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Marijuana and Alcohol
A Driver Performance Study

California Dept. of Justice, Sacramento

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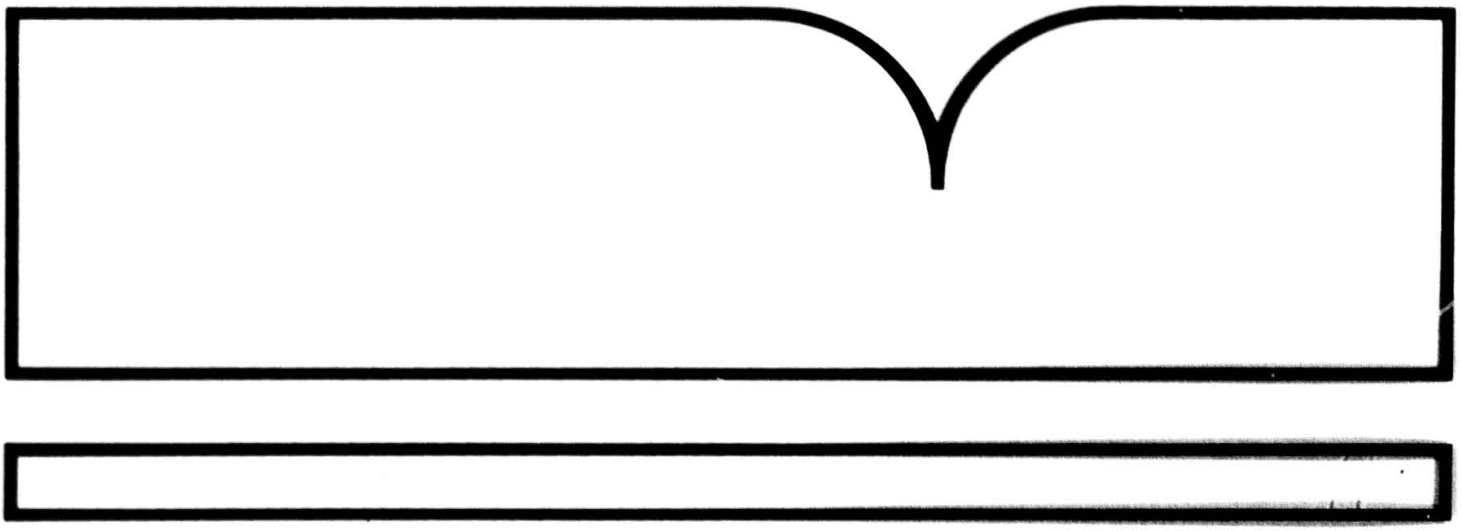

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16. Abstract (Limit: 200 words)

Approximately 80 volunteers male marijuana and alcohol users received one of four experimental treatments: (1) marijuana, (2) alcohol, (3) marijuana and alcohol, or (4) double placebo. After consumption, each subject drove a vehicle over a test coure which simulated a number of real-world driving conditions. Four post-drug runs were involved, separated by one-hour intervals. The subject's performance was rated by an in-car examiner, ourside observers, and computerized vehicle measurements. Blood and urine specimens were extracted after each run to establish levels of tetrahydrocannabinol (THC), serum carboxy, and alcohol. A variety of multivariate statistical techniques were applied in evaluating treatment effects. Both marijuana and alcohol had significant effects on driving performance, and the effects were particularly detrimental under the both-drugs treatment. The effects of marijuana were more rapid than those of alcohol and somewhat less severe for most tasks.

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MARIJUANA AND ALCOHOL: A DRIVER PERFORMANCE STUDY

- A FINAL REPORT -

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CALIFORNIA DEPARTMENT OF JUSTICE
OFFICE OF THE ATTORNEY GENERAL
SACRAMENTO, CALIFORNIA

SEPTEMBER 1986

CALIFORNIA
OFFICE OF TRAFFIC SAFETY
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PREFACE

This is the final report of the MARIJUANA and ALCOHOL DRIVER PERFORMANCE STUDY conducted as part of the Project (#087902), funded by the Office of Traffic Safety, entitled: FORENSIC PROCEDURES FOR THE PRESENCE OF MARIJUANA IN BLOOD. This performance study was conducted between June and September 1981.

The reason that publication of this report was delayed well beyond the end of the project in June 1984 was because of the loss of critical staff, and consultants, who were primarily responsible for the monumental task of analyzing the large amount of data generated, and preparing the final results and conclusions of this publication.

Publication of this report would not have been possible without the dedicated efforts of Raymond Peck, of the Department of Motor Vehicles, who is singly responsible for completing the detailed and complex statistical analyses and preparing the final results, conclusions and discussion for publication. The computerized data reduction and computer runs were performed by Dr. Neal Grossen of the Research Consulting Corporation. Dr. Grossen was also responsible for devising the factor analysis strategy used to identify the relevant performance dimensions.

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Dr. James Ketchum M.D., Private Consultant

Neal Grossen, Ph.D., Foundation of Calif. State University, Sacramento, CA.

The Bureau of Forensic Services further acknowledges the assistance and support given by all Bureau personnel who participated in the planning, organization, and implementation of the various phases of this project.

Credit is also due to the many student assistants whose hard work and dedication contributed greatly towards the successful completion of the Marijuana and Alcohol Driver Performance Study which was a major part of this project.

EXECUTIVE SUMMARY

This paper summarizes Phase II of an effort to characterize and quantify the effects of marijuana use, alone and in combination with alcohol, on driving performance. An earlier study (Department of Justice [DOJ] Incidence Study), funded by the Office of Traffic Safety, indicated that Delta 9-THC was present in a significant proportion of the submitted blood samples drawn from California drivers detained by Highway Patrol Officers because of ostensibly impaired driving performance (Zimmerman, Bager, Soares, Hollister and Reeve, 1983). Phase I of this grant suggested that volunteers given ad lib doses of marijuana by inhalation were subsequently considered impaired when required to perform the standard field sobriety tests (Reeve, Grant Robertson, Gillespie and Hollister, 1983). These impairment ratings persisted for up to three hours after smoking and were associated with mean hemolyzed blood concentrations of Delta 9-THC measure from the subjects. One limitation of the Phase I study was the absence of experimental controls for placebo bias. Phase II is designed to extend this research to include actual driving performance within the confines of a rigorously controlled experimental design. Subjects received a standard dose of marijuana by inhalation, and a double blind control condition was included to minimize experimenter and subject bias. In addition, since alcohol was observed in a large number of cases in association with Delta 9-THC during the DOJ Incidence Study, the present study included alcohol and marijuana plus alcohol as additional experimental conditions.

The specific objectives of the study are listed below:

1. To determine the singular and combined effects of marijuana and alcohol on driving performance on a closed-course drive range.

2. To determine if there is a relationship between the ranges of Delta 9-THC in blood and/or alcohol in breath and measures of driving performance.

3. To determine if the various driving performance measures are differently affected by marijuana and alcohol ingestion.

4. To determine the relationship between the time following marijuana and/or alcohol ingestion and driving performance.

5. To determine the interrelationship among the performance factors affected by the marijuana and alcohol ingestion.

6. To determine whether marijuana, alone or in combination with alcohol, results in impairment that can be reliably detected through external observation of the driving and standard field sobriety tests.

It was beyond the scope of this study to establish a definitive relationship between performance decrements on the various driving range maneuvers and impaired driving ability on public streets. Instead, the study measured performance of various driving range maneuvers related to "real world" driving. One limitation of using a closed-course drive range is that some behavioral domains that are known to be critical to accident avoidance, such as risk taking and response to other vehicles, are not tapped. In spite of these limitations,

the impairment of any skill component of a drive task has potential safety implications and is therefore deserving of serious scrutiny. In addition, some driver-behavior investigators, such as McPherson and McKnight (1981), suggest that deficiencies on the skill and motor components of the drive task may indirectly degrade accident avoidance components. Such an effect would be mediated by a reduction in the "spare capacity" for handling emergency situations that could occur in drivers whose low skill level requires use of large portions of their perceptual and attentional capacity to maneuver a vehicle through traffic.

Research Design

Subjects were randomly assigned to the four treatment combinations created by the following 2 x 2 factorial design:

1) alcohol plus marijuana placebo;
2) marijuana plus alcohol placebo;
3) marijuana and alcohol; and
4) double placebo.

Following treatment, each subject completed four performance trials at one-hour intervals. Thus, the design can be characterized as a 2 x 2 x 4 factorial with repeated measures on the trial factor. The subjects also completed a practice (non-scored) and a pretreatment baseline run. The assignment procedures and independent variables are described below:

1. Alcohol and placebo treatments. Subjects received either active or placebo alcoholic drinks. Active drinks consisted of 1.05 ml of ethyl alcohol per kg of body weight administered as 80 proof (40% ethanol) vodka mixed with 3 parts of orange juice. The total dosage was dispensed in 3 drinks, consumed at 10-minute intervals. With elimination and absorption consideration, this dosage was calculated to produce a peak blood-alcohol concentration of approximately .08% mg. The placebo drinks consisted of plain orange juice with a very small amount of vodka floated on the drink surface to disguise the absence of ethanol.

2. Marijuana and placebo treatments. Subjects were administered either an active marijuana cigarette or a placebo cigarette. The active marijuana was standardized material rolled into a 1.0 g cigarette issued by the National Institute on Drug Abuse (NIDA) containing 1.9% Delta 9-THC. The placebo marijuana cigarettes were comprised of marijuana in which all active cannabinoids had been removed.

   The subjects smoked the cigarette in an ad lib fashion, but were encouraged to finish the entire cigarette within a 10-minute period.

3. Assignment of treatments. Each subject was randomly assigned to one treatment condition until a minimum of 20 subjects had completed the study within each of the four treatment conditions. The randomization was accomplished through use of a table of random digits. Four participants were removed from the data because of chemical indications of drug use within the prescribed "no use" period prior to driving.

Performance Measures

Seven types of dependent variables were used to evaluate posttreatment performance, as described below:

1. Rating of vehicle handling and skill by in-car license examiners and outside raters.

2. Computerized vehicle measures (speed, accelerator reversals, brake presses, steering control, and lateral placement).

3. Standard field sobriety test ratings by CHP officers.

4. Impairment ratings by officers in a following car.

5. Self assessment ratings by subjects.

6. Risk assessment task.

7. Performance on two psychomotor tasks (critical tracking task and brief interval time estimation task).

Measures were obtained at baseline and at each of the four posttreatment trials. In addition to the above measures, blood samples were taken following each trial and assayed for blood alcohol concentration, Delta 9-THC and serum carboxy THC-7.

STATISTICAL ANALYSES AND FINDINGS

The several hundred variables were reduced to a manageable number of dimensions through a series of factor analyses. Multiple discriminant function analysis, canonical correlation analysis, and analysis of variance were used to evaluate the effects of treatment on the performance dimensions. The objective of these analyses was to determine how each performance measure, singly and in combination with the entire set of measures, was affected by the drug conditions. A probability level of $p \leq .05$ was usually required for making significance claims, meaning that differences which would be expected to occur by chance more than five times in 100 were not regarded as true effects.

The posttreatment differences between the groups on the reduced set of variables were evaluated within each run and on the composite of all runs (3-6 combined) through a stepwise multiple discriminant function (MDF) procedure. The 29 variables or composites from the factor analysis comprised the discriminators and the four treatments formed the grouping dimension.

Using a significance level of $p \leq .25$ and a requirement that a performance variable discriminate on at least 2 runs, the following 12 variables were selected for retention:

1.	STOPS	Errors made in stopping
2.	COGNIT	The cognitive factor from the field sobriety test.
3.	STOUCH	Total cones and stanchions touched.
4.	ATTEMPTS	Total risk task attempts.

5.	SDOWN	Total cones and stanchions knocked down.
6.	BSPEED	Speed control rating from inside rater.
7.	BISKILLS	Skill performance rating from inside rater.
8.	UFAILED	Failure to follow directions on urban drive.
9.	ESTCHIC	Time estimated by driver to traverse chicane.
10.	POSTOP	Number of errors in stop position.
11.	SMPH	Miles per hour in the extended drive (speedometer covered).
12.	CTT	Critical tracking task.

The discriminant analyses were then rerun on each trial to derive optimal functions for this common pool of 12 variables. Table 13* shows the resulting discriminant functions for each run and the total composite (runs 3-6 combined). Since four groups were being discriminated, a maximum of three functions combined were evaluated per each analysis. The test for all three functions was significant at the $p<.01$ level on each of the runs. The second function was significant in runs 3 ($p\leq.01$) and 5 ($p\leq.05$). The third function was not significant on any of the runs and is, therefore, not shown. The Wilks Lambda's associated with all three functions combined ranged from .29 (run 3) to .38 (run 4). Since these Lambda's represent the percentage of unexplained variance, a lower value indicates greater discrimination (explained variance) and hence a larger treatment effect. The results in Table 13 therefore indicate that, as expected, the effects of treatment were largest at run 3. Somewhat unexpected, however, was the increase in treatment effect from run 5 to run 6.

The final two columns of Table 13 show the significant discriminant functions for the composite runs analysis (runs 3-6 combined). The two significant functions accounted for 64.3% and 22.3% of the explained variance, respectively. The Wilk's Lambda value indicates that the functions explained 67% (1 minus .33) of the between group variance on the composite runs ($p\leq.001$).

Figure 1 shows a two-dimensional plot of the group centroids for functions 1 and 2 of the composite-runs analysis. Looking first at function 1, note that the maximum separation is between the double placebo (P) and the both-drugs (B) group. Since the placebo group's centroid was on the positive end of the scale, increasing scores on function 1 were associated with non-drugged performance and decreasing scores with drugged performance. The marijuana group (M) and alcohol group (A) centroids fell between the two extremes, with marijuana closer to the both-drugs group.

Separation on the second function is less clear, with the double placebo group (P) having a negative centroid, the M and A groups having positive centroids, and the both-drugs group falling almost at the 0 plane.

*The tables and figures referenced in this section appear at the end of the full report.

Canonical Analysis of Treatment Effects

A series of canonical correlations was computed to determine the amount of variance accounted for by each treatment combination. These analyses provided multivariate tests of the main and interactive effects of marijuana and alcohol on the above 12 variables. Details of the analyses are described in Biasotti et al (1986) but, stated briefly, involved creating dummy variables for the treatment group vector and computing the contribution of each effect term to the size of the canonical correlation.

The results are shown in Table 16 for each run and for the four posttreatment runs combined. The main effects of marijuana and alcohol were highly significant for all trials. Although significant non-additivity (interaction) occurred at runs 3 and 6, the interaction variance was much lower than the main effects and was non-significant at runs 4 and 5 and for runs 3-6 combined. It can, therefore, be concluded that the effects of the two drugs were largely additive.

Looking at the main effects within trial, note that the alcohol effect is consistently the larger of the two, although the differences are modest. There are also differences in the time gradients, with alcohol exerting its largest effect at run 4 and marijuana at run 3. Probably the most surprising finding is the significant increase in the interaction at the last trial. Inspection of the treatment group means 10 for the individual variables, along with the multivariate centroids, indicated that this interaction was due to a deterioration in the performance of the both-drugs group during Trial 6.

The results shown in the last two columns of Table 16 warrant further comment. The first of these summary columns is simply the average of the four individual trial effects. The last column is based on the canonical analysis of the composite performance scores (runs 3-6 combined). Thus, the first summary preserves any differential effect of treatment across trials, whereas the latter ignores trial interactions by collapsing the trials into a single composite.

It is important to understand the above distinction because the relative size of the alcohol and marijuana effects depends on which summary is used. Using the mean-percent-variance-explained across the four trails as a criterion, the marijuana effect is smaller than the alcohol effect (25.1% vs. 31.4%). However, the reverse is true for the composite-runs summary. This seeming conflict indicates the presence of a trial x alcohol interaction. In other words, the structure of the performance decrements for alcohol varied across trials and these effects were obscured when the trials were collapsed into a single composite. Under these conditions, the average of the individual trials provides a more accurate reflection than does the composite analysis. As indicated above, this average indicates that alcohol had a larger effect on performance than did marijuana.

Ancillary Measures

The results for some of the ancillary measures and other variables of special interest are summarized below.

Following car, field sobriety tests, and overall LRE and self-ratings. Figures 15, 16 and 17 display plots of means on selected rating variables. The most outstanding feature of these data is the poorer performance of the both-drugs group on every variable. Note also the remarkable similarity of the inside rater (LRE), self and officer ratings (Figure 16). In general, the impairment associated with either marijuana or alcohol alone was perceived as minimal and slightly greater for alcohol. However, since these results are in terms of the composite runs, they tend to underestimate the immediate effects of the drugs.

The results on the following-car measure (FALLCAR) produced clear-cut evidence of a treatment effect (Figure 17). Subjects receiving both drugs would have been "stopped" or "pulled over" about 60% of the time by the CHP officer, compared to about 15% of the placebo subjects. Alcohol alone and marijuana alone resulted in stopped scores of 50% and 32%, respectively.

Impairment Questionnaire. Subjects completed an impairment questionnaire after the third and fifth drives for the purpose of assessing subjective perception of change resulting from drugs. The scores on the 10 impairment items were summed and tabulated by treatment and trial (run 53 and 5). The results are presented below:

Treatment	Run 3	Run 5
Placebo	10.0	5.57
Marijuana	22.7	5.26
Alcohol	21.2	7.20
Both	29.3	18.05

These findings are very consistent with the subjective self-assessment ratings presented earlier, particularly with respect to the tendency for the both-drugs condition to lengthen the duration of impairment.

BITE and CTT. The mean BITE and CTT scores by treatment groups and run are presented in Table 3. On each measure, higher scores indicate better performance.

Although there was a definite trend on the BITE task for the placebo group to perform best, and the both-drugs group poorest, none of the analysis of variance tests produced a significant difference at $p \leq .05$. The results are nevertheless suggestive of impaired time estimation, particularly when both drugs are combined.

The results on the CTT measure show that the alcohol and both-drugs groups did significantly worse (lower means) than did the placebo and marijuana groups, indicating a clear-cut alcohol effect. The differences reached statistical significance ($p \leq .05$) at runs 4 and 5 and on the total composite (runs 3-6 combined). Somewhat surprisingly, there is not even a directional trend toward marijuana impairment on the CTT. The scores for the placebo do not change materially from run-to-run, whereas all three drug groups tended to improve. Thus, there is no evidence of residual practice effects, but there is clear indication of reductions in impairment on the CTT over time.

Blood Levels

One of the objectives of the study was to determine if variations in blood levels were associated with variations in driving performance. To evaluate the statistical relationship between the drugs and 12 performance measures, the actual blood levels of the substances (THC, carboxy, and BAC) were recorded for each of the runs for a canonical analysis with the 12 performance measures.

The mean blood levels by trial are presented in Table 44. The results conform fairly well with what was expected based on the amounts consumed and time from dosage. The alcohol group peaked at Trial 4 (.08%) and declined to a BAC of .04% at Trial 6. The marijuana and both-drugs group achieved peak THC levels ($\bar{x}$ = 69.6 & 54.3) at Trial 3, and the levels had dissipated precipitously by the next trial ($\bar{x}$ = 13.1 & 13.4). The difference between the marijuana and both groups on serum carboxy levels was not expected and, at first glance, suggests that alcohol interferes with the creation of serum carboxy. Although the difference persisted throughout the trials, the authors are inclined to dismiss this finding due to variations in serum carboxy among the subjects prior to treatment.

The only other notable finding in Table 44 is the variation in BAC levels between the alcohol-only and both-drugs groups. The results suggest that the presence of THC/serum carboxy delayed the absorption of alcohol. At Trial 3, the alcohol-only group achieved a BAC of .07% compared to .05% for both groups. However, the 2 groups were at parity at trials 5 and 6.

Since there was some variation in the blood levels actually attained by the subjects within a given treatment and trial, it was possible to explore whether these variations were correlated with differences in driving performance. In other words, did subjects who attained higher-than-average levels tend to exhibit more performance detriment than those who attained lower levels? This question was pursued by the previously mentioned canonical correlation analysis in which scores on the 12-variable performance vector were correlated with the three quantitative blood levels and dummy-coded treatment vector.

Table 45 shows the contribution of the blood levels to explaining variation on the performance vector. Note that in all cases the inclusion of blood alcohol level increased the size of the alcohol effect and that the increase was statistically significant on runs 3 and 4 ($p \leq .05$) and approached significance on runs 5 and 6. In contrast, the contribution of serum THC or serum carboxy alone was always less than the contribution of treatment group designation. However, when both serum carboxy and serum THC were included, there was an increase in the marijuana effect on runs 4-6. The increase on run 4 was statistically significant ($p \leq .05$). At run 4, the net effect (explained variance) increased from 62% to 72% ($p \leq .05$).

In general, these results indicate that knowledge of a person's exact blood levels on all three measures slightly increased the ability to predict performance on the driving tasks during runs 4 and 5. In addition, the results indicated that the marijuana effect is mediated by the joint operation of serum THC and serum carboxy. Either substance alone does poorly as a predictor of performance.

The above analysis has limited sensitivity due to the restricted range of variability of the blood levels. This stems from the fact that the experimental design was intended to minimize variability by administering a constant dosage to each subject and then testing each subject at fixed intervals from dosage.

CONCLUSIONS

The Main and Interactive Effects of Alcohol and Marijuana - General Characteristic

The results of the univariate and multivariate analyses indicate that both substances affected driving performance. The MDF analyses resulted in two significant linear composite (functions) of the 12 performance measures which were most consistently and uniquely impacted by the treatments. These two functions explained between 62% to 70% of the between-group variance on the 12 measures across the 4 posttreatment trials. Approximately 60% of the explained variance was attributed to the first function compared to 20% for the second function. The first function produced maximum discrimination between placebo and the both-drugs group, with marijuana alone and alcohol alone occupying intermediate positions between these two performance extremes. Inspection of the group means on the 12 variables which defined the function indicated the both-drug polarity was generally indicative of impaired performance.

The second function tended to separate the marijuana and alcohol groups from the placebo group, with the both-drugs group occupying an intermediate position.

An attempt was made to interpret the "meaning" of the two functions by inspection of the standardized discriminant function coefficients and structure loadings (Table 14). Persons scoring higher (better) on the first function produced higher estimates of the speed at which they could drive the chicane and drove more quickly through it; drove at lower and more appropriate speeds when the speedometer was covered, as well as on most other parts of the course; drove more cautiously; made more accelerator reversals on the extended drive; performed better on the detour task; performed better on the field sobriety tests; were judged to be less impaired by the officer; rated their driving and field sobriety performance higher (less impaired); received higher overall rating from the driver's license examiner (LRE) and made fewer driving errors on the drive course. This function clearly reflects between-group variation and performance decrement on a wide variety of measures: (1) subjective self ratings; (2) LRE ratings of speed control, overall performance, cautiousness and number of driving errors; (3) officer field sobriety and vehicle control ratings; (4) objective measures of performance (number of stanchions knocked down, accelerator reversals, urban drive speed); and (5) number of attempts on the risks task. Since this function produced maximum separation between the both-drugs and placebo group, with the latter falling at the positive end of the first function, higher scores on most of the preceding variables were associated with not being drugged, and lower scores with being exposed to alcohol and marijuana combined. The nature and range of variables affected indicate that exposure to the combined marijuana/alcohol condition resulted in impaired vehicular control and an accurate subjective awareness of the impaired performance.

Inspection of the second function indicated that higher scores on the function were associated with the marijuana and alcohol condition, and the structure loadings indicated that it is primarily a measure of impaired stopping. By far, the highest loading on the function was on the variable POSTOP, which correlated + .69 with the function. This variable represents the LRE's rating of proper stopping position on all stopping maneuvers throughout the drive range. Since the variable was scaled so that low scores indicate proper stopping position (1=smooth stop, 2= abrupt or misjudged stop, and 3=rolling or not stop), the high positive correlation between the function and variable is indicative of a higher proportion of improper stop ratings among marijuana and alcohol subjects. Unfortunately, the method of scaling did not distinguish between stopping too soon or too late relative to the sign.

Four other stopping measures also had significant positive loadings on the second function: (1) SSTOP (stopping errors on the speedometer covered segment of the course); (2) ESTOP (stopping errors on the extended drive); (3) STOPS (total number of stopping errors on all segments of the course); and (4) BIPOST (bipolar rating of stop position cautiousness). A fifth stopping measure, BISTOP, yielded a negative loading (-.31), indicating that stopping position caution on the speedometer-covered portion of the course was negatively associated with the function.

Taken together, the above results indicate that marijuana and alcohol affected stopping behavior, but that the negative effects were reduced when both drugs were combined. The mechanism underlying what appears to be a suppressive interaction is not clear, and interpretation is further complicated by the above-mentioned scaling problems. One possibility is that the separate drugs produced different types of stopping errors (e.g., delayed vs. early stops), which cancelled out when both were combined.

The main and interactive multivariate effects of marijuana and alcohol on the 12-variable discriminant functions were evaluated through a series of canonical analyses, and an analysis of variance procedure was used to evaluate univariate effects. The canonical analyses indicated that both substances had highly significant multivariate main effects on all four posttreatment runs. The marijuana condition explained between 23.1% to 29.2% of the variance on the 12 most discriminating performance measures, averaging 25.1% for all 4 trials. The alcohol condition produced a somewhat larger effect, accounting for 29.9% to 32.3% of the performance variance, and yielding an average effect of 31.4% for all 4 trials. The multivariate effects were largely additive, although small, but significant, interactions did occur on Trials 3 and 6 where the respective explained-variance totals for the marijuana by alcohol interactions were 12.0% and 12.9% respectively.

<u>The Main and Interactive Effects of Marijuana - Detailed Characteristics of Effects Within Trials</u>

The above discussion has primarily focused on the effects on all posttreatment trials combined into single composite measures. It has also been limited to the 12-variable core selected through the multiple discriminate function analysis (MDF).

A complete understanding of the results of the experiment require interpretation of effects within and across trial and consideration of the many variables not included in the final MDF analyses.

The evaluation of treatment x trial interaction was achieved through a test of slope differences on the trial factor. Since the trials occurred at fixed points in time from the single drug ingestion, the trial slopes represent the effects of time, and the slope differences reflect temporal differences in each treatment group's performance gradient. These tests indicated significant slope difference on each of the 12 measures selected from the MDF analysis. A major source of the difference was marijuana's tendency to result in maximum impairment in the first posttreatment trial, while alcohol's effect maximized at trial 4 and did not decline as rapidly as the marijuana effect. The both-drugs group tended to show the impairment gradient that would be expected from the component drugs, evidencing maximum impairment at both trials 3 and 4 and consistently showing greater impairment than either marijuana or alcohol alone. In addition, the combination of the two substances significantly lengthened the duration of effects, resulting in a rather remarkable increase in impairment from trial 5 to 6. The emergence of a synergistic marijuana-alcohol interaction at trial 6 suggests an effect mediated by some residual mechanism occurring when the two substances are combined. An obvious intuitive explanation would be that marijuana and alcohol interact to produce greater fatigue and "hang-over" effects.

Other results of interest are the findings on various peripheral measures, such as FALLCAR, field sobriety tests, self-rating and the CTT. In general, the subject self-rating of impairment was among the more sensitive indicators of treatment and tended to parallel the objective measures. The fact that the self-rating impairment indices closely mirror both the objective indicators and the overall LRE and officer rating provides confirmation for the reliability and validity of the results.

That the officers in the following-car (FALLCAR) were able to detect driving impairment with a significant degree of accuracy is notable. The FALLCAR results also clearly reveal the different time gradients for marijuana and alcohol. The marijuana subjects were detectable only at run 3, whereas alcohol peaked at run 4, and the both-drugs group was detected as impairment on all runs except 6.

The results on the CTT variable are surprising in that marijuana alone did not produce evidence of impairment, which is in conflict with prior research findings. This complex psychomotor task was originally devised to detect alcohol impairment. The fact that alcohol and marijuana plus alcohol combined did produce impairment would seem sufficient to dispel the hypothesis that marijuana finding can be attributed to same type of error or procedural artifact.

Surprisingly, the marijuana-only condition resulted in fewer stanchions being knocked down at trial 3, where the level of intoxication was actually greatest. However, it is important to note that persons receiving marijuana or marijuana and alcohol tended to drive more slowly through the chicane than did the placebo and alcohol-alone subjects. Speed through the chicane was measured by the vehicle line sensor, which produced the following elapsed times for the placebo, alcohol, marijuana, and both-drugs treatments, respectively: 16.7, 16.3, 17.3, and 18.4. Although the differences were not statistically significant ($p = .22$), it is instructive to note that other researchers have found that marijuana tends to cause persons to compensate for subjective impairment by reducing task difficulty through reduced vehicle speed.

Blood Level

One of the objectives of the study was to assess the feasibility of developing an objective chemical index of marijuana impairment. Although the results did show that the quantitative levels of THC and carboxy combined resulted in some increase in the ability to explain performance variations, the practical and theoretical implication of the finding are not entirely clear. Experimental replication of the study and use of a wider range of marijuana-dose levels is needed before the feasibility of establishing a quantitative threshold can be fully evaluated. We are not optimistic about the prospects for development of quantitative thresholds for marijuana impairment.

Traffic Safety Implications

There is a vast amount of empirical evidence documenting the effects of marijuana on a wide array of human performance measures - cognitive, psychomotor and affective. Although the literature has clearly established that marijuana affects all three domains and results in detriments in the ability to perform many psychomotor and cognitive tasks, the evidence is somewhat more equivocal on the question of actual driving skill and even more equivocal on the question of those aspects of driving skill that are related to safety and accident avoidance.

Authorities are therefore not in agreement on the traffic safety threat posed by marijuana use (Warren and Simpson, 1980). In a recent series of papers, McBay and Owens (1980) and Mason and McBay (1984, 1985) concluded that marijuana is a relatively minor factor in traffic accidents and they questioned the feasibility of relating impairment to specific levels of THC. Although many of their criticisms of past studies are both astute and pertinent, we believe these same limitations prevent forming unqualified opinions in any direction about the role of marijuana in traffic accidents. Many of the conclusions formed by McBay and his associates are based on the failure to find a substantial incidence of THC in the blood or plasma levels of drivers killed in single vehicle accidents in North Carolina. Considerable caution is necessary in generalizing incidence data from North Carolina to a state like California. Not only are there likely to be large differences in marijuana usage, there may be also differences in drive task complexity between such states and in the use of cannabis in conjunction with vehicle travel.

In addition, Moskowitz (1985) has recently pointed out that behavioral impairment and subjective intoxication are still manifest after THC has dissipated from blood. This factor results in an unknown proportion of false negative findings from an analysis of accident victim blood specimens. Nevertheless, the point remains that the traffic safety implication of marijuana use must ultimately be based on direct evidence of its causal role in increasing accident risk. This necessitates establishing accurate "population-at-risk" baselines for: (1) the incidence at which persons drive under various levels of THC alone; (2) the same incidence in combination with alcohol; and (3) the same incidence in combination with other drugs. The fact that marijuana is so often detected in conjunction with alcohol makes it difficult to establish a case against marijuana since any increase in relative risk could be due to alcohol alone. Establishing incident rates for the above risk groups would facilitate interpretation of the respective incident rates among accident-involved drivers.

Probably the most consistent and important finding of this study was the demonstration of an additive marijuana/alcohol effect on a wide array of performance measures. If one accepts the thesis that marijuana in conjunction with alcohol makes people "drunker", then it follows that marijuana in this context increases accident risk. A public policy implication of such a thesis might be to reduce the illegal, per se, BAC level for persons detected with both substances in their blood.

The question of the traffic safety risk posed by marijuana alone is not as clear cut as the risk presented by marijuana and alcohol in combination. Although evidence of impairment was identified in both the present and numerous past studies, the translation of this evidence into inferences about accident causation presents numerous difficulties. Before explaining why, we offer a dissenting opinion from a recent comprehensive review of the literature by Moskowitz (1985):

> "It should be clear from the above review that there is more than sufficient experimental evidence to conclude that marijuana seriously impairs psychomotor performance required for driving. Among the areas which exhibited overwhelming evidence for impairment were: A. Coordination...; B. Tracking; C. Perception; D. Vigilance; E. Driving and flying performance measured by simulators; F. Driving performance on the road...
>
> Clearly, marijuana is a substance which produces serious behavioral toxicological effects. Any situation in which safety both for self and others depends upon alertness and capability of control of man-machine interaction precludes the use of marijuana."

Based on the present study and past evidence, we agree that marijuana undoubtedly impairs psychomotor abilities that are functionally related to skillful driving and that driving skill itself may be impaired, particularly at high dose levels or among naive subjects. Given these facts alone, Moskowitz's implicit recommendation that people not drive after consuming marijuana should obviously be heeded. However, the extent to which marijuana-impaired driving causes accidents cannot be deduced from the present study, nor any of the studies cited by Moskowitz. Our more conservative posture to this question is based on the following rationale:

1. In their multidisciplinary investigation of traffic accidents, Treat et al (1979) identified "improper lookout" and excessive speed as the two most frequent human factor causes of accidents. Although improper lookout may involve some of the perceptual and information-processing components affected by marijuana, it is more closely related to the search and scan strategies utilized by drivers in anticipating and detecting potential conflicts. In the only study of marijuana's impact on traffic visual search behavior, Moskowitz et al (1976) found no evidence of a negative effect on search and scan behavior. Excessive speed can be best viewed as a reflection of attitude toward risk, risk assessment and aggressiveness. Several investigators have reported that marijuana reduces risk taking propensity and driving speed.

Because of these compensating tendencies, it is presently not possible to assess the net impact of marijuana as a causal agent in traffic accidents. Although some increased accident risk appears likely, the magnitude of the risk remains obscure.

2. Most of the laboratory marijuana studies which have shown the greatest psychomotor impairment have utilized tasks that are only abstractly related to driving. Although divided attention and tracking are required for driving, it does not necessarily follow that performance on a highly novel and complex laboratory task designed to magnify performance decrements are correlated with actual "real-world" performance in a vehicle. Harano, Peck and McBride (1975) evaluated a large array of psychomotor measures, including divided attention, and concluded that none were important predictors of a driver's accident propensity. The fact that attempts to measure response to simulated accident situations has not consistently detected a marijuana-induced decrement, even at high dose levels, underscores the need for more research (Stein et al, 1983).

Future Research Needs

In addition to the need for improved epidemiological studies mentioned earlier, the relationship between marijuana consumption and driving behavior can be clarified by a research design possessing the following characteristics:

1. A multi-method/multi-criterion approach in which subjects perform relevant psychomotor, driving simulator, and actual driving tasks. The utilization of different measurement domains will permit an assessment of the multivariate effects across domain, leading to more generalizable characterizations of the extent and locus of marijuana-induced impairment.

2. At least three dose levels of marijuana should be used (none, moderate, and high) in order to obtain a greater range of THC variation for investigating dose-response relationships.

3. Frequency of prior marijuana usage should be treated as an experimental factor by selecting subjects who substantially vary on use rate. At least three levels should be employed--light user, moderate user, and heavy users. Such a design would permit an evaluation of treatment x use frequency interaction, resulting in a better understanding of whether acquired tolerance and accommodation are important factors in influencing impairment.

4. An independent groups design with repeated measurement trials should be employed in preference to latin square-design in which each subject receives all treatments. Individual differences in drug response and experimental error could be reduced through matching and analysis of covariance procedures.

5. The design should include some tasks under reduced-illumination to simulate night driving conditions. Serious accidents more often occur at night, and there is reason to suspect that marijuana-induced impairment would be greater under night driving conditions.

Further research is also needed to validate the relationship between tasks (or simulators) designed to detect drug impairment and actual driving behavior, as measured by driving performance tests and accident involvement rates.

INTRODUCTION

This report represents Phase II of an effort to characterize and quantify the effects of marijuana use, alone and in combination with alcohol, on driving performance. An earlier study (DOJ Incidence Study), funded by the Office of Traffic Safety, indicated that Delta 9-THC was present in a significant proportion of the submitted blood samples drawn from California drivers detained by highway patrol officers because of ostensibly impaired driving performance (Zimmerman et al 1983). Phase I of this grant also suggested that volunteers given ad lib doses of marijuana by inhalation were subsequently considered impaired when required to perform the standard field sobriety test (Reeve et al 1983). These impairment ratings persisted for up to three hours after smoking and were associated with hemolyzed blood concentrations of Delta 9-THC measured from the subjects. One limitation of the Phase I study was the absence of experimental controls for placebo bias. Phase II is designed to extend this research to include actual driving performance within the confines of a rigorously controlled experimental design. Subjects received a standard dose of marijuana by inhalation, and a double blind control condition was included to minimize experimenter and subject bias. In addition, since alcohol was observed in a larger number of cases in association with Delta 9-THC during the DOJ Incidence Study, the present study included alcohol and marijuana plus alcohol combined as additional experimental conditions.

The specific objectives of the present study were:

A. To determine the singular and combined effects of marijuana and alcohol on a number of measures of driving performance.

B. To determine if there is a relationship between the range of Delta 9-THC in blood and/or alcohol in breath and measures of driving performance.

C To determine if the various driving performance measures are differently affected by marijuana and alcohol ingestion.

D. To determine the relationship between the time following marijuana and/or alcohol ingestion and driving performance impairment.

E. To determine the interrelationship among the performance factors affected by the marijuana and alcohol ingestion.

F. To determine whether marijuana, alone or in combination with alcohol, results in impairment that can be reliably detected through external observation of the driving and standard field sobriety tests.

It was beyond the scope of this study to establish a definitive relationship between subject's performance decrements on the various driving range maneuvers and impaired driving ability on public streets. Instead, the study focused on performance decrements of various driving maneuvers on a closed-driving course that are related to "real world" driving. One limitation of using a closed-course for driving trials is that some behavioral domains that are known to be critical to accident avoidance are not tapped. For example, search and scanning strategies, hazard detection, and risk taking are not likely to arise for a

driver involved in repeated trials over a closed-course driving range and in the absence of other vehicles. In spite of these limitations, the impairment of any skill component of the above task has potential safety implications and is therefore deserving of serious scrutiny. In addition, some driver-behavior investigators, such as McPherson and McKnight (1981), suggest that deficiencies in skill and motor components of the driver task may indirectly degrade accident avoidance components. Such an effect would be mediated by a reduction in the "spare capacity" for handling emergency situations that could occur in drivers whose low skill level requires use of large portions of their perceptual and attentional capacity to maneuver a vehicle through traffic.

The following sections present an overview of past studies on an effect of marijuana and marijuana and aclohol combined on human performance variables related to driving behavior. Because the detrimental effects of alcohol on human performance have been so well documented, the numerous alcohol studies reviewed are not summarized here. Suffice it to say that accident investigation studies are virtually unanious in showing that alcohol is present in over 50% of all fatal accidents. The reader interested in more detailed information can reference Carpenter (1962) and Perrine (1974,1975).

The Effects of Marijuana on Driving

Until the identification and synthesis of Delta 9-THC as the main active substance of cannabis (Mechoulam, 1973) and subsequent forensic tests to quantify it, evidence about marijuana and driving was largely based on anecdotal evidence and self report surveys. In a survey of 246 college students licensed to drive in Western Ontario, Smart (1974) found that 10% of those who reported having had an accident in the previous year indicated that their accidents had occurred after using marijuana. Smart does not clearly specify whether these drivers had been simultaneously using alcohol or other psychoactive drugs. Respondents to a survey conducted by Grilly (1981) revealed that alcohol was perceived to be more detrimental than marijuana in driving.

Actual performance studies concerning marijuana use have largely involved simulate driving tasks or, less frequently, closed-course driving procedures (Crancer et al 1969, Rafaelsen et al 1973; and Tinklenberg 1972). Simulator studies provide a way of measuring specific sub-sets of the drive task, such as risk-taking, tracking, car control, and sensory perceptual skill. These simulator studies have often failed to find detriment in performance after THC treatment. Moskowitz et al (1972) did not find significant impairment in car control and tracking aspects of a driving simulator after doses of 50-200 ug/kg smoke-delivered THC. They did, however, find dose-related decrements in detection responses for the marijuana treatment groups. Likewise, Rafaelsen (1973) found little effect on simulated driving except at oral doses of 12 or 16mg. At these levels, Rafaelsen demonstrated that cannabis (as well as alcohol) increased the time required to brake and then start from a stopped position. Concurrently, while the alcohol treatment resulted in an increase, marijuana decreased the number of gear changes compared to baseline performance. In simulated instrument flying by experienced pilots, Janowsky et al (1976) found definite deterioration only after very large doses of 90 ug/kg of smoke-delivered THC.

Crancer (1969) found speedometer and steering errors after subjects smoked about 22 mg in a simulated driving task. However, since improvement occurred in half the subjects on some scores, and insufficient controls were imposed, these results may be attributable to practice effects. Further criticisms of the experimental design and drug treatments and the lack of clarity of response scores have been made in regard to this study (Moskowitz, 1972).

In a study of marijuana and driving performance, Dott (1972) used a closed-loop simulator with two independent lanes to allow assessment of a passing task. Subjects receiving THC displayed decreased risk-taking behavior and greater hesitancy except when confronted by an emergency situation wherein decision time per se was not impaired. Dott concluded that THC did not appear to affect "decision time" (response to an emergency), but altered the subject's perception of what constituted an acceptable passing gap. Ellingstad et al (1973) replicated the Dott study and obtained similar results.

Kielholz et al (1973) conducted a simulated driving study utilizing an information density-changing task which included a steering wheel and pedal depression device. Marijuana-impaired subjects showed unchanged simple reaction time and lengthened complex reaction time. Subjects had difficulty attending to multiple sets of objects and demonstrated "tunnel-vision". Dose-related increases in risk-taking were also found in the marijuana-impaired subjects. Some effects were reported lasting up to 10 hours after marijuana ingestion.

While these studies provide evidence that driving performance is likely to be altered by marijuana, they do not demonstrate the impact of marijuana use on actual driving performance. In order to make this assessment, closer approximations, such as closed-course driving and actual traffic involvement studies, are necessary.

Moskowitz (1976) reports that the earliest closed-course driving study examining marijuana effects was conducted by North Carolina Highway Safety Research Center. In this study, subjects drove through seven varied cone patterns. No difference was found between marijuana treatments and non-treatment groups. Hansteen et al (1976) conducted a closed-course study and observed decrements in car handling performance following marijuana use. In this experiment doses of 21 and 88 ug Delta 9-THC/kg alone or in after drug treatment on six laps of a 1.1.-mile course and then again three hours later on three laps of the course. Increases were reported in the number of cones overturned in a slalom, but outside observers were unable to detect an increase in "rough-handling" behavior caused by marijuana alone; whereas, alcohol adversely affected both of these performance measures. No differences in speed were found, with the exception of the high marijuana group which drove 7% slower than the other groups. Hingson (1982) urges cautious interpretation of these results, however, because of the artificial driving circumstances employed.

In the first study in which subjects were allowed to drive on unrestricted city streets under the influence of marijuana, Klonoff (1974) gave modest doses of THC via smoked marijuana to 43 male and 21 female student volunteers and measured driving performance on the city streets of Vancouver, British Columbia. Further driving performance on a closed-course containing tunnels, subjective ratings provided by DMV examiners who accompanied the drivers through all driving portions of the study. Eleven behavioral components were rated to provide further evidence of impairment. They included general driving habits,

cooperation, judgment, speed, and concentration. Results from this study indicated that even low doses of marijuana impaired driving with greater accompanying heart rate elevation on the closed-course where driving tasks were more exacting. On the closed-course, marijuana subjects showed some impairment in recalling the proper order of tasks to be followed on the track as well as a loss of discrimination of internal and external course markers. Even greater performance decrements were found in the actual street traffic setting. These decrements in actual street driving included missing traffic lights or stop signs, passing without sufficient caution, not adjusting to changes in traffic flow, lack of awareness of pedestrians or stationary vehicles, preoccupation with traffic signals and slowed response to green light signals (Klonoff, 1974). Considerable variation among individual drivers was observed, with some actually showing improvement after marijuana, particularly in the street-driving task. In a general review of the literature, Moskowitz (1976) credited Klonoff's study as having greatest "face validity", but commented that the subjective variables rated by the instructors provide little insight into the specific pharmacological actions of the drug.

Casswell (1979) examined the effects of marijuana in an actual driving situation and found that the number of steering corrections made by subjects decreased, as did their average vehicle speed. Casswell suggested that marijuana-impaired drivers tended to compensate for the drug effect by decreasing overall speed, thus reducing the rate of information processing required.

A number of studies have been conducted on the effects of marijuana upon specific psychomotor tasks believed to be related to accident avoidance. Sharma and Moskowitz (1973) tested reaction time to a light signal detection tack designed to simulate traffic light detection. They found that light signal detection errors increased significantly between each of three levels of marijuana (50, 100, or 200 ug Delta 9-THC/kg body weight condition). The number of false alarms was not affected by any of the marijuana conditions. Dogoloff (1981) cited studies showing similar delays in reaction time to "stop" and "go" signals under marijuana conditions. Dogoloff also found that after smoking marijuana, drivers had a consistently lower performance than non-marijuana smokers when required to divide attention between concentrating on following a car at a safe speed and identifying a danger signal.

Reduced tracking performance has been shown for rotary pursuit tracking by Roth et al (1973), Clark et al (1974), and Manno et al (1970) for varied levels of marijuana use. Casswell and Marks (1973) required subjects to perform a divide attention compensatory tracking task while under the influence of extracted THC or placebo cigarettes in high or low dosage (500 ug/kg and 250 ug/kg active material respectively). Absorption was indicated by significant increases in pulse rate. Significantly more central and peripheral light signals were missed for both THC levels.

Bech et al (1973) showed that 300 ug/kg of cannabis had the effect of delaying braking-time by 16% to 23%. After 500 ug/kg of cannabis, the braking-time delay was increased by 66% above baseline braking-time. In a separate condition, subjects were given 70 mg/kg of alcohol and demonstrated braking-time delay increases of 44% baseline performance.

Effects of Marijuana and Alcohol in Combination on Driving Performance

Examining the influence of marijuana and alcohol in combination on the performance of tasks related directly to driving is an involved task. Correlation between blood concentration and behavioral performance has not been clearly established for marijuana. The complexity of defining safe levels of THC in combination with alcohol becomes more apparent when the effect of various pharmacological parameters are examined. For example, although alcohol and THC are able to produce dose-related decrements in both individual driving skills and actual driving performance, it cannot be assumed that their combined effect will be additive. Adams et al (1975), for example, reported that glare-recovery was slowed by each substance individually and produced a sub-additive effect for combined use. Belgrave et al (1979) used a battery of cognitive and psychomotor tasks to obtain four driving-skill factors, each of which was significantly impaired by alcohol in moderate dosage and further augmented by modest doses of THC.

Macavoy and Marks (1975) studied the effects of alcohol and marijuana on a task requiring subjects to monitor and report visual events occurring in both central and peripheral visual field - a task related to the requirements of actual driving. Low doses of THC were found to cause definite impairment, as did moderate doses of alcohol, with a tendency for the drug in combination to cause an antagonistic effect. The effect was such that alcohol offset the decrement in divided attention brought about by THC in the responses of experienced users. An extension of this study, however, showed no evidence for such an interaction effect.

Belgrave et al (1979) also performed an extensive study into the effects of combined marijuana and alcohol use. The treatment conditions were marijuana alone, alcohol alone, combined marijuana/alcohol and a placebo. The performance variables included: 1) standing steadiness; 2) simple and complex reaction time; 3) the Vienna Discrimination Apparatus; 4) a pursuit motor task; 5) an arithmetic task to measure concentration and attention; and 6) a word construction test. A factor analysis resulted in four rotated factors: 1) reaction and speed; 2) cognitive functioning; 3) standing steadiness; and 4) psychomotor coordination. Both THC and ethanol produced significant decrements on the first factor, while alcohol produced decrements in the standing steadiness and psychomotor coordination factors. THC caused a significant deterioration in performance on all four rotated factors. The combined treatment group showed no more than an additive since there was no evidence of interaction.

The findings of Belgrave et al (1979) indicate that the effects of combined marijuana/alcohol tend to be additive. Some authors have disputed this claim and suggest that, depending on the measurement task, some performance variables show synergistic and antagonistic effects. Macavoy (1975) found, for example, that in a divided attention performance task, marijuana caused significant decrements in performance, while produced no effect. The double-dose subjects showed interactive effects depending on whether they were users or non-users of cannabis. The non-users showed a synergistic effect, while the users showed antagonistic effects. One of the first attempts to demonstrate the effects of moderate levels of marijuana and alcohol alone and in combination with a closed-driving course was made by Casswell (1979). Casswell found that vehicle velocity tended to increase under alcohol and under alcohol and marijuana

combined. Furthermore, lateral positioning of the vehicle in the roadway tended to become more variable under the combined condition. Conversely, given marijuana alone, the number of steering corrections decreased as did average vehicular speed. However, there were problems in the procedures for administering drug doses, resulting in great variability in the time lapses between administering drugs.

Smiley et al (1974) conducted a closed-course investigation in an attempt to gauge some of the required skills involved in the driving task. These investigators found that the accuracy with which drivers were able to stop at a traffic signal deteriorated after alcohol treatment. Less adverse effects were observed on each of the driving performance measures than with alcohol alone. The only exception to this was a significant reduction in response time to a light signal which flashed at random intervals throughout each trial. Alcohol and marijuana together appeared to improve performance over that obtained under the alcohol alone. Stopping accuracy varied greatly for subjects in the alcohol and marijuana combined group with some subjects obtaining their worst scores, while others, their best scores on this task. Measures of steering movement were based on a power spectral density function of steering wheel angle which was calculated for each subject for each drug condition. The alcohol and marijuana-combined group demonstrated the greatest area under the power spectral density curve, thus showing the greatest amplitude in steering change. No clear speed variation trend occurred. However, subjects in the placebo condition drove at significantly greater speeds than did subjects in any of the active drug conditions (alphas ranging from $p > .10$ to $p < .01$). Subjects given only alcohol drove faster than subjects in the combined group ($p < .05$).

In the more recent closed-course study conducted by Attwood et al (1981), driving performance was observed under marijuana and alcohol combined, marijuana alone, alcohol alone and a placebo control. Two levels of alcohol and two levels of marijuana were used. Eight subjects were assigned to the treatment conditions using a repeated measures design, and various driving parameters were measured electronically. Multivariate techniques demonstrated significant discrimination between each treatment on a number of variables, but univariate comparisons did not produce results which exceeded chance significance. Since multivariate methods capitalize on chance relationships, especially when employed on small samples, the Attwood findings must be reviewed with caution until replicated.

Sutton (1983) evaluated the effects of marijuana, alcohol, and marijuana-alcohol combined on the ability of subjects to drive on an experimental driving range. Using a double-blind, repeated-measures design and nine subjects, he found that only the combined marijuana-alcohol condition resulted in significant impairment. The significant synergistic interaction was evident in both ratings of in-car raters and a following patrol officer. The patrol officer was able to identify every trial involving the combined-drug group. In commenting on the findings, Sutton expressed surprise that the marijuana-alone condition did not result in impairment, in view of earlier studies and in view of "the elaborate efforts made in this study to maximize marijuana intoxication". (The marijuana condition was comprised of a 2% Delta 9 cigarette and the alcohol condition was calibrated to produce a BAC of .06%.)

In two separate but related experiments, Allen et al (1982), and Stein et al (1983) used a simulator to investigate the separate and combined effects of alcohol and marijuana. The first study employed two alcohol levels (0 and .10%) and three marijuana levels (0, 50, and 100 ug Delta 9-THC/kg body weight). The alcohol condition was found to consistently impair most of the measured functions, including increased simulated accidents and violations. The marijuana conditions, however, did not result in consistent impairment, nor was there evidence of an interactive effect.

The second experiment was essentially a replication of the first, except with higher marijuana concentrations (0, 100, and 200 ug Delta 9/kg). The only reliable effect due to marijuana alone was a tendency to drive more slowly. However, there was a significant interaction between alcohol and marijuana on simulated accidents, with the alcohol and high-marijuana-dose group involved in significantly more accidents than other conditions.

Although the mechanism of the alcohol-marijuana interaction is unknown, and the function is highly complex, there can be little doubt that the substances are at least additive in their effects on a number of driving-related tasks.

Epidemological Accident Investigations

Several researchers have done both survey studies of the incidence of marijuana in the body fluids of fatal accident victims in an attempt to document the accident risk from driving under the influence of marijuana. Grilly (1981) found that the majority of surveyed respondents viewed marijuana as detrimental to driving skills. These respondents also viewed marijuana not to be as detrimental as other commonly-used substances such as, alcohol, barbiturates, narcotics, and LSD. In a survey of 256 college students licensed to drive in Western Ontario, Smart (1974) found that 10% of those respondents who reported having an accident in the past year also indicated that the accident occurred after using marijuana. Sterling-Smith (1973) concluded that marijuana use occurred more frequently (16%) prior to fatal vehicular accidents than in matched controls who had no accidents. Sterling-Smith (1976) also found that of matched fatal-accident drivers and non-accident drivers, the fatal-accident driver was more likely to use marijuana (45%) than were the control drivers (34%). Teale et al (1977), using radioimmunoassay (RIA) to identify cannabinoids, found the 4.5% of the injured drivers in England and Wales contained Delta 9-THC. Woodhouse (1974) found of 710 fatally-injured drivers, 38% had been "in contact" with cannabis. Cimbura et al (1982) found cannabinoids to be present in 12% of 484 drivers and pedestrians fatally injured in Ontario. Using a large sample of 1,792 people arrested for driving under the influence, a study conducted by the California Department of Justice indicated that marijuana was present in 12% to 15% of the samples tested (Zimmerman et al 1983).

McBay and Owens, 1980, and Mason and McBay, 1985, justifiably criticize most studies on the grounds that the low blood levels of marijuana found, and the low incidence rate observed, indicate a relatively insignificant factor in automobile accidents. Owens (1981) found detectable levels (3 ug/L) of THC in the blood of 5.9% of a sample of drivers killed in single-vehicle fatal accidents in North Carolina. In contrast, 67% of the sample had ethanol level of .09% or higher. Terhune and Fell (1982) evaluated blood specimens from 497 drivers injured in accidents in the vicinity of Rochester, New York. THC was

detected in 10% of the sample, and alcohol was detected in 25%. Among the positive THC specimens, 51% also had detectable ethanol levels. The alcohol and THC subjects were more often judged responsible for the accident than drug-free and other drug groups. More important, they point out that no study to date has included the necessary controls for establishing the "population-at-risk" baseline necessary to make epidemiologically sound estimates of marijuana's casual role in traffic accidents. Nevertheless, the apparent frequent use of marijuana in conjunction with driving, particularly among drivers in the young, high-risk age groups and in frequent combination with alcohol, is sufficient cause for concern.

Overview and Implications

Although most of the studies reviewed indicate that marijuana impairs driving-related psychomotor and perceptual skills, there is still a notable amount of variability and conflict in the results of these prior investigations. Moreover, the question of the combined effect of alcohol and marijuana has not been as extensively evaluated. The preponderance of evidence indicates that the effects of the two drugs are primarily additive, but some investigators have reported both antagonistic and synergistic interactions. Some of the conflicting results can be potentially explained by: (1) differences in the performance tasks, (2) differences in marijuana dosage, (3) subject differences in the prior use frequency of marijuana and alcohol (tolerance and adaptation), (4) differences in the measurement sensitivity and reliability of the performance measures, (5) differences in research design, particularly with respect to repeated measures vs. independent group designs, and (6) experimental artifacts.

Most of the studies reviewed have not tested for other drugs or established the quantitative level of THC attained by their subjects. The research design of the present study was developed to answer the objectives listed at the outset of this paper. These objectives, in part, emanated from the aforementioned ambiguities in the existing state of knowledge regarding marijuana's impact on driving behavior. The research design, which is detailed in the next section of this report, has the following salient characteristics:

1. Included a closed-course driving range and a wide range of other performance modalities in order to permit a better understanding of the underlying drive performance dimensions and to provide an assessment of the multivariate impact of marijuana and alcohol on driving performance;

2. Utilized independent groups design in assigning subjects to treatment conditions because of concern over the mathematical assumption required for repeated measures design, and potential confounding due to memory and carry-over effects (Gaito, 1958 and 1961);

3. Screen for presence of other drugs;

4. Measured performance over several spaced trials following drug administration in order to establish treatment effect time gradient, and;

5. Tested blood specimens prior to each trial to establish the obtained level of alcohol and cannabinoids for each subject.

It is believed that his combination of refinements has not been employed in any prior study and would clarify some of the ambiguities surrounding the single and combined effects of marijuana and alcohol on driving.

METHOD

Research Design

Subjects were randomly assigned to the four treatment combinations created by the following 2 x 2 factorial design:

(1) Alcohol plus marijuana placebo;

(2) Marijuana plus alcohol placebo;

(3) Marijuana and alcohol and;

(4) Double placebo.

Following treatment, each subject completed four driving performance trials, spaced approximately one hour apart. Thus, the design can be characterized as a 2 x 2 x 4 factorial with repeated measures on the trial factor. The assignment procedures and independent variables are described below.

1. Alcohol and Placebo Treatments. Subjects received either active or placebo alcoholic drinks. Active drinks consisted of 1.05 ml of ethyl alcohol per kg of body weight administered as 80 proof (40% ethanol) vodka mixed with 3 parts of orange juice. The total dosage was dispensed in 3 drinks, consumed at 10-minute intervals. With elimination and absorption considerations, this dosage is calculated to produce a peak blood-alcohol concentration of approximately .08% mg. The placebo drinks consisted of plain orange juice with a very small amount of vodka floated on the drink surface to disguise the absence of ethanol.

2. Marijuana and Placebo Treatments. Subjects were administered either an active marijuana cigarette or a placebo cigarette. The active marijuana was standardized material rolled into a 1.0 g cigarette issued by the National Institute on Drug Abuse (NIDA) containing 1.9% Delta 9-THC. The placebo marijuana cigarettes were similar to active marijuana in overall appearance but were free of all cannabinoids.

   The subjects smoked the cigarette in an ad-lib fashion but were encouraged to finish the entire cigarette within a 10-minute period.

3. Assignment of Treatments. Each subject was randomly assigned to one treatment condition until a minimum of 20 subjects had completed the study within each of the four treatment conditions. The randomization was accomplished through use of a table of random digits. Data was actually collected for 102 subjects to assure an adequate region for error (subject deletion, etc.).

   Four participants were removed from the data because of indications of drug use within the prescribed "no use" period prior to driving. This is discussed further in the Results section.

Subject Selection

Subjects in the experiment were primarily recruited from college campuses throughout the Sacramento area. They consisted of 102 males selected from

approximately 300 volunteers. These males were U.S. citizens between 21 and 35 years of age, having no felony convictions and possessing a relatively good driving record. Each subject's California driving record was examined to verify that none had a two-point offense over the prior three-year period. In addition, their driving records were screened for one-point offenses not to exceed four accumulated in one year, six in two years, or eight in three years (Grossen et al 1981).

Selection criteria required moderate use of both alcohol and marijuana (not necessarily used in combination). This assured that each subject was well acquainted with the effects of the drugs, but were not heavy users of either drug. Moderate alcohol use was defined as a consumption of no less than 6 oz. and no more the 35 oz. of 40% ethyl alcohol or its equivalent per week. Moderate marijuana use was determined by a personal questionnaire and a psychiatric-medical interview, and had to conform to the following criteria:

1. First use at least two years prior to testing.

2. Sufficient use to produce familiarity with the sensations and effects on performance which are characteristic of marijuana intoxication, and some experience in the use of these drugs while driving an automobile.

3. Evidence of ability to smoke moderately large amounts (comparable to or higher than those to be issued in the experiment) without severe discomfort, i.e., "bad trips" or grossly aberrant behavior.

4. Recent use consisting of 1-to-7 average potency (5-20 mg THC content) "joints" weekly for the past 3 months (not more than 1 daily, nor less than 1 weekly).

5. Evidence from laboratory analysis of urine samples taken prior to and during testing that subject has not used significant amounts of other psychoactive drugs in the period immediately prior to administration of alcohol and/or marijuana.

6. Evidence from laboratory RIA assay of blood drawn prior to smoking marijuana that subject has not recently smoked or ingested significant quantities of marijuana.

Necessarily, considerable reliance had to be placed upon subject's own testimony in judging the degree to which they had been "regularly using" marijuana and alcohol. This was evaluated critically in a psychiatric interview. A psychiatrist specializing in both psychopharmacology and clinical aspects of substance abuse conducted these interviews.

The Minnesota Multiphasic Personality Inventory (MMPI) was administered to each subject in an attempt to minimize any adverse psychological reactions during drug-treatment testing. Only subjects scoring under 80 on each of the standard clinical scales of the MMPI were included in the study. A few subjects scored between 70 and 79 and were only included based on satisfactory psychiatric interview results. These criteria have been adopted by the U.S. Army Medical Research Laboratories at Edgewood Arsenal, Maryland, in the selection of military volunteers for studies with psychoactive drugs. This application of the MMPI has also been employed by Burns and Moskowitz (1980), Sharma (1975), and Crancer et al (1969) to "exclude persons showing a combination of psychological stress and inflexible defense patterns". All subjects were

considered physically healthy based on medical information obtained in the psychiatric interview and personal history questionnaire. Each subject completed a two-page questionnaire (Grossen et al 1981) providing basic demographic and general health information, driving experience, and information regarding experiences with alcohol, marijuana and other drugs. Other requirements for participation included the absence of significant amounts of other psychoactive drugs in the period immediately prior to participation. Four subjects who qualified for the study in all other aspects were not used in the final analysis because significant levels of cocaine and/or amphetamines were detected in urine samples collected just prior to driving on the course.

All of the subjects were informed as to the nature and requirements of the study and provided with an informed consent document. The Informed Consent Document (Grossen et al 1981) outlined all possible risks involved in participating in this study. All potential participants were assured confidentiality and freedom from prosecution for any activities required of them during their participation in the study. Upon satisfactory completion of their participation, they were paid $50 plus-or-minus funds accrued from the risk task.

Subject's Safety

The risk to all participants was minimized by the following measures:

1. Careful attention to selection of physically and psychologically healthy subjects known to have good driving records.

2. Seat belt and shoulder harness were required for each vehicle occupant.

3. Limitation of dosage to levels normally used socially and demonstrated by previously published studies to produce only moderate impairment of cognitive, psychomotor and physical performance abilities.

4. Presence of a physician on location throughout all sessions in which drugs were administered.

5. Housing of subjects in air-conditioned recreational vehicles equipped with lavatory and running water and stocked with emergency medical supplies and equipment adequate to provide emergency treatment.

6. Availability of emergency transportation (including police helicopter) to assist in the movement of any injured or seriously-ill volunteer to a nearby medical facility.

7. Limitation of subject's exposure to only one day at the driving range.

8. Minimum number of outside observers utilized on the driving range.

Performance Assessment

Driving performance was assessed on a closed-driving course. The course configuration was designed to sample a subset of critical elements of the driving task. Based on systems analyses of the drive task, human-factor theorists ordinarily differentiate driving into four subtasks:

a. Search-and-scan subtasks--processes in which information from the environment impinges upon the driver through the driver's conscious manipulations of sensory receptors.

b. Perceptual subtask--processes related to the driver's identification and recognition of relevant clues within the driving environment.

c. Decision-making subtask--decision making and judgemental processes involved in translating perceived cues into motor responses.

d. Physical-responses subtask--motor response processes involved in the lateral and vertical maintenance of vehicle in the driving environment.

While marijuana and/or alcohol may affect all four components, this study principally provided indices of impairment within the realm of physical response. However, detection of impairment within any of the activities constituting the physical response subtask may represent impacts on motor activities through indirect impacts on some of the higher level tasks (e.g., impaired decision-making, increased risk-taking, impaired time-sharing between information sources, impaired time-perception, etc.).

Driving Course

The driving course utilized during the experiment was located at the California Highway Patrol Academy in Bryte, California. Appendix II presents schematics of the driving course. (Grossen et al 1981) A portion of the course consisted of a skills and urban complex and was located in the northeast corner of the academy. The "extended driving area" is located at the west end.

A typical drive through the complete course required approximately 12 minutes. The order of progression was through the chicane, left turn and stop. Then into the forced lane change and recovery stop and detour. The course then led to the urban driving area and, upon completion, to the extended drive where one lap was completed. The extended drive ended at the risk task exercise which led back to the skills driving complex.

The skills driving complex measured specialized skill functions: the ability to react swiftly and appropriately to emergency situations and to assess potential danger. The driving tasks consisted of (1) chicane, (2) forced lane change, (3) urban drive, and (4) risk task. Each individual task was separated either by right- and left-hand turns, stop signs, straightaways, or various combinations of each.

The chicane was a 500 foot (155.4 m) meandering channel 9-10 feet (3 m) in width. Bordering each side of the chicane were 40, 3.5 foot (1.1 m) stanchions placed an average of 12 feet (3.6 m) apart. Subjects were encouraged to negotiate the chicane as rapidly as safe driving permitted, touching or hitting the fewest number of stanchions possible.

The forced lane change was analogous to an accident or obstacle avoidance situation. Decisions were required to correctly react in a forced choice driving situation. The simulation consisted of three, 12 foot (3.5 m) wide lanes set apart by traffic cones. At the beginning of each lane were 3 overhead light traffic signals (19 ft. (5.8 m) above the pavement) set on "green". The

vehicle was driven towards the center lane when approaching the simulator. Sixty feet (18 m) from the signals, the vehicles crossed over a pneumatic activating hose which electronically switched two of the green lane signals to red or all light signals to red. The drivers were required to respond to the remaining green light or rapidly come to a smooth, safe stop in the case where all lights were red. The light combinations used throughout the experiment were randomly selected. Subjects were required to maintain the speed of the vehicles between 30 mph (48 kph) and 34 mph (54.4 kph). Failure to do so was tantamount to failure of the exercise. The speed of the vehicle was electronically timed at the critical decision point by automatic timers. The subject was instructed to repeat the task upon failure. Only one repeat was allowed for any given run. The rationale for tight control of vehicular speed through the forced lane change procedure was twofold. First, correctly performing the exercise at speeds greater than 34 mph was extremely difficult, even for the practiced and unimpaired drivers. Second, performance of the task at speeds below 30 mph was relatively easy. Thus, for the forced lane change to have potential discriminating power, it was necessary to impose a minimum speed requirement.

Upon completion of the forced lane change, the vehicles were maneuvered back into the center lane and were stopped at a stanchion located in the center lane, 142 ft. (43.3 m) beyond the overhead lights. The automobiles were then backed out of the chute, stopped and "detoured" through a left curve of two rows of cones placed 9-10 ft. (3 m) apart and stopped at a stop sign. The cars entered the urban drive, a varying course determined by one of six written instructions presented to the subject to direct him through a series of street grids consisting of 7, north-south streets and 4, east-west streets. This exercise simulated an urban drive over unfamiliar and varying routes. Drivers were instructed to obey all street signs and to exercise the proper rules of the road. The subjects were directed upon completion of this drive (duration two-to-three minutes) to the extended drive portion of the course.

The second portion, or extended driving course, consisted of a 2-mile (3.2 km) track designed primarily to simulate open-road travel, with problems similar to those encountered in this type of driving. Included were a road detour and a stop. At two points along the course, 25 and 35 mph speed limit signs were posted. After coming to a stop at the stop sign located approximately three-quarters-of-a-mile from the end of the extended drive, the car's speedometer was covered, and the subject was asked to accelerate to 35 mph and maintain that speed. The speed of the vehicle at this point was monitored by the inside rater (LRE) with a driver obscured-view, digital speed indicator. The speed was also continuously recorded on the split image videotape from the speed sensor.

Exiting the extended drive, the vehicle was stopped and the speedometer was uncovered prior to entering the risk task.

The risk task was designed to measure the subject's willingness to gamble on the accuracy of his perception of road objects and his driving ability (Grossen et al 1981, Appendix 3A). The driver waged a sum of money on his ability to traverse a series of variable-width gates, one which was impossible to clear without hitting the delineating stanchions. In another closed-course driving study, Casswell, 1979, employed a similar risk-task in which subjects were required to use their judgement in deciding if they could negotiate a gap varying in width from .0 to .5 m wider than the vehicle. In the present study, the gap varied in width from 2.02 to 1.71 m and the subject was required to maintain a speed of 30 mph. (The width of the vehicles was 1.71 m.) Upon completion of the risk task, subjects had negotiated one complete circuit of the course and returned to the starting, parked position.

The driving course was designed to sample the following driving functions and sub-tasks: (1) steering, (2) braking, (3) lateral road placement, (4) risk, (5) speed and (6) reaction to forced lane changes. Various measures of performance were derived from these tasks. They included various reaction time and quality measures, decision making skills and vehicle control, cognitive and coordination skills. The specific driving skills included stopping and speed control, steering errors and control, and eye/hand coordination. The operational definition of each variable at its most molecular level of measurement can be deduced by reference to the appropriate exhibit or appendix of the protocol (Grossen et al 1981).

Use of Vehicles

The two vehicles utilized on the driving course were 1978 Mercury Zephyrs powered by 302 cu inch V-8 engines. The cars were equipped with radial tires, power brakes, power steering, air conditioning, and automatic transmissions. Dual brake systems were added to each vehicle to enable front seat passengers to slow or stop the cars independently of the drivers. Each subject was assigned to one-of-the-two vehicles for use on the driving course and drove all trials in that car.

Automated Recording Procedures

Some measures of driving performance were collected by electronic sensors and video tape recording equipment. The complete data recording system was housed in the trunk of the test vehicles. It consisted of six components: (1) a video cassette recorder; (2) a video screen splitter; (3) a recorder charger-adapter; (4) an AC/DC power inverter; (5) a power supply filter; and (6) a 12V 550A auxiliary power supply.

Each vehicle was equipped with two video cameras and four sensors: speed, brake, accelerator, and steering. One camera was mounted on top of the vehicle and provided a wide-angle, black-and-white image of the roadway in front of the vehicle. The other camera was located in the trunk and was focused on a 20 cm x 1.5 cm LED bar containing the sensors' light emitting diodes (LEDs). The two camera images were merged together to form a single picture.

The LED bar contained, from left-to-right, 1 diode for the speed sensor, 10 diodes for the brake sensor, 10 diodes for the accelerator sensor, and 20 diodes for the steering sensor. The speed sensor LED, by pulsing with each wheel rotation, provided a measure of velocity. Brake and accelerator pedal movement was indicated by an increase in the number of lit LEDs from 0 to a level proportioned to the amount the pedal was depressed. The steering sensor, when centered, lit 10 of its 20 LEDs. Ninety degrees of steering wheel rotation to the left would linearly decrease the number of lit LEDs from ten to zero. Similarly, rotation to the right would linearly increase the number of lit LEDs from 10 to 20.

The video cassette recorder (VCR) captured 5 measurements per frame at a rate of 30 frames per second. After the video recording had been completed, the video cassette tapes were processed using a digital graphics circuit board (32 Kilobyte random access memory) mounted in a Northstar microcomputer chassis (64 kilobyte random access memory). The video signal transmitted to the digital graphic (DG) printed circuit board was an analog signal. The DG board converted the analog signal to a digital signal at a threshold level adjusted by contrast control. The digitized video signal was sent simultaneously to a video monitor and to the circuit boards' 32K RAM.

To measure the number of LEDs lit by each vehicle-mounted sensor, a program was written to check the value of the bit at that location in the computer memory associated with a specific LED or set of LEDs. The line "sensor", on the other hand, was a computer-generated measure of the position of the vehicle in the lane. This was achieved by measuring the vehicle's distance from the white-fog line on the side of the road, which was easily distinguished from the asphalt once the image had been digitized and read into the computer memory (RAM). To determine the value to be assigned to the line sensor, a screen location (i.e., memory address) several feet in front of the vehicle was chosen as a reference point. Under the control of the computer program, the number of zero bits (black) between the reference point and the white-fog line were tallied. When a one bit (white) was found, the tally ceased. This measured value, the number of zero bits, represents the distance in arbitrary units of the vehicle from the fog line. This sensitive measure was able to detect change as small as one cm. The measurement procedure was repeated at a rate of 30 frames/second. When the digitization of a section of the course was completed, the contents of the 64K RAM was written onto a 5-1/4 inch diskette. The first four moments of the digitized signals were computed for each of the four sensor variables.

Following digitization, it was found that sensor malfunction had introduced a considerable number of errors. A series of programs were written to process the sensor data; identifying and removing errors where possible. The data reconstruction process utilized algorithms similar to those commonly used in the filtering and smoothing of continuous data. The reconstruction process and results are described in detail by Kerslake (1983). Although the first four moments of the score distributions were computed for each subject, and "difference scores" were computed to represent continuous and discrete changes in the score values over the course of each trial, only the first moment of the non-differenced scores was used for this analysis.

Other drug studies which have employed analog recording procedures include Attwood et al (1981), Casswell (1979) and Smiley et al (1974).

Subjective Dependent Variables

Outsider Rater Procedures - Observations of specific task performance were made by specially-trained raters stationed at strategic locations on the course.

a. Chicane - Two or more raters were stationed along the chicane. The lead rater recorded the time through the chicane and the total number of stanchions touched or knocked over as observed by all of the raters. The scoring sheet and instructions are in Appendix 3B, page 96, of the Protocol, (Grossen et al 1981). The raters were responsible for observing touched or downed stanchions along a particular span of the chicane and for reporting this to the lead rater. After each drive, the chicane raters were responsible for repositioning any stanchions that had been knocked out of place.

b. Forced lane change - At least one rater was stationed at the forced lane change and was required to control the sequence of light changes and score the vehicular performance. Instructions, including randomized light changes, and scoring sheets are in Appendix 3C, page 99, of the Protocol. This rater also monitored time to complete the task and directed a repeat of the exercise if the task was completed at either an excessive or reduced speed.

c. Risk task - A rater was responsible for setting stanchions and "dollar-value" signs at the three gates of the risk task. Each rater was responsible for all settings and scoring related to his gate. Instructions and scoring sheets for the risk task are in Appendix 3A, page 67, of the Protocol.

License Registration Examiners Procedures

Four specifically-trained California DMV License Registration Examiners (LREs) provided further measures of driving performance. The LRE scoring form is shown in Appendix II. It included both subjective judgements of driving performance on specific items throughout the course as well as objective speed and elapsed-time measures. Detailed instructions for using the rating form and assessing the scores are contained in Appendix 6B and 6C of the 1981 Protocol. During each trial, an LRE accompanied the driver and had access to a dual braking system. They were required to avoid reference to performance ratings when in the presence of the subjects. The LREs were required to review a comprehensive set of instructions. They included precise techniques for reducing experimenter bias and variance in the LRE ratings. Specific instructions presented item-by-item directions for scoring each individual task specified on the scoring sheets. Each LRE rater was assigned to one car and rated the performance of all subjects driving that vehicle. Subjects and LREs were randomly assigned to the vehicles and remained with that vehicle throughout the day. The LREs were never informed of what treatments the subjects had received. At no time were the LREs allowed to communicate with subjects other than in the course of the driving task.

To prevent any systematic rater-bias from confounding the treatments, an attempt was made to assign the same proportion of each treatment condition to the LREs. A chi-square test of independence between LREs and treatment did not approach significance.

The LREs were trained until interrater consistency was high and in conformance with the intent of each measurement dimension. In some cases, dimensions and criteria were altered to eliminate ambiguity. A small-scale, pilot study was conducted prior to running subjects in which one of the research staff drove the course and was rated by the LREs. The reliabilities were high, and there were no significant differences between the raters.

The assignment of the same LRE to all trials of a given subject was a logistical requirement. It would have been desirable to have different LREs rate each trial, or at least every other trial per subject. Such a procedure would have assured independence between trials and minimized the potential for halo effects.

California Highway Patrol Following Car

California Highway Patrol (CHP) Officers observed and evaluated vehicular motion on the extended drive and made assessments of impairment comparable to actual highway evaluations. The CHP officer followed the subject's vehicle in an unmarked car. Utilizing a cassette recorder, the officer verbally recorded all of his/her observations and judgements of the subject's driving performance, just as he/she would have non-verbally assessed the subject's driving abilities if he/she were following on the highway. Each officer was instructed to verbally record his/her "stream of thought" while observing the subject's

vehicle. He/she was also asked to estimate the distance from the subject's car and any landmarks which might serve as convenient points of reference during his/her monologue. The officers were unaware of what treatment the subjects received, in order to prevent bias and criterion contamination.

Field Sobriety Test

To each subject, upon completion of each experimental drive, a second CHP Officer explained and demonstrated the tasks involved in the field sobriety test (FST). Eight separate tasks were included. They were: (1) Romberg body sway; (2) finger to nose; (3) heel-to-toe; (4) standing on one foot; (5) finger count; (6) hand pat; (7) counting backwards; and (8) reciting the alphabet. Scoring forms and instructions to the administering officer are in Appendix 8 of the 1981 Protocol. The administering officer used a scale of either 1 - 4 or 1, 2, and 4 to rate performance on each task with the scale being anchored on satisfactory performance of the task. Using body sway as an example, the following scale would apply: (1) slight sway, (2) missed, (3) pronounced sway, and (4) failed performance. Precise instructions for applying this scale were included for the officers' review. After completing the individual ratings for each task, the officer assessed the subject's overall impairment on a scale of 0 - 9, where 0 represented no noticeable impairment and 9 represented extremely impaired performance. The officer then asked the subject to assess his own overall driving and FST performance on a similar 0 - 9 point scale. For this scale, 0 represented no noticeable impairment and 9 represented "the greatest impairment that the subject had ever experienced while smoking marijuana, drinking alcohol, or both". All subject, LRE, and officer ratings were double-blind. That is, neither the subject nor rater were informed of the treatment the subject received. In addition, the FST and car-following tasks always utilized different officers to prevent cross contamination.

Miscellaneous Measures

Although driving skills were central to the current investigation, time on the course was less than 20-minutes-per-hour and it was therefore possible to schedule other observations during the intervening periods. Several additional variables were chosen on the basis of their medical and psychological significance, as well as their use by other investigators in previous studies (e.g., Weil and Zinberg 1968; Tinklenberg et al 1972; Renault et al 1974; Peter et al 1975; Milstein et al 1975; Manno et al 1979; Kiplinger 1971; Galanter et al 1972; Belgrave et al 1979).

a. Blood Pressure (BP) and Heart Rate (HR) - These physiological measures are traditionally monitored when drugs having significant autonomic effects are used in human subjects. Heart rate characteristically increases following the ingestion of alcohol and is even more noticeable after smoking marijuana. Blood pressure sometimes declines precipitously after marijuana ingestion, and occasionally lightheadedness or even syncope may result. During the study, each subject's blood pressure was recorded by a registered nurse at hourly intervals while the subject was seated in a relaxed position after a period of minimal activity. HR was counted for 30 seconds at these times.

b. Symptom/Sign Rating Scale (SRS) - Because the symptoms (subjective observations) and signs (objective observations) accompanying use of alcohol and marijuana were potentially valuable predictors of driving performance and correlates of intoxication, the SRS was developed for tapping a variety

of these measures. A psychiatrist or registered nurse systematically observed subjects for signs of intoxication (e.g., euphoria, verbosity, irritability, incoordination, slurred speech, suffused conjuctiva, paranoid suspiciousness, hyperactivity, etc.) and recorded the degree to which each was present on a simple scale (0 = not at all; 1 = slightly or to a mild degree; 2 = considerably or to a marked degree). Subjects, likewise, rated the degree to which certain characteristic symptoms were present (e.g., feeling "high" or "stoned", confident, sleepy, dull, irritable, etc.). Observations were recorded on a special form (Protocol, Grossen et al 1981, Appendix 10C, R182) at hourly intervals.

c. Critical Tracking Task (CTT) - The CTT was developed specifically to detect alcohol impairment and has been found to be sensitive to relatively low-alcohol doses if administered after stable baseline performance has been established (Klein and Jex, 1975). Although evidence of sensitivity to marijuana is less clear cut, some investigators have reported evidence of a dose-related, negative effect (Sharma, 1975). The detailed operations involved in the CTT are contained in Appendix 5 of the 1981 Protocal.

Summarized briefly, the CTT presents an electronically controlled interactive visual display (cathode-ray tube), which must be controlled through manual operation of a joy stick or dial. The stimulus task involves keeping a randomly oscillating needle within the lateral boundary of the display by appropriate manipulation of the response dial. The task increases in difficulty in response to a subject's performance until the subject is no longer able to control the oscillating needle. Each subject completed a series of CTT trials until a learning plateau was achieved. The task automatically terminates when the subject is no longer able to control the stimulus and the score is then recorded. Higher scores indicate that a subject was able to control the stimulus for a longer period and achieve a higher level of difficulty.

d. Brief Interval Time Estimation (BITE) - This task measured time distortion effects of drug treatment by requiring the subject to reproduce a brief interval of time upon presentation. The BITE involved repeated production of a constant brief interval (5 seconds). The subject attempted to stop a timepiece as close to five seconds as he was able. He was allowed to verbally count or tap out the five-second interval and was then required to stop the counter at precisely five seconds. A digital watch/timer by Casio Computer Co., LTD. was used to perform this task. Details and scoring sheets for the BITE task are found in Appendix 4, page 110, of the Protocol.

Subjects were given 50 practice trials prior to driving, at which time the average error was less than 0.1 of a second. Similar tasks were utilized by Sidell and Pless (1970), who used a variable time analyzer, and Walker (1960), who used a zero input tracking analyzer.

e. Impairment Questionnaire - The purpose of this questionnaire was to assess the subject's perceptions of any changes that may have occurred as a result of their drug-alcohol treatment. It was important to investigate the relationship between perceived change in abilities and actual change in driving performance as measured by objective criteria. The questionnaire was given to the subjects after the third and fifth driving trials. The complete impairment questionnaire, devised by Tart (1977), is found in the Protocol.

f. Exit Questionnaire - This questionnaire was given to the subjects upon completion of all other testing procedures. Its primary focus was to determine the effect of learning on performance. It contained questions to assess and compare the subject's prior experience with driving while impaired to the experience of driving in the experimental condition. Other questions assessed learning strategies employed during the experiment. It was believed that subjects with greater experience in drug-impaired driving would possibly perform the driving tasks differently than subjects with less comparable experience.

General Procedure

Preliminary learning sessions for performing the CTT and BITE were conducted during an evening of the week prior to the subject's driving session assignment. This initial learning session consisted of 50 repetitions of the BITE distributed over 5 sets of 10 repetitions each. Interspersed between sets, the CTT was practiced in sets of 5 for a total of 25 repetitions. Subjects were examined for color blindness, height and weight, and then reminded that they were not to consume any drugs 24 hours prior to their driving day.

Actual driving for the study was done on weekends throughout the summer of 1981. Twelve subjects were assigned to drive on each experimental weekend (six on Saturday, and six on Sunday).

On each day of driving, the six designated subjects were picked up at their homes. When the sixth subject was in the limousine-van, a tape recorded set of comprehensive instructions was played enroute to the driving course. These instructions appear in Appendix 2B of the Protocol. Upon arrival, subjects were assigned an identifying letter which was worn throughout the day. The subjects drove in the sequential order of these letters consisting of "A" through "F". Subjects A, C, and E were assigned to one of the cars and subjects B, D, and F were assigned to the other. In their assigned car, subjects were driven together through the entire course to familiarize them with the driving route and the tasks to be performed. The drivers for this familiarization run were experimenters who could answer any remaining questions the subjects may have had.

Subjects were then given a standard breakfast. Breakfast and lunch were provided in order to control the calorie intake of each subject.

The first subject (subject A) then began a practice drive intended to allow him to adjust to the assigned car and the course. This drive was not scored in any way. Ten minutes into the first subject's initial drive, the second subject (subject B) began his practice drive. This 10-minute lag assured that the two cars would never be in the same section of the course at the same time. This progression continued until all of the subjects completed the practice drive. Although the drive was not scored, a license registration examiner rode with the subject to assure safety and to familiarize the subjects with the accompanying observer. After completing the practice drive, each subject performed 25 repetitions of the BITE and at least 25 repetitions of the CTT. Again, this was considered practice and was not scored. Each subject, in turn, proceeded to the clinical station, at which time his blood pressure was measured and ca 27 ml blood and ca 20 ml urine were collected. During this period, a battery of clinical questions pertaining to the general health and mental status of the subject was assessed and recorded.

Upon completion of this preliminary round, the subjects, in turn, performed the first scored drive of the day. These drives were again staggered at 10-minute intervals. This preimpairment trial provided baseline measures of performance on the driving course and on the other measures. For this trial, each subject progressed through the driving course in the same order as previously described. Completing the skills, urban and extended driving portions of the course, the subject parked the car and was led to the field sobriety test area where he was instructed in the eight separate coordination and/or cognitive skills tasks presently used by law enforcement agencies to detect signs of alcohol impaired performance. The field sobriety tests were conducted by a California Highway Patrol Officer, and the entire test was videotaped.

After completing the field sobriety, test the subject entered the BITE and CTT station to perform another 20-minute block of tests.

Completing this block of tasks, the subject returned to the clinical station to begin the impairment session. During this time, he was given 30 minutes in which to drink three drinks containing either a combination of alcohol and orange juice in proportion to his height and weight or a combination of orange juice with a small amount of alcohol floated on top to mask the placebo condition. The subject was asked to consume the entire amount of each drink.

Following the administration of alcohol, the subject was given a marijuana cigarette and asked to smoke as much of it as he was able. The 1.0 g (NIDA) cigarettes contained either 1.9% Delta 9-THC or marijuana with Delta 9-THC extracted. The subject was allowed 10 minutes in which to smoke ad lib, consuming as much of the cigarette as he could. The entire cigarette was consumed in almost all instances. This was succeeded by another 20-minute-clinical block which consisted of drawing a second blood sample, monitoring blood pressure and taking a breath sample with an INTOXILYZER Breath-Alcohol testing device. The breath-alcohol reading was concealed from the subject, and only the administering "bartender" observed and documented the reading.

The subject then began his first impairment drive. This, as well as the three remaining drives, was scored in the same manner as the baseline drive. All tasks and routes were identical to those of the baseline drive, with the single exception that there was a different urban route for each drive. The subject, therefore, drove a total of six different routes through the urban drive area. During every driving session, each subject was instructed, by the accompanying LRE, to follow all normal driving rules and precautions.

Each remaining driving session was succeeded by a field sobriety test, a 20-minute BITE and CTT session, and a 20-minute clinical session. Therefore, each remaining cycle duplicated the first impairment run with the following exceptions: After the third and fifth drives and following the BITE and CTT sessions, the subjects completed a modified version of the define-impairment questionnaire developed by Tart (1977). After completing the BITE and CTT on the fifth drive, the subject was given a standard lunch consisting of a hamburger and a non-caffinated soft drink. During the clinical examination period following the sixth driving run, the subject provided a second urine sample and completed the exit questionnaire.

Each subject was accompanied by the same LRE during each subsequent drive, and instructions and precautions were read to him by the LRE. All conversations in the car and at the field sobriety test station were audio-tape recorded.

Between driving tasks, the subjects were not allowed to talk with experimenters or raters and were continuously observed. They were allowed to talk among themselves. All subjects were identically instructed and monitored.

Prizes

As part of the tape-recorded set of instructions, it was explained to the subjects that prizes would be awarded to the subject achieving the highest and second-highest performance scores of the day. The person with the highest accumulation of points received his choice of either a chilled bottle of wine or two, six-packs of beer. The person with the second highest point accumulation received a six-pack of beer.

Subjects were told that to accumulate points, their best strategy would be to drive using all normal driving precautions and procedures as would be used in other normal driving situations.

The prizes were awarded in an effort to keep the driving day interesting for the subjects and to motivate them to drive and perform the tasks as well as they could.

After the sixth and final driving session, subjects completed a questionnaire assessment of their perceived performance and contribution to the study. When the sixth and final subject had completed all tasks, the prizes were awarded, and subjects were debriefed and driven home.

Quality Control

Experimental quality control was divided into two areas of responsibility. One individual insured that subjects moved from station-to-station at 10-minute intervals. The same individual was responsible for the double-blind administration of treatments, and proper collections and recording of biological specimens.

A different individual was on-site to monitor the quality and completeness of data entries. This individual insured that raters and electronic equipment were in position and operational. The monitors also had the responsibility for all data acquisition and recording techniques, under the supervision of the quality control manager.

Final Data Reduction

The selection of the final reduced set of performance measures was based on a series of factor analyses. The first step in this process was to perform separate factor analyses within each of the four basic measurement domains: (1) license registration examiner (inside rater) measures; (2) vehicle sensor measures; (3) outside rater measures; and (4) field sobriety test measures. The risk task measures were included as part of the LRE factor analysis. Three risk variables were evaluated: (1) number of attempts on the Risk Task; (2) quality of performance as judged by the LRE; and (3) the monetary value of the successful attempts or risk choice. A principal component analysis (Harmon, 1960) was employed to factor each of the domains using the SPSS computer program (version 7). Eigenvalues of one or more were required for retaining factors, and the varimax rotational procedure was used to achieve simple orthogonal structure.

The second phase of the data reduction consisted of a "global analysis" in which the factors and their defining variables from the domain analyses were combined into one analysis and factored a second time.

The within-domain factor scores used as input for this global analysis were created by simply summing the variables possessing the highest-factor loadings for a given variable, usually after converting to standardized form. This technique for computing composite scores was elected over the more conventional multiple regression weighting procedure because of its computational simplicity. For additional rationale, see Tryon and Bailey (1960).

The primary purpose of the global factor analysis was not to further reduce dimensionality but, rather, to provide insights on the nature and structure of the total array of performance measures. As such, the primary output of interest was the factor-loading matrix portraying the correlation between the resultant factors and each variable. It should be noted that other outputs of this analysis, such as the communality and variance allocations, contain spurious elements, due to the substantial redundancies among some variables (i.e., the inclusion of both factors and their composite variables from the previous domain analyses).

Summarized below are additional methodological details pertaining to each of the factor analyses:

1. LRE Domain - It will be recalled that the Urban Drive portion of each run was varied slightly to minimize familiarity effects. This strategy created a potential source of variance within and across runs, since the Urban Drive LRE ratings were no longer based on identical task elements. This complication was handled by first factoring the data without the Urban Drive scales and then performing separate factor analyses on these omitted scales.

   Before performing the preceding factor analyses, the 76 items on the scoring sheet were reduced to 61 variables by deleting items with inadequate variability and extreme skewness. A 61-variable correlation matrix was produced within each run and for runs 2-6 combined. Run 1 was a training trial and is not included in the analysis. These matrices were factor analyzed, and a similar analysis was repeated for the urban drive scales. Based on the similarity of the factor structure across runs and a desire to maximize the reliability of the subjective LRE scales, it was decided to select the factor solution which resulted from the total composite run correlation matrix.

   Although the selection of the number of factors was based on inspection of the total composite run analyses, in most instances only the baseline (Run 2) scores were used in determining the exact variables to be used in forming the factor composite scores. Scale differences resulting from the particular urban route assigned to the subjects were removed through simple normalization (averaging) procedures.

2. Outside Rater Domain - Seven variables were factor analyzed, and the factor scores were based only on the run 2 analysis.

3. Sensor Measure Domain - After reconstruction of these data in accordance with previously described procedures, separate factor analyses were run on the 18 vehicle sensor variables for each of the 6 runs. The factor solution for run 2 was used to compute composite scores.

4. Field Sobriety Test Domain - Performance on the nine variables at baseline (run 2) were factor analyzed and the factor scores computed based on that result.

5. Global Analysis - The factor scores and the defining variables from the preceding analyses were subjected to another factor analysis, along with certain other variables of a prior interest (the BITE, CTT, and car-following ratings). As described previously, the primary purpose of this global analysis was to obtain information on the structural relationship between the various domains. Based on an evaluation of the within-domain and global-factor analyses, 21 measures were selected as criterion measures for evaluating treatment effects. Six additional variables were included due to their substantial importantance, producing a total of 27 measures. (This set was increased to 29 and 30 for some analyses.)

Reliability Analysis

The stability of the measurements was determined by computing test-retest correlations across the separate runs for the factor composites and variables identified from the various factor analyses. The following between-run correlations were computed for each measure: (1) average of all pair-wise correlations between runs, including run 2; (2) average of all pair-wise correlations between runs, excluding run 2; (3) average pair-wise correlations between run 2 and each of the post-treatment runs--that is,

$$\frac{(R_{23,} + R_{24,} + R_{25,} + R_{26})}{4}$$

The systematic manipulation of run 2 in computing the above averages was due to the baseline function of this run. It was hypothesized that the post treatment trials would be influenced by both drug- and learning-effects.

If so, then the inclusion or exclusion of run 2 should have a substantial effect on the test-retest correlations. This logic also reveals a limitation of these test-retest correlations as measures of reliability. Since most of the subjects were treated after run 2 and each subject repeated the route several times, one would expect nonrandom variations in the performance scores across trials. Such effects would tend to attenuate the intrinsic test-retest reliability of the measures. The alternative of using only the placebo group to estimate reliability was rejected because of the small sample size.

Evaluation of Treatment Group Equivalency

Although the random assignment procedures would be expected to preclude bias in the composition of the four treatment groups, a small number of subjects had to be deleted for a variety of reasons (instrument malfunction, presence of other drugs in blood, etc.).

Three sets of analyses were conducted to verify the equivalency of the final groups on pretreatment variables. First, the groups were compared on some of the biographical and interview variables through chi-square and F tests. Second, the groups were compared on the final 27 performance factors, as measured at baseline (run 2). This was done through 27 separate one-way ANOVAS. Finally, a multiple-discriminant-function analysis (MDF) was run on the baseline scores for the 12 most significant discriminators of postdrug performance. This latter analysis was considered the most important because it

measures whether the groups were biased on the measures showing the strongest treatment effects. The Wilks' Lambda statistic was used to evaluate the statistical significance of the discriminant functions. A non-significant outcome at run 2 would be interpreted as evidence of treatment group equivalency.

Test for Potential Covariates

In order to increase the sensitivity of the experiment, a comprehensive search was made for potential covariates -- that is, variables which were substantially correlated with post-treatment performance.

Two pools of variables were evaluated. The first set consisted of five background items from the screening interview form. The matrix of correlations between these variables and the post-treatment performance measures on runs 3-6, combined, were inspected for the presence of statistically meaningful correlations.

The 12 best discriminators of treatment effects comprised the second pool of variables. Performance on each of these measures at baseline (run 2) was regressed against their respective post-treatment counterparts at run 3 and runs 3-6 combined.

The above analysis, in essence, represents the relationship between pre-test and post-test performance and was expected to yield high correlations. As such, the baseline measures were considered to be likely covariates. However, the standard analysis of covariance model requires use of a common regression slope, thereby assuming that the relationship between covariates and the dependent variable is the same for each treatment.

This assumption of parallel slopes was evaluated by testing the significance of the increase in multiple R which occurred when using separate regressions for each treatment (Kerlinger and Pedhazur, 1973).

Evaluation of Treatment Effects

The methods used to evaluate the impact of treatment on driving performance can be divided into five categories: (1) multivariate evaluation of main and interactive effects; (2) univariate evaluation of moderator effects (treatment by subject variable interactions); (3) univariate evaluation of treatment effect time gradients; (4) the mediating effects of cannabinoid and alcohol blood levels; and (5) miscellaneous correlational analyses of selected variables. These analyses are described in detail below:

1. Multivariate Evaluation of Main and Interactive Effects - Treatment effects were evaluated through a series of stepwise multiple-discriminant-function analyses (MDF) in which the 4 treatments comprised the groupings, and the 27 performance dimensions, identified above, comprised the classification variables. Separate MDFs were run for each of the 4 post-treatment trials and for the 4 trials combined. Alpha level of $P < .25$ was used to enter and retain variables in the discriminant functions, and variables which were not significant at this level on 2 or more trials were deleted. After deletion, the MDFs were recomputed on the remaining variables. The requirement of significance on two or more trials was imposed to increase the stability of the discriminant functions; the recomputation of the MDFs on a common core of variables facilitated comparison across trials.

The Wilks' Lambda statistic was used as the significance criterion for extracting functions, with $P \leq .05$ required for significance. Since 4 treatment groups (k) were involved, a maximum of 3 functions (k-1) were extracted in each discriminant analysis. All analyses were computed using the SPSS step-wise MDF routine (version 7).

Interpretation of the functions was based on inspection of the standardized discriminant function weights and, more importantly, the structure loading matrices (Tabachnick & Fidell, 1983). This latter statistic represents the simple correlation between the linear composite scores from each function and a given variable. These loadings were produced for both the variables included in the MDF analyses and for the entire pool of available variables.

The contributions of marijuana, alcohol and their interaction to explaining variations in performance were evaluated through a series of canonical correlations in which the independent variable vector was comprised of the treatment group dummy codes, and the dependent variable vector consisted of the significant discriminators identified from the MDF analyses. The following sequences of effects were evaluated by comparing the increments in the percent of variance explained by the successive canonical models: (1) alcohol dummy code; (2) alcohol and marijuana dummy code; (3) alcohol, marijuana and the alcohol x marijuana interaction dummy codes. A second sequence was also run in which the entry sequence of marijuana and alcohol was reversed. All analyses were run separately for runs 3-6 and 3-6 combined.

The percent of variance explained by the models was calculated as the complement of the Wilks' Lambda statistic (1 -**X**). To avoid sequence bias in the main effect estimates, the results from the two sequences for entering marijuana and alcohol were averaged.

2. Univariate Evaluation of Main and Interactive Effects - The final set of significant discriminators was evaluated univariately through a series of 2 x 2 x 2 analyses of variance in which the three factors were: (1) performance on a baseline task (run 2) or demographic variable; (2) the alcohol treatment condition; and (3) the marijuana condition. The significant discriminators from the MDF analyses comprised the dependent variables.

   The primary focus of this analysis was on the two- and three-way interaction terms involving the background and baseline variables -- that is, on variables which might moderate, or interact with, response to treatment. The following five background variables, in addition to the performance measures at baseline, were evaluated: (1) weekly alcohol use; (2) weekly marijuana use; (3) experience driving under alcohol; (4) experience driving under marijuana; and (5) years of driving experience.

3. Univariate Evaluation of Treatment Effect Time Gradients - A usable number, repeated measures program was not available to the computing facility which performed the majority of the computer data analysis. It was, therefore, necessary to evaluate differences in treatment duration and time gradients through use of discriminant function (MDF) and regression programs. For the MDF analyses, the treatment groups comprised the grouping dimension, and performance on each variable by trial provided the discriminators for a given MDF. Separate analyses were done for each of the performance measures found to be significant in the MDF analyses described earlier. One set of

analyses was done with run 2 included and another set with the baseline run excluded. Although not an explicit test of slope difference hypotheses (treatment x trial interaction), certain patterns of nonlinearity and interaction can be inferred from the number of significant functions extracted. The extraction of more than one significant function would suggest that the pattern of means over trials differed as a function of treatment.

A more explicit test of slope differences was achieved by testing the homogeneity of the within-treatment regressions of trials on performance. This test is the same test used to test for equivalency of regression slopes in many standard analysis of covariance programs and is described in standard references, such as Kerlinger and Pedhazur (1973).

4. Mediating Effects of Cannabinoid and Alcohol Blood Levels - Three sets of analyses were performed to evaluate the significance of the alcohol and cannabinoid blood/serum levels. The first consisted of an analysis of mean levels by trial and treatment to establish the peak blood/serum levels attained by the treatments and the blood/serum metabolization rate of the active chemical constituents.

   The last two analyses sought to evalute the correlation between the actual blood or serum levels and performance decrements. This was done through a series of within-trial and between-trial analyses. The within-trials analysis consisted of a series of canonical analyses in which the quantitative blood levels were included as additional quantitative factors, and the various post-treatment performance measures served as response variables. The magnitude and significance of the increases in the explained variance of the models was evaluated for each additional quantitative factor (blood alcohol, serum THC, and serum THC-carboxy). These analyses were done for each of the post-treatment runs, separately, and for the runs 3-6 combined.

   The between-trials analyses evaluated the extent to which performance variations across trials (trial effects) could be explained by individual variations in blood levels across trials. This was done in two stages: (1) Canonical analyses were run within each trial using the significant discriminators from the MDF as the Y vector and the three blood level variables as the X vector; (2) The resultant pairs of canonical scores were computed for each subject within runs and then stored as new variables for a between-runs canonical analysis. In this latter analysis, the four canonical scores from the previous Y vector (i.e., trial performance measures) comprised a new Y vector, and the canonical scores from previous X vector (trial blood levels) comprised a new X vector. Conceived in this way, the output from the second canonical analysis represents the multivariate relationship between variations in blood levels and performance across trials. Two complete sets of these two-stage, canonical analyses were generated - one with run 2 included and the other excluding run 2.

5. Miscellaneous Correlational Analyses - The relationship between certain subsets of variables was evaluated through an analysis of the appropriate correlation and discriminant function structure (loading) matrices. The variable sets involved in these analyses were the FST, CTT, BITE, risk task, forced lane change, car-following observation, subjective self ratings, and biographical variables.

Several limitations of the statistical analysis warrant mention at this juncture. Most of the statistical techniques used assume a linear parametric model of normally distributed performance scores and homogenous within-group variances. These assumptions were not satisfied by many of the criterion measures obtained in this study. Although MDF and analysis of variance have been found to be relatively robust against violations of the standard parametric assumptions, the obtained significance levels are best viewed as approximations of the true values. Another problem relates to the tendency for MDF analysis to capitalize on chance relationships, particularly when a stepwise variable selection procedure is used. As a result, the discriminating power of the functions are inflated and the accuracy of the functions in discriminating between groups will shrink when applied to an independent sample. For these reasons, we have elected not to report the discriminant function classification matrices.

Analytical Methods for the Detection of Alcohol

The reliability, accuracy, and specificity of methods applied to the quantitation of alcohol in blood, breath, and urine as a measure of impairment of driving skills have been universally established as evidenced by the many chemical test laws now in effect both nationally and internationally (Mason & Dubowski, 1974).

Breath alcohol analysis utilizing the CMI Intoxilyzer Model 4011AW was the method selected for determining all blood alcohol concentrations (BAC) reported in this study. The Intoxilyzer instrument is capable of measuring the alcohol concentration in vapor samples generated by a Breath Alcohol Simulator with a precision and accuracy of 5% of the true alcohol concentration. The alcohol concentration determined from a breath sample is expressed in terms of an equivalent blood alcohol concentration based on the established, average, conversion ratio of 2100 ml Breath to 1 ml Blood.

The 2100:1 alveolar breath/blood conversion ratio has, since 1950, been the accepted standard for the calibration of all evidential breath testers (EBT) designed to analyze alveolar breath samples to determine an equivalent BAC. This ratio is based on Harger et al, comprehensive studies of the partition of ethanol between air and water, blood and urine over a range of temperatures. Many hundreds of breath-to-blood ratio case studies and controlled correlation studies conducted since 1950 with EBT calibrated on the 2100:1 ratio have clearly shown the true average to be closer to 2300:1. Because the intoxilyzers are calibrated using the 2100:1 ratio, we expect that the results obtained underestimate the true BAC in most cases by about 8-to-10% of the BAC value and seldom, if ever, overestimate the BAC by more than .01% (Dubowski, 1974; Jones, 1981).

Methods Applied to the Analysis of Cannabinoids in Body Fluids

The methods for cannabinoid analysis used in this study were selected to fulfill two main objectives: (1) to determine if a correlation could be established between quantitative levels of Delta 9 THC and/or other THC metabolites in blood and/or serum samples; and (2) to determine to what degree the methods applied to the cannabinoid analysis in blood and/or serum would meet the requirements of accuracy and specificity required for eventual implementation in a forensic DUI Program.

When this study was being planned in early 1981, it appeared from the literature and consultation with other researchers that some combination of radio-immunoassay (RIA) and GC/MS techniques, as applied to whole blood or serum samples obtained from suspected DUI drivers, would provide the requisite accuracy and specificity needed for establishing a correlation between quantitative levels of Delta 9-THC and/or THC metabolites and observed symptoms of DUI.

Based on the information available at the time, the following methods for cannabinoid analysis were used for the analysis of blood and serum collected from the subjects in this study:

1. A commercial RIA reagent kit utilizing 3H labelled Hapten for the quantitative determination of Delta 9 THC and total cannabinoids in blood and serum samples. These kits were obtained from Immunanalysis Corp., Glendale, California, 91204.

2. A non-commercial kit, utilizing 125I labelled Hapten for the quantitative determination of Delta 9 THC and COOH-THC in blood and serum samples. These kits were made available for research purposes by NIDA through the Research Triangle Institute, Research Triangle Park, North Carolina, 27702.

3. Quantitative determination of Delta 9-THC in blood and serum by GC/MS as reported by A. Wong, et al, (NIDA Monograph 42).

Blood And Urine Sampling Protocol - Blood specimens were collected by venipuncture from the arm of each of six subjects for each drive day by the following schedule:

1. A specimen prior to consuming alcohol or smoking marijuana was drawn immediately prior to the start of the baseline drive.

2. Four additional specimens were taken at hourly intervals after treatment and immediately before each of the four subsequent performance drives.

3. Urine samples were collected from all subjects prior to receiving their prescribed treatment, and a second urine sample was collected after the last driving session to be used to screen for drugs not included in the treatment.

Each of the five hourly sample withdrawals consisted of whole blood and serum as follows:

1. One 7 ml Vacutainer containing EDTA and NAF yielding, on average, about 6 ml of whole blood per subject drawn.

2. Two 10 ml Vacutainers containing no anticoagulant or preservative. These tubes were centrifuged immediately after drawing and yielded about 8 ml of serum per subject drawn.

The 6 subjects per day x 5 sample draws per subject gave a total of 30 whole blood and 30 serum samples per drive day. The 17-drive days, conducted between

June 27 and September 23, 1981, yielded approximately 500 blood and 500 serum samples. Each of the 500 blood and 500 serum samples were aliquoted into 4 sub-samples: 1 for each of the 2 RIA labs, the GC/MS lab, and a reserve sample.

Each of these 30 blood and serum samples were aliquoted within 1-or-2 hours after withdrawal into either 2 ml or 4 ml Teflon stoppered glass vials according to the Protocol shown in Table 1.

These vials were assigned blind-code numbers, placed in special styrofoam holders, frozen on the day of withdrawal, and maintained frozen until needed for analysis by each of the following methods and laboratories.

1. Tritiated (3H) RADIOIMMUNOASSAY (RIA) for Delta 9-THC and total Cannabinoids by Dr. R. Baselt at University of California at Davis,with kits and directions supplied under contract with Immunanalysis Corp., Glendale, California, 91204.

2. IODINE 125 (125 I) RIA for Delta 9-THC and Carboxy-Delta 9 THC at the California Department of Justice, Bureau of Forensic Services, Toxicology Unit, with experimental kits and directions supplied by Research Triangle Institute (RTI) under contract to NIDA.

3. Gas Chromatograhy/Mass Spectrometry (GC/MS) of all samples reported as positive by either of the RIA methods, and selected negative RIA results were analyzed for Delta 9 THC by the California Analytical Laboratories, Sacramento, California, by the method described by Dr. A. Wong (Wong et al 1982).

Table 1 also presents a summary of how the samples were divided and distributed among the three laboratories. The samples given to the two RIA labs consisted of 1 ml aliquots of blood and serum contained in 2 ml glass vials. The samples for GC/MS analysis consisted of 2 ml aliquots of blood and serum contained in 4 ml vials. Any reserve remaining, i.e., 2 to 3 ml, was placed in 4 ml vials. The headspace allowed in each sample vial was to prevent the glass vial from breaking when frozen.

With each set of 30 subject vials of blood and serum samples for each drive day, blind coded control samples in identical vials at 7 levels of Delta 9-THC (0, 3, 5, 7, 10, 15, and 30 ng/ml) were included with each set of subject vials at the rate of 2 blind controls for every 8 randomly ordered subject samples. No blind carboxy-THC controls were used.

A 1-time batch of 25 ng/ml Delta 9 THC secondary standard was prepared by DOJ project personnel using a pure (98.6%) GC/MS validated standard (by RTI) sample of Delta 9-THC in ethanol and maintained at (4 C). This secondary standard of Delta 9 THC was utilized by DOJ to spike the "blind" control specimens for RIA and GC/MS analysis. Equal 25 ml aliquots of this secondary standard were provided to the 3 analytical labs to prepare known calibration standards which were run with each set of subject samples along with calibration standards supplied with the RIA kits.

The blind control and subject samples were maintained frozen until completion of the analyses, (one-to-four weeks for the RIA analyses, and up to six months for the GC/MS analyses). These analyses were performed on positive samples only.

Pre- and post-urine samples were screened with the ROCHE-DIAGNOSTICS Abuscreen RIA kits for the following drugs: Morphine/Codeine; Amphetamines; Barbiturates; Cocaine; PCP; Methaqualone; Benzodiozepines; and Cannabinoids. All of the analyses performed for all subjects' blood, breath, and urine samples are summarized in Appendix I, Table 1.

RESULTS

Sample Description

Data for the experiment was collected from a total of 100 subjects. After extensive editing of the data, a total of 83 subjects were retained for analysis. The first 11 subjects were used as pilot subjects leaving 89 subjects in the experimental pool. In 6 cases blood and urine samples collected prior to drug treatments were found to contain high levels of other drugs indicating probable drug use just prior to participation. Consequently, these subjects were excluded from the final statistical analysis. (See Appendix I, table 1).

Table 2 summarizes several background and demographic characteristics by treatment group. The overall average consumption was 4.35 "joints" per week. The average number of times subjects reported driving under the influence of alcohol ranged from 18.5 times for the alcohol only group to 28.8 times for the marijuana only group. The average number of times subjects reported driving under the effects of marijuana was quite similar to the number of times they reported driving under the influence of alcohol, averaging 21.20 times and 21.58 times, respectively.

The significance of the differences between the groups on each of the variables in table 2 was evaluated through Chi-Square and F Tests. None of the differences were statistically significant ($p > .05$), indicating that the four groups can be considered random samples from a common population.

Table 3 presents the total number of subjects for which data was collected, total subjects removed, total number of subjects analyzed and percent of subjects removed by each treatment condition. The percentage removed ranged from 8% for the marijuana and alcohol groups to 20% for each of the other three groups. Although this variation in deletion rate was not statistically significant ($p > .05$), the more critical question is whether or not it introduced a bias on the comparability of the groups with respect to the driving performance measures. This question is evaluated in a subsequent section of the report.

Domain Data Reduction Analysis

As described in the methods section, separate factor analyses were conducted on the performance measures within each of the four primary measurement domains. The major objective of this strategy was to reduce the large number of variables to a smaller and more manageable number of reliable dimensions.

The results of the analyses are summarized in table 4. Based on the 1.0 eigenvalue criterion, the inside rater domain reduced to 21 factors, the outside rater domain to 1 factor, the field sobriety test to 4 factors, and the vehicle sensor domain to 6 factors. The first 10 factors for the inside rater (LRE) domain accounted for 38.4% of the total variable score variance. The small amount of variance extracted by each factor, particularly after the first two factors, indicates that many of the LRE measures have limited generality. It was therefore decided to retain only the first 10 LRE factors for further analysis.

The outside rater domain produced only one factor, which accounted for 61.4% of the total score variance. Since these dimension consisted of simple counts of the number of cones touched or knocked down during the skill tasks, a complex factor structure was not anticipated, and the factor which emerged was essentially a count of the number of touched stanchions.

The first three factors of the FST domain and the first two of the sensor domain were retained for further analyses. The percent of total variance explained by these factors was 56.7% and 43.8%, respectively.

In addition to selecting measures for further analyses based on the preceding factor analyses, several variables were included on the basis of substantive apriori considerations. These were: number of stanchions knocked down, critical tracking task (CTT), brief interval time estimation (BITE), CHP officers and subjects overall impairment rating of subjects' impairment (OFF RATE and SELF RATE). Finally, the elapsed time/on various segments of the course was included as a measure of vehicle speed. This measure was chosen instead of the speed sensor because the latter failed to form a clear-cut interpretable factor in the vehicle sensor factor analysis.

One final variable, CHP car following rating (FALLCAR), was not included in the global analysis but was retained as a performance measure in subsequent analyses of treatment effects.

The final set of variables and the results of the global factor analysis are described below.

Global Factor Analysis

Based on the preceding analyses, a total of 73 factors or variables were selected for further factoring. The major purpose of this final "global" analysis was to obtain factor loadings across the various measurement domains, thereby providing information on the total structure of the measurements and on the correlation between the domain factors and variables. The 73 variables consisted of 16 composite scores or factors from the domain analyses and 57 raw variables. As described in the the methods section, the composites were not based on regression-derived factor scores, but rather on the simpler procedure of summing the standardized defining variables (Tryon & Bailey, 1970). It should be emphasized that the inclusion of both factors and some of their component variables in the same analysis (part-whole relations) inflated the extracted factor variance allocations and commonalities, requiring an additional adjustment, described below.

The factor loading matrix, after varimax rotation, is shown in table 5 and the percent of variance (after rotation) explained by each of the 21 extracted factors is summarized in table 6. Only loadings of .20 or greater are included in the loading matrix. The percentage variance allocations after removal of the spurious part/whole components are shown in table 7. These adjustments were computed by deleting the loading for the factor composite whenever the variable components were also included.

The 21 factors explained 81.75% of the total variance in the 73 variables. Keep in mind that since most of these 73 variables are the product of previous domain factor analyses, the 81.75% does not refer to the variance in the entire pool of

original measures. This percentage would be lower and would also contain more measurement error and idiosyncratic variance.) After the eighth factor, the incremental percent of variance begins to flatten, indicating the extraction of less useful factors.

The first factor can be best characterized as a coordination factor with high loadings on three tasks from the field sobriety test including the right- and left-foot balance and the finger count tasks. The officers' overall impairment rating loaded most highly on this factor. Factor 2 appears to be a stopping factor with the extended drive stop, bipolar extended, and urban drive stops among the high-loading variables. High loadings on the BISTOP composite variable provide further substantiation. Factor 3 was characterized by several forced lane change measures. Factor 4 was characterized by the three measures of speed assessed by LREs during the speedometer-covered portion of the extended drive. Factor 5 was characterized by measures of steering and associated lane-position measures during turns and on the speedometer-covered portion of the extended drive. Factor 6 appears to be a measure of overall errors in speed and lane-position control. Factor 7 was characterized by a high loading on the LRE-assessed backing-up task, and factor 8 had a high loading on the bipolar speed measure from the extended drive. Factor 9 was characterized by stopping position of measures, and factor 10 was defined by attempts on the risk task (attempting the risk task and the total number of attempts made).

The above factors and the remaining 11 are described in table 7. The sensor factor from the vehicle sensor domain, which was a measure of overall steering, accelerator changes and brake presses, did not load very highly on any of the factors. Its highest loading (.41) was on factor 3, and it also had loadings of over .30 on several other factors. The time and line measures of the vehicle sensor domain characterized factors 13 and 19, respectively. Factor 13 is, therefore, a measure of driving speed, and factor 19 represents lateral deviation from the right border of the road. The fact that the bipolar speed rating during the urban drive (BUSPEED) also loaded heavily on factor 13 provides further indication of a speed interpretation. The BITE and CTT loaded most highly on factor 18, indicating significant shared variance between complex reaction/motor control and time estimation. Factor 15 is clearly a measure of the quality of response to the risk task. Factor 12 is characterized by the degree of recklessness on steering through the turns of the urban drive. The final two factors represent the outside rater domain.

Test/Retest Correlations

The consistency (temporal stability) of the performance scores were evaluated by computing various sets of pair-wise correlations between trials as described in the methods section. Table 8 presents these results for each of 28 variables of interest including the 27 derived from the domain and global factor analyses. The first 4 columns in table 4 present run-by-run pair-wise correlations between driving trials 3, 4, 5, and 6 and the baseline trial (run 2). Averages of sets of pair-wise correlations between runs are shown in columns 5-7. Column 5 contains the grand average of all pairwise correlations across driving trials including run 2. The average pair-wise correlation between post-treatment runs, and is shown in column 6 and the average correlation between the post-treatment runs and run 2 is presented in column 7.

Looking at the overall average row at the bottom of the table 8, note that the average pair-wise correlation between runs for all variables was .61 (runs 2-6). The average using just runs 3-6 was slightly higher (.66), and the average between runs 2 and 3-6 slightly lower. Although somewhat lower than the conventional psychometric standard for "high" reliability in test construction ($r \geq .80$), these test/retest correlations must be interpreted within the context of the present experimental design. Three-fourths of the subjects were treated between the second and third trial, and each subject was "tested" five times. Any intrinsic reliability of the measures would be attenuated by differential response to treatment, learning effects, and differential-fatigue factors.

Several of the specific measures produced very low test/retest correlations. Most notable in this regard were time (sensor domain), COGNIT, and eye-hand coordination (FST domain), both of the outside rater variables, risk, BSPEED, BSTEER, BISKILL, UFAILED, FLRESPONSE, FLRERUN, OVERALL, and FALLCAR.

In general, the sensor domain produced the most stable measurements. This conclusion is further supported by the results for the 5 sensor variables prior to forming composite factors (see final five variables in table 8). The average of all pair-wise correlations (runs 2-6) ranged from .48 (speed) to .72 (accelerator). The low reliability of the time factor is surprising since the line sensor measured the elapsed time required to traverse various parts of the course. It, therefore, should be highly related to the speed sensor and equally reliable. Yet, the correlation between the speed and elapsed time across the various trials was low. Although the reason for this are not clear, the speed sensor was potentially distorted by the aforementioned signal loss and error in reconstructing the missing values. Another difference is that elapsed time includes stopping time, whereas the speed sensor monitored miles per hour while the vehicle was moving.

The overall LRE rating is the only LRE variable showing a marked alteration in reliability due to treatment. Note, for example, the dramatic reduction in test/retest correlation which occurred when the baseline trial is included in the correlation (runs 3-6 with 2 and runs 2-6 vs. runs 3-6).

Evaluation of Assignment Bias

To measure potential bias among the variables, a series of one-way univariate ANOVAs were performed on each of 27 performance variables at baseline (run 2). Table 9 presents the variables in conjunction with their associated F values and significance levels. Only two of the analyses were statistically significant. The driver's position during stops (POSTOP) was significant in run 2 ($F = 3.25$, $p < .05$). Inspection of the means for this variable indicate that the group that was to receive the marijuana treatment generally made more stop position errors than did the other groups; conversely, the group designated to be the placebo tended to make fewer stop position errors than the other groups. The other significant variable was the driver's speed (SMPH) on the extended portion of the driving course ($F = 3.33$, $p < .05$). Inspection of the means for this variable indicate that the group designated to receive alcohol and marijuana traversed the extended portion of the course about two miles per hour faster than the other groups.

Although these results appear to suggest some degree of bias in treatment assignment, it would be expected that 1.5 variables would be statistically significant by chance alone based on 27 variables tested at significance level $p \leq .05$. Since the observed results were very close to chance expectations, it is likely that the significance observed on two variables reflects random variation within a common population. It was therefore concluded that there was no compelling evidence of bias.

The above conclusion was further evaluated through a single multivariate test. As described in the methods section, a multiple discriminant function analysis (MDF) was performed using the four treatments as the grouping factor and 12 performance measures at baseline as discriminators. The 12 discriminators were those subsequently found to be the most significant discriminators of posttreatment performance, including the two variables described above (POSTOP and SMPH). As would be expected if groups were drawn at random from a common population, the discrimination at baseline did not approach significance as tested by the Wilk's Lambda criterion ($p > .25$). Thus, the conclusion of treatment group equivalence at baseline was confirmed.

Covariate Search

To supplement the assessment of bias among the measures and to increase the power of subsequent analysis, an attempt was made to determine if there were any measures that could be utilized as covariates. Items from the background questionnaire and variables from the baseline run were correlated with those of the total (composite) run.

Baseline Performance Measures. Although there was no complelling evidence of bias with respect to performance at baseline, inclusion of the baseline measures as covariates in analyses of posttreatment performance might be warranted if these measures proved to be significant predictors of scores on the subsequent trials. Normally, scores on pre- and post-tests are highly correlated, a fact which was confirmed by some of the preceding test/retest correlations.

The relationships between runs 2 and 3 were evaluated through a series of regression analyses on each of the 12 most significant discriminators of posttreatment performance. On each measure, the baseline (run 2) scores for each treatment group comprised the predictor variable and performance on run 3 was used as the dependent criterion variable. Significant relationships were found on seven of the measures; the R^2s are summarized in table 10. In several instances, the correlations were very substantial and would normally justify the use of the baseline measures as covariates. However, the analyses also indicated that the relationships between pre- and posttreatment performance varied significantly among the four treatment groups on all measures except one -- BISKILLS. In some cases, the heterogeneity of slopes was substantial. Note, for example, the difference in the R^2 between a common- and separate-slopes model for the CTT measure (.53 vs. .66).

Although there are methods for performing an analysis of covariance utilizing different within-treatment-regression slopes, they are very complex to execute and difficult to interpret. It was therefore decided not to include baseline measures as covariates even though in some instances the increase in precision might have justified the distortion produced by violating the homogeneity of slopes assumption. The fact that the groups were essentially equivalent on the baseline measures weighed heavily in this decision.

It is instructive to consider the substantive meaning of the slope differences in their own right. The fact that the relationship between pre- and post-treatment performance differed as a function of treatment indicates the presence of significant treatment x baseline score interaction. In the context of the present study, these interactions mean that the magnitude of a treatment's effect on performance was influenced by the subject's performance levels at baseline. The nature and direction of some of these interactions are explored in a subsequent section of the report.

Background Questionnaire Items. Data from the background questionnaire offered some additional variables which could potentially be used as covariates. The background questionnaire contained questions about the subject's drug use, experience driving under the influence of drugs, and overall years of driving experience.

The simple correlation coefficients between the 5 background variables and each of the 29 performance measures (runs 3-6 combined) selected following the global factor analysis are shown in table 11. Years of driving experience and experience driving under alcohol provided the largest number of significant correlations -- 7 and 10, respectively. However, none of the correlations were even moderately high, and the overall results suggest that post-treatment performance was largely independent of the five background characteristics. It was therefore decided not to include any of the background variables as covariates.

Discriminant Function Analysis of Treatment Effects Within Run

The post-treatment differences between the groups on the reduced set of variables were evaluated within each run (3-6) and on the composite of all runs through a step-wise discriminant function procedure. The 29 variables or composites from the factor analyses comprised the discriminators and the four treatments formed the grouping dimension.

Table 12 presents the significant variables. Four variables were consistently significant discriminators for every run. They were STOPS, POSTOP, ESTCHIC, and CTT. Several other variables were significant on two or more runs. Although the Wilk's Lambdas seem to indicate that treatment effects actually increased over time, this is more likely the result of a greater capitalization on chance in the latter trials, which contained a larger number of marginally significant discriminators.

Using a significance level of $p \leq .25$ and a requirement that a performance variable discriminate on at least 2 runs, the following 12 variables were selected for retention:

1.	STOPS	Errors made in stopping.
2.	COGNIT	The cognitive factor from the Sobriety Test.
3.	STOUCH	Total cones and stanchions touched.
4.	ATTEMPTS	Total risk task attempts.
5.	SDOWN	Total cones and stanchions knocked down.
6.	BSPEED	Speed control from inside rater.
7.	BISKILLS	Skills portion of driver's performance.
8.	UFAILED	Failure to follow directions on urban drive.
9.	POSTOP	Number of errors in stop position.

10. ESTCHIC Time estimated by driver to traverse Chicane.
11. SMPH Miles per hour in the extended drive.
12. CTT Critical tracking task

The discriminant analyses were then rerun on each trial to derive optimal functions on this common pool of the 12 best variables. Table 13 shows the discriminant functions resulting from these variables for each run and for the composite of the runs. The test for all three functions combined was significant at $p \leq .01$ on each of the runs. The second function was significant on runs 3 ($p \leq .01$) and 5 ($p \leq .05$). The third function was not significant on any of the runs and is, therefore, not shown. The Wilk's Lambda statistic associated with all three functions ranged from .29 (run 3) to .38 (run 4). Since these Lambdas represent the percentage of unexplained variance, a lower value indicates greater discrimination (explained variance) and hence a larger treatment effect. The percentage explained variance can be obtained by subtracting the Lambdas from one. (The reader should note, however, that this value is not the same as the variance-explained percentage shown in Table 13. This latter percentage is the relative percentage of the total explained variance attributed to each function. Since the Wilk's Lambda statistic is a measure of total variance, it provides a better index of the absolute magnitude of the treatment effects.) The results in table 13 indicate that, as expected, the effects of treatment were largest at run 3. Somewhat unexpected, however, was the increase in treatment effect from run 5 to run 6.

The final two columns of table 13 show the significant discriminant functions for the composite runs analysis (runs 3-6 combined). The functions accounted for 64.3% and 22.3% of the explained variance, respectively. The Wilk's Lambda value indicates that the functions explained 67% (1 minus .33) of the between group variance on the composite runs ($p \leq .001$).

Figure 1 shows a two-dimensional plot of the group centroids for functions 1 and 2 of the composite runs analysis. Looking first at function 1, note that the maximum separation is between the double placebo (P) and the both drugs (B) group. Since the placebo group's centroid was on the positive end of the scale, increasing scores on function 1 were associated with nondrugged performance and decreasing scores with drugged performance. The marijuana group (M) and alcohol group (A) centroids fall between the two extremes, with marijuana closer to the both-drugs group.

Separation on the second function is a bit less clear, with the double placebo group (P) having a negative centroid, the M and A groups having positive centroids, and the both-drugs group falling almost along the 0 plane.

Although the centroid plots provide information on how the functions separate the groups, they provide no information on what the functions are measuring; nor can we even infer from the value of the centroid plots whether a positive or negative score indicates a performance detriment. To some extent, the characteristics of the functions can be inferred from the standardized discriminant function weights associated with the discriminators (table 13). However, a better procedure is to base interpretation of the functions on the structure loading matrix, as described below.

Table 14 shows the structure or "loading" matrix for the first two functions for all variables yielding correlations of .26 higher ($p \leq .01$) for the composite run.

Since the structure loading are simple correlation between the discriminant function scores of the subjects and their scores on each variable, they provide valuable information on what each function is really measuring. It should be noted that these correlations have more powerful causal implications than ordinary correlation coefficients because they represent correlations with that portion of the total score variance which has been causally affected by the treatments (the between treatment variance). Although the loading matrix is usually limited to the variables contained in the discriminant functions, the procedure here was to calculate the correlations for the entire pool of 73 variables.

The variable loading highest on function 1 was ESTCHIC - the subject's estimate of the speed at which he could traverse the chicane. The positive correlation of .52 indicates that increasing scores on the first function were substantially associated with increasing subjective speed estimates. Since the centroid plots, above, indicated that high positive scores on function 1 were associated with the placebo condition and high negative scores with the double drug condition, the positive correlation with ESTCHIC suggests that drugged subjects perceived themselves as less skillful. The subject's speed, when the speedometer was covered (SMPH and SMPH 1-3), was also substantially correlated with the first function. The negative coefficients indicate that persons with higher scores on this function tended to drive more slowly when the speedometer was covered compared to persons with lower scores. The function was also inversely associated with most other measures of vehicle speed. Taken together, these results indicate that the drugged groups drove faster than the placebo even though they perceived themselves as less skillful. Two exceptions to this trend were on elapsed time through the chicane and urban drive (CLI6 and ULI6). The respective correlations of -.40 and -.30 indicate that higher scoring subjects took less time and hence drove more quickly on these portions of the course.

The LRE bipolar measures showed a tendency for higher scores on the function to be associated with greater-than-average cautiousness. Among other relationships of interest, the structure loadings indicate that high scores on the function were associated with better performance on the detour task, more frequent accelerator reversals on the extended drive (EACI), less impairment on the field sobriety tests (FST), higher self-ratings, and a higher overall LRE rating (OVERALL). The correlations for the variables ERROR and ATTEMPTS indicate that persons scoring higher on function 1 tended to make fewer total errors (LRE rating) on the course and fewer attempts at the risk task.

The second function is primarily a measure of stopping performance, as indicated by POSTOP, BIPOST, STOPS, SSTOP, and ESTOP. These measures are all from the LRE rating form and represent various errors or deviations in stopping. By far the most important was POSTOP, which correlated .69 with the second function. Persons scoring high on this function tended to stop too soon or too late relative to the signs. As shown by the centroid plots, this function tended to separate the marijuana and alcohol groups from the placebo, with the latter receiving lower (better) scores. The both drugs group fell in between these extremes, suggesting a suppressive interaction on stopping performance.

In comparing the signs of the standardized discriminant function coefficients in table 13 with the structure loadings in table 14, it will be noted that some of the signs have reversed (ESTCHIC, SMPH, ATTEMPTS, COGNIT, UFAILED, and SDOWN). This phenomenon, which is analogous to the concept of a suppressor effect in multiple regression analysis (Cohen & Cohen, 1975) means that a variable's influence when adjusted for multicollinearity with other variables is different from its simple direct relationship with the treatment groups. Considerable caution must therefore be used in interpreting the discriminant function coefficients. Interpretation of such effects is further complicated when more than one significant function is extracted, because each function is orthogonally adjusted for the other and separates the groups in different ways. It is, therefore, necessary to examine each function with respect to the group centroids, the structure loadings, and the discriminant function coefficients in order to fully comprehend the nature of the treatment effects. Inspection of the within group means for each discriminating variable is also informative.

The group means for the 12 final outcome variables are presented in table 15. Inspection of the means indicates that the group receiving both marijuana and alcohol performed worse on STOPS, STOUCH, ATTEMPTS, BSPEED, UFAILED, ESTCHIC, SMPH, and CTT. Specifically, the both-drugs group tended to make more stopping errors, touch more cones or stanchions, make more attempts at the risk task, drive more recklessly on the course, not follow directions as well on the urban route, estimate their chicane speed to be slower, drive the extended course faster, and have lower scores on the CTT measures.

The group receiving alcohol only tended to do worse on the driving performance measures than the group receiving marijuana only, and the marijuana-only group tended to perform worse than the placebo group with the exception of the SDOWN variable. Surprisingly, the marijuana-only group knocked down the fewest number of stanchions and did not exhibit even a trend toward a negative effect on the CTT.

The reader is cautioned that the above portrayal of means is merely a description of directional trends which have not been tested for statistical significance. An inferential analysis of the overall and pair-wise differences appears in a later section.

Canonical Analysis of Treatment Effects

To determine the amount of variance accounted for by each treatment combination, a series of canonical correlations were performed. These analyses provided multivariate tests of the main and interactive effects of marijuana and alcohol on performance.

The two variable sets consisted of the treatments (alcohol, marijuana, or alcohol and marijuana) designated as dummy variables and the 12 final discriminators obtained in the discriminant function analyses described above. The canonical correlations between the two variable sets were computed hierarchically using two step-wise sequences for representing the treatment set: sequence A- (1) marijuana alone, (2) marijuana alone + alcohol alone, (3) marijuana alone + alcohol alone + both drugs; sequence B- (1) alcohol alone, (2) alcohol alone + marijuana alone, (3) alcohol alone + marijuana alone + both drugs. By comparing the incremental increase in the percentage variance explained (1.0 minus Wilk's Lambda) at each step, the main and interactive multivariate effects of the treatments were assessed.

As explained in the methods section, the two entry sequences were necessitated by the small nonorthogonality introduced through subject deletion. The unequal samples rendered the main effect estimates sensitive to the entry sequence, and this bias was removed by averaging the two solutions.

The results are shown in table 16 for each run and for the four posttreatment runs combined. The main effects of marijuana and alcohol were highly significant for all trials. Although significant nonadditivity (interaction) occurred at runs 3 and 6, the interaction variance was much smaller than the main effects and was non-significant at runs 4 and 5 and for runs 3-6 combined. It can, therefore, be concluded that the effects of the two drugs were largely additive.

Looking at the main effects within trial, note that the alcohol effect is consistently the larger of the two, although the differences are modest. There are also differences in the time gradients, with alcohol exerting its largest effect at run 4 and marijuana at run 3. Probably the most surprising finding is the significant increase in the interaction at the last trial. Inspection of the treatment group means for individual variables, along with the multivariate centroids, indicates that this interaction was due to a comparative increase in the impairment of the both-drugs group during trial 6.

The results shown in the last two columns of table 16 warrant discussion. The first of these summary columns is simply the average of the four treatment effects across the four trials. The last column is based on the canonical analysis of the composite performance scores (runs 3-6 combined). Thus, the first summary preserves any differential effect of treatment within trials, whereas the latter ignores trial interactions by collapsing the trials into a single composite.

It is important to understand the above distinction because the relative size of the alcohol and marijuana effects depends on which summary is used. Using the mean percent variance explained across the four trials as a criterion, the marijuana effect is smaller than the alcohol effect (25.1% vs. 31.4%). However, the reverse is true for the composite runs summary. This seeming conflict indicates the presence of a significant trial x alcohol interaction. In other words, the structure of the performance decrements for alcohol varied across trials and these effects were obscured when the trials were collapsed to form a composite. Under these conditions, the average of the individual trials provides a more accurate reflection than does the composite analysis. As indicated above, this average indicates that alcohol had a larger effect on performance than did marijuana.

Detailed Analysis of the Effects over Time

Since the research design involved multiple trials for each subject (repeated measures), the effects of time and time x treatment interaction were of central importance to the study. In other words, to what extent did performance vary across trial and did the time gradients vary among the treatments? Recall from the methods section that these questions correspond to the test of the trial effect and trials x treatment interaction in a repeated measures ANOVA, but that the nonavailability of a repeated measures program required a combination of two different approaches.

The first consisted of separate multiple discriminant function (MDF) analyses on each of the 12 significant discriminators; mean performance on each trial for a given measure comprised the discriminators and the four treatments comprised the groups. The second approach was a test of the significance of the difference between the within-group-trials slope. This latter test is essentially a test of difference in rates of change across trial, whereas the former is a test for overall differences in the elevation and pattern of the vector of trial means.

Discriminant Function Analysis. The major objective of the discriminant function analysis described above was to determine the number of significant functions which resulted when the individual trials were used as discriminators. As was the case in the previous MDF analyses of treatment effects, a maximum of three functions were extractable since four groups were being discriminated. Two sets of analyses were performed -- one using all five trials (including run 2) as discriminators and the other using only the four post-treatment trials. Since each of the 12 multivariate discriminators were evaluated, a total of 24 discriminant functions were required.

The rationale behind viewing the number of significant functions as a measure of treatment x trial interaction warrants further comment and qualification. If the relationship between trial and performance were approximately linear and identical for each treatment group, one would expect a maximum of one significant function or eigenvalue for a given performance measure. The existence of two or three significant eigenvalues would imply that the dispersion pattern in group means across trials differed among the treatment groups in a complex way. It should be noted that this test is not an explicit test of slope differences since it is sensitive to both the elevation and profile of the trial means. The reader is referred to O'Brien and Kaiser (9185) for a discussion on the formulation of repeated measures problems as multivariate (MANOVA) models.

The results of the analyses are summarized in tables 17 and 18. Plots of treatment means across trials are shown in figures 2-13. In neither of the analyses (tables 17 and 18) did more than one significant function emerge. The analyses with the baseline run excluded produced statistically significant single functions on five measures -- STOUCH, SDOWN, ESTCHIC, SMPH, and CTT. The inclusion of the baseline run reduced the significant discriminators to the latter three.

Since it was not the major purpose of these analyses to evaluate univariate treatment effects, per se, the interpretation of the significant functions will not be examined in any detail. The major thrust of these results is that performance within each trial adds very little to the ability to discriminate the treatment groups. The fact that no significant discrimination occurred for most of the measures may seem paradoxical in view of the highly significant treatment effects identified in the previous sections. However, these effects were based on multivariate linear composites of the 12 performance measures, which take into account the covariance among the measures. As is frequently the case in MDF analysis, variables which are not significant by themselves becomes significant in conjunction with other correlated measures. There would also be some loss in power in using the trials as discriminators in situations where trial effects were minimal.

Although there are some exceptions, analysis of figures 2-13 indicate that the largest performance difference across the post-treatment trials (3-6) was between the double placebo and both-drugs group, with the latter almost always showing poorer performance. Perhaps the most interesting and significant finding occurred on the critical tracking task (Figure 13). This measure produced the largest eigenvalue on both of the MDF analyses and showed very clear-cut differences across the post-treatment trials. Looking at figure 13, note that the double placebo and marijuana groups have very similar means across each trial and almost flat trial gradients. Thus, there is no evidence that marijuana impaired this function or that performance varied across the trials. In contrast, the alcohol and both-drugs group show marked detriment. The slope of their gradients is quadratic, beginning at trial 3, asymptoting at trial 4 and beginning to dissipate at trial 5. However, even at trial 6 performance had not returned to the baseline level.

The results for ESTCHIC are also clear-cut, with only the both-drugs groups showing an effect throughout all trials. The effect is particularly notable at run 3, then gradually declining. Since ESTCHIC is the subjects' estimates of how fast they could drive through the chicane, this effect is a measure of subjective intoxication and indicates that subjects receiving both drugs perceived themselves as significantly impaired. In contrast, marijuana and alcohol alone were not perceived as affecting performance on this task.

Figure 12 displays the results on speed maintenance (at 35 mph) with the speedometer covered. The double-placebo group was able to maintain speed at 35 mph on all trials, whereas the three drug groups drove at above 35 mph on each run. The both-drugs group was again the poorest, approaching 40 mph at trial 4.

The means across trials for STOUCH and SDOWN are shown in figures 4 and 5. These effects are complex and difficult to interpret. Figure 4 suggests that marijuana improved performance on the first post-treatment trial (run 3), and that the combination of marijuana and alcohol produced an increasing decline at trials 4 and 5. Since the subjective and objective effects of marijuana were generally largest at run 3, the fact that the marijuana group touched fewer stanchions at run 3 is surprising. However, results on one speed through the chicane measure (CLI6) indicates that the marijuana group also drove slower than the other groups during run 3. This could explain the better performance on STOUCH.

The results for SDOWN (figure 5) show a markedly different profile which, in some respects, complements the effects shown on STOUCH. That is, there is a tendency for large effects for a given treatment-trial on one measure to be associated with a smaller effect on the other. Thus, if the two performance sets were added to create a "sum of cones touched or knocked down" measure, the composite would show less evidence of a treatment effect.

In interpreting the preceding figures, some may be tempted to conceptualize the results in terms of "change" or "difference" scores in which the post-treatment means are subtracted from their baseline levels (run 2). It should be recalled, however, that the baseline differences did not exceed chance expectation. As a result, any between-group differences at run 2 could represent measurement error rather than "true score" differences. Under these circumstances, difference scores are subject to distortions.

Analysis of Slope Differences. A multiple-regression procedure was used to evaluate the homogeneity of the within group trials slopes (Tabachnick & Fidell, 1983, page 192). This technique provides an explicit test of differences in rates of change across trials. It was applied to each of the 12 performance measures, both with and without run 2 included.

All 24 analyses indicated statistically significant slope differences ($p \leq .05$). The nature of these differences can be deduced from inspection of the figures and several have already been discussed in connection with the above analyses. In most instances, the major source of the slope differences related to differences between the placebo and the three drug groups, particularly the both-drugs group. The both-drugs group was atypical in showing an increasing performance decrement at trial 6 on several measures. Another consistent difference was between the marijuana and alcohol groups. Marijuana tended to have a peak effect at trial 3, whereas, alcohol more often peaked at trial 4.

Detailed Interaction Analysis

This section presents results of the analyses of the interaction effects between marijuana, alcohol, various baseline and background variables, and driving performance. As such, the major focus is on whether the univariate effects of alcohol and marijuana are additive with each other and with selected subject characteristics. The earlier multivariate analyses of treatment effects indicated that marijuana and alcohol were largely additive in their effects but that significant interactions did occur on runs 3 and 6.

The subject characteristics which formed the pool of potential moderator variables were selected from the baseline (run 2) performance factors and the background questionnaire. Twenty variables were included from the performance domain and five from the questionnaire.

A 2 x 2 x 2 factorial analysis of variance program was used to test the main and interactive effects of the treatment and moderator variables on each of the 12 post-treatment measures identified in the previous discriminant function analysis. The first factor was alcohol, the second marijuana, and the third consisted of a moderator variable dichotomized into low or high scores. The split was made as close as possible to the median in order to approximately equalize the marginal distributions and cell sizes.

Because of the large number of separate ANOVAs (12 x 25 = 300), it was necessary to limit the analysis to effects on the post-treatment composite scores (runs 3-6 combined).

Baseline Measures. The 240 (12 x 20) ANOVAs which included a baseline measure are summarized in Tables 19-38. Each table represents inclusion of 1 of the 20 baseline performance measures. The entries in the table represent the obtained p-value for each source of variation. Seven sources of variance were tested per analyses: (1) main effect of alcohol; (2) main effect of marijuana; (3) main effect of the moderator; (4) marijuana x alcohol interaction; (5) marijuana x moderator interaction; (6) alcohol x moderator interaction; and (7) marijuana x alcohol x moderator interaction.

Although the primary objective of these analyses was to assess two and three-way interactions involving the baseline measure, we will first briefly touch on (1) main effects and (2) two and three-way moderator interactions before proceeding to the main results of interest.

1. Marijuana and Alcohol. Alcohol generally yielded a significant effect ($p \leq .05$) on four measures -- BSPEED, STOUCH, CTT, and SMPH. Effects approaching or exceeding significance on some of the analyses were also noted for ATTEMPTS (risk task), COGNIT, and BISKILL.

   Since the same 12 dependent variables were used in evaluating the interactions with subject characteristics (moderators), the question arises as to why the main effects of alcohol (and marijuana) were not the same in each table. The variation is due to the differing amounts of nonorthogonality in combination with the differing main effects of the moderator variables. Although an attempt was made to split each moderator variable as closely to the median as possible, some of the moderators still deviated substantially from a 50/50 split. In addition, the treatment groups did not have equal samples due to subject deletion. Since each main effect is statistically adjusted for the other main effects in the model, the main effects of alcohol and marijuana vary (slightly) as a function of which moderator variable was included. In general, a main effect which is significant only in the presence of only or a few of the moderators is open to question.

   With the above caveat in mind, inspection of tables 19-38 indicate a consistent main effect of marijuana on 3 measures--COGNIT, SMPH, and ESTCHIC. A suggestive main effects also occurred on ATTEMPTS (risk task) in several of the analyses.

   The only consistent evidence of a significant marijuana x alcohol interaction occurred on POSTOP. This interaction was significant on all analyses except the one which employed baseline POSTOP performance as a moderator (table 22). However, even in this instance, the p-value was suggestive ($p = .124$). Since there was some evidence that the treatment groups were not equivalent on POSTOP at baseline, the reduction in significance of the interaction when POSTOP-2 was included could indicate a removal or reduction in bias through operation of the POSTOP-2 main effect. Nevertheless, the fact that the interaction was statistically significant on 19 of the 20 analyses and at least suggestive on the other, lends some credence to its reality. Inspection of treatment group means (table 15) indicates that the interaction was suppressive in nature. That is, the two drugs together ($\bar{x} = 4.95$) resulted in better stopping performance than either marijuana ($\bar{x} = 5.59$) or alcohol ($\bar{x} = 5.52$) alone.

   Only two other marijuana x alcohol interactions were statistically significant. One was on ESTCHIC when RISK-2 was a moderator and the second was for STOPS with CTT-2 as the moderator. Given the large number of interactions which were tested and the lack of significant M x A interaction on these measures in the other ANOVAs, it is likely that these represent "chance significance."

2. Two and Three-way Moderator Interactions. The analyses of variance produced 10 significant two-way marijuana x moderator interactions, 11 significant alcohol x moderator interactions, and 12 significant three-way interactions. Since a total of 240 tests of each of the above two-way and three-way hypotheses were made, one would expect to obtain by chance alone approximately 12 significant results at the .05 level for each of the interaction tests (240 x .05 = 12). The obtained results are, therefore consistent with the hypothesis of no true two-way and three-way interactions involving the baseline performance measures. This inference leads to the conclusion that the effects of marijuana and alcohol did not vary as a function of a subject's baseline performance. Although this conclusion seems to conflict with the previous analyses of slope differences, the regression slopes were based only on trial 3, whereas the present interactions are based on runs 3-6 combined. Given the lack of sufficient evidence of nonadditivity, no attempt will be made to describe or interpret any of the specific interactions that reached significance, with one exception. Note from table 34 that the marijuana x SMPH-2 interaction on SMPH was significant at $p < .02$. Since this was the only one of the 33 significant interactions in which the dependent variable and moderator involved the same measure, there is a somewhat stronger substantive basis for describing its structure. Inspection of the cell means indicates that marijuana-positive subjects who drove below the median speed during baseline (speedometer covered) drove very close to the target speed of 35 mph after being drugged ($\bar{x}$ = 35.3 mph), while higher-speed-marijuana subjects showed an increase in speed after consumption ($\bar{x}$ = 39.5). In contrast, marijuana-negative subjects who drove more quickly at baseline showed no increase in speed during the post-treatment trials.

Biographical Measures. The 60 ANOVAs (5 x 12) using the five biographical measures are summarized in tables 39-44. Since the main effects of marijuana and alcohol and the M x A interaction were already presented in connection with the preceding analyses, they will not be discussed further. The novel aspects of the present ANOVAs relate to main effects and interactions involving the biographical measures.

It is readily apparent from inspection of tables 39-43 that very few of the effect terms were statistically significant. The analyses resulted in four significant main effects for the biographical measures, three significant alcohol x biographical interactions, no significant marijuana x biographical interactions, and one significant three-way interaction. As was the case with the baseline-interaction analysis, these numbers do not exceed chance expectation (60 x .05 = 3). Therefore, there is no compelling basis for concluding that response to the treatment was influenced by subject's prior driving experience, frequency of marijuana/alcohol usage, or experience driving while impaired.

The fact that so few of the main effects of the biographical variables were significant is surprising since data was presented earlier showing that these measures had modest, but significant, correlations with several of the performance measures (table 11). This difference is probably due to the attenuation and information loss caused by collapsing the measures into simple dichotomies.

Miscellaneous Univariate Analyses. One limitation to the above analyses is that only the 12 performance variables selected from the multivariate analyses were used in evaluating the treatment effects and interactions. As was the case with the evaluation of treatment effects over time, relatively few of the measures were significantly affected by treatment when considered individually. However, these analyses have not addressed the question of treatment effects on the many performance measures not selected through the multivariate analyses.

To explore this issue, the means on the 73 variables and factors identified from the within-domain analyses were tabulated by treatment group and trials. The results for all variables which yielded one-way ANOVAs significant at $p \leq .10$ are shown in Appendix III, tables 1-5. Although detailed consideration of these results is reserved for the discussion section of the report, a few comments are in order here. First, the number of significant ($p \leq .05$) univariate F ratios is substantial. The maximum number of significant effects occurred at runs 3 and 4, where there were, respectively, 19 and 14 significant differences. At run 6, the number declined to 7, but the composite (runs 3-6) yielded significant effects on 15 variables. Given the differences in the significant variables and the declining magnitude of effects across trials, it is clear that performance on runs 3-6 combined has provided a conservative estimate of the treatment effects.

Blood Levels Analysis

Analytical Results for the Detection of Alcohol and Cannabinoids in Body Fluids. The peak blood alcohol concentration (BAC) values for all positive alcohol subjects used in the performance analyses were closely distributed around the target BAC value of .08%, as indicated in Appendix I, table 1. With the exception of 2 out of 42 subjects, the remaining subjects fell within .02% of the target value of .08%, which is an expected variance when calculating alcohol dose based on body weight and drinking period.

The use of two different RIA methods and a GC/MS confirmation method might appear redundant but proved to be a fortunate decision because of the many unanticipated problems which occurred with the RIA methods as well as the confirmatory GC/MS analysis (Hanson et al, 1983). Based on the comparison of methods, the 125 I-RIA analysis of the subject serum samples for Delta 9-THC and COOH-THC were the only methods sufficiently accurate and reliable for a statistical correlation with the driving performance measures.

The results of the RIA analysis performed on each subject's serum, pre- and post-urine, and concurrent percent BAC are summarized in Appendix I, table 1.

Basal Levels of Cannabinoids in Serum (Plasma) of Chronic Marijuana Users. There were 12 test subjects for whom the analysis of serum (plasma) indicated "basal" levels of cannabinoids in the 2-to-10 ng/ml level range. Of the subjects, five of the 12 were in the placebo marijuana group, and 7 were in the marijuana treatment group. The initial serum cannabinoid results for these 12 subjects are summarized in table B of the Appendix I.

The relatively high and constant Delta 9-THC levels found in all samples from the five placebo marijuana subjects, shown in table B, strongly suggests that basal levels of cannabinoids may persist at significant levels as a result of chronic marijuana use.

The relatively high Delta 9-THC levels found in the pretreatment samples of the seven subjects shown in Table B who received the marijuana treatment would indicate that some of these subjects may have used marijuana on the same day the samples were taken.

Where there was sufficient reserve sample, further confirmatory analyses were conducted by the NIDA research group at RTI as indicated in table C of Appendix I. This limited analysis conducted by NIDA/RTI confirmed the initial RIA results from the I 125 RIA analyses (table B).

To further examine the above hypothesis, 4 of the same 12 subjects were recalled in June 1982 for a follow-up study. These four subjects gave four serum samples spaced about two hours apart while under constant observation of project staff without receiving any marijuana treatment. The subjects were asked to abstain from any marijuana use for at least 48 hours as had been done during the drive study. These samples were analyzed by the Center of Human Toxicology, University of Utah, Salt Lake City. The results reported for these serum samples are summarized in table D, of Appendix I. These results further supported the contention that significant basal levels of cannabinoids can exist for many hours in "chronic" marijuana users. The general behavior and the extremely high levels found in subject 8/8/81-F strongly suggest that he did not abstain from marijuana use for 48 hours as requested.

Further work is necessary with respect to documenting THC levels in body fluids (serum) of chronic marijuana users.

A comparison and evaluation of the accuracy, sensitivity, and reliability of the two RIA and GC/MS methods used for cannabinoid analysis for this study are discussed by Hanson et al, 1983. The significant conclusions are summarized as follows: Either of the RIA methods appears capable of measuring THC and COOH-THC concentrations for up to three hours after usage in persons smoking marijuana. Serum is a better specimen than blood in terms of accuracy, detectability, reproducibility, and specificity. A parallel determination of COOH-THC concentrations is desirable for forensic purposes. The level of this metabolite provides a useful confirmation of the presence of THC and indicates elapsed time since inhalation of marijuana. Accurate method calibration is essential, and reference standards in a biological matrix should be available to allow analysts to gauge their accuracy while performing THC and COOH-THC determinations.

Correlation with Performance Measures. One of the objectives of the study was to determine if variations in blood level were associated with variations in driving performance. To evaluate the magnitude of the statistical relationship between the drugs and 12 performance measures, the actual blood levels of the substances (THC, carboxy, and alcohol) were recorded for each of the runs for correlation with the 12 performance measures.

The mean blood levels by trial are presented in table 44. The results conform fairly well with what was expected based on the amounts consumed and time from dosage. The alcohol group peaked at trial 4 (.08%) and declined to a BAC of .04% at trial 6. The marijuana and both drugs group achieved peak THC levels ($\bar{x}$ = 69.6 & 54.3) at trial 3, and the levels had dissipated precipitously by the next trial ($\bar{x}$ = 13.1 & 13.4). The difference between the marijuana and both-

drug groups on serum carboxy levels was not expected and at first glance suggests that alcohol interferes with the creation of serum carboxy. Although the difference persisted throughout the trials, the authors are inclined to dismiss this finding due to variations in serum carboxy among the subjects prior to treatment.

The only other notable finding in table 44 is the variation in BAC levels between the alcohol-only and both-drugs groups. The results suggest that the presence of THC/serum carboxy delays the absorption of alcohol. At Trial 3, the alcohol-only group achieved a BAC of .07% compared to .05% for the both-drug groups. However, the two groups were at parity at trials 5 and 6.

Since there was some variation in the blood levels actually attained by the subjects within a given treatment and trial, it was possible to explore whether these variations were correlated with differences in driving performance. In other words, did subjects who attained higher-than-average levels tend to exhibit more performance detriment than those who attained lower levels? This question was pursued by the previously mentioned canonical correlation analysis in which scores on the 12 variable performance vector were correlated with the three quantitative blood levels and dummy-coded treatment vector. The interaction terms were created by forming the appropriate two-way cross-product terms among the independent variables. The results are summarized in table 45.

Table 45 shows the contribution of the blood levels to explaining variation on the performance vector. Note that in all cases the inclusion of blood alcohol level increased the size of the alcohol effect and the increase was statistically significant on runs 3 and 4 ($p < .05$); the increase approached significance on runs 5 and 6. In contrast, the contribution of serum THC and serum carboxy alone was always less than the contribution of the treatment group designation. However, when both serum carboxy and serum THC were included, there was an increase in the marijuana effect on runs 4-6. The increase on run 4 was statistically significant ($p < .05$).

In all cases, the size of the interaction effects declined by inclusion of the blood levels, but the total explained variance (marijuana + alcohol) still increased at runs 4 and 5. At run 4, for example, the net effect (explained variance) increased from 62% to 72% ($p < .05$).

In general, these results indicate that knowledge of a person's exact blood levels on all three measures slightly increased the ability to predict performance on the driving tasks during runs 4 and 5. In addition, the results indicate that the marijuana effect was mediated by the joint operation of serum THC and serum carboxy. Either substance alone does poorly as a predictor of performance.

The above analysis has limited sensitivity due to the restricted range of variability of the blood levels. This stems from the fact that the experimental design was intended to minimize variability by administering a constant dosage to each subject and then testing each subject at fixed intervals from dosage. Any analysis applied to variations within trials necessarily ignores variation across trials. The change in blood levels over trials is obviously much greater than subject variations within trials due to the passage of time.

The above recognition led to the following question: To what extent are changes in blood levels over trials associated with between-trial variations in performance?

This question was evaluated through a two-stage, canonical analysis procedure. Recall from the methods section that canonical analysis produces linear composites of two sets of variables (vectors) which are weighted so as to maximize the correlation between the sets. Each trial was subjected to a canonical correlation analysis in which the 3 blood levels comprised the X vector and the 12 performance measures comprised the Y vector. The weights for each set of variables were then used to compute two scores for each subject -- one from the optimally-weighted-blood-level vector and the other from the optimally-weighted-performance-variable vector. These pairs of computed scores can be viewed as representing the relationship between blood levels and performance within trial. To determine the structure of the relationships across trials, these computed scores were subjected to a second canonical analysis in which the two variable sets were comprised of the canonical scores for each trial. That is, each subject received a canonical blood level score and a canonical performance score for trials 2, 3, 4, 5, and 6, and these pairs of scores were subjected to a second commical analysis. The analyses were done with and without trial 2 included.

The results for the analysis including run 2 are shown in tables 46 and 47, and those excluding run 2 are summarized in tables 48 and 49. Attention is directed to tables 46 and 48 since they are the results of primary interest.

The analysis which included the baseline run (table 46) resulted in three significant functions, whereas the analysis of post-treatment runs produced two significant functions (table 48). The percent of variance explained by the 2 sets were, respectively, 88. and 83. Thus, optimum linear composites of the blood levels and performance functions can be constructed which explain a large portion of the systematic variation in performance over trials. However, the meaning of the canonical variates is unclear and does not provide unequivocal evidence of a temporally isomorphic relationship. For example, the largest coefficients on the first function tend to occur at different trials, and there are some inexplicable sign reversals (table 47 and 49). These types of interpretative ambiguities frequently occur with canonical correlation analysis due to the complex partialling of intercorrelated variates and capitalization on chance relationships (Cohen and Cohen, 1983). Nevertheless, there are some interesting structural similarities, such as the weights for runs 4 and 5 on the second function in table 49.

Figures 14A and 14D portray covariation in mean performance and mean blood levels over the four post-treatment trials. These data are presented for their descriptive and heuristic interest and must be interpreted with caution because of their aggregate form and lack of tests of significance. They do show instances of intuitively plausible concordance between performance and blood levels, but they also show instances of improved group performance in the presence of elevated blood levels.

Special Issues

A number of auxillary measures were incorporated to monitor parametors of special interest. CHP Officers in a "FALLCAR" and a field sobriety test were used to assess the officers' ability detect drug impairment. Questionnaires were developed to provided subjective ratings of impairment. The BITE and CTT were incorporated to assess drug effect on more standardized psychomotor measures. Finally, the forced lane change maneuver and risk task were included as measures of relevant cognitive- and emergency-response modalities.

CHP Following Car and Field Sobriety Tests. The correlation between these tests and the scores for the first two discriminant functions are presented in table 50. Recall that the multivariate analysis of treatment effects resulted in two statistically significant functions containing 12 performance measures. Simple correlations between these function scores and individual variables provide information on the characteristics and structure of the treatment effects.

Six of the variables were significantly correlated with the first function, but only one was correlated with the second function. (The first function is by far the most important and reflects differences between all of the treatments, particularly between the both-drugs group and placebo.)

The subject's rating of his performance on the FST and drive range produced the highest correlation (- .40), and the direction of the relationship indicates that increased performance decrement following treatment was associated with a perception of poorer performance. That is, subjects whose overall performance was indicative of treatment impairment tended to rate their performance as more impaired. All of the other significant correlations show a similar direction. The finger count, counting backward and officer's rating produced the highest correlations among the FST measures; the following car measure was also highly significant in the expected direction. Figures 15-17 display plots of means on selected rating variables. The most outstanding feature of these data is the poorer performance of the both-drugs group on every variable. Note also the remarkable similarity of the LRE, self and officer ratings (figure 16). In general, the impairment associated with either marijuana or alcohol alone was perceived as minimal and slightly greater for alcohol. However, since these results are in terms of the composite runs, they tend to underestimate the immediate effects of the drugs.

The results on the following car measure produced clear-cut evidence of a treatment effect (figure 17). Subjects receiving both drugs would have been "stopped" or "pulled over" about 60% of the time by the following CHP officers, compared to about 15% of the placebo subjects. Alcohol alone and marijuana alone resulted in stopped scores of 50% and 32%, respectively. The analysis of variance of these differences was highly significant ($p < .01$, Appendix III, table 5).

Table 51 indicates that the FST and following car measures were significantly correlated with several of the performance measures. The means for the FST and following car are shown by individual trials in Tables 52-55. The different time gradients for the marijuana and alcohol effect is evident on the following car measure. At Run 3, the marijuana and both groups would have been "stopped" 59% of the time compared to 42% of the alcohol subjects. By the next run (4),

the marijuana group's stopped percentage dropped dramatically (29%) while the alcohol group increased to 56%. The both-drug group above 50% at all runs except run 6. The differences were statistically significant at runs 4 and 5 ($p < .05$) and almost significant at run 3 ($p < .10$).

Curiously, the other subjective ratings do not show a corresponding differential in the marijuana and alcohol time gradients. The subjects in all three drug groups rated themselves most impaired at run 3. The same was true of the officer's overall FST rating. However, both drugs in combination resulted in significantly greater impairment and much longer lasting impairment on the FST ratings. Even at trial 6, the both-drug group showed clear impairment on the officer's and self-ratings, while the other groups have returned to baseline or placebo levels.

Impairment Questionnaire. Subjects completed an impairment questionnaire after the third and fifth drives for the purpose of assessing their subjective perception of change as a result of treatment. The complete questionnaire is reproduced in the 1981 Protocol (Grossen et al). Table 56 presents the correlation between the items from the impairment questionnaire at run 3 and the two scores from the significant discriminant functions. All of the questionnaire items were significantly correlated ($p < .05$) with the first function, and three items were significantly correlated ($p < .05$) with the scores from the second function. The three items were self-control, speed, and time estimation. All correlations with the first function were negative. All correlations with the second function were positive. The single highest correlation on the first function was on speed estimation ($r = -.43$). The direction of this and the other relationships indicate that increased subjective impairment was always associated with increased impairment on the performance functions.

Table 57 presents the impairment questionnaire items obtained after run 5 correlated with the scores from the two significant discriminant functions. All but one item (self-control) was significantly correlated ($p < .05$) with the first function. Time and speed estimation were the only items that correlated significantly with the second function. The structure loadings between runs 3 and 5 are very similar, indicating a temporal robustness to the structure of the impairment mechanisms.

The simple correlations between each of the 10 impairment items and the 27 performance factors at runs 3 and 5 are presented in tables 57 and 58. These correlations are conceptually distinct from the previous structure coefficients in that they do not represent relationships with treatment effects. Rather, these correlations indicate the extent to which a given pair of variables covary for the entire sample of subjects. As such, they are of less interest than the structure coefficients. Although a substantial number of the correlations are statistically significant, there is considerable variation between the two runs. Taken together, the LREs rating of the subject's total performance (OVERALL) yielded the highest correlation with the 10 impairment ratings on both runs, with several correlations in the .30 to .40 range. ESTCHIC also produced consistently moderate correlations on most of the variables, particularly item 6 (coordination) and item 7 (reaction time) on run 3. Both correlations were .43, indicating that greater perceived impairment on these attributes was associated with slower estimates of chicane speed. UFAIL was moderately correlated with

most of the run 5 impairment items but not with many of the items on run 3. In general, the overall results indicate a moderate relationship between poorer performance and lower self-ratings.

To further explore the relationship between treatment and perceived impairment, the scores on the 10 impairment items were summed and tabulated by treatment and trial (runs 3 and 5). The results are presented below:

TREATMENT	RUN 3	RUN 5
Placebo	10.0	5.57
Marijuana	22.7	5.26
Alcohol	21.2	7.20
Both	29.3	18.05

These findings are very consistent with the subjective self-assessment ratings presented earlier, particularly with respect to the tendency for the both-drugs condition to lengthen the duration of impairment. An analysis by individual item at run 3 indicated significant treatment differences on all items except reaction time (#7), spatial abilities (#3), and auditory ability (#2). The intercorrelation among items was substatial and the largest treatment differences occurred on items 1 (visual), 4 (memory), 5 (self control), and 9 (time estimation).

A better understanding of the mean ratings can be gained by dividing each mean by the number of items (10) on the impairment questionnaire and comparing the per item means to the scaled values on the questionnaire. Recalling that 0 = normal and 9 = severe impairment, it can be seen that all treatments were associated with relatively moderate impairment ratings. Even the both drugs group received an average score of only 2.9 per item. It is interesting to note that the placebo averaged 1.0, indicating that a substantial number of subjects in the control group believed they had received an active substance.

Exit Questionnaire. In an attempt to gain further comprehension of the subjective experience under the drug treatments, each subject completed an exit questionnaire upon conclusion of all other driving and peripheral tasks. This questionnaire consisted of 25 items covering previous drug experiences and the subjects' reactions to the experimental drive tasks. The questionnaire items are defined in table 59. The complete questionnaire appears in the 1981 Protocol (Grossen et al).

Each item from the questionnaire was correlated with the two discriminant function scores to assess the relationship between the objective functions and subjective experience. The correlations are presented in table 60. The subject's rating of "produced high" correlated positively with both functions ($p < .055$). Thus, the degree of the "high" was associated with greater performance impairment ($r = .45$). Driving frequency under a similar "high" was negatively correlated with both functions ($p < .05$) suggesting that subjects felt that they would not normally drive given a similar drug reaction. Perceived drug impairment on various segments of the course also produced substantial correlations in the expected direction.

Since the exit questionnaire asked each subject to guess what treatment he received, it is possible to assess the adequacy of the placebo condition. The 77 subjects produced the following distribution: No drug=6, marijuana alone = 10; alcohol alone = 16; both drugs = 49. Clearly, the majority of the double placebo subjects were fooled by the placebo, and there was a strong tendency for subjects to erroneously believe they had received both alcohol and marajuana.

Background Variables. The correlations between five background characteristics and scores on the first two discriminant functions are summarized in table 61. None of the correlations are statistically significant, indicating that treatment response was not associated with any of the five background factors. This result is consistent with the previous analysis of interaction effects.

BITE and CTT. Table 62 presents the correlations between the BITE and CTT measures and the two significant discriminant functions. BITE scores for Runs 4 and 5 were significantly correlated ($p < .01$) with the first function as was the BITE composite. BITE scores on Run 3 were negatively correlated ($p < .01$) with the second function, as was the BITE composite ($p < .05$).

The means presented by treatment groups for each run for the BITE are presented in table 63. Although there was a definite trend on the BITE task for the placebo group to perform best, and the both-drugs group to perform most poorly, none of the analysis of variance tests produced a significant difference at ($p < .05$). The results are nevertheless suggestive of impaired time estimation, particularly when both drugs are combined.

The results in table 62 indicate that the CTT correlated positively ($p < .05$) with the first function at run 4 and negatively with the second function for runs 4 and 5 ($p < .05$). The CTT means by treatment for each run (table 63). The CTT differences clearly indicate impairment due to alcohol since the alcohol-alone and both-drugs groups were notably inferior to the marijuana and placebo groups. The differences reached statistical significance on runs 4 and 5 and on the total composite (run 3-6 combined). Somewhat surprisingly, there is not even a directional trend toward marijuana impairment on the CTT. The scores for the placebo do not change materially from run to run, whereas all three drug groups tended to improve. Thus, there is no evidence of residual practice effects but there is clear indication of reductions in impairment on the CTT over time.

Table 64 presents correlations between performances on the BITE and CTT with 27 dependent variables. Both tests provided significant correlations with several of the performance measures, and the correlations tended to be higher at run 3.

Forced Lane Change and Risk Task. Two special driving maneuvers were incorporated to examine specific emergency-response and cognitive-risk assessment under the drug treatments. Performance on the forced lane change maneuver was assessed for five different parameters. Correlations between these measures and the two significant discriminant functions are presented in table 65. The overall composite factor for the forced lane change provided the only significant correlation ($p < .05$), which occurred on the second function.

The means for each measure on the forced lane change are presented in table 66. The results in section 3 of the table show that all groups performed the task using a great deal of caution. Slight deviations from over-cautiousness occurred only in the drug groups but did not come close to approaching recklessness. During the initial drug-treatment runs, the alcohol-only group and the group receiving both drugs more frequently repeated the task due to failure to maintain the required speed. This was particularly true of the both-drugs group at run 3. However, large differences among treatments were not evident.

Table 67 presents correlations between the risk task measures andh the two significant discriminant function scores. The choice to perform the task was significantly correlated with the first function ($p < .05$) and quality of performance was significantly correlated ($p < .01$) with the second function. The number of attempts required to traverse. The channel in the risk task was positively correlated with the first function ($p < .01$). The composite-risk factor was positively correlated ($p < .05$) with the second function. The correlation pattern suggests that the placebo condition may have led to more attempts to perform the risk task and with greater success. The means for the risk task measures (table 68) show that the placebo group tended to perform the task more cautiously than did the other groups, and along with the alcohol only group, tended to attempt the task more frequently.

DISCUSSION AND CONCLUSIONS

The present study was designed to measure the effects on driving performance of a fixed "normative" dose (1 cigarette) of marijuana, both alone and in combination with alcohol. In the Introduction Section of the report, this general objective was subdivided into the following specific objectives:

1. To determine the singular and combined effects of marijuana and alcohol on a number of measures of driving performance.

2. To determine if there is a relationship between the ranges of Delta 9-THC in blood and/or alcohol in breath and measures of driving performance.

3. To determine if the various driving performance measures are differently affected by marijuana and alcohol ingestion.

4. To determine the relationship between the time following marijuana and/or alcohol ingestion and driving performance impairment.

5. To determine the interrelationship among the performance factors affected by the marijuana and alcohol ingestion.

6. To determine whether marijuana, alone or in combination with alcohol, results in impairment that can be reliably detected through external observation of the driving and standard field sobriety tests.

These objectives, as restated below provide the framework for summarizing and interpreting the preceding results. The conclusions formulated from this overview are then discussed in relation to previous research findings and theory relating to marijuana's effect on driving performance and traffic safety. The discussion concludes with a consideration of policy implications, study limitations and future research needs. Since the primary focus of the study was on the effects of marijuana, and marijuana in combination with alcohol, the alcohol findings are discussed only briefly, serving more as a referent for interpreting the marijuana effects.

The Main and Interactive Effects of Alcohol and Marijuana - General Characteristics

The results of the univariate and multivariate analyses indicate that both substances affected driving performance. The Multiple Discriminant Function (MDF) analysis resulted in two significant linear composites (function) of the 12 performance measures which were most consistently and uniquely affected by the treatments. These two functions explained between 62% and 70% of the between-group variance on the 12 measures across the 4 post treatment trials. Approximately 60% of the explained variance was attributed to the first function compared to 20% for the second function. The first function produced maximum discrimination between the placebo and both-drugs group, with marijuana alone and alcohol alone occupying intermediate positions between these two performance extremes. Inspection of the group means on the 12 variables which defined the function indicated the both-drugs polarity was generally indicative of impaired performance.

The second function, tended to separate the marijuana and alcohol groups from the placebo group, with the both drugs groups occupying an intermediate position.

An attempt was made to interpret the meaning of the two functions by inspection of the standardized discriminant function coefficients and structure loadings. Persons scoring higher (better) on the first function produced higher estimates of the speed at which they could drive the chicane; and drove more quickly through it; drove at lower and more appropriate speeds when the speedometer was covered, as well as on most other parts of the course; drove more cautiously; made more accelerator reversals on the extended drive; performed better on the detour task, performed better on the field sobriety tests; were judged to be less impaired by the officer; rated their c iving and field sobriety performance higher (less impaired); received higher overall ratings from the driver's license examiner (LRE) and made fewer driving errors on the drive course. This function clearly reflects between-group variation and performance decrement on a wide variety of measures: 1) subjective self-rating; 2) LRE ratings of speed control, overall performance, cautiousness and number of driving errors; 3) officer field sobriety and vehicle control rating; 4) objective measures of performance (number of stanchions knocked down, accelerator reversals, urban drive speed; and 5) number of attempts on the risks task). Since this function produced maximum separation between the both-drugs and placebo groups, with the latter falling at the positive end of the first function, higher scores on most of the preceding variables were associated with not being drugged, and lower scores with being exposed to alcohol and marijuana combined. The nature and range of variables affected indicate that exposure to the combined marijuana/alcohol condition resulted in impaired vehicular control and an accurate subjective awareness of the impaired performance.

Inspection of the second function indicated that higher scores on the function were associated with the marijuana and alcohol conditions, and the structure loadings indicated that it is primarily a measure of impaired stopping. By far the highest loading on the function was on the variable POSTOP, which correlated + .69 with the function. This variable represents the LREs rating of proper stopping position on all stopping maneuvers throughout the drive range. Since the variable was scaled so that low scores indicate proper stopping position (1 = smooth stop, 2 = abrupt or misjudged stop, and 3 = rolling or not stop), the high positive correlation between the function and variable is indicative of a higher proportion of improper stop ratings among marijuana and alcohol subjects. Unfortunately, the method of scaling did not distinguish between stopping too soon or too late relative to the sign.

Four other stopping measures also had significant positive loadings on the second function: 1) SSTOP (stopping errors on the speedometer covered segment of the course; 2) ESTOP (stopping errors on the extended drive); 3) STOPS (total number of stopping errors on all segments of the course), and 4) BIPOST (bipolar rating of stop position cautiousness). A fifth stopping measure, BISTOP, yielded a negative loading (- .31), indicating that stopping position caution on the speedometer-covered portion of the course was negatively associated with the function.

Taken together, the above results indicate that marijuana and alcohol affected stopping performance but, that the negative effects were reduced when both drugs were combined. The mechanism underlying what appears to be a of suppressive interaction is not clear and interpretation is further complicated by the above mentioned scaling problem. One possibility is that the separate drugs produced different types of stopping errors (delayed vs. early stops), which cancelled out when both were combined.

The main and interactive multivariate effects of marijuana and alcohol on the 12-variable discriminant functions were evaluated through a series of canonical analyses, and an analysis of variance procedure was used to evaluate univariate effects. The canonical analyses indicated that both substances had highly significant multivariate main effects on all four posttreatment runs. The marijuana condition explained between 23.1% to 29.2% of the variance on the 12 most discriminating performance measures, averaging 25.1% for all 4 trials. The alcohol condition produced a somewhat larger effect, accounting for 29.9% to 32.3% of the performance variance, and yielding an average effect of 31.4% for all 4 trials. The multivariate effects were largely additive, although small but significant interactions did occur on trials 3 and 6, where the respective explained-variance totals for the marijuana by alcohol interactions were 12.0% and 12.9%, respectively.

The factorial analyses of variance of univariate effects on each of the 12 discriminating variables provided additional confirmation on the locus of treatment effects and on their interactions with variables that were considered to be potential moderators of treatment effects (baseline performance on the 12 discriminating variables and 5 background characteristics representing prior alcohol/drug use and driving experience). These analyses indicated that marijuana had a significant main effect on three measures: 1) COGNIT, 2) SMPH, and 3) ESTCHIC. A suggestive main effect also occurred on attempts (risk task). Inspection of the means indicated that the marijuana resulted in poorer performance on the cognitive factor of the field sobriety test; higher speed while the speedometer was covered; a lower self-assessment of the fastest speed the subject could drive through the chicane, and more attempts required to complete the risk task. The alcohol condition produced a significant main effect on four measures: 1) BSPEED, 2) STOUCH, 3) CTT, and 4) SMPH. Suggestive main effects were also noted on attempts (risk task), COGNIT, and BISKILL. Inspection of the means for these variables indicated that all of the effects were suggestive of impairment. No consistent evidence for a marijuana/alcohol interaction emerged from these analyses, nor of an interaction with any of the baseline and background factors. Thus, these analyses support the conclusion that the effects of marijuana and alcohol are largely additive with each other and with the background and baseline performance characteristics of the subject sample.

The above conclusion of general additivity requires some qualification and further discussion. Recall that the detailed interaction analysis was limited to the 12 variables identified from the discriminant function analysis and was further limited to the total runs-composite (trials 3-6 combined). Since the effects of marijuana were most acute at run 3, and declined thereafter, the use of the composite measure could have masked some of the effects. In addition, interaction effects on variables other than "the best 12" were not evaluated.

There was, in fact, evidence of a small, but statistically significant, interaction between marijuana and alcohol at run 3, cited above in connection with the canonical analyses of treatment effects. Further evidence of non-additivity also emerged in connection with the homogeneity of slopes test for the regression of baseline (run 2) performance on posttreatment performance (runs 3-6 combined). The slope differences were particularly dramatic for the CTT and ESTCHIC variables, suggesting that response to treatment varied as a function of a subject's baseline level on these measures. The structure and substantive meaning of these interactions is not clear and seem in conflict with the failure to find significant treatment x baseline interactions in the analyses of variance. Two reasons can be advanced to explain the above paradox. First, the baseline measures were collapsed into dichotomous categorical factors for the analysis of variance tests, whereas the slopes test treated each variable as a continuous measure. If relationships and interactions among the measures were approximately linear, the categorical approach would significantly reduce the sensitivity for detecting interactions. Second, the slopes test was based on a slightly smaller sample size (N = 63) due to the deletion of cases with missing values on some of the measures.

The Main and Interactive Effects of Marijuana -- Detailed Characteristics of Effects Within Trials

The above discussion has primarily focused on the effects on all post-treatment trials combined into single composite measures. It has also been limited to the 12-variable core selected through the multiple discriminant function analysis. A complete understanding of the results of the experiment requires interpretation of effects within and across trial and a consideration of the many variables not included in the final MDF analyses.

The evaluation of treatment x trial interaction was achieved through a test of slope differences on the trial factor. Since the trials occurred at fixed points in time from the single drug ingestion, the trial slopes represent the effects of time, and the slope differences reflect temporal differences in each treatment group's performance gradient. These tests indicated significant slope differences on each of the 12 measures selected from the MDF analysis. A major source of the difference was marijuana's tendency to result in maximim impairment in the first post-treatment trial while alcohol's effect maximized at trial 4 and did not decline as rapidly as the marijuana effect. The both-drugs group tended to show the impairment gradient that would be expected from the component drugs, evidencing maximum impairment at both trials 3 and 4 and consistently showing greater impairment than either marijuana or alcohol alone. In addition, the combination of the two substances significantly lengthened the duration of effects, resulting in a rather remarkable increase in impairment from trials 5 to 6. The emergence of a synergistic marijuana by alcohol interaction at trial 6 suggests an effect mediated by some residual mechanism occurring when the two substances are combined. An obvious intuitive explanation would be that marijuana and alcohol interact to produce greater fatigue and "hang-over" effects.

Further insight into the effects of maijuana and alcohol can be gained by reference to the analyses of variance within trial on each of the 73 variables that were created prior to the data reduction. All variables significant at $P < .10$ are summarized in Appendix III, tables 1-5. The maximum number of effects occurred on runs 3 and 4, which yielded 29 and 24 significant F ratios, respectively, at the .10 level of significance. These numbers reduce to 19 and 14 at $P \leq .05$.

Individual t-tests and Bonferroni contrasts between each treatment and the control condition indicate that the majority of the both-drugs versus placebo differences are statistically significant ($P < .05$). A much smaller proportion of the placebo contrasts involving marijuana and alcohol separately reached significance, which would be expected based on the results of the MDF analysis.

The alcohol effect is larger than the marijuana effect on most of the variables from each domain -- subjective self-ratings, officer ratings, LRE ratings and objective measures. Among the exceptions to this rule was performance on stopping maneuvers at run 3, which was discussed previously. This finding suggests that marijuana may degrade the ability to judge distance and/or speed, resulting either in stopping to soon or too late.

Other results of interest are the findings on various peripheral measures, such as FALLCAR, field sobriety tests, self-rating and the CTT. In general, the subject self-rating of impairment was among the more sensitive indicators of treatment and tended to parallel the objective measures. This was also true of other self ratings not shown in these tables, but presented in the results section. The fact that the self-rating impairment indices closely mirrored both the objective indicators and the overall LRE and officer rating provides confirmation for the reliability and validity of the results.

That the officers in the following car (FALLCAR) were able to detect driving impairment with a significant degree of accuracy is notable. The FALLCAR results also clearly revealed the different time gradients for marijuana and alcohol. The marijuana subjects were detectable only at run 3, whereas alcohol peaked at run 4, and the both-drugs group was detected as impaired on all runs, except run 6. The impairment cues are evidently different than produced by the field sobriety test, since the latter did not produce consistently significant discrimination.

The results on the CTT variable are surprising in that marijuana alone did not produce impairment on any trial, which is in conflict with prior research findings. This complex psychomotor task was originally devised to detect alcohol impairment. The fact that alcohol and marijuana plus alcohol combined did produce impairment would seem sufficient to dispel the hypothesis that the marijuana finding can be attributed to some type of error or procedural artifact. Some possible reasons for the finding are discussed below in connection with the review of relevant literature.

Surprisingly, the marijuana-only condition resulted in fewer stanchions being knocked down at trial 3--where the level of intoxication was actually greatest. However, it is important to note that persons receiving marijuana or marijuana and alcohol tended to drive more slowly through the chicane than did the placebo and alcohol-alone subjects. Speed through the chicane was measured by the vehicle-line-sensor (CL16), which and produced the following elapsed times for the placebo, alcohol, marijuana, and both-drugs treatments, respectively: 16.7, 16.3, 17.3, and 18.4. Although the differences were not statistically significant ($p=.22$), it is instructive to note that other researchers have found that marijuana tends to cause persons to compensate for subjective impairment by reducing task difficulty through reduced vehicle speed.

Blood Levels

One of the objectives of the study was to assess the feasibility of developing an objective chemical index of marijuana impairment. Although the results did show that the quantitative levels of THC and carboxy combined resulted in some increase in the ability to explain performance variations, the practical and theoretical implication of the finding are not entirely clear. Experimental replication of this finding; using a wider range of marijuana-dose levels is needed before the feasibility of establishing a quantitative threshold can be fully evaluated. We are not optimistic, however, over the prospects for developing a scientifically defensible "illegal per se" threshold.

Relation to Prior Studies

As indicated by the extensive literature review summarized in the first section of the present study, there is a vast amount of empirical evidence documenting the effects of marijuana on a wide array of human performance measures -- cognitive, psychomotor and affective. Although the literature has clearly established that marijuana affects all three domains and results in detriments in the ability to perform many psychomotor and cognitive tasks, the evidence is somewhat more equivocal on the question of actual driving skill and even more equivocal on the question of those aspects of driving skill that are related to safety and accident avoidance. Any attempt at formulating a comprehensive and coherent theory on the effects of cannabis on driving performance, or reconciling the various empirical outcomes of different studies are complicated by the differing circumstances unique to each investigation. Among the variables to be considered are: (1) the specific performance task; (2) the frequency of prior marijuana usage and the strength of the typical THC concentration of the marijuana; (3) previous experience driving after consuming marijuana; (4) the amount and strength of the experimental dosage; (5) mode of ingestion (oral or smoke); and the type of research design employed in each study. The demonstration of behavioral change following ingestion, and the magnitude of the performance change, can obviously be affected by each of the above variables. Finally, there is the problem of equating change with detriment and then generalizing the inferred detriment to real world accident avoidance behavior. With the above cautions in mind, we will attempt to reach some conclusions as to how the present findings articulate with the findings of other investigators.

Studies of marijuana's impact on driving performance can be grouped into four general categories:

1. Laboratory Studies - These studies employ as performance criteria specific psychomotor tasks that are considered to be relevant to driving. Much of the work of Moskowitz and his associates using divided attention and visual tracking tasks fall into this category (Moskowitz, 1976). Similarly, studies by Belgrave et al (1979), Chesher (1976), and Sharma (1975) employed "task relevant" psychomotor variables in evaluating the effects of marijuana or marijuana in combination with alcohol.

2. Simulation Studies - These studies use response to simulated driving display tasks as performance measures. The simulators that have been employed vary greatly in the fidelity and complexity of the simulated tasks, but all involve response to dynamic traffic displays by manipulating vehicle control panels (steering wheel, accelerator, and brake). Examples of such studies are those of Rafaelsen et al (1973), Stein et al (1983), Dott (1972), and Crancer (1969).

3. Closed-Course Drive Range Studies - These studies measure the ability to drive a vehicle over a course designed to tap typical vehicle control response to actual driving situations. Examples of such studies are those of Klonoff (1974), Sutton (1983), Attwood et al (1981), and Casswell (1979).

4. On-Street Drive Studies - These studies attempt to measure impairment of the ability to drive in actual traffic situations such as might be encountered on a typical driver's license test. The study by Klonoff (1974), cited above, used on-street driving performance as its primary evaluation measure.

5. Epidemiological Studies of Accident-Involved Drivers and Victims - These studies attempt to determine the presence of cannabis or its metabolites in the tissue fluids of accident cases and establish cause/effect relationships by reference to an assumed or measured "population-at-risk" baseline. Although a number of studies have attempted to measure the presence of cannabis in the body fluids of fatal and injured accident victims (Owens et al 1983; Williams et al 1985; Warren et al 1981; Terhune et al 1982; Cimbura et al 1982.), none of these studies have developed the normative baseline or "population-at-risk" indices necessary to establish cause/effect relationships. (As a proxy to conventional "population-at-risk" measurement, some did provide relative-risk estimates based on indirect statistical models, such as induced exposure and accident culpability indices).

With the few exceptions discussed below, we believe that the measurement domains and research design of the present study are too dissimilar to warrant detailed comparison with the results of studies from Areas 1 and 2. (The implications of Study Area 5 are discussed later in connection with traffic safety ramifications of marijuana and driving). One area of conflict between the present and prior studies is the failure of marijuana alone to exhibit even a suggestive detrimental impact on the CTT measure. The critical tracking task was originally designed to detect alcohol impairment, and has been shown to be sensitive to relatively moderate levels of alcohol. Sharma and moskowitz (1975), reported that a dose of 200 mcg THC resulted in significant impairment lasting throughout the 4 hours of a repeated measures test session. Remarkably, the impairment was almost as great in the fourth hour as in the first hour after ingestion. More recently, Barnett et al (1985), using data from the above Sharma study, investigated in more detail the effects of the three-dose levels of marijuana (100, 200, and 250 mcg/kg) on divided attention, visual search and CTT performance. All three measures exhibited dose-related marijuana effects which, in the case of the CTT, lasted roughly 7.1 hours.

Given this evidence, we would be inclined to dismiss the present result as an artifact were it not for the fact that both the alcohol and both-drugs groups did show the expected decrement. Any error or procedural artifact would be expected to have affected all groups, since assignment to the experimental conditions was random and was rigorously monitored.

Two explanations are proposed here for consideration: The first is the differences in experimental design between this and the above two studies. The present study utilized an independent-groups design and treated the trials condition as a collapsed factor for most of the analyses. In contrast, the Sharma and Barnett et al, studies used repeated-measures designs in which each subject received all experimental conditions. Repeated-measures designs have greater sensitivity than independent-group designs when the very strong mathematical assumptions on which the designs are based are satisfied; these assumptions are often not satisfied in experiments on human subjects. Although it is clear from the inspection of pre- and post-treatment means that creation of pre- vs. post-difference scores or use of analysis of covariance would not have altered the results, one can always posit that the randomization did not completely control for idiosyncratic differences in drug response. However, there is absolutely no evidence from the present study to substantiate this thesis, and it seems unlikely that this would have occurred with the sizable samples used to randomize subject differences.

A more reasonable hypothesis is that the subjects were not equivalent in terms of their prior marijuana use (frequency and potency). There is evidence to believe that persons accommodate, at least partially, to the effects of marijuana through acquired tolerance and/or experience in performing tasks while intoxicated. Even though learning new tasks is generally impeded by marijuana use, there is evidence that recall of material originally learned while intoxicated is greatest during subsequent periods of intoxication, i.e., "state dependent learning" (Darley et al 1974). If one accepts the preceding evidence and line of reasoning, it seems clear that investigations of marijuana-induced performance decrement can produce conflicting results if based on subjects with different levels of tolerance and experience.

It appears that the subjects in the present study were, in fact, heavier users than subjects in the Sharma et al, and Barnett et al, studies. The latter studies required that subjects current use frequency not exceed three cigarettes per week and imposed a minimum lifetime use of only 10 episodes. The present study required subjects to be current users of one to seven "joints" per week, to have been users for at least two years, and to have experience with marijuana that was "at least" as potent as that used in the study.

It is instructive to note that the average impairment/intoxication rating given at trial 3 by subjects in the marijuana-alone group was only 3.41 on a scale of 1-9. This, combined with the extremely high serum carboxy levels of some of the subjects at baseline, suggests a chronic use of high-potency marijuana that in some cases exceeded the self-reported use frequency. In any event it seems clear that the subjects of this study were heavier users then than used in the Sharma et al, and Barnett et al, studies and many of the other studies cited in the literature. This speculation is also consistent with the prevalent availability and use of high potency "sinsemilla" variety of marijuana grown in northern California.

Variations in acquired tolerance and accomodation could also explain conflicting results of other investigators. For example, most studies, including the present, have not produced psychomotor decrements of the magnitude, consistency, and duration of those reported by Belgrave et al (1979). These

investigators used orally-ingested THC on subjects whose use history appeared quite moderate. In addition, the oral ingestion of THC--a mode not normally encountered and which produces slower but more enduring effect--further confounds interpretation and any generalization to what would be expected from smoking normative-dose levels.

Several of the other results obtained here articulate well with those reported by other investigators. The ability of the police observers in the following car to detect impairment, particularly in subjects who received both marijuana and alcohol, is consistent which the results of Sutton (1983). However, neither alcohol or marijuana alone could be detected in Sutton's study, which is in conflict with the present results.

The fact that marijuana and alcohol exerted a relatively additive effect on many driving behaviors is also consistent with prior research findings. The consistency of this effect and the range of the affected behaviors is notable. The emergence of a significant synergistic interaction between marijuana and alcohol in the fourth hour after ingestion is a novel finding. The mechanism underlying such an effect requires further study.

One limitation of the study was the lack of emergency response and accident avoidance tasks. A forced lane change (FLC) maneuver was included to tap some of the same psychomotor components required to avoid accidents; it was not consistently affected by any of the drug conditions (other than a tendency for the drugged subjects to more often drive below the minimum speed threshold). Although across-study comparisons are tenuous, the failure to find a marijuana main effect on this task is consistent with Stein et al (1983), finding that even a substantial dosage of marijuana (200 mg/kg) had no effect on the ability to avoid accidents on a simulated drive task. However, Stein did find evidence of a marijuana by alcohol interaction on accident avoidance, with lower doses (200 mg/kg) of marijuana decreasing the negative effect of alcohol and larger doses (200 mg/kg) accentuating alcohol's negative effects.

An important limitation of the forced lane change maneuver as a proxy for accident avoidance is that it was introduced as a discrete "off-line" task rather than integrated into the drive course. It therefore does not really measure vigilance and divided attention, which are critical components of accident avoidance behavior and attributes which are more likely to be affected by marijuana (Sharma and Moskowitz, 1973; Moskowitz, 1976).

Since this study utilized a closed-course-drive range, detailed comparison with prior research using a similar task mode is in order (one such study by Sutton was already alluded to above). Several other such studies have been reported in the literature (Klonoff, 1974; Attwood, 1980; Smiley et al 1974; Casswell, 1977; and Hansteen et al 1976).

Hansteen found that marijuana (88 mg/kg THC) resulted in a significant increase in the number of cones overturned on the slalom portion of the course but did not lead to increased erratic vehicle handling as judged by raters, whereas alcohol impaired both measures. In contrast, the present study found that marijuana significantly reduced the number of cones knocked over in the chicane task, but that the marijuana subjects also drove more slowly through the chicane. The both-drug group also reduced their speed compared to the placebo, but hit the same number of cones as the placebo. The alcohol-alone group tended to drive the fastest and also tended to hit the most cones.

Casswell found that marijuana alone decreased vehicle speed and course steering corrections. In contrast, alcohol and marijuana plus alcohol decreased fine steering reversals and increased variation in lateral placement of the vehicle. Casswell concluded that marijuana subjects tended to compensate for their perceived impairment by reducing vehicle speed, thereby reducing task difficulty and information processing demands. This conclusion in consistent with our finding, cited above, concerning marijuana's affect on the chicane task. However, marijuana did not result in reduced speed on most parts of the course and was associated with increased speed when the speedometer was covered. This can easily be attributed to the fact that most parts of the course were of minimal difficulty and did not require compensation. Although there was little evidence from the present study to show impact on steering reversals, marijuana and alcohol did affect steering control as rated by the in-car observer. The failure of the vehicle steering sensor to detect change in the present study could be due to the sensor defects and signal loss alluded to in the Methods Section.

The study by Smiley (1974) is one of the few examples of a marijuana by alcohol interaction in which marijuana appeared to reduce the negative effects of alcohol. Such an interaction occurred on stopping accuracy. In contrast, the present research showed that marijuana was associated with more stopping errors than alcohol, but there was evidence of significant negative interaction (interference) in which the combination of alcohol and marijuana resulted in better stopping position than marijuana alone.

Attwood (1981) found no evidence of any univariate effects of marijuana and alcohol on drive-range performance as measured by an instrumented vehicle. However, he did find significant multivariate effects. The strong tendency of multivariate methods to capitalize on chance relationships in small samples requires that Attwoods results be interpreted with caution until replicated.

The drive range portion of the study by Klonoff (1974) most resembles the present study in method and scope. With respect to the drive course tasks, Klonoff found that the higher marijuana dose (one cigarette of 1.2% THC) resulted in an increase in the number of cones hit on a slalom task, risk task, a funnel task, two tunnel maneuvers, and total composite score. No effects occurred on the back-up and corner tasks. The low-dose condition resulted in detriment on one of the tunnel tasks, the cornering task, and the total composite score. Effects on braking distance were suggestive but equivocal. Klonoff also found evidence of marijuana-induced improvement on various in-car observer ratings of on-street performance. The largest effects occurred on rating of judgment, care, and concentration (since these are highly subjective ratings, the possibility of some observer-halo bias should be noted, particularly if the raters were able to accurately guess the treatment condition).

Klonoff also concluded that while the majority of the subjects showed impairment, that a substantial minority actually improved. Several variables were analyzed as potential moderators of treatment response, including prior experience driving while under the influence of marijuana, and none produced evidence of interaction. (The present study also found no evidence of interaction on similar background variables.)

The present study was not analyzed in a way which would permit direct comparison with Klonoff's conclusion that some subjects improved following marijuana ingestion. However, we do know from the discriminant function classification matrices (which were not presented) that a small number (n=2) of marijuana subjects could not be differentiated from placebo subjects at trial 3 based on their discriminant-function scores. This finding implies that some users drive as well after consuming marijuana as does the typical user when not under the influence of marijuana. This, of course, does not necessarily mean that the misclassified subjects were not impaired, because these analyses did not take into account performance at baseline. It is therefore possible that the misclassified marijuana subjects represent persons who possess an extraordinarily high degree of skills, which was reduced to an average level by the marijuana treatment. This hypothesis and its articulation with Klonoff's findings will require a more in-depth analyses of the present data.

Traffic Safety Implications

Authorities are not in agreement on the traffic safety threat posed by marijuana use (Warren and Simpson, 1980). In a recent series of papers, McBay and Owens (1980) and Mason and McBay (1984/1985) concluded that marijuana is a relatively minor factor in traffic accidents and they questioned the feasibility of relating impairment to specific levels of THC. Although many of their criticisms of past studies are both astute and pertinent, we believe these same limitations prevent forming unqualified opinions in any direction about the role of marijuana in traffic accidents. Many of the conclusions formed by McBay and his associates are based on the failure to find a substantial incidence of THC in the blood or plasma levels of drivers killed in single vehicle accidents in North Carolina. Considerable caution is necessary in generalizing incidence data from a state like North Carolina to California. Not only are there likely to be large differences in marijuana usage, there may also be large differences in drive task complexity and the likely use of cannabis in conjunction with vehicle travel.

In addition, Moskowitz (1985) has recently pointed out that behavioral impairment and subjective intoxication are still manifest after THC has dissipated from the blood. This factor results in an unknown proportion of false negative findings from an analyses of accident victims blood specimens. Nevertheless, the point remains that the traffic safety implications of marijuana use must ultimately be based on direct evidence of its causal role in increasing accident risk. This necessitates establishing accurate "population-at-risk" baselines for (1) the incidence at which persons drive under various levels of THC alone; (2) the same incidence in combination with alcohol; and (3) the same incidence in combination with other drugs. The fact that marijuana is so often detected in conjunction with alcohol makes it difficult to establish a case against marijuana since any increase in relative risk could be due to alcohol alone. Establishing incident rates for the above risk groups would facilitate interpretation of the respective incident rates among accident-involved drivers.

Probably the most consistent and important finding of this study was the demonstration of an additive marijuana/alcohol effect on a wide array of performance measures. If one accepts the thesis that marijuana in conjunction with alcohol makes people "drunker", then it follows that marijuana in this context increases accident risk. A public policy implication of such a thesis might be to reduce the illegal, per se, BAC level for persons detected with both substances in their system.

The question of the traffic safety risk posed by marijuana alone is not as clear-cut as the risk presented by marijuana and alcohol in combination. Although evidence of impairment was identified in both the present and numerous past studies, the translation of this evidence into inferences about accident causation presents numerous difficulties. Before explaining why, we offer a dissenting opinion from a recent comprehensive review of the literature by Moskowitz (1985):

> "It should be clear from the above review that there is more than sufficient experimental evidence to concludde that marijuana seriously impairs psychomotor performance required for driving. Among the areas which exhibited overwhelming evidence for impairment were: A. coordination....; B. tracking; C. perception; D. vigilance; E. driving and flying performance measured by simulators; F. driving performance on the road.... Clearly, marijuana is a substance which produces serious behavioral toxicological effects. Any situation in which safety both for self and others depends upon alertness and capability of control of man-machine interaction precludes the use of marijuana."

Based on the present study and past evidence, we agree that marijuana undoubtedly impairs psychomotor abilities that are functionally related to driving and that driving skill itself may be impaired, particularly at high dose levels or among naive subjects. Given these facts alone, Moskowitz's implicit recommendation that people not drive after consuming marijuana should obviously be heeded. However, the extent to which marijuana-impaired driving causes accidents cannot be deduced from the present study, nor any of the studies cited by Moskowitz. Our more guarded posture to this question is based on the following rationale:

1. In their multidisciplinary investigation of traffic accidents, Joscelyn and Treat (1976) identified "improper lookout" and excessive speed as the two most frequent human factor causes of accidents.

   Although improper lookout involves some of the attentional and information-processing elements affects by marijuana, it is more closely related to the search and scan strategies utilized by drivers in anticipating and detecting potential conflicts. In the only study of marijuana's impact on traffic visual search behavior, Moskowitz et al (1976) found no evidence of a negative effect on this skill. Excessive speed can be best viewed as a reflection of attitude toward risk, risk assessment and aggressiveness. Several investigators have reported that marijuana reduces risk taking propensity and driving speed. Because of these compensating tendencies, it is presently not possible to assess the net impact of marijuana as a causal agent in traffic accidents. Although some increased accident risk appears likely, the magnitude of the risk remains obscure.

2. Many of the laboratory marijuana studies which have shown the greatest psychomotor impairment have utilized tasks that are only abstractly related to driving. Although divided attention and tracking are required for driving, it does not necessarily follow that performance decrement on a laboratory task designed to maximize task demands in order to identify individual differences and impairment are correlated with actual real-world performance in a vehicle. Correlational studies have consistently found very low or non-significant relationships between the recorded accident tests, including divided attention (Harano, et al 1975). The fact that attempts to measure response to simulated accidents have not consistently detected a marijuana-induced decrement, even at high dose levels, underscores the need for more research (Stein et al 1983).

Future Research Needs

In addition to the need for improved epidemiological studies mentioned earlier, the relationship between marijuana consumption and driving behavior can be clarified by a research design possessing the following characteristics:

1. A multi-method/multi-criterion approach in which subjects perform relevant psychomotor, driving simulator, and drive range tasks. The utilization of different measurement domains will permit an assessment of the multivariate effects across domain, leading to more generalizable characterizations of the extent and locus of marijuana-induced impairment.

2. At least three dose levels of marijuana should be used (none, moderate, and high) in order to obtain a greater range of THC variation and to better evaluate dose-response relationships.

3. Frequency of prior marijuana usage should be treated as an experimental factor by selecting subjects who vary substantially on use rate. At least three levels should be employed--light users, moderate users, and heavy users. Such a design would permit an evaluation of treatment x use frequency interaction resulting in a better understanding of whether acquired tolerance and accommodation are important factors in influencing impairment.

4. An independent group design with repeated measurement trials should be employed in preference to latin square and repeated measures designs in which each subject receives all treatments. Individual differences could be controlled through matching and analysis of covariance procedures.

5. The design should include some tasks under reduced-illumination to simulate night driving conditions. Serious accidents more often occur at night, and there is reason to suspect that marijuana-induced impairment would be accentuated by reduced visibility and night driving conditions.

Further research is also needed to validate the relationship between tasks (or simulators) designed to detect drug impairment and real-world driving.

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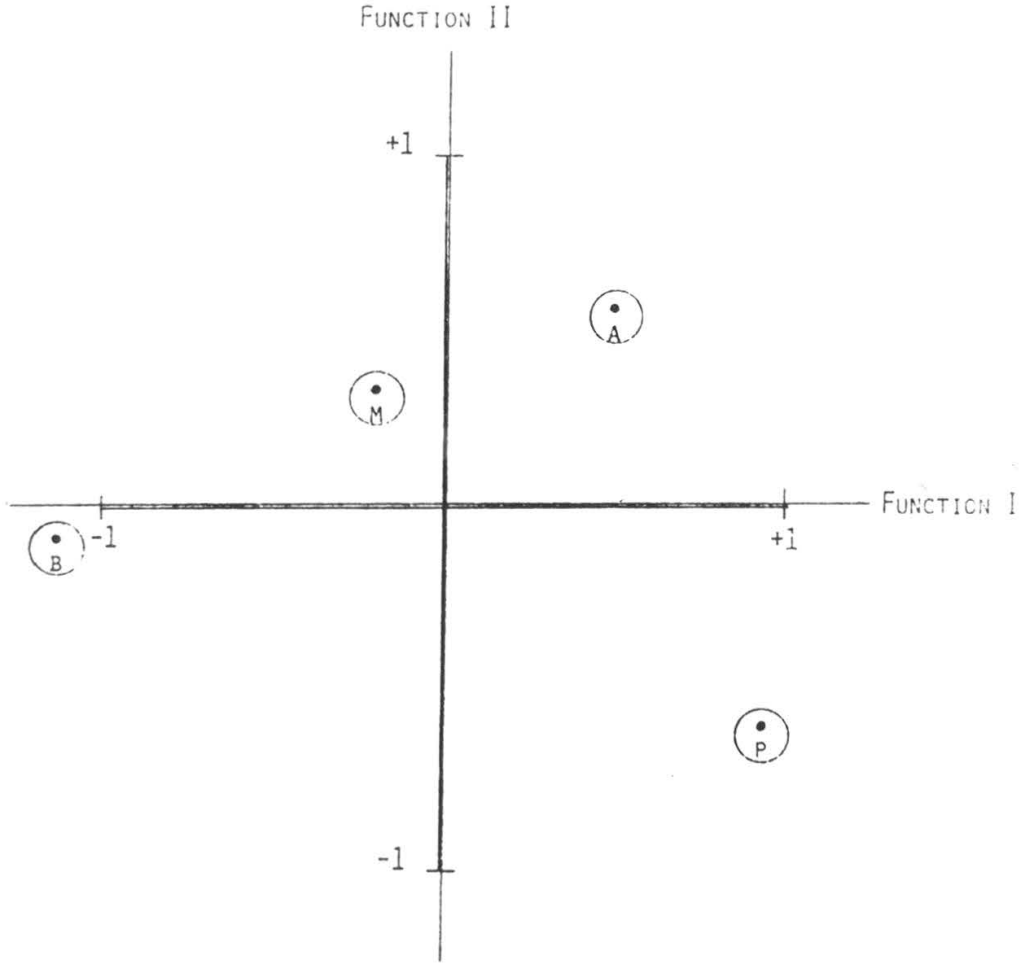


Figure 1

OVERALL DISCRIMINANT FUNCTIONS BY GROUP CENTROIDS
(RUNS 3 - 6 COMBINED)

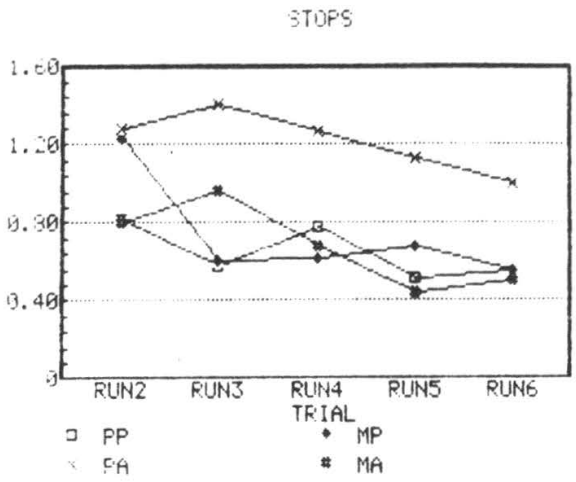


Figure 2 STOPPING ERRORS PRESENTED ACROSS TRIALS BY TREATMENT GROUP (RUNS 2-6)

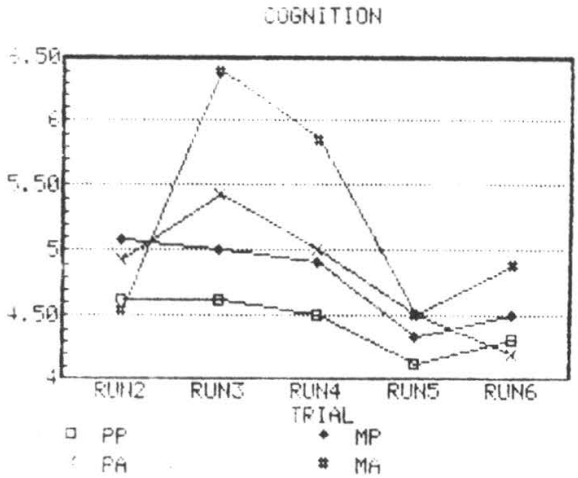


Figure 3 FIELD SOBRIETY COGNITION TASKS PRESENTED ACROSS TRIALS BY TREATMENT GROUP (RUNS 2-6)

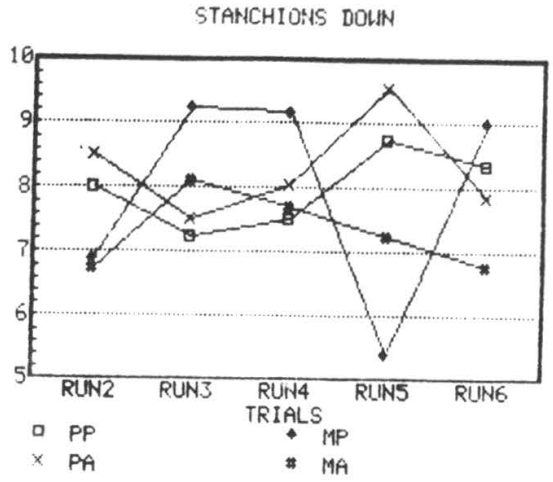


Figure 4 NUMBER OF STANCHIONS TOUCHED PRESENTED ACROSS TRIALS BY TREATMENT GROUP (RUNS 2-6)

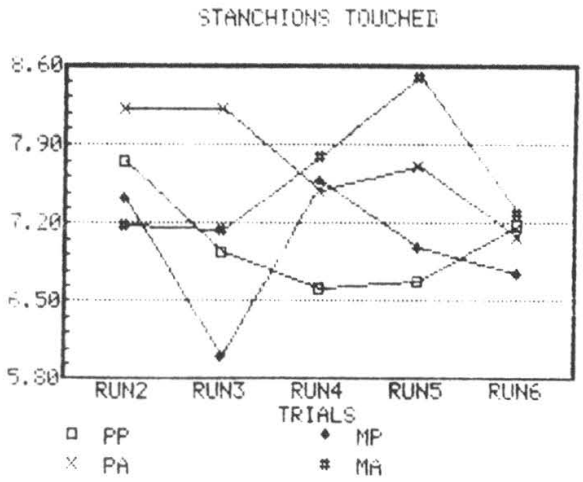


Figure 5 NUMBER OF STANCHIONS DOWN PRESENTED ACROSS TRIALS BY TREATMENT GROUP (RUNS 2-6)

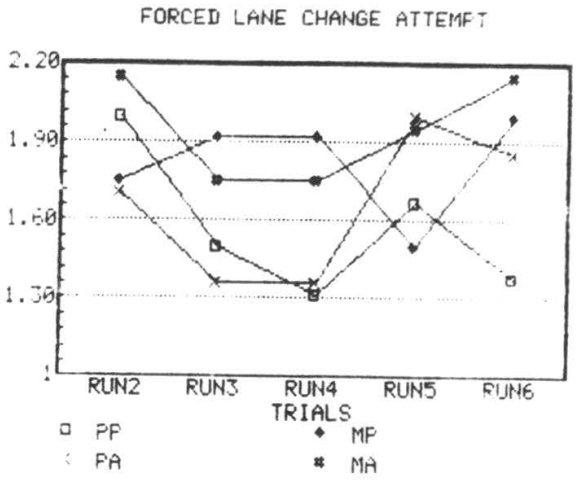


Figure 6 ATTEMPTS THROUGH THE FORCED LANE CHANGE PRESENTED ACROSS TRIALS BY TREATMENT GROUP (RUNS 2-6)

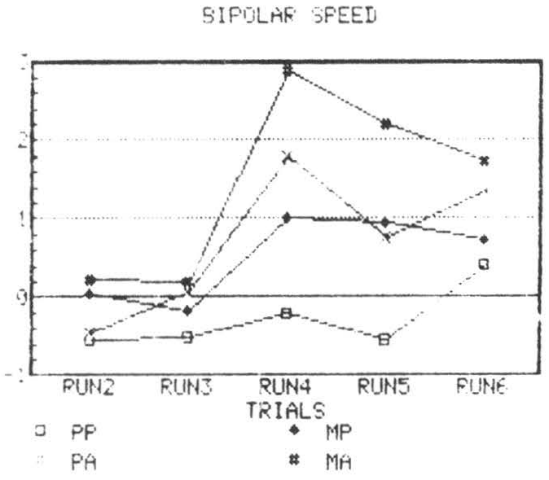


Figure 7 BIPOLAR SPEED CONTROL PRESENTED ACROSS TRIALS BY TREATMENT GROUP (RUNS 2-6)

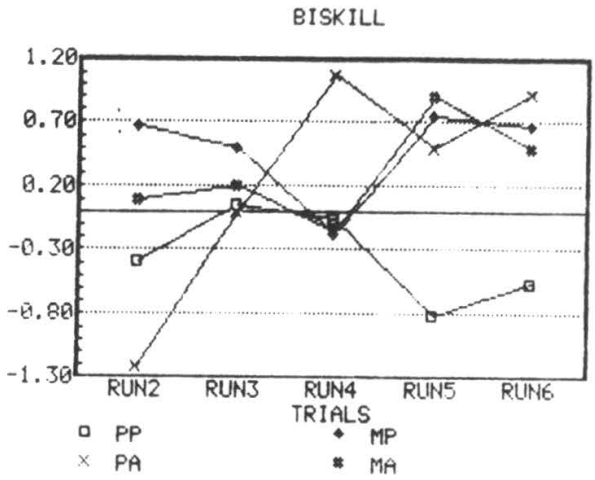


Figure 8 PERFORMANCE ON BIPOLAR SKILLS TASKS PRESENTED ACROSS ACROSS TRIALS BY TREATMENT GROUP (RUNS 2-6)

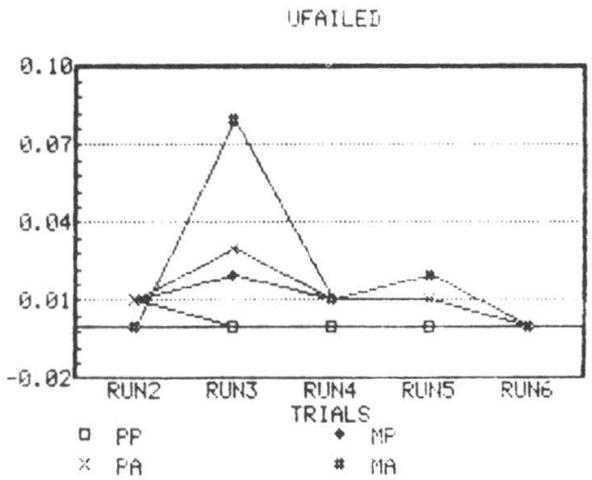


Figure 9 FAILURE TO FOLLOW URBAN DRIVE INSTRUCTIONS PRESENTED ACROSS TRIALS BY TREATMENT GROUP (RUNS 2-6)

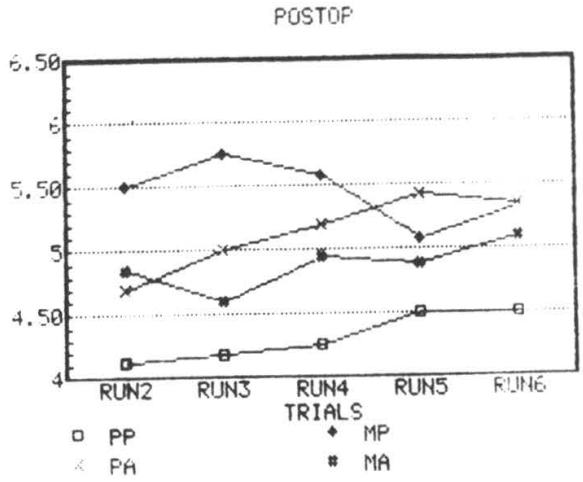


Figure 10 POSITION OF STOP PRESENTED ACROSS TRIALS BY TREATMENT GROUP (RUNS 2-6)

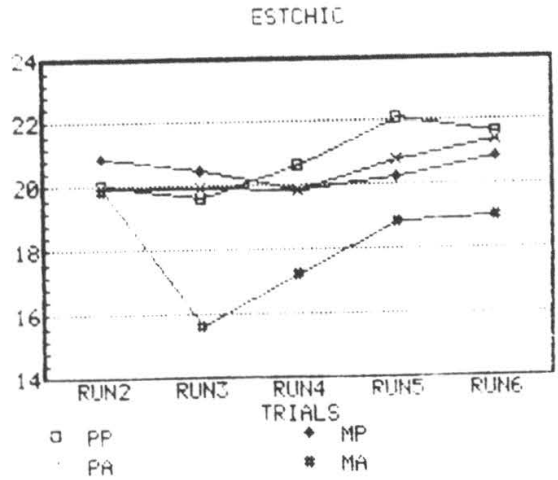


Figure 11 SUBJECT SPEED ESTIMATES THROUGH THE CHICANE PRESENTED ACROSS TRIALS BY TREATMENT GROUP (RUNS 2-6)

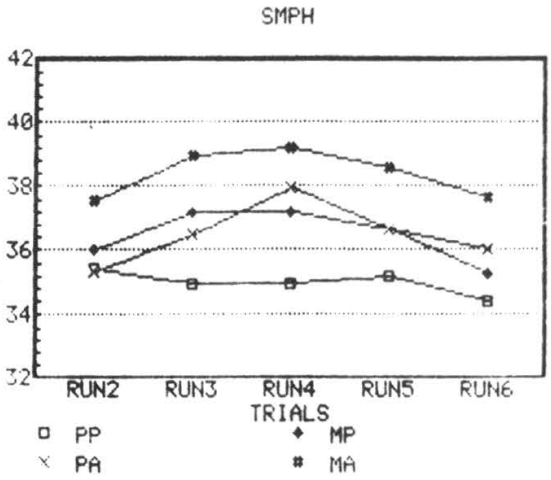


Figure 12 MILES PER HOURS THROUGH THE SPEEDOMETER COVERED PORTION OF THE EXTENDED DRIVE PRESENTED ACROSS TRIALS BY TREATMENT GROUP (RUNS 2-6)

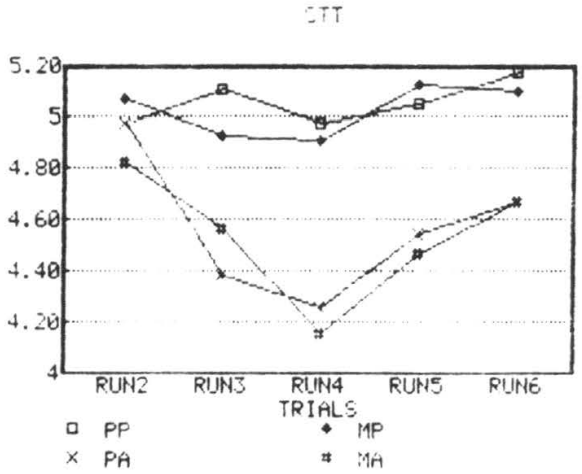
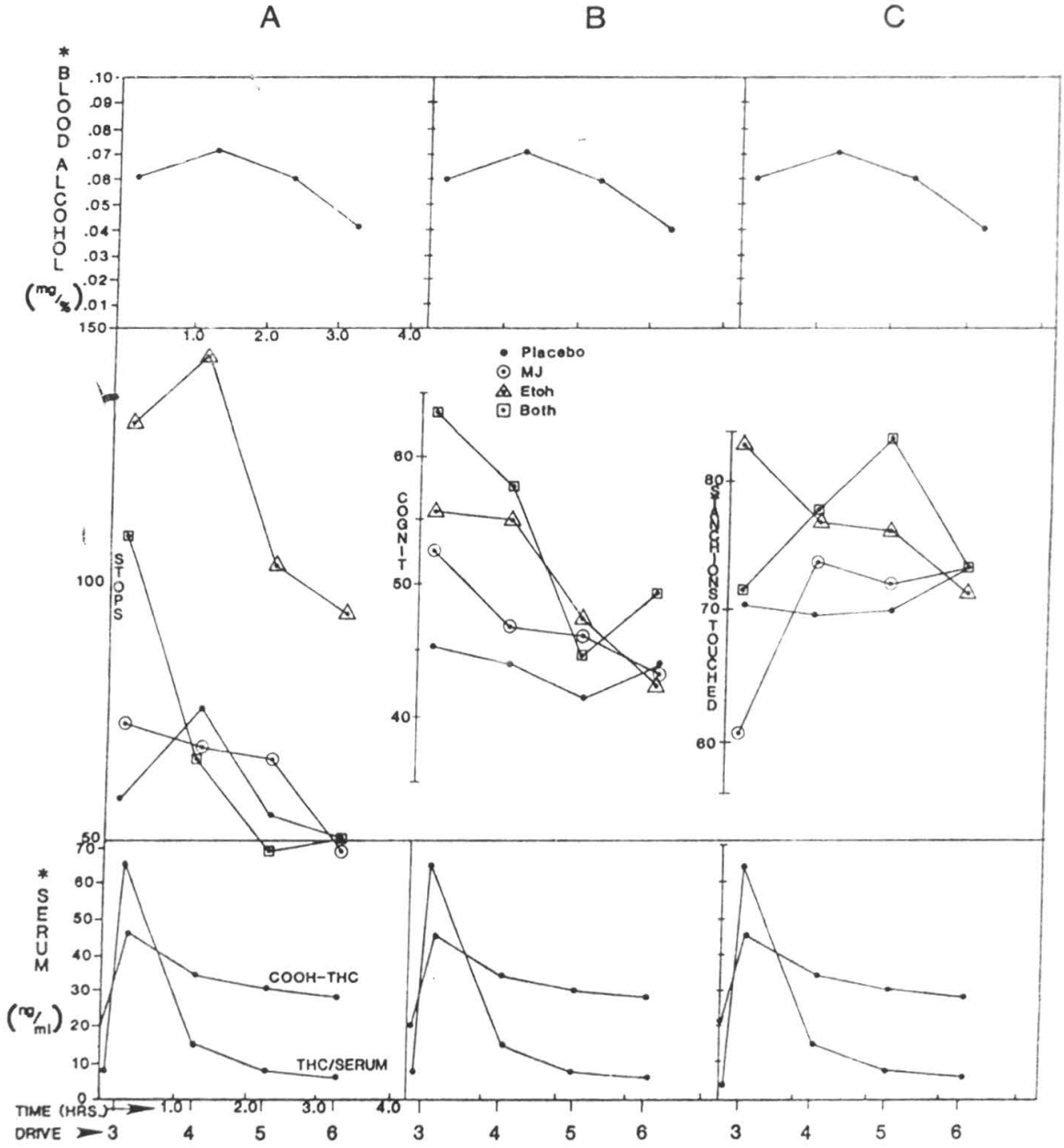


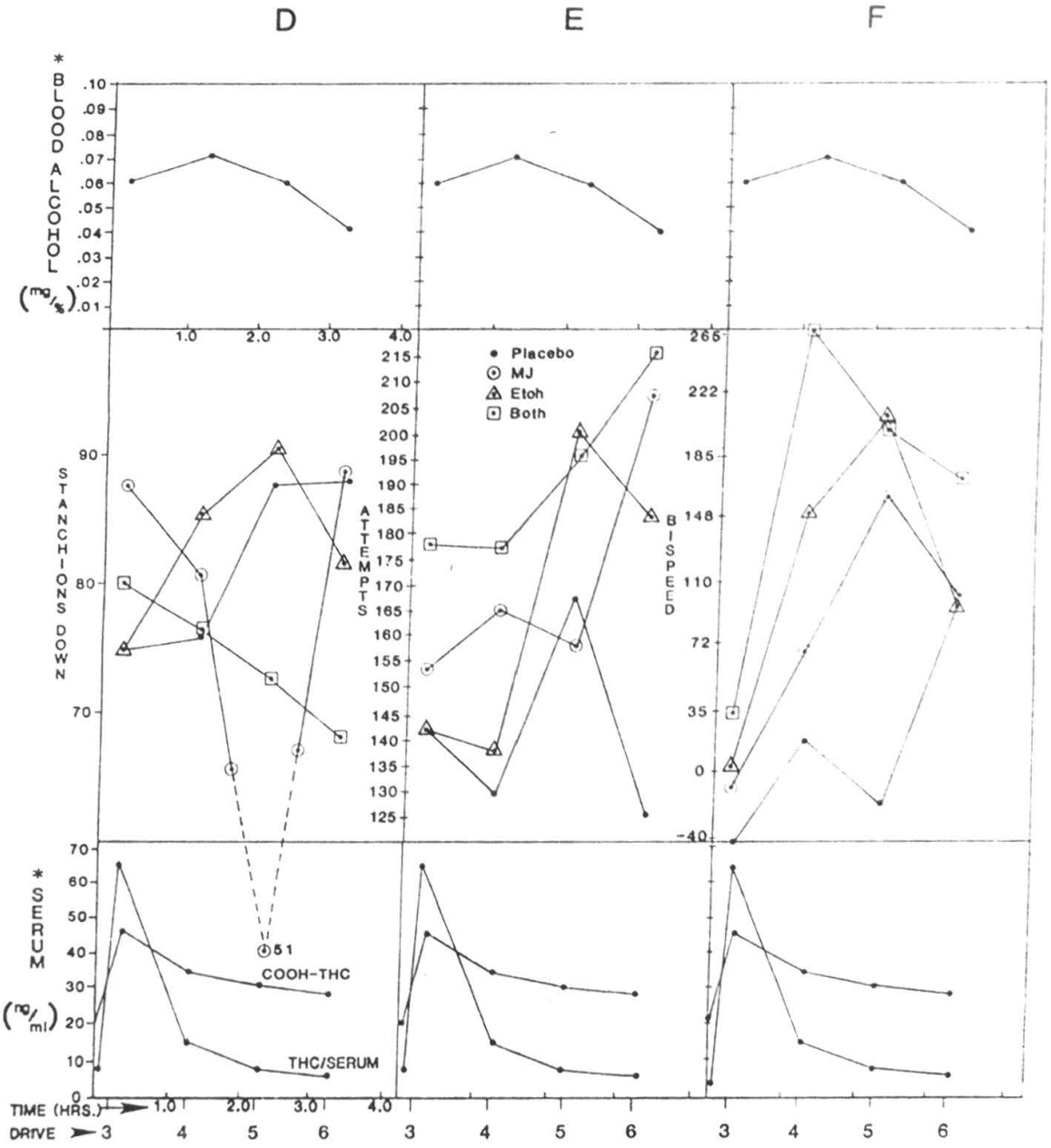
Figure 13 CRITICAL TRACKING TASK PRESENTED ACROSS TRIALS BY TREATMENT GROUP (RUNS 2-6)

Fig. 14 A - PERFORMANCE FACTORS BY BLOOD LEVELS *



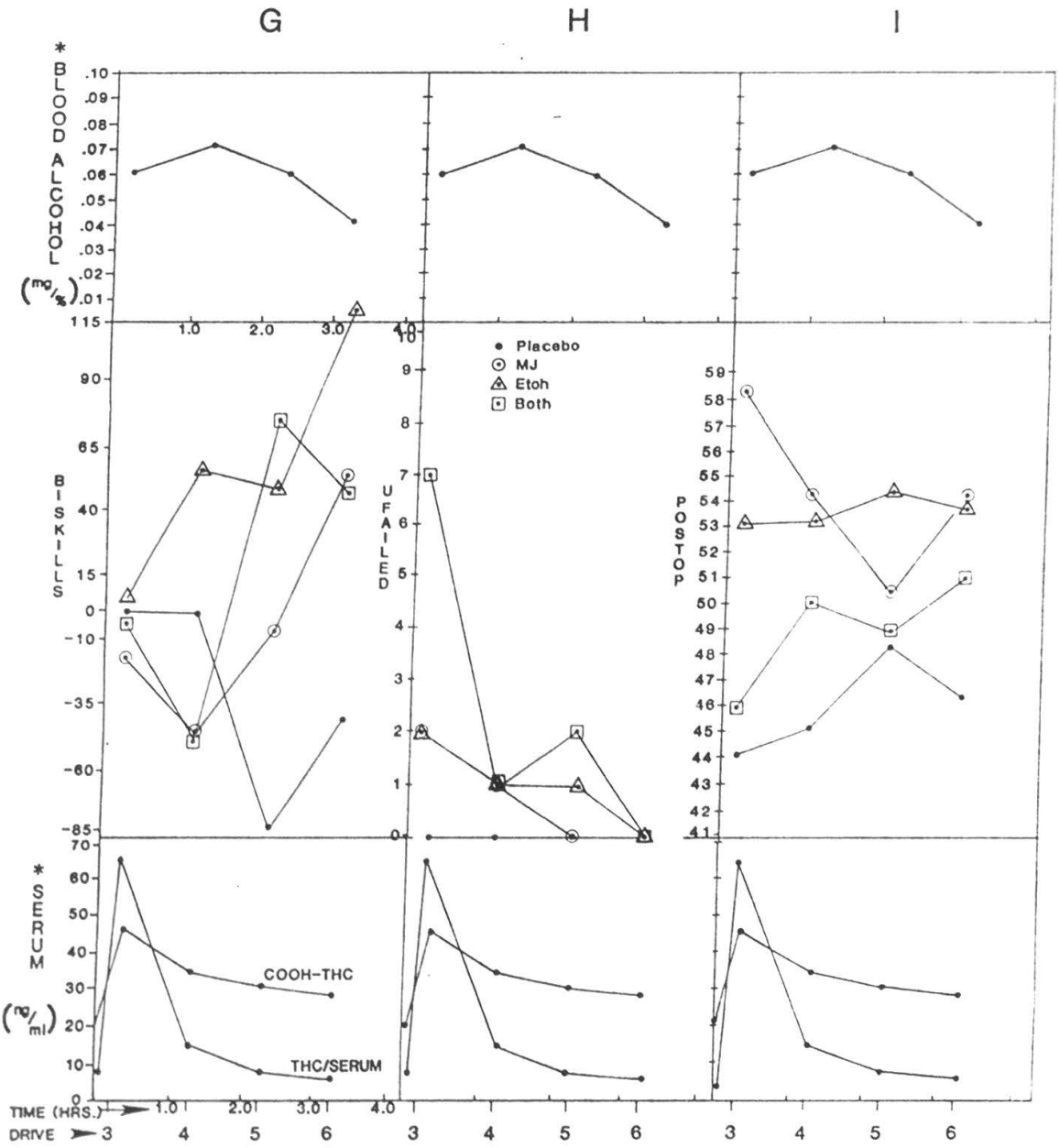
* AVERAGE OVER ALL GROUPS

Fig. 14B - PERFORMANCE FACTORS BY BLOOD LEVELS *



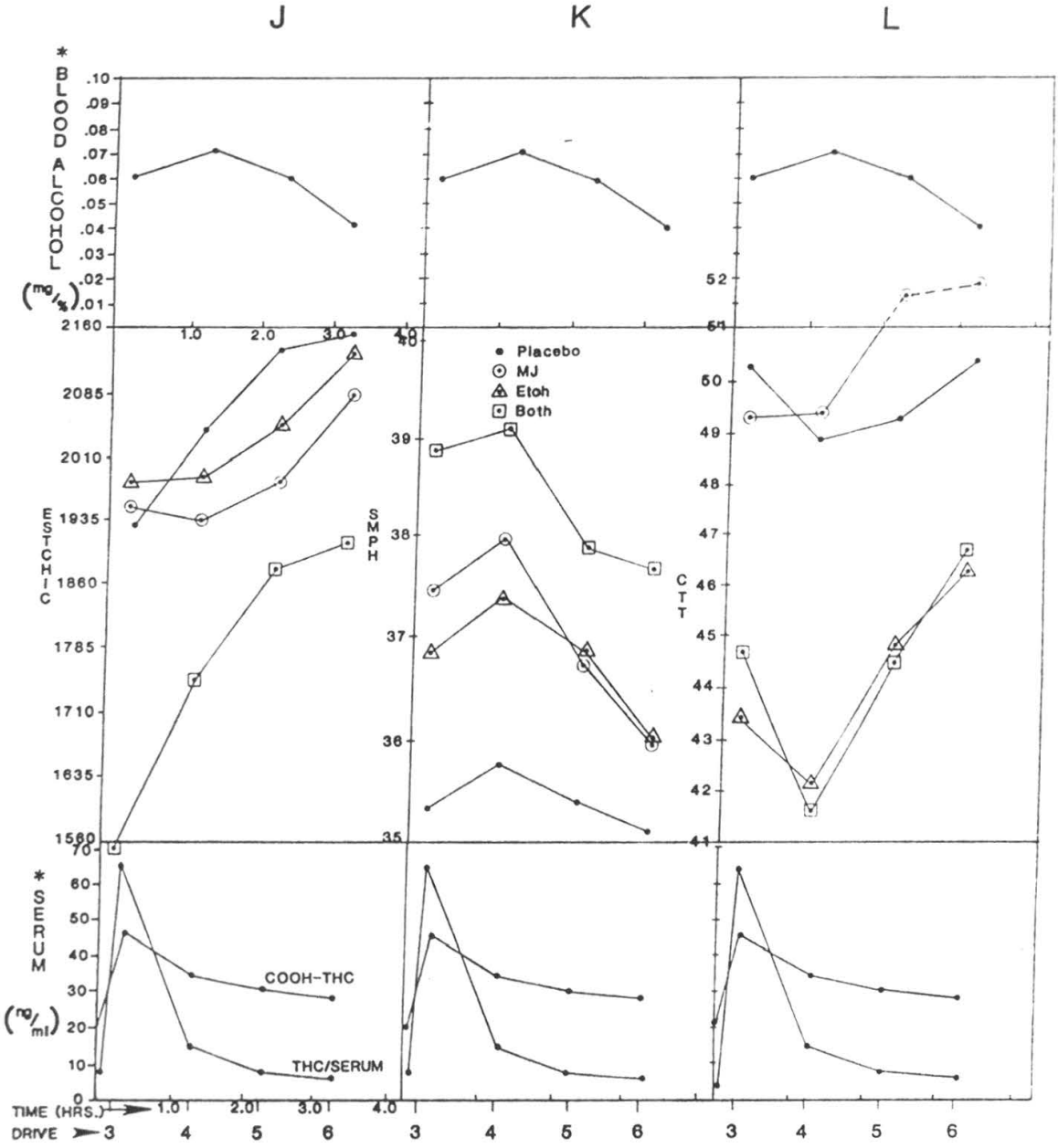
* AVERAGE OVER ALL GROUPS

Fig. 14C - PERFORMANCE FACTORS BY BLOOD LEVELS *

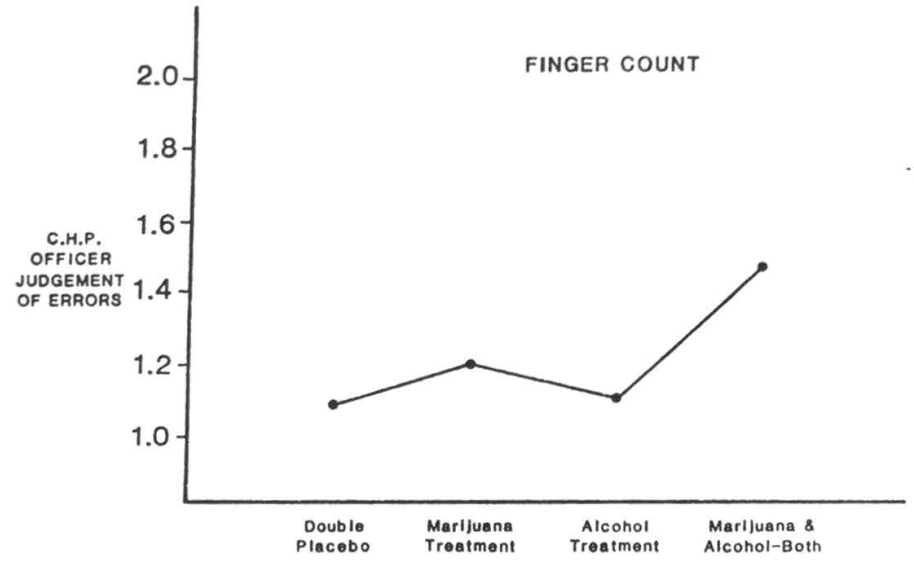


* AVERAGE OVER ALL GROUPS

Fig. 14D - PERFORMANCE FACTORS BY BLOOD LEVELS *



* AVERAGE OVER ALL GROUPS



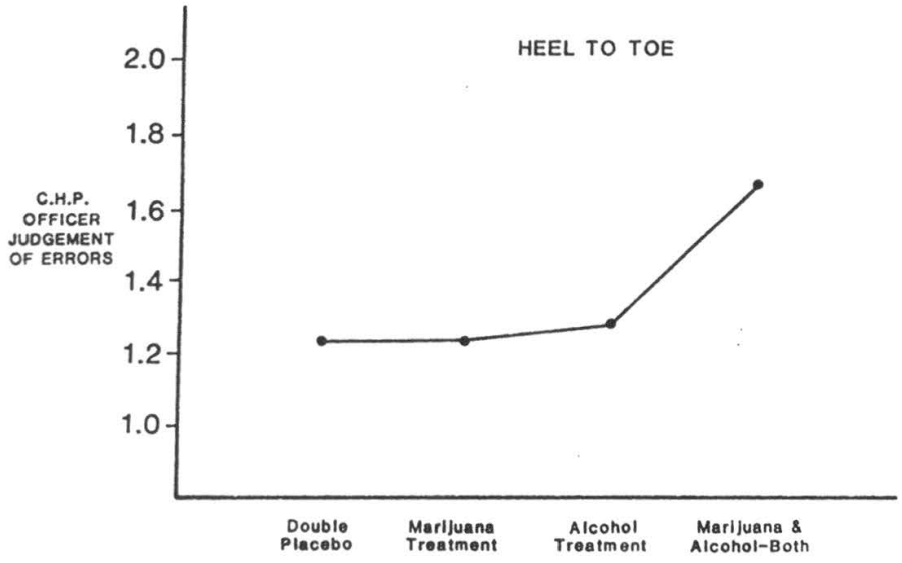
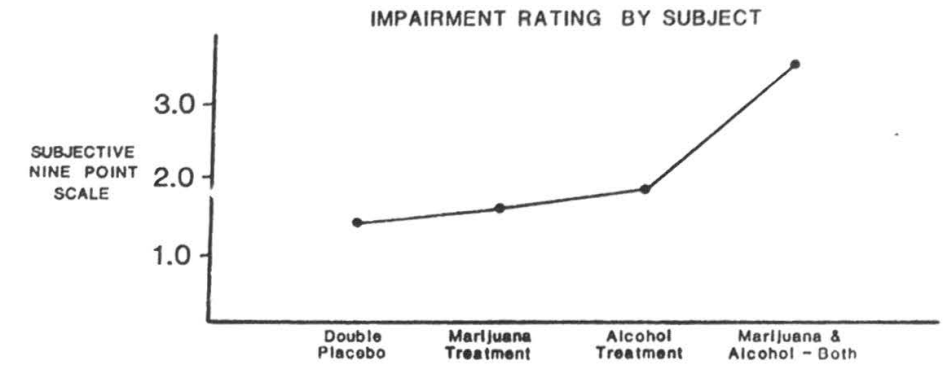
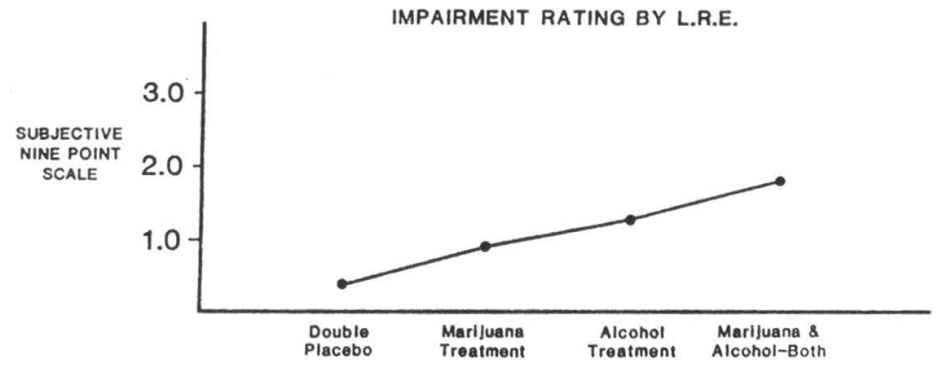


Figure 15

POST TREATMENT PERFORMANCE ON TWO FIELD SOBRIETY TASKS (RUNS 3 - 6 COMBINED)





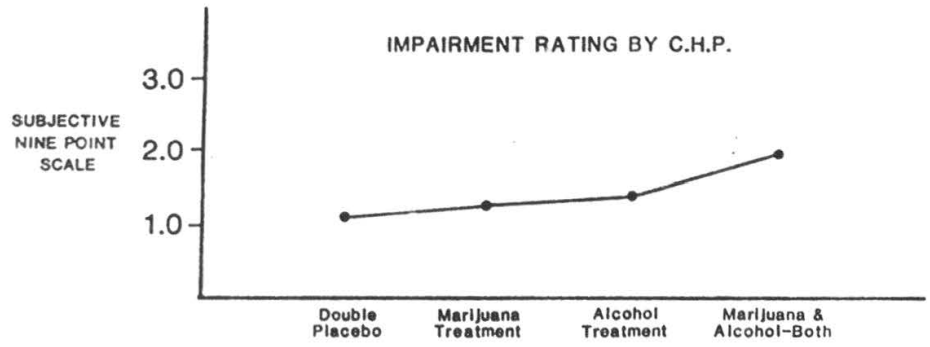


Figure 16

SUBJECTIVE IMPAIRMENT RATINGS BY TREATMENT GROUP (RUNS 3 - 6 COMBINED)

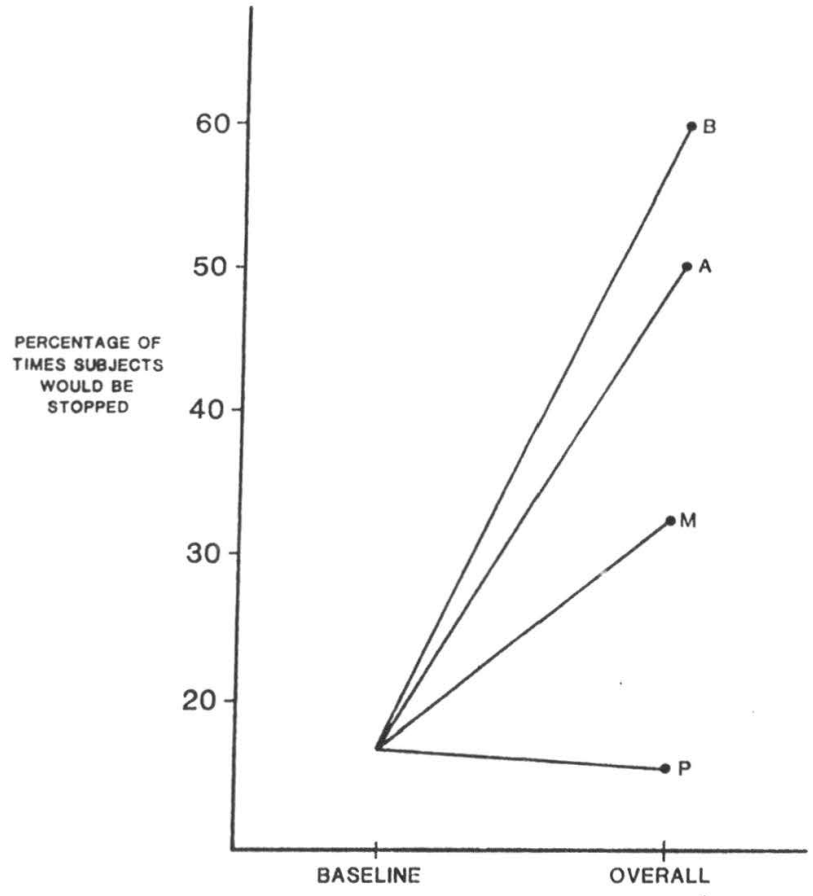


Figure 17

TREATMENT EFFECTS ON CH CAR - FOLLOWING RATING (RUNS 3 - 6 COMBINED)

Table 1 Blood and Serum Subsampling Procedures

For Serum: B-D #6441 Vacutainer, 10 ml sterile
(2 tubes/subject) (No perservative or coating)

For Blood: B-D #6472 Vacutainer, 7 ml sterile
(1 tube/subject) (EDTA 7 mg + 17.5 mg NaF)

Total subjects samples = 100
6 subjects/day X 5 blood + 5 serum/subject's = 30 blood + 30 serum/day

Vacutainer subsampling into glass/Teflon capped vials
(Blind code numbered)

Type Sample	Distribution of Subsample Vials		
	Two RIA Labs	GC/MS Lab	Reserve
Blood	1 ml samples into 2 X 2 ml vials = 2 ml	2 ml samples into 4 ml vial = 2 ml	2 to 3 ml samples into 4 ml vial
Serum	same	same	same
Total Vials/Day	120 X 2 ml	60 X 4 ml	60 X 4 ml

Table 2 Summary Of Subjects Personal History By Treatment Group

Discription	Treatment* P/P	P/A	M/P	M/A	All Groups Combined
Number of subjects	25	25	26	24	99
Average age in years	26.9	25.2	24.9	26.0	25.75
Average education in years	15.3	14.6	14.3	14.7	14.73
Average drinks per week	10.2	9.0	6.8	6.7	8.18
Average joints per week	3.4	6.4	3.4	4.2	4.35
Average age of first marijuana use in years	17.5	16.7	16.3	15.8	16.58
Average driving experience in years	10.5	9.6	8.8	9.9	9.70
Average times driven under influence of alcohol	28.3	18.5	28.8	20.7	21.58
Average times driven under influence of marijuana	19.4	21.1	18.5	25.8	21.20
Percentage not married	60.0	64.0	65.4	70.1	65.6
Percentage Caucasian	88.0	88.0	96.2	95.8	92.9
Percentage professional drivers	0	4.0	15.4	8.3	7.1
Percentage with moving violations (3 year)	40.0	60.0	38.5	37.5	43.4
Percentage with reportable accidents (5 years)	20.0	32.0	30.8	37.5	29.3
Percentage with DUI convictions	0	0	0	4.2	1.0

* Treatment Code: P/P = Placebo Marijuana and Alcohol; P/A = Placebo Marijuana and Alcohol;
M/P = Marijuana and Placebo Alcohol; M/A = Marijuana and Alcohol.

Table 3 Number of Subjects By Treatment Condition

Double Placebo			Alcohol Only		
Total Run - - - - - - - - -		25	Total Run - - - - - - - - -		25
Total Removed For:			Total Removed For:		
Pilot Data - - - -	3		Pilot Data - - - -	4	
Drug Contamination	2		Drug Contamination	1	
Total		5	Total		5
Total Analyzed		20	Total Analyzed		20
Percent Removed		20%	Percent Removed		20%
Marijuana Only			**Alcohol and Marijuana**		
Total Run - - - - - - - - -		26	Total Run - - - - - - - - -		24
Total Removed For:			Total Removed For:		
Pilot Data - - - -	2		Pilot Data - - - -	2	
Drug Contamination	3		Drug Contamination	0	
Total		5	Total		2
Total Analyzed		21	Total Analyzed		22
Percent Removed		19%	Percent Removed		.08%

Table 4 Summary of Within-Domain Factor Analyses

Domain	Number of variables correlated	Number of factors extracted*	Percent of total variance explained	Percent of variance explained by first principal component (unrotated)
Inside Rater (LRE)	60	21	61.7	8.3
Outside Rater	7	1	61.4	61.4
Field Sobriety Test	9	4	68.8	29.9
Vehicle Sensor	18	6	74.1	32.0

* Based of number of eigenvalues greater than 1.0.

Table 5 Rotated Factor Loading Matrix - Global Analysis (Varimax Rotation)

	Factor									
Variables	1	2	3	4	5	6	7	8	9	10
Uspeed		.24					.26	-.21	-.31	
Uturns	.21	.21				.31				
Ustops		.75							-.20	
Ufailed	.21	.29				-.20				
Chicane							.42			
Forlane			-.86							
Backup			-.26				.72			
Postop		-.21							.87	
Elanepos						.85				
Esteer					.27	.39	.49	.23		
Espeed								-.51	-.46	
Estop		.69								
Detour	.25				.25	.65				
Sspeed				.34			.23		-.44	
Slanepos					.85					
Ssteer					.85		.25			
Sstop	.27									-.93
Riskch										
Riskqu										.20
Estchic		.28		.31		-.33				
Flremind			.81							
Flmph			.90							
Flresp			.60							
Flrerun								.21	-.22	
Smph1				.78						.23
Smph2				.93						
Smph3				.94						
Romb		.43						.30	-.26	
Fingnose										
Heeltoe	.73									
Righfoot	.84									
Leftfoot	.61									
Fingcout	.68									
Handcoun	.43		-.20		.20					
Countbac								-.33		
Alph	.37									
Offrate	.68								.23	
Selfrate										
Bichic							-.20			
Bforl			.88				-.70			
Biback			.27						.87	
Bipost		-.21								
Bielane					.28	.76				
Biesteer		.20	-.20			.28	.42			
Biespeed								.84		
Biestop		.82								
Bidet	.31					.62				
Bisspeed				.49				.52		
Bislane					.77					
Bissteer					.85		.25			
Bisstop		.30								
Buspeed										
Buspeer										
Bustops		.78								
CTT	-.42				-.26		.27			
BITE										
Smph				.97						
Sensor	-.21	.29	.40		-.22					
Lines										
Time				-.28						
Stops	.20	.78								
Errors			-.30		.26	.51	.57			
Risk										-.61
Coord	.90									
Cognit	.77									
Eyehand		.21								.21
Stouch									-.20	
Sdown			.20							.92
Attempts								.88		
Bspeed				.26						
Bsteer		.20		.61	.60					
Bistop		.91								
Biskills			.62				-.32			

Table 5 Rotated Factor Loading Matrix - Global Analysis (continued)

Variables	Factor 11	12	13	14	15	16	17	18	19	20	21
USPEED	.20		.63						-.20		
UTURNS		.72				.23					
USTOPS						.20		-.27			
UFAILED		.49	.46	-.69							
CHICANE											
Forlane						.20					
Backup											
Postop										-.28	
Elanepos											
Esteer							.25		.24		
Espeed		.25							.22		
Estop		.32					-.32	.23			
Detour									-.21		
Sspeed	.36		.21								
Slanepos											
Ssteer					.32	.71					
Sstop											
Riskch					.84						
Riskqu	.26								-.28		
Estchic	.26										
Flremind											
Flmph			-.30	-.40							
Flresp				-.60					-.21		
Flrerun											
Smph1											
Smph2											
Smph3											-.35
Romb	.24										
Fingnose	.90										
Heeltoe											
Righfoot					.24			-.31		.34	
Leftfoot								.25	-.25	-.33	
Fingcout							.22		-.23	.35	.32
Handcout						.81				.23	
Countbac											.25
Alph			.23		-.41				.34		
Offrate	.20										
Selfrate		-.35		.21		.32	.29	.50			
Bichic				.79							
Bforl						.22					
Biback											
Bipost											
Bielane					.20		.35				
Biesteer											
Biespeed											
Biestop							-.21				
Bidet		.29							.24	.30	
Bisspeed											
Bislane											
Bissteer								.74			
Bisstop											
Buspeed			-.79								
Buspeer		-.78									
Bustops							-.46	.25			.25
CTT							-.74				
BITE											
Smph						-.33		-.30			
Sensor		-.32							.65		
Lines					.26						
Time	-.21		.68								
Stops		.30				.23					
Errors				-.27							
Risk	.23				.67						
Coord											.25
Cognit						.25					
Eyehand	.89									-.72	
Stouch					.28	-.20					.70
Sdown											
Attempts											
Bspeed			-.22								
Bsteer								.25			
Bistop											
Biskills				.52							

Table 6 Total Percent Of Variance Explained By Each Of 21 Final Factors From Global Factor Analysis Adjusted For Redundant Variance

		Percent of Variance		Cumulative Percent of Variance	
Factor	Eigenvalue	Prior To Adjustment	Adjusted	Prior To Adjustment	Adjusted
1	8.73850	12.0	11.74	12.0	11.74
2	6.64960	9.1	8.99	21.0	20.73
3	5.81619	8.0	7.90	29.0	28.63
4	4.84857	6.6	5.13	35.7	33.76
5	4.36177	6.0	6.30	41.7	40.06
6	3.87837	5.3	5.29	47.0	45.35
7	3.31530	4.5	4.52	51.5	49.87
8	2.90219	4.0	4.14	55.5	54.01
9	2.30981	3.2	3.33	58.7	57.34
10	2.03114	2.8	2.55	61.4	59.89
11	2.02750	2.8	2.49	64.2	62.38
12	1.85514	2.5	2.61	66.8	64.99
13	1.76940	2.4	2.20	69.2	67.60
14	1.72179	2.4	2.31	71.5	69.91
15	1.56006	2.1	2.08	73.7	71.99
16	1.51540	2.1	2.06	75.8	74.05
17	1.39449	1.9	2.05	77.7	76.10
18	1.30013	1.8	1.62	79.4	77.72
19	1.18239	1.6	1.22	81.1	78.94
20	1.13260	1.6	1.43	82.6	80.37
21	1.02419	1.4	1.38	84.0	81.75

Table 7 Final Set of Variable Composites Selected From Global Analysis

Factor	Definition	Variables Comprizing Factor	* Variance	Included in set of Final 12
Inside Rater Domain:				
STOPS	The total number of errors the driver make stopping at all of the stops combined on the course.	USTOPS + ESTOP + SSTOP		*
ERRORS	The total number of errors in speed control, turn control and lane postion control in the drive.	USPEED + UTURNS + CHICANE + FORLANE + BACKUP + ELANEPOS + ESTEER + SEPEED + DETOUR + SSPEED + SLANEPOS + SSTEER		
RISK	This variable consisted of the driver's speed control on the Risk Task and his overall quality of response.	RISKCH + RISKQU		*
ATTEMPTS	This variable reflected the total number of times the driver attempted a risk in the Risk Task.	ATTEMPTS		
BISTEER	This variable reflected the driver's tendency to be reckless or cautious during turning manuvers.	BIESTEER + BISSTEER + BUSTEER + BIELANE + BISLANE		
BISPEED	This variable reflected the driver's tendency to be reckless or cautious in regard to controlling speed.	BIESPEED + BISSPEED + BUSPEED		*
BISTOP	This variable reflected the driver's tendency to be reckless or cautious during stopping manuvers.	BIESTOP + BISSTOP + BUSTOPS		
BISKILLS	This variable reflected the driver's tendency to be reckless or cautious during the Chicane and Forced Lane Change portions of the drive.	BICHIC + BFORL + BLBACK		*
UFAILED	This variable was the sum of the number of times the driver failed to follow instructions during the course of the Urban Drive.	UFAILED		*
POSTOP	This variable reflected the ability of the driver to position the vehicle at the stop line during all stopping manuvers.	POSTOP		*
ESTCHIC	This variable was the estimated time it would take the driver according to his own estimate, to traverse the Chicane portion of the drive.	ESTCHIC		*
FLREMIND	This variable was the number of times that the Inside Rater had to remind the driver of the speed requirement of the Forced Lane Change.	FLREMIND		
FLRESP	This variable reflected the quality of the driver's response to the Forced Lane Change.	FLRESP		

Table 7 (Continued) Final Set of Variable Composites Selected From Global Analysis

Factor	Definition	Variables Comprizing Factor	% Variance	Included in Set of Final 12
FLRERUN	This variable reflected the total number of times the driver was required to rerun the Forced Lane Change.	FLRERUN		
SMPH	This variable was the average speed the driver travelled on the Extended portion of the course.	SMPH1 + SMPH2 + SMPH3/3		*
OVERALL	This variable was the overall subjective rating of degree of impairment of the driver as provided by the Inside Rater.	OVERALL	61.7%	
Outside Rater Domain:				
STOUCHED	The total number of cones and stanchions touched by the driver in the skills portions of the course.	STOUCHED		*
SDOWN	The total number of cones and stanchions knocked down by the driver in the skills portions of the course.	SDOWN	63.4%	*
Sensor Domain:				
SENSOR	This variable was a combination of the brake, steering, and accelerator sensor variables across all portions of the course.	CAC1 + UAC1 + EAC1 + SAC1 + CBR1 + UBR1 + EBR1 + SBR1 + CST1 + UST1 + EST1 + SST1		
LINES	This variable reflected the degree to which the driver deviated from a fixed distance from the edge of the road over the Urban and Extended portions of the course.	ULT1 + ELI1 + SLI1		
TIME	The total time the driver took to complete the entire drive.	ULI6 + ELI6 + SLI6	72.9% (52.4)	
Field Sobriety Test Domain:				
COGNITION	This factor consisted of the hand count, the counting backwards, and the finger count tasks.	FINGCOUNT + HANDCOUNT + COUNTBAC + ALPH		*
COORDINATION	This factor consisted of the heel to toe, standing on the left foot, and standing on the right foot tasks.	HEELTOE + LEFTFOOT + RIGHTFOOT		
EYEHAND	This factor consisted of the romderg body sway task and the finger to nose task.	ROMB + FINGNOSE	68.8% (56.7)	

Table 7 (Continued) Final Set of Variable Composites Selected From Global Analysis

Factor	Definition	Variables Comprizing Factor	% Variance	Included in set of Final 12
Special Variables Domain*:				
FALLCAR	This variable reflected the CHP following car officer's tendency to want to stop the driver for probable cause.	FALLCAR		
CTT	This variable was the scores obtained on the Critical Tracking Task.	CTT3 + CTT4 + CTT5 + CTT6/4		*
BITE	This was the overall score obtained during all runs on the Brief Interval Time Estimation task.	BITE3 + BITE4 + BITE5 + BITE6/4		

Table 8 Between-Run Correlations For 28 Variables From Global Factor Analysis

Variable (By Domain)	WITH RUN 2				AVERAGE OF:		
	Run 3	Run 4	Run 5	Run 6	Runs 2-6	Runs 3-6	Runs 3-6 with 2
SENSOR DOMAIN:							
SENSOR	.85	.47	.46	.46	.67	.74	.56
LINES	.75	.74	.67	.56	.71	.74	.68
TIME	.43	.23	-.03	.15	.19	.19	.19
FIELD SOBRIETY TEST DOMAIN:							
COORD	.41	.55	.63	.55	.57	.59	.53
COGNIT	.26	.14	.36	.05	.31	.34	.26
EYE-HAND	.25	.42	.43	.06	.26	.24	.29
OUTSIDE RATER DOMAIN:							
S TOUCH	.20	-.11	-.15	.01	.10	.18	-.01
S DOWN	-.25	-.00	.08	.12	-.01	-.01	-.01
INSIDE RATER DOMAIN:							
STOPS	.65	.63	.60	.50	.57	.56	.59
ERRORS	.53	.47	.39	.29	.56	.66	.42
RISK	.31	.22	.38	.09	.21	.18	.25
ATTEMPTS	.31	.52	.54	.27	.38	.36	.41
B SPEED	.16	.16	.20	.32	.42	.56	.21
B STEER	.52	.32	.12	.17	.35	.39	.28
BI STOPS	.57	.50	.41	.44	.41	.36	.48
BI SKILL	.35	.23	.20	.20	.30	.33	.24
U FAILED	-.02	.20	.17	-.04	.06	05	.07
P.O. STOP	.62	.65	.52	.45	.59	.62	.56
Fst. CHIC	.52	.57	.47	.51	.61	.67	.51
F.L.REMIND	-.02	-.03	-.02	.01	-.01	.00	-.01
F.L.RESPONSE	.31	.34	.10	.36	.26	.25	.27
F.L.RERUN	.10	.15	.19	-.07	.15	.19	.09
SMPH	.65	.57	.57	.46	.65	.71	.56
OVERALL	.24	.18	.26	.24	.51	.71	.23
SPECIAL VARIABLES DOMAIN:							
FALL CAR	.34	.02	.35	.21	.25	.26	.22
OFF RATE	.34	.29	.50	.47	.48	.53	.40
CTT	.40	.69	.74	.68	.61	.66	.62
BITE	.63	.73	.58	.49	.65	.68	.60
*ACCELERATOR	.79	.62	.67	.68	.72	.73	.69
SPEED	.16	.51	.33	.41	.48	.57	.35
*STEERING	.77	.23	.28	.32	.58	.69	.40
LINES	.75	.74	.66	.55	.71	.74	.67
*BRAKING	.79	.65	.51	.40	.58	.58	.59
OVERALL AVERAGE					.61	.66	.54

*Prior to being combined with other variables forming the "SENSOR" factor.

Table 9 Bias For Baseline Measures (run 2) From Univariate Analyses of Variance

Variable	F value	Variable	F value
SENSOR	0.30	BISKILLS	0.12
LINES	0.95	CLI6	0.10
TIME	0.51	UFAILED	0.61
STOPS	0.66	POSTOP	3.25*
ERRORS	0.49	ESTCHIC	0.22
RISK	1.57	FLREMIND	0.10
COORD	0.50	FLRESPONSE	1.41
COGNIT	0.12	FLRERUN	0.51
EYEHAND	0.66	SMPH	3.33*
STOUCHED	0.75	OVERALL	0.69
SDOWN	0.91	FALLCAR	0.17
ATTEMPTS	0.59	OFFRATE	1.09
BSPEED	1.51	CTT	1.33
BSTEER	1.58	BITE	0.04

*$p < .05$

Table 10 Test For Homogeneity of the Within Treatment Regression Slopes Between Baseline (run 2) and Post-Treatment Performance (runs 3-6, combined) On Each Measure

Variable*	R^2 Using Combined	R^2 Using Separate Slopes	F Ratio of Differences	Significance
STOPS	.49	.53	2.92	.05
ATTEMPTS	.37	.40	4.24	.01
BISKILLS	.12	.18	0.01	NS
POSTOP	.49	.51	9.36	.01
ESTCHIC	.36	.54	12.25	.01
SMPH	.41	.50	7.19	.01
CTT	.53	.66	18.54	.01

* Includes only variables with correlations equal to or greater than .30.

(12)

Table 11 Subject Background Variables Correlated With 29 Composite Performance Variables

Performance Variables (Runs 3-6)	Subject Background Variables				
	Driving Experience	Average Weekly Alchhol Intake	Driving Experience Under Alcohol	Average Weekly Marijuana Intake	Driving Experience Under Marijuana
SENSOR	.03	.00	.12	.12	.19*
LINES	-.15	.18*	-.02	.12	-.07
TIME	.04	.08	-.19*	-.09	-.17
STOPS	.06	-.06	-.27*	-.08	-.10
ERRORS	.10	-.12	-.22*	-.17	-.07
RISK	.05	.35*	.25*	.10	-.03
COORD	.08	-.13	-.13	-.02	-.02
COGNIT	.04	-.13	-.10	.11	.01
EYEHAND	.28*	.05	-.14	.05	-.01
STOUCH	.27*	-.12	-.19*	.33*	.10
SDOWN	-.00	.22*	.01	.10	.03
ATTEMPT	.25*	-.15	-.04	.20*	-.03
BSPEED	.08	-.09	.01	.03	.06
BSTEER	.19*	-.05	-.03	-.10	-.11
BISTOP	.21*	.04	-.06	.07	-.02
BISKILS	-.20*	-.14	.01	-.16	-.10
CLI7	.14	-.00	-.21*	.06	-.11
UFAILED	.03	.13	-.10	-.09	-.07
POSTOP	.07	-.01	.18*	.13	.09
ESTCHIC	-.07	.09	.17	-.01	.04
FLREMIND	-.03	.06	.08	.08	.11
FLRESP	.12	-.01	-.02	.29*	.02
FLRERUN	-.05	-.03	-.17	-.03	-.18*
SMPH	.11	-.06	-.18*	-.04	-.15
OVERALL	-.01	-.06	-.14	-.11	-.10
FALLCAR	.17	-.12	-.17	.11	-.04
OFFRATE	.11	-.10	-.25*	.08	-.02
CTT	-.33*	.03	.10	-.35*	-.02
BITE	-.01	.12	.23*	-.00	.20*

* $P < .05$

Table 12 Significant Variables For Each Discriminant Function Analysis To Establish Best Discriminators

	Run 3	Run 4	Run 5	Run 6	Run Total
	STOPS	STOPS	STOPS	STOPS	STOPS
	POSTOP	POSTOP	POSTOP	POSTOP	POSTOP
	ESTCHIC	ESTCHIC	ESTCHIC	ESTCHIC	ESTCHIC
	CTT	CTT	CTT	CTT	CTT
	SMPH	SMPH	BISKILLS	BISKILLS	SMPH
	STOUCH	SENSOR	SDOWN	SDOWN	STOUCH
	ATTEMPTS	FLRERUN	UFAILED	UFAILED	UFAILED
	OVERALL	COGNIT	BSPEED	COGNIT	BSPEED
		OFFRATE	BISTOP	LINES	ATTEMPTS
			FALLCAR	FLRESP	TIME
				FLREMIND	ERRORS
					BLANE
	- - - -	- - - -	- - - -	- - - -	- - - -
Lambda:	.313*	.318*	.302*	.261*	.265*
Number of Significant Functions**	3	2	2	2	2

* Wilk's Lambda for all 3 functions.
** $p < .05$

Table 13 Standardized Discriminant Functions For Final 12 Variables

VARIABLE	DRIVE 3		DRIVE 4		DRIVE 5		DRIVE 6		TOTAL COMPOSITE DRIVES	
	FUNCTIONS I	II	FUNCTIONS I	II	FUNCTIONS I	II	FUNCTIONS I	II	FUNCTIONS I	II
STANCHIONS TOUCHED	-0.22	0.67	-0.08	.28	0.12	0.05	-0.10	-0.04	-0.72	0.81
ESTIMATED CHICANE (DEVIATION)	-0.73	0.44	-0.48	.38	-0.51	0.42	-0.81	0.12	-0.87	0.41
S MPH (SPEED COVERED)	0.70	0.00	0.43	-.13	0.39	-0.11	0.38	0.09	0.68	-0.28
ATTEMPTS (RISK TASK)	0.47	-0.09	0.24	-.39	0.22	-0.01	0.90	0.22	0.63	-0.19
COGNIT	0.32	0.39	0.51	.07	-0.12	0.06	-0.15	-0.77	0.46	0.04
URBAN FAILURES	0.23	-0.07	0.20	-.06	0.42	0.21	0.17	-0.54	0.42	-0.11
STANCHIONS DOWN	0.14	0.00	-0.11	.10	0.60	0.60	-0.32	0.49	-0.28	0.02
POSITION OF STOP	-0.22	0.37	0.16	.27	-0.05	0.31	0.47	0.44	0.16	0.93
STOPPING ERRORS	0.27	0.68	-0.16	.54	-0.34	0.34	0.49	0.66	0.16	0.59
BIPOLAR SKILL	0.18	-0.10	-0.01	.30	0.18	0.08	0.80	0.39	0.21	0.17
CTT	-0.24	-0.63	-0.58	-.42	-0.46	-0.35	-0.15	-0.04	-0.15	-0.19
BIPOLAR SPEED	-0.22	0.17	0.06	-.16	0.46	-0.44	-0.25	-.28	-0.09	0.45
Variance explained (%)	55.1	27.9	64.6	21.9	55.3	31.8	64.7	24.9	64.3	22.3
Canonical correlation	0.68	0.56	0.67	.46	0.63	0.53	0.71	0.53	0.69	0.49
Wilks' Lambda	0.29	0.54	0.38	.67	0.36	0.61	0.30	0.62	0.33	0.64
Significance*	.001	.01	.01	.21	.001	.05	.001	.11	.001	.07

*Note: The significance of the first function is actually a test of all three functions combined whereas the significance of the second function is a test of the variance explained after the first function has been removed. The third function, which was not significant in any of the runs, represents the variance explained after the first two functions have been extracted. All subsequent discriminant functions tables should be interpreted in this fashion.

Table 14 Structure (Loading) Matrix - First Two Discriminant Functions (Runs 3 - 6 Combined)

(Correlations > .26, p < .01)

Function 1		Function 2	
EAC1	.34	UTURNS	.27
CLI6	-.40	POSTOP	.69
ULI6	-.30	ESTOP	.33
USPEED	-.28	SSTOP	.33
UFAILED	-.31	RISK QU	.28
ESTEER	-.26	SMPH2	.27
DETOUR	-.41	OVERALL	.28
SSPEED	-.32	CTT6	-.28
RISKCH	.25	BITE3	-.27
ESTCHIC	.52	BI POST	.37
SMPH1	-.39	BIE SPEED	.33
SMPH2	-.48	BIS SPEED	.28
SMPH3	-.37	BIS STOP	-.31
OVERALL	-.38	STOPS	.36
FALL CAR	-.27	ERRORS	.28
FING COUNT	-.30	BSPEED	.31
COUNTBAC	-.26		
OFFRATE	-.26		
SELFRATE	-.40		
SDOWN	.27		
ATTEMPTS	-.30		
BITE4	.31		
BITE5	.30		
BUSTOPS	.30		
BIS SPEED	-.42		
BIS STEER	-.32		
SMPH	-.45		
ERRORS	-.30		
COGNIT	-.25		
BITE	.25		
BSTEER	-.24		
BSPEED	-.25		
BISTOP	.25		

Table 15 Treatment Group Means On 12 Final Variables (Runs 3 - 6 Combined)

Variable	Placebo	Marijuana	Alcohol	Both
STOPS	1.35	1.76	2.14	1.61
COGNIT	4.34	4.59	4.66	5.09
STOUCH	28.10	25.89	28.95	29.21
SDOWN	32.15	29.05	31.25	28.04
ATTEMPTS	5.50	6.37	6.20	7.26
BSPEED	-0.89	-0.26	0.16	0.35
BSKILLS	-0.26	-0.21	0.49	0.11
UFAILED	0.00	0.05	0.04	0.10
POSTOP	4.55	5.59	5.52	4.95
ESTCHIC	20.38	19.59	20.18	17.67
SMPH	34.93	36.69	36.32	38.16
CTT	4.98	5.01	4.49	4.44

Table 16 Percent Of Variance Accounted For By Treatments On Canonical Functions

Effect	RUN 3		RUN 4		RUN 5	
	Percent of Variance Explained	p	Percent of Variance Explained	p	Percent of Variance Explained	p
Marijuana	29.2	.001	23.1	.001	23.1	.001
Alcohol	29.9	.001	32.3	.001	31.5	.001
Alcohol x Marijuana Interaction . . .	12.0	.05	7.1	N.S.	8.9	N.S
Total Explained . .	71.1	.001	62.4	.001	63.5	.001

	RUN 6		$\bar{X}$ (Trials 3-6)		ALL POST TREATMENT RUNS COMBINED	
	Percent of Variance Explained	p	Percent of Variance Explained	p	Percent of Variance Explained	p
Marijuana	24.8	.001	25.1	.001	33.2	.001
Alcohol	31.9	.001	31.4	.001	24.2	.001
Alcohol x Marijuana Interaction . . .	12.9	.05	10.2	N.S.	9.1	N.S.
Total Explained . .	69.6	.001	66.7	.001	66.5	.001

Table 17 Eigenvalues For Repeated Measures Discriminant Functions On Each Of The Twelve Performance Measures (Baseline Run Excluded)

Variable	Function I	Function II
STOPS	.14152	.06535
COGNIT	.18824	.13303
STOUCH	.29882 *	.09546
SDOWN	.37807 *	.08865
ATTEMPTS	.22442	.08453
BSPEED	.21859	.01537
BISKILLS	.15227	.05414
UFAILED	.13416	.01550
POSTOP	.20638	.01892
ESTCHIC	.34944 *	.08319
SMPH	.36481 *	.03202
CTT	.43620 *	.02778

* p < .05. Based on Wilk's Lambda with all functions included.

Table 18 Eigenvalues For Repeated Measures Discriminant Functions On Each Of The Twelve Performance Measures (Baseline Run Included)

Variable	Function I	Function II
STOPS	.15418	.10587
COGNIT	.30029	.06637
STOUCH	.29627	.11290
SDOWN	.37482	.08603
ATTEMPTS	.20250	.12097
BSPEED	.30147	.05522
BISKILLS	.13544	.12457
UFAILED	.17486	.02365
POSTOP	.31390	.07433
ESTCHIC	.49839 *	.10763
SMPH	.40701 *	.13402
CTT	.67729 **	.05050

* p < .05; ** p < .01. Significance based on Wilk's Lambda with all functions included.

Table 19 Interaction Analysis - Baseline STOPS Score x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Significance Levels)

	Post Treatment Performance Measures (Run 3-6)											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.268	.513	.100	.077	.043	.004	.000	.150	.218	.012	.741	.098
M	.499	.523	.016	.095	.315	.978	.721	.952	.196	.006	.234	.009
STOPS 2	.000	.196	.305	.554	.898	.819	.790	.712	.959	.322	.969	.414
A x M	.436	.004	.807	.915	.372	.660	.711	.817	.795	.444	.556	.126
A x STOPS 2	.456	.952	.346	.974	.411	.011	.004	.740	.737	.377	.588	.133
M x STOPS 2	.108	.700	.689	.612	.098	.662	.334	.983	.633	.652	.981	.811
A x M x STOPS 2	.402	.042	.251	.388	.887	.095	.481	.507	.277	.944	.931	.962

Table 20 Interaction Analysis - Baseline ERRORS Score x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Significance Levels)

	Post Treatment Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.770	.366	.147	.057	.035	.005	.000	.107	.238	.009	.756	.080
M	.608	.466	.020	.084	.342	.979	.712	.945	.219	.006	.240	.009
ERRORS 2	.149	.108	.109	.362	.344	.937	.510	.059	.361	.348	.662	.663
A x M	.270	.007	.834	.789	.467	.725	.732	.594	.757	.662	.575	.081
A x ERROR 2	.556	.137	.861	.466	.061	.264	.791	.173	.503	.653	.815	.026
M x ERRORS 2	.796	.125	.848	.357	.484	.462	.956	.926	.539	.099	.354	.299
A x M x ERRORS 2	.027	.098	.344	.490	.651	.172	.965	.078	.550	.265	.405	.939

Table 21 Interaction Analysis - Baseline RISK Score x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Levels Of Significance)

	Post Treatment Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.705	.454	.130	.058	.033	.005	.000	.147	.222	.008	.697	.064
M	.716	.555	.021	.178	.292	.728	.819	.880	.255	.008	.156	.005
RISK 2	.806	.915	.663	.004	.597	.034	.518	.501	.207	.313	.226	.301
A x M	.222	.008	.999	.717	.459	.819	.712	.708	.991	.381	.512	.036
A x RISK 2	.271	.767	.725	.766	.699	.234	.917	.989	.151	.373	.615	.017
M x RISK 2	.718	.453	.462	.342	.329	.104	.943	.876	.950	.071	.570	.459
A x M x RISK 2	.465	.717	.715	.108	.669	.719	.506	.022	.727	.873	.022	.194

Table 22 Interaction Analysis - Baseline POSTOP Score x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Levels Of Significance)

	Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.748	.172	.121	.069	.030	.005	.000	.163	.228	.007	.724	.057
M	.823	.098	.011	.082	.290	.941	.485	.751	.109	.004	.278	.049
POSTOP 2	.021	.000	.456	.589	.768	.868	.159	.172	.177	.592	.730	.010
A x M	.100	.124	.603	.924	.909	.564	.756	.543	.754	.991	.512	.051
A x POSTOP 2	.933	.308	.073	.749	.041	.478	.340	.914	.380	.024	.968	.456
M x POSTOP 2	.609	.357	.773	.505	.568	.468	.813	.549	.751	.828	.113	.888
A x M x POSTOP 2	.398	.317	.311	.385	.456	.137	.775	.814	.888	.246	.457	.550

Table 23 Interaction Analysis - Baseline SENSOR Score x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Levels Of Significance)

	Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.609	.513	.116	.049	.026	.004	.000	.136	.219	.004	.775	.094
M	.968	.805	.013	.053	.307	.757	.646	.915	.226	.001	.303	.024
SENSOR 2	.035	.052	.557	.138	.963	.055	.477	.368	.637	.040	.367	.012
A x M	.222	.010	.745	.789	.746	.887	.746	.855	.848	.509	.539	.066
A x SENSOR 2	.495	.414	.226	.738	.235	.605	.725	.595	.630	.731	.687	.980
M x SENSOR 2	.464	.207	.605	.900	.007	.372	.795	.236	.163	.006	.454	.936
A x M x SENSOR 2	.205	.839	.796	.082	.819	.741	.024	.405	.205	.346	.694	.134

Table 24 Interaction Analysis - Baseline COGNIT x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Levels Of Significance)

	Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.656	.447	.076	.076	.037	.008	.000	.201	.223	.011	.694	.067
M	.728	.551	.008	.111	.370	.811	.829	.707	.204	.007	.210	.007
COGNIT 2	.448	.858	.027	.191	.093	.018	.166	.000	.824	.341	.520	.820
A x M	.289	.009	.724	.919	.576	.872	.724	.484	.822	.454	.531	.054
A x COGNIT 2	.722	.761	.457	.245	.394	.897	.284	.458	.821	.608	.381	.161
M x COGNIT 2	.480	.881	.559	.607	.022	.688	.551	.167	.348	.406	.491	.077
A x M x COGNIT 2	.042	.286	.324	.124	.751	.361	.237	.120	.247	.189	.816	.052

Table 25 Interaction Analysis - Baseline BSTEER Score x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Significance Levles)

	Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.752	.545	.137	.068	.035	.010	.000	.196	.215	.009	.791	.083
M	.588	.464	.019	.047	.290	.927	.651	.983	.221	.009	.276	.024
BSTEER 2	.564	.097	.931	.039	.605	.597	.278	.164	.732	.581	.865	.063
A x M	.270	.012	.973	.782	.656	.639	.975	.919	.788	.630	.645	.130
A x BSTEER 2	.677	.755	.286	.865	.615	.978	.397	.943	.559	.505	.163	.889
M x BSTEER 2	.853	.479	.912	.053	.033	.603	.318	.088	.616	.322	.578	.760
A x M x BSTEER 2	.772	.652	.663	.139	.705	.729	.412	.021	.333	.269	.800	.451

Table 26 Interaction Analysis - Baseline BSPEED Score x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Levels Of Significance)

	Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	AMPH	SDOWN	ESTCHIC
A	.666	.550	.107	.075	.044	.008	.000	.243	.228	.011	.720	.061
M	.747	.731	.013	.120	.428	.906	.768	.534	.220	.009	.223	.006
BSPEED 2	.621	.161	.482	.535	.239	.603	.777	.001	.747	.683	.853	.253
A x M	.306	.010	.970	.814	.650	.795	.567	.976	.877	.639	.444	.073
A x BSPEED 2	.804	.581	.328	.832	.656	.816	.645	.284	.769	.295	.214	.527
M x BSPEED 2	.607	.452	.744	.216	.252	.762	.003	.921	.222	.582	.936	.205
A x M x BSPEED 2	.052	.571	.288	.277	.365	.651	.999	.406	.285	.874	.576	.233

Table 27 Interaction Analysis - Baseline ESTCHIC Score x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Levels Of Significance)

	Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.705	.450	.176	.019	.046	.005	.000	.097	.147	.006	.847	.202
M	.688	.560	.017	.067	.327	.176	.759	.979	.181	.006	.235	.004
ESTCHIC 2	.936	.894	.219	.004	.418	.906	.167	.129	.149	.243	.304	.000
A x M	.226	.012	.903	.955	.541	.733	.638	.944	.564	.625	.673	.202
A x ESTCHIC 2	.686	.741	.964	.917	.748	.048	.777	.319	.235	.528	.364	.643
M x ESTCHIC 2	.366	.522	.481	.142	.302	.990	.974	.171	.595	.750	.790	.810
A x M x ESTCHIC 2	.539	.695	.833	.392	.487	.468	.424	.423	.588	.484	.731	.985

Table 28 Interaction Analysis - Baseline STOUCH Score x Marijuana x Alcohol On 12 Post Treatment Measures (Cell Entries Represent Levels Of Significance)

	Post Treatment Performance Measure											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.733	.401	.144	.101	.047	.009	.000	.179	.259	.013	.757	.068
M	.731	.591	.012	.035	.189	.858	.621	.956	.147	.002	.215	.016
STOUCH 2	.755	.687	.470	.021	.094	.270	.364	.678	.324	.039	.780	.409
A x M	.417	.006	.958	.854	.500	.790	.697	.803	.738	.299	.517	.097
A x STOUCH 2	.385	.826	.551	.739	.864	.999	.751	.797	.493	.402	.888	.597
M x STOUCH 2	.169	.297	.199	.951	.297	.819	.391	.865	.549	.141	.475	.155
A x M x STOUCH 2	.287	.003	.821	.470	.259	.505	.757	.643	.598	.666	.189	.588

Table 29 Interaction Analysis - Baseline LINES Score x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Levels Of Significance)

	Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.687	.456	.131	.064	.036	.006	.000	.158	.234	.008	.747	.084
M	.723	.561	.019	.091	.409	.996	.727	.955	.231	.005	.235	.010
LINES 2	.667	.965	.831	.751	.109	.831	.866	.963	.539	.635	.598	.955
A x M	.392	.012	.891	.840	.427	.709	.621	.861	.841	.675	.487	.085
A x LINES 2	.084	.516	.916	.186	.753	.854	.195	.555	.710	.484	.276	.270
M x LINES 2	.666	.893	.328	.945	.054	.844	.387	.378	.562	.248	.780	.827
A x M x LINES 2	.811	.618	.958	.737	.864	.604	.661	.705	.801	.148	.013	.401

Table 30 Interaction Analysis - Baseline COORD Score x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Levels Of Significance)

	Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.706	.381	.105	.065	.032	.005	.000	.150	.177	.009	.744	.087
M	.681	.799	.028	.118	.264	.996	.484	.832	.280	.004	.202	.013
COORD 2	.956	.018	.115	.609	.444	.795	.016	.067	.093	.447	.641	.813
A x M	.301	.004	.927	.822	.552	.639	.878	.861	.850	.644	.377	.129
A x COORD 2	.793	.622	.366	.451	.444	.228	.073	.482	.390	.411	.082	.874
M x COORD 2	.448	.795	.752	.906	.453	.311	.123	.023	.177	.453	.622	.906
A x M x COORD 2	.110	.817	.437	.911	.048	.060	.598	.335	.320	.107	.492	.813

Table 31 Interaction Analysis - Baseline CTT Score x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Levels Of Significance)

	Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.921	.438	.299	.071	.032	.004	.001	.184	.246	.010	.650	.054
M	.847	.558	.007	.107	.336	.891	.727	.976	.195	.006	.230	.011
CTT 2	.046	.914	.036	.864	.772	.368	.000	.862	.872	.995	.650	.392
A x M	.032	.047	.558	.878	.483	.475	.549	.735	.967	.699	.150	.064
A x CTT 2	.054	.273	.787	.752	.517	.466	.558	.765	.942	.226	.460	.071
M x CTT 2	.159	.033	.324	.789	.510	.635	.289	.789	.318	.915	.007	.513
A x M x CTT 2	.318	.656	.413	.964	.118	.367	.715	.703	.501	.108	.590	.433

Table 32 Interaction Analysis - Baseline BISTOP Score x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries REpresent Levels Of Significance)

	Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.170	.947	.219	.020	.053	.006	.000	.115	.065	.019	.939	.158
M	.997	.768	.022	.051	.356	.998	.795	.974	.099	.009	.273	.014
BISTOP 2	.000	.002	.516	.040	.602	.785	.481	.417	.023	.353	.255	.204
A x M	.235	.008	.962	.563	.601	.897	.622	.689	.346	.534	.699	.090
A x BISTOP 2	.692	.250	.791	.257	.744	.840	.305	.360	.011	.872	.747	.381
M x BISTOP 2	.803	.938	.584	.083	.600	.353	.207	.890	.184	.751	.361	.737
A x M BISTOP 2	.388	.924	.384	.492	.918	.340	.391	.321	.096	.524	.251	.896

Table 33 Interaction Analysis - Baseline BISKILLS Score x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Levels Of Significance)

	Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.834	.341	.055	.115	.049	.008	.000	****	.258	.014	.721	.027
M	.691	.559	.014	.078	.311	.977	.739	****	.197	.004	.225	.008
BISKILLS 2	.370	.236	.034	.053	.353	.673	.512	****	.608	.132	.912	.010
A x M	.332	.006	.795	.960	.677	.924	.889	.***	.851	.442	.418	.085
A x BISKILLS 2	.719	.488	.951	.141	.332	.470	.599	.***	.707	.057	.924	.775
M x BISKILLS 2	.631	.438	.221	.895	.171	.179	.425	.***	.364	.191	.178	.290
A x M BISKILLS 2	.475	.494	.995	.081	.388	.323	.539	.***	.493	.389	.293	.785

Table 34 Interaction Analysis - Baseline SMPH Score x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Levels Of Significance)

	Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.688	.364	.119	.086	.045	.005	.000	.205	.154	.009	.994	.068
M	.703	.502	.015	.106	.349	.908	.726	.891	.158	.002	.264	.008
SMPH 2	.769	.205	.793	.215	.349	.172	.703	.170	.208	.000	.004	.470
A x M	.301	.008	.888	.774	.436	.998	.695	.590	.496	.123	.542	.076
A x SMPH 2	.915	.304	.311	.639	.269	.560	.678	.940	.265	.553	.388	.303
M x SMPH 2	.726	.657	.620	.573	.573	.125	.406	.299	.168	.014	.071	.617
A x M x SMPH 2	.543	.239	.347	.139	.893	.645	.731	.787	.153	.309	.999	.190

Table 35 Interaction Analysis - Baseline SDOWN Score x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Levels Of Significance)

	Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.707	.438	.125	.063	.031	.005	.000	.153	.217	.009	.732	.076
M	.732	.714	.016	.087	.343	.984	.710	.901	.265	.007	.213	.008
SDOWN 2	.729	.195	.799	.753	.799	.771	.859	.677	.391	.813	.778	.784
A x M	.337	.014	.796	.904	.668	.678	.871	.630	.805	.519	.712	.118
A x SDOWN 2	.386	.331	.392	.875	.140	.581	.349	.245	.559	.677	.269	.849
M x SDOWN 2	.985	.911	.626	.069	.422	.079	.201	.988	.949	.100	.170	.555
A x M x SDOWN 2	.106	.068	.436	.899	.264	.970	.014	.411	.733	.813	.816	.023

Table 36 Interaction Analysis - Baseline TIME Score x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Levels Of Significance)

	Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.662	.506	.172	.060	.020	.002	.000	.084	.229	.003	.674	.077
M	.652	.610	.018	.196	.411	.986	.642	.798	.163	.005	.268	.002
TIME 2	.465	.938	.586	.027	.055	.163	.773	.014	.374	.129	.312	.027
A x M	.276	.006	.630	.971	.575	.438	.770	.836	.757	.728	.554	.112
A x TIME 2	.490	.663	.050	.619	.246	.494	.675	.720	.565	.354	.527	.134
M x TIME 2	.560	.695	.608	.291	.254	.260	.254	.528	.508	.340	.053	.006
A x M x TIME 2	.588	.346	.096	.472	.327	.604	.045	.225	.576	.265	.896	.082

Table 39 Interaction Analysis - Driving Experience (DEXPER) x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Levels Of Significance)

	Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.545	.195	.066	.122	.052	.004	.000	.347	.175	.012	.834	.041
M	.788	.318	.112	.139	.475	.982	.961	.400	.146	.010	.096	.003
DEXPER	.623	.376	.334	.762	.548	.020	.009	.248	.406	.631	.888	.108
A x M	.160	.001	.771	.782	.766	.504	.774	.740	.644	.884	.410	.101
A x DEXPER	.504	.833	.735	.646	.718	.255	.038	.975	.341	.108	.394	.33
M x DEXPER	.132	.288	.655	.790	.385	.057	.965	.440	.448	.344	.655	.233
A x M x DEXPER	.933	.855	.339	.868	.392	.900	.475	.763	.301	.589	.164	.267

Table 40 Interaction Analysis - Weekly Alcohol Use (AWEEK) x Marijana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Levels Of Significance)

	Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.635	.189	.049	.162	.030	.013	.000	.386	.171	.009	.786	.043
M	.725	.323	.085	.162	.386	.795	.785	.387	.149	.009	.085	.004
AWEEK	.593	.693	.993	.262	.322	.853	.676	.094	.527	.188	.563	.180
A x M	.204	.001	.765	.807	.773	.592	.680	.519	.545	.585	.571	.081
A x AWEEK	.969	.682	.524	.280	.422	.303	.054	.884	.461	.401	.617	.226
M x AWEEK	.862	.095	.065	.963	.267	.503	.714	.712	.172	.279	.150	.540
A x M x AWEEK	.690	.009	.277	.569	.107	.628	.812	.186	.105	.969	.130	.503

Table 41 Interaction Analysis - Alcohol Driving Experience (ADRIVE) x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Levels Of Significane)

	Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.555	.222	.050	.119	.039	.011	.000	.282	.190	.016	.853	.059
M	.888	.564	.085	.214	.342	.857	.674	.534	.125	.013	.110	.005
ADRIVE	.179	.003	.792	.029	.189	.624	.360	.466	.260	.458	.505	.655
A x M	.281	.000	.757	.957	.619	.580	.921	.636	.546	.712	.713	.150
A x ADRIVE	.392	.234	.155	.353	.570	.788	.324	.906	.307	.881	.029	.246
M x ADRIVE	.555	.695	.378	.124	.251	.809	.330	.859	.474	.906	.714	.324
A x M x ADRIVE	.378	.718	.628	.547	.716	.325	.232	.509	.761	.969	.904	.191

Table 37 Interaction Analysis - Baseline EYEHAND Score x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Levels Of Significance)

	Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.716	.452	.123	.067	.029	.005	.000	.150	.218	.006	.730	.071
M	.453	.512	.029	.095	.449	.859	.817	.877	.253	.006	.229	.004
EYEHAND 2	.012	.542	.046	.830	.042	.107	.427	.426	.272	.204	.888	.064
A x M	.154	.011	.961	.963	.382	.658	.719	.606	.836	.338	.507	.124
A x EYEHAND 2	.197	.934	.847	.813	.599	.756	.910	.130	.561	.021	.568	.161
M x EYEHAND 2	.717	.869	.695	.507	.609	.539	.163	.565	.476	.077	.062	.806
A x M x EYEHAND 2	.978	.411	.691	.371	.127	.551	.124	.416	.628	.184	.432	.408

Table 38 Interaction Analysis - Baseline BITE Score x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Levels Of Significance)

	Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.640	.439	.100	.074	.040	.006	.000	.153	.227	.008	.684	.094
M	.694	.549	.015	.091	.311	.973	.744	.958	.198	.006	.200	.010
BITE 2	.168	.890	.123	.151	.018	.737	.602	.967	.653	.589	.478	.484
A x M	.317	.009	.783	.769	.373	.716	.779	.757	.798	.650	.473	.114
A x BITE 2	.515	.464	.832	.173	.951	.408	.793	.487	.478	.208	.007	.780
M x BITE 2	.802	.234	.845	.277	.285	.262	.186	.800	.570	.162	.503	.331
A x M x BITE 2	.483	.111	.786	.966	.358	.608	.560	.142	.283	.995	.262	.597

Table 42 Interaction Analysis - Weekly Marijuana Use (MWEEK) x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Levels Of Significance)

	Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.580	.184	.042	.134	.043	.014	.000	.291	.162	.016	.897	.075
M	.736	.329	.082	.145	.435	.776	.751	.471	.153	.013	.099	.007
MWEEK	.780	.390	.381	.630	.663	.151	.377	.960	.159	.992	.375	.669
A x M	.156	.001	.586	.738	.593	.523	.813	.607	.645	.736	.531	.126
A x MWEEK	.101	.026	.864	.536	.614	.347	.962	.550	.111	.617	.095	.969
M x AWEEK	.097	.267	.327	.962	.590	.264	.516	.872	.716	.528	.793	.837
A x M x AWEEK	.103	.104	.090	.331	.798	.872	.017	.699	.650	.427	.870	.897

Table 43 Interaction Analysis - Marijuana Driving Experience (MDRIVE) x Marijuana x Alcohol On 12 Post Treatment Performance Measures (Cell Entries Represent Levels Of Significance)

	Performance Measures											
Variation Source	STOPS	POSTOP	COGNIT	ATTEMPTS	BSPEED	STOUCH	CTT	BISKILLS	UFAILED	SMPH	SDOWN	ESTCHIC
A	.509	.291	.026	.120	.033	.014	.000	.301	.165	.017	.800	.052
M	.628	.249	.105	.150	.570	.892	.803	.495	.236	.009	.118	.009
MDRIVE	.230	.071	.123	.860	.134	.425	.721	.797	.169	.403	.498	.235
A x M	.162	.003	.251	.925	.541	.602	.661	.565	.928	.997	.367	.102
A x MDRIVE	.479	.908	.001	.311	.900	.436	.234	.910	.203	.727	.072	.521
M x MDRIVE	.140	.970	.367	.257	.842	.893	.519	.396	.977	.142	.792	.252
A x M x MDRIVE	.938	.818	.239	.988	.879	.750	.393	.590	.858	.595	.855	.574

Table 44 Average Blood Levels Of Drugs For Each Post-Treatment Run *

RUN	DRUG	UNITS	CONDITION			
			Placebo	Marijuana	Alcohol	Both
3	Alcohol	% BAC	0.00	0.00	0.07	0.05
	Serum THC (S-1)	ng/ml	0.80	69.6	3.0	54.3
	Serum Carboxy (S-2)	ng/ml	NA	46.1	NA	73.6
4	Alcohol	% BAC	0.00	0.00	0.08	0.07
	Serum THC (S-1)	ng/ml	0.41	13.1	1.7	13.4
	Serum Carboxy (S-2)	ng/ml	NA	36.0	NA	30.8
5	Alcohol	% BAC	0.00	0.00	0.06	0.06
	Serum THC (S-1)	ng/ml	0.00	7.4	1.8	7.6
	Serum Carboxy (S-2)	ng/ml	NA	31.5	NA	25.9
6	Alcohol	% BAC	0.00	0.00	0.04	0.04
	Serum THC (S-1)	ng/ml	0.00	4.8	2.2	4.9
	Serum Carboxy (S-2)	ng/ml	NA	27.3	NA	23.0

NA = No analysis
* See Appendix 1, Table A for individual subject results.

Table 45 Percent Of Variance Accounted For By Blood And Serum Levels

Run	Source of Variation	Marijuana Effect	Alcohol Effect	Interaction Effect	Total
3	Treatment Group	29	30	12	71
	Serum THC and Blood Alcohol	09	39	13	61
	Serum Carboxy and Blood Alcohol	12	39	09	60
	Both Serum Levels and Blood Alcohol	23	39	03	65
4	Treatment	24	31	07	62
	Serum THC Only and Blood Alcohol	19	41	05	62
	Serum Carboxy Only and Blood Alcohol	22	41	07	70
	Both Serum Levels and Blood Alcohol	30	41	01	72
5	Treatment Group	23	37	4	64
	Serum THC Only and Blood Alcohol	14	44	3	61
	Serum Carboxy Only and Blood Alcohol	19	44	2	65
	Both Serum Levels and Blood Alcohol	26	44	2	72
6	Treatment Group	29	28	13	70
	Serum THC Only and Blood Alcohol	22	34	1	57
	Serum Carboxy Only and Blood Alcohol	18	34	8	60
	Both Serums and Blood Alcohol	34	34	1	69

Table 46 Canonical Correlation Functions For 12 Performance Measures With 3 Blood Level Measures Including Baseline

Canonical Function	Canonical Correlation	Percent of Variance (1-Wilks Lambda)	Significance
1	.84	.88	.000*
2	.64	.61	.000*
3	.50	.35	.004*
4	.36	.13	.105
5	.03	.00	.842

* Canonical variates used in the analysis.

Table 47 Canonical Variate Weights For Performance And Blood Level Measures Across All Runs Including Baseline

Variable Set	Canonical Variate		
Blood Levels	1	2	3
Run 2 (Baseline)	-0.07974	-0.05441	0.91499
Run 3	-0.46319	-0.07333	-0.37507
Run 4	-0.52873	-1.03065	0.50235
Run 5	0.25615	-1.25032	-0.36365
Run 6	0.17398	-0.11644	-0.71117
Performance Measures			
Run 2 (Baseline)	0.04569	-0.15488	0.98776
Run 3	-0.42309	0.45231	-0.37529
Run 4	-0.07415	-1.12710	-0.12963
Run 5	0.43325	-1.01291	-0.38507
Run 6	-0.39157	-0.20942	-0.13509

Table 48 Canonical Correlation Functions For 12 Performance Measures With 3 Blood Level Measures - Treatment Runs Only

Canonical Function	Canonical Correlation	Percent of Variance (1-Wilks Lambda)	Significance
1	.83	.84	.000*
2	.64	.48	.000*
3	.37	.13	.080
4	.01	.00	.913

* Canonical variates used in the analysis.

Table 49 Canonical Variate Weights For Performance And Blood Level Measures Across Treatment Runs

Variable Set	Canonical Variate	
Blood Levels	1	2
Run 3	0.38488	0.12885
Run 4	0.56728	0.94182
Run 5	-0.27972	1.29562
Run 6	-0.13785	0.19443
Performance Measures		
Run 3	0.40824	-0.42152
Run 4	0.06961	1.16235
Run 5	-0.42622	1.05454
Run 6	0.40491	0.20756

Table 50 Correlations Between the CHP Car Following and Field Sobriety Test Variables and the Two Significant Discriminant Functions of Post Treatment Performance

	Functions	
Variable	1	2
Following Car	-.27**	.12
Romberg Test	.03	-.12
Finger to Nose	-.07	-.01
Heel to Toe	-.22*	-.01
Right Foot Balance	-.11	.03
Left foot Balance	.02	-.03
Finger Count	-.30**	.14
Hand Count	-.18	.01
Counting Backwards	-.26**	.01
Alphabet	.05	-.01
Officer's Rating	-.26**	.19*
Subject's Rating	-.40***	.08
FST Total	-.18	.01

* $p < .05$
** $p < .01$
*** $p < .001$

Table 51 Correlations For Subject Impairment As Rated By Following CHP And FST Officers With 27 Experimental Factors On Run 3

Factors	Following Car	FST Officer Rating
ACCELERATOR	-.03	.20*
BRAKING	.12	.11
STEERING	.01	.16
LINES	.10	.05
TIME	.05	-.07
STOPS	.03	.30*
ERRORS	.24*	.41*
RISK	.06	.25*
COORDINTATION	.08	.69*
COGNITION	.07	.35*
EYEHAND	.06	.34*
STOUCH	-.01	.06
SDOWN	.05	-.33*
ATTEMPTS	-.13	-.01
BSTEER	.15	.31*
BSPEED	.04	-.12
BLANE	.29*	.40*
BISTOP	.01	.01
BISKILLS	.14	.01
UFAILED	.14	-.07
POSTOP	.16	.04
ESTCHIC	-.15	-.17
FLREMIND	.14	.04
FLRESPONSE	.25*	-.10
FLRERUN	.05	.08
SMPH	.19*	.10
OVERALL	.22*	.29*

* $p < .05$

Table 52 Standardized Means For CHP Following Car And Field Sobriety Test Measures By Treatment For Run 3

Variable	Placebo	Marijuana	Alcohol	Both
Following Car	1.21	1.59	1.42	1.59
Romberg Test	1.47	1.29	1.32	1.23
Finger to Nose	1.42	1.65	2.16	1.73
Heel to Toe	1.26	1.76	1.58	2.00
Right Foot Balance	1.32	1.35	1.74	1.95
Left Foot Balance	1.37	1.53	1.74	1.64
Finger Count	1.21	1.59	1.58	1.95
Hand Count	1.16	1.53	1.42	1.45
Count Backward	1.16	1.18	1.26	1.77
Alphabet	1.00	1.00	1.32	1.14
Officer's Rating	1.63	2.18	2.84	2.95
Subject's Rating	2.26	3.41	3.95	4.77

Table 53 Standardized Means For CHP Following Car And Field Sobriety Test Measures By Treatment For Run 4

Variable	Placebo	Marijuana	Alcohol	Both
Following Car	1.15	1.29	1.56	1.57
Romberg Test	1.30	1.12	1.28	1.57
Finger to Nose	1.40	1.59	1.56	1.62
Heel to Toe	1.50	1.24	1.50	1.90
Right Foot Balance	1.30	1.47	1.56	1.81
Left Foot Balance	1.15	1.59	1.44	1.71
Finger Count	1.00	1.35	1.33	1.43
Hand Count	1.30	1.12	1.44	1.71
Count Backward	1.00	1.12	1.50	1.33
Alphabet	1.15	1.18	1.11	1.29
Officer's Rating	1.35	1.82	2.11	2.81
Subject's Rating	1.70	2.47	2.39	4.27

Table 54 Standardized Means For CHP Following Car And Field Sobriety Test Measures By Treatment For Run 5

Variable	Placebo	Marijuana	Alcohol	Both
Following Car	1.11	1.17	1.21	1.52
Romberg Test	1.16	1.17	1.21	1.14
Finger to Nose	1.11	1.13	1.11	1.29
Heel to Toe	1.37	1.33	1.21	1.57
Right Foot Balance	1.32	1.67	1.37	1.52
Left Foot Balance	1.26	1.56	1.37	1.24
Finger Count	1.00	1.17	1.16	1.24
Hand Count	1.05	1.44	1.16	1.05
Alphabet	1.00	1.00	1.21	1.10
Officer's Rating	0.89	1.22	.84	1.29
Subject's Rating	1.05	1.05	1.42	3.19

Table 55 Standardized Means For CHP Following Car And Field Sobriety Test Measures By Treatment For Run 6

Variable	Placebo	Marijuana	Alcohol	Both
Following Car	1.10	1.21	1.24	1.35
Romberg Test	1.05	1.00	1.00	1.00
Finger to Nose	1.25	1.21	1.12	1.10
Heel to Toe	1.20	1.29	1.24	1.35
Right Foot Balance	1.05	1.21	1.35	1.20
Left Foot Balance	1.30	1.21	1.29	1.00
Finger Count	1.05	1.07	1.12	1.35
Hand Count	1.05	1.07	1.06	1.15
Count Backward	1.20	1.29	1.00	1.35
Alphabet	1.15	1.00	1.00	1.05
Officer's Rating	0.45	0.57	0.47	0.90
Subject's Rating	0.45	0.57	0.64	2.20

Table 56 Impairment Questionaire Items (Runs 3 & 5) Correlated With The Two Significant Discriminant Functions

Questionaire Items	Run 3 Function 1	Run 3 Function 2	Run 5 Function 1	Run 5 Function 2
Visual Abilities	-0.3086 P=0.002	0.1510 P=0.088	-0.2415 P=0.014	0.1723 P=0.061
Auditory Abilities	-0.2341 P=0.017	0.1072 P=0.169	-0.2476 P=0.012	0.0884 P=0.215
Spatial Abilities	-0.2714 P=0.007	0.1785 P=0.054	-0.3038 P=0.003	0.0516 P=0.323
Memory Abilities	-0.3713 P=0.000	0.1397 P=0.105	-0.3193 P=0.002	0.0130 P=0.454
Self Control	-0.3134 P=0.002	0.2948 P=0.004	-0.1353 P=0.113	0.1100 P=0.163
Coordination	-0.3015 P=0.003	0.1622 P=0.073	-0.2435 P=0.014	0.1349 P=0.113
Reaction Time	-0.3378 P=0.001	0.0626 P=0.288	-0.2827 P=0.005	0.1327 P=0.117
Speed Estimation	-0.2661 P=0.008	0.2339 P=0.017	-0.3903 P=0.000	0.0961 P=0.049
Time Estimation	-0.4294 P=0.000	0.1879 P=0.045	-0.4226 P=0.000	0.1839 P=0.049
Distance Estimation	-0.2994 P=0.003	0.0944 P=0.199	-0.2812 P=0.005	0.1013 P=0.183

Table 57 Correlations* For Tart Impairment Questionaire With 27 Experimental Factors On Run 3

	Test Items									
Factors	1	2	3	4	5	6	7	8	9	10
ACCELERATOR	-.05	-.16	-.22	.00	-.03	.02	-.06	-.01	-.05	-.13
BRAKING	.00	-.10	-.05	.05	.07	.06	.07	.17	.12	-.04
STEERING	-.04	-.18	-.22	-.07	.00	.06	-.05	-.03	-.07	-.18
LINES	-.02	-.02	-.06	-.03	-.05	-.08	-.10	-.08	-.16	.02
TIME	.07	-.09	.14	-.02	-.12	-.05	.04	.04	.10	.00
STOPS	.13	-.02	.01	-.08	.21	.09	-.01	.01	.04	-.08
ERRORS	.21	-.14	.16	.19	.25	.29	.16	.15	.18	.11
RISK	.21	.02	.15	.18	.30	.15	.27	.07	.04	.33
COORDINATION	.11	.10	.11	.16	.30	.28	.16	.08	.16	.17
COGNITION	.21	.28	.23	.20	.10	.24	.17	.19	.25	.22
EYEHAND	-.04	.01	-.08	.02	.02	.05	-.02	-.02	-.05	.04
STOUCH	.32	.15	.11	-.01	.09	.23	.17	.15	.04	.11
SDOWN	.02	-.05	-.16	-.19	-.16	-.23	-.12	-.24	-.24	-.14
ATTEMPTS	.03	.14	.01	.16	.04	.09	.10	.16	.15	.05
BSTEER	.26	-.13	.30	.24	.32	.30	.26	.23	.26	.21
BSPEED	.02	.04	.17	.20	.12	.10	.19	.24	.28	.11
BLANE	.21	.02	.17	.18	.20	.28	.17	.13	.17	.13
BISTOP	-.20	-.07	-.15	.00	-.14	-.15	-.11	-.02	-.11	.02
BISKILLS	-.06	-.01	.08	-.03	.07	-.08	.00	.00	.05	-.13
UFAILED	.02	.04	.02	-.03	.04	.01	-.02	.04	.04	-.02
POSTOP	.13	.12	.19	.14	.14	.16	.16	.23	.17	.21
ESTCHIC	-.31	-.08	-.23	-.22	-.07	-.43	-.43	-.11	-.23	-.23
FLREMIND	-.14	.04	-.21	-.04	-.10	.00	-.09	-.08	.02	-.04
FLRESPONSE	.08	.02	.02	-.08	-.07	-.01	.03	.01	-.10	.08
FLRERUN	.29	.27	.27	.20	.21	.32	.23	.17	.15	.25
SMPH	.24	.19	.19	.33	.24	.21	.31	.30	.38	.22
OVERALL	.31	.39	.39	.31	.41	.35	.29	.27	.22	.25

* Correlations above .19 significant at $p < .05$.

Table 58 Correlation* For Tart Impairment Questionaire With 27 Experimental Factors On Run 5

	Test Items									
Factor	1	2	3	4	5	6	7	8	9	10
ACCELERATOR	.23	.15	.27	.20	.25	.16	.22	.16	.16	.19
BRAKING	.22	.11	.20	.16	.22	.26	.21	.22	.23	.22
STEERING	.09	.08	.03	.14	.07	.17	.13	.07	.06	.04
LINES	-.10	-.13	-.17	.01	-.11	-.02	-.14	.05	.01	-.06
TIME	-.28	-.28	-.03	-.22	-.16	-.28	-.12	-.25	-.23	-.29
STOPS	.00	-.07	-.00	.06	.23	-.23	.00	-.01	.00	-.03
ERRORS	.13	.03	.29	.13	.25	.23	.22	.25	.30	.40
RISK	-.02	.07	.05	.16	-.13	-.07	-.01	.08	.03	-.02
COORDINATION	-.06	.17	.04	-.02	.24	.14	.07	.05	.04	.12
COGNITION	-.10	.05	.07	.01	.16	.17	.08	.11	.14	.14
EYEHAND	-.12	-.05	-.02	-.08	-.07	-.11	-.06	-.07	-.01	-.01
STOUCH	-.03	.05	-.09	.01	.02	.06	.01	-.03	.02	.11
SDOWN	.03	-.02	.02	-.02	.19	.09	.08	-.04	-.02	.04
ATTEMPTS	.05	.03	.01	-.06	.04	.11	.14	.11	.09	.10
BSTEER	-.01	.03	.14	.20	.11	.18	.19	.29	.19	.22
BSPEED	.01	-.08	.11	.03	-.03	.00	-.03	-.07	.17	.03
BLANE	-.04	.03	.05	.04	.09	.13	.07	.19	.14	.16
BISTOP	-.05	.06	-.07	.13	-.12	-.10	-.04	-.06	-.08	-.14
BISKILLS	.06	-.03	.13	.15	.05	.09	.10	.11	.11	.09
UFAILED	.24	.34	.00	.33	.28	.43	.20	.24	.27	.38
POSTOP	.21	.15	.00	.11	.11	.17	.20	.18	.15	.13
ESTCHIC	-.23	-.22	-.23	-.25	-.10	-.15	-.11	-.20	-.22	-.16
FLREMIND	-.05	-.10	-.11	-.15	-.32	-.20	-.16	-.13	-.13	-.17
FLRESPONSE	.06	.04	.02	.02	.00	-.03	.04	.02	.06	-.03
FLRERUN	.06	.13	.15	.12	.31	.19	.16	.12	.12	.20
SMPH	.23	.00	.24	.19	.10	.18	.17	.28	.32	.21
OVERALL	.19	.08	.33	.14	.23	.29	.24	.30	.30	.38

* Correlations above .19 significant at $p < .05$.

Table 59 Glossary Description Of Exit Questionaire Items As Entered In Analyses

Item Label	Description
1. Drug Received	Drug believed to have been received.
2. Produced High	Rate of "High" produced.
3. Strategy Chicane	During which run was strategy for Chicane learned.
4. Practice Chicane	Did practice aid in learning Chicane?
5. Drug Chicane	Did drugs interfer with performance?
6. Strategy FLC	During which run was strategy for Forced Lane Change learned.
7. Practice FLC	Did practice aid in learning Forced Lane Change?
8. Drug FLC	Did drugs interfer with performance.
9. Strategy Urban	During which run was strategy for the Urban Drive learned.
10. Practice Urban	Did practice aid in learning the Urban Drive?
11. Drug Urban	Did drugs interfer with performance?
12. Drug Extended	Did drugs interfer with performance on the Extended Drive?
13. Strategy Risk	During which run was strategy for the Risk task learned.
14. Practice Risk	Did practice aid in learning the Risk task?
15. Drug Risk	Did drugs interfer with performance on the Risk task?
16. Feedback	Did receiving feedback about performance help overall.
17. Feed Chicane	Did receiving feedback about performance help on the Chicane?
18. Feed FLC	Did receiving feedback about performance help on the Forced Lane Change?
19. Feed Urban	Did recieving feedback about performance help on the Urban drive routes?
20. Feed Extended	Did recieving feedback about performance help on the Extended drive?
21. Feed Risk	Did recieving feedback about performance help on the Risk task?
22. Compensation	Was compensation for effects of alcohol or marijuana necessary?
23. Driving Frequency	Frequency with which driving normally occured under similar impairment.
24. Similar high	Was the "high" under driving similar to state subject normally drove after drug consumption?
25. Real Conditions	Were drug-alcohol treatments representative of actual conditions under which people drive under the influence of the drugs.

Table 60 Correlations Of Exit Questionaire Items With The Two Discriminant Functions

Item Label	Function 1	Function 2
Drug Received	0.1270	0.0570
	P=0.137	P=0.312
Produced High	0.4492	0.2105
	P=0.000	P=0.034
Strategy Chicane	-0.1297	-0.0030
	P=0.132	P=0.490
Practice Chicane	0.0605	0.0590
	P=0.302	P=0.306
Drug Chicane	-0.3231	-0.0633
	P=0.002	P=0.293
Strategy FLC	-0.1087	0.1794
	P=0.175	P=0.060
Practice FLC	-0.1178	0.0128
	P=0.155	P=0.456
Drug FLC	-0.0109	-0.0348
	P=0.463	P=0.383
Strategy Urban	-0.0216	0.1345
	P=0.427	P=0.123
Practice Urban	-0.0400	-0.0545
	P=0.366	P=0.320
Drug Urban	-0.4127	-0.0455
	P=0.000	P=0.348
Drug Extended	-0.3330	0.0058
	P=0.002	P=0.480
Strategy Risk	-0.1002	-0.0801
	P=0.195	P=0.246
Practice Risk	-0.2397	0.0340
	P=0.019	P=0.385
Drug Risk	-0.1916	-0.0206
	P=0.049	P=0.430
Feedback	-0.0919	0.0624
	P=0.215	P=0.296
Feedback-Chicane	-0.0762	-0.0748
	P=0.257	P=0.260
Feedback FLC	-0.1255	-0.0508
	P=0.140	P=0.332
Feedback Urban	-0.1830	-0.0242
	P=0.057	P=0.418
Feedback Extended	0.0873	0.0821
	P=0.227	P=0.240
Feedback Risk	-0.4052	-0.0746
	P=0.000	P=0.261
Compensation	-0.1388	-0.2150
	P=0.116	P=0.031
Driving Frequency	-0.2247	-0.2381
	P=0.025	P=0.019
Similar high	0.3145	0.0142
	P=0.003	P=0.452
Real Conditions	-0.2123	-0.-114
	P=0.033	P=0.461

Table 61 Subject Background Variables Correlated With Two Discriminant Functions From Final Variable Set

Background Variable	Function I	Function II
Number of Years Driving	0.02 p=0.426	0.02 p=0.424
Average Weekly Intake of Alcohol	-0.04 p=0.370	0.01 p=0.467
Average Experience Driving Under the Influence of Alcohol	-0.16 p=0.089	-0.01 p=0.480
Average Weekly Intake of Marijuana	-0.06 p=0.303	0.05 p=0.344
Average Experience Driving Under the Influence of Marijuana	-0.14 p=0.109	-0.01 p=0.450

Table 62 Correlations Of BITE And CTT Measures With The Two Significant Discriminant Functions (Run 3 - 6, combined)

Variable	Function 1	Function 2
BITE - Run 1	-.03	-.05
BITE - Run 2	.04	.01
BITE - Run 3	.14	-.27**
BITE - Run 4	.31**	-.14
BITE - Run 5	.30**	-.13
BITE - Run 6	.13	-.16
BITE - Composite	.25*	-.20*
CTT - Run 1	.07	-.16
CTT - Run 2	.07	-.10
CTT - Run 3	.09	-.01
CTT - Run 4	.22*	-.23*
CTT - Run 5	.13	-.23*
CTT - Run 6	.17	-.28**
CTT - Composite	.17	-.19*

* $p < .05$
** $p < .01$

Table 63 Treatment Means For BITE And CTT Measures

BITE

Run Number	Placebo	Marijuana	Alcohol	Both
Run 3	60.63	53.82	56.16	52.55
Run 4	62.52	56.47	58.95	53.43
Run 5	58.32	57.58	62.30	49.90
Run 6	57.45	53.93	52.33	57.40
Total	59.89	55.20	57.55	52.89

CTT

Run Number	Placebo	Marijuana	Alcohol	Both
Run 3	5.03	4.93	4.34	4.47
Run 4	4.89	4.94	4.21	4.16
Run 5	4.93	5.17	4.48	4.45
Run 6	5.04	5.20	4.63	4.67
Total	4.98	5.01	4.49	4.45

Table 64 Correlations Between BITE And CTT and 27 Performance Factors On Run 3 & 5

	Run 3		Run 5	
Factors	BITE	CTT	BITE	CTT
ACCELERATOR	-.07	.06	.00	-.10
BRAKING	-.17	-.00	-.03	-.12
STEERING	.10	.02	.08	-.20*
LINES	.08	-.08	-.06	.07
TIME	.00	.21*	.05	.17
STOPS	-.18*	.24*	-.08	-.11
ERRORS	-.32*	-.16	-.25*	-.11
RISK	-.15	-.23*	-.18*	.02
COORDINATION	-.21*	-.02	-.20*	-.09
COGNITION	-.21*	-.09	-.18*	-.04
EYEHAND	.03	-.17	-.01	.08
STOUCH	-.14	.03	.05	-.11
SDOWN	.14	.08	.16	-.06
ATTEMPTS	.14	.05	.18*	-.16
BSTEER	-.36*	-.04	-.09	-.21*
BSPEED	.15	-.00	-.04	-.06
BLANE	-.36*	-.28*	-.12	-.29*
BISTOP	-.01	-.51*	-.00	-.07
BISKILLS	-.05	-.07	.15	-.06
UFAILED	-.10	-.09	-.12	-.08
POSTOP	-.10	.12	.04	-.01
ESTCHIC	.18*	.18*	.11	.14
FLREMIND	.02	.04	.03	.23*
FLRESPONSE	-.01	-.07	.16	.04
FLRERUN	-.04	-.23*	-.17	-.12
SMPH	-.07	.10	-.16	.01
OVERALL	-.31*	-.13	-.29*	-.16

* $p < .05$

Table 65 Correlations Of Forced Lane Change Measures With The Two Significant Discriminant Functions

Variable	Function 1	Function 2
Forced Lane Change Composite	.06	.22*
Reminded of Speed	-.15	.09
Response	-.07	-.00
Rerun	-.05	-.05
Bipolar Composite	.02	.12

* $p < .05$

Table 66 Treatment Means For Forced Lane Measures Across Runs

Run	Placebo	Marijuana	Alcohol	Both Marijuana & Alcohol
A	Forced Lane Change Composite Factor			
3	0.95	0.71	1.16	1.05
4	0.65	0.64	1.11	0.71
5	0.68	0.44	1.05	0.61
6	0.90	0.64	0.71	0.75
B	Forced Lane Change Reminded Of Speed			
3	2.00	2.00	1.95	1.95
4	1.85	2.00	1.83	1.90
5	1.79	2.06	1.95	1.95
6	2.00	2.00	1.88	2.05
C	Forced Lane Change Quality Of Response			
3	1.00	1.18	1.21	1.09
4	1.10	1.05	1.00	1.19
5	0.95	1.06	1.11	1.00
6	1.00	1.07	1.18	1.05
D	Forced Lane Change Rerun			
3	0.21	0.35	0.37	0.77
4	0.20	0.12	0.44	0.48
5	0.32	0.11	0.42	0.10
6	0.40	0.14	0.00	0.20
E	Bipolar Forced Lane Change			
3	0.53	0.12	0.21	0.41
4	0.45	0.06	0.11	0.05
5	-0.26	0.00	0.11	0.43
6	0.10	0.07	0.71	0.25

Table 67 Correlations For Risk Task Measures With 2 Significant Discriminant Functions

Variable	Function 1	Function 2
Risk Choice	.25*	-.00
Risk Quality	-.13	.28**
Risk Attempts	-.30**	.08
Risk Composite	.01	.22*

* $p < .05$
** $p < .01$

Table 68 Treatment Means For Risk Task Measures Across Runs

Run	Placebo	Marijuana	Alcohol	Both Marijuana & Alcohol
A	Risk Choice			
3	4.58	4.35	4.42	4.27
4	4.50	4.29	4.72	4.24
5	4.32	4.50	4.05	4.10
6	5.00	3.79	4.24	3.80
B	Risk Quality			
3	0.32	1.00	1.42	1.14
4	0.80	1.12	1.50	1.05
5	0.84	0.83	0.84	1.00
6	0.95	0.43	0.65	0.55
C	Risk Attempts			
3	1.37	1.52	1.37	1.85
4	1.30	1.65	1.28	1.76
5	1.63	1.50	1.95	1.95
6	1.25	2.02	1.76	2.15
D	Risk Composite			
3	4.89	5.35	5.84	5.41
4	5.30	5.41	6.22	5.29
5	5.16	5.33	4.89	5.10
6	5.95	4.21	4.88	4.35

APPENDIX I, TABLE A

TABLE OF ALL ^{125}I RIA ANALYSES PERFORMED ON EACH SUBJECT'S SERUM AND URINE SAMPLES WITH CONCURRENT PERCENT BAC, IN ORDER OF DRIVE DATE

GLOSSARY FOR TABLE HEADINGS (1) TO (8); AND ABBREVIATIONS USED

(1) DRIVE DATE: The day and date of Driving Impairment Study.

(2) SUBJECT: The code used to identify each test subject on each drive day.

(3) TREATMENT: The randomly assigned treatment condition administered between baseline drive (#2) and first post treatment drive (#3).

P/P = double placebo
P/A = Placebo marijuana/active alcohol
M/P = active marijuana/placebo alcohol
M/A = active marijuana/active alcohol

(4) TYPE ANAL: Only the serum samples results were used for correlation with performance measures. The types of analyses applied to the subject serum samples and abbreviations used:

S-1 = ^{125}I RIA for Δ^9-THC
S-2 = ^{125}I RIA 9=COOH-THC
BAC = Breath alcohol Concentration determined. with Intoxilyzer Model 4011AW.
NA = Not analyzed, or analysis was in error.
NS = No sample obtained, or available for analysis.

NOTE: The ^{125}I RIA kits were made available for research purposes by NIDA through RESEARCH TRIANGLE INSTITUTE, RESEARCH TRIANGLE, N.C.

(5) RESULTS: Serum Samples: in ng/ml and % BAC by Drive #. RIA results < 2 ng/ml are reports as negative (0). Percent BAC results are rounded to two significant figures by truncating the third figure.

(6) URINE SCREEN, OTHER DRUGS: PRE and POST treatment urine samples were collected from subjects prior to receiving their prescribed treatment and after the last driving session. These samples were screened for the following drugs using ROCHE DIAGNOSTICS ^{125}I RIA Kits (ABBREVIATIONS USED):

AMPH = AMPHETAMINE/METHAMPHETAMINE
BARB = BARBITURATES
MORP = MORPHINE/CODEINE
COCA = COCAINE/METABOLITE
PCP = PHENCYCLIDINE
METHQ = METHAQUALONE
DIAZP* = DIAZEPINES
CANNAB* = CANNABINODS

(+) = POSITIVE within the established sensitivity and calibration limits of the particular RIA kit applied.
(-) = NEGATIVE no reaction with sensitivity and calibration limits.
(*Experimental, not commercially available when applied)

(7) REMARKS: as noted.

(8) SUBJECT DATA USED OR REJECTED:
U = Subjects and data used for correlation with performance measures analyzed.
U* = Subject "THC" results used for follow-up study of basal levels of cannabinoids in plasma of chronic marijuana users.
R = Subjects rejected for correlation with performance measures.

All the data collected for the 6/27, 28/81 drive dates was considered a "pilot" and not used for correlation with performance measures.

APPENDIX I TABLE A

(1) Drive Day & Date	(2) Subj.	(3) Treat-ment	(4) Type of Anal.	(5) Results: Serum Samples in ng/ml & % BAC, By Drive #					(6) Urine Screen other Drugs		(7) Remarks	(8) Subj. Data Used or Reject.
				2	3	4	5	6	Pre	Post		
SAT. JUN. 27, 1981	A	P/A	S-1 S-2 BAC	Ø NA .00	Ø NA .06	2.7 NA .06	Ø NA .05	Ø NA .03	(-)	(-)		R
	B	P/P	S-1 S-2 BAC	Ø NA .00	2.9 NA .00	3.1 NA .00	3.9 NA .00	6.7 NA .00	(-)	(+) Cannab	Indicates "THC" Use After Driv. #2	R
	C	M/A	S-1 S-2 BAC	Ø NA .00	62 NA .08	11 NA .06	6.4 NA .03	3.3 NA .01	(-)	(+) Cannab		R
	D	M/P	S-1 S-2 BAC	3 NA .00	97 NA .00	18 NA .00	11 NA .00	2.4 NA .00	(-)	(+) Cannab		R
	E	P/A	S-1 S-2 BAC	6.3 NA .00	2.3 NA .04	2.9 NA .04	3.4 NA .02	3.7 NA .00	(+) Cannab	(+) Cannab		R
	F	P/A	S-1 S-2 BAC	Ø NA .00	Ø NA .06	Ø NA .07	Ø NA .05	Ø NA .03	(-)	(+) Cannab		R
SUN. JUN. 28, 1981	A	P/P	S-1 S-2 BAC	NA NA .00	3.5 NA .00	Ø NA .00	2.4 NA .00	2.0 NA .00	(+) Cannab	(-)		R
	B		S-1 S-2 BAC								Subject did not show	R
	C	P/P	S-1 S-2 BAC	3.2 NA .00	3.2 NA .00	2.7 NA .00	2.3 NA .00	2.2 NA .00	(-)	(+) Cannab		R*
	D	M/A	S-1 S-2 BAC	6.4 NA .00	72 NA .04	16 NA .07	9.8 NA .07	7.9 NA .05	?	?	No urine samples obtained	R
	E	P/A	S-1 S-2 BAC	2.9 NA .00	3.1 NA .04	2.8 NA .05	2.7 NA .05	2.5 NA .03	(-)	(-)		R
	F	M/P	S-1 S-2 BAC	Ø NA .00	52 NA .00	21 NA .00	7.3 NA .00	3.9 NA .00	(-)	(-)		R

APPENDIX I TABLE A (continued)

(1) Drive Day & Date	(2) Subj.	(3) Treat-ment	(4) Type of Anal.	(5) Results: Serum Samples in ng/ml & % BAC, By Drive # 2	3	4	5	6	(6) Urine Screen other Drugs Pre	Post	(7) Remarks	(8) Subj. Data Used or Reject.
SAT. JUL. 11, 1981	A	P/P	S-1 S-2 BAC	Ø NA .00	Ø NA .00	NS NS .00	NS NS .00	NS NS .00	(-)	(-)	No blood draw Driv. # 4, 5, 6	U
	B	P/P	S-1 S-2 BAC	Ø NA .00	Ø NA .00	Ø NA .00	Ø NA .00	Ø NA .00	(-)	(-)		U
	C	M/P	S-1 S-2 BAC	Ø 2.8 .00	25 17 .00	NS NS .00	3.7 25 .00	2.8 15 .00	(-)	(-)		U
	D	M/A	S-1 S-2 BAC	Ø Ø .00	27 12 .09	3 8.7 .07	Ø 12 .05	2.6 7.7 .04	(-)	(-)		U
	E	M/A	S-1 S-2 BAC	2.9 26 .00	66 31 .06	8.3 29 .05	5.7 20 .03	3.6 9 .01	(+) Cannab	(+) Cannab		U
	F	M/A	S-1 S-2 BAC	Ø 2.6 .00	37 14 .04	7.3 14 .04	3.2 12 .04	Ø 11 .02	(+) Cannab	(+) Cannab		U
SUN. JUL. 12, 1981	A	M/P	S-1 S-2 BAC	2.4 15 .00	43 47 .00	12 37 .00	7 29 .00	5.9 30 .00	(-)	(-)		U
	B	P/A	S-1 S-2 BAC	Ø NA .00	Ø NA .08	Ø NA .08	Ø NA .06	Ø NA .04	(-)	(-)		U
	C	P/P	S-1 S-2 BAC	Ø NA .00	2.4 NA .00	Ø NA .00	Ø NA .00	Ø NA .00	(-)	(+) Cannab		U *
	D	M/P	S-1 S-2 BAC	14 106 .00	111 182 .00	30 109 .00	19 99 .00	16 92 .00	(+) Cannab	(+) Cannab		U
	E	P/A	S-1 S-2 BAC	3.2 NA .00	2.8 NA .07	Ø NA .05	Ø NA .04	2.5 NA .03	(-)	(+) Cannab		U
	F	M/A	S-1 S-2 BAC	Ø Ø .00	10 10 .05	2.8 9.3 .06	Ø 7.2 .05	Ø 6.8 .03	(-)	(+) Cannab		U

APPENDIX I TABLE A (continued)

(1) Drive Day & Date	(2) Subj.	(3) Treat-ment	(4) Type of Anal.	(5) Results: Serum Samples in ng/ml & % BAC, By Drive #					(6) Urine Screen other Drugs		(7) Remarks	(8) Subj. Data Used or Reject.
				2	3	4	5	6	Pre	Post		
SAT. JUL. 18, 1981	A	M/P	S-1 S-2 BAC	9.8 77 .00	33 89 .00	17 56 .00	12 44 .00	11 52 .00	(+) Cannab Coca	(+) Cannab Coca	Subj. Admitted Coca. Use on Driv Day	R *
	B	M/P	S-1 S-2 BAC	Ø Ø .00	18 18 .00	4.2 5.1 .00	2.6 4.5 .00	Ø 4.1 .00	(-)	(-)		U
	C	P/P	S-1 S-2 BAC	Ø Ø .00	Ø NA .00	Ø NA .00	Ø NA .00	Ø NA .00	(-)	(-)		U
	D	M/A	S-1 S-2 BAC	Ø Ø .00	20 15 .05	4.8 14 .07	3.6 12 .06	Ø 4.1 .04	(-)	(-)		U
	E	M/P	S-1 S-2 BAC	Ø 11 .00	45 38 .00	14 37 .00	5.3 22 .00	3.7 25 .00	(+) Cannab	(+) Cannab		U
	F	P/A	S-1 S-2 BAC	5.2 58 .00	5.0 51 .06	4.3 NA .09	4.6 NA .07	4.1 NA .05	(+) Cannab	(+) Cannab		U
SUN. JUL. 19, 1981	A	M/P	S-1 S-2 BAC	Ø Ø .00	43 31 .00	11 28 .00	2.9 21 .00	Ø 21 .00	(-)	(+) Cannab		U
	B	M/P	S-1 S-2 BAC	Ø 24 .00	43 37 .00	9.5 28 .00	5.1 19 .00	3.8 16 .00	(+) Cannab	(+) Cannab		U
	C	P/P	S-1 S-2 BAC	Ø Ø .00	Ø NA .00	Ø NA .00	Ø NA .00	Ø NA .00	(-)	(-)		U
	D	P/A	S-1 S-2 BAC	8.8 96 .00	7.2 NA .11	7.6 NA .08	6.3 NA .06	6.5 NA .04	(+) Cannab Coca	(+) Cannab Coca	Subject Admitted Coca Use on Driv. Day	R *
	E	P/A	S-1 S-2 BAC	Ø 6.8 .00	Ø NA .09	Ø NA .09	Ø NA .07	Ø NA .06	(+) Cannab	(-) Cannab		U
	F	M/A	S-1 S-2 BAC	Ø 16 .00	221 69 .05	29 60 .07	14 68 .05	9.8 56 .03	(-)	(+) Cannab		U

(1) Drive Day & Date	(2) Subj.	(3) Treatment	(4) Type of Anal.	(5) Results: Serum Samples in ng/ml & % BAC, By Drive #					(6) Urine Screen other Drugs		(7) Remarks	(8) Subj. Data Used or Reject.
				2	3	4	5	6	Pre	Post		
SAT. AUG. 1, 1981	A	P/P	S-1 S-2 BAC	2.8 6 .00	Ø NA .00	Ø NA .00	Ø NA .00	Ø NA .00	(+) Cannab Coca	(+) Cannab Coca		U
	B	P/A	S-1 S-2 BAC	Ø 3.8 .00	Ø NA .05	Ø NA .08	Ø NA .07	Ø NA .05	(+) Cannab Coca	(+) Cannab Coca	Serum Coca. By RIA (-)	U
	C	P/A	S-1 S-2 BAC	Ø 2.9 .00	Ø NA .08	Ø NA .10	Ø NA .08	Ø NA .06	(+) Cannab	(-)		U
	D	P/A	S-1 S-2 BAC	Ø Ø .00	Ø NA .08	Ø NA .09	Ø NA .07	Ø NA .06	(-)	(+) Cannab		U
	E	M/A	S-1 S-2 BAC	7.5 51 .00	85 54 .06	13 62 .07	12 50 .05	8.5 53 .03	(+) Cannab	(+) Cannab		U *
	F	P/A	S-1 S-2 BAC	10 159 .00	11 138 .09	7.4 NA .07	9.3 NA .05	8.5 NA .04	(+) Cannab Coca	(+) Cannab	Serum Coca By RIA (-)	U *
SUN. AUG. 2, 1981	A	P/P	S-1 S-2 BAC	Ø NA .00	3 NA .00	Ø NA .00	Ø NA .00	Ø NA .00	(-)	(-)		U
	B	M/P	S-1 S-2 BAC	3.6 8.2 .00	121 52 .00	18 55 .00	13 48 .00	7 40 .00	(-)	(+) Cannab		U
	C	M/P	S-1 S-2 BAC	3.8 22 .00	110 47 .00	NS NS .00	4 34 .00	2.7 37 .00	(+) Cannab	(+) Cannab		U
	D	M/P	S-1 S-2 BAC	Ø 2.5 .00	33 11 .00	3.8 8.4 .00	Ø 6.6 .00	Ø 5.6 .00	(-)	(+) Cannab.		U
	E	P/P	S-1 S-2 BAC	2.4 3.2 .00	Ø NA .00	Ø NA .00	Ø NA .00	Ø NA .00	(+) Cannab	(-)		U
	F	M/A	S-1 S-2 BAC	5.6 10 .00	112 29 .05	27 35 .07	13 38 .05	8.4 29 .03	(+) Cannab	(+) Cannab		U

APPENDIX I TABLE A (continued)

(1) Drive Day & Date	(2) Subj.	(3) Treatment	(4) Type of Anal.	(5) Results: Serum Samples in ng/ml & % BAC, By Drive #					(6) Urine Screen other Drugs		(7) Remarks	(8) Subj. Data Used or Reject.
				2	3	4	5	6	Pre	Post		
SAT. AUG. 8, 1981	A	M/A	S-1 S-2 BAC	Ø 25 .00	36 12 .03	7.7 12 .06	3.9 12 .05	2.2 8.4 .03	(-)	(+) Cannab		U
	B	M/A	S-1 S-2 BAC	Ø Ø .00	83 56 .03	22 34 .06	7.8 18 .05	4.5 17 .03	(-)	(+) Cannab		U
	C	P/P	S-1 S-2 BAC	Ø Ø .00	Ø NA .00	Ø NA .00	Ø NA .00	Ø NA .00	(-)	(-)		U
	D	P/P	S-1 S-2 BAC	2.1 5.3 .00	3.2 NA .00	Ø NA .00	2.1 NA .00	Ø NA .00	(+) Cannab	(+) Cannab		U
	E	M/P	S-1 S-2 BAC	Ø Ø .00	22 10 .00	4 6.6 .00	2.4 3.9 .00	Ø 3.2 .00	(-)	(+) Cannab		U*
	F	P/A	S-1 S-2 BAC	9.5 143 .00	12 126 .06	11 NA .11	12 NA .09	10 NA .07	(+) Cannab	(+) Cannab		U
SUN. AUG. 9, 1981	A	M/A	S-1 S-2 BAC	Ø 4.6 .00	21 24 .05	4.4 17 .07	6.2 13 .06	2.2 17 .05	(+) Cannab	(+) Cannab		U
	B	P/A	S-1 S-2 BAC	2.1 17 .00	2.3 NA .06	2.9 NA .07	4.0 NA .05	4.0 NA .03	(+) Cannab	(+) Cannab		U
	C	P/A	S-1 S-2 BAC	Ø Ø .00	Ø NA .09	Ø NA .10	Ø NA .08	Ø NA .06	(-)	(-)		U
	D	M/P	S-1 S-2 BAC	Ø Ø .00	49 45 .00	14 29 .00	5.1 22 .00	3.7 19 .00	(-)	(+) Cannab		U
	E	M/A	S-1 S-2 BAC	Ø 10 .00	113 33 .05	40 32 .07	16 23 .07	8.1 15 .06	(+) Cannab	(+) Cannab		U
	F	P/P	S-1 S-2 BAC	Ø 20 .00	3.5 NA .00	Ø NA .00	2.2 NA .00	2.6 NA .00	(+) Cannab Coca	(+) Cannab Coca	Serum Coca By RIA (+)	R

APPENDIX I TABLE A (continued)

(1) Drive Day & Date	(2) Subj.	(3) Treatment	(4) Type of Anal.	(5) Results: Serum Samples in ng/ml & % BAC, By Drive #					(6) Urine Screen other Drugs		(7) Remarks	(8) Subj. Data Used or Reject.
				2	3	4	5	6	Pre	Post		
SAT. AUG. 15, 1981	A	P/P	S-1 S-2 BAC	Ø Ø .00	3.2 NA .00	3.1 NA .00	Ø NA .00	Ø NA .00	(-)	(-)		U
	B	M/A	S-1 S-2 BAC	Ø 2.0 .00	21 7.4 .06	8.1 7.8 .08	3.8 6.2 .07	4.4 8.6 .05	(-)	(-)		U
	C	P/A	S-1 S-2 BAC	3.3 6.3 .00	2.1 NA .05	3.0 NA .06	Ø NA .05	2.9 NA .04	(+) Cannab	(+) Cannab		U
	D	M/A	S-1 S-2 BAC	Ø Ø .00	24 22 .06	10 21 .09	6.5 17 .07	6.0 15 .05	(+) Morp. Diap	(+) Morp Diazp Cannab	Serum Morp. By RIA (-)	U
	E	P/P	S-1 S-2 BAC	Ø Ø .00	2.7 NA .00	2.3 NA .00	Ø NA .00	Ø NA .00	(-)	(-)		U
	F	P/P	S-1 S-2 BAC	Ø 2.1 .00	2.3 NA .00	2.7 NA .00	Ø NA .00	Ø NA .00	(+) Cannab	(-)		U
SUN. AUG. 16, 1981	A	P/P	S-1 S-2 BAC	Ø Ø .00	Ø NA .00	Ø NA .00	Ø NA .00	Ø NA .00	(-)	(-)		U
	B	M/A	S-1 S-2 BAC	Ø 16 .00	35 36 .03	10 28 .06	6.3 26 .07	5.8 30 .05	(-) Cannab	(+) Cannab		U
	C	M/P	S-1 S-2 BAC	5.5 52 .00	117 92 .00	22 76 .00	19 75 .00	12 62 .00	(+) Cannab	(+) Cannab		U
	D	M/P	S-1 S-2 BAC	3.3 21 .00	87 80 .00	18 37 .00	13 38 .00	9.9 43 .00	(+) Cannab	(+) Cannab		U
	E	P/A	S-1 S-2 BAC	Ø Ø .00	3.8 NA .08	Ø NA .07	Ø NA .06	Ø NA .05	(-)	(-)		U
	F	P/A	S-1 S-2 BAC	Ø Ø .00	Ø Ø .08	Ø Ø .08	Ø Ø .06	Ø Ø .05	(-)	(-)		U

APPENDIX I TABLE A (continued)

(1) Drive Day & Date	(2) Subj.	(3) Treatment	(4) Type of Anal.	(5) Results: Serum Samples in ng/ml & % BAC, By Drive # 2	3	4	5	6	(6) Urine Screen other Drugs Pre	Post	(7) Remarks	(8) Subj. Data Used or Reject.
SUN. AUG. 23, 1981	A	P/P	S-1 S-2 BAC	Ø 18 .00	Ø NA .00	Ø NA .00	Ø NA .00	Ø NA .00	(+) Cannab	(+) Cannab		U
	B	M/P	S-1 S-2 BAC	Ø 4.4 .00	76 17 .00	23 20 .00	9.2 19 .00	4.7 26 .00	(-)	(+) Cannab		U
	C	P/P	S-1 S-2 BAC	7.7 96 .00	7.7 NA .00	6.5 NA .00	NS NS .00	NS NS	(+) Cannab Coca Amph	(+) Cannab Coca Amph	Serum Amph. By RIA (+)	R*
	D	M/P	S-1 S-2 BAC	22 86 .00	131 85 .00	37 76 .00	25 111 .00	26 103 .00	(+) Cannab	(+) Cannab Coca Amph	Serum Coca (-) Amph (+) By RIA	R*
	E	P/A	S-1 S-2 BAC	2.4 Ø .00	Ø NA .08	Ø NA .07	Ø NA .05	Ø NA .04	(-)	(+)		U
	F	M/A	S-1 S-2 BAC	Ø 9.2 .00	50 40 .07	15 30 .08	7 21 .06	5.3 20 .05	(+) Cannab	(+) Cannab		U
SAT. SEP. 5, 1981	A	P/A	S-1 S-2 BAC	Ø 11 .00	Ø NA .06	Ø NA .08	Ø NA .05	Ø NA .03	(+) Cannab	(+) Cannab		U
	B	P/P	S-1 S-2 BAC	Ø Ø .00	Ø NA .00	Ø NA .00	Ø NA .00	Ø NA .00	(-)	(-)		U
	C	M/A	S-1 S-2 BAC	7.8 43 .00	51 74 .09	21 58 .09	15 42 .08	12 42 .06	(+) Cannab	(+) Cannab		U*
	D	M/P	S-1 S-2 BAC	NA NA .00	45 28 .00	15 19 .00	5.5 18 .00	2.9 11 .00	(+) Cannab	(+) Cannab		U
	E	M/P	S-1 S-2 BAC	Ø 4.1 .00	38 32 .00	8.5 22 .00	4.2 23 .00	2.8 16 .00	(-)	(+) Cannab		U
	F	P/A	S-1 S-2 BAC	3.3 14 .00	4.2 NA .05	3.4 NA .06	2.3 NA .04	2.9 NA .02	(+) Cannab Coca	(+) Cannab Coca	Serum Coca By RIA (-)	U

APPENDIX I TABLE A (continued)

(1) Drive Day & Date	(2) Subj.	(3) Treatment	(4) Type of Anal.	(5) Results: Serum Samples in ng/ml & % BAC, By Drive # 2	3	4	5	6	(6) Urine Screen other Drugs Pre	Post	(7) Remarks	(8) Subj. Data Used or Reject.
SUN. SEP. 6, 1981	A	P/P	S-1	Ø	Ø	Ø	Ø	Ø	(+)	(+)		U
			S-2	8.4	NA	NA	NA	NA	Cannab	Cannab		
			BAC	.00	.00	.00	.00					
	B	M/A	S-1	Ø	69	16	8.7	5.1	(+)	(+)		U
			S-2	22	52	45	42	39	Cannab	Cannab		
			BAC	.00	.03	.06	.06	.04				
	C	M/A	S-1	Ø	24	6	2.6	Ø	(-)	(+)		U
			S-2	3.6	9.9	9.1	5.5	2.8		Cannab		
			BAC	.00	.04	.06	.06	.04				
	D	P/A	S-1	6.2	8.9	6.2	6.5	5.7	(-)	(-)		U*
			S-2	10	11	NA	NA	NA				
			BAC	.00	.07	.07	.05	.02				
	E	P/P	S-1	Ø	Ø	Ø	Ø	Ø	(-)	(-)		U
			S-2	Ø	NA	NA	NA	NA				
			BAC	.00	.00	.00	.00	.00				
	F	M/P	S-1	Ø	136	25	11	6	(+)	(+)		U
			S-2	22	41	28	32	26	Cannab	Cannab		
			BAC	.00	.00	.00	.00	.00				
MON. SEP. 7, 1981	A	M/P	S-1	3.3	159	19	4.9	2.6	(+)	(+)		U
			S-2	3.8	35	41	39	31	Diazp	Cannab Diazp		
			BAC	.00	.00	.00	.00	.00				
	B	P/P	S-1	Ø	2.9	Ø	2.1	Ø	(-)	(-)		U
			S-2	Ø	NA	NA	NA	NA				
			BAC	.00	.00	.00	.00	.00				
	C	P/A	S-1	Ø	3.2	Ø	Ø	Ø	(+)	(+)		U
			S-2	5.7	NA	NA	NA	NA	Cannab	Cannab		
			BAC	.00	.07	.08	.06	.05				
	D	P/P	S-1	Ø	Ø	Ø	Ø	Ø	(+)	(+)		U
			S-2	8.3	NA	NA	NA	NA	Cannab	Cannab		
			BAC	.00	.00	.00	.00	.00				
	E	M/A	S-1	3.9	10	25	14	6.8	(+)	(+)		U
			S-2	26	110	69	60	43	Cannab	Cannab		
			BAC	.00	.07	.08	.06	.05				
	F	M/P	S-1	Ø	23	5.1	2.5	Ø	(-)	(-)		U
			S-2	Ø	19	22	23	21				
			BAC	.00	.00	.00	.00	.00				

APPENDIX I TABLE A (continued)

(1) Drive Day & Date	(2) Subj.	(3) Treatment	(4) Type of Anal.	(5) Results: Serum Samples in ng/ml & % BAC, By Drive # 2	3	4	5	6	(6) Urine Screen other Drugs Pre	Post	(7) Remarks	(8) Subj. Data Used or Reject.
SUN. SEP. 13, 1981	A	M/A	S-1 S-2 BAC	Ø Ø .00	40 30 .09	16 17 .08	7.1 16 .09	5.3 15 .07	(+) Cannab	(+) Cannab		U
	B	P/P	S-1 S-2 BAC	Ø Ø .00	Ø NA .00	Ø NA .00	Ø NA .00	Ø NA .00	(-)	(-)		U
	C		S-1 S-2 BAC								Subject Did Not Show	R
	D	M/A	S-1 S-2 BAC	2.7 60 .00	40 74 .05	19 66 .06	10 50 .05	8 57 .03	(+) Cannab	(+) Cannab		U
	E	M/P	S-1 S-2 BAC	8.5 45 .00	118 89 .00	23 76 .00	16 61 .00	14 57 .00	(+) Cannab	(+) Cannab		U*
	F	P/A	S-1 S-2 BAC	2.7 12 .00	5.1 NA .06	Ø NA .07	Ø NA .06	3.4 NA .04	(+) Cannab	(+) Cannab		U
	A		S-1 S-2 BAC									
	B		S-1 S-2 BAC									
	C		S-1 S-2 BAC									
	D		S-1 S-2 BAC									
	E		S-1 S-2 BAC									
	F		S-1 S-2 BAC									

APPENDIX I TABLE B

Subject Serum Samples Selected for Follow-Up Study

on "BASAL" Cannabinoid Level

Driv. Date and Subj. Treatment	Subj.-Driv. #	Serum RIA (ng/ml) By DOJ-TOX-LAB		Reserve Samples Selected for Reanalysis by NIDA/RTI	Remarks
		Δ^9 - THC	9-COOH-THC		
(1) 7/19/81 P/A	D-2	8.8	96	D-3 Combined with D-4; ca 3 ML	Subject Rejected For Correlation with Performance Measures.
	D-3	7.2	NA		
	D-4	7.6	NA		
	D-5	6.3	NA		
	D-6	6.5	NA		
(2) 8/1/81 P/A	F-2	10	159	F-5, ca 2 ML	Subject Rejected For Correlation With Performance Measures.
	F-3	11	138		
	F-4	7.4	NA		
	F-5	9.3	NA		
	F-6	8.5	NA		
(3) 8/8/81 P/A	F-2	9.5	143	F-4 Combined with F-6, ca 2 ML	Sample Leaked In Transet., Insuff. Sample For Analysis
	F-3	12	126		
	F-4	11	NA		
	F-5	12	NA		
	F-6	10	NA		
(4) 8/23/81 P/P	C-2	7.7	9.6	C-3, ca 2 ml.	Sample Leaked In Transet., Insuff. Sample For Analysis.
	C-3	7.7	NA		
	C-4	6.5	NA		
	C-5	N.S	NA		
	C-6	N.S	NA		
(5) 9/6/81 P/A	D-2	6.2	10	D-5, ca 1 ml.	
	D-3	8.9	11		
	D-4	6.2	NA		
	D-5	6.5	NA		
	D-6	5.7	NA		
(6) 6/28/81 M/A	D-2	6.4	NA	D-2, ca 2 ml.	
	D-3	72	NA		
	D-4	16	NA		
	D-5	9.8	NA		
	D-6	7.9	NA		
(7) 7/12/81 M/P	D-2	14	106	D-2, ca 2 ml.	
	D-3	111	182		
	D-4	30	109		
	D-5	19	99		
	D-6	16	92		
(8) 7/18/81 M/P	A-2	9.8	77	-No Rserve Sample	Subject Rejected For Correlation with Performance measures.
	A-3	33	89		
	A-4	17	56		
	A-5	12	44		
	A-6	11	52		

APPENDIX I TABLE B (continued)

Driv. Date and Subj. Treatment	Subj.- Driv. #	Serum RIA (ng/ml) By DOJ-TOX-LAB		Reserve Samples Selected for Reanalysis by NIDA/RTI	Remarks
		Δ^9- THC	9-COOH-THC		
(9)	E-2	7.5	51	-No Reserve Sample	Subject Rejected For Correlation with Performance measures.
	E-3	85	54		
8/1/81	E-4	13	62		
	E-5	12	50		
M/A	E-6	8.5	53		
(10)	D-2	22	86	-No Reserve Sample	Subject Rejected For Correlation with Performance measures.
	D-3	121	85		
8/23/81	D-4	37	76		
	D-5	25	111		
M/P	D-6	26	103		
(11)	C-2	7.8	43	-No Rserve Sample	
	C-3	51	74		
9/5/81	C-4	21	58		
	C-5	15	42		
M/A	C-6	12	42		
(12)	E-2	8.5	45	E-2, ca 2 ml.	
	E-3	118	89		
9/13/84	E-4	23	76		
	E-5	16	61		
M/P	E-6	14	57		

NOTE: (1) Treatment Code: M= Marijuana; A= Alcohol; P= Placebo

(2) Subject Driv. # (Samples 2 to 6) taken at one hour intervals. Treatment given between Driv. # 2 (baseline) and Driv. # 3.

APPENDIX I TABLE C

Results of Selected Samples from Table 2 Sent

to NIDA/RTI for Reanalysis, Nov. 1981

Samples Driv. Date/ Subj.-Driv. #	(ng/ml) Δ^9 - THC:			(ng/ml) 11-nor-Δ^9-THC-9-COOH
	DOJ TOX LAB (^{125}I RIA)	RTI (^{125}I RIA)	RTI (GC/MS)	By RTI - (^{125}I RIA)
7-19-81, D-3	7.2	9.6	5.2	47.5
D-4	7.6	9.2	3.8	40.2
8-1-81, F-5	9.3	10.6	2.3	90.1
8-8-81, F-4	10.8	14.6	NA	61.8
F-6	10.4	12.9	3.8	66.9
8-23-81, E-3	7.7	11.1	9.5	72.6
9-6-81, D-5	6.5	8.8	2.1	14.7
6-28-81, D-2	6.4	7.9	1.9	26.7
7-12-81, D-2	13.4	16.4	7.0	93.4
8-1-81, E-2	7.5	8.8	2.2	48.1
9-13-81, E-2	8.5	10.4	2.1	31.8

NOTE: Because of the limited sample size, and loss of some sample in transit, no 9-COOH-THC analysis was performed.

APPENDIX I TABLE VI

Results from Follow-Up Study of "BASAL" Cannabinoid Levels In Serum Samples fo Selected Subjects from Table 2, June 1982

Initial Driv. Date-Subj. (Follow-Up Sample)	Follow-Up Sample Time/ Type Anal.	GC/MS Results, ng/ml (1)				Remarks
		1st	2nd	3rd	4th	
8/8/81-F	Time	0850	1040	1240	1435	On Initial Driv.
	Δ^9 - THC	6.0	5.3	5.5	4.8	Date Subj. F
(6-5-82)	11-OH-THC	1.7	1.6	2.8	3.1	Treatment
	9-COOH-THC	177	205	185	187	was P/A
9/6/81-D	Time	0815	1025	1235	1415	On Initial Driv.
	Δ^9 - THC	1.2	1.1	1.0	0.9	Date Subj. D
(6-4-82)	11-OH-THC	<0.1	<0.1	0.8 (2)	0.1	Treatment
	9-COOH-THC	13.1	10.7	10.2	10.0	was P/A
8/23/81-D	Time	0840	1030	No	1425	On Intitial Driv.
	Δ^9 - THC	4.0	3.3	|	2.5	Date Subj. D
	11-OH-THC	1.6	1.4	Sample	0.4	Treatment
	9-COOH-THC	43	49	↓	40	Was M/P
9/5/81-C	Time	0845	1035	1235	1430	On Initial Driv.
	Δ^9 - THC	1.3	1.1	1.2	1.1	Date Subj. C
(6-5-82)	11-OH-THC	<0.1	<0.1	<0.1	0.5	Treatment
	9-COOH-THC	43	36	30	36	was M/A

NOTES: (1) GC/MS Analyses of these samples conducted by R.L. Foltz Ph.D., Center for Human Toxicology, University of Utah, Salt Lake City

(2) Contribution for Intering Peak

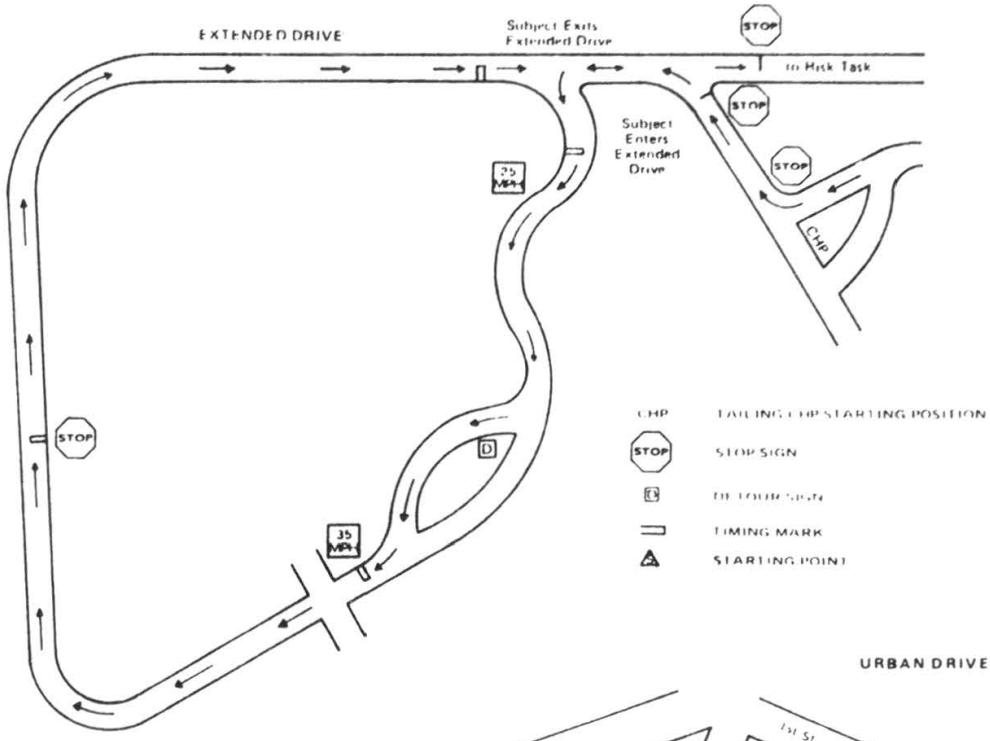
APPENDIX II

A. SCHEMATIC OF DRIVING COURSE

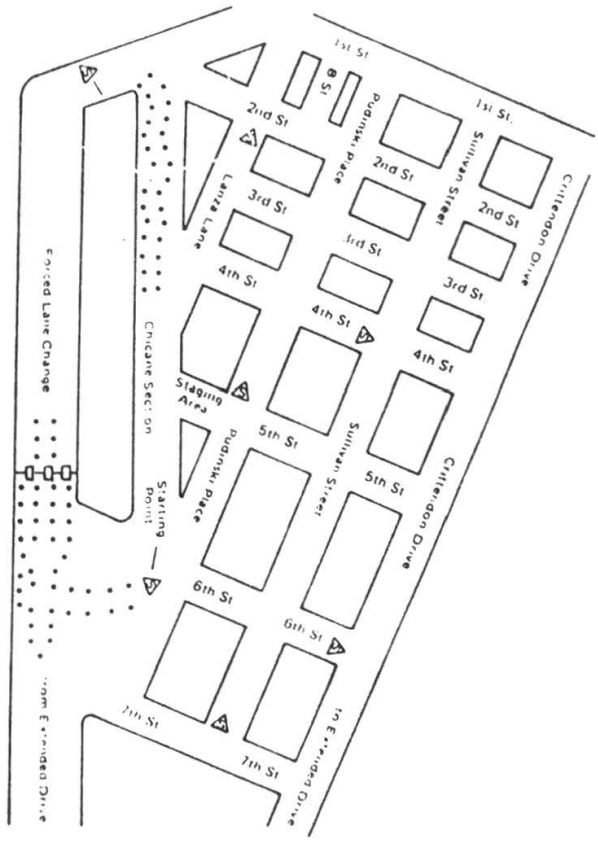
B. LICENSE REGISTRATION EXAMINER'S (LRE) SCORING FORMS

RE: GROSSEN ET AL, PERFORMANCE STUDY EXPERIMENTAL PROTOCOL, OTS. PROJECT 089702, SEPTEMBER 28, 1981.

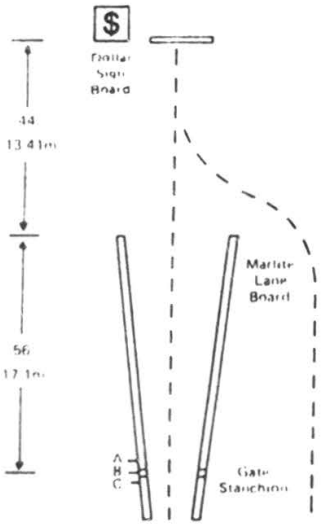
FIGURE 3 – MAP OF TEST AREAS



URBAN DRIVE



RISK TASK



Minimum speed through the Risk Task is 25 mph (40 kph).

Driver may drive around the channel without penalty as long as no wheel touches the white Marlite lane boards and no stanchions are touched. The minimum speed of 25 mph (40 kph) must be maintained.

This exercise is continued two more times in sequence with a shifting placement of the gate widths (A, B or C position).

The Dollar Sign Board will display either a $1, a $2, or a $5 sign.

The relationship between dollar value and gate setting is completely random.

Gate Stanchion Settings

A 79.5" (2.02m)

B 73.5" (1.87m)

C 67.5" (1.71m)

Failure occurs when:

1. Minimum speed of 25 mph is not maintained.
2. Gate stanchions are hit by vehicle.
3. Marlite lane boards in front of gate are knocked [illegible]

Rater (Name) _____

Subject I.D. #_____ Run #_____

Time _____ Date _____

1. Start

*** "I will be unable to answer most of your questions! Drive the chicane as fast as you can, hitting as few stanchions as possible. How fast do you feel you can drive the chicane?"

*** Speed Est. By Subject
_____ mph

*** Starting Speed
Slow 1 2 3 4 5 Fast

2. Chicane

*** Steering Control
Overcautious 1 2 3 4 5 Reckless

*** Speed Control
Overcautious 1 2 3 4 5 Reckless

*** Time to Complete Chicane
_____ Seconds
_____ mph (from chart)

3. Stop Sign

*** Stop
( )Abrupt Stop
( )Misjudged Stop Distance
( )Smooth Stop
( )Rolling Stop
( )No Stop- Reduced Speed

*** Position at Stop
( )Good
( )Average
( )Poor

4. Forced Lane Change

*** "Maintain a speed of between 30 and 34 MPH. If you do not drive within this speed range you will be required to repeat this task once."

*** Starting Speed
Slow 1 2 3 4 5 Fast

5. First Forced Lane Change

*** 30 mph Approach
( )Reminded of Speed
( )Not Reminded of Speed

*** Speed at Timing Mark
_____ mph

*** Response to Lights
( )Correct
( )Incorrect
( )None - Froze

*** Quality of Response
Overcautious 1 2 3 4 5 Reckless

*** Stop
( )Abrupt Stop
( )Misjudged Stop Distance
( )Smooth Stop
( )Rolling Stop
( )No Stop Reduced Speed

6. Second Forced Lane Change (optional)

*** 30 mph Approach
( )Reminded of Speed
( )Not Reminded of Speed

*** Speed at Timing Mark
_____mph

*** Response to Lights
( )Correct
( )Incorrect
( )None - Frose

*** Quality of Response
Overcautious 1 2 3 4 5 Reckless

*** Stop
( )Abrupt Stop
( )Misjudged Stop Distance
( )Smooth Stop
( )Rolling Stop
( )No Stop Reduced Speed

7. Back up and Detour

*** Reverse Start
Slow 1 2 3 4 5 Fast

*** Reverse Stop
( )Abrupt Stop
( )Misjudged Stop Distance
( )Smooth Stop
( )Rolling Stop
( )No Stop- Reduced Speed

*** Forward Starting Speed
Slow 1 2 3 4 5 Fast

8 Urban Drive

*** "The Speed limit for the urban drive is 25 mph Follow all normal driving procedures using all driving rules and precautions I will not be able to help you interpret the driving route; however, if you miss a turn I will have you back up in order to stay on the course"

*** Go to route instructions

Rater (Name) _____

Subject I.D. # _____ Run # _____

Time _____ Date _____

Appendix 6C

ROUTE #2

1. Start (straight onto 6th St.)
   *** Starting Speed
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

2. Left turn onto Sullivan St.
   *** Left Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

3. Left turn onto 4th St.
   *** Left Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

4. Right turn onto Pudinski Pl.
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

5. Left turn onto 3rd St.
   *** Left Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

6. Right turn onto Lanza Lane
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

7. Stop sign on Lanza Lane
   *** Stop
   ( ) Abrupt Stop
   ( ) Misjudged Stop Distance
   ( ) Smooth Stop
   ( ) Rolling Stop
   ( ) No Stop - Reduced Speed
   ( ) Failure to Comply

8. Right turn on 1st St.
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

9. Right turn onto Pudinski Pl.
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

10. Left turn onto 2nd St.
    *** Left Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

11. Right turn onto Sullivan St.
    *** Right Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

12. Stop sign on Sullivan St.
    *** Stop
    ( ) Abrupt Stop
    ( ) Misjudged Stop Distance
    ( ) Smooth Stop
    ( ) Rolling Stop
    ( ) No Stop - Reduced Speed
    ( ) Failure to Comply

13. Left turn onto 7th St.
    *** Left Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

14. Right turn onto Crittendon Drive
    *** Right Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

DRIVE FROM URBAN SKILLS COURSE TO EXTENDED DRIVE

Rater (Name) _____

Subject I.D. # _____ Run # _____

Time _____ Date _____

ROUTE #3

Appendix 6C

1. Start (Left onto Pudinski Pl.)
   *** Starting Speed
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

2. Right turn onto 5th St.
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

3. Stop sign on 5th St.
   *** Stop
   ( ) Abrupt Stop
   ( ) Misjudged Stop Distance
   ( ) Smooth Stop
   ( ) Rolling Stop
   ( ) No Stop - Reduced Speed
   ( ) Failure to Comply

4. Left turn onto Sullivan St.
   *** Left Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

5. Left turn onto 4th St.
   *** Left Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

6. Right turn onto Lanza Lane
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

7. Stop sign on Lanza Lane
   *** Stop
   ( ) Abrupt Stop
   ( ) Misjudged Stop Distance
   ( ) Smooth Stop
   ( ) Rolling Stop
   ( ) No Stop - Reduced Speed
   ( ) Failure to Comply

8. Right turn onto 1st St.
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

9. Right turn onto Sullivan St.
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

10. Right turn onto 3rd St.
    *** Right Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

11. Yield sign on 3rd St.
    *** Speed
    Slow 1 2 3 4 5 Fast
    *** Compliance to Yield
    Overcautious 1 2 3 4 5 Reckless

12. Left turn onto Pudinski Pl.
    *** Left Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

13. Stop sign on Pudinski Pl.
    *** Stop
    ( ) Abrupt Stop
    ( ) Misjudged Stop Distance
    ( ) Smooth Stop
    ( ) Rolling Stop
    ( ) No Stop - Reduced Speed
    ( ) Failure to Comply

14. Left turn onto 5th St.
    *** Left Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

15. Stop sign on 5th St.
    *** Stop
    ( )Abrupt Stop
    ( )Misjudged Stop Distance
    ( )Smooth Stop
    ( )Rolling Stop
    ( )No Stop- Reduced Speed
    ( ) Failure to Comply

16. Right turn onto Sullivan St.
    *** Right Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

17. Left turn onto 6th St.
    *** Left Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

18. Stop sign on 6th St.
    *** Stop
    ( ) Abrupt Stop
    ( ) Misjudged Stop Distance
    ( ) Smooth Stop
    ( ) Rolling Stop
    ( ) No Stop - Reduced Speed
    ( ) Failure to Comply

19. Right turn onto Crittendon Drive
    *** Right Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

DRIVE FROM URBAN SKILLS COURSE TO EXTENDED DRIVE

Rater (Name) _____

Subject I.D. #_____ Run # _____

Time _____ Date _____

ROUTE #4

1. Start (left onto Pudinski Pl.)
   *** Starting Speed
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

2. Left turn onto 4th St.
   *** Left Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

3. Right turn onto Lanza Lane
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

4. Stop sign on Lanza Lane
   *** Stop
   ( ) Abrupt Stop
   ( ) Misjudged Stop Distance
   ( ) Smooth Stop
   ( ) Rolling Stop
   ( ) No Stop - Reduced Speed
   ( ) Failure to Comply

5. Right turn onto 2nd St.
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

6. Left turn onto B St.
   *** Left Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

7. Right turn onto 1st St.
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

8. Right turn onto Sullivan St.
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

9. Right turn onto 4th St.
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

10. Left turn onto Pudinski Pl.
    *** Left Turn
    Sharp 1 2 3 4 5. Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

11. Stop Sign on Pudinski Pl.
    *** Stop
    ( ) Atrupt Stop
    ( ) Misjudged Stop Distance
    ( ) Smooth Stop
    ( ) Rolling Stop
    ( ) No Stop - Reduced Speed
    ( ) Failure to Comply

12. Left turn onto 5th St.
    *** Left Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

13. Stop sign on 5th St.
    *** Stop
    ( ) Abrupt Stop
    ( ) Misjudged Stop
    ( ) Smooth Stop
    ( ) Rolling Stop
    ( ) No Stop - Reduced Speed
    ( ) Failure to Comply

14. Right turn onto Sullivan St.
    *** Right Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

15. Stop sign on Sullivan St.
    *** Stop
    ( ) Abrupt Stop
    ( ) Misjudged Stop Distance
    ( ) Smooth Stop
    ( ) Rolling Stop
    ( ) No Stop Reduced Speed
    ( ) Failure to Comply

16. Left turn onto 7th St.
    *** Left Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

17. Right turn onto Crittendon Dr.
    *** Right Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

DRIVE FROM URBAN SKILLS COURSE TO EXTENDED DRIVE

Rater (Name) _____

Subject I.D. #_____ Run # _____

Time _____ Date _____

ROUTE #5

1. Start (straight onto 6th St.)
   *** Starting Speed
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

2. Left turn onto Sullivan St.
   *** Left Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

3. Left turn onto 5th St.
   *** Left Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

4. Right turn onto Pudinski Pl.
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

5. Left turn onto 2nd St.
   *** Left Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

6. Right turn onto Lanza Lane
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

7. Right turn onto 1st St.
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

8. Right turn onto Sullivan St.
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

9. Right turn onto 4th St.
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

10. Left turn onto Pudinski Pl.
    *** Left Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

11. Stop Sign on Pudinski Pl.
    *** Stop
    ( ) Abrupt Stop
    ( ) Misjudged Stop Distance
    ( ) Smooth Stop
    ( ) Rolling Stop
    ( ) No Stop - Reduced Speed

12. Left turn onto 6th Ave.
    *** Left Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

13. Right turn onto Sullivan St.
    *** Right Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

14. Stop Sign on Sullivan St.
    *** Stop
    ( ) Abrupt Stop
    ( ) Misjudged Stop Distance
    ( ) Smooth Stop
    ( ) Rolling Stop
    ( ) No Stop - Reduced Speed

15. Left turn onto 7th St.
    *** Left Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

16. Right turn onto Crittenden Drive
    *** Right Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

DRIVE FROM URBAN SKILLS COURSE TO EXTENDED DRIVE

Appendix C

Rater (Name) _____

Subject I.D. # _____ Run # _____

Time _____ Date _____

ROUTE #6

1. Start (left onto Pudinski Pl.)
   *** Starting Speed
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

2. Right onto 5th St.
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   ( ) Failure to Comply

3. Stop sign on 5th St.
   *** Stop
   ( ) Abrupt
   ( ) Misjudged Stop Distance
   ( ) Smooth Stop
   ( ) Rolling Stop
   ( ) No Stop - Reduced Speed
   ( ) Failure to Comply

4. Left onto Sullivan St.
   *** Left Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

5. Le onto 3rd St.
   *** Left Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

6. Yield sign on 3rd St.
   *** Speed
   Slow 1 2 3 4 5 Fast
   *** Compliance to Yield
   Overcautious 1 2 3 4 5 Reckless

7. Right onto Lanza Lane
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

8. Stop sign on Lanza Lane
   *** Stop
   ( ) Abrupt
   ( ) Misjudged Stop Distance
   ( ) Smooth Stop
   ( ) Rolling Stop
   ( ) No Stop - Reduced Speed
   ( ) Failure to Comply

9. Right onto 2nd Street
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast

10. Right onto Pudinski Pl.
    *** Right Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

11. Left onto 4th St.
    *** Left Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

12. Stop sign on 4th St.
    *** Stop
    ( ) Abrupt
    ( ) Misjudged Stop Distance
    ( ) Smooth Stop
    ( ) Rolling Stop
    ( ) No Stop - Reduced Speed
    ( ) Failure to Comply

13. Right turn onto Sullivan St.
    *** Right Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

14. Stop sign on Sullivan St.
    *** Stop
    ( ) Abrupt Stop
    ( ) Misjudged Stop Distance
    ( ) Smooth Stop
    ( ) Rolling Stop
    ( ) No Stop - Reduced Speed
    ( ) Failure to Comply

15. Left onto 7th St.
    *** Left Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

16. Right onto Crittendon Dr.
    *** Right Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast

DRIVE FROM URBAN SKILLS COURSE TO EXTENDED DRIVE

Rater (Name) _____

Subject I.D.# _____ Run # _____

Time _____ Date _____

ROUTE #7

Appendix 6C

1. Start (left onto Pudinski Pl.)
   *** Starting Speed
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

2. Right turn onto 4th St.
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

3. Stop sign on 4th St.
   *** Stop
   ( ) Abrupt Stop
   ( ) Misjudged Stop Distance
   ( ) Smooth Stop
   ( ) Rolling Stop
   ( ) No Stop - Reduced Stop
   ( ) Failure to Comply

4. Left turn onto Sullivan St.
   *** Left Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

5. Left turn onto 3rd St.
   *** Left Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

6. Yield sign on 3rd St.
   *** Speed
   Slow 1 2 3 4 5 Fast
   *** Compliance to Yield
   Overcautious 1 2 3 4 5 Reckless

7. Right turn onto Lanza Lane
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

8. Stop sign on Lanza Lane
   *** Stop
   ( ) Abrupt Stop
   ( ) Misjudged Stop Distance
   ( ) Smooth Stop
   ( ) Rolling Stop
   ( ) No Stop - Reduced Speed
   ( ) Failure to Comply

9. Right turn onto 1st St.
   *** Right Turn
   Sharp 1 2 3 4 5 Wide
   *** Speed of Turn
   Slow 1 2 3 4 5 Fast
   ( ) Failure to Comply

10. Right turn onto Sullivan St.
    *** Right Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

11. Right turn onto 4th St.
    *** Right Turn
    Sharp 1 2 3 4 5
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

12. Left turn onto Pudinski Pl.
    *** Left Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

13. Stop sign on Pudinski Pl.
    *** Stop
    ( ) Abrupt Stop
    ( ) Misjudged Stop
    ( ) Smooth Stop
    ( ) Rolling Stop
    ( ) No Stop - Reduced Speed
    ( ) Failure to Comply

DRIVE FROM URBAN SKILLS COURSE TO EXTENDED DRIVE

14. Left turn onto 5th St.
    *** Left Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

15. Stop sign on 5th St.
    *** Stop
    ( ) Abrupt Stop
    ( ) Misjudged Stop
    ( ) Smooth Stop
    ( ) Rolling Stop
    ( ) No Stop Reduced Speed
    ( ) Failure to Comply

16. Right turn onto Sullivan St.
    *** Right Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

17. Left turn onto 6th St.
    *** Left Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

18. Stop sign on 6th Street
    *** Stop
    ( ) Abrupt Stop
    ( ) Misjudged Stop Distance
    ( ) Smooth Stop
    ( ) Rolling Stop
    ( ) No Stop - Reduced Speed
    ( ) Failure to Comply

19. Right turn onto Crittendon Dr.
    *** Right Turn
    Sharp 1 2 3 4 5 Wide
    *** Speed of Turn
    Slow 1 2 3 4 5 Fast
    ( ) Failure to Comply

1. Drive to Extended Drive

*** First Lane Position Marker
( )Out of Lane- Right
( )Right of Lane
( )Center of Lane
( )Left of Lane
( )Out of Lane- Left

*** Second Lane Position Marker
( )Out of Lane- Right
( )Right pf Lane
( )Center of Lane
( )Left of Lane
( )Out of Lane- Left

*** Steering Control
Overcautious 1 2 3 4 5 Reckless

*** Speed Control
Overcautious 1 2 3 4 5 Reckless

2. Stop Signs

*** Stop
( )Abrupt Stop
( )Misjudged Stop Distance
( )Smooth Stop
( )Rolling Stop
( )No Stop- Reduced Speed

*** Starting Speed
Slow 1 2 3 4 5 Fast

*** Stop
( )Abrupt Stop
( )Misjudged Stop Distance
( )Smooth Stop
( )Rolling Stop
( )No Stop- Reduced Speed

3. START EXTENDED DRIVE

*** "Drive the Extended Drive Course as if it were a two lane road. Obey all traffic rules and signs."

*** Left Turn
Sharp 1 2 3 4 5 Wide
( )Failure to Comply

*** Starting Speed
Slow 1 2 3 4 5 Fast

*** Left Turn Entering Course
Sharp 1 2 3 4 5 Wide
( )Failure to Comply

*** Speed of Turn
Slow 1 2 3 4 5 Fast

4. Segment One

*** First Lane Position Marker
( )Out of Lane- Right
( )Right of Lane
( )Center of Lane
( )Left of Lane
( )Out of Lane- Left

*** Second Lane Position Marker
( )Out of Lane- Right
( )Right of Lane
( )Center of Lane
( )Left of Lane
( )Out of Lane- Left

*** Third Lane Position Marker
( )Out of Lane- Right
( )Right of Lane
( )Center of Lane
( )Left of Lane
( )Out of Lane- Left

*** Fourth Lane Position Marker
( )Out of Lane- Right
( )Right of Lane
( )Center of Lane
( )Left of Lane
( )Out of Lane- Left

*** Steering Control
Overcautious 1 2 3 4 5 Reckless

*** Speed Control
Overcautious 1 2 3 4 5 Reckless

5. Segment Two

*** Quality of Response to Detour Sign
Overcautious 1 2 3 4 5 Reckless

*** Steering Control Through Detour
Overcautious 1 2 3 4 5 Reckless

*** Speed Control Through Detour
Overcautious 1 2 3 4 5 Reckless

*** First Lane Position Marker
( )Out of Lane Right
( )Right of Lane
( )Center of Lane
( )Left of Lane
( )Out of Lane Left

*** Second Lane Position Marker
( )Out of Lane Right
( )Right of Lane
( )Center of Lane
( )Left of Lane
( )Out of Lane Left

*** Steering Control
Overcautious 1 2 3 4 5 Reckless

*** Speed Control
Overcautious 1 2 3 4 5 Reckless

6. Segment Three

***Stop Sign
( )Abrupt Stop
( )Misjudged Stop Distance
( )Smooth Stop
( )Rolling Stop
( )No Stop Reduced Speed

*** Position at Stop
( )Good
( )Average
( )Poor

IMPORTANT- place cover over speedometer before starting.

*** Starting Speed
Slow 1 2 3 4 5 Fast

6. Continued

*** First Lane Position Marker
( )Out of Lane- Right
( )Right of Lane
( )Center of Lane
( )Left of Lane
( )Out of Lane- Left

*** Speed Monitored
_____ mph

*** Second Lane Position Marker
( )Out of Lane- Right
( )Right of Lane
( )Center of Lane
( )Left of Lane
( )Out of Lane- Left

*** Third Lane Position Marker
( )Out of Lane- Right
( )Right of Lane
( )Center of Lane
( )Left of Lane
( )Out of Lane- Left

*** Fourth Lane Position Marker
( )Out of Lane- Right
( )Right of Lane
( )Center of Lane
( )Left of Lane
( )Out of Lane- Left

*** Speed Monitored
_____ mph

*** Steering Control
Overcautious 1 2 3 4 5 Reckless

*** Speed Control
Overcautious 1 2 3 4 5 Reckless

*** Maximum Speed Segment 3.
_____ mph

*** Stop Sign
( )Abrupt Stop
( )Misjudged Stop
( )Smooth Stop
( )Rolling Stop
( )No Stop- Reduced Speed

*** Position at Stop
( )Good
( )Average
( )Poor

IMPORTANT- remove speedometer cover.

7. Risk Task

*** "The minimum speed through each gate of the risk task is 25 mph. Dollar values for each gate are posted at the decision point. You may choose to drive through or to drive around each gate individually. By driving through a gate you may gain or lose the amount posted. By driving around a gate you will not gain or lose any amount."

*** Starting Speed
Slow 1 2 3 4 5 Fast

*** Quality of Response
Overcautious 1 2 3 4 5 Reckless

*** Driver Choice
( )Drove Through
( )Drove Around

*** Speed and Risk Code
MPH_____
Risk #_____

*** Quality of Response
Overcautious 1 2 3 4 5 Reckless

***Driver Choice
( )Drove Through
( )Drove Around

*** Speed and Risk Code
MPH_____
Risk #_____

*** Quality of Response
Overcautious 1 2 3 4 5 Reckless

*** Driver Choice
( )Drove through
( )Drove Around

*** Speed and Risk Code
MPH _____
Risk # _____

In your opinion, do you feel this driver is:

Not Impaired Definitely Impaired
0 1 2 3 4 5 6 7 8 9

*** Comments:

APPENDIX III

TABLES 1-5

By RAYMOND PECK (1985)

APPENDIX III, TABLE 1

Significant Univariate Discriminators of Post-Treatment Performance
(Run 3 - Initial Variable Pool = 72)

Variable	Treatment means				F-ratios	Significance level (p)*
	Placebo	Marijuana	Alcohol	Both		
FALLCAR.....	1.21	1.59	1.42	1.59	2.36	.078
USPEED.....	0.12	0.20	0.26	0.33	3.47	.020
UTURNS.....	0.18	0.34	0.30	0.39	3.43	.021
CHICANE.....	0.74	1.94†	2.16†	1.64	2.48	.067
POSTOP.....	4.05	6.35†	5.52†	4.33	4.59	.005
ELANEPOS.....	1.32	1.06	1.37	2.50†	2.84	.044
ESTEER.....	0.58	1.12	1.37	1.91†	3.47	.020
ESPEED.....	0.47	0.59	0.95	1.50†	3.14	.030
SLANEPOS.....	0.32	0.47	0.53	1.50†	4.32	.007
SSTEER.....	0.05	0.18	0.16	0.50	3.28	.026
RISKQU.....	0.32	1.00	1.42†	1.14†	2.17	.099
FINGNOSE	1.42	1.65	2.16†	1.73	2.21	.094
HEELTOE.....	1.26	1.76	1.58	2.00†	3.00	.036
COUNTBAC.....	1.16	1.18	1.26	1.77	2.64	.056
ALPH.....	1.00	1.00	1.32	1.14	2.40	.075
OFFRATE.....	1.63	2.18	2.84†	2.95†	3.29	.025
SELFRATE.....	2.26	3.41†	3.95†	4.77†	5.31	.002
BIPOST.....	4.42	5.82†	5.32†	4.59	4.59	.005
BIELANE.....	0.79	0.94	0.95	2.14†	2.61	.058
BISTOP.....	0.08	-0.15	0.45	-0.18	2.47	.068
BISSPEED.....	0.16	0.24	0.37	0.86	2.43	.070
BISSTEER.....	0.05	0.18	0.16	0.50	3.27	.026
COGNIT.....	4.53	5.29	5.58	6.32†	2.68	.053
STOUCH.....	7.05	6.06	8.32†	7.14	3.15	.029
ESTCHIC.....	19.65	20.14	20.54	14.09†	6.98	.000
SMPH.....	34.12	37.66†	36.62†	39.92†	4.95	.003
ERRORS.....	6.66	9.89†	9.88†	14.44†	5.04	.003
BSTEER.....	1.44	1.72	1.97	4.67†	3.04	.034
OVERALL.....	0.68	1.94†	2.05†	2.91†	6.01	.001

*$p < .10$; $df = \frac{3}{73}$

† Treatment vs. placebo contrast significant at $p \leq .05$ (Bonferonni t test)

6098-1

APPENDIX III, TABLE 2

Significant Univariate Discriminators of Post-Treatment Performance
(Run 4 - Initial Variable Pool = 72)

Variable	Treatment means				F-ratios	Significance level (p)*
	Placebo	Marijuana	Alcohol	Both		
STOPS	0.75	0.69	1.42	0.67	2.39	.075
COGNIT	4.45	4.76	5.39	5.76†	2.69	.053
BSPEED	0.18	0.62	1.40	2.65†	2.73	.050
POSTOP	4.45	5.41†	5.33†	4.95	2.53	.064
ESTCHIC	20.35	19.35	19.89	17.29†	3.65	.017
SMPH	35.45	37.96†	37.43†	39.05†	4.42	.007
CTT	4.91	4.94	4.27†	4.16†	8.78	.000
ERRORS	6.19	7.74	9.35†	12.49†	4.20	.009
OVERALL	0.70	1.24	1.61†	2.00†	3.25	.027
COORD	3.95	4.29	4.50	5.43†	2.63	.057
FALLCAR	1.15	1.29	1.56	1.57	3.38	.023
UTURNS	0.20	0.34	0.26	0.41	3.41	.022
ESTEER	0.65	0.71	1.33	1.43	2.60	.059
ESTOP	0.35	0.29	0.83	0.38	2.72	.051
DETOUR	0.35	0.65	0.39	0.95	2.47	.069
SLANEPOS	0.15	0.06	0.33	1.00†	4.54	.006
ROMB	1.30	1.12	1.28	1.57	2.38	.076
HEELTOE	1.50	1.24	1.50	1.90	2.29	.086
COUNTBAC	1.00	1.12	1.50	1.33	2.94	.039
OFFRATE	1.35	1.82	2.11	2.81†	5.27	.002
SELFRATE	1.70	2.47	2.39	4.29†	8.10	.001
BIPOST	4.45	5.41†	5.33†	4.95	2.53	.064
BISSPEED	0.00	0.41	0.83†	1.00†	3.78	.014
BISLANE	0.15	0.06	0.11	1.00†	4.92	.004

*$p \leq .10$; $df = \frac{3}{72}$

† Treatment vs. placebo contrast significant at $p \leq .05$ (Bonferonni t test)

6098-2

APPENDIX III, TABLE 3

Significant Univariate Discriminators of Post-Treatment Performance
(Run 5 - Initial Variable Pool = 72)

Variable	Treatment means				F-ratios	Significance level (p)*
	Placebo	Marijuana	Alcohol	Both		
STOUCH.....	7.00	7.06	7.63	8.33†	2.47	.069
SDOWN.....	8.79	5.33	9.16	7.24	6.22	.001
BSPEED.....	-0.22	0.80	0.71	2.47†	3.62	.017
ESTOHIC.....	21.47	19.78†	20.53	18.71†	2.20	.095
CTT.....	4.93	5.20	4.53	4.45	4.75	.004
ERRORS.....	7.27	6.37	7.30	11.35†	3.08	.032
OVERALL.....	0.42	0.83	0.84	1.38†	2.82	.045
FALLCAR.....	1.11	1.17	1.21	1.52	4.07	.001
BITE.....	58.32	57.50	62.47	49.90	2.25	.090
ELANEPOS.....	0.84	0.61	1.00	1.81†	2.39	.076
ESTEER.....	0.79	0.89	0.84	1.62†	2.23	.092
ESTOP.....	0.26	0.44	0.68	0.29	2.36	.079
DETOUR.....	0.42	0.56	0.32	1.10†	4.14	.009
SSPEED.....	0.58	0.50	0.95	1.38†	4.13	.009
SELFRATE.....	1.05	1.06	1.42	3.19†	7.74	.000
BISSPEED.....	-0.26	0.39	0.63†	1.19†	7.78	.000

*$p \leq .10$; $df = \frac{3}{73}$

† Treatment vs. placebo contrast significant at $p \leq .05$ (Bonferonni t test)

6098-3

APPENDIX III, TABLE 4

Significant Univariate Discriminators of Post-Treatment Performance
(Run 6 - Initial Variable Pool = 72)

Variable	Treatment means				F-ratios	Significance level (p)*
	Placebo	Marijuana	Alcohol	Both		
COGNIT.....	4.45	4.43	4.18	4.90	3.11	.032
ATTEMPTS.....	1.25	2.07†	1.76	2.15†	3.79	.014
ESTCHIC.....	21.60	20.86	21.24	19.00†	2.84	.044
CTT.....	5.04	5.20	4.72	4.67	2.43	.073
RISK.....	5.95	4.21	4.88	4.35	2.71	.052
OVERALL.....	0.35	0.36	0.76	1.05	2.88	.042
RISKCH.....	5.00	3.79	4.24	3.80	4.32	.008
SELFRATE.....	0.45	0.57	0.65	2.20†	8.20	.000
BIDET.....	0.15	0.64	0.47	0.15	2.35	.081
BISSPEED.....	0.00	0.36	0.76†	0.85†	3.28	.026

*$p \leq .10$; $df = \frac{3}{67}$

† Treatment vs. placebo contrast significant at $p \leq .05$ (Bonferonni t test)

6098-4

APPENDIX III, TABLE 5

Significant Univariate Discriminators of Post-Treatment Performance
(Run Total 3-6 - Initial Variable Pool = 75)

Variable	Treatment means				F-ratios	Significance level (p)*
	Placebo	Marijuana	Alcohol	Both		
BSPEED.....	-0.89	-0.26	0.16†	0.35†	2.32	.081
POSTOP.....	4.55	5.59†	5.53†	4.95	3.47	.020
ESTCHIC.....	20.38	19.59	20.18	17.67†	3.99	.011
SMPH.....	34.93	36.69†	36.32	38.16†	3.29	.025
CTT.....	4.98	5.01	4.49	4.45	4.65	.005
ERRORS.....	7.80	8.84	9.59†	12.91†	3.61	.017
BSTEER.....	0.97	0.34	0.41	2.22†	2.43	.071
FLREMIND.....	1.89	2.00	1.91	1.97	2.30	.084
OVERALL.....	0.53	1.09	1.29	1.79†	5.33	.002
UTURNS.....	0.85	1.09	1.04	1.35	2.23	.092
POSTOP.....	4.55	5.59†	5.53†	4.95	3.47	.020
ESTEER.....	0.79	0.89	1.09	1.55†	2.90	.040
ESTOP.....	0.28	0.36	0.65	0.37	2.23	.092
DETOUR.....	0.31	0.66	0.36	0.79	3.31	.024
SSPEED.....	0.60	0.71	0.86	1.22	2.74	.049
SLANEPOS.....	0.31	0.42	0.40	1.00†	3.80	.013
OFFRATE.....	1.05	1.37	1.48	1.88†	3.00	.035
SELFRATE.....	1.33	1.79	1.99	3.39†	7.95	.000
CTT4.....	4.91	4.93	4.31	4.19†	8.08	.000
CTT5.....	4.95	5.11	4.55	4.47	3.93	.011
CTT6.....	5.04	5.13	4.74	4.68	2.33	.080
BITE5.....	58.20	57.26	61.90†	49.70	2.46	.070

*$p \leq .10$; $df = \frac{3}{78}$

† Treatment vs. placebo contrast significant at $p \leq .05$ (Bonferonni t test)

6098-5