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Effects of Cocaine on Human Aggression

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LICATA, A., S. TAYLOR, M. BERMAN AND J. CRANSTON. *Effects of cocaine on human aggression.* PHARMACOL BIOCHEM BEHAV **45**(3) 549–552, 1993.—Thirty male undergraduates received either a placebo, low dose (1 mg/kg), or high dose (2 mg/kg) of orally administered cocaine. Subjects were then given the opportunity to administer electric shocks to an increasingly aggressive fictitious opponent during a competitive reaction-time task. Aggression was defined as the intensity of shock the subject was willing to set for his adversary. The results of this study indicate that subjects in the high-dose cocaine condition reacted more aggressively than placebo subjects irrespective of level of provocation.

Aggression Cocaine Human

COCAINE has become a common drug of abuse during the past decade (2). It is estimated that 20 to 30 million people in the United States have used cocaine and that over 5 million are current users (8). Researchers also reported a concurrent increase in cocaine-related street violence (6).

There is considerable controversy concerning the relationship between cocaine consumption and aggressive behavior in humans. Several investigators proposed that cocaine, a psychomotor stimulant, can instigate aggression (5,6,10). In contrast, a number of researchers questioned the widespread belief that cocaine precipitates aggressive behavior. For example, Carr and Meyers argue, "the relationship of cocaine to violence derives more from fears and a longstanding bias against the drug than from any empirical data" (1). The results of the few studies designed to investigate the effects of cocaine on animal aggression are equivocal (11,12), and no experimental evidence for cocaine-induced human aggression exists.

A number of models have been proposed to account for the relationship between cocaine and violence (4,14,15). According to the psychopharmacological model, aggression is the direct result of the ingestion of the drug. The systemic model refers to the traditionally violent interactions that result from the production and distribution of an expensive and illicit commodity. The economic-compulsive model depicts violence as the instrumental behaviors used to support the habitual use of a drug. According to Goldstein, there are no empirical data to suggest how much drug-related violence is due to each of these three models (4). However, contemporary drug experts generally agree that systemic or economic influences account for most of the violence perpetrated by, or directed at, drug users (4,14,15).

The purpose of this study was to investigate the relationship between the psychopharmacological effects of an acute dose of cocaine and aggression in humans. It appears critical to determine if such a relationship exists rather than to arbitrarily dismiss this model in favor of the economic or systemic models of cocaine-related aggression.

Subjects who had consumed varying amounts of cocaine were given the opportunity to shock an increasingly provocative fictitious opponent while competing in the Taylor reaction-time (RT) task (16). Studies using the Taylor paradigm demonstrated the facilitation of aggression by alcohol (17) and diazepam (19), as well as the inhibition of aggression by marijuana (13).

METHOD

Subjects

Subjects were 30 male undergraduates, 18–25 years of age. Subjects were recruited by sign-up folders placed in buildings on campus. The possible use of drugs was not mentioned in the folder. Volunteers were contacted by phone and informed that the experiment concerned the effects of various drugs on perceptual-motor performance, that they would be paid $2.00 per hour for their participation, and that the experiment would be conducted at the University Health Center. To ensure the efficacy of the drug manipulation, subjects were asked to refrain from the use of drugs or alcohol for 24 h prior to the study and from eating for 4 h before the experimental session.

Procedure

Upon arriving at the health center, the subject was asked to read and sign a consent form that stated that the study would involve the use of electric shock and administration of either a placebo, a stimulant, a sedative, or a minor tranquilizer and that his health records would be reviewed by health center personnel. The specific use of cocaine was not men-

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tioned in the informed consent form. Also, subjects were not told which drug they would receive. This procedure was followed to control for subject expectancy effects and prevent the subject from associating a controversial drug, cocaine, with a particular subjective experience. The subject confirmed in the consent form, and to the experimenter, that he had not used drugs or alcohol in the last 24 h or ingested food in the last 4 h.

Subjects agreed to complete several questionnaires regarding their personal health. The responses to these questionnaires were examined by the first author to assure that each subject met the safety criteria for participation set by the health center personnel. The director of the health center, a physician, screened the health records of potential subjects to determine if any medical contraindications to their participation appeared in their health history.

Subjects' drug use history was assessed by means of a drug experience questionnaire. Subjects who reported they had no prior drug experience, and those who reported heavy use of drugs (i.e., several times a week or more), were excluded from the study. Thus, subjects in this study had some prior experience with drugs. To minimize the possibility that subjects would expect to receive cocaine, or any other particular drug, subjects were questioned about their experience with general classes of drugs (i.e., stimulants, hallucinogens, tranquilizers). While cocaine was given as an example of a stimulant, no specific assessment was made of cocaine use.

Each subject was randomly assigned to either a "low-dose" condition (1 mg/kg body weight of cocaine HCl placed in a gelatin capsule), "high-dose" condition (2 mg/kg body weight of cocaine HCl placed in a gelatin capsule), or an inactive placebo condition (a capsule containing just inert filler). To ensure subjects' safety, no participant received more than 250 mg cocaine HCl. The high-dose condition represents a moderately efficacious dose as indicated by pharmacological studies (18). The oral route of administration used in this study has been found to be about as efficient a mode of ingestion as intranasal administration (20). Intranasal administration was not used because of the increased probability that subjects would identify the drug consumed as cocaine.

Blood pressure and subjective impressions of drug effects were assessed four times during the experimental session: a) shortly after subjects' arrival (baseline); b) approximately 65 min after drug consumption (pretask); c) immediately following participation in the RT task (posttask), approximately 95 min after the consumption of the drug; and d) prior to debriefing (approximately 2 h and 15 min after drug consumption). A modification of the Addiction Research Center Inventory (ARCI) was used to assess the subjective effects of drug ingestion (9). This inventory consisted of 40 true–false questions, such as, "I would be happy all the time if I felt as I do now" and "My thoughts come more easily than usual." Validity for the ARCI is supported by numerous studies assessing the subjective and physiological effects of psychomotor stimulants, including cocaine (3).

After drug consumption, subjects were monitored by the experimenter for the next 65 min while waiting for the task to begin. This time interval allowed the drug to reach its peak pharmacological activity level (18).

The subject was next escorted to the experimental cubicle and seated in front of the subject task board. A concentric-ring electrode was attached to the subject's nondominant wrist. The experimenter next informed the subject that he was "Subject A" and that he would be competing with another subject ("Subject B") in the adjoining room. The experimenter then excused himself, ostensibly to prepare the second subject (a fictitious opponent) for the experiment.

Shock "unpleasantness" thresholds for the subject and "opponent" were then determined by administering a series of increasing shock intensities. The subject informed the experimenter when he first felt the shock and also when the shock was definitely unpleasant. This procedure was repeated for the opponent and overheard by the subject over an intercom. Shock calibration instructions, task instructions, and the verbal responses of the fictitious opponent were all tape recorded and controlled by the experimenter.

The subject was informed by means of a tape-recorded message that he was competing in a reaction-time task with another subject in an adjoining room. At the beginning of each trial, the subject was instructed to select (by pressing 1 of 10 buttons) the intensity of shock he wished his opponent to receive. He was informed that the level of shock he selected would be administered to his opponent at the end of the trial if he were faster than his opponent on the reaction-time task and that he would receive the shock his opponent selected for him if his opponent was faster. Regardless of who won, at the end of each trial a feedback light indicated the shock intensity that had been set for the subject by his opponent before the reaction-time trial. Thus, the subject was led to believe that either he or his opponent would receive a shock, depending upon the outcome of the competition, and both could select the intensity of shock the other would receive. In actuality, the frequency of wins and losses (50%) and the intensity of the shock were controlled by the experimenter. To establish the shock intensities to be administered to each subject, the intensity judged by the subject as "definitely unpleasant" during the shock threshold determination was designated as "10" (maximum shock intensity); "9" was set at 95% percent of the maximum, "8" at 90% of the maximum, and so forth.

Each subject then competed with the opponent for a series of 21 trials. These consisted of the first trial, three six-trial blocks of increasing provocation (trials 2–7, 9–14, 16–21), and two transition trials (trials 8 and 15). The purpose of the transition trial was to ensure credibility by smoothing the transition between blocks. The mean feedback settings for blocks one, two, and three, indicating increasing shocks selected by the opponent, were 2.5, 5.5, and 8.5, respectively. These feedback settings were defined as low, medium, and high provocation, respectively. The subject never received the maximum shock feedback setting (10).

Following the reaction-time task, the subject completed a posttask questionnaire. The subject was then debriefed, paid, and released.

RESULTS

Aggression was defined as the intensity of shock subjects selected for their opponent to receive. Mean shock settings as a function of drug dose and provocation are presented in Fig. 1. To determine the effect of cocaine on aggression across blocks of increasing provocation, a three × three (drug condition × blocks) split-plot analysis of variance (ANOVA) was performed on subjects' mean shock settings. According to this analysis, the main effect of drug dose approached significance, $F(2, 27) = 2.76, p = 0.08$. Planned pairwise comparisons using Dunn's correction and separate variance estimates (7) indicated that high-dose cocaine subjects ($M = 6.60$) set

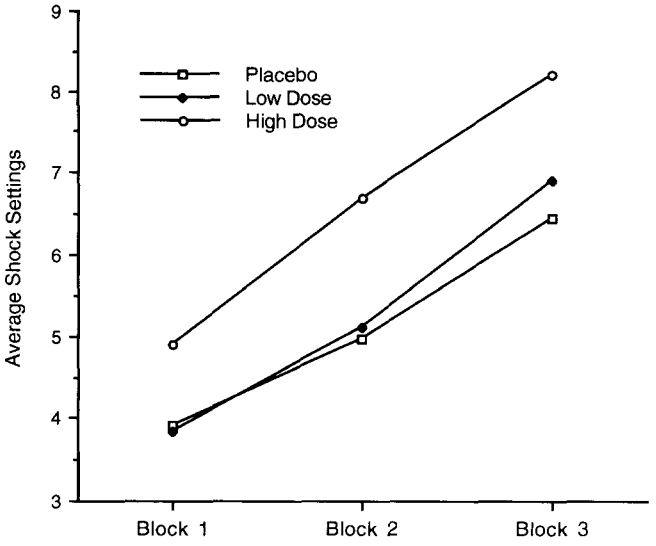

FIG. 1. Mean shock setting as a function of drug dosage and level of provocation.

significantly higher average shocks than placebo subjects ($M = 5.11, t = 2.64, p = 0.02$). However, low-dose subjects ($M = 5.28$) did not differ from placebo subjects on average shock settings ($t = 0.26, p = 0.80$). The main effect of provocation from this analysis was significant, $F(2, 54) = 41.48$, $p < 0.001$. Subjects' mean shock settings for blocks one through three were 4.21, 5.59, and 7.18, respectively. The interaction of drug dose and blocks, however, was not significant, $F(4, 54) = 0.33, p = 0.86$.

An inspection of Fig. 1 suggests that the mean shock settings for the placebo and high-dose cocaine groups diverged as a function of provocation. To further explore this possibility, a two × three split-plot ANOVA was performed on the shock settings set in the high-dose and placebo conditions. As expected, the dose main effect was significant at less than the 0.05 level. However, the interaction of drug dose and block was nonsignificant, $F(2, 36) = 0.82$, $p = 0.45$. Thus, subjects in the high-dose cocaine condition tended to set more intense shocks than subjects in the placebo condition, and subjects in both conditions appeared to increase their shock setting in response to increasing provocation at approximately the same rate.

It will be recalled that blood pressure and a measure of subjective effects were assessed four times during the study. A three × four (drug condition × time) split-plot ANOVA performed on mean systolic blood pressure yielded a significant main effect for dose, $F(2, 27) = 3.43$, $p < 0.05$. Mean systolic blood pressures for the placebo, low-dose, and high-dose conditions were 107.25, 110.50, and 115.40, respectively. The main effect of time was also significant, $F(3, 81) = 17.22$, $p < 0.001$. Mean systolic blood pressures for the four times were 107.87, 111.57, 115.07, and 107.87, respectively. The dose × time interaction was also significant, $F(6, 81) = 3.90$, $p < 0.01$. According to this interaction, systolic blood pressures of both the low- and high-dose groups steadily increased from baseline to posttask, while the systolic blood pressure of the placebo group did not significantly increase from the baseline.

A three × four (drug condition × time) split-plot ANOVA performed on mean diastolic blood pressure indicated that the main effect of drug dose was not significant at the 0.05 level, $F(2, 27) = 1.37$. Mean diastolic blood pressure readings for placebo, low-, and high-dose conditions were 72.27, 71.90, and 75.17, respectively. The main effect for time was significant, $F(3, 81) = 25.64$, $p < 0.001$. Mean diastolic blood pressures for times one through four were 69.30, 74.07, 76.70, and 72.40, respectively. The dose × time interaction approached significance, $F(6, 81) = 1.89$, $p < 0.10$. Subsequent Newman-Keuls tests revealed significant increases in diastolic blood pressure from baseline to pretask and posttask following ingestion of both low and high doses of cocaine but not following ingestion of the placebo.

The effects of drug dose and time on mean subjective "highness" as assessed by the ARCI were examined in a three × four (drug condition × time) split-plot ANOVA. The main effect for dose was not found to be significant. Mean highness scores for placebo, low-, and high-dose conditions were 16.17, 16.62, and 15.50, respectively. The main effect for time was significant, $F(3, 81) = 16.94$, $p < 0.001$. Mean highness scores as a function of time were 15.13, 18.40, 16.43, and 14.43, respectively. The dose × time interaction was also significant, $F(6, 81) = 4.82$, $p < 0.001$. According to this interaction, subjects in the high-dose condition reported a significant increase in the degree of subjective highness from baseline to the second measure. Subjects in the low-dose and placebo conditions did not evidence an increase in subjective highness. In addition, the groups did not differ in subjective highness during the third and last measurement time. Thus, subjects in the high-dose cocaine condition reported a significantly different subjective experience from other subjects just prior to the RT task.

Subjects' reaction times were monitored for each trial. A three × three (drug condition × blocks) split-plot ANOVA was performed on subjects' mean reaction times. Results of this analysis indicated that neither the main effect for drug, nor the drug × blocks interaction, was significant. Thus, cocaine did not appear to affect performance on the RT task.

It will be recalled that each subject reported when the shock was definitely unpleasant during the threshold determination prior to the RT task. This shock was the maximum shock the subject thought he could receive. A one-way ANOVA was performed on subjects' shock "unpleasantness" thresholds. According to this analysis, the main effect of dose was not significant, $F(2, 27) < 1$. Mean shock thresholds for the placebo, low-, and high-dose conditions were 8.9, 9.4, and 10.6 mA, respectively. An ANOVA was also performed on subjects' posttask ratings, based on a seven-point scale, of how "painful" the shocks felt. This analysis revealed that the main effect for dose was nonsignificant, $F(2, 27) = 1.80$. Mean pain ratings due to shock for the placebo, low-, and high-dose conditions were 2.4, 2.6, and 2.4, respectively. Thus, neither the objective measures of shock unpleasantness nor the subjective perceptions of pain varied as a function of cocaine dosage.

Subjects were also asked to indicate, posttask, which drug they thought they consumed. The list of choices included various tranquilizers, stimulants, and depressants, as well as the categories "no drug" and "other." Only two subjects selected cocaine as the drug consumed. One of these subjects received a placebo; the other received a low dose of cocaine. None of the subjects in the high-dose condition selected cocaine. Thus, there is little evidence that expectancy effects, related to pre-

conceptions about specific drugs, influenced the results reported in this study.

DISCUSSION

This experiment provided subjects who had ingested either a placebo, low, or high dose of cocaine with the opportunity to aggress against a provocative opponent. During the first block of trials, the fictitious opponent set relatively low shock levels for subjects. Under this low level of provocation, subjects in the high-dose cocaine condition behaved more aggressively than subjects in the placebo condition. The same pattern of results was revealed under medium and high levels of provocation. Thus, subjects in the high-dose condition behaved more aggressively than subjects in the placebo condition irrespective of degree of provocation.

It has been suggested that aggression by cocaine users may simply be the by-product of the compulsive and violent activities necessary to procure the drug (4,6,14,15). The results of this study suggest that this perspective may be in need of revision. Clearly, the aggressive responding exhibited by high-dose cocaine subjects was not influenced by economic or systemic pressures related to the procurement of the drug. Instead, the observed aggression was due to the psychopharmacological effects of cocaine.

It seems plausible that the emphasis placed on the systemic/economic model of cocaine-instigated aggression results from the lack of experimental investigations in the literature. There are several methods available to study the effects of acute dosages of drugs such as cocaine on aggression (e.g., naturalistic observation, self-report, analysis of crime statistics, etc.). However, experimental methodology, in which aggressive behavior following the consumption of cocaine is monitored under highly controlled conditions, would appear to be most suitable for determining whether ingestion of cocaine enhances aggressive responding.

One possible explanation for the aggression observed in the high-dose cocaine condition involves changes in pain perception. Aggressive behavior might be facilitated or inhibited if cocaine altered the perceived painfulness of the shock. According to the results of this study, however, there were no differences in either objective or subjective pain thresholds among the three drug conditions.

The most parsimonious explanation for the current findings is that cocaine-instigated aggression was mediated by changes in arousal (21). There is considerable evidence supporting this interpretation of the results. High-dose cocaine subjects exhibited the greatest physiological arousal on the blood pressure indices. Further, only subjects in the high-dose cocaine condition reported significant changes in subjective highness.

Contrary to the common psychopharmacological perspective that aggression results primarily from toxic paranoia exhibited during chronic cocaine use (4,6), the results of this study demonstrated that a single dose of cocaine, in a nonaddicted population, could instigate the expression of aggression. One must, of course, exercise some caution in generalizing these results to other subject populations or settings. Future research should investigate the influence of cocaine on the aggressive behavior of more experienced or chronic users. It should also be recognized that the experimental situation differed in many ways from the everyday social interactions experienced by cocaine users.

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