SESSION II

CHILDHOOD NUTRITION

Chairman: PROFESSOR OTTO WOLFF

Influence of nutrition in childhood on the origins of coronary heart disease

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Summary

Both the quality and the quantity of food ingested are relevant to the genesis of risk factors for coronary heart disease and the two are inseparable. Nevertheless they have a major common pathway through hypertension, which may well be the most important consequence of a high-protein, high-carbohydrate, high-fat, high-energy and high-sodium diet.

Because body fatness is a strongly genetically determined characteristic and because it evolves over the whole period of childhood a vigorous and sustained programme of health education is required at all levels. The aim of such a programme is to effect a small shift in the fatness of the whole population; such a shift would dramatically and disproportionately reduce the incidence of obesity. Since the morbidity and mortality which is found in obese subjects arises primarily from cardiovascular disease in general, and coronary heart disease in particular, nutritional influences have obvious relevance to the prevention of coronary heart disease.

Introduction

Both the quality and quantity of the diet in childhood seem likely to have a part to play in the genesis of risk factors for coronary heart disease. The constituents of the diet are important in determining many of the risk factors and the relationship of the quality as well as of the quantity of the diet has to be borne in mind.

Quality of dietary intake

Man eats a diet containing fat, protein and carbohydrates and, generally, without trying too hard,

manages to consume what we have come to call a balanced mixed diet. But diets, like everything else, are subject to fashion as well as to custom. Thus international epidemiological comparisons can be very fruitful.

Fat intake in children is associated with elevations of plasma lipids and especially of plasma cholesterol. The nature of the fat in terms of fatty acid chain length and composition may be relevant to the causation of atheroma but it is not the purpose of this paper to discuss it. There is, however, a considerable amount of data on the fatty acid composition of adipose tissue in obese and non-obese children, which can be summed up as follows (Brook, 1972).

The fatty acid composition of the triglyceride of adipose tissue reflects the fatty acid composition of the dietary fat. Adipose tissue of obese children contains significantly greater amounts of oleic (C.18:1) and palmitoleic (C.16:1) acids than that of control subjects and these fatty acids are also elevated in plasma triglyceride and non-esterified fatty acids. It appears that the proportion of palmitoleic acid in plasma triglyceride reflects the dietary intake of carbohydrate (Segall *et al.*, 1970). Consequently, the pattern in obese children is taken as evidence of increased carbohydrate feeding. Carbohydrate feeding may also affect the levels of plasma lipids, especially of triglyceride, but the major impact of carbohydrate is on insulin output.

Most fat is taken in conjunction with protein in childhood and there is little doubt that in the developed countries normal protein requirements are exceeded many times over. Whether this matters is not known but just as fats go with protein, so also

does sodium and in terms of being a blood pressure regulator, this is important, since hypertension is a major risk factor of coronary heart disease.

Much of the relationship between obesity and coronary heart disease may well be explained by the fact that, broadly speaking, levels of blood pressure mirror degrees of overweight. In children the data properly to discuss the relevance of obesity and salt intake to hypertension do not exist. Weight reduction variably affects hypertension; although it is sometimes accompanied by a fall in blood pressure, it is not clear whether the fall results from a loss of body fat or from a reduction in salt intake.

Quantity of dietary intake

To stress again the individuality of dietary requirements would be unnecessary but the prerequisite of obesity is, of course, the persistent intake of food in excess of requirements. As yet we have very little idea of what controls the intake of food in humans even though we know that they, like animals, can be induced, through lesions in the ventromedial nucleus of the hypothalamus, to increase food intake, to decrease activity and rapidly to gain weight. There is evidence that this syndrome of hypothalamic obesity is mediated by a rise in circulating plasma insulin and that this rise is the cause and not the result of the hyperphagia (Bray, 1977). Since vagotomy not only prevents but also reverses the hyperinsulinism of experimental animals subjected to hypothalamic surgery and abolishes the consequent weight gain, a neural mechanism has to be invoked to explain the phenomenon.

The question of whether the hyperinsulinism of simple obesity is the result or the cause of obesity is a vexed one but there seems little doubt that once it exists it perpetuates the obese state. Insulin resistance in obesity appears to occur because there is a diminution in insulin receptors in the obese state consequent upon hyperinsulinaemia; the level of circulating insulin inversely affects the concentration of insulin receptors (Bar, Harrison and Roth, 1977). As a result of this internal regulation it is possible to construct a model to explain the relevance of hyperinsulinaemia to obesity.

Increased food, and particularly carbohydrate food, raises plasma glucose levels; the consequent hyperinsulinaemia depresses the concentration of insulin receptors, which results in insulin resistance and a further rise in blood glucose. The consequent production of insulin ensures that a lipogenic stimulus is maintained and the only way this cycle can be broken is to withhold carbohydrates. The immediate fall in plasma insulin levels, with a consequent rise in the concentration of the insulin receptors, which follows institution of dietary control, is conspicuous. It occurs long before any sig-

nificant adipose mass change (Brook and Lloyd, 1973).

Adipose cells

Obese subjects, both adults and children, have large fat cells. In obese infants the number of adipose cells is also greater and seems to increase consistently during childhood, in marked contrast to normal subjects in whom it now appears that growth of the adipose organ, at least between the ages of 2 and 8 years, is largely achieved through increases in cell size. To have large fat cells is disadvantageous from the point of view of responses to hormonal lipolytic signals; in particular adipose cell responsiveness to adrenalin is known to be reduced and the responsiveness to other lipolytic hormones may also be blunted. Once again, a vicious circle is entered.

At last some longitudinal data on fat-cell development are beginning to appear (Knittle, 1977). From these data it seems that infants of obese parents may have a genetically determined propensity to hypercellularity. Whether in the long term there is a relationship between the degree of obesity and the number of adipose cells is still not known but the finding of an increased number of adipose cells in the normal weight infants of obese parents is extremely interesting.

Genetics of body fat

Hitherto it has been fairly widely held that obesity is one end of the normal spectrum of body fat and is a relatively homogeneous condition. Whilst it would generally be accepted that hypothalamic obesity, the obesity of Cushing's syndrome and the Prader-Labhart-Willi syndrome, to take three examples, are different from 'simple' obesity, it now seems likely that this too comprises a spectrum of conditions. In short, obesity is a symptom and a sign, not a disease entity.

There is now abundant evidence that obese infants do not necessarily remain fat (Poskitt and Cole, 1977); it is impossible to predict at the ages of 7 or 11 years how fat a person is going to be at the age of 16 years (Borjeson, 1977). There is every reason to believe that body fatness is an evolving quantity and not a fixed one. This is not to say that genetic influences are unimportant. The heritability of body fatness in childhood is high (Brook, Huntley and Slack, 1975; Borjeson, 1976; Garn and Clark, 1976) and children of obese parents are themselves at risk from becoming obese. Nevertheless there is, as yet, very little information on the values for heritability of body fatness in adults and they may be quite different from those in children.

Heritability is a measure of the proportion of the variation of a characteristic which is due to genetic influences. It is an estimate which applies, however,

TABLE 1. Longitudinal correlation coefficients of measurements of skin fold thicknesses obtained in childhood and repeated 15 years later

Age at first measurement	N	Males /females		pooled <i>r</i>
		<i>r</i> for triceps	<i>r</i> for subscapular	
3-5	27/22	0.41/0.45	0.47/0.40	0.43/0.42
6-8	26/36	0.48/0.57	0.52/0.50	0.50/0.54
9-11	28/36	0.62/0.55	0.57/0.47	0.60/0.51
12-14	20/28	0.66/0.12	0.74/0.36	0.70/0.24

only to the population in which it is measured. It may well change with sex, age, and race, to name only three variables, and it may be quite different for, say, triceps skin fold compared to subscapular skin fold. The data for adults are not available and are badly needed in the context of the causation of obesity in adulthood which is when obesity is mainly associated with the morbidity and mortality of coronary heart disease.

Natural history of body fatness

As far as obese children are concerned, it is impossible to predict what will happen to their obesity. As yet there are no longitudinal data on whether how fat you are as a child affects how fat you are as an adult. The author has set about trying to answer this question, which is particularly critical to the prevention of obesity. His co-worker, Dr Lorna Hawk, has been following-up subjects, who were aged 3 to 15 years when they were last seen, 15 years later. The data on the longitudinal correlation coefficients of the children of the first sixty complete families are shown in Table 1.

Here it is evident that in both sexes there is an increase in correlation coefficients of first and second measurements of triceps skin fold, subscapular skin fold and of the two skin folds pooled as the subjects got older at the time of the first measurement. This means that the older a child is, the more it resembles itself as an adult and this is, of course, quite inimical to the idea of a critical period for the determination of adult body fatness. There is a conspicuous lack of correlation in girls when the first measurement is made in puberty and this suggests that 'puppy fat' of puberty may not be a myth, at least in girls, and that what happens at that age may not be very relevant later. Taken overall these data clearly show that body fatness evolves and is not determined.

Part of the reason for the difficulty in girls may well be due to the much greater increase in body fat in puberty in girls and to the much greater variation in body fatness in adult women compared to adult men. The Ten-State Nutrition Survey (Garn and Clark, 1976) makes this last point very clearly.

Implications for prevention of obesity

A characteristic with high heritability needs a

strong and sustained environmental influence to alter it. That it can be altered is self-evident: there is no one who cannot lose weight, even if some people require very few calories to do so. The present indications are that no time is necessarily preferable to another for the prevention of obesity. Consequently a sustained programme of health education directed at all ages is required to shift the whole population distribution of body fatness downwards.

Tanner and Whitehouse (1975) recorded a marked increase in the standard values of skin fold thickness compared to the values previously published in 1962. Borjeson (1977), however, found that in Stockholm the whole child population of 7-year-olds in 1975 was not much fatter than a similar population in 1950, although the fat children now are much fatter than they were 25 years ago. In any screening programme, therefore, the level at which intervention is required to prevent obesity in individual subjects is probably lower than we are accustomed to providing.

Acknowledgment

I am very grateful to Dr Lorna Hawk for allowing me to use a very preliminary analysis of a small amount of her data to illustrate this paper (*see under* 'Natural history of body fatness').

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