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Soil-transmitted nematode infections among school-age children in Rakai District, Uganda: A study of infection prevalence, intensity, and effectiveness of treatment

Jensen Reckhow

May 2014

Submitted to the Yale School of Public Health
In partial fulfillment of the requirements for the degree of Master of Public Health in Epidemiology of Microbial Disease & Global Health



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Thanks are also due to the many other people who have helped with this project along the way. This project would not have been possible without the hard work of these important people. Thank you to Lisa Harrison for your endless patience with me during laboratory trainings, supply ordering, and the IRB submission process. Thank you to Debbie Humphries for helping me develop targeted and meaningful statistical analysis plans. Thank you to Kaveh Khoshnood, for your guidance, words of encouragement and for helping me keep my efforts in context to ensure a meaningful outcome.

Finally, mwebale nyo to all of my friends at Hope for African Children. Mugagga: thank you for guiding me to become a better microscopist. Betty: thank you for taking time to teach me about your community, and for engaging me in projects and important decisions at HAC. Eddie: thank you for serving as a de-facto bodyguard and for always keeping me safe and comfortable. Julie: thank you for assisting me every step of the way, for being a wonderfully patient Luganda instructor, and for being a constant source of happiness throughout the summer. Keneth and Elysia, thank you for believing in this project, for remaining calm through endless logistical pitfalls, and for working so hard to ensure my experience was a good one. You are a constant source of inspiration.

ABSTRACT

One billion people are currently infected with at least one soil-transmitted nematode (STN), and over 161,600 school-aged children in sub-Saharan Africa live in areas where the prevalence exceeds 20%. STN infections cause malnutrition and cognitive deficits that limit productivity and may contribute to endemic poverty. Despite this significant and recognized disease burden, research on these diseases remains piecemeal; the majority of scientific understanding of these conditions is derived from a handful of small studies. Data regarding prevalence, intensity, and effectiveness of treatment of STN infections in Rakai District, Uganda is particularly limited, and the Ministry of Health has discontinued surveillance in the area due to financial constraints. A cross-sectional study of 269 school-aged children was conducted in Rakai District to address this knowledge gap. Fecal samples were collected by household and analyzed using light microscopy. Demographic and behavioral risk factors for infection were assessed via questionnaire. Subjects who were infected with any of the three major soil-transmitted nematodes (hookworm, *Ascaris lumbricoides*, *Trichuris trichiura*) were offered treatment with single dose oral albendazole (400 mg). The prevalence of hookworm, *Ascaris*, and *Trichuris*, was 55.0%, 49.4%, and 21.2%, respectively, with 70.6% of all subjects infected with at least one STN. In a univariate analysis, young age was associated with hookworm ($p = 0.0239$), *Ascaris* ($p = 0.0186$), and any STN ($p = 0.0010$) infection. Having a recent history of malaria was a risk factor for hookworm ($p = 0.0308$), *Ascaris* ($p = 0.0301$), and any STN ($p = 0.0251$). Moderate/heavy infection intensity was associated with increasing poly-parasitism (OR = 5.2) and treatment failure (OR = 2.3). In a multivariate analysis, recent history of malaria and low weight/height were significant predictors of hookworm (adjusted OR: 1.86, 0.95) or *Ascaris* (adjusted OR: 1.93, 0.94) infections. Pig ownership was a strong predictor of *Trichuris* infection (adjusted OR: 3.38). The cure rate/egg reduction rates following albendazole treatment were as follows: hookworm: 58/79%; *A. lumbricoides*: 74/92%; *T. trichiura*: 82/98 %. In conclusion, this study confirms a high prevalence of three major STN infections in Rakai District, as well as an association with malaria and poor nutritional status. Single dose albendazole therapy exhibited reduced effectiveness, especially against hookworm, in this polyparasitized population. We recommend that future deworming programs in Rakai integrate efforts to modify behavioral risk factors, along with monitoring for treatment effectiveness and emerging anthelminthic resistance.

INTRODUCTION

Specific Objectives of the Investigation

In contrast to HIV/AIDS, malaria, and tuberculosis, the neglected tropical diseases (NTDs) have historically been underrepresented in global health research and interventions. Of the seventeen diseases included within this group, soil-transmitted nematode (STN) infections and schistosomiasis pose the most significant health burdens, affecting over one billion people worldwide (Soukhathammavong et al. 2012). Combined, these helminthiases are responsible for an estimated loss of at least 44 million Disability Adjusted Life Years (DALYs)—more than the 36 million caused by malaria, and approaching the 47 million attributed to tuberculosis (Hodges et al. 2012). Though STN infections are believed to be responsible for over 135,000 deaths annually, and schistosomiasis for over 200,000 annual deaths in sub-Saharan Africa alone, the burden and etiology of these diseases remain poorly characterized (Kabatereine et al. 2011).

Table 3.1.1 Estimated number of disability-adjusted life years (DALYs) (in thousands) by cause (neglected tropical disease), and by WHO region (excluding the European Region)[a], 2004

Neglected tropical disease	World[b]	WHO region				
		African	Americas	Eastern Mediterranean	South-East Asia	Western Pacific
Human African trypanosomiasis	1 673	1 609	0	62	0	0
Chagas disease	430	0	426	0	0	0
Schistosomiasis	1 707	1 502	46	145	0	13
Leishmaniasis	1 974	328	45	281	1 264	51
Lymphatic filariasis	5 941	2 263	10	75	3 525	65
Onchocerciasis	389	375	1	11	0	0
Leprosy	194	25	16	22	118	13
Dengue	670	9	73	28	391	169
Trachoma	1 334	601	15	208	88	419
Ascariasis[c]	1 851	915	60	162	404	308
Trichuriasis[c]	1 012	236	73	61	372	269
Hookworm disease[c]	1 092	377	20	43	286	364

[a] Source: *The global burden of disease: 2004 update* (1).
[b] Because estimates from the European Region were omitted from the table, numbers for the regions may not always add up to the world's total.
[c] Soil-transmitted helminthiases.
The published sources from which these tables are based should be consulted for details of the costs involved.

("Working to overcome the global impact of neglected tropical diseases," World Health Organization, 2010)

In 2000, Uganda became the first country to launch national schistosomiasis and STN infection control programs. These, along with other NTD control programs, were streamlined to avoid redundancy in resource distribution through the establishment of the National Control Program for the Integrated Control of Neglected Tropical Diseases in 2007 (Parker and Allen 2011). Yet the Ministry of Health of Uganda remains pressed for resources in combatting these diseases and continues to face challenges related to the efficient coordination and integration of disease control services (Kabatereine et al. 2005). Furthermore, adequate surveillance data is severely lacking: current prevalence estimates for soil-transmitted nematode infections range from 0% to nearly 90%, and vary widely, even between neighboring districts (Kabatereine et al. 2005). These disparities are likely due to the fact that prevalence estimates are drawn from exceptionally low sample sizes: in most cases, fewer than fifteen individuals are studied in any given location (Parker and Allen 2011). Data on Rakai District is especially lacking. There is currently no prevalence data on record for hookworm infection, and collection of surveillance data for other helminth infections ceased in 2008 (Uganda Vector Control Division, 2013). See Appendix 1 for a comprehensive listing of all data on record at the Vector Control Division regarding STN prevalence in Rakai and neighboring Masaka Districts in Uganda. The helminthiasis disease profile of this region is poorly understood, and additional data regarding prevalence, intensity, and responsiveness to treatment of such infections is desperately needed.

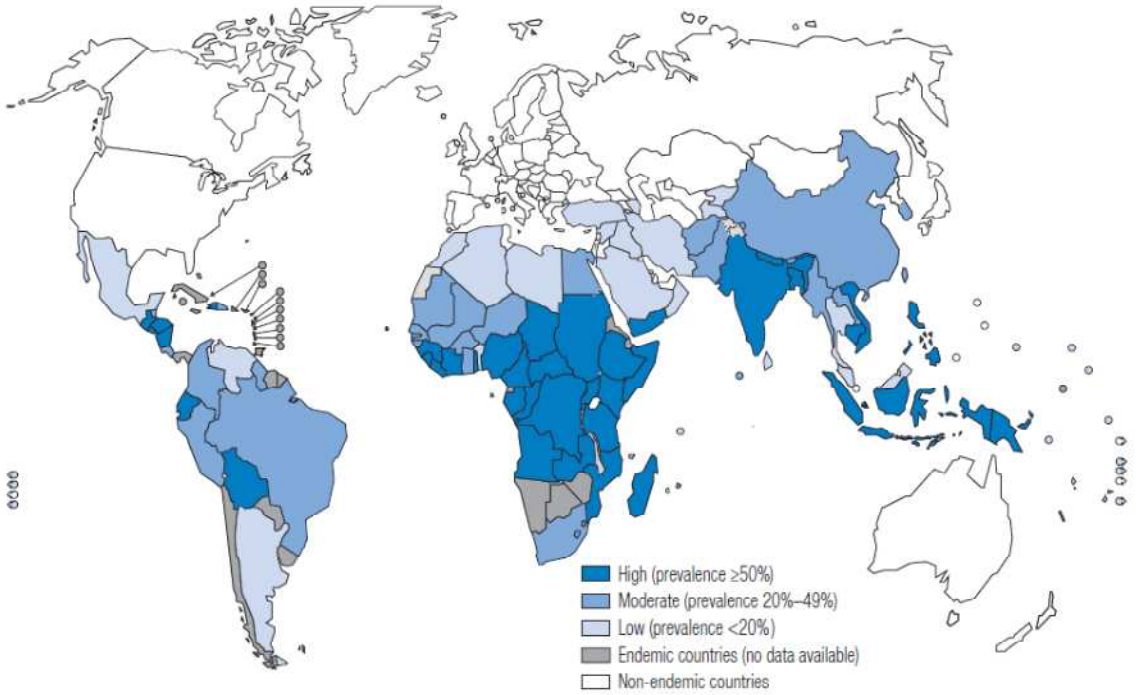
The intent of this investigation is to contribute to current understanding of the disease profile of helminth infections in Rakai District. The specific objectives of the investigation are as follows:

1. To estimate the prevalence and intensity of infection caused by *Schistosoma sp., Necator americanus, Ancylostoma duodenale, Ascaris lumbricoides,* and *Trichuris trichiura* among school-age children residing in Kabuwoko Parish, Kirumba Sub-County of Rakai District, Uganda; and
2. To assess responsiveness to the WHO-recommended anthelmintic treatment of the aforementioned infections.

Helminthiases: A Global Health Problem

Helminthiases are the most widespread of all NTDs (Soukhathammavong et al. 2012). Helminthiasis can result from infection by a number of different helminths, the most globally significant of which are the blood fluke *Schistosoma* species, the hookworms *Necator americanus* and *Ancylostoma duodenale*, the roundworm *Ascaris lumbricoides*, and the whipworm *Trichuris trichiura*. Historically, helminths have had a global distribution (Hotez et al. 2008). Today, these parasites are most commonly found in sub-Saharan Africa, East Asia, China, India, and South America, (Mascarini-Serra 2011). The World Health Organization (WHO) estimates that over 880 million children alone are in need of treatment for disease caused by the soil-transmitted nematodes, making STN infections among the most prevalent in the world.

Fig. 5.17.1 Distribution of soil-transmitted helminthiases, worldwide, 2009



Distribution of soil-transmitted helminthiases is focal in many countries. For the detailed epidemiological situation in countries, please refer to *Preventive chemotherapy and transmission control databank*. Geneva, World Health Organization, 2010 (available at: http://www.who.int/neglected_diseases/ preventive_chemotherapy/databank/en/index.html: accessed January 2009).

("Working to overcome the global impact of neglected tropical diseases," World Health Organization, 2010)

The sequelae of helminth infections are varied and unique. Though most cases of helminthiasis are asymptomatic, these infections are known to exert a subtle but profound fitness cost on their hosts. Acute disease is rare, but the gradual decreases in physical and cognitive health incurred during chronic infection can significantly reduce productivity and earning potential, both of which are immensely difficult to measure (Soukhathammavong et al. 2012, Bethony et al. 2006). Furthermore, co-infection with multiple helminths, as well as with one or more helminths and another disease—both of which are common—are believed to result in unique disease susceptibility and outcomes, though these interactions remain scientifically elusive (Kabatereine et al.

2011). NTDs, and helminthiases in particular, remain poorly understood, but their global health significance is becoming increasingly clear.

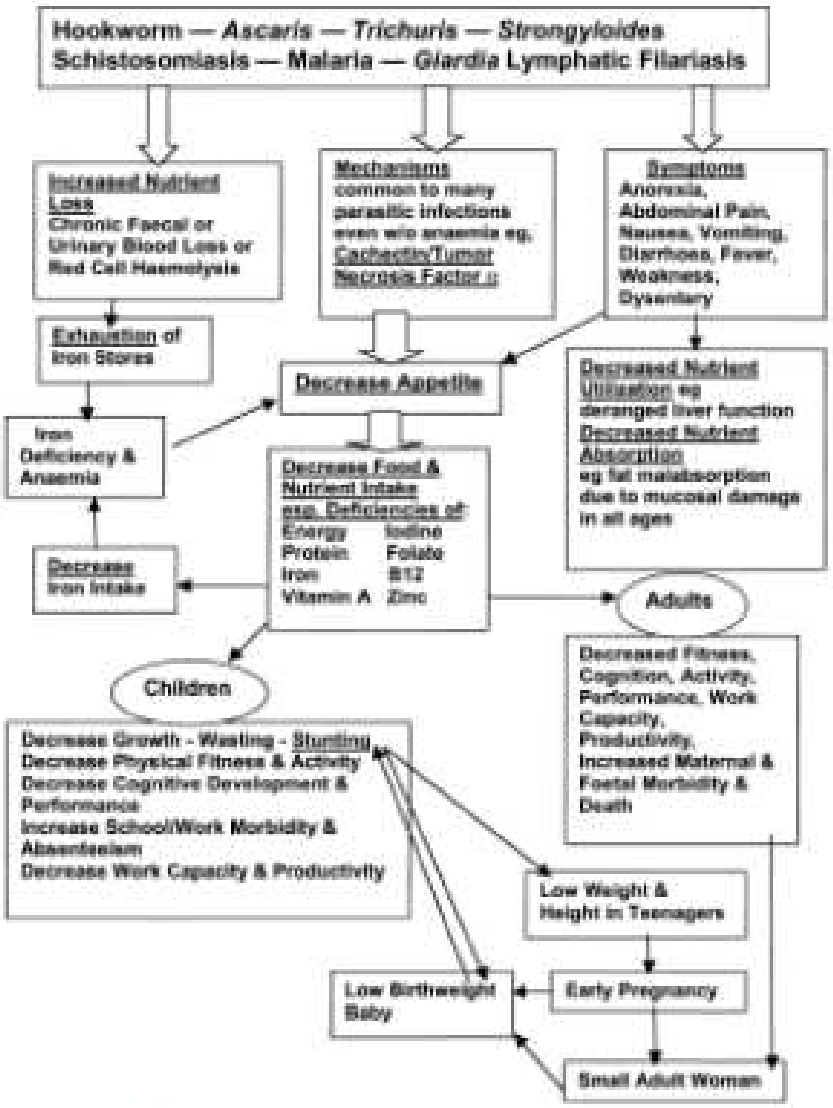


Fig. 1. Conceptual framework: How parasites cause/aggravate malnutrition and retard development. Adapted from Stephenson & Holland, 1987 and ACC/SCN, 1992.

Mechanisms of physical and cognitive growth impairment in helminth infections. (Stephenson et al. 2000)

Though the mechanistic details remain largely uncharacterized, the clinical outcomes of most cases of severe helminthiasis manifest as physical and cognitive impairments, many of which may be lasting and irreversible (Hodges et al. 2012).

Because the effects are often subtle and particularly difficult to quantify, understanding of the health and economic impacts of these diseases remains vague and largely suggestive (Hodges et al. 2012). However, it is clear that the disease sequelae of helminthiases have the potential to impair school performance, physical productivity, and wage-earning potential for the individual, which may lower national productivity and even gross national product when considered in aggregate (Hodges et al. 2012).

Addressing the health burden presented by helminthiases remains a critical global objective. Many nations have national disease control programs, though few have integrated these services, despite the fact that such integration would help streamline resource distribution, as the endemicity profiles of many NTDs (and helminthiases) overlap significantly (Kabatereine et al. 2011). Furthermore, the observed synergism between helminth infection and the outcome of other co-infections suggests that successful control of one disease may result in reductions of another disease without direct treatment, further supporting the case for integrated control (Kabatereine et al. 2011). However, programmatic inefficiencies remain, partly due to poor organization, and partly due to limited scientific understanding of how these parasites function and how best to control them.

Helminths are unique in the parasitic world, as their transmission dynamics differ distinctly from viral and bacterial infections: helminths cannot reproduce inside of a host (Hotez et al. 2004). Furthermore, disease and transmission appear to be functions of infection intensity within the individual: there is evidence that individuals are predisposed to either heavy or light infections, and that the egg output per worm decreases as the number of worms harbored by an individual increases (Sabatelli et al.

2008). Infection intensity, rather than the number of people harboring infection, is of greater interest, as heavier worm burdens are more prone to causing disease (Sabatelli et al. 2008). Because of this, population-wide study is essential to improving scientific understanding of helminthiases. Control strategies rely on targeting those members of the population who harbor the greatest numbers of helminths; understanding the determinants of infection intensity within a population will be essential to any successful control strategy (Sabatelli et al. 2008).

Study Hypotheses & Primary Goals

Though poorly understood, the health and productivity burdens of helminthiases are clearly globally significant. Controlling these diseases is essential. Furthermore, it is becoming increasingly clear that the dynamics of disease and transmission are highly specific at the community level, suggesting that local data and tailored intervention programs will be critical for effective and efficient disease control (Sabatelli et al. 2008). Theoretical rationale for emerging resistance to treatment is sound and supported by limited data; increasing surveillance data on this subject will be indispensible in strengthening the case for the urgency of new treatment options (Humphries et al. 2011). This project was designed with these knowledge gaps in mind, and attempts to make a small but important contribution to the field by providing a snapshot estimate of the current situation faced by one poorly studied community in an endemic area.

The fundamental goal of this study is to characterize the prevalence of helminthiases among school-age children in Kabuwoko Parish, Kirumba Sub-County of Rakai District, Uganda. Embedded within this goal are the objectives of estimating prevalence and intensity of each helminth, in addition to commenting on associated

demographic risk factors and other noteworthy patterns of disease distribution. The assessment of treatment effectiveness hopes to shed light on the relevance of this concern to the community of interest, with a broader goal of hinting at the potential extent of drug resistance and supporting the need for the development of alternative treatment options. The data collected may be useful for the Ministry of Health of Uganda, which suffers from resource limitations that have prevented adequate surveillance in this region. Finally, the structure of this study hopes to inspire future projects that capitalize on the mutually beneficial process of outsourcing surveillance activities to students with significant resource networks. Such a system allows for students to engage in a meaningful international research project, while relieving resource-stained agencies of the burden of conducting routine surveillance. In this case, the Ministry of Health of Uganda will have access to surveillance data they are as of yet unable to collect, while the student will receive considerable field research experience.

It is expected that helminths will be found in this community, as anecdotal evidence from community members and prevalence data from neighboring districts suggest that helminths survive well in the region. Overall treatment effectiveness is expected to be high, as anecdotal reports suggest that treatment has rarely been made available. Hypothesized demographic risk factors include poor personal hygiene and sanitation habits, the possession and use of shoes, and the possession of animals, as these have all been reported to be risk factors in previous studies (Bethony et al. 2006, Brooker et al. 2008, Humphries et al. 2011).

BACKGROUND

Disease caused by soil-transmitted nematodes and schistosomes account for over 40% of the global NTD burden (Krauth et al. 2012). The establishment of national and international control programs since the turn of the millennium has brought helminthiases into scientific focus, prompting a significant increase in research into the biology, etiology, and control of these diseases. Increased and integrated understanding of these diseases has contributed to a multifaceted global control strategy that capitalizes on helminth biology, infrastructural and behavioral risk factors, and chemotherapeutic interventions (Kabatereine et al. 2011).

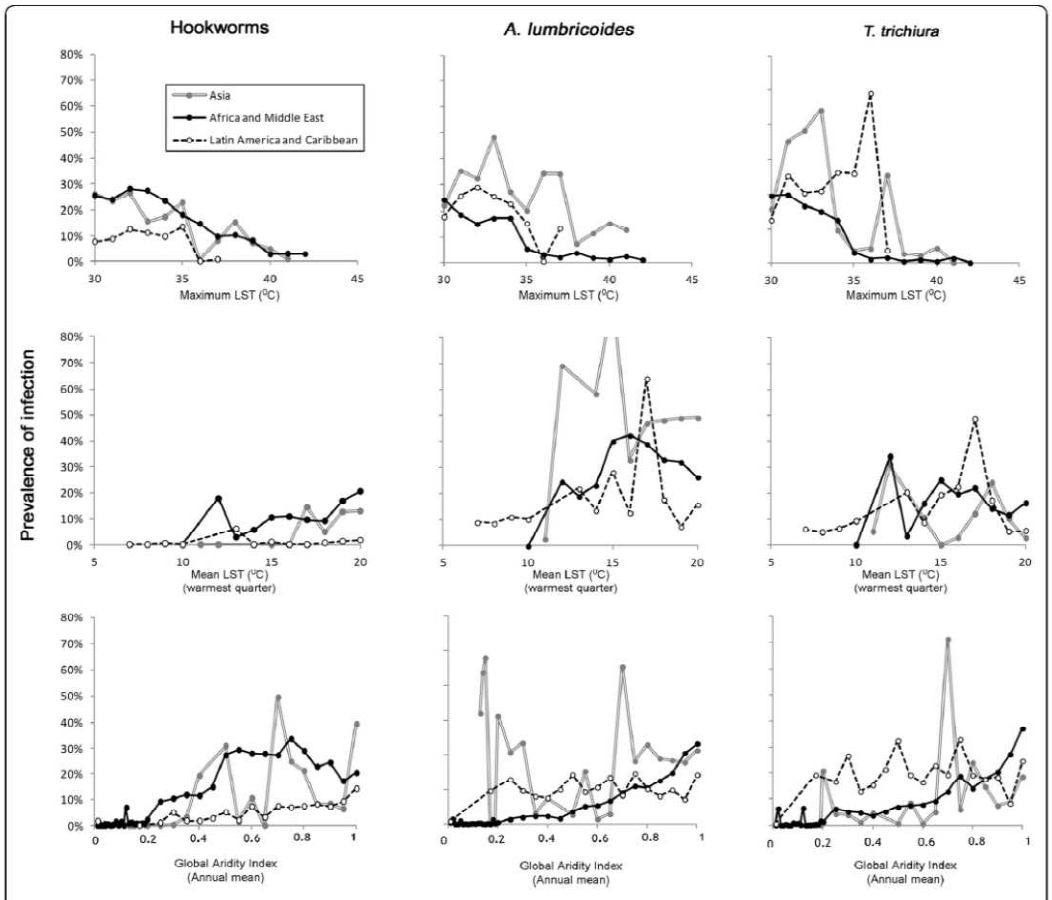


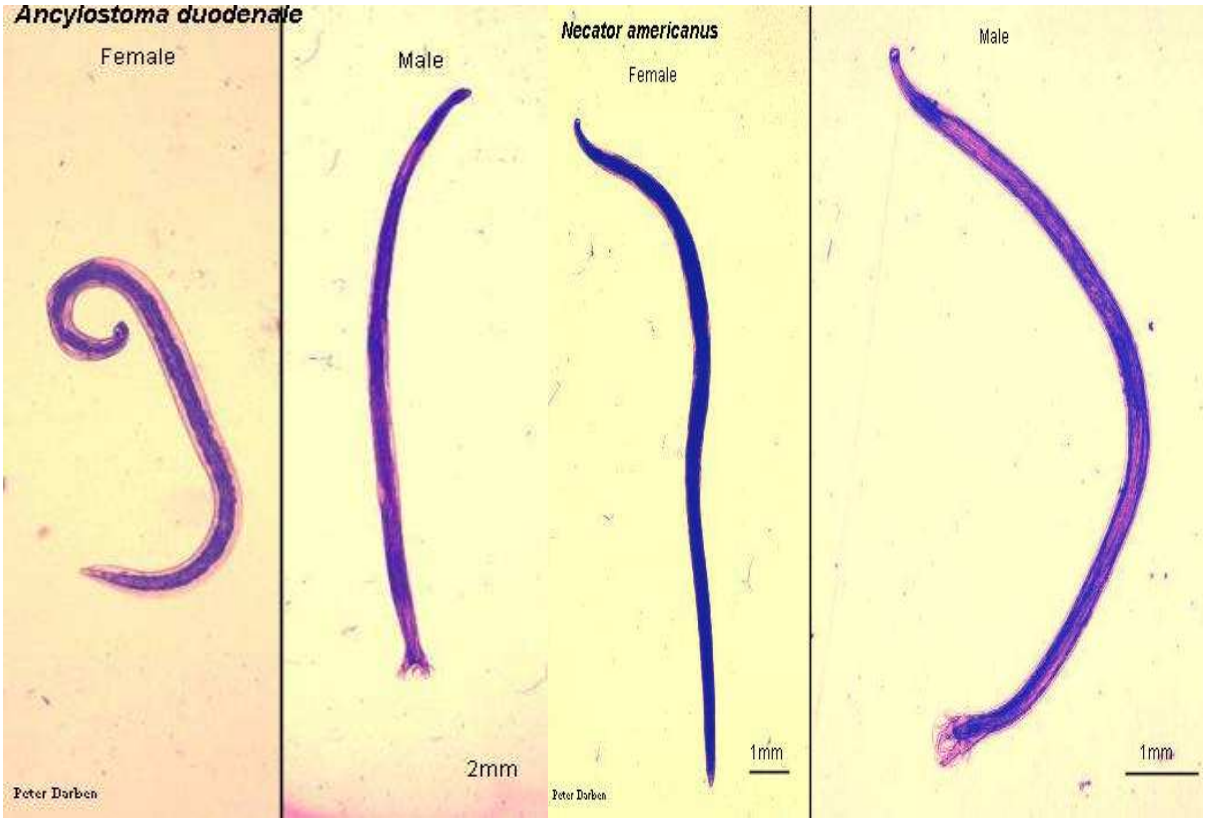
Figure 2 Relationship between climatic factors and prevalence of STH infection. Climatic factors estimated from interpolated weather station data, and prevalence of STH infection from 4,840 unique survey locations world-wide, stratified by world region. Estimates were derived for each survey point location and data for locations experiencing the same temperature are averaged for presentation.

Soil-transmitted nematodes thrive in tropical climates, which in part explains their predominance in tropical regions of the world. (Pullan and Brooker 2012)

Hookworm Disease

Between 576 and 740 million people are infected with hookworms worldwide, such that the disease outranks fellow NTDs African trypanosomiasis, Dengue fever, Chagas disease, leprosy, and schistosomiasis in DALYs (Loukas et al. 2005, Bethony et al. 2006). Over 44 million pregnant women are infected, 7.5 million of whom reside in sub-Saharan Africa (Kabatereine et al. 2005). Hookworm disease is endemic throughout Uganda (Brooker et al. 2004). Both infection prevalence and intensity appear to increase with age in endemic areas (Pullan et al. 2010).

Ancylostomatidae, the family of strongyle nematodes, contains 18 genera capable of parasitizing a wide range of mammalian hosts to produce hookworm disease (Loukas et al. 2005). *Necator americanus* is the dominant species responsible for human hookworm infection, though *Ancylostoma duodenale* also boasts a wide distribution (Hotez et al. 2004). Though these two species are primarily responsible for hookworm infection in humans, several zoonotic species are capable of causing minor infection in humans. *A. ceylanicum*, *A. caninum*, and *A. braziliense*, which typically infect cats and dogs, can cause minor cutaneous symptoms and eosinophilia in humans, but do not result in egg-bearing infections (Kabatereine et al. 2005).



Morphology of A. duodenale (left) and N. americanus (right) adults. (http://missinglink.ucsf.edu/lm/virus_and_parasites/hookworm.html)

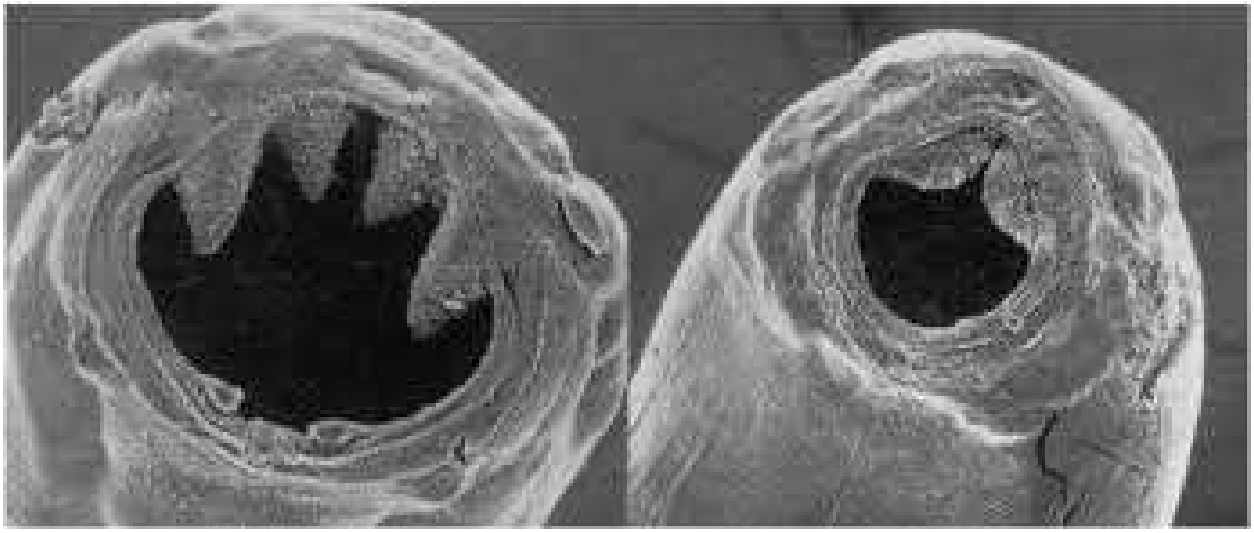
The life cycles of the various hookworm species are nearly identical, with some notable nuances. Transmitted through the fecal-oral and fecal-cutaneous routes, the life cycle of all hookworms begins when eggs are shed into the environment in the feces of an infected individual (Loukas et al. 2005). Under optimal soil conditions, released eggs will hatch into stage one larvae (L1), which are mobile and begin feeding on microorganisms in the soil (Loukas et al. 2005). These larvae will undergo two moults, developing first into stage two larvae (L2), which are also mobile and feeding, and finally into stage three larvae (L3). L3 larvae are encapsulated by a cuticular sheath, and, though still mobile, no longer feed and thus become developmentally arrested (Loukas et al. 2005). These larvae will migrate to higher ground if possible (traveling to the top of

a blade of grass, for example) to maximize the likelihood of contacting a potential host (Loukas et al. 2005). Only the L3 stage is capable of initiating infection (Kabatereine et al. 2005). Vertical transmission has also been hypothesized for *A. duodenale*; it is believed that infected mothers may transmit infective L3 to neonates through colostrum and breast milk, though this transmittion route is not confirmed (Kabatereine et al. 2005).

Both *N. americanus* and *A. duodenale* L3 may attach to the skin of a human upon contact, and will migrate along the surface of the skin in search of a hair follicle for penetration (Loukas et al. 2005). Once the larva has entered the follicle, it will migrate towards a blood or lymphatic capillary and will be passively transported through the circulatory system (Loukas et al. 2005). At this point, the larva has reactivated, and development resumes (Loukas et al. 2005). When the larva reaches the pulmonary microcirculatory system (typically ten days after initial infection), it migrates to the tracheal alveoli, bursts through the alveolar wall into the lumen, and is swept up through the lung cavity in mucus. The larva will then be coughed up, re-swallowed, and transmitted down into the gut (Loukas et al. 2005, Kabatereine et al. 2005). In addition to skin penetration, *A. duodenale* larvae are also capable of causing oral infection, in which the larvae are swallowed and transmitted directly to the gut (Kabatereine et al. 2005).

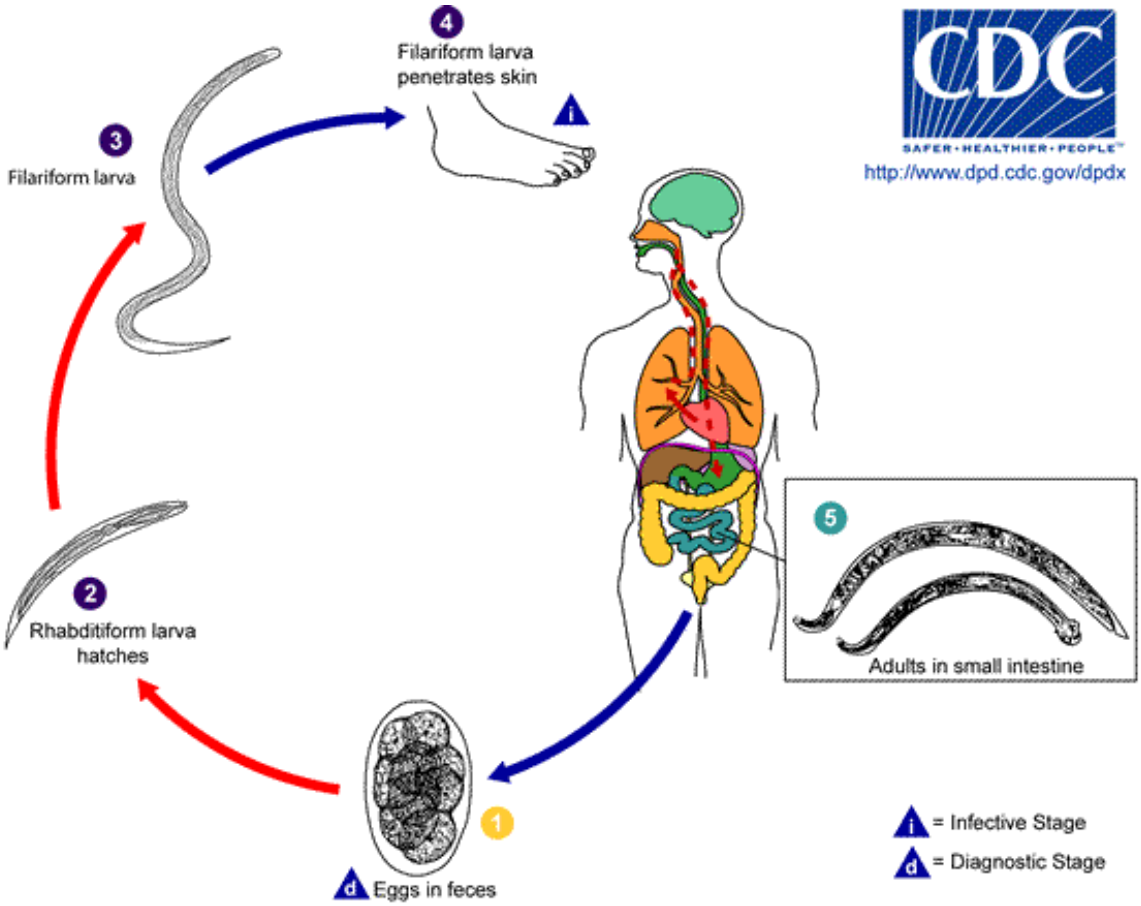
En route, the larva moults once more into a stage four (L4) larva, which now possesses a primordial buccal capsule and a developing genital system (Loukas et al. 2005). Once the small intestine is reached, the larva attaches to microvilli, begins feeding, and develops into an adult (Kabatereine et al. 2005). Adult worms enjoy a fully

formed buccal capsule, which serves primarily to anchor the worm in the upper portion of the small intestine (Bethony et al. 2006). The buccal capsule will contain either teeth (*Ancylostoma* species) or cutting plates (*N. americanus*), and allows the worms to suck up clumps of villi so that they may be stably anchored in the mucosa (Loukas et al. 2005).



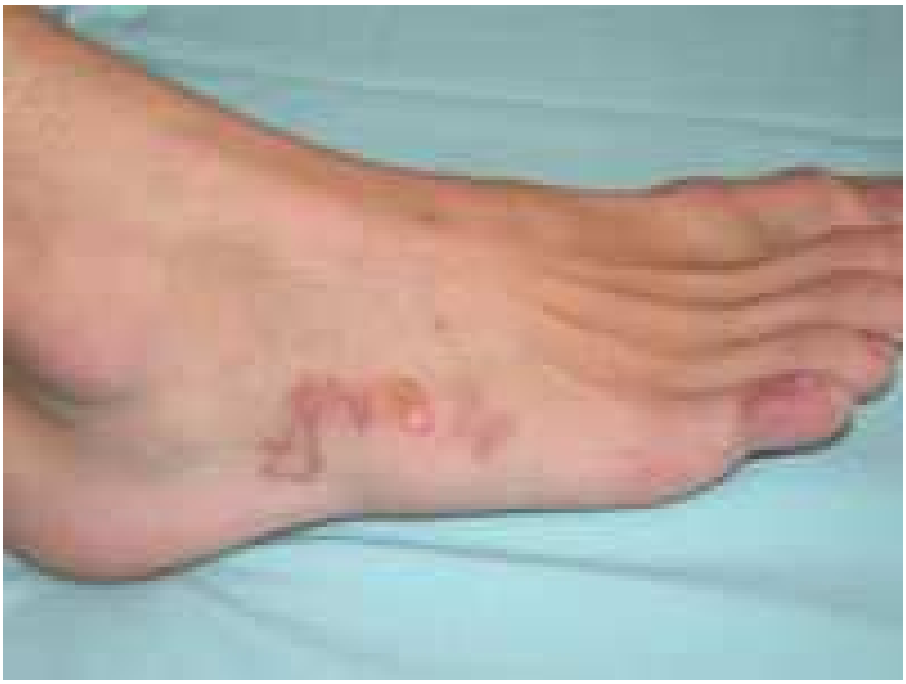
Morphological differences in the buccal capsule of A. duodenale (left) and N. americanus (right). (https://encrypted-tbn0.gstatic.com/images?q=tbn:ANd9GcQYpUFHWciY06qd0Z0I1 jlmu83va3dRBnUZCoTgkKRlsaob8n2y4Q)

The worms feed on blood components such as hemoglobin, releasing proteases and anticoagulant peptides to ensure continuous blood flow and adequate tissue maceration (Loukas et al. 2005). Adult *N. americanus* females are 7-13 mm in length and produce 9,000-10,000 eggs each day after mating and feeding; *A. duodenale* adult females are 8-13 mm long and may produce between 25,000 and 30,000 eggs per day (Bethony et al. 2006). Adults of both species typically survive for 5-7 years in a human host (Bethony et al. 2006).



Hookworm Life Cycle, Centers for Disease Control (http://www.cdc.gov/parasites/hookworm/biology.html)

Though nearly 80% of infections are asymptomatic, symptoms may be seen shortly following initial infection. In highly endemic areas, repeated cutaneous exposure to hookworm larvae may result in a pruritic, erythematous, papular rash, known commonly as “ground itch,” and more formally as cutaneous larva migrans. This symptom occurs as the immune system mounts a response against the antigenic stimulation of penetrative L3 (Bethony et al. 2006).

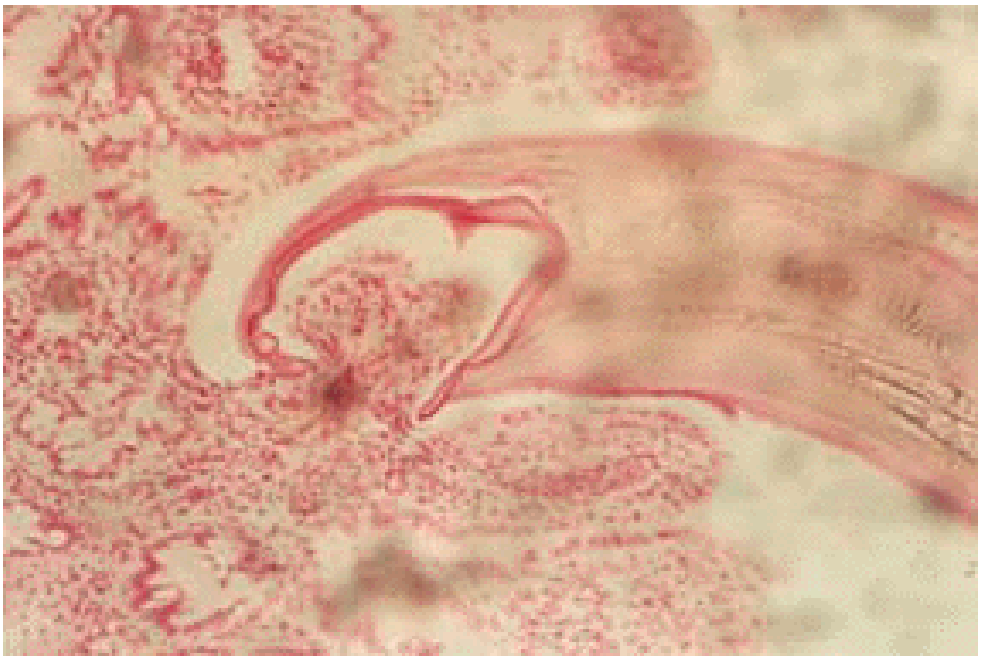


Cutaneous larva migrans caused by hookworm infection. (http://www.dermnetnz.org/arthropods/larva-migrans.html)

Ten days after infection, a cough or sore throat may develop as the hookworm migrates through the lungs and pulmonary vasculature (Kabatereine et al. 2005). In rare cases, the immune response to the parasite in this stage may be so robust that it causes mild pneumonitis lasting up to one month (Kabatereine et al. 2005). When *A. duodenale* infection results from oral contamination, Wakana Disease, which is characterized by nausea, vomiting, pharyngeal irritation, cough, dyspnea, and hoarseness, may result (Kabatereine et al. 2005). Hypothermia severe enough to mask the fever caused by malaria co-infection is also commonly observed (Kabatereine et al. 2005). Eosinophilia is typically seen 5-9 weeks after infection; this reflects the broad antigenic challenge presented by hookworm invasion that prompts a TH2-type immune response (Kabatereine et al. 2005). Interestingly, this tends to wane once adult worms establish in the small intestine, hinting at the deployment of a mechanism of immune suppression (Kabatereine et al. 2005). Chronic epigastric pain, nausea, dyspnea, palpitations,

headache, fatigue, and impotence have also been observed in conjunction with hookworm infection (Kabatereine et al. 2005).

The most significant clinical disease outcomes that result from hookworm infection are due to the mechanical damage caused by the worm's attachment to the intestinal mucosa and its migration through somatic tissues. Though *A. duodenale* typically causes more daily blood loss than *N. americanus*, the intestinal symptoms caused by both species tend to be fairly indistinguishable (Kabatereine et al. 2005). The main disease outcome caused by hookworm infection is iron-deficiency anemia as a direct consequence of unsustainable intestinal blood loss (Kabatereine et al. 2005). Hookworms feed off of the intestinal blood supply and cause additional blood loss due to generalized tissue maceration; this results in anemia when the daily rate of loss exceeds the daily intake and cumulative reserves of iron in the host (Kabatereine et al. 2005). In some cases, this blood loss may also result in hypoalbuminemia, reflecting a net loss in host protein reserves (Kabatereine et al. 2005). Hypoproteinemia may present as anasarca, a condition characterized by extreme general edema, most frequently affecting the face, lower limbs, and belly (Kabatereine et al. 2005). Typically, a worm burden between 40 and 160 worms is required to induce anemia; however, this varies depending on the iron status and nutritional habits of the host, as well as the relative fitness of the worms harbored (Kabatereine et al. 2005). Once anemia develops, a direct correlation can be observed between infection intensity and subsequent reductions in hemoglobin, serum ferritin, and protoporphyrin levels, highlighting the effect hookworms have on blood integrity of the host (Kabatereine et al. 2005).



Adult hookworm attached the intestinal epithelia.
(http://www.path.cam.ac.uk/~schisto/general_parasitology/parasitology_nematode_examples.html)

Chronic hookworm disease can result in lasting, sometimes irreversible, effects that vary with the age and general health status of the host. Retardation of physical growth, as well as profound effects on memory, reasoning ability, and reading comprehension have been associated with hookworm infection, and have a particularly detrimental effect on children, as they are in a dynamic developmental state (Kabatereine et al. 2005). Impaired cognitive development in children harboring hookworm infection has been shown to reduce school attendance and performance, leading to long-term reductions in overall productivity and wage-earning potential (Kabatereine et al. 2005). As children tend to have lower stores of iron than adults, they are especially susceptible to the anemia caused by hookworm infection and the resulting sequelae (Kabatereine et al. 2005). Women of child-bearing age also have notably low iron reserves; hookworm infection has been shown to induce anemia in pregnant women that results in increased maternal mortality, impaired lactation,

premature birth, and low birth weight, all of which increase the risk of morbidity and mortality of the child (Kabatereine et al. 2005). Disease interaction has been noted in individuals co-infected with hookworm and malaria, HIV/AIDS, and/or tuberculosis, though the nature of these interactions is poorly characterized thus far (Pullan et al. 2011).

While research on hookworm disease has been quite extensive, there remain many significant gaps in scientific understanding of the behavior of these helminths, the mechanisms by which they cause disease in humans, and the nature of the immune system interactions therein. Though this parasite has been researched extensively—receiving more attention than many other NTDs—knowledge is limited, and additional research is desperately needed to improve understanding of how this parasite operates to produce disease.

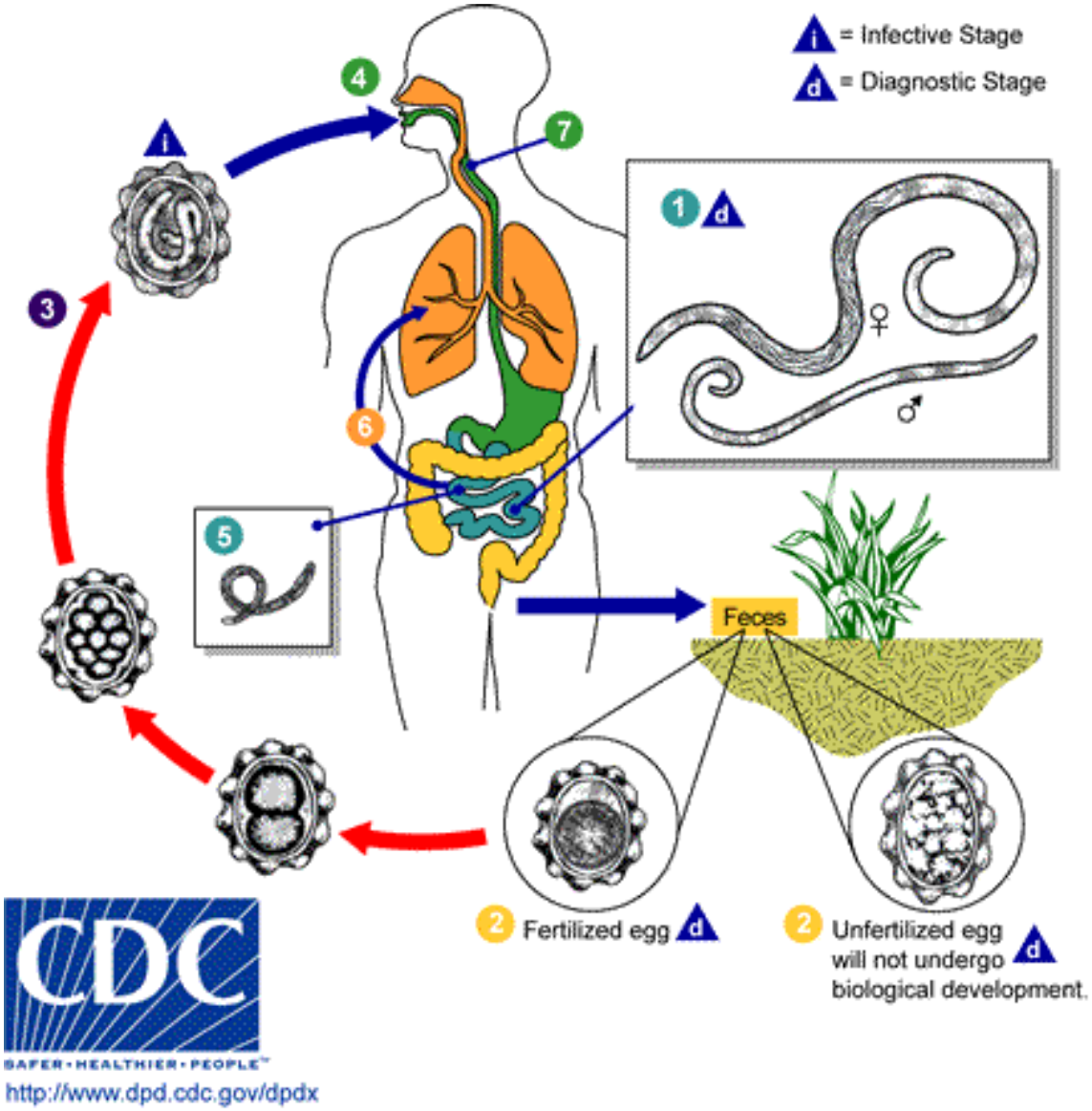
Ascariasis

Between 807 and 1,221 million people in the world are currently infected with *Ascaris lumbricoides*, a parasitic intestinal nematode that is the causative agent of ascariasis disease (Bethony et al. 2006). Endemic in tropical and subtropical climates worldwide, ascariasis is most common to sub-Saharan Africa, Southeast Asia, and the Pacific Islands, though significant infection prevalence is also observed in Latin America, the Middle East, and China (Bethony et al. 2006). Seventy-three percent of infections occur in Asia and 12% occur in Africa (O'Lorcain and Holland 2000). The average prevalence in Uganda is low, hovering around 5-10%, though estimates vary widely by region, and ranges from 0% to nearly 90% (Kabatereine et al. 2005). Infection

is more common along coastal regions, where moisture and temperature conditions are conducive to egg and larval survival (Kabatereine et al. 2005). *A. lumbricoides* is the largest nematode known to parasitize the human intestine (Centers for Disease Control).

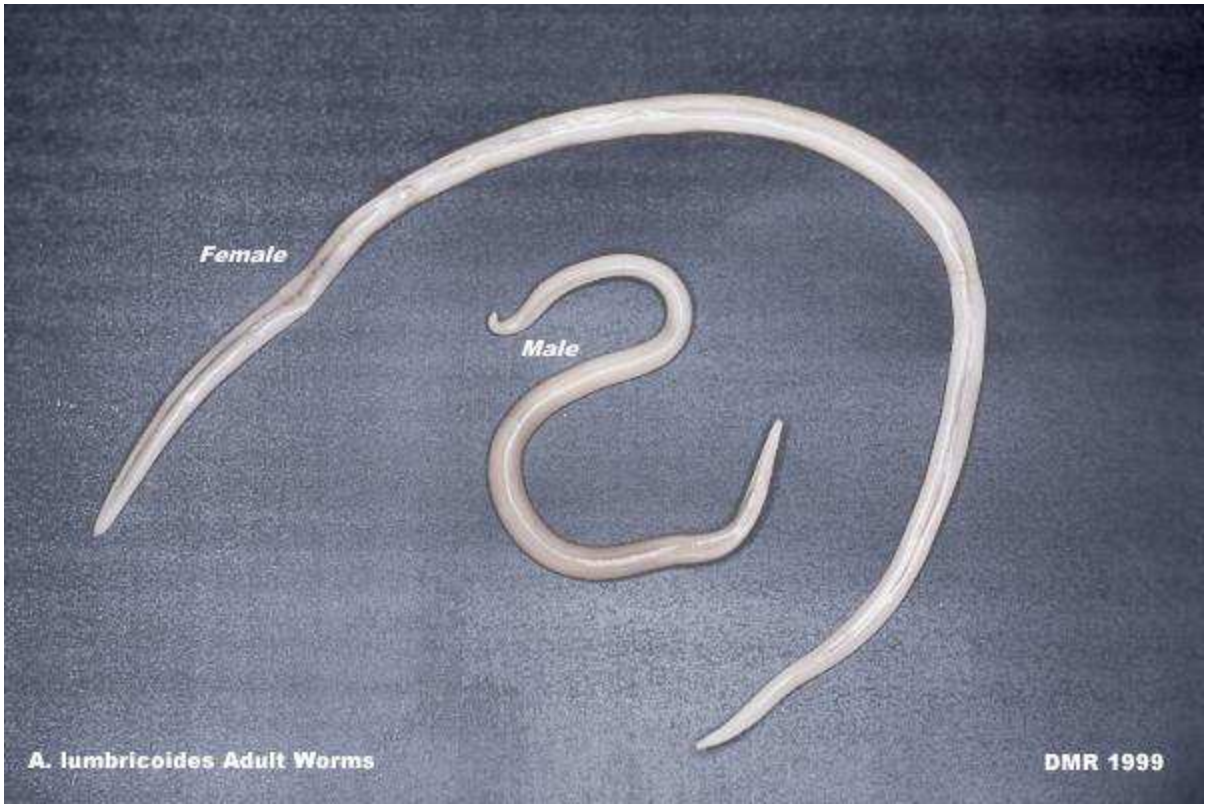
Like other intestinal nematodes, *A. lumbricoides* begins its life cycle in the egg stage and is shed in the feces of an infected host. Both fertilized and unfertilized eggs may be shed into the environment, but only fertilized eggs are capable of causing infection. In the event that eggs are shed in soil, and under appropriate temperature and moisture conditions, fertile eggs will embryonate and become infective within an average of 18 days (Centers for Disease Control). If ambient humidity is too low, or if the temperature is too high, fertilized eggs will not embryonate and will not be capable of developing into larvae upon infection (Brooker et al. 2004, Mascarini-Serra 2011). Even so, *A. lumbricoides* eggs are particularly hardy: their characteristic lipid coating makes them resilient in a variety of environmental conditions, and embryonated eggs may survive for up to 15 years in the environment under adequate conditions (O'Lorcain and Holland 2000). Humans may contact infectious eggs through accidental ingestion of contaminated soil, typically by consuming unwashed vegetables or by placing dirty hands in the mouth (this is more common among children) (Centers for Disease Control). Swallowed eggs hatch into larvae in the duodenum and invade the intestinal mucosa to access the circulatory system (Centers for Disease Control, O'Lorcain and Holland 2000). When the portal vein is reached, the larvae are carried passively by the circulatory system (through a mechanism similar to the migration of hookworm larvae) to the lungs (Centers for Disease Control). Here they mature for 10-14 days, then

penetrate the alveolar wall of the trachea, entering the lumen of the respiratory system (Centers for Disease Control). The larvae then ascend the bronchial tree and throat, and are coughed up and swallowed once more (Centers for Disease Control). This time, when the larvae reach the small intestine, they stop travelling and mature into adults. Many larvae will die en route if they end up in inappropriate tissues, where they can cause a chronic granulomatous immune response that manifests in the creation of scar tissue around the rogue worm (O'Lorcain and Holland 2000).



Life Cycle of A. lumbricoides*, Centers for Disease Control*

A. lumbricoides mature for 2-3 months within the host, and are able to survive as adults for 1-2 years. Like other nematodes, adult females are larger than adult males; the females range in length from 20-35 cm, while the males are just 15-30 cm long. After mating and feeding, female worms produce an average of 200,000 eggs per day (Centers for Disease Control, Bethony et al. 2006). *A. lumbricoides* may colonize all parts of the small intestine, and feed on digested food contained therein. Unlike hookworm, *A. lumbricoides* infection prevalence and intensity tend to peak in childhood; the majority of infections in endemic areas are among those 5-15 years of age (Bethony et al. 2006).



Adult male and female Ascaris lumbricoides*. (http://www.practicalscience.com/alworm2.jpg)*

The clinical manifestations of ascariasis vary widely. The majority of infections, particularly light ones, are asymptomatic; only 8-15% of cases have associated morbidity (O'Lorcain and Holland 2000). The first symptom to appear is a nonspecific cough and verminous pneumonia, which typically results from the worms migrating through the respiratory tract (Centers for Disease Control). Other symptoms may result from mechanical blockage caused by the worms, and include abdominal pain and distention, as well as intestinal obstruction with a variety of clinical outcomes. For example, bowel infarction and/or intestinal perforation may result if a bolus of worms obstructs the intestine. This is particularly common in children, whose intestines tend to have a smaller lumen diameter (Bethony et al. 2006).

Child with abdominal distention caused by heavy ascariasis infection. (http://endtheneglect.org/2009/12/night-1-ascariasis/)

Worms may also become lodged in the appendix (resulting in disease manifestations indistinguishable from appendicitis) and the bile duct (causing biliary colic, cholecystitis, cholangitis, pancreatitis, and hepatic abscess) (Bethony et al. 2006). When a host becomes feverish due to another ailment, *A. lumbricoides* adults may migrate out of the body via the anus or nasopharygeal openings (Bethony et al. 2006). Though mechanical complications of ascariasis are rare, there is potential for serious negative health outcomes when the worms obstruct critical transport systems in their human hosts.

A bolus of Ascaris lumbricoides*.*
(http://www.organicnutrition.co.uk/faqs/faq-detoxing.htm)

Symptomatic ascariasis may also cause digestive and nutrition problems, which can have more serious long-term implications. Both lactose intolerance and vitamin A malabsorption are common symptoms of disease; these have been associated with impaired growth and physical fitness in addition to reduced school attendance (Bethony

et al. 2006). Because of these growth impairments, *A. lumbricoides* may contribute to long-term reductions in school completion and subsequent career success and lifetime productivity.

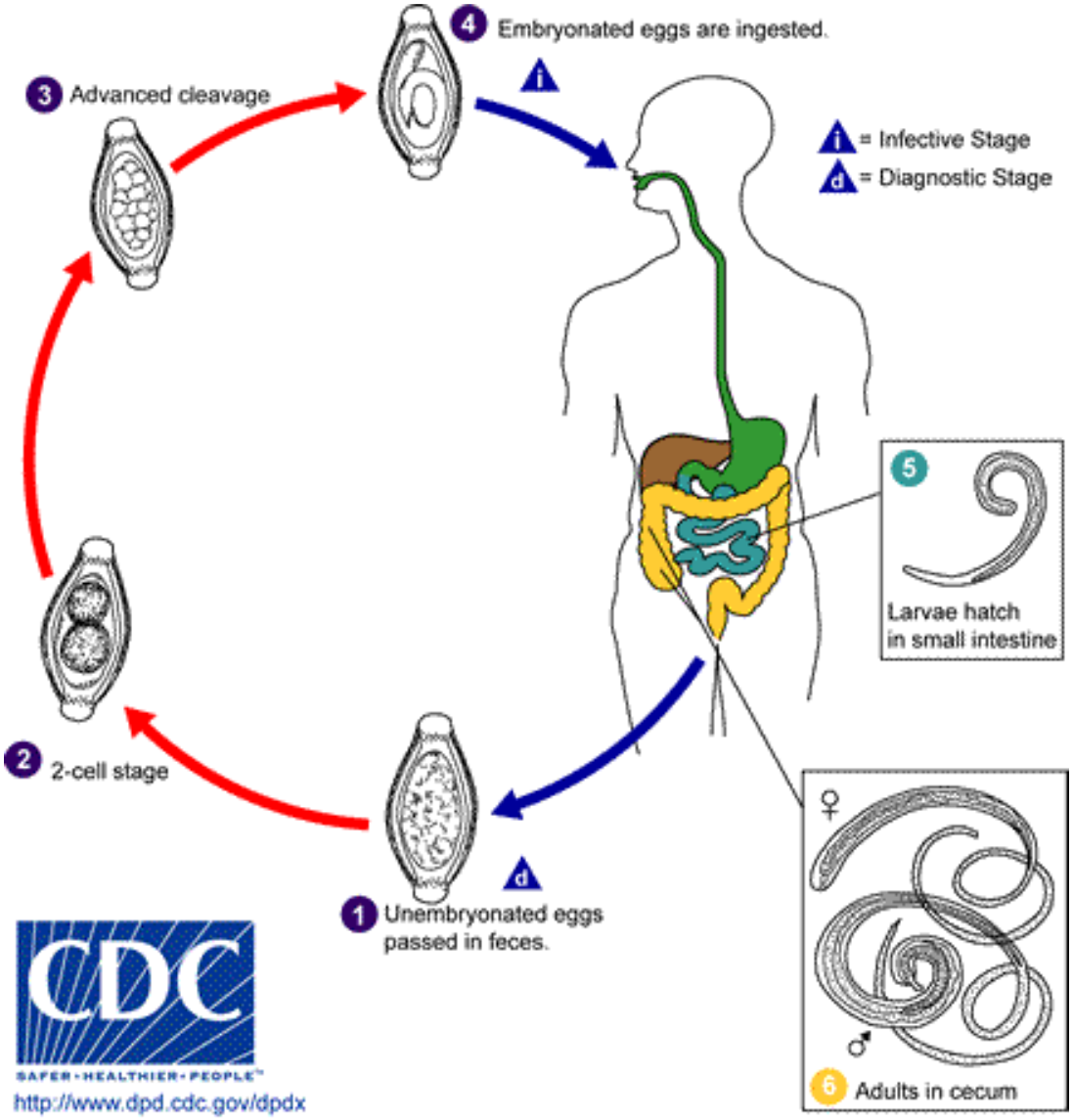
Trichuriasis

Trichuriasis, the disease caused by infection with *Trichuris trichiura*, affects between 604 and 795 million individuals worldwide, and is the third most common roundworm to parasitize humans (Bethony et al. 2006, Centers for Disease Control). Though humans serve as the primary host of *T. trichiura*, pigs, lemurs, and monkeys may also harbor infection (Stephenson et al. 2000). The global distribution of *T. trichiura* (also known as whipworm) is similar to that of *A. lumbricoides*; the majority of infections occur in Asia (over 400 million), with significant but lower infection rates occurring in Africa (over 160 million) and other tropical regions (Stephenson et al. 2000, Bethony et al. 2006). The prevalence of *T. trichiura* in Uganda is highly variable; the estimated average prevalence is approximately 5%, though estimates range from 0% to 70% across the different districts (Kabatereine et al. 2005). Within Uganda, *T. trichiura* is particularly prevalent along the shores of Lake Victoria near Masaka and Rakai Districts, where the soil composition is mineral hydromorphic and thus exceptionally conducive to the development of this nematode (Kabatereine et al. 2011). Like ascariasis, the burden of trichuriasis is borne primarily by children between the ages of 5 and 15; with increasing age thereafter, infection prevalence and intensity appear to decline (Bethony et al. 2006).

Adult male and female Trichuris trichiura. *(http://plpnemweb.ucdavis.edu/nemaplex/images/ttrichmf.jpg)*

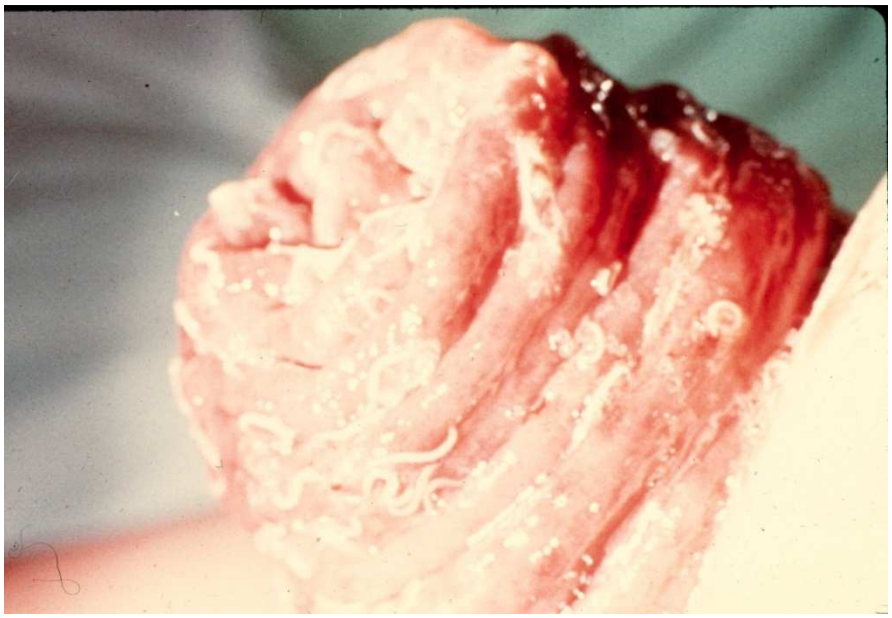
Unembryonated *T. trichiura* eggs enter the environment when passed in the feces of an infected host (Centers for Disease Control). When environmental conditions are appropriate, shed eggs will develop into a 2-cell stage, followed by an advanced cleavage stage, and finally by embryonation, producing infectious eggs within 15-30 days (Centers for Disease Control). Infectious eggs may then be ingested with contaminated fruits or vegetables that have not been adequately washed or peeled, or by the accidental ingestion of contaminated soil on the hands (Centers for Disease Control). Once ingested, embryonated eggs will hatch in the small intestine, releasing larvae that mature in the gastrointestinal tract and establish as adults in the caecum or ascending colon (Centers for Disease Control). The thinner, anterior end of the worm will lodge within the intestinal epithelia while the wider, posterior end remains free in the

intestinal lumen, such that the worms are fixed to their location (similar to the aforementioned hookworm species) (Bethony et al. 2006). Thus, the adult whipworm is both an intracellular and an extracellular parasite (Bethony et al. 2006). Adult female worms begin oviposition 60-70 days after infection and typically shed between 3,000 and 5,000 eggs each day, though daily depositions of up to 20,000 eggs have been observed (Centers for Disease Control, Bethony et al. 2006). Adult worms are between 3 and 5 cm long and may survive for 1-2 years in the colon (Bethony et al. 2006).



Life Cycle of Trichuris trichiura*, Centers for Disease Control*

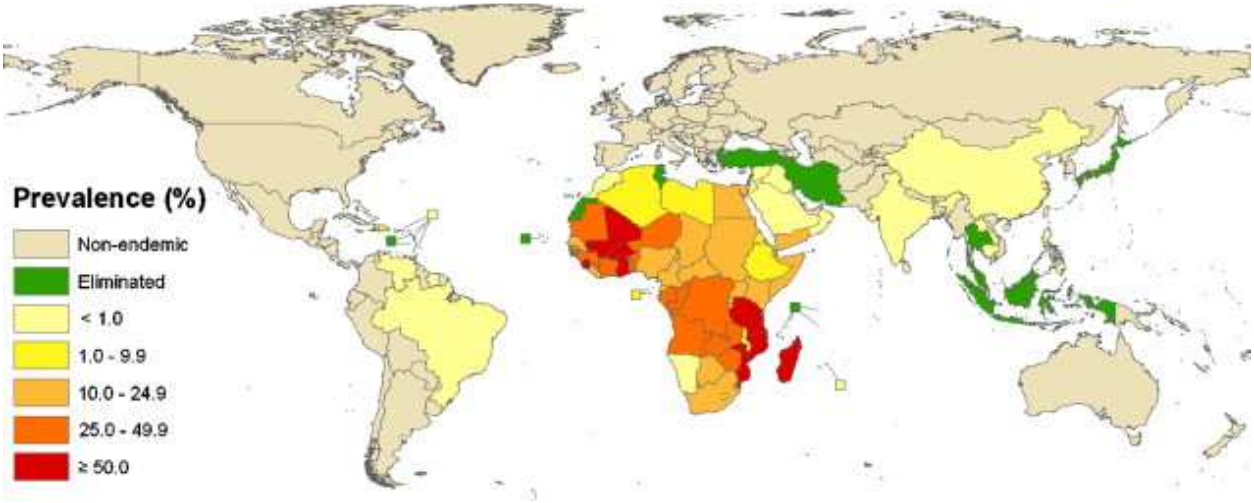
Light *T. trichiura* infections tend to be asymptomatic, and heavy infections are typically associated with increasingly severe disease. Inflammation at the site of attachment is common; this results in broad colitis when many worms are present (Bethony et al. 2006). Over time, this colitis may develop into a syndrome that is symptomatically similar to irritable bowel syndrome, with such characteristic sequelae as chronic abdominal pain, anemia and resulting growth impairment, and finger clubbing (Bethony et al. 2006). Trichuris dysentery syndrome (TDS) may also develop over time, and is defined by chronic dysentery and resulting rectal prolapse (Bethony et al. 2006). The frequency with which painful stools that contain mucus, blood, and water are passed tends to correlate directly to infection intensity (Centers for Disease Control). Infection with *T. trichiura* tends to result in more severe disease than does infection with the other soil-transmitted nematodes of interest, though its geographical distribution and health burden are notably less extensive.



Rectal prolapse caused by heavy T. trichiura *infection.*
(http://www.stanford.edu/class/humbio103/ParaSites2002/trichuriasis/trichsymptoms.html)

Schistosomiasis

Schistosomiasis is caused by infection with one of the five schistosome species: *S. mansoni*, *S. haematobium*, *S. japonicum*, *S. mekongi*, and *S. intercalatum*. *S. mansoni* has the broadest distribution; this species is endemic throughout parts of Africa (particularly the Great Lakes region and the Nile River Valley), South America (Brazil, Venezuela, and Suriname), and the Caribbean (Dominican Republic, Guadeloupe, Martinique, and Saint Lucia) (Centers for Disease Control). The second most prevalent species, *S. Haematobium*, is found in the Nile River Valley as well, and also in North Africa and parts of the Middle East (Centers for Disease Control). *S. japonicum* is endemic to Indonesia and parts of China and Southeast Asia (Centers for Disease Control). *S. mekongi*, found in Cambodia and Laos, and *S. intercalatum*, found in parts of Central and West Africa, are less common (Centers for Disease Control). All organisms responsible for schistosomiasis are digenetic trematodes (Centers for Disease Control). In addition to the species mentioned here, species that typically parasitize birds and mammals may cause cutaneous disease in humans, but will not successfully establish infection (Centers for Disease Control).



Global Distribution of Schistosomiasis. (http://www.infectionlandscapes.org/2012/06/schistosomiasis.html)

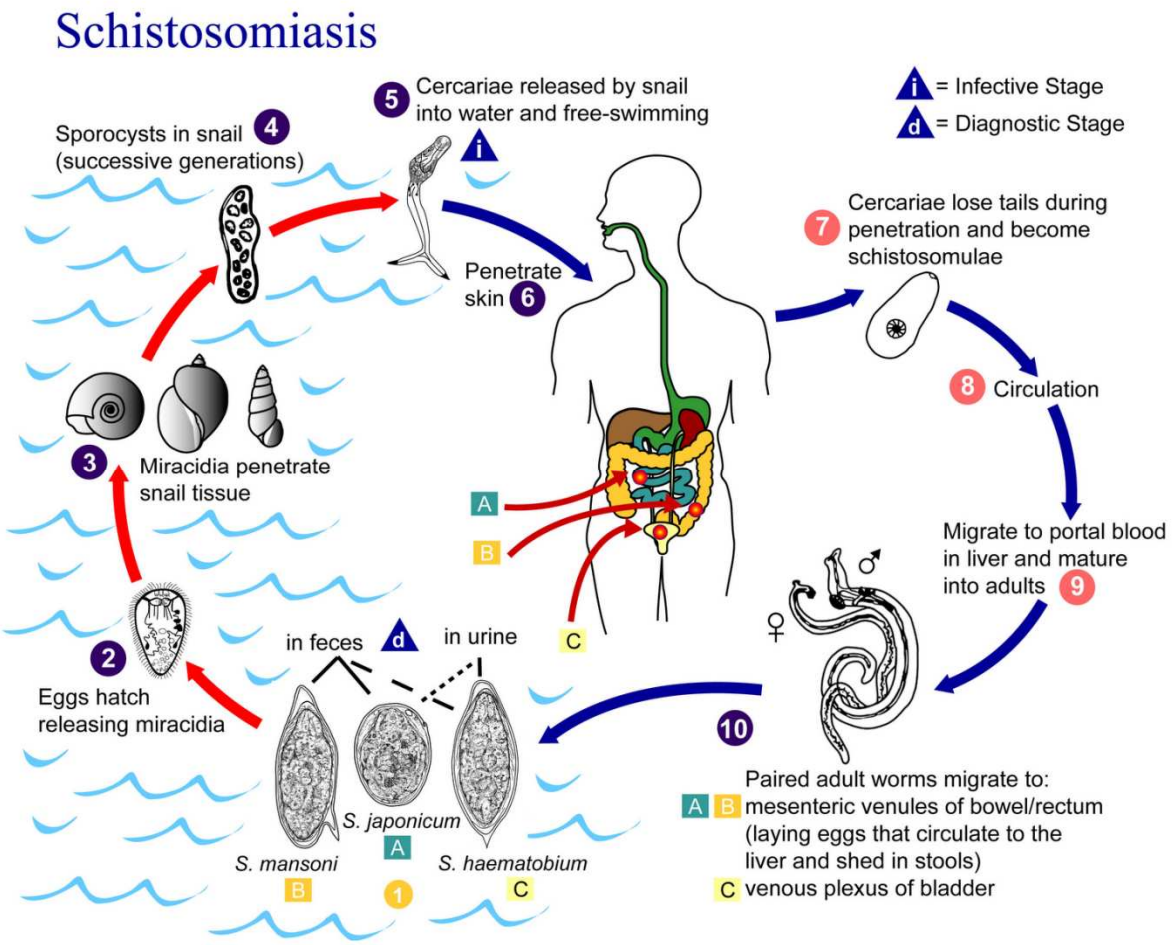
Over 200 million people harbor schistosomal infections worldwide (Hodges et al. 2012). In Uganda, *S. mansoni* is believed to be exclusively responsible for all schistosomal infections, and is found primarily around large rivers and lakes such as Lake Victoria (Kabatereine et al. 2011). Four million people within the country are infected, and nearly 17 million are at risk (Kabatereine et al. 2011). Uganda is supported by the Schistosomiasis Control Initiative, which works closely with the nation's own Vector Control Division to combat the disease (Kabatereine et al. 2011). Infection with *S. mansoni* is rarely observed in Rakai District (Kabatereine et al. 2011).

The life cycle of *S. mansoni* is similar to those of other related blood flukes and begins with the shedding of eggs in the feces and urine of an infected human host. If temperature and light conditions are optimal, eggs shed into freshwater will hatch, releasing motile miracidia. Hatched miracidia swim in search of a viable host; freshwater snails of the genus *Biomphalaria* are optimal hosts during this stage of the parasitic life cycle.

Biomphalaria snail. (http://www.infectionlandscapes.org/2012/06/schistosomiasis.html)

Entering the snail's foot, miracidia develop into sporocysts, undergoing rapid multiplication and two developmental transformations, ultimately presenting as cercariae, the infective state of the schistosome. Infectious cercariae are released from the snail and into the surrounding water during daylight hours, which conveniently overlaps with the time during which their next host (a human) is most likely to also be in the water. Once released, cercariae can survive for up to 48 hours in freshwater, actively searching for a human host.

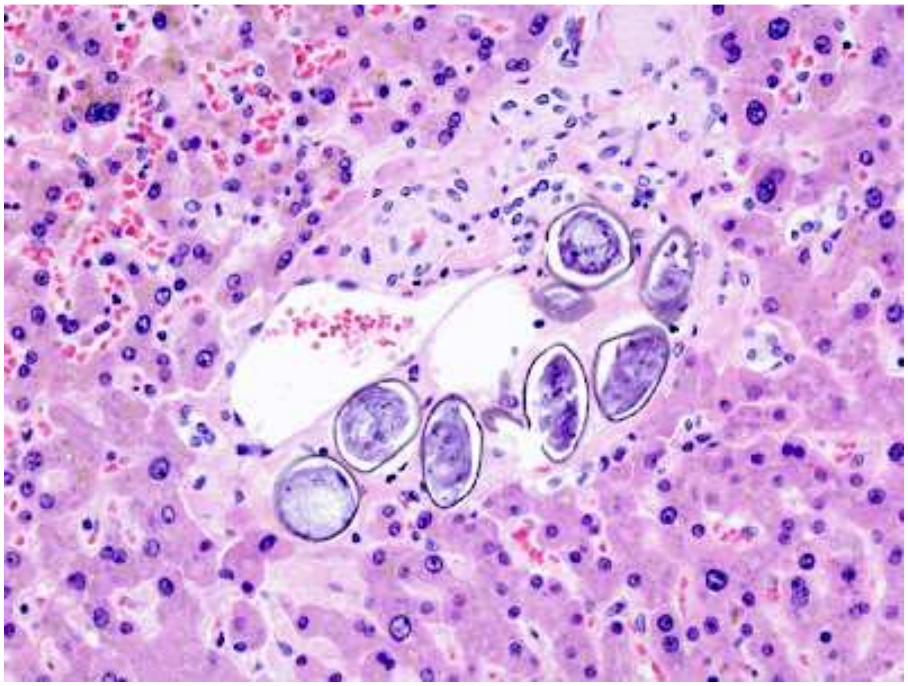
Upon contact with human skin, the cercariae shed their bifurcated tail, attach to the skin, and then creep along the surface in search of a possible point of entry, typically a hair follicle. Penetrative cercariae are classified as schistosomal larvae, and are termed schistosomulae. Each schistosomula may remain in the skin for several days before entering the circulatory system; the ultimate site of residence in the hepatoportal circulatory region is reached 15 days after infection. All schistosomulae develop into sexually mature adults upon contact with a larva of the opposite sex, and begin producing eggs at least 32 days after entering the host. Female worms will deposit eggs in small venules of the hepatoportal and perivesical systems; the eggs then migrate towards the intestinal lumen and are expelled intermittently and in small quantities into the environment through feces (Centers for Disease Control).



Life Cycle of Schistosoma sp., Centers for Disease Control

Symptomatic disease following infection by a schistosome is a result of the host immune response to the eggs, rather than any toxic mechanism induced by the worms (Centers for Disease Control). Even so, disease can be severe, and both acute and chronic symptoms can develop. Acute symptoms include a skin rash that may develop within days of the initial infection due to antigenic stimulation during cutaneous larval migration, in addition to Katayama Fever, a condition characterized by chills, diarrhea, eosinophilia, cough, and muscle aches as a result of the movement of worms through somatic tissues during the first two months of infection. More chronic symptoms may include abdominal pain, hepatosplenomegaly, and blood in the urine and stool as the

worms deposit eggs that initiate a prolonged immune response. Long-term infections can result in chronic anemia, eventually leading to malnutrition and cognitive impairment. The unintentional deposition of eggs throughout somatic tissues has the potential to induce a number of complications, including hepatic perisinusoidal egg granulomas, Symmers' pipe stem periportal fibrosis, portal hypertension, and even embolic egg granulomas in the brain or spinal cord. Over time, repeated infection may result in permanent and severe damage to the liver, intestine, spleen, lungs, and bladder due to excessive scarring, and has been linked to the development of bladder cancer. Schistosomiasis can range from asymptomatic infection to one resulting in death; the propensity for symptomatic infection remains poorly understood (Centers for Disease Control).



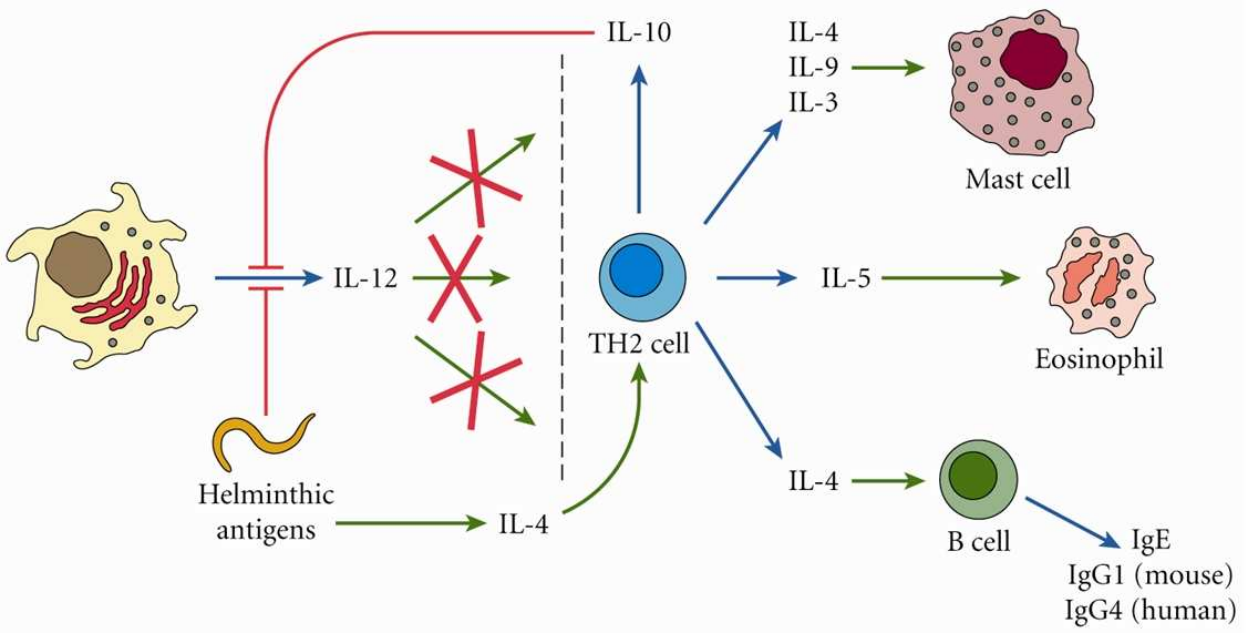
Granulomatous immune response to embedded Schistosoma mansoni *eggs. (http://www.infectionlandscapes.org/2012/06/schistosomiasis.html)*

Immunomodulation

Interactions between parasitic helminths and the human immune system are complex, reactive, and fluid—a phenomenon that is consistent with the reality that these parasites and their human hosts have coevolved for many, many years. The response mounted is exceptionally multifaceted, as helminths are responsible for a range of antigenic stimulation that stems from their ability (and need) to persist within the host in several different forms (as eggs, larvae, and adults). The fact that many of these helminths are nonetheless capable of establishing chronic infection—often persisting for a year or more—suggests that the coevolution of these parasites and their hosts has resulted in a uniquely harmonized system of immunosuppression and immunomodulation that allows the parasites to thrive while minimizing damage to their host.

A T_H2 adaptive immune response is one of the immunological hallmarks of helminth infection and constitutes the host's primary defense mechanism against these pathogens (Bethony et al. 2006). This response begins with naïve CD4+ cells differentiating into the T_H2 subtype following stimulation by an antigen presenting cell (APC) that has contacted helminth antigen; these cells go on to secrete IL-4, IL-5, and IL-13, resulting in the activation of mast cells, plasma cells specific for parasite-neutralizing IgG4 antibody, eosinophils, and tissue-repairing macrophages (Abbas and Lichtman 2009). The activated leukocytes will then release a wave of toxic granules to attack the helminth, and the macrophages will work to minimize damage to the surrounding tissue (Abbas and Lichtman 2009). In theory, this multifaceted, robust, and

expertly coordinated response should successfully control attempted colonization by a parasitic helminth.

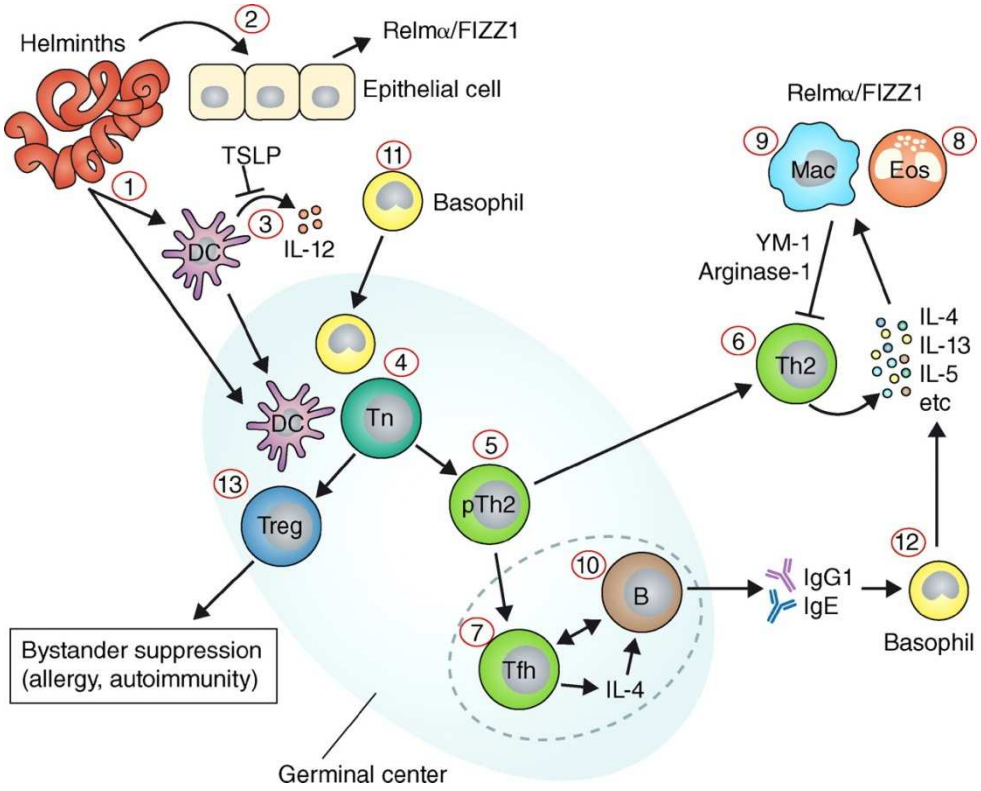


TH2 response to helminth infection. (http://www.ppdictionary.com/parasites_1.htm)

However, these helminths often persist, suggesting an additional level of complexity to the immune response and counterattack from the parasite. Though research on the subject is limited, there is evidence that helminths are capable of "distracting" the human immune system, effectively diluting the T_H2 response and diverting immunological resources to less effective avenues of attack. Helminth antigens may bind to IgG1, IgG4, IgM, IgD, and IgA antibodies; the lack of specificity of this antibody affinity suggests that the infection induces both a T_H1 and a T_H2 response (McSorley and Loukas 2010). This results in a mixed cytokine response that is beneficial for the helminth, as the presence of integrated cytokine feedback mechanisms dilutes the strength of any one response (Loukas et al. 2005). The human immune system is capable of responding to many different pathogens using a number

of distinct cascading mechanisms; helminth infection prevents any of these from becoming too successful by providing nonspecific activation of several at once.

In addition to diluting the effectiveness of the T_H2 response by also inducing a mostly ineffectual T_H1 response, there also seems to be evidence that parasitic helminths prompt excessive activity from regulatory T cells, which serve to limit most immunological activity (Loukas et al. 2005). This is evidenced by an otherwise unexplained elevation in circulating IL-10, a cytokine typically secreted by T_{REG} cells (Loukas et al. 2005). While T_{REG} cells ordinarily serve to benefit the host by minimizing excessive damage to host tissues caused by overzealous activated immune cells, in this case it serves to benefit the helminths, which, by co-opting this immunological check system, prevent the human immune system from mounting a response sufficient to kill the worms (Loukas et al. 2005). The extreme activation of regulatory T cells has effects beyond the helminth infection; chronically infected individuals have been shown to exhibit hypo-responsiveness to immunological challenge by other infections as well, highlighting the immunosuppressive actions of the helminths (Loukas et al. 2005). Such a finding has significant implications for co-infection of individuals harboring helminths, as these individuals are less likely to be able to successfully combat these additional pathogens.



General immunomodulatory hallmarks of helminth infection. (Maizels et al. 2009)

In addition to these hallmark mechanisms, several other immunologically resistant factors have been identified in specific helminths and specific stages of helminth development within the host. *A. lumbricoides*, for example, has been shown to secrete a pepsin inhibitor (PI-3) that protects the worms from digestion in the highly acidic environment of the stomach (Bethony et al. 2006). By inhibiting pepsin, the vulnerable larvae are able to survive stomach digestion, allowing them to safely reach and colonize the small intestine. These worms are also known to secrete glycoconjugates that bind to phosphorylcholine to suppress lymphocyte proliferation, an act which antagonizes the adaptive immune response against the infection (Bethony et al. 2006). TsMIF, a compound secreted by *T. trichiura*, inhibits the migration of peripheral blood mononuclear (PMN) cells by competing with macrophage inhibitory factors; this serves the same purpose of preventing effector cells of the T_H2 response

from reaching their target (Bethony et al. 2006). *T. trichiura* has also been shown to induce a significant distracting T_H1 response. In fact, the excessive presence of TNF-α, which is symbolic of the T_H1 response, actually appears to be severe enough to cause pathological appetite loss and wasting in individuals with heavy *T. trichiura* infections (Stephenson et al. 2000). The chronic inflammation associated with these infections is also believed to play a role in the anemia and stunting observed in many infected individuals (Stephenson et al. 2000).

The immunological profile of hookworm infection has been more thoroughly investigated and is thus better understood than those of other helminth infections. IgE levels increase during L3 migration through the body, suggesting that the T_H2 response is mounted shortly after infection (Kabatereine et al. 2005). Adult hookworms produce T-cell apoptotic factor, an integrin antagonist that prevents proper binding to host CD11b and CD18, and a factor that cleaves eotaxin responsible for monocyte chemotaxis (Hotez et al. 2004). Each of these secretions serves to limit an immunological response, either by blocking or destroying effector molecules and cells (Hotez et al. 2004).

The ability of helminths to control and redirect the immune responses mounted by their hosts is noteworthy. Unfortunately, scientific understanding of the immunological processes at work in helminth infections remains piecemeal, and additional insight is urgently needed to progress efforts to control disease and prevent immunosuppression, subsequent disease, and excess disease susceptibility caused by these parasites.

Risk Factors & Prevention

There is strong scientific and political consensus that helminthiases are largely diseases of rural poverty (Hotez et al. 2004). Risk factors are consistent across the different forms of disease, and relate largely to the strength of sanitation infrastructure, individual hygiene and sanitation behaviors, and the presence and robustness of health education (Centers for Disease Control, Dumba et al. 2008, Mascarini-Serra 2011, O'Lorcain and Holland 2000, Sabatelli et al. 2008). Analysis and differentiation of these risk factors has served as a basis for the design of prevention strategies, many of which highlight risk reduction as a cost-effective preventive measure.

Several studies have been conducted in communities in Uganda to assess risk factors of the various STN diseases and schistosomiasis. A population-based study conducted in 2010 found the main risk factors of STN infection to be older age, previous exposure to anthelmintic treatment, less frequent use of shoes, having a mud floor, and a lower level of education of the head of the household (Pullan et al. 2010). Host genetic factors were not found to be significant, but household clustering did occur, suggesting that transmission often occurs in or around the home (Pullan et al. 2010). The findings of this study suggest that prevention interventions should target behavior in and around the home, perhaps by improving household hygiene, and reinforcing such behavior changes with education.

A smaller study conducted in Luweero District, which is located in central Uganda near Lake Victoria, found similar results. This study highlighted that the main risk factors for helminthiases were poor personal and environmental hygiene practices, naming the method of anal cleaning, latrine maintenance practices, presence of livestock, hand

washing methods, house floor material, accessibility of water, and age and education level of the subject as key determinants of disease outcome (Dumba et al. 2008). This study found that individuals with poorly maintained latrines, either as a product of host behavior or a lack of water resources for maintaining proper hygiene, were significantly more likely to harbor a helminth infection (Dumba et al. 2008). This finding has strong theoretical support, as wet, muddy latrines provide an ideal habitat for the maturation of STN eggs and larvae (Dumba et al. 2008). Thus, when infected individuals deposit eggs in and around the latrine (the spread of which is facilitated by a less hygienic method of anal cleaning, such as sliding), these eggs are more likely to develop into an infectious form (Dumba et al. 2008). The presence of pigs also increases transmission, as they may ingest contaminated human feces and shed eggs once more, thus facilitating the dispersal of potentially infectious eggs (Dumba et al. 2008). The coupling of environmental risk factors (poor latrine maintenance, presence of pigs) with personal hygiene risk factors (inadequate washing) results in a predictable increase in helminthiasis prevalence (Dumba et al. 2008).

The shoreline of Lake Victoria has been a popular site for scientific study of helminthiases, as the populations in this area tend to have elevated prevalence of all four disease types (schistosomiasis, ascariasis, hookworm disease, and trichuriasis) (Kabatereine et al. 2011). A study conducted in these lakeside communities in 2011 suggested that the high population density and permissible water environments facilitate increased transmission of helminth infection, highlighting that this is disproportionately detrimental to island communities, which tend to be more remote and thus have less consistent access to treatment services (Kabatereine et al. 2011). Lake Victoria is one

of the few locations within Uganda in which schistosomiasis is endemic; many outside cases are believed to be the product of travel to this area (Kabatereine et al. 2011). This study corroborated previous findings that socioeconomic status and hygiene behavior are significant risk factors for helminthiasis (Kabatereine et al. 2011).

The consistency of determined risk factors has resulted in a fairly harmonious intervention framework that tends to revolve around reducing environmental risk by improving sanitation infrastructure (typically through the construction of pit latrines), and reducing exposure through behavior modification, typically through the provision of shoes and promotion of proper hygiene (World Health Organization, Centers for Disease Control). The limited success of such interventions suggests that the nuances behind identified risk factors are important determinants of the disease profile. For example, though providing pit latrines may seem like an excellent way to isolate human feces from future human contact, thus interrupting transmission, there is evidence that constructing pit latrines actually does the opposite (Mascarini-Serra, Freeman et al. 2013). If pit latrines are not used or maintained properly, the area surrounding them may become a hotspot for helminth egg and larvae development (Mascarini-Serra 2011). Relatedly, the provision of shoes does not ensure their use; if individuals continue to travel barefoot, especially in and around pit latrines, the provision of shoes is unlikely to result in a reduction in transmission (Mascarini-Serra 2011). When interpreting risk factors for use in designing prevention strategies, it is essential to thoroughly consider the context behind the observations to ensure that the interventions address the fundamental risk in a realistic and effective manner.

Given the complexity surrounding the effective translation of risk factor analysis into worthwhile prevention strategies, alternative methods are desperately needed. Infrastructure-based interventions tend to be costly, and behavior-based interventions run the risk of having low adherence. Short-term interventions do little to control transmission in highly endemic areas, due largely to the long-term viability of helminth eggs in the environment: there are many opportunities for transmission, and prevention will need to continue indefinitely to truly reduce transmission. This suggests a need for long-term therapeutic interventions, or, better yet, the implementation of vaccination control schemes.

Vaccine-based prevention programs are particularly desirable in the control of helminthiases because they circumvent the need to remove environmental reserves of helminths (a daunting and costly task). Vaccines thus pose a single-step control strategy for interrupting infection, disease, and transmission (Bethony et al. 2006). However, funding constraints and a limitation of viable animal models have seriously impeded efforts to develop such vaccines (Bethony et al. 2006).

It has been shown that dogs immunized with radiation-attenuated *A. caninum* are immunologically protected from future disease upon additional helminthic challenge, suggesting that a human vaccine may be possible (Loukas et al. 2005). The absence of observed sterilizing immunity in this and other cases is not of great concern, as the observed reductions in worm burden are still sufficient to prevent disease (Loukas et al. 2005). In light of this finding, much research has been done to elucidate the biochemical pathways at work in helminth parasitization of humans with the hope of identifying viable vaccine targets. Ideal vaccine targets may prevent penetration or migration of helminth

larvae through body tissues, or attack adult larvae at their ultimate site of residence (Loukas et al. 2005). It is believed that a combined vaccine with both such targets would maximize effectiveness in reducing the worm burden.

Observation of partial immunity in canines following vaccination against A. caninum aspartic protease. (Loukas et al. 2005)

To date, hookworm disease is the only helminthiasis for which vaccine candidates in the late stage of development exist. Two potential vaccines, one which targets Ancylostoma secreted protein-2 of *N. americanus* (Na-ASP-2), and one which targets glutathione S transferase of *N. americanus* (Na-GST-1) are currently undergoing clinical testing in Brazil (Sabatelli et al. 2008). Both of these targets are larval proteins involved in penetration and migration; targeting these proteins would compromise the

ability of *N. americanus* to establish infection in a human host (Loukas et al. 2005). Though research on vaccine candidates for other helminthiases is more limited, understanding of immunological interactions between the host and the parasite may lead to future insights within this realm.

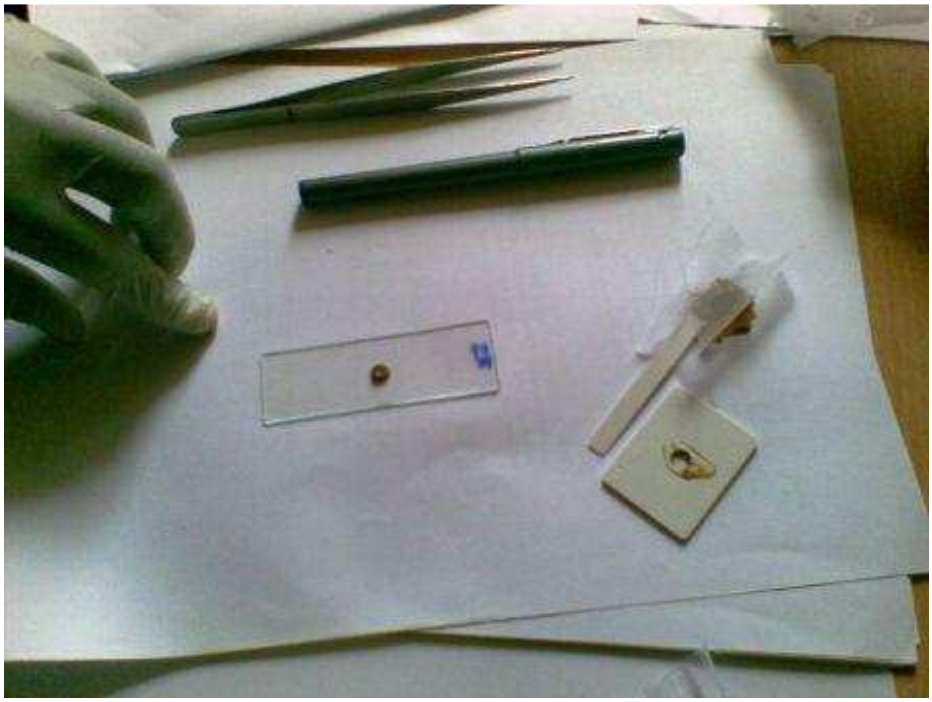
As with all diseases, prevention of helminthiases relies fundamentally on an integration of approaches. The most successful means of controlling helminth infection thus far has been economic development and systematic infrastructural developments, as evidenced by the eradication of many helminthic diseases from many developed countries (Kabatereine et al. 2011). Yet such changes remain out of the reach of many endemic regions, calling for alternative, less resource-intensive strategies. As risk factors become more clearly understood at the community and biological levels, more targeted, cost-effective prevention programs may be designed to purposefully prevent transmission of parasitic helminths.

Diagnosis

The Kato Katz technique, recommended by the WHO, is regarded as the most effective method for detecting helminth infection in rural, resource-poor settings (Tarafder et al. 2010, World Health Organization). The materials required for this technique are simple, and can be purchased together in a Kato Katz kit, which contains templates (plastic pieces with a central hole of known volume), a roll of nylon (to be used as a sieve), a roll of cellophane, and plastic spatulas. Additional required materials include newspaper, a hard surface such as a ceramic tile, glycerol-malachite green

solution, microscope slides and cover slips, and appropriate protection equipment (World Health Organization).

The technique involves filling the template hole with a sieved sample of fresh stool using the spatula, and carefully removing the template such that the molded sample is left on a microscope slide placed underneath the template (Tarafder et al. 2010). The sample is then covered with a piece of cellophane soaked in the green staining solution, and the prepared slide is left to dry for 10-30 minutes before microscopic analysis of its contents (Tarafder et al. 2010). Once the slide is dry, a trained individual will be able to identify and count any eggs that are present.



Preparation of a sample using the Kato Katz technique. (http://www.ihsnet.org.in/SHG/Kato%20Katz%20Method.htm)

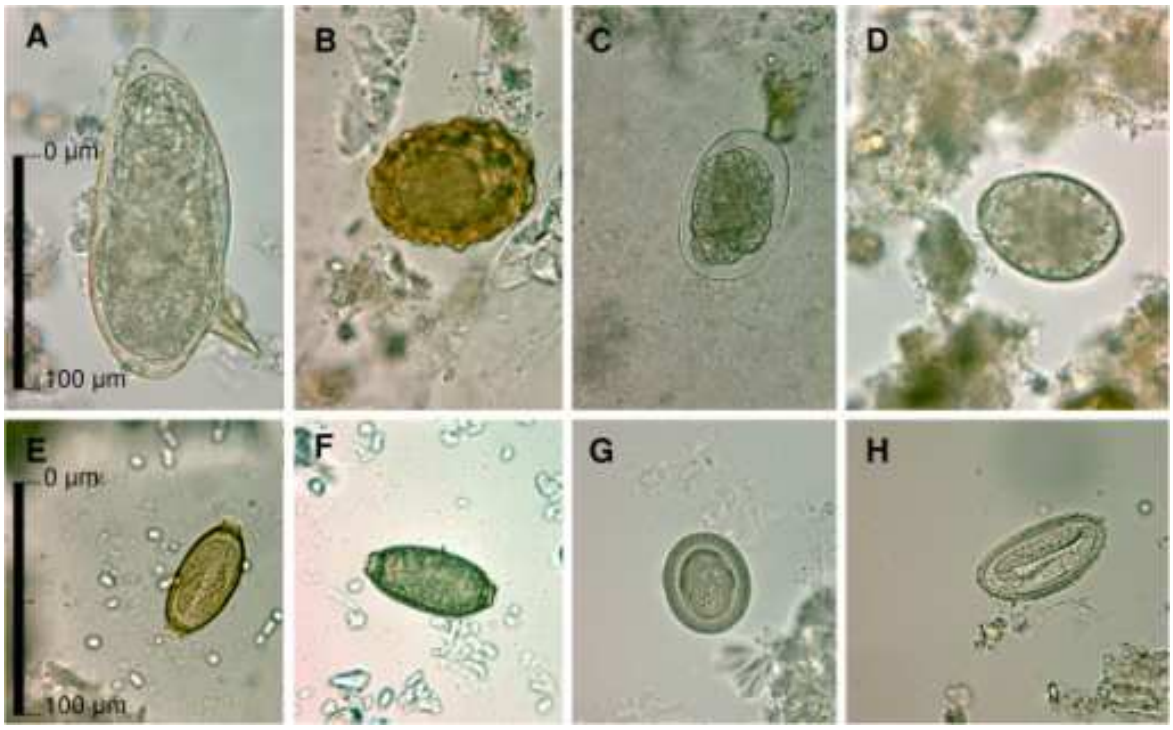
The Kato Katz technique is widely recommended because it is cost-effective, simple, and quick to perform, making it ideal for low resource usage (Habtamu et al. 2011). The main alternative test, FLOTAC, is more sensitive to helminth infections, but

at the cost of being more complex (and therefore requiring a more skilled technician) and more costly, both in terms of time and resources (Habtamu et al. 2011, Speich et al. 2010). A study conducted in Tanzania in 2010 estimated the cost of Kato Katz to be 1.73 USD per test, in contrast to a cost of 2.35 USD for each FLOTAC test, further highlighting the economic favorability of Kato Katz (Speich et al. 2010). The majority of the materials required for Kato Katz come in an inclusive kit; the only major materials not included are microscope slides and the staining solution, which can easily be procured and prepared in a laboratory. The green staining solution is forgiving, and can be produced by a technician with a low level of expertise. Furthermore, Kato Katz can be used to assess both infection presence and infection intensity while requiring a very small sample of stool (World Health Organization). The chart below delineates egg count thresholds (measured as eggs per gram) typically used to characterize infection intensity of the major helminths (Stephenson et al. 2000). Hookworms are listed as one category, as hookworm eggs are not morphologically distinguishable (Kabatereine et al. 2005):

Helminth	**Light Intensity**	**Moderate Intensity**	**Heavy Intensity**
Ascaris lumbricoides	1 – 4,999 epg	5,000 – 49,999	50,000 +
Hookworms	1 – 1,999	2,000 – 3,999	4,000 +
Schistosoma mansoni	1 - 99	100 - 399	400 +
Trichuris trichiura	1 - 999	1,000 – 9,999	10,000 +

However, it is important to note that the Kato Katz technique is not a gold standard and comes with significant limitations. There is a limited window during which a prepared slide must be viewed; after 30-60 minutes of drying, hookworm eggs present in a sample may have collapsed and degraded (Tarafder et al. 2010). This requires

strategic and rapid reading of slides following preparation, especially in the likely case that many samples are being analyzed in succession. Furthermore, the technique is not 100% sensitive, and is particularly prone to missing light infections (Krauth et al. 2012). A 2012 study on the distribution of helminth eggs within a fecal smear suggested that, while there is no clear spatial distribution pattern of eggs in samples, sample homogenization significantly improves detection of *Schistosoma spp.*, as these eggs are more likely to be distributed unevenly in a sample (Krauth et al. 2012). Because this technique uses a very small volume of stool, it is possible that the sample selected may contain no eggs, even if there are eggs present elsewhere in the original stool sample. A study conducted on 271 school-age children in Ethiopia in 2011 found the Kato Katz technique to have 76.6% sensitivity for detecting whipworm infection, 67.8% sensitivity for detecting roundworm infection, and 19.6% sensitivity for detecting hookworm infection (Habtamu et al. 2011). The notably low sensitivity presented by these findings suggests that results of this technique should be interpreted conservatively, and also calls for the use of repeated testing and sample homogenization to improve the likelihood of accurately detecting an infection. In spite of these shortcomings, Kato Katz remains the most efficacious and cost-effective diagnostic method for helminth detection in resource-poor environments.



Morphological features of selected parasite eggs of note: Schistosoma mansoni (A), Ascaris lumbricoides (B), hookworm (C), Trichuris trichiura (E) (Becker et al. 2013)

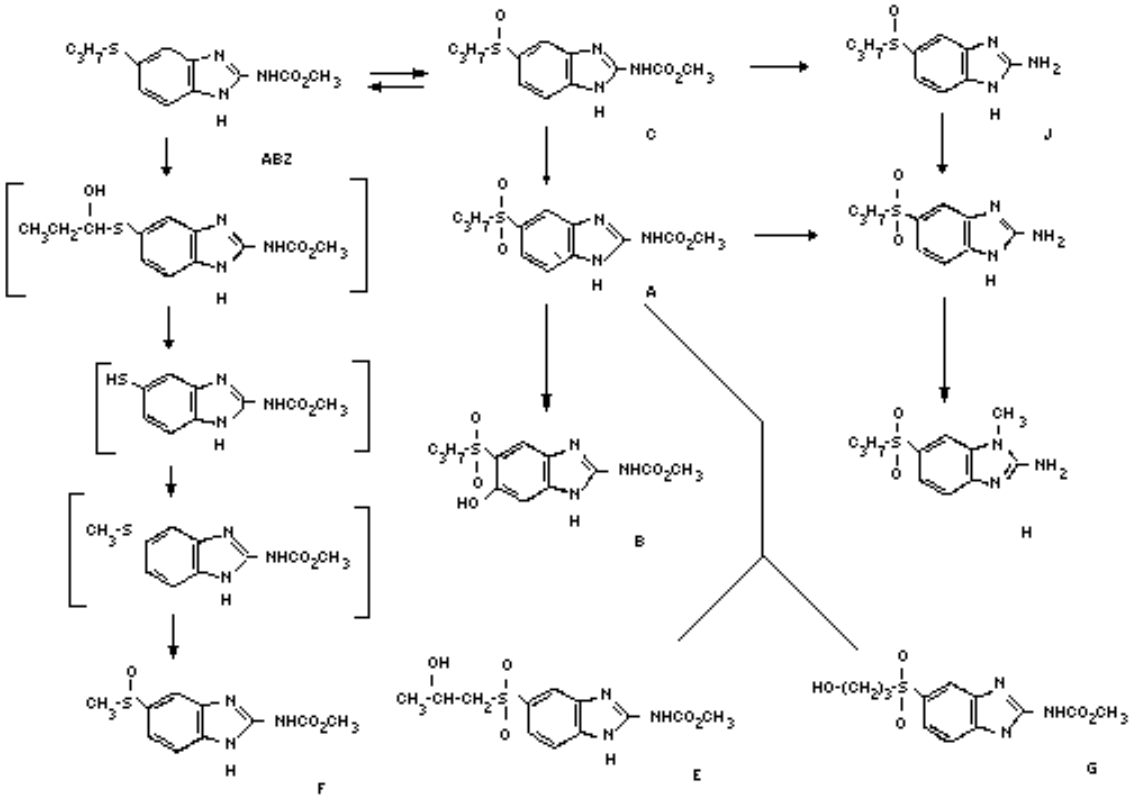
Treatment

Several options exist for chemotherapeutic treatment of helminthiases, the goal of which is to remove adult worms from the body (Bethony et al. 2006). The WHO currently endorses the use of albendazole in the treatment of soil-transmitted helminthiases and the use of praziquantel in the treatment of schistosomiasis (Bethony et al. 2006, Mascarini-Serra 2011). Mebendazole, levamisole, and pyrantel pamoate are also viable alternatives for STN treatment (Mascarini-Serra 2011). The following table summarizes the current standard treatment guidelines for helminthiases (Bethony et al. 2006):

Helminthiasis	Drug	Dose
Ascariasis	Albendazole	400 mg once
	Mebendazole	100 mg twice a day for 3 days OR 500 mg once
	Pyrantel pamoate	11 mg/kg for 3 days
	Levamisole	25 mg/kg once
Hookworm	Albendazole	400 mg once
	Mebendazole	100 mg twice a day for 3 days
	Pyrantel pamoate	11 mg/kg for 3 days
	Levamisole	2.5 mg/kg once; repeat after 7 days if infection is heavy
Trichuriasis	Albendazole	400 mg for 3 days
	Mebendazole	100 mg twice a day for 3 days OR 500 mg once
Schistosomiasis	Praziquantel	60 mg/kg once

The most common treatment option for STN diseases is a single dose benzimidazole drug, either albendazole or mebendazole (Bethony et al. 2006). Both drugs are broad-spectrum anthelmintics, and operate by binding to nematode β-tubulin. This binding action inhibits microtubule polymerization in the parasite, causing death of adult worms within several days (Bethony et al. 2006). Mebendazole is not absorbed well from the gastrointestinal tract, so its therapeutic activity is confined to adult worms residing within the intestines (Bethony et al. 2006). Albendazole, on the other hand, is absorbed more completely, and is metabolized in the liver to a sulphoxide derivative that distributes well throughout many somatic tissues (Bethony et al. 2006). Because this drug is able to reach high concentrations in tissues throughout the body, it attacks both adult worms within the intestines and tissue-migrating larvae (Bethony et al. 2006). The two benzimidazole drugs are comparably effective against ascariasis, though albendazole is typically more effective against hookworm infections, and neither drug is particularly effective against trichuriasis (Bethony et al. 2006). Several doses are sometimes recommended to ensure hookworm and trichuriasis infections are cured.

Fig. 1: Proposed Metabolic Pathways of Albendazole



Albendazole metabolism. End products pictured interfere with β-tubulin polymerization. (http://www.inchem.org/documents/jecfa/jecmono/v25je02.htm)

Systemic toxicity is rarely seen in these drugs when administered at the aforementioned doses, but transient abdominal pain, diarrhea, nausea, dizziness, and headache have been reported in their use (Bethony et al. 2006). Typically, however, both drugs are successful in reducing the worm burden such that it is below the threshold of disease without any side effects (Hotez et al. 2004). Studies suggest that both drugs are embryotoxic and teratogenic when administered to pregnant rats, so there is concern about administering either drug to children younger than one year of age and to pregnant women (Bethony et al. 2006). Pyrantel pamoate and levamisole are acceptable, though less effective, alternative treatments for hookworm and

ascariasis, but must be administered by body weight, making their administration slightly more complicated (Bethony et al. 2006).

Praziquantel is recommended in the treatment of trematodes such as the blood flukes responsible for schistosomiasis, and is widely used as an anthelmintic for veterinary cases (Hodges et al. 2012). Though the details of its mechanism of action are still poorly understood, it is likely that the drug operates by offsetting the balance of membrane-based calcium ion channels, causing an influx of Ca^{2+} into parasitic cells that ultimately results in death of the worms (Doenhoff et al. 2009). Though this drug is highly effective and solely recommended in the treatment of schistosomiasis, its use is associated with a number of side effects, most of which result from the host immune response to released contents of killed worms (Hodges et al. 2012). Even when given after a full meal, praziquantel has been associated with abdominal pain, nausea, vomiting, diarrhea, and dizziness (Hodges et al. 2012). The severity of the side effects often depends on the location of the schistosomes; it is recommended that patients with cerebral infections be hospitalized during treatment, as the rapid and intense immune response to the death of flukes in and around the brain may cause life-threatening seizures (Doenhoff et al. 2009). This is particularly concerning, as experiences and anecdotes of the side effects of this drug have resulted in many endemic communities refusing to take it, complicating treatment programs (Hodges et al. 2012, Parker and Allen 2011).

The most widely accepted and employed method for treating helminthiases is the use of mass drug administration (MDA), which involves a mass distribution of drugs, free of charge, to children (and sometimes adults) in endemic areas (Parker and Allen

2011). The patents on the WHO-recommended benzimidazole anthelmintic treatments have expired, so these drugs may be produced cheaply by generic manufacturers, removing economic barriers that would otherwise prevent their widespread, global use (Bethony et al. 2006). Furthermore, both benzimidazole drugs can be distributed as a single dose tablet, allowing for untrained professionals, such as school teachers, to take charge of their distribution (Hotez et al. 2004). Because of this, and the fact that school-age children tend to have the highest concentration of heavy infections of ascariasis, trichuriasis, and schistosomiasis, many MDA programs operate out of schools (Hotez et al. 2004). It is recommended that treatment frequency be dependent on the intensity of transmission (or the rate of re-infection) within a region, though such metrics are not always available (Mascarini-Serra 2011). MDA programs are quite cheap, and operate on economies of scale, such that the cost per individual decreases when treatment coverage within a community increases (Brooker et al. 2008). In Uganda, the average cost per child treated is estimated to be 0.54 USD, with a cost-effectiveness of 3.19 USD per case of anemia averted (Brooker et al. 2008).

Children receiving anthelmintic tablets as part of National Health Week in Rwanda. (http://www.legatum.org/initiative/Rwanda-and-Burundi-Tropical-Disease-Control)

The World Bank has stated that regular deworming of children is “one of the most cost-effective health interventions a developing nation can undertake” (Hodges et al. 2012). There is a large body of evidence suggesting that school-based deworming improves iron and hemoglobin status, physical growth, cognition, and educational achievement, while reducing school absenteeism (Hotez et al. 2004). Such programs are also believed to confer broader benefits to the community by reducing transmission and lowering the overall burden of disease (Hotez et al. 2004). It is believed that regular treatment will maintain the burden of infection below levels at which disease would result, and this has been corroborated by many studies (Bethony et al. 2006, Hodges et al. 2012). In 2011, the WHO reported that 30% of all school-age children in endemic areas had received treatment; in 2013, over 189 million deworming tablets had been donated to maintain and further this effort (World Health Organization). Substantial improvements in maternal anemia, birth weight, and infant mortality have been observed when MDA programs are extended to include women at risk for pregnancy (Bethony et al. 2006).

Though MDA programs are widely supported, there is increasing evidence that their effectiveness in controlling hookworm is limited, that they neglect critical groups in need of treatment, and that they may actually support the development of drug resistance. Unlike ascariasis and trichuriasis, hookworm disease is not disproportionately concentrated in children, so targeting treatment to younger people does not effectively control disease in many cases (Hotez et al. 2004, Pullan et al. 2010). Furthermore, due to the unique transmission dynamics of helminth infections, high treatment coverage rates do not necessarily correlate to effective disease control:

the primary goal of treatment is to reduce the prevalence of heavy infections, or those infections most likely to result in disease (Hodges et al. 2012). School-based deworming programs tend to disproportionately target children from stable families (who are more likely to be at school on any given day), while systematically missing hard to reach children (Dumba et al. 2008, Hodges et al. 2012). Thus, a region may boast high coverage rates, but still maintain steady transmission if the heaviest infections, and thus the cases most likely to transmit, are missed by the program (Hodges et al. 2012).

Regional data on program effectiveness are rare, and there are substantial theoretical concerns for the emergence of resistance following such widespread use of preventive chemotherapy (Parker and Allen 2011). Data from a 2006 review on STN infections found that reinfection is nearly inevitable in endemic areas, even with the massive and frequent delivery of anthelmintic therapy: hookworm prevalence may reach 80% of pretreatment levels within 30-60 months, ascariasis may return to 55% of pretreatment levels within just 11 months, and trichuriasis may return to 44% of pretreatment levels within 17 months (Bethony et al. 2006). While regular treatment tends to be successful at reducing the overall worm burden, reinfection continues, which may help select for drug resistance over time (Bethony et al. 2006).

In fact, such drug resistance is widespread among livestock nematodes, and has been attributed to the fact that anthelmintics are administered frequently to livestock kept in close proximity with limited gene flow (Soukhathammavong et al. 2012). This may not directly predict emerging resistance among human nematodes, however, as the human parasites tend to reproduce more slowly, and are thus subjected to less frequent treatment (Bethony et al. 2006). Furthermore, treatment of human nematodes

is largely targeted to high-risk populations (Bethony et al. 2006). Nonetheless, exceptionally low cure rates have been observed in isolated cases; resistance appears to be emerging in Southeast Asia and perhaps some parts of sub-Saharan Africa (Soukhathammavong et al. 2012).

	Albendazole (*n* = 89)	**Mebendazole (*n* = 82)**	**Albendazole (*n* = 89)**	***Mebendazole (n = 82)***
No. of hookworm-infected patients	89 (100)	82 (100)	57 (64.0)	*67 (81.7)*
No. of children cured (cure rate, %)	n.a.	n.a.	32 (36.0)	*15 (17.6)*[a]
Light infection (1–1,999 EPG)	72 (80.9)	67 (48.2)	55 (61.8)	*59 (72)*
No. of children cured (cure rate, %)	n.a.	n.a.	17 (19.1)	*8 (9.8)*[b]
Moderate infection (2,000–3,999 EPG)	9 (18.0)	7 (46.7)	2 (2.2)	*6 (7.3)*
No. of children cured (cure rate, %)	n.a.	n.a.	7 (7.9)	*1 (1.2)*[c]
Heavy infection (≥4,000 EPG)	8 (1.1)	8 (1.1)	0 (0)	*2 (2.4)*
No. of children cured (cure rate, %)	n.a.	n.a.	8 (9)	*6 (7.3)*[d]
GM fecal egg count (range), EPG	859.1 (699.0–1,057.0)	707.0 (559.0–894.3)	63.0 (34.0–116.0)	*147.3 (90.0–242.0)*
Egg reduction rate, %	n.a.	n.a.	86.7	*76.3*[e]

[a]*OR 0.4 [95% CI (0.2–0.8; P = 0.01)] comparison of treatment outcomes between mebendazole vs. albendazole;*

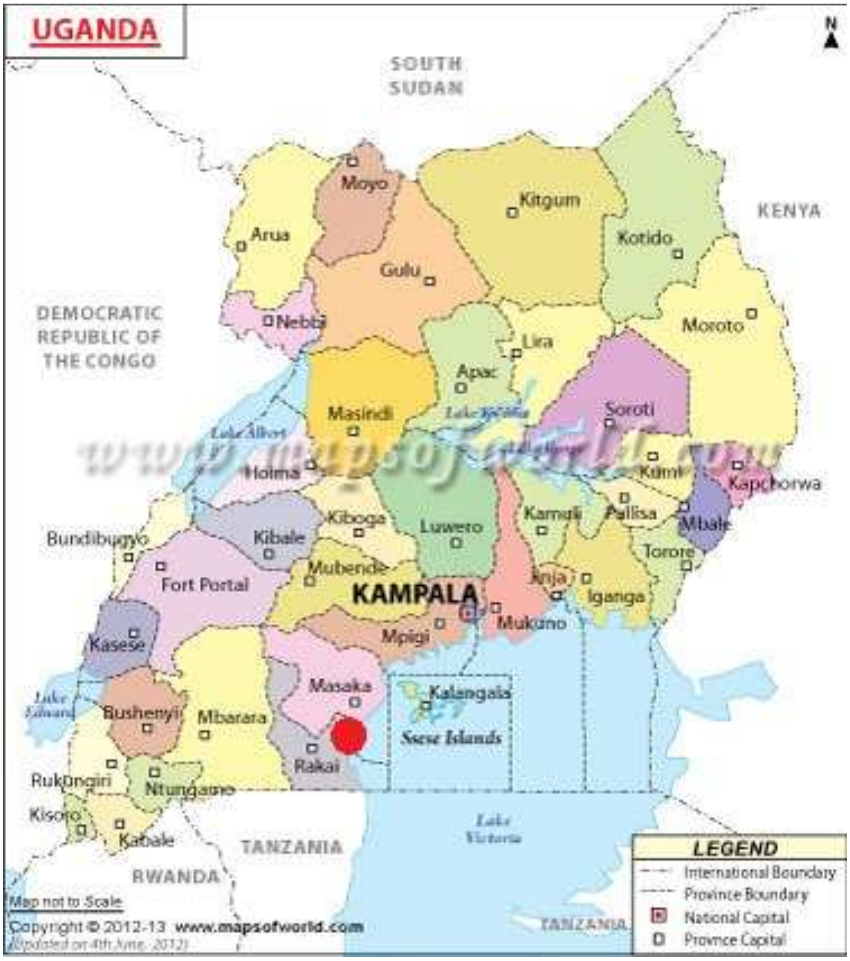
Evidence of albendazole and mebendazole treatment failure in Lao PDR, Soukhathammavong et al. 2013

New treatment options are being developed; nitazoxonide, which is currently used in the treatment of giardiasis and cryptosporidiosis, and tribendimidine have shown some anthelmintic activity, and their success in combined anthelmintic therapy regimens is currently being evaluated (Bethony et al. 2006). However, no new therapies are in the late stages of development, so emerging resistance remains a critical concern, as there are no viable alternative options to date (Bethony et al. 2006).

METHODS

Study Location

This study was conducted in Kabuwoko Parish (which is centrally located within Kirumba Sub-County of Rakai District, Uganda), between June and August 2013, through a joint partnership among the Yale School of Public Health, the non-governmental organization Hope for African Children, and the Kabuwoko Health Centre III. This study location was chosen because of existing relationships with the community and because information on the area was lacking.



Map of Uganda. Kirumba Sub-County, highlighted in red, is located in Rakai District. (http://www.mapsofworld.com/uganda/)

The first reported case of HIV in Uganda was in Rakai District, and the region has been notably stricken by the disease (Sewankambo et al. 1994). As a result, the population of this community consists primarily of young children and their elderly caregivers; young adults, and young male adults in particular, are notably absent. Kabuwoko Parish is a rural community in which the majority of families rely on subsistence farming for survival. There is no running water in the community, and electricity access is limited to a few central areas, and is not reliable. Very little reliable demographic information exists on this community.

Left: Road to Bukunda Village. Right: Home in Dwaniro Village.

Hope for African Children (HAC), a registered non-governmental organization operating out of Kabuwoko Village in Rakai District, Uganda, assisted with on-the-ground support for this project. The HAC staff helped coordinate efforts prior to the arrival of Jensen Reckhow in Uganda by organizing meetings with the LC Chairmen and the community public to introduce and explain the project in a comfortable setting. The organization was also responsible for establishing the partnership with Kabuwoko Health Centre III, where all of the in-country laboratory analysis took place. As an organization that regularly conducts home visits and administers questionnaires to their

members, HAC was able to provide staff to assist with translation during the consent, assent, and questionnaire processes.

Staff member Julie Namazzi posing with sign post outside of HAC Headquarters.

Kabuwoko Health Centre III agreed to serve as a partner in this study by administering single-dose albendazole and praziquantel treatment to all willing study participants found to be infected with helminths. The staff of Kabuwoko Health Centre III, which include both qualified nurses and doctors who are trained and certified in treatment administration, both administered treatment and were responsible for coordinating related activities, including post-treatment care, as needed. This partnership allowed for the research team to assess pre- and post-treatment helminth burdens without dealing directly with treatment administration and related care, while ensuring that study participants had access to treatment and associated care from qualified professionals.

Top Left: Kabuwoko Health Centre III. Top Right: Laboratory space for Kato Katz analysis. Bottom: Laboratory space in health centre.

Study Population

The study population included all school-age children (those between the ages of 4 and 14, inclusive) who live in Bukira, Bukunda, Busowe, Dwaniro, Kabuwoko, Kindulwe, Kabonera, and Segero Villages, located in Kirumba Sub-County of Rakai District, Uganda. The initial proposal called for the selection of study participants using a

random number generator and rosters from five of the regional primary schools. However, due to complications with coordinating with school officials, it was determined that home-based selection would be more feasible. Initial potential study subjects were identified through simple random selection from the roster of Hope for African Children members. All siblings of identified potential participants who resided within the same compound as the original identified potential participant were also eligible and invited to participate in this study. Members of the study population who are mentally disabled, or whose only guardian is mentally disabled, were excluded from this study, as it was deemed difficult to obtain fair and reasonable consent from these potential participants. There was no exclusion of study participants on the basis of sex nor health status, unless a potential participant was deemed to be too ill to complete the tasks required of participation (i.e., the participant is too ill to give informed assent, answer questions, or provide a stool sample). See Appendix 2 for consent forms used in this study.

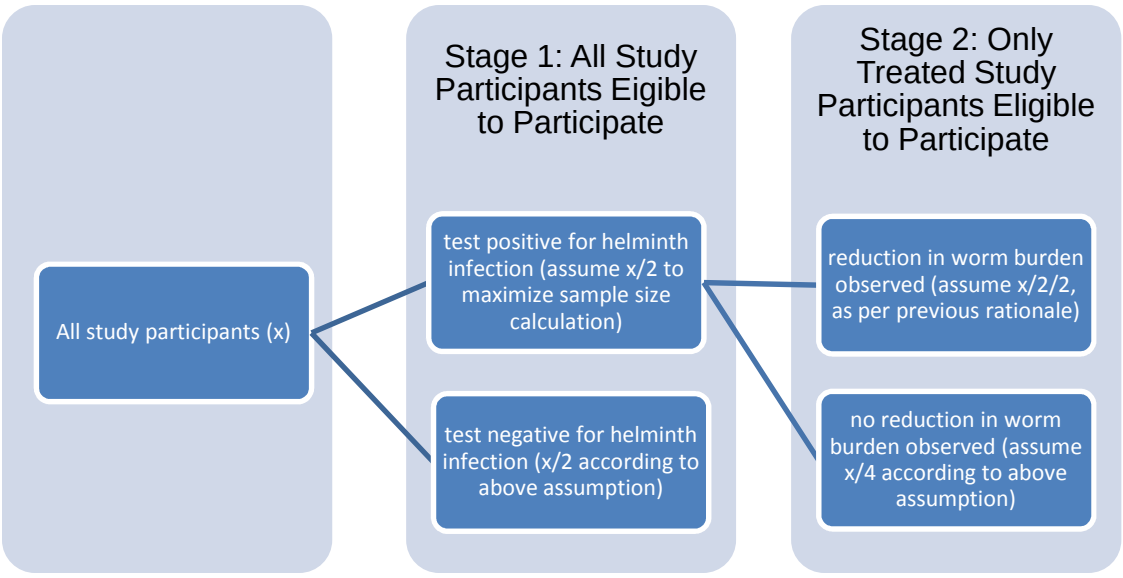
The study population was limited to school-age children because this is the age bracket that is most susceptible to helminth infection and most likely to suffer from associated health problems. Children are more likely to engage in the behaviors that facilitate transmission of helminth diseases: children are more likely to play outside and interact with contaminated soil, and are less likely to remember to follow recommended hygiene behaviors like washing vegetables and hands before eating. Because their behavior allows for increased exposure to helminths, this population is most likely to develop helminth infections. The most severe health outcomes related to helminth infection have to do with inhibition of growth (both physical and cognitive). As discussed earlier, because children are still growing, they are more likely to suffer problems in

these areas than adults who face a similar worm burden but who are essentially done growing. Thus, children (both those in school and those unable to attend) represent the most vulnerable population when it comes to helminth infection, so it is reasonable to focus research efforts on them.

Once the study population was selected, the target sample size was determined after considering mathematical requirements for the study to have reasonable accuracy in its results and after considering the feasible scope of the study given time constraints. All mathematical calculations were made such that the sample size determined would allow for a prevalence estimate that is within 10 percentage points of the true prevalence, estimated with 95% confidence. This level of accuracy was deemed sufficient for this study, as it is one of several accepted standards for epidemiological studies. The following formula was used to calculate the sample size:

$$n = \left(\frac{z_{1-\alpha/2}}{\omega}\right)^2 (\pi(1-\pi))$$

Where n indicates the sample size required given a confidence interval designated by the z-score $z_{1-\alpha/2}$ (for 95% confidence, this value is 1.96) for a population with a true prevalence proportion π to produce an estimate within ω (0.1 for 10 percentage points in this case) of the true proportion (Elashoff and Lemeshow 2013). In these calculations, it was assumed that the true proportion of infected persons within this study population was unknown. In an effort to produce the most conservative calculation, a π value of 0.5 was selected, as this value would produce the highest required sample size. The sample size calculation was made to solve for x according to the following diagram documenting the groups into which study participants may fall during this study:



Thus, assuming the proportion which would result in the most conservative sample size estimate at each stage (0.5), the above equation was solved, resulting in a value of 97 for n. This calculation indicates that the sample size must be such that at least 97 study participants are included in any given round of the study. Because the number of study subjects involved in the second stage of the study is at most the same size as the number involved in the first stage (either all of the participants are treated, or fewer than all are), this number is the minimum number of study participants for the second stage of the study. Because this stage represents, at a most conservative estimate, half of the total participant group, the minimum total number of study participants was calculated to be 97*2 = 194. Thus, at least 194 study participants would be required to achieve estimates for both the proportion of the study population found to harbor worms and the proportion of those found to respond to treatment that are within 10 percentage points of the true values, within 95% confidence.

Because the mathematically derived value seemed well within the reasonable scope of this study given the timeframe, the target sample size was increased to 250 to

account for the likely loss of participants over the course of the study. Based on prior experience and knowledge of conditions in the field, the research team determined that obtaining 250 study participants was a reasonable, realistic goal that satisfied the study's statistical requirements. Thus, the study was set to include at least 250 school-age children as study participants.

Study Design

In preparation for this study, Hope for African Children hosted two community-wide meetings to introduce the project and field questions and concerns related to the upcoming study. At these meetings, the nature of the project was presented, as well as the implications of participating. The purpose of these meetings was to ensure that the community as a whole felt comfortable and familiar with the project before it was allowed to proceed; the research team felt it was imperative to introduce the project in a familiar environment, free from any external pressures, so that community members would feel at ease to ask any and all questions and to express concern. As Hope for African Children hosts community-wide meetings fairly regularly to share updates on their work, this type of meeting setting was familiar and comfortable.

The staff at Hope for African Children also met with the LC1 and LC5 Chairmen to gain approval for the project. In these meetings, the nature of the project was explained, as well as requirements of participation. Like the community-wide meetings, this was an opportunity for the LC Chairmen to express concerns over the project and talk through the intricate details so that they fully understood what it would entail before approving it.

Because all of the meetings hosted by Hope for African Children resulted in enthusiastic support from the community members and leaders, it was confirmed that preparations for the project should move forward swiftly.

Before the study officially began, the full research team met with the LC1 and LC5 Chairmen once more to discuss the project, to provide the Chairmen an opportunity to meet and discuss the protocol with the main on-the-ground researchers, Bazanya Mugagga, Julie Namazzi and Jensen Reckhow. Similarly, another community-wide meeting like the ones already hosted by HAC was ordered, to allow everyone the opportunity to meet the research team and ask questions or express concerns in person.

Once everyone had been introduced to the research team and had an opportunity to learn about the study, ask questions, and express concerns in an informal setting, the process of selecting potential participants and collecting consent and assent from them and their guardians commenced. Potential participating children and homes were identified using random selection from the Hope for African Children roster. All children within the desired age range (4-14 years) within each selected home were eligible for inclusion in the study.

Once potential study participants were selected, the research team traveled to the homes of all potential participants to coordinate the remainder of the study activities. Upon arrival at a potential study participants' home, the research team began by collecting oral consent from the parent or guardian, which was documented using a thumbprint on the form. If consent was given, the research team then collected assent from all eligible children in the home in the same way, again using a thumbprint to

document assent. Consent and assent were taken by Jensen Reckhow, who was authorized to do so by Yale University, while Julie Namazzi served as a translator. If any potential study participant was found to be too ill or otherwise incapable of providing assent or completing the required protocol for the study (evidenced by their inability to complete such tasks), they were at this point excluded from the study. In any case where either consent or assent is not given, the visit was terminated and there was no further contact with the household. In cases where both consent and assent were obtained, the research team then introduced the materials transfer consent and assent forms in the same manner. Study participants and their guardian were not required to give all forms of consent/assent; study participants were able to choose to opt out of the materials transfer component of the study at no cost to them. The full consent/assent process took 15-20 minutes, allowing for ample time for questions and concerns to be addressed. Once this full consent/assent process had been completed, the research team moved on to administer the prepared questionnaire. The questionnaire was administered to the parent or guardian from whom consent was collected, in reference to each child from whom assent was collected, individually. See Appendix 3 for the details of the questionnaire.

Jensen Reckhow conducting a questionnaire with a family in Bukira Village.

Once the questionnaire had been completed, all study participants were provided with stool cups, collection spoons, newspaper, and soap. The process of stool collection was explained thoroughly by both Jensen Reckhow and Bazanya Mugagga, who requested that study participants do their best to collect a stool sample that evening or early the next morning, by defecating on the newspaper and then using the collection spoon to scoop a sample into their pre-labeled stool cup. After sealing the stool cup, ensuring its outside rim is clear, and putting it aside, the remaining fecal matter was to be disposed of in the nearest pit latrine, using the newspaper to prevent human contact with the remaining product. Upon completion of this task, the study participants were urged to wash up using the soap provided to ensure that this process did not detract from his/her personal hygiene. Bazanya Mugagga explained the importance of following this protocol exactly and providing an honest sample, in response to concerns

expressed by the staff at HAC regarding participant compliance. For example, he explained why it is essential that the sample provided came from the designated study participant, and explained that it is okay if the designated participant was unable to produce a sample by the next morning, and that it is more important for the sample to be from the designated study participant than for it to be returned promptly. Following this discussion, the research team thanked the family for agreeing to participate in this study, and explained that they would return the following morning to collect all prepared samples. Due to time constraints, participants were also told to return to the HAC office within the next two days to learn their infection status and be referred for treatment if necessary. As the HAC office was on the way to school for most study participants, little hardship was incurred through these visits to the office, and the majority of study participants actually brought their samples directly to the office on their own.

Collected samples were transported by the research team to Kabuwoko Health Centre III using a biohazardous specimen carrier, where they were analyzed by Bazanya Mugagga and Jensen Reckhow. The Kato Katz technique was used to identify the presence and concentration of *Necator americanus*, *Ancylostoma duodenale*, *Ascaris lumbricoides*, *Trichuris trichiura*, and *Schistosoma* eggs in each sample provided. The number of eggs seen in the given sample was then extrapolated based on the known size of the sample to provide an estimated helminth burden, measured as eggs per gram of stool (epg). Based on these extrapolated calculations, this technique allowed for study participants to be categorized as having a light, moderate, heavy, or non-existent infection, based on WHO standards for evaluating helminth infection

intensity. Egg counts were conducted by both Bazanya Mugagga and Jensen Reckhow in an effort to improve accuracy, though only one Kato Katz slide was prepared.

Light microscope used to identify helminth eggs in fecal samples.

A physical record of all study participants found to harbor helminth infections was kept at Kabuwoko Health Centre III and was monitored by a member of the research team during all hours of operation throughout the study. While laboratory work is being done, Bazanya Mugagga and Jensen Reckhow were responsible for watching over this list; when Kabuwoko Health Centre III was not in operation, this list was stored in a locked cabinet along with all other sensitive data stored at the health center.

All samples proven to harbor no helminth infection were discarded (transferred to a biodegradable collection bag that was disposed of in the health center biohazard waste pit at the end of each work day). The samples that did harbor helminth eggs were cultured to produce larvae using a modified Baermann method. The traditional Baermann method involves mixing a fecal sample with bone charcoal at a 1:5 ratio and incubating the mixture for 11 days at ambient temperature (Suwansaksri et al. 2003).

This is meant to simulate environmental conditions under which STN eggs may hatch into larvae and mature (Suwansaksri et al. 2003). Following the incubation period, the mixture is transferred to a kimwipe and placed in a funnel apparatus filled with water (Suwansaksri et al. 2003). The mixture is left to stand for 14-18 hours, during which time it is expected that any larvae cultured in the mixture will migrate into the water, effectively resulting in a larval extraction (Suwansaksri et al. 2003). Due to resource limitations, the cultures were kept in open tupperware containers on the floor of the laboratory, covered with kimwipes. Following the 11 day incubation period, the mixtures were placed in a funnel apparatus filled with water. The larvae then migrated out of the fecal mixture and were collected at the base of the funnel after standing for an average of 16 hours. A small sample from each preparation was examined under the microscope to confirm the presence of larvae; successful samples were gravitationally concentrated and preserved in an ethanol-base solution.

Baermann Funnels used to extract larvae from feces/charcoal mixture.

The filtration apparatus in the funnel ensured that only helminth larvae were collected in the final sample, and that these larvae were fully isolated from any genetic and biological material from the original study participant. Final samples isolated using this technique consisted only of harvested helminth larvae and a small amount of water (verified by viewing a small portion of each sample under the microscope to observe the larvae). The samples were stored in the laboratory at Kabuwoko Health Centre III throughout the duration of the study, such that only Jensen Reckhow had access to them while they were in Uganda. Following completion of the study, these samples were returned to Yale University with Jensen Reckhow as per the conditions set forth by the relevant Materials Transfer Agreement. See Appendix 4 for the text of the Materials Transfer Agreement used in conjunction with this study.

The aforementioned protocol was repeated for all study participants. Throughout the duration of the study, Hope for African Children extended invitations to Center Days to all study participants. Center Days, which occur every Saturday, are typically for HAC members only, and consist of a day of character-building activities with two free meals. The community is familiar with this HAC program, and participation was high, likely due to the provision of complimentary meals. This provided an excellent opportunity for treatment administration. Madame Goletti, the primary doctor at the health center, came down to the HAC office to administer treatment during each Center Day over the course of the study. Madame Goletti was experienced with administering this type of treatment.

Left: HAC staff coordinating children to receive albendazole treatment.
Right: Children after receiving anthelmintic therapy (boxes pictured are empty).

An ample supply of 400 mg albendazole tablets and 600 mg praziquantel tablets were provided to Kabuwoko Health Centre III free of charge for use in tandem with this study. While treatment administration was not a component of this study, it was hoped that it would occur in parallel to the efforts proposed here so that its impact could be evaluated. To this end, treatment was purchased by the research team for qualified staff at Kabuwoko Health Centre III to administer to study participants throughout the duration of the study. Unused treatment doses and other equipment were donated to Kabuwoko Health Centre III for future use as necessary in the community.

Left: Jensen Reckhow explaining functionality of donated equipment to Madame Goletti. Right: Julie Namazzi, Jensen Reckhow, and Madame Goletti celebrating donated equipment and anthelmintics.

A single-dose 400 mg albendazole treatment was recommended for study participants found to harbor infections by *Necator americanus*, *Ancylostoma duodenale*, *Ascaris lumbricoides*, and *Trichuris trichiura* and a weight-dependent dose of 40 mg/kg (~2-5 tablets of 600 mg doses) of praziquantel treatment was recommended for study participants found to harbor *Schistosoma* infections. These treatments are approved and widely recommended for the age range of the study population (with no study participants under the age of 4, there was no uncertainty about the safety of these drugs). The praziquantel treatment regimen is exactly in line with the WHO-recommended standard of treatment as well as the standard treatment protocol recommended by the Ministry of Health of Uganda. The albendazole treatment regimen is in line with WHO recommendations as well, but contradicts the standard mebendazole treatment regimen that is accepted in Uganda. The decision to follow an alternative treatment protocol to that which is typically administered in Uganda was due to evidence-based research and understanding of the mechanisms of the two drugs available and their relative effectiveness when treating the diseases in question. As discussed earlier, mebendazole acts almost exclusively in the gut, making it an excellent treatment option for destroying parasitic worms that reside in the intestines, and a relatively non-toxic one, due to its poor absorption rates. Albendazole, on the other hand, is an effective parasite-killing agent in the gut as well as throughout body tissues, making it more effective as a holistic treatment option. The following table summarizes the results of a selection of research studies conducted on the comparative effects of albendazole and mebendazole treatment regimens, and suggests that

albendazole treatment has generally been found to be more effective for treating the STN infections under review in this study:

Hookworm Infection CR	Roundworm Infection CR	Whipworm Infection CR	Details of the Study
Albendazole: 84.3% Mebendazole: 9.1%	100% 100%	67.4% 43.3%	Jongsuksuntiqul et al., 1993, Thailand
Albendazole: 92.4% Mebendazole: 50%	83.5% 79.6%	67.8% 60.6%	Muchiri et al., 2001, Western Kenya
Albendazole: 81.8% Mebendazole: 17.2%	100% 100%	Egg reduction: 45.7% 15%	Bartoloni et al, 1993, Bolivia
Albendazole: 72% Mebendazole: 15%	88% 92%	53% 36%	Keiser and Utzinger, 2010, Meta-analysis

All study participants treated for helminth infection during this study were contacted for follow up seven days after receiving treatment. The homes of these participants were visited again, and new stool cups, collection spoons, newspaper, and bars of soap were provided for each participating child who had received treatment. This visit was identical to the first visit, with the exception that consent and assent were not collected for a second time (as this part of the protocol was already explained in the original forms) and the questionnaire was not repeated. Similar to the first visit, the study participant was asked to prepare their stool sample in the evening or early the following morning, and the prepared sample was to be retrieved from their home the following morning by the research team. The provided samples were processed in the same manner as the original one was; Kato Katz was used to quantify helminth infection, and the Baermann Technique was used to culture positive samples.

Following completion of the study in Uganda, isolated larval specimens were transported back to Yale University by commercial plane as checked baggage with Jensen Reckhow, as per the terms of the Materials Transfer Agreement. It was

essential that these specimens be transported to Yale University, because the subsequent analytical techniques to be used were developed and modified in the Cappello Lab at Yale, and this laboratory is well equipped to manage all aspects of the protocol. Unfortunately, Kabuwoko Health Centre III does not have the capacity to conduct genetic analysis on these specimens, and none of the parties involved in this study had access to other facilities in Uganda where this could be done easily and in a timely manner. Because the specimens will degrade over time, transferring them to the facilities at Yale University made the most sense and was most likely to yield quality results that would constitute a useful and viable body of research.

Post-Collection Laboratory Analyses

DNA was extracted from the harvested larval samples upon their arrival at Yale University using the QIAamp DNA Stool Mini Kit manufactured by Qiagen. This kit was selected because it is specifically designed to extract “genomic, bacterial, viral, and parasite DNA from fresh or frozen human stool.” The utility of this technique comes from its use of a distinctive adsorptive resin that removes PCR inhibitors commonly present in stool samples. The procedure takes less than one hour, and was deemed an efficient and straightforward method to use for extracting larval DNA from less-than-ideal samples (Qiagen).

Following DNA extraction, a speciating polymerase chain reaction technique was used to differentiate between *Necator americanus* and *Ancylostoma duodenale* hookworm specimens (as well as to identify erroneous samples to be discarded). A number of techniques were tested, and a technique that amplified the mitochondrial

cytochrome oxidase I (COX-1) gene proved to be most effective for these samples. The technique selected involves amplifying 585-bp fragments of the COX-1 gene in a given egg, larval, or adult hookworm sample, and using gel electrophoresis to determine whether the fragment is present (Zhan et al. 2001). The technique uses species-specific primers, so the electrophoresed gel will clearly identify which species is/are present.

Future laboratory analysis will be required to test the hypothesis that there is a genetic basis for anthelmintic resistance in the STN population endemic to the study location. These efforts will involve sequencing the β-tubulin gene that is the target of these therapies, and attempting to glean insights from observed correlations between gene SNPs and treatment effectiveness.

Statistical Analyses & Rationale

Statistical analyses for this study were conducted using the SAS and R statistical programming packages. All of the data collected during the study was entered into Excel twice to ensure accuracy in data reporting. While the majority of the data collected were used in their existing form for analysis, several questions were combined to produce indices for socioeconomic status, dietary diversity, and hunger status. Dietary diversity was assessed following recommendations from the Food and Agriculture Organization, and hunger was evaluated in line with the FANTA household hunger scale developed by USAID (Food and Agricultural Organization, United States Agency for International Development). The socioeconomic index was derived from a model in use in a longitudinal study the Cappello Lab is currently conducting in Ghana, and is in line with traditional asset-based proxy models typically used to asses socioeconomic

status where income and cash flows are either difficult to measure or culturally irrelevant (Humphries et al. 2011, Vyas and Lilani 2006). The chart below delineates how the socioeconomic indicator was built from the questionnaire responses:

Question from Questionnaire	Points Added to SES Indicator, by Response
What is the main material of the floor?	Natural floor – 1 Natural floor covered with mats – 2 Cement floor - 3
What is the main material of the roof?	Metal – 1 Cement - 2
Does any member of the household own agricultural land?	Yes – 1 No – 0
Does any member of the household own at least one cow, goat, chicken, pig, or duck?	Yes – 1 No – 0
What is the main source of water for members of your household?	Borewell or contained rainwater – 1 Other - 0
What kind of toilet facility do members of the household use?	Pit latrine – 1 Bush – 0
Where does the child get medical care if he/she is sick?	Government clinic – 1 Private clinic – 2 Private clinic in a more urbanized area – 3
Cumulative hunger score	No hunger risk – 1 Some hunger risk - 0

In addition to these, weight for height and body mass index parameters were also built for use in data analysis. As few people in this region keep track of birth dates, there was not sufficient data to assess weight-for-age, height-for-age, or BMI-for-age, which are the typical metrics of size used in this type of study. Instead, weight for height and BMI were used in isolation, as these require only weight and height data. Though both were formally analyzed, the results for BMI were not used in interpretations, as it is not accepted to generalize BMI data for children. Such data was only used to corroborate associations observed with regard to the weight for height metric.

The majority of analyses were executed using SAS. A univariate analysis, in the form of a frequency distribution, was conducted on all data metrics collected. The purpose of this analysis was to determine general sample population averages for the different parameters assessed. Univariate analysis was the simplest way to procure summary statistics for the population as a single sample. Bivariate analyses were then conducted to highlight relationships between different classes of variables, with a focus on comparisons between demographic and behavioral factors (data collected from the questionnaires) and health outcomes (laboratory data). All of the health outcome variables, and many of the demographic and behavioral variables, were categorical, some binary and others nominal. Bivariate analyses conducted on categorical variables were assessed using the chi squared test. Correlations between outcome variables and continuous variables were assessed using an F test analysis of variance, which assessed differences between the mean of the continuous variable when the sample was stratified by the outcome variable. These analyses allowed for the identification of statistically significant relationships among the data collected during the study, without regard to the direction or nature of the relationships.

Logistic regressions were then run on each class of predictor parameters to produce unadjusted odds ratios assessing relative risk of one outcome versus another among different subsets of the population. Logistic regression was used because the outcomes assessed were categorical (as discussed above). This analysis supplemented the chi squared and F tests with information regarding the direction and magnitude of each association.

As is standard among research projects that aim to assess risk factors for helminth infection, the bivariate analyses were followed by the construction of logistic regression models to tease out the effects of individual risk factors on a number of outcomes. Logistic regression was chosen for this purpose because all outcome variables of interest were categorical rather than continuous. A standard logistic model was used for the binary outcome variables, and a multinomial logistic model was used for the ordinal ones. The logistic regression analysis allowed for the measurement of adjusted associations; it was able to tease out which variables retained or gained significance in predicting the outcome when other relevant variables were held constant. This allowed for the isolation of specific effects, which is useful when aiming to understand the fundamental causes of outcomes of interest. Linear regression was not used in this study, because none of the outcome variables of interest were truly continuous.

Three model-building logistic regressions were used: the first assessed the significance of age, sex, socioeconomic status, and dietary diversity on the outcome of interest. Such a model is typically used in this field, as these are the factors most commonly predicted to be associated with STN infection and related health outcomes. As data on these parameters was complete for the full study population, the full dataset was used in the evaluation of this model. However, as these were not always the parameters found to be significant in this study, a second logistic regression was also used. The second regression built a model using backward selection, taking into consideration all available parameters. This second model was used to reveal the unique combinations of predictors found in this particular study. No parameters were

excluded from this analysis on the basis of co-linearity, as logistic regression is fairly good at controlling for this inherently. Additionally, no parameters were excluded on the basis of presumed irrelevance, because data in this field is limited, and it was assumed that valuable information could be gleaned from any relationships found, whether expected or not. Thus, every variable analyzed for which there was sufficient data available was included in this selection process. For this second model, only those individuals for whom complete data on all parameters was available were included. This ensured consistency in the population analyzed by the regression model, and ensured that the backward selection procedure progressed appropriately. The second model represents the most parsimonious model reached. If a parsimonious model was reached when more than five predictors remained, the procedure continued until the next most parsimonious model was found. This was done because models with many insignificant predictors do not bear scientific relevance, even if they are statistically sound.

Unfortunately, many of the outcomes did not have a substantial number of predictors. Because of this, the second logistic regression model was reported twice: once using only those predictors that were included in the final, most parsimonious model, and once using the five most significant predictors, regardless of statistical significance. Given the small sample size, this decision was made to ensure that potentially important predictors did not go without consideration due to stringent significance requirements. Appendix 5 documents the backward selection procedures used to construct each of these models.

Beyond the analysis done using SAS, the assessments regarding treatment effectiveness were done using an interface for the R statistical package eggCounts hosted by the University of Zurich. This program analyzes pre- and post- treatment Kato Katz data for each helminth species to determine a 95% confidence interval for the fecal egg count reduction (FECR) using a paired t-test. This standard procedure is fitting for such an analysis: FECR seeks to detect the change in the average number of eggs counted per sample before and after treatment (i.e., the means of two paired samples); a paired t-test is the appropriate statistical test for detecting such a difference. The cure rate was determined by calculating the percentage of individuals whose samples were positive for a given helminth species whose second sample was negative for that species, documenting the percentage of individuals found to have been cured of a detected infection following treatment administration.

RESULTS

Participation & Characteristics of the Study Population

Of the 301 eligible children approached, 269 agreed to and participated in this study. Eight villages within Kirumba Sub-County were represented in the study population. Segero Village had the greatest level of representation among the study population (22.7%), while Bukunda Village had the lowest (3.0%). Study participants were Catholic (63.6%), Christian (31.2%), and Muslim (5.2%), with nearly equal sex representation (52.8% females and 47.2% males). The average age among study participants was 8.7 years. Study participants represented all levels of school enrollment, with some individuals in nursery (14.5%), primary (63.6%), and secondary

(5.2%) school, in addition to some not enrolled (16.7%). Over half of the study population did not report wearing shoes (52.8%), but 42.4% of participants claimed to wear shoes daily. Two-thirds of the sample had not been dewormed in the past year. Only one-quarter of the children had slept under a bednet the night before the questionnaire, and 27.1% had had malaria within the last year. The majority of households owned at least one pig (78.8%), and most heads of household were farmers (85.9%). Most heads of household completed some primary school (55.3%), and a smaller portion completed at least some secondary school (27.9%). The average child had a diet that represented 3.4 dietary groups (out of a possible 16), and the average socioeconomic score was 8.6 out of a possible 13. The average weight for height was 0.21 and the average BMI was 16.9. A detailed breakdown of the characteristics of the study population is included in Table 1, featured below.

Table 1. Univariate Analysis of the Sample[1]

Characteristic	N	% of Sample
Age Group		
4 to 5 years	71	26.4
6 to 10 years	105	39.0
11 to 14 years	93	34.6
Community		
Bukira	36	13.4
Bukunda	8	3.0
Busowe	47	17.5
Dwaniro	10	3.7
Kabonera	33	12.3
Kabuwoko	39	14.5
Kindulwe	35	13.0
Segero	61	22.7
Sex		
Female	142	52.8
Male	127	47.2
Religion		
Catholic	171	63.6
Christian	84	31.2
Muslim	14	5.2

Characteristic	N	% of Sample
Schooling		
Not Enrolled	45	16.7
Nursery	39	14.5
Primary	171	63.6
Secondary	14	5.2
Shoe Usage		
Never	142	52.8
Every Week	13	4.8
Every Day	114	42.4
Deworming History		
Past 12 months	89	33.5
More than 12 months	177	66.5
Bednet Use		
Uses Net	69	25.7
Does not use net	200	74.4
Pig Ownership		
Owns pigs	212	78.8
Does not own pigs	57	21.2
Head of Household Education		
None	41	16.8
Some Primary School	135	55.3
Some Secondary School	68	27.9
Head of Household Occupation		
Farmer	231	85.9
Other	38	14.1
Malaria History		
Past 12 months	73	27.1
More than 12 months	196	72.9
Hookworm Infection	148	55.0
***A. lumbricoides* Infection**	133	49.4
***T. trichiura* infection**	57	21.2
Infection Intensity		
No Infection	79	29.4
Light	175	65.1
Moderate/Heavy	15	5.6
Multiplicity		
No infection	79	29.4
One species	76	28.3
Two species	80	29.7
Three species	34	12.6
Average Dietary Diversity Score	3.4 ± 1.1	NA

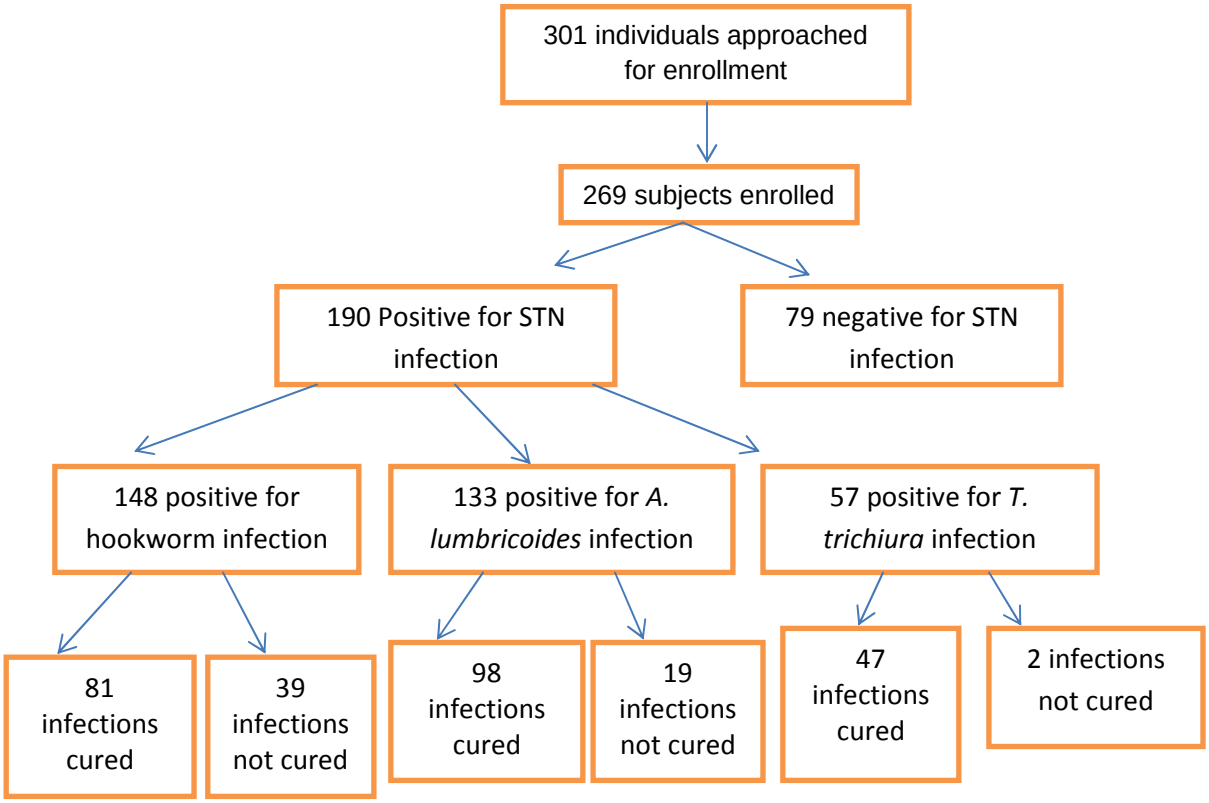
Characteristic	N	% of Sample
Average Socioeconomic Index	8.6 ± 1.4	NA
Average Weight/Height	20.5 ± 6.0	NA
Average Body Mass Index	16.9 ± 4.1	NA

[1] Numbers may not sum to 269 due to missing data, and column percentages may not sum to 100% due to rounding.

70.6% of the study population was found to harbor STN infection during the pre-treatment analysis, with prevalences of 55.0%, 49.4%, and 21.2% for hookworm, *A. lumbricoides*, and *T. trichiura* infection, respectively. Of the 190 individuals infected by STNs, 39 of them harbored only hookworm infection (20.5%), 33 harbored only *A. lumbricoides* infection (17.4%), and 4 harbored only *T. trichiura* infection (2.1%), such that 28.3% of the population had a mono-infection. Sixty one individuals were infected with both hookworm and *A. lumbricoides* (32.1%), while 14 were infected with hookworm and *T. trichiura* (7.4%) and only 5 were infected with *A. lumbricoides* and *T. trichiura* (2.6%); 29.8% of the study population was infected by two distinct STN species. Thirty-four individuals, or 12.6% of the population, were infected by all three STN species of interest. Nine percent of the infections were moderate or heavy; 91% were of light intensity.

Schistosoma sp. and *Taenia sp.* infections were also identified among the study population (4.1% and 1.1%, respectively), but were excluded from subsequent analyses for several reasons. Firstly, the prevalence for each of these infections was quite low, such that analyses of correlation with other parameters assessed in the study would be unable to yield significant or meaningful results. Such analysis would not contribute useful findings to the study. Furthermore, it would be difficult to assess whether the *Schistosoma sp.* infections were acquired within the study location, as the majority of the individuals found to harbor the infection had recently travelled to and swam in Lake

Victoria as a part of a school trip. Lake Victoria is a known reservoir of *Schistosoma* cercariae, so it is possible that these infections developed following exposure outside of the study location (Kabatereine et al. 2011). The uncertainty regarding the source of the infection, coupled with the low prevalence in the area, complicate the validity of any subsequent analyses assessing demographic and behavioral risks associated with the infection. On the other hand, the source of the *Taenia sp.* infections is well established, and is likely due to the consumption of undercooked pork (Centers for Disease Control). In this case, the low prevalence alone precludes additional analysis. Future analytic data in this study thus excludes these two classes of infections.



Characteristics of study enrollment. Numbers may not sum to totals due to missing data.

Description of the Sample by Infection Status

Tables 2a and 2b provide detailed information about the study population in terms of infection status. Age was found to be significantly associated with the occurrence of STN infection, such that the youngest individuals were more likely to harbor an infection than the oldest individuals: children between the ages of 11 and 14 were only one quarter as likely to harbor STN infection as children 5 and younger ($p = 0.001$). Village of residence was not found to bear any significant association with infection status. Males were slightly less likely to harbor an STN infection than females (OR = 0.71), but this was not significant ($p = 0.2$). Religion, bednet use by the child, whether the child had been dewormed within the past year, head of household education level, and whether the head of the household was a farmer were not associated with an outcome of STN infection. The odds of being infected were highest among children enrolled in nursery school, and lowest among those in secondary school, but this association was not significant ($p = 0.07$). Surprisingly, children who claimed to wear shoes daily were 2.29 times as likely as children who never wore shoes to harbor an infection, and this difference was significant ($p = 0.01$). Children who had suffered from malaria within the past year were more than two times as likely to harbor STN infection than children who had not had the disease in the past year ($p = 0.025$).Children who owned pigs were 71% more likely to harbor STN infection than those who did not, but this finding was not significant ($p = 0.8$). Socioeconomic status and dietary diversity bore no relation to infection status. Average weight for height was higher among the group of non-infected children ($p = 0.004$). Suprisingly, no association was seen between BMI and STN infection status ($p = 0.4$). Dietary diversity and

socioeconomic status bore no relation to infection status (p = 0.4 and p = 0.5, respectively).

Table 2a. Description of the sample according to infection status[1]

Characteristic	STN Infection		p Value[2]
	Yes (N = 190)	**No (N = 79)**	
Age Group			**0.0010**
4 to 5 years (N = 71)	60 (31.6)	11 (13.9)	
6 to 10 years (N = 105)	76 (40.0)	29 (36.7)	
11 to 14 years (N = 93)	54 (28.4)	39 (49.4)	
Community			0.8873
Bukira (N = 36)	25 (13.2)	11 (13.9)	
Bukunda (N = 8)	6 (3.2)	2 (2.5)	
Busowe (N = 47)	31 (16.3)	16 (20.3)	
Dwaniro (N = 10)	6 (3.2)	4 (5.1)	
Kabonera (N = 33)	23 (12.1)	10 (12.7)	
Kabuwoko (N = 39)	30 (15.8)	9 (11.4)	
Kindulwe (N = 35)	23 (12.1)	12 (15.2)	
Segero (N = 61)	46 (24.2)	15 (19.0)	
Sex			0.2073
Female (N = 142)	105 (55.3)	37 (46.8)	
Male (N = 127)	85 (44.7)	42 (53.2)	
Religion			0.5232
Catholic (N = 171)	122 (64.2)	49 (62.0)	
Christian (N = 84)	60 (31.6)	24 (30.4)	
Muslim (N = 14)	8 (4.2)	6 (7.6)	
Schooling			0.0695
Not Enrolled (N = 45)	35 (18.4)	10 (12.7)	
Nursery (N = 39)	32 (16.8)	7 (8.9)	
Primary (N = 171)	116 (61.1)	55 (69.6)	
Secondary (N = 14)	7 (3.7)	7 (8.9)	
Shoe Usage			**0.0161**
Never (N = 142)	90 (47.4)	52 (65.8)	
Every Week (N = 13)	9 (4.7)	4 (5.1)	
Every Day (N = 114)	91 (47.9)	23 (29.1)	
Deworming History			0.2075
Past 12 months (N = 89)	67 (35.8)	22 (27.9)	
More than 12 months (N = 177)	120 (64.2)	57 (72.2)	
Bednet Use			0.9355
Uses Net (N = 69)	49 (25.8)	20 (25.3)	
Does not use net (N = 200)	141 (74.2)	59 (74.7)	
Pig Ownership			0.0849
Owns pigs (N = 212)	155 (81.6)	57 (72.2)	

Characteristic	STN Infection		p Value[2]
	Yes (N = 190)	**No (N = 79)**	
Does not own pigs (N = 57)	35 (18.4)	22 (27.9)	
Head of Household Education			0.7959
None (N = 41)	28 (16.4)	13 (17.8)	
Some Primary School (N = 135)	97 (56.7)	38 (52.1)	
Some Secondary School (N = 68)	46 (26.9)	22 (30.1)	
Head of Household Occupation			0.4064
Farmer (N = 231)	161 (84.7)	70 (88.6)	
Other (N = 38)	29 (15.3)	9 (11.4)	
Malaria History			**0.0251**
Past 12 months (N = 73)	59 (31.1)	14 (17.7)	
More than 12 months (N = 196)	131 (69.0)	65 (82.3)	
Average Dietary Diversity Score	3.4 $\pm$ 1.1	3.5 $\pm$ 1.3	0.4284
Average Socioeconomic Index	8.7 $\pm$ 1.4	8.6 $\pm$ 1.4	0.5171
Average Weight/Height	19.8 $\pm$ 6.1	22.1 $\pm$ 5.5	**0.0044**
Average Body Mass Index	16.7 $\pm$ 4.3	17.2 $\pm$ 3.6	0.3923

[1]Numbers may not sum to 269 due to missing data, and column percentages may not sum to 100% due to rounding.
[2]P value for analysis of variance F-test (continuous variables) or χ^2 test (categorical variables).

Table 2b. Unadjusted associations between study variables and infection status

Characteristic	N[1]	% Infected	OR (95% CI)[2]
Age Group			
4 to 5 years	71	84.5	1.00
6 to 10 years	105	72.4	0.48 (0.22, 1.04)
11 to 14 years	93	58.1	0.25 (0.12, 0.55)
Community			
Bukira	36	69.4	0.74 (0.30, 1.86)
Bukunda	8	75.0	0.98 (0.18, 5.37)
Busowe	47	67.0	0.63 (0.27, 1.46)
Dwaniro	10	60.0	0.49 (0.12, 1.97)
Kabonera	33	69.7	0.75 (0.29, 1.93)
Kabuwoko	39	76.9	1.09 (0.42, 2.80)
Kindulwe	35	65.7	0.63 (0.25, 1.55)
Segero	61	75.4	1.00
Sex			
Female	142	73.9	1.00
Male	127	66.9	0.71 (0.42, 1.21)
Religion			
Catholic	171	71.4	1.00
Christian	84	71.4	1.00 (0.56, 1.79)
Muslim	14	57.1	0.54 (0.18, 1.62)
Schooling			

Characteristic	N[1]	% Infected	OR (95% CI)[2]
Not Enrolled	45	77.8	1.00
Nursery	39	82.1	1.31 (0.44, 3.84)
Primary	171	67.8	0.60 (0.28, 1.31)
Secondary	14	50.0	0.29 (0.08, 1.01)
Shoe Usage			
Never	142	63.4	1.00
Every Week	13	69.2	1.30 (0.38, 4.43)
Every Day	114	79.8	2.29 (1.29, 4.05)
Deworming History			
Past 12 months	89	75.3	1.45 (0.81, 2.57)
More than 12 months	177	67.8	1.00
Bednet Use			
Uses Net	69	71.0	1.03 (0.56, 1.87)
Does not use net	200	70.5	1.00
Pig Ownership			
Owns pigs	212	73.1	1.71 (0.93, 3.16)
Does not own pigs	57	61.4	1.00
Head of Household Education			
None	41	68.3	1.00
Some Primary School	135	71.9	1.19 (0.56, 2.53)
Some Secondary School	68	67.7	0.97 (0.42, 2.23)
Head of Household Occupation			
Farmer	231	69.7	0.71 (0.32, 1.59)
Other	38	76.3	1.00
Malaria History			
Past 12 months	73	80.8	2.09 (1.09, 4.02)
More than 12 months	196	66.8	1.00
Average Dietary Diversity Score	269	NA	0.91 (0.72, 1.15)
Average Socioeconomic Index	269	NA	1.06 (0.88, 1.28)
Average Weight/Height	269	NA	0.94 (0.90, 0.98)
Average Body Mass Index	269	NA	0.97 (0.92, 1.04)

[1]Numbers may not sum to 269 due to missing data.

[2]Unadjusted odds ratios were calculated using logistic regression.

The results of the logistic regression models built to predict an outcome of STN infection can be found below in Table 2c. Of the parameters considered in the first logistic regression model (age, sex, dietary diversity, socioeconomic status index), only age was found to be significant. Increasing age was associated with a lower risk of

infection (adjusted OR = 0.86, p = 0.0001), as was expected from the bivariate analysis. No association was observed between any of the other parameters and STN infection.

Five significant predictors were found when all parameters were considered in an adjusted association model, so only one model was reported for the second logistic regression. Notably, having been dewormed within the past 12 months resulted in a two-fold increased risk of having an STN infection (p = 0.04). Owning a pig and having had malaria in the past year also appeared to increase the odds of being infected (adjusted OR = 2.01 and 2.23, respectively). Infected individuals had, on average, a 15% reduction in weight for height when compared to uninfected individuals, after controlling for deworming history, pig ownership, malaria history, and BMI (p = 0.0001).

Table 2c: Logistic Regression Models to Predict STN Infection

Characteristic	**Adjusted OR (95% CI)**	**p**
Model 1		
Age	0.86 (0.79, 0.93)	**0.0001**
Sex (Ref = female)	0.77 (0.45, 1.34)	0.3581
Dietary Diversity	0.88 (0.69, 1.12)	0.2989
Socioeconomic Status	1.08 (0.89, 1.32)	0.4438
Model 2		
Deworming History	2.00 (1.03, 3.88)	**0.0419**
Pig Ownership	2.01 (1.02, 3.96)	**0.0442**
Malaria History	2.23 (1.09, 4.55)	**0.0278**
Weight/Height	0.85 (0.78, 0.92)	**0.0001**
BMI	1.16 (1.03, 1.31)	**0.0181**

Risk Factors for Individual Helminth Infections

Bivariate analyses of demographic and behavioral risk factors stratified by specific types of helminth infections can be found in Tables 3a and 3b (stratified by hookworm infection), 4a and 4b (stratified by *A. lumbricoides* infection), 5a and 5b (stratified by *T. trichiura* infection), and 6a (stratified by type of co-infection). Logistic

regression models can be found in Tables 3c (modeling hookworm infection), 4c (modeling *A. lumbricoides* infection), and 5c (modeling *T. trichiura* infection).

Table 3a. Description of the sample by hookworm infection status[1]

Characteristic	Hookworm Infection		p Value[2]
	Yes (N = 148)	**No (N = 121)**	
Age Group			**0.0239**
4 to 5 years (N = 71)	48 (32.4)	23 (19.0)	
6 to 10 years (N = 105)	57 (38.5)	48 (39.7)	
11 to 14 years (N = 93)	43 (29.1)	50 (41.3)	
Community			0.8749
Bukira (N = 36)	19 (12.8)	17 (14.1)	
Bukunda (N = 8)	5 (3.4)	3 (2.5)	
Busowe (N = 47)	23 (15.5)	24 (19.8)	
Dwaniro (N = 10)	4 (2.7)	6 (5.0)	
Kabonera (N = 33)	20 (13.5)	13 (10.7)	
Kabuwoko (N = 39)	23 (15.5)	16 (13.2)	
Kindulwe (N = 35)	18 (12.2)	17 (14.1)	
Segero (N = 61)	36 (24.3)	25 (20.7)	
Sex			0.4805
Female (N = 142)	81 (54.7)	61 (50.4)	
Male (N = 127)	67 (45.3)	60 (49.6)	
Religion			0.5371
Catholic (N = 171)	98 (66.2)	73 (60.3)	
Christian (N = 84)	42 (28.4)	42 (34.7)	
Muslim (N = 14)	8 (5.4)	6 (5.0)	
Schooling			0.0664
Not Enrolled (N = 45)	29 (19.6)	16 (13.2)	
Nursery (N = 39)	27 (18.2)	12 (9.9)	
Primary (N = 171)	86 (58.1)	85 (70.3)	
Secondary (N = 14)	6 (4.1)	8 (6.6)	
Shoe Usage			0.2905
Never (N = 142)	72 (48.7)	70 (57.9)	
Every Week (N = 13)	7 (4.7)	6 (5.0)	
Every Day (N = 114)	69 (46.6)	45 (37.2)	
Deworming History			0.2082
Past 12 months (N = 89)	54 (36.7)	35 (29.4)	
More than 12 months (N = 177)	93 (63.3)	84 (70.6)	
Bednet Use			0.9917
Uses Net (N = 69)	38 (25.7)	31 (25.6)	
Does not use net (N = 200)	110 (74.3)	90 (74.4)	
Pig Ownership			0.3135
Owns pigs (N = 212)	120 (81.1)	92 (76.0)	

Characteristic	Hookworm Infection		p Value[2]
	Yes (N = 148)	No (N = 121)	
Does not own pigs (N = 57)	28 (18.9)	29 (24.0)	
Head of Household Education			0.5365
None (N = 41)	21 (15.6)	20 (18.4)	
Some Primary School (N = 135)	79 (58.5)	56 (51.4)	
Some Secondary School (N = 68)	35 (25.9)	33 (30.3)	
Head of Household Occupation			0.7005
Farmer (N = 231)	126 (85.1)	105 (86.8)	
Other (N = 38)	22 (14.9)	16 (13.2)	
Malaria History			**0.0308**
Past 12 months (N = 73)	48 (32.4)	25 (20.7)	
More than 12 months (N = 196)	100 (67.6)	96 (79.3)	
Average Dietary Diversity Score	3.4 $\pm$ 1.1	3.5 $\pm$ 1.8	0.4866
Average Socioeconomic Index	8.7 $\pm$ 1.4	8.6 $\pm$ 1.4	0.5525
Average Weight/Height	19.8 $\pm$ 6.2	21.3 $\pm$ 5.6	**0.0441**
Average Body Mass Index	16.8 $\pm$ 4.3	17.0 $\pm$ 3.8	0.6974

[1]Numbers may not sum to 269 due to missing data, and column percentages may not sum to 100% due to rounding.
[2]P value for analysis of variance F-test (continuous variables) or χ^2 test (categorical variables).

Table 3b. Unadjusted associations between study variables and hookworm infection status

Characteristic	N[1]	% Infected with Hookworm	OR (95% CI)[2]
Age Group			
4 to 5 years	71	67.6	1.00
6 to 10 years	105	54.3	0.57 (0.30, 1.07)
11 to 14 years	93	46.2	0.41 (0.22, 0.78)
Community			
Bukira	36	52.8	0.78 (0.34, 1.78)
Bukunda	8	62.5	1.16 (0.25, 5.29)
Busowe	47	48.9	0.67 (0.31, 1.43)
Dwaniro	10	40.0	0.46 (0.12, 1.81)
Kabonera	33	60.6	1.07 (0.45, 2.54)
Kabuwoko	39	59.0	1.00 (0.44, 2.26)
Kindulwe	35	51.4	0.74 (0.32, 1.70)
Segero	61	59.0	1.00
Sex			
Female	142	57.0	1.00
Male	127	52.8	0.84 (0.52, 1.36)
Religion			
Catholic	171	57.3	1.00
Christian	84	50.0	0.75 (0.44, 1.26)
Muslim	14	57.1	0.99 (0.33, 2.99)

Characteristic	N[1]	% Infected with Hookworm	OR (95% CI)[2]
Schooling			
Not Enrolled	45	64.4	1.00
Nursery	39	69.2	1.24 (0.50, 3.10)
Primary	171	50.3	0.56 (0.28, 1.10)
Secondary	14	42.9	0.41 (0.12, 1.40)
Shoe Usage			
Never	142	50.7	1.00
Every Week	13	53.9	1.13 (0.36, 3.54)
Every Day	114	60.5	1.49 (0.91, 2.47)
Deworming History			
Past 12 months	89	60.7	1.39 (0.83, 2.34)
More than 12 months	177	52.5	1.00
Bednet Use			
Uses Net	69	55.1	1.00 (0.58, 1.74)
Does not use net	200	55.0	1.00
Pig Ownership			
Owns pigs	212	56.6	1.35 (0.75, 2.43)
Does not own pigs	57	49.1	1.00
Head of Household Education			
None	41	51.2	1.00
Some Primary School	135	58.5	1.34 (0.67, 2.71)
Some Secondary School	68	51.5	1.01 (0.47, 2.19)
Head of Household Occupation			
Farmer	231	54.6	0.87 (0.44, 1.75)
Other	38	57.9	1.00
Malaria History			
Past 12 months (N = 73)	73	65.8	1.84 (1.05, 3.22)
More than 12 months (N = 196)	196	51.0	1.00
Average Dietary Diversity Score	269	NA	0.93 (0.75, 1.15)
Average Socioeconomic Index	269	NA	1.05 (0.89, 1.25)
Average Weight/Height	269	NA	0.96 (0.92, 1.00)
Average Body Mass Index	269	NA	0.99 (0.93, 1.05)

[1]Numbers may not sum to 269 due to missing data.
[2] Unadjusted odds ratios were calculated using logistic regression.

Age, history of malaria, and weight for height were all significantly associated with hookworm infection. Similar to the findings for all STN infections, the prevalence of hookworm infection declined with age (children ages 6-10 were 57% as likely as

children 4-5 to harbor hookworm infection; children ages 11-14 were only 41% as likely, p = 0.02). Relatedly, the difference in infection prevalence by age was only statistically different among the youngest (4 and 5 years old) and oldest (11 and 14 years old) groups of children. Shoe usage was not significantly associated with hookworm infection, though it appeared as though daily use of shoes increased the risk of infection (OR = 1.49, with never wearing shoes as the referent, and p = 0.07). A history of malaria was associated with an increased risk of hookworm infection (OR = 1.84, p = 0.03). High weight for height was slightly protective against infection (OR = 0.96, p = 0.04). None of the other parameters assessed were associated with hookworm infection.

Table 3c: Logistic Regression Models to Predict Hookworm Infection

Characteristic	**Adjusted OR**	**p**
Model 1		
Age	0.89 (0.83, 0.96)	**0.0028**
Sex	0.92 (0.55, 1.56)	0.7594
Dietary Diversity	0.89 (0.71, 1.12)	0.3282
Socioeconomic Status	1.09 (0.91, 1.31)	0.3425
Model 2		
Malaria History	1.86 (1.03, 3.35)	**0.0396**
Weight/Height	0.95 (0.91, 0.99)	**0.0189**
Model 3		
Deworming History	1.81 (1.00, 3.26)	**0.0489**
Malaria History	1.85 (1.01, 3.40)	**0.0473**
Weight / Height	0.89 (0.82, 0.96)	**0.0017**
BMI	1.12 (1.01, 1.25)	**0.0352**
Pig Ownership	1.37 (0.72, 2.59)	0.3379

Age was the only significant predictor of hookworm infection in the logistic regression model controlling for sex, dietary diversity, and socioeconomic status. This model suggested that older age was protective against hookworm infection, and the association was highly significant (adjusted OR = 0.89; p = 0.003). Backward selection yielded a model with only two significant predictors of hookworm infection: history of

malaria infection and weight for height. Having suffered from malaria within the past year was associated with an 86% increase in the odds of having hookworm infection when controlling for differences in weight for height (p = 0.04). Unsurprisingly, individuals with lower weight for height were more likely to have hookworm (adjusted OR = 0.95, p = 0.02). The five most significant predictors of hookworm infection were deworming history, malaria history, weight for height, BMI, and pig ownership; all predictors except pig ownership were found to be significant in a model controlling for the other parameters mentioned. Children who had been dewormed in the past year and children who had had malaria within the past year were substantially more likely to harbor hookworm infection (adjusted OR = 1.81 and 1.85, respectively). Once again, having a low weight for height, but a higher BMI, was associated with an increase in the odds of hookworm infection. Owning at least one pig raised the odds of having hookworm infection 37% when controlling for the other factors mentioned here, though this was not significant (p = 0.3).

Table 4a. Description of the sample by A. lumbricoides *infection status*[1]

Characteristic	*A. lumbricoides* Infection		p Value[2]
	Yes (N = 133)	**No (N = 136)**	
Age Group			**0.0186**
4 to 5 years (N = 71)	43 (32.3)	28 (20.6)	
6 to 10 years (N = 105)	54 (40.6)	51 (37.5)	
11 to 14 years (N = 93)	36 (27.1)	57 (41.9)	
Community			0.9533
Bukira (N = 36)	16 (12.0)	20 (14.7)	
Bukunda (N = 8)	4 (3.0)	4 (2.9)	
Busowe (N = 47)	26 (19.6)	21 (15.4)	
Dwaniro (N = 10)	5 (3.8)	5 (3.7)	
Kabonera (N = 33)	15 (11.3)	18 (13.2)	
Kabuwoko (N = 39)	18 (13.5)	21 (15.4)	
Kindulwe (N = 35)	16 (12.0)	19 (14.0)	
Segero (N = 61)	33 (24.8)	28 (20.6)	
Sex			0.3543
Female (N = 142)	74 (55.6)	68 (50.0)	
Male (N = 127)	59 (44.4)	68 (50.0)	

Characteristic	*A. lumbricoides* Infection		p Value[2]
	Yes (N = 133)	**No (N = 136)**	
Religion			0.2498
Catholic (N = 171)	81 (60.9)	90 (66.2)	
Christian (N = 84)	47 (35.3)	37 (27.2)	
Muslim (N = 14)	5 (3.8)	9 (6.6)	
Schooling			0.2463
Not Enrolled (N = 45)	26 (19.6)	19 (14.0)	
Nursery (N = 39)	21 (15.8)	18 (13.2)	
Primary (N = 171)	82 (61.7)	89 (65.4)	
Secondary (N = 14)	4 (3.0)	10 (7.4)	
Shoe Usage			**0.0108**
Never (N = 142)	58 (43.6)	84 (61.8)	
Every Week (N = 13)	7 (5.3)	6 (4.4)	
Every Day (N = 114)	68 (51.1)	46 (33.8)	
Deworming History			0.6973
Past 12 months (N = 89)	42 (32.3)	47 (34.6)	
More than 12 months (N = 177)	88 (67.7)	89 (65.4)	
Bednet Use			0.7555
Uses net (N = 69)	33 (24.8)	36 (26.5)	
Does not use net (N = 200)	100 (75.2)	100 (73.5)	
Pig Ownership			0.5149
Owns pigs (N = 212)	107 (80.5)	105 (77.2)	
Does not own pigs (N = 57)	26 (19.6)	31 (22.8)	
Head of Household Education			0.8564
None (N = 41)	20 (16.5)	21 (17.1)	
Some Primary School (N = 135)	69 (57.0)	66 (53.7)	
Some Secondary School (N = 68)	32 (26.5)	36 (29.3)	
Head of Household Occupation			0.1403
Farmer (N = 231)	110 (82.7)	121 (89.0)	
Other (N = 38)	23 (17.3)	15 (11.0)	
Malaria History			**0.0301**
Past 12 months (N = 73)	44 (33.1)	29 (21.3)	
More than 12 months (N = 196)	89 (66.9)	107 (78.7)	
Average Dietary Diversity Score	3.4 $\pm$ 1.1	3.5 $\pm$ 1.2	0.2844
Average Socioeconomic Index	8.6 $\pm$ 1.4	8.7 $\pm$ 1.4	0.8261
Average Weight/Height	19.8 $\pm$ 6.1	21.2 $\pm$ 5.8	**0.0462**
Average Body Mass Index	16.7 $\pm$ 4.2	17.0 $\pm$ 4.0	0.5887

[1]Numbers may not sum to 269 due to missing data, and column percentages may not sum to 100% due to rounding.
[2]P value for analysis of variable F-test (continuous variables) or χ^2 test (categorical variables).

Table 4b. Unadjusted associations between study variables and A. lumbricoides *infection status*

Characteristic	N[1]	% Infected with *A. lumbricoides*	OR (95% CI)[2]
Age Group			
4 to 5 years	71	60.6	1.00
6 to 10 years	105	51.4	0.69 (0.37, 1.27)
11 to 14 years	93	38.7	0.41 (0.22, 0.77)
Community			
Bukira	36	44.4	0.68 (0.30, 1.55)
Bukunda	8	50.0	0.85 (0.19, 3.71)
Busowe	47	55.3	1.05 (0.49, 2.26)
Dwaniro	10	50.0	0.85 (0.22, 3.23)
Kabonera	33	45.5	0.71 (0.30, 1.66)
Kabuwoko	39	46.2	0.73 (0.33, 1.63)
Kindulwe	35	45.7	0.72 (0.31, 1.65)
Segero	61	54.1	1.00
Sex			
Female	142	52.1	1.00
Male	127	46.5	0.80 (0.49, 1.29)
Religion			
Catholic	171	47.4	1.00
Christian	84	56.0	1.41 (0.84, 2.39)
Muslim	14	35.7	0.62 (0.20, 1.92)
Schooling			
Not Enrolled	45	57.8	1.00
Nursery	39	53.9	0.85 (0.36, 2.02)
Primary	171	48.0	0.67 (0.35, 1.31)
Secondary	14	28.6	0.29 (0.08, 1.08)
Shoe Usage			
Never	142	40.9	1.00
Every Week	13	53.9	1.69 (0.54, 5.29)
Every Day	114	59.7	2.14 (1.30, 3.54)
Deworming History			
Past 12 months	89	47.2	0.90 (0.54, 1.51)
More than 12 months	177	49.7	1.00
Bednet Use			
Uses Net	69	47.8	0.92 (0.53, 1.59)
Does not use net	200	50.0	1.00
Pig Ownership			
Owns pigs	212	50.5	1.22 (0.68, 2.19)
Does not own pigs	57	45.6	1.00
Head of Household Education			
None	41	48.8	1.00
Some Primary School	135	51.1	1.10 (0.55, 2.21)

Characteristic	N[1]	% Infected with *A. lumbricoides*	OR (95% CI)[2]
Some Secondary School	68	47.1	0.93 (0.43, 2.03)
Head of Household Occupation			
Farmer	231	47.6	0.59 (0.29, 1.19)
Other	38	60.5	1.00
Malaria History			
Past 12 months	73	60.3	1.82 (1.06, 3.15)
More than 12 months	196	45.4	1.00
Average Dietary Diversity Score	269	NA	0.89 (0.72, 1.10)
Average Socioeconomic Index	269	NA	0.98 (0.83, 1.16)
Average Weight/Height	269	NA	0.96 (0.92, 1.00)
Average Body Mass Index	269	NA	0.98 (0.93, 1.04)

[1] Numbers may not sum to 269 due to missing data.
[2] Unadjusted odds ratios were calculated using logistic regression.

When analyzed by *A. lumbricoides* infection status, age, shoe usage, malaria history, and weight for height were all found to yield significant associations with the outcome. Older children were less likely to harbor an infection ($p = 0.02$). Infection prevalence varied widely by village of residence, and no association was seen between *A. lumbricoides* infection and sex, though slightly fewer of the infected individuals were males (44.4%, $p = 0.35$). Religion, deworming history, bednet use, pig ownership, schooling, and the education level and occupation of the head of the household appeared to have no bearing on *A. lumbricoides* infection status. Shoe usage, however, was significantly associated with infection ($p = 0.01$), and individuals who wore shoes daily were more than twice as likely to be infected with *A. lumbricoides*. Children who had had malaria in the past year were nearly two times as likely to have *A. lumbricoides* infection as those who had not had the disease (OR = 1.82, $p = 0.03$). Uninfected individuals had higher dietary diversity, socioeconomic status, weight for height, and BMI, though only the weight for height association proved to be significant ($p = 0.05$).

Table 4c: Logistic Regression Models to Predict A. lumbricoides *Infection*

Characteristic	**Adjusted OR**	**p**
Model 1		
Age	0.90 (0.83, 0.97)	**0.0041**
Sex	0.87 (0.52, 1.47)	0.6063
Dietary Diversity	0.91 (0.73, 1.14)	0.4244
Socioeconomic Status	0.99 (0.83, 1.19)	0.9374
Model 2		
Malaria History	1.93 (1.08, 3.45)	**0.0255**
Weight / Height	0.94 (0.90, 0.99)	**0.0102**
Model 3		
Malaria History	1.95 (1.07, 3.54)	**0.0285**
Weight / Height	0.89 (0.83, 0.96)	**0.0036**
BMI	1.11 (0.99, 1.23)	0.0642
HH Occupation	0.49 (0.23, 1.04)	0.0631
Religion (Ref: Catholic) Christian Muslim	 1.31 (0.73, 2.35) 0.51 (0.15, 1.69)	 0.3611 0.2683

As was observed in the models of hookworm and STN infection generally, age was the only significant predictor of infection with *A. lumbricoides* when sex, dietary diversity, and socioeconomic status were also considered (p = 0.004). When all parameters were considered in a backward selected model, only malaria history and weight for height were found to be significant predictors of *A. lumbricoides* infection. Having had malaria in the past year increased the odds of being infected by 93% (p = 0.025) when controlling for weight for height; having a higher weight for height lowered the odds of being infected by 6% (p = 0.01) when controlling for malaria history. The five most significant predictors found in the model were malaria history, weight for height, BMI, whether the head of the household was a farmer, and the religion of the household. High BMI again was found to increase the odds of infection (adjusted OR = 1.11), though the effect was not significant (p = 0.06). Interestingly, children who lived in farming households and Muslim households were less likely to be infected (adjusted OR = 0.49 and 0.51, respectively) than children from non-farming households and Catholic

households, but neither effect bore statistical significance when controlling for malaria history, weight for height, and BMI.

Table 5a. Description of the sample by T. trichiura *infection status*[1]

Characteristic	*T. trichiura* Infection		p Value[2]
	Yes (N = 57)	**No (N = 212)**	
Age Group			0.7736
4 to 5 years (N = 71)	17 (29.8)	54 (25.5)	
6 to 10 years (N = 105)	22 (38.6)	83 (39.2)	
11 to 14 years (N = 93)	18 (31.6)	75 (35.4)	
Community			0.6273
Bukira (N = 36)	7 (12.3)	29 (13.7)	
Bukunda (N = 8)	1 (1.8)	7 (3.3)	
Busowe (N = 47)	12 (21.1)	35 (16.5)	
Dwaniro (N = 10)	0 (0.0)	10 (4.7)	
Kabonera (N = 33)	9 (15.8)	24 (11.3)	
Kabuwoko (N = 39)	10 (17.5)	29 (13.7)	
Kindulwe (N = 35)	6 (10.5)	29 (13.7)	
Segero (N = 61)	12 (21.1)	49 (23.1)	
Sex			0.7855
Female (N = 142)	31 (54.4)	111 (52.4)	
Male (N = 127)	26 (45.6)	101 (47.6)	
Religion			0.2956
Catholic (N = 171)	35 (61.4)	136 (64.2)	
Christian (N = 84)	21 (36.8)	63 (29.7)	
Muslim (N = 14)	1 (1.8)	13 (6.1)	
Schooling			0.4424
Not Enrolled (N = 45)	12 (21.1)	33 (15.6)	
Nursery (N = 39)	10 (17.5)	29 (13.7)	
Primary (N = 171)	31 (54.4)	140 (66.0)	
Secondary (N = 14)	4 (7.0)	10 (4.7)	
Shoe Usage			0.2013
Never (N = 142)	26 (45.6)	116 (54.7)	
Every Week (N = 13)	5 (8.8)	8 (3.8)	
Every Day (N = 114)	26 (45.6)	88 (41.5)	
Deworming History			0.7687
Past 12 months (N = 89)	20 (35.1)	69 (33.0)	
More than 12 months (N = 177)	37 (64.9)	140 (67.0)	
Bednet Use			**0.0237**
Uses Net (N = 69)	8 (14.0)	61 (28.8)	
Does not use net (N = 200)	49 (86.0)	151 (71.2)	
Pig Ownership			**0.0098**
Owns pigs (N = 212)	52 (91.2)	160 (75.5)	
Does not own pigs (N = 57)	5 (8.8)	52 (24.5)	

Characteristic	*T. trichiura* Infection		p Value[2]
	Yes (N = 57)	**No (N = 212)**	
Head of Household Education			0.2683
None (N = 41)	5 (9.4)	36 (18.9)	
Some Primary School (N = 135)	32 (60.4)	103 (53.9)	
Some Secondary School (N =68)	16 (30.2)	52 (27.2)	
Head of Household Occupation			0.0908
Farmer (N = 231)	45 (79.0)	186 (87.7)	
Other (N = 38)	12 (21.1)	26 (12.3)	
Malaria History			0.3956
Past 12 months (N = 73)	18 (31.6)	55 (25.9)	
More than 12 months (N = 196)	39 (68.4)	157 (74.1)	
Average Dietary Diversity Score	3.4 $\pm$ 1.1	3.5 $\pm$ 1.8	0.4866
Average Socioeconomic Index	8.7 $\pm$ 1.4	8.6 $\pm$ 1.4	0.5525
Average Weight/Height	19.8 $\pm$ 6.2	21.3 $\pm$ 5.6	**0.0441**
Average Body Mass Index	16.8 $\pm$ 4.3	17.0 $\pm$ 3.8	0.6974

[1]Numbers may not sum to 269 due to missing data, and column percentages may not sum to 100% due to rounding.
[2] P value for analysis of variable F-test (continuous variables) or χ^2 test (categorical variables).

Table 5b. Unadjusted associations between study variables and T. trichiura *infection status*

Characteristic	N[1]	% Infected with *T. trichiura*	OR (95% CI)[2]
Age Group			
4 to 5 years	71	23.9	1.00
6 to 10 years	105	21.0	0.84 (0.41, 1.73)
11 to 14 years	93	19.4	0.76 (0.36, 1.61)
Community			
Bukira	36	19.4	1.19 (0.42, 3.33)
Bukunda	8	12.5	0.70 (0.08, 6.25)
Busowe	47	25.5	1.69 (0.68, 4.16)
Dwaniro	10	0.00	NA
Kabonera	33	27.3	1.84 (0.69, 4.94)
Kabuwoko	39	25.6	1.70 (0.66, 4.38)
Kindulwe	35	17.1	1.02 (0.35, 2.98)
Segero	61	19.7	1.00
Sex			
Female	142	21.8	1.00
Male	127	20.5	0.92 (0.51, 1.66)
Religion			
Catholic	171	20.5	1.00
Christian	84	25.0	1.30 (0.70, 2.40)
Muslim	14	7.1	0.30 (0.04, 2.36)

Characteristic	N[1]	% Infected with *T. trichiura*	OR (95% CI)[2]
Schooling			
Not Enrolled	45	26.7	1.00
Nursery	39	25.6	0.95 (0.36, 2.52)
Primary	171	18.1	0.61 (0.28, 1.31)
Secondary	14	28.6	1.10 (0.29, 4.18)
Shoe Usage			
Never	142	18.3	1.00
Every Week	13	38.5	2.79 (0.84, 9.22)
Every Day	114	22.8	1.32 (0.72, 2.43)
Deworming History			
Past 12 months	89	22.5	1.10 (0.59, 2.03)
More than 12 months	177	20.9	1.00
Bednet Use			
Uses Net	69	11.6	0.40 (0.18, 0.90)
Does not use net	200	24.5	1.00
Pig Ownership			
Owns pigs	212	24.5	3.38 (1.28, 8.91)
Does not own pigs	57	8.8	1.00
Head of Household Education			
None	41	12.2	1.00
Some Primary School	135	23.7	2.24 (0.81, 6.18)
Some Secondary School	68	23.5	2.22 (0.75, 6.59)
Head of Household Occupation			
Farmer	231	19.5	0.52 (0.25, 1.12)
Other	38	31.6	1.00
Malaria History			
Past 12 months (N = 73)	73	24.7	1.32 (0.70, 2.49)
More than 12 months (N = 196)	196	19.9	1.00
Average Dietary Diversity Score	269	NA	0.84 (0.64, 1.12)
Average Socioeconomic Index	269	NA	1.00 (0.82, 1.24)
Average Weight/Height	269	NA	1.02 (0.97, 1.07)
Average Body Mass Index	269	NA	1.05 (0.98, 1.12)

[1] Numbers may not sum to 269 due to missing data.
[2] Unadjusted odds ratios were calculated using logistic regression.

Unlike the associations seen for hookworm and *A. lumbricoides* infection, the associations between demographic and behavioral risk factors and the occurrence of *T. trichiura* infection did not correlate very well with the overall trends for STN infection.

Age, village of residence, sex, religion, deworming history, socioeconomic status, dietary diversity and BMI did not correlate to this infection outcome. Bednet use appeared to protect against *T. trichiura* infection (p = 0.02). Children who owned pigs were more than three times as likely to have *T. trichiura* infection (p = 0.01). Additionally, infected children had a lower weight for height (p = 0.04).

Table 5c: Logistic Regression Models to Predict T. trichiura *Infection*

Characteristic	**Adjusted OR**	**p**
Model 1		
Age	0.97 (0.89, 1.06)	0.5014
Sex	1.10 (0.60, 2.04)	0.7553
Dietary Diversity	0.87 (0.65, 1.15)	0.3250
Socioeconomic Status	1.05 (0.85, 1.30)	0.6530
Model 2		
Pig Ownership	3.04 (1.14, 8.16)	**0.0270**
Bednet Use	0.45 (0.20, 1.02)	0.0563
Model 3		
Bednet Use	0.34 (0.14, 0.80)	**0.0139**
Pig Ownership	3.13 (1.14, 8.58)	**0.0263**
HH Occupation	0.39 (0.17, 0.90)	**0.0273**
Dietary Diversity	0.77 (0.56, 1.04)	0.0896
BMI	1.07 (1.00, 1.15)	0.0620

Age, sex, dietary diversity, and socioeconomic status all were not significant predictors of *T. trichiura* infection when considered in tandem. Pig ownership and bednet use were the only predictors included in the final parsimonious model derived from backward selection. In this model, pig ownership increased the odds of being infected by 204% (p = 0.03). Bednet use appeared to have a protective effect, lowering the risk of infection by 55%. The five most significant predictors also included dietary diversity, BMI, and whether the head of the household was a farmer. Living with a farmer head of household decreased the odds of infection substantially (adjusted OR = 0.39, p = 0.03). Greater dietary diversity lowered the risk of roundworm infection, but the effect was not significant (p = 0.09). The effect of BMI was likewise minimal (p = 0.06).

Table 6a. Description of the sample by type of infection[1]

Characteristic	Type of Infection								p Value[2]
	None (N = 79)	HW Only (N = 39)	AL Only (N = 33)	TT Only (N = 4)	HW+AL (N = 61)	HW+TT (N = 14)	AL+TT (N = 5)	HW+AL+TT (N = 34)	
Age Group									**0.0100**
4 to 5 years (N = 71)	11 (13.9)	12 (30.8)	8 (24.4)	2 (50.0)	23 (37.7)	3 (21.4)	2 (40.0)	10 (29.4)	
6 to 10 years (N = 105)	29 (36.7)	15 (38.5)	16 (48.5)	1 (25.0)	23 (37.7)	6 (42.9)	2 (40.0)	13 (38.2)	
11 to 14 years (N = 93)	39 (49.4)	12 (30.8)	9 (27.3)	1 (25.0)	15 (24.6)	5 (35.7)	1 (20.0)	11 (32.4)	
Community									0.8099
Bukira (N = 36)	11 (13.9)	5 (12.8)	4 (12.1)	1 (25.0)	9 (14.8)	3 (21.4)	1 (20.0)	2 (5.9)	
Bukunda (N = 8)	2 (2.5)	2 (5.1)	1 (3.0)	0 (0.0)	2 (3.3)	0 (0.0)	0 (0.0)	1 (2.9)	
Busowe (N = 47)	16 (20.3)	4 (10.3)	6 (18.2)	0 (0.0)	9 (14.8)	1 (7.1)	2 (40.0)	9 (26.5)	
Dwaniro (N = 10)	4 (5.1)	1 (2.6)	2 (6.1)	0 (0.0)	3 (4.9)	0 (0.0)	0 (0.0)	0 (0.0)	
Kabonera (N = 33)	10 (12.7)	4 (10.3)	3 (9.1)	0 (0.0)	7 (11.5)	4 (28.6)	0 (0.0)	5 (14.7)	
Kabuwoko (N = 39)	9 (11.4)	7 (18.0)	5 (15.2)	2 (50.0)	8 (13.1)	3 (21.4)	0 (0.0)	5 (14.7)	
Kindulwe (N = 35)	12 (15.2)	5 (12.8)	3 (9.1)	1 (25.0)	9 (14.8)	1 (7.1)	1 (20.0)	3 (8.8)	
Segero (N = 61)	15 (19.0)	11 (28.2)	9 (27.3)	0 (0.0)	14 (23.0)	2 (14.3)	1 (20.0)	9 (26.5)	
Sex									0.3363
Female (N = 142)	37 (46.8)	19 (48.7)	17 (51.5)	3 (75.0)	38 (62.3)	9 (64.3)	4 (80.0)	15 (44.1)	
Male (N = 127)	42 (53.2)	20 (51.3)	16 (48.5)	1 (25.0)	23 (37.7)	5 (35.7)	1 (20.0)	19 (55.9)	
Religion									0.8628
Catholic (N = 171	49 (62.0)	29 (74.4)	17 (51.5)	3 (75.0)	41 (67.2)	9 (64.3)	4 (80.0)	19 (55.9)	
Christian (N = 84)	24 (30.4)	7 (18.0)	16 (48.5)	1 (25.0)	16 (26.2)	5 (35.7)	1 (20.0)	14 (41.2)	
Muslim (N = 14)	6 (7.6)	3 (7.7)	0 (0.0)	0 (0.0)	4 (6.6)	0 (0.0)	0 (0.0)	1 (2.9)	
Schooling									**0.0297**
Not Enrolled (N = 45)	10 (12.7)	6 (15.4)	4 (12.1)	1 (25.0)	13 (21.3)	2 (14.3)	1 (20.0)	8 (23.5)	
Nursery (N = 39)	7 (8.9)	9 (23.1)	3 (9.1)	1 (25.0)	10 (16.4)	1 (7.1)	1 (20.0)	7 (20.6)	
Primary (N = 171)	55 (69.6)	22 (56.4)	25 (75.8)	2 (50.0)	38 (62.3)	10 (71.4)	3 (60.0)	16 (47.1)	
Secondary (N = 14)	7 (8.9)	2 (5.1)	1 (3.0)	0 (0.0)	0 (0.0)	1 (7.1)	0 (0.0)	3 (8.8)	
Shoe Usage									**0.0157**
Never (N = 142)	52 (65.8)	24 (61.5)	11 (33.3)	2 (50.0)	29 (47.5)	6 (42.9)	5 (100.0)	13 (38.2)	
Every Week (N = 13)	4 (5.1)	2 (5.1)	2 (6.1)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	5 (14.7)	
Every Day (N = 114)	23 (29.1)	13 (33.3)	20 (60.6)	2 (50.0)	32 (52.5)	8 (57.1)	0 (0.0)	16 (47.1)	
Deworming History									0.8808
Past 12 months (N = 89)	22 (27.9)	20 (51.3)	10 (32.3)	1 (25.0)	17 (28.3)	4 (28.6)	2 (40.0)	13 (38.2)	
More than 12 months (N = 177)	57 (72.1)	19 (48.7)	21 (67.7)	3 (75.0)	43 (71.7)	10 (71.4)	3 (60.0)	21 (61.8)	
Bednet Use									0.1145
Uses net (N = 69)	20 (25.3)	14 (35.9)	10 (30.3)	1 (25.0)	17 (27.9)	1 (7.1)	0 (0.0)	6 (17.7)	
Does not use net (N = 200)	59 (74.7)	25 (64.1)	23 (69.7)	3 (75.0)	44 (72.1)	13 (92.9)	5 (100.0)	28 (82.4)	
Pig Ownership									**0.0407**
Owns Pigs (N = 212)	57 (72.2)	31 (7.5)	26 (78.8)	4 (100.0)	46 (75.4)	13 (92.9)	5 (100.0)	30 (88.2)	
Does not own pigs (N = 57)	22 (27.9)	8 (20.5)	7 (21.2)	0 (0.0)	15 (24.6)	1 (7.1)	0 (0.0)	4 (11.8)	
Head of Household Education									0.5496
None (N = 41)	13 (17.8)	6 (18.8)	7 (24.1)	0 (0.0)	10 (17.5)	2 (14.3)	0 (0.0)	3 (9.4)	
Some Primary School (N = 135)	38 (52.1)	21 (65.6)	13 (44.8)	2 (50.0)	31 (54.4)	5 (35.7)	3 (100.0)	22 (68.9)	
Some Secondary School (N = 68)	22 (30.1)	5 (15.6)	9 (31.0)	2 (50.0)	16 (28.1)	7 (50.0)	0 (0.0)	7 (21.9)	
Malaria History									0.2060
Past 12 months (N = 73)	14 (17.7)	12 (30.8)	10 (30.3)	0 (0.0)	19 (31.2)	3 (21.4)	1 (20.0)	14 (41.2)	
More than 12 months (N = 196)	65 (82.3)	27 (69.2)	23 (69.7)	4 (100.0)	42 (68.8)	11 (78.6)	4 (80.0)	20 (58.8)	
Infection Intensity									**<0.0001**
None (N = 79)	79 (100.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	
Light (N = 143)	0 (0.0)	36 (100.0)	33 (100.0)	4 (100.0)	56 (100.0)	14 (100.0)	4 (100.0)	28 (100.0)	
Treatment Efficacy									0.0949
Cured (N = 99)	NA	13 (54.2)	25 (86.2)	2 (100.0)	30 (57.7)	10 (71.4)	3 (75.0)	16 (55.2)	
Not Cured (N = 55)	NA	11 (45.8)	4 (13.8)	0 (0.0)	22 (42.3)	4 (28.6)	1 (25.0)	13 (44.8)	
Moderate/Heavy (N = 11)	0 (0.0)	3 (7.7)	0 (0.0)	0 (0.0)	5 (8.2)	0 (0.0)	1 (20.0)	6 (17.7)	
Average Dietary Diversity Score	3.5 ± 1.3	3.5 ± 1.1	3.4 ± 1.1	4.0 ± 0.8	3.4 ± 1.0	3.2 ± 0.9	3.2 ± 0.4	3.2 ± 1.2	0.8189
Average Socioeconomic Index	8.6 ± 1.4	8.9 ± 1.3	8.6 ± 1.2	8.5 ± 1.3	8.6 ± 1.6	8.5 ± 1.2	9.0 ± 1.2	8.7 ± 1.4	0.9015
Average Weight/Height	22.1 ± 5.5	19.8 ± 5.7	19.6 ± 5.5	19.0 ± 4.3	19.0 ± 5.4	21.0 ± 7.6	22.0 ± 7.2	21.0 ± 7.6	0.1004

Characteristic	Type of Infection								p Value[2]
	None (N = 79)	HW Only (N = 39)	AL Only (N = 33)	TT Only (N = 4)	HW+AL (N = 61)	HW+TT (N = 14)	AL+TT (N = 5)	HW+AL+TT (N = 34)	
Age Group									**0.0100**
Average Body Mass Index	17.2 ± 3.6	16.6 ± 4.5	16.2 ± 4.0	16.1 ± 2.9	16.3 ± 3.1	17.3 ± 5.2	19.0 ± 6.7	17.6 ± 5.5	0.6075

[1] Numbers may not sum to 269 due to missing data, and column percentages may not sum to 100% due to rounding.
[2] P value for analysis of variable F-test (continuous variables) or χ^2 test (categorical variables)

When each type of infection (the three types of mono-infection, hookworm-whipworm co-infection, hookworm-roundworm co-infection, roundworm-whipworm co-infection, and triple infection) was analyzed separately, only hookworm and *A. lumbricoides* mono-infection, hookworm-roundworm co-infection, and infection by all three STN species produced reportable findings (further analysis of these variants was conducted, though is not pictured in a table here). This may be due to sample size limitations, as the sample in this study was not large enough to reasonably stratify participants into eight subgroups. Hookworm mono-infection was more common among individuals who slept under a bednet (p = 0.03), and among individuals who had been dewormed within the past year (OR = 2.68, p = 0.04). Individuals with *A. lumbricoides* mono-infection were disproportionately likely to be cured; only 9% of these infections were not cured (p = 0.006). No Muslim children harbored *A. lumbricoides* as a mono-infection. Over 90% of all hookworm-roundworm co-infections were of light intensity, though this co-infection also accounted for 33.3% of all moderate and heavy infections. Nearly two thirds of all hookworm-roundworm co-infections occurred in females, but the difference was not of statistical significance (p = 0.09). Moderate and heavy infections were significantly more likely to be triple infections; 40% of all moderate and heavy infections were triple infections (p = 0.02). A history of malaria was associated with increased prevalence of this co-infection; individuals with this condition were nearly twice as likely as other study participants to have suffered malaria within the past year

(p = 0.05). A logistic regression model of hookworm mono-infection found that deworming history was the only significant predictor of developing this type of infection: children who had been dewormed in the past year were 2.68 times as likely to harbor hookworm mono-infection (p = 0.05). Individuals with moderate or heavy infections were over 5 times as likely to have a triple infection as any other infection type (p = 0.02). The lowest cure rates were seen in hookworm mono-infection (54.2%) and triple STN infection (55.2%). Both *A. lumbricoides* and *T. trichiura* were more likely to be cured when occurring as mono-infections, and least likely to be cured when occurring as triple infections.

Predictors of Treatment Success, Light Infection Intensity, and Low Polyparasitism of Infection

Bivariate analyses on the predictors of treatment success can be found in Tables 7a and 7b; logistic regression models to predict this outcome are reported in Table 7c. Tables 8a, 8b, and 8c provide information on bivariate and logistic regression associations that predict infection intensity; the same predictions for polyparasitism of infection are reported in Tables 9a, 9b, and 9c.

Table 7a. Description of the sample of infected individuals by treatment effectiveness[1]

Characteristic	Infection Cured		p Value[2]
	Yes (N = 99)	**No (N = 55)**	
Age Group			0.9023
4 to 5 years (N = 50)	31 (31.3)	19 (34.6)	
6 to 10 years (N = 62)	41 (41.4)	21 (38.2)	
11 to 14 years (N = 42)	27 (27.3)	15 (27.3)	
Community			0.1768
Bukira (N = 17)	15 (15.2)	2 (3.6)	
Bukunda (N = 4)	3 (3.0)	1 (1.8)	
Busowe (N = 26)	19 (19.2)	7 (12.7)	
Dwaniro (N = 5)	3 (3.0)	2 (3.6)	

Characteristic	Infection Cured		p Value[2]
	Yes (N = 99)	**No (N = 55)**	
Kabonera (N = 19)	9 (9.1)	10 (18.2)	
Kabuwoko (N = 24)	16 (16.2)	8 (14.6)	
Kindulwe (N = 19)	9 (9.1)	10 (18.2)	
Segero (N = 40)	25 (25.3)	15 (27.3)	
Sex			0.3205
Female (N = 87)	53 (53.5)	34 (61.8)	
Male (N = 67)	46 (46.5)	21 (38.2)	
Religion			**0.0457**
Catholic (N = 97)	64 (64.7)	33 (60.0)	
Christian (N = 51)	34 (34.3)	17 (30.9)	
Muslim (N = 6)	1 (1.0)	5 (9.1)	
Schooling			0.9724
Not Enrolled (N = 29)	18 (18.2)	11 (20.0)	
Nursery (N = 26)	16 (16.2)	10 (18.2)	
Primary (N = 93)	61 (61.6)	32 (58.2)	
Secondary (N = 6)	4 (4.0)	2 (3.6)	
Shoe Usage			0.6421
Never (N = 72)	49 (49.5)	23 (41.8)	
Every Week (N = 7)	4 (4.0)	3 (5.5)	
Every Day (N = 75)	46 (46.5)	29 (52.7)	
Deworming History			0.5271
Past 12 months (N = 51)	31 (32.0)	20 (37.0)	
More than 12 months (N = 100)	66 (68.0)	34 (63.0)	
Bednet Use			0.5399
Uses net (N = 38)	26 (26.3)	12 (21.8)	
Does not use net (N = 116)	73 (73.7)	43 (78.2)	
Pig Ownership			0.3832
Owns Pigs (N = 126)	83 (83.8)	43 (78.2)	
Does not own pigs (N = 28)	16 (16.2)	12 (21.8)	
Head of Household Education			0.5047
None (N = 21)	14 (16.1)	7 (13.7)	
Some Primary School (N = 79)	52 (59.8)	27 (52.9)	
Some Secondary School (N = 38)	21 (24.1)	17 (33.3)	
Head of Household Occupation			0.8979
Farmer (N = 128)	82 (82.8)	46 (83.6)	
Other (N = 26)	17 (17.2)	9 (16.7)	
Malaria History			0.5637
Past 12 months (N = 46)	28 (28.3)	18 (32.7)	
More than 12 months (N = 108)	71 (71.7)	37 (67.3)	
Infection Intensity			0.1762
Light (N = 143)	94 (94.6)	49 (89.1)	
Moderate/Heavy (N = 11)	5 (5.1)	6 (10.9)	

Characteristic	Infection Cured		p Value[2]
	Yes (N = 99)	**No (N = 55)**	
Multiplicity			0.2226
One Species (N = 55)	40 (40.4)	15 (27.3)	
Two Species (N = 70)	43 (43.4)	27 (49.1)	
Three Species (N = 29)	16 (16.2)	13 (23.6)	
Average Dietary Diversity Score	3.4 ± 1.1	3.4 ± 1.0	0.7273
Average Socioeconomic Index	8.5 ± 1.3	9.1 ± 1.6	**0.0155**
Average Weight/Height	19.8 ± 5.9	19.6 ± 7.1	0.8579
Average Body Mass Index	16.8 ± 4.6	16.5 ± 4.6	0.7207
Average Time between Last Meal & Treatment	3.9 ± 2.8	3.3 ± 2.1	0.1518

[1] Numbers may not sum to 269 due to missing data, and column percentages may not sum to 100% due to rounding.
[2] P value for analysis of variable F-test (continuous variables) or χ^2 test (categorical variables).

Table 7b. Unadjusted associations between study variables and treatment effectiveness

Characteristic	N[1]	% Cured	OR (95% CI)[2]
Age Group			
4 to 5 years	50	62.0	1.00
6 to 10 years	62	66.1	1.20 (0.55, 2.60)
11 to 14 years	42	64.3	1.10 (0.47, 2.58)
Community			
Bukira	17	88.2	4.50 (0.90, 22.47)
Bukunda	4	75.0	1.80 (0.17, 18.91)
Busowe	26	73.1	1.63 (0.56, 4.78)
Dwaniro	5	60.0	0.90 (0.14, 6.02)
Kabonera	19	47.4	0.54 (0.18, 1.63)
Kabuwoko	24	66.7	1.20 (0.41, 3.47)
Kindulwe	19	47.4	0.54 (0.18, 1.63)
Segero	40	62.5	1.00
Sex			
Female	87	60.9	1.00
Male	67	68.7	1.41 (0.72, 2.75)
Religion			
Catholic	97	66.0	1.00
Christian	51	66.7	1.01 (0.50, 2.11)
Muslim	6	16.7	0.10 (0.01, 0.92)
Schooling			
Not Enrolled	29	62.1	1.00
Nursery	26	61.5	0.98 (0.33, 2.91)
Primary	93	65.6	1.17 (0.49, 2.76)
Secondary	6	66.7	1.22 (0.19, 7.82)

Characteristic	N[1]	% Cured	OR (95% CI)[2]
Shoe Usage			
Never	72	68.1	1.00
Every Week	7	57.1	0.63 (0.13, 3.03)
Every Day	75	61.3	0.75 (0.38, 1.47)
Deworming History			
Past 12 months	51	60.8	0.80 (0.40, 1.61)
More than 12 months	100	66.0	1.00
Bednet Use			
Uses Net	38	68.4	1.28 (0.59, 2.79)
Does not use net	116	62.9	1.00
Pig Ownership			
Owns pigs	126	65.9	1.45 (0.63, 3.33)
Does not own pigs	28	57.1	1.00
Head of Household Education			
None	21	66.7	1.00
Some Primary School	79	65.8	0.96 (0.35, 2.67)
Some Secondary School	38	55.3	0.62 (0.20, 1.87)
Head of Household Occupation			
Farmer	128	64.1	0.94 (0.39, 2.29)
Other	26	65.4	1.00
Malaria History			
Past 12 months	46	60.9	0.81 (0.40, 1.65)
More than 12 months	108	65.7	1.00
Infection Intensity			
Light	143	65.7	1.00
Moderate/Heavy	11	45.5	0.43 (0.13, 1.50)
Multiplicity of Infection			
One species	55	72.7	1.00
Two species	70	61.4	0.60 (0.28, 1.28)
Three species	29	55.2	0.46 (0.18, 1.18)
Average Dietary Diversity Score	154	NA	0.95 (0.70, 1.29)
Average Socioeconomic Index	154	NA	0.74 (0.57, 0.95)
Average Weight/Height	154	NA	1.01 (0.95, 1.06)
Average Body Mass Index	154	NA	1.01 (0.94, 1.09)
Average Time Between Last Meal & Treatment	154	NA	1.12 (0.96, 1.31)

[1]Numbers may not sum to 190 (total infected) due to missing data.
[2] Unadjusted odds ratios were calculated using logistic regression.

Interestingly, the bivariate analysis assessing associations between demographic and behavioral factors on treatment effectiveness found that only religion and socioeconomic status correlated significantly with the outcome. Muslim individuals were significantly less likely than Catholic individuals to have a cured infection (OR = 0.10; p = 0.045); the difference between Christian individuals and Catholic ones was minor (OR = 1.01). Age, school enrollment, and shoe usage did not appear to be related to treatment effectiveness. Treatment success varied widely by village; some saw cure rates below 50% while others saw cure rates approaching 90%. Though the differences by village were not statistically significant, they were substantial. Treatment was more likely to be successful in males, but not significantly so (OR = 1.41, p = 0.3). Bednet use, a history of malaria, deworming history, and factors related to the head of the household did not impact treatment success. Dietary diversity, weight for height, and BMI appeared to have minimal and unclear effects on treatment success. Higher socioeconomic status, however, was surprisingly associated with a lower likelihood of treatment success (OR = 0.74, p = 0.015). Average time between the last meal and treatment was not significantly associated with treatment success.

Table 7c: Logistic Regression Models to Predict Treatment Failure

Characteristic	Adjusted OR	p
Model 1		
Age	1.01 (0.91, 1.11)	0.8639
Sex	0.71 (0.36, 1.41)	0.3237
Dietary Diversity	1.05 (0.76, 1.45)	0.7629
Socioeconomic Status	1.35 (1.05, 1.75)	**0.0195**
Model 2		
Socioeconomic Status	1.40 (1.08, 1.82)	**0.0116**
Model 3		
Socioeconomic Status	1.58 (1.18, 2.11)	**0.0020**
Time between Last Meal & Treatment	0.88 (0.73, 1.05)	0.1660
Sex	0.47 (0.21, 1.09)	0.0783
Pig Ownership	0.29 (0.10, 0.82)	**0.0200**
Multiplicity Two Three	 2.26 (0.92, 5.59) 4.02 (1.31, 12.40)	 0.0772 **0.0153**

The first logistic regression, which assessed the predictive value of age, sex, dietary diversity, and socioeconomic status on treatment effectiveness, found that only socioeconomic status served as a significant predictor. Surprisingly, higher socioeconomic status was associated with treatment failure, and this finding was significant (OR = 1.35, p = 0.02). In the second logistic regression model, only socioeconomic status was significant, but its effect was attenuated slightly (OR = 1.4). The five most important predictors of treatment effectiveness, beyond socioeconomic status, included sex, pig ownership, polyparasitism of the infection, and the average time between the last meal and treatment. Females were more than 50% more likely to suffer from treatment failure, but the effect was not significant (p = 0.08). The longer the time lag between the last meal and treatment, the greater the success rate of the treatment, but this was also not significant (p = 0.2). Pig owners were more likely to have successful treatment (OR = 0.29, p = 0.02). Interestingly, increasing polyparasitism was associated with treatment failure; individuals with three infections

were four times as likely as those harboring mono-infection to have unsuccessful treatment (p = 0.015).

Table 8a. Description of the sample by infection intensity[1]

Characteristic	Infection Intensity			p Value[2]
	None (N = 79)	**Light (N = 175)**	**Moderate/Heavy (N = 15)**	
Age Group				**0.0037**
4 to 5 years (N = 71)	11 (13.9)	54 (30.9)	6 (40.0)	
6 to 10 years (N = 105)	29 (36.7)	69 (39.4)	7 (46.7)	
11 to 14 years (N = 93)	39 (49.4)	52 (29.7)	2 (13.3)	
Community				0.9416
Bukira (N = 36)	11 (13.9)	23 (13.1)	2 (13.3)	
Bukunda (N = 8)	2 (2.5)	6 (3.4)	0 (0.0)	
Busowe (N = 47)	16 (20.3)	28 (16.0)	3 (20.0)	
Dwaniro (N = 10)	4 (5.1)	6 (3.4)	0 (0.0)	
Kabonera (N = 33)	10 (12.7)	20 (11.4)	3 (20.0)	
Kabuwoko (N = 39)	9 (11.4)	29 (16.6)	1 (6.7)	
Kindulwe (N = 35)	12 (15.2)	22 (12.6)	1 (6.7)	
Segero (N = 61)	15 (19.0)	41 (23.4)	5 (33.3)	
Sex				0.3547
Female (N = 142)	37 (46.8)	98 (56.0)	7 (46.7)	
Male (N = 127)	42 (53.2)	77 (44.0)	8 (53.3)	
Religion				0.3292
Catholic (N = 171)	49 (62.0)	112 (64.0)	10 (66.7)	
Christian (N = 84)	24 (30.4)	57 (32.6)	3 (20.0)	
Muslim (N = 14)	6 (7.6)	6 (3.4)	2 (13.3)	
Schooling				0.1343
Not Enrolled (N = 45)	10 (12.7)	31 (17.7)	4 (26.7)	
Nursery (N = 39)	7 (8.9)	28 (16.0)	4 (26.7)	
Primary (N = 171)	55 (69.6)	109 (62.3)	7 (46.7)	
Secondary (N = 14)	7 (8.9)	7 (4.0)	0 (0.0)	
Shoe Usage				**0.0267**
Never (N = 142)	52 (65.8)	83 (47.4)	7 (46.7)	
Every Week (N = 13)	4 (5.1)	7 (4.0)	2 (13.3)	
Every Day (N = 114)	23 (29.1)	85 (48.6)	6 (40.0)	
Deworming History				0.4240
Past 12 months (N = 89)	22 (27.9)	61 (35.5)	6 (40.0)	
More than 12 months (N = 177)	57 (72.2)	111 (64.5)	9 (60.0)	
Bednet Use				0.8638
Uses net (N = 69)	20 (25.3)	46 (26.3)	3 (20.0)	
Does not use net (N = 200)	59 (74.7)	129 (73.7)	12 (80.0)	
Pig Ownership				0.1997
Owns pigs (N = 212)	57 (72.2)	142 (81.1)	13 (86.7)	

Characteristic	Infection Intensity			p Value[2]
	None (N = 79)	Light (N = 175)	Moderate/Heavy (N = 15)	
Does not own pigs (N = 57)	22 (27.9)	33 (18.9)	2 (13.3)	
Head of Household Education				0.5928
None (N = 41)	13 (17.8)	27 (17.1)	1 (7.7)	
Some Primary School (N = 135)	38 (52.1)	87 (55.1)	10 (76.9)	
Some Secondary School (N = 68)	22 (30.1)	44 (27.9)	2 (15.4)	
Head of Household Occupation				0.6910
Farmer (N = 231)	70 (88.6)	148 (84.6)	13 (86.7)	
Other (N = 38)	9 (11.4)	27 (15.4)	2 (13.3)	
Malaria History				**0.0026**
Past 12 months (N = 73)	14 (17.7)	50 (28.6)	9 (60.0)	
More than 12 months (N = 196)	65 (82.3)	125 (71.4)	6 (40.0)	
Multiplicity				**0.0475**
One species (N = 76)	NA	73 (41.7)	3 (20.0)	
Two species (N = 80)	NA	74 (42.3)	6 (40.0)	
Three species (N = 34)	NA	28 (16.0)	6 (40.0)	
Average Dietary Diversity Score	3.5 $\pm$ 1.3	3.4 $\pm$ 1.0	3.1 $\pm$ 1.6	0.3561
Average Socioeconomic Index	8.6 $\pm$ 1.4	8.7 $\pm$ 1.4	8.4 $\pm$ 1.4	0.5888
Average Weight/Height	22.1 $\pm$ 5.5	20.0 $\pm$ 6.2	18.4 $\pm$ 4.2	**0.0106**
Average Body Mass Index	17.2 $\pm$ 3.6	16.7 $\pm$ 4.5	16.7 $\pm$ 2.1	0.6929

[1] Numbers may not sum to 269 due to missing data, and column percentages may not sum to 100% due to rounding.
[2] P value for analysis of variable F-test (continuous variables) or χ^2 test (categorical variables).

Table 8b. Unadjusted associations between study variables and infection intensity

Characteristic	N[1]	% Light Infection	OR (95% CI)[2]
Age Group			
4 to 5 years	60	90.0	1.00
6 to 10 years	76	90.8	1.10 (0.35, 3.45)
11 to 14 years	54	96.3	2.89 (0.56, 14.97)
Community			
Bukira	25	92.0	1.09 (0.20, 6.01)
Bukunda	6	100.0	NA
Busowe	31	90.3	0.88 (0.20, 3.96)
Dwaniro	6	100.0	NA
Kabonera	23	87.0	0.63 (0.14, 2.88)
Kabuwoko	30	96.7	2.73 (0.31, 24.53)
Kindulwe	23	95.7	2.08 (0.23, 18.80)
Segero	46	89.1	1.00
Sex			
Female	105	93.3	1.00
Male	85	90.6	0.69 (0.24, 1.98)

Characteristic	N[1]	% Light Infection	OR (95% CI)[2]
Religion			
Catholic	122	91.8	1.00
Christian	60	95.0	1.70 (0.45, 6.41)
Muslim	8	75.0	0.27 (0.05, 1.51)
Schooling			
Not Enrolled	35	88.6	1.00
Nursery	32	87.5	0.74 (0.17, 3.20)
Primary	116	94.0	1.64 (0.45, 5.91)
Secondary	7	100.0	NA
Shoe Usage			
Never	90	92.2	1.00
Every Week	9	77.8	0.30 (0.05, 1.70)
Every Day	91	93.4	1.20 (0.39, 3.71)
Deworming History			
Past 12 months	67	91.0	0.82 (0.28, 2.43)
More than 12 months	120	92.5	1.00
Bednet Use			
Uses Net	49	93.9	1.43 (0.39, 5.28)
Does not use net	141	91.5	1.00
Pig Ownership			
Owns pigs	155	91.6	0.66 (0.14, 3.08)
Does not own pigs	35	94.3	1.00
Head of Household Education			
None	28	96.4	1.00
Some Primary School	97	89.7	0.32 (0.04, 2.63)
Some Secondary School	46	95.7	0.82 (0.07, 9.42)
Head of Household Occupation			
Farmer	161	91.9	0.84 (0.18, 3.95)
Other	29	93.1	1.00
Malaria History			
Past 12 months	59	84.8	0.27 (0.09, 0.79)
More than 12 months	131	95.4	1.00
Multiplicity of Infection			
One species	76	96.1	1.00
Two species	80	92.5	0.51 (0.12, 2.10)
Three species	34	82.4	0.19 (0.05, 0.82)
Average Dietary Diversity Score	190	NA	1.49 (0.81, 2.76)
Average Socioeconomic Index	190	NA	1.17 (0.80, 1.70)
Average Weight/Height	190	NA	1.05 (0.95, 1.16)
Average Body Mass Index	190	NA	1.00 (0.89, 1.14)

[1]Numbers may not sum to 190 (total infected) due to missing data.
[2] Unadjusted odds ratios were calculated using logistic regression.

A bivariate analysis on the relationships between demographic and behavioral factors and the resulting intensity of infection suggested that age, shoe usage, malaria history, polyparasitism of the infection, and weight for height all bore significant associations to the outcome. Older children tended to have lighter infections than younger children (OR of light infection among children 11-14 years compared to children 4-5 years = 2.89; OR among children 6-10 years compared to children 4-5 years = 1.10, p = 0.004). Infection intensity varied widely among infected individuals in the different villages. Schooling was not associated with infection intensity, but none of the secondary school children studied harbored moderate or heavy STN infections. Notably, the children who wore their shoes weekly were more likely to have heavier STN infections than both those who did not wear shoes at all and those who wore them daily (p = 0.03). No difference in infection intensity was observed when considered by deworming treatment history. Though bednet use appeared to not have a meaningful relationship with infection intensity, having had malaria in the past year significantly increased the odds of having a heavier STN infection (OR of having a light infection = 0.27, p = 0.003). Factors related to the head of the household did not appear to relate to infection intensity of the child; socioeconomic status, pig ownership, dietary diversity, and BMI were likewise not implicated in this outcome. In contrast, polyparasitism of the infection was significantly associated with infection intensity (p = 0.05): light infections were increasingly less common in individuals harboring multiple STN species (OR of light infection among those with duplicitous infections = 0.51; OR among those with three STN species = 0.19). Perhaps not surprisingly, weight for height was lower among children with heavier infections (p = 0.01).

Table 8c: Logistic Regression Models to Predict Moderate/Heavy Infection

Characteristic	Adjusted OR	p
Model 1		
Age	0.86 (0.72, 1.03)	0.1000
Sex	1.46 (0.50, 4.27)	0.4931
Dietary Diversity	0.68 (0.37, 1.23)	0.2021
Socioeconomic Status	0.89 (0.61, 1.29)	0.5291
Model 2		
Malaria History	6.66 (1.72, 25.80)	**0.0060**
SES Score	0.71 (0.44, 1.14)	0.1524
Multiplicity Two STNs Three STNs	 2.20 (0.40, 12.23) 7.51 (1.34, 42.05)	 0.3683 **0.0219**
Model 3		
Deworming History	1.39 (0.37, 5.22)	0.6234
HH Education Some Primary School Some Secondary School	 3.02 (0.33, 27.72) 1.68 (0.13, 21.59)	 0.3279 0.6897
Malaria History	7.12 (1.77, 28.67)	**0.0057**
SES Score	0.71 (0.44, 1.15)	0.1613
Multiplicity Two STNs Three STNs	 2.21 (0.38, 12.66) 6.51 (1.13, 37.47)	 0.3751 **0.0358**

Adjusted analyses found no significant predictors of moderate/heavy infection when considering only age, sex, dietary diversity, and socioeconomic status. When all parameters were considered, the most parsimonious model included malaria history, socioeconomic status, and multiplicity as key predictors of moderate/heavy infection. In this model, having had malaria within the past year was associated with a 566% increased risk of having a heavier infection (p = 0.006). Additionally, have a triple co-infection increased the risk of having a heavier worm burden by 551% when compared to individuals harboring mono-infections (p = 0.035). The top five predictors of heavier infections also included deworming history and education level of the head of the household, but neither of these parameters bore a significant association to the outcome.

Table 9a. Description of the sample by multiplicity of infection[1]

Characteristic	Number of Infections				p Value[2]
	0 (N = 79)	**1 (N = 76)**	**2 (N = 80)**	**3 (N = 34)**	
Age Group					**0.0214**
4 to 5 years (N = 71)	11 (13.9)	22 (29.0)	28 (35.0)	10 (29.4)	
6 to 10 years (N = 105)	29 (36.7)	32 (42.1)	31 (38.8)	13 (38.2)	
11 to 14 years (N = 93)	39 (49.4)	22 (29.0)	21 (26.3)	11 (32.4)	
Community					0.9567
Bukira (N = 36)	11 (13.9)	10 (13.2)	13 (16.3)	2 (5.9)	
Bukunda (N = 8)	2 (2.5)	3 (4.0)	2 (2.5)	1 (2.9)	
Busowe (N = 47)	16 (20.3)	10 (13.2)	12 (15.0)	9 (26.5)	
Dwaniro (N = 10)	4 (5.1)	3 (4.0)	3 (3.8)	0 (0.0)	
Kabonera (N = 33)	10 (12.7)	7 (9.2)	11 (13.8)	5 (14.7)	
Kabuwoko (N = 39)	9 (11.4)	14 (18.4)	11 (13.8)	3 (8.8)	
Kindulwe (N = 35)	12 (15.2)	9 (11.8)	11 (13.8)	3 (8.8)	
Segero (N = 61)	15 (19.0)	20 (26.3)	17 (21.3)	9 (26.5)	
Sex					0.1082
Female (N = 142)	37 (46.8)	39 (51.3)	51 (63.8)	15 (44.1)	
Male (N = 127)	42 (53.2)	37 (48.7)	29 (36.3)	19 (55.9)	
Religion					0.7514
Muslim (N = 14)	6 (7.6)	3 (4.0)	4 (5.0)	1 (2.9)	
Christian (N = 84)	24 (30.4)	24 (31.6)	22 (27.5)	14 (41.2)	
Catholic (N = 171)	49 (62.0)	49 (64.5)	54 (67.5)	19 (55.9)	
Schooling					0.1681
Not Enrolled (N = 45)	10 (12.7)	11 (14.5)	16 (20.0)	8 (23.5)	
Nursery (N = 39)	7 (8.9)	13 (17.1)	12 (15.0)	7 (20.6)	
Primary (N = 171)	55 (69.6)	49 (64.5)	51 (63.8)	16 (47.1)	
Secondary (N = 14)	7 (8.9)	3 (4.0)	1 (1.3)	3 (8.8)	
Shoe Usage					**0.0030**
Never (N = 142)	52 (65.8)	37 (48.7)	40 (50.0)	13 (38.2)	
Every Week (N = 13)	4 (5.1)	4 (5.3)	0 (0.0)	5 (14.7)	
Every Day (N = 114)	23 (29.1)	35 (46.1)	40 (50.0)	16 (47.1)	
Deworming History					0.2124
Past 12 months (N = 89)	22 (27.9)	31 (41.9)	23 (29.1)	13 (38.2)	
More than 12 months (N = 177)	57 (72.2)	43 (58.1)	56 (70.9)	21 (61.8)	
Bednet Use					0.3013
Uses net (N = 69)	20 (25.3)	25 (32.9)	18 (22.5)	6 (17.7)	
Does not use net (N = 200)	59 (74.7)	51 (67.1)	62 (77.5)	28 (82.4)	
Pig Ownership					0.2540
Owns Pigs (N = 212)	57 (72.2)	61 (80.3)	64 (80.0)	30 (88.2)	
Does not own pigs (N = 57)	22 (27.9)	15 (19.7)	16 (20.0)	4 (11.8)	
Head of Household Education					0.6971
None (N = 41)	13 (17.8)	13 (20.0)	12 (16.2)	3 (9.4)	
Some Primary School (N = 135)	38 (52.1)	36 (55.4)	39 (52.7)	22 (68.8)	
Some Secondary School (N = 68)	22 (30.1)	16 (24.6)	23 (31.1)	7 (21.9)	

Characteristic	Number of Infections				p Value[2]
	0 (N = 79)	**1 (N = 76)**	**2 (N = 80)**	**3 (N = 34)**	
Head of Household Occupation					0.3418
Farmer (N = 231)	70 (88.6)	67 (88.2)	68 (85.0)	26 (76.5)	
Other (N = 38)	9 (11.4)	9 (11.8)	12 (15.0)	8 (23.5)	
Malaria History					0.0669
Past 12 months (N = 73)	14 (17.7)	22 (29.0)	23 (28.8)	14 (41.2)	
More than 12 months (N = 196)	65 (82.3)	54 (71.1)	57 (71.3)	20 (58.8)	
Infection Intensity					**0.0475**
Light (N = 175)	NA	73 (96.1)	74 (92.5)	28 (82.4)	
Moderate/Heavy (N = 15)	NA	3 (3.9)	6 (7.5)	6 (17.6)	
Treatment Efficacy					0.2226
Cured (N = 99)	NA	40 (72.7)	43 (61.4)	16 (55.2)	
Not Cured (N = 55)	NA	15 (27.3)	27 (38.6)	13 (44.8)	
Average Dietary Diversity Score	3.5 $\pm$ 1.3	3.5 $\pm$ 1.1	3.4 $\pm$ 1.0	3.2 $\pm$ 1.2	0.5724
Average Socioeconomic Index	8.6 $\pm$ 1.4	8.8 $\pm$ 1.3	8.6 $\pm$ 1.5	8.7 $\pm$ 1.4	0.7724
Average Weight/Height	22.1 $\pm$ 5.5	19.6 $\pm$ 5.5	19.5 $\pm$ 5.9	21.0 $\pm$ 7.6	**0.0203**
Average Body Mass Index	17.2 $\pm$ 3.6	16.4 $\pm$ 4.2	16.6 $\pm$ 3.8	17.6 $\pm$ 5.5	0.4026

[1] Numbers may not sum to 269 due to missing data, and column percentages may not sum to 100% due to rounding.
[2] P value for analysis of variance F-test (continuous variables) or χ^2 test (categorical variables).

Table 9b. Unadjusted associations between study variables and multiplicity[1]

Characteristic	N	% Mono-infection	OR (95% CI)[2]	% Double Infection	OR (95% CI)	% Triple Infection	OR (95% CI)
Age Group							
4 to 5 years (N = 71)	60	36.7	1.00	46.7	1.00	16.7	1.00
6 to 10 years (N = 105)	76	42.1	1.26 (0.63, 2.52)	40.8	0.79 (0.40, 1.56)	17.1	1.16 (0.61, 2.18)
11 to 14 years (N = 93)	54	40.7	1.19 (0.56, 2.53)	38.9	0.73 (0.35, 1.53)	20.4	1.04 (0.52, 2.06)
Community							
Bukira (N = 36)	25	40.0	0.87 (0.32, 2.33)	52.0	1.85 (0.69, 4.96)	8.0	1.06 (0.43, 2.60)
Bukunda (N = 8)	6	50.0	1.30 (0.24, 7.14)	33.3	0.85 (0.14, 5.16)	16.7	1.22 (0.24, 6.06)
Busowe (N = 47)	31	32.3	0.62 (0.24, 1.60)	38.7	1.08 (0.42, 2.75)	29.0	0.54 (0.23, 1.24)
Dwaniro (N = 10)	6	50.0	1.30 (0.24, 1.60)	50.0	1.71 (0.31, 9.42)	0.0	NA
Kabonera (N = 33)	23	30.4	0.57 (0.20, 1.65)	47.8	1.56 (0.57, 4.31)	21.7	0.61 (0.25, 1.54)
Kabuwoko (N = 39)	30	46.7	1.14 (0.45, 2.87)	36.7	0.99 (0.38, 2.56)	16.7	1.09 (0.47, 2.55)
Kindulwe (N = 35)	23	39.1	0.84 (0.30, 2.32)	47.8	1.56 (0.57, 4.31)	13.0	0.94 (0.37, 2.36)
Segero (N = 61)	46	43.5	1.00	37.0	1.00	19.6	1.00

Characteristic	N	% Mono-infection	OR (95% CI)[2]	% Double Infection	OR (95% CI)	% Triple Infection	OR (95% CI)
Sex							
Female (N = 142)	105	37.1	1.00	48.6	1.00	14.3	1.00
Male (N = 127)	85	43.5	1.30 (0.73, 2.34)	34.1	0.55 (0.30, 1.00)	22.4	1.01 (0.59, 1.73)
Religion							
Muslim (N = 14)	8	37.5	0.89 (0.20, 3.91)	50.0	1.26 (0.30, 5.27)	12.5	0.99 (0.26, 3.77)
Christian (N = 84)	60	40.0	0.99 (0.53, 1.87)	36.7	0.73 (0.39, 1.38)	23.3	0.84 (0.47, 1.50)
Catholic (N = 171)	122	40.2	1.00	44.3	1.00	15.6	1.00
Schooling							
Not Enrolled (N = 45)	35	31.4	1.00	45.7	1.00	22.9	
Nursery (N = 39)	32	40.6	1.49 (0.55, 4.07)	37.5	0.71 (0.27, 1.89)	21.9	1.32 (0.54, 3.24)
Primary (N = 171)	116	42.2	1.60 (0.72, 3.56)	44.0	0.93 (0.44, 1.99)	13.8	1.64 (0.81, 3.34)
Secondary (N = 14)	7	42.9	1.64 (0.31, 8.59)	14.3	0.20 (0.02, 1.82)	42.9	0.81 (0.18, 3.66)
Shoe Usage							
Never (N = 142)	90	41.1	1.00	44.4	1.00	38.2	1.00
Every Week (N = 13)	9	44.4	0.90 (0.49, 1.62)	0.0	NA	55.6	0.34 (0.10, 1.25)
Every Day (N = 114)	91	38.5	1.15 (0.29, 4.56)	44.0	1.16 (0.65, 2.06)	17.6	0.87 (0.50, 1.50)
Deworming History							
Past 12 months (N = 89)	67	46.3	1.54 (0.84, 2.83)	34.3	0.60 (0.32, 1.11)	19.4	1.14 (0.53, 2.44)
More than 12 months (N = 177)	120	35.8	1.00	46.7	1.00	17.5	1.00
Bednet Use							
Uses net (N = 69)	49	51.0	1.84 (0.95, 3.55)	36.7	0.74 (0.38, 1.45)	12.2	0.56 (0.22, 1.46)
Does not use net (N = 200)	141	36.2	1.00	44.0	1.00	19.9	1.00
Pig Ownership							
Owns Pigs (N = 212)	155	39.6	0.87 (0.41, 1.82)	41.3	0.84 (0.40, 1.75)	19.4	1.86 (0.61, 5.67)
Does not own pigs (N = 57)	35	42.9	1.00	45.7	1.00	11.4	1.00
Head of Household Education							
None (N = 41)	28	46.4	1.00	42.9	1.00	10.7	1.00
Some Primary School (N = 135)	97	37.1	0.68 (0.29, 1.59)	37.1	0.90 (0.38, 2.10)	22.7	2.44 (0.67, 8.87)
Some Secondary School (N = 68)	46	34.8	0.62 (0.24, 1.61)	34.8	1.33 (0.52, 3.43)	15.2	1.50 (0.35, 6.33)
Head of Household Occupation							
Farmer (N = 231)	161	41.6	1.58 (0.68, 3.69)	42.2	1.04 (0.46, 2.31)	16.2	0.51 (0.20, 1.26)
Other (N = 38)	29	31.0	1.00	41.4	1.00	27.6	1.00
Malaria History							
Past 12 months (N = 73)	59	37.3	0.85 (0.45, 1.60)	39.0	0.83 (0.44, 1.55)	23.7	1.73 (0.80, 3.71)
More than 12 months (N = 196)	131	41.2	1.00	43.5	1.00	15.3	1.00

Characteristic	N	% Mono-infection	OR (95% CI)[2]	% Double Infection	OR (95% CI)	% Triple Infection	OR (95% CI)
Infection Intensity							
Light	175	41.7	1.00	42.3	1.00	16.0	1.00
Moderate/Heavy	15	20.0	0.35 (0.10, 1.28)	40.0	0.91 (0.31, 2.67)	40.0	3.50 (1.15, 10.61)
Treatment Efficacy							
Cured	99	40.4	1.81 (0.88, 3.70)	43.4	0.80 (0.41, 1.54)	16.2	0.62 (0.27, 1.42)
Not Cured	55	27.3	1.00	49.1	1.00	23.6	1.00
Average Dietary Diversity Score	190	NA	1.16 (0.88, 1.53)	NA	0.96 (0.73, 1.27)	NA	0.82 (0.56, 1.21)
Average Socioeconomic Index	190	NA	1.09 (0.88, 1.34)	NA	0.92 (0.75, 1.13)	NA	1.00 (0.76, 1.31)
Average Weight/Height	190	NA	0.42 (0.00, 52.23)	NA	0.21 (0.00, 25.70)	NA	41.55 (0.13, >99.99)
Average Body Mass Index	190	NA	0.97 (0.90, 1.04)	NA	0.99 (0.93, 1.06)	NA	1.05 (0.98, 1.13)

[1]Numbers may not sum to 190 (total infected) due to missing data.
[2] Unadjusted odds ratios were calculated using logistic regression.

Infection polyparasitism, or the number of distinct STN infections, was significantly associated with age, shoe usage, and weight for height. Curiously, children between the ages of 6 and 10 were 26% more likely to have a mono-infection and 21% less likely to have a double infection as children between 4 and 5 ($p = 0.02$). Children who wore shoes every day were approximately equally likely to have a mono-infection as a double infection. The proportion of infections that were of light intensity decreased as polyparasitism increased; 96.1% of mono-infections were light, while only 82.4% of triple infections were light ($p = 0.05$). Dietary diversity, socioeconomic status, and BMI were not associated with polyparasitism. In contrast, weight for height was strongly associated with this parameter, but inconsistently so, such that children with no STN infection and children with triple infections had elevated weight for height ($p = 0.02$).

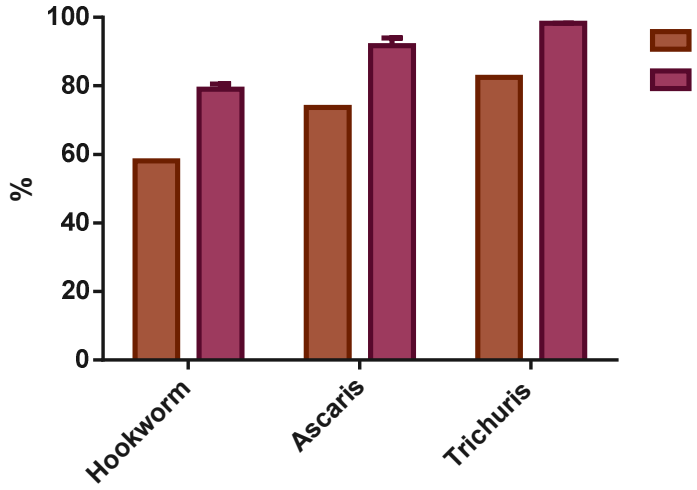
Table 9c: Logistic Regression Models to Predict Triple Co-Infection

Characteristic	Adjusted OR	p
Model 1		
Age	1.00 (0.92, 1.08)	0.9221
Sex	0.99 (0.58, 1.69)	0.9633
Dietary Diversity	0.85 (0.65, 1.10)	0.2044
Socioeconomic Status	0.96 (0.79, 1.16)	0.6383
Model 2		
Intensity	3.76 (1.27, 11.17)	**0.0171**
Bednet Use	0.49 (0.26, 0.94)	**0.0322**
Model 3		
Bednet Use	0.41 (0.21, 0.81)	**0.0102**
HH Occupation	0.49 (0.22, 1.06)	0.0709
Dietary Diversity	0.87 (0.67, 1.14)	0.3219
BMI	1.06 (0.99, 1.13)	0.1111
Intensity	3.55 (1.18, 10.64)	**0.0239**

A logistic regression modeling the effect of age, sex, dietary diversity, and socioeconomic status on polyparasitism found no significant predictors. When constructed using backward selection of all available potentially predictive parameters, infection intensity and bednet use were the only significant predictors ($p = 0.02$ and $p = 0.03$, respectively). The five most important predictors also included whether the head of the household was a farmer, dietary diversity, and BMI. Children who slept under bednets and children with lighter infections were much less likely to suffer from triple co-infections, but both effects were slightly attenuated after controlling for head of household occupation, dietary diversity, and BMI. Having a farmer head of household protected against triple co-infection, as did increased dietary diversity. The effect of BMI was slight and insignificant.

The cure rate and egg reduction rates were calculated for albendazole with respect to each type of helminth. For hookworm, the cure rate was 58.1% and the fecal egg reduction rate (FECR) was 79.0% (95% CI = 77.4% - 80.5%). For *A. lumbricoides*,

the cure rate was 73.7% and the FECR was 91.8% (95% CI = 89.2% - 94.0%). The cure rate for *T. trichiura* was 82.5%, and the FECR was 98.3% (95% CI = 96.4% - 98.3%).



DISCUSSION

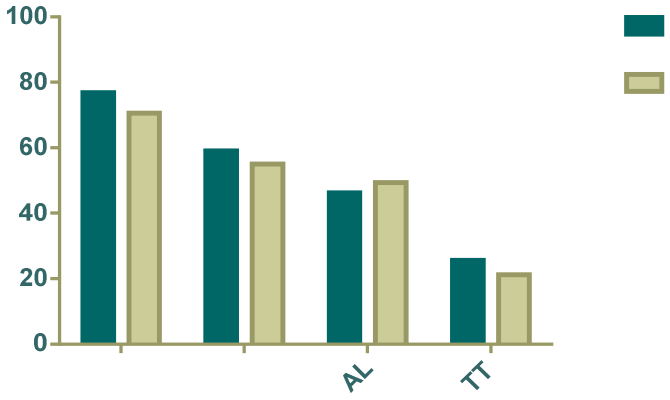
The statistical analyses conducted in this study yielded a number of noteworthy findings. The results can most easily be understood when considered as risk factors for infection, stratified by external factors (relating to the community and household environment) and individual factors (relating to the physiology and behavior of the child), and separately as risk factors for certain infection outcomes.

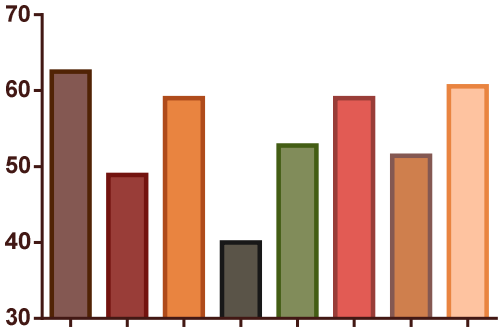
Implications of Community, Environmental, & Household Risk Factors

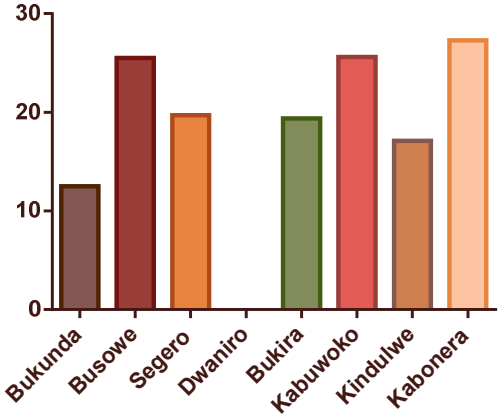
The data from this study suggest that environmental and structural factors may play influential roles in STN infection occurrence.

Though not statistically significantly so, infection rates did vary somewhat by village, suggesting that where a child lives may underlay a portion of the infection risk

they face. Kabuwoko Village, for example, had high infection prevalence when considered by most categories (overall STN infection, hookworm infection, and *T. trichiura* infection), but was below average in terms of prevalence of *A. lumbricoides*. Interestingly, the prevalence in Bukira Village, which directly neighbors Kabuwoko, was consistently lower.







Kabuwoko Village is more densely populated than many of the other villages, and, as the primary village of the parish, serves as a social hub, while Bukira Village

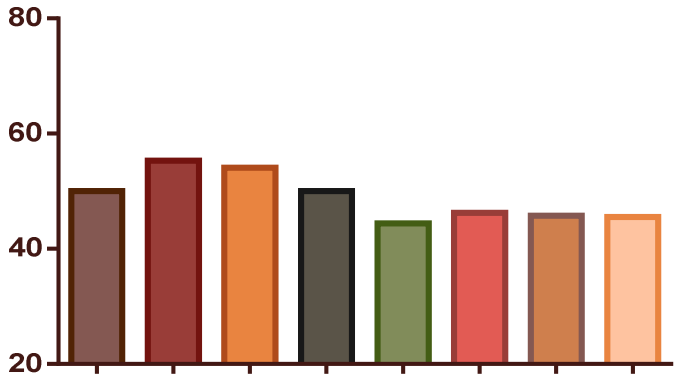
consists of more dispersed homes that house small families. There are several potential pathways by which these factors may help explain increased STN infection prevalence in Kabuwoko. Pit latrines, houses, and gardens all exist in closer proximity when population density is higher; this may increase transmission by encouraging more frequent contact between residents and high-risk transmission areas, like the ground surrounding pit latrines. Reiss et al and Halpenny et al discussed the association between hookworm infection prevalence and population density, positing that prevalence increases with population density, but decreases with urbanization, in support of earlier findings (Reiss et al. 2013, Halpenny et al. 2013). Additionally, as many of the primary schools and regional meeting spaces are located in Kabuwoko, the pit latrines in this area receive more traffic than usual. This may allow for infectious material to be brought in from a range of other villages, and may explain the heightened transmission observed in this population.

	Cycle 1			Cycle 2				
		Ascaris			*Ascaris*		Hookworm	
	n	Infected	Uninfected	n	Infected	Uninfected	Infected	Uninfected
Regional Factors								
Residence in high prevalence cluster, %		-	-	144			41(25–58)	20(14–29)*
Household Factors								
Household Density, km^2	131	45±9	31±3	144	31±9	35±3	39±7	33±3
Wealth Index, *HWI*[2]	128	0.17±0.13	0.20±0.07	140	−0.06±0.16	0.16±0.06	0.06±0.1	0.16±0.06
Mother's Education, *yrs*	131	3.1±0.7	3.5±0.3	143	2.1±0.8	3.7±0.33	3.5±0.7	3.5±0.3
# People/Room	123	5.3±0.6	5.2±0.3	143	5.5±0.6	5.5±0.2	5.8±0.5	5.5±0.3
Individual Factors								
Cycle 1 Baseline Infection, *epg*	122	10463±3119	1737±478**	172	2895±1549	612±193*	91±39	4±2**
Age, *mo*	155	32±2	31±1	200	22±3	27±1	29±2	25±1
Female, %	155	48(33–65)	52(44–61)	200	45(27–65)	52(45–59)	47(33–61)	53(45–61)
Height for age								
Z score	130	−2.8±0.2	−2.6±0.1	170	−2.6±0.2	−2.4±0.1	−2.6±0.2	−2.4±0.08
Stunting, %	130	84(67–93)	70(60–78)	170	78(54–92)	70(63–77)	77(61–88)	70(61–77)
Latrine use, %	135	9(2–24)	18(11–26)	185	10(2–31)	15(10–21)	12(5–26)	15(10–22)

[1]Summary statistics presented are mean ±SEM or % (95% CL).
[2]Asset-based index, weights derived from the first component of Principle Components Analysis

Risk factors for STN infection in Panama reveal that denser populations in rural communities have higher infection prevalence. (Halpenny et al. 2013)

Dwaniro had the lowest prevalence of both hookworm and *T. trichiura*; this may be due to the fact that few families in Dwaniro own land, and to the fact that the village exists more as a linear stretch of homes along a road than as a cluster of houses more typical of a village structure. The sprawling, non-agricultural nature of this village may be less conducive to transmission than one in which homes are tightly clustered and intermingle with pit latrines, agricultural land, and livestock grazing areas. This conjecture is supported by a number of studies in which livestock ownership, high population density, and less hygienic pit latrine use have been associated with higher STN infection prevalence (Freeman et al. 2013, Humphries et al. 2011, Reiss et al. 2013). The prevalence of *A. lumbricoides* was fairly consistent across villages, which may be a reflection of the prolonged environmental stability of *A. lumbricoides* eggs (which may persist for up to 15 years), or of uniformity in the risk factors specific for this infection (though this is less likely, as many of the risk factors are shared by other soil-transmitted nematodes).



In contrast, the prevalence of both hookworm and *T. trichiura* varied more substantially by village, suggesting that these pathogens may be slightly more sensitive to environmental factors, or that the environmental factors that affect egg viability and transmission are less well met in some villages. Though the implications remain unclear, there is some evidence, even within this small sample, that transmission varies by village and may be influenced by related environmental or behavioral factors.

Household factors also appear to play a role in transmission. Muslim families consistently exhibited lower infection prevalence than both Catholic and Christian families. This finding was not significant, but it is probable that the lack of significance is due to the low representation of Muslim families in this sample. The difference may be explained by different hygiene and behavioral practices. Muslim families do not own pigs, and pig ownership was associated with increased infection prevalence in all cases. Yet the evidence that this is the cause for the religion-based disparity in infection risk is not definitive; other behavioral differences may be responsible as well. This hypothesis is supported by the fact that religion was non-significant in multivariate analysis, while pig ownership was consistently significant. Furthermore, previous studies have not found an association between religion and STN infection.

The socioeconomic status indicator measure used in this study did not appear to have any bearing on any of the infection outcomes. This finding was contrary to several other studies which have found a negative association between STN infection and wealth (De Silva et al. 1996, Al-Mekhlafi et al. 2007). However, Halpenny et al likewise found no significant association between a constructed wealth index and STN infection (Halpenny et al. 2013). There is both theoretical and evidence-based support for this

association, as wealthier families typically have improved access to health care, education, and hygiene practices. While it is possible that socioeconomic status has little bearing on infection risk or outcome in this population, or that there is not enough variability in socioeconomic status among the members of the study population to draw any relevant conclusions, it is also possible that the indicator used did not sufficiently capture differences in wealth across the households reviewed. Wealth is notoriously difficult to measure, and the standard indicators used in this type of research may not have been quite so relevant in this community. For example, the primary water source for a home is more a product of individual preference than wealth, as all people have access to all water sources in the area (and all of the sources are fairly far from residential spaces). Housing material is also often only vaguely related to wealth, as most families have lived in the same home for many generations; the wealth status of the family at the time the house was constructed may be quite different from the family's current wealth status. Similarly, owning land does not exactly correlate with wealth. Of course, families that do not own agricultural land have fewer resources than those who do, but there is also great variability in wealth among those who do own agricultural land. The amount of land owned is not uniform across families, and those with smaller plots are more financially strained. This variability was not captured in the socioeconomic status indicator; in effect this measure was redundant with the food insecurity measure, as the only variability captured here was the effect sought in the food insecurity measure (whether the family had enough food for itself). Many of the factors assessed in the socioeconomic status indicator may not have sufficiently reflected differences in wealth.

Health care, however, is extremely related to financial resource capacity in this community: families who can afford them will use private health facilities over public ones, as public facilities often lack basic and essential services, in addition to being heavily plagued by employee absenteeism. Livestock ownership is also indicative of wealth; livestock are both a status symbol and a source of income and sustenance in this region. Because these two indicators are influenced less by non-financial factors and capture the gradient of wealth fairly well, these were useful metrics for building a socioeconomic status index in this population.

Relatedly, the FAO dietary diversity index may not have quite captured what it intended to assess in this study population. Dietary diversity in this community was, on average, fairly low, such that the breadth of the dietary diversity index failed to capture much of the gradient in this population. The average dietary diversity score within the population was 3.4, out of a possible 16. Most of the points were awarded to groups of foods; the variability of diet within this population occurred more within these groups then across them. Thus, this index obscured some of the nuance of diet in this population, reflecting only gross differences, of which there were few.

The analysis from this study highlights the need for new standards in this type of research that accommodate nuances observed in different geographical and cultural settings. Socioeconomic indicators that are relevant in one context may not serve as well in another, and demanding uniformity in the metrics used in this field of research may obscure important associations as a result of poor specificity of the measure. It is important to standardize protocols so that research may be compared, but this must be

balanced with the understanding that wealth and dietary diversity mean different things in different places.

Fortunately, other household-level metrics, such as pig ownership, did appear to be useful for analysis of infection risk. Though only significant in unadjusted association in the case of *T. trichiura* infection, pig ownership was consistently associated with an increased risk of STN infection and was featured in nearly every logistic regression model. The relationship between pig ownership and STN transmission is not well understood, though there is speculation on potential causative pathways, as discussed earlier (Traub et al. 2004). Why this effect was greatest for *T. trichiura* is also unclear, and has not been observed in other studies. The significance of pig ownership appears to have been overstated in the unadjusted associations, as the strength and significance of the association decreased in the logistic regression models. This highlights the fact that pig ownership is tied to other predictive factors measured in this study, such as socioeconomic status, head of the household occupation, and religion.

Community parents receiving new piglets as part of an income generating project managed by Hope for African Children.

Factors related to the head of the household had varying significance, and reservations in interpreting these results are warranted. In many families, the head of the household is not particularly influential in the life of the child, and their wealth does not always trickle down to all members of the family. Thus, while it might be reasonable to presume that having a more educated head of the household would lead to better household hygiene practices and greater financial stability in the family, in experience they do not appear to be very tightly linked in this community. Consequently, it is not particularly surprising that the education level of the head of the household did not significantly correlate with any of the STN infections or with an STN infection in general, despite exhibiting a correlation in other studies (Sanchez et al. 2013, Mekhlafi et al. 2007, Conlan et al. 2012).

Previous studies have suggested that farming increases STN transmission, which may increase transmission for the family due to household clustering of infections (Humphries et al. 2011, Halpenny et al. 2013). This was not observed in this study. In fact, when present, the association went in the other direction, such that farming households were less likely to harbor an infection than non-farming households. This may be explained by the fact that the majority of families in this community farm, regardless of whether farming is the primary occupation of the head of the household. Most parents and most children spend at least some time each day working the earth; the exposure to potentially contaminated soil, or the exposure to soil that may be contaminated by an infected individual, is not well captured by this parameter. Perhaps, when the head of the household is primarily a farmer, the children are not required to take on as many of the farming responsibilities, and therefore are subject to reduced

exposure when compared to children in families where the head of the household is less involved with farm work. Most households rely on farming for sustenance; there is a certain amount of farming to be done by all households. Thus, if the head of the household is not doing it, someone else must take on the responsibility; if this is a child, then their risk of acquiring STN infection may increase as a result. This correlation is merely speculation at this point; further research assessing how much time individual family members spend farming, as well as their behaviors during that time, would be required before any conclusions could be drawn.

Table 2
Risk factors for hookworm infection

	Adjusted odds ratio*	*P* value	95% CI
Children			
Malaria	2.84	0.03	1.11, 7.26
ES IgG 2nd quartile	5.43	0.007	1.59, 18.54
ES IgG 3rd quartile	4.97	0.02	1.26, 19.68
ES IgG 4th quartile	6.24	0.005	1.72, 22.65
Adults			
Body mass index	0.71	< 0.001	0.60, 0.85
Does not wear shoes	4.06	0.004	1.57, 10.53
Does not use a latrine	6.10	0.001	2.09, 17.54
Farmer	4.89	0.001	1.90, 12.61

*Adjusted for age, gender, and community.

Multiple logistic regression reveals that occupational exposures of farming may increase the risk of hookworm infection in Kintampo, Ghana. (Humphries et al. 2013)

As characteristics of the environment and household are somewhat removed from the child, associations between these parameters and infection outcomes must be taken with some hesitation. These associations are incredibly useful for deriving hypotheses about potential interaction pathways, and help guide future research, but do not provide particularly reliable results in and of themselves. It is both possible and likely

that the characteristics of the resident village, socioeconomic status, pig ownership, and education level and occupation of the head of the household bear some relevance to STN infection outcomes, and this research helps suggest how each factor may be important. Future studies may better elucidate the pathways through which these structural factors influence transmission. Highlighting these pathways will be useful in designing ecological-level interventions to reduce STN transmission, which may lead to sustainable and cost-effective reductions in infection.

Implications of Risk Factors Related to the Child

Beyond structural factors, whose role in STN transmission is indirect, are behavioral and physiological factors of the child, which may bear more direct relevance to transmission pathways.

Age was a significant predictor of STN infection in general, as well as of hookworm and roundworm infection. The effect was similar for all helminths reviewed, such that the youngest children were at the greatest risk of infection, and risk diminished with age. This finding is contrary to what Sanchez et al. found when analyzing STN infections among children in rural communities in Honduras (Sanchez et al. 2013). Though few studies have analyzed prevalence variation by age among children, a study conducted in Thailand reviewing defecation patterns and other risk factors for STN infection among rural populations found that open defecation was more common among younger children (Chongsuvivatwong et al. 1996). This finding may help explain the patterns observed in this study population. It is presumable that younger children are more likely to play in the dirt and are less likely to be vigilant about

cleaning themselves afterwards. Anecdotally, in this community they are also more likely to defecate in the open and to use a sliding technique rather than leaves to wipe themselves afterwards. As children mature, these behaviors become likely less common, which may reduce transmission risk.

Additionally, it is possible that children become somewhat immunotolerant to STN infection with age and repeated exposure. This may occur through two possible pathways, as discussed by Allen and Maizels in a recent review on immunity to helminths (Allen and Maizels 2011). It is well known that the immune system matures with age, reaching a peak in late adulthood (Abbas and Lichtman 2009). It is also well known that immune responses tend to be more rapid and robust in repeat exposures to previously encountered pathogens (Abbas and Lichtman 2009). It is possible that the age-based variation is due to factors relating to the immune system, but this cannot be confirmed given current limited understanding of how these factors interact.

Furthermore, it is possible that the association observed is due to co-linearity with more direct predictors of STN infection risk. This is suggested by the fact that age did not appear to be a significant predictor in any of the adjusted association logistic regression models. A potential co-linear relationship may exist with the weight for height parameters, as the relationship between weight and height is known to vary with age (World Health Organization). At least one body size index was a strong predictor in all multivariate analyses; this may be the cause for the observed age association. Regardless of the reason, the association between age and infection status may be useful in guiding future interventions; the data from this study suggests that greater

impact may be derived from interventions that disproportionately target younger children.

Though the correlation between sex and infection status was not statistically significant in any case, the association was consistently skewed such that females were more likely to harbor infection than males. In addition, sex was found to be one of the five most influential parameters in determining the outcome of STN infection of any type. The lack of significance in all observed associations may be due to the fact that the difference between the sexes is slight or inconsistent, such that the sample size of this study was insufficient to capture the difference. It is possible that this difference is due to as of yet unidentified pathways between host immune response to soil-transmitted nematodes and changing hormone profiles during puberty. This association has not been well examined, and a significant sex-based variation in prevalence has not been reported among studied populations of children. A more likely explanation is that hygiene and behavior differences between the sexes account for some of the disparity. In this community, female children are more likely than their male counterparts to be relieved of the opportunity to go to school in lieu of being recruited for additional farm work. Females are also likely to have less adequate hygiene behaviors, as available soap and shoes are preferentially reserved for males. Females are frequently prohibited from playing soccer, and instead play netball (similar to volleyball). As soccer involves footwork, the boys will sometimes wear shoes for the sport, while girls almost always remain barefoot for netball. These and other differences in the behavior exhibited by males and females may contribute to divergent exposure risk, and may thus account for the consistent, though minor, sex-based difference in STN infection. Furthermore, the

fact that these parameters are somewhat removed from the sex-based association may help explain why this parameter failed to reach significance in this study, even if it is associated with divergent risk.

Left: boys playing soccer in uniform (with shoes). Right: girls playing netball (barefoot).

The relationship between school enrollment and infection status was likewise statistically insignificant, but there is still room for conceptual speculation. Firstly, it is important to note that this parameter is especially likely to interact with other assessed parameters, such as age, sex, and shoe usage. Younger children are less likely to be enrolled in school (it is rare for a child under 6 years of age to be enrolled). As previously discussed, distinct differentiated sex roles are common in this community; females are less likely to be enrolled in school than their male siblings. Lastly, as shoes are a required component of the school uniform and a scarce resource otherwise, children enrolled in school are more likely to have shoes than those not enrolled. It is possible that the strong interactions with these parameters, and the fact that the direction of association varied among the parameters, may have resulted in a non-significant association with schooling despite its being a parameter relevant for consideration.

In the case of any STN infection and hookworm infection specifically, risk appeared to be greatest among nursery school children, followed by those not enrolled, primary school attendees, and lastly, secondary school children. A similar case was noted for *A. lumbricoides* infection risk, with the exception that those not enrolled in school faced the greatest infection risk. The disparity here is likely due to household factors that preferentially increase *A. lumbricoides* transmission: transmission of this helminth appeared to be more significantly associated with head of the household occupation. As discussed earlier, children not enrolled in school are more likely to engage in farm work, and the increased significance of this factor may explain, at least in part, the shift in risk by school status observed. Beyond this minor disparity, the patterns discussed seem to be reflective of age and associated behavior-related patterns: hygiene likely improves as children age and progress through school. Those not enrolled in school are of all ages but are predominately younger, explaining why they fit in between nursery and primary school kids in terms of infection risk. Curiously, the risk profile for *T. trichiura*, when stratified by school, did not match the others. For this helminth, infection risk was greatest among those in secondary school, followed by those not enrolled, those in nursery, and those in primary. This may just be an artifact, as relatively few children in the study population were enrolled in secondary school to begin with, but it is worth discussing because it does diverge from the trends observed for other STN infections (which also suffered from having relatively low representation from secondary school enrollees). This divergence remains in line with existing knowledge of STN risk profiles, which suggest that risk is greatest among children between the ages of 5 and 15, but which fail to delineate risk differences within those

childhood years (Bethony et al. 2006). Thus, it is possible that this difference is typical and has simply been poorly examined thus far. Other explanations include the possibility that older children are exposed more regularly to environments that are specifically conducive to *T. trichiura* embryonation, or that these individuals are less likely to receive anthelminthic treatment (which, due to its high effectiveness in this population, may actually have artificially lowered the prevalence in other, more frequently treated groups). As the relationship between these parameters has not yet been well characterized, there remains much room for speculation on the roots of this association.

Inextricably related to school enrollment is the usage of shoes, which displayed a highly idiosyncratic relationship to all types of STN infection in this study population. It is widely believed, though not well supported by scientific evidence, that shoe usage decreases STN transmission by preventing larval penetration of the feet (Freeman et al. 2013). Several studies have reported an inverse association between shoe usage/ownership and STN infection (Humphries et al. 2013, Tadesse 2005). However, the metrics used to assess shoe wearing behavior vary across studies; this variation appears to affect the results substantially.

Table 3: Hookworm prevalence among Babile town schoolchildren, Eastern Ethiopia, 2001

	Hookworm infection				
	Yes No (%)	No * No (%)	Total No (%)	χ^2	P-value
Shoe					
Present	26 (6.5)	371 (93.5)	397 (100)		
Absent	2 (11.1)	16 (88.9)	18 (100)	0.57	0.34
Protective shoe					
Present	18 (6.3)	267 (93.7)	285 (100)		
Absent	8 (7.1)	104 (92.9)	112 (100)	0.09	0.76
Shoe wearing habit					
Always	8 (3.9)	199 (96.1)	207 (100)		
Sometimes/ not at all	20 (9.6)	188 (90.4)	208 (100)	5.45	0.01

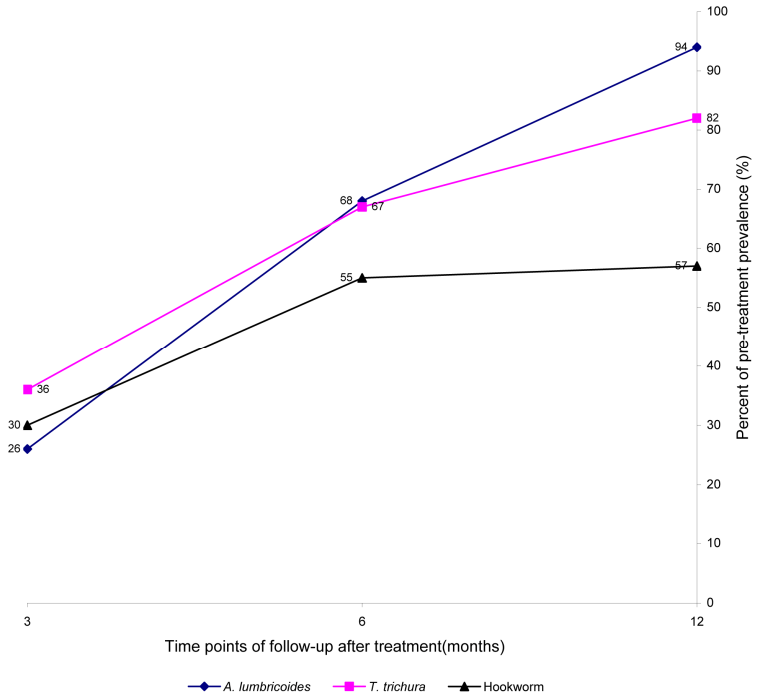
* No ova, larvae or adult of hookworm seen

Evidence of the protective effect of quality shoes against hookworm infection in Ethiopia. (Tadesse 2005)

The results of this study found shoe usage to be a potentially irrelevant factor in transmission, or one that was poorly assessed by the questionnaire, as children who wore shoes daily were much more likely to be infected than both children who wore shoes only weekly and children who did not wear shoes at all. This observation serves to highlight the futility of such a measure as a proxy for shoe-wearing behavior: though better than a metric that simply assesses whether a child owns shoes rather than whether they wear them, this metric remains flawed. Children in this community value shoes very highly; wearing them is a privilege. As a required and costly part of the school uniform, shoes are well-maintained and protected from damage. In many cases, this means the shoes are only worn at school and during religious worship: the shoes are carried on the walk to school, and are often removed for playing outside. Though children may be wearing their shoes at some point every day, they may not truly be reducing exposure to STN infection.

Yet the association does appear to be significant in several cases, suggesting a more substantive force may be at work, rather than simple misclassification. It is possible that indoor chores are delegated to the children who do not have shoes, and that the children who do have them do not wear them consistently while working outdoors. Many possible explanations can be speculated, but none can be confirmed as of yet; additional behavioral data would be required to elucidate more directly plausible pathways. It is interesting also to note that, though significant in unadjusted associations, shoe usage was not implicated in any of the logistic regression models. This suggests overlap with other assessed parameters, further highlighting the potential ineffectuality of this metric in understanding and explaining STN transmission.

The relationship between deworming history and STN infection risk was also an interesting one. This parameter was not significant in any unadjusted associations, and confidence intervals were consistently wide, so there is no apparent association between these two parameters. A meta-analysis of studies analyzing reinfection rates found that, three months after treatment, infection prevalence of *Ascaris*, *Trichuris*, and hookworm reached 26%, 36%, and 30% of pre-treatment levels respectively (Jia et al. 2012). These high reinfection rates suggest that little correlation would be observed between infection status and recent deworming history beyond this three month window. School-based deworming had taken place 4 months prior to the study in this community, so it is unsurprising that little correlation was seen between recent deworming history and infection; this finding is in line with studies that have observed similarly high rates of reinfection.



Estimate of reinfection rates for soil-transmitted nematodes based on a review of the literature. (Jia et al. 2012)

Despite being non-significant in univariate assessment, the parameter was among the five most influential for both STN infection in general and for hookworm infection. In both cases, the association was statistically significant and suggested that children who had been dewormed in the past year were more likely to harbor infection than those who had not been dewormed. Pullan et al similarly found that previous anthelmitic treatment was a risk factor for current STN infection (Pullan et al. 2011). This is a very interesting finding, as it completely goes against expectation: anthelmintic treatment is supposed to decrease infection prevalence, not increase it. It is possible that this association is a relic of emerging treatment failure of hookworm infections in this community: the more a child is treated, the less effective the treatment becomes or the more likely to child is to pick up a new infection that is resistant to treatment. Following this logic, it is reasonable that this pattern would only be observed in hookworm infection cases, as the cure rates for both *A. lumbricoides* and *T. trichiura* were exceptionally high. Treatment failure has been observed previously in Ghana and Southeast Asia (Humphries et al. 2011, Soukhathammavong et al. 2012). This association is conceptually intriguing, but it is important to keep in mind that future investigation would be required to confirm or reject this idea.

Somewhat surprisingly, bednet usage and incidence of malaria did not appear to correlate with one another; one was associated with infection status while the other was not. This suggests one of two things: either the bednets being used in this community are not effective in preventing malaria (likely due to holes in the nets, improper use, or evasive behaviors by the mosquitoes), or they are not being used as claimed. The questionnaire was designed to rule out the second option; several questions were

asked to indirectly assess the actual use of the bednet, but this remains a possibility. Either way, the lack of association here is of concern, as malaria prevention is critical in this endemic region.

Beyond the public health importance of disease control, the importance of malaria prevention is further evidenced in this study by the increased infection prevalence seen for all STN infections in children who had suffered from malaria within the past year. Malaria history was significant in both unadjusted and adjusted associations, with the exception of *T. trichiura* infection (in which case bednet use and malaria history curiously switched places). Literature on the subject suggests that it is actually STN infection that increases susceptibility to malaria, rather than the other way around (Humphries et al. 2011). Thus, these data are in line with existing understanding of STN immunomodulation, and are great cause for concern as they serve to highlight a major aspect of the public health importance of this study: STN infections are abundant, and may be contributing to increases in malaria transmission, notably underwriting the burden of infectious disease among children in this region.

Another major public health implication of STN infection in this community is evidenced by associations seen between infection status, weight for height, and BMI. In all cases, except for *T. trichiura* infection, both low weight for height and low BMI appeared to be associated with infection. However, these parameters should not be interpreted in isolation, as both weight for height and BMI are somewhat age-dependent. Both characteristics were found to be predictors in all of the adjusted associations produced by logistic regression, even after controlling for age-associated variation (except for *T. trichiura*, for which only BMI was found to be an important

predictor). Interestingly, in all cases, low weight for height, but high BMI, appeared to be associated with a higher prevalence of infection. The weight for height finding is both intuitive and supported by the literature: chronic helminth infections are known to be associated with, and believed to be partly causative of, growth stunting. Parasitic helminths extract nutrients from their human hosts, and can cause anemia and protein deficiency, both of which impede growth rate (Hotez et al. 2004).

Table 5. Comparisons of nutritional status and helminth infections.

Helminth infections	Nutritional status		Row total	Row %	95% CI	*P* value	Percentage attribution risk	Population attribution risk
	HAZ							
	Stunted	Normal						
Infected	20	49	69	29	0.8–2.8	0.2	26.6	5.8
Uninfected	77	286	363	21				
	BAZ							
	Underweight	Normal						
Infected	6	63	69	8.7	0.7–5.5	0.2	46	79
Uninfected	17	346	363	5				
	MUAC							
	MAM	Normal						
Infected	14	55	69	20.3	0.6–2.3	0.7	10.3	19.1
Uninfected	66	297	363	18.2				

A greater proportion of stunted children were found to harbor STN infection in Wakiso District, Uganda. (Lwanga et al. 2012)

The opposing association observed with BMI is simply an artifact of the modeling procedure: both parameters influence the outcome in the same way, and are strongly correlated with one another (as they are both derivatives of weight and height data), but the association with weight for height and the outcome is stronger than the association for BMI and the outcome (weight for height is significant even in unadjusted associations, while BMI is not). Thus, to correct for the lower strength of the association, BMI acts to pull the estimate towards the null. Either way, the effect is the same: infected children tend to have lower weight for height (and therefore lower BMI), and the

difference is both statistically significant and relevant. Growth stunting can have major implications for development later on in life, and can hinder both cognitive ability and physical productivity, both of which can precipitate and maintain poverty (Stephenson et al. 2000, Pullan et al. 2010). By corroborating earlier findings that STN infection is associated with poor physical growth, this study highlights the importance of curbing STN infection as a means to increase productivity and reduce poverty.

The associations among risk factors related to the child provide key insights that may guide future interventions to cater to the most at-risk populations. This study suggests that younger children face an increased risk of STN infection, and highlights the detrimental effects such infections have on physical growth and susceptibility to malaria. The lack of association between deworming history and infection prevalence is concerning, as it suggests limited long-term effectiveness of anthelmintics in controlling infection prevalence in this community.

Implications of Risk Factors that Characterize the Infection

The relationships between polyparasitism, infection intensity, and treatment effectiveness reveal key insights into the biology of STN infections, in addition to highlighting areas of concern for disease control.

The high prevalence of polyparasitism relative to mono-parasitism is of note and in line with existing literature (Sanchez et al. 2013). Co-infections pose unique obstacles to disease control and speak to the need for integrated disease management; the fact that co-infections are more prevalent in this community emphasizes the need for the

development of strategic interventions that can simultaneously address infections by multiple STN species.

Monoparasitism	**1 species (n = 129)**	*T. trichiura*	113 (87.6)
		A. lumbricoides	9 (7.0)
		Hookworms	7 (5.4)
		Total monoparasitism	**129 (55.6)**
Polyparasitism	**2 species (n = 76)**	*T. trichiura* & *A. lumbricoides*	59 (77.6)
		T. trichiura & Hookworms	15 (19.7)
		A. lumbricoides & Hookworms	2 (2.6)
		Sub-total	**76 (73.8)**
	3 species (n = 27)	All three STH species	**27 (26.2)**
		Total polyparasitism	**103 (44.4)**

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Evidence of high prevalence of polyparasitism among school-age children. (Sanchez et al. 2013)

Very few determinants of polyparasitism were identified in this study. It is possible that this is due to the low sample size when study participants are stratified by multiplicity. Because of this limitation, associations that were not found to be significant but which exhibited consistent trends will still be discussed. No association was observed between school enrollment, sex, religion, and socioeconomic status and polyparasitism. However, age was a key determinant of polyparasitism; younger children were more likely to have more co-infections than older children. Sanchez et al. also found that polyparasitism was more common among younger children (Sanchez et al. 2013). This finding is likely explained by age-related exposure behaviors; younger children adhere less to hygienic behaviors than older children.

Children who owned pigs were overrepresented in the more multiplicitous infections; this is likely due to increased exposure as explained earlier. In contrast, the proportion of children from farming households decreased as the number of co-infections increased, suggesting that having a head of household who is a farmer may

be associated with a lower risk of harboring multiple helminth infections. This shift in risk to the child may again be due to guardians spending more time farming, rather than children, thereby decreasing the risk of environmental exposure to the children studied. These findings are in line with those found for risk factors faced by the child.

The findings related to having a recent history of malaria, dietary diversity, and weight for height may provide some insights into how the overall health of the child is implicated in the polyparasitism of STN infection. The more helminths harbored by a child, the greater the risk of having had malaria in the past 12 months. Mazigo et al. and Midzi et al. found similar results when studying co-infection with soil-transmitted nematodes, *S. mansoni*, and *Plasmodium falciparum* among schoolchildren in Tanzania and Zimbabwe, respectively (Mazigo et al. 2010, Midzi et al. 2008).

Table 4 Association of helminths with *Plasmodium falciparum* infection among primary schoolchildren living in a farming area in Zimbabwe

Parasite combination	Overall *n* (%)	Malaria positive *n* (%)	Malaria negative *n* (%)	X^2 (*P*-value)	Odds ratio (95% CI)
Schistosoma haematobium					
Examined	920	140	780		
Positive	546 (59.3)	85 (60.7)	461 (59.1)	0.13 (0.72)	1.07 (0.74–1.55)
Schistosoma mansoni					
Examined	906	132	774		
Positive	155 (17.1)	34 (25.8)	121 (15.6)	8.15 (0.004)	1.87 (1.21–2.90)
Hookworm					
Examined	905	131	774		
Positive	117 (12.9)	31 (23.7)	86 (11.1)	15.68 (<0.001)	2.48 (1.56–3.93)
Trichuris trichiura					
Examined	905	131	774		
Positive	11 (1.2)	3 (2.3)	8 (1.0)	1.47 (0.21[a])	2.24 (0.59–8.57)
Ascaris lumbricoides					
Examined	905	131	774		
Positive	8 (0.9)	1 (0.8)	7 (0.9)	0.03 (1.000[a])	0.84 (0.10–6.91)

[a] *P*-value based on Fisher's exact test.

Children with Schistosoma sp., *hookworm, and* Trichuris *infection all faced an increased risk of malaria co-infection. (Midzi et al. 2008)*

This may suggest that children become increasingly immunocompromised with each additional helminth infection, which intuitively makes sense: each helminth acts in

a unique way to suppress and evade the immune system, so, together, multiple helminths will provide broader immunosuppression. That this manifests as a correlation between increased incidence of malaria and increasing polyparasitism is not particularly surprising. This finding is both in line with existing literature and cause for concern, as it highlights the compounded negative health outcomes for children who reside in areas endemic for multiple soil-transmitted nematodes.

Relatedly, dietary diversity appears to be incrementally lower with each additional helminth, and was among the top five predictors of triple co-infection. The changes between poly-parasitism groups were subtle, but this is largely due to the fact that the dietary diversity scores did not exhibit wide variety to begin with: when the measure is, on average, between 3 and 4, any change will be small in value. Nonetheless, this relationship between poor dietary diversity has been discussed in the literature (Sanchez et al. 2013). Poor diet may weaken the immune system, leaving these children more susceptible to all three STN species (McSorley and Loukas 2010). It is also possible that the poor dietary diversity measure is a reflection of a larger picture of limited resource availability: perhaps the child is not well cared for at home and does not maintain proper hygiene; perhaps the limited capacity of the family farm has led to minimal productivity and therefore poverty. There are a number of plausible pathways by which low dietary diversity, either as a function of limited resource availability or neglect, may compromise immune function and thereby increase susceptibility to multiple soil-transmitted nematode infections. Future studies may attempt greater specificity in the dietary diversity metric to increase visibility of meaningful differences in diet within a population with limited overall dietary diversity.

The trend in weight for height poses a similar concern, though with opposing causality: this too appears to decrease with increasing polyparasitism (the blip seen in children with three helminth infections is likely due to the fact that *T. trichiura* infections are more common in older children generally, and they tend to have higher weight for height). Yet unlike dietary diversity, the trend in weight for height is most likely a reflection of both a cause and an outcome of STN polyparasitism: the lower a child's weight for height, the less robust their immune system, and the less able they are to fight off STN challenge (McSorley and Loukas 2010, Dumba et al. 2008, Sanchez et al. 2013). On the other hand, the more infections a child harbors, the more likely they are to be underweight, as they suffer from poor nutrient absorption and growth stunting (Loukas et al. 2005). The feedback loop at play here is of great concern, as physical growth during childhood has many implications for health during adulthood: children who suffer from stunted growth are less likely to reach their full physical and cognitive capacity, which may decrease opportunities available to them later in life while limiting overall productivity (Pullan et al. 2010). In this way, STN infections pose a chronic and significant threat to the ability of this community to rise out of poverty and successfully develop. Thus, controlling STN infections must be a key priority in the quest to eliminate endemic poverty in this area.

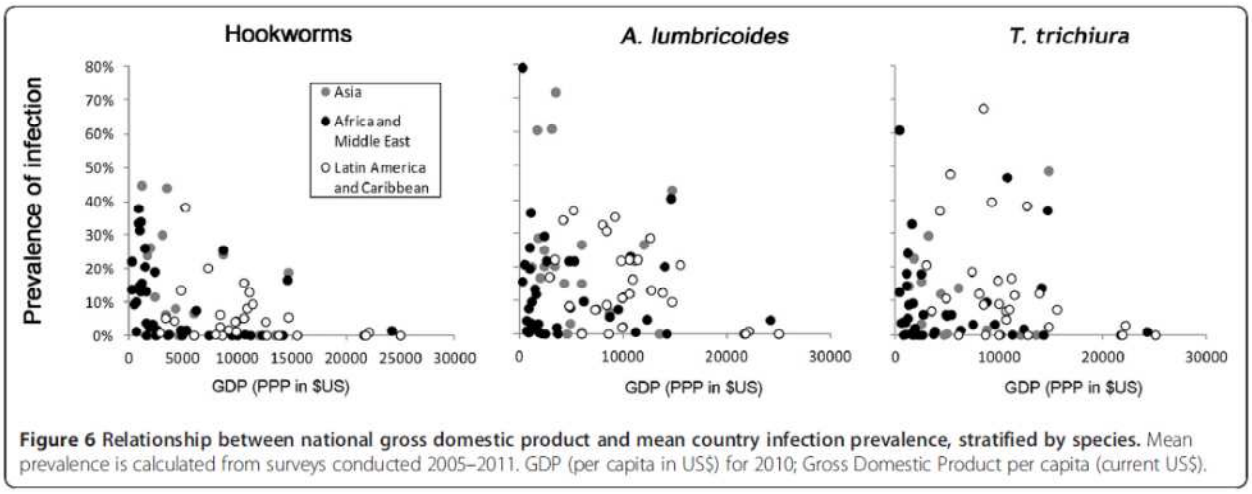


Figure 6 Relationship between national gross domestic product and mean country infection prevalence, stratified by species. Mean prevalence is calculated from surveys conducted 2005–2011. GDP (per capita in US$) for 2010; Gross Domestic Product per capita (current US$).

GDP tends to be lower in countries with high STN prevalence, highlighting the substantial economic cost posed by morbidities of these diseases. (Pullan and Brooker, 2012)

Another notable finding regarding infection polyparasitism is the fact that infection intensity increases with polyparasitism, while treatment effectiveness decreases. The correlation with infection intensity provides further evidence for the collaborative immunosuppressive effect enacted by co-infecting helminths: if helminths do in fact synergistically impair immune functionality, then it makes sense for there to be more worms when there are more species present. If multiple species can take hold readily in a host, then multiple worms of a given species ought to be able to do so as well. This phenomenon has been observed in rural Honduras and northern Rwanda (Sanchez et al. 2013, Mupfasoni et al. 2009).

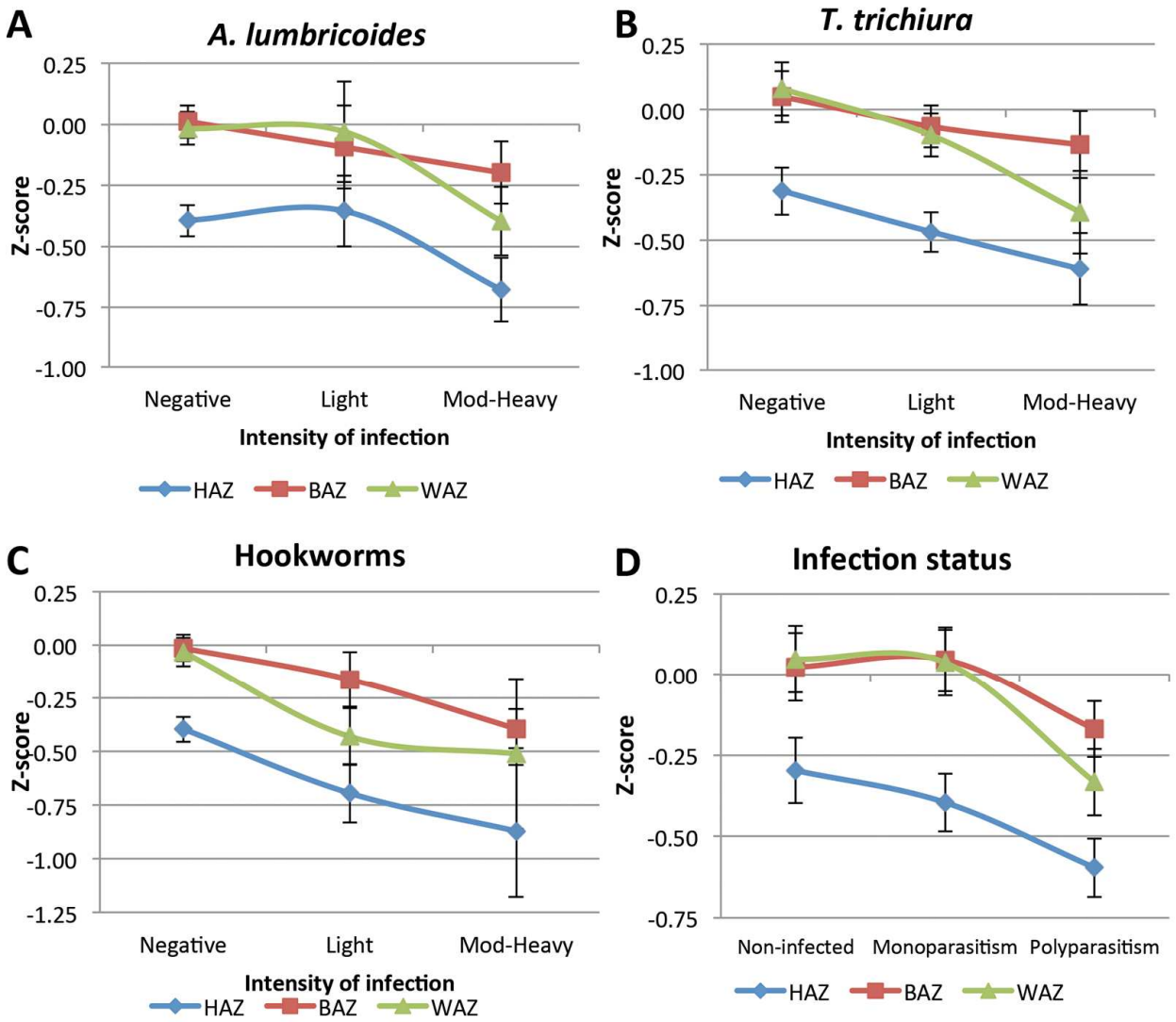
The fact that polyparasitism and heavier worm burden are associated with reduced treatment effectiveness is quite troubling, as co-infection is common in this community. This relationship highlights the importance of developing novel interventions that will both reduce co-infection prevalence and infection intensity, as anthelmintic therapy appears to be most effective in low intensity mono-infections. The relationship between these parameters should be further assessed in future studies.

To identify causal pathways that lead to polyparasitism and heavy worm burdens, both the risk factors for higher polyparasitism and the risk factors for greater infection intensity must be reviewed. Luckily, many of the risk factors are shared by these two outcomes: like higher polyparasitism infections, moderate and heavy STN infections are associated with pig ownership, a recent history of malaria infection, low dietary diversity, and low weight for height. The mechanisms responsible for these associations are likely similar, as both outcomes (high polyparasitism and high intensity) result in more helminths taking hold in a human host. However, where there was more variability in polyparasitism, there appear to be more direct associations with infection intensity, suggesting that this parameter may be more directly affected by the assessed risk factors.

Moderate and heavy infections are more common in younger children, while lighter infections are more common in older children. This is discordant with the understanding that worm burden should increase with age, due to increased exposure and longevity of the infection (Pullan et al. 2010). Possible explanations may include changing exposures (younger children are less hygiene-conscious and may be more prone to eating unwashed foods and travelling barefoot), developing partial immunity (over time children may become less susceptible to STN infection), or to increasing frequency of treatment (older children are more likely to be enrolled in school and to have been treated for STN infection recently enough to not have acquired a new *Trichuris* or *Ascaris* infection at the time of the study).

Infection intensity was inversely correlated with both weight for height and BMI, both of which were among the top five predictors of infection intensity when controlling

for all other paramters. This finding is in line with other studies that have shown diminished nutritional status to be associated with heavier STN infection (Pullan et al. 2010, Sanchez et al. 2013). It is curious that the effect is understated in this population, but the fact that both parameters were included in the top five predictors of infection intensity suggests their importance nonetheless.

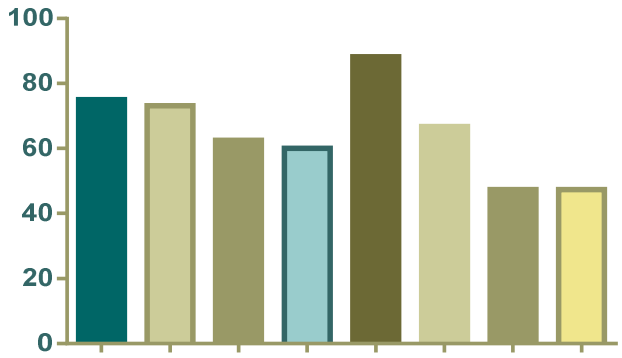


Increasing infection intensity is associated with lower height-for-age, BMI-for-age, and weight-for-age among schoolchildren in Honduras. (Sanchez et al. 2013)

Beyond the discussion of risk factors that lead to various types and degrees of STN infection is an equally important discussion of how well these infections can be

managed once they occur. The findings of this study related to albendazole treatment effectiveness are both unusual and concerning. As expected, the parameters most related to treatment effectiveness were structural or related to the worm itself, rather than to the child (treatment effectiveness ought to depend on susceptibility of the helminths, rather than on risk factors established by the host). Socioeconomic status, village, polyparasitism, sex, pig ownership, and the time between the last meal and treatment were the only parameters that bore any meaningful association to treatment success that have not yet been discussed.

Socioeconomic status and village of residence reflect structural factors that may increase the odds of contacting a resistant helminth: cure rates varied impressively among villages, ranging from nearly 90% in Bukira to only 47% in Kabonera and Kindulwe.



These findings were not significant, but this may be explained by low sample sizes when study participants were stratified by village. Kabonera and Kindulwe also have the greatest Muslim representation; this may account for the religion-based disparity

observed. The most plausible explanation is that the helminths that reside in Kabonera and Kindulwe are slightly less responsive to albendazole treatment than those that reside in the other villages. Such variety in cure rates across villages within the same geographic region has recently been observed in Ghana (Humphries et al. 2011). These two villages are somewhat isolated from the others, and each is home to an active religious community that is responsible for administering regular health-based interventions and is well-equipped to do so. Therefore, it is possible that the helminths in these areas have had greater exposure to albendazole and are beginning to develop resistance. Of course, this cannot be concluded with any reasonable degree of certainty from the data available, as no statistically meaningful associations were found between deworming history and treatment success. The fact that higher socioeconomic status was associated with higher rates of treatment failure, does, however, help corroborate this point: because deworming treatment is not consistently administered by schools in this area, many families choose to purchase treatment themselves. This is only an option for families that can afford such treatment. It is possible, then, that the families with a higher disposable income are spending more money to deworm their children regularly, and are thereby slowly contributing to the development of treatment failure in this area. These hypotheses are in line with current theoretical speculation about how anthelmintic resistance may occur among helminths that parasitize humans (Humphries et al. 2013, Geerts and Gryseels, 2001). Whether these different cure rates are evidence of anthelmintic resistance remains unclear in the absence of laboratory confirmation. However, the findings suggest that this is in urgent need of clarification, as anthelmintic resistance would pose a major threat to STN disease control.

	Livestock	Man
Treatment frequency (No./year)		
Gastro-intestinal nematodes	1–3	1–3
Onchocerca volvulus	NA	1–2
Schistosoma spp.	NA	Up to 1–2
Single drug regimens		
Gastro-intestinal nematodes	BZ or IVM	ALB
O. volvulus	NA	IVM
Schistosoma spp.	NA	PZQ
Targeting of treatments		
Kind of treatment	Mass	Target
Coverage (%)	± 100	< 80
Underdosing		
Weight underestimation	+++	+
Economic reasons	+++	++
Substandard drugs	+++	++

NA: not applicable; ALB: albendazole; BZ: benzimidazoles; IVM: Ivermectin; PZQ: praziquantel.

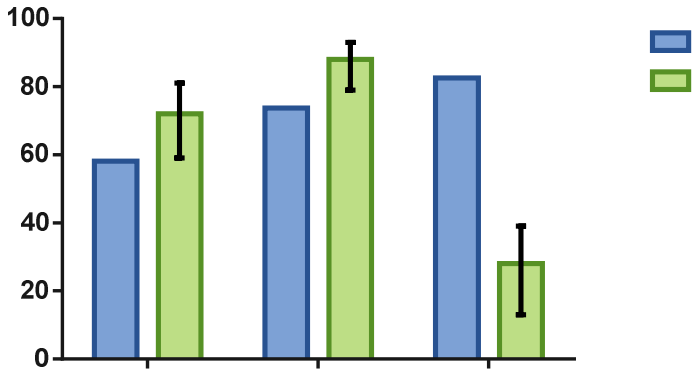
Risk factors associated with anthelmintic resistance in livestock and humans. (Geerts and Gryseels 2002)

Other findings related to cure rate reflect the physiology of the helminths and the internal environment of the host: children who had eaten more recently before taking the medication were less likely to be cured, as were children who had more worms and more types of helminths. The relationship between the time elapsed between the last meal and treatment administration has been minimally assessed; but albendazole is believed to be better absorbed by an empty stomach (Humphries, personal communication). This relationship is supported by the data in this study, as the children who experienced successful treatment tended to have last eaten at least 4 hours prior to treatment. The relationships between treatment effectiveness and polyparasitism and

intensity have already been discussed; the more worms present, the less likely the treatment is to cure the infection. This is perhaps due to the fact that when there are more helminths present, each worm is exposed to a smaller fraction of the active elements of the treatment, which may dilute its effect, similar to the phenomenon relating having a full stomach to treatment effectiveness. More research is needed to confirm these pathways, but the data from this study provides promising insights that suggest what the next steps should be in improving understanding of how treatment effectiveness is determined within a host.

Perhaps in line with the idea that the drug effect is diluted when many worms are present is the abnormally high cure rate observed for *T. trichiura*. A systematic review and meta-analysis of treatment effectiveness against STN infections found a cure rate of single-dose albendazole on *T. trichiura* infection to be 28% (95% CI 13% - 39%) (Keiser and Utzinger 2008). The cure rate and egg reduction rate in this study, however, approached 100%. The disparity between these numbers is enormous and perplexing. It is possible that the cure rate was unusually high because the infections were typically quite light; the majority of samples reflected burdens of only 24-72 eggs per gram (light intensity). Thus, it is plausible that each worm was subjected to a higher dose of treatment than in other studies where heavier infections and co-infections predominated. It is unlikely that this is an artifact of the lack of specificity in the Kato Katz technique, as any detection limits would apply to all of the helminths, and would artificially inflate cure rates (and deflate infection rates) for all species studied. It is also possible that *T. trichiura* worms in this area are just particularly susceptible to this treatment, although further evidence would be required to make that case. Lastly, it is

possible that this finding is in fact not unusual, and that the existing data just are not comprehensive enough to have taken note of the wide range of susceptibility among populations of *T. trichiura*. No matter the cause, this is, given current information, an unusual finding that warrants further investigation.



Unlike the rates seen for *T. trichiura* infection, the cure rates and egg reduction rates for hookworm and *A. lumbricoides* were in line with the literature reviewed by Keiser and Utzinger (2008). The fact that these findings are in line with the literature helps give credence to the rates observed for *T. trichiura*. Despite being within the realm of observed rates, the effectiveness of albendazole against hookworm remains concerning. The World Health Organization has stated that cure rates below 80% and egg reduction rates below 90% warrant concern for emerging anthelmintic resistance, and provide support for maintaining vigilant surveillance of treatment effectiveness in the region (World Health Organization). Both observed rates for treatment effectiveness against hookworm fall below these thresholds, corroborating earlier studies that have

also found possible evidence of emerging anthelmintic resistance in hookworm (Humphries et al. 2013, Soukhathammavong et al. 2012).

Ethical Concerns of the Study

Despite significant efforts to reduce ethical issues in this study, some concerns did arise over the course of its execution. The major concern was the fact that, as with all studies, enrollment had to be limited due to resource constraints, and not all at-risk individuals could be included. It is especially difficult to explain logistical limitations of a scientific study to people in communities that are unfamiliar with such work and the strict guidelines within which it is to be conducted. Throughout the course of the study, it was not uncommon for parents of included children or children from more distant villages to come to the Hope for African Children office to ask why they had not been recruited for enrollment in the study. No scientific researchers had ever come to Kabuwoko Parish before, and the community members were unfamiliar with cross-sectional studies. Though enrollment took place within the guidelines established by both the Yale University and Makerere University Institutional Review Boards, it is important to remember that scientific research is inherently exclusive. While it is well understood that the outcomes of the research are hoped to benefit communities at large, it is important not to lose sight of those who are left behind when research studies face resource limitations.

Another set of concerns arose within this study due to cultural differences and difficulties in communication. Many community members were under the impression that Jensen Reckhow was a doctor capable of diagnosing and treating a variety of

complex health problems. Not an issue brought on by lack of transparency or miscommunication, this confusion arose because the term “doctor” is loosely applied in this community and does not come with educational qualifications. Few rural healthcare workers in Rakai District are in fact certified doctors, but the term is used nonetheless, and the same level of expertise expected. When presented with medical cases, the research team referred individuals to receive care from the designated staff at Kabuwoko Health Centre III, and did not offer any specific medical advice.

Conducting public health research in a region in which the research team and study participants face a language barrier is immensely difficult, and requires significant forethought if it is to occur in a highly ethical fashion. Jensen Reckhow had spent time in the community before conducting this research, and was a trusted and valued friend by many of its residents. Establishing relationships like this is imperative to ensuring a productive, comfortable experience for all parties involved.

Limitations of the Study & Recommendations for the Field

Despite providing a useful body of information regarding the profile of STN infections in Kabuwoko Parish of Uganda, this study, and the field of research to which it contributes, is not without significant limitations. Perhaps most importantly, this study was small. It is difficult to characterize a community from such low enrollment rates, yet this type of study is not foreign to the field: the meta-analysis discussed above included 20 studies, yet treatment effectiveness estimates were made using data on only several hundred individuals (Keiser and Utzinger 2008). Studies in this field tend to be small and isolated; in many cases fewer than 300 individuals from a single village are

included. Thus, the accepted beliefs within the field are based on a handful of small studies. This creates, at best, a spotty picture of what the disease distribution, etiology, and risk factors really look like. Generalizations based on such limited data must be made with caution; greater emphasis ought to be put on conducting larger studies that may better capture regional variability and the true breadth of disease manifestations. Thus, as with most other studies in this field, the findings presented here must be interpreted with hesitation, as they make reflect abnormalities in the population rather than the norm.

Another limitation of this study was the use of a single Kato Katz test for diagnosing an individual. The technique has limited specificity to begin with, and typically two samples are taken per individual to minimize error (Tarafder et al. 2010). However, due to time and resource constraints, this was not possible. Given that, it is likely that some of the egg counts do not accurately reflect infection intensity, and that some infections were missed altogether. Because all samples suffered from the same lack of specificity and sensitivity, the internal associations are more likely to be fairly accurate; the identification of risk factors and potential causal pathways for STN infection would not be likely to change were the egg counts all made more accurate. However, the prevalence of STN infection may increase; it is reasonable to assume that estimates provided in this study are conservative.

While it would of course be better to have more accurate data and STN infection prevalence estimates, the data here already speak to major concerns in this area. The overall prevalence of STN infection is over 70%—well in excess of the thresholds establish by the WHO at which regular anthelmintic treatment is recommended (annual

treatment if above 20%; biannual treatment if above 50%). Anecdotal evidence and records from the Vector Control Division of the Ministry of Health suggest that these targets are far from being met: routine deworming in Rakai District ceased in 2008 (Vector Control Division). It is hoped that the findings of this study will help support a larger case for further investment in surveillance work in this region—and throughout the country—to ensure that currently used estimates accurately reflect current conditions on the ground. In an area as highly endemic as this one, there is an excellent case for routine intervention. This case is further supported by the evidence of emerging resistance highlighted in this study—if this is a real threat, it will need to be monitored vigilantly.

The findings of this study contribute to an existing body of research that hopes to characterize the risks and outcomes of STN infections. By highlighting some of the primary risk factors, such as pig ownership and personal hygiene, this research may be useful in guiding future interventions that target the populations most at risk. On the other hand, this research also helps characterize the negative outcomes of STN infection—namely poor physical development and increased malaria risk in spite of bednet use—that may help push the urgency of the issue. Lastly, the associations between intensity, polyparasitism, and treatment effectiveness point to the importance of upstream control measures (prevention) in making downstream control measures (treatment) more effective. By complimenting existing knowledge with information from a new study site, this research supports scientific understanding of STN infections and how they can be managed.

APPENDIX 1

Data collected from the Ministry of Health of Uganda, Vector Control Division

PREVALENCE DATA FOR MASAKA DISTRICT, 2010

Site	No. Examined	Schisto.		Hookworm		Ascaris		Trich.	
		No. +	Prev.	No. +	Prev.	No. +	Prev.	No. +	Prev.
Bukakata	15	3	20	1	6.7	3	20	7	46.7
Bulingo	15	0	0	4	26.7	0	0	3	20
Dimo	21	1	4.8	8	38.1	7	33.3	10	47.6
Kabasese	16	2	12.5	0	0	4	25	6	37.5
Kakyanga	16	3	18.8	0	0	1	6.3	4	25
Kamuwunga	16	1	6.3	2	12.5	1	6.3	6	37.5
Kasa	15	10	66.7	0	0	6	40	12	80
Kaziru	16	1	6.3	1	6.3	3	18.8	10	62.5
Kisuku	22	5	22.7	0	0	0	0	6	27.3
Lambu	15	11	73.3	2	13.3	0	0	5	33.3
Makonzi	15	3	20	2	13.3	0	0	4	26.7
Malembo	16	0	0	0	0	4	25	12	75
Mitondo	12	0	0	3	25	0	0	3	25
Nabugabo	15	1	6.7	3	20	0	0	2	13.3
Namirembe	15	0	0	2	13.3	1	6.7	6	40

PREVALENCE DATA FOR RAKAI DISTRICT, 1997

Site	No. Examined	Schisto.		Hookworm		Ascaris		Trich.	
		No. +	Prev.	No. +	Prev.	No. +	Prev.	No. +	Prev.
Kyebe	127	no data	8.2	no data	no data	no data	no data	no data	no data
Kyebe	148	no data	69.4	no data	no data	no data	no data	no data	no data
Kyebe	12	no data	6.1	no data	no data	no data	no data	no data	no data
Lwamaggwa	56	no data	16.3	no data	no data	no data	no data	no data	no data
	73	no data	0	no data	no data	no data	no data	no data	no data

PREVALENCE DATA FOR RAKAI DISTRICT, 2008

Site	No. Examined	Schisto.		Hookworm		Ascaris		Trich.	
		No. +	Prev.	No. +	Prev.	No. +	Prev.	No. +	Prev.
Misozi	59	0	0	no data	no data	8	13.6	11	18.6
Kasensero	53	1	1.9	no data	no data	8	15.1	17	32.1
Kakiri	60	0	0	no data	no data	0	0	5	8.3
Malemba	62	0	0	no data	no data	1	1.6	13	21
Ssemuto	61	0	0	no data	no data	0	0	9	14.8
Lwanga	60	1	1.7	no data	no data	0	0	4	6.7
St. Jude Bbale Kanagisa	59	2	3.4	no data	no data	2	3.4	7	11.9
Lugando	60	1	1.7	no data	no data	0	0	6	10
Ndolo	59	0	0	no data	no data	2	3.4	3	5.1
Kakunyu	58	0	0	no data	no data	1	1.7	2	3.4

APPENDIX 2

Consent and Assent Forms Used in the Study

Adult Consent Forms

ADULT/PARENTAL PERMISSION FORM
FOR PARTICIPATION IN A RESEARCH PROJECT
HOPE FOR AFRICAN CHILDREN
AND
YALE UNIVERSITY SCHOOL OF MEDICINE

HIC Proposal Title: The epidemiology of geohelminths.
Study Title: Helminth Infections among school-age children in Rakai District, Uganda
Makerere IRB Chairperson: *Paul Kutyabami (paulkutyabami@yahoo.com)*
Principal Investigator: *Keneth Kiyija, Hope for African Children*
Principal Investigator: *Bayanza Mugagga, Kabuwoko Health Clinic*
Principal Investigator: *Michael Cappello, MD, Yale University School of Medicine, New Haven CT USA*
Co-investigator: *Jensen Reckhow*
Funding Source: *Yale-Collaborative Action Project and The Thomas Rubin and Nina Russel Global Health Fellowship administered by the Yale School of Public Health*

Invitation to Participate and Description of Project

We are inviting **you and your chil**d to participate in a research study designed to look at infectious worms in your community. We believe that worm infections are common in your area, and these infections can lead to a number of health problems. We would like to take a closer look at infection rates and responses to treatment among residents of your community, and request that **you and your child** participate. We hope to enroll about 250 participants in this study, which is being conducted by *Hope for African Children* with *Yale University*.

We want to ensure that you have a good sense of the risks and benefits of participation in this study before you make a decision about your participation. This permission form details the research study, and a member of the research team will talk it through with you. This discussion will cover all aspects of the research process: our purpose for conducting this work, the procedures that will be performed and any associated risks therein, potential benefits and available alternative treatments. Once you have a good understanding of the study and feel capable of making an informed judgment about participation, you will be asked if you wish for **you and your child** to participate; if so, you will be asked to sign this form.

Description of Procedures

This study will be conducted this summer between June and August. If **you and your child** agree to participate, we will ask **you and your child** a series of questions about your family's habits, including bednet, latrine, and water usage. These factors may affect your child's risk of worm-related disease. If **you and your child** agree to participate in the study we will ask **your child** to provide a stool sample in a container we will provide. We will ask **your child** to bring the sample to school as soon as possible after the child has passed the stool. The stool sample will be analyzed in the laboratory for hookworm infection.

If **your child** is infected, we will escort **your child** to the health clinic to receive medical treatment. The treatment will be administered orally. Ten to fourteen days after treatment, we will collect another stool sample from **your child**. This will be used to determine if the treatment was effective.

We are always available and happy to answer questions you may have.

Risk and Inconvenience Involved

This study involves minimal risk for **you or your child**. The collection of stool involves minimal risks, as does the treatment regimen. We expect that **you and your child**'s participation in this study will take no longer than 2-5 hours this summer.

Benefits

By participating in this study, **you and your child** will benefit by learning if your child is infected with worms. If **your child** is infected, **your child** will be referred to medical treatment of the infection. Your community will also benefit from this study, as the knowledge gained about the extent of worm infections in your community and how individuals respond to treatment may help efforts to control these diseases in the future.

Economic Considerations

Participation in this study will not cost you anything but a small amount of your time.

Treatment Alternatives/Alternatives

You and your child's participation in this study is completely voluntary.
You can choose for **you and your child** to not participate in this study.
You may withdraw **you and your child** from this study at any time without losing any regular medical care.
Please ask as many questions as you like so that you understand this study.

Confidentiality

The information that we gather from this study will be returned to Yale University in the United States. Your identifiable information that is obtained in connection with this study will remain private and confidential. It will NOT be disclosed to anyone without your permission as required by U.S. law. Examples of information that we are legally required to disclose include abuse of a child or elderly person, or certain reportable diseases.

We will store your answers to questions and all information about **your child**'s infection status by code and not by name. All other information that we have with **your child**'s identity will be kept in locked files. After five years it will be destroyed. When the results of the research are published or discussed in conferences, no information will be included that would reveal **you or your child**'s identity unless your specific consent for this activity is obtained.

Representatives from the Yale University Human Investigation Committee may inspect our study records during internal auditing procedures. However, these individuals are legally required to keep all information confidential.

In Case of Injury

If **your child** is injured as a result of participation in this study, please contact Madam Goletti or Madam Josephine at the health clinic in Kabuwoko where you can obtain free medical care. Other than care for injuries due to participation in this study, no additional financial compensation for injury or lost wages is available.

Voluntary Participation and Withdrawal

You are free to choose for **you and your child** not to participate and if **you and your child** do become subjects, you are free to withdraw from this study at any time during its course. If you so choose, the answers you provided and any notes we have regarding your child's infection status will be deleted from the research database. If you choose for **you and your child** not to participate or if you withdraw, it will not harm your relationship with your own doctors.

Questions

We have used some technical terms in this form. Please feel free to ask about anything you don't understand and to consider this research and the consent form carefully—for as long as you feel is necessary—before you make a decision about participating.

Authorization

I have read (or someone has read to me) this form and have decided to participate in the project described above. Its general purposes, the particulars of involvement and possible hazards and inconveniences have been explained to my satisfaction. My signature also indicates that I have received a copy of this consent form.

Name of Subject:_____

Signature:_____

*Relationship:*___

Date:_____

_____ _____
Signature of Principal Investigator Date

or

_____ _____
Signature of Person Obtaining Consent Date

If you have further questions about this project or if you have a research related problem, you may contact the Principal Investigator Keneth Kiyija (256 782 744 608). If you have any questions concerning your rights as a research subject, you may contact the Makerere University Medical School Institutional Review Board in Kampala.

THIS FORM IS NOT VALID UNLESS THE FOLLOWING BOX

HAS BEEN COMPLETED BY THE HIC OFFICE

THIS FORM IS VALID ONLY THROUGH: _____. INITIALED: _____

ADULT/PARENTAL PERMISSION FORM
FOR
PARTICIPATION IN A RESEARCH PROJECT
HOPE FOR AFRICAN CHILDREN
AND
YALE UNIVERSITY SCHOOL OF MEDICINE

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Co-investigator: *Jensen Reckhow*
Funding Source: *Yale-Collaborative Action Project and The Thomas Rubin and Nina Russel Global Health Fellowship administered by the Yale School of Public Health*

OBUBAKA OBUTITTA OKWENYIGILA MUKUNONYEREREZZA

Tukuyita gwe n'omwanawo okwetaba mu musomo gw'okunonyerereza okulaba ebiwuka ebirwaza Ku kyalo kyamwe.Tukiliza nti ebiwuka bino bilibuli wamu mukitudu kyamwe,ela nga bileeta endwade nyinji.Twandiyagade okwekanya endwade n'obujanjabi bwa'bantu ku kyalo kyamwe,ela tusaba gwe n'omwanawo okwetaba mukunonyereza kuno.Tusubila okufuna abantu bibiri mu atano(250) Mukunonyeleza kuno okutekedwawo aba Hope For African Children With Yale University.

Twagala oku kakasa nti omanyi emitawana ne birugi ebiri mu musomo guno ngatonasalawo ku gwenyigilamu.Olupapula luno lunyonyola ebiri mu musomo guno, ela omu kubali mukibinja ekinonyereza ajja kukyogeramu nawe. Okwegeyamu kuno kujja kutwaliramu ebisexerwa byaffe byona ekubiri mu kunonyereza okugenda mu maaso: Ekisexerwa kyafe okukola omulimu guno,Emitendera eginakolebwa era n'emitwana egiri mu. Ebirungi ebisoboka n'engeri endala ez'obujjanjabi eziwo. Bwobaga otegede bulunji omusomo guno,era nga owulira osobola okusalawo okwenyigira mu. Ojakusabibwa oba wandiyagadde gwe n'omwana wo okwenyigira mu, singa kiba wekityo, ojja kusabibwa okuteka omukono ku lupapula luno.

OKUNYONYOLA EMITENDERA

Okusoma kuno kujja twalibwa musomo mukyeya kyo gw'omukaaga ne wakati w'ogwomunana. Singa gwe n'omwana wo mukiliza okwetaba mu, tujja kubabuzza yo ebibuzza ebikwata kumbera ya waka. Nga Obutimba bwensiri, toilet, n'amazzi gemukozesa. Bino byandiba ebyakabenje eri omwana ng'obulwadde obuletebwa enjoka z'omulubuto. Singa gwe n'omwana wo muliza okwetaba musomo guno tujja kusaba omwana wo atuutele obubi obubi bwe mu kikebe kyetunaba tumuwadde. Tujja kusaba omwana oyo alete ekikebe ekyo kusomero amangu dala. Obubi obwo bujjakutwalira bwekenenyezebwe mu labalatore oba omwana alina mu enjoka eziyitibwa enfaana.

Singa omwana wo asangibwa ngalina enjoka ezo, tujja kumuwerekera ko awafunibwa obujjanjabi era afune eddagala. Tujja funa obubi bw'omwana oyo obulala tubwekenenye okula nti eddagala lyakola bulungi.

Wetuli ebanga lyona era tuli basanyufu okudamu ebibuzzo byemulina.

OBUZIBU N'OKUTAWANYIZIBWA OKULIMU.
Omusomo guno gulimu obuzibu butono eri omwana wo. Nga okuleta obubi bwe, nemitendera gy'okujanjaba omwana. Tusubira omwana eyetabye mu musomo guno ajakutwala obudde obutasuka ssawa biri ne kitundu ($2^{1/2}$hours).

BYETUFUNAMU
Mukwtaba obwetabi musomo guno, omwana wo ajjakumanya singa ab'atawanyizibwa obulwadde obw'enjokka. N'abantu bekyalo kyo bajja kufuna mu musomo guno, amagezi agakwata ku bulwadde obusasanyibwa enjokka era nabuli muntu ayinza atya okujjanjaba oba kuyamba okwewala obulwadde buno mubisera ebijja mumaso.

OKUTUNULIRA EBY'ENFUNNA
Okwataba mu musomo guno tewetaga kusasula sente yonna naye okujjako obudde obutono enyo.

OBUJJANJABI OBW'ENGERI ENDALA
Gwe n'omwana wo okwetaba mu musomo guno kwa bwanakyewa.
Osobola okulonda wo gwe n'omwana wo obutetaba musomo guno.
Osobola gwe n'o mwana wo okuva mu musomo guna essawa yona.
Tuyambe obuzze ebibuzzo bingi nga bwoyagala osobole okutegera omusomo guno.

EBYEKYAMA.
Obubaka oba amawulire getukunganya okuva mu musomo guno bujja twalibwa ku YALE UNIVERSITY mu Amerika. Obubaka obufunidwa obukwatagana n'omusomo guno bujja kusigala nga bwakyama. Tebujja kufulumizibwa oba kubulirwa muntu yenna nga tokirizza nga bwekyetagisibwa mu matekka g'Amerika. Obumu ku bubaka bwetuyina okwanjula oba okufulumya mu matekka bwebwo nga okutulugunya omwana n'endwadde ezetagisibwa okwogerako n'okunonyerezebwa ko.

Tujja kuterka bulungi okudda mu kwo eri ebibuzzo n'obubaka obukwata kugwe n'omwana wo mungeri obulamu bwe webuyimiridde mungeri ya namba so si mulinyalye. Obubaka obulala obwendabika bwetulira obukwatako gwe n'omwana wo bujja kugalibwa mu fayilo. Wewanayitawo emyakka ettano bijjakusanyizibwa wo. Singa ebivudde mukunonyerezza bifulumizibwa oba bikubaganyizibwako ebirilowoozo mu lukungana, tewali bubaka bukwata kugwe n'omwana wo bujja kwogerwa ko okujjako nga ekitundu ekyo ekiniddwa kikwatako.

Akyikirira akakyiko akakulira okunonyerezza mu setendekero erya YALE ayinza okwekenenya ebivudde mu musomo guno ng'ali mukuteka ebintu mumitendera. Naye, buli muntu alina okukuma obubaka bwona nga bwakyama.

SINGA WAGWAWO AKABENJE OBA OBUVUNNE
Singa omwana wo afuna obuvunne nga engeri y'okwetaba mu musomo guno, tukirira Mukyala Goletti oba Josephine ku dwaliro e Kabuwoko woyinza okufuna obujjanjabi obwobwerere mukifo ky'okujanjaba ekiwundu atenga kyajja lwa kwetaba mu musomo guno. Tewali sente zina kudizibwa olw'ekiwundu oba omusaala gukulirindiridde.

OKWETABA N'O KUVAMU KWA BWANAKYEWA.
Oliwadembe gwe n'omwna wo obutetaba era singa mufuka omulamwa, muli baddembe okuva oba okuleka omusomo guno essawa yonna nga gugenda maaso.singa olonda wo nti okudamu kwewawadeyo nebyetulira ebikwata okubulamu bw'omwana bisimulwe bijja kusimurwa. Singa osalawo obutetaba oba obuteba mu, tekijja kutta nkolagana eriwo wakati wo n'abasawo.

EBIBUZZO
Tukozeseza enjogera oba olulimi olwekikugu mu fomu, oliwaddembe okubuzza ku kintu kyonna kyo tategedde era no kwekakasa olupapula luno olw'okunonyereza bulungi singa oba wetazze nga tonasalawo kwenyigiramu.

OKUKIRIZIBWA

Nsomye (oba waliwo ansomedde) mu lupapula (form) luno era nsazewo to kwenyigira mu pulojekiti eyogedwako wagulu. Kya migaso mingi, buli mutaawana oguli oba ogusobola okubawo gwo gedwako mu bukakafu bwange. Omukono gwange oguteredwa ku lupapula luno kitegeza nti nange lufunye ko era nensoma mu.

Erinya ly'esomo: ……..

Omukono: …….

Enkolagana: ……

Enaku z'omwezzi: ……

……..
……
Omukono gw'akulira okunonyereza Enaku z'omwezzi

Oba

……
……..
Omukono gw'oyo Akirizza Enaku z'omwezzi

Bwoba olina ebibuzzo ebirala ebikwata ku musomo guno (project) oba olina ekizibu ekyefananyirizako kukunonyereza kuno, Oobola okutukiririra akulirira okunonyereza kuno Keneth Kiyijja (256 782 744 608). Bwoba olina ekibuzo kyonna ekikwata ku demberyo nga gwe gwebanonyerezako, osobola okolagana ne Makerere University Medical School Institutional Review Board mu Kampala.

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Principal Investigator: *Bayanza Mugagga, Kabuwoko Health Clinic*
Principal Investigator: *Michael Cappello, MD, Yale University School of Medicine, New Haven CT USA*
Co-investigator: *Jensen Reckhow*
Funding Source: *Yale-Collaborative Action Project and The Thomas Rubin and Nina Russel Global Health Fellowship administered by the Yale School of Public Health*

Invitation to Participate and Description of Specimen Transfer

Thank **you and your child** for agreeing to participate in the study on worm infections being conducted by *Hope for African Children* and *Yale University*. We would like to request that, in addition to your participation in this study, you allow for us to keep a portion of the stool sample **you and your child** have provided for further research.

We want to ensure that you have a good sense of the risks and benefits of participation in this aspect of the study before you make a decision about your participation. This permission form details the nature of the continued research beyond the initial part of the study to which you have already agreed to participate, and a member of the research team will talk it through with you. This discussion will cover all aspects of this part of the research process: our purpose for conducting this work, where and how your samples will be taken, the procedures that will be performed and any associated risks therein, potential benefits, and how your privacy will be guaranteed. Once you have a good understanding of the study and feel capable of making an informed judgment about participation, you will be asked if you wish for **you and your child** to participate; if so, you will be asked to sign this form.

Overview of the Cappello Lab at Yale University
The Cappello Lab is a research facility located at Yale University in New Haven, Connecticut, in the United States of America. This laboratory conducts research on worm infections, running studies very similar to the one in which you are currently enrolled, in Ghana, Uganda, and Guatemala. The research team is interested in studying how worms cause disease in humans and how the worms respond to medical treatments. The goal of these studies is ultimately to develop better treatment options, and even a vaccine. In many of these studies, samples collected on-site are transported back to Yale in the United States for further scientific analysis, primarily for genetic sequencing.

Description of Procedures

If **you and your child** agree to participate, a portion of the stool sample provided by **your child** will be put aside for this study if your child is found to have worms. That portion of the sample will be cultured so that the worm eggs in the sample are able to develop into larvae. This will be done at Kabuwoko Health Centre III, where **you and your child** typically receive medical treatment when needed, in a secure setting so that only the research team has access to it. Once the larvae are cultured, they will be stored in a secure safe at the Health Centre until the rest of the study is finished, along with other samples provided by other people participating in this study. At that point, nobody will be able to tie any of the samples to original study participants—not even the people running the study. The sample provided by your child will only be identifiable by a code number, and will not be tied to your name at all. The sample will also only contain material from the worms with which your child was infected, and will not contain any human material from your child. Come August, your child's sample will be transferred to the Cappello Lab at Yale University in Jensen Reckhow's checked baggage on a commercial flight.

When your child's sample arrives at the Cappello Lab at Yale University, it will be stored in a locked cabinet until it is ready for use. Researchers at Yale University will use laboratory techniques to uncover the genetic code of the worms found in the sample, and will use that code to look at how the worms found in your child's stool respond to treatment. When this study is over, the samples will be disposed of forever as biohazard waste. Professionals will remove the samples to ensure they are properly and completely destroyed.

We are always available and happy to answer questions you may have.

Risk and Inconvenience Involved

This study involves minimal risk for **you or your child**. Participating in this part of the study does not require any additional time or effort from you, and we will ensure that any samples you provide to Yale University will not be tied to your name in any way, so that the samples provided cannot be traced back to you or your child. There is no risk that this identifying information will come out at any time, because it will never be recorded in relation to the sample you provide for this purpose.

Benefits

By participating in this part of the study, **you and your child** will not experience any additional benefits beyond those gained in the other parts of the study to which you have already agreed to participate. Your community may benefit from this part of the study, as the knowledge gained how individuals in your community respond to treatment may help efforts to control these diseases in the future.

Economic Considerations

Participation in this study will not cost you anything beyond the costs already explained in other parts of the study.

Confidentiality

No identifying information related to you or your child will be recorded for this part of the study, so the confidentiality of **you and your child** is guaranteed for this part of the study.

Voluntary Participation and Withdrawal

You are free to choose for **you and your child** not to participate and if **you and your child** do become subjects, you are free to withdraw from this study at any time during its course. **You and your child** may choose not to participate in this part of the study and may still participate in the other portion to which you have already agreed to participate. If you choose for **you and your child** not to participate or if you withdraw, it will not harm your relationship with your own doctors.

Questions

We have used some technical terms in this form. Please feel free to ask about anything you don't understand and to consider this research and the consent form carefully—for as long as you feel is necessary—before you make a decision about participating.

Authorization

I have read (or someone has read to me) this form and have decided to participate in the specimen transfer project described above. Its general purposes, the particulars of involvement and possible hazards and inconveniences have been explained to my satisfaction. My signature also indicates that I have received a copy of this consent form.

Name of Subject:_____

Signature:_____

*Relationship:*___*_____*

Date:_____

_____ _____
Signature of Principal Investigator Date

or

_____ _____

Signature of Person Obtaining Consent Date

If you have further questions about this project or if you have a research related problem, you may contact the Principal Investigator Keneth Kiyija (256 782 744 608). If you have any questions concerning your rights as a research subject, you may contact the Makerere University Medical School Institutional Review Board in Kampala.

THIS FORM IS NOT VALID UNLESS THE FOLLOWING BOX

HAS BEEN COMPLETED BY THE HIC OFFICE

THIS FORM IS VALID ONLY THROUGH: _____. INITIALED: _____

ADULT/PARENTAL PERMISSION FORM
FOR SPECIMEN TRANSFER
HOPE FOR AFRICAN CHILDREN
AND
YALE UNIVERSITY SCHOOL OF MEDICINE

HIC Proposal Title: The epidemiology of geohelminths.
Study Title: Helminth Infections among school-age children in Rakai District, Uganda
Makerere IRB Chairperson: *Paul Kutyabami (paulkutyabami@yahoo.com)*
Principal Investigator: *Keneth Kiyija, Hope for African Children*
Principal Investigator: *Bayanza Mugagga, Kabuwoko Health Clinic*
Principal Investigator: *Michael Cappello, MD, Yale University School of Medicine, New Haven CT USA*
Co-investigator: *Jensen Reckhow*
Funding Source: *Yale-Collaborative Action Project and The Thomas Rubin and Nina Russel Global Health Fellowship administered by the Yale School of Public Health*

Oyanirizibwa okwetaba mu kunonyereza kw'ebiwuka.

Webaale nnyo gwe no'mwana wo okukiriza okwetaba mu musomo gw'ebilwadde bwe biwuka by'omulubuto ogwatekebwawo ekitongole kya "Hope for African children and Yale University". Olwokwenyigirakwo mu musomo guno twandyagadde okubasaba okukuma obumu ku bukyafu bwamwe okwongera okubunonyerezako.

Twandyagadde okusoka okukumanyisa ku bulabe awamu n'emigaso egiri mu kwetaba mu musomo nga tonakola kusalawo kwo. Foomu eno eyokukiriziganya eraga ebivudde mu kunonyereza kw'omusomo gwe wakiriza okwetabamu,era omu kubanonyereza ajja kukyogeraamu naawe. Olukungana luno lugenda kubamu ebikwata ku binonyerezeddwako era ye nsonga lwaki tukola olukungana luno: Tujja kwongera ku wa era lwaki? Tugenda kozesa ebintu bino.Engeri gye tugenda obikwatamu,obulabe obubilimu era tubikuume nga byakyama.Buli anaba ayize era nga awulira ayagala okweyongerayo n'omusomo guno oba okubeera ekitundu fu ffe gwe oba omwana wo ojja kuteeka omukono ku foomu yaffe.

Ebikwata ku "Capello Lab eri ku Yale University."

“Capello Lab” kye kifo ekinonyereza nga kisangibwa ku Yale Universty mu “New Haven” esangibwa mu Amerika “Lab” eno enonyereza ku bikwata ku biwuka by’mulubuto nga etekateka emisomo nga guno gwolimu mu nsi ezenjawulo nga-: Ghana,Uganda ne Guatemala.Abanonyereza basayo omwoyo ku ngeri gyebijjajabibwamu. Ekisexerwa ky’omusomo guno kwe kufuna obujjajabi n’engeri y’okugema ebiwuka bino.

Byetugerezako tubizayo ku Yale University mu Amerika okwongera okunonyereza.

Okunyonyola mu mitendera.

Omuzadde n’omwana webaba bakiriza,omwana ajja kugibwako obubi era omwana bwaba asangiddwa n’obuwuka.obubi bujja kutelekebwa okutuusa nga amaggi agabaddemu gafuse ebiwuka era kino kijja kukolebwa e kabuwoko Health centre iii era eno okujjajabibwa gye kunabeera naye byona bijja kumibwa nga byakyama.tewali kirala kyonna kyetubetaaza yadde erinnya olwo byonna bijja kutwalibwa mu capello lab mu yale university mu mwezi gwa August.

Olwo no bajja kukozesa ebiwuka bino okusobola okufuna eddaggala eribijjajaba era nga okunonyereza kuwedde byona bijja kusanyizibwawo.

Obulabe n’okutataganya okulimu.

Mu kunonyereza kuno temuli buzibu bwona bwe mugenda kusanga era tetulina kirala kyetubetaza era tewali kigenda kuzulibwa nti kivudde mwono oba ono.

Byetufunamu.

Omugaaso gw’omusomo guno,tusuubira era nga tikiriza nti buli omu ajja kuyiga okuziyiza ebiwuka bino.

Ebyetagisa (ebisaale).

Tewali bisaale birala byetaagisa okugyako ebyabagambiddwa.

Bijja kuba byekusifu.

Byonna ebinazulibwa bijja kumibwa nga byakyama.

Engeri y’okwetabaamu

Omuntu yenna wa ddembe okwetaba mu musomo guno era yenna aba awulira nga ayagala okulekulira wa ddembe era kino tekigya kugyawo kolagaana ye wakati n’abasawo.

Ebibuzo.

Nga tonaba kola kusalawo kwo oli wa ddembe okubuza yenna gwe kikwatako.

Authorization

I have read (or someone has read to me) this form and have decided to participate in the specimen transfer project described above. Its general purposes, the particulars of involvement and possible hazards and inconveniences have been explained to my satisfaction. My signature also indicates that I have received a copy of this consent form.

Name of Subject:_____

Signature:_____

*Relationship:*___

Date:_____

_____ _____
Signature of Principal Investigator Date

or

_____ _____
Signature of Person Obtaining Consent Date

If you have further questions about this project or if you have a research related problem, you may contact the Principal Investigator Keneth Kiyija (256 782 744 608). If you have any questions concerning your rights as a research subject, you may contact the Makerere University Medical School Institutional Review Board in Kampala.

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Child Assent Forms

CHILD ASSENT FORM
FOR PARTICIPATION IN A RESEARCH PROJECT
HOPE FOR AFRICAN CHILDREN
AND
YALE UNIVERSITY SCHOOL OF MEDICINE

HIC Proposal Title: The epidemiology of geohelminths.
Study Title: Helminth Infections among school-age children in Rakai District, Uganda
Makerere IRB Chairperson: *Paul Kutyabami (paulkutyabami@yahoo.com)*
Principal Investigator: *Keneth Kiyija, Hope for African Children*
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Principal Investigator: *Michael Cappello, MD, Yale University School of Medicine, New Haven CT USA*
Co-investigator: *Jensen Reckhow*
Funding Source: *Yale-Collaborative Action Project and The Thomas Rubin and Nina Russel Global Health Fellowship administered by the Yale School of Public Health*

Why am I here?

We are asking you to take part in a research study because we are trying to learn more about parasites, who has them, and how we can better treat people with them. We are inviting you to be in the study because you live in a community where parasites are common.

Why are they doing this study?

We want to learn more about the general everyday behaviors of people in your community. Some of these behaviors may cause people to get parasites more often, and some of them may not. We want to know how to best prevent and treat parasite infection.

What will happen to me?

In this study, we will come to your house and ask your parents to answer some questions. We will ask you a few questions, too.

We will give you a container and ask for you to give us a stool sample. You will bring the collected stool sample to school. We will examine your stool sample in a laboratory and look for parasite eggs. If we find parasites, we will take you to the health clinic and they will give you medicine to try to get rid of them.

We will ask you to submit another stool sample 10-14 days after being treated. If there are still parasite eggs in the sample, we will again take you to the health clinic for treatment.

Will the study hurt?

No- this study only involves talking and submitting a stool sample.

Will the study help me?

The study may help us figure out how parasites are passed from person to person in your community. This may help us figure out how we can prevent you from getting sick with parasites in the future. If you have parasites now, you will be treated.

What if I have any questions?

You can ask any questions that you have about the study. If you have a question later that you didn't think of now, you can ask me next time.

Do my parents know about this?

This study was explained to your parents and they said that you could be in it. You can talk this over with them before you decide.

Do I have to be in the study?

You do not have to be in the study. No one will be upset if you don't want to do this. If you don't want to be in this study, you just have to tell them. You can say yes now and change your mind later. It's up to you.

Writing your name on this page means that that you agree to be in the study, and know what will happen to you. If you decide to quit the study all you have to do is tell the person in charge.

_____ _____
Signature of Child Date

_____ _____
Signature of Researcher Date

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Lwaki ndiwano?
Tukusaba wetabe mu musomo gw'okunonyereza kubanga tugezako okumanya ebisingawo kubiwuka, ani abirina era ani asobola okujjanjaba abantu aba birina. Tukuyita okubera mu musomo kubanga oli mutuzze wekitundu ekyo awali ebiwuka.

Lwaki bakola omusomo guno?
Twagala okuyigga ebisingawo kumpiisa z'abantu eza bulijjo mu kitundu kyo. Empiisa ezimu kuzino zisobola okuletera abantu ebiwuka buli kasera, nera ebimu bisobola obutaleta. Twagala okumanya tusobola tutya okubyewala n'okujanjaba obulwadde bw'ebiwuka.

Kiki ekinantukako?
Mu musomo guno, tujja kujja ewamwe era tujja kusaba bazadde bo okutudamu ebibuzo ebimu. Nawe tujja kukubuzza yo ebibuzzo ebitonotono.

Tujja ku kuwa omukebe era tukusabe otuwe obubi bwo butuno nyo. Ojakuleta omukebe ogwo ku somero. Tujja kwekenenya obubi obwo mu labalatore era tulabe amaggi g'ebiwuka ebyo. Singa osangibwa ng'olina ebiwuka, tujja kutwala awajanjabirwa era bajja kuwa eddagala eribijanjaba.

Tujja kusaba olete obubi obulala nga wayise wo enaku kumi oba kumi nanya (10 – 14) ng'omazze okujanjabibwa. Bweganaba amaggi gakyaliko, tujjakudamu tukutwale ofune obujanjabi.

Omusomo gulumya?
Nedda – omusomo gulimu kwogela na kuwayo bubibwo.

Omusomo gunanyamba?
Omusomo guno gusobola otuyamba okumanya ngeriki ebiwuka gyebitabula okuva ku muntu omu okuda kumulala ku kyalo. Gusobola otuyamba okumanya ngeri ki gyetuyinza okuziyizza gwe okufuna obulwadde bwebiwuka mu kisera kijja maaso. Bwoba ng'olina ebiwuka kati, ogenda kujanjabibwa.

Singa mba nganyina ebibuzzo byange?
Osobola okubuzza ebibuzzo byona byolina ebikwata ku musomo guno. Singa oba olina ebibuzzo byona ng'omusomo byotalowoozezako kati, osobola okumbuzza omulundi omulala

Bazadde bange bamanyi kino?
Omusomo guno nyonyoledwa bulungi eri abazzadde bo era nebagamba nti ogubere mu. Osobola okwogera ko nabo ku lw'omusomo guno nga tonasalawo.

Nyina okubera mu musomo guno?
Tolina kubera mu musomo guna. Tewali ajja kunyigira singa oba toyagadde ku kikola. Singa oba toyagala kwetaba musomo guno, oyina okubagamba. Osobola okugamba nti oja kwetaba mu ate n'okyusa endowoozayo nga wayisewo akasera. Kiri eri gwe?

Okuwandiika erinya lyo ku lupapula luno kitegezza nti okirizza okwetaba mu musomo guno, era bera ng'omanyi ekinakutukako. Singa oba oyagala okuva mu musomo guno, ky'olina okola kwe kutegezza oyo gwekikwata ko.

_____ _____
Omukono gw'omwana enaku z'omwezzi

_____ _____
Omukono gw'omunonyereza enaku z'omwezzi

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CHILD ASSENT FORM
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Co-investigator: *Jensen Reckhow*
Funding Source: *Yale-Collaborative Action Project and The Thomas Rubin and Nina Russel Global Health Fellowship administered by the Yale School of Public Health*

Why am I here?

We are asking you to take part in an additional aspect of the research study to which you have already agreed to participate because we are trying to learn more about the genetic identities of parasites so that we may develop better way to treat them. For this part of the study, we want to take some of the parasites we found in the stool sample you provided us with back home to the United States. We are inviting you to be in the study because you live in a community where parasites are common.

Why do they need my sample?

We want to look at the genetics of the parasites we found in your stool sample. To do that, we have to take the samples back to the United States so we can access the equipment necessary to process them. We want to do this because we think it will help us in our efforts to come up with a better treatment plan for parasites.

What will happen to me?

Nothing else will happen to you, beyond what you have already heard from the last time we talked. If you want to hear any of that again, just let us know, and we can review that section of the last form you signed. For this part of the study, we will take a portion of the stool sample you already provided, and we will take the parasites out of it. The parasites will then be taken to the United States so we can study how they infect humans and how they respond to treatment. The sample you provide will be kept with other samples, and once we collect it, nobody will be able to tell that it's yours anymore.

Will the study hurt?

No- again, this part of the study does not involve anything extra on your end, and the other parts of the study won't hurt either. Nobody will be able to tell that you have participated in this process, either, once the sample is collected.

Will the study help me?

The study may help us figure out how parasites hurt people in your community, and may help us figure out how to get rid of them.

What if I have any questions?

You can ask any questions that you have about the study. If you have a question later that you didn't think of now, you can ask me next time.

Do my parents know about this?

This study was explained to your parents and they said that you could be in it. You can talk this over with them before you decide.

Do I have to be in the study?

You do not have to be in the study. No one will be upset if you don't want to do this. If you don't want to be in this study, you just have to tell them. You can say yes now and change your mind later. It's up to you.

Writing your name on this page means that that you agree to be in this part of the study, and know what will happen to you. If you decide to quit this part or all of the study all you have to do is tell the person in charge.

_____ _____
Signature of Child Date

_____ _____
Signature of Researcher Date

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Lwaki ndi wano?
Tubasaba mutwegateko mu kwongera okunonyereza kwe wakiriza nga tunonyereza n'okuyiga ku biwuka bw'omusaayi kituyambe engeri gyetuyiza okubujjajabamu. Mu kusoma kuno,twagala okukozesa obuwuka bwetusanga mu bubi bwamwe nga tuzeyo mu Amerika.

Lwaki twetaga obubi bwo?
Twagala okutunulira ebika by'ebiwuka bye tusanze mu bubi bwamwe.Okukola kino twetaga okuzaayo ebimu bye tukebede mu Amerika. Twagala okukola kino kubanga tulowoza kijja kutuyamba okufuna obujjajabi.

Kiki ekinantukako?
Tewali kijja kukutukako okusinzira kwebyo bwetwayogera omulundi ogwayita. Bwoba wetaga okukudiramu tubulire tukunyonyole tusobole okutunulira akatundu akali ku foomu eyo gyewasiyininga. Mu kusoma kuno tujja kutwala a katundu ka sampo eyo jewatuwa tugijjemu obuwuka obwo. Obuwuka obwo bujja kutwalibwa mu America tusobole okulaba engeri gwebuyinza okukosa omubiri gwo muntu era tulabe negeri jetuyinza okubujjajjabamu.

Okusoma kuno kunanyigiriza?
Nedda,okusoma kuno tekujja kunyigiriza muntu y'enna era tewali agenda kumanya nti buno obubi bwono oba bwono.

Okusoma kuno kunanyamba?
Okusoma kuno kujja kutuyamba okumanya engeri obuwuka gye'bulumamu oba gyebukosamu omubiri n'abantu.

Bwemba nina ebibuuzo?
Oyinza okubuuza ebibuuzo byona byolina mu kunonyereza kuno. Bwoba ng'olina ekibuuzo kyobade tosubira kati, oyinza okumbuza ekiseera ekirara.

Bazadde bange kino bakimanyiko
Omusomo guno gwa nyonyolebwa bazadde bo era nebakiriza nti osobola okugwetabamu. Oyinza okwogelako nabo nga tonasalawo.

Nina okuba mu musomo guno?
Tolina kuba mu musomo guno. Tewali ajja kukunenya bwoba toyagala kugwetabamu. Bwoba toyagala bategeze. Oyinza okukiriza kati nokyusa ebilowozobyo.
Okuwandiika erinyalyo kulupapula luno kitegeza nti okiliza okwetaba mu musomo gunno era omanyi ekinabawo. Bwoba oyagala okuva mu musomo guno tegeza oyo ovunanyizibwako.

_____ _____
Signature of Child Date

_____ _____
Signature of Researcher Date

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INITIALED: _____

APPENDIX 3

Questionnaire

IDENTIFICATION	
CHILD NAME **DATE OF BIRTH** **AGE** **SEX** **HEIGHT** **WEIGHT** **SCHOOL/CLASS** **CHILD ID #** **HEAD OF HOUSEHOLD** **RESPONDENT NAME** **RELATIONSHIP OF RESPONDENT TO CHILD** **ADDRESS** **COMMUNITY** **INTERVIEWER NAME** **TRANSLATOR NAME** **QUESTIONNAIRE ANSWERS REVIEWED** **INITIALS**	

1. SOCIOECONOMIC INDICATORS		
QUESTIONS AND FILTERS	CODING CATEGORIES	ENTER #
What is the main material of the floor?	NATURAL FLOOR……..……..1 MATS COVERING FLOOR ……2 CEMENT FLOOR…..……..3	
What is the main material of the roof?	Thatch ……..1 Metal ……2 Other ……66	
What type of fuel does the household mainly use for cooking?	CHARCOAL ……..5 FIREWOOD/STRAW ……....6 DUNG……....7 OTHER_____66 (SPECIFY)	
Does any member of the household own agricultural land?	YES……1 NO……2 DON'T KNOW……..……..88	
Does any member of the household own at least one: 1.6a 1.6b 1.6c 1.6d	How many? COW……..……- GOAT……..……- POULTRY…..……- PIG……..……..-	
How far is the household from the nearest health facility? 1.8a 1.8b 1.8c 1.8d 1.8e	LESS THAN 1KM……..…..1 BETWEEN 1 AND 5KM..……2 BETWEEN 5 AND 10KM…….....3 GREATER THAN 10KM..……....4 DON'T KNOW……88	

How many people in the household? 1.9a 1.9b 1.9c 1.9d 1.9e	Total number _____ ≤ 5 yrs _____ 6-11 yrs _____ 12-15 yrs _____ Women > 15 _____ Men > 15 _____	
What is the primary religion of the household?	Muslim Christian Traditional (specify tribe) Other	

2. HUNGER			
Each of the questions in the following table is asked with a recall period of four weeks or 30 days. The respondent is first asked an occurrence question—that is, whether the condition in the question happened at all in the past four weeks (yes or no). If the respondent answers "yes" to an occurrence question, a frequency-of-occurrence question is then asked to determine whether the condition happened rarely (once or twice), sometimes (three to ten times), or often (more than ten times) in the past four weeks.			
NO.	Begin each question with "In the past four weeks…"	CODING CATEGORIES	ENTER #
2.1	…was there ever no food to eat of any kind in your household because of lack of resources to get food?	YES……..1 NO…. ……2 DON'T REMEMBER…..88 REFUSED……77	SKIP to Q22.2 if No (2)
2.1a	How often did this happen?	RARELY……..…1 SOMETIMES..……....2 OFTEN…..……....3	
2.2	…did you or any household member go to sleep at night hungry because there was not enough food?	YES……..1 NO…. ……2 DON'T REMEMBER.....88 REFUSED……77	SKIP to Q2.3 if No (2.9)
2.2a	How often did this happen?	RARELY……..…1 SOMETIMES..……....2 OFTEN…..……....3	
2.3	…did you or any household member go a whole day and night without eating anything because there was not enough food?	YES……..1 NO…… ……2 DON'T REMEMBER…..88 REFUSED……77	
2.3a	How often did this happen?	RARELY……..…1 SOMETIMES..……....2 OFTEN…..……....3	

3. HOUSEHOLD WATER SOURCES		
QUESTIONS AND FILTERS	CODING CATEGORIES	ENTER #
What is the main source of water for members of your household?	BOREHOLE……..... 1 DUG WELL..……..…… 2 RAINWATER…..…… 3 SURFACE WATER…… 4 OTHER_____ 66 (SPECIFY)	
Where is the water source located?	< 1KM FROM HOUSE……..2 ≥ 1KM FROM HOUSE……..3	

Do you do anything to the water to make it safer before drinking it?	YES……..1 NO…… ……2 DON'T KNOW……88	SKIP to Q4.1 if NO (2)
What do you do to the water to make it safer before drinking it?	YES NO BOIL……1 2 ADD ALUM……1 2 STRAIN THROUGH CLOTH……1 2 FILTER……1 2 LET IT SIT AND SETTLE…….1 2 OTHER_____ 66 (SPECIFY)	

3. TOILET FACILITIES			
NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	ENTER #
4.1	What kind of toilet facility do members of the household use?	YES NO PIT LATRINE……1 2 COMPOST……1 2 BUCKET……1 2 BUSH OR FIELD……1 2 OTHER_____ 66 (SPECIFY)	
4.2	Is this a public toilet facility?	YES…… ……..1 NO……2 DON'T KNOW……88	(SKIP TO Q5.1 if YES)
4.3	How many people use this facility?	ENTER # _____ DON'T KNOW ……..88	

3. EXPOSURE/DISEASE PREVENTION			
NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	ENTER #
5.1	Does your household have any mosquito nets that can be used while sleeping?	YES……..1 NO…… ……..2	SKIP to Q5.6 IF NO
5.2	How many mosquito nets does your household have? IF 7 OR MORE NETS, RECORD '7'.	NUMBER OF NETS……	
5.3	Does the child sleep under the mosquito net?	YES……1 NO……2 DON'T KNOW……...88	
5.4	How long ago did you obtain the mosquito net? IF MORE THAN 3 YEARS AGO, ENTER 55	MONTHS AGO…….. DON'T KNOW……...88	

5.5	How long ago was it last soaked or dipped? IF MORE THAN 3 YEARS AGO, ENTER 55	MONTHS AGO…….. DON'T KNOW……..……...88	
5.6	Did anyone sleep under the net last night?	YES……1 NO……2 DON'T KNOW……..……...88	
5.7	Has any member of your household had deworming medication in the past year?	YES……1 NO……2 DON'T KNOW……...88	
5.8	Has any member of your household had a fever in the last month?	YES……1 NO……2 DON'T KNOW……..……...88	
5.9	Has any member of your household had malaria in the past year?	YES……1 NO……2 DON'T KNOW……..……...88	
5.10	Does the child own shoes?	YES……1 NO……2 DON'T KNOW……..……...88	
5.11	If yes, how often does the child wear shoes?	ALMOST ALWAYS……1 SOMETIME EVERY DAY…… ……2 SOMETIME EVERY WEEK……3 RARELY……4 DON'T KNOW……..……...88	

3. HOUSEHOLD LISTING. Please identify other people in the household.
If more than 7, select in the following order (1) children 6-11 yrs, (2) children ≤ 5 yrs, (3) women > 15-45 yrs, (4) teens 12-15 yrs, (5) men > 15

LINE NO.	RELATIONSHIP TO INDEX CHILD	SEX	RESIDENCE	AGE	EDUCATION LEVEL	OCCUPATION	SHOES	BED NET USAGE
One line per person living or usually present in the household	What is the relationship of (#) to the index child?	Is (#) male or female?	Does (#) usually live here?	How old is (#)? IN YEARS	None...1 Primary…2 Jr High...3 Sr High...4 Vocational...5 Tertiary…6 Post Grad…7	SELF-DESCRIBED Farmer...1 Small trader...2 Student…3 None…4 Other (specify)	OWNS SHOES?	SLEPT UNDER BED NET LAST NIGHT?
(1)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Head of Household		M F 1 2	YES NO 1 2				YES NO 1 2	YES NO 1 2

Mother or Caregiver		1 2	1 2				1 2	1 2
03		1 2	1 2				1 2	1 2
04		1 2	1 2				1 2	1 2
05		1 2	1 2				1 2	1 2
06		1 2	1 2				1 2	1 2
07		1 2	1 2				1 2	1 2

CODES FOR RELATIONSHIP TO INDEX CHILD
01 = PARENT
02 = BROTHER/SISTER
03 = HALF SISTER/HALF BROTHER
04 = AUNT/UNCLE
05 = GRANDPARENT
06 = OTHER RELATIVE
07 = NOT RELATED
08 = DON'T KNOW

3. DIETARY DIVERSITY SCORE (INDEX CHILD ONLY)			
NO.	DID THE PARTICIPATING CHILD EAT THE FOLLOWING FOODS DURING THE DAY OR AT NIGHT?	YESTERDAY	IN THE PREVIOUS WEEK
8.1	Milk such as tinned, powdered, or fresh animal milk?	YES NO DK 1 2 88	YES NO DK 1 2 88
8.3	Bread, rice, noodles, or other foods made from grains?	1 2 88	1 2 88
8.4	Pumpkin, carrots, squash or sweet potatoes that are yellow or orange inside?	1 2 88	1 2 88
8.5	White potatoes, white yams, manioc, cassava, or any other foods made from roots?	1 2 88	1 2 88
8.6	Any dark green, leafy vegetables?	1 2 88	1 2 88
8.7	Ripe mangoes, papayas, or (INSERT ANY OTHER LOCALLY AVAILABLE VITAMIN A-RICH FRUITS)?	1 2 88	1 2 88
8.8	Any other fruits or vegetables?	1 2 88	1 2 88

8.9	Liver, kidney, heart or other organ meats?	1 2 88	1 2 88
8.10	Any meat, such as beef, pork, lamb, goat, chicken or duck?	1 2 88	1 2 88
8.11	Eggs?	1 2 88	1 2 88
8.12	Fresh or dried fish or shellfish?	1 2 88	1 2 88
8.13	Any foods made from beans, peas, lentils or nuts?	1 2 88	1 2 88
8.14	Cheese, yogurt or other milk products?	1 2 88	1 2 88
8.15	Any red palm oil or foods made with red palm oil?	1 2 88	1 2 88
8.16	Any other oil, fats, or butter, or foods made with any other oils, fats or butter?	1 2 88	1 2 88
8.17	Any sugary foods such as chocolates, sweets, candies, pastries, cakes or biscuits?	1 2 88	1 2 88

3. CHILD HEALTH INDICATORS (INDEX CHILD ONLY)			
NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	ENTER
9.1	When was the last time the INDEX CHILD consulted a healthcare worker?	IN THE LAST WEEK……..1 IN THE LAST MONTH……..2 IN THE LAST YEAR……..3 MORE THAN ONE YEAR……..4 NEVER……5 DON'T KNOW……88	
9.2	Where does the child get medical care if he/she is sick?	MASAKA HOSPITAL……....1 LOCAL CLINIC……..2 LOCAL HEALER……..3 FAMILY MEMBER……..4 DON'T KNOW……88 OTHER_____66 (SPECIFY)	
9.3	Where does the child get medications if he/she needs them?	MASAKA HOSPITAL……....1 LOCAL CLINIC……..2 LOCAL DRUG SELLER ……..3 LOCAL HEALER……..3 FAMILY MEMBER……..4 DON'T KNOW……88 OTHER_____66 (SPECIFY)	
9.4	Does the child have a health card?	YES……1 NO……2 DON'T KNOW……88	

9.5	Has the child ever received a vaccine?	YES……1 NO…(SKIP REMAINING QUESTIONS)……2 DON'T KNOW……88	
9.6	If so, against what disease(s) was he/she vaccinated?	TETANUS……...1 TYPHOID……...2 POLIO……3 DIPTHERIA……..4 YELLOW FEVER……5 TUBERCULOSIS (BCG)……...6 RABIES……7 MUMPS……8 MEASLES……9 RUBELLA……10 PERTUSSIS.....11 DON'T KNOW……..88 OTHER _____66 (SPECIFY)	
9.7	Vaccinations confirmed on health card?	YES……1 NO……2	
9.8	Where did the child get the vaccinations?	MASAKA HOSPITAL……....1 LOCAL CLINIC……..2 LOCAL HEALER……..3 FAMILY MEMBER……..4 DON'T KNOW……88 OTHER_____66 (SPECIFY)	

THANK YOU for all of your help. We are very grateful for your time.

ENDAGA	
ELINYA Y'OMWANA **AMAZALIBWA** **EMYAKKA** **EKIKULA** **OBUWANVU** **OBUZITTO** **ESSOMERO/EKIBIINA** **ENAMBA Y/OMWANA #** **OMUKULU W'OMAKKA** **ELINYA LY'OYO AVUNANYIZIBWA KUMWANA** **AMUYITA ATYA** **ENDAGIRIRO** **EKITUNDU WASANGIBWA** **ELINYA LY'OYO ABUZZA** **ELINYA LYO MUVUNUZI** **OLUPAPULA LW'EBIBUZZO LUTUNUDWA MU**	

3. EBIRAGA EMBEERA EYABULIJJO			
NO.	EBIBUZZO N'ENSENGEJJA	ENSENGEKA	YINGIZA #
1.1	Biki ebikola wansi oba eddiro ly'enyumba?	Takka lyoka……1 Mikeka gyegibaka ……2 Wasimetingibwa……...3	

1.2	Biki ebikola akasolya ?	Suubbi ……..1 byuma ……2 Ebirala ……66	
1.3	Biki byemukozesa okufumba?	Amaanda ……..5 Enku ……....6 Obussa……....7 Ebirala_____66 (Yawula	
1.4	Waliwo Omu Mumakka alina ettakka awalimirwa?	Yee……..1 Nedda……2 Simanyi……..88	
1.5	Waliwo Omu Mumakka alina ekimu kubino? 1.6a 1.6b 1.6c 1.6d	Bimekka? Ente……- Embuzzi……- Enkoko……- Embizzi……..-	
1.6	Banga ki Amakka lyegesudde okuva awafunibwa eby'obulamu? 1.8a 1.8b 1.8c 1.8d 1.8e	Kitundu kya kiro mitta($^{1/2}$KM)……..1 Wakati 1 Ne 5KM……2 Wakati5 Ne 10KM……....3 Wasukka mu 10KM……....4 Simanyi……88	
1.7	Abantu bamekka abali makka? 1.9a 1.9b 1.9c 1.9d 1.9e	Omuwendo gwonna _____ ≤ 5 yrs _____ 6-11 yrs _____ 12-15 yrs _____ Abakazi > 15 _____ Abaami > 15 _____	
1.8	Ddini ki eyasokka mu makka?	Ya bayisilamu Nzikiriza ya kristu Nzikiriza y'abyabuwangwa (yawula ekikka) Ebirala	

1. ENJALA			
Buli ekimu ku bubibuzzo bino ebiri mu mezza kibuziddwa okusinzira wakati webanga elya wikki enya oba enaku asatu (30 days). Oyo abuzibwa yasose kubuzibwa oba nti embeera eri mukibuzo yali emutuseko mubanga eriyise erya wikki enya (yee oba Nedda). Singa abuzibwa addamu "yee" ku kibuzzo ekibuzidwa, ebibuzzo ebiwerako bijja ku mubuzibwa okumanya embeera eyatukawo olindi (gumu oba ebiri), ebisera ebisinga (essatu ku kumi), oba buli kasera (emirundi gisoba mu kumi) mu wikki enya ezayitta.			
NO.	Tandika buli kibuzzo "mu wikki ennya ezayita…"	Ensengeka	Yingiza #

2.1	…wali obulidwako emmere ey'okuwa ab'omukka go kubanga tolina busobozi bwakufuna mu mmere?	Yee……...1 Nedda…. ……2 Sijukira …..88 Bagigana ……77	Bukka paka Q22.2 singa addamu nedda (2)
2.1a	Kino kitusewo emirundi emekka?	Lumu na lumu……..…1 Ebanga liyisewo.……....2 Emirundi mingi…..…….....3	
2.2	…Gwe oba omu ku bomumakka go yali asuzze ko enjala olw'okuba tewali mmere emalaa?	Yee……...1 Nedda…. ……2 Sijukira.....88 Yagigana.……77	Buka paka Q2.3 singa kiba nti nedda (2.9)
2.2a	Kino kyatukawo emirundi emekka?	Lumu na lumu……..…1 Ebisera ebimu.……....2 Buli kisera…..…….....3	
2.3	…Gwe oba omu ku b'omumaka go yali asibyeko era nasula nga talidde kintu kyona olw'okuba tewali mmere emala?	Yee……...1 Nedda.…… …….2 Sijukira…..88 Bajigana.……77	
2.3a	Kino kyatukawo emirundi emekka?	Lumu na lumu……..…1 Ebisera ebimu.……....2 Buli kisera…..…….....3	

4. AMAZZI AGAKOZEBWA AWAKKA			
NO.	EBIBUZZO N'ENSENGEJJA	ENSENGEKA	YINGIZZA #
3.1	Abo mu makka go amazzi bagajja wa?	Bowa ……..…..... 1 Kuluzi...……..…… 2 Mulembeka ga nkubba…..…… 3 Kiddiba…..…… 4 Walala_____ 66 (yawula)	
3.2	Amazzi gasangibwa wa?	Tewenka kiro meitta emu……..2 Wasuka kiro meitta emu ……..3	
3.3	Waliwo ekintu kyona kyokola amazzi okugafula amalungi nga tonaganywa?	Yee……..1 Nedda…… ……2 Simanyi……88	Bukawo paka Q4.1 singa agamba nedda (2)

3.3a	Kiki kyola amazzi okubera amalungi nga temunaganwa?	YES NO Gafumbibwa……1 2 Tugatamu Omunyu…….....1 2 Gayisa mu lugoye…..……..1 2 Kusengejja ….……...1 2 Ogalinda kutekka…….1 2 Engeri endala_____ 66 (yawula)	

3. KABUYONJO/ LATULINI			
NO.	EBIBUZZO	ENSENGEKKA	YINGIZA #
4.1	Kabuyonjo kikaki abawakka gyebakozesa?	Yee Nedda Ya kinya ……..……..…....1 2 Ya kuyola...……..……..1 2 Kikebe.……...1 2 Munsiko ……..……1 2 Engeri endala_____ 66 (yawula)	
4.2	Kabuyonjo eyo yakyalo kyonna?	Yee…… ….……..1 Nedda……2 Simanyi……88	(Buka paka Q5.1singa agamba nti yee)
4.3	Abantu bamekka abakozesa kabuyonjo eno?	Yingiza # _____ Simanyi ……..88	

3. OKUZIYIZA ENDWADDE			
NO.	EBIBUZZO	ENSENGEKA	YINGIZA #
5.1	Abo mu makka go balina obutimba bwensiri bwebasola okozesa nga bebasse?	Yee……..….1 Nedda…… ……...2	Buka paka Q5.6 singa Nedda
5.2	Mu makka go waliyo obutimba bwe nsiri bumekka? Singa buli 7 oba okusinga wo, wandiika '7'.	Namba y'obutimba……	
5.3	Omwana asula mu katimba ke nsiri?	Yee……1 Nedda……2 Simanyi……..……...88	
5.4	Omazze bangaki lye wafuniramu akatimba kensiri? Singa lisuka mu myaka 3 emabegga, Yingiza 55	Wayisewo omwezzi…….. Simanyi……..……...88	
5.5	Kakoma ddi okunyikibwa mu ddagala? Singa lisuka mu myaka 3 emabegga, Yingiza 55	Wayisewo omwezzi …….. Simanyi ……..……...88	

5.6	Waliwo omuntu yenna eyasuzze mu katimba ekiro ekyayise?	Yee……1 Nedda……2 Simanyi……..88	
5.7	Waliwo muntu yenna mu bomu makka go eyafuna endagala ly'ebiwuka omwaka oguwedde?	Yee……1 Nedda……2 Simanyi……..88	
5.8	Waliwo omuntu yenna kubo mumakka go eyalwala omusujja omwezzi oguwedde?	Yee……1 Nedda……2 Simanyi……..88	
5.9	Waliwo omuntu yenna kubo mumakka go eyalwala omusujja gw'ensiri mu mwaka oguwedde?	Yee……1 Nedda……2 Simanyi……..88	
5.10	Abaana balina engatto?	Yee……1 Nedda……2 Simanyi……..88	
5.11	Oba yee, Azambala emirundu emekka?	Kumpi buli kisera……1 Ekisera kimu na kimu olunaku…… ……2 Ekisera kimu na kimu ewiki……3 Lumu na lumu……4 Simanyi……..88	

3. ENSENGEKA Y'AMAKKA. Bambi oyogere abantu bolina mu makka.
Singa basukka mu 7, Londa mu mitendera (1) Abaana 6-11 yrs, (2) Abaana wansi 5 yrs, (3) Abakyala okuva15-45 yrs, (4) Abavubuka 12-15 yrs, (5) Abaami okuva 15

Olunyiriri.	Enkolagala n'omwana ayogerwako	Ekikula	Gy'abera	Emyaka	Yasoma kyenkanawa	Omulimo	Engatto	Akozessa akatimba k'ensiri
Walugand a oba wa munju akyikiridde omwana.	Omwana ayogerwako akuyitta atya oba mulina nkolagana ki?	Muwala oba mulenzi?	Abeera wo awaka buli kisera?	Alina emyaka emekka?	tewali…1 Primary…2 Siniya ezisoka...3 Yamalako siniya...4 Tendekero lye byemikono...5 Tendekero lye byokutunga…6 Yatikirwa …7	Gweyogerera Mulimi...1 Musubuzi ...2 Musomi…3 Tewali…4 Birala (yawula)	Oyina engatto?	Wasuzze mu katimba k'ensiri ekiro ekyayise?
(1)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Omukulu w'amakka		M F 1 2	Yee Nedda 1 2				Yee Nedda 1 2	Yee Nedda 1 2
Maama oba mulabirizi?		1 2	1 2				1 2	1 2

03		1 2	1 2				1 2	1 2
04		1 2	1 2				1 2	1 2
05		1 2	1 2				1 2	1 2
06		1 2	1 2				1 2	1 2
07		1 2	1 2				1 2	1 2

Enamba ez'enkolangana waki w'omwana:
01 = Bazadde
02 = Muganda we omulenzi oba Omuwala
03 = muganda omutto omuwala/omulenzi
04 = sega/kojja
05 = jjaaja
06 = aboluganda abalala
07 = siwalunganda
08 = simanyi

3. OBUBONERO BWEBIKA BY'EMMERE (Ku mwana yeka)			
NO.	Omwana eyetabyemu oba ayogerwako yalidde kummere eno wamanga emisana oba ekiro?	EGULO	MU WIKI EWEDDE
8.1	Amaata okugezza ag'omukkebe, ag'obuwunga oba ag'ente?	YEE NEDDA SIMANYI 1 2 88	YEE NEDDA SIMANYI 1 2 88
8.3	Omugatti, omucere, noodles, oba emmere endala eva munsigo?	1 2 88	1 2 88
8.4	Ensujju, carrots, squash oba lumode omuganda owakyenvu munda oba kacungwa?	1 2 88	1 2 88
8.5	Lumonde omweru, endagu, manioc, muwogo, oba emmere endala yona eva mumirandira?	1 2 88	1 2 88
8.6	Emmere endirwa?	1 2 88	1 2 88
8.7	Emiyembe egyengedde, papayas, or (yingiza mu ekibala ekirara ekirana ekirisa kya Vitamin A)?	1 2 88	1 2 88
8.8	Ebibala ebirala oba enva endirwa?	1 2 88	1 2 88

8.9	Ekibumba, Ensiggo, Omutima oba enyama endala?	1 2 88	1 2 88
8.10	Enyama yona, okugezza ey'omukebbe,embizzi, endigga, embuzzi, enkoko oaba embatta?	1 2 88	1 2 88
8.11	Amaggi?	1 2 88	1 2 88
8.12	Ekyenyanja ekibisi oba ekikalu oba mukene?	1 2 88	1 2 88
8.13	Emmere yonna eva mu bijanjalo, kawo oba mu binyebwa?	1 2 88	1 2 88
8.14	Omuzigo, bongo oba ebiva mu matta byonna?	1 2 88	1 2 88
8.15	Butto ava mu binazzi oba emmere eva mu binazzi?	1 2 88	1 2 88
8.16	Butto omulala yenna, amasavu, oba omuzigo, oba emmere eva mubutto oba amasavu n'omuzigo?	1 2 88	1 2 88
8.17	Eby'okulya ebiwomerera okugeza nga chocolates, sweets, candies, pastries, cakes oba biscuits?	1 2 88	1 2 88

3. EBILAGA OBULAMU BW'OMWANA (Omwana ayogerwako yekka)			
NO.	EBUBIZZO	ENSENGEKA	YINGIZA
9.1	Ddi omwana weyasembayo okutwlibwa eri omusawo akwatibwako?	MU WIKKI EWEDDE……..1 MU MWEZI OGUWEDDE……...2 MU MWAKA OGUWEDDE……...3 WAYISEWO OWAKKA GUMU……..4 TATWALIBWA NGA YO……5 SIMANYI……88	
9.2	Wa omwana gyafunira obujanjabi bwaba mulwadde?	EDWALIRO LY'EMASAKA……....1 AKALWALIRO KO KUKYALO……2 OMUJANJABI WO KUKYALO……...3 WALUGANDA……..4 SIMANYI……88 KIRALA_____66 (YAWULA)	

9.3	Wa omwana gyafunira obujanjabi bwaba abwetazzze?	EDWALIRO LY'EMASAKA……......1 AKALWALIRO KO KUKYALO..……..2 OMUTUNZI WEDDAGALA KUKYALO ……..3 OMUJANJABI WO KUKYALO……..3 WALUGANDA.……..4 SIMANYI……..…88 KIRALA_____66 (YAWULA)	
9.4	Omwana alina ekipande?	YEE……..1 NEDDA……2 SIMANYI……88	
9.5	Omwana yali agemedwa ko?	YEE……1 NEDDA……..(Buka ebibuzzo ebiddirira) ……2 SIMANYI……88	
9.6	Singa Abera ngayagemesebwa, Yagemesebwa ndwade ki?	TETANUS……..1 TYPHOID……...2 POLIO……3 DIPTHERIA……..4 YELLOW FEVER……5 TUBERCULOSIS (BCG)……...6 RABIES……7 MUMPS……8 OMULANGIRA……9 RUBELLA……10 PERTUSSIS.....1 SIMANYI……..88 OBULALA_____66 (YAWULA)	
9.7	Dozi zo kugema kw'omwana ziragibwa ku kipande?	YEE……1 NEDDA.……2	
9.8	Abaaba wabagemeseza wa?	MASAKA HOSPITAL……....1 AKALWALIRO KO KUKYALO..……..2 OMUJJANJABI WO KUKYALO……..3 OMUJJANJABI WA FAMULE……..4 SIMANYI……..…88 EBIRALA_____66 (YAWULA)	

Webale nyo obuyambi bwo. Twe yanziza obudde bwotuwadde.

APPENDIX 4

Materials Transfer Agreement

MTO.12990

Biological Material Transfer Agreement
("AGREEMENT")
1. PROVIDER: **Uganda National Council for Science and Technology**
On behalf of:
Kabuwoko Health Centre III
P.O. Box 40, Kalisizo, Rakai District, Uganda
2. PROVIDER SCIENTIST: **Bazanya Mugagga, M.D.**
3. RECIPIENT: **Yale University**
Grant & Contract Administration
47 College Street, Suite 203, New Haven, CT 06510 U.S.A
4. RECIPIENT SCIENTIST: **Michael Cappello, M.D.**, Professor of Pediatrics
On behalf of:
Jensen Reckhow, Yale School of Public Health
5. ORIGINAL MATERIAL: **Helminth Larvae**
6. RESEARCH PURPOSE: The requested material consists of helminth larvae harvested from fecal samples using the Baermann Method. The samples, obtained from children in Rakai District, Uganda, will include both pre- and post-treatment helminth specimens. The overall goal of the research is to isolate genetic factors that relate to the potential emergence of anthelminthic resistance in Rakai District, Uganda.For each larval sample, the DNA will be extracted and sequenced, with amplification focused specifically on the beta-tubulin gene, which is believed to be the target of anthelminthic drugs. Genetic polymorphisms will be analyzed with respect to observed treatment response in an effort to correlate specific genetic factors of this gene with treatment resistance. (RESEARCH PURPOSE is approved by RECIPIENT IRB (HIC) #1304011926.
7. RESEARCH LOCATION: Michael Cappello Laboratory
Yale University School of Medicine
Child Health Research Center 464 Congress Avenue, New Haven, CT 06520
I. Definitions:
1. MATERIAL: ORIGINAL MATERIAL, PROGENY, and UNMODIFIED DERIVATIVES. The MATERIAL shall not include: (a) MODIFICATIONS, or (b) other substances created by the RECIPIENT through the use of the MATERIAL which are not MODIFICATIONS, PROGENY, or UNMODIFIED DERIVATIVES.
2. PROGENY: Unmodified descendant from the MATERIAL, such as virus from virus, cell from cell, or organism from organism.
3. UNMODIFIED DERIVATIVES: Substances created by the RECIPIENT which constitute an unmodified functional subunit or product expressed by the ORIGINAL MATERIAL. Some examples include: subclones of unmodified cell lines, purified or fractionated subsets of the ORIGINAL MATERIAL, proteins expressed by DNA/RNA supplied by the PROVIDER, or monoclonal antibodies secreted by a hybridoma cell line.
4. MODIFICATIONS: Substances created by the RECIPIENT which contain/incorporate the MATERIAL.
5. COMMERCIAL PURPOSES: The sale, lease, license, or other transfer of the MATERIAL or MODIFICATIONS to a for-profit organization. COMMERCIAL PURPOSES shall also include uses of the MATERIAL or MODIFICATIONS by any organization, including RECIPIENT, to perform contract research, **MTO.12990**

to screen compound libraries, to produce or manufacture products for general sale, or to conduct research activities that result in any sale, lease, license, or transfer of the MATERIAL or MODIFICATIONS to a for-profit organization. However, industrially sponsored academic research shall not be considered a use of the MATERIAL or MODIFICATIONS for COMMERCIAL PURPOSES per se, unless any of the above conditions of this definition are met.
6. NONPROFIT ORGANIZATION(S): A university or other institution of higher education or an organization of the type described in section 501(c)(3) of the Internal Revenue Code of 1954 (26 U.S.C. 501(c)) and exempt from taxation under section 501(a) of the Internal Revenue Code (26 U.S.C. 501(a)) or any nonprofit scientific or educational organization qualified under a state nonprofit organization statute. As used herein, the term also includes government agencies.
II. Terms and Conditions of this Agreement:
1. The PROVIDER retains ownership of the MATERIAL, including any MATERIAL contained or incorporated in MODIFICATIONS.
2. The RECIPIENT retains ownership of: (a) MODIFICATIONS (except that, the PROVIDER retains ownership rights to the MATERIAL included therein), and (b) those substances created through the use of the MATERIAL or MODIFICATIONS, but which are not PROGENY, UNMODIFIED DERIVATIVES or MODIFICATIONS (i.e., do not contain the ORIGINAL MATERIAL, PROGENY, UNMODIFIED DERIVATIVES). If either 2 (a) or 2 (b) results from the collaborative efforts of the PROVIDER and the RECIPIENT, joint ownership may be negotiated.
3. The RECIPIENT and the RECIPIENT SCIENTIST agree that the MATERIAL:
(a) is to be used solely for teaching and academic research purposes;
(b) will not be used in human subjects, in clinical trials, or for diagnostic purposes involving human subjects without the written consent of the PROVIDER;
(c) is to be used only at the RECIPIENT organization and only in the RECIPIENT SCIENTIST's laboratory under the direction of the RECIPIENT SCIENTIST or others working under his/her direct supervision; and
(d) will not be transferred to anyone else within the RECIPIENT organization without the prior written consent of the PROVIDER.
4. The RECIPIENT and the RECIPIENT SCIENTIST agree to refer to the PROVIDER any request for the MATERIAL from anyone other than those persons working under the RECIPIENT SCIENTIST's direct supervision. To the extent supplies are available, the PROVIDER or the PROVIDER SCIENTIST agrees to make the MATERIAL available, under a separate implementing letter to this Agreement or other agreement having terms consistent with the terms of this Agreement, to other scientists (at least those at NONPROFIT ORGANIZATION(S)) who wish to replicate the RECIPIENT SCIENTIST's research; provided that such other scientists reimburse the PROVIDER for any costs relating to the preparation and distribution of the MATERIAL.
5. (a) The RECIPIENT and/or the RECIPIENT SCIENTIST shall have the right, without restriction, to distribute substances created by the RECIPIENT through the use of the ORIGINAL MATERIAL only if those substances are not PROGENY, UNMODIFIED DERIVATIVES, or MODIFICATIONS.
(b) Under a separate implementing letter to this Agreement (or an agreement at least as protective of the PROVIDER's rights), the RECIPIENT may distribute MODIFICATIONS to NONPROFIT ORGANIZATION(S) for research and teaching purposes only.
(c) Without written consent from the PROVIDER, the RECIPIENT and/or the RECIPIENT SCIENTIST may NOT provide MODIFICATIONS for COMMERCIAL PURPOSES. It is recognized by the RECIPIENT that such COMMERCIAL PURPOSES may require a commercial license from the PROVIDER and the PROVIDER has no obligation to grant a commercial license to its ownership interest in the MATERIAL incorporated in the MODIFICATIONS. Nothing in this paragraph, however, shall prevent the RECIPIENT
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from granting commercial licenses under the RECIPIENT's intellectual property rights claiming such MODIFICATIONS, or methods of their manufacture or their use.
6. The RECIPIENT acknowledges that the MATERIAL is or may be the subject of a patent application. Except as provided in this Agreement, no express or implied licenses or other rights are provided to the RECIPIENT under any patents, patent applications, trade secrets or other proprietary rights of the PROVIDER, including any altered forms of the MATERIAL made by the PROVIDER. In particular, no express or implied licenses or other rights are provided to use the MATERIAL, MODIFICATIONS, or any related patents of the PROVIDER for COMMERCIAL PURPOSES.
7. If the RECIPIENT desires to use or license the MATERIAL or MODIFICATIONS for COMMERCIAL PURPOSES, the RECIPIENT agrees, in advance of such use, to negotiate in good faith with the PROVIDER to establish the terms of a commercial license. It is understood by the RECIPIENT that the PROVIDER shall have no obligation to grant such a license to the RECIPIENT, and may grant exclusive or non-exclusive commercial licenses to others, or sell or assign all or part of the rights in the MATERIAL to any third party(ies), subject to any pre-existing rights held by others and obligations to the Federal Government.
8. The RECIPIENT is free to file patent application(s) claiming inventions made by the RECIPIENT through the use of the MATERIAL but agrees to notify the PROVIDER upon filing a patent application claiming MODIFICATIONS or method(s) of manufacture or use(s) of the MATERIAL.
9. Any MATERIAL delivered pursuant to this Agreement is understood to be experimental in nature and may have hazardous properties. The PROVIDER MAKES NO REPRESENTATIONS AND EXTENDS NO WARRANTIES OF ANY KIND, EITHER EXPRESSED OR IMPLIED. THERE ARE NO EXPRESS OR IMPLIED WARRANTIES OF MERCHANTABILITY OR FITNESS FOR A PARTICULAR PURPOSE, OR THAT THE USE OF THE MATERIAL WILL NOT INFRINGE ANY PATENT, COPYRIGHT, TRADEMARK, OR OTHER PROPRIETARY RIGHTS.
10. Except to the extent prohibited by law, the RECIPIENT assumes all liability for damages which may arise from its use, storage or disposal of the MATERIAL. The PROVIDER will not be liable to the RECIPIENT for any loss, claim or demand made by the RECIPIENT, or made against the RECIPIENT by any other party, due to or arising from the use of the MATERIAL by the RECIPIENT, except to the extent permitted by law when caused by the gross negligence or willful misconduct of the PROVIDER.
11. This agreement shall not be interpreted to prevent or delay publication of research findings resulting from the use of the MATERIAL or the MODIFICATIONS. The RECIPIENT SCIENTIST agrees to provide appropriate acknowledgement of the source of the MATERIAL or co-authorship to PROVIDER SCIENTIST in accordance with International Committee of Medical Journal Editors (ICMJE) Guidelines (Medical Education, 1999, 33, 066-078) in all publications.
12. The RECIPIENT agrees to use the MATERIAL in compliance with all applicable statutes and regulations, including Public Health Service and National Institutes of Health regulations and guidelines such as, for example, those relating to research involving the use of animals or recombinant DNA.
13. This Agreement will terminate on the earliest of the following dates: (a) when the MATERIAL becomes generally available from third parties, for example, though reagent catalogs or public depositories or (b) on completion of the RECIPIENT's current research with the MATERIAL, or (c) on thirty (30) days written notice by either party to the other, or (d) three (3) years from the date of final authorized signature on this AGREEMENT, provided that:
(i) if termination should occur under 13(a), the RECIPIENT shall be bound to the PROVIDER by the least restrictive terms applicable to the MATERIAL obtained from the then-available resources; and
(ii) if termination should occur under 13(b) or (d) above, the RECIPIENT will discontinue its use of the MATERIAL and will, upon direction of the PROVIDER, return or destroy any remaining MATERIAL. The RECIPIENT, at its discretion, will also either destroy the MODIFICATIONS or remain bound by the terms of this agreement as they apply to MODIFICATIONS;
and ---------- ----------~----------
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(iii) in the event the
PROVIDER
terminates this Agreement under 13(c) other than for breach of this Agreement or for cause such as an imminent health risk or patent infringement, the
PROVIDER
will defer the effective date of termination for a period of up to one year, upon request from the RECIPIENT,
to permit completion of research in progress.
Upon
the effective date of termination, or if requested, the deferred effective date of termination, RECIPIENT will discontinue its use of the MATERIAL and will, upon direction of the
PROVIDER,
return or destroy any remaining MATERIAL. The RECIPIENT, at its discretion, will also either destroy the MODIFICATIONS
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remain bound by the terms of this agreement as they apply to MODIFICATIONS. 14. Both parties shall discuss in good faith to enable the amicable resolution of matters, arising in connection with the interpretation or performance hereof as well as the matters which are not expressly set forth in this AGREEMENT. This AGREEMENT shall be interpreted and construed in accordance with the laws of the country of the defending patiy, namely laws of the State of Connecticut, in cases where the RECIPIENT is the defending party, or the laws of Uganda, in cases where
PROVIDER
is the defending party. Unless specified otherwise, reference in this agreement to a statute refers to that statute as it may be amended, or to any restated or successor legislation of comparable effect. 15 . Paragraphs 6, 9, and
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shall survive termination. 16. The MATERIAL is provided at no cost, or with an optional transmittal fee solely to reimburse the
PROVIDER
for its preparation and distribution costs. RECIPIENT SCIENTIST will be responsible for any costs to transfer the MATERIAL to RECIPIENT. **Uganda National Council for Science and Technology** Signed:
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Authorized Institutional Official Date: ----------~---------- Name: Title: Acknowledged by:
PROVIDER
SCIENTIST: Signed: Date: Name: Bazanya Mugagga, M.D. Title: **Yale University** Name: Donald B. Wiggin Title: Contract (MT A) Manager Grant & Contract Administration Acknowledged by: RECIPIENT SCIENTIST: Signed: Date: ---------- Name: Michael Cappello, M.D. Title: Professor ofPediatrics Signed:
Dffie:
_____
___
Name: Jensen Reckhow Title: MPH Candidate,
Yale
Public
Health

APPENDIX 5

Backward Selection Model Building Procedures

Backward Selection Process for Modeling STN Infection

Variables included in full model: age, village, sex, religion, schooling, shoe usage, deworming history, bednet use, pig ownership, head of household education, head of household occupation, malaria history, dietary diversity, socioeconomic status, weight/height, BMI

Model	# Parameters (DF)	Likelihood Ratio χ^2	Δ DF	Δ χ^2	Critical Value	Conclusion
Full Model	28	48.2831	--	--	--	--
-Village	21	43.3402	7	4.9429	14.067	Δ χ^2 < Critical Value, so removing village makes the model more parsimonious.
-Shoe Usage	19	33.2198	2	10.1204	5.991	Δ χ^2 < Critical Value, so removing shoe usage makes the model more parsimonious.
-SES Score	18	33.2175	1	0.0023	3.841	Δ χ^2 < Critical Value, so removing SES score makes the model more parsimonious.
-Bednet Use	17	33.1411	1	0.0764	3.841	Δ χ^2 < Critical Value, so removing bednet use makes the model more parsimonious.
-Religion	15	32.2162	2	0.9249	5.991	Δ χ^2 < Critical Value, so removing religion makes the model more parsimonious.
-HH Ed.	13	31.6468	2	0.5694	5.991	Δ χ^2 < Critical Value, so removing HH education makes the model more parsimonious.
-DDS	12	31.4181	1	0.2287	3.841	Δ χ^2 < Critical Value, so removing dietary diversity makes the model more parsimonious.
-Age	10	30.5723	2	0.8458	5.991	Δ χ^2 < Critical Value, so removing age makes the model more parsimonious.
-Schooling	7	29.7465	3	0.8258	7.815	Δ χ^2 < Critical Value, so removing schooling makes the model more parsimonious.
-Sex	6	28.5931	1	1.1534	3.841	Δ χ^2 < Critical Value, so removing sex makes the model more parsimonious.
-HH Occ.	5	27.4726	1	1.1205	3.841	Δ χ^2 < Critical Value, so removing HH occupation makes the model more parsimonious.
-Pigs	4	23.4634	1	4.0092	3.841	Δ χ^2 > Critical Value, so removing pig ownership

						does NOT improve the model. Pig ownership is added back in to the final model.

Backward Selection Process for Modeling Hookworm Infection

Variables included in full model: age, village, sex, religion, schooling, shoe usage, deworming history, bednet use, pig ownership, head of household education, head of household occupation, malaria history, dietary diversity, socioeconomic status, weight/height, BMI

Model	**# Parameters (DF)**	**Likelihood Ratio χ^2**	**Δ DF**	**Δ χ^2**	**Critical Value**	**Conclusion**
Full Model	28	24.1221	--	--	--	--
-Village	21	22.5211	7	1.601	14.067	Δ χ^2 < Critical Value, so removing village makes the model more parsimonious.
-Religion	19	22.2369	2	0.2842	5.991	Δ χ^2 < Critical Value, so removing religion makes the model more parsimonious.
-Age	17	22.0416	2	0.1953	5.991	Δ χ^2 < Critical Value, so removing age makes the model more parsimonious.
-Schooling	14	21.9460	3	0.0956	7.815	Δ χ^2 < Critical Value, so removing schooling makes the model more parsimonious.
-HH Occ.	13	21.9025	1	0.0435	3.841	Δ χ^2 < Critical Value, so removing HH occupation makes the model more parsimonious.
-HH Ed.	11	20.9590	2	0.9435	5.991	Δ χ^2 < Critical Value, so removing HH education makes the model more parsimonious.
-Sex	10	20.8312	1	0.1278	3.841	Δ χ^2 < Critical Value, so removing sex makes the model more parsimonious.
-SES Score	9	20.6194	1	0.2118	3.841	Δ χ^2 < Critical Value, so removing SES score makes the model more parsimonious.
-Shoe Usage	7	18.0934	2	2.5260	5.991	Δ χ^2 < Critical Value, so removing shoe usage makes the model more parsimonious.
-Bednet Use	6	17.8819	1	0.2115	3.841	Δ χ^2 < Critical Value, so removing bednet use makes the model more parsimonious.
-DDS	5	17.5470	1	0.3349	3.841	Δ χ^2 < Critical Value, so removing dietary diversity makes the model more parsimonious.
-Pig Ownership	4	16.6277	1	0.9193	3.841	Δ χ^2 < Critical Value, so removing pig ownership makes the model more

						parsimonious.
-Deworming History	3	12.9637	1	3.6640	3.841	$\Delta \chi^2$ < Critical Value, so removing deworming history makes the model more parsimonious.
-BMI	2	9.3974	1	3.5663	3.841	$\Delta \chi^2$ < Critical Value, so removing BMI makes the model more parsimonious.
-Malaria History	1	5.0321	1	4.3653	3.841	$\Delta \chi^2$ > Critical Value, so removing malaria history does NOT improve the model. Malaria history is added back in to the final model.

Backward Selection Process for Modeling Ascaris *Infection*

Variables included in full model: age, village,sex, religion, schooling, shoe usage, deworming history, bednet use, pig ownership, head of household education, head of household occupation, malaria history, dietary diversity, socioeconomic status, weight/height, BMI

Model	**# Parameters (DF)**	**Likelihood Ratio χ^2**	**Δ DF**	**$\Delta \chi^2$**	**Critical Value**	**Conclusion**
Full Model	28	39.2375	--	--	--	--
-Schooling	25	37.7963	3	1.4412	7.815	$\Delta \chi^2$ < Critical Value, so removing schooling makes the model more parsimonious.
-Pig Ownership	24	37.7824	1	0.0139	3.841	$\Delta \chi^2$ < Critical Value, so removing pig ownership makes the model more parsimonious.
-Village	17	31.4138	7	6.3686	14.067	$\Delta \chi^2$ < Critical Value, so removing village makes the model more parsimonious.
-Age	15	31.0630	2	0.3508	5.991	$\Delta \chi^2$ < Critical Value, so removing age makes the model more parsimonious.
-HH Ed.	13	30.0271	2	1.0359	5.991	$\Delta \chi^2$ < Critical Value, so removing HH education makes the model more parsimonious.
-DDS	12	29.9191	1	0.1080	3.841	$\Delta \chi^2$ < Critical Value, so removing dietary diversity makes the model more parsimonious.
-Shoe Usage	10	20.3740	2	9.5451	5.991	$\Delta \chi^2$ < Critical Value, so removing shoe usage makes the model more parsimonious.
-SES Score	9	20.3380	1	0.0360	3.841	$\Delta \chi^2$ < Critical Value, so removing SES score makes the model more parsimonious.
-Bednet Use	8	20.1127	2	0.2253	5.991	$\Delta \chi^2$ < Critical Value, so removing bednet use makes the model more parsimonious.

-Deworming History	7	19.8426	1	0.2701	3.841	$\Delta \chi^2$ < Critical Value, so removing deworming history makes the model more parsimonious.
-Sex	6	19.3080	1	0.5346	3.841	$\Delta \chi^2$ < Critical Value, so removing sex makes the model more parsimonious.
-Religion	4	16.7854	2	2.5226	5.991	$\Delta \chi^2$ < Critical Value, so removing religion makes the model more parsimonious.
-HH Occ.	3	14.0992	1	2.6862	3.841	$\Delta \chi^2$ < Critical Value, so removing HH occupation makes the model more parsimonious.
-BMI	2	11.2561	1	2.8431	3.841	$\Delta \chi^2$ < Critical Value, so removing BMI makes the model more parsimonious.
-Malaria	1	6.1617	1	5.0944	3.841	$\Delta \chi^2$ > Critical Value, so removing malaria history does NOT improve the model. Malaria history is added back in to the final model.

Backward Selection Process for Modeling Trichuris *Infection*

Variables included in full model: age, village, sex, religion, schooling, shoe usage, deworming history, bednet use, pig ownership, head of household education, head of household occupation, malaria history, dietary diversity, socioeconomic status, weight/height, BMI

Model	**# Parameters (DF)**	**Likelihood Ratio χ^2**	**Δ DF**	**$\Delta \chi^2$**	**Critical Value**	**Conclusion**
Full Model	28	50.6806	--	--	--	--
-Age	26	49.7746	2	0.9060	5.991	$\Delta \chi^2$ < Critical Value, so removing age makes the model more parsimonious.
-Village	19	33.0620	7	16.7126	14.067	$\Delta \chi^2$ < Critical Value, so removing village makes the model more parsimonious.
-Schooling	16	31.9676	3	1.0944	7.815	$\Delta \chi^2$ < Critical Value, so removing schooling makes the model more parsimonious.
-Religion	14	29.9245	2	2.0431	5.991	$\Delta \chi^2$ < Critical Value, so removing religion makes the model more parsimonious.
-SES Score	13	29.9209	1	0.0036	3.841	$\Delta \chi^2$ < Critical Value, so removing SES score makes the model more parsimonious.
-Sex	12	29.7666	1	0.1543	3.841	$\Delta \chi^2$ < Critical Value, so removing sex makes the model more parsimonious.
-HH Ed.	10	29.4152	2	0.3514	5.991	$\Delta \chi^2$ < Critical Value, so

						removing HH education makes the model more parsimonious.
-Shoe Usage	8	27.8020	2	1.6132	5.991	$\Delta \chi^2$ < Critical Value, so removing shoe usage makes the model more parsimonious.
-Malaria History	7	27.1455	1	0.6565	3.841	$\Delta \chi^2$ < Critical Value, so removing malaria history makes the model more parsimonious.
-Weight/ Height	6	23.7354	1	3.4101	3.841	$\Delta \chi^2$ < Critical Value, so removing weight for height makes the model more parsimonious.
- Deworming History	5	21.0139	1	2.7215	3.841	$\Delta \chi^2$ < Critical Value, so removing deworming history makes the model more parsimonious.
-DDS	4	17.8368	1	3.1771	3.841	$\Delta \chi^2$ < Critical Value, so removing dietary diversity makes the model more parsimonious.
-BMI	3	15.0120	1	2.8248	3.841	$\Delta \chi^2$ < Critical Value, so removing BMI makes the model more parsimonious.
-HH Occ.	2	11.6648	1	3.3472	3.841	$\Delta \chi^2$ < Critical Value, so removing HH occupation makes the model more parsimonious.
-Bednet Use	1	7.5859	1	4.0789	3.841	$\Delta \chi^2$ > Critical Value, so removing bednet use does NOT improve the model. Bednet use is added back in to the final model.

Backward Selection Process for Modeling Treatment Failure

Variables included in full model: age, village, sex, religion, schooling, shoe usage, deworming history, bednet use, pig ownership, head of household education, head of household occupation, malaria history, dietary diversity, socioeconomic status, weight/height, BMI, intensity, multiplicity, time between last meal and treatment

Model	**# Parameters (DF)**	**Likelihood Ratio χ^2**	**Δ DF**	**Δ χ^2**	**Critical Value**	**Conclusion**
Full Model	32	42.4696	--	--	--	--
-HH Ed.	30	39.8692	2	2.6004	5.991	$\Delta \chi^2$ < Critical Value, so removing HH education makes the model more parsimonious.
-Weight/ Height	29	39.8660	1	0.0032	3.841	$\Delta \chi^2$ < Critical Value, so removing weight for height makes the model more parsimonious.
-	28	39.8474	1	0.0186	3.841	$\Delta \chi^2$ < Critical Value, so removing deworming

Deworming History						history makes the model more parsimonious.
-Malaria History	27	39.8136	1	0.0338	3.841	$\Delta \chi^2$ < Critical Value, so removing malaria history makes the model more parsimonious.
-DDS	26	39.7824	1	0.0312	3.841	$\Delta \chi^2$ < Critical Value, so removing dietary diversity makes the model more parsimonious.
-Shoe Usage	24	39.3076	2	0.4748	5.991	$\Delta \chi^2$ < Critical Value, so removing shoe usage makes the model more parsimonious.
-Village	17	29.6694	7	9.6382	14.067	$\Delta \chi^2$ < Critical Value, so removing village makes the model more parsimonious.
-Schooling	14	27.8795	3	1.7899	7.815	$\Delta \chi^2$ < Critical Value, so removing schooling makes the model more parsimonious.
-Age	12	27.5772	2	0.3023	5.991	$\Delta \chi^2$ < Critical Value, so removing age makes the model more parsimonious.
-HH Occ.	11	27.2712	1	0.3060	3.841	$\Delta \chi^2$ < Critical Value, so removing HH occupation makes the model more parsimonious.
-BMI	10	26.5304	1	0.7408	3.841	$\Delta \chi^2$ < Critical Value, so removing BMI makes the model more parsimonious.
-Intensity	9	25.7841	1	0.7463	3.841	$\Delta \chi^2$ < Critical Value, so removing intensity makes the model more parsimonious.
-Religion	7	21.2262	2	4.5579	5.991	$\Delta \chi^2$ < Critical Value, so removing religion makes the model more parsimonious.
-Bednet Use	6	19.7088	1	1.5174	3.841	$\Delta \chi^2$ < Critical Value, so removing bednet use makes the model more parsimonious.
-Time to Treatment	5	17.4524	1	2.2564	3.841	$\Delta \chi^2$ < Critical Value, so removing time between the last meal and treatment makes the model more parsimonious.
-Sex	4	15.0711	1	2.3813	3.841	$\Delta \chi^2$ < Critical Value, so removing sex makes the model more parsimonious.
-Multiplicity	2	10.0063	2	5.0648	5.991	$\Delta \chi^2$ < Critical Value, so removing multiplicity makes the model more parsimonious.

-Pig Ownership	1	6.9452	1	3.0611	3.841	$\Delta \chi^2$ < Critical Value, so removing pig ownership makes the model more parsimonious. The only remaining predictor is significant, so model selection stops.

Backward Selection Process for Modeling Moderate/Heavy Infection

Variables included in full model: age, village, sex, religion, schooling, shoe usage, deworming history, bednet use, pig ownership, head of household education, head of household occupation, malaria history, dietary diversity, socioeconomic status, weight/height, BMI, multiplicity

Model	**# Parameters (DF)**	**Likelihood Ratio χ^2**	**Δ DF**	**Δ χ^2**	**Critical Value**	**Conclusion**
Full Model	30	34.7703	--	--	--	--
-DDS	29	34.7702	1	0.0001	3.841	$\Delta \chi^2$ < Critical Value, so removing dietary diversity makes the model more parsimonious.
-Village	22	29.0576	7	5.7126	14.067	$\Delta \chi^2$ < Critical Value, so removing village makes the model more parsimonious.
-Weight/ Height	21	29.0574	1	0.0002	3.841	$\Delta \chi^2$ < Critical Value, so removing weight for height makes the model more parsimonious.
-Schooling	18	26.0412	3	3.0162	7.815	$\Delta \chi^2$ < Critical Value, so removing schooling makes the model more parsimonious.
-Shoe Usage	16	25.3160	2	0.7252	5.991	$\Delta \chi^2$ < Critical Value, so removing shoe usage makes the model more parsimonious.
-Pig Ownership	15	23.1812	1	2.1348	3.841	$\Delta \chi^2$ < Critical Value, so removing pig ownership makes the model more parsimonious.
-Age	13	19.7049	2	3.4763	5.991	$\Delta \chi^2$ < Critical Value, so removing age makes the model more parsimonious.
-Sex	12	19.6666	1	0.0383	3.841	$\Delta \chi^2$ < Critical Value, so removing sex makes the model more parsimonious.
-Bednet Use	11	19.5456	1	0.1210	3.841	$\Delta \chi^2$ < Critical Value, so removing bednet use makes the model more parsimonious.
-BMI	10	19.3224	1	0.2232	3.841	$\Delta \chi^2$ < Critical Value, so removing BMI makes the model more parsimonious.
-HH Occ.	9	19.1445	1	0.1779	3.841	$\Delta \chi^2$ < Critical Value, so removing HH occupation makes the model more parsimonious.
-Religion	7	17.7775	2	1.3670	5.991	$\Delta \chi^2$ < Critical Value, so

						removing religion makes the model more parsimonious.
-HH Ed.	5	16.3617	2	1.4158	5.991	$\Delta \chi^2$ < Critical Value, so removing HH education makes the model more parsimonious.
-Deworming History	4	15.7313	1	0.6304	3.841	$\Delta \chi^2$ < Critical Value, so removing deworming history makes the model more parsimonious.
-Multiplicity	2	9.3539	2	6.3774	5.991	$\Delta \chi^2$ > Critical Value, so removing multiplicity does NOT improve the model. Multiplicity is added back in to the final model.

Backward Selection Process for Modeling Triple Co-Infection

Variables included in full model: age, village, sex, religion, schooling, shoe usage, deworming history, bednet use, pig ownership, head of household education, head of household occupation, malaria history, dietary diversity, socioeconomic status, weight/height, BMI, intensity

Model	**# Parameters (DF)**	**Likelihood Ratio χ^2**	**Δ DF**	**Δ χ^2**	**Critical Value**	**Conclusion**
Full Model	29	26.0449	--	--	--	--
-SES Score	28	26.0447	1	0.0002	3.841	$\Delta \chi^2$ < Critical Value, so removing SES score makes the model more parsimonious.
-Religion	26	23.9159	2	2.1288	5.991	$\Delta \chi^2$ < Critical Value, so removing religion makes the model more parsimonious.
-Village	19	20.8982	7	3.0177	14.067	$\Delta \chi^2$ < Critical Value, so removing village makes the model more parsimonious.
-Age	17	20.8104	2	0.0878	5.991	$\Delta \chi^2$ < Critical Value, so removing age makes the model more parsimonious.
-Schooling	14	20.0779	3	0.7325	7.815	$\Delta \chi^2$ < Critical Value, so removing schooling makes the model more parsimonious.
-Deworming History	13	20.0218	1	0.0561	3.841	$\Delta \chi^2$ < Critical Value, so removing deworming history makes the model more parsimonious.
-Pig Ownership	12	19.9512	1	0.0706	3.841	$\Delta \chi^2$ < Critical Value, so removing pig ownership makes the model more parsimonious.
-HH Ed.	10	19.0744	2	0.8768	5.991	$\Delta \chi^2$ < Critical Value, so removing HH education makes the model more parsimonious.
-Shoe	8	17.5781	2	1.4963	5.991	$\Delta \chi^2$ < Critical Value, so removing shoe usage

Usage						makes the model more parsimonious.
-Sex	7	17.4082	1	0.1699	3.841	$\Delta \chi^2$ < Critical Value, so removing sex makes the model more parsimonious.
-Weight/ Height	6	17.0668	1	0.3414	3.841	$\Delta \chi^2$ < Critical Value, so removing weight for height makes the model more parsimonious.
-Malaria History	5	16.4706	1	0.5962	3.841	$\Delta \chi^2$ < Critical Value, so removing malaria history makes the model more parsimonious.
-DDS	4	15.4869	1	0.9837	3.841	$\Delta \chi^2$ < Critical Value, so removing dietary diversity makes the model more parsimonious.
-BMI	3	13.2544	1	2.2325	3.841	$\Delta \chi^2$ < Critical Value, so removing BMI makes the model more parsimonious.
-HH Occ.	2	10.9596	1	2.2948	3.841	$\Delta \chi^2$ < Critical Value, so removing HH occupation makes the model more parsimonious.
-Bednet Use	1	6.3731	1	4.5865	3.841	$\Delta \chi^2$ > Critical Value, so removing bednet use does NOT improve the model. Bednet use is added back in to the final model.

APPENDIX 6

SAS Code Used for Final Featured Statistical Analyses

```
libname epi 'C:\Users\jdr9\Downloads';
proc import
        datafile =
'c:\users\jdr9\downloads\EpiData1.xls'
        dbms = xls
        out = epi.one
        replace;
run;
data deworm2;
        set epi.one;
/*recoding variables to facilitate
meaningful analysis*/
/*recoding shoe usage variable to include
class level variable and a binary variable -
does shoe usage matter, or shoe possession,
or neither?*/
        if shoes = 1 or shoes = 2 then do;
                hasshoes = 1; end;
        else if shoes =3 then do;
                hasshoes = 0; end;
/*recoding cure and intensity variables for
logistic modeling*/
        cure=SUM(HWCure, ALCure, TTCure);
        if cure = 0 then do;
                cure = 1; end;
        else if cure >=1 then do;
                cure = 0; end;
        int=SUM(HWint, ALint, TTint);
        if int = 0 then do;
                int = 1; end;
        else if int >= 0 then do;
                int = 2; end;
        else if int = . then do;
                int = 0; end;
        if int = 0 then intensity = .;
/*creating class level and binary variables
for head of household education: does level
of education matter, or just having any
education at all, or neither?*/
        if hhed = 1 then hhbinary = 0;
                else if hhed >=2 then
hhbinary = 1;
/*creating class level variable for
polyparasitism*/
        if 1 <= infection <= 3 then wormnum =
1;
                else if infection = 0 then
wormnum = 0;
                else if 4 <= infection <= 6
then wormnum = 2;
                else if infection = 7 then
wormnum = 3;
/*creating dummy variables for logistic
modeling*/
        if 0 <= religion <= 2 then do;
                chris = (religion = 1);
```

```
                mus = (religion = 0);
        end;
        if 1 <= hhed <= 3 then do;
                some_prim = (hhed = 2);
                some_sec = (hhed = 3);
        end;
        if 1<= int <= 2 then do;
                mhtol = (int = 2);
        end;
        if 0 <= agegroup <= 3 then do;
                sixten = (agegroup = 1);
                elevpls = (agegroup = 2);
        end;
        if 0 <= schooling <= 3 then do;
                nurs = (schooling = 1);
                prim = (schooling = 2);
                sec = (schooling = 3);
        end;
        if 1 <= shoes <= 3 then do;
                daily = (shoes = 1);
                weekly = (shoes = 2);
        end;
        if 0 <= wormnum <= 3 then do;
                one = (wormnum = 1);
                two = (wormnum = 2);
                three = (wormnum = 3);
        end;
        if village = 'Bukira' or village =
'Bukunda' or village = 'Busowe' or village =
'Dwaniro' or village = 'Kabonera' or village
= 'Kabuwoko' or village = 'Kindulwe' or
village = 'Segero' then do;
                bukira = (village =
'Bukira');
                bukunda = (village =
'Bukunda');
                busowe = (village =
'Busowe');
                dwaniro = (village =
'Dwaniro');
                kabonera = (village =
'Kabonera');
                kabuwoko = (village =
'Kabuwoko');
                kindulwe = (village =
'Kindulwe');
        end;
        if infection = 1 then hwonly = 1;
                else if infection ne 1 then
hwonly = 2;
        if infection = 2 then alonly = 1;
                else if infection ne 2 then
alonly = 2;
        if infection = 3 then ttonly = 1;
                else if infection ne 3 then
ttonly = 2;
        if infection = 4 then hwal = 1;
                else if infection ne 4 then
hwal = 2;
        if infection = 5 then hwtt = 1;
                else if infection ne 5 then
hwtt = 2;
        if infection = 6 then altt = 1;
                else if infection ne 6 then
altt = 2;
        if infection = 7 then hwaltt = 1;
                else if infection ne 7 then
hwaltt = 2;
        if int = 1 then intensity = 0;
                else if int = 2 then
intensity = 1;
        if wormnum = 1 then worm = 1;
                else if wormnum = 2 then worm
= 2;
                else if wormnum = 3 then worm
= 3;
        if 1<=worm<=3 then do;
                twow = (worm = 2);
                threew = (worm = 3);
        end;
run;
/*checking recoding work for errors*/
proc freq;
        tables shoes*hasshoes
        cure*hwcure*alcure*ttcure
        int*intensity*hwint*alint*ttint
        hhed*hhbinary
        infection*wormnum*worm
        religion*chris*mus
        hhed*some_prim*some_sec
        int*mhtol
        agegroup*sixten*elevpls
        schooling*nurs*prim*sec
        shoes*daily*weekly
        wormnum*one*two*three
        village*bukira*bukunda*busowe*dwaniro
*kabonera*kabuwoko*kindulwe
        infection*hwonly*alonly*ttonly*hwal*h
wtt*altt*hwaltt
        wormnum*worm*twow*threew
        / list missing;
        run;
/*unadjusted associations for categorical
variables: chisq test for p value; logistic
for ORs*/
proc freq;
        tables (agegroup village gender
religion schooling shoe usage dewormed
childnet pigs hhed hhocc malaria) * infected
/ chisq relrisk;
run;
proc logistic;
        model infected = sixten elevpls;
run; quit;
proc logistic;
        model infected = bukira bukunda
busowe dwaniro kabonera kabuwoko kindulwe;
run; quit;
proc logistic;
        model infected = chris mus;
run; quit;
proc logistic;
        model infected = nurs prim sec;
run; quit;
proc logistic;
        model infected = weekly daily;
run; quit;
proc logistic;
        model infected = some_prim some_sec;
run; quit;
proc logistic;
        model infected = dds;
run; quit;
proc logistic;
        model infected = ses_score;
run; quit;
proc logistic;
        model infected = wh;
run; quit;
proc logistic;
        model infected = bmi;
run; quit;
```

```
proc freq;
       tables (agegroup village gender
religion schooling shoe usage dewormed
childnet pigs hhed hhocc malaria) * hw /
chisq relrisk;
run;
proc logistic;
model hw = sixten elevpls;
run; quit;
proc logistic;
       model hw = bukira bukunda busowe
dwaniro kabonera kabuwoko kindulwe;
run; quit;
proc logistic;
       model hw = chris mus;
run; quit;
proc logistic;
       model hw = nurs prim sec;
run; quit;
proc logistic;
       model hw = weekly daily;
run; quit;
proc logistic;
       model hw = some_prim some_sec;
run; quit;
proc logistic;
       model hw = dds;
run; quit;
proc logistic;
       model hw = ses_score;
run; quit;
proc logistic;
       model hw = wh;
run; quit;
proc logistic;
       model hw = bmi;
run; quit;

proc freq;
       tables (agegroup village gender
religion schooling shoe usage dewormed
childnet pigs hhed hhocc malaria) * al /
chisq relrisk;
run;
proc logistic;
       model al = sixten elevpls;
run; quit;
proc logistic;
       model al = bukira bukunda busowe
dwaniro kabonera kabuwoko kindulwe;
run; quit;
proc logistic;
       model al = chris mus;
run; quit;
proc logistic;
       model al = nurs prim sec;
run; quit;
proc logistic;
       model al = weekly daily;
run; quit;
proc logistic;
       model al = some_prim some_sec;
run; quit;
proc logistic;
       model al = dds;
run; quit;
proc logistic;
       model al = ses_score;
run; quit;
proc logistic;
       model al = wh;
run; quit;
proc logistic;
       model al = bmi;
run; quit;

proc freq;
       tables (agegroup village gender
religion schooling shoe usage dewormed
childnet pigs hhed hhocc malaria) * tt /
chisq relrisk;
run;
proc logistic;
       model tt = sixten elevpls;
run; quit;
proc logistic;
       model tt = bukira bukunda busowe
dwaniro kabonera kabuwoko kindulwe;
run; quit;
proc logistic;
       model tt = chris mus;
run; quit;
proc logistic;
       model tt = nurs prim sec;
run; quit;
proc logistic;
       model tt = weekly daily;
run; quit;
proc logistic;
       model tt = some_prim some_sec;
run; quit;
proc logistic;
       model tt = dds;
run; quit;
proc logistic;
       model tt = ses_score;
run; quit;
proc logistic;
       model tt = wh;
run; quit;
proc logistic;
       model tt = bmi;
run; quit;

proc freq;
       tables (agegroup village gender
religion schooling shoe usage dewormed
childnet pigs hhed hhocc malaria intensity
cure) * infection / chisq;
run;

proc freq;
       tables (agegroup village gender
religion schooling shoe usage dewormed
childnet pigs hhed hhocc malaria intensity
worm) * cure / chisq relrisk;
run;
proc logistic;
       model cure = sixten elevpls;
run; quit;
proc logistic;
       model cure = bukira bukunda busowe
dwaniro kabonera kabuwoko kindulwe;
run; quit;
proc logistic;
       model cure = chris mus;
run; quit;
proc logistic;
       model cure = nurs prim sec;
run; quit;
proc logistic;
```

```
        model cure = weekly daily;
run; quit;
proc logistic;
        model cure = some_prim some_sec;
run; quit;
proc logistic;
        model cure = dds;
run; quit;
proc logistic;
        model cure = ses_score;
run; quit;
proc logistic;
        model cure = wh;
run; quit;
proc logistic;
        model cure = bmi;
run; quit;
proc logistic;
        model cure = mhtol;
run; quit;
proc logistic;
        model cure = twow threew;
run; quit;
proc logistic;
        model cure = treattime;
run; quit;

proc freq;
        tables (agegroup village gender
religion schooling shoe usage dewormed
childnet pigs hhed hhocc malaria worm) * int
/ chisq;
run;
proc logistic;
        model int = sixten elevpls;
run; quit;
proc logistic;
        model int = bukira bukunda busowe
dwaniro kabonera kabuwoko kindulwe;
run; quit;
proc logistic;
        model int = gender;
run; quit;
proc logistic;
        model int = chris mus;
run; quit;
proc logistic;
        model int = nurs prim sec;
run; quit;
proc logistic;
        model int = weekly daily;
run; quit;
proc logistic;
        model int = dewormed;
run; quit;
proc logistic;
        model int = childnet;
run; quit;
proc logistic;
        model int = pigs;
run; quit;
proc logistic;
        model int = some_prim some_sec;
run; quit;
proc logistic;
        model int = dds;
run; quit;
proc logistic;
        model int = ses_score;
run; quit;
proc logistic;
        model int = wh;
run; quit;
proc logistic;
        model int = bmi;
run; quit;
proc logistic;
        model int = twow threew;
run; quit;

proc freq;
        tables (agegroup village gender
religion schooling shoe usage dewormed
childnet pigs hhed hhocc malaria intensity
cure) * wormnum / chisq;
run;
proc logistic;
        model wormnum = sixten elevpls;
run; quit;
proc logistic;
        model wormnum = bukira bukunda busowe
dwaniro kabonera kabuwoko kindulwe;
run; quit;
proc logistic;
        model wormnum = gender;
run; quit;
proc logistic;
        model wormnum = chris mus;
run; quit;
proc logistic;
        model wormnum = nurs prim sec;
run; quit;
proc logistic;
        model wormnum = weekly daily;
run; quit;
proc logistic;
        model wormnum = dewormed;
run; quit;
proc logistic;
        model wormnum = childnet;
run; quit;
proc logistic;
        model wormnum = pigs;
run; quit;
proc logistic;
        model wormnum = some_prim some_sec;
run; quit;
proc logistic;
        model wormnum = dds;
run; quit;
proc logistic;
        model wormnum = ses_score;
run; quit;
proc logistic;
        model wormnum = wh;
run; quit;
proc logistic;
        model wormnum = bmi;
run; quit;
proc logistic;
        model wormnum = mhtol;
run; quit;
/*unadjusted associations for continuous
variables: ANOVA for means stratified by
category, GLM for p values*/
proc sort;
        by infected;
run;
proc means;
        class infected;
        var dds ses_score wh bmi;
run;
```

```
proc glm;
       class infected;
       model dds = infected;
run;
proc glm;
       class infected;
       model ses_score = infected;
run;
proc glm;
       class infected;
       model wh = infected;
run;
proc glm;
       class infected;
       model bmi = infected;
run;
proc sort;
       by hw;
run;
proc means;
       class hw;
       var dds ses_score wh bmi;
run;
proc glm;
       class hw;
       model dds = hw;
run;
proc glm;
       class hw;
       model ses_score = hw;
run;
proc glm;
       class hw;
       model wh = hw;
run;
proc glm;
       class hw;
       model bmi = hw;
run;
proc sort;
       by al;
run;
proc means;
       class al;
       var dds ses_score wh bmi;
run;
proc glm;
       class al;
       model dds = al;
run;
proc glm;
       class al;
       model ses_score = al;
run;
proc glm;
       class al;
       model wh = al;
run;
proc glm;
       class al;
       model bmi = al;
run;
proc sort;
       by tt;
run;
proc means;
       class tt;
       var dds ses_score wh bmi;
run;
proc glm;
       class tt;
       model dds = tt;
run;
proc glm;
       class tt;
       model ses_score = tt;
run;
proc glm;
       class tt;
       model wh = tt;
run;
proc glm;
       class tt;
       model bmi = tt;
run;
proc sort;
       by infection;
run;
proc means;
       class infection;
       var dds ses_score wh bmi;
run;
proc glm;
       class infection;
       model dds = infection;
run;
proc glm;
       class infection;
       model ses_score = infection;
run;
proc glm;
       class infection;
       model wh = infection;
run;
proc glm;
       class infection;
       model bmi = infection;
run;
proc sort;
       by cure;
run;
proc means;
       class cure;
       var dds ses_score wh bmi treattime;
run;
proc glm;
       class cure;
       model dds = cure;
run;
proc glm;
       class cure;
       model ses_score = cure;
run;
proc glm;
       class cure;
       model wh = cure;
run;
proc glm;
       class cure;
       model bmi = cure;
run;
proc glm;
       class cure;
       model treattime = cure;
run;
proc sort;
       by int;
run;
proc means;
       class int;
       var dds ses_score wh bmi;
run;
```

```
proc glm;
       class int;
       model dds = int;
run;
proc glm;
       class int;
       model ses_score = int;
run;
proc glm;
       class int;
       model wh = int;
run;
proc glm;
       class int;
       model bmi = int;
run;
proc sort;
       by wormnum;
run;
proc means;
       class wormnum;
       var dds ses_score wh bmi;
run;
proc glm;
       class wormnum;
       model dds = wormnum;
run;
proc glm;
       class wormnum;
       model ses_score = wormnum;
run;
proc glm;
       class wormnum;
       model wh = wormnum;
run;
proc glm;
       class wormnum;
       model bmi = wormnum;
run;

/*logistic regression adjusted models*/
proc logistic;
       class infected;
       model infected = age gender dds
ses_score;
run; quit;
proc logistic;
       class hw;
       model hw = age gender dds ses_score;
run; quit;
proc logistic;
       class al;
       model al = age gender dds ses_score;
run; quit;
proc logistic;
       class tt;
       model tt = age gender dds ses_score;
run; quit;
proc logistic;
       class cure;
       model cure = age gender dds
ses_score;
run; quit;
proc logistic;
       class intensity (ref = '0');
       model intensity = age gender dds
ses_score;
run; quit;
proc logistic descending;
       class worm;
       model worm = age gender dds
ses_score;
run; quit;
data logregset;
       set deworm2;
       where age ne . and gender ne . and
religion ne . and schooling ne . and shoes
ne . and dewormed ne . and childnet ne . and
pigs ne . and hhed ne . and hhocc ne . and
malaria ne . and dds ne . and ses_score ne .
and wh ne . and bmi ne .;
run;
proc logistic;
       class infected;
       model infected =
              /*sixten elevpls*/
              /*bukira bukunda busowe
dwaniro kabonera kabuwoko kindulwe*/
              /*gender*/
              /*chris mus*/
              /*nurs prim sec*/
              /*daily weekly*/
              dewormed
              /*childnet*/
              pigs
              /*some_prim some_sec*/
              /*hhocc*/
              malaria
              /*dds*/
              /*ses_score*/
              wh
              bmi;
run;
proc logistic;
       class hw;
       model hw =
              /*sixten elevpls*/
              /*bukira bukunda busowe
dwaniro kabonera kabuwoko kindulwe*/
              /*gender*/
              /*chris mus*/
              /*nurs prim sec*/
              /*daily weekly*/
              /*dewormed*/
              /*childnet*/
              /*pigs*/
              /*some_prim some_sec*/
              /*hhocc*/
              malaria
              /*dds*/
              /*ses_score*/
              wh
              /*bmi*/;
run;
proc logistic;
       class al;
       model al =
              /*sixten elevpls*/
              /*bukira bukunda busowe
dwaniro kabonera kabuwoko kindulwe*/
              /*gender*/
              /*chris mus*/
              /*nurs prim sec*/
              /*daily weekly*/
              /*dewormed*/
              /*childnet*/
              /*pigs*/
              /*some_prim some_sec*/
              /*hhocc*/
              malaria
              /*dds*/
```

```
                /*ses_score*/
                wh
                /*bmi*/;
run;
proc logistic;
        class tt;
        model tt =
                /*sixten elevpls*/
                /*bukira bukunda busowe
dwaniro kabonera kabuwoko kindulwe*/
                /*gender*/
                /*chris mus*/
                /*nurs prim sec*/
                /*daily weekly*/
                /*dewormed*/
                /*childnet*/
                pigs
                /*some_prim some_sec*/
                /*hhocc*/
                /*malaria*/
                /*dds*/
                /*ses_score*/
                /*wh*/
                /*bmi*/;
run;
proc logistic;
        class cure;
        model cure =
                /*sixten elevpls*/
                /*bukira bukunda busowe
dwaniro kabonera kabuwoko kindulwe*/
                /*gender*/
                /*chris mus*/
                /*nurs prim sec*/
                /*daily weekly*/
                /*dewormed*/
                /*childnet*/
                /*pigs*/
                /*some_prim some_sec*/
                /*hhocc*/
                /*malaria*/
                /*dds*/
                ses_score
                /*wh*/
                /*bmi*/
                /*mhtol*/
                /*twow threew*/
                /*treattime*/;
run;
proc logistic;
        class intensity (ref = '0');
        model intensity =
                /*sixten elevpls*/
                /*bukira bukunda busowe
dwaniro kabonera kabuwoko kindulwe*/
                /*gender*/
                /*chris mus*/
                /*nurs prim sec*/
                /*daily weekly*/
                /*dewormed*/
                /*childnet*/
                /*pigs*/
                /*some_prim some_sec*/
                /*hhocc*/
                malaria
                /*dds*/
                ses_score
                /*wh*/
                /*bmi*/
                /*twow threew*/;
run;
proc logistic descending;
        class worm;
        model worm =
                /*sixten elevpls*/
                /*bukira bukunda busowe
dwaniro kabonera kabuwoko kindulwe*/
                /*gender*/
                /*chris mus*/
                /*nurs prim sec*/
                /*daily weekly*/
                /*dewormed*/
                childnet
                /*pigs*/
                /*some_prim some_sec*/
                /*hhocc*/
                /*malaria*/
                /*dds*/
                /*ses_score*/
                /*wh*/
                /*bmi*/
                mhtol;
run;
```

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