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Pharmacological manipulation of hoarding: Further analysis of amphetamine isomers and pimozide

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Dose-response relationships were established for the effect of d- and l-amphetamine on hoarding latency, the time course of hoarding, and the size of the total hoard. Both isomers displayed a marked effect on hoarding latency with the d isomer approximately twice as potent as the l form. The effects of the isomers on latency were reflected in the temporal profiles and in the sizes of the accumulated hoards. In a further experiment, pimozide—a dopamine blocking agent—drastically reduced the overall level of hoarding but had only a minor effect on hoarding latency. Pimozide exerted selective action on the amphetamine isomers, markedly antagonizing the effect of d-amphetamine on latency but displaying little effect on the l form. The results indicate that pharmacological manipulation can reduce hoarding activity in the rat by means of a number of distinct mechanisms.

Results from a number of investigations have raised the possibility that both the feeding and hoarding behavior of rodents may share a common neurophysiological substrate. For example, hoarding in the rat is modified by lesions of the neocortex (Stamm, 1953), limbic cortex (Stamm, 1954), and anterior hypothalamus (Vanderwolf, 1967), all of which zones have been implicated in ingestive behaviors (e.g., Mogenson & Huang, 1973). Moreover, for the hypothalamus, which traditionally has been firmly implicated in the control of food intake, experiments involving hypothalamic stimulation and lesioning have suggested that the controls for feeding and hoarding may be closely interrelated but not identical (Blundell & Herberg, 1973; Herberg & Blundell, 1967, 1970).

The elucidation of neurochemical mechanisms of feeding has prompted studies designed to explore the role of brain chemistry in hoarding. Current explanations favor a role for brain catecholamines in feeding (e.g., Grossman, 1975; Mogenson, 1974), and although intrahypothalamic injections of norepinephrine which induce feeding failed to initiate hoarding activity (Blundell & Herberg, 1970), other studies have reported that injections of amphetamine reduce both feeding and hoarding (Blundell, 1971; Zucker & Milner, 1964). Since the effects of amphetamine on feeding may be largely mediated via catecholamine mechanisms (e.g., Baez, 1974; Carey & Goodall, 1975; Holtzman & Jewett, 1971.), these data may provide some evidence for mechanisms involved in hoarding. Further understanding of the mechanisms mediating the action of amphetamine has been achieved through a comparison of the neurochemical and behavioral effects of the d and l isomers (e.g., Taylor & Snyder, 1971). Since the isomers appear to exert differing actions on brain dopamine and noradrenaline, this technique can be used to shed light upon which of these catecholamines makes the dominant contribution to the maintenance of specific behavior paterns. Recently, Nyby, Belknap, and Thiessen (1974) applied this procedure in an investigation of feeding and hoarding in the gerbil (*Meriones unguiculatus*). The results of this study suggested a role for noradrenergic neurons in the effect of amphetamine on feeding and hoarding.

The present investigation was designed to further examine the actions of the isomers of amphetamine in the rat by establishing dose response curves for the effects of the isomers on feeding and hoarding. Moreover, since Segal (1975) has suggested that the major distinction between the behavioral actions of d- and l-amphetamine may depend upon time differences in the activity of the drugs, the first experiment considered closely the temporal profiles of hoarding behavior. In addition, because of the observation that amphetamine anorexia appears to be partially accounted for by a blockade of the onset of eating (Blundell, Latham, & Leshem, 1976; Blundell & Leshem, 1975), it seemed necessary to include a measure of the latency to the onset of hoarding. Consequently, in addition to the traditional measure of the size of the total hoard, this experiment embodied information about the hoarding latency, the

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temporal profile of hoarding activity, and the rate at which hoarding was carried out.

A second experiment was designed to further investigate the relative contributions of dopamine and noradrenaline to the maintenance of hoarding behavior by observing the effect of a specific dopamine blocking agent and by administering this blocker in combination with the amphetamine isomers.

EXPERIMENT 1

Method

Animals and Apparatus. Male hooded rats (mean weight 325 g) were housed singly in wire mesh cages (310 × 230 × 200 mm) partly covered with darkened cardboard and provided with a water bottle and cotton wool for nesting material. These home-cages were attached to a wire mesh tunnel (310 long × 80 mm square) which led to a small compartment (160 × 160 × 160 mm) that formed the food store. When required, the rats were allowed access to the food store by means of a sliding door which normally separated the home cage from the hoarding tunnel.

Procedure and Design. The animals were allowed to become habituated to the cages and to the experimental room for 2 weeks prior to the beginning of any experimental procedures. When hoarding trials were initiated, the rats were deprived of food at 6:00 o'clock each evening and were then allowed access to the hoarding tunnels (by removing the separating doors) at 10:00 o'clock the following morning. A small tin tray containing a known number of pellets (diet 41B) was placed in the food store, and the rats were allowed to eat and hoard for a period of 1 h. At this time, the partition door was closed, the hoard was counted, and the total hoard was adjusted to 20 pellets which were left in the home compartments until 6:00 o'clock. This procedure was maintained for 1 full week to ensure that all rats would display hoarding behavior in the experimental situation. A similar procedure was adopted during 1 further week, except that the rats were given a sham intraperitoneal injection 30 min before the beginning of each daily hoarding trial. This strategy was devised in order to allow the rats to become accustomed to the stress of the injection procedure prior to the initiation of the experiment proper and was designed to minimize the influence of change in emotionality upon hoarding (Bindra, 1948).

During the course of the experiment, seven drug conditions were administered: 0.5, 1.0, and 2.5-mg/kg doses of d- and l-amphetamine and a control solution of .9% w/v NaCl. A within-subjects design was used in order to reduce the influence of large interindividual differences in hoarding activity, and each of the 10 rats received each drug condition once, with a period of at least 72 h separating successive injections. A counterbalanced order of presentation was used to minimize temporal order effects, and the experimental manipulations were performed under double blind conditions. Drug injections were made 30 min before the rats were allowed access to the hoarding tunnels. A 12-h light-dark cycle was used, with the light phase lasting from 0730 until 1930 h.

In order to derive temporal profiles of hoarding behavior, the number of pellets hoarded was noted at 15, 30, 45, 60, 90, 120, 150, 180, 210, and 240 min after the beginning of each hoarding trial. Moreover, an exact record was made of the time at which the first pellet was transported from the store into the home compartment—the hoarding latency. At the end of each trial, the hoard was adjusted to 20 pellets and was left in the home cage until 6:00 o'clock in the evening of the following day. This procedure was adopted to allow the rats to feed freely for 32 h after drug injection in order to avoid any sudden fall in body weight which could be brought about by a combination of anorexic drug action and experimental food restriction.

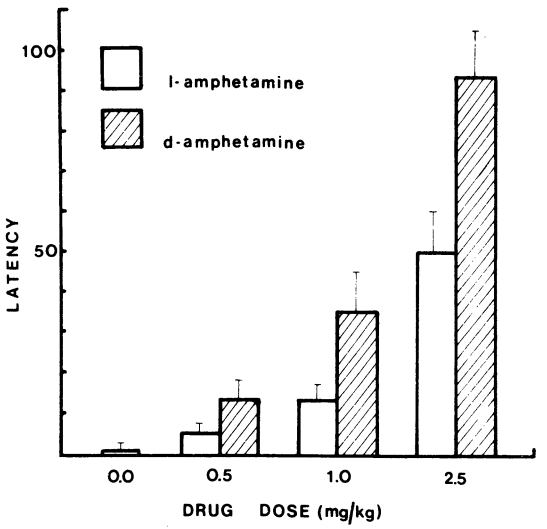

Figure 1. Dose-response relationship for d- and *l*-amphetamine on hoarding latency (minutes). Diagram shows means and standard errors.

The data were computer analyzed, using repeated measures analysis of variance procedures, and planned *a priori* two-sample comparisons were carried out when appropriate.

Results and Discussion

The effects of the drug injections on hoarding latency are shown in Figure 1, and it can be seen that all drugs gave rise to a noticeable increase in latency [$F(6,54) = 12.54$, $p < .01$]. In addition, two further features of these data deserve comment: First, a clear dose-effect relationship is apparent for both isomers, with the larger doses of the drugs producing the longer latencies. Second, d-amphetamine displayed a more potent effect on latency than did l-amphetamine—the period of suppression of the onset of hoarding for the d isomer being approximately twice as long as that brought about by the equivalent dose of l-amphetamine [$t(9) = 5.08$, $p < .01$, two-tailed test for pooled doses].

Figure 2 shows the temporal profiles on hoarding activity over the full 4 h of the experimental trials, and the dose-effect relationships observed for the latency measure are again reflected in the total number of pellets hoarded. All drugs reduced the total number of pellets hoarded over the 4-h period and the dose-effect relationships were preserved in the temporal profiles. In addition, d-amphetamine generally reduced hoarding to a greater extent than the l isomer, a finding consistent with previous work on the gerbil (Nyby, Belknap, & Thiessen, 1974).

From these data, it could be inferred that the amphetamine isomers separately influence the hoarding latency, the time course of hoarding activity, and the total number of pellets hoarded. However, the inset graph in Figure 2 shows the pooled hoarding

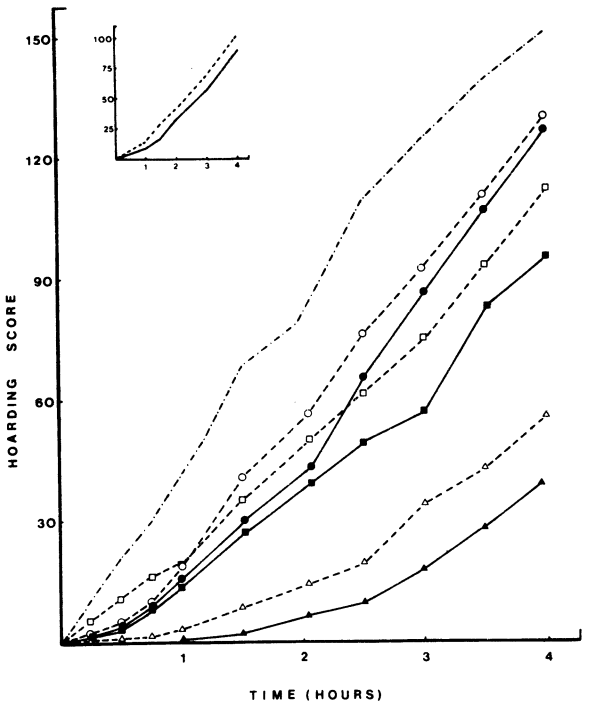

Figure 2. Temporal profiles of hoarding activity measured over a 4-h period. Open symbols with dashed lines indicate scores for *l*-amphetamine; dark symbols and continuous lines show data for d-amphetamine. Circular symbols, 0.5 mg/kg; square, 1.0 mg/kg; and triangles, 2.5 mg/kg. Broken line with no symbols, saline control injections. The small insert graph shows the profiles for pooled doses of *l*-amphetamine (dashed lines) and d-amphetamine (continuous line).

scores for the differing doses of the amphetamine isomers, and inspection of these summarized data illustrates that the difference in potency between the d and l isomers is much smaller than the 2:1 ratio (d:l) apparent for hoarding latency. Indeed, considering the total number of pellets hoarded over 4 h, the potency ratio is approximately 10:9 [t(9) = 0.5, n.s., two-tailed test for pooled doses], and it is clear that the potency ratio will vary according to the time elapsed since injection. Moreover, inspection of the temporal profiles after 90 min, by which time rats would be expected to have begun hoarding (see latency measures in Figure 1), indicates that the rate of hoarding is comparable for the two isomers (d-amphetamine, 0.50 pellets/min; l-amphetamine, 0.49). This suggests that once rats have actually started to hoard, the isomers do not differentially influence the rate at which hoarding occurs. Consequently, the major influence of the amphetamine isomers in this experiment seems to have been a marked suppression of the onset of hoarding activity. The observed effects of the amphetamine isomers on the temporal profiles of hoarding and upon the size of the total hoard appear, in large part, to be consequences of the effect of the drugs upon latency.

The prominence of the effect of amphetamine on hoarding latency directs attention to the nature of the mechanism underlying this action. One possibility is that the drug exerts inhibitory influence on a physiological mechanism responsible for the initiation of integrated behavior sequences. On the other hand, it could also be argued that amphetamine engenders stereotypic behavior or randomly directed locomotor activity, either of which could prevent the commencement of hoarding. It is possible that a consideration of the catecholaminergic mediation of the action of the isomers could provide a means of identifying whether or not amphetamine blocks the onset of hoarding by inhibiting the initiation of behavior or by merely interfering with the occurrence of any controlled activity. It has been noted earlier that the ratio of the potencies of d- and l-amphetamine for suppressing the onset of hoarding was 2:1. Significantly, it has been suggested that potency ratios of this order indicate dopaminergic mediation of drug action (e.g., Baez, 1974; Taylor & Snyder, 1971). In order to investigate the possibility that a dopaminergic mechanism may be involved in the initiation of hoarding, the following experiment examined the effect of hoarding of a dopamine blocking agent with fairly specific action—pimozide (Anden, Butcher, Corrodi, Fuxe, & Ungerstedt, 1970). The experiment was also designed to detect antagonisms of the effects of amphetamine by pimozide.

EXPERIMENT 2

Method

Animals and Apparatus. The rats and the hoarding compartments were identical to those used in Experiment 1. Consequently, all the subjects were experienced hoarders and were fully habituated to the experimental techniques.

Procedure and Design. This experiment was begun three weeks after the end of Experiment 1 and all rats were put on the 16-h deprivation schedule and given daily hoarding trials for one week prior to the commencement of drug injections. This procedure was adopted in order to ensure that the natural baseline level of hoarding of these rats had not been disturbed by the experimental manipulations in Experiment 1.

The experiment conformed to a 3×2 repeated measures factorial design in which each animal received six drug treatments, the order of presentation being counterbalanced to prevent bias. Before every hoarding trial, the rats received two injections. Thirty minutes before the onset of the trial, the rats were given injections of either d-amphetamine (2.5 mg/kg), l-amphetamine (2.5 mg/kg), or .9% w/v NaCl. In addition, at 4 h prior to these injections the rats received a dose of .45 mg/kg pimozide or vehicle.[1] This time interval was chosen since it has been shown that pimozide becomes maximally effective as a dopamine blocker between 2 and 8 h after administration (Soudijn & Wijngaarden, 1972). Moreover, in a separate dose-response study, we have shown that this dose of pimozide can effectively antagonize amphetamine anorexia when the drugs are administered with these temporal relationships (Latham, Strupp, & Blundell, Note 1). As in Experiment 1, measures of the number of pellets hoarded was noted at specified time intervals and a record was kept of the exact hoarding latency of each animal. In

addition, the feeding latency was measured by noting the time at which each animal began to eat a food pellet. Double-blind conditions were maintained throughout the experiment.

Results

Figure 3 shows the effect of the drug treatments on hoarding latency. In keeping with the results of Experiment 1, the analysis of variance revealed a significant main effect for the amphetamine treatment [$F(2,4) = 32.8$, $p < .01$], indicating that the amphetamine isomers produced differential effects on hoarding latency. This effect is clearly displayed in Figure 3. There was no significant main effect for the pimozide conditions [$F(1,18) = 0.34$, n.s.], and Figure 3 indicates that pimozide alone produced only a small change in the hoarding latency when compared with the control condition. However, the ANOVA revealed a significant interaction between the amphetamine and pimozide factors [$F(2,18) = 6.5$, $p < .01$], suggesting that pimozide was exerting a selective effect upon the amphetamine isomers. This supposition is confirmed by inspection of Figure 3, which shows that pimozide markedly antagonized the effect of d-amphetamine upon hoarding latency but had little influence on the action of the l-isomer.

Figure 3 shows the latencies for the rats to begin eating, and it can be seen that in general the drug treatment produced a similar pattern of effects: the d-amphetamine isomer gave rise to longer latencies than the l-isomer, and pimozide markedly attenuated the effect of d-amphetamine while marginally increasing the latencies for l-amphetamine. However, there is one important difference between the hoarding and eating latency scores, for pimozide alone produced no lengthening of the eating latencies. Indeed, the mean eating latency scores were 0.55 min for saline control injections and 0.54 min for the pimozide-plus-saline condition.

Figure 4 shows the temporal profiles of hoarding for the six drug treatment conditions, and it can be seen that the profiles for these doses of the amphetamine isomers are similar to those displayed in Experiment 1 (Figure 2). However, while pimozide produced only a slight effect on hoarding latency Figure 4 shows that this drug drastically reduced the level of established hoarding behavior [$t(9) = 7.4$, $p < .01$]; over the course of 4 h, pimozide-treated animals hoarded only a few pellets more than did rats given d-amphetamine. But Figure 4 also reveals that this time course of the effect of pimozide on pellets hoarded seems to differ from that of the amphetamine isomers: the hoarding profile of pimozide confirmed to a negatively accelerating curve, while the profiles for the amphetamine isomers showed an increased rate of hoarding over time.

The marked interaction between pimozide and the amphetamine isomers for hoarding latency was not fully reflected in the hoarding profiles or in the sizes

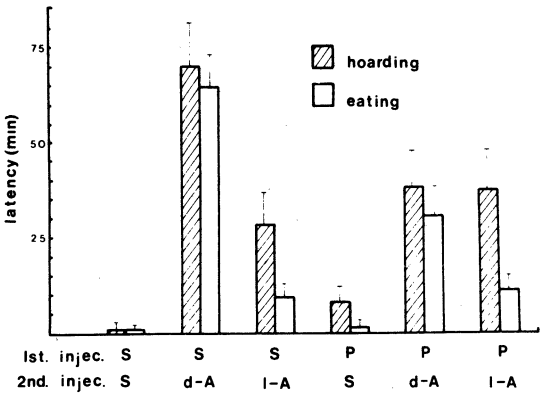

Figure 3. Effect of pimozide (P) injections on the hoarding and eating latencies arising from injections of saline (S), d-amphetamine (d-A), and l-amphetamine (l-A). Diagram shows means and standard errors. See text for statistical treatment.

of the total hoards. In keeping with the effects shown in Figure 3, pimozide had little influence on the action of l-amphetamine on total number of pellets hoarded. However, in contrast to the effect of pimozide on the hoarding latency of d-amphetamine, there was little modification of the hoarding profile or of the size of the final hoard [$t(9) = 0.7$, n.s.].

GENERAL DISCUSSION

The results of these experiments have demon-

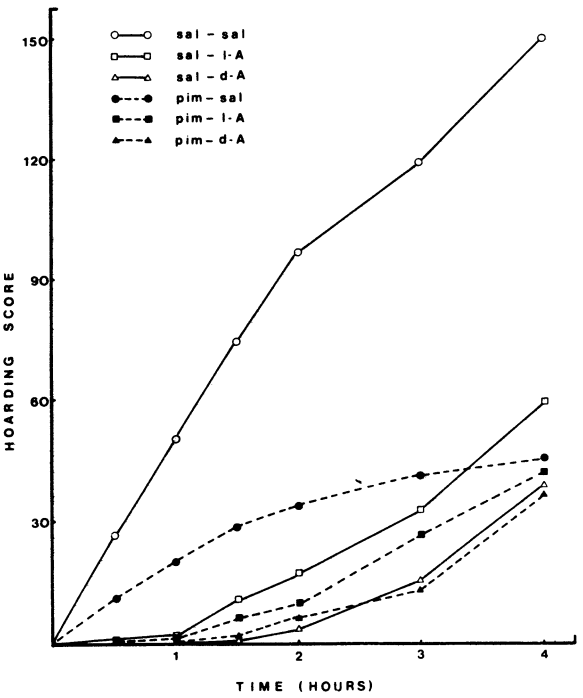

Figure 4. Temporal profiles of hoarding scores showing the effect of pimozide on the action of d- and l-amphetamine. See text for details.

strated that a reduction in hoarding behavior in the rat can be brought about through at least two distinct processes following pharmacological manipulation. Both pimozide and the amphetamine isomers gave rise to a clearly measurable diminution in the total number of pellets accumulated by the rats during the hoarding trials. However, this overall reduction in the size of the hoard was achieved by means of separate processes for the two classes of drug. While the amphetamine isomers reduced hoarding predominantly by suppressing the onset of hoarding activity, pimozide displayed only a minor effect on latency but reduced hoarding by curtailing the frequency of the hoarding journeys once hoarding had begun. It is worth noting that a measure of hoarding behavior based upon the number of pellets collected in a single time period (e.g., 1 h) would have failed to detect any difference between the modes of action of pimozide and the amphetamine isomers. Moreover, it is possible that a battery of measures of hoarding, including hoarding latency, the time course of hoarding, and the size of the total hoard, may throw light upon the mechanisms through which pharmacological agents modify this pattern of behavior.

For the amphetamine isomers, the data indicated that both the d and l forms reduced overall hoarding behavior by delaying the onset of hoarding journeys. One possible explanation for this observation is that the hoarding activity has been displaced by competing activities induced by the drugs. Significantly, amphetamine is known to produce behavioral topography known as stereotypy, which is dependent upon a dopaminergic mechanism (Fog, 1972). In turn, amphetamine-induced stereotypy can be blocked by pimozide (Ellinwood & Balster, 1974). Since pimozide significantly antagonized the action of d-amphetamine on hoarding latency (Experiment 2), this finding suggests that at least part of the debilitating effect of amphetamine on hoarding can be accounted for by drug-induced stereotypy interfering with the onset of the behavior. As pimozide also antagonizes the action of d-amphetamine on eating latency (Figure 3), stereotypy may provide a partial explanation for the anorexic action of high doses of amphetamine. The suggestion that stereotypy plays a role in suppressing the onset of hoarding (and eating) is supported by the observation that pimozide failed to antagonize the effect of l-amphetamine on hoarding latency. Since l-amphetamine does not produce stereotypic behavior at the doses used in this study (Segal, 1975), an antistereotypic action of pimozide would not be expected. Accordingly, it seems that while the action of d-amphetamine on hoarding latency may be partially mediated by drug-induced stereotypy, this explanation cannot account for the effect of the l-isomer. Moreover, it is noticeable that pimozide did not completely counteract the effect of d-amphetamine on latency. These observations suggest that, for both d- and l-amphetamine, a part of the suppression on the onset of hoarding is brought about through a mechanism which is not antagonized by pimozide; in turn, this suggests that the mechanism may not be dopaminergic.[2] One possibility is that the action of the amphetamine isomers on latency is mediated by an adrenergic or noradrenergic mechanism. This notion is in keeping with the findings of Leibowitz (1976), showing that one way in which the anorexic effect of intrahypothalamic amphetamine may be counteracted is by β-adrenergic blockers. Consequently, it seems that both adrenergic and dopaminergic systems may be involved in the effect of amphetamine on hoarding latency.

In contrast to the amphetamine isomers, pimozide brought about a drastic reduction in hoarding activity while displaying only a minor effect on hoarding latency. Moreover, this slight effect of pimozide on the onset of hoarding seems to have been brought about by factors different from those responsible for the amphetamine latency. While the amphetamine isomers suppressed the onset of eating and hoarding (Figure 3), it was noticeable that pimozide had no action on eating latency. Consequently, it can be deduced that at the beginning of each hoarding trial, pimozide-treated animals left the home cages as rapidly as saline-treated animals and made the journey down the hoarding tunnel to the food store. However, while saline-treated rats typically carried the first pellet back to the home compartment before eating, it was observed that pimozide-treated rats began eating in the food store. Accordingly, the small hoarding latency observed after pimozide injections seems to be entirely accounted for by the rats' eating in the outer compartment and thereby delaying the return trip of the first hoarding journey. The finding that pimozide exerted no effect on eating latency and showed only a small indirect effect on hoarding latency suggested that blockade of dopamine receptors does not markedly interfere with the initiation of these two activities. However, pimozide gave rise to a marked slowing effect on hoarding, which suggests that dopamine blockade impairs the rate at which hoarding behavior can be carried out. This suggestion is quite in keeping with the observation that pimozide severely slows the rate of eating (Blundell & Latham, in press).

The results of these experiments have shown that different parameters of rats' hoarding behavior may be differentially influenced by pharmacological agents, and have suggested that hoarding is a pattern of behavior under complex catecholaminergic control. In particular, it seems necessary to distinguish between mechanisms responsible for the

initiation and for the maintenance of hoarding behavior; this division has some similarity to the distinction between primary and modulatory processes (Cole & Gay, 1974). Hoarding behavior is susceptible to modification by agents such as amphetamine, which appears to act primarily on the mechanism responsible for the initiation of the activity, and by drugs such as pimozide, which show a dominant action on the maintenance of hoarding. This pattern of effects suggests at least a dual neurochemical controlling system. While recent work on hoarding in the gerbil (Nyby, Belknap, & Thiessen, 1974) has suggested a role for noradrenergic neurons in the amphetamine-induced suppression of hoarding, the results of the present study suggest that the initiation of hoarding can be disrupted by means of a dopaminergic process and suppressed by a noradrenergic system. In addition, it seems that a dopaminergic mechanism may be responsible for maintaining the rate of hoarding activity. Since it has recently been proposed that serotonin contributes to the overall regulation of feeding behavior in addition to the catecholamines (Blundell, 1975, 1977), further experiments seem to be required to investigate the influence of serotonergic manipulation on hoarding activity.

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NOTES

1. Pimozide was dissolved in a minimal amount of hot 0.01N tartaric acid solution and made up to required concentration with distilled water.

2. Alternatively, it could be argued that a dopaminergic mechanism is involved which may be susceptible to antagonism by other dopamine blocking agents such as haloperidol, butaclomol, α-flupenthixol, or sulpiride, but not by pimozide.

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