Journal of Experimental Psychology: General
1976, Vol. 105, No. 1, 3–46

Learned Helplessness: Theory and Evidence

Steven F. Maier
University of Colorado

Martin E. P. Seligman
University of Pennsylvania

SUMMARY

In 1967, Overmier and Seligman found that dogs exposed to inescapable and unavoidable electric shocks in one situation later failed to learn to escape shock in a different situation where escape was possible. Shortly thereafter Seligman and Maier (1967) demonstrated that this effect was caused by the uncontrollability of the original shocks. In this article we review the effects of exposing organisms to aversive events which they cannot control, and we review the explanations which have been offered.

There seem to be motivational, cognitive, and emotional effects of uncontrollability. (a) Motivation. Dogs that have been exposed to inescapable shocks do not subsequently initiate escape response in the presence of shock. We review parallel phenomena in cats, fish, rats, and man. Of particular interest is the discussion of learned helplessness in rats and man. Rats are of interest because learned helplessness has been difficult to demonstrate in rats. However, we show that inescapably shocked rats do fail to learn to escape if the escape task is reasonably difficult. With regard to man, we review a variety of studies using inescapable noise and unsolvable problems as agents which produce learned helplessness effects on both instrumental and cognitive tasks. (b) Cognition. We argue that exposure to uncontrollable events interferes with the organism's tendency to perceive contingent relationships between its behavior and outcomes. Here we review a variety of studies showing such a cognitive set. (c) Emotion. We review a variety of experiments which show that uncontrollable aversive events produce greater emotional disruption than do controllable aversive events.

We have proposed an explanation for these effects, which we call the learned helplessness hypothesis. It argues that when events are uncontrollable the organism learns that its behavior and outcomes are independent, and that this learning produces the motivational, cognitive, and emotional effects of uncontrollability. We describe the learned helplessness hypothesis and research which supports it.

Finally, we describe and discuss in detail alternative hypotheses which have been offered as accounts of the learned helplessness effect. One set of hypotheses argues that organisms learn motor responses during exposure to uncontrollable shock that compete with the response required in the test task. Another explanation holds that uncontrollable shock is a severe stressor and depletes a neurochemical necessary for the mediation of movement. We examine the logical structure of these explanations and present a variety of evidence which bears on them directly.

What follows are three instances of the phenomenon to be explained:

1. When placed in a shuttle box an experimentally naive dog, at the onset of the first electric shock, runs frantically about, until it accidentally scrambles over the barrier and escapes the shock. On the next trial, the dog, running frantically, crosses the barrier more quickly than on the preceding trial. Within a few trials the animal becomes very efficient at escaping and soon learns to avoid shock altogether. After about 50 trials the dog becomes nonchalant and stands in front of the barrier. At the onset of the signal for shock, the dog leaps gracefully across and rarely gets shocked again. But dogs first given inescapable shock in a Pavlovian hammock show a strikingly different pattern. Such a dog's first reactions to shock in the shuttle box are much the same as those of a naive dog. He runs around frantically for about 30 sec, but then stops moving, lies down, and quietly whines. After 1 min. of this, shock terminates automatically. The dog fails to cross the barrier and escape from shock. On the next trial, the dog again fails to escape. At first he struggles a bit and then, after a few seconds, seems to give up and passively accept the shock. On all succeeding trials, the dog continues to fail to escape.

2. A college student is confronted with a series of 25 letter anagrams, each with the same pattern, 34251. He has a little trouble with the first one, taking about 45 sec to solve it. He solves each of the next three in about 30 sec, and now he sees the pattern. Each of the last 16 anagrams is solved immediately. In striking contrast is the college student who has first faced a series of discrimination problems which are unsolvable or a series of loud tones which are inescapable. He works hard on the first anagram, trying many rearrangements of letters, but fails to solve it in the 100 sec allowed. He fails to solve the second one also. The third anagram, a relatively easy one, he solves, after about 60 sec. He fails to solve the next eight, appearing to give up after about 60 sec with each one. He then solves six in a row, but very slowly, and finally sees the pattern. He now solves the final three immediately.

3. A naive rat is placed in a shuttle box and trained to escape from shock. Shock terminates immediately upon his running to the opposite side; he learns readily, and escapes efficiently. A second rat who had received inescapable shock earlier in another apparatus learns just as well as the first rat to escape in the shuttle box. Now, however, the contingency between crossing the shuttle box and shock termination is obfuscated; shock does not terminate immediately upon crossing, but only after 3 sec elapse. The first rat continues to escape readily, learning to bridge a 3-sec delay of reinforcement. The second rat, however, fails to respond; on other trials, he runs across during shock, but overall he shows no learning curve.

We believe these three phenomena are all instances of "learned helplessness," instances in which an organism has learned that outcomes are uncontrollable by his responses and is seriously debilitated by this knowledge. This article explores the evidence for the phenomenon of learned helplessness, and discusses a variety of theoretical interpretations. Since the phenomenon results from experience with uncontrollable outcomes, we begin by defining uncontrollability.

Uncontrollability and the Instrumental Training Space

Learning theorists have usually viewed the relations between instrumental responding and outcomes to which organisms are sensitive in terms of the conditional probability of an outcome or reinforcer following a response $p(\mathrm{RF/R})$, which can have values ranging from 0 to 1.0. At 1.0, every response produces a reinforcer or outcome (continuous reinforcement). At 0, a response never produces a reinforcer (extinction). Intermediate values represent various degrees of partial reinforcement.

The preparation of this manuscript was supported by Grant MH26827-01 to Steven F. Maier and Grant MH19604-01 to Martin E. P. Seligman.

Requests for reprints should be sent to Steven F. Maier, Department of Psychology, University of Colorado, Boulder, Colorado 80302.

One conditional probability, however, is an inadequate description of the relations between response and outcomes about which an organism may learn. Important events can sometimes occur when no specific response has been made, and it would be a woefully maladaptive organism that was insensitive to such a contingency. Rather than representing environmental contingencies as occurring along a single dimension, we think instrumental training can be better described using a two-dimensional space, as shown in Figure 1. The x-axis $p(\mathrm{RF/R})$ represents the traditional dimension, the conditional probability of an outcome following a response. Orthogonal to the conditional probability of an outcome, given a response, is the conditional probability of an outcome occurring in the absence of that response $p(\mathrm{RF/\bar{R}})$. This dimension is represented along the y-axis. We assume that organisms are sensitive to variations along *both* dimensions conjointly, and the empirical meaning of this assumption is that systematic changes in behavior should occur with systematic changes along both dimensions. There is considerable convergence of opinion and evidence among learning theorists today that organisms can indeed learn about the contingencies within this instrumental training space, including the crucial 45° line (e.g., Catania, 1971; Church, 1969;; Gibbon, Berryman, & Thompson, 1974; Maier, Seligman, & Solomon, 1969; Rescorla, 1967, 1968; Seligman, Maier, & Solomon, 1971; Wagner, 1969; Weiss, 1968). Thus an organism may learn the extent to which food occurs when it does *not* make a specific response along with learning the extent to which food occurs when it *does* make a specific response.

Consider a few examples. In Figure 1, Point *a* (1.0,0) is a case of continuous reinforcement: The subject is always reinforced for response R, and is never reinforced if it fails to make R. Point *b* (0,1.0) is a case in which the subject is never reinforced for making the designated R, and is always reinforced for refraining from R (differential reinforcement of other behavior). Consider Point *c* (.5,.2): Here the subject

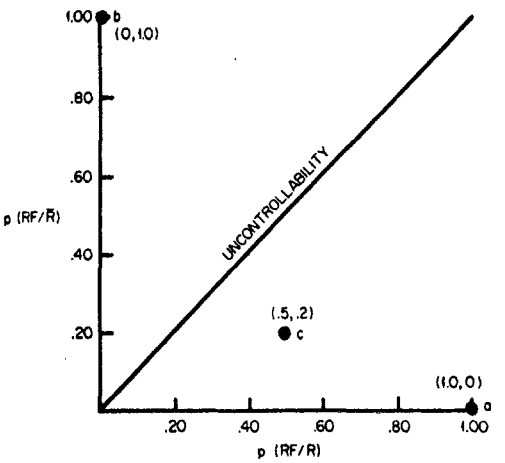

FIGURE 1. The response-reinforced contingency space. $p(\mathrm{RF/R})$ = conditional probability of an outcome following a response, $p(\mathrm{RF/\bar{R}})$ = conditional probability of an outcome occurring in the absence of that response.

is reinforced 50% of the times that it makes R, but even if it fails to make R, it is reinforced 20% of the time.

The traditional training procedures arrayed along the x-axis have been thoroughly explored by many experimenters (e.g., Ferster & Skinner, 1957; Honig, 1966). The points in the training space which do not fall along the x-axis have not, however, been systematically investigated. Consider the points that lie along the 45° line (x, y, where $x = y$). Whether or not the subject responds, the density of reinforcement is the same. The conditional probability of an outcome, given a specific response, *does not differ* from the conditional probability of reinforcement in the absence of that response. The outcome is independent of responding.

The concepts of controllability and uncontrollability are defined within this instrumental training space. Any time there is something the subject can do or refrain from doing that changes what it gets, it has control. Specifically, a response R stands in a relation of *control* to a reinforcer RF if and only if

$$p(\mathrm{RF/R}) \neq p(\mathrm{RF/\bar{R}}). \qquad (1)$$

Furthermore, when a response will not change what the subject gets, the response

and reinforcer are independent. Specifically, a response R stands in relation of independence to a reinforcer RF if and only if

$$p(\mathrm{RF/R}) = p(\mathrm{RF/\bar{R}}). \qquad (2)$$

When this is true of all emitted responses (as in Pavlovian conditioning) the subject *cannot control* the reinforcer, and the reinforcer is defined as *uncontrollable.*

How can we tell that the phenomena we will discuss result from experiencing uncontrollable outcomes as opposed to merely experiencing the outcome itself? To put it another way, how can we tell whether helplessness is a psychological phenomenon as opposed to merely being the result of physical stimulation?

There is a simple and elegant experimental design which isolates the effects of controllability from the effects of the outcome being controlled. In this "triadic" design, three groups are used: One group receives as its pretreatment an outcome that it can control by some response. A second group is "yoked"—it receives *exactly* the same physical outcome as its counterpart in the first group, but there is no response the yoked subject can make which modifies these outcomes. A third group receives no pretreatment. Later, all groups are tested on a new task.

Helplessness does not result from trauma per se: In the studies we discuss, deficits do not occur in the groups that control shock, but only in the yoked group (Hiroto & Seligman, 1975; Maier, 1970; Maier, Anderson, & Lieberman, 1972; Seligman & Beagley, 1975; Seligman & Maier, 1967).[1] The triadic design is a direct test of the hypothesis that learning that shock is uncontrollable, and not shock per se, causes helplessness. Here is an example of how the triadic design is used: Seligman and Maier (1967) used three groups of eight dogs. An escape group was trained in a hammock to turn off shock by pressing a panel with its nose. A yoked group received shocks identical in number, duration, and pattern to the shocks delivered to the escape group. The yoked group differed from the escape group only with respect to the instrumental control which it had over shock; while pressing the panel did not affect the programmed shocks in the yoked group, panel pressing terminated shock in the escape group. A naive control group received no shock in the hammock.

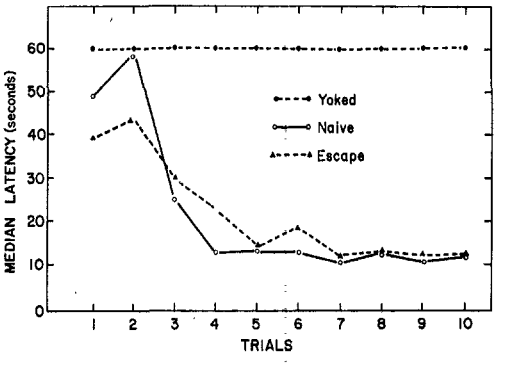

FIGURE 2. Median response latency in a shuttle box for dogs given escapable, yoked inescapable, or no shock in a harness. (The yoked group did not learn to escape.) (From "Failure to Escape Traumatic Shock" by Martin E. P. Seligman and Steven F. Maier, *Journal of Experimental Psychology,* 1967, *74,* 1–9. Copyright 1967 by the American Psychological Association. Reprinted by permission.)

Twenty-four hours after the hammock treatment, all three groups received escape/avoidance training in a shuttle box. Figure 2 shows the results of this experiment. The escape group and the naive control group performed well in the shuttle box. They jumped the barrier readily. In contrast, the yoked group was significantly slower to respond than the escape group and the naive control group. Six of the eight subjects in the yoked group failed completely to escape shock. So it was *not the shock itself,* but the inability to control the shock, that produced failure to respond.

EFFECTS OF UNCONTROLLABILITY

Having defined the objective conditions under which uncontrollability occurs and

[1] It should be mentioned that Church (1963) has argued against the use of the yoked group as a *control* group for instrumental learning. This argument is not relevant to helplessness experiments in which the yoked group is the experimental group, and the other groups are each control groups.

delineated the kind of experimental design which isolates the effects of uncontrollability from the effects of stimulation per se, we now review the deficits produced by uncontrollable outcomes. In general, when an organism experiences uncontrollable events, three deficits often ensue: motivational, cognitive, and emotional.

a) The motivation to respond in the face of later aversive events seems to wane. b) Moreover, even if the subject does respond and the response succeeds in producing relief, the subject often has difficulty learning that the response worked. c) Finally, emotional balance may be distributed; depression and anxiety, measured in a variety of ways, may predominate. The motivational deficits produced by helplessness are in many ways the most striking, so we turn to them first for review and analysis.

Motivational Deficits

Dogs. These sets of experiments form the base of the pyramid on which we construct a theory of learned helplessness, so these studies will be examined thoroughly. The behavior of dogs exposed to inescapable shock seems to typify what many species do when they are faced with uncontrollability.

Here is the typical procedure that produces learned helplessness in the dog (Overmier, 1968; Overmier & Seligman, 1967; Seligman & Groves, 1970; Seligman & Maier, 1967; Seligman, Maier, & Geer, 1968). On the first day, the subject is strapped into a hammock and given 64 inescapable electric shocks, each 5.0 sec long and of 6.0 mA (moderately painful) intensity. The shocks are not predicted by any signal and they occur randomly in time. Twenty-four hours later, the subject is given 10 trials of signalized escape/avoidance training in a two-way shuttle box. The dog must jump over the barrier from one compartment into the other to escape or avoid shock. Shocks can occur in either compartment, so there is no *place* that is always safe, but the *response* of shuttling or jumping always leads to shock termination.

The onset of a signal (light dimming) begins each trial, and the signal stays on until the trial ends. The interval between the start of the signal and the shock is 10 sec. If the dog jumps the shoulder-high barrier during this interval, the signal terminates and shock is prevented. Failure to jump during the signal-shock interval leads to a 4.5-mA shock which remains until the dog jumps the barrier. If the dog fails to jump the barrier within 60 sec after signal onset, the trial automatically ends.

Between the years 1965 and 1969 the behavior of about 150 dogs that received prior inescapable shock was studied. Of these, two thirds (about 100) did not learn to escape and went through the striking sequence of behaviors that we described. The other one third seemed completely normal. Like naive dogs, they escaped efficiently. There was no intermediate outcome. Interestingly enough, of the several hundred naive dogs who had been given shuttle box training, about 95% have been efficient responders. The other 5% failed to learn even with no prior inescapable shock. We believe that the prior history of these dogs before they arrived at the laboratory may determine whether a naive dog will show a learned helplessness effect and whether a dog given inescapable shock will be immune to this effect. When we discuss the theory and prevention of the learned helplessness effect below, we will be more explicit about how to immunize against failure to escape.

Since dogs exposed to inescapable shock seem to be physically capable of jumping the barrier, the behavioral deficit must have a psychological base. Occasionally, they jump the barrier between trials. Further, if a dog has been sitting and taking shock after shock on the left side of the box, and the door on the right side is opened at the end of the session, he will often come bounding across to escape from the box altogether

The learned helplessness effect in the dog occurs in a variety of situations and is easily produced. Within limits, it does not depend on the use of any particular shock parameters. We have varied the frequency, intensity, duration, and temporal pattern of shocks, and still produced the effect. Furthermore, it does not matter if the inescapable shock is or is not preceded by a signal. Finally, it does not matter what apparatus

the inescapable shocks are given in or where the escape/avoidance training takes place. The shuttle box and hammock are interchangeable. If the dog is given inescapable shock in the shuttle box and then required to press panels with its head to escape in the hammock, he is still helpless. Further, after uncontrollable shock, dogs are not only debilitated at fleeing from the shock itself (escape), but they also seem to be unable to prevent or "avoid" shock. Overmier (1968) gave dogs inescapable shock in the hammock and then tested them in a shuttle box. If the dog jumped after the signal went on, but before the shock came on, he could avoid the shock. But no escape was allowed, for if the dog failed to jump in the signal-shock interval, the barrier was closed and inescapable shock occurred. The helpless dogs failed to avoid, just as they had failed to escape. So, dogs previously exposed to inescapable shocks failed to cope adaptively with signals for shock as well as with shock.

Debilitation of response initiation as a consequence of uncontrollable outcomes has been reported in cats, rats, mice, birds, primates, fish, and man, as well as in dogs. The learned helplessness effect seems rather general among species that learn.

Cats. Thomas and Balter (in press) reported an effect in cats which seems identical to learned helplessness in dogs. (See also Masserman, 1943, 1971; Seward & Humphrey, 1967; Zielinski & Soltysik, 1964, for other reports of debilitation in cats caused by inescapable shock). They designed a hammock for cats and gave them inescapable shock. When later placed in a cat shuttle box, those cats failed to escape. Like dogs, they sat and took the shock. Thomas and Balter also reported that blocking activity of the septal area of the brain breaks up helplessness, and directly stimulating the septum electrically produces a learned helplessness-like effect. When we discuss the theory and therapy for helplessness, we will examine these physiological data in more detail.

Fish. Following inescapable shock, fish also show poor escape and avoidance responding. Padilla, Padilla, Ketterer, and Giacolone (1970) gave inescapable shock to goldfish and then tested them in an aquatic shuttle box. These fish were slower to avoid than naive controls. (See Behrend & Bitterman, 1963; Bintz, 1971; Frumkin & Brookshire, 1969; Padilla, 1973, for related goldfish data.)

Rats. Until recently, it has proven difficult to produce a learned helplessness effect in rats. A substantial number of experiments were performed involving inescapable shock; by and large, however, these showed rather small, if any, effects on later response initiation. (Maier et al., 1969, and Seligman et al., 1971, reviewed the complicated literature, and the interested reader is referred there for details. See also Anderson, Cole, & McVaugh, 1968; de Toledo & Black, 1967; Dinsmoor & Campbell, 1965a, 1965b; Looney & Cohen, 1972; Mullin & Mogenson, 1963; Weiss, Krieckhaus, & Conte, 1968, for representative experimental studies.) Unlike dogs, a rat given prior inescapable shock was typically only a bit slower to escape shock on the first few trials, or slower to acquire avoidance—it did not fail to learn.

After intensive experimentation, however, several investigators have now independently produced substantial learned helplessness effects in rats. (Maier, Albin, & Testa, 1973; Maier & Testa, 1975; Seligman & Beagley, 1975; Seligman, Rosellini, & Kozak, 1975.)[2] In doing so, one crucial factor emerged—the response used in the test for learned helplessness must be difficult, and not something the rat does very readily. So, for example, if rats are first exposed to inescapable shock and then tested on a simple escape response like pressing a bar *once* (FR-1), or fleeing to the other side of a shuttle box, no deficits are found. If, however, the response requirement is increased—so the bar must be pressed three times in order for shock to end (FR-3 lever pressing) or the rat has to run across the shuttle box and back (FR-2 shuttling)—then the rat that has experienced inescapable shock fails to escape. In con-

[2] In passing it should be mentioned that mice have been reported to show response deficits following inescapable shock (Braud, Wepman, & Russo, 1969).

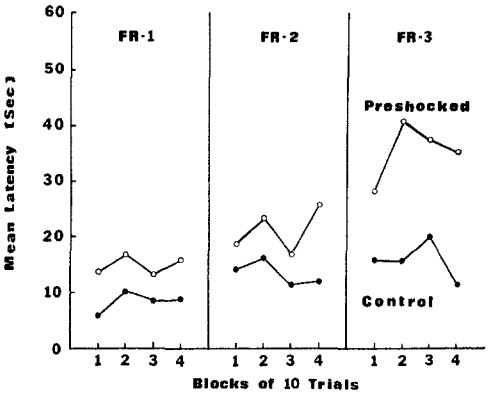

FIGURE 3. Mean latency to press a lever to escape shock in an FR-1, FR-2, and FR-3 schedule, for rats given inescapable or no shocks. (The yoked subjects escaped very poorly only on the FR-3.) (From "Learned Helplessness in the Rat" by Martin E. P. Seligman and Gwyneth Beagley, *Journal of Comparative and Physiological Psychology,* 1975, *88,* 534–541. Copyright 1975 by the American Psychological Association. Reprinted by permission.)

trast, rats that have had prior escapable shock or no shock learn even more difficult responses without difficulty (see Figure 3).

Hannum, Rosellini, & Seligman (in press) recently extended these findings to development. Three groups of rats received four sessions of escapable, inescapable, or no shock shortly after they were weaned. At 90 days of age they were tested on an FR-3 lever press escape task. Rats that had received inescapable shock at weaning failed to escape shock. Rats that had received escapable shock or no shock escaped well (see Figure 4).

Man. What are the laboratory effects of inescapable trauma in man? Like the dog, cat, rat, and fish, when a human being is faced with noxious events that it cannot control, its motivation to respond seems to be reduced.

Hiroto's study (1974) is representative and it replicated in college students the findings on dogs. In a triadic design, subjects in his escape group received loud noise which they learned to turn off by button pushing. The subjects in the inescapable group received the same noise, but the noise was independent of their responding. A third group received no noise. All the groups were then taken to a hand shuttle box; in order to escape noise the individual had only to move his hand from one side to the other. Both the no-noise and escape group learned readily to shuttle with their hands. Like other species, however, the human inescapable group failed to escape and avoid; rather, most sat passively and took the aversive noise. (For other learned helplessness experiments in man, see Fosco & Geer, 1971; Glass & Singer, 1972; Hiroto & Seligman, 1975; Klein, Fencil-Morse, & Seligman, in press, Krantz, Glass, & Snyder, 1974; Miller & Seligman, 1975a, Note 1; Racinskas, 1971; Rodin, 1975, Roth, 1973; Roth & Bootzin, 1974; Roth & Kubal 1975; Thornton & Jacobs, 1971.)

Hiroto's design was actually more complicated and had two other important factors. Half the subjects in each of the three groups were told that how they did in the shuttle box was a test of skill; the other half were told that how they did was governed by chance. Those who received chance instructions tended to respond poorly in all groups. Finally, the personality dimension of "external vs. internal locus of control of reinforcement" was also varied in Hiroto's design (see Lefcourt, 1966; Rotter, 1966; James, Note 2), with half of all the students in each group being "externals" and half

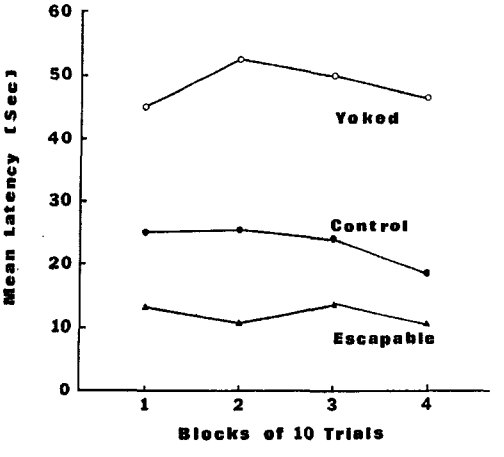

FIGURE 4. Mean escape response latency for rats given either escapable, yoked inescapable, or no shocks as infants (Hannum, Rosellini, & Seligman, in press). The rats given inescapable shocks as infants did not learn to escape.)

"internals." An external is a person who believes, as shown by his answers on a personality inventory, that reinforcements occur in his life by chance or luck and are beyond his control. An internal believes that *he* controls his own reinforcers and that skill will out. Hiroto found that externals became helpless in his experiment more easily than did internals (see also Dweck, 1975). So three independent factors, the laboratory procedure of uncontrollability, the cognitive set induced by chance instructions, and externality, each produced a learned helplessness effect. Given this convergence, Hiroto concluded that these three factors all erode motivation to escape by contributing to the expectation that responding and relief are independent.

This concludes the survey of motivational deficits produced by exposure to uncontrollable aversive events in different species. It seems to be generally true that uncontrollability produces deterioration in the readiness of dogs, cats, rats, fish, monkeys, and men to respond actively to trauma.

Generality. Does uncontrollability produce a habit limited to situations very like the ones in which uncontrollability is experienced, or does it produce a more general effect? The question we are really asking here is "Is helplessness just an isolated set of habits or does it involve a more basic change?" We believe that what is learned when the environment is uncontrollable can have consequences for a wide range of behavior.

At the lowest level of generality, helplessness transfers from some types of apparatus to others, as long as shock occurs in both situations; dogs given inescapable shock in a hammock fail to escape later in a shuttle box. But does what is learned transfer to aversive situations not involving shock? Braud et al. (1969) used a triadic design with mice. One group could escape shock by climbing up a pole, a second group was yoked, and a third group received no shock. All groups were then placed in an alley flooded with water and had to swim out in order to escape. The yoked group was poorest at escaping from water. (See McCulloch & Bruner, 1939, for similar findings

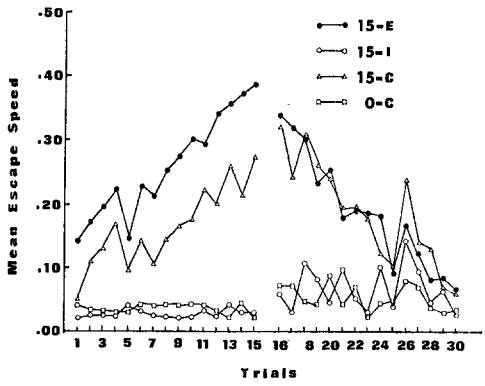

FIGURE 5. Mean speed to escape a nonrewarded goalbox. (The group designated by E previously received escapable shocks, the group designated by I received inescapable shocks, and the groups designated by C received no shocks. The groups designated by 15 received rewarded runway acquisition training, whereas the group designated by 0 did not. The rats given inescapable shocks [15-I] showed no tendency to escape the frustrating goalbox.) (From "Frustration and Learned Helplessness" by Robert A. Rosellini and Martin E. P. Seligman, *Journal of Experimental Psychology: Animal Behavior Processes,* 1975, *1,* 149–157. Copyright 1975 by the American Psychological Association. Reprinted by permission.)

in the rat, also for the earliest apparent learned helplessness study in the literature.) Rosellini and Seligman (1975) performed an experiment in which shock-induced learned helplessness may have transferred to frustration. Three groups of rats received escapable shock, inescapable shock, or no shock. After this pretreatment, they learned to run down an alleyway in order to get food in a goal box. Food was present on every trial in the goal box. Once they had learned, food was no longer placed in the goal box. So during this extinction procedure, the rats ran down the alleyway into the goal box where they expected food, but found none. This has been shown to be a "frustrating" and aversive experience for a rat (Amsel, Rashotte, & MacKinnon, 1966). The rats were then given an opportunity to jump out of the goal box and escape this frustration. Rats that had experienced escapable shock or no shock escaped the goal box readily. Rats that had experienced inescapable shock did not escape from the goal box. (See Figure 5.) The effect of ex-

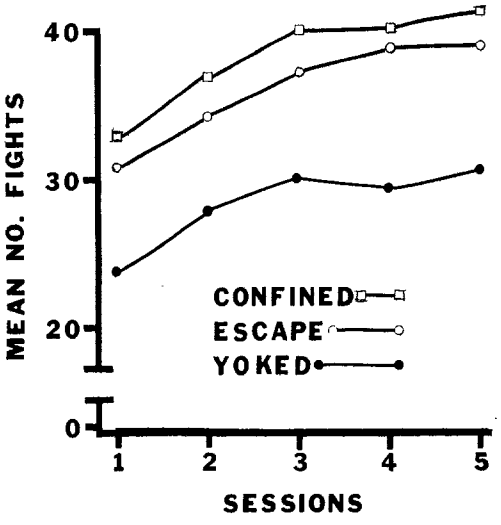

FIGURE 6. Shock-induced fighting frequency, for each of five test sessions, for rats that had received escapable, yoked inescapable, or no shocks. (Inescapable shocks depressed fighting.) (From "Influence of Control of Shock on Subsequent Shock-Elicited Aggression" by Steven F. Maier, Christine Anderson, and David A. Lieberman, *Journal of Comparative and Physiological Psychology*, 1972, *81*, 94–100. Copyright 1972 by the American Psychological Association. Reprinted by permission.)

posure to one kind of uncontrollable event generalized to another—frustration.

Another possible instance of the transfer of a learned helplessness effect is related to "shock-elicited aggression." If two rats are placed in a small enclosure and repeatedly shocked, the shocks frequently come to elicit attack and aggressive posturing. Maier et al., (1972) gave rats escapable shock, inescapable shock, or no shock, and then placed these rats in a shock-elicited aggression situation. As shown in Figure 6, the amount of shock-elicited aggression was lower in the rats previously exposed to inescapable shock than it was in the rats given escapable or no shock (also see Payne, Anderson, & Murcurio, 1970; Powell & Creer, 1969). In a related study (unpublished data) we found that dogs that had received inescapable shock as puppies lost out in competition for food (only one nose fits into a coffee cup full of Alpo) with dogs that received no shock or escapable shock. So it seems that exposure to uncontrollable aversive events retards the initiation of aggressive as well as defensive responses. (See Brookshire, Littman, & Stewart, 1961, Experiment 6; and Anderson & Paden, 1966, for other evidence on intra-aversive transfer.)

Does experiencing uncontrollable aversive events have effects on nonaversively motivated behavior? Recently, Hiroto and Seligman (1975) and Miller and Seligman (1975) systematically explored the transfer of helplessness between instrumental tasks and cognitive tasks. Three groups of college students received escapable, inescapable, or no loud noise. They were then switched to a nonaversive anagrams test, and time to solve a series of 20 anagrams like IATOP was recorded. Students who had received inescapable loud noise failed to find the solutions more often than the escape group and the no-noise group. So exposure to uncontrollable aversive events retards solution of cognitive problems in a nonaversive situation.

Are the debilitating effects of uncontrollability only produced by uncontrollable *trauma?* What happens to response initiation when preceded by a history of uncontrollable outcomes which are not traumatic? Hiroto and Seligman (1975) tried to produce a learned helplessness effect using unsolvable discrimination problems. Just as a solvable discrimination problem is controllable in the same sense that an escapable shock is controllable, an unsolvable discrimination problem is uncontrollable in the same sense that an inescapable shock is uncontrollable. The conditional probability of success given any response (e.g., to the left) is the same as the probability of success if the alternate response (e.g., to the right) is made.

With the formal similarity of unsolvability and inescapability in mind, Hiroto and Seligman (1975) and Klein et al. (in press) gave three groups of college students four sets of solvable, unsolvable, or no-discrimination problems. Then all groups were given the hand shuttle box with loud noise to be escaped. Individuals who had solvable discrimination problems or no prior problems escaped noise readily. The unsolvable group failed to escape the noise. So

we believe that response initiation to control noxious events may be impaired by experience with uncontrollable reward.

Equally intriguing is the possibility that delivery of appetitive events independent of behavior may produce a failure to learn responses to procure such events, an effect analogous to learned helplessness. Engberg, Hansen, Welker, and Thomas (1973) trained one group of pigeons to press a treadle to obtain food. A second group of pigeons received food delivered independently of behavior, and a third group received only a small amount of magazine training. Following this pretreatment, all pigeons were tested with a key peck autoshaping procedure. In autoshaping (Brown & Jenkins, 1968) the pigeon key is periodically illuminated for a brief period of time, and food is presented at the end of each key illumination period whether or not a peck has occurred. Pigeons normally acquire key pecking under such an arrangement. The result was that the group given previous treadle press training was fastest to acquire key pecking and the group given noncontingent food was the slowest to acquire key pecking. Engberg et al. interpreted their results as constituting an appetitive analogue to learned helplessness; food was delivered independently of behavior, and this retarded the acquisition of a response to procure food.

However, the Engberg et al. experiment has been criticized on a number of grounds (Gamzu, Williams, & Schwartz, 1973). First, Gamzu et al. argued that autoshaping is not a reasonable task to use in attempting to establish an appetitive analogue to learned helplessness because autoshaped key pecks are largely controlled by Pavlovian contingencies (Moore, 1973) rather than instrumental contingencies; learned helplessness is a debilitation of instrumental responding. Second, Gamzu et al. noted that it is easy to account for the Engberg et al. experiment in terms of competing motor responses. Both the group given treadle training and the group given noncontingent food would have been expected to learn a motor response incompatible with key pecking (treadle responding and standing by the grain hopper, respectively). However, Engberg et al. removed the treadle during the autoshaping test, and so it could be argued that the stimulus controlling the competing behavior was removed for the treadle group. The stimulus controlling any competing behavior was not removed for the noncontingent food group, and this difference could account for the results. In response to these criticisms Welker (1974) conducted a further investigation of the phenomenon. One group of pigeons was trained to peck a key for food, a second was given noncontingent food, and a third was given only a small amount of magazine training. All pigeons were then given a test in which treadle pressing produced food on a continuous reinforcement schedule. The key was present during the treadle test, so it could not be argued that the stimulus controlling the competing behavior was removed for the contingent food group. The results were the same as in Engberg et al.; noncontingent food presentation retarded acquisition. Taking a hint from the helplessness experiments in rats (see pp. 8–9), Welker repeated his experiment with an FR-3 treadle response as the test task. That is, the treadle had to be pressed three times in order to produce food. This magnified the size of the observed effect.

A potentially related finding was recently reported by Bainbridge (1973). At 50 days of age, rats were given a solvable black–white discrimination problem, an unsolvable problem, or no problem. Twenty days later they were tested with the same tasks but different stimuli, or in a Hebb–Williams maze based on spatial cues. The rats given an initial unsolvable problem performed very poorly in both the same and the different apparatus. Thus exposure to appetitive events delivered independently of behavior may interfere with the acquisition of appetitively motivated responses, just as exposure to noncontingent shock interferes with the acquisition of shock-motivated responses.

So we believe that the psychological state produced by uncontrollability may undermine response initiation quite generally. Dogs, rats, cats, fish, and people make fewer responses to escape shock after receiving

uncontrollable shock. Furthermore, these motivational deficits may not be limited to shock or even noxious events. Escape from frustration, aggressive behavior, and even the propensity to solve anagrams might be undermined by inescapable aversive events. Conversely, uncontrollable rewards disrupt escape from loud noise, learning to procure food, and competitiveness. However, it should be stressed that many of the experiments cited here are subject to alternative interpretation, and much more research will be required before the true extent of these effects will be known. Clearly, there must be a limit to the degree of generalization of learned helplessness effects, and these limits must be explored and delineated. We cannot yet say what factors will prove important. All that can be said now is that there appears to be some degree of transfer.

Cognitive Deficits

We have argued that a major consequence of experience with uncontrollable events is *motivational;* uncontrollable events undermine the motivation to initiate voluntary responses to control future events. Experience with uncontrollable events may also have a second consequence that is cognitive; experience with uncontrollability may produce a difficulty in learning that responses have succeeded, even when responding is actually successful. Uncontrollability may retard the perception of control.

This phenomenon seems to appear in helpless dogs, rats, and men. Occasionally, a naive dog sits and takes shock on the first three or four trials in the shuttle box, and then on the next trial jumps the barrier and escapes shock successfully for the first time. Once a *naive* dog makes one response that produces shock termination, it learns rapidly. On all further trials, the subject responds vigorously and goes on to learn to avoid shock altogether. But dogs that first received inescapable shock were different in this respect also. About one third of them go through a similar pattern—sitting through shock on the first three or four trials and then escaping successfully on the next. These dogs, however, then revert to taking the shock, and they fail to escape on future trials. It appears as if one success is just not enough to make a dog that has experienced inescapable shock learn that his responding now produces shock termination.

Miller and Seligman (1975) and Klein et al. (in press) found that such a negative cognitive set results from uncontrollability in man. Three groups of students received escapable, inescapable, or no loud noise. Then they confronted two new tasks, a task of skill and a task of chance. On each of 10 trials in the skill task, they sorted 15 cards into 10 categories of shape, attempting to complete it within 15 sec. Unknown to them, the experimenter arranged to have them succeed or fail on any given trial by saying that time was up before they had finished or after. So they went through a prearranged run of successes and failures. At the end of each trial, the subjects rated (on a 0–10 scale) what they thought their chances of succeeding on the next trial would be. Subjects who were previously exposed to inescapable noise showed very little change in their expectancy for successes after each new success and failure. They had difficulty perceiving that their response would affect succeeding or failing. Control subjects and subjects who had escaped noise showed large expectancy changes following each success and failure. This showed that they believed outcomes to be dependent on their actions. The three groups did not differ in expectancy changes following success and failure in a "chance" task that they perceived as a guessing game. So inescapable loud noise produced a cognitive set in which people believed that success and failure is independent of their own skilled actions within the experimental situation, and they therefore had difficulty perceiving that skilled responses were effective.

Hiroto and Seligman (1975), Miller and Seligman (Note 1) and Klein et al. (in press) also reported this negative cognitive set in another form. After escapable, inescapable, or no noise, as the reader will recall, students had to solve anagrams (see p. 11). Two kinds of deficits emerged. Inescapable noise interfered with their ability to solve any given anagram. In addition, there was a pattern to the 20 ana-

TABLE 1

MEAN NUMBER AND STANDARD DEVIATION OF CONSECUTIVE SUCCESSES BEFORE PATTERN SOLUTION

Subject	Noise		
	Inescapable	Escapable	None
Depressed			
M	5.9	4.2	7.1
SD	2.53	1.36	1.22
Nondepressed			
M	6.9	4.5	3.7
SD	3.36	1.58	.86

Note. Adapted from Miller and Seligman (Note 1). Inescapable noise increased the number of successes required in nondepressed subjects.

grams to be solved; each was arranged with its letters in this order: 53124, for example, ISOEN, DERRO, OURPG, etc. As can be seen in Table 1, students who had received inescapable noise required about seven consecutive successes before catching on to the pattern; students who received escapable noise or no noise needed about three consecutive successes. Unsolvable discrimination problems, incidentally, produced the same disruption of anagram solution as did inescapable noise.

The existence of a negative cognitive set produced by independence between responding and outcomes bears on an important issue in learning theory. When two events are presented independently of each other, for example, a tone and a shock presented at random, does the subject learn anything at all about the tone or does he merely come to ignore it? A helplessness point of view holds that men and animals actively learn that responses and outcomes are independent of each other, and one way the learning is manifested is by the difficulty they later have learning that the response produces the outcome when it does. Merely substitute *shock* for *outcome* and *tone* for *response*. This suggests that organisms should also actively learn when a tone and shock are independent and that organisms show this by having trouble learning later that the tone is followed by the shock when it actually is. Rescorla (1967) held the contrary view. Independence between a tone and shock is a neutral condition in which nothing is learned. Recent investigation has shown that active learning does indeed occur when conditioned stimuli (CSs) and unconditioned stimuli (USs) are independently presented. Mellgren and Ost (1971) reported that a group for which CSs were presented independently of food took longer to learn later that the CSs were associated with the food than did naive rats (or even rats for whom the CSs predicted the *opposite* relationship with food). Kemler and Shepp (1971) showed that irrelevant stimuli were most slowly learned about when they became the relevant stimuli for the solution of a discrimination problem for children. Thomas, Freeman, Svinicki, Burr, and Lyons (1970) showed that pigeons that had two colors presented independently of food tended not to discriminate later between two line tilts, one of which predicted the presence of food and the other its absence. MacKintosh (1973) also reported retardation of conditioning by prior CS–US independence.

So independence between two stimuli may produce active learning, and this learning seems to retard the ability of rats, pigeons, and men to learn later that the stimuli depend on one another. This evidence coheres with the effects of response-outcome independence on cognition and bolsters the hypothesis that such independence retards the ability of the organism to perceive that his responding has contingent consequences (also see the discussion of experiments by Maier & Testa, 1975, pp. 24–25).

Emotional Deficits

Our first hint that uncontrollable aversive events had emotional as well as motivational and cognitive consequences came when we found that the motivational effects dissipated in time under some circumstances. Overmier and Seligman (1967) found that after one session of inescapable shock in the hammock, dogs were helpless in the shuttle box 24 hours later. If, however, the dogs were not tested in 24 hours, but at 48, 72, or 168 hours after inescapable shock, they were normal. This is the only circumstance we know under which helplessness dissipates in time. If multiple sessions of inescapable

shock are given to the dog in the hammock, he will fail to escape a week later in the shuttle box (Seligman & Groves, 1970). Further, if the dog is reared from birth in laboratory cages, having minimal experience with control over events, small amounts of inescapable shock produce nontransient helplessness (Seligman & Groves, 1970). Finally, no time course has been observed in rats; following one session of inescapable shock, rats fail to escape 5 minutes, 1 hour, 24 hours, and 168 hours later. It must be borne in mind however, that under one circumstance—a single session of inescapable shock in dogs of unknown past history—helplessness dissipates in time, and such a time course hints of a transient emotional disturbance.

Other evidence indicates the emotional consequences of uncontrollability. One widely quoted study (Brady, Porter, Conrad, & Mason, 1958) is related to uncontrollability and helplessness, but appears to show *less* emotionality with uncontrollability. Two groups of four monkeys were given shocks; half of them—the "executives"—had control over the shock and could avoid them by bar pressing. The other four were yoked, or helpless, since they could not modify shock. The executives formed stomach ulcers and died, but the helpless monkeys did not. Unfortunately, this result may be an artifact of the way the monkeys were assigned to the two groups. All eight monkeys were placed on the executive schedule originally, and the first four to start the lever pressing became the executives. The last four became the yoked subjects. It is possible that the more emotional a monkey is, the more quickly it begins to bar press when it is shocked (Sines, Cleeland, & Adkins, 1963). So the four most emotional animals may have become the executives, and the four least emotional may have become the yoked subjects.

Jay Weiss (1968, 1971a, 1971b, 1971c) has recently repeated the executive monkey design with rats. (See also Moot, Ceballa, & Crabtree, 1970.) Three groups of rats were randomly assigned to the triadic design. The executive animals showed fewer and less severe ulcers than the yoked animals. Moreover, the yoked rats lost more weight, defecated more, and drank less than the executive. So rats given inescapable shock show more stress or emotional effects when measured by stomach ulcers than rats who can control shock.

There is further evidence in rats that uncontrollable shock produces more severe emotional reactions than does controllable shock. Mowrer and Viek (1948) shocked two groups of rats while the rats were eating. One group could control the shock by jumping in the air, and the other group received uncontrollable shock. The rats getting uncontrollable shock subsequently ate less than those controlling shock (see Brimer & Kamin, 1963; Desiderato & Newman, 1971; Lindner, 1968; and Payne, 1972, for an ongoing controversy surrounding these data). In an analogous human study by Hokanson, DeGood, Forrest, and Brittain (1971), subjects performed a symbol matching task while being shocked. The schedules were individually arranged so that each subject received an average of one shock every 45 sec. Subjects in the controllability group were allowed to take as many time-outs as they wished whenever they wanted. A yoked control group received the same number of time-outs at the same times. Measures of blood pressure taken at 30-sec intervals indicated that yoked subjects showed consistently higher blood pressures. (See Averill & Rosenn, 1972; Bandler, Madaras, & Bem, 1968; Corah & Boffa, 1970; Elliot, 1969; and Stotland & Blumenthal, 1964, for related studies in humans using a variety of other measures of emotional arousal. This is a complex and inconsistent literature and is reviewed by Averill, 1973.)

Hearst (1965) found that the presentation of uncontrollable shocks resulted in a breakdown of a well-trained appetitive discrimination in rats. During uncontrollable shock his rats no longer discriminated between two stimuli, one of which signaled the presence and the other the absence of food. During controllable shock the appetitive discrimination was maintained.

Such breakdown of appetitive discriminations is reminiscent of the classic work on "experimental neurosis." The concept of

experimental neurosis is not a homogeneous one; neither is it well defined. Unlike the other studies reviewed, controllability has not been manipulated explicitly to produce the "neurosis." Yet from an operational point of view, we can speculate that the lack or loss of control may be important in their etiology. Typically, an animal is restrained in some type of harness seriously limiting what it can do. Often the procedure is Pavlovian, and by definition the organism has no control over the onset or the offset of the stimuli presented. In Shenger-Krestnikova's (in Pavlov, 1927) classic experiment, an appetitive discrimination deteriorated and signs of distress were noted when the dog could no longer tell the difference between the rewarded and nonrewarded stimulus. In the work of Liddell, James, and Anderson (1934), sheep developed a range of maladaptive behaviors following uncontrollable electric shock. Masserman (1943) trained monkeys to feed in response to a signal and made them "neurotic" by presenting a fear-arousing stimulus during feeding. Without "therapy" these monkeys remained disturbed almost indefinitely. According to Masserman (1943):

> Markedly different, however, was the case of animals that had been taught to manipulate various devices that actuated the signals and feeder because in this way they could exert at least partial control over their environment. This stood them in good stead even after they were made neurotic in as much as when their hunger increased they gradually made hesitant, but spontaneous, attempts to reexplore the operation of the switches, signals and food boxes, and were bolder and more successful as food began to reappear. (p. 82)

In a dramatic primate study, Stroebel (1969) trained a group of rhesus monkeys to air-condition their overheated chamber by pressing a lever, and also to control loud noise, annoying light, and mild shocks by pressing the same lever. He then retracted the lever so that it could still be seen, but could no longer be pressed. *No further physical stressors were presented.* Initial responding was frantic. Brain temperature became irregular and the circadian rhythm was disrupted.

> As rhythm disturbance developed, members of this . . . group of subjects began to show lassitude and weakness; their fur became knotted, mottled and poorly groomed; behaviorally they performed unpredictably if at all on the right hand lever problems, pausing often for naps and rest. The behaviors exhibited by these animals were clearly nonadaptive in nature; for example, two subjects spent hours in catching "imaginary" flying insects, one subject masturbated almost continuously, three subjects became almost compulsive hair pullers, and all tended to show movement stereotypy alternating with an almost total lack of interest in their external environment. (Stroebel, 1969, p. 97)

It is not clear whether there can be any one theory which can account for "experimental neuroses," nor is it clear whether all these phenomena are closely related. But uncontrollability is prominently present, and emotional disruption is the frequent result.

In summary, three types of disruption seem to be caused by uncontrollability of aversive events in the laboratory: The motivation to respond is reduced, the propensity to perceive success is undermined, and emotionality is modified. These effects hold across a variety of situations and species. We need a theory to put all this together and we will now propose one.

Learned Helplessness Theory

The basic facts about learned helplessness effects in the laboratory have now been arrayed before the reader. What must an adequate theory of helplessness accomplish? It must account for the three facets of the effects of uncontrollable aversive events: motivational, cognitive, and emotional. It must be testable; there should be experiments that can be performed which would confirm it if it is true, or disconfirm it if it is false. Finally, it must be applicable outside the laboratory—it must be useful in explaining any helplessness effects found in the real world. This final requirement is beyond the scope of this article, but the interested reader should see Seligman (1975).

The theory to be presented is straightforward, and the groundwork for it has been prepared by the way we laid out the data above. It accounts directly for the motivational and cognitive deficits and, with an additional premise, can account for the emotional disturbance. The theory has been tested in several ways, and as a bonus it

predicts ways to eliminate helplessness effects and ways to prevent them.

When an organism is faced with an outcome that is independent of his responses, he sometimes learns that the outcome is independent of his responses.

This is the cornerstone of our view and probably seems obvious to all but the most sophisticated learning theorist. You will recall our discussion of the response contingency space (Figure 1). Learning theorists would much prefer that the kinds of contingencies that can be learned about be as simple as possible. First they believed that the most that could be learned about was a simple pairing of a response and an outcome, and the pairing of the response with the absence of the outcome. But this had to be broadened to include partial reinforcement, with the subject integrating both kinds of pairings. So what could be learned about was broadened to the probability of an outcome given a response. Then, it was shown that organisms could also learn about the probability of an outcome given that it did not make the indicated response. The added step which our view makes is that the organism can learn about both these probabilities conjointly, that variation of experience corresponding to the points in the response contingency space will produce systematic particular, we have argued that exposing changes in behavior and cognition. In organisms to the 45° line, in which the probability of the outcome is the same whether or not the response of interest occurs, produces learning. Behaviorally, this learning should tend to produce lack of response initiation to control the outcome; cognitively it should produce a belief in the inefficacy of responding and difficulty in learning that responding succeeds: and emotionally when the outcome is traumatic it might produce emotional changes.

The basic triadic design employed in all the helplessness studies reviewed above is, of course, directly relevant to the premise that men and animals learn about, and form expectations concerning, independence between outcome and responses. So, for example, in the Seligman and Maier (1967) study only the yoked dogs later failed to escape shock, while the dogs who could escape by panel pressing and the dogs who were not shocked did not fail to respond later. Clearly something different happened to the dogs that received shock independently of their responses. We believe they learned that responding was futile and therefore expected future responding to shock to be futile. We have gone to pains to give an *objective* definition of *uncontrollability* and *response–outcome independence*. This is because the theory is basically a three-step affair.

Information about contingency →
Cognitive representation of the contingency
(learning, expectation, perception, belief) →
Behavior

An organism must begin with the information about the contingency between response and outcome. This information is a property of the world out there, a set of stimuli, and not a property of the receiver. We have carefully defined the kind of information that can be called information that a response and an outcome are independent.

But the middle step in the chain is crucial and is easily overlooked. The information about the contingency must be processed and transformed into a cognitive representation of the contingency.[3] Such a representation has been variously called "learning that outcome and response are independent," "perceiving that response and outcome are independent," or "believing that response and outcome are independent." We prefer to call the representation "the expectation that responding and an outcome are independent."

A person or animal can be exposed to the environmental contingency in which an outcome response are independent, yet not form such an expectation. Immunization, as we shall see later in this article (refer to p. 25), is an example. Conversely, a per-

[3] For attempts to spell out in detail the relationship between the contingency information and its cognitive representation, the interested reader should consult, for example, Kelly (1967, 1972), Weiner, Frieze, Kukla, Reed, Rest, and Rosenbaum (1971) from an attribution theory point of view; Irwin (1971) and Seligman and Johnston (1973) from a cognitive-learning theory point of view; as well as Lazarus (1966) and Stotland (1969).

son can show a helplessness effect without being exposed to the contingency as such; he can merely be told that events are uncontrollable.

Glass and Singer (1972) have done an extensive series of studies on the role of controllability in reducing stress and have found that merely telling a human subject about controllability duplicates the effects of actual controllability. They attempted to mimic the stress of the urban environment by having their subjects (college students) listen to a very loud melange of sound: two people speaking Spanish, one person speaking Armenian, a mimeograph machine, a calculator, and a typewriter. When subjects could *actually* turn off the noise by button pushing, they were more presistent in problem solving, they found the noise less irritating, and they did better at proofreading than yoked subjects.

Glass and Singer also presented another group of subjects with the same noise, but this time it was uncontrollable. However, this group had a panic button and was told: "You can terminate the noise by pressing the button. But we'd prefer you not do it." None of the subjects in fact tried to turn off the noise. All they had was the false information that they could control the noise if they had to. These subjects did just as well as the subjects who actually controlled the noise. Actual controllability and actual uncontrollability may often produce corresponding expectations. But this type of experiment, in which the expectation is invalid, highlights the fact that it is the expectation and not the objective conditions of controllability that is the cornerstone of our theory. (See also Geer, Davison, & Gatchel, 1970; Geer & Maisel, 1972; Klein et al., in press; Langer, 1974; Pervin, 1963; and Stotland & Blumenthal, 1964, for related effects of perceived control.)

So the first step of the theory is that the organism acquires an expectation of response-outcome independence, when outcomes are uncontrollable. The second step in the theory is the *means* by which the expectation of response–outcome independence produces the effects associated with helplessness.

We assume that the incentive to initiate voluntary responses in a traumatic situation is partly produced *by the expectation that responding produces relief. In the absence of this incentive, voluntary responding will decrease in likelihood. When a person or animal has learned that relief is independent of responding, the expectation that responding produces relief is negated, and therefore response initiation is reduced.* Most generally put, the incentive to initiate voluntary responses to control any outcome (e.g., food, sex, shock termination) comes, in part, from the expectation that responding produces that outcome. (See Bolles, 1972, for a discussion of expectational mechanisms and incentive motivation.)[4] In the absence of this incentive, voluntary responding will decrease in likelihood. When a person or animal has learned the outcome is independent of responding, the expectation that responding will produce the outcome, and therefore response initiation, decreases.

The way in which this undermining of motivation works has been seen with crystal clarity in a human helplessness experiment (Thornton & Jacobs, 1971). Following inescapable shock, college students sat and took the shock. When asked why they did not respond appropriately, *60% of the subjects reported they had no control over shocks, so why try.* These subjective reports suggest that a belief in uncontrollability undermines the incentive to initiate responses.

We also assume that *learning that an outcome is independent of a response makes it more difficult later to learn that responses produce that outcome.*

Response–outcome independence is an active form of learning, and like any other active form of learning, it can proactively interfere with contravening forms of learning through an associative interference. At

[4] It should also be mentioned that innately elicited struggling is another source of responding in a traumatic situation, but it is the waxing and waning of *voluntary* (outcome-sensitive) responses which is our concern here. This does not deny that innate responses can be transformed into voluntary responses (Schwartz & Williams, 1972).

first the dog is not a passive recipient of shock but emits many responses, but each of the responses is unrelated to shock termination. The dog might, for example, turn his head and shock might happen to go off on the trial, but on other trials he might turn his head and shock would not go off. Shock would also terminate when he had not turned his head. Now when he is tested in the shuttle box and jumps the barrier, which in reality causes shock termination, the dog has trouble learning this. This is because he has had many experiences in which a pairing between a response and shock termination proved spurious and, as for head turning, he still expects that shock will just as likely go off if he fails to jump the barrier. Such a dog may return to taking shock even after he makes one or two "successful" jumps. In contrast, a naive dog has no interfering expectation that shock termination is independent of responding, so one experience with barrier jumping leading to shock termination is sufficient for him to learn. For some evidence regarding such associative interference see the discussion of Maier and Testa (1975), pp. 24–25.

This, then, is the theory of helplessness. The expectation that an outcome is independent of responding (a) reduces the motivation to control that outcome and (b) interferes with learning that responding controls the outcome.

Seligman (1975) has recently extended this theory to changes in emotionality. He argued that when a traumatic event first occurs it causes a heightened state of emotionality, which has been called "fear." The fear continues until the subject learns that he can or cannot control the trauma. If the subject learns he can control the trauma, fear is reduced and may disappear altogether. If the subject learns that he cannot control the traumatic event, fear decreases and is replaced with depression. For a more detailed discussion see Seligman (1975).

Alternative Explanations

Now that we have described the learned helplessness hypothesis and the manner in which it is able to account for the phenomena under consideration, we will describe some theoretical positions which have been advanced as alternatives to the helplessness hypothesis. It should be noted at the outset that these hypotheses have been offered as explanations of only the fact that organisms exposed to inescapable shock later fail to learn to escape and avoid shock in a shuttle box, rather than as explanations of the broad range of phenomena considered above. Thus we will describe these theoretical positions in the context of the basic learned helplessness effect. These alternative theories can be divided into two groups, motivational accounts and motor accounts.

Motivational Alternatives

Adaptation. The adaptation hypothesis maintains that a subject adapts to shock during pretreatment with inescapable shocks and is therefore not sufficiently motivated to escape from shock in the shuttle box. The hypothesis is inadequate:

1. Adaptation to repeated, intense shock has never been demonstrated (Church Lo-Lordo, Overmier, Solomon, & Turner, 1966).

2. It is unlikely that very much adaptation could persist for as long as 24 hours and beyond.

3. In experiments with dogs, the dogs do not look as though they are adapted; they howl, defecate, and urinate to the first shock presentation in the shuttle box. On later trials, the dogs are passive; but they whimper and jerk with the shock.

4. We have disconfirmed the adaptation hypothesis experimentally. Raising the shock level in the shuttle box should increase motivation to escape. However, Overmier and Seligman (1967) found that increasing the shock level from 4.5 mA to 6.5 mA does not eliminate the interference effect.

5. A series of escapable shocks in the hammock does not produce failure to escape in the shuttle box (Seligman & Maier, 1967), while the same shocks, if *inescapable,* do produce failure to escape. By this hypothesis, both conditions should lead to equal adaptation to shock and to similar behavior in the test situation, but they do not.

6. Seligman and Maier (1967) gave one group of dogs 10 trials of escape/avoidance training in a shuttle box *before* treatment with inescapable shock in the harness. Following exposure to inescapable shocks in the harness, the dogs escaped and avoided shock normally when returned to the shuttle box. Thus prior exposure to controllable shock "immunized" the dogs against the interfering effects of exposure to inescapable shocks. This result follows from the helplessness hypothesis because prior experience with controllable shock should proactively interfere with the subject's learning that shock is uncontrollable and should also allow the subject to discriminate between the places where shocks are controllable and uncontrollable. However, this result is not consistent with the adaptation hypothesis. Prior exposure to escapable shock in the shuttle box should not eliminate any adaptation produced by inescapable shocks in the harness.

7. If the subject is tested and fails to escape 24 hours after inescapable shocks, the learned helplessness effect will persist in chronic form; the subject will fail to escape on later opportunities. The helplessness hypothesis suggests a way to eliminate chronic failure to escape. By this hypothesis, the dog does not try to escape because he does not expect that any instrumental response will produce shock termination. By forcibly exposing the dog to the escape and avoidance contingencies, this expectation might be altered. This type of training by "putting through" has been used by others with mixed success (Loucks, 1935; Miller & Konorski, 1928; Woodbury, 1942). Seligman et al. (1968) reasoned that forcibly dragging the dog from side to side in the shuttle box, in such a way that the dog's changing compartments terminated shock, might effectively expose the dog to the response-reinforcement contingency. This was the case. The experimenter pulled three chronically helpless dogs back and forth across the shuttle box with long leashes. This was done during CS and shock, while the barrier was absent. After being pulled across the center of the shuttle box (and thus terminating shock and CS) 20, 35, and 50 times, respectively, each dog began to respond on his own. Then the barrier was replaced, and the subject continued to escape and avoid. The recovery from helplessness was complete and lasting. This type of exposure to the escape and avoidance contingencies should not affect any adaptation to shock that might have been present. Seligman et al. (1975) replicated these findings in rats.

Sensitization. Perhaps the inescapable shocks received in the harness sensitize the subject to shock so that it is too motivated to enable it to make organized responses in the shuttle box. This hypothesis is inadequate. (a) Sensitization explains the inefficiency of responding but not the absence of responding. (b) Lowering the shock level in the shuttle box should permit the subject to make organized responses. However, Overmier and Seligman (unpublished data) found that the interference effect is not attenuated when shock in the shuttle box is reduced to 3.0 mA. (3) Arguments 5, 6, and 7 in the previous paragraph, which invalidate the adaptation hypothesis, also invalidate the sensitization argument.

Motor Activity Alternatives

Within the last 2 years three hypotheses have been offered as explanations of the learned helplessness effect which are more difficult to dismiss than are sensitization and adaptation. These explanations have the advantage that they are able to account for the fact that the learned helplessness effect is determined by the controllability of the shock during the initial treatment. We have classified them as motor activity hypotheses because they all maintain that exposure to inescapable shock interferes with subsequent shuttle box acquisition because it changes motor activity. Two of these hypotheses argue that exposure to inescapable shock establishes a motor response that is incompatible with shuttling and therefore competes with shuttling. Thus these two hypotheses argue that the organism does not learn shuttling because it is performing a motor response that prevents the occurrence of shuttling. The remaining hypothesis maintains that inescapable shock is a powerful stressor

and depletes a neurochemical necessary for the occurrence of movement. Thus this view holds that inescapably shocked animals fail to acquire shuttling because they cannot move sufficiently.

Incompatible motor response theories. As already noted, two different incompatible motor response explanations of the learned helplessness effect have been proposed. The first to be described was offered by Bracewell and Black (1974) and was suggested by three experiments. In their first experiment they examined the effects of restraint and inescapable shock upon simple FR-1 shuttlebox escape/avoidance acquisition. They found that restraint in the absence of any exposure to inescapable shock produced a decrement in FR-1 shuttlebox acquisition, but that exposure to inescapable shock in the absence of restraint did not produce a reliable decrement. In their second experiment Bracewell and Black found that exposure to "high" intensity (1.0 mA) inescapable shock produces a small decrement in FR-1 shuttle box acquisition even when delivered in the unrestrained condition. In their final experiment Bracewell and Black explicitly punished movement by arranging a positive correlation between movement and shock intensity. This procedure led to a decrement in subsequent FR-1 shuttle box acquisition.

Bracewell and Black (1974) proposed the following interpretation of their data:

> One explanation that accounts for these results is the operant conditioning of responses incompatible with the shuttle box escape response that occurred in the third case [where movement was explicitly punished], also occurred in the first two. That is, restrained rats may have been punished for struggling and consequently, learned to hold still, and unrestrained rats may have learned to hold still or to perform some other incompatible response to reduce the high intensity shock. (p. 67)

Thus Bracewell and Black (1974) argued that because explicit punishment of movement produced a retardation in shuttle box acquisition, the other instances of retarded acquisition might also be due to punishment of movement during the pretreatment phase. They went on to argue that the learned helplessness effect might be explainable in the same terms. It should be noted that this hypothesis can explain why escapable shock does not produce a subsequent decrement. If shock is escapable, the organism learns a response that completely eliminates shock (the escape response) and so does not have to learn a response that only mitigates shock (the putative incompatible response). It need only be argued that the former is not incompatible with shuttling but the latter is.

We will present a large amount of data which we feel to be inconsistent with the Bracewell and Black (1974) hypothesis. However, these experiments also bear on the second competing motor response theory and so they will not be presented until that theory has been described. Nevertheless, a few comments are appropriate here. First, the fact that explicit punishment of movement produces a decrement in subsequent escape/avodiance acquisition does *not* imply that other procedures which produced escape/avoidance decrements do so because they punish movement. There are undoubtedly many ways to produce poor escape acquisition, and it is unlikely that all operate through a similar mechanism. Paralyzing the animal with a drug would retard escape acquisition, but this does not mean that the debilitating effect of inescapable shocks occurs because inescapable shock induces paralysis. Thus the retarding effect of movement punishment does not imply that inescapable shock retards acquisition because it punishes movement. Moreover, the explicit punishment of movement in the Bracewell and Black experiment led to a *much smaller* escape acquisition decrement than did inescapable shock delivered to restrained subjects. The punishment procedure increased the subsequent mean escape latency across the 10 test trials from the control level of 2.4 sec to 9.1 sec. The inescapable shock procedure resulted in a 21.5-sec mean escape latency. So it can be argued that the explicit training of an incompatible response did not duplicate the effects of exposure to inescapable shock in restrained subjects, and so the argument that the movement punishment effect accounts for the inescapable shock effect seems questionable. It can, of course, be argued that a more effective or different

punishment procedure might yield a larger escape decrement, but that remains to be demonstrated.

Further, it is not clear that Bracewell and Black (1974) employed a test procedure that is generally sensitive to learned helplessness effects. In order for an effect to be called a learned helplessness effect, it must be demonstrated that the effect is caused by the *controllability* of the events delivered during pretreatment. There is no evidence that decrements in *FR-1* shuttle box acquisition produced by prior exposure to shock are caused by the inescapability of the shocks in rats. There is not a single study employing a triadic design in rats (see p. 6) that has found appropriate effects with FR-1 shuttling as the dependent variable. In fact, there have been both reported (Anderson, Schwendiman & Payne, Note 3; Maier et al., 1973; Seligman & Beagley, 1975) and unreported (Carder, Leaf, Note 4) failures to find *any* effect of prior exposure to shock on FR-1 shuttle box escape acquisition in the rat. It is to be noted that both Anderson et al. and Maier et al. (1973) delievered inescapable shock to restrained rats and still failed to observe any effects. There are a number of differences between the various FR-1 shuttle box experiments, and it is not known which are responsible for the difference in results. Nevertheless, the dependent variable used by Bracewell and Black may not be one that is sensitive to helplessness effects in rats and so the implications of their results for learned helplessness is difficult to assess.

A further problem concerns the generality of the effects found by Bracewell and Black (1974). It will be recalled that they found restraint in the absence of shock to produce a subsequent escape deficit and inescapable shock in the absence of restraint to produce only a small escape decrement. However, Maier et al. (1973) used an escape/avoidance task known to be sensitive to helplessness effects in rats (see p. 8) and found no effect of restraint alone. Cohen (1970) found no effect of restraint on shuttle box escape/avoidance acquisition in dogs. Further, Looney and Cohen (1972), Seligman and Beagley (1975), and Seligman et al. (1975) reported large escape deficits produced by exposure to inescapable shock in unrestrained rats. Again there are many differences between studies, but it is clear that restraint does not always produce a subsequent escape decrement nor does inescapable shock have to be delivered to restrained subjects for a large effect to occur.

A second incompatible motor response theory has been proposed by Anisman and Waller (1973). They argued that exposure to shock induces response repertoire changes in the organism, and if no coping response is available, "freezing" becomes the organism's dominant reaction to shock. Freezing would, of course, compete with the occurrence of active responding in a subsequent escape/avoidance task. As evidence for their hypothesis, Anisman and Waller cite a variety of studies which show that manipulations designed to increase freezing have a detrimental effect on avoidance behavior, and manipulations designed to reduce freezing facilitate avoidance performance. So, for example, strong shock, which causes more freezing in rats, produced more interference with shuttle avoidance than weak shock (Anisman & Waller, 1972) and scopalamine, a drug that reduces freezing, makes rats better avoiders (Anisman, 1973).

A number of comments are appropriate. First, the experiments discussed by Anisman and Waller are not directly relevant to the learned helplessness effect. They are all studies of effects on *avoidance,* not escape. There is no evidence that the manipulations described by Anisman and Waller affect *escape* acquisition. In fact, escape latencies are not even reported in many of the experiments (e.g., Anisman, 1973). The learned helplessness effect is a failure to escape—to flee from shock itself—a more profound debilitation than failure to prevent shock. Second, even if it could be shown that inducing freezing debilitates escape, this does not mean that inescapable shock interferes with escape by inducing freezing. In addition, dogs do not seem to freeze, people who receive unsolvable discrimination problems or inescapable noise do not freeze, and organisms that receive noncontingent food do not freeze, yet these conditions may all produce

helplessness. Finally, why does inescapable shock, but not escapable shock, produce freezing (if it does) in rats? We have not been able to think of an answer that does not imply that the rat has learned that shock is inescapable, and this is the heart of the helplessness view.

Experimental evidence. We feel that there are six lines of evidence that bear on the incompatible motor response theories, and each will be described.

1. It has already been noted that rats exposed to inescapable shock often do not fail to learn an FR-1 shuttle box response. This has been investigated by Maier et al. (1973), and in agreement with a number of previous investigators they found that rats exposed to inescapable shock under a wide range of parameters subsequently responded as rapidly as did controls in a two-way shuttle box escape/avoidance acquisition test. However, they noted that the rats in their experiments seemed to acquire this shuttling escape response in a manner different from that exhibited by dogs. The rats responded very rapidly, but the response was as rapid on the very first acquisition trial as it was after 30 trials—the acquisition curves were flat. The mean response latency from shock onset on the very first shuttle box trial was 2.67 sec. The escape responding of dogs in a shuttle box is quite different. Averaged across experiments, the naive control dogs in the Maier, Overmier, and Seligman experiments took slightly over 30.0 sec of shock before escaping on the first trial and did not emit short latency escape responses until the fourth trial. This suggested to Maier et al. (1973) that an interference effect in rats might be found if a more slowly acquired escape response were used as the test task. The escape response they used was one in which the rats had to cross the shuttle box *twice* in order to terminate shock. However, these FR-2 trials were preceded by five ordinary shuttle box trials (FR-1), since pilot investigation revealed that naive rats sometimes fail to acquire the FR-2 shuttling response if such prior training was not provided. The result was that a large interference effect appeared—half of the rats exposed to inescapable shocks did not learn the FR-2 escape response even though they responded rapidly on the five FR-1 trials, while all control rats learned the FR-2 escape response. It should be noted that we have replicated this effect several times, as have other laboratories (Seligman & Beagley, 1975; Weisman, Note 5).

The fact that rats exposed to inescapable shock will learn FR-1 shuttling but will not learn FR-2 shuttling might be explained in a variety of ways. The helplessness hypothesis asserts that prior exposure to inescapable shock interferes with the subsequent formation of associations between responding and shock termination and, in addition, reduces the organism's incentive to attempt to escape. From the foregoing discussion it is clear that crossing the shuttle box *once* is a high probability initial response to shock in the rat, perhaps a species-specific defense response (Bolles, 1970) and, at least on early trials, may be elicited. Consistent with this view of shuttle box crossing, Maier et al. (1973) found that if conditions were arranged such that there was no contingency between crossing the shuttle box and shock termination, naive rats would still cross the shuttle box in response to shock with short latencies for about 10 trials.

It should be noted that this strong tendency of the rat to cross the shuttle box rapidly in response to shock guarantees that the preshocked rats will be exposed to the FR-1 contingency a relatively large number of times even if such a rat is slow to learn. In addition, the contingency between crossing the shuttle box and shock termination is relatively simple under the FR-1 procedure, and so the preshocked rats might learn FR-1 escape even though associative interference is actually present. If associative interference were present, some difference in FR-1 acquisition between inescapably shocked and control rats, at least on early trials, might be expected. However, such a difference could be masked by the elicited nature of shuttling on early trials. Consistent with this argument, Testa, Juraska, and Maier (1974) found that FR-1 shuttling *extinguished* more rapidly in groups given previous exposure to inescapable shock than in

controls, even though these groups did not differ in the acquisition of FR-1 shuttling.

The FR-2 shuttling response, on the other hand, is not as probable an initial unconditioned response to shock as is FR-1 shuttling, and presents a more complicated contingency between crossing the shuttle box and shock termination than does FR-1 shuttling. Thus the associative interference produced by learning that shock is inescapable might be able to prevent the acquisition of can also explain why exposure to inescap- the FR-2 shuttle box response.

Both competing motor response hypotheses able shock has little or no effect on FR-1 shuttling. The competing motor response theories could argue that the competing response is not strong enough to interfere with the highly elicited FR-1 but can compete with the initially weaker FR-2. In addition the FR-2 procedure exposes the rat to more shock than does the FR-1 procedure, and it could be argued that this intensifies the competing response.

Although these hypotheses are not mutually exclusive, they point to different aspects of the FR-2 procedure as the crucial feature in producing an interference effect. The helplessness hypothesis emphasizes the difficulty of learning the contingency between crossing the shuttle box and shock termination inherent in the FR-2 procedure, while the competing response explanations emphasize the physical difficulty of the FR-2 response and/or the large amount of shock to which the FR-2 procedure exposes an organism. There are two obvious ways to determine which of these aspects of the FR-2 procedure is crucial. The first is to simplify the contingency between crossing the shuttle box and shock termination while at the same time maintaining the physical difficulty and shock exposure characteristics of the FR-2. The second is to arrange a complex contingency between shuttling and shock termination while at the same time reducing the physical difficulty of the response and the extent of shock exposure. Maier and Testa (1975) have conducted experiments using each of these strategies.

The contingency between crossing the shuttle box and shock termination should be relatively difficult to learn with the FR-2 procedure, for the obvious reason that shock does not terminate after each crossing. In the Maier et al. (1973) FR-2 experiment, shock was simultaneously presented to the grids of *both* chambers of the shuttle box, so that there was no consequence for the first response of the FR-2, not even a momentary interruption of shock presentation. The contingency between the shuttle response and shock termination can be improved by arranging a brief interruption of shock following the first response in the FR-2. It should be noted that such a procedure will not change the effortfulness of the FR-2 nor will it have an appreciable effect on the duration of shock exposure produced by the FR-2 procedure, if the interruption in shock is very brief.

Maier and Testa (1975) conducted precisely the experiment suggested above. It was found that although inescapably shocked rats fail to learn ordinary FR-2 shuttling, they learn as well as nonshocked controls when there is a 1-sec interruption of shock after the first crossing of the FR-2. Since the only obvious difference between the procedures which did and did not yield a learned helplessness effect was the nature of the contingency between responding and shock termination, it seems likely that associative factors are strongly involved.

The previous experiment suggests that the inescapably shocked rats learn the FR-1 task but do not learn the FR-2 task because the FR-1 task contains an obvious contingency between shuttling and shock termination while the FR-2 task contains a degraded contingency. If this is the case, then it ought to be possible to produce a learned helplessness effect with a single crossing of the shuttle box if the contingency between the single crossing and shock termination were degraded.

In a second experiment, Maier and Testa (1975) degraded the contingency between a single crossing of the shuttle box and shock termination by interposing a time delay between crossing of the shuttle box and shock termination. Thus the required escape response was no more effortful than the ordinary FR-1 task which does not

TABLE 2

DESIGN OF THE MAIER AND RHOADES EXPERIMENT

Group	Phase 1	Phase 2	Phase 3
ES-IS	Escapable wheel turn	Inescapable tube	Escapable shuttle box
IS-IS	Inescapable wheel turn	Inescapable tube	Escapable shuttle box
NS-IS	No-shock wheel turn	Inescapable tube	Escapable shuttle box
NS-NS	No-shock wheel turn	No-shock tube	Escapable shuttle box

yield an interference effect, but contained an obscured contingency between shuttling and shock termination, as does the FR-2 task which does yield an interference effect. We found that a delay in shock termination as short as 1 sec severely retarded the acquisition of FR-1 shuttle box escape in rats previously exposed to inescapable shocks, but had no effect on subjects not previously exposed to inescapable shocks. It should be noted that a procedure designed to control for the extra amount of shock produced by the delay revealed that this factor did not account for the results. Thus prior exposure to inescapable shock will retard the acquisition of even low-effort escape responses if the contingency between the escape responses and shock termination is made less obvious.

Taken together, the results of these two experiments indicate that the nature of the contingency between the escape response and shock termination is more important than are the effort and shock exposure characteristics of the escape response in determining whether an interference effect will occur. Inescapably shocked rats fail to learn FR-2 shuttle box escape because the contingency between crossing the shuttle box and shock termination is here more complex, not because the FR-2 response is effortful or exposes the subject to long shock durations. It is therefore difficult to account for the rat interference effect with an explanation that does not posit an associative deficit following inescapable shock.

The incompatible motor response hypotheses cannot handle these data. It is difficult to conceive of a motor response that would interfere with FR-2 shuttling when there is no shock interruption, but not when there is. Similarly, what motor response could be incompatible with FR-1 shuttling if there is a short delay in shock termination but not incompatible when there is no delay?

2. It will be recalled (p. 20) that Seligman and Maier (1967) found that prior exposure to controllable shock immunizes the organism against the deleterious effects of exposure to uncontrollable aversive events. This finding might seem inconsistent with the incompatible motor response hypotheses since prior exposure to escapable shock should not prevent the establishment of the incompatible shock-mitigating response or freezing during exposure to uncontrollable shock. However, in the Seligman and Maier experiment the subjects' initial experience with controllable shock was given with the same task that was used as the test task following exposure to inescapable shock. That is, the subjects were first given escape training in a shuttle box, then given inescapable shock in a harness, and then tested in a shuttle box. Thus the immunization effect may not have been caused by the organisms' initial control over shock but rather by the acquisition of the specific response later used as a test.

The confounding of initial control over shock with the acquisition of the test task response allows the incompatible motor response theories to account for the Seligman and Maier immunization experiment. They could argue that the Seligman and Maier procedure strengthened shuttling sufficiently to overcome the putative competing response.

Clearly, what is needed is an experiment which separates the effects of initial control over shock and the prior strengthening of the specific response later to be tested. An unpublished experiment by Maier and Rhoades meets this requirement. (See also Seligman et al., 1975). The design of the

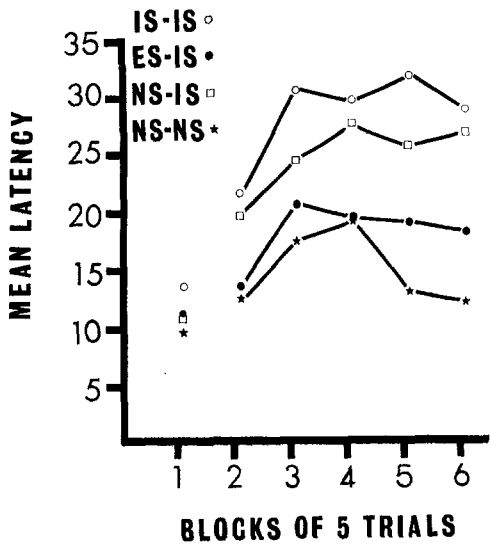

FIG. 7. Mean shuttle box FR-2 escape response latency for rats previously given either escapable shock (ES), yoked inescapable shock (IS), or no shock (NS) in a wheel-turn apparatus, and then given either inescapable shock (IS) or no shock (NS) in a restraining tube (Maier & Rhoades, unpublished data, 1975). (The rats given escape training in the wheel-turn box and then given inescapable shocks in the restraining tube [ES-IS] were not helpless in the shuttle box.)

experiment can be seen in Table 2. The experiment involved three phases and four groups of rats. In the first phase one group of rats was trained to escape shock by turning a small wheel with their paws, a second group received yoked inescapable shock in a wheel-turn box, and two groups were placed in the wheel-turn box but not shocked. In the second phase both of the shocked groups and one of the control groups received inescapable shock in a restraining tube, and the remaining group was only restrained. In the third phase all groups were given five trials of FR-1 followed by 25 trials of FR-2 escape/avoidance training in a shuttle box, our standard helplessness test task.

The results found by Maier and Rhoades are seen in Figure 7. Prior experience with escapable shock in the wheel-turn situation mitigated the interfering effects of inescapable shock on shuttle box escape acquisition. Although prior experience with escapable shock in the wheel-turn situation did not completely eliminate the effects of exposure to inescapable shock, the immunizing effect was reliable. It is difficult for competing response theories to account for an immunizing effect that is caused by the experience of control per se rather than by the prior strengthening of the test response. It is difficult to argue that learning to turn a small wheel with the paws in a wheel-turn box directly strengthens running in a shuttle box. This wheel-turn escape training should not have an immunizing effect, but it does.

3. If inescapable shock interferes with subsequent escape acquisition because it has led to the development of a motor response incompatible with the escape response, then the explicit reinforcement of such a response should duplicate the effects of exposure to inescapable shock. As already noted, Bracewell and Black (1974) found that the explicit punishment of movement led to a small decrement in subsequent acquisition in a shuttle box. However, this experiment is difficult to interpret for the reasons already noted (see pp. 21–22). Another study using this strategy was reported by Maier (1970). One group of dogs was trained to escape shock by holding still, a response directly incompatible with jumping over a hurdle in a shuttle box. The dogs were restrained in a hammock and panels were placed ¼ in. (.64 cm) above and ¼ in. (.64 cm) to each side of the dog's head. The dogs could not prevent the onset of shock, but they could terminate shock by *not touching* any of the panels for a specified period of time once the shock came on. Since shock elicits head movements in restrained dogs, the dogs were required to actively inhibit movements elicited by shock. Because the panels were positioned ¼ in. (.64 cm) from the dog's head, the dogs could refrain from touching the panels only by remaining almost perfectly motionless, by freezing. A second group of dogs received inescapable shock yoked to the first group and a third received no pretreatment. Following this treatment all groups received 12 days of testing in a shuttle box.

If inescapable shock produces a learned helplessness effect because it induces freezing or leads to the punishment of movement, then the group taught to inhibit movement as

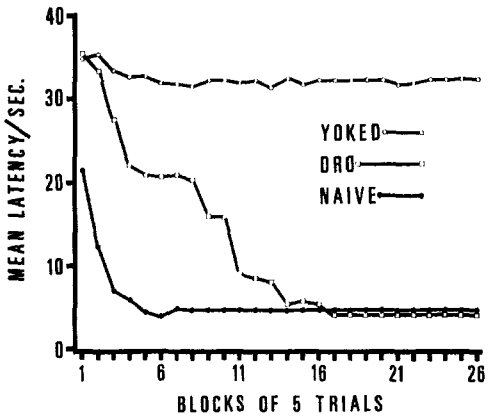

FIGURE 8. Mean latency to respond in a shuttle box for dogs previously given training to escape shock by holding still (DRO), given yoked inescapable shocks, or given no shocks. (The yoked group did not learn, whereas the group taught to hold still did learn.) (From "Failure to Escape Traumatic Shock: Incompatible Skeletal Motor Responses or Learned Helplessness?" by Steven F. Maier, *Learning and Motivation,* 1970, *1,* 157–170. Copyright 1970 by Academic Press. Reprinted by permission.)

a means of escaping shock should later escape at least as poorly as the group given inescapable shock. The learned helplessness hypothesis makes a different prediction. Even though the dogs have learned an incompatible motor response, they have also learned that they have control over shock; and even if they should be slow to learn to escape in the shuttle box, they should eventually learn. In contrast, dogs given inescapable shocks should not learn to escape in the new situation; experiences of failure to escape on the early trials in the new situation should strengthen the assumed cause of interference.

The results of this experiment can be seen in Figure 8. The group (labeled DRO) trained to perform a response incompatible with shuttling was slow to learn shuttling, but all subjects in this group eventually learned. In contrast, half of the subjects given inescapable shock entirely failed to learn across the 12 days of testing. Thus the training of a response demonstrably incompatible with shuttling (negative transfer was produced) did not duplicate the effects of inescapable shock, and this is inconsistent with competing motor response explanations of the learned helplessness effect. It could, of course, be argued that exposure to inescapable shock establishes a response that is stronger or even more incompatible with shuttling than the one trained in the present experiment; the burden of proof, however, would clearly be shifted onto such an argument.

4. It will be recalled that Bracewell and Black (1974) argued that movement occurs during inescapable shock and is punished, presumably because it hurts to struggle. Alternatively, Bracewell and Black suggested that the subject might find some other motor response that reduces the pain produced by shock. This position predicts that if the subject is not allowed to move during exposure to inescapable shock, no learned helplessness should result. If movement does not occur it cannot produce an increment in pain and so be punished, and if movement does not occur the subject cannot find a motor response that mitigates shock.

Overmier and Seligman (1967) prevented movement during exposure to inescapable shock by the administration of curare. Curarization during exposure to inescapable shock did not prevent or even reduce the subsequent learned helplessness effect. It seems to us that this experiment is a direct disconfirmation of the Bracewell and Black (1974) hypothesis.[5] However, Bracewell and Black have argued that this curare experiment does not necessarily contradict their position. Their argument is that movement-related neural processes might be adventitiously reinforced or punished during curarization: "A possibility exists, therefore, that the onset and termination of shock could act inadvertently to reinforce superstitious movement related neural responses in curarized dogs" (Bracewell & Black, 1974, p. 55). It should be carefully noted that this is different from their original position. The Bracewell and Black hypothesis

[5] It should be noted that this experiment has no implications for the Anisman and Waller (1973) competing motor response theory. There is no reason to believe that curarization during inescapable shock should reduce any subsequent freezing.

argued that an incompatible response is acquired because it produces a positive outcome of reduced pain or discomfort. Thus Bracewell and Black provided a mechanism whereby the incompatible response is acquired, an *explicit* reinforcement mechanism. But in the case of the curarized subject Bracewell and Black do *not* argue that "the movement related neural processes" reduce pain and are therefore explicitly reinforced since there is no obvious way that such processes could reduce pain. Thus Bracewell and Black argue that in the case of curarized subjects movement-related neural processes are *adventitiously* rather than explicitly reinforced, and this seems gratuitous. Although there is evidence that movement-related neural processes can be explicitly punished (Black, Young, & Batenchuk, 1970), there is no evidence that they can be adventitiously or superstitiously reinforced. Further, even if such processes could be adventitiously reinforced or punished there is no reason to assume that the overt movement produced by such a process would be incompatible with shuttling. It could just as easily facilitate shuttling. To assume that it will be incompatible is, again, gratuitous.

5. We have already described an experiment by Rosellini and Seligman (1975) (see p. 10) in which rats were given escapable shock, inescapable shock, or no shock, and were then trained to traverse a runway for food. Following training, the rats were extinguished and then allowed to escape from the presumably frustrating goal box. The result was that the rats previously exposed to inescapable shock were slow to escape the goal box but did not differ from controls in either acquisition or extinction. If a motor response is established during exposure to inescapable shock, it is difficult to see why it would interfere with escape from the goal box but not interfere with runway acquisition and extinction.

6. The incompatible motor response theories have difficulty dealing with situations that do not employ electric shocks or other noxious unconditioned stimuli. The particular competing response hypotheses offered by Bracewell and Black (1974) and by Anisman and Waller (1973) cannot account for appetitive analogues to the learned helplessness effect (see p. 12) in animals or learned helplessness effects in human beings induced by exposure to loud noises (see p. 9). Moreover, it is difficult to see how *any* incompatible motor response view could account for the noise-escape failure produced by unsolvable discrimination problems in human subjects (see p. 11). It should be noted that the incompatible motor response hypotheses which we have discussed were designed to account only for the effects of inescapable shock in animals, and it could be argued that these other effects are different phenomena and therefore need not all be explained by the same theory. Such a move seems ad hoc and our preference is for that theory which is able to integrate the largest amount of data.

Motor Activation Deficit Hypothesis

A somewhat different motor response explanation of the learned helplessness phenomenon has been offered by Weiss et al., (1975). Their explanation of the learned helplessness effect is as follows:

> When animals were exposed to severe inescapable shock, a deficiency in central noradrenergic activity occurred, deriving in part at least, from the depletion in the level of norepinephrine seen in this condition. As a consequence of this noradrenergic deficiency the animals could mediate only a limited amount of motor activity, an amount insufficient for learning and performance of the correct response in the shuttle avoidance task on which they were tested. On the other hand, animals exposed to the same shocks but able to control them did not develop this noradrenergic deficiency and therefore could mediate sufficient motor behavior to perform adequately. (p. 7)

A number of factors led Weiss et al. (1975) to develop this explanation of the learned helplessness effect. First, Miller and Weiss (1969) argued that the dissipation of the learned helplessness effect over a 48-hour interval following one session of exposure to inescapable shock in dogs suggests mediation by a time-dependent physiological change rather than by learning. They argued that things that are learned do not disappear across an interval as short as 48 hours. Second, it is known that central catecholamines have a time course of recovery fol-

lowing depletion (e.g., Rech, Bovys, & Moore, 1966) and are involved in the mediation of movement (e.g., Herman, 1970). Third, Weiss (1968, 1971a, 1971b, 1971c) has shown that rats given inescapable shock undergo greater stress than do rats given equivalent escapable shocks, as measured by gastric lesions, loss of body weight, plasma steroid levels, and fearfulness. Finally, this led Weiss, Stone, and Harrell (1970) to assay whole brain norepinephrine following exposure to escapable or yoked inescapable shock. They found norepinephrine levels to be lower following exposure to inescapable shock. This finding has been replicated in separate brain regions by Weiss, Pohorecky, Dorros, Williams, Emmel, Whittlesey, and Case (Note 6) and has been shown to occur regardless of the amount of motor activity required to escape and avoid shock (Weiss, Pohorecky, Emmel, & Miller, cited in Weiss et al., 1975). Following these demonstrations of effect on endogenous norepinephrine levels, Weiss, Pohorecky, McMenima, Berkeley, and Jaffe (cited in Weiss et al., 1975) estimated norepinephrine reuptake and release in vivo by infusion of radioactive (3H) norepinephrine. They found that reuptake occurred at a higher rate in rats previously exposed to inescapable than escapable shocks. This reuptake difference could account for the differences in the level of endogenous norepinephrine.

Weiss et al. (1975) present 12 experiments in support of the notion that the learned helplessness effect occurs because inescapable shock is a severe stressor, produces a temporary depletion of norepinephrine, and thus depresses the movement the organism can emit below what is required for shuttle box acquisition. We will describe each of these 12 experiments. In the first three experiments, Weiss et al. attempted to show that a procedure which deplete norepinephrine but does not involve the factors stressed by the learned helplessness hypothesis duplicates the learned helplessness phenomenon. It is known that a forced swim in 2 °C water rapidly depletes brain norepinephrine levels and reduces its release (Stone, 1970a, 1970b). Thus the motor activation deficit hypothesis would expect a forced swim in cold water to produce a learned helplessness effect. However, a forced swim in warm water (28 °C) does not produce a depletion of brain norepinephrine and so the motor activation deficit hypothesis would not expect a warm swim to produce a learned helplessness effect. Weiss et al. argued that animals forced to swim in either warm or cold water should experience the same lack of control, and that the learned helplessness hypothesis would thus expect similar effects resulting from a warm or a cold swim. In Experiment 1, rats were put in either warm (28 °C) or cold (2 °C) water for either 3.5 or 6.5 minutes. Thirty minutes later the rats were all given an FR-1 shuttle box shock escape/avoidance acquisition test. The results were that the rats exposed to cold swim were severely retarded in shuttle box acquisition, while the warm-swim subjects showed normal acquisition.

Experiment 1 led Weiss et al. (1975) to conclude that cold swim duplicated the failure-to-learn aspect of the learned helplessness phenomenon. Another feature of the learned helplessness phenomenon is that prior training to escape in the shuttle box eliminates the interfering effects of subsequent exposure to inescapable shock. In Experiment 2 Weiss et al. gave rats 25 trials of FR-1 shuttle box training 24 hours before a 3.5 minute cold swim. The rats were again tested in the shuttle box 30 minutes after the cold swim. The pretraining in the shuttle box reduced the deficit produced by cold swim. Another feature of the learned helplessness phenomenon is that it has a time course in dogs following a single session of inescapable shock. In Experiment 3 Weiss et al. gave rats a cold swim and then tested them in a shuttle box 30 minutes, 2 hours, or 48 hours later. With a 48-hour delay, there was no longer any detectable effect of the cold swim. Further, rats tested at the 30-minute point were retested 48 hours later. A deficit was still present upon retest, as was the case with learned helplessness. These three experiments led Weiss et al. to conclude that cold swim duplicates the effects of inescapable shocks.

The next six experiments employed a different strategy. In these experiments, Weiss et al. attempted to find situations in which the motor deficit and the learned helplessness hypotheses make different or opposed predictions. The motor activation deficit hypothesis implies that the magnitude and the likelihood of finding a failure to learn to escape depends on the amount of motor activity required to escape. Weiss et al. claimed that the learned helplessness hypothesis would not expect the amount of effort required to escape to be a potent variable. Experiment 4 by Weiss et al. demonstrated that the detrimental effects of cold swim are strengthened by increasing the height of the barrier in the shuttle box, and Experiment 5 showed that cold swim did not produce a deficit in learning to escape shock when the escape response only required restrained rats to poke their nose through a hole in the front of the tube in which they were confined. In Experiment 6 Weiss et al. attempted to demonstrate that exposure to inescapable shock interferes with acquisition of escape behavior in their FR-1 test task. They mentioned that they have had difficulty in producing such an effect, but showed that a 50-min. exposure to 4.0 mA 2.0-sec shocks occurring every 20 sec produces an escape deficit when the rat is tested 30 min later. Experiment 7 showed that such shocks interfered with shuttle box but not with nose-poke acquisition, as predicted by the motor deficit hypothesis.

The next three experiments by Weiss et al. are based on data (Zigmond & Harvey, 1970) which indicate that the central noradrenergic response to stress habituates with repeated exposure to stress. That is, the degree of norepinephrine depletion produced by a stressor is much reduced if the organism has been repeatedly stressed previously. This leads the motor activation deficit hypothesis to predict that repeated exposures to the stressful condition should *reduce* any subsequent deficit in a test task. The learned helplessness hypothesis surely does not make such a prediction. In Experiments 8 and 9 Weiss et al. found that 14 sessions of exposure to cold swim or inescapable shock eliminated the FR-1 shuttle box escape deficit produced by cold swim or inescapable shock. In addition, these experiments confirmed the finding that norepinephrine is not depleted after repeated stress.

A variety of drug treatments were explored by Weiss et al. in the final three experiments. Experiment 10 found that tetrabenazine, a relatively nonspecific depletor of monoamines, produced poor performance in a shuttle box 30 minutes after administration. Experiment 11 showed that repeated administration of tetrabenazine did not affect shuttle box performance. Finally, Experiment 12 found that pargyline, a drug which inhibits monoamine oxidase and therefore prevents intraneuronal degradation of monoamines, prevented inescapable shock from affecting FR-1 shuttle box performance.

Discussion of the motor activation deficit hypothesis. We have presented the entire logical and evidential bases of the motor deficit hypothesis without comment or interruption because it presents, at least on the surface, a convincing case and should be seen as such by the reader. However, we feel that every step in the argument which suggested the motor deficit hypothesis and each piece of data which seems to support the hypothesis is questionable and open to alternative interpretation, and we will discuss each, point by point. Following this discussion we will present a variety of lines of evidence which we feel are inconsistent with the motor deficit hypothesis.

It will be recalled that a physiological depletion hypothesis was first suggested by Miller and Weiss (1969), who argued that the time course of the learned helplessness effect is not consistent with the notion that something learned during exposure to inescapable shock is the cause of the effect. They argued that if something is learned it should still be present 48 hours later. Although this point is not crucial to the motor deficit hypothesis, it should be pointed out that it is highly debatable. Proactive and retroactive interference produce memory losses in animals (Maier, Allaway, & Gleitman, 1967; Maier & Gleitman, 1967) as well as man, and both proactive and retroactive interference effects generally increase

with time since learning. It is possible that prior experiences with learning to control events could interfere with the retention of the learning that shocks are uncontrollable after only one sesison with inescapable shocks. In addition, there is ample evidence in animals for retention losses over short intervals of time (see D'Amato, 1973; Spear, 1973).

More importantly, Weiss et al. (1975) ignored aspects of the learned helplessness time course data which are not as congenial to their hypothesis as is the fact that the effect does not occur after 48 hours following a single experience with inescapable shocks. Seligman and Groves (1970) have shown that the time course of the learned helplessness effect is eliminated in dogs if the dogs are given four sessions rather than one session of inescapable shock. Such dogs failed to learn to escape even when tested 7 days after their last exposure to inescapable shock. Further, if dogs are raised in laboratory cages and thus deprived of extensive experience controlling events, only two sessions of inescapable shock are required to produce nontransient learned helplessness effects (Seligman & Groves, 1970). It should be noted that these are precisely the results that would be expected from a memory interpretation of the time course and are not explicable by the motor deficit hypothesis in any obvious way. Norepinephrine levels should certainly be recovered by 7 days following exposure to inescapable shock. Finally, Seligman and Beagley (1975) failed to find a time course of learned helplessness with rats as subjects after only one session of inescapable shock. Rats given inescapable shock failed to learn to escape when tested 7 days after exposure to a single session of inescapable shock. This finding is particularly damaging to the motor deficit hypothesis because this hypothesis rests on a base of data obtained with rats, not dogs, as subjects. It would be interesting if rats raised in a natural environment, rather than in laboratory cages, showed a transient helplessness effect. In summary, the transient effect required by the motor deficit hypothesis occurs only under one condition—a single session of inescapable shock with mongrel dogs not raised in the laboratory. The time course data taken as a whole does not invalidate a learning interpretation and may be more consistent with the learned helplessness hypothesis than with the motor activation deficit hypothesis.

Of greater importance in establishing the plausibility of the motor activation deficit hypothesis are the several demonstrations by Weiss and his colleagues that exposure to inescapable shock is highly stressful and reduces endogenous norepinephrine levels while exposure to escapable shock is less stressful and does not lead to such a reduction. It is this that allows the motor deficit hypothesis to explain why the learned helplessness effect occurs and is determined by the controllability of the initial shock. Several comments are in order. Weiss et al. (1975) argue that the learned helplessness effects which we have found are caused by norepinephrine depletion produced by the inescapable shock which *we* deliver. Therefore, it is *required* that Weiss and his colleagues demonstrate that *our* inescapable shock conditions produce intense stress and norepinephrine depletion. It is thus crucial to inquire whether the experiments by Weiss and his colleagues employed conditions roughly similar to ours. Table 3 summarizes some of the relevant aspects of our rat helplessness experiments and the Weiss studies. As can be seen, the behavioral helplessness studies use 1.0-mA shocks with session lengths lasting from 1 to 1½ hours. The rat's behavior is tested 24 hours or longer after the inescapable shock session. The conditions used by Weiss and his colleagues *bear no resemblance at all* to these conditions. All but one of the norepinephrine studies exposed the subjects to a *minimum of 20 hours* of inescapable shock, and shock intensity was a *minimum of 3.0 mA*. Further, norepinephrine was typically assayed *immediately* following the end of the inescapable shock session. It is worth noting that the one norepinephrine study which deviated from these parameters, even though still very different from our parameters (Weiss et al., 1970, Experiment 1), did *not* find depletion of norepinephrine in the group given inescapable shocks. This experiment

TABLE 3

SUMMARY OF THE PARAMETERS USED IN THE RAT LEARNED HELPLESSNESS EXPERIMENTS

Experiment	Maximum shock level	Session length	Time between inescapable shock and test
Behavioral helplessness			
Maier, Albin, & Testa	1.0 mA	1½ hr.	24 hr.
Goeckner, Greenough, & Maier	1.0 mA	1 hr.	24 hr.
Testa, Juraska, & Maier	1.0 mA	1½ hr.	24 hr.
Maier & Testa	1.0 mA	1½ hr.	24 hr.
Seligman & Beagley	1.0 mA	1½ hr.	24 hr.
Seligman, Rosellini, & Kozak	1.0 mA	1½ hr.	5 min.; 1 hr.; 24 hr.; 168 hr.
Seligman, Hannum, & Rosellini	1.0 mA	1½ hr.	65 days
Ulcers, steroids			
Weiss (1968), Experiment 2	1.6 mA	21 hr.	12 hr.
Weiss (1971a)	4.0 mA	48 hr.	none
Weiss (1971b)	4.0 mA	48 hr.	none
Weiss (1971c)	4.0 mA	48 hr.	none
Norepinephrine			
Weiss, Stone & Harrell, Experiment 1	3.0 mA	3 hr.	20 min.; 40 min.
Weiss, Pohorecky, Dorros, Williams, Emmel, Whittlesey, & Case	3.4 mA	20 hr.	none
Weiss, Pohorecky, Emmel, & Miller	not known	24 hr.	none
Weiss, Pohorecky, Emmel, McMenima, Berkeley, & Jaffe	not known	20 hr.	2 min.

found a reliable overall difference in norepinephrine levels between inescapable, escapable, and no-shock groups. However, the inescapable group did not differ reliably from the no-shock group, and the overall difference was due to an *elevation* of norepinephrine in the escapable shock group.

It should be clear that, in order to explain the learned helplessness effect, the motor activation deficit hypothesis requires that a 1-hour session employing 1.0-mA shocks (occurring about one per minute) produce a depletion in norepinephrine which is still present 24 hours following the termination of the inescapable shock session. There is not a shred of evidence for such an effect. It is a curious argument that holds a behavioral deficit which occurs 24 hours after a 1-hour session with 1.0-mA shocks is attributable to norepinephrine depletion because norepinephrine levels are low when measured immediately after massive 20-hour sessions with 3.4- or 4.0-mA shocks.

At this point it should be noted that Weiss et al. (1975) have restricted their explanation to only the *dog* helplessness experiments, and not the rat helplessness experiments. However, all of the data obtained by Weiss and his colleagues came from rats, and so we feel that a comparison of their parameters with those used in our rat studies has been proper. How can one argue that data obtained from rats apply to data obtained from dogs but not to parallel data obtained from rats? Further, the parameters used in our dog studies also differ massively from those used by Weiss et al.—we have typically used 64 shocks spread over about 1½ hours and have tested the dogs 24 hours later.

Moreover, Weiss et al. (1975) have reported that endogenous levels of norepinephrine are depleted in rats given inescapable shock *only if the rats were group housed prior to the experiment.* That is, in all of the neurochemical studies referred to above, the rats were group housed before treatment. If the rats were individually housed before treatment inescapable shock did not deplete endogenous norepinephrine levels. This may

be because individual housing in rats induces increased activity of tyrosine hydroxylase (Segal, Knapp, Kuczenski, & Mandell, 1973), a rate-limiting enzyme in the synthesis of norepinephrine. It should be noted that the rats in all of our experiments have been *individually housed* prior to treatment. In fact, Goeckner, Greenough, and Maier (1974) manipulated housing conditions from weaning to adulthood and found that inescapable shock produced a learned helplessness effect whether the rats were group housed or individually housed.

We conclude that the neurochemical data said to provide plausibility to the motor activation deficit explanation of learned helplessness, in fact, provide little if any support.

We now turn to a consideration of the 12 experiments reported by Weiss et al. (1975) as supporting the motor deficit interpretation of the learned helplessness effect. Recall that the first three experiments were designed to show two things: (a) that a treatment which depletes norepinephrine (cold swim) but cannot be said to lead to the learning that aversive events and responding are independent produces all of the characteristics of the learned helplessness phenomenon; (b) that a procedure which involves the same degree of uncontrollability as the first but does not deplete norepinephrine (warm swim) does not produce a learned helplessness effect. We do not feel that the experiments actually demonstrate that which is claimed.

Recall that Weiss et al. compared the effects of a cold swim and a warm swim on FR-1 shuttle box acquisition and argued that the learned helplessness hypothesis would expect equivalent effects from these two treatments since they involve equal uncontrollability. This is not correct. The learned helplessness hypothesis does not argue that failure to learn to escape shock results from exposure to sheer uncontrollability, but by exposure to uncontrollable *aversive* events. For example, the learned helplessness hypothesis would not expect failure to escape shock to result from exposure to a light presented independently of behavior. Cold swim and warm swim are certainly not equally aversive, and warm swim may not be aversive at all. Further, although we do not wish to argue in this direction, it is not clear to us why cold swim cannot be described as a condition in which escape from the highly aversive freezing water is independent of behavior. Cold swim produces muscular debilitation while warm swim does not. Thus the trauma in a cold swim may indeed be more uncontrollable than in a warm swim.

More important than whether the learned helplessness hypothesis predicts equivalent effects of cold and warm swim, we do not believe that Weiss et al. (1975) have demonstrated that cold swim produces a behavioral deficit that is the same as the learned helplessness effect. Learned helplessness is defined as an effect resulting from the uncontrollability of aversive events. As noted previously, FR-1 shuttle box acquisition may not be sensitive to such effects in rats, and a variety of experimenters have failed to find any effect of inescapable shock on subsequent FR-1 shuttle box acquisition. There are a few experiments which find an effect of inescapable shock on subsequent FR-1 shuttle box acquisition (e.g., Weiss et al., 1975, Experiment 6) but these experiments omit comparison of the effects of equivalent escapable and inescapable shock so it cannot be concluded that a learned helplessness effect with FR-1 shuttling has been demonstrated. Since Weiss et al. employed FR-1 shuttling as their dependent variable, they may have been using a test that is not sensitive to learned helplessness effects.

Further, Weiss et al. (1975) used a 30-minute interval between cold swim and the behavioral task, whereas our learned helplessness experiments use a 24-hour interval between inescapable shock exposure and test. Thus Weiss et al. must demonstrate that cold swim produces a deficit 24 hours later, not 30 minutes later. Experiment 3 of Weiss et al. examined the time course of the cold-swim effect, but it does not help us here because Weiss et al. did not have a 24-hour time point. Their experiment only involved testing at 30 minutes, 2 hours, or 48 hours. An examination of the Weiss et al.

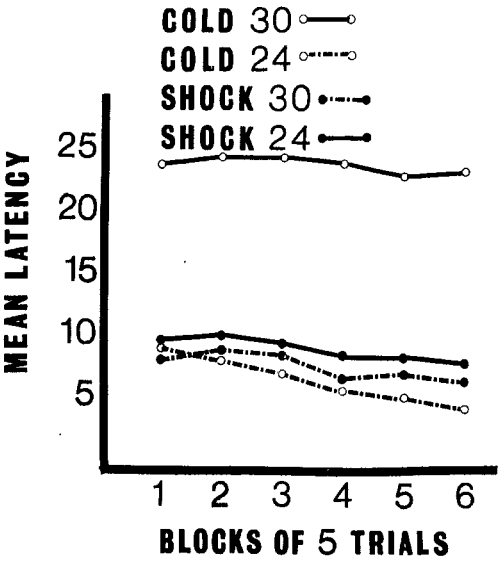

FIG. 9. Mean shuttle box FR-1 escape response latency for rats given either cold swim or inescapable shocks 3 minutes or 24 hours before the shuttle box test (Jackson & Maier, unpublished data 1975). (Only the rats given cold swim 30 minutes before the test failed to respond.)

data reveals that the cold-swim effect was almost absent at 2 hours. However, the 24-hour point is still needed, and Maier has now supplied the missing point. Jackson and Maier (unpublished data) gave rats 3.5 minutes of cold swim in 2 °C water and tested them in a FR-1 shuttle box task 30 minutes or 24 hours later. The results can be seen in Figure 9. Jackson and Maier found the same debilitation as found by Weiss et al. 30 minutes after cold swim, but there was *no deficit at all* if 24 hours had intervened. It should also be noted that the learned helplessness effect in rats, the effects of exposure to uncontrollable shock on subsequent escape acquisition in a task sensitive to controllability, may not show any dissipation with time.

Another aspect of Experiment 3 is worthy of comment. Weiss et al. (1975) note that dogs that were tested 24 hours after exposure to inescapable shock and that failed to learn to escape, would fail to learn again if retested later. That is, the failure became permanent. Weiss et al. retested the rats that failed to learn to escape 30 minutes following cold swim after an additional 48 hours. A deficit was still present and Weiss et al. concluded that this duplicates the corresponding characteristic of the learned helplessness phenomenon in dogs. We do not think so. In the dog experiments, there was no improvement at all during retest; the dogs showed no tendency to learn. This was not true of the rats in the Weiss et al. study. There were 25 trials on the retest, and by the last block of 5 trials the response latency of the cold swim rats was down to about 7 sec, as compared with about 4 sec in a control group. In fact, in a later experiment (Experiment 7) the retest effect disappeared completely, although Weiss et al. did not comment on this aspect of the data.

The final experiment (Experiment 2 in Weiss et al., 1975) said to reveal the identity of the cold swim and learned helplessness effects is the immunization effect. It is worth noting that although Weiss et al. found prior training in the shuttle box to reduce the effects of cold swim on shuttle box performance, the reduction was not complete; some debilitation still occurred. The immunizing effect on learned helplessness is *complete* in both dogs (Seligman & Maier, 1967) and rats (Seligman et al., 1975) when the immunization treatment is given in the shuttle box.

We conclude that the experiments provided by Weiss et al. (1975) do not demonstrate that the behavioral deficit produced by cold swim is the same as the deficit produced by the uncontrollable shocks used in the learned helplessness experiments. Even more convincing to us than the discrepancy between the Weiss et al. and the learned helplessness experiments are our observations of subjects 30 minutes after cold swim, the point at which Weiss et al. tested such subjects. A severe motor debilitation *does* appear to be present. The rats have great difficulty moving and simply remain motionless when placed on a table. Further, such rats have some difficulty righting themselves when placed on their sides. When they are removed from the 2 °C water they are com-

pletely limp. Stone (1970b) has extensively studied the effects of cold swim. Observing the rats after a 20-minute swim in 14.5 °C water, Stone notes:

> Shortly after the stress, the swim stressed rats appear exhausted; they lie in a prostrate position, are unable to move their rear limbs, and have difficulty righting themselves when laid on one side. During the later stages of recovery, the animals remain immobile in a crouched position and show ptosis with continued and violent shivering. (p. 53)

We have observed similar behavior after 3.5 minutes in 2 °C water.

The inescapable shocks used in our learned helplessness experiments do not produce anything like these obvious signs of motor debilitation. As noted previously, the shocks which we use have no effect on runway acquisition 24 hours later, nor do they affect FR-1 shuttle box performance. We doubt that Weiss's subjects could even locomote to the end of a runway 30 minutes after cold swim. Thus we conclude that Weiss et al. (1975) are indeed correct in arguing that cold swim produces a motor deficit, perhaps via norepinephrine depletion. However, there is no reason to believe that the levels of inescapable shock used in the learned helplessness dog or rat experiments produce a motor deficit 24 hours later.

The factors just discussed are also important in interpreting the subsequent experiments by Weiss et al. in which the motor activation deficit hypothesis and the learned helplessness hypothesis were said to make opposed predictions. Experiments 4 and 5 demonstrated that the amount of motor activity required to perform the task response given 30 minutes after cold swim determined whether a deficit occurred, and thus supported the notion that cold swim results in a temporary motor deficit. These experiments do support this notion, but there is nothing in the learned helplessness position which denies that a cold swim produces a motor deficit. The learned helplessness position does not predict this, but there is no reason why it should.

Experiments 6 and 7 of Weiss et al. (1975) use electric shocks during pretreatment, but 150 4.0-mA shocks were delivered whereas the learned helplessness experiments with rats used 64 to 80 1.0-mA shocks. The level of shock used by Weiss et al. is almost of tetanizing intensity and may indeed lead to a trauma-induced motor deficit 30 minutes later. Our dog experiments used 64 6-mA shocks, and testing occurred 24 hours later. It is difficult to compare shock intensities across species as different as dogs and rats, but our observations indicate 4 mA in rats to be much more severe than 6 mA in dogs. Jackson and Maier (unpublished data) investigated whether exposure to shocks characteristic of learned helplessness experiments would produce the sort of deficit reported by Weiss et al. Rats were exposed to 64 1.0-mA shocks and tested in an FR-1 shuttle box task either 30 minutes or 24 hours later. As can be seen in Figure 10, these shocks did not produce a deficit even 30 minutes later. Thus Experiments 6 and 7 employed conditions very different from those employed in the learned helplessness experiments with rats and may indeed be correctly interpreted in terms of a motor deficit. The fact that 150 4.0-mA shocks produce a motor deficit 30 minutes later does not imply that 64 1.0-mA shocks produce a motor deficit 24 hours later.

The conceptual base for Experiments 8 and 9 may indeed be adequate for a test between the motor activation deficit and learned helplessness explanations of the learned helplessness phenomenon. Weiss et al. noted that the reduction in norepinephrine levels produced by exposure to stressors habituates with repeated exposure to the stressor. Thus if inescapable shock produces a learned helplessness effect because it produces a motor deficit through norepinephrine depletion, repeated exposure to inescapable shock should not produce a learned helplessness effect. The learned helplessness hypothesis certainly does not make this prediction. It will be recalled that Weiss et al. found that rats repeatedly exposed to cold swim or to 4.0-mA shocks did indeed perform well when tested on FR-1 shuttling. On the surface this result seems quite deci-

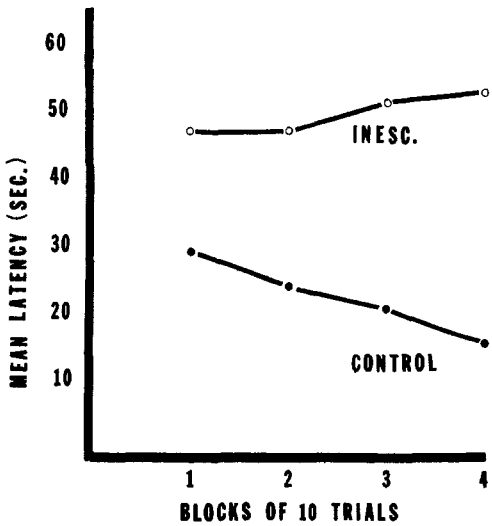

FIGURE 10. Mean FR-3 lever press latencies for rats given 15 days of inescapable shocks or no shock (Seligman & Rosellini, unpublished data). (The inescapable shock group did not learn.)

sive. However, it should be recognized that the Weiss et al. logic demands that repeated exposure to the *same shock conditions* as employed in learned helplessness experiments prevents a deficit on a task *known to be sensitive* to the degree of control which organisms have over initial shock experience.

Thus we have each independently repeated the Weiss et al. (1975) experiment with the shock parameters normally used in our laboratories and with test tasks known to be sensitive to learned helplessness of controllability effects. Rosellini and Seligman (unpublished data) gave one group 15 daily sessions of 1.0-mA inescapable shocks. A second group received no treatment. All three groups were then tested in an FR-3 level pressing task. Maier employed 10 days of exposure to either escapable, inescapable, or no shocks in a wheel-turn apparatus and then tested his subjects in an FR-2 shuttling task. The shock level was 1.0 mA. The results of these experiments can be seen in Figures 10 and 11. It is clear that repeated exposure to 1.0-mA shocks does not reduce the deficit seen in tasks known to be sensitive to learned helplessness effects.

Many of the comments made thus far are also appropriate to Experiments 10 through 12. Recall that in these experiments Weiss et al. (1975) found that pharmacological depletion of monoamines produced poor FR-1 shuttle box performance 30 minutes later, and that pharmacological blockade of monoamine degradation prevented the effects of inescapable shock 30 minutes later in a FR-1 shuttle box task. This is consistent with the notion that Weiss et al. are studying a motor debilitation mediated by monoamines, but as already noted, this may be a very different phenomenon from learned helplessness.

We conclude that the experiments presented by Weiss et al. do not strongly support the motor activation deficit hypothesis as an explanation of the learned helplessness effect. It should be stressed that we are *not* arguing that the learned helplessness hypothesis can account for the data presented by Weiss et al. We think it likely that Weiss et al. were quite correct in their interpretation of their own data. Weiss et al. have demonstrated a number of very interesting effects, but they may not be

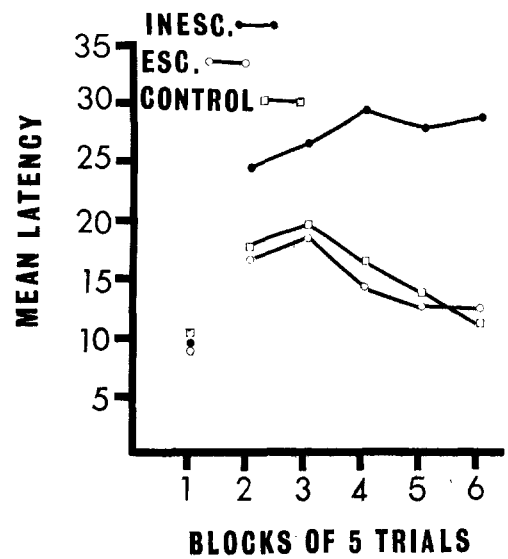

FIGURE 11. Mean FR-2 shuttle box response latencies for rats given 10 days of escapable, yoked inescapable, or no shocks (Maier, unpublished data, 1975). (The inescapable shock subjects failed to learn.)

closely related to learned helplessness effects. The basic similarity is that in both cases animals failed to escape shock, but there may be many roots to producing such a deficit, and they may not reduce to one explanation. The deficit produced by exposure to extremely traumatic events as used by Weiss et al. may be produced by a very different mechanism from the deficits produced by exposure to much less traumatic *uncontrollable* aversive events in the learned helplessness experiments.

Other evidence. There are a number of findings not considered by Weiss et al. which are difficult for the motor activation deficit hypothesis to explain. Many of these have already been described and only a brief discussion is necessary.

1. We have already described experiments by Maier and Testa (1975) which indicate that although inescapably shocked rats are poor at learning FR-2 shuttling, they will learn as well as controls when there is a brief break in shock after the first response of the FR-2. In an analogous fashion, inescapably shocked rats readily learned FR-1 shuttling when shock terminated immediately after the response, but failed to learn when shock termination was briefly delayed. The motor activation deficit hypothesis cannot explain why the behavior of inescapably shocked subjects is so strongly controlled by the nature of the contingency between responding and shock termination in the test task. The same amount of motor activity is required to perform an FR-2 response whether or not there is a brief break in shock, but the outcomes are very different. Similarly, the amount of motor activity required to make a single crossing of a shuttle box is the same whether or not shock termination is slightly delayed, but the outcome is dependent on this factor. Again, mention should be made that Weiss et al. restricted their explanation to learned helplessness in the dog, but this is ad hoc and it does not seem justifiable to restrict consideration to only a small part of the data.

2. Recall that Seligman and Maier (1967) found that prior experience with controllable shock in a shuttle box eliminated the interfering effects of subsequent inescapable shocks on shuttle box performance. The motor activation deficit hypothesis is able to account for this immunizing effect by arguing that pretraining in the shuttle box reduces the amount of motor behavior required during testing because the subject does not have to search for the correct response—it has already been trained. We have presented new data (p. 26) indicating that prior experience with controllable shock has an immunizing effect even when such prior experience is given with a different response from that later used in testing. The motor activation deficit hypothesis cannot explain this effect. Prior training to turn a wheel with the paws should not reduce the amount of motor activity required to find the correct response in a shuttle box.

3. Seligman, Maier, and Geer (1968) and Seligman and Beagley (1975) found that escape deficits in dogs and rats already failing to escape could be eliminated by forcible exposure to the escape contingency. It is hard to see how the motor deficit hypothesis can account for this therapy effect. Does forcible exposure to the escape contingency produce a rapid recovery of norepinephrine levels? It is interesting to note the Abramson and Seligman (unpublished data) have found that forcible exposure to the escape contingency eliminates the behavioral deficit even in rats whose norepinephrine levels have been depleted by injection of α-Methyl-para-tyrosine.

4. We have several times referred to the fact that the learned helplessness effect does not have a time course in dogs when the dogs are given a number of sessions of inescapable shock (Seligman & Groves, 1970) and may not have a time course in rats at all (Seligman & Beagley, 1975). This is directly opposed to the expectations of the motor deficit hypothesis.

5. Hannum et al. (in press) found that rats given four sessions of inescapable shock shortly after weaning failed to learn to escape as adults. Such a failure did not result if the shocks were escapable. Do four sessions

of inescapable shock shortly after weaning produce a permanent depletion of norepinephrine?

6. Although Weiss et al. (1975) have explicitly limited the score of their hypothesis to learned helplessness experiments employing electric shocks to animals (even, on an ad hoc basis, to dogs), mention should be made that there are a large number of experiments outside this domain which can be integrated by the learned helplessness hypothesis but not by the motor activation deficit hypothesis. Examples are the effects of exposure to inescapable noise and unsolvable problems on problem-solving behavior in humans (see p. 13), and the possible analogues to shock-induced learned helplessness produced by delivery of appetitive events independently of behavior (see p. 12) in animals.

Conclusions

We feel that the burden of the evidence is that the motor theories here discussed cannot account for the learned helplessness phenomenon. It seems to us that the central shortcoming of these theories is that they are performance theories. That is, they claim that inescapably shocked subjects are later less likely to emit the test task response, not that they are less likely to learn from an exposure to the contingency between responding and relief if they do respond. The learned helplessness hypothesis also argues for a performance deficit (the reduced incentive motivation proposition), but in addition argues that learning will be undermined (the associative interference proposition). We feel that the Maier and Testa (1975) experiments (see p. 24) offer conclusive evidence that exposure to inescapable shock has an effect on associative processes as well as on motivational processes.

This should *not* be taken to mean that we feel that the processes discussed by Bracewell and Black (1974), Anisman and Waller (1973), and Weiss et al. (1975) do not exist and do not influence behavior. Organisms can, of course, learn motor responses in one situation that are able to interfere with the acquisition of motor responses in a different situation. Further, the data presented by Weiss et al. do convincingly point to the conclusion that severe trauma induces a transient motor deficit perhaps mediated by norepinephrine depletion. What we dispute is that these processes are sufficient to account for all or even many of the learned helplessness effects. We also dispute that these hypotheses handle the data better than the learned helplessness hypothesis, and we feel that a stronger case has been presented for the learned helplessness hypothesis.

It should be noted, however, that the learned helplessness hypothesis is not without its problems. We will list those that loom largest.

1. The time course of learned helplessness effects has yet to be satisfactorily explained. Recall that one session of inescapable shock produces only a transient effect in dogs, while four sessions of inescapable shock seem to produce a more permanent effect. Moreover, there are data to indicate that the learned helplessness effect may be permanent in rats even after one session of inescapable shock.

This pattern of results is difficult to explain. The learned helplessness theory could argue (see pp. 30–31) that previous experience controlling events proactively interferes with the memory that shock is inescapable. Proactive interference is known to increase with time from learning and to decrease with additional training, and so the dog effects can be explained. But, why do rats show a permanent effect after one session? A possibility concerns the rearing conditions of our subjects. Our dogs were not laboratory- or cage-reared, while the rats we used were raised in cages. Perhaps this reduced the likelihood that the rats encountered many immunizing experiences with controllable events. Consistent with the argument, Seligman and Groves (1970) found that only two sessions of inescapable shock were required to produce a permanent learned helplessness effect in cage-reared beagles while four sessions were required to produce a permanent effect in mongrels raised outside the laboratory. However, such an explanation of the time course effects

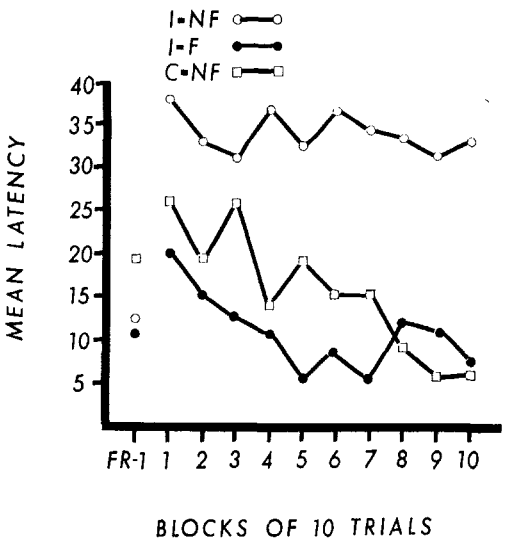

FIG. 12. Mean FR-3 lever press latencies for rats previously given either inescapable (I) or no (C) shocks and then given noise feedback (F) or no feedback (NF) during lever press escape training (Jackson, Tomie, & Maier, Note 7). (Tone feedback eliminated failure to escape.)

is entirely post hoc and is not supported by an independent body of evidence. Additional experimental support will be required before any confidence can be placed in this interpretation.

2. Exposure to inescapable shock produces a deficit on some escape tasks but not on others. Maier et al. (1973) found that exposure to inescapable shock interfered with FR-2 shuttling but not with FR-1 shuttling in rats. Maier et al. were able to account for this difference in terms of the elicited nature of FR-1 shuttling in rats. In an analogous fashion, Seligman and Beagley (1975) found that inescapable shock has no effect on FR-1 lever pressing in rats. However, Seligman and Beagley did not find a differential effect of escapable and inescapable shock on FR-2 lever pressing. A clear learned helplessness effect only emerged with FR-3 lever pressing. It is not obvious why this should be so (Seligman & Beagley, 1975). Further, Weiss et al. (1975) found no effect of inescapable shock on a nose-poke escape response. The Maier et al. explanation does not apply to the Weiss et al. data because a clear acquisition function for nose-poke responding was found.

It might seem that the amount of physical activity involved in these various tasks is a determining factor. However, recall that Maier and Testa (1975) found that inescapably shocked rats learn FR-2 shuttling if a brief break in shock occurs after the first response, and they fail to learn FR-1 shuttling if shock termination is delayed. Here physical activity cannot be a factor. The Maier and Testa experiments suggest that the amount of feedback produced by the response could be a crucial factor. A recent experiment by Jackson, Tomie, and Maier (Note 7) supports this notion. Jackson et al. began by replicating the Seligman FR-3 effect. As shown in Figure 12, rats given inescapable shock were very poor at FR-3 lever press escape responding. Another group of inescapably shocked rats was also tested in the FR-3 lever pressing situation. However, a 100-msec burst of white noise was sounded following each lever press, thus providing feedback. As can be seen in Figure 12, these rats learned rapidly. It should be noted that a warning signal was used in the Weiss et al. (1975) nose-poke experiment. This signal terminated with the response, thus possibly providing feedback. Most interesting is the fact that the only study in the literature (Bracewell & Black, 1974) to find an effect of inescapable shock on FR-1 shuttling in the rat did not use a warning signal. Again, much more work will be needed before any confidence can be placed in such an explanation.

Mention should be made that the helplessness effect has seemed more fragile in rats than in dogs or humans. Both of us have encountered occasional difficulty in producing a reliable phenomenon. The feedback effect discussed previously may help to explain this fragility—we may be using test tasks with a relatively large amount of *intrinsic* feedback.

3. The learned helplessness hypothesis is vague in its specification of boundary conditions. Further, there is little empirical evidence regarding such boundary conditions. Should exposure to loud noise presented in-

dependently of behavior interfere with the acquisition of responses that escape electric shock? Should exposure to uncontrollable shock interfere with the acquisition of responses that procure food? Should exposure to noncontingent food interfere with the acquisition of responses that terminate shock? Should exposure to uncontrollable shock interfere with Pavlovian conditioning? Many further questions of this sort could easily be posed. The learned helplessness hypothesis does not make any predictions regarding the results of such experiments. Further, there are very few experiments directed at answering these questions. The learned helplessness hypothesis will have to become more specific, and experiments designed to delineate these boundary conditions will have to be conducted.

4. A final problem concerns the relationship between the subject's perception of independence between responding and outcome and the conditions which lead to such a perception (see p. 17). In our experiments we have arranged conditions in which the relationship between responding and outcome is objectively independent. However, subjects do not always respond to random relationships as if they were random[6] (e.g., Bruner & Revusky, 1961; Hake & Hyman, 1953; Naylor & Clark, 1968), and it is well known that subjective probabilities do not reflect objective probabilities with accuracy (cf. Kahneman & Tversky, 1972). Thus we will have to specify the conditions under which the perception of independence develops—only these conditions should lead to learned helplessness.

Before closing a few comments are in order.

It must seem to the reader that a great deal of theoretical confusion surrounds the learned helplessness phenomenon. A number of investigators have proposed alternative accounts of the same behavioral phenomenon and have presented experimental evidence which they feel supports their position and refutes the learned helplessness hypothesis. We, on the other hand, have questioned the implications of these experiments for the learned helplessness hypothesis. Why all this confusion? We feel that a large part of this confusion stems from an error of inference noted previously. The logical structure of many of the experiments which have been presented as inconsistent with the learned helplessness hypothesis have the following form: (a) A procedure is found which produces an escape deficit, (b) this procedure is hard to interpret in terms of the learning of independence between responding and shock termination, and (c) therefore the learned helplessness hypothesis is contradicted. This seems to us to be poor logic. The learned helplessness hypothesis does not argue that all escape deficits are produced by learning that responding and shock termination are independent. There are undoubtedly many ways to produce poor escape performance and there is no reason to expect that all of these operate through a single mechanism.

Just as there may be many different ways to produce escape-learning deficits, exposure to inescapable shock may itself produce a variety of changes in the organism, not just one. Under some conditions, organisms might learn a motor response, stress might be induced, and the organism might learn that its behavior is independent of shock termination. A complete explanation of the influence of exposure to inescapable aversive events on later behavior might involve a consideration of each of these factors. Thus we do not view the various theoretical positions that have been here discussed as incompatible: They might be complementary. The existence of one of these processes does not imply the nonexistence of the others. We feel that we have presented convincing evidence for the reality of the processes specified by the learned helplessness hypothesis; however, this evidence does not imply that other processes do not also occur.

It might seem that this makes the learned helplessness hypothesis difficult to test, but this is not so. A variety of predictions made

---

[6] Thanks go to Christopher Peterson for these references.

by the learned helplessness hypothesis have been tested and confirmed (e.g., Hiroto & Seligman, 1975; Maier, 1970; Maier et al., 1973; Maier & Testa, 1975; Overmier & Seligman, 1967; Seligman & Beagley, 1975; Seligman & Groves, 1970; Seligman & Maier, 1967; Seligman, Maier, & Geer, 1968; Testa, Juraska, & Maier, 1974) Failures of these predictions would have been evidence against the learned helplessness position. In addition there are many new ways in which the hypothesis could be tested. For example the *logic,* if not the execution, underlying Experiments 8 and 9 of Weiss et al. (1975) seems to us to be adequate to test the learned helplessness position. The coming years should see a number of such tests and a more detailed elaboration of the learned helplessness hypothesis.

Finally, we would like to say a word about the cognitive nature of our position. The learned helplessness hypothesis has been stated in cognitive languages, whereas most of the alternative views have been stated in S–R language. We have found it difficult to even approach the sort of phenomena that we have tried to explain within an S–R framework, and have found the cognitive theorizing to be more fruitful and to reflect more accurately those processes that we feel to be reflected in behavior. Many other investigators of learning and motivation in lower organisms seem to have reached a similar conclusion (e.g., Bolles, 1972; Irwin, 1971; Kamin, 1969; Seligman & Johnston, 1973; Terry & Wagner, 1975; Wagner, Rudy, & Whitlow, 1973). The next few years should determine the value of such an approach.

REFERENCES NOTES

1. Miller, W., & Seligman, M. E. P. *Depression, learned helplessness, and the perception of reinforcement.* Manuscript submitted for publication, 1975.
2. James, W. H. *DeKalb survey tests: Student opinion survey form I-E, I.* Author's copyright, 1963.
3. Anderson, D. C., Schwendiman, G., & Payne, R. *Prior unsignalled inescapable shock trauma in the rat.* Paper presented at the meeting of the Western Psychological Association, San Diego, March 1968.
4. Carder, B., & Leaf, R. Personal communication, 1973.
5. Weisman, R. Personal communication, 1974.
6. Weiss, J. M., Pohorecky, L. A., Dorros, K., Williams, S., Emmel, D., Whittlesey, M., & Case, E. *Coping behavior and brain norepinephrine turnover.* Paper presented at the meeting of the Eastern Psychological Association, Washington, D.C., May 1973.
7. Jackson, R. L., Tomie, A., & Maier, S. F. *Learned helplessness in rats: Effects of response produced feedback.* Paper presented at Rocky Mountain Psychological Association, Salt Lake City, May 1975.

REFERENCES

Amsel, A., Rashotte, N. E., & MacKinnon, J. R. Partial reinforcement effects within subject and between subjects. *Psychological Monographs,* 1966, *80* (20, Whole No. 628).

Anderson, D. C., Cole, J., & McVaugh, W. Variations in unsignaled inescapable preshock as determinants of responses to punishment. *Journal of Comparative and Physiological Psychology,* 1968, *65* (3, Pt. 2).

Anderson, D. C., & Paden, P. Passive avoidance response learning as a function of prior tumbling trauma. *Psychonomic Science,* 1966, *4,* 129–130.

Anisman, H. Effects of pretraining compatible and incompatible responses on subsequent one-way and shuttle-avoidance performance in rats. *Journal of Comparative and Physiological Psychology,* 1973, *82,* 95–104.

Anisman, H., & Waller, T. G. Facilitative and disruptive effect of prior exposure to shock on subsequent avoidance performance. *Journal of Comparative and Physiological Psychology,* 1972, *78,* 113–122.

Anisman, H., & Waller, T. G. Effects of inescapable shock on subsequent avoidance performance: Role of response repertoire changes. *Behavioral Biology,* 1973, *9,* 331–355.

Averill, J. R. Personal control over aversive stimuli and its relationship to stress. *Psychological Bulletin,* 1973, *80,* 286–303.

Averill, J. R., & Rosenn, M. Vigilant and nonvigilant coping strategies and psychophysiological stress reactions during the anticipation of electric shock. *Journal of Personality and Social Psychology,* 1972, *23,* 128–141.

Bainbridge, P. L. Learning in the rat: Effect of early experience with an unsolvable problem. *Journal of Comparative and Physiological Psychology,* 1973, *82,* 301–307.

Bandler, R. J., Jr., Madaras, G. R., & Bem, D. J. Self-observation as a source of pain perception. *Journal of Personality and Social Psychology,* 1968, *9,* 205–209.

Behrend, E. R., & Bitterman, M. E. Sidman avoidance in the fish. *Journal of the Experimental Analysis of Behavior,* 1963, *13,* 229–242.

Bintz, J. Between- and within-subject effect of shock intensity on avoidance in goldfish (*Carassius auratus*). *Journal of Comparative and Physiological Psychology,* 1971, *75,* 92–97.

Black, A. H., Young, G. A., & Batenchuk, C. Avoidance training of hippocampal theta waves in flaxedilized dogs and its relation to movement. *Journal of Comparative and Physiological Psychology,* 1970, *70,* 15–24.

Bolles, R. C. Species-specific defense reactions in avoidance learning. *Psychological Review,* 1970, *77,* 32–48.

Bolles, R. C. Reinforcement, expectancy, and learning, *Psychological Review,* 1972, *79,* 394–409.

Bracewell, R. J., & Black, A. H. The effects of restraint and noncontingent pre-shock on subsequent escape learning in the rat. *Learning and Motivation,* 1974, *5,* 53–69.

Brady, J. V., Porter, R. W., Conrad, D. G., & Mason, J. W. Avoidance behavior and the development of gastroduodenal ulcers. *Journal of the Experimental Analysis of Behavior,* 1958, *1,* 69–72.

Braud, W., Wepmann, B., & Russo, D. Task and species generality of the "helplessness" phenomenon. *Psychonomic Science,* 1969, *16,* 154–155.

Brimer, C. J., & Kamin, L. J. Disinhibition, habituation, sensitization and the conditioned emotional response. *Journal of Comparative and Physiological Psychology,* 1963, *56,* 508–516.

Brookshire, K. H., Littman, R. A., & Stewart, C. N. Residue of shock trauma in the white rat: A three factor theory. *Psychological Monographs,* 1961, *75,* (10, Whole No. 514).

Brown, P., & Jenkins, H. M. Autoshaping of the pigeons key-peck. *Journal of the Experimental Analysis of Behavior,* 1968, *11,* 1–8.

Bruner, A., & Revusky, S. H. Collateral behavior in humans. *Journal of the Experimental Analysis of Behavior,* 1961, *4,* 349–350.

Catania, A. C. Elicitation, reinforcement, and stimulus control. In R. Glaser (Ed.), *The nature of reinforcement.* New York: Academic Press, 1971.

Church, R. M. Response suppression. In B. A. Campbell & R. M. Church (Eds.), *Punishment and aversive behavior.* New York: Appleton-Century-Crofts, 1969.

Church, R. M., LoLordo, V. M., Overmier, J. B., Solomon, R. L., & Turner, L. H. Cardiac responses to shock in curarized dogs: Effects of shock intensity and duration, warning signal, and prior experience with shock. *Journal of Comparative and Physiological Psychology,* 1966, *62,* 1–7.

Cohen, P. S. Interference effects of escapable shock upon subsequent acquisition of escape-avoidance responding. *Journal of Comparative and Physiological Psychology,* 1970, *71,* 484–486.

Corah, N. L., & Boffa, J. Perceived control, self-observation, and response to aversive stimulation. *Journal of Personality and Social Psychology,* 1970, *16,* 1–4.

D'Amato, M. Delayed matching and short-term memory in monkeys. In G. H. Bower, (Ed.), *The psychology of learning and motivation* (Vol. 7). New York: Academic Press, 1973.

Desiderato, O., & Newman, A. Conditioned suppression produced in rats by tones paired with escapable or inescapable shock. *Journal of Comparative and Physiological Psychology,* 1971, *77,* 427–443.

De Toledo, L., & Black, A. H. Effects of preshock on subsequent avoidance conditioning. *Journal of Comparative and Physiological Psychology,* 1967, *63,* 493–499.

Dinsmoor, J. A., & Campbell, S. L. Escape-from-shock training following exposure to inescapable shock. *Psychological Reports,* 1956, *2,* 43–49. (a)

Dinsmoor, J. A., & Campbell, S. L. Level of current and time between sessions as factors in "adaptation" to shock. *Psychological Reports* 1956, *2,* 441–444. (b)

Dweck, C. S. The role of expectations and attributions in the alleviation of learned helplessness. *Journal of Personality and Social Psychology,* 1975, *31,* 674–685.

Dweck, C. S., & Repucci, N. D. Learned helplessness and reinforcement responsibility in children. *Journal of Personality and Social Psychology,* 1973, *25,* 109–116.

Elliot, R. Tonic heart rate: Experiments on the effects of collative variables lead to an hypothesis about its motivational significance. *Journal of Personality and Social Psychology,* 1969, *12,* 211–228.

Engberg, L. A., Hansen, G., Welker, R. L., & Thomas, D. R. Acquisition of key-pecking via autoshaping as a function of prior experience: "Learned laziness"? *Science,* 1973, *178,* 1002–1004.

Ferster, C. B., & Skinner, B. F. *Schedules of Reinforcement.* New York: Appleton-Century-Crofts, 1957.

Fosco, F., & Geer, J. H. Effects of gaining control over aversive stimuli after differing amounts of no control. *Psychological Reports,* 1971, *29,* 1153–1154.

Frumkin, K., & Brookshire, K. H. Conditioned fear training and later avoidance learning in goldfish. *Psychonomic Science,* 1969, *16,* 159–160.

Gamzu, E. R., Williams, D. A., & Schwartz, B Pitfalls of organism and concept: "Learned laziness"? *Science,* 1973, *101,* 367–368.

Geer, J. H., Davison, G. C., & Gatchel, R. I. Reduction of stress in humans through nonveridical perceived control of aversive stimulation. *Journal of Personality and Social Psychology,* 1970, *16,* 731–738.

Geer, J. H., & Maisel, E. Evaluating the effects of the prediction-control confound. *Journal of Personality and Social Psychology,* 1972, *23,* 314–319.

Gibbon, J., Berryman, R., & Thompson, R. L. Contingency spaces and measures in classical and instrumental conditioning. *Journal of the Experimental Analysis of Behavior,* 1974, *21,* 585–605.

Glass, D. C., & Singer, J. E. *Urban stress: Experiments on noise and social stressors.* New York: Academic Press, 1972.

Goeckner, D. J., Greenough, W. T., & Maier, S. F. Escape learning deficit after overcrowded rearing in rats: Test of a helplessness hypothesis. *Bulletin of the Psychonomic Society,* 1974, *3,* 54–56.

Hake, H. W., & Hyman, R. Perception of the statistical structure of a random series of binary symbols. *Journal of Experimental Psychology,* 1953, *45,* 64–74.

Hannum, R. D., Rosellini, R. A., & Seligman, M. E. P. Retention and immunization of learned helplessness from weaning to adulthood. *Developmental Psychology,* in press.

Hearst, E. Stress induced breakdown of an appetitive discrimination. *Journal of the Experimental Analysis of Behavior,* 1965, *8,* 135–146.

Herman, Z. The effects of noradrenaline on rat's behaviour. *Psychopharmacologia,* 1970, *16,* 369–374.

Hiroto, D. S. Locus of control and learned helplessness. *Journal of Experimental Psychology,* 1974, *102,* 187–193.

Hiroto, D. S., & Seligman, M. E. P. Generality of learned helplessness in man. *Journal of Personality and Social Psychology,* 1975, *31,* 311–327.

Hokanson, J. E., DeGood, D. E., Forrest, M. S., & Brittain, T. M. Availability of avoidance behaviors in modulating vascular-stress responses. *Journal of Personality and Social Psychology,* 1971, *19,* 60–68.

Honig, W. K. (Ed.). *Operant conditioning: Research and application.* New York: Appleton-Century-Crofts, 1966.

Irwin, F. W. *Intentional behavior and motivation: A cognitive theory.* Philadelphia: Lippincott, 1971.

Kahneman, D., & Tversky, A. Subjective probability: A judgment of representativeness. *Cognitive Psychology,* 1972, *3,* 430–454.

Kamin, L. J. Predictability, surprise, attention, and conditioning. In B. A. Campbell & R. M. Church (Eds.), *Punishment and aversive behavior,* New York: Appleton-Century-Crofts, 1969.

Kelley, H. H. Attribution theory in social psychology. In D. Levine (Ed.), *Nebraska Symposium on Motivation.* Lincoln: University of Nebraska Press, 1967.

Kelley, H. H. *Causal schemata and the attribution process.* Morristown, N. J.: General Learning Press, 1972.

Kemler, D., & Shepp, B. The learning and transfer of dimensional relevance and irrelevance in children. *Journal of Experimental Psychology,* 1971, *90,* 120–127.

Klein, D. C., Fencil-Morse, E., & Seligman, M. E. P. Depression, learned helplessness, and the attribution of failure. *Journal of Personality and Social Psychology,* in press.

Krantz, D. S., Glass, D. C., & Snyder, M. L. Helplessness, stress level, and the coronary prone behavior pattern. *Journal of Experimental Social Psychology,* 1974, *10,* 284–300.

Langer, E. J. *The illusion of control.* Unpublished doctoral dissertation, Yale University, 1974.

Lazarus, R. S. *Psychological stress and the coping process.* New York: McGraw-Hill, 1966.

Lefcourt, H. N. Internal versus external control of reinforcement: A review. *Psychological Bulletin,* 1966, *65,* 206–220.

Liddell, H. S., James, W. T., & Anderson, O. D. The comparative physiology of the conditioned motor reflex: Based on experiments with the pig, dog, sheep, goat and rabbit. *Comparative Physiology Monograph,* 1934, *11*(1), Serial No. 89.

Lindner, M. *Hereditary and environmental influences upon resistance to stress.* Unpublished doctoral dissertation, University of Pennsylvania, 1968.

Looney, T. A., & Cohen, P. S. Retardation of jump-up escape responding in rats pretreated with different frequencies of noncontingent electric shock. *Journal of Comparative and Physiological Psychology,* 1972, *78,* 317–322.

Loucks, R. B. The experimental delimitation of neural structures essential for learning: The attempt to condition striped muscle responses with faradization of the sigmoid gyri. *Journal of Psychology,* 1935, *1,* 5–44.

MacKintosh, N. J. Stimulus selection: Learning to ignore stimuli that predict no change in reinforcement. In R. A. Hindle & J. Stevenson-Hinde (Eds.), *Constraints on learning.* New York: Academic Press, 1973.

Maier, S. F. Failure to escape traumatic shock: Incompatible skeletal motor response or learned helplessness? *Learning and Motivation,* 1970, *1,* 157–170.

Maier, S. F., Albin, R. W., & Testa, T. J. Failure to learn to escape in rats previously exposed to inescapable shock depends on nature of escape response. *Journal of Comparative and Physiological Psychology,* 1973, *85,* 581–592.

Maier, S. F., Allaway, T. A., & Gleitman, H. Proactive inhibition in rats after prior reversal: A critique of the spontaneous recovery hypothesis. *Psychonomic Science,* 1967, *9,* 63–65.

Maier, S. F., Anderson, C., & Lieberman, D. A. Influence of control of shock on subsequent shock-elicited aggression. *Journal of Comparative and Physiological Psychology,* 1972, *81,* 94–100.

Maier, S. F., & Gleitman, H. Proactive interference in rats. *Psychonomic Science,* 1967, *7,* 25–26.

Maier, S. F., Seligman, M. E. P., & Solomon, R. L. Pavlovian fear conditioning and learned helplessness. In B. A. Campbell & R. M. Church (Eds.), *Punishment.* New York: Appleton-Century-Crofts, 1969.

Maier, S. F., & Testa, T. J. Failure to learn to escape by rats previously exposed to inescapable shock is partly produced by associative interference. *Journal of Comparative and Physiological Psychology,* 1975, *88,* 554–564.

Masserman, J. H. *Behavior and neurosis.* Chicago: University of Chicago Press, 1943.

Masserman, J. H. The principle of uncertainty in Neurotegenesis. In H. D. Kimmel (Ed.), *Experimental psychopathology.* New York: Academic Press, 1971.

McCulloch, T. L., & Bruner, J. S. The effects of electric shock upon subsequent learning in the rat. *Journal of Psychology,* 1939, *7,* 333–336.

Mellgren, R. L., & Ost, J. W. P. Discriminative stimulus preexposure and learning of an operant discrimination in the rat. *Journal of Comparative and Physiological Psychology,* 1971, *77,* 179–187.

Miller, S., & Konorski, J. Sur une forme particuliere des reflexes conditionels. *Comptes rendus hebdomadaires des séances et mémoires de la Société de Biologie et filiales,* 1928, *99,* 1155–1157.

Miller, W., & Seligman, M. E. P. Depression in humans. *Journal of Abnormal Psychology,* 1975, *84,* 228–238.

Miller, W., & Weiss, J. M. Effects of somatic or visceral responses to punishment. In B. A. Campbell & R. M. Church (Eds.), *Punishment and aversive behavior.* New York: Appleton-Century-Crofts, 1969.

Moore, B. R. The role of directed Pavlovian reactions in simple instrumental learning in the pigeon. In R. A. Hinde & J. Stevenson-Hinde (Eds.), *Constraints on learning.* New York: Academic Press, 1973.

Moot, S. A., Ceballa, R. P., & Crabtree, J. M. Instrumental control and ulcertation in rats. *Journal of Comparative and Physiological Psychology,* 1970, *71,* 405–410.

Mowrer, O. H., & Viek, P. An experimental analogue of fear from a sense of helplessness. *Journal of Abnormal and Social Psychology,* 1948, *43,* 193–200.

Mullin, A. D., & Mogenson, G. J. Effects of fear conditioning on avoidance learning. *Psychological Reports,* 1963, *13,* 707–710.

Naylor, J. C., & Clark, R. D. Intuitive interference strategies in interval learning tasks as a function of validity magnitude and sign. *Organizational Behavior and Human Performance.* 1968, *3,* 378–399.

Overmier, J. B. Interference with avoidance behavior: Failure to avoid traumatic shock. *Journal of Experimental Psychology,* 1968, *78,* 340–343.

Overmier, J. B., & Seligman, M. E. P. Effects of inescapable shock upon subsequent escape and avoidance learning. *Journal of Comparative and Physiological Psychology,* 1967, *63,* 28–33.

Padilla, A. M. Effects of prior and interpolated shock exposures on subsequent avoidance learning by goldfish. *Psychological Reports,* 1973, *32,* 451–456.

Padilla, A. M., Padilla, C., Ketterer, T., & Giacolone, D. Inescapable shocks and subsequent avoidance conditioning in goldfish (*Carrasius auratus*). *Psychonomic Science,* 1970, *20,* 295–296.

Pavlov, I. P. *Conditioned reflexes.* New York: Dover, 1927.

Payne, R. J. Alterations of Sidman avoidance baselines by CS paired with avoidable or unavoidable shock. *Psychological Reports,* 1972, *31,* 291–294.

Payne, R., Anderson, D. C., & Murcurio, J. Preshock-produced alterations in pain-elicited fighting. *Journal of Comparative and Physiological Psychology,* 1970, *71,* 258–266.

Pervin, L. A. The need to predict and control under conditions of threat. *Journal of Personality,* 1963, *31,* 570–585.

Powell, D. A., & Creer, T. L. Interaction of developmental and environmental variables in shock-elicited aggression. *Journal of Comparative and Physiological Psychology,* 1969, *69,* 219–225.

Racinskas, J. R. *Maladaptive consequences of loss or lack of control over aversive events.* Unpublished doctoral dissertation, Waterloo University, Ontario, Canada, 1971.

Rech, R. H., Bovys, H. K., & Moore, K. Alterations in behavior and brain catecholamine levels in rats treated with α-methyltyrosine. *Journal of Pharmacology and Experimental Therapeutics,* 1966, *153,* 412–419.

Rescorla, R. A. Pavlovian conditioning and its proper control procedures. *Psychological Review,* 1967, *74,* 71–79.

Rescorla, R. A. Probability of shock in the presence and absence of the CS in fear conditioning. *Journal of Comparative and Physiological Psychology,* 1968, *66,* 1–5.

Rodin, J. Overcrowding, perceived choice and response to controllable and uncontrollable outcomes. *Journal of Experimental Social Psychology,* in press.

Rosellini, R., & Seligman, M. E. P. Learned helplessness and escape from frustration. *Journal of Experimental Psychology: Animal Behavior Processes,* 1975, *1,* 149–158.

Roth, S. *The effects of experimentally induced expectancies of control: Facilitation of controlling behavior or learned helplessness?* Unpublished doctoral dissertation, Northwestern University, 1973.

Roth, S., & Bootzin, R. R. Effects of experimentally induced expectancies of external control: An investigation of learned helplessness. *Journal of Personality and Social Psychology,* 1974, *29,* 253–264.

Roth, S., & Kubal, L. The effects of noncontingent reinforcement on tasks of differing importance: Facilitation and learned helplessness effects. *Journal of Personality and Social Psychology,* 1975, *32,* 680–691.

Rotter, J. B. Generalized expectancies for internal versus external control of reinforcement. *Psychological Monographs,* 1966, *80,* (1, Whole No. 609).

Schwartz, B., & Williams, D. R. Two different kinds of key peck in the pigeon: Some properties of responses maintained by negative and positive response-reinforcer contingencies. *Journal of the Experimental Analysis of Behavior,* 1972, *18,* 201–216.

Segal, D. S., Knapp, S., Kuczenski, R. T., & Mandell, A. J., The effects of environmental isolation on behavior and regional rat brain tyrosine hydroxylase and tryptophan hydroxylase activities. *Behavioral Biology,* 1973, *8,* 47–53.

Seligman, M. E. P. Helplessness. *On depression, development, and death.* San Francisco: W. H. Freeman, 1975.

Seligman, M. E. P., & Beagley, G. Learned helplessness in the rat. *Journal of Comparative and Physiological Psychology,* 1975, *88,* 534–541.

Seligman, M. E. P., & Groves, D. Non-transient learned helplessness. *Psychonomic Science,* 1970, *19,* 191–192.

Seligman, M. E. P., & Johnston, J. C. A cognitive theory of avoidance learning. In F. J. McGuigan & D. B. Lumsden (Eds.), *Contemporary approaches to conditioning and learning.* Washington, D. C.: V. H. Winston, 1973.

Seligman, M. E. P., & Maier, S. F. Failure to escape traumatic shock. *Journal of Experimental Psychology,* 1967, *74,* 1–9.

Seligman, M. E. P., Maier, S. F., & Geer, J. The alleviation of learned helplessness in the dog. *Journal of Abnormal and Social Psychology,* 1968, *73,* 256–262.

Seligman, M. E. P., Maier, S. F., & Solomon, R. L. Unpredictable and uncontrollable aversive events. In F. R. Brush (Ed.), *Aversive conditioning and learning.* New York: Academic Press, 1971.

Seligman, M. E. P., Rosellini, R. A., & Kozak, M. Learned helplessness in the rat: Reversibility, time course, and immunization. *Journal of Comparative and Physiological Psychology,* 1975, *88,* 542–547.

Seward, J. P., & Humphrey, G. L. Avoidance learning as a function of pretraining in the cat. *Journal of Comparative and Physiological Psychology,* 1967, *63,* 338–341.

Sines, J. O., Cleeland, C., & Adkins, J. The behavior of normal and stomach lesion susceptible rats in several learning situations. *Journal of Genetic Psychology,* 1963, *102,* 91–94.

Spear, N. E. Retrieval of memory in animals. *Psychological Review,* 1973, *80,* 163–194.

Stone, E. A. Behavioral and neurochemical effects of acute swim stress are due to hypothermia. *Life Sciences,* 1970, *9,* 877–888. (a)

Stone, E. A. Swim-stress-induced inactivity: Relation to body temperature and brain norepinephrine, and effects of *d*-amphetamine. *Psychosomatic Medicine,* 1970, *32,* 51–59. (b)

Stotland, E. *The psychology of hope.* San Francisco: Jossey-Bass, 1969.

Stotland, E., & Blumenthal, A. The reduction of anxiety as a result of the expectation of making a choice. *Canadian Journal of Psychology,* 1964, *18,* 139–145.

Stroebel, C. F. Biologic rhythm correlates of disturbed behavior in the rhesus monkey. *Bibliotheca Primatologica,* 1969, *9,* 91–105.

Terry, W. S., & Wagner, A. R. Short-term memory for "surprising" versus "expected" unconditioned stimuli in Pavlovian conditioning. *Journal of Experimental Psychology: Animal Behavior Process,* 1975, *1,* 122–134.

Testa, T. J., Juraska, J. M., & Maier, S. F. Prior exposure to inescapable electric shock in rats effects extinction behavior after the successful acquisition of an escape response. *Learning and Motivation,* 1974, *5,* 380–392.

Thomas, D. R., Freeman, F., Svinicki, J. G., Burr, D. E. S., & Lyons, J. Effects of extradimensional training on stimulus generalization. *Journal of Experimental Psychology Monograph,* 1970, *83,* (1, Pt. 2).

Thomas, E., & Balter, A. Learned helplessness: Amelioration of symptoms by cholinergic blockade of the septum. *Science,* in press.

Thornton, J. W., & Jacobs, P. D. Learned helplessness in human subjects. *Journal of Experimental Psychology,* 1971, *87,* 369–372.

Wagner, A. R. Stimulus selection and a "modified continuity theory". In G. H. Bower & J. T. Spence (Eds.), *The psychology of learning and motivation* (Vol. 3). New York: Academic Press, 1969.

Wagner, A. R., Rudy, J. W., & Whitlow, J. W. Rehearsal in animal conditioning. *Journal of Experimental Psychology,* 1973, *97,* 407–426.

Weiner, B., Frieze, I., Kukla, A., Reed, L., Rest, S., & Rosenbaum, R. M. *Perceiving the causes of success and failure.* Morristown, N. J.: General Learning Press, 1971.

Weiss, J. M. Effects of coping responses on stress. *Journal of Comparative and Physiological Psychology,* 1968, *65,* 251–260.

Weiss, J. M. Effects of coping behavior in different warning signal conditions on stress pathology in rats. *Journal of Comparative and Physiological Psychology,* 1971, *77,* 1–13. (a)

Weiss, J. M. Effects of coping behavior with and without a feedback signal on stress pathology in rats. *Journal of Comparative and Physiological Psychology,* 1971, *77,* 22–30. (b)

Weiss, J. M. Effects of punishing the coping response (conflict) on stress pathology in rats. *Journal of Comparative and Physiological Psychology,* 1971, *77,* 14–21. (c)

Weiss, J. M., Glazer, H. I., & Pohorecky, L. A. Coping behavior and neurochemical changes: An alternative explanation for the original "learned helplessness" experiments. In *Relevance of the psychopathological animal model to the human.* New York: Plenum Press, 1975.

Weiss, J. M., Krieckhaus, E. E., & Conte, R. Effects of fear conditioning on subsequent avoidance behavior. *Journal of Comparative and Physiological Psychology,* 1968, *65,* 413–421.

Weiss, J. M., Stone, E. A., & Harrell, N. Coping behavior and brain norepinephrine in rats. *Journal of Comparative and Physiological Psychology,* 1970, *72,* 153–160.

Welker, R. E. *"Learned laziness": A replication and extension.* Unpublished doctoral dissertation, University of Colorado, 1974.

Woodbury, C. B. A note on "passive" conditioning. *Journal of General Psychology,* 1942, *27,* 359–361.

Zielinski, K., & Soltysik, S. The effect of pretraining on the acquisition and extinction of the avoidance reflex. *Acta Biologiae Experimentalis,* 1964, *24,* 73–87.

Zigmond, M., & Harvey, J. Resistance to central norepinephrine depletion and decreased mortality in rats chronically exposed to electric foot shock. *Journal of Neuro-Visceral Relations,* 1970, *31,* 373–381.

(Received June 30, 1975)