SUPPRESSIVE AND FACILITATIVE EFFECTS OF SHOCK INTENSITY AND INTERRESPONSE TIMES FOLLOWED BY SHOCK

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Although response-dependent shock often suppresses responding, response facilitation can occur. In two experiments, we examined the suppressive and facilitative effects of shock by manipulating shock intensity and the interresponse times that produced shock. Rats' lever presses were reinforced on a variable-interval 40-s schedule of food presentation. Shock followed either long or short interresponse times. Shock intensity was raised from 0.05 mA to 0.4 mA or 0.8 mA. Overall, shock contingent on long interresponse times punished long interresponse times and increased response rates. Shock contingent on short interresponse times punished short interresponse times and decreased response rates. In Experiment 1, raising the range of interresponse times that produced shock enhanced these effects. In Experiment 2, the effects of shock intensity depended on the interresponse times that produced shock. When long interresponse times produced shock, low intensities increased response rates. High intensities decreased response rates. When short interresponse times produced shock, high shock intensities punished short interresponse times and decreased response rates more than low intensities. The results may explain why punishment procedures occasionally facilitate responding and establish parameters for future studies of punishment.

Key words: punishment, electric shock, shock intensity, interresponse times, lever press, rats

Azrin and Holz (1966) defined punishment as the process by which a response-dependent stimulus reduces the future probability of the response. In the typical procedure for studying punishment, food occasionally is delivered contingent on a rat's lever presses. Once responding stabilizes, a punishment contingency is added by delivering response-dependent electric shock through a grid floor. Punishment is assessed by comparing responding before and after the addition of the shock contingency.

Although adding a shock contingency often decreases response rates, it can also increase

rates (Appel, 1968; Arbuckle & Lattal, 1992; Filby & Appel, 1966; Galbicka & Branch, 1981; Lande, 1981; Sizemore & Maxwell, 1985). This paradoxical effect has been observed with a variety of subjects (e.g., monkeys, rats, and pigeons), operants (e.g., lever presses and key pecks), and reinforcers (e.g., monkey chow, pellets, and access to grain). Whether shock increases or decreases response rates may depend on several factors including the schedule of shock, the schedule of reinforcement, the manner of introducing shock, shock duration, shock frequency, shock intensity, and the interresponse times (IRTs) followed by shock (Arbuckle & Lattal, 1992; Azrin & Holz, 1966; Baron, 1991; Boe, 1966; Church, 1963; Sizemore & Maxwell, 1985). Of present interest are two of these factors: shock intensity and the IRTs followed by shock.

Typically, response facilitation is more likely when the shock intensity is low (Appel, 1968; Arbuckle & Lattal, 1992; Filby & Appel, 1966; Lande, 1981; Sizemore & Maxwell, 1985). For example, Filby and Appel reported that 0.2-mA and 0.4-mA shocks increased response rates, but shock intensities of 0.6 mA and

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higher decreased response rates. Similar results were reported by Appel (1968), Arbuckle and Lattal (1992), Lande (1981), and Sizemore and Maxwell (1985).

Whether shock increases or decreases response rates may also depend on the IRTs followed by shock (e.g., Arbuckle & Lattal, 1987; 1992; Galbicka & Branch, 1981; Sizemore & Maxwell, 1985). Galbicka and Branch used shock schedules with and without an IRT contingency to assess the role of IRTs in determining responses rates. They reinforced squirrel monkeys' lever presses according to a variable interval (VI) 60-s schedule and arranged 1.0-mA shock according to a differential IRT schedule or a random-ratio (RR) schedule. Under both shock schedules, approximately one out of every thirty responses was followed by shock, but only one schedule included an IRT contingency. In the differential IRT condition, shock depended on long IRTs, whereas in the RR-30 condition, shock was independent of IRTs. When long IRTs produced shock, response rates increased. Long IRTs were punished, and shorter IRTs became more frequent. Expanding the range of IRTs followed by shock to include shorter IRTs caused response rates to increase further. By contrast, when shock delivery was independent of IRTs, response rates decreased. Short IRTs were punished, and long IRTs became more frequent. Under the RR schedule, expanding the portion of responses followed by shock decreased response rates further. Arbuckle and Lattal (1992, Experiment 2) extended these findings by delivering shock contingent on either long or short IRTs. Although the effects on overall response rates were inconsistent across subjects, shock affected the IRT distributions by punishing the IRTs that produced shock.

The results of Galbicka and Branch (1981) are interesting not only because they demonstrate that response-dependent shock can increase response rates but also because they establish that IRTs can serve as functional units under punishment contingencies. Their results suggest that cases of response facilitation on response-dependent shock schedules may be attributable to the punishment of long IRTs rather than responses. Given that the facilitative effects of shock on response rates seem to be limited to low shock intensities, one may wonder whether the punishment of long IRTs

is specific to low intensities as well. In Galbicka and Branch's study, shock intensity was held constant at 1.0 mA. This prevented any interactive effects of shock intensity and the IRTs that produce shock. To assess any interactive effects of these variables, shock intensity must be manipulated when IRTs are the functional units.

Sizemore and Maxwell (1985) investigated the combined effects of shock intensity and the IRTs that produce shock. They reinforced rats' lever presses with food on a VI 40-s schedule. Across conditions, shock intensity was raised from 0.1 mA to 0.4 mA in 0.1-mA steps. Two experiments differed in terms of the IRTs specified by the shock contingency. In the first experiment, all "long" IRTs (IRTs greater than 8 s) produced shock. In the second experiment, all "intermediate" IRTs (IRTs greater than 8 s and less than 12 s) produced shock. The lowest intensity of 0.1 mA increased response rates regardless of the IRTs that produced shock. By contrast, the effects of higher shock intensities depended on the IRTs that produced shock. When long IRTs produced 0.2-mA shock, response rates increased slightly. Higher shock intensities decreased and occasionally eliminated responding. When intermediate IRTs produced 0.2-mA, 0.3-mA, or 0.4-mA shock, shock decreased response rates but did not eliminate responding. Analyses of the IRT distributions showed that shock punished the targeted IRTs in both experiments, however, punishment of these became less differential as the intensity was raised.

Sizemore and Maxwell's (1985) results provide additional support that IRTs can serve as functional units under punishment contingencies. They also show that shock intensity and the IRTs that produce shock interact to determine whether shock suppresses or facilitates responding. When high-intensity shock was contingent on long IRTs (Experiment 1), response rates decreased. One reason for the decrease in response rates may be that shock elicited other responses, such as freezing, which interfered with the ability to complete the operant response. Another possibility is that the temporal reach of shock depends on the shock intensity. If so, raising the shock intensity may also raise the temporal reach of shock and increase the number of IRTs captured by the punishment contingency. At

low intensities, the effect of shock may be localized to the IRTs closest in time to the shock. If long IRTs produce shock, long IRTs decrease in frequency, and response rates increase (e.g., Arbuckle & Lattal, 1992; Sizemore & Maxwell, 1985). If short IRTs produce shock, short IRTs decrease in frequency and response rates decrease (e.g., Arbuckle & Lattal, 1992). At high shock intensities, the temporal reach of shock may extend to a longer sequence of IRTs, which most likely includes a variety of IRT durations. Under these circumstances, various IRT classes would decrease in frequency, and response rates would decrease.

There is no reason, however, to assume that changes in the temporal reach of a behavioral consequence are limited to cases where the consequence is a punisher. Raising the intensity, or magnitude, of any behavioral consequence may increase its temporal reach regardless of whether the consequence is a punisher or a reinforcer.

Doughty and Richards (2002) demonstrated how the magnitude of a consequence affects the temporal reach of its behavioral effect. They examined the effects of reinforcer magnitude on responding under differential-reinforcement-of-low-rate (DRL) schedules. On a DRL schedule, an IRT longer than a specified criterion produces a reinforcer. In the first experiment, rats' lever presses produced either 30 μ l or 300 μ l of water on a DRL schedule. The experiment was divided into three phases. In the first phase, IRTs longer than 72 s produced 30 μ l or 300 μ l of water, depending on the rat. After responding stabilized, the amount of water was reversed such that rats that were previously given 30 μ l were given 300 μ l and vice versa. In the second phase, the reinforcement schedule was changed to a DRL18-s schedule. In the third phase, the DRL remained unchanged but the water amounts alternated each session. Across phases, 300 μ l of water produced higher response rates than 30 μ l, indicating that 30 μ l was more effective in the differential reinforcement of long IRTs. Analyses of the IRT distributions revealed that as the size of the reinforcer increased, the distributions became more positively skewed, and intermediate and short IRTs increased in frequency. In other words, when the reinforcer was large, the differential reinforcement of long IRTs

became less differential and generalized to other classes of IRTs. Similar results also were obtained by Kirshenbaum, Brown, Hughes, and Doughty (2008).

The experiments of Doughty and Richards (2002) and Kirshenbaum et al. (2008) support the idea that the temporal reach of a behavioral consequence increases as the magnitude of the consequence is raised. Arbuckle and Lattal (1992) suggested that a similar outcome may occur with punishment. Results from Sizemore and Maxwell's (1985) first experiment, in which high shock intensities punished all classes of IRTs, are consistent with but do not directly establish the idea that more IRTs are affected by shock as shock intensity is raised. More direct evidence of the changes in the temporal reach of shock as it applies to punishment is needed.

To fully understand the interactive effects of shock intensity and the IRTs followed by shock, and consequently, to understand how behavioral units might vary as a function of shock intensity, several issues must be clarified. One is the role of shock intensity, specifically high intensities. In Sizemore and Maxwell's (1985) first experiment, the subjects' contact with 0.4-mA shock was limited. This made it difficult to assess the effects of the highest intensities. Furthermore, high shock intensities were confounded with increases in the cumulative exposure to shock. This was due to manipulating shock intensity in ascending order. Gradually raising, as opposed to lowering or randomizing, the shock intensity is a common experimental strategy in studies of punishment because it minimizes the risk of sensitization (e.g., Azrin & Holz, 1966). Nevertheless, manipulating shock intensity in this way makes it difficult to disentangle the effects of shock intensity from the effects of cumulative exposure to shock. Additionally, in Sizemore and Maxwell's study, the range of shock intensities was small, spanning only 0.3 mA. Using a broader range of intensities might reveal effects that were absent with narrow ranges. Sizemore and Maxwell's study also leaves unanswered questions about the IRTs that produce shock. For example, in their experiments, shock targeted long and intermediate IRTs. Fewer studies have included shock contingencies targeting short IRTs (cf. Arbuckle & Lattal, 1992). Lastly, Sizemore and Maxwell defined the IRTs that produced shock

in absolute terms. This may have produced inconsistent contact with the shock contingencies across subjects, as the IRT distributions presumably varied across individuals.

The purpose of the present study was to clarify the conditions under which shock suppresses or facilitates responding by examining the effects of shock intensity and the IRTs that produce shock. To address previous concerns regarding the limited range of shock intensities and contact with shock contingency, the present study included five shock intensities varied across a 0.75-mA range. This is more than twice the range used by Sizemore and Maxwell (1985). Additionally, we used two different arrangements of the experimental manipulations to separate the effects of shock intensity from those of the cumulative exposure to shock. To fully understand the role of the IRTs that produce shock, we delivered shock contingent on short IRTs as well as long IRTs and defined the IRTs that produced shock in relative terms to maintain more consistent contact with the shock contingencies across conditions, subjects, and shock intensities. Lastly, we manipulated a third variable, the range of IRTs susceptible to shock, not studied by Sizemore and Maxwell. Previous research by Galbicka and Branch (1981) indicates that the range of IRTs susceptible to shock may influence whether shock suppresses or facilitates responding, but few studies have examined how this variable interacts with shock intensity.

In two experiments, rats' lever presses were reinforced with food on a VI 40-s schedule. Once responding stabilized, response-dependent shock was added. We manipulated the range of IRTs susceptible to shock and raised the shock intensity from 0.05 mA to 0.4 mA or 0.8 mA. Shock delivery was contingent on either long IRTs or short IRTs. Because previous research with shock has shown differences in suppression levels according to how shock intensity is raised (e.g., Azrin & Holz, 1966), the experiments differed in their arrangements of the experimental manipulations. In the first experiment, the portion of the IRT distribution susceptible to shock was raised prior to raising the shock intensity. In the second experiment, shock intensity was raised before the portion of the IRT distribution susceptible to shock was raised.

GENERAL METHOD

Subjects

Experimentally naive male Sprague-Dawley rats (*Rattus norvegicus*) served as subjects, 8 in each experiment. They were maintained at 80% ($\pm 2\%$) of their free-feeding weights by food obtained during the experimental sessions and, if necessary, by supplemental feedings at least 30 min after the sessions. Weights were adjusted on a monthly basis according to growth charts provided by the supplier. The rats were housed individually in a room with a 12:12 hr reversed light/dark cycle. This research was approved by the Institutional Animal Care and Use Committee at West Virginia University.

Apparatus

Sessions were conducted in four identical operant chambers enclosed in ventilated sound-attenuating chests (Med Associates Inc., St. Albans, VT). Each chamber was 29 cm long, 22 cm high, and 24 cm deep. The ceiling and side walls were constructed of Plexiglas, and the end walls were made of stainless steel. The floor consisted of 19 stainless-steel rods 0.5 cm in diameter spaced approximately 1.3 cm apart. On the front wall were two retractable levers. Each lever was 4.4 cm wide, 1.3 cm thick, and protruded 1.9 cm into the chamber when inserted. The inside edges of the levers were spaced 11.4 cm apart (5.7 cm from the middle of the wall). The tops of the levers were positioned 8 cm from the floor. Approximately 5 cm above each lever was a white cue light (No. 1820 bulb). During all experimental sessions, only the left lever was inserted. The right lever remained retracted and inoperative. Responses on the left lever were reinforced occasionally with 45-mg food pellets dispensed into a magazine centered on the front wall. The rat's head entries into the magazine were detected with a photocell. Punishing stimuli consisted of scrambled foot shock controlled by a constant-current shock generator (Med Associates ENV-413). General illumination was provided by a houselight (No. 1820 bulb) located on the back wall. White noise (85 dB) masked extraneous sounds. Experimental events were controlled and recorded using a computer with Med Associates interfacing hardware and MED-PC IV© software.

Procedure

Sessions usually were conducted 7 days per week at approximately the same time each day. Sessions lasted until 60 reinforcers were delivered or 60 min had elapsed. A 3-min pre-session delay allowed the rats to recover from any disruptive effects of handling. During the pre-session delay, the chamber was darkened, and the levers were retracted. The start of the session was signaled by onset of the houselight and insertion of the left lever. During delivery of a reinforcer, the houselight was turned off and remained off until 2 s elapsed after the rat's head entered the food magazine.

Preliminary Training

Magazine training was conducted to establish the reinforcing efficacy of pellet delivery. Then, each lever press produced food. Once the lever press was acquired, rats were exposed to a VI schedule of reinforcement (Fleshler & Hoffman, 1962) consisting of 15 intervals presented randomly without replacement. Over the course of 8 to 10 sessions, the mean interval was raised from VI 5-s to VI 40-s, at which point preliminary training ended. The use of a VI 40-s schedule follows Sizemore and Maxwell's (1985) procedure. The independent variables were shock intensity and the IRTs eligible for shock. For some rats, relatively long IRTs were targeted by presenting shock contingent on IRTs greater than the criterion t (an IRT $> t$ schedule). For others, relatively short IRTs were targeted by presenting shock contingent on IRTs less than the criterion t (an IRT $< t$ schedule).

Design

Table 1 summarizes the experimental design. Both experiments were organized into four phases. Each phase consisted of a baseline condition followed by five shock conditions. The baseline consisted simply of a VI 40-s schedule of pellet delivery. Phases were defined in terms of the shock intensity or the percentile used to establish t during the five shock conditions that followed the baseline. In Experiment 1, shock intensity was held constant within a phase and raised across phases, beginning with an intensity of 0.05 mA. The conditions within each phase differed in terms of the percentile used to establish t . In

Experiment 2, t was manipulated across phases, either by raising the percentile used to establish t or by lowering it. In this latter experiment, t was held constant within each phase, and the conditions within the phase differed in terms of shock intensity. Within each experiment, rats were exposed to one of two shock schedules. For some rats, shock was delivered on an IRT $> t$ schedule. For others, shock was delivered on an IRT $< t$ schedule. Details on the individual shock schedules are described below. Using two different shock schedules in each experiment yielded four experimental arrangements. Although previous research suggests that shock intensity and shock contingencies involving IRTs have interactive effects, it is unclear whether these effects depend on how manipulations in shock intensity and shock contingencies are arranged. Previous research with shock intensity shows that raising the shock intensity rapidly may have a greater effect than when those same intensities are reached gradually (Azrin & Holz, 1966). The present experiments provided an additional test of the generality of the effects of shock intensity and the IRTs that produce shock by varying how manipulations of these variables were arranged.

Because response rates during the shock conditions could reflect preexisting differences in the response rates from the initial baseline rather than an effect of the shock contingency, matching was used to assign rats to the four experimental arrangements. Rats were ranked from highest to lowest on the basis of their stabilized response rates in the initial baseline. Rats with ranks 1 through 4 were then assigned to the four experimental arrangements using random assignment without replacement. This process was repeated until all 16 rats were assigned to an experimental arrangement.

Baseline

In each baseline, lever pressing was reinforced on a VI 40-s schedule, and no shock was administered. This condition remained in effect for a minimum of 13 sessions and until responding was judged stable. Stability judgments considered response rates over the last six sessions. Responding was judged stable when the difference between the mean of the first block of three sessions and the mean of the second block was within 10 % of the overall

Table 1

Shock intensities and the percentiles used to establish t across experiments. Note that every phase began with a baseline phase in which food was delivered on a variable interval schedule and no shock was administered.

Experiment	Shock Schedule	Rat	Phase	Shock Intensity and Percentile Used to Establish t Across Shock Conditions			
				Shock 1		Shock 2	
				Intensity (mA)	Percentile	Intensity (mA)	Percentile
1	IRT > t	JL5, JL8, JL14, JL16	1	0.05	90th	0.05	80th
			2	0.1	90th	0.1	80th
			3	0.2	90th	0.2	80th
			4	0.4	90th	0.4	80th
	IRT < t	JL1, JL3, JL15, JL17	1	0.05	10th	0.05	20th
			2	0.1	10th	0.1	20th
			3	0.2	10th	0.2	20th
			4	0.4	10th	0.4	20th
	IRT > t	JL2, JL11, JL13, JL19	1	0.05	80th	0.1	80th
			2	0.05	70th	0.1	70th
			3	0.05	60th	0.1	60th
			4	0.05	50th	0.1	50th
2	IRT < t	JL7, JL10, JL12, JL18	1	0.05	20th	0.1	20th
			2	0.05	30th	0.1	30th
			3	0.05	40th	0.1	40th
			4	0.05	50th	0.1	50th

mean, and there was an absence of trend as judged visually.

Shock Conditions

Following each baseline, rats were exposed to five experimental conditions in which food was presented on a VI 40-s schedule and shock was presented on either an IRT > t schedule or an IRT < t schedule. If food and shock were scheduled to be presented after the same response, shock was delivered immediately and food delivery was postponed until after the next response. The first response within a new interfood interval was ineligible for shock. On the IRT > t schedule, each IRT that exceeded the temporal criterion t was followed by shock. On the IRT < t schedule, each IRT less than t was followed by shock. The value of t was determined by cumulating the IRTs from the stable baseline sessions, calculating the deciles of the cumulative IRT distribution, and setting t to one of these deciles (details below). All shocks lasted 100 ms. The following shock intensities were used: 0.05 mA, 0.1 mA, 0.2 mA, 0.4 mA, and 0.8 mA.

Each shock condition was designed to last five sessions but was terminated early (a) if responding was suppressed below 10% of the

baseline rate for two consecutive sessions, or (b) if the IRT distributions of the most recent two sessions shifted outside the range of the IRTs that produce shock. The first situation occurred in Experiment 2 (details presented in the report of Experiment 2). The second situation never occurred. When a shock condition was terminated early, the entire phase was also terminated (i.e., the remaining shock conditions within the phase were cancelled), and the baseline condition for the next phase was started.

EXPERIMENT 1

METHOD

Table 2 shows the number of baseline sessions, shock intensities, and values of t for the four phases. (Results for Rat JL14 are limited to the first phase because this rat became ill during the second baseline condition and was removed from the study.) Baseline conditions lasted until responding stabilized (13 to 44 sessions), and shock conditions lasted five sessions for all rats except JL14. For 4 rats, shock was administered according to an IRT > t schedule. For

Table 1
(Extended)

Shock Intensity and Percentile Used to Establish t Across Shock Conditions					
Shock 3		Shock 4		Shock 5	
Intensity (mA)	Percentile	Intensity (mA)	Percentile	Intensity (mA)	Percentile
0.05	70th	0.05	60th	0.05	50th
0.1	70th	0.1	60th	0.1	50th
0.2	70th	0.2	60th	0.2	50th
0.4	70th	0.4	60th	0.4	50th
0.05	30th	0.05	40th	0.05	50th
0.1	30th	0.1	40th	0.1	50th
0.2	30th	0.2	40th	0.2	50th
0.4	30th	0.4	40th	0.4	50th
0.2	80th	0.4	80th	0.8	80th
0.2	70th	0.4	70th	0.8	70th
0.2	60th	0.4	60th	0.8	60th
0.2	50th	0.4	50th	0.8	50th
0.2	20th	0.4	20th	0.8	20th
0.2	30th	0.4	30th	0.8	30th
0.2	40th	0.4	40th	0.8	40th
0.2	50th	0.4	50th	0.8	50th

the other 4 rats, shock was administered according to an $IRT < t$ schedule. Across phases, shock intensity was raised from 0.05 mA to 0.4 mA as shown in the table. Within phases, the percentiles used to establish t were based on the baseline condition at the outset of each phase and were lowered or raised across the shock conditions. Under this arrangement, the portion of the IRT distribution that was susceptible to the shock contingency was raised gradually at each shock intensity.

RESULTS

Analyses for all experiments are based on the six stable baseline sessions and all shock sessions (five per condition unless noted otherwise). Tables 3 and 4 list the mean reinforcement rates. Across phases, reinforcement rates were similar. Within a phase, reinforcement rates in the baseline and shock conditions were similar, with rates in the shock conditions no more than 15% below the baseline rates. Reinforcement rates were stable from session to session, as shown by standard deviations of 0.06 reinforcers per minute or less. In most cases, the obtained reinforcement rates were close to the programmed rates of

1.5 reinforcers per minute, suggesting that changes in the IRT distributions or response rates should be attributed to the shock contingency and not to changes in reinforcement rates. The exception was Rat JL17, for whom the obtained reinforcement rates in the last two phases were 70% to 85% less than the programmed rate. Interestingly, this effect emerged in the baseline condition from the third phase and was not a carryover from the last shock condition in the second phase.

Tables 3 and 4 also list the mean shock rates. Across phases, raising the shock intensity had no systematic effect on the shock rates. The exception was Rat JL16, who had lower shock rates at the two highest intensities. Similar shock rates across phases indicate that the IRT distributions and response rates in the shock conditions were similar across phases. Within a phase, as the range of IRTs susceptible to shock was expanded (i.e., the percentile was lowered or raised), shock rates increased, with rates 3 to 5 times faster at the 50th percentile than at the 90th or 10th percentile. This suggests that while expanding the range of IRTs susceptible to shock may have caused the IRT distributions to shift away from the IRTs

Table 2

Experiment 1: Number of baseline sessions, shock intensities, and values of t (in s) for the four phases.

					Shock Conditions										
Shock Schedule	Rat	Phase	Baseline Sessions	Shock Intensity (mA)	t (s) at Each Percentile of Baseline IRT Distribution										
					90	80	70	60	50	10	20	30	40	50	
IRT > t	JL5	1	37	0.05	2.58	1.31	0.96	0.77	0.66	—	—	—	—	—	
		2	14	0.1	2.14	0.96	0.69	0.55	0.46	—	—	—	—	—	
		3	33	0.2	2.43	1.20	0.80	0.62	0.50	—	—	—	—	—	
		4	13	0.4	2.27	0.81	0.57	0.46	0.39	—	—	—	—	—	
	JL8	1	40	0.05	6.14	3.97	2.86	1.66	1.04	—	—	—	—	—	
		2	24	0.1	5.54	3.27	1.31	0.90	0.73	—	—	—	—	—	
		3	24	0.2	7.10	4.98	2.72	1.25	0.94	—	—	—	—	—	
		4	13	0.4	9.90	5.70	1.99	1.28	0.99	—	—	—	—	—	
	JL14	1	38	0.05	4.22	1.73	1.05	0.78	0.62	—	—	—	—	—	
	JL16	1	38	0.05	2.72	1.18	0.78	0.58	0.44	—	—	—	—	—	
		2	13	0.1	2.78	1.00	0.51	0.38	0.31	—	—	—	—	—	
		3	42	0.2	3.79	1.39	0.85	0.62	0.49	—	—	—	—	—	
		4	16	0.4	5.43	2.79	1.13	0.71	0.54	—	—	—	—	—	
	IRT < t	JL1	1	36	0.05	—	—	—	—	—	0.26	0.31	0.38	0.45	0.54
			2	32	0.1	—	—	—	—	—	0.32	0.40	0.47	0.54	0.64
			3	25	0.2	—	—	—	—	—	0.27	0.33	0.38	0.44	0.52
4			13	0.4	—	—	—	—	—	0.30	0.34	0.38	0.43	0.49	
JL3		1	38	0.05	—	—	—	—	—	0.27	0.33	0.38	0.43	0.52	
		2	13	0.1	—	—	—	—	—	0.30	0.36	0.41	0.47	0.53	
		3	30	0.2	—	—	—	—	—	0.27	0.34	0.40	0.46	0.53	
		4	13	0.4	—	—	—	—	—	0.26	0.35	0.41	0.48	0.57	
JL15		1	40	0.05	—	—	—	—	—	0.18	0.25	0.34	0.52	0.83	
		2	18	0.1	—	—	—	—	—	0.35	0.68	1.13	1.74	2.42	
		3	30	0.2	—	—	—	—	—	0.27	0.53	1.02	1.80	2.59	
		4	14	0.4	—	—	—	—	—	0.28	0.64	1.29	1.95	2.67	
JL17		1	34	0.05	—	—	—	—	—	0.27	0.37	0.48	0.62	0.80	
		2	13	0.1	—	—	—	—	—	0.40	0.50	0.60	0.72	0.88	
		3	44	0.2	—	—	—	—	—	0.40	0.53	0.67	0.83	1.04	
		4	14	0.4	—	—	—	—	—	0.52	0.69	0.83	1.00	1.29	

Note: Results for Rat JL14 are limited to the first phase because this rat was injured during the second baseline condition and was removed from the study.

that produced shock, these shifts were not severe enough to avoid shock.

To assess changes in the IRT distributions, a two-way (Shock Intensity $\times$ Percentile), repeated-measures ANOVA was conducted on the differences between the percentage of responses followed by shock and the percentage that would have been followed by shock if the IRT distributions in the shock conditions were the same as the baseline distributions (e.g., 10% when t was set to the 90th percentile, 20% when t was set to the 80th percentile, etc.). Rat JL14 was dropped from the analysis. For rats exposed to the IRT > t shock schedule, there was a significant main effect of the range of IRTs eligible for shock, that is, the percentile, on the IRT distributions ($F(4,8) = 102.82, p < .01$) Post-hoc comparisons for this and all

other experiments were made using the Least Significance Difference test, and alpha was set to .05. All of the differences were statistically significant. There was not a main effect of intensity ($F(3,6) = 3.12, p > .05$) or a significant interaction between intensity and the range of IRTs eligible for shock ($F(12,24) = 1.69, p > .05$)

For the rats exposed to the IRT < t shock schedule, there also was a significant main effect of the range of IRTs eligible for shock, or percentile, on the IRT distributions ($F(4,12) = 17.48, p < .01$). Post-hoc comparisons revealed the following significant differences: IRT distributions at the 40th and 10th percentile, the 40th and 20th percentile, the 50th and 10th percentile, the 50th and 20th percentile, the 50th and 30th percentile, and

the 50th and 40th percentile. There was not a significant main effect of intensity ($F(3,9) = 0.87$, $p > .05$) or a significant interaction between intensity and the range of IRTs eligible for shock ($F(12, 36) = 0.62$, $p > .05$).

Changes in the IRT distributions can also be assessed with Figures 1 and 2. These figures show the percentage of responses followed by shock for rats exposed to the $IRT > t$ shock schedule (Figure 1) and the $IRT < t$ shock schedule (Figure 2). Recall that each phase began with a baseline condition without shock, and the IRT distribution from the stable sessions defined the percentiles to be followed by shock in the subsequent conditions. If the IRT distribution in the shock conditions matched the baseline distributions, the percent of responses followed by shock would be those indicated by the dashed reference line in each panel. Deviations from the dashed reference line indicate shifts in the IRT distributions relative to the baseline. Functions above the dashed reference line indicate the IRT distributions shifted toward the IRTs that produced shock, that is, the targeted IRTs increased in frequency. Functions below the dashed reference line indicate the IRT distributions shifted away from the IRTs that produced shock, that is, the targeted IRTs decreased in frequency. The degree to which the obtained functions deviate from the dashed reference line indicates the size of the shift in the IRT distributions. Greater deviations represent greater shifts in the IRT distributions. By using the proportion of responses followed by shock, as opposed to the absolute number of responses followed by shock, we can assess shifts in the targeted IRTs as well as the overall IRT distributions. As the relative frequency of targeted IRTs decrease, the relative frequency of IRTs that were not targeted by the shock contingency increases, and vice versa.

In Figure 1, all of the functions fall below the dashed reference line showing that shock contingent on long IRTs punished long IRTs. Across phases, raising the shock intensity did not affect the IRT distributions. Within each phase, expanding the range of IRTs susceptible to shock (i.e., lowering the percentile used to define t) punished long IRTs and shifted the IRT distributions towards shorter IRTs. Given that the size of the deviation of the function from the reference line is greater at

the 60th and 50th percentiles than at the 90th and 80th percentiles, it seems that shock produced larger shifts in the relative IRT frequency distributions as the range of IRTs eligible for shock was expanded.

Figure 2 shows a similar analysis for rats exposed to the $IRT < t$ schedule. For most rats, the functions usually fell below the reference line meaning that shock contingent on short IRTs punished short IRTs. Across phases, raising the shock intensity had no effect on the IRT distributions. Within a phase, expanding the range of IRTs susceptible to shock punished short IRTs further, and the IRT distributions shifted toward long IRTs. Shock produced greater shifts in the IRT distributions as the range of IRTs eligible for shock was expanded. This effect is best seen in the results of JL17. Here, in all of the phases, the differences between the obtained functions and the reference line are greater at the 40th and 50th percentiles, when larger portions of the IRT distributions were susceptible to shock, than at the 10th and 20th percentiles, when smaller portions of the IRT distributions were susceptible to shock.

Proportional response rates were calculated by dividing the response rate from each shock session by the mean baseline response rate. These session-by-session proportions were then averaged to create a mean over the five sessions of each shock condition. A two-way (Shock Intensity $\times$ Percentile), repeated-measures ANOVA was conducted with Rat JL14 omitted from the analysis. For the rats exposed to the $IRT > t$ shock schedule, there was a significant main effect of the range of IRTs eligible for shock on proportional response rates ($F(4,8) = 15.26$, $p < .01$). Post-hoc comparisons revealed the following differences as statistically significant: response rates at the 90th and 80th percentiles, the 90th and 60th percentiles, and the 80th and 60th percentiles. There was not a main effect of intensity ($F(3,6) = 2.82$, $p > .05$) or an interaction between intensity and the range of IRTs eligible for shock ($F(12,24) = 1.25$, $p > .05$).

For the rats exposed to the $IRT < t$ shock schedule, there was a significant main effect of the range of IRTs eligible for shock on proportional response rates ($F(4,12) = 6.19$, $p < .01$). Post-hoc comparisons revealed the following significant differences: proportional response rates at the 50th and 10th percentiles,

Table 3

Experiment 1: Reinforcement and shock rates for rats exposed to the $IRT > t$ shock schedule. Mean rates are listed with standard deviations in parentheses, based on the stable six sessions of baseline conditions and all five sessions of the shock conditions.

			Rat JL5			
Phase	Condition	Percentile	Reinforcers per min		Shocks per min	
1 Shock = 0.05 mA	Baseline		1.45	(0.01)	-	-
	Shock 1	90th	1.44	(0.01)	4.51	(0.16)
	Shock 2	80th	1.45	(0.01)	9.11	(0.53)
	Shock 3	70th	1.45	(0.03)	13.46	(0.67)
	Shock 4	60th	1.43	(0.01)	15.84	(1.08)
	Shock 5	50th	1.44	(0.01)	20.83	(0.78)
2 Shock = 0.1 mA	Baseline		1.44	(0.02)	-	-
	Shock 1	90th	1.44	(0.01)	6.27	(0.54)
	Shock 2	80th	1.45	(0.00)	11.73	(0.51)
	Shock 3	70th	1.44	(0.01)	17.73	(0.41)
	Shock 4	60th	1.45	(0.01)	25.11	(1.23)
	Shock 5	50th	1.45	(0.01)	32.37	(1.11)
3 Shock = 0.2 mA	Baseline		1.46	(0.01)	-	-
	Shock 1	90th	1.44	(0.01)	5.87	(0.37)
	Shock 2	80th	1.44	(0.01)	10.42	(0.61)
	Shock 3	70th	1.44	(0.01)	14.31	(1.29)
	Shock 4	60th	1.43	(0.01)	18.72	(0.92)
	Shock 5	50th	1.44	(0.01)	21.86	(0.78)
4 Shock = 0.4 mA	Baseline		1.45	(0.00)	-	-
	Shock 1	90th	1.44	(0.01)	6.67	(0.21)
	Shock 2	80th	1.43	(0.01)	13.16	(0.93)
	Shock 3	70th	1.43	(0.01)	17.84	(0.52)
	Shock 4	60th	1.44	(0.01)	22.07	(1.17)
	Shock 5	50th	1.42	(0.00)	26.05	(1.69)

the 50th and 20th percentiles, and the 50th and 40th percentiles. There was not a main effect of intensity ($F(3,9) = 1.01, p > .05$) or a significant interaction ($F(12, 36) = 1.29, p > .05$).

Proportional response rates are shown in Figures 3 and 4. Hereafter, mean proportions at least one standard deviation above the reference line will be considered instances of facilitation, and those at least one standard deviation below the reference line will be considered instances of suppression. As shown in Figure 3, shock contingent on long IRTs usually facilitated responding. Across phases, proportional response rates were similar when 0.1-mA, 0.2-mA, and 0.4-mA shock was delivered, and shock intensity did not appear to have an effect on responding. Within a phase, shock facilitated responding the most when a large portion of the IRT distribution was susceptible to shock, that is, at the 60th and 50th percentiles.

Figure 4 shows proportional response rates for those rats exposed to an $IRT < t$ shock schedule. Shock contingent on short IRTs did

not always affect response rates. Across phases, raising the shock intensity did not affect responding. Within a phase, expanding the portion of the IRT distribution susceptible to shock suppressed responding. This is evidenced by lower response rates at the 40th and 50th percentiles than at the 10th and 20th percentiles.

DISCUSSION

Overall, the shock schedule had an effect on IRT distributions and response rates. Shock delivered contingent on long IRTs punished long IRTs and increased response rates. Shock contingent on short IRTs punished short IRTs and decreased response rates, although the effects on response rates were not very robust. Regardless of the IRTs that produced shock, across phases, raising the shock intensity did not affect IRT distributions or response rates. Within a phase, raising the range of IRTs eligible for shock punished the IRTs targeted by shock. There was no interaction between shock intensity and the range of IRTs suscep-

Table 3
(*Extended*)

Rat JL8		Rat JL14		Rat JL16	
Reinforcers per min	Shocks per min	Reinforcers per min	Shocks per min	Reinforcers per min	Shocks per min
1.38 (0.02)	- -	1.42 (0.01)	- -	1.44 (0.01)	- -
1.37 (0.01)	1.74 (0.12)	1.42 (0.01)	3.81 (0.45)	1.43 (0.01)	5.25 (0.47)
1.38 (0.03)	3.84 (0.15)	1.41 (0.01)	8.36 (0.42)	1.44 (0.01)	11.16 (0.24)
1.39 (0.01)	5.20 (0.22)	1.40 (0.02)	10.28 (0.76)	1.43 (0.01)	14.06 (0.74)
1.37 (0.01)	6.26 (0.33)	1.41 (0.01)	12.27 (0.87)	1.43 (0.01)	16.79 (1.18)
1.37 (0.01)	9.20 (0.30)	1.38 (0.02)	12.86 (0.82)	1.42 (0.01)	21.21 (0.83)
1.41 (0.01)	- -	- -	- -	1.44 (0.01)	- -
1.38 (0.02)	2.59 (0.28)	- -	- -	1.42 (0.01)	5.62 (0.21)
1.38 (0.02)	4.96 (0.41)	- -	- -	1.42 (0.01)	11.03 (0.16)
1.37 (0.00)	7.30 (0.10)	- -	- -	1.43 (0.01)	14.19 (0.48)
1.33 (0.01)	9.84 (0.65)	- -	- -	1.42 (0.01)	20.70 (2.56)
1.34 (0.01)	12.70 (1.07)	- -	- -	1.42 (0.01)	27.01 (2.11)
1.38 (0.01)	- -	- -	- -	1.43 (0.01)	- -
1.37 (0.01)	1.71 (0.17)	- -	- -	1.41 (0.01)	4.26 (0.40)
1.37 (0.01)	2.85 (0.19)	- -	- -	1.40 (0.03)	9.06 (0.24)
1.34 (0.00)	4.21 (0.30)	- -	- -	1.40 (0.02)	12.06 (0.78)
1.35 (0.03)	7.79 (0.20)	- -	- -	1.39 (0.02)	13.77 (1.06)
1.29 (0.05)	9.75 (0.49)	- -	- -	1.38 (0.01)	14.49 (1.39)
1.35 (0.02)	- -	- -	- -	1.41 (0.01)	- -
1.34 (0.02)	1.18 (0.19)	- -	- -	1.40 (0.01)	3.19 (0.30)
1.34 (0.01)	2.69 (0.10)	- -	- -	1.39 (0.02)	5.90 (0.54)
1.32 (0.01)	4.65 (0.14)	- -	- -	1.36 (0.01)	7.24 (0.27)
1.32 (0.03)	6.21 (0.60)	- -	- -	1.37 (0.02)	9.00 (0.59)
1.29 (0.03)	7.31 (0.61)	- -	- -	1.35 (0.03)	11.40 (0.77)

tible to shock. The effects were fairly consistent within and across rats.

The findings are consistent with previous research in a few ways. First, the punishment of long IRTs and response facilitation by shock delivered on an $IRT > t$ schedule was also reported by Arbuckle and Lattal (1992) and Sizemore and Maxwell (1985). Second, like Galbicka and Branch (1981), the present experiment found that when shock targeted long IRTs, expanding the range of IRTs followed by shock exacerbated the effects of shock. We also extended these findings to short IRTs. Third, the finding that short IRTs were sensitive to shock punishment contingencies is consistent with the results of Arbuckle and Lattal (1992).

Interestingly, when shock targeted short IRTs, response rates did not change very much. This may be due, in part, to the mathematical relation between response rates and IRTs. In general, when shock is delivered on a differential schedule, it produces graded effects in IRT distributions and response rates (e.g., Galbicka & Branch, 1981; Sizemore &

Maxwell, 1985), however, changes in IRT distributions do not always manifest as changes in response rates. It is possible that response rates are more sensitive to changes in long IRTs than to changes in short IRTs. For example, in Arbuckle and Lattal's (1992) research, shock punished short IRTs without producing consistent concomitant changes in response rates. By contrast, in Galbicka and Branch's (1981) and Sizemore and Maxwell's (1985) research, shock punished long IRTs and did affect response rates.

Alternatively, the limited effect on response rates may be caused by low response rates in the baseline. When baseline response rates are low, there is limited room for response rates to decrease without facing the countervailing factors of reinforcement. By contrast, when baseline response rates are high, there is more room for these rates to decrease without decreasing the rate of reinforcement. Low baseline response rates may also explain why, in some cases, shock contingent on short IRTs increased responding. When absolute response

Table 4

Experiment 1: Reinforcement and shock rates for rats exposed to the $IRT < t$ shock schedule. Mean rates are listed with standard deviations in parentheses, based on the stable six sessions of baseline conditions and all five sessions of the shock conditions.

Phase	Condition	Percentile	Rat JL1			
			Reinforcers per min		Shocks per min	
1	Baseline		1.42	(0.02)	-	-
Shock = 0.05 mA	Shock 1	10th	1.42	(0.01)	5.17	(1.81)
	Shock 2	20th	1.42	(0.01)	19.25	(4.84)
	Shock 3	30th	1.42	(0.02)	21.40	(7.82)
	Shock 4	40th	1.41	(0.02)	16.13	(4.88)
	Shock 5	50th	1.35	(0.02)	7.35	(3.25)
2	Baseline		1.35	(0.02)	-	-
Shock = 0.1 mA	Shock 1	10th	1.36	(0.00)	1.42	(0.46)
	Shock 2	20th	1.37	(0.02)	6.58	(1.55)
	Shock 3	30th	1.37	(0.03)	9.99	(3.87)
	Shock 4	40th	1.35	(0.02)	6.70	(2.68)
	Shock 5	50th	1.35	(0.03)	10.70	(3.99)
3	Baseline		1.39	(0.01)	-	-
Shock = 0.2 mA	Shock 1	10th	1.38	(0.04)	2.01	(1.09)
	Shock 2	20th	1.36	(0.02)	3.83	(1.45)
	Shock 3	30th	1.38	(0.01)	10.03	(2.60)
	Shock 4	40th	1.37	(0.02)	10.02	(1.65)
	Shock 5	50th	1.39	(0.01)	14.43	(5.03)
4	Baseline		1.37	(0.02)	-	-
Shock = 0.4 mA	Shock 1	10th	1.39	(0.01)	2.10	(0.82)
	Shock 2	20th	1.39	(0.03)	5.59	(1.54)
	Shock 3	30th	1.38	(0.02)	7.76	(2.75)
	Shock 4	40th	1.38	(0.01)	11.37	(1.39)
	Shock 5	50th	1.34	(0.02)	13.08	(4.83)

rates are low, small increases or decreases translate into large differences in relative rates. Unlike previous research (e.g., Appel, 1968; Filby & Appel, 1966; Galbicka & Branch, 1981; Lande, 1981; Sizemore & Maxwell, 1985), shock intensity did not affect IRT distributions or response rates, and there was no interaction between shock intensity and the range of IRTs eligible for shock. This may be because the shock duration was too brief, the no-shock baseline strengthened responding, or shock intensity was manipulated across phases. If the shock duration of 100 ms was too brief, low and high shock intensities may have been functionally equivalent. In addition, the return to a no-shock baseline between phases may have strengthened responding, particularly long IRTs, in such a way that responding became resistant to shock in the subsequent shock conditions. Previous research suggests that responding can become resistant to punishment when punishment periods are repeatedly separated by periods of no punishment, sometimes referred to as vacations from punishment (Orme-Johnson, 1967). Lastly, shock intensity

may not have had an effect because it was raised across phases rather than within phases. Shock intensity is another way of conceptualizing the punisher magnitude. Research on reinforcement magnitude has shown that manipulations made across phases or on a simple schedule produce less reliable effects than those made within phases or on concurrent schedules (Bonem & Crossman, 1988; Catania, 1963). The same may be true with punisher magnitude.

EXPERIMENT 2
METHOD

Table 5 shows the number of baseline sessions, percentiles used to establish t , and values of t . During the second baseline, Rat JL7 became injured with a pulled muscle and response rates decreased. Remedial training was conducted by changing the schedule of food delivery to a fixed-ratio (FR) 1 schedule. Once responding recovered, food was delivered on a VI 5-s schedule, and the mean

Table 4
(Extended)

Rat JL3		Rat JL15		Rat JL17	
Reinforcers per min	Shocks per min	Reinforcers per min	Shocks per min	Reinforcers per min	Shocks per min
1.41	(0.02)	-	-	1.44	(0.01)
1.42	(0.01)	4.66	(0.79)	1.42	(0.01)
1.42	(0.01)	9.09	(0.99)	1.42	(0.02)
1.42	(0.01)	13.13	(2.99)	1.41	(0.01)
1.42	(0.01)	16.45	(2.79)	1.42	(0.02)
1.40	(0.01)	17.26	(1.55)	1.40	(0.01)
1.42	(0.00)	-	-	1.36	(0.02)
1.41	(0.01)	4.90	(1.31)	1.39	(0.02)
1.42	(0.01)	9.12	(1.96)	1.38	(0.01)
1.43	(0.01)	12.24	(1.60)	1.38	(0.02)
1.42	(0.01)	14.13	(1.10)	1.39	(0.01)
1.40	(0.01)	12.70	(2.06)	1.34	(0.01)
1.43	(0.02)	-	-	1.40	(0.01)
1.42	(0.01)	3.75	(1.10)	1.38	(0.01)
1.41	(0.02)	6.89	(2.72)	1.39	(0.01)
1.41	(0.01)	8.02	(2.38)	1.38	(0.02)
1.39	(0.01)	10.71	(2.68)	1.38	(0.03)
1.41	(0.01)	16.98	(4.80)	1.34	(0.02)
1.42	(0.01)	-	-	1.37	(0.04)
1.41	(0.01)	3.59	(1.18)	1.37	(0.02)
1.40	(0.01)	5.79	(1.45)	1.37	(0.02)
1.39	(0.03)	8.25	(1.96)	1.38	(0.00)
1.40	(0.02)	12.19	(4.04)	1.38	(0.01)
1.38	(0.01)	10.42	(3.41)	1.36	(0.02)
				2.75	(0.32)
				2.43	(1.22)
				3.75	(1.00)
				5.54	(1.03)
				9.56	(2.27)
				-	-
				1.33	(0.05)
				1.32	(0.02)
				1.31	(0.04)
				1.33	(0.03)
				1.35	(0.03)
				1.28	(0.06)
				1.25	(0.03)
				1.26	(0.04)
				1.24	(0.03)
				1.28	(0.06)
				1.29	(0.01)
				1.28	(0.02)
				1.04	(0.40)
				1.22	(0.04)
				1.25	(0.04)
				1.24	(4.04)
				1.18	(0.10)
				-	-
				1.24	(0.42)
				3.11	(0.87)
				4.88	(0.80)
				6.24	(1.50)
				5.79	(2.04)
				-	-
				1.22	(0.77)
				1.93	(0.68)
				2.61	(1.17)
				5.33	(1.69)
				6.02	(1.65)
				-	-
				0.49	(0.42)
				1.61	(1.44)
				0.98	(0.44)
				3.41	(1.63)
				3.25	(0.80)
				-	-
				0.53	(0.17)
				1.61	(0.96)
				2.58	(0.77)
				2.76	(1.46)
				2.22	(1.37)

interval was raised gradually until the standard VI 40-s schedule was reached. For all other rats, baseline conditions lasted until responding stabilized (13 to 56 sessions) and shock conditions lasted five sessions (unless otherwise noted). For 4 rats, shock was administered according to an $IRT > t$ schedule. For the other 4 rats, shock was administered according to an $IRT < t$ schedule.

RESULTS

For Rat JL2, results at the 70th percentile exclude the last shock condition because 0.4-mA shocks suppressed responding below 10% of the baseline rate, and the 0.8-mA condition was not conducted. For all other conditions, analyses are based on the six stable baseline sessions and all five shock sessions.

Tables 6 and 7 list the mean reinforcement rates. Across phases, reinforcement rates were similar for most rats. Within a phase, at 0.05 mA, 0.1 mA, and 0.2 mA, reinforcement rates were close to the programmed rates. Reinforcement rates in these conditions were within 0.1 reinforcers per minute of the

baseline rates and stable, with standard deviations of 0.05 reinforcers per minute or less. When 0.4-mA shock was delivered, reinforcement rates decreased for some rats by nearly 80%. When 0.8-mA shock was delivered, reinforcement rates were often low, dropping to 30% to 60% below the baseline rates, and in some cases, fell to near zero. The decrease at 0.8 mA was much more severe for the rats exposed to the $IRT > t$ schedule than for those exposed to the $IRT < t$ schedule. Given these findings, a loss of reinforcement cannot explain changes in IRT distributions or response rates in the 0.05-mA, 0.1-mA, and 0.2-mA conditions but may explain low response rates in the 0.4-mA and 0.8-mA shock conditions, at least when shock was delivered on an $IRT > t$ schedule.

Tables 6 and 7 also list the mean shock rates. Because the range of IRTs eligible for shock was raised across phases, we would expect the shock rates to increase across phases, too, assuming that the baseline distributions remained the same across phases. For rats exposed to the $IRT > t$ shock schedule, shock

Figure 1. Experiment 1: Percent of responses followed by shock for rats exposed to the $IRT > t$ shock schedule. Error bars extend one standard deviation above and below the mean of each shock condition. The reference line in each panel indicates the percentage of responses that would have been followed by shock if the IRT distributions in the shock conditions matched the baseline distributions. Percentiles are listed in descending order because this order reflects the increase in the portion of the IRT distribution susceptible to the shock contingency. Note that the scale of the ordinate is different for Rat JL8 and Rat JL5.

rates increased across phases as expected, but for rats exposed to the $IRT < t$ shock schedule, shock rates were similar across phases, suggesting that the baseline IRT distributions differed across phases. Within a phase, shock rates depended on the shock schedule. For rats exposed to the $IRT > t$ schedule, shock rates were similar from 0.05 mA to 0.4 mA but dropped by approximately 50% at 0.8 mA. Rat JL13 was the exception. For rats exposed to the $IRT < t$ schedule, shock rates decreased as the intensity was raised, indicating that fewer

short IRTs were emitted. In these conditions, the IRT distributions shifted away from short IRTs so much that the rats were able to avoid shock by responding slowly.

To assess changes in the IRT distributions, a two-way (Shock Intensity $\times$ Percentile), repeated-measures ANOVA was conducted on the differences between the percentage of responses followed by shock and those indicated by the reference line. Rat JL2 was omitted from the analysis. For rats exposed to the $IRT > t$ schedule, there was no effect of the range

Figure 2. Experiment 1: Percent of responses followed by shock for rats exposed to the $IRT < t$ shock schedule. Error bars extend one standard deviation above and below the mean. The reference line in each panel indicates the percentage of responses that would have been followed by shock if the IRT distributions in the shock conditions matched the baseline distributions. Note that the scale of the ordinate varies across rats.

of IRTs susceptible to shock ($F(4,8) = 3.60$, $p > .05$) or shock intensity ($F(3,6) = 2.47$, $p > .05$). There was not an interaction between shock intensity and the range of IRTs susceptible to shock ($F(12, 24) = 0.19$, $p > .05$).

For rats exposed to the $IRT < t$ schedule, there was a main effect of the range of IRTs susceptible to shock, that is, the percentile, on IRT distributions ($F(3,9) = 31.20$, $p < .01$). All post-hoc comparisons were statistically significant. There was a main effect of intensity ($F(4,12) = 48.34$, $p < .01$). All post-hoc comparisons involving 0.2 mA, 0.4 mA, or 0.8

mA were statistically significant. There was also a significant interaction between the range of IRTs susceptible to shock and shock intensity ($F(12, 36) = 4.03$, $p < .01$).

Figures 5 and 6 show shifts in the IRT distributions for rats exposed to the $IRT > t$ schedule (Figure 5) and the $IRT < t$ schedule (Figure 6). In these figures, the reference lines increased across phases because the range of IRTs eligible for shock was expanded across phases. The relevant feature, therefore, is not the raising of the obtained functions across phases but the deviations between these

Figure 3. Experiment 1: Proportion of baseline response rates across all shock conditions for rats exposed to the IRT > t shock schedule. Error bars extend one standard deviation above and below the mean. The dashed lines represent one standard deviation above and below the mean baseline response rate.

Figure 4. Experiment 1: Proportion of baseline response rates across all shock conditions for rats exposed to the IRT < t shock schedule. Error bars extend one standard deviation above and below the mean. The dashed lines represent one standard deviation above and below the mean baseline response rate.

Table 5

Experiment 2: Number of baseline sessions, shock intensity, and values of t (in s) for the four phases.

Shock Schedule	Rat	Phase	Baseline Sessions	Percentile of Baseline IRT Distribution	Shock Conditions				
					t (s) at Each Shock Intensity				
					0.05	0.1	0.2	0.4	0.8
IRT > t	JL2	1	38	80	1.08	1.08	1.08	1.08	1.08a
		2	17	70	0.49	0.49	0.49	0.49a	—
		3	13	60	0.40	0.40	0.40	0.40	0.40a
		4	14	50	0.38	0.38	0.38	0.38	0.38a
	JL11	1	40	80	2.26	2.26	2.26	2.26	2.26a
		2	27	70	0.87	0.87	0.87	0.87	0.87a
		3	41	60	0.50	0.50	0.50	0.50	0.50
		4	16	50	0.32	0.32	0.32	0.32	0.32b
	JL13	1	39	80	1.81	1.81	1.81	1.81	1.81
		2	26	70	0.74	0.74	0.74	0.74	0.74
		3	23	60	0.58	0.58	0.58	0.58	0.58
		4	13	50	0.47	0.47	0.47	0.47	0.47
	JL19	1	37	80	5.56	5.56	5.56	5.56	5.56
		2	19	70	3.11	3.11	3.11	3.11	3.11a
		3	42	60	1.08	1.08	1.08	1.08	1.08b
		4	15	50	0.33	0.33	0.33	0.33	0.33a
IRT < t	JL7	1	41	20	0.32	0.32	0.32	0.32	0.32
		2	56c	30	0.41	0.41	0.41	0.41	0.41
		3	14	40	0.77	0.77	0.77	0.77	0.77
		4	15	50	1.59	1.59	1.59	1.59	1.59
	JL10	1	40	20	0.37	0.37	0.37	0.37	0.37
		2	13	30	0.63	0.63	0.63	0.63	0.63
		3	42	40	0.34	0.34	0.34	0.34	0.34
		4	15	50	0.47	0.47	0.47	0.47	0.47
	JL12	1	39	20	0.23	0.23	0.23	0.23	0.23
		2	13	30	0.33	0.33	0.33	0.33	0.33
		3	41	40	0.49	0.49	0.49	0.49	0.49
		4	14	50	0.74	0.74	0.74	0.74	0.74b
	JL18	1	33	20	0.25	0.25	0.25	0.25	0.25
		2	21	30	0.40	0.40	0.40	0.40	0.40
		3	31	40	0.36	0.36	0.36	0.36	0.36
		4	15	50	0.44	0.44	0.44	0.44	0.44

^a Only 2 sessions were conducted in this condition. ^b Only 3 sessions were conducted in this condition. ^c Total includes sessions in which JL7 was trained on a VI 40-s schedule of pellet delivery and excludes the 31 sessions of remedial training described in the text.

functions and the reference lines. Although the statistical analyses showed no significant effects for rats exposed to the IRT > t shock schedule, from Figure 5, it appears that shock contingent on long IRTs punished long IRTs except when shock intensity was 0.8 mA. For all rats except JL13, 0.8-mA shock decreased responding so severely that the few responses that were emitted terminated long IRTs, thereby increasing the frequency of long IRTs relative to baseline.

As shown in Figure 6, shock contingent on short IRTs punished short IRTs. Across phases, expanding the range of IRTs susceptible to shock shifted the IRT distributions away from

short IRTs and toward long ones. This is shown by slightly greater deviations of the functions from the reference line across phases. Within phases, raising the shock intensity punished IRTs other than those followed by shock, and the IRT distributions shifted toward long IRTs. This is evidenced by the greater differences between the functions and the reference line at high intensities than at low intensities. At 0.8 mA, for all rats except JL12, very few responses were followed by shock. In these conditions, the IRT distributions shifted away from short IRTs so much that the rats were able to avoid shock by responding slowly. For most rats, the effects of shock intensity became

greater as the range of IRTs susceptible to shock was raised.

To assess changes in proportional response rates, a two-way (Percentile $\times$ Shock Intensity), repeated-measures ANOVA was conducted. As noted previously, there were no data for Rat JL2 at the 70th percentile phase during the 0.8-mA condition. The very low response rates in and early termination of the preceding 0.4-mA condition suggested that 0.8-mA shock would have suppressed responding further. For this reason, zeroes were entered for Rat JL2 at the 70th percentile phase during the 0.8-mA condition. For rats exposed to the $IRT > t$ shock schedule, there was a main effect of shock intensity on proportional response rates ($F(4,12) = 12.02$, $p < .01$). All post-hoc comparisons at 0.8 mA were statistically significant. There was no main effect of the range of IRTs eligible for shock, that is, the percentile, ($F(3,9) = 1.07$, $p > .05$) or an interaction between the range of IRTs eligible for shock and shock intensity ($F(12,36) = 1.78$, $p > .05$).

For rats exposed to the $IRT < t$ shock schedule, there was a main effect of shock intensity on proportional response rates ($F(4, 12) = 60.83$, $p < .01$). All of the post-hoc comparisons involving 0.4 mA or 0.8 mA were statistically significant as was the difference between response rates at 0.1 mA and 0.2 mA. There was not a main effect of the range of IRTs susceptible to shock ($F(3,9) = 0.65$, $p > .05$) or an interaction between shock intensity and the range of IRTs susceptible to shock ($F(12,36) = 1.83$, $p > .05$).

Proportional response rates are shown in Figures 7 and 8 and were calculated as described for Figure 3. As shown in Figure 7, shock contingent on long IRTs usually increased responding except at 0.8 mA. Within a phase, intensities of 0.4 mA or less usually facilitated responding whereas 0.8-mA shock usually suppressed responding, and in some cases, it suppressed responding to near zero. Rat JL13 was the exception. For this rat, 0.8-mA shock facilitated responding half of the time.

Figure 8 shows that shock contingent on short IRTs usually suppressed responding, except for Rat JL12, for whom response rates in the shock conditions were often similar to those in the baseline conditions. Within a phase, high shock intensities suppressed responding more than low shock intensities. At

0.05 mA, response rates were usually 10% to 20% lower than baseline levels whereas at 0.8 mA, response rates were often 40% to 60% lower than baseline levels. Interestingly, response rates in the 0.8-mA condition were not as low when shock was followed short IRTs as they were when shock followed long IRTs.

DISCUSSION

Overall, the effects of shock depended on the shock schedule. Shock contingent on long IRTs increased response rates except when 0.8-mA shock was used. The IRT distributions in the shock conditions differed from the baseline distributions but did not significantly differ from one another, perhaps because data from one rat was omitted. Raising the range of IRTs eligible for shock had no effect on the IRT distributions or response rates. Shock intensity did not affect the IRT distributions but did affect response rates. Specifically, shock intensities of 0.4 mA or less increased response rates, whereas 0.8-mA shock decreased response rates dramatically for all rats except one. There was no interaction between the range of IRTs eligible for shock (i.e., the percentile) and shock intensity.

In most cases, shock contingent on short IRTs punished short IRTs and decreased responding. Expanding the range of IRTs susceptible to shock punished short IRTs further but did not affect response rates. Shock intensity affected both IRT distributions and response rates. In general, high shock intensities punished short IRTs and decreased response rates more than low shock intensities. These effects were consistent within and across rats.

Findings from the present experiment are consistent with previous research in several ways. First, the current experiment showed that shock could facilitate or suppress responding by delivering shock contingent on long IRTs or short IRTs, respectively. Similar findings on response facilitation were reported by Galbicka and Branch (1981), Arbuckle and Lattal (1992; Experiment 2), Sizemore and Maxwell (1985), and in Experiment 1. Arbuckle and Lattal (1987; 1992, Experiment 2) also found that shocks contingent on short IRTs suppressed responding. Second, in the present experiment, shock contingent on short IRTs punished these IRTs. Both Arbuckle

Table 6

Experiment 2: Reinforcement and shock rates for rats exposed to the $IRT > t$ shock schedule. Mean rates are listed with standard deviations in parentheses, based on the stable six sessions of baseline conditions and all five sessions of the shock conditions.

Phase	Condition	Intensity (mA)	Rat JL2			
			Reinforcers per min		Shocks per min	
1	Baseline		1.43	(0.01)	-	-
80th percentile	Shock 1	0.05	1.44	(0.01)	8.52	(0.63)
	Shock 2	0.1	1.43	(0.01)	7.74	(0.27)
	Shock 3	0.2	1.42	(0.02)	7.14	(0.26)
	Shock 4	0.4	1.40	(0.02)	6.40	(0.62)
	Shock 5	0.8	0.04	(0.04) ^b	0.25	(0.22)
2	Baseline		1.43	(0.02)	-	-
70th percentile	Shock 1	0.05	1.40	(0.01)	17.94	(0.94)
	Shock 2	0.1	1.42	(0.01)	15.42	(0.31)
	Shock 3	0.2	1.36	(0.13)	12.05	(1.39)
	Shock 4	0.4	0.01	(0.01) ^b	0.19	(0.02)
	Shock 5	0.8	-	-	-	-
3	Baseline		1.44	(0.02)	-	-
60th percentile	Shock 1	0.05	1.41	(0.01)	24.48	(2.45)
	Shock 2	0.1	1.41	(0.01)	24.88	(3.41)
	Shock 3	0.2	1.42	(0.01)	23.50	(1.18)
	Shock 4	0.4	0.90	(0.65)	14.22	(10.20)
	Shock 5	0.8	0.00	(0.00) ^b	0.09	(0.01)
4	Baseline		1.45	(0.01)	-	-
50th percentile	Shock 1	0.05	1.41	(0.01)	27.52	(2.93)
	Shock 2	0.1	1.42	(0.02)	26.51	(1.08)
	Shock 3	0.2	1.38	(0.07)	21.26	(1.31)
	Shock 4	0.4	1.15	(0.40)	15.83	(5.88)
	Shock 5	0.8	0.00	(0.00) ^b	0.15	(0.08)

^a Only 3 sessions were conducted in this condition. ^b Only 2 sessions were conducted in this condition.

and Lattal (1992; Experiment 2) and Experiment 1 reported similar findings. Third, shock intensity affected response rates. Several studies have reported an effect of shock intensity. While some studies have reported response facilitation with low shock intensities (e.g., Appel, 1968; Filby & Appel, 1966; Lande, 1981; Sizemore & Maxwell, 1985) and response suppression with high ones (e.g., Azrin, 1956, 1959, 1960; Azrin & Holz, 1961; Azrin, Holz, & Hake, 1963; Holz, Azrin, & Ulrich, 1963), others have reported response suppression across all intensities used, with high shock intensities suppressing response rates more than low shock intensities. These last studies include those by Filby and Appel (1966) and Hake, Azrin, and Oxford (1967) among others (e.g., Appel & Peterson, 1965; Azrin, 1959; Azrin & Holz, 1961; Azrin et al., 1963; Borocz, Storms, & Broen, 1964; Sizemore & Maxwell, 1985). Fourth, severe suppression occurred when 0.8-mA shock was delivered on an $IRT > t$ shock schedule. Severe suppression by high shock intensities has been found by Filby and

Appel (1966) and Azrin (1959). Finally, as in Experiment 1, there was no interaction between the shock intensity and the range of IRTs susceptible to shock on response rates. In contrast to Experiment 1, expanding the range of IRTs susceptible to shock did not affect response rates or IRT distributions. This may be due to habituation across phases. Because each phase consisted of 25 shock sessions, the cumulative number of shocks increased dramatically across phases. (Rat JL2 was the exception, with less than 20 shock sessions in the 70th percentile phase.) This may have made the shock delivered in the later phases less effective. This habituation, if present, would have been confounded with raises in the range of IRTs susceptible to shock. Additionally, in the present study, the range of IRTs susceptible to shock was raised four times, as opposed to five in Experiment 1. Moreover, the 80th percentile, as opposed to the 90th, was used as the initial value of t . Recall that in Experiment 1, the greatest changes in IRT distributions and response

Table 6
(Extended)

Rat JL11		Rat JL13		Rat JL19	
Reinforcers per min	Shocks per min	Reinforcers per min	Shocks per min	Reinforcers per min	Shocks per min
1.43	(0.01)	-	-	1.44	(0.00)
1.43	(0.01)	6.75	(0.71)	1.44	(0.01)
1.42	(0.01)	5.83	(0.52)	1.43	(0.01)
1.44	(0.01)	5.21	(0.68)	1.42	(0.01)
1.43	(0.04)	5.12	(0.28)	1.42	(0.01)
0.03	(0.01) ^b	0.14	(0.10)	0.86	(0.42)
1.38	(0.03)	-	-	4.37	(2.11)
1.36	(0.05)	11.44	(0.73)	0.36	(0.50)
1.40	(0.02)	10.19	(1.08)	1.38	(0.01)
1.42	(0.01)	10.36	(0.57)	1.38	(0.01)
1.21	(0.30)	7.82	(2.02)	1.34	(0.03)
0.03	(0.00) ^b	0.25	(0.05)	1.34	(0.05)
1.40	(0.03)	-	-	1.34	(0.02)
1.39	(0.03)	19.15	(1.63)	1.35	(0.02)
1.41	(0.02)	16.90	(1.17)	0.03	(0.00) ^b
1.40	(0.03)	16.11	(0.86)	10.65	(0.40)
1.41	(0.02)	15.15	(0.59)	-	-
1.04	(0.24)	8.06	(2.83)	1.28	(0.04)
1.42	(0.01)	-	-	1.29	(0.02)
1.39	(0.00)	30.86	(5.09)	1.26	(0.05)
1.38	(0.02)	23.09	(2.20)	1.28	(0.05)
1.38	(0.02)	22.05	(2.90)	1.24	(0.03)
1.35	(0.05)	21.83	(4.04)	1.24	(0.03)
0.50	(0.62) ^a	6.65	(8.55)	0.41	(0.58) ^a
				1.22	(0.03)
				1.27	(0.02)
				1.24	(0.03)
				1.27	(0.04)
				1.28	(0.02)
				0.00	(0.00) ^b
				0.03	(0.01)

rates occurred when the range of IRTs eligible for shock was raised for the first time (i.e., the percentile used for t was lowered from the 90th to the 80th). Subsequent manipulations may produce less dramatic differences. By excluding the 90th percentile as a value of t , the present experiment may have missed dramatic changes in IRT distributions and response rates, resulting in the range of IRTs susceptible to shock having no effect. Lastly, as noted previously, the use of a no-shock baseline between each shock phase may have acted as vacations from punishment and, when repeated, may have made responding resistant to punishment (Orme-Johnson, 1967).

It is interesting that in both experiments, there was no effect for the variable manipulated across phases. However, when the same variable was manipulated within phases, an effect was observed. Perhaps some variables only exert an effect when manipulated quickly within phases rather than slowly across phases, as was previously discussed with reinforcer magnitude (e.g., Bonem & Crossman, 1988).

In the present experiment, although 0.8-mA shock typically suppressed responding, the amount of suppression depended on the IRTs followed by shock. On the $IRT > t$ shock schedule, 0.8-mA shock severely suppressed responding. By contrast, on the $IRT < t$ shock schedule, responding decreased but was still maintained at 0.8 mA. Previous research shows that 0.8-mA shock not only decreases but often eliminates responding (Appel, 1968; Filby & Appel, 1966; Boroczi et al., 1964; Dworkin, Bimle, & Miyauchi, 1989). The inconsistent effects of 0.8-mA shock in Experiment 2 may be better understood by examining how each shock schedule interacted with shock-elicited behavior. Suppose that, regardless of the shock schedule, 0.8-mA shock elicited responses, such as freezing, that were incompatible with lever pressing (e.g. Azrin, 1956; Galbicka & Platt, 1984). On the $IRT > t$ shock schedule, freezing would have caused the IRTs to become longer, and the probability of shock would have increased. Under this arrangement, responses elicited by high shock intensities may have interfered with the rat's ability to avoid shock.

Table 7

Experiment 2: Reinforcement and shock rates for rats exposed to the $IRT < t$ shock schedule. Mean rates are listed with standard deviations in parentheses, based on the stable six sessions of baseline conditions and all five sessions of the shock conditions.

Phase	Condition	Intensity (mA)	Rat JL7			
			Reinforcers per min		Shocks per min	
1	Baseline		1.32	(0.02)	-	-
20th percentile	Shock 1	0.05	1.32	(0.03)	2.14	(0.71)
	Shock 2	0.1	1.28	(0.03)	2.38	(0.39)
	Shock 3	0.2	1.29	(0.04)	2.94	(1.23)
	Shock 4	0.4	1.27	(0.04)	1.11	(0.98)
	Shock 5	0.8	1.18	(0.07)	0.13	(0.12)
2	Baseline		1.32	(0.03)	-	-
30th percentile	Shock 1	0.05	1.29	(0.02)	2.28	(0.93)
	Shock 2	0.1	1.29	(0.02)	2.63	(1.04)
	Shock 3	0.2	1.29	(0.01)	1.67	(0.28)
	Shock 4	0.4	1.30	(0.01)	2.01	(0.51)
	Shock 5	0.8	1.29	(0.01)	0.46	(0.19)
3	Baseline		1.30	(0.01)	-	-
40th percentile	Shock 1	0.05	1.31	(0.01)	3.18	(1.64)
	Shock 2	0.1	1.32	(0.02)	2.74	(1.43)
	Shock 3	0.2	1.32	(0.01)	1.98	(0.67)
	Shock 4	0.4	1.31	(0.02)	0.93	(0.49)
	Shock 5	0.8	1.29	(0.01)	0.46	(0.52)
4	Baseline		1.34	(0.01)	-	-
50th percentile	Shock 1	0.05	1.33	(0.02)	3.14	(0.72)
	Shock 2	0.1	1.33	(0.01)	2.61	(0.93)
	Shock 3	0.2	1.33	(0.01)	2.20	(0.62)
	Shock 4	0.4	1.32	(0.02)	1.91	(0.71)
	Shock 5	0.8	1.31	(0.01)	0.21	(0.07)

^a There were only 3 sessions in this condition.

By contrast, on the $IRT < t$ shock schedule, if high shock intensities elicited freezing, the IRT would still become longer, but on this shock schedule, longer IRTs would reduce the probability of shock. Hence, on this schedule, the responses elicited by high shock intensities helped the rat to avoid shock. Although responding under both shock schedules may be influenced by the same shock-elicited responses, on the $IRT > t$ shock schedule, the elicited responses produced additional shocks whereas on the $IRT < t$ shock schedule, the elicited responses produced fewer shocks.

GENERAL DISCUSSION

In two experiments, we examined the suppressive and facilitative effects of shock by manipulating the shock schedule, shock intensity, and the IRTs that produced shock. In general, shock contingent on long IRTs punished long IRTs and increased response rates, except when 0.8-mA shock was used. Shock contingent on short IRTs punished

short IRTs and decreased response rates. Changes in the IRT distributions were not always reflected in response rates. Expanding the range of IRTs susceptible to shock (i.e., the percentile used to establish t) usually exacerbated the effects of shock when this variable was manipulated within phases (Experiment 1) but not across phases (Experiment 2). Shock intensity also had an effect when it was manipulated within phases (Experiment 2) but not across phases (Experiment 1). Low intensities increased or decreased responding when the IRTs that produced shock were long or short, respectively. High intensities consistently decreased responding, although this decrease was greatest when long IRTs produced shock. There was not a statistically significant interaction between the shock intensity and the range of IRTs susceptible to shock.

The present results extend previous findings by examining a broader range of IRT durations and shock intensities. We also examined the effects of the range of IRTs

Table 7
(Extended)

Rat JL10		Rat JL12		Rat JL18	
Reinforcers per min	Shocks per min	Reinforcers per min	Shocks per min	Reinforcers per min	Shocks per min
1.45	(0.01)	-	-	1.47	(0.00)
1.43	(0.01)	9.24	(0.70)	1.46	(0.00)
1.43	(0.01)	8.91	(1.88)	1.46	(0.01)
1.42	(0.01)	5.94	(2.25)	1.47	(0.01)
1.40	(0.06)	2.97	(1.55)	1.45	(0.00)
1.25	(0.14)	0.18	(0.14)	1.43	(0.01)
1.38	(0.01)	-	-	1.45	(0.00)
1.38	(0.01)	6.26	(1.64)	1.46	(0.00)
1.37	(0.03)	4.63	(1.13)	1.46	(0.00)
1.38	(0.01)	5.22	(1.35)	1.46	(0.01)
1.38	(0.02)	1.17	(1.30)	1.46	(0.00)
1.36	(0.01)	0.31	(0.16)	1.45	(0.01)
1.40	(0.01)	-	-	1.45	(0.01)
1.38	(0.02)	11.56	(3.69)	1.44	(0.01)
1.39	(0.01)	11.00	(2.49)	1.43	(0.01)
1.39	(0.01)	5.87	(1.58)	1.44	(0.00)
1.40	(0.00)	9.79	(2.84)	1.44	(0.01)
1.34	(0.06)	0.54	(0.42)	1.43	(0.01)
1.38	(0.01)	-	-	1.45	(0.01)
1.38	(0.01)	14.17	(4.19)	1.44	(0.01)
1.37	(0.01)	9.41	(2.71)	1.44	(0.00)
1.37	(0.01)	8.15	(1.86)	1.44	(0.01)
1.36	(0.03)	7.92	(3.35)	1.44	(0.01)
1.32	(0.03)	0.41	(0.20)	1.42	(0.02) ^a
				1.30	(0.92)
				1.39	(0.03)

susceptible to shock and included a shock contingency targeting short IRTs. In doing so, these findings help to identify the variables and procedural arrangements that determine facilitation or suppression under response-dependent shock schedules. Although the shock intensity, range of IRTs susceptible to shock, and type of IRTs that produced shock were all important determinants of responding, the former two only exerted an effect when they were manipulated quickly, that is, within phases, as opposed to slowly, that is, across phases. Similar findings have been reported when reinforcer magnitude is manipulated within phases or on concurrent schedules (Bonem & Crossman, 1988; Catania, 1963). Although the reason for this is unclear, one possibility is that the punishing or reinforcing efficacy of a stimulus is determined by the local context and recent history, and gradual manipulations of intensity or magnitude are not salient.

In the present study, low shock intensities and the punishment of long IRTs were necessary for response facilitation. These

findings are relevant in applied settings where practitioners may be reluctant to employ punishment procedures for fear of inadvertently increasing problem behavior (Axelrod, 1990; Azrin & Holz, 1966; Hutchinson, 1977; Newsome, Favell, & Rincover, 1983; Pazulinec, Meyerrose, & Sajwaj, 1983). In applied settings, the use of punishment is sometimes reserved for cases of intense, albeit low-rate, problem behavior that has not been sufficiently reduced through other methods. Given that the punishment of long IRTs facilitated responding in the current study, it is possible that putative punishers fail to decrease behavior in applied settings because the punisher has followed an intense, but low-rate, problem behavior and punished long IRTs rather than response rates. In addition, the practical constraints of applied settings may make it difficult to arrange quick increases in punisher intensity, causing therapists to rely on more gradual, ineffective manipulations. More research on the arrangement of punisher manipulations in applied settings is needed.

Figure 5. Experiment 2: Percent of responses followed by shock for rats exposed to the $IRT > t$ shock schedule. Error bars extend one standard deviation above and below the mean. The reference line indicates the percent of baseline responses that satisfied the shock contingency.

SHOCK INTENSITY

Previous research indicates that shock intensity is one of the most powerful determinants of responding, and high shock intensities are often assumed to decrease, if not eliminate, responding regardless of other variables such as the shock schedule (Azrin, 1959, 1960; Azrin & Holz, 1961; Azrin et al., 1963; Boe, 1971; Church, Raymond, & Beauchamp, 1967; Camp, Raymond, & Church, 1967; Holz, 1968; Holz et al., 1963; Karsh, 1962). The results of Experiment 2 show that when shock is contingent on short IRTs,

responding can still be maintained at high shock intensities, such as 0.8 mA. What seems important is the manner in which shock-elicited behavior affects contact with the shock and reinforcement schedules. Schedules that deliver shock contingent on short IRTs often arrange avoidance of the shock through low response rates. Responding slowly is more beneficial than not responding because the former still produces food reinforcement whereas the latter does not. Given this, it appears that shock intensity does not always trump other factors of responding. Rather, it interacts with them. High shock intensities

Figure 6. *Experiment 2*: Percent of responses followed by shock for rats exposed to the $IRT < t$ shock schedule. Error bars extend one standard deviation above and below the mean. The reference line indicates the percent of baseline responses that satisfied the shock contingency.

may eliminate responding when shock is contingent on long IRTs but not when it is contingent on short IRTs. Because many of the IRTs generated by schedules of reinforcement are short (Anger, 1956; Arbuckle & Lattal, 1992, Experiment 1; Blough, 1963; Blough & Blough, 1968; Doughty & Richards, 2002; Galbicka & Branch, 1981; Holz & Azrin, 1963; Peele, Casey, & Silberberg, 1984; Shimp, 1968, 1969; Shull, Gaynor, & Grimes, 2001; Sizemore & Maxwell, 1985), shock delivered independently of IRTs is more likely to follow

short IRTs than other IRTs. This may explain why punishment sometimes decreases but does not eliminate responding.

TEMPORAL REACH

In Experiment 2, low shock intensities increased or decreased response rates depending on the IRTs that produced shock whereas high shock intensities decreased response rates regardless of the IRTs that produced shock. One explanation for this is that, at low shock intensities, the punishing effects of

Figure 7. Experiment 2: Proportion of baseline response rates across all shock conditions for rats exposed to the IRT > t shock schedule. Error bars extend one standard deviation above and below the mean. The dashed lines represent one standard deviation above and below the mean baseline response rate. Note the changes in the scaling of the ordinate for Rat JL19.

Figure 8. Experiment 2: Proportion of baseline response rates across all shock conditions for rats exposed to the IRT < t shock schedule. Error bars extend one standard deviation above and below the mean. The dashed lines represent one standard deviation above and below the mean baseline response rate.

shock were confined to the IRTs preceding shock and increased or decreased response rates accordingly. By contrast, at high shock intensities, shock punished the IRTs preceding shock as well as other classes of IRTs, thereby producing a greater decrease in responding.

The decrease in classes of IRTs that did not produce shock seems noteworthy and may be due to changes in the temporal reach of shock. It is possible that the temporal reach of a punisher depends on its intensity. Raising the shock intensity may also raise the temporal reach of shock and increase the number of IRTs affected by the punishment contingency. According to this account, when low intensities are used, the effects of shock would be localized to the IRTs closest in time to the shock. If long IRTs produce shock, long IRTs would decrease in frequency, and response rates would increase (e.g., Arbuckle & Lattal, 1992; Sizemore & Maxwell, 1985). If short IRTs produce shock, short IRTs would decrease in frequency and response rates would decrease (e.g., Arbuckle & Lattal, 1992). At high shock intensities, the temporal reach of shock may extend to a longer sequence of IRTs, which most likely includes a variety of IRT durations. Under these circumstances, various IRT classes would decrease in frequency, and response rates would decrease.

In the present study, changes in the temporal reach of shock may explain why the effects of shock intensity on IRT distributions were statistically significant when short IRTs produced shock but not when long IRTs produced shock. We can assume that the temporal reach of a given intensity is fixed, regardless of the IRTs that produce shock. If so, then shock would presumably affect the IRTs contiguous with shock along with any other IRTs within the temporal reach. Because short IRTs take up less of the temporal reach, shock contingent on short IRTs may allow more IRTs to be captured by the temporal reach than shock contingent on long IRTs.

Punishment studies involving electric shock provide a unique way of studying temporal reach because of the psychophysics of the punisher. Studies on reinforcement magnitude with non-humans typically involve raising the number of pellets delivered or the duration of access to grain. In these cases, reinforcer delivery is inevitably delayed by virtue of the psychophysical properties of the

stimuli used. By contrast, punisher magnitude is often manipulated by varying shock intensity. The psychophysical properties of electric shock are such that these stimuli are experienced almost immediately, perhaps making the latter paradigm a more appropriate one for examining temporal reach. To evaluate the temporal reach account further, future studies of punishment should examine the effects of IRT shock schedules with and without a delay contingency. Of particular interest is whether similar results are obtained when the punisher is a stimulus other than electric shock.

Alternatively, it is possible that high shock intensities simply have stronger punishing effects than low shock intensities. When shock is contingent on IRTs, the class of IRTs targeted by the shock contingency is punished, and these punishing effects generalize to other classes of IRTs. If so, high shock intensity would not decrease responding by capturing members of more classes of IRTs but would punish the class of IRTs that produced shock more effectively than low intensities would. This punishing effect may generalize to other classes of IRTs.

PROCEDURAL IMPLICATIONS

A notable feature of the obtained results is the degree to which responding quickly recovered during the no-shock baselines, particularly when high intensities were used. Some of the previous studies on response-dependent shock have cited complete or irreversible suppression as disadvantages of studying punishment in a steady-state design (Appel, 1961; Axelrod, 1990; Azrin, 1959, 1960; Azrin & Holz, 1966; Boe & Church, 1967; Hake et al., 1967). Yet, in the current study, response recovery was fast and reliable. Several procedural details may be responsible for the response recovery observed. First, while shock intensity was high at times, shock duration was only 100 ms, which is relatively brief when rats are used as subjects. Other commonly used durations include 400 ms, 500 ms, and 1000 ms (Boe, 1966; Boroczi et al., 1964; Church et al., 1967; Camp et al., 1966; Seligman & Campbell, 1965). Second, although the shock schedule may have seemed dense, it was relatively lean when compared with previous research. In fact, in early studies on punishment, it was common to deliver

shock on an FR-1 schedule (Azrin, 1959, 1960; Azrin & Holz, 1961; Boe, 1966; Boroczi et al., 1964; Holz et al., 1963; Karsh, 1962; Seligman & Campbell, 1965). In the present study, usually less than 50% of responses were followed by shock, except when 0.8-mA shock was used. Third, exposure to shock was limited to 60 min per session. In previous research, animals were sometimes exposed to high-intensity shock for several sessions or for several hours within a session (e.g., Azrin, 1956; Azrin & Holz, 1966). By imposing a 60 min time limit, the current study reduced the likelihood that decreases in responding were due to satiation to food reinforcement rather than to the shock itself. Additionally, conditions were terminated early in the few cases where responding was suppressed severely, and the number of sessions at a given shock intensity was limited to five. These procedural details may be worth considering when designing future studies on punishment, particularly those employing a steady-state design.

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