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MALNUTRITION AND INFECTION IN A TYPICAL RURAL GUATEMALAN VILLAGE: LESSONS FOR THE PLANNING OF PREVENTIVE MEASURES

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A long-term prospective study of fetal and postnatal growth and possible influences on growth was initiated in 1963 in a Guatemalan village. A high proportion of the infants were of low birth weight and they were more at risk to death in the first year of life than infants with higher birth weights. The latter also had a better chance of surviving the critical weaning period. There was a very high prevalence of infection in infancy and childhood, and a high proportion of infants with elevated cord IgM levels suggests that there had been intrauterine antigenic stimulation.

Postnatal growth was generally satisfactory for the first 8–16 weeks of life but by one year the majority of the children were below the standard for height and weight, and growth continued to be unsatisfactory until the third or fourth year. There was evidence that growth was being retarded by infections and that they had their greatest effect during the second year of life. It is suggested that maternal infections are an important component of the etiology of fetal growth retardation and that planning of priorities for maternal and child health in developing countries should take this into account.

INTRODUCTION

Approximately two-thirds of the total world's population live under deficient socio-economic conditions. The populations in such ecosystems, whether in Asia, Africa, or Latin America, show high illiteracy rates, are exposed to poor environmental sanitation, and have deficient nutrient intakes. Under such circumstances, the individual has less opportunity to express his optimal genetic potential in terms of growth and development. Observations made in many developing nations have revealed that retardation in physical growth is remarkably similar in different geographical regions, pointing out to a universal phenomenon (Béhar, 1968). Retardation in physical growth, within the limits usually observed has no significance *per se;* however, it acquires great significance when used as an indicator of a similar degree of retardation in physiological and psychological development as many studies throughout the world seem to indicate. Furthermore, the high frequency of physical retardation in children often is accompanied by high rates of morbidity and mortality. Therefore, physical growth can be considered an indicator of inadequate nutrition and poor health.

It seems of paramount importance to know the relative significance of the factors associated with deficient growth and development as a basis for the design and implementation of measures that will prevent or correct such deficiencies in a more efficient manner and with the minimum of effort. Traditionally, deficient diets have been recognized as main determinants of growth failure in children of developing nations. Infection, however, has emerged as an important factor, often as important or more important than the particular diet or a region. Due to deficient sanitation, crowding, poor housing, and chronic malnutrition, infection is present at all stages of human growth, including the period of fetal development. A wealth of available technology could be applied to improve the situation of nutrition and infection in most areas. However, there are unknowns requiring further scrutiny in order to establish better the nature of the problem and be able to make optimal use of the knowledge and resources.

INCAP FIELD STUDY OF MALNUTRITION AND INFECTION

The objective of this communication is to comment on selected observations obtained in a typical highland Indian village of Guatemala, Santa María Cauqué, where INCAP has conducted a

long-term prospective study during the past 9 years (Mata, Urrutia and García, 1967). Experience has shown that, although there are differences in ways of life with villages in the lowlands of Guatemala, the principal factors that lead to malnutrition and infectious diseases are fundamentally the same throughout the country. The community then is quite typical and representative of many localities of Guatemala, and appears to be similar, from the public health standpoint, to communities in the highlands of Mexico, Central and South America. This makes the present observations applicable to a larger geographical region. Furthermore, the growth patterns of children in the study village are remarkably similar to those reported for children in many African and Asian populations. Therefore, it is expected that the information obtained in the Guatemalan village has a wider general significance.

Observations began in Santa María Cauqué in 1963 after establishing rapport with the villagers, mainly through implementation of a health clinic. A long-term prospective study was initiated in 1964 with the objective of better understanding the basic problems of poor growth performance and the interactions of growth, infection and diet. The village presently has 1,500 inhabitants; its characteristic features are a low socio-economic development, deficient housing, poor sanitation, and deficient diets (Mata, Urrutia and García, 1967; Mata, Urrutia and Lechtig, 1971). Seventy per cent or more of the energy and proteins consumed are derived from corn, a food whose protein is deficient in lysine and tryptophan. Beans, rice, bread, vegetables and a variety of weeds and fruits are consumed in small amounts. Animal proteins are consumed only occasionally and mainly by the adults.

Fetal Growth

Virtually all babies born during the period of research were studied shortly after birth. This was achieved by having nurses living in the village, and obtaining good rapport with the folk midwives and the community as a whole. A high frequency of fetal growth retardation was observed; of the total births, 9 percent were premature by weight and gestational age, and an additional 32 percent were "small for date" (Table I) (Mata, 1971). The high frequency of low birth weight infants cannot be explained solely on the basis of high altitude, (10,000 feet and higher) since the village was at 6,000 feet elevation. No data on fetal wastage were produced by this study. When these results and other information on fetal infection became evident, collection of data on fetal loss was begun in 1972. The limited findings cannot be presented yet.

TABLE I

Fetal growth in 367 live newborns. Santa María Cauqué, Guatemala, 1964-1971 (Means ± S. D., and range).

Time of gestation, weeks	Birth weight, g	Number	Percentage of all babies	Birth weight for group
< 37	[a]	33	9.0	1,713 ± 368 (783 – 2,565)
> 37	> 2,500	116	31.6	2,272 ± 248 (1,600 – 2,493)
> 37	> 2,500	218	59.4	2,789 ± 218 (2,504 – 3,562)

[a] All but one had birth weight less than 2,500 g.

The study revealed a good correlation between fetal growth and survival. Infants with low birth weight died more often during the first year of life. This became evident when newborns were grouped in four categories (Table II) arranged according to the Mean ± S.D. for birth weight in this series of babies (2,540 ± 409 g) (Mata, 1971). Babies born with at least 2,900 g survived the first year of life, despite an environmental situation that favored malnutrition and frequent infection. An examination of the survival of cohort children that had been (or could have been) followed for at least three years, showed that those born with a birth weight of 2,900 g or more, had managed to live through the critical second and third years. This period of life coincides with a time when weaning is completed and infection and malnutrition are more evident. On the other hand half of infants born with a weight of less than 2,100 g died in the first year of life, two-fifths died in the neonatal period and three-fifths in the post-natal infant period. This mortality was despite the availability in the village of medical treatment provided by an experienced pediatrician (Mata, 1971; Urrutia, Mata and García, 1971). The association of low birth weight and increased mortality holds for other Guatemalan villages, and appears to be a universal phenomenon.

Most of the information on fetal growth available for developing nations has been derived from hospital records and reflects the situation of the middle and high socio-economic strata, and of selected groups of the low strata with access to

TABLE II

Infant deaths in relation to birth weight in 323 live newborns. Santa María Cauqué, Guatemala, 1964–1970.[a]

Birth weight, g	Number of infants	Deaths[b]					
		Neonatal		Post-neonatal		Total	
		Number	%	Number	%	Number	%
< 2,131	42	8	19	13	31	21	50
2,131–2,539	89	3	3	3	3	6	7
2,540–2,949	152	2	1	7	5	9	6
> 2,949	40		0		0		0
Total	323	13	4	23	7	36	11

[a] Adapted from Mata *et al.* (1972a).

[b] This Table pertains to 323 infants observed for at least one year, among the original 367 (Table I). Attrition was due to migration or to the fact that not all infants were one year old when the Table was made.

hospital services. There is practically no information regarding fetal growth in the lower rural socio-economic groups of developing nations, nor is there much knowledge regarding the significance of low birth weight in post-natal life (except for prematures by weight and gestational age). Therefore, it seems mandatory to conduct similar studies in other regions of the world to expand the present observations. This acquires greater importance in view of the limited emphasis of care during pregnancy in the public health programs of many developing countries.

Possible Causes of Fetal Growth Retardation

The two most likely causes of fetal growth retardation and prematurity are inadequate maternal nutrition and infection during pregnancy. Although there is a wealth of evidence in experimental animals stressing the importance of maternal nutrition on fetal growth, epidemiological observations and experimental studies in humans, although suggestive, are not conclusive (Rosa and Thurshen, 1970; Thomson, 1963); more research is needed to establish this concept for the human host. INCAP studies pointed out a close association between caloric intake during pregnancy and fetal growth (Lechtig *et al.*, 1972). Calorie supplementation during pregnancy resulted in better fetal growth for women ordinarily living on a deficient diet. In the village studied the nutrient intake of mothers was definitely deficient in calories, protein, vitamin A and riboflavin (Mata *et al.*, 1972a). Furthermore, protein was derived from corn, which has a low biological value. Although the iron intake appeared adequate, it was derived mainly from pulses and other vegetables, and therefore, it was not readily available to the host.

Maternal nutrition is not the only environmental factor influencing fetal growth (a general misconception in developing nations where malnutrition is prevalent). The Santa María Cauqué study revealed an overwhelming force of infection affecting the human host at all ages and virtually at all times. This influence of infection has become apparent through the systematic microbiological study of rural children from birth to age three years (Mata, Urrutia and García, 1967; Mata, Urrutia and Lechtig, 1971; Mata *et al.*, 1972b). The overwhelming infection seen in such a population suggests that maternal and fetal infection is much greater than in industrialized societies. In this regard, examination of umbilical cord sera of more than 90 percent of the newborn population revealed that approximately one half contained elevated values of immunoglobulin M (IgM) (Table III). Since this macromolecule normally does not cross the placental barrier, its presence in the blood of

TABLE III

Cord serum immunoglobulins (mg/ml) in 306 neonates. Santa María Cauqué, Guatemala, 1964–1971.[a]

Class	Mean ± S.E.	Range	Percent of high values[b]
IgM	0.371 ± 0.024	0.0 – 3.2	56.2
IgA	0.087 ± 0.012	0.0 – 1.7	29.4
IgG	13.90 ± 0.247	6.85–28.8	

Source: Mata *et al.*, (1972b).

[a] All cord serum specimens with an IgM/IgA ratio below 1.5 were excluded from the tabulation, suspected of being admixed with maternal blood.

[b] High values: IgM > 0.20 mg/ml
IgA > 0.10 mg/ml

the neonate is presumed indicative of synthesis of fetal antibody as a response to antigenic stimulation. A possible explanation for the high frequency of elevated IgM in the newborn population of Santa María Cauqué is increased infection in the mother and in the fetus. The fetus may produce antibody to antigens present in the maternal circulation, or more likely, to agents replicating in the embryonic membranes, or in the fetal tissues. Studies in industrialized societies have indicated that a high percentage of intrauterine infections lead to high levels of cord IgM. These observations, confirmed in two additional highland and four lowland Guatemalan villages, as well as in similar areas of Peru (Lechtig and Mata, 1971; 1972), suggest an increased frequency of intrauterine antigenic stimulation.

Possible congenital infections on the study village were investigated: five of 60 newborns appeared infected with enteroviruses at birth; the prevalence of *Toxoplasma* infection among 300 neonates was about 10 times that occurring in industrial societies; at least 1.5 percent of the babies had been infected *in utero* with cytomegaloviruses (CMV) or another herpesvirus (Mata *et al.*, 1972b). The investigations conducted at INCAP aimed at only a few of the known agents that cause intrauterine infection. It is known that many other viruses, as well as bacterial agents may infect the fetus (Monif, 1969; Davies, 1971). The present observations suggest that the total frequency of intrauterine infection could be more elevated than reported herein.

Studies in industrialized countries have pointed out that intrauterine infections, particularly those caused by rubella, cytomegaloviruses and *Toxoplasma* are a cause of low birth weight, and of deficient physical and mental post-natal development. The INCAP study has not yet provided evidence on how much of the high frequency of

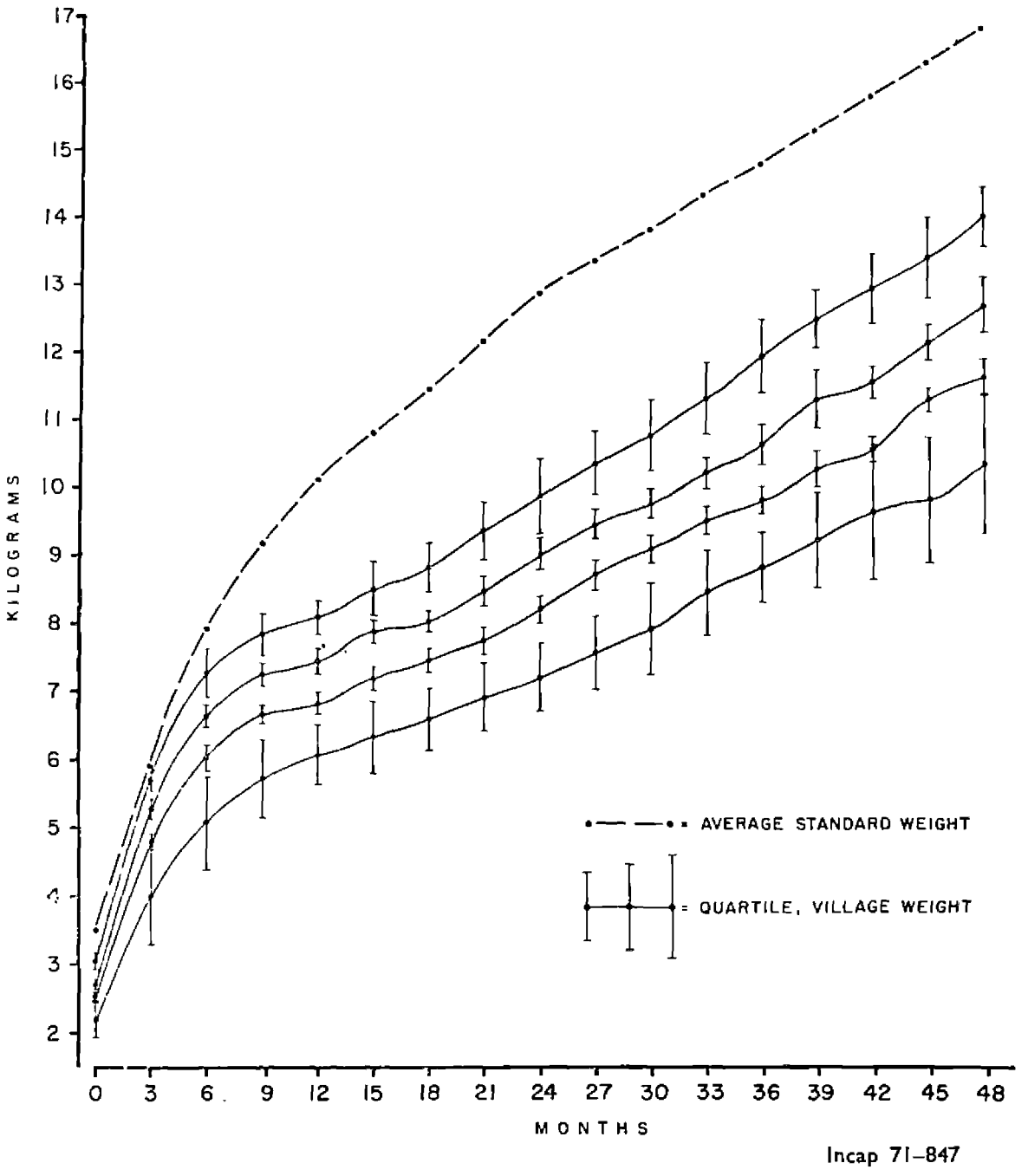

FIGURE 1 Average weight curve of village children, by quartiles, compared with the Jackson and Kelly standard for Iowa children. Santa María Cauqué, Guatemala, 1964–1970 (Mata *et al.*, 1972b).

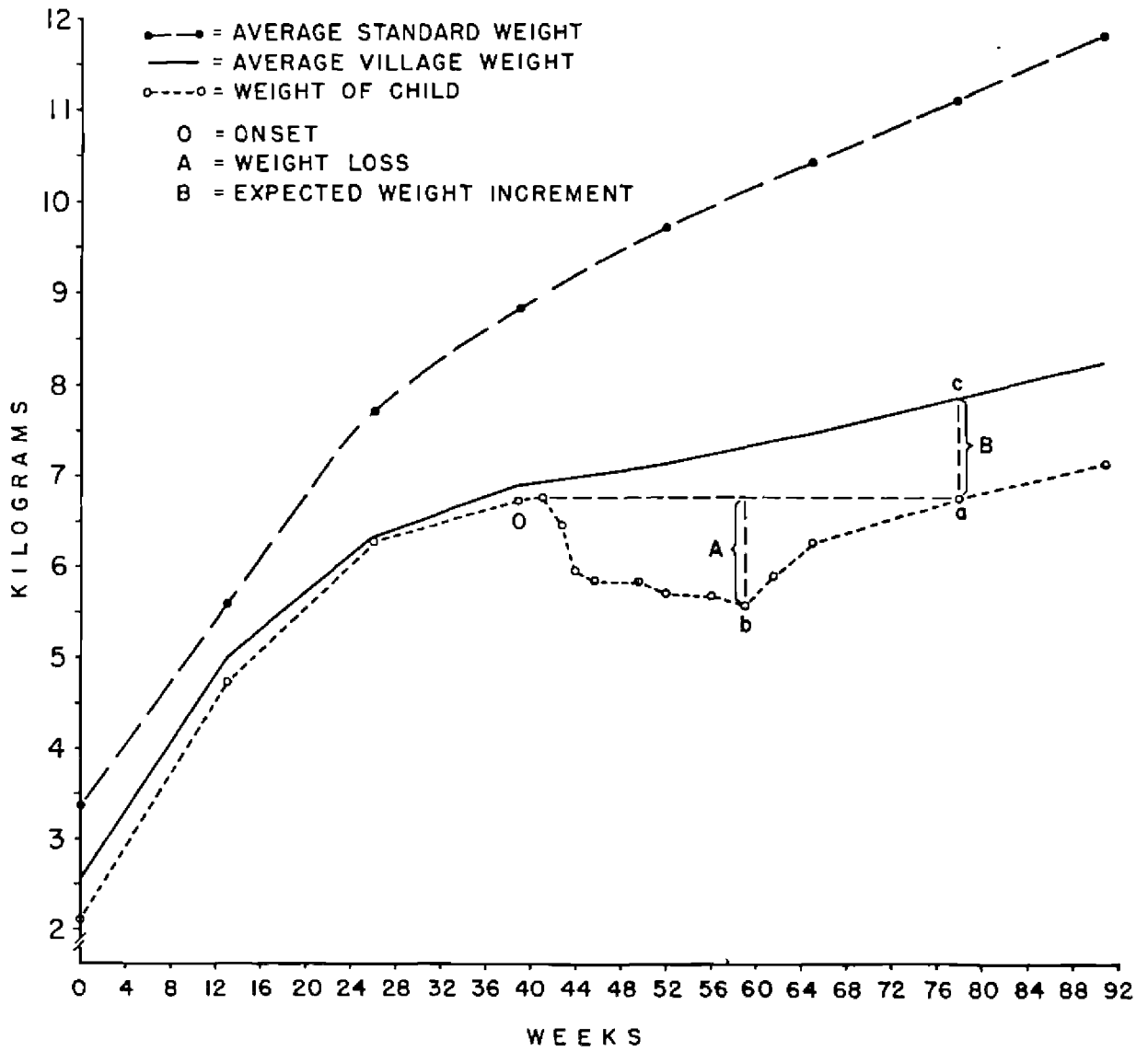

FIGURE 2 In this boy from Santa María Cauqué, there was severe weight loss following the onset of whooping cough; recuperation was also prolonged. The child's weight is compared with the average weight curve for village children and the Jackson and Kelly standard. (Mata *et al.*, 1972b).

fetal growth retardation is due to intrauterine infection. Nevertheless current knowledge relating to the effect of maternal and fetal infection on fetal growth and development, and the evidence that fetal malnutrition and infection are more prevalent in the study area, point to their likely interrelationship.

Post-Natal Growth Retardation

Most infants in Santa Maria Cauqué showed an adequate growth rate in the first eight to 16 weeks of life, despite the low birth weights observed. Height and weight curves in the first few months had the same slopes as those observed in children of industrialized societies (Figure 1.) Around the third month of life, infants departed from most accepted standards, and by one year of age, the great majority were below the standard for height and weight (Mata *et al.*, 1972b). The decreased growth rates continued throughout the second year of life. After the third or fourth year, children usually improve their dietary intake, and are already immune to many of the infections. Thus the growth rate improves to reach values that are similar to those of well-nourished children. Growth curves (weight and height) are parallel to those of the standard (Béhar, 1968); and indicate that there is no significant catch-up; throughout this time their weight for height is usually within normal limits.

It must be pointed out that all infants in this community are almost exclusively breast-fed during the first three months of life, with very small amounts of complementary foods given during the rest of the first year. Solid foods in significant amounts (corn preparations, bread, noodles, beans, vegetables) are progressively introduced only after one year of age. In the series of birth weight values the mode for complete weaning was 28 months. Cow's milk is not given during the weaning process or thereafter.

Careful examination of the growth curves of the children participating in this prospective study demonstrates weight losses resulting from infections

such as diarrheal disease, measles, bronchopneumonia and whooping cough, as illustrated in Figure 2 (Mata *et al.*, 1972b). The negative effect of infection on nutrition, growth, and survival is more marked in the first two years of life (Mata *et al.*, 1972b; Gordon, Wyon and Ascoli, 1967; Puffer, Serrano and Dillon, 1971). The mechanisms comprise reduction of intake of food, impaired absorption and utilization of nutrients, increased losses and demands for nitrogen, and other physiologic alterations. In the metabolic ward, infections have been shown to be responsible for a host of cell-function alterations and significant nitrogen losses (Beisel *et al.*, 1967). The negative nitrogen balance may last for days or weeks (Scrimshaw, Taylor and Gordon, 1968). Even asymptomatic infections like those caused by attenuated viruses may induce negative nitrogen balances in children recovered from malnutrition (Gandra and Scrimshaw, 1961), a fact to be remembered in view of the high rates of infection experienced by poor children at all times (Mata *et al.*, 1972a). Animal experimentation (Holt, Halac, and Kajdi, 1962) showed greater nitrogen losses with higher nitrogen intake when the animals were submitted to stress (infection or trauma). Based on this work it has been suggested that, although infectious disease induces a greater negative nitrogen balance in well-nourished children than in malnourished ones (Holt, Halac and Kajdi, 1962), the significance for poorly nourished children is expectedly greater. The reason for this is that these children are receiving poor diets, they have decreased tissue reserves and, therefore, they cannot cope with the increased demands required during the process of defense and repair. Their poor dietary intake also limits the repletion period that follows an infection in well-nourished individuals (Scrimshaw, Taylor and Gordon, 1968).

Consequently, it is easy to understand why children in developing societies easily lose weight during illness, and why they are often precipitated into severe protein-calorie malnutrition. Furthermore, in the particular study village, and in many other preindustrial communities, food is often suppressed or substantially diminished when the child is ill, unfavorable qualitative changes are introduced in the diet, and equivocal treatments are instituted.

Finally, there is also evidence that frequent subclinical infections, for example with enteroviruses, may be related to growth. It has been found that children that had experienced more infections with enteric viruses in the first three or six months of life had poorer growth curves than children who had fewer infections in the same period of time (Mata *et al.*, 1972a).

COMMENTS

Although some of these observations are still preliminary, several conclusions of interest to public health practice in preindustrialized areas of the world can be derived. In the first place, the very high proportion (around 40 percent) of babies weighing less than 2,500 g at birth, the majority of whom are born at term, should be emphasized. This proportion seems to be more realistic than those usually reported in preindustrial countries, which are derived from hospital statistics, and, therefore, only represent selected segments of the population. The greater mortality risk for low birth weight babies, particularly prematures, during the neonatal period has been documented (Mata, 1971; Mata *et al.*, 1972a; Corsa *et al.*, 1952; U.S. Department of Health, Education and Welfare, 1972). Of importance is the fact that, under the conditions of the study village, this high risk persists throughout the first year of life (Mata *et al.*, 1971; Mata *et al.*, 1972a). Since low birth weight may be associated mainly with conditions of poor health in the mother, the evidence here presented further substantiates the value of infant mortality as an indicator of the overall health situation of population groups. Significant efforts are being made in underdeveloped nations to reduce the exceedingly high rates of infant mortality. In this regard, specific goals were set by the American countries in the Charter of Punta del Este (Organización de Estados Americanos, 1967), but the majority of nations failed to attain these goals during the first decade (Organización Panamericana de la Salud, 1972). What seems clear is that programs predominantly oriented towards infants and small children would be limited in their action if the overall health condition of the population, and particularly of pregnant women, is not properly considered.

Prenatal care programs in developing countries often are patterned after those of developed nations. Frequently, great emphasis is given to measures oriented toward detection or prevention of complications such as eclampsia or hemolytic disease due to Rh incompatibility. But both of these conditions are known to be extremely rare

diseases in these indigenous populations, while not enough attention is given to the much more common and important problem of malnutrition and infectious disease.

With respect to maternal factors associated with low birth weight, no final conclusions can be derived from the observations. Nevertheless, there is evidence from other INCAP studies that maternal malnutrition, particularly energy deficiency (Lechtig, Habicht, de León and Guzmán 1972) plays a role. This finding is supported by the frequent observation of deficient diets and malnutrition in mothers from our study village, (Mata, Urrutia and Lechtig, 1971; Mata *et al.*, 1972a). We suggest the hypothesis that maternal infections, with or without replicative phenomena in fetal tissues, are an important component of the etiology of fetal growth retardation. Further studies are needed to confirm this hypothesis; at the same time, these observations could provide a more rational basis for identification of priorities for maternal and child health planning in developing countries.

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REFERENCES

Béhar, M. (1968). Prevalence of malnutrition among preschool children of developing countries. *In* Scrimshaw N.S. and J. E. Gordon (Eds.), *Malnutrition, Learning, and Behavior*. Proceedings of an International Conference co-sponsored by the Nutrition Foundation, Inc. and the Massachusetts Institute of Technology, held at Cambridge, Mass., March 1–3, 1967. The MIT Press, Cambridge, Mass., p. 30–42.

Beisel, W. R., W. D. Sawyer, E. D. Ryll and D. Crozier (1967). Metabolic effects of intracellular infections in man. *Ann. Int. Med.* **67**, 744.

Corsa, L., T. F. Pugh, T. H. Ingalls and J. E. Gordon (1952). Premature birth as a problem of human populations. *Amer. J. Med. Sci.* **224**, 343.

Davies, P. A. (1971). Bacterial infection in the fetus and newborn. *Arch. Dis. Child.* **46**, 1.

Gandra, Y. R. and N. S. Scrimshaw (1961). Infection and nutritional status. II. Effect of mild virus infection induced by 17-D yellow fever vaccine on nitrogen metabolism in children. *Amer. J. Clin. Nutr.* **9**, 159.

Gordon, J. E., J. B. Wyon and W. Ascoli (1967). The second year death rate in less developed countries. *Amer. J. Med. Sci.* **254**, 357.

Holt, L. E., E. Halac Jr. and C. N. Kajdi (1962). The concept of protein stores and its implications in diet. *J. Amer. Med. Assoc.* **181**, 699.

Lechtig, A., J. P. Habicht, E. de León and G. Guzmán (1972). Influencia de la nutrición materna sobre el crecimiento fetal en poblaciones rurales de Guatemala. II. Suplementación alimentaria. *Arch. Latinoamer. Nutr.* **22**, 117.

Lechtig, A. and L. J. Mata (1971). Levels of IgG, IgA, and IgM in cord blood of Latin American newborns from different ecosystems. *Rev. lat-amer. Microbiol.* **13**, 173.

Lechtig, A. and L. J. Mata (1972). IgM and C3 in serum of Peruvian mothers and cord blood of their infants. (Cartas al Editor). *Arch. Latinoamer. Nutr.* **22**, 309.

Mata, L. J. (1971). Field studies on nutrition and infection. *Protein Advisory Group Bulletin No.* **11**, Protein Advisory Group of the United Nations, New York, p. 18.

Mata, L. J., J. J. Urrutia, C. Albertazzi, O. Pellecer and E. Arellano (1972a). Influence of recurrent infections on nutrition and growth of children in Guatemala. *Amer J. Clin. Nutr.* **25**, 1267.

Mata, L. J., J. J. Urrutia, A. Cáceres and M. A. Guzmán (1972b). The biological environment in a Guatemalan rural community. *In* White, P. L. (Ed.), *Proceedings Western Hemisphere Nutrition Congress III, August 30-September 2, 1971, Miami Beach, Florida.* Futura Publishing Co., Inc., Mount Kisko, N.Y., p. 257–264.

Mata, L. J., J. J. Urrutia and B. Garcia (1967). Effect of infection and diet on child growth: experience in a Guatemalan village. *In* Wolstenholme, G. E. W. and M. O'Connor (Eds.), *Nutrition and Infection.* J. and A. Churchill Ltd., London, Great Britain, p. 112–126. (CIBA Foundation Study Group No. 31).

Mata, L. J., J. J. Urrutia and A. Lechtig (1971). Infection and nutrition of children of a low socio-economic rural community. *Amer. J. Clin. Nutr.* **24**, 249.

Monif, G. R. G. (1969). *Viral Infections of the Human Fetus.* 1st ed. The MacMillan Company, London, p. 164

Organización de Estados Americanos (OEA) (1967). Documento Oficial, Sec. XII. 1, Rev. 2 (español), p. 30.

Organización Panamericana de la Salud (1972). *Informe Anual del Director de la Oficina Sanitaria Panamericana*, **1971**. Washington, D.C., p. 371. (Documento Oficial de la OPS No. 116).

Puffer, R. R., C. V. Serrano and A. Dillon (1971). *Inter-American Investigation of Mortality in Childhood.* Pan American Health Organization (PAHO/WHO), Washington, D.C., p. 160.

Rosa, F. W. and M. Thurshen (1970). Fetal nutrition *Bull. Wld Hlth Org.* **43**, 785.

Scrimshaw, N. S., C. E. Taylor and J. E. Gordon (1968). *Interactions of Nutrition and Infection.* WHO Monograph Series No. 57. World Health Organization, Geneva, p. 329.

Thomson, A. M. (1963). Prematurity: Socio-economic and nutritional factors. *Modern Problems in Pediat.* **8**, 197.

Urrutia, J. J., L. J. Mata and B. García (1971). Relación entre el peso del recién nacido y su supervivencia. *Arch. Latinoamer. Nutr.* **21**, 222.

U.S. Department of Health, Education and Welfare (1972). *A Study of Infant Mortality from Linked Records, by Birth Weight, Period of Gestation, and other Variables. United States,* **1960** *Live-Birth Cohort.* DHEW Publication No. (HSM) 72–1055. Department of Health, Education and Welfare, Bethesda, Maryland. p. 90.