differential quantity of reward on acquisition and performance of maze habit. Journal of Comparative & Physiological Psychology, 1954, 47, 44-46.
MARX, M. H., & BROWNSTEIN, A. J. Effects of incentive magnitude on running speeds without competing responses in acquisition and extinction. Journal of Experimental Psychology, 1963, 65, 182-189.
NEURINGER, A. J. Effects of reinforcement magnitude on choice and rate of responding. Journal of the Experimental Analysis of Behavior, 1967, 10, 417-424.
PEREBOOM, A. C., & CRAWFORD, B. M. Instrumental and competing bheavior as a function of trials and reward magnitude. Journal of Experimental Psychology, 1958, 56, 82-85.
PUBOLS, B. H., JR. Incentive magnitude, learning, and performance in animals. Psychological Bulletin, 1960, 57, 89-115.
ROSEN, A. J. Incentive-shift performance as a function of magnitude and number of rewards. Journal of Comparative & Physiological Psychology, 1966, 62, 487-490.
SCHRIER, A. M. Effects of an upward shift in amount of reinforcer on runway performance of rats. Journal of Comparative & Physiological Psychology, 1967, 64, 490-492.
SPENCE, K. W. *Behavior theory and conditioning.* New Haven: Yale University press, 1956.
VOGEL, J., MIKULKA, P., & SPEAR, N. E. Effect of interpolated extinction and level of training on the "depression effect." Journal of Experimental Psychology, 1967, 72, 51-60.
WAGNER, A. R. Effects of amount and percentage of reinforcement and number of acquisition trials on conditioning and extinction. Journal of Experimental Psychology, 1961, 62, 234-242.
ZEAMAN, D. Response latency as a function of the amount of reinforcement. Journal of Experimental Psychology, 1949, 39, 466-483.

Attenuation of stimulus sensitivity by scopolamine

KENNETH BROWN and DAVID M. WARBURTON*
Queen's University, Belfast, Northern Ireland

Five male albino rats were trained on a DRL 15-sec schedule of reinforcement and injected with six doses of scopolamine hydrobromide. Analysis of the results by means of signal-detection theory indicated that the effects were due to modifications of stimulus sensitivity rather than changes in response bias. This was interpreted as evidence against a response-inhibition hypothesis and suggests that there were changes in the stimulus input.

A number of studies have shown that scopolamine hydrobromide, a peripheral cholinergic blocking agent (Goodman & Gilman, 1965) increased responding in situations requiring response suppression (Heise, Laughlin, & Keller, 1970; Warburton & Groves, 1969). Other studies (Warburton, 1969) have demonstrated similar behavioral effects when cholinergic function was modified by anticholinesterases. This disruption has been attributed to either impairment of stimulus control (e.g., Heise et al, 1970) or to response disinhibition (Bignami, 1967). These hypotheses were tested by using a form of analysis based on the theory of signal detection (TSD). This theory originated in work on electronic communication systems and has been extended to sensory psychology (Green & Swets, 1966). It has been particularly useful in psychophysics because it yields independent estimates of a S's sensitivity to stimuli, d', and his response bias, β.

The present experiment applied the TSD analysis to performance in a situation involving response suppression, the differential reinforcement of low rate (DRL). In the DRL schedule a hungry animal is reinforced with food only if his responses are spaced at greater than a specific interval, while responses within this time limit reset the interval (Ferster & Skinner, 1957). The nature of the discriminative stimulus controlling the spaced responding is unknown, but the TSD analysis still enabled the relative changes in sensitivity and bias to be examined.

METHOD

The Ss were five adult male albino rats (Wistar strain), housed individually and maintained at 80% of their "free-feeding" weight.

They were trained to leverpress in a Grason-Stadler (Type E 3125 B) operant box using 45-mg Noyes pellets of food. Thereafter, they were reinforced for spacing their responses at intervals increasing progressively from 5 to 15 sec over a period of 6 days. Supplements of 15 g of dry laboratory diet were given after each daily session. They were maintained on the DRL 15 sec for 50 1-h sessions until the behavior was stable. Prolonged training was needed to satisfy the TSD assumption of well-practiced Ss, i.e., Ss that have an ordinal scale of likelihood ratio that a particular event was caused by a signal rather than noise (Green & Swets, 1966).

After stable performance was established, doses of 0, 0.0625, 0.125, 0.25, 0.5, and 0.75 mg/kg of scopolamine hydrobromide were injected intraperitoneally on alternate days, according to a Latin square design, providing that performance returned to the baseline level on the intervening day. Previous studies had shown that this interval between injections was sufficient (Warburton & Groves, 1969), and our data confirmed this.

RESULTS

The experimental results were recorded in terms of the number of responses, the number of reinforcements, and the interresponse times (IRTs), using 10 3-sec intervals. From the IRT data, estimates were made of the probability of responding in a particular time interval, on the condition that the animal reached the initial boundary of that interval and so had an opportunity for responding during the interval, the IRTs/OPs analysis used by Anger (1963). From these values, the mean probability of a response was calculated for each animal over four intervals of 3 sec, prior to the availability of reinforcement, giving the "false-alarm" rate, p(S/n). The results for the first 3-sec interval were not used in this calculation because they represented deprivation conditions, apparatus, etc. (Harzem, 1969). The mean probability of a response for the five intervals between 16 and 30 sec was computed to give the "hit" rate, p(S/s). It was possible to average these values because of the relative stability of the IRTs/OPs measure within both the pre- and postreinforcement intervals. The false-alarm rates and hit rates of each animal for each dose level were plotted on double probability graph paper, and an index of sensitivity, d'_e, was estimated for each one by the procedure suggested by J. P. Egan and described in Green & Swets (1966). The individual changes in sensitivity for each dose are plotted in Fig. 1.

These dose-response functions show that the sensitivity to the signal decreased inversely with dose in all animals. It should be emphasized that the computation assumes that the underlying noise and signal + noise distributions are both Gaussian and equal, but, even if they were not, the relationships between d'_e and dose level would be unchanged.

A discriminative index of the hit rate over the false-alarm rate was calculated for each dose level for each S. There was no overlap between the indices for the 0- and 0.0625-mg/kg dose levels, and no overlap between these two and the other four dose

*Present address: Department of Psychology, Reading University, Reading, England.

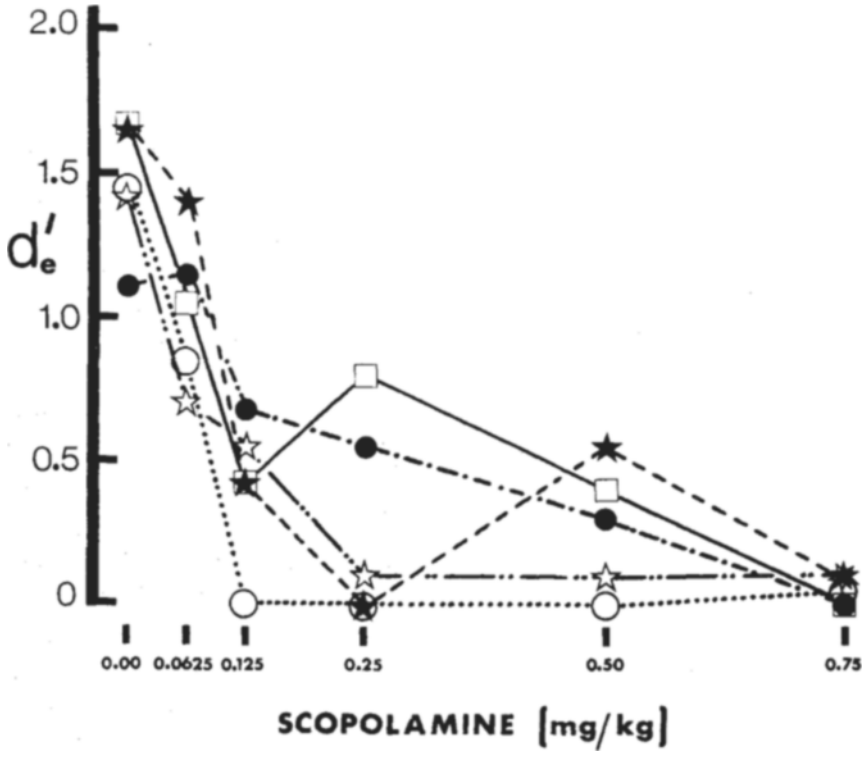

Fig. 1. Individual changes in stimulus sensitivity, d'_e, with six doses of scopolamine.

levels. A Wilcoxon matched-pairs signed-ranks test (Siegel, 1956) shows that there were significant differences ($p < .05$) between the 0- and 0.0625-mg/kg dose levels and all the other dose levels, and between the 0- and 0.0625-mg/kg dose levels. The present authors are not aware of any study that has used and detected a dose as low as 0.0625 mg/kg in rats, indicating the sensitivity of a TSD analysis.

The mean false-alarm and hit rates for the group of five animals were calculated for each dose level and are shown in Fig. 2.

These values lie on a straight line running from the 0, 1 coordinate to an intercept on the positive diagonal. The 0, 1 coordinate represents perfect discrimination, while the positive diagonal represents chance discrimination. The intercept on the positive diagonal at 0.185 gives the response bias of the group when the signal cannot be discriminated from noise, i.e., $d' = 0$.

DISCUSSION

The results contradict the hypothesis that the scopolamine-induced disruption of behavior was the outcome of response disinhibition (e.g., Bignami, 1967; Russell, 1966). This hypothesis noted that responses competing with the reinforced response are suppressed during training and postulated that response suppression is mediated by a cholinergic system in the brain. Thus, drugs such as cholinolytics and high doses of anticholinesterases which block cholinergic function will "release" these suppressed responses. In terms of TSD, theories of response disinhibition would predict decreases in the response criterion, giving a greater number of "hits" and "false alarms" but leaving the animal's sensitivity to stimuli, d'_e, unchanged. This notion is clearly disproved by the results of this experiment, which showed decreases in d'_e.

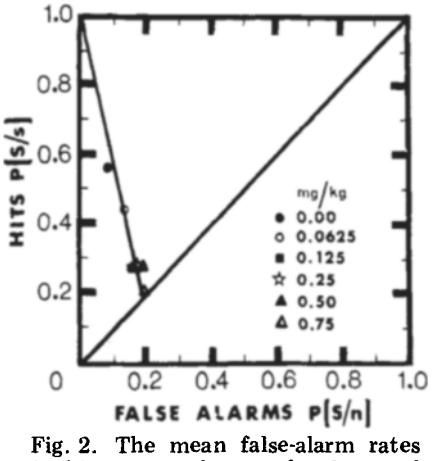

Fig. 2. The mean false-alarm rates and hit rates for each dose of scopolamine.

It was not possible, of course, to calculate the absolute changes in the signal-to-noise ratio, d', in the present experiment because the signal is unknown, but unpublished experiments from our laboratory, using specifiable stimuli, have enabled the calculation of the absolute changes in d', and the present findings have been substantiated.

The results of these experiments clearly indicate that scopolamine modifies behavior by reducing the signal-to-noise ratio and not by changing the animal's response criterion. In perceptual theory, attention has been interpreted as a process of stimulus selection produced by increases in the signal-to-noise ratio in specific sensory systems (Treisman, 1964) and impairment of attention would be expected to produce a change in sensitivity with little consistent change in the criterion. Thus, our results are consistent with the hypothesis that scopolamine disrupts "attention" mechanisms by impairing the ascending cholinergic reticular pathways (Warburton & Russell, 1969).

REFERENCES

ANGER, D. The role of temporal discriminations in the reinforcement of Sidman avoidance behavior. Journal of the Experimental Analysis of Behavior, 1963, 6, 477-506.

BIGNAMI, G. Anticholinergic agents as tools in the investigation of behavioral phenomena. In H. Brill (Ed.), *Neuro-psycho-pharmacology*. Amsterdam: Excerpta Medica, 1967. Pp. 819-830.

FERSTER, C., & SKINNER, B. F. *Schedules of reinforcement*. New York: Appleton-Century-Crofts, 1957.

HARZEM, P. Temporal discrimination. In R. M. Gilbert and N. S. Sutherland (Eds.), *Animal discrimination learning*. London and New York: Academic Press, 1969. Pp. 299-334.

HEISE, G. A., LAUGHLIN, N., & KELLER, C. A behavioral analysis of reinforcement withdrawal. Psychopharmacologia (Berlin), 1970, 16, 345-368.

GOODMAN, L. S., & GILMAN, A. *The pharmacological basis of therapeutics*. New York: Macmillan, 1965.

GREEN, D. M., & SWETS, J. A. *Signal detection theory and psychophysics*. New York: Wiley, 1966.

RUSSELL, R. W. Biochemical substrates of behavior. In R. W. Russell (Ed.), *Frontiers of physiological psychology*. New York: Academic Press, 1966. Pp. 185-245.

SIEGEL, S. *Nonparametric statistics*. New York: McGraw-Hill, 1956.

TREISMAN, A. Selective attention in man. British Medical Bulletin, 1964, 20, 12-16.

WARBURTON, D. M. Behavioral effects of central and peripheral changes in acetylcholine systems. Journal of Comparative & Physiological Psychology, 1969, 68, 56-64.

WARBURTON, D. M., & GROVES, P. The effects of scopolamine on habituation of acoustic startle in rats. Communications in Behavioural Biology, 1969, 3, 289-293.

WARBURTON, D. M., & RUSSELL, R. M. Some behavioral effects of cholinergic stimulation in the hippocampus. Life Sciences, 1969, 8, 617-627.