Predictors and Nutritional Consequences of Intestinal Parasitic Infections in Rural Ecuadorian Children

by Mamie-Eleanor Sackey,[a] M. Margaret Weigel,[a] *and* Rodrigo X. Armijos[b,c]

[a] *Community and International Nutrition and Health Section, Department of Human Nutrition, Foods, and Exercise, Virginia Polytechnic Institute and State University, Virginia, USA*

[b] *Laboratorio de Inmunologia, Division de Medicina Tropical y Nutricion, Universidad Central del Ecuador, Quito, Ecuador*

[c] *Dept de Microbiologia, Instituto Nacional de Higiene 'Leopoldo Izquieta Perez', Ministerio de Salud Publica, Quito, Ecuador*

Summary

The study investigated the prevalence, risk factors, and nutritional consequences of intestinal parasitic infections (IPI) in rural Ecuadorian children. A total of 244 children aged 0.2–14 years were studied. The data were collected using a structured questionnaire, anthropometry, and laboratory analysis of blood and fecal samples. The results showed that 90 per cent of the subjects were infected with at least one pathogenic IPI: 51 per cent with helminths, 37.6 per cent with protozoa, and 21.4 per cent with both. *Giardia*-infected children had a risk for stunted growth that was twice that of other children (51.7 vs. 33.1 per cent; OR = 2.16, 95 per cent CI = 1.13–4.15; *p* = 0.01). They also had significantly reduced mean hemoglobin levels compared with their non-infected counterparts (11.8 ± 1.5 vs. 12.2 ± 1.4 g/dl; *p* = 0.023). However, the proportion diagnosed with iron-deficiency anemia was slightly, but not significantly, increased (29.4 vs. 24.3 per cent). The most consistent predictor of *Giardia* and other protozoal IPI risk was a high intra-/peri-domicilliary concentration of domestic animals. Children who lived in such households had an infection risk that was two to five times greater than that of their non-infected counterparts. The data indicate that *Giardia intestinalis* infection has an adverse impact on child linear growth and hemoglobin. They also suggest that domestic animals may be an important reservoir for *Giardia* and other intestinal protozoal infections observed in the Ecuadorian children studied.

Introduction

Intestinal parasitic infections (IPI) constitute a global health burden. They are estimated to affect approximately 3.5 billion persons worldwide and cause clinical morbidity in 450 million, many of these being children in developing countries.[1] Intestinal parasitic infections, mostly helminths, have been linked with poorer nutritional outcomes, including an increased risk for nutritional anemias, protein-energy malnutrition (PEM), and growth deficits in children.[1–3] However, the published evidence has not been entirely consistent regarding the impact of these on child growth and iron status.[4] This is especially true of protozoal infections such as *Giardia intestinalis*[5,6] about which, less is known.

Intestinal infections caused by parasites, bacteria, and viruses were ranked as the fifth leading cause of child mortality in Ecuador in 1998.[7] However, despite their assumed importance for public health, only a few studies have been published[8–12] documenting the prevalence of IPIs in Ecuadorian children and only one of these has identified any factors associated with IPI risk.[8] None have directly examined IPI impact on children's nutritional status. Thus, it was decided to investigate the prevalence, predictors and consequences of IPIs on growth and iron status in rural children living in a tropical rainforest area of northwest Ecuador.

Acknowledgements

The study was supported in part by the Dr Howard Massey Food and Nutrition Scholar Award and the Hepler Summer Graduate Research Fellowship from Virginia Tech (MES). Laboratory and logistical support were provided by the Fundacion International Biociencias in Quito, Ecuador. The authors would like to acknowledge the expert technical assistance of Dr Manuel Palacios, Josue Correa and Jorge Salazar (Universidad Central del Ecuador), Lorena Quintero (Fundacion Biociencias), Drs Mae Moreno, Noria Escheveria, Janet Heredia and Isabelle Pallares (Universidad San Francisco de Quito), Carmen Land (University of Missouri-Columbia) and Jeffrey Stanaway (Virginia Tech). We also thank the children, parents, teachers and community leaders of Paraiso de Amigos, La Paz, Bosque de Oro I, Bosque de Oro II and Cristobal Colon where the study was conducted.

Correspondence: Dr M. M. Weigel, Department of HNFE, 205 Wallace Annex, Virginia Tech, Blacksburg, Virginia, 24061-0430, USA. E-mail <mweigel@vt.edu>.

Methods and Materials

Study protocol
The survey study was conducted during a 2.5 month period (June–August 2000) in five hamlets (Paraiso de Amigos, La Paz, Bosque de Oro I, Bosque de Oro II and Cristobal Colon) located within a rural tropical rainforest area in Pichincha Province, Ecuador. Institutional approval to conduct the study was given by the Virginia Tech Institutional Review Board and the local partner institution, Fundacion Internacional 'Biocencias'. Meetings were first held with local community leaders and the parents/guardians of prospective subjects to explain the study and request their participation. Children whose parents/guardians gave their informed written consent were enrolled in the study.

The study data were collected during house-to-house visits except in instances where the parents/guardians requested another location, which was usually the local schoolhouse. Data were collected using a structured questionnaire, anthropometry, and laboratory analysis of blood and fecal samples. Additional qualitative data were obtained through the use of key informants and by direct observation. The parents/guardians of the subjects received oral and written notification of test results. Children diagnosed with IPIs and/or iron-deficiency anemia were given the appropriate anti-parasitic treatment or iron supplements. Those identified with PEM or other medical conditions were given written referrals to local health services. The parents/guardians of subject families received individual nutrition counseling performed by qualified nutritionists and when requested, had the fecal samples of other members analysed for IPIs. In addition, each participating child received vitamin supplements. These medications, supplements, and services were provided without cost to the families.

Data collection
A structured questionnaire with open- and closed-ended questions collected information from the parents/guardians of subjects regarding sociodemographic, housing, water and sanitation characteristics. These included subject age, sex, and ethnicity, marital status, education and primary occupations of both parents, community residence site, family size and composition, house construction, water, sanitation, and garbage disposal, hygiene practices and the type and number of domestic animals living in and around the home.

Subject hemoglobin levels were determined using the HemoCue ß-Hemoglobin photometer HemoCue (Angelholm, Sweden). The instrument was calibrated daily with the control cuvette supplied by the manufacturer. The cut-off points used to identify iron-deficiency anemia were based on CDC recommended values for child age and sex.[13]

Weight was measured to the nearest 0.5 kg using a portable scale (Detecto, USA). Standing height in children aged ≥24 months was measured to the nearest 0.1 cm using a portable stadiometer. For children under 24 months, recumbent length was measured to the nearest 0.1 cm with an infant measuring board that had a fixed headboard and a movable footboard. Subject weight-for-age, weight-for-height, and height-for-age measurements were calculated using the EPINUT program of Epi-Info, version 6.[14] This compared subject measurements to an international National Center for Health Statistics growth reference curves.[15] The cut-off point used to identify malnourished children was less than –2 standard deviation units (SD) below the NCHS reference median. Children whose weight-for-height, height-for-age, and weight-for-age fell below –2 SDs were classified, respectively, with acute malnutrition (wasting), chronic malnutrition (growth stunting), or global malnutrition.

The subjects contributed three serial stool samples which were analysed for the presence of IPIs. The analyses were performed by certified laboratory technicians on the day of collection in the field laboratory of the Puerto Quito community clinic. After gross examination of fecal sample characteristics, a direct wet smear was prepared by emulsifying 2 mg of feces on a glass slide. A drop of Lugol's solution was applied to the first half of the split sample and isotonic solution to the second. The samples were covered with a coverslip and systematically observed at low (10 ×) and high magnifications (40 ×) with a light microscope in order to identify the presence of mature parasites, cysts or eggs. Children which had at least one stool sample in which an IPI was identified were classified as positive for that parasite.

Data analysis
Descriptive data are reported as means with standard deviations, frequency counts and percentages. The bivariate analyses used contingency table analysis with χ^2 to investigate differences between proportions. Factors identified as significant ($p < 0.05$) were subsequently analysed using multiple logistic regression analysis which adjusted for potential confounders. The crude and adjusted odds ratio obtained from the bivariate and multiple logistic regression analyses are presented in Tables 2–4 with their respective 95 per cent confidence intervals. Students *t*-test and one-way analysis of variance were used to test mean differences. The preliminary analyses examined the impact of: (1) parasite presence vs. absence, (2) parasite burden (low, medium, high), and (3) polyparasitism on child iron status and growth. However, only the results of those which considered the former are presented since the others yielded no significant associations with nutritional status or did not result in increased

explanatory power over that of IPI presence vs. absence.

TABLE 1
Subject and household characteristics

Characteristics	*X* ± SD	No. (%)
Child age (months)	63.4 ± 40.3	
Child ethnicity (% mestizo)		181 (88.3)
Child sex (% female)		132 (54.1)
Maternal occupation: (% housewife)		168 (83.6)
Paternal occupation: (% agriculturalist)		163 (79.5)
Maternal education (years completed)	4.3 ± 2.6	
Paternal education (years completed)	4.3 ± 2.6	
Maternal marital status (% legally married)		59 (30.1)
Household size	6.5 ± 2.1	
Household composition		
Parents	1.9 ± 0.4	
Other adults	2.6 ± 1.0	
Children < 13 years	3.1 ± 1.5	
Adolescents 13–18 years	0.8 ± 1.0	
Home ownership (%)		114 (56.7)
Home electricity (%)		68 (33.8)
Home construction (% wood/cane)		125 (62.5)
Household water source(s)		
River only		69 (34.8)
Open well only		46 (23.2)
Spring only		41 (20.7)
Municipal piped-in only		17 (8.6)
Municipal and other sources		12 (6.1)
Well and other non-municipal sources		8 (4.0)
River and other non-municipal sources		5 (2.5)
Household sanitary facilities		
Inside toilet		13 (6.4)
Outside toilet or latrine		134 (66.1)
Open ground		50 (24.6)
Household wastewater elimination		
Open ground		115 (58.1)
Septic tank		47 (23.7)
River or ravine		28 (14.1)
Other		8 (4.0)
Household garbage disposal		
Burn		55 (27.4)
Open ground		57 (28.4)
River or ravine		15 (7.5)
Bury		32 (15.9)
Municipal pick-up		19 (9.5)
Mixed		23 (11.4)
Home distance to garbage disposal site (m)	40.2 ± 16.3	
Drinking water treatment		
Boil		133 (66.2)
Chlorinate		18 (9.0)
Boil and chlorinate		6 (3.0)
Mixed (treatment and non-treatment)		5 (2.5)
No treatment		39 (19.4)
Food preparation water treatment		
Boil		56 (29.0)
No treatment		137 (71.0)
Households with domestic animals living inside or in close proximity to home (%)		190 (94.5)
No. domestic animals living inside or in close proximity to home	22.7 ± 22.3	

Results

Ninety per cent of the child subjects were infected with at least one pathogenic IPI: 51 per cent with helminths, 37.6 per cent with protozoa, and 21.4 per cent with both. The most common pathogenic species identified were *Ascaris lumbricoides* (39.7 per cent), *Giardia intestinalis* (25.2 per cent), *Trichuris trichiura* (19.7 per cent), *Entamoeba histolytica/dispar* (18.5 per cent), and *Ancylostoma duodenale* (1.7 per cent). Table 1 shows the characteristics of the subjects and their households. Most household water was reported to come from the local river, open wells, springs, or other unprotected sources. Few parents/guardians reported that their households consistently boiled their drinking water for more than 5 min or treated it with chlorine. Fewer still said that they used treated water in preparing foods. Close to 95 per cent of subject households had chickens, pigs, dogs, cows, horses, and other domestic animals living inside or in close proximity to the family's living quarters. These averaged approximately 23 animals per household.

Table 2 indicates that children infected with *G. intestinalis* had significantly reduced mean serum hemoglobin levels compared with the others. Iron-deficiency anemia was detected in 26.4 per cent of the total sample. However, the proportion of *Giardia*-infected children with iron-deficiency anemia was slightly, but not significantly, increased over that of non-infected children (29 vs. 24 per cent). No significant associations were detected between infection with the other IPIs, serum hemoglobin, and the risk for anemia.

Nearly 40 per cent of the children in the sample had stunted growth, 21.5 per cent had global malnutrition and 4.1 per cent were wasted. Table 3 indicates that children with intestinal parasitic infections were twice as likely as non-infected children to suffer from growth stunting or chronic malnutrition. Likewise, those infected with *G. intestinalis* also had double the risk for stunted growth. However, no significant associations were identified between any of the intestinal parasitic infections identified in the study and the risk for either acute malnutrition or global malnutrition.

Table 4 shows the results of the bivariate analyses that investigated IPI risk. As indicated, residence (Paraiso de Amigos), close geographic proximity to neighbors, and high intra/peri-domilliciary domestic animal concentration (> 20 animals) were associated with a significantly increased risk for infection with *G. intestinalis.* Subjects whose parents were legally married were at reduced risk. No significant associations were identified between *Giardia* infection and child gender, age, other parental characteristics,

TABLE 2
Association between intestinal parasitic infection and child iron status

Intestinal parasite species	Serum hemoglobin (g/dl)		Iron-deficiency anemia[a]	
	X ± SD	F-ratio	No. (%)	OR (95% CI)
G. intestinalis present (51)	11.8 ± 1.5*	5.2	15 (29.4)	1.30 (0.6–2.8)
G. intestinalis absent (144)	12.2 ± 1.4		35 (24.3)	1.00
E. histolytica/dispar present (42)	12.4 ± 1.5	<1.0	11 (26.2)	1.00 (0.47–2.2)
E. histolytica/dispar absent (152)	12.0 ± 1.3		39 (25.7)	
A. lumbricoides present (84)	12.3 ± 1.3	1.7	16 (19.0)	0.53 (0.27–1.1)
A. lumbricoides absent (111)	12.0 ± 1.5		34 (30.6)	1.00
T. trichiura present (43)	12.3 ± 1.1	<1.0	10 (23.3)	0.85 (0.38–1.9)
T. trichiura absent (152)	12.1 ± 1.5		40 (26.3)	1.00
A. duodenale present (4)	11.8 ± 0.6	<1.0	1 (25.0)	0.95 (1.0–9.3)
A. duodenale absent (192)	12.1 ± 1.5		50 (26.0)	1.00

[a] Cut-off points based on CDC[13] criteria according to age and sex.

* $p = 0.023$.

TABLE 3
Association between intestinal parasitic infection and child growth status

Intestinal parasite species	Growth stunting		Wasting		Global malnutrition	
	No. (%)	OR (95% CI)	No. (%)	OR (95% CI)	No. (%)	OR (95% CI)
Any parasitic infection	66 (43.1)	2.10 (1.10–3.99)*	7 (5.0)	1.80 (0.37–0.92)	37 (24.0)	1.69 (0.82–3.46)
No parasitic infection	20 (27.0)	1.00	2 (2.9)	1.00	12 (15.8)	1.00
G. intestinalis present	30 (51.7)	2.16 (1.13–4.15)**	1 (2.0)	0.38 (0.50–3.1)	15 (25.9)	1.42 (0.71–2.84)
G. intestinalis absent	56 (33.1)	1.00	8 (5.1)	1.00	34 (19.8)	1.00
E. histolytica/dispar present	17 (41.5)	1.22 (0.61–2.43)	0 (0.0)	–	9 (21.4)	1.00 (0.44–2.27)
E. histolytica/dispar absent	68 (36.8)	1.00	9 (5.1)		40 (21.4)	
A. lumbricoides present	39 (42.9)	1.38 (0.80–2.38)	3 (3.7)	0.80 (0.2–3.15)	24 (26.1)	1.60 (0.85–3.01)
A. lumbricoides absent	47 (34.6)	1.00	6 (4.7)	1.00	25 (18.1)	1.00
T. trichiura present	19 (41.3)	1.20 (0.62–2.32)	1 (2.4)	0.49 (0.06–4.0)	11 (23.9)	1.20 (0.56–2.60)
T. trichiura absent	67 (37.0)	1.00	8 (4.8)	1.00	38 (20.6)	1.00
A. duodenale present	1 (25.0)	0.55 (0.60–5.30)	0 (0.0)	–	1 (25.0)	1.24 (0.13–1.22)
A. duodenale absent	85 (37.9)	1.00	9 (4.4)		48 (21.1)	1.00

* $p = 0.023$; ** $p = 0.01$

water, sanitation and other household indicators. The multiple logistic regression analysis (Table 4) investigated which factors made significant independent contributions to *Giardia* risk. The previously identified factors of residence and domestic animal concentration retained their associations with increased risk, and parental marital status with decreased risk. However, the contribution of close home proximity to neighbors was no longer apparent and was dropped from the model.

As Table 4 also indicates, residence (Paraiso de Amigos), high domestic animal concentration, drinking water source (river), and paternal occupation (agriculturalist) were associated with an increased risk for *Amoeba histolytica/dispar* infection in the bivariate analyses, while young age (< 84 months) and adequate household garbage disposal were associated with a reduced risk. No significant associations were identified between the other factors measured and infection risk. As Table 4 indicates, the previously identified associations of high animal density, and adequate garbage disposal with infection risk remained significant in the multiple logistic regression analysis. However, the factors of

TABLE 4
Factors associated with the risk for intestinal parasitic infections

	No. (%)	OR (95% CI)	*p*	Adjusted OR (95% C.I.)	*p*
1. Giardia intestinalis infection					
Residence					
Paraiso de Amigos	13 (44.8)	2.81 (2.17–6.72)	0.02	3.53 (1.23–10.1)	0.018
Other	46 (22.4)	1.00		1.00	
Maternal marital status					
Legally married	6 (10.9)	0.25 (0.10–0.61)	0.003	0.28 (0.11–0.74)	0.010
Other	44 (33.3)	1.00		1.00	
No. of domestic animals living in/around home					
> 20	26 (36.1)	2.33 (1.20–4.57)	0.019	3.78 (1.68–8.49)	0.001
≤ 20	22 (19.5)	1.00		1.00	
House proximity to nearest neighbor					
≤ 100 m	39 (33.6)	2.36 (1.14–4.92)	0.03	1.26 (0.54–2.91)	NS
> 100 m	12 (17.6)	1.00		1.00	
2. Amoeba histolytica/dispar infection					
Residence					
Paraiso de Amigos	24 (40.7)	5.59 (2.61–12.0)	0.0001	1.68 (0.20–15.13)	NS
Other	19 (10.9)	1.00		1.00	
Subject age					
≤ 84 months	24 (13.5)	0.08 (0.04–0.18)	0.0001	0.49 (0.22-1.1)	NS
> 84 months	19 (35.2)			1.00	
Paternal occupation					
Agriculturalist	37 (23.9)	4.10 (1.02–8.00)	0.047	2.78 (0.60–12.1)	NS
Other	2 (7.1)	1.00		1.00	
No. of domestic animals living in/around home					
> 20	23 (31.9)	2.30 (1.14–4.62)	0.019	2.29 (1.1–4.98)	0.035
≤ 20	19 (17.0)	1.00		1.00	
Source of household water					
River water only	20 (31.3)	2.23 (1.04–4.80)	0.039	1.73 (0.79–3.78)	NS
Other	21 (16.9)	1.00		1.00	
Household garbage					
Adequate disposal[a]	15 (14.9)	0.41 (0.19–0.87)	0.019	0.36 (0.17–0.80)	0.012
Inadequate disposal	27 (30.0)	1.00		1.00	
3. Trichuris trichuria infection					
Residence					
La Paz	18 (30.5)	2.31 (1.16–4.58)	0.025	1.84 (0.87–3.87)	NS
Other	28 (16.0)	1.00		1.00	
Subject age					
≤ 60 months	13 (10.9)	0.31 (0.15–0.67)	0.0016	0.36 (0.16–0.81)	0.013
> 60 months	32 (28.1)	1.00		1.00	
Maternal education					
≤ 3 years	26 (32.5)	2.62 (1.29–5.32)	0.011	1.96 (0.93–4.13)	NS
> 3 years	16 (15.5)	1.00		1.00	

[a] Garbage disposed of by burning, burying and pickup.

household water source, paternal occupation, age and residence were no longer evident.

Table 4 reveals that residence in La Paz and a low maternal education (≤ 3 years) were associated with an increased risk for *T. trichuria* infection. Younger subject age (≤ 60 months) was associated with a decreased risk. No significant associations were observed between *Trichuris* infection and the other indicators investigated. The multiple logistic regression analysis results revealed that although subject age retained its respective identified contributions to trichuriasis risk, that of residence and education were no longer evident.

The mothers of the children identified with ascariasis were slightly, but significantly, less well educated compared with those without the infection, i.e., 3.8 ± 2.7 years vs. 4.7 ± 2.8 years; $p = 0.022$). However, no significant associations were identified between *Ascaris* infection and the sociodemographic and other indicators measured in the study. None of the indicators measured in the study predicted the risk for hookworm infection. This may be the result of insufficient statistical contrast related to the low hookworm infection frequency.

Discussion

The study investigated the prevalence, predictors and nutritional consequences of intestinal parasitic infections in rural Ecuadorian children. The pattern of a high prevalence and predominance of helminth infection concurs with that noted for other tropical child groups living in the Ecuadorian Amazon[8,11] but differs from that reported for children living in subtropical[12] and highland areas[10] of the same province.

The observed relationship between *Giardia* infection and hemoglobin is consistent with authors who linked the parasite with significantly reduced iron status in children living in Venezuela,[16] the Gaza[17] and Egypt[18] but differs from de Morais and associates[6] who reported that Brazilian children with asymptomatic infection showed no evidence of reduced hemoglobin levels compared to non-infected controls and did not benefit from oral iron therapy. On the other hand, Jimenez and associates[16] showed that *Giardia*-infected children who were given a single dose of secnidazole had significantly increased mean hemoglobin values by 15 days post-treatment.

Most of the risk for growth stunting found in children with IPI infection appeared to be attributable to the robust contribution of *G. intestinalis* infection rather than other species. Previously published studies have been inconsistent with respect to the association of giardiasis and growth stunting. For example, several authors have reported significant reductions in the linear growth of *Giardia*-infected children[5,19] and experimental mammals.[20] Others have demonstrated increased height and weight in children treated for giardiasis.[21] Furthermore, Fraser and colleagues[5] have reported finding that growth stunting followed rather than preceded *Giardia* infection in their prospective study of Bedouin infants. It has been suggested that *Giardiasis* may cause nutrient malabsorption and other adverse changes.[2,22,23] The resulting deficits in growth may be exacerbated where PEM is already present.[24]

In contrast to these findings, other authors have reported the lack of a significant association between giardiasis and growth stunting in young infants[25] or older children.[26] While differences in study design and methodology may be responsible for some of these between-study discrepancies, population differences in infection intensity and burden, nutritional status, breastfeeding, patterns of growth, and other attributes appear responsible for others. Evidence in support of the importance of population differences has been noted by Fraser and colleagues[5] who reported that the impact of giardiasis on child growth was different for two populations who lived in the same geographical area of Israel but who had distinct lifestyle and environmental characteristics.

The most consistent predictor of protozoal IPI risk was a high intro-/peri-domicilliary concentration of domestic animals. Children who lived in such households had a risk for *Giardia, E. histolytica/diapar*, and *Blastocysis hominis* infection that was two to five times higher than that of those living in homes where fewer animals were present. Pigs, dogs, cats, chickens, cattle and horses have been identified as possible reservoirs for *Giardia* and other common protozoal IPIs.[27–33] In the Ecuadorian group studied, this may occur through contamination of the children's drinking water, their home environment, objects they play with, and via blowing dust which contains parasite cysts and mature forms.

Several caveats should be considered when evaluating the study results. First, self-reported data obtained by interview may be subject to significant under- or over-reporting of specific characteristics depending on whether the respondents believe these to be socially desirable. However, the use of direct observation during the home visits allowed for the verification of many of the reported household attributes. This information was also cross-checked with school teachers and other family members when possible. It is also possible that the detection methods used may not have been sensitive enough to detect all cases of *G. intestinalis* and other IPIs. Several newer techniques are reported to have higher sensitivity compared with the conventional methods used in the present study.[34] However, we attempted to offset this limitation by analysing three serial fecal samples per subject, an approach which is reported to increase the yield of protozoa, for example, by an additional 23 per cent for *A. histolytica* and 11.3 per cent for *G. intestinalis.*[34] Although one study has reported that growth faltering occurred subsequent to *Giardia* infection,[5] the cross-sectional design of the present investigation did not allow for the definitive determination of whether infection itself augmented the risks for stunted growth and decreased hemoglobin or whether malnutrition increased infection susceptibility. Prospective cohort studies are needed to clarify this question and the mechanisms responsible. In addition, future studies should investigate the role of domestic animals in infection transmission since this risk factor is potentially modifiable.

References

1. World Health Organization. Intestinal Parasites. Available from: http:/www.who.int/ctd/intpara/burdens.htm (accessed August 2001).
2. Stephenson LS, Latham MC, Ottesen EA. Malnutrition and parasitic helminth infections. Parasitology 2000; 121: S23–38.
3. Stephenson LS, Latham MC, Ottesen EA. Global malnutrition. Parasitology 2000; 121: S5–22.

4. Northrop-Clewes CA, Roushman EK, Mascie-Taylor CGN, Lunn PG. Anthelmintic treatment of rural Bangladeshi children: effect on host physiology, growth, and biochemical status. Am J Clin Nutr 2001; 73: 53–60.
5. Fraser D, Bilenko N, Deckelbaum RJ, Dagan R, El-On J, Naggan L. *Giardia lamblia* carriage in Israeli Bedouin infants: risk factors and consequences. Clin Infect Dis 2000; 30: 419–24.
6. De Morais MB, Suzuki HU, Corral JN, Macgado NL, Neto UF. Asymptomatic giardiasis does not affect iron absorption in children with iron deficiency anemia. J Am Coll Nutr 1996; 15: 434–38.
7. Sempertegui R, Naranjo P, Padilla M. Panorama Epidemiologico del Ecuador. Ministerio de Salud Publica, Quito, Ecuador, 1995.
8. San Sebastian MS, Santiago S. Control of intestinal helminths in schoolchildren in Low-Napo, Ecuador: impact of a two-year chemotherapy program. Rev Socied Bras Med Trop 2000; 33: 69–73.
9. Levav M, Mirsky AF, Scahnatz PM, Castro S, Cruz ME. Parasitic infection in malnourished school children effects on behaviour and EEG. Parasitology 1995; 110: 103–11.
10. Ortega F. A cost-effectiveness analysis of anthelminthic intervention for community control of ascariasis: Traditional vs. pharmaceutical therapy. Oregon State University, Dissertation Abstracts Online 2000; 61, no. 06B: 3025.
11. Benefice E, Barral H. Differences in the life style and nutritional status between settlers and Siona-Secoya Indians living in the same Amazonian milieu. Ecol Food Nutr 1991; 25: 307–22.
12. Weigel MM, Armijos RX, Zurita C, Racines J, Reddy A, Mosquera J. Cutaneous leishmaniasis and nutritional status in rural Ecuadorian children. J Trop Pediatr 1995; 41: 22–8.
13. Centers for Disease Control. Recommendations to prevent and control iron deficiency in the United States. MMWR 1998; 47: 11–13.
14. Dean AG, Dean JA, Coulombier D, *et al*. Epi-info, Version 6, A Word-Processing, Database, and Statistics Program for Public Health on IBM-compatible Microcomputers. Center for Disease Control, Atlanta, GA; 2000.
15. Dibley MJ, Goldsby JB, Staehling NW, Trowbridge FL. Development of normalized curves for the international growth reference: historical and technical considerations. Am J Clin Nutr 1987; 46: 736–48.
16. Jimenez JC, Rodriguez N, Di Prisco MC, Lynch NR, Costa V. Haemoglobin concentrations and infection by *Giardia intestinalis* in children: effect of treatment with secnidazole. Ann Trop Med Parasitol 1999; 93: 823–27.
17. Shubair ME, Yassin MM, Al-Hindi AI, *et al*. Intestinal parasites in relation to haemoglobin level and nutritional status of school children in Gaza. J Egyp Soc Parasitol 2000; 30: 365–75.
18. Curtale F, Nabil M, El Wakeel A, Shamy MY, and the Behera Survey Team. Anaemia and intestinal parasitic infections among school age children in Behera Governorate, Egypt. J Trop Pediatr 1998; 44: 323–28.
19. Farthing MJG, Mata L, Urrutia JJ, Kronmal RA. Natural history of *Giardia* infection of and children in rural Guatemala and its impact physical growth. Am J Clin Nutr 1986; 43: 395–405.
20. Astiazaran-Garcia H, Espinosa-Cantellano M, Castanon G, Chavez-Munguia B, Martinez-Palomo A, *Giardia lamblia*: effect of infection with symptomatic and asymptomatic isolates on the growth of gerbils (*Meriones unguiculatus*). Exp Parasitol 2000; 95: 128–35.
21. Gupta MC, Urrutia JJ. Effect of periodic antiascaris and antigiardia treatment on nutritional status of preschool. Am J Clin Nutr 1982; 36: 79–86.
22. Solomons NW. Giardiasis: nutritional implications. Rev Infect Dis 1982; 4: 859–69.
23. Eckmann L, Gillin FD. Microbes and microbial toxins: paradigms for microbial-mucosal interactions I. Pathophysiological aspects of enteric infections with the lumen-dwelling protozoan pathogen *Giardia lamblia*. Am J Physiol Gastrointest Liver Physiol 2001; 280: G1–6.
24. Leitch GJ, Udezulu IA, He Q, Visvesvara GS. Effects of protein malnutrition on experimental giardiasis in the Mongolian gerbil. Scan J Gastroenterol 1993; 28: 885–93.
25. Lunn PG, Hezekioh EO, Northrop-Clewes CA, Boyce SA. *Giardia intestinalis* is unlikely to be a major cause of poor growth of rural Gambian infants. J Nutr 1998; 129: 872–77.
26. Saldiva SR, Silveira AS, Philippi ST, *et al*. *Ascaris-trichuris* association and malnutrition in Brazilian children. Pediatr Perinat Epidem 1999; 13: 89–98.
27. Mahmud MA, Chappell C, Hossain MM, Habib M, Dupont HL. Risk factors for development of first symptomatic *Giardia* infection among infants of a birth cohort in rural Egypt. Am J Trop Med Hyg 1995; 53: 84–8.
28. Fayer R, Trout JM, Graczyk TK, Lewis EJ. Prevalence of *Cryptosporidium, Giardia* and *Eimeria* infections in post-weaned and adult cattle on three Maryland farms. Vet Parasitol 2000; 93: 103–12.
29. Thompson RCA. Giardiasis as a re-emerging infectious disease and its zoonotic potential. Int J Parasitol 2000; 30: 1259–67.
30. Acerrano, A. H_2uh-O; the aqua's not so pura. Sports Afield 1997; 217: 64–72.
31. Bednarska M, Bajer A, Sinski E. Calves as a potential reservoir of *Cryptosporidium parvum* and *Giardia* sp. Ann Agric Environ Med 1998; 5: 135–38.
32. Devera R, Requena I, Velasquez V, Castillo H, Gonzalez R. Pigs as reservoirs of *Blastocystis* spp. in a rural community of the state of Bolivar, Venezuela. Enferm Infecc Microbiol Clin 1999; 17: 422.
33. Sullivan PS, DuPont HL, Arafat RR, *et al*. Illness and reservoirs associated with *Giardia lamblia* infection in rural Egypt: the case against treatment in developing world environments of high endemicity. Am J Epidemiol 1988; 127: 1272–81.
34. Garcia LS. Practical Guide to Diagnostic Parasitology. Washington, DC, ASM Press, 1999.