Weaning Infants with Malnutrition, Including HIV

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Abstract

A normal pregnancy and adequate lactation performance should produce at 6 months of life a healthy baby, who has a weight and height within the limits of international growth norms. When that does not happen and the child is either too small (or too big), i.e. ‘malnourished’, strong determinants will have been maternal health, combined with environmental stress to the baby. In discussing differential strategies for weaning and complementary feeding, the distinction must first be made between true clinical malnutrition and simply deviant growth. The former needs rehabilitation therapy, which is beyond the scope of this discussion. For deviant poor growth, one must devise a regimen that removes an infant from any low-weight danger zone for increased early mortality risk. Thereafter, one can address the emerging scientific evidence that rapid accelerated catch-up growth has implications for increased metabolic derangement and chronic disease risk in childhood and beyond. Human immunodeficiency virus (HIV), infecting either mother or mother and offspring, is one of the emerging situations that will produce malnutrition before a child is due to be weaned. It will also often induce early weaning. Attention to specific micronutrient supplementation is recommended in HIV-seropositive and malarial infants.

Introduction

Adequate early nutrition is the most essential element in assuring adequate growth and appropriate cognitive and functional development of the infant. Given the evolutionary nature of maternal milk, its ability to support normative growth is axiomatic. Moreover, it is the consensus of public health opinion that exclusive breastfeeding through at least the first 4 [1], if not a full 6 months of life [2], followed by timely and appropriate introduction of foods to complement maternal milk intake, is the ideal dietary pattern during the first year of life.
The nutritional status of a fetus and nursing infant is almost totally dependent upon the mother and maternal factors. If maternal health and nutrition are satisfactory during gestation, and adequate lactation and exclusive breastfeeding are established, a 6-month-old infant should be well-nourished at the moment of initiating the weaning process. Given the degree to which appropriate early feeding should be protective both of normal growth and good health [3], the association of the term ‘malnutrition’ with the initiation of weaning may seem somewhat tautological on its face.

Hence, to get oriented on logical bearings of this topic, we must take a step backwards and consider the implicit query: Under what circumstances would a child emerging from the early months of life, and presumably exclusively breastfed, be classified as ‘malnourished’ at the point of the life course at which complementary feeding is first indicated? To address this, requires an examination of the multiple definitions of ‘malnutrition’ in order to understand the connotations and implications of each, and their application in evaluation. Moreover, if growth or body composition have been truly retarded or distorted despite exclusive breastfeeding, we must identify any noxious process(es) that compromised the usually protective and privileged situation of exclusive breastfeeding.

What Constitutes ‘Malnutrition’ at the Initiation of Weaning?

The primary indicator of adequate nutrition during infancy is adequate growth, that is having anthropometric indices (length and weight) that fall within the boundaries of a standard reference growth curve, and having a trajectory of growth that tracks an appropriate velocity month by month. The National Center for Health Statistics growth curve [4], the traditional international standard for decades, was criticized for the influence of energy-dense formula feeding and lack of ethnic diversity in its constituent population. In April 2006, United Nations agencies adopted a new standard for growth [5]. The reference populations for the first 2 years of life were a combination of select cohorts from 6 geographic locations, maintaining ideal practices of infant and toddler feeding practices, including 4 months of exclusive breastfeeding and at least 1 year of maternal milk offering [6]. According to the framers of the new World Health Organization (WHO) growth curve, it is meant to be ‘prescriptive’, that is to be the universal standard for all children independent of social class, ethnicity or geographical origin [6].

Micronutrient malnutrition adds another dimension to nutritional adequacy at the time of weaning. The breast milk content of certain nutrients, such as iron and zinc, is low and becomes progressively more marginal with the passage of lactation. To the extent that the complementary feeding regimen chosen will correct any deficits in micronutrients, however, any utility of diagnosing iron or zinc deficits would be impractical and academic.
Poor Growth in Early Infancy

Beyond the appropriate macronutrient forms and micronutrient content of human milk, infants benefit from the passive immunity transferred in utero from the mother’s circulation via the placenta and from the hygienic nature of maternal milk, combined with its immunoprotective properties. Hence, substantial infections should be a rarity during the first months of life. It is important to distinguish three scenarios of ‘malnutrition’ (table 1).

Severe Protein-Energy Malnutrition in Infancy

The aforementioned biological factors generally protect a breastfed infant through the first semester of life, but unique situations of early infectious stress or true maternal lactation failure can produce the clinical protein-energy malnutrition syndromes of kwashiorkor (edematous) and marasmus (inanition). In such situations, one common principle is to continue breastfeeding through the child’s recuperation. So, to some extent, the therapeutic recovery feeding regimen is a variety of complementary feeding.

However, with this severity of malnutrition and clinical illness the regimens are unique and specialized. A consultancy of the WHO and the Nestlé Foundation renewed a focus on the nuances of rehabilitation of severe protein-energy malnutrition [7]. This regimen has improved child survival [8]. A consideration of the clinical management of severe malnutrition is beyond the scope of the present discussion. Suffice it to say, however, that the principles discussed in the rest of this chapter and in other contributions to this volume come into practice when the severely malnourished child emerges from intensive therapy, and is returned to an age-appropriate diet.

Pathological Factors Contributing to Poor Growth before 6 Months of Age

When severe growth faltering is observed in supposedly exclusively breastfed 6-month-old children of privileged circumstances, the leading possibilities to be considered are: (1) not indeed having received exclusive lactation, (2) presence of congenital or hereditary anomalies, or (3) infant abuse. When it occurs in underprivileged conditions, the leading possibilities to be considered are: (1) having suffered substantial fetal malnutrition (small birth size), (2) not indeed having received exclusive lactation, and (3) presence of an infection.
What infections are relevant in the first 6 months of life? It should be remembered that breastfed infants are relatively resistant to some of the more notorious tropical diseases. For instance, intestinal helminths are not usually contracted until a child begins to crawl, and it takes months for the eggs to mature into adult worms. Recurrent diarrheal disease emerges when the weaning period begins, but is not likely to have occurred during exclusive breastfeeding. Infancy is a period of unique susceptibility to two epidemic diseases, however; we shall use them as examples in this analysis. Malaria can begin early in life, insofar as the children have not yet acquired immunity; it adversely affects growth early in life [9]. Human immunodeficiency virus (HIV) can be transmitted in utero, during birth or from the breast milk of an infected mother, but convenient early diagnosis is confounded by the presence of maternal antibodies.

**Ecological and Environmental Factors Contributing to Poor Growth before 6 Months of Age**

It was shown in elegant studies with Peruvian infants from Lima’s squatter slums that, despite exclusive breastfeeding, growth may not follow the same trajectory as in their peers in the United States [10]. Based on poor growth in poultry and livestock raised in unsanitary conditions, we postulated that chronic immunostimulation might explain some of the variance of poorer growth, even of appropriately fed infants [11]. This scenario was based on a presumed mechanism of ‘damage’, i.e. metabolic disruption by the burden of ambient microbes.

A complementary (and not mutually exclusive) alternative scenario for slow early growth is based on a proposition of programmed ‘adaptation’ [12]. This theory postulates that the fetus senses (predicts) – and fetal growth responds to – the conditions of the external environment in which it will have to survive. The prospect of deprivation predisposes to adapting with a more compact body habitus and a more efficient metabolism.

Unhygienic and deprived ecological circumstances of rural agrarian or urban slum communities invoke modulators of – or signaling of messages for – limited early-life growth. In the face of enduring squalid deprivation, it would seem preferable, moreover, to influence growth by removing the adverse conditions, rather than by pushing trophic stimulation strategies to force attainment of the standard growth pattern.

**Guidelines for Complementary Feeding in ‘Infant Malnutrition’**

The foregoing discussion raises some doubts on the assurance with which, short of overt clinical signs and symptoms, we can label a certain pattern of growth as constituting ‘malnutrition’; meanwhile, we recognize that a certain
‘hidden hunger’ for sufficient intake of micronutrients may coexist with – or without – states of slower growth. Ongoing research provides us with emerging consideration on the causes and consequences of deviant growth patterns in infancy, but the practical aspects of improving complementary feeding are still grounded in what household economics, cultural acceptability and established science and experience will allow for.

**Theoretical Considerations for Anthropometric ‘Deviation’**

When is being small in size a consequence of injury or damage, and when is it an attempt at adaptation to adverse ambient circumstances? The need to ponder such a question has arisen with the emergence of the school of biological thinking known as developmental origins of health and disease. The theory of predictive adaptive responses is predicated on a life-long, phenotypic programming of metabolic response in relation to accessing and storing nutrients [12]. If the prediction is for nutrient scarcity, the phenotype for nutrient management will be one of compulsive retention. If the environmental signal projects abundance, then a looser control of nutrient disposal will dominate the metabolic phenotype.

It now seems, however, that it is not only smallness of size, but also undergoing a period of intense and rapid compensatory growth, that forms and solidifies the life-long metabolic disposition toward excessive nutrient retention and chronic disease [13]. Multiple epidemiological observations, recently summarized [14], confirm an association between early small size, rapid ‘catch-up’ growth or both and risks of obesity, metabolic disorders and malignancies from childhood onward. This predisposition to disease is now attributed to the fetal and infant adaptations for scarcity becoming dysadaptive at a point in later life when, unanticipated, the dietary offering of sodium, sugar, fat, and energy becomes abundant.

These considerations raise the philosophical quandary as to whether the feeding regimen for a child who was born small or faltered in growth trajectory during the first semester of life should aim: (1) to stimulate growth despite the ecological conditions (trophic intent), or (2) to release the infant from environmental barriers to growth providing an adequate offering of nutrients (enabling intent). The former is prescriptive for the host; the latter is adaptive by the host. A series of correlates for the latter assumption of adaptation are provided in table 2.

Future food technology research could seek to develop simple techniques to provide a filler substance to an infant’s diet that would dilute its caloric density without jeopardizing micronutrient status nor severely impairing growth and development. Additional cost, cultural acceptability, infant’s discomfort from hunger, and disruption of family dining patterns are among the considerations to be addressed in such dietary modulation of infant growth.

Balanced against the concern for long-term chronic disease risk are a series of clear and present dangers from low body size and slow growth for
infants in developing countries. The clinical consequences of low birth weight and growth faltering for mortality risk have been well documented for decades. Pelletier et al. [15] documented that underweight (low weight for age) is a contributing factor in over half the deaths in children under 5 years of age. Whether the low weight is a marker of a child at risk or a true mediator of his demise, this association has inculcated a clinical and public health imperative to grow a small child out of the weight zone of high mortality risk.

We confront the paradox that (1) being below a certain weight in infancy conveys a certain excess risk of infectious mortality [15], whereas (2) rapid accelerated growth in infancy conveys a predisposition to metabolic derangement in childhood [14] and chronic morbidity in adult life [13]. To the extent that small body size truly exposes infants to risk of adverse outcomes for infections in the unsanitary and derived environments of developing countries, it would be at some peril to infant health if we would too lightly discard conventional wisdom and practice of rapidly recovering normative weights.

Practical and Prescriptive Considerations for Anthropometric Deviation

Whatever the theoretical considerations for a child that is small, there cannot – and should not – be substantial deviation from the WHO’s practical principles of adapting a child to the household dietary scheme [16]. The constraints on nutrient density and bioavailability in the common grains and tubers, milled and mashed to produce traditional weaning foods in developing countries, have been meticulously documented in a classic analysis by Brown and coworkers [17, 18] for United Nations agencies’ publications, and are discussed elsewhere in this workshop.

As to the limits of absorbing the zinc and iron in contemporary complementary diets, premasticated meat was presumably the basis for the evolutionary complementary food of hunting clans. Solving the low nutrient density and poor bioavailability issues for diverse micronutrients should

Table 2. Admonitions for the protection of the infant born under adverse circumstances in a low-income developing society

- Do not be so small as to be at higher risk of death from infectious diseases
- Achieve and maintain a ‘protective’ relative weight compatible with survival
- Beyond that, however, do not be in a hurry – nor in an obligation – to achieve the median weight for your age (or higher), especially if your length has not extended in a normative fashion
- Rather maintain a normative partition between lean and fat tissues in relation to your attained length
- Seek a dietary pattern consistent with your in utero adaptation, restricted if you were of low birth weight or there was a rapid early growth spurt, and more liberal if you were of normal birth weight and early growth
cause us to heed recent calls for early introduction of animal protein [19], and even consider cooked meat and visceral organs for initiating weaning [20].

Good caring practices in the household are indispensable for successfully feeding an infant of whatever growth attainment. Social scientists at the Cornell University developed a ‘best-practice complementary feeding behaviors’ framework which identified the following elements: ‘what is fed, how food is prepared and given, who feeds the child, when food is fed (frequency and scheduling), and the feeding environment (where)’ [21]. Clearly many household members, including slightly older siblings, participate in infant feeding. Factors such as the microbiological purity of the water used to prepare porridges and gruels need to be assured.

Theoretical Considerations for Infection-Related Growth Retardation

We have isolated two contemporary infectious scourges – malaria and HIV – that can impair infant growth virtually from the time of conception through early infancy. In fact, even if an infant is not infected with the HIV virus, exposure to maternal antiretroviral medications in utero and in breast milk can adversely affect growth. These infections represent situations where the nuances of feeding and care at the initiation of weaning have more profound, and weighty considerations for the health of the infant and the well-being of the family is in the balance.

In these two diseases of intracellular pathogen origin, theoretical concerns for adverse effects of iron and zinc exposure have arisen. A recent supplement field trial in a malarial area of Africa confirmed excess mortality with oral iron supplementation [22], with no adversity from oral zinc. Trials elsewhere on the continent in HIV-infected infants and toddlers uncovered no untoward effects with either zinc [23] or iron [24], but theoretical concerns remain.

Practical and Prescriptive Considerations for Infection-Related Growth Retardation

The emotional and financial burdens of malaria and HIV call for pragmatism and practicality in the management of complementary feeding. Moreover, as shown in a recent study in Côte d’Ivoire, when weaning was initiated much earlier by HIV-seropositive mothers, there were negative consequences for growth [25]. Nevertheless, the basic principles of complementary feeding are as operative in these infections as with all infants [16–18].

When financial and logistical conditions permit, however, some additional strategies in complementary feeding may improve outcomes. For instance, since avoiding ambient infections, especially in the immune deficiency states of infants living with HIV is a priority, even more attention to water purity and food hygiene is desirable. Probiotics and hyperimmune bovine colostrum, moreover, may have a role in prophylaxis and treatment of diarrhea [26]. For instance, the protozoa, Cryptosporidium parvum, produces a profuse,
intractable diarrheal state resistant to antimicrobials, but susceptible to the antibodies in bovine colostrum concentrates. Periodic vitamin A supplementation has proven protective of growth and health in both HIV- and malaria-infected infants and toddlers and is recommended [27, 28]. Zinc supplementation may also be protective [23]. In infected infants, there is untapped promise in a series of supplement formats, such as sprinkles (a powder), spreads (analogous to nut butters), and foodLETS (crushable flavored tablets), recently devised to provide selected micronutrients directly to complementary dishes or diets prepared at home [29].

### Elements for a Research Agenda

The fundamental public health goal surrounding considerations for infants with poor growth is assuring their short-term survival and health through their second semester of infancy and into the toddler period. Thereafter, appropriate growth and full psychomotor and physiological development are to be encouraged. Balancing practical issues of early mortality risk with predicted adverse long-term health outcomes in the management of low weight, we lack complete knowledge and wisdom for assessing our patients and setting optimal goals for dietary intake and growth. We also lack tools and recipes for formulating diets to achieve less rapid weight recovery without endangering health, nutrition or ultimate height attainment. Based on the sum of present considerations, elements of a research agenda to address the pressing issues have been outlined in table 3.
Conclusion

It is not expected that an exclusively breastfed infant would be lagging in growth attainment or velocity after the 6th month of life when complementary feeding should begin. Not all growth failure and nonnormative body composition, moreover, represent ‘malnutrition’ in a universal connotation. It may be due variously to phenotypic adaptation or nutrient imbalance, each of which has differential relevance to short-term survival and long-term health. Our first imperative is to understand the theoretical principles that would influence assessment of nutritional implications of observed growth. We then need to dominate the practicalities of current best practices for routine feeding and application of micronutrient supplements. A review of the topic reveals a number of uncertainties and unresolved issues that lend themselves to continuing investigation. The end would be refining assessment of ‘small’ infants to assign the most appropriate goals for their growth velocity and micronutrient nutrition while developing affordable and acceptable complementary feeding guidelines and practices to achieve the safest and most harmonious outcomes.

References


Discussion

Dr. Michaelsen: You mentioned that you have some problems with prescriptive growth, but I didn't really understand. Are you suggesting an alternative?

Dr. Solomons: I have a problem with the notion of prescriptive in many ways. I never mentioned them but I am glad you asked the question. What they basically say is that you should be within a certain channel on a reference growth chart. Let us go back to how the children were selected and treated to become part of the data-set. There was a master prescription. The prescription is to be well fed, to be perfectly fed by contemporary standards, including a period of being exclusively breastfed. It is unfair to assume to be on a trajectory created by exposure to an optimum environment
and feeding regime, if he or she has a different start in life. We need to focus on the conditions that permit the growth pattern on the WHO standards, not on the channels within the standards themselves. That is my problem with prescriptive growth. In my opinion, prescriptive has to start with the conditions and not the trajectory.

Dr. Cabus: I have read some papers about people with AIDS who interrupt breastfeeding, and when breastfeeding is combined with food, it is easier to get the virus. Could you comment on this?

Dr. Solomons: There is science and very systematic observations. Up to 4 months for any suckling, there is a certain risk of the mother passing on the virus to the child. If the mother is not willing to undertake that risk she should never let the milk flow from her breast into the child. The evidence suggests, however, that up to 4 months of age, if she doesn't get things like mastitis or cracking of the nipples that will enhance the infection, the child will have a minimum risk of contracting the virus as long as she doesn't use any other food besides breast milk. We mean truly exclusive breastfeeding. Now at 4 months other foods are going to be started anyway, so at that point rather than continuing to give breast milk, which is the recommendation for any other situation, breastfeeding should abruptly stop because the combination of milk and solid foods produces more risk of viral transmission. This could be related to changes in permeability of the intestinal wall. Now the other thing to discuss is how safe artificial formula feeding from day zero is, and to me this is a question of the purity of the water. The products are fine, but can water sanitation be managed? It is possible, then that is a viable alternative. The question if there is more risk of death when artificial formulas are used is valid if the water is infected, but if the water is pure there is no risk, as in a developed country. It is not an easy scenario but if it can be managed then that is another successful way to prevent the transmission. Of course the child could already be infected and that is something that is easily discovered with all the sophistication of virus counts done by antigen detection. The child has the maternal antibody, or children who have positive mothers will have the antibody until that clears in the child's own antibody. So resources and the direction of resources is the major issue of consideration in an AIDS-infected environment. This is my understanding of why abrupt weaning at 4 months or never breastfeeding are the only two recommended alternatives, and mixed feeding is definitely not recommended.

Dr. Haschke: The WHO has a clear policy on this. We should not assume that the majority of mothers, in particular in southern Africa, are living under conditions where they don't have access to clean water. In South Africa, for example, it is the program of the government that more than 90% of the population should have access to clean water, including all the people living in settlements and even in big cities where the HIV rate is very high. So here the government has clearly decided to offer food programs for these children. The same is true for Botswana and other countries where HIV rates are high. For informal settlements, I think that there definitely should be an alternative to food programs, for example exclusive breastfeeding, which is the other way to enhance child survival.

Dr. Pazvakavambwa: I just wanted to clarify the issue of formula feeding in HIV. There was a CDC study in Botswana in which the government provided formula and antiretrovirus drugs to HIV-infected children. But then there was a study in which the mortality in formula-fed babies, where the water was said to be freely available and of good quality, was compared with the mortality in breastfed babies, and it was found that the mortality in breastfed babies was much lower than in exclusively formula-fed babies [1]. So I think that even in the so-called richer developing countries one has to be very careful before prescribing exclusive formula feeding in the presence of HIV.

Dr. Solomons: As a confounder there is of course a self-selective nature. On the one hand, there are mothers who choose to breastfeed and those who choose to
formula feed; so you can never truly eliminate the maternal confidence factor from an observation.

**Dr. Bulusu:** Going back to this WHO prescriptive growth that you were mentioning. In the child development program in India, these growth charts are being used, and today each and every village community level worker is aware of grades 1, 2, 3, and 4 malnutrition because the charts are more like a guideline for them. Growth monitoring is done at the village level, so even an illiterate worker or mother can see just on the basis of the charts that her child has fallen, so improvement is needed. In the positive deviance program of UNICEF, which is being tried in India, just by showing the care and feeding practices of one mother sets an example for the other mothers in the village. When one mother with the same socioeconomic status is able to improve her child's condition why can't the other mothers do so? Perhaps I am wrong, but I look at this growth chart not as a directive that your child has to be there but probably as a guideline to see where your child stands and how best can we improve the status of the child.

**Dr. Solomons:** It would be different, if 'standards' were taken as a guideline, or 'reference' means knowing where the child is in relationship to where he might be going. We have to be very careful when using those two concepts. Prescriptive to me means standard [2]; 'reference' is compared to anything that can be considered to be a comparative reference. The choice of words is important. What I assert is that it is dangerous and a motive for action when a child is losing channels. But maintaining one's channels or gaining channels are different. So either of those two is all right. What I am concerned about is gaining channels too rapidly; that is to start below the red line and be about the green line in 4 months. Now the next question is at age 7, 8, 15 years and so forth, as to what has that rapid growth brought. Especially in India where, as I mentioned yesterday in my response to your talk, there is the Indian paradox which means that body composition is quite different. As a regional phenomenon mothers have a median BMI of 18.5, that is what we consider the lower limit of normal. So half your population is by definition chronically energy malnourished. I don't believe they are, I don't believe that term fits. Similarly you have the so-called thin-fat babies [3]. Babies are small but they are fatty, and the fat is both visceral and subcutaneous. Both of which seem to be primed for metabolic insulin-resistant. So what happens when a thin-fat baby is born to a mother who is eating high glycemic index food? Then the situation is worse for that baby than for the one in Paris whose evolution and adaptation has been conditioned for over the last 500 years. The French baby is better off in opulence than the rural migrant in India. Now it may only require one generation for Indian babies to receive an external message of affluence, so they will no longer be programmed for retention but programmed for easy excretion.

**Dr. Shahkhalili:** What would happen if small babies did not have catch-up growth during the suckling and weaning period? Could it happen later? Would these babies with delay/lack of early catch-up growth have a risk for later metabolic disease?

**Dr. Solomons:** I can't give you a general answer, and I don't want to because I don't think the science guides us well. But clinically, if you take two children with exactly the same situation, one of them may do well with growth and the other may not. So I don't know the predictors of the diagnostic subtleties to determine which type is which. But I hope we can learn that and then it would be a management issue, not on a public health level but on an individualized level by a practitioner or care taker. What I am basically saying – which the public health people don't want to hear from me – is that to respond to low size at birth, we need to depend upon more of the art of clinical judgment rather than the standard of prescriptive management to determine how the child should grow. The battle is not to be so small as to be at risk of death and dying early, because it is better to be alive and diabetic later rather than to be dead early. But
using that as a principle on how to manage growth, I think the study by Demmelmair et al. [4] is very important because it should give us a handle on both the ethical considerations and the dietary management that can make people grow more slowly. There are certain ways that if you give children complementary feeding they will cross channels and grow right back up to the green line, at least in weight, in a very short period of time. I think at least the science suggests that this pattern has more adverse consequences. Slower growth, however, is harder to produce. We don’t have a lot of experience with controlled slow growth. I think randomized trials, such as the one happening in Europe [4], should perhaps happen in developing countries like India, which produces two patterns of growth that should be looked at in terms of the metabolic consequences in later life. As I emphasized it is better to survive to be able to think about going on to later life. So the global imperative is for the child to survive, and then worry about how they are going to be metabolically better off, because we now have the science and the concepts to think of a better quality of life after surviving.

Dr. Ibe: Why does the WHO describe this growth chart as prescriptive?

Dr. Solomons: I don’t know; go to the website or look at Dr. Garcia’s talk [2]. The WHO calls it prescriptive, I don’t.

Dr. Ibe: We use the growth chart as a medical tool and it is called the route to a healthy child for the mothers because it is visual. They look at it and see how their babies are doing; they show esthetic growth.

Dr. Solomons: Living in our country (Guatemala), the goal would be to show steady growth: don’t lose growth, don’t go down. That would be my first mandate, and my second would be don’t grow upwardly on the curve too fast if there is the option to grow more slowly. But going down, weighing less than before, that is a bad sign. It is a sign of immediateness and then all the therapeutic efforts to save the children from death have to be quickened. But if they are growing on a good slope on the chart, I don’t care whether they are between the red lines or below the red lines of the center of the reference distribution. I do care if they are above the red lines (i.e. overweight). That was in my paper but I didn’t talk about macrosomia and large babies, another problem, not so much a problem in Nigeria, India and Guatemala, but maybe in Paris where an occasional baby can weigh more than 5 kg at birth.

Dr. Michaelsen: You mentioned the CHOP study [4] and several others have also mentioned it. It is a randomization study, randomization to formulas with different protein contents starting from early after delivery, so it will not really give us an answer on an effect during the weaning period which is a period where we don’t know very much about programming.

Dr. Solomons: At 6 months, they get off the low protein or the higher protein diet. But it is still about growth and not about feeding. So the notion then would be to learn. I hoped that that was embodied in my research agenda. There is also follow-up formula but they still have to eat solid foods with it. How do you then maintain the moderate and not accelerated growth once you start feeding other than infant formula? That is the question.

Dr. Agostoni: May I give you some details on this since we are part of the protein trial with infant formulas that has just been mentioned (the CHOP project). The low protein group and the high protein group go on up to 12 months because they switch to two follow-on formulas with low and high protein content, respectively, so they stop the low and the high protein schedule at 12 months. As far as their dietary intakes from solids and other sources, they are going to be integrated too.

Dr. Brunser: As I want to close the session, I want to say two things to Dr. Solomons. First of all, your lecture was, as always, very thought-provoking. The second thing is that you will have to change the picture of your mosquito: you wanted me to say that the mosquito in your slide was the malaria mosquito, Anopheles, but as it turns out, it is the yellow fever mosquito, Stegomyia fasciata.
References

1 Kuhn L, Peterson I: Options for prevention of HIV transmission from mother to child, with a focus on developing countries. Paediatr Drugs 2002;4:191–203.