

*EFFECTS OF SLEEP DEPRIVATION ON
FREE-OPERANT AVOIDANCE*CRAIG H. KENNEDY, KIM A. MEYER,
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Two studies examined effects of sleep deprivation on free-operant avoidance by rats. In Experiment 1, a 5-s shock–shock (SS) interval and 20-s response–shock (RS) interval produced baseline performances, which were reestablished after each experimental manipulation. Once baselines were established, animals were exposed to 24, 48, or 96 hr of sleep deprivation and equivalent periods of home cage and food restriction as a control condition. Compared to baseline, sleep deprivation increased response rates by increasing the proportion of brief interresponse times (IRTs); response rates changed little in the control conditions. Percentage of shocks avoided did not systematically change across conditions. In Experiment 2, the RS interval was manipulated (10, 20, and 40 s), while the SS interval (5 s) and level of sleep deprivation (48 hr) were held constant. Across RS intervals, sleep deprivation increased response rates via a shift toward brief IRTs. In addition, sleep deprivation increased the percentage of shocks avoided as an inverse function of RS intervals.

Key words: sleep deprivation, avoidance, negative reinforcement, lever press, rats

Avoidance responding is maintained by the postponement or reduction of noxious stimuli (Hineline, 1981; Sidman, 1953a). For example, in free-operant avoidance, a shock may be delivered every 10 s (shock–shock [SS] interval) unless an animal presses a lever; each lever press postpones the stimulus for 30 s (response–shock [RS] interval). If an animal intermittently presses the lever within the RS interval, shock is avoided and responding is negatively reinforced. As long as RS intervals exceed SS intervals, such contingencies typically yield highly stable responding, with response rates increasing as RS and SS intervals become briefer.

Most studies of negatively reinforced responding focus on the effects of environmental contingencies. But avoidance responding can also be affected by physiological changes. For example, avoidance responding tends to be increased by stimulants such as cocaine, *d*-amphetamine, and methylphenidate (Barrett, 1976; Stretch & Skinner, 1967) and decreased by sedatives such as alcohol, chlordiazepoxide, and pentobarbital (Ator,

1979; Barrett, 1976; Barrett, Dworkin, & Zuccarelli, 1977; Waller & Waller, 1962). In addition, physiological manipulations have selective effects on positively versus negatively reinforced responding. For example, chlordiazepoxide increases appetitively reinforced responding but decreases avoidance responding (Ator, 1979).

Although psychoactive compounds have been a primary focus of research, other physiological variables may also be of scientific interest. If the introduction of an event alters an otherwise steady state of responding, studying its functional effects on behavior may provide a more complete account of behavior–environment relations (Sidman, 1960; Thompson & Boren, 1977). One type of physiological change that has received little attention in operant research is sleep deprivation. Our interest in sleep deprivation and operant behavior arose from applied studies that focused on the problem behavior of students with developmental disabilities. Several studies have documented correlations between bouts of sleep deprivation and increases in negatively reinforced problem behavior (Horner, Day, & Day, 1997; Kennedy & Ikonen, 1993; Kennedy & Meyer, 1996; O'Reilly, 1995). It is perhaps relevant that, in laboratory animals, sleep deprivation has been reported to impair acquisition of shuttle avoidance (Joy & Prinz, 1969; Smith, 1996) and increase aggression, sensitivity to stimulation,

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and activity levels (Albert, Cicala, & Siegel, 1970; Boyaner, 1970; Brock, Hamdi, Ross, Payne, & Prasad, 1995; Hicks, Moore, Findley, Hirshfield, & Humphrey, 1978; Hicks, Moore, Hayes, Phillips, & Hawkins, 1979; Ogilvie & Broughton, 1976). To date, however, no laboratory study has directly manipulated sleep deprivation to study its effects on negatively reinforced responding (cf. Ulrich, Brierton, Mabry, & Stachnik, 1965).

The present research was a preliminary examination of the effects of sleep deprivation on avoidance responding. Experiment 1 compared the effects of different levels of sleep deprivation on a constant free-operant avoidance baseline. Experiment 2 examined the effects of a constant level of sleep deprivation on avoidance baselines maintained under different RS intervals.

EXPERIMENT 1

METHOD

Subjects

Subjects were 4 experimentally naive Sprague-Dawley male rats (R1, B2, G2, and R3) individually housed with 24-hr ad libitum access to water and food except during sleep deprivation and control conditions. At the beginning of the experiment, rats were approximately 100 days of age and weighed between 423 g and 455 g. Throughout the experiment, a 12:12 hr light/dark cycle, with lights on beginning at 6:00 a.m., was in effect.

Apparatus

Modular operant conditioning chambers (Coulbourn Instruments E10-TC), 28 cm high, 20 cm long, and 26 cm wide, were used. Each chamber was individually housed in a sound-attenuating container. Chamber walls consisted of two translucent plastic side panels and aluminum rear and instrument panels. On each instrument panel a lever was placed 3.5 cm from the left chamber wall and 7.5 cm above the floor. The lever was 3.3 cm wide, protruded 1.5 cm from the instrument panel, and required 0.25 N of downward force to operate. A 28-V DC light located in the center of the ceiling illuminated the chamber. During experimental sessions, 80 dB white noise was presented by a speaker located at the top of the instrument panel.

Floors consisted of 18 stainless steel rods spaced 1.3 cm apart. A constant-current shock generator and scrambler delivered 1-mA shocks, 0.5 s in duration (Coulbourn Instruments E13-14). All events in the operant chambers were controlled by Coulbourn Instruments L2T2 hardware and software run by a Compaq[®] 386 computer.

Procedure

Avoidance schedule and daily sessions. Sessions, lasting 60 min, were conducted at the same time each day for each rat, 7 days per week (unless a sleep deprivation or control condition specified otherwise). White noise and ceiling illumination marked the start of each session and continued throughout the session. A free-operant avoidance task (Sidman, 1953a, 1953b) was in effect at the beginning of the first session. When a shock occurred, in the absence of an intervening response, every 5 s thereafter another shock was delivered (SS interval) until a lever press occurred. Each subsequent lever press postponed the next shock for 20 s (RS interval). Training continued until there were 10 consecutive sessions in which (a) the rat avoided at least 80% of the programmed shocks per session and (b) response rate trends were stable on visual inspection. This required between 10 and 27 sessions across subjects. Throughout the experiment, data were collected separately for the first 10 min of each session (warm-up period) and the remaining 50 min (experimental period). Only data from the experimental period were used in the analysis. The same avoidance schedule (5-s SS interval; 20-s RS interval) was employed throughout the remainder of the experiment.

Sleep deprivation. Once the initial training criteria were met for a rat, it was initially exposed to one of three sleep deprivation (24, 48, or 96 hr) or one of three control conditions (24, 48, or 96 hr; see below for specifics of control conditions). Following exposure to either a sleep deprivation or control condition, a rat was immediately placed in an operant chamber for a 1-hr avoidance session. When a session was completed, rats were returned to their vivarium cages. Daily avoidance sessions continued for 3 to 5 days, after which rats were exposed to a new sleep deprivation or control condition if avoidance and

response rate were similar to previous baseline levels for two sessions. During each condition, if one member of the pair (see below) met the stability criterion in fewer sessions than the other pair member, the 1st rat was continued on the condition until the 2nd rat also met the criterion. This pattern was repeated until each animal had four exposures to each level of sleep deprivation and three exposures to each control condition.

Sleep deprivation was manipulated using the pedestal-over-water method (Morden, Mitchell, & Dement, 1967). Pairs of rats (R1 and B2; G2 and R3), both having met the response and avoidance criteria stated previously, were placed in sleep deprivation tanks containing two platforms, each measuring 9 cm in diameter and positioned 9 cm apart from each other and the tank walls. Tanks were cylindrical, approximately 1 m high and 0.5 m in diameter. One rat was placed on each platform. The platforms were positioned 1 cm above 15 cm of water in the tank, allowing animals to remain seated on a pedestal or move between pedestals, but not to lie down. Rats remained in this environment for the duration of the sleep deprivation condition, except for daily 1-hr ad libitum access to food and water in individual home cages.

Rats have three stages of sleep, two stages of slow-wave sleep and rapid-eye-movement (REM) sleep (e.g., Portas et al., 1998). The pedestal-over-water technique selectively deprives rats of 90% to 99% of REM sleep but less than 10% of slow-wave sleep (Maloney, Mainville, & Jones, 1999). This occurs because muscle atonicity is produced at the onset of REM sleep, and animals begin to fall into the water and are awakened. REM and total sleep deprivation have produced similar physiological and psychological effects (e.g., Kushida, Bergmann, & Rechtschaffen, 1989). Because REM sleep deprivation is less intrusive and produces effects equivalent to total sleep deprivation in rats, REM sleep deprivation was employed in this experiment.

Control conditions. Sleep deprivation conditions differed from baseline in two ways: (a) There were no daily avoidance sessions and (b) access to food and water was restricted to 1 hr per day. To control for potential effects of the home cage and food and water restriction, these conditions were presented without

sleep deprivation. Control conditions lasted for 24, 48, or 96 hr.

RESULTS

Figure 1 shows the percentage of shocks avoided for each rat across baseline, sleep deprivation, and control conditions. The percentage of avoided shocks was calculated by dividing the number of shocks avoided per session by the total number of possible shocks per session and multiplying by 100%. Baseline sessions are the final two sessions conducted prior to an experimental manipulation. There was no systematic change in shock avoidance as a function of sleep deprivation. Individual-session data indicated little variability across sessions for each animal. It should be noted, however, that all animals avoided most shocks.

Figure 2 shows, for each rat, the mean number of responses per minute across baseline, sleep deprivation, and control conditions. Baseline data are from the final two sessions prior to the introduction of each sleep deprivation or control condition. There were a total of 39 to 41 baseline sessions, along with four replications of each sleep deprivation condition and three replications of each control condition for each animal. Rates of responding increased as a function of sleep deprivation for each rat; response rates usually were unaffected by the control conditions. Figure 3 summarizes response rates for each animal from the data presented in Figure 2. Response rates for 3 animals peaked at 48 hr of sleep deprivation, with response rate for Rat R1 peaking at 96 hr.

In addition to increased rates of responding, sleep deprivation also had variable effects on response rate from one exposure to the next for each animal (see Figure 2). For example, R1 had a mean of 9.1 responses per minute following 24 hr of sleep deprivation, but individual session values ranged from 7.3 to 12.4 responses per minute. Overall, R1's response rate during baseline was 7.6, with a relatively stable pattern across sessions, except for sessions following 48 hr or 96 hr of sleep deprivation. In the session following sleep deprivation, response rates tended to be elevated relative to other baseline sessions (data not shown). In sessions following 24 hr of sleep deprivation, there was an increase in response rate only once (which was similar in

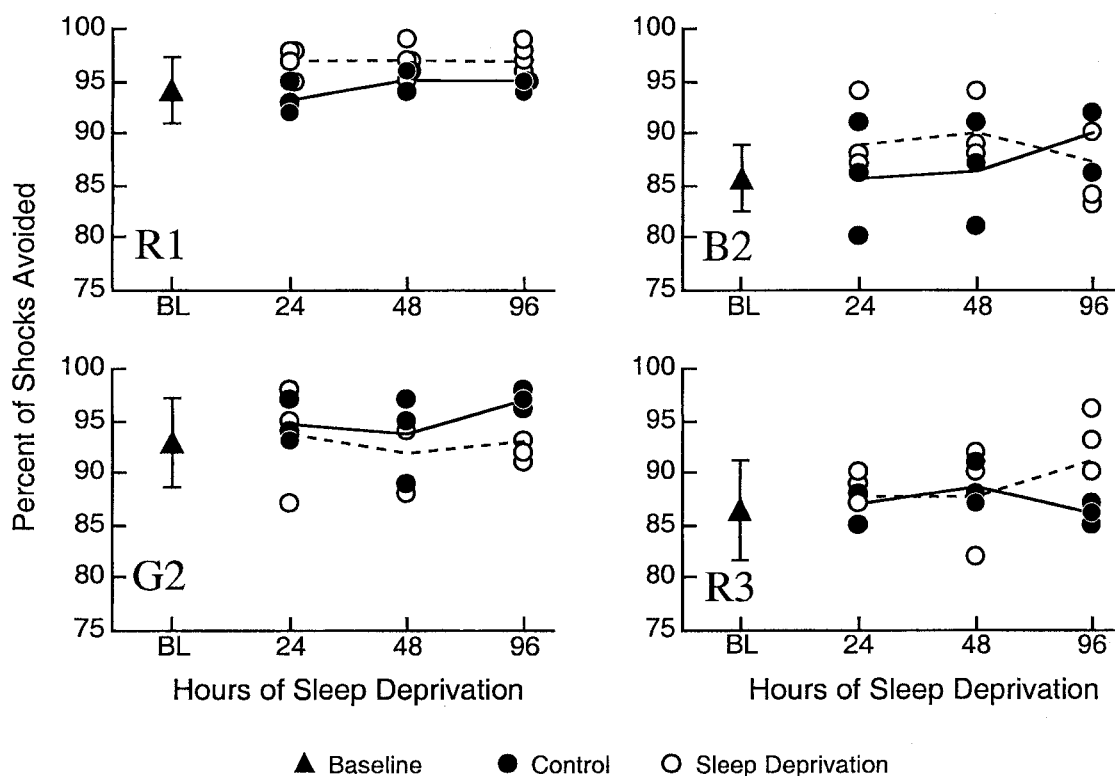


Fig. 1. Percentage of shocks avoided by Rats R1, B2, G2, and R3. Data are summarized for each subject for baseline sessions (closed triangles; bars indicate variation as 1 *SD*). Individual sessions and condition means following sleep deprivation (open circles and broken lines) or control conditions (closed circles and solid lines) are arrayed for 24 hr, 48 hr, and 96 hr.

effect to 48 hr and 96 hr of sleep deprivation), with response rates following 24 hr of sleep deprivation showing marginal differences from baseline. Sleep deprivation for 48 hr and 96 hr for this animal produced variable but elevated response rates. Similar variability was observed for the other subjects' avoidance responding (Figures 2 and 3).

Table 1 presents the interresponse times (IRTs) (in 2-s bins up to 20 s, the RS interval) across baseline, sleep deprivation, and control conditions. IRTs, calculated as the length of time between successive responses, provide an index of the temporal distribution of responding (Anger, 1956). Brief IRTs increased for Rat R1 during sleep deprivation and control conditions, with sleep deprivation effects pronounced at 48 hr and 96 hr. The increase was largest for brief IRTs, but continued through to 10-s IRTs. For Rats G2, R3, and B2, increases in IRT frequency occurred only under sleep deprivation. IRTs of 1 to 6 s in-

creased across most conditions (with the exception of increased 10.1-s to 16-s IRTs for B2 at 96 hr of sleep deprivation). Because sleep deprivation increased brief IRTs, little, if any, improvement in shock avoidance would be expected, particularly when subjects were proficient shock avoiders.

EXPERIMENT 2

We systematically replicated Experiment 1 by assessing the effects of sleep deprivation across differing response rates generated by different RS intervals. We selected the 48-hr level of sleep deprivation because it had the greatest effect on avoidance behavior in the previous experiment. In that the control conditions included in Experiment 1 did not affect responding, they were omitted in Experiment 2.

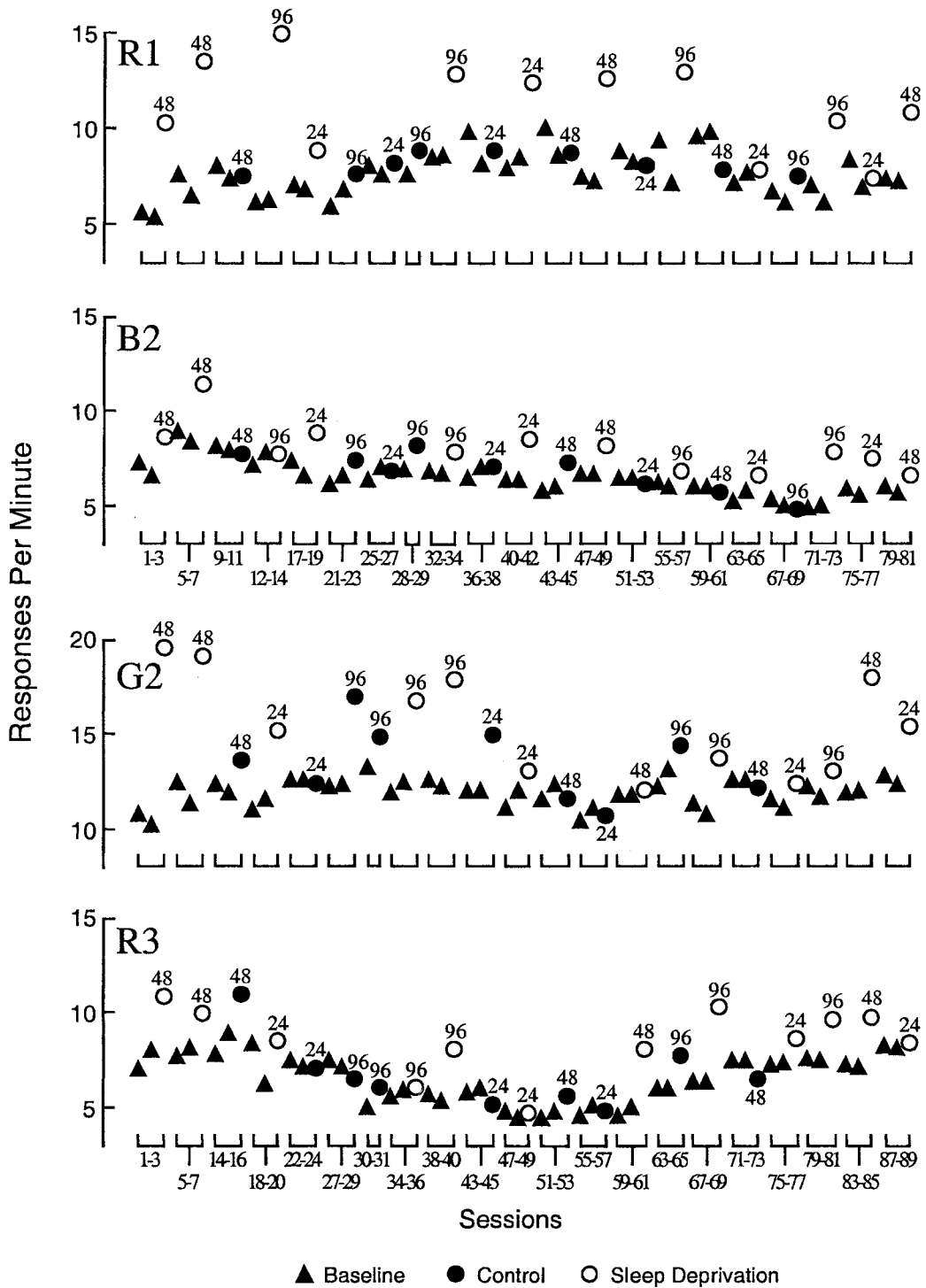


Fig. 2. Session-by-session data for Rats R1, B2, G2, and R3. Data represent the final two baseline sessions prior to a sleep deprivation or control session. Session numbers are the same for Rats R1 and B2 and for Rats G2 and R3. Data are presented as the number of avoidance responses per minute across baseline (triangles), sleep deprivation (open circles), and control conditions (closed circles). Numbers above open circles represent the number of hours of sleep deprivation or control condition.

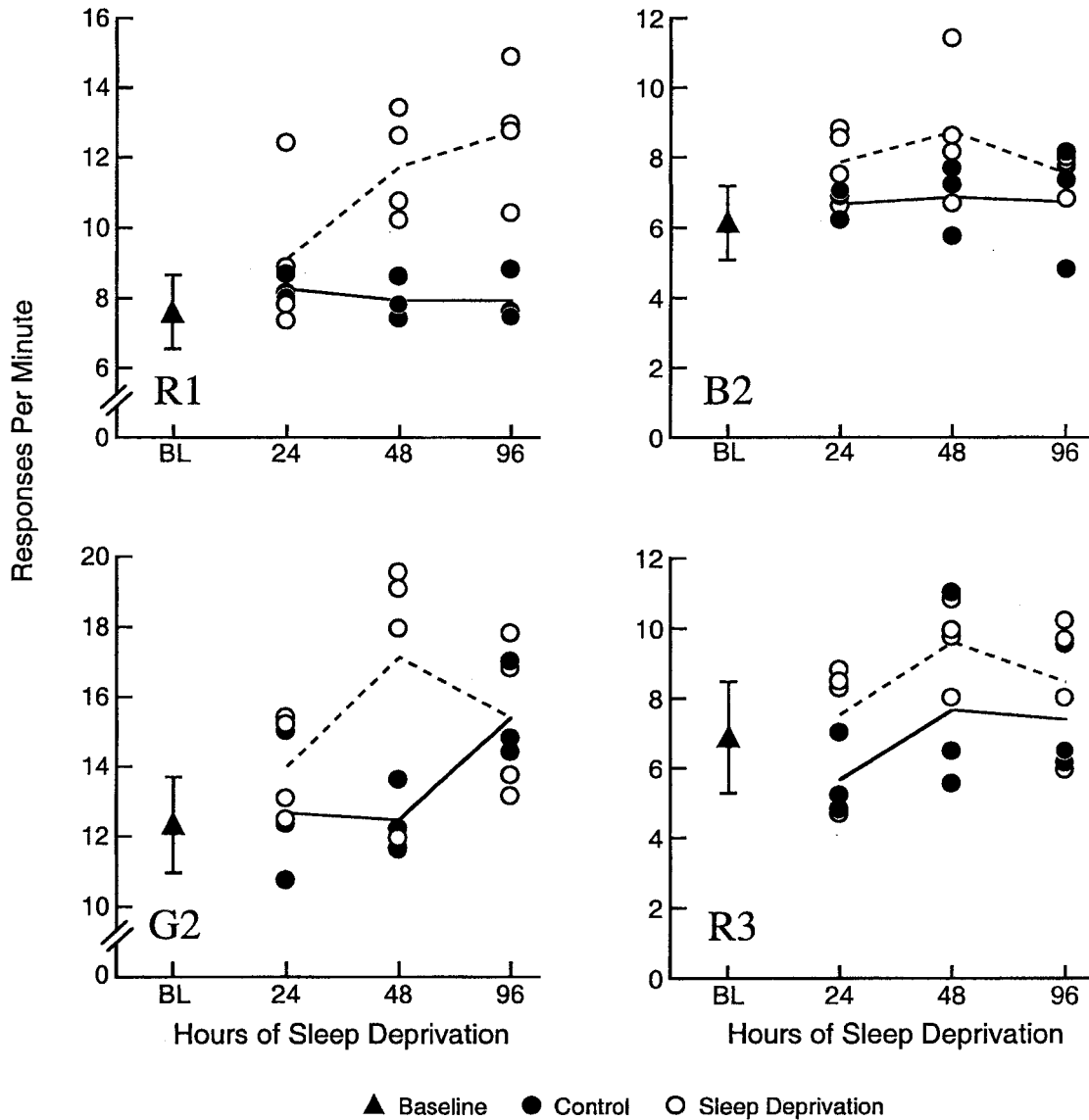


Fig. 3. Response rates for Rats R1, B2, G2, and R3. Data are summarized as the mean responses per minute for each subject for baseline sessions (closed triangles). Individual sessions and condition means following sleep deprivation (open circles and broken lines) or control conditions (closed circles and solid lines) are arrayed for 24 hr, 48 hr, and 96 hr. Bars indicate variation as 1 SD.

METHOD

Procedure

Subjects and Apparatus

Subjects were 4 experimentally naive Sprague-Dawley male rats (R2, R4, P2, and P4). Initially, each rat was between 90 and 140 days of age and weighed between 371 and 471 g. Light/dark cycles, housing conditions, and the apparatus were as in Experiment 1.

The initial training procedures were the same as the previous experiment (the SS was 5 s; the RS was 20 s), with all sessions lasting 60 min and being conducted 7 days per week. Across animals, 12 to 34 daily sessions were required to meet the training criterion. Once training was completed, a subject was ex-

Table 1
Interresponse-time (IRT) distributions across baseline, sleep deprivation, and control conditions. Data indicate the average number of IRTs per session.

Rat	Condition	IRT (s)									
		0-2	2.1-4	4.1-6	6.1-8	8.1-10	10.1-12	12.1-14	14.1-16	16.1-18	18.1-20
R1	Baseline	116	39	24	22	30	31	29	28	25	10
	24 hr SD ^a	165	50	39	37	25	33	32	24	23	6
	24 hr control	168	39	23	21	20	19	23	27	23	7
	48 hr SD	240	57	56	43	42	34	17	19	11	5
	48 hr control	157	31	27	18	19	21	24	32	25	9
	96 hr SD	304	66	56	52	50	39	26	16	10	3
	96 hr control	145	31	25	25	23	24	37	30	19	7
G2	Baseline	325	66	53	31	28	23	24	16	15	7
	24 hr SD	391	80	55	34	27	24	14	18	13	5
	24 hr control	349	71	42	32	29	25	23	24	14	4
	48 hr SD	543	93	53	29	30	17	19	11	9	3
	48 hr control	317	75	52	36	26	22	20	17	9	6
	96 hr SD	455	90	54	41	26	22	16	12	11	4
	96 hr control	401	114	73	46	39	23	24	13	10	5
R3	Baseline	113	27	27	20	27	25	33	20	19	6
	24 hr SD	144	47	31	18	11	12	13	15	19	7
	24 hr control	75	28	26	12	11	10	9	12	20	7
	48 hr SD	210	68	42	23	23	16	16	17	13	5
	48 hr control	144	47	40	15	16	11	11	17	8	4
	96 hr SD	171	58	37	26	15	15	14	12	10	5
	96 hr control	107	40	27	17	12	12	16	19	19	10
B2	Baseline	112	17	19	14	10	13	13	18	24	11
	24 hr SD	182	27	21	13	16	13	12	21	18	8
	24 hr control	125	24	22	12	15	15	14	14	13	10
	48 hr SD	284	43	40	16	20	17	24	22	25	9
	48 hr control	127	27	19	16	15	17	19	17	16	10
	96 hr SD	163	34	25	21	22	37	35	37	20	11
	96 hr control	130	24	25	18	10	6	6	14	21	8

^a SD = sleep deprivation.

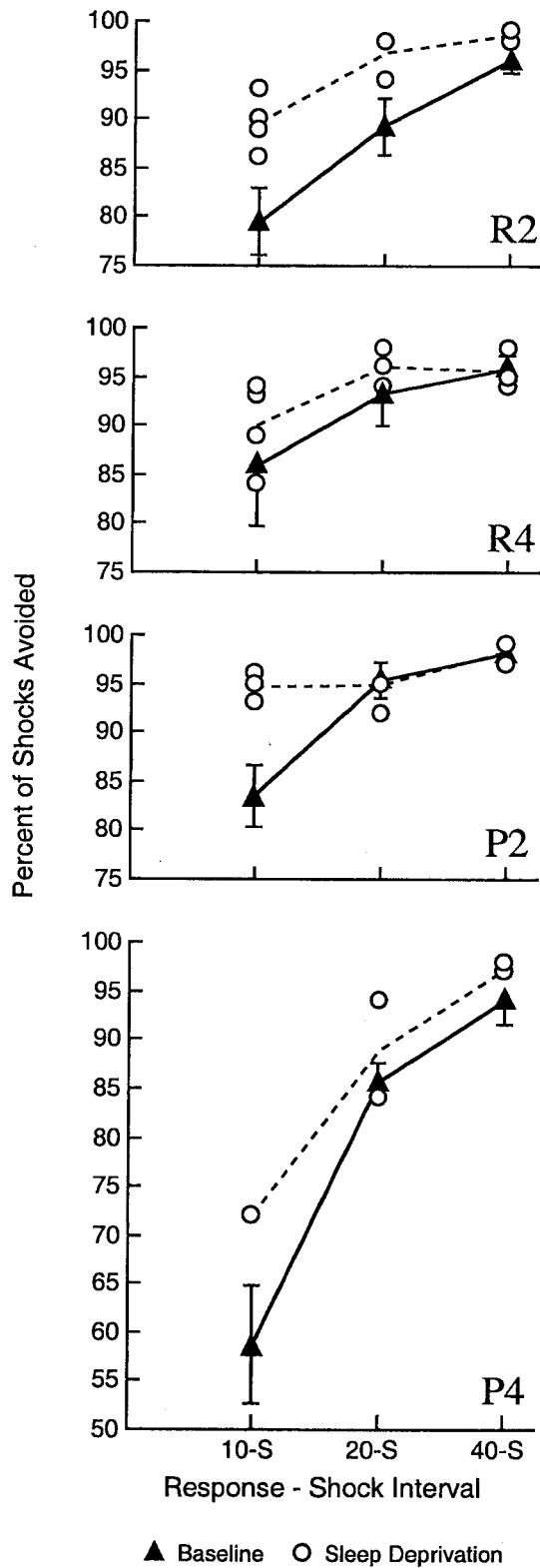
posed to 48 hr of sleep deprivation and then was tested in an avoidance session using the 20-s RS interval. Once the sleep deprivation test was completed, the animal resumed its regular avoidance session schedule the next day; however, the RS interval was changed to 40 s. When 5 days had elapsed with the 40-s RS interval avoidance procedure, a rat was sleep deprived for 48 hr and then was tested in an avoidance session using the 40-s RS interval. Following this sleep deprivation test, the animal was exposed to another RS interval for 5 days and then was tested in a session using that RS interval following 48 hr of sleep deprivation. The sequence of RS interval exposures for each animal was 20 s, 40 s, 10 s, 40 s, 10 s, 20 s, 10 s, 40 s, 20 s, and 10 s. However, due to a computer software failure, the final 10-s and 20-s RS interval conditions were omitted for Rats P2 and P4. The sleep deprivation procedures outlined in Experiment 1 were followed, except that all expo-

sure were 48 hr in length. Rats R2 and R4 and Rats P2 and P4 were paired in tanks during sleep deprivation.

RESULTS

Figure 4 shows the percentage of shocks avoided by each animal across RS intervals for the two baseline sessions preceding sleep deprivation and the sleep deprivation condition. Three subjects (R2, R4, and P2) avoided at least 80% of shocks per session. Relative to baseline, each of these subjects avoided a greater percentage of shocks at RS 10 s under 48 hr of sleep deprivation. There was no systematic difference between baseline and sleep deprivation conditions for Rats R2, R4, and P2 as the RS interval increased. The 4th subject (P4) had a lower percentage of avoidance for RS 10-s intervals, but showed the same general pattern across RS intervals as the other animals.

Response-rate data are presented for each



animal in Figure 5. The data represent the mean number of responses per minute across baseline RS intervals and following 48 hr of sleep deprivation. Baseline data are from the final two sessions prior to each sleep deprivation condition. As in Experiment 1, 48 hr of sleep deprivation increased avoidance response rates. Overall, shorter RS intervals generated higher response rates in baseline and following sleep deprivation. Figure 6 presents these data in chronological order and shows that greater differentiation emerged among RS interval response rates over the course of the study.

The increased response rates associated with sleep deprivation were reflected in increases in brief IRTs across RS intervals and subjects. Table 2 shows baseline and sleep deprivation IRT distributions in 2-s bins from the sessions shown in Figure 5. In general, sleep deprivation increased brief IRTs. For all subjects this increase was particularly pronounced between 1-s and 6-s IRTs. Together, these findings appear to replicate those of Experiment 1 and extend those findings to a range of RS intervals.

GENERAL DISCUSSION

Sleep deprivation had several effects on the free-operant avoidance performances of rats. In both experiments, rates of avoidance responding increased after 48 hr of sleep deprivation. Sleep deprivation also affected the percentage of shocks avoided under certain conditions. In Experiment 1, when RS intervals were held constant, sleep deprivation resulted in no consistent change in shock avoidance across 24, 48, and 96 hr of sleep deprivation. In Experiment 2, when RS values were manipulated, shock avoidance increased when animals were sleep deprived as an inverse function of the RS interval duration.

Experiment 1 demonstrated an increased probability of IRTs ≤ 6 s, particularly ≤ 2 -s

Fig. 4. Percentage of shocks avoided across RS intervals by Rats R2, R4, P2, and P4. Data are arrayed for each subject across 10-, 20-, and 40-s RS intervals during baseline sessions (closed triangles and solid lines). Individual sessions and condition means following sleep deprivation (open circles and broken lines) are presented for 10-, 20-, and 40-s RS intervals. Bars indicate variation as 1 SD.

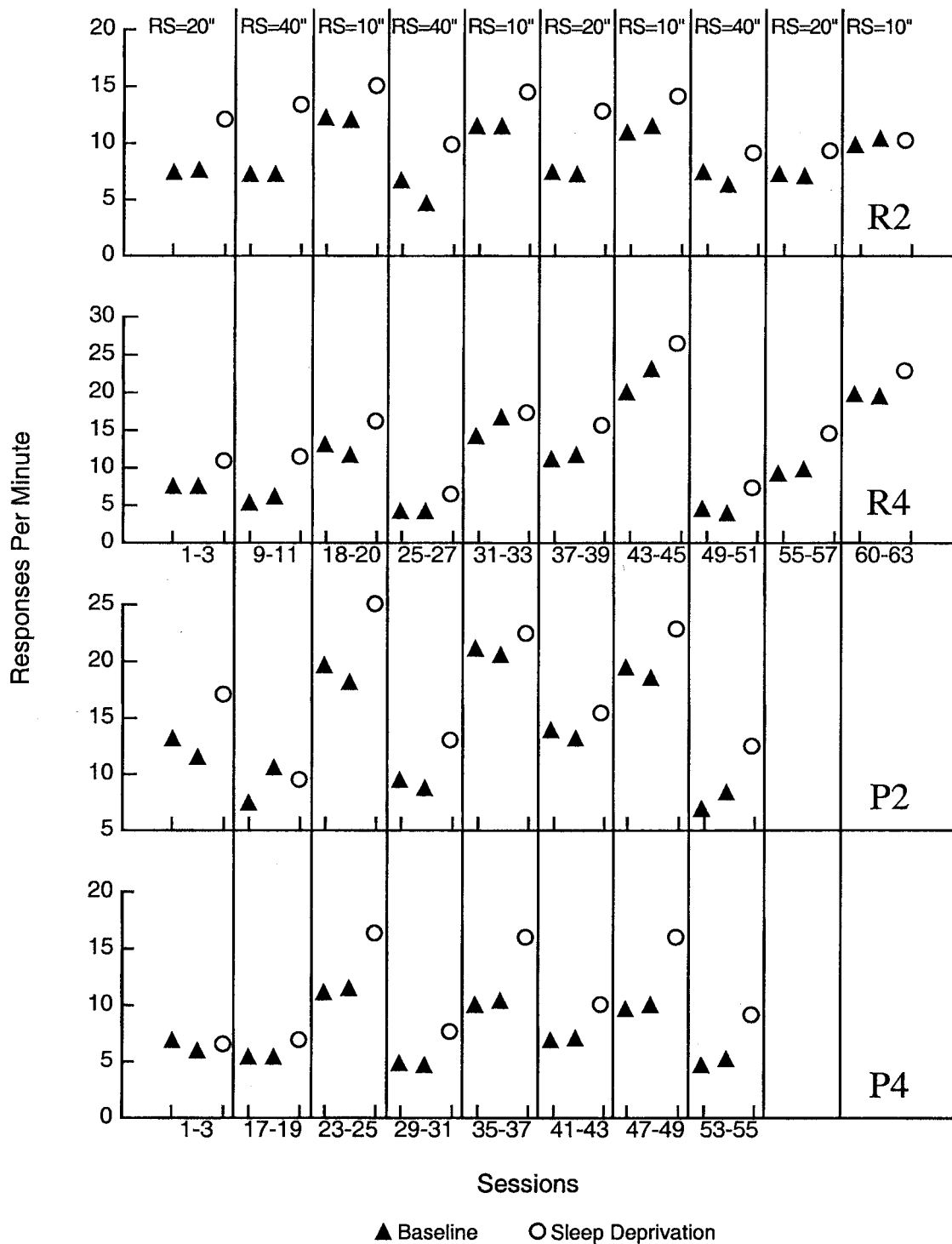


Fig. 5. Session-by-session data for Rats R2, R4, P2, and P4. Data represent the final two baseline sessions prior to sleep deprivation. Session numbers are the same for Rats R2 and R4 and for P2 and P4. Data are presented as the number of avoidance responses per minute across baseline (closed triangles) and 48-hr sleep deprivation conditions (open circles). Values at the top of each panel indicate the RS interval.

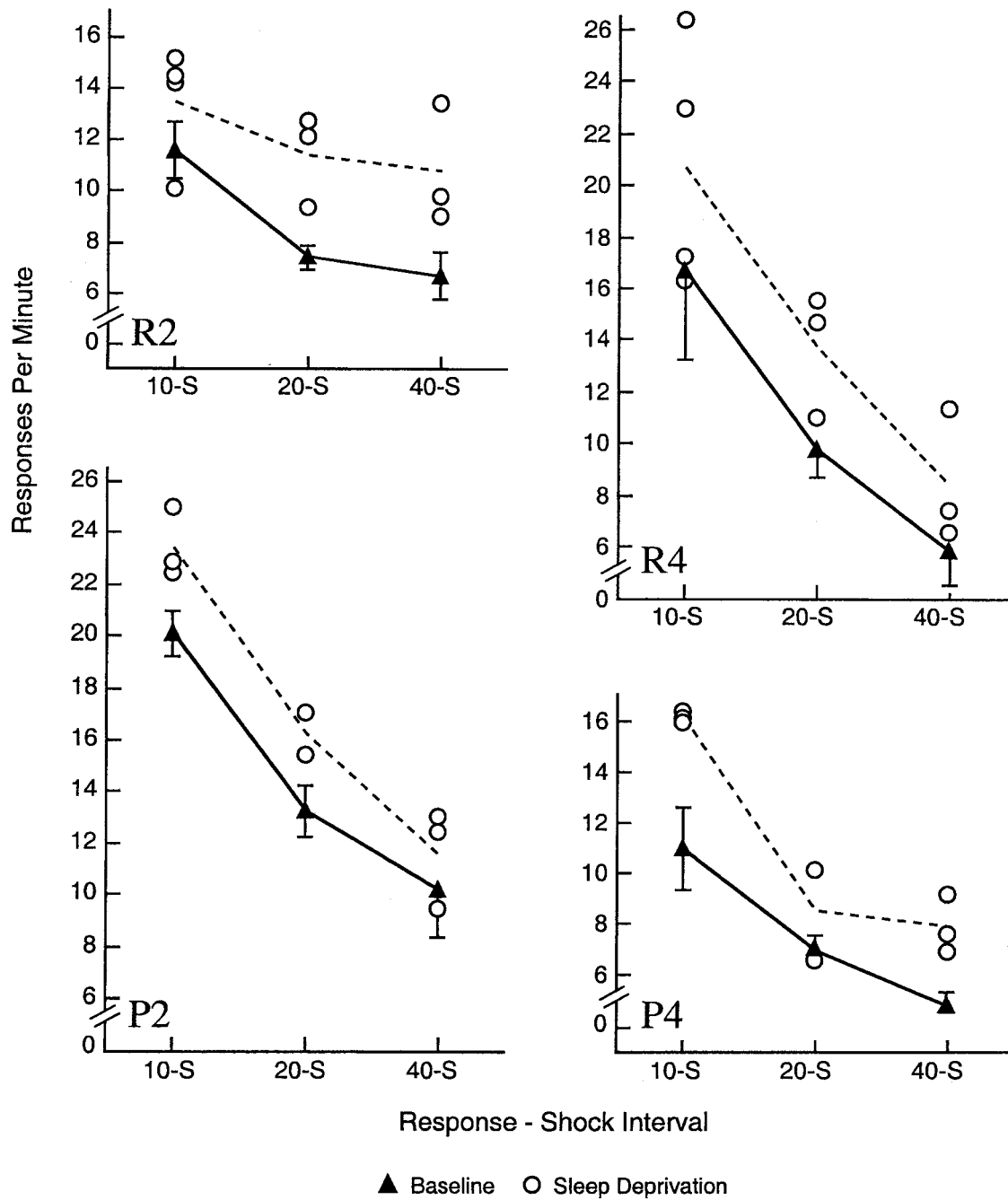


Fig. 6. Response rates for Rats R2, R4, P2, and P4. Data are arrayed as the mean responses per minute for each subject across 10-, 20-, and 40-s RS intervals during baseline sessions (closed triangles and solid lines). Individual sessions and condition means following sleep deprivation (open circles and broken lines) are presented for 10-, 20-, and 40-s RS intervals. Bars indicate variation as 1 SD.

Table 2

Interresponse-time (IRT) distributions across baseline and 10-, 20-, and 40-s response-shock (RS) intervals. Data indicate the average number of IRTs per session.

Rat	Condition	IRT (s)				
		0-2	2.1-4	4.1-6	6.1-8	8.1-10
R2	10-s RS baseline	202	82	89	53	51
	10-s RS SD ^a	217	122	131	102	64
	20-s RS baseline	109	43	44	30	24
	20-s RS SD	206	74	80	53	60
	40-s RS baseline	116	42	39	25	18
R4	40-s RS SD	251	60	42	43	41
	10-s RS baseline	569	151	83	59	32
	10-s RS SD	634	253	152	72	18
	20-s RS baseline	314	119	63	34	32
	20-s RS SD	325	172	107	66	35
P2	40-s RS baseline	201	101	63	36	24
	40-s RS SD	256	87	71	52	26
	10-s RS baseline	359	110	58	74	69
	10-s RS SD	416	108	92	83	58
	20-s RS baseline	181	80	35	13	13
P4	20-s RS SD	342	135	41	26	21
	40-s RS baseline	105	42	17	12	11
	40-s RS SD	212	49	33	20	15
	10-s RS baseline	211	63	32	16	15
	10-s RS SD	403	112	69	33	23
P4	20-s RS baseline	109	38	36	19	14
	20-s RS SD	164	50	32	34	13
	40-s RS baseline	84	27	16	11	10
	40-s RS SD	166	46	38	18	18

^a SD = sleep deprivation.

IRTs. Because Experiment 1 arranged a 20-s RS interval for each lever press, brief bursts of responding would not necessarily increase the overall number of shocks avoided. This observation is supported by the results of Experiment 2, in which RS intervals were systematically manipulated. Sleep deprivation increased response rates over baseline rates at each RS interval, with a corresponding increase in brief IRTs. With longer RS intervals (e.g., 40 s), the increase in brief IRTs did not increase the number of shocks avoided, because changes in the pattern of responding did not contact the avoidance contingency. Moreover, for briefer RS intervals (e.g., 10 s), the brief IRTs were more closely (temporally) related to shock postponement and increased the number of shocks avoided. In addition, the animals were less effective at avoiding shocks under shorter RS intervals, providing greater opportunity for improved performance.

Why does sleep deprivation increase avoidance responding? It is possible that the over-

all rate of an animal's activity is increased, with lever pressing being one of the affected topographies. Previous research on activity levels and sleep deprivation suggests that sleep deprivation increases general activity (Albert et al., 1970; Boyaner, 1970; Brock et al., 1995). This hypothesis could be tested by replicating the current study, or some variant, and measuring activity levels prior to and following daily sessions as well as before and after sleep deprivation.

A second possible explanation for the response-rate increases is that sleep deprivation alters the organism's sensitivity to environmental stimulation. With rats, greater shock intensities result in higher rates of avoidance responding (Weiss & Laties, 1963). Research relating to pain thresholds and sleep deprivation (Hicks et al., 1978) suggests that the more sleep deprived an animal is, the lower its tolerance to noxious stimulation. In the current study it is possible that sleep deprivation lowered an animal's pain threshold and thus increased response rate.

The generality of our findings to other forms of sleep deprivation is unknown. At least two other methods suggest themselves for further analysis. One method is the disk-over-water technique that produces total sleep deprivation (Rechtschaffen, Gilliland, Bergmann, & Winter, 1983) rather than selectively producing REM sleep deprivation. It should be noted, however, that the physiological effects of prolonged total sleep deprivation have not differed qualitatively from prolonged REM sleep deprivation (Kushida et al., 1989), suggesting that a similar process underlies the effects of the two approaches. As an alternative approach, producing intermittent sleep patterns that more closely resemble the types of sleep deprivation observed in humans with sleep disorders may be worthy of study.

A logical extension of the current study would be to analyze the effects of sleep deprivation on both positively and negatively reinforced responding. At issue is whether the effects of sleep deprivation are specific to negative reinforcers, or whether the rate-increasing effect of sleep deprivation also occurs for positively reinforced responding. Such an analysis could shed light on the selective versus general effects of sleep deprivation on activity level previously discussed.

An important step for this line of research would be not only to demonstrate the robustness of sleep deprivation effects on operant behavior but also to delimit the boundary conditions of such effects.

The present findings begin to address a number of questions that have arisen out of applied research for people with developmental disabilities (Carr, Reeve, & Magito-McLaughlin, 1996; Cataldo & Harris, 1982; Kennedy & Meyer, 1998; Thompson & Symons, *in press*). Previous studies have indicated a relation between bouts of sleep deprivation and increases in the rate of problem behavior (Horner *et al.*, 1997; Kennedy & Itkonen, 1993; Kennedy & Meyer, 1996; O'Reilly, 1995). In particular, self-injury and aggression maintained by negative reinforcement increased in frequency following a night of disrupted sleep. Some researchers have cautioned that sleep deprivation is not related to observed changes in responding and have suggested that those changes are only correlational in nature (e.g., Iwata, 1994). The current findings suggest a role for sleep deprivation in increasing rates of negatively reinforced behavior and offer a starting point from which to clarify the findings of previous applied studies. In addition, future analyses suggested by the current experiments may provide an approach for studying interactions between biological events, such as sleep deprivation, and operant behavior.

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