

# LABORATORY IDENTIFICATION OF DRUG USE BASED ON OBSERVABLE SIGNS AND SYMPTOMS

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## Abstract

The heuristics of the Drug Evaluation and Classification Program (DECP) were evaluated for detecting drug impairment and identifying the type of the impairing drug. A quantitative model, based on stepwise logistic regression of systematic observations made by Drug Evaluation Experts (DREs), provided higher levels of drug identification, and lower rates of errors than the DREs. For impairments caused by cannabis, alprazolam, and amphetamine, the model yielded sensitivity levels greater than 60% and specificity levels greater than 90 percent. For codeine, with a specificity of nearly 90% the sensitivity was only 20%. The regression models' Phi correlations with the actual drug dosing were three times as high as the DREs' correlations with respect to cannabis, twice as high with respect to alprazolam, equal (and poor) with respect to codeine, and over 50 times as high with respect to amphetamine (where the DREs performance was worse than chance). In conclusion, the DECP is a useful tool for drug detection and identification, though to be effective its training procedures should focus on better integration of information from signs and symptoms indicative of drug impairment.

## Introduction

The Drug Evaluation and Classification Program (DECP) is used to train police officers to become Drug Recognition Experts (DREs) who can (a) detect impairment from drugs, and (b) identify the type of drug as a CNS stimulant, a CNS depressant, a narcotic/analgesics, phencyclidine (PCP), cannabis, a hallucinogen or an inhalant. The complete evaluation is a 12-step procedure that includes an evaluation of the subject's general appearance, a structured set of examinations of vital signs and symptoms, several tests of the pupils' responses to light, psychophysical tests, and an interview. Previous evaluations of the validity of the DECP suffered from either lack of methodological controls, or from very limited data (Adler and Burns, 1994; Bigelow et al. 1985; Compton, 1986; Heishman et al., 1996). This study evaluated the DECP's potential for detecting and identifying drug impairment on the basis of systematic data collection by DREs. Four drugs were used in this study: Cannabis, alprazolam (a depressant), codeine (a narcotic analgesic), and amphetamine (a stimulant). The validity of the DECP was tested by stepwise logistic regression functions that related drug impairment to specific signs and symptoms noted on the DECP forms. In a separate study the validity of the DREs' conclusions was assessed by measuring their sensitivity (true positives) and specificity (correct rejections) levels. Since the scientific validity of the DECP is based on the physical evidence, it was important to see to what extent the physical signs and symptoms by themselves can account for specific drug impairments.

## Method

All subjects were self-admitted regular users of the study drugs, in good health (based on a blood analysis and examination of vital signs), and provided a written informed consent. Each subject was individually tested, and each testing session was preceded by a medical evaluation and test for drugs. The testing session started with a pre-dosing test battery consisting of cognitive performance tests and tests of oculo-motor control. After the tests' completion the subject was given one more examination of vital signs, and if all signs were normal, then the subject was dosed by a nurse under a doctor's supervision. The DREs were told that the subjects may be under the influence of none, one, or a combination of two or more drugs, and that the drugs could belong to any of the categories they were trained to identify except hallucinogens and inhalants. The DRE evaluations of drug impairment followed the DECP guidelines except that no interview or any discussions with the subjects were allowed. The evaluation included examination of vital signs, psychophysical tests for motor control, nystagmus, and pupil response to light. A detailed description of the DECP testing procedure is provided elsewhere (Kosnoski, et al., 1998; NHTSA, 1991). Each subject was tested on six sessions, separated from each other by at least 48 hours. Each subject was orally administered each of the three drug levels (placebo, low dose and high dose) twice. The order of the dose levels was counterbalanced between subjects. The total number of sessions with each drug and each level of each drug varied from a low of 23 sessions to a high of 28 sessions, for a total of 302 sessions.

## Results and Discussion

**Cannabis** dosing was significantly associated with bloodshot eyes (much more likely to have bloodshot eyes than placebo-dosed group), slowed pupil reaction to light, larger pupil size both in darkness and under direct light, and an elevated pulse rate that dropped in the course of the session. Stepwise logistic regression analyses (without the psychophysical tests that did not add any explanatory power) yielded three significant variables - average pulse rate, pupil reaction to light, and pupil size under direct light. The strength of the regression model was determined by the % of concordant pairs, and by the sensitivity and specificity that it can provide. In the present analysis, to be conservative, instead of using one cut-off level of  $P\text{-hat} = .5$ , two cut-off levels were used; with  $P\text{-hat} \geq .7$  classified as not drugged,  $.3 < P\text{-hat} < .7$  classified as undecided, and  $P\text{-hat} < .3$  as drugged. While this approach may reduce the sensitivity of the test (letting some people pass as questionable), it does reduce the number of false alarms. The regression yielded the frequencies listed in Table 1.

This function provides the following levels of performance for cannabis identification:

Sensitivity =  $24/49 = 0.49$     Specificity =  $77/100 = 0.77$     Concordance = 86.7%  
False Alarms =  $2/100 = 0.02$     Misses =  $12/49 = 0.24$     Discordance = 12.9% (ties=0.4%)

In summary, the three variables - pupil diameter in direct light, reaction to light, and average pulse rate - provide a relatively good prediction of cannabis dosing, with a very low rate of false alarms, and approximately 50% rate of correct drug impairments. Furthermore, this good performance came with an acceptable rate (23%) of undecided cases.

**Alprazolam**-dosed subjects performed significantly worse on the one-leg-stand (total score of 1.65 for placebo vs. 4.61 for alprazolam) ( $t = -6.62$ ,  $df = 73.3$ ,  $p < .001$ ). On the Romberg balance test their average sway was twice as large as in the placebo-dosed condition (1.3 degrees for placebo vs. 2.8 degrees for alprazolam) ( $t = -3.33$ ,  $df = 56.8$ ,  $p = .002$ ); in the walk-and-turn test they had more points deducted (1.71 vs 4.04,  $t = -6.55$ ,  $df = 69.3$ ,  $p < .001$ ); and they lost more

points in the finger-to-Nose test (1.23 vs 1.90,  $t=-2.43$ ,  $df = 72.9$ ,  $p= .017$ ). Subjects dosed with alprazolam were more likely to have slowed or no pupil reaction to light was observed (Chi Square = 10.14,  $p= .006$ ), and were more likely to have nystagmus (Chi Square = 10.87,  $p= .001$ ) than under the placebo condition. Pupil size both in the dark and under direct light was slightly but significantly smaller in the alprazolam condition than in the placebo condition [6.30 mm vs. 6.88 mm ( $t= 2.98$ ,  $df =147$ ,  $p= .003$ ), and 3.17 mm vs. 3.57 mm. ( $t=2.73$ ,  $df =147$ ,  $p= .007$ ), respectively]. Pulse rate was higher under alprazolam dosing [75.5 beats/minute vs. 67.9 beats/min in the placebo condition ( $t=-5.24$ ,  $df = 149$ ,  $p< .001$ )]. Temperature in the placebo condition was significantly higher than in the alprazolam-dosed condition (97.6 vs. 96.9;  $t=5.54$ ,  $df = 148$ ,  $p= < .001$ ).

Table 1. Allocation of subjects to impaired by a specific drug vs. unimpaired (or impaired by other drugs), using a logistic regression based on drug-dosed and placebo-dosed subjects.

<b>Cannabis</b>				
Predicted Condition				
True Condition	Unimpaired	Cannabis-Impaired	Undecided	Total
Placebo	77	2	21	100
Cannabis	12	24	13	49
Total	89	26	34	149
<b>Alprazolam</b>				
Predicted Condition				
True Condition	Unimpaired	Alprazolam-Impaired	Undecided	Total
Placebo	82	5	10	97
Alprazolam	10	27	9	46
Total	92	32	19	143
<b>Codeine</b>				
Predicted Condition				
True Condition	Unimpaired	Narcotic/Anal. Impaired	Undecided	Total
Placebo	53	1	43	97
Codeine	1	14	32	47
Total	54	15	75	144
<b>Amphetamine</b>				
Predicted Condition				
True Condition	Unimpaired	Stimulant-Impaired	Undecided	Total
Placebo	75	4	18	97
Amphetamine	7	23	18	48
Total	82	27	36	145

Stepwise logistic regression analysis yielded five significant measures: convergence, average pulse rate, temperature, horizontal gaze nystagmus and the walk-and-turn test. With  $.3 < P\text{-hat} < .7$  classified as ‘undecided’, the regression yielded the frequencies listed in Table 1 and the following levels of performance.

Sensitivity =  $27/46 = 0.59$     Specificity =  $82/97 = 0.85$     Concordance = 92.1%  
 False Alarms =  $5/97 = 0.05$     Misses =  $10/46 = 0.22$     Discordance = 7.8%

In summary, the five variables – Convergence, Avg. Pulse rate, Temperature, HGN-sum, and Walk-and-turn – provide quite a good prediction of alprazolam dosing, with a very low rate of false alarms (5%), and a good rate of correct identifications of impairment (59%). Furthermore this good performance came with a fairly low rate (13%) of undecided cases.

**Codeine** dosing was significantly associated only with a slightly smaller pupil size than in the placebo condition. This was true both in the dark (6.28 mm vs. 6.88 mm,  $t=2.78$ ,  $df=74.5$ ,  $p=.007$ ), and in direct illumination (3.08 mm vs. 3.57 mm,  $t=3.35$ ,  $df=148$ ,  $p=.001$ ). Temperature in the placebo condition was very slightly but significantly higher than in the codeine-dosed condition (97.6 vs. 97.4;  $t=2.40$ ,  $df=126.6$ ,  $p=.018$ ).

Stepwise logistic regression analyses (without the psychophysical tests that did not add any explanatory power) yielded only two predictive measures: pupil diameter in direct light, and temperature. The regression yielded the frequencies listed in Table 1, and the following levels of performance:

Sensitivity =  $14/47 = 0.30$       Specificity =  $53/97 = 0.53$       Concordance = 67.1%  
False Alarms =  $1/97 = 0.01$       Misses =  $1/46 = 0.02$       Discordance = 30.8%

In summary, the two variables – pupil diameter in direct light and temperature – are not very sensitive to codeine dosing. Even with .3 and .7 cutoff levels (i.e., .31-.69 range as justification for a retest), we obtained only a moderate rate (30%) of correct identification of codeine impairment (though we still obtained only a 2% rate of false alarms). These acceptable rates of performance came at a price of approximately 50% rate of ‘undecided’ cases (i.e. in need of a retest).

**Amphetamine** dosing was significantly associated with slightly poorer convergence, but with marked increase in pulse rate (that remained elevated throughout the session), and a marked increase in systolic and diastolic blood pressure. Convergence was significantly poorer in the amphetamine-dosed condition than in the placebo-dosed condition (3.4 vs. 3.0,  $t=-2.74$ ,  $df=130.6$ ,  $p=.007$ ). Pulse rate under amphetamine dosing remained high throughout the evaluation session. Average pulse rate under amphetamine dosing was 80.8 beats/minute, whereas under placebo dosing it was 67.9 beats/min. ( $t=-9.25$ ,  $df=151$ ,  $p<.001$ ). Both systolic blood pressure was high relative to the placebo condition (128 vs. 119;  $t=4.25$ ,  $df=151$ ,  $p<.001$ ), and diastolic blood pressure was higher (80 vs. 74;  $t=4.14$ ,  $df=151$ ,  $p<.001$ ). Stepwise logistic regression analyses (with or without the results on the four psychophysical tests) yielded only two significant predictors: average pulse rate and the total blood pressure. When we classified all cases with  $.3 < P\text{-hat} < .7$  as ‘undecided’, the regression yielded the frequencies listed in Table 1, and the following levels of performance:

Sensitivity =  $23/48 = 0.48$       Specificity =  $75/97 = 0.77$       Concordance = 90.7%  
False Alarms =  $4/97 = 0.04$       Misses =  $7/48 = 0.15$       Discordance = 9.1% (ties = 0.2%).

In summary, the two variables – average pulse rate and total blood pressure - provide a good prediction of amphetamine dosing, with a very low rate of false alarms (4%), and moderate rate (48%) of correct identifications. This comes at a cost of 25% of undecided cases that must be reevaluated.

**The DECP diagnostic guidelines** are summarized in the form of a matrix that specifies which signs and symptoms should be associated with each of the drug categories. The comparisons between the DECP guidelines and the effects obtained in this validation study are summarized in

Table 2. The table contains the average score obtained for each drug for each of the measures evaluated, and an indication of whether or not that average score was significantly different from the placebo condition (based on ANOVAs and Dunnett’s post-hoc tests for interval variables, and Chi Square tests for categorical measures). Starred entries indicate that the mean for that drug is significantly different (at  $p < .05$ ) from the mean in the placebo condition. The ‘Up’, ‘Dn’, and ‘Yes’ codes next to some of the entries are the drug impairment indicators noted in the DECP matrix. They refer to scores that are expected to be up (Up) relative to no impairment or down (Dn) relative to no impairment. When a symptom is simply supposed to be present (such as nystagmus) it is denoted by a ‘yes’. Shaded cells indicate an agreement between the DRE matrix and the results obtained here.

Table 2. Mean effects of each drug on ocular and vital signs (see text for explanations).

Measure	Cannabis	Alprazolam	Codeine	Amphetamine	Placebo	ANOVA
Nystagmus (Score)	2.78	3.80* Yes	2.39	2.51	2.62	F(4,295)=8.11, $p < .001$
Pupil Diam (in dark)	7.37* Up	6.30*	6.28* Dn	6.89 Up	6.88	F(4,290)=9.48, $p < .001$
Pupil Diam (in light)	4.13* Up	3.17*	3.08* Dn	3.73 Up	3.57	F(4,293)=12.45, $p < .001$
Pulse Average	84.8* Up	75.5* Dn	66.2	80.8* Up	67.9	F(4,295)=41.81, $p < .001$
Blood Pres. (Systolic)	118.3 Up	117.1 Dn	123.1 Dn	128.4* Up	119.7	F(4,295)=8.86, $p < .001$
Blood Pres. (Diastolic)	72.7 Up	75.1 Dn	72.5 Dn	80.3* Up	73.8	F(4,295)=7.13, $p < .001$
Blood Pres. (Syst+Dias)	191.1 Up	192.2 Dn	195.6 Dn	208.6* Up	193.5	F(4,295)=10.24, $p < .001$
Temperature	97.5	97.0*	97.4 Dn	97.7 Up	97.6	F(4,294)=10.66, $p < .001$
RT to light (% slowed)	44.9*	44.9* Yes	31.3? Yes	23.5 Yes	20.8	Chi <sup>2</sup> =15.13, $p = .004$
Converg. %impaired	71.4* Yes	77.6* Yes	53.1	47.1	59.8	Chi <sup>2</sup> =13.44, $p = .009$

\* Significantly different from the placebo condition, based on Dunnett test with  $p < .05$ .

+ An analysis conducted on the dichotomous variable HGN revealed a similar effect

? With no post hoc tests for Chi Square, only most extreme differences from placebo are starred.

From the table above it can be seen that there is a partial agreement between the results obtained in this controlled drug dosing environment and the DECP matrix. Of the 29 expected impairment signs (indicated by ‘Up’, ‘Dn’, or ‘Yes’), 13 were confirmed in this study (starred and shaded). Of the 16 signs that were not confirmed, 15 were not significantly different from the placebo condition; one (Pulse Average) was significantly different from the placebo but in the opposite-than-expected direction. Four were significantly different from the placebo but not predicted to be so.

## **Conclusions and Recommendations**

This validation study demonstrate that the DECP is useful for detecting drug-impaired people, and identifying three of the four drug categories evaluated: cannabis, depressants, and stimulants. The DECP guidelines for data interpretation and drug identification are not totally consistent with the regression models that yielded the high levels of drug identification. One benefit of the quantitative models, is that their decision cutoff levels can be adjusted to different –socially acceptable – rates of false identifications of drug impairments (false alarms), without sacrificing the sensitivity of the identification. Thus, with the exception of codeine, the models derived for cannabis, alprazolam, and amphetamine can be very useful to the DREs.

For the most part, the signs and symptoms that were significantly associated with each of the drugs were consistent with those indicated by the DECP, thus validating the DECP. The specific consistent associations (all relative to the placebo conditions) were enlarged pupil, raised pulse rate, and lack of convergence for cannabis impairment; horizontal gaze nystagmus, slowed pupil reaction, and lack of convergence for alprazolam (depressant) impairment; constricted pupil for codeine (narcotic/analgesic) impairment; and raised pulse rate and blood pressure for amphetamine (stimulant) impairment. On the other hand the study DID NOT confirm some of the signs and symptoms expected from the DECP. These included raised blood pressure for cannabis impairment, low pulse rate and low blood pressure for alprazolam impairment, low blood pressure, low temperature, and slowed pupil reaction for codeine impairment, and enlarged pupils, raised temperature, and slowed pupil reaction for amphetamine impairment.

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