THE ROLE OF THERMOREGULATORY DEVELOPMENT IN PERFORMANCE OF HEAT-REINFORCED AUTOSHAPING IN CHICKS

by

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When chicks are run in a heat autoshaping procedure its regularly observed that after the level of autoshaped responding peaks it then begins to drop and continues to drop until little or no responding occurs at all. The overall goal of this study was to provide supporting evidence for the belief that this regularly occurring decrease in responding is the result of thermoregulatory development in chicks. A cold escape response hierarchy was developed to account for this proposed causal relationship. On the basis of this hypothesis a prediction was made that when the effectiveness of the chick's innate thermoregulatory responses to cold were reduced, there would be an increase in the occurrence of the learned operant thermoregulatory response of pecking the CS+.

This prediction was tested in Experiment 1, a heat autoshaping study in which a 10 mg/kg dose of propranolol was used to reduce the effectiveness of chick's innate thermoregulatory reactions to cold. As predicted, compromising the chick's innate thermoregulatory abilities resulted in an increased level of CS+ pecking. By demonstrating that the prediction was correct, the results of this experiment supported the proposed hypothesis on which the prediction was based. However, these results could not be regarded as conclusive evidence because the observed increase in responding could have been the result of some general activity stimulating effect of propranolol that was independent of its detrimental thermoregulatory effect.

Experiment 2 was designed to address this issue. The nature of propranolol's autoshaped response stimulating effect was examined in Experiment 2 by evaluating the drug's effect on performance in an autoshaping paradigm where thermoregulatory ability was not a relevant performance variable. A food autoshaping procedure was used for this purpose. Also, in Experiment 2 an attempt was made to replicate the results of Experiment 1 using a slightly modified heat autoshaping procedure.

In the heat autoshaping procedure of Experiment 2, a 10 mg/kg injection of propranolol caused no change in the chicks CS+ pecking activity. These results, which show that the above prediction about the drug's effect on the level of responding was incorrect, do not support the proposed hypothesis. This same dosage also caused no change in the response levels of the food autoshaping subjects. In both the Heat and Food groups 15 and 20 mg/kg doses of propranolol resulted in equivalent decreases in the level of responding.

The effects of these three different doses of propranolol suggest that a dose-response curve may exist for the drug's effect on autoshaped response levels. In such a dose-response relationship there would be an optimum dose of propranolol that would cause an increase in responding. A 10 mg/kg dose apparently was the optimum dose in the 16 day old chicks in Experiment 1, but fell short of the optimum dose for the 21 day old chicks in the second experiment. This suggests that the optimum dose might be age-dependent.

Further research on this hypothesis should next focus on demonstrating the existence of such a dose-response relationship in the heat autoshaping paradigm. Once an optimum dose is identified, the issue of whether propranolol really can result in an increase in autoshaped responding could be clarified. If the drug truly does produce such an increase, then the nature of its stimulating effect would have to be examined again using the optimum dose in a food autoshaping study.

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The Autoshaping Paradigm

One of the recent developments in experimental psychology for the evaluation and study of animal behavior and learning is the autoshaping procedure (Terrace, 1981). This procedure contains elements of both classical and operant conditioning. In the standard autoshaping experiment an unrestrained subject in a Skinner-box style experimental chamber is exposed to a series of pairings of a conditioned stimulus (CS) with an unconditioned stimulus (US). Over the course of such training the subjects acquire the behavior of approaching and pecking the CS despite the fact that such behavior in no way alters the predetermined sequence of CS-US pairings.

The true nature of the autoshaped response is a strongly debated issue. Some investigators hold that it is a classically conditioned response (Terrace, 1981) while others argue that it is an operant response (Herrnstein, 1977). In the standard autoshaping study the CS+ event is immediately followed by the onset of the US. This arrangement lends itself to adventitious reinforcement of responses to the CS+, an occurrence which supports the arguement that the autoshaped response is an operant response. However, it is difficult to explain the origin of the first autoshaped response from the operant perspective. This is not a problem for the classical conditioning theorists who have developed several reasonable Pavlovian accounts of the origin of the first autoshaped response (Terrace, 1981). Also, the observation that the autoshaped response resembles the unconditioned response (UR) lends further support to the classical conditioning interpretation of the response.

In the first published autoshaping study (Brown & Jenkins, 1968), pigeons learned to peck a CS composed of an illuminated key manipulandum paired with a food US. Since then the autoshaping procedure has been extensively studied

using different animals and a wide variety of CSs and USs. The procedure has also been widely employed as a tool for evaluating the learning ability of experimental animals. For example, in a study by Cunningham, Francisco, Kocarnik, and Metcalfe (1984), the effect of hyperoxic incubation on the learning ability of chicks was examined with a heat autoshaping procedure (Wasserman, 1973). In this procedure, chicks learn a discrimination between a colored keylight paired with heat reinforcement (CS+) and a different colored keylight that is unpaired with reinforcement (CS-). Conditioning takes place in experimental chambers maintained at a mean temperature of 9° C. Heat reinforcement is provided by a 5 s operation of a 250 W infrared bulb suspended above the ceiling of the chamber. Conditioning usually begins when the chicks are 2 days of age. Each conditioning session is 12 min in duration and involves 12 5-s presentations of both the CS+ and CS- for a total of 24 trials per session. Typically each subject receives two training sessions each day and training is usually carried out for between 7 and 10 days.

Linakis and Cunningham (1980) used a food autoshaping procedure (Brown & Jenkins, 1968) to evaluate how the learning ability of chicks was affected by exposure to ethanol during incubation. This procedure also involved the conditioning of a discrimination between two different colored keys. Instead of using heat reward in a cold environment, food reward was given to food-deprived subjects. Conditioning with food cannot begin until the chicks are 4 days of age because for at least the first 3 days after hatching the chicks continue to use their internalized yolk sac as their primary food source and food deprivation has no effect until this internal food supply is exhausted.

A comparison of the data from the studies described above reveals substantial similarities and one striking difference between the chick's performance in the heat and food procedures. Specifically, at the beginning of both heat and food rewarded training the mean percentage of trials with at least

one peck (%TWP) in each training session rises at a regular rate reaching peak levels by the second or third day of training for both the CS+ and CS-. The peak level for the CS+ is much greater than the peak level for the CS-. This difference between the two stimuli reflects the chick's capacity to discriminate between the reinforced and nonreinforced stimuli. After peaking, the rate of responding to the CS- in both paradigms drops to a very low level and remains at this level for the rest of the study. Responding to the CS+ also begins to drop after peaking, however, in the food paradigm this drop is relatively small. Responding to the CS+ in the food experiment will remain at or near the peak level for the remainder of the study. High levels of responding to the CS+ in the food autoshaping paradigm have been maintained for up to 18 days (Linakis & Cunningham, 1980).

However, this is not the case for responding to the CS+ in the heat paradigm. After reaching peak level, responding to this CS+ also drops slightly but instead of leveling out at or near the peak level, performance continues to decrease. Responding to the heat CS+ will decrease regularly over the course of the study until the response is either present at a very low level or is absent altogether in some subjects. By Day 7 in most of the heat autoshaping studies conducted in this laboratory some chicks exhibit a near zero level of responding; usually by Day 10 most subjects exhibit little responding to either CS. This phenomenon has been observed to be a regular occurrence in all the heat autoshaping experiments run in this lab. This performance decrease is quite consistent, always beginning on the second or third day of training. The pattern and rate of this decrease is quite similar from study to study. This decrease in performance will be referred to here as the "drop-off effect."

The observation that responding to the CS+ drops off in the heat but not in the food autoshaping paradigm suggests that there are qualitative differences in the factors that influence performance of the autoshaped response in the two

paradigms. At least part of this difference lies in the source of motivation for the autoshaped response and also in the type of reinforcer used in each procedure. In the food procedure, hunger brought on by food deprivation serves as the source of motivation; the reinforcer is delivery of food. In the heat study, on the other hand, the thermal imbalance produced by the cool chamber is the source of motivation and the reinforcement is the relief from the cold provided by the heat radiated from the heat lamp. In the food procedure, reinforcement is available only during the brief, scheduled US deliveries. This is not necessarily true for the heat procedure because other forms of heat reinforcement are available during the cold exposure (Schmidt & Rautenberg, 1975) besides the scheduled heat lamp US deliveries. Specifically, the chick's own innate thermoregulatory abilities can provide reinforcing relief from the cold. A chick's thermoregulatory reactions to the cold can occur throughout the entire training session; they are not limited to any particular time during a trial or session. Therefore, in the heat autoshaping procedure, some form of heat reinforcement is available throughout the entire session, not just during the scheduled US deliveries.

At hatching, the chick's innate thermoregulatory responses to a cold load are highly ineffective. The chick's thermoregulatory abilities mature rapidly during the first week after hatching and likewise its responses to cold are increasingly effective in maintaining a stable core temperature. On the basis of repeated experimentation and observation it appears that the rapid improvement of the chick's thermoregulatory abilities is correlated with the drop-off effect. The remainder of this paper is devoted to an explanation of exactly how thermoregulatory development could be responsible for the drop in performance and also to a description of an experimental approach intended to verify the causal nature of this proposed relationship.

Thermoregulation in Chicks

A background on thermoregulation in chickens is important to understanding the causal relationship suggested above. The chicken is a homeotherm that engages in two distinct classes of thermoregulatory responses to maintain a stable core temperature when exposed to cold (Bligh, 1973). One class of responses is composed of distinctive postures and patterns of skeletal muscle movements the animal will perform when exposed to the cold. This class of responses is referred to as the "physical" thermoregulatory reactions in the avian physiology literature. The second class of responses is composed of autonomically controlled heat conserving and heat generating reactions that occur within the body in response to cold. This second class of responses is referred to as the "autonomic" class of thermoregulatory responses in the avian physiology literature. Both the physical and autonomic thermoregulatory mechanisms are concerned with control of the rate of heat loss from the body. The autonomic mechanisms are further concerned with increments in the generation of heat to compensate for heat lost from the body.

When exposed to a cold load the chicken engages in species-specific physical responses that reduce the rate of heat loss from the body. These species-specific physical reactions include feather fluffing which increases the insulative protection of the body. The feathers provide the chicken with a highly effective thermal shield and serve a critical role in the adult chicken's defense against cold (Horowitz, Scott, Hillman, & Van Tienhoven, 1978). Chickens will also assume postures that cover poorly insulated surfaces of the body through which heat is lost at high rates, the result being a decrease in the loss of body heat (Freeman, 1971). This is observed in chickens as tucking the head under a wing thereby covering the comb and wattles. Another

characteristic posture is sitting or squatting which serves to cover the legs, feet, and underside of the torso. This sitting reaction to cold has been shown to reduce the rate of heat loss nearly 50% (Freeman, 1971, p. 1130). Fundamentally, these postural changes serve to reduce the total surface area of the body that is effective in heat loss and to reduce the overall surface to volume ratio. The physical class of thermoregulatory responses also includes behavioral responses to cold. Behavioral responses include locomotion out of uncomfortably cold areas and heat-reinforced conditioned responses performed in cold experimental settings. For convenience in later discussions in this thesis, the behavioral responses will be regarded as a separate class of thermoregulatory responses.

Autonomic responses to cold involve both increments in heat generation and reductions in the rate of heat loss. Mammals possess two types of autonomically controlled reactions to cold, shivering and nonshivering thermogenesis. Shivering thermogenesis involves autonomically controlled minute, rhythmic extensions and contractions of normally voluntary muscle. This reaction also leads to a reduction in the rate of heat loss since it causes vasoconstriction in cutaneous vasculature (Bligh, 1973, chap. 6).

Adult and particularly neonate mammals possess the autonomic response to cold known as nonshivering thermogenesis (Bligh, 1973, chap. 6), the generation of heat through the breakdown of fat stored in brown adipose tissue. This breakdown is caused by the release of epinephrine from sympathetic nerves that innervate this tissue. Administration of a beta adrenergic receptor blocker like propranolol can inhibit this thermogenic response. Nonshivering thermogenesis is most prevalent in neonate mammals who are born with large stores of brown fat. Nonshivering thermogenesis is the neonate mammal's major response to cold. As the mammal ages, the occurrence of nonshivering thermogenesis decreases, while in the mature adult mammal the response is absent

though possible.

Adult birds also exhibit shivering thermogenesis in response to cold. Nonshivering thermogenesis, however, is absent and not thought to be possible in adult birds (Bligh, 1973, chap. 21). This belief is supported by the observation that administration of adrenergic receptor blocking agents, which will inhibit nonshivering thermogenesis, has no detrimental effects on the adult bird's ability to thermoregulate. Also, avians do not possess brown adipose tissue, which is thought to be necessary for nonshivering thermogenesis. Instead they have white adipose tissue which is quite different in its physiological and biochemical properties. Although clearly absent in the adult there is strong evidence that some type of nonshivering thermogenic response exists in the chick (Wekstein and Zolman, 1967, 1969; Freeman, 1971, p. 1134). Among the evidence is the observation that when exposed to a cold load chicks show an increased metabolic rate accompanied by an increase in plasma levels of the products of the breakdown of stored fats. This increase in metabolism could result from both shivering and nonshivering responses to cold. Administration of propranolol has been successful in blocking this increase in metabolism (Wekstein and Zolman, 1967, 1969), however, it does not prevent thermogenic fat breakdown. This supports the conclusion that propranolol inhibits the chick's shivering thermogenic response (Freeman, 1971, p. 1136). Chicks exhibit thermogenic fat breakdown in response to an adminstration of norepinephrine suggesting that this reaction to cold is sympathetically mediated. However, this seems unlikely since it has also been demonstrated that blocking of adrenergic receptors does not inhibit this response (Freeman, 1971, p. 1137). Since norepinephrine has a strong thermolytic effect in adult birds, it is considered unlikely that norepinephrine mediates nonshivering thermogenesis in chicks. According to Freeman (1971, p.1138) nonshivering thermogenesis in chicks is most likely to be mediated by either glucagon or thyroid hormone (T3).

As in mammals, nonshivering thermogenesis disappears as the chicken grows.

Up until Day 19 of incubation the chicken embryo is essentially a poikilotherm although it does exhibit a minor, transient metabolic rate increase when thermally stressed (Romijn & Lokhorst, 1955). At hatching, the chick exhibits sustained increases in metabolism when exposed to cold. This is believed to reflect the operation of shivering and nonshivering thermogenic responses (Freeman, 1971, p. 1134). The newly hatched chick's thermoregulatory abilities, however, are highly ineffective and can maintain a stable core temperature only at the warmest of thermoneutral temperatures (Sherry, 1981). The poor thermoregulatory abilities of the newly hatched chick are attributed to such factors as the poor insulative quality of down, and to limited fat and carbohydrate stores. The neonate chick has a high surface to volume ratio which favors a high rate of heat loss from the body. Also, the chick's skin is highly water permeable which results in a high rate of heat loss through evaporation of water out of the body.

Because of its poor thermoregulatory capacity and its high rate of heat loss, the newly hatched chick requires an external heat source in order to maintain a stable core body temperature (Sherry, 1981). In the natural setting this heat source is the brooding mother hen. The neonate chick undergoes a variety of rapid developments that result in substantial improvement in the effectiveness of its thermoregulatory abilities. Within a week of hatching the chick is able to maintain a stable core body temperature without the aid of an external heat source and can effectively maintain a stable core temperature under a moderate cold load. This rapid improvement primarily reflects a decreasing rate of heat loss from the body resulting from physical and anatomical developments that occur in the chick. Contributing developments include changes in the relative proportions of the body that result in a more favorable surface to volume ratio. Also, there is a decrease in the water

permeability of the skin which results in a reduction of heat loss through water evaporation. Another important development is the replacement of down by feathers which possess a relatively superior insulative capacity (Sherry, 1981). According to the literature, the thermoregulatory abilities of the domestic chicken are thought to be fully developed anywhere from 2 to 5 weeks after hatching.

The Chick Has a Repertiore of Cold Escape Responses

Considerable thermoregulatory development occurs as the chicks grow over the course of the heat autoshaping study. At the beginning of the study, when chicks are 2 days old, the 12 min cold exposure produces core temperature drops of up to 4° C. This large drop reflects the neonate chick's relatively poor ability to maintain a stable core temperature under a cold load. As discussed above, in the first week after hatching the chick's thermoregulatory system becomes increasingly more effective. This improvement is reflected in the steadily decreasing size of the temperature drop produced by the 12 min cold exposure over the first week of the heat study. Usually by Day 7 of the heat study the cold exposure already produces little or no change in core temperature.

This rapid improvement in the growing chick's thermoregulatory ability is presumed to underlie the drop-off effect. A motivational hypothesis has been developed as one possible account of this causal relationship. As a homeotherm, the chick is able to maintain its body core temperature within a very narrow range known as the set point. Both the adult and neonate chicken have a set point temperature, although it is possible that the chick's is lower than the adult's. Also the range of temperatures composing the set point may be much broader in the chick than in the adult. If the chicken's core temperature drops out of the set point range, then the chicken can be considered to be in a state of thermal motivation. The state of thermal motivation can be conceived of as a drive to raise or lower core temperature back to set point. The further the chicken's core temperature deviates from set point, the greater the bird's level of thermal motivation.

Any measurement of body temperature that shows the drop in body temperature produced by the cold exposure could serve as an index of the chick's level of thermal motivation. In the heat autoshaping experiment each chick's rectal temperature is measured shortly before and immediately after the 12 min cold exposure. The pre-exposure temperature can be considered to represent the chick's set point temperature. The post-exposure rectal temperature is a measure of the chick's body temperature after 12 min at 9° C. The difference between a chick's pre and post-exposure rectal temperatures will show how far below set point the cold exposure caused the chick's core body temperature to drop. This difference can be used as an index of the bird's level of thermal motivation.

It is possible that a measured drop in rectal temperature is a relatively coarse indicator of the chick's level of thermal motivation. Schmidt (1976) conducted a study which suggests that the temperature of primary concern to the avian homeotherm's thermoregulatory system is the temperature of its spinal cord and rostral brain stem. This study demonstrated that in adult pigeons the rate of heat-reinforced operant responding in a cold setting was more closely related to spinal cord and rostral brain stem temperature than to body core temperature.

Even when their movements are restricted to the cold experimental chamber and locomotion away from the cold is not possible, chickens still possess other behavioral means of escaping the cold load. Horowitz et al. (1978) demonstrated that in a cold setting where performance of an operant keypecking response leads to heat reinforcement, adult hens engaged in two different behaviors to escape the cold. Normal hens, that had learned a heat-reinforced operant response, were observed to sit and fluff their feathers throughout a 1 h cold exposure and never performed the operant response during this exposure. A variety of body temperature measurements showed that these hens were able to maintain a nearly normal body temperature by engaging in this sitting response. As mentioned

above, sitting and fluffing the feathers is a basic avian heat-conserving reaction that helps the bird to maintain a stable core temperature when exposed to a cold load. These same hens were then shaved so that they could not use their innate postural reactions to cold in order to maintain a stable core temperature under a cold load. Without its insulating feathers, the chicken loses body heat at a very high rate when exposed to a cold load, more than it can compensate for with thermogenesis. When the shaved birds were placed in the cold operant setting, they performed the heat-reinforced response at a steady rate. Using the operant response, shaved hens raised the temperature in the chamber and maintained it at a level at which they were able to maintain a normal body temperature.

Presumably, if no means of behavioral thermoregulation were available, shaved hens would be unable to maintain a normal body temperature (Wekstein & Zolman, 1967). In the Horowitz et al. study (1978) then, shaved hens were able to use the heat-reinforced operant response to escape the body cooling effects of the cold load. However, when these birds had feathers, they escaped the body cooling effects of the cold load by engaging in the innate thermoregulatory reaction of sitting and feather fluffing. The heat-reinforced operant response and the innate sitting and feather fluffing response can both be considered behaviors that enable the hen to escape the body cooling effects of the cold load, i.e., escape responses.

In the heat autoshaping experiment, when chicks are placed in the cold experimental chamber, they are observed to engage in a variety of different behaviors. Most of the 12 min session, however, is spent engaging in three specific behaviors: pecking the CS+, squatting or sitting with fluffed feathers. It is possible to regard these behaviors as escape responses. Sitting or squatting with fluffed down is one of the chick's innate thermoregulatory reactions to a cold load. In the study discussed above, normal adult hens

escaped the body cooling effects of the cold load by sitting with fluffed feathers. Similarly, if the chick's thermoregulatory abilites are relatively mature, the innate thermoregulatory reaction of squatting or sitting with fluffed down enables the chick to escape the body cooling effects of the cold load. From this perspective it is possible to regard the observed behaviors of sitting or squatting with fluffed down as escape responses. It should be noted here that these heat-conserving responses of sitting or squatting with fluffed feathers may be accompanied by autonomically controlled thermogenesis.

In the heat autoshaping study the 5 s heat US delivered by the heat lamp composes 5 s of escape from the cold. For the short period the lamp operates, the area below the lamp is substantially warmer than 9° C. In the above discussion of the nature of the autoshaped response it was pointed out that this response could be an adventitiously reinforced operant response. The autoshaped response of pecking the CS+ is always followed by the heat US within a few seconds. Combining these two points, it is possible to regard the CS+ pecking response as an escape response. Specifically, as an operant escape response, pecking the CS+ always leads to 5 s of warmth provided by the heat lamp. Horowitz et al. (1978) demonstrated that shaved adult hens, who could not rely on their innate thermoregulatory reactions, engaged in a heat-reinforced operant pecking response to escape the cold. Similarly, chicks in the autoshaping study engage in an adventitiously heat-reinforced CS+ pecking response to temporarily escape the cold.

The three behaviors observed most often during the heat autoshaping sessions can all be regarded as escape responses. The chick then, possesses a repertoire of different behaviors that lead to escape from the body cooling effect of the cold. This repertiore contains both learned and innate escape responses. Schmidt and Rautenberg (1975) showed that such learned and innate responses can in some cases occur together while in other cases they are

incompatible and cannot be performed together. In this particular study, adult pigeons under a heat load learned a cold-air reinforced operant pecking response. The pigeons were observed to perform the operant response in combination with certain innate thermoregulatory responses to heat. However, some innate reactions, when they occurred, were incompatible with performance of the operant pecking response. A steady rate of operant responding would be interrupted by performance of such a response. One such innate reaction involves the pigeon laying down on one side while extending the wing on the other side of the body. It is not possible for the pigeon to engage in this innate postural response and to stand and peck at the same time. In this particular case, performance of the innate reaction to the heat load prevents or blocks the performance of the learned operant thermoregulatory response.

Schmidt and Rautenberg (1975) presented an outline of a motivational hypothesis to account for how ongoing cold-reinforced operant responding could suddenly be interrupted by performance of an incompatible innate response to the heat load. The fundamental idea they presented was that the different responses composing the pigeon's repertiore of learned and innate reactions to heat will each be elicited at a different level of thermal motivation. They stated that certain innate reactions to heat are elicited at a lower level of thermal motivation than the learned cold-reinforced operant response. However, it appears that the notion of a response hierarchy may be sufficient to explain these observations without appealling to motivational level. This will be considered in the following section.

The Chick Has an Escape Response Hierarchy

As described above, the chick has a repertiore of cold escape behaviors containing learned and innate reactions to the cold load. The escape responses the chick engages in most frequently during the cold exposures are sitting or squatting with fluffed feathers and the autoshaped CS+ pecking response. These escape responses can be considered to be organized into a response hierarchy. The response at the top of the hierarchy will be elicited first in the presence of thermal motivation; if this response is ineffective the second response in the hierarchy will be performed.

In the proposed hierarchy, the innate thermoregulatory responses of sitting or squatting with fluffed feathers together compose the response at the top of the hierarchy. Again, these heat-conserving postural responses are accompanied by autonomically controlled increases in heat production. The learned thermoregulatory response of autoshaped pecking of the CS+ is the second response in the hierarchy. The CS+ pecking response is elicited in the presence of thermal motivation only if the innate postural responses are ineffective.

Support for this proposed order can be found in the Horowitz et al. study (1978). When normal hens that had learned the heat-reinforced operant response were exposed to the cold load, the first escape response these birds engaged in was the innate thermoregulatory reaction of sitting with fluffed feathers. These normal hens never performed the operant response. Feathered hens performing this sitting response were able to maintain nearly normal body temperatures throughout the 1 h cold exposure. Shaving these hens made heat-conserving innate postural responses to cold like sitting ineffective. Shaving the hen essentially eliminated the sitting reaction from the hen's repertiore of thermoregulatory respopnses to cold. Shortly after being placed

in the cold chamber, shaved hens began to perform the heat-reinforced operant response at a high rate. These hens were able to raise and maintain the temperature of the chamber at a level at which their body temperature was nearly normal.

When the innate postural reaction to cold was not available, the hens engaged in the learned thermoregulatory response. However, when the innate response was available, these birds never engaged in the learned response. As long as the normal hen is able to effectively maintain a steady, near normal body temperature with the sitting response, there is no need for the next response in the hierarchy to be elicited. This explains why normal hens never performed the heat-reinforced pecking response. However, when these birds were shaved, they were unable to maintain core temperature by sitting. Consequently, upon exposure to the cold load, the learned thermoregulatory response was elicited.

A consideration of the relative energy efficiency of the two escape responses supports the proposed order within the hierarchy. When exposed to a cold load the chick will engage in autonomically controlled heat generation to compensate for its rate of heat loss. The greater the bird's rate of heat loss, the more energy it must invest in compensatory heat generation. In the heat autoshaping study, when chicks have been observed pecking the CS+, they are always standing and are usually moving about near the response key. Although the responses are not evidently incompatible, chicks sitting or squatting never peck the response key, even when positioned immediately in front of the key. As mentioned earlier, when sitting or squatting with fluffed feathers, the chick has a greatly reduced rate of heat loss. However, when the chick is standing and moving about while engaging in the CS+ pecking response, it probably has a relatively higher rate of heat loss. Therefore, when the chick is performing the CS+ pecking response it must invest more energy in thermogenesis than if it

were performing the heat-conserving innate postural responses. The order of responses within the hierarchy then could be based on the relative energy efficiency of each escape response.

The hypothesized escape response hierarchy operates in accordance with the concept that effective responses will be reinforced while ineffective responses will be extinguished. Specifically, an effective escape response is a response that reduces thermal motivation by providing relief from the body cooling effects of the cold load. An ineffective response is one that does not provide relief from the cold load resulting in the extinction of that response and performance of the next response in the hierarchy.

The escape response hierarchy could account for the proposed causal relationship between thermoregulatory development and the drop-off effect in the following manner. On the early days of the autoshaping study when the CS+ pecking level is high, the chick's thermoregulatory abilities are considered to be relatively immature. Shortly after being exposed to the cold, innate postural responses are elicited. However, because the chick's thermoregulatory abilities are immature, the innate postural reaction is ineffective and is extinguished. This response is replaced by the next response in the hierarchy, i.e., the CS+ pecking response, which is continuously reinforced by the heat US.

On the later days of the study when the CS+ pecking response level is low, the chick's thermoregulatory abilities are considered to be relatively matured. Again, shortly after being exposed to the cold load, the innate postural reaction to cold is elicited. At this age, however, the chick's relatively mature thermoregulatory reactions are able to provide escape from the body cooling effects of the cold load and are reinforced. As a consequence, the learned thermoregulatory response is not elicited.

Experiment 1

A preliminary study has been conducted that supports the escape response hierarchy account of the drop-off effect. The design of this study was based on the assumption that reducing the effectiveness of the chick's innate postural response to cold will cause it to be extinguished and lead to an increase in the occurrence of the autoshaped CS+ pecking response. The effectiveness of the innate postural response can be impaired pharmacologically with the beta adrenergic receptor blocker propranolol (Freeman, 1971). Wekstein and Zolman (1967, 1969) demonstrated that in chicks up to 20 days of age propranolol produces a lower baseline body core temperature, and impairs the chick's ability to maintain a stable core temperature when exposed to a cold load. As discussed above, propranolol probably inhibits autonomically mediated shivering thermogenesis that accompanies the postural response. Proranolol will, like shaving, essentially remove the innate postural reaction from the chick's repertoire of escape responses. Like the shaved hens who could not engage in the innate postural response to the cold load, the chicks under the influence of propranolol should also show an increase in the occurrence of the learned thermoregulatory response.

The basic design of this experiment is as follows. When chicks were an average of 2 days old, they began training in the heat autoshaping procedure. Training was conducted daily until the overall CS+ pecking levels of the group of chicks had dropped-off. The test of the effects of propranolol on performance was run on the day following the last day of training. On the Test day subjects were divided into two groups. The subjects in one group received an injection of propranolol, while the subjects in the other group were given an injection of saline. Shortly after receiving their injections, subjects were

run through one standard heat autoshaping session. Any significant difference found between the mean CS+ levels of these two groups was attributed to the effects of propranolol.

Method

Subjects

Fertilized chicken eggs (White Leghorn) were obtained from the Department of Poultry Science at Oregon State University (Corvallis, Oregon). One hundred forty eggs with a mean weight of 57.8 g (range 54 to 61 g) were incubated in two Marsh Roll-X forced air incubators (No. RX1A). Seventy eggs were set in each incubator. During the first 19 days of the 21 day incubation period eggs were turned automatically each hour. At the beginning of day 20 of incubation eggs were set for hatching.

Monitoring of the hatch began late in day 20 of incubation when it was apparent that the first chicks were going to hatch. The contents of the incubators were checked on an hourly basis to see if any chicks had hatched. When a chick was found to be totally free of its shell it was weighed and banded for identification. The chick was returned to the incubator for an additional hour. After this hour chicks were moved to a warm room (33-38 $^{\circ}$C) where they were housed individually in stainless-steel, wire mesh cages (24.5 X 17.5 X 17.5 cm). This room was continuously illuminated throughout the study. The chicks had free access to food and water in their cages.

This experiment was run as part of a larger project that was concerned with the effects of hyperoxic incubation conditions on the learning ability of chicks. The 70 eggs in one incubator were incubated under normoxic (room air) conditions. The 70 eggs in the other incubator exposed to air that was 60%

oxygen from Day 15 to Day 18 of incubation. The hyperoxic incubation conditions were created in accordance with a procedure described by Cunningham et al. (1984). Analysis of the data from this experiment showed that the condition of incubation factor (hyperoxic or normoxic) had no significant effect on the chick's learning or performance. This factor was dismissed and was not considered in the results section.

Apparatus

Autoshaping was conducted in four identical experimental chambers (30 X 28 X 33 cm). Three walls of each chamber were composed of sanded sheet-aluminum panels. The fourth wall was composed of clear acrylic and was hinged so that it served as the door of the chamber. The top of the chamber was covered with 1/4" wire mesh and its floor was composed of a piece of masonite covered by paper towels. The chick's movement within the chamber was restricted by a V-shaped wire mesh barrier. The barrier limited the chick's movements to a triangular area (21.0 X 21.0 X 30.0 cm) in front of the panel containing the response key.

The response key was a frosted plastic disk 2.5 cm in diameter (BRS/LVE, Model PPK-002). The key was situated 10 cm above the chamber floor, in the center of one of the aluminum walls. An in-line mini-projector (Industrial Electronics Engineers Inc., Model 00010-01-XXXX-44) mounted behind the response key was used to project red and green circles onto the key. The red light was created using Kodak Filter No. 29, the green light with Filter No. 60. A houselight was mounted at the top and center of the wall opposite the response key. The houselights, which were on throughout each autoshaping session, were miniature light bulbs (No. 1820) wired in parallel to a 24 Volt DC power supply.

Each chamber was enclosed within a separate 10.4 cubic foot (291 liter) refrigerator (Kenmore Model 564.8600110) maintained at a mean temperature of 9°

C during autoshaping sessions. Located above the center of each experimental chamber was an infrared heat lamp bulb (250 W). These bulbs were located about 32 cm above the floor of each chamber. An Apple II computer and electromechanical devices were used to control stimulus events and record response data.

Procedure

Training Phase. The heat autoshaping procedure commenced 22 days after the beginning of incubation. At this point the average age of the 32 chicks that began the study was 38 h (range 28 to 49 h). Of these 32 chicks, 6 did not complete the study because of either illness or death. Thus, on the final day of the study only 26 subjects remained. The statistical analyses of behavioral, temperature, and weight data involved only the data from these 26 subjects.

All subjects were exposed to two daily sessions of the discriminative autoshaping procedure inside the refrigerated experimental chambers. On the average the second session of each day began 2.5 h after the start of the day's first session. Subjects underwent a total of 13 consecutive days of training. A test of the hypothesis using propranolol was conducted on the fourteenth and last day of the study.

Ten min before the beginning of the first session of each day, the chicks were individually transported from their home cage to the laboratory in translucent white plastic half-gallon pitchers. On arrival in the lab, each chick's pre-session or baseline rectal temperature was measured using a digital readout electronic thermometer (Yellow Springs Instruments, Model 2600) connected to a thermister probe (YSI, Model 402). While the chick was held still on a flat surface, the probe was inserted 2.3 cm into the chick's rectum, and a temperature reading was taken after 45 s (to the nearest 0.1°C). Each

chick's weight was then measured and recorded. After these preliminary procedures, which took about 10 min to complete for a group of four chicks, the chicks were placed individually into separate experimental chambers. The autoshaping session was started as soon as all the chicks were in their chambers and the refrigerator doors were shut. The houselights were turned on simultaneously in each chamber at the beginning of the autoshaping session. In the short span of time between being placed in the chamber and the start of the session, the chick's are in the dark. The chicks do not peck in the dark and this prevents them from responding before the computer is monitoring their pecking behavior at the start of the session.

Each autoshaping session was composed of a sequence of 12 heat reinforced CS+ trials and 12 nonreinforced CS- trials arranged in orders described by Fellows (1967). The CS+ was a 5 s illumination of the response key with the red light. The offset of CS+ was followed immediately by the onset of the heat lamp US which operated for 5 s. The CS- was a 5 s illumination of the response key with the green light. Over the course of a session, subjects were exposed to a total of 24 stimulus presentations. Each trial was separated by a mean intertrial interval (ITI) of 30 s (range 25 to 40 s). The average duration of a single session of autoshaping was 14 min.

At the end of the last trial of a session the houselights were turned off and a second timer was switched on. Each chick's post-session rectal temperature was measured shortly after the end of the day's first session. The procedure followed for measuring post-session rectal temperatures was identical to the one used for taking pre-session temperatures. Subjects were removed from their experimental chamber and processed individually at 90 s intervals. Thus, the first chick in each group of four spent a total of 15.5 min in the cold chamber. The last chick to have its post-session rectal temperature measured spent a total of 20 min in the cold chamber. After all the chicks in the group

had their post-session temperatures recorded they were returned to their home cages.

On the second session of each day of autoshaping there were no weight or temperature measurements. Upon arriving in the laboratory chicks were promptly placed into their separate experimental chambers and the session was started as soon as all the refrigerator doors were shut. At the end of this session, all the subjects were removed from their experimental chambers within 30 s of the end of the session. All subjects were exposed to the cold chamber for about 15 min during the second session of each day. After removal from the chambers, subjects were returned to their home cages.

Test Phase. At the beginning of the Test day (Day 14) the group of 26 subjects was divided into two groups on the basis of the total number of keypecks they made on Day 13, the last day of the training phase. Assignments were made such that both groups had equivalent distributions of high, medium, and low activity subjects. One of these groups was randomly designated the propranolol group, the other the saline control group. Subjects were an average of 16 days old at this point in time. On the Test day, subjects were exposed to only one session of the heat autoshaping procedure. Each subject's pre-session weight was used to calculate the volume of the injection the subject would receive during the test procedure. The 12 subjects of the propranolol group received an IP injection (1 ml/100 g of body weight) of a 1 mg/ml soloution of propranolol hydrochloride (i.e., 10 mg/kg). The 14 subjects of the saline control group received equivalent (1 ml/100 g) IP injections of physiological saline.

Forty min before beginning the test session, subjects were transported from their home cages to the laboratory where their baseline rectal temperature was measured. Immediately after the temperature measurement, each subject was

given its designated injection. When all the subjects were injected, they were returned to their home cages. Subjects remained in the home cages for 30 min, the span of time Wekstein and Zolman (1967) consider to be sufficient for the development of the effects of propranolol on thermoregulation. The subjects were then transported back to the laboratory and promptly placed into separate experimental chambers and run through a session of autoshaping. This session was otherwise no different from the 24 trial sessions used during the training phase. After the session was over, each subject's post-session rectal temperature was measured following the same procedure used on the preceding 13 days.

Behavioral Data

Over the course of each autoshaping session, the computer recorded the number of keypecks made by each subject during the CS+, CS-, US, and ITI periods of each trial. Because the CS+ pecking response is considered to be the learned thermoregulatory response in the escape response hierarchy, the CS+ pecking data are of greatest relevance for evaluating whether this experiment supports the proposed hypothesis. The amount of keypecking activity that took place during the CS+ periods of a single session was quantified using two measures. The first measure was the average number of pecks made during each 5 s CS+ presentation (PECKS/CS+). The second measure was the percentage of all CS+ trials of a session on which at least one peck was recorded during the CS+ presentation (% CS+ TWP). For the purpose of demonstrating that chicks acquired the discrimination between the CS+ and CS-, similar measurements were made for the CS- trials. A variety of ANOVAs was run on the PECKS/CS and % CS TWP data of all subjects from the training and test phases. These analyses will be described below in the Results section.

Observations

Direct visual observation and classification of the behavior of 16 chicks during the autoshaping session was carried out during the second session on Days 2 and 13 of the training phase. Behavior was observed at three points during the course of each trial of these two sessions. These points were the 5 s CS, the 5 s US periods, and the 5 s period immediately preceding the onset of the CS, i.e., the ITI.

The overall purpose of this procedure was to evaluate what the chicks were doing when they were not keypecking. The learned thermoregulatory response within the proposed response hierarchy, again, is the CS+ pecking response. According to the hypothesis tested by this experiment, chick's whose thermoregulatory abilites are relatively mature will engage in innate postural responses to cold rather than peck the CS+. Therefore, a more specific objective of the observation procedure was to determine whether or not the chicks were performing the innate postural responses of sitting or squatting during CS+ presentations on which no response occurred. Despite the chick's capacity to discriminate between the CS+ and CS-, the chick will occasionally peck the key during CS- presentations and even during the ITI. The purpose of this procedure, again, was to identify what chicks did when they were not pecking the key. Therefore, all observations (CS+, CS-, and ITI) during which a keypeck was observed were discarded.

On accepted observation periods, the behavior observed was classified in accordance with a five category system, modeled after a behavior observation system developed by Holland (1977) for classifying the behavior of rats in an operant conditioning setting. The five categories of behavior were (1) sitting,

(2) standing or squatting, (3) moving about the chamber, (4) pecking or rubbing the wall immediately surrounding the response key, and (5) stretching or wing spreading during the operation of the heat lamp.

Often more than one behavior occurred during a single accepted observation period. In this case the behavior that occurred for most of the period was the one recorded for this observation. This behavior category system is still under development. There have not yet been any assessments of the inter-observer reliability of this system.

Results

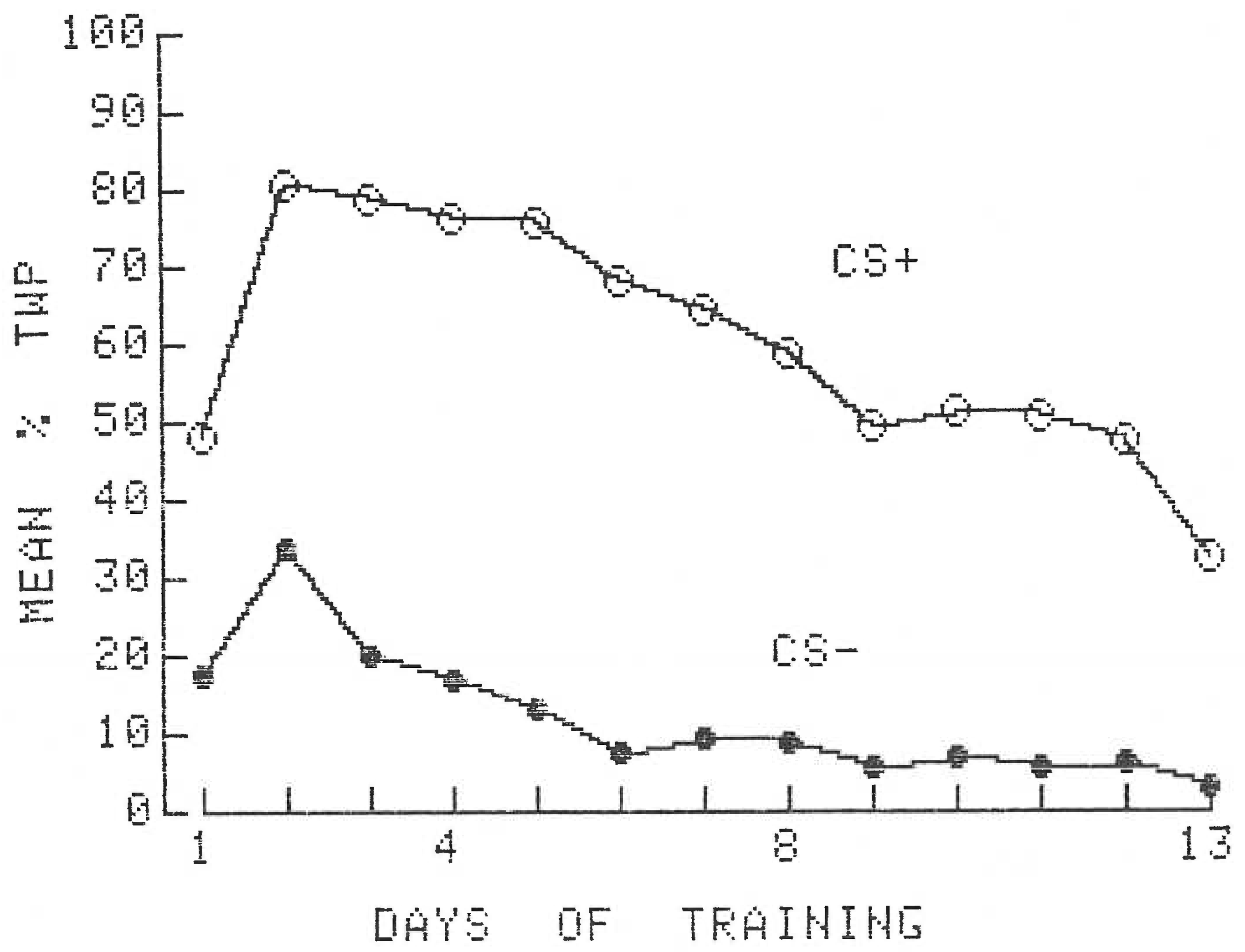
Acquisition

The %TWP and PECKS/CS scores of all subjects were averaged together across the two sessions of each day of training to yield overall group mean %TWP and mean PECKS/CS values for each day of training. Figure 1 shows the mean % CS+ TWP and mean % CS- TWP values over the 13 days of the training phase. On Day 1 subjects responded on a greater percentage of CS+ than CS- presentations. Responses to both stimuli, as well as the difference between pecking to the CS+ and CS- continued to increase up to Day 2. On Day 3 the percentage of CS- trials with a response began to drop and continued to drop until Day 6. Responding to CS- trials remained at this low level for the remainder of the training phase. After reaching a peak level on Day 2, the percentage of CS+ trials with a response remained at equally high levels through to Day 5. On Day 6 the CS+ response level began to drop and continued to drop on the remaining days of the training phase. Response levels to both the CS+ and CS- were at their lowest on the last day of the training phase, Day 13.

On Day 1 subjects responded on an average of 47% of CS+ and 17% of CS- trials. These levels increased to 80% of CS+ and 34% of CS- trials on Day 2. Responding to the CS- peaked on Day 2 and then dropped to 7% of CS- trials by Day 6. On Days 6 to 13 subjects responded on an average of 6% of CS- trials. The lowest level of CS- responding occurred on Day 13 when this level dropped to 3%. After peaking on Day 2, the level of CS+ responding dropped regularly on the remaining days of this phase. The lowest level of CS+ responding was observed on Day 13 when these subjects responded on 32% of CS+ trials.

An overall two-way ANOVA was conducted on the % TWP data from all 26

Figure 1. The overall mean %TWP values for the CS+ and CS- over the 13 days of the training Phase.



MEAN % TWP
100
90
80
70
60
50
40
30
20
10
0
CS+
CS-
1
4
8
13
DAYS OF TRAINING

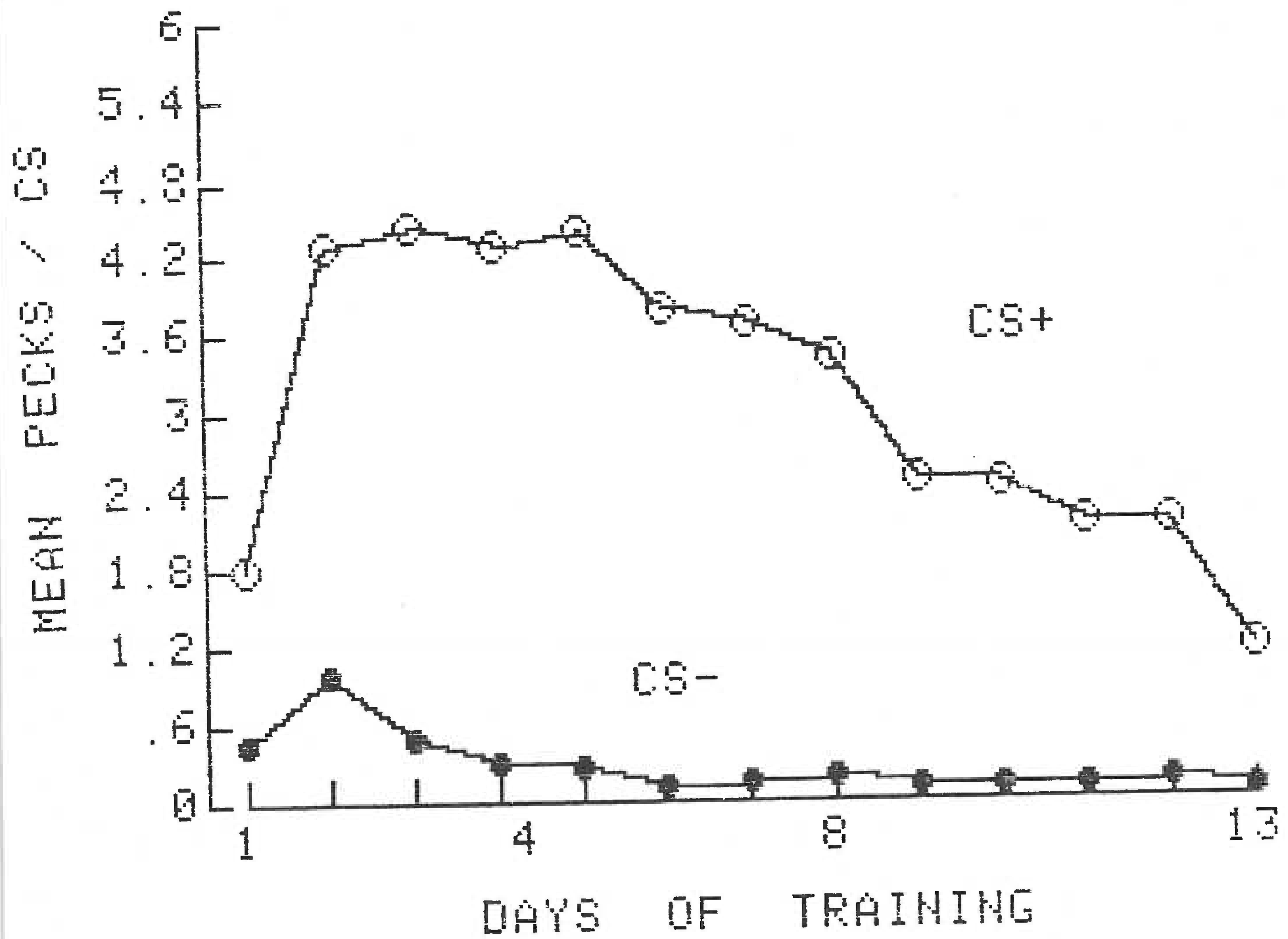
subjects over the 13 days of the training phase. The two factors were the within-group factors type of stimulus, i.e., CS+ or CS-, and day-of-training. This analysis showed that there was a significant effect of both type of stimulus [F (1,25) = 212.79, $p < .001$] and day-of-training factors [F (12,300) = 25.11, $p < .001$]. The interaction of these factors was also significant [F (12,300) = 10.13, $p < .001$].

A one-way within-group ANOVA in which the single factor was type of stimulus was conducted on the % TWP data of all subjects from Day 1. This analysis showed that on Day 1, subjects responded on a significantly greater percentage of CS+ than CS- presentations [F (1,25) = 86.93, $p < .001$]. A similar ANOVA run on the % TWP data from Day 2 showed that the difference between the CS+ and CS- continued to be significant [F (1,25) = 184.20, $p < .001$] on this day also.

The drop in responding to the CS- from Day 2 to Day 6 was evaluated with a one-way within-group ANOVA that compared the % CS- TWP data from Days 2 and 6. This analysis showed that subjects responded on a significantly smaller percentage of CS- presentations on Day 6 than on Day 2 [F (1,25) = 70.36, $p < .001$]. The drop in responding to the CS+ from Day 2 to Day 13 was evaluated with a similar ANOVA of the % CS+ TWP data from Days 2 and 13. This analysis showed that subjects responded on significantly fewer [F (1,25) = 82.14, $p < .001$] CS+ presentations on Day 13 than on Day 2. Analysis of the Day 13 % TWP data of the entire group showed that subjects still responded on a significantly larger number of CS+ than CS- presentations [F (1,25) = 49.64, $p < .001$] at the end of the training phase.

Figure 2 shows the mean PECKS/CS+ and mean PECKS/CS- values over the 13 days of the training phase. On Day 1 subjects pecked more often during CS+ than during CS- presentations. Both the number of pecks to both stimuli and the difference between the stimuli continued to increase on Day 2. On Day 3 the

Figure 2. The overall mean PECKS/CS values for the CS+ and CS- over the 13 days of the training phase.


MEAN PECKS / CS
6
5.4
4.8
4.2
3.6
3
2.4
1.8
1.2
.6
0
CS+
CS-
1
4
8
13
DAYS OF TRAINING

mean number of pecks per CS+ continued to rise slightly, but the mean number of pecks per CS- dropped substantially and continued to drop until Day 6. Responding to the CS- remained at this low level for the remainder of the training phase. From Day 2 to Day 5 the mean number of pecks per CS+ was at or near its peak level. On Day 6 the mean PECKS/CS+ value began to drop and continued to decrease over the remainder of the training phase. The mean PECKS/CS+ and mean PECKS/CS- measures were both at their lowest levels of the training phase on Day 13.

On Day 1 the subjects made an average of 1.8 PECKS/CS+ and 0.4 PECKS/CS-. These rates rose on Day 2 to 4.3 PECKS/CS+ and 1.0 PECKS/CS-. The rate of CS- responding peaked on Day 2 and then fell to 0.1 PECKS/CS- on Day 6. On Days 6 to 13 subjects made an average of 0.1 PECKS/CS-. The subjects made an average of 4.3 PECKS/CS+ on Days 2 to 5 with the peak level of 4.4 PECKS/CS+ occurring on Day 5. After peaking the rate of CS+ pecking dropped regularly over the remaining days of this phase. The lowest rate of CS+ pecking was observed on Day 13 when subjects made 1.2 PECKS/CS+

An overall two-way ANOVA was run on the mean PECKS/CS data of all subjects from all 13 days of the training phases. The two factors were the within-group factors type of stimulus and day-of-training. This analysis showed a significant effect of both type of stimulus [F (1,25) = 58.78, p < .001] and day-of-training factors [F (12,300) = 12.91, p < .001]. The interaction between the factors was also significant [F (12,300) = 11.42, p < .001]. A one-way within-group ANOVA in which the single factor was type of stimulus was run on the PECKS/CS data from all subjects on Day 1. This ANOVA showed that on Day 1, subjects pecked significantly more frequently during CS+ than during CS- presentations [F (1,25) = 50.95, p < .001]. A similar ANOVA run on the PECKS/CS data from Day 2 showed that subjects continued to peck significantly more often during CS+ than CS- presentations [F (1,25) = 86.71, p < .001] on

this day as well.

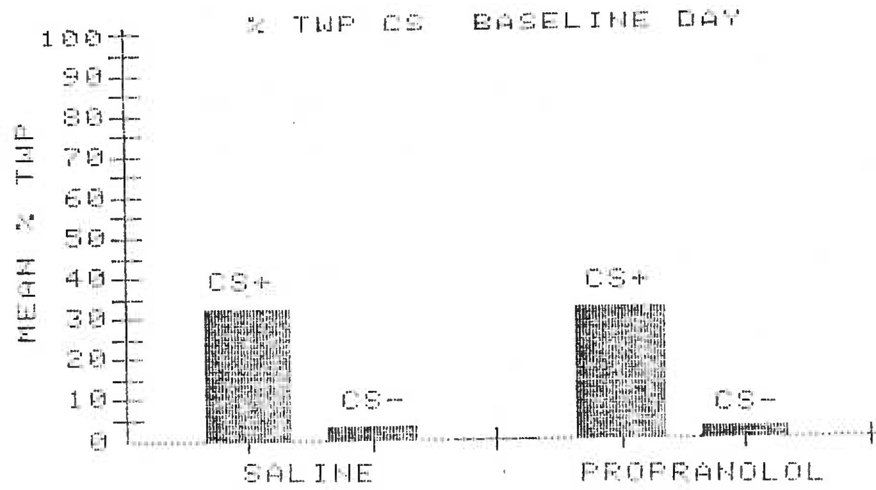
The decrease in CS- pecking from Day 2 to Day 6 was evaluated with a one-way ANOVA comparing the PECKS/CS- data of all subjects from Days 2 and 6. This analysis showed that subjects pecked significantly less frequently [F (1,25) = 26.85, p < .001] during CS- presentations on Day 6 than Day 2. The drop in CS+ pecking from Day 5 to Day 13 was examined using a one-way ANOVA that compared PECKS/CS+ data from all subjects on Days 5 and 13. This analysis showed that subjects pecked significantly less often [F (1,25) = 46.60, p < .001] during CS+ presentations on Day 13 than on Day 5. An analysis of the PECKS/CS data of all subjects from Day 13 showed that subjects still pecked significantly more frequently during CS+ than during CS- presentations [F (1,25) = 26.77, p < .001] at the end of the training phase.

Test Phase

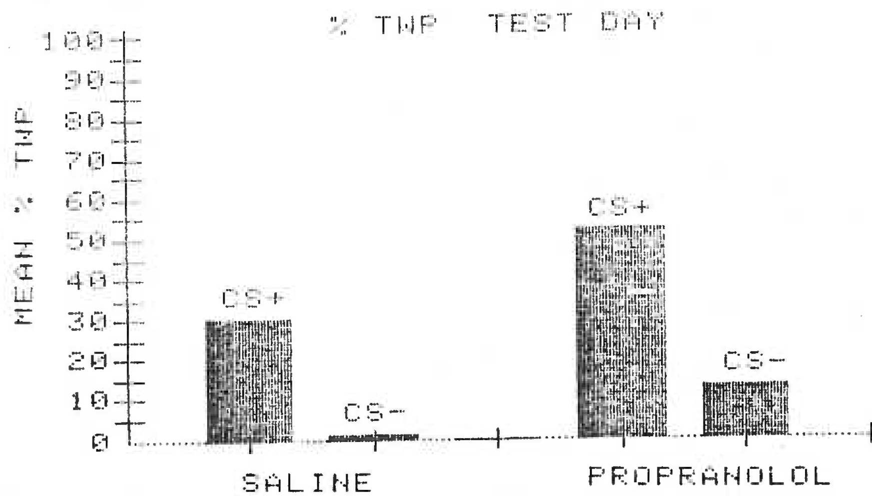
Figure 3A shows the mean % TWP values on Day 13, the last day of the training phase, for the propranolol and saline groups. This bar graph shows that at the end of the training phase both groups responded on nearly the same percentage of CS+ presentations. They also responded on equivalent percentages of CS- presentations. Figure 3B shows the mean % TWP values for these groups on the Test day after these subjects had been injected with either propranolol or saline. On the Test day the propranolol group responded on a much larger percentage of both CS+ and CS- presentations than did the saline group. Together the graphs show that the response levels of the saline group on Day 13 and the Test day were equivalent. The response levels of the propranolol group, however, were much higher on the Test day than on Day 13. An overall three-way ANOVA was carried out on the % TWP data of both groups from Day 13 and the Test day. One of factor was the between-group factor type of treatment, i.e., propranolol vs saline. The remaining factors were the within-group factors type

Figure 3. The mean %TWP values for the CS+ and CS- of the saline and propranolol groups on (A) Day 13, the last day of the training phase, and (B) the Test day.

(A)



(B)



of stimulus and day of testing. This analysis showed a significant effect of both the type of stimulus [F (1,24) = 58.82, p < .001] and day of study factors [F (1,24) = 8.38, p < .01]. The type of treatment factor was not found to be significant [F (1,24) = 2.11]. However, the interaction of the type of treatment and day of testing factors was significant [F (1,24) = 4.50, p < .01]. All other possible interactions were found to be nonsignificant by this analysis.

On Day 13 the saline subjects responded on an average of 32% of all CS+ presentations and only 3% of all CS- presentations. The propranolol subjects responded on an average of 32% of CS+ presentations and 2% of CS- presentations on Day 13. The % TWP data from both groups on Day 13 was evaluated with a two-way ANOVA in which the factors were type of treatment and type of stimulus. This analysis showed that both the saline [F (1,13) = 36.55, p < .001] and propranolol subjects [F (1,11) = 16.47, p < .01] responded on a significantly greater percentage of CS+ than CS- presentations on Day 13. The analysis also showed that there was no significant difference in the responding levels of the two groups [F (1,24) = 0.01] on Day 13.

On the Test day saline subjects responded on an average of only 30% of all CS+ presentations, while the propranolol subjects responded on 52% of these presentations. Also, on the Test day the saline subjects responded on an average of only 1% of all CS- presentations while the propranolol subjects responded on 13% of these presentations. An analysis of this data showed that on the Test day all the subjects continued to respond on a significantly greater [F (1,24) = 45.73, p < .001] percentage of CS+ than CS- presentations. However, the propranolol subjects responded on a significantly larger [F (1,24) = 5.96, p < .05] percentage of all stimulus presentations than did saline subjects.

Two-factor ANOVAs were carried out separately on the % TWP data from the

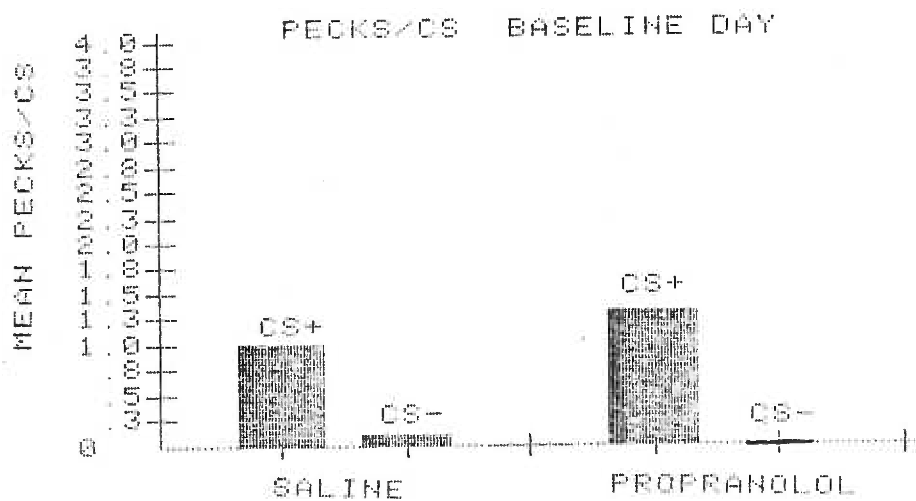
individual groups from Day 13 and the Test day. The factors were the within-group factors of type of stimulus and day of testing. Analysis of the % TWP data of the saline group showed that there was no significant difference [F (1,13) = 0.47] between this group's overall response levels on Day 13 and the Test day. A similar analysis of the % TWP data of the propranolol group showed, however, that these subjects responded on a significantly larger [F (1,11) = 20.02, p < .001] percentage of all stimulus presentations on the Test day than they did on Day 13.

The mean PECKS/CS values of the propranolol and saline groups on Day 13 are plotted in Figure 4A. On Day 13 both groups pecked nearly the same number of times during each CS+ presentation. Also, both groups pecked nearly the same number of times during CS- presentations on Day 13. Figure 4B shows the PECKS/CS values of these groups on the Test day. This plot shows that the propranolol group pecked much more often during CS+ presentations than the saline group did. However, both groups pecked nearly the same number of times on CS- presentations on the Test day. Together the graphs show that the saline group's overall pecking levels are nearly the same on both Day 13 and the Test day. The propranolol group also pecked nearly the same number of times during CS- presentations on both days. However, the propranolol subjects pecked more times during CS+ presentations on the Test day than on Day 13.

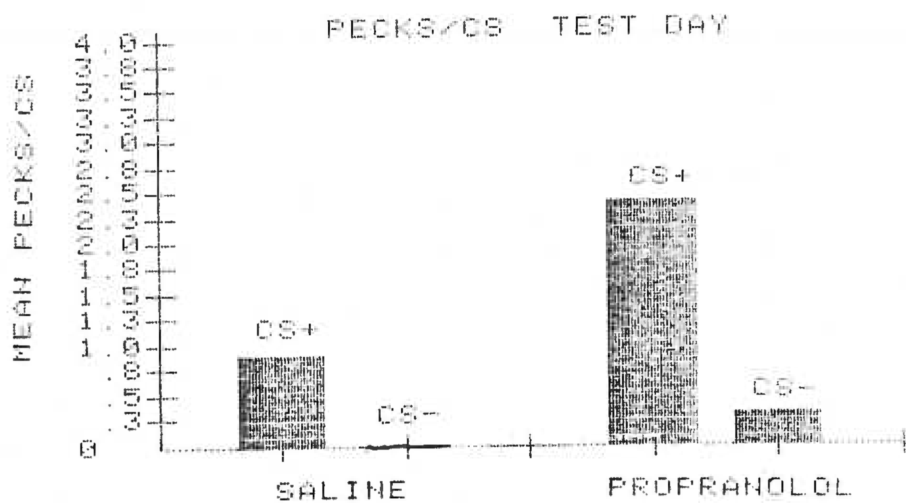
An overall three-way ANOVA was run on the PECKS/CS data from both groups on Day 13 and the Test day. One factor was the between-group factor of type of treatment. The other factors were the within-group factors type of stimulus and day of testing. This analysis showed a significant effect of both the type of stimulus [F (1,24) = 26.10, p < .001] and day of testing factors [F (1,24) = 5.09, p < .05]. The interaction of these factors was also significant [F (1,24) = 4.43, p < .05]. The type of treatment factor was found to be nonsignificant [F (1,24) = 3.07]. However, the interaction of the type of

Figure 4. The mean PECKS/CS values for the CS+ and CS- of the saline and propranolol groups on (A) Day 13 of the training phase and (B) the Test day.

(A)



(B)



treatment and day of study factors was significant [F (1,24) = 10.13, p < .01] as was the interaction between all 3 factors [F (1,24) = 9.58, p < .01]. All other possible interactions were found to be nonsignificant.

On Day 13 propranolol subjects made an average of 1.3 pecks during CS+ presentations and an average of 0 pecks during each CS-, while the saline subjects made an average of 1.0 peck during each CS+ and 0.1 pecks during each CS-. On the Test day while the saline subjects made an average of only 0.9 pecks during each CS+, the propranolol subjects made an average of 2.4 pecks during these stimulus presentations. There was little difference, however, between the group's mean PECKS/CS- values. On the Test day the saline subjects made an average of 0 pecks during each CS- and the propranolol subjects made an average of 0.3 pecks during these presentations.

The PECKS/CS data from both groups was compared on individual days (13 and Test) using one-way within-group ANOVAs in which the factor was type of stimulus. Analysis of only the Day 13 data from both groups showed that all subjects pecked significantly more [F (1,24) = 26.44, p < .001] during CS+ than during CS- presentations. This analysis also showed that on Day 13 there was no significant difference [F (1,24) = 0.30] between the overall mean pecking levels of the propranolol and saline groups. A similar analysis of the Test day data showed that both groups continued to peck significantly more often [F (1,24) = 23.03, p < .001] during CS+ than during CS- presentations. However, the analysis shows that on the Test day the overall pecking level of the propranolol group was significantly greater [F (1,24) = 5.56, p < .05] than that of the saline group.

Comparisons between each group's PECKS/CS data from Day 13 and the Test day were made using two-way ANOVAs in which the factors were the within-group factors type of stimulus and day of testing. This analysis showed that the saline group's overall pecking levels on Day 13 and Test day were not

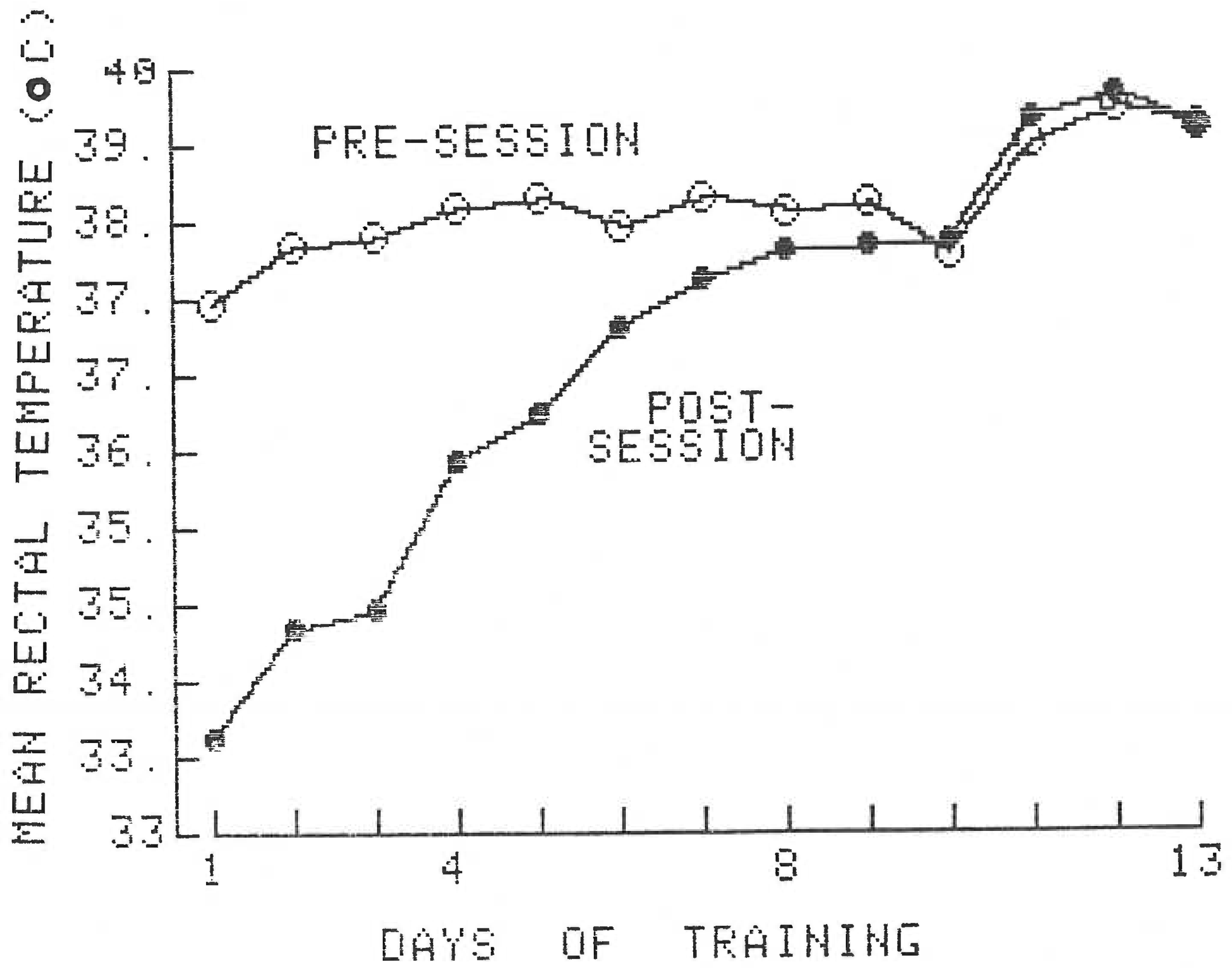
significantly different [F (1,13) = 1.08]. The overall pecking level of the propranolol group, on the other hand, was significantly higher [F (1,11) = 8.22, $p < .05$] on the Test day than it was on Day 13.

Temperature Data

Training Phase. Figure 5 shows the overall mean pre and post-sessions rectal temperatures of all subjects over the 13 days of the training phase. On the first day of the training phase subjects were, on the average, 2 days old. The mean pre-session temperature was 37.8° C on Day 1 and rose slightly to 38.3° on Day 2. From Day 2 to Day 10 pre-session rectal temperature changed very little and was an average of 38.6° C over this period. On Day 11 mean pre-session rectal temperature rose to 39.3° C and remained above 39.0° for the remainder of the study. Pre-session rectal temperature on the last 3 days of the training phase was an average of 39.6° C. A one-way within-group ANOVA in which the factor was day of training was run on the pre-session temperature data of all subjects from all 13 days of the training phase. This analysis showed that day of training was significant [F (12,300) = 28.43, $p < .001$].

The plot of post-session rectal temperature in Figure 5 shows the progressive reduction in the magnitude of the temperature drop caused by the cold exposure over the course of the experiment. On Day 1 the subjects's temperatures dropped an average of 4.0° C. This drop reduced to 3.5° C on Day 2. This progressive decrease in the magnitude of the temperature drop continued on the following days. Drops of less than 1° C were observed on Days 6 and 7. On Days 8 and 9 the temperature drop was an average of 0.4° C. Post-session temperature averaged 0.2° C higher than the mean pre-session temperature on Days 10, 11, and 12. On the last day of the training phase the post-session temperature of 39.4° C was equal to the pre-session temperature. On Day 13 the

Figure 5. The overall mean pre and post-session rectal temperatures over the 13 days of the training phase.


MEAN RECTAL TEMPERATURE (oC)
40
39.
38.
37.
37.
36.
35.
35.
34.
33.
33
PRE-SESSION
POST-
SESSION
1
4
8
13
DAYS OF TRAINING

cold exposure, on the average, produced no change in rectal temperature. A one-way within-group ANOVA in which the factor was day of training was run on the post-session temperature data of all the subjects over all 13 days of the training phase. This analysis showed that day of training was significant [F (12,300) = 148.42, $p < .001$]. An analysis of the temperature changes (pre-session temperature - post-session temperature) of all subjects from all 13 days of the training phase showed that the decrease in the magnitude of the mean temperature change over the course of the training phase was significant [F (12,300) = 129.98, $p < .001$].

The difference between this group's pre and post-session temperatures was evaluated with a t test only on those days when post-session temperature was equal to or lower than pre-session temperature. These tests showed that the difference between this group's pre and post-session temperatures was significant on the first 9 days of the Acquisition phase.

Test Phase. Table 1 lists the mean pre-session rectal tempertures of the saline and propranolol groups on the last day of the Acquisition phase and on the Test day. The pre-session temperatures of both groups were equivalent on each day. The pre-session temperatures of both groups were slightly higher on the Test day than on Day 13. An overall two-way ANOVA was run on the pre-session rectal temperature data of the propranolol and saline groups from Day 13 and the Test day. One factor was the between-group factor type of treatment and the other was the within-group factor day of testing. This analysis showed that day of testing was significant [F (1,24) = 8.37, $p < .01$]. This reflects the fact that the pre-session temperatures of both groups were slightly higher on Day 13 than on the Test day. The analysis also showed that the type of treatment [F (1,24) = 0.02] and the interaction of the treatment and day of testing factors [F (1,24) = 0.14] were nonsignificant.

Table 1. The mean pre-session rectal temperatures of the saline and propranolol groups on Day 13, the last day of the training phase, and on the Test day.

Table 1

Test Phase Pre-Session Temperatures

(o C)

Group	Day	
	Baseline	Test
Propranolol	39.5	39.2
Saline	39.5	39.3

A one-way between-group ANOVA in which the factor was type of treatment found that the difference between the two group's pre-session temperatures on the Test day was not significant [F (1,24) = 0.09]. The difference between the Day 13 and Test day pre-session temperatures of each group was evaluated with a one-way within-group ANOVA in which the factor was day of testing. This analysis showed that the difference between the saline group's Day 13 and Test day pre-session temperatures was not significant [F (1,13) = 2.63]. However, the analysis showed that the propranolol group's pre-session temperature was significantly greater [F (1,11) = 10.31, p < .01] on Day 13 than on the Test day.

Figure 6 shows the mean changes in the temperatures of the saline and propranolol groups caused by the 12 min exposure to 9° C on Day 13 and the Test day. On Day 13 the propranolol group's temperature increased slightly while the saline group's temperature did not change. On the Test day the cold exposure caused the saline group's temperature to increase slightly. The propranolol group's temperature, however, dropped substantially on the Test day.

An overall two-way ANOVA was run on the temperature changes of the propranolol and saline groups over Day 13 and the Test day. One factor was the between-group factor type of treatment and the second was the within-group factor day of testing. This analysis showed a significant effect of both the type of treatment [F (1,24) = 14.41, p < .001] and day of testing factors [F (1,24) = 13.42, p < .01]. The interaction between these factors was significant [F (1,24) = 17.06, p < .001]. The difference between the two group's mean temperature changes on individual days was examined with a one-way between-group ANOVA in which the factor was type of treatment. This analysis showed that on Day 13, the difference between the temperature changes of two groups was not significant [F (1,24) = 0.50]. On the Test day, however, the size of the drop in the propranolol subject's rectal temperature was

Figure 6. The mean change in the rectal temperatures of the saline (S) and propranolol (P) groups after a 12 min exposure to 9° C on Day 13, the last day of the training phase (Baseline), and on the Test day.

significantly greater [F (1,24) = 23.56, p < .001] than the size of the increase in the saline subjects.

The difference between the Day 13 and Test day temperature changes of each group were evaluated with a one-way within-group ANOVA in which the factor was day of testing. Analysis of the difference between the saline group's temperature change on Day 13 and the Test day showed that it was not significant [F (1,13) = 0.29]. This analysis did show, however, that the size of the propranolol group's temperature change on the Test day was significantly greater [F (1,11) = 16.56, p < .01] than it was on Day 13.

The Pearson product moment correlation coefficient (r) between a group's temperature drop and both its % CS+ TWP and PECKS/CS+ values were calculated on the baseline day and test day. On the baseline day the correlation between the propranolol group's temperature change and and its % CS+ TWP value was not significant [df = 10, r = -0.27]. This was true for the correlation between the group's temperature drop and PECKS/CS+ values on the baseline day [df = 10, r = -0.26]. The correlation between the saline group's baseline day temperature change and % CS+ TWP values [df = 12, r = +0.37] was not significant. The correlation between this group's baseline day temperature change and PECKS/CS+ values [df = 12, r = +0.37] was also not significant.

The correlation between the propranolol group's test day temperature drop and % CS+ TWP values was not significant [df = 10, r = +0.43] but was stronger than the same correlation on the baseline day. This was true for the correlation between this group's temperature drop and PECKS/CS+ values [df = 10, r = +0.44] as well. The correlation between the saline group's test day temperature change and its % CS+ TWP values was not significant [df = 12, r = -0.03] and was weaker than the same correlation on the baseline day. Again, this was also true for the correlation between this group's test day temperature drop and PECKS/CS+ values [df = 12, r = -0.05].

Observation Data

It should be noted again at this point that the overall purpose of this procedure was to identify what chicks were doing during observations when a pecking response did not occur. Of specific focus was what chicks did on CS+ presentations on which a pecking response did not occur.

Figure 7A shows the results of the ITI period observations on Day 2 (excluding observations on which pecking was observed). Each bar in the graph represents the total percentage of all the Day 2 ITI observations on which a particular nonpecking behavior was recorded. This figure shows that on Day 2 during ITI periods the most prevalent behavior was standing still (2). The results of the Day 13 ITI observations are shown in Figure 7B. Standing still was still the most prevalent behavior during ITI periods on Day 13, the last day of the training phase. Together Figures 7A and 7B show that the frequency of standing still is nearly the same on Days 2 and 13. The frequency of sitting (1) rose while the frequency of wall rubbing (4) decreased over this period. Keypecking was observed on less than 5% of all ITI observations on both Days 2 and 13.

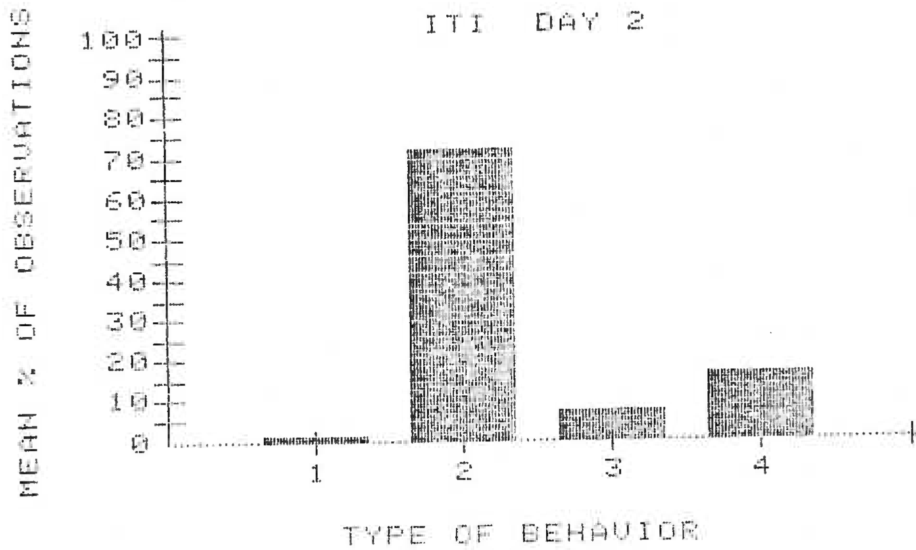
The frequency of each nonpecking behavior on Day 2 was compared to its frequency on Day 13 with a one-way within-group ANOVA in which the factor was day of observation. On Day 2 standing was recorded on 72% of all ITI observations. This value dropped slightly to 68% on Day 13. The difference between the frequency of standing during the ITI on Day 2 and 13 was not significant [F (1,15) = 0.12]. Wall rubbing was recorded on 16% of all ITI observations on Day 2 and on 7% of the Day 13 observations. This frequency difference between Day 2 and Day 13 was not significant [F (1,15) = 4.85]. The frequency of sitting during the ITI rose from 1% on Day 2 to 18% on Day 13. Analysis of this difference found that sitting occurred significantly more often

Figure 7. The mean percentages of the ITI period observations on which each of the 4 different types of nonkeypecking behaviors were recorded on (A) Day 2 and (B) Day 13 of the training phase.

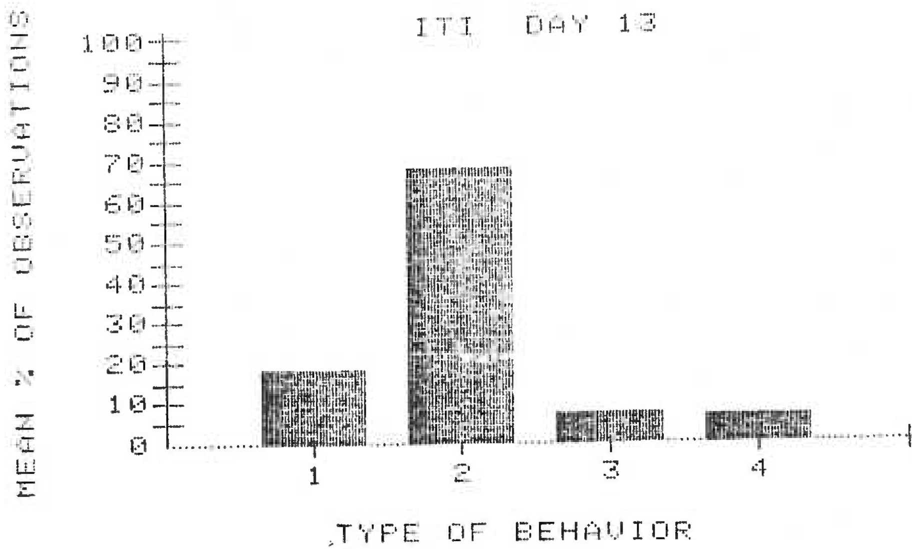
BEHAVIOR TYPE

1 = SITTING
2 = STANDING
3 = MOVING ABOUT
4 = PECKING WALL AROUND RESPONSE KEY

(A)



(B)



[F (1,15) = 6.20, p < .05] during ITI periods on Day 13 than Day 2.

The results of the CS+ period observations on Day 2 are plotted in Figure 8A (excluding observations on which keypecking was observed). Keypecking was, however, the most prevalent behavior during Day 2 CS+ observations. Keypecking was recorded on 94% of these observations on Day 2. Nonpecking behavior was recorded on only 6% of all the Day 2 CS+ observations. Figure 8B shows the results of the Day 13 CS+ period observations. Standing still and keypecking were the most prevalent behaviors on Day 13 CS+ periods. However, keypecking accounted for a total of only 37% of these observations on Day 13. The frequency of both sitting and standing increased from Day 2 to Day 13. Standing still was recorded on 5% of all CS+ period observations on Day 2. This value increased to 34% on Day 13. Analysis of this increase showed that the frequency of standing still was significantly greater [F (1,15) = 26.36, p < .001] on Day 13 than on Day 2. Sitting, a behavior which was never recorded during CS+ period observations on Day 2, was recorded on 15% of these observations on Day 13. Analysis of this increase showed that the frequency of sitting was significantly higher [F (1,15) = 4.78, p < .05] on Day 13 than on Day 2.

Figure 9A shows the results of the CS- period observations on Day 2 (excluding observations on which keypecking occurred). Keypecking occurred on 35% these observations. Standing and wall rubbing were the most prevalent nonpecking behaviors observed on Day 2 CS- observations. The results of the CS- period observations on Day 13 are plotted in Figure 9B. Standing still is the most prevalent nonpecking behavior during Day 13 CS- periods. Keypecking occurred on only 2% of these Day 13 observations. Figures 9A and 9B together show that the frequency of wall rubbing decreased from Day 2 to Day 13. The frequency of sitting and standing still, however, both increased over this period.

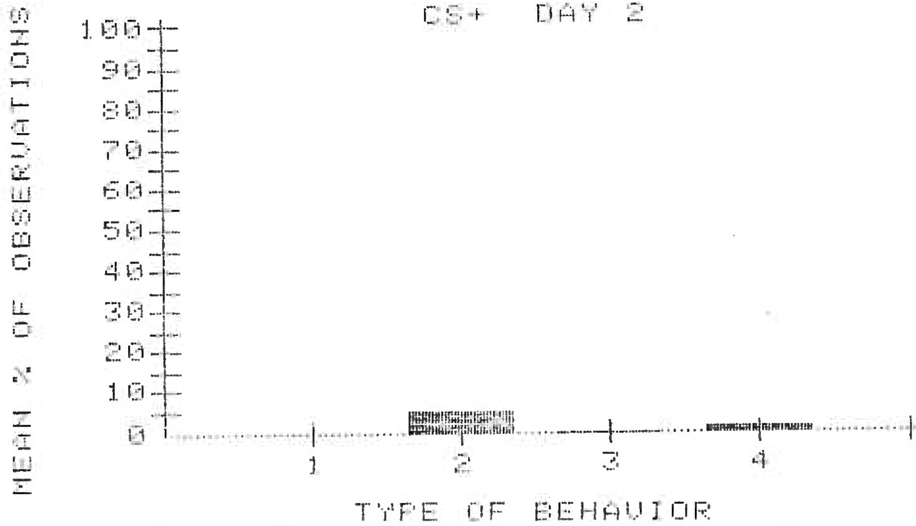
Wall rubbing was recorded on 22% of all CS- observations on Day 2 and on

Figure 8. The mean percentages of the CS+ period observations on which each of the 4 different types of nonkeypecking behaviors were recorded on (A) Day 2 and (B) Day 13 of the training phase.

BEHAVIOR TYPE

1 = SITTING
2 = STANDING
3 = MOVING ABOUT
4 = PECKING WALL AROUND RESPONSE KEY

(A)



(B)

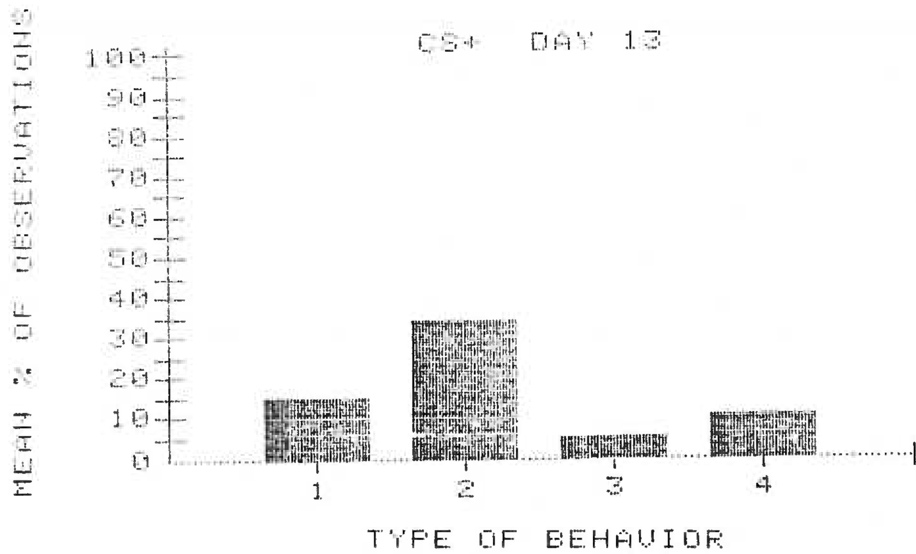
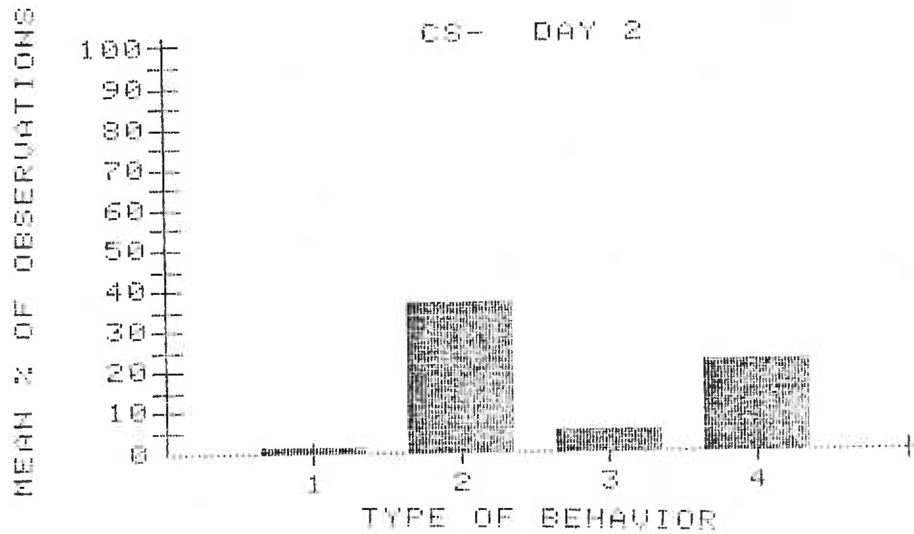


Figure 9. The mean percentages of the CS- period observations on which each of the 4 different types of nonkeypecking behaviors were recorded on (A) Day 2 and (B) Day 13 of the training phase.

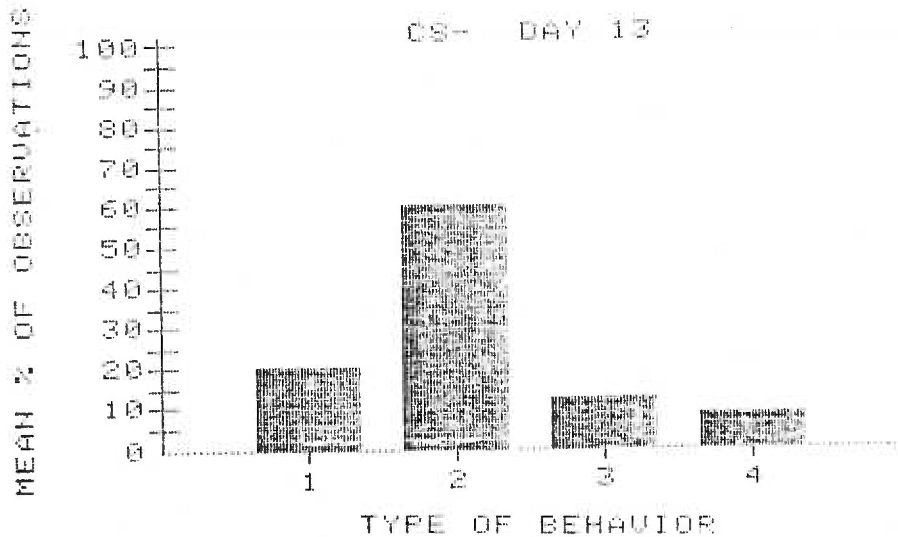
BEHAVIOR TYPE

1 = SITTING
2 = STANDING
3 = MOVING ABOUT
4 = PECKING WALL AROUND RESPONSE KEY

(A)



(B)



8% on Day 13. The frequency of wall rubbing was significantly less [F (1,15) = 15.29, $p < .001$] on Day 13 than on Day 2. Standing still, recorded on 37% of all CS- observations on Day 2, was recorded on 60% of these observations on Day 13. Analysis of this increase showed that the frequency of standing was significantly greater [F (1,15) = 7.20, $p < .05$] on Day 13 than on Day 2. Sitting was observed on 1% of all CS- period observations on Day 2 and 20% on Day 13. The frequency of sitting during the CS- was significantly higher [F (1,15) = 6.32, $p < .05$] on Day 13 than on Day 2.

Discussion

The results of this experiment provide some support for the proposed escape response hierarchy hypothesis as one possible explanation of the relationship between thermoregulatory development and the drop-off effect. According to the hypothesis, as the chick's thermoregulatory abilities improved the innate postural responses to cold would displace and prevent the occurrence of the learned thermoregulatory response of pecking the CS+. There are observational data that support this prediction. On Day 2 keypecking occurred on nearly all the CS+ observations. On Day 13, however, keypecking accounted for only about 40% of all these observations. Standing still and sitting were recorded on most of the CS+ observations on which a keypeck did not occur. The behavior of sitting during the cold is a documented innate heat-conserving postural reaction to cold. Assuming that the behavior of standing is also a postural reaction to cold, these results show that on Day 13 CS+ observations during which keypecking was not recorded, chicks were usually engaged in an innate postural response to cold.

According to the proposed hypothesis, injecting the chick's with propranolol should cause an increase in the occurrence of the learned thermoregulatory response of pecking the CS+. The Test procedure results show that this occurred. The level of autoshaped CS+ pecking increased significantly in the chicks that had been injected with propranolol while the level continued to drop in the saline control group. The Test day rectal temperature data shows that propranolol reduced the chick's ability to maintain a stable core temperature during the cold exposure. While the exposure produced little or no change in the temperatures of saline subjects, it did cause significant drops in propranolol subjects.

Conclusions about the validity of the proposed causal relationship between thermoregulatory development and the drop-off effect are only tentative because of the lack of knowledge of propranolol's general effects on autoshaped responding in chicks. It is entirely possible that propranolol is a general stimulator of keypecking activity. If this were the case, the increased occurrence of CS+ pecking in the propranolol group would be the result of this stimulating effect, and not to the drug's detrimental thermoregulatory effects.

Experiment 2

The purpose of this experiment was to replicate the finding of Experiment 1 and to determine what effects propranolol has on autoshaped keypecking behavior independent of its detrimental effects on thermoregulatory ability. The design of this study represents an effort to conduct a procedure very similar to the heat autoshaping procedure under conditions in which thermoregulatory ability does not affect the level of autoshaped responding. The food-reinforced autoshaping paradigm offers a reasonable setting for such a test. In the heat autoshaping procedure, chicks are motivated to escape the aversive cold of the experimental chamber. One such means of escape is the programmed delivery of heat from an infrared bulb. In the food study, however, food-deprived chicks are motivated to eat in order to satiate their hunger, and programmed deliveries of a food US are made available during training. Beyond this difference in the sources of motivation and form of US, these two paradigms are identical. Unrestrained chicks are exposed to a CS+ that is always followed by reinforcement and to a CS- that is never reinforced. As a result of such exposure, chicks develop the behavior of regularly pecking the CS+, this being the autoshaped keypecking response. In both procedures acquisiton of this discrimination is apparent during the first training session and is usually complete by the second or third session of training. Because of the similarities of the heat and food paradigms it was assumed that the latter would be an ideal setting for determining the general effects of propranolol on autoshaped keypecking levels independent of the drug's thermoregulatory effects.

In Experiment 2, one group of chicks was run in the heat autoshaping procedure while a second group was run in the food procedure. Both groups were exposed to the same number and sequence of CS+ and CS- trials on each day of training. Both groups were conditioned daily until the performance level in the

heat autoshaping group had dropped off. On the following day half the subjects in each group received an injection of propranolol, the other half, the saline control subjects, received an injection of physiological saline. Following the injection, subjects were exposed to another session of the same autoshaping procedure they had been exposed to on the preceding days. At the beginning of the test session subjects in both the Heat and Food groups had been exposed to the same number of days and sessions of autoshaping. Also all subjects had been exposed to equal numbers of reinforced CS+ and nonreinforced CS- trials.

One of the major procedural differences between the heat and food paradigms is in the age at which conditioning usually commences. Chicks are, on the average, nearly 2 days old when heat autoshaping normally starts. In the food procedure, however, conditioning does not begin until the subjects are 6 days old. At hatching chicks still have a substantial supply of food in their internalized yolk sacks. Food deprivation therefore has little effect on chicks until they are around 4 days old. Once they are 4 days old, chicks that have been food deprived for 24 h are trained to eat from the food hopper. After a second day of this training, chicks are ready to begin the autoshaping procedure at 6 days of age. In order for subjects in the Heat and Food groups of this drug evaluation to be the same age, both groups began their respective autoshaping procedures when all chicks were 6 days old. Thus, subjects in the heat procedure began training 4 days later than normal.

Another major procedural difference is that subjects in the food study are food deprived while subjects in the heat study have free access to food. In order to eliminate this difference the heat study subjects were also food deprived in the proposed drug evaluation. Both groups had free access to food for a total of 3.5 hours each day. Another reason exists for food depriving the heat subjects. If propranolol is indeed a general stimulator of autoshaped keypecking behavior, then the question of how hunger might alter this effect is

raised. Hunger could possibly eliminate the behavior stimulating effects of the drug. If this were so, the stimulating effects of propranolol would not be observed in the autoshaped keypecking behavior of food-deprived chicks. It has already been demonstrated that propranolol causes an increase in the level of heat autoshaped keypecking. If this is due to propranolol's general stimulating effect, and hunger negates this effect, then there should be no increase in the level of heat autoshaped responding in food-deprived subjects injected with propranolol. Heat autoshaping subjects were food-deprived to control for the possibility that hunger negates the stimulating influence of propranolol.

Rectal temperature measurements are an important part of the heat procedure. Body temperature, however, is not a relavent variable in the food procedure. In order to insure that subjects in both the heat and food procedures were handled equally, food subjects also had their pre and post-session rectal temperatures measured in this study. One final difference between the two procedures is that in the food procedure subjects are usually housed together as a group in a brooder. In the heat study, however, subjects are isolated from each other, being housed individually in small cages. In order to eliminate this difference in the drug evaluation procedure, all subjects, in both the Heat and Food groups were housed individually in separate cages.

Methods

Subjects

At the beginning of the study sixteen chicks were run in both the heat and food procedures for a total of 32 subjects in this experiment. Two subjects in the food procedure died during the course of the study. The Acquisition phase

results from the food procedure were based on the data of the 14 surviving subjects. Two food procedure subjects failed to perform the autoshaped keypecking response regularly during the Acquisition phase. These two subjects were not run during the Test phase, therefore a total of 12 subjects were run in the food procedure during the Test phase. As in Experiment 1, these chicks were hatched in the lab from fertile eggs (White Leghorn) obtained from the Department of Poultry Science at Oregon State University. The incubation and hatching procedures were similar to those outlined in Experiment 1. No hyperoxic incubation procedure was carried out in this experiment so only one incubator with 70 eggs was set. Chicks were allowed free access to food until the food deprivation procedure began the day the subjects were 3 days old. From this point subjects were limited to only 3.5 h of free access to food on each of the remaining days of the study. The daily food access period began after the end of the second daily training session. Chicks had free access to water in their cages throughout the study.

Apparatus

Heat Autoshaping. The heat autoshaping procedure was run in the same apparatus described in Experiment 1.

Food Autoshaping. A standard key-pecking panel for pigeons (Grason Stadler Pigeon Station, E1184JA-1) was mounted inside a Grason Stadler Animal Chest (Model E3125 AA-3). With the pigeon station in place the inside dimensions of the experimental chamber were 33.0 X 33.0 X 36.0 cm. This entire arrangement was housed within a walk-in sound-attenuating chamber manufactured by the Industrial Acoustics Company, Inc. (inner dimensions: .91 X 1.98 X 1.02 m). Ventilation was provided by fans located in the animal chest and

sound-attenuating chamber.

The floor of the experimental chamber was composed of a galvanized steel tray lined with paper towels and covered with wire mesh. A food hopper delivered chicken feed (Carnation Chick Starter mix) through an apperature (5.1 X 4.0 cm) located in the center of the key pecking panel 6.0 cm above the floor. A frosted BRS/LVE response key 2.5 cm in diameter was situated 10.0 cm above the floor, 3.0 cm to the left of the food hopper apperature. An in-line mini-projector (Industrial Electronics Engineers Inc., Model 00010-01-XXXX-44) was mounted behind the response key. The projector was used to project red and green circles onto the key. The red light projection was created with a Kodak Filter No. 29, the green projection with Filter No. 60.

A houselight was located at the top and in the center of the chamber wall opposite the key-pecking panel. The houselight was a miniature light bulb (#1820) wired to a 28 V DC power supply. The houselight was illuminated throughout the course of the training sessions. Another miniature light bulb was located in the food hopper apperature. When the food hopper was raised to provide the chick with a food US, this light was illuminated. When the hopper was lowered out of the chick's reach, this light was turned off. Outside light was eliminated by covering the window of the sound-attenuating chamber on the inside with layers of construction paper and aluminum foil. An Apple II minicomputer and electromechanical devices controlled stimulus events and recorded keypecking.

Procedure

Heat Autoshaping. The heat autoshaping procedure was nearly identical to the procedure described in Experiment 1. The exception being that training

began when chicks were an average of 6 days old rather than 2. Each session was composed of a sequence of 12 heat-reinforced CS+ and 12 nonreinforced CS- trials arranged in orders described by Fellows (1967), with a mean ITI of 30 s between trials. Each 24 trial session lasted around 14 min. Subjects were exposed to two sessions of autoshaping each day of the training phase. As in Experiment 1, the baseline rectal temperatures and weights of each subject were measured shortly before the beginning of each day's first session. Post-session rectal temperatures were recorded shortly after the end of this session. Weights and temperatures were not measured during the second session of each day. Food deprivation procedures for these subjects were the same as those outlined below for the food autoshaping subjects.

Hopper Training. Before beginning the food autoshaping procedure subjects were first taught to eat food from out of the food hopper in a procedure known as hopper training. Early on the day when the chick's had a mean age of 3 days, food was removed from their cages. Up until this point subjects had continuous free access to food. Early on the following day hopper training commenced. Subjects were brought from their home cages to the lab where their weights were measured. After all the subjects were weighed they were placed into separate experimental chambers and the hopper training session was started. Hopper training on the first day consisted of 30 consecutive 20 s food presentations separated by a mean ITI of 40 s. When the hoppers were not operating the hopper was dropped out of the chick's reach and the space containing the hopper was dark. When the hoppers were operating they were raised so that chicks had easy access to food within them. Also, the space containing the hopper was illuminated during this period.

Subjects were observed repeatedly throughout the session to see whether they had eaten from the raised food hoppers. At the end of the session each

chick's crop or gullet was examined to see whether any food had been eaten. After the session the chick's were returned to their home cages where they had free access to food for the following 3.5 h. After this period of free access, food was removed and was not available again until after training had been completed on the following day. On the second and last day of hopper training the hopper presentations were shortened from 20 to only 5 s in duration. If a subject failed to eat during either of the sessions it was exposed to an extra session of magazine training on that day.

Food Autoshaping. When chicks were an average of 6 days old, they commenced training in the food autoshaping procedure. Subjects were exposed to 2 sessions of autoshaping each day of the training phase. Shortly before the beginning of the first session each subject's pre-session rectal temperature and weight was measured. The subjects were then placed into their separate chambers and the session started. This was marked inside the chambers by switching on the houselights. As in the heat autoshaping procedure, the food autoshaping session was composed of a sequence of 12 reinforced CS+ and 12 nonreinforced CS- trials, with a mean ITI of 30 s between trials. These 24 trial sessions had a total duration of around 14 min. The CS+ was a 5 s illumination of the response key with a red light. The offset of the CS+ was immediately followed by the onset of the food US, a 5 s raising of the food hopper. The CS- was a 5 s illumination of the response key with a green light.

The end of the last trial of each session was marked by switching off the houselights. As in the heat procedure, subjects were removed from their chambers and had their post-session rectal temperatures measured at 90 s intervals.

Temperatures and weights were not measured on the second food autoshaping session of each day. After the end of the second session of each day, subjects

were returned to their home cages where they had free access to food for the following 3.5 h. Subjects in the heat procedure were also exposed to this schedule of food deprivation. Training in both groups was carried out until the day on which the percentage of CS+ trials on which the heat autoshaping group responded had dropped to a level that was significantly lower than its peak level and also lower than the level on the first day of training. Autoshaped keypecking behavior in the food subjects was quantified using the %TWP and PECKS/CS measures outlined in Experiment 1.

Test Phase. The propranolol test was conducted in both groups the day after the end of the training phase. At the end of the last day of training, subjects in both procedures were divided into propranolol and saline groups. Chicks were distributed into two divisions on the basis of the total number of pecks they made on the last day of training. Subjects were distributed such that the two groups had equivalent numbers of high, medium and low activity subjects. One group was randomly designated to be the propranolol group, and the other group the saline control group. The propranolol and saline groups in the heat procedure were processed in accordance with the test procedure outlined in Experiment 1. The food subjects were processed in nearly the same manner.

The effects of three different doses (10, 15, and 20 mg/kg) of a 1 mg/ml solution of propranolol hydrochloride on autoshaped responding were assessed in the Test phase. It should be noted that initially only one dosage of propranolol (10 mg/kg) was to be evaluated. The results from this dosage, however, led to the decision to evaluate the two larger doses in order to collect some preliminary data on a possible dose-response relationship between the size of the dose of propranolol and its effects on CS+ pecking activity. Dose and order of receiving each dose are confounded in this preliminary investigation, however, this Test procedure was not designed to evaluate a

dose-response relationship.

Each subject's weight was measured shortly before the beginning of each test procedure. The subject's pre-session weight was used to calculate the size of the injection it received on that particular test day. On Day 1 of the Test phase, when subjects had a mean age of 21 days, the propranolol subjects in both procedures received a 10 mg/kg injection of propranolol, the dose used in Experiment 1. The saline subjects in both procedures were given 1 cc/100 g of body weight injections of physiological saline.

On the next day, all subjects accidently had free access to food for nearly 24 h. No drug test was run on this day. Both the Heat and Food autoshaping groups were run through one session of their respective types of autoshaping. This day was not considered in the results.

On the following day, after all subjects were back on the correct food deprivation schedule, the second dose of propranolol was tested. Propranolol subjects in both procedures were given a 20 mg/kg injection of propranolol and all saline subjects received 2 cc/100 g of body weight injections of saline. The third dose of propranolol was tested on the following day. On Day 3 all propranolol subjects received a 15 mg/kg injection of propranolol and all saline subjects received 1.5 cc/100 g of body weight injections of saline on Day 3.

Thirty-five min before the beginning of each day's test autoshaping session, all subjects were brought to the lab where their pre-session temperatures were measured and they received their designated injections. After the injections the subjects were returned to their home cages. Thirty min later the subjects were returned to the lab and run through one session of the autoshaping procedure they had been exposed to on all previous days. Each subject's post-session rectal temperature was measured shortly after the end of this session.

Results

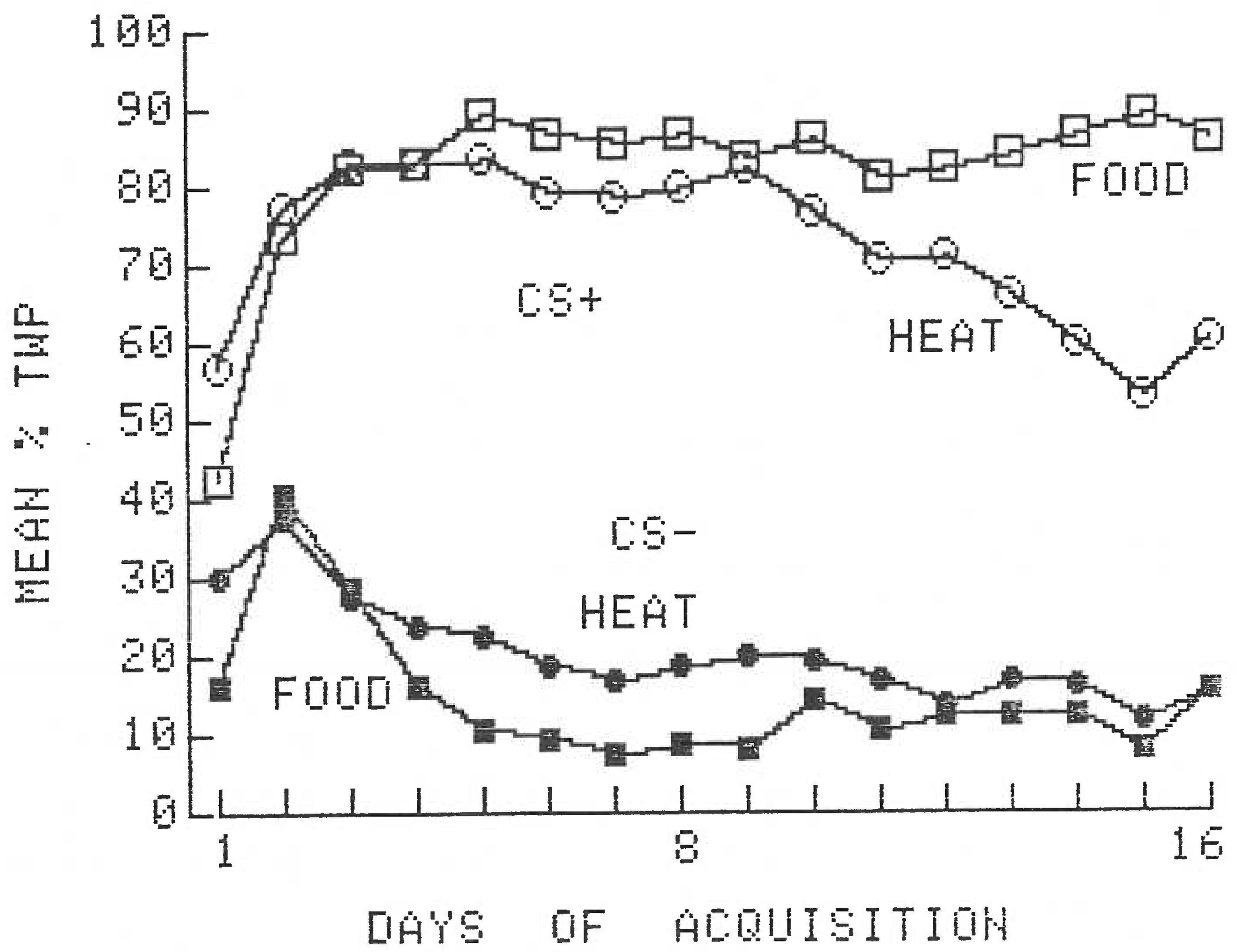
Acquisition: % TWP

Figure 10 shows the % TWP data of the Heat and Food groups on the 16 days of the Acquisition phase. The % CS+ TWP levels of both groups rose on Days 1, 2, and 3. The Heat group's % CS+ TWP level peaked on Day 3 and then remained slightly below this level until Day 9. The CS+ response level of this group dropped regularly on Days 10 to 15, then rose again slightly on Day 16. The Day 15 level was the Heat group's lowest % CS+ TWP level of the Acquisition phase. The Food group's % CS+ TWP level continued to rise until Day 5 and then remained at or near this peak level for the remainder of this phase. On Days 1 and 2 the Heat group's % CS+ TWP level was greater than that of the Food group. On Days 5 to 16 the Food group's % CS+ TWP level was higher than the Heat group's.

The % CS- TWP levels of both groups rose on Days 1 and 2. Responding to the CS- peaked in both groups on Day 2 and then dropped until Day 7. The Heat group's % CS- TWP level remained at or near its Day 7 level for the rest of this phase. This was true for the Food group as well. The % CS- TWP level of the Heat group was higher than that of the Food group on Day 1 and was higher again on Days 4 to 15. The Food group's CS- response level was never greater than the Heat group's.

An overall three-way ANOVA was run on the % TWP data of all subjects over the 16 days of the Acquisition phase. One of these factors was the between-group factor type of autoshaping, the other two factors were the within-group factors of type of stimulus and day of training. This analysis showed that the type of stimulus factor [F (1,28) = 239.04, $p < .001$] and its interaction with the day of training factor [F (15,420) = 18.40, $p < .001$] were significant. This reflects the observation that all subjects

Figure 10. The overall mean % CS+ TWP and % CS- TWP values of the Heat and Food autoshaping groups on the 16 days of the Acquisition phase.



MEAN % TWP
100
90
80
70
60
50
40
30
20
10
0
1
8
16
DAYS OF ACQUISITION
CS+
FOOD
HEAT
CS-
HEAT
FOOD

responded on a greater percentage of CS+ than CS- trials on all days of this phase and also that this difference was greater on the later days of this phase. The day of training factor was also significant [F (15,420) = 6.95, p < .001]. The type of autoshaping factor and its interaction with the type of stimulus factor were both not significant. However, the interaction between the type of autoshaping factor and the day of training factor was significant [F (15,420) = 3.59, p < .01], as was the interaction of all three factors [F (15,420) = 4.58, p < .001].

The difference between the two group's % CS+ TWP levels was examined each day with a one-way between-group ANOVA. This series of analyses showed that CS+ response levels of these groups were significantly different on only two days. On Days 15 [F (1,28) = 12.29, p < .001] and 16 [F (1,28) = 5.44, p < .05] the Heat group responded on a significantly fewer CS+ trials than the Food group. A similar series of analyses on the two group's % CS- TWP levels found that the CS- responding levels of the groups were not significantly different on any day of this phase.

The drop in responding to the CS- observed in the Food subjects between Days 2 and 7 was examined with a one-way within-group ANOVA. This analysis showed that these subjects responded on significantly fewer [F (1,13) = 19.61, p < .001] CS- trials on Day 7 than on Day 2. A similar analysis showed that the Food group's level of responding to the CS+ on Day 5 when it peaked was not significantly greater [F (1,13) = 0.66] than the level recorded on Day 16, the last day of acquisition training.

The drop in the Heat group's level of responding to the CS+ from Day 9 to Day 16 was evaluated with one-way within-group ANOVAs. These analyses showed that the Heat subjects responded on significantly more CS+ trials [F (1,15) = 26.27, p < .001] on Day 9 than on Day 15 when responding to the CS+ was at its lowest level. Analysis of the increase in responding to the CS+ from Day 15 to

Day 16 showed that the level on Day 16 was not significantly greater [F (1,15) = 2.62] than the level on Day 15. The level of responding to the CS+ on Day 16 was also significantly lower [F (1,15) = 15.14, $p < .01$] than the level on Day 9. A similar analysis showed that Heat subjects responded on significantly more [F (1,15) = 9.36, $p < .01$] CS- trials on Day 2 than on Day 7.

Acquisition: Pecks/CS

Figure 11 shows the mean PECKS/CS data for both the Heat and Food groups on the 16 days of the Acquisition phase. The rate of CS+ pecking increased rapidly in both groups on Days 1 and 2 and peaked in both groups on Day 3. After peaking the CS+ pecking rate of the Heat group dropped at a regular rate for the remainder of the study. The PECKS/CS+ level of the Food group dropped slightly after peaking and then remained at slightly lower than peak levels on the remaining days of the study. The CS+ pecking rate of the Heat group was greater than the Food group's rate until Day 10. On Days 12 to 16 the Heat group's CS+ pecking rate was lower than the Food group's.

The CS- pecking rates of both groups rose on Days 1 and 2. Both group's CS- pecking rates peaked on Day 2 and then dropped until Day 6. The CS- pecking rates of both groups then remained at a steady low level for the rest of this phase. The CS- pecking rates of both groups were equivalent on all days except Day 1 when the Heat group's rate was slightly higher than the Food group's rate. An overall three-way analysis was run on the PECKS/CS data of all subjects over the 16 days of Acquisition training. One of these factors was the between-group factor type of autoshaping. The other two factors were the within-group factors type of stimulus and day of training. This analysis showed that the type of stimulus factor [F (1,28) = 99.01, $p < .001$] and its interaction with the day of training factor [F (15,420) = 12.61, $p < .001$] were both significant. This reflects the observation that both groups pecked the CS+ at a higher rate

Figure 11. The overall mean PECKS/CS+ and PECKS/CS- values of the Heat and Food autoshaping groups on the 16 days of the Acquisition phase.

MEAN PECKS/CS

7
6.3
5.6
4.9
4.2
3.5
2.8
2.1
1.4
.7
0

HEAT

FOOD

CS+

CS-

HEAT

FOOD

1
8
16

DAYS OF ACQUISITION

than the CS- on all days of this phase and also that this difference was greater around the midpoint of this phase. The day of training factor was also significant [F (15,420) = 9.70, p < .001]. The type of autoshaping factor and its interaction with each within-group factor were nonsignificant. However, the interaction of all three factors was significant [F (15,420) = 5.15, p < .001].

The difference between the two group's CS+ pecking rates was examined each day with a one-way between-group ANOVA. This series of analyses showed that this difference was significant only on Day 1 when the Heat group pecked the CS+ at a significantly higher rate [F (1,28) = 6.28, p < .05] than the Food group. A similar series of analyses on the group's PECKS/CS- data showed that the CS- pecking rates of the two group's was significantly different, again, only on Day 1 when the Heat group pecked the CS- at a significantly higher rate [F (1,28) = 6.38, p <.05] than the Food group.

The drop in the Food group's level of CS- pecking that occurred between Days 2 and 5 was evaluated with a one-way within-group ANOVA. This analysis showed that the Food group made significantly fewer pecks [F (1,13) = 21.25, p < .001] during CS- presentations on Day 5 than Day 2. The drop in the Food group's level of CS+ pecking from Day 3 to Day 9 was examined with a similar ANOVA. This analysis showed that the Food group pecked the CS+ at a significantly lower rate [F (1,13) = 5.02, p < .05] on Day 9 than on Day 3. Although the minor increase in this group's rate of CS+ pecking from Day 9 to Day 14 was not significant, the Day 14 level was not significantly lower than the Day 3 level [F (1,13) = 0.71]. The Food group's CS+ pecking level on Day 16 was not significantly lower [F (1,13) = 2.03] than its peak level on Day 3.

The drop in the Heat group's level of CS- pecking from Day 2 to Day 6 was examined with a one-way within-group ANOVA. This analysis showed that Heat subjects pecked significantly less frequently [F (1,15) = 13.52, p < .01]

during CS- presentations on Day 6 than Day 2. The drop in pecking to the CS+ from Day 3 to Day 15 was evaluated with a similar analysis. This ANOVA showed that these subjects made significantly fewer pecks [F (1,15) = 31.60, p < .001] during the CS+ on Day 15 than on Day 3. The Heat group's CS+ pecking rate on Day 16 was not significantly greater [F (1,15) = 0.69] than it had been on Day 15. Heat subjects also made significantly less pecks [F (1,15) = 26.42, p < .001] during CS+ presentations on Day 16, the last day of acquisition training, than on Day 3.

Test: % TWP

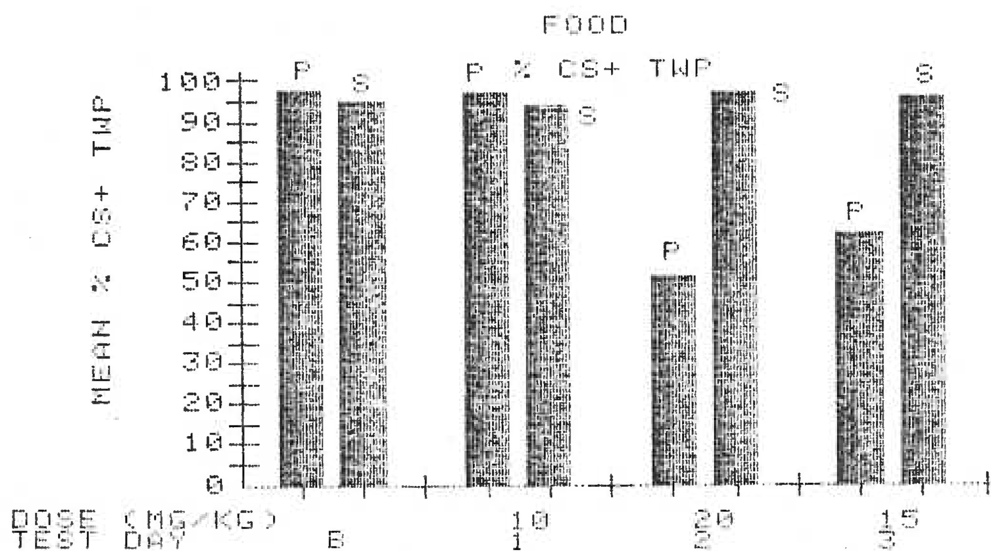
Beacuse of the differences in the response levels of the Heat and Food groups present at the end of the Acquisition phase, Test phase ANOVAs were run separately on the data from these two groups.

Food Autoshaping. Figure 12A shows the mean % CS+ TWP data for the propranolol and saline divisions of the Food group over the days of the Test phase. Figure 12B shows the mean % CS- TWP Test phase data of these groups. The graphs show that the response levels of the Saline-Food group to both the CS+ and CS- changed little over the course of this phase. After a 10 mg/kg injection on Day 1, response levels of the propranolol (Prop) group remained at the group's baseline levels. However, response levels of these subjects fell below their baseline levels after a 20 mg/kg injection on Day 2 and again on Day 3 after a 15 mg/kg injection.

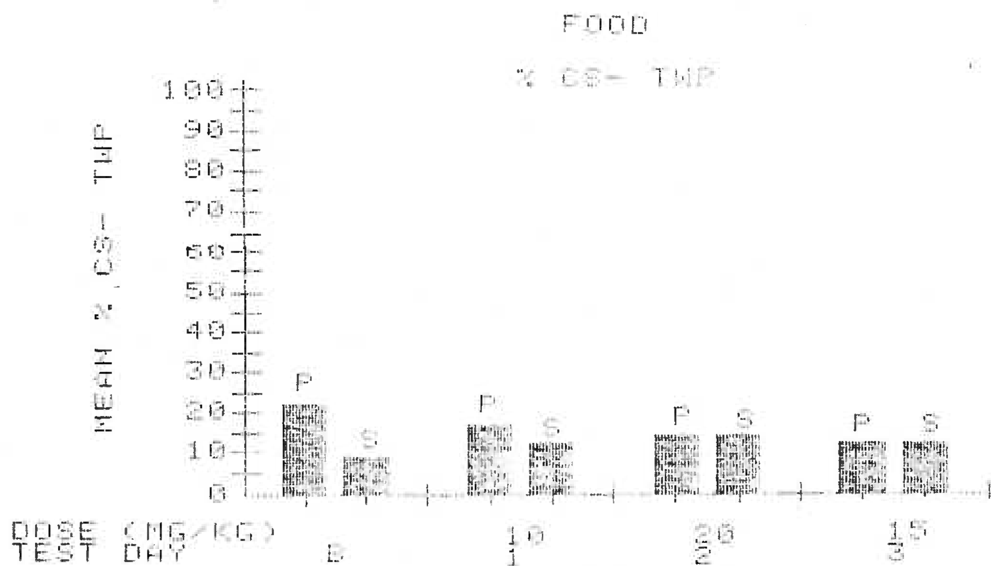
The Test phase data of all the Food group subjects were evaluated with an overall three-way ANOVA. One of these factors was the between-group factor type of treatment (propranolol or saline). The others were the within-group factors type of stimulus and day of testing. This analysis showed that the type of stimulus factor was significant [F (1,10) = 211.84, p < .001] reflecting the

Figure 12. The mean % CS+ TWP (12A) and % CS- TWP (12B) values of the propranolol (P) and saline (S) divisions of the Food autoshaping group on the baseline (B) and three drug test days of the Test phase.

(A)



(B)



observation that these subjects responded on more CS+ than CS- trials on all the days of this phase. The day of testing factor was also significant [F (3,30) = 5.74, $p < .01$]. The type of treatment factor was not found to be significant, however, there was a significant interaction between the type of treatment and day of testing factors [F (3,30) = 7.99, $p < .001$]. A variety of followup analyses were run to identify the source of this significant effect.

Between-group differences in response levels were examined on each day with a one-way between-group ANOVA. Analysis of the baseline day data showed that there was no significant difference [F (1,10) = 0.96] between the overall baseline response levels of the saline and propranolol groups. Although the baseline level of responding to the CS- was higher in the Prop-Food than in the Saline-Food group, this difference was not significant [F (1,10) = 0.83]. The response levels of these two treatment groups were not significantly different on Day 1 after the Prop-Food group had received a 10 mg/kg injection. After a 20 mg/kg injection of propranolol on Day 2, Prop-Food subjects responded on significantly fewer [F (1,10) = 9.64, $p < .05$] CS+ trials than Saline-Food subjects. The difference between the Day 2 CS- response levels of these two groups was not significant [F (1,10) = 1.35]. The Prop-Food subjects responded on fewer CS+ trials than their saline counterparts again on Day 3 after a 15 mg/kg injection. This difference, however, was found to be nonsignificant [F (1,10) = 3.43].

Within-group differences between response levels on the baseline and test days were evaluated with one-way within-group ANOVAs. The Prop-Food group's CS+ and CS- response levels after a 10 mg/kg injection on Day 1 were not significantly different from its CS+ and CS- response levels on the baseline day. After the 20 mg/kg injection on Day 2, propranolol subjects responded on significantly fewer [F (1,5) = 10.79, $p < .05$] CS+ trials than they had on the baseline day. This group's Day 2 level of responding to the CS- was not

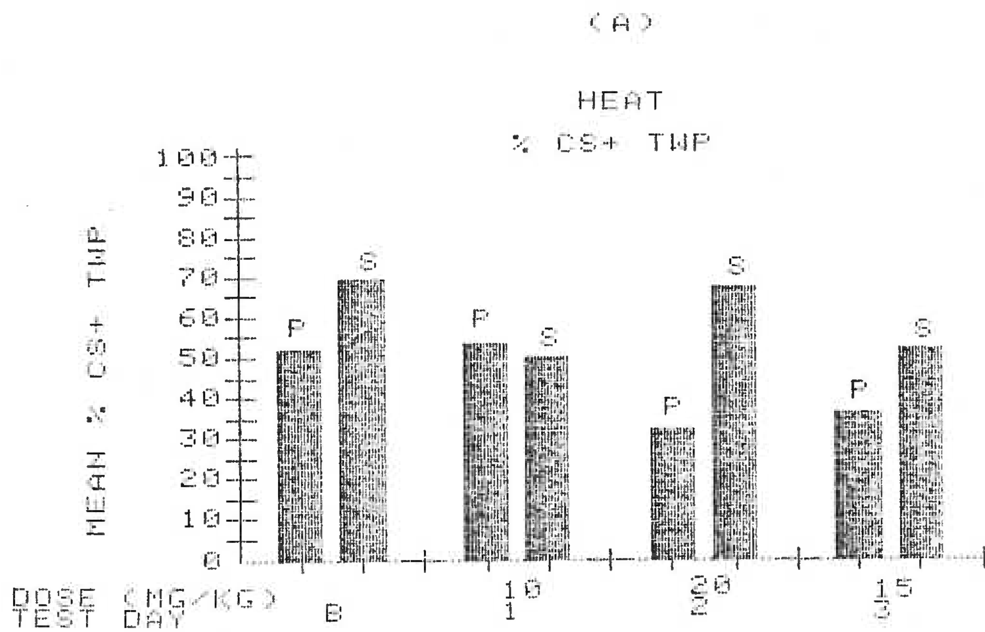
significantly different [F (1,5) = 2.23] than its baseline level. Although response levels to both stimuli fell after the 15 mg/kg injection on Day 3, these levels were not significantly lower than the baseline CS+ [F (1,5) = 4.30] and CS- [F (1,5) = 3.06] response levels.

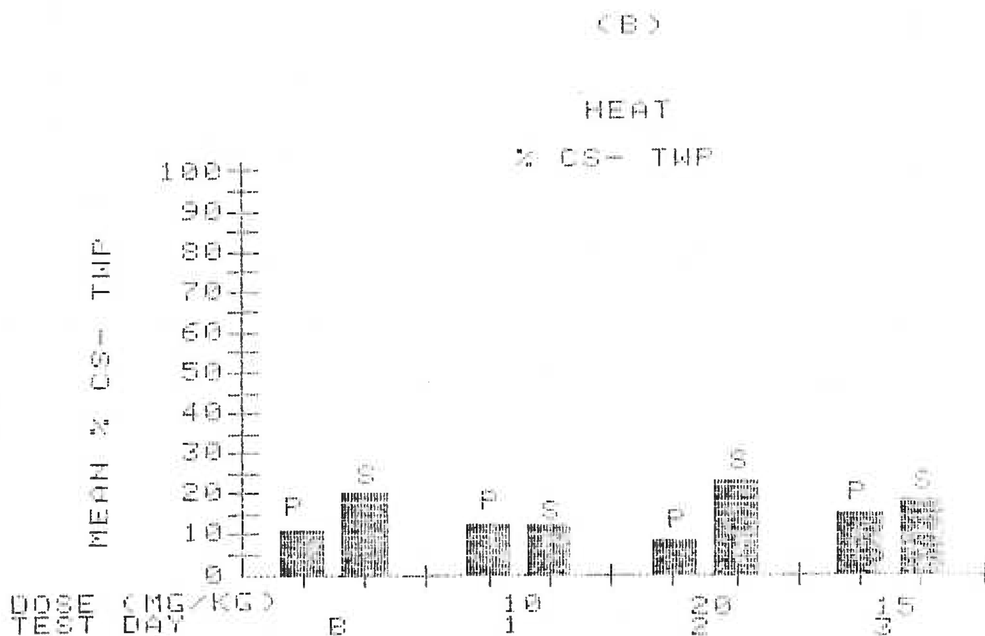
Heat Autoshaping. Figure 13A shows the mean % CS+ TWP data of the saline and propranolol divisions of the Heat group over the days of the Test phase. Figure 13B shows the mean % CS- TWP Test phase data for these subjects. The figures show that after a 10 mg/kg injection, the response levels of the Prop-Heat group remained at baseline levels. This group's level of responding to the CS+ dropped below their baseline level after a 20 mg/kg injection of propranolol on Day 2 and again on Day 3 after a 15 mg/kg injection. In the Saline-Heat group, response levels of both stimuli dropped below the group's baseline levels on Day 1. These response levels rose back to near baseline on Day 2 and then fell again on Day 3.

An overall three-way ANOVA was run on the % TWP Test phase data of all subjects in the Heat group. One of these factors was the between-group factor type of treatment, the others were the within-group factors type of stimulus and day of testing. This analysis showed that the type of stimulus factor was significant [F (1,14) = 45.13, $p < .001$] reflecting the observation that all these subjects responded on more CS+ than CS- trials on each day of the Test phase. The type of treatment and day of testing factors were both found to be nonsignificant, however, the interaction between these two factors [F (3,42) = 3.36, $p < .05$] was the only significant interaction in this analysis. The following analyses were carried out to locate the source of this significant effect.

Differences between the response levels of the Saline-Heat and Prop-Heat groups on each day of this phase were evaluated with a one-way between-group

Figure 13. The mean % CS+ TWP (13A) and % CS- TWP (13B) values of the propranolol (P) and saline (S) divisions of the Heat autoshaping group on the baseline (B) and three drug test days of the Test phase.


(A)
HEAT
% CS+ TWP
MEAN % CS+ TWP
100
90
80
70
60
50
40
30
20
10
0
P
S
P
S
P
S
P
S
DOSE (MG/KG)
TEST DAY
B
10
1
20
2
15
3


(B)
HEAT
% CS− TWP
MEAN % CS− TWP
100
90
80
70
60
50
40
30
20
10
0
P
S
P
S
P
S
P
S
DOSE (MG/KG)
TEST DAY
B
10
1
20
2
15
3

ANOVA. Although the baseline response levels of the Saline-Heat group are higher than those of the Prop-Heat group, the overall response levels of these two groups were not significantly different [F (1,14) = 1.19]. The differences between the two group's baseline CS+ [F (1,14) = 1.33] and CS- [F (1,14) = 0.61] response levels were not significant. On Day 1 after a 10 mg/kg injection of propranolol, the overall response level of the Prop-Heat group was not significantly different [F (1,14) = 0.01] from that of the Saline-Heat group. After a 20 mg/kg injection on Day 2, however, the Prop-Heat subjects responded on significantly fewer [F (1,14) = 4.16, p < .05] CS+ trials than their saline counterparts. The CS- response levels of these two groups were not significantly different [F (1,14) = 1.54] on Day 2. On Day 3, after a 15 mg/kg injection, the Prop-Heat subjects again responded on fewer CS+ trials than the Saline-Heat subjects, however, this difference was found to be nonsignificant [F (1,14) = 1.09].

The differences between each group's response levels on the baseline and test days were examined with one-way within-group ANOVAs. After a 10 mg/kg injection on Day 1 the Prop-Heat group responded on nearly the same percentage of CS+ trials as it had on the baseline day, the difference was not significant. After the 20 mg/kg injection on Day 2, Prop-Heat subjects responded on significantly fewer CS+ trials [F (1,7) = 8.21, p < .05] than they had on the baseline day. This group's level of responding to the CS+ was lower than baseline again on Day 3 after a 15 mg/kg injection, however, this difference was not significant [F (1,7) = 1.90]. This series of analyses also showed that the Saline-Heat group responded on significantly fewer [F (1,7) = 12.60, p < .01] CS+ trials on Day 1 than on the baseline day. This drop was probably the result of the drop-off effect. The Saline-Heat group's levels of CS+ responding on Days 2 and 3 were not significantly lower than its baseline day level. A similar drop was not observed in the Prop-Heat group on Day 1. It is possible

that this absence of a similar drop was due to a drug effect that countered this drop by causing a slight increase in responding to the CS+.

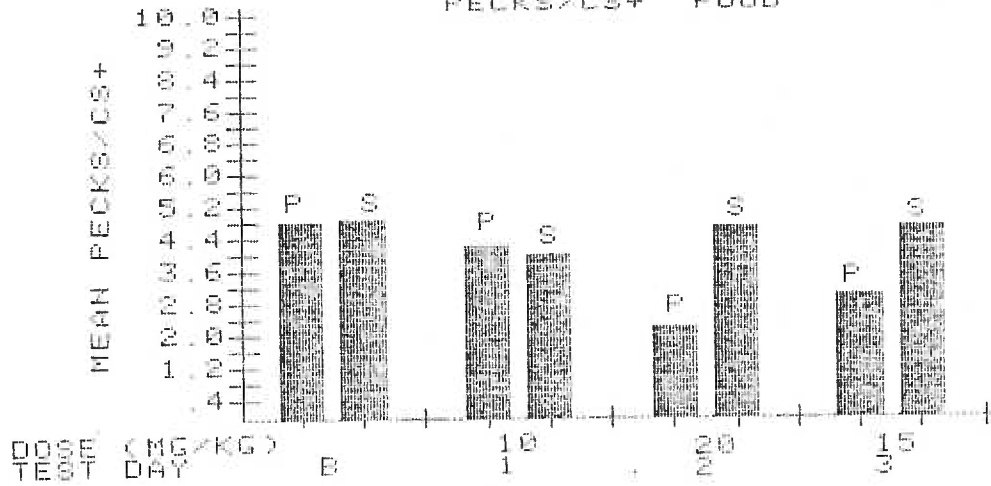
Test: Pecks/CS

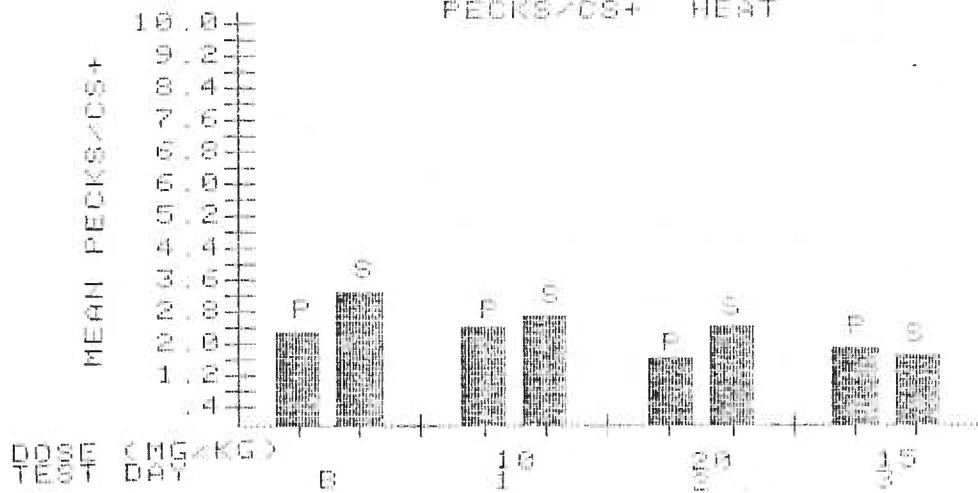
Food Autoshaping. Figure 14A shows the mean PECKS/CS+ data of the propranolol and saline divisions of the Food group over the days of the Test phase. This graph shows that the Prop-Food and Saline-Food group's levels of CS+ pecking were equivalent on the baseline day and again on Day 1. On Days 2 and 3 the Saline-Food group's rate of pecking the CS+ was greater than that of the Prop-Food group. The two group's rates of CS- pecking were equivalent on the baseline and all test days. The CS- pecking rates of both groups ranged between only 0.0 and 0.4 PECKS/CS- over the four days of this phase.

An overall three-way ANOVA was run on the PECKS/CS Test phase data of all the subjects in the Food group. One of these factors was the between-group factor type of autoshaping. The other two factors were the within-group factors type of stimulus and day of testing. This analysis showed that the type of stimulus factor was significant [F (1,10) = 38.00, $p < .001$] reflecting the observation that this group pecked the CS+ at a higher rate than the CS- on all days of this phase. The type of treatment and day of testing factors were both nonsignificant. Also, none of the possible factor interactions were found to be significant. Despite the absence of any significant interaction effects a series of followup analyses was run on this data that uncovered certain significant details missed by the overall analysis.

The difference between a group's response levels on the baseline and test days were analyzed with one-way within-group ANOVAs. The Prop-Food group's rate of CS+ pecking was lower than its baseline level on all test days. The CS+ pecking rate of the Prop-Food group after a 10 mg/kg injection on Day 1 was not significantly lower than its baseline day rate. The rate of CS+ pecking was almost significantly lower [F (1,5) = 5.87, $p < .059$] than the baseline

Figure 14. The mean PECKS/CS+ values of the propranolol (P) and saline (S) divisions of the Food (14A) and Heat (14B) autoshaping groups on the baseline (B) and three drug test days of the Test phase.


(A)
PECKS/CS+ FOOD
MEAN PECKS/CS+
10.0
9.2
8.4
7.6
6.8
6.0
5.2
4.4
3.6
2.8
2.0
1.2
.4
P
S
DOSE (MG/KG)
TEST DAY
B
10
1
20
2
15
3


(B)
PECKS/CS+ HEAT
MEAN PECKS/CS+
10.0
9.2
8.4
7.6
6.8
6.0
5.2
4.4
3.6
2.8
2.0
1.2
.4
P
S
DOSE (MG/KG)
TEST DAY
B
10
1
20
2
15
3

level after the 20 mg/kg injection on Day 2 and was significantly lower than baseline [F (1,5) = 6.51, $p < .05$] after the 15 mg/kg injection on Day 3. The Saline-Food group's rates of CS+ pecking on Days 2 and 3 were equivalent to its baseline rate. On Day 1, this group's rate of CS+ pecking fell below its baseline rate, but the difference was not significant [F (1,5) = 2.25].

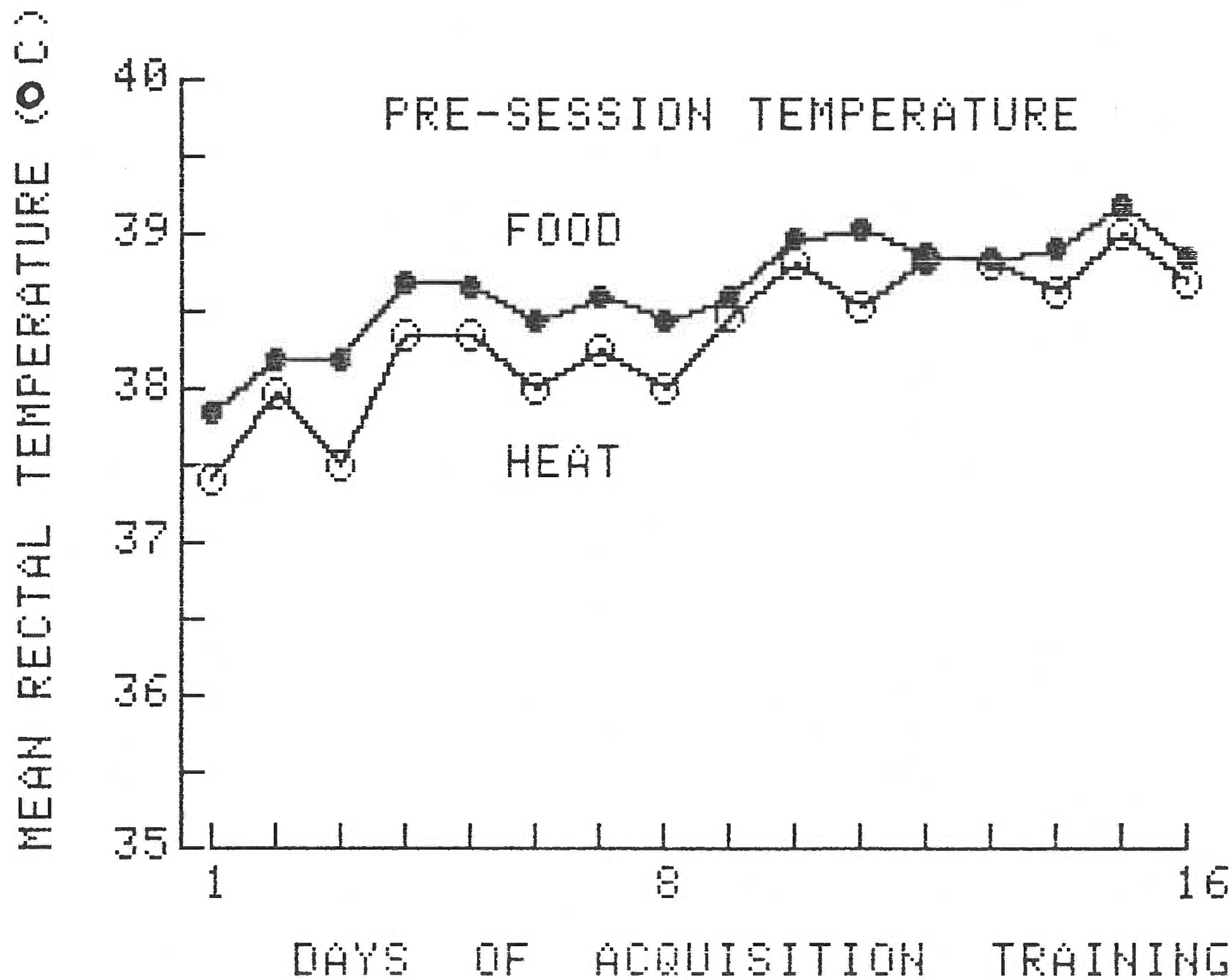
Heat Autoshaping. Figure 14B shows the mean PECKS/CS+ data of the propranolol and saline divisions of the Heat group over the days of the Test phase. This figure shows that the Prop-Heat group's rate of CS+ pecking fell below its baseline rate on Day 2 after a 20 mg/kg injection and again on Day 3 after a 15 mg/kg injection. The Saline-Heat group's rate of CS+ pecking decreased regularly on all test days. The Prop-Heat group's rate of CS+ pecking was lower than that of the Saline-Heat group on the baseline day and on Day 2. The CS+ pecking rates of these two group's were equivalent on Days 1 and 3.

An overall three-way ANOVA was carried out on the PECKS/CS data of all Heat group subjects over all four days of the Test phase. One of these factors was the between-group factor type of treatment. The other two factors were the within-group factors type of stimulus and day of testing. This analysis showed that the type of stimulus factor was significant [F (1,14) = 17.01, $p <$ .01]. The type of treatment and day of testing factors were both nonsignificant. Also, none of the possible factor interactions were significant.

Temperature Data

Training Phase: Pre-Session Temperatures. Figure 15 shows the mean pre-session rectal temperatures of the Heat and Food groups on the 16 days of the Acquisition phase. Subjects were 6 days old at the beginning of this phase. The graph shows that the pre-session temperatures of both groups increased slightly over the first half of this phase. Pre-session temperatures of both

Figure 15. The mean pre-session rectal temperatures of the Food and Heat autoshaping groups on the 16 days of the Acquisition phase.


PRE-SESSION TEMPERATURE
FOOD
HEAT
MEAN RECTAL TEMPERATURE (°C)
40
39
38
37
36
35
1
8
16
DAYS OF ACQUISITION TRAINING

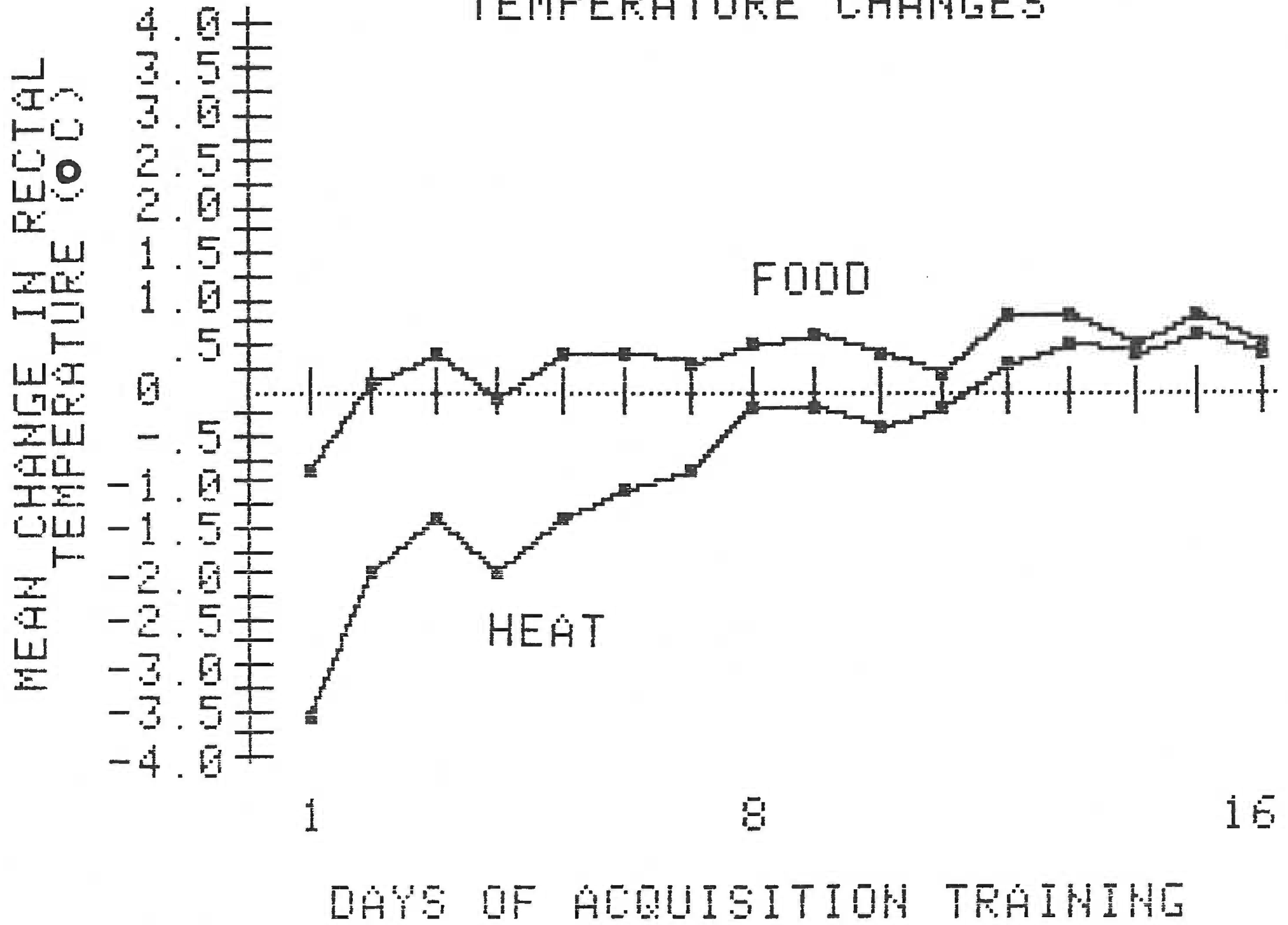
groups leveled out during the second half of this phase. The Food group's pre-session temperatures were slightly higher than those of the Heat group on the first 8 days. The two group's pre-session temperatures were equivalent on the last 8 days of this phase.

An overall two-way ANOVA was run on the pre-session temperature data of all subjects over the 16 days of the Acquisition phase. One factor was the between-group factor type of autoshaping, the other was the within-group factor day of training. This analysis showed that the day of training factor was significant [F (15,420) = 24.91, $p < .001$]. The type of autoshaping factor and the interaction between these two factors were both nonsignificant.

Training Phase: Temperature Changes. Figure 16 shows the change in the Food group's rectal temperatures caused by a 12 min exposure to room temperature (24° C) over the 16 days of the Acquisition phase. This figure also shows the change in the Heat group's temperature produced by the 12 min exposure to 9° C over the days of this phase. The largest temperature drop in both groups occurred on Day 1. On Days 2 and 3 the Food group's temperature increased a fraction of a degree and then dropped slightly again on Day 4. From Days 5 to 16 the Food group's temperature increased slightly in response to the room temperature exposure. The magnitude of this increase was greatest on Days 12, 13, and 15. The Heat group's temperature continued to drop in response to the cold exposure from Day 2 to Day 11. The magnitude of this drop decreased regularly over these days. From Days 12 to 16 the cold exposure caused the Heat group's temperature to increase slightly.

On the first 7 days of this phase the Heat group's temperature change after exposure to 9° C was larger than the Food group's temperature change after exposure to room temperature. The size of the two group's temperature changes

Figure 16. The mean changes in the rectal temperatures of the Food autoshaping group after a 12 min exposure to room temperature (24° C) and in the Heat autoshaping group after a 12 min exposure to 9° C on the 16 days of the Acquisition phase.



TEMPERATURE CHANGES
MEAN CHANGE IN RECTAL TEMPERATURE (°C)
FOOD
HEAT
0
1
8
16
DAYS OF ACQUISITION TRAINING

were equivalent on the remaining days of Acquisition training.

An overall two-way analysis was run on the temperature change data of all subjects on the 16 days of this phase. One of these factors was the between-group factor type of autoshaping, the other was the within-group factor day of training. This analysis showed that the type of autoshaping [F (1,28) = 38.30, $p < .001$] and day of training factors [F (15,420) = 42.94, $p < .001$] were both significant. The interaction between these two factors was also significant [F (15,420) = 12.16, $p < .001$].

The difference between the magnitude of the two group's temperature changes was examined each day with a one-way between-group ANOVA. This series of analyses showed that the temperature change in the Heat group was significantly larger than the change in the Food group's temperature on the first 11 days of this phase. The difference between the two group's temperature changes was not significant on Days 12 to 16.

The change in the size of the Food group's temperature increase over the days of this phase was found to be significant [F (15,195) = 11.95, $p < .001$] by a one-way within-group ANOVA. A similar analysis showed that the change in the size of the Heat group's temperature drop was also significant [F (15,225) = 36.13, $p < .001$].

The difference between the Heat group's pre and post-session temperatures was evaluated with a t test only on those days when the group's post-session temperature was equal to or lower than its pre-session temperature. These tests showed that the difference between the Heat group's pre and post-session temperatures was significant on the first 6 days of the Acquisition phase.

Test: Pre-Session Temperatures. Table 2 lists the mean pre-session temperatures of the Saline and Propranolol divisions of both the Heat and Food groups for the four days of the Test phase. Overall, the pre-session temperatures of all 4 groups were equivalent on all days of the Test phase.

Table 2. The mean pre-session temperatures of the propranolol (Prop) and saline divisions of the Food and Heat autoshaping groups on the four days of Test phase.

Table 2

Test Phase Pre-Session Temperatures

($^{\circ}$C)

Group	Baseline	Test Day 1	2	3
Prop-Food	39.0	39.2	38.6	38.7
Saline-Food	38.8	38.8	38.9	38.7
Prop-Heat	38.8	38.7	38.8	38.8
Saline-Heat	38.6	38.6	38.8	38.7

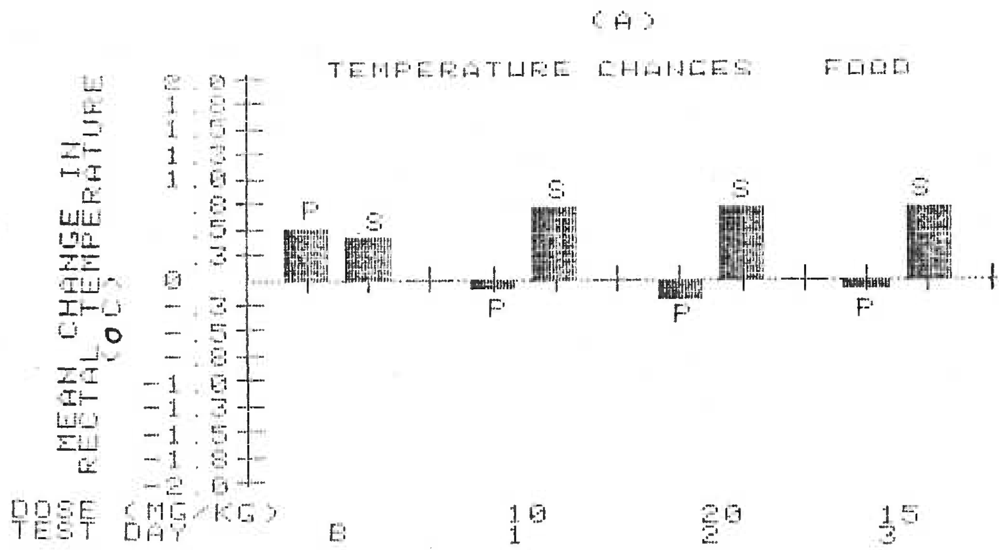
An overall three-way ANOVA was run on the pre-session temperature data of all subjects over the four days of the Test phase. Two of these factors were the between-group factors type of autoshaping and type of treatment (propranolol or saline). The remaining factor was the within-group factor day of testing. This analysis showed that none of these factors or their interactins were significant.

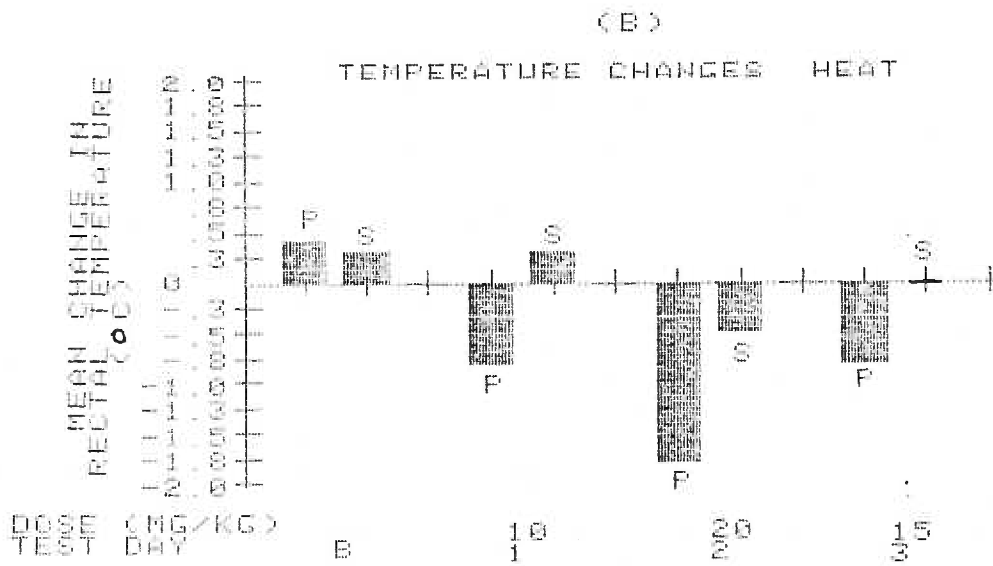
Test: Temperature Changes / Food. Figure 17A shows the mean changes in the rectal temperatures of the Prop-Food and Saline-Food groups caused by a 12 min exposure to room temperature (24° C) on the four days of the Test phase. The temperatures of both groups increased slightly on the baseline day. The Prop-Food group's temperature dropped slightly on all three drug test days. The drops caused by the 10 mg/kg injection on Day 1 and the 15 mg/kg injection on Day 3 were equivalent. The drop caused by the 20 mg/kg injection on Day 2 was larger than the drops on Days 1 and 3.

The Saline-Food group's temperature increased slightly on all drug test days. The size of this mean increase was the same on all three days. This group's test day increases were all greater than its baseline day increase. The magnitudes of the two group's temperature increases on the baseline day were equivalent. The size of the Saline-Food group's temperature increase on the three drug test days was larger than the size of the drops in the Prop-Food group's temperature on these days.

An overall two-way ANOVA was run on the temperature changes of all Food group subjects over these four test days. One factor was the between-group factor type of treatment, the other was the within-group factor day of testing. This analysis showed that the type of treatment factor was significant [F $(1,10) = 15.98$, $p < .01$]. The day of testing factor and the interaction between these two factors were both nonsignificant.

Figure 17. The mean changes in the rectal temperatures of the propranolol (P) and saline (S) divisions of the Food autoshaping group (17A) after a 12 min exposure to room temperature (24° C) and of the Heat autoshaping group (17B) after a 12 min exposure to 9° C on the baseline (B) and three drug test days of the Test phase.


(A)
TEMPERATURE CHANGES FOOD
MEAN CHANGE IN RECTAL TEMPERATURE (°C)
DOSE (MG/KG)
TEST DAY
B
10
1
20
2
15
3


(B)
TEMPERATURE CHANGES HEAT
MEAN CHANGE IN RECTAL TEMPERATURE (°C)
DOSE (MG/KG)
TEST DAY
B
10
1
20
2
15
3

Test: Temperature Changes / Heat. Figure 17B shows the mean changes in the rectal temperatures of the Prop-Heat and Saline-Heat groups caused by the 12 min exposure to 9° C on the four days of the Test phase. The temperatures of both groups increased slightly on the baseline day. The Prop-Heat group's temperature dropped on all three drug test days. The drops following a 10 mg/kg injection on Day 1 and after 15 mg/kg injection on Day 3 were equivalent. The drop following a 20 mg/kg injection on Day 2 was larger than the drops that occurred on Days 1 and 3.

The Saline-Heat group's temperature rose slightly on Day 1, dropped slightly on Day 2, and did not change at all on Day 3. The increase on Day 1 was equivalent to this group's temperature increase on the baseline day. The size of the group's temperature drop on Day 2 was slightly larger than the size of this group's temperature increases on the baseline day and Day 1. The magnitudes of the two group's temperature increases on the baseline day were equivalent. The size of the drops in the Prop-Heat group's temperature was greater than the size of the Saline-Heat group's temperature changes on the three drug test days.

An overall two-way ANOVA was run on the temperature changes of all Heat group subjects on the four days of this phase. One of these factors was the between-group factor type of treatment, the other was the within-group factor day of testing. This analysis showed that the type of treatment [F (1,14) = 6.12, $p < .05$] and day of testing factors [F (3,42) = 12.29, $p < .001$] were both significant. The interaction between these two factors was also significant [F (3,42) = 3.01, $p < .05$].

The difference between the group's temperature changes was examined on each day with a one-way between-group ANOVA. On the baseline day the increases in the temperatures of both groups were not significantly different [F (1,14) = 0.24]. The temperature change was significantly greater in the Prop-Heat

group than in the Saline-Heat group after a 10 mg/kg injection on Day 1 [F $(1,14) = 6.46$, $p < .05$] and again on Day 3 after a 15 mg/kg injection [F $(1,14) = 5.57$, $p < .05$]. Although the Prop-Heat group's temperature drop was substantially larger than the Saline-Heat group's after a 20 mg/kg injection on Day 2, the difference was not found to be significant [F $(1,14) = 3.92$]. This is probably due to the large variation in the size of the temperature drops of the Prop-Heat group and the relatively smaller variation in the size of the temperature changes in the Saline-Heat group.

The differences between a groups temperature changes on the baseline and test days were examined with a series of one-way within-group ANOVAs. The Prop-Heat group's temperature change after a 10 mg/kg injection on Day 1 was significantly greater [F $(1,7) = 12.38$, $p < .01$] than its baseline day change. This was true after a 20 mg/kg injection on Day 2 [F $(1,7) = 14.09$, $p < .01$] and again on Day 3 after a 15 mg/kg injection [F $(1,7) = 14.78$, $p < .01$]. The differences between this group's temperature drops on Days 1 and 2 and Days 2 and 3 were equal. However, while the difference between Days 1 and 2 was significant [F $(1,7) = 5.88$, $p < .05$], the same difference between the drops on Days 2 and 3 was not [F $(1,7) = 4.89$].

The Saline-Heat group's temperature change on the baseline day was not significantly different from its change on Day 1 [F $(1,7) = 0.05$] and Day 3 [F $(1,7) = 0.98$]. This group's temperature change on Day 2 was significantly greater than it was on both the baseline day [F $(1,7) = 12.04$, $p < .05$] and Day 1 [F $(1,7) = 6.15$, $p < .05$]. The Saline-Heat group's temperature change on Day 2, however, was not significantly different [F $(1,7) = 2.48$] from its change on Day 3. Also, the group's temperature changes on Days 1 and 3 were not significantly different [F $(1,7) = 0.84$].

Discussion

The results from the heat autoshaping part of Experiment 2 do not support the proposed causal relationship between thermoregulatory development and the drop-off effect. Based on the proposed hypothesis, a prediction was made that propranolol should cause an increase in the response levels of chicks in the Heat study. In this experiment, however, none of the three dosages of propranolol caused an increase in the level of responding, and the two larger doses caused decreases in performance.

Experiment 2 was designed to show that the increases in performance observed after an injection of propranolol in Experiment 1 were not due to some general activity stimulating effect of the drug. Such a demonstration would have supported the conclusion that the observed performance increase was due to the drug's detrimental effects on a chick's ability to maintain a stable body temperature. This was to be achieved here by showing that propranolol would not influence the level of autoshaped responding in the Food paradigm where thermoregulatory ability was not a relevant variable in the control of performance.

The results from the food autoshaping part of Experiment 2 do demonstrate that propranolol does not have a general response activity stimulating effect. Propranolol did not cause an increase in the Food group's level of autoshaped responding at any of the three dosages. As in the Heat group, the two larger doses of propranolol caused a decrease in the response levels of the Food subjects. From the results of both the Heat and Food groups it can be seen that the 10 mg/kg dose of propranolol had no effect on the chick's responding behavior. The 15 and 20 mg/kg doses, however, both caused decreases in the chick's level of autoshaped responding.

Propranolol, again, is a drug that can disrupt a chick's ability to maintain a stable body temperature under a cold load. This capacity of the drug can be seen in the temperature data from the Heat group. During the Test phase the 12 min exposure to 9°C resulted in a slight temperature increase in the Saline-Heat group. In the Prop-Heat group the cold exposure caused a temperature drop of nearly 1 C after both the 10 and 15 mg/kg injections of propranolol. The 20 mg/kg injection resulted in a nearly 2°C drop in temperature. On the basis of the size of the drop in temperature, the 20 mg/kg dose had twice the effect of the two smaller dosages on the chick's thermoregulatory ability.

General Discussion

In Experiment 1, the 10 mg/kg injection of propranolol did, as predicted, cause an increase in autoshaped responding. On the other hand, in Experiment 2, the same injection of propranolol caused no change in the level of responding, and two larger doses (15 and 20 mg/kg) caused decreases in responding. The following is an attempt to account for this inconsistency.

There were two notable differences between the propranolol tests in these two experiments. In Experiment 1 subjects were 16 days old on the Test day when they received a 10 mg/kg injection of propanolol. In Experiment 2 subjects were 21 days old when they received this injection. In order to control for procedural differences between the heat and food paradigms, heat autoshaping subjects were food-deprived throughout Experiment 2. Heat autoshaping subjects were not food-deprived in Experiment 1.

As mentioned in the introduction to Experiment 2, one possible outcome of the procedure could be the negation of propranolol's effect on response levels by some effect of food deprivation. So one possible account of the inconsistency is that in Experiment 2, hunger prevented the increase in responding caused by a 10 mg/kg injection of propranolol in Experiment 1.

In Experiment 2 the observation that a 10 mg/kg dose of propranolol caused no change in responding, while the two larger doses caused a decrease in responding suggests the existence of a dose-response curve. In this dose-response relationship, propranolol is believed to have two different effects on the chick and its behavior. One effect is to reduce the effectiveness of the chick's innate thermoregulatory mechanisms. This effect leads to an increased level of thermal motivation when the chick is exposed to a cold load. The above hypothesis holds that this increase in motivation will be reflected as an increase in the chick's CS+ pecking activity. The second effect

of this drug, observed at the larger dosages in Experiment 2, is to cause a depression in the chick's level of general somatic activity, which includes keypecking.

These two effects of propranolol are combined in the proposed dose-response relationship as follows. At smaller dosages propranolol will cause increases in the chick's level of thermal motivation that will lead to increased CS+ pecking activity. Also, these smaller doses will cause no depression in somatic activity. As the size of the dose increases propranolol will continue to cause increases in CS+ pecking through its effects on thermal motivation and it will also cause some depression of this responding through its effects on somatic activity. The observed level of CS+ pecking at these dosages would be the net result of the drugs response stimulating and depressing effects. At larger dosages the drug's somatic activity depressing effect would be larger than its stimulating effect, the result being either no change or a decrease in the chick's CS+ pecking level. At some point on this dose-response curve there is an optimum dosage at which propranolol's response stimulating and depressing effects would balance out to result in the largest possible increase in CS+ pecking.

In Experiment 1 the 10 mg/kg injection presumably was near this optimum dosage for these 16 day old, nonfood-deprived chicks. However, in Experiment 2 this same dosage was not near the optimum in these 21 day old, food-deprived chicks. Assuming such a dose-response relationship exists, it is possible that the optimum dosage shifted such that the optimum dosage for the 21 day old subjects was different and possibly higher than the optimum dosage for the 16 day old subjects. This shift could be the result of age and/or food deprivation.

One final explanation focuses on propranolol's detrimental effects on the chick's ability to maintain a stable body core temperature during a cold load.

Propranolol, again, is a beta adrenergic receptor blocker that is believed to prevent the chick's autonomically-mediated shivering thermogenesis response to cold. This thermogenic reaction compensates for heat lost from the body during the cold exposure. It is possible that a 21 day old chick's postural reactions to cold are so effective in controlling heat loss that the absence of shivering thermogenesis has little effect on the ability to maintain a stable core temperature. Thus, even if propranolol did effectively prevent shivering thermogenesis, the postural reactions alone could still effectively provide escape from the cold. Despite this loss of propranolol's thermoregulatory effect, larger doses could still cause a depression of the CS+ pecking levels of these older chicks's through the drug's somatic activity depressing effect.

Summary and Conclusions

The overall goal of this study was to provide supporting evidence for the belief that the drop-off effect observed in heat autoshaping studies is the result of thermoregulatory development in chicks. A response hierarchy hypothesis was developed to account for this causal relationship. The final form of this hypothesis was a cold escape response hierarchy. On the basis of this hypothesis a prediction was made that when the effectiveness of the chick's innate thermoregulatory responses to cold were reduced, there would be an increase in the occurrence of the learned operant thermoregulatory response of pecking the CS+.

This prediction was tested in Experiment 1, a heat autoshaping study in which a 10 mg/kg dose of propranolol was used to reduce the effectiveness of chick's innate thermoregulatory reactions to cold. As predicted, compromising the chick's innate thermoregulatory abilities resulted in an increased level of CS+ pecking. By demonstrating that the prediction was correct, the results of

this experiment supported the proposed hypothesis on which the prediction was based. However, these results could not be regarded as conclusive evidence for the proposed hypothesis because the observed increase in responding could have been the result of some general activity stimulating effect of propranolol that was independent of its detrimental thermoregulatory effect.

Experiment 2 was designed to address this issue. The nature of propranolol's autoshaped response stimulating effect was examined in Experiment 2 by evaluating the drug's effect on performance in an autoshaping paradigm where thermoregulatory ability was not a relevant performance variable. A food autoshaping procedure was used for this purpose. Also, in Experiment 2 an attempt was made to replicate the results of Experiment 1 using a slightly modified heat autoshaping procedure.

In the heat autoshaping procedure of Experiment 2, a 10 mg/kg injection of propranolol caused no change in the chicks CS+ pecking activity. These results, which show that the above prediction about the drug's effect on the level of responding was incorrect, do not support the proposed hypothesis. This same dosage also caused no change in the response levels of the food autoshaping subjects. In both the Heat and Food groups 15 and 20 mg/kg doses of propranolol resulted in equivalent decreases in the level of responding.

The effects of these three different doses of propranolol suggest that a dose-response curve may exist for the drug's effect on autoshaped response levels. In such a dose-response relationship there would be an optimum dose of propranolol that would cause an increase in responding. A 10 mg/kg dose apparently was the optimum dose in the 16 day old chicks in Experiment 1, but fell short of the optimum dose for the 21 day old chicks in the second experiment. This suggests that the optimum dose might be age-dependent.

Further research on this hypothesis should next focus on demonstrating the existence of such a dose-response relationship in the heat autoshaping paradigm.

Once an optimum dose is identified, the issue of whether propranolol really can result in an increase in autoshaped responding could be clarified. If the drug truly does produce such an increase, then the nature of its stimulating effect would have to be examined again using the optimum dose in a food autoshaping study.

There are nondrug means for reducing the effectiveness of the chick's innate thermoregulatory responses to cold. Wekstein and Zolman (1967) demonstrated that removing a chick's feathers can reduce its ability to maintain a stable body core temperature while under a cold load. The chicks feathers were removed by a topical application of a calcium thioglycolate solution. Such a nondrug approach to reducing the effectiveness of the chick's innate responses to cold could also be used to test the proposed hypothesis, possibly without the complications of the drug approach.

References

Bligh, J. (1973). Temperature regulation in mammals and other vertebrates. London: North-Holland.

Brown, P. L., & Jenkins, H. M. (1968). Autoshaping of the pigeon's key-peck. Journal of the Experimental Analysis of Behavior, 11, 1-8.

Budgell, P. (1971). Behavioral thermoregulation in the barbary dove. Animal Behavior, 19, 524-531.

Cunningham, C. L., Francisco, D. L., Kocarnik, D., & Metcalfe. J. (1984). The effects of hyperoxic incubation on learning behavior in chicks. Manuscript submitted for publication.

Fellows, B. J. (1967). Chance stimulus sequences for discrimination tasks. Psychological Bulletin, 67, 87-92.

Freeman, B. M. (1971). Body temperature and thermoregulation. In D. J. Bell & B. M. Freeman (Eds.), Physiology and Biochemistry of the Domestic Fowl: Vol. 2. (pp.1126-1145). New York: Academic Press.

Herrnstein, R. J. (1977). Doing what comes naturally: A response to Professor Skinner. American Psychologist, 32, 1013-1016.

Holland, P. C. (1977). Conditioned stimulus as a determinant of the form of the Pavlovian conditioned response. Journal of Experimental Psychology: Animal Behavior Processes, 3, 77-104.

Horowitz, K. A., Scott ,N. R., Hillman, P. E., & Van Tienhoven, A. (1978). Effects of feathers on instrumental thermoregulatory behavior in chickens. Physiology & Behavior, 21, 233-238.

Linakis, J. G., & Cunningham, C. L. (1980). The effects of embryonic exposure to alcohol on learning and inhibition in chicks. Neurobehavioral Toxicology, 2, 243-251.

Romijn, C., & Lokhorst, W. (1955). Chemical heat regulation in the chick embryo. Poultry Science, 34, 649-654.

Schmidt, I. (1976). Effect of central thermal stimulation on the thermoregulatory behavior of the pigeon. European Journal of Physiology, 363, 271-272.

Schmidt, I., & Rautenberg, W. (1975). Instrumental thermoregulatory behavior in pigeons. Journal of Comparative Physiology, 101, 225-235.

Sherry, D. F. (1981). Parental care and the development of thermoregulation in red jungle fowl. Behavior, 76, 250-279.

Terrace, H. S. (1981). Autoshaping and two-factor learning theory. In C. H. Locurto, H. S. Terrace & J. Gibbon (Eds.), Autoshaping and Conditioning Theory. (pp. 1-15). New York: Academic Press.

Wasserman, E. A. (1973). Pavlovian conditioning with heat reinforcement produces stimulus-directed pecking in chicks. Science, 181, 875-877.

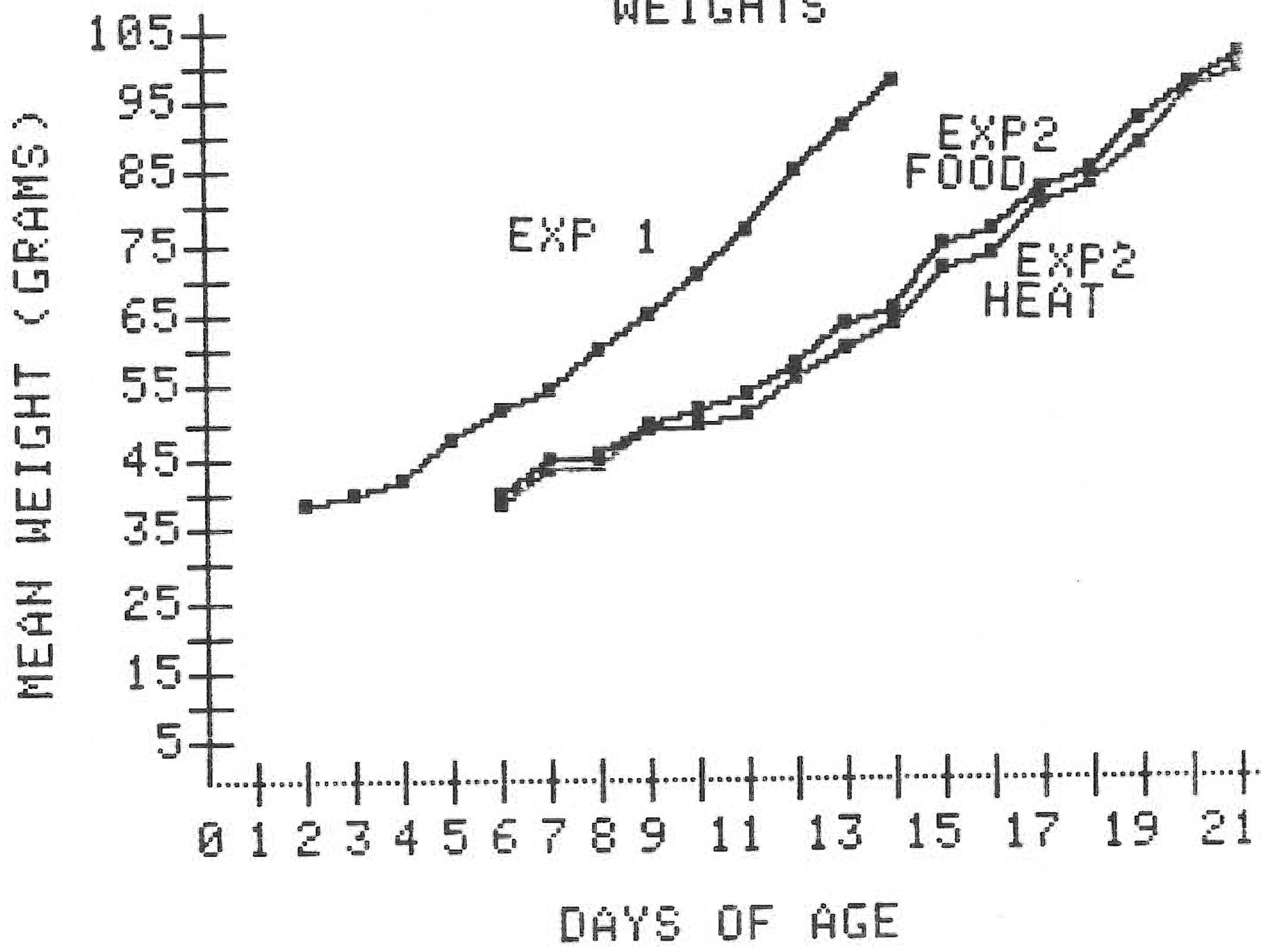
Wasserman, E. A., Hunter, N. B., Gutowski, K. A., & Bader, S. A. (1975). Autoshaping chicks with heat reinforcement: The role of stimulus-reinforcer and response-reinforcer relations. Journal of Experimental Psychology: Animal Behavior Processes, 1, 158-169.

Weiss, B., & Laties, V. G. (1961). Behavioral thermoregulation. Science, 133, 1334-1338.

Wekstein, D. R., & Zolman, J. F. (1967). Homeothermic development of the young chick. Proceedings of the Society for Experimental Biology and Medicine, 125, 294-297.

Wekstein, D. R., & Zolman, J. F. (1969). Ontogeny of heat production in chicks. Federation Proceedings, 28, 1023-1028.

Figure A1. The mean weights of the of the Heat autoshaping group in Experiment 1 over the 13 days of the Acquisition phase in this study and of the Heat and Food autoshaping groups in Experiment 2 over the 16 days of the Acquisition phase in this study.


WEIGHTS
MEAN WEIGHT (GRAMS)
105
95
85
75
65
55
45
35
25
15
5
EXP 1
EXP2
FOOD
EXP2
HEAT
0 1 2 3 4 5 6 7 8 9 11 13 15 17 19 21
DAYS OF AGE