Urban Poverty: Effects on Prenatal Nutrition

Abstract. *Infants of poor (as measured by an index) mothers were 15 percent smaller than infants of nonpoor mothers. Infants from poor families had multiple anatomic evidences of prenatal undernutrition.*

Perinatal mortality rates are higher in the United States than in many other nations (*1*). An excess of infants of low weight at birth accounts for much of this high perinatal mortality (*1*). Low birth weight and perinatal death are much more common in families of low socioeconomic status than in families that are better off (*1, 2*). We now identify undernutrition as the cause of low birth weight in a group of infants born of poor urban mothers.

Material was examined from 445 consecutive autopsies on stillborn and newborn infants at Babies Hospital, New York City. Gestational ages calculated from the mother's last menstrual period ranged from 20 to 44 weeks. One hundred ninety-three cases were excluded from further consideration because there were fetal or maternal disorders that may have affected fetal growth (*3, 4*). These included multiple births, hypertension and other manifestations of maternal toxemia, maternal diabetes mellitus, major congenital malformations in the newborn, chromosomal disorders in the newborn, any evidence of chronic fetal infection, and erythroblastosis fetalis. The remaining 252 cases were classified as poor or nonpoor, on the basis of weekly income and family size, the standard being the poverty-index tables (U.S. Social Security Administration) (*5*). This index is not generous. In 1966 for a family of four, it provided only 75 cents a day per person for total food expenditures (*5*). Of the infants, 49 were born to poor families, and 203 were born to nonpoor families. Thirty-seven percent of the poor and 27 percent of the nonpoor infants were stillborn. All other infants died within 48 hours of birth. Both the poor and the nonpoor constitute high-risk groups. The mean number of gestations for poor mothers was 3.9 and for the nonpoor 2.8. In the poor, 85 percent of the pregnancies before the current one resulted in surviving children; the figure was 76 percent for the nonpoor.

Table 1. Mean organ and body weights (± 1 S.D.) in newborn infants of poor and nonpoor families in percent of "normal" published values (*6*). Body length: poor infants, 97 ± 7; nonpoor infants, 103 ± 10; difference of 6 percent, $P < .005$.

Item	Poor	Nonpoor	Difference
Body	92 ± 18	107 ± 23	15*
Brain	101 ± 19	107 ± 25	6†
Heart	90 ± 27	105 ± 28	15*
Liver	83 ± 22	104 ± 31	21*
Spleen	81 ± 45	104 ± 49	23*
Thymus	66 ± 31	104 ± 49	38*
Kidney	91 ± 34	101 ± 37	10†
Adrenal	77 ± 38	102 ± 46	25*
Placenta	84 ± 35	88 ± 29	4‡

* $P < .005$; † $P < .05$; ‡ $P > .1$.

Weights of organs and body measurements were calculated in percent of mean values for "normal" infants (*6*). A mean percent of these "normal" values was then calculated for each organ or body measurement (Table 1). The method of point counting was used to determine the relative size of abdominal subcutaneous adipose cells (*7*).

The mean gestational age for both groups was 29 weeks. Mean thickness of abdominal subcutaneous fat was 2.9 mm ± 1.0 (1 S.D.) in the poor infants and 4.5 mm ± 1.2 in the nonpoor infants ($P < .05$). Mean volume of individual adipose cells in this locus was 2.92 ± 1.40 (arbitrary units) in poor infants and 4.47 ± 1.46 in nonpoor infants ($P < .05$).

Undernutrition appears responsible for the prenatal growth retardation in infants from poor families. Both mass of adipose tissue and size of individual fat cells were smaller in the infants from poverty families. Such infants had thymus, spleen, liver, and adrenal glands relatively smaller than brain, kidneys, heart, and skeletal bones. This particular ranking of organ growth has often been observed in both human beings and animals who have experienced chronic alimentary undernutrition (*3, 8*).

It has also been observed in a variety of placental and uterine disorders in which the flow of nutrients to the growing fetus was restricted (*4*). Since all multiple births and cases with a known uterine or placental disorder were excluded from the current study, it is possible that maternal malnutrition during gestation contributed to the fetal undergrowth. Other environmental or genetic factors have not been excluded, but most families in the two economic groups lived in the same area of New York City. The racial balance in the two economic groups was also similar.

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References and Notes

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Postpartum Psychiatric Reactions: Time of Onset and Sex Ratio of Newborns

Abstract. *There is no predominance of male infants born to women who develop schizophrenic symptoms within 1 month after delivery. Moreover, there is no difference in the sex ratio of infants born to schizophrenic women whose onset of symptoms occurred within the first 10 days postpartum, which is the period of rapid hormonal changes that is most likely to reflect postnatal influences of fetal sex.*

Taylor (*1*) studied state hospital records of 26 pregnancies where the mother developed schizophrenic symptoms during the first month after delivery. Twenty-two of the offspring were male and four were female. From this finding, he postulated a causal relation between birth of a male infant and postpartum schizophrenia. He specu-