conclusively, particularly with regard to systematic observations (rather than ratings) of aggressive acts. Second, whereas stimulant medication leads to increases in social compliance, the question of its effects on explicitly prosocial behavior (e.g., social initiation or leadership) is indeterminate. To help resolve such questions, we recently investigated stimulant effects on (a) compliant vs. prosocial behaviors and (b) disruptive vs. aggressive behaviors in a naturalistic summer research program for boys with ADD. The 25 participants, aged 6–12, were placed on a double-blind trial of placebo, 0.3 mg/kg or 0.6 mg/kg methylphenidate hydrochloride over a 3-week period, in a within subjects/crossover fashion, with each dosage lasting for one week. Scan-sampling procedures were used to record ongoing social behavior in naturalistic classroom and playground settings, and reliable observations of the distinct social behaviors in question were made. Clear medication effects on aggression were found. An overall main effect was clearly significant, and individual response patterns showed that all subjects clinically appraised as both hyperactive and aggressive showed at least a 50% reduction of aggression on medication. Interestingly, for only several subjects did the 0.6 mg/kg dosage provide any greater benefit than did the low (0.3 mg/kg) dose. Effects on prosocial behavior were less striking, although for certain older (ages 9–12) boys who had presumably learned basic prosocial skills previously, medication effects were dramatic. The clinical and methodological significance of these findings include the importance of analysis of individual response patterns in addition to group means.

METHYLPHENIDATE AND ADD CHILDREN'S SOCIAL BEHAVIOR: INDIVIDUAL DIFFERENCES, DOSE RESPONSE. William E. Pelham, Jr. Western Psychiatric Institute and Clinic, University of Pittsburgh School of Medicine, Pittsburgh, PA.

A number of studies have evaluated the dose effects of psychostimulants on ecologically valid measures of functioning in Attention Deficit Disorder (ADD) children. Typical studies have included evaluations of doses ranging from 0.15 to 0.75 mg/kg methylphenidate (MPH) or equivalent, and they have focused on a variety of dependent measures, tapping both social and cognitive spheres. Despite relatively similar dose ranges and dependent measures, however, these studies have yielded somewhat inconsistent results. For example, some report only linear effects of stimulants on measures of cognitive functioning, while others report that beneficial stimulant effects peak at relatively low doses. Similarly, some studies report beneficial stimulant effects on ADD children's social behaviors, both prosocial and antisocial, but other studies have failed to support these findings. It is the thesis of this paper that the discrepancies result from individual differences in drug effects, both across children within response domains and across dependent measures within children. Data to support this argument are reported for 17 ADD children who underwent a within-subject evaluation of placebo, 0.3 and 0.6 mg/kg MPH, with dose randomized over days. Dependent measures were gathered over 5 to 9 days per condition, and included observations of classroom and playground behavior, measures of classroom seat work, mathematics and reading performance (completion rate and accuracy), and frequency counts of positive peer interactions, negative verbalizations, conduct problems, rule-following behavior and noncompliance to adult requests. Analyses of group data consistently revealed linear effects of MPH, within the dose range evaluated, across most dependent measures. However, very few children exhibited the linear trends that characterized the group data. On some dependent measures, not a single child's data were reflected in the group mean. Furthermore, the shapes of the dose-response curves across the dependent measures consistently varied within children. The results have implications for psychopharmacology with ADD children, particularly regarding the notion of a "medication responder."

INVITED ADDRESS

Substance Abuse Treatment and Policy: Contributions of Behavioral Pharmacology

Thomas J. Crowley, University of Colorado Health Sciences Center, Denver, CO

Chair: *John Grabowski,* University of Texas Health Sciences Center, Houston, TX

SYMPOSIUM

Progress in Understanding the Behavioral and Neurobiological Effects of Cocaine

Chair: *Steven I. Dworkin,* Louisiana State University School of Medicine, Shreveport, LA

Discussant: *John Grabowski,* University of Texas Health Sciences Center, Houston, TX

LONG-TERM EXPOSURE TO COCAINE: OVERVIEW AND SOME CURRENT DATA. William L. Woolverton. University of Chicago, Chicago, IL.

(Abstract not available)

REPEATED COCAINE ADMINISTRATION AND SCHEDULE-CONTROLLED BEHAVIOR. Marc N. Branch. University of Florida, Gainesville, FL.

Recent research has shown that effects of repeated cocaine administration can be altered by behavioral variables. Among the recently discovered important factors are the parameters of schedules of positive reinforcement. Tolerance to effects of repeated cocaine administration has been found to depend strongly on the parameter value of fixed-ratio schedules, but not at all on the parameter value of fixed-interval schedules. Variable-ratio and variable-interval schedules have produced intermediate effects, i.e., for some subjects, tolerance has been schedule-parameter dependent, whereas for others, it has not.

REINFORCING AND CARDIOVASCULAR EFFECTS OF COCAINE IN MONKEYS AND HUMANS. Charles W. Schindler, Jonathan R. Katz, Steven R. Goldberg, Ro Nemeth-Coslett and Jack E. Henningfield. National Institute on Drug Abuse Addiction Research Center, Baltimore, MD.

The effects of IV cocaine administration were determined in both squirrel monkeys and human volunteers. In monkeys, the ability of cocaine to maintain self-administration behavior and the cardiovascular effects of cocaine were determined in separate groups. In humans, self-administration and cardiovascular measures were taken concurrently. In monkeys, where higher doses could be administered, cocaine produced significant increases in blood pressure at