LSD or DOB?

SIR: In "Mania Associated with LSD Ingestion" (November 1981 issue), C. Raymond Lake, M.D., Ph.D., and associates described a patient who experienced a typical manic episode 3 weeks after taking an oral dose (on a slip of blotter paper) of what he thought was LSD. Chemical confirmation of the substance ingested was impossible. Dr. Lake and associates believed that "the medium, cost, time until onset, duration of symptoms, and their characteristics strongly indicate" the agent taken was LSD or an LSD analogue. They also stated that because of high potency, allowing effective doses in the microgram range, "LSD is the only drug of abuse dispensed on a slip of blotter paper." This mistaken information may have weighed unduly in their virtual acceptance of the substance involved as LSD. May we suggest that an alternative possibility exists?

Another hallucinogen is purveyed in blotter paper units. That agent is 4-bromo-2,5-dimethoxyamphetamine, known frequently in laboratory studies as DOB but also as bromo-DMA (1). According to 19 anonymous brief notations in the Drug Enforcement Administration bulletin *Microgram* (vols. VI–XIV, 1973–1981), since 1972 DOB has repeatedly been found sold in blotter paper doses in the United States, New Zealand, Australia, the United Kingdom, West Germany, and Greece. Extractable drug averaged .93–2.8 mg per dosage unit for analyses from the United States. Delliou (1) found individual squares to contain 1.4–4.6 mg. In most sales of DOB on blotter paper, it was being misrepresented as LSD (caveat emptor!), to which it is unrelated chemically.

A clinical study of DOB (2) found .2 mg to be the minimum detectable oral dose; the highest dose tried, 2.0 mg, produced "intellectual and emotional stimulation" and perceptual enhancement but not perceptual distortions or hallucinations. However, outside of clinical trials DOB reportedly causes not only hallucinations but also "disorientation often leading to panic state" (1).

LSD is rapidly absorbed after an oral dose and initial symptoms usually appear within a few minutes; visual illusions and perceptual distortions occur within the second or third hour (3). First perception of drug action occurred as late as 2–4 hours after oral doses of DOB in 4 of 13 subjects (2). A pharmacokinetic study (4) showed that DOB (or a metabolite) began to concentrate in the brain only 3 hours after an oral dose. DOB is said (1) to have a longer duration, 12–24 hours, than LSD, 8–12 hours. Both of these characteristics of DOB match those of the drug ingested in Dr. Lake and associates' case—slow onset (3 hours) and a long recovery time (18–24 hours)—as well as or better than do those of LSD.

Thus, it appears that DOB could have been the psychotomimetic agent encountered by the patient described by Dr. Lake and associates. In view of this possibility, their description might better have been of "mania associated with ingestion of a psychotomimetic (or hallucinogenic) agent."

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Dr. Lake and Ms. Moriarty Reply

SIR: It is possible that the drug ingested by our patient, Mr. A, was not LSD but, rather, 4-bromo-2,5-dimethoxyamphetamine (DOB). We thank Dr. Davis for suggesting DOB as a possible alternative, one that we had not considered. However, we believe it is likely that Mr. A did, in fact, ingest LSD.

The relevant issue is the frequency with which DOB is sold as LSD in the United States. Some statistics are available from PharmChem Laboratories, a public service drug analysis laboratory that publishes information about street drugs (1). Of 76 street samples of alleged LSD received by PharmChem in 1980, 64 (84%) contained exclusively LSD. Only 4 of the 76 samples (5.2%) contained drugs other than LSD.

If Mr. A had ingested DOB, it is uncertain whether the dose would have been potent enough to cause the perceptual distortions described in our report. In the clinical tests described by Shulgin and associates, a dose of 2.0 mg of DOB caused no perceptual distortion in humans. According to Dr. Davis, U.S. street samples of DOB average .93–2.8 mg per dosage unit. Thus, the most potent dose Mr. A would be likely to have taken, 2.8 mg, is only .8 mg greater than a dose that caused no perceptual distortion in humans. Also, LSD (as well as DOB) fits the description of time until symptom onset in our case. Because DOB doses in the United States are relatively small and substitutes were apparently infrequently sold as LSD in the United States in 1980, it is probable that our patient ingested LSD.

In conclusion, we feel that the evidence points to LSD as the drug in question but fully agree that without a positive urine drug screen no positive identification can be made. That other potent hallucinogenic drugs can be distributed on blotter paper is important because treatment of overdose with amphetamine (or an amphetamine analogue such as DOB) differs from that of LSD overdose.

REFERENCE

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