in their overall topography.

This apparent similarity between taste aversion learning and optimal predation response to model/mimic systems is not without problems. Taste aversion learning has been characterized by the

specific salience of taste cues, while most documented cases of mimicry involve visual cues (Alcock, 1975). But, closer inspection reveals two lines of reasoning that may counter this objection: (1) The prepotence of taste cues (see above, pp. 16-30, for a detailed discussion) has been firmly established only in rats and guinea pigs. In quail, the only other species specifically tested for cue modality hierarchy, vision was found dominant. This has led Rozin and Kalat (1971) to suggest, "that the critical dimension for poison-based aversion learning may not be 'taste versus other modalities' but 'eating-related cues versus other cues' . . . . Eating-related cues would be whatever type of cue--gustatory, visual, or otherwise--a particular species uses to identify food." Further work may show vision to be the dominant modality in poison-based aversion learning for animals that frequently encounter visual mimicry. If this proves to be the case, a within-modality cue hierarchy might also be found, with "warning colors," oranges and yellows, being most salient. This possibility is not entirely hypothetical: Johnson, Beaton, and Hall (1975) have shown that green monkeys can form poison-based aversions to strictly visual cues and that aversions directed toward yellow cues are more pronounced and more long lasting than aversions toward blue cues. Since, however, Johnson et al.'s work was not even indirectly concerned with the question of mimicry, their results must be treated with caution in this context. (2) Even granting that most poison-based aversion learning is limited to taste cues and that most cases of mimicry involve visual cues, the two phenomena may still be closely related. Brower's work on the blue jay and the monarch

butterfly (summarized in Brower, 1969) suggests strongly that the jay's learned aversion is first directed to the taste of the butterfly, then secondarily transferred to the appearance of the butterfly. Perhaps even with visual mimicry, taste cues are still particularly important.

In summary, although the existence of poison-based aversion learning in animals not known to encounter model/mimic systems indicates that dealing with mimicry is not an essential function of the phenomenon, the similarity of the two and the demonstrable usefulness of poison-based aversion learning for predation on models and mimics suggests that the apparent relationship between them may not be entirely fortuitous. And, if further work does show a greater salience for "warning colors" in known predators of model/mimic systems, the possibility of a significant functional relationship will be enhanced.

Finally, the practical relationship between poison-based aversion learning and predation upon model/mimic systems needs to be mentioned: the close similarity between the two systems suggests that taste aversion paradigms might provide a useful experimental tool for studying mimicry. Czaplicki, Porter, and Wilcoxon (1975) have already employed poison-based aversion learning to demonstrate the possibility of olfactory mimicry, and the phenomenon could also be used to test the parameters predicted from various optimizing theories. Few areas in theoretical ecology have such a tractable laboratory paradigm at their disposal. It is hoped that more workers begin to exploit it.

Selection of Specific Nutrients

Even when faced with an incredibly complex assortment of potential foods of widely differing nutritional values, animals are capable of selecting a balanced and healthy diet. This has been demonstrated in the field (by the mere existence of healthy animals) and in the laboratory (Richter, 1943). Considerable evidence exists to show that this selection is under both innate and learned control. In a major review, Nachman and Cole (1971) observed:

> In actual fact it appears that there are at least two quite different mechanisms by which preferences or aversions for specific foods develop as a function of need state:
> (1) A nutritional deficiency may result in a direct change in the acceptability or palatability of particular substances. It is as if the innate preference-aversion curve for a substance changes in direct response to some physiological state. The principle and perhaps only example of this type of change in preference is the specific hunger for sodium salts.
> (2) The second mechanism involved in specific hunger behavior is seen in the much more general phenomenon in which animals in a need state will learn to develop a preference or an aversion to particular foods as a result of the beneficial or toxic consequences of ingesting those foods.

Since this second mechanism may represent an important extension of the poison-based taste aversion learning phenomenon, evidence for it will be considered in some detail.

Although it was established fairly early that B-vitamin-deficient rats could learn to prefer a diet rich in their needed vitamin (Harris, Clay, Hargreaves, and Ward, 1933; Scott and Quint, 1946), the question of precisely how such learning occurs has been addressed only recently. The primary assumption had been that the rats were learning to associate the beneficial effects of recovery from the deficiency with the taste of the new diet, thus coming to prefer the new diet. However, after it was shown that the anorexia

associated with B_1 vitamin deficiency was specific to the familiar, deficient diet and that the initial preferences for novel foods were not necessarily specific for vitamin rich-foods (Rozin, 1965; Rodgers and Rozin, 1966; Rozin and Rodgers, 1967), Rozin suggested that the rats might be learning an aversion to the deficient food rather than a preference for the enriched food. He tested this (Rozin, 1967) by comparing the response of vitamin-deficient rats toward a deficient food with the response of rats toward a food adulterated with quinine. He also tested the response of recovered vitamin-deficient rats toward the food originally associated with the deficiency. He concluded,

> Observation of deficient rats strongly suggests that a specific aversion to a familiar deficient diet occurs and persists even after the rats have recovered . . . . Most important, the fact that hungry recovered rats avoided the familiar deficient diet, when no other foods were available (thus preferring no food to familiar deficient diet), strongly suggests that the familiar deficient diet is aversive.

He went on to note the similarity of these findings with those of poison-based taste aversion studies and suggested that thiamine deficiency might be considered a slow acting poison.

One problem remained, however: both Richter (1953) and Rzoska (1953) had reported that poisoning resulted in a generally heightened neophobic food reaction, while the thiamine deficiency studies indicated a neophilic response. Rozin (1968) noted that this might be simply the result of procedural differences (in poisoning studies rats were offered familiar-safe versus novel-safe, while in deficiency studies rats were offered familiar-deficient versus novel-safe) and he tested for this possibility by comparing the responses of poisoned or deficient rats to familiar-safe, familiar-unsafe (toxin or deficiency

associated), and novel-safe foods. The results were clear: "[P]oisoning or deficiency produce an increased aversion for the 'poison vehicle' (paleophobia) and heightened suspicion of new diets (neophobia) in wild and domestic rats." This caused him to conclude that, "specific hungers (other than sodium) and poisoning responses are fundamentally similar, and consist of a learned (conditioned) aversion for the bad diet, and an increased noephobia."

Rozin's work thus appears to establish a much wider potential functional application for the poison-based aversion learning phenomenon. However, since the special case of innate control of sodium specific hunger has been well established (see evidence in reviews by Nachman and Cole, 1971; Rozin and Kalat, 1971, 1972; Rozin, 1975), might it not be that the learning involved in thiamine deficiency is another special case? Or, more specifically, is it possible, using deficiencies other than vitamin B_1, to demonstrate learning phenomena similar to those found by Rozin? The answer is yes: The reaction to riboflavin and pyridoxine deficiency seems identical to that of thiamine (Rozin and Rogers, 1967), the learned nature of the specific hunter for calcium has been established (Scott, Verney, and Morrisey, 1950; Frumkin, 1975), and the rejection of amino-acid-imbalanced diets appears to be a learned response (Leung, Rogers, and Harper, 1968; Booth and Simson, 1971; Simson and Booth, 1974). Clearly the phenomenon is not restricted to thiamine deficiencies.

However, it must be acknowledged that learned aversions do not explain specific hungers in total. A number of studies have indicated that rats learn to prefer flavors associated with increases

in physiological well being--for example, with recovery from thiamine deficiency (Garcia, Ervin, Yorke, and Koelling, 1967; Zahorik, Maier, and Pies, 1974), with hunger satiation (Revusky, 1967), and with the quenching of thirst (Revusky, 1968b, 1974). But, a direct comparison of the strength of aversions produced by thiamine deficiency with the strength of preferences produced by recovery from deficiency (Seward and Greathouse, 1973) showed the aversions to be much stronger than the preferences. Thus, although learned preferences may play some role in specific hungers, the major part seems reserved for learned aversions.

In summary, it has been shown that poison-based taste aversion learning and specific hunger learning are related, that learned specific hungers are widespread, and that learned aversions play a greater role than learned preferences in these specific hungers. Therefore, the important functional role of taste aversion learning in the selection of specific nutrients seems well established.

Optimal Foraging Models

Recently many attempts have been made to derive models which can describe diet selection by animals. (Schoener, 1971, offers a theoretical review, while Krebs, 1973, offers a behavioral review.) All of these models share one fundamental assumption: "that nature pursues economy in all her workings" (Rosen, 1967). Given this assumption that optimization does occur, the individual models further assume that predators can estimate the quantity and quality of prey in their environment, and then, after making additional simplifying assumptions, the models finally conclude by showing how an optimizing

predator should select its diet given a particular distribution and abundance of prey types. Most authors have defined optimality by net caloric gain (for example: Schoener, 1969, 1971; Emlen, 1966, 1968; Pulliam, 1974; Griffiths, 1975; Rapport, 1971; Charnov, 1976; Werner and Hall, 1974; MacArthur and Pianka, 1966), while a few have included specific nutrient constraints (Pulliam, 1975; Westoby, 1974), and others have considered the special problems presented by mimicry (Holling, 1965; Estabrook and Jesperson, 1974; Bobisud and Potratz, 1976). Despite their differing emphases, most of these have reached the same general conclusion: The breadth of an optimizing predator's diet will vary inversely with overall prey availability. However, they differ in their specific predictions depending upon their specific assumptions.

A few authors have attempted to test these predictions and their efforts have produced inconclusive results. Werner and Hall (1974) presented bluegills with varying densities of different sized Daphnia and found that the fish did vary their diet breadth as predicted. Krebs, Ryan, and Charnov (1974) examined the foraging tactics of chickadees and concluded that the birds' behavior was more consistent with an optimal foraging model than with a hunting by expectation model (see Krebs, 1973, for a discussion of the hunting by expectation model). Charnov (1976) re-examined data (obtained earlier by Holling) on the prey selection of mantids and concluded that the mantids' behavior was as predicted from optimal foraging models. However, Emlen and Emlen (1975) offered Swiss-Webster mice seeds of varying net energetic value and obtained results which could be fitted to an optimal foraging model only after extensive a posteriori

manipulation and adjustment. Furthermore, Kear (1962) and Willson (1971) both found that finches' seed preferences cannot be predicted simply on the basis of efficiency in handling. Since an optimal foraging model which defined optimality by net caloric gain (as most have) would predict that Emlen and Emlen's mice and Kear's and Willson's finches should feed according to efficiency, Krebs (1973) has commented, "the predictions made by generalized optimal foraging models are on the whole too simplistic to provide much guidance in studying behavioral mechanisms of predation." If, as Krebs has claimed, the models are incapable of generating interesting predictions, the question arises: Is the difficulty in the application of the models or in their design? I think both.

Whenever a model is applied, care must be taken to insure that the parameters fed into the model have been accurately estimated so that the predictions generated will be truly related to the situation being tested. Since optimal diet theory is concerned with a choice of diet breadth based upon prey actually encountered, an experimenter must use as a model parameter effective prey abundance, as determined by the predator's perceptual capacities, rather than absolute prey abundance, as determined by the experimenter's manipulations. Likewise, the rank ordering of prey types in terms of value must be based upon the predator's assessment of value, not the experimenter's. In their 1974 paper, Werner and Hall took pains to do just that. First, they presented a carefully reasoned argument to show why their measure of value (size class) should be equivalent the bluegills' measure of value. Then, to insure that their measure

of density would also be the same as that of the fish, they measured the bluegills' ability to detect the various sized prey, then scaled their density factors accordingly. As mentioned above, their results were in good agreement with the predictions from theory.

On the other hand, Emlen and Emlen (1975), whose results did not easily fit those predicted by theory, were not as careful in their determination of the model parameters. As their estimate of abundance they used absolute abundance of the prey types. This was not unreasonable, as the two seed types were apparently readily distinguishable by the mice. However, to estimate relative value they determined the average time required to eat each of the seed types. Then, knowing that the two seeds had equal caloric value and basing their estimates of value strictly upon net caloric gain, they considered the most rapidly eaten seed the most valuable. However, they noted that the seeds differed not only in ease of consumption, but also in taste, color, size, hardness, and probably odor. Thus, if the mice possessed any innate value judgements (food preferences) based upon taste, color, size, hardness, or odor which were as strong or stronger than their learned value judgement based upon ease of consumption, then Emlen and Emlen's estimation of relative prey value was simply incorrect and their theoretical predictions were quite unrelated to the situation actually being tested. The possibility of innate value judgements based on taste is not hypothetical. A genetic component in taste preferences has been demonstrated in rats (Nachman, 1959) and in laboratory mice (McClearn and Rodgers, 1961). This points up a major problem in the application of these diet selection

models: The estimates of value and density must be based upon the animal's assessment, not the experimenter's. Ideally, a completely independent set of experiments should first be used to determine the predator's perceptual capabilities and its preferences (value judgements). Only then can a test of optimization be performed which will yield results unconfounded by inaccurate parameter estimation.

The possibility that Emlen and Emlen's results were confounded by the interaction of innate versus learned value judgements also points out a difficulty in the design of optimal diet selection models: All of the models require that the animals be able to assess the value of prey items in their environment, yet very few of them postulate how the animals might do this. Specifically, there has been little or no concern regarding whether this assessment is made on the basis of genetically stored information or learned information. Since the once heated nature/nurture controversy has been fairly well resolved as a draw (see discussion in Alcock, 1975), it would seem reasonable to suggest that both mechanisms might be involved. But if two mechanisms are involved, the possibility of antagonism between them exists, as postulated in the comments above on the results of Emlen and Emlen. Since this potential antagonism between innate and learned food preferences is not merely theoretical, having been clearly demonstrated in the failure of sodium-deficient animals, but not of controls, to learn poison-based aversions toward salty tastes (Frumkin, 1975; Weisinger, Parker, and Skorupski, 1974), a complete diet selection model should include the possibility of genetic control, the possibility of learned control, and the

possibility of interaction between them. None of the extant models does this. To be sure, the models dealing with model/mimic systems of necessity invoke learning and the general model of Westoby (1974) also invokes learning, but neither of these provides for genetic control. Furthermore, Westoby's model is distinctly incomplete in that it makes no allowance for modifications in diet breadth in response to varying overall food availability. Meanwhile, the bulk of the models (e.g., Schoener, 1969, 1971; Emlen, 1966, 1968; Pulliam, 1974; Griffiths, 1975; Rapport, 1971; Charnov, 1976; Werner and Hall, 1974; MacArthur and Pianka, 1966) provide specifically for neither learned nor genetic control, but simply assume some control.

Perhaps learning's absence in diet selection models has been due to the lack of learning studies in the ecological literature. Or perhaps it has arisen from a desire to produce minimally cluttered models. Whatever the reason, however, I suggest that the issues discussed in regard to the Emlen and Emlen (1975) paper indicate that the time has come to place completeness above simplicity in the design of the models. And, I suggest that the vast psychological literature dealing with taste preferences, particularly the literature dealing with taste aversion learning, can provide powerful insight into the mechanisms involved in learned food value assessment and could be profitably incorporated into a model allowing for the interaction of genetic and learned control. Furthermore, I believe that such an integrated system would be capable of incorporating the problems of toxin avoidance, plant secondary compounds, predation on model/mimic systems, specific nutrient selection, and optimal diet breadth into

a unified model of diet selection, capable of yielding interesting predictions. An effort to produce such a model will be attempted in the Discussion Section (page 172) of this dissertation.

Functionally Oriented Study of Taste Aversion Learning

Introduction

In the preceding sections an effort has been made to establish the potential functional importance of taste aversion learning in several aspects of feeding. And, it has been suggested that an integration of learning theory and foraging theory could lead to a better understanding of diet selection. However, no suggestions have been made regarding the methods to be employed in this endeavor. This section will attempt to remedy that omission by: (1) evaluating the relevance of work that has already been done on taste aversion learning, (2) discussing the problems of functional analysis in general, and (3) offering specific suggestions for future work.

Current Studies

The existing body of taste aversion literature is indirectly of great significance to a functional analysis of the phenomenon, in that it has provided the extensive background information upon which such an analysis must be based. However, the direct relevance of this literature to a truly functional analysis is marginal, simply because that problem has not been foremost in the minds of the experimenters. As a result their work has not been designed to provide the type of information which is useful for such analysis. Furthermore, all of the studies have either been done on the white

rat or on a variety of species separated by wide phyletic gulfs. The white rat studies suffer from the possibility that their findings may be tainted by domestication. Since several authors (Richter, 1953; Rzoska, 1953; Galef and Clark, 1971b; Rozin, 1968) have reported finding taste aversion learning differences between wild and domestic rats, this problem is particularly disconcerting. The studies on other species, although contributing some functional information (for example, see the discussion above, p. 35, on Toxin Avoidance), generally suffer from the limits placed on them by the phylogenetic diversity of the animals used. If animals differ in many respects besides the behavior under consideration, comparisons between them are difficult to interpret. King (1968) has stated the matter forcefully: "Comparative studies of learning . . . are worthless if motor patterns vary among the species. [For example,] if one species jumps or runs more than the other, learning may depend upon the frequency of these responses more than on the capacity to learn." Since stimulus intensity is also known to affect learning (see Gray, 1965, for a review), he might have added that variations in sensory capacities also limit the meaningful interpretation of comparative studies. This is particularly relevant to taste aversion studies, since Kare (1971), in a review on the comparative study of taste, observed, "Each species has a sense of taste which was apparently evolved complementary to its survival. Unlike other physiological systems, which tend to have a universal functional character, diverse taste characteristics are encountered in closely related species or individuals."

Despite these limitations on the relevance of most taste aversion learning studies to a functional analysis, two efforts (Westoby, 1974; Rozin, 1975) have been made to go beyond the glib assertion of "obvious adaptive value" (cf. above, p. 30) and to incorporate taste aversion studies into a more general discussion of diet selection. However well conceived these efforts, they suffer from different, but symmetric, problems: each has placed too much emphasis on his own discipline. Rozin does a thorough job of reviewing the psychological literature on diet selection, but makes no attempt to relate these findings to ecological considerations. Indeed, he dismisses the regulation of caloric intake in less than a page (in a 56-page review), yet caloric intake has been the primary currency used in calculating cost/benefit ratios for ecological foraging models. Westoby, on the other hand, has developed a diet selection model for large herbivores that, while deriving from the ecological perspective, also includes some findings from taste aversion studies: "The psychological literature offers what is necessary if foods are to be chosen in accord with the nutritional benefits they bring. . .--'long-delay learning.'" Despite the fact that his model not only encompasses, but thoroughly depends upon psychological concepts ("Much turns upon whether these animals have and exercise long-delay learning mechanisms."), Westoby shows an insufficient grasp of the literature dealing with specific hungers and learned taste aversions. For example, his model is primarily built around the long-delay positive reinforcing values of foods providing essential nutrients despite the findings of Seward and Greathouse (1973) that learned aversions are much stronger than

learned preferences in the recovery from nutrient deficiency and despite the suggestions from recent evidence (Simson and Booth, 1973) that long-delay learning may be less applicable to nutritional reinforcement than to toxin avoidance.

Thus, we see that the bulk of the taste aversion literature is not directly relevant to a functional analysis of the phenomenon and that the only efforts made to date to incorporate taste aversion studies into a general theory of diet selection have not been set upon a sufficiently broad foundation. Obviously, the time has come to offer suggestions for remedying these difficulties. However, before actual experiments are proposed, the next section will consider some of the general difficulties of functional analysis. Then the concluding section will offer specific suggestions for future work.

Functional Analysis

The function of a behavior may be considered on two levels. First is the proximate level--the immediate effect of the behavior. Statements at this level are simple and completely subject to empirical verification. However, they are not without problems. For example, in vertebrates the heart has the effect of causing the blood to circulate throughout the body, and few would dispute the legitimacy of the claim, "In vertebrates, the heart has the function of causing the blood to circulate." But, the heart also has the effect of causing audible heartsounds, yet many would object to the statement, "In vertebrates, the heart has the function of causing an audible heartbeat." This objection derives from the second level of the meaning of function--the ultimate effect upon the animal's survival value.

Since this relationship between function and adaptive value (or adaptedness) is pervasive, Williams (1966) has virtually equated the two and stated,

> One should never imply that an effect is a function unless he can show that it is produced by design [natural selection] and not happenstance. The mere fact of the effect's being beneficial from one or another point of view should not be taken as evidence of adaptation. [For example,] under these rules it is entirely acceptable to conclude that a turtle leaves the sea to lay eggs, but not that a lemming enters it to commit suicide.

Although this equation of function with survival value eliminates the confusion surrounding statements such as, "The heart has the function of causing an audible heartbeat," it does, unfortunately, bring with it all of the teleological stickiness that often contaminates discussions of adaptation. However, this problem is not insoluble. Indeed, Tinbergen (1963) has said:

> I have always been amazed, and I must admit annoyed as well, when I met, among fellow-zoologists, with the implied or stated opinion that the study of survival value must necessarily be guesswork, and that exact experimentation on the problem is in principle not possible. I am convinced that this is due to a confusion of the study of natural selection with that of survival value. While I agree that the selection pressures which must be assumed to have moulded a species' past evolution can never be subjected to experimental proof, and must be traced indirectly, I think we have to keep emphasizing that the survival value of the attributes of present-day species is just as much open to experimental inquiry as is the causation of behavior or any other life process.

While few current authors would claim that the assessment of function is <u>in principle</u> impossible (even Williams, who claimed in <u>Adaptation and Natural Selection</u>, 1966, that, "biologists have no logically sound and generally accepted set of principles and procedures for answering the question: 'What is its function?'", went on to devote the last chapter of that book to establishing criteria to be used in

assessing function), most would claim that it is difficult and not to be accomplished by a brief inspection of "obvious adaptive value."

Several authors (for example, Beer, 1973; Dewsbury, 1973a, 1973b; Hailman, 1965; Hinde, 1970; Hinde and Tinbergen, 1958; King, 1968; Klopfer and Hailman, 1967; 1972a, 1972b; Lorenz, 1969, 1970, 1971; Tinbergen, 1951, 1963, 1972a, 1972b) have specifically addressed the problem of functional analysis of behaviors. Despite differences in emphasis, their combined thinking may be fairly summarized as: Two principal methods may be employed in the study of behavioral function. The first is experimental manipulation, either natural or artificial, and the second is comparison, either phylogenetic or ecological.

The first is the most favored, as it involves quantification and replication, but it is the least used, as it is difficult to apply in practice. Examples of this type are Kruuk's (1964) study on colonial nesting in black-headed gulls, Tinbergen's work (1972a) on the relationship of nest spacing and egg shell removal to predation in gulls, and Blest's (1957a, 1957b) demonstration of the function of eye spot displays in lepidoptera. All of these studies share one attribute in common: each studied a behavior which intimately involved a physical object that could be experimentally manipulated: nests could be moved, egg shells could be added, and the visibility of eye spots could be modified. It is this need to have some <u>thing</u> to manipulate that has prevented the application of this method to behaviors in general. The controlled experimental modification of a single behavior is, if not in some cases impossible,

considerably more difficult than the modification of physical objects.

The second method, the comparative, is less precisely quantifiable and thus less preferred. However, since it can be applied in circumstances when direct experimental intervention cannot, it has been frequently used--occasionally with great success (for many examples, see discussions by Hinde and Tinbergen, 1958; Tinbergen, 1972a, 1972b; Lorenz, 1970, 1971). The method, as it is commonly perceived, is aimed at adaptive correlation, either by the study of adaptive radiation (by comparing the differences in closely related, but ecologically diverse species) or of adaptive convergence (by studying behavior similarities in ecologically similar, but phylogenetically diverse species). But, as with any other method, certain restrictions must be adhered to if optimal results are to be obtained. The most important restrictions are those which insure comparability of results. As has been noted above, if animals differ in too many details besides the variable in question, interpretation is difficult. Dewsbury (1973b) has pointed out that although wide phyletic comparisons may provide general bits of information of some interest, they are rife with procedural and interpretational difficulties. But, he continues, "Many of the problems inherent in work with widely different taxa are minimized in work within more restricted taxonomic units . . . . This method is particularly useful when a good sample of the species comprising the genus or family is available for study." He then goes on to recommend mice of the genus Peromyscus as being particularly well suited for a comparative study, and concludes,

"[A] comprehensive approach within a restricted taxonomic unit . . . may represent the greatest hope for success with the method of adaptive correlation." Hinde (1970) has gone even further, recommending the study of intraspecific, or subspecies, differences, "as they lead to study of evolutionary changes in progress."

Thus, although many authors freely admit the drawbacks of comparative adaptive correlation studies, they have been forced to admit their usefulness:

> Adaptive correlations must be treated as what they are--somewhat speculative attempts to relate behavior to selective pressures. Such data are not the result of precise, controlled laboratory experiments and cannot be evaluated as such. [But,] the questions they are designed to answer are of such importance that some tolerance of ambiguity may be appropriate (Dewsbury, 1973b).

Even Hailman, who once criticized Lorenz for failing to understand the scientific method (see Lorenz, 1971, page xix), has seen fit actually to employ the comparative method of adaptive correlation and has concluded that although it may not be entirely satisfactory in quantitative rigor, "It is . . . a vast improvement over glibly assigning a 'selective advantage' to a particular character just because to do so seems 'reasonable' a priori" (Hailman, 1965).

Furthermore, the comparative method should not simply be considered a slightly distasteful method of last resort, to be employed only if experimentation is impossible, but also as a useful, perhaps essential, prerequisite to experimentation. For example, Tinbergen's (1972a) study of the adaptive value of egg shell removal has been cited above as an experimental test of adaptive value. However, Tinbergen was unable to perform this demonstration until a comparative investigation had allowed him to winnow the many potential functions

to find that one most reasonable to test. In fact, an examination of Tinbergen's very successful study reveals the following procedure: (1) identify, characterize, and name the phenomenon; (2) postulate potential functions for the phenomenon; (3) use comparative information to eliminate some of these functions; and (4) experimentally test the remaining functions. Since this stepwise procedure is clearly more parsimonious than a random testing of arbitrarily chosen potential functions, it is perhaps time to recognize that the comparative method is not merely an acceptable, but also a desirable approach to the problem of functional analysis.

In summary, most, if not all, authors agree that the best method to be employed in the study of function is experimental manipulation. However, they also agree that this is often not immediately feasible and that the comparative adaptive correlation method then becomes the procedure of choice.

Future Work

The preceding materials have endeavored to show that taste aversion learning is a powerful phenomenon, deserving of attention as a potentially important contributor to the diet selection mechanisms of animals. Yet the preceding materials have also endeavored to show that the existing taste aversion literature cannot provide a sufficient foundation upon which to develop such a general and functional interpretation of the phenomenon, and that this limitation derives primarily from the concentration of taste aversion studies upon either domesticated animals or animals of widely divergent taxa. In terms of the stepwise analysis of function derived from Tinbergen:

(1) the phenomenon has been too narrowly characterized, having been studied in depth only among domestic animals, (2) very little attention has been paid to potential functions other than toxin avoidance (see discussion above, beginning on page 34), (3) a comparative study, free from the great problems inherent in comparisons across wide phyletic gulfs, has yet to be attempted, and (4) until more information has been generated from steps 1 through 3, experimental testing is completely out of reach.

The question, then, is how best to remedy these problems. This must be approached from a theoretical and a practical view point. From a consideration of the steps above, the theoretical approach seems obvious: First, a detailed investigation of taste aversion learning must be performed in a nondomesticated species. Second, a systematic consideration of potential functions for taste aversion learning must be carried out (this has been attempted above, pp. 34-54). Third, a comparative investigation should be performed in an effort to link ecological variables to taste aversion variables. Finally, once these other steps have been performed, enough information, hopefully, will have been obtained to allow for a meaningful experimental investigation. On the practical side, the question is: which species will make the best experimental preparation? For the detailed investigation in a nondomesticated species the criteria are reasonably apparent: the animals should be abundant, not too specialized in their diet so that the results will be more generally applicable, easy and inexpensive to obtain and rear, and relatively small so that large numbers may be handled conveniently. For the

comparative study, the optimal animals would belong to a genus that has many species and subspecies occurring in a variety of habitats. Preferably some closely related species or subspecies should occupy distinctly different habitats so that adaptive radiation might be studied, while other more distantly related members of the genus should occupy similar habitats so that adaptive convergence might also be studied. And finally, it would be most advantageous if the same animals could be used in both the in-depth and the comparative studies. Are there animals that meet all of these criteria? The answer is: yes, mice of the genus Peromyscus. Barry (1975) has discussed the many advantages of Peromyscus as subjects for laboratory studies, and Dewsbury (1973b) has already been mentioned as specifically recommending Peromyscus for comparative studies. As a consequence of all these considerations, I have chosen to perform an investigation of taste aversion learning in Peromyscus. That study will be reported in the remainder of this dissertation.

GENERAL METHODS

Introduction

The series of experiments reported in this dissertation has been designed to study various attributes of the taste aversion learning phenomenon in mice of the genus Peromyscus. Methods and techniques common to all of the experiments are described in this General Methods section. Specific methods employed in individual experiments are described with those experiments.

Experimental Subjects

The mice used in these studies were from two taxa: Peromyscus maniculatus bairdi and Peromyscus maniculatus blandus. The P. m. bairdi were further subdivided into a semi-domestic group and an F_1-from-wild-caught group. The domestic P. m. bairdi were derived from mice maintained in the laboratory since their original capture near Ann Arbor, Michigan, in 1948. The F_1 P. m. bairdi were the first generation of laboratory-born animals from original stocks captured on the Michigan State University campus and at Dansville, Michigan. The P. m. blandus were F_2 and F_3 descendants from original stocks which were wild caught in 1970, 1974, and 1975 near Rodeo, Hidalgo County, New Mexico.

Maintenance

The animals were housed in two separate rooms during these studies: a breeding room (colony room) and an experimental room. Both rooms were fixed on the same 15 hours light, 9 hours dark cycle. In both rooms the temperature ranged from 20°C to 25°C and the relative humidity from 50% to 90%.

The mice were housed in plastic laboratory cages measuring 6" by 12" by 6" deep, with wire mesh lids. Wayne Breeder Blox and water were provided ad libitum to the animals in the colony room. Wayne Breeder Blox were provided ad libitum to the animals in the experimental room, but the fluid availability varied according to the experimental paradigm. None of the animals was used in more than one experiment and none had experience with a fluid other than tap water nor a food other than lab chow prior to its participation in an experiment. All mice were provided with a bedding of wood shavings and with cotton nesting material.

Young mice were housed with their parents until weaning at 21 days of age. Mice used as adults (100 to 160 days of age) were housed as litter mates until the experimental procedures were begun. All tests were conducted upon animals housed individually.

Basic Schedule

In all of these studies a flavored fluid, rather than a flavored solid food, was used. Toxicosis was induced by the intraperitoneal (IP) injection of a lithium chloride solution. Injection of the LiCl, rather than including it in the drinking fluid, was

chosen in order to deny the animals the flavor of LiCl as an additional, and essentially uncontrollable, cue.

To produce predictable drinking at a given time, the mice were placed on a regular cycle of fluid-deprivation/fluid-availability. Pilot studies had indicated that simply restricting the fluid availability to 20 minutes per day, as has been done in several rat studies, would produce an unacceptably high mortality rate. However, very low mortality was observed when 24-hour periods of fluid deprivation were alternated with 24-hour periods of ad lib. drinking. Thus, the basic schedule outlined in Figure 5 and Table 1 was developed and adhered to in all of these studies, except the pilot studies.

A step by step discussion of Figure 5 is required to explain this schedule: On day 0 the animals are in their colony cages and have water available until 1300 hours, when they are placed individually into experimental cages. Their water is removed, beginning a 24-hour period of water deprivation. On day 1, a drinking tube filled with water is placed on the cage, left for 20 minutes, then removed and the amount consumed recorded. The arrow at this point in Figure 5 indicates the recording of data. Immediately following the recording of data, the tube is refilled, replaced upon the cage, and left in position for approximately 24 hours. On day 2, the drinking tube is removed from the cage and the amount consumed recorded. Again, an arrow indicates this recording of data. This alternation of fluid-availability/fluid-deprivation is then continued for as many days as required for the individual experiment. As can be seen from Figure 5,

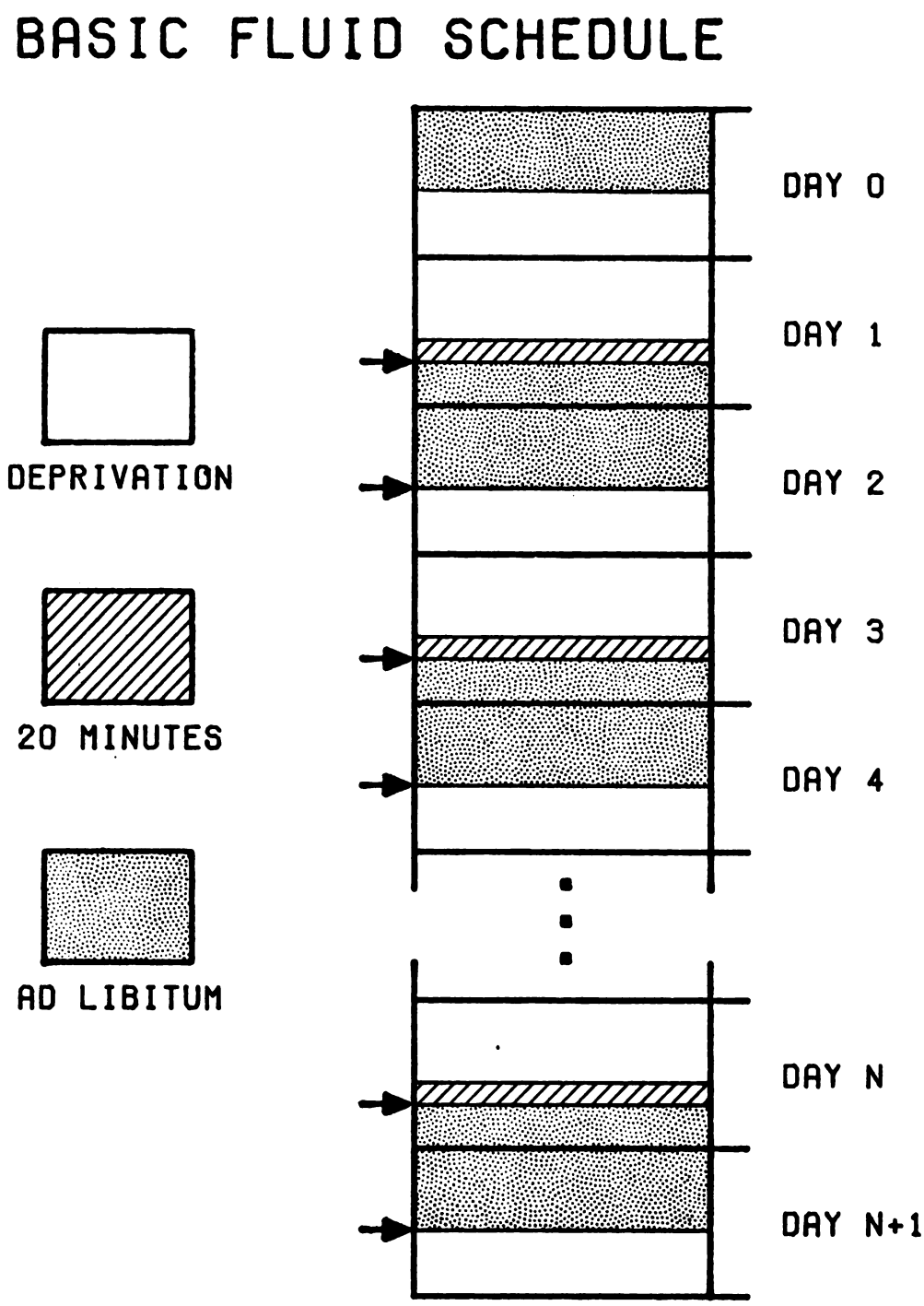


Fig. 5.--Basic fluid deprivation/availability schedule followed in all experiments. The arrows indicate times of data recording.

Table 1.--Basic fluid deprivation/availability schedule followed in all experiments.

Day	Procedure	Data Recorded
0	Animal placed in apparatus, fluid removed	Nothing
1	Fluid tube placed on cage for 20 minutes; then removed and consumption recorded; then refilled and replaced on cage.	Fluid consumption during the 20-minute period
2	Fluid tube removed from cage; consumption recorded; tube not replaced.	Consumption during the previous 24 hours
3	Fluid tube placed on cage for 20 minutes; then removed and consumption recorded; then refilled and replaced on cage.	Fluid consumption during the 20-minute period
4	Fluid tube removed from cage; consumption recorded; tube not replaced.	Consumption during the previous 24 hours
.	.	.
.	.	.
.	.	.
n	Same as 1	Same as 1
n + 1	Same as 2, plus animal removed from apparatus and experiment terminated.	Same as 2

this schedule involves data recording on every day, but on the odd numbered days of the paradigm the data represent fluid consumption during a 20-minute period following a 24-hour deprivation, while on the even numbered days the data represent total fluid consumption during a period of approximately 24 hours.

In every experiment, tap water was offered during both the 20-minute and the 24-hour drinking periods until nearly all animals had learned the schedule and were drinking reliably during the 20-minute periods. Once this criterion was obtained, the individual experiments were begun. Each experiment followed the same basic schedule given in Figure 5 and Table 1, differing only in the presentation of fluids other than tap water during the 20-minute drinking periods. Injections, when given, immediately followed a 20-minute drinking period. Injections were administered by moving the animal and its cage to a large handling arena located at the doorway joining the experimental room and the breeding (colony) room. The animal was removed from its individual cage briefly for the actual administration of the injection. Following injection, the animal was immediately returned to its cage and the cage returned to its original location in the experimental room. Because of the well known interaction between diurnal biorhythms and toxicosis (Bünning, 1973; Luce, 1971), the 20-minute drinking periods began at the same time every day: 1300 hours.

Assignment to Treatment Groups

Because the experiments were long lasting, because they required that the animals rapidly learn the basic fluid schedule, and because they involved the injection of toxic substances, some animals

were dropped from an experiment for the following reasons: (1) failure to drink at least 0.5 ml of fluid on the day of injection (this was necessary to insure the flavor/toxicosis contingency); (2) death (all data from animals not surviving the entire experiment were dropped); and (3) pregnancy (prior to experimental use animals were housed as littermates--this resulted in occasional pregnancies).

The unpredictable nature of these drops, together with space and animal number limitations, made it necessary to schedule the animals assigned to various treatment groups with the following considerations: (1) a desired size (n) was determined for each treatment group; (2) some number of animals (less than n) was assigned to each treatment group and begun on the appropriate experiment; (3) as space and/or mice became available, additional mice were randomly selected and assigned to the treatment groups in a balanced manner; (4) as soon as n animals completed a given treatment, no further animals were assigned to that treatment; (5) if by chance more than n animals completed a given treatment, only the first n to complete the treatment were used in the analysis; (6) data analysis was not begun until all treatment groups were completed. Thus, all treatments had the same number of animals completing the experiment although different numbers may have been initially assigned to each treatment group.

Flavor Stimuli

In all of the experiments a 20% (grams:milliliters) sucrose solution was used. This was prepared using commercially available cane sugar and distilled water. In the experiment dealing with the effects of various flavors, three additional flavors were used: (1) a sour

solution, prepared by diluting 0.75 ml of 37% HCl in 500 ml of distilled water; (2) a bitter solution, prepared by dissolving 3.0 grains of quinine sulfate in 400 ml of distilled water; and (3) a salty solution, prepared by dissolving 5.85 grams of NaCl in 1000 ml of distilled water (giving a 0.1 molar solution). All solutions were mixed the day of use.

The concentration of any flavor used in a taste aversion study must fall within two limits: (1) it must be strong enough that the animals can detect it, and (2) it must not be so strong that the animals do not accept it. Within these limits the choice of concentration is somewhat arbitrary, although Dragoin (1971) indicated that the strength of an aversion formed by rats was dependent upon the concentration of the flavor offered as the CS. However, he explained his results on the basis of detectability, as described above. For a preferred flavor, such as sucrose, the range of detectable and acceptable concentrations is rather wide. Since a 20% sucrose solution had been used successfully in studies on white rats (Revusky, 1968; Revusky and Bedarf, 1967), and since pilot studies indicated that this concentration was both detectable and acceptable to Peromyscus, this concentration was chosen for these studies. For other flavors, the range between acceptability and detectability may be very narrow. As a result, extensive pilot studies were carried out to determine the appropriate concentrations for the sour, bitter, and salty flavors.

Measurement of Consumption

The measurement of fluid consumption requires some type of reasonably accurate calibrated drinking bottle. Early pilot studies indicated that a very acceptable, yet inexpensive (less than 15¢ each), device could be produced by modifying a 10 cc disposable plastic syringe in the following manner: (1) the standard tip of the syringe is removed by passing a 1/4" drill bit through the tip; (2) the opening thus produced is further reamed and smoothed by redrilling with a 5/16" drill bit (this step is essential to prevent rough bits of plastic from capturing air bubbles and blocking the flow of fluid); (3) a drinking spout is manufactured by cutting 8 mm OD glass tubing to the desired length, then rounding the opening slightly in a flame; (4) the spout is attached to the modified syringe with a collar of 1/4" ID Tygon tubing. The resulting calibrated drinking tube can be easily filled, in a manner similar to a fountain pen, simply by dipping the tip into the desired fluid and pulling the plunger--this allows all tubes to be filled to the same level. Fluid consumption can then be read from the calibrations on the syringe. Readings to 0.1 ml can be obtained from 5 ml and 10 ml syringes.

Statistical Methods

The primary statistical tool employed throughout this dissertation was the t-test. This simple but powerful test has the great advantage of being both easy to apply and easy to interpret. Since most of my experiments yielded results that could most readily be interpreted in terms of pairwise comparisons, it seemed both reasonable and prudent to use the t-test. However, whenever multiple

comparisons are made within one experiment, there exists with any test an increased overall probability of committing some Type I error (false rejection of the null hypothesis). That is, with multiple comparisons in an experiment, the results must be interpreted with caution if an excessive danger of some Type I error is to be avoided.

Just how cautious this interpretation must be can be shown using the binomial expansion to consider the case when N independent comparisons are to be made. Assume that the null hypothesis is true and let p = the probability that each test will individually show no significance and let q = the probability that each test will show apparent, but spurious significance (note that $p + q = 1.0$). That is, according to customary usage, let $q = \alpha$ and let $p = (1 - \alpha)$. Then the expansion of $(p + q)^N$ can be used to determine the overall probability of encountering any spuriously significant results in the N comparisons. Note that p^N gives the probability that no Type I error will occur in any of the tests and that $1 - p^N$ gives the overall probability that one or more Type I errors will occur. Thus, $1 - p^N$ gives the value of α for the experiment as a whole. With $p = 0.95$ (i.e., with each $\alpha = 0.05$) the value of $1 - p^N$ increases rapidly with N. However, as p increases (i.e., as q decreases), the effect of increasing N on the overall probability of Type I error is greatly reduced. Figure 6 gives the overall α as a function of N, the number of independent comparisons employed in the experiment. As is readily apparent, if the α for each comparison is ≤ 0.05, the probability of committing some Type I error rapidly increases with increasing N. With only three comparisons the probability of committing some Type I error has

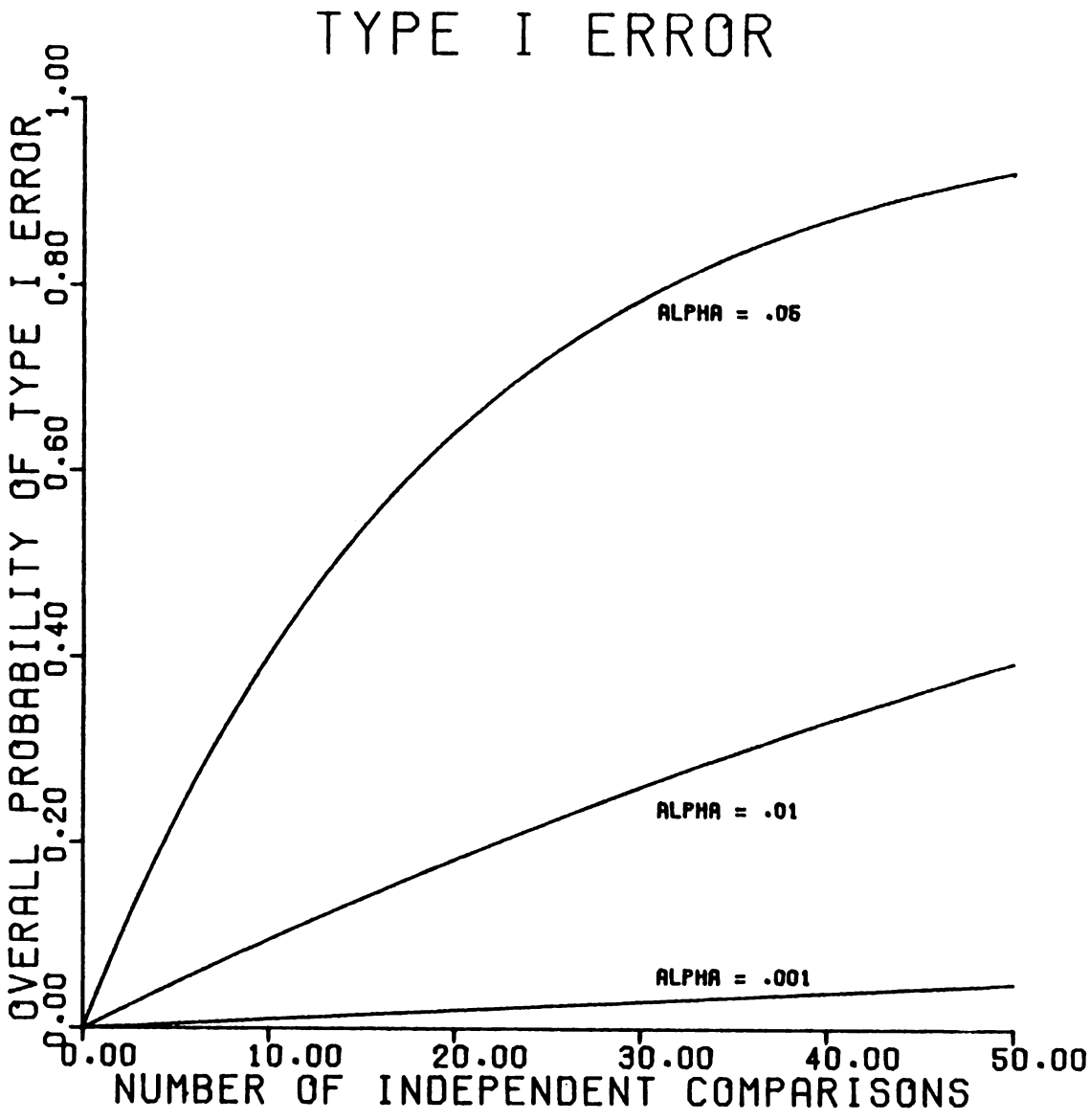


Fig. 6.--The overall probability of committing any Type I error in a multiple comparison experiment as a function of the total number of comparisons and the alpha level of each individual comparison.

increased to ≤ 0.14, with 15 comparisons it is ≤ 0.54, and with 50 comparisons it is ≤ 0.92. When the α for each comparison is ≤ 0.01, there is more protection against Type I error, but even then once N exceeds 10 the probability of some Type I error begins to increase unacceptably. However, with $\alpha \leq 0.001$ for each comparison, the protection against any Type I error is great. Even with 50 comparisons the overall likelihood of any Type I error is ≤ 0.05. Thus, Figure 6 shows that in experiments involving multiple independent comparisons, results that appear significant at the $\alpha \leq 0.001$ level are well protected against Type I error and may be interpreted confidently, but results that appear significant at $0.001 \leq \alpha \leq 0.05$, and particularly at $0.01 \leq \alpha \leq 0.05$, are only poorly protected and must be regarded with caution.

Since pilot studies indicated that taste aversion learning is a powerful phenomenon in Peromyscus which readily produces differences at the $\alpha \leq 0.001$ level even with moderate sample sizes, the advantages of the t-test were felt to outweigh the disadvantages of multiple comparisons. Hence, the t-test was widely employed throughout this dissertation. However, in those experiments with cross-classified designs, analysis of variance was used since it provided the most straightforward testing of main effects.

PILOT STUDY I

Introduction

The first essential step in assessing the significance of taste aversion learning to Peromyscus is the demonstration that the phenomenon occurs in Peromyscus. For methodological reasons I was also interested in determining if equimolar LiCl and NaCl solutions have equivalent tastes to Peromyscus.

Purposes

The purposes of this experiment are twofold: (1) to determine if mice of the genus Peromyscus will learn to avoid drinking a distinctly flavored fluid if consumption of that fluid causes toxic effects, and (2) to determine if equimolar LiCl and NaCl solutions have equivalent tastes to Peromyscus.

Test Subjects

All of the animals in this study were experimentally naive adult (male and female) F_1 P. m. bairdi reared according to the description in the General Methods Section.

Procedure

The animals were housed individually and randomly assigned to 1 of 4 treatment groups (initially n = 13 for each, but differential mortality reduced the final numbers to 11, 12, 10 and 10 for groups

1 - 4, respectively). All animals were placed on a restricted fluid-availability schedule, with water available only during a 20-minute period (1300 - 1320) every day. After 8 days of water consumption on this schedule, the different treatment schedules were begun. During the 20-minute drinking period of day 9, groups 1 and 2 were given 0.75 ml of a 0.2 molar LiCl solution, and groups 3 and 4 were given 0.75 ml of a 0.2 molar NaCl solution. On days 10 and 11, all groups were given water. On day 12, groups 1 and 3 were offered 0.2 molar LiCl and groups 2 and 4 were offered 0.2 molar NaCl. This drinking period was limited to 10 minutes since Nachman (1963) and Rusiniak, Garcia, and Hankins (1976) have shown that for rats the onset of toxic effects from ingested lithium occurs approximately 8 - 12 minutes following the beginning of a drinking bout. Figure 7 summarizes these treatments.

Results

The results of this experiment are given in Figure 8. T-test comparisons were made among the means to determine differences. Both groups 1 and 2 showed suppressed consumption, were not significantly different from each other ($t = 1.742$, $df = 21$), and were significantly different from groups 3 and 4 ($|t| \geq 7.190$, $df \geq 19$, $p \leq 0.001$). Groups 3 and 4 were also significantly different from each other ($t = 9.113$, $df = 18$, $p \leq 0.001$).

Conclusions

The hypothesis that Peromyscus can form learned taste aversions is supported by the suppressed consumption of group 1 relative to

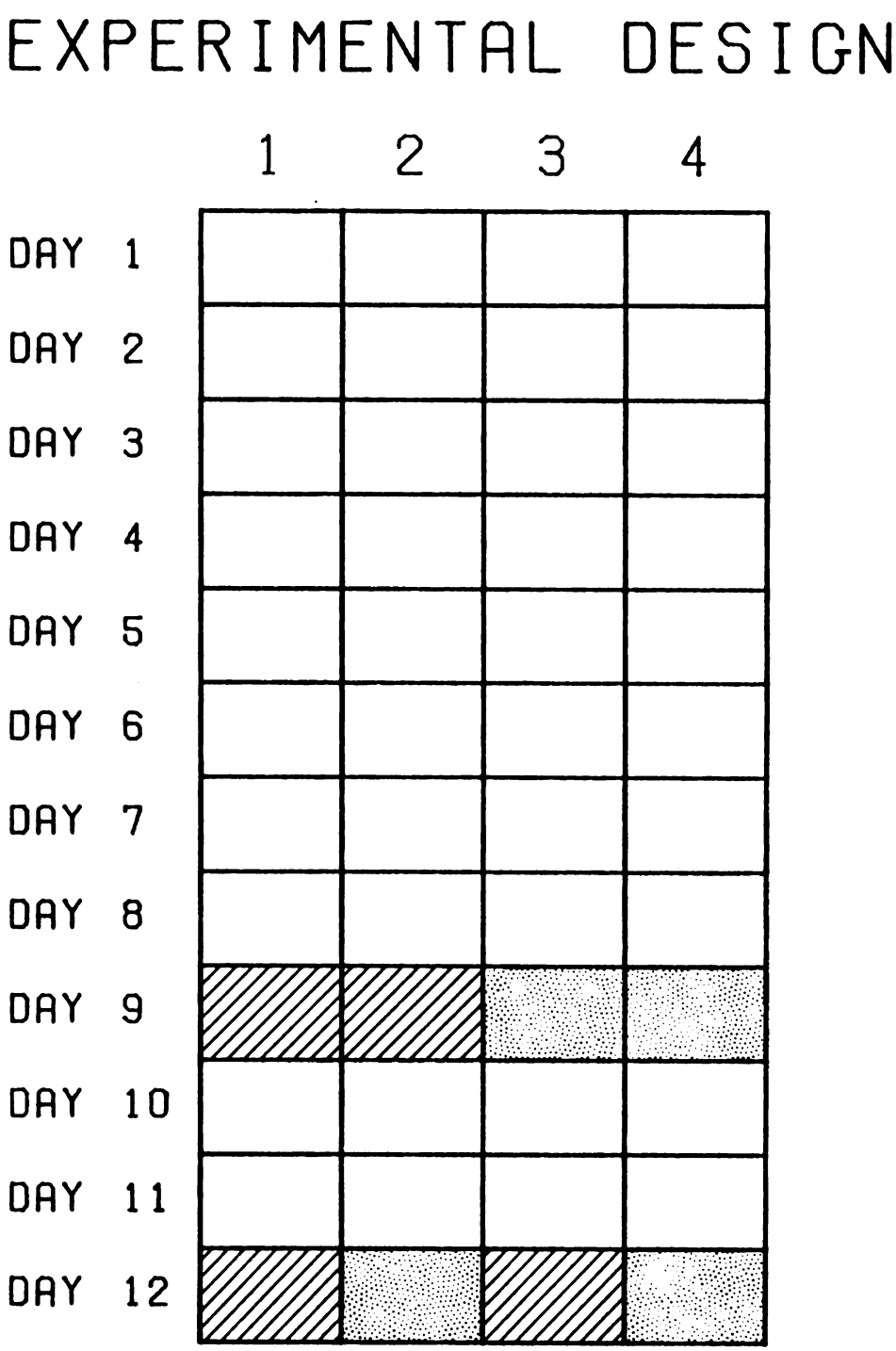


Fig. 7.--Basic fluid presentation schedule for Pilot Study I. On the days shaded with crosshatching, LiCl was offered, on the stippled days, NaCl was offered. On the remaining days, water was offered.

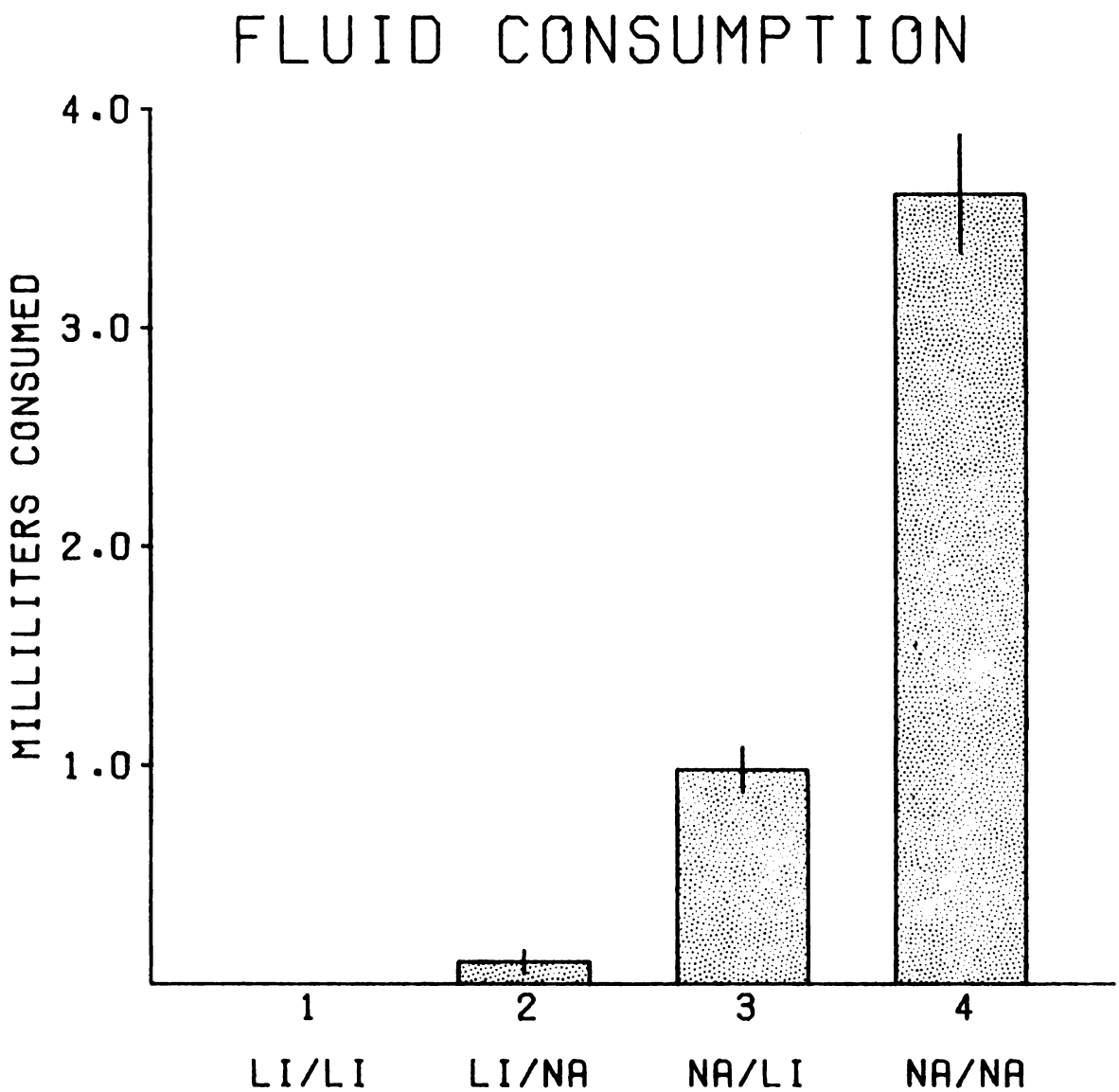


Fig. 8.--Mean fluid consumption (± 1 SE) of test groups in Pilot Study I on day 12. Salts given on days 9 and 12 are shown below abscissa.

group 3. However, the hypothesis that equimolar LiCl and NaCl solutions have equivalent tastes to Peromyscus is supported by the apparent generalization of the aversion evidenced in the suppressed consumption of group 2, but is contradicted by, or is at best inconsistent with, the significant difference between groups 3 and 4. It is possible to rescue the equivalent taste hypothesis by suggesting that the two solutions do taste the same (as supported by the lack of difference between groups 1 and 2), but that the toxic effects of LiCl appear so rapidly in Peromyscus that consumption of the 0.2 molar LiCl solution was terminated much earlier in the 10-minute period than was consumption of the 0.2 molar NaCl. Since the data from this pilot shed no light on this modified hypothesis, the only strong conclusion that can be drawn from this pilot is that Peromyscus can learn to avoid distinctly flavored fluids that have toxic effects.

PILOT STUDY II

Introduction

Pilot Study I gave inconsistent results regarding the equivalence of taste of equimolar LiCl and NaCl solutions to Peromyscus. However, it was noted that the apparent inconsistency could be eliminated if it were further hypothesized that the onset of the toxic effects of LiCl occurred so rapidly in Peromyscus that total fluid consumption in a 10-minute interval would be differentially affected for LiCl and NaCl solutions.

Purpose

The purpose of this experiment is to determine, in Peromyscus, if there is any indication that the onset of LiCl-induced toxicosis occurs within 5 minutes of the beginning of consumption.

Test Subjects

All of the animals in this study were experimentally naive adult (male and female) F_1 P. m. bairdi reared according to the description in the General Methods Section.

Procedure

The animals were housed individually in plastic cages and assigned randomly to 1 of 2 treatment groups (n = 13 for each). All animals were placed on the same restricted 20-minute per day water

availability schedule as employed in Pilot Study I. For 8 days tap water was offered to both groups. On day 9 group 1 was offered 0.2 molar LiCl and group 2 was offered 0.2 molar NaCl. Each individual animal's consumption was monitored and recorded in 10-second intervals for a total of five minutes (with time 0 taken as the moment the animal first contacted the drinking spout).

Results

The results of this experiment are given in Figure 9. The figure shows that cumulative consumption for the two groups was virtually identical for the first 90 seconds, then began to diverge rapidly, becoming significantly different at 2:00 minutes (t-test, $p \leq 0.05$). The difference from 2:00 minutes to 5:00 minutes is striking--none of the LiCl animals drank any additional fluid during these last three minutes, while all of the NaCl animals continued to drink throughout the final three minutes.

Conclusions

These results are certainly consistent with the equal taste, but rapid onset of toxic effects hypothesis. They also agree nicely with the results of Pilot Study I: In Pilot I animals freely drinking LiCl consumed a mean of 0.98 (± 0.10) ml, while in this experiment mean LiCl consumption was 0.85 (± 0.09) ml. (The free consumption of NaCl was markedly less here than in Pilot I, but this experiment involved only a 5-minute drinking period while Pilot I offered a 10-minute period.)

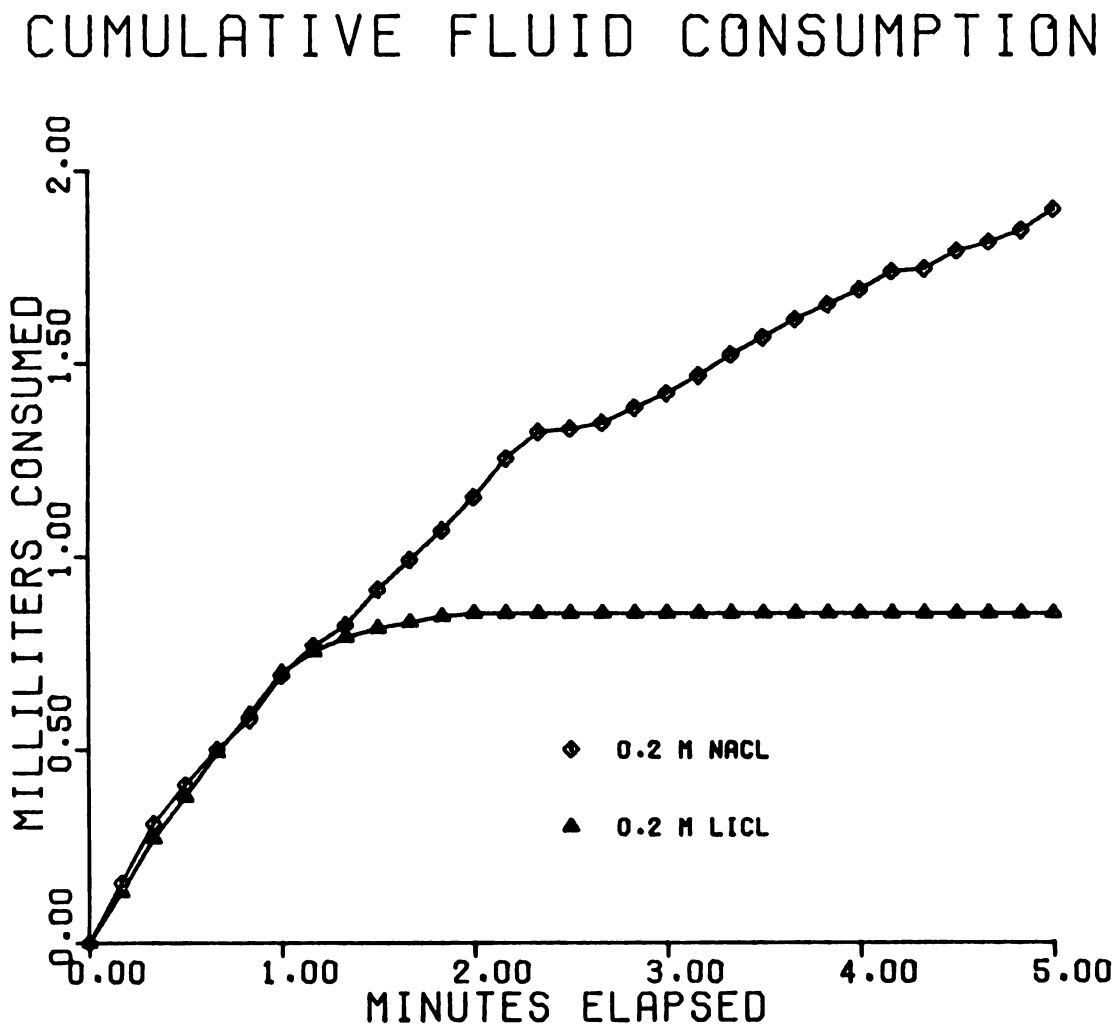


Fig. 9.--Cumulative consumption by animals drinking LiCl or NaCl solutions.

Although these results are consistent with the early onset of toxicosis hypothesis it is readily conceded that they do not establish that hypothesis--these results could just as easily be explained by a difference in a delayed aftertaste. However, it is established that some difference is detected by the mice very rapidly after the beginning of drinking--so rapidly, in fact, that any attempt to continue taste aversion studies on Peromyscus using the ingestion of LiCl would seriously confound decreased consumption produced by a learned aversion with decreased consumption due to the rapid onset of toxicosis or aftertaste. Consequently, the primary conclusion drawn from this pilot study is: Although Peromyscus initially respond identically to LiCl and NaCl solutions (possibly indicating equivalent tastes), a rapid and distinct divergence in cumulative consumption of the two fluids indicates they must be in some way distinguishable by Peromyscus.

BASIC TASTE AVERSION LEARNING I

Introduction

Although the results of the first pilot study indicated that Peromyscus can learn to avoid a distinctly flavored toxic fluid after one experience with that fluid, the results of the second pilot study indicated that there was apparently only a 90-second delay between onset of drinking and onset of toxic effects, and presumably no delay between termination of drinking and onset of toxic effects. Thus, the experimental design employed in the pilot studies provided no evidence that the mice would be capable of forming aversions if a delayed illness were paired with a distinctive flavor, nor did the pilots give any evidence regarding appropriate dosages for delayed illness.

Purposes

The purposes of this experiment are twofold: (1) to determine if adult F_1 P. m. bairdi form an aversion to a distinctly flavored fluid if that fluid is followed by a delayed lithium-induced toxicosis; and (2) to determine if the degree of such an aversion is affected by the dosage of toxin employed.

Test Subjects

All of the animals in this study were experimentally naive adult (male and female) F_1 *P. m. bairdi* reared according to the description in the General Methods section.

Procedure

The animals were housed individually in plastic cages and randomly assigned to 1 of 11 treatment groups (n = 10 for each group). All treatment groups were assigned to have one experience with sucrose (20%) followed by an injection of either a toxin or a control substance. The specific assignments were as follows: (1) no injection control, (2) 0.3 milliequivalents of LiCl per kilogram of body weight, (3) 0.3 mEq/kg NaCl, (4) 1.0 mEq/kg LiCl, (5) 1.0 mEq/kg NaCl, (6) 3.0 mEq/kg LiCl, (7) 3.0 mEq/kg NaCl, (8) 6.0 mEq/kg LiCl, (9) 6.0 mEq/kg NaCl, (10) 9.0 mEq/kg LiCl, and (11) 9.0 mEq/kg NaCl. The various dosages were obtained by varying the concentration of the solute rather than by varying the volume of the injectant.

In order to develop a regular drinking schedule in the animals, all of the groups were given water according to the basic fluid schedule (Table 1 and Figure 5) for 4 days. On day 4 each animal was weighed. On day 5 each animal was offered sucrose solution during the 20-minute drinking period, then immediately injected (IP) with the assigned substance and dose. Additional drinking fluid was withheld from the animals for 2 hours following the injections. Then drinking tubes filled with water were placed on the cages and left there for 22 hours. On day 6 the drinking during this 22-hour period

was recorded and the drinking tubes removed from the cages. On day 7 sucrose was offered to all of the animals during their 20-minute drinking bout. Following this 20-minute drinking bout the experiment was terminated.

Results

The results of this experiment are given in Figure 10. The difference between the groups injected with LiCl and NaCl is obvious. Statistical analysis (all ways pairwise t-tests) indicated that none of the NaCl groups was different from the no injection control group ($|t| \leq 1.874$, df = 18) and that none of the NaCl groups was different from any other NaCl group ($|t| \leq 1.271$, df = 18). The 0.3 mEq LiCl group was not different from the no injection group ($t = 1.091$, df = 18), the 1.0 mEq LiCl was slightly different from the no injection group ($t = 2.409$, df = 18, $p \leq 0.05$), and the 3.0, 6.0, and 9.0 mEq LiCl groups were all strikingly different from the no injection group ($|t| \geq 5.231$, df = 18, $p \leq 0.001$). Although different from the no injection group, the 1.0 mEq LiCl group was not different from the 1.0 mEq NaCl group ($t = -0.709$, df = 18). However, the 3.0, 6.0, and 9.0 mEq LiCl groups were all quite different from their matching NaCl groups ($|t| \geq 4.375$, df = 18, $p \leq 0.001$).

Conclusions

In terms of the stated purposes of this experiment, the conclusions to be drawn from the data are obvious: adult F_1 *P. m. bairdi* will avoid drinking a sucrose solution after a single pairing of sucrose consumption with a delayed lithium-induced illness, and the

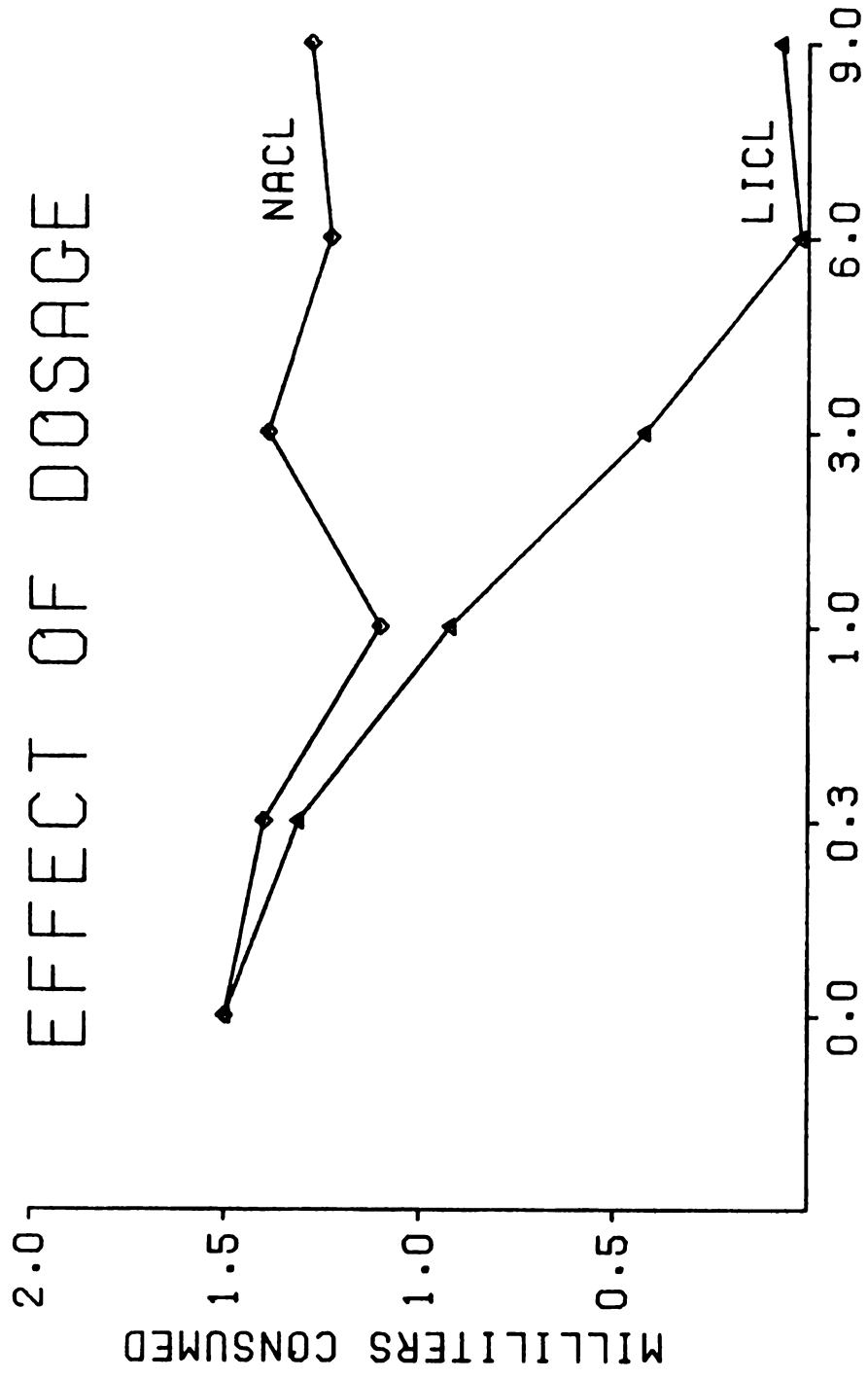


Fig. 10.--The mean effect of injectant dosage upon learned taste aversions with a delayed illness schedule.

degree of the aversion is influenced by the dosage of toxin given to the animal. However, the shape of the LiCl curve in Figure 10 shows that definite limits exist on the effect of dosage upon aversion--that is, no detectable changes in the degree of aversion are likely to occur if the dosage is decreased below 0.3 mEq or if the dosage is increased beyond 9.0 mEq. These ceiling and floor values provide guidelines which must be considered when taste aversion learning experiments using Peromyscus are designed.

BASIC TASTE AVERSION LEARNING II

Introduction

The previous experiment showed that mice which had experienced the taste of sucrose followed by a delayed lithium-induced illness subsequently avoided drinking a sucrose solution. This was taken as evidence that Peromyscus form learned taste aversions, and it was clear that the strength of these aversions is directly related to the dosage of toxin used to produce the illness. However, two alternative hypotheses exist which also could explain the results of Figure 10: (1) exposure to lithium alone is sufficient to alter the animals' reaction to sucrose--the sucrose/lithium contingency is unnecessary; or (2) the apparent aversions observed are not specific to sucrose at all, but rather represent a reduced tendency to drink any fluid presented in the experimental paradigm.

Purposes

The purposes of this experiment are twofold: (1) to determine whether the decrease in preference for sucrose shown to occur following lithium-induced toxicosis is actually dependent upon the sucrose/lithium contingency, or is derived from the toxicosis alone; and (2) to determine if the apparent aversion to sucrose is indeed specific to sucrose or is simply an aversion to drinking.

Test Subjects

All of the animals in this study were experimentally naive adult (male and female) F_1 *P. m. bairdi*, reared according to the description in the General Methods section.

Procedure

The animals were housed individually in plastic cages and randomly assigned to 1 of 4 treatment groups (n = 10 for each group). Treatment group 1 was assigned to receive a lithium injection following the ingestion of water; treatment group 2 was assigned to receive a sodium injection following the ingestion of water; treatment group 3 was assigned to receive a lithium injection following the ingestion of sucrose (20% w/v) solution; treatment group 4 was assigned to receive a sodium injection following the ingestion of sucrose solution.

In order to develop a regular drinking schedule in the animals, all of the groups were given water according to the basic fluid schedule (Figure 5 and Table 1) for 12 days. On day 12 each animal was weighed. On day 13 the different treatment group procedures were begun: the animals in group 1 were given water during their 20-minute drinking period, then immediately injected (IP) with a 0.6 M LiCl solution; the animals in group 2 were given water and similarly injected with a 0.6 M NaCl solution; the animals in group 3 were given a 20% weight/volume sucrose solution during their 20-minute drinking period, then similarly injected with the LiCl solution; the animals in group 4 were given a 20% sucrose solution, then similarly injected with the NaCl solution. The volume of the injections was adjusted so

that each animal received 9.0 mEq of solute per kilogram of body weight. Additional drinking fluid was withheld from the animals for 2 hours following the injections. Then drinking tubes filled with water were placed on the cages and left there for 22 hours. On day 14 the drinking during the previous 22-hour period was recorded and the drinking tubes removed from the cages. On day 15 water was offered to the animals during the 20-minute drinking period and consumption during this period was recorded. Then the tubes were refilled with water and replaced on the cages for 24 hours. On day 16 the tubes were removed from the cages and the water consumption during the previous 24 hours was recorded. On day 17 all groups were offered a 20% w/v sucrose solution during their 20-minute drinking period. The amount of sucrose drunk was recorded, and fresh tubes were filled with water and placed on the cages for 24 hours. On day 18 the water consumption during the previous 24 hours was recorded, the animals were again weighed, and the experiment was terminated. Table 2 summarizes these treatments.

Results

The results of this experiment are given in Figure 11. Since Figure 11 shows data for 6 consecutive days, a day by day consideration of the figure seems appropriate:

day 12 - This graph represents 24-hour water consumption for all groups on the day before the initiation of the different treatment group procedures. Analysis of variance indicates that no significant differences exist.

Table 2.--Experimental design for basic taste aversion experiment II.

Day	Treatment Group			
	1	2	3	4
13	20-minute water drinking, followed by LiCl injection	20-minute water drinking, followed by NaCl injection	20-minute sucrose drinking, followed by LiCl injection	20-minute sucrose drinking, followed by NaCl injection
14	22-hour water drinking	22-hour water drinking	22-hour water drinking	22-hour water drinking
15	20-minute water drinking	20-minute water drinking	20-minute water drinking	20-minute water drinking
16	24-hour water drinking	24-hour water drinking	24-hour water drinking	24-hour water drinking
17	20-minute sucrose drinking	20-minute sucrose drinking	20-minute sucrose drinking	20-minute sucrose drinking

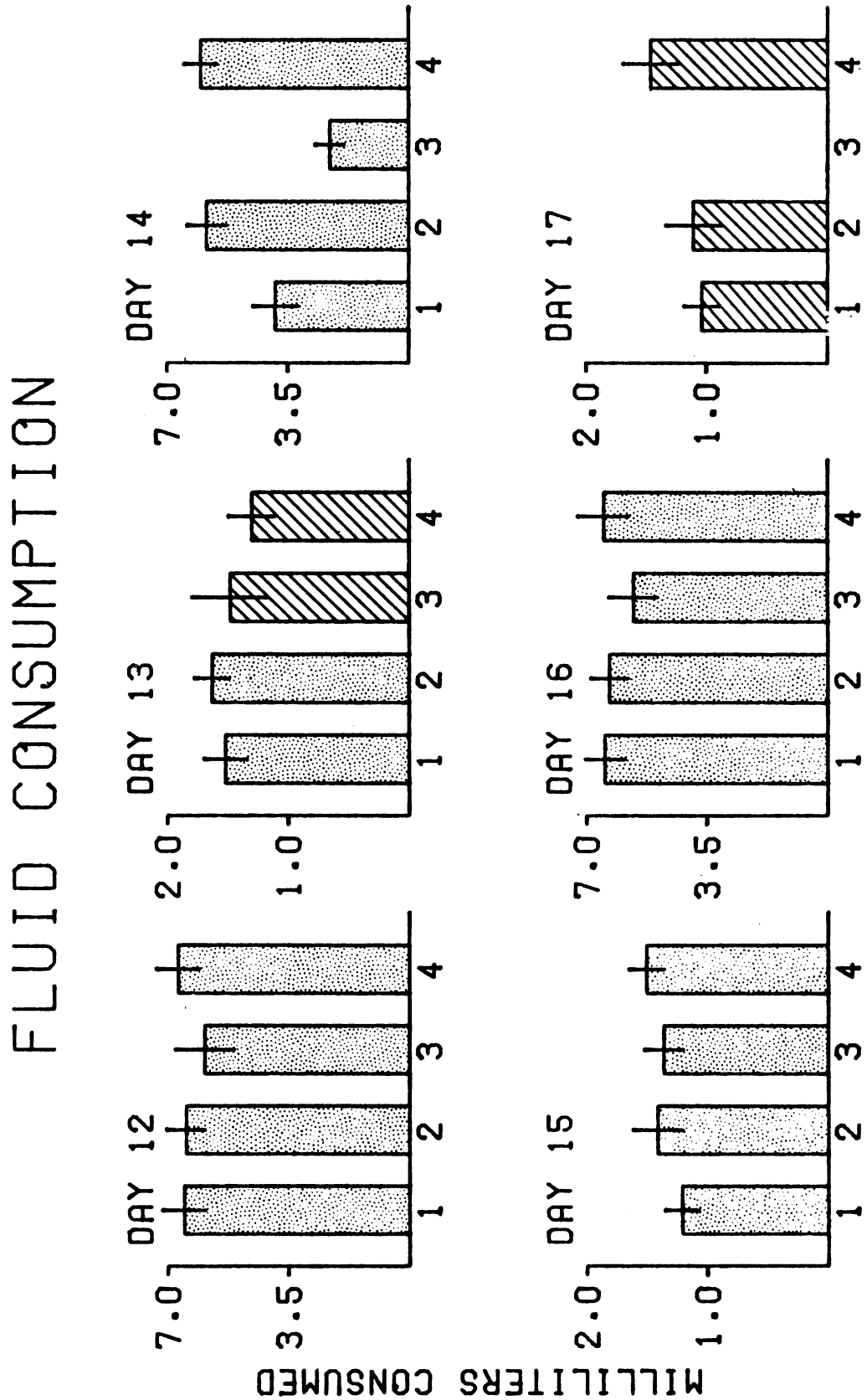


Fig. 11.--Mean fluid consumption (± 1 SE) over 6 days. The stippled bars represent water consumption, the striped bars sucrose consumption.

day 13 - This graph represents 20-minute water consumption for groups 1 and 2, and 20-minute sucrose consumption for groups 3 and 4. Again, analysis of variance indicates that no significant differences exist. NOTE: immediately following this 20-minute drinking period the animals were injected.

day 14 - This graph shows the 22-hour water consumption for the period beginning 2 hours after the injections. Both of the lithium-injected groups (group 1, water followed by lithium, and group 3, sucrose followed by lithium) show significantly depressed consumption (see Table 3 and Table 4). No difference exists between groups 1 and 3, nor between groups 2 and 4.

day 15 - This graph gives the 20-minute water consumption on the second day after the injections. Analysis of variance indicates that no significant differences exist.

day 16 - This graph gives the 24-hour water consumption recorded on the third day following the injections. Again, analysis of variance shows no significant differences.

day 17 - This graph gives the 20-minute sucrose consumption for all groups. Group 3 does not appear, as none of the animals in this group drank <u>any</u> sucrose. Thus, an obvious difference exists between group 3 and the other groups. No significant differences exist among groups 1, 2, and 4 (see Table 5 and Table 6).

Table 3.--Analysis of variance on water consumption on day 14 in basic taste aversion learning test II.

Factor	df	Mean Square	F	Prob.
Injectant	1	81.796	29.759	$\leq .001$
Preinjection flavor	1	5.041	1.834	NS
Injectant x Flavor	1	7.569	2.754	NS
Error	36	2.749		
Total	39	ss = 98.950		

Table 4.--Duncan's New Multiple Range Test on water consumption of day 14 in basic taste aversion learning test II. Those means not subtended by the same line are significantly different ($p \leq .05$).

group	suc/Li	wat/Li	wat/Na	suc/Na
mean	2.27	3.85	5.84	6.00

Table 5.--Analysis of variance on sucrose consumption of day 17 in basic taste aversion learning test II.

Factor	df	Mean Square	F	Prob.
Injectant	1	5.852	18.917	$\leq .001$
Preinjection Flavor	1	1.190	3.847	NS
Injectant x Flavor	1	4.830	15.614	$\leq .001$
Error	36	0.309		
Total	39	SS = 11.137		

Table 6.--Duncan's New Multiple Range Test on sucrose consumption on day 17 in basic taste aversion learning test II. Those means not subtended by the same line are significantly different ($p \leq .05$).

group	suc/Li	wat/Li	wat/Na	suc/Na
mean	0.00	1.04	1.11	1.46

Conclusions

On day 17 sucrose was refused by group 3 (which had lithium toxicosis contingently paired with sucrose ingestion), but not by group 1 (which received lithium following water ingestion). This indicates that the aversion to sucrose shown by group 3 was not the result of the lithium injection *per se*, but rather was the result of the contingency between sucrose ingestion and lithium poisoning. On days 15 and 16, no significant differences existed in water consumption among the groups. This indicates that the aversion produced by the sucrose/lithium contingency was specific to sucrose and not a generalized aversion to drinking. The fact that both of the lithium-injected groups showed a significant decrease in water consumption on day 14 is most reasonably explained by noting that the consumption on day 14 represents water consumption during a period when the animals were under the direct influence of the lithium toxicosis. Observations of animals under the influence of lithium (Radomski, Fuyat, Nelson, and Smith, 1950; Nachman, 1963; and my observations) indicate that these animals are particularly lethargic and do not engage in any activities--drinking included. However, whatever the source of this difference between the two lithium-injected groups and the two sodium-injected groups, it is important to note that it has completely disappeared by day 15--the second day following the injection. Therefore, in *Peromyscus* tests which intend to measure the learned effects of lithium toxicosis (without fear of being confounded by interactions with nonlearned effects) should never be conducted during the 24-hour

period immediately following the induction of the illness, but they may be held at any time thereafter.

In summary, the data indicate that: (1) adult P. m. bairdi are capable of forming a learned aversion to sucrose following a single pairing of sucrose consumption with delayed lithium-induced illness; (2) this aversion is not simply the effect of lithium injection per se; and (3) this aversion is not a general aversion to drinking. Furthermore, the fact that the sucrose/lithium group formed an aversion to sucrose, but the water/lithium group did not form an aversion to water indicates that the contingent flavor must in some way be "distinctive." However, this experiment provides no information regarding that distinctiveness; that is, there is no indication whether the distinctiveness is provided by the novelty, or by the sweetness, or by some other attribute of the sucrose.

EFFECT OF FLAVOR

Introduction

In the Basic Taste Aversion Learning II experiment it was noted that apparently a flavor must be "distinctive" if an animal is to associate it with toxicosis. Since all of the experiments in this series have employed a 20% sucrose solution exclusively as the flavor paired with toxicosis, it is theoretically possible, albeit intuitively implausible, that the "distinctiveness" resides in the specific flavor of the sucrose solution. There is nothing in the data presented so far that could contradict the assertion that Peromyscus are capable of forming taste aversions only to a sucrose solution. This experiment will test that assertion. It will also test whether an aversion to one novel flavor generalizes to another novel flavor.

Purposes

The purposes of this experiment are twofold: (1) to determine if Peromyscus can form aversions toward flavors other than that of a 20% sucrose solution--specifically, to determine if Peromyscus can form aversions to fluids that are distinctly sour, salty, or bitter; and (2) to determine if an aversion directed toward one of these basic flavors (sweet, sour, salty, bitter) is generalized toward the others.

Test Subjects

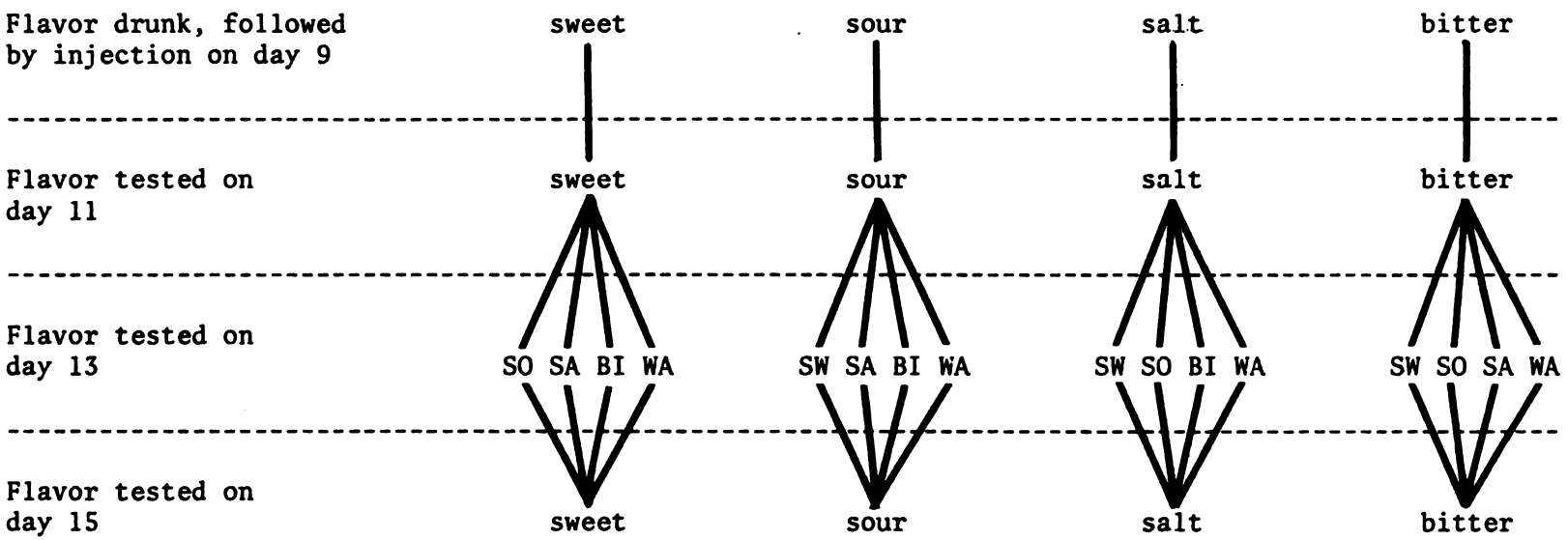
All of the animals in this study were experimentally naive adult (male and female) F_1 *P. m. bairdi* reared according to the description in the General Methods section.

Procedure

The animals were housed individually in plastic cages and randomly assigned to 1 of 8 treatment groups (n = 16 for each). The different treatment groups were each assigned to have 1 of the 4 basic flavors paired with either a NaCl or a LiCl injection. The specific assignments were (1) sucrose/Li, (2) sucrose/Na, (3) HCl/Li, (4) HCl/Na, (5) NaCl/Li, (6) NaCl/Na, (7) quinine/Li, and (8) quinine/Na. (Note: See the General Methods section, page 71, for a description of the actual concentrations used for each flavor.)

All of the animals were given water on the basic fluid schedule of Figure 5 and Table 1 for 8 days. On day 9 the different groups were offered their assigned flavors, followed by their assigned injections (IP, 6.0 mEq/kg as 0.4 molar solutions). On day 10 all groups received water. On day 11, each group was tested on the flavor which it had received paired with injection--this test was to determine if an aversion had been formed to each flavor. On day 12 all groups received water. On day 13, each group was divided into 4 subgroups and each subgroup was tested on a flavor other than the one paired with injection (see Table 7 for an illustration of this procedure); this test was to determine if the aversion directed toward the flavor paired with injection would generalize to the other novel flavors. On day 14 all groups received water. On day 15, all groups were

Table 7.--Fluid presentation schedule for the effect of flavor test.



Note: SW = sweet, SO = sour, SA = salt, BI = bitter, WA = water.

retested on the flavor that they had had originally paired with injection--this test was necessary to determine if the aversion to the primary flavor was still intact on the trial following the generalization trial.

Results

The results on the first, second, and third test trials are given in Figures 12, 13, and 14, respectively. Asterisks in these figures indicate the level of significance (following convention, * = $p \leq 0.05$, ** = $p \leq 0.01$, *** = $p \leq 0.001$) of a t-test comparing the means of the LiCl and NaCl groups illustrated. These results are discussed in the following section.

Conclusions

The first question to be answered is, did the animals form aversions to all four flavors? Figure 12 shows clearly that they did. However, the responses of the groups drinking the salty flavor (both the LiCl and the NaCl injected groups) were significantly different from those drinking the other flavors. This might indicate that the 0.1 molar NaCl solution employed as the salty taste cue was too weak for the animals to detect it clearly, were it not for the fact that extensive pilot studies had indicated the 0.1 molar concentration to be the optimal concentration for producing learned aversions. This leaves a number of alternate explanations for the unique response to the salty flavor: (1) Salt solution may be less thirst quenching than the other flavors. This could explain the increased consumption by the control group, but it does not explain why the poisoned group

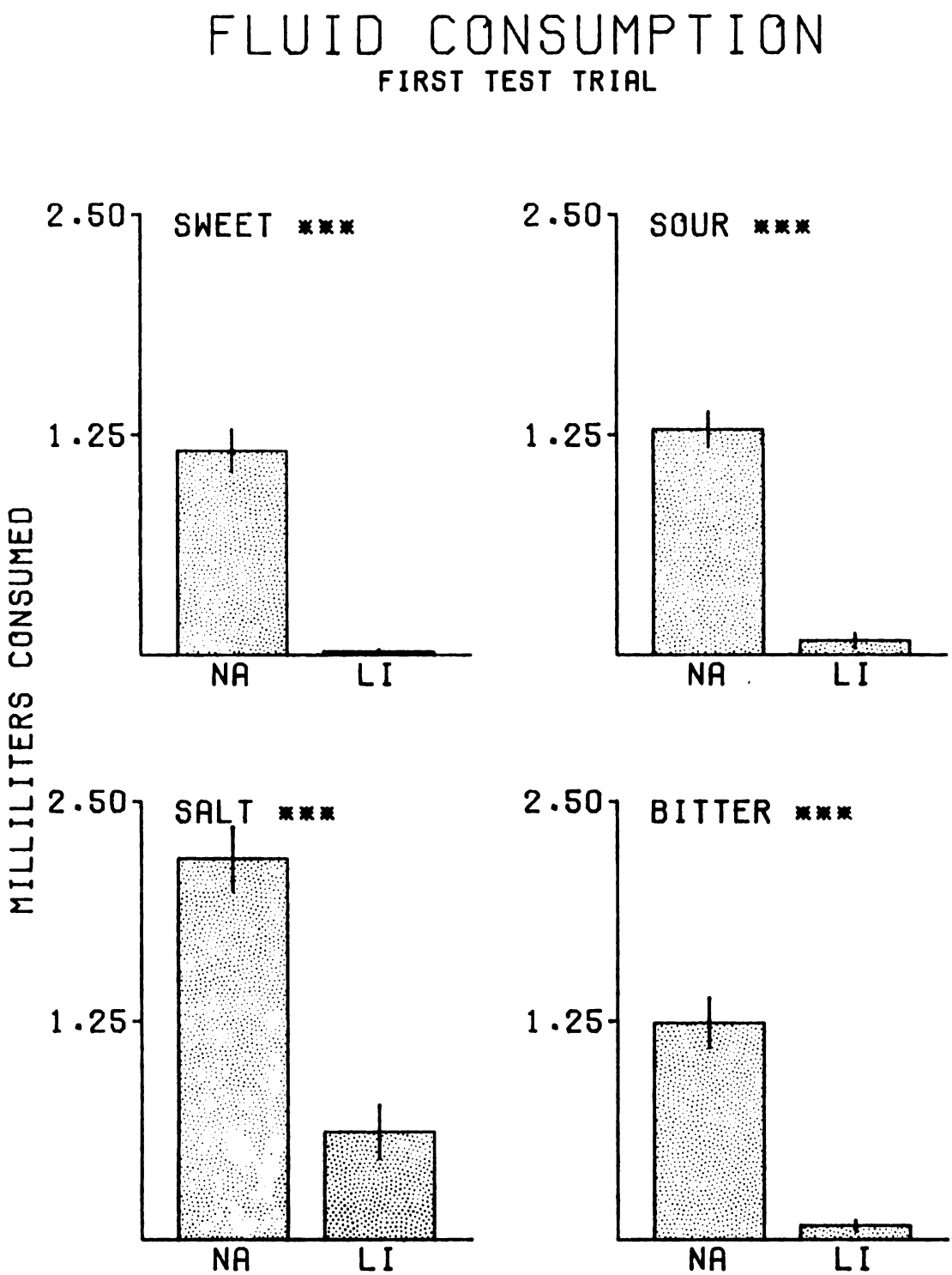


Fig. 12.--Mean fluid consumption (± 1 SE). The asterisks indicate the level of significance of a t-test comparing the LiCl group with the NaCl group.

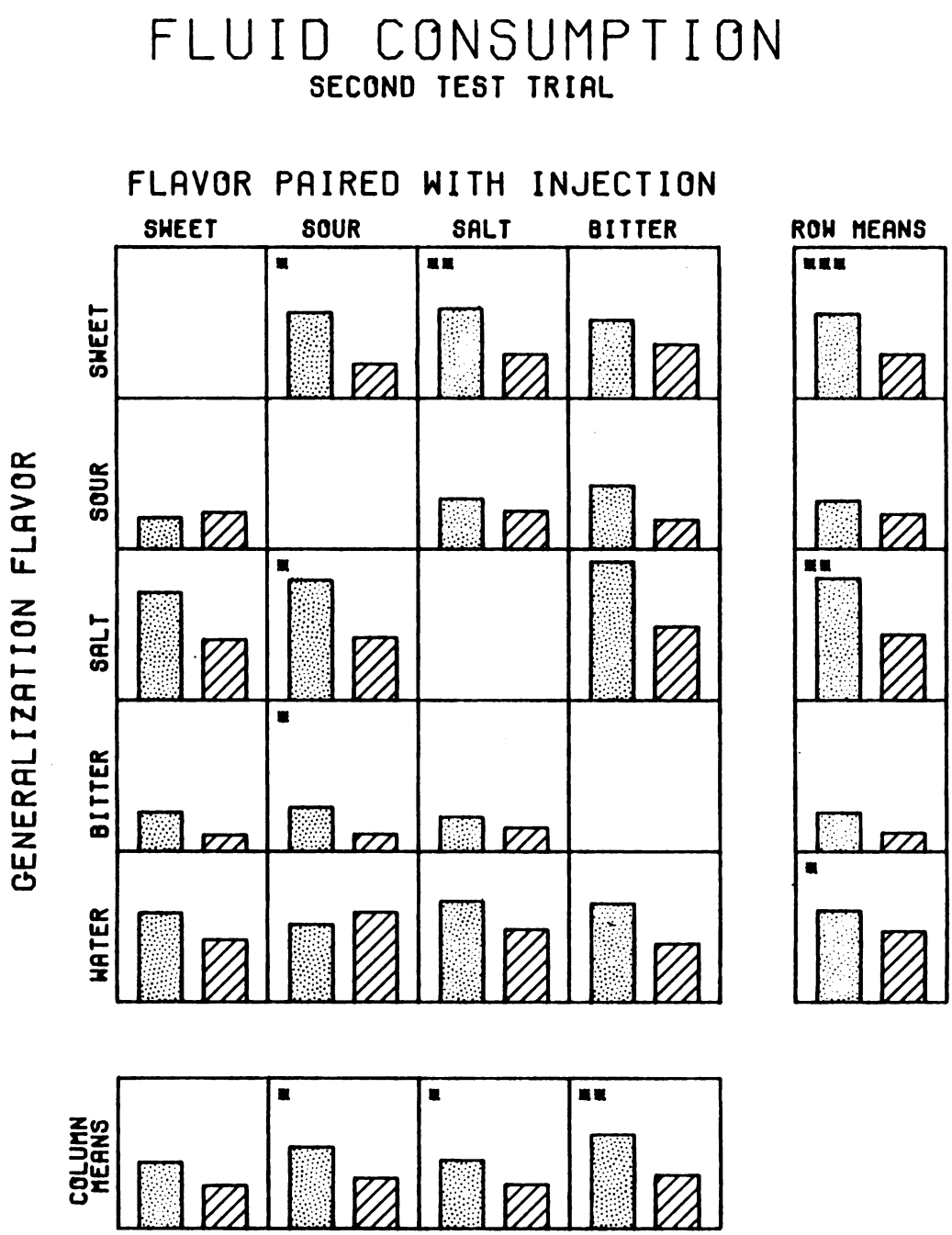


Fig. 13.--Mean fluid consumption of the generalization flavor. The asterisks indicate the level of significance of a t-test comparing the NaCl injected group (stippled bar) with the LiCl injected group (crosshatched bar).

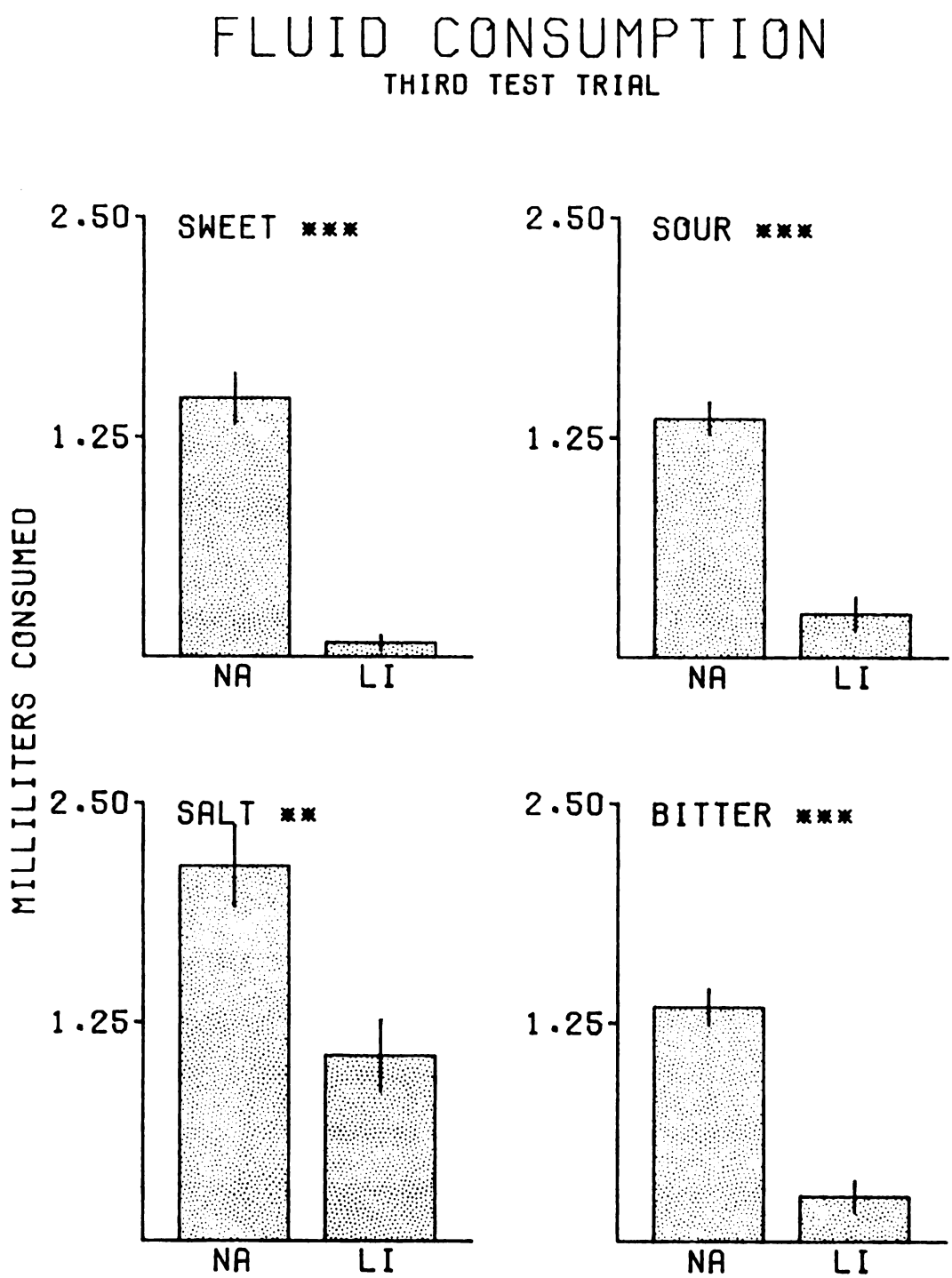


Fig. 14.--Mean fluid consumption (± 1 SE). The asterisks indicate the level of significance of a t-test comparing the LiCl group with the NaCl group.

failed to form as complete an aversion as the poisoned groups drinking the other flavors. (2) There may be a specific interaction between the flavor and the toxin. Radomski, Fuyat, Nelson, and Smith (1950) reported that a major component of lithium toxicity is severe sodium depletion, and Weisinger, Parker, and Skorupski (1974) found that rats would not form an aversion to salty water if formalin-induced sodium deficiency were used as the paired illness, even though the same treatment was effective in producing an aversion to other flavors and other toxins were effective in producing an aversion to the salty flavor. Although this could explain why the group poisoned on LiCl did not form a complete aversion, it cannot explain why the nonpoisoned group drank more salty water than the nonpoisoned groups drinking the other flavors. (3) The diet provided for the mice may have produced a slight, but chronic sodium deficiency. This could explain both the increased consumption of salty water by the controls (since it is known that sodium-deficient rats recognize and preferentially consume salty flavors [Hander, 1965]), and the failure of the poisoned animals to form complete aversions (since Frumkin, 1975, found it impossible to induce taste aversions toward salty flavors in rats with pre-existing sodium deficiencies). As this third possibility explains the unique response of both the poisoned and the nonpoisoned groups, it seems to be a more attractive hypothesis than the other two. However, since further experimentation is clearly necessary to determine the precise origin of the observed results, this difference between the groups drinking a salty flavor and the other groups must, for the present, stand as an interesting, but unexplained phenomenon.

The next question to be answered is, did the aversions formed generalize to other flavors? Since Figure 14 shows clearly that all of the flavor groups still showed strong aversions on the trial following the generalization trial, analysis of the generalization results may begin with the reasonable assumption that at the time of the generalization test each flavor group possessed a strong aversion to its primary flavor. Figure 13 gives the results of the generalization test (an inspection of Table 7 will help clarify Figure 13). Again, asterisks indicate the level of significance of a t-test comparing the two means.

The results in Figure 13 show that some generalization does occur. The LiCl-injected groups drank less than the NaCl-injected groups in 14 of the 16 independent paired comparisons. The probability of this occurring if there were really no difference between them is given by the binomial probability distribution as 0.0018. However, a comparison of the values shown in the boxes labelled "row means" with Figures 12 and 14 indicates that the generalized aversion was not as strong as the primary aversion.

At this point, it would be interesting to determine if there were any specific interactions between the various flavors of poisoning and flavors of generalization. Unfortunately, there are only 8 animals in each specific-interaction cell (4 LiCl and 4 NaCl), thus making the demonstration of significant differences most difficult. But, a faint pattern does appear discernible in the data: generalization is most pronounced away from sour and toward sweet. That is, Figure 13 shows that all of the significance that appears in

the specific interactions (the boxes not labelled as "means") occurs only in groups poisoned on sour or tested on sweet. However, the indistinctness of this pattern, coupled with the small number of animals involved, renders this conclusion tenuous at best and purely speculative at worst. Obviously, replication with increased sample size is called for.

Finally, attention must be paid to the significant difference shown for the row means for water consumption in Figure 13. At first glance this result suggests that the poisoned animals are learning to avoid drinking per se--a finding in sharp contrast with the results obtained in the Basic Taste Aversion Learning II experiment (page 91, above). However, when many different groupings of animals are made, as in this effect-of-flavor experiment, it is always possible that a chance juxtaposition will cause a spuriously significant result. That appears to be the case with this "significant" difference in water consumption appearing in the row means for water consumption of Figure 13. A comparison of the water consumption of these two groups on the day before treatment shows an equally large and significant difference. Most importantly, the mean water consumption of the lithium-injected water-drinking animals on the last 20-minute water day before treatment was 1.49 (± 0.08) ml. On the day showing the apparent generalized aversion to drinking (the day represented in Figure 13) the lithium-injected animals' mean water consumption was 1.46 (± 0.11) ml. Obviously these animals have not acquired a generalized aversion to drinking, as their post-treatment water consumption is virtually identical to their pre-treatment water consumption.

Therefore, the apparently significant difference in mean fluid consumption between the lithium- and sodium-injected animals drinking water on day 13 does appear to be merely the result of a chance juxtaposition. It should also be noted that an analysis of the pre-treatment water consumption for all other compared groups in Figure 13 revealed no other pre-treatment significant differences.

In summary, the results of this experiment indicate that: (1) Peromyscus can and do form strong taste aversions toward sweet, sour, salty, and bitter fluids if ingestion of these fluids is followed by LiCl-induced illness, and (2) there is some tendency for Peromyscus to generalize an aversion from one novel flavor to another.

EFFECT OF NOVELTY

Introduction

In the Conclusions to the Basic Taste Aversion Learning II experiment (page 91) it was noted that apparently flavors used successfully in the formation of learned aversions must be in some way "distinctive." The suggestion that the "distinctiveness" might reside in the sweetness per se of the flavor has been tested and rejected in the Effect of Flavor experiment. However, the suggestion that the novelty of the flavor might contribute to its "distinctiveness" has not yet been tested in Peromyscus.

The importance of novelty in the taste aversion phenomenon is also of great relevance in assessing the potential contribution of the phenomenon to overall foraging strategy. Studies on laboratory rats have indicated that absolute novelty is necessary for the formation and maintenance of maximum aversions after a single toxic experience. Vogel and Clody (1972), Elkins (1973), Fenwick, Mikulka, and Klein (1975), and Klein, Mikulka, and Hamel (1976) have demonstrated that as few as 3 safe exposures to a fluid will significantly reduce a rat's ability to acquire a learned aversion to that flavor, and that only 1 safe exposure will greatly facilitate the extinction of an aversion. Siegel (1974) showed that only 1 safe exposure was required to affect significantly the acquisition of an aversion.

These results, particularly the dramatic effect upon extinction produced by a single safe exposure, suggest that absolute novelty of the experimental flavor is important in both the acquisition and the maintenance of learned taste aversions. This in turn obviously affects the potential generality of the phenomenon: If a taste must be relatively novel for an aversion to be learned and to be maintained, the taste aversion phenomenon may contribute significantly to an animal's overall foraging strategy only under relatively limited conditions. However, it is possible that the distinct loss of "neophobia" observed in domestic rats as compared with wild rats (Barnett, 1958; Barnett, 1963) might account for this apparent requirement of absolute novelty. If this were true, the results cited above (Elkins, 1973; Fenwick et al., 1975; Klein et al., 1976; Siegel, 1974) might not even be applicable to feral rats, much less generalizable to Peromyscus.

Purpose

The purpose of this experiment is to test the effect of relative flavor novelty upon the acquisition and maintenance of taste aversions in Peromyscus.

Test Subjects

All of the animals in this study were experimentally naive adult (male and female) F_1 P. m. bairdi reared according to the description in the General Methods section.

Procedure

The animals were housed individually in plastic cages and randomly assigned to 1 of 5 treatment groups (n = 13 for each).

Treatment group 1 was assigned to receive no prior safe exposure to sucrose, then a sucrose/LiCl pairing. Treatment groups 2 and 3 were assigned to receive a single safe exposure to sucrose, then a sucrose/LiCl pairing. Treatment group 4 was assigned to receive 10 safe exposures to sucrose, then a sucrose/LiCl pairing. Treatment group 5 was assigned to receive no safe sucrose exposure, then a sucrose/NaCl pairing. See Figure 15 for an illustration of this design.

All of the animals were given water on the basic fluid schedule of Figure 5 and Table 1 for 8 days. On day 9 the individual treatment schedules were begun. Figure 15 shows these individual schedules. On day 31 all animals were offered sucrose, followed by an IP injection of 9.0 mEq/kg of LiCl or NaCl (as a 0.6 molar solution). Twelve extinction trials were administered by presenting sucrose solution to all animals on the odd numbered days from day 33 through day 55.

Results

The results showing the effect of relative novelty upon aversion acquisition are given in Figure 16. T-tests indicated that groups 1, 2, and 3 were not different from each other ($|t| \leq 0.659$, $df = 24$), but were all different from group 4 ($|t| \geq 3.780$, $df = 24$, $p \leq 0.001$) and from group 5 ($|t| \geq 9.300$, $df = 24$, $p \leq 0.001$). Furthermore, group 4 was different from group 5 ($t = 4.090$, $df = 24$, $p \leq 0.001$).

Figure 17 gives the results showing the effect of relative novelty upon aversion maintenance. A group was considered to have extinguished its aversion when a t-test comparing its mean with that of group 5 (the NaCl-injected group) showed no significant difference.

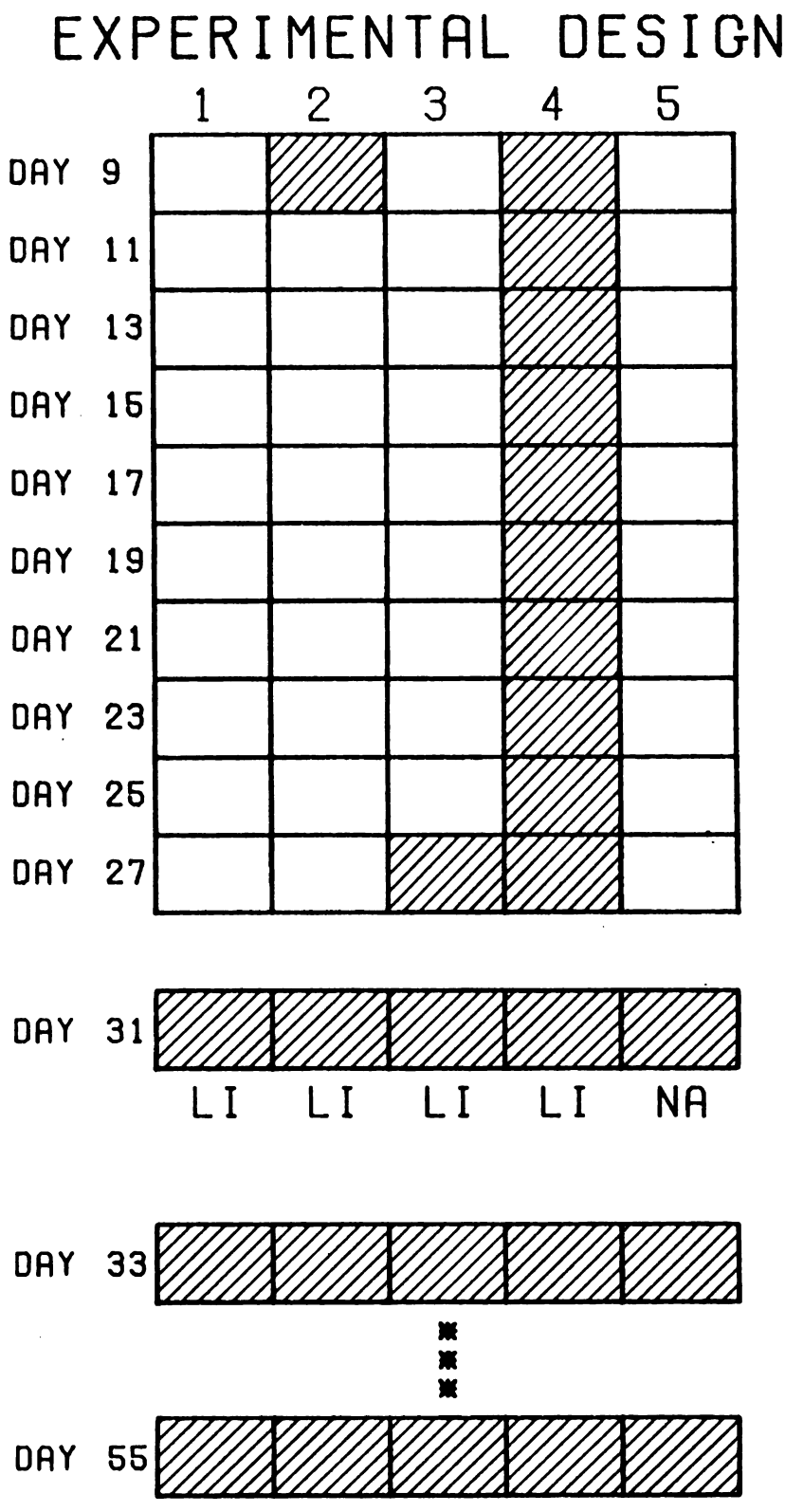


Fig. 15.--Schedule of fluid presentations and injections in the Effect of Novelty experiment. Sucrose was offered on all of the crosshatched days illustrated and also on the odd numbered days between day 33 and day 55. Water was offered on all other days.

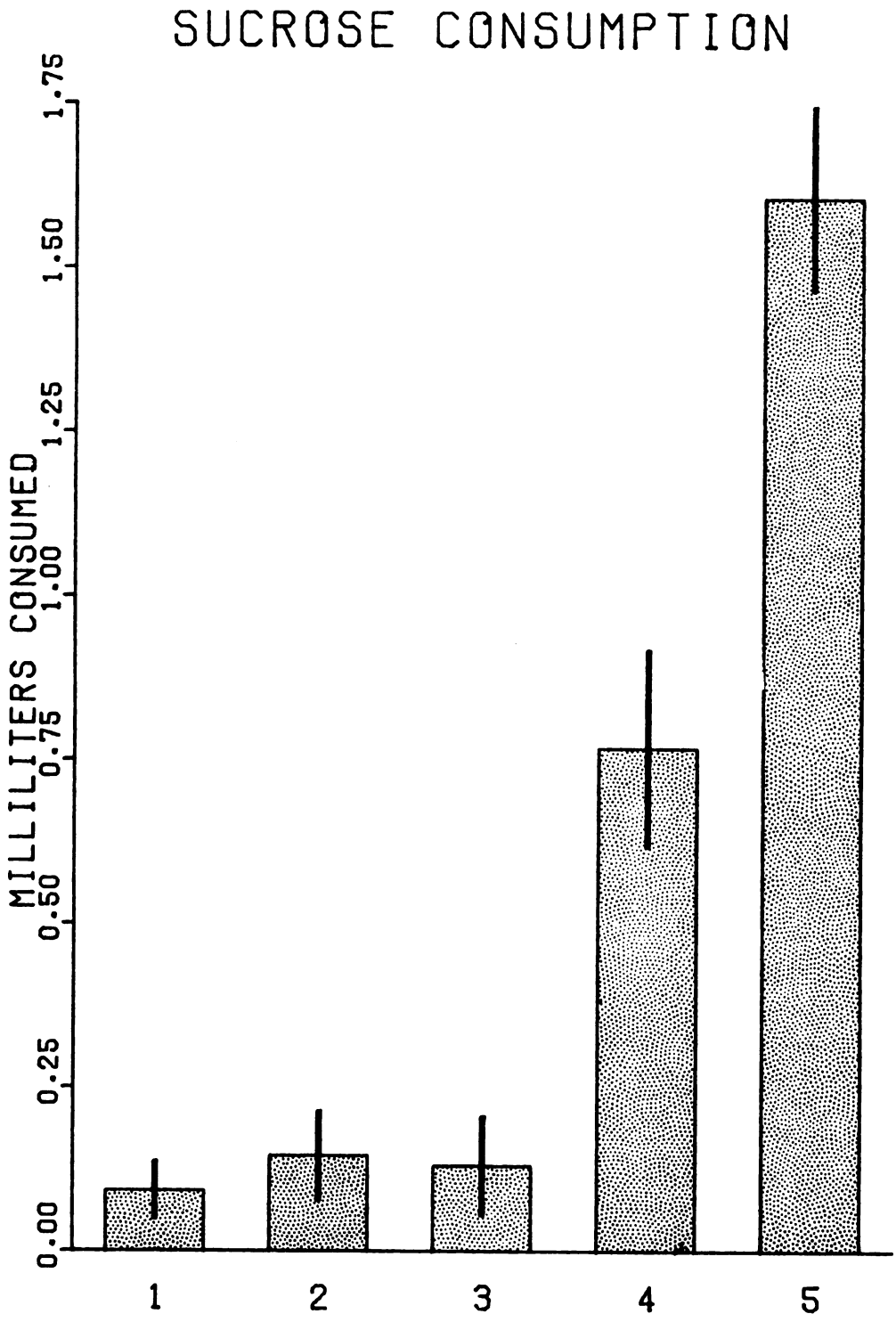


Fig. 16.--Mean sucrose consumption (± 1 SE) on the first trial following the sucrose/injection contingency. The numbers below the abscissa indicate the treatment groups as defined in Figure 15.

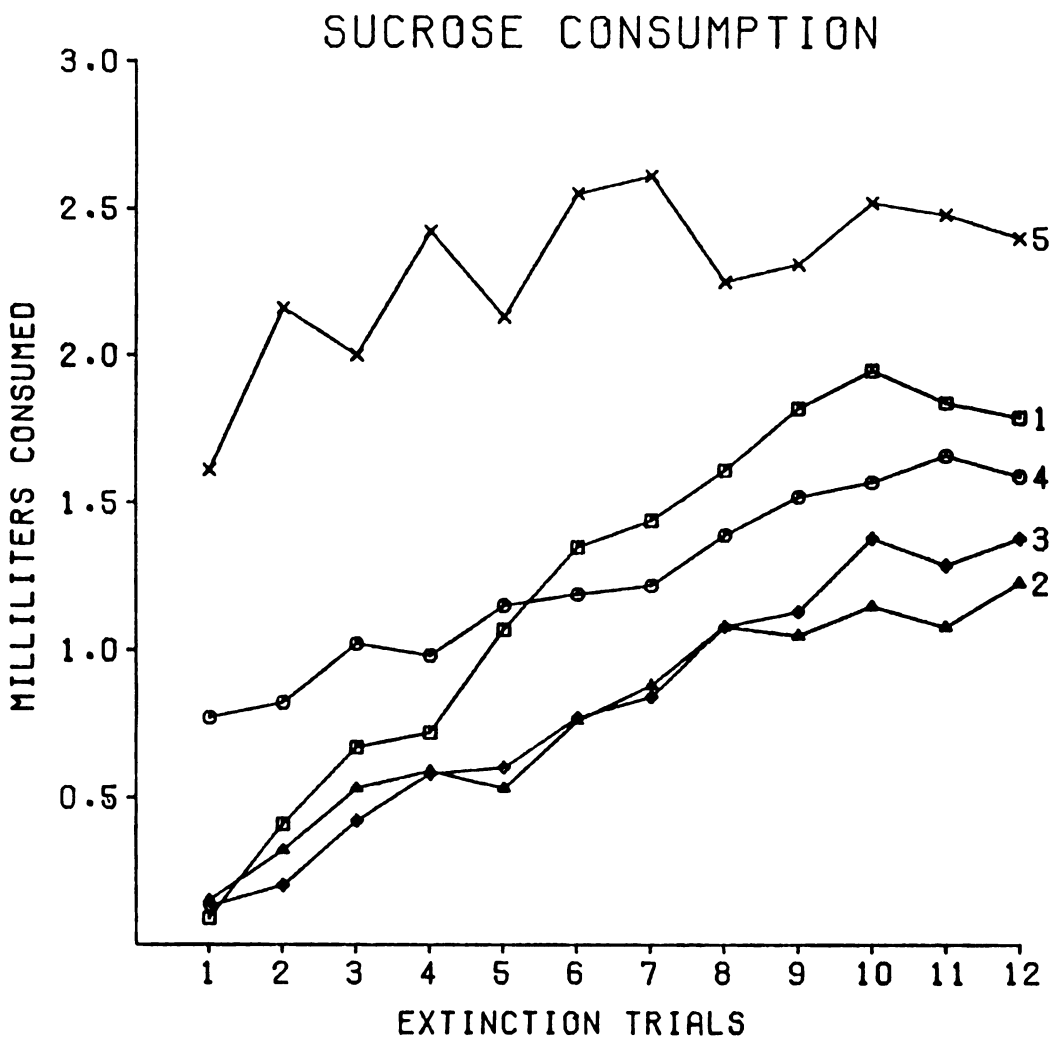


Fig. 17.--Mean sucrose consumption over 12 extinction trials. The numbers to the right of the curves indicate the treatment groups as defined in Figure 15.

Only group 1 (the no-prior-safe-experience group) ever attained this criterion, doing so on extinction trial 9.

Conclusions

The significant difference on the first extinction trial between groups 1 (no-safe-prior) and 4 (10-days-safe-prior) show that familiarization with a flavor reduces the ability of Peromyscus to form a taste aversion toward that flavor. However, the equally significant difference between group 4 (10-days-safe-prior) and group 5 (sodium control) shows that 10 days of safe exposure in no way eliminates the ability to form aversions. Similarly, the lack of differences among the no-prior and the 1-day-prior groups shows that a slight decrease in novelty has no effect upon aversion acquisition. A comparison of these results with those of Elkins (the rat study most comparable in design to this one) shows that the effect of relative novelty upon aversion acquisition is roughly the same in Peromyscus as in laboratory rats.

However, in both of the rat studies involving extinction data (Elkins, 1973; Fenwick et al., 1975) it was found that even 1 day of safe exposure caused a marked increase in the rate of aversion extinction as compared with animals which had had no prior safe experience. In striking contrast, Figure 17 shows that for Peromyscus the no-prior-safe-experience group showed the most rapid extinction. In fact, only the no-prior-safe-experience group ever attained the extinction criterion. On extinction trial 12, groups 2 and 3 (1-day prior-safe-exposure) were still different from group 5 at the

$p \lesssim 0.001$ level, while group 4 (10-days-safe-exposure) was still different at $p \lesssim 0.01$.

It is always tempting, whenever striking and unexpected results are encountered in an unreplicated experiment, to suggest that they may have arisen from a powerful chance deviation. For example, there is a pronounced change in the slope of the group 1 curve between extinction trials 4 and 5. Might not this have come about as the result of a single outlier animal? An inspection of the raw data indicates, however, that this is not the case. On trial 5, 6 animals in group 1 drank 1.5 ml or more, while in groups 2 and 3 only 2 animals drank 1.5 ml or more.

Although the absence of an outlier animal in group 1 argues against the chance explanation of these results, a replication certainly seems advisable. Until such a replication is performed, a conservative interpretation of these results is warranted. Therefore, despite the indication from these data that in Peromyscus the relationship between novelty and extinction is exactly the converse of that found in rats, I choose only to assert that this experiment shows that the relationship in Peromyscus is not the same as in rats. That is, in Peromyscus the relative familiarity of the flavor paired with toxicosis may affect the degree of aversion formed, but an aversion toward a slightly familiar flavor appears to extinguish no more rapidly than one toward a wholly novel flavor.

EFFECT OF CHOICE

Introduction

In the Effect of Novelty experiment (pp. 112-119) it was found that extinction of the aversions began almost immediately. An inspection of Figure 17 shows that there was an appreciable decrease in the aversion with every extinction trial. This very rapid onset of extinction is rather inconsistent with the hypothesis that taste aversion learning might play an important role in these animals' feeding strategies--that is, taste aversion learning seems capable of powerfully affecting only those first few feedings immediately following the toxic experience. On the other hand, the rate of extinction suggested by the data given in Figure 17 might be somewhat misleading. These data represent sucrose consumption when sucrose was the only fluid available after a 24-hour fluid deprivation period. Since these rather stringent conditions could easily be expected to force a rapid extinction of the aversion, the extinction curve of Figure 17 might be giving a distorted view of the duration of acquired taste aversions. Therefore, the present experiment has been designed to assess the effect of choice upon the duration of acquired taste aversions.

Purpose

The purpose of this experiment is to determine the duration of an acquired taste aversion under conditions in which the animals

are allowed to choose between water and the flavor associated with toxicosis.

Test Subjects

All of the animals in this study were experimentally naive adult (male and female) F_1 *P. m. bairdi*, reared according to the description in the General Methods section.

Procedure

The animals were housed individually in plastic cages and randomly assigned to 1 of 2 treatment groups (n = 20 for each group). Treatment group 1 was assigned to receive a sodium chloride injection following the ingestion of sucrose (20% w/v solution); treatment group 2 was assigned to receive a lithium chloride injection following the ingestion of sucrose. As the experiment progressed, four animals in group 1 proved to be pregnant and were dropped from the experiment, leaving n = 16 in group 1. One animal in group 2 died following the injection and 2 proved to be pregnant, leaving n = 17 in group 2.

In order to develop a regular drinking schedule in the animals, both of the groups were given water according to a schedule slightly modified from the basic fluid schedule of Table 1. The modifications were: (1) during any drinking bout two tubes of fluid were offered, rather than one, and (2) the 20-minute drinking periods were extended to 30 minutes. After 8 days on this schedule, each animal's drinking data for the 4 previous 30-minute drinking periods were examined and a preferred position determined, based on the total fluid consumption at the two locations. On day 9 each animal was

offered a single tube, in its preferred position, containing a 20% w/v sucrose solution. Immediately following the 30-minute drinking period with sucrose the group 1 animals were injected (IP) with 9.0 mEq/kg of body weight of NaCl as 0.6 M solution. The group 2 animals were similarly injected with a 0.6 M LiCl solution. Water was withheld for an additional 2 hours, then two water tubes were placed on the cage for 22 hours. Then the animals were returned to the modified fluid schedule outlined above for 64 days. During the 24-hour ad lib. drinking periods water was available in both tubes. During the 30-minute drinking periods sucrose was available in the preferred position tube, water in the other tube.

This method of controlling for position preference by always placing the sucrose solution in the preferred position was decided upon following a pilot study which indicated that nonpoisoned animals (i.e., NaCl-injected) tended to show a strong position preference which introduced excessive, spurious variance if the positions of the flavors were shifted about according to a random schedule. However, the schedule used in this experiment gives results which confound the effect of a flavor aversion with the effect of a position aversion. Therefore, to provide tests which could unconfound these variables, fluid presentation was extended for two additional cycles following those depicted in Figure 18. On the first of these additional 30-minute drinking periods, sucrose was presented in the nonpreferred position, water in the preferred. On the second additional 30-minute drinking period, water was presented in both positions.

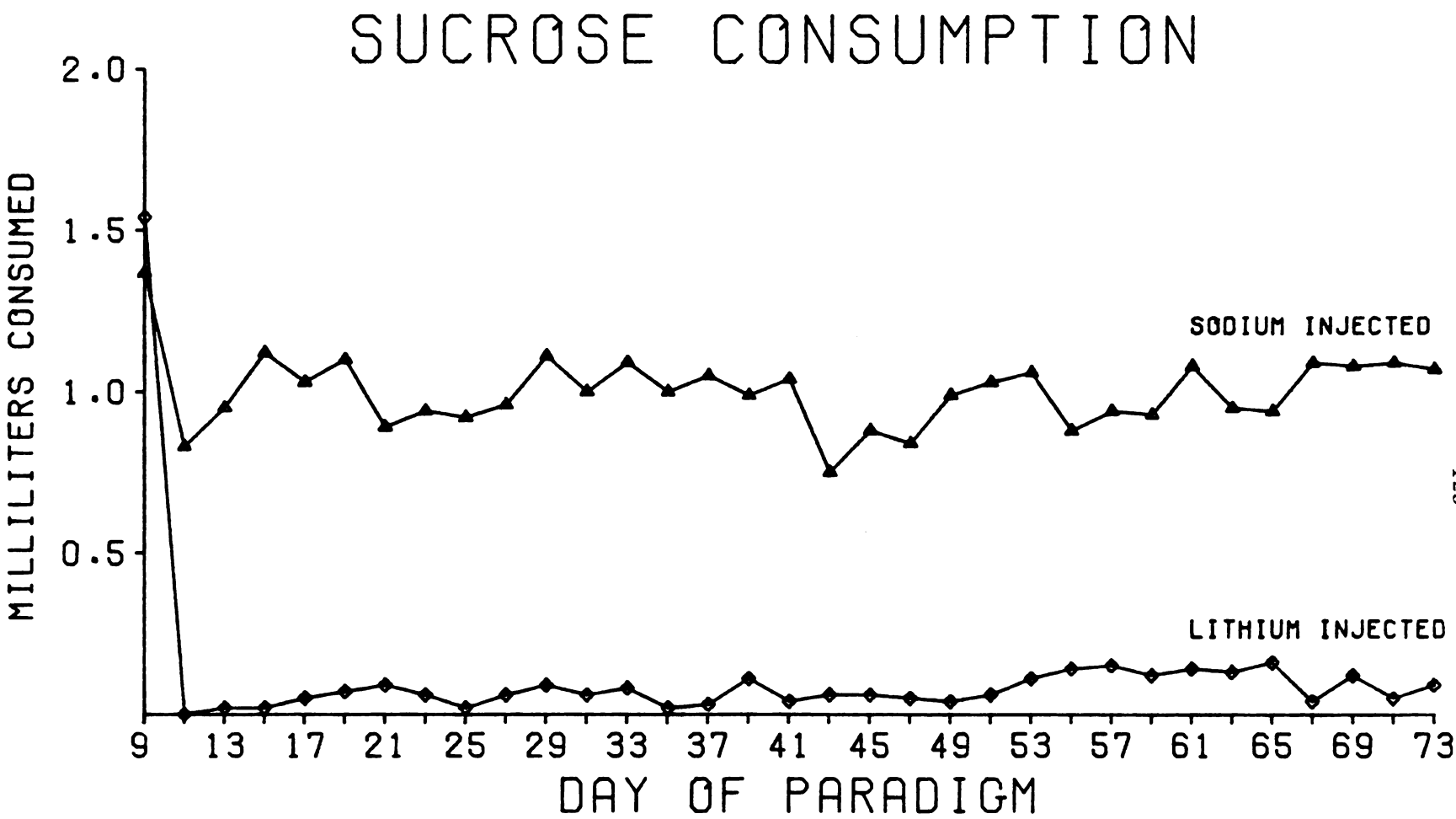


Fig. 18.--Mean sucrose consumption in the preferred position.

Results

Table 8 gives the fluid consumption in the preferred position beginning on day 9 (the day of the injection) and ending on day 73 (the last day when sucrose was presented in the preferred position). The data on the even-numbered days of the schedule indicate water consumption during a 24-hour period, while the data on the odd-numbered days indicate sucrose consumption during a 30-minute period. No significant differences exist between the treatment groups on any of the 24-hour water drinking periods (minimal acceptable significance being $p \leq .01$, due to the large number of comparisons). A highly significant difference ($p \leq .001$) in sucrose consumption between the lithium-injected and the sodium-injected groups was observed in every 30-minute drinking period following the sucrose/injection pairing. As can be seen from Figure 18, which graphically depicts the sucrose drinking data, the aversion shown by the lithium-injected animals to the sucrose continued virtually undiminished throughout the 64 days of testing. Table 9 gives the total fluid consumption (water plus water on the even-numbered days, water plus sucrose on the odd-numbered days) for both groups. No significant differences (again with $p \leq .01$ as the minimal acceptable significance) exist between the treatment groups on any of the days except day 10, which was the 22-hour period immediately following the injections. Figure 19 gives the results for the two additional cycles following the termination of the basic experiment. Figure 19-A gives the data from day 73, the last day of the basic paradigm, for reference. Figure 19-B gives the data for day 75, when sucrose was offered in the nonpreferred position.

Table 8.--Mean fluid consumption in the preferred position--data from even-numbered days represent water consumption in a 24-hour period, data from odd-numbered days represent sucrose consumption in a 30-minute period. The data are: mean (standard error). The t-test values represent comparisons between the mean of group 1 and the mean of group 2. On the odd-numbered days the significance of the t-test was determined according to Cochran's t'-test of difference between two means with heterogeneous variance (Snedecor and Cochran, 1967). The t-tests of the even-numbered days were standard tests.

Day	Group 1	Group 2	t-value	df
9	1.37 (0.14)	1.54 (0.16)	-0.8215	31
10	3.57 (0.48)	1.93 (0.50)	2.3606	31
11	0.83 (0.14)	0.00 (0.00)	6.0184***	15/16
12	4.13 (0.54)	4.25 (0.62)	-0.1478	31
13	0.95 (0.13)	0.02 (0.01)	7.1500***	15/16
14	3.89 (0.65)	3.18 (0.52)	0.8614	31
15	1.12 (0.14)	0.02 (0.01)	8.2523***	15/16
16	4.03 (0.79)	4.15 (0.56)	-0.1264	31
17	1.03 (0.12)	0.05 (0.04)	7.8961***	15/16
18	3.93 (0.63)	3.46 (0.52)	0.5747	31
19	1.10 (0.13)	0.07 (0.06)	7.4832***	15/16
20	3.95 (0.59)	2.77 (0.45)	1.6038	31
21	0.89 (0.15)	0.09 (0.06)	4.9064***	15/16
22	3.94 (0.76)	3.58 (0.46)	0.4167	31
23	0.94 (0.10)	0.06 (0.05)	8.0333***	15/16
24	4.38 (0.72)	3.65 (0.63)	0.7630	31
25	0.92 (0.16)	0.02 (0.02)	5.8059***	15/16
26	4.56 (0.74)	3.31 (0.48)	1.4344	31

Table 8.--Continued.

Day	Group 1	Group 2	t-value	df
27	0.96 (0.13)	0.06 (0.05)	6.7529***	15/16
28	4.29 (0.68)	2.95 (0.59)	1.4914	31
29	1.11 (0.14)	0.09 (0.08)	6.4243***	15/16
30	4.65 (0.64)	2.82 (0.60)	2.0830	31
31	1.00 (0.12)	0.06 (0.05)	7.2711***	15/16
32	4.54 (0.65)	3.17 (0.51)	1.6703	31
33	1.09 (0.16)	0.08 (0.06)	5.8504***	15/16
34	4.19 (0.71)	3.48 (0.56)	0.7907	31
35	1.00 (0.12)	0.02 (0.02)	8.6046***	15/16
36	3.46 (0.55)	2.90 (0.52)	0.7409	31
37	1.05 (0.16)	0.03 (0.02)	6.4611***	15/16
38	3.97 (0.66)	2.61 (0.48)	1.6827	31
39	0.99 (0.12)	0.11 (0.07)	6.2486***	15/16
40	3.41 (0.61)	2.38 (0.33)	1.5184	31
41	1.04 (0.15)	0.04 (0.03)	6.6656***	15/16
42	3.39 (0.54)	2.88 (0.41)	0.7592	31
43	0.75 (0.13)	0.06 (0.04)	5.1646***	15/16
44	4.15 (0.60)	2.71 (0.56)	1.7571	31
45	0.88 (0.14)	0.06 (0.04)	5.9234***	15/16
46	4.51 (0.63)	3.06 (0.46)	1.8737	31
47	0.84 (0.13)	0.05 (0.04)	5.9579***	15/16
48	4.34 (0.67)	2.29 (0.42)	2.6219	31
49	0.99 (0.16)	0.04 (0.03)	5.8359***	15/16

Table 8.--Continued.

Day	Group 1	Group 2	t-value	df
50	3.84 (0.64)	2.54 (0.43)	1.7048	31
51	1.03 (0.15)	0.06 (0.03)	6.6639***	15/16
52	3.56 (0.65)	2.84 (0.46)	0.9175	31
53	1.06 (0.15)	0.11 (0.07)	5.7792***	15/16
54	3.89 (0.66)	3.04 (0.51)	1.0178	31
55	0.88 (0.13)	0.14 (0.09)	4.6686***	15/16
56	3.89 (0.60)	2.77 (0.51)	1.4387	31
57	0.94 (0.15)	0.15 (0.10)	4.5652***	15/16
58	3.94 (0.69)	2.87 (0.48)	1.2902	31
59	0.93 (0.14)	0.12 (0.07)	5.2067***	15/16
60	3.72 (0.66)	2.97 (0.50)	0.9100	31
61	1.08 (0.15)	0.14 (0.08)	5.7112***	15/16
62	4.21 (0.67)	2.51 (0.48)	2.0872	31
63	0.94 (0.16)	0.13 (0.07)	4.7848***	15/16
64	3.76 (0.58)	2.74 (0.55)	1.2773	31
65	0.95 (0.17)	0.16 (0.09)	4.1962***	15/16
66	4.10 (0.70)	2.47 (0.46)	1.9727	31
67	1.09 (0.16)	0.04 (0.03)	6.8570***	15/16
68	3.74 (0.63)	2.66 (0.47)	1.3899	31
69	1.08 (0.17)	0.12 (0.08)	5.1563***	15/16
70	3.41 (0.64)	2.74 (0.42)	0.8892	31
71	1.09 (0.20)	0.05 (0.04)	5.3832***	15/16
72	3.79 (0.60)	3.15 (0.48)	0.8474	31

Table 8.--Continued.

Day	Group 1	Group 2	t-value	df
73	1.07 (0.17)	0.09 (0.06)	5.3999***	15/16

Note: ** denotes $p \leq 0.01$; *** denotes $p \leq 0.001$.

Table 9.--Mean fluid consumption totaled from the preferred and unpreferred positions--data from even-numbered days represent water consumption in a 24-hour period, data from odd-numbered days represent water plus sucrose consumption in a 30-minute period. The data are: mean (standard error). The t-test values represent comparisons between the mean of group 1 and the mean of group 2. All tests were standard t-tests.

Day	Group 1	Group 2	t-value	df
9	1.37 (0.14)	1.54 (0.16)	-0.8215	31
10	6.55 (0.88)	3.19 (0.48)	3.3970**	31
11	1.80 (0.16)	1.51 (0.16)	1.2698	31
12	7.22 (0.88)	7.18 (0.68)	0.0329	31
13	2.08 (0.18)	1.78 (0.16)	1.2416	31
14	6.68 (0.78)	6.71 (0.69)	-0.0293	31
15	2.12 (0.19)	1.66 (0.15)	1.9107	31
16	7.59 (1.12)	7.22 (0.68)	0.2862	31
17	2.18 (0.18)	1.62 (0.16)	2.2836	31
18	7.09 (0.77)	7.29 (0.65)	-0.2056	31
19	2.13 (0.14)	1.89 (0.18)	1.0180	31
20	7.29 (0.89)	6.92 (0.68)	0.3333	31
21	2.06 (0.15)	1.66 (0.13)	1.9910	31
22	7.03 (0.95)	7.04 (0.65)	-0.0141	31
23	2.19 (0.13)	1.78 (0.14)	2.1535	31
24	7.00 (0.79)	7.14 (0.64)	-0.1334	31
25	2.14 (0.13)	1.73 (0.12)	2.3073	31
26	7.62 (0.95)	7.26 (0.30)	0.2982	31
27	2.20 (0.14)	1.71 (0.16)	2.2683	31
28	7.51 (0.95)	7.19 (0.69)	0.2745	31

Table 9.--Continued.

Day	Group 1	Group 2	t-value	df
29	2.24 (0.15)	1.80 (0.17)	1.9750	31
30	7.01 (0.89)	7.25 (0.65)	-0.2145	31
31	2.25 (0.20)	1.76 (0.16)	1.9040	31
32	7.19 (0.83)	7.20 (0.62)	-0.0121	31
33	2.12 (0.18)	1.76 (0.17)	1.4236	31
34	7.68 (1.07)	7.88 (0.88)	-0.1500	31
35	2.14 (0.23)	1.36 (0.24)	2.3125	31
36	6.56 (0.81)	6.21 (0.66)	0.3295	31
37	2.36 (0.21)	1.76 (0.13)	2.4575	31
38	7.03 (0.93)	6.29 (0.63)	0.6632	31
39	2.10 (0.15)	1.82 (0.17)	1.2254	31
40	6.96 (0.87)	6.45 (0.61)	0.4893	31
41	2.14 (0.19)	1.79 (0.14)	1.4869	31
42	6.55 (0.84)	6.45 (0.55)	0.1042	31
43	2.01 (0.19)	1.75 (0.15)	1.0578	21
44	7.51 (0.94)	6.42 (0.62)	0.9729	31
45	2.24 (0.22)	1.86 (0.16)	1.4282	31
46	7.09 (0.94)	6.65 (0.58)	0.4093	31
47	2.16 (0.22)	2.00 (0.16)	0.6066	31
48	6.92 (0.83)	6.12 (0.51)	0.8318	31
49	2.21 (0.22)	1.86 (0.19)	1.2239	31
50	7.15 (0.79)	6.86 (0.77)	0.2584	31
51	2.29 (0.23)	1.79 (0.14)	1.8273	31

Table 9.--Continued.

Day	Group 1	Group 2	t-value	df
52	7.10 (0.84)	6.87 (0.57)	0.2290	31
53	2.41 (0.20)	2.08 (0.20)	1.1993	31
54	7.33 (0.81)	6.68 (0.54)	0.6756	31
55	2.30 (0.18)	2.08 (0.19)	0.8429	31
56	7.63 (0.86)	6.61 (0.54)	1.0168	31
57	2.19 (0.19)	1.99 (0.17)	0.7749	31
58	7.58 (0.86)	7.09 (0.64)	0.4587	31
59	2.26 (0.20)	1.99 (0.22)	0.9035	31
60	7.09 (0.92)	6.74 (0.63)	0.3213	31
61	2.38 (0.19)	2.15 (0.22)	0.7682	31
62	7.34 (0.92)	6.58 (0.59)	0.7136	31
63	2.48 (0.22)	2.25 (0.22)	0.7462	31
64	7.56 (0.97)	7.14 (0.62)	0.3661	31
65	2.22 (0.24)	2.04 (0.17)	0.6381	31
66	7.70 (0.94)	6.86 (0.57)	0.7754	31
67	2.66 (0.22)	2.17 (0.21)	1.5744	31
68	6.83 (0.81)	6.61 (0.69)	0.2129	31
69	2.57 (0.24)	2.11 (0.20)	1.4635	31
70	7.04 (1.03)	6.84 (0.69)	0.1649	31
71	2.49 (0.22)	2.07 (0.18)	1.4952	31
72	7.08 (0.97)	6.69 (0.67)	0.3314	31
73	2.43 (0.24)	2.12 (0.18)	1.0453	31

Note: ** denotes $p \leq 0.01$; *** denotes $p \leq 0.001$.

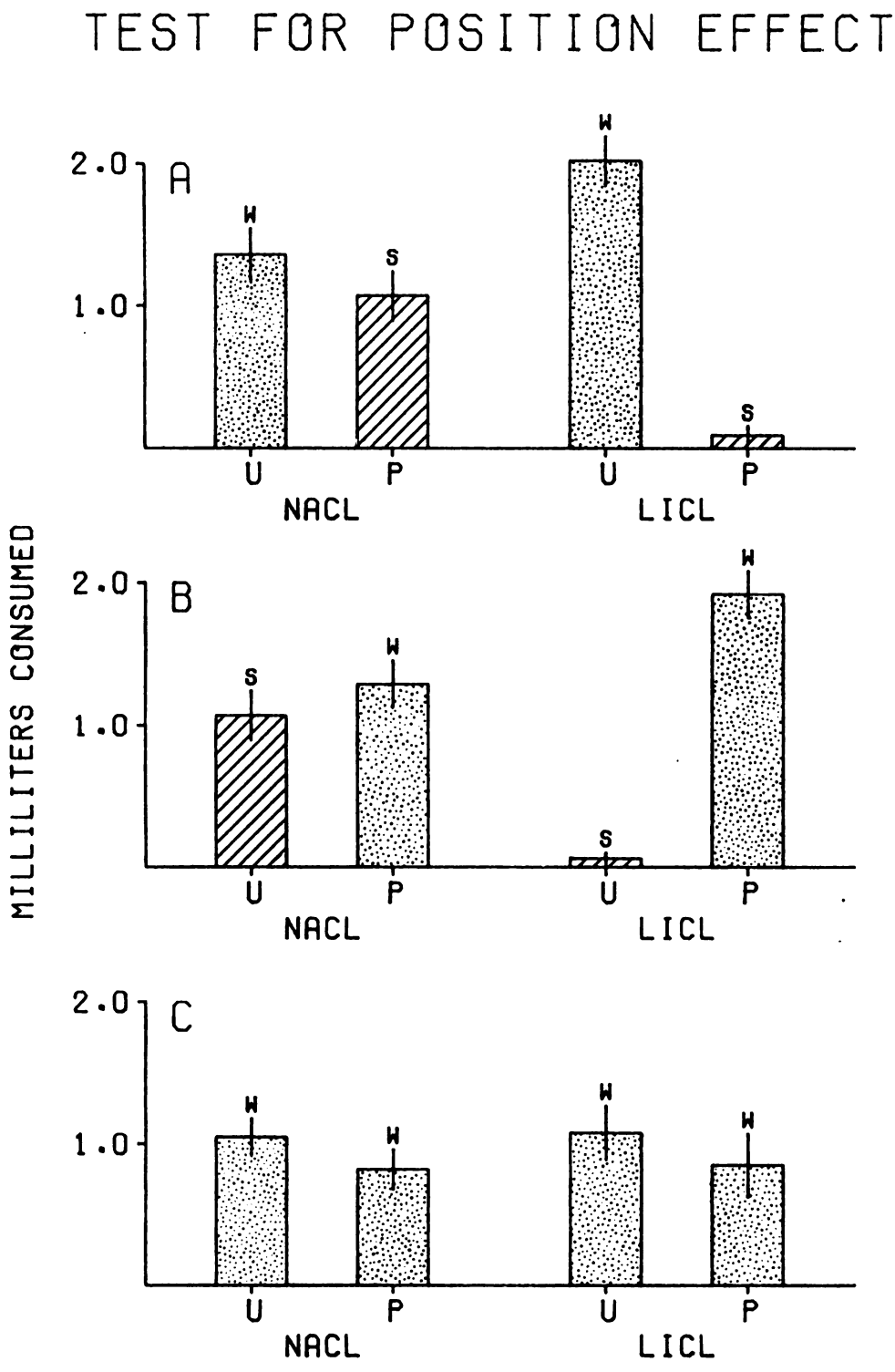


Fig. 19.--Mean fluid consumption (± 1 SE) in the preferred (P) and unpreferred (U) positions. Crosshatched bars represent sucrose consumption, stippled bars water consumption.

Figure 19-C gives the data for day 77, when water was presented in both positions.

Conclusions

The effect of choice seems clear--the strength of the aversion was unaffected by 32 unreinforced presentations of the conditioned flavor over a period of 64 days. However, because of the nature of the experimental design, two alternative interpretations must be considered before it may be concluded that the aversion was directed at the flavor of sucrose: (1) since the 30-minute drinking period always occurred at the same time of day, the aversion could have been toward drinking during the time of day associated with poisoning, or (2) since the sucrose was always offered in the same location, the aversion could have been directed to the position associated with poisoning.

The possibility of a temporal aversion is eliminated upon comparison of Tables 8 and 9. Table 8 shows a highly significant difference between the treatment groups in sucrose consumption in each of the 30-minute drinking periods (the data from the odd-numbered days). Table 9 shows no significant differences in total fluid consumption during these same 30-minute periods. This lack of difference in total fluid consumption rules out temporal aversion.

As noted above in the discussion of procedure, the confounding of taste and position aversions was anticipated and was tested by (1) reversing the sucrose and water positions, and (2) presenting water in both positions. Figure 19-A shows the fluid consumption in both groups on the last day (day 73 of Figure 18) with

sucrose in the preferred position. Figure 19-B shows the fluid consumption with sucrose in the nonpreferred position. The nearly perfect mirror imagery of the two figures shows convincingly that the animals are tracking the flavor, not the position of the solutions. Further confirmation is shown in Figure 19-C which shows the fluid consumption on day 77, when water was offered in both positions. There is obviously no difference in the position preference of the NaCl and the LiCl groups.

With the dismissal of the two alternative interpretations of the data, the conclusion is clear: in adult F_1 P. m. bairdi taste aversion learning can be a long lasting phenomenon. A single flavor/toxicosis pairing is capable of altering an animal's flavor preferences for at least two months, provided alternate flavors are available. Since Peromyscus have a life expectancy in the wild of approximately 6 months (Howard, 1949), and since these animals were at least 4 months old at the beginning of the experiment, it is possible to restate the conclusions in a more dramatic manner: A single toxic experience with a novel flavor can cause a Peromyscus to avoid that flavor for the rest of its life, even if it re-encounters that flavor as often as every other day.

EFFECT OF AGE AND GENETICS

Introduction

The overall purpose of the experiments reported thus far has been to determine the basic attributes of taste aversion learning in Peromyscus while simultaneously analyzing these attributes to determine if they are consistent with the hypothesis that taste aversion learning might play an important role in overall foraging strategy. The results of these experiments may be summarized: (1) Single trial taste aversion learning does occur in Peromyscus, even if the illness follows the flavor ingestion by an appreciable delay; (2) Although there is some generalization from the flavor specifically associated with toxicosis, the greatest aversion is specific to the flavor paired with illness; (3) Although acquisition of the aversion is somewhat reduced by safe familiarization with the flavor, extinction of the aversion is not accelerated, and may even be delayed, by safe familiarization with the flavor; and (4) If extinction is not forced, the aversion is long lasting. These results are all compatible with the hypothesis that taste aversion learning might contribute to overall foraging strategy. Indeed, if taste aversion learning acts in such a way that it enables an animal to avoid a flavor once paired with illness for the rest of its life, it can profoundly influence the animal's foraging strategy in regard to the types of food eaten.

With these considerations in mind, I decided to shift the emphasis of my research from further elucidation of the fundamental properties of taste aversion learning toward a functional analysis by attempting to relate the phenomenon to three independent biological variables, which I list below along with some of my predictions concerning the effects of the variables: (1) age--if any time in a mammal's life involves especially high risks of ingesting toxins, it is the period immediately following weaning. Animals at this time are selecting items for their diet with virtually no learned information regarding these items' relative safety or toxicity. (2) Habitat/subspecies--if animals live in habitats with different probabilities of encountering toxins, they might show different abilities to form taste aversions. (3) Domestication--if taste aversion learning is actively maintained in a population by strong selection pressure, the relaxation of that pressure associated with 30 years of laboratory breeding might have some effect upon taste aversion learning. Any correlations detected between these variables and taste aversion learning would provide a powerful first step in the functional analysis of the phenomenon.

Purposes

The purposes of this experiment are threefold: (1) to determine the effect of age differences upon taste aversion learning in *Peromyscus*; (2) to determine the effect of habitat/subspecies differences (specifically to contrast a desert subspecies, *P. m. blandus*, with a grassland subspecies, *P. m. bairdi*) upon taste aversion

acquisition in Peromyscus; and (3) to determine the effect of domestication upon taste aversion acquisition in P. m. bairdi.

Test Subjects

All of the animals in this study were experimentally naive adult and young of P. m. blandus, of P. m. bairdi F_1 from wild caughts, and of domestic P. m. bairdi reared according to the description in the General Methods section.

Procedure

The procedure was intended to test the effect of age, subspecies, and domestication upon the acquisition of a LiCl-induced taste aversion to a 20% sucrose solution. Since this involved the simultaneous testing of a number of variables, a fully filled three-way factorial design was employed. Figure 20 illustrates this design. Twenty-five animals were assigned to each of the 30 cells. The age categories were: young = 25 to 30 days of age, old = 100 to 160 days. The genetics categories were: A = P. m. blandus, B = P. m. bairdi F_1, and C = P. m. bairdi domestic. The dosage categories were: 1 = no injection controls, 2 = 1.0 mEq/kg LiCl, 3 = 3.0 mEq/kg LiCl, 4 = 9.0 mEq/kg LiCl, and 5 = 9.0 mEq/kg NaCl. The no injection controls were necessary to determine if differences existed in the baseline sucrose consumption among the various age and genetic categories. The three different LiCl dosages were employed because of the potential problems arising from ceiling and floor effects as discussed above (page 90) in the Basic Taste Aversion Learning I experiment. That is, since there was no way to predict the direction nor the strength of

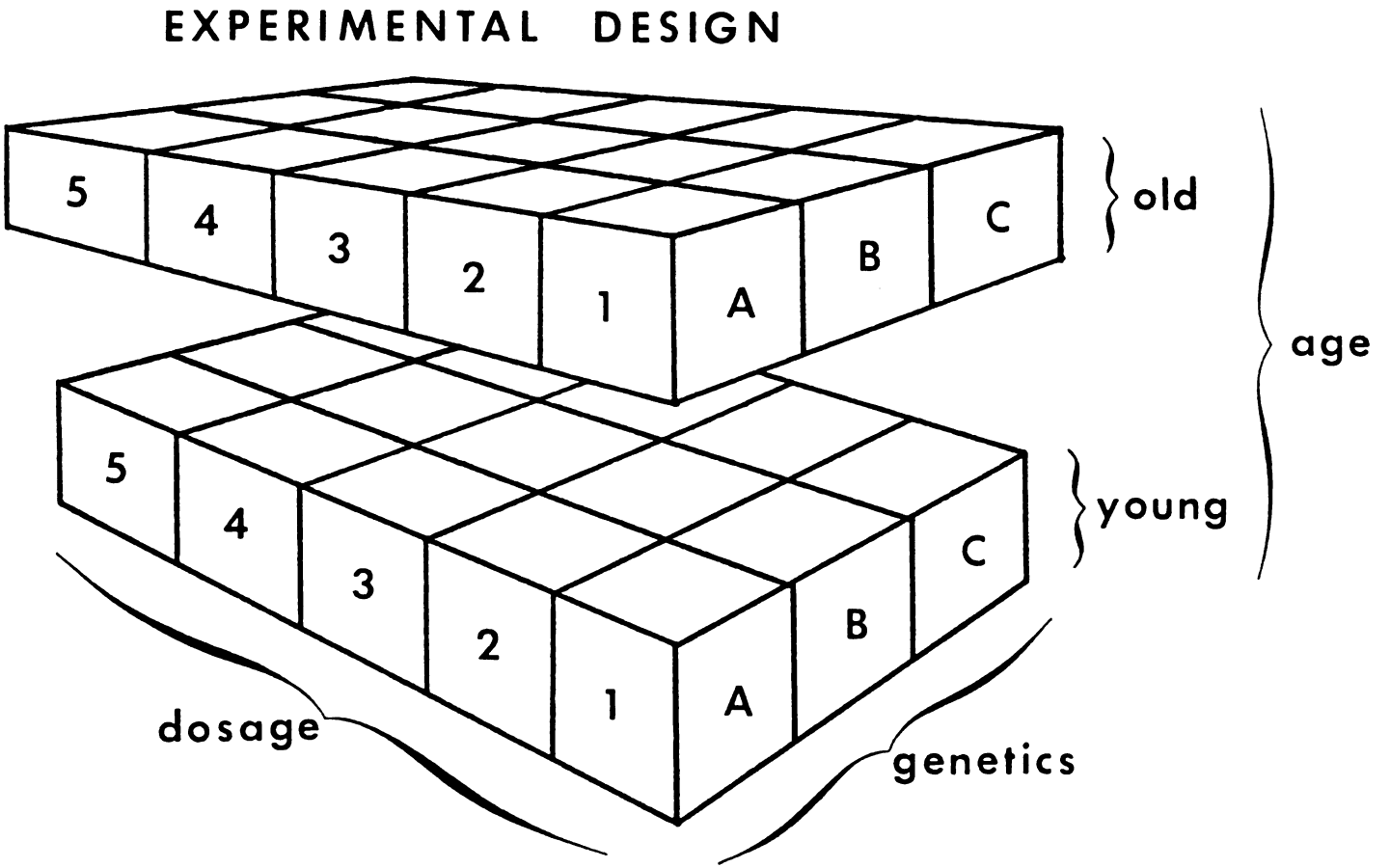


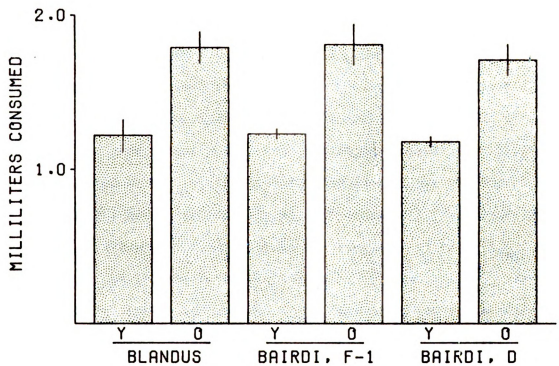
Fig. 20.--Factorial experimental design. See text for an explanation of the categories.

the various age and genetic effects, it was important to design the experiment so that any results obtained would not be obscured by being too close to the limits of either floor or ceiling. Thus, bearing in mind the dosage effect data given in Figure 10, these three LiCl dosages were selected. The 9.0 mEq/kg NaCl controls were included to determine if any differences detected among the LiCl groups could be attributed simply to differences in response to injection.

Animals were picked from their respective age and genetic categories, assigned randomly to 1 of the 5 dosage categories, and housed individually in plastic cages. They were then offered water on the basic fluid schedule of Figure 5 and Table 1 for 4 days. On day 5 each group was offered a 20-minute presentation of 20% sucrose solution, then immediately injected with the assigned substance. The volume of the injectant was held constant for all groups at 0.015 ml/gm of body weight. Therefore, the dosage was controlled by varying the molarity of the injected fluid. The no injection animals were handled in the same manner as the injected animals, except that no injection was administered. On day 6 all animals received water. On day 7 all animals were offered 20% sucrose for 20 minutes.

Analysis

With this design, analysis of the results must proceed in a stepwise fashion. The first question to be answered is, do the results of the 6 no injection control groups (the 6 groups of dosage level 1 in Figure 20) indicate differences in baseline sucrose consumption? Figure 21 shows clearly that such differences do exist. Thus, before analysis of the injected groups can proceed, the data must be



ANALYSIS OF VARIANCE

SOURCE OF VARIANCE	DF	MS	F
TREATMENTS			
AGE			
OLD VS YOUNG	1	11.648	47.891**
GENETIC LINE			
P. M. BAIRDI VS P. M. BLANDUS	1	0.009	0.040
BAIRDI DOMESTIC VS BAIRDI F-1	1	0.137	0.563
INTERACTIONS			
AGE WITH BAIRDI VS BLANDUS	1	0.004	0.017
AGE WITH DOMESTIC VS F-1	1	0.023	0.093
ERROR	144	0.243	
TOTAL	149	SS=46.845	

Fig. 21.--Mean sucrose consumption (± 1 SE) of the no injection control groups. Asterisks indicate the level of significance. Y = young; O = old.

standardized to eliminate this baseline difference. Weight-specific standardization was rejected because it introduced P. m. bairdi vs. P. m. blandus differences while eliminating the age difference. Therefore, it was decided to standardize the data of the injected groups in terms of the expected value for noninjected animals of the same age and genetic category. Each individual animal's consumption is represented as X_{dagn}, where d indicates dosage level, a indicates age level, g indicates genetic level, and n indicates replicate (25 per cell). The mean for any cell in the design is given by $\bar{X}_{dag}$, and the mean for any of the 6 noninjected cells is given by $\bar{X}_{lag}$. Thus, each animal's actual consumption, X_{dagn}, was converted to an index of consumption, I_{dagn}, according to $I_{dagn} = X_{dagn}/\bar{X}_{lag}$. That this successfully removes the baseline differences can be demonstrated by performing the index conversion upon the data for the no injection animals themselves and then recalculating the means for these groups. When this is done, all of the means are standardized to 1.0, but the variance remains intact since the standardization is performed upon each data point individually.

With the data of the injection groups standardized to remove baseline differences, analysis of the effect of age and genetic differences upon aversion acquisition may proceed. The next question is, were any ceiling or floor effects encountered? Figure 22 presents the mean index consumption values for all of the injection groups arranged by genetic and age categories. No indication of floor or ceiling effects exists, as a relatively clean dose/response effect is apparent in each age and genetic category. The absence of floor or ceiling

INDEX VALUES ON FIRST TEST DAY

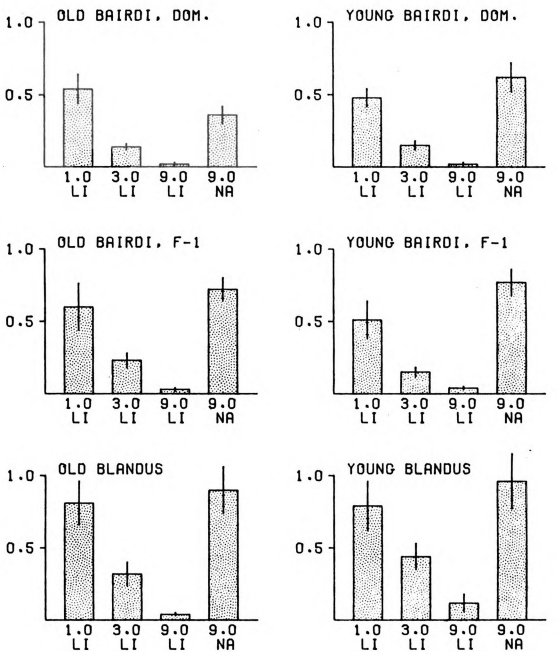


Fig. 22.--Mean index consumption data (± 1 SE) for all injected groups arranged according to age and genetic category. Numbers below abscissa give injectant dosage in mEq/kg. Numbers on ordinate give fluid consumption index values.

effects for the three different LiCl dosages enables these groups to serve essentially as replicates of the basic age-subspecies-domestication question, thereby providing strong corroboration if the results of the three levels of LiCl are in agreement.

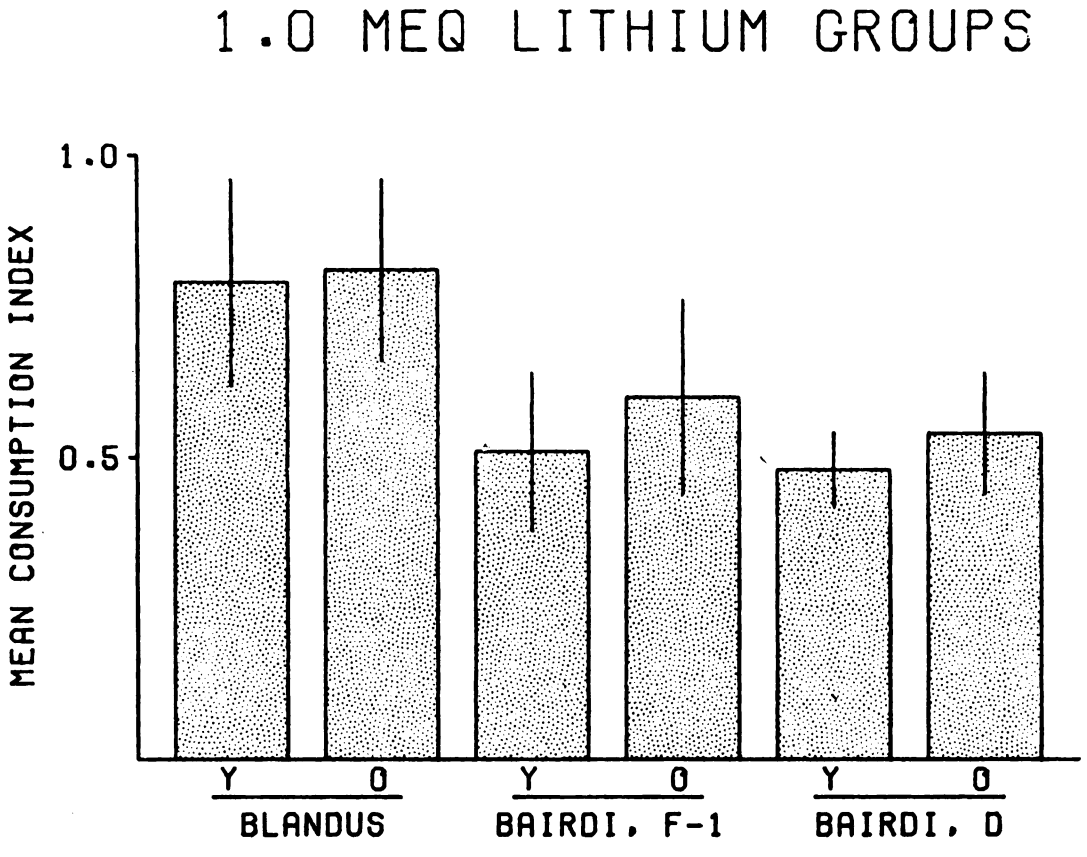
The data were examined using analysis of variance with a priori orthogonal contrasts so that the effects of all the relevant variables could be assessed with a single statistical operation. See Snedecor and Cochran (1967), pages 346 through 349, for a detailed description of this procedure.

Results

The results and statistical analyses for the LiCl levels and the NaCl level are given in Figures 23 through 26, respectively. Asterisks are given in the figures to indicate the level of significance of the various tests. Following convention, * = $p \leq 0.05$, ** = $p \leq 0.01$, *** = $p \leq 0.001$, **** = $p \leq 0.0001$, and ***** = $p \leq 0.00001$. Due to the complexity of the experimental design, the results will be presented and considered in a stepwise fashion.

Treatment Effects

Figures 23, 24, and 25 give the results for the 1.0, 3.0, and 9.0 mEq LiCl groups. As can be seen, there is no evidence of a main age effect, nor an age interaction at any of the 3 LiCl dose levels. Likewise, there is no evidence of a domestication effect at any of the 3 levels. Since each LiCl dose level represented an F test with a good sample size (df = 1/144), and since all 3 levels gave very low F values for these treatments, this experiment gives strong evidence that age

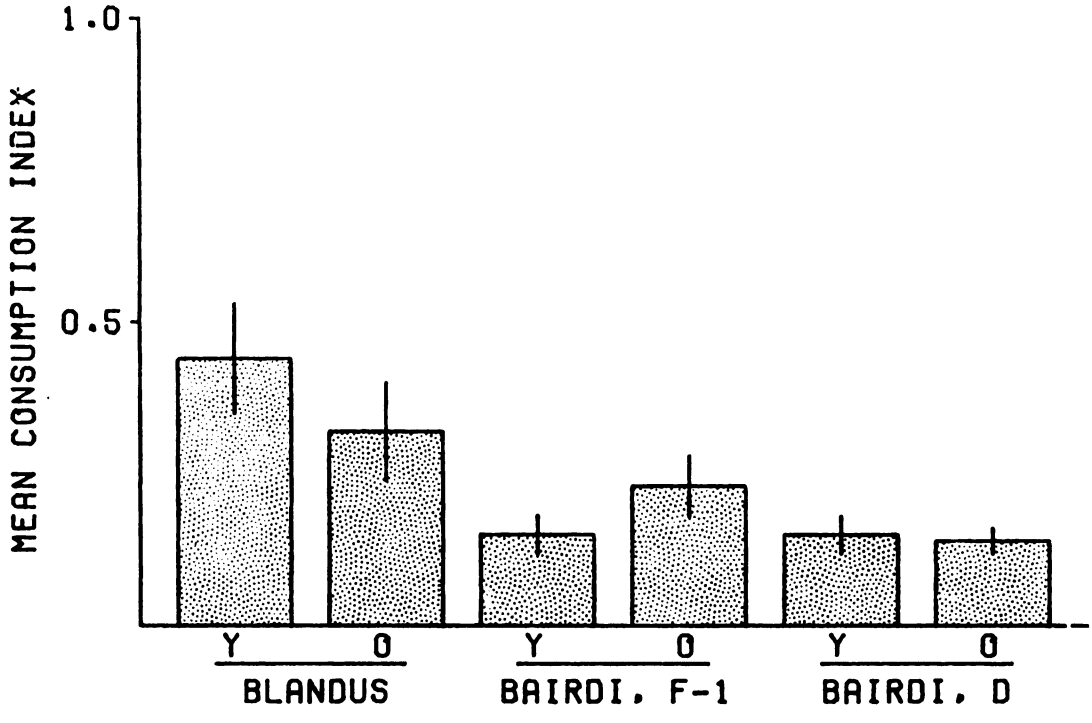


ANALYSIS OF VARIANCE

SOURCE OF VARIANCE	DF	MS	F
TREATMENTS			
AGE			
OLD VS YOUNG	1	0.121	0.946
GENETIC LINE			
P. M. BAIRDI VS P. M. BLANDUS	1	2.411	18.765****
BAIRDI DOMESTIC VS BAIRDI F-1	1	0.047	0.364
INTERACTIONS			
AGE WITH BAIRDI VS BLANDUS	1	0.30	0.232
AGE WITH DOMESTIC VS F-1	1	0.003	0.026
ERROR	144	0.128	
TOTAL	149	SS=21.115	

Fig. 23.--Mean index consumption data (± 1 SE) for the 1.0 mEq/kg LiCl groups. Asterisks indicate the level of significance of the F test. Y = young; O = old.

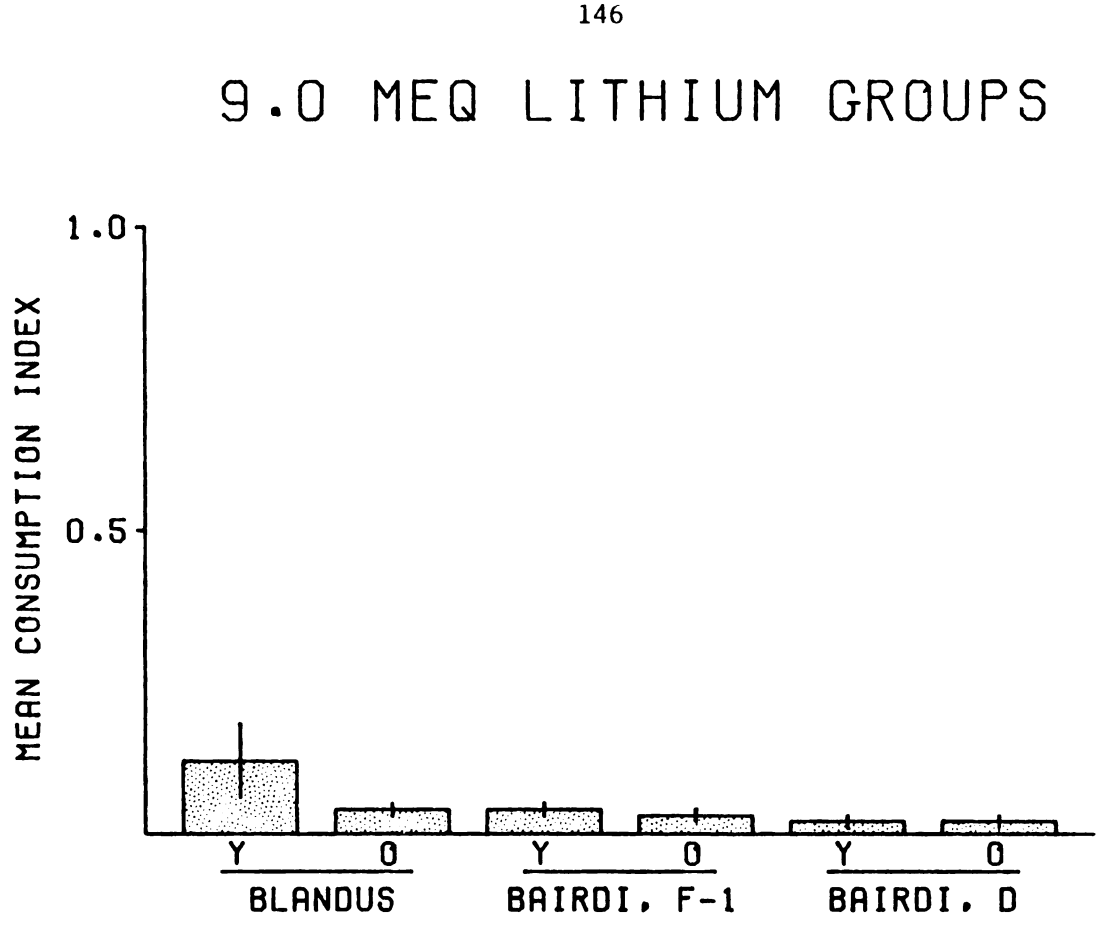
3.0 MEQ LITHIUM GROUPS



ANALYSIS OF VARIANCE

SOURCE OF VARIANCE	DF	MS	F
TREATMENTS			
AGE			
OLD VS YOUNG	1	0.019	0.392
GENETIC LINE			
P. M. BAIRDI VS P. M. BLANDUS	1	1.530	30.802*****
BAIRDI DOMESTIC VS BAIRDI F-1	1	0.045	0.902
INTERACTIONS			
AGE WITH BAIRDI VS BLANDUS	1	0.193	3.878
AGE WITH DOMESTIC VS F-1	1	0.050	1.003
ERROR	144	0.050	
TOTAL	149	SS= 8.992	

Fig. 24.--Mean index consumption data (± 1 SE) for the 3.0 mEq/kg LiCl groups. Asterisks indicate the level of significance of the F test. Y = young; O = old.



ANALYSIS OF VARIANCE

SOURCE OF VARIANCE	DF	MS	F
TREATMENTS			
AGE			
OLD VS YOUNG	1	0.028	2.159
GENETIC LINE			
P. M. BAIRDI VS P. M. BLANDUS	1	0.107	8.175**
BAIRDI DOMESTIC VS BAIRDI F-1	1	0.008	0.606
INTERACTIONS			
AGE WITH BAIRDI VS BLANDUS	1	0.050	3.848
AGE WITH DOMESTIC VS F-1	1	0.000	0.002
ERROR	144	0.013	
TOTAL	149	SS= 2.074	

Fig. 25.--Mean index consumption data (± 1 SE) for the 9.0 mEq/kg LiCl groups. Asterisks indicate the level of significance of the F test. Y = young; O = old.

and domestication have no effect upon taste aversion learning in Peromyscus. However, these same tests gave very strong evidence of a difference between the desert subspecies, P. m. blandus, and the grassland subspecies, P. m. bairdi. In the 1.0 mEq/kg LiCl groups this difference was significant at the $p \leq 0.0001$ level; in the 3.0 mEq/kg, LiCl groups the significance level was $p \leq 0.00001$; and even in the 9.0 mEq/kg LiCl groups, where consumption by all animals was pushed nearly to the floor, the difference was still detectable at the $p \leq 0.01$ level. The large sample sizes used and the agreement in the results across all 3 LiCl doses provides very strong corroboration for the conclusion that there is a striking difference in the response of P. m. bairdi and P. m. blandus to a sucrose/LiCl contingency treatment.

Interpretation of Effects

Alternate Hypotheses. Even with such overwhelming evidence of subspecies performance differences, conclusions regarding differences in taste aversion learning cannot be claimed to have been unequivocally demonstrated. With this design, subspecies differences in relative salience of the taste or toxic cues could explain the results. If it could be established that these results were produced solely by differences in taste salience or solely by differences in toxicity, the importance of these findings for an analysis of taste aversion learning would be greatly limited. On the other hand, if it could be shown that neither taste differences alone nor toxic differences alone are sufficient to explain the results, then the significance

of the findings is greatly enhanced. Since this experimental design involves 2 measured variables (dose of toxin and sucrose consumption) and 3 hypothesized sources of variation (taste salience, toxic effect, and aversion learning), analysis of these alternate explanations cannot proceed directly. But, additional manipulations do allow for indirect analysis.

Effect of Taste. It is possible to explain the results of all three LiCl dosage groups (Figures 23, 24, and 25) by asserting that a 20% sucrose solution tastes differently to P. m. blandus and P. m. bairdi and that this difference makes the flavor cue differentially salient for the two subspecies. Since variations in conditioned stimulus intensity have a well documented effect upon learning in general (see Gray, 1965, for a review) and taste aversion learning in particular (Dragoin, 1971; Nowlis, 1974), this hypothesized difference in flavor salience could explain the results presented thus far.

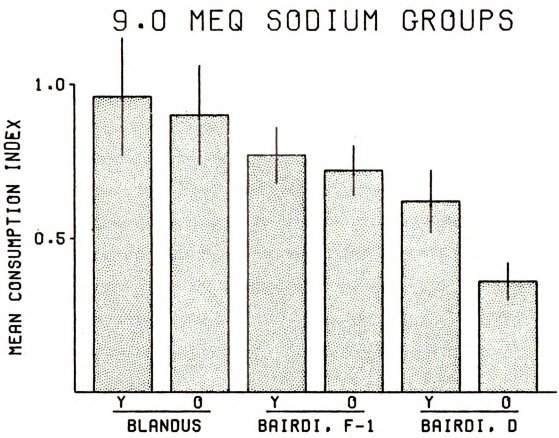
This claim may be easily tested: If the only difference between P. m. bairdi and P. m. blandus in taste aversion learning is a difference in taste salience, then any time a 20% sucrose solution is employed as a flavor cue in a taste aversion experiment involving these animals, results similar to those on Figures 23, 24, and 25 should always be produced, regardless of the toxin employed. More simply, holding the flavor constant while changing the toxin provides a test of the hypothesis that differences in taste salience are solely responsible for the subspecies differences observed.

Since an injection of 9.0 mEq/kg of body weight of 0.6 molar NaCl solution provides a severe osmotic shock potentially leading to

diuresis and concomitant dehydration, the results of the 9.0 mEq/kg NaCl groups, given in Figure 26, provide such a test. As is apparent, a rather different set of results is obtained when hyperosmotic saline solution is used as the toxic agent in place of a LiCl solution: Not only is there a strong P. m. bairdi vs. P. m. blandus effect, there is also a powerful domestication effect ($F = 14.801$, $df = 1/144$, $p \leq 0.001$) and a slight age effect as well ($F = 4.859$, $df = 1/144$, $p \leq 0.05$). The distinct divergence between these results and those obtained when LiCl is employed argues against the "taste salience only" hypothesis.

It is possible to counter by suggesting that the results of the 9.0 mEq/kg NaCl groups derive from a sampling error, a chance departure from the expected. After all, when the statistical analyses of all the LiCl and NaCl groups are considered together, some 20 orthogonal contrasts have been performed. Therefore, the chance occurrence of an unexpected result significant at the $p \leq 0.05$ level, such as the age effect observed in Figure 26, is not only possible, but actually expected. Likewise, it is not altogether unreasonable to suggest that even the apparently highly significant ($p \leq 0.001$) domestication effect could also derive from chance.

Since simple empiricism rather than eloquent rhetoric is the best rebuttal to this assertion, further experiments were carried out in which sucrose consumption was paired with 3.0 mEq/kg and 1.0 mEq/kg NaCl. Recall that the close agreement among the results for the 3 LiCl dosages strongly supports the conclusion that Figures 23, 24, and 25 accurately reflect the relative aversion strengths produced when



ANALYSIS OF VARIANCE

SOURCE OF VARIANCE	DF	MS	F
TREATMENTS			
AGE			
OLD VS YOUNG	1	0.543	4.859*
GENETIC LINE			
P. M. BAIRDI VS P. M. BLANDUS	1	3.194	28.603*****
BAIRDI DOMESTIC VS BAIRDI F-1	1	1.663	14.801***
INTERACTIONS			
AGE WITH BAIRDI VS BLANDUS	1	0.078	0.701
AGE WITH DOMESTIC VS F-1	1	0.270	2.419
ERROR	144	0.112	
TOTAL	149	SS=21.820	

Fig. 26.--Mean index consumption data (± 1 SE) for the 9.0 mEq/kg NaCl groups. Asterisks indicate the level of significance of the F test. Y = young; O = old.

20% sucrose is paired with LiCl. That is, with a sucrose/LiCl contingency the expected relative aversion strengths are: P. m. blandus << P. m. bairdi F_1 = P. m. bairdi domestic. On the other hand, Figure 26 suggests that with a sucrose/NaCl contingency the expected relative aversion strengths are: P. m. blandus < P. m. bairdi F_1 << P. m. bairdi domestic. But this result has been challenged as possibly deriving from random sampling error. To test this challenge, two additional experiments were carried out in which groups of adult P. m. blandus, P. m. bairdi F_1, and P. m. bairdi domestic (n = 25 for each) had sucrose consumption paired with injections of 3.0 or 1.0 mEq/kg of NaCl. The results of these additional experiments (which followed exactly the procedure outlined above, page 137, except that 3.0 and 1.0 mEq/kg of NaCl was injected) are given in Figures 27 and 28. The data for day 5, sucrose consumption immediately preceding injection, are given to show that no pretreatment differences existed among the groups. T-tests on the means for the trial following injection (day 7) showed that the relative aversion strengths at both the 1.0 and 3.0 mEq/kg doses were P. m. blandus = P. m. bairdi F_1 < P. m. bairdi domestic. This is in nice agreement with the results at 9.0 mEq/kg NaCl (the absence of a P. m. blandus vs. P. m. bairdi F_1 effect at the lower concentrations of NaCl is easily explained by noting the occurrence of a ceiling effect), thus corroborating the challenge to the "taste salience only" hypothesis. Differences in taste salience alone simply cannot explain all of the results obtained in these experiments. Since the 3 LiCl replicates are in agreement with each other and the

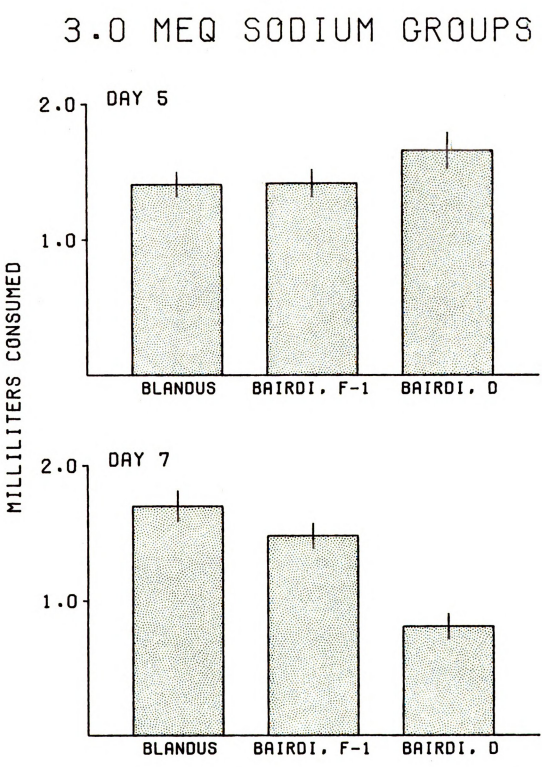


Fig. 27.--Mean sucrose consumption (± 1 SE) immediately preceding injection (day 5) and on the first test trial following injection (day 7). All groups were injected with 3.0 mEq/kg of NaCl.

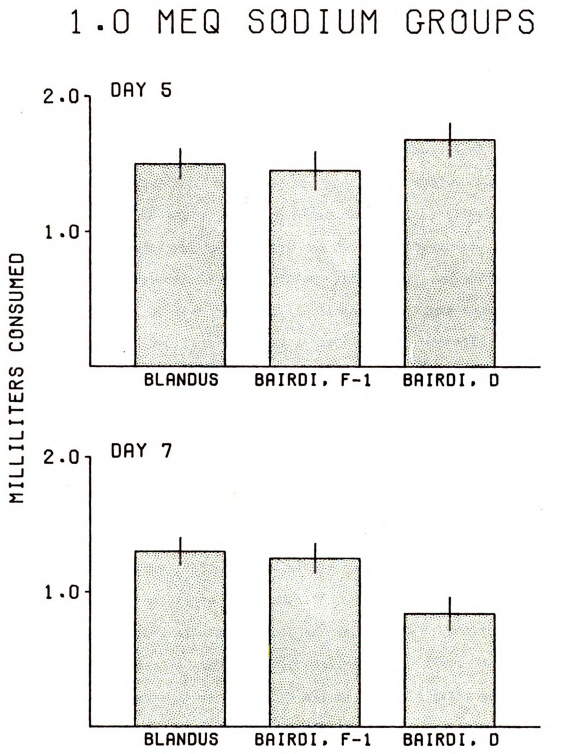


Fig. 28.--Mean sucrose consumption (± 1 SE) immediately preceding injection (day 5) and on the first test trial following injection (day 7). All groups were injected with 1.0 mEq/kg of NaCl.

3 NaCl replicates are in agreement, differences in taste salience alone must be rejected as the explanation for these results.

Effect of Toxin. Even with the rejection of the "taste salience only" hypothesis, it is still possible to explain all of the results obtained thus far, including the additional sodium corollaries, by claiming that the toxins employed exert differential illness-inducing effects upon the various age and genetic categories, and that the apparent differences in degree of aversion simply reflect differences in the level of induced illness. Since it is known that both LiCl toxicosis and NaCl loading produce dehydration (Radomski, Fuyat, Nelson, and Smith, 1950; Schou, 1957), and since the desert subspecies (most likely the best adapted to handle dehydration) always formed the weakest aversion, the direction of the observed differences fits nicely with this "toxic effect only" hypothesis. The additional difference between P. m. bairdi F_1 and P. m. bairdi domestic produced by NaCl injections can reasonably be explained by postulating that 30 years of access ad libitum to water has severely weakened the domestic strain's tolerance to dehydration.

Although this hypothesis that the sole cause of all the observed differences is simply a differential toxic effect is perfectly and reasonably consistent with all of the data gathered thus far, it is not a mere "catch all" hypothesis, but is subject to a simple test. Just as the "taste salience only" hypothesis could be tested by holding the flavor constant and varying the toxin, so the "toxic effect only" hypothesis can be tested by holding the toxin constant and varying the flavor. That is, if all of the differences

can be explained solely by differential toxic effects, then the results obtained with sucrose and a given toxin should hold for any other flavor and that toxin. Therefore, an additional experiment was run in which groups (n = 25) of adult P. m. blandus, P. m. bairdi F_1, and P. m. bairdi domestic had a quinine solution (3.0 grains of quinine sulfate in 800 ml distilled water) paired with an injection of 3.0 mEq/kg LiCl. This particular toxin and dose was chosen because it was the least likely to encounter ceiling or floor effects. Also, because I had no data indicating whether differential baseline consumption of quinine solution might occur, a group (n = 25) from each taxon was run with no injection and all of the data were standardized, $I_{dgn} = X_{dgn}/\bar{X}_{1g}$, as described above (page 141). The results for the three injected groups are given in Figure 29. Recall that with 3.0 mEq/kg LiCl the strengths of the aversions toward sucrose were P. m. blandus << P. m. bairdi F_1 = P. m. bairdi domestic, with the difference between P. m. blandus and P. m. bairdi being significant at the $p \leq 0.00001$ level (see Figure 24). However, t-tests on the quinine data of Figure 29 showed that when quinine was paired with 3.0 mEq/kg LiCl no difference, even at the $p \leq 0.20$ level, existed between any pair of means ($|t| \leq 0.9340$, df = 48). Clearly, a difference in toxic effect alone cannot account for all of the data now accumulated.

Summary

The rejection of the "taste salience only" and the "toxic effects only" hypotheses does not prove that differences in taste aversion learning have been demonstrated. Their rejection does,

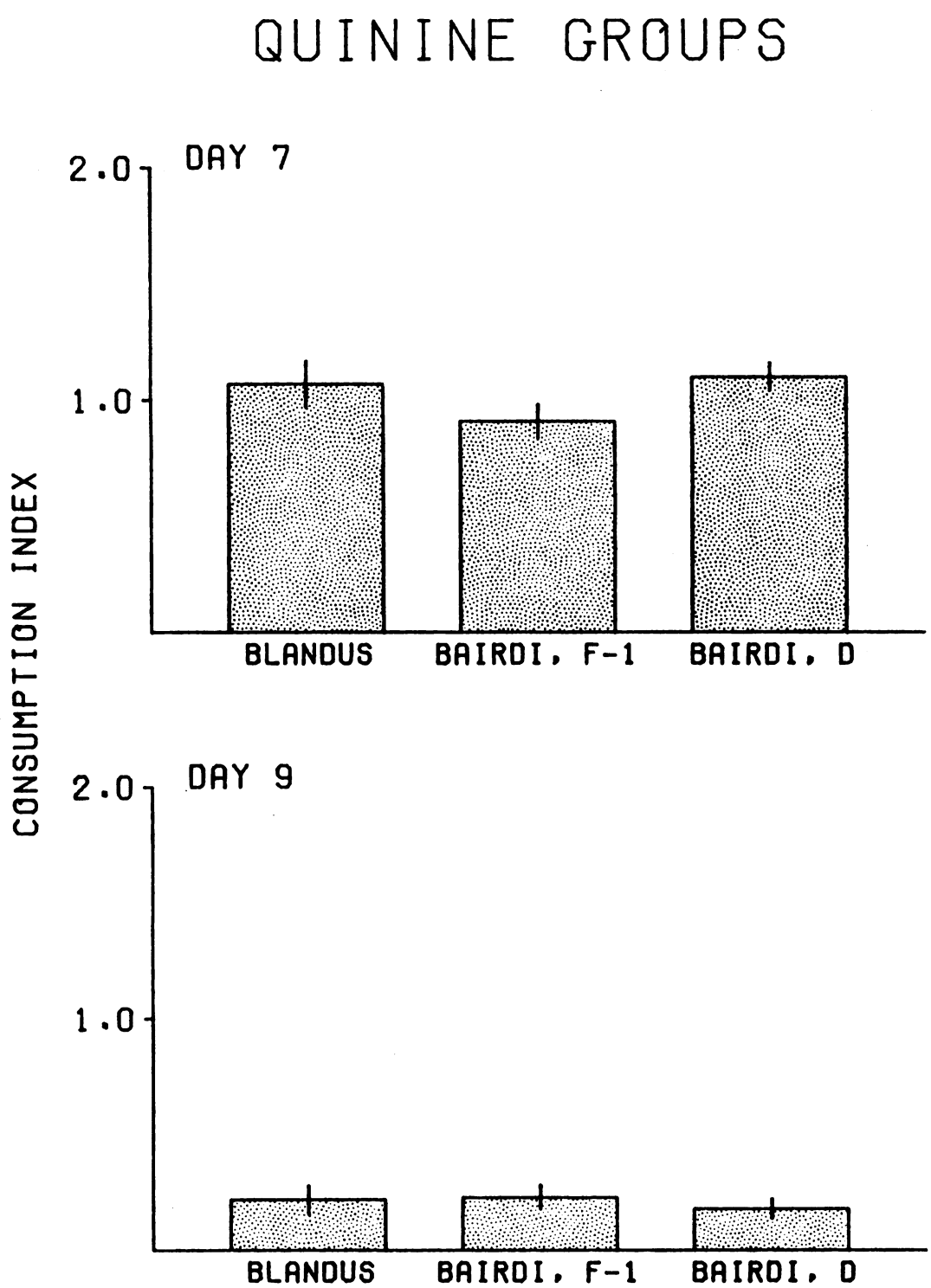


Fig. 29.--Mean consumption index data (± 1 SE) immediately preceding injection (day 7) and on the first trial following injection (day 9). All groups were injected with 3.0 mEq/kg of LiCl.

however, establish that either differences in taste aversion learning are occurring, or that a complex interaction of differences in taste salience, toxic effect, and taste aversion learning is occurring. In either event, the differences between P. m. blandus and P. m. bairdi in a taste aversion learning paradigm have been shown to be distinct, real, and of complex origin--thus potentially of great interest.

Conclusions

Age

No evidence for an age effect appeared in any of the three replicates using a sucrose/LiCl contingency (Figures 23, 24, and 25). This strongly supports the conclusion that an age difference, specifically that of weanlings versus adults, has no effect upon taste aversion acquisition in Peromyscus. However, there was an apparent age effect at the $0.025 < p < 0.05$ level in the sucrose/NaCl groups (Figure 26). Since the results of experiments involving multiple statistical comparisons must be interpreted with caution, and since this age effect was an unreplicated finding among many tests, I am reluctant to attach great importance to it and prefer to simply note that additional studies will be necessary to determine its validity. In any event, this possible evidence of a weak age effect with a sucrose/NaCl contingency is far overshadowed by the strong evidence against such an effect with a sucrose/LiCl contingency.

Subspecies

In the tests involving a sucrose/LiCl pairing, a powerful P. m. blandus versus P. m. bairdi effect was obvious with P. m. blandus

showing a distinctly weaker aversion. With a sucrose/NaCl pairing, the difference between P. m. blandus and P. m. bairdi disappeared and was replaced with an F_1 versus domestic effect in the P. m. bairdi. Finally with a quinine/LiCl pairing, no differences were found between any two groups.

These mixed, but replicated findings indicate that strong taste aversion acquisition differences do exist between P. m. blandus and P. m. bairdi. As discussed above, these results also show that these differences may not be attributed solely to simple differences in taste or toxin salience. However, at this point it is impossible to separate differences produced by differential taste aversion learning from differences produced by a complex interaction of taste salience, toxin salience, and taste aversion learning.

Domestication

As with age, the three sucrose/LiCl replicates show no evidence of a domestication effect upon aversion acquisition. However, with a sucrose/NaCl contingency, a moderately strong domestication effect was found in three replicates. Although this finding did allow a rejection of the "taste salience only" hypothesis, it did not provide any basis for determining the cause of the specific effect of NaCl injections upon taste aversion acquisition in domestic P. m. bairdi.

Although any attempt to answer this question in the absence of further data must involve speculation, I must confess to a suspicion that 30 years of laboratory rearing with water available ad libitum may have severely reduced the ability of the domestic P. m. bairdi to withstand salt loading. This speculation could be tested in a

number of ways--one of the simplest would be to look for differential mortality in groups of F_1 and domestic P. m. bairdi maintained with saline solution as the only available drinking fluid. Similarly, the LD_{50} for injected saline could be determined for the two groups.

Whatever the final answer, the present experiment has established that no domestication effects on taste aversion acquisition exist in P. m. bairdi when 20% sucrose solution is followed by an IP injection of LiCl, but that a domestication effect does exist if NaCl is employed as the injectant.

Summary

In the introduction to this experiment it was noted that "any correlations detected between these variables and taste aversion learning would provide a powerful first step in the functional analysis of the phenomenon." Such a first step has been made with these findings, particularly the very strong differences discovered between P. m. blandus and P. m. bairdi. It is tempting to begin immediate speculation linking the results of this experiment with the habitat differences encountered by a desert and a grassland subspecies. It is important, however, to bear in mind that all of the data generated by the many mice used in this experiment provide only a single test of the desert versus grassland question. Before too great a web of speculation has been spun, much additional work is needed.

At least two distinct paths are available for further research. The first would involve an effort to disentangle the relationships among taste salience, toxin salience, and aversion learning.

This could be done by continuing to vary the flavor and the toxin. The generation of dose/response curves relating flavor intensity to aversion acquisition could test the taste salience question, and the use of toxins with distinctly different physiological effects could test for specific toxin interactions. Finally, using taste cues in both taste aversion paradigms and other learning paradigms could check for differences unique to taste aversion acquisition. Despite its potential interest, this approach runs the risk of bogging down in a mire of detail long before any data susceptible to functional analysis have been generated. The second path could avoid this mire by directly pursuing the relationship of taste aversion learning to habitat. That is, if the present experiment were repeated with a different desert subspecies and a different grassland subspecies, and if the replicate produced results consistent with those already obtained, then a pattern linking specific differences in taste aversion learning with specific habitats would begin to emerge. From this linkage new hypotheses and tests of hypotheses could readily be generated. Finally, once a better understanding of the relationship of habitat to taste aversion learning has been established, an investigation into mechanism (i.e., into taste salience, toxin salience, and aversion learning) could proceed with less fear of miring in unessential detail.

Whatever path is ultimately followed, the melding of taste aversion studies with the theory of foraging strategies promises interesting new insights for both.

GENERAL DISCUSSION

Introduction

In the literature review it was pointed out that this dissertation would: (1) carry out an in-depth investigation of taste aversion learning in a nondomesticated species, and (2) begin a functional analysis of taste aversion learning by linking it to other aspects of diet selection. In this section the results obtained in these studies will be discussed in terms of these two goals. For clarity and simplicity, the two topics will be discussed separately and the results of the experiments will be treated in the order in which they were presented.

Taste Aversion Learning

The most important question when analyzing these results in terms of the existing taste aversion literature is: do these findings support or contradict the results obtained with other animals, particularly laboratory rats? Insofar as the present data support findings already obtained with white rats, they suggest the generality of the taste aversion phenomenon and the need to incorporate it into a general theory of diet selection. Wherever the present findings disagree with those previously obtained, they require an effort to detect the possible causes and significance of the differences. Of course, differences that are merely quantitative are of much less

importance than those that are qualitative. Thus, this section will attempt to relate the findings of this dissertation to results in the existing literature, noting the similarities and examining the differences.

The first experiment (pages 75-81, above), in which mice drank 0.2 molar LiCl and then were tested on 0.2 molar LiCl or 0.2 molar NaCl, produced results that were in close agreement with the most comparable rat study (Nachman, 1963a). This suggests that despite Kare's (1971) warning that taste sensitivities often vary greatly even between closely related species, equimolar LiCl and NaCl solutions taste similarly to both rats and Peromyscus. However, the companion pilot study, in which the cumulative consumption was recorded for mice drinking 0.2 molar LiCl or 0.2 molar NaCl (Figure 9, page 84), gave results for the deermice that were noticeably different from those reported for rats. Nachman (1963a) has provided a pair of mean cumulative drinking curves for rats drinking 0.12 molar LiCl and 0.12 molar NaCl that do not begin to diverge until 4 minutes have passed and in which the LiCl curve does not level off until 8 minutes have elapsed. Similarly, Rusiniak, Garcia, and Hankins (1976) give individual cumulative drinking curves for 7 rats drinking 0.12 molar LiCl solutions. Their results show that the first rat to stop drinking did so after 6 minutes, while the median rat stopped at 10 minutes, and one continued to drink throughout the 15-minute drinking period. The present work, on the other hand, found that 13 mice drinking 0.2 molar LiCl all stopped consumption completely by 2 minutes into the drinking period. However, even with these findings I am disinclined

to attribute a great deal of significance to this apparent rat/mouse difference. First, the rats were drinking a 0.12 molar solution, whereas the deermice were drinking a 0.2 molar solution, and it is reasonable to expect a more rapid onset of toxic symptoms from a more concentrated solution. Second, Rusiniak et al.'s rats had had prior safe experience with an equimolar NaCl solution and therefore would be more likely to show a delayed reaction. Third, mice are considerably smaller than rats. If the onset of toxicosis in any way depends upon the rate at which Li^+ ions are distributed throughout the body, it is perfectly reasonable that the mice should show an earlier onset of symptoms since they have both a smaller body and a higher weight-specific cardiac output than the rats. Finally, although rats are at least 10 times heavier than Peromyscus, they drank fluid at a rate only 3 times greater than the mice. Thus, they were acquiring their dose at a lower rate per unit body weight than were the mice. Since all of these factors could reasonably be expected to produce differences of the sort observed, it seems likely that the observed differences could be explained by them.

The next two experiments (pages 86-100) were intended to determine: (1) if Peromyscus could form an aversion to a flavor when the onset of illness followed the presentation of the flavor by 20 minutes, (2) the dose/response relationship between quantity of toxin and degree of aversion, and (3) if pseudoconditioning is involved in the formation of taste aversions by Peromyscus. The delayed illness and pseudoconditioning tests gave results in agreement with the extensive rat literature: long-delay learning can occur in Peromyscus

and pseudoconditioning is not implicated. However, the dose/response curve generated for the mice is somewhat different from one reported for rats (Nachman and Ashe, 1973). For example, mice injected with 0.3 mEq LiCl/kg of body weight drank 87% as much as their noninjected controls, while rats at that dosage drank only 56% of the control amount. At 1.0 mEq/kg, mice drank 73% while rats drank 17%. At 3.0 mEq/kg mice drank 33% while rats drank 0%. At 6.0 and 9.0 mEq/kg the mice drank 0%, but Nachman and Ashe did not test rats at these dose levels. These comparisons show a consistent displacement of the mouse and rat dose/response curves suggesting that the mice form considerably weaker aversions than do the rats. However, there were some important differences between the two experimental designs: (1) the rats were given 15% sucrose solution, the mice 20%, and (2) the rats were injected immediately following a 10-minute drinking period, the mice immediately following a 20-minute drinking period. Since recent studies have shown that the duration of CS presentation, the length of the CS/US interval, and the CS intensity all affect the formation of taste aversions (Bond and Harland, 1975; Andrews and Braveman, 1975; Barker, 1976; Dragoin, 1971), it is possible that these procedural differences are responsible for the apparent differences. In any event, a simple quantitative difference between the degree of aversion produced in rats and mice by similar toxin doses is hardly eventful. Much more important is the demonstration that Peromyscus, like rats, do form strong aversions after long CS-US intervals and that sensitization is not involved.

The next experiment (pages 101-111) in which four groups of deermice had four different flavors paired with illness and then were tested for the generalization of their aversions to other flavors, has no direct counterpart in the extant literature on other species. However, several separate studies have shown that rats can form aversions to a variety of flavors: acetic acid (Zahorik, 1972), apple juice (Hankins, Garcia, and Rusiniak, 1973), cat food (Wise and Albin, 1973), milk (Berger, 1972), hydrochloric acid (Dragoin, 1971), quinine (Braun and Snyder, 1973), sodium chloride (Kalat and Rozin, 1970), sucrose (Nachman and Ashe, 1973), and many others (see an exhaustive listing in Riley and Baril, 1976). But, no work has been done to test the generalization of aversions from one flavor to several others of distinctly different taste quality. To be sure, Domjan (1975) has shown that in rats aversions to saccharin generalize strongly to casein hydrolysate solution but not to dilute vinegar. However, when all of his results are examined, it appears that he has merely demonstrated that, to a rat, a 0.2% sodium saccharin solution has a taste very similar to that of a 5% casein hydrolysate solution. Despite the lack of directly comparable material in the literature on generalization of taste aversions, the present study was able to show that deermice, like rats, form strong taste aversions to a variety of flavors paired with a delayed illness, and that these aversions are relatively specific to the flavor paired with toxicosis.

In the next experiment, dealing with the effects of relative CS novelty upon aversion acquisition and maintenance, it was found that the acquisition data in mice were very similar to those found in

rats, but the maintenance data were distinctly different (see discussion on pages 112-119). Since Barnett has demonstrated that domestic rats show much less neophobia than do wild rats (Barnett, 1958, 1963), domestication may be implicated in this rat/mouse difference. If domestication does prove to be the cause, this experiment will have provided the first indication that some qualitative characteristics of taste aversion learning reported in rats may actually be artifacts of domestication and thus of limited use to a generalized theory of diet selection. A test of this possibility could be easily performed by carrying out an experiment on effects of novelty upon feral (or F_1-from-wild-caught) rats. Until such a test is performed, or until similar tests have been performed on enough other species to determine which is the more general response, the data regarding the effect of relative CS novelty upon aversion extinction should probably not be included in a general model.

In the duration of aversion experiment (pages 120-134), it was found that an aversion to sucrose solution, acquired after a single pairing with illness, lasted undiminished for 66 days, if mice had access to unflavored water at the same time as the sucrose solution. Although relatively little work on rats or other species has involved extended periods of extinction trials, one early paper by Garcia, Kimeldorf, and Koelling (1955) showed that rats which had had saccharin paired with radiation subsequently avoided saccharin almost completely for 12 days of continuous access to a bottle of saccharin and a bottle of water, and that consumption of saccharin did not return to baseline levels even after an additional 51 days of continuous access. A

recent study by Carey (1973) found that amphetamine-induced aversions to saccharin persisted undiminished for 50 days of continuous two-bottle fluid availability. Also, Grote and Brown (1973), using rats with a cyclophosphamide-induced aversion to saccharin, have shown that the rate of extinction (when only saccharin is available) is influenced by the fluid-deprivation level of the animals during the tests. They found that rats tested after 23 hours of fluid deprivation began to extinguish their aversion on the third or fourth trial and had completely lost it by the fifteenth. However, rats tested while only 10-hours deprived showed no extinction for 13 trials and were only drinking 50% of control level after 27 trials. Thus, it appears that the deermouse findings and the rat findings are in fairly close accord with regard to extinction. That is, if extinction is not forced by high deprivation levels, taste aversions are long lasting.

The final experiment (pages 135-160) tested the effects of age, domestication, and subspecies upon aversion acquisition and found that neither age nor domestication produced a detectable effect, while subspecies produced a highly significant effect. Since this was a fairly complex experiment, the results from these three tests will be discussed individually.

Age differences could be expected from two theoretical considerations: (1) Weanling mammals are selecting their own diet for the first time and would therefore be experiencing an exceptionally high risk of unwittingly ingesting a toxin. Consequently natural selection might act to provide them with a particularly sensitive taste aversion learning capability. (2) Taste aversion learning may

involve learning to learn. That is, an animal may require a great deal of experience with various sensory cues and various gastro-intestinal effects before it learns to specialize its association of gastro-intestinal consequences to food-related cues. The first of these considerations suggests that young animals should form stronger aversions than old animals. The second suggests that young animals should form little or no aversions. Grote and Brown (1971b) have shown that weanling rats can form taste aversions after a single flavor/toxicosis pairing. However, tests to determine whether young rats form stronger or weaker aversions than do old rats have not been definitive. Richter (1953) reported, "young wild Norways develop such responses more readily than do older rats," but he offered no data to support this contention. Grote and Brown (1971a) reported a significant age difference in aversion formation (cyclophosphamide-induced aversion to saccharin) between 21- and 70-day-old rats, with the young animals forming the stronger aversions: After a low dose of toxin the young rats drank 5.27 ml of the test solution, while the older animals drank 9.33 ml. After a high dose of toxin, the young drank 2.85 ml, the old 5.85 ml. However, since the authors did not adjust their data to standardize out age-specific differences in general fluid consumption, and since their young control animals drank .85 ml while their old control animals drank 16.77 ml, it is likely that their apparent difference in aversion formation is only an artifact. Also, Grote and Brown, in another study (1971b), reported that previously unpublished work (Grote's 1971 doctoral dissertation from the University of North Carolina) had found no differences in

aversion acquisition between weanling and mature rats. Thus, the absence of an age effect reported in the present dissertation is not without anticipation. However, since the previously published results were either unsupported by data (Richter, 1953), or supported by minimal data (Grote and Brown, 1971a, used only 3 or 4 animals per group), this work provides the first really powerful test and rejection of age differences in aversion acquisition. When all of the lithium groups of the present study are considered together, this dissertation compared the aversion acquisition of 225 young mice with that of 225 old mice and found no indication of a difference. Therefore, at least for Peromyscus, any hypothesis that predicts aversion acquisition differences between weanlings and adults must be soundly rejected.

Domestication effects upon aversion acquisition can be expected if taste aversion learning is a highly labile trait maintained in feral populations by strong selection pressure. Quantitative taste aversion learning differences between domesticated and feral rats have been well documented (Richter, 1953; Rozin, 1968). However, this present work found no domestication effect when comparing first-generation, laboratory-born P. maniculatus bairdi with P. m. bairdi deriving from a stock that has been bred in captivity for 30 years. Although this difference between rats and mice may at first seem surprising, it becomes less so after noting that the mice have simply experienced 30 years of laboratory breeding, with no active selection for "domesticated" traits, while the modern domesticated rat is the product of over 100 years of active selection and intense inbreeding (Richter, 1949, 1954, 1959). Thus, the fact that the "domesticated"

deermice have experienced much less genetic intervention than the white rat may explain the failure to find a domestication effect in Peromyscus. This failure is still of some interest, however, in that it indicates that the taste aversion learning phenomenon is not rapidly or easily modified by the relaxation of selection pressures which is assumed to accompany laboratory rearing.

Subspecies differences are to be expected if the subspecies are chosen from different habitats or niches which place different demands upon the taste aversion learning capacities of the animals. Genetic drift could also produce subspecies differences. Although strain differences have been reported among lines of domestic rats (Dragoin, 1971; Ader, 1973), these findings are not directly relevant to the subspecies comparison made in this work as the rat studies did not involve differences that could be linked to habitat differences, nor did the rat studies find particularly powerful differences. Since no other work exists in which comparisons have been made on the taste aversion learning capabilities of closely related species or subspecies these present findings of exceptionally strong subspecies differences ($p < 0.00001$, see Figures 23, 24, and 25, pages 144-146) must stand alone. Although it would be possible to speculate upon the specific causes of these observed differences and upon the specific linkages between these differences and the habitats of the mice involved, I am reluctant to do so, since this entire 750-mouse experiment represents only a single test of one grassland type versus one desert type. As an example of the hazards inherent in drawing conclusions from too few comparisons, Rozin (1975) has performed three

separate flavor-preference tests upon domestic rats and has found that they prefer Hebrew National over Genoa salami, gefilte fish over shrimp, and Mogen David over Virginia Dare wine. Yet, even with these three independent replicates he is reluctant to postulate kosher dietary selection in domestic rats. Likewise, with only 1 replicate, I am reluctant to postulate specific habitat-linked taste aversion differences in Peromyscus. However, I do intend to repeat this experiment using other desert and grassland species and subspecies of Peromyscus. If similar patterns are found, I will be led to feel that it would be worthwhile to explore causative hypotheses for specific connections between taste aversion learning and the habitats.

In summary, the in-depth research on taste aversion learning in Peromyscus performed in this dissertation has given results that for the most part are very similar to those reported for laboratory rats. (The only major exception was the reversed effect of CS novelty upon aversion extinction.) This indicates that the basic attributes of taste aversion learning demonstrated in studies on laboratory rats may not be attributed to the peculiarities of domesticated animals, but must instead be recognized as behaviors in the repertoire of naturally occurring populations. Since they can no longer be dismissed as domestication artifacts, and since taste aversion learning has been shown to produce profound and long lasting modifications to an animal's flavor preferences, it is time to attempt an integration of taste aversion learning into a general theory of diet selection. The next section will begin to do this.

Diet Selection

Throughout this dissertation frequent reference has been made to the need for an integrated theory of dietary selection which incorporates the predictions from ecological models and the findings of laboratory research on dietary preferences. Nearly all of the optimization models for foraging require that the animal be able to assess the relative value and density of the various prey types that it may encounter, then feed in such a manner that the diet is selected from those prey of highest value. Usually the assumption is made that value is based upon net caloric gain, but occasionally other measures of value are used (see discussion above, pages 48-53). Very few of the models discuss the potential methods by which the predator might assess the prey's value--they merely assume that the predator can and does do so. This oversimplification greatly restricts the applicability of these models to real situations. For example, a model based solely upon net caloric gain may be able to predict accurately the dietary selection of naive blue jays feeding upon a population of butterflies which includes Viceroys, but it would no longer be able to predict the dietary selection of those same blue jays once they had had an opportunity to attack Monarchs. The optimization models do not allow for the dramatic re-estimation of prey values that can occur when a predator encounters a noxious or toxic prey. That is, a significant failing of the optimization models is their assumption of a stable prey value hierarchy that does not change over the time scale in which the model is operating. This simple assumption is not necessary, as a fair body of psychologically oriented research exists

which can provide some insight into the food value assessment mechanisms employed by many animals. In fact, I believe that this literature is extensive enough to allow the development of a "black box" flow chart model which shows the interrelationships of the various decisions that go into an animal's assessment of a food's value. Figure 30 offers such a model.

To explain both the model and its symbolism, I will list the decision making and learning processes involved in a predator's encounter with a prey: (1) Starting at the top of the figure, a predator encounters (either as the result of an overt search or in some other manner) a prey of the i^{th} class, P_i. (2) Immediately upon detecting the particular prey item, the predator has acquired a bit of information regarding the availability of the i^{th} prey class, and the predator can then modify its estimate, D_{P_i}, of the density of the i^{th} class. (3) This modification in D_{P_i} may in turn lead to a modification of the predator's assessed value, V_{P_i}, of prey of the i^{th} class. (4) After assessing the value, V_{P_i}, of the prey encountered, the predator compares that value with V_m, a minimum acceptable level determined by the predator's hunger state. (5) If $V_{P_i} \geq V_m$, the predator will pursue P_i. If not, it will reject P_i. (6) After pursuing P_i the predator will either fail to capture it, whereupon it may modify its estimate of the prey's value, V_{P_i}, or it will catch it. (7) Upon capture and attempted ingestion, the predator will experience the immediate effects of the prey. (8) If these immediate effects are "bad" (the predator may have just captured a bumblebee instead of a robber fly), the predator will reject P_i and can then

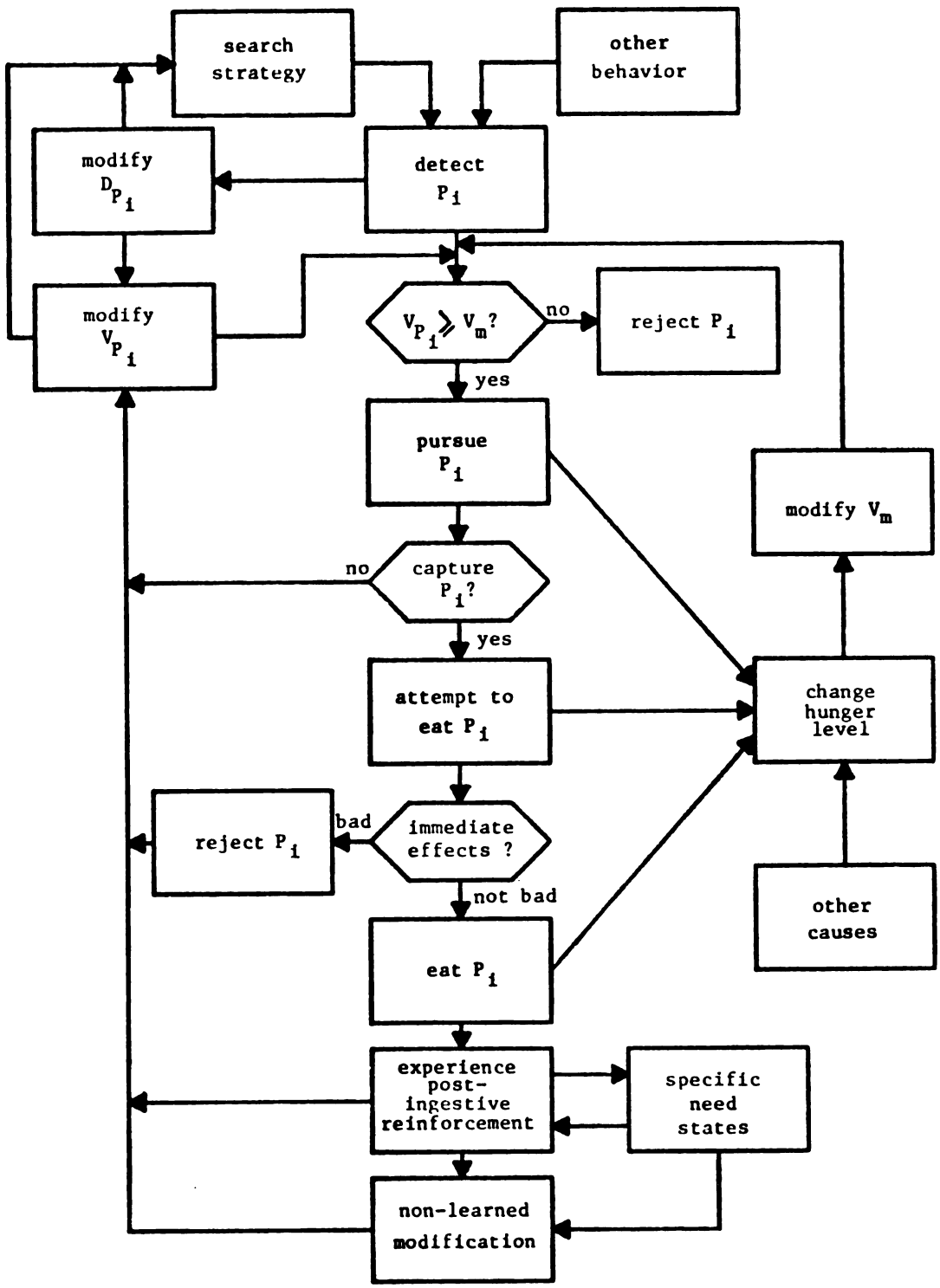


Fig. 30.--A flow chart representation of the decision making and learning processes involved in dietary selection.

re-estimate the prey's value, V_{P_i}. (9) If the immediate effects are not bad, the predator will eat P_i. (10) Meanwhile, throughout this entire process of pursuit the predator has been consuming calories producing a concomitant modification of V_m. Other causes, such as changes in ambient temperature may also have been affecting V_m. (11) After eating P_i the predator will experience the longer term effects of ingestion, i.e., it will experience "post-ingestive reinforcement." If this reinforcement is positive, e.g., relief from dietary deficiency symptoms, the predator may re-evaluate V_{P_i} upwards. If this reinforcement is negative, e.g., toxicosis, the predator may re-evaluate V_{P_i} downwards. These post-ingestive effects of the food may either affect or be affected by specific dietary need states of the animal. (12) Certain specific need states, e.g., sodium deficiency, may produce a nonlearned modification of V_{P_i}. (13) Finally, modifications in V_{P_i} or D_{P_i} may lead to modifications in the predator's search strategy. (Since this model is primarily concerned with dietary selection rather than search strategy, all such modifications of search strategy have been compressed into one box of the model.)

For a nonquantified model of this type to be of value, it must be fully justified and fairly complete. That is, no loops or processes should be included which are not required by data, and no data should exist which cannot be reasonably explained by the model. Furthermore, the model should be capable of producing testable predictions. If these criteria can be met, the model may be considered to offer an approximation of reality worthy of serious consideration. Then, further data gathering, designed around the concepts of the

model, may be performed in an effort to evaluate the sufficiency of the model and to quantify the processes involved.

The first step, a justification of the processes of the model, will be presented here: (1) As it is apparent that a predator can encounter potential prey either inadvertently or as the result of an overt search, the two encounter possibilities of the model are easily justified. (2) Predators do seem to be able to assess the relative density of prey items in their environment and to modify their search strategy and their assessment of the prey's value accordingly. Sudden switching of prey preferences in predators faced with prey classes of varying density is an example of this type of behavior. Murdoch, Avery, and Smyth (1975) offer both experimental data and a literature review in support of this phenomenon. (3) Justifying the assumption that the decision to pursue or not pursue involves a comparison of two values ($V_{P_i} \gtrless V_m$?) can be accomplished by showing that a predator's acceptance or rejection of a food item can be controlled independently by V_{P_i} or V_m. If animals under the same level of deprivation (thus presumably with the same V_m's) show differential acceptance of the same food class after differential prior experience with that food class (i.e., after assigning different V_{P_i}'s), the influence of a varying V_{P_i} measure upon food acceptability will be demonstrated. The dose/response curve for sucrose acceptance given in Figure 10 above (page 89) is such a demonstration. All of the mice in this figure were under the same level of deprivation, yet there was a significantly different acceptance of sucrose among the groups with different LiCl experiences. Similarly, if animals under different

levels of deprivation (i.e., with different V_m's) show differential acceptance of foods with fixed V_{P_i}'s, the influence of a varying V_m measure upon food acceptability will be demonstrated. Smigel and Rosenzeig (1974) have shown that seed selection by rodents is affected as predicted by deprivation. Young (1945) found that the acceptability of various foods to rats went up with increasing deprivation. Brown (1969) observed that deprivation will cause pigeons to accept seeds rejected under nondeprived conditions. Also, Grote and Brown's (1973) demonstration that the rate of extinction of a taste aversion is affected by deprivation level during testing further supports the concept of a varying V_m. (4) The box allowing a modification of V_{P_i} following the failure to catch P_i simply allows predators to learn not to pursue inaccessible prey. (5) The decision following capture (immediate effects?), along with the provision for modifying V_{P_i}, allows learning following an immediate noxious experience. It is separated from the longer term "post-ingestive reinforcement" because these two types of learning may show some significant differences (see the discussion of the special attributes of taste aversion learning in the literature review above) and because some data regarding two step learning processes necessitate the two decision levels: Brower's (1969) discussion of a blue jay's learned rejection of Monarch butterflies shows that the Monarch must first be ingested so that the blue jay can, via "post-ingestive reinforcement," assign a low V_{P_i} to prey with the taste of the Monarch. Then on subsequent encounters the blue jay assigns a low V_{P_i} to prey with the appearance of the Monarch after associating the Monarch's appearance with its taste via

"immediate effects" learning. Finally, after the blue jay assigns a very low V_{P_i} to insects with the appearance of the Monarch, no prey with that appearance are even pursued. (6) The possibility for long-delay "post-ingestive reinforcement" leading to modifications of V_{P_i} is included to allow for taste aversion learning, specific hungers, and other long-delay, food-related learning phenomena. This is shown interacting with specific need states because the make up of a meal can obviously influence subsequent specific need states and because the existence of specific need states can influence the quality of the post-ingestive reinforcement (cf. Weisinger, Parker, and Skorupski's, 1974, demonstration of the interaction between sodium deficiency, hypoglycemia, and a rat's ability to learn illness-based aversions toward sweet and salty flavors). (7) The possibility of nonlearned modifications in V_{P_i} arising from specific need states is included because of the well established fact that sodium deficiency produces a nonlearned preference for salty flavors (Rozin, 1975; Nachman and Cole, 1971). The need for the model to include <u>separate</u> control of learned and nonlearned modifications of V_{P_i}'s is established by evidence showing that a nonlearned preference for salty flavors, produced by a sodium deficiency, can antagonize a simultaneous illness-based learned aversion toward salty flavors (Frumkin, 1971, 1975; Weisinger, Parker, and Skorupski, 1974; Cullen, 1970b; Fregly, 1958; Nachman, 1962, 1963b). (8) Finally, provisions are made for the influence of ingestive behaviors and other factors upon hunger level. This in turn is shown to modify V_m, which then modifies the acceptability of various foods by changing the outcome of "$V_{P_i} \gtrless V_m$?"

Beukema (1968), in particular, has argued strongly that predator hunger level should be systematically included in any models that purport to explain diet selection or other predator-prey interactions.

If we assume, then, that all of the compartments in this flow chart are necessary to explain the existing data, we must next ask, are they sufficient to explain the data? At this point, a few assumptions, not specified in the figure, must be included: (1) the animals will never be perfectly reliable in their assessment and recall of V_{P_i}, D_{P_i}, or V_m. That is, each value will always be plus or minus some error term, e. (2) There is an innate starting input into the system. That is, all newborn animals bring with them an initial set of V_{P_i}'s which will then be modified by experience in accord with that species' learning abilities. Burghardt has provided evidence for innate V_{P_i}'s in his studies of prey preferences in newborn garter snakes (1969, 1975). (3) If a predator encounters several different prey items simultaneously, it will first rank the prey by V_{P_i}, then test the greatest V_{P_i} against V_m. With these three additional assumptions, it is possible to "walk" a number of examples through the model to show that it is capable of explaining them. The case of the two step learning process involved in a blue jay's aversion to Monarch butterflies has already been discussed. Grote and Brown's (1973) finding that deprivation level influences the extinction rate of a learned taste aversion also fits nicely with the model: Under states of high deprivation V_m is very low so that the V_{P_i} for the flavor associated with poisoning is not much less than V_m. An occasional conjunction of error terms such that $V_{P_i} \pm e \geq V_m \pm e$ allows the

animal to sample the flavor, P_i. Since no illness is associated with ingestion, post-ingestive reinforcement resets V_{P_i} a bit higher. Thus, on subsequent encounters the probability that $V_{P_i} \pm e \geq V_m \pm e$ is increased. With each safe ingestion of P_i, V_{P_i} is set higher and higher until the probability that $V_{P_i} \pm e \geq V_m \pm e$ approaches unity. At that point the aversion is fully extinguished. However, if the animal is tested under low deprivation, it comes into the situation with a relatively high V_m. Consequently the probability that $V_{P_i} \pm e \geq V_m \pm e$ is fairly low, and, on the average, several trials will have to occur before by chance $V_{P_i} \pm e$ does exceed $V_m \pm e$ and extinction can begin. (Note that a similar interaction of V_{P_i} and V_m could also explain the slow extinction of animals offered the aversive flavor and a safe flavor simultaneously--i.e., a two bottle extinction test.) The effect of deprivation level upon diet breadth predicted by optimal foraging models (e.g., Emlen, 1966, 1968; MacArthur and Pianka, 1966; Schoener, 1971; Werner and Hall, 1974) and observed by Smigel and Rosenzweig (1974) in heteromyids, by Young (1945) in rats, and by Brown (1969) in pigeons could also be explained by an interaction of V_{P_i} and V_m. Similarly the well established ability of vertebrate predators to learn to avoid noxious prey (Brower, 1969; Duncan and Shepard, 1965; Shideler, 1973; Alcock, 1971) can be explained by the "immediate effects" learning, and taste aversion learning and learned specific hungers can be explained by the "post-ingestive reinforcement" learning.

By now it begins to appear that this is an explain-all sort of model--useless for its ability to explain anything. This is

emphatically not the case. Implicit in the model, but thus far not explicitly stated, is the belief that the relative importance of these different processes will vary from predator to predator. After the dietary selection of a particular species has been studied sufficiently, it will be possible to provide quantitative estimates of the relative contributions of the different processes for that species. Then the model can be used to produce very specific, quantitative, and testable predictions regarding that species' diet selection strategy.

This approach to model building is not without precedent. Beukema (1969), in discussing the usefulness of deductive models in ecology, argues strongly in favor of an empirical model such as this one. In this type of model, behavior studies are first used to establish the basic attributes of the system, and only then is deduction employed to produce testable predictions. Beukema goes on to argue that the development of a quantitatively general theory of diet selection should not be attempted by pooling the data from a variety of species, since predators are likely to differ one from the other in the specifics of their predation strategies. Rather, one predator should be studied in depth to provide compelling evidence regarding that predator's strategy. The present model is precisely of that type. Only the qualitative aspects of the model are developed from data pooled from a variety of predators. Studies on individual species can then be used to provide species-specific quantification.

If it is now granted that this model is both necessary and sufficient to explain the variety of studies thus far considered, one last question remains: Is it <u>really</u> necessary to invoke such a

complicated model, as yet lacking in quantification, to explain dietary selection? Might not a simplified model relying only upon net caloric gain to assign value be sufficiently accurate to accomplish most of the goals of a theory of dietary selection? The latter question must be answered in the negative if it can be shown that the complicated model is required not only to explain an occasional taste aversion study, but also to explain many much more general studies. A number of lines of evidence can be presented to show the general need for the complicated model: (1) Several investigations of dietary preference have produced results at variance with the predictions of optimal foraging strategy models based exclusively on caloric considerations (e.g., Emlen and Emlen, 1975; Kear, 1962; Willson, 1971). (2) Many laboratory studies have shown that learning processes are important in establishing and modifying dietary selection preferences in animals. All of the taste aversion studies discussed above in the literature review and those presented in the body of this dissertation fall into this category. In addition, Hogan (1965) has shown that young Burmese jungle fowl (Gallus gallus spadiceus) are, upon first encounter, quite fearful of a mealworm and only learn its value as food after much experience. In terms of the present model, the experience is required to elevate the V_{P_i} for mealworms from its low initial value. Coppinger's (1969, 1970) studies of blue jay predation on butterflies have indicated that the jays are highly reluctant to take novel prey--that is, learning processes play a great role in regulating the acceptability of various prey types to blue jays. For his doctoral work, Rabinowitch (cited in Coppinger, 1969) raised chickens on

monotonous diets of either wheat or milo seeds. At 42 days of age the chicks raised on wheat were offered only milo and vice versa. Half of the animals starved to death rather than sample the novel food--a powerful example of the importance of experience upon diet selection. Finally, several workers, using artificial and natural model/mimic systems, have clearly established animals' abilities to learn to avoid noxious prey (e.g., Brower, 1969; Duncan and Shepard, 1965; Shideler, 1973; Alcock, 1971). (3) Field studies have shown that learning to avoid noxious or toxic prey can significantly modify predators' feeding preferences under natural or semi-natural conditions. Morrell and Turner (1970) showed that "wild (although admittedly urbanized) birds do indeed learn that prey (although admittedly artificial prey) is unpalatable, and later reject both it and its perfect mimic." Gustavson, Kelly, Sweeney, and Garcia (1976) distributed sheep carcasses laced with LiCl on a 3000-acre sheep ranch in southeastern Washington. They found that, "A comparison of this year's sheep losses with the rancher's past records suggested a 30-60% reduction in sheep killing following this application of taste aversion conditioning in the field." Brower, Cook, and Croze (1967), in a field study in Trinidad, released control moths and moths which had been painted to become artificial mimics of an indigenous unpalatable butterfly. They predicted that the protected mimics should be recovered, upon trapping, in greater numbers than the controls. In actuality, they found that more controls than artificial mimics were recovered. However, when they analyzed their data over time, they discovered that the mimics had had an initial advantage, but that this

had rapidly become a disadvantage. The authors then postulated that they had released so many artificial mimics over so long a time that they had swamped the model/mimic system causing the predators to learn either to discriminate between the models and mimics or to begin feeding upon both the models and the mimics. To test this, they repeated the experiment using fewer mimics over a much reduced time scale (3 days versus 24 days). In this case, they did recover the predicted excess of mimics, thus apparently supporting their swamping hypothesis. If this interpretation is correct, their data indicate that the local predators rapidly learned to shift their prey preferences in response to varying risks of toxicosis. (4) Freeland and Janzen (1974) have argued persuasively that the presence of secondary compounds in plants presents herbivores with special problems in dietary selection which are best handled by a learning strategy. Several authors have pointed out that plant secondary compounds are widespread and have suggested that they primarily serve a defensive role (McKey, 1974; Levin, 1971; Janzen, 1969; Wittaker and Feeny, 1971). Although the overall importance of plant secondary compounds in determining the diet selection strategies of herbivores has yet to be established, Freeland (1973) has gone so far as to suggest that they may be so important as to control vole population cycles. If true, this would certainly suggest an important role for learned dietary preferences in this species. It has been established that some mammalian herbivores can learn to avoid naturally occurring toxic plant products. In a study on the L-DOPA content of Mucuna seeds, Bell and Janzen (1971) found that local mammals learned to avoid the toxic mature seeds:

> All three of the medium-sized seed-eating mammals (Sciurus, Dasyprocta, Cuniculus) that live in areas of Central America where Mucuna seeds grow reject the opened seeds after eating a small amount (0.5 to 1 g) when first offered them. These same animals feed readily on immature seeds of Mucuna and on other large legume seeds that are protected from insects by other than chemical means.

Also, Sherbrooke (1976) found that Perognathus penicillatus, P. intermedius, and Dipodomys merriami refused, after initial sampling, "to eat [toxic] jojoba seeds, even while undergoing severe weight loss and finally death. Probably death results from starvation rather than the toxic effects of ingested seed." He has also stated (personal communication), "My observations . . . lead me to believe that jojoba seed rejection by these species is a learned aversion rather than an innate aversion to a new food item." Thus, empirical evidence (in addition to theoretical reasoning) shows that animals can and do learn to avoid naturally occurring toxic foods.

All of the material just presented (the failure of some tests of caloric optimization models to obtain the predicted results, the many laboratory and field studies showing the importance of learning in dietary selection, the widespread evidence of naturally occurring toxic compounds, and the demonstration that animals do learn to avoid these naturally occurring toxins) suggests that a complicated model involving many different methods by which a predator may modify its assessment of food value is in fact necessary if dietary selection is to be generally explained. Given this, and given that the literature review and the research presented in this dissertation have shown that taste aversion learning is a powerful molder of dietary preferences, the need to incorporate taste aversion learning into a general theory

of diet selection is, finally, obvious. The development and quantification of a definitive, integrated model along the lines suggested in Figure 30 will be a lengthy task, and I am not so presumptuous as to believe that the present work has done more than to initiate this effort. However, as Lao Tzu (translated in Baskin, 1972) has noted, "A journey of a thousand miles begins with a single step."

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