# Preshock, scopolamine, and genetic strain as determinants of the effects of punishment

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*A 1.0 mg/kg dose of scopolamine was more effective in attenuating preshock-produced augmentation of a response to test punishment than was a 0.5 mg/kg dose. Further, both the preshock and drug effects were dependent upon the strain of S.*

In another paper (Anderson & Payne, 1967) we demonstrated that scopolamine attenuated preshock-produced augmentation of response to test shock punishment due to its analgesic rather than amnesic action. Several questions emerged from this and other similar studies. First, because we did not always obtain this drug effect for different strains, genetic factors were thought to be involved. Further, because of failure to demonstrate the proactive preshock effect and of an unusual dose-response relationship reported by Berry & Stark (1965), this latter variable was included as part of the present study.

**Method**

The Ss were 40 male, naive, albino rats, 90-100 days of age at the beginning of experimentation. Half were acquired from the Northwest Rodent Supply Co., Pullman, Wash., and the other half from the Holtzman Co., Madison, Wis.

The preshock chamber was 6 in. wide x 10 in. long x 8 in. high, inside. The grids of the floor and the stainless steel walls were wired as separate electrodes. The preshock source was a tube-regulated, dc, constant-current device (Campbell & Teghtsoonian, 1958) set at 1.25 mA. Shock was scrambled.

The test apparatus was a 6-ft straight alley which provided total running times (obtained by a system of photorelays and clocks).

All Ss initially were trained to run the alley for food. Eight groups then were formed on the basis of their asymptotic running times ($F < 1$). Four groups were Holtzman and four were Pullman Ss. Three groups of each strain were administered preshock treatment which consisted of placement in the chamber for 3-1/2 min, the last 3 min of which involved continuous shock presentation. This was repeated on each of five consecutive days. One preshocked group from each strain was injected (IP) with a 1.0 mg/kg dose of scopolamine·HBr 20 min prior to the treatment. Another preshocked group from each strain was administered (IP) a 0.5 mg/kg scopolamine·HBr injection in an equal volume. Two additional groups from each strain were preshocked following injections with equivalent volumes of saline. The last set of two groups were not preshocked, but were injected with saline 20 min prior to placement in the treatment chamber. Strangely, one S from each of the Holtzman groups died on the day following the last injection (leaving four per group).

It is to be noted that the Dosage and Drug variables were "nested" under the Preshock condition. Nevertheless, the findings of this report were considered an important additional extension of our prior work in this area.

Testing involved return of all Ss to the alley. Following five food-retraining trials, food was removed, the food cup cleaned, and two shock punishments (60 V ac; 0.5-sec duration) were administered immediately in front of the food cup to all Ss. Three full-alley, shock-recovery trials were administered on each of the next three consecutive days.

**Results**

We analyzed the mean log total running times of all eight groups on the five food-retraining trials immediately preceding test shock punishment. Here, unlike any of our previous preshock studies, Preshock ($p < .01$), Strain ($p < .05$), and Preshock by Strain interaction ($p < .05$) effects were obtained. However, by the last retraining trial, group performances were equivalent and asymptotic ($Fs < 1$).

Figure 1 represents the performances, by trial, of the Pullman Ss on the three shock-recovery test days. Here a strong Preshock effect, collapsed over Trials and Days, was obtained ($p < .001$). Further, the action of scopolamine in attenuating the proactive preshock effect clearly was manifest in that both scopolamine-injected, preshocked groups exhibited running times

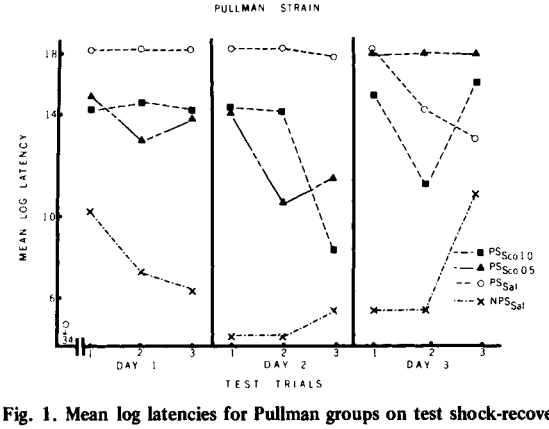


**Fig. 1. Mean log latencies for Pullman groups on test shock-recovery trials. The far left-hand column represents the combined mean log latencies for the four groups on the food-retraining trial immediately preceding test punishment. The legend is explained in the procedure.**

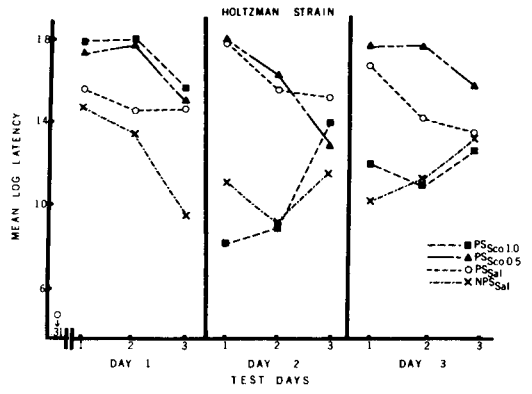


Fig. 2. Mean log latencies on test shock-recovery trials for the Holtzman groups. The far left-hand column represents the mean log latencies for Holtzman groups (combined) on the food-retraining trial immediately preceding test punishment. The legend is explained in the procedure.

intermediate between the saline-injected, preshocked and the control groups. Interestingly, while the Dosage effect was not apparent on Test Days 1 and 2, it was on Test Day 3. Here, the 1.0 mg/kg-scopolamine group exhibited less suppression than the 0.5 mg/kg group (which, in turn, exhibited more suppression than on previous test days).

Figure 2 represents the mean log running times for the Holtzman groups over the same test trials of Fig. 1. Neither a Preshock nor a Drug effect was observed on Test Day 1. However, by Day 2 both effects were more pronounced. Here the saline-injected, preshocked group exhibited more suppression (almost complete) than the saline-injected controls. Further, the 1.0 mg/kg-scopolamine group exhibited less suppression than did the 0.5 mg/kg group. Indeed, the performance of the higher dosage group was identical to that of controls, while the performance of the other was exactly that of the saline-injected, preshocked Ss. Test Day 3 was somewhat confusing because the Preshock effect was not reliable. Nevertheless, the trend was in the predicted direction and the results, in general, paralleled those of Test Day 2.

## Discussion

Pretest-punishment alleyway performance differences typically have not been observed in our other preshock studies. Fortunately, however, since all achieved equivalent asymptotic performances, the effects of test shock were not confounded with baseline differences.

Taking the shock-recovery data of Figs. 1 and 2 together, the analgesic property of the higher scopolamine dosage demonstrably was greater than that of the intermediate level or of saline. Here, our data corresponded with most of the dose-response studies involving scopolamine (e.g., Herrnstein, 1958; Leaf & Muller, 1966), and suggested a monotonically increasing action in attentuation of the proactive preshock effect over the range of dosages employed. These findings were viewed as having important implications for use of this drug as an anesthetic agent for treatments which have potent aversive consequences.

It should be mentioned that unusual side effects occasionally were observed at higher scopolamine dosages. In one preliminary preshock study, a group of five scopolamine-injected Ss died within 24 h following the fifth shock treatment. Further, following preshock, Ss occasionally gnawed the flesh away from both the ventral and dorsal surfaces of their hind paws.

Last, observation of the scopolamine-injected Ss during preshock treatment did not suggest that the drug reduced motoric activity. All preshocked Ss scrambled violently, squealed, and evinced pronounced autonomic activity during the first 60-80 sec of the preshock treatment on each day. The latter portion of the treatment period involved collapse of Ss to the grid floor in apparent exhaustion.

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