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Modeling Signal Features of Escape Response: Effects of Cessation Conditioning in “Learned Helplessness” Paradigm

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Six experiments examined the effects of signaling the termination of inescapable shock (cessation conditioning) or shock-free periods (backward conditioning) on later escape deficits in the learned helplessness paradigm, using rats (Sprague-Dawley and Bantin-Kingman). A cessation signal prevented later performance deficits when highly variable inescapable shock durations were used during pretreatment. The inclusion of short minimum intertrial intervals during pretreatment did not alter the benefits of cessation conditioning but eliminated the protection afforded by a safety signal. The beneficial effects of both cessation and backward signals were eliminated when a single stimulus signaled shock termination and a shock-free period. Finally, a combination of cessation and backward signals was found to be most effective in immunizing against the effects of subsequent unsignaled, inescapable shock on later escape performance. These data suggest that cessation conditioning may be crucial to the prophylactic action of an escape response.

Exposure to unsignaled, inescapable electric shock impairs later measures of cognitive (e.g., Jackson, Alexander, & Maier, 1980), vegetative (e.g., Dess, Minor, & Brewer, 1989; Weiss, Goodman, Ambrose, Webster, & Hoffman, 1984), and motor performance (e.g., Anisman, deCatanzaro, & Remington, 1978; Weiss, Stone, & Harrell, 1970) in rats and other species. This learned helplessness effect (Maier & Seligman, 1976; Overmier & Seligman, 1967) or distress syndrome (Minor, Dess, & Overmier, in press) is prevented if a subject is able to escape shock or exert some form of behavioral control over the stressor. Although research in this paradigm historically has focused on the adverse impact of uncontrollable aversive events, attention recently has shifted to the escape response, the mechanisms underlying its prophylactic action, and the issue of whether behavioral control is uniquely stress reducing (e.g., Jackson & Minor, 1988; Mineka, Cook, & Miller, 1984; Minor et al., in press; Overmier, 1988; Volpicelli, Ulm, Alexander, & Seligman, 1983; Warren, Rosellini, & Maier, in press).

One explanation of escape responding views the instrumental contingency as central to its stress-reducing capacity. Learned helplessness theorists (Maier & Seligman, 1976; Seligman, Maier, & Solomon, 1971) have argued that rats learn the independence between responding and outcomes during exposure to inescapable shock and develop an expectation that future events will be similarly uncontrollable. This expectation of helplessness generalizes to later test tasks to serve as a cognitive mediator of associative and motivational deficits. Escapable shock, on the other hand, engenders expectations of control or mastery (e.g., Maier & Seligman, 1976; Volpicelli et al., 1983), which are incompatible with cognitions of helplessness and thereby eliminate the cause of test impairment. From this view, behavioral control should be uniquely suited to reducing the distress and performance deficits associated with uncontrollable shock.

Alternatively, stress reduction could be accomplished as an emergent consequence of escape responding. For instance, Weiss (1971a, 1971b) attributed the benefits of behavioral control to the stimulus properties of responding. Escape responses were assumed to generate “relevant feedback,” defined as stimuli correlated with the absence of the stressor, to mitigate the impact of traumatic shock. Recent elaborations on this view argue that such proprioceptive stimuli acquire fear-inhibiting properties because of their consistent association with shock termination and a shock-free period to reduce chronic fear, the principal cause of helplessness (Jackson & Minor, 1988; Mineka et al., 1984; Minor et al., in press).

According to this account, subjects are unable to discriminate between periods of danger and safety during exposure to unsignaled, inescapable shock and remain chronically afraid during the interval between shock trials as a result (Mower & Vick, 1948; Seligman, 1968). Prolonged exposure to this condition results in asymptotic context fear conditioning and mobilizes nonassociative, endogenous processes that contribute to later performance deficits (Minor & LoLordo, 1984; Weiss et al., 1981). An escape response prevents helplessness because stimuli generated by the act of escaping acquire safety-signal properties, thereby reducing the total amount of time spent in fear during the pretreatment session. Less fear is conditioned to the pretreatment context, and the nonassociative consequences of chronic fear are prevented.

This feedback hypothesis suggests that behavioral control should be only one of several operations that modulate the
The greatest benefit of an escape response may stem from the production of stimuli that predict shock termination. Mowrer (1960) suggested that stimuli signaling the cessation of shock should elicit a relief reaction and acquire secondary-reinforcing properties. If so, cessation conditioning could serve as a source of fear inhibition that is not represented by a safety signal and accounts for the added benefit of behavioral control in the above studies.

Unfortunately, data on cessation conditioning are scant and equivocal. Some studies suggest that a stimulus presented shortly before the end of each conditioning trial acquires fear-inhibitory properties (Moscovitch, 1972) and directly mitigates the unconditioned reaction to electric shock (Segundo, Galeano, Sommer-Smith, & Roig, 1961). Cessation stimuli also appear to have some of the properties of an escape response that are not represented by a backward signal–shock relation (Moscovitch, 1972). However, other investigators have suggested that the procedure results in excitatory conditioning (Mowrer & Aiken, 1954) and retards inhibitory backward conditioning (Moscovitch & LoLordo, 1968). Thus, although the ability to predict shock termination could contribute importantly to the prophylactic action of an escape response, the robustness of cessation conditioning and its relevance to helplessness phenomena are unclear.

The present study examined the effects of signaling the cessation of inescapable shock during pretreatment on later shuttle-escape performance, a traditional measure of helplessness in rats (Maier, Albin, & Testa, 1973). Of particular interest was whether cessation conditioning yields the beneficial characteristics of an escape response that are not represented in a backward signal–shock relation.

### Experiment 1

Experiment 1 used pretreatment shock parameters similar to those used by Moscovitch (1972) in his successful demonstration of inhibitory cessation conditioning in dogs. The shocks in this study were longer and more variable in duration than is typical in a helplessness experiment. However, if a cessation signal acquires fear-inhibitory properties because it signals shock termination, then its predictive value should be greatest when trial durations are unpredictable (cf. Moscovitch & LoLordo, 1968). Thus, an effect of cessation conditioning in the helplessness paradigm seemed most likely when Moscovitch's shock parameters were used.

Rats were exposed to variable-duration inescapable tail shocks, inescapable shock in conjunction with a 3-s cessation signal, or restraint in tubes. As a control for any nonassociative effect of presenting stimuli during the pretreatment session, other rats were exposed to the signal in a random, withindtrial relation with inescapable shock. The random signal was presented once per trial, but was equally likely during any 3 s of the total shock duration. If cessation conditioning depends on the reliable correlation between a signal and shock termination, then there should be no benefit of this random relation. All rats were tested for shuttle-escape performance 24 hr later.

### Method

**Subjects.** Thirty-two male Sprague-Dawley rats (Rattus norwegicus), weighing 300–340 g, were obtained from the Bantin–Kingman Company (Fremont, CA) and housed in individual cages, with free access to food and water, in a colony room maintained on a 12:12-
The experiment was conducted during the light portion of the cycle.

Apparatus. Pretreatment occurred in clear Plexiglas restraining tubes, measuring 23 cm in length and 6 cm in diameter. Adjustable front walls prevented rats from moving forward in the tubes. A rat's tail extended through the rear door of each tube and was taped to a plastic rod. Unscrambled shock was delivered from one of four constant-current shock generators (Lafayette Instrument Co., Model 82400) to electrodes attached to the rat's tail with electrode paste and tape. Each tube was housed in a sound-attenuating enclosure containing an exhaust fan that masked extraneous noises. Illumination was provided by a 7-W houselight located in the center of the rear wall of the attenuating enclosure. An 80-dB (SPL: re 2,000 dynes/cm²) 2000-Hz tone could be delivered from a speaker located in the upper right-hand corner of the rear wall of each enclosure.

Escape testing was conducted in two identical (45 cm x 20 cm x 20 cm) shuttleboxes (BRS-LVE Model 146-40). Each shuttlebox was divided into two equal compartments by a metal barrier that had an 8 x 7 cm center opening flush with the grid floor. The floor consisted of 2-mm diameter stainless-steel rods spaced 1.1 cm apart center to center. Scrambled shock was delivered to the grid floor from one of two Grason-Stadler (Series 700) shock generators. The floor was pivoted in the center and a response was recorded when a microswitch on either side of the chamber was displaced. The shuttleboxes were constantly illuminated by two 6-W lamps located in the center of each end wall. Each shuttlebox was housed in a sound-attenuating chest, containing an exhaust fan that masked extraneous noise. Experimental events were programmed and data recorded by Commodore 64C microcomputers.

Procedure. Rats were assigned randomly to one of four groups of 8 subjects each. Three groups were exposed to 60, 1.0-mA inescapable tail shocks on a variable time, 60-s schedule (range: 30 to 150 s) during pretreatment. Shock duration averaged 8.0 s on a trial (range: 4 to 22 s). Group 1 was exposed to inescapable shock in the absence of any signal; Group C was presented with a compound cessation signal, consisting of a 2000-Hz tone and blackout of the houselight, 3 s prior to the termination of each inescapable shock; and Group RAN received one presentation of the compound signal per trial in a random, within-trial relation (RAN), or restraint (R) in tubes. All rats were tested for shuttle-escape performance 24 hr later.

All groups were tested for escape performance in the shuttleboxes 24 hr later. Rats received 5 trials during which a single shuttle crossing terminated shock (FR-1 trials), followed by 25 trials during which a rat had to cross from one side of the shuttlebox to the other, and then return, to terminate shock (FR-2 trials). Shock terminated automatically if the appropriate response requirement was not met within 40 s of shock onset. Shock intensity was 0.6 mA on each trial. Both FR-1 and FR-2 trials were presented on a variable time, 60-s schedule (range: 20 to 230 s); however, 3 min intervened between trial types (cf. Minor & LoLordo, 1984). In this and subsequent experiments, statistical decisions were made with alpha = .05; only significant F ratios are reported.

Results and Discussion

Figure 1 shows mean shuttle-escape latencies in blocks of five trials for each group. Escape latencies did not differ among groups during FR-1 trials, F(3, 28) = 1.21, p < .05. Group differences were evident, however, when the response requirement was increased to an FR-2. Preexposure to inescapable shock (Group I) or inescapable shock in conjunction with a random signal (Group RAN) resulted in large performance deficits relative to restrained controls (Group R). By contrast, the ability to predict shock termination during pretreatment (Group C) prevented any performance deficit.

Group differences were analyzed by computing a mixed-design analysis of variance (ANOVA: Group x Trial Block) on FR-2 escape latencies. The ANOVA yielded a significant main effect of group, F(3, 28) = 25.81, and a significant Group x Trial Block interaction, F(12, 112) = 2.35. Newman–Keuls post hoc comparisons of the overall mean escape latency for the FR-2 trials indicated that Groups I and RAN, which did not differ from one another, were significantly slower to escape than were Groups C and R. Performance in Groups C and R was not significantly different.

The outcome of this experiment is inconsistent with the idea that shuttle-escape deficits or other measures of helplessness result from an inability to control traumatic aversive events. The presentation of a signal during the last few seconds of each trial did not alter the operational uncontrollability of pretreatment shocks, yet later escape deficits were prevented in Group C. Because presentations of the stimulus randomly within trials yielded no such protection (Group RAN), the performance of Group C cannot be attributed to a nonassociative effect of the stimuli and suggests that the benefits of a cessation signal stemmed from the ability to predict shock termination.

These data are consistent with anxiety-based interpretations of distress and helplessness, which attribute performance deficits to an inability to modulate intense, chronic fear during inescapable shock (Gray, 1982; Jackson & Minor, 1988; Minor et al., in press; Weiss et al., 1982; Weiss & Simson, 1985). Any operation that reduces fear during the pretreatment session should afford some measure of protection against later impairment. In this regard, Moscovitch's (1972) earlier dem-
onstration of the potent fear-inhibiting properties of cessation signals is consistent with the benefit derived from the procedure in the present study.

Experiment 2

The pretreatment shock durations used in Experiment 1 were longer and more variable in duration than is typical when trial durations are yoked to the escape latencies of a master rat. It is unclear whether cessation conditioning occurs when shorter, less variable shock durations are used. Mowrer and Aiken (1954) failed to find evidence of inhibitory cessation conditioning using fixed-duration shocks. By contrast, highly variable shock durations were used in the two successful demonstrations of the phenomenon (Moscovitch, 1972; Segundo et al., 1961). Although other procedural differences could account for the discrepancy between these studies, they serve to question the relevance of the results of Experiment 1 to the role of cessation conditioning in escape learning and the standard yoked helplessness experiment.

 Experiment 2 was conducted to assess the effectiveness of a signal for the cessation of inescapable shock in preventing later escape deficits when pretreatment shock durations were yoked to the escape latencies of a master rat. Rats were exposed to escapable tail shock, yoked inescapable shock, yoked inescapable shock with a cessation signal, or restraint in wheel-turn chambers during pretreatment and were tested for shuttle-escape performance 24 hr later. The cessation signal was presented for 1 s prior to the end of each pretreatment trial in the appropriate group.

Method

Subjects. Thirty-two male albino rats (Bantin–Kingman; Rattus norvegicus), weighing 280–310 g, were housed as in Experiment 1.

Apparatus. Pretreatment was conducted in four identical wheel-turn chambers, measuring 14 cm x 11 cm x 17 cm. Two circular pieces of Plexiglas (10 cm in diameter) were connected by 12 (7 cm long, 0.6 cm diameter) stainless-steel rods to form a cylindrical wheel. The wheel entered the front wall of the chamber 3.5 cm from the ceiling and extended 2.5 cm into its interior. A rat’s tail extended through an aperture in the rear door of a chamber and was taped to a Plexiglas rod. Unscrambled shock was delivered from one of three constant-current shock generators (Lafayette Electronics, Model 82400) to electrodes attached with tape to the rat’s tail. Contact was facilitated with electrode paste. The wheel-turn boxes were located in individual sound-attenuating chests illuminated by a 7-W bulb. Later escape testing was conducted in the shuttleboxes described for Experiment 1.

Procedure. Rats were assigned randomly to one of four groups of 8 rats each and exposed to escapable shock, yoked inescapable shock, yoked inescapable shock with a cessation signal, or restraint in wheel-turn chambers. Escape rats (E) received 100 unsignaled shock-escape trials on a variable time, 60-s schedule (range: 30 to 150 s). Wheel-turn responses were ineffective during the first 1.0 s of each trial. Thereafter, shock terminated when the rat completed a 360-degree turn of the wheel or after 60 s without a response. Escape latencies for each rat in Group E were recorded and used to determine trial durations for two inescapably shocked rats (Y: yoked shock) such that all three rats received the same number, pattern, intensity, and durations of shock. Responses by yoked rats had no effect on shock onset or termination. One yoked group (YC) received compound stimulus presentations, consisting of the onset of a 2000-Hz tone and the simultaneous blackout of the houselight, 1 s prior to the termination of each trial; shocks were unsignaled for the other yoked group (Y). Shock intensity for all three groups was 0.6 mA during the first 20 trials and was incremented by 0.1 mA for every block of 20 trials thereafter to a maximum intensity of 1.0 mA during Trials 81–100. Other rats were simply restrained (Group R) in wheel-turn chambers for a comparable amount of time and received no shock. All groups were tested for shuttle-escape performance 24 hr later, as in Experiment 1.

Results and Discussion

All escape rats rapidly learned to turn the wheel to escape shock during pretreatment. Mean escape latency during the first block of 20 trials was 3.36 s and decreased significantly over training to 2.27 s during the last 20 trials, $F(4, 28) = 2.77$.

The results of the shuttle-escape test are shown in Figure 2. Although escape latencies did not differ during the FR-1 trials ($F < 1$), clear group differences emerged when the response requirement was increased to FR-2. Rats that could escape pretreatment shocks (Group E) performed as well as restrained controls (Group R). Signaling shock termination during pretreatment (Group YC) was less effective in preventing FR-2 test deficits than was an escape contingency; nonetheless, some benefit of cessation conditioning was evident from the comparison of Group YC to the rats receiving pretreatment shocks without a signal (Group Y).

A mixed-design ANOVA (Group x Trial Block) conducted on FR-2 escape latencies yielded a significant main effect of group, $F(3, 28) = 13.45$, and a significant Group x Trial Block interaction, $F(12, 112) = 2.49$, indicating that latencies in Group YC increased over FR-2 trials as those in Group E decreased. Newman–Keuls pairwise comparisons of overall
FR-2 latencies indicated that Group Y was significantly slower to respond than were Groups YC, E, and R. Moreover, rats in Group YC were slower to escape than were Groups E and R, which did not differ from one another.

These data again indicate that the ability to predict shock termination during inescapable shock attenuates later performance deficits. Nonetheless, cessation conditioning (Group YC) was less effective in modulating the impact of pretreatment shocks than was behavioral control (Group E). Rats in Group YC also were slower to respond during testing than were restrained controls (Group R). Because statistically equivalent performance occurred between restrained and cessation groups in Experiment 1, this latter outcome suggests that the present procedure may have been less effective in promoting cessation conditioning.

Although Groups E and YC received identical exposure to shock during pretreatment, the predictive value of proprioceptive and exteroceptive stimuli probably differed in the two conditions. In Group E, proprioceptive stimuli generated by within-trial escape responses necessarily were strongly correlated with shock termination. However, whether the fixed-duration "cessation" cue was uniquely associated with shock termination in Group YC depended on the escape performance of the master rat. Although a minimum trial duration of 1.0 s was programmed into the wheel-turn escape contingency, some rats responded with such short latencies that total trial durations only varied between 1.2 and 1.8 s. For YC rats, an exteroceptive stimulus presented during the last 1.0 s of trials varying within these limits was not a unique signal for shock termination: A greater percentage of the total shock duration on a trial occurred in the presence of the signal than in its absence, which might have reduced the overall benefit derived from the signal. Other master rats acquired the escape response more slowly and produced longer, more variable trial durations, which increased the correlation between the YC signal and shock termination. Thus, E rats showing the greatest mastery of the escape contingency may have precluded cessation conditioning in their YC partner, whereas E rats that performed inconsistently may have enhanced cessation conditioning in the YC rat.

Some evidence that this relationship between groups affected the outcome of the experiment was obtained by computing the correlation between mean test latencies in YC rats and measures of pretreatment escape performance in their Group E partners. Although mean pretreatment latencies in Group E were not predictive of YC test performance, \( r(6) = -0.13, p > .05 \), a reasonably strong (albeit not statistically significant) negative correlation was obtained between the trial-to-trial variability in Group E wheel-turn latencies (as measured by the standard deviation) and YC shuttle-escape latencies, \( r(6) = -0.52, p > .10 \). Master rats that responded unreliably across pretreatment trials enhanced the value of the cessation signal and its modulatory effect on test performance in their YC partners, and vice versa.

The failure to find a closer match between the prophylactic action of an escape response and that of a cessation signal does not necessarily mean that the two operations are independent. Although it has been convenient to assume that proprioceptive feedback from the escape response acquires signal value during training, there is reason to question the assumption. For instance, monkeys deprived of proprioceptive, proprioceptive, and exteroceptive feedback are still capable of avoidance responding (Taub & Berman, 1968). Such data argue against the conceptions presented here and for a unique process of behavioral control (but see Gray, 1975, for an alternative explanation). Taub and Berman (1968) and Mowrer and Viek (1948), however, suggested that the secondary-reinforcing or signal value of a response is encoded with the motor program. Although the effects of response-generated proprioceptive feedback might be modeled by yoking exteroceptive stimulus presentations to each response of a master rat, one cannot know when and if such incipient behavior acquires stimulus properties or occurs on a trial. Thus, the failure to find identical effects of escape and cessation-signal operations in the present study may have been a modeling problem—it is quite possible that we have yet to find an effective combination of stimulus and temporal parameters that yields cessation conditioning with relatively short-duration shocks.

Rather than attempting to draw strong conclusions through direct comparison of cessation and escape relations in the yoked control procedure, information concerning the relatedness of these operations can be obtained by assessing their sensitivity to various manipulations. For instance, the protective effects of escape responses and safety stimuli have been dissociated under several conditions (Warren et al., in press).

A stronger case for a role of cessation conditioning in escape responding could be made if cessation stimuli retain their prophylactic action when safety signals do not. This general strategy was used in the following experiments.

**Experiment 3**

Although presentation of a stimulus immediately after inescapable shock usually prevents later measures of distress and helplessness, any beneficial effect of the manipulation is lost when short minimum ITIs are included in the schedule of shock delivery (Rosellini et al., 1986; Rosellini et al., 1987). Because backward inhibitory conditioning is similarly impaired by a manipulation of the minimum ITI (Moscovitch & LoLordo, 1968), the protection afforded by a safety signal probably is derived from the ability to predict relatively long shock-free periods. Moreover, the fact that the loss of any benefit of a safety signal under a short minimum ITI is correlated with a loss in inhibitory strength is consistent with the idea that the protection accruing from an operation is related to its fear-inhibiting potential (Jackson & Minor, 1988; Minor et al., in press). Nonetheless, escape responses retain their modulatory effect regardless of the distribution of ITIs (Rosellini et al., 1987), which questions the relevance of the safety-signal manipulation to escape responding and suggests that these two operations might work in fundamentally different ways to reduce the impact of a stressor.

**Cessation conditioning differs from standard inhibitory conditioning procedures in that a putative inhibitor is established in the presence of shock.** Because a cessation signal is contiguous with shock within a trial, increasing the proximity of shocks between trials by reducing the minimum ITI might
be expected to have little effect on the signal's inhibitory properties. Indeed, Moscovitch (1972) provided preliminary evidence that the use of short minimum ITIs does not substantially alter inhibitory cessation conditioning. Although the relative impact of a short minimum ITI on backward and cessation relations has not been assessed within a single experiment, a comparison across experiments (Moscovitch, 1972; Moscovitch & LoLordo, 1968) suggests that it is more detrimental to a backward procedure. Thus, if escape-generated stimuli become signals for shock termination and a signal-free period, the use of a short minimum ITI might eliminate the benefits of a safety signal but leave intact any protection accruing from cessation conditioning.

Experiment 3 determined whether the distribution of pretreatment ITIs differentially affects the ability of cessation and safety stimuli to prevent later shuttle-escape deficits. Independent groups of rats were exposed to unsignaled, inescapable shock, inescapable shock in conjunction with a 3-s cessation signal, inescapable shock followed by a 3-s safety stimulus, or restraint in tubes. The highly variable shock durations from Experiment 1 were used to facilitate cessation conditioning. One cessation group and one safety-signal group received brief additional shocks 5 s after a small percentage of the trials. These added shocks were intended to mimic the effect of shortening the minimum ITI but allowed us to maintain more features of the baseline shock schedule between groups, without dramatically changing the overall severity of the pretreatment session (cf. Anderson et al., 1987). All rats were tested for shuttle-escape performance 24 hr later.

**Method**

**Subjects and apparatus.** Forty-eight male albino rats (Bantin-Kingman), weighing 280–310 g, were housed as in Experiment 1. The apparatus was the same as in Experiment 1.

**Procedure.** Rats were assigned randomly to one of six groups of 8 rats each and exposed to 60, 1.0-mA, variable-duration inescapable tail shocks (I), inescapable shock with a cessation signal (C), inescapable shock followed by a safety signal (B: backward conditioning), or restraint (R) in tubes. The baseline schedule of shock delivery was identical to the one used in Experiment 1. The cessation signal consisted of the onset of a 2000-Hz tone and blackout of the houselight and was presented 3 s before shock terminated on each trial. Backward conditioning groups received at least one compound signal for 3 s immediately after each trial. One cessation group (C-AS) and one feedback group (B-AS) received additional 1.0-mA, fixed-duration (1-s) inescapable shocks 5 s after a randomly selected 17% of the trials (i.e., 10 additional shocks over 60 trials). Additional shocks were not accompanied by stimulus presentations in either group. All six groups were tested for shuttle-escape performance 24 hr later, as in Experiment 1.

**Results and Discussion**

Figure 3 shows shuttle-escape latencies in blocks of five trials for all groups in Experiment 3. Rats were exposed to variable-duration inescapable shocks (I), inescapable shock in conjunction with a cessation signal (C), inescapable shock followed by a backward (safety) signal (B), or restraint (R) in tubes. One cessation- (C-AS) and one backward-signal (B-AS) group received brief additional shocks 5 s after a randomly selected 17% of the pretreatment trials. All rats were tested for shuttle-escape performance 24 hr later.

A mixed-design ANOVA (Group × Trial Block) conducted on FR-2 escape latencies yielded significant main effects of group, \( F(5, 42) = 35.17 \), and trial block, \( F(4, 168) = 3.93 \). The interaction between factors was not statistically significant. Newman–Keuls comparisons revealed that overall mean latencies in Groups I and B-AS during the FR-2 trials were not significantly different; however, both groups were significantly slower to respond than were Groups C, C-AS, B, and R, which did not differ from one another.

These data replicate the findings of Experiment 1 concerning the protection afforded by a cessation signal and further demonstrate that such stimuli retain their prophylactic action under conditions in which a safety signal does not. Although cessation and safety stimuli were equally prophylactic when the minimum ITI (30 s) was relatively long, a safety signal failed to protect against the effects of inescapable shock on shuttle-escape performance when added shocks occasionally occurred in close proximity to the termination of the backward cue. The benefits of a cessation signal, however, were not significantly attenuated by the presentation of additional shocks.

It should be noted that the use of added shocks in this study is not the traditional method of shortening the minimum ITI. However, the reductions in the benefit of a safety signal are consistent with and extend previous work using a more traditional manipulation (Moscovitch, 1972; Moscovitch & LoLordo, 1968; Rosellini et al., 1987). Furthermore, the
added shock procedure allowed us to maintain more facets of the baseline shock schedule across conditions (e.g., session length and overall severity).

The interval between the termination of the safety signal and the occurrence of an added shock was 2 s in Group B-AS, whereas 5 s intervened between the termination of the cessation signal and an added shock in Group C-AS. Thus, cessation and backward conditioning could have been differentially affected by added shocks in the present study or a manipulation of the minimum ITI in other work (Moscovitch, 1972; Moscovitch & LoLordo, 1968) simply because of this difference in signal-shock interval. Cessation conditioning might have been equally impaired if added shocks had occurred in closer proximity to the termination of the signal. However, more recent work (Trauner & Minor, 1989) has shown that a cessation signal continues to protect against later escape deficits, regardless of whether added shocks are presented 2 s or 5 s after a trial.

The ability of cessation and safety stimuli to protect against stress-induced performance deficits and their sensitivity to the minimum ITI in the present experiment corresponds closely to earlier work on inhibitory conditioning in these procedures (Moscovitch, 1972; Moscovitch & LoLordo, 1968; Rosellini, DeCola, & Warren, 1986, Rosellini et al., 1987; Segundo et al., 1961; Siegel & Domjan, 1971). When a procedure results in inhibitory fear conditioning, performance deficits are prevented. Small changes in a procedure that retard inhibitory conditioning similarly reduce the prophylactic action of the signal relation in the helplessness paradigm.

The present data provide circumstantial evidence for a role of cessation conditioning in escape responding insofar as both operations retain their stress-reducing capacity under short minimum ITIs. Escape-related stimuli may become signals for shock termination and a shock-free period when relatively long ITIs are employed. Loss of one source of protection (safety feedback) when the minimum ITI is reduced would still leave a second protective relation (cessation conditioning) that is capable of preventing shuttle-escape deficits in isolation.

Experiment 4

Although we have argued here and elsewhere (Minor et al., in press) that response-generated stimuli become signals for shock termination and a shock-free period during escapable shock, it is unclear whether a single stimulus can yield both predictions simultaneously. Moscovitch and LoLordo (1968) reported that backward inhibitory conditioning was retarded when the conditional stimulus (CS) overlapped with the last few seconds of shock on each trial, suggesting that inhibitory cessation and backward conditioning do not simply summate. If fear inhibition is critical to the level of protection afforded by escape responses and exteroceptive events, then stimuli signaling both shock termination and a shock-free period should be less prophylactic than are stimuli signaling either relation alone.

Experiment 4 examined the relative benefit derived from a stimulus signaling the termination of inescapable shock and a shock-free period. Rats were exposed to the baseline shock parameters that yielded a protective signal in Experiments 1 and 3. The effect of combining predictive relations in a single stimulus was assessed by presenting a compound stimulus continuously during the last 3 s of each trial and the first 3 s of the ITI. Other rats received similar treatment except that separate elements of the compound were used as cessation and backward stimuli.

Method

Subjects and apparatus. Forty-eight male albino rats (Bantin-Kingman), weighing 290–320 g, were housed as in Experiment 1. The apparatus was the same as in Experiment 1.

Procedure. Rats were assigned randomly to one of six groups of eight rats each. Five groups were restrained in tubes and exposed to sixty 1.0-mA inescapable tail shocks according to the baseline schedule of shock delivery described in Experiment 1. These groups received stimulus presentations in one of the following relations with shock: (a) Shocks were unsignaled (Group I); (b) a compound cessation signal (tone + darkness) was presented for the last 3 s of each shock trial (Group C); (c) the compound stimulus was presented during the last 3 s of shock and the first 3 s of the ITI so that it contained both cessation and backward relations (Group CB); or (d) different elements of the compound served as cessation and backward signals—that is, tone served as the cessation signal and darkness as the backward signal (Group CB-TD), or vice versa (Group CB-DT). Other rats (Group R) were simply restrained in tubes for the same time period and received no shock. All rats were tested for shuttle-escape performance 24 hr later.

Results and Discussion

The results of the shuttle-escape test are shown in Figure 4. Escape latencies did not differ among groups during the FR-
1 trials \((F < 1)\). Escape performance on the remaining FR-2 trials was determined by the presence of cessation or safety signals and the nature of the signaling event. Although the presentation of a compound stimulus during the final seconds of each pretreatment trial (Group C) prevented the deleterious effects of inescapable shocks on later escape latencies (Group I), the benefit of that signal was markedly attenuated when the stimulus continued into the ITI (Group CB). However, in comparison to restrained controls (Group R), a combination of cessation and backward relations was prophylactic when different elements of the compound signaled each relation (Groups CB-TD and CB-DT).

A mixed-design ANOVA (Group \(\times\) Trial Block) conducted on FR-2 escape latencies yielded significant main effects of group, \(F(5, 42) = 9.23\), and trial block, \(F(4, 168) = 3.20\), and a significant Group \(\times\) Trial Block interaction, \(F(20, 168) = 1.88\). Newman–Keuls post hoc comparisons of the pooled means for all FR-2 trials indicated that rats in Group I were significantly slower to respond than were Groups CB, C, CB-TD, CB-DT, and R. Moreover, Group CB had longer latencies than did Groups C, CB-TD, CB-DT, and R, which did not differ from one another.

These data indicate that the ability of cessation and backward conditioning to modulate the impact of inescapable shock is markedly reduced when a single stimulus is used. Given the equivalent shuttle-escape latencies in Groups C and R, the comparatively poor performance of Group CB cannot be attributed to a general failure to obtain cessation conditioning. The detrimental effect of the CB manipulation also was not the simple product of signaling more than one relation within an experiment, because Groups CB-TD and CB-DT showed no deficit in test performance.

The poor test performance of Group CB may have resulted in part from the generalization of fear within the CB stimulus during conditioning. Moscovitch and Lloreda (1968) argued that cessation CSs are likely to have both excitatory and inhibitory properties. The reduction in inhibitory conditioning when a backward CS was overlapped with shock could result from the generalization of fear from the cessation to the backward components of the stimulus, thereby reducing the ability of the CB cue to prevent later escape deficits.

The performance of Group CB, however, cannot be explained solely on the basis of a loss of backward inhibitory conditioning. Presumably, the overall excitatory potential of the CB cue was no greater than that of a cessation CS alone. Even if this level of fear conditioning eliminated any advantage to pairing the stimulus with shock in a backward manner, the CB cue still should have had the net effect of a cessation CS, which is prophylactic in isolation. This logic suggests that both cessation and backward conditioning were retarded in Group CB, although for reasons that are not altogether clear.

The presentation of the same stimulus during the last seconds of shock and the first seconds of the ITI in Group CB was intended to model the signal properties of responding at these times during escapable shock. The performance of Group CB suggests that escape responding should be impaired if response-generated stimuli become signals for both shock termination and a shock-free period. However, the idea that escape-generated stimuli become signals for shock termination and a shock-free period is questioned by the present data only to the extent that such interoceptive stimuli are continuous and uniform. Responses made in the presence of shock and those occurring in the ITI can have different topographies and force characteristics (Davis, 1977; Davis & Burton, 1974), and they may generate discriminable interoceptive stimuli to create a condition more like that in Groups CB-TD or CB-DT than in Group CB. Thus, a contribution of posttrial feedback to the beneficial effects of an escape response cannot be ruled out on the basis of this experiment and may depend on the similarity between stimuli generated by within-trial and intertrial responses.

**Experiment 5**

Experience with escapable shock prior to exposure to inescapable shock usually prevents later deficits in escape performance (Seligman et al., 1975; Williams & Maier, 1977). This proactive effect of escape training has been termed immunization and provides some of the strongest evidence for learned helplessness theory. According to this account, subjects initially learn the positive contingency between responding and shock termination during exposure to escapable shock and develop the expectation that future events will be similarly controllable. This expectation of mastery (Volpicelli et al., 1983) proactively interferes with the learning that shock is uncontrollable during subsequent exposure to inescapable shock, thereby eliminating the cause of distress and helplessness during the final test phase.

The immunization phenomenon is conceptually problematic for an anxiety-based interpretation. Although response-generated stimuli may acquire predictive value to mitigate fear during initial escape training, it is unclear why this training would alter the capacity of subsequent inescapable shock to elicit fear and cause helpless effects. Moreover, Maier and Warren (1988) recently found that initial experience with a safety signal following inescapable shock did not serve as an effective immunizing agent, suggesting that the immunization phenomenon may be independent of the stimulus properties of responding. Indeed, Maier and Warren (1988) argued that qualitatively different mechanisms subserve the beneficial effects of escape responses and exteroceptive stimulus feedback.

Another possible explanation for Maier and Warren’s data, however, is that their procedure was not sufficiently sensitive to the potential proactive effects of the safety-signal manipulation. The anxiety hypothesis (Jackson & Minor, 1988; Minor et al., in press) suggests that escape responses and safety signals should have effects of the same nature, but not necessarily of identical magnitude. The interoceptive stimuli generated by an escape response and the exteroceptive stimuli used to model their effects probably differ along a number of dimensions (e.g., information content, saliency, and duration) that influence rate of conditioning and overall predictive value. In this regard, exteroceptive feedback may have a prophylactic action that is quantitatively smaller than that of escape. If this is the case, it should be possible to override any beneficial effect of the manipulation, particularly under highly stressful conditions. Unfortunately, Maier and Warren (1988)
used a large number (100) of unusually severe (1.6 mA) shocks during initial immunization training and subsequent exposure to inescapable shock. Thus, the apparent qualitative differences between escape and safety-signal contingencies as immunizing agents actually may have resulted from quantitative differences in conditioning and the relatively greater prophylactic action of escape.

Experiment 5 assessed the ability of a safety signal to immunize against the effects of subsequent inescapable shock under less stressful parameters than were used by Maier and Warren (1988). Rats initially received 60 trials of escapable tail shock (1.0 mA) in tubes on Day 2, followed by a shuttle-escape test on Day 3.

Method

Subjects and apparatus. Thirty-two male albino rats (Bantin-Kingman), weighing 300-340 g, were housed as in Experiment 1. The wheel-turn chambers used during immunization training (Phase 2) were described in Experiment 2. Restraining tubes (Phase 2) and shuttleboxes (Phase 3) were described in Experiment 1.

Procedure. Rats were assigned randomly to one of four groups of eight rats and exposed to 60 trials of escapable tail shock (E), yoked inescapable shock (Y), yoked inescapable shock followed by a 5-s compound stimulus (tone + darkness) presentation (YS), or restraint (R) in wheel-turn chambers. Trials were presented on a VT 60-s schedule (range = 30-150 s). Wheel-turn responses by the E rat were ineffective in terminating shock during the first 0.8 s of each trial. Thereafter, shock terminated for all rats when the E rat completed a 360° turn of the wheel. Shock intensity was 0.6 mA during the first 20 trials and was incremented by 0.1 mA for each block of 20 trials thereafter, as in Experiment 2.

On Day 2, rats in the three shocked groups (i.e., EI, YI, and YSI) were exposed to one hundred, 1.0-mA unsignaled, inescapable tail shocks on a variable time, 60-s schedule in restraining tubes. The fourth group (RR) was restrained in tubes for an equivalent amount of time and received no shock. All groups were tested for shuttle-escape performance on Day 3.

Results and Discussion

All rats in Group EI rapidly learned to turn the wheel to escape shock during the immunization phase. Escape latencies were relatively fast throughout training and decreased only slightly from a mean of 3.45 s during the first 20 trials to 1.96 s during the last 20 trials. This change in escape latencies did not attain a conventional level of statistical significance, $F(3, 21) = 2.58, p > .08$.

The results of the shuttle-escape test are shown in Figure 5. A statistically significant performance deficit occurred during the FR-1 trials for rats receiving yoked inescapable shock during the immunization phase (Groups YI and YSI) in comparison to the groups exposed to escapable shock (EI) or restraint (RR). $F(3, 28) = 5.31$. The poor performance of the yoked groups was augmented when the shuttle requirement was increased. Groups YI and YSI showed equally large performance deficits during the FR-2 trials. Thus, the presentation of a stimulus in a backward relation with inescapable shock failed to immunize against the adverse effects of subsequent unsignaled, inescapable shock on test performance.

By contrast, initial exposure to escapable shock prevented a stress-induced deficit in test performance. Rats in Group EI performed as well as the controls receiving simple restraint in earlier sessions (Group RR).

A mixed-design ANOVA (Group × Trial Block) conducted on FR-2 escape latencies yielded significant main effects of group, $F(3, 28) = 32.62$, and trial block, $F(4, 112) = 5.31$. The interaction between variables was not statistically significant. Newman-Keuls comparisons indicated that Groups YI and YSI, which did not differ in FR-2 escape latencies, were significantly slower to respond than were Groups EI and RR, which did not differ from one another.

The results of this experiment are consistent with those of Maier and Warren (1988) in demonstrating that initial experience with a safety signal does not immunize against the adverse effects of subsequent unsignaled, inescapable shock on shuttle-escape performance. Because comparatively moderate shock parameters were used in the present study, it is unlikely that Maier and Warren's failure to find similar effects of escape and safety-signal contingencies was due to the use of a highly stressful procedure that masked a proactive effect of the exteroceptive event. As such, these experiments suggest that posttrial safety is not sufficient for immunization to occur.

Experiment 6

The previous experiment does not necessarily rule out a role of response-generated stimuli in the immunization phenomenon. Signaling the cessation of shock may be more critical to immunization phenomena than is feedback concerning safety. Another possibility is that an escape response is generally more protective because of the combined predic-
tive values of response-generated stimuli. This possibility suggests that an immunization effect might occur when both cessation and safety feedback are modeled with exteroceptive event presentations during inescapable shock.

Experiment 6 compared the effectiveness of a cessation signal and a combination of cessation and safety signals as immunizing agents. Because only weak evidence of protection by a cessation signal was obtained when shock durations were yoked to the escape latencies of a master rat (Experiment 2), a direct comparison of these signal conditions with escapable shock was not possible. During the initial training phase, independent groups of rats were exposed to variable-duration inescapable tailshocks, inescapable shock with a cessation signal, or inescapable shock accompanied by both cessation and safety signals. Rats in these three groups were exposed to fixed-duration inescapable shocks 24 hr later. The restraining devices and other aspects of the conditioning context were similar in these first two phases to maximize generalization of the effects of immunization training to inescapable shock treatment on Day 2. All rats were tested for shuttle-escape performance on Day 3.

If the stimulus features of an escape response contribute to the immunization phenomenon, one of the two signal conditions in Phase 1 should work proactively to attenuate the effects of Phase 2 inescapable shocks on shuttle-escape performance. Specifically, if the immunization effect requires the combined contribution of cessation and backward conditioning, then performance deficits should be alleviated only in the group exposed to both signal types in Phase 1. On the other hand, if cessation conditioning is largely responsible for proactive transfer, then either of the two Phase 1 signal conditions should improve test performance.

Method

Subjects and apparatus. Thirty-two male albino rats from Ban
tin–Kingman, weighing 311–330 g, were housed as in Experiment 1. The apparatus was the same as in Experiment 1.

Procedure. Rats were assigned randomly to one of four groups of eight rats each. Three groups were exposed to 60 variable-duration inescapable tailshocks in restraining tubes as in Experiment 1. One of these groups (CI) received compound stimulus presentations (tone + darkness) during the last 3 s of each trial; a second group (CB-I) received tone presentations during the last 3 s of each trial, followed by a 3-s blackout of the houselight; and the third group (II) was exposed to unsignaled shock. Twenty-four hr later all rats in these groups were exposed to one hundred, 5-s (1.0 mA) inescapable tailshocks on a variable time, 60-s schedule in restraining tubes. A fourth group (RR) was restrained in tubes for an appropriate amount of time in both phases and received no shock. Shuttle-escape testing was conducted on Day 3 for all groups.

Results and Discussion

The results of the Phase 3 shuttle-escape test are shown in Figure 6. Escape latencies did not differ among groups during the FR-1 trials. A large performance deficit occurred for the rats receiving inescapable shock during Phases 1 and 2 (Group II). By contrast, initial exposure to inescapable shock in conjunction with a cessation signal (Group CI) partially immunized against the effects of subsequent inescapable shock on test performance. This immunization effect was enhanced when different stimuli signaled shock termination and a shock-free period (Group CB-I).

A mixed-design ANOVA (Group × Trial Block) conducted on FR-2 escape latencies yielded significant main effects of group, \( F(3, 32) = 11.65 \), and trial block, \( F(4, 128) = 2.43 \), and a marginally significant Group × Trial Block interaction, \( F(12, 128) = 1.82 (p > .051) \). Newman–Keuls post hoc comparisons indicated that Group II had significantly longer FR-2 escape latencies than did Groups CI, CB-I, and RR. Moreover, Group CI had a significantly longer overall escape latency than did Group RR. Groups CB-I and RR did not differ from one another.

These data are inconsistent with learned helplessness theory. The immunization experiment is a natural extension of the logic used to explain the basic helplessness effect (cf. Maier & Seligman, 1976; Seligman et al., 1975). If learning that shock termination is independent of responding during inescapable shock proactively interferes with learning the positive escape contingency during shuttle testing, then initially training subjects on a positive escape contingency should prevent the basic helplessness effect. Such preliminary training should instill cognitions of mastery that proactively interfere with the development of helplessness cognitions during subsequent exposure to inescapable shock, thereby eliminating the cause of shuttle-escape deficits in the final test phase. However, the present data clearly indicate that response learning is not a necessary condition for immunization to occur. The presentation of a cessation signal or the combination of cessation and backward signals did not change the operational uncontrollability of the immunizing shocks. Yet the typical deficit in shuttle-escape performance produced by Phase 2 inescap-
able shocks was partially reversed in Group Cl and completely eliminated in Group CB-I.

A potential caveat to this conclusion is that the conditions of Phases 1 and 2 were selected to maximize generalization in the present study. Other investigations of the immunization phenomenon have attempted to minimize the similarity between these phases. At the same time, however, the similarity between the immunization phase and the test phase often has been maximized. For instance, in the seminal study of immunization (Seligman & Maier, 1967), dogs were trained on an escape-avoidance task in a shuttlebox, exposed to inescapable shock in a different apparatus, and then tested in the shuttlebox used for immunization training. Similarly, in their study of immunization in rats, Williams and Maier (1977) used grid shock during the immunization and test phases, with interpolated tail shock. The extent to which similarity across phases influences the immunization effect has not been analyzed directly and could have important implications for interpretations of the phenomenon.

Immunization does not appear to be a simple artifact of conditioning an active response to shock during the immunization phase (Anisman, Irwin, Beauchamp, & Zacharko, 1983; Minor et al., in press). One explanation for the basic helplessness effect is that an inactive pattern of responding (Anderson, Crowell, Cunningham, & Lupo, 1979; Anisman et al., 1978; Glazer & Weiss, 1976) or motoric depression (Weiss et al., 1981; Weiss & Simson, 1985) is established during exposure to inescapable shock. This pattern recurs during later testing to interfere with the performance of active movements. From this view, preliminary exposure to escapable shock in the immunization paradigm prevents test deficits by training an active instrumental response to shock that proactively interferes with the development of inactive responses during inescapable shock. Of course, this direct effect of an instrumental escape contingency on responding cannot be modeled with stimulus presentations during inescapable shock and, therefore, cannot account for the present findings.

The fact that a combination of cessation and backward signals (Group CB-I) was somewhat more effective as an immunizing agent than was a cessation signal alone (Group Cl) suggests that the immunization phenomenon may be related to the total level of stress-reduction or inhibitory conditioning accomplished during initial training. Initial escape training should be particularly effective as an immunizing agent if this is the case. An escape contingency potentially adds not only the dimensions of predictability represented by the signal conditions of the present study, but also may allow other stress-reducing mechanisms to operate that are not directly related to prediction or control (see Minor et al., in press, for a thorough discussion).

General Discussion

This investigation provides clear evidence that deficits in shuttle-escape performance are dramatically attenuated when the cessation of inescapable preshocks is signaled by an exteroceptive stimulus. This modulatory effect of cessation conditioning depends on the relationship between the signal and shock termination, as random within-trial presentations of a signal afforded no protection against later escape deficits (Experiment 1). Moreover, cessation signals retained their prophylactic action under conditions that reduced the effectiveness of a backward signal-shock relation. The inclusion of short minimum ITIs in the pretreatment schedule of shock delivery eliminated the benefits of a safety signal but did not substantially alter the prophylactic action of a cessation signal (Experiment 3). The presentation of a safety signal during inescapable shock also failed to immunize against the adverse effects of subsequent inescapable shock on test performance (Experiment 5). On the other hand, a cessation signal was partially effective as an immunizing agent, although its effects were greater when combined with a safety signal (Experiment 6).

These data are incompatible with the learned helplessness explanation of shuttle-escape deficits (e.g., Maier & Seligman, 1976; Overmier & Seligman, 1967). Signalizing the cessation of inescapable shock did not alter the operational uncontrollability of pretreatment shocks, yet later deficits in escape performance were prevented. Furthermore, the effectiveness of cessation conditioning as an immunizer is inconsistent with the type of proactive interference mechanism used to explain not only the immunization phenomenon but the basic helplessness effect as well. Behavioral control clearly is not necessary for immunization to occur. Consequently, the learning of response-outcome dependence or the development of mastery cognitions cannot be the only mechanism capable of mitigating the impact of subsequent inescapable shock in the immunization paradigm. As such, it seems very unlikely that the learning of response-outcome independence is responsible for the deleterious effects of inescapable shock on later shuttle-escape performance.

Nonassociative interpretations of signal effects also have difficulty accounting for the results of the present experiments. Maier and Keith (1987) suggested that presenting a stimulus immediately after inescapable shock serves as a posttrial distractor that disrupts rehearsal of information concerning the dependence of responding and shock termination. Subjects fail to develop helplessness cognitions as a result, and therefore are not impaired during testing. This hypothesis, however, is of questionable relevance to the study of cessation signals. Cessation conditioning does not involve the posttrial presentation of stimulus events and, therefore, should not disrupt memory processing during the ITI. The explanation also has difficulty accounting for several findings concerning the effects of a safety signal following inescapable shock. For instance, reducing the minimum ITI during the pretreatment session (Experiment 3) eliminated the ability of a safety signal to prevent later shuttle-escape deficits. As Rosellini and his colleagues have noted previously (e.g., Rosellini & DeCola, 1988; Rosellini et al., 1987; Warren et al., in press), there is little reason to assume that changing the minimum ITI should alter the effectiveness of a safety stimulus as a posttrial distractor. Similarly, it is difficult to understand why overlapping a backward CS with shock (Experiment 4) would alter posttrial rehearsal processes. Because both of these manipulations retard backward inhibitory conditioning (Moscovitch & LoLordo, 1968), their effects in the helplessness paradigm seem to be more amenable to an associative interpretation.
The Modulator Analysis and Fear Inhibition

The present results can be interpreted within the general framework of a modulator analysis (Minor et al., in press; Overmier, 1988). This approach attributes the consequences of exposure to unsigned, inescapable shock to the stressor per se. More complex procedures (e.g., signaled, inescapable shock and escapable shock) are viewed as potential modulators (either amplifiers or attenuators) of the baseline stress reaction and are analyzed by adding the appropriate contiguity to the baseline procedure and comparing the two conditions. If the two procedures produce differential outcomes, then the added operation is assumed to generate a psychological context that modulates the stress baseline.

The modulator analysis of stress effects is largely atheoretical and nonspecific with respect to the nature of the baseline stress reaction or the psychological context that is generated by modulator variables (Overmier, 1988). However, as we have argued here, one possibility is that exposure to unsigned, inescapable shock generates a baseline of intense, chronic fear that causes subsequent performance deficits (see Minor et al., in press, and Weiss & Simson, 1985, for a thorough discussion). From this view, any added operation that reduces the intensity or chronicity of fear during pretreatment should serve as an effective modulator of helplessness effects. Thus, anxiety reduction provides a unifying mechanism of action for modulator operations, including escape and signal–shock contingencies.

One way in which modulator contingencies might work to reduce anxiety during pretreatment is by acquiring fear-inhibitory properties. The ability of a safety signal to reduce the level of fear conditioned to the pretreatment context (Jackson & Minor, 1988; Mineka et al., 1984; Rosellini et al., 1987) and to eliminate later deficits in escape performance (Anderson et al., 1987; Jackson & Minor, 1988) or other measures of pathology (Overmier & Murison, 1989; Overmier et al., 1985) is closely linked to inhibitory conditioning and the prediction of safety during the ITI.

The benefits of a cessation signal could be similarly dependent on fear inhibition. Although Mowrer and Aiken (1954) reported that signaling the termination of shock resulted in excitatory fear conditioning in rats, only five conditioning trials were given before testing. In addition, the properties of the cessation CS were assessed by response-contingent presentations of the signal for a pretrained appetitive operant, a procedure that is highly sensitive to weak excitatory potentials (Heth, 1976; Heth & Rescorla, 1973). As Mowrer and Aiken noted, this type of procedure was unlikely to reveal inhibitory potentials, because there was no fear to inhibit in the test and the occurrence of the CS in the appetitive condition was likely to “remind” subjects of aversive conditioning. In this regard, evidence of inhibitory cessation conditioning has been obtained following more extensive training and when an aversive test was used to measure the signal’s inhibitory properties. Segundo et al. (1961) reported that a cessation CS mitigated the unconditioned reaction to shock. Moscovitch (1972) further demonstrated that the inhibitory potential of a cessation CS transfers to a different situation to suppress ongoing escape–avoidance responding.

Nonetheless, there is reason to question whether the effectiveness of a cessation signal as a modulator stems entirely from the development of an inhibitory association. The present data suggest that cessation and backward signals work in different ways to prevent helplessness effects. These relations were differentially sensitive to a manipulation of the minimum ITI (Experiment 3), could not be signaled by a single stimulus (Experiment 4), and exerted different proactive effects in the immunization paradigm (Experiments 5 and 6). Although fear inhibition may be accomplished with different associative mechanisms (e.g., Holland & Lamarrhe, 1984), the exact nature of the association in cessation and backward conditioning and how these mechanisms affect fear are not known. Several standard inhibitory conditioning procedures produce a CS that simultaneously carries excitatory and inhibitory associations. Collateral excitatory associations can mask the expression of inhibition, which is revealed only after the excitatory potential is extinguished through repeated presentation of the CS alone (Williams & Overmier, 1988; Williams, Travis, & Overmier, 1986). As Moscovitch and Lordingo (1968) have argued, cessation CSs are very likely to develop strong excitatory potentials due to their contiguity with shock to mask the expression of inhibition. Thus, from the perspective of the anxiety hypothesis, signaling shock termination should not have resulted in effective modulation of test deficits if fear reduction was accomplished solely through the expression of an inhibitory association.

Rather than modulating the impact of inescapable shock through a direct inhibitory association, cessation signals may reduce fear through a more circuitous route. The work of Segundo et al. (1961) suggests that the benefits of a cessation signal derive, in part, from counterconditioning. Following extensive training, the presentation of a cessation CS eliminated the defensive behavior typically seen throughout the earlier portion of the shock trial. The CS also eventually elicited the behavior and cortical responses that were normally observed immediately after a conditioning trial. Interestingly, any excitatory association of a cessation CS with shock could contribute to this process. Conditioned fear stimuli frequently produce a potent hypalgesia (e.g., Fanselow, 1981)—some pain inhibition may be necessary to suppress the defensive reaction to shock before posttrial behavior is transferred to the CS. Such processes could work to “devalue” (Holland & Straub, 1979; Rescorla, 1973) the representation of shock prior to posttrial memory processing (Wagner, 1978). Joint rehearsal of a functionally weaker unconditional stimulus intensity with ambient stimuli should reduce the asymptotic level of fear conditioning in the situation (see Mackintosh, 1974, for a review). Thus, whereas a backward signal–shock relation appears to work by inhibiting fear during the ITI, signaling shock termination may reduce the maximum level of fear by altering the effectiveness of the UCS.

Regardless of the specific mechanism by which cessation signals modulate the impact of shock, these data are generally consistent with the idea that cessation conditioning contributes importantly to the beneficial effects of an escape response. Like an escape response, a cessation signal retained its prophylactic effect under short minimum ITIs (Experiment 3) and proactively altered the ability of unsigned, inescapable
shock to impair test performance in the immunization paradigm (Experiment 6). These data suggest that prediction and control are not independent modulatory dimensions. Much of the prophylactic action of an escape response is likely to result from the acquired signal value of response-generated stimuli and attendant reductions in fear during the pretreatment session. A possible qualification to this argument is that signaling shock termination during pretreatment afforded somewhat less protection against later escape deficits than did the availability of an escape response when shock durations were yoked in Experiment 2. However, as argued earlier, this difference probably resulted from a failure to equate the conditioning of exteroceptive and interoceptive stimuli in the two conditions and an inherent bias in the yoked-control procedure toward superior performance in the master group (cf. Church, 1964).

References


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