Results

One hundred and five children were tested. Their ages ranged from 6 to 17 years. Thirty of the 38 children in Group I had positive tests (78%), while 8 of 29 tested in Group II were positive (27%). There were no positive tests in the 28 children in Group III.

Table I summarizes the results of the skin testing.

Discussion

These results certainly implicate the German roach as a potent sensitizing agent in exposed populations. Sensitization most likely develops by inhalation of their emanations or disintegrating parts, or possibly by the ingestion of the excretions or parts of the roach itself. One might have expected more of the children in Group II to have positive tests because so many of them lived in homes contaminated by roaches at one time or are presently exposed to them in school or in the homes of relatives or friends. The testing material appears to be non-irritating. The children in Group III all have multiple, positive skin tests to various inhalant, pollen, food, and mold allergens, yet no one reacted to extracts of the German roach, even when tested with solutions containing as much as 2,400 PNU per cc of the material.

The important question not answered by this study is, what role does this sensitivity to the roach play in the etiology of the patient's symptoms? There is a suggestion that it may be quite important in some patients. Patients with positive intradermal reactions have reacted to inhalation of the insect extract with clinical wheezing.[3] In the present study, three children with positive tests developed mild wheezing, and another developed nasal symptoms 10 to 20 minutes following the skin test injection. Also, several children showed definite exacerbation of their symptoms when visiting homes where roaches were abundant, while others had a definite relief of symptoms when they moved from environments heavily contaminated by the cockroach. Of course, other factors may have been important in these instances.

There are many children living in slum areas with perennial, persistent, nasal symptoms and wheezing who appear resistant to the usual modes of allergic therapy. In this group of patients, a clinical sensitivity to the roach, which contaminates their environment, may be one of the factors perpetuating their symptoms. Certainly, further investigation is required to fully evaluate it's importance.

Summary

There is a definite tendency for allergic children exposed to cockroaches to develop positive skin tests. Suburban children, who were not exposed but had the same allergic problems of wheezing and nasal symptoms, had no positive tests. There is a suggestion that this sensitivity may play a role in perpetuating allergic symptoms in some children. The cockroach seems to be an important potential allergen in susceptible populations.

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LSD Exposure *in Utero*

In spite of the many recent reports on the damaging effects of LSD on human chromosome *in vitro*[1,2] and *in vivo*[3-6] and its ability to produce congenital malformations in rodents,[7] the teratogenic potential of this drug in humans is still uncertain. Two infants with limb malformations who had been exposed to LSD *in utero* have thus far been reported.[8,9] On the other hand, Cohen, *et al.*,[5] reported nine children and Hulten, *et al.*,[6] one child, all of whom had been exposed to LSD *in utero* and had no obvious birth defects although chromosome damage was apparent. Hecht and his co-workers[9] have pointed out the need for additional data on infants who had *in utero* exposure to LSD, regardless of the presence or absence of congenital malformations, so that the teratogenic properties of the drug could be evaluated. In view of this request, we wish to report a clinically normal child with a normal

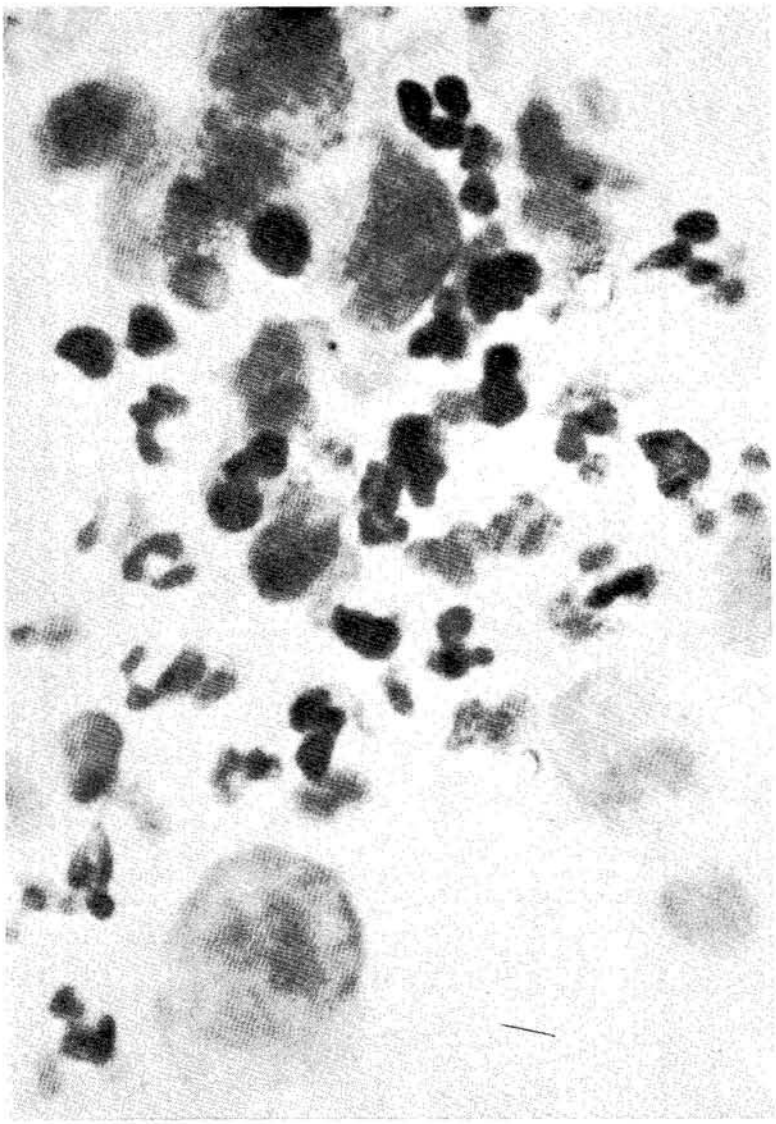

FIG. 1. Micronuclei are shown among normal-sized, blast-like nuclei.

karyotype, who was exposed to LSD repeatedly during the first 4 months of fetal life.

CASE REPORT

This family consists of a 37-year-old male, his 36-year-old wife, and their 8-month-old son who was exposed to LSD-25 during the first 4 months *in utero*. The parents ingested LSD regularly since October 1963, all of which was obtained from a British drug firm in pure form. On this basis they feel that their estimates of dosage are accurate. During the course of ingestion (Table I) they also consumed peyote and smoked cannabis regularly.

While using LSD, peyote, and cannabis, the mother became pregnant. Her last menstrual period was on May 19, 1966, and she thinks that she conceived on May 29, 1966, a day on which she ingested 320 μg and her husband ingested 800 μg of LSD. LSD was also ingested during the second, third, and fourth months of pregnancy, as shown in Table I. In October, realizing she was pregnant, she discontinued LSD ingestion during the remainder of the pregnancy and the period of lactation, although she used cannabis regularly. Other than three buttons of peyote on September 5, the mother denies ingesting any other drugs.

The pregnancy was otherwise uneventful and the child was born February 23, 1967, 2 weeks prior to the expected date of confinement. He

TABLE I

LSD INGESTION DURING PREGNANCY

Date	*μg LSD Ingested*	
	Mrs. X	*Mr. X*
October	2,500	2,500
November 1963–July 1964	500–1,500	500–1,500
July 1964–May 1966	300–1,000 (every 3–4 wk)	300–1,000 (every 3–4 wk)
May 29, 1966*	320	800
July 10, 1966	320	320
August 7, 1966	320	320
September 5, 1966	160	none
September 27, 1966	320	320
October 1966–June 1967	300–1,000 (every 3–4 wk)	300–1,000 (every 3–4 wk)
July 4, 1967	320	300
August 1967	none	640
October 1967	320	800
Total intake	20,000	50,000

* Presumed date of conception.

weighed 6 lb, 8 oz at birth and was judged to be completely normal then and at 8 months of age. Dermatoglyphics were unremarkable.

Leukocyte cultures were started on both parents and the 8-month-old infant 5 weeks after the father ingested 800 μg and the mother ingested 320 μg of LSD Lymphocytes from a patient with Fanconi's anemia (positive control) and from one of the author's (negative control) were simultaneously cultured.

Numerous breaks were found in the chromosomes from the positive control. The chromosomes from both parents, their child, and the negative control lacked abnormal chromosomal associations or a significant number of breaks.

The only unusual cytological finding was a large number of micronuclei scattered individually and sometimes in groups in the cells cultured from the father's blood (Fig. 1). These micronuclei were distinctive from the normally appearing degenerating polymorphonuclear cells usually seen in leukocyte cultures both on the basis of size and number. None of the other samples exhibited these micronuclei nor have they been seen in this laboratory in any sample before or since. Micronucleated cells in leukocyte cultures of patients who had ingested LSD have once before been reported.[10]

COMMENT

There are several reports in which LSD has been implicated in chromosomal damage, both *in vitro*[1,2] and *in vivo*.[3-6] Other reports have failed to show chromosomal damage in individuals taking LSD.[10,11] Since LSD users usually take other drugs at the same time and LSD purchased from a vendor often contains substances other than LSD (e.g., amphetamines), it is difficult to be certain whether the observed *in vivo* chromosomal damage is due to LSD, to the other drugs, or to a synergistic action requiring both.

Hulten, *et al.*,[6] recently reported that an infant exposed to LSD *in utero* had an increased frequency of breaks at 5 weeks of age, but these aberrations had disappeared at 8 months. Since the infant son of our family was studied at 8 months of age, we cannot exclude the possibility that chromosomal damage would have been demonstrable earlier in life.

Idanpaan-Heikkila and Schoolar[12] recently presented evidence of transplacental movement of radioactive LSD in mice, indicating that fetal exposure to this drug may occur following maternal ingestion. However, the published data on the teratogenic action of pure LSD are controversial both in rodents[13,14] and humans.[5,8,9] Some have attempted to explain this on the basis of the timing of the embryo's exposure to the drug. Our family had an apparently normal child who was conceived during a period of LSD ingestion and was again exposed to the drug during the second, third, and fourth months *in utero*.

This report is not meant to imply that LSD is without danger to the embryo. It merely demonstrates that intra-uterine exposure to pure LSD at the times indicated in Table I, in addition to consistent exposure to cannabis, does not necessarily lead to abortion, persistent chromosome damage, or readily recognizable congenital malformations.

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Gangrene of an Extremity in the Newborn

Gangrene of an extremity in a newborn infant appears to be a rare phenomenon. A review of the literature by Askue and Wong[1] in 1952 included 52 cases, 30 of which involved a lower extremity. Etiologic factors producing the gangrene are obscure in most cases. Possible causative mechanisms such as trauma, infection, pressure, and arterial thrombosis have been suggested but rarely proven.

FIG. 1. Gangrenous right leg of newborn infant with sharp line of demarcation in upper thigh.

This report describes a second born twin who had gangrene of the right lower limb at birth caused by arterial obstruction from a dissecting intramural hematoma of the femoral artery.

Case Report

This male infant was the second born twin of a gravida 2, para I, 26-year-old, white mother. Pregnancy had been normal until 24 weeks' gestation, when severe back pain developed. At this time a 21 pounds gain in weight during 1 month was recorded. There was no history of maternal trauma or drug ingestion. Roentgenograms revealed the presence of twins with signs of fetal death in the presenting infant. Forty-eight hours after the mother's admission the first twin was delivered spontaneously. No amniotic fluid escaped, the infant was a stillborn, macerated male infant, 26 cm in crown-heel length and estimated to be 22 weeks' gestation. After a pudendal block, the mem-