Environmental Contamination and Chronic Inflammation Influence Human Growth Potential

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Short stature (stunting) having become a diagnostic standard for chronic malnutrition, has introduced some confusion and thus, development of an incomplete research and programmatic paradigm regarding the determination of the size of the infant and young child, specifically regarding the role of diet. An adequate supply of nutrients is necessary, but not sufficient to guarantee normal linear growth. Hygienic factors also profoundly influence growth.

Livestock reared in unsanitary conditions grow poorly and produce reduced amounts of meat. The science of animal husbandry recognizes that either cleaning up the environmental conditions or adding antibiotics to pig fodder can improve growth and enhance meat production. The antitrrophic effects of mobiliizing and maintaining an inflammatory response directly antagonize the processes of anabolic metabolism (1). It is not an exaggeration to say that, due to poverty and misery, many of the underprivileged of the world experience living conditions that are tantamount to a dirty pig sty.

Indeed, dietary deficiency is not the main determinant of impaired growth in early life. Based on a review of the literature, Brown and Solomons have suggested that somewhat <40% of the variance in linear growth retardation can be ascribed to diet (2). Campbell et al., in a study from the Gambia in this issue (3), acknowledge with their second reference citation (4) some inspiration that they gained from a theory-oriented review article which my colleagues and I wrote a decade ago. It provided them with the insight that “an alternative explanation for infants in developing countries is that faltering growth occurs as a consequence of chronic or recurrent exposure to infection brought about by living in an unhygienic and unsanitary environment.” As a point of historical fact, it was not infection (if that is to be understood as recurrent episodes of symptomatic illness studied in Campbell et al., references 6, 7 and 27) that we were focusing on in our 1993 review (4). Rather, our focus was the continuous burden of chronic immunostimulation by environmental antigens that keeps the acute phase response working overtime. This phenomenon is so elegantly, ethically, and even quantitatively genes to the systemic milieu will require redress (3). Thus, along with collateral and synergistic improvements in the way that low income societies select and prepare their diet, attention to avoiding infections, reducing inequalities and caring for their environment will reduce the barriers to the growth process. Such a comprehensive public health approach should permit children to be bigger under environmental circumstances in which becoming bigger is truly better.

Understanding growth retardation is not the exclusive domain of the nutrition community. We must unite with other professions in intersectorial and multifaceted research and programmatic efforts that focus on the foods we eat as being but one component of our interaction with the total environment (5). Clearly within such an effort, attention to the intestinal health of tropical populations, specifically averting environmental enteropathy, and thereby sealing the permeability leaks that admit antigens to the systemic milieu will require redress (3). Thus, along with collateral and synergistic improvements in the way that low income societies select and prepare their diet, attention to avoiding infections, reducing inequalities and caring for their environment will reduce the barriers to the growth process. Such a comprehensive public health approach should permit children to be bigger under environmental circumstances in which becoming bigger is truly better.

LITERATURE CITED