

alcohol on adult-child interactions were to be studied. All of the subjects participated in a 20-minute interaction with a child whom they thought had been similarly recruited for the study but who was actually a child actor. In half of the conditions, the child actor enacted a role characteristic of an ADD/CD child, and in half, the child enacted a normal child role. Prior to the interaction, half of the adults drank a sufficient amount of ethanol to raise their BAL to 0.05 and half drank a nonalcoholic beverage. The interactions were videotaped and scored to examine the nature of the strategies that the adults used to control the child's behavior. In addition, individual characteristics of the adults were examined to predict the nature of their interactions with the children.

RISK FOR ALCOHOLISM: PSYCHOPHYSIOLOGICAL HYPERREACTIVITY TO AVERSIVE AND NONAVERSIVE STIMULATION. Peter R. Finn, Jordan B. Peterson and Robert O. Pihl. McGill University, Montreal, Quebec, Canada.

Previous research has shown that men with multigenerational family histories (MFH) of alcoholism are autonomically hyperreactive to unavoidable shock and more sensitive to the reactivity dampening effects of alcohol when compared to controls. The present study was designed to test the hypothesis that MFH men are hyperreactive to stimulation in general, reflected in their reactivity to avoidable and unavoidable aversive stimulation and nonaversive 'orienting' tones. The electrodermal orienting response to tones and the autonomic (cardiovascular, electrodermal and muscle tension) response to a shock delivery procedure was measured under alcohol and no alcohol consumption conditions in MFH men and family history negative (FH-) men. The results showed a consistent pattern of autonomic hyperreactivity to all stimulus conditions, and a consistent pattern of reactivity dampening in MFH men only. The MFH men's hyperreactivity to stimulation is hypothesized to reflect a centrally mediated dysfunction in the modulation of sensory responsivity. The effect of alcohol in MFH men may reinforce drinking as a way to normalize responsivity, and this may promote excessive alcohol intake.

MANIPULATING EXPECTANCIES AS A MEANS OF ALTERING ALCOHOL CONSUMPTION. Renelle F. Massey and Mark S. Goldman. University of South Florida, Tampa, FL.

Researchers report a close relationship between alcohol expectancies (i.e., beliefs that alcohol makes people sociable, brave, sexy, etc.) and levels of alcohol use/abuse. To test experimentally the theoretical utility of expectancy as an intervening variable, a program designed to alter expectancies was developed which included a drinking experience. Reduction of alcohol consumption as a result of this program was compared with a state-of-the-art program and no treatment. Alcohol consumption significantly decreased in the month following participation in the expectancy program, but not in the control and traditional alcohol abuse prevention programs. Expectancy theory may have significant potential for alcohol abuse prevention.

EFFECT OF THE WAITING LIST ON ADMISSION TO DRUG TREATMENT. John L. Black and Michael P. Dolan. Dallas VA Medical Center; Hugh Tenison. Terrell State Hospital, Terrell, TX; and Jan Blumentrit. North Texas State University, Denton, TX.

Although drug abuse is a growing problem with increasing demand for drug treatment, limited treatment resources necessitate that clients wait for admission. This study assessed the effect of waiting for drug treatment. Applicants to a drug abuse treatment program were monitored over a 12-month period. Of the 587 applicants, 58.6% were admitted. The most common reason for nonadmission was inability to reach the applicant (19.4%), followed by the applicant not reporting for the scheduled screening (11.6%). Procedures designed to increase treatment admissions from waiting lists were identified.

THE USE OF MULTIPLE DEPENDENT VARIABLES TO CHARACTERIZE DRUG EFFECTS. Adelbert W. Price, Richard R. McKnight, Judy M. Plaisance and Reginald V. Fant. Nicholls State University, Thibodaux, LA.

Response rate, force of response, and an index of response chain performance were employed in an assessment of the effects of a stimulant (*d*-amphetamine), a depressant (pentobarbital), and a neuroleptic drug (pimozide). While all three drugs produced significant ($p \leq 0.05$) dose-related decrements in responding, the use of the additional behavioral measures allowed for a clear discrimination among the effects of these drugs. Pentobarbital and pimozide, but not *d*-amphetamine, produced significant ($p \leq 0.05$) alterations of the chaining index. Only pentobarbital significantly ($p \leq 0.05$) altered the force of responses. These data clearly support the use of multiple behavioral measures to characterize the effects of psychoactive drugs.

HALOPERIDOL BLOCKS REACQUISITION OF AN OPERANT DURING ONE-TRIAL LEARNING. Joseph H. Porter, Jenny L. Wiley and William R. Faw. Virginia Commonwealth University, Richmond, VA.

After acquisition training to traverse a straight runway for ten 45 mg food pellets (single trial/day), food-deprived rats ($n=8$ /group) were tested without food reward until running latencies met an extinction criterion. Then, a single priming trial with food reward was conducted with four groups receiving 0.03, 0.10 or 0.30 mg/kg haloperidol or vehicle (V+F) injections. A fifth group received vehicle but no food reward (V+E). The food prime resulted in reacquisition of operant running on the following test day for the V+F group; however, haloperidol blocked this effect. Thus, haloperidol blocked the incentive motivational properties of the food reinforcement.

HOW HALOPERIDOL SLOWS FIXED-RATIO RESPONDING: A QUANTITATIVE TOUR DE FORCE. Stephen C. Fowler and Ruey-Ming Liao. University of Mississippi, University, MS.

Hungry rats were trained to grasp and pull a wire ball attached to a force transducer, and after response rates had stabilized on a fixed-ratio 20 schedule of liquid-food reinforcement, effects of low doses of haloperidol (0.04, 0.08, 0.16 mg/kg) were evaluated. A combination of quantitative methods, including a new procedure for quantifying key attributes of cumulative records, revealed that haloperidol reduced rate of response in three ways (in increasing order of importance): it slowed the high-speed contractions of the forelimb that occur during the ratio run; it lengthened the postreinforcement time; and it caused an abrupt cessation in responding before the session ended. All three effects were

observed at the lowest dose tested. Response slowing reflected the temporal domain (as opposed to the force domain) motor effects of low doses of haloperidol.

THE EFFECTS OF CODEINE ON AGGRESSION: A TIME COURSE STUDY. Ralph Spiga, Don R. Cherek and John D. Roache. University of Texas Health Sciences Center, Houston, TX.

The effects of codeine on aggressive responding were studied in a controlled laboratory condition. Aggressive responding was defined as the subject subtracting points from an ostensible person. Aggressive responding was provoked by a fictitious person when they subtracted points exchangeable for money from the subject. A nonaggressive response option was monetarily reinforced. Codeine at all doses diminished aggressive responding relative to placebo. While codeine had no effect on nonaggressive responding at lower doses (25 mg/70 kg and 50 mg/kg), at the highest dose codeine increased nonaggressive responding compared to placebo.

CROSS-TOLERANCE AND SENSITIVITY TO OPIOIDS IN MORPHINE-TREATED PIGEONS. Rebecca M. Craft, Mitchell J. Picker and Linda A. Dykstra. University of North Carolina at Chapel Hill, Chapel Hill, NC.

Response-rate decreasing effects of several opioid agonists were determined in pigeons responding under a fixed-ratio 30 schedule of food presentation. Following determination of acute dose-effect curves, pigeons were injected once daily with 56 mg/kg morphine, resulting in a 1-log rightward shift in the morphine, *l*-methadone and ethylketocyclazocine dose-effect curves. In contrast, the cyclazocine and bremazocine curves were shifted to the left, whereas the U50,488 curve remained unchanged. Results suggest that in morphine-tolerant pigeons, morphine, *l*-methadone and ethylketocyclazocine share mu agonist properties, cyclazocine and bremazocine share mu antagonist properties, and U50,488 effects are unrelated to mu opioid receptor activity.

CROSS-TOLERANCE TO OPIOIDS IN MORPHINE-TREATED ANIMALS. Pamela Doty, Mitchell J. Picker and Linda A. Dykstra. University of North Carolina at Chapel Hill, Chapel Hill, NC.

Cross-tolerance to various mu and kappa opioid agonists was evaluated in morphine-tolerant squirrel monkeys using cumulative dosing procedures. Lever-press responding was maintained under a multiple FR30 schedule of food presentation. In monkeys given 3.0 mg/kg of morphine twice daily, the dose-effect curve for the rate-decreasing effects of morphine shifted 1/2 to 3/4 log unit to the right. Dose-effect curves for *l*-methadone, ethylketazocine (EKC) and U50,488 were determined prior to and during the chronic regimen. Results indicated approximately a 1/2 log unit shift to the right for the *l*-methadone dose-effect curve and no shift for EKC or U50,488.

TOLERANCE TO A MORPHINE CUE: ROLE OF MORPHINE MAINTENANCE DOSE. Elizabeth S. Steigerwald. Wayne State University, Detroit, MI; Christine A. Sanerud. The Johns Hopkins University School of Medicine, Baltimore, MD; and William J. Lipinski, Mechele D. Doty and Alice M. Young. Wayne State University, Detroit, MI.

Experiments examined the ability of several chronic doses of morphine to confer tolerance to a morphine discriminative stimulus. Rats were trained to discriminate saline and 3.2 mg/kg morphine under RF schedules of food delivery. Morphine generalization gradients were determined before, during and after chronic drug treatment. For chronic treatment, separate groups of rats received saline or selected doses of morphine for 14 to 18 day periods while discrimination training was suspended. A final group received 36 mg/kg/day pentobarbital. Repeated administration of saline, 10 mg/kg morphine, or 36 mg/kg pentobarbital produced no tolerance. In contrast, 36 and 100 mg/kg morphine produced marked tolerance to the morphine cue, albeit accompanied by marked suppression of response rates. It appears that the magnitude of tolerance developed to a morphine cue is dependent on the maintenance dose employed for chronic treatment.

COMBINATION OF BUPRENORPHINE WITH NALOXONE IN HUMANS. Linda L. Weinhold, George E. Bigelow and Kenzie L. Preston. The Johns Hopkins University School of Medicine, Baltimore, MD.

(Abstract not available)

DISCRIMINATIVE PROPERTIES OF BREMAZOCINE AND FENTANYL IN PIGEONS. Mitchell J. Picker and Linda A. Dykstra. University of North Carolina at Chapel Hill, Chapel Hill, NC.

Pigeons were trained to discriminate a dose of either bremazocine or fentanyl from water using a two-key drug discrimination procedure. During substitution tests, the kappa agonists bremazocine, U50,488 and tifluadom produced bremazocine-like but not fentanyl-like stimulus effects. The kappa agonists, ethylketocyclazocine, ketocyclazocine and nalorphine, and the mu agonists, fentanyl and morphine, produced fentanyl-like but not bremazocine-like stimulus effects. During tests of antagonism, the A50 doses of naloxone or Mr2266 in combination with the training dose of bremazocine were approximately equivalent, whereas in fentanyl-trained pigeons, the A50 dose of naloxone was approximately 1 log-unit smaller than the A50 dose of Mr2266.

ROLE OF DOPAMINE IN THE EFFECTS OF PENTAZOCINE AND TRIPELENNAMINE. Thomas J. Hudzik and Barbara L. Slifer. University of New Orleans-Lakeside, New Orleans, LA.

The present study was designed to identify the role of dopaminergic mechanisms in the mediation of the effects of pentazocine and tripeleNNamine. Utilizing milk intake as the dependent variable, dose-effect curves were constructed for these drugs in both the absence and presence of various dopaminergically active compounds. Pentazocine interacted in an additive manner with haloperidol and was antagonized by raclopride, while the effects of tripeleNNamine failed to be modified in a manner consistent with its hypothesized dopaminergic activity. These data lend further support to the notion that pentazocine may exert some of its effects via the dopaminergic system.

DO KAPPA EFFECTS EXPLAIN "T'S & BLUES"? Lynn A. Cones and Barbara L. Slifer. University of New Orleans-Lakeside, New Orleans, LA.