Laboratory Validation Study of Drug Evaluation and Classification Program: Ethanol, Cocaine, and Marijuana*

Stephen J. Heishman[1,2,†], Edward G. Singleton[2,3], and Dennis J. Crouch[4]

[1]Addiction Research Center, National Institute on Drug Abuse, Baltimore, Maryland; [2]Department of Psychiatry and Behavioral Sciences, Johns Hopkins University School of Medicine, Baltimore, Maryland; [3]Department of Psychology, Morgan State University, Baltimore, Maryland; and [4]Center for Human Toxicology, University of Utah, Salt Lake City, Utah

Abstract

The Drug Evaluation and Classification (DEC) program is used by police agencies to determine if individuals are behaviorally impaired because of drug use, and, if impaired, to determine the class of drug(s) causing the impairment. Although widely used, the validity of the DEC evaluation has not been rigorously tested. The primary goal of this study was to determine the validity of the variables of the DEC evaluation in predicting whether research volunteers had been administered ethanol, cocaine, or marijuana; a secondary goal was to determine the accuracy of trained police officers (Drug Recognition Examiner, DRE) in detecting whether subjects had been dosed with ethanol, cocaine, or marijuana. Community volunteers (n = 18) with histories of drug use received ethanol (0, 0.28, 0.52 g/kg), cocaine (4, 48, 96 mg/70 kg), and marijuana (0, 1.75, 3.55% THC) in a double-blind, randomized, within-subjects design. A single drug dose or placebo was administered during each of nine experimental sessions, and blood samples were obtained before and periodically after dosing. With the exception of marijuana, plasma drug concentration was at or near the observed maximum during the DEC evaluation. The ability of the DEC evaluation to predict the intake of ethanol, cocaine, or marijuana was optimal when using 17–28 variables from the evaluation. When DREs concluded impairment was due to drugs other than ethanol, their opinions were consistent with toxicology in 44% of cases. These findings suggest that the DEC evaluation can be used to predict accurately acute administration of ethanol, cocaine, or marijuana, and that predictions of drug use may be improved if DREs focused on a subset of variables.

Introduction

Motor vehicle accidents are the leading cause of death in the United States for people aged 1 to 34, and ethanol is a factor in nearly half of traffic fatalities each year (1). Several studies have examined the role of drugs other than ethanol; however, measuring the prevalence rate of drugs in victims of vehicular accidents has yielded inconsistent data. A recent national study of fatally injured drivers from seven geographically diverse states found a prevalence rate of 6.4% for drugs other than ethanol; marijuana (6.7%) and cocaine (5.3%) were the two most prevalent drugs detected (2). In contrast, studies conducted in metropolitan areas or single states typically have reported greater drug prevalence rates among fatally injured drivers, ranging from 19 to 37% for marijuana and from 8 to 20% for cocaine (3–6). Relatively high prevalence rates were also found in subjects stopped for reckless driving; urine drug testing indicated 33% were positive for cannabinoids, 13% were positive for cocaine metabolites, and 12% were positive for both (7). It is important to note that drug prevalence rates do not imply driver impairment; however, most drug-related vehicular accidents presumably involve impaired cognitive or psychomotor abilities. Currently, for drugs other than ethanol, we have few standardized methods for identifying drug-induced behavioral impairment.

The Drug Evaluation and Classification (DEC) program, which was originally developed by the Los Angeles Police Department (LAPD) during the 1970s, grew out of a need to document legally if a driver was impaired because of drug use. The DEC program consists of a standardized evaluation conducted by a trained police officer (Drug Recognition Examiner, DRE[1]) and the toxicological analysis of a biological specimen. The evaluation involves examination of the suspect's appearance, behavior, eyes, performance of four field sobriety tests (FST), vital signs, and questioning of the suspect (8). From the evaluation, the DRE concludes (a) if the suspect is behaviorally impaired, (b) if the impairment is drug-related, and (c) the drug class or combination of classes likely to be causing the impairment. The DEC program uses seven drug classes: central nervous system (CNS) depressants, CNS stimulants, hallucinogens, phencyclidine, narcotic analgesics, inhalants,

* A preliminary report of this study was presented at the 1995 SOFT meeting in Baltimore, Maryland.

† Address correspondence to Stephen J. Heishman, Biology of Dependence Section, NIDA Addiction Research Center, P.O. Box 5180, Baltimore, MD 21221

1 The acronym DRE, as originally developed by the Los Angeles Police Department, referred to Drug Recognition Expert. However, appellate courts have ruled that the term *Expert* is appropriate because the court determines whether a witness is an expert. Challenges have also been raised by the forensic toxicology profession. The International Association of Chiefs of Police has retained the acronym DRE, but now recognizes the terms *Examiner* or *Evaluator* in addition to *Expert* for those officers certified in the DEC program. Drug Recognition Examiner is the title used in this paper.

and cannabis. Results of the toxicological analysis either confirm or rebut the DRE's drug class opinion.

Since the early 1980s, the National Highway Traffic Safety Administration (NHTSA) has overseen the development and implementation of the DEC program, and, in 1987, the International Association of Chiefs of Police (IACP) became the national certifying agency for DREs. The DEC program is currently functional in 29 states and the District of Columbia and has experienced an increase in participating agencies of 300% since 1991 (9). A study of the impact of the DEC program on traffic enforcement and adjudication in 11 DEC jurisdictions in 5 states (10) indicated that the number of impaired driving arrests was unchanged from before to after the DEC program was implemented; however, mean blood alcohol concentration (BAC) of DWI suspects was lower in the year after the program was begun compared with nine agencies without the DEC program. The study also found that DRE cases typically result in a conviction on an impaired driving charge, but that DREs are rarely required to testify in court. In the 1980s, Los Angeles Municipal Courts began accepting expert testimony from DREs (11). Since 1991, courts in Arizona and New York have held that DRE testimony meets the Frye standard and is thus admissable. Courts in Colorado, Florida, and Minnesota have held that DRE testimony is admissable and that the Frye standard is inapplicable (9,12).

Although widely used, the validity of the DEC program has been evaluated under limited laboratory and field conditions. In the only laboratory study to date (13), four DREs from the LAPD evaluated drug-experienced research volunteers who were dosed under double-blind conditions on separate days with d-amphetamine (15 or 30 mg, p.o.), diazepam (15 or 30 mg, p.o.), secobarbital (300 mg, p.o.), and marijuana (1.3% or 2.8% Δ^9-tetrahydrocannabinol [THC], 12 puffs). Results indicated that DREs correctly identified the drug class in 91.7% of cases when subjects were judged impaired, correct identifications were dose dependent, and the DEC evaluation resulted in a very low false-positive rate (1.3%). Several field studies of the DEC program have also indicated that a high percentage of DRE opinions were confirmed by toxicological analysis. A study in Los Angeles reported that when DREs concluded suspects were impaired, any drug was detected in blood in 94% of cases, and the named drug class was confirmed in 79% of cases (11). In the adjudication study discussed previously (10), laboratory tests found any drug in 85% of cases and confirmed drug class opinions in 64% of cases when suspects were judged to be impaired. Finally, a recent study of the Arizona DEC program from 1989 to 1993 indicated that DRE impairment decisions were supported by toxicology in 84% of cases (12).

These studies suggest the DEC program, as used in the field, can accurately determine if an individual has used a drug sometime before the evaluation. However, the validity of the individual variables of the DEC evaluation as predictors of drug intake has not been rigorously examined. Thus, the primary goal of this study was to determine the validity of the DEC evaluation variables in predicting if research volunteers had been administered ethanol, cocaine, or marijuana. A secondary goal was to determine the accuracy of the DREs' evaluations in detecting if subjects had been dosed with ethanol, cocaine, or marijuana. The ultimate goal of this and future studies is to refine the DEC evaluation by determining which variables are best predictors of drug intake across a range of drug classes, thereby aiding the DREs in their decision process.

Methods

Research subjects

Participants were 18 community volunteers (14 men, 4 women) who ranged in age from 23 to 40 years (mean = 30.5). Before the study, subjects were given thorough psychiatric and physical examinations and were interviewed about past and current drug use. Subjects reported a history of alcohol use ranging from 3 to 25 years (mean = 14.6), marijuana use ranging from 7 to 20 years (mean = 14.5), and cocaine use ranging from 1 to 15 years (mean = 6.7). During the 2 weeks before admission, subjects reported using alcohol an average of 4.4 times, marijuana an average of 3.8 times, and cocaine an average of 5.2 times. Sixteen subjects were smokers who averaged 13 cigarettes per day. Subjects provided written informed consent according to guidelines for the protection of human subjects of the Department of Health and Human Services and were paid for their participation.

Drug Recognition Examiners

Twenty-eight DREs (25 men, 3 women) representing eight states participated in the study. All DREs were certified according to IACP standards (14). Certification requires 9 days of classroom instruction on the DEC evaluation, relevant legal issues, behavioral and physiolgical effects of the seven drug classes (see Introduction), and a written examination. Candidates are then required to complete a minimum of 12 DEC evaluations on arrested individuals. They must evaluate persons under the influence of at least three of the seven drug classes, and 75% of evaluations must be corroborated by toxicological analysis. A final written examination must also be passed.

In this study, DREs had no contact with research subjects before or after the DEC evaluation. DREs were told that the study might involve ethanol and five classes of drugs (CNS depressants, CNS stimulants, phencyclidine, narcotic analgesics, and cannabis), that all combinations of ethanol and those drugs could be administered, and that, on some sessions, subjects would receive no active drug. DREs were not permitted to interrogate subjects, except for two questions about physical defects and vision problems. They were also instructed not to converse with other DREs in forming their opinions about behavioral impairment and the drug class causing the impairment. They were observed at all times by the NHTSA supervisor to ensure compliance with these instructions and proper conduct of the modified DEC evaluation.

Drug administration

Ethanol was administered as 80-proof vodka mixed with Sprite® (250-mL constant volume) in doses of 0.28 and 0.52 g/kg. These doses were selected to produce peak BACs of about 30 and 60 mg/dL, respectively. Placebo consisted of 250 mL Sprite® with 1–2 mL of 80-proof vodka floated on the surface. Subjects drank the beverage gradually over a 5-min period. Subjects swallowed two opaque gelatin capsules, which always contained inactive lactose, as they were drinking the beverage.

Cocaine hydrochloride (Mallinkrodt Medical, St. Louis, MO) was administered intranasally in doses of 4, 48, and 96 mg/70 kg. Doses were calculated on the basis of the salt. Lactose was added to the lower doses to maintain a constant amount of 96 mg/70 kg. The 4 mg/70 kg dose was used as a placebo because it has been shown to numb the nasal mucosa but not to produce measurable plasma cocaine levels or subjective and cardiovascular effects (15). Subjects were given the powder on paper and prepared two lines using thin cardboard. When instructed, subjects insufflated one line into each nostril using a straw within 2 min.

Marijuana cigarettes were supplied by the National Institute on Drug Abuse (Rockville, MD) and contained either 0 (placebo), 1.75, or 3.55% THC by weight, equivalent to approximately 0, 16, and 32 mg THC, respectively. Two marijuana cigarettes (total of 16 puffs) were smoked according to a paced procedure: 40-s interpuff interval, ad lib puff duration, 10-s breathhold duration (16). This resulted in complete or nearly complete pyrolysis of each cigarette. Total smoking time was approximately 12 min.

Doses were administered under double-blind conditions according to a randomized, Latin square design. Drug was administered in four forms at each session: beverage, capsules, powder, and cigarettes. For each session, only one drug was active, the others placebo. Drug administration lasted 20 min, and the order was always ethanol, capsules, cocaine, and marijuana, based on time to peak drug effect and duration of behavioral intoxication.

Experimental

Subjects were informed that the purpose of the study was to investigate the effects of commonly abused drugs on behavior, mood, and performance. Subjects were told that they would be observed by law enforcement officials who would attempt to determine if they had received active drug(s). They were instructed not to discuss with the DREs what drug(s) they thought they received. Each subject participated in nine 6-h sessions, which were separated by at least 48 h. Subjects were instructed not to use any illicit drug or alcohol within 24 h of experimental sessions. Subjects reported to the laboratory by 8:00 a.m. and were discharged that same afternoon after drug effects had dissipated. Research staff evaluated subjects to determine physical and mental status. If this evaluation determined that a subject was unable to be tested, the subject was sent home, and that session was rescheduled. If subjects passed the evaluation, they provided an observed urine specimen and submitted to an ethanol breath test using a hand-held Alco-sensor III (Intoximeters, St. Louis, MO). If the breath reading exceeded 0.00%, the subject was sent home and the session rescheduled. Urine drug testing was performed with immunoassays (EMIT® II reagents; Syva Co., San Jose, CA) for amphetamines, barbiturates, benzodiazepines, cocaine metabolites, methadone, opiates, phencyclidine, and cannabinoids. An indwelling catheter was then inserted in a nondominant forearm vein. A battery of measures (vital signs, pupillometry, blood, subjective ratings, and performance) was assessed predrug and at specific postdrug times. The DEC evaluation began 10 min after drug administration ended and lasted about 25 min.

DEC evaluation

The protocol was an abridged version of the DEC evaluation used in law enforcement contexts (8). In this study, DREs did not question subjects about recent drug use, nor did they interrogate subjects to solicit admissions about drug use. DREs recorded their observations on an evaluation form that was developed for the DEC program (8).

The preliminary portion of the evaluation lasted 3–4 min and began with a breath test for BAC using an Alco-sensor IV (Intoximeters). DREs recorded information about physical defects, corrective lenses, appearance of the eyes, and visual impairment. Using a pencil, DREs assessed eye tracking and nystagmus, pupillary size, and condition of eyelids. Pulse was measured while subjects were seated.

The next segment involved examination of the eyes and performance of four FSTs, which lasted 15 min. Subjects' eyes were tested for horizontal gaze nystagmus, vertical nystagmus, and convergence. The FSTs were Romberg Balance (RB), Walk and Turn (WT), One Leg Stand (OLS), and Finger to Nose (FN). The RB assessed body sway and tremor while subjects stood for 30 s with feet together, arms at sides, head tilted back, and eyes closed. The WT test required subjects to take nine heel-to-toe steps along a straight line marked on the floor, turn, and return with nine heel-

Table I. Mean* (Standard Error) Data of 17 Variables from DEC Evaluation that Best Predicted Presence or Absence of Ethanol

	Ethanol dose (g/kg)		
DEC Variable	0	0.28	0.52
Horizontal gaze nystagmus	0.6 (0.2)	2.3 (0.4)	3.2 (0.3)
Breath, normal†	63.0 (0.1)	11.1 (0.1)	5.5 (0.1)
Walk and turn cues	2.1 (0.5)	2.5 (0.9)	3.4 (0.9)
Internal clock	28.1 (1.4)	30.9 (4.3)	29.6 (2.7)
One leg stand aborted†	94.4 (0.03)	100.0 (0.0)	94.4 (0.1)
Oral temperature (°F)	97.6 (0.1)	97.5 (0.1)	97.5 (0.1)
Sum of pulses	199.5 (3.6)	203.9 (5.3)	213.2 (7.4)
Face, normal†	94.4 (0.03)	94.4 (0.1)	88.9 (0.1)
Attitude, relaxed†	7.4 (0.04)	5.6 (0.1)	11.1 (0.1)
Rebound dilation of eyes†	27.8 (0.1)	16.7 (0.1)	11.1 (0.1)
Speech, slurred†	1.9 (0.02)	11.1 (0.1)	27.8 (0.1)
Turn errors†	55.6 (0.1)	50.0 (0.1)	66.7 (0.1)
Breath, cigarette†	3.7 (0.03)	0.0 (0.0)	5.6 (0.1)
Muscle tone, abnormal†	13.0 (0.1)	22.2 (0.1)	44.4 (0.1)
Speech, confused†	9.3 (0.04)	16.7 (0.1)	11.1 (0.1)
One leg stand count†	85.2 (0.1)	88.9 (0.1)	77.8 (0.1)
Walk and turn steps†	94.4 (0.03)	94.4 (0.1)	94.4 (0.1)

* $n = 18$
† Data are percentage of cases exhibiting variable.

to-toe steps. The OLS assessed balance by having subjects stand on one leg, with the other leg elevated in a stiff-leg manner 15 cm off the floor for 30 s. Subjects were given a brief rest between right and left leg testing. In the FN test, subjects stood as in the RB and brought the tip of the index finger of the left or right hand (as instructed) directly to the tip of the nose. DREs then measured pulse, blood pressure, and oral temperature.

The final portion involved further examination of the eyes and lasted about 8 min. Using a hand-held template, DREs estimated the diameter of each pupil to the nearest 0.5 mm under conditions of ambient room light, nearly total darkness, indirect light, and direct light. While illuminating the eyes under direct light from a penlight for 15 s, DREs assessed constriction of the pupils and fluctuation of pupillary diameter. Lastly, DREs measured pulse and assessed muscle tone, attitude, coordination, speech, breath odor, and facial appearance.

DREs retired to an isolated room to decide if subjects were impaired. If a conclusion of impaired was reached, DREs recorded their prediction of the drug class(es), including ethanol, that were causing the impairment. If a conclusion of not impaired was reached, DREs could indicate a non-impairing dose of ethanol or drug and identify the drug class.

Collection and analysis of blood samples

Blood samples were collected on ice in 7-mL Monoject® (Sherwood Medical, St. Louis, MO) tubes containing sodium fluoride and potassium oxalate. Samples were centrifuged, and the plasma was transferred to silanized borosilicate tubes for storage at –20°C. Samples were analyzed at the Center for Human Toxicology (Salt Lake City, UT).

Ethanol

Plasma (0.5 mL) was analyzed for ethanol concentration with the Abbott TDx® (Abbott Diagnostics, Chicago, IL) automated REA® immunoassay (17–19). The TDx® instrument was calibrated from 25 to 300 mg/100 mL ethanol. Each batch of samples contained quality control samples at concentrations of 40, 100, and 250 mg/100 mL. The limit of quantitation (LOQ) was 10 mg/100 mL. Intra- and interassay percent coefficients of variation (%CV) at the three ethanol control concentrations were less than 8.5.

Cocaine

Analysis of cocaine, benzoylecgonine (BZE), and ecgonine methylester (EME) was performed by solid-phase extraction (SPE) and positive-ion chemical-ionization mass spectrometry (20) using 1-mL aliquots of controls, calibrators, and subject plasma. Drug-free plasma was fortified with drug reference solutions to produce calibration curves from 5 to 500 µg/L. Controls were analyzed at 25, 100, and 300 µg/L. Plasma samples suspected of containing concentrations exceeding 500 µg/L were diluted with drug-free plasma, and a 1-mL volume of the dilution was extracted. Deuterium-labeled cocaine, BZE, and EME were used as internal standards. Samples were derivatized with *N*-methyl-*N*-(tert-butyldimethylsilyl)-trifluoroacetamide prior to gas chromatography–mass spectrometry (GC–MS) analysis (21). The analyses were performed using a Finnigan MAT (San Jose, CA) 4500 MS equipped with a 9610 GC, INCOS software, and a CTC A200S (Finnigan MAT) autosampler. The MS was operated in the positive-ion detection mode with a combination of methane and ammonia as the reagent gas. Chromatographic separation was achieved on a 15-m × 0.32-mm DB-5® fused-silica capillary column (J&W Scientific, Folsom, CA). The LOQ for cocaine and metabolites was 5.0 µg/L. For all analytes at 10, 25, 100, and 200 µg/L, the intra- and interassay %CVs were less than 8.8 and from 6.5 to 12.7, respectively.

Marijuana

Analysis of THC and 11-nor-9-carboxy-Δ^9-tetrahydrocannabinol (THCCOOH) was performed by SPE and negative ion chemical ionization MS (22,23) using 1-mL aliquots of controls, calibrators, and subject plasma. Calibrators were prepared by fortifying drug-free plasma with THC and THCCOOH in the concentration range of 0.5 to 100 µg/L. Controls were analyzed at 5 µg/L for THC and 20 µg/L

Table II. Results of Discriminative Function Analysis of 17 Predictor Variables and 5 Best Predictors from DEC Evaluation for Ethanol

Ethanol: 17 variables

Sensitivity	= 94.4
Specificity	= 92.6
False pos.	= 7.4
False neg.	= 5.6
Efficiency	= 93.3

Actual condition	Predicted condition: not dosed with drug	Predicted condition: dosed with drug
not dosed with drug	50	4
dosed with drug	2	34

Ethanol: 5 variables

Sensitivity	= 80.6
Specificity	= 92.6
False pos.	= 7.4
False neg.	= 19.4
Efficiency	= 87.8

Actual condition	Predicted condition: not dosed with drug	Predicted condition: dosed with drug
not dosed with drug	50	4
dosed with drug	7	29

for THCCOOH. Samples suspected of containing concentrations exceeding the calibration curve were diluted with drug-free plasma, and a 1-mL volume of the dilution was extracted. Deuterium-labeled THC and THCCOOH were used as internal standards. The eluant from the SPE was divided into two equal fractions. One fraction was derivatized with trifluoroacetic anhydride for the quantitation of THC. The second fraction, containing THCCOOH, was derivatized with BF_3–methanol and then with trifluoroacetic anhydride. The GC–MS analysis used the instrumentation described previously for cocaine; however, for this analysis, the MS was operated in the negative ion detection mode with methane as the reagent gas. Chromatographic separation was achieved on a 15-m × 0.32-mm DB-1® fused-silica capillary column (J&W Scientific) with a 1-µm film thickness using hydrogen as the carrier gas. The LOQ for THC was 0.5 µg/L and was 1.0 µg/L for THCCOOH. Interassay %CVs at 1.6 µg/L were 8.6 and 7.0 and at 25 µg/L were 9.5 and 1.6 for THC and THCCOOH, respectively.

Data analysis

The DEC examination yielded 100 variables, which were transformed into 76 variables to approximate the DREs' evaluation process. The transformed data from 9 sessions for 18 subjects yielded a data set containing 162 cases. Analysis of plasma samples indicated that two subjects absorbed little or no THC from marijuana cigarettes; both active, but not placebo, marijuana sessions for the two subjects were excluded from all statistical and descriptive analyses. Thus, marijuana data were analyzed for 16 subjects, and there was a total of 158 valid cases.

The transformed data were subjected to three forward stepwise discriminant analyses (24) that reduced the transformed data to subsets of 17–28 variables comprising the best predictors of the presence or absence of each of the three study drugs. These subsets of best-predictor variables for each drug were then subjected to separate discriminant function analyses that predicted and classified whether subjects were dosed or not dosed with each drug. Similar discriminant function analyses were then conducted using only the five best predictors for each drug. The resultant data were interpreted using the following definitions:

- True positive (TP). Subjects were dosed with active drug and identified as dosed.
- True negative (TN). Subjects were not dosed with active drug and identified as not dosed.
- False positive (FP). Subjects were not dosed with active drug and identified as dosed.
- False negative (FN). Subjects were dosed with active drug and identified as not dosed.

The above parameters were then used to calculate the following measures of predictive accuracy of the DEC evaluation (25,26):

- Sensitivity. TP × 100/(TP + FN) = the incidence of TP results in the sample when the evaluation was conducted with dosed subjects (ie., the probability that a dosed subject was identified as dosed).
- Specificity. TN × 100/(TN + FP) = the incidence of TN results in the sample when the evaluation was conducted with non-dosed subjects (i.e., the probability that a non-dosed subject was identified as not dosed).
- False positive rate. FP × 100/(FP + TN) = the percentage of subjects in the sample identified as dosed by the evaluation when they were not dosed.
- False negative rate. FN × 100/(FN + TP) = the percentage of subjects in the sample identified as not dosed when they were dosed.
- Efficiency. (TP + TN) × 100/(TP + TN + FP + FN) = the percentage of all subjects in the sample identified accurately, whether dosed or not dosed.

Table III. Mean* (SE) Data of 17 Variables from DEC Evaluation that Best Predicted Presence or Absence of Cocaine

DEC Variable	Cocaine dose (mg/70 kg) 4	48	96
Sum of blood pressures	198.0 (2.6)	207.7 (4.9)	213.7 (4.6)
Sum of pulses	199.5 (3.6)	210.9 (7.8)	222.1 (6.1)
Oral temperature (°F)	97.6 (0.1)	97.2 (0.2)	97.2 (0.2)
Eyes, normal†	61.1 (0.1)	33.3 (0.1)	44.4 (0.1)
Sum of pupil diameters	39.8 (0.8)	40.8 (1.4)	42.7 (1.4)
Breath, marijuana†	27.8 (0.1)	27.8 (0.1)	16.7 (0.1)
Breath, normal†	63.0 (0.1)	38.9 (0.1)	66.7 (0.1)
Sum of left and right eye range†	23.2 (0.1)	13.9 (0.1)	13.9 (0.1)
Muscle tone, abnormal†	13.0 (0.1)	27.8 (0.1)	44.4 (0.1)
Sway, Romberg balance	1.3 (0.2)	1.0 (0.3)	1.3 (0.3)
Walk and turn steps†	94.4 (0.03)	94.4 (0.1)	94.4 (0.1)
Speech, low volume†	18.5 (0.1)	11.1 (0.1)	16.7 (0.1)
Face, flushed or red†	1.9 (0.02)	11.1 (0.1)	5.6 (0.1)
One leg stand cues	2.5 (0.5)	1.8 (0.4)	1.4 (0.6)
Speech, confused†	9.3 (0.04)	16.7 (0.1)	11.1 (0.1)
Attitude, relaxed†	7.4 (0.04)	5.6 (0.1)	5.6 (0.1)
Rebound dilation of eyes†	27.8 (0.1)	33.3 (0.1)	27.8 (0.1)

* $n = 18$
† Data are percentage of cases exhibiting variable.

Results

Plasma data

During each experimental session, eight blood samples were obtained at predrug baseline and 1-, 16-, 45-, 80-, 110-, 170-, and 260-min postdrug. A complete report of the pharmacokinetic data will be published elsewhere. We report here plasma drug concentration from the 16-min postdrug sample, which was obtained halfway through the DEC evaluation.

Ethanol (0.28 and 0.52 g/kg) produced mean ± standard error (SE) peak plasma concentrations of 24.3 ± 2.2 and 54.4 ± 6.0 mg/dL,

respectively, at 16-min postdrug, which was about 30 min after drinking ended. Ethanol concentration declined over the course of the session, reaching baseline levels at 170- (low dose) and 260-min (high dose) postdrug.

At 16-min postdrug, cocaine (48 and 96 mg/70 kg) produced mean plasma concentrations of 74.7 ± 7.2 and 180.5 ± 17.1 ng/mL, respectively. Placebo cocaine (4 mg/70 kg) did not produce measureable plasma levels of cocaine or cocaine metabolites. Peak cocaine plasma levels were obtained at 45–80 min. At 16 min, BZE concentrations were 95.4 ± 27.7 and 210.7 ± 47.3 ng/mL, and EME levels were 10.8 ± 3.0 and 26.1 ± 6.3 ng/mL for low- and high-dose cocaine, respectively.

Marijuana produced peak plasma concentrations immediately after smoking, which declined at 16 min to 15.4 ± 3.0 and 28.2 ± 4.2 ng/mL for 1.75% and 3.55% THC, respectively. Concentrations of THCCOOH reached maximum concentrations of 18.4 ± 3.6 and 27.7 ± 4.2 ng/mL at 16 min after low and high marijuana doses, respectively.

DEC evaluation

Ethanol

The stepwise discriminant analysis of the 76 transformed variables resulted in a subset of 17 variables that were the best predictors of the presence or absence of ethanol (Canonical $R = .85, p < .001$). Results of the breathalyzer reading were not included in the analysis because it was a direct indicator of ethanol administration. Table I presents these 17 variables in descending order of predictive weight and also presents mean values for each variable for placebo, low, and high ethanol doses. A brief description of these variables follows: presence of horizontal gaze nystagmus, abnormal breath odor, increased errors on WT test, estimation of 30 s during RB test, inability to complete OLS test, low oral temperature, increased sum of three pulse recordings, abnormal facial appearance, relaxed attitude, lack of rebound dilation of the pupils under direct illumination, slurred speech, increased errors on executing the turn on WT test, cigarette breath odor, abnormal muscle tone, confused speech, miscounting during OLS test, and number of steps in WT test.

The discriminant function comprising these 17 variables predicted the presence or absence of ethanol with extremely high accuracy (Table II). The model was equally accurate in predicting the presence (sensitivity) and absence of ethanol (specificity); false positive and false negative rates were very low. The discriminant function using the five best predictive variables (Canonical $R = .77, p < .001$) resulted in less sensitivity and a greater false-negative rate than the 17-variable model (Table II). Specificity and false positive rate were the same for both models, whereas overall predictive efficiency was slightly less with the 5-variable compared with the 17-variable model.

Table IV. Results of Discriminative Function Analysis of 17 Predictor Variables and 5 Best Predictors from DEC Evaluation for Cocaine

Cocaine: 17 variables

Sensitivity	= 88.9
Specificity	= 96.3
False pos.	= 3.7
False neg.	= 11.1
Efficiency	= 93.3

Actual condition	Predicted condition: not dosed with drug	Predicted condition: dosed with drug
not dosed with drug	52	2
dosed with drug	4	32

Cocaine: 5 variables

Sensitivity	= 75.0
Specificity	= 83.3
False pos.	= 16.7
False neg.	= 25.0
Efficiency	= 80.0

Actual condition	Predicted condition: not dosed with drug	Predicted condition: dosed with drug
not dosed with drug	45	9
dosed with drug	9	27

Cocaine

The stepwise discriminant analysis of the 76 transformed variables resulted in a subset of 17 variables that were the best predictors of the presence or absence of cocaine (Canonical $R = .78, p < .001$). Table III presents these 17 variables in descending order of predictive weight and also presents mean values for each variable for placebo, low, and high cocaine doses. A brief description of these variables is as follows: increased sum of systolic and diastolic blood pressure, increased sum of three pulse recordings, low oral temperature, eyes that did not appear normal, sum of the pupillary diameter measures during four illumination conditions, breath odor that was not marijuana-like, abnormal breath odor, sum of the pupillary diameter range of left and right eyes, abnormal muscle tone, decreased body sway during RB test, number of steps in WT test, speech that was not low in volume, facial appearance that was flushed or red, decreased errors on OLS test, confused speech, attitude that was not relaxed, and rebound dilation of the pupils under direct illumination.

The discriminant function comprising these 17 variables predicted the presence or absence of cocaine with high accuracy (Table IV). The model had greater specificity than sensitivity and a very low false-positive rate.

The discriminant function using the five best predictive variables (Canonical $R = .65$, $p < .001$) was less accurate in predicting the presence or absence of cocaine compared with the 17-variable model (Table IV). Decreases were especially evident in sensitivity with increases in false-positive and false-negative rates. As a result, predictive efficiency of the 5-variable function was less than that of the 17-variable model.

Marijuana

The stepwise discriminant analysis of the 76 transformed variables resulted in a subset of 28 variables that were the best predictors of the presence or absence of marijuana (Canonical $R = .92$, $p < .001$). Table V presents these 28 best-predictor variables in descending order of predictive weight and also presents mean values for each variable for placebo, low, and high marijuana doses. A brief description of these variables is as follows: increased sum of three pulse recordings, droopy eyelids, low oral temperature, abnormal speech, lack of rebound dilation of the pupils under direct illumination, sum of the pupillary diameter measures during four illumination conditions, increased sum of systolic and diastolic blood pressure, low volume speech, increased body sway during RB test, incoherent speech, abnormal pupillary reaction to light, eyes that did not appear normal, bloodshot eyes, abnormal muscle tone, miscellaneous abnormal appearance of eyes, abnormal facial appearance, increased eye or body tremors during FN test, increased errors on executing the turn on WT test, less than the complete number of steps in WT test, decreased errors on FN test, increased errors on WT test, marijuana breath, abnormal breath odor, lack of hippus of the pupils under direct illumination, failure of eyes to converge, stale breath odor, cigarette breath odor, and slurred speech.

Table VI shows that the discriminant function comprising these 28 variables predicted the presence or absence of marijuana with extremely high accuracy, resulting in minimal false positive and false negative rates. The discriminant function using the five best predictive variables (Canonical $R = .78$, $p < .001$) resulted in only slightly less sensitivity and specificity compared with the 28-variable model; false positive and false negative rates were low (Table VI). Predictive efficiency was high with both models.

Table V. Mean* (Standard Error) Data of 28 Variables from DEC Evaluation that Best Predicted Presence or Absence of Marijuana

DEC Variable	Marijuana dose (% THC)		
	0	1.75	3.55
Sum of pulses	199.5 (3.6)	244.6 (11.2)	262.5 (9.5)
Eyelids, droopy†	14.8 (0.1)	56.3 (0.2)	50.0 (0.1)
Oral temperature (°F)	97.6 (0.1)	97.5 (0.2)	97.3 (0.3)
Speech, normal†	64.8 (0.1)	25.0 (0.1)	6.3 (0.1)
Rebound dilation of eyes†	27.8 (0.1)	6.3 (0.1)	18.8 (0.1)
Sum of pupil measures	39.8 (0.8)	41.3 (1.0)	42.9 (1.7)
Sum of blood pressures	198.0 (2.6)	205.3 (6.8)	209.0 (6.9)
Speech, low volume†	18.5 (0.1)	25.0 (0.1)	18.8 (0.1)
Sway, Romberg balance	1.3 (0.2)	1.7 (0.4)	2.3 (0.4)
Speech, incoherent†	1.9 (0.02)	6.3 (0.1)	12.5 (0.1)
Pupil reaction to light, abnormal†	33.3 (0.1)	68.8 (0.1)	56.3 (0.1)
Eyes, normal†	61.1 (0.1)	37.5 (0.1)	18.8 (0.1)
Eyes, bloodshot†	1.9 (0.02)	12.5 (0.1)	12.5 (0.1)
Muscle tone, abnormal†	13.0 (0.1)	43.8 (0.1)	43.8 (0.1)
Eyes, other†	3.7 (0.03)	12.5 (0.1)	0.0 (0.0)
Face, normal†	94.4 (0.03)	68.8 (0.1)	87.5 (0.1)
Tremors, finger to nose†	33.3 (0.1)	50.0 (0.3)	37.5 (0.2)
Turn errors†	55.6 (0.1)	62.5 (0.1)	75.0 (0.1)
Walk and turn steps†	94.4 (0.03)	93.8 (0.1)	93.8 (0.1)
Finger to nose cues†	16.7 (0.1)	6.3 (0.1)	18.8 (0.1)
Walk and turn cues	2.1 (0.5)	4.3 (0.9)	5.0 (0.7)
Breath, marijuana†	27.8 (0.1)	31.3 (0.1)	31.3 (0.1)
Breath, normal†	63.0 (0.1)	50.0 (0.1)	37.5 (0.1)
Hippus†	7.4 (0.04)	6.3 (0.1)	6.3 (0.1)
Convergence of eyes†	24.1 (0.1)	31.3 (0.1)	0.0 (0.0)
Breath, stale†	3.7 (0.03)	6.3 (0.1)	25.0 (0.1)
Breath, cigarette†	3.7 (0.03)	6.3 (0.1)	6.3 (0.1)
Speech, slurred†	1.9 (0.02)	6.3 (0.1)	31.3 (0.1)

* $n = 16$
† Data are percentage of cases exhibiting variable.

DRE predictions

Table VII presents drug dose administered, predose urine drug test results, DRE prediction of drug class(es) causing impairment, and consistency of prediction with toxicology using IACP standards for each of the 162 experimental sessions. Table VIII and Figure 1 present summaries of these data regarding DREs' predictions. Table VIII shows the number of impairment and no impairment decisions when placebo or any active drug was administered in the session. For these data, sensitivity and specificity were relatively low, and false positive and false negative rates were high. Figure 1 shows DRE decisions for each drug as a function of dose. Although DREs knew the BAC reading, a substantial number of subjects dosed with active ethanol were judged not impaired; however, there was a dose-related increase in ethanol-induced impairment decisions. DREs concluded that the majority of subjects dosed with cocaine were not impaired. Across the three doses, DREs concluded that subjects were dosed with drugs other than cocaine more than with cocaine. Decisions regarding marijuana showed orderly dose-related trends; predictions of impairment due to marijuana increased and predictions of no impairment decreased with marijuana dose.

To obtain and maintain IACP certification, DREs' drug-class predictions must be consistent with toxicological analysis. Consistency does not require DREs' predictions to correspond exactly to the drugs identified in toxicological tests of body fluids. Under IACP standards (14), the following rules apply: (a) if the DRE concludes a subject is impaired by

only one drug class, toxicology must confirm some drug in that class; (b) if the DRE names two drug classes, toxicology must confirm at least one of them; and (c) if the DRE names three or more drug classes, toxicology must confirm at least two. DREs are not expected to identify all drugs taken by a subject, only those that account for the observed impairment. The rules for consistency treat ethanol as a special case. Because DREs administer a breath test, they know if the subject has ingested ethanol. Thus, if the DRE names ethanol and some other drug class, toxicology must confirm the other class, and if the DRE names ethanol and two other classes, toxicology must confirm at least one of the other classes. IACP standards accept a quantitative or qualitative analysis of blood, urine, saliva, or any body tissue or fluid that produces an identification of a drug or metabolite to confirm the DRE's opinion.

Of the 158 valid DEC examinations, DREs concluded impairment was present in 81 cases, which were compared with toxicology to assess the consistency of DREs' predictions. Toxicology confirmed the presence of a drug class if (a) an active dose of that drug was administered on that session or (b) the predose urine drug test for that session was positive for the drug class. Of the 81 impairment predictions, toxicology was positive for any drug(s) in 75 cases (92.6%). Under IACP standards, DREs' predictions were consistent with toxicology in 41 cases (50.6%). These 41 consistent cases included 9 in which the DRE concluded the subject was impaired by ethanol alone. Because the DRE's breath test provided a priori confirmation of ethanol, an ethanol-only prediction was guaranteed to be consistent. Excluding those 9 cases resulted in 72 predictions that named some nonethanol drug class. The DREs' predictions were consistent with toxicology in 32 cases (44.4%).

Drug metabolites identified in the predose urine tests were responsible, in part, for the consistency of the DREs' opinions (see Table VII). Of the 81 impairment predictions, DREs correctly named the class of drug administered in the experimental session 40 times. That includes 18 cases where the correctly named drug was ethanol. Thus, in 63 cases where the DREs concluded impairment was not due to ethanol, they correctly named the class of drug administered 22 times (34.9%). In 9 other cases, the DREs' prediction was consistent only because it named one or more drug class found in the urine test, which accounted for 29.0% (9 of 31) of the consistent predictions that involved drugs other than ethanol.

Positive predose urine tests for drugs other than cocaine metabolites or cannabinoids had little impact on the DREs' consistency. Of the 158 total cases, 3 were positive for opiates; in each case, the DRE concluded no impairment. Of the 81 impairment predictions, DREs concluded 27 were under the influence of narcotics, depressants, or both, usually in combination with ethanol, stimulants, or marijuana. None of the 27 cases were confirmed by the urine drug test.

Table VI. Results of Discriminative Function Analysis of 28 Predictor Variables and 5 Best Predictors from DEC Evaluation for Marijuana

Marijuana: 28 variables

Sensitivity	=100.0
Specificity	= 98.1
False pos.	= 1.9
False neg.	= 0.0
Efficiency	= 98.8

Actual condition	Predicted condition: not dosed with drug	Predicted condition: dosed with drug
not dosed with drug	53	1
dosed with drug	0	32

Marijuana: 5 variables

Sensitivity	= 90.6
Specificity	= 92.6
False pos.	= 7.4
False neg.	= 9.4
Efficiency	= 91.9

Actual condition	Predicted condition: not dosed with drug	Predicted condition: dosed with drug
not dosed with drug	50	4
dosed with drug	3	29

Discussion

The goals of this study were to determine the validity of the DEC evaluation variables and the accuracy of the DREs' evaluation in predicting whether research volunteers had been administered ethanol, cocaine, or marijuana. Using discriminant function analysis, it was found that 17–28 variables of the DEC evaluation predicted the presence or absence of each of the three drugs with a high degree of sensitivity and specificity and low rates of false-positive and false-negative errors. The five best predictive variables were nearly as accurate as the entire subsets of 17–28 variables. When DREs concluded subjects were impaired by ethanol or drugs or both, their predictions were consistent with toxicological analysis in 51% of cases. When ethanol-only decisions, which were guaranteed to be consistent with toxicology, were excluded, DREs' predictions were consistent in 44.0% of cases.

Criterion-related validation procedures were used to determine the validity of the DEC evaluation. Criterion-related validity refers to accuracy of a test in predicting performance by comparing actual performance with an independent criterion, that the test is

Table VII. Drug Administered, Predrug Urine Test, Drug Recognition Examiner (DRE) Prediction of Drug(s) Causing Impairment, and Consistency of DRE Prediction with Toxicology According to IACP Standards for each Session for 18 Subjects

Subject	Session	Drug dose	Urine drug test	DRE Prediction	Consistency
1	1	Cocaine-placebo	Negative	ni*	na†
	2	Ethanol-placebo	Negative	ni	na
	3	THC‡-placebo	Negative	ni	na
	4	Ethanol-high	Negative	ni	na
	5	Cocaine-high	Negative	ni	na
	6	Ethanol-low	Negative	ni	na
	7	THC-low	Negative	Stim§	No
	8	THC-high	Negative	Stim	No
	9	Cocaine-low	Negative	ni	na
2	1	Ethanol-low	Cann#	Alc‖	Yes
	2	THC-low	Cann	Cann, Stim, Narc**	No
	3	Cocaine-low	Cann	ni	na
	4	Ethanol-placebo	Cann	ni	na
	5	Cocane-placebo	Cann	ni	na
	6	THC-high	Cann	Stim, Cann	Yes
	7	Cocaine-high	Cann	ni	na
	8	THC-placebo	Negative	ni	na
	9	Ethanol-high	Cann	ni	na
3	1	Ethanol-placebo	Negative	ni	na
	2	THC-placebo	CM††	ni	na
	3	Cocaine-placebo	Negative	ni	na
	4	THC-high	CM	Stim	Yes
	5	Ethanol-high	CM	Alc	Yes
	6	THC-low	CM	Cann	Yes
	7	Cocaine-low	CM	ni	na
	8	Cocaine-high	CM	ni	na
	9	Ethanol-low	CM	ni	na
4	1	THC-low	CM	Depr‡‡	No
	2	Ethanol-low	CM	Alc, Cann	No
	3	THC-placebo	CM, Cann	ni	na
	4	Ethanol-placebo	CM, Cann	Cann	Yes
	5	Cocaine-high	CM, Cann	Depr, Cann	Yes
	6	Ethanol-high	CM	Alc	Yes
	7	Cocaine-placebo	CM, Cann	ni	na
	8	Cocaine-low	CM, Cann, Op§§	ni	na
	9	THC-high	CM	Cann, Depr	Yes
5	1	THC-high##	CM	ni	na
	2	Cocaine-high	CM	Cann	No
	3	Ethanol-high	CM	Alc	Yes
	4	THC-low##	CM, Cann	ni	na
	5	Ethanol-low	CM	Alc, Depr	No
	6	THC-placebo	CM	Cann	No
	7	Cocaine-placebo	CM	Stim	Yes
	8	Cocaine-low	CM	Cann	No
	9	Ethanol-placebo	Stim, Cann	Narc	No

* ni = Not impaired
† na = Not applicable. Consistency of DRE prediction applies only when impairment is observed.
‡ THC = Δ^9-tetrahydrocannabinol administered via smoked marijuana.
§ Stim = CNS stimulant.
# Cann = Cannabinoids.
‖ Alc = Alcohol (ethanol).
** Narc = Narcotic.
†† CM = Cocaine metabolites.
‡‡ Depr = CNS depressant.
§§ Op = Opiates.
Session excluded from data analyses because of little or no THC absorption

Table VII continued. Drug Administered, Predrug Urine Test, Drug Recognition Examiner (DRE) Prediction of Drug(s) Causing Impairment, and Consistency of DRE Prediction with Toxicology According to IACP Standards for each Session for 18 Subjects

Subject	Session	Drug dose	Urine drug test	DRE Prediction	Consistency
6	1	Cocaine-high	Negative	Cann*	No
	2	Ethanol-high	Negative	Alc†, Cann, Depr‡	No
	3	THC§-high	Negative	Cann	Yes
	4	Cocaine-low	Negative	Cann, Stim#	Yes
	5	THC-low	Negative	Cann, Depr	Yes
	6	Cocaine-placebo	CM‖	Stim, Depr	Yes
	7	Ethanol-placebo	Negative	Depr	No
	8	Ethanol-low	Negative	Alc, Cann	No
	9	THC-placebo	Negative	Depr, Cann	No
7	1	Ethanol-high	Negative	Alc	Yes
	2	THC-high	Negative	Cann	Yes
	3	Cocaine-high	Negative	Stim	Yes
	4	Ethanol-low	CM	Alc, Stim	Yes
	5	Cocaine-low	CM	ni	na
	6	Ethanol-placebo	CM	Depr, Cann	No
	7	THC-placebo	CM	Depr, Cann	No
	8	THC-low	Negative	Depr, Stim	No
	9	Cocaine-placebo	Negative	ni**	na††
8	1	Ethanol-high	Negative	Alc	Yes
	2	THC-placebo	Negative	ni	na
	3	Cocaine-placebo	CM	ni	na
	4	Cocaine-high	Negative	ni	na
	5	THC-high	CM, Cann	Cann	Yes
	6	THC-low	CM	Depr	No
	7	Cocaine-low	CM	ni	na
	8	Ethanol-low	Negative	ni	na
	9	Ethanol-placebo	CM	ni	na
9	1	Cocaine-high	Negative	Cann	No
	2	Ethanol-placebo	Negative	Narc‡‡, Cann	No
	3	THC-placebo	Negative	ni	na
	4	THC-high	Negative	Stim	No
	5	Ethanol-high	Negative	ni	na
	6	Ethanol-low	CM, Op§§	ni	na
	7	THC-low	CM	ni	na
	8	Cocaine-low	Negative	ni	na
	9	Cocaine-placebo	Negative	ni	na
10	1	Ethanol-placebo	CM	Cann	No
	2	Cocaine-low	CM, Cann	Stim	Yes
	3	Ethanol-low	CM, Cann	ni	na
	4	Cocaine-placebo	CM	ni	na
	5	THC-placebo	CM, Cann	ni	na
	6	Cocaine-high	CM	ni	na
	7	Ethanol-high	CM	Alc	Yes
	8	THC-high	CM, Cann	Cann	Yes
	9	THC-low	CM, Cann	ni	na

* Cann = Cannabinoids.
† Alc = Alcohol (ethanol).
‡ Depr = CNS depressant.
§ THC = Δ^9-tetrahydrocannabinol administered via smoked marijuana.
# Stim = Stimulant.
‖ CM = Cocaine metabolites.
** ni = Not impaired.
†† na = Not applicable. Consistency of DRE prediction applies only when impairment is observed.
‡‡ Narc = Narcotic.
§§ Op = Opiates.

Table VII continued. Drug Administered, Predrug Urine Test, Drug Recognition Examiner (DRE) Prediction of Drug(s) Causing Impairment, and Consistency of DRE Prediction with Toxicology According to IACP Standards for each Session for 18 Subjects

Subject	Session	Drug dose	Urine drug test	DRE Prediction	Consistency
11	1	Ethanol-low	Negative	Alc*, Depr†	No
	2	Cocaine-high	CM‡	Cann§	No
	3	Ethanol-high	Negative	Alc	Yes
	4	Cocaine-low	Negative	Cann, Depr	No
	5	THC#-low§§	Negative	Cann, Depr	Yes
	6	THC-placebo	CM	Depr	No
	7	Cocaine-placebo	CM	Narc, Depr	No
	8	Ethanol-placebo	CM	ni**	na††
	9	THC-high§§	CM	Stim‡‡, Narc	Yes
12	1	THC-placebo	CM	Cann	No
	2	Ethanol-low	CM, Cann	ni	na
	3	THC-low	CM, Cann	Stim, Cann	Yes
	4	Ethanol-placebo	CM	ni	na
	5	Cocaine-placebo	Negative	Stim	No
	6	Ethanol-high	CM	Alc	Yes
	7	THC-high	CM	Cann	Yes
	8	Cocaine-high	CM	ni	na
	9	Cocaine-low	Negative	Cann	No
13	1	THC-placebo	Cann	Cann, Stim	Yes
	2	Cocaine-placebo	CM	Cann	No
	3	Ethanol-placebo	CM	Cann	No
	4	Cocaine-high	CM, Cann	Stim	Yes
	5	THC-high	CM	Cann	Yes
	6	Cocaine-low	Negative	ni	na
	7	Ethanol-low	CM	ni	na
	8	Ethanol-high	Stim, Cann	ni	na
	9	THC-low	CM	Stim, Cann	Yes
14	1	Cocaine-placebo	CM	Cann	No
	2	THC-high	Negative	Cann, Stim	Yes
	3	Ethanol-placebo	Negative	Cann, Stim, Narc	No
	4	Ethanol-high	Negative	Alc, Depr	No
	5	Cocaine-high	CM	ni	na
	6	Cocaine-low	CM	ni	na
	7	Ethanol-low	Negative	Alc, Cann	No
	8	THC-low	Negative	ni	na
	9	THC-placebo	CM	ni	na
15	1	THC-placebo	Negative	Cann	No
	2	Cocaine-placebo	Cann	Cann	Yes
	3	Cocaine-low	Cann	Cann, Depr	Yes
	4	THC-high	Cann	Cann	Yes
	5	Cocaine-high	Cann	ni	na
	6	THC-low	Cann	ni	na
	7	Ethanol-low	Cann	ni	na
	8	Ethanol-placebo	Cann	ni	na
	9	Ethanol-high	Cann	Alc, Cann, Depr	Yes

* Alc = Alcohol (ethanol).
† Depr = Depressant.
‡ CM = Cocaine metabolites.
§ Cann = Cannabinoids.
# THC = Δ^9-tetrahydrocannabinol administered via smoked marijuana.
‖ Narc = Narcotic.
**ni = Not impaired.
††na = Not applicable. Consistency of DRE prediction applies only when impairment is observed.
‡‡Stim = Stimulant.
§§Session excluded from data analyses because of little or no THC absorption.

designed to predict (27). Because the DEC evaluation is used to predict drug intake, drug dose was chosen as the criterion measure in this study. Another criterion that could be used to validate the DEC evaluation is behavioral impairment. However, this would require a behavioral test that was independent of the DEC evaluation and was sensitive to the impairing effects of ethanol, cocaine, and marijuana. Behavioral impairment is typically defined by whatever ability (sensory, psychomotor, cognitive, etc.) is being measured, and it is now clear that drugs differentially affect various neuropsychological processes (28,29). Until a broad range of drugs and doses are tested on the DEC evaluation and independent performance tests under laboratory conditions, it is difficult to assess the validity of the DEC evaluation with respect to behavioral impairment criteria. Such validation is critically needed, however, because the current means of confirming a DRE's prediction of impairment is the presence of parent drug or metabolite in blood or urine, which, with the exception of ethanol, provides little, if any, information concerning behavioral impairment (11,30,31).

The validity of the DEC evaluation was examined by developing mathematical models based on discriminant functions that identified which subsets of variables best predicted whether subjects were dosed with placebo or each active drug. The subsets consisted of 17 variables for ethanol (Table I), 17 variables for cocaine (Table III), and 28 variables for marijuana (Table V). The greater number of variables for marijuana was probably due to marijuana's more pervasive effects. For all three drugs, the discriminative function testing this subset of variables predicted the presence or absence of drug with a high degree of sensitivity and specificity and low false-positive and false-negative rates (Tables II, IV, and VI).

One purpose of developing a predictive model was to reduce the large set of variables generated by the DEC evaluation to the smallest subset of variables that would account for behavioral

Table VII continued. Drug Administered, Predrug Urine Test, Drug Recognition Examiner (DRE) Prediction of Drug(s) Causing Impairment, and Consistency of DRE Prediction with Toxicology According to IACP Standards for each Session for 18 Subjects

Subject	Session	Drug dose	Urine drug test	DRE Prediction	Consistency
16	1	Cocaine-low	Cann*	ni†	na‡
	2	Ethanol-low	CM§, Cann	ni	na
	3	THC#-low	Negative	Narc††, Cann	Yes
	4	Cocaine-placebo	Cann	ni	na
	5	THC-placebo	Cann	ni	na
	6	Ethanol-high	CM, Cann	ni	na
	7	THC-high	Cann	ni	na
	8	Ethanol-placebo	Cann	ni	na
	9	Cocaine-high	CM, Cann	Stim**	Yes
17	1	THC-low	CM, Cann	Depr††	No
	2	Ethanol-high	CM	Depr, Cann	No
	3	THC-high	CM, Cann	ni	na
	4	Ethanol-low	CM, Cann	ni	na
	5	Cocaine-low	Cann	ni	na
	6	Cocaine-placebo	CM, Cann	ni	na
	7	Ethanol-placebo	CM, Narc	ni	na
	8	THC-placebo	CM	ni	na
	9	Cocaine-high	CM, Cann	ni	na
18	1	Cocaine-placebo	Negative	ni	na
	2	THC-low	Negative	Cann	Yes
	3	Cocaine-low	Negative	ni	na
	4	THC-placebo	Negative	ni	na
	5	Ethanol-placebo	Negative	ni	na
	6	THC-high	Negative	Cann	Yes
	7	Cocaine-high	Negative	ni	na
	8	Ethanol-high	CM	ni	na
	9	Ethanol-low	Negative	ni	na

* Cann = Cannabinoids.
† ni = Not impaired.
‡ na = Not applicable. Consistency of DRE prediction applies only when impairment is observed.
§ CM = Cocaine metabolites.
# THC = Δ^9-tetrahydrocannabinol administered via smoked marijuana.
‖ Narc = Narcotic.
**Stim = CNS stimulant.
††Depr = CNS depressant.

differences between conditions of drug and placebo. Because the number of variables in the best-predictor subset was still relatively large (17–28), it seemed desirable to reduce the subset of predictor variables further. If the predictive accuracy of the five best variables were comparable with that of the subset, it would be easier for DREs to focus on five variables rather than the entire subset. For ethanol, the specificity and false-positive rate of the 17-variable model and the 5-variable model were comparable; however, sensitivity was less with the 5-variable model. For cocaine, the 17-variable model was superior to the 5-variable model on all measures of accuracy. For marijuana, the predictive performance of the 28-variable model was comparable with that of the 5-variable model. However, if comparisons are ignored, the overall predictive efficiency of the 5-variable model for each drug ranged from 80 to 92%, indicating a high degree of predictive validity.

The predictor variables reflect some of the known effects of each drug. Interestingly, three of the five best-predictor variables for ethanol (nystagmus, WT, and OLS errors) and cocaine (increased blood pressure and pulse, pupillary dilation) are diagnostic criteria for ethanol and cocaine intoxication according to the *Diagnostic and Statistical Manual of Mental Disorders* of the American Psychiatric Association (32). Ethanol has been shown to increase nystagmus and other eye movements (33,34), and impairment on the WT and OLS tests is consistent with ethanol-induced ataxia and loss of motor coordination (16,35). Increased blood pressure and pulse are indicative of cocaine's cardiovascular effects (36), and increased pupillary diameter has also been reported for cocaine (37). The slight improvement in performance on the RB and OLS tests is consistent with reports of cocaine and other stimulants enhancing psychomotor and attentional abilitites (38,39). The best predictor of marijuana was increased pulse, which is the most reliable physiological sign of acute marijuana use (40,41). Marijuana has also been found to produce pupillary dilation, nystagmus, and transient visual disturbances (37). The marijuana-induced impairment observed in this study on the FSTs and performance impairment reported by others (42) may be related to impaired highway driving caused by marijuana (43).

Table VIII. DREs' Predictions* When Subjects Were Administered Placebo or Any Active Dose of Ethanol, Cocaine, and Marijuana

Sensitivity	= 56.7
Specificity	= 59.3
False pos.	= 40.7
False neg.	= 43.3
Efficiency	= 57.6

Actual condition	Predicted condition: not dosed with drug	Predicted condition: dosed with drug
not dosed with drug	32	22
dosed with drug	45	59

*A prediction of a subject being dosed with a drug was equivalent to a prediction of impairment.

The secondary goal of this study was to determine the accuracy of the DREs' evaluations in detecting if an individual had been dosed with ethanol, cocaine, or marijuana. When DREs judged subjects as impaired, their drug class predictions were consistent with toxicology in half the cases, and when ethanol-only decisions were excluded, consistency fell to 44% of cases. Nearly one-third of nonethanol impairment decisions were consistent because of drug metabolites identified in predose urine specimens. However, it is highly unlikely that this contributed to behavioral impairment because the blood sample obtained at the time of the DEC evaluation for each of these cases tested negative for the parent drug. It is widely recognized that a positive urine drug test does not indicate behavioral impairment (30,31).

The ethanol data indicated that some DREs concluded no impairment in the face of measureable BACs, confirming that low to moderate doses of ethanol were administered. In addition, the fact that an equal number of impairment and no impairment decisions were made for ethanol suggests that these doses represent a threshold for detection of behavioral impairment with the DEC evaluation (see Figure 1).

These data clearly indicate that the variables of the DEC evaluation alone did not permit DREs to predict impairment and drug intake with the consistency that the IACP requires for certification. There were several differences between the controlled laboratory conditions of this study and the field conditions under which DREs normally conduct the DEC evaluation that may have contributed to this outcome:

- In this study, DREs evaluated research subjects who were not dosed with active drug on some sessions. In the field, there is preliminary evidence (e.g., impaired driving, drugs or drug paraphernalia in possession) that makes it more likely that the individual has used drugs, and the DREs are aware of this before conducting the DEC evaluation.
- In the field, the odor of marijuana on a suspect's breath and the presence of crystalline particles in the nostrils are strongly indicative of marijuana and cocaine, respectively. In this study, these easily observable cues were meaningless or possibly misleading, despite the fact that DREs were instructed to ignore them.
- An abbreviated DEC evaluation was used in this study that was different from the standardized test used in the field.
- In the field, DREs attempt to supplement information from the DEC evaluation by interviewing suspects and often receive incriminating statements or confessions of drug use; no such interviewing was permitted in the study.
- Although participating DREs were instructed to be conservative in assessing subjects' impairment, the fact that their conclusions would not lead to prosecution or conviction may have led them to speculate more freely about a subject's dose condition than they would in an actual law enforcement context.

- Although the doses of cocaine and marijuana in this study were moderate to high, suspected drug-impaired drivers encountered by DREs may have used greater drug doses or have been using drugs for a longer period of time than did subjects in this study and thus may exhibit clearer clinical and behavioral signs of impairment.
- In this study, DREs were told that several drug classes and drug combinations might be administered, but only ethanol, cocaine, and marijuana were administered individually.

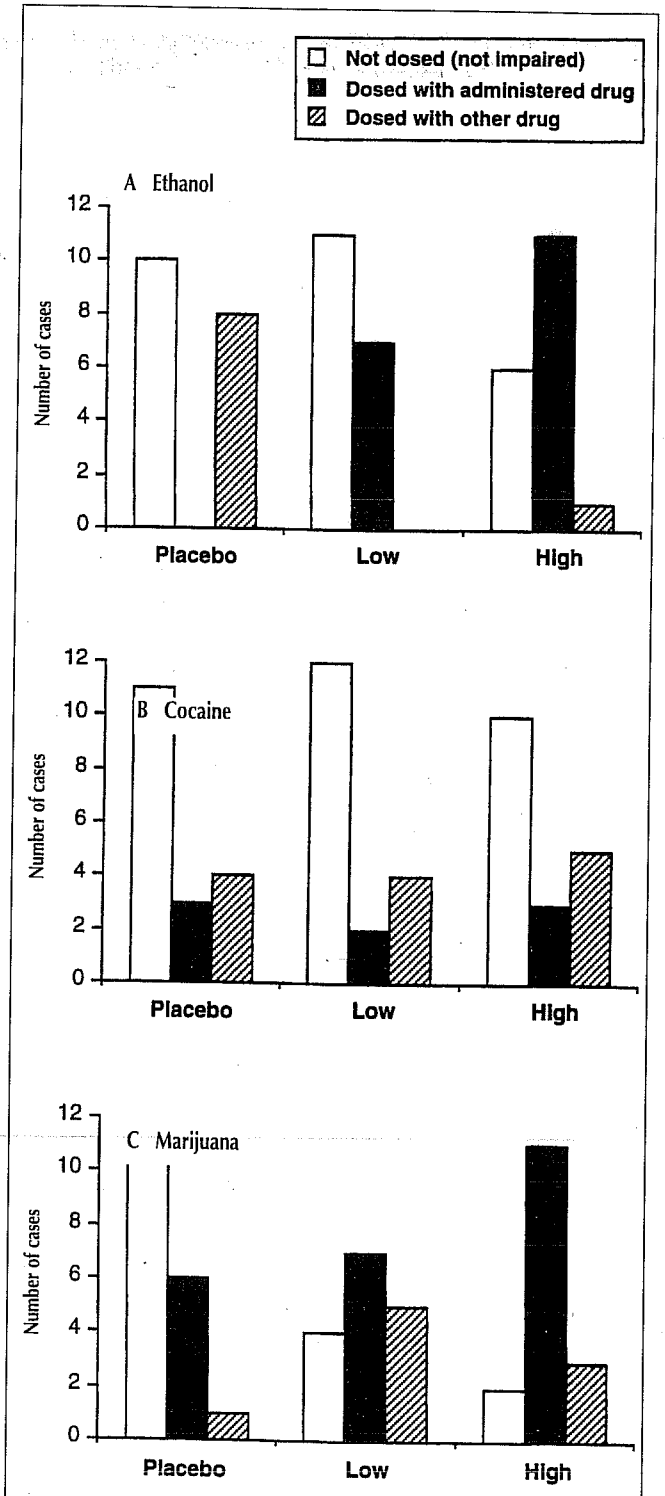

Figure 1. Number of cases in which DRE concluded subject was either not impaired, was impaired and dosed with the administered drug alone or in combination with other drugs, or was impaired and dosed with a drug(s) other than the administered drug as a function of placebo, low, and high doses of (A) ethanol, (B) cocaine, and (C) marijuana. For ethanol and cocaine, $n = 18$; for marijuana, $n = 16$.

The mathematical models predicted the presence of drugs with greater accuracy than did the DREs. This suggests that predictive accuracy would be improved if attention were focused on the best predictors identified by the mathematical models; however, any comparison between the DREs' accuracy and that of the mathematical models should be made cautiously. The intent of this study was not such a comparison, but rather an independent evaluation of the validity of the DEC evaluation itself and the predictive accuracy of the DREs as they used the information from the evaluation to reach their decisions. Given that comparisons are inevitable, however, there are important differences between the decision-making process in humans and that of mathematical models.

Whereas the DREs were limited in their ability to identify the presence of drug for the reasons discussed previously, the statistical models were designed to identify the variables of the DEC evaluation that best predicted the presence or absence of drug. Thus, the mathematical models were strongly biased toward making correct predictions. The complex mathematical calculations on 76 variables required for the predictive models were handled by a computer, which is far superior to humans in its ability to integrate such a vast amount of data and generate an accurate prediction.

In this study, the 162 cases comprised data from 18 research participants who were each evaluated nine times by the same or different DREs. Thus, the results of this study are markedly influenced by the characteristics of the 18 participants, and their data may not generalize to the larger society. However, this is always a limitation of laboratory research with relatively small numbers of subjects. A more field-relevant, but less practical, experimental design would have used 162 different participants and randomly assigned equivalent numbers of participants to each DRE. Such a design would have resulted in a much wider range of responses on the variables of the DEC evaluation, which would have generalized more readily to field situations.

Conclusion

The results of this study lead to several conclusions. With respect to the predictive validity of the DEC evaluation, individual variables of the test can be used to predict accurately acute administration of ethanol, cocaine, or marijuana. This predictive validity was optimal when predictions were made using 17–28 variables from the DEC evaluation. In addition, five of these variables could be used to make predictions that were nearly as accurate. These findings suggest that predictions of impairment and drug use may be improved if DREs focused on a subset of variables associated with each drug class, rather than the entire DEC evaluation.

Acknowledgments

This study was financially supported by NHTSA and the Intramural Research Program of NIDA. We would like to thank Jack Oates, Ted Anderson, and Robin Mayer of NHTSA for their assistance in coordinating the participation of DREs and for critical reading of earlier versions of the manuscript. We also thank Sgt. Ken Clark, Virginia State Police, for serving as on-site supervisor of DREs; Dr. Robert Keenan for providing medical coverage; Richard Taylor for supervising many of the daily aspects of the study; and Joyce Lutz, Anna Francis-Wood, Janeen Nichels, and Diana Lafko for their excellent nursing and technical assistance throughout the study.

References

1. Morbidity and Mortality Weekly Report. Update: Alcohol-related traffic fatalities–United States, 1982–1993. *MMWR* **43:** 861–67 (1994).
2. K.W. Terhune, C.A. Ippolito, D.L. Hendricks, J.G. Michalovic, S.C. Bogema, P. Santinga, R. Blomberg, and D.F. Preusser. The incidence and role of drugs in fatally injured drivers. National Highway Traffic Safety Administration, Report No. DOT HS 808 065. U.S. Department of Transportation, Washington, DC (1992).
3. A.F. Williams, M.A. Peat, D.J. Crouch, J.N. Wells, and B.S. Finkle. Drugs in fatally injured young male drivers. *Public Health Rep.* **100:** 19–25 (1985).
4. C.A. Soderstrom, A.L. Trifillis, B.S. Shankar, W.E. Clark, and R.A. Crowley. Marijuana and alcohol use among 1023 trauma patients. *Arch. Surg.* **123:** 733–37 (1988).
5. R.D. Budd, J.J. Muto, and J.K. Wong. Drugs of abuse found in fatally injured drivers in Los Angeles County. *Drug Alcohol Depend.* **23:** 153–58 (1989).
6. P.M. Marzuk, K. Tardiff, A.C. Leon, M. Stajic, E.B. Morgan, and J.J. Mann. Prevalence of recent cocaine use among motor vehicle fatalities in New York City. *J. Amer. Med. Assoc.* **263:** 250–56 (1990).
7. D. Brookoff, C.S. Cook, C. Williams, and C.S. Mann. Testing reckless drivers for cocaine and marijuana. *New Engl. J. Med.* **331:** 518–22 (1994).
8. National Highway Traffic Safety Administration. Drug evaluation and classification program, briefing paper. U.S. Department of Transportation (1991).
9. Preusser Research Group, Inc. Drug evaluation and classification program: history and growth. U.S. Department of Transportation, National Highway Traffic Safety Administration, Letter Report (1995).
10. D.F. Preusser, R.G. Ulmer, and C.W. Preusser. Evaluation of the impact of the drug evaluation and classification program on enforcement and adjudication. National Highway Traffic Safety Administration, Report No. DOT HS 808 058. U.S. Department of Transportation, Washington, DC (1992).
11. R.P. Compton. Field evaluation of the Los Angeles Police Department drug detection procedure. National Highway Traffic Safety Administration, Report No. DOT HS 807 012. U.S. Department of Transportation, Washington, DC (1986).
12. E.V. Adler and M. Burns. Drug recognition expert (DRE) validation study. Final Report to Governor's Office of Highway Safety, State of Arizona. (1994).
13. G.E. Bigelow, W.E. Bickel, J.D. Roache, I.A. Liebson, and P. Nowowieski. Identifying types of drug intoxication: Laboratory evaluation of a subject-examination procedure. National Highway Traffic Safety Administration, Report No. DOT HS 806 753. U.S. Department of Transportation, Washington, DC (1985).
14. International Association of Chiefs of Police. National Standards of the Drug Evaluation and Classification Program. IACP Advisory Committee on Highway Safety (1993).
15. M.W. Fischman and C.R. Schuster. Cocaine effects in sleep-deprived humans. *Psychopharmacology* **72:** 1–8 (1980).
16. S.J. Heishman, M.L. Stitzer, and G.E. Bigelow. Alcohol and marijuana: comparative dose effect profiles in humans. *Pharmacol. Biochem. Behav.* **31:** 649–55 (1988).
17. Y.H. Caplan and B. Levine. The analysis of ethanol in serum, blood, and urine: A comparison of the TDx REA ethanol assay with gas chromatography. *J. Anal. Toxicol.* **10:** 49–52 (1986).
18. Abbott Laboratories Diagnostic Division, Assay Manual: Ethanol. 1987, pp 22.33–22.41.
19. Abbott Laboratories Diagnostic Division, Assay Manual: Ethanol. 1995, pp 1–6.
20. D.J. Crouch, M.E. Alburges, A.C. Spanbauer, D.E. Rollins, D.E. Moody, and A.A. Chasin. Analysis of cocaine and its metabolites from biological specimens using solid-phase extraction and positive ion chemical ionization mass spectrometry. *J. Anal. Toxicol.* **19:** 352–58 (1995).
21. M.R. Harkey, G.L. Henderson, and C. Zhou. Simultaneous quantitation of cocaine and its major metabolites in human hair by gas chromatography/chemical ionization mass spectrometry. *J. Anal. Toxicol.* **15:** 260–65 (1991).
22. R.L. Foltz, K.M. McGinnis and D.M. Chinn. Quantitative measurement of delta-9-tetrahydrocannabinol in physiological specimens using capillary column gas chromatography negative ion mass spectrometry. *Biomedical Mass Spec.* **10:** 316–23 (1983).
23. United Chemical Technologies, Inc. Clean Screen® Extraction Column Applications Manual. Horsham, PA, 1994, p 40.
24. E.J. Pedhazur. Mulitple *Regression in Behavioral Research.* Holt, Rinehart and Winston, New York, NY, 1982.
25. H.I. Kaplan and B.J. Sadock. Epidemiology, Biostatistics, and Social Psychiatry, 6th ed. Williams and Wilkins, Baltimore, 1991, pp 133–42.
26. S.D. Ferrara, L. Tedeschi, G. Frison, G. Brusini, F. Castagna, B. Bernardelli, and D. Soregaroli. Drugs-of-abuse testing in urine: statistical approach and experimental comparison of immunochemical and chromatographic techniques. *J. Anal. Toxicol.* **18:** 278–91 (1994).
27. A. Anastasi. Psychological Testing, 6th ed. Macmillan, New York, NY, 1988.
28. I. Grant. Alcohol and the brain: neuropsychological correlates. *J. Consult. Clin. Psychol.* **55:** 310–24 (1987).
29. I. Hindmarch. Psychomotor function and psychoactive drugs. *Br. J. Clin. Pharmacol.* **10:** 189–209 (1980).
30. Consensus Development Panel. Drug concentrations and driving impairment. *J. Amer. Med. Assoc.* **254:** 2618–21 (1985).
31. Scientific consensus conference: Clinical pharmacologic implications of urine screening for illicit substances of abuse. American Society for Clinical Pharmacology and Therapeutics, San Diego, CA, 1988.
32. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders,* 4th ed. American Psychiatric Association, Washington, DC, 1994.
33. M.J. Cohen, S.L. Schandler, and B.D. Naliboff. Psychophysiological measures from intoxicated and detoxified alcoholics. *J. Stud. Alcohol* **44:** 271–82 (1983).

34. V. Jantti, A.H. Lang, E. Keskinen, I. Lehtinen, and A. Pakkanen. Acute effects of intravenously given alcohol on saccadic eye movements and subjective evaluations of intoxication. *Psychopharmacology* **79:** 251–55 (1983).
35. S.E. Lukas, B.W. Lex, J.P. Slater, N.E. Greenwald, and J.H. Mendelson. A microanalysis of ethanol-induced disruption of body sway and psychomotor performance in women. *Psychopharmacology* **98**: 169–75 (1989).
36. K.L. Preston, J.T. Sullivan, P. Berger, and G.E. Bigelow. Effects of cocaine alone and in combination with mazindol in human cocaine abusers. *J. Pharmacol. Exp. Ther.* **267:** 296–307 (1993).
37. N.J. McLane and D.M. Carroll. Ocular manifestations of drug abuse. *Surv. Ophthalmol.* **30:** 298–313 (1986).
38. B. Weiss and V.G. Laties. Enhancement of human performance by caffeine and the amphetamines. *Pharmacol. Rev.* **14:** 1–36 (1962).
39. S.T. Higgins, W.K. Bickel, J.R. Hughes, M. Lynn, M.A. Capeless, and J.W. Fenwick. Effects of intranasal cocaine on human learning, performance and physiology. *Psychopharmacology* **102:** 451–58 (1990).
40. S.J. Heishman, M.L. Stitzer, and J.E. Yingling. Effects of tetrahydrocannabinol content on marijuana smoking behavior, subjective reports, and performance. *Pharmacol. Biochem. Behav.* **34:** 173–79 (1989).
41. T.H. Kelly, R.W. Foltin, and M.W. Fischman. Effects of smoked marijuana on heart rate, drug ratings and task performance by humans. *Behav. Pharmacol.* **4:** 167–78 (1993).
42. L.D. Chait and J. Pierri. Effects of smoked marijuana on human performance: A critical review. In *Marijuana/cannabinoids: Neurobiology and neurophysiology.* L. Murphy and A. Bartke, Eds., CRC Press, Boca Raton, FL, 1992, pp 387–423.
43. H.W.J. Robbe. *Influence of marijuana on driving.* University of Limburg Press, Maastricht, 1994.

Manuscript received March 26, 1996;
revision received May 16, 1996.