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ORIGINAL COMMUNICATION

Childhood stunting in Northeast Brazil: the role of *Schistosoma mansoni* infection and inadequate dietary intake

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Objective: To evaluate the relationship between stunting, *Schistosoma mansoni* infection and dietary intake in schoolchildren.
Design: This is a cross-sectional study. Two stool samples were obtained from each child and examined quantitatively for the presence of *S. mansoni, Ascaris lumbricoides* and *Trichuris trichiuria* eggs. Information on dietary intake, and demographic, biologic and socioeconomic variables was elicited during the in-home survey. Logistic regression was used to evaluate the association between stunting (height for age < -2s.d.), parasitic infection and food consumption.
Setting: The study was carried out in the city of Nazaré, located in the Recôncavo region of the State of Bahia, northeastern Brazil.
Subjects: The sample consisted of 461 children 7–14 y old, 228 boys and 233 girls, recruited from public schools.
Results: Of the children studied, 55.1% presented with *S. mansoni* infection and 22.1% were stunted. The median protein, lipid and carbohydrates intake were 47.8, 36.0 and 248.2 g/day, respectively. The median caloric consumption was 1527.0 kcal (6388.97 kJ/day). The analysis indicated that children heavily infected ($\geq$400 eggs/g of stool) with *S. mansoni* had a 2.74-fold (95% CI: 1.32–5.67) higher risk of stunting than uninfected children, and those with inadequate intake of lipid ($<$36 g/day) had a 1.83-fold (95% CI: 1.05–3.20) increased risk of stunting compared to those with adequate diets.
Conclusion: Heavy *S. mansoni* infection and inadequate dietary intake of fat in schoolchildren play a significant and independent role in the development of stunting. This meaning that nutritional interventions in this age group in *S. mansoni* endemic areas must include the diagnosis and treatment of the infection associated with dietary measures.
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Introduction

Linear growth deficit has been most frequently studied in children less than 5 years of age in underdeveloped countries. The deficit begins in the first 4 – 6 months of postpartum life (Waterlow, 1994), and is most accentuated in the first 3 y of life. Between 3 and 5 y of age, the rate of decline decreases; however, in many cases, compromised stature persists throughout the individual's life (ACC/SCN, 2000).

The prevalence and determinants of linear growth deficit in school-age children and adolescents have been studied less frequently, although it is an important health issue for this segment of the population. It is estimated that the percentage of school-aged children with linear growth deficit in the different Latin America countries varies between 7.5

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and 50.6% (ACC/SCN, 2000, p 14). In Brazil, there are no available nationwide, population-based studies that characterize the anthropometric state of schoolchildren and adolescents, and a few studies have investigated the patterns of linear growth in children between 5 y and the onset of adolescence (Sichieri & Allam, 1996; Andrade *et al*, 2003). The results of a study carried out in a small city in Bahia, however, indicate that the deficit in linear growth reaches approximately 11% of school-aged children and 11.7% of the adolescents (Assis *et al*, 2002a). In Salvador, one of the most influential cities of the Northeast, malnutrition assessed by body mass index (BMI) was identified in 11% of adolescents (Assis *et al*, 2002b).

Although the underlying determinants of stunting for children in this age group are ill defined, they are likely to be similar to the determinants for children less than 5 y of age (de Onis *et al*, 2000). It is also likely that these same determinants explain the maintenance of linear growth deficits in older children. Factors such as low wages, inadequate food consumption, increased burden of diseases, inadequate sanitation and hygienic conditions are also associated with the health and nutrition status of children and adolescents. Clearly, inadequate intake of nutrients, particularly of total calories, protein, fat and micronutrients, is associated with physical growth deficit in preschoolchildren (ACC/SCN, 2000). However, adequate dietary consumption does not guarantee normal physical growth, since other morbid events (eg acute or chronic infections) negatively affect this complex process contributing to the onset or maintenance of growth deficits in children.

Parasitic infections, particularly ascariasis, schistosomiasis and hookworm, are extremely prevalent in underdeveloped countries (WHO, 1999a), and are among the most common sources of morbidity in children and adolescents. Because parasitic infections cause anorexia and poor absorption of nutrients and promote the deviation of nutrients to the organism's defense mechanisms, they contribute to the onset or exacerbation of weight and height deficits, as well as to specific nutritional deficiencies (ACC/SCN, 1997). In the acute phase, the infection induces an immune response and the production of cytokines (Tomkins & Watson, 1989; Ross *et al*, 2002), which can directly affect the process of bone formation and remodeling required for the growth of long bones (Stephensen, 1999).

The different species of *Schistosma* have been shown to be associated with linear growth retardation in children (Stephenson *et al*, 1989; McGarvey *et al*, 1993; Parraga *et al*, 1996). In the northeastern region of Brazil, *Schistosoma mansoni* is the only species of schistosome; however, it is one of the most prevalent parasitic infections in the region. This parasitic infection is known to exert a negative effect on the anthropometric status of school-age children even in low–moderate level of infection (Parraga *et al*, 1996; Assis *et al*, 1998). Allen (1994) postulated that the association between the effects of parasitic infection on physical growth could be modified by changes in dietary intake.

The Brazilian Northeast, the site of this study, although the birthplace of the nation, is one of the poorest regions of the country. A large percentage of its population lives under marginal living conditions in material terms. There is inadequate basic sanitation, resulting in an elevated prevalence of the diseases of want, infection and parasites (Monteiro, 1995; Duarte *et al*, 2002). Among the parasitic diseases, schistosomiasis is one of the most frequent in the region, affecting several million individuals, while as a consequence of mass chemotherapy its severe form has decreased (Katz, 1998). Studies carried out in various regions of the State of Bahia (Bina & Prata, 2003) and in other regions of the Northeast (Coutinho *et al*, 1997) have identified a high prevalence of schistosomiasis. This study aimed to further analyze the relationship between stunting, *S. mansoni* infection and dietary intake as a cofactor in the development of morbidity.

Population and methods

Population

The study population was derived from a sample of 1047 students from 17 of the 21 public schools of the town of Nazaré, State of Bahia, Brazil, selected to participate in a large study on growth and *S. mansoni* infection. The characteristics of the study population, the methods and other results have been published elsewhere (Parraga *et al*, 1996; Assis *et al*, 1998). At baseline, 50% of the study population was selected at random using the students' serial numbers for a demographic and nutritional survey, stool examinations and anthropometric measurements. Complete data were obtained for 461 students. The survey included data on the dietary intake of the child and socioeconomic and sanitary conditions of his/her household. The power of the sample was 80% at a statistical level of significance of 0.05 to detect a difference in risk for stunting depending on infection status considering the prevalence of stunting to be 22.1%. This calculation assumes the inclusion of 12 variables in the logistic regression model.

Ethical committees at the Federal University of Bahia and Case Western Reserve University approved the study protocol. The guardians and the children were fully informed about all the aspects of the study, and signed informed consent was obtained from the guardians of all participants.

Height measurement

Height was measured according to the recommendations of the World Health Organization (WHO, 1983), by appropriately trained nutritionists who were unaware of the child's infection status. Each child was measured twice, standing on a flat surface using a Ross Stadiometer (Ross Laboratories, Columbus, OH, USA) with an error of ± 0.1 cm. The mean of the two measurements was calculated and entered as the child's height. In keeping with WHO recommendations (WHO, 1983), the measurement method was standardized

prior to the beginning of the study. Each technician performed two height measurements on 10 different children who were not part of the study population. The two measurements were taken blindly (ie separately by two different technicians), observing a sufficient time interval to prevent the first measurement from influencing the second. The two measurements were compared to evaluate reproducibility. To evaluate accuracy, the measurements made by one of the authors were compared with the measurements made by the nutrition technicians. The measurements of the technicians were found to be highly correlated, both before ($r=0.94$–1.00) and after data collection ($r=0.80$–1.00) (Parraga *et al*, 1996). The age of the child was obtained from a birth certificate. Examinations were conducted in the same schools where the participants studied, on pre-established dates and times.

Nutritional survey

The information on the dietary intake was elicited from a 24-h recall method. The children and their mothers provided all information. The interviews were conducted in the subject's homes by registered nutritionists. They were fully trained to collect the data in a standardized way. In order to assist the subjects in quantifying the food portion consumed, an album with full-scale pictures showing foods and utensils was used (Araújo *et al*, 1993). Measurement cups were used to estimate the quantity of ingested liquids. The quantitative nutrient value of all foods consumed was estimated using a computer program (Ancao *et al*, 1996).

Parasitological examinations

Stool samples were collected in plastic cups that had been previously labeled, numbered and distributed by technicians of the National Health Foundation (Fundação Nacional de Saúde — FNS), who also provided instructions for the collection and analysis of fecal examinations. The Kato–Katz method was used to quantify infection by *S. mansoni* (eggs/g of stool) and to identify the presence of *Ascaris lumbricoides* and *Trichuris trichiura* (Katz & Pellegrino, 1972).

All children whose feces were found to contain *S. mansoni* eggs were asked to provide a second sample in order to determine the intensity of infection. The mean of the two egg counts (multiplied by 24) was used to determine the intensity of infection (number of eggs/g of stool). Data quality control was performed by two laboratory technicians not involved in the study, who re-read 10% of the slides.

Socioeconomic survey

Household crowding, years of maternal education and household appliances were used as proxy variables to assess the socioeconomic status of each family. The children's guardian provided the requested information during the in-home survey.

Data entry

Data were entered in the program EPI-INFO. Statistical analyses were performed with the SAS statistical package (SAS System for Windows, version 8, 2000).

Statistical analysis

Proportions were used as the statistical parameter in the descriptive analysis. Logistic regression was used to measure the association between physical growth with *S. mansoni* infection and the energy and nutrient intake. Sex, age and the majority of the socioeconomic variables were associated with both nutritional status and parasitic infection, meaning that they are potential confounders of the principal association studied. Therefore, in the logistic regression, we adjusted for these variables. The logistic model was also adjusted for concurrent infections with *A. lumbricoides* and *T. trichiuria* to control for possible effects of these parasites on linear growth. The risks were expressed as odds ratios with two-sided 95% confidence intervals.

Prior to the final analysis, the unadjusted bivariate logistic regression analysis was used to evaluate the individual effect of all the independent and covariables on linear growth. The contribution of each variable in the model was assessed by the maximum likelihood estimation. All variables that were found to be associated with growth deficit in this exploratory analysis at the level of $P<0.25$ were included in the multivariate analysis (Hosmer & Lemeshow, 1989).

Anthropometric status was measured using the 'height-for-age' *z*-scores. Children with a *z*-score value higher than or equal to -2 standard deviations (s.d.) above the expected value were characterized as eutrophic, while those with deficits lower than -2s.d. were characterized as stunted. This variable was dichotomized (all subjects were classified as either stunted or eutrophic) and considered in the model as the dependent variable, while the intensity of *S. mansoni* infection and energy and nutrient intake constituted independent variables. The intensity of *S. mansoni* infection was categorized as light, moderate and severe (1–99, 100–399 and ≥ 400 eggs/g of stool, respectively) (WHO, 1998). Age was dichotomized into groups: 7–9 and 10–14 y old. These divisions were based on the WHO classification for the end of infancy and the beginning of adolescence (10 y) (WHO, 1995), and also because a second growth spurt occurs in adolescence (Allen & Gillespie, 2001). The median of total calories, protein, carbohydrates and lipids in the diet was estimated for each child. Values below the median were considered as inadequate intake. The level of years of maternal education was categorized into low (under 5 y of study) and high (above 5 y of study) degrees. Variables that indicated possession of household goods (stove, refrigerator, radio) were categorized in 0 (no) and 1 (yes), and crowding was categorized as low if <5 inhabitants/household and high if ≥ 5 inhabitants/household. When indicated, the variables were transformed into their respective dummy variables.

Results

The study population was composed of 49.5% males and 50.5% females; 28.4% were between 7 and 9 y old, and 71.6% were between 10 and 14 y. Infection with *S. mansoni* was identified in 55.1% of the study population. The majority of these infections were classified as light (49.2%), followed by moderate (30.7%) and severe (20.1%). *A. lumbricoides* and *T. trichiuria* infections were observed in 71.8 and 67.9% of the children, respectively. Linear growth deficit (defined as height-for-age *z*-score <-2s.d.) was identified in 22.1% of the subjects. Years of maternal education were classified as low in 39.3% of cases. A stove was present in 85.3% of the households, radio in 79.2% and refrigerator in 57.7%. The majority of children lived in overcrowded households (64.4%) (Table 1). The intensity of infection did not differ by age or sex (Table 2). The median dietary intake of calories was 1527 kcal/day (6388.97 kJ/day) and the median protein, lipid and carbohydrate intakes were, 47.8, 36.0 and 248.2 g/day, respectively (Table 3). No significant interaction was found between any of the variables.

The bivariate analysis indicated that children with moderate and severe *S. mansoni* infections were, respectively, 1.92 (95% CI: 1.05–3.50) and 3.0 (95% CI: 1.52–5.77) times more likely to have linear growth deficit than those who were not infected. The children in the group 10–14 y old were more likely to be stunted than those younger — 7–9 y of age (OR 1.98; 95% CI: 1.15–3.42). Also, household crowding (OR 2.40; 95% CI: 1.36–4.22) or absence of a refrigerator (OR 1.67; 95% CI: 1.04–2.70) was associated with a high prevalence of stunting. Further, a deficit of height-for-age was found to be significantly associated with dietary intake below the median: proteins (OR 1.59; 95% CI: 1.02–2.48) and lipids (OR 1.98; 95% CI: 1.26–3.11) (Table 4).

The subsequent analysis was to fit a multivariate model including all variables with a *P*-value <0.25 in the bivariate analysis. Although the infection with *A. lumbricoides* ($P=0.95$) and total caloric intake ($P=0.25$) might be excluded under this criteria, they were included in the multivariate analysis, because they have been identified by others as important predictors of stunting (Caulfield *et al*, 1995). It is worth noting that when the two variables were included in the multivariate model, the coefficient of the Wald statistics increased from 38.26 to 41.42 ($P=0.0002$).

Table 1 Biological, demographic and socioeconomic characteristics of the study population, and distribution of intensity of *S. mansoni, A. lumbricoides* or *T. trichiura* infection status

Variables	N	%
Sex		
Male	228	49.5
Female	233	50.5
Age (y)		
7–9	131	28.4
10–14	330	71.6
S. mansoni infection		
Egg-negative	207	44.9
Infected	254	55.1
Intensity of S. mansoni infection (eggs/g of stool)		
Light (1–99)	125	49.2
Moderate (100–399)	78	30.7
Severe ($\geq$400)	51	20.1
Infected with *A. lumbricoides*	331	71.8
Infected with *T. trichiura*	313	67.9
Height-for-age		
≥-2 s.d.	359	77.9
<-2 s.d.	102	22.1
Level of maternal formal education		
High (<5 y of study)	280	60.7
Low (≥5 y of study)	181	39.3
Possession of stove	393	85.3
Possession of radio	365	79.2
Possession of refrigerator	266	57.7
Household crowded*	297	64.4

$N=461$.
*>5 inhabitants per household.

Table 2 Distribution of biological characteristics of the study population by intensity of *S. mansoni* status

	*Sex**				*Age (y)***			
	Male		*Female*		*7–9*		*10–14*	
Intensity of S. mansoni *infection (eggs/g of stool)*	n	%	n	%	n	%	n	%
No. infected	101	48.8	106	51.2	64	30.9	143	69.1
Light (1–99)	64	51.2	61	48.8	35	28.0	90	72.0
Moderate (100–399)	34	43.6	44	56.4	22	28.2	56	71.8
Severe ($\geq$400)	29	56.9	22	43.1	10	19.6	41	80.4
Total	228	49.5	233	50.5	131	28.4	330	71.6

$N=461$.
*$P=0.50$.
**$P=0.46$.

Table 3 Intake median of energy and dietary nutrients

Energy/dietary nutrient	*Median*
Energy	1.527 kcal/day (6388.97 kJ/day)
Protein	47.8 g/day
Lipids	36.0 g/day
Carbohydrates	248.2 g/day

$N = 461$.

This is an indication that these two variables together were important in the fitting of the multivariate model (Hosmer & Lemeshow, 1989). After adjusting for potential confounding variables (age, sex, years of maternal education, household crowding, possession of refrigerator, and *A. lumbricoides* and *T. trichiuria* infection), the final model retained severe *S. mansoni* infection (OR 2.74; 95% CI: 1.32–5.67) and lipid intake below the median (OR 1.83; 95% CI: 1.05–3.20) as the major factors associated with stunting (Table 5).

Discussion

Of the variables studied, *S. mansoni* infection and inadequate consumption of dietary lipids, independent of each other, were the most relevant factors for the high prevalence of stunting (22.1%) in this population. Specifically, *S. mansoni* infection without clinical expression increased the chance of stunting in school-aged children by 2.74-fold (95% CI: 1.32–5.67) compared to those not infected, and lipid intake below the median value nearly doubled the risk of stunting (OR 1.83; 95% CI: 1.05–3.20). These associations persisted even after controlling for demographic and socioeconomic variables, and for the presence of *A. lumbricoides* and *T. trichiura* coinfections.

The various studies involving the relationship between growth and parasitic infection have reached different conclusions depending on the parasite species and the populations studied. Randomized placebo controlled clinical trials tend to provide evidence favoring the recommendation for treatment of these infections as a strategy for improving the health and nutritional status of children (WHO, 1999b). A major part of these studies evaluated the effect of *A. lumbricoides* or *T. trichiuria* infections (Stephenson *et al*, 1993; Northrop-Clews *et al*, 2001; de Silva, 2003), and few assessed *S. mansoni* infection (Assis *et al*, 1998). Also, the great majority of these studies involve preschoolchildren, while studies involving older children are scarce. In contrast with these studies, a meta-analysis of the literature up to 1999 (Dickson *et al*, 2000) indicated that no morbidity could be ascribed to geohelminth infections. This analysis, however, did not address specific species, such as hookworms, or other helminthes, such as schistosomes.

Stunting can be the result of multiple individual and environmental factors in addition to parasitic infection; thus, multivariable logistic regression analysis was used to assess the independent influence of dietary intake. In the unadjusted regression analysis, low consumption of lipids, proteins and carbohydrates each contributed significantly to

Table 4 Crude odds ratio (OR) between stunting and demographic and socioeconomic variables, adequacy of calories and macronutrients intake, and *S. mansoni, A. lumbricoides* or *T. trichiura* infection status

Variables	*OR**	*95% CI*	P-*value**
Intensity of infection (eggs/g of stool)			
No. infected	1	—	
Light (1–99)	0.98	0.55–1.76	0.95
Moderate (100–399)	1.92	1.05–3.5	0.03
Severe ($\geq$400)	3.00	1.52–5.77	$<$0.01
Age (y)			
7–9	1		
10–14	1.98	1.15–3.42	0.01
Sex			
Male	1		
Female	0.76	0.50–1.17	0.21
Maternal level of education			
High	1	—	
Low	1.47	0.90–2.38	0.11
Household crowding			
High	1	—	
Low	2.40	1.36–4.22	$<$0.01
Possession of refrigerator			
Yes	1	—	
No	1.67	1.04–2.70	0.05
Possession of radio			
Yes	1	—	
No	1.08	0.56–2.07	0.63
Possession of stove			
Yes	1	—	
No	1.52	0.67–3.44	0.36
A. lumbricoides			
Not infected	1	—	
Infected	0.58	0.31–1.11	0.95
T. trichiura			
Not infected	1	—	
Infected	1.07	0.56–2.04	0.17
Total dietary calories*			
$\geq$Median	1	—	
$\leq$Median	1.29	0.83–2.01	0.25
Total dietary proteins*			
$\geq$Median	1	—	
$\leq$Median	1.59	1.02–2.48	0.04
Total dietary lipids*			
$\geq$Median	1		
$\leq$Median	1.98	1.26–3.11	$<$0.01
Total dietary carbohydrates *			
$\geq$Median	1		
$\leq$Median	1.36	0.88–2.12	0.17

$N = 461$. *Maximum likelihood estimative.

Table 5 Odds ratio estimate (adjusted) for association between stunting, *S. mansoni* infection and dietary protein of diets*

Variables	*Odds ratio*	*95% CI*	P-*value*
S. mansoni infection (eggs/g of stool)			
No. infected	1	—	—
Light (1–99)	0.96	0.52–1.78	0.89
Moderate (100–399)	1.92	1.00–3.68	0.05
Severe (≥400)	2.74	1.32–5.67	<0.01
Total dietary lipid*			
≥Median	1		
≤Median	1.83	1.05–3.2	0.03

$N = 461$.
*Controlled for age, sex, maternal level of education, household crowding; (>5 inhabitants per domicile), possession of refrigerator, and presence of *A. lumbricoides* and *T. trichiuria*. Maximum likelihood estimate.

stunting. Following adjustment for sex, age and socio-economic status, however, only decreased lipid consumption was retained in the model. Thus, low lipid consumption was independently more critical than poor intake of the other nutrients. In rural populations in Brazil, low consumption of fat has the most significant impact on the availability of energy from the diet (Assis *et al*, 2002a). Although protein consumption was generally adequate and would be correlated with fat in most European or American diets, most protein consumed in this population was of vegetable origin and, thus, low in fat.

In 1910, the North American diet averaged 30% of calories from fat. This rose in the mid-1970s to 40%, but according to the USDAs Continuing Survey of Food Intakes by Individuals this fell in the 1990s to just over 30% (Lin *et al*, 2001). In this Brazilian community, fat provided only 21% of calories for children and adolescents. This level of intake is not necessarily bad. The American Heart Association recommends that less than 30% of calories come from fat in children above the age of 2 y. Even when 15–20% of calories come from fat, studies indicate that fat-associated nutritional factors, such as vitamins A, D and E and long-chain polyunsaturated essential fatty acids, should not be compromised (Fisher *et al*, 1997; Seidell, 1998), however, it points out that the good and bad aspects of fat consumption are not easily compared between modernizing and postmodern populations. For this reason, American Heart Association's recommendations for nutritional requirements of children are population-specific. The minimum for an American population of relatively sedentary children is 20% of calories from fat. An average of 21% is likely to be inadequate for more active rural populations in developing countries where the nutritional content of other foods may be poor. In addition, many of the subjects of this study were infected with multiple parasites as well as undergoing puberty. At this phase of growth, energy demand increases in response to the increased growth rate (Caulfield *et al*, 1995) creating a need for additional energy consumption.

The relationship between parasitic infection and growth is complex and involves a network of multiple determinants from the social, economic and physical environment in which a child lives. The diet itself is often deficient in zinc and vitamin A, a condition other studies suggest is worsened by schistosome infection (Oberhelman *et al*, 1998; Ferreira and Coutinho, 1999; de Silva, 2003).

S. mansoni infection appears to act both directly and indirectly to decrease linear growth by complex mechanisms. There are indications that the infection can affect the absorption and loss of nutrients. The major organ for deposition of eggs and the subsequent inflammatory response is the intestinal wall and mucosa (Ross *et al*, 2002). Although not often recognized, *S. mansoni* infection is associated with a significant increase in watery diarrhea, bloody diarrhea and blood in stools (Sukwa *et al*, 1986). Eggs are retained in gut and produce inflammation of the mucosa, hyperplasia of cells and gut ulceration (Ross *et al*, 2002). This is associated with poor absorption of protein, fat (Coutinho *et al*, 1997) and vitamin A (Friis *et al*, 1996).

The infection may also work indirectly by reducing hormones needed for growth, such as growth hormone (Nilson *et al*, 1993) and insulin-like growth factor-1 (Remer & Manz, 1999), and maturation. There is likewise evidence that chronic inflammation reduces steroids need for maturation and development, such as dehydroepiandrosterone and its sulfate derivate (Fulford *et al*, 1998). Malnutrition also affects the production of steroid hormones as well as the production and differentiation of immunological factors (Nilson *et al*, 1993). Chronic stimulation of the immune system also may result in decreased intake as a result of stimulation of TNF α (Stephenson *et al*, 1993). The higher metabolic cost of immune stimulation, and its repercussions on the hormonal systems, the low intake of nutrients and the anorexia provoked by the infection can be used to explain the association between schistosomiasis and linear growth in prepubertal and pubertal individuals.

The situation can become even more critical when the intake of fat in the diet is low, as in the consumption profile observed in the children studied here. These potential mechanisms could explain the finding that severe intensity *S. mansoni* infection and inadequate consumption of dietary lipids are the most relevant factors for the high prevalence of stunting (22.1%) in this population.

The data presented here result from a cross-sectional study and, as a consequence, in themselves have a limited interpretation. It is not possible to identify and correct for all confounding factors. Also cause and effect between parasitic infection and nutritional status cannot be established. Further, the 24-h recall method has its own methodological and analytic allimitations (Willett, 1990). Despite these reservations, the results of this study are supported by our own studies (Parraga *et al*, 1996; Assis *et al*, 1998) and those of others showing an association between stunting and schistosome infection (McGarvey *et al*, 1993; Stephenson, 1993).

The relationship between infection with *S. mansoni* and physical growth must also be modified by other factors. It is known that children infected with *S. mansoni* generally live in poor areas, in inadequate environmental conditions at risk of several other infections and with inadequate diet (Corbett *et al*, 1992). In this sense, the children are already at increased risk of stunting independent of the *S. mansoni* infection. It is, therefore, possible that the relationship between *S. mansoni* and physical growth is due to a confounding of these several factors. However, the data presented here and elsewhere (Parraga *et al*, 1996; Assis *et al*, 1998) favor the idea of an independent effect of *S. mansoni* infection on the physical growth in this group.

Lima e Costa *et al* (1988) also observed that heavy egg burdens ($\geq$500 eggs/gram) as much as clinical signs of morbidity are associated with weight-for-height deficit in children 4.5–14.4 y of age. This relationship is not always observed (Proietti *et al*, 1992) in areas of low endemicity. However, from the review by Ferreira and Coutinho (1999), it can be concluded that there is a relationship between infection with *S. mansoni* and nutritional compromise in the parasitized population.

Heavy *S. mansoni* infection and low intake of fat were both strong predictors of faltering growth in schoolchildren in this region of Brazil. This study further emphasizes the importance of local environmental conditions and food intake in determining the appropriate amount of fat to be included in the diet. Therefore, chronic parasitic infections should be included, as one important factor in the explanatory framework for growth failure, especially in the linear growth of older children in underdeveloped countries.

In Brazil, after more than 20 y of the implementation of a national schistosomiasis mansoni control program, there is some evidence of a reduction of the severe clinical forms associated with S. *mansoni* infection (Katz, 1998; WHO, 1999b) despite a lesser reduction in the prevalence of infection. However, the observations made here and in other studies on the association between stunting and *S. mansoni* infection and inadequate diet leads us to conclude that programs directed at control of schistosomiasis mansoni must focus on all forms of infection and merely not the most severe, if an impact on nutritional status is included as one of the targets of a comprehensive system of healthcare.

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