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Sustainable control of Ascaris lumbricoides (worms) in a rural, disease endemic and developing community: A systems approach

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Sustainable Control of *Ascaris Lumbricoides* (Worms) in a Rural, Disease Endemic and Developing Community: A Systems Approach

by

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DEDICATION

Corthia “Anne” Clarke: my sister and my best friend, when you left to be with God, I was very sad and even now I miss you very much. At the same time I am happy because “to live is Christ and to die is great gain”. You were my greatest chair leader in form and I cannot imagine how awesome you are now that you are transformed and a part of my “cloud of witnesses”. We had many dreams, and this was one of them, so I am dedicating this work to you. I promise to honor your memory by continuing on with the others. Your unconditional love for me, taught me that it was okay to be myself, thank you! I love you honey – bunny. Tell God mi sey howdie.

Monica Annmarie Gray: Monica, exactly eighteen years ago, in pure innocence, you expressed the desire to go to the highest level possible in education. It was only when you got to UWI, that you realized that there was further to go and so on you went. You have worked hard and sacrificed much, but have never really stopped to say, thank you to yourself. So, this work is also dedicated to you, Monica Annmarie Gray. What a wonderful journey! This manuscript is a testament of your ability to dream, plan and accomplish goals thru prayer, help from family, friends, and foes, and diligent work. Since that time all those years ago, you have come up with a lot more dreams, bigger dreams and I know that there will be many more. Monica, I hope that as you embark on these new dreams, you will one day look back on this document and draw strength and the necessary courage to go forward towards those endeavors. This accomplishment, while great, will pass, so continue to give birth to you. Continue to grow. Continue to dream. Continue to be you. I am so proud of you, Dr. Monica Annmarie Gray.

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I thank you, my God, my father for being: faithful, loving and kind. This manuscript is evidence, proof positive that you fulfill your promises. Thank you.

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NOTE TO READER

The original of this document contains color that is necessary for understanding the data. The original dissertation is on file with the USF library in Tampa, Florida.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

SUSTAINABLE CONTROL OF ASCARIS LUMBRICOIDES (WORMS) IN A RURAL, DISEASE ENDEMIC AND DEVELOPING COMMUNITY: A SYSTEMS APPROACH

Monica Annmarie Gray

ABSTRACT

Parasitic infections, inadequate sanitation, and poor nutrition represent major etiologies that operate in synergy to cause some of the world's most disabling diseases. Citizens of developing nations, especially children living in rural areas, are the most affected. Current research and subsequent interventions have attempted to solve these issues using vertical interventions aimed at minimizing specific health outcomes. This approach does not consider the interaction among causes and the interrelationship between human beings and their environment. Challenges solved in this manner often fail to produce sustainable results or worse, create new problems.

This project proposed the systems approach framework to address these challenges. The systems thinking dynamical modeling software, STELLA®, was used to model the conditions that promoted and/or hindered *Ascaris lumbricoides* and other gastrointestinal parasitic diseases in the rural developing community of Paquila,

Guatemala. The interventions chosen were: administration of anti – helminthic drugs, supplying protein nutrition, and an excreta management system that allowed for effluent recycling to crop production. A new design for a Solar Latrine was proposed and the solar heating and microbial deactivation processes were modeled using the commerically available, Finite Element Method software COMSOL®.

From the simulations, disease eradication was most likely to occur when at least 50% of the host population were treated every 3 months for 2 years or more with an anti – helminthic drug of 94% efficacy or better, latrine coverage and usage were at least 70%, and nutrition was provided at about 1.1 g protein per kg (human mass) per day. Given the climatic conditions in Paquila and the proposed latrine design, sustained treatement temperatures of up to 65^{o}C were possible in the fecal materail and with a minimum of 1 month (4 months maximum) retention time, it was concluded that the resulting humanure would meet US EPA Class A Biosolids microbial requirements.

1 INTRODUCTION

1.1 Problem statement

Parasitic organisms, inadequate sanitation, poor nutrition, and their synergistic interactions represent major etiologies of the world's most disabling diseases. Over half the world's population does not have access to improved sanitation (Jimenez *et al.*, 2006). Intestinal parasitic infections, which are usually associated with lack of sanitation, affect an estimated 3.5 billion people worldwide (Corrales *et al*., 2006; Santiso, 1997). The poor nutritional status of those affected increases; their susceptibility to infection, duration and degree of morbidity, and likelihood of mortality (Gendrel *et al.*, 2003). The questions this research undertakes are: given that these same challenges have been successfully dealt with in developed nations, can they be sustainably solved in a rural, disease – endemic, developing community, and if so, what will it take?

1.2 Current approach

The traditional approach to problem solving has been; isolation of each effect, determination of the dominant cause(s) and suggestion of vertical intervention programs, whose effectiveness are measured by quantifying specific health outcomes (Buchholz *et al*., 2007; Novick *et al.,* 2008). Therefore, areas endemic for the above conditions receive combinations of discipline – specific programs such as medication (Watkins *et al.*, 1996), excreta disposal (Corrales *et al.*, 2006; Pruss and Mariotti, 2000), water (Caslake *et al.*, 2004; Mcguigan *et al.*, 1998; Walker *et al.*, 2004), water and excreta disposal (Esrey *et al*., 1991), personal and domestic hygiene (Curtis and Cairncross,

2003a; Feachem, 1984), and school feeding programs (Hall, 2007; Stephenson *et al*., 2000). The community is then evaluated for any improvement in the disease outcome of interest, such as reduction in the number of worms per person or variation in diarrheal incidence over the intervention period (Muller *et al.*, 1989).

Methods of intervention and analysis used are usually not standardized across disciplines and thus, collected data can be highly unstructured and tend to lack external validity (Fewtrell *et al.*, 2005; Heller *et al.*, 2003; Varghese *et al.*, 2008). This approach has facilitated a number of innovations in individual areas such as water supply engineering, but has led to fragmentation of the public health delivery system. This outlook has persisted despite emerging evidence that problems solved in this manner often fail or worse, create new problems (Corrales *et al.*, 2006; Espinosa *et al*., 2008; Stepek *et al.,* 2006; Sterman, 2006).

1.3 Research approach

This research proposes a systems approach to solving these challenges. In this framework, the human – parasite relationship is considered the axis around which social and ecological conditions revolve to create and maintain parasite persistence (Buchholz *et al.*, 2007; Holling, 2001). Parasite endemicity is viewed therefore as a self – organizing collective behavior or emergent property of the host – parasite – environmental continuum. This by definition is a complex system (Boccara, 2004).

The systems approach recognizes the inherent nonlinearity of the interactions among system agents which is accounted for when modeling the controlling mechanisms that lead to emergence (Buchholz *et al.*, 2007; Holling, 2001). For this work, key interventions found in the literature such as improvements in sanitation and nutritional status, and mass chemotherapy are chosen and then dynamically modeled singly and concomitantly, to determine the sustainability of either approach. This

research hypothesized that, given the synergistic interaction among system variables, it will take an effective complement of interventions to sustainably resolve the issues in the system rather than the usual individual applications. This approach encourages interdisciplinary input, acknowledges that the whole is greater than the sum of its parts, provides solutions that will be more readily integrated into the community's culture, and is therefore more likely to be sustainable (Buchholz *et al.*, 2007; Coreil *et al.,* 2001).

1.4 Goals and overview

The overarching aim is to model the critical components that characterize the conditions required for the sustainable control of parasitic infections in a rural, disease – endemic, developing community typified by poor sanitation and nutrition. The project has two main goals:

- Development of STELLA® models that include combinations of the human – parasite relationship, mass chemotherapy, crop production, and human excreta management, and
- Design and then modeling in COMSOL®, of a high – rate Solar Latrine to determine the extent to which pathogens can be predictably deactivated in human excreta.

This document has seven chapters. The current chapter summarizes the motivation for considering the problem under investigation and the specific strategies that will be undertaken to develop appropriate solutions. Chapter 2 discusses the conceptual framework adopted to limit the scope of study. Details of the methodologies to be pursued within the proposed framework are advanced in Chapter 3. Chapter 4 presents the modeling of the host – parasite populations and the impact of chemotherapeutic interventions on the mean worm burden. Soybean cultivation, human excreta recycling to crop cultivation and the impact of nutrition and chemotherapy are

modeled in Chapter 5. Chapter 6 covers the design of a high – rate Solar Latrine, modeling of the inactivation process and the impact of combined chemotherapy, nutrition and latrine interventions. Finally, Chapter 7 summarizes the findings, conclusions, limitations and assumptions, and future direction for this work. This project combines the disciplines and sub – disciplines of Environmental and Agricultural Engineering, and Public Health.

2 BACKGROUND AND SCOPE

2.1 Introduction

Infectious diseases occur worldwide, however, developing countries are characterized by much higher incidence and prevalence rates (Coreil *et al.*, 2001; Esrey *et al.*, 1991). The etiologic agents are primarily transmitted via the fecal – oral route (Tinuade *et al.*, 2006). Compromised diets and inadequate sanitation, conditions that are more often than not indigenous to rural developing areas, operate individually and concomitantly to predispose community members to reoccurring infections (Thein – Hlaing and Myat Lay, 1990; Venkatachalam and Patwardhan, 1953). Over time equilibrium develops between host population and infectious agents that results in disease endemicity (Bundy and Golden, 1987).

About a hundred years ago these conditions epitomized the experiences of developed countries such as the United States (Burstrom *et al.*, 2005; Spencer *et al.*, 1967; Woldemicael, 2000). In retrospect, it was the confluence of social and ecological factors which aided and/or hindered sustainable transfer of solutions to these challenges (Curtis and Cairncross, 2003b). Similarly, for developing countries, these circumstances arise out of and are driven by concomitants of socio – economic underdevelopment and an environment that facilitate the proliferation of pathogens (Santiso, 1997; Ukoli, 1984). These generating factors present unique barriers against and opportunities for sustainable resolutions (Richmond and Peterson, 2001). It is therefore important to understand the synergistic interactions among the microbes, human hosts, and their

environments, in order to propose solutions that are economically viable, culturally sensitive and ecologically sustainable.

2.2 Epidemiological models

Epidemiological models are conceptual models that are used to represent the environmental factors that regulate and promote host – microbe interactions (Webber and Rutala, 2001). The Triangle and Wheel models for infectious diseases will be discussed here. Regardless of the form these models take, they are fundamentally based on the "chain of infection" assumption. That is, an infection is only possible if the following are in place (Oleckno, 2002):

- The pathogen has some reservoir outside the host where it can survive until it is able to come in contact with its definitive host, for example soil.
- The susceptible person is exposed to the pathogen. That is, the individual comes in contact with the microbe, such as using containers contaminated with fecal matter.
- There is some route and transport mechanism between the reservoir and the host through which the organism can enter the host, such as through the host's food supply.

2.2.1 Triangle model

This model proposes that disease occurs when there is an imbalance among host, agent and environmental factors (Oleckno, 2002). Host factors include personal traits and behaviors, genetic predispositions and immunologic differences which influence the probability for disease and degree of morbidity. Conditions external to host and pathogen that facilitate the disease process are considered an environmental factors and include physical, biological, social or combinations of these. Time delays

associated with developmental phases, incubation time and period of infectivity play very important roles in the stability of host – microbe relationship and subsequent disease endemicity within the human community. The epidemiologic triangle is illustrated below in Figure 2.1.

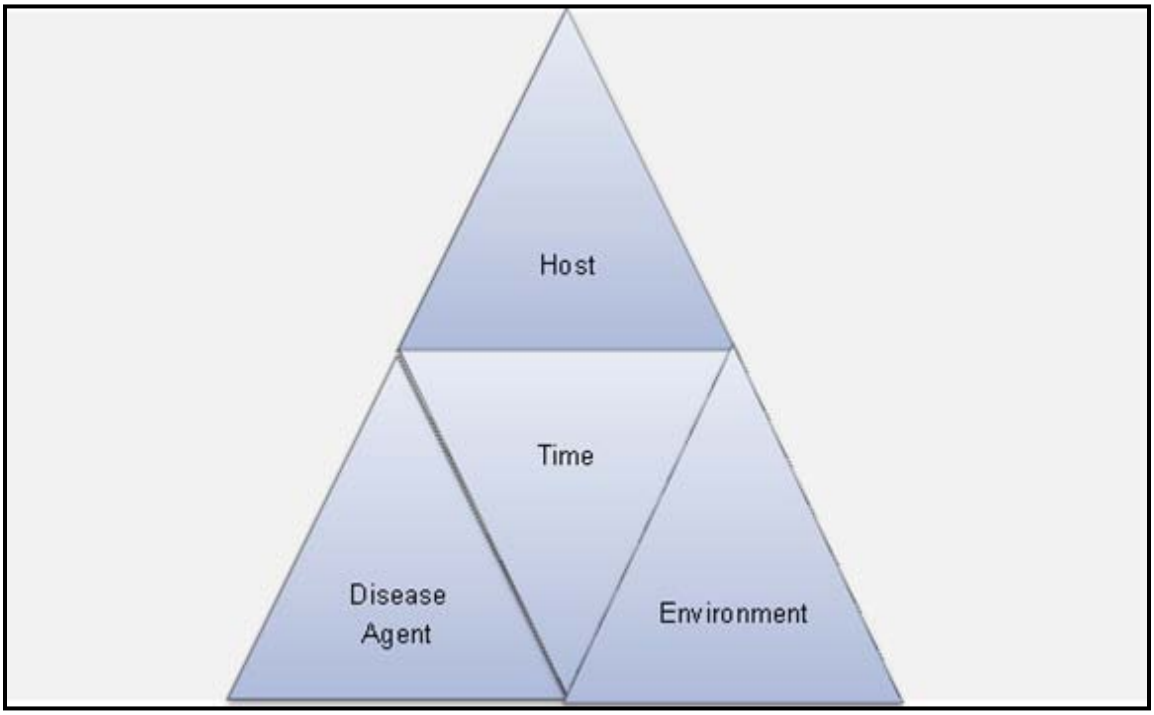

Figure 2.1: Epidemiologic triangle (adapted from Webber and Rutala (2001))

2.2.2 Wheel model

The Wheel model has an agent – host – environment paradigm similar to that of the Triangle model but these factors are conceptualized differently. The hosts with their inherent characteristics form the core across which interactions with biological (including pathogens), physical and social environments take place (Webber and Rutala, 2001). This model is adopted for this research with a minor change. This Modified Wheel model has at its core the host and the microorganism with their inherent proximate

characteristics which facilitate their dependence on and regulation of exchanges with the physical and social environment. This model is illustrated in Figure 2.2:

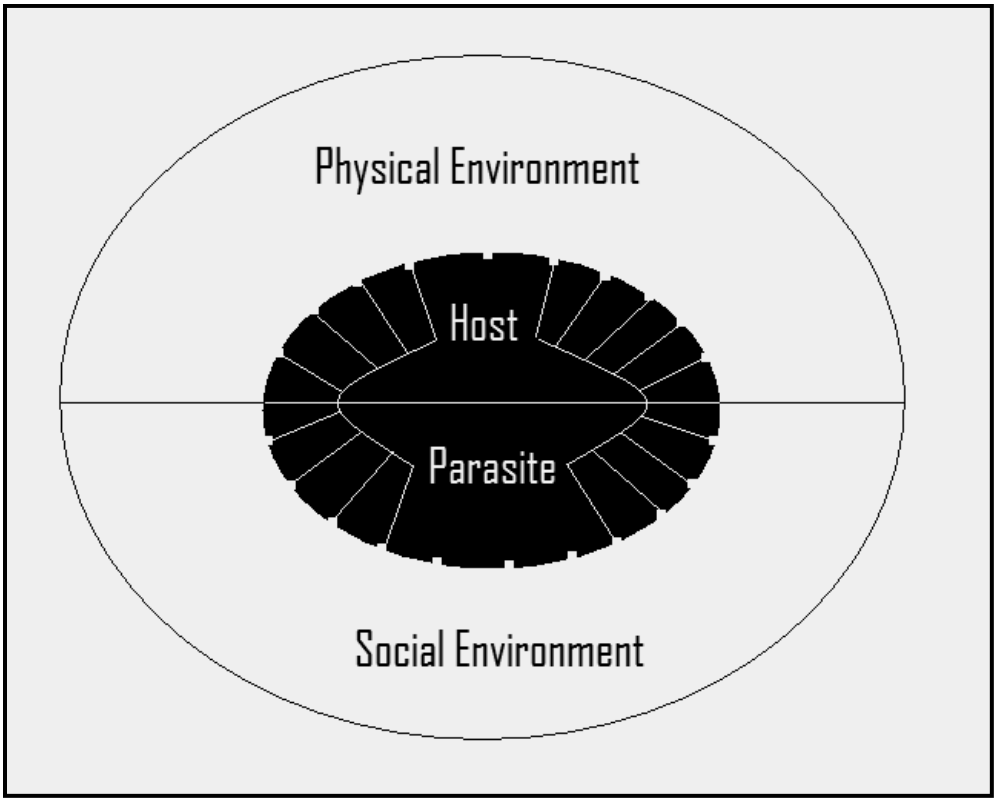

Figure 2.2: Modified wheel model (adapted from Webber and Rutala (2001))

2.3 Infectious diseases

The preceding discussion was advanced without formal definition and classification of infectious diseases, which will be addressed now. Moore (2002) defines a disease as any condition that creates harm to an individual's well – being through a distinct pathological process having characteristic signs and symptoms. In general, diseases may be classified according to the duration of the illness, the incidence and prevalence in a community, or by the causative agents (Nadakavukaren, 2000). An acute disease is of relatively short duration, the individual is likely to survive and the effects tend to be reversible, otherwise, the disease is said to be chronic (Moore, 2002). Endemic refers to the expected prevalence of a disease in a particular community

(Oleckno, 2002). If there is an unexpected outbreak among a large number of individuals, the disease is considered epidemic (Nadakavukaren, 2000). Infectious or communicable diseases occur when microbes such as bacteria, viruses and parasites are transmitted directly or indirectly among human beings and/or animals (Cairncross and Feachem, 1983). It should be noted that these categories are not mutually exclusive. Therefore, an infectious disease can be acute or chronic and endemic or epidemic within an individual or community, respectively.

There are many ways of categorizing communicable diseases. The conventional system is according to the pathogenic agents, for example, bacterial (Typhoid), viral (Dengue), protozoal (Malaria) and helminthic (Ascariasis) (Cairncross and Feachem, 1983; Heymann, 2004). Strictly speaking, protozoa (unicellular) and helminthes (multi – cellular animals) represents the two main categories of parasites (Stepek *et al.*, 2006). However, this definition is normally relaxed to include bacteria, viruses and protozoa as microparasites and helminthes as macroparasites, thereby grouping all infectious pathogens under the parasitic umbrella (Anderson and May, 1992; Santiso, 1997). A more practical approach is to classify according to the mechanism of transmission, for example fecal – oral, water – and excreta – related diseases (Cairncross and Feachem, 1983). This work focuses on infectious diseases that are transmitted via the fecal – oral route and demarcate the pathogens according to microparasites, those that cause diarrheal diseases, and macroparasites, those responsible for true parasitic infections (see Table 2.1).

Table 2.1: Classification of fecal – oral infectious diseases and associated pathogens

Categories	Infection	Pathogen
Diarrheal Diseases	Cholera	Bacteria
	E. coli diarrhea	Bacteria
	Shigellosis (bacillary dysentery)	Bacteria
	Cryptosporidiosis	Protozoa
	Giardiasis	Protozoa
	Rotavirus diarrhea	Virus
Parasitic Infections	Ascariasis	Helminthes
	Trichuriasis	Helminthes
	Hookworm	Helminthes

2.3.1 Diarrheal diseases and parasitic infections

Although all pathogens discussed above can cause diarrhea, these diseases are generally associated with microparasites (Dobson, 1988; Feachem, 1984; Gendrel *et al.*, 2003). When microparasites are ingested they simultaneously develop and multiply to produce more infective stages. Infectious diarrheal diseases tend to be acute and the etiological organisms are sometimes able to confer immunity to the host after an episode (Dobson, 1988). Macroparasites, in contrast, tend to produce chronic, asymptomatic, debilitating diseases, and usually do not similarly reward the hosts for their trouble (Stepek *et al.*, 2006). The organism develops into the adult life stage without replication (Anderson and May, 1992). The host and pathogen adapt to each in a true parasitic relationship (Markell *et al*., 1986). For both types of organisms, however, infection usually occurs when transmission stages are passed into the environment with excreta and come in contact with a susceptible host.

Globally, infective diarrhea is a leading cause of morbidity and mortality especially among children, ranking third of the top fatal childhood diseases (Curtis and Cairncross, 2003a; Nguyen *et al.*, 2006). For example, in the United States there are about 4 million diarrheal related hospitalizations annually (Heymann, 2004). Worldwide, children suffer about 1.5 billion bouts annually, with a median of 2 – 3 episodes (Kosek *et al.,* 2003; Meddings *et al*., 2004). However, children living in developing countries that are most affected, accounting for about 90% of the 3 million deaths claimed by these diseases annually (Curtis and Cairncross, 2003a; Meddings *et al.*, 2004; Tinuade *et al.*, 2006).

Parasitic infections are normally caused by metazoans (multi – cellular animals) of which the most medically important are the helminthes or worms. This group includes cestodes (tapeworms), trematodes (flukes) and nematodes (roundworms) (Moore, 2002; Stepek *et al.*, 2006). The gastrointestinal nematodes: hookworms, *Trichuris trichiura* and *Ascaris lumbricoides* are among the most prevalent and are of great public health importance (O'Lorcain and Holland, 2000; Stephenson *et al.*, 2000). There are more than one billion cases associated with each organism (Naish *et al.*, 2004). An estimated 50% of the world's population harbors at least one, with most infected with all three simultaneously, resulting in 60, 000 deaths annually (Glickman *et al.*, 1999; Smith *et al.*, 2001). These organisms are associated with intestinal blockages, cognitive impairment and malnutrition, especially anemia (Curtale *et al*., 1998; Stephenson *et al*., 2000). As is the case for microparasites, children under 5 years old in developing communities are disproportionately affected (Saldiva *et al.,* 1999).

This demarcation between diarrheal and parasitic diseases is really an academic and clinical convenience. In reality, infectious diseases usually occur simultaneously and as a result, differential diagnosis for the causative agent of over half the diarrheal cases

have not been possible (Curtis and Cairncross, 2003b). It is easy to see why this may occur. For example, during a diarrheal episode, the intestinal hurry may expel both micro and macroparasites and deciding which caused what becomes moot. To further complicate the issue, invading pathogenic protozoal/bacterial/viral agents may exacerbate helminth infections and vice versa (Boes and Helwigh, 2000). Since all forms of microbial intestinal inflammation tend to have similar pathological symptoms, the definitive etiological agent is usually underdetermined (Stephenson *et al.*, 2000).

From a public health perspective it is therefore more important to consider the transmission modality in order to prescribe sustainable interrupting intervention as opposed to trying to diagnose specific pathogens. This is the strategy adopted for this work. As discussed above, diarrheal and parasitic diseases are usually of fecal origin and the vector that mediates the transmission is excreta. Therefore, to determine if the host's living area has been contaminated by feces, environmental samples are tested for indicator organisms that are known to be exclusively associated with excreta (Droste, 1997).

2.3.2 Indicator organisms

Indicator organisms are widely used to determine the sanitary quality of environmental samples (Pachepsky *et al.*, 2006). For this research, *A. lumbricoides* was chosen to represent infectious disease organisms because it has many qualities of an ideal indicator organism, and is a better indicator organism for identifying fecal contamination than traditional total and fecal coliforms (Ishitani *et al.*, 2005; Muller *et al.*, 1989). The following is a discussion of the characteristics of an ideal indicator organism as put forward by Droste (1997) and Hazen (1988) and the ability of *A. lumbricoides* to fulfill these requirements:

"*Indicator must be present when pathogens are present and absent when pathogens are not and must originate in the digestive tract of humans only*". In general, organisms that cause diarrheal diseases and parasitic infections are almost always transmitted by the fecal – oral route (Curtis and Cairncross, 2003a; Feachem, 1984). *A. lumbricoides* can only survive to adulthood in human intestine, and is therefore exclusively associated with the pathogenic source (Crompton, 1989). Further, in areas endemic for *A. lumbricoides*, poly – parasitism is usually common (Fleming *et al.*, 2006; Quihui – Cota *et al.*, 2004; Saldiva *et al.*, 1999). The infection transmission stages, the eggs, are passed out in feces along with all other potential pathogens making it a great clinical and environmental indicator (Muller *et al*., 1989). In contrast, contemporary indicators, such as members of the coliform group, can occur in humans, animals, soils and vegetation, and thus can be present in the absence of any identifiable source of fecal pollution (Droste, 1997). In addition, these indicator bacteria may not be appropriate for the tropics, where water sources are of higher temperature and nutrient levels, conditions which promote extra – intestinal re – growth (Moe *et al.,* 1991). Therefore, the presence of *A. lumbricoides* eggs is a definite confirmation of fecal contamination.

"*The indicator should occur in high numbers and its density correlate with health hazards associated with the pollution source*". Estimates of over 10^{14} *A. lumbricoides* eggs are released into the environment daily worldwide (Anderson and May, 1985). The worm burden determines the morbidity and mortality potential of infection (Guyatt and Bundy, 1991). Fecal egg counts are indirectly correlated to the health hazard posed by *A. lumbricoides*. The number of eggs produced by the mature female is relatively constant, so assuming a 1:1 female – male ratio, the number of worms harbored by an individual can be ascertained (Hall and Holland, 2000). Once in the environment, the

eggs do not reproduce and can therefore predict the prevalence and incidence rates in a community (Muller *et al*., 1989).

"It should approach the resistance to disinfectants and environmental stress including toxic materials, of the most resistant pathogen potentially present at significant levels in the sources". That is, the indicator should survive longer than pathogens in the extra – intestinal environment. The eggs of *A. lumbricoides* are able to survive under extreme natural and treatment conditions (Arfaa, 1984). They are resistant to adverse conditions of low temperature, desiccation and strong chemicals, and can remain viable in soil for at least 7 years (Brownell and Nelson, 2006). However, high pH and temperatures, and direct sunlight are lethal (Capizzi – Banas and Schwartzbrod, 2001). It should be noted that because the method of detection does not include culturing the eggs, their inactivation does not interfere with being able to deduce fecal contamination.

"Should be easily, rapidly and reliably identified and enumerated, and analysis should be inexpensive". Definitive diagnosis is by identifying the characteristic eggs in fecal and environmental samples. The demand for mass – examination in Japan led to the development of a new stool examination procedure, the cellophane thick smear technique (Kobayashi *et al.,* 2006). This method proved to be so simple, sensitive and economical that it was standardized by the World Health Organization (WHO) (Ash *et al.*, 1994). Soil samples require a different approach and a standardized method is still being developed (Gessel *et al.*, 2004).

"The indicator should not itself be pathogenic". *A. lumbricoides* is pathogenic to human beings. However a surrogate, *Ascaris suum*, the species that infects pigs is available for use in experimental studies, since the two species are morphologically and biologically similar (Crompton *et al.*, 1989; WHO, 1967). *A. suum* is easier to obtain in large numbers (Brownell and Nelson, 2006), with experiments in pigs serving as useful

models to elucidate pathology of *A. lumbricoides* in humans (Boes and Helwigh, 2000), and shows host specificity (Anderson and May, 1985). *A. suum* serves as an excellent model because its life cycle in pigs is similar to *A. lumbricoides* in human beings, and the pig is metabolically and physiologically similar to humans as is obvious from its extensive use in biochemical research (Boes *et al.*, 1998; Carrera *et al.*, 1984).

2.4 *Ascaris lumbricoides (Ascaris)*

Each infectious agent has inherent features that determine its pathogenic success. These include its size, nutrient requirement for reproduction and development, and tolerance of environmental conditions. These factors together determine how well the organism will colonize its reservoir and/or host, the number of members required to cause illness (pathogencity) and case fatality rate or virulence of the organism.

For example, *Ascaris* is one of the most accomplished parasites and the worldwide prevalence is testament of its ability to resist insults from seasonal changes and public health interventions such as mass chemotherapy (Anderson and May, 1982). Research has shown that the longevity of the adult worm, female fecundity, the environmental resistance of the eggs and the resulting time delays in parasite production and transmission represent biological features that contribute to *Ascaris* endemicity (May and Anderson, 1978). While population processes such as nonlinearity between infection intensity and host death rates, aggregated worm distribution among community members and density – dependent constraints on parasite population growth within individual hosts interact to regulate and maintain the *Ascaris* – human relationship (Anderson and May, 1978; Crompton *et al.*, 1989). The following sections will discuss these characteristics and describe how they contribute to the organism's global notoriety.

2.4.1 Adult worm

The adult worm causes Ascariasis*,* which is the most common and prevalent intestinal nematode infection worldwide (Peng *et al.*, 2003; Sahba and Arfaa, 1967; Thein – Hlaing *et al.*, 1984). An estimated 1.5 billion persons are infected with *Ascaris,* resulting in approximately 10,000 deaths annually (Brownell and Nelson, 2006; Cooper *et al.*, 2001; de Silva *et al.*, 1997a). Humans usually contract infection by ingesting eggs containing second or third stage larvae (O'Lorcain and Holland, 2000; Peng *et al.*, 2003). Triggered by specific physiological factors like the presence of carbon dioxide and temperature of 38 ^{o}C, second stage larvae hatch in the walls of the duodenum (Clarke and Perry, 1988; Crompton, 2001). The larvae then embark on an amazing journey through multiple organs (see Figure 2.3). They first penetrate the gut wall and enter the blood circulatory system (Markell *et al.*, 1986). They reach the liver about 6 hours after infection and undergo moulting (Heymann, 2004). Within 9 – 10 days the third stage larvae arrive at the lungs where they continue to grow (O'Lorcain and Holland, 2000). About 20 days after initial ingestion, the fourth stage larvae move up the trachea and are swallowed to reenter the small intestine (Heymann, 2004). It takes about another month for juveniles to become sexually developed adults.

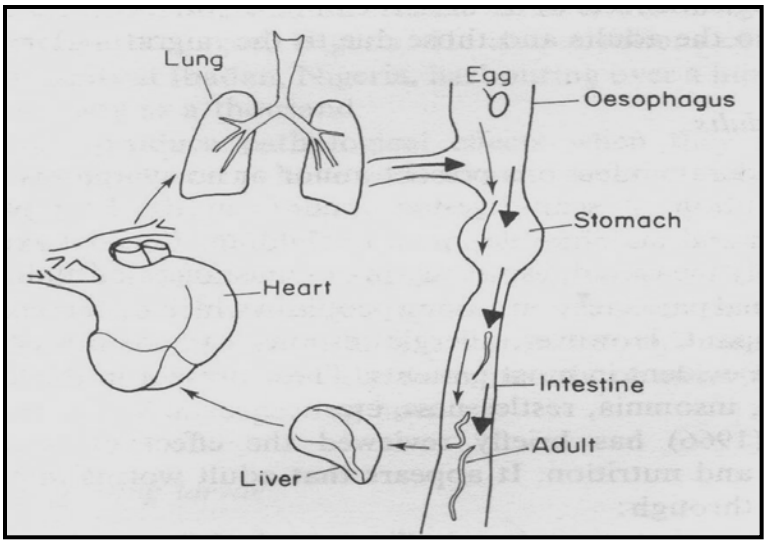

Figure 2.3: Larval migration of *Ascaris* in human beings (Ukoli, 1984)

The average lifespan of the adult worms is about a year but a maximum of 2 years is possible, which is very long for a parasite (Bethony *et al.*, 2006). The adult worms are the largest of the intestinal nematodes of humans and most closely resemble the common garden earthworms, *Lumbricus*, after which they are named (Markell *et al.*, 1986). The mature females occasionally reach 49 cm in length while the males are seldom over 30 cm (Brown and Cort, 1927). The very high fecundity of the female worm is attributable to its large size (Hall and Holland, 2000). A gravid female worm have been purported to be able to lay up to 200,000 eggs per day (Arfaa, 1984)! Thus, the long lifespan and high egg production rate maintain a continuously high supply of the infective stages in the environment and subsequently increase the risk of infections to susceptible host.

Ascaris is dioecious and polygamous, that is, both sexes are required to produce embryonated (fertilized eggs that can develop to become infective) and males mate with multiple females respectively (Croll *et al.*, 1982). As a result, an infected person may produce unfertilized and fertilized eggs or a mixture of both depending on mating activities of the worm population inside a given host (Peng *et al.*, 2003). The mating

probability is a function of the worm density and is very high for *Ascaris* because the number of worms in the host population is not normally distributed but tends to cluster, with the majority being harbored by a small number of persons (Boes *et al.*, 1998; O'Lorcain and Holland, 2000). There are two major reasons for this phenomenon. These are: differences in human behavior such as eating and personal hygiene habits, and the heterogeneity in the spatial distribution of the infective eggs (Anderson, 1982). This distribution of worm numbers among hosts ensures that there is always a portion of the human population producing fertilized eggs (Schmid and Robinson, 1972; Schulz and Kroeger, 1992). Also, for those hosts with light and moderate infections, the worms' survival and fecundity are not reduced by density – dependent host immunological responses as with the case of heavy infestations (Anderson and May, 1982). Thus, maximum egg production and worm life expectancy rates are possible at lower worm burdens.

During larval migration some hosts may develop pneumonitis and asthmatic attacks (Markell *et al.*, 1986). In general, *Ascariasis* is clinically symptomless, but becomes less so as the intensity, number of worms per host increases (Komiya and Yanagisa, 1964; Margolis *et al.*, 1982; Sahba and Arfaa, 1967). Light infections of worm density less than 20 worms per host usually present minor symptoms unless adult worms undergo uncharacteristic migration to pancreas, bile ducts, gallbladder or liver (Crompton, 1989; Hall and Holland, 2000). Children experience temporary growth retardation, which is completely reversible upon treatment and improved nutrition (de Silva *et al.*, 1997a). Heavy infections of worm burdens greater than 40 worms per host are likely to cause death (Hall and Holland, 2000; Thein – Hlaing *et al.*, 1987). Intestinal obstruction (see Figure 2.4) is the most common of the severe complications associated

with high worm burdens and usually results in death especially in children (de Silva *et al.*, 1997b).

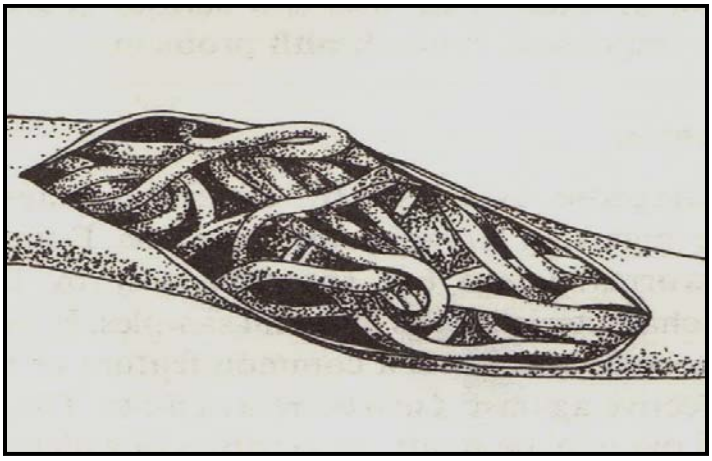

Figure 2.4: *Ascaris* blocking small intestine (Ukoli, 1984)

The actual worm burden cannot be ascertained without anthelminthic treatment, therefore fecal egg concentration is the typical surrogate (Hall and Holland, 2000). Egg counts give an indirect measure of the intensity of infection and are expressed as eggs per gram of feces (epg). It is assumed that the greater the epg the higher the density (number of worms per unit volume of organ) of sexually mature female worms in the intestine (Margolis *et al.*, 1982; O'Lorcain and Holland, 2000). Light infections are defined by less than 5000 epg, while greater 50,000 epg constitutes heavy worm burden (WHO, 1967).

2.4.2 *Ascaris'* eggs

Ascaris' eggs are typical of those of the phylum *Nematoda*. One of the features responsible for the success of *Ascaris* and other nematodes is the structure and chemical composition of the egg shell that makes it resistant to harsh environmental conditions (see Figure 2.5). The main function of the shell is to maintain a homeostatic environment for the developing embryo and protect it from adverse environmental conditions as it passes from the host (Wharton, 1983). The three inner fundamental

layers are formed from secretion by a fertilized oocyte (egg produced after female mates) (Wharton, 1980a). These include an inner lipid layer (ascaroside layer), a middle chitinous layer and an outer vitelline layer (Bartley *et al.*, 1996; Wharton, 1980a). *Ascaris* possesses an additional outer layer, a sort of "final finish" that the female adds to the eggs as they leave her uterus (Foor, 1967).

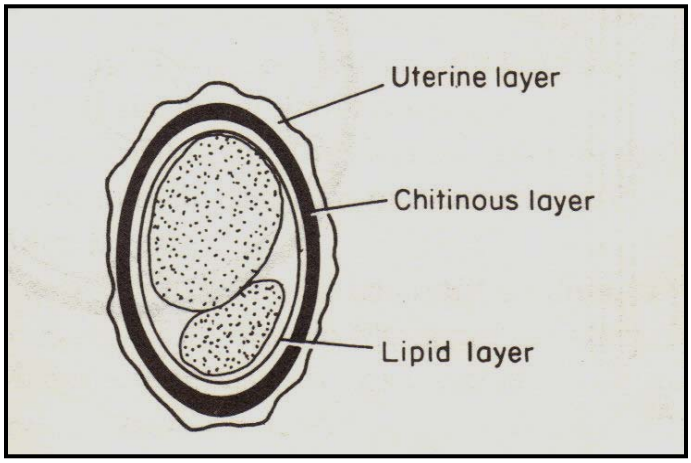

Figure 2.5: *Ascaris*' egg showing the basic layers of the shell (Ukoli, 1984)

2.4.2.1 Ascaroside (lipid) layer

The chemical composition of the lipid layer in *Ascaris* is very unique: 75% of a special class of fats called ascarosides and 25% protein (Brownell and Nelson, 2006; Wharton, 1980a). It immediately surrounds the embryo and is most responsible for the eggs' thermal resistance (ascarosides have high melting point of 82 ^{o}C) and relative impermeability to toxic substances (Bird and Mcclure, 1976; Ukoli, 1984). This layer is permeable to oxygen (developing egg is an obligate aerobe), organic solvents, and small amounts of water vapor, but is hydrophobic (Clarke and Perry, 1980; Passey and Fairbairn, 1955). The permeability of the ascaroside layer varies. For example, there is an increase in permeability during external incubation between 44 – 65 ^{o}C and hatching in the host's alimentary tract (Barrett, 1976).

2.4.2.2 Chitinous layer

The chitinous layer is the thickest layer of the shell and is an engineering wonder. It consists of a series high tensile strength chitin microfibers dispersed in a protein coat that is able to withstand deforming forces (Wharton, 1983). The fibers are orientated at random, with the resulting arrangement resembling interconnecting ridges (like roof trusses) which provide structural strength to the eggs and protect it against mechanical damage.

2.4.2.3 Vitelline layer

This layer consists of lipoprotein (fat – protein) similar to the lip layer. This layer is usually thin but may become thickened as the egg becomes fully formed and is not usually visible under a light microscope (Ukoli, 1984). It is permeable to organic solvent and melts at approximately 70 ^{o}C (Fairbairn, 1957).

2.4.2.4 Uterine layer

The uterine layer is composed of glycoprotein which is progressively stabilized by a quinine – tanning process, analogous to cuticle hardening in insects, as the egg leaves the host (Clarke and Perry, 1988). For example, if the eggs are taken prematurely from the uterus before this final “spit shine” the egg – shell is colorless and soluble in acids, alkalis and various enzymes and does not completely embryonate in direct sunlight (Fairbairn, 1957). However, when they are fully developed and are passed out in feces, the eggs are brown and insoluble in all reagents except sodium hypochlorite (Wharton, 1983). It has been hypothesized that the development of color that occurs during embryo formation (development of second stage larva) protects the egg from the harmful effects of ultraviolet, which coincidentally is the most resistant phase of the life cycle (Black *et al.*, 1982; Fairbairn, 1957).

2.4.2.5 *Ascaris'* egg structure and its persistence

The *Ascaris* shell is able to slow, but not completely prevent water vapor loss. The water loss rate is dependent on the surrounding relative humidity and temperature (Wharton, 1979). After exposure to above 60 – 65 ^{o}C for 3 days the ability of the egg shell to slow down the rate of water loss disappears and the egg collapses as a result of desiccation (Wharton, 1980b). Oxygen consumption and water loss is higher at higher temperatures, which corresponds to the higher developmental rate (Brown, 1928; Wharton, 1980a). Embryonated (infective) eggs can withstand desiccation better than unembryonated since they consume oxygen more slowly (Brown and Cort, 1927).

The infective eggs are dormant and can survive in the soil for several years (Barrett, 1976; Komiya and Kobayashi, 1965). In addition, due to the average relative lifespans of the egg, worm and human populations; 2 – 6 weeks, 1 year and 69 years respectively, the infective stages are assumed to always be in steady state (May and Anderson, 1978). Therefore, high egg output, over a relatively long reproduction time, coupled with potentially high survival rates of infective stages provides a continuous stream of opportunity for disease transmission and maintenance in the host's community.

2.5 Human – *Ascaris* population dynamics

Proximate factors are those hosts' characteristics that influence the level of exposure to pathogenic organisms, susceptibility to infection, morbidity of the resulting disease and subsequent health outcome (Webber and Rutala, 2001). For instance, research shows that children under 15 years old and certain families tended to reacquire pre – control worm intensities after chemotherapy stops (Crompton, 1989; Thein –

Hlaing *et al.*, 1987). The following sections will discuss the interrelationship between age, gender and ethnicity, and an individual's predisposition to infection.

2.5.1 Age

The host's age is an important determining factor for disease prevalence and intensity because of its association with exposure rates and ability to resist infection. For example, the prevalence of *Ascaris* infection normally increase rapidly during early childhood to as much as 92% among school aged children up to 15 years old but tapers to about 65% for adults in endemic areas (Croll *et al.*, 1982; O'Lorcain and Holland, 2000; Thein – Hlaing *et al.*, 1984). However, in hyper – endemic areas 100% prevalence rates in adult age classes are not uncommon (Anderson, 1980b; Young *et al.*, 2007). The trend for the variation in the number of worms per person is not so easy to describe since intensity is a function of the host's physiology and density – dependent constraints. That is, children because of their small gut size are not physically able to host as many worms as their adult counterparts (de Silva *et al.*, 1997b). Also, as the number of worms increase, competition of increasingly scarce resources hinders worm growth and establishment (Bottomley *et al.*, 2007).

Homes with small children are more likely to have yards contaminated with fecal matter (Schulz and Kroeger, 1992). The eggs of helminthes tend to follow an aggregated distribution, with high concentrations close to residences and around latrines where children tend to frequent and are therefore more exposed to the infective stages (Muller *et al.*, 1989; Schulz and Kroeger, 1992; Thein – Hlaing *et al.*, 1984). In addition, while *Ascaris* infection does not impart lasting immunity to the host, it has been hypothesized that exposure to repeated infection during early life may induce some level of protection to adults (O'Lorcain and Holland, 2000). This may account in part for the relatively low infection intensities found in adult members of endemic communities. Finally, age –

related differences in incidence and prevalence may be due to changes in the pattern contact with infectious stages due to changes in roles and responsibilities as children grow older (Okyay *et al.*, 2004).

2.5.2 Gender

Females generally have higher disease prevalence rates than males (Crompton, 1988). These may represent differences in exposure rates that arise due to culturally – defined roles. For example a female who has to handle children's feces on a regular basis may be more exposed to much higher concentrations of microparasites than her male counterpart that works outside the home. On the other hand, the male may be more exposed to soil – transmitted helminthes such as *Ascaris* if he works in fields fertilized with night – soil (Curtale *et al.*, 1998). However, these results can be confounded by age and cultural factors. For example, in an area where pica (habit of eating soil) is practiced, boys ages 1 – 5 tended to have higher prevalence rates, while female rates are higher within the 11 – 18 age groups (Glickman *et al.*, 1999).

2.5.3 Ethnicity

Infectious disease incidence is normally higher among certain ethnic groups (Kightlinger *et al.*, 1998)., It has been found, however that ethnicity in these cases is a proxy for socio – economic status, which is a more valid explanation for the observed differences (Coreil *et al.*, 2001). It is possible that cultural behaviors as well as genetic differences may also create heterogeneity which causes a particular group of persons to be more susceptible to an infectious agent or enhance the pathogencity and virulence of the organism.

2.6 Physical environment

The physical environment plays an important role in the promotion and establishment of diseases (Stephenson *et al.*, 2000). *Ascaris*, as well as other infectious diseases pathogens, are usually endemic in areas that have inadequate excreta disposal, low quality water supply, poor housing, and moist and warm climates (Crompton *et al.*, 1985; Santiso, 1997). For this work, these factors will be classified into two groups, namely, the natural and built environments. The natural environment is defined in the usual sense and comprises the geographical location of the community, and its resulting climate and ecology. The built environment consists of the type of housing and the sanitation infrastructure available to the community.

2.6.1 Geographical location

The energy from the sun modifies, controls and determines the climate of an area (Moore, 2002). Most developing countries are geographically located in the tropics between latitudes 35 ^{o}N and 35 ^{o}S and consequently receive the greatest amounts of solar insolation (Eggers – Lura, 1979). These regions are usually warm and humid, conditions that shorten the developmental cycles of plants and animals (Santiso, 1997). As a result, over 40% of the world's plants and animals make the tropics their home (Nadakavukaren, 2000). Thus, while parasites can be found everywhere in the world, they are most abundant and persistent in these communities (Stromberg, 1997). For example, low prevalence rates are normally reported in countries with drier climates, since the infective stage requires a high relative humidity to survive (Crompton, 1988).

Annual seasonal variations can influence the intensity of disease transmission (Thein – Hlaing *et al.*, 1984). For example, contamination of yard soil was found to be higher during the rainy season than during the dry seasons (Schulz and Kroeger, 1992).

In addition, changing weather conditions determine planting and harvesting seasons, consequently increasing the contact rate between community members and infective stages and resulting in very high parasite transmission (Gunawardena *et al.*, 2004). Recycling night – soil to crops has been shown to be a major source of gastrointestinal infections, therefore, peak prevalence rates have been observed to coincide with crop cycles (Kobayashi *et al.*, 2006). For parasites with lifespans greater than a year, as is the case for *Ascaris*, these patterns do not significantly affect their net stability (Thein – Hlaing *et al.*, 1984). Seasonal factors are therefore more relevant in determining when the reproduction and transmission rates are at their lowest in order to maximize the outcomes of control measures.

2.6.2 Housing

Generally, the poorer the quality of housing and community services, the more likely infectious diseases will persist resulting in higher prevalence rates (O'Lorcain and Holland, 2000; Webber and Rutala, 2001). The risk of mortality is 58% lower among children born in households with a good environment than among those born to lower quality housing conditions, even after controlling for socioeconomic variables (Woldemicael, 2000). Similar statistics were observed for overcrowding (Schulz and Kroeger, 1992). Dirt floors can be excellent transmission loci especially for soil – transmitted helminthes (Grimason *et al.*, 2000).

2.6.3 Water supply

The water supply diffusion rate (percentage of population serviced by a potable water supply system) is usually very slow for developing communities (Ishitani *et al.*, 2005). Contact with contaminated water results in up to 60 billion episodes of gastrointestinal illness annually most of whom are under age five (Caslake *et al.*, 2004;

Curtis and Cairncross, 2003a; Walker *et al.*, 2004). Children from households that use water from rivers and lakes are 44% more likely to die from diarrheal diseases than their counterparts who have access to piped supplies, even after controlling for demographic and socio – economic factors (Woldemicael, 2000).

The literature is very conflicting on the benefits and health outcomes from water supply interventions (Fewtrell *et al.*, 2005; Gasana *et al.*, 2002; Huttly *et al.*, 1997). For instance, Fewtrell *et al.* (2005) reported that increasing the amount of water, irrespective of purity has been shown to improve health. In areas where environmental fecal contamination is high, water supply improvements no matter how high the quality offer very little health impact (Esrey *et al.*, 1991). Thus, while it seemed intuitive that providing water of high quality and quantity should correct these insults, this is generally not the case.

2.6.4 Excreta disposal

Promiscuous defecation by children and unhygienic disposal of their feces by adults play a more important role in determining childhood growth, morbidity and mortality, than does water quality, especially where the prevalence of diarrhea is high (Esrey *et al.*, 1991; Jinadu *et al.*, 2004; Schulz and Kroeger, 1992). For example, a child born to a household without toilet facility is at 64% more risk of dying from parasitic diseases than one with such amenities (Woldemicael, 2000). The type of disposal facility was found to be important, with flush toilets having a greater impact on mortality reduction than pit latrines (Esrey *et al.*, 1991). For developing countries however, the required physical infrastructure and water resources needed for contemporary “flush” toilets are generally nonexistent or insufficient to meet the demands of the rapidly growing populations, rendering their application unsustainable (Langergraber and Muellegger, 2005). For example, Schulz and Kroeger (1992) found that if sewage

system were inadequate, homes with “flush” toilets had yards that were equally contaminated with *Ascaris*’ eggs as those with latrine systems. The lack of proper disposal systems can therefore lead to groundwater contamination, resulting in further infections (Gannon *et al.*, 1991).

As a result, only 67% of the population of developing countries have adequate facilities for excreta disposal (Palamuleni, 2002). There is however a drive to provide latrines in response to the Millennium Development Goals (Waterkeyn and Cairncross, 2005). Since the eggs of soil – transmitted helminthes are not immediately infective, any kind of latrine that helps to avoid fecal contamination of the floor, yard, or fields will limit transmission, however, hygiene practices are very important (Muller *et al.*, 1989). For example, if an earth floor latrine is poorly maintained, it can become a focal point for disease transmission (Grimason *et al.*, 2000). Dirty latrines may result in higher disease incidence than would occur if people were practicing widely scattered open defecatation (Cairncross and Feachem, 1983).

2.7 Social environment

The human hosts, their behavioral and cultural practices represent the social environment. These are intermediate and distal factors that cause community members to be exposed to or protected from infection but do not influence disease occurrence directly (Coreil *et al.*, 2001). These include host density, individual health behaviors (hygiene practices, preexisting conditions, diet and nutrition), and socio – economic status.

2.7.1 Population

The population of developing countries has been increasing steadily and is expected to account for more than 95% global projected growth over the next 1 – 2

decade (Moore, 2002). While there is an exodus from rural to urban areas, it is the former that will account for the bulk of this growth (Kosek *et al.*, 2003). The subsequent overcrowding can lead to conditions favorable for the efficient transmission of pathogens, resulting in higher intensity infections among households with more members (O'Lorcain and Holland, 2000).

2.7.2 Hygiene

From the above discussions, it can be concluded that it is not enough to construct affordable latrines and provide clean water, but hygiene education interventions is also essential for success. Traditionally hygiene interventions are typically of two types, those focusing on health and hygiene education, and those promoting hand washing with soap and water (Fewtrell *et al.*, 2005; Jinadu *et al.*, 2004). Human activities are not always in their best interest. For example cultural beliefs that consider fecal matter from children to be innocuous can cause community members to be nonchalant during handling, which can lead to higher infection risks especially in areas where diarrheal diseases are prevalent (Yeager *et al.*, 1999). Therefore behavioral interventions are crucial to the success of control programs (Webber and Rutala, 2001).

2.7.3 Preexisting infections and polyparasitism

Conditions that encourage *Ascaris* endemicity also support many other gastrointestinal parasites. Thus, where diarrheal diseases are endemic, polyparasitism is usually also common (Keiser and Utzinger, 2008).

2.7.4 Diet and nutrition

Specific dietary habits can increase the host's risk for infection or be protective against disease. For example, in areas where geophagia (soil – eating) is culturally

practiced, participants are at higher risk of ingesting soil – dwelling pathogenic organisms and a normally found to have infection intensities above community average (Geissler *et al.*, 1998; Glickman *et al.*, 1999; Young *et al.*, 2007). Eating uncooked fruits and vegetables that have been fertilized with human excreta may also lead to higher disease incidence (Feachem *et al.*, 1983).

One of the most important factors that determines the magnitude of morbidity and likelihood of mortality from infectious diseases in endemic areas is the nutritional status of the host (Boes and Helwigh, 2000). Under nutrition at any age can compromise the host defense systems (Stephenson *et al.*, 2000). However, young children and pregnant women are particularly vulnerable because of their inherently high nutritional demand (Bundy and Golden, 1987). Further, the additional metabolic requirements from the pathogens put them in less favorable health conditions to resist other insults (Bundy and Golden, 1987).

One third of young children in developing countries experience linear growth retardation or stunting in early childhood as a result of chronic undernutrition (Morgan, 2005; Saldiva *et al.*, 1999). *Ascariasis* and diarrhea are known to play a major role in the etiology of childhood malnutrition (O'Lorcain and Holland, 2000). This is because nutritional, especially protein – energy, deficiencies often cause suppression of immune – response, which can lead to unrestrained establishment and increased survival of parasites (Gendrel *et al.*, 2003; Stephenson *et al.*, 2000). For example children with average burden of 26 worms were reported to have lost about 4 g of protein daily intake due to parasitic interference with the digestive process (Stephenson *et al.*, 2000; WHO, 1967). Periodic deworming of *Ascaris* – infected pre – school children have been shown to improved growth in areas where protein – energy malnutrition is common (Stephenson, 1980).

Other nutrients of special importance include fat, carbohydrate, vitamin A and iron (Stephenson, 1980). Fecal fat excretion has been shown to decrease after deworming (Macinko *et al.*, 2006). The typical diet in developing countries derives 75% of total calorie intake from carbohydrates and so any interference with absorption can have serious consequences (Carrera *et al.*, 1984). Reduced absorption of vitamin A have been associated with protein deficiencies (Stephenson, 1980; Woodruff and Wright, 1984).

However, the malnutrition – infection interaction is not confined to a linear, one – way causal relationship. That is, nutritional deficiencies tend to promote and intensify infections as well as infections may promote nutritional imbalances due to increased energy requirements to fight them (Boes and Helwigh, 2000). On the other hand, as the host becomes more malnourished, worm burden and fecundity may be reduced as nutrients become increasingly unavailable (Bundy and Golden, 1987). Parasitic infections can and often do cause decreased food intake (Saldiva *et al.*, 1999). Thus, infectious diseases may affect nutritional status as well as pre – existing nutritional status may increase the risk of and/or exacerbate illness (Stephenson *et al.*, 2000).

2.7.5 Socio – economic status

Socio – economic factors represent the availability of resources that promote life, health and wellbeing. These include but are not limited to, household and community economic status, type of residences and physical infrastructure, health care availability, mother's education, and political stability (Woldemicael, 2000). The social capacity of the community is also important and includes the ability of members to come together and solve common challenges (Coreil *et al.*, 2001).

Throughout history and in nearly every country, the poor has been identified as the population most at risk for adverse health outcomes (Morgan, 2005). There is usually

a culture of entrepreneurship embedded in the social heritage of peoples of developing countries (Brentlinger *et al.*, 2007; Ukoli, 1984). According to the Global Entrepreneurship Monitoring (GEM) report, which measures what fraction of a country's adult population that has attempted or started a business, developing nations usually have the highest numbers. The rationale proposed has been that individuals are usually forced to seek their own employment because of high unemployment rates or the contradiction between industry requirements and cultural outlook (Johansson, 2008).

Control programs, where they have been mounted, have underestimated the socio – cultural and human behavioral factors which play a part in enhancing transmission of infection (Brentlinger *et al.*, 2007). In addition they have underutilized an important resource that is virtually a staple in developing communities, that is, social capacity (Coreil *et al.*, 2001). Social networks are usually extensive and are reminiscent of small towns in developed countries.

2.8 Proposing sustainable solutions

An individual's health status is a dynamic equilibrium among host factors, characteristics of the infectious agent, and environmental influences occurring over time (Webber and Rutala, 2001). Parasitic diseases are prevalent in the tropics because of the combined effects of ecological and climatic factors, dietary and sanitation constraints, human behavioral and cultural practices, population density, and socio – economic conditions. The warm and humid climates of these areas facilitate faster development and proliferation of large numbers infectious agents (Ukoli, 1984). The climatic conditions also encourage human behavior that increases contact between infectious stages and susceptible individuals. *Ascaris* was chosen to represent these pathogens because it has several characteristics of an ideal indicator organism (Muller *et al.*, 1989; Schulz and Kroeger, 1992).

Ascaris, as well as other infectious disease pathogens are usually endemic in areas of developing countries that have high population densities and low socio – economic status (Crompton *et al.*, 1985; Santiso, 1997). However, population density impacts disease occurrence more on a community rather than a national level and is usually surrogated by low socio – economic status. This is evidence by the fact that some of the richest countries have the highest population density without the associated infectious disease endemicity (Johansson, 2008). The economic wealth that fulfills the physical needs of the community is a protective factor against disease transmission, however, socio – cultural practices can have more influence on the occurrence and spread of parasitic diseases (Ukoli, 1984).

Poor nutrition is known to interfere with the ability of children to benefit from educational programs which can lead to other socio – economic status issues and is a major cause of morbidity and death (United Nations, 1991). Controlling any enteric parasite means dealing with at least two populations, the pathogen infesting the host and the infective stages in the environment. Providing nutritional supplement and mass chemotherapy may help to decrease morbidity and mortality rates within the host population but it does nothing to stop the transmission stages.

There are disagreements in the literature about the benefits of sanitation interventions, similar to those of the results of water improvement studies. In fact, a number of researches have evidenced the failure of improved safe water supply and excreta disposal to sustainably combat infectious diseases (Schulz and Kroeger, 1992). That is, improvements in sanitation facilities may significantly reduce prevalence of infection, however, morbidity problems may linger (Asaolu *et al.*, 2002). The threshold – saturation theory (see Figure 2.6) has been used to explain this counterintuitive finding (Shuval *et al.*, 1981). The theory states that in communities with very low socio –

economic status, the health of members will not respond to any improvements in the sanitation infrastructure, resulting in an initial lag phase or threshold. The rationale is that there are so many transmission routes for disease and the personal hygiene and nutritional status of members are so low that these interventions will not succeed in eliminating enough to have a significant impact. As individuals' and community's socio – economic status increases the community is able to respond to improvements in the physical environment, but at some point further improvements show diminishing return on investment (Asaolu *et al.*, 2002).

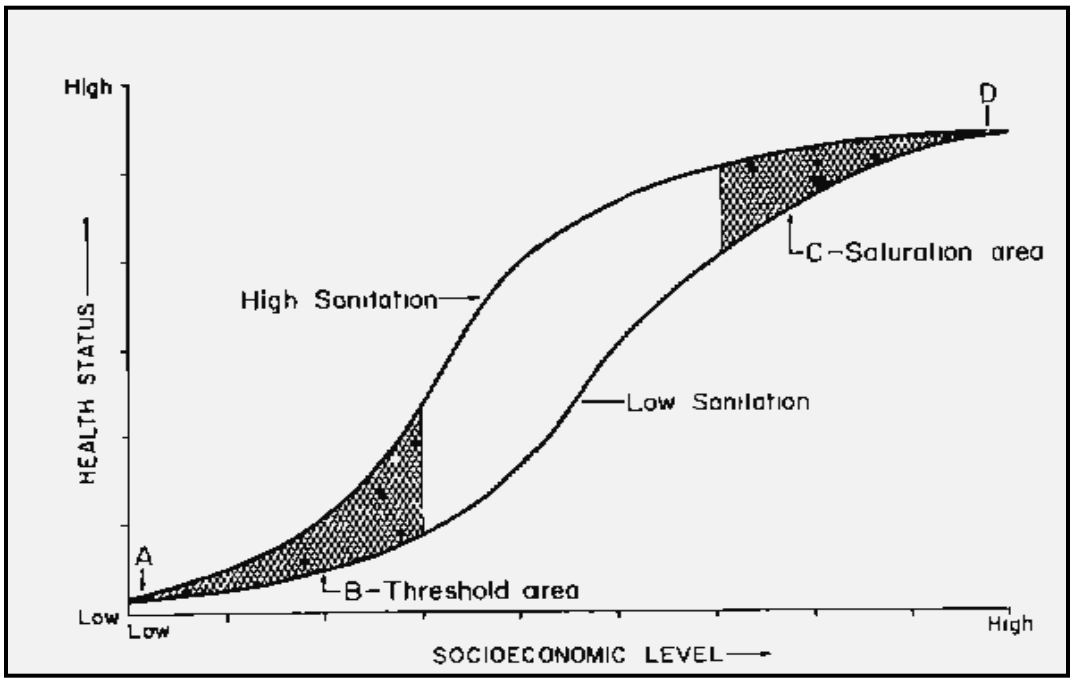

Figure 2.6: Threshold – saturation theory (Shuval *et al.*, 1981)

In addition to the probability of failure to decrease disease morbidity and mortality, simply providing latrines or drilling wells does not increase the social or economic capital of the community. That is, even if members help in construction, the process does not strengthen the social structure and encourage the community to solve its own problem. A simple latrine does not make use of a valuable resource that can be

recycled to crop production. With the high economic and ecological cost of chemical fertilizers, recycling excreta will in a single move, improve both nutritional and fiscal status. This type of integrated approach has been shown to work (Brentlinger *et al.*, 2007; Checkley *et al.*, 2004; Jensen *et al.*, 2005; Meddings *et al.*, 2004; Root, 2001; Shuval *et al.*, 1981; WHO, 2002). For example, in malaria eradication programs it has been found that bed net programs are more sustainable when distribution is coordinated through local shopkeepers (Brentlinger *et al.*, 2007; Goodman *et al.*, 2007). Motivated by a business opportunity, shopkeepers were encouraged to keep up supply, thus health promotion was channeled through a social structure that was already well integrated into the local community (Foster, 1991; Goodman *et al.*, 2006).

The synergistic interactions among the factors discussed above imply that interventions targeting any one social service are likely to be wasted unless comprehensive and coordinated actions are undertaken. In addition, education and training programs are also essential in improving nutritional practices, especially in instruction of low – income women on the value of breast – feeding and on the preparation of balanced and uncontaminated food for infants and children (United Nations, 1991).

2.9 Summary and conclusions

History has shown that parasitic diseases, inadequate sanitation and poor nutrition with their associated morbidity and mortality can be resolved. The question that remains therefore is whether sustainable solutions can be found for these challenges in a rural and developing community setting. The Modified Wheel epidemiological model was employed as a framework to elucidate the controlling mechanisms in the host – parasite relationship that lead to endemicity and the key interventions found in the

literature that have been shown to have some measure of success in controlling adverse effects.

The overarching goal of this dissertation is to propose economically viable, culturally sensitive and ecologically sustainable solutions for controlling fecal – oral transmitted infectious diseases in a rural and developing community. By definition, sustainability is development that efficiently utilizes present resources to fulfill current needs, while facilitating the ability of future generations to meet their own needs (Wright, 2002). An implicit deduction is that for every challenge there are available resources and, if wisely applied, such solutions can be integrated within the social fabric of a community, such that future generations will be able to independently maintain them.

To satisfy these criteria, disease control must be integrated with other aspects of land use and development, improvement in agricultural practice, and education. That is, a broad – spectrum resource improvement program which will generate the capacity in the people to seek solutions to future problems. This research is proposing a systems approach that will establish links among the various aspects of ecology, engineering and agriculture, human behavior, education and culture for sustainably breaking the host – parasite – environment continuum.

3 METHODOLOGY

3.1 Background

There are at least twenty species of pathogenic microorganisms that are found exclusively in the human intestines and are passed out with feces to contaminate the environment to cause diarrhea and parasitic infections in others or the host (Curtis and Cairncross, 2003a). These microbes have a variety of developmental and transmission stages, but all have similar biological characteristics that determine the persistence of their relationship with the host. *Ascaris* plays dual roles of clinical as well as environmental indicator organism (Muller *et al.*, 1989). Medically, the presence of eggs in fecal samples is indicative of an established worm population (Peng *et al.*, 2003). In addition, because *Ascaris* tend to occur simultaneously with other infectious agents, its presence may point to poly – parasitism (Fleming *et al.*, 2006). Eggs found in environmental samples such as yard soil definitively verify fecal contamination (Uga *et al.*, 1995)

While the mode of transmission (eggs, larvae or arthropod vector), life cycle (direct versus indirect), and propagation (cyclo – developmental or cyclo – propagative) for *Ascaris* do not mirror exactly what occurs with all gastrointestinal infectious disease pathogens, the conditions under which these organisms and their transmission stages exist and flourish, and their routes of infection are similar (Curtis and Cairncross, 2003a). Thus, a fundamental assumption of this research project is that creating the conditions that sustainably control *Ascaris* will in effect facilitate the suppression of other infectious diseases. This is in part due to the fact that compared to parasitic infections caused by

viruses and bacteria, *Ascaris* is very resistant to control strategies (Anderson and May, 1982).

Research has shown that the stability of any microbial population depends on the life cycle stages that is most affected by density – constraints, analogous to a rate determining step in a chemical reaction (Churcher *et al.*, 2006). The first step towards proposing sustainable solutions to the challenges described in Chapter 1 is therefore to detail the life cycle of *Ascaris.*

Ascaris epitomizes a macroparasite with a direct life cycle (see Figure 3.1) (Crompton, 2001). That is, the organism does not use an intermediate host in its developmental cycle (Heymann, 2004). Their eggs undergo obligatory development in the soil and are therefore referred to as soil – transmitted helminthes (Cairncross and Feachem, 1983; Curtale *et al.*, 1998). While in the soil, fertilized eggs moult to second stage larva, which is the infective stage (Brown, 1928). This process takes about 2 – 4 weeks depending on the environmental conditions such as temperature, moisture and solar insolation (Croll *et al.*, 1982). When infective eggs are ingested, they hatch and develop while journeying through the body as described in Section 2.4.1, a process that takes about 2 months (Murrell *et al.*, 1997). The sexually mature worms mate and consequently produce eggs that pass out into the environment.

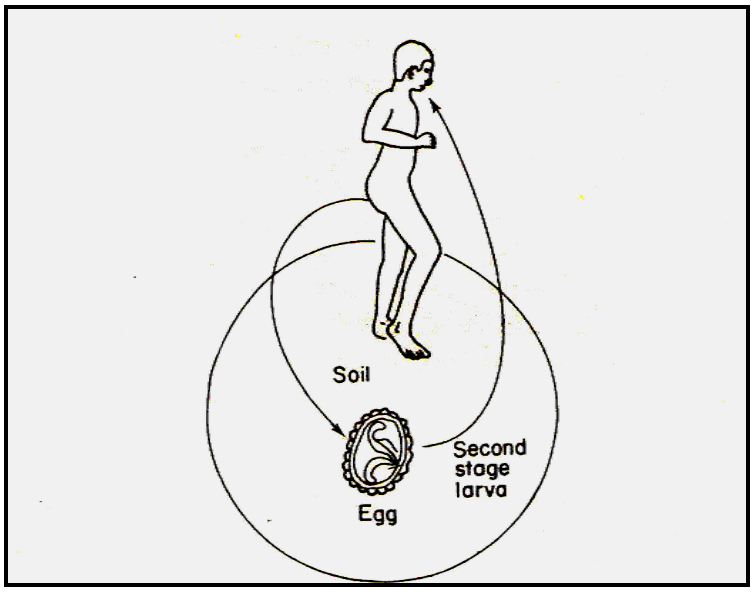

Figure 3.1: Life cycle of *Ascaris* (Ukoli, 1984)

Based on the density – dependent constraint principle mentioned above, the establishment of the worm population and egg production are the rate determining steps in the infection cycle (Churcher *et al.*, 2006). However, the longevity of the eggs in the soil provides a continual source of reinfection that can dominate the influence of those processes in determining disease entrenchment (Anderson and May, 1992; Churcher *et al.*, 2006). Therefore the proposed strategy is to interrupt the developmental cycle of the pathogenic organisms with interventions that target these leverage points (Webber and Rutala, 2001). This includes periodic mass treatment, crop production, hygiene education, and inactivating eggs in soil and excreta (Komiya and Kunii, 1964).

Worm establishment is a function of the host's immune resistance to the invading parasite (Churcher *et al.*, 2006). Thus, providing adequate protein – energy will assist the immune system in suppressing the number of larvae that survive the journey through the body and ultimately reduce worm density (Bradley and Jackson, 2004; King *et al.*, 2005). Since the worms cannot survive outside the host, expelling them by mass chemotherapeutic treatment will instantaneously remove the entire populations, offering

the hosts immediate relief from disease symptoms (Watkins and Pollitt, 1996). However, because there is a population of eggs still in the environment reinfection will occur. Research has shown that after one mass chemotherapy intervention pre – control prevalence and intensity levels were achieved within 1 year and egg production restarted in as little as 2 – 3 months (Kightlinger *et al.*, 1995; Soeripto, 1991; Thein – Hlaing *et al.*, 1987). Therefore repeated applications with concurrent sanitation and hygiene programs are necessary (Arfaa, 1984).

The three main transmission routes for infective eggs are from feces – contaminated surfaces and materials, from fields that have been fertilized with night – soil to workers and by consumption of uncooked plants grown in these fields (Feachem *et al.*, 1983). Providing water and training in hygiene practices in washing surfaces, containers and hands would likely eliminate the first route. Inactivating the eggs in excreta before it is used in crop production will over time reduce the other two transmission routes. Therefore the excreta needs to be safely contained to prevent further environmental contamination and then treated to obtain a parasite free product.

In summary, mass chemotherapy, Solar Latrine with treatment and crop production with treated excreta are proposed. Individual and integrated simulations of these interventions are being used to explore the minimum length of time needed to reduce the risk of reinfection in the community. Mass chemotherapy offers immediate relief to community members and stops the flow of eggs into the soil reservoir. Since there is a store of infective eggs already in the soil it is expected that reinfection is going to occur. Therefore mass chemotherapy will be repeated ad hoc. The Solar Latrine will require the addition of soil which more than likely will come from the area surrounding the homes that is known to have the highest concentrations of eggs. Infective eggs will therefore be deactivated over time. Recycling treated excreta to soybean cultivation will

provide protein rich crops to strengthen the host's immune system (defenses) and thus enable them to resist future infections. In addition this will improve soil structure and fertility. Hygiene education is also essential to interrupt the fecal – oral transmission routes.

3.2 Objectives and subtasks

As discussed in Chapter 1 above, the overall aim is to model the conditions that are required to eradicate parasitic infection in order to compare the sustainability of the systems approach versus traditional vertical intervention approach. This is will be accomplished through a variety of objectives and subtasks as listed below.

3.2.1 Objective 1

- Dynamical modeling of systems' components in STELLA®:
    - Model human – parasite population dynamics,
    - Model parasite infection dynamics in response to mass chemotherapy control measures, and
    - Model crop production using treated humanure as a form of excreta management.

3.2.2 Objective 2

- Develop integrated models:
    - Develop nutrition, sanitation and mass chemotherapy strategies,
    - Determine the best complement to sustainably control infectious diseases in community.

3.2.3 Objective 3

- Design and model a high – rate Solar Latrine:
    - Design a Solar Latrine that treats fecal material using energy from the sun to deactivate microbes by increasing temperature of the product,
    - Calculate hourly solar insolation for the selected site using EXCEL® ,
    - Using data from solar tables and acquired average weather conditions, model the heating, and deactivation processes in COMSOL® to determine the extent to which pathogens can be predictably deactivated in human excreta.

3.3 Study design

3.3.1 Systems approach

A collection of components that work together to produce a unique quality is called a system (Fisher, 2005). Systems theory is based on the assumption that all types of systems have common characteristics regardless of their unique internal structures (Skyttner, 2005). That is, communities characterized by parasite endemicity have similar sets of interdependent controlling processes even if the behavior of individual hosts and the structure of the specific locality are different. Systems approach consists of systems thinking and systems dynamics.

Systems thinking is a methodology used to identify and solve phenomena operating in and arising out of a larger environment (Shiflet and Shiflet, 2006). The interrelationships are conceptualized using causal loop mapping and parts integration techniques as opposed to the traditional linear cause – effect – isolation approach (Richmond and Peterson, 2001). Systems dynamics is using computer simulations to model the global dynamics of the systems components to understand rather than predict

the behavior of the system over time (Ford, 1999; Shiflet and Shiflet, 2006). This approach is considered more realistic and valuable because it can reveal emergent properties that result from nonlinear interactions among systems components and subsequent feedback mechanisms, which are not readily obvious during piecewise investigations. Thus, systems thinking and dynamical modeling can explore critical leverage points, effectiveness, as well as the unintended and counterintuitive effects of public health interventions.

Considering the lifecycle of *Ascaris*, the interactions occurring among the host – microbe – environment are very complicated, however this characteristic complexity emerges from a small number of controlling mechanisms such as biological and population processes described in Section 2.4 above (Boccara, 2004; Holling, 2001). For this research the key factors found in literature that adequately describe the structures that hinder or promote parasite endemicity are modeled separately and simultaneously in STELLA® to identify and understand the general dynamics of the system. From these simulations, an optimal complement of interventions can be derived that will successfully and sustainably control infectious disease. Once accomplished, the successful solutions can be applied across different communities with similar systems emergence attributes or tailored to facilitate disparities unique to a given location (Novick *et al*., 2008).

3.3.2 STELLA®

The STELLA® software is specifically designed for modeling the dynamics of highly interdependent systems (Hannon and Ruth, 2001). The software allows one to represent complex systems conceptually through a series of simple building blocks that represent the controlling processes operating to produce an emergent behavior (Ford, 1999). An icon – based graphical interface in the form of “Stock and Flow” diagrams is used to represent the concepts of systems thinking. The model equations are

automatically generated and made accessible beneath the model layer (see Figure 3.2 and Table 3.1). All generated equations for the STELLA® models presented are made available in the Appendix B.

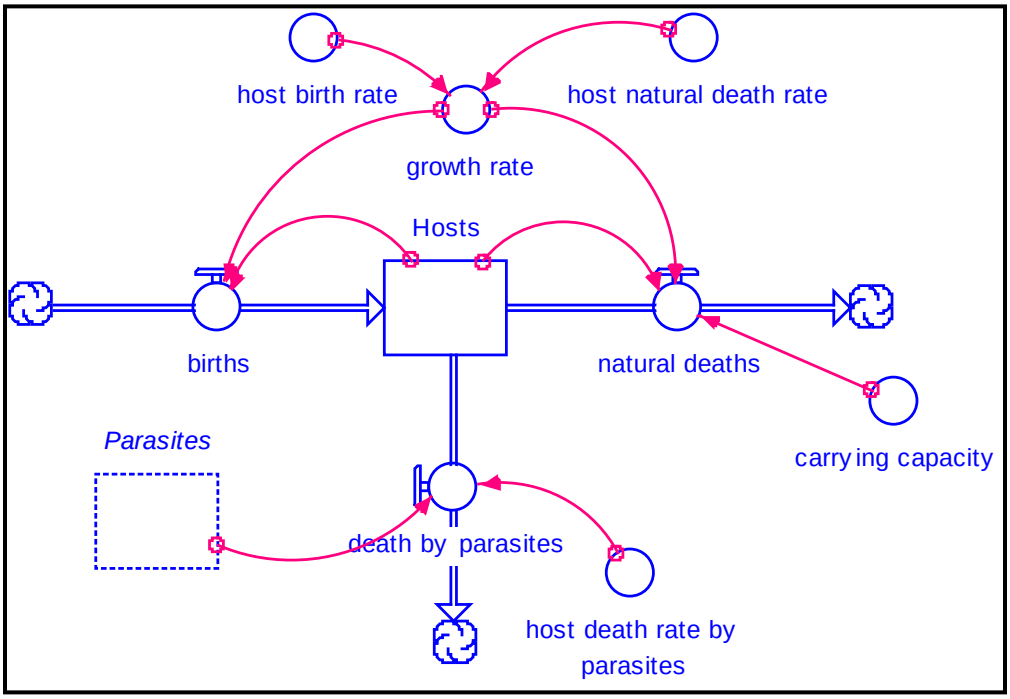

Figure 3.2: Systems thinking representation of host dynamics in STELLA®

Table 3.1: Automatically generated model equations in STELLA®

```
Hosts(t) = Hosts(t - dt) + (births - natural_deaths - death_by_parasites) * dt

INIT Hosts = 150 {host}

INFLOWS:
births = growth_rate * Hosts {host/time}

OUTFLOWS:
natural_deaths = growth_rate * Hosts * Hosts / carrying_capacity {host/time}
death_by_parasites = Parasites * host_death_rate_by_parasites {host/time}

carrying_capacity = 200 {host}
growth_rate = host_birth_rate - host_natural_death_rate {host/host/time}
host_birth_rate = 3 {1/time}
host_death_rate_by_parasites = 0.5 {host/parasite/time}
host_natural_death_rate = 1 {1/time}
```

3.3.3 COMSOL®

The microbial inactivation in the Solar Latrine was modeled using the multi – physics, Finite Element Method (FEM) software COMSOL®. The multi – physics capability of COMSOL® means that it can handle partial differential equations describing different physical processes such as those governing heat transfer, evaporation and microbial inactivation and is able to solve them simultaneously over a given domain or geometry. In the FEM the partial differential equation is transformed into an integral expression and, the domain and boundary conditions are divided into elements resulting in a mesh (see Figure 3.3) with a number of nodal points (Hughes, 2000; Zienkiewicz, 1983). Numerical approximation of the integral provides an approximate solution over each finite element and its contribution summed at each node (Hughes, 2000). The advantages of FEM are its ability to handle any arbitrary geometry, general, constant or varying boundary conditions and heterogeneous materials (Akin, 1994).

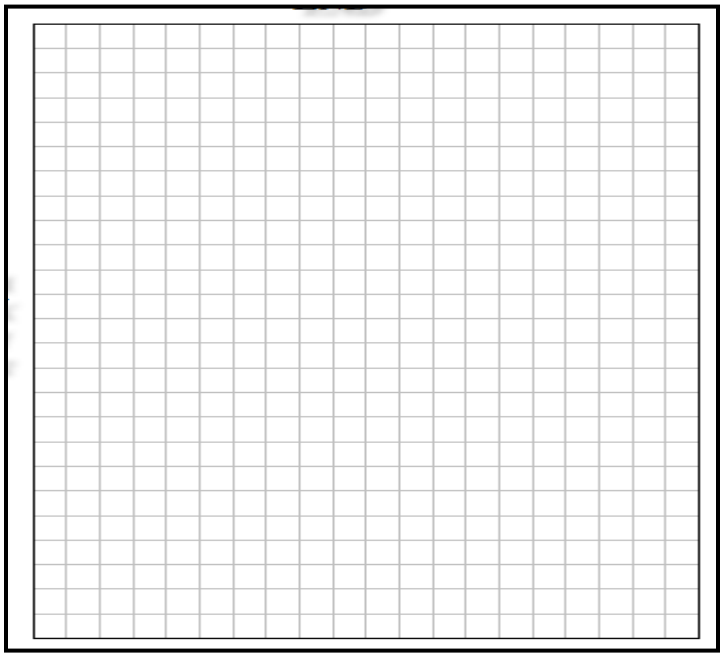

Figure 3.3: Finite element mesh in COMSOL® for a rectangular geometry

3.4 Site selection

The following criteria were used to select the country and ultimately the study community:

- Human excreta and/or sludge reuse in agriculture,
- Currently cultivated or have the ability to grow soybean,
- Infection disease endemicity,
- Poor sanitation, and
- Practice agricultural sun drying.

3.4.1 Study village

The village of Paquila, Guatemala was chosen as the model site because it was considered representative of this region and the above criteria. It is about 10 km^2 and located about 1 ½ hours south of QuetzaItenango and 2 ½ hours west of the capital, Guatemala City (see Figure 3.4). Geographically, Guatemala is located in Central America and is bordered by El Salvador, Honduras, Belize and Mexico. The climate is predominantly tropical with very little temperature variation throughout the year. The rainy season is from May to October with average annual rainfall of about 1,300 mm. It is the most densely populated country in Central America with about 75% of the population living in rural areas (CIA, 2008).

Figure 3.4: Map of Guatemala showing village of Paquila (see star below Coatepeque) (CIA, 2008)

After the 1976 earthquake, several excreta disposal programs were undertaken to bring latrines to rural areas (Strauss *et al.*, 1990). At first simple latrines were installed, however, they were socially rejected because they were difficult to construct on rocky underground and in areas with high groundwater table. The pits would flood during the raining season, the contents would smell and attract flies. The community members went back to open defecation. Following this initial failure, a double – vault latrine with urine separation call Dry Alkaline Fertilizer Family (DAFF) was introduced and recycling latrine contents was encouraged (Plenty, 2008).

Also in 1976, Plenty International, a non – governmental organization based in Tennessee went to Guatemala to help with the rebuilding efforts. In an effort to sustainably reduce malnutrition, they started a soybean farm extension program that provided technical and financial assistance for economically disadvantaged families and

organizations who were interested in learning how to grow soybeans and other dry legumes in rotation with traditional staples, improve family nutrition and food security and, increase annual cash income. This led to the construction of a Mayan owned and operated soy dairy (Alimentos San Bartolo) in the village of San Bartolo, Solola, about 50 miles north of Paquila. Today this facility is managed by the Mayan community development organization, ADIBE, employs eight people and produces a reliable and inexpensive source of protein in the form of soy milk, ice cream, tofu and other products for sale locally and nationally (Plenty, 2008).

There is no specific development program for housing, road construction and environmental sanitation being carried out in the area. In February of 2003 two Christian missionaries, Jim and Dianne Thompson, moved from Asheville, North Carolina and started a base clinic in Paquila (Boca Costa Medical Mission, 2004). Before 2004, only about half the village had access to clean water. An extensive water project by the Thompsons in the summer of 2004 brought access to piped water the rest of the community. Today, there are about four other satellite clinics that serve over 45 villages in “The Boca Costa de Solola” area of Southwestern Guatemala and a developing referral relationship with a hospital offering 24 hours emergency care 45 minutes away in Mazantenango. Over 30% of the patients are seen for gastrointestinal parasitic infections with the highest proportion suffering with intestinal worms (see Figure 3.5 and Table 3.2).

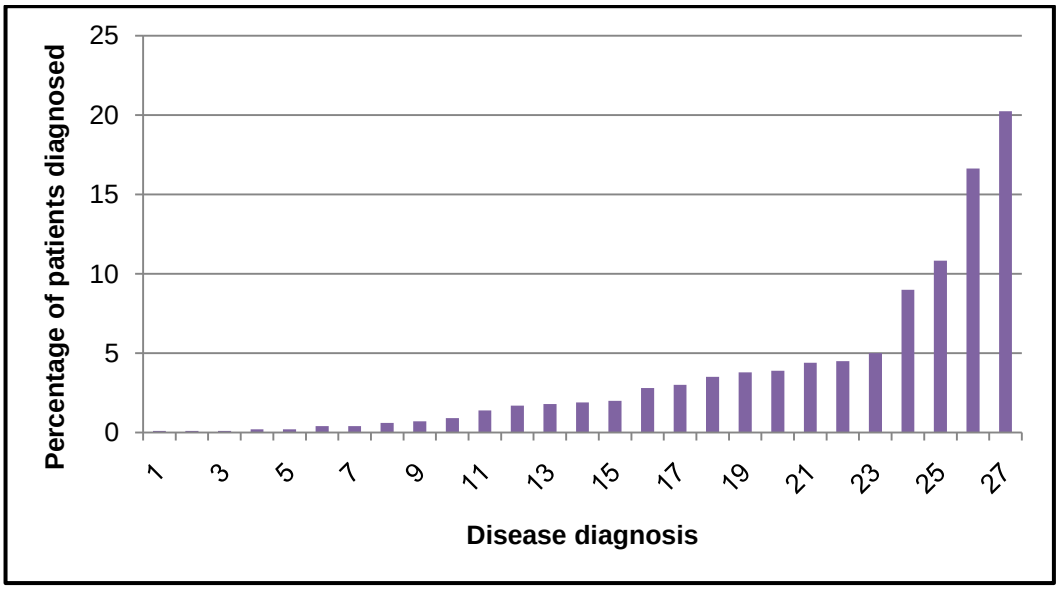

Figure 3.5: Breakdown of the disease diagnosis at area clinics (Boca Costa Medical Mission, 2004)

Table 3.2: Breakdown of the disease diagnosis at area clinics (Full table in Appendix A)

Number code	Disease diagnosis	Percentage of patients diagnosed
11	Bacterial dysentery	1.40
22	Skin infection (fungal)	4.50
23	Gastritis	5.00
24	Amebic dysentery / Giardia	9.00
25	Other: general pain, vitamins, only	10.83
26	Respiratory infections	16.64
27	Intestinal worms	20.24

3.4.2 Study population

The population of Paquila is about 3500 indigenous Mayan. The primary language is Quiche with Spanish secondary. It has one of the highest infant and maternal mortality rates, with 50% of infants dying before age 5. Paquila is a typical agricultural village and relatively isolated, with an extended family unit structure. The people of the villages are mostly subsistence farmers who grow coffee, banana, sugar

cane, corn, rice, root and vegetable crops, and rubber. Children usually start working in the fields by age 5 years and are encouraged to farm a small plot of land next to the main field of their household by age 14. The typical house is a single room hut that is primarily used for sleeping. It is constructed of mud wall, thatched roof with dirt floor. The preceding information was acquired from the Boca Costa Mission's website or through personal communication with the Thompsons.

Due to the relative isolation of the community, infections can be assumed to occur only by intra – community transfers and not from the imported infective stages. Sun drying of agricultural products and brick mean that relevant skills needed to utilize a proposed Solar Latrine are in place. The clinic ensures primary health care and has helped to engender the trust of the community. The successful soybean project in the neighboring community creates potential for inter – community transfer of technology. Villages like Paquila are prime candidates for successful and sustainable control and eradication of *Ascariasis* and other infectious disease (Arfaa, 1984; Komiya and Kunii, 1964; Thein – Hlaing *et al.*, 1984).

4 EPIDEMIOLOGICAL MODEL

4.1 Introduction

This chapter covers the host – parasite relationship that formed the core of the Modified Wheel Epidemiological conceptual framework discussed in Section 2.2.2. Moore (2002) agreed with this strategy of first establishing population dynamics before attempting to propose solutions to environmental health challenges. That is, it is important to first determine the reproduction and transmission rates, life expectancy, and pathogencity of the parasite within the human community before suitable control methods can be prescribed (Boes and Helwigh, 2000).

To review, the establishment of a parasite in a community and its subsequent entrenchment result from a number of inherent biological and population processes that are detailed by organism's lifecycle (see Section 3.1). While endemicity emerges from the confluence of host – parasite – environment interactions, it is the proximate factors such as, female fecundity and longevity, environmental resistance of infective stages, density – dependent constraints on parasite population, and nonlinearity associated with parasite induced host deaths that directly influence the stability of the host – parasite relationship (Anderson and May, 1982).

The overall goal is to simulate population dynamics and to determine how to prevent, reduce or eliminate infection hazard, morbidity and mortality to community members. The specific objectives include:

- Model the host – parasite dynamics,
- Determine the conditions that influence stability, and

- Determine the effects of chemotherapeutic control measures on parasite endemicity.

Consequently, this chapter has three main sections. The chapter begins with a review of general population dynamics that occur in nature with special attention to predator – prey interactions on which the proposed model is based. The model is then translated into STELLA® to determine stability and optimal leverage points for interventions. Finally, a model simulating mean worm burden in response to mass chemotherapy is developed to determine eradication requirements.

Parasite population biology and ecology have been extensively modeled (Anderson and May, 1978; Bradley and May, 1978; Churcher *et al.*, 2006; Crofton, 1971; Dobson, 1988; Macdonald, 1961; Pielou, 1969; White and Grenfell, 1997). However, there is a lack of conformity in the use of notations, their definitions and dimensions. Through out the literature, equations are presented with a plethora of symbols representing the same variable, units not specified and/or inconsistent units even by the same authors. For example, Anderson (1980b) used the symbol (β) to represent density – dependent constraint on host mortality. While in the same year used it to mean the contact rate between hosts and parasitic infective stages (Anderson, 1980a). More recently (Kretzschmar and Adler, 1993) used the same notation to represent host birth rate. Table 4.1 gives a list of the nomenclature adopted in the proposed Human – *Ascaris* model. Similar tables are located throughout the chapter to represent variables as they are introduced in those sections to create clarity and transparency, and reduce confusion.

Table 4.1: Nomenclature and definitions used in Human – *Ascaris* model

Symbol	Description	Units
H	Magnitude of host population at time, t	host
P	Magnitude of worm population at time, t	worm
W	Magnitude of infective egg population at time, t	egg
M	Population mean (ratio of the average number of adult worms to each host) at time, t	worm/host
r	Host growth rate (birth rate – natural death rate)	host/host/time
K	Village carrying capacity of the host population	host
$d_{h,n}$	Host natural death rate	host/host/time
$d_{h,p}$	Host mortality rate due to worm induced death	host/worm/time
$d_{p,n}$	Worm's natural death rate	worm/worm/time
$p(i)$	Probability that a host contains (i) number of worms	[ ]
λ	Egg production rate by adult worms	egg/worm/time
β	Proportion of eggs ingested by individuals in a given time interval; contact rate between infective eggs and hosts	egg/egg/host/time
γ	Rate of inactivation of eggs in the environment; (d_2/time)	egg/egg/time
d_1	Number of ingested eggs that hatch and survive to adulthood	worm/egg
d_2	Proportion of eggs that survive environmental conditions to become infective	egg/egg
ω_f	Proportion of female worms in a metapopulation; all worms in all hosts	[ ]
Φ	Probability that a female worm will mate in an infrapopulation; worms in one host	[ ]
k	Negative binomial clumping parameter, denotes worm dispersion among host population	worm/host

4.2 Population dynamics

4.2.1 General population dynamics

Table 4.2: Nomenclature and definitions used in Section 4.2.1

Symbol	Description	Units
N	Magnitude of species population at time, t	species
r	Species/Prey/Host population growth rate (birth rate – death rate)	1/time
K	Carrying capacity of area	species

The population growth rate of a species in a given area is normally generalized by the following mathematical function (Lotka, 1956):

$$\frac{dN}{dt} = f(N) \qquad [4.1]$$

Where (N) is the number of a given species living in the area at time, (t) and whose future value is a function of the current state of the population (Bartlett, 1960; Boccara, 2004). For natural population growth (due to death and birth processes only, assuming no immigration or emigration), the simplest model for $f(N)$ is the Verhulst logistic equation, for which detailed derivation and rationale can be found in (Hutchinson, 1978; Pielou, 1969):

$$\frac{dN}{dt} = rN\left(1 - \frac{N}{K}\right) \qquad [4.2]$$

The model satisfies the following assumptions (Hutchinson, 1978):

- Each individual has at least one parent like itself, and
- If the area occupied by the individuals is finite and there is no adverse event to cause extinction, the population will increase at a rate (r = birth rate – death rate) up to the carrying capacity, (K) which is determined by environmental resistance. (rN) is the biotic potential of the organisms, that is, the maximum

growth where neither scarcity (K) nor intra – species crowding (N^2) limits reproduction (Pielou, 1969).

4.2.2 Predator – prey dynamics

Table 4.3: Nomenclature and definitions used in Section 4.2.2

Symbol	Description	Units
N_1	Magnitude of prey population at time, t	prey
N_2	Magnitude of predator population at time, t	predator
d	Death rate of predators	1/time
α	Contact rate between predator and prey	1/predator/time
η	Conversion efficiency of eaten preys to new predators	predator/prey

Equation [4.2] describes the population dynamics of a single species, however in nature, organisms of different species do not live in isolation but interact with each other in two main ways; competition for common environmental resources or one use the other as a food source (Leslie and Gower, 1960). This work advances the latter relationship, commonly generalized as the predator – prey model. The Lotka – Volterra equations are the simplest deterministic representation of the predator – prey interaction (Maynard Smith, 1974; Pielou, 1969). Equations [4.3] are modified versions of the origin formulation, accounting for density and resource constraints ($\frac{N_1}{K}$) on the prey population:

$$\frac{dN_1}{dt} = rN_1\left(1 - \frac{N_1}{K}\right) - \alpha N_1 N_2$$
$$\frac{dN_2}{dt} = \eta\alpha N_1 N_2 - dN_2 \qquad [4.3]$$

Where (r) is the growth rate of the prey (N_1), (α) is the contact rate between predator (N_2) and prey deaths resulting from predation is given by ($\alpha N_1 N_2$). (d) is the

death rate of the predator while birth rate is directly proportional to prey – predator interaction ($\eta\alpha N_1 N_2$), with the prey to predator offspring conversion efficiency (η). A unique characteristic of this model is that of damped population oscillations around a fixed equilibrium (Lapage, 1963). That is, when the prey population increases predator – prey contact goes up with concomitant increases in predation and predator birth rates. This feeds back negatively to reduce host numbers with subsequent slowing in the growth of the predator population.

The derivation of this system of equations is based on a number of simplifying assumptions, as follows (Maynard Smith, 1974):

- If preys are able to avoid predation, their population growth is determined by the logistic model in equation [4.2],
- Both species move and interact randomly, similar to molecules in a chemical reactions,
- The predator's feeding time is much smaller than the time between feeding, so it is reasonable to assume that the rate at which a prey gets eaten is proportional to their population density ($N_1 N_2$),
- Eaten preys are instantaneously converted to new predators. That is, there are no developmental time delays,
- Time is a continuous variable since successive generations overlap allowing the use of differential equations to represent dynamics (Anderson and May, 1978), and
- The population densities of both species are only functions of time, not the age, sex or genotype of their members. Thus, the rate of change of population densities of predator and prey can be represented by ordinary differential equations (May and Mclean, 2007).

This model is analogous to the collision theory in chemical kinetics (Lotka, 1956). This conceptualization only crudely represents the predator – prey dynamics because predators tend to deliberately seek out preys and there is a time lapse between eating a prey, metabolic assimilation and subsequent birth of an offspring. The deterministic nature of these equations also makes them ecologically unrealistic (Maynard Smith, 1968). For example, a fundamental assumption is that the population size must be infinite (detailed in Section 4.2.5 below), which is not possible in a finite area (Bartlett, 1957). In addition, they ignore random fluctuations characteristic to biological and population processes (Boccara, 2004; Maynard Smith, 1974). In spite of these limitations, however, the predator – prey model is valuable as a point of departure that can be customized to more accurately mirror biological interactions of the host – parasite population dynamics. The following sections will detail modifications to the system of equations in [4.3] to make them more representative of the biological and population processes that occur in host – parasite relationships.

4.2.3 Host – parasite dynamics

The host – parasite relationship is a unique manifestation of the predator – prey model and is considered to be mathematically equivalent (Anderson and May, 1978; Pielou, 1969). An increase in the host population results in increased host – parasite contact, which leads to higher rates of infection and average parasite burden per host. As the number of parasite per host increases, the rate of infection induced host deaths also increases creating negative feedback to reduce the parasite population, resulting in population oscillations characteristic of predator – prey dynamics (Pielou, 1969).The encounters are similarly not random, but are functions of host and parasite behavioral patterns. A minor difference in the two systems is manifested in the absolute numbers of the analogous population members. That is, preys are normally the more abundant of

the two species in the predator – prey relationship. In the host – parasite model, parasites, which are the “predators”, tend to have population sizes much larger than their hosts.

4.2.4 Deterministic host – parasite dynamics

In general, parasites have two types of life cycles, indirect (more than one hosts) and direct (one host). *Ascaris* epitomizes parasites with direct life cycles (see Figure 4.1). The parasite has two distinct populations, the adult worms infesting human hosts and eggs dispersed in the environment (Usher and Williamson, 1974).

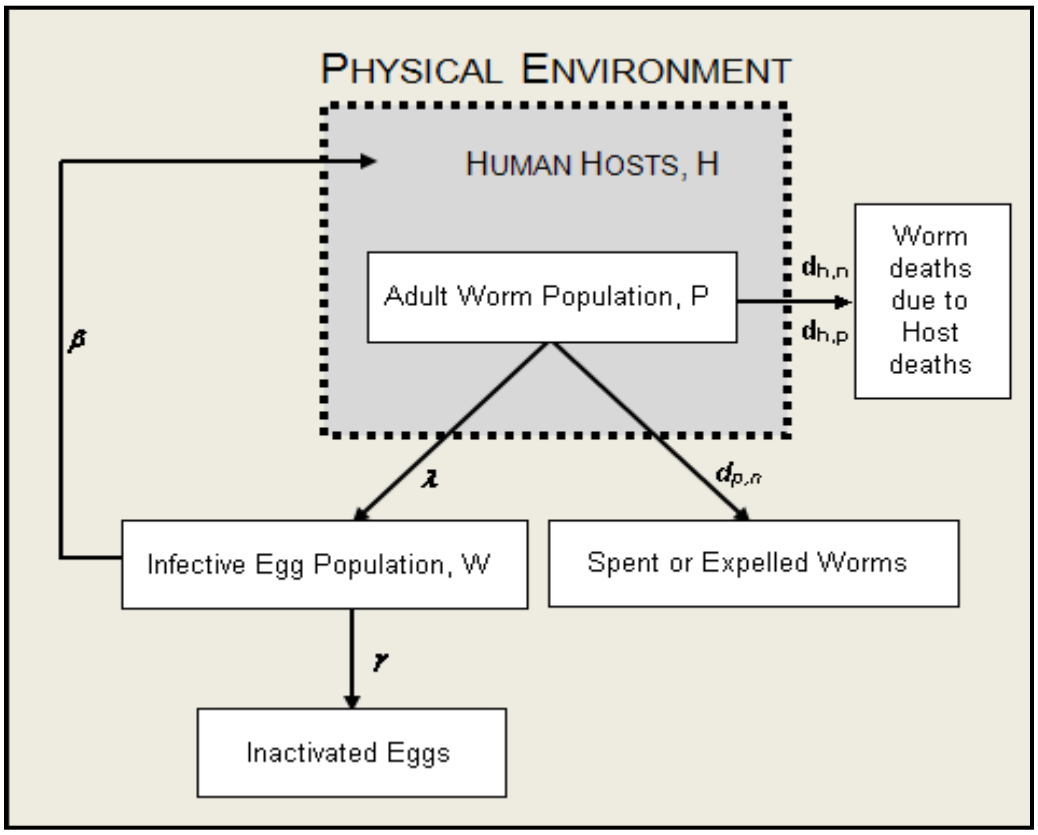

Figure 4.1: Flow chart of human – *Ascaris* population dynamics

4.2.4.1 Deterministic host population equations

Table 4.4: Nomenclature and definitions used in Sections 4.2.4.1 and 4.2.4.2 (Boccara, 2004)

Symbol	Reference Symbol*	Description	Units
H	H	Magnitude of host population at time, t	host
P	P	Magnitude of worm population at time, t	worm
$d_{h,p}$	s	Host mortality rate due to worm induced death	host/worm/time
r_p	r_p	Parasite population growth rate	1/time
c_h	c	Worm carrying capacity of each host	worm/host

*Reference Symbol: notation used by the reference cited in the table heading

The prey population of equation [4.3] is adopted here to represent the host population in the host – parasite model. As before, in the absence of parasites, the host is assumed to growth logistically, limited only by the availability of environmental resources. For parasitic infection not every encounter results in death of the host. Thus, the contact rate, (α) is now redefined as parasite induced host death rate, ($d_{h,p}$), which is assumed constant for the deterministic representation. In reality death only occurs at high worm burdens, which in turn depends on the probability distribution of the worms among community members. This is accounted for in the stochastic model presented in Section 4.2.5.1 below. The host dynamics from equation [4.3] is now:

$$\frac{dH}{dt} = rH\left(1 - \frac{H}{K}\right) - d_{h,p}HP \qquad [4.4]$$

4.2.4.2 Deterministic parasite population equations

The predator population dynamics in equation [4.3] assumes a constant per capita death rate given by ($-dN_2$) and a birth rate that is proportional to the availability of preys leading to ($\eta\alpha N_1 N_2$). However, in reality density (number of worms per organ) is limited by physical capacity of the host, infrapopulation competition for available

resources of resources and the host's immunological response which increases with infection intensity (Anderson, 1998; Englund, 1988; Loukas *et al.*, 2000). To account for these constraint, a logistic – type model similar to equation [4.2], has been proposed where the carrying capacity of an individual host, analogous to (K) is given by ($c_h H$) and intra species competition given by (P^2) (Boccara, 2004):

$$\frac{dP}{dt} = r_p P\left(1 - \frac{P}{c_h H}\right) \qquad [4.5]$$

4.2.4.3 System of deterministic equations for host – parasite dynamics

The system of equations representing the host – parasite dynamics is represented in equation [4.5]. These equations are just two of many variations possible, through combining different terms and making other assumptions about the ecology of the species.

$$\frac{dH}{dt} = rH\left(1 - \frac{H}{K}\right) - d_{h,p} HP$$
$$\frac{dP}{dt} = r_p P\left(1 - \frac{P}{cH}\right) \qquad [4.6]$$

4.2.5 Stochastic host – parasite population dynamics

Table 4.5: Nomenclature and definitions used in Section 4.2.5 (Maynard Smith, 1974)

Symbol	Description	Units
P	Magnitude of parasite population at time, t	parasite
P_o	Initial magnitude of parasite population at time, t = 0	parasite
$\hat{P}$	Average parasite population	parasite

The preceding discussion was limited to deterministic representations of the host – parasite population dynamics and are therefore subjected to the inherent limitations of

that class of models (Maynard Smith, 1974). For example, consider the deterministic model for an exponentially growing parasite population:

$$\frac{dP}{dt} = r_p P \qquad [4.7]$$

The number of individuals at time (t) is thus given by the well known solution:

$$P = P_o\, e^{r_p t} \qquad [4.8]$$

The deterministic assumption is that a fraction of ($r_p \mathrm{dt}$) individuals are born over a short time interval, (dt) (Maynard Smith, 1974). The corresponding stochastic model assumption is, for the time period (dt), an individual produces one offspring with probability, ($r_p \mathrm{dt}$) and no offspring with probability ($1 - r_p \mathrm{dt}$) (Bartlett, 1960). Therefore whole instead of fractional individuals are reproduced at each time step. The mean number of individuals ($\hat{P}$) and the variance of (P) can then be calculated at time (t) by (May, 1974):

$$\hat{\hat{P}} = P_o\, e^{r_p t}$$
$$var(P) = P_o\, e^{2r_p t}(1 - e^{-r_p t}) \qquad [4.9]$$

The resulting population mean ($\hat{P}$) is the analogue of the solution for the deterministic model in equation [4.7] for replicate populations with initial size (P_o). The variance of (P) measures any differences in the population sizes after a time step. The coefficient of variation (CV); ratio of the standard deviation to the mean, is the best method for comparing dispersion among populations and is given by (Bradley and May, 1978):

$$CV = \frac{\sqrt{var(P)}}{\hat{P}} = \frac{\sqrt{P_o\, e^{2r_p t}(1 - e^{-r_p t})}}{P_o\, e^{r_p t}} \qquad [4.10]$$

$$\lim_{t \to \infty} CV \to \frac{1}{\sqrt{P_o}}$$

Thus, if (P_o) is large, there is very little deviation among the population means, which tend to the mean in equation [4.8]. Since the stochastic and deterministic means are equivalent, it can be concluded that for infinitely large (P_o) both models are equally representative of the population dynamics (Bartlett, 1960; May, 1974).

In order for deterministic models to more accurately describe the host – parasite relationship, more complicated equations are required. One method to overcome this limitation is to develop hybrid models consisting of deterministic models while allowing for stochastic variations (Anderson and May, 1978; Pielou, 1969). These models can then be developed to maintain the ecological and biological fidelity of the populations. This approach has been adopted for the *Ascaris* – human population dynamics based on the predator – prey model in equation [4.3] presented here and is described in the following sections.

4.2.5.1 Stochastic host population equation

Table 4.6: Nomenclature and definitions used in Section 4.2.5.1

Symbol	Description	Units
i	Worm burden	worm/host
$p(i)$	Probability of a host containing i parasites	[ ]
$d_{h,p}(i)$	Death rate among hosts with i parasites	host/worm/time

For the host population (H) dynamics (Anderson, 1978, 1980a, 1982; Anderson and May, 1978, 1992; May and Anderson, 1978):

- As a first approximation, there is no density – dependent constraint on the growth rate (r), leading to exponential instead of logistic reproduction similar to equation [4.6]. Instead, the host population is assumed to be regulated by parasitic activities (Anderson, 1980a).

- The rate of parasite induced host mortality is a function of the worm burden, (i). That is, the more worms a host harbors the more likely death will result due to parasite induced complications such as abdominal obstruction, which is especially true for children (Thein – Hlaing and Myat Lay, 1990). If $p(i)$ is the probability that a given host contains (i) number of worms, then the death rate among those with (i) parasites is given by $d_{h,p}(i)$. The death rate will therefore depend on the number of parasite per host and the assumed probability distribution of $p(i)$. The total parasite induce deaths among host is given by:

$$H\sum_{i=0}^{\infty} d_{h,p}(i).p(i) \qquad [4.11]$$

- The host equation from [4.3] then becomes:

$$\frac{dH}{dt} = rH - H\sum_{i=0}^{\infty} d_{h,p}(i).p(i) \qquad [4.12]$$

4.2.5.2 Stochastic worm population equation

Table 4.7: Nomenclature and definitions used in Section 4.2.5.2

Symbol	Description	Units
W	Magnitude of egg population at time, t	egg
d_1	number of ingested eggs that become established worms	worm/egg
τ_1	Time period between egg ingestion and established worm egg production; prepatent period	time
β	Contact rate between host and infective eggs; host's ingestion rate of infective eggs	egg/egg/host/time
$d_{h,n}$	Death rate of host due to cause other than parasites	host/host/time
$d_{p,n}(i)$	Death rate of parasites as a function of infrapopulation competition	worm/worm/time

For the worm population (P) dynamics (Anderson, 1978, 1980a, 1982; Anderson and May, 1978, 1992; May and Anderson, 1978):

- When infective eggs of *Ascaris* are ingested only a portion (d_1) will survive the prepatent period (τ_1), time between infection and when the larva finally return to the small intestine and develop to reproductive maturity. Assuming that the number of worms established in all host (P) is a linear function of the number hosts (H), and infective eggs in the environment (W), then the total number of established worms is given by equation [4.12]:

$$d_1 \beta W H \qquad [4.13]$$

- The rate of change of the worm population is the difference between number of worms established in the human population and the losses due to various death processes. Parasite mortalities have three components; natural deaths of worm and host, and host deaths as a result of high parasite burdens (Anderson and May, 1978). These are discussed in turn below.
- Losses due to parasite natural host deaths at a rate of ($d_{h,n}$). That is, when individuals die, the worms die with them, assuming that the worm burden is not high enough to cause these deaths. The total number of worms lost in this manner is:

$$d_{h,n}.H\sum_{i=0}^{\infty} i.p(i) \qquad [4.14]$$

- Losses due to parasite induced deaths. From equation [4.10] the number of host dying as a result of high worm burden was given by $H\sum_{i=0}^{\infty} d_{h,p}(i).p(i)$. Therefore the product of the number host dying and the average worm burden per host (P/H) gives the total number of worms dying with them:

$$P\sum_{i=0}^{\infty} d_{h,p}(i).p(i) \qquad [4.15]$$

- Losses due to worms dying naturally due to worms being spent or host's immunological responses. The natural life expectancy for an average *Ascaris* worm is about 1 year. However, as the worm burden increase, the host immunological response is heightened which results in a higher mortality rate $d_{p,n}(i)$, among the parasites. As a first approximation $d_{p,n}(i)$ is considered constant and is given by ($d_{p,n}$). This is a reasonable assumption, since as the number of worms increases the likelihood of host death increases, which is accounted for in equation [4.14]. Total worm death due to natural causes is given by:

$$d_{p,n}.P \qquad [4.16]$$

- The parasite equation from [4.3] then becomes:

$$\frac{dP}{dt} = d_1\beta WH - H.d_{h,n}\sum_{i=0}^{\infty} i.p(i) \qquad [4.17]$$
$$-P\sum_{i=0}^{\infty} d_{h,p}(i).p(i) \; - d_{p,n}.P$$

4.2.5.3 Stochastic egg population equation

Table 4.8: Nomenclature and definitions used in Section 4.2.5.3

Symbol	Description	Units
W	Magnitude of egg population at time, t	egg
$\lambda(i)$	Rate of egg production as a function of parasite density	egg/worm/time
ω_f	Proportion of female worm; assume to be 1:1 ratio	[ ]
Φ	Probability that female worm will mate	[ ]
τ_2	Time period between eggs exiting host and developing to become infective to host	time
d_2	Proportion of eggs produced that survive environmental conditions to become infective	egg/egg
γ	Inactivation rate of eggs in the environment; (d_2/time)	egg/egg/time

The infective egg population (W) dynamics (Anderson, 1978, 1980a, 1982; Anderson and May, 1978, 1992; May and Anderson, 1978):

- The rate of change of infective eggs in the environment is a function of the fecundity of the established worm population, ingestion by host and the rate of inactivation as a result of harsh ambient conditions.
- Research as shown that egg production $\lambda(i)$, affected by the worm burden (i) of the host (Croll *et al.*, 1982). In addition because *Ascaris* is dioecious (both sexes required for infective egg production) and polygamous (a single male will mate with multiple females), the fertility rate depends on the proportion of female worms in the population, (ω_f) and probability that a given female will mate, (Haukisalmi *et al.*, 1996). Egg production for the entire established worm population in the host is given by:

$$\omega_f . \Phi . H \sum_{i=0}^{\infty} \lambda(i) . p(i) \qquad [4.18]$$

- The eggs are not immediately infective when released into the environment but require a developmental period (τ_2) before they are able to cause disease in the host population. During this time, the developing embryo is particularly vulnerable and many die from exposure to harsh ambient conditions such as direct exposure to sunlight and desiccation. Therefore only a proportion (d_2) will survive to become pathogenic.
- Losses are due to environmental inactivation at a rate (γ), and ingestion by host (βWH) as describe in equation [4.16]. The rate of change of eggs in the environment is given by:

$$\frac{dW}{dt} = d_2\omega_f.\Phi.H\sum_{i=0}^{\infty}\lambda(i).p(i) - \gamma W - \beta WH \qquad [4.19]$$

4.2.5.4 System of stochastic equations for host – parasite dynamics

The three populations are represented in equation [4.19] below. The following discussion will involve further explanation of the various population and biological processes involved in parasite – host dynamics and how these lead to stability and subsequent disease endemicity. This analysis will then be applied to evaluating the effects of various control strategies on the dynamics of the parasitic population in this and ensuing chapters.

$$\frac{dH}{dt} = rH - H\sum_{i=0}^{\infty}d_{h,p}(i).p(i)$$

$$\frac{dP}{dt} = d_1\beta WH - H.d_{h,n}\sum_{i=0}^{\infty}i.p(i) - P\sum_{i=0}^{\infty}d_{h,p}(i).i.p(i) - d_{p,n}.P \qquad [4.20]$$

$$\frac{dW}{dt} = d_2\omega_f.\Phi.H\sum_{i=0}^{\infty}\lambda(i).p(i) - \gamma W - \beta WH$$

4.2.5.5 Statistical distribution and spatial pattern of worms among hosts

Table 4.9: Nomenclature and definitions used in Section 4.2.5.5

Symbol	Description	Units
M	Mean worm burden	worm/host
$\mathrm{E_t}(i)$	"First moment" define a mean worm burden, M	worm/host
$\mathrm{E_t}(i^2)$	"Second moment" define as variance, $Var\ (i)$ by (Bliss and Fisher, 1953)	worm/host
k	Clumping parameter of the negative binomial distribution	worm/host
$\mathrm{D} = \frac{\gamma}{\beta}$	"Scanning power" of infective eggs; number of host acquiring infection by (Macdonald, 1965)	host

From the three governing equations above, the number of worms per host (i) is an important variable, whose value depends on the statistical distribution of its frequency. In general discrete ecological data are observed to fall into three categories; underdispersed (evenly dispersed), random and overdispersed. These spatial patterns are represented by the positive binomial, Poisson and negative binomial probability distributions respectively (Anderson, 1980a). The latter two distributions are particularly relevant to parasitic organisms and will be discussed further here.

Consider a community endemic for *Ascaris*, with each individual carrying (i) number of worms, ($i = 0, 1, 2, \ldots n$). If each worm were randomly and independently assigned to a host, then their dispersion would be considered random. A sample from this host population would show that the number of worms per host is a Poisson variable (Pielou, 1969). This distribution assumes that the maximum density (number of worm in small intestine) is the same for each host and that each host has the same probability of being infected by a worm (Maynard Smith, 1968). Thus, the mean and the variance of the observed frequency distributions of the number of worms per host are equal for this

distribution (Bhattacharyya, 1977). The mean and variance of the Poisson distribution is given by (Anderson and May, 1978):

$$M \equiv \mathrm{E_t}(i) = \frac{P}{H}$$
$$Var\ (i) \equiv M \qquad [4.21]$$

However, certain segments of the host population are more at risk for acquiring infection and higher worm burdens due heterogeneous distribution of infective eggs, differential habits and susceptibility to infection among community members (Wakelin, 1987). For example, older hosts are physiologically able to carry more worms and children are more likely to be infected because of behavioral habits such as playing in dirt. From field studies of *Ascaris* infections, the variance of the observed frequency distribution of the number of worms per host is usually much greater than the mean and a clumped pattern of both infection incidence and egg location is typically observed (May, 1977; Wong *et al.*, 1991). That is, a minority of the host population is infested with the majority of the worm population, referred to as “wormy people” in Norman Stoll’s 1947 seminal work (Stoll, 1999); reprinted.

This means that the greater proportion of the worm population is exposed to severe “crowding effects” (Anderson and May, 1992). Population processes such as parasite mortality and fecundity are greatly influenced by parasite burden, which has been shown to regulate parasite transmission and establishment (Churcher *et al.*, 2006; Medica and Sukhdeo, 2001; Uznanski and Nickol, 1980). Overdispersed or aggregated distribution, therefore, has important implications for host – parasite stability and by extension parasite endemicity (Boes *et al.*, 1998).

The degree of aggregation is measured by the parameter (k), when the intensity has a negative binomial distribution. (k) is an intrinsic property of the clumping pattern of the worms that is independent of mean worm burden. For example, in general, the

worm's natural death rate is greatest in hosts with higher worm densities as resources become limiting in the small intestine. However, unless these hosts are also dying, they will still have higher than average worm burdens due to their higher risk behaviors. Thus, the overall population mean (P/H) is reduced but the spatial arrangement denoted by (k) is unchanged. In terms of measuring the success of an intervention, it will be shown later in this chapter that because of this phenomenon, morbidity may be greatly reduced but disease prevalence and incidence remain unchanged. The mean and variance of the negative binomial distribution is given by Bliss and Fisher (1953):

$$M \equiv \mathrm{E_t}(i) = \frac{P}{H}$$
$$Var\ (i) \equiv \mathrm{E_t}(i^2)\ \equiv \mathrm{M} + \frac{\mathrm{M}^2}{\mathrm{k}} = \frac{P}{H}\left(1 + \frac{P}{kH}\right) \qquad [4.22]$$

Low values of (k) indicate very high variance or dispersion from the population mean, that is, pronounced worm aggregation. The opposite is true for high values. It is interesting to note that as (k) becomes infinitely large the variance equals the mean (equation [4.23]); that is, the frequency distribution of the worm burden becomes Poisson.

$$\lim_{k\to\infty}\left(M + \frac{M^2}{k}\right) \to M$$
$$Var\ (i) \equiv \mathrm{M} = \frac{P}{H} \qquad [4.23]$$

4.2.6 Simplifying host, worm and egg population dynamics

The birth, death and transmission processes described by the equations of [4.19] exhibit random characteristics and are subjected to density – dependent constraints. These features are captured by the worm burden and its probability distribution among individuals in the host population as discussed in Section 4.2.5.5 above. The

overdispersed distribution was chosen because it most accurately mirrored the biological and population processes of parasitic organisms. However, an important departure from the most influential models found in literature will first be dealt with.

4.2.6.1 Units inconsistency in Anderson and May (1978)

Table 4.10: Nomenclature and definitions used in Anderson and May (1978) and May and Anderson (1978)

Reference symbol	Equivalent symbol*	Description	Units from reference
a	r =(a - $d_{h,n}$)	Host birth rate	/host/time
b	$d_{h,n}$ [host/host/time]	Host natural death rate	/host/time
α	$d_{h,p}$ [host/worm/time]	Host mortality rate due to worm induced death	/host/time
λ	λ [egg/worm/time]	Egg production rate by adult worms	/worm/time
μ	$d_{p,n}$ [worm/worm/time]	Worm's natural death rate	/worm/time
$H_o = \frac{\gamma}{\beta}$	$\mathrm{D} = \frac{\gamma}{\beta}$ [host]	Transmission efficiency constant/Scanning power	unspecified
M	M [worm/host]	Mean worm burden	worm/host
$\mathrm{E_t}(i)$	M [worm/host]	"First moment" define a mean worm burden, M	worm/host
$\mathrm{E_t}(i^2)$ see below	see [4.22] above	"Second moment"; mean – square number of parasites per host	worm/host
k	k [worm/host]	Clumping parameter of the negative binomial distribution	unspecified
β	β [egg/egg/host/time]	Egg transmission rate per host	/host/time

*Notation and units used in proposed Human – *Ascaris* model of this work

In their ground breaking work, Anderson and May (1978) proposed a system of equations that are foundational to this work and countless others over the past 30 years. However, on closer inspection there are fundamental flaws. For example, as proposed, the units are inconsistent. Consider equation (7) from their paper:

$$\frac{dH}{dt} = (a - b)H - \alpha P \qquad [4.24]$$

Dimensionally equation [4.23] is as follows using the units in Table 4.10:

$$\frac{host}{time} = \frac{1}{time}.host - \frac{1}{time}.worm \qquad [4.25]$$

That is, (αP) has units of worm/time instead of the required host/time to ensure unit – consistency.

Similarly equation (9):

$$\frac{dP}{dt} = \frac{\lambda PH}{H_o + H} - P(b + \mu + \alpha) - \alpha\frac{P^2}{H} \qquad [4.26]$$

Dimensionally equation [4.25] is as follows:

$$\frac{worm}{time} = \frac{[egg.worm^{-1}.time^{-1}].worm.host}{host} - worm.\frac{1}{time} - \frac{1}{time}\frac{worm^2}{host} \qquad [4.27]$$

Unit – inconsistencies occur in two places, $\frac{\lambda PH}{H_o+H}$ having units of egg/time and $\alpha\frac{P^2}{H}$ units of worm^2/host/time, when the correct units should be worm/time. These inconsistencies will be addressed in the proposed models in the following sections.

4.2.6.2 Hybridized equations for host population

The parasite pathogencity rate $d_{h,p}(i)$ is defined as a function of the worm burden. This relationship is assumed to linear for this work because previous works have determined that nonlinear representations do little to improve the accuracy (Crofton, 1971). Therefore, $d_{h,p}(i) = d_{h,p}.i.$ By definition $\sum_{i=0}^{\infty} i.p(i)$ is defined as the expected number of (i) at time (t) or the population mean worm burden and is denoted by $\mathrm{E_t}(i)$. Substituting both these values into the host equation of [4.19] simplifies to equation [4.27] where $\mathrm{E_t}(i)$ depends on the spatial distribution of the worms among the hosts.

$$\frac{dH}{dt} = rH - d_{h,p}.H.\mathrm{E_t}(i) \qquad [4.28]$$

For overdispersed distributions, $\mathrm{E_t(i)} = \frac{\mathrm{P}}{\mathrm{H}}$. Equation [4.27] now becomes:

$$\frac{dH}{dt} = rH - d_{h,p}.P \qquad [4.29]$$

Dimensionally equation [4.28] is as follows:

$$\frac{host}{time} = \frac{1}{time}.host - \frac{host}{worm/time}.worm \qquad [4.30]$$

4.2.6.3 Hybridized equations worm population

Assuming constant egg productivity rate (λ) independent of density – constraints as a first approximation and substituting $\lambda(i) = \lambda.i$ and the identity $\mathrm{E_t}(i)$, the infective egg population equation of [4.19] becomes:

$$\frac{dW}{dt} = d_2\lambda P - \gamma W - \beta WH \qquad [4.31]$$

Unit – consistency check:

$$\frac{egg}{time} = \frac{egg}{egg}.\frac{egg}{\frac{worm}{time}}.worm - \frac{egg}{\frac{egg}{time}}egg - \frac{1}{host/time}.egg.host \qquad [4.32]$$

The life expectancy of the host, worm and egg populations differ significantly by several orders of magnitudes as shown in Table 4.11 below. Thus, density of the infective stages in the environment can be assumed to equilibrate instantaneously, relative to the variations in the other populations, to $\frac{\mathrm{dW}}{\mathrm{dt}} = 0$. Rearranging equation [4.30] to solve for number of infective eggs in the environment, (W) gives:

$$W = \frac{d_2\lambda P}{\gamma + \beta H} \qquad [4.33]$$

Table 4.11: Relative lifespans of human, worm and egg populations in the lifecycle of *Ascaris* (CIA, 2008)

Population	Lifespan (years)
Human	69
Adult worm	1
Ascaris egg	0.1

Macdonald (1961) introduced the concept of "scanning power" which when applied to *Ascaris*, is the number of host that infective eggs will succeed in coming into contact with and surviving to adulthood. The "scanning power", (D) is define as the ratio of the mortality rate of the eggs, (γ) and proportion of eggs ingested by human hosts, (β). Substituting $\mathrm{D} = \frac{\gamma}{\beta}$ into equations [4.32] gives:

$$\beta W = \frac{d_2 \lambda P}{D + H} \qquad [4.34]$$

Substituting equation [4.33] into the worm population equation of [4.19] gives:

$$\frac{dP}{dt} = \frac{d_1 d_2 \lambda P H}{D + H} - H.d_{h,n} \sum_{i=0}^{\infty} i.p(i) - P \sum_{i=0}^{\infty} d_{h,p}(i).i.p(i) - d_{p,n}.P \qquad [4.35]$$

From above the parasite induced host deaths was assumed to be $d_{h,p}(i) = d_{h,p}.i$. The total death rate among hosts caused by heavy worm burden is given by:

$$P \sum_{i=0}^{\infty} d_{h,p}(i).i.p(i) \equiv d_{h,p} \mathrm{PE_t}(i^2) \qquad [4.36]$$

If the worms' natural mortality rate, ($d_{p,n}$) is assumed to be proportional to the worm burden, then $d_{p,n}(i) = d_{p,n} i$. Substituting this identity and equation [4.35] into [4.34] gives:

$$\frac{dP}{dt} = \frac{d_1 d_2 \lambda P H}{D + H} - H d_{h,n} \mathrm{E_t}(i) - P d_{h,p} \mathrm{E_t}(i^2) - d_{p,n}.P \qquad [4.37]$$

Anderson and May (1978) defined the second moment $\mathrm{E_t}(i^2)$ as the mean – square number of worms per host. For overdispersed distribution $\mathrm{E_t}(i^2)$ was defined as:

$$\mathrm{E_t}(i^2) \equiv M + M^2\left(\frac{k+1}{k}\right) \qquad [4.38]$$

Thus giving equation (13):

$$\frac{dP}{dt} = \frac{\lambda PH}{H_o + H} - P(b+\mu+\alpha) - \alpha\frac{P^2}{H}\left(\frac{k+1}{k}\right) \qquad [4.39]$$

Resulting in similar inconsistencies from equation [4.26]:

$$\frac{worm}{time} = \frac{[egg.worm^{-1}.time^{-1}].worm.host}{host} - worm.\frac{1}{time} - \frac{1}{time}\frac{worm^2}{host} \qquad [4.40]$$

However, Bliss and Fisher (1953) in their equally seminal work define the second moment of the negative binomial as given in equation [4.21] above, where $\mathrm{E_t}(i^2) \equiv M + \frac{M^2}{k}$. Thus, equation [4.36] becomes:

$$\frac{dP}{dt} = \frac{d_1 d_2 \lambda PH}{D+H} - d_{h,n}H.\frac{P}{H} + P.d_{h,p}\left(\frac{P}{H} + \frac{P^2}{kH^2}\right) - d_{p,n}P$$
$$\therefore \frac{dP}{dt} = \frac{d_1 d_2 \lambda PH}{D+H} - P\left(d_{h,n} + d_{p,n} + \frac{P.d_{h,p}}{H}\right) - P.d_{h,p}\left(\frac{P^2}{kH^2}\right) \qquad [4.41]$$

In terms of units, equation [4.40] becomes:

$$\frac{worm}{time} = \frac{\left[\frac{egg}{egg}\right]\left[\frac{worm}{egg}\right]\left[\frac{egg}{worm/time}\right].worm.host}{host} - worm.\frac{1}{time} - worm.\frac{host}{worm/time}.\frac{worm^2}{\frac{worm}{host}.host^2} \qquad [4.42]$$

4.2.6.4 System of hybridized equations for host – parasite dynamics

The three populations represented in equation [4.19] are now simplified to two equations given by [4.42]. These will be translated to STELLA® for further analysis and the results presented in Section 4.3 below.

$$\frac{dH}{dt} = rH - d_{h,p}.P$$
$$\frac{dP}{dt} = \frac{d_1 d_2 \lambda PH}{D+H} - P\left(d_{h,n} + d_{p,n} + \frac{P.d_{h,p}}{H}\right) - P.d_{h,p}\left(\frac{P^2}{kH^2}\right) \quad [4.43]$$

4.2.7 Population dynamics in terms of mean worm burden, M

Epidemiological interventions are interested in determining and reducing parasite reproduction, infection transmission, average worm burden, and ultimately disease incidence and prevalence in the entire human population. In the above discussion the host – parasite dynamics were represented by the absolute values of population members, total host (H) and parasite (P). In reality, one cannot determine the total number of worms in the host population without treating everyone to induce parasite expulsion. Instead a sample of host is usually chosen and their worm burden determined (usually indirectly by counting the number of eggs in the host's feces). From this, the average parasite prevalence, given an assumed probability distribution (say the negative binomial), is ascertained and the appropriate steps are then taken. For a chemotherapy intervention, these steps include choosing the type of mass treatment strategy, target population, medication delivery frequency and time period, and the proportion of persons to receive medication at each treatment. These decisions are therefore best made in terms of the host population's mean worm burden (M). Expressing equation [4.19] in terms of (P/H) gives:

$$\frac{dP}{dt} = d_1\beta WH - H.d_{h,n}\sum_{i=0}^{\infty} i.p(i) - P\sum_{i=0}^{\infty} d_{h,p}(i).i.p(i) - d_{p,n}.P$$

$$\frac{d\left(\frac{P}{H}\right)}{dt} = d_1\beta W - d_{h,n}\sum_{i=0}^{\infty} i.p(i) - \frac{P}{H}\sum_{i=0}^{\infty} d_{h,p}(i).i.p(i) - d_{p,n}.\frac{P}{H} \quad [4.44]$$

From the above assumptions, equation [4.43] can be rewritten as:

$$\frac{dM}{dt} = d_1\beta W - d_{h,n}\mathrm{E_t}(i) - M.d_{h,p}\mathrm{E_t}(i^2) - d_{p,n}.M$$

$$\frac{dM}{dt} = d_1\beta W - M(d_{h,n} + d_{p,n}) - M.d_{h,p}\mathrm{E_t}(i^2) \quad [4.45]$$

Setting the egg population equation from [4.18] to zero, assuming the negative binomial distribution and solving for (W) gives:

$$\frac{dW}{dt} = d_2.\omega_f.\Phi\,(M,k).H\sum_{i=0}^{\infty}\lambda(i).i.p(i) - \gamma W - \beta WH = 0$$

$$W = \frac{d_2.\omega_f\Phi(M,k).H.\sum_{i=0}^{\infty}\lambda(i).i.p(i)}{\gamma + \beta H} \quad [4.46]$$

It is common to assume a 1:1 sex ratio, so ($\omega_f = \frac{1}{2}$) (Croll *et al.*, 1982). Substituting in equation [4.45] for $\sum_{i=0}^{\infty} i.p(i)$ with (M) and $\lambda(i)$ with $\lambda(i) = \lambda_0\, e^{-\theta i}$, where ($\theta$) is a measure of the density – dependent constraint on reproduction and (λ_0) is the maximum eggs production without those constraints gives(Anderson, 1982):

$$W = \frac{\frac{1}{2}.d_2\Phi(M,k).H.M.\lambda_0\, e^{-\theta i}}{\gamma + \beta H} \quad [4.47]$$

Substituting equation [4.46] into equation [4.44] gives:

$$\frac{dM}{dt} = d_1\beta\frac{\frac{1}{2}.d_2.\Phi(M,k).H.M.\lambda_0\, e^{-\theta i}}{\gamma + \beta H} - (d_{h,n} + d_{p,n})M - Md_{h,p}\mathrm{E_t}(i^2) \quad [4.48]$$

Substituting $\mathrm{D} = \frac{\gamma}{\beta}$ as in equation [4.33] above give:

$$\frac{dM}{dt} = d_1 \frac{\frac{1}{2}.d_2.\Phi(M,k).H.M.\lambda_0\, e^{-\theta i}}{D+H} - (d_{h,n} + d_{p,n})M - Md_{h,p}\mathrm{E_t}(i^2) \quad [4.49]$$

Ascaris worms are dioecious and polygamous, therefore, the likelihood of worms in a given host mating to produce fertilized eggs, denoted by the mating function ($\Phi(M,k)$), depends on the number of worms (i) present and is its probability distribution $p(i)$ (Anderson and May, 1992). Assuming negative binomial distribution:

$$\Phi\,(M,k) = 1 - \left(1 + \frac{M}{2k}\right)^{-(1+k)} \quad [4.50]$$

Substituting equations [4.49] and $\mathrm{E_t}(i^2) \equiv M + \frac{M^2}{k}$into [4.47] gives equation [4.50]:

$$\frac{dM}{dt} = \frac{\frac{1}{2}.d_2.d_1.\left[1 - \left(1 + \frac{M}{2k}\right)^{-(1+k)}\right].H.M.\lambda_0\, e^{-\theta i}}{D+H} - (d_{h,n} + d_{p,n})M - Md_{h,p}\left[\frac{M^2}{k} + M\right] \quad [4.51]$$

In terms of units:

$$\frac{worm}{host/time} = \frac{\left[\frac{egg}{egg}\right]\left[\frac{worm}{egg}\right].host.\left[\frac{worm}{host}\right].\left[\frac{egg}{worm/time}\right]}{host} - \frac{1}{time}.\frac{worm}{host} - \frac{worm}{host}.\frac{host}{worm/time}.\left[\frac{\left(\frac{worm}{host}\right)^2}{\frac{worm}{host}} + \frac{worm}{host}\right] \quad [4.52]$$

4.2.7.1 Basic reproductive rate

Ascaris has a complex lifecycle with many distinct developmental stages and by extension many population determining rate processes. The overall aim of any interventions is to somehow reduce the reproductive or transmission potential of the

parasite. For example, a nutrition program may increase the host's immunity to invading parasites, which lowers the number of established worm which subsequently reduces egg production and ultimately the rate of infection. In the same way, mass chemotherapy, remove the adult worm population and ceases egg production, at least temporarily. The basic reproductive rate (R_0) captures all these reproductive and transmission processes into one parameter and is defined as the expected number of sexually mature female offsprings that one female will produce in her lifetime in the absence of density – dependent constraints on the infrapopulation (Anderson, 1985; Thomas and Weber, 2001).

For a fertilized female *Ascaris* worm, (R_0) is a function of the net output of transmission stages which depends on her fecundity (λ) and the array of developmental and death processes the offsprings are subjected to. For example, only a proportion, (d_2) of produced eggs are embryonated upon exit from the host and are able to survive the 2 – 3 week development in the environment before they become infective. The rate of ingestion is a function of the rate of infective egg mortality (γ) and their rate of contact with the host population (β). Once ingested, again only a portion of the larvae, (d_1) are able to withstand the host's immunological defenses to make it back to the small intestine. While in the intestine, the worms are subjected to various density – independent death processes such as dying of "natural" causes ($d_{p,n}$), and dying when the host dies of other causes except parasite induced ($d_{h,n}$). Rearranging equation [4.50] gives:

$$\frac{dM}{dt} = (d_{h,n} + d_{p,n})M\left[\frac{\frac{1}{2}.d_2.d_1.H.\left[1-\left(1+\frac{M}{2k}\right)^{-(1+k)}\right].\lambda_0}{(d_{h,n}+d_{p,n})\,(D+H)} - 1 - \frac{d_{h,p}}{(d_{h,n}+d_{p,n})}\left[\frac{M^2}{k}+M\right]\right] \quad [4.53]$$

The basic reproductive rate is therefore given by:

$$R_0 = \frac{\frac{1}{2}.d_2.d_1.H.\left[1-\left(1+\frac{M}{2k}\right)^{-(1+k)}\right].\lambda_0}{(d_{h,n}+d_{p,n})\,(D+H)} \quad [4.54]$$

Substituting equation [4.53] into [4.52] gives:

$$\frac{dM}{dt} = M\left[(d_{h,n}+d_{p,n})(R_0-1) - d_{h,p}\left[\frac{M^2}{k}+M\right]\right] \quad [4.55]$$

In practice, the basis reproductive rate is used as a measure of parasite stability in the host community. That is, when $R_0 = 1$, each female worm replaces itself in the next generation and the parasite is said to be endemic (Thein – Hlaing *et al.*, 1991). Below this threshold, the organism is unable to maintain itself and is subsequently eradicated. In the field, (R_0) is usually approximated using models similar to equation [4.54] and estimates of the required variables (e.g.M, k) obtained as a result of mass chemotherapy (Anderson and May, 1992). (R_0) is therefore a very useful bench mark to measure an intervention's success and will be adopted for this work.

4.2.8 Control by chemotherapy

Table 4.12: Nomenclature and definitions used in Section 4.2.8

Reference Symbol	Description	Units
c	Excessive worm deaths due to chemotherapy	1/time
g	Number of community member treated at each application	host/time
h	Cure rate of drug per dose; proportion of worms expelled	worm/worm/host
R_o	Basic reproductive rate	[ ]

A chemotherapeutic intervention is the administration of medication to expel the adult life stages of the parasite from the human hosts. There are three main types; mass treatment (random application to a proportion or all community members), targeted treatment (administration to a specific group such as school aged children) and selective treatment (say to individuals with high fecal egg count) (Anderson, 1989). Due to the availability of increasingly effective, cheap and safe drugs, this is one of the most widely employed method of controlling parasitic infections (Anderson and May, 1985). In addition, it is the quickest method of preventing and reducing morbidity associated with helminth infections and has been recognized by the World Health Assembly who recommended frequent treatment of school – aged children (Keiser and Utzinger, 2008). The following sections will consider interventions that subscribe to mass treatment where at each administration the drug is given to a group of randomly selected individuals from among community members. The total number of worms expelled, (c) is given by (Anderson and May, 1992):

$$c = -\ln(1 - gh) \qquad [4.56]$$

Where (g) is the number of persons treated per treatment interval and (h) is the drug efficacy. The proportion of worms expelled in a single treatment for four of the most common drugs used to treat soil transmitted helminthes are listed in Table 4.13:

Table 4.13: Proportion of host's worm burden kill by drug in a single treatment (Keiser and Utzinger, 2008)

Drug	Cure rate, h (%/host)
Albendazole (400mg)	93.9
Mebendazole (500mg)	96.5
Pyrantel pamoate(10mg/kg)	87.9
Levamisole (2.5mg/kg)	91.5

Including this new worm death rate into equation [4.50] gives:

$$\frac{dM}{dt} = \frac{\frac{1}{2}.d_2.d_1.\left[1-\left(1+\frac{M}{2k}\right)^{-(1+k)}\right].H.M.\lambda_0\, e^{-\theta i}}{D+H} - (d_{h,n}+d_{p,n})M - cM - Md_{h,p}\left[\frac{M^2}{k}+M\right] \quad [4.57]$$

Rearranging as before to obtain a form of the basic reproductive rate R_o:

$$\frac{dM}{dt} = \frac{\frac{1}{2}.d_2.d_1.\left[1-\left(1+\frac{M}{2k}\right)^{-(1+k)}\right].H.M.\lambda_0\, e^{-\theta i}}{D+H} - (d_{h,n}+d_{p,n}+c)M - Md_{h,p}\left[\frac{M^2}{k}+M\right] \quad [4.58]$$

Let $\tilde{R}$ be a new basic reproductive rate in terms the excess worm deaths, c:

$$\tilde{R} = \frac{\frac{1}{2}.d_2.d_1.\left[1-\left(1+\frac{M}{2k}\right)^{-(1+k)}\right].H.M.\lambda_0\, e^{-\theta i}}{(d_{h,n}+d_{p,n}+c)(D+H)} \quad [4.59]$$

Then equation [4.58] becomes:

$$\frac{dM}{dt} = M\left[(d_{h,n}+d_{p,n}+c)\left(\tilde{R}-1\right) - d_{h,p}\left[\frac{M^2}{k}+M\right]\right] \quad [4.60]$$

As before for the parasite to be eradicated $\tilde{R} < 1$

$$\tilde{R} = \frac{\frac{1}{2}.d_2.d_1.\left[1-\left(1+\frac{M}{2k}\right)^{-(1+k)}\right].H.M.\lambda_0\, e^{-\theta i}}{(d_{h,n}+d_{p,n}+c)(D+H)} < 1$$

$$\frac{\frac{1}{2}.d_2.d_1.\left[1-\left(1+\frac{M}{2k}\right)^{-(1+k)}\right].H.M.\lambda_0\, e^{-\theta i}}{(D+H)} < (d_{h,n}+d_{p,n})+c \qquad [4.61]$$

Rearranging in terms of c gives:

$$\frac{\frac{1}{2}.d_2.d_1.\left[1-\left(1+\frac{M}{2k}\right)^{-(1+k)}\right].H.M.\lambda_0\, e^{-\theta i}}{(d_{h,n}+d_{p,n})(D+H)} < 1+\frac{c}{(d_{h,n}+d_{p,n})} \qquad [4.62]$$

The left hand side is actually (R_o), therefore the number of worms expelled during chemotherapy must be greater than a critical number for eradication to occur:

$$c > (R_o-1)(d_{h,n}+d_{p,n}) \qquad [4.63]$$

Therefore, the critical proportion of persons that must be treated at each treatment interval is obtained by solving for (g) in equation [4.55] and substituting for (c) from equation [4.63] to give:

$$g_c = \frac{1-e^{-c}}{h}$$

$$g_c = \frac{1-e^{-(R_o-1)(d_{h,n}+d_{p,n})}}{h} \qquad [4.64]$$

Another important epidemiological parameter is the disease prevalence, number of persons infected with worms in the community. For the negative binomial distribution, disease prevalence (p_d) is given by (Guyatt *et al.*, 1990):

$$p_d = 1-\left(1+\frac{M}{k}\right)^{-k} \qquad [4.65]$$

4.3 Dynamical modeling in STELLA®

4.3.1 Step 1: Reproducing host – parasite trajectories from literature

The first stage of the modeling process was to reproduce the trajectories from (Anderson and May, 1978) and compare the results obtained after translating into STELLA®. Table 4.14 and Table 4.15 give the initial population and parameter values obtained from Figure 4 of the article. Figure 4.2 and Figure 4.3 illustrate the STELLA® representation of the population equations given by [4.65] and [4.66] respectively. These equations are equivalent to equations (7) and (13) (Anderson and May, 1978) but rewritten in terms of the notations used in this work, the corresponding symbols used by those authors are also given in the tables. The results and discussion of this first step is given in the subsection following.

Table 4.14: Population parameters for host model (Anderson and May, 1978)

Description	Symbol	Value	Units	Reference symbol
Hosts	H	100	host	H
Parasites	P	200	worm	P
Host birth rate	a	3.0	host/host/time	a
Host natural death rate	$d_{h,n}$	1.0	host/host/time	b
Host mortality rate due to parasite induced death	$d_{h,p}$	0.5	host/worm/time	α

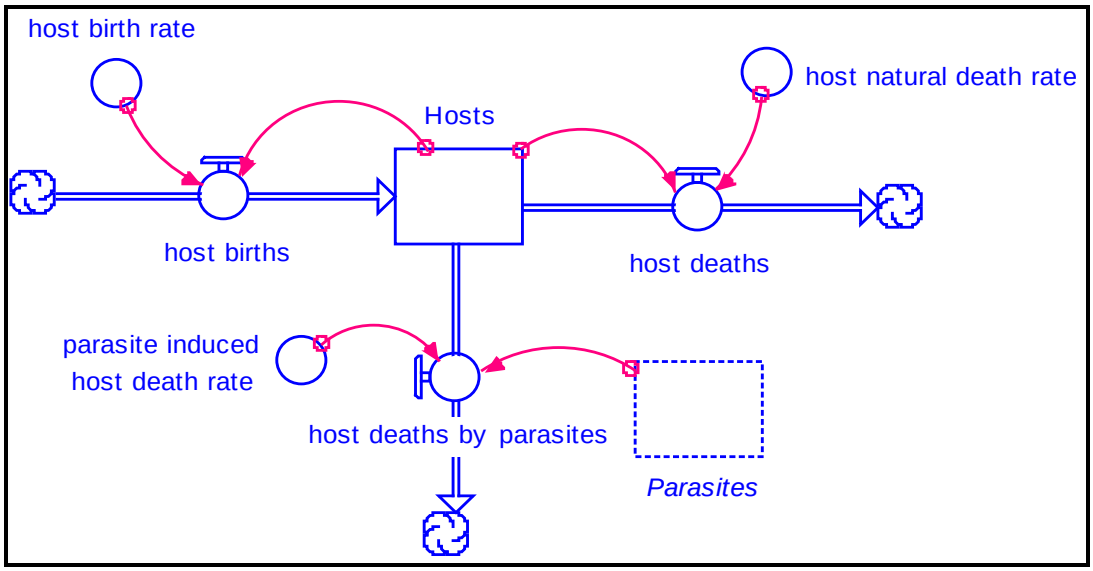

Figure 4.2: STELLA® representation of host's equation

$$\frac{dH}{dt} = aH - d_{h,n}H - d_{h,p}.P \qquad [4.66]$$

Table 4.15: Population parameters for parasite equation (Anderson and May, 1978)

Description	Symbol	Value	Units	Reference
Parasites	P	200	worm	P
Egg production rate by adult worms	λ	6.0	egg/egg/time	λ
Parasite carrying capacity	$\frac{d_{h,p}P^2}{H}\left(\frac{k+1}{k}\right)$	-	worm^2/host/time	$\frac{\alpha P^2}{H}\left(\frac{k+1}{k}\right)$
Clumping parameter	k	2.0	unspecified	k
Parasite natural death rate	$d_{p,n}$	0.1	worm/worm/time	μ
Transmission efficiency	D	10	host	H_o

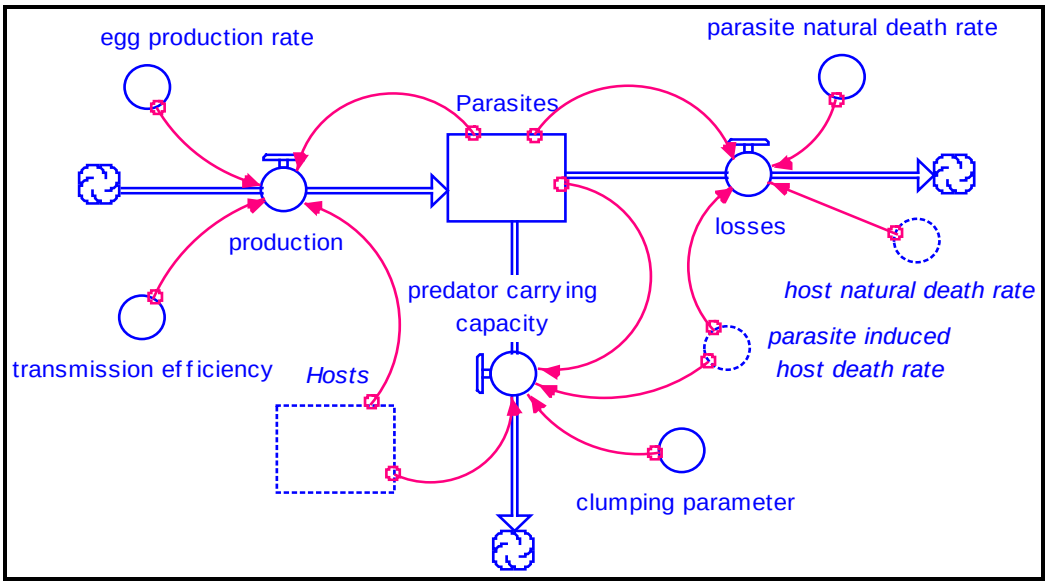

Figure 4.3: STELLA® representation of worm's equation

$$\frac{dP}{dt} = \frac{\lambda PH}{D+H} - P\left(d_h + d_p + d_{h,p}\right) - d_{h,p}\frac{P^2}{H}\left(\frac{k+1}{k}\right) \qquad [4.67]$$

4.3.1.1 Step 1 results and discussion: reproducing trajectories from literature

The STELLA® output compared well with the graph presented in the article with defining features such as the characteristic oscillations in the populations occurring in similar locations. Minor differences, such as the value of the maximums might be due to the fact that the initial values were estimated as they were not explicitly stated by the authors and could have been different from those used in their work. An interesting finding was that while host and parasite maximums occurred simultaneously, the maximum parasite burden occurred a time step later, see Table 4.16 and Figure 4.4 below.

Table 4.16: STELLA® output population values for host – parasite equation from Anderson and May (1978)

Time (years)	Hosts	Parasites	Mean parasite burden
0	100.00	210.00	2.10
1	108.27	606.44	5.60
2	37.61	218.73	5.82
3	24.82	98.49	3.97
4	30.20	105.06	3.48
5	35.98	139.56	3.88
6	34.81	145.51	4.18
7	32.24	131.77	4.09
8	32.05	126.77	3.96
9	32.90	130.22	3.96
10	33.17	132.91	4.01
11	32.92	132.23	4.02
12	32.76	131.07	4.00
13	32.81	131.05	3.99
14	32.88	131.47	4.00
15	32.88	131.58	4.00

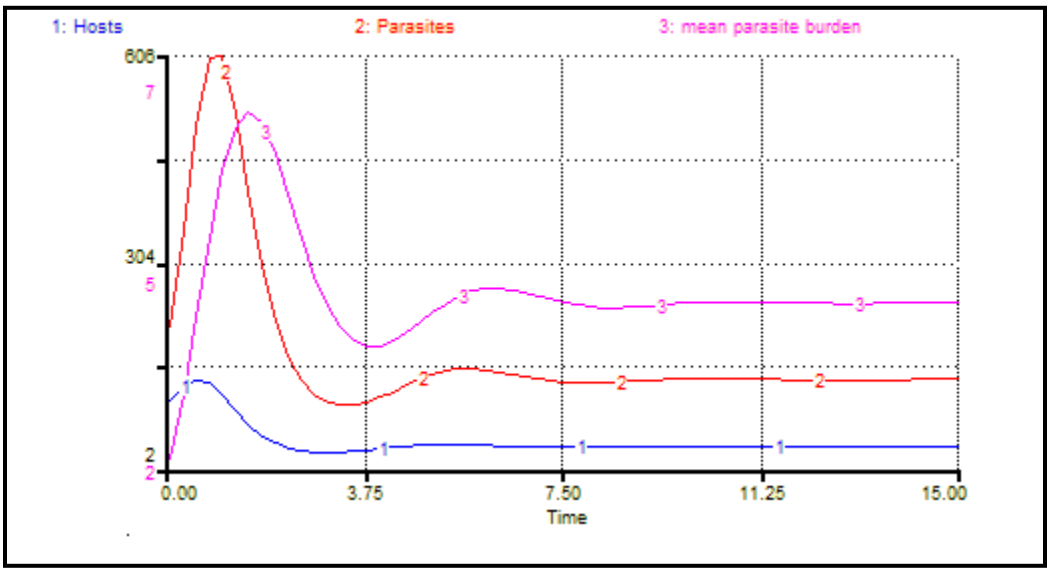

Figure 4.4: STELLA® reproduction of Figure 4 from Anderson and May (1978) with population mean added

4.3.2 Determining conditions for parasite dynamics in Paquila

The next step was to model the study population using the system of equations developed in [4.42]. Once established, what – if scenarios were conducted to determine

the effects of varying variables that represent key parasite population processes on worm burden and disease prevalence in the host community. The values chosen for the variables were either taken from literature in similar study sites or are values known to be true for Paquila through personal communications with Dianne and Jim Thompson; missionaries in the village. For those values taken from articles the appropriate reference is given in the population parameter tables below.

Croll *et al.* (1982) found that the average worm burden for an agricultural village similar to Paquila had a mean worm burden (M) of 22 worms/host. For this exercise, a mean worm burden of 20 worms/host was chosen instead to mimic the 2:1 parasite to host ratio from the Step 1, resulting in an initial parasite population of 7000. The host birth and death rates were estimated from the country's population values. There is some concern for committing an ecological fallacy (applying global results to local level), however, it could be argued that since the majority of the population lived in rural areas, these population rates are weighted towards those groups of persons (CIA, 2008; Oleckno, 2002). In lieu of actual values for pathogencity of *Ascaris*, the parasite induced host death rate was used for hookworm, another soil transmitted helminth (Anderson, 1980b).

The fecundity of the female *Ascaris* worm is legendary with proposed average daily production of up to 200,000 eggs (Brown and Cort, 1927; Jungersen *et al.*, 2000). However, these values were obtained from only two cases and without differentiating the fertilized status of the eggs (Brown and Cort, 1927). Fertilized eggs are more epidemiologically important and a tremendous amount of the eggs that exit the host are unfertilized (Peng *et al.*, 2003). Thus for this model a conservative value of 20 fertilized eggs per day per female worm was chosen, which is reasonable since the average person comes in contact (ingests) 9 – 20 infective eggs annually (Wong *et al.*, 1991).

Anderson and Gordon (1982) define the transmission efficiency as the number of newly – born cohort in host population, as such the number of live births in this community was used to estimate (D) for this study. This is a reasonable assumption since in disease endemic areas infection is recycled continually with new incidence occurring only within newborns.

Table 4.17: Host population parameters for Paquila

Description	Symbol	Value	Units	Reference
Hosts	H	3500	host	
Parasites	P	7000	worm	(Croll *et al.*, 1982)
Host birth rate	a	29/1000	host/host/year	(Cia, 2008)*
Host natural death rate	$d_{h,n}$	5.27/1000	host/host/year	(Cia, 2008)*
Host mortality rate due to parasite induced death	$d_{h,p}$	5 e^{-05}	host/worm/year	(Anderson, 1980b)

*This value is that for country of Guatemala

$$\frac{dH}{dt} = rH - d_{h,p}.P$$

$$\frac{dP}{dt} = \frac{d_1 d_2 \lambda PH}{D+H} - P\left(d_{h,n} + d_{p,n} + \frac{P.d_{h,p}}{H}\right) - P.d_{h,p}\left(\frac{P^2}{kH^2}\right) \quad [4.43]$$

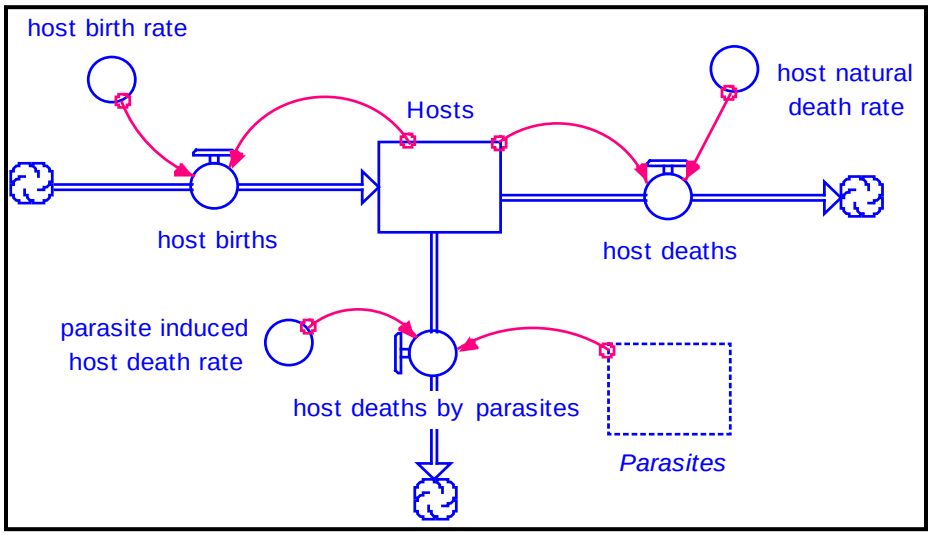

Figure 4.5: STELLA® representation of host's equation for Paquila (equation [4.43])

Table 4.18: Parasite population parameter for Paquila

Description	Symbol	Value	Units	Reference
Parasites	P	7000	worm	
Fertilized egg production rate by adult female worms	λ	7300	egg/egg/year	
Parasite natural death rate	$d_{p,n}$	1.0	worm/worm/year	(Croll *et al.*, 1982)
Transmission efficiency	D	100	host	(Anderson and Gordon, 1982)*
Parasite carrying capacity	$\frac{d_{h,p}P^2}{kH}$	-	worm/year	
Egg survival	d_1	0.01	egg/egg	(Larsen and Roepstorff, 1999)
Egg hatching	d_2	0.02	worm/egg	(Wong *et al.*, 1991)
Egg production transmission	$d_1 d_2 \lambda$	-	1/year	
Saturation	$\frac{H}{D+H}$	-		

*These authors define transmission efficiency as newly born cohort of host

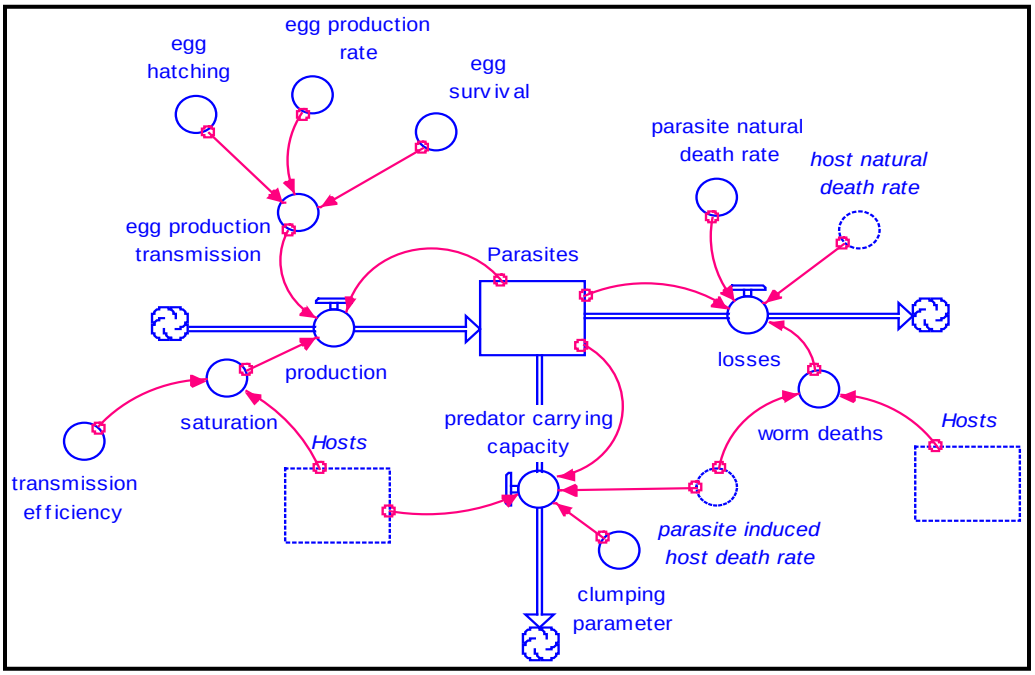

Figure 4.6: STELLA® representation of parasite's equation for Paquila (equation [4.43])

4.3.2.1 Step 2 results and discussion: host – parasite dynamics

A model similar to the host – parasite model simulated in Step 1 above was developed for the village of Paquila as shown in Figure 4.5 and Figure 4.6. However this was based on the system of equations listed in equation [4.43] using the default values presented in Table 4.17 and Table 4.18. The result is given in Figure 4.7. There is some parasite regulation of host population but not with the severity seen in the article by Anderson and May (1978). This is in part due to the much lower parasite induced host death rate seen in human populations compared to smaller species that the article modeled.

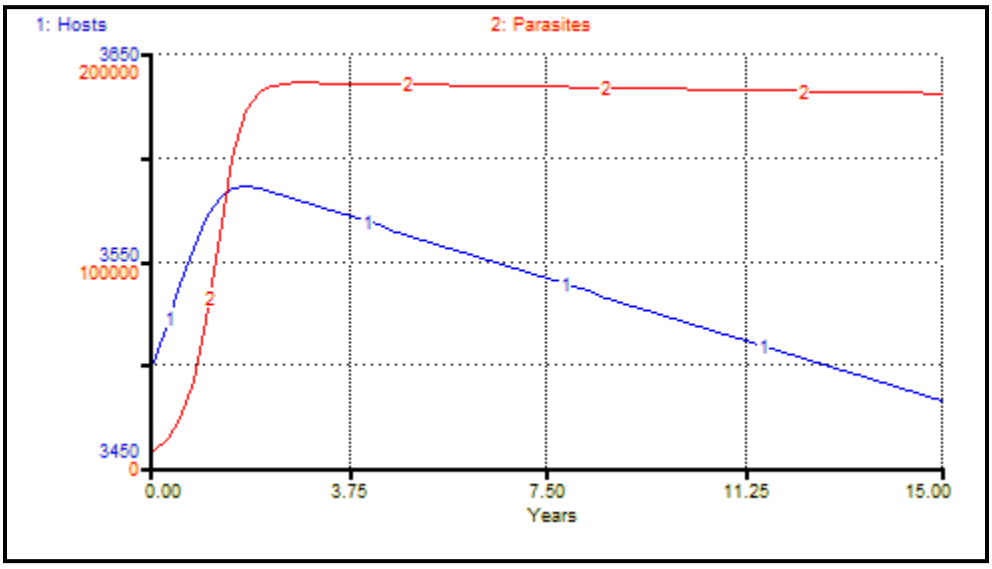

Figure 4.7: Host – parasite dynamics for Paquila

4.3.2.2 Step 2 results and discussion: varying egg survival, d_2

Under moist shady conditions *Ascaris* eggs are known to survive at least 7 years in the soil with a maximum of up to 15 years reported (Black *et al.*, 1982). However, on average only about 1% survive the developmental period to become infective, the majority being inactivated by sunlight or desiccated due to high temperatures (Larsen

and Roepstorff, 1999). The model was run for varying egg survival rates to determine the response of the mean worm burden of the host population.

From Table 4.20 and Figure 4.7 a mere 10% increase in the deactivation rate (or 10% decrease in egg survival) decreases the potential maximum mean worm burden from 44 to 9 worms/host at year 15. A further 10% decrease reduced the worm intensity to 50% of what it was at the beginning of the simulation. This simulation is mimicking what occurs in a sanitation treatment system such as a Solar Latrine which will be explored in Chapter 6. The following are of note; the nonlinearity of the responses (small changes can create big results), results occur over time, and changing one variable may not be enough to eradicate parasite sustainably from community.

Table 4.19: Mean worm burden of Paquila in response to varying egg survival rates

Time (years)	Mean worm burden $d_2 = 0.008$	Mean worm burden $d_2 = 0.009$	Mean worm burden $d_2 = 0.01$
0	2.00	2.00	2.00
1	1.92	2.21	2.54
2	1.84	2.44	3.24
3	1.76	2.69	4.12
4	1.69	2.98	5.25
5	1.62	3.30	6.68
6	1.56	3.65	8.49
7	1.50	4.04	10.78
8	1.44	4.48	13.62
9	1.39	4.97	17.08
10	1.34	5.51	21.21
11	1.29	6.11	25.91
12	1.24	6.77	30.98
13	1.20	7.51	36.05
14	1.16	8.32	40.70
15	1.12	9.21	44.60

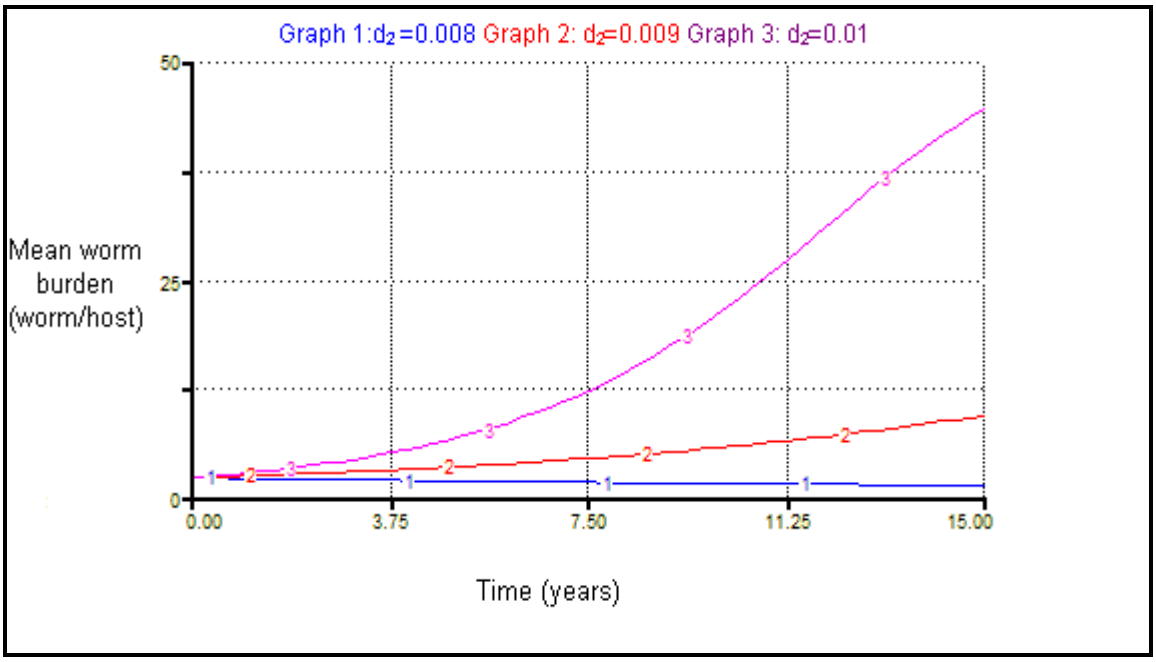

Figure 4.8: Mean worm burden of Paquila in response to varying egg survival rates

4.3.2.3 Step 2 results and discussion: varying worm natural death rate, $d_{p,n}$

The adult worms of *Ascaris* are very long lived with life spans up to 2 years with 1 being the average (Crompton, 2001). The model was run for 25% and 50% decreases in worms' average life expectancy. The latter simulation showed that it is possible to eradicate the parasites (mean worm burden < 1) in about 7 years, as seen in Table 4.20 and Figure 4.9. As a point of clarification, a mean worm burden of 1 leads to production of unfertilized eggs (if the 1 worm present were female) since at least 2 worms are needed to successfully mate (Churcher *et al.*, 2005). This begs the question, what practical intervention can be sustainably applied for 7 years to achieve this level of success? Increasing the rate at which the adult worms die can be done by fortifying the host's immune system via nutritional supplement (Chapter 5) or through chemotherapy (Section 4.3.3 below).

Table 4.20: Mean worm burden of Paquila in response to varying worm life expectancies

Time (years)	Mean worm burden $d_{p,n} = 1.0$	Mean worm burden $d_{p,n} = 1.25$	Mean worm burden $d_{p,n} = 1.5$
0	2.00	2.00	2.00
1	2.96	2.30	1.79
2	4.37	2.65	1.61
3	6.45	3.06	1.44
4	9.51	3.52	1.30
5	13.94	4.07	1.17
6	20.18	4.69	1.05
7	28.48	5.42	0.95
8	38.38	6.26	0.86
9	48.36	7.23	0.77
10	56.45	8.34	0.70
11	61.76	9.61	0.63
12	64.72	11.06	0.57
13	66.23	12.70	0.52
14	66.97	14.53	0.47
15	67.34	16.56	0.42

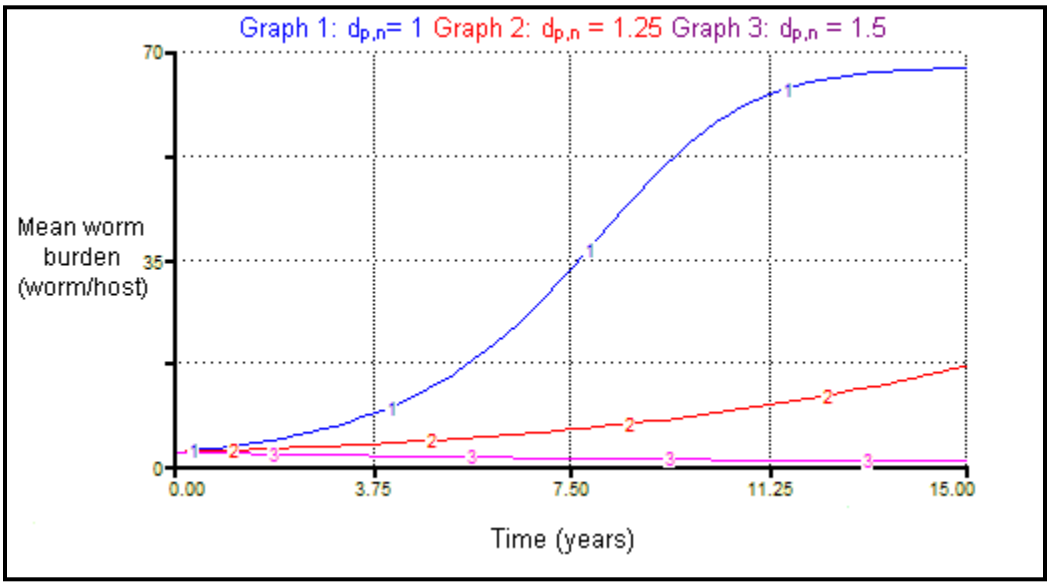

Figure 4.9: Mean worm burden of Paquila in response to varying worm life expectancies

4.3.2.4 Step 2 results and discussion: varying parasite induced host death rate, $d_{h,p}$

Parasite – induced host deaths is a measure of the pathogencity of the worms, that is, the number of worms needed to cause death in an average host. Thus, for the same host population size a small $d_{h,p}$ (say $5\mathrm{e}^{-6}$) means that a large number of worms are required. While for a large $d_{h,p}$ (say $5\mathrm{e}^{-3}$) represents a very lethal parasite. This is illustrated in the results below.

The drastic difference in the mean worm burden at year 15 is predominantly attributable to host population dying (see Table 4.22). All species have an average pathogencity. However, host factors such as compromised immunity (due to nutritional deficiencies) can increase an organism's ability to cause mortality.

Table 4.21: Mean worm burden of Paquila in response to varying parasite pathogencity

Time (years)	Mean worm burden $d_{h,p} = 5\text{e}^{-6}$	Mean worm burden $d_{h,p} = 5\text{e}^{-5}$	Mean worm burden $d_{h,p} = 5\text{e}^{-3}$
0	2.00	2.00	2.00
1	2.54	2.54	2.44
2	3.24	3.24	2.91
3	4.13	4.12	3.40
4	5.27	5.25	3.85
5	6.73	6.68	4.25
6	8.59	8.49	4.56
7	10.98	10.78	4.79
8	14.03	13.62	4.96
9	17.94	17.08	5.06
10	22.91	21.21	5.13
11	29.22	25.91	5.18
12	37.16	30.98	5.21
13	47.01	36.05	5.22
14	58.97	40.70	5.23
15	73.03	44.60	5.24

Table 4.22: Host population of Paquila in response to varying parasite pathogencity

Time (years)	Host $d_{h,p} = 5\text{e}^{-6}$	Host $d_{h,p} = 5\text{e}^{-5}$	Host $d_{h,p} = 5\text{e}^{-3}$
0	3,500.00	3,500.00	3,500.00
1	3,584.01	3,583.64	3,544.59
2	3,670.02	3,669.17	3,581.53
3	3,758.08	3,756.60	3,610.12
4	3,848.24	3,845.91	3,630.33
5	3,940.53	3,937.09	3,642.85
6	4,035.01	4,030.12	3,648.94
7	4,131.70	4,124.92	3,650.07
8	4,230.65	4,221.41	3,647.64
9	4,331.90	4,319.48	3,642.77
10	4,435.48	4,418.98	3,636.31
11	4,541.40	4,519.78	3,628.85
12	4,649.69	4,621.74	3,620.76
13	4,760.35	4,724.80	3,612.29
14	4,873.38	4,828.97	3,603.61
15	4,988.76	4,934.38	3,594.80

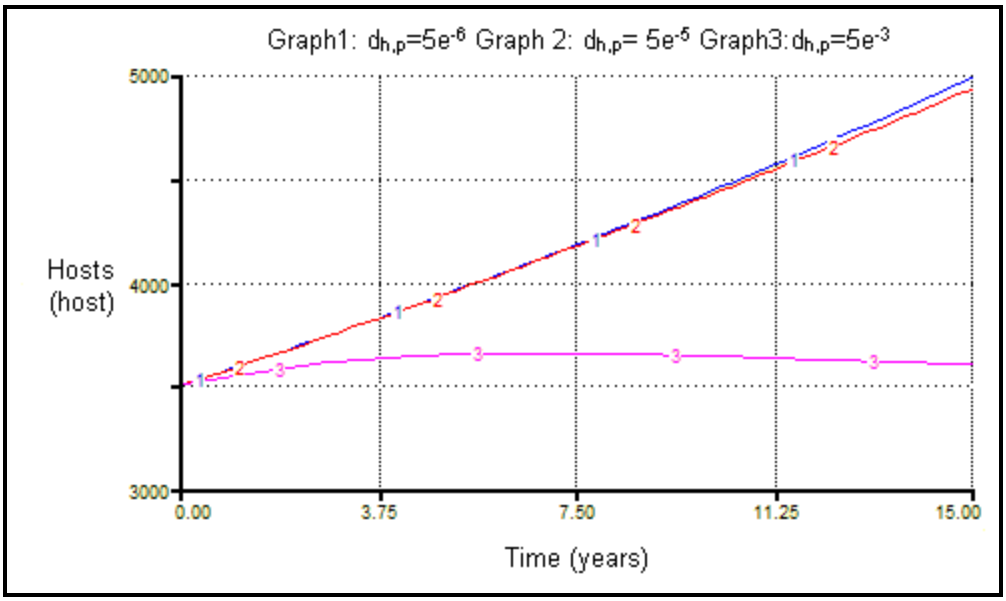

Figure 4.10: Host population of Paquila in response to varying parasite pathogencity

4.3.2.5 Step 2 results and discussion: clumping parameter, k

The clumping parameter represents the degree to which worm numbers are aggregated or clumped in the host population. Compared to viral and bacterial disease, helminth offsprings are not immediately infectious yet are able to persist in communities that have low population densities unlike their pathogenic counterparts (Anderson, 1982). This is in part attributable to their high transmission efficiencies and tendency for a large portion of the worm population to aggregate in a small number of human host, ensuring a continual and abundant supply of infective stages (Macdonald, 1965). This can have unexpected implications for mean worm burden and disease prevalence as is seen from the result of running the model for varying clumping factor.

From the results below, large changes in average worm intensity resulted in very little impact on the actual number of persons infected in the community, that is, there was little impact on disease prevalence in the community. For example, a 75% change in mean worm burden had a corresponding 1% change in disease prevalence. As the

clumping factor becomes larger (more random distribution), large swings in worm intensities resulted in higher changes in prevalence. This has been corroborated by various researchers (Anderson and May, 1992; Croll *et al.*, 1982). Thus, depending on the aggregation of worms among community members, an intervention program may be very successful at reducing morbidity and mortality, but have very little impact on the number of infected persons.

Table 4.23: Mean worm burden and disease prevalence in Paquila for varying clumping parameter, k

Time (years)	Mean worm burden $k = 5.7\mathrm{e}^{-3}$	Prevalence* $k = 5.7\mathrm{e}^{-3}$	Mean worm burden $k = 5.7\mathrm{e}^{-1}$	Prevalence* $k = 5.7\mathrm{e}^{-1}$
0	20.00	0.05	20.00	0.87
1	7.93	0.04	24.37	0.88
2	6.49	0.04	29.15	0.89
3	5.92	0.04	34.03	0.90
4	5.64	0.04	38.64	0.91
5	5.49	0.04	42.65	0.92
6	5.41	0.04	45.86	0.92
7	5.37	0.04	48.26	0.92
8	5.34	0.04	49.95	0.92
9	5.33	0.04	51.10	0.92
10	5.33	0.04	51.88	0.92
11	5.33	0.04	52.40	0.92
12	5.33	0.04	52.75	0.92
13	5.34	0.04	52.99	0.92
14	5.34	0.04	53.16	0.93
15	5.35	0.04	53.30	0.93

*Prevalence was calculated using equation [4.64]

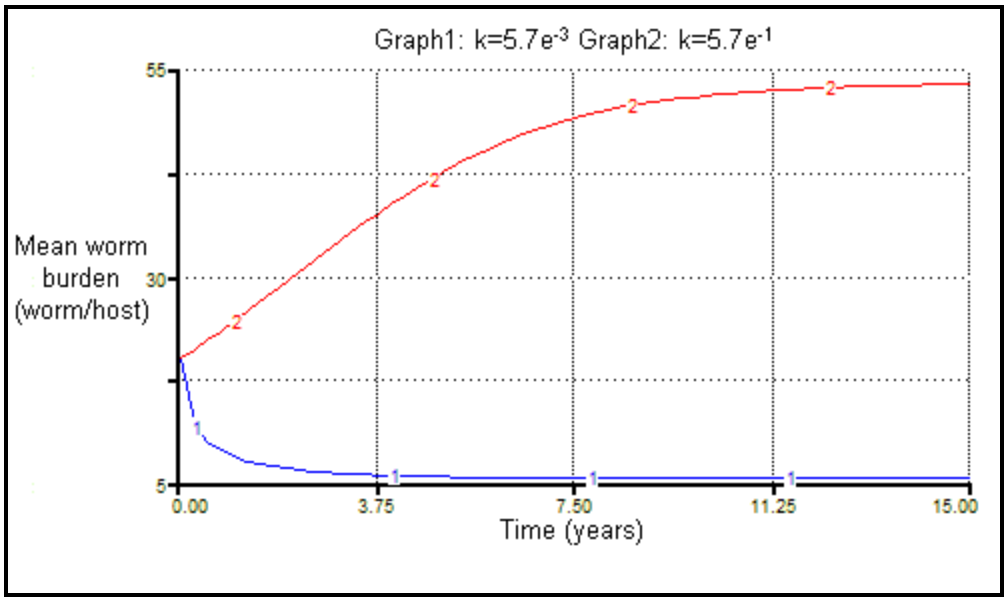

Figure 4.11: Mean worm burden in Paquila for varying clumping parameter, k

4.3.3 Modeling population mean with chemotherapy

From the simulation in Section 4.3.2.3 above reduction in the life expectancy of the adult worms can significantly reduce mean worm burdens. One method of accomplishing this reduction is through administering medication *en masse* to the host population. The resulting population mean was modeled according to equation [4.67]. The model variables as they appear in the STELLA® model are presented in Table 4.24 with their corresponding values and/or equations. The model was first simulated with varying values of the basic reproductive rates. Various what – if scenarios were then conducted by modifying the proportion of persons receiving medication at each treatment interval, the drug cure rates, frequency of treatment and the length of the intervention. The results of the response of the population mean worm burden are presented in the sections below.

$$\frac{dM}{dt} = M(d_{h,n} + d_{p,n})(R_0 - 1) - cM - d_{h,p}M\left[\frac{M^2}{k} + M\right] \qquad [4.68]$$

Table 4.24: Model parameters for the chemotherapy simulation

Description	Symbol	Value	Units	Reference
Population mean	M	20	worm/host	
Basic reproduction rate	R_0	1.5	-	
Ro1	$R_0 - 1$	-	-	
Clumping factor	k	0.57		
Host natural death rate	$d_{h,n}$	5.27/1000	host/host/year	(Cia, 2008)*
Host mortality rate due to parasite induced death	$d_{h,p}$	5 e^{-05}	host/worm/year	(Anderson, 1980b)
Parasite natural death rate	$d_{p,n}$	1	worm/worm/year	
Proportion treated	g	0.27	host/host/time	
Drug efficacy, cure rate	h	-	worm/worm	see Table 4.13
Treatment frequency	tf	4 times	/year	
chemo = IF(TIME < 2) THEN(Population__Mean * PULSE(chemo_rate,0,treatment__frequency)) ELSE(Population__Mean * 0) {worm/host/time}				
Excess death rate due to chemotherapy	c	chemo_rate = - LOGN(1 - drug_efficacy * proportion_treated) {1/time}; equation [4.63]		

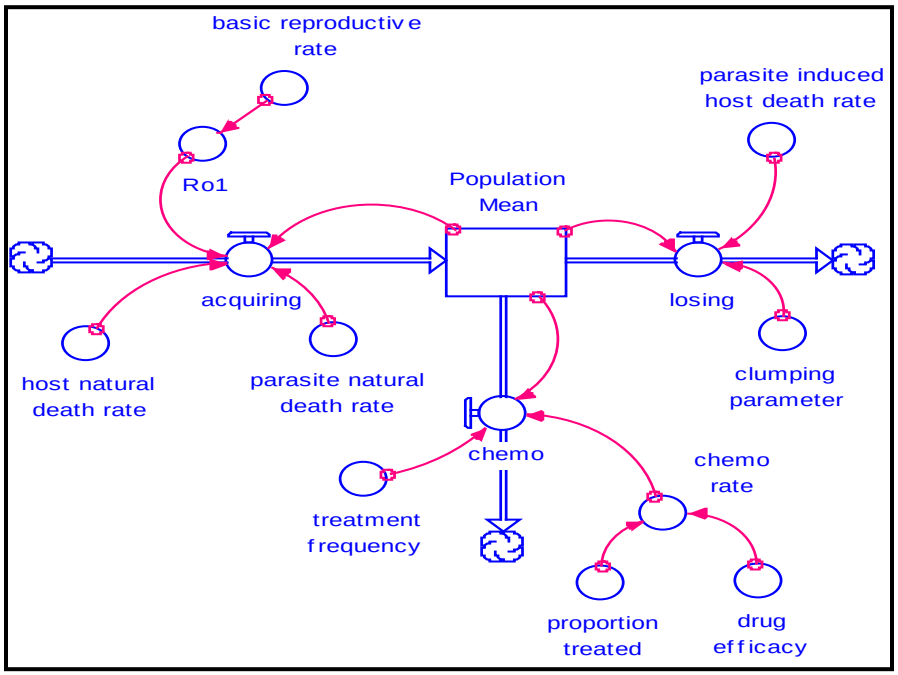

Figure 4.12: STELLA® representation of chemotherapy model for equation [4.68]

4.3.3.1 Step 3 results and discussion: mean worm burden as a function of R_0

As discussed above when the value of R_0 = 1 the parasite is unable to maintain its population and the mean worm burden decreases exponentially as shown in Figure 4.13. It should be noted that this does not occur rapidly (it took 15 years for an average 6 worms/person reduction). This is in part due to the store of infective eggs in the environment. Therefore, chemotherapy and nutrition may be used to reduce (R_0), however if eggs in the environment are not deactivated the disease will persist. A relatively small increase in the worms' basic reproductive rate resulted in a significant increase in the average worm burden (see Figure 4.14).

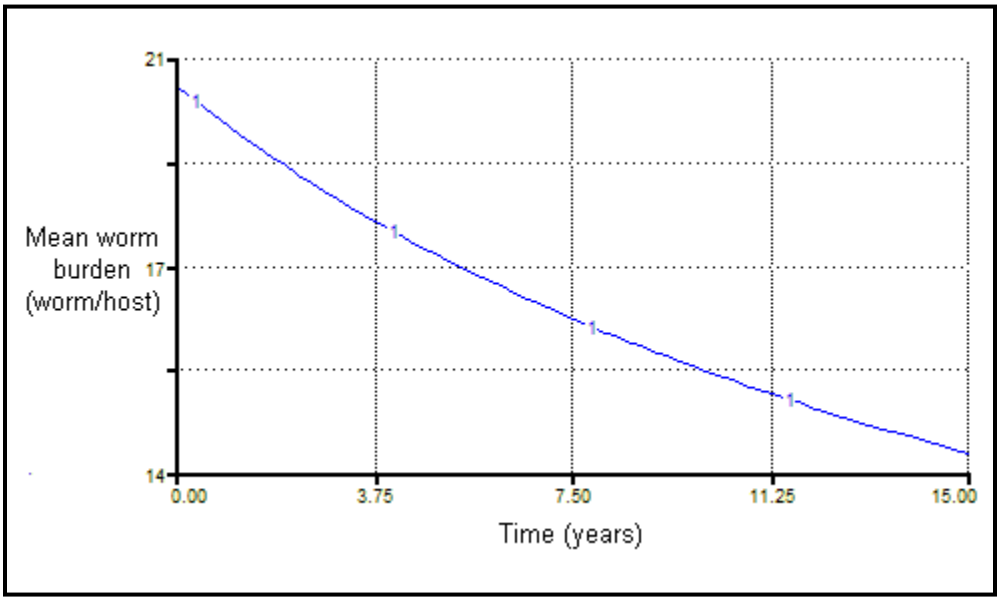

Figure 4.13: Variation of mean worm burden when R_o = 1

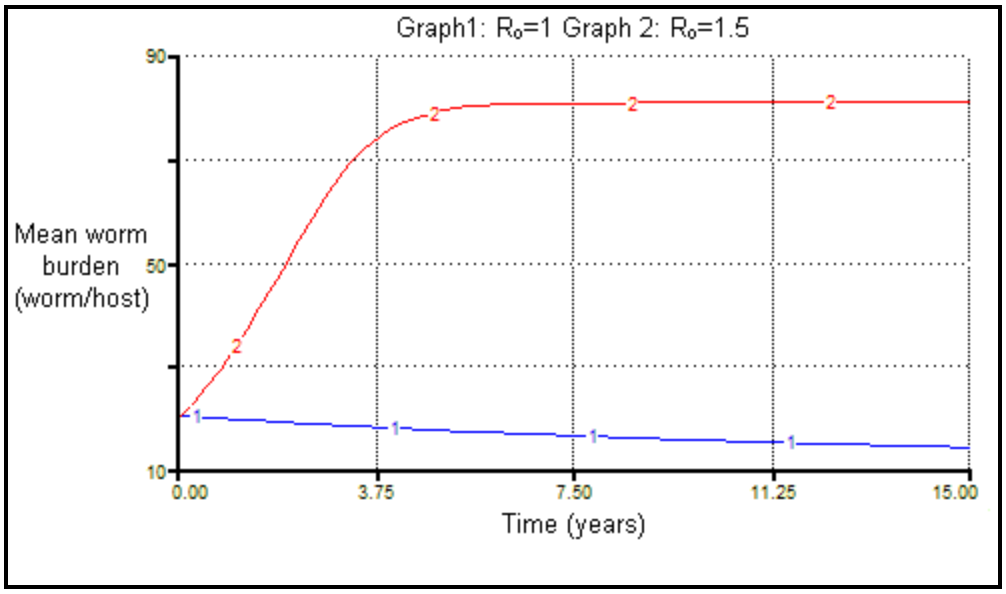

Figure 4.14: Mean worm burden dynamics for different values of R_o

4.3.3.2 Step 3 results and discussion: using different drugs

The average treatment efficacies of four of the most commonly used anti – helminthes are given in Table 4.13. The cure rates range from 88 – 97%. Three runs were made using 88, 93 and 97% for intervention periods of 2 and 5 years with drug administration occurring every 3 months to 27% of community members. Four treatments per year was used as the default interval because the transmission cycle of *Ascaris* form egg production to soil development to infection to sexual maturity requires a minimum of 3 months (WHO, 1967).

For all trials, mean worm burden increased and exceeded pre – control levels after treatment stopped (Figure 4.15). It has been hypothesized that exposure to repeated infection during early life may induce some level of protective immunity, but this is quickly lost when the individual is worm – free such as during anti – helminthic interventions resulting in post – treatment burdens that are greater than endemic levels (O'Lorcain and Holland, 2000).

For the least potent drugs this recovery time is usually equal to the length of the treatment period (2 or 5 years). However, as the efficacy of the drugs increases mean worm burden is suppressed for longer periods. When treatment continued for 2 years and then stopped, the ultimate worm burden at the end of 15 years was the same for all drugs regardless of the cure rate. For longer a treatment period drug efficacy had a more significant effect on the final infection intensity; that is, a 97% kill rate kept reinfection substantially lower relative to other schemes (see Table 4.25). Figure 4.15 shows the dynamics of the mean worm burden if the program were run for all 15 years. Under this scheme the parasite burden decreased below 1 worm/host after about 5 – 6 years for all 3 drugs.

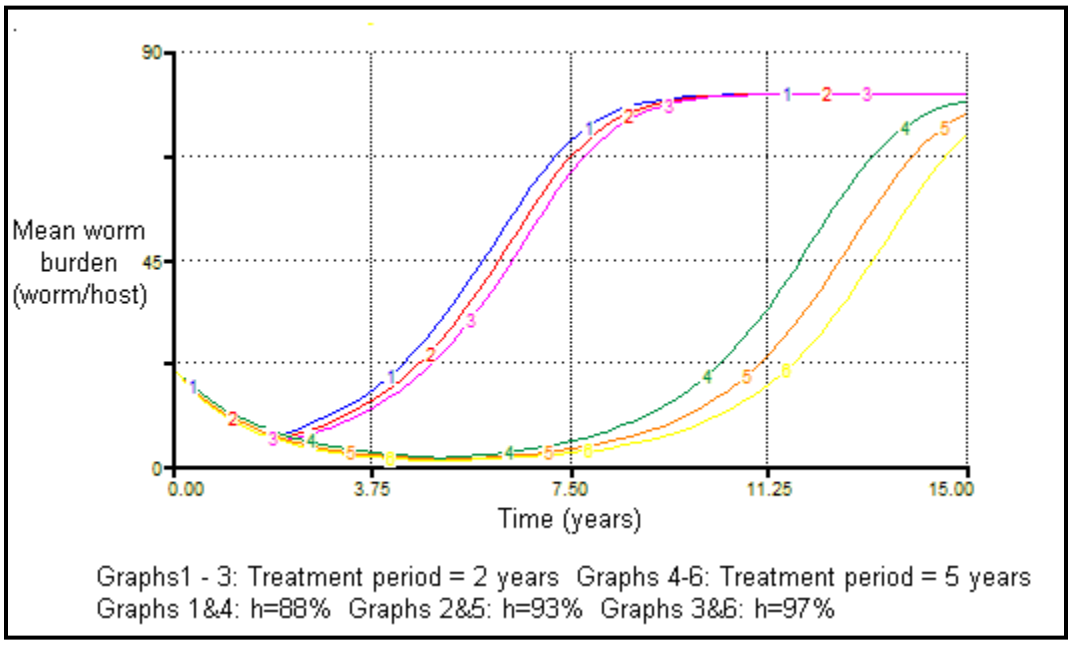

Figure 4.15: Chemotherapy application for treatment periods of 2 and 5 years with different drugs

Table 4.25: Chemotherapy application for treatment periods of 2 and 5 years with different drugs

Time (years)	Mean worm burden $h = 88\%$	Mean worm burden $h = 93\%$	Mean worm burden $h = 97\%$	Mean worm burden $h = 88\%$	Mean worm burden $h = 93\%$	Mean worm burden $h = 97\%$
	Treatment time = 2 years			Treatment time = 5 years		
0	20.00	20.00	20.00	20.00	20.00	20.00
1	11.30	10.41	9.72	11.30	10.41	9.72
2	6.50	5.53	4.83	6.50	5.53	4.83
3	11.10	9.44	8.25	3.77	2.95	2.41
4	18.74	16.01	14.03	2.19	1.58	1.20
5	30.82	26.62	23.50	1.27	0.84	0.60
6	47.44	42.05	37.76	2.18	1.45	1.03
7	64.32	59.61	55.35	3.73	2.48	1.77
8	74.98	72.57	70.03	6.39	4.25	3.03
9	79.17	78.35	77.42	10.91	7.27	5.19
10	80.41	80.19	79.92	18.44	12.39	8.86
11	80.75	80.69	80.62	30.36	20.86	15.05
12	80.83	80.82	80.80	46.86	33.97	25.12
13	80.86	80.85	80.85	63.86	51.18	40.02
14	80.86	80.86	80.86	74.76	67.19	57.65
15	80.86	80.86	80.86	79.09	76.27	71.44

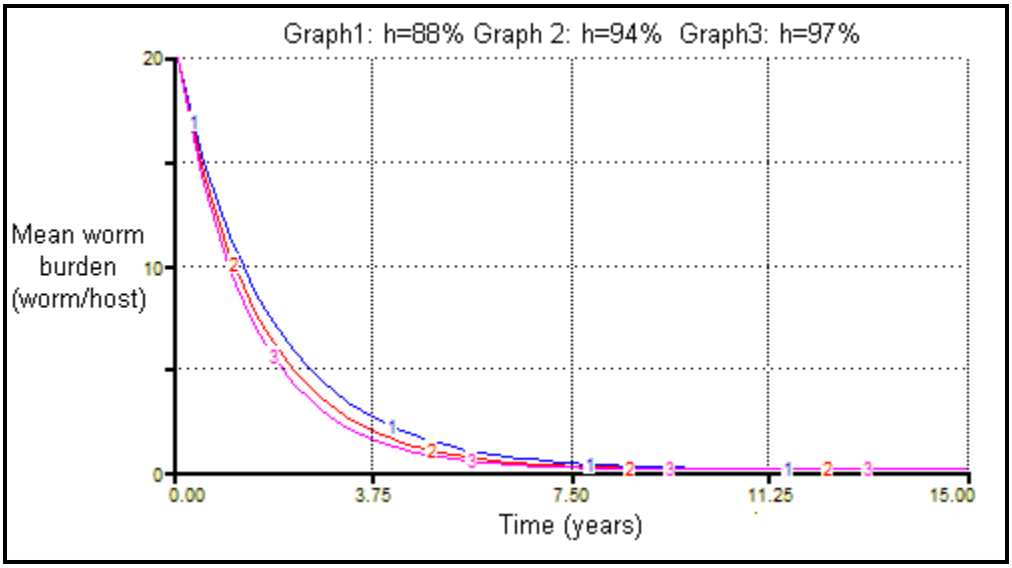

Figure 4.16: Mean worm burden dynamics for treatment period of 15 years using different drugs

4.3.3.3 Step 3 results and discussion: varying proportion treated

The chemotherapy scheme chosen is based on ad hoc random selection individuals at the time of each treatment application. The critical percentage of persons that must be treated in order to eradicate the parasite is given by equation [4.64] above. From that equation the required number of person to be randomly chosen at each treatment are 45, 42 and 41% for medication with cure rates of 88, 93 and 97% respectively. For this simulation 27, 45 and 50% were chosen to be treated for 2 years at 4 treatments per year.

For all simulations the mean increased again after treatment stopped. However the times to re – acquire pre – control levels were different. For example the time taken for the mean to get back to 20 worms/host was 2, 7 and 9 years for proportion treated at 27, 45 and 50%, respectively. Within 2 years the mean worm burden was reduced to less than 1 worms/host when 45 and 50% of the population was treated.

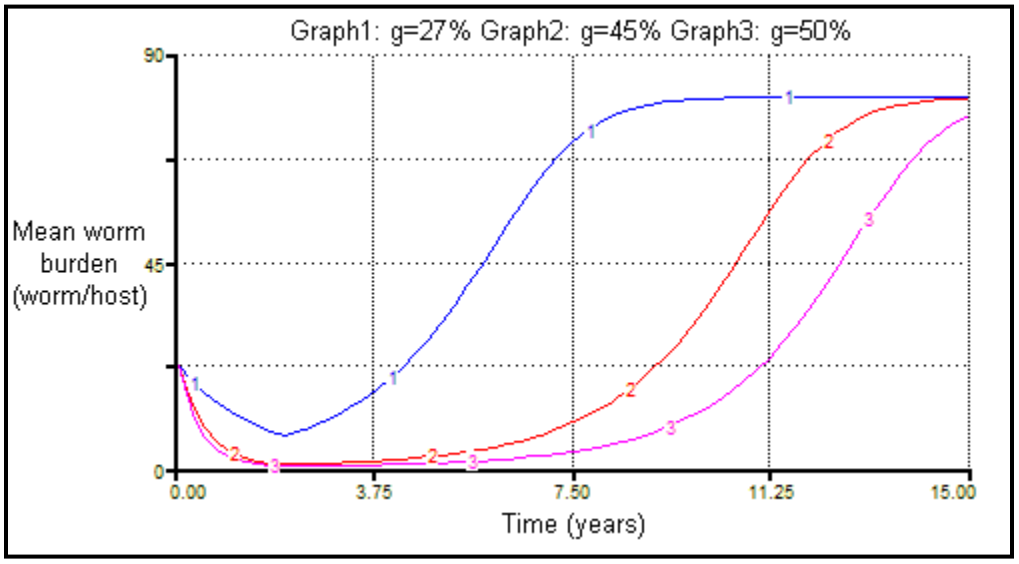

Figure 4.17: Mean worm burden dynamics for varying proportion of population treated

Table 4.26: Mean worm burden dynamics for varying proportion of population treated

Time (years)	Mean worm burden $g = 27\%$	Mean worm burden $g = 45\%$	Mean worm burden $g = 50\%$
0	22.00	22.00	22.00
1	11.96	3.27	1.91
2	6.66	0.50	0.17
3	11.36	0.86	0.29
4	19.17	1.47	0.50
5	31.47	2.52	0.86
6	48.22	4.31	1.47
7	64.95	7.38	2.53
8	75.27	12.57	4.33
9	79.26	21.15	7.41
10	80.44	34.40	12.62
11	80.75	51.67	21.22
12	80.84	67.55	34.51
13	80.86	76.42	51.79
14	80.86	79.62	67.63
15	80.86	80.54	76.45

4.3.3.4 Step 3 results and discussion: varying treatment length and frequency

In previous simulations the default number of treatments was taken as every 3 months (4 times per year). Fallah *et al.* (2002) recommended intervals of 2 months but cautioned that drug resistance and inability to sustainably implement such a strategy on a large scale may lead to failure, compromising instead with every 4 (3 times per year) or 6 months (twice per year).To determine the level of response to frequency and length of treatment, the model was run for 4, 2 and 1 times per year, and 2 and 5 years respectively. At each trial, only 27% of the population was treated. The results are presented in Figure 4.18 and Table 4.27.

For all treatment trials the mean increased to and above pre – control levels after treatment stopped. All treatments returned to the same mean worm burden at year 15 except when the population was treated every treated every 3 months for 5 years. Also only this treatment achieved a mean worm burden below 1 worm/host. The rapidity of

the observed return differed most markedly for the number of treatments per year. Thus, for those receiving four treatments per year the mean infection intensity returned to 20 worms/host 3 – 6 years depending on the treatment period and in less than 1 year for twice per year frequency. When treatment occurred once per year the mean never fell below the initial value regardless of how long the intervention continued.

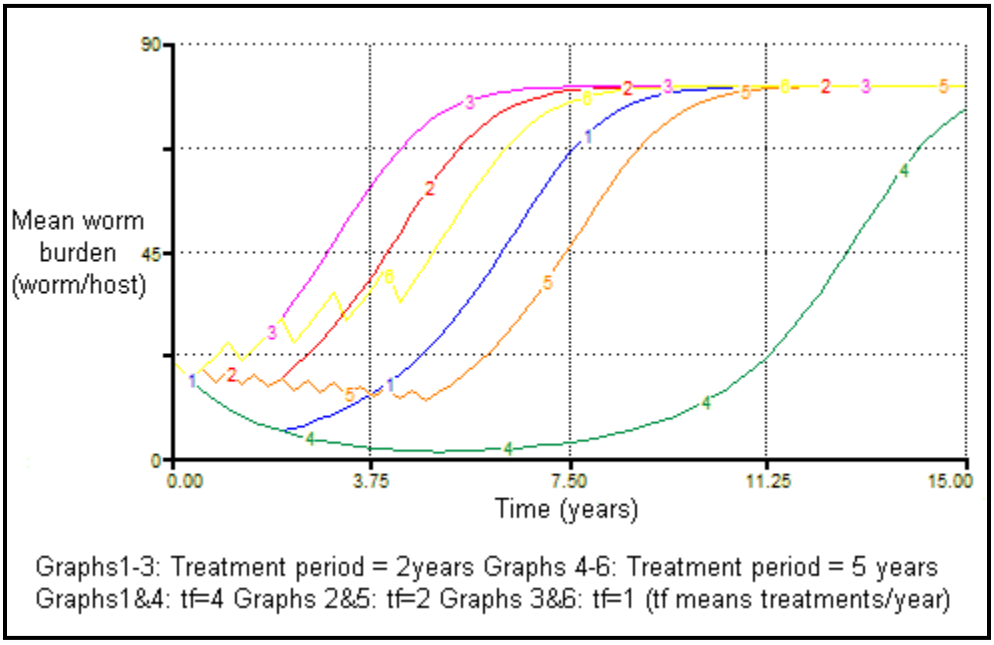

Figure 4.18: Mean worm burden dynamics for varying treatment period and frequency

Table 4.27: Mean worm burden dynamics for varying treatment period and frequency

Time (years)	Mean worm burden $tf = 4$	Mean worm burden $tf = 2$	Mean worm burden $tf = 1$	Mean worm burden $tf = 4$	Mean worm burden $tf = 2$	Mean worm burden $tf = 1$
	Treatment time = 2 years			Treatment time = 5 years		
0	20.00	20.00	20.00	20.00	20.00	20.00
1	10.23	18.41	24.70	10.23	18.41	24.70
2	5.34	17.03	30.01	5.34	17.03	30.01
3	9.13	28.21	46.43	2.80	15.81	35.65
4	15.50	44.13	63.49	1.47	14.72	41.21
5	25.82	61.52	74.58	0.78	13.75	46.19
6	40.97	73.59	79.04	1.33	23.04	63.30
7	58.59	78.71	80.38	2.28	37.12	74.49
8	71.99	80.29	80.74	3.91	54.66	79.01
9	78.15	80.71	80.83	6.69	69.59	80.37
10	80.13	80.82	80.86	11.41	77.25	80.74
11	80.67	80.85	80.86	19.26	79.87	80.83
12	80.81	80.86	80.86	31.60	80.60	80.86
13	80.85	80.86	80.86	48.39	80.80	80.86
14	80.86	80.86	80.86	65.08	80.85	80.86
15	80.86	80.86	80.86	75.33	80.86	80.86

*tf means treatments per year

4.3.3.5 Cost – effectiveness of best and worst case scenarios

The drug of choice for Paquila is Albendazole (Boca Costa Medical Mission, 2004). It is chewable, has relatively few side effects and cost effective. Cost is about US$0.20 per dose (1 tablet), which is about 4 – 10 times the cost for individual diagnosis and is therefore recommended for *en masse* instead of selective treatment (WHO, 2002). Assuming there are on average about 4216 persons in Paquila over the next 15 years, then the cost for treating 25% of the population (1139 persons) over 5 years once per year is about US$ 1139. It will cost almost eight times as much to treat 50% of the same community 4 times per year for 5 years. However the disease would be eradicated in 2 years and mean worm burden would not increase immediately after treatment stopped (see Figure 4.19), while in the former case the money would have been poorly spent since there is little result to show for it. This is similar to recommendation in

literature that for a chemotherapy intervention to be successful, treatment must be given to a proportion of the population above that indicated by the critical value as calculated by equation [4.64], for a greater than the maximum life expectancy of the longest lived developmental stage, which for *Ascaris* is the adult worm and is on average 2 years (Anderson and May, 1992).

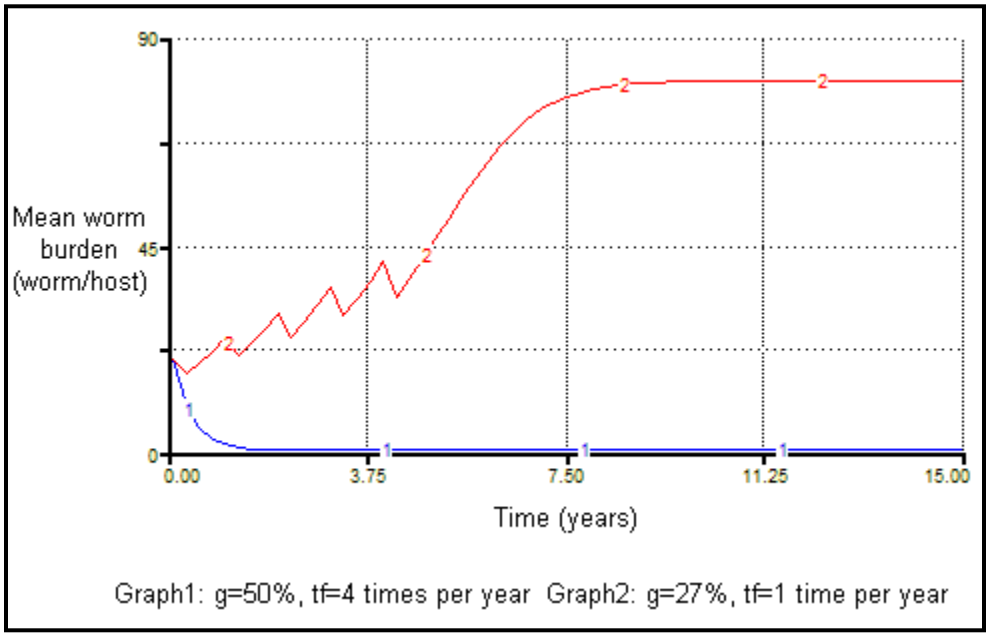

Figure 4.19: Comparing effectiveness of two possible treatment strategies

4.4 Summary and conclusions

The aim of epidemiological modeling is to determine who is affected, and how to prevent, reduce or eliminated the risk of infection. This was covered when modeling for the conditions that promote parasite endemicity in Step 2. In Step 3 the aim was to determine how long it takes to eradicate worms using mass chemotherapy only.

In general, there the response of the mean worm burden was characterized by nonlinearity to changes in the variables governing the population processes, small changes cause big results, after treatment stopped the hosts are rapidly reinfected and large changes in worm burden does not necessitate commensurate reductions in

disease incidence and prevalence in the community. Egg survival, parasite induced host deaths and natural parasite deaths seemed to be the rate determining processes in the life cycle of the parasite. The following are conclusions that are more specific to each simulation:

- Population processes
    - There is some degree of parasite regulation on the host population,
    - A treatment system that deactivates greater than 20% of infective eggs is required to sustainable eradicate the parasite,
    - A 50% decrease in adult worm life expectancy must be maintained for about 7 years to suppress the mean worm burden below unity,
    - If parasite pathogencity is high enough, a significant swing in mean disease intensity can be a result of host rather than worms dying and,
    - When the distribution of the number of worms per host is highly aggregated large changes in mean worm burden produces very little changes in disease prevalence.
- Sustainability and success of chemotherapy program
    - There are a variety of drugs use to treat parasitic infections and each has a different level of efficacy. While reinfection occurred after all trials stopped, drugs that had high cure rates suppressed post – control rebound more,
    - The longer the treatment time and the higher the cure rate of the medicine being applied the more successful the intervention,
    - The higher the number of persons treated in each interval the longer post – control rebound is suppressed and the more likely the intervention to eradicate disease,

- Treatment every 3 months for 5 years was the best scheme, however this was the most expensive and,
- Applying treatment once per year did not affect mean worm burden, prevalence and thus morbidity. Therefore, treatment must be administered at regular intervals, in systematic manner, over an economically viable time scale and must be accompanied by other control measures for sustainable eradication to occur.

5 NUTRITION MODEL

5.1 Background

Parasitic organisms are mainly transmitted via the fecal – oral route: from feces – contaminated surfaces, fields that have been fertilized with unsanitized excreta, and by consuming under cooked or raw plants grown in these fields (Curtis and Cairncross, 2003a; Feachem *et al.*, 1983; Santiso, 1997). The poor nutritional status of those affected exacerbates their, susceptibility to infection, duration and degree of morbidity, and likelihood of mortality (Gendrel *et al.*, 2003; Santiso, 1997).

Every day each human being produces between 20 – 1500 g (wet weight basis) of fecal matter containing up to 8 g of nitrogen, 2 g phosphorus and 3 g of potassium as well as various micronutrients, assuming urine is collected separately (Feachem *et al.*, 1983; Schouw *et al.*, 2002b). Annual nutrient production is equivalent to the amount of commercial fertilizer needed to cultivate 250 kg of cereal, the approximate yearly per capita required food intake (Heinonen –Tanski and Van Wijk – Sijbesma, 2005; WHO, 1985). It is only logical therefore to recycle excreta to crop production.

However, the average person also excretes 10^{10} – 10^{15} microbes per gram of fecal material, some of which can be pathogenic (Vinneras *et al.*, 2003a). Therefore, excreta must be treated to ensure microbial quality before it can be safely reused. This chapter will cover the production of fecal matter and its use to supply the agronomic requirements during soybean cultivation as part of a nutrition program. Microbial inactivation of humanure will be dealt with in Chapter 6.

5.1.1 Protein nutrition and parasitic infections

Protein – calorie malnutrition is the most common and significant cause of immune deficiency in developing countries and is usually associated with parasitic infections (Gendrel *et al.*, 2003; Woodruff and Wright, 1984). The malnutrition – infection interaction, however, is not confined to a linear, one – way causal relationship. That is, a diet with protein deficiencies facilitates the growth and establishment of parasites which in turn create nutritional imbalances due to increased energy requirements to fight them, deceased food intake, and interference with protein absorption and metabolism (Boes and Helwigh, 2000; Stephenson *et al.*, 2000; Venkatachalam and Patwardhan, 1953). On the other hand, as the host becomes more malnourished, worm burden and fecundity may be reduced as nutrients become unavailable (Bundy and Golden, 1987). Studies have shown that when a diet high in protein (skimmed milk) is administered almost all parasitic infections are eradicated (Bundy and Golden, 1987; Venkatachalam and Patwardhan, 1953).

5.1.2 Soybean

In proposing soybean, it should be noted that this is not a promotion for monoculture (an image normally associated with this crop), with its attendant ecological shortcomings, but rather crop rotation and intercropping with traditional staples. Such practices are well known to be a more sustainable method of agricultural production. In addition, soybean is being used here as a nutrient equivalent (a sort of nutrient “indicator organism”). That is, if it is not possible to cultivate soybean, then the calculations presented can be translated to a more culturally and ecologically appropriate protein dense crop. Nevertheless, soybean was chosen for this project because it has several

qualities that makes it ideal for a protein – nutrient intervention.The rationales for choosing soybean are:

- Due to its position as the world's primary source of protein, it has been extensively studied and therefore detailed information is readily available for model input (Liu, 1997; Smith and Circle, 1978; University of Nebraska –Lincoln, 2007),
- It is the only known complete source of protein among plant – based food; contains all the essential amino acids that must be provided because of the body's inability to synthesize them and then some (Liu, 1997),
- Direct use is a form of primary consumption (diet based on vegetation), which is more efficient in terms of energy conversion and utilization; a significant amount of energy is wasted at each trophic level change (Moore, 2002),
- Food and Agricultural Organization of the United Nations and the World Health Organization (FAO/WHO) have used egg and milk as bench marks for protein nutrition. However, 75% of Guatemalan Mayans are lactose intolerant (Boca Costa Medical Mission, 2004; Plenty, 2008; WHO and FAO, 1973, 1985). In addition Ascaris infection is know to exacerbate this condition (Carrera et al., 1984),
- The crop was introduced to a neighboring community over 20 years ago and has been woven into their social fabric, as well as technical support through extension services is available (Plenty, 2008),
- Soybean is a legume and therefore fixes nitrogen. It passes this benefit along when intercropped or rotated with traditional staples (Ghosh et al., 2004; Smith and Circle, 1978), and

- Agronomic retention time (planting to harvesting) is relatively short; about 3 – 4 months (Liu, 1997; Plenty, 2008; Smith and Circle, 1978).

5.1.3 Goals and objectives

The main goal of this chapter is to simulate the response of the population mean worm burden to a nutritional intervention. Unless otherwise cited, model inputs and recommendations for agricultural and nutrition planning were obtained from: Plenty, (2008), University of Nebrask – Lincoln (2007), and WHO and FAO (1973, 1985). The specific objectives are:

- Model soybean cultivation,
- Model the effect of protein nutrition on the parasite induced host death rate in the population mean worm burden dynamics, and
- Model effect of nutrition and chemotherapy on population mean worm burden.

5.2 Excreta model development

5.2.1 Excreta and nutrient production

In rural areas of developing countries, the average adult daily excreta output approximately 0.35 kg feces and 1.2 kg urine (Feachem *et al.*, 1983). Strictly speaking, excreta refers to urine production but is generally used to mean both together, but for this project it is used to mean fecal material only. Typical nitrogen content is approximately 5% (dry weight bases), of which a third is released yearly (Heinonen – Tanski and Van Wijk – Sijbesma, 2005). The nitrogen content of urine is significantly higher than that of feces (Heinonen –Tanski and Van Wijk – Sijbesma, 2005; Schouw *et al.*, 2002a), however, this work focuses on the latter for the following reasons:

- Nitrogen losses associated with urine storage are much higher, producing high concentrations of ammonia which significantly reduces its shelf life (Heinonen – Tanski and Van Wijk – Sijbesma, 2005),
- Soybean requires a high organic matter content that is absent from urine (Plenty, 2008; University of Nebraska – Lincoln, 2007),
- Organic matter is known to improve soil structure, increase the soil's ability to resist drought and erosion and promote salt tolerance of plants (Chambers et al., 2003), and
- The nitrogen in feces becomes available over time, thus reducing the potential for groundwater contamination upon application (Melse and Verdoes, 2005).

5.2.2 STELLA® excreta simulation

For this simulation, "Latrine Content" refers to the combined total capacity of all the latrines in the community assuming each household has and uses this facility (see Figure 5.1). Based on rate of production and capacity of the latrine and solar vaults (details in Chapter 6), it is expected that the latrine will be emptied every 4 months. Excreta that is not immediately used for soybean production is stored for later use. The simulation result is given in Figure 5.2. The graph shows that fecal matter will be removed from latrine vaults to the solar vault every 4 months, with a four – month offset separating the vaults. Thus, for the first year, processed excreta will not be harvested in time for the soybean planting season which occurs around May.

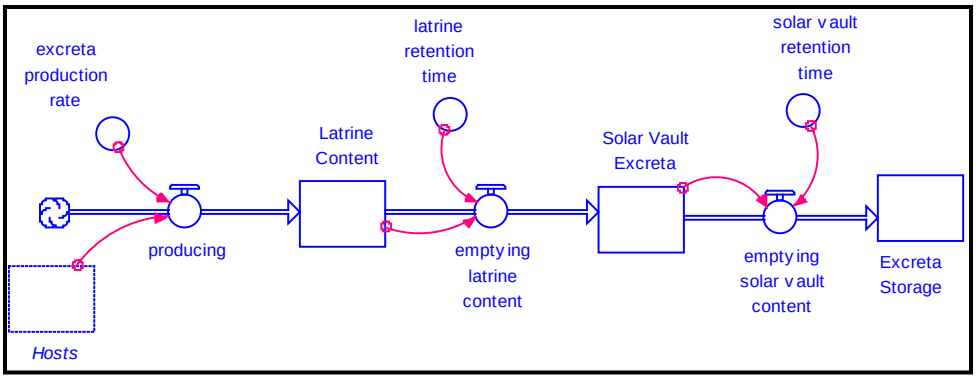

Figure 5.1: STELLA® representation of excreta production

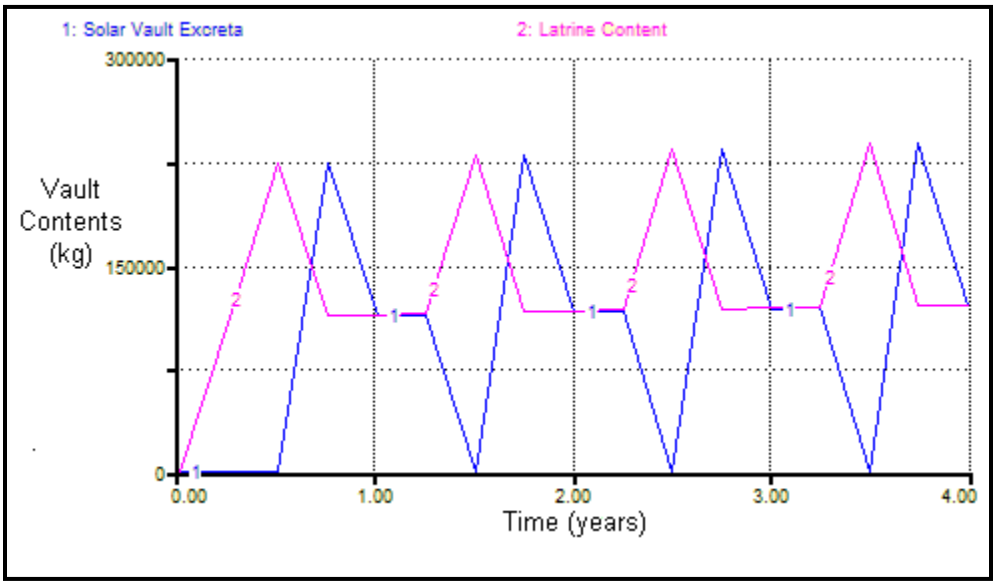

Figure 5.2: Result of excreta production and processing in solar and latrine vaults

5.3 Soybean model development

5.3.1 Nutrient requirements of hosts

When dietary protein is derived from a single vegetable source such as soybean, the daily recommended intake is 1.1 g per kg of body weight. Assuming a 70 kg person the yearly protein intake would be 28 kg (1.1 * 70* 365). In general the recommended range is 0.8 – 1.5 g/kg/d and 0.66 g/kg/d for basal metabolic maintenance. For maintenance: 0.66 g/kg/d for adults and 0.67 g/kg/d for children.

Dried soybeans contain on average 40% protein by weight of which only about 70% is biologically available depending on preparation (National Soybean Research Lab, 2008). So for a 70 kg person with intake of 1.1 g/kg/d, 100 kg soybean will cover his 28 kg protein yearly requirement (28/0.4 kg soybean).

Table 5.1: Protein and soybean requirements

Metabolic requirement	Protein requirement		
	Daily (g/kg/d)	Yearly (kg/year)*	Soybean equivalent*
Maintenance	0.66	16.9	60
Lower limit	0.80	20.4	73
Single veg. source	1.10	28.1	100
Maximum	1.50	38.3	137

*Assuming a 70 kg person

5.3.2 Land requirement

Assuming available land is fixed, the arable land determines the carrying capacity of the village. The village sits on an area of about 10.36 km^2, therefore using the percentage arable land for Guatemala, approximately 1.37 km^2 (13.22%) can be used for crop production (CIA, 2008).

The average crop yield for soybean in Guatemala is 39 kg of soybean for every 1 kg seed planted (28 kg seeds produced 1089 kg soybeans per acre ($4*10^{-3}$ km^2)). Thus, each person requires about 2.6 kg (100 kg soybean/person / 39 kg soybean/1 kg seed) seeds planted on his behalf resulting in total requirement of 102.6 kg soybeans per year (20% factor of safety is added to 100 kg requirement during simulation).

From the planting rate of 28 kg seeds produced 1089 kg soybeans per acre ($4*10^{-3}$ km^2) each person requires $3.7*10^{-4}$ km^2. The total carrying capacity of the village is then approximately 3700 persons (1.37 km^2/$3.7 * 10^{-4}$ km^2/person). The

number of seeds per kg of soybean depends on the variety; for this work 1 kg of soybean is taken have 5280 seeds.

5.3.3 Nitrogen demand requirement from humanure

For the yield given above, crop nitrogen demand over the entire growing season is 35910 kg of N/km^2 (see Table 5.2). Since it is a legume, soybean will fulfill 75% of this from soil nitrogen (existing soil nitrogen and mineralized soil organic matter nitrogen), acquiring the rest through fixation. However, applying more than 50% of the required total demand is counterproductive as this prevents the nodules from fixing atmospheric nitrogen, increases the likelihood of nitrogen contamination due to excess residual nitrates at the end of the growing season, and has been shown to increase plant susceptibility to certain diseases (University of Nebraska – Lincoln, 2007).

Typical nitrogen fraction in excreta is about 11% on a dry weight basis and only a third is bio – available each year (Heinonen – Tanski and Van Wijk – Sijbesma, 2005; Tarkalson *et al.*, 2006). In manure application it is typical to expect 15 – 35% losses due to ammonia volatilization (Sogaard *et al.*, 2002). However, conditions in the latrine vaults can be reasonably assumed to be anaerobic and pH around 7, hence in the presence of urease, urea is converted to the ammonium ion (NH_4^+) (Montangero and Belevi, 2007).

Table 5.2: Soybean nutrient uptake at 1089 kg soybeans per acre (4*10^{-3} km^2) yield

Nutrient	Seed	Stover*	Total
N (kg/km^2)	21432	14478	35910

*Stover: leaves, stalks and pods left in field after harvest

The results from Table 5.3 show that in the early stages humanure may have to be supplemented by chemical fertilizer depending on the ambient nitrogen content of the village soil. However, after successive crop seasons the nitrogen fixed from the air,

mineralized from soil organic matter from previous seasons and continued available excreta from the human population will cover the required demand.

Table 5.3: Percentage soybean nitrogen demand fulfilled by humanure

Variable	Minimum	Maximum
Hosts	3500	4990
Excreta production, kg	447,125	637,473
Area under cultivation, km^2	1.28	1.37
Required nitrogen demand (35910 kg N/km^2), kg N	45,965	49,197
Recommended excreta application (up to 50%), kg N	22, 983	24,598
Available nitrogen in excreta in 1st year, kg N	16,820	23,981
Percent demand fulfilled by humanure	37%	49%

5.3.4 STELLA® soybean simulation

The host and parasite model is similar to those presented in Chapter 4, only now the parasite induced death rate is being modified by a "multiplier" which modifies the normal parasite induced death rate over time based on the ratio of available to desired nitrogen (see Figure 5.3). The assumption is, as the amount of nitrogen increases in the host's diet, his ability to fight infection is strengthened and the pathogencity of the worms against the host is reduced (Anderson *et al.*, 1979). To simulate this, the planting rate was varied to produce different amounts of soybean per host. It was assumed that currently the host population is getting just enough protein for maintenance which results in the default pathogencity used in Chapter 4.

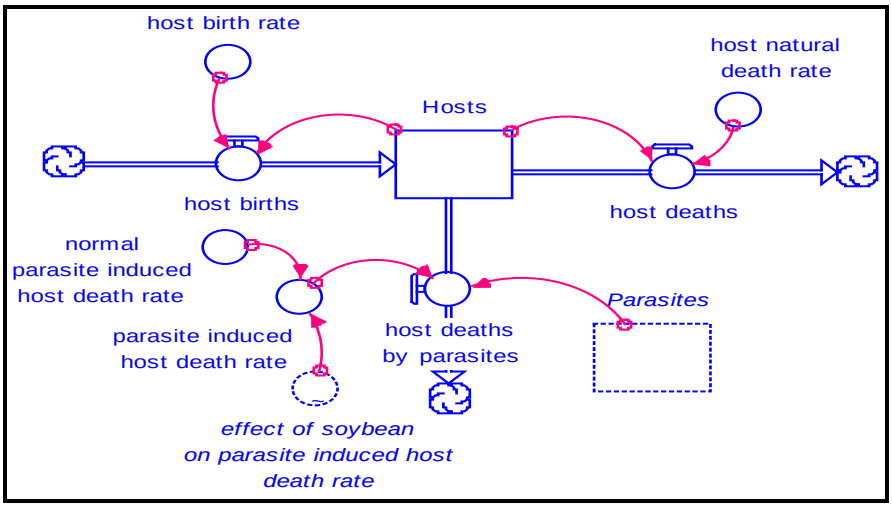

Figure 5.3: STELLA® representation of host population illustrating effect of nutrition on host's survival

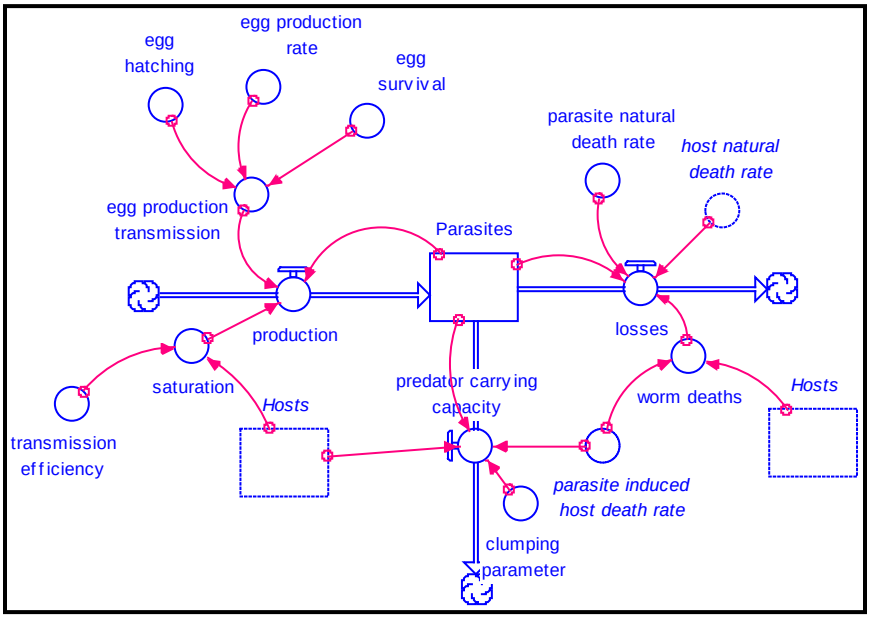

Figure 5.4: STELLA® representation of parasite population illustrating effect of nutrition on parasite's survival

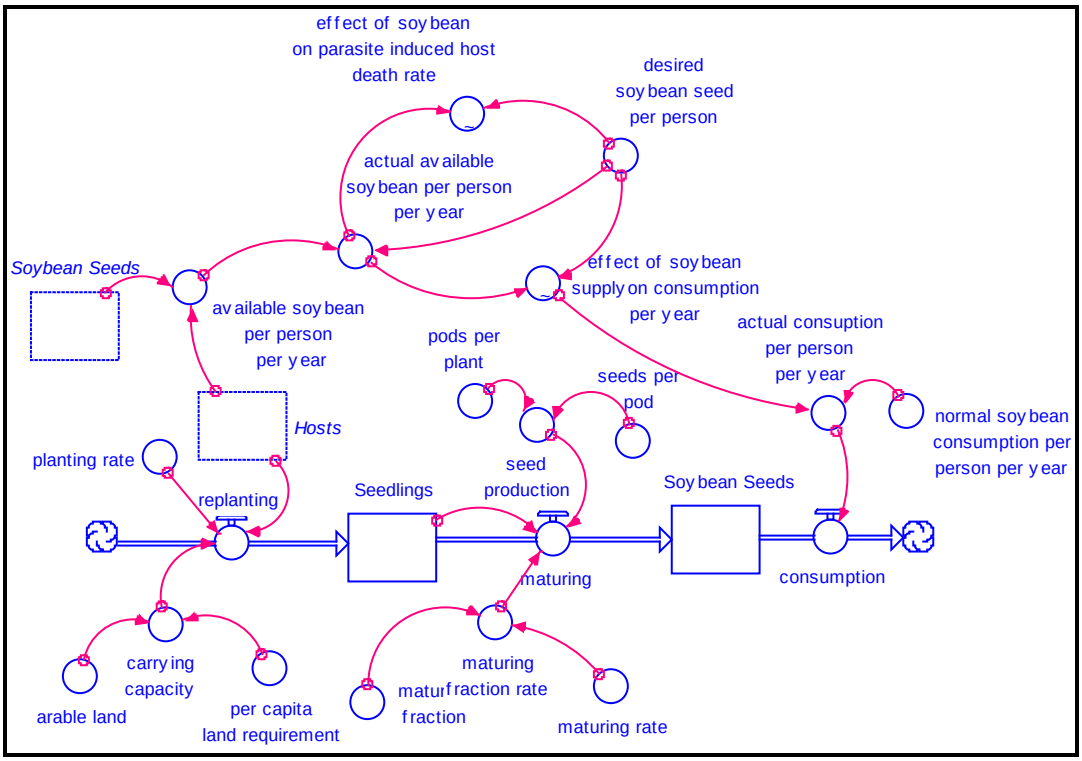

Figure 5.5: Soybean production cycle with effect of supplying nutrient

Table 5.4 shows that the results of providing nutrition for metabolic maintenance are similar to those seen in Chapter 4. As nutrition is increased more hosts are surviving, an almost 300 hosts difference by the end of the 15 – year run. When replanting was removed the mean worm burden increased drastically but was suppressed for the nutrition interventions (see Table 5.5). A counterintuitive observation can be seen with the increase of mean worm burden with increasing nutrition. The possible reasons for this is, nutrition was assumed to affect only the host's ability to resist death from the parasite but did not change the natural death rate of the parasite. Hence, nutrition by itself will increase the life expectancy of the host population but chemotherapy is needed to reduce the mean worm burden.

Table 5.4: Host population at different levels of protein interventions

Time (years)	Hosts default from Chapter 4	Hosts base protein	Hosts minimum protein	Hosts required protein	Hosts maximum protein
0	3,500.00	3,500.00	3,500.00	3,500.00	3,500.00
1	3,582.59	3,562.39	3,562.39	3,562.39	3,562.39
2	3,655.87	3,623.19	3,623.77	3,624.97	3,626.63
3	3,714.39	3,682.41	3,684.40	3,688.55	3,694.26
4	3,772.43	3,740.29	3,744.81	3,754.27	3,767.42
5	3,831.36	3,797.36	3,805.83	3,823.74	3,848.17
6	3,891.19	3,854.40	3,868.55	3,898.95	3,929.78
7	3,951.95	3,912.39	3,934.27	3,978.62	4,012.38
8	4,013.65	3,972.43	4,004.45	4,058.33	4,096.17
9	4,076.31	4,035.68	4,080.69	4,138.17	4,181.50
10	4,139.93	4,103.30	4,157.19	4,218.34	4,268.83
11	4,204.53	4,176.45	4,233.20	4,299.20	4,358.76
12	4,270.14	4,251.14	4,309.09	4,381.16	4,451.98
13	4,336.76	4,325.35	4,385.30	4,464.71	4,549.27
14	4,404.41	4,399.53	4,462.29	4,550.30	4,651.59
15	4,473.10	4,474.15	4,540.48	4,638.34	4,760.14

Table 5.5: Mean worm burden for different levels of protein interventions

Time (years)	Mean worm burden - no intervention	Mean worm burden base protein	Mean worm burden minimum protein	Mean worm burden required protein	Mean worm burden maximum protein
0	2.00	2.00	2.00	2.00	2.00
1	21.20	2.47	2.47	2.47	2.47
2	133.58	3.03	3.03	3.03	3.04
3	164.12	3.67	3.68	3.70	3.73
4	164.59	4.40	4.43	4.50	4.59
5	164.64	5.21	5.29	5.45	5.69
6	164.69	6.08	6.24	6.62	7.01
7	164.74	7.01	7.33	8.05	8.57
8	164.79	8.00	8.59	9.66	10.38
9	164.83	9.07	10.12	11.40	12.44
10	164.88	10.28	11.71	13.22	14.77
11	164.92	11.69	13.26	15.05	17.42
12	164.97	13.14	14.68	16.85	20.49
13	165.01	14.42	15.95	18.60	24.18
14	165.05	15.51	17.05	20.33	28.92
15	165.10	16.41	18.01	22.06	35.68

5.4 STELLA® integrated population dynamics

5.4.1 Chemotherapy and nutrition

The STELLA® model for the host's population is similar to that of Figure 5.3 above. Figure 5.6 illustrates an additional pathogen loss through chemotherapy. The best chemotherapy strategy was found to be treating 50% of the population, every 3 months for 5 years with a drug that was 94% efficacious. This program was adopted for this simulation; only the treatment period was reduced to 2 years. The results in Tables 5.6 and 5.7 show that an additional 219 lives, over the maximum achieved in the above simulation, were saved and that the worms are virtually eradicated without the rebound seen with chemotherapy alone. It should be noted that the ultimate populations for all types of intervention were similar; this is due to the fact that the carrying capacity has been exceeded as people are living longer and so saturation occurs. This has been observed in malaria eradication programs (Barlow, 1967). Thus it is necessary to promote family planning in conjunction with these interventions.

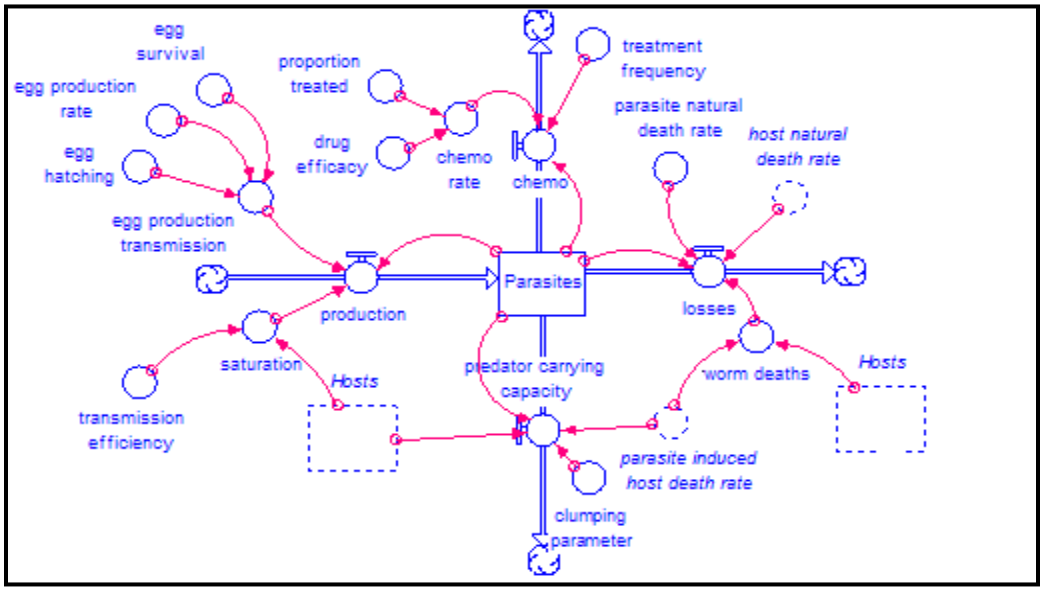

Figure 5.6: STELLA® representation of parasite population illustrating nutrition and chemotherapy

Table 5.6: Host population at different levels of protein interventions with chemotherapy

Time (years)	Hosts - no intervention	Hosts base protein	Hosts minimum protein	Hosts required protein	Hosts maximum protein
0.00	3,500.00	3,500.00	3,500.00	3,500.00	3,500.00
1.00	3,575.52	3,575.52	3,575.52	3,575.52	3,575.52
2.00	3,660.83	3,660.86	3,660.87	3,660.88	3,660.90
3.00	3,748.46	3,748.49	3,748.50	3,748.51	3,748.53
4.00	3,838.17	3,838.21	3,838.22	3,838.24	3,838.27
5.00	3,930.01	3,930.08	3,930.09	3,930.12	3,930.16
6.00	4,024.05	4,024.14	4,024.16	4,024.20	4,024.24
7.00	4,120.31	4,120.44	4,120.47	4,120.53	4,120.58
8.00	4,218.85	4,219.05	4,219.09	4,219.17	4,219.23
9.00	4,319.72	4,320.01	4,320.07	4,320.17	4,320.23
10.00	4,422.97	4,423.38	4,423.47	4,423.58	4,423.65
11.00	4,528.62	4,529.22	4,529.34	4,529.46	4,529.55
12.00	4,636.73	4,637.59	4,637.74	4,637.87	4,637.98
13.00	4,747.32	4,748.55	4,748.72	4,748.87	4,749.00
14.00	4,860.43	4,862.15	4,862.34	4,862.52	4,862.69
15.00	4,976.08	4,978.46	4,978.67	4,978.88	4,979.10

Table 5.7: Mean worm burden for different levels of protein interventions with chemotherapy

Time (years)	Mean worm burden - no intervention	Mean worm burden base protein	Mean worm burden minimum protein	Mean worm burden required protein	Mean worm burden maximum protein
0	2.00	2.00	2.00	2.00	2.00
1	0.07	0.07	0.07	0.07	0.07
2	0.00	0.00	0.00	0.00	0.00
3	0.00	0.00	0.00	0.00	0.00
4	0.00	0.00	0.00	0.00	0.00
5	0.00	0.00	0.00	0.00	0.00
6	0.01	0.01	0.01	0.01	0.01
7	0.01	0.01	0.01	0.01	0.01
8	0.01	0.01	0.01	0.01	0.01
9	0.01	0.01	0.01	0.01	0.01
10	0.01	0.01	0.01	0.01	0.01
11	0.02	0.02	0.02	0.02	0.02
12	0.02	0.02	0.02	0.02	0.02
13	0.03	0.03	0.03	0.03	0.03
14	0.04	0.04	0.04	0.04	0.04
15	0.05	0.05	0.05	0.05	0.05

5.5 Summary and conclusions

The aim of this chapter was to model the human population in response to various nutrition regimes and compare vertical interventions of nutrition or deworming, with an integrated program. The following is a summary of the results obtained:

- Excreta production and nutrient recycling
    - Excreta production provides enough nitrogen to meet the demand of soybean cultivation, and
    - In the first year chemical fertilizer may have to be used to start the project, depending on the fertility of the soil.
- Nutrition intervention
    - In the first year, a feeding program will be necessary while the soybean crop matures,
    - Depending on the level on nutrition provided, up to 300 lives can be saved,
    - Nutrition significantly reduces worm burden but was not able to eradicate the worms from among the host population, and
    - (Stephenson, 1980) found that when protein deficient hosts were dewormed, growth rates increased 20 – 35. The simulation found that number of host surviving increase about 11% when supplied with protein.
- Sustainability and success of integrated program
    - A further 219 lives were saved with the introduction of the chemotherapy program,
    - Compared to chemotherapy only a shorter treatment period is necessary for eradication, for example, only 2 years compared to the 5 previously recommended, and

- As these programs achieve success the population will expand. To avoid unsustainable population growth, family planning education is also necessary,

Eradicating parasitic infection from a community is a balancing act among community resources, health and living status. As the nutritional status is improved and worm burden decreased, the population will expand beyond its carrying capacity, which can feed back to cause excess deaths due to scarcity. Thus, in addition to chemotherapy and nutritional programs, family planning must also be promoted. While the mean worm burden did not rebound as previously seen in Chapter 4, there was some reinfection (starting in year 6) due to the presence of infectious eggs in the environment. Thus, a latrine intervention is necessary.

6 SOLAR HIGH – RATE LATRINE

6.1 Background

As seen in Chapter 5, fecal material is a valuable resource that can be recycled to lifesaving crop production. However, improper handling and disposal facilitate the transmission of parasitic organisms, which are the cause of approximately 1.5 billion bouts of infectious diarrhea and 3 million deaths annually in children alone (Kosek *et al.*, 2003; Meddings *et al.*, 2004). Therefore, before fecal matter can be used in crop cultivation, its microbial quality must first be assured. In developing countries the most common methods of excreta sanitation are the “drop and store” options of latrines, addition of chemicals, and composting (Jimenez, 2007; Langergraber and Muellegger, 2005; Vinneras *et al.*, 2003a).

6.1.1 Excreta treatment in developing countries

Traditionally, pit latrines consisted of an unlined hole in the ground surrounded by a simple cover to provide privacy (Grimason *et al.*, 2000). The Ventilated Improved Pit (VIP) latrine consists of a prefabricated concrete floor over the drop zone, a superstructure, and a ventilation pipe to reduce odor and prevent fly infestations (Cairncross and Feachem, 1983). The latter is being widely promoted and installed in response to the Millennium Development Goal (MDG) of globally reducing the proportion of persons currently without adequate sanitation from 50 to 25% by 2015 (Jimenez *et al.*, 2006; Langergraber and Muellegger, 2005). In these systems microbial inactivation is a function of storage time, based on the assumption that most microorganisms die

naturally upon exiting their host and are exposed to harsh environment conditions (Corrales *et al.*, 2006; Jimenez, 2007). However, these systems can become transmission loci where proper hygiene is not practiced resulting in higher incidence of diseases than where open defecation is practiced, can contaminate ground water sources and do not allow for reuse due to high effluent concentrations of resistant pathogens (Banks *et al.*, 2002; Jensen *et al.*, 2005; Vinneras *et al.*, 2003b).

Ash or lime is usually added to latrine contents to enhance microbial die off during storage by increasing the pH (Capizzi – Banas *et al.*, 2004). While more effective than simply storing, the efficacy of the ashes is contingent on the source of the wood, which limits quality control (Vinneras *et al.*, 2003a). Effluent quality is more predictable when lime is used, where pH above 12 is guaranteed, but its use can be economically challenging due to high cost (Capizzi – Banas *et al.*, 2004).

Sustained temperatures of up to 70 ^{o}C, which will deactivate most pathogens, can be achieved in composting systems, but for them to work, specific carbon to nitrogen ratio, moisture contents and aeration rates must be achieved and maintained, that are not possible without specialized knowledge (Heinonen – Tanski and Van Wijk – Sijbesma, 2005; Redlinger *et al.*, 2001). In addition, as much as 40% nitrogen and 60% organic carbon can be loss during processing (Fares *et al.*, 2005).

6.1.2 Solar Latrines

Most developing countries are located in warm humid climates and receive up to 3000 hours of sunshine per year (Eggers – Lura, 1979). Solar Latrines are therefore particularly suited for countries in the tropics. Solar Latrines are modified VIP latrines which utilize the thermal energy from sunlight to inactivate microbes and were introduced in Central America in the early 1990s. Over the years several updates have been introduced because the systems were not able to achieve and maintain the

elevated temperatures required for inactivation due to several design flaws (Corrales *et al.*, 2006). Earlier versions were poorly oriented to the sun, were constructed under trees which blocked solar insolation, and had vault covers that were opaque to energy rich light rays (metallic cover that does not allow visible light through). In addition, there has not been a rigorous analysis of the heat transfer that results from solar flux into the vault.

6.1.3 Goals and objectives

This portion of the research has two main goals with several accompanying objectives as follows:

- Design a Solar Latrine and develop a multi – physics model for simultaneous heating of and microbial inactivation in latrine contents based on Fourier's and Fick's Laws
    - Propose a new latrine design,
    - Develop solar tables for the study village, and
    - Model heating of latrine contents using Finite Element Method package, COMSOL®, to determine if effluent excreta can meet US EPA Class A Biosolids quality standards.
- Simulate the population response to a latrine intervention
    - Develop STELLA® model to represent latrine intervention, and
    - Model population mean worm burden to combined interventions of chemotherapy, soybean and latrine.

6.2 Solar Latrine design and modeling

6.2.1 Current design description

Current Solar Latrines are similar to other VIP designs; in that the liquid fraction of excreta is separated from solids using a urine – diverting toilet bowl (see Figure 6.1). Urine diversion reduces the emptying frequency and leaching hazard to the ground and surface waters, and produces effluent with lower moisture contents (Heinonen – Tanski and Van Wijk – Sijbesma, 2005). Typically, the foundation, envelopes of the vaults and superstructure are made from standard concrete blocks and poured concrete. The latrine and solar vaults are above ground, which reduces the risk of groundwater contamination through seepage. A vent pipe carries off excess odors and prevents fly infestations.

Excreta accumulate in a pile in the drop zone and must be manually pushed and shoveled, if access is provided at all. Once a substantial pile builds up, the material is pushed back towards the solar vault for thermal processing. Therefore, both vaults are open to each other, which leads to parasitic heat losses through the toilet pedestal and vent pipe. Sunlight is made up of several types of electromagnetic radiations (Goswami *et al.*, 2000). Thermal radiation is one portion of the radiation spectrum and consists of infrared, visible and ultraviolet wavelengths, and heat up objects on contact or is emitted when matter is heated (Yüncü *et al.*, 1987). Metallic materials are opaque to light in the visible range and incident energy heats first the material before energy is emitted. These energy conversions have associated heat losses. Thus the metallic covers of the current Solar Latrine models are not very efficient at heating the fecal material in the vault below.

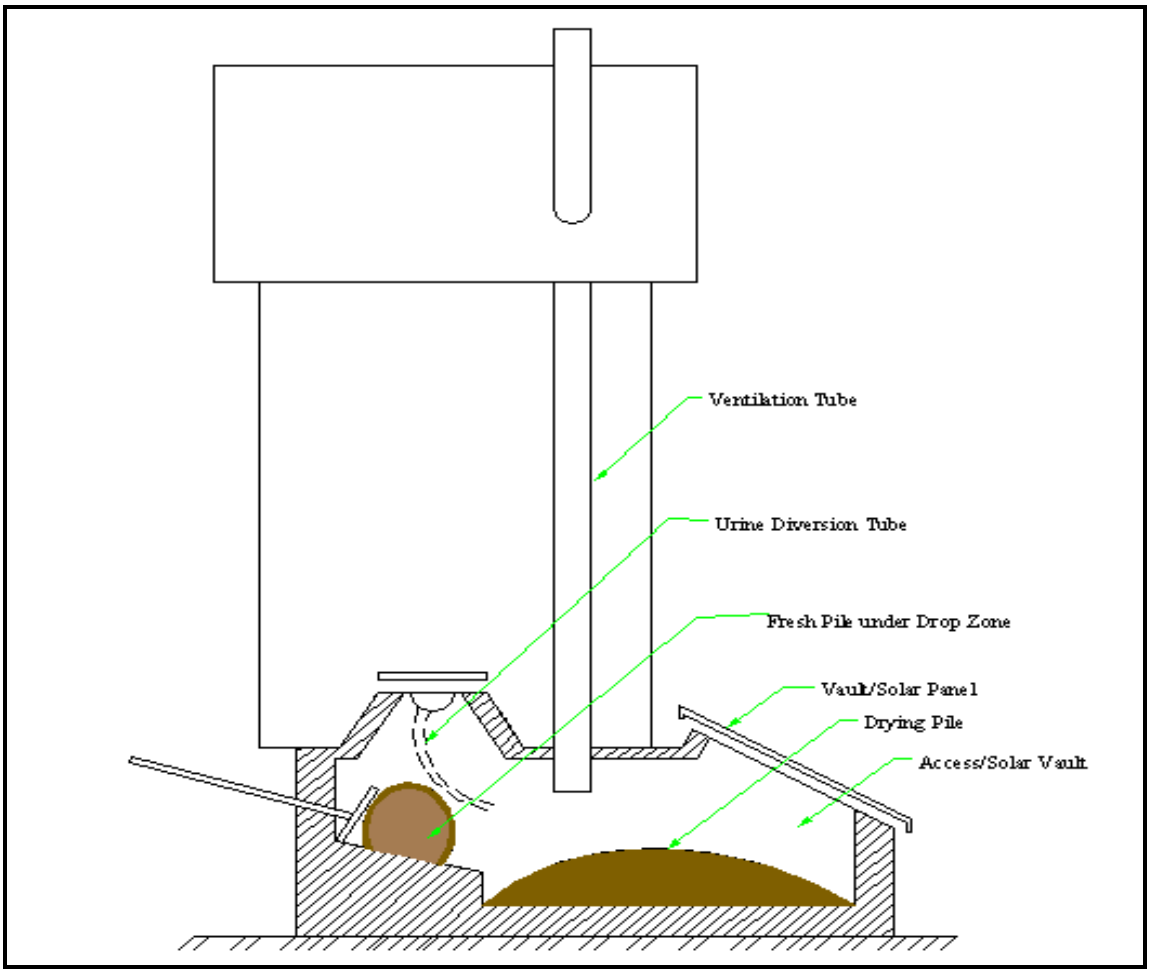

Figure 6.1: Current Solar Latrine design

6.2.2 New Solar Latrine design

The proposed design is a modification of the Solar Latrine in Figure 6.1. The major changes included: addition of a drum under the drop zone to collect fecal matter, closing off the solar vault from the drop zone, replacing the metal solar panel with a light transparent glazing, and addition of a water collection system for a hygiene station. Figure 6.2 shows an isometric cut – away view of the entire arrangement.

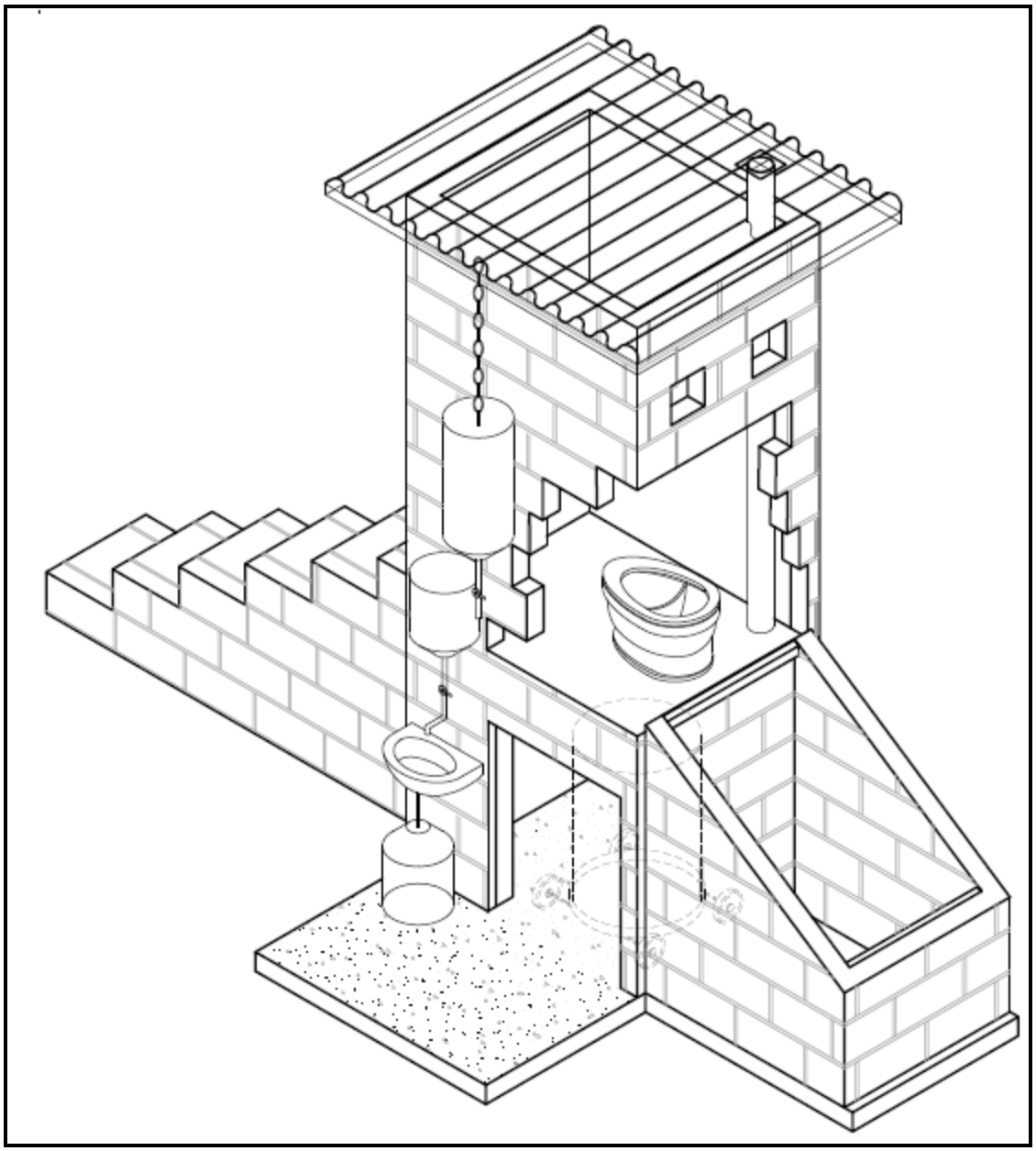

Figure 6.2: Isometric cut – away view of new Solar Latrine design

The proposed design has new several features:

- A 1/8 ” thick single polycarbonate glazing with shading coefficient of 0.98 (opaque to only 2% of incoming solar energy) was chosen to replace the metal vault panel, is inclined at 29.53^{o} (latitude + 15^{o}) resulting in greater insolation

throughout the year (ASHRAE, 1997; Jain and Jain, 2004; Kreider and Kreith, 1981).

- The solar vault is completely blocked off from the drop zone, instead an access door is constructed thru the side of the latrine vault.
- The vent pipe is split into a “Y” entering both vaults (see Figure 6.3). The potion entering the solar vault “T’s” off, running along the entire width and is perforated to prevent short – circuiting of air flow over the material being processed. This portion can be removed and the orifice capped during the heating phase of processing.
- A 55 – gallon cylindrical drum is placed in the drop zone to store excreta during the filling phase, which is removed for treatment and replaced with an empty one once capacity is reached. This promotes safer handling of the potentially hazardous material. Once removed from the drop zone, the drum can be opened to form two semicircular troughs; hence it is given the name “Solar Processing Trough (SPT)”. Details are provided in Figure 6.4. A drum cart is provided for easier transfer of SPT from latrine to solar vault.
- Taking advantage of the high rainfall of the area, a rain collection system is provided for hand washing (after toilet use and SPT handling). This system features a novel PVC chain link water guide, which eliminates the need for cleaning associated with traditional gutters (see Figure 6.5).
- Gravel resulting from concrete construction on the latrine can be placed in the solar vault to form a rock bed to provide heat when the sun is not shining (Figure 6.6). A rock bed is uni – directional heat exchanger, that is, during the day it takes in energy and at night (or sunless days) releases it (Kreider, 1989).

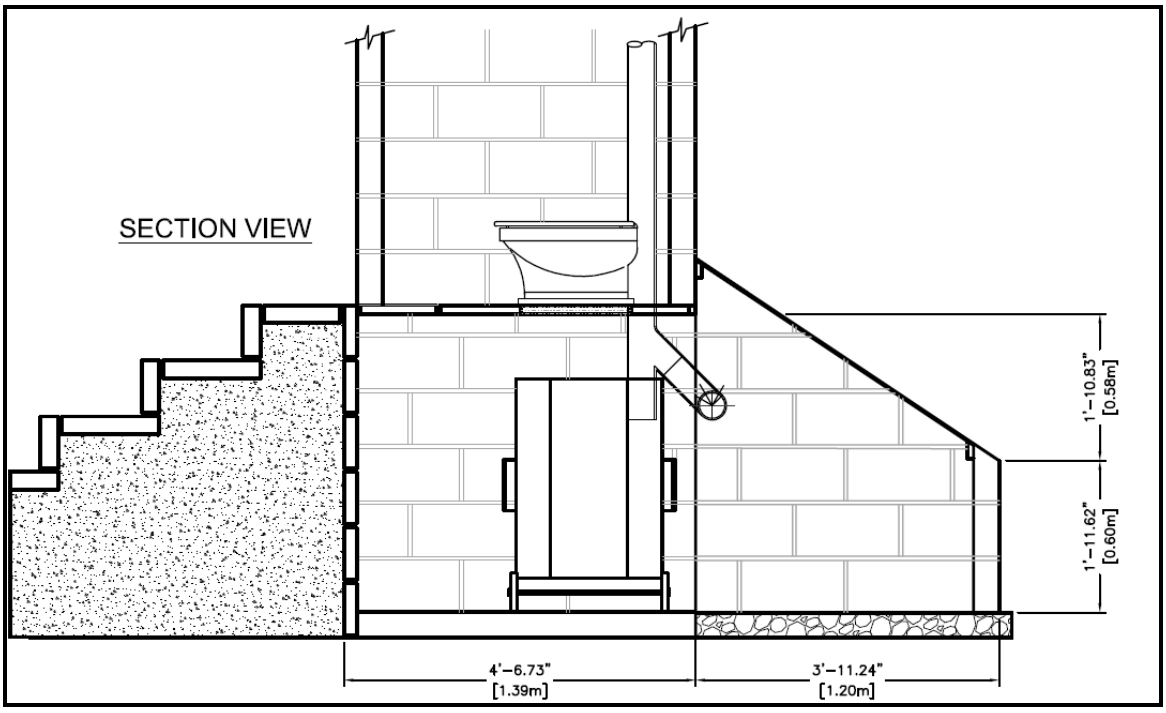

Figure 6.3: Section view thru new Solar Latrine design

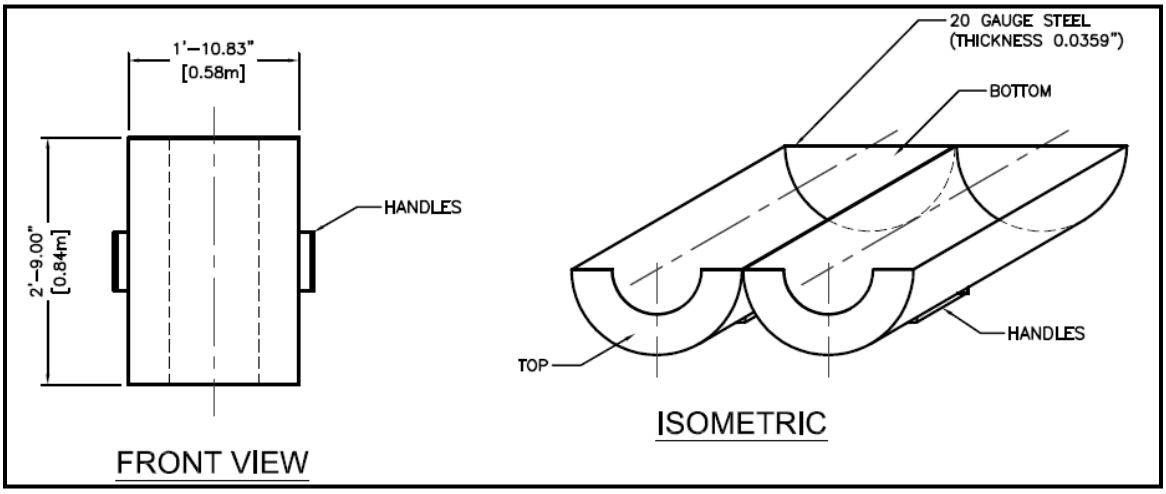

Figure 6.4: Detail view of Solar Processing Trough (SPT)

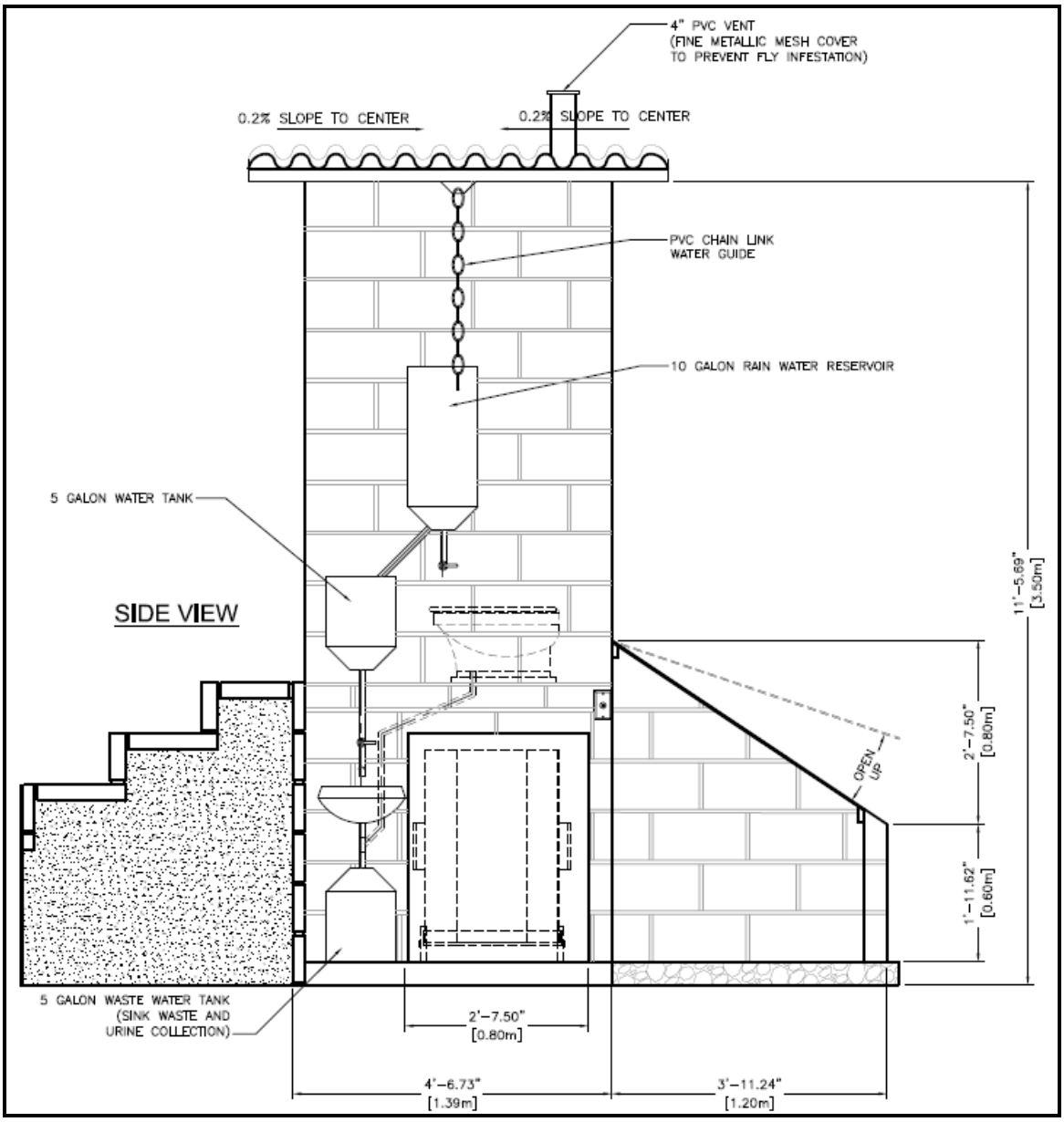

Figure 6.5: Side view showing details of water collection system

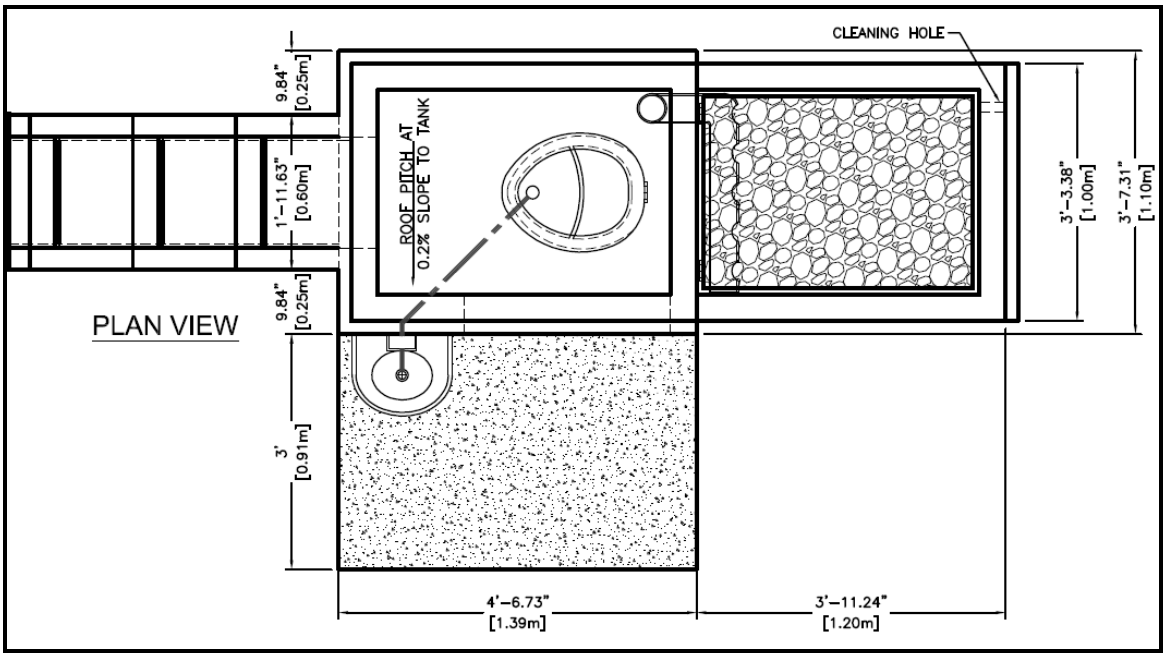

Figure 6.6: Plan view of new Solar Latrine showing perforated vent pipe in solar vault

6.2.2.1 Solar Processing Trough (SPT) capacity requirement calculations

In rural areas of developing countries, the average adult daily excreta output is 350 g feces and 1.2 kg (0.32 gallons) urine (Feachem *et al.*, 1983). For 4 adult equivalents (2 adult and 4 children) total production is 168 kg (370 lbs) assuming latrine harvesting is carried out every 4 months. Fecal matter is about 80 – 95% water so the density was taken as 1000 kg/m^3. Thus, the volume required is 0.168 m^3 (44 gallons). A standard 0.21 m^3 (55 gallons) – drum was chosen, providing 20% extra volume to allow for addition of ash, soil or other desiccating materials. Using the Manufacturers Standard Gauge for steel sheet (41.82 lbs/ft^2/in thickness), a 20 gauge (0.0359 in thick) 55 – gallon metal drum weighs approximately 13 kg (28.5 lbs) resulting in total at capacity weight of 181 kg (398 lbs) which can be readily lifted by two adult men. Based on 0.32 gallon output and allowing for hand washing, the 5 – gallon waste water tank needs to be emptied about every 3 days.

6.2.3 Determination of the total instantaneous radiation on vault glazing

Table 6.1: Symbols used in developing solar tables

Symbol	Description	Units
δ_s	Solar declination angle	deg
I_c	Total instantaneous solar radiation incidence on glazing	W/m^2
$I_{b,c}$	Direct beam radiation	W/m^2
$I_{d,c}$	Diffuse radiation	W/m^2
$I_{r,c}$	Ground reflected radiation	W/m^2
$I_{b,N}$	Beam radiation normal to sun's rays	W/m^2
I	Extraterrestrial solar radiation	W/m^2
I_o	Solar constant	W/m^2
A_c	Area of vault glazing (solar transparent cover)	m^2
ρ	Ground reflectance	[ ]
k_o	Optical depth	[ ]
C	Sky diffusion factor for a given month	[ ]
n	The day number	[ ]
L	Latitude of the location	deg
α	Solar altitude	deg
a_s	Solar azimuth	deg
h_s	Solar hour angle	deg
a_w	Orientation angle of the solar vault	deg
θ	Angle of incidence of beam radiation on glazing	deg
β	Angle of tilt of solar vault panel	deg

*Standard angular measurements applied, e.g. north is considered positive.

The total instantaneous solar radiation (I_c) incidence on the vault glazing of area (A_c), is a function of the vault location latitude (L), the solar declination (δ_s), solar altitude (α), solar azimuth (a_s), angle of incidence of beam radiation (*Cos* θ), ground reflectance (ρ), and weather conditions. Equations and the following discussion can be obtained from any standard solar engineering text and unless otherwise stated were

acquired from Davidson and Chavez (1996), Duffie and Beckman (1974, 1980) Goswami *et al.* (2000), Kreider and Kreith (1981), Kreider (1989), Kreider and Joint (1975), Kreith and Kreider (1978), Wieder, (1982), Wu *et al. (*1975), and Yüncü *et al.* (1987). A conceptual model is presented in Figure 6.7.

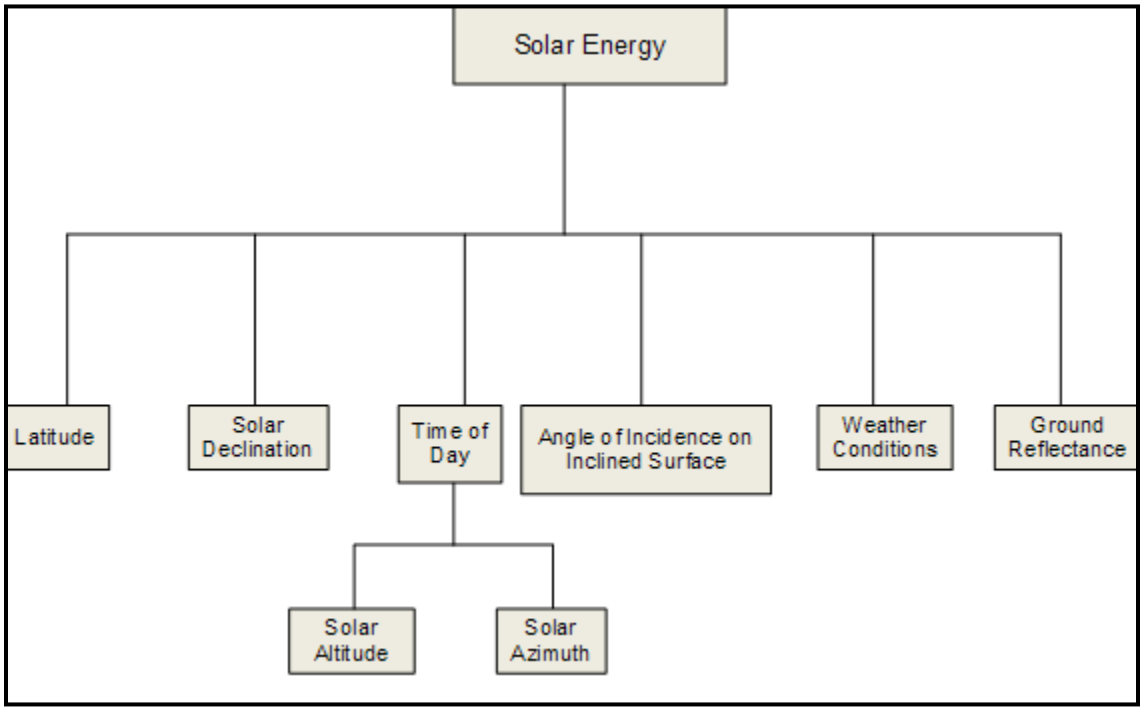

Figure 6.7: Conceptual model of solar insolation on a horizontal or inclined surface

The total instantaneous solar radiation (I_c) incidence on solar panel of area (A_c) is given by:

$$I_c = I_{b,c} + I_{d,c} + I_{r,c} \qquad [6.1]$$

Where ($I_{b,c}$) is the direct beam radiation and is given by:

$$I_{b,c} = Cos\,\theta \cdot I_{b,N} \qquad [6.2]$$

Where (θ) is the angle of incidence of the direct beam radiation on the panel is calculated as follows:

$$Cos\,\theta = Cos\alpha . \cdot Cos(a_s - a_w) \cdot Sin\beta + Sin\alpha \cdot Sin\beta \qquad [6.3]$$

Where (a_w) is the orientation angle of the vault, (when vault is facing south, a_w = *0*), (a_s) is the solar azimuth angle (angle formed on the horizontal plane of the earth's surface as it moves across the sky and is measured from the south) and is given as follows:

$$Sin\,a_s = \frac{Cos\,\delta_s \cdot Sin\,h_s}{Cos\,\alpha} \qquad [6.4]$$

Where (α) is the solar altitude angle at a given time of the day and is computed from the following equation:

$$Sin\alpha = Sin\,L \cdot Sin\,\delta_s + Cos\,L \cdot Cos\delta_s \cdot Cos\,h_s \qquad [6.5]$$

Where (L) is the latitude of the location under consideration, (δ_s) the declination angle, which is given by:

$$\delta_s = 23.45^o \cdot Sin\left[\frac{360}{365} \cdot (284 + n)\right] \qquad [6.6]$$

(h_s), the solar hour angle and is given by:

$$h_s = \frac{15}{hour} \cdot (hours\ from\ solar\ noon) \qquad [6.7]$$

From equation [6.1] ($I_{b,N}$), is the instantaneous solar beam radiation normal to sun's rays, given by:

$$I_{b,N} = C_n \cdot I \cdot \ell^{-k_o / \sin\alpha} \qquad [6.8]$$

Where (C_n) is the clearness number, (k_o) the optical depth, (both a function of weather conditions), (I) the extraterrestrial solar radiation, which is computed as follows:

$$I = I_o\left[1 + 0.034 \cdot Cos\left(\frac{360 \cdot n}{365.25}\right)\right] \qquad [6.9]$$

Where I_o = 1367 W/m^2 is the solar constant (total energy intensity measured just outside the earth's atmosphere) and (n) is the day number corresponding to the date under consideration. Example for January 1st, n = 1.

From equation [6.1], $I_{d,c}$ is the diffused radiation and is given by:

$$I_{d,c} = C \cdot I_{b,N} \cdot Cos^2(\beta/2) \qquad [6.10]$$

Where (C) is the sky diffusion factor for the month in question (function of weather conditions), (β) is the tilt angle (angle of inclination) of the solar vault glazing (recommended: Latitude of area + 15^o). From equation [6.1] ($I_{r,c}$) is the ground – reflected solar radiation and is given by:

$$I_{r,c} = \rho \cdot I_{b,N} \cdot (Sin\alpha + C) \cdot Sin^2(\beta/2) \qquad [6.11]$$

Where (ρ) is the ground reflectance and depends on the surrounding vegetation.

Therefore using equations [6.1 – 6.11] the total solar radiation on the vault can be calculated for every hour of every day of the year, for any location in the world.

6.2.3.1 Solar insolation and climatic data for study village

Solar tables were developed for Paquila, Guatemala (latitude 14.53 ^{o}N longitude 91.51 ^{o}S). An excerpt from solar tables showing hourly solar insolation for 1 year on surfaces inclined at various angles and facing different directions that were developed in EXCEL® using equations [6.1 – 6.11] is given in Figure 6.8. The calculations were compared with NASA's 22 – years monthly averages for accuracy (NASA, 2008). Data for a south – facing surface, inclined at 29.53^o was abstracted from the tables, while temperature, cloud cover, number of clear sky and no sun days, and rainfall data were retrieved from the NASA website (NASA, 2008). From the data, it was determined that the months of May to August had the lowest average solar radiation, zero days of average clear sky days, the highest number of black days and highest rainfall amounts.

Average temperatures were not significantly different from the rest of the year. Data for this four – month period were used for model simulation. The complete data set for solar insolation is given in the Appendix C.

Solar Position and Insolation Values for 14.53 Degrees North Latitude (Paquila, Guatemala)

Date	Solar Times		Solar Position				$I_{b,N}$	I_h	South					South-East					East				
	h_{sr}	h_{ss}	hr	h_s	α	a_s	(W/m²)	(W/m²)	4.5	14.5	24.5	29.5	90.0	4.5	14.5	24.5	29.5	90.0	4.5	14.5	24.5	29.5	90.0
1-May	5:44 AM	6:15 PM	3:00	-135	0.0	136.9	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			4:00	-120	0.0	123.2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			5:00	-105	0.0	111.0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			6:00	-90	3.7	104.4	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
			7:00	-75	17.9	101.3	252	108	104	93	81	74	26	118	139	156	163	159	126	164	198	213	261
			8:00	-60	32.2	98.7	510	333	327	308	282	266	64	352	389	415	424	320	366	432	486	509	491
			9:00	-45	46.6	96.5	659	558	552	530	495	473	96	579	614	633	637	378	592	656	703	720	546
			10:00	-30	61.0	94.6	742	739	735	712	671	644	119	755	777	780	773	352	765	809	832	835	477
			11:00	-15	75.5	93.4	785	855	852	829	785	755	133	863	865	844	826	264	868	881	871	858	329
			12:00	0	89.6	180.0	798	895	892	869	824	794	138	892	870	825	795	138	893	871	827	797	138
			13:00	15	75.5	93.4	785	855	852	829	785	755	133	863	865	844	826	264	868	881	871	858	329
			14:00	30	61.0	94.6	742	739	735	712	671	644	119	755	777	780	773	352	765	809	832	835	477
			15:00	45	46.6	96.5	659	558	552	530	495	473	96	579	614	633	637	378	592	656	703	720	546
			16:00	60	32.2	98.7	510	333	327	308	282	266	64	352	389	415	424	320	366	432	486	509	491
			17:00	75	17.9	101.3	252	108	104	93	81	74	26	118	139	156	163	159	126	164	198	213	261
			18:00	90	3.7	104.4	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
			19:00	105	0.0	111.0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			20:00	120	0.0	123.2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			21:00	135	0.0	136.9	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			Surface Daily Totals				6695	6080	6032	5815	5451	5218	1013	6227	6436	6481	6440	3083	6327	6755	7007	7065	4348

Figure 6.8: Excerpt from solar insolation tables for Paquila showing data for May 1

6.2.4 Heating and microbial inactivation model development and performance

6.2.4.1 Microbial quality requirements

The microbial standard for Class A Biosolids from the US EPA's Part 503 Biosolids Rule was used as the bench mark for effluent quality. The rule requires that biosolids to be applied to land must undergo treatment that reduces pathogenic bacteria, enteric viruses and viable helminth ova (US EPA, 1992). The microbial criteria for Class A Biosolids are listed in Table 6.2 and were chosen because once achieved there is no public entry or crop harvest restriction requirement after land application (Lewis and Gattie, 2002).

Table 6.2: Criteria for meeting Class A requirements (US EPA, 1992)

Parameters	Limit	Units
Total fecal coliform	1000	Most Probable Number (MPN)/g Total Solid (TS, dry weight)
Salmonella	3	MPN/4g TS
Enteric viruses	<1	Plaque Forming Units (PFU)/4g TS
Helminth/Protozoa	<1	Ova/4g TS

6.2.4.2 Process criteria requirements

The eggs of helminthes are very resistant to environmental insults (Verle *et al.*, 2003). For example, research has shown that the eggs of *Ascaris* can withstand temperature ranges 60 – 65 ^{o}C and have been know to remain viable in soil for up to 15 years (Bird and Mcclure, 1976; Fairbairn, 1957; Komiya and Kobayashi, 1965; Wharton, 1979). Recommendations for excreta recycling from double vault latrines in Guatemala have been at least 18 months at temperatures 18 – 20 ^{o}C (Strauss, 1991).

Ascaris was therefore the indicator organism – of – choice for this research. Under laboratory conditions heating to 60 ^{o}C for 3 – 5 minutes was shown to destroy all eggs (Arfaa, 1984). From Figure 6.9, it was determined that to achieve Class A requirement for this parameter, a minimum retention time should be about 1 month with a temperature 45 ^{o}C. Due to heterogeneity of latrine contents, uncertainty in weather conditions and diurnal variations in solar insolation, a minimum processing time of 4 months with temperatures up to a maximum of 65 ^{o}C was targeted. The underlying assumptions are: these conditions are favorable for the inactivation of *Ascaris'* eggs, if they are destroyed then other pathogenic organisms will be too and thus, microbial quality of the “humanure” can be sufficiently assured for agricultural purposes.

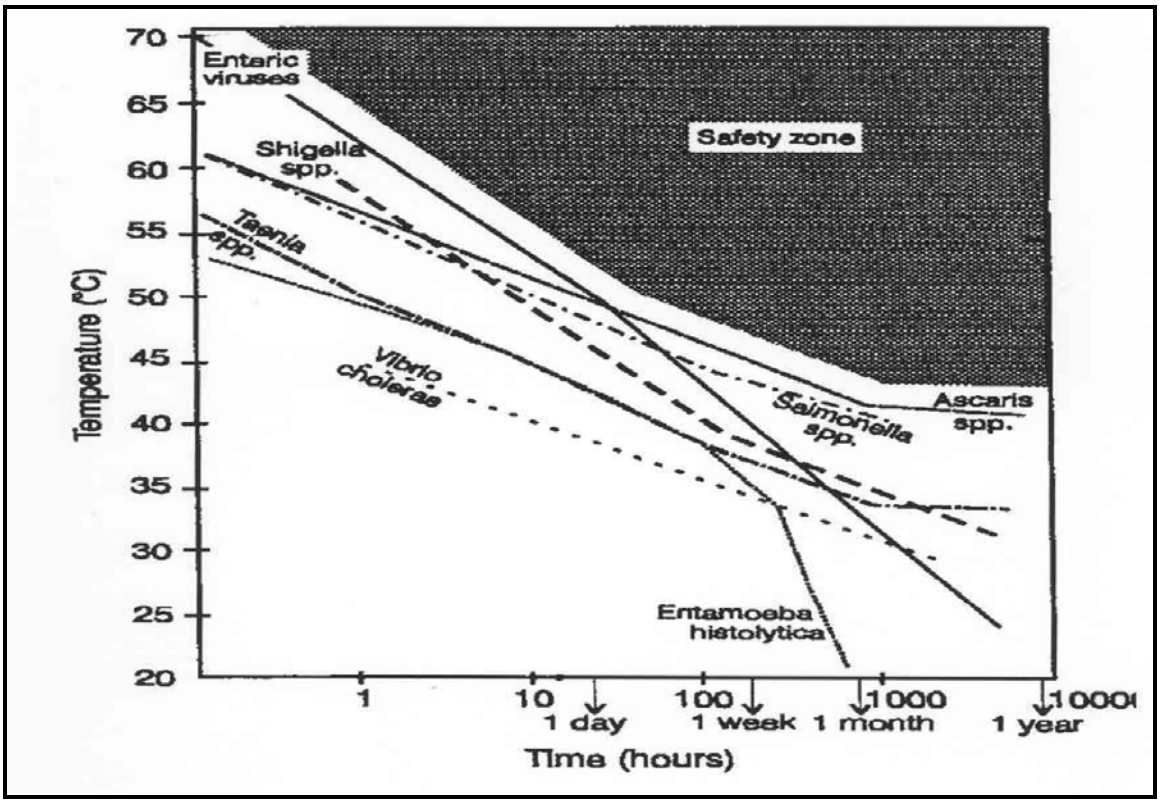

Figure 6.9: Processing time required to deactivate microorganisms at specific temperatures (Feachem et al., 1983)

6.2.5 Numerical methods

The aim was to model the thermal sanitation of the excreta, to determine the temperature profiles and microbial concentration as a function of treatment time. The problem was set up as a 2D symmetrical transient heat conduction problem for temperature with transport for the destruction of microbes. Two differential equations, connected by the temperature changes in the product, were solved simultaneously in the model, one for heat transfer (equation [6.12]) and one for microbial transport (equation 6.15]) using the Finite Element Method. To make use of the symmetry of the container, only half the length of the SPT was modeled (Figure 6.10). The boundary conditions were represented by the convective flux of solar radiation through the vault glazing. All other boundaries were considered to be insulated (Thorvaldsson and Janestad, 1999).

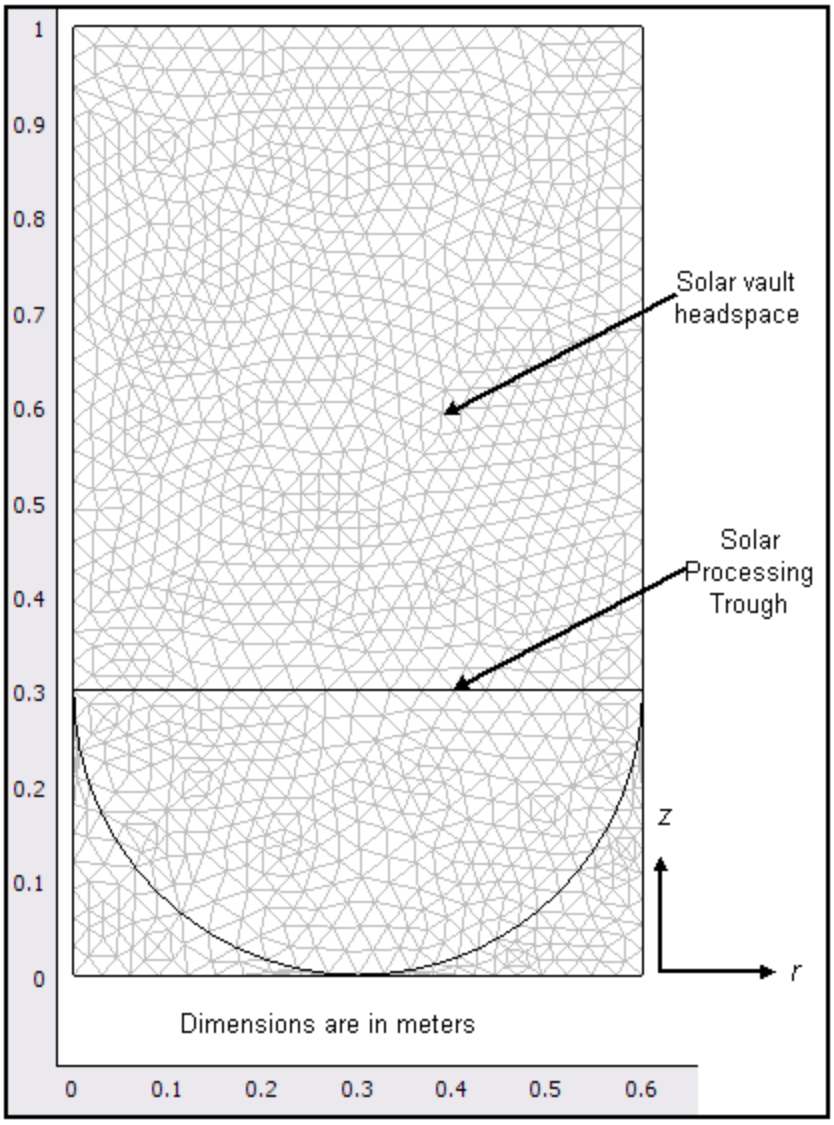

Figure 6.10: Solar Processing Trough as modeled in COMSOL® showing finite element's mesh

Table 6.3: Nomenclature used in numerical modeling

Symbols	Description	Units
T	Temperature of excreta in SPT	K
k	Thermal conductivity of drum material	W/m/K
ρ	Density of fecal matter	kg/m^3
C_p	Specific heat at constant pressure of fecal matter	J/kg/K
c_A	Concentration of *Ascaris*' eggs in fecal matter	(# of microbes)/kg
D	Diffusivity of *Ascaris*' eggs	m^2/s
r_A	Microbial inactivation rate	mol(#)/m^3/s
E_a	Activation energy	kJ/mol
R	Gas constant	J/K/mol
q_o	Inward heat flux	W/m^2
h_c	Convective heat transfer coefficient of vault air	W/m^2/K
T_{inf}	Atmospheric temperature (outside vault)	K
U_o	Overall coefficient of heat transfer	W/m^2/K
A	Overall area of the vault	m^2

6.2.5.1 Heat transfer

Equation [6.12] was derived from Fourier's Law for heat conduction to determine the energy balance over a reference element in the product. The temperature *T(r, z, t)* at position *(r, z)* at time *t* was calculated as:

$$\frac{\partial T}{\partial t} = \frac{k}{\rho C_p}\left(\frac{1}{r}\frac{\partial}{\partial r}\left(r\frac{\partial T}{\partial r}\right) + \frac{\partial^2 T}{\partial z^2}\right) \qquad [6.12]$$

The following boundary conditions were applied:

$$k\frac{\partial T}{\partial t} = q_o + h_c(T_{inf} - T) \qquad [6.13]$$

The inward heat flux, (q_o), is the net of the solar radiation through glazing and the convection gains or losses between vault envelope and ambient air due to temperature differences. This was determined from the following equation (see Table 6.4 below for definitions):

$$q_o = SHGC * solar\_flux(t) + U_o A\left(T_{inf} - T\right) \quad [6.14]$$

6.2.5.2 Microbial inactivation

Equation [6.15] was derived from Fick's Law for mass diffusion to determine concentration of microbes over a reference element in the product. *Ascaris*' eggs are non – motile and thus their diffusivity was set to zero, and all boundaries are considered insulated towards diffusion. The microbial concentration *c(r, z, t)* at position *(r, z)* at time, (*t*) was calculated as:

$$\frac{\partial c}{\partial t} = D\left(\frac{1}{r}\frac{\partial}{\partial r}\left(r\frac{\partial T}{\partial r}\right) + \frac{\partial^2 T}{\partial z^2}\right) - r_A$$
$$r_A = k_1 c_A e^{-\frac{E_a}{RT}} \quad [6.15]$$

6.2.5.3 Model simulation

2D heat and mass transfer was modeled by solving equations [6.12 – 6.15] numerically using an unconditionally stable Finite Element Method, Implicit (backward) Euler (Thorvaldsson and Janestad, 1999). The total heating time was 4 months (2952 hours) with time step size 1 second (varying the time step did not cause significant deviations in the results). Input variables are listed in Table 6.4:

Table 6.4: COMSOL® input variables

Variable	Definition	Values
$T(t_o)$	Initial temperature of atmosphere and vault content	295 [K]
T_{inf}	22 – years of average hourly atmospheric temperature	Text file [K] (NASA, 2008)
k	Thermal conductivity of drum material	0.55 [W/m/K]
ρ	Density of fecal matter	1000 [Kg/m$^{3]}$
C_p	Specific heat at constant pressure of material	4200 [J/Kg/K]
E_a	Activation energy	1.6 *10^5 [KJ/mol]
D	Diffusivity of *Ascaris*' eggs	0 [m^2/s]
R	Gas constant	8.314 [KJ/mol/K]
k_1	Decay rate of microbes	2.31 x 10^{21} [/s]
h_c	Convective heat transfer coefficient	2.36 [W/m^2/K] (Axaopoulos *et al.*, 2001)
SHGC	Solar heat gain coefficient for glazing	0.85 [ ] (ASHRAE, 1997)
solar_flux	Hourly solar insolation on inclined surface, (I_c)	Text file [W/m^2]
U_o	Heat loss coefficient of concrete envelope of vault	0.44 [KW/m^2/K] (Axaopoulos *et al.*, 2001)
A	Area of vault envelope	1.44 [m^2]

6.2.5.4 Results and discussion

Figure 6.11 shows the temperature fronts at the end of the simulation. Temperatures ranged from 295 – 343 K (22 – 70 ^{o}C), with an average of 331K (55 ^{o}C) at location (0.3, 0.15) of SPT. The diurnal variation in the solar flux drove the temperature variation which is illustrated by Figure 6.12. There was a 2 – day lag before required treatment temperatures (55 – 65 ^{o}C) were achieved.

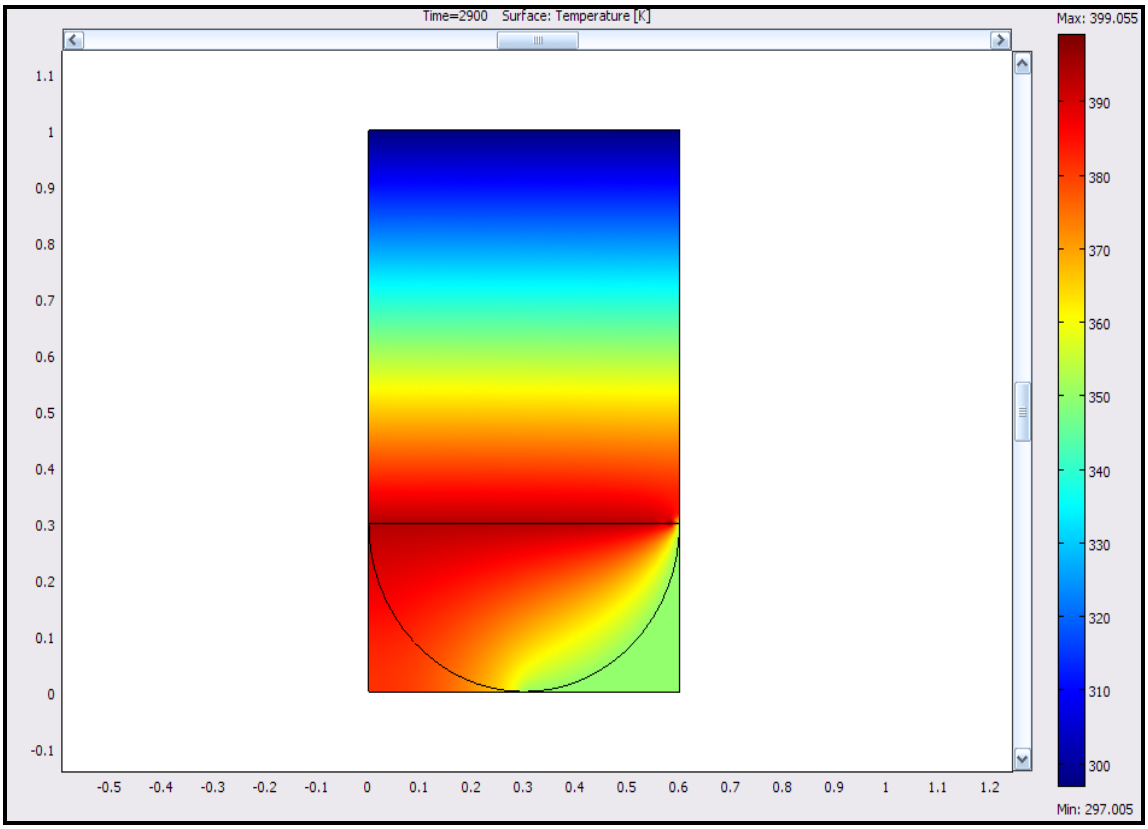

Figure 6.11: Surface plot showing temperature fronts at time t = 2900 hours

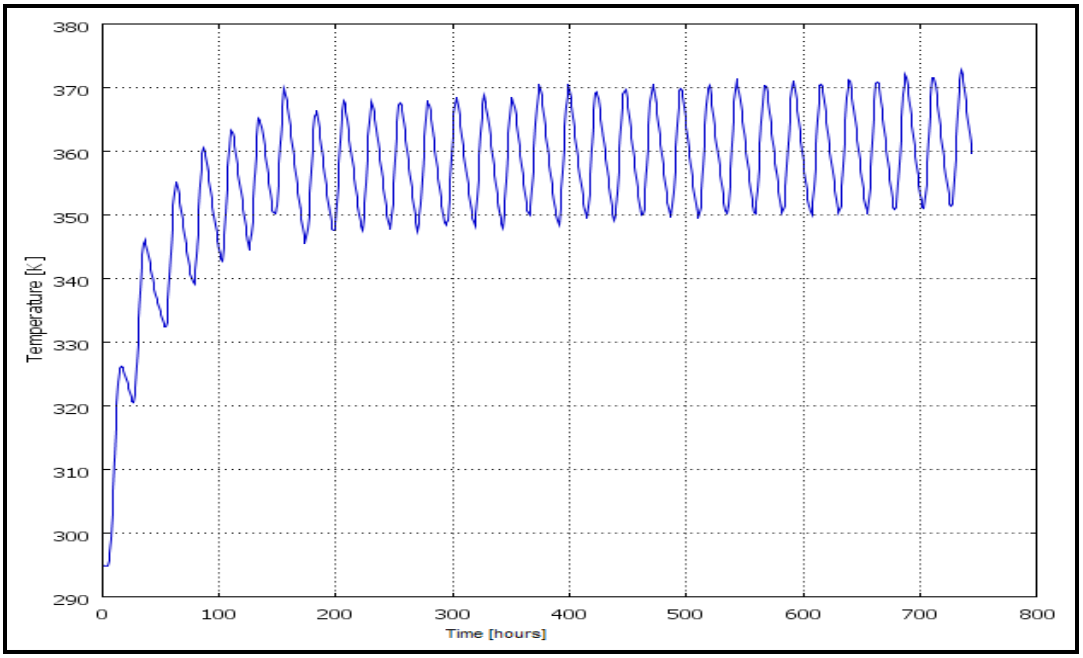

Figure 6.12: Temperature variation at location (0.3, 0.15) of SPT

At the temperatures that were achieved in the SPT it required at least 3 days before the organisms were totally inactivated and Class A status could be achieved as shown in Figure 6.13. The results of the above simulations indicate that the proposed design is able to safely contain and treat excreta to obtain a parasite free product. Quality assurance can be even better if ashes are also added when available. In Japan, where one of the most successful infectious disease program was implemented, sodium nitrite (ovicide) and calcium superphosphate are added to excreta (buffer and fertilizer) to increase egg die off (Komiya and Kunii, 1964).

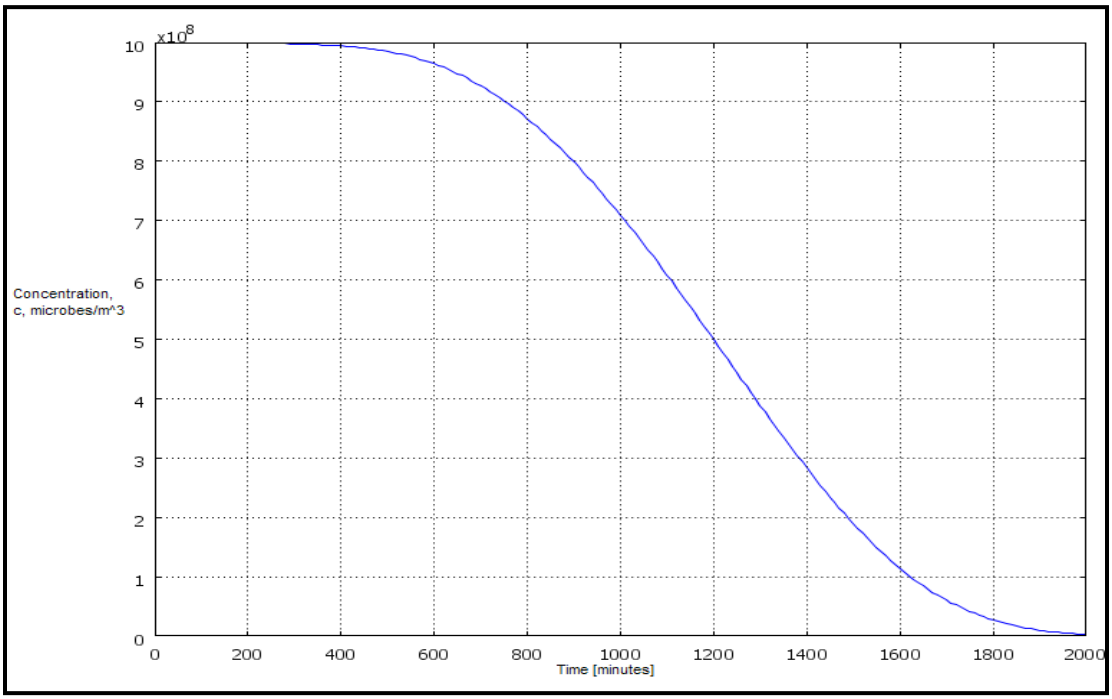

Figure 6.13: Concentration of microbes at (0.3, 0.15) as function of time

One of the most effective methods of controlling infectious diseases is to interrupt the developmental cycle and transmission routes of the pathogenic organisms (Webber and Rutala, 2001). Safe stool disposal will keep parasites out of the domestic area frequented by children, while treating excreta before it is used in crop production will

overtime eliminate the other transmission routes. However, fulfilling this mandate is challenging due to high prevalence rates and the subsequently high concentrations of pathogens that must be inactivated before reuse is possible. In addition the installed systems need to be low cost, very easy and simple to operate in order to be sustainable. The proposed modified Solar Latrine design fulfilled these requirements:

- The process is economically sustainable because it utilizes a renewable and "freely" available source of energy. Compared to traditional pit latrine the investment is not significantly more and the system pays for itself both financially (reduced the need for commercial fertilizers) and socially (reduced morbidity and mortality) (Eggers – Lura, 1979),
- Makes use of a technology that is already being use, is embedded into the culture and is thus familiar to individuals. Reduces learning curve and cognitive dissonance associated with learning a new skill, and self efficacy is already in place,
- Tackles both public health issues to improve nutritional status while preventing infectious diarrheal diseases,
- The SPT significantly limits the contact between human beings and the hazardous material and makes for easy handling and transportation,
- This design can be retrofitted to existing latrines, that is, it can be used to update earlier models,
- In tropical climates there is on average three crop cycles throughout the year. This scheme matches the agronomic rates so farmers are less likely to use unsanitized "humanure" (Jensen et al., 2005),
- Innovation can be married to community economy which will increase the likelihood of success and sustainability (create a labor market for excreta

collection and storage, latrine construction, etc), From an economical perspective the process is virtually volume independent (Caslake et al., 2004), and

- Unlike synthetic fertilizers (which do not improve soil structure), the nutrients in night – soil are slowly released over time, thereby reducing the likelihood of nitrogen and phosphorous groundwater contamination. It also contains an organic carbon fraction which improves soil structure (Jimenez et al., 2006).

6.2.6 Summary

In general, however, latrines are usually abandoned once filled (Simms *et al.*, 2005). This has caused reintroduction of communities into the class of persons "without access to improved sanitation" and destruction of the latrines as farmers try to get to the contents (Jensen *et al.*, 2005). The results showed that there is an initial time lag of about 2 days before desired treatment temperatures (55 – 65 ^{o}C) were achieved. Under average solar insolation conditions, the microbial concentration in a family's 170 kg quarterly output can be lowered to US EPA Class A Biosolids levels. May to August is considered the worse solar insolation period and it is from this period that data was abstracted to input into the heating and inactivation model. A 4 – month retention time is recommended due to uncertainties in weather conditions especially during the rainy season, which was modeled here. Even so, this is significantly less than the 12 – 18 months currently prescribed for other latrine systems.

6.3 STELLA® modeling of solar latrine and integrated intervention

6.3.1 Solar Latrine intervention

Table 6.5: Symbols used in modeling Solar Latrine intervention

Reference symbol	Description	Units
c_w	Excessive egg deaths due to latrine	1/time
g_w	Number of community member using latrine over each retention time	host/time
h_w	Kill rate of latrine per dose; proportion of worms inactivated	egg/egg/host

6.3.1.1 Evaluating the stationary egg population assumption

In Chapter 4, the assumption that the infective egg population does not change over time because of the relative differences in the life expectancies among the three populations. As a result the differential equation for the egg population was subsumed into that of the parasite. For this simulation each population is considered separately. Therefore the first trial was to determine if the assumption held. Figures 6.14 – 6.16 show the populations separated:

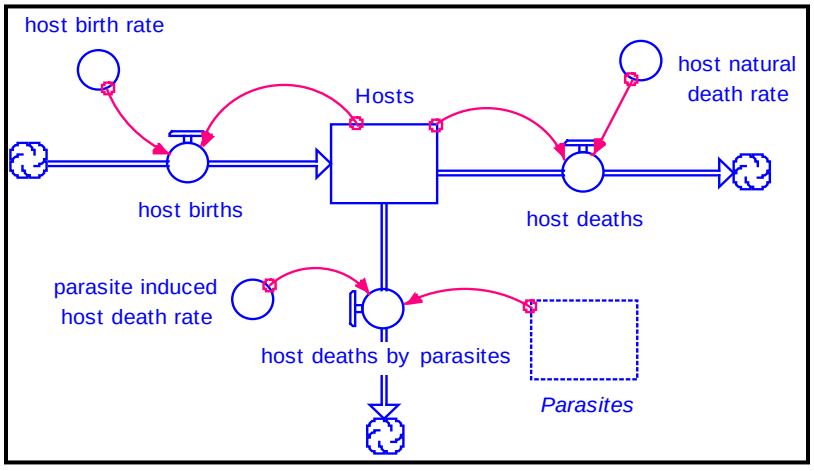

Figure 6.14: STELLA® model of host population with all three populations separated

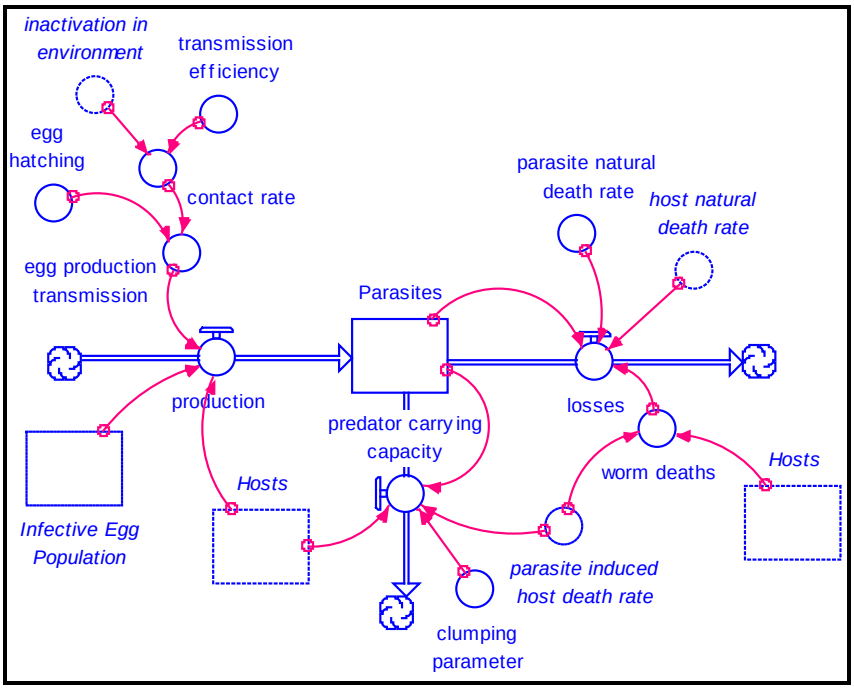

Figure 6.15: STELLA® model of parasite population with all three populations separated

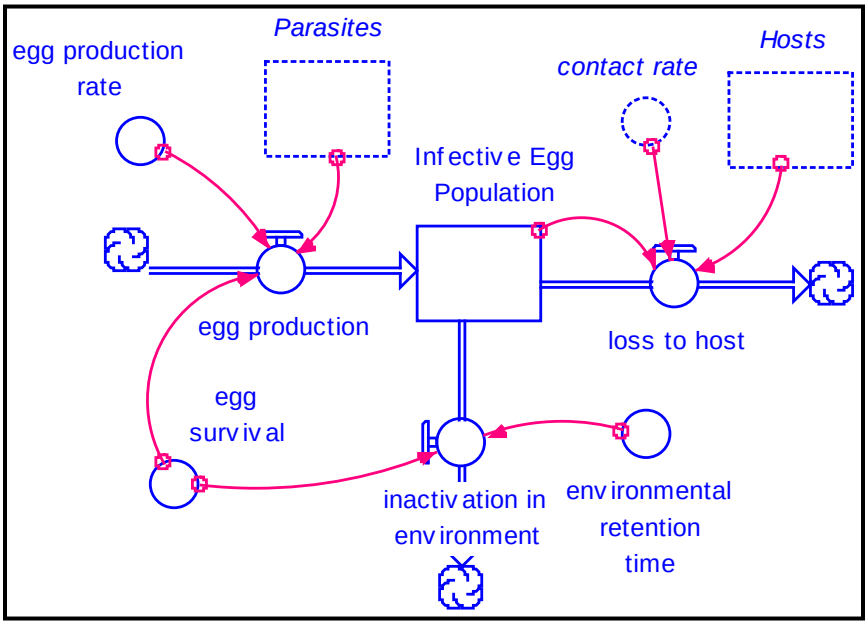

Figure 6.16: STELLA® model of egg population with all three populations separated

6.3.1.2 Results and discussion of stationary egg population assumption

From the results in Table 6.6, the final host and mean worm burden are very similar. In the early time periods, there were major differences, but over time equilibrium was established as the rate of change in the egg population goes to zero. Thus, the assumption was indeed accurate and thus previous interventions can be modeled using this method with comparisons possible.

Table 6.6: Comparison of the host population and mean worm burden dynamics in response to assumption

Time (years)	Host population – assumption	Mean worm burden – assumption	Host population – no assumption	Mean worm burden – no assumption	Egg population
0	3,500.00	2.00	3,500.00	2.00	0
1	3,582.59	21.20	3,583.04	9.48	261878.03
2	3,655.87	133.58	3,665.83	31.61	891526.99
3	3,714.39	164.12	3,743.63	90.71	3000086.57
4	3,772.43	164.59	3,810.59	147.68	8420227.56
5	3,831.36	164.64	3,871.58	163.82	13418858.83
6	3,891.19	164.69	3,931.78	167.08	14935372.21
7	3,951.95	164.74	3,992.58	167.70	15245236.81
8	4,013.65	164.79	4,054.24	167.81	15302735.71
9	4,076.31	164.83	4,116.85	167.83	15313012.59
10	4,139.93	164.88	4,180.42	167.84	15314801.11
11	4,204.53	164.92	4,244.97	167.84	15315104.74
12	4,270.14	164.97	4,310.52	167.84	15315155.02
13	4,336.76	165.01	4,377.08	167.84	15315163.15
14	4,404.41	165.05	4,444.67	167.84	15315164.43
15	4,473.10	165.10	4,513.30	167.84	15315164.62

6.3.1.3 Solar Latrine intervention

Modeling a Solar Latrine intervention in this manner is entirely new and there was no precedence in literature. Therefore, the effect of the latrine intervention was conceptualized in the following manner:

- The Solar Latrine's vault is acting as a chemotherapeutic agent, but instead of worms, the target is the eggs,
- If people are assumed to use the latrine randomly (similar to choosing members to treat randomly), then the excess deaths among the egg population (c_w) is given by an equation similar to that equation [4.56] for parasites. Where (h_w) is the efficacy of the latrine in deactivating the eggs (assumed to be 99% effective) and (g_w) is the number of persons using the latrine over a treatment period,

$$c_w = -\ln(1 - g_w h_w) \qquad [6.16]$$

- Therefore the solar rate (Figure 6.17) is analogous to the "chemo rate" used for parasites and is the rate at which the latrines remove infective eggs from the environment.

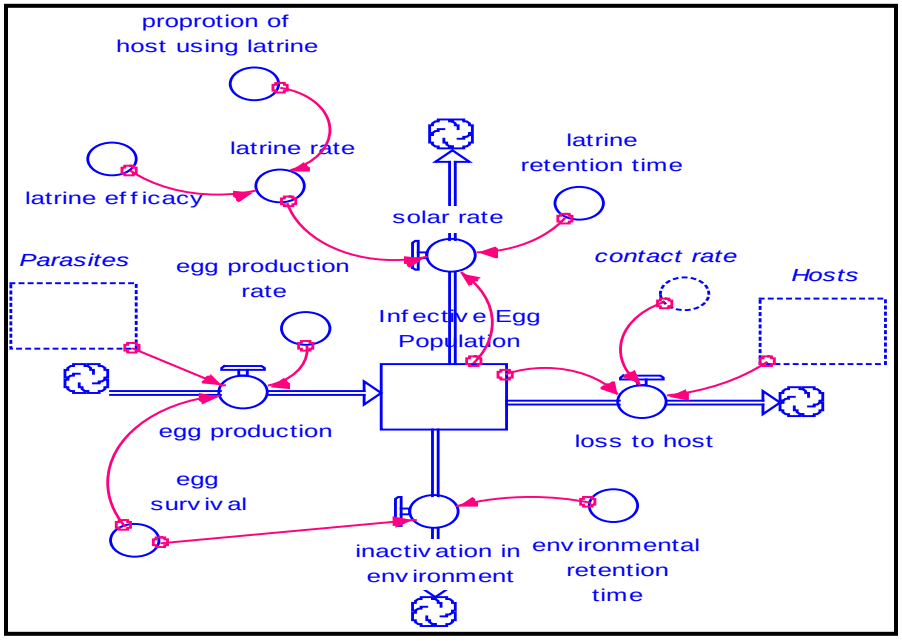

Figure 6.17: STELLA® model of egg population with latrine intervention

6.3.1.4 Results and discussion of latrine intervention

A variable environmental retention time was added to the model to indicate the life expectancy of the infective stage in the environment and was taken to be about 1 ½ months (0.125). Previously, this was assumed to be 0.1 years, but due to software idiosyncrasies (time steps needing to be $1/2^n$), this value was chosen. The model was run with all the default values previously used, but for differing number of host using the system. From the results in Figure 6.18 and Tables 6.7 and 6.8, the minimum number of persons required to use the latrine system for eradication to be possible is about 70%, with significant changes in mean worm burdens occurring at 30%. This agrees with (Muller *et al.*, 1989) who found that at least 20% of household population needs to use latrine to make any difference in fecal contamination.

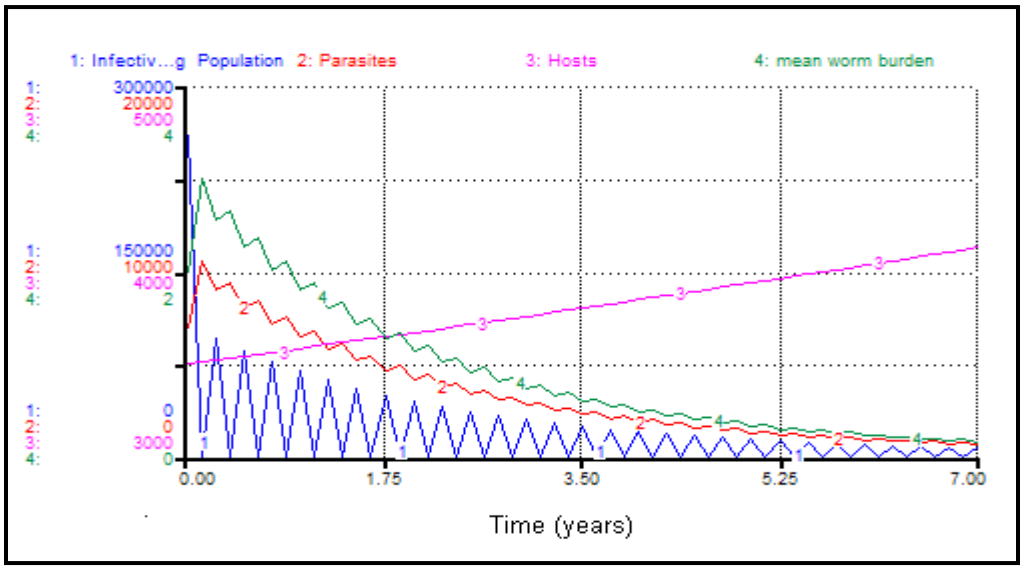

Figure 6.18: Infective egg, worm and host population and mean worm burden to latrine intervention

Table 6.7: Response of mean worm burden to different rates of population use in latrine intervention

Time (years)	Mean worm burden 0% using latrines	Mean worm burden 10% using latrines	Mean worm burden 30% using latrines	Mean worm burden 50% using latrines	Mean worm burden 70% using latrines	Mean worm burden 90% using latrines
0	2.00	2.00	2.00	2.00	2.00	2.00
1	9.48	8.22	5.83	3.67	2.22	1.80
2	31.61	24.14	12.60	5.32	1.88	1.14
3	90.71	65.69	27.12	7.82	1.61	0.73
4	147.68	121.86	54.83	11.64	1.40	0.48
5	163.82	144.19	89.34	17.44	1.23	0.32
6	167.08	148.90	108.66	26.03	1.10	0.22
7	167.70	149.91	114.44	37.88	0.99	0.15
8	167.81	150.26	116.11	51.90	0.91	0.11
9	167.83	150.48	116.88	64.73	0.84	0.08
10	167.84	150.68	117.46	73.40	0.79	0.06
11	167.84	150.87	118.00	78.08	0.75	0.04
12	167.84	151.05	118.52	80.51	0.73	0.03
13	167.84	151.24	119.03	81.96	0.71	0.03
14	167.84	151.41	119.52	83.02	0.70	0.02
15	167.84	151.59	120.00	83.92	0.70	0.02

Table 6.8: Response of host population to different rates of population use in latrine intervention

Time (years)	Host 0% using latrines	Host 10% using latrines	Host 30% using latrines	Host 50% using latrines	Host 70% using latrines	Host 90% using latrines
0	3,500.00	3,500.00	3,500.00	3,500.00	3,500.00	3,500.00
1	3,583.04	3,583.10	3,583.24	3,583.38	3,583.47	3,583.50
2	3,665.83	3,666.48	3,667.60	3,668.48	3,669.00	3,669.14
3	3,743.63	3,746.95	3,752.10	3,755.22	3,756.63	3,756.93
4	3,810.59	3,819.15	3,834.66	3,843.42	3,846.41	3,846.89
5	3,871.58	3,884.37	3,912.58	3,932.75	3,938.36	3,939.05
6	3,931.78	3,948.11	3,986.09	4,022.71	4,032.55	4,033.44
7	3,992.58	4,012.34	4,058.31	4,112.60	4,129.01	4,130.11
8	4,054.24	4,077.48	4,131.06	4,201.67	4,227.81	4,229.11
9	4,116.85	4,143.61	4,204.83	4,289.58	4,328.98	4,330.50
10	4,180.42	4,210.76	4,279.77	4,376.75	4,432.59	4,434.32
11	4,244.97	4,278.96	4,355.90	4,464.07	4,538.69	4,540.63
12	4,310.52	4,348.21	4,433.24	4,552.26	4,647.34	4,649.50
13	4,377.08	4,418.54	4,511.83	4,641.69	4,758.59	4,760.97
14	4,444.67	4,489.96	4,591.67	4,732.55	4,872.51	4,875.12
15	4,513.30	4,562.48	4,672.79	4,824.91	4,989.15	4,992.01

6.3.2 Simultaneous Solar Latrine and chemotherapy interventions

The next step in the modeling process was to add chemotherapy. At first only 27% of the population was treated every 3 months with Albendazole (94% efficacy) for 2 years. The model was then simulated with the proportions of host using the latrine as given above. The results are given in Tables 6.9 and 6.10. The model was then run assuming 50% of the population was treated. These results were given in Tables 6.11 and 6.12.

6.3.2.1 Results and discussion for simultaneous latrine and chemotherapy interventions

When both interventions were employed the minimum number of persons required to use the latrine system for eradication to be possible is dropped from 70% to 50%, with significant changes in mean worm burdens again occurring at 30%, and total eradication occurring in about 6 years at 90% toilet usage. When the number of hosts treated was increased to 50%, the required percent usage dropped to 30% from 50%, however, the ultimate worm burden rebounded to pre – control levels. Total eradication was now possible at 70% toilet utilization in as little as 2 years. Significant changes in the number of hosts saved as a result of the addition chemotherapy occurred at lower usages. When the majority of the population started using the latrines, chemotherapy showed a smaller impact on the host's life expectancy.

Table 6.9: Response of mean worm burden to different rates of population use in latrine intervention with 27% of host receiving chemotherapy

Time (years)	Mean worm burden 0% using latrines	Mean worm burden 10% using latrines	Mean worm burden 30% using latrines	Mean worm burden 50% using latrines	Mean worm burden 70% using latrines	Mean worm burden 90% using latrines
0	2.00	2.00	2.00	2.00	2.00	2.00
1	4.99	4.17	2.68	1.45	0.73	0.54
2	8.75	6.14	2.59	0.80	0.19	0.09
3	30.49	18.88	5.82	1.20	0.16	0.06
4	89.14	53.83	12.92	1.79	0.14	0.04
5	147.52	112.87	28.54	2.73	0.12	0.03
6	163.92	142.51	58.54	4.21	0.11	0.02
7	167.12	148.90	93.92	6.59	0.10	0.01
8	167.71	150.18	111.50	10.44	0.09	0.01
9	167.82	150.56	116.27	16.67	0.08	0.01
10	167.83	150.78	117.62	26.47	0.08	0.00
11	167.84	150.97	118.29	40.65	0.08	0.00
12	167.84	151.15	118.83	57.44	0.07	0.00
13	167.84	151.34	119.33	71.54	0.07	0.00
14	167.84	151.51	119.82	79.61	0.07	0.00
15	167.84	151.68	120.30	83.28	0.07	0.00

Table 6.10: Response of host population to different rates of population use in latrine intervention with 27% of host receiving chemotherapy

Time (years)	Host 0% using latrines	Host 10% using latrines	Host 30% using latrines	Host 50% using latrines	Host 70% using latrines	Host 90% using latrines
0	3,500.00	3,500.00	3,500.00	3,500.00	3,500.00	3,500.00
1	3,583.34	3,583.39	3,583.48	3,583.58	3,583.64	3,583.66
2	3,668.18	3,668.47	3,668.95	3,669.31	3,669.50	3,669.54
3	3,753.08	3,754.42	3,756.20	3,757.11	3,757.45	3,757.51
4	3,833.00	3,838.37	3,844.62	3,846.91	3,847.51	3,847.60
5	3,901.74	3,914.72	3,933.05	3,938.71	3,939.74	3,939.85
6	3,964.18	3,982.62	4,019.11	4,032.47	4,034.18	4,034.31
7	4,025.82	4,048.12	4,099.84	4,128.06	4,130.89	4,131.04
8	4,088.06	4,113.94	4,176.01	4,225.28	4,229.92	4,230.09
9	4,151.20	4,180.65	4,251.13	4,323.71	4,331.32	4,331.52
10	4,215.30	4,248.39	4,326.92	4,422.68	4,435.16	4,435.38
11	4,280.39	4,317.17	4,403.83	4,521.17	4,541.49	4,541.73
12	4,346.49	4,387.01	4,481.95	4,618.09	4,650.37	4,650.63
13	4,413.60	4,457.94	4,561.31	4,713.11	4,761.85	4,762.14
14	4,481.75	4,529.97	4,641.94	4,807.13	4,876.01	4,876.32
15	4,550.96	4,603.12	4,723.86	4,901.46	4,992.91	4,993.25

Table 6.11: Response of mean worm burden to different rates of population use in latrine intervention with 50% of host receiving chemotherapy

Time (years)	Mean worm burden 0% using latrines	Mean worm burden 10% using latrines	Mean worm burden 30% using latrines	Mean worm burden 50% using latrines	Mean worm burden 70% using latrines	Mean worm burden 90% using latrines
0	2.00	2.00	2.00	2.00	2.00	2.00
1	2.16	1.69	0.90	0.35	0.11	0.06
2	1.64	1.01	0.29	0.05	0.00	0.00
3	6.15	3.27	0.67	0.07	0.00	0.00
4	21.12	9.93	1.50	0.10	0.00	0.00
5	67.59	29.97	3.39	0.16	0.00	0.00
6	136.64	79.98	7.77	0.24	0.00	0.00
7	161.86	132.02	17.91	0.38	0.00	0.00
8	166.79	147.37	40.25	0.61	0.00	0.00
9	167.66	150.22	77.74	0.99	0.00	0.00
10	167.81	150.84	107.28	1.63	0.00	0.00
11	167.83	151.09	116.71	2.71	0.00	0.00
12	167.84	151.29	118.92	4.59	0.00	0.00
13	167.84	151.46	119.70	7.87	0.00	0.00
14	167.84	151.64	120.23	13.62	0.00	0.00
15	167.84	151.81	120.70	23.54	0.00	0.00

Table 6.12: Response of host population to different rates of population use in latrine intervention with 50% of host receiving chemotherapy

Time (years)	Host 0% using latrines	Host 10% using latrines	Host 30% using latrines	Host 50% using latrines	Host 70% using latrines	Host 90% using latrines
0	3,500.00	3,500.00	3,500.00	3,500.00	3,500.00	3,500.00
1	3,583.57	3,583.60	3,583.66	3,583.73	3,583.76	3,583.77
2	3,669.21	3,669.32	3,669.50	3,669.63	3,669.69	3,669.70
3	3,756.58	3,756.96	3,757.41	3,757.61	3,757.68	3,757.69
4	3,844.50	3,845.97	3,847.31	3,847.69	3,847.78	3,847.79
5	3,929.29	3,934.84	3,939.12	3,939.93	3,940.04	3,940.05
6	4,003.53	4,019.29	4,032.55	4,034.36	4,034.51	4,034.53
7	4,068.82	4,093.83	4,126.83	4,131.03	4,131.25	4,131.27
8	4,132.30	4,162.33	4,220.13	4,229.98	4,230.31	4,230.32
9	4,196.23	4,230.14	4,309.06	4,331.23	4,331.74	4,331.76
10	4,261.04	4,298.69	4,391.55	4,434.80	4,435.61	4,435.62
11	4,326.84	4,368.26	4,470.91	4,540.66	4,541.96	4,541.98
12	4,393.66	4,438.90	4,550.37	4,648.71	4,650.87	4,650.89
13	4,461.50	4,510.63	4,630.87	4,758.73	4,762.39	4,762.41
14	4,530.39	4,583.48	4,712.62	4,870.29	4,876.58	4,876.60
15	4,600.35	4,657.45	4,795.67	4,982.54	4,993.51	4,993.53

6.3.3 Integrated Solar Latrine, chemotherapy and nutrition interventions

The final step in the modeling process was to combine all three interventions. For the first iteration, 27% of the population was treated with anti – helminthic medication and nutrition was provided at the required amount of 1.1 g/kg/d. The proportion of the population receiving treatment was then increase to 50% with all other variables except the proportion of persons using latrines remained constant. The results are given in Tables 6.13 and 6.14, and Tables 6.15 and 6.16 respectively.

6.3.3.1 Results and discussion for simultaneous Solar Latrine, chemotherapy, and nutrition interventions

Once resources became a limiting factor through the fixed area of arable land counterintuitive results occurred. For example, even while providing optimal nutrition, the worm burden increased above previous numbers for those not having any latrine intervention. This is as a result of hosts dying as the carrying capacity of the land was reached and surpassed. Thus mean worm burden was reduced below 1 worm/host at 50% toilet usage and with a reduction in absolute ultimate value (mean worm burden 83.28 to 39.89), however, over 300 more hosts died as a result compared to when intervention with only Solar Latrine and chemotherapy. When the chemotherapy rate was increased to 50% the worm burden decreased by about 100% saving the lives of 271 individuals at 50% latrine usage.

Table 6.13: Response of mean worm burden to different rates of population use in latrine intervention with 27% of host receiving chemotherapy and all having optimal protein supplement

Time (years)	Mean worm burden 0% using latrines	Mean worm burden 10% using latrines	Mean worm burden 30% using latrines	Mean worm burden 50% using latrines	Mean worm burden 70% using latrines	Mean worm burden 90% using latrines
0	2.00	2.00	2.00	2.00	2.00	2.00
1	4.86	4.07	2.63	1.43	0.72	0.53
2	7.96	5.73	2.49	0.78	0.18	0.09
3	20.43	14.83	5.41	1.16	0.16	0.06
4	32.64	26.40	10.86	1.72	0.14	0.04
5	45.49	38.51	19.37	2.58	0.12	0.03
6	53.64	46.83	29.12	3.92	0.11	0.02
7	58.33	51.10	36.07	5.99	0.10	0.01
8	62.73	54.83	39.88	9.12	0.09	0.01
9	67.78	59.01	42.88	13.53	0.08	0.01
10	74.01	64.10	46.03	19.00	0.08	0.00
11	82.01	70.53	49.69	24.61	0.07	0.00
12	92.81	78.98	54.11	29.37	0.07	0.00
13	108.48	90.63	59.61	33.19	0.07	0.00
14	134.18	108.03	66.68	36.53	0.07	0.00
15	188.05	138.01	76.20	39.89	0.07	0.00

Table 6.14: Response of host population to different rates of population use in latrine intervention with 27% of host receiving chemotherapy and all having optimal protein supplement

Time (years)	Host 0% using latrines	Host 10% using latrines	Host 30% using latrines	Host 50% using latrines	Host 70% using latrines	Host 90% using latrines
0	3,500.00	3,500.00	3,500.00	3,500.00	3,500.00	3,500.00
1	3,554.53	3,556.70	3,561.25	3,566.10	3,569.26	3,569.92
2	3,596.59	3,607.90	3,627.69	3,643.11	3,651.48	3,653.29
3	3,603.97	3,637.09	3,691.37	3,724.17	3,737.84	3,740.36
4	3,579.50	3,634.50	3,742.62	3,806.14	3,826.65	3,829.77
5	3,567.54	3,634.53	3,783.99	3,889.40	3,917.87	3,921.47
6	3,569.32	3,646.01	3,821.86	3,974.35	4,011.48	4,015.43
7	3,571.04	3,655.78	3,855.48	4,060.24	4,107.46	4,111.68
8	3,577.25	3,669.25	3,886.59	4,144.07	4,205.77	4,210.25
9	3,589.18	3,687.76	3,919.98	4,224.32	4,306.46	4,311.19
10	3,607.40	3,711.87	3,957.15	4,299.80	4,409.58	4,414.55
11	3,632.44	3,742.07	3,998.65	4,370.60	4,515.19	4,520.40
12	3,664.96	3,778.90	4,044.83	4,438.60	4,623.34	4,628.78
13	3,705.84	3,823.04	4,096.08	4,506.37	4,734.10	4,739.77
14	3,756.38	3,875.37	4,152.83	4,575.93	4,847.52	4,853.42
15	3,818.72	3,937.23	4,215.62	4,648.45	4,963.66	4,969.79

Table 6.15: Response of mean worm burden to different rates of population use in latrine intervention with 50% of host receiving chemotherapy and all having optimal protein supplement

Time (years)	Mean worm burden 0% using latrines	Mean worm burden 10% using latrines	Mean worm burden 30% using latrines	Mean worm burden 50% using latrines	Mean worm burden 70% using latrines	Mean worm burden 90% using latrines
0	2.00	2.00	2.00	2.00	2.00	2.00
1	2.14	1.68	0.90	0.35	0.11	0.06
2	1.62	0.99	0.28	0.04	0.00	0.00
3	5.93	3.21	0.66	0.07	0.00	0.00
4	17.54	9.27	1.47	0.10	0.00	0.00
5	35.66	22.85	3.30	0.16	0.00	0.00
6	49.48	39.35	7.39	0.24	0.00	0.00
7	56.31	48.50	15.87	0.38	0.00	0.00
8	60.91	53.02	28.57	0.60	0.00	0.00
9	65.67	56.94	38.42	0.96	0.00	0.00
10	71.32	61.35	43.48	1.58	0.00	0.00
11	78.40	66.70	47.02	2.62	0.00	0.00
12	87.62	73.39	50.61	4.39	0.00	0.00
13	100.27	82.10	54.78	7.41	0.00	0.00
14	119.04	93.99	59.82	12.41	0.00	0.00
15	151.02	111.51	66.12	19.95	0.00	0.00

Table 6.16: Response of host population to different rates of population use in latrine intervention with 50% of host receiving chemotherapy and all having optimal protein supplement

Time (years)	Host 0% using latrines	Host 10% using latrines	Host 30% using latrines	Host 50% using latrines	Host 70% using latrines	Host 90% using latrines
0	3,500.00	3,500.00	3,500.00	3,500.00	3,500.00	3,500.00
1	3,565.70	3,567.19	3,570.27	3,573.44	3,575.33	3,575.64
2	3,638.74	3,643.75	3,651.95	3,657.92	3,660.78	3,661.24
3	3,706.02	3,719.56	3,736.66	3,745.25	3,748.53	3,749.02
4	3,745.64	3,782.04	3,821.23	3,834.61	3,838.40	3,838.91
5	3,755.16	3,822.68	3,904.65	3,926.06	3,930.43	3,930.96
6	3,758.92	3,845.38	3,985.66	4,019.67	4,024.66	4,025.21
7	3,761.31	3,859.72	4,060.46	4,115.46	4,121.16	4,121.73
8	3,765.92	3,871.97	4,118.99	4,213.25	4,219.98	4,220.56
9	3,775.92	3,888.13	4,161.57	4,312.92	4,321.16	4,321.76
10	3,792.23	3,909.61	4,198.82	4,414.25	4,424.77	4,425.39
11	3,815.37	3,936.94	4,237.83	4,516.87	4,530.86	4,531.50
12	3,845.91	3,970.55	4,280.80	4,620.10	4,639.50	4,640.15
13	3,884.52	4,010.96	4,328.40	4,722.87	4,750.74	4,751.41
14	3,932.13	4,058.81	4,381.04	4,823.53	4,864.65	4,865.34
15	3,990.11	4,114.96	4,439.10	4,919.99	4,981.30	4,982.00

6.4 Summary and conclusions

The aim of this chapter was to design a Solar Latrine and model the heating and microbial inactivation process occurring within, and model the response of the host and mean worm burdens to various combinations of Solar Latrine, chemotherapy and nutrition interventions. The following is a summary of the results obtained and a proposition of the most sustainable intervention strategy found:

- Solar Latrine design and process model
    - As designed, temperatures of up to 55 – 65 ^{o}C can be achieved and sustained in the solar vault, and
    - A four – month retention time is enough to produce US EPA Class A Biosolids from human excreta even under the most solar unfriendly conditions.
- Vertical and integrated intervention strategies
    - Vertical integration of individual strategies may not be enough to sustainably eradicate parasitic disease in an endemic community,
    - Combining strategies does not necessarily produce positive additive effects,
    - Eradication is possible if a least 50% of the host population were treated every 3 months for at least 2 years with a drug of at least 94% efficacy, latrine coverage and usage were at least 70%, and nutrition were provide at about 1.1 g protein per kg (human mass) per day, and
    - Family planning must also be promoted simultaneously.

7 CONCLUSIONS AND FUTURE STUDIES

7.1 Summary

Preventable infectious diarrheal diseases claim the lives of and cause a tremendous amount of morbidity in many children living in rural areas of developing countries. These diseases are primarily caused by parasitic organisms transmitted via the fecal – oral route. Poor protein nutrition weakens the immune system against infections and their associated morbidity. Given that these challenges have been successfully addressed in developed countries such as the United States, the main thrusts of this research were to determine if they might be similarly solved and what will it take to do so sustainably in the context of a developing community.

Current strategies include single vertical interventions that address individual causes such as nutritional deficiencies, poor sanitation and the pathogenic organisms. A community's health, however, results from a confluence of host – parasite population and biological processes that are facilitated by the physical and social environment in which they occur. This project proposes a systems approach instead. Models representing single and combined interventions were developed to determine if a systems approach could more sustainably eradicate endemic parasitic diseases from a rural community whose livelihood centered on agriculture. That is, the resistance of the model parasite, *Ascaris lumbricoides,* to various insults (chemotherapy, sanitation and nutrition intervention) was explored for Paquila, a rural and agricultural community located in the southwestern highlands of Guatemala.

7.2 Limitations and assumptions

The results produced here are limited by the scope and associated assumptions. These included:

- This study is limited to only those infectious disease that are transmitted via the fecal – oral route and are cause by parasitic microorganisms,
- Ascaris was used as an indicator organism because given its ability to resist environmental conditions and association with other diarrheal agents. However, this organism has its own biological characteristics that may not be applicable to all diarrhea causing pathogens,
- It was assumed that all Solar Latrines have the same inactivation rates and performance, in reality this might not be the case,
- In addition to microorganisms humans also excrete other elements such as heavy metals and pharmaceuticals and personal care products (PCPPs). There is technology being developed that is able to sequester heavy metals using micro – organisms and natural plant extracts. With regards to PCPPs these occur in minute quantities, there limited plant uptake and has not show signs of bioaccumulation or concentration (WHO, 2006),
- As presented, this model does not allow for increases in land yield due to technological breakthroughs; better seeds, pesticides or new ways of farming. In reality the carrying capacity of arable land has increased due to these factors in recent years,
- Also the model does not consider that as food shortages occur, planting density will likely increase which will lead to soil degradation and reduced fertility,
- The carrying capacity of the village was determined by the amount of arable land that was available for crop production only, in reality it is the sum of all the

limiting factors that control the population in the defined area should be consider in the computation. This is therefore a conservative estimate, and

- For chemotherapy it was assumed that the persons to be treated would be drawn randomly from the population. There is research that has suggested that treating those more heavily infected individuals may be more effective (Anderson, 1985).

7.3 Findings and conclusions

From the STELLA® models developed, various what – if scenarios and sensitive analysis were conducted. The major findings and conclusions from the simulations were as follows:

- The rate determining steps were: life expectancy of the adult worms, rate of egg production and the survival rate of eggs in the environment,
- The rate of reinfection to levels observed before chemotherapy was very rapid. Thus, chemotherapy must be accompanied with other strategies and needed to be continually applied for at least 2 years (Croll *et al.*, 1982),
- It will ideally take at least 2 – 5 years for disease to be sustainably controlled. However, this is contingent on the ability and willingness of the community to acquire and accept the new skills respectively. In general it takes about 1 – 2 generations for a major technical innovation to become a societal staple. This time can be significantly reduced and the probability of success increased if the intervention dove – tails an already established process, such as sun drying of excess agricultural product and sun drying of latrine contents (Spencer *et al.*, 1967).,

- For the nutrition intervention, it is important to note that feeding programs must be in place for the first year, independent of soybean cultivation, since it will take about a year before soybeans harvest and excreta production are synchronized,
- (Fewtrell *et al.*, 2005) found that point of use water availability was very effective in reducing disease incidence. The provision of water supply for washing hands could increase the success of the proposed program,
- The systems approach was shown to be more sustainable because of the cost effectiveness of utilizing an existing and abundant resources (sunlight), can be easily applied in tandem with current interventions, the community members are empowered by being able to contribute to the solution and by producing their own food, increases independence and socio – economic status, and is easily integrated into the community's social and cultural structure (Coreil *et al.*, 2001),
- (Muller *et al.*, 1989) suggested that latrines must be used by at least 20% of the hosts' population, which was confirmed by the simulations. However, to ensure eradication it was observed that 70% usage was required, and
- As the interventions succeed in eradicating the organisms, the population will increase over time, thus birth control methods must also be promoted (Barlow, 1967; Goodman *et al.*, 2006).

7.4 Future studies

The models developed in this work could be modified to produce age – appropriate effective didactic tools, which could be used to teach students about the link between feces and being sick and how to prevent disease occurrence. The transparent solar vault could be used as an important talking point to start the conversation about fecal matter,

its associated health risk and reuse benefits, a discussion that is currently taboo in many cultures.

One very important variable not considered in this study is maternal education and its impact on disease and health status of household members. Research has shown that as maternal education increases the fertility rate decreases and the health of children increases drastically (Moore, 2002; United Nations, 1991). In addition this variable is closely linked with socioeconomic status as indicated by the Threshold theory in Chapter 2 (Gorter *et al.*, 1998). It would of interest to determine the minimum level of economic empowerment necessary to encourage the community to address and sustainably solve their own health challenges.

Drying is an important part of excreta processing, however water and vapor diffusion were not considered here. These physics would help to represent the treatment process more realistically. Future studies would address this.

The village of Paquila, Guatemala is ideal for the interventions presented here; has a primary health care system in place, water is available to all and it is in close proximity to the extension services required to start a soybean program. Success in curtaining this highly visible disease could serve as an entry point into promoting and tackling other community challenges. A successful intervention program here could enable this village to serve as a model community for countless others with similar health issues and disease sustaining mechanisms. In addition an actual intervention would substantiate these findings and suggestions, which could then be tailored to the specific needs of a community.

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APPENDICES

Appendix A

Table A – 1: Complete breakdown of disease diagnosis at area clinics (Boca Costa Medical Mission, 2004)

Disease diagnosis	Patients seen/%
Emergency	0.10
Encephalopathy	0.10
Hemorrhagia	0.10
Asthma	0.20
Diabetic	0.20
Hepatitis	0.40
Surgical (recommended)	0.40
Seizure disorder	0.60
Hypertension	0.70
Bacterial vaginitis	0.90
Bacterial dysentery	1.40
Ear infection	1.70
Yeast infection	1.80
Allergies	1.90
Pregnant	2.00
Skin infection: fungal	2.80
Parasite skin (scabies / lice)	3.00
Dentist	3.50
Anemia	3.80
Urinary infection	3.90
Eye infection	4.40
Skin infection: bacterial	4.50
Gastritis	5.00
Amebic dysentery / Giardia	9.00
Other: general pain, vitamins, only	10.83
Respiratory infections	16.64
Intestinal (worms)	20.24

Appendix B

Table B – 1: Host – parasite STELLA® generated equations from Anderson and May (1978)

```
Hosts(t) = Hosts(t - dt) + (host_births - host_deaths - host_deaths_by_parasites) * dt
INIT Hosts = 100 {hosts}

INFLOWS:
host_births = Hosts * host_birth_rate {host/time}

OUTFLOWS:
host_deaths = Hosts * host_natural_death_rate {host/time}
host_deaths_by_parasites = Parasites * parasite_induced__host_death_rate
{parasite/time}

Parasites(t) = Parasites(t - dt) + (production - losses - predator_carrying__capacity) * dt
INIT Parasites = 210 {parasites}

INFLOWS:
production = (egg_production_rate * Parasites * Hosts) / (transmission_efficiency +
Hosts) {parasite/time}

OUTFLOWS:
losses = (host_natural_death_rate + parasite_natural_death_rate +
parasite_induced__host_death_rate) * Parasites {parasite/time}
predator_carrying__capacity = (parasite_induced__host_death_rate * Parasites  *
Parasites * (clumping_parameter + 1))/ (Hosts *clumping_parameter)
{parasites^2/host/time}

clumping__parameter = 0.57
clumping_parameter = 2.0
egg_production_rate = 6 {1/time}
host_birth_rate = 3.0 {1/time}
host_natural_death_rate = 1.0 {1/time}
mean_parasite_burden = Parasites/Hosts
mean_worm_burden = Parasites/Hosts {worm/host}
parasite_induced__host_death_rate = 0.5 {hosts/parasite/time}
parasite_natural_death_rate = 0.1 {1/time}
prevalence = 1- (1 + (mean_worm_burden/clumping__parameter))^-
clumping__parameter
transmission_efficiency = 10 {hosts}
```

Appendix B (Continued)

Table B – 2: Host – parasite STELLA® generated equations for Paquila

```
Hosts(t) = Hosts(t - dt) + (host_births - host_deaths - host_deaths_by_parasites) * dt
INIT Hosts = 3500 {hosts}

INFLOWS:
host_births = Hosts * host_birth_rate {host/time}

OUTFLOWS:
host_deaths = Hosts * host_natural__death_rate {host/time}
host_deaths_by_parasites = Parasites * parasite_induced__host_death_rate {host/time}

Parasites(t) = Parasites(t - dt) + (production - losses - predator_carrying__capacity) * dt
INIT Parasites = 7000 {parasites}

INFLOWS:
production = egg_production_transmission * Parasites * saturation {worm/time}

OUTFLOWS:
losses = (host_natural__death_rate + parasite_natural__death_rate + (worm_deaths *
Parasites)) * Parasites {worm/time}
predator_carrying__capacity = Parasites * parasite_induced__host_death_rate *
(Parasites * Parasites/(clumping__parameter *Hosts * Hosts)) {worm/time}

clumping__parameter = 0.57 {worm/host}
egg__hatching = 0.05 {worm/egg}
egg__survival = 0.01 {egg/egg}
egg_production__rate = 7300 {egg/worm/year}
egg_production_transmission = egg__hatching * egg__survival * egg_production__rate
{worm/egg * egg/egg * egg/worm/time}
host_birth_rate = 0.029 {1/year}
host_natural__death_rate = 0.00527 {1/year}
mean_worm_burden = Parasites/Hosts
parasite_induced__host_death_rate = 0.00005 {host/worm/year}
parasite_natural__death_rate = 1.15 {1/year}
saturation = Hosts/(Hosts + transmission__efficiency) {host/(host+host)}
transmission__efficiency = 100 {host}
worm_deaths = parasite_induced__host_death_rate/Hosts {1/worm/time}
```

Appendix B (Continued)

Table B – 3: STELLA® generated equations for population mean with chemotherapy

```
Population__Mean(t) = Population__Mean(t - dt) + (acquiring - losing - chemo) * dt
INIT Population__Mean = 20 {worm/host}

INFLOWS:
acquiring = (host_natural_death_rate  + parasite_natural_death_rate) * Ro1 *
Population__Mean {worm/host/time}

OUTFLOWS:
losing = parasite_induced_host_death_rate * Population__Mean * (((Population__Mean
* Population__Mean) /clumping__parameter) + Population__Mean) {worm/host/time}
chemo = IF(TIME < 5)  THEN(Population__Mean *
PULSE(chemo_rate,0,treatment__frequency)) ELSE(Population__Mean * 0)
{worm/host/time}

basic_reproductive_rate = 1.5
chemo_rate =  - LOGN(1 - drug_efficacy * proportion_treated)
clumping__parameter = 0.57 {worm/host}
drug_efficacy = 0.9 {worm/worm}
host_natural_death_rate = 0.00527 {1/time}
parasite_induced_host_death_rate = 0.00005 {host/worm/time}
parasite_natural_death_rate = 1.15 {1/time}
proportion_treated = 0.27 {host/host}
Ro1 = basic_reproductive_rate - 1
treatment__frequency = 0.33 {every 3 months}
```

Appendix B (Continued)

Table B – 4: STELLA® generated equations for excreta production

```
Excreta_Storage(t) = Excreta_Storage(t - dt) + (emptying_solar_vault_content) * dt
INIT Excreta_Storage = 0 {kg excreta}

INFLOWS:
emptying_solar_vault_content = PULSE(Solar_Vault_Excreta, 0.66,
solar_vault_retention__time)

Hosts(t) = Hosts(t - dt) + (host_births - host_deaths - host_deaths__by_parasites) * dt
INIT Hosts = 3500 {hosts}

INFLOWS:
host_births = Hosts * host_birth_rate {host/time}
OUTFLOWS:
host_deaths = Hosts * host_natural__death_rate {host/time}
host_deaths__by_parasites = Parasites * parasite_induced__host_death_rate
{host/time}
Latrine_Content(t) = Latrine_Content(t - dt) + (producing - emptying_latrine_content) * dt
INIT Latrine_Content = 0 {kg excreta}

INFLOWS:
producing = Hosts * excreta_production_rate {kg excreta/year}
OUTFLOWS:
emptying_latrine_content = PULSE(Latrine_Content, 5/12, latrine__retention_time)

Parasites(t) = Parasites(t - dt) + (production - losses - predator_carrying__capacity) * dt
INIT Parasites = 7000 {parasites}

INFLOWS:
production = egg_production_transmission * Parasites * saturation {worm/time}
OUTFLOWS:
losses = (host_natural__death_rate + parasite_natural__death_rate + (worm_deaths *
Parasites)) * Parasites {worm/time}
predator_carrying__capacity = Parasites * parasite_induced__host_death_rate *
(Parasites * Parasites/(clumping__parameter *Hosts * Hosts)) {worm/time}

Solar_Vault_Excreta(t) = Solar_Vault_Excreta(t - dt) + (emptying_latrine_content -
emptying_solar_vault_content) * dt
INIT Solar_Vault_Excreta = 0 {kg}

INFLOWS:
emptying_latrine_content = PULSE(Latrine_Content, 5/12, latrine__retention_time)

OUTFLOWS:
emptying_solar_vault_content = PULSE(Solar_Vault_Excreta, 0.66,
solar_vault_retention__time)
```

Appendix B (Continued)

Table B – 4 (continued)

clumping__parameter = 0.57 {worm/host} egg__hatching = 0.05 {worm/egg} egg__survival = 0.01 {egg/egg} egg_production__rate = 7300 {egg/worm/year} egg_production_transmission = egg__hatching * egg__survival * egg_production__rate {worm/egg * egg/egg * egg/worm/time} excreta_production_rate = 0.35 * 365 {kg excreta/person/day * 365 day/year = kg excreta/year} host_birth_rate = 0.029 {1/year} host_natural__death_rate = 0.00527 {1/year} latrine__retention_time = 0.33 { 0.33DT = 4months or 1/3year} parasite_induced__host_death_rate = 0.00005 {host/worm/year} parasite_natural__death_rate = 1.15 {1/year} saturation = Hosts/(Hosts + transmission__efficiency) {host/(host+host)} solar_vault_retention__time = 0.33 {100% removal} transmission__efficiency = 100 {host} worm_deaths = parasite_induced__host_death_rate/Hosts {1/worm/time}

Appendix B (Continued)

Table B – 5: STELLA® generated equations for the effect of nutrition on host's survival

```
Hosts(t) = Hosts(t - dt) + (host_births - host_deaths - host_deaths__by_parasites) * dt
INIT Hosts = 3500 {hosts}

INFLOWS:
host_births = Hosts * host_birth_rate {host/time}

OUTFLOWS:
host_deaths = Hosts *  host_natural__death_rate {host/time}
host_deaths__by_parasites = Parasites * parasite_induced__host_death_rate
{host/time}

Parasites(t) = Parasites(t - dt) + (production - losses - predator_carrying__capacity) * dt
INIT Parasites = 7000 {parasites}

INFLOWS:
production = egg_production_transmission * Parasites * saturation {worm/time}
OUTFLOWS:
losses = (host_natural__death_rate + parasite_natural__death_rate + (worm_deaths *
Parasites)) * Parasites {worm/time}
predator_carrying__capacity = Parasites * parasite_induced__host_death_rate *
(Parasites * Parasites/(clumping__parameter *Hosts * Hosts)) {worm/time}

Seedlings(t) = Seedlings(t - dt) + (replanting - maturing) * dt
INIT Seedlings = Hosts * replanting

INFLOWS:
replanting = IF(Hosts<carrying__capacity) THEN(PULSE((planting_rate*Hosts), 5/12,1))
ELSE (PULSE((planting_rate*carrying__capacity), 5/12,1))
OUTFLOWS:
maturing = Seedlings *maturing__fraction_rate * seed__production {plants/year}

Soybean_Seeds(t) = Soybean_Seeds(t - dt) + (maturing - consumption) * dt
INIT Soybean_Seeds = 184000000{soybean seeds}

INFLOWS:
maturing = Seedlings *maturing__fraction_rate * seed__production {plants/year}

OUTFLOWS:
consumption = actual_consuption_per_person_per_year
actual_available_soybean_per_person__per_year = min
(available_soybean_per_person__per_year, desired__soybean_seed_per_person)
{trees/person}
```

Appendix B (Continued)

Table B – 5 (continued)

actual_consuption_per_person_per_year =
normal_soybean_consumption_per_person_per_year *
effect_of_soybean_supplyon_consumption_per_year
arable_land = 1.37 {km^2}
available_soybean_per_person__per_year = (Soybean_Seeds/ Hosts) {soybean seeds/person/year}
carrying__capacity = arable_land/per_capita_land_requirement {host}
clumping__parameter = 0.57 {worm/host}
desired__soybean_seed_per_person = 528000 {soybean seeds/person/year}
egg__hatching = 0.02 {worm/egg}
egg__survival = 0.01 {egg/egg}
egg_production__rate = 7300 {egg/worm/year}
egg_production_transmission = egg__hatching * egg__survival * egg_production__rate {worm/egg * egg/egg * egg/worm/time}
host_birth_rate = 0.029 {1/year}
host_natural__death_rate = 0.00527 {1/year}
maturing__fraction = 0.45
maturing__fraction_rate = maturing__fraction/maturing_rate
maturing_rate = 4/12 {years}
mean_worm_burden = Parasites/Hosts
normal_parasite_induced_host_death_rate = 0.00005 {host/worm/year}
normal_soybean_consumption_per_person_per_year = 528000 {soybean seeds/person/year}
parasite_induced__host_death_rate = normal_parasite_induced_host_death_rate *effect_of_soybean__on_parasite_induced_host_death_rate {host/worm/year}
parasite_natural__death_rate = 1.15 {1/year}
per_capita_land_requirement = 3.661E-4 {km^2/host}
planting_rate = 0
pods_per_plant = 35 {pods/plant}
prevalence = 1-(1+(mean_worm_burden/clumping__parameter))^(-clumping__parameter)
saturation = Hosts/(Hosts + transmission__efficiency) {host/(host+host)}
seed__production = pods_per_plant * seeds_per_pod
seeds_per_pod = 3 {seeds/pod}
transmission__efficiency = 100 {host}
worm_deaths = parasite_induced__host_death_rate/Hosts {1/worm/time}
effect_of_soybean__on_parasite_induced_host_death_rate =
GRAPH(actual_available_soybean_per_person__per_year / desired__soybean_seed_per_person)
(0.00, 100), (0.2, 10.0), (0.4, 0.1), (0.6, 0.01), (0.8, 0.001), (1, 0.001), (1.20, 0.001), (1.40, 0.001)

Appendix B (Continued)

Table B – 5 (continued)

effect_of_soybean_supplyon_consumption_per_year = GRAPH(actual_available_soybean_per_person__per_year / desired__soybean_seed_per_person) (0.00, 0.00), (0.2, 0.2), (0.4, 0.3), (0.6, 0.4), (0.8, 0.5), (1.00, 1.00), (1.20, 2.00), (1.40, 3.00), (1.60, 5.00), (1.80, 10.0), (2.00, 20.0)

Appendix B (Continued)

Table B – 6: STELLA® generated equations for the effect of nutrition and chemo on host's survival

```
Hosts(t) = Hosts(t - dt) + (host_births - host_deaths - host_deaths__by_parasites) * dt
INIT Hosts = 3500 {hosts}

INFLOWS:
host_births = Hosts * host_birth_rate {host/time}

OUTFLOWS:
host_deaths = Hosts *  host_natural__death_rate {host/time}
host_deaths__by_parasites = Parasites * parasite_induced__host_death_rate
{host/time}

Parasites(t) = Parasites(t - dt) + (production - losses - predator_carrying__capacity -
chemo) * dt
INIT Parasites = 7000 {parasites}

INFLOWS:
production = egg_production_transmission * Parasites * saturation {worm/time}

OUTFLOWS:
losses = (host_natural__death_rate + parasite_natural__death_rate + (worm_deaths *
Parasites)) * Parasites {worm/time}
predator_carrying__capacity = Parasites * parasite_induced__host_death_rate *
(Parasites * Parasites/(clumping__parameter *Hosts * Hosts)) {worm/time}
chemo = IF(TIME < 2)  THEN(Parasites * PULSE(chemo_rate,0,treatment_frequency))
ELSE(Parasites * 0) {worm/time}

Seedlings(t) = Seedlings(t - dt) + (replanting - maturing) * dt
INIT Seedlings = Hosts * replanting

INFLOWS:
replanting = IF(Hosts<carrying__capacity) THEN(PULSE((planting_rate*Hosts), 5/12,1))
ELSE (PULSE((planting_rate*carrying__capacity), 5/12,1))

OUTFLOWS:
maturing = Seedlings *maturing__fraction_rate * seed__production {plants/year}

Soybean_Seeds(t) = Soybean_Seeds(t - dt) + (maturing - consumption) * dt
INIT Soybean_Seeds = 184000000{soybean seeds}

INFLOWS:
maturing = Seedlings *maturing__fraction_rate * seed__production {plants/year}
```

Appendix B (Continued)

Table B – 6 (continued)

OUTFLOWS:
consumption = actual_consuption_per_person_per_year
actual_available_soybean_per_person__per_year = min
(available_soybean_per_person__per_year, desired__soybean_seed_per_person)
{trees/person}
actual_consuption_per_person_per_year =
normal_soybean_consumption_per_person_per_year *
effect_of_soybean_supplyon_consumption_per_year
arable_land = 1.37 {km^2}
available_soybean_per_person__per_year = (Soybean_Seeds/ Hosts) {soybean
seeds/person/year}
carrying__capacity = arable_land/per_capita_land_requirement {host}
chemo_rate = - LOGN(1 - drug__efficacy * proportion_treated)
clumping__parameter = 0.57 {worm/host}
desired__soybean_seed_per_person = 528000 {soybean seeds/person/year}
drug__efficacy = 0.94 {worm/worm}
egg__hatching = 0.02 {worm/egg}
egg__survival = 0.01 {egg/egg}
egg_production__rate = 7300 {egg/worm/year}
egg_production_transmission = egg__hatching * egg__survival * egg_production__rate
{worm/egg * egg/egg * egg/worm/time}
host_birth_rate = 0.029 {1/year}
host_natural__death_rate = 0.00527 {1/year}
maturing__fraction = 0.45
maturing__fraction_rate = maturing__fraction/maturing_rate
maturing_rate = 4/12 {years}
mean_worm_burden = Parasites/Hosts
normal_parasite_induced_host_death_rate = 0.00005 {host/worm/year}
normal_soybean_consumption_per_person_per_year = 528000 {soybean
seeds/person/year}
parasite_induced__host_death_rate = normal_parasite_induced_host_death_rate
*effect_of_soybean__on_parasite_induced_host_death_rate {host/worm/year}
parasite_natural__death_rate = 1.15 {1/year}
per_capita_land_requirement = 3.661E-4 {km^2/host}
planting_rate = 0
pods_per_plant = 35 {pods/plant}
prevalence = 1-(1+(mean_worm_burden/clumping__parameter))^(-
clumping__parameter)
proportion_treated = 0.5 {host/host}
saturation = Hosts/(Hosts + transmission__efficiency) {host/(host+host)}
seed__production = pods_per_plant * seeds_per_pod
seeds_per_pod = 3 {seeds/pod}
transmission__efficiency = 100 {host}
treatment_frequency = 0.25 {every 3 months}

Appendix B (Continued)

Table B – 6 (continued)

worm_deaths = parasite_induced__host_death_rate/Hosts {1/worm/time}

effect_of_soybean__on_parasite_induced_host_death_rate =
GRAPH(actual_available_soybean_per_person__per_year /
desired__soybean_seed_per_person)
(0.00, 100), (0.2, 10.0), (0.4, 0.1), (0.6, 0.001), (0.8, 0.001), (1, 0.001), (1.20, 0.001), (1.40, 0.001)

effect_of_soybean_supplyon_consumption_per_year =
GRAPH(actual_available_soybean_per_person__per_year /
desired__soybean_seed_per_person)
(0.00, 0.00), (0.2, 0.2), (0.4, 0.3), (0.6, 0.4), (0.8, 0.5), (1.00, 1.00), (1.20, 2.00), (1.40, 3.00), (1.60, 5.00), (1.80, 10.0), (2.00, 20.0)

Appendix B (Continued)

Table B – 7: STELLA® generated equations for all three populations separated

```
Hosts(t) = Hosts(t - dt) + (host_births - host_deaths - host_deaths_by_parasites) * dt
INIT Hosts = 3500 {hosts}

INFLOWS:
host_births = Hosts * host_birth_rate {host/time}

OUTFLOWS:
host_deaths = Hosts * host_natural__death_rate {host/time}
host_deaths_by_parasites = Parasites * parasite_induced__host_death_rate {host/time}
Infective_Egg__Population(t) = Infective_Egg__Population(t - dt) + (egg_production -
loss_to_host - inactivation_in__environment) * dt
INIT Infective_Egg__Population = 0

INFLOWS:
egg_production = egg__survival*egg_production__rate*Parasites {egg/year}
OUTFLOWS:
loss_to_host = contact_rate*Infective_Egg__Population*Hosts
inactivation_in__environment = egg__survival/environmental_retention__time
Parasites(t) = Parasites(t - dt) + (production - losses - predator_carrying__capacity) * dt
INIT Parasites = 7000 {parasites}

INFLOWS:
production = egg_production_transmission*Infective_Egg__Population*Hosts
{worm/time}

OUTFLOWS:
losses = (host_natural__death_rate + parasite_natural__death_rate + (worm_deaths *
Parasites)) * Parasites {worm/time}
predator_carrying__capacity = Parasites * parasite_induced__host_death_rate *
(Parasites * Parasites/(clumping__parameter *Hosts * Hosts)) {worm/time}
clumping__parameter = 0.57 {worm/host}
contact_rate = inactivation_in__environment/transmission__efficiency
egg__hatching = 0.05 {worm/egg}
egg__survival = 0.01 {egg/egg}
egg_production__rate = 7300 {egg/worm/year}
egg_production_transmission = egg__hatching* contact_rate {worm/egg * egg/egg *
egg/worm/time}
environmental_retention__time = 0.125
host_birth_rate = 0.029 {1/year}
host_natural__death_rate = 0.00527 {1/year}
mean_worm_burden = Parasites/Hosts
parasite_induced__host_death_rate = 0.00005 {host/worm/year}
parasite_natural__death_rate = 1.15 {1/year}
transmission__efficiency = 100 {host}
worm_deaths = parasite_induced__host_death_rate/Hosts {1/worm/time}
```

Appendix B (Continued)

Table B – 8: STELLA® generated equations for host's population response to latrine intervention

```
Hosts(t) = Hosts(t - dt) + (host_births - host_deaths - host_deaths_by_parasites) * dt
INIT Hosts = 3500 {hosts}

INFLOWS:
host_births = Hosts * host_birth_rate {host/time}

OUTFLOWS:
host_deaths = Hosts * host_natural__death_rate {host/time}
host_deaths_by_parasites = Parasites * parasite_induced__host_death_rate {host/time}

Infective_Egg__Population(t) = Infective_Egg__Population(t - dt) + (egg_production -
loss_to_host - inactivation_in__environment - solar_rate) * dt
INIT Infective_Egg__Population = 260610

INFLOWS:
egg_production = egg__survival*egg_production__rate*Parasites {egg/year}

OUTFLOWS:
loss_to_host = contact_rate*Infective_Egg__Population*Hosts
inactivation_in__environment = egg__survival/environmental_retention__time
solar_rate = IF(TIME<15)
THEN(Infective_Egg__Population*PULSE(latrine_rate,0,latrine__retention_time))
ELSE(Infective_Egg__Population*0)

Parasites(t) = Parasites(t - dt) + (production - losses - predator_carrying__capacity) * dt
INIT Parasites = 7000 {parasites}

INFLOWS:
production = egg_production_transmission*Infective_Egg__Population*Hosts
{worm/time}
OUTFLOWS:
losses = (host_natural__death_rate + parasite_natural__death_rate + (worm_deaths *
Parasites)) * Parasites {worm/time}
predator_carrying__capacity = Parasites * parasite_induced__host_death_rate *
(Parasites * Parasites/(clumping__parameter *Hosts * Hosts)) {worm/time}

clumping__parameter = 0.57 {worm/host}
contact_rate = inactivation_in__environment/transmission__efficiency
egg__hatching = 0.05 {worm/egg}
egg__survival = 0.01 {egg/egg}
egg_production__rate = 7300 {egg/worm/year}
egg_production_transmission = egg__hatching* contact_rate {worm/egg * egg/egg *
egg/worm/time}
environmental_retention__time = 0.125
host_birth_rate = 0.029 {1/year}
```

Appendix B (Continued)

Table B – 8 (continued)

host_natural__death_rate = 0.00527 {1/year}
latrine__retention_time = 0.25
latrine_efficacy = 0.99
latrine_rate = -LOGN(1-latrine_efficacy*proprotion_of__host_using_latrine)
mean_worm_burden = Parasites/Hosts
parasite_induced__host_death_rate = 0.00005 {host/worm/year}
parasite_natural__death_rate = 1.15 {1/year}
proprotion_of__host_using_latrine = 0.4 {host/host}
transmission__efficiency = 100 {host}
worm_deaths = parasite_induced__host_death_rate/Hosts {1/worm/time}

Appendix B (Continued)

Table B – 9: STELLA® generated equations for host's population response to latrine and chemo interventions

```
Hosts(t) = Hosts(t - dt) + (host_births - host_deaths - host_deaths_by_parasites) * dt
INIT Hosts = 3500 {hosts}

INFLOWS:
host_births = Hosts * host_birth_rate {host/time}

OUTFLOWS:
host_deaths = Hosts * host_natural__death_rate {host/time}
host_deaths_by_parasites = Parasites * parasite_induced__host_death_rate {host/time}

Infective_Egg__Population(t) = Infective_Egg__Population(t - dt) + (egg_production -
loss_to_host - inactivation_in__environment - solar_latrine) * dt
INIT Infective_Egg__Population = 260610

INFLOWS:
egg_production = egg__survival*egg_production__rate*Parasites {egg/year}

OUTFLOWS:
loss_to_host = contact_rate*Infective_Egg__Population*Hosts
inactivation_in__environment = egg__survival/environmental_retention__time
solar_latrine = IF(TIME<15)
THEN(Infective_Egg__Population*PULSE(latrine_rate,0,latrine__retention_time))
ELSE(Infective_Egg__Population*0)

Parasites(t) = Parasites(t - dt) + (production - losses - predator_carrying__capacity -
chemo) * dt
INIT Parasites = 7000 {parasites}

INFLOWS:
production = egg_production_transmission*Infective_Egg__Population*Hosts
{worm/time}

OUTFLOWS:
losses = (host_natural__death_rate + parasite_natural__death_rate + (worm_deaths *
Parasites)) * Parasites {worm/time}
predator_carrying__capacity = Parasites * parasite_induced__host_death_rate *
(Parasites * Parasites/(clumping__parameter *Hosts * Hosts)) {worm/time}
chemo = IF(TIME < 2)  THEN(Parasites * PULSE(chemo_rate,0,treatment_frequency))
ELSE(Parasites * 0) {worm/time}
chemo_rate =  - LOGN(1 - drug__efficacy * proportion_treated)
clumping__parameter = 0.57 {worm/host}
contact_rate = inactivation_in__environment/transmission__efficiency
drug__efficacy = 0.94 {worm/worm}
egg__hatching = 0.05 {worm/egg}
```

Appendix B (Continued)

Table B – 9 (continued)

egg__survival = 0.01 {egg/egg}
egg_production__rate = 7300 {egg/worm/year}
egg_production_transmission = egg__hatching* contact_rate {worm/egg * egg/egg * egg/worm/time}
environmental_retention__time = 0.125
host_birth_rate = 0.029 {1/year}
host_natural__death_rate = 0.00527 {1/year}
latrine__retention_time = 0.25
latrine_efficacy = 0.99
latrine_rate = -LOGN(1-latrine_efficacy*proprotion_of__host_using_latrine)
mean_worm_burden = Parasites/Hosts
parasite_induced__host_death_rate = 0.00005 {host/worm/year}
parasite_natural__death_rate = 1.15 {1/year}
proportion_treated = 0.50 {host/host}
proprotion_of__host_using_latrine = 0.4
transmission__efficiency = 100 {host}
treatment_frequency = 0.25 {every 3 months}
worm_deaths = parasite_induced__host_death_rate/Hosts {1/worm/time}

Appendix B (Continued)

Table B – 10: STELLA® generated equations for host's population response to latrine, chemo and nutrition interventions

```
Hosts(t) = Hosts(t - dt) + (host_births - host_deaths - host_deaths_by_parasites) * dt
INIT Hosts = 3500 {hosts}

INFLOWS:
host_births = Hosts * host_birth_rate {host/time}

OUTFLOWS:
host_deaths = Hosts * host_natural__death_rate {host/time}
host_deaths_by_parasites = Parasites * parasite_induced__host_death_rate {host/time}

Infective_Egg__Population(t) = Infective_Egg__Population(t - dt) + (egg_production -
loss_to_host - inactivation_in__environment - solar_latrine) * dt
INIT Infective_Egg__Population = 260610

INFLOWS:
egg_production = egg__survival*egg_production__rate*Parasites {egg/year}

OUTFLOWS:
loss_to_host = contact_rate*Infective_Egg__Population*Hosts
inactivation_in__environment = egg__survival/environmental_retention__time
solar_latrine = IF(TIME<15)
THEN(Infective_Egg__Population*PULSE(latrine_rate,0,latrine__retention_time))
ELSE(Infective_Egg__Population*0)

Parasites(t) = Parasites(t - dt) + (production - losses - predator_carrying__capacity -
chemo) * dt
INIT Parasites = 7000 {parasites}

INFLOWS:
production = egg_production_transmission*Infective_Egg__Population*Hosts
{worm/time}

OUTFLOWS:
losses = (host_natural__death_rate + parasite_natural__death_rate + (worm_deaths *
Parasites)) * Parasites {worm/time}
predator_carrying__capacity = Parasites * parasite_induced__host_death_rate *
(Parasites * Parasites/(clumping__parameter *Hosts * Hosts)) {worm/time}
chemo = IF(TIME < 2)  THEN(Parasites * PULSE(chemo_rate,0,treatment_frequency))
ELSE(Parasites * 0) {worm/time}

Seedlings(t) = Seedlings(t - dt) + (replanting - maturing) * dt
INIT Seedlings = Hosts * replanting
```

Appendix B (Continued)

Table B – 10 (continued)

INFLOWS:
replanting = IF(Hosts<carrying__capacity) THEN(PULSE((planting_rate*Hosts), 5/12,1))
ELSE (PULSE((planting_rate*carrying__capacity), 5/12,1))

OUTFLOWS:
maturing = Seedlings *maturing__fraction_rate * seed__production {plants/year}

Soybean_Seeds(t) = Soybean_Seeds(t - dt) + (maturing - consumption) * dt
INIT Soybean_Seeds = 184000000{soybean seeds}

INFLOWS:
maturing = Seedlings *maturing__fraction_rate * seed__production {plants/year}

OUTFLOWS:
consumption = actual_consuption_per_person_per_year
actual_available_soybean_per_person__per_year = min
(available_soybean_per_person__per_year, desired__soybean_seed_per_person)
{trees/person}
actual_consuption_per_person_per_year =
normal_soybean_consumption_per_person_per_year *
effect_of_soybean_supplyon_consumption_per_year
arable_land = 1.37 {km^2}
available_soybean_per_person__per_year = (Soybean_Seeds/ Hosts) {soybean
seeds/person/year}
carrying__capacity = arable_land/per_capita_land_requirement {host}
chemo_rate = - LOGN(1 - drug__efficacy * proportion_treated)
clumping__parameter = 0.57 {worm/host}
contact_rate = inactivation_in__environment/transmission__efficiency
desired__soybean_seed_per_person = 528000 {soybean seeds/person/year}
drug__efficacy = 0.94 {worm/worm}
egg__hatching = 0.05 {worm/egg}
egg__survival = 0.01 {egg/egg}
egg_production__rate = 7300 {egg/worm/year}
egg_production_transmission = egg__hatching* contact_rate {worm/egg * egg/egg *
egg/worm/time}
environmental_retention__time = 0.125
host_birth_rate = 0.029 {1/year}
host_natural__death_rate = 0.00527 {1/year}
latrine__retention_time = 0.25
latrine_efficacy = 0.99
latrine_rate = -LOGN(1-latrine_efficacy*proprotion_of__host_using_latrine)
maturing__fraction = 0.45
maturing__fraction_rate = maturing__fraction/maturing_rate
maturing_rate = 4/12 {years}

Appendix B (Continued)

Table B – 10 (continued)

```
mean_worm_burden = Parasites/Hosts
normal_parasite_induced_host_death_rate = 0.00005 {host/worm/year}
normal_soybean_consumption_per_person_per_year = 528000 {soybean
seeds/person/year}
parasite_induced__host_death_rate = normal_parasite_induced_host_death_rate *
effect_of_soybean__on_parasite_induced_host_death_rate {host/worm/year}
parasite_natural__death_rate = 1.15 {1/year}
per_capita_land_requirement = 3.661E-4 {km^2/host}
planting_rate = 11175
pods_per_plant = 35 {pods/plant}
proportion_treated = 0.5 {host/host}
proprotion_of__host_using_latrine = 0.4
seed__production = pods_per_plant * seeds_per_pod
seeds_per_pod = 3 {seeds/pod}
transmission__efficiency = 100 {host}
treatment_frequency = 0.25 {every 3 months}
worm_deaths = parasite_induced__host_death_rate/Hosts {1/worm/time}

effect_of_soybean__on_parasite_induced_host_death_rate =
GRAPH(actual_available_soybean_per_person__per_year /
desired__soybean_seed_per_person)
(0.00, 100), (0.2, 10.0), (0.4, 0.1), (0.6, 0.001), (0.8, 0.001), (1, 0.001), (1.20, 0.001),
(1.40, 0.001)

effect_of_soybean_supplyon_consumption_per_year =
GRAPH(actual_available_soybean_per_person__per_year /
desired__soybean_seed_per_person)
(0.00, 0.00), (0.2, 0.2), (0.4, 0.3), (0.6, 0.4), (0.8, 0.5), (1.00, 1.00), (1.20, 2.00), (1.40,
3.00), (1.60, 5.00), (1.80, 10.0), (2.00, 20.0)
```

Appendix C

Table C – 1: Solar incidence radiation on the south – facing Solar Latrine panel in Paquila, Guatemala for the months May to August

Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c
1	0	41	0	81	432	121	0
2	0	42	0	82	589	122	0
3	0	43	0	83	691	123	0
4	0	44	0	84	726	124	0
5	0	45	0	85	691	125	0
6	1	46	0	86	589	126	1
7	72	47	0	87	432	127	73
8	245	48	0	88	245	128	246
9	431	49	0	89	73	129	434
10	587	50	0	90	1	130	590
11	689	51	0	91	0	131	693
12	724	52	0	92	0	132	728
13	689	53	0	93	0	133	693
14	587	54	1	94	0	134	590
15	431	55	73	95	0	135	434
16	245	56	245	96	0	136	246
17	72	57	432	97	0	137	73
18	1	58	588	98	0	138	1
19	0	59	690	99	0	139	0
20	0	60	725	100	0	140	0
21	0	61	690	101	0	141	0
22	0	62	588	102	1	142	0
23	0	63	432	103	73	143	0
24	0	64	245	104	246	144	0
25	0	65	73	105	433	145	0
26	0	66	1	106	589	146	0
27	1	67	0	107	692	147	0
28	73	68	0	108	727	148	1
29	245	69	0	109	692	149	73
30	431	70	0	110	589	150	246
31	587	71	0	111	433	151	434
32	689	72	0	112	246	152	591
33	725	73	0	113	73	153	694
34	689	74	0	114	1	154	729
35	587	75	0	115	0	155	694
36	431	76	0	116	0	156	591
37	245	77	0	117	0	157	434
38	73	78	1	118	0	158	246
39	1	79	73	119	0	159	73
40	0	80	245	120	0	160	1

Appendix C (Continued)

Table C – 1 (continued)

Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c
161	0	201	436	241	0	281	73
162	0	202	593	242	0	282	1
163	0	203	696	243	0	283	0
164	0	204	731	244	0	284	0
165	0	205	696	245	0	285	0
166	0	206	593	246	1	286	0
167	0	207	436	247	73	287	0
168	0	208	247	248	248	288	0
169	0	209	73	249	437	289	0
170	0	210	1	250	595	290	0
171	0	211	0	251	698	291	0
172	0	212	0	252	734	292	0
173	0	213	0	253	698	293	0
174	1	214	0	254	595	294	1
175	73	215	0	255	437	295	73
176	247	216	0	256	248	296	249
177	435	217	0	257	73	297	439
178	592	218	0	258	1	298	597
179	695	219	0	259	0	299	701
180	730	220	0	260	0	300	736
181	695	221	0	261	0	301	701
182	592	222	1	262	0	302	597
183	435	223	73	263	0	303	439
184	247	224	248	264	0	304	249
185	73	225	436	265	0	305	73
186	1	226	594	266	0	306	1
187	0	227	697	267	0	307	0
188	0	228	732	268	0	308	0
189	0	229	697	269	0	309	0
190	0	230	594	270	1	310	0
191	0	231	436	271	73	311	0
192	0	232	248	272	249	312	0
193	0	233	73	273	438	313	0
194	0	234	1	274	596	314	0
195	0	235	0	275	699	315	0
196	0	236	0	276	735	316	0
197	0	237	0	277	699	317	0
198	1	238	0	278	596	318	1
199	73	239	0	279	438	319	74
200	247	240	0	280	249	320	250

Appendix C (Continued)

Table C – 1 (continued)

Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c
321	440	361	0	401	74	441	445
322	598	362	0	402	1	442	605
323	702	363	0	403	0	443	710
324	738	364	0	404	0	444	746
325	702	365	1	405	0	445	710
326	598	366	74	406	0	446	605
327	440	367	251	407	0	447	445
328	250	368	442	408	0	448	253
329	74	369	601	409	0	449	74
330	1	370	705	410	0	450	1
331	0	371	741	411	0	451	0
332	0	372	705	412	0	452	0
333	0	373	601	413	0	453	0
334	0	374	442	414	1	454	0
335	0	375	251	415	74	455	0
336	0	376	74	416	252	456	0
337	0	377	1	417	444	457	0
338	0	378	0	418	604	458	0
339	0	379	0	419	708	459	0
340	0	380	0	420	744	460	0
341	0	381	0	421	708	461	0
342	1	382	0	422	604	462	1
343	74	383	0	423	444	463	74
344	250	384	0	424	252	464	253
345	441	385	0	425	74	465	446
346	600	386	0	426	1	466	607
347	703	387	0	427	0	467	712
348	739	388	0	428	0	468	748
349	703	389	0	429	0	469	712
350	600	390	1	430	0	470	607
351	441	391	74	431	0	471	446
352	250	392	251	432	0	472	253
353	74	393	443	433	0	473	74
354	1	394	602	434	0	474	1
355	0	395	707	435	0	475	0
356	0	396	743	436	0	476	0
357	0	397	707	437	0	477	0
358	0	398	602	438	1	478	0
359	0	399	443	439	74	479	0
360	0	400	251	440	253	480	0

Appendix C (Continued)

Table C – 1 (continued)

Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c
481	0	521	75	561	451	601	0
482	0	522	0	562	613	602	0
483	0	523	0	563	719	603	0
484	0	524	0	564	755	604	0
485	0	525	0	565	719	605	0
486	1	526	0	566	613	606	0
487	74	527	0	567	451	607	75
488	254	528	0	568	256	608	258
489	447	529	0	569	75	609	453
490	608	530	0	570	0	610	617
491	713	531	0	571	0	611	723
492	750	532	0	572	0	612	759
493	713	533	0	573	0	613	723
494	608	534	0	574	0	614	617
495	447	535	75	575	0	615	453
496	254	536	255	576	0	616	258
497	74	537	450	577	0	617	75
498	1	538	612	578	0	618	0
499	0	539	717	579	0	619	0
500	0	540	753	580	0	620	0
501	0	541	717	581	0	621	0
502	0	542	612	582	0	622	0
503	0	543	450	583	75	623	0
504	0	544	255	584	257	624	0
505	0	545	75	585	452	625	0
506	0	546	0	586	615	626	0
507	0	547	0	587	721	627	0
508	0	548	0	588	757	628	0
509	0	549	0	589	721	629	0
510	0	550	0	590	615	630	0
511	75	551	0	591	452	631	75
512	255	552	0	592	257	632	258
513	448	553	0	593	75	633	455
514	610	554	0	594	0	634	618
515	715	555	0	595	0	635	725
516	751	556	0	596	0	636	761
517	715	557	0	597	0	637	725
518	610	558	0	598	0	638	618
519	448	559	75	599	0	639	455
520	255	560	256	600	0	640	258

Appendix C (Continued)

Table C – 1 (continued)

Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c
641	75	681	458	721	0	761	75
642	0	682	622	722	0	762	0
643	0	683	729	723	0	763	0
644	0	684	766	724	0	764	0
645	0	685	729	725	0	765	0
646	0	686	622	726	0	766	0
647	0	687	458	727	76	767	0
648	0	688	260	728	261	768	0
649	0	689	76	729	460	769	0
650	0	690	0	730	626	770	0
651	0	691	0	731	733	771	0
652	0	692	0	732	770	772	0
653	0	693	0	733	733	773	0
654	0	694	0	734	626	774	0
655	75	695	0	735	460	775	75
656	259	696	0	736	261	776	261
657	456	697	0	737	76	777	461
658	620	698	0	738	0	778	627
659	727	699	0	739	0	779	735
660	764	700	0	740	0	780	772
661	727	701	0	741	0	781	735
662	620	702	0	742	0	782	627
663	456	703	76	743	0	783	461
664	259	704	261	744	0	784	261
665	75	705	459	745	0	785	75
666	0	706	624	746	0	786	0
667	0	707	731	747	0	787	0
668	0	708	768	748	0	788	0
669	0	709	731	749	0	789	0
670	0	710	624	750	0	790	0
671	0	711	459	751	75	791	0
672	0	712	261	752	260	792	0
673	0	713	76	753	459	793	0
674	0	714	0	754	625	794	0
675	0	715	0	755	733	795	0
676	0	716	0	756	770	796	0
677	0	717	0	757	733	797	0
678	0	718	0	758	625	798	0
679	76	719	0	759	459	799	75
680	260	720	0	760	260	800	262

Appendix C (Continued)

Table C – 1 (continued)

Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c
801	462	841	0	881	75	921	470
802	629	842	0	882	0	922	639
803	737	843	0	883	0	923	749
804	774	844	0	884	0	924	786
805	737	845	0	885	0	925	749
806	629	846	0	886	0	926	639
807	462	847	75	887	0	927	470
808	262	848	264	888	0	928	266
809	75	849	465	889	0	929	76
810	0	850	633	890	0	930	0
811	0	851	742	891	0	931	0
812	0	852	779	892	0	932	0
813	0	853	742	893	0	933	0
814	0	854	633	894	0	934	0
815	0	855	465	895	76	935	0
816	0	856	264	896	265	936	0
817	0	857	75	897	468	937	0
818	0	858	0	898	637	938	0
819	0	859	0	899	746	939	0
820	0	860	0	900	784	940	0
821	0	861	0	901	746	941	0
822	0	862	0	902	637	942	0
823	75	863	0	903	468	943	76
824	263	864	0	904	265	944	267
825	464	865	0	905	76	945	471
826	631	866	0	906	0	946	641
827	739	867	0	907	0	947	751
828	777	868	0	908	0	948	789
829	739	869	0	909	0	949	751
830	631	870	0	910	0	950	641
831	464	871	75	911	0	951	471
832	263	872	264	912	0	952	267
833	75	873	467	913	0	953	76
834	0	874	635	914	0	954	0
835	0	875	744	915	0	955	0
836	0	876	782	916	0	956	0
837	0	877	744	917	0	957	0
838	0	878	635	918	0	958	0
839	0	879	467	919	76	959	0
840	0	880	264	920	266	960	0

Appendix C (Continued)

Table C – 1 (continued)

Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c
961	0	1001	76	1041	478	1081	0
962	0	1002	0	1042	649	1082	0
963	0	1003	0	1043	760	1083	0
964	0	1004	0	1044	799	1084	0
965	0	1005	0	1045	760	1085	0
966	0	1006	0	1046	649	1086	0
967	76	1007	0	1047	478	1087	76
968	268	1008	0	1048	270	1088	272
969	473	1009	0	1049	76	1089	481
970	643	1010	0	1050	0	1090	653
971	753	1011	0	1051	0	1091	765
972	791	1012	0	1052	0	1092	804
973	753	1013	0	1053	0	1093	765
974	643	1014	0	1054	0	1094	653
975	473	1015	76	1055	0	1095	481
976	268	1016	269	1056	0	1096	272
977	76	1017	476	1057	0	1097	76
978	0	1018	647	1058	0	1098	0
979	0	1019	758	1059	0	1099	0
980	0	1020	796	1060	0	1100	0
981	0	1021	758	1061	0	1101	0
982	0	1022	647	1062	0	1102	0
983	0	1023	476	1063	76	1103	0
984	0	1024	269	1064	271	1104	0
985	0	1025	76	1065	479	1105	0
986	0	1026	0	1066	651	1106	0
987	0	1027	0	1067	763	1107	0
988	0	1028	0	1068	801	1108	0
989	0	1029	0	1069	763	1109	0
990	0	1030	0	1070	651	1110	0
991	76	1031	0	1071	479	1111	76
992	269	1032	0	1072	271	1112	273
993	474	1033	0	1073	76	1113	482
994	645	1034	0	1074	0	1114	655
995	756	1035	0	1075	0	1115	768
996	794	1036	0	1076	0	1116	806
997	756	1037	0	1077	0	1117	768
998	645	1038	0	1078	0	1118	655
999	474	1039	76	1079	0	1119	482
1000	269	1040	270	1080	0	1120	273

Appendix C (Continued)

Table C – 1 (continued)

Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c
1121	76	1161	485	1201	0	1241	76
1122	0	1162	659	1202	0	1242	0
1123	0	1163	772	1203	0	1243	0
1124	0	1164	811	1204	0	1244	0
1125	0	1165	772	1205	0	1245	0
1126	0	1166	659	1206	0	1246	0
1127	0	1167	485	1207	76	1247	0
1128	0	1168	274	1208	276	1248	0
1129	0	1169	76	1209	488	1249	0
1130	0	1170	0	1210	664	1250	0
1131	0	1171	0	1211	777	1251	0
1132	0	1172	0	1212	816	1252	0
1133	0	1173	0	1213	777	1253	0
1134	0	1174	0	1214	664	1254	0
1135	76	1175	0	1215	488	1255	76
1136	274	1176	0	1216	276	1256	278
1137	484	1177	0	1217	76	1257	491
1138	657	1178	0	1218	0	1258	668
1139	770	1179	0	1219	0	1259	782
1140	809	1180	0	1220	0	1260	821
1141	770	1181	0	1221	0	1261	782
1142	657	1182	0	1222	0	1262	668
1143	484	1183	76	1223	0	1263	491
1144	274	1184	275	1224	0	1264	278
1145	76	1185	487	1225	0	1265	76
1146	0	1186	661	1226	0	1266	0
1147	0	1187	775	1227	0	1267	0
1148	0	1188	814	1228	0	1268	0
1149	0	1189	775	1229	0	1269	0
1150	0	1190	661	1230	0	1270	0
1151	0	1191	487	1231	76	1271	0
1152	0	1192	275	1232	277	1272	0
1153	0	1193	76	1233	490	1273	0
1154	0	1194	0	1234	666	1274	0
1155	0	1195	0	1235	779	1275	0
1156	0	1196	0	1236	819	1276	0
1157	0	1197	0	1237	779	1277	0
1158	0	1198	0	1238	666	1278	0
1159	76	1199	0	1239	490	1279	76
1160	274	1200	0	1240	277	1280	278

Appendix C (Continued)

Table C – 1 (continued)

Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c
1281	493	1321	0	1361	76	1401	500
1282	670	1322	0	1362	0	1402	679
1283	784	1323	0	1363	0	1403	795
1284	824	1324	0	1364	0	1404	835
1285	784	1325	0	1365	0	1405	795
1286	670	1326	0	1366	0	1406	679
1287	493	1327	76	1367	0	1407	500
1288	278	1328	280	1368	0	1408	282
1289	76	1329	496	1369	0	1409	76
1290	0	1330	674	1370	0	1410	0
1291	0	1331	789	1371	0	1411	0
1292	0	1332	828	1372	0	1412	0
1293	0	1333	789	1373	0	1413	0
1294	0	1334	674	1374	0	1414	0
1295	0	1335	496	1375	76	1415	0
1296	0	1336	280	1376	281	1416	0
1297	0	1337	76	1377	498	1417	0
1298	0	1338	0	1378	677	1418	0
1299	0	1339	0	1379	793	1419	0
1300	0	1340	0	1380	833	1420	0
1301	0	1341	0	1381	793	1421	0
1302	0	1342	0	1382	677	1422	0
1303	76	1343	0	1383	498	1423	76
1304	279	1344	0	1384	281	1424	282
1305	494	1345	0	1385	76	1425	501
1306	672	1346	0	1386	0	1426	681
1307	786	1347	0	1387	0	1427	798
1308	826	1348	0	1388	0	1428	838
1309	786	1349	0	1389	0	1429	798
1310	672	1350	0	1390	0	1430	681
1311	494	1351	76	1391	0	1431	501
1312	279	1352	280	1392	0	1432	282
1313	76	1353	497	1393	0	1433	76
1314	0	1354	675	1394	0	1434	0
1315	0	1355	791	1395	0	1435	0
1316	0	1356	831	1396	0	1436	0
1317	0	1357	791	1397	0	1437	0
1318	0	1358	675	1398	0	1438	0
1319	0	1359	497	1399	76	1439	0
1320	0	1360	280	1400	282	1440	0

Appendix C (Continued)

Table C – 1 (continued)

Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c
1441	0	1481	75	1521	516	1561	0
1442	0	1482	0	1522	699	1562	0
1443	0	1483	0	1523	817	1563	0
1444	0	1484	0	1524	858	1564	0
1445	0	1485	0	1525	817	1565	0
1446	0	1486	0	1526	699	1566	0
1447	75	1487	0	1527	516	1567	79
1448	283	1488	0	1528	292	1568	294
1449	503	1489	0	1529	79	1569	519
1450	683	1490	0	1530	0	1570	702
1451	800	1491	0	1531	0	1571	821
1452	840	1492	0	1532	0	1572	862
1453	800	1493	0	1533	0	1573	821
1454	683	1494	0	1534	0	1574	702
1455	503	1495	79	1535	0	1575	519
1456	283	1496	292	1536	0	1576	294
1457	75	1497	515	1537	0	1577	79
1458	0	1498	697	1538	0	1578	0
1459	0	1499	815	1539	0	1579	0
1460	0	1500	855	1540	0	1580	0
1461	0	1501	815	1541	0	1581	0
1462	0	1502	697	1542	0	1582	0
1463	0	1503	515	1543	79	1583	0
1464	0	1504	292	1544	293	1584	0
1465	0	1505	79	1545	517	1585	0
1466	0	1506	0	1546	701	1586	0
1467	0	1507	0	1547	819	1587	0
1468	0	1508	0	1548	860	1588	0
1469	0	1509	0	1549	819	1589	0
1470	0	1510	0	1550	701	1590	0
1471	75	1511	0	1551	517	1591	78
1472	284	1512	0	1552	293	1592	294
1473	504	1513	0	1553	79	1593	520
1474	685	1514	0	1554	0	1594	704
1475	802	1515	0	1555	0	1595	823
1476	842	1516	0	1556	0	1596	864
1477	802	1517	0	1557	0	1597	823
1478	685	1518	0	1558	0	1598	704
1479	504	1519	79	1559	0	1599	520
1480	284	1520	292	1560	0	1600	294

Appendix C (Continued)

Table C – 1 (continued)

Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c
1601	78	1641	522	1681	0	1721	77
1602	0	1642	708	1682	0	1722	0
1603	0	1643	827	1683	0	1723	0
1604	0	1644	868	1684	0	1724	0
1605	0	1645	827	1685	0	1725	0
1606	0	1646	708	1686	0	1726	0
1607	0	1647	522	1687	77	1727	0
1608	0	1648	295	1688	296	1728	0
1609	0	1649	78	1689	525	1729	0
1610	0	1650	0	1690	711	1730	0
1611	0	1651	0	1691	831	1731	0
1612	0	1652	0	1692	872	1732	0
1613	0	1653	0	1693	831	1733	0
1614	0	1654	0	1694	711	1734	0
1615	78	1655	0	1695	525	1735	77
1616	295	1656	0	1696	296	1736	297
1617	521	1657	0	1697	77	1737	527
1618	706	1658	0	1698	0	1738	714
1619	825	1659	0	1699	0	1739	834
1620	866	1660	0	1700	0	1740	876
1621	825	1661	0	1701	0	1741	834
1622	706	1662	0	1702	0	1742	714
1623	521	1663	78	1703	0	1743	527
1624	295	1664	296	1704	0	1744	297
1625	78	1665	523	1705	0	1745	77
1626	0	1666	709	1706	0	1746	0
1627	0	1667	829	1707	0	1747	0
1628	0	1668	870	1708	0	1748	0
1629	0	1669	829	1709	0	1749	0
1630	0	1670	709	1710	0	1750	0
1631	0	1671	523	1711	77	1751	0
1632	0	1672	296	1712	297	1752	0
1633	0	1673	78	1713	526	1753	0
1634	0	1674	0	1714	712	1754	0
1635	0	1675	0	1715	833	1755	0
1636	0	1676	0	1716	874	1756	0
1637	0	1677	0	1717	833	1757	0
1638	0	1678	0	1718	712	1758	0
1639	78	1679	0	1719	526	1759	77
1640	295	1680	0	1720	297	1760	298

Appendix C (Continued)

Table C – 1 (continued)

Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c
1761	528	1801	0	1841	75	1881	532
1762	715	1802	0	1842	0	1882	722
1763	836	1803	0	1843	0	1883	844
1764	878	1804	0	1844	0	1884	886
1765	836	1805	0	1845	0	1885	844
1766	715	1806	0	1846	0	1886	722
1767	528	1807	76	1847	0	1887	532
1768	298	1808	298	1848	0	1888	299
1769	77	1809	530	1849	0	1889	75
1770	0	1810	718	1850	0	1890	0
1771	0	1811	839	1851	0	1891	0
1772	0	1812	881	1852	0	1892	0
1773	0	1813	839	1853	0	1893	0
1774	0	1814	718	1854	0	1894	0
1775	0	1815	530	1855	75	1895	0
1776	0	1816	298	1856	299	1896	0
1777	0	1817	76	1857	531	1897	0
1778	0	1818	0	1858	721	1898	0
1779	0	1819	0	1859	842	1899	0
1780	0	1820	0	1860	884	1900	0
1781	0	1821	0	1861	842	1901	0
1782	0	1822	0	1862	721	1902	0
1783	76	1823	0	1863	531	1903	74
1784	298	1824	0	1864	299	1904	299
1785	529	1825	0	1865	75	1905	533
1786	717	1826	0	1866	0	1906	723
1787	838	1827	0	1867	0	1907	845
1788	879	1828	0	1868	0	1908	887
1789	838	1829	0	1869	0	1909	845
1790	717	1830	0	1870	0	1910	723
1791	529	1831	75	1871	0	1911	533
1792	298	1832	298	1872	0	1912	299
1793	76	1833	531	1873	0	1913	74
1794	0	1834	719	1874	0	1914	0
1795	0	1835	841	1875	0	1915	0
1796	0	1836	883	1876	0	1916	0
1797	0	1837	841	1877	0	1917	0
1798	0	1838	719	1878	0	1918	0
1799	0	1839	531	1879	75	1919	0
1800	0	1840	298	1880	299	1920	0

Appendix C (Continued)

Table C – 1 (continued)

Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c
1921	0	1961	73	2001	536	2041	0
1922	0	1962	0	2002	727	2042	0
1923	0	1963	0	2003	850	2043	0
1924	0	1964	0	2004	892	2044	0
1925	0	1965	0	2005	850	2045	0
1926	0	1966	0	2006	727	2046	0
1927	74	1967	0	2007	536	2047	71
1928	299	1968	0	2008	299	2048	299
1929	534	1969	0	2009	72	2049	536
1930	724	1970	0	2010	0	2050	729
1931	846	1971	0	2011	0	2051	852
1932	888	1972	0	2012	0	2052	894
1933	846	1973	0	2013	0	2053	852
1934	724	1974	0	2014	0	2054	729
1935	534	1975	73	2015	0	2055	536
1936	299	1976	299	2016	0	2056	299
1937	74	1977	535	2017	0	2057	71
1938	0	1978	726	2018	0	2058	0
1939	0	1979	849	2019	0	2059	0
1940	0	1980	891	2020	0	2060	0
1941	0	1981	849	2021	0	2061	0
1942	0	1982	726	2022	0	2062	0
1943	0	1983	535	2023	72	2063	0
1944	0	1984	299	2024	300	2064	0
1945	0	1985	73	2025	536	2065	0
1946	0	1986	0	2026	728	2066	0
1947	0	1987	0	2027	851	2067	0
1948	0	1988	0	2028	893	2068	0
1949	0	1989	0	2029	851	2069	0
1950	0	1990	0	2030	728	2070	0
1951	73	1991	0	2031	536	2071	70
1952	299	1992	0	2032	300	2072	299
1953	534	1993	0	2033	72	2073	537
1954	725	1994	0	2034	0	2074	729
1955	848	1995	0	2035	0	2075	853
1956	890	1996	0	2036	0	2076	895
1957	848	1997	0	2037	0	2077	853
1958	725	1998	0	2038	0	2078	729
1959	534	1999	72	2039	0	2079	537
1960	299	2000	299	2040	0	2080	299

Appendix C (Continued)

Table C – 1 (continued)

Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c
2081	70	2121	538	2161	0	2201	67
2082	0	2122	731	2162	0	2202	0
2083	0	2123	855	2163	0	2203	0
2084	0	2124	897	2164	0	2204	0
2085	0	2125	855	2165	0	2205	0
2086	0	2126	731	2166	0	2206	0
2087	0	2127	538	2167	68	2207	0
2088	0	2128	299	2168	299	2208	0
2089	0	2129	69	2169	538	2209	0
2090	0	2130	0	2170	732	2210	0
2091	0	2131	0	2171	856	2211	0
2092	0	2132	0	2172	899	2212	0
2093	0	2133	0	2173	856	2213	0
2094	0	2134	0	2174	732	2214	0
2095	70	2135	0	2175	538	2215	53
2096	299	2136	0	2176	299	2216	265
2097	537	2137	0	2177	68	2217	495
2098	730	2138	0	2178	0	2218	684
2099	854	2139	0	2179	0	2219	806
2100	896	2140	0	2180	0	2220	848
2101	854	2141	0	2181	0	2221	806
2102	730	2142	0	2182	0	2222	684
2103	537	2143	68	2183	0	2223	495
2104	299	2144	299	2184	0	2224	265
2105	70	2145	538	2185	0	2225	53
2106	0	2146	731	2186	0	2226	0
2107	0	2147	855	2187	0	2227	0
2108	0	2148	898	2188	0	2228	0
2109	0	2149	855	2189	0	2229	0
2110	0	2150	731	2190	0	2230	0
2111	0	2151	538	2191	67	2231	0
2112	0	2152	299	2192	298	2232	0
2113	0	2153	68	2193	538	2233	0
2114	0	2154	0	2194	732	2234	0
2115	0	2155	0	2195	857	2235	0
2116	0	2156	0	2196	899	2236	0
2117	0	2157	0	2197	857	2237	0
2118	0	2158	0	2198	732	2238	0
2119	69	2159	0	2199	538	2239	52
120	299	2160	0	2200	298	2240	265

Appendix C (Continued)

Table C – 1 (continued)

Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c
2241	496	2281	0	2321	50	2361	495
2242	685	2282	0	2322	0	2362	685
2243	807	2283	0	2323	0	2363	808
2244	849	2284	0	2324	0	2364	850
2245	807	2285	0	2325	0	2365	808
2246	685	2286	0	2326	0	2366	685
2247	496	2287	51	2327	0	2367	495
2248	265	2288	264	2328	0	2368	262
2249	52	2289	495	2329	0	2369	49
2250	0	2290	685	2330	0	2370	0
2251	0	2291	807	2331	0	2371	0
2252	0	2292	849	2332	0	2372	0
2253	0	2293	807	2333	0	2373	0
2254	0	2294	685	2334	0	2374	0
2255	0	2295	495	2335	50	2375	0
2256	0	2296	264	2336	263	2376	0
2257	0	2297	51	2337	495	2377	0
2258	0	2298	0	2338	685	2378	0
2259	0	2299	0	2339	808	2379	0
2260	0	2300	0	2340	850	2380	0
2261	0	2301	0	2341	808	2381	0
2262	0	2302	0	2342	685	2382	0
2263	52	2303	0	2343	495	2383	48
2264	264	2304	0	2344	263	2384	262
2265	495	2305	0	2345	50	2385	494
2266	685	2306	0	2346	0	2386	685
2267	807	2307	0	2347	0	2387	808
2268	849	2308	0	2348	0	2388	850
2269	807	2309	0	2349	0	2389	808
2270	685	2310	0	2350	0	2390	685
2271	495	2311	50	2351	0	2391	494
2272	264	2312	263	2352	0	2392	262
2273	52	2313	495	2353	0	2393	48
2274	0	2314	685	2354	0	2394	0
2275	0	2315	807	2355	0	2395	0
2276	0	2316	850	2356	0	2396	0
2277	0	2317	807	2357	0	2397	0
2278	0	2318	685	2358	0	2398	0
2279	0	2319	495	2359	49	2399	0
2280	0	2320	263	2360	262	2400	0

Appendix C (Continued)

Table C – 1 (continued)

Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c
2401	0	2441	47	2481	493	2521	0
2402	0	2442	0	2482	684	2522	0
2403	0	2443	0	2483	807	2523	0
2404	0	2444	0	2484	849	2524	0
2405	0	2445	0	2485	807	2525	0
2406	0	2446	0	2486	684	2526	0
2407	48	2447	0	2487	493	2527	44
2408	261	2448	0	2488	259	2528	258
2409	494	2449	0	2489	45	2529	492
2410	685	2450	0	2490	0	2530	683
2411	808	2451	0	2491	0	2531	806
2412	850	2452	0	2492	0	2532	849
2413	808	2453	0	2493	0	2533	806
2414	685	2454	0	2494	0	2534	683
2415	494	2455	46	2495	0	2535	492
2416	261	2456	260	2496	0	2536	258
2417	48	2457	493	2497	0	2537	44
2418	0	2458	684	2498	0	2538	0
2419	0	2459	807	2499	0	2539	0
2420	0	2460	850	2500	0	2540	0
2421	0	2461	807	2501	0	2541	0
2422	0	2462	684	2502	0	2542	0
2423	0	2463	493	2503	45	2543	0
2424	0	2464	260	2504	258	2544	0
2425	0	2465	46	2505	492	2545	0
2426	0	2466	0	2506	684	2546	0
2427	0	2467	0	2507	807	2547	0
2428	0	2468	0	2508	849	2548	0
2429	0	2469	0	2509	807	2549	0
2430	0	2470	0	2510	684	2550	0
2431	47	2471	0	2511	492	2551	43
2432	261	2472	0	2512	258	2552	257
2433	494	2473	0	2513	45	2553	491
2434	685	2474	0	2514	0	2554	683
2435	807	2475	0	2515	0	2555	806
2436	850	2476	0	2516	0	2556	848
2437	807	2477	0	2517	0	2557	806
2438	685	2478	0	2518	0	2558	683
2439	494	2479	45	2519	0	2559	491
2440	261	2480	259	2520	0	2560	257

Appendix C (Continued)

Table C – 1 (continued)

Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c
2561	43	2601	490	2641	0	2681	39
2562	0	2602	682	2642	0	2682	0
2563	0	2603	805	2643	0	2683	0
2564	0	2604	847	2644	0	2684	0
2565	0	2605	805	2645	0	2685	0
2566	0	2606	682	2646	0	2686	0
2567	0	2607	490	2647	40	2687	0
2568	0	2608	255	2648	253	2688	0
2569	0	2609	42	2649	489	2689	0
2570	0	2610	0	2650	681	2690	0
2571	0	2611	0	2651	804	2691	0
2572	0	2612	0	2652	846	2692	0
2573	0	2613	0	2653	804	2693	0
2574	0	2614	0	2654	681	2694	0
2575	42	2615	0	2655	489	2695	38
2576	256	2616	0	2656	253	2696	252
2577	491	2617	0	2657	40	2697	487
2578	682	2618	0	2658	0	2698	679
2579	806	2619	0	2659	0	2699	803
2580	848	2620	0	2660	0	2700	845
2581	806	2621	0	2661	0	2701	803
2582	682	2622	0	2662	0	2702	679
2583	491	2623	41	2663	0	2703	487
2584	256	2624	254	2664	0	2704	252
2585	42	2625	489	2665	0	2705	38
2586	0	2626	681	2666	0	2706	0
2587	0	2627	805	2667	0	2707	0
2588	0	2628	847	2668	0	2708	0
2589	0	2629	805	2669	0	2709	0
2590	0	2630	681	2670	0	2710	0
2591	0	2631	489	2671	39	2711	0
2592	0	2632	254	2672	252	2712	0
2593	0	2633	41	2673	488	2713	0
2594	0	2634	0	2674	680	2714	0
2595	0	2635	0	2675	803	2715	0
2596	0	2636	0	2676	846	2716	0
2597	0	2637	0	2677	803	2717	0
2598	0	2638	0	2678	680	2718	0
2599	42	2639	0	2679	488	2719	38
2600	255	2640	0	2680	252	2720	251

Appendix C (Continued)

Table C – 1 (continued)

Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c	Hour	Solar_flux/I_c
2721	486	2761	0	2801	35	2841	482
2722	678	2762	0	2802	0	2842	674
2723	802	2763	0	2803	0	2843	798
2724	844	2764	0	2804	0	2844	840
2725	802	2765	0	2805	0	2845	798
2726	678	2766	0	2806	0	2846	674
2727	486	2767	36	2807	0	2847	482
2728	251	2768	249	2808	0	2848	245
2729	38	2769	484	2809	0	2849	34
2730	0	2770	677	2810	0	2850	0
2731	0	2771	800	2811	0	2851	0
2732	0	2772	843	2812	0	2852	0
2733	0	2773	800	2813	0	2853	0
2734	0	2774	677	2814	0	2854	0
2735	0	2775	484	2815	35	2855	0
2736	0	2776	249	2816	247	2856	0
2737	0	2777	36	2817	482	2857	0
2738	0	2778	0	2818	675	2858	0
2739	0	2779	0	2819	798	2859	0
2740	0	2780	0	2820	841	2860	0
2741	0	2781	0	2821	798	2861	0
2742	0	2782	0	2822	675	2862	0
2743	37	2783	0	2823	482	2863	33
2744	250	2784	0	2824	247	2864	244
2745	485	2785	0	2825	35	2865	480
2746	678	2786	0	2826	0	2866	673
2747	801	2787	0	2827	0	2867	796
2748	844	2788	0	2828	0	2868	839
2749	801	2789	0	2829	0	2869	796
2750	678	2790	0	2830	0	2870	673
2751	485	2791	35	2831	0	2871	480
2752	250	2792	248	2832	0	2872	244
2753	37	2793	483	2833	0	2873	33
2754	0	2794	676	2834	0	2874	0
2755	0	2795	799	2835	0	2875	0
2756	0	2796	842	2836	0	2876	0
2757	0	2797	799	2837	0	2877	0
2758	0	2798	676	2838	0	2878	0
2759	0	2799	483	2839	34	2879	0
2760	0	2800	248	2840	245	2880	0

Appendix C (Continued)

Table C – 1 (continued)

Hour	Solar_flux/I_c	Hour	Solar_flux/I_c
2881	0	2921	6
2882	0	2922	0
2883	0	2923	0
2884	0	2924	0
2885	0	2925	0
2886	0	2926	0
2887	32	2927	0
2888	243	2928	0
2889	479	2929	0
2890	672	2930	0
2891	795	2931	0
2892	838	2932	0
2893	795	2933	0
2894	672	2934	0
2895	479	2935	6
2896	243	2936	95
2897	32	2937	230
2898	0	2938	351
2899	0	2939	431
2900	0	2940	459
2901	0	2941	431
2902	0	2942	351
2903	0	2943	230
2904	0	2944	95
2905	0	2945	6
2906	0	2946	0
2907	0	2947	0
2908	0	2948	0
2909	0	2949	0
2910	0	2950	0
2911	6	2951	0
2912	98	2952	0
2913	233		
2914	354		
2915	435		
2916	464		
2917	435		
2918	354		
2919	233		
2920	98		

ABOUT THE AUTHOR

Monica Annmarie Gray was born in Montego Bay, Jamaica. She graduated with a Bachelors of Science (*Summa Cum Laude/* First Class Honors) from the University of the West Indies, St. Augustine Campus, Trinidad in Agricultural Engineering and a minor in Bio – systems Engineering. She then went on to complete a Master of Science in Biological Engineering at the University of Georgia, Athens.

While at the University of South Florida, Tampa, Monica undertook a PhD in Civil and Environmental Engineering in Water Resources Engineering and a Master of Public Health in Environmental and Occupational Health. Two of her life's most awesome dreams are to fly an F-16 (Fighting Falcon) Fighter Jet and to be Jamaica's first woman Prime Minister.