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An Hallucinogenic Amphetamine Analog (DOM) in Man*

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Summary. The amphetamine analog, 2,5-dimethoxy-4-methylamphetamine (DOM), was studied in 18 volunteer subjects given single doses ranging from 2 to 14 mg. The former was a threshold dose, with definite psychotomimetic effects being evident from doses over 5 mg. The clinical syndrome greatly resembled that of the LSD-mescaline-psilocybin series of drugs, including its time-course. Somewhat more sedation was produced by DOM than would have been expected from the others, despite concomitant evidence of peripheral sympathetic stimulation. Just as with the other drugs, DOM increased plasma free fatty acids, decreased phosphorus and creatinine clearance, decreased circulating eosinophils and had little effect on catecholamine excretion. Performance of psychometric tests was impaired. Chlorpromazine treatment concurrently was found to attenuate the reaction. Tolerance rapidly developed when the drug was used chronically by patients.

Key-Words: Amphetamine — Hallucinogens — Drug Addiction — Psychopharmacology — Phenylethylamines.

The 2,5-dimethoxy, 4-methyl analog of amphetamine has been identified as the material found in several samples of the widely used hallucinogenic drug known as "STP". This compound is one of s series of synthetic amphetamine analogs, and was known by the abbreviation of DOM before becoming part of the hippie drug culture. Other analogs of amphetamine, such as trimethoxyamphetamine, 3-methoxy-4,5-methylenedioxyamphetamine (MMDA), or 3,4-methylenedioxyamphetamine (MDA), have been studied clinically and found to have psychotomimetic effects comparable to mescaline. (SHULGIN, 1964; ALLES, 1959.) The action of these compounds is reputed to be long and they are considerably more potent than mescaline itself. DOM represented a somewhat different chemical configuration from the above compounds. Previously, it had been the experience that even minor changes in the mescaline molecule

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severely impaired clinical activity. For instance, 3,4-dimethoxy-phenylethylamine, which lacks such a substituent at the 5-position, is inactive (HOLLISTER and FRIEDHOFF, 1966). Based on animal studies, it was concluded that 3,4,5-methoxy substitution was a requisite for hallucinogenic action of mescaline analogs (SMYTHIES *et al.*, 1967). Consequently, the variant in structure represented by DOM was of interest in terms of structure-activity relationships.

Methods

Subjects

Volunteer subjects were selected on the criteria of being in good general and mental health, not taking any current drugs, and not undergoing psychotherapy. Non-smokers were preferred as no smoking was permitted on the test day. Subjects must have been fasting (no food or calorie-containing beverages) from 7:00 P.M. the night preceding each test. During the day preceding the test, as well as the test days, they were to have avoided bananas, cheese, and alcohol. Subjects were permitted to terminate participation upon request.

Procedure

Procedures were done as follows for obtaining four different kinds of data:

Clinical. A self-reporting scale, the Clyde Mood Scale (CMS) was used prior to treatment and at 1, 3 and 5 hours after (CLYDE, 1963). A second form of self-report was a Symptom-Sign Questionnaire especially devised for past studies with psychotomimetic drugs. This questionnaire was completed soon after completion of the trial. A third source of data was tape recordings of brief conversations with the subject prior to the drug and at 30 min intervals during the first seven hours. Time cues were provided with each recording so that a systematic reconstruction of each clinical syndrome was possible by auditing the tapes.

Physiological. A battery of tests, called the Physiological Battery, was administered prior to treatment and at 2, 4 and 8 hours after. These included measurements, within the region of clinical accuracy, of pupil size, activity of deep tendon reflexes, sitting and standing blood pressure, resting pulse rate and oral temperature. Electrocardiograms were taken prior to treatment and between 4 and 5 hours after treatment in eight subjects.

Psychometric. Two forms of the Repetitive Psychometric Measures were used, the Number Facility Test (NF) and the Flexibility of Closure Test (FC). (MORAN, 1959.) Both have been sensitive to psychotomimetic drug effects in the past. These were obtained prior to treatment and 1, 3 and 5 hours after.

Biochemical. Urine collections were made over three accurately timed 2-hour periods, beginning at 7:00 A.M. and ending at 1:00 P.M. These 2-hour specimens were used to measure total creatinine, phosphorus and vanilmandelic acid (VMA) excretion (CROUT and ABRAHAM, 1962). Blood specimens were taken for determination of plasma free fatty acid (FFA) levels, glucose, total leucocyte and absolute eosinophil counts (TROUT *et al.*, 1960). Determination of serum creatinine and phosphorus were made at times appropriate for calculating clearances at each 2-hour period.

Doses of Drug

The drug was administered as 1 or 2 mg/capsules with lactose filler, given orally with water on an empty stomach. Eighteen subjects received separate trials of drug with the following doses employed: 2, 3, 4, 5, 6, 7, 8(2), 9, 10(2), 11, 12(4), 13 and 14 mg. In proportion to weight, doses ranged from 30 to 220 μg/kg, with a medium level of 132 μg/kg. Four subjects received doses of the drug along with simultaneous doses of chlorpromazine. One patient received 10 mg and 200 mg respectively and another 12 and 200 mg. Neither of these patients received a second dose of drug. Two others received 12 and 50 mg and 14 and 200 mg respectively, as well as receiving the former doses of the amphetamine analog alone on a separate occasion.

Two subjects, recruited from inpatients at the hospital, volunteered for a study of tolerance. They were treated before one of two trials with doses of 1 mg twice daily on the first day, increasing to 4 mg three times daily on the eighth day with the challenge dose of 12 mg being given on the ninth day. A month later, the same dose was repeated without any pretreatment.

The setting was "experimental", the room being austere and set in a series of laboratories and research offices. As little prior suggestion of effects as possible was given within the limits of providing adequate prior information for consent. Much of the first four hours after the drug was administered was spent in testing of one sort or another. After that, subjects were allowed to eat and the pace became somewhat more relaxed. On most trials, two subjects received drug concurrently with relatively free communication between them.

Results

Clinical Syndromes

The clinical syndrome from DOM varied widely depending on the dose. The lowest dose administered, 2 mg, was threshold with mild but definite effects. Doses in excess of 5 mg almost always produced appreciable psychotomimetic effects. Because the nature of the psychotomimetic

Table 1. *Development of clinical syndrome—tape-recording data*

Time	Somatic	Perceptual	Psychic
30 min	Feelings of warmth Tingling sensations Dizziness, light-headed Sweaty palms Eyes feel heavy	Blurred vision Voices distant, echo effect	Slight mental confusion Laughing, euphoric Difficulty thinking, speaking
1 hour	Restless or relaxed Nausea, vomiting alaise, sick Muscle quivering, tremors Fullness in head Fearful	Visual illusions Increased contrasts Increased colors Multiple surfaces Increased after-images Light patterns, eyes closed Objects vibrate Hearing more acute, voices echo-like Sounds perceived as lights	Hilarity, laughing Impaired thinking, concentration Difficulty in speaking
$1^1/_2$ hrs	Same, plus Muscle fatigue Palpitation Tachycardia Increased nasal, ocular secretion	Visual illusions Same, plus Halos around objects Colored patterns, eyes open Increased peripheral vision Stroboscopic effects Time sense distorted, time meaningless	Mood changeable, usually happy Loss of control of thoughts Poor concentration Loss of sense of reality
2 hrs	Same, plus Relaxed or drowsy Poor coordination Cold or warm feelings Trembling, tense Pressure in chest	Same, plus Kaleidoscopic patterns Spatial distortions Difficulty focussing	Same, plus Rare paranoid or hostile thinking
$2^1/_2$ hrs	Same, plus Coughing	Same, plus Objects move Surfaces undulate Patterns from sounds	Same, plus Irritable Feel separated from body
3 hrs	Same, plus Limbs heavy	Same, plus Altered perspectives Distorted body image Awareness of skeletal structure	Same, plus Increased self-esteem Fluctuating levels of impairment Some waning of effects
4 hrs	Sweating Headache "Drunk", drowsy Lack of hunger Residual nausea	Same, plus Faces distorted Enhanced sensory experiences	Same, plus Poor motivation Feeling of decrescendo

Table 1 (continued)

Time	Somatic	Perceptual	Psychic
5 hrs	Sweaty Restless, tense Nausea Sleepy, drowsy	Most perceptual effects Still present, but waning	Same, plus Mild incoherence Tendency toward acting-out Feeling of "coming-down"
6 hrs	Shaky, tense Appetite poor Some nausea Headache Muscles tight Fatigue, sleepy, drowsy	Definite waning of all perceptual effects	Some mildly depressed Residual impairment in some Some relatively clear
7 hrs	Physical exhaustion "Hang-over" Headache Hungry	Most effects dissipated	Minimal residual impairment Usually happy

effect of this drug was of major interest in the study, the majority of subjects received doses in excess of 5 mg. Both the intensity and duration of action seemed somewhat related to the actual dose used, perhaps more so than to the dose per weight basis.

As judged by the data obtained from tape-recorded interviews, clinical effects were often appreciated within 30 min after drug administration, reaching a peak between 3 to 5 hours, following which there was a gradual subsidence over the next few hours. With low doses, subjects were usually completely clear by 7 hours. The predominant somatic, perceptual and psychic symptoms and their development in time are shown in Table 1. Questionnaire data supported the same general type of clinical syndrome. Those questions most often answered positively or showing the greatest degree of intensity are summarized in Table 2.

Mean scores on the six Clyde Mood Scale factors are summarized in Table 3 for the 16 subjects who received only DOM and for the 4 subjects who received DOM and chlorpromazine. The drug when given alone caused a marked decrease in clear-thinking and an increase in dizziness. These two changes have been reported by subjects from other psychotomimetic drugs we have tested. Dividing subjects into "high" and "low" dose groups as determined by a median dose revealed that the higher doses altered these two symptoms appreciably more than the lower ones. In addition to these changes, subjects given chlorpromazine reported decreased friendliness, increased sleepiness and increased discomfort.

Table 2. *Clinical syndrome—questionnaire data*

Somatic
Nausea
Appetite decreased more often than increased
Increased sweating
Feelings of heat slightly more often than cold
Paresthesias
Tension, tremors
Fatigue
Perceptual
Blurred vision
Double or multiple images
Vibration of objects
Visual hallucinations
Shapes distorted
Objects appeared lighter
Colors more vivid
Body looked distorted
Details stood out
Contrasts increased
Time slower subjectively
Hearing increased
Psychic
Difficulty in attending
Aware of environment
Happy, uncontrolled laughter
Control of thoughts lost at times
Difficulty in expression
Words inadequate to describe experience
Mind flooded with thoughts
Mind occasionally blank
Memory poorer at times
Easily distracted
Good memory for drug experience

These latter changes are what might be expected from normal subjects given substantial doses of chlorpromazine alone.

Physiological Measurements

The predominant findings were evidence of sympathetic stimulation: pupillary dilatation, increased deep tendon reflexes, tremor and increased pulse rate (Table 4). Although the majority of subjects remained in their normal state of alertness, some showed definite drowsiness early in the course of drug effect; no direct stimulation was noted. Comparing "high" versus "low" dose groups, increases in pupil size, blood pressure and pulse rate were clearly more related to the higher doses of drug. Only three subjects had a temperature rise of 1° C or more during the test

Table 3. *Mean scores on Clyde mood scale factors*

	16 Subjects Drug alone				4 Subjects Drug and Chlorpromazine			
Mood Factor/Time	Pre	1 hrs	3 hrs	5 hrs	Pre	1 hrs	3 hrs	5 hrs
Friendly	51	51	55	51	52	42	38	38
Aggressive	49	49	49	47	44	45	48	44
Clear-Thinking	49	41[a]	37[a]	41	50	37	37	38
High dose	49	38	34	41				
Low dose	50	44	39	41				
Sleepy	51	49	53	51	44	50	58	44
Dizzy	47	62[a]	62[a]	59[a]	49	66	63	53
High dose	48	70	62	61				
Low dose	48	54	62	56				
Unhappy	40	39	38	41	39	36	53	53

[a] $p = .001$, t-test, correlated means, 2-tail.

period; all were in the "high" dose group. Despite a subjective feeling of weakness, none was detectable clinically in any subject.

All of the eight subjects on whom EKGs were taken received doses of drug at the higher ranges (11 to 14 mg). Five subjects showed a change in the EKG 4 to 5 hours after drug, with slightly lower, rounder T-waves in leads 1 and 2, and V2 and 6. One subject, who had been pretreated with the drug for 8 days prior to challenge with a 12 mg dose showed no change in the EKG. In one subject, the T-waves initially did not have the usual peaking, but became more normal in appearance after the drug. One patient who received 200 mg of chlorpromazine along with 12 mg of the drug, showed changes typical of the phenothiazine effect: flattened, lower T-waves and lower ST segments in lead 1, 2, 3, AVL and V2-5.

Biochemical Tests

Free fatty acid (FFA) mobilization was clearly evident from this drug, the degree resembling that we have observed from other psychotomimetics, with the peak being reached at 4 hours. This increase in FFA occurred in the face of a slight increase in plasma glucose levels (Table 5). Both creatinine and phosphorus clearance were reduced significantly during the 2-hour period following the drug as compared with the immediately preceding 2-hour period. Reduction was of comparable degree for both clearances. VMA excretion was not significantly changed, although a trend toward increased excretion was noted in the "high" dose group. Total leucocytes were increased but directly counted total circulating eosinophils were decreased substantially. These changes were

Table 4. *Physiological changes in subjects receiving drug alone*

	Change from Baseline at		
	2 hrs	**4 hrs**	**7 hrs**
Obtunded consciousness	6	4	0
Tremor	8	7	4
Inc. deep tend. reflexes	14	14	7
Pupils dilated (mm diam)	15	14	10
High dose, mean	2.1	2.1	1.7
Low dose, mean	1.4	0.8	0.4
Blood pressure (mm Hg)			
Systolic, sitting			
High dose, mean	11.5	14.5	9.4
Low dose, mean	− 3.1	0.2	0.7
Systolic, standing			
High dose, mean	10.9	12.6	7.7
Low dose, mean	− 1.2	− 3.4	− 4.5
Diastolic, sitting			
High dose, mean	4.5	1.9	2.5
Low dose, mean	1.4	2.4	− 3.1
Diastolic, standing			
High dose, mean	9.5	4.0	2.6
Low dose, mean	1.0	2.1	− 6.3
Pulse rate, sitting			
High dose, mean	8.4	19.6	23.9
Low dose, mean	3.2	1.2	9.2
Pulse rate, standing			
High dose, mean	9.9	23.3	25.5
Low dose, mean	− 1.1	− 2.0	5.7
Temp. (degrees cent.)			
Mean	+ 0.1°	+ 0.3°	+ 0.4°

similar in the four patients treated concurrently with chlorpromazine as in the sixteen who received doses of the drug by itself. Although there were some differences between the responses of "high" and "low" dose groups, these were smaller in the case of these biochemical measurements than in other comparisons.

Psychometric Tests

Both psychometric tests showed signs of impairment, mostly attributable to the responses of the "high" dose group (Table 6). The number of problems attempted, as well as those correctly done, were significantly decreased for the Number Facility Test at 3 to 5 hours. The number of

Table 5
Biochemical and laboratory measurements from subjects receiving DOM alone

	Control	2 hrs	4 hrs	5 hrs
Free Fatty Acids (FFA)	644	885[b]	1352[b]	1200[b]
High dose, mean	636	887[a]	1630[b]	852[a]
Low dose, mean	654	882[a]	1074[b]	1093[b]
Glucose	94	99	99	100
Creatinine clearance				
High dose, mean	89	83		
Low dose, mean	109	92		
Phosphorus clearance				
High dose, mean	12.1	8.8[a]		
Low dose, mean	11.4	6.8[a]		
VMA excretion				
High dose, mean	3.1	3.5	3.1	
Low dose, mean	3.0	2.9	2.7	
Eosinophils				
High dose, mean	175		45[b]	
Low dose, mean	163		73[a]	
Total leucocytes				
High dose, mean	5100		8900[a]	
Low dose, mean	7500		8800	

Control-average of 3 determinations in case of FFA and glucose; 2 determinations in case of eosinophils and total leucocytes; 120 min period in case of clearance tests.

FFA—μmol/l; glucose—mg/per 100 ml; eosinophils and leucocytes—cells/cu.ml; VMA>μgm/mg creatinine.

[a] $p < .05$; [b] $p < .01$; t-test of correlated means.

Table 6. *Performance on psychometric tests of subjects receiving DOM alone*

	Pre		1 hrs		3 hrs		5 hrs	
	Att	Corr	Att	Corr	Att	Corr	Att	Corr
Number Facility								
High dose, mean	46	44	31	27	13[a]	9[a]	18[a]	14[a]
Low dose, mean	54	50	51	49	44	41	50	47
Flex. of Closure								
High dose, mean	21	17	25	8[a]	19	3[a]	21	7[a]
Low dose, mean	24	16	27	20	23	16	26	18

[a] $p < .01$, t-test correlated means, compared to pre-.
Att—problems attempted; Corr—number correct.

correct figure drawings in the Flexibility of Closure Test was significantly decreased at 3 hours. The main difference between the tests was that the percent of accuracy in relation to attempts remained rather constant for the NF test but declined appreciably for the FC test.

Interactions with Chlorpromazine

Despite the relatively few cases involved, it appeared that chlorpromazine ameliorated the clinical syndrome rather than aggravating it. One subject, who received 14 mg of drug, with 200 mg of chlorpromazine at one time and without it on the other, experienced a markedly different reaction with the addition of chlorpromazine; psychotomimetic effects were diminished (though by no means absent) and marked sedation was evident. Another subject who had 12 mg of drug and 50 mg of chlorpromazine on one occasion and none on the other had a definite psychotomimetic experience on both trials but the effects were appreciably greater in the absence of the phenothiazine. One subject who received a 200 mg dose of chlorpromazine along with 12 mg of drug had a generally disagreeable experience, mostly due to the effects of chlorpromazine; another who received 200 mg along with 10 mg had a generally mild effect with no apparent discomfort. It seems highly doubtful that chlorpromazine is a specific antagonist for any of the psychotomimetics, but its over-riding sedative effects may ameliorate some aspects of the reactions.

Chlorpromazine had little effect on the physiological or biochemical measures. In general, subjects treated with both drugs had higher pulse rates than might have been expected from DOM alone, and more sleepiness and loss of strength were also observed. Systolic blood pressure rose in the sitting position, but diastolic pressure tended to fall; in the standing position, the expected orthostatic hypotensive effect of chlorpromazine was made apparent by a fall in both pressures. Free fatty acids rose about as much from the combination of drugs as from the drug alone (mean pre-treatment value of 617 to peak value of 1420 μmol/L at 4 hours). Plasma glucose rose substantially more (mean value of 92 mg per 100 ml pre-treatment to 105 to 110 during first 5 hours). The fall in total eosinophils and rise in leucocytes was comparable to that observed with DOM alone. Psychometric tests showed far more impairment from the drug combination than from DOM alone.

Tolerance

Pretreatment with graduated and increasing doses of DOM produced a state of partial tolerance. The clinical syndromes reported by both patients who received 12 mg challenge doses following pretreatment were much milder than might have been anticipated and considerably less than they experienced 4 weeks later after the same dose without

pretreatment. On the latter occasion, the clinical phenomena closely resembled those described above. Many of the physiological measures were attenuated by pretreatment, although slight changes in deep reflexes, pupil size and blood pressure and pulse were still detected in the tolerant state. One patient, who seemed to have developed more clinical tolerance than the other, also had a lesser rise in FFA (252 to 729 in tolerant state; 261 to 1108 μmol/L in the non-tolerant state). As compared with their performance on the psychometric tests during the two doses of DOM, both patients showed a marked decrease in the number of problems attempted in the non-tolerant state.

Discussion

The clinical syndromes reported resembled those from the LSD-mescaline-psilocybin group of drugs in virtually every respect, both quantitatively and qualitatively (HOLLISTER and HARTMAN, 1962). The same was true for the timecourse. One would judge this compound to be 40 to 50 times as potent as mescaline, but until some questions about the purity of the material used in this study are settled, no firm estimates can be made.

The strong sympathomimetic actions of this drug were also clearly evident and in this regard also resembled closely the drugs of the LSD-mescaline-psilocybin series. If there were any differences in this regard, it was that DOM had more direct effects on blood pressure and heart rate than the others. The fact that three laboratory measurements consistently altered by the LSD group of drugs were similarly altered by DOM (increased FFA, decreased phosphorus and creatinine clearance, and decrease in eosinophils) confirms the similarity of action. These biochemical effects were not appreciably blocked by the concurrent use of chlorpromazine, which is not very surprising, as most are probably mediated through beta-adrenergic receptors and chlorpromazine is essentially an alpha-receptor blocking drug.

Although reports in the press had indicated that some adverse reactions might be expected if chlorpromazine were administered as an antidote to patients with "STP" intoxication, such was not the case in our study. In view of the great resemblance of DOM to LSD or mescaline, it seemed reasonable that chlorpromazine should have a similar attenuating interaction, which is what impelled us to try the combination out in four subjects. Quite possibly some of the earlier difficulties in treating "bad trips" from this drug with chlorpromazine were due to overenthusiastic use of the latter drug or use of preparations containing mixtures of DOM with centrally-acting anticholinergic drugs.

The development of tolerance was also expected, as this had also been reported for all drugs in the LSD-mescaline-psilocybin series. Very

likely, some cross-tolerance might also be expected, especially between DOM and mescaline.

Although blind controls were not used in the present study, it would be absolutely beyond belief that subjects could so consistently report a similar clinical syndrome from an essentially unknown drug. To mimic the physiological and biochemical changes would also be impossible. Finally, these were as clear evidences of a dose-response relationship as one can ever achieve in human subjects given drugs of this rype. The numerous non-drug variables which alter responses in humans are well known and do not permit a highly accurate determination of such relationships. Still, an independent but concurrent study of the same drug in which threshold doses (2 to 3.2 mg) were used revealed considerably different responses in subjects than were encountered with the larger doses used in the present study (SNYDER, FAILLACE, and HOLLISTER, 1967).

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