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Relationship of weight gain in infancy to subcutaneous fat and relative weight at $10\frac{1}{2}$ years of age*

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Mellbin, T. and Vuille, J.-C. (1976). *British Journal of Preventive and Social Medicine*, **30**, 239-243. **Relationship of weight gain in infancy to subcutaneous fat and relative weight at $10\frac{1}{2}$ years of age.** In a representative sample of 895 schoolchildren, aged between 9 years 10 months and 11 years 2 months, the risk of being overweight or obese was compared between those who had gained weight rapidly during infancy and those whose weight gain had been normal. A substantially increased risk ratio was found only in boys for whom a correlation analysis showed that the total weight gain during the first year of life was associated with the total body mass in relation to height, more or less independently of the degree of fatness at $10\frac{1}{2}$ years of age. In girls, a direct but very weak association was found between weight gain in infancy and the degree of fatness at $10\frac{1}{2}$ years. The implications of these findings with respect to aetiology and the possibilities of prevention are briefly discussed.

Previous investigations show that rapid weight gain in infancy is not a specific precursor of overweight at seven and at $10\frac{1}{2}$ years of age, and that there are marked sex differences (Mellbin and Vuille, 1973, 1976). Overweight was defined as the deviation of measured weight from standard weight for height. Obviously, this parameter not only reflects obesity—that is, an increased mass of adipose tissue, but it also includes cases with a heavy lean body mass. In so far as overweight is seen as a health hazard, only the increase of adipose tissue is probably of any significance (Mann, 1974). In the present study an attempt was made to answer the question whether and to what degree, excessive weight gain in infancy as a possible indicator of overnutrition is associated with an increased mass of body fat later in life. Experimental evidence suggests that the number of adipocytes present in the adult organism is relatively stable, that it is largely dependent on the rate of replication of these cells during the first year or years of life, and that this early replication is influenced by nutrition (Knittle and Hirsch, 1968). If these mechanisms are also valid in human beings, weight gain in infancy, which can be assumed to be positively correlated to the rate of replication of fat cells, should show a close correlation with estimates of the amount of adipose tissue later in life. In studies on large population samples direct measurements of total body fat are not feasible, but measurements of skinfold thickness seem to provide reliable estimates of the total mass of adipose tissue (Durnin and Rahaman, 1967; Durnin and Womersley, 1974).

Material and Methods

The original sample of 972 seven-year-olds for whom reliable weight data from the first year of life were available has been described previously (Mellbin and Vuille, 1973, 1976). Of these children, 895 (92%; 424 boys and 471 girls) were still living in the city of Uppsala or its suburbs when they were 9 years 10 months to 11 years 2 months old (average: $10\frac{1}{2}$

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years). These children were measured and weighed as described (Mellbin and Vuille, 1973). The same person who performed these measurements also determined the triceps and the subscapular skinfold thicknesses of every child, using a Harpenden caliper and the technique described by Tanner and Whitehouse (1962). Readings were made to the nearest 0·1 mm.

As in the previous reports, the weight data from the first year of life were used to assign every child to one of two cohorts:

Rapid weight gain was defined as either short spurts of excessive weight increase (above the 97th percentile during any four-month period), a total weight gain during the first 12 months exceeding the 90th percentile of the study population, or an attained weight at 12 months exceeding the 90th percentile. All children who fulfilled none of these criteria were placed in the *normal weight gain* cohort.

Moderate and *severe overweight* were defined as relative weight (100 × measured weight/standard weight for height) exceeding 110% and 120%, respectively.

Two degrees of obesity were defined according to the sum of the two skinfold values, choosing the limits in such a way that the same numbers of children were included in each of the two categories as in the two categories of overweight. This requirement, which was necessary in order to make the risk estimates for obesity comparable with those for overweight, was fulfilled by setting the limits for the sum of the skinfolds at 20·7 and 27·5 mm for boys and at 26·0 and 33·0 mm for girls. These values correspond to the 80–85th and 90–95th percentiles in British children (Tanner and Whitehouse, 1962) and to the 86th and the 95th percentiles in our material.

Results

The percentage distributions of skinfold values for children in the *rapid weight gain* and *normal weight gain* cohorts is shown in Figs 1 and 2. There is no marked difference between the two cohorts: for girls in particular there is almost complete overlapping. Table I summarizes the medians, the mean values, and the standard deviations for the triceps, subscapular, and combined skinfolds, and for all sex/weight-gain subgroups. The figures agree with those of Tanner and Whitehouse (1962) within one millimetre. All three parameters differ significantly between the sexes. The small differences between the

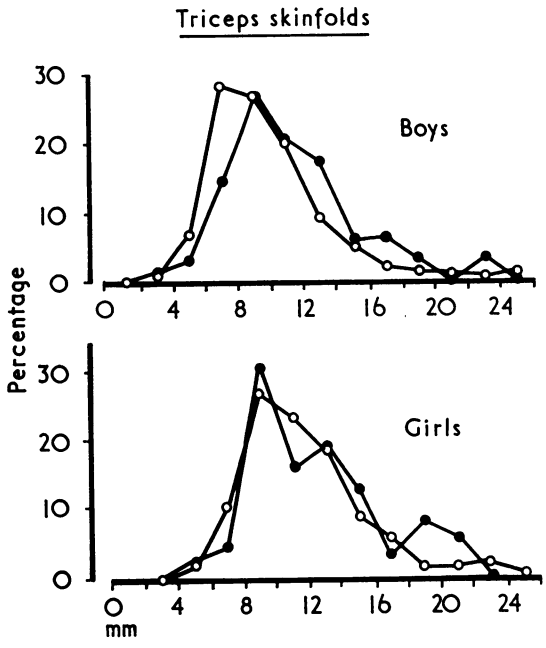

FIG. 1. Triceps skinfold values. Percentage distribution of the children in the *rapid weight gain* (filled circles) and the *normal weight gain* cohort (open circles).

rapid and the normal weight gain cohorts in boys are statistically significant both for the triceps and subscapular values separately and for their sum.

Table II shows the risk ratios of the two cohorts for *overweight* or *obesity*. The asterisks indicate the probability of the ratios being equal to 1·0. A substantially increased risk, with values of 3·32 and 2·05, was found for overweight but not for obesity in boys, whereas in girls none of the risk ratios was significantly different from 1·0.

The observation that the risk ratios in boys were higher for overweight than for obesity prompted an analysis of the hypothesis that the velocity of weight gain in infancy might be a predictor of lean body mass later in life rather than of the amount of fat. For this purpose the correlations between relative weight, skinfold values, and total weight gain during the first year of life were computed. In this way the whole range of values was considered, in contrast to the dichotomous categories used for the calculation of the risk ratios. In order to neutralize the contribution of fat to the relative weight, partial correlations were also computed. Triceps, subscapular, and combined skinfolds gave almost identical results; only those for the sum of the

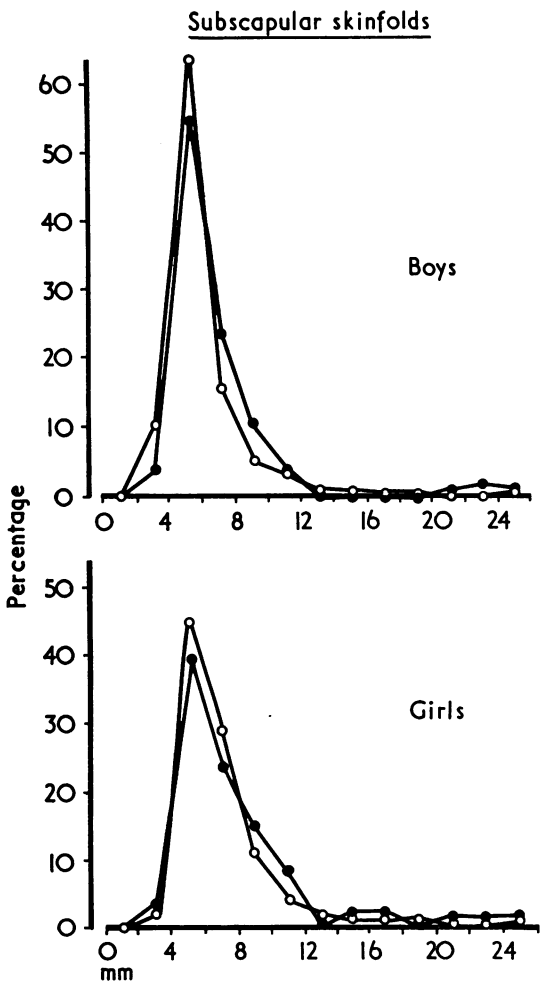

FIG. 2. Subscapular skinfold values. Percentage distribution of the children in the *rapid weight gain* (filled circles) and the *normal weight gain* cohort (open circles).

skinfolds are therefore presented (Table III). The following observations may be made:

1. Higher zero order correlations were obtained in boys, a finding which is consistent with the results presented above.

2. The partial correlations disclosed a particularly interesting pattern: In boys, the correlation to relative weight is affected only slightly if the interaction with skinfold thickness is taken into account.

In contrast, when controlling for relative weight, the correlation to skinfold thickness is totally abolished. In other words, weight gain in infancy seems to be related primarily to the total body mass in relation to height later in life, and it appears to be significant for the degree of subsequent fatness only in so far as this is a correlate of lean body mass. In girls, the reverse pattern is seen: the correlation to skinfold thickness is relatively stable, but that to relative weight is neutralized through controlling for the interaction with skinfold thickness—that is, there seems to be a weak but direct association between weight gain in infancy and the degree of fatness later in life.

DISCUSSION

The results presented in this study may be criticized on the grounds that no quantitative data on the nutrition during the first year of life were available, nor were there any direct measurements of the accumulation of body fat during this critical period. However, Fomon *et al.* (1975) found no evidence of a linear relationship between calorie consumption and fat deposition in human infants, at least not within the normal range. Similarly, Sveger *et al.* (1975) reported only minor differences in mean calorie consumption between groups of normal and overweight or obese infants. Thus, inclusion of nutritional data in our set of variables would probably not have helped very much. It could be argued, however, that if the estimation of the risk of future obesity had been based on skinfold data from the infant population rather than on the pattern of weight gain, the relationship between risk factor and outcome would have been more specific and hence quantitatively more important. However, the data from other studies do not support this objection. In the prospective study on 211 children by Hernesniemi, Zachmann, and Prader (1974), the correlations between skinfold measurements at 39 weeks and at 15 years were no higher than the correlations found in our study, and when the increments of skinfold thicknesses between 13 and 39 weeks were taken as the dependent variables, no significant correlations were found at all. In an attempt to derive an obesity index for infants, Crawford *et al.* (1974) studied 22 anthropometric variables in selected groups of clearly lean and clearly obese infants. An equation including weight gain from birth to six months, suprailiac skinfold and waist circumference gave the best discrimination between leanness and obesity, weight gain being the best single indicator of obesity at six months. Thus, the data published so far do not support the assumption that substantially different results might have been expected in our study if other estimates of fat deposition during the first year of life had been used.

TABLE I
SKINFOLD DATA

Site of Measurement	Sex	Weight Gain in Infancy	Median	Mean	SD	*t* Values
Triceps	Boys	Rapid	10·5	11·0	3·81	> $t = 3·18^{**}$
		Normal	9·1	9·7	3·42	
		Total	9·3	10·0	3·56	$t = 7·69^{***}$
	Girls	Rapid	11·7	12·3	3·86	
		Normal	11·0	11·7	3·79	> $t = 1·51$
		Total	11·2	11·9	3·81	
Subscapular	Boys	Rapid	5·7	6·7	3·71	> $t = 2·79^{**}$
		Normal	5·2	5·7	2·53	
		Total	5·2	6·0	2·89	$t = 6·48^{***}$
	Girls	Rapid	6·6	8·1	4·48	
		Normal	6·2	7·3	3·72	> $t = 1·87$
		Total	6·2	7·5	3·95	
Triceps + subscapular combined	Boys	Rapid	16·1	17·7	7·15	> $t = 3·16^{**}$
		Normal	14·2	15·4	5·65	
		Total	14·4	16·0	6·13	$t = 7·52^{***}$
	Girls	Rapid	18·3	20·3	7·89	> $t = 1·67$
		Normal	17·0	19·0	7·09	
		Total	17·3	19·4	7·33	

$^{**}P < 0·01$ $^{***}P < 0·001$

TABLE II
RISK RATIOS OF *RAPID WEIGHT GAIN* VERSUS *NORMAL WEIGHT GAIN* COHORTS FOR OVERWEIGHT AND OBESITY AT 10½ YEARS OF AGE

Criterion	Risk Ratio	
	Boys	Girls
Overweight		
Relative weight > 110%	2·05**	1·48
Relative weight > 120%	3·32**	1·61
Obesity		
Skinfold† >20·7 mm (boys)	1·75*	
>26·0 mm (girls)		1·45
Skinfold >27·5 mm (boys)	1·68	
>33·0 mm (girls)		1·75

† Sum of triceps and subscapular
*P <0·05 **P <0·01

$$\text{Risk ratio} = \frac{\text{No. overweight in } \textit{rapid weight gain} \text{ cohort} / \text{total no. in } \textit{rapid weight gain} \text{ cohort}}{\text{No. overweight in } \textit{normal weight gain} \text{ cohort} / \text{total no. in } \textit{normal weight gain} \text{ cohort}}$$

TABLE III
COEFFICIENTS OF CORRELATION OF TOTAL WEIGHT GAIN IN FIRST YEAR OF LIFE, SUM OF TRICEPS AND SUBSCAPULAR SKINFOLDS AT 10½ YEARS, AND RELATIVE WEIGHT AT THAT AGE

	Weight Gain	Relative Weight (*controlling for* skinfolds)	Skinfolds (*controlling for* relative weight)
Weight gain		0·274***	0·191***
Sum of triceps and subscapular skinfolds		*0·205****	*−0·045*
			0·794***
Relative weight (*controlling for* skinfolds)	0·136**		
	−0·013		
Skinfolds (*controlling for* relative weight)	0·173***	0·824***	
	*0·109**		

BOYS
GIRLS

*P <0·05 **P <0·01 ***P <0·001
Figures in italics indicate partial correlations

In two previous investigations (Mellbin and Vuille, 1973, 1976) we found a significant relationship between the pattern of weight gain in infancy and later overweight only in boys. The present analysis has now shown that this sex-specific phenomenon at 10½ years is almost exclusively due to an association between rapid weight gain in infancy and an increased total body mass in relation to height, whether or not this overweight is accompanied by an increased mass of adipose tissue. The relationship between rapid weight gain in infancy and later obesity (defined as skinfold thickness above certain limits) is weak, and there is no appreciable sex difference.

It has been suggested (Sjöström and Björntorp, 1974) that especially for the type of obesity that is characterized by an increased number of adipocytes, early onset and poor prognosis on the chances of successful weight reduction, could be caused by overnutrition in infancy. Naeye (1969) showed that increased cellularity in adipose tissue might be associated with hypercellularity in other tissues also. It appears likely that this may apply to many of the boys in our *rapid weight gain* cohort. Sjöström and Björntorp are inclined to see the cause of this

syndrome in environmental factors, especially nutrition. To the best of our knowledge there is as yet no proof of this assumption; the syndrome could equally well be genetically founded; in any case it appears difficult to explain the sex difference by the nutritional hypothesis. The lack of marked association between *rapid weight gain* in infancy and later 'pure' fatness—that is, fatness without concomitant increase of lean body mass, may be regarded as indicating that pure obesity appears later in life, probably under the influence of environmental factors, and that it corresponds to the hypertrophic type in the classification of Sjöström and Björntorp (1974).

Unfortunately, these results reinforce earlier conclusions (Mellbin and Vuille, 1973, 1976) that the prospects for prevention of obesity through more rigorous control of infant feeding practices are not very bright, at least not in Sweden, where childhood obesity is as serious a problem as in other countries in spite of a high degree of compliance with official recommendations concerning infant nutrition (Sveger *et al.*, 1975).

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