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NEUROPSYCHOLOGICAL DEFICITS IN CHRONIC COCAINE ABUSERS

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A basic neuropsychological assessment battery was given to thirty-seven chronic freebase cocaine ("crack") abusers. The following tests were used: Wechsler Memory Scale (Wechsler, 1945), Rey-Osterrieth Complex Figure (Osterrieth, 1944) (copy and immediate reproduction), Verbal Fluency (semantic and phonologic), Boston Naming Test (Goodglass, Kaplan, & Weintrab, 1983), Wisconsin Card Sorting Test (Heaton, 1981) and Digit-symbol from the WISC (Wechsler, 1974). In general, performance was lower than expected according to their age and eductational level. Subjects showed significant impairment in short-term verbal memory and attention subtests. Neuropsychological test scores were correlated with lifetime amount of cocaine used, suggesting a direct relationship between cocaine abuse and cognitive impairment. A pattern of cognitive decline is proposed.

Keywords: Cocaine abuse; neuropsychology.

During the last several years, a vast importance has been given to the medical and social analysis of drug abuse (e.g., Pérez, 1987). This has been particularly true with regard to the effects of alcohol, marijuana and LSD (Adams, Rennik, Schooff & Keegan, 1975; Carlin & Trupin, 1977; Fletcher et al., in press; Grant, Adams, Carlin & Rennik, 1977; Parsons & Farr, 1981; Satz, Fletcher & Sutker, 1976; Wright & Hogan, 1972). In general, these studies have shown the existence of some cognitive deficits with chronic drug abuse; these effects appear particularly evident with regard to memory, attention, and abstraction abilities (Bruhn & Maage, 1975; Bruhn et al., 1981; Carlin, 1986; Carlin, Strauss, Grant & Adams, 1978; Parsons & Farr, 1981). This has led to the proposal of the existence of a group of toxic dementias (e.g., Ardila & Rosselli, 1986; Marsden, 1985). Much evidence has already been established in regard to alcoholic dementia and its manifestations of impairments in short-term memory, attentional deficits, and spatial defects (Victor & Adams, 1985).

Research on the cognitive effects of the chronic use of alkaloids extracted from the coca leaf (*Erythroxylum coca*) has been, however, particularly scarce. The acute effects of cocaine on behavior are much better known. It seems clear that due to the stimulant effect of cocaine on the nervous system the subject displays euphoria, restlessness, and increased energy (Gawing & Ellinwood, 1988; Hartmann, 1988). Cocaine intoxication can also produce some psychiatric symptoms (Gawin & Kleber, 1986; Miller, Gold & Milman, 1989). Frequent users may develop panic attacks (Washton & Gold, 1984), paranoid ideation, depression, anxiety, and loss of motivation (Washton & Tatarsky, 1984); violent behavior is commonly seen also among cocaine patients (Manschreck et al., 1988). When given chronically cocaine can produce some be-

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havior disorders similar to those seen in schizophrenic psychosis (Wyatt, Fawcett, & Kirch, 1989).

Freebase cocaine ("crack") has been associated with cerebrovascular complication (Kaye & Fainstat, 1987; Levine et al., 1987; Golbe & Merkin, 1986). Cocaine intoxication can produce seizures, cardiac arhythmias, and respiratory arrest (Miller, Gold, & Milman, 1989). Although the exact mechanism of cocaine on the nervous system is not presently understood, it has been hypothesized that it produces a direct neurotoxin effect or multiple microinfarctions due to hemodynamic changes. Drowsiness, unsteady gait and seizures have been correlated with passive smoke inhalation in children exposed to the smoke of free cocaine used by their adult caretakers (Bateman & Heagarty, 1989). The fetal effects of "crack" with its use during pregnancy have been recently studied by Cherukuri et al. (1988).

The cognitive consequences of chronic cocaine use are less clearly defined. Adamse, O'Malley, Heaton and Gawin (unpublished) have suggested a correlation between the amount of cocaine consumed and the severity of cognitive impairment. Washton and Gold (1984) reported that 57% of the cocaine abusers interviewed experienced memory problems. Press (1983) found better (although nonsignificant) performance of normals when compared with cocaine abusers on the Luria-Nebraska Neuropsychological Battery. According to the author, verbal memory subtests were the most significantly impaired in cocaine abusers. Low scores in the range of impairment on Trials A and B have also been reported in freebase cocaine users (Reitan & Wolfson, 1985). Adamse et al. (unpublished) administered a neuropsychological screening battery to 20 chronic cocaine abusers and compared their performance with normal controls. The poorest performance was observed on the Symbol-Digit Modalities Test, Arithmetic Test and Story Memory. The authors suggested an underlying deficit in attention and memory.

The purpose of the present study was to analyze further the cognitive effects of chronic cocaine abuse.

METHOD

Subjects

Thirty-seven freebase cocaine ("crack") abusers (23 males and 14 females) with a mean age of 28.86 (SD = 5.18; range = 20–46) were studied. The subjects' mean educational level was 10.86 (SD = 1.20). All subjects met the criteria of substance dependence according to the DSM-III-R (1984) but were abstinent for approximately 30 days (mean 27.35) before the evaluation. They were in an inpatient rehabilitation program in Miami (Florida) at the moment of the neuropsychological testing. The subjects had selected "crack" as their main drug for 16.64 months (on average) before the neuropsychological evaluation. All subjects were considered heavy users (average 9.27 grams per week). All were polydrug users before using "crack". However, once they started using "crack" they did not use any other drug, except for marijuana occasionally. No subject presented a major positive neurological (head trauma, epilepsy, etc.), or psychiatric (DSM-III-R, 1984) disorder. At the time of the evaluation, no subject was taking any medication that might affect the central nervous system. Subjects were nonpaid volunteers.

Instruments

All subjects were assessed in abstraction, language, constructional and memory

abilities, using the following neuropsychological tests: (1). Wechsler Memory Scale (WMS) (Wechsler, 1945). It includes the following subtests: Information, Orientation, Mental Control, Digits, Logical Memory, Visual Reproduction and Associative Learning. (2). Rey-Osterrieth Complex Figure (Osterrieth, 1944). Copy and immediate reproduction. Taylor's scoring system was used (Lezak, 1983). (3). Verbal Fluency. Two different conditions were used: Semantic verbal fluency (animals), and phonological verbal fluency (FAS). (4). Boston Naming Test (Goodglass, Kaplan, & Weintraub, 1983). (5). Wisconsin Card Sorting Test (WCST) (Heaton, 1981). (6). Digit-symbol from the WISC-R (Wechsler, 1974).

Procedure

Subjects were interviewed to determine eligibility. Using a structured interview, subjects were asked about demographic data, developmental, family, and medical history, and history of substance abuse. Subjects who met the criteria for participation in the study were then administered a battery of neuropsychological tests. Administration time was approximately 90 minutes, divided into two sessions.

RESULTS

Results of the WMS, Rey-Osterrieth Complex Figure, and Verbal Fluency Tests, were compared both with the norms collected by Ardila, Rosselli, and Puente (in press), matching age and educational level, and with the norms presented by Lezak (1983). For the rest of the tests, normative data presented in the test manuals were used. Table I presents the results obtained on the WMS. Only for Information, Logical Memory and Associative Learning Subtests, the drug abuser group scores are one or more standard deviations below the normative group scores. Associative Learning subtest was the most impaired: the mean score in Associative Learning subtest is almost two standard deviations below the normative means used. For the cocaine-abuser group

TABLE I
Mean scores and standard deviations (in brackets) obtained in the Wechsler Memory Scale subtests

SUBTEST	COCAINE ABUSERS	NORMATIVE DATA (Ardila et al., in press)	(Lezak, 1983) (Wechsler, 1945)
Information	4.73(0.83)	5.30(0.56)	
Orientation	4.84(0.44)	4.80(0.50)	
Mental Control	5.43(2.26)	4.95(2.52)	
Logical Memory	6.59(1.96)	13.43(3.69)	9.28(3.10)
Digits	8.76(2.15)	9.05(2.22)	
Visual Reproduction	6.78(2.66)	8.25(2.70)	11.00(2.73)
Associative Learning	10.81(2.20)	15.60(3.25)	15.75(2.81)
Total	47.94	62.39	
Memory Quotient	84*		
	79**		

*According to Ardila et al.'s norms
**According to Wechsler's norms

TABLE II
Mean scores and standard deviations (in brackets) obtained in the neuropsychological tests

SUBTEST	COCAINE ABUSERS	NORMATIVE DATA (Ardila et al., in press)	(Lezak, 1983) (Goodglass et al., 1983)
Rey Figure (copy)	31.43(5.02)	29.33(4.06)	32.00
Rey Figure (memory)	18.36(6.55)	19.44(5.66)	22.00
Verbal Fluency (animals)	14.21(3.11)	14.03(4.72)	18.00
Verbal Fluency (FAS)	32.20(2.79)	34.26(4.30)	31-44
Boston Naming Test	49.94(6.03)		55.73(4.42)

the Memory Quotient calculated with regard to the normative data was one standard deviation below the expected normative score.

Table II presents the results for the Rey-Osterrieth Complex Figure, Verbal Fluency, and Boston Naming Tests. There were no significant differences between the experimental and normative group means in the copy of the Rey-Osterrieth Complex Figure, and Verbal Fluency subtests. However, in the immediate reproduction of the Rey-Osterrieth Complex Figure, and in the Boston Naming Test, the drug-abuser group scored about one standard deviation below the expected score.

The Digit-symbol subtest mean raw scores in the cocaine group was 41.45 (SD = 8.78) that corresponds in average to a scale score of 4 (mean = 10) for 16-year-old children. On the Wisconsin Card Sorting Test the mean total number of errors was 33.35 (SD = 21.75); the normals' mean is 21.60 errors (SD = 16.70) (Heaton, 1981).

The score in each of the subtests was correlated with lifetime cocaine use and with the estimated amount of drug intake. Table III presents these correlations. In general, correlations with lifetime use of crack were higher than correlations with weekly amount of cocaine use. There was a significant correlation between lifetime drug abuse and the scores in the Digits subtest of the WMS and Memory Quotient, and with the Rey-Osterrieth Complex Figure immediate reproduction condition. Amount intake correlated significantly only with Information subtest of the WMS, and with the Boston Naming test. Other correlations were lower and nonsignificant.

TABLE III
Correlations between cocaine use and scores in the neuropsychological subtests

Subtest	lifetime (in months)	p	amount	p
Information	−0.10	0.26	−0.29	0.04
Orientation	−0.24	0.07	0.18	0.13
Mental Control	−0.21	0.09	0.01	0.47
Logical Memory	−0.23	0.07	−0.14	0.19
Digits	−0.36	0.01	−0.08	0.29
Visual Reproduction	−0.01	0.49	−0.02	0.43
Associative Learning	−0.22	0.08	−0.20	0.10
Memory Quotient	−0.34	0.01	−0.07	0.32
Rey Figure (copy)	−0.15	0.17	−0.17	0.15
Rey Figure (memory)	−0.47	0.001	−0.17	0.15
Fluency (animals)	−0.07	0.33	−0.06	0.34
Fluency (FAS)	−0.15	0.25	0.10	0.26
Boston Naming Test	−0.14	0.19	−0.31	0.03
Digit Symbol	−0.22	0.08	−0.13	0.21
Wisconsin	−0.19	0.12	−0.16	0.16

DISCUSSION

Our results, although preliminary, point to a moderate but significant cognitive impairment associated with cocaine abuse. This cognitive impairment is reflected particularly in verbal memory (Logical Memory and Associative Learning) subtests, and to a lesser degree, nonverbal memory (immediate reproduction of the Rey-Osterrieth Complex Figure and Visual Reproduction from the WMS) tests. The Memory Quotient was significantly decreased in cocaine abusers, and negatively correlated with lifetime cocaine use; Digit-Symbol subtest scores were also significantly decreased. Abstracting ability (Wisconsin Card Sorting Test) and naming (Boston Naming Test) scores were about one standard deviation below the expected scores; while Verbal Fluency, Rey-Osterrieth Complex figure copy, and Information scores were virtually normal. This pattern of impairment is, in general, consistent with the impairment usually found in stimulant-abusers (Adams et al., 1975; Carlin, 1986; Carlin et al., 1977, 1978; Washton & Gold, 1984). The significant correlation observed with lifetime amount of cocaine use supports the assumption that cocaine is responsible for the lowered performance in the neuropsychological tests.

According to our results, freebase cocaine abuse, under the conditions of our sample, is associated with a moderate but significant cognitive impairment. However, this cognitive impairment follows a specific pattern: short-term verbal memory (Logical Memory, and Associative Learning substest, and Memory Quotient) and attentional (Digit-Symbol and Digits subtests) abilities are most sensitive, while abstracting (Wisconsin Card Sorting Test), naming (Boston Naming Test), short-term nonverbal memory (Visual Reproduction and Rey-Osterrieth Complex Figure, immediate reproduction) are less impaired than short-term verbal memory and attention. Verbal fluency (semantic and phonologic), constructional abilities (Rey-Osterrieth Complex Figure, copy), and long-term memory (Information subtest) are virtually unimpaired. It would be expected that changing the conditions of the sample (lower or higher amounts of cocaine intake, longer or shorter lifetime use, mixture with additional drugs) the level of cognitive impairment would be different, although the general pattern, similar.

Our results are consistent with previous research on neuropsychological effects of cocaine abuse. Press (1983) underlines the verbal memory deficits in cocaine abusers, while Adamse et al. (unpublished) suggest an underlying defect in concentration and memory. Low scores in attentional tasks such as the Trials tests have been also reported (Reitan & Wolfson, 1985). Consequently, it seems consistent that memory and attention are most senstive to chronic cocaine abuse, probably followed by abstraction and naming abilities. It is important to note that these are the general cognitive deficits usually found in chronic drug abusers (Bruhn & Maage, 1975; Bruhn et al., 1981; Carlin, 1986; Carlin et al., 1978; Parsons et al., 1981), eventually leading to a toxic dementia.

REFERENCES

Adams, K.M., Rennik, P., Schooff, K., & Keegan, J. (1975). Neuropsychological measurement of drug effects: polydrug research. *Journal of Psychodelic Drugs, 7*, 151–160.

Adamse, M., O'Malley, S., Heaton, R.K., & Gawin, F.H. (unpublished). Neuropsychological assessment in chronic cocaine abusers.

Ardila, A., & Rosselli, M. (1986). *Neuropsicología del fenómeno del envejecimiento.* Medellin (Colombia): Prensa Creativa.

Ardila, A., Rosselli, M., & Puente A. (in press). *Neuropsychological assessment: A clinical guide.* New York: Plenum.

Bateman, D.A., & Heagarty, M.C. (1989). Passive freebase cocaine (crack) inhalation by infants and toddlers. *American Journal of Disabled Children, 143*, 25–27.
Bruhn, P., Arlien-Soberg, P., Glydensted, C., & Christensen, E.L. (1981). Prognosis in chronic toxic encephalopathy. *Acta Neurologica Scandinavica, 64*, 259–272.
Bruhn, P., & Maage, N. (1975). Intellectual and neuropsychological functions in young men with heavy and long-term patterns of drug abuse. *American Journal of Psychiatry, 132*, 397–401.
Carlin, A.S. (1986). Neuropsychological consequences of drug abuse. In I. Grant & K.M. Adams (Eds.) *Neuropsychological assessment of neuropsychiatric disorders*. New York: Oxford University Press, pp. 478–497/
Carlin, A.S., Strauss, F.F., Grant, I., & Adams, K.M. (1978). Prediction of neuropsychological impairment in polydrug abuse patients. *Addictive Behavior, 5*, 229–234.
Carlin, A.S., & Trupin, E. (1977). The effects of long term chronic cannabis use on neuropsychological functioning. *International Journal of Addiction, 12:*, 617–624.
Cherukuri, R., Minkoff, H., Feldman, J., Parekh, A., & Glass, L. (1988). A cohort study of alkaloidal cocaine ("crack") in pregnancy. *Obstetric-Gynecology, 72*, 147–151.
Diagnostic Statistical Manual of Mental Disorders-III-R (1984). Washington: American Psychiatric Association.
Fletcher, J.M., Page, J.B., Francis, D.J., Krauskopf, D., Trup, W.P., & Satz, P. (in press). Cognitive effects of chronic cannabis abuse. *New England Journal of Medicine*.
Gawin, F.H., & Kleber, H. (1986). Abstinence symptomatology and psychiatric diagnosis in cocaine abusers. *Archives of General Psychiatry, 43*, 107–113.
Gawin, F.H., & Ellinwood, E.H. (1988). Cocaine and other stimulants. Action, abuse and treatment. *New England Journal of Medicine, 318*, 1173–1182.
Golbe, L.I., & Merkin, M.D. (1986). Cerebral infarction in a user of free-base cocaine ("crack"). *Neurology, 36*, 1602–1604.
Goodglass, H., Kaplan, E., & Weintraub, S. (1983). *Boston naming test*. Philadelphia: Lea Febiger.
Grant, I., Adams, H.K., Carlin, A.S., & Rennik, P.M. (1977). Neuropsychological deficits in polydrug abusers. *Drug and Alcohol Dependence, 2*, 91–108.
Hartmann, D.E. (1988). *Neuropsychological toxicology*. New York: Pergamon Press.
Heaton, R. (1981). *Winsconsin Card Sorting Test: Manual*. Odessa: Psychological Assessment Resources, Inc.
Kaye, B.R., & Fainstat, M. (1987). Cerebral vasculitis associated with cocaine abuse (letter). *JAMA, 257*, 1175.
Levine, S.R., Washington, J.M., Jefferson, M.F., Kiernan, S.W., Moen, M., Feit, H., & Welsh, K.M. (1987). Crack cocaine-associated stroke. *Neurology, 37*, 1849–1853.
Lezak, M.D. (1983). *Neuropsychological assessment*. New York: Oxford University Press.
Manschreck, T.C., Laughery, J.A., Weisstein, C.C., Allen, D., Humblestone, B., Neville, M., Podlewski, H., & Mitra, N. (1988). Characteristics of freebase cocaine phychosis. *Yale Journal of Biological Medicine, 61*, 115–122.
Marsden, C.D. (1985). Assessment of dementia. In J.A.M. Frederiks (Ed.) *Handbook of clinical neurology, vol. 46: Neurobehavioral disorders*. Amsterdam: Elsevier, pp. 221–232.
Miller, N.S., Gold, M.S., & Milman, R.L. (1989). Cocaine. *American Family Physician, 39*, 115–120.
Osterrieth, P.A. (1944). Le test de copie d'une figure complexe. *Archieves de psychologie, 30*, 206–356.
Parsons, E.S., & Farr, S.D. (1981). The neuropsychology of alcohol and drug abuse. In S.B. Filskov, T.S. Boll (Eds.), *Handbook of clinical neuropsychology, vol I*. New York: Wiley, pp. 320–365.
Pérez, A. (1987). *Cocaína: surgimiento y evolución de un mito*. Bogotá: Catálogo Cientifico.
Press, R.J. (1983). *The neuropsychological effects of cocaine and opiate use*. Ann Arbor: University Microfilms International.
Reitan, R.M., & Wolfson, D. (1985). *The Halstead-Reitan Neuropsychological Battery. Theory and Clinical Interpretation*. Tucson, Arizona: Neuropsychology Press.
Satz, P., Fletcher, J.M., & Sutker, L.S. (1976). Neuropsychological, intellectual, and personality correlates of chronic marihuana use in native Costa Ricans. In R. Dornbush, A.M. Freedman, & M. Fink (Eds.) *Chronic cannabis use. Annals of the New York Academy of Sciences, 282*, 266–306.
Victor, M., & Adams, D.R. (1985). The alcoholic dementias. In J.A.M. Frederiks (Ed.), *Handbook of clinical neurology, vol 46: Neurobehavioral disorders*. Amsterdam: Elsevier, pp. 335–350.
Washton, A.M., & Gold, M.S. (1984). Chronic cocaine abuse: Evidence and adverse effects on health and functioning. *Psychiatric Annals*, 733–743.
Washton, A.M., & Tatarsky, A. (1984). Adverse effects of cocaine abuse. *National Institute of Drug Abuse: Research Monograph Series, 49*, 247–254.
Wechsler, D. (1945). A standarized memory test for clinical use. *The Journal of Psychology, 19*, 87–95.
Wechsler, D. (1974). *WISC-R: Manual*. New York: Psychological Corporation.

Wright, M., & Hogan, T.P. (1972). Repeated LSD ingestion and performance on neuropsychological tests. *Journal of Nervous and Mental Disease, 154*, 432–438.

Wyatt, R.J., Fawcett, R., & Kirch, D. (1989). A possible animal model of defect state schizophrenia. In S.C. Schulz, & C.A. Tamminga (Eds.), *Schizophrenia: Scienctific progress*. New York: Oxford University Press, pp. 184–189.