methadone treatment. Drug usage was monitored in follow-up by weekly urine surveillance until illicit drug use returned to 50% over a 60-day period. Half of the participants remained abstinent for at least 30 days after completing the contract (M=101.5 days, SD=188.4 days). Latency to relapse was at least 100 days for 13 of the 20 participants (M=230.9 days, SD=213.5 days).

NICOTINE EFFECTS ON HUMAN AVOIDANCE RESPONDING. Don R. Cherek, Joel L. Steinberg. Dept. of Psychiatry, Louisiana State University Medical Center; Thomas H. Kelley. Veterans Administration Medical Center, Shreveport, LA; Neal L. Benowitz. Dept. of Medicine, University of California, San Francisco.

Male subjects were administered nicotine gum or took varying numbers of puffs on research cigarettes which delivered either 0.42 or 2.14 mg of nicotine prior to sessions. During sessions, lever pressing was maintained by avoidance of point loss (1 point=10 cents) on a free-operant avoidance schedule. Nicotine gum (2–8 mg) resulted in no change in avoidance responding in some subjects. Puffs on high nicotine cigarettes produced increased avoidance responding in all subjects. The behavioral effects of nicotine were determined by historical exposure levels and not nicotine blood levels.

BRAIN SITES INVOLVED IN THE BEHAVIORAL EFFECTS ON INTRAVENTRICULARLY ADMINISTERED (−)-NICOTINE. Victor J. DeNoble and Paul C. Mele. Virginia Commonwealth University.

Fifteen hooded rats were trained to lever press for food under a fixed ratio (FR) 32 schedule. All rats were implanted with two cannulae, one in the lateral ventricle (LV) and the second in one of the following brain structures: dorsal hippocampus (DH), locus ceruleus (LC), lateral hypothalamus (LH), reticular formation (RF), or the vestibular nucleus (VN). All rats when infused with 5.0 μg (−)-nicotine (LV) or 0.25 μg in the VN showed an increased latency to complete the first ratio (mean=8.2±1.3 min). When lidocaine (5.0 μg) was applied to the RF the latency to complete the first ratio following 5.0 μg (−)-nicotine infusion into the LV was decreased by 55%. Lidocaine in the VN completely blocked the effect of LV (−)-nicotine. Neither lidocaine nor (−)-nicotine had any effect on responding when applied to the other brain structures.

MEMORY ENHANCEMENT WITH CLONIDINE IN SCHIZOPHRENIA. Robert B. Fields, Jules Rosen, Jeffrey Peters and Daniel P. Van Kammen. Veterans Administration Medical Center, Pittsburgh, PA.

This double-blind study examined the effect of clonidine, a central alpha$_2$ adrenergic agonist, on the memory functioning of hospitalized schizophrenics. Eight patients were tested while drug free and while on stable doses of clonidine (0.8 to 1.4 mg/day) for approximately five weeks. Memory test scores were significantly better during the clonidine trials. This difference could not be explained by changes in general cognitive functioning, sustained attention, or level of psychosis as no significant differences in these indices were found. The present findings are consistent with recent reports which suggest that clonidine may enhance memory functioning only when memory deficits exist.

OBJECTIVE AND SUBJECTIVE AGGRESSION MEASURES: EFFECTS OF ALCOHOL AND DIAZEPAM. Joel L. Steinberg, Don R. Cherek and Thomas H. Kelly. Dept. of Psychiatry, Louisiana State University Medical Center.

Diazepam was administered to nine normal human subjects to determine its acute effects on aggressive behavior in a behavioral laboratory setting. The subjects were adult males who were screened by psychiatric, physical, and laboratory examinations to be in good health and to exclude subjects with mental disorders or substance abuse. Aggressive behavior was defined as the delivery of an aversive stimulus to another person. Specifically, the subject would press a button on an FR 10 schedule which ostensibly subtracted money from a fictitious person with whom the subject was told he was paired. Aggressive responses were provoked by random subtractions of money from the subject which were attributed to the other person. Aggressive responses were maintained by a provocation-free interval (PFI), of 500 sec or 125 sec, according to an escape contingency. A non-aggressive response option was concurrently available by pressing a button to earn points exchangeable for money, on an FR 100 schedule. Diazepam was administered double blind in doses of 0, 2.5, 5, and 10 mg per 70 kg of body weight, in a repeated measures design in which each subject received each dose for three sessions. Eight subjects showed decreases in aggressive responses after 10 mg/70 kg diazepam, and one subject showed increases at this dose. Diazepam produced small and variable effects on non-aggressive, monetary reinforced responding which did not appear to correlate with the rate of aggressive responding, thus indicating a relatively specific effect on aggressive behavior.

At the end of each dose-response determination, the subjects were administered a single dose of 0.5 g/kg of alcohol. The correlation coefficient between the effects of alcohol and diazepam on aggressive responses in 11 subjects from two separate studies was 0.918 ($p<0.001$). The data suggest that subjects who exhibit increases in aggressive responses following alcohol administration appear likely to increase aggressive response following diazepam. Preliminary results also indicate that subjects who had low scores on the Buss-Durkee Hostility Inventory showed decreased aggressive responses after diazepam administration.

PHARMACOLOGICAL CHARACTERIZATION OF SUPERSENSITIVITY TO NALTREXONE IN SQUIRREL MONKEYS. Charles P. France and William H. Morse. Harvard Medical School.

Repeated weekly exposure to naltrexone was examined in squirrel monkeys responding under fixed-ratio schedules of food presentation or stimulus-shock termination. After six weekly naltrexone exposures the food-controlled (F) monkeys were 300- to 1000-fold more sensitive than the shock-controlled (S) monkeys to the rate-suppressing effects of naltrexone. The difference in sensitivity between F and S monkeys was 3- to 10-fold for MR 2266 and WIN 44,441, and at

least 100-fold for naloxone; there was no difference for nalorphine, morphine, U 50,488, or pentobarbital, suggesting supersensitivity in non-dependent squirrel monkeys results, in part, from opioid antagonist actions.

AMNESTIC PROPERTIES OF THE BENZODIAZEPINES. William T. Kirk and Roland R. Griffiths. Dept. of Psychiatry, Johns Hopkins University.

The cognitive, subjective and psychomotor effects of two benzodiazepines and pentobarbital were examined, at doses that were selected to produce comparable levels of sedation, in healthy, male volunteers recruited from the community at large. Results demonstrated a time-related impairment in psychomotor performance (eye-hand coordination and balance) following administration of active compounds which were reflected in the subjective ratings of drug effects. Additional performance deficits in picture recognition and number recall were observed suggesting that these compounds have amnestic as well as sedative properties.

PHYSOSTIGMINE-INDUCED ANALGESIA IN MATURE AND SENESCENT RATS. Janet S. Knisely. Dept. of Pharmacology and Toxicology, Medical College of Virginia; and Robert J. Hamm. Virginia Commonwealth University.

To investigate the role of the cholinergic system in the production of analgesia during aging, rats (3-month, 17-month and 25-month) were injected with physostigmine (0.015625, 0.0625 or 0.25 mg/kg). Before drug administration, baseline pain sensitivity was assessed using three tail-flick trials. Following the injections, tail-flick latencies were measured at 5 minute intervals for 30 minutes and at 45, 60, 75 and 90 minutes. Post-drug tail-flick latencies were converted to percent maximum possible effect (% MPE) and were analyzed by a 3(Age) × 4(Dose) × 11(Time) analysis of variance. The analysis revealed no age-related change in physostigmine-induced analgesia however, there were main effects of Dose and Time and all interactions were significant ($p<0.001$) except Age × Dose. Thus, increasing the dose of physostigmine enhanced analgesia and the analgesia displayed, varied across time. A lack of age-related differences in analgesia produced by physostigmine is in agreement with other research which has demonstrated that stimulation of the cholinergic system produces an equivalent or increased pharmacological responsivity in aged animals.

AUTONOMIC HYPER-REACTIVITY, SENSITIVITY TO ALCOHOL AND GENETIC RISK FOR ALCOHOLISM. Peter R. Finn and Robert O. Pihl. McGill University.

A genetic predisposition in the etiology of alcoholism in some individuals is indicated from adoption studies of the sons of alcoholics. A high risk paradigm was used to compare the degree of autonomic nervous system (ANS) reactivity to signalled shock and the effect of alcohol on ANS reactivity in 3 groups (high, moderate and low risk) of 12 non-alcoholic males divided according to the extent of family history for alcoholism. The high risk subjects were significantly more reactive to the shock procedure on cardiovascular and electrodermal measures when sober, and alcohol significantly reduced their reactivity more so than the other two groups.

The methodology and results of this study have relevance for (1) the etiology of alcoholism in high risk males, (2) high risk paradigms in alcohol research, (3) tension reduction models of alcohol consumption.

VASOPRESSIN ENHANCES MEMORY FOR PROSE. Bill E. Beckwith, Thomas V. Petros, Paula Bergloff and Robin Staebler. University of North Dakota.

The effects of treatment with DDAVP on memory in healthy and adult human males was investigated. Each subject received 60 micrograms of DDAVP intranasally and then heard six narrative passages of prose presented at differing rates of presentation. Proportion of recall was measured at high, medium, and low levels of importance of idea units within the passage. Treatment with DDAVP facilitated recall for both high and medium importance idea units. Treatment did not interact with rate of presentation. These findings provide further evidence for the modest facilitation provided by acute administration of DDAVP on human memory.

THE THEORETICAL MODEL: AROUSAL, COERCION AND THERAPY AS PREVENTION METHODS. Arthur P. Sullivan. New York City Board of Education, New York, NY; Robert Guglielmo. NYC Family Court Mental Health Services, New York, NY; and Roxane Polak. Hofstra University.

Substance use is taxonomized into psychologically adaptive use (experimental and recreational) and maladaptive (abuse and addictive). Descriptors which differentiate persons who use substances abusively or addictively from those who do not and seem characteristically resistant are examined. Etiological considerations are used to construct three methods for preventing, in the sense of lessening the likelihood or intensity of, abuse and addiction. *Coercive* methods are proposed for the immature and unintelligent, consisting primarily in using group enforcement procedures to enforce drug-free norms imposed on the group from without. *Arousal* methods are suggested for those whose distress from which relief is sought in substance use is primarily environmental. *Therapy* or counseling procedures are recommended for those suffering imperceptible distress of which they do not become aware until the drug experience brings immediate but brief respite. Distress caused by inadequate self esteem is explored in terms of origin, course of treatment, and prognosis.

PEER-GROUP COUNSELING TO PREVENT SUBSTANCE ABUSE. Barbara A. Taylor. Lord Stirling School, Glen Ridge, NJ.

The New York Model for drug prevention is a multiphasic, multi-level program which includes primary and junior high school classroom education programs, with peer-group counseling for selected students. Peer-group counseling, led by trained personnel, can be an effective tool in preventing substance abuse in students who have personal, school, social or family difficulties. Initially, these students often feel vulnerable and inadequate in facing difficult situations so they often avoid any uncomfortable experiences (classwork, competition). They use maladaptive