

Is the Child with Poor Growth Achievement More Likely to Die of the Measles?

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EASLES is typically a benign child-hood illness, but an attack carries the risks of complications and sequelae. These may range from residual behavior disorders with little physical dysfunction to more immediate serious complications and even death.

Despite the widespread use of measles vaccine, the number of deaths attributed to measles in the United States has only halved in the ten years between 1957 and 1966.

In developing countries, high case mortality rates from measles have been associated with malnutrition rather than with differences in viral virulence.² The recent reports of malnutrition in the United States ³ suggest that malnourished infants and children in this country are also at particular risk.

There is no single objective index of nutritional status. This makes identification of the individual malnourished child in the community difficult. But since one of the first effects of serious malnutrition in infants and children is growth retardation, it can be argued that children who are clearly "underweight" or "underheight" for their age may be looked upon as more vulnerable to serious measles.

In order to test this hypothesis we looked into the past growth records of 155 fatal cases of measles. Useable height, weight and age data were procurable for 75 cases (68 Michigan children and seven California children).

From the Nutrition Unit, School of Public Health, The University of Michigan, Ann Arbor, Mich. 48104. These data indicated that approximately one-half of the girls and one-third of the boys were at or below the third percentile of Iowa standards in height and weight prior to their terminal illness (Table 1). Shortness of stature was particularly marked in girls under five years of age. At five years of age and over, in height achievement both sexes showed approximately one out of four at or below the third percentile.

The record of height provides a reliable index of growth performance prior to the onset of the illness. It is influenced by pathologic processes and inherited ethnic influences. Six of the children were reported to be suffering from mongolism (Down's syndrome).

No other pathologic conditions that might have been responsible for the deficit in height were revealed in the death certificates. The deaths were scattered throughout a broad cross section of the several racial groups which constitute the population of Michigan.

Weight is not as good an index of growth, and hence the weight deficits also observed might have been attributable to the terminal illness. Low-birth-weight babies tend to be underweight for age, but since birthweight is not recorded on death certificates it could not be determined whether immature or premature babies were contributing to the underweight group.

Since the present sample is small, the results may be looked upon as suggestive rather than conclusive. They do show, nevertheless,

GROWTH RETARDATION AND MEASLES

TABLE 1. Fatal Measles Cases Failing to Exceed the Third Percentile for Height and Weight by Age and Sex

	Under 5 Years		5 Years and Over		All Ages	
	Male	Female	Male	Female	Male	Female
	No. %	No. %	$\overline{\text{No.}}$ $\frac{c_{e}}{\sqrt{e}}$	No. %	No. %	No. %
Height Weight	8 38.1 9 36.0	11 64.6 15 62.5	2 28.5 2 25.0	3 25.0 6 33.0	10 35.7 11 33.3	14 48.4 21 50.0

that most of the children in the sample who died from measles between 1958 and 1967 were both underweight and underheight. That this was due to malnutrition at the time of the terminal illness is conjectural. The more important question is: Are children who are "underweight" and "underheight" for age from any cause more likely to succumb to measles than those of normal stature? If subsequent studies lead to an affirmative answer to this question, then special efforts

should be made to identify these children and to make sure that they receive the protection of vaccination.

References

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- Gordon, J. E., Jansen, A. A. J. and Ascoli, W.: Measles in rural Guatemala. J. Pediat. 66: 779, 1965.
- 3. Schaefer. A.: Report to the Senate Select Committee on Nutrition and Human Needs, April 1970.

Single Cell Protein

Single cell protein could be produced industrially in almost unlimited quantities, independently of agricultural land or climate. It is thus the subject of much current interest and research and can be expected to increase mankind's protein resources, whether used for animal or for human feeding. . . .

The main sources of single cell protein are yeasts and bacteria. Yeasts have been consumed for many years, but only in small quantities per person. During the Second World War, food yeast (Torula utilis) was consumed in increased

quantities in Germany, the USSR, and some other countries, providing valuable B-complex vitamins and making a moderate contribution to protein supplies.

A species of algae has long been consumed by populations around Lake Chad, Africa, but human experience of eating single cell protein of bacterial origin is rare.

Even when demonstrably free from acute and chronic toxicity, the amount of single cell protein that could be consumed is probably limited by its RNA content, among other possible factors. Rapidly growing cells have a high concentration of ribonucleic acid, which in man and higher apes can be excreted only as uric acid. High uric acid levels in blood and urine are unacceptable because they can result in renal stones or gout in susceptible individuals.

It is reasonable that most single cell proteins will first be used in animal feeding over several generations before they are introduced into human diets; their use in animal feeding will release conventional proteins for human nutrition.

—IVHO Chronicle, February 1970.

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