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Relation of Poverty and Race to Birth Weight and Organ and Cell Structure in the Newborn

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Extract

Based on studies of birth weights, there has long been speculation that poverty, inadequate maternal nutrition, and race might alter prenatal growth by influencing fetal nutrition. In the present study, undernutrition was identified as the cause of low birth weight in a group of infants born to poor urban mothers in the United States. A postmortem, quantitative, morphological study of body size, organ size, and cellular structure demonstrated that the infants of 83 poor mothers had many anatomic abnormalities recognized as characteristic of undernutrition. Such abnormalities were not found in infants from 386 families with incomes above the poverty line. There were almost no differences in body or organ growth among various racial groups when the comparisons were between families of similar economic status.

Speculation

Is the low birth weight in infants born to poor urban mothers due to abnormal nutrition during pregnancy, the mother's earlier development, or uterine or placental abnormalities? If maternal malnutrition is responsible, the exact nutritional deficiencies should be promptly identified so that preventive programs can be developed. There is also a pressing need to know whether or not the moderate undernutrition observed in the present study alters brain composition and subsequent mental and motor performance.

Introduction

In recent years considerable attention has been focused on unfavorable perinatal death rates in the United States, which are significantly higher than those recorded in a number of nations with a lower standard of living [14]. Most of this excessive mortality is centered in families of low socioeconomic status [4, 8, 14, 15, 29]. High perinatal mortality rates in nonwhites are presumably due to their over-all low socioeconomic standing [14, 32]. Children of poor families in other nations also have an excessive perinatal mortality [3, 6, 34, 35]. Inadequate maternal prenatal care has been cited by some and rejected by others as the factor principally responsible for the large perinatal losses of the poor [1, 16, 31, 34]. It is widely agreed that birth weights are lower in the poor and that such low weights are associated with their high perinatal mortality [3, 6, 8, 13–15, 29, 32, 35]. A recently published preliminary study demonstrated that infants born to poor urban families had certain anatomic abnormalities characteristic of undernutrition [25]. It was speculated that this undernutrition might contribute to their increased perinatal mortality. The present much

Table I. Weight and length data by income[1]

	Income: percentage of poverty line			
	Less than 100%[2]	100–149%	150–199%	Over 200%[3]
Length[4]				
Body	98 ± 9	104 ± 9[5]	104 ± 9[5]	102 ± 10[5]
Weight[4]				
Body	94 ± 17	110 ± 21[5]	111 ± 25[5]	107 ± 21[5]
Brain	102 ± 17	114 ± 28[5]	111 ± 23[5]	110 ± 25[5]
Heart	91 ± 24	114 ± 55[6]	109 ± 28[5]	107 ± 29[5]
Liver	88 ± 24	110 ± 32[5]	106 ± 29[5]	101 ± 32[5]
Spleen	83 ± 44	106 ± 52[6]	105 ± 51[6]	98 ± 51[6]
Thymus	74 ± 36	103 ± 50[5]	111 ± 51[6]	98 ± 48[5]
Kidney	92 ± 35	102 ± 34[6]	108 ± 37[6]	96 ± 38[7]
Adrenal	84 ± 39	100 ± 37[5]	108 ± 45[6]	94 ± 45[6]
No. of cases	83	102	79	205

[1] Mean organ and body measurements in newborn infants from poor and nonpoor families in percentage of "normal" published values [12]. They are divided into groups by representing their incomes in percentage of the poverty line values recorded in the Social Security Administration index [28]. *P* values compare each of the nonpoor groups with the poor group.
[2] Poor.
[3] Private and semiprivate, nonpoor.
[4] Values are given in mean ± SD.
[5] $P < 0.005$.
[6] $P < 0.05$.
[7] $P > 0.1$.

larger study has revealed additional features of undernutrition in such neonates. In addition, data are supplied on how the degree of poverty and race influence prenatal nutrition.

Patients

Material was examined from 1002 consecutive autopsies on stillborn and newborn infants whose tissues were well preserved at Babies Hospital, New York City. Gestational ages calculated from the last menstrual period ranged from 20 to 44 weeks, and 449 of the cases were excluded from further consideration because of fetal or maternal disorders that may have affected fetal growth. Fetal conditions that led to exclusion included major congenital malformations, chromosomal disorders, chronic infection, a hemolytic process, or multiple births. Maternal factors that led to exclusion were hypertension or other manifestations of toxemia, diabetes mellitus, chronic gestational infection, cancer, cardiac disease, exposure to teratogenic drugs, or placental abnormalities such as large infarcts or prolonged partial separation.

Data on income were available on 469 of the remaining 553 cases, and they were classified as poor or nonpoor on the basis of weekly income and family size with the use of urban poverty index tables developed in the US Social Security Administration [28]. Weekly income was determined through personal interview by a clinical registrar at the time the patient was registered for antenatal care in the outpatient clinic at Sloan Hospital for Women in New York City. With 1966 taken as a base line, the Social Security index was corrected for earlier and later changes in the economy by means of the consumer price index. In 1966 for a family of four it provided only 75 cents a day per person for total food expenditures [28]. A few patients without specific income data were classified as nonpoor because they had private or semiprivate status in the hospital.

With the use of the aforementioned criteria, 83 of the infants were born to poor and 386 to nonpoor families. The nonpoor were divided into subgroups by expression of their incomes in percentage of the poverty line values recorded in the Social Security Administration index. Infants were classified for race or origin according to the mother's self-identification or birthplace. Included were 190 white, 179 black, 4 oriental, and 96 Puerto Rican or Cuban infants. Except for the private and semiprivate cases, almost all the mothers received their prenatal care in the antenatal clinic at the Sloan Hospital for Women.

Methods

Organ weights and body measurements were obtained from autopsy protocols. In each case, weights and measurements were calculated in percentage of mean values for "normal" infants as recommended by Gruenwald and Minh [12]. Since Gruenwald's tables start at a gestational age of 24 weeks, "normal" values for the period from 20 to 24 weeks were interpolated from the tables of Schulz *et al.* [30]. A mean percentage of these normal published values was then calculated for each organ or body measurement for each classified group (Table I).

The method of point counting was used to determine the approximate percentage of various components comprising a tissue such as the percentage of nuclei in Wharton's jelly [7]. The volume of hematopoietic tissue in the liver was determined by multiplication of the percentage of these cells in a given volume by the recorded weight of the organ in grams.

Previous studies have shown that nuclear size of an individual cell type is almost the same in normal newborn infants and those with a variety of fetal growth disorders [18, 20, 21, 24, 27]. On the assumption that the same thing was true in the current study, total cytoplasmic volume of a cell type in an organ was divided by total nuclear volume to obtain the relative cytoplasmic volume per cell. Skeletal muscle fibers, hepatic parenchymal cells, and adrenal fetal zone cells

Table II. Various measurements in newborn infants of poor and nonpoor mothers[1]

	Gestational ages 20–29 weeks		Gestational ages 30–42 weeks	
	Poor	Nonpoor	Poor	Nonpoor
Abdominal wall subcutaneous fat thickness, mm	0.92 ± 0.64	1.70 ± 0.68 ($P < 0.005$)	2.00 ± 1.09	2.77 ± 1.66 ($P < 0.1$)
Adipose cells area, μ^2	306 ± 173	395 ± 157 ($P < 0.05$)	434 ± 229	617 ± 232 ($P < 0.025$)
Skeletal muscle fibers, cytoplasm/cell	3.9 ± 1.3	5.7 ± 1.5 ($P < 0.005$)	6.1 ± 1.7	10.8 ± 4.3 ($P < 0.005$)
Liver, cytoplasm/hepatic cell,	4.2 ± 1.2	4.9 ± 1.5 ($P < 0.05$)	4.1 ± 1.2	6.3 ± 3.4 ($P < 0.01$)
Liver, volume hematopoietic tissue	4.3 ± 2.7	5.5 ± 3.7 ($P < 0.1$)	3.9 ± 2.3	5.6 ± 4.4 ($P < 0.1$)
Adrenal glands, fetal zone, cytoplasm/glandular cell	7.2 ± 1.9	10.4 ± 4.0 ($P < 0.005$)	8.1 ± 2.7	9.9 ± 4.3 ($P < 0.1$)
Umbilical cord, % nuclei in Wharton's jelly	2.9 ± 1.9	1.9 ± 0.4 ($P < 0.05$)	2.5 ± 2.0	1.6 ± 0.7 ($P < 0.1$)
No. of cases	25	24	21	22

[1] Except where specified, values are in arbitrary units.

were specifically selected for such study because their cytoplasmic mass is much reduced by undernutrition [9, 17–19, 21–23, 26]. We determined the size of adipose cells by counting the number of such cells located within a premeasured grid and dividing the area of the grid by the number of adipose cells within it. All these quantitative histological measurements were made on 46 infants of poor and 46 infants of nonpoor families. These 92 cases were selected on a random basis from the two larger groups. All tissues were fixed in buffered, neutral formalin for 48 hr. Student's *t* test (two-tailed) was used to evaluate the significance of all data. To avoid bias, all anatomic data on individual cases were collected without knowledge of the families' economic status.

Results

Body weight at autopsy for the infants from poor families was 13–17% less than the mean value for infants from nonpoor families (Table I). The mean gestational age for both groups was 29 weeks, and the relative distribution of infants at the various gestational ages was similar in the poor and nonpoor groups. Body length and all organ weights were smaller in the infants from poor families. In these infants, weights of thymus, spleen, liver, and adrenal glands were disproportionately smaller than weights of other organs (Table I). Differences in brain weight between the two groups were small. Mean thickness of abdominal subcutaneous fat was significantly less in the infants from poor families (Table II).

The mean volume of individual adipose cells from the abdominal wall was significantly smaller in the neonates from poor families (Table II). Similar differences in the two groups were noted in measurements of the mean cytoplasmic volume of individual skeletal muscle fibers, hepatic parenchymal cells, and glandular cells in the fetal zone of the adrenal glands. The smaller weight of the liver and adrenal glands in infants of the poor was mainly due to a smaller volume of cytoplasm in individual parenchymal cells because the number of these cells was not much different in the poor and nonpoor groups (Tables II and III).

Table III. Number of cells in two organs[1]

	Poor	Nonpoor	*P*	No. of cases	
				Poor	Nonpoor
Liver, no. hepatic cells					
20–25 weeks	212 ± 80	190 ± 62	>0.1	14	13
26–30 weeks	359 ± 67	393 ± 138	>0.1	12	12
31–35 weeks	533 ± 224	609 ± 339	>0.1	12	10
36–42 weeks	861 ± 334	1064 ± 308	>0.1	8	11
Adrenal gland, fetal zone, no. of glandular cells					
20–25 weeks	15.4 ± 6.6	14.2 ± 6.2	>0.1	14	13
26–30 weeks	19.8 ± 5.1	18.4 ± 10.3	>0.1	14	12
31–35 weeks	38.6 ± 18.1	36.8 ± 11.6	>0.1	12	10
36–42 weeks	35.4 ± 18.9	44.1 ± 15.8	>0.1	8	11

[1] Values are relative rather than absolute.

Infants from poor families had a greater percentage of nuclei in Wharton's jelly than those from nonpoor families, reflecting a smaller mass of jelly per nucleus in the former group (Table II). Total volume of hematopoietic tissue in the liver was somewhat greater in infants of the nonpoor (Table II). Even when represented in proportion of mean liver weight, there was

Table IV. Weight and length data by race, poor[1]

	Caucasian	Black	Puerto Rican
Length			
Body	99 ± 4	99 ± 7	99 ± 6
Weight			
Body	97 ± 15	91 ± 18	99 ± 14
Brain	106 ± 15	98 ± 15	113 ± 21
Heart	89 ± 9	90 ± 27	93 ± 21
Liver	95 ± 24	85 ± 24	92 ± 22
Spleen	71 ± 26	87 ± 65	90 ± 48
Thymus	74 ± 32	70 ± 33	79 ± 42
Kidney	98 ± 41	87 ± 33	106 ± 33
Adrenal	88 ± 20	83 ± 42	87 ± 39
No. of cases	13	49	19

[1] Mean organ and body measurements in Caucasian, black, and Puerto Rican newborn infants from poor families in percentages of "normal" published values [12]. P is greater than 0.1 in all of the intergroup comparisons, except for brain ($P < 0.05$) and kidney ($P < 0.1$) comparisons between blacks and Puerto Ricans.

Table V. Weight and length data by race, nonpoor[1]

	Caucasian	Black	Puerto Rican
Length			
Body	103 ± 8	104 ± 9	103 ± 11
Weight			
Body	109 ± 20	108 ± 24	109 ± 24
Brain	109 ± 21	113 ± 27	112 ± 28
Heart	111 ± 31	107 ± 31	110 ± 52
Liver	106 ± 35	103 ± 29	103 ± 25
Spleen	104 ± 56	98 ± 48	108 ± 48
Thymus	100 ± 47	102 ± 48	111 ± 55
Kidney	101 ± 38	97 ± 36	104 ± 34
Adrenal	103 ± 44	97 ± 43	93 ± 35
No. of cases	177	130	77

[1] Mean organ and body measurements in Caucasian, black, and Puerto Rican newborn infants from nonpoor families in percentages of "normal" published values [12]. P is greater than 0.1 in all of the intergroup comparisons.

Table VI. Population characteristics[1]

	Poor	Nonpoor	P
Mother's age	26.5 ± 6.6	26.7 ± 6.0	>0.1
Mother's height	63.5 ± 3.1	63.9 ± 2.7	>0.1
Living children, no.	2.26 ± 2.10	1.18 ± 1.40	<0.1
Total no. of pregnancies	3.93 ± 2.60	2.85 ± 1.96	>0.1
Newborn body weight in % of control values			
0–1 living children in family	94.9 ± 18.9	109.0 ± 22.6	<0.05
2–3 living children in family	94.9 ± 14.1	107.1 ± 19.6	<0.05
4 or more living children in family	90.0 ± 16.5	109.0 ± 16.1	<0.05

[1] Values are mean ± SD.

slightly more hematopoietic tissue in the nonpoor groups (4%). Differences in body and organ measurements were insignificant between the income-classified subgroups above the poverty line (Table I). Families on New York City welfare rolls were all above the poverty line and showed no neonatal undernutrition. Groups based on race or geographic origin also showed few differences when they had similar economic status (Tables IV and V). The differences between brain weights in blacks and Puerto Ricans in Table IV are unexplained.

The mothers' ages and heights were similar in the poor and nonpoor groups (Table VI). The poor mothers had a greater number of recorded pregnancies than the nonpoor. There was a small decrease in relative birth weights with increasing family size in the poor group (Table VI).

Discussion

In the current study newborn infants from poor urban families were somewhat undergrown for gestational age by comparison with infants with more prosperous parents. Organ structures studied postmortem were not the same in infants of poor families as they were in infants with more prosperous parents. If organ structure in the latter group is considered to be normal, organs are abnormal in the infants from poor families, the abnormalities being characteristic of undernutrition. In infants from poor families, thymus, spleen, liver, and adrenal glands were more undergrown than kidneys, heart, and skeletal bones; there was almost no abnormality in brain weights. This particular ranking of relative organ growth has been repeatedly observed in a number of placental and uterine disorders that restrict the flow of nutrients to the growing fetus [10, 17, 21, 26]. This growth pattern also has frequently been reported in both children and young animals with chronic postnatal undernutrition [19, 22].

Microscopic abnormalities in the newborn infants of the poor are also characteristic of undernutrition. These features include a subnormal mass of cytoplasm in adipose cells and skeletal muscle fibers. The subnormal size of several visceral organs in these infants is also due to a reduced mass of cytoplasm in individual parenchymal cells; the number of parenchymal cells in these organs is near normal. These features are also characteristic of undernutrition [26]. In contrast, in most non-nutritional fetal growth disorders studied to date, organs are small because they have a subnormal number of cells whereas cell size is normal or increased

[20, 24, 26]. Thus, both gross and microscopic features of organ structure in newborn infants from poor families point to undernutrition as the cause of the retarded prenatal growth.

The undernutrition in newborn infants of the poor might be related to uterine or placental abnormalities that restrict the flow of nutrients to the fetus or it might be due to inadequate maternal nutrition during pregnancy. There is no evidence that uterine or placental disorders were responsible for the growth retardation observed in the current study. Cases with recognized uterine or placental abnormalities were specifically excluded from the analysis. Furthermore, infants of the poor had none of the anatomic evidences of chronic antenatal hypoxia characteristically induced by uterine or placental abnormalities. When present, such placental or uterine disorders presumably induce anatomic evidence of hypoxia by restricting gas exchange with the fetus. The most easily quantitated fetal abnormality related to chronic hypoxia is an increased mass of largely erythroid hematopoietic tissue in the liver [21, 23]. Infants of poor families in the current study had a smaller absolute and relative volume of hepatic hematopoietic tissue than the infants of nonpoor families.

Maternal malnutrition during gestation provides the simplest explanation for the undernutrition found in newborn infants of the poor. Although lay opinion easily relates maternal diet to fetal nutrition, scientific data are surprisingly sparse for human beings. There was a decline of birth weights during World War II of about 200 g in both Japan and Holland during periods of widespread hunger [11, 33]. The decline was about 500 g during the intense hunger of the siege of Leningrad [2]. Unfortunately, accurate gestational ages are not available for these studies so that it is not possible to analyze the individual roles of prematurity and retarded prenatal growth in the genesis of the reduced birth weights. Since World War II there has been an increase in birth weights in Japan, caused entirely by an increased rate of third trimester fetal growth [11]. It has been postulated that undernutrition exerts its main influence in late gestation and that the increasing birth weights are explained by dietary improvements during pregnancy [11]. Unfortunately, the Japanese study did not include good dietary data [11]. Data in the current study indicate that maternal nutrition might also affect midgestational fetal growth (Table II). Older small studies have demonstrated a direct relation between the quantity and quality of maternal diet and birth weights [5]. There is also the possibility that mother's childhood nutrition and growth may influence subsequent fetal growth [3]. Unfortunately, our study provides no information about parasites or infection in the mothers of the undernourished neonates.

Infant mortality rates of nonwhites are about double those of whites in the US [14]. Low birth weights are more than twice as common in nonwhites, but the white infant does not become significantly larger than the nonwhite until the last month of gestational life [13, 14]. Since the greatest effect of undernutrition on fetal growth may be in late gestation, one might suspect that nutritional factors are responsible for the reported racial differences in fetal growth and neonatal mortality. Data in the present study support this hypothesis. There were few differences in body or organ growth among the various racial groups when the comparisons were between families of similar economic status.

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