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AUTHOR Horowitz, Sheldon
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ABSTRACT

The effects of alcohol on the developing fetus are examined. Noted is the existence of both structural problems (such as microcephaly and cardiac anomalies) and behavioral problems (such as mental retardation and speech and language deficits). The potential damage of alcohol at a very early stage of fetal development is discussed. It is thought that children without physical signs may nevertheless have potential learning difficulties demonstrated by a wide range of performance deficits. The author describes a current study of the cognitive, educational, and behavioral effects of alcohol in children of mothers who drank alcohol during pregnancy. (CL)

FETAL ALCOHOL EFFECTS IN CHILDREN:

COGNITIVE, EDUCATIONAL, and BEHAVIORAL CONSIDERATIONS

Presentation at 61st Annual CEC Convention, 1983

Submitted by: Sheldon Horowitz
Educational Supervisor
Department of Psychiatry
Center for Mental Health
Interfaith Medical Center
555 Prospect Place
Brooklyn, N. Y. 11238

The focus of this paper will be to overview alcohol effects, not from the perspective of the user/abuser, but rather from that of the victim. It has become clear over the past decade that early maternal alcohol consumption has the potential to cause (or at least contribute to) the increased incidence of certain traits and behaviors in offspring. These characteristics, appearing in clusters in the most severely affected offspring, have been called the Fetal Alcohol Syndrome, (Lemoine, Harousseau, Borteryu, and Menuet, 1968; Jones and Smith, 1973), and in lesser affected children as Fetal Alcohol Effects (Shaywitz, Cohen, and Shaywitz, 1980; Shaywitz, Caparulo, and Hodgson, 1981). This paper will survey the teratogenic effects of alcohol, making mention of both structural and behavioral findings from the current literature.

A teratogen may be defined as a substance that can adversely affect fetal development and, in many cases, present a recognizable pattern of dysmorphic conditions and suspect behavioral effects. Structural teratogens such as thalidomide, dilantin, (Hanson and Smith, 1975), trimethadione, (Zackai, Mellman, Neiderer, and Hanson, 1975), and Warfarin, (Shaul and Hall, 1977) are known to have potentially damaging effects on the developing fetus. Cigarettes

have been indicted as having the potential to contribute both to the abnormal physiological development of the unborn child and to a delay or deficit in the neurodevelopmental integrity of the maturing central nervous system (Holsclaw and Topham, 1978; Dunn, McBurney, Ingram and Hunter, 1977). Cigarette smoking in combination with alcohol abuse has been further implicated in recent findings that suggest that not only do these habits coincide with great frequency, but that there may be a close parallel relationship to the use of caffeine and illicit drugs in these same individuals (Kuzma and Kissinger, 1981). Behavioral teratogens, or substances that have been said to cause changes in behavior in the absence of gross structural damage, may include methadone, (Hall, Panti, and Wilson, 1980), amphetamines, (Mulkovich and van den Berg, 1977), L.S.D., (Cohen and Shiloh, 1977-78), and heroin, (Stone, 1971). In question is also the potential risk of excessive exposure to x-ray (Brent, 1980), and maternal hypertermia, (Fleet, Graham, Harvey, and Smith, 1980).

Alcohol is, in many ways, unique in its teratogenic status. While findings are often confounded by multiple substance abuse, poor nutrition, stress, and any number of other variables, there seems to be a general consensus that both structural and behavioral characteristics may be attributable to early alcohol abuse. Structural features, including microcephaly, shortened palpebral fissures, joint, limb, and cardiac anomalies, and thin vermillion with inconspicuous or absent philtrum have been cited,

(Lemoine, Harousseau, Borteryu, and Menuet, 1968; Hanson, Streissguth, and Smith, 1978), and are almost always accompanied by interuterine and postnatal growth deficiency, (Golden, Sokol, Kuhnert and Bottoms, 1982). Behavioral characteristics of infants born to alcohol involved mothers include failure to thrive and decreased alertness (Landesman-Dwyer, Keller, and Streissguth, 1978), weak sucking (Martin, Martin, Streissguth and Lund, 1979), and retarded motor development (Golden, Sokol, Huhnert, and Bottoms, 1982). Behavoral and cognitive impairments, most notably mental retardation, (Streissguth, Herman, and Smith, 1978), speech and language deficits (Iosub, Fuchs, Bingol, and Gromisch, 1981) and moderate to severe learning difficulties (Shaywitz, Cohen, and Shaywitz, 1980; Shaywitz, Caparulo and Hodgson, 1981) are almost always found to accompany structural features, and, in an increasingly large number of cases, are being noted in the absence of dysmorphic identfying features.

It is virtually impossible to account for the wide range of variables that effect measurement outcomes in studies using human subjects. This is especially true in the case of alcohol use as much of the qualitative and descriptive data required are collected by verbal report, and are often provided by persons who, given histories of general deprivation and/or multiple substance abuse, are less than reliable sources of information. While alcohol has been indicted as the major cause of teratogenic effects in children, maternal variables such as age, liquid

intake, use of medication, and exposure to caffeine and cigarettes must be considered as potential risk factors. Obstetric factors, sensitivity to foods and heat, response to stress, and metabolic and hormonal levels have been cited as potential risk factors for some women. Majewski, (1981), noting these risk factors, further suggested that the incidence and severity of embryopathy was closely related to the type of alcohol consumed and the stage of maternal alcoholic involvement. A recent study. (Fisher, Atkinson, Burnap, Jacobson, Sehgal, Scott, and Van Thiel, 1982), reported that many of the features of FAS may be attributable to a restriction in the placental transfer of nutrients essential for fetal growth, resulting in an ethanol-associated selective fetal malnutrition. This finding is supported by independent studies of the effects of thiocynate, a substance common in beer, (Bottoms, Judge, Kuhnert, and Sokol, 1982), and high and low protein diets in combination with alcohol, (Weiner, Shoemaker, Koda, and Bloom, 1981). In both of these investigations, selective malnutrition was posited to be a corollary to already identifiable teratological alcohol effects.

One of the first attempts to control and document the effects of in utero alcohol exposure was offered by Kathleen Sulik (1981). Using seven day old mice (corresponding to the 3rd week of gestation in humans), she was able to replicate the development of many of the same anomalous physiological

features that were observed in human (i.e. shortened palpebral fissures, absent philtrum, small cranial size) as well as, such neurological findings as abnormal ventrical size and agenesis corpus callosum. This study is especially remarkable in that it indictes alcohol as a potentially damaging substance at so early a time during pregnancy. Many women are not aware that they are pregnant three weeks after conception.

Mental retardation is clearly the most disabling and frequently reported consequence of excessive maternal alcohol consumption, yet studies vary considerably in their accounts of mean and range I.Q. scores (Shaywitz, Cohen and Shaywitz, 1980; Streissguth, Barr, Martin, and Herman, 1980; Darby, Streissguth, and Smith, 1981). Cited to be among the most commonly recognized neurological disorders along with Down's Syndrome, and spina bifida, (Abel, 1980), I.Q. scores of FAS children tend to remain stable (Streissguth, Herman, and Smith, 1976), despite environmental enrichment and foster home placements (Jones, Smith, Streissguth, and Myrianthopoulos, 1974).

While there is little doubt that early maternal alcohol abuse can result in moderate to severe learning impairment in children, few efforts have been made to describe the characteristics of children who do not manifest the severe dysmorphic features of FAS but who are the offspring of alcohol abusive mothers. These FAE children in many ways resemble a population of youngsters who are referred to as learning disabled in that they present a wide range of performance deficits in the ab-

sence of clear etiology and are without apparent physical stigmata. I am currently conducting a study of the cognitive educational, and behavioral effects of alcohol in children. The subjects are all between the ages of 7 - 12 years, share similar socioeconomic status, and are being assessed using a common set of normative and qualitative measures. Subjects are categorized as normal, learning disabled (LD), fetal alcohol effects (FAE), and fetal alcohol syndrome (FAS), and performance is being compared on measures of I.Q., academic achievement, receptive language, and motor speed. Additional data such as maternal age at delivery, cigarette smoking and multiple substance abuse, auditory-visual acuity, and class placement in school is also being collected.

The rationale for this ongoing study is to provide a better sampling and description of characteristics in learning impaired children whose mothers drank during pregnancy. The implications of the data, however, may extend beyond the diagnostic and educational realms and into such areas as health care, nutrition, obstetrics, pediatrics, nursing, social work, neurology, psychology, and psychiatry. Alcohol related effects adhere to no racial, class, or environmental boundaries and the likelihood of early detection in non-chronic drinkers is poor at best.

The coordination of efforts among multidisciplinary service delivery personnel and a shared commitment by the educational and medical communities regarding the implications of these and other data for improved instruction and management is clearly the direction of choice.

Abel, E.L., The Fetal Alcohol Syndrome: Behavioral Teratology. Psychological Bulletin, 1980, 87, 29-50. (a)

Brent, R.L., X-ray, Microwave, and Ultrasound: The real and the unreal hazzards. Pediatric Annals, 1980, 9 469-473.

Bottoms, S.F., Judge, N.E., Kuhnert, P.M., and Sokol, R.S. Thiocynate and drinking during pregnancy. Alcoholism: Clinical and Experimental Research, 1982, 6, 3, 391-395.

Cohen, M.M., and Shiloh, Y., Genetic toxicology of lysergic acid diethylamide (LSD-25). Mutat. Res., 1977-1978, 47, 183-209.

Darby, B.L., Streissguth, A.P., Smith, D.W., A preliminary follow-up of eight children diagnosed as Fetal Alcohol Syncrome in Infancy. Neurobehav. Toxic. and Teratology, 1981, 3, 157-159.

Dunn, G.H., McBurney, A.K., Ingram, S., and Hunter, C.M., Maternal cigarette smoking during pregnancy and the child's subsequent development. II. Neurological and intellectual maturation to the age of 6½ years. Canadian Journal of Public Health, 1977, 68, 43-50.

Fisher, S.E., Atkinson, M., Burnar, J.K., Jacobson, S., Sengal, P.K., Scott, W., and Van Thiel, D.H. Ethanol-Associated Malnutrition: A contributing factor in the Fetal Alcohol Syndrome. Alcoholism: Clinical and Experimental Research, 1982, 6, 2, 197-201.

Fleet, H.B., Graham, J.M., Harvey, M.A., and Smith, D.W. Patterns of malformations resulting from the teratogenic effects of first semester hyperthermia. Pediatric Research, 1980, 14, 587.

Golden, N.L., Sokol, R.J., Kuhnert, B.R., and Bottoms, S. Maternal alcohol use and infant development. Pediatrics, 1982, 70, 6, 931-934.

Hall, J.Q., Panti, R.M., and Wilson, K.M., Maternal and fetal sequelae of anticoagulation during pregnancy. American Journal of Medicine, 1980, 68, 129-139.

Hanson, J.W., and Smith, D.W., The Fetal Hydantoin Syndrome. Journal of Pediatrics, 1975, 87, 285.

Hanson, J.W., Jones, K.I., and Smith, D.W., Fetal Alcohol Syndrome: Experience with 41 patients. Journal of the American Medical Association, 1976, 235, 1458-1460.

Holsclaw, D.S. Jr., and Topham, A.L., The effects of smoking on fetal, neonatal, and childhood development. Pediatric Annals, 1978, 7, 201-221.

Iosub, S., Fuchs, M., Bingol, N., and Gromisch, D.S. Fetal Alcohol Syndrome revisited. Pediatrics, 1981, 68, 4, 475-479.

Jones, K.L., and Smith, D.W., Recognition of the Fetal Alcohol Syndrome in early infancy. Lancet, 1973, 2, 999-1001.

Jones, K.L., Smith, D.W., Streissguth, A.P., and Myrianthopoulos, N.C., Outcomes in offspring of chronic alcoholic women. Lancet, 1974, 1, 1076-1078.

Kuzma, J.W., Kissinger, D.G., Patterns of alcohol and cigarette use in pregnancy. Neurobehav. Toxic. and Teratology, 1981, 3, 211-221.

Landesman-Dwyer, S., Keller, L.S., and Streissguth, A.P. Naturalistic observations of newborns: Effects of maternal alcohol intake. Alcoholism: Clinical and Experimental Research, 1978, 2, 171-177.

Lemoine, P., Harousseau, H., Borteryu, J.P., and Menuet, J.C. Children of alcoholic parents: Anomalies observed in 127 cases. Ouest Medicale, 1968, 21, 476-482.

Majewski, F., Alcohol embryopathy: Some facts and speculations about pathogenesis. Neurobehav. Toxic and Teratology, 1981, 3, 129-144.

Martin, D.C., Martin, J.C., Streissguth, A.P., and Lund, C.A. Sucking frequency and amplitude in newborns as a function of maternal drinking and smoking. In M. Galanter, (ed.) Currents in Alcoholism, Vol. V, Grune and Stratton, N.Y.: 1979. pp. 359-366.

Mulkovich, L. and van den Berg, B.J. Effects of antenatal exposure to anorectic drugs. American Journal of Obstetrics and Gynecology, 1977, 129, 637-642.

Shaul, W.L., and Hall, J.G., Multiple congenital anomalies associated with oral anticoagulants. American Journal of Obstet. Gynecol., 1977, 127, 191.

Shaywitz, S.E., Caparulo, B.K., and Hodgson, E.S., Developmental language disability as a consequence of prenatal exposure to ethanol. Pediatrics, 1981, 68, 6, 850-855.

Shaywitz, S.E., Cohen, D.J., and Shaywitz, B.A., Behavior and learning difficulties in children of normal intelligence born to alcoholic mothers. The Journal of Pediatrics, 1980, 96, 6, 978-982.

Stone, M.L., et al., Narcotic addiction in pregnancy. American Journal of Obstetrics and Gynecology, 1971, 109, 716.

Streissguth, A.P., Herman, C.S., and Smith, D.W., Stability of intelligence in the Fetal Alcohol Syndrome: A preliminary report. Alcoholism: Clinical and Experimental Research, 1978, 2, 2, 165-170.

Streissguth, A.P., Landesman-Dwyer, S., Martin, J.C., and Smith, D.W., Teratogenic effects of alcohol on humans and laboratory animals. Science, 1980, 209, 353-361.

Sulik, K.K., Johnson, M.C., and Webb, M.A., Fetal Alcohol Syndrome: Embryogenesis in a mouse model. Science, 1981, 214, 20, 936-938.

Wiener, S.G., Shoemaker, W., Koda, L.Y., and Bloom, F.E., Interaction of ethanol and nutrition during gestation: Influence on maternal and offspring development in the rat. Journal of Pharmacology and Experimental Theraputics, 1981, 216, 572-579.

Zackai, E.H., Mellman, W.J., Neiderer, B., and Hanson, J.W., The Fetal Trimethadione Syndrome. Journal of Pediatrics, 1975, 87, 280-284.