



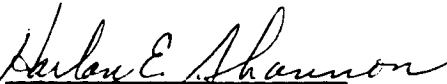
behavior and its deleterious effects, then it should remain available for clinical use. Morphine is an example of such a drug. On the other hand, if the perniciousness of a drug outweighs its therapeutic utility, then it should not be available for clinical use.

3. The premise that "whenever clinical descriptions are available for the effects of psychoactive drugs, these descriptions must of necessity supercede results from animal studies" is without merit. It has been documented numerous times that whenever the practitioner or scientist and the client or subject are knowledgeable as to the fact that a drug is being administered, subjective bias will strongly influence the subsequent conclusions as to the utility of the drug. Too many people are prone to a "placebo effect." That is, a person may respond in a manner which is in the expected therapeutic direction regardless of what has been given, including a placebo or sugar pill, if they believe that the "tablet" given should be therapeutically effective. For this reason, it has become well established that clinical descriptions of psychoactive drugs are valid only when both the practitioner or scientist and the client or subject are unaware of whether a drug or placebo has been given. Such studies are termed "double-blind, placebo controlled." To date, no such studies or clinical experience have been reported for MDMA, even though there has been ample opportunity. Thus, there are no valid clinical descriptions of the effects of MDMA to date. Therefore, we must rely solely on the available animal data.
4. The available animal data, as reviewed by Dr. Nichols, indicates that MDMA is predominantly amphetamine-like. This point should not be clouded by the use of such terms as "sympathomimetic." Amphetamine

is a sympathomimetic, as is MDMA, as Dr. Nichols concedes. While Dr. Nichols is correct in his assertion that the pharmacology of S-(+) or racemic MDMA is not adequately described by current pharmacologic definition, he presents no evidence to indicate that MDMA differs in any fundamental way from amphetamine.

5. The human studies cited by Dr. Nichols in support of the contention that S-(+) MDMA or racemic MDMA are different from amphetamine are anecdotal reports. There is no evidence that the human subjects would have been able to tell the difference between MDMA and amphetamine if they had been given comparable doses on separate occasions when they and the practitioner or scientist were unaware as to which drug the client or subject had been given.
6. Dr. Nichols states that "one therefore could not predict, a priori, whether it [MDMA] had therapeutic value, without adequate clinical trials." As discussed above, an adequate clinical trial would be a "double-blind, place controlled" trial. There has been ample opportunity for practitioners to conduct such a trial. The argument that MDA was popularly marketed as the "love drug" and MDMA has less hallucinogenic potential than MDA, and therefore by insinuation a better drug, is without merit. PCP also has been marketed on the street as a "love drug".

I declare under penalty of perjury that the foregoing is true and correct. Dated May 17, 1985.

  
Harlan E. Shannon

CURRICULUM VITAE

Name: Harlan E. Shannon

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University of Redlands, Redlands, CA	B.A.	1969	Psychology
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