accordance with that information they might have gotten from experienced bees before they left the hive. Furthermore, the distribution of dark recruits once more resembles that of the light recruits, even though recruits from each hive would not have had access to information provided by experienced bees from the other hive.

That trained dark bees from the experimental hive could successfully recruit hive mates to the 500-m site is evident from a tally made after the control hive had been removed from the area. At that time 38 regular foragers succeeded in recruiting 49 new bees in 70 minutes (while only two landed at a control station at 350 m).

Results from the first experiment, in which regular foragers visited only the experimental site and 74 percent of all recruits caught had landed at that site, do not differ markedly from the results of earlier step-experiments of von Frisch and co-workers (*1*). The act of regularly switching the dishes, by itself, did not appreciably alter the distribution from that expected on the basis of the dance-communication hypothesis [in one respect, though, these results are not directly comparable to those reported earlier, because unmarked bees were collected upon landing, whereas von Frisch and co-workers tallied them as they approached the dish (*2*)].

Results from the second experiment (in which three of the four sites were made more similar to each other by having equal and regular visitation at each site), however, show a drastic alteration of the distribution of dark recruits at the various sites. Regular dark foragers from the experimental hive could provide information concerning the location of the 400-m site only, but most of the recruited bees from that hive arrived at nearer sites. Only 33 percent arrived at that site about which information would have been provided in the dance maneuver.

The close correspondence between the distributions of dark and light recruits in the second experiment emerges more clearly in the remarkable similarity between the distributions of dark and light bees obtained in the third experiment (*9*). Again, neither distribution resembles what one might expect on the basis of the dance-communication hypothesis. That is, bees returning to the control hive would be able to collectively furnish direction information concerning all sites, whereas bees returning to the experimental hive would be able to furnish information concerning the distance and direction of the 500-m, experimental, site only.

Furthermore, both distributions obtained in the third experiment exhibit an interesting symmetry about the geometric center of all sites. The percentage of recruited bees arriving at each site from both hives closely correlates with the distance of each site from the center of moment of all sites (*9*). Of all the distributions, that of the dark recruits in this third experiment reveals this most clearly. Whereas only 9 percent of the dark recruits arrived at the experimental site, 85 percent arrived at one of the two sites closest to the center of all sites.

The particular distribution of recruits may well arise from the fact that, even if all stations are equal in attractiveness, they would not necessarily be similar in their spatial relationship. The 300- and 400-m stations have at least one station on either side of them, but the end stations each have other stations in only one direction.

The results from the present experiments indicate that previous step-experiments (which contribute to the interpretation that recruited bees use the distance information contained in the dance maneuver) lack at least two essential controls in their design. For a step-experiment to be properly controlled, experimental and control sites should be as similar to each other as possible. Even if various sites are identical in attractiveness to bees, however, the geometry of the arrangement of various sites apparently funnels recruits toward the center of all sites (*2*).

Clearly, the dance maneuver executed by successful bees contains information related to the distance between the hive and the food source. My results, however, are not consistent with the interpretation that recruited bees use that information in arriving at the appropriate distance from the hive before they orient to the specific odor of the food. These results indicate, instead, that recruited bees apparently use other information after leaving their hive (including odor of hive mates or other bees) in the process of orienting to a particular food site visited by bees.

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9. The data published herein were not subjected to a statistical analysis for various reasons, in particular because the most interesting set of comparisons that could be made are not stochastically independent. Without a better understanding of the mechanisms contributing to the distribution of bees at feeding sites, one cannot compute expectations for comparison to the experimental results.
   The data obtained from this type of experiment suggest that the distribution may be multinomial with parameters functionally related to the distance from the geometric center of the stimulus sources. This preliminary notion of the mechanism involved is easily testable. However, we intend to do this by prospective experimentation—not by retrospective tests of goodness of fit to the data presented here. This will give us a probabilistic structure within which we can frame meaningful null hypotheses.
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Effects of Magnesium Pemoline and Dextroamphetamine on Human Learning

Abstract. *Two central nervous system stimulants, magnesium pemoline and dextroamphetamine, were tested to see if they facilitate learning in human subjects. Subjects under placebo learned faster than the subjects under any of the several doses of magnesium pemoline; however, none of these differences reached statistical significance. Subjects who received dextroamphetamine learned significantly more slowly than those who received placebo.*

The stimulant magnesium pemoline has been reported (*1*) to enhance learning and memory in the rat. According to Glasky and Simon (*2*) magnesium pemoline facilitates the synthesis of nucleic acids in the brains of rats. They argue that this finding is relevant to the biochemical findings which suggest that RNA synthesis or protein synthesis, or both, underlie memory and learning. Consonant with Glasky and Simon's biochemical findings is the

behavioral evidence of Plotnikoff (*3*), who reported that magnesium pemoline facilitates the learning of a simple avoidance response in rats selected as "slow learners," but that such rats tested after receiving the stimulants methamphetamine and methylphenidate did not learn differently from control rats that had received saline. Recently, Cameron (*4*) has been reported as stating that magnesium pemoline improves the memory of certain older patients suffering from intellective deterioration.

Few studies have dealt directly with the effects of stimulants on associative processes (*5*). We now present our initial findings with humans on how magnesium pemoline and dextroamphetamine affect the acquisition rate in a learning task in a normal population that is intellectually above average. We found that magnesium pemoline did not facilitate learning and that dextroamphetamine interfered with learning.

Our experimental subjects were 30 male university-student volunteers. A double-blind procedure was followed, and each subject was given a single oral administration of one drug. The five drug conditions used in this study were: (i) magnesium pemoline, 25 mg; (ii) magnesium pemoline, 12.5 mg; (iii) magnesium pemoline, 6.25 mg; (iv) dextroamphetamine, 15 mg; and (v) placebo. The subjects who received 12.5 mg and 6.25 mg of magnesium pemoline were tested after the tests of the other drug conditions were completed.

The apparatus included a visual display consisting of a row of eight small neon lamps arranged on a backboard which rested on a table 2.4 m in front of the subject. On the table directly before the subject were eight response keys. Individual lights served as the stimuli in the experimental tasks. Two tasks were used in the experiment. The simpler one was a reaction-time task in which the correct response to each light was to press the button directly in front of it. The learning task, with which this report is concerned, required the subject to learn which single key was the correct response to each light when the keys were randomly assigned, except that the correct button was never immediately in front of the light associated with it.

An interval of 500 msec intervened between a response and the presentation of the next stimulus. Any key press, erroneous or correct, caused the next stimulus in the sequence to appear. Error-feedback information was given, over earphones, to the subject by means of a brief tone immediately after a wrong response, but the error did not delay presentation of the next stimulus. Both stimulus and response, and whether the response was correct or incorrect, were automatically recorded for each trial.

The first day of the experiment was a training session to familiarize the subjects with the apparatus, and only the reaction-time task was administered. On the day of the experiment (1 day after training for those subjects who received 12.5- and 6.25-mg doses of magnesium pemoline, 3 days after training for all other subjects) testing was begun 2½ hours after the drug was administered. The first problem was the reaction-time task. Next, the subject was instructed to strive for maximum accuracy in the learning task. The learning session consisted of 840 trials which were balanced for frequency of stimuli.

The data for each subject were grouped into 15 blocks of 56 trials, and the proportion of correct responses in each block was calculated (Fig. 1). An arcsine transformation (*6*) was performed on these proportions to stabilize their variances, and the subsequent analyses were based on these transformations. The data analysis produced a measure of the subject's rate of learning as indicated by the change with practice in the probability of making correct responses.

The learning rate was calculated by the equation

$$LR = \frac{\text{asymptote} - \text{mean trial-blocks 2–9}}{\text{asymptote} - \text{trial-block 1}}$$

where LR is the learning rate, asymptote is the percentage of correct responses at asymptote, and where mean trial-blocks 2–9 and trial-block 1 refer to the percentage of correct responses made in each case. The method was suggested by Anderson (*7*).

The estimate of asymptotic performance was the subject's mean over trial-blocks 10 through 15; the first trial-block score was used as an estimate of initial performance. Thus the denominator of the ratio expresses the range of a subject's learning during practice, and the numerator represents the difference between a subject's asymptotic performance and his level of performance during the period of greatest learning. Since it was desirable to test the

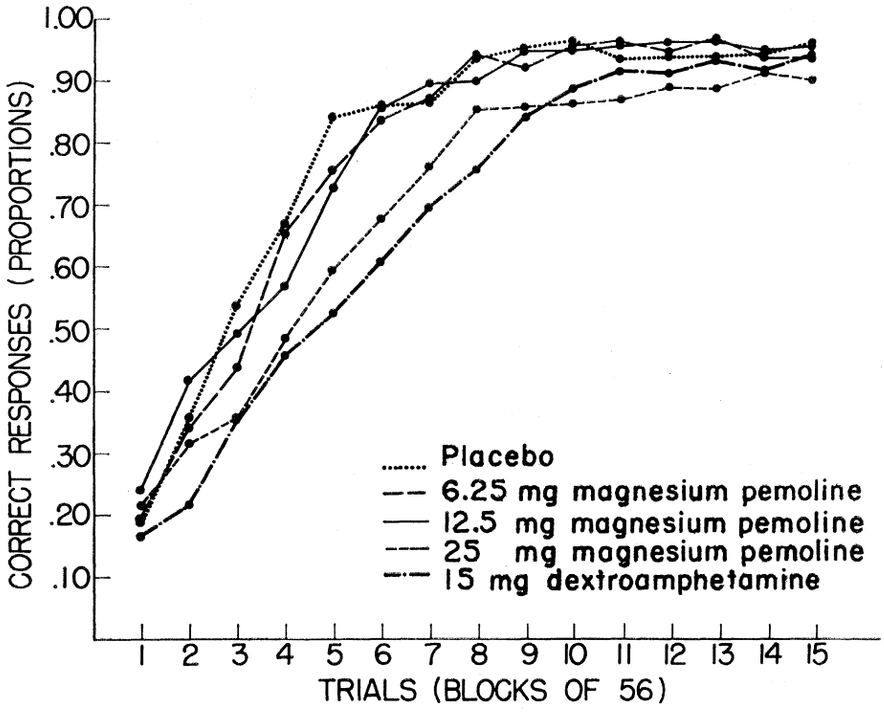

Fig. 1. Learning curves of the five drug conditions: placebo, 6.25 mg of magnesium pemoline, 12.5 mg of magnesium pemoline, 25 mg of magnesium pemoline, and 15 mg of dextroamphetamine. Each trial block consists of 56 trials balanced for order and frequency of stimuli. The ordinate value gives the proportion of correct responses for each trial block.

Table 1. Means and standard deviations of the learning rate scores for the drug conditions.

Dose (mg)	Mean	Standard deviation
	Placebo	
	.275	.084
	Magnesium pemoline	
6.25	.333	.045
12.5	.356	.077
25.0	.490	.077
	Dextroamphetamine	
15.0	.506	.055

curves for differences in learning rate during the period of greatest growth, it was necessary to estimate this segment of the curve. The learning curve was averaged for all subjects and each trial, beginning with trial 14, was compared with the last trial with Dunnett's test against a control (*8*). The first trial significantly different ($t = 2.74$, df 14/350, $P<.05$, one-tailed test) from trial 15 (asymptote) was trial 8. Consequently, the period of greatest learning was indexed by the average of the proportions of correct responses over trials 2 through 9. This value was used to calculate the learning rate.

A significant difference in the average value of this score indicates that the functions which relate practice to learning differ. For the various drug conditions Fig. 1 shows the learning curves and Table 1 shows the means of the learning rate scores (smaller values indicate more rapid improvement). Mean learning rate was fastest under placebo and increasingly slower under 6.25 mg of magnesium pemoline, 12.5 mg of magnesium pemoline, 25 mg of magnesium pemoline, and 15 mg of dextroamphetamine. Each active drug condition was compared with the placebo condition, with Dunnett's test against a control. Dextroamphetamine was the only drug condition significantly different from placebo ($t = 2.31$, df 5/25, $P< .10$, two-tailed test), the dextroamphetamine group showed a slower rate of learning than the placebo group. The rate of learning was not significantly different from placebo for any of the doses of magnesium pemoline. However, the higher the dosage of magnesium pemoline, the slower the mean rate of learning.

The direction of the differences between placebo and the various doses of magnesium pemoline is in agreement with what is known about the behavioral effects of magnesium pemoline in animals: higher doses inhibit learning (*9*). Only doses below this inhibitory range have been reported to facilitate behavior. In our study, however, learning rate scores under the lower doses of magnesium pemoline looked progressively more like the placebo scores. None of the results of our study indicated that in such subjects magnesium pemoline enhances learning. Other studies have indicated that acute doses of magnesuim pemoline enhance learning in rats (*3*). We did not find this to be true in human beings.

A moderately high dose of dextroamphetamine significantly slowed the rate of learning. The effects of the amphetamines on complex performance, in nonfatigued organisms, suggest that this class of stimulants interferes with performance. The amphetamines increase arousal (*10*), and high levels of arousal are detrimental to the acquisition of complex new associations (*11*). Such an interpretation may explain our findings with subjects under dextroamphetamine, since, while they learned more slowly than subjects under placebo, nevertheless the dextroamphetamine group in the simpler reaction-time task had significantly faster reaction times than the placebo group, and yet without any decrease in accuracy (*12*).

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Reserpine and Hypothalamic Mediation

Dominic (*1*) demonstrated that the rauwolfian alkaloid reserpine, a common tranquilizer, inhibits the olfactory blockage of pregnancy induced by the urine of strange male mice. He interprets this to offer ". . . direct evidence of hypothalamic mediation in the male-induced olfactory blockage of ovoimplantation in mice." This interpretation I question; and it should be acknowledged that obtaining good evidence of this is not so facile.

Reserpine causes general reduction in responsiveness to all stimuli, including alarming stimuli, of which the urine of a strange male may be one. At a more fundamental, albeit not necessarily more relevant, level, reserpine acts physiologically by depleting stores of catecholamines and serotonin—substances thought to serve neurotransmitter or neuromodulatory functions—and by impairing mechanisms for reconstituting these stores. But this effect is by no means specific to the hypothalamus; it is manifest in all parts of the central and peripheral sympathetic nervous systems (*2*).

Hypothalamic mediation of olfactory-induced blockage of implantation is probable and had indeed been suggested before (*3*). But experimental evidence of such mediation is not provided by the effects of this relatively nonspecific drug reported by Dominic.

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Dominic [*Science* **152**, 1764 (1966)] suggests that the effect of reserpine in blocking the failure of pregnancy in newly mated female mice produced by exposure to fresh urine from alien males was due to the hypothalamic action of reserpine.

This conclusion may be sound, but in humans reserpine frequently produces marked swelling of the nasal mucosa and blockage of the nasal air-