

**INTESTINAL PARASITIC INFECTIONS: PREVALENCE, RISK FACTORS AND
CONSEQUENCES FOR CHILD GROWTH, IRON STATUS AND DEVELOPMENT IN
RURAL ECUADOR.**

Mamie- Eleanor Sackey

Thesis submitted to the Faculty of the Virginia Polytechnic and State University in partial
fulfillment of the requirements for the degree of

MASTERS OF SCIENCE
in
Human Nutrition Foods and Exercise

Mary-Margaret Weigel, Chair

Barbeau William, E

Anne M. Zajac

July 18, 2001

Blacksburg, VA

Keywords: intestinal parasites, children, risk factors, growth, nutrition, psychomotor
development, Ecuador

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by

Mamie-Eleanor Sackey

Mary Margaret Weigel, Chairperson

Department of Human Nutrition, Foods and Exercise.

(Abstract)

Intestinal parasitic infections (IPI's) are considered to be a public health problem of global importance by the World Health Organization. The present epidemiologic survey study investigated the prevalence, risk factors, and consequences of pathogenic IPI's on the growth, nutrition and psychomotor development of 244 Ecuadorian children aged 0.2-14 years. The study was conducted in five rural hamlets located in a tropical rainforest area in northwest Ecuador. The study data were obtained by means of a structured questionnaire, a developmental screening examination, anthropometry, and lab analysis of blood and fecal samples. Data analysis was conducted using appropriate bivariate and multivariate statistical techniques.

The study results revealed that 90% of the child subjects were infected with at least one pathogenic IPI species. Fifty-one percent were identified with helminthic infections, 37.6% with protozoal infections, and 21.4% were infected with both. The most common intestinal parasites detected were *Ascaris lumbricoides* (39.7%), *Giardia intestinalis* (25.2%), *Trichuris trichiura* (19.7%), *Entamoeba histolytica/dispar* (18.5%), *Blastocystis hominis* (13.3%), and *Ancylostoma duodenale* (1.7%). The prevalence of growth stunting (40%) and iron-deficiency anemia (26%) also was high. Children infected with *Giardia* exhibited a risk for stunted growth that was twice that of their non-infected counterparts (51.7% vs. 33.1%; OR=2.16, 95% C.I.= 1.13-4.15; p= 0.01). They also had significantly reduced mean blood hemoglobin levels compared to non-infected children 11.8 ± 1.5 .g/dL vs. 12.2 ± 1.4 g/dL; p= 0.023) but the proportion with iron-deficiency anemia was slightly but not significantly increased (29.4% vs. 24.3%). The characteristic most consistently associated with risk for pathogenic protozoal IPI's was a high density of domestic animals living in and around the home. Children who lived in such households had a risk for infection that was 2-5 times greater than others. This suggests that

domestic animals were important reservoirs for IPI infection in the child group studied. Contrary to the *a priori* hypothesis, no gender, ethnic, nor age differences in infection risk were identified except for *Trichuris* infection, which was reduced in younger children contrary to expectations. Mass or targeted chemotherapy combined with health education and promotion are needed to reduce the cycle of infection and re-infection and the negative impact of these on child growth and iron status. Health education and promotion messages can be incorporated into other types of programs already in place in local schools and by the Ecuadorian Ministries of Public Health, Education, and Social Welfare and other agencies.

ACKNOWLEDGEMENTS

My acknowledgements go to the following organizations for awarding me the funds, which made possible this research: Howard Massey Food and Nutrition Scholar Award, HEPLER Summer Research Fellowship and the Fundacion Internacional Biociencias.

I would like to express my sincere thanks to Dr. Mary-Margaret Weigel for all the support and guidance she gave me. Quick to point out my mistakes and even quicker to encourage me I have learnt a lot under your guidance. Needless to say you have been a great mentor to me both as an advisor, lecturer and friend. My sincere thanks also go to my committee members Dr. William Barbeau and Dr. Anne Zajac. I am deeply grateful for all your patience and kind words of advice.

I also thank Ms. Carmen Land (Univ. Mo-Colombia), Jeff Stanaway (Virginia Tech), the nutrition team from the University of San Francisco de Quito, the Fundacion Biociencias research team and the medical students from the Central University of Quito. Together we formed a great team and I learnt a lot working with you all. Sincere thanks goes also to the children and parents in rural Ecuador, for their kindness in letting myself and the rest of research team into the privacy of their homes and lives. Without your willing participation this research would never have been realized.

I would also like to my parents both in Ghana and in the States: Rev & Mrs. A. A Sackey and Mr. & Mrs. G. A Kofi, you all contributed to make my stay here as pleasant and stress free as possible. Special thanks go also to my sister and friend Michelle Penn whose wise counsel, patience and spiritual strength helped see me through the tough days and also to my friend James and the Navigators may the Lord truly bless you all.

I extend my thanks also to all the faculty and staff of the HNFE department especially to Dr. Forrest Thye for all the help and advice given me.

Finally to the one person to whom I owe my life and through whom I had the strength to go from day to day, the Lord and Savior Jesus Christ I dedicate this paper and raise my hands in deep and sincere thanks.

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Chapter 1: INTRODUCTION

Intestinal parasitic infections (IPI's) caused by pathogenic helminthic and protozoal species are endemic throughout the world. They affect an estimated 3.5 billion persons and cause clinical morbidity in approximately 450 million (WHO 2000). Developing countries are reported to be the most affected and within these, the majority of the cases occur among school aged children (WHO 1996;Montresor et al. 1998). The distribution of intestinal parasitic infection depends on many factors. These include sociodemographic variables associated with poverty such as reduced access to adequate sanitation, potable water, and health care as well as the prevailing climatic and environmental conditions (Mata 1982;WHO 1996; Montresor et al. 1998). The economic burden caused by hookworm, roundworm and whipworm infections is high. This was recently estimated by Stephenson and colleagues (2000 *b*) to cost 39.0 million disability-adjusted life years (DALYs).

Young children are reported to be disproportionately affected by IPI's compared to adults due to their increased nutritional requirements and less developed immune systems (Scrimshaw 1994). Intestinal parasitic infections in this age group have been linked with significantly reduced growth (Stephenson et al. 1993) and an increased risk for protein-energy malnutrition (Stephenson et al.2000*a*) including growth stunting (Oberhelman et al 1998; Tsuyuoka et al 1999), iron-deficiency anemia (Stoltzfus et al. 1997), and reduced cognitive/psychomotor development (Nokes et al 1992; Callendar et al. 1994; Sakti et al. 1999).

The global prevalence of the soil-transmitted helminthes is high. Recent estimates indicate that approximately 1472 million people have roundworm infection, 1298 million have hookworm infection, and 1049 million have whipworm infection (Crompton 1999). In the Americas region, the respective prevalence of roundworm, hookworm and whipworm is estimated at 182 million, 152 million and 135 million respectively (Chan et al., 1994). The nutritional consequences of intestinal infections caused by helminthic species are substantial. For example, ascariasis has been linked with decreased fat, protein and vitamin A absorption (O'Lorcain and Holland 2000), iron deficiency anemia (Curtale et al. 1993; Stoltzfus et al. 1997) and in the case of severe infection, intestinal obstruction (de Silva et al. 1997). Trichuriasis often occurs in tandem with ascariasis. It is estimated to cause morbidity in 87-133 million persons

worldwide (Chan et al. 1994). Heavy trichuriasis burdens have been linked with iron-deficiency anemia (Stoltzfus et al. 1997; Chan et al. 1994), growth retardation, and reduced cognitive development in children (Chan 1994). Morbidity caused by hookworm infection affects 159 million persons per year (Chan et al, 1994). The major health consequence of hookworm infection is anemia. For example, Stoltzfus et al. (1997) have reported that hookworm was responsible for ~25% of all anemia cases and 35% of those caused by iron-deficiency among the 3,595 Zanzibarian school children they studied. It is also a major cause of anemia in pregnant women (Stephenson et al. 2000b).

The global prevalence of intestinal infections caused by pathogenic protozoal species is also reported to be high. *Entamoeba histolytica*, the cause of amoebiasis, is estimated to inflict severe disease in 48 million individuals around the globe (Petri and Singh 1999). Infections caused by *Entamoeba histolytica* result in the deaths of ~100,000 persons per year, second only to another protozoal infection, malaria (WHO 1997). Severe *E. histolytica* infections in children often cause dysentery and bloody diarrhea; amebic liver abscess may also occur although they are less common (Petri and Singh 1999).

The worldwide prevalence of giardiasis is suspected to be in the millions but precise estimates are not available. *Giardia intestinalis* is the most common protozoal infection of the human small intestine (Eckmann and Gillin 2001). It also is one of the first intestinal protozoal infections found in developing country infants (Farthing et al 1986). *Giardia* infection is associated with the malabsorption of fats, carbohydrates, and vitamins, especially vitamins A and B₁₂ (Solomons 1982). Recent authors also have linked it with reduced serum hemoglobin levels (Curtale et al. 1998).

Intestinal parasitic infections can adversely impact host nutritional status in several different ways. Specifically, they can depress appetite and food intake (Stephenson et al. 1993; Hadju et al. 1996), compete for micronutrients (Hlaing 1993; Stoltzfus et al. 1997), cause nutrient malabsorption (Solomons 1982; Hlaing 1993) or blood loss resulting in the loss of iron or other essential nutrients (Stoltzfus et al. 1998). Some infections can have a negative effect on metabolic or nutrient excretion rates (Stephenson et al. 2000 b).

The precise impact of IPI on child nutrition, growth and development appears to depend on the species and burden (Chan et al. 1994; Oberhelman et al. 1998) and host immune response (Eckmann and Gillin 2000). Although most previous authors have concentrated on severe infection, several recent studies have shown that those which are of light-moderate intensity also can have negative consequences for child growth and health (Simeon et al. 1995; Wilson et al. 1999). Different from most gastrointestinal bacterial and viral infections, IPI's tend to be chronic. Thus, their impact on host nutrition tends to be longer-term (Oberhelman et al. 1998).

Despite recent advances, a number of important questions still remain unanswered regarding intestinal parasitic infection risk and its consequences in child populations. For example, it is still not clear whether certain age, gender, and ethnic groups are more likely to become infected. It also is uncertain at which age the negative nutritional consequences associated with IPI first become evident. In addition, the evidence has not been entirely consistent regarding the precise impact of individual parasite species nor the effect of polyparasitism on specific indicators of child nutritional status. Few data have been published about the effects of IPI's on child psychomotor development. Finally, most prior authors have concentrated on the nutritional, psychomotor and health impacts of pathogenic helminthes rather than on protozoal infections such as amoebiasis and giardiasis even though the latter are often more prevalent in most developing populations (El Molla 1990; Yassin et al 1999).

The three major objectives of the present study were to investigate: (1) the prevalence, (2) risk factors, and (3) consequences of intestinal parasitic infections with respect to the growth, iron status, and psychomotor development of infants and children living in an endemic tropical rainforest area of rural northwest Ecuador. This developing population was chosen because it is reported to be characterized by high population prevalences of intestinal parasitic infection (MSP 1992), protein-energy malnutrition (Friere et al. 1984; Varrea et al., 1997), and iron-deficiency anemia (MSP 2001). Fifty six percent of the country's inhabitants are reported to live below the level of poverty according to government statistics with an additional 38% of people in the rural areas living in extreme poverty (SIISE 2001*a*). Most homes, especially those in rural areas, also have substandard housing, water and sanitation as well as decreased access to health care services (SIISE 2001 *a-c*, MSP 2001). Several studies conducted during the mid-to-late

1990's reported that approximately 50-70% of the rural residents investigated in the tropical-subtropical region of NW Ecuador were infected with one or more pathogenic intestinal parasite species. The IPI's reported as most common in the adult and child groups surveyed were *E. histolytica/dispar*, *G. intestinalis*, *A. lumbricoides*, *T. trichiura* (Weigel et al. 1992; Weigel & Armijos 1995; Weigel et al. 1995). Likewise, the prevalence of child growth stunting was 38% and iron-deficiency anemia, 12% (Weigel et al. 1995).

CHAPTER 2: LITERATURE REVIEW

Intestinal Parasitic Infections

Intestinal parasitic infections (IPI's) enjoy a wide global distribution. They are estimated to affect an estimated 3.5 billion people, most of whom are children residing in developing countries (WHO 2000). The major IPI's of global public health concern are the protozoal species *Entamoeba histolytica* and *Giardia intestinalis* and the soil transmitted helminthes *Ascaris lumbricoides*, *Trichuris trichiura*, and hookworm (WHO 1999;WHO 2000). The incidence and prevalence of these parasitic pathogens varies both between and within countries. The majority of infections are associated with poverty conditions such as reduced access to safe drinking water, adequate sanitation and hygiene, housing, and inadequate access to health care (Mata 1982;Montresor et al. 1998). They also are affected by poor family and community hygiene and sanitation practices and prevailing climatic and environmental conditions (Jemaneh 1998). These conditions lay the stage for the continuous transmission of the IPI's (Mata 1982; Montresor et al. 1998; Crompton 1999).

Intestinal Protozoal Infections

The protozoa are an extremely diverse group of unicellular organisms occurring in almost all of the ecological niches known to humans, including the bottom of hot springs and the edges of ice flows. Even though the majority of protozoa occur as free-living organisms in the soil, moist, marine or freshwater environments, a substantial number also exist as mutualists, commensals or parasites (Melhorn 1988; Katz et al. 1989). Protozoan parasites are known to affect all species of vertebrates and many invertebrates. They are able to adapt to life in virtually all body sites of their hosts. Their characteristic high infectivity enhances their pathogenicity within the host (Katz et al 1989; Neva and Brown 1994).

***Entamoeba Histolytica/Dispar* Infection**

Entamoeba histolytica infection is common in most developing countries. It also is becoming more frequent in the United States and other developed countries as the result of increasing tourism abroad and a rising number of refugees and other immigrants and non-immigrants originating in endemic countries (Petri and Singh 1999). The two species *Entamoeba histolytica* and *Entamoeba dispar* are morphologically identical but pathologically distinct (WHO 1997; Petri and Singh 1999). Together they infect around 10% of the world's population (Walsh 1986). However, only *E. histolytica* is capable of causing disease (WHO 1997). Colonization with *E. dispar* is said to be three times more common than *E. histolytica* in developing countries while it is ten times more common in developed nations (Petri and Singh 1999).

Entamoeba histolytica is reported to be responsible for approximately 50 million cases of invasive amoebiasis (Petri and Singh, 1999) and upwards of 100,000 deaths/year (WHO 1997). Thus, it is ranked second only to malaria as the cause of mortality due to a protozoal infection (WHO1997). The parasite normally inhabits the large intestine but is also capable of invading other organs such as the liver, brain and spleen (Petri and Singh 1999). The majority of amoebic infections are reported to occur in Central America, South America, Africa and Asia. These are often associated with poor water and food hygiene and sanitation practices (Petri and Singh 1999). Humans are the most significant reservoir of infection even though morphologically identical amoebae have been isolated from certain domestic and wild animals (Katz et al. 1989).

The lifecycle of *E. histolytica* includes the infective cyst and the invasive trophozoite forms. Infection is acquired primarily through the ingestion of infective cyst forms present in fecally contaminated water and food. It also can be transmitted by person-to-person contact (Petri and Singh 1999). The quadrinucleate cysts measure approximately 10 – 20µm in diameter and are resistant to gastric juices present in the human stomach. The amoeba excysts in the small intestine by releasing four metacystic amoebae that divide into eight trophozoites which then move down to the large intestine (Neva and Brown 1994; Katz et al.1989; Garcia 1999; Petri and Singh1999). Intestinal stasis allows the amoeba to establish a foothold in the cecal area of the

large intestine even though a portion may be swept along to the sigmoidorectal region or even out of the body (Neva and Brown 1994). The chances of establishing a foothold are improved when the volume of food intake is low and the number of parasites is high (Neva and Brown 1994).

The trophozoites measure approximately 10-60µm in size (Garcia 1999). These adhere to the colonic mucin glycoproteins via a galactose and N – acetyl-D- galactosamine specific lectin (Stanley and Reed 2000). Host colonic cells are killed via the induction of an apoptotic cascade (Petri and Singh 1999). Colonic lesions formed by the trophozoites vary from mucosal thickenings to characteristic flask shaped ulcerations to necrosis of the intestinal wall (Neva and Brown 1994; Petri and Singh 1999). The trophozoites are typically found in the periphery of the necrotized tissues. Reproduction is by binary fission or the formation of cysts. These pass out with the stool and are immediately infective (Neva and Brown 1994). In the extraintestinal invasion no cysts are formed and trophozoites proliferate solely by binary fission (Katz et al.1989).

Asymptomatic infection with *E. histolytica* is defined as the presence of cysts in stools in the absence of colitis or extraintestinal infection. These healthy carriers may pass millions of cysts in the stool per day as the trophozoites multiply in the intestinal lumen (Petri and Singh 1999, WHO 1997, Neva and Brown 1994). Approximately, 90% of all intestinal *E. histolytica* infection are asymptomatic. However, even asymptomatic infection is associated with a small but significantly increased risk for developing invasive amoebiasis (Petri and Singh 1999; Mohamed et al 2000). Clinical symptoms of acute intestinal amoebiasis include diarrhea, bloody stool that may contain necrotic mucous, abdominal pain, tenderness and fever (Petri and Singh 1999). Symptoms of amoebic liver abscess usually involve fever, right upper abdominal tenderness/ pain, weight loss and colitis (Katz et al.1989; Neva and Brown 1994; Petri and Singh 1999). Amebic liver abscess is 7-10 times more common in men than women but this sex difference in risk has not been reported for children (Petri and Singh 1999).

Amoebiasis infection can be controlled through proper treatment and/or disposal of raw sewage and maintaining clean water supply including the protection of open wells, springs and

rivers from contamination with sewage and feces. The risk for infection can also be reduced via the adequate boiling of drinking water or treatment of water with chlorine or iodine. The exterior of raw vegetables and fruits should also be washed with soap and soaked in vinegar for 15 minutes prior to consumption (Petri and Singh 1999).

***Giardia* Infection (*Giardia intestinalis*)**

Giardiasis is caused by infection with the enteric pathogenic intestinal flagellate, *Giardia intestinalis*. It is considered the most common human intestinal protozoan infection. Humans are the preferred host but morphologically identical organisms have been isolated in dogs, beavers, ungulates and other mammals (Katz et al 1989). The frequent availability of hosts and reservoirs for the protozoan has contributed to the high prevalence of this infection in many industrialized countries (Juckett, 1996). The transmission of the parasite is mainly by the fecal-oral route but also occurs by human-to-human transmission (Mata 1982; Garcia 1999).

Giardia exists in two forms: the infective cyst and the not invasive trophozoite which typically inhabits the duodenum and upper jejunum of humans (Neva and Brown, 1994). The round or oval shaped cysts, which are the infective form of the protozoan, are approximately 11–14 µm long and 7-10 µm wide (Garcia 1999). After ingestion the cysts pass unharmed by gastric acid through the stomach to the small intestine. Excystation normally occurs in the duodenum. The trophozoites resist the ordinary peristaltic movements by adhering to the microvillous border of the epithelial cells using a ventral disc-shaped sucker (Solomons, 1982). Infection with *Giardia* is usually confined to the upper small intestine but also has been observed in the bile duct and gall bladder of ill patients (Ibid). Conditions of achlorhydria and hypochlorhydria in the host's gut are reported to enhance *Giardia* proliferation (Lopez et. al 1978, Brown and Neva 1994). Identification of the parasite is usually made by microscopic examination of direct fecal smears for either trophozoites or cysts in the feces. Clinical symptoms of giardiasis include diarrhea, steatorrhea, epigastric pain, wasting, hypoalbuminemia and impaired absorption of folate and vitamin B₁₂ (Neva and Brown 1994; Solomons 1982).

Intestinal Helminth Infections

The three major soil transmitted helminthes considered to be of global public health concern are *Ascaris lumbricoides* (roundworm), *Trichuris trichiura* (whipworm), and *Ancylostoma duodenale* or *Necator americanus* (hookworm). Over one billion of the world's population is estimated to be infected with these parasites; two billion are more said to be at risk (Montresor et al. 1998). Children are reported to be at especially increased risk for severe infections and the morbidity and mortality associated with these (Chan et al 1994). Helminth, infection has been linked with an increased risk for several nutritional anemias, protein-energy malnutrition, and reduced physical growth and development in infants and children (Connolly and Kvalsvig 1993; Nokes and Bundy 1994; Stephenson et al. 2000b).

Roundworm Infection (Ascaris Lumbricoides)

Ascaris lumbricoides, a soil transmitted helminths, is reported to infect at least one-fourth of the world's population (Crompton 1994). Annual morbidity associated with the parasite has been estimated by the WHO at 60,000 with another 250 million people said to be at risk for acquiring the infection (Montresor et al. 1998). Both domestic and wild animals are common reservoirs for *A. lumbricoides*. It should be noted that some controversy exists regarding whether *Ascaris suum*, the pig round worm, is genetically identical to *A. lumbricoides* (Anderson 1995; Peng et al. 1998).

Ascaris lumbricoides is a robust parasite. This quality is due, in part, to the resilient nature of its eggs, which are capable of surviving a wide range of hot and cold temperatures, chemicals, chemical disinfectants and other extreme conditions (Neva and Brown 1994). The eggs of *Ascaris* are one of the most resilient of the helminth eggs and can remain infective for years embedded in the soil (Gilgen and Mascie-Taylor 2000; Crompton 1994). *Ascaris* is the largest of the human intestinal parasitic nematodes. Mature male and females can grow from 15–30 cm and 20- 35cm in length, respectively. Both usually inhabit the jejunum where they feed on the semi-digested food present in the host (Neva and Brown 1994). They also secrete antitrypsins

and thus, are capable of adequately competing with the host system for ingested proteins. They resist the normal peristaltic movement of the gut by assuming an S-shaped configuration, pressing their outer cuticle against the columnar epithelium of the host. This allows them to move against peristalsis (Katz et al. 1989). The mature female lays approximately 200,000 eggs per day, which then pass out in the feces in an unembryonated form (Thein Hlaing 1993; Crompton 1994). Under favorable conditions, such as adequate moisture and shade the eggs embryonate to form the second stage larva in which they remain until they are subsequently ingested (Thein Hlaing 1993; Crompton 1994).

Infection is acquired through the ingestion of infective eggs from fecally contaminated food or water. Since the eggs are very sticky, they readily adhere to raw fruits and vegetables, which are washed with contaminated water or fertilized with contaminated night soil. In highly endemic areas, *Ascaris* eggs may be found on eating or cooking utensils, or under the fingernails. They also may circulate in household dust and air where they are inhaled or swallowed (O’Lorcain and Holland 2000).

Ingested eggs hatch in the duodenum to release the larvae, which penetrate the intestinal wall to reach the venules. From here, the larvae are carried passively to the liver via portal circulation. The larvae subsequently migrate to the heart through the inferior *vena cava* and are carried to the lungs by the pulmonary circulation where they lodge in the alveolar spaces where and undergo their second and third molt. Next, third stage larvae break out of the alveolar space and migrate up the trachea into the pharynx where they are swallowed. After arriving at the small intestine, they undergo one more molt before they are transformed into the mature worm. These mature forms then mate and lay more eggs (Neva and Brown 1994). This process, which starts with the ingestion of infective eggs and ends with the production of eggs, takes approximately 2-3 months (Thein Hlaing 1993). Adult worms have a life span of 1-2 years. Adult females can generate eggs for up to 1 year (Thein Hlaing 1993; O’Lorcain and Holland 2000).

The location and burden of the worms mostly determines the type and degree of morbidity observed in the host. During the migratory phase, large numbers of larvae may induce host sensitization resulting in asthma, coughing, shortness of breath, fever, skin rash and

eosinophilia (O’Lorcain and Holland 2000; Neva and Brown 1994). After the worms mature in the small intestine, clinical signs and symptoms may include abdominal pain and distension, nausea, vomiting and anorexia. Vitamin A, fat and protein malabsorption also may be present (ibid). Less frequent clinical complications include intestinal obstruction, intestinal perforation, and blockage of the bile duct, acute pancreatitis or appendicitis due to the migration of worms.

Whipworm Infection (*Trichuris trichiura*)

Trichuriasis or whipworm infection is caused by *Trichuris trichiura*. The infection is estimated to affect around 1049 million persons worldwide. Of these, 114 million are children of preschool age and 233 million are of school age (Chan 1997). Humans are the primary host for infections caused by *Trichuris trichiura* but the species has been detected in some non-human primates (Horii and Usui, 1985).

The mature male and female whipworm inhabit the transverse and descending colons where they embed their narrow anterior portion in the host’s columnar epithelium cells to obtain nutrients (Gilgen and Mascie-Taylor 2000). The worms feed on the enterocyte syncytium but may also ingest enterocytes, leucocytes and mucosal fluids if they happen to penetrate below the basal membrane of the same (Pawloski 1984). Their posterior wider portion hangs in the gut lumen allowing the effective emission of eggs. Both sexes live for a span of approximately two years. The adult female can deposit from 3000 – 5000 eggs per day (Katz et al 1989). Fertilized eggs deposited with the feces are undeveloped and must embryonate before they can become infective. The duration of egg maturation is approximately 18–25 days if the ambient temperature is optimal, i.e., 20 – 30°C (Katz et al 1989; Neva & Brown 1994). *Trichuris* eggs are more sensitive to the environment compared to those of *A. lumbricoides* eggs. Temperatures that fall below –9°C or rise above 52°C kill the eggs. Direct sunlight within 15 hours also kills the eggs (Stephenson et al. 2000).

Infection with *Trichuris* occurs via the oral-fecal route caused by the ingestion of infective eggs from contaminated food, hands or water. These then pass through the stomach to the small intestine where they hatch. The larvae penetrate the cells of the small intestine coming

to lie above the lamina propria where they undergo four molts. The immature adults emerge and are passively transported to the large intestine where they mature and embed their thin, whip-like anterior into the columnar cells. The adult whipworm develops within 60–90 days after initial infection (Mehlhorn 1988; Katz et al 1989; Neva and Brown 1994; Stephenson et al 2000).

Trichuriasis signs and symptoms mostly depend on infection burden. Light infections are reported to sometimes cause non-specific responses such as anorexia and urticaria (Stephenson et al 2000). Symptoms associated with moderate infections include epigastric and lower abdominal pain, vomiting, diarrhea, flatulence and weight loss. Heavy infection is associated with mucoid bloody diarrhea, and in very severe cases, rectal prolapse due to frequent straining. In severe infections, the worms may be observed embedded in the edematous rectal mucosa accompanied by moderate eosinophilia. Furthermore, growth stunting associated with decreased collagen synthesis has been reported in children (Neva and Brown 1994; Katz et al 1989; Stephenson et al 2000).

Hookworm Infection

The two major species of hookworm known to infect humans are *Necator americanus* and *Ancylostoma duodenale*. Eggs of both species are identical and morphological identification can be done on the basis of the adult morphology (Katz et al. 1989; Pawloski et al. 1991) or by the study of the larval stages cultured from eggs by the Harada-Mori method (Pawloski et al. 1991). Hookworms are estimated to infect 151 million people worldwide and cause mortality in another 65,000 (Montresor et al. 1998). No animal reservoirs have been identified for hookworms (Katz et al 1994). Mature hookworms typically inhabit the jejunum where they attach to the intestinal mucosa with their ventral teeth (*A. duodenale*) or cutting plates (*N. americanus*) (Katz et al 1989; Neva and Brown 1994). The worms derive their nourishment by feeding on the villous tissue and sucking blood at the point of attachment (Katz et al 1989; Gilgen & Mascie-Taylor 2000). Average blood losses of 0.1– 0.2 ml of blood per day have been reported for *A. duodenale* and 0.02 ml/day for *Nectator americanus* (Roche & Layrisse 1966).

Ancylostoma duodenale is the only hookworm species endemic to Ecuador where the present study was conducted. Thus, the remainder of the description of the hookworm life cycle will focus on this species. Katz and associates (1989) have reported that the adult female passes approximately 28,000 eggs per day. Both the eggs and larvae are very sensitive to exposure to the environment and do not survive for more than a month in the soil (Sorensen et al. 1994). Human infection is acquired through penetration of the skin by the infective third stage filariform larvae (Pawloski et al. 1991; Neva and Brown 1994). Penetration usually occurs in bare feet with skin abrasions, areas on and between the toes, or through a hair follicle (Neva and Brown 1994). The larvae then enter the circulation and are carried to the lungs. During their fourth stage of development, the larvae break out of the alveoli and migrate up the bronchi and trachea to the pharynx where the larvae are involuntarily swallowed and pass down to the small intestine (Katz et al 1989; Crompton 2000). Clinical signs and symptoms of hookworm include pneumonia in heavily infected children during larval migration and epigastric pain and iron deficient anemia during the intestinal phase of the infection (White et al. 1986; Maxwell et al.1987; Crompton 2000).

The Public Health Importance of Intestinal Parasitic Infections

IPI's are considered a public health problem of worldwide importance for reasons of their high prevalence, widespread distribution, and effects on health (Montresor et al. 1998; WHO 1999; Stephenson et al 2000). IPI risk is reported to be elevated in infants and children compared to other age groups. In addition, they are reported to disproportionately suffer from the nutritional (Thein Hlaing 1993; Hadju et al 1997; Stephenson et al 2000), health, and developmental consequences of IPI's (Nokes et al 1992; Callendar et al 1994; Simeon et al 1995; Sakti et al 1999). Morbidity and mortality caused by IPI's is usually more pronounced in children compared to adults due to their higher nutritional requirements and less mature immune systems (Guyatt 2000). Furthermore, the risk for poor clinical outcomes is reported to be increased in those children who were already malnourished prior to becoming infected (Scrimshaw 1994; Stephenson et al 2000 *a*). The chronic malnutrition-IPI cycle which commonly begins during childhood in many developing populations also has been linked with decreased work capacity and productivity in adolescents and adults (Guyatt 2000).

The World Health Organization (WHO) promotes the use of presumptive antihelminth treatment as the foundation of IPI control activities in endemic populations (WHO 1996; Montresor et al. 1998) while still recognizing the need for longer-term economic, social, behavioral and environmental solutions to reduce or eliminate sources of re-infection (WHO 1996; Montresor et al. 1998; WHO 2000). This need was underscored two decades ago by Mata (1982) in his review on the control and prevention of parasitic infections. There, he stated that, “the issue of control has become difficult as one must take into consideration not only the nature and natural history of the parasites but also the biological characteristics and behavior of the humans involved and the environmental circumstances under which they dwell” (pp 871).

Impact of Intestinal Parasitic Infections on Child Growth, Anemia and Psychomotor Development

Intestinal Parasitic Infections and Child Growth

The synergistic cycle of infection, immunity, and malnutrition in children is well documented (Chandra 1999; Chandra 1997). Infants, toddlers and other young children are reported to be most vulnerable to the adverse nutritional effects of IPI's. One reason is that they often suffer from an increased IPI burden associated with a greater exposure to these infectious agents by virtue of unsanitary practices associated with child development (e.g., playing in contaminated dirt and water, sucking on dirty fingers and other objects, etc.). Growing children also have high nutritional requirements. (Scrimshaw 1994). Their less mature immune systems, especially in those aged < 6 years, can reduce their ability to mount strong immune defense to infectious agents. Thus, they are more likely to suffer from the adverse consequences of infection, which can affect energy and nutrient intake, transport, metabolism and excretion (Chandra 1990; Chandra 1999).

Several studies have documented that children infected with roundworm are more likely to suffer from growth stunting compared to those who are not infected. O' Lorcaín & Holland

(2000) reported that some stunting is caused by infection-induced anorexia. The result is decreased caloric and nutrient intake. In addition, infected children have a decreased ability to digest and absorb the nutrients ingested. Likewise, hookworm infection is commonly associated with poor child growth. This appears to be the result of the loss of blood, which contains iron and other essential nutrients needed for growth. Another apparent reason is due to the intestinal inflammation caused by the infection. This lowers the absorption of nutrients and may also aggravate hypoalbuminemia (Warren et al. 1993). Furthermore, a heavy whipworm burden has been observed to negatively impact growth as a result of the frequent dysentery common with this infection (Callender et al. 1994) as well as nutrient loss induced by gastrointestinal bleeding.

A growing body of evidence now exists which has implicated both acute and chronic *Giardia* infection with reduced growth in humans (Farthing et al. 1986; Fraser et al. 2000) and experimental mammals (Astiazaran-Garcia et al. 2000). Moreover, Gupta and colleagues (1982) have demonstrated that Guatemalan children infected with *Giardia* experienced significant increases in height and weight in the six months following anti-parasitic treatment. These results differ from that of Lunn and associates (1999) and Saldiva and associates (1999) who reported being unable to detect any significant differences in recumbent length in *Giardia*-infected versus non-infected infants.

Malnutrition is reported to commonly occur in association with acute *E. histolytica* infection (Mata 1982). The rapid transit time associated with frequent diarrheal episodes is responsible for decreased nutrient absorption. In addition, children who suffer from liver abscess and other forms of extra-intestinal amoebic infections are reported to experience metabolic alterations, which also affect their nutritional status (Petri and Singh 1999). Weigel and associates (1996) have reported finding a significant association between asymptomatic amoebiasis in pregnant Ecuadorian women and reduced fetal growth. However, little is known about whether or not asymptomatic amoebiasis has any significant impact on child growth subsequent to birth. Asymptomatic amoebiasis which occurs after initial acute infection is over, is reported to be highly prevalent in both children and adults residing in developing countries.

Intestinal Parasitic Infections and Anemia

Prior studies have linked intestinal parasitic infections with nutritional anemias. For example, ascariasis is reported to increase the risk for vitamin A (Thein Hlaing 1993) and iron-deficiency anemia (Curtale et al. 1993). Likewise, malabsorption caused by giardiasis has been linked with folate and vitamin B₁₂ deficiencies (Solomons 1982).

The relationship between hookworm infection and iron-deficiency anemia in vulnerable maternal-child groups is well documented (WHO 1996; Brooker et al.1999). However, recent authors have reported that intestinal infection with non-hookworm species may negatively impact iron status (Cooper et al. 1992; Callender et al.1994). For example, Wilson and associates (1999) linked asymptomatic non-hookworm polyparasitosis with significantly decreased mean hemoglobin levels in Colombian schoolboys. Weigel and colleagues (1996) have reported that asymptomatic amoebiasis in pregnant Ecuadorian women was associated with significantly reduced hemoglobin levels and an increased risk for iron-deficiency anemia.

Severe burdens of whipworm infection have been linked with iron deficiency anemia in children (Cooper et al. 1992). Anemia is considered one of the cardinal signs of *Trichuris* dysentery syndrome. Since the whipworm does not ingest erythrocytes, the observed anemia is probably the result of blood loss due to chronic mucosal inflammation and ulceration at the point of insertion (Gilgen and Mascie-Taylor 2000). Neva and Brown (1994) have estimated average blood losses of ~0.005 mls/day in heavily infected persons.

Giardia infection has been associated with reduced serum hemoglobin levels (Curtale et al. 1998) as well as anemia in children (Shubair et al. 2000) although the possible mechanism(s) for this phenomenon remain unknown.

Intestinal Parasitic Infections and Psychomotor Development

Intestinal parasitic infections have been suggested to have a negative influence on child cognitive and psychomotor development (Nokes et al. 1992; Callender et al. 1994; Oberhelman et al. 1998; Sakti et al. 1999). One of the mechanisms by which cognitive impairment is hypothesized to occur relates to the ability of certain parasites, especially hookworm and heavy whipworm infection (Nokes and Bundy 1994), to affect host iron status and cause iron deficiency and anemia (Stoltzfus et al. 1998; Brooker et al. 1999) thus affecting brain iron metabolism (Youdim et al. 1989). Iron deficiency and anemia may also indirectly affect cognitive development through their adverse effects on appetite and reduced energy and nutrient intake (Stephenson et al. 1993; Stephenson et al. 2000b).

The results of a randomized, double-blinded, placebo-controlled clinical study conducted by Nokes and associates (1992) in school children infected with moderate-heavy parasite burdens demonstrated that antiparasite treatment was associated with significant improvement in auditory short-term memory, scanning, and long-term memory retrieval. Callender et al. (1994) also reported seeing significant improvements in locomotor development, height, weight and hemoglobin levels after one year of antihelminthic treatment in children diagnosed with *Trichuris* dysentery syndrome. A recent study conducted by Oberhelman et al. (1998) noted that high ascaris loads were associated with an increased risk for having a suspect developmental test in a large sample of Nicaraguan children. Hadidjaja et al. (1998) also demonstrated significant improvements in cognitive test results in their study of 336 Indonesian children (6-8 years) after treatment for *Ascaris* infection.

The Context of Health and Illness in the Republic of Ecuador: An Overview

The Republic of Ecuador is a developing South American nation situated between the countries of Colombia to the north, and Peru to the south. It is bounded on the west by the Pacific Ocean and on the east by the Peruvian Amazon. Official government statistics report that the prevalence of poverty is high. Seventy seven percent of the rural population lives below the poverty threshold; 38% in extreme poverty (SIISE 2001a; MSP 2001). The country is also

characterized by reduced access to clean drinking water, sanitation, and health care (SIISE 2001 *a-d*; MSP 2001). The access of rural populations to these basic services is even more reduced. For example, only 9% of homes have access to piped-in potable water and only 11 % have adequate sewage disposal (SIISE 2001*b,c*). The unstable public health care system has been made even more so by a continued series of economic crises which reduced funds for this sector from US\$ 17 million/year during the 1995-1996 fiscal year to a only US \$8 million in 1999 (SIISE 2001*d*).

This worsening economic situation and decreased health care spending is reflected in the high mortality and morbidity rates reported for the population especially in the more vulnerable subgroups, i.e., women of reproductive age and children. For example, the maternal mortality rate in Ecuador (15/10,000 births) is elevated compared to most of the rest of Latin America (PAHO 2001). Likewise, iron-deficiency anemia affects from 45-90% of pregnant women and 40-50% of non-pregnant women (Weigel et al. 1994, 1996; Weigel, unpublished data 2001). In addition, one-fifth of young children are vitamin A deficient and half have iron deficiency anemia (MSP 2001).

Around 50% of children < 6 years in Ecuador are reported to suffer from some form of protein-energy malnutrition (PEM), the figure rises to 70% in the rural highlands (MSP 2001*a*). Prior authors have reported that the population prevalence of acute malnutrition or wasting is relatively low affecting only 1-2% of infants and other young children. The most common form of PEM is chronic malnutrition or growth stunting. Studies carried out over the past two decades indicate that from 45%-67% of all infants and preschoolers have stunted growth (Friere et al. 1995; Weigel et al. 1995; Varrea et al. 1997; PAHO 1998; SIISE 2001*e*). A recent survey of 30,000 low-income children showed evidence of retarded linear growth as early as 9-12 months of age (Varrea et al. 1997). The high chronic malnutrition prevalence recorded for Ecuadorian child groups exceeds the cut-off point recommended by the WHO for initiating public health interventions (WHO/CDC 1996).

The prevalence of intestinal parasitic infections and the morbidity and mortality associated with them are reported to be elevated in Ecuador (MSP 2001). The most frequent

IPI's reported for the general population include amoebiasis, giardiasis, ascariasis, and trichuriasis (MSP 2001). Hookworm infection is less common and mostly confined to tropical areas along the Pacific coast and in Amazonia (Sebastian and Santi 2000). Several studies conducted in subtropical and tropical areas of the provinces of Pichincha, Imbaburra and Manabi in the northwest of the country identified infection with *Entamoeba histolytica/dispar* as the most common IPI in adults and children followed by giardiasis, ascariasis and trichuriasis (Weigel 2001, unpublished data; Weigel and Armijos 1996; Weigel et al. 1995). No cases of hookworm cases were found.

CHAPTER 3: OBJECTIVES AND HYPOTHESES

Objectives

The overall objective of the present study was to investigate the prevalence, risk factors and nutritional and developmental consequences of intestinal parasitic infections in rural Ecuadorian children living in the Pichincha Province. The specific objectives of the study were:

1. To determine the prevalence of intestinal parasitic infections in rural children
2. To identify the socio-demographic, gender, ethnic, environmental and other predictors of IPI risk in the children.
3. To determine the association between IPI and the risk for child malnutrition, iron-deficiency anemia and retarded psychomotor development.

Hypotheses

1. Boys and infants/toddlers are more likely to suffer from IPI's and multiple IPI's compared to girls and older children because they are more likely to come into contact with contaminated water, dirt, food, feces, and other sources of infection through play and other behaviors.
2. Children with IPI's have increased risk for stunted growth, iron-deficiency anemia and delayed psychomotor development compared to non-parasitized children,
3. Greater IPI burdens are associated with increased risk for malnutrition and developmental delays.
4. Infants and toddlers suffer from greater nutritional and developmental consequences of IPI due to their greater growth-related nutritional requirements and less mature immune systems.
5. The negative growth, iron status, and developmental consequences of IPI become first evident at around nine months of age subsequent to weaning.

CHAPTER 4: MATERIALS AND METHODS

Study Design and Description of the Study Site

This cross-sectional survey study was conducted in five small rural hamlets (Paraiso de Amigos, Bosque de Oro I, Bosque de Oro II, La Paz, and Cristobal Colon) in the Puerto Quito Canton (county), situated in the northwest corner of Ecuador in the Pichincha Province. The study site was located in an area of dense tropical rainforest approximately 125 miles from the capital city of Quito. The mean elevation of the area is approximately 300 meters above sea level. The mean annual temperature recorded in this region ranges from 24-38°C and the humidity averages around 80%. Motorized access to the study communities was limited and often required 0.5-2 hours of walking on dirt paths.

Subject Selection

The five rural communities that participated in the study were selected by convenience sampling. Within these, all children between the ages of 0–6 years were recruited for participation. However, during the course of the study it became necessary to also recruit older children (6–14 yrs) to augment the sample size due to a lower than expected number of children in the 0-6 age category.

Informed Consent

Permission to conduct the study was obtained from the Institutional Review Board for Research Involving Human Subjects at the Virginia Polytechnic and State University and from the local partner institution's (Fundacion Biocencias) Ethics Committee.

Study Protocol

Training Sessions

Prior to the initiation of the research, an intensive one-week training session was carried out for all the research team members. The predominantly Ecuadorian research team was comprised of three medical doctors, eight medical students, two graduate students and two laboratory technicians apart from the main researchers. Training and standardization sessions for the study included: guidance in the administration of the Denver Developmental Screening Test anthropometric measurements, sensitivity to cultural/traditional norms in the administration of the structured questionnaire, and practice in the collection of capillary blood samples with Hemocue photometer. Analyses of the fecal samples were performed by trained laboratory technicians.

Pilot Study

A one-week pilot study was conducted in Puerto Quito, a small town located in the study area. This town was selected because it served as a central point for many of the surrounding villages and thus had a steady stream of inhabitants from the neighboring villages. Approximately 30 children between the ages of 9 – 84 months were involved in the pilot study.

Main Study

Before the initiation of the study in each community, meetings with the school principals, local community leaders, and the communities themselves were conducted for the purpose of discussing the purpose of the project and to ask for permission to carry it out. The study data were collected during house-to-house visits to the homes of the subjects in the five study communities. In some cases, however, when such visits were not feasible parents/guardians were met at the local school. The purpose of the study was explained to all parents/guardians of prospective subjects. They were also informed about the other basic elements of the study including procedures, possible risks and benefits, confidentiality and the freedom to withdraw without penalty. Those parents/guardians that gave permission for their children to participate

were required to sign a written consent form, which repeated the information given during the verbal consent procedure (Appendix 1).

The study data were obtained using a structured questionnaire, with open and close-ended items, a developmental screening examination, anthropometry, and laboratory analysis of blood and fecal samples. After signing the consent form, each parent/guardian was given a brief overview about the format and length of the interview. Parents and guardians were interviewed with the questionnaire (Appendix 2), which gathered information on household sociodemographic indicators, the child's medical history, breastfeeding and weaning practices. The Denver Developmental Screening Test was administered to children aged ≤ 60 months. The anthropometric measurements of each child were then taken and these included the height, weight, mid-upper arm circumference including the head circumference for children under 24 mos. After this approximately four to five drops of blood were donated for the serum hemoglobin assessment. Finally, the children's parents/ guardians were given plastic containers with well-fitted lids for the collection of the three daily fecal samples. These samples were collected each morning.

Child subjects identified with a pathogenic intestinal parasitic infection received antiparasitic treatment without cost. In addition, all children involved in the study were given a vitamin A supplement and those diagnosed with iron-deficiency anemia were given iron tablets. All families in the study received nutrition counseling performed by trained nutritionists.

Data Collection Instruments

Questionnaire

The open and closed-ended questionnaire collected information from the parents/guardians of subjects regarding child and household sociodemographic, housing, water and sanitation characteristics. These items included subject age, sex, and ethnicity, parental marital status, family size and composition, house construction, water, sanitation, and garbage disposal, characteristics and the presence versus absence, type and density of domestic animals living in and around the home.

Anthropometry

The anthropometric data collected included weight, height or recumbent length (children < 24 months), mid-upper arm and thigh circumference, and head circumference (children < 24 months). 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 0.1cm using a portable stadiometer. For children who were < 24 months of age, recumbent length was measured to the nearest 0.1cm with an infant measuring board having a fixed head board and a movable footboard. Measurements of the mid-upper arm circumference and mid-thigh circumferences were measured to the nearest 0.1 mm using a semi-flexible insertion tape measure (Ross Labs, USA), as was head circumference in children <24 months of age. All measurements were made after removal of shoes, socks, hair adornments, soiled diapers or any other heavy items, which might interfere with the accuracy of the measurements.

Subject weight-for-age, weight-for-height, and height-for-age measurements were calculated using the EPINUT program (Epi info, version 6). This compared subject measurements to an international NCHS growth reference curves (Dibley et al. 1987). The results were expressed in standard deviation units or Z-scores. The cut-off points used to identify malnourished children were: < - 2 standard deviations from the reference median. The < - 2 SD cut-off is equivalent to the reference population in which 2.3% fall below this point. Children whose weight-for-height were < - 2 SD's were classified as having acute malnutrition, those with a height-for-age < - 2 SD's with linear growth retardation (i.e., stunted growth or chronic malnutrition) and those whose weight-for-age was < - 2 SD's with global malnutrition.

Iron Status

Subject iron status was determined using the HemoCue B-Hemoglobin photometer *HemoCue*® (Hemocue AB Angelholm, Sweden). The instrument employs the cyanmethemoglobin (HiCN) method to determine total hemoglobin concentration in capillary blood samples. Sterile disposable surgical lancets were used to obtain approximately 0.2 ml of

capillary blood from a finger prick of the middle or ring finger (children ≥ 9 mos.) or heel pricks (children < 9 mos.). Care was taken to avoid “milking” the subjects during the blood draw. The first 2-3 drops of drawn capillary blood were wiped away and the subsequent drop collected in a disposable microcuvette, which contained the reagent in a dry form. The blood-filled microcuvette was analyzed immediately. The function of the photometer was checked daily using the supplied control cuvette that was standardized to give a reading of 12.8 ± 0.3 g/dl. The cutoff points used to identify iron-deficiency anemia were based on CDC recommended values adjusted for child age and sex (MMWR 1998).

Denver Developmental Screening Test

The psychomotor development of children between the ages of 1 month and 6 years was assessed using a Spanish version of the Denver Developmental Screening Test (DDST). The DDST, which is used routinely by the Ecuadorian Ministry of Public Health (MSP 1991), is an internationally recognized developmental test. The DDST tests subjects on four component scales: social, language, gross motor and fine motor. Items on the test for each section of the DDST are age-specific and designed to correspond to a broad range of heterogeneous developmental skills, which the child should normally have attained, by a certain chronological age (Frankenburg et al, 1992) i.e. the ability to hop or stand on one leg (gross motor) or the ability to stack a number of blocks (fine motor).

Items on the test were scored as pass, risk, fail, no opportunity to respond, or refusal to perform. The results were interpreted as follows: (1) Pass, no failures on age-specific component(s), (2) One failure - risk on that component, and (3) More than one failure - retardation on that component.

Identification of Intestinal Parasitic Infections

The subjects contributed three consecutive stool samples for the intestinal parasite analysis. Experienced, certified laboratory technicians analyzed these on the day of collection in the field laboratory of the Biociencias Foundation Puerto Quito community clinic. After gross

examination of the sample characteristics, a direct wet fecal smear was prepared by emulsification of 2mg of the feces on a glass slide in one drop of Lugol's iodine. The sample was covered with a coverslip and systematically observed at low and high magnifications using a light microscope in order to identify any mature parasites, cysts or eggs present.

Data Analysis

The descriptive data were analyzed with standard techniques such as means with standard deviations, frequency counts and percentages. Simple bivariate analysis using 2x2 contingency table analyses with X^2 were utilized to investigate differences between proportions. Factors identified as significant ($P < 0.05$) in these analyses were subsequently analyzed 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 the tables with their respective 95% confidence intervals. Student's t-test and one-way analysis of variance (ANOVA) were used to test mean differences. The latter controlled for the influence of confounders where applicable.

CHAPTER 5: RESULTS

Subject Characteristics

Sociodemographic Characteristics

The sociodemographic characteristics of the child subjects and their households are displayed in Table 1. As indicated, the mean age of the 244 children living in the five rural tropical communities studied was 63.4 ± 40.3 months; 80% were under the age of seven years. Female children comprised slightly over half (54.1%) of the sample and 88% were of mestizo ethnicity (i.e., mixed Spanish-Quechua descent). The size of the households in which the children resided averaged 6.5 ± 2.1 members. Both parents were present in most households and many children also had grandparents or other adult relatives living with them.

As Table 1 also indicates, a majority of the children's parents were either legally married or lived together in stable common law unions. Most subject mothers (83.6%) reported that their primary occupation was housewife. In contrast, most of their fathers (79.5%) indicated that they were engaged in subsistence farming and/or in the production of small cash crops (e.g., palm oil, coffee, cacao, black pepper, passion fruit, bananas). A majority of the children's parents had completed at least three years of formal schooling. Close to 60% of their homes were owned by their families (56.7%); another one-quarter occupied homes in exchange for working as caretakers of the property on which they and their families lived, and the rest rented or had other arrangements.

Table 1. Subject & Household Sociodemographic Characteristics

Child Characteristics	F (%) or x ± SD
Place of Residence	
La Paz	60 (24.6)
Bosque de Oro 1	66 (27.0)
Bosque de Oro 2	10 (4.1)
Paraiso de Amigos	29 (11.9)
Cristobal Colon	79 (32.4)
Child Ethnicity (% mestizo)	181 (88.3)
Age (months)	63.4 ± 40.3
Sex (% female)	132 (54.1)
Household Characteristics	
Maternal Occupation: (% Housewife)	168 (83.6)
Paternal Occupation: (% Agriculturalist)	163 (79.5)
Maternal education (yrs completed)	4.3 ± 2.6
Paternal Education (yrs completed)	4.3 ± 2.6
Maternal marital status:	
Legally married	59 (30.1)
Common law marriage	120 (61.2)
Other	17 (8.7)
Paternal marital status:	
Legally married	62 (32.6)
Common law union	116 (61.1)
Other	12 (6.3)
Household Size	6.5 ± 2.1
Household Composition	
Parents	1.9 ± 0.4
Other adults	2.6 ± 1.0
Children < 13 yrs.	3.1 ± 1.5
Adolescents 13-18 yrs	0.8 ± 1.0
Type of Home Ownership	
Own	114 (56.7)
Rent	19 (9.5)
Borrowed	11 (5.5)
Caretaker	51 (25.4)
Other	6 (3.0)

Home Construction, Water, and Sanitary Characteristics

Table 2 describes the home construction, water, and sanitary characteristics of subject households. As shown, over 60% of the homes were constructed from wood or other plant materials. Only a third had access to electricity for home illumination. Most households reported that they used outside pit latrines or the open ground as their home sanitary facilities. Close to 60% of households said that they disposed of wastewater directly on the open ground; fewer than 2% said their homes were connected to a municipal sewage system. A majority said that they discarded their household garbage on the open ground. These surface dumps were described as located an average of only 40.2 ± 16.3 meters from their homes.

Table 2 also shows that the principal source of water for many houses was from the Puerto Quito river, followed by home wells and surface springs. Even though 79% of households reported treating their drinking water, only 31.3% said that they used treated water when preparing food for their families. Fewer than 10% said that they did not use treated water for bathing. The major form of water treatment was by boiling although some families also said that they used chlorine. However, fewer than 4% of the households who reported that they boiled drinking water said that they allowed it to come to a rolling boil for at least five minutes as recommended by WHO. Approximately 82% of the interviewed parents/guardians reported consistent hand washing after bathroom use. Most subject homes (95%) were characterized by the presence of free roaming domestic animals in and around the house (22.7 ± 22.3 animals). These animals included poultry, pigs, dogs, cats, horses, and guinea pigs (used as food animals).

Table 2. Home Environmental and Sanitary Characteristics

Characteristic	F (%) / $\bar{x} \pm SD$
Type of Home Illumination:	
Electricity only	68 (33.8)
Kerosene/gasoline lamps only	45 (22.4)
Candles only	44 (21.9)
Mixed types	35 (17.4)
Other	9 (4.5)
Home Construction	
Cement block	36 (18.0)
Wood/cane/panbill	125 (62.5)
Mixed	28 (14.0)
Other	11 (5.5)
Home Water Source	
River only	69 (34.8)
Open well only	46 (23.2)
Spring only	41 (20.7)
Municipal piped-in only	17 (8.6)
Municipal & other sources	12 (6.1)
Well & other non-municipal sources	8 (4.0)
River & other non-municipal sources	5 (2.5)
Home Sanitary Facilities	
Inside toilet	13 (6.4)
Outside toilet	19 (9.4)
Outside latrine	115 (56.7)
Open ground	50 (24.6)
Home Sewage Elimination	
Open ground	115 (58.1)
Septic tank	47 (23.7)
River or ravine	28 (14.1)
Municipal sewage system	3 (1.5)
Mixed	5 (2.5)
Home Garbage Disposal	
Burn	55 (27.4)
Open ground	57 (28.4)
River or ravine	15 (7.5)
Bury	32 (15.9)
Municipal	19 (9.5)
Mixed	23 (11.4)
Mean distance from house to site of garbage disposal (meters)	40.2 \pm 16.3
Household Reports Treating Drinking Water (% yes)	158 (79.0)
Household Reports Treating Bath Water (% yes)	15 (7.7)
Household Reports Treating Water Used for Washing Foods Prior to Consumption (% yes)	62 (31.3)

Prevalence of Intestinal Parasitic Infections

The results of the stool sample analyses revealed that the most common non-pathogenic parasites detected in subject stool samples were *Entamoeba coli* (22.6%), *Endolimax nana* (7.7%) and *Iodamoeba bütschelii* (2.1%). Ninety percent of the child subjects were infected with one or more pathogenic or presumed to be pathogenic intestinal parasite. Fifty-one percent had helminthic infections, 37.6% had protozoal infections and 21.4% were infected with both. The most common intestinal parasite detected was *Ascaris lumbricoides* (39.7%), followed by *Giardia intestinalis* (25.2%), *Trichuris trichiura* (19.7%), *Entamoeba histolytica/dispar* (18.5%), *Ancylostoma duodenale* (1.7%) and *Blastocystis hominis* (13.3%). The distribution of light (+), medium (++), or heavy (+++) burdens of parasitic infections were:

<i>Ascaris lumbricoides</i>	+ (51.1%), ++ (33.1%), +++ (15.8%)
<i>Giardia intestinalis</i>	+ (64.0%), ++ (35.6%), +++ (0%)
<i>Trichuris trichiura</i>	+ (74.1%), ++ (20.4%), +++ (5.6%)
<i>Entamoeba histolytica/dispar</i>	+ (56.7%), ++ (31.7%), +++ (11.7%)
<i>Ancylostoma duodenale</i>	+ (75%), ++ (25%), +++ (0%)
<i>Blastocystis hominis</i>	+ (89.2%), ++ (8.1%), +++ (2.7%)

Controversy remains regarding whether *B. hominis* should be classified as a commensal or a human pathogen. For the purpose of this study, it was decided to treat it as a potential pathogen. The rationale for this is based on the results of several recent studies which have linked the protozoal parasite with diarrhea and other gastrointestinal complaints in the absence of other pathogenic IPI's and their recommendations that the infection requires treatment (Brites et al. 1997; Nimri 1993).

Nutritional Status Indicators

Iron Status.

The mean serum hemoglobin concentration, as measured with the HemoCue analyzer, was 12.05 ± 1.5 g/dL. Over one quarter (26.4%) of the children were identified as having iron deficiency anemia and another 3.9 % were diagnosed as borderline for the condition, based on the CDC criteria (MMWR 1998).

Child Growth.

The continuous and categorical anthropometric indicators of child nutritional status are shown in Table 3. As indicated, close to 40% of the child subjects were classified with chronic malnutrition or growth stunting, based on a low height-for-age (<-2 SD). Global malnutrition or low weight-for-age (<-2 SD) was identified in 21.5% of the subjects. Acute malnutrition or wasting (low weight for height, <-2 SD) was observed in 4.1%. No significant sex, or ethnic differences were observed in proportion to children identified with chronic, acute nor global malnutrition. None of the children were observed to have edema, which could have interfered with the weight measurements.

Psychomotor Development

The Denver Developmental Screening Test was administered to the 132 subjects who were aged ≤ 6 years. The results found that only eleven children (9.4%) failed the overall examination. A total of eight (6.7%) failed the gross motor portion, eleven (9.5%) the fine motor portion, fifteen (13.2%) the language portion and four (3.4%), the social development portion of the exam.

Table 3. Child Nutrition Indicators

Indicators	$\bar{x} \pm SD/ f (\%)$
Anthropometric Indicators	
Weight (kg)	16.7 \pm 7.6
Height (cm)	100.9 \pm 20.2
Head circumference (< 24 mos) (cm)	44.7 \pm 2.3
Mid-upper arm circumference (cm)	17.1 \pm 2.1
Thigh circumference (cm)	29.7 \pm 7.0
Malnutrition	
Chronic malnutrition(stunting)	89 (37.4)
Acute malnutrition (wasting)	9 (4.1)
Global malnutrition	242 (21.5)
Iron Indicators:	
Serum hemoglobin (g/dL)	12.05 \pm 1.5
Iron-deficiency anemia	55 (26.8)
Borderline for iron-deficiency anemia	8 (3.9)

Factors Associated with the Risk for Intestinal Parasitic Infection.

Simple bivariate analysis was used to determine which factors were associated with the risk for infection with intestinal parasites. The results of these are presented as crude odds ratios with 95% confidence intervals in (Tables 4-8). The tables also show the results of the multiple logistic regression analyses, which identified which of the indicators made significant independent contributions to the risk for intestinal parasitic infection.

***Giardia intestinalis* Infection**

Factors identified as associated with the risk for *G. intestinalis* infection in the bivariate analyses are displayed in Table 4. Infection with *Giardia* was observed to be more common among children who lived in the hamlet of Paraiso de Amigos whose households were situated \leq 100 meters from their closest neighbor, and who had a high density (≥ 20) of domestic animals living in or around their homes. In contrast, children whose parents were legally married had reduced risk for giardiasis. No significant associations were identified between *Giardia* and child gender, age, ethnicity, education of parents, home ownership, household water source, home wastewater elimination, garbage disposal, drinking water treatment nor the other factors measured.

In the multiple logistic regression analysis, residence in Paraiso de Amigos and of domestic animal density retained their positive association with risk of giardiasis (Table 4). Likewise, parental marital status was still found to be associated with reduced giardiasis risk. However, when the influence of the confounders was taken into account, the contribution of home proximity to nearest neighbor was no longer evident and was dropped from the final model.

Table 4. Factors Associated with the Risk for *Giardia intestinalis* Infection

Factor	f (%)	OR (95% C.I.)	P	AOR (95% C.I.)	P
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 meters	39 (33.6)	2.36 (1.14-4.92)	0.03	1.26 (0.54-2.91)	NS
> 100 meters	12 (17.6)	1.00		1.00	

***Entamoeba histolytica/dispar* Infection**

The results of the simple bivariate analyses of factors associated with risk of *E. histolytica/dispar* infection, displayed in Table 5, showed a high risk of infection among child subjects who resided in Paraiso de Amigos, whose families had a high density of child animals living in or around the house, used rivers as their household source of water and whose fathers were engaged in agricultural work. In contrast, a reduced risk of infection was observed in younger children (< 84 months) and those whose families reported that they adequately disposed of their household garbage (municipal pickup/burning/burying vs. open ground/ravines/river). No significant associations were identified between gender, ethnicity, treatment of water source, or type of toilet facility, other indicators and amoebiasis risk.

The multiple logistic regression analysis results indicated that animal density, subject age and adequate garbage disposal maintained their previously identified significant associations. However, the contributions of water source, paternal occupation and place of residence identified previously were no longer apparent and were eliminated from the model.

***Blastocystis hominis* Infection**

Table 6 shows which factors were found to be significantly associated with the risk for *Blastocystis hominis* infection. As indicated, the simple bivariate analysis results revealed that children who lived in the hamlet of La Paz, whose homes were characterized by high animal density and who used only paraffin candles for lighting their homes were at increased risk for infection. In contrast reduced infection risk was associated with home ownership, maternal housewife occupation, adult hand washing practices, younger subject age (\leq 108 months) and wooden home construction. The multiple logistic regression results, shown in Table 6, indicated

Table 5. Factors Associated with the Risk for *Entamoeba Histolytica/Dispar* Infection

Factor	f (%)	OR (95% C.I.)	P	AOR (95% C.I.)	P
Residence					
Paraiso de Amigos	24 (40.7)	5.59 (2.61-12.04)	0.000001	1.68 (0.20-15.13)	NS
Other	19 (10.9)	1.00		1.00	
Subject age					
≤ 84 mos	24 (13.5)	0.08 (0.04-0.18)	0.000001	0.49 (0.22-1.1)	NS
> 84 mos.	19 (35.2)			1.00	
Paternal Occupation					
Agriculturalist	37 (23.9)	4.10 (1.02-18.00)	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*	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	

* burn, bury, municipal pickup

Table 6. Factors Associated with the Risk for *Blastocystis hominis* Infection

Factor	f (%)	OR (95% C.I.)	P	AOR (95% C.I.)	P
Residence					
La Paz	24 (41.3)	17.0 (6.8-42.5)	0.0001	5.44 (1.9-15.3)	0.001
Other	7 (0.04)	1.00		1.00	
Home ownership					
Own home	7 (7.0)	0.17 (0.06-0.46)	0.0001	0.20 (0.07-0.57)	0.003
Other	24 (28.6)	1.00		1.00	
No. of domestic animals in/around home					
> 20	24 (33.8)	7.73 (3.12-19.2)	0.0001	5.1 (1.8-14.4)	0.002
≤ 20	7 (6.2)	1.00		1.00	
Maternal Primary Occupation					
Housewife	21 (13.1)	-----	0.033*		NS
Other	9 (29.0)				
Handwashing behavior of adult caretaker after bathroom use					
Always washes	19 (12.3)	0.29 (0.12-0.69)	0.008	0.68 (0.20-2.15)	NS
Does not always Wash	11 (33.3)	1.00		1.00	
Subject Age					
≤ 108 mos	20 (10.2)	0.25 (0.10-0.63)	0.0017	0.67 (0.55-1.50)	NS
> 108 mos.	11 (31.4)	1.00		1.00	
Home illumination					
Use candles only	15 (35.7)	4.51 (1.85-11.1)	0.0004	1.70 (0.56-5.24)	NS
Other	16 (11.0)	1.00		1.00	
Home construction					
Wood only	12 (1.0)	0.21 (0.09-0.46)	0.00003	0.45 (0.20-1.29)	NS
Other	28 (35.0)	1.00		1.00	

* Fisher's exact test *P*-value

that the significant associations previously identified during the bivariate analyses were retained with the exception of subject age, maternal occupation and home construction type.

***Trichuris trichiura* Infection**

Table 7 shows that residence in La Paz and a low level of maternal education (≤ 3 years) were both associated with an increased risk for trichuriasis. Younger subject age (≤ 60 months) was associated with a decreased risk. No significant associations were observed between *Trichuris* infection and subject gender, household water source, toilet facilities, garbage disposal and the other factors measured. The multiple logistic regression analysis determined which factors made significant independent contributions to the risk infection with *Trichuris*. Although subject age and maternal education retained their previously identified contributions to the risk for trichuriasis, that of residence in La Paz was no longer evident and was dropped from the model.

***Ascaris lumbricoides* Infection**

The bivariate analysis with Students' t-test revealed that the mothers of the children identified with ascariasis were less well educated than 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.

***Ancylostoma duodenale* (Hookworm) Infection**

No significant associations were identified between any of the factors measured in the study and the risk for hookworm infection, ostensibly because of the reduced number of cases detected, i.e., only four which did not permit sufficient contrast in the statistical analyses.

Table 7. Factors Associated with the Risk for *Trichuris trichiura* Infection

Factor	f (%)	OR (95% C.I.)	P	AOR (95% C.I.)	P
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 mos	13 (10.9)	0.31 (0.15-0.67)	0.0016	0.36 (0.16-0.81)	0.013
> 60 mos.	32 (28.1)	1.00		1.00	
Maternal education					
≤ 3 yrs.	26 (32.5)	2.62 (1.29-5.32)	0.011	1.96 (0.93-4.13)	NS
> 3 yrs.	16 (15.5)	1.00		1.00	

Intestinal Parasitic Infection and Nutritional Status

Iron Status

As displayed in Table 8, the results of the analysis of variance showed that children with *G. intestinalis* infection exhibited significantly reduced mean serum hemoglobin levels ($p=0.023$) compared to those without evidence of the parasite. The analysis adjusted for the influence of chronological age and sex on hemoglobin levels. However, despite having lower average hemoglobin levels, children who suffered from giardiasis were not at significantly increased risk for iron-deficiency anemia compared to their non-infected counterparts. In addition, the association of *Giardia* with reduced serum hemoglobin was not correlated with age and no significant association of IPI and reduced serum hemoglobin and/or IDA was observed in children under 10 months of age.

No significant associations were detected between infection with *Entamoeba histolytica/dispar*, *Ascaris lumbricoides*, *Trichuris trichiura*, or *Ancylostoma duodenale* and mean serum hemoglobin levels (Table 8). Likewise, infection with these was not associated with the risk for iron-deficiency anemia. Furthermore, for all the parasites a higher burden of infection was not associated with decreased hemoglobin levels nor did it appear to affect the risk for anemia.

Child Growth

Table 9 shows that children with intestinal parasitic infections were twice as likely as those without infection to show evidence of reduced linear growth or growth stunting. Likewise, children infected with *G. intestinalis* had a risk for stunted growth that was increased by 2.2 times compared to those in whom the parasite was not detected. None of the other parasitic infections was found to be significantly associated with stunted growth. In addition, 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 9).

Table 8. Association Between Intestinal Parasitic Infection and Child Iron Status

Intestinal Parasite Species	Serum Hemoglobin (g/dL)*		Iron-Deficiency Anemia**	
	X ± S.D.	F-	f (%)	OR (95% CI)
<i>G. intestinalis</i> present (51)	11.8 ± 1.5***	5.2	15 (29.4)	1.30 (0.6-2.8)
<i>G. intestinalis</i> absent (144)	12.2 ± 1.4		35 (24.3)	1.00
<i>E. histolytica/dispar</i> present (42)	12.4 ± 1.5	< 1.0	11 (26.2)	1.00 (0.47-2.2)
<i>E. histolytica/dispar</i> absent (152)	12.0 ± 1.3		39 (25.7)	
<i>A. lumbricoides</i> present (84)	12.3 ± 1.3	1.7	16 (19.0)	0.53 (0.27-1.1)
<i>A. lumbricoides</i> absent (111)	12.0 ± 1.5		34 (30.6)	1.00
<i>T. trichiura</i> present (43)	12.3 ± 1.1	< 1.0	10 (23.3)	0.85 (0.38-1.9)
<i>T. trichiura</i> absent (152)	12.1 ± 1.5		40 (26.3)	1.00
<i>E. nana</i> present (18)	12.5 ± 1.3	< 1.0	4 (22.2)	0.81 (0.25-2.6)
<i>E. nana</i> absent (176)	12.1 ± 1.4		46 (26.1)	1.00
<i>A. duodenale</i> present (4)	11.8 ± 0.6	< 1.0	1 (25.0)	0.95 (1.0-9.3)
<i>A. duodenale</i> absent (192)	12.1 ± 1.5		50 (26.0)	1.00

* controlled for subject age and sex

** cut-off points based on CDC (2000) criteria according to age and sex

*** p = 0.023

Table 9. Association Between Intestinal Parasitic Infection and Child Growth Status

Intestinal Parasite Species	HAZ		WHZ		WAZ	
	F (%)	OR (95% CI)	F (%)	OR (95% CI)	F (%)	OR (95% CI)
Any parasitic infection	66 (43.1)	2.10 (1.10-3.99)*	7 (5.0)	1.80(0.37-8.92)	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
<i>G. intestinalis</i> 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)
<i>G. intestinalis</i> absent	56 (33.1)	1.00	8 (5.1)	1.00	34 (19.8)	1.00
<i>E. histolytica/dispar</i> present	17 (41.5)	1.22 (0.61-2.43)	0 (0.0)	-----	9 (21.4)	1.00 (0.44-2.27)
<i>E. histolytica/dispar</i> absent	68 (36.8)	1.00	9 (5.1)		40 (21.4)	
<i>A. lumbricoides</i> 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)
<i>A. lumbricoides</i> absent	47 (34.6)	1.00	6 (4.7)	1.00	25 (18.1)	1.00
<i>T. trichiura</i> 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)
<i>T. trichiura</i> absent	67 (37.0)	1.00	8 (4.8)	1.00	38 (20.6)	1.00
<i>E. nana</i> present	5 (29.4)	0.66 (0.22-1.94)	2 (13.3)	4.1 (0.7-21.7)	4 (22.2)	1.10 (0.33-3.36)
<i>E. nana</i> absent	81 (38.8)	1.00	7 (3.6)	1.00	45 (21.3)	1.00
<i>A. duodenale</i> present	1 (25.0)	0.55 (0.60-5.30)	0 (0.0)	-----	1 (25.0)	1.24 (0.13-1.22)
<i>A. duodenale</i> absent	85 (37.9)	1.00	9 (4.4)		48 (21.1)	1.00

* p= 0.023 ** p= 0.01

Moreover, differences in the level of infection (+, ++, +++) were not associated with the risk for growth stunting, wasting or global malnutrition.

Similarly, with regards to age no significant association of IPI with stunting was observed for children under ten months of age. The results found that only one child out of the nine children less than 10 months (11.1%) who were positive for IPI was stunted. In addition, no association with stunting was also observed for *Giardia* in isolation or in combination with any other IPI for children under 10 months.

Intestinal Parasitic Infection and Psychomotor Development

The results of the simple biivariate analyses were unable to detect any significant associations between any of the intestinal parasitic infections identified and the risk for failing the Denver Developmental Screening exam. However, it is unclear whether these results were due to the real lack of an association or a lack of contrast caused by only a few children failing the exam, which did not permit meaningful statistical analysis.

CHAPTER 6: DISCUSSION

DISCUSSION

The survey-design study investigated the prevalence, risk factors, and the nutritional and development consequences of intestinal parasitic infections in rural Ecuadorian children. It was hypothesized *a priori* that boys and young children would be at increased risk for IPI's and polyparasitosis compared to girls and older children since their behavior would make them more likely to come into contact with contaminated water, dirt, food, feces, and other sources of infection. It also was hypothesized that children with IPI's, especially younger ones, would be at increased risk for stunted growth, iron-deficiency anemia and delayed psychomotor development compared to non-parasitized children. Greater IPI burdens were postulated to be associated with increased risk for malnutrition and developmental delays. Infants and toddlers were predicted to suffer from greater nutritional and developmental consequences of IPI due to their greater growth-related nutritional requirements. Finally, it was hypothesized that the negative growth, iron status, and developmental consequences, assumed to be associated with IPI's, would first become evident at around nine months of age after weaning had occurred.

IPI Prevalence

The results revealed that the prevalence of IPI was elevated in the study population. Ninety percent of the children were identified as having one or more of the following parasites present in their stool samples: *Ascaris lumbricoides*, *Trichuris trichiura*, *Giardia intestinalis*, *Anclostoma duodenale*, *Entamoeba histolytica/dispar* or *Blastocystis hominis*. These results are consistent with those of previous studies conducted in nearby subtropical areas, which reported IPI population prevalence in the 92-96% range (Weigel et al. 1992; Weigel et. al, 1995). Similar to what has been noted for other tropical groups living in the Amazon (Benefice and Barral 1991; Sebastian and Santi 2000) and Pacific Coast (Weigel, unpublished data, 2001), ascariasis was found to be the most prevalent IPI identified. Several cases of hookworm infection were also found. The observed pattern is different from that reported for rural subtropical (Weigel et al. 1995; Weigel et al., 1996) and highland groups (Ortega 2000) living at higher altitudes in the

same province. These authors reported that by *E. histolytica/dispar* infection was the most prevalent IPI followed by *Giardia*, *Ascaris* and *Trichuris* infections. They also reported being unable to identify hookworm infection in any of the child or adult subjects they examined.

It is noted however, that even though a high prevalence of intestinal parasitic infections (90%) was observed for this population the distribution of the IPI could have been underreported due to the reduced sensitivity (50 – 70%) of the parasitological technique employed (Watt et al. 1986; Nunez-Fernandez et al 1991; Abo-Shehada 1992; Garcia 1999). The reduced sensitivity of this technique was however, largely combated by the analysis of 3 separate fecal samples - a technique widely observed to increase the sensitivity of any coproparasitological analysis (Garcia 1999). As noted by Garcia (1999) additional examinations of fecal samples increase the yield of protozoa by 22.7% for *E. histolytica* and 11.3% for *G. intestinalis*.

No observation of *Cryptosporidium parvum* an IPI observed in children was observed in this study. This was attributed to the coproparasitological technique employed as the oocysts of this parasite are difficult to detect in the absence of special staining techniques such as the modified acid fast, Kinyoun's and Giemsa methods or with the use of immunoassay methods both of which were not employed in this study (Garcia 1999).

Similar to the current study, only a minority of rural homes in Ecuador have potable water supply (9%) or adequate sewage disposal (11%) (SIISE, 2001b-c). The high prevalence of poor water and inadequate sanitation, e.g. unlined pit latrines (56.7%) sited close to unprotected surface water as well as the common practice of open field defecation (24.6%) could also have accounted for the high prevalence of *A. lumbricoides*. These are known to be significant routes for the fecal-oral transmission of *A. lumbricoides* and *T. trichiura*. This is especially true for *A. lumbricoides* with its highly resilient ova (O'Lorcain and Holland 2000). Likewise, in the study area, almost all household water comes from the local river, springs and open wells which are unprotected from the droppings of wild and domestic animals. Furthermore, household wastewater is frequently discharged into the local river, which further promotes the transmission of pathogenic intestinal protozoa and helminthes downriver.

Iron-Deficiency Anemia

Anemia caused by iron deficiency is reported to be the most frequent nutritional problem of public health importance in Ecuador. Previous authors have reported that the condition affects 40-90% of all pregnant women (Friere 1989; Weigel et al. 1992; Weigel et al.1996; Gil Ramos 1998) and from 12-50% of children (MSP 2001; Estrella et al. 1996). The results of the current study concur since over one-quarter of the children in the study were diagnosed with iron-deficiency anemia according to their blood hemoglobin values. This prevalence was doubled compared to figures reported for rural children living in a subtropical area of the same province (Weigel et al. 1995).

The study identified the presence of a significant negative association between *Giardia* infection and mean blood hemoglobin levels. The proportion identified with iron-deficiency anemia also was increased from 24% in non-infected children to 29% in infected children although this difference did not achieve statistical significance. The negative impact of *Giardia* infection on child iron status observed in the present work is consistent with that noted in recent studies of children from other developing countries. Specifically, Curtale and associates (1998) have linked *Giardia* infection with significantly reduced mean hemoglobin levels in Egyptian children. Likewise, Shubair and associates (2000) found that schoolchildren in the Gaza infected with *Giardia* had significantly reduced hemoglobin levels and were more likely to present with anemia caused by iron deficiency than their non-infected counterparts.

The precise mechanism by which *Giardia* causes negative changes in child iron status is still under investigation. However, it does not seem unreasonable to suggest that malabsorption of the mineral may be involved. Specifically, giardiasis has been linked with the reduced uptake of vitamins B₁₂ and folate (Solomons, 1982; Egger et al.1990) due to malabsorption (Solomons, 1982; Eckmann and Gillin 2001). The parasite also is known to selectively colonize the duodenum and upper jejunum (Solomons, 1982), the major site at which iron absorption occurs (Despopoulos and Silbernagl 1991; Berdanier 1995; Brody 1999).

Physical Growth

The most prevalent form of protein-energy malnutrition identified in the child study subjects was chronic malnutrition or growth stunting. This affected about four of every ten children examined. In contrast, acute malnutrition or wasting was found in fewer than 5%. These figures are in close agreement with what has been reported for studies in similarly aged children in nearby rural counties of the same province (Weigel et al. 1995; Moreano et al. 1994).

As expected, children infected with intestinal parasites were at double the risk for chronic malnutrition, i.e., stunted growth. This finding is consistent with recently published studies, which linked IPI with significantly reduced linear growth in Brazilian (Saldiva et al. 1999), Colombian (Wilson et al. 1999) and Nicaraguan children (Oberhelman et al. 1998). Most of the increased risk in our Ecuadorian sample, however, appeared to be due to the strong contribution of *Giardia* infection. Infection with this species also doubled the risk for stunted growth. This is seen in the odds ratio and adjusted odds ratios with their 95% CI's which were almost identical to that of the combined IPI.

Our results concur with those published by several previous authors who implicated chronic as well as acute giardiasis with significant reductions in the rates of linear growth in Israeli (Fraser et al. 2000) and Guatemalan children (Farthing et al. 1986) and in experimentally infected gerbils (Astiazaran-Garcia et al. 2000). Moreover, Gupta and colleagues (1982) have demonstrated that Guatemalan children infected with *Giardia* experienced significant increases in height and weight in the six months following anti-parasitic treatment.

On the other hand, the results differ from Lunn and associates (1999) who reported being unable to detect any significant differences in recumbent length in *Giardia*-infected versus non-infected infants living in rural Gambia. However, their study may not be directly comparable since they studied 2-8 month old infants whose immunocompetence, growth patterns and nutritional requirements were probably quite different from older children. More importantly, all of the children in their study were still being fully or partially breastfed. This contrasts with our sample where only 2/18 of the children in this age range were still nursing. The immunoprotective effects of human breast milk against parasites, bacteria and viruses in the

infant gut are well documented. Indeed Lunn and associates (1999) themselves noted that *Giardia* infections in their older subjects who received little or no breast milk appeared to be more severe and to have compromised their growth. Similarly, both Gupta and associates (1982) and Farthing and associates (1986) reported that growth faltering became more evident during the 2nd year of life when the children in their study were no longer breastfeeding.

Our study findings also differ from those published by Saldiva and associates (1999). These authors reported being unable to observe a significant association between *Giardia* infection (alone or combined with other IPI's) in the 520 rural Brazilian children they studied. Their subjects ranged in age from 1–12 yrs. The reason for the observed discrepancy is not immediately evident. The children in both studies were of similar age. The prevalence of pathogenic IPI's was also comparable except for *Giardia*, which was increased in their study (i.e. 44% vs. 25.2%). In contrast, the better housing and potable water conditions, which characterized the Brazilian children, suggested that they enjoyed better socioeconomic conditions compared to our study subjects. This appeared to be reflected in their improved nutritional status since the prevalence of both stunting (11.5% vs. 37.9%) and wasting (0.6% vs. 4.0%) was much reduced in the Brazilian children. Unfortunately, between-study comparisons of iron nutriture cannot be made since they did not collect data on hemoglobin or other biochemical iron status measures. We suggest that the improved nutritional status of the children in their study may account for the apparent lack of association between *Giardia* infection and growth stunting.

The precise mechanisms responsible for the adverse effects of *Giardia* infection on the nutritional status of experimental animals and humans remains under investigation. One of the ways in which *Giardia* infection may cause growth stunting is through the reduced absorption of macro- and micronutrients in the host gastrointestinal tract (Solomons 1982; Egger et al.1990; Eckmann & Gillin 2001). Acute or chronic diarrhea associated with giardiasis may further exacerbate the situation (Ibid).

On the other hand, due to the cross-sectional design of the study, one cannot be certain whether the infection itself increased the risk for growth stunting or whether the state of chronic

malnutrition, increased the susceptibility for acquiring the parasite. This also may be true for the association found between *Giardia* infection and decreased iron status in the current study. Eckmann and Gillin (2001) have reported that the clinical signs and symptoms as well as duration of infection are highly variable even in immunocompetent individuals infected by the parasite strain. They suggest that both immune and non-immune factors may influence clinical outcome. Host immune response to *Giardia* plays a major role in whether or not the organism is able to attach itself to the intestinal epithelium, time of clearance once it is established, and the development of protective immunity (Fraser 1994).

Nutritional status is a known to be a principal determinant of immune response. Studies conducted over the past several decades have linked PEM with significant reductions in normal cellular immune function, phagocytic function, complement system, secretory IgA levels and cytokine production (Chandra 1999). Likewise, iron deficiency has been reported to increase host susceptibility and the clinical severity of many parasitic infections (Tompkins & Watson 1989). Similar to PEM, iron deficiency is associated with decreased cell-mediated immune function especially delayed cutaneous hypersensitivity reactions, lymphocyte activation of blastocysts, and altered neutrophil and macrophage killing functions, and lymphocyte production (Chandra 1990, 1997, 1999). Larger cohort and clinical studies are needed in this population to clarify whether *Giardia* infection causes chronic malnutrition and/or poor iron status or the converse? Future studies also should investigate the mechanisms, if any, by which this occurs.

Risk Factors for IPI

The one factor most consistently associated with risk for protozoal intestinal parasitic infections was the density of domestic animals living inside and around the home. The most common animals identified were chickens, ducks, pigs, dogs, cats, horses, donkeys, mules and guinea pigs. High animal density appeared to increase the risk for *Giardia*, *Entamoeba histolytica/dispar*, and *Blastocystis hominis* infection from 2.3 to 5.1 times over that of households where fewer animals were present. *Giardia* (Sullivan et al. 1988; Benardska et al. 1998; Fayer et al. 2000) and other protozoa (Doyle *et al.* 1990; Acerrano, 1997; Devera et al. 1999) are reported to be often found in the feces of common domestic animals. These have

been implicated as natural reservoirs involved in the transmission of the disease to humans. In the specific case of the Ecuadorian children, this probably occurs by way of fecal contamination of major drinking water sources from the local river, open shallow wells, and springs. Infection transmission also may occur by children coming into contact with fecally contaminated objects, substances, and the animals themselves and subsequent ingestion of the protozoa via unwashed hands. It also is possible that the cysts and mature forms are transmitted by blowing dust and wind.

No consistent pattern of risk was identified for infection with the helminths, *Ascaris*, *Trichuris*, and *Anclystoma*. However, in the case of the latter, this was probably due to the low infection prevalence (n=4). Also contrary to the a priori hypothesis, no gender differences were identified regarding the risk for acquiring IPI's. Likewise, infants and other young children under the age of five years (< 5 yrs) either had comparable risk for IPI's compared to their older counterparts or in the case of *Trichuris*, were at decreased risk for infection. The reason for this unexpected finding is not clear but it may be the result of a relatively uniform exposure to IPI's across all age and gender groups. The association of age with *Trichuris* however, corresponded with findings in other research such as that by Bundy et al 1987 in St Lucia where the increase in *Trichuris* prevalence was observed to occur rapidly with age and remain high and relatively constant in adulthood (Bundy et al 1987). Similarly, previous research showed no significant association between sex and parasitic infection (Shubair et al 2000, Oberhelman et al 1998, Raj Mahendra et al 1997).

One possible reason for the lack of significant ethnic differences in IPI prevalence, risk and nutritional consequences may be a lack of contrast caused by the relative dearth of minority subjects in the study. However, observations made during visits to both mestizo and minority households suggested that the household sanitation and hygiene practices of the two groups were not distinguishable from each other.

CHAPTER 7: SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

Summary and Conclusions

The results of the survey indicated that IPI's were frequent in the rural Ecuadorian children studied since 90% were infected with at least one pathogenic species. Fifty-one percent of the children were identified with helminthic infections, 37.6% had protozoal infections and 21.4% were infected with both. The most common intestinal parasite detected was *Ascaris lumbricoides* (39.7%), followed by *Giardia intestinalis* (25.2%), *Trichuris trichiura* (19.7%), *Entamoeba histolytica/dispar* (18.5%), *Blastocystis hominis* (13.3%) and *Ancylostoma duodenale* (1.7%). The IPI frequency and distribution is consistent with the pattern reported for children in other rural tropical areas of the country.

Growth stunting and iron-deficiency anemia affected 40% and 26%, respectively, of all study children. Children infected with *Giardia* exhibited a risk for stunted growth that was twice that of their non-infected cohorts. They also had significantly reduced mean blood hemoglobin levels although their risk for iron-deficiency anemia was not significantly increased over that of other children. These findings were of considerable interest especially since at the time of study the children did not demonstrate evidence of acute clinical intestinal infection. The present work adds to the growing body of evidence that like acute infection, asymptomatic giardiasis also may have detrimental consequences for child health and growth. On the other hand, given the cross-sectional nature of the study design, it also is possible that children who were already chronically malnourished may have experienced altered immune function and thus increased susceptibility to acquiring infection compared to better their nourished counterparts.

The characteristic most consistently associated with risk for pathogenic protozoal IPI's was a high density of domestic animals living in and around the home. Children who lived in households with a high animal density had an infection risk that was 2-5 times greater than others. This finding suggests these putative reservoirs may represent an important link in IPI transmission to humans. Contrary to the *a priori* hypothesis, no gender, ethnic, or age differences

in infection risk were identified excepting *Trichuris* infection, which was reduced in younger children contrary to expectations.

In conclusion, the elevated prevalence of intestinal parasitic infections and their nutritional consequences identified in this study, especially that of *Giardia*, indicates the need for mass or targeted chemotherapy among the child population. However, this is only a short-term solution whose costs may outweigh benefits since re-infection typically occurs within a short time if concomitant changes in household and community sanitation do not occur to interrupt the chain of infection. The study data suggest that one low-cost solution to reducing IPI's in this particular group is by decreasing the proximity of one putative reservoir (domestic animals) to homes in order to reduce contamination of inside and outside play areas, water and food supplies. Other low technology, low-cost solutions include the covering of open water sources, improving sanitary food and water storage and handling, and basic personal hygiene. Furthermore, the discharge of untreated household waste-water into the local river and streams should be discouraged as well as the frequent practice of defecation on the open ground and near these drinking water sources. These positive changes can be accomplished through health education and promotion activities targeted at the children and their families. The Ministries of Public Health, Education, and Social Welfare currently have other programs operating in the area, which could be used to better promote household and community sanitation and IPI control.

“Sustainable health, especially for children, is not possible without good environmental sanitation”(WHO. Press Release WHO/18)

Future Research

The majority of prior studies have focused on the effect of hookworm, roundworm and other helminthes on child nutrition status and the impact of chemotherapeutic interventions on the same. Few authors have investigated the adverse effects of asymptomatic *Giardia* and *Entamoeba histolytica* infection, on the health and nutritional status of children. The present study suggests that asymptomatic giardiasis has an important negative impact on child growth and iron status. Thus, it is important to conduct follow-up studies to determine the specific

mechanisms by which the *Giardia* infection affects child growth, iron status, and the absorption of other micro- and macro-nutrients. In addition, future authors should investigate whether regular chemotherapy with metronidazole or other anti-protozoal agents in combination with the hygiene and sanitation improvements mentioned, have a significant positive impact on reducing child morbidity and improving nutritional status.

REFERENCES

- Abo-Shehada, M.N. (1992). A critical study into the fecal analysis routine in practice in Jordan. *Trop Geogr Med* 44, 369-72
- Acerrano, A. (1997). H2uh-O; the aqua's not so pura. *Sports Afield* 217,64-72.
- Albonico, M., Crompton, D.W. & Savioli, L. (1999). Control strategies for human intestinal nematode infections. *Advanced Parasitology*. 42, 277-341.
- Anderson, R.M. & May, R.M. (1985). Helminth infections of humans: mathematical models, population dynamics and control. *Advances in Parasitology* 24, 1-101.
- Astiazaran-Garcia H, Espinosa-Cantellano M, Castanon G, Chavez-Munguia B, Martinez-Palomo A. (2000). *Giardia lamblia*: effect of infection with symptomatic and asymptomatic isolates on the growth of gerbils (*Meriones unguiculatus*). *Experimental Parasitology* 95,128-35.
- Awasthi, S. & Pande, VK. (1997). Prevalence of malnutrition and intestinal parasites in preschool slum children in lucknow. *Indian Pediatrics* 34, 599-605.
- Benefice E. & Barral H. (1991). Differences in the life style and nutritional status between settlers and Siona-Secoya Indians living in the same Amazonian milieu. *Ecology Food and Nutrition* 25,307-322.
- Berdanier, C.D. (1998). *Advanced Nutrition Micronutrients*. Boston, London, New York: CRC Press.

- Booth M., Bundy D.A., Albonico, M., Chwaya, H.M., Alawi, K.S. & Savioli L. (1998). Associations among multiple geohelminth species infections in schoolchildren from Pemba Island. *Parasitology* 116, 85-93.
- Brites, C., Barberino, M.G., Bastos, M.A., Sampaio, Sa. M. & Silva, N. (1997) Blastocystis hominis as a potential cause of diarrhea in AIDS patients: a report of six cases in Bahia, Brazil. *Brazil Journal of Infectious Disease* 1, 91-94.
- Brody, T. (1999). *Nutrition Biochemistry*, 2nd ed. San Diego, New York, Boston, London, Sydney, Tokyo, Toronto:Academic Press.
- Brooker, S., Peshu N., Warn, P.A., Mosobo, M., Guyatt, H.L., Marsh, K., & Snow, R.W. (1999). The epidemiology of hookworm infection and its contribution to anaemia among pre-school children on the Kenyan coast. *Transactions of the Royal Society of Tropical Medicine and Hygiene* 93, 240-6.
- Bundy, D.A. (1994) Immunoepidemiology of intestinal helminthic infections. The global burden of intestinal nematode disease. *Transactions of the royal Society of Tropical Medicine and Hygiene* 88, 259-61.
- Callender, J.E., Grantham-McGregor, S.M., Walker, S.P. & Cooper, E.S. (1994) Treatment effects in *Trichuris* dysentery syndrome. *Acta Paediatrica* 83, 1182-7.
- Centers for Disease Control and Prevention. Recommendations to Prevent and Control Iron Deficiency in the United States. MMWR 1998;47 (No. RR-3):[11- 13]
- Chan, M.S., Medley, G.F., Jamison, D. & Bundy, D.A. (1994). The evaluation of potential global morbidity attributable to intestinal nematode infections. *Parasitology* 109, 373-87.
- Chan, M.S. (1997). The Global Burden of Intestinal Nematode Infections - Fifty years on. *Parasitology Today* 13, 438-443.

Chandra RK. (1997). Nutrition and the immune system: an introduction. *American Journal Clinical Nutrition* 66, 460S-463S.

Chandra RK. (1999). Nutrition and immunology: from the clinic to cellular biology and back again. *Proceedings of the Nutrition Society* 58, 681-3.

Chandra RK. (1991). McCollum Award lecture. Nutrition and immunity: lessons from the past and new insights into the future. *American Journal of Clinical Nutrition*. 53,1087-101.

Connolly, K.J. & Kvalsvig, J.D. (1993). Infection , nutrition and cognitive performance in children. *Parasitology* 107, S187-S200.

Crompton, D.W.T. (1999). How much human helminthiasis is there in the world? *Journal of parasitology* 85, 397-403.

Crompton, D.W.T. (2000). The public health importance of hookworm infection. *Parasitology* 121, S39-S50.

Curtale, F., Tilden R, Muhilal, Vaidya Y, Pokhrel RP, & Guerra R. (1993). Intestinal helminths and risk of anaemia among Nepalese children. 35, 159-66.

Curtale, F., Nabil, M., El Wakeel, A., Shamy, M.Y. and the Behera Survey Team. (1998). Anaemia and intestinal parasitic infections among school age children in Behera Governorate, Egypt. *Journal of Tropical Pediatrics* 44, 323-28.

deSilva, N.R., Jayapani, V.P. & de Silva H.J. (1996). Socioeconomic and behavioral factors affecting the prevalence of geohelminths in preschool children. *Southeast Asian Journal of Tropical Medicine and Public Health* 1, 36-42.

deSilva, N.R., Guyatt, H.L. & Bundy, D.A. (1997). Morbidity and mortality due to Ascaris-induced intestinal obstruction. *Transactions of the Royal Society of Tropical Medicine and Hygiene* 91, 31-6.

Despopoulos, A. & Silbergnagl, S. (1991). *Color Atlas of Physiology*. 4th Edn. George Thieme Verlag, Stuttgart.

Devera R, Requena I, Velasquez V, Castillo H, Gonzalez R. (1999) Pigs as reservoirs of Blastocystis spp. in a rural community of the state of Bolivar, Venezuela. *Enferm Infecc Microbiol Clin* 17, 422.

Dibley, M.J., Goldsby, J.B., Staehling, N.W. & Trowbridge, F.L. (1987). Development of normalized curves for the international growth reference: historical and technical considerations. *American Journal of Clinical Nutrition* 46,736-48.

Doyle, P.W., Helgason, M.M., Mathias, R.G. & Proctor, E.M. (1990). Epidemiology and pathogenicity of *Blastocystis hominis*. *Journal of Clinical Microbiology* 28, 116-121.

Eckmann, L. & Gillin F.D. (2001). Microbes and microbial toxins: paradigms for microbial-mucosal interactions I. Pathophysiological aspects of enteric infections with the lumen-dwelling protozoan pathogen *Giardia lamblia*. *American Journal of Physiology and Gastrointestinal Liver Physiology* 280, G1-G6.

El Molla MA. (1990). Intestinal Parasites. Final Report of the Health Examination Survey. *Ministry of Health, Cairo*. Publication 38/1.

Estrella R, Estevez E, Yopez R., et al. (1996). Valores y limites de referencia de hemoglobina en niños de 8 a 10 años de edad en tres niveles de altitud en Ecuador. In: Berger J, San Miguel S, Arze M, Fernandez E, Aguyo VM, *Anemia Por Deficiencia de Hierro en la Region Andina*, pp 77-96. La Paz, Bolivia: ORSTOM.

Farthing, M.J.G., Mata, L., Urrutia, J.J. & Kronmal, R.A. (1986). Natural history of Giardia infection of and children in rural Guatemala and its impact physical growth. *American Journal Clinical Nutrition* 43, 395-405.

Fayer R, Trout JM, Graczyk TK, Lewis EJ. (2000). Prevalence of Cryptosporidium, Giardia and Eimeria infections in post-weaned and adult cattle on three Maryland farms. *Vet Parasitol.* 93,103-12.

Food and Agriculture Organization of the United Nations. (1997). *Quality control of wastewater for irrigated crop production*. Water reports - 10. Rome, Italy.

Frankenburg, W.K., Dodds, J., Archer, P., Shapiro, H. & Bresnick, B. (1992). The Denver II: a major revision and restandardization of the Denver developmental screening test. *Pediatrics* 89, 91 – 97.

Fraser D, Bilenko N, Deckelbaum RJ, Dagan R, El-On J, Naggan L. (2000). Giardia lamblia carriage in Israeli Bedouin infants: risk factors and consequences. *Clin Infect Dis*, 30, 419-24.

Friis, R.H. & Sellers, T.A. (1999). *Epidemiology for Public Health Practice*. 2nd ed. Maryland:Aspens Publishers, Inc.

Garcia, L.S. (1999). *Practical Guide to Diagnostic Parasitology*. Washington, D.C.: ASM Press.

Gibson, R.S. (1990). *Principles of Nutritional Assessment*. New York, Oxford:Oxford University Press, Inc.

Gilgen, D. & Mascie-Taylor, C.G.N. (2000). The effect of anthelmintic treatment on helminth infection and anaemia. *Parasitology* 122, 105-110.

Gonzalez-Ruiz, A. & Wright, S.G. (1998). Disparate amoebae. *The Lancet* 351, 1672-2.

Grantham-McGregor, S.M., Powell, C.A., Walker, S.P. & Himes, J.H. (1991). Nutritional supplementation, psychosocial stimulation, and mental development of stunted children: the Jamaican Study. *Lancet* 338,1-5.

Grantham-McGregor, S.M., Walker, S.P., Himes, J.H. & Powell, C.A. (1993). The effect of nutritional supplementation and stunting on morbidity in young children: the Jamaican study. *Transactions of the Royal Society of Tropical Medicine and Hygiene* 87, 109-13.

Gupta, M.C & Urrutia J.J. (1982). Effect of periodic antiascaris and anti giardia treatment on nutritional status of preschool. *American Journal Clinical Nutrition*. 36,79-86.

Guyatt, H.L. & Bundy D.A.P. (1993). Estimation of intestinal nematode prevalence: influence of parasite mating patterns. *Parasitology*. 107, 99-106.

Guyatt, H. (2000). Do intestinal nematodes affect productivity in adulthood? *Parasitology Today* 16, 153-58.

Hadju, V., Stephenson, L.S., Abadi, K., Mohammed, H.O., Bowman, D.D. & Parker, R.S. (1996). Improvements in appetite and growth in helminth-infected schoolboys three and seven weeks after a single dose of pyrantel pamoate. *Parasitology* 113, 497-504.

Hadju, V., Abadi, K. & Stephenson, L.S. (1997). Relationships between soil-transmitted helminthiases and growth in urban slum schoolchildren in Ujung Pandang, Indonesia. *International Journal of Food Science and Nutrition* 48, 85-93.

Haque, R., Ali, I.M. & Petri, W.A. (1999). Prevalence of immune response to *Entamoeba Histolytica* infection in preschool children in Bangladesh. *American Journal of Tropical Medicine and Hygiene* 60, 1031-34.

Horii, V. & Usui, M. (1985). Experimental transmission of *Trichuris* ova from monkeys to man. *Transactions of the Royal Society of Tropical Medicine and Hygiene* 79, 423.

Jemaneh L. (1998). Comparative prevalences of some common intestinal helminth infections in different altitudinal regions in Ethiopia. *Ethiopia Medical Journal* 36, 1-8.

Juckett, G. (1996). Intestinal protozoa. *American Family Physician* 53, 2507-2516

Kaneda Y, Horiki N, Cheng X, Tachibana H and Tsutsumi Y. (2000) Serologic response to *Blastocystis hominis* infection in asymptomatic individuals. *Tokai J Exp Clin Med* 25, 51-6

Katz, M., Despommier, D.D. & Gwadz, R.W. (1989). *Parasitic Diseases*. 2nd ed. New York Inc: Springer-Verlag.

Keusch, G.T., Migasena, P. (1982). Biological Implications of Polyparasitism. *Reviews of Infectious Diseases*. 4, 880-82.

Kidala D., Greiner T. & Gebre-Medhin, M. (2000). Five-year follow-up of a food-based vitamin A intervention in Tanzania. *Public Health Nutrition* 3, 425-431.

Lopez, M.E., Mata, L., Lizano, C. & Gamboa, F. (1978) Duodeno – jejunal infection in the child with protein energy malnutrition. *Rev. Med Hosp. Natl. Ninos* 13,53-62,

Lunn, P.G., Hezekioh, E.O., Northrop-Clewes, C.A. & Boyce, S.A. (1998). *Giardia intestinalis* is unlikely to be a major cause of poor growth of rural Gambian infants. *Journal of Nutrition* 129, 872-77.

Marei, M.A., Al-Hamshary, A.M.S., Abdalla, K.F. & Abdel-Maabooud, A.I. (1998). A study on secretory IgA in malnourished children with chronic diarrhoea associated with parasitic infections. *Journal of Egyptian Society of Parasitology* 28, 907-13.

Marsden, P.D. (1982) Treatment and control of Parasitic Diseases. *Reviews of Infectious Diseases* 4, 885-90.

Martinez Perez, A. & Justiniani Cedeno, N.E. (1999). Incidencia de parasitosis intestinales en pacientes pediaticos hematologicos de 1 a 15 anos de edad. *Revista Alergia Mexico* XLVI, 26-29.

Mata, L.J. (1978). *The children of Santa María Cauqué*. Cambridge, MA: MIT Press.

Mata L. (1982). Sociocultural Factors in the Control and Prevention of Parasitic Diseases. *Reviews of Infectious Diseases* 4, 871-79.

Maxwell CJ, Hussain R, Nutman TB, et al. (1987) The clinical and immunologic responses of normal human volunteers to low dose hookworm (*Necator americanus*) infection. *American Journal of Tropical Medicine Hygiene* 37,126-134,

McGarvey, S.T., Aligui, G., Daniel, B.L., Peters, P., Olveda, R. & Olds, G.R. (1992) Child growth and schistosomiasis japonica in northeastern Leyte, the Philippines: cross-sectional results. *American Journal of Tropical Medicine Hygiene* 46,571-81

McGarvey, S.T., Wu, G., Zhang, S., Wang, Y., Peters, P., Olds, G.R. & Wiest, P.M. (1993). Child growth, nutritional status, and schistosomiasis japonica in Jiangxi, People's Republic of China. *American Journal of Tropical Medicine and Hygiene* 48, 547-53.

Mehlhorn, H. (1988) *Parasitology in focus: facts and trends*. Berlin, Heidelberg: Springer-Verlag.

Mendez, M.A. & Adair, L.S. (1999). Severity and timing of stunting in the first two years of life affect performance on cognitive tests in late childhood. *Journal of Nutrition* 129, 1555-1562.

Montresor, A., Crompton, D.W.T., Hall, A., Bundy, D.A.P. & Savoli, L. (1998). Guidelines for the evaluation of soil-transmitted helminthiasis and schistosomiasis at community level.

Division of Control of Tropical Parasites Unit, World Health Organization : Geneva, Switzerland, WHO/CTD/SIP/98.1)

Moreano M, Carrasco F, Bacallao J. Desnutricion y Condiciones socioeconomicas en el Ecuador. Quito: SISVAN/CONADE/UNICEF/PAHO/WHO, 1994.

MSP 2001. MODERSA. Ministerio de Salud Pública. *Proyecto de Modernización de Servicios de Salud "Modersa"*. Available at: <http://www.msp.gov.ec> Accessed June 2001.

Neva, F.A. & Brown, H.W. (1994). *Basic Clinical Parasitology*, 6th ed. Norwalk, Connecticut: Appleton and Lange.

Nimri, L.F. (1993). Evidence of an epidemic of *Blastocystis hominis* infections in preschool children in northern Jordan. *Journal of Clinical Microbiology* 31, 2706 – 8.

Nokes, C., Cooper, E.S., Robinson, B.A. & Bundy, D.A. (1991). Geohelminth infection and academic assessment in Jamaican children. *Transactions of the Royal Society of Tropical Medicine and Hygiene* 85, 272-3.

Nokes, C., Grantham-McGregor, S.M., Sawyer, A.W., Cooper, E.S. & Bundy, D.A. (1992). Parasitic helminth infection and cognitive function in school children. *Proceedings. Royal Society of London. Series B. Biological Sciences* 247, 77-81.

Nokes, C. & Bundy, D.A.P. (1994). Does helminth infection affect mental processing and educational achievement? *Parasitology Today* 10:14-18.

Northrop-Clewes, C.A., Roushman, E.K., Mascie-Taylor, C.G.N. & Lunn, P.G. (2001). Anthelmintic treatment of rural Bangladeshi children: effect on host physiology, growth, and biochemical status. *American Journal of Clinical Nutrition* 73, 53-60.

Nunez- Fernandez, F.A., Sanjurjo Gonzalez, E., & Finlay Villalvilla, C.M. (1991) Comparison of several coproparasitological techniques for the diagnosis of soil-transmitted intestinal helminthiasis. *Rev Inst Med Trop Sao Paulo* 33,403-6

Oberhelman, R.A., Guerrero, E.S., Fernandez, M.L., Silio, M., Mercardo, D., Comiskey, N., Ihenacho, G. & Mera, R. (1998). Correlations between intestinal parasitosis, physical growth, and psychomotor development among infants and children from rural Nicaragua. *American Journal of Tropical Medicine and Hygiene* 58, 470-5.

O'Lorcain, P. & Holland, C.V. (2000). The public health importance of *Ascaris lumbricoides*. *Parasitology* 121, S51-S71.

Ortega F. *A cost-effectiveness analysis of anthelmintic intervention for community control of ascariasis: Traditional vs. pharmaceutical therapy*. Oregon State University, Dissertation Abstracts Online 2000; 61, no. 06B (2000): p. 3025 (ISBN: 0-599-79373-2)

OTA. *Healthy Children: Investigating the Future*. US. Congress, Office of Technology Assessment publication No OTA-H-345. Government Printing Office, 1988.

Paul, I., Gnanamanti, G. (1998). Re-infection estimation of soil-transmitted helminths among slum school children in Visakhapatnam, Andhra Pradesh. *Journal of Communicable Disease* 30, 245-49.

Pawlowski, Z.S., Schad, G.A. & Stott, G.J. (1991). *Hookworm Infection and Anemia*. Geneva, World Health Organization

Peng, W., Anderson, T.J.C., Zhou, X. & Kennedy, M.W. (1998). Genetic variation in sympatric *Ascaris* populations from humans and pigs in China. *Parasitology* 117, 355-361.

Petri, W.A. & Singh, U. (1999). Diagnosis and management of Amebiasis. *Clinical Infectious Diseases* 29, 1117-25.

Raj Mahendra S., Sein, K.T., Anuar, A.K. & Mustaffa, B.E. (1997). *Intestinal Helminthiasis in relation to height and weight of early primary school children in Northeastern Peninsular Malaysia* 28, 314-320.

Roche, M. & Layrisse, M. (1966). The nature and causes of "hookworm anemia". *American Journal Of Tropical Medicine and Hygiene* 15,1031-02.

Rousham, E.K. & Mascie-Taylor C.G.N. (1994). An 18-month study of the effect of periodic anthelmintic treatment on the growth and nutritional status of pre-school children in Bangladesh. *Annals of Human Biology* 21, 314-24.

Sakti, H, Nokes, C, Hertanto, W.S., Hendratno, S, Hall, A, Bundy D.A. & Satoto. (1999). Evidence for an association between hookwork infection and cognitive function in Indonesian school children. *Tropical Medicine and International Health* 4, 322-34.

Saldiva, S.R., Silveira A.S., Philippi S.T., Torres D.M., Mangini A.C., de Souza Dias, da Silva, R.M., Buratini, M.N., & Massad. E. (1999). *Ascaris-Trichuris* association and malnutrition in Brazilian children. *Pediatric and Perinatal Epidemiology* 13, 89-98.

Scrimshaw, N.S. (1994). The consequences of hidden hunger for individuals and societies. *Food and Nutrition Bulletin* 15, 3-24.

Sebastian M.S. & Santiago S. (2000). Control of intestinal helminths in schoolchildren in Low-Napo, Ecuador: impact of a two-year chemotherapy program. *Revista da Sociedade Brasileira de Medicina Tropical* 33, 69-73.

Shils, E.M., Olson, J.A., Shike, M. & Ross A.C. (1999). *Modern Nutrition in Health and Disease*, 9th ed. Philadelphia, Baltimore, New York, London: Lippincott Williams and Wilkins.

Shubair, M.E., Yassin, M.M., Al-Hindi, A.I., Al-Wahaidi, A.A., Jadallah, S.Y. & Abu Shaaban N. Al-Dein S. (2000). *Intestinal parasites in relation to haemoglobin level and nutritional status of school children in Gaza* 30, 365-75.

SIISE 2001a. La década de en cifras - *La pobreza y la extrema pobreza de consumo*. Available at: <http://www.siise.gov.ec> Accessed June 2001.

SIISE 2001b. La década de en cifras - *El acceso a agua segura en la vivienda*. Available at: <http://www.siise.gov.ec> Accessed June 2001.

SIISE 2001c. La década de en cifras - *El acceso a la red de alcantarillado*. Available at: <http://www.siise.gov.ec> Accessed June 2001.

SIISE 2001d. La década de en cifras - *La inversión pública en educación y salud*. Available at: <http://www.siise.gov.ec> Accessed June 2001.

SIISE 2001e. La década de en cifras - *La desnutrición de los niños/as*. Available at: <http://www.siise.gov.ec> Accessed June 2001.

Simeon, D.T., Grantham-McGregor, S.M., Callender, J.E., & Wong, M.S. (1995). Treatment of *Trichuris trichiura* infections improves growth, spelling scores and school attendance in some children. *Journal of Nutrition* 125, 1875-83.

Solomons, N.W. (1982). Giardiasis: Nutritional implications. *Reviews of Infectious Diseases*. 4, 859-69.

Stanley, S.L., Reed, S.L. (2001). Microbes and microbial toxins: paradigms for microbial-mucosal interactions VI. *Entamoeba histolytica*: parasite-host interactions. *American Journal Physiology and Gastrointestinal Liver Physiology* 280, 1049-54.

Stephenson L.S., Latham M.C., Adams E.J., Kinoti, S.N. & Pertet A. Physical fitness, growth and appetite of Kenyan school boys with hookworm, *Trichuris trichiura* and *Ascaris lumbrocooides* infections are improved four months after a single dose of albendazole. *Journal of Nutrition* 123, 1036-46.

Stephenson, L.S., Latham, M.C. & Ottesen E.A. (2000a). Global malnutrition. *Parasitology* 121, S5-S22.

Stephenson, L.S., Latham, M.C. & Ottesen, E.A. (2000b). Malnutrition and parasitic helminth infections. *Parasitology* 121, S23-S38.

Stephenson, L.S., Holland, C.V. & Copper, E.S.(2000). The public health significance of *Trichuris trichiura*. *Parasitology* 121,S73-S95.

Stoltzfus, R.J., Albonico, M., Tielsch, J.M., Chwaya, H.M. & Savioli, L. (1997). Linear growth retardation in Zanzibari school children. *American Society for Nutritional Sciences* 127, 1099-05.

Stoltzfus, R.J., Chwaya, H.M., Tielsch, J.M., Schulze K.J., Albonico, M. & Savioli, L. (1997). Epidemiology of iron deficiency anemia in Zanzibari school children: importance of hookworms. *American Journal of Clinical Nutrition* 65, 153-159.

Stoltzfus, R.J., Albonico M., Chwaya H.B., Tielsch J.M., Schulze K.J. & Savioli, L. (1998). Effects of the Zanzibar school-based deworming program on iron status of children. *American Journal of Clinical Nutrition* 68, 179-186.

Sullivan, P.B., Marsh, M.N., Phillips, M.P., Dewitt, O., Neale, G.,Cevallos, A.M., Yamsin, P. & Farthing, M.J.G. (1990). Prevalence and treatment of giardiasis in chronic diarrhoea and malnutrition. *Archives of Disease in Childhood* 65, 304-306

Taamasri, P., Mungthin, M., Rangsin, R., Tongupprakarn, B., Areekul, W. & Leelayoova, S. (2000). Transmission of intestinal blastocystis related to the quality of drinking water. *Southeast Asian Journal of Tropical Medicine and Public Health* 31,112-7

Thein Hlaing, Myat, L.K., Hlaina, M. & Maung, M. (1990). Role of ascariasis in surgical abdominal emergencies in the Rangoon Children's Hospital, Burma. *Annals of Tropical Paediatrics* 10, 53-60.

Thein Hlaing. (1993). Ascariasis and childhood malnutrition. *Parasitology* 107, S125-S136.

Tompkins A, Watson F. Malnutrition and Infection: A Review. ACC/SCN State of the Art Policy Discussion Paper No. 5. Geneva: United Nations, 1989.

Tsuyuoka, R., Bailey, J.W., d'Avila A.M., Guimaraes, N., Gurgel, R.Q., et al. (1999). Anemia and intestinal parasitic infections in primary school students in Aracaju, Brazil. *Cad Saude Publica* 15, 413-21.

Valencia, M.E., McNeill, G., Haggarty, P., Moya, S.Y., Pinelli, A., Quihui, L. & Davalos R. (1995). Energetic consequences of mild *Giardia intestinalis* infestation in Mexican children. *American Journal of Clinical Nutrition* 61, 860-5.

Varrea Teran J, Fornasini M, Weigel MM, Betancourt J, Mantilla G. (1997). *Analisis del Estado Nutricional de 32,000 Ninos de 0-59 meses, al Ingreso en el O.R.I. (Operacion Recate Infantil)*. [Analysis of the Nutritional Status of 32,000 Children Aged 0-59 Months, Prior to Supplementation at Entry in the O.R.I. Program] Final Technical Report, ORI Program, Ministry of Social Welfare, Quito, Ecuador, 12/97.

Walsh, J.A. (1986). Problems in Recognition and Diagnosis of Amebiasis: Estimation of the Global Magnitude of Morbidity and Mortality. *Reviews of Infectious Diseases* 8, 228-38.

Warren, K.S., Bundy, D.A.P., Anderson, R.M., Davis, A.R., Henderson, D.A., Jamison, D.T., Prescott, N. & Senft, A. (1993). *Helminth Infection*. Oxford University Press, Oxford.

Watkins, W.E. & Pollitt, E. (1996). Effects of removing *Ascaris* on the growth of Guatemalan schoolchildren. *Pediatrics* 97, 871-876.

Watt, R.M., Philip A, Wos, S.M. & Sam, G. J. (1986). Rapid assay for immunological detection of *Trichomonas vaginalis*. *Journal of Clinical Microbiology* 24, 551- 5.

Weigel, M.M., Armijos, R.X., Monaco, M.H., Izurieta, R., Racines, R.J., Zurita, C. & Jaramillo, G. (1992). Nutritional and health status of rural women colonists in the subtropical lowlands of northwest Ecuador. *Ecology of Food and Nutrition*. 29, 25-43.

Weigel, M.M. & Armijos, R.X. (1995). *Rapid Assessment of the El Corazon and Magdalena Alta Communities*: Final Technical Report to the Center for Tropical Rainforest Investigations, Quito, Ecuador, 1995 pp1-35

Weigel, M.M. & Castro Morillo NP. The food acquisition, dietary practices, and nutritional status of minority women of African descent living in tropical South America. *Ecology of Food and Nutrition* 19,1-33, 2000

Weigel M.M., Narvaez WM, Lopez A, et al.(1991) Prenatal diet, nutrient intake, and pregnancy outcome in an urban Ecuadorian population. *Arch Latinoamer Nutr* 26,21-37

Weigel MM, Calle A, Vega M, et al. (1996). The effect of chronic intestinal parasitic infection on maternal-perinatal outcome. *International Journal of Gynecology and Obstetrics* 52:9-17.

White, C.J., Maxwell, C.J. & Gallin, J.I. (1986). Changes in the structural and functional properties of human eosinophils during experimental hookworm infection. *Journal of Infectious Disease* 159, 778 – 783.

Wilson, W.M., Dufour, D.L., Staten, L.K., Baarac-Nieto, M., Reina, J.C. & Spurr, G.B. (1999). Gastrointestinal parasitic infection, anthropometrics, nutritional status, and physical work capacity in Colombian boys. *American Journal of Human Biology* 11, 763-71.

World Health Organization (1994). *Bench Aids for the Diagnosis of Intestinal Parasites*. Geneva, WHO.

World Health Organization (1996). *Report of the WHO Informal Consultation on the Use of Chemotherapy for the Control of Morbidity due to Soil-Transmitted Nematodes in Humans* (WHO/CTD/SIP/96.2). Schistosomiasis and Intestinal Parasites Unit, Division of Control of Tropical Diseases, WHO, Geneva, Switzerland.

World Health Organization (1997). *Report of a Consultation of Experts on Amoebiasis* (WHO/PAHO/UNESCO). WHO Weekly Epidemiological Record No. 14. Geneva, World Health Organization.

World Health Organization (1999). *Monitoring Helminth Control Programmes* (WHO/CDC/SIP/99.3). Guidelines for monitoring the impact of control programmes aimed at reducing morbidity caused by soil-transmitted helminths and schistosomes, with particular reference to school-age children, WHO, Geneva, Switzerland.

World Health Organization (2000). *Intestinal Parasites*. Available at: <http://www.who.int/ctd/intpara/burdens.htm>. Accessed May 2000.

World Resources Institute. (1990). *World Resources*. Washington, D.C. World Resources Institute.

Yassin, M., Shubair, M.E., Al-Hindi, A.I. & Jadallah, S.Y. (1999). Prevalence of intestinal parasites among school children in Gaza city, Gaza strip. *J. Egypt. Society of Parasitology* 29, 365-73.

APPENDICES

APPENDIX A: CONSENTMENT FORM CONSENTIMIENTO

AL LEER Y/O ESCUCHAR ESTE CONSENTIMIENTO, MI HIJO/HIJA, O DEPENDIENTE ACEPTAMOS PARTICIPAR EN EL PROGRAMA NUTRICIONAL SOBRE, *“INFECCIONES POR PARASITOS INTESTINALES: PREVALENCIA, FACTORES DE RIESGO Y CONSECUENCIAS POR MALNUTRICION EN SU PESO Y DESARROLLO, EN NIÑOS”*

Mi hijo / hija _____ y yo aceptamos voluntariamente el participar en el programa dirigido por el Dr. Rodrigo Armijos y la Dra. Margarita Weigel. Los puntos del programa es determinar que parásitos intestinales infectan y alteran la nutrición tanto en peso como en desarrollo en mi hijo/hija y otros niños de la comunidad.

Yo he sido informada que el programa es importante y gratuito en donde se indica que a mi hijo/hija se le realizará exámenes de coproparasitario (muestras de heces) por tres días consecutivos para determinar si el niño/niña necesita tratamiento para parásitos intestinales. También se realizara preguntas basicas acerca de mi hijo/hija y la historia familiar, y medica del niño/niña (como si alguna vez utilizo antiparasitarios, suplementos vitamínicos/ minerales y también las conducciones de vivienda). Mi hijo será sometido a un examen médico de rutina, y a un chequeo nutricional, luego participar en un screening para que se le realice el examen de Denver para control de desarrollo. Se realizará algunas preguntas acerca de la alimentación del niño/niña, un recordatorio dietético de 24 horas, finalmente se tomará una pequeña cantidad de sangre del niño/niña en capilares para determinar en el niño/niña presenten anemia.

He sido informada también que a mi hijo se le realizará exámenes de rutina que se harán dentro de los servicios de salud utilizando un tiempo requerido aproximadamente de 1-2 horas y en caso necesario se podrá alargar un poco mas. Los riesgos par la realización de estos exámenes son mínimos, aquellos incluyen la posibilidad de un poquito de molestia causada por la colección de una pequeña muestra de sangre (una gota) en el sitio de punción.

Los beneficios de participar en el programa pueden incluir exámenes de laboratorio gratuitos de parásitos intestinales, anemia, diagnóstico medica como nutricional y la administracion de medicación de acuerdo a los resultados de exámenes y en caso necesario, referencia del niño a centros hospitalarios en las ciudades de Sto Domingo de los Colorados o Quito. Otros beneficios importantes incluye también el mejorar la salud y nutrición en los niños que viven en la comunidad, y en otros sitios tanto del Ecuador como de otros países.

DECLARO QUE NO RECIBIRE NI EXIGIRE BENEFICIO ECONOMICO NI DE OTRA INDOLE POR LA PARTICIPACION DE MI HIJO/HIJA EN EL PROGRAMA, EXCEPTO LOS ARRIBA MENCIONADOS.

Entiendo que el personal del programa hará respetar la privacidad y el anonimato por la participación de mi hijo /hija y también los resultados de los exámenes servirán para determinar el tratamiento adecuado. Entiendo que mi hijo/hija y yo tenemos plena libertad de retirarnos del programa en cualquier momento. Todos mis preguntas e inquietudes han sido respondidas satisfactoriamente por el personal del programa y si tuviera en el futuro otras preguntas podre solicitar amplia información del mismo.

A pesar de todas las precauciones se tengan en cuenta, de presentarse alguna complicación mi hijo/hija o mi dependiente serán asistido inmediatamente por el personal del programa recibiendo atención médica y tratamiento adecuado.

Si usted tienes preguntas o inquietudes por favor contactarse con los médicos de la Fundación Biociencias en Puerto Quito o con los directores del programa en las oficinas de la fundación en Quito – Dr. Rodrigo Armijos o Dra. Weigel, Instituto Biomedicina (4to piso), Iquique y Sodiro S/N

Facultad de Ciencias Medicina, Telef: Directo: 222- 310.

MI FIRMA O HUELLA INDICA QUE HE LEIDO Y/O ESCUCHADO, COMPRENDIDO, Y ACEPTADO QUE YO Y MI DEPENDIENTE HIJO/HIJA, SEAN INCLUIDOS EN EL PROGRAMA Y QUE HE RECIBIDO UNA COPIA DE ESTE DOCUMENTO

.....
Fecha

.....
Firma del responsable

.....
No de C.I

.....
Firma del Investigador

.....
Firma del testigo

APPENDIX B: STRUCTURED QUESTIONNAIRE

INFECCIÓN DE PARASITOS INTESTINALES: PREVALENCIA, FACTORES DE RIESGO Y CONSECUENCIAS POR MALNUTRICIÓN CREMEINTO, Y DESARROLLO EN NIÑOS ECUATORIANOS EN ZONAS RURALES.

Fecha: ____/____/____
 día mes año

Sitio de Intervención: _____

PARTE 1. CARACTERISTICAS SOCIODEMOGRAFICAS

Nombre del niño: _____

Fecha de nacimiento: ____/____/____
 día mes año

Sexo del niño: F M

Dirección Domiciliaria: _____

Nombre de la Madre:

_____/_____/_____
 Apellidos Nombres

Etnicidad: Mestizo Afro-Ecuatoriano Indígena Otros: _____

Ocupación (primaria): _____

Educación (años completos): _____

Estados Civil: Casada Unión Libre Soltera Separada Divorciada Viuda

Nombre del Padre:

_____/_____/_____
 Apellidos Nombres

Etnicidad: Mestizo Afro-Ecuatoriano Indígena Otros: _____

Ocupación (primaria): _____

Educación (años completos): _____

Estados Civil: Casado Unión Libre Soltero Separado Divorciado Viudo

Información de otros familiares que viven en casa:

Nombres:	Edad	Sexo	Relación
----------	------	------	----------

1. _____
2. _____
3. _____
4. _____
5. _____
6. _____

Nombre: _____
Fecha: _____
Lugar: _____

2. CARACTERISTICAS DE LA VIVIENDA

La casa dónde usted vive es?

_____ Propria _____ Arrendada
_____ Otro (_____)
_____ No sé/ no recuerda / no responde

A que distancia está su casa de sus vecinos más cercanos?

_____ metros.
_____ No sé/ no recuerda / no responde.

Qué tipo iluminación tiene su casa?

_____ Eléctrica _____ Kerosene/gasoline (candil)
_____ Velas _____ Otras
_____ No sé/ no recuerda / no responde.

Qué tipo de construcción tiene su casa?

_____ Bloque _____ Mixta
_____ Madera _____ Otras
_____ No sé/ no recuerda / no responde.

La vivienda cuenta con agua de?

_____ Agua potable municipal _____ Pozo séptico
_____ Lluvia _____ Vertiente
_____ Agua de Rio _____ Otras
_____ No sé/ no recuerda / no responde.

En su casa tiene?

_____ Servicio higienico dentro de la casa _____ Tasa sanitaria fuera de la casa
_____ Letrina dentro de la casa _____ Letrina fuera de la casa
_____ Campo abierto _____ Otros
_____ No sé/ no recuerda / no responde.

La eliminacion de aguas servidas de su casa es?

_____ Municipal _____ Pozo séptico
_____ Río o quebrada _____ Campo abierto
_____ Otras _____ No sé/ no recuerda / no responde

Como elimina usted la basura?

_____ Quemar _____ Rivera de rio o quebrada
_____ Enterrar _____ Enterando en fosas
_____ Campo abierto _____ Otras _____ No sé/ no recuerda / no responde

Nombre: _____
Fecha: _____
Lugar: _____

A qué distancia esta su casa del sitio donde usted elimina la basura?

_____ metros
_____ No sé/ no recuerda / no responde

Usted desinfecta (o trata) el agua?

Si	No		Si	No	
_____	_____	Tomar	_____	_____	Lavar los alimentos
_____	_____	Bañarse	_____	_____	Otro
_____	_____	No sé/ no recuerda / no responde			

Si desinfecta el agua (o trata) que utiliza:

Describe lo que utiliza para consumir el agua, preparar los alimentos (ej. hervir, cloro, otros).

_____ No sé/ no recuerda / no responde.

Usted después de salir del baño, letrina, o después de cambiar el pañal o asear a sus niños se lava las manos?

_____ Siempre	_____ Ocasionalmente
_____ Frecuentemente	_____ Cuando se acuerda
_____ Nunca	_____ No sé/ no recuerda / no responde

Su niño después de salir del baño o letrina se lava las manos?

_____ Siempre	_____ Ocasionalmente
_____ Frecuentemente	_____ Cuando se acuerda
_____ Nunca	_____ No sé/ no recuerda / no responde

Tiene animales domésticos que viven en la casa o alrededor de ella

_____ Si
_____ No _____ No sé/ no recuerda / no responde

Si la respuesta es sí, que animal's tiene, cuántos animales tiene?

Animal	Número	Libres	Encerrados

_____ No sé/ no recuerda / no responde

Nombre: _____
Fecha: _____
Lugar: _____

INSTRUCCIONES

**DIBUJAR LA LOCALIZACION DEL AREA DONDE VIVE, FACILIDADES DE
HYGIENE, SERVICIOS DE AGUA, DONDE VIVEN LOS ANIMLES DOMESTICOS,
ELIMINACION DE ECRETAS.**

A large, empty rectangular box with a thin black border, intended for the student to draw the location of their living area, hygiene facilities, water services, domestic animals, and waste disposal.

Nombre: _____
Fecha: _____
Lugar: _____

3. CARNET DE SALUD (Pregunte y mire l carnet de salud y verifique la información).

Información al nacimiento:

Peso: _____ gramos
Talla: _____ cm
PC: _____ cm
PB: _____ cm

Nacimiento:
_____ prematuro (< de 37 semanas)
_____ normal

Recordatorio post natal con el Carnet de Salud (Evolución de Crecimiento).

1. Edad: _____ meses
Peso: _____ gramos
Talla: _____ cm
PC: _____ cm
PB: _____ cm

6. Edad: _____ meses
Peso: _____ gramos
Talla: _____ cm
PC: _____ cm
PB: _____ cm

2. Edad: _____ meses
Peso: _____ gramos
Talla: _____ cm
PC: _____ cm
PB: _____ cm

7. Edad: _____ meses
Peso: _____ gramos
Talla: _____ cm
PC: _____ cm
PB: _____ cm

3. Edad: _____ meses
Peso: _____ gramos
Talla: _____ cm
PC: _____ cm
PB: _____ cm

8. Edad: _____ meses
Peso: _____ gramos
Talla: _____ cm
PC: _____ cm
PB: _____ cm

4. Edad: _____ meses
Peso: _____ gramos
Talla: _____ cm
PC: _____ cm
PB: _____ cm

9. Edad: _____ meses
Peso: _____ gramos
Talla: _____ cm
PC: _____ cm
PB: _____ cm

5. Edad: _____ meses
Peso: _____ gramos
Talla: _____ cm
PC: _____ cm
PB: _____ cm

10. Edad: _____ meses
Peso: _____ gramos
Talla: _____ cm
PC: _____ cm
PB: _____ cm

Nombre: _____
Fecha: _____
Lugar: _____

Historia de Vacunación (Carnet de Salud)

Vacuna	Recibio (✓)	Edad del niño en que recibió la vacuna
BCG1	_____	_____
BCG2	_____	_____
PVO1	_____	_____
PVO2	_____	_____
PVO3	_____	_____
DPT1	_____	_____
DPT2	_____	_____
DPT3	_____	_____
Antisarampionosa	_____	_____
Otras	_____	_____
	_____	_____

Condiciones Médicas

Su niño ha presentado alguna de estas enfermedades (actuales o pasadas):

_____ Asma _____ IRA _____ Paperas _____ Desnutrición
_____ Bronquitis _____ IVU _____ Varicela _____ otros _____
_____ Alergias _____ Diarrea _____ Parasitos intestinal
_____ Neumonía _____ Sarampión _____ Gripes
_____ No sé/ no recuerda / no responde

Durante el último mes (4 semanas), su niño ha presentado alguna enfermedad?

_____ Si
_____ No _____ No sé/ no recuerda / no responde

Si la respuesta es sí:

Tipo de enfermedad (s)	Cuando?
_____	_____
_____	_____
_____	_____

Su niño ha estado hospitalizado?

_____ Si
_____ No _____ No sé/ no recuerda / no responde

Si la respuesta es si:

Por qué estuvo hospitalizado? Cuando? Por cuánto tiempo estuvo hospitalizado?

_____ No sé/ no recuerda / no responde

Nombre: _____
Fecha: _____
Lugar: _____

4. HISTORIA ALIMENTARIA

Su bebé recibió calostro?

- _____ Si
_____ No
_____ No sé/ no recuerda / no responde

Su bebé recibió seno materno?

- _____ Si Por cuánto tiempo? _____
_____ No sé/ no recuerda / no responde

Por qué dejó de dar el pecho? _____

_____ No sé/ no recuerda / no responde.

Historia de introducción de alimentos sólidos.

Alimentos	Edad de introduccion de alimentos (meses)
_____ Frutas	_____
_____ Cereal	_____
_____ Huevos	_____
_____ Vegetales	_____
_____ Legumbres	_____
_____ Carne	_____
_____ Pescado	_____
_____ Mariscos	_____
_____ Dulces	_____
_____ Otros	_____
_____ No sé/ no recuerda / no responde	

Nombre: _____
Fecha: _____
Lugar: _____

24- RECORDATORIO DE LA DIETA EN LAS ULTIMAS 24 HORAS

PROLOGO AQUI

El día de la semana en que se hace el recordatorio (debe ser el día anterior) :

Lunes Martes Miercoles Jueves Viernes Sabado Domingo

La dieta del día de ayer es: _____ normal
 _____ día especial o de fiesta
 _____ otro

Su niño ayer comió:

_____ lo mismo
_____ menos de lo usual Por qué? _____
_____ más de lo usual Por qué ? _____
_____ No sé/ no recuerda / no responde

Su niño últimamente ha tomado suplementos vitamínicos?

_____ Si Que? _____ Dosis: _____ Frecuencia; _____ No
_____ No
_____ No sé/ no recuerda / no responde

Su niño últimamente a tomado medicación?

_____ Si Que? _____ Dosis: _____ Frecuencia; _____
_____ Si Que? _____ Dosis: _____ Frecuencia; _____ No
_____ No
_____ No sé/ no recuerda / no responde

Nombre: _____
Fecha: _____
Lugar: _____

5. MEDIDAS ANTROPOMETRICAS (actual).

Peso: _____ (kg)

Talla: (del niño menor de 2 años de la corona a los talones: _____ (cm).

PB: _____ (cm).

PC: (< 2 años) _____ (cm).

Circunferencia del muslo: _____ (cm)

DX: _____

Nombre: _____

Fecha: _____

Lugar: _____

6. HEMOGLOBINA (con Hemocue)

_____ g/DL

DX: _____

Nombre: _____
Fecha: _____
Lugar: _____

7. TAMISAJE DE DESARROLLO MENTAL (versión ecuatoriana DENVER II)

Tes edad cronológica del niño _____ meses.

Componentes del tes	Medidas de Desarrollo mental
Motor grueso	_____ = _____
Motor fino	_____ = _____
Lenguaje	_____ = _____
Social	_____ = _____
TOTAL	_____ = _____

Nombre: _____
 Fecha: _____
 Lugar: _____

8. PARASITOS INTESTINALES

Ejemplo1

Fecha	Parásito	Carga parasitaria	Comentarios	Técnicas de Laboratorio

Ejemplo2

Fecha	Parásito	Carga parasitaria	Comentarios	Técnicas de Laboratorio

Ejemplo3

Fecha	Parásito	Carga parasitaria	Comentarios	Técnicas de Laboratorio

VITA

MAMIE – ELEANOR SACKEY

Education:

M.S. Community and International Nutrition, July 2001.

Virginia Polytechnic Institute and State University (Virginia Tech), Blacksburg, VA.

Thesis topic: “Intestinal Parasitic Infection: Prevalence, Risk Factors and Consequences for Malnutrition, Growth and Development in Rural Ecuadorian Children”.

GPA: 3.68/4.0

Bachelor of Science, Biochemistry, June 1997.

University of Ghana, Legon, Ghana, West Africa.

Research: Classification of Cowpea Varieties by DNA Fingerprinting.

Professional Experience:

Research Project, Rural Northeast Ecuador, June–August 2000.

Survey Design

Interview administration

Hemoglobin status evaluation

Intestinal Parasite evaluation

Anthropometric evaluation

Developmental screening

Database management

Graduate Teaching Assistant, August 1999- Present.

Department of Human Nutrition Foods and Exercise, Virginia Tech.

Metabolic Nutrition HNFE 3025

Conducted weekly class review sessions

Developed and graded quizzes, exams and project reports.

Advised and assisted students with course work.

Methods of Human Nutritional Assessment Laboratory HNFE 3034

Organized laboratory set-up and cleaning.

Supervised and assisted students in lab procedures.

Designed and graded quizzes and exams.

Graded lab reports, project reports and displayed grades in online spreadsheet database.

Scheduled biweekly office hours to advise and assist students with assignments.

Research Assistant, November 1998- April 1999.

Diabetes Management Center: Korle-bu Teaching Hospital, Ghana, West Africa.

Assistant supervisor and team member of research staff.

Blood serum analyst for non-communicable disease survey.

Supervised Genetic analysis of type 2 diabetes mellitus survey: participant eligibility determination, participant screening blood and urine biochemical analysis, bioelectric impedance analysis, organized and scheduled clinical investigations, data entry.

Administered and analyzed of questionnaires for Diabetes Survey.

Dietetic Assistant, February – September 1998.

Korle-bu Teaching Hospital, Ghana, West Africa.

Counseled outpatients under supervision of registered dietitians.

Understudied counseling of inpatients and participated in ward rounds.

Researched and interpreted statistical data on trends in diet therapy compliance.

Assisted in designing and editing diet-therapy educational materials.

Teacher (National Service), December 1992- August 1993.

All Saints Anglican School, Ghana, West Africa.

Fifth grade teacher- taught reading, writing, arithmetic, english comprehension, english composition, general science, religious studies, geography, agriculture and history to 5th grade students.

Developed, administered and graded class assignments, homework and exams.

Structured class work to differing abilities of pupils.

Taught english comprehension and physical education to 1st- 7th grade students.

UN Working Languages and Level:

English - Level C

French - Level A - B

Spanish - Level A

Awards & Academic Activities:

Department of Human Nutrition Foods and Exercise Summer 2000 Fellowship

Paul Massey Food and Nutrition Scholar Award 2000.

Local Coordinator and Member of “The European International Model United Nations”
Conference (*TEIMUN), Den Haag, Netherlands 1997.

Member of the General Assembly of TEIMUN, July 1996.