Gaseous Krypton Fluoride

Mass spectrometric analysis (*1*) of krypton fluoride, prepared by J. G. Malm and C. L. Chernick by the electric discharge method with which A. V. Grosse *et al.* (*2*) first prepared KrF_4, yielded Kr^+ and KrF^+ ions as shown in Fig. 1 (*3*).

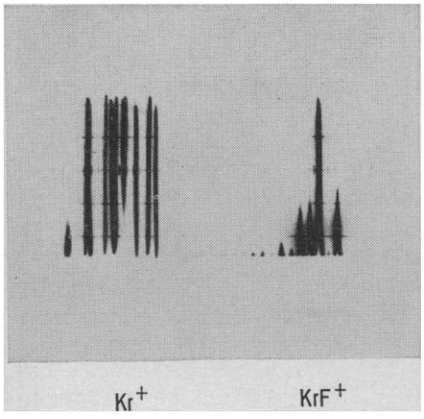

Fig. 1. Mass spectrum of krypton fluoride.

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References and Notes

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3. Based on work performed under the auspices of the U.S. Atomic Energy Commission.

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Punishment and Shock Intensity

Abstract. *The degree of suppression of on-going, food-motivated behavior induced by punishing electric shock was exponentially related to the intensity of the aversive stimulus. No evidence for recovery from these effects during punishment sessions was observed.*

On-going behavior can be suppressed or eliminated for a time by punishment. In pigeons, there is a decline in response rate when electric shock is first introduced after each peck at a plastic disk for food reward, but the amount of this suppression has been reported to be unrelated to the intensity of the punishing stimulus (*1*) if the shock is of sufficient strength to induce any suppression at all. This result has not, however, been generalized to include other species. Moreover, the effects of continued exposure to punishment are not completely understood. The belief has predominated that behavior is suppressed so long as shock is present and that response rates return to preshock levels (recover) only when the punishment is withdrawn (*2*). Azrin, however, reported that recovery from the effects of what he called mild punishment can occur while the shock continues to follow each response (*3*) and that the degree of recovery is a function of the intensity of shock (*1*).

In Azrin's experiments, shock was delivered to the bird's pubis bone through implanted electrodes (*4*). Results with grid shock have recently been reported (*5, 6*) which indicate that brief exposure to punishment might have relatively permanent effects, in that rates do not return to preshock levels even after the punishment contingency is removed. For example, squirrel monkeys were trained (*5*) to press a lever to obtain food pellets on an intermittent schedule of reinforcement. A 0.5-sec, 1-ma shock was then introduced through the lever and grid floor after each response. The experimental sessions lasted for 8 hours. Bar pressing was completely inhibited for 20 days (160 hr) after a maximum of 70 shocks were received; even when the shock was disconnected, no responding occurred in 30 additional 8-hour experimental sessions. Storms *et al.* (*6*) trained hooded rats to work for food and later introduced a 1-ma shock after every response. This procedure again resulted in the complete suppression of food-maintained behavior. The rats were tested for 3 days without punishment after a 2-week rest period and no recovery was observed in three of the four animals.

There appears, in summary, to be some evidence in support of every possible effect of repeatedly exposing animals to punishing shock. Either recovery occurs while the punishment contingency is present (*1*), occurs only after the contingency is withdrawn (*2*), or does not occur at all (*5, 6*). It is likely that the discrepancies in experimental results are a function of intensity parameters, species differences in sensitivity to shock, length of the exposure period, or method of shock administration. The present investigation (*7*) was designed to test the first of these possibilities, that is, to relate both initial suppression (rate during the first day at each intensity) and recovery (rate during subsequent punishment sessions) to shock intensity.

The situation studied is not uncommon in psychological laboratories and resembles that of Appel and of Storms *et al.* A strain of rats (Sprague-Dawley) obtained from the Holtzman Co., a box with a grid floor (R. Gerbrands model C), and a commercial shock generator and scrambler (Grason-Stadler model E1064GS) were used.

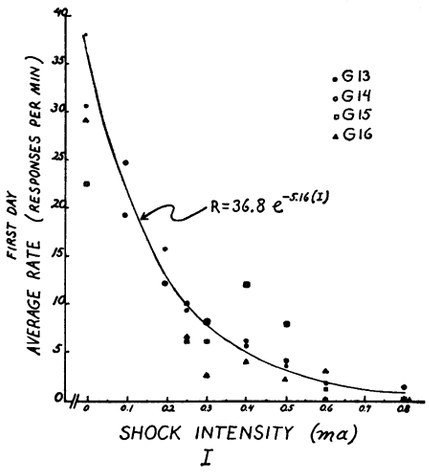

Fig. 1. Average rate of responding for four albino rats (G13, G14, G15, and G16) during the first 90-minute session at each shock intensity. Bar presses were intermittently reinforced with food at 1-minute intervals and regularly punished with shock. The curve was fitted by the method of least squares to the log of the mean rate and is for the equation indicated on the graph.

Four 90-day old male rats were placed on restricted food intake consisting of whatever food they received during the experimental sessions plus sufficient additional Wayne laboratory pellets to maintain them at a constant body weight. They were also trained to press a lever. Every response was initi-

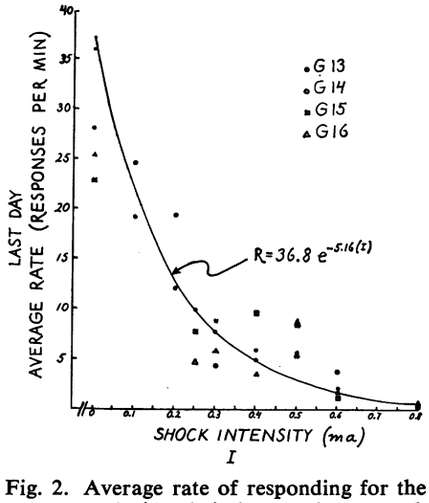

Fig. 2. Average rate of responding for the same rats during their last session at each shock intensity. The curve is the same as in Fig. 1.

ally reinforced with 0.05 ml of a liquid diet of sweetened milk, eggs, and vitamins; later, the schedule was changed so that there was a mean interval between food presentations of 1 min (VI). When rates stabilized on the VI schedule, a 0.5-sec shock delivered through the bar and grid floor was introduced after every response. The intensity of shock was gradually increased for all animals in an ascending order from 0.1 to 0.8 ma in an effort to replicate approximately the procedure of Azrin (*1*) and to avoid the difficulty (if not the impossibility) of reconditioning bar pressing after behavior is completely suppressed by severe punishment (*5, 6*). Each animal was tested at each intensity until its behavior did not change from day to day. This usually required 7 to 10 days. After the last session at a given shock level, there were 5 days under the VI schedule without shock.

At the intensities studied, the rates returned to preshock levels in the absence of the punishment contingency (during VI sessions). Figure 1 shows that the degree of initial suppression, the rate of bar pressing (R) on the 1st day at each intensity (I), is an inverse function of the severity of punishment. This is generally true for each animal as well as for the group data which is not shown. The curve is for the equation $R=36.8\ e^{-5.16(I)}$; it was fitted to the log of the mean rate during the first day of exposure to each intensity by the method of least squares.

Average rates for each animal during its last day at each shock level are plotted in Fig. 2. No animal showed any systematic change in rate from day 1 to any subsequent punishment session at any intensity no matter how long it was exposed to a given shock level (up to 12 days in the present experiment and 15 days in a subsequent study). Therefore, the points in Fig. 1 would be expected to overlap with those of Fig. 2 as indeed they do, and the curve which was fitted to the data of the 1st day may also describe all of the punishment data. Thus, under the conditions of the present experiment, no recovery from shock-induced suppression occurs at any intensity while punishment is being administered and the amount of suppression is an exponential function of the intensity of shock.

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7. Supported by U.S. Public Health Service research grant MY-3363, from the National Institute of Mental Health. I thank Dr. D. X. Freedman for his support, Dr. N. J. Peterson for his advice and assistance, and Miss A. R. West for testing the animals.

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Allergic Encephalomyelitis: Rapid Induction without the Aid of Adjuvants

Abstract. *Rapid production of allergic encephalomyelitis has heretofore required injection of nervous tissue emulsified in immunologic adjuvants. Adjuvants are not required if large doses of a potent nervous-tissue antigen and a highly susceptible strain of rats are used. Susceptibility was increased in animals inoculated beforehand with pertussis vaccine.*

Experimental allergic encephalomyelitis (EAE) was first produced in monkeys by repeated injections of homogenates and extracts of nervous tissue (*1*). The course of treatments lasted many months. One or a few injections of nervous tissue has caused the disease in only an occasional mouse, dog, rabbit, or rat (*2, 3*). With the introduction of Freund's adjuvant, allergic encephalomyelitis could be produced rapidly and regularly after one or a few injections of nervous tissue emulsified in adjuvant (*4*). It is thought that the paraffin-oil emulsion facilitates dispersion of nervous-tissue antigens from the local depot to lymph nodes and other immunologically active sites, and saves the antigen from excessively rapid destruction. The oil and the killed mycobacteria of the adjuvant may promote a cellular environment favorable for sensitization (*5*).

That, in rats, the mycobacterial component of the adjuvant was dispensable was shown when rats developed EAE after a single injection of nervous tissue emulsified in mineral oil without mycobacteria ("incomplete" adjuvant)(*3*). We found this procedure particularly effective in producing EAE in the Charles River CD F (Fischer 344) strain of inbred rats, at least when the antigen was guinea pig spinal cord (*6*). In addition, treatment with pertussis vaccine enhances the susceptibility of mice (*7*) and rats (*8*) to EAE. Based on these considerations, we have developed a procedure for rapid and regular production of experimental allergic encephalomyelitis in rats without the aid of adjuvants.

Female rats, 9 to 11 weeks old, of CD F strain (Charles River Breeding Laboratories) were given free access to Purina Laboratory Chow and tap water. Each received 0.6 or 1.2 ml of pertussis vaccine (Lederle, Phase I, approximately 60 billion organisms per milliliter) diluted to 3.0 ml with saline, intraperitoneally, 4 days before challenge. Previously frozen neural tissues were homogenized with a small amount of distilled water. The volume of each injection was 0.05 ml which contained 40 mg of tissue (wet weight). Injections were made intracutaneously under light ether anesthesia.

A single injection of homogenate of guinea pig spinal cord in the right foot pad was ineffective. However, three, five, or ten injections (120, 200, or 400 mg) in the right foot pad produced the disease in 2 to 3 weeks after the first injection. The incidence and severity were proportional to the dosage (Table 1) but did not depend on whether the injections were given simultaneously on the 1st day or were spread out over a 1- or 2-week period. Omission of prior treatment with pertussis vaccine reduced the effectiveness of injections of nervous tissue. Clinical signs were weakness or paralysis, especially of hind limbs, loss of weight, and occasionally, urinary incontinence. All rats were killed 21 days after the first injection. Histologic examination of spinal cord and brain revealed vascular

Table 1. Production of experimental allergic encephalomyelitis without adjuvants.

Rats (No.)	Dose of cord (mg)*	EAE characteristics	
		Clinical incidence (%)	Histologic severity (0–4)†
	Pertussis treatment		
6	40	0	0
11	120	9	1.0
25	200	24	1.3
40	400	68	3.0
	No treatment		
16	400	31	1.1

* Wet weight of guinea pig spinal cord tissue, as 80 percent aqueous homogenate, divided into 1 to 10 injections all in right foot pad. Different schedules of administration are combined for brevity (injections simultaneous or spread out over 1 to 2 weeks). † Group average, graded individually from 0 to 4 according to number and severity of lesions in spinal cord and hindbrain.