each successive day (Fig. 1). The monkey administered morphine to himself at a low rate both before and after a saline injection. On day 2 an injection of nalorphine (0.1 mg/kg) produced little initial change in self-administration, but after a 20- to 25-minute delay a large increase in responding appeared which continued for 20 to 30 minutes. With repeated presentations of this dose of nalorphine the delay in appearance of increased self-administration diminished. By day 4, the increased rate of self-administration responding appeared within 2 minutes of the nalorphine injection. The other two monkeys showed similar responses. The change in responding observed with repeated administration of nalorphine might have been due to the novelty of the drug effect. We would expect, however, that a novelty effect would have been demonstrated as a decrease in the number of administrations of morphine after repeated injections of nalorphine, rather than an increase. If we assume that the administration of nalorphine to morphine-dependent monkeys produces aversive stimulation which can be reduced by administration of morphine, then the decreased self-administration response latencies after repeated nalorphine injections may reflect the development of conditioned escape or avoidance responding. Nalorphine may not be unique in this regard—rats self-administer certain barbiturates at a higher relative rate shortly after the brief presentation of electric shock (*6*). In our second set of observations, we explored the possibility that previously neutral environmental stimuli can elicit conditioned changes in the pattern of morphine self-administration after repeated withdrawal episodes.

After the initial injections of nalorphine, a form of classical conditioning was begun. A stimulus (flashing red light) was presented once a day at the same time for 10 minutes before and 30 minutes after an intravenous injection of saline or nalorphine. After four pairings of light and saline injection, the light was presented once a day in association with an intravenous injection of 0.1 mg of nalorphine per kilogram of body weight. The light and the stimulus associated with the injection procedure might thus be viewed as conditioned stimuli (CS) and the nalorphine injection as an unconditioned stimulus (US). After ten pairings of light and nalorphine injection, a control trial was conducted by omitting the light-injection pairing. The control trial was followed by five daily test trials with light–saline injection pairings.

No change in the number of administrations of morphine was produced by intravenous saline injections during the initial trials (days 1 to 4) (Fig. 2). During conditioning trials (days 5 to 14), intravenous injections of nalorphine (0.1 mg/kg) increased the frequency of administration of morphine in the 30-minute period following the injection. After the tenth conditioning trial (day 14), a control trial, without a light-injection pairing was conducted and the rate of self-administration was similar to that of days 1 to 4. Thus, conditioning had not altered the base-line performance of the monkeys. The first test (pairing of light and saline injection, day 16), after the tenth conditioning trial, resulted in large increases in the number of self-administrations of morphine during the 30 minutes following the saline injection. The self-administration rate of the three monkeys after the injection was three to five times greater than that seen after the initial light–saline injection trials (days 1 to 4). With repeated pairings of light and saline injection (days 16 to 20), this conditioned response rapidly disappeared. Reconditioning training was then conducted (days 21 to 30) and results closely paralleled those in the initial conditioning sessions. On the first day of the subsequent test (days 32 to 34) the animals showed a large increase in the number of self-administrations of morphine.

We noted (*2*) that pairing of a red light CS with a nalorphine US suppressed food-reinforced lever pressing during the interval between CS onset and US onset. No change in self-administration was seen in the present study, however, during the 10-minute interval between CS onset and injections of saline or nalorphine.

That the monkeys increased their responding to saline injections although they did not increase their responding to the light CS preceding the injections indicates that the stimuli associated with injections had acquired the property of increasing self-administration of morphine. A stimulus complex consisting of pairing of light and saline injection acquires conditioned reinforcing properties after a number of response-contingent pairings of light and morphine injection. During extinction conditions, response-contingent presentations of this stimulus complex produces large, but transitory, increases in response rate previously reinforced with morphine (*7*). Thus, stimuli associated with either the nalorphine-induced withdrawal syndrome or with morphine reinforcement can acquire conditioned properties which result in their playing an important role in the control of self-administration of drugs.

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References and Notes

1. S. R. Goldberg and C. R. Schuster, *J. Exp. Anal. Behav.* **10**, 235 (1967); S. Irwin and M. H. Seevers, *J. Pharm Exp. Therap.* **116**, 31 (1956).
2. A. Wikler, *Brit. J. Addict.* **57**, 73 (1961); ———, in *Narcotics*, D. M. Wilner and G. G. Kassenbaum, Eds. (McGraw-Hill, New York, 1965).
3. ——— and F. T. Pescor, *Psychopharmacologia* **10**, 225 (1967).
4. For further details of intravenous narcotic self-administration procedures in primates, see G. A. Deneau, T. Yanagita, M. H. Seevers, *Psychopharmacologia*, in press; C. R. Schuster, in *Use of Nonhuman Primates in Drug Evaluation*, H. Vagtborg, Ed. (Univ. of Texas Press, Austin, 1968), p. 283.
5. G. A. Deneau and M. H. Seevers, *Advan. Pharmacol.* **3**, 267 (1964).
6. J. D. Davis, G. C. Lulenski, N. E. Miller, *Int. J. Addict.* **3**, 207 (1968).
7. C. R. Schuster and J. H. Woods, *ibid.*, p. 223.
8. This report is based on a dissertation submitted to the Graduate School of the University of Michigan by S.R.G. in partial fulfillment of the requirements for the Ph.D. degree. Supported by NIMH grant 5R-10MH-12084; M. H. Seevers, principal investigator.
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Olfactory Stimuli and the "Pseudo-Extinction" Effect

Abstract. *Continuously rewarded rats show a decrease in running speed on a runway recently traversed by other rats undergoing experimental extinction. This "pseudo-extinction" effect is caused by discriminable odors emitted by extinction subjects. These odors could be confounding variables in studies using forms of aversive stimulation.*

The influence of olfactory stimuli on the albino rat in a variety of situations has been studied. The results are inconsistent with several experiments demonstrating patterned responding within differential conditioning, single and double alternation, and straight runway situations (*1*). The hypothesis has been advanced that discriminable odors elicited by certain specifiable conditions rather

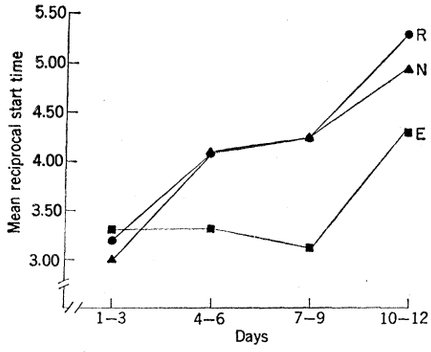

Fig. 1. Mean starting speed for recipient paper subjects over 12 days of testing. Trials were conducted on new paper (*N*), extinction-traversed paper (*E*), and reward-traversed paper (*R*).

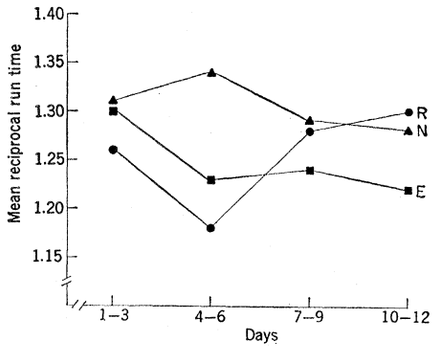

Fig. 2. Mean running speed for recipient paper subjects over 12 days of testing. Trials were conducted on new paper (*N*), extinction-traversed paper (*E*), and reward-traversed paper (*R*).

than learning control the rat's behavior in many experimental settings. These odors apparently modify, in an unconditioned manner, the instrumental performance of another animal.

We studied this "odor" hypothesis in an attempt to account for an as yet unexplained experimental result, the "pseudo-extinction" effect (*2*). This effect is a decrease in running speed of continuously food-rewarded control subjects tested after extinction trials of another experimental group in the same apparatus. Runway performance was observed in a group of continuously reinforced subjects under three conditions: trials in which the apparatus floor was clean paper, trials in which the paper floor was recently traversed by a single reinforced subject, and trials in which the paper floor was recently traversed by a single subject undergoing experimental extinction. If the paper floor is a prime receptacle of odorous chemical substances emitted by the rat, if rats undergoing experimental extinction emit a discriminable odor capable of modifying another rat's runway performance, and if no such odor is released during reward training, then trials run on extinction paper should be slower than either new or reward paper trials, and the latter two should not differ.

The subjects were 20 male albino rats from 100 to 120 days old. A straight, uncovered runway (1.2 m long, 46 cm high, and 9 cm wide) was used to minimize the concentration of odors within the apparatus. The entire apparatus was hinged along one side so that it could be tilted to permit replacement of the paper floor (adding machine paper). New paper was kept on a roll outside the apparatus at the start box end. The paper floor was changed by pulling the paper through the length of the apparatus and out the goal box end. No other controls for odor traces, such as cleansing the apparatus walls or spraying with disinfectant, were employed. A 97-mg reinforcement pellet was placed in a clear plastic coaster, which rested directly on the paper floor in the goal box. Start and running times were measured by two standard electric timers. They were controlled by a microswitch operated by the starting gate and by two photoelectric units, one located 4 cm beyond the 30-cm-long start box and the other 4 cm before the 30-cm-long goal box.

One week before preliminary training, subjects were handled, tamed, and placed on a food deprivation schedule (22 hours) maintained throughout the experiment. Subjects were given 12 g of Purina Lab Chow daily. On each of the 3 days before preliminary training, randomly selected groups of four subjects were allowed 10 minutes to explore the apparatus; new paper floors were provided for each group of animals. As preliminary training, all subjects were run individually and given a food reward; four trials were conducted daily on five consecutive days. The rat was removed from its home cage and placed in the start box facing away from the starting gate; after 10 seconds the gate was opened. When the subject entered the goal box the goal gate was closed; the subject was confined until it had consumed the reward pellet, and then was removed and returned to its home cage. The paper floor was then removed, and new paper inserted for the next animal.

For experimental training, subjects were divided into four groups of five subjects prior to the first day of experimental training. The ten animals whose last 2 days' performance was most stable were assigned to two experimental squads, on the supposition that the animals whose performance was least variable would provide a good base line by which to assess the effects of odor traces. Of the ten remaining animals, the five fastest and five other subjects were assigned to extinction and continued-reinforcement conditions, respectively. It was thought that those running fastest would experience the greatest frustration to experimental extinction, and hence maximize the emission of any differential odors. For identification purposes these last two groups were called extinction trace (ET) and reward trace (RT) subjects. Their sole function was to lay an odor trail on the paper floor which an experimental animal would traverse 1 minute later. In addition to trials in which new paper served as the apparatus floor (N), experimental animals received extinction-traversed paper (E) and reward-traversed paper (R) to run on. On a given test day, after a warm-up trial on new paper, each experimental subject received three trials, one each of N, R, and E. It should be noted that experimental subjects were reinforced on all trials. The trials differed solely on the basis of the previous history of the paper floor. Order of the three kinds of trials (N, R, and E) was counterbalanced within a 3-day block of trials. Testing continued for a total of 12 days (four 3-day blocks of trials).

All RT animals received two daily rewarded trials as in preliminary training. All ET subjects received two daily trials, but pellets were no longer presented in the food cup. For ET subjects, goal box entry resulted in 30-second confinement, and failure to enter the goal box led to 120-second confinement in the remainder of the apparatus. Fecal boluses were removed and urine was blotted by the experimenter with a paper towel. It was observed that no RT subjects urinated or defecated during the testing period. All ET subjects urinated.

Start and running times were converted to reciprocals and are referred to as starting and running speeds, respectively. Figure 1 shows the starting speed results. While nondifferential performance was evidenced during the first quarter of testing, subsequent starting speeds were slower on extinction paper than were trials on new or reward paper. This decrease in performance on extinction-trace paper maintained itself from day 4 until the end of the test-

ing period. Analysis of variance yielded significant main effects of treatment conditions ($F = 7.58$, d.f. 2/18, $P < .01$) and blocks of days ($F = 5.06$, d.f. 3/27, $P < .01$). As the interaction was insignificant ($F < 1.0$ overall treatment means were compared by the Newman-Keuls procedure (*3*). The E trials were significantly slower ($P < .05$) than N or R trials. Figure 2 shows the running speeds were slower on extinction as were again slower than trials on new paper after the first three test days. Trials on reward paper displayed a more aberrant trend measured by running speed than by starting speed. The R trials were slower than E and N trials over the first half of testing. However, from day 7 until the end of the testing period, performance on reward paper was coincident with trials on new paper; both were faster than trials on extinction paper. Analysis of variance yielded only a significant main effect of treatment conditions ($F = 3.66$, d.f. 2/18, $P < .05$). Again the interaction was insignificant ($F = 1.74$, d.f. 6/54, $P > .10$) and comparison of the overall treatment means was made by the Newman-Keuls procedure. The E trials were significantly slower ($P < .05$ than N, but not R trials.

These results indicate that the odor trace of a rat undergoing experimental extinction can significantly disrupt the performance of a subsequently run animal that was continuously reinforced. This disruption has previously been termed the "pseudo-extinction" effect and was evidenced as slower starting speeds on E as compared to N and R trials and slower running speeds on E as compared to N trials. This suggests that the mere traversal of another subject is not sufficient to disrupt the succeeding animal's performance. Rather, the state of the animal laying the trace seems to be critical in the elicitation of competing behaviors within the experimental animals. The pattern of results evidenced by the two dependent variables was different. There is the possibility that the repeated testing procedure had differential effects on running than on starting times, this influencing the time course of the observed effects.

Our experiment does not discriminate between qualitative and quantitative odor effects since experimentally extinguished animals were on the paper floor longer than rewarded animals. Nor does it identify the olfactory stimuli involved, particularly whether these olfactory stimuli are isolable from those of the excretory products deposited by the ET animals. The experiment does, nonetheless, demonstrate the importance of olfactory stimuli to the "pseudo-extinction" effect.

Rats can discriminate odors from animals of the same species put under stress by electric shock (*4*). Experimental extinction is apparently a situation capable of producing the emission of some olfactory stimulus which, when present on the paper floor of a subsequently run animal, elicits some behavior which interferes with running for food reward. Such odor effects appear to be an important, potential confounding variable in studies where learning rather than the transmission of information between conspecifics by chemical means is investigated (*5*). Results from situations involving noxious stimulation, such as electric shock or nonreward, which seem likely to increase the probability of odor emission, should be reevaluated because of such confounding. Control for odor effects would seem desirable if interpretation of experimental outcomes is to be unambiguous.

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References and Notes

1. J. H. McHose and H. W. Ludvigson, *Psychonom. Sci.* **6**, 485 (1966); J. H. McHose, *ibid.* **9**, 281 (1967); H. W. Ludvigson and D. Sytsma, *ibid.*, p. 283; J. H. McHose, L. L. Jacoby, P. A. Meyer, *ibid.*, p. 401.
2. D. W. Lauer and T. S. Carterette, *J. Comp. Physiol. Psychol.* **50**, 334 (1957).
3. B. J. Winer, *Statistical Principles in Experimental Design* (McGraw-Hill, New York, 1962), pp. 80–85.
4. J. G. Valenta and M. K. Rigby, *Science* **161**, 599 (1968).
5. Although technical difficulties have precluded the use of and reference to the term "pheromone," the general similarity of the odor effects in the present experiment to pheromones should be noted. For a discussion of pheromone effects in vertebrates, see K. K. Gleason and J. H. Reynierse [*Psychol. Bull.* **71**, 58 (1969)].

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Occurrences of $CaCO_3 \cdot H_2O$ and Its Naming

In the report by Marschner (*1*) of the formation of the compound $CaCO_3 \cdot H_2O$ ("hydrocalcite") in scales deposited from cold waters, the statement that "it has hitherto not been observed in nature" is incorrect; the compound was first observed in 1959 in bottom sediments from Lake Issyk-Kul, Kirgizia, by Sapozhnikov and Tsvetkov (*2*), whose analysis gave $CaCO_3 \cdot 0.65H_2O$. In 1964, Semenov (*3*) showed that the optical and x-ray data for the material corresponded to those for the well-known synthetic compound, hexagonal $CaCO_3 \cdot H_2O$. The x-ray powder diffraction data differ slightly in spacings and intensities from those of Marschner but undoubtedly refer to the same compound.

A second occurrence of $CaCO_3 \cdot H_2O$ was reported in 1963 by Carlström (*4*), who found it in trigonal crystals ($a = 6.100$ Å, $c = 7.553$ Å) among the statoconia of the tiger shark *Galeocerdo cuvier*.

Semenov (*3*) named the material monohydrocalcite, and this name has priority over Marschner's "hydrocalcite." The latter name is doubly unacceptable, because it had already been used by Kosman in 1892 to designate material that was perhaps $CaCO_3 \cdot 2H_2O$ or $CaCO_3 \cdot 3H_2O$ (*5*). This is an excellent example of unnecessary confusion in the mineralogical nomenclature that could easily have been avoided if the proposed new name had been referred to the Commission on New Minerals and Mineral Names, International Mineralogical Association.

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References and Notes

1. H. Marschner, *Science* **165**, 1119 (1969).
2. D. G. Sapozhnikov and A. I. Tsvetkov, *Dokl. Akad. Nauk SSSR* **124**, 402 (1959).
3. E. I. Semenov, *Kristallografiya* **9**, 109 (1964); abstract in *Amer. Mineral.* **49**, 1151 (1964).
4. D. Carlström, *Biol. Bull.* **125**, 441 (1963).
5. See E. S. Dana, *System of Mineralogy* (Wiley, New York, ed. 7, 1951), vol. 2, p. 227.
6. Publication authorized by the director, U.S. Geological Survey.

6 October 1969

Hard Clam Pumping Rates: Energy Requirement

The paper by Hamwi and Haskin (*1*) on oxygen consumption and pumping rates in *Mercenaria mercenaria* seems to draw a conclusion not warranted by the data they presented. I have reproduced their Fig. 2, from which they conclude that pumping rate may be regulated by oxygen require-