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Maternal regulation of fetal growth

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The birth weight of the mothers, sibs, maternal aunts and their children were compared with those of 400 full-term, appropriate-for-gestational age, 181 true premature, 200 small-for-gestational age, and 261 large-for-gestational age neonates. Except for true prematures, a close correlation was found between the weight of the newborns and their mothers and maternal relatives in each case. The findings support the Ounsted theory that the rate of fetal growth is influenced by a familial component with maternal transmission. This regulation does not operate in true prematurity where the effect of environmental and pathological factors seems to prevail over the familial and genetic features.

In the last two decades many efforts have been made to identify the factors responsible for the great variation in mean birth weight both between and within ethnic groups. It has been shown that the socio-economic situation, maternal nutrition, smoking habits, height of the parents, parity and high altitude may all influence the rate of fetal growth. Familial low birth weight has also long been known but the data on genetic factors are still rather speculative [5]. Considering the theory of Ounsted and Ounsted [2, 3] that through maternal regulatory genes the mother's own intrauterine experience affects her reproductive performance, we have made an attempt to compare the birth weight of neonates with those of their mothers, sibs, maternal aunts and cousins.

Material and Methods

In the neonatal units of three hospitals in County Győr-Sopron, West-Hungary, birth weight, length, head circumference and gestational age of a total of 2149 consecutive newborn infants were determined. Gestational age was calculated from the day of the last normal menstruation period. If this was not possible or a remarkable discrepancy between calculated week of pregnancy and somatic or neurological maturity was noted, the baby was excluded from the study.

The mothers of the infants were personally interviewed and their age, height, prepregnancy weight, profession, qualification, social status, and smoking habit were registered. Inquiries were made also about their previous diseases, spontaneous and artificial abortions, and labours. Special care was taken to find out the birth weight of the mothers themselves, that of their other children, of their sisters, and of the children of the latter.

At final evaluation only those families

were considered in which the mother could give reliable information on her own birth weight. Thus, the family data of 1042 neonates were involved in the study, who could be grouped according to gestational age and birth weight as follows.

1. Full-term appropriate-for-gestational age (AGA) newborns with a gestational age from 37 to 42 weeks, and with a birth weight between the 10th and 90th percentile values of the Hungarian standard. 400 neonates fulfilled these requirements.

2. True premature neonates with a gestational age under 37 weeks, whose birth weight fell between the 10th and 90th percentile curves of the local chart. This group included 181 infants.

3. Small-for-gestational age (SGA) newborns, whose birth weight was under the 10th percentile, irrespective of their gestational age. 200 babies were regarded as SGA.

4. Large-for-gestational age (LGA) infants, whose birth weight was above the 90th percentile, irrespective of their gestational age. 261 neonates belonged to this group.

Student's t test, correlation coefficients, and regressions were used to evaluate the data.

Results

The analysis of the effect of some maternal and environmental factors on fetal growth confirmed the well established facts that the weight of

Table I

Age, height and prepregnancy weight of the mothers of infants of the four groups examined (mean ± S. D.)

	Age (year)	Height (cm)	Prepregnancy weight (kg)
Full-term AGA	23.9±4.3	163±5.7	59.7±8.9
SGA	24.2±4.7	159±6.6[c]	55.8±10.4[c]
True prematures	24.5±5.0	161±6.6[b]	57.5±9.8[a]
LGA	25.6±4.4[c]	165±5.9[c]	66.4±11.1[c]

Significance of the difference from the mean of the full-term AGA mothers:
[a] = $p < 0.01$
[b] = $p < 0.005$
[c] = $p < 0.001$

Table II

Birth weight of neonates, their mothers, sibs, maternal aunts and their children in the four groups examined (mean ± S. D.)

	True prematures		SGA		Full-term AGA		LGA	
	N	Birth weight	N	Birth weight	N	Birth weight	N	Birth weight
Index neonates	181	2.16±0.55	200	2.43±0.29	400	3.29±0.30	261	4.17±0.27
Mothers	181	3.02±0.63	200	2.75±0.48	400	3.30±0.74	261	3.68±0.58
Sibs	137	3.04±0.41	114	2.54±0.47	457	3.23±0.38	215	3.79±0.37
Maternal aunts	90	3.24±0.54	83	2.65±0.56	214	3.28±0.41	113	3.80±0.49
Children of maternal aunts	87	3.10±0.44	84	2.73±0.46	145	3.24±0.33	80	3.80±0.41

the neonate was positively correlated to height, weight, qualification and standard of life of the mother, while previous abortions, smoking and diseases during the pregnancy predisposed to lower birth weight. Thus, these factors are not dealt with in detail, only age, height and prepregnancy weight of the mothers are shown in Table I.

Average birth weight of the neonates and those of the mothers and maternal relatives is summarized in Table II.

As shown by the figures, a fairly small variation in birth weight was observed when full-term AGA infants were compared with their mothers, sibs, maternal aunts and cousins. The differences between the various family members examined were not significant statistically, on the other hand, when calculating correlation coefficients (r), a significantly close correlation was demonstrated in each case.

Similar tendencies were verified in the case of both SGA and LGA infants. The birth weight of SGA children correlated well to the birth weight of their mothers ($r = 0.312$; $p < 0.001$), and even the sibs, aunts and their children were born with a weight of remarkably less than 3.00 kg. The mothers and other family members of LGA infants were also heavy newborns, and there was a close correlation between probands and mothers ($r = 0.326$; $p < 0.001$), and between probands and sibs ($r = 0.511$; $p < 0.001$), respectively. At the same time, the true prematures proved to be significantly lighter than their mothers whose birth weight was considerably higher than that of the SGA mothers ($p < 0.005$). This was valid also for sibs, aunts and the children of aunts, whose mean birth weight nearly reached that of the full-term AGA infants and their relatives.

Discussion

Small and light women have generally smaller babies than those who are tall and heavy [6]. Ounsted and Scott [4] showed, however, that this generalization is not applicable throughout the whole spectrum. In their survey the proportion of heavy women in the SGA group did not differ from the full-term AGA group, whereas in the LGA group it was six times greater. In our material the mothers of LGA infants were remarkably heavier and moderately taller than the mothers of full-term AGA babies, while the mothers of SGA neonates proved to be significantly smaller and lighter. Since the majority of the infants of these three groups was born after the 37th week of gestation, this roughly means that maternal stature essentially affected the birth weight of near-term and term babies. At the same time, the measures of the mothers of true prematures differed only slightly from those of the full-term eutrophic group. This suggests that maternal height and weight have a less significant influence on the termination of preg-

nancy before the 37th week of gestation.

When examining the familial variation of fetal growth rate, our findings in the SGA, full-term AGA and LGA groups do offer support to the Ounsted theory. A strong familial tendency of intrauterine growth retardation was seen in the maternal relatives of SGA babies, and a predisposition to large birth weight was verified among the relatives of LGA infants. This familial component of growth variation is certainly operative on the female side. The male side was not investigated in this study, but its influence was excluded in the survey of Johnstone and Inglis [1]. The question whether the maternal transmission of the tendency to overgrowth or growth retardation is of merely genetic origin or it can be attributed to microsocial factors such as dietary habits as a result of shared learning experience in the family, cannot be answered.

Whatever the mechanism, the maternal regulation probably does not operate in the case of true prematurity. It seems most likely that the too early termination of a pregnancy is caused rather by various diseases, known and/or as yet unidentified biological and environmental factors than by maternal regulatory and genetic effects. As already mentioned, maternal stature, being itself a genetically controlled property, has a limited influence on premature birth.

In conclusion, normal or pathological fetal growth certainly depends on several endogenous and exogenous factors, the overlapping of which should be taken in account in the different types of birth weight variations. Our findings suggest that one of the essential differences between SGA and true premature infants is that maternal regulation plays a major role in intrauterine growth retardation but is insignificant in prematurity. This underlines again that statistics seeking the causes leading to low birth weight should always consider the gestational age of the neonate.

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