The first study concerns the intelligence test results of 21 influenzal meningitis survivors, each of whom was paired with a near age sibling. All patients were ill before the age of 3, during 1960–64. All were tested at age of 6 to 15 years. The mean IQ of the postmeningitic (PM) children was 85 while that for sibling controls (C) was 97 ($p = 0.04$). It was found that 6 of 21 (29%) of the PM subjects were 15 IQ points (1 S.D.) and 2 (10%) were more than 30 IQ points (2 S.D.) lower than C while no C had an IQ which was 15 points below that of his affected sibling.

The second study compares psychological test performance of 25 post-bacterial meningitic children, *considered to be free of sequelae*, who were enrolled in regular public school classes in the first 3 grades. Each was matched by age, sex, social class and classroom membership with a peer control. All subjects were administered a battery of psychological tests. The results indicated that with each of the following, the PM subjects had significantly lower mean scores than peer controls. On the Illinois Test of Psycholinguistic Abilities, the psycholinguistic ages differed at the 0.004 level. On the Frostig Developmental Test of Visual Perception, the perceptual quotient differed at the 0.08 level. On the Peabody Picture Vocabulary Test, the vocabulary quotient differed at the 0.03 level.

It is concluded that these survivors of bacterial meningitis function intelligently at a significantly lower level than their sibling or peer controls.

155 *Psychosocial Study of Childhood Poisoning: A Five-year Follow-up.* JAMES A. MARGOLIS, Univ. of Washington Sch. of Med., Seattle Wash. and Yale Univ. Sch. of Med., New Haven, Conn. (introduced by Albert J. Solnit).

Fifty-two families were involved in a retrospective study of childhood poisoning in 1963. The study population consisted of 20 poison repeaters, 19 single ingestors and 13 controls and their families. The data indicated that childhood poisoning was the result of an abnormal parent-child interaction characterized by behavior problems in the child, limited parent-child relatedness, marital tension and a tense and distant family atmosphere.

The present study was undertaken to determine if these children and their families could still be differentiated by psychosocial variables, 5 years after the initial study and 7 to 10 years after the poisoning episode.

During the interim none of the subjects ingested poisons, nor did their siblings and there was not a shift to other types of accidents. Poison repeaters were continuing to have more behavior problems than single ingestors and they in turn were more deviant than controls. The families could not be differentiated socioeconomically, but there continued to be more stress in the homes of the poisoned subjects, especially therepeaters.

In conclusion, childhood poisoning, especially when repetitive was found to be related not only to behavior problems of the child and family maladaptation at the time of the ingestions, but also significantly predicts later problems in the child and his family. The present study offers further evidence that childhood poisoning is more than just a chance event and may be the result of a pathological parent-child relationship that has long lasting consequences requiring early intervention.

156 *Unmet Needs of Parents of and Individuals with Sickle Cell Anemia (SCA).* CHARLES F. WHITTEN and JOYCE KIRKLAND, Wayne State Univ., Detroit.

Interviews with 50 mothers of children with SCA and 50 adults with SCA reveal that too frequently: (1) Parents believe that their children will not live beyond the teens. (2) Children are pressured to eat 'blood building foods' to improve the anemia and life expectancy. (3) Children's physical activities are unduly restricted. (4) Parents nor children know how to cope with the child's small stature or the teasing it engenders. (5) Parents are not aware that the annoying enuresis may be secondary to SCA. (6) The child's feelings about the disease are not even explored. (7) Parents and adults have uncertainties about the desirability of sicklers marrying and having children. (8) Prior to the birth of a child with SCA parents are not aware of their potential for having children with the disease. (9) One parent blames the other for the child's SCA. (10) Teenagers and adults do not seek training in sedentary occupations. (11) Employers reject applicants with SCA for jobs within their capabilities. These undesirable features appear to exist because: (1) Physicians have not informed, have inadequately informed, or have inaccurately informed sicklers and their parents, or physicians have not used a mode of presentation which promotes understanding and retention, (2) genetic, occupational and psychological counseling have been neglected, (3) employers have not been informed that with a few allowances sicklers can be successful employees. Thus, we need to develop a multidisciplinary public education and anticipatory guidance program. This could significantly improve the lives of many victims of SCA and their parents by enabling them to make realistic and healthy social and psychological adjustments.

157 *Correlation Between Plasma and Liver Concentrations of Vitamins A and E in Patients with Cystic Fibrosis (CF).* BARBARA A. UNDERWOOD and CAROLYN R. DENNING, Inst. of Nutrition Sci. and Dept. of Ped., Columbia Univ. Coll. of Physicians and Surgeons, New York, NY.

This study was designed to determine if the low plasma concentrations of vitamins A and E often seen in children with CF are a reflection of tissue depletion or of a defect in transport mechanisms. Ten patients with CF, ranging in age from 9 to 23 years, were studied. Plasma concentrations of vitamins A and E and carotene were measured in 7 of the 10 patients during the 18 months prior to death. At autopsy all 10 subjects had repeat measurements of these parameters plus an assessment of liver concentrations of vitamin A and α-tocopherol. All patients had received regular supplementation with vitamin A at twice the recommended daily allowance for several years; however, plasma levels of this vitamin were consistently below normal and carotene values were remarkably low in all subjects. Nine of the 10 CF patients had normal or increased vitamin A concentrations in the liver. Eight of the 10 patients received no supplemental vitamin E and had plasma vitamin E levels less than 0.4 mg/100 ml and liver concentrations of α-tocopherol less than half that of the control group. This study has shown that the low plasma concentrations of vitamin A in patients with CF do not reflect inadequate liver stores and suggests a defect in the mechanism by which vitamin A is transported from its storage depot in the liver. In contrast, the low plasma concentrations of vitamin E do correlate with decreased liver stores of α-tocopherol. (Supported by grants from the National Cystic Fibrosis Research Foundation and the Nutrition Foundation.)