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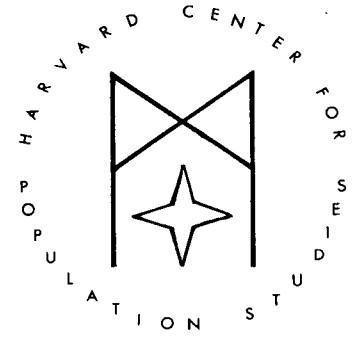

HARVARD UNIVERSITY
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PUBLIC HEALTH IN RURAL INDIA:

The Case of Excreta Disposal

by

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PREFACE

The writing of this thesis has been a source of great personal pleasure. That this would be so was far from evident two and a half years ago when I started on this venture. I was not only intimidated by the challenge this represented to my creative powers, but I was in the midst of a severe conflict concerning the compatibility of my professional training with my psychological need for involvement with people at a micro level in my work and my political concern for specific groups in the populations of poor countries. The ongoing work of which this thesis is the first part represents a resolution of these conflicts in a way that I scarcely thought possible. The enormous sensitivity of Professor Harold Thomas to my personal, political and intellectual concerns at that and, indeed, at all other times during the past five years, has been a source of great support to me.

Professor Gerrit v.R.Marais of the University of Cape Town fostered my interest in public health problems and was instrumental in my decision to come to Harvard. My two primary intellectual debts at Harvard are to my thesis adviser, Professor Thomas,and to Professor Roger Revelle. Professor Thomas gave me the rare combination of freedom and critical advice which I needed; Professor Revelle gave generously of his enthusiasm and scientific insights. I have benefitted greatly, too, from the knowledge and experience

which others have shared freely with me. In particular I would like to thank Drs.G.A.Schad, Ashok Khosla, Amulya Reddy, Gerald Winfield, A.K.Roy, G.D.Agrawal, G.J.Mohanrao, K.A.Pisharoti, Prodipto Roy, Lalit Nath, S.J.Arceivala, S.Subba Rao, David Donaldson, Pat Owens, G.D.Spangler, B.Cvjetanovic, F.James Levinson and David Bradley for their help.

This research, which included two trips to the Indian sub-continent, was funded by a Ford Foundation Grant to the Harvard University Center for Population Studies for "Research in Planning and Development of Water and Other Natural Resources in the Indian sub-continent". Our program officer, Dr.Sadik Toksoz of the Ford Foundation, New Delhi, assisted me in every possible way and our program director at Harvard, Professor Peter Rogers, gave generously of his support. I would like to acknowledge, too, the considerable contribution which Kate Gordon made to this thesis. To all my colleagues and friends on this program, but especially to James Gavan, Dick Tabors, Mahesh Chaturvedi, Vimal Shah, Ramesh Bhatia, P.S.Rao and Elizabeth Gibson, I would like to express my thanks for creating such a supportive personal and intellectual environment.

I feel considerable sadness at leaving Cambridge and the many close friends I have here. My friendships with Jochen Kühner, Glenn Withers, Carol Shea and Jack de Long have been, and are, especially important to me. My relationship with Ellis Waingrow

has been profoundly valuable to me.

Finally, in recognition of their unstinting love, support and understanding, I would like to dedicate this thesis to my parents, Dick and Thelma Briscoe.

John Briscoe
Cambridge, Massachusetts,
February 1976.

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SYNOPSIS

It is a basic tenet of public health that the high mortality and morbidity levels prevailing in poor countries are causally related to the level of environmental sanitation in these areas. One of the more prominent of these environmental problems is that pertaining to the removal and disposal of human excreta. While technical solutions to this classic sanitary engineering problem have been devised and successfully implemented in high income countries, the problems associated with the disposal of human excreta in most poor countries, and in the rural areas of these countries in particular, have yet to be adequately addressed. In undertaking this dissertation the hope was that a multi-disciplinary approach using some of the concepts and tools of "systems analysis" would provide new insights into the nature of rural excreta disposal problems and that fruitful non-traditional approaches to the practical issues involved would emerge.

The fundamental questions in this field, to which this dissertation is a response, are:

I) What is the appropriate allocation of government resources to the water supply and sanitation sector?

II) What is the appropriate intra-sectoral apportionment of the resources allocated to this sector?, and

III) What specific institutional structures and technological options should be fostered in order to initiate the

desired changes in defecation habits?

Chapter One is a descriptive account of the role of sanitation in social welfare. While there is no primary research in this chapter, it is useful as an effort to address some of the issues raised by Question I, in that the connections between a micro problem and those macro issues which are of direct concern to decision makers are made explicit. In the context of this dissertation, Chapter One specifies the overall matrix, a few of the elements of which are explored in the subsequent chapters.

In Chapters Two through Five we work our way from the micro level to the macro level, examining in depth a few of the many pertinent questions which arise.

In Chapter Two the economic concepts which have been developed for examining environmental problems in developed countries are applied to develop a framework in which to evaluate sanitation programs. Those programs which have been undertaken in rural India are examined in terms of this framework. The fundamental assumption is that the crucial factor obstructing the transformation of defecation practices in rural India is neither the absence of an appropriate technology nor the existence of a set of anachronistic beliefs, but is the perception by the populace that there are no significant tangible benefits which will accrue to the individual if that person changes his or her traditional defecation habits. Particular

attention is focussed on the factors upon which individual motivations to change traditional habits are contingent. This orientation is quite different from that which has been, and is, the norm used by planners of environmental health works and gives rise to a radically different set of attractive institutional and technological options. In Chapter Two an attempt is made to quantify the orders of magnitude of the "internal" benefits for a few of the more promising options. In summary, then, Chapter Two contributes substantially to a clarification of the way in which micro-level change may be promoted (the concern of Question III).

The analysis in Chapter Two suggests that the most propitious technological options may be those in which human excreta is treated as an agricultural and energy resource rather than as an undesirable waste product. Chapter Three consists of a more detailed analysis of the potential and actual role of night soil as a fertilizer source, and an assessment of the effects of the agricultural regime on the internal benefits to be derived from a composting operation in a village in West Bengal. A major contribution of this exercise is the collection and refining of much of the basic data on which any analysis of the economic viability of the proposed programs must be based. The chapter is also useful in developing an analytic method for appraising the tangible benefits associated with "excreta-use" programs and in assessing the effects on these benefits of policy decisions in this sector (e.g. subsidies of

communal latrines and bio-gas plants) and exogenous changes (e.g. a change in the price and availability of chemical fertilizers or a change in the crop types in the area).

While the microeconomic theory behind the models constructed in the latter part of this chapter is well established, the economic factors underlying the use of traditional fertilizer sources has been the subject of little analysis. This analysis is a significant contribution towards understanding the dynamics of the supply of and demand for organic fertilizers in a village economy.

The most important single factor inhibiting the rational allocation of resources within the public health sector (the concern of Question II) is the absence of quantitative estimates of the linkages between health interventions and disease incidence. In Chapter Four this complex problem is addressed through the medium of mathematical epidemiological models. The present analysis contributes to the existing knowledge by, first, elucidating the factors which govern the behaviour of several complex disease simulation models and using these insights to suggest some shortcomings of these models, and, second, by developing a simulation model for a soil transmitted helminthic disease. No such model has previously appeared in the literature.

A set of simple analytic models for different diseases are developed which are useful in interpreting the results of several existing complex simulation models and in elucidating the nature

of the implicit equilibrating mechanisms. An important consequence of this enquiry is the demonstration of the necessity for including non-linearities (such as density dependent effects or immunity) in models of soil-transmitted helminths. It is suggested that the exclusion of these ecological factors in the other systems examined constitutes a major limitation of these models.

Where the etiology of a disease is clearly understood these models are useful both in simulating the effects of parameter changes and in identifying paramount data deficiencies. These models are expected to contribute to empirical investigations into the health consequences of environmental changes by specifying the expected form of the relationships between various disease measures and the independent variables which may affect these measures, thus ensuring that statistical (e.g. multiple regression) models reflect the etiological realities of different diseases. Where the etiology is unclear (as in the case of the diarrheas) the assumptions which must be built into the model are unclear and valid simulation models can not be constructed. These models can be used in this case, however, to facilitate the choice of the appropriate statistical model for an empirical analysis.

The bulk of Chapter Four is devoted to the development of a set of deterministic simulation models for hookworm infestation in rural West Bengal. These models proved highly useful in providing a framework in which to organize the considerable

body of data on hookworm which has been assembled by parasitologists and epidemiologists. In the present context the models are useful in providing a mechanism for mapping sanitation changes into changes in the level of infestation in a community. The usefulness of these models for purposes of policy formation will remain severely limited until a body of data relating sanitation changes to changes in the incidences of different diseases becomes available.

In Chapter Five an attempt is made to quantify some of the health effects of a change in defecation practices in demographic and economic terms (the concern of Question I). Most previous analyses of this sort have taken the health changes as given; a feature of the present approach is the coupling of epidemiological and demographic models. Given the paucity of adequate epidemiological models, this chapter quantifies the effects of the changes, in only typhoid fever and hookworm infestation, which may arise from changed sanitation practices. While little light is shed on the many problems which arise in assessing the economic consequences of health changes in poor countries, the latter part of this chapter indicates the ways in which existing data may be manipulated to obtain meaningful estimates of the economic and nutritional consequences of changes in defecation habits.

While this thesis contributes to the extension of knowledge in several of the specific areas examined, the major contribution is the development of a framework in which to examine

issues of excreta disposal. This framework is radically different from that which has been used in the past in that it does not isolate the engineering and epidemiological aspects but builds on these fundamental issues to understand how people may be motivated to change age-old defecation habits and to address questions of the allocation of government resources to this sector.

CHAPTER ONE

INTRODUCTION

Summary

In the first section of this chapter the questions which motivated this research are specified, and the progress which has been made in attempting to answer some of these questions is outlined. In the next section attention is drawn to some of the biases which are brought to this analysis of this subject. The body of this chapter is devoted to an exploration of the links between improved excreta disposal practices and disease, nutrition, economic development and demographic change in rural India. The final section outlines some characteristics of a village in West Bengal which are pertinent to this study.

CONTENTS

1.1. THE QUESTIONS ADDRESSED

In the First Five Year Plan the Government of India allocated 0.8% and 0.35% of the total Plan expenditure to rural and urban water supply and sanitation respectively. By the Fifth Plan (1974-1979) the proportion allocated to this sector had increased considerably, with 1.5% of the budget being spent on rural, and 1.15% on urban, water supply and sanitation. The intra-sectoral allocations, too, had changed. While 83% of the rural allocation was spent on the sanitary latrine program in the First Plan, all of the rural allocation was spent on water supply after the Third Plan (World Health Organization [1974c]).

An examination of the way in which these inter- and intra-sectoral allocations have been determined reveals that the basis is largely intuitive rather than scientific. It is the objective of this dissertation to develop a systematic approach to these questions and to build some of the basic analytic blocks on which the rational allocation of resources depends.

This dissertation begins and ends with the larger issue, namely that of the role of sanitation in social welfare. In Chapter One the relationships between improved sanitation and health, nutrition, agriculture, population and economic development are discussed. In Chapter Five a methodology for translating health changes into terms relevant to inter-sectoral resource allocation questions is developed and an attempt

made to quantify some of these effects which may emanate from changed rural excreta disposal practices. In Chapter Four the complex problem of translating environmental changes into changes in mortality and morbidity rates is addressed by developing a set of mathematical epidemiological models for different fecal-borne diseases. While the development of a comprehensive set of these models is proscribed by the lack of knowledge concerning the etiology of an important subset of these diseases and by the paucity of data on the effects of environmental changes on most of the diseases, these models provide a useful heuristic, and hopefully eventually operational, approach to resource allocation questions within the public health sector.

In their seminal work on rural domestic water supply in East Africa, White, Bradley and White (1974) suggest that two primary sets of problems need to be addressed: "One relates to how much and what kind of improvement in supplies is desirable...(while) the second set of problems relates to the practical organization and means to be used to improve the supplies." A similar duality arises with respect to rural excreta disposal. While Chapters One, Four and Five have focussed on the first question, in Chapters Two and Three some issues pertinent to the second question are addressed.

In Chapter Two past and present rural excreta programs in India are examined and a radically different approach from that

which has been standard in the past is suggested. An analysis of the factors which may motivate individuals to change under each of these program types suggests that this new approach may be at least less intrinsically failure bound in trying to effect change in traditional habits. In Chapter Three some of the issues raised in Chapter Two are explored in greater depth. In particular, Chapter Three is an attempt to quantify the relationships between defecation practices and the production of organic fertilizers and to build some quantitative models for understanding the role of these fertilizers in village agriculture. In this chapter an attempt is made, too, to quantify some of the "internal" benefits and costs associated with different excreta disposal technologies.

As will be emphasised repeatedly throughout the text, this dissertation provides no definitive answers to these extremely complex questions, but rather represents the tentative first step along a long and unclear road. In Chapter Six the significant contributions of this study are summarized and the directions in which this work should proceed are mapped out.

1.2. SOME LIMITATIONS AND BIASES

In attempting to alter the traditional defecation habits of a people, we are dealing with a sensitive personal issue which is deeply embedded in the culture of a community. That which can be contributed by a Western engineer to the understanding of so complex an issue in an alien culture is, perforce, limited.

Sanitation has been recognised as an important problem by the leaders of modern India. Gandhiji (1924) laid great emphasis on this issue and considered that "the one thing we can and must learn from the West is the science of sanitation." We recognise that this focus may be confined to those Indians who have had prolonged exposure to "Occidentals [who] come from a clean and tidy material world, in which dirt, squalor and disorder are sins," (Chaudhuri [1966]), yet take our cue from these leaders.

In this analysis we will develop a framework in which to examine the agricultural, epidemiological and economic implications of different rural sanitation programs. While policy implications will be drawn from this analysis it should be emphasized that there are other equally important dimensions to the problem which are touched peripherally at best.

M. N. Srinivas (1975) has emphasized the importance of recognizing pollution-purity ideas when examining issues pertaining to human wastes. In Chapter Two evidence on the acceptability of the removal and disposal alternatives which are considered will be presented. Pollution-purity notions are implicit in

these data and thus the question is addressed, albeit indirectly.

The assumptions concerning the emergence of an adequate institutional structure for the implementation of the proposed programs are rudimentary. In essence we assume that if there is a demonstrable and clearly perceived benefit to the individual for following a certain course of action the appropriate institutional structure will be forthcoming.

That the social system itself may prove to be a major impediment to improving the welfare of a group of people is dramatically illustrated by the Government of India program to provide "bhangis" (scavengers or sweepers) with wheelbarrows for night soil collection. Isaacs (1965) has shown how a generic disinterest of caste Hindu officials in social efforts directed towards improving the lot of the Harijans, the misuse of funds by local bodies, the obstacles raised by the private contractors who hold the night-soil-removing concessions and a reluctance to change on the part of the bhangis themselves made the implementation of this program impossible. "You can start with the idea of providing a scavenger with a wheelbarrow but you end up not only having to rebuild your cities and towns but also having to make over the social system while you are at it. The scavenger's lot is clearly not a happy one, but whoever would change his lot has to shoulder the whole burden of backwardness." The appropriate response to this complexity, namely to be realistic but not resigned, is exemplified by Myrdal (1968):

> The fact that attitudes toward personal and public hygiene are woven into the very fabric of society - including not only levels of living and in particular, literacy and education, but also social stratification, sexual mores, religious taboos and rites, and superstitious ideas about how diseases are caused and transmitted - does not imply that campaigns to improve habits of hygiene are hopeless. A study of health conditions in South Asia leaves one with the impressions that there is an immense need for such campaigns.

Most of the data used in this analysis are typical of a village of one thousand inhabitants in Singur thana of West Bengal. Dube (1955) has drawn attention to "the great variation in Indian villages. . . in the factors of size, population, land area, ethnic composition, caste constitution, the pattern of land ownership, the structure of authority and the degree of contact with urban areas." The village spatial structure assumed in this analysis is "a cluster of houses and huts with the fields lying all around" (Srinivas [1955]). Srinivas has pointed out that this structure is not found in Assam, parts of Gujarat and the west coast of Southern India. India is a vast and varied land. The conclusions emerging from this study apply only to a village of the assumed structure; considerable caution should be exercised in generalizing the conclusions which emerge.

1.3. THE CHOICE OF THE TOPIC : WHY RURAL? WHY EXCRETA?

The means to the end of improving the welfare of a people are generally far less clear than the desirability of that end. In this dissertation we wish to explore a few of the issues which arise in evaluating the effects of a specific program on human welfare in the hope that analysis of this sort facilitates choice among alternatives when resources are limited. While we do attempt to evaluate the link between public health programs and other dimensions of welfare such as per capita income, this analysis is probably best seen as an attempt to construct one of the blocks on which rational allocation of resources in this field of public health rests. Exclusive concentration on excreta disposal should not be seen, therefore, as an attempt to beg the larger question as to which, in Wolman's (1974) words, "battlegrounds should have priority in public health," but is rather an attempt to block off a manageable piece of the problem.

The focus on the rural population in this dissertation was motivated by the fact that health problems arising from inadequate sanitation are greatest in rural areas. In a World Health Organization publication, Gotaas (1956) draws attention to this:

> Although population densities in urban areas are high - which increases the possibilities of disease transmission - the rate at the present time for those communicable diseases spread through inadequate environmental sanitation is highest among the rural population of the world. Insanitary disposal of wastes is the major factor contributing to this paradox.

These environmental conditions partially account for the wide disparity in death rates in rural and urban India: recent Government of India data (Panikar [1975]) give a crude death rate of 18.9 per 1000 in rural areas while this rate is but 10.3 per 1000 in urban areas. While some of this difference may be accounted for by different age structures in urban and rural populations, using life expectancy, a measure which is not affected by age structure, these differences persist. The urban and rural life expectancies are 60 and 47 years respectively (Gwatkin [1975]).

Despite the importance of rural environmental health problems, sanitary engineers in India have devoted the bulk of their attention to urban environmental problems. The Fourth Five Year Plan (Government of India [1968]) explicitly drew attention to this bias:

> The problem of sanitation in towns other than those with sewerage schemes has so far been dealt with from the point of view of improving the conditions of those in unclean occupations; no reference was made to the problem of sanitation in the villages. It is necessary for public health departments and research organizations to take special interest in the problem.

Agrawal (1966) has speculated that the indifference to rural areas arises for economic (urban communities are richer), organizational and technical (western bias) reasons.

Saunders and Warford (1974) have pointed out that "...any investment emphasis on rural areas will, on an overall basis,

result in a high to low income redistribution since rural populations are generally poorer than urban populations and since the major portion of the national revenue (on a per person basis), which is usually generated primarily from output and income-based taxes, comes from the higher income urban areas." This dissertation is intended to be part of a larger effort to attain a more equitable welfare distribution and, in particular, counteract the urban biases in development planning.

The problem of intimate contact between man and human excreta is one which has largely been resolved in the western world but which "remains to be (adequately) confronted in the rest of the world"(Wolman [1974]). Gandhi (1937) identified this as one of the primary rural development problems in India:

> The very first problem the village worker will solve is its (the village's) sanitation. It is the most neglected of all problems that baffle workers and that undermine physical well-being and breed disease. If the worker became a voluntary bhangi (sweeper), he would begin by collecting nightsoil and turning it into manure and sweeping village streets. He will tell people how and where they should perform daily functions and speak to them on the value of sanitation and the great injury caused by its neglect.

Pitambar Pant (1972) has suggested that "the greatest (environmental) problem of all (in India) is the disposal of human wastes." A considerable amount of research and extension work in this field has been carried out by international organizations and countries, including India, which have perceived improved excreta disposal conditions to be a key to improved

health. That this work has been unsuccessful in significantly altering traditional defecation habits in rural India is apparent in the descriptions of those habits by public health workers (Johns Hopkins CMRT [1970], Dutt [1962], Pillai [1941], Rao [1969]).

The technological choices which have been made and the types of programs which have been implemented in India are reflective of the fact that this problem has been considered the province of sanitary engineers and public health workers exclusively. The objective in examining this issue anew is to determine whether a somewhat broader approach, in which elements of engineering, economics, agriculture and epidemiology are examined, provides useful insights into the significance of improved excreta disposal as a public health measure and into the relationships between technological choice and program structure on the one hand, and sustained change (or lack thereof) in defecation practices, on the other hand.

Although the focus here is exclusively on the problem of excreta removal and disposal, the connections between excreta disposal and water supply are important and would be taken into account in an extension of this work. On the one hand, "for the latrine programme to develop adequately, adequate water is a prerequisite, hence the water supply and latrine programmes should tie into each other as closely as possible" (Government of India [1962]). This relationship is supported by an empiri-

cal study conducted by the Planning Research and Action Institute in Lucknow (1972), which found that the "acceptance of latrines was negatively correlated to the distance of drinking water sources." On the other hand, where there is an adequate water supply program but no rural sanitation program "the expected measure of benefits from the water supply programme may be denied to rural communities"(World Health Organization [1970]). Van Zijl's investigation of the relationship between rural water supply and diarrhea concludes that "[while] the availability of water reduces the incidence of diarrhea, without further sanitary improvements it probably does not reduce the incidence of diarrhea sufficiently to solve the problem"(1966).

My focus on excreta disposal rather than on water supply or other environmental improvements is motivated partially by an extant bias against excreta programs in India (the World Health Organization [1974b] has warned of the dangers inherent in India's "heavy investment in rural water supplies, to the almost total exclusion of rural sanitation"), and partially by the challenging nature of the problems which have been encountered in rural latrine programs, which "have had a chequered career...in India, Bangladesh and Burma...owing to lack of sustained efforts and failure to capture community interest, acceptance and self-generating involvement"(World Health Organization [1974b]). With regard to the latter, this dissertation is seen as a response to Wolman's exhortation to "undertake deep

enquiry into the human motivations that have prevented greater success. . . . in excreta removal in rural populations"(Wolman [1965]).

1.4. SANITATION AND SOCIAL WELFARE

In this section we examine the relationships between the methods of removal and disposal of human excreta in rural areas of India and health, agriculture, economic development and population. In the analysis carried out in later chapters, an effort will be made to quantify some of these links.

1.4.1. Sanitation and Health

1.4.1.1. Communicable Diseases

The prevalence of illness due to communicable diseases is extremely high throughout South Asia (Myrdal [1968]). The most widespread diseases in these countries are probably those transmitted by human feces (International Bank for Reconstruction and Development [1975], Government of India [1962]). These include cholera, typhoid fever, bacillary dysentery, salmonellosis, amebic dysentery, diarrhea, ascariasis, ancylostomiasis, poliomyelitis and infective hepatitis. In this section we present some Indian data on the prevalence of these diseases and examine the relationships between improved excreta disposal practices and reduction in illness and death.

Estimates of disease-specific morbidity and mortality rates in India vary widely due to regional differences (Das [1962]), definitional problems (Saha [1967]) and unreliable data (Dutt [1962]). The World Health Organization (1974b) suggests that

"illness resulting from the lack of safe water and sanitation is probably much more widespread and severe than is apparent from recorded vital statistics, because of deficiencies in reporting and recording cases."

The crude death rate in India in 1972 has been estimated at 16.9 per 1000 population (Panikar [1975]). The Special Study on Morbidity, conducted under the National Sample Survey (1962), estimates the prevalence of illness to be about 30% in rural areas. Data published by scientists at the National Environmental Engineering Research Institute (Mohanrao [1974] and Dave [1973]) suggest a similar overall morbidity figure.

Some estimates of the percentage of mortality and morbidity in the Indian subcontinent attributable to fecal-borne diseases are given in Table 1.1. These data indicate that these diseases are a major cause of ill-health in India.

	Mortality	Morbidity
Mohanrao (1974)	30%	50-60%
Gordon et al. (1965)	about 20%	67%
Pisharoti (1967)	67%	67%
Chandrasekhar (The Central Public Health Engineering Research Institute [1964])	33%	
The Planning Research and Action Institute (1970)		50%
Bhaskaran (1962)	60%	
Rao (1969)		21% (of those treated)
Rambhadran (1972)	28%	(fevers and diarrhea)
Roy (1975)		70%
Government of India (1969)	54%	(respiratory and gastro-intestinal)
Mosley (1968)	14%	(diarrhea only)
Robinson (1967)	(over) 30%	

Table 1.1. Percentage Mortality and Morbidity Due to Fecal-Borne Diseases

In a recent study Panikar (1975) examines the causes of the low mortality rates in Kerala (rural and urban death rates are 9.4 and 7.8 per mille respectively, while the corresponding rates for the country as a whole are 18.9 and 10.3) and concludes that the decline in mortality may be traced to public health and sanitation programmes rather than to the extension of the modern medical care delivery system.

Few careful quantitative studies of the effect of latrine use on health have been conducted. Scrimshaw's (1970) study of diarrhea in Guatemala concluded that privies as used in the villages had no influence on the diarrheas of children in the first two years of life. For adults and the population as a whole, the presence of privies did contribute to a lower incidence of acute diarrheal disease. An extensive World Health Organization Project on diarrheal diseases in seven poor countries found that "whereas basic sanitation adds only a little to the reduction of diarrheal incidence, complete sanitation can reduce it markedly" (van Zijl [1966]). After improvements were made in the excreta disposal facilities of a community in Georgia, reported diarrheal rates were reduced by 50% (McCabe [1957]). Field observations by Cvjetanovic (1971) suggest that the construction of latrines reduced the transmission of typhoid from carriers by 50%. A recent World Health Organization study in the Philippines (Azurin and Alvero [1974]) shows that a rural latrine program reduced the incidence of cholera by 68% over a

five year period. The World Health Organization (1973) reports that a program for the sanitary handling and disposal of excreta in Peru resulted in a reduction in hookworm prevalence among school children from 99% to 58% in four years. Data from the Planning Research and Action Institute (1970) in Lucknow indicate that the prevalence of hookworm infestation in a rural community dropped from 100% to 48% in three years as a result of universal use of latrines. The sharp decline in gastro-intestinal diseases observed by the All India Institute of Hygiene and Public Health (Majumdar [in the Central Public Health Engineering Research Institute (1964)]) in Singur, West Bengal was attributed largely to the introduction of sanitary latrines and safe water supply through tubewells.

Mohanrao (1974) describes "the practice of open defecation [as] one of the main causes of the high rate of recycling of enteric infections in villages;" the All India Institute of Medical Sciences (1966) implicates"the contaminated water supply, the unclean environment (insanitary disposal of human wastes), and malnutrition as contributory factors in the high incidence of diarrheal disease among children in Haryana;" the World Health Organization (1974b) believes that "in areas where cholera is endemic the lack of sanitary privies creates serious problems during emergencies;" Jelliffe (1966) suggests that "weanling diarrhea is produced by the ingestion of organisms that are not usually pathogenic, especially in the large numbers to which a child is exposed in an unsanitary environment;" the

World Health Organization expert committee on enteric diseases (1964) states that "the traditional acceptance of environmental sanitation as the fundamental feature in the long-term control of acute diarrheal disease in total population is wholly justified;" and Jungalwalla (Government of India [1964]), Anwikar (1973) and Mathur and Prasad (in the Central Public Health Engineering Research Institute [1964]) believe that successful disposal of human excreta could cut down the incidence of disease in India by 20 to 30%.

Myrdal's (1968) opinion that "disposing of human excreta is perhaps the gravest sanitation problem [in South Asia]" is shared, then, by many public health workers, who regard the provision of sanitary privies in rural areas as "the sine qua non of rural sanitation (which) as a single self-contained measure can yield spectacular results in the reduction of enteric infections and helminthic infestations" (World Health Organization), and who believe that "the residual burden of preventable illness in South Asia is not likely to be eased without strenuous efforts to remedy the grossly defective environmental sanitation conditions under which most of the people live"(Mani [1965]).

1.4.1.2. Nutrition

In recent years marasmus and kwashiorkor have increasingly been recognised as important diseases in poor countries. As a result of the demonstration that infection, and particularly diarrheal infection, frequently precipitates these diseases in children living at or near subsistence nutrition levels, increased attention has been focussed on the relationship between infection and malnutrition. The evidence which has been accumulated demonstrates conclusively the existence of such a relationship.

The interaction between nutrition and infection has been elucidated by Scrimshaw et al. (1959):

> A basic biologic fact has inadequate recognition. The interaction between nutrition and infection is dynamic, frequently characterized by synergism and less commonly by antagonism. The mistaken impression that this interrelation is of secondary importance does little harm in countries where malnutrition is rare. Where both malnutrition and infection are serious, as they are in most tropical and technically underdeveloped countries, success in control of either condition commonly depends on the other. Problems of malnutrition and infectious disease are inter-dependent in clinical management of patients and in public health programs.

The incidence of Malabsorbtion:

Baker and Mathan (1972a) found that xylose malabsorbtion was present in 92% of pre-school children and 51% of the adults studied in Southern India;Keusch et al. (1972) report that 50% of Peace Corps personnel in Pakistan developed an incapacity to absorb xylose and vitamin B_{12}.

While the relationship between xylose malabsorbtion and nutrient malabsorbtion is not well established, it is likely (Baker [1972b]) that xylose malabsorbtion indicates nutrient malabsorbtion in the long run. Given the vast number of people exhibiting xylose malabsorbtion ("almost every study involving a general population in a tropical area has shown a prevalence rate between 30% and 50% [Baker (1972b)]), and given the fact that the majority of people in these tropical areas live on marginal diets, it is certain that malabsorbtion is of considerable economic and public health significance.

Mechanisms by which Infection affects Nutrition:

Almost all infections induce changes capable of affecting the absorbtive system of the host (Scrimshaw [1959]).

Protein:

The factors underlying the adverse effects of bacterial and protozoal infections have been outlined by Gopolan and Srikantia (1973):

> There is some impairment in the absorbtion of nitrogen from the intestinal tract, but this impairment is not very pronounced. Even in severe diarrhea the fall in absorbtion of dietary nitrogen has been found to be small - from a normal 90% to about 70%. The more significant finding has been marked negative nitrogen balance arising as a result of increased excretion of nitrogen in the urine....Much of the increased nitrogen in the urine comes from a breakdown of muscle. An additional route by which an increased amount of nitrogen is lost

> is through sweat, particularly when there is much febrile sweating.

The effect of intestinal helminths is quite different. Helminthic infections interfere directly with protein utilization by competing with the host for nitrogen. Studies in Singur (Rao and Sen [1969]) revealed that hookworm infestation of a mild nature definitely interferes with protein digestion and absorbtion and that "a child with a moderate load of hookworms in its intestines always remains in a negative nitrogen balance irrespective of its protein intake." African data (reported in Scrimshaw [1968]) show that adults with heavy hookworm infection absorb significantly less nitrogen (62.5%) than worm-free subjects on the same diet (73.3%). An Indian study (quoted in Young and Scrimshaw [1974]) demonstrated that fecal nitrogen in children with moderately heavy ascariasis was thrice as high before deworming as it was after treatment. The nutritional consequences of helminthic infections are often over-emphasized, however.

> The mere presence of a helminth does not justify an assumption that it has clinical or metabolic significance. The relative importance of these infections in contributing to protein malnutrition is often over-emphasized for the simple reason that intestinal helminths are visible or are readily demonstrable microscopically whereas other infectious agents or the action of nutrients can be recognized only by complicated technical procedures. (Scrimshaw [1968])

Effect on Other Nutrients:

The nutritional status with regard to nutrients other than protein is also adversely affected by infectious diseases: data on vitamin A, thiamine, ascorbic acid and iron deficiencies due to the presence of infectious diseases are given by Scrimshaw et al. (1968). The effect of hookworm infestation on the iron balance of a subject, for instance, is of far more importance than the effect on nitrogen excretion.

> The sucking motions of hookworms lead to the loss of large quantities of blood and iron; in subjects who receive an inadequate supply of food iron, as is the case in many rural populations, such blood loss is of great importance in the causation of anaemia in the tropics. (World Health Organization [1973])

Behavioral Effects:

Infection may also interfere with food intake through loss of appetite and withholding of food (Gopalan [1973]). In the Punjab Wyon (quoted in Call and Levinson [1973]) found that "a mother, recognising the increased incidence of diarrheal infection when supplemental foods are given, and recognising the incidence of mortality from the infection, makes a conscious, seemingly sensible decision to withhold such foods." Cravioto (quoted in Barnett and Muller [1974]) has observed a similar phenomena in rural Mexico. "The mothers were reluctant to give their babies milk because many of them died of diarrhea. Proteins such as milk, eggs and meat are excellent breeding grounds for

bacteria while protein-poor substances such as gruel are not. Cravioto concludes that the price of survival for the marginales in a society unable or unwilling to improve sanitation is malnutrition."

Quantitative Assessment of These Effects:

Some estimates of the nutritional effects of specific infections are available: Woolley (1970) has estimated that the annual basic caloric requirement rises by 5.7% for a single attack of typhoid in a year. Data presented by Crowley, Pollack and Brockett (1956) on the effect of hookworm infestation on the nutritional status of Chinese troops suggest (see Chapter Five) that an adult infested with 45 worms will waste 2.75% of the calories ingested. The 1965 Food and Agriculture Organization/World Health Organization Expert Group on Protein Requirements (WHO [1965a]) suggested that obligatory nitrogen losses for adults be increased by 10% to allow for periodic stress in ordinary living due to minor infections, psychological factors and the like, but the 1973 Expert Group (WHO [1973]) cautioned that

> the quantitative effects of infections on the protein needs of an individual cannot be stated, since they are likely to vary with the frequency, severity, and nature of the infection and other host factors, including nutritional status.

Revelle has examined differences between nutritional intakes and requirements and has reported that "...existing scanty

evidence suggests that malabsorbtion may result in wastage of something like 10% of the food eaten in India."(Pugwash [1974])

The Role of the Environment:

The etiology of "tropical enteropathy", the subclinical malabsorbtion syndrome, remains unclear. Indications are that the cause is neither subclinical malnutrition nor a ubiquitous food toxin but that it "is [probably] related to exposure to a contaminated environment"(Baker and Mathan [1972a]), since "all areas in which this enteropathy occurs are those where there is a high contamination of the environment"(Baker [1972c]) and biopsies indicate that all adults in Thailand "have an abnormal flora in the jejunum, possibly related to continuous environmental contamination"(Keusch [1972]). Lindenbaum et al. (1972) have suggested the following mechanism for relating environmental contamination to subclinical malabsorbtion:

> The high incidence of recurrent diarrheal episodes in Bengali infants and children as well as in Peace Corps Volunteers shortly after arrival in Pakistan may indicate that early in life, as a result of fecal contamination of food and water, there are repeated episodes of symptomatic small intestinal infection, such as the traveller's diarrhea syndrome that may be caused by certain strain of E. coli. Such mild enteric infections may be associated with profound impairment of absorbtive function. It is possible that, as Indians and Pakistanis grow to adulthood and are exposed repeatedly to such agents, they no longer show clinical symptoms with each episode of infection, but the process of subclinical small bowel injury is perpetuated.

Environmental sanitation programs, then, may "influence the prevalence and distribution of infectious disease, which may

in turn affect nutrient intakes and needs"(WHO/FAO [1973]). Berg (1973) has suggested that "although measures such as protected water supplies, waste disposal and other forms of environmental sanitation...are not normally thought of as nutrition measures, they may be significant determinants of nutritional status or, more likely, essential in combination with food-related measures." Mitra (1974) characterizes the human body in a poor country as a "leaky nutrition bucket" and sees adverse environmental conditions as seriously hampering nutrition programs since "if the leaks are not plugged much more than necessary will be poured in to compensate for the leakage."

1.4.2. Sanitation and Agriculture

The connection between the health and agricultural effects of excreta disposal practices is frequently presented as a trade-off between the agricultural benefits and health costs of nightsoil use as a fertilizer. A World Health Organization Expert Committee on Environmental Sanitation (WHO [1950]), for instance, "recognised the agricultural advantage to be gained from the use of nitrogen matter on the soil... [but] considered that safety alone should be the primary consideration in disposal of excreta and sewage." The approach taken in this dissertation is quite different. Improved excreta utilization practices are seen as benefitting both agricultural production, through the provision of a source of organic manure, and health.

Gotaas (1956) explains that "humus from night soil, manure and garbage...contains nitrogen, phosphorus and potash which are vital to the continuing fertility of the soil." Stelmach (1966) has reviewed the literature on the effect of organic matter on soil fertility (the "tilth"effect) and drawn the following conclusions:

> Soil physical conditions are generally improved by additions of organic matter. Soil aggregation, moisture holding capacity, reduction of run-off, and soil temperature are greatly improved either by the organic matter directly, or indirectly by byproducts of organic matter decomposition. The microbial population is greatly increased when organic matter is added to the soil. The exchange capacity of soil increases with an increase of organic matter in the soil, and the leaching of plant nutrients decreases.

The Indian Council of Agricultural Research (Garg et al. [1971]) has pointed out that high crop yields induced by the addition of artificial fertilizers reduce the productive capacity of soils if the secondary and trace elements are not replenished. This replenishment may be an important function of organic manures. This ICAR publication also draws attention to the "experience of users of organic manures [which] has indicated that the quality of crops, particularly fruits and vegetables, is superior to the quality obtained with chemical fertilizers."

A major difficulty which arises in assessing the agricultural value of organic manures is the paucity of suitable data. We digress briefly here to investigate the reasons for the lack of reliable studies in this area.

Schatz (1966) has examined the neglect to which research in this area has been subject in the United States and has concluded that it "is due in large part to the chemical fertilizer industry...which was generally not interested in humus or soil organic matter because there was little if any money to be made from these substances." Martin-Leake (1966), a British biologist who spent his life working in the Gangetic plain, found that in India, too, the agricultural institutions are subsidized by, and have become appendages to, the powerful chemical fertilizer industries. Thus, while throughout rural India animal and vegetable wastes are composted and the products spread on the fields, it appears that in the Indian agricultural research establishment

there has not been a single attempt by an agricultural economist to investigate the factors underlying the supply of, and demand for, organic fertilizers at a village level. The situation with regard to energy sources is similar. Revelle (1975) has estimated that in rural India human energy and the working energy of domestic animals make up 24% of the total energy use, "non-commercial sources" such as firewood, vegetable wastes and cow dung account for 67%, while "commercial"sources account for a mere 9%. The importance of the "non-commercial" sector is clear: the paucity of studies on this sector can almost certainly be ascribed to the same constellation of forces which has led to the neglect of research on "non-commercial" fertilizers.

Changes in the price of oil over the past few years have fostered a renewed awareness of the value of these non-commercial resources. The National Committee on Science and Technology (1974a), for instance, states that "the energy and fertilizer shortages have made organic fertilization and utilization of non-commercial energy derived from dung, firewood and vegetable matter more important than ever," and "high fertilizer prices, the energy crisis, foreign exchange shortages and soil erosion in less developed countries all justify more attention for organic manures." Advocates of village level development through intermediate technology have been in powerful positions in India since Independence (K.N. Raj [1975]) but have been able to effect few lasting changes in this area. Translation of the present awareness into meaningful change depends on the force which is

brought to bear on those people and institutions whose power and privilege may be threatened and who have barred progress of this sort for so long. In the words of A.K.N. Reddy (1974):

> However convincing the arguments for alternative technologies, it is obvious that attempts to implement such a plan will arouse the opposition of most groups in the elite which have a vested interest in western technology. Whether this opposition will be overcome will depend upon whether the Government has the will to implement such a plan, and, in the last analysis, whether the pressure of the poor and the committed can generate that will when it is absent or strengthen it when it wavers.

In assessing the possibilities of using organic manures derived from human wastes, it is important to examine the oft-stated belief that "there are religious and social objections to the use of human excreta"(Richardson [1956]). A conference on the social and cultural factors affecting environmental sanitation in India (Government of India [1956a]) brought together many prominent anthropologists and agricultural experts. The consensus was that "in the mind of the cultivator his occupation has priority over his sanitary condition." In some areas the villagers have perceived the value of nightsoil manure and have spontaneously instituted programs for collecting night soil, using it with wastes and spreading the compost on the fields. In Shigli, a town in Karnataka, "hundreds of men and women of all castes work all the year round in collecting night soil manure" (Basavakumar [1975]). In towns throughout India where compost is produced the demand for the manure far outstrips the supply

(CPHERI [1970a]), indicating that those social and religious taboos which may exist do not preclude the use of mature compost.

In Chapter Three we compare the present use of organic manures in Indian agriculture with the potential which is available in human, animal and crop wastes and village refuse. The total quantity of nitrogen contained in these sources amounts to about 60 lbs of nitrogen per cultivated acre per year. Calculations suggest that it may be realistic to capture about 35 lbN/acre/year in India whereas present use is at the level of about 8 lbN per acre per year. Even with the other sources of organic manure, namely cow dung and vegetable wastes, having alternative uses, the agricultural input from organic sources and particularly from human wastes and village refuse, can be greatly increased.

1.4.3. Sanitation and Economic Development

Ragner Nurkse's (1957) description of "the vicious circle of poverty" in which "a poor man may not have enough to eat; being underfed his health may be weak; being physically weak, his working capacity is low, which means that he is poor, which in turn means that he will not have enough to eat; and so on," is painfully appropriate for many rural Indians. In Asian Drama Myrdal (1968) has repeatedly stressed the interdependence of health and economic conditions in South Asia:

> We find poverty - in its widest sense - to be a primary cause of the stubborn survival of conditions that gravely menace health, though at the same time inferior health conditions help to preserve poverty by lowering labor input and efficiency. There can be no warrant for leaving health out of the development picture. Ill health is a very serious deterrent to a rise in labour input and efficiency in the underdeveloped countries in South Asia.

Ill health may affect economic output in a variety of ways. Truncated working lives and absenteeism due to illness may reduce the availability of labour. If work opportunities are limited, economic output may not change greatly as a result of increased labour availability, but in rural areas, where labour requirements are seasonal and peaked, output may be expected to increase. The productivity of workers may be impaired by reduced physical and mental ability associated with high morbidity levels. Again, the magnitude of this effect is related to the stringency with which other constraints inhibit productivity. If

a rural sanitation program improves the level of health, it is possible for a country to cut back on some of the expenditures which are incurred for health and medical services and funnel the freed resources into income-generating channels. Wastage of other resources, such as food, may also be curtailed by improved health. There may, too, be indirect effects on economic output associated with the mortality, fertility and migration changes emanating from programs of environmental improvement. Finally, changes in attitudes and organizations engendered by environmental health programs may induce increases in output.

While these links between improved health and economic output are intuitively plausible, "a review of the literature reveals a lack of consensus in this area"(Feldstein et al.[1973]).

Malenbaum (1973) has analysed the relationship between health factors and economic output across twenty-two poor countries in Asia, Latin America and Africa. "The influence of health factors on economic output [appeared] to be quantitatively large relative to the influence of other factors, including agricultural inputs such as labour and commercial fertilizer"(Saunders and Warford [1974]). Malenbaum inferred "that the sequence from health input to economic output followed a motivational rather than physical path," and suggested "that additional health facilities can motivate workers in economically backward areas to achieve greater output in small-scale industries like agriculture and home industries."

Weisbrod et al. (1973) conducted an empirical study of the effects of infestation with schistosomiasis on labour productivity of workers on a banana estate and light industry plant in St. Lucia. The tentative conclusion emerging from this study was that "the cost of schistosomiasis infection for males is a reduction, not in market production and earnings, but leisure." Doubt has been cast on the universality of the results since "this study was plagued not only by the normal problems of field studies, but also by the valid question of whether or not schistosomiasis and the other diseases are sufficiently severe on St. Lucia to affect productivity"(Saunders and Warford [1974]).

Theodore Schultz (1966) examined the 1918-19 influenza epidemic in India in order to test the hypothesis that the marginal product of a part of the labour force in agriculture in poor countries is zero. This analysis revealed that the epidemic led to a significant fall in agricultural production in the Ganges valley.

A number of studies have attempted to assess the impact of improved nutritional status on worker productivity. In a recent study on construction and rubber plantation workers in an area of Indonesia where hookworm prevalence was 85% and about 44% suffered from iron-deficiency anemia, Basta showed that "treatment of the anemic workers with elemental iron for 60 days, at a cost of 13 US cents per labourer, resulted in an increase in productivity of approximately 19%"(IBRD [1975]).

Satyanarayana et al. (1972) examined the effects of improved

protein and caloric intakes on the working efficiency of Indian coal miners and found that "[an] improvement in nutritional status alone may not result in increased productivity, unless other operational constraints are removed."

A 1949 study on the effect of improved nutrition on the output of German miners has been used to show that malnutrition is depriving India of 49% of the physical capacity of her labour force (Correa [1973]) and that a 40% rise in builders' wages would have improved nutrition enough to raise productivity by 50% in the construction of Bhakra Nangal Dam (Healy[1965]). White, Bradley and White's (1972) comment on the effects of improved domestic water on health, namely that "[the] confident and vigorous claims have often been extrapolated beyond the evidence on which they are based," is applicable to many estimates of the economic impact of improved health and nutrition, too, the above two cases being somewhat extreme examples. The evidence on the effects of improved sanitation on economic output, then, is mixed. Quantitative estimates of this relationship should be made with extreme caution, taking into account all of the available empirical evidence and making allowances for the differences in other (non-health) constraints which may vary widely with both area and occupation.

Government policies towards environmental infrastructural development may also have income distributional implications. If the national government bears part of the cost of a rural water supply or excreta disposal program this will generally result in

a redistribution of income within the country from urban to rural and from higher to lower income groups (Saunders and Warford [1974]).

Muscat and Berg (1974) have analysed the role of nutrition in development. The quote which follows seems equally appropriate with respect to rural sanitation programs.

> ...malnutrition can [not] be treated or overcome in isolation from other elements of the socio-economic framework... better nutrition alone is [not] a panacea for underdevelopment. On the contrary, nutrition is one of the many interrelated determinants of human performance requiring advancement. We do question whether the relative importance of nutrition, its role among the many factors, has been given the attention it deserves. We also suggest that under certain circumstances nutrition may be a pre-condition to the advancement of these factors.

In concluding this section on the relationships between sanitation programs and economic factors we note that changes in the balance of payments situation and shifts of energy, fertilizer and crop prices may affect the economic viability of programs, such as those based on bio-gas or composting plants, in which human waste is viewed as a resource. Recent increases in the prices of fossil fuels and inorganic fertilizers, for instance, "have substantially increased the net benefits from the installation of gobargas plants"(Henderson [1975]).

1.4.4. Sanitation and Population

Much attention has been focussed recently on the "population problem" in developing countries. In this section we examine the ways in which excreta disposal programs may affect the underlying demographic variables.

Mortality:

Historical demographers have examined the causes for the fall in death rates during the 18th and 19th centuries in Europe. McKeown and Brown (1955) found that the mortality decline in England was not the result of medical treatment or a change in the nature of infectious diseases, but was due to changes in environmental factors such as housing, water supply, waste disposal and nutrition. Stolnitz,in his survey of the causes of mortality declines,"emphasizes the increased control of governments and societies over various types of public health measures and sanitation"(Leibenstein [1970]).

In an earlier section of this chapter evidence was presented to support the hypothesis that improved excreta disposal practices in rural India will have a significant impact on the health of the population. On historical, a priori, and empirical grounds, then, one concludes, with Mitra (1974), that the completion of the public health revolution in India, including environmental sanitation in villages and towns, is bound to have substantial effects on mortality.

Fertility:

While a fall in mortality will, ceteris paribus, result in a corresponding increase in the rate of population growth, this effect is complicated by relationships between the factors underlying the mortality decline, and the decline itself, with fertility.

Frisch (1974) has examined the biological determinants of female fecundity and has suggested that "when the observed fertility rate of a population is lower than is normally found in a well nourished, non-contracepting population....the lower fertility could be explained wholly or in part by: delayed menarche, longer than normal adolescent sterility, irregular menstrual function or the complete absence of menses due to fluctuating food supplies, higher pregnancy wastage, which also accompanies undernutrition and longer lactational amenorrhea." If, as suggested earlier, excreta disposal problems affect the nutritional status of women, fecundity may be thereby increased.

Against the certainty that reduced mortality will result in a population spurt "is the hope that a lowering of child mortality rates now will have an important effect in later years on reducing the population growth rate"(Berg [1973]). The mechanism through which "the decline in mortality rates...seems to operate as an inducement for reduced family size"(Leibenstein [1970]) is "that [a couple] need no longer build up a reserve

pool of labour by raising a large number of children and that [they] can expect all or most of the children born to survive up to and through adult working span of life" (Mitra [1974]).

T.Paul Schultz (1974) has surveyed the literature on the determinants of fertility and found that "multiple regression analysis based on both individual and grouped data indicate that the relationship between fertility and child mortality is positive and statistically significant in such varied environments as Bangladesh (1951-61), Puerto Rico (1950-60), Taiwan (1964-69), Chile (1960) and the Philippines (1968)."

A considerable body of recent research has focussed on the response of fertility to infant and child mortality at the level of the reproductive couple. Preston (1975),summarizing this work, states that "..in most of the score of populations for which data are available....most families are unable or unmotivated to replace a deceased child with another live birth, additional to those which would have been expected in the absence of a child death." The "replacement effect" seems to be strongest at the lowest and highest developmental levels, apparently because the mechanism is primarily a biological one that operates through breastfeeding. In Bangladesh, for instance, Chaudhury, Khan and Chen (1976) found that the average interval to the next birth when the latest child dies in infancy is about 12 months shorter than when the latest child survives.

Since the birth interval in breastfeeding, non-contracepting populations is often around 2.5 years, the measured replacement effect can be as high as 40%, although a level of 25% is more common (Preston [1975]). Preston points out that in India this biological mechanism is "often reinforced by conventional differences in residential and sleeping arrangements and frequency of intercourse that depend on the survival or death of an infant."

If compensation for a change in mortality is to be complete, then, fertility must be affected by the community level of mortality through channels other than the replacement of deceased children within a family (Preston [1975]). There are insufficient data available to establish the magnitude of these contextual effects.

We conclude that,while it is clear that declining infant and child mortality will lead to a reduction in fertility, the magnitude of this reduction is unlikely to be such that population growth rates will not increase.

Migration:

Since "relative levels of environmental quality in rural and urban areas are one of the factors determining rural-urban migration (Revelle [1973]), improved sanitary conditions in rural areas may reduce the flow of people into urban centres. Saunders and Warford (1974) found that "there is little evidence that in the short run a rural water supply program will have any effect

on migration" and suggest that rural health programs may instead exacerbate migration problems by developing a healthier, potentially more productive rural population for whom employment opportunities may not exist.

The other side of the sanitation-population coin is that "the severity of human excreta disposal problems may increase at a more rapid rate than the actual growth in numbers"(Revelle [1973]). This effect may arise from certain geometric relationships: assuming a settlement to be circular and that waste generation is linearly related to population size (or area), the average transportation distance to the perimeter rises proportionately to the radius, and the area:perimeter (or waste generation:disposal area) ratio also rises proportionately to the radius. As man:land ratios in rural areas rise, areas used for defecation are likely to contract and the interactions between man and feces thereby increase. The availability of space for construction in villages has proved "a big handicap in the extension of latrines" (Planning Research and Action Institute [1969]). This handicap is likely to become more serious still as population densities increase.

1.5. Description of a Village in West Bengal

In Chapters Three, Four and Five we examine the implications of different excreta disposal practices in a hypothetical village in West Bengal which has a population of one thousand people. A village of about this size in rural West Bengal has been descrived by Kochar (1974). We extract the following description of Bandipur village in Hooghly District from Kochar's study.

> The climate of deltaic West Bengal is characterized by high rainfall during the monsoon (mid-June through September), hot summers (April to mid-June) and mild winters. Bandipur is the oldest and largest of the dozen villages in a two square mile area. It has a post office, a regular market, a bi-weekly regional market, a Primary Health Center, two high schools and a number of specialists and skilled artisans [whom] the people in the neighbouring villages often use.
>
> The settlement pattern is "loosely clustered," which allows the formation of numerous small patches and corridors of grass, shrubs and bushes, small plantation and barren land within the residential area. Most of the cultivable land holdings close to the village are small and are owned by cultivators. The outlying larger holdings of the absentee land-lords are farmed on share-cropping basis. The overall human density is 2240 persons per square mile within this dense settlement area. Thirty-two percent of the village area is devoted to fields, 9% to bamboo groves, 8% under banana groves, 17% under ponds and canals, 15% under bushes and tree groves, and 20% is residential area.
>
> While the villagers can reach Calcutta in about 1 1/2 hours by frequent train services, the social life of the village is not entirely different from what one reads in accounts written a hundred years ago. The thatched mud houses, dirt paths, bullock carts, small farms, traditions and superstitions still exist hand in hand with new dress and foods, radios and furniture, wider knowledge and political awareness.

Caste status is very important in day-to-day interpersonal interactions. Separate castes often have separate hamlets (called paras). The day-to-day activities and social life of the families is largely confined to their own para and the adjoining paras.

Rice is the main crop of the area. Pulses are not grown in the area. Many households maintain a small vegetable garden close to the house. Most fields are fallow from after the winter harvest (December) until the onset of the monsoon (June).

A house generally consists of a one- or two-room residential hut, a raised verandah with kitchen enclosure, a cattleshed, a courtyard generally enclosed by walls and a pond close by. The average household comprises about seven people and five domestic animals. Water is stored within the houses in pitchers or buckets for drinking and cooking. Tube wells are available to all households and provide drinking water. All other needs for water (such as for cleaning, washing, bathing) are fulfilled at the ponds. Ponds frequently adjoin a defecation area.

Personal and indoor cleanliness are vigorously pursued. A bath every day in the pond is the norm. Although soap is generally kept in every house, its use for bathing is generally avoided, except for washing hair. Casual washing of feet, hands and mouth is repeated numerous times during the day within the house and in course of numerous visits to the ponds. During bathing and washing a handful of water is repeatedly taken into mouth and spat out into the pond.

In rural Bengali culture, defecation behaviour, fecal pollution, and interaction with the "polluted" habitats is tied up with some cultural "idioms". Most important of these is the idiom of purity and pollution. For a rural Bengali household the social, ethical and ritualistic implications of habits are intermingled with the notions about sacred and profane, sin and virtue, pure and impure. Human feces, or objects and places directly or indirectly contaminated with human feces are "impure". Since impurity is contagious, fecally polluted objects or persons are taboo. Cultural norms require that these be avoided. Some purificatory ritual must be performed to restore the normal level of "sacredness" to individuals or things so defiled. This concept is reflected in prescriptions about ablution, change of clothes, avoidance of defecation grounds, taboo for defecation in fields under food crops, and avoidance, in general, of contact with feces. However, these notions are followed varyingly in different situations. Bamboo groves are recognized as a kind of "latrine" in local parlance.

Ablution is done soon after defecation by entering a pond and rubbing the peri-anal skin with water in a squatting posture. This is followed by rubbing the hands with soil as a purifactory act. Termination of defilement is symbolized by taking by hands a mouthful of water and spitting it out. Only the left hand must be used for ablution. Rural Bengalis scrupulously avoid the use of left hand for eating or handling any food materials. These norms are followed with high conformity. A visit to a fecally polluted bamboo grove for any purpose would normally require similar rituals.

Most of the people visit the same defecation grounds and tend to choose the same area of the defecation ground for defecation. This leads to a heavy fecal infestation in highly localized areas.

Only about 9% of the households reported having some kind of a latrine, and only 1% of the stools were passed in latrines.

About 49% of the people own some kind of footwear but less than 1% claimed using footwear when they go for defecation.

CONTENTS

2.1. A Framework in which to Develop Government Policy on Rural Sanitation

In this dissertation we examine rural excreta removal and disposal problems in India using the apparatus of economic analysis "to try to understand why old institutions have not been effective and to attempt to invent new control methods that are more satisfactory"(Thomas [1974]).

In Feldstein's (1970) work on health planning in South Korea he assumes that "...the relations between health improvement and economic development can be implicitly taken into account in the weights given in the planner's preference function to reduced mortality in different age groups, to reduced disability and to the direct economic consequences." Levinson (1974) advocates the use of a similar criterion with "...the appropriate weights based on [the] perceived political and socioeconomic importance" of the different elements. We follow this emerging pattern in health program evaluation by entertaining a number of pre-selected policy options and mapping out the "response function" for each of these options. The presentation to the planners will then be of the form of "output" (including economic, health, agricultural and possible energy components) and "input" (comprising analytic, administrative - see Footnote 1 - and economic costs) vectors for different program choices. (The relationship of this criterion to other economic efficiency criteria is discussed in Footnote 2.)

CHAPTER TWO

APPROACHES TO RURAL EXCRETA PROGRAMS IN INDIA

Summary

In this chapter we use economic theory to suggest ways in which excreta disposal programs in India may be examined. Attention is focussed on the motivations underlying changes in traditional defecation practices. Existing programs in India are evaluated in this framework and a different approach, in which incentives for both participation and maintenance play a prominent role, is suggested.

The analysis of excreta disposal, and other environmental, problems "is to a large extent an application of the general principles of ... externalities"(Dorfman and Dorfman [1974]). Technological externalities refer to "more or less direct effects, other than price changes, that one decision unit may impose on another...[and which] can, and in many instances do, prevent the market mechanism from functioning efficiently"(Davis and Damien [1974]). The major external effect associated with the disposal of human wastes arises because individual defecation practices affect the health of others in the community. Other externalities arise through the effect of organic wastes on the soil, which may lead to reduced erosion and increased soil water retention capacity, and from aesthetic considerations.

Economists see the control of the use of environmental resources as occurring through the ordinary economic arrangements, through government regulation or through economic or financial inducement. The existence of significant technological externalities argues against the first method of control of defecation practices in rural India while the second method is inappropriate both in terms of personal privacy and in terms of the administrative capacity of the government. These same considerations make the levying of a charge or tax on those who do not change their traditional defecation habits an infeasible proposition and we therefore think in terms of influencing behaviour by means of subsidies in this analysis. (In Footnote 3 we discuss

briefly the implications of this intervention in the context of the theory of welfare economics.)

2.2. Rural Excreta Programs in India: A Review and Some New Directions

In the post-Independence period considerable sums were invested in rural excreta disposal programs. The major program in this area was the Government of India Research-cum-Action Project which was partially funded by the Ford Foundation during the 1950's. This project continues today although the levels of funding and activity have declined considerably. Similar programs have been undertaken by, among others, the Planning Research and Action Institute (PRAI) of the Uttar Pradesh government, the All-India Institute of Hygiene and Public Health at their rural health center in Singur, West Bengal, the Gandhigram Institute of Rural Health and Family Planning in Tamil Nadu, and the National Environmental Engineering Research Institute (NEERI) in Nagpur. The approach taken in these programs has been remarkably uniform. Much effort has been directed to designing a latrine which is suitable for rural use and acceptable to rural people. After development of a suitable design the installation of latrines in rural areas has been closely linked with health education of the rural people. The hypothesis was (and is) that education concerning the negative health effects of the present defecation practices would result in a demand by villagers for latrines. In many cases at least part of the capital cost of the latrine was subsidized to encourage acceptance.

The most striking impression from observations of, and dis-

cussions about, these programs is their lack of success in effecting any significant transformation in village defecation habits. In the words of the World Health Organization (1974b), "Rural latrine programs initiated in India, Bangladesh and Burma...have had a chequered career owing to lack of sustained efforts and failure to capture community interest, acceptance and self-generating involvement." In the areas covered by these projects about 4% of the population use latrines on a permanent basis (Subba Rao [1964], Daniel [1975], Irrulapan [1975]). Mitra (1974) has pointed out that "in any social welfare programme for communities with great income and social disparities, the measure of success of a delivery system consistently improves as one moves up from low to high income groups." With the meagre psychological returns to workers in these areas, the existing programs have effectively abandoned the effort to promote acceptance of latrines by the lower socio-economic groups; the effort that is expended is almost exclusively directed towards the upper strata in the village hierarchy. In this regard rural latrine programs are "like so many efforts at rural development in India - the cooperatives, agricultural extension, irrigation schemes, the rural health system - [which have been] designed to help the poor [but have] ended up providing subisdized services to the rich"(Cassen [1975]).

The failure of health education programs to create a demand for rural latrines stems from both the difficulties involved in demonstrating that long-established defecation habits do have adverse

health effects and the perception that the benefits associated with improved habits will largely be "external" benefits.

This failure to distinguish between socially desirable solutions and the individual motivations on which the attaining of such solutions is contingent has affected the technological choices which have been made, too. Medically-trained people frequently oversee public health projects and have tended to be almost exclusively concerned with the "safety" of the excreta disposal method, while other planners have recommended the technology with the lowest cost-benefit ratio. Scant attention has been paid to the incentives for individual adoption which are implicit in the technologies.

That the above technological choices and implementation strategies have been followed to this day is remarkable since a considerable body of data on rural latrine programs in India accords with the findings of research in motivation in suggesting reasons for the failure of these programs.

Rovenstock (1960) has examined "what research in motivation suggests for public health," and has indicated that there are three principles of motivation in public health:

1) Preventive or therapeutic behavior relative to a given health problem in the individual is determined by the extent to which he sees the problem as having both serious consequences and a high probability of occurrence in his case and the extent to which he believes that some course of action open to him will be effective in reducing the threat.

2) Behavior emerges out of frequent conflict among motives and among courses of action. Where motives themselves

> conflict and compete for attention, those which have the highest value for the individual will actually be aroused. Health matters - at least in the individual who believes himself to be healthy - are probably not as potent as are certain other motives, especially economic and social ones...
>
> 3) Health related motives may not always give rise to health-related behavior and conversely health-related behavior may not always be determined by health-related motives. [Emphasis added.]

Dutt (1962) has found that the two primary motivations for participating in latrine programs in rural India are prestige and the perception of economic advantage. The Research-cum-Action project at Poonamallee (1963) reported that "[the villagers'] preoccupation is more with economic pursuits rather than trying to find ways and means to make better their health conditions." The Planning, Research and Action Institute (1969)"found that health was not the reason for acceptance of a latrine," but that the major reasons for acceptance of latrines were "the provision of certain conveniences in addition to conferring a higher social status....From the data it appears that most of the people were not able to visualize the impact of the use of latrines on the health of the community and therefore, they valued them only in terms of personal gain. This is further supported by almost similar views expressed by the respondents when asked about the advantages and disadvantages of having latrines."(Krishna [1968]) Srivastava (1969) examined sanitary composting in rural areas and found "that people are more receptive to change if some immediate advantage is readily available. This emerges from the observation that for almost all the persons mentioned, the main stimulus

for their adoption of composting practice was to obtain better manure. They realised the importance of control of flies and thereby prevention of the spread of diseases, but since the reduction in the morbidity of such diseases was not noticeable, it could not motivate them."

The way in which motives other than those relating to health may be harnessed to improve public health is illustrated by an examination of night soil disposal practices in mainland China. For centuries Chinese farmers have valued night soil as a fertilizer but have paid a grave price in terms of health for use of this resource. Exhortations to farmers to desist from this practice were ineffective in motivating change: the agricultural benefits were clear and "internal", the health effects less clear and largely "external" (see Scott [1956] and Winfield [1948]). Over the last twenty-five years rural health in China has improved dramatically (Unakul [1975]), due partially to the fact that the government of the People's Republic has persuaded farmers to use an adaptation of the Bangalore (hot fermentation) method for composting of their organic wastes. It is clear that farmers would not use their night soil in ways which they did not perceive to be personally beneficial either in pre-revolutionary (see Hinton [1966]) or contemporary (see Wittfogel [1970]) China. The change in practice was made possible by demonstrating that the new method would provide a superior fertilizer.

The lesson in the above is that, given the fact that the

Chinese people were used to using latrines, it was not an insurmountable task for a government sincerely interested in improving living conditions to improve the method of use of this resource. In India the existing situation is one degree worse: here a major, perhaps the crucial, problem is to bring about a change in the defecation habits of the majority. If a rural people were accustomed to using latrines the improvements that have taken place in rural hygiene in China could be envisioned, the difference in institutional, climatic and cultural systems notwithstanding.

There are two distinct types of technological choices which must be addressed: the means of collection of the excreta, and the method of excreta treatment.

Individual collection may be coupled with communal treatment. Night soil collection and transportation options are effectively limited to manual collection and bullock cart or manual transportation in rural India, and any program advocating individual collection and communal treatment therefore implies an extension of the scavenging system, in the short run at least. Economic and health considerations (of both the scavengers and the general population) argue against such a solution (Kirk [1949]): the political tide has been, and continues to be, such that this technological possibility need not be considered further. Individual collection, therefore, is tied to individual treatment and communal collection to communal treatment.

While rural excreta programs in India have focussed largely on

the adoption of latrines by individual households, a number of factors suggest that this may not be the appropriate policy. The factors include the following:

i) Pisharoti (1967) has estimated that about 50% of Indian households have insufficient space for the installation of a latrine. In U.P. the Planning, Research and Action Institute found that the percentage of rural households with adequate space for a latrine varied between 80% and 29% for different districts. "The non-availability of space in many houses was due to the clustering of houses in such a way that a courtyard or open space was not available in the proximity of the house. This was especially so among the lower socio-economic groups."(PRAI [1969]) If any program is intended to have a significant effect on the habits of poor people, this factor imposes a serious constraint on the efficacy of individual latrines. With rapid population growth in rural areas this situation is likely to deteriorate further.

ii) The initial capital cost of a household latrine of the type recommended by the National Environmental Engineering Research Institute is about Rs 250 (Handa [1975]). This is a sizeable investment for the average rural family whose total income is about Rs 2000 annually with the result that "only the affluent members of the rural society who already enjoy better health for reasons of a higher

level of living and better diet can afford one"(Kawata [1965]). On a per capita basis communal latrines may be cheaper due to economies of scale in construction, higher "load factors" and reduced collection and transportation costs.

iii) For bio-gas plants, one of the more promising technological options which will be considered, individual units are not a feasible proposition except for the wealthy who also have several head of cattle. This matter has been examined by the National Committee on Science and Technology (1974a) which has concluded "that the community gas plants alone can help the poorer sections."

iv) PRAI (Krishna [1968]) has found that "only 50% (of latrines installed) were in use: 41% were not in use because their "purdah" walls had crumbled, and 25% because their pits had caved in. Other reasons for non-use were: filling of pits (10%), blockages (7%) and wrong siting (4%)". Kirk (1949) has suggested that"it is easier to ensure adequate structural maintenance for communal than individual units."

v) A much smaller educational effort may be required to ensure participation in a communal scheme than to persuade households to construct their own latrines.

These advantages notwithstanding, communal latrines have not been favourably regarded in India. The Ministry of Health (Govern-

ment of India [1966]) has found that "a public latrine is more a public nuisance now. As it belongs to everybody, nobody takes care of it. Very rarely the public latrine has a caretaker to look after it." The failure of communal latrines is almost always at base a failure to maintain these facilities adequately. In analysing different technological and institutional options, specific attention must be paid to those factors which provide incentives for maintenance.

By emphasizing communal latrines in this discussion it is not our intention to suggest that private latrines should not be used or that technical assistance should not be offered to those who wish to build latrines. We suggest that village sanitation programs should encourage the use of private latrines by those who could afford them, but should focus most resources on communal facilities for the poor.

In the same vein, by pointing out the shortcomings of the standard health education approach in stimulating a demand for latrines, we do not intend to suggest that health education is not a vital element in improving rural gastrointestinal health. Our intention is to show that this may not be the most efficient approach to changing traditional defecation habits.

In Chapters Three, Four and Five of this dissertation the complexities involved in estimating the benefits associated with changes in defecation practices are examined. In concluding this chapter we wish to make some tentative suggestions concerning the structure of appropriate village excreta disposal

programs and examine the orders of magnitude of the "internal" costs and benefits involved, and the resulting incentives for adoption of, and participation in, such programs. In deliberating on the institutional structures, we have taken heed of Berg's (1973) comment that experience with a variety of programs designed to improve the quality of life of the rural poor suggests that "those that avoid extensive administrative mechanisms are especially attractive."

In Appendix 2 we estimate the internal costs and benefits associated with several excreta disposal schemes in which latrines are attached to bio-gas plants. Two ownership systems are considered. In the first, those who will be using the gas from the digester as a cooking fuel own and operate the facility. In the second arrangement, we assume that an entrepreneur constructs the facility and pays villagers a certain amount for consistent use of his latrine. Both of these arrangements seem interesting in that the incentives for use of the latrines and maintenance of the facility are clear and "internal". The programs examined are:

1) A latrine-cum-bio-gas plant which provides cooking fuel to about six families, using:
    1.1) human excreta only, and with the facility owned and operated by an entrepreneur who pays villagers for use of his latrine;
    1.2) both human excreta and rice straw in the digester, with:

1.2.1) community ownership and operation, or

1.2.2) the facility owned and operated by an entrepreneur.

2) A large plant owned and operated by an entrepreneur and fueled by both human excreta and rice straw, which provides fuel for a diesel engine which is used either for small scale industrial purposes or for irrigation.

The calculations indicate that the most promising arrangement is one in which the community owns and operates a plant which is fed with both human wastes and rice straw. With presently available subsidies a community of about six families could meet almost all their cooking requirements and recoup an investment of about Rs 5500 in under 3 1/2 years. If a centralized, non-seasonal demand for bio-gas as a substitute for diesel existed (e.g., for small scale industrial purposes), an entrepreneur could construct a latrine-cum-bio-gas scheme, pay villagers one rupee each a month for consistent defecation in his latrine and make about a 13% return on his investment.

In Section 3.3 of Chapter Three a similar but more sophisticated analysis is performed for the case in which human excreta and village wastes are composted with rice straw. This analysis indicates that, while the benefit/cost ratio for such an operation may be quite high, the profit is unlikely to exceed about Rs 4 per capita per year. If we envision a scheme in which the individuals group together because of the return which each per-

son will receive for participation (as proposed for the bio-gas case above) the individual return is so small that it would be unlikely that this scheme would appear attractive. This operation would be a proposition only if incentive payments were not necessary to induce villager participation in the scheme. In this case the project would be attractive to either a local government agency or to an entrepreneur (assuming the standard subsidies on the capital cost of communal latrines and the refuse collection costs to be available).

These calculations are based on data of mixed quality and caution should be exercised in interpreting the results. Pilot projects on these lines in different parts of India would facilitate a refining of the cost and benefits estimates and would indicate whether the incentives could form the basis of a self-sustaining change in defecation habits in rural India.

Footnotes to Chapter Two

1) If certain benefits are theoretically shown to be available through resource re-allocation by the state, it does not necessarily follow that state action should be recommended. "As with collective goods, there is a case for collective action to achieve optimum allocation. But all this means is that if the state can ascertain and enforce a move to the optimum position at a cost less than the gain to be had, and if it can do this in a way which does not have unfavourable effects on income distribution, then it should take action"(Turvey [1963]).

2) Dorfman and Dorfman (1972) outline four efficiency criteria which are used in conjunction with an equity criterion as a basis for policy evaluation. The first pair of efficiency criteria are those relating to utility, namely Pareto optimality and social welfare maximization. In using the former, it is necessary to invoke an equity criterion to discriminate between Pareto-optimal points, whereas the "sharp utility criterion" has equity implicit in the definition of the social welfare function. The difficulties inherent in the definition and measurement of individual and social utility functions have led to the ubiquitous use of the second pair, the production efficiency criteria, in assessment of economic performance. The first of these criteria, the "broad efficiency criterion," is similar to that of Pareto optimality but concentrates on measurable outputs of goods and services and environmental quality rather than utility. Productive efficiency, which is measured by this criterion, is a necessary but not sufficient condition for Pareto optimality. The remaining efficiency criterion is GNP maximization. This criterion, despite its well-documented shortcomings, such as enshrinement of existing income distribution and difficulty in including non-monetary benefits and costs, is the most widely used. Use of the "broad productivity criterion" avoids many of the difficulties of the "sharp criterion" since it does not require either evaluation of the benefits in terms of a common numeraire or summing or discounting these benefits. The major shortcoming is that it is likely to be indecisive in choosing amongst alternatives.

3) Pigou (1932) distinguished between the case in which a person renders disservices and compensation is not given to the injured party, and the case in which the person renders services for which he receives no payment. Coase (1960), however, has shown that the responsibility for damage incurred is, from a resource allocation viewpoint, "a reciprocal one, falling no less on the afflicted party than on the perpetrator." We therefore can look at the health externalities in two ways which are logically equivalent: We can consider the result of

the present action of the individual to be the imposition of an external cost on other individuals, or we can consider the result of altered sanitation practice by an individual to be the conferring of external benefits on other individuals. Public policy is not dictated entirely by efficiency criteria, however. It is presumably some concern with equity which has led to "the classic form of government intervention, [being] the payment of a subsidy to units whose actions confer external economies on other units, and the levying of taxes upon those entities whose actions confer external diseconomies on other units"(Davis and Kamien [1974]). Administrative considerations suggest that subsidies are more appropriate than taxes in rural India and we therefore choose to think in terms of altered individual defecation practice as conferring external benefits on the community.

CHAPTER THREE

MODELS FOR ASSESSING THE AGRICULTURAL IMPLICATIONS OF SANITATION PROGRAMS

Summary

The purpose of this chapter is to develop a set of models for examining the relationships between the excreta disposal practices and the agricultural regime in a typical village of one thousand inhabitants in West Bengal. In the first section the potential contribution of organic fertilizers is estimated and compared with the actual use in rural India. The next section sketches the context in which the subsequent analysis is set by quantifying the nitrogen flows in a Bengal rice paddy. In the remainder of the chapter an optimization model,for a scheme in which human excreta and village refuse are composted with rice straw,is developed, the demand for organic fertilizers in this village is estimated and these supply and demand models are embedded in a micro-economic framework. Attention is paid to the effects of the availability of alternative organic and inorganic fertilizers on the economic viability of the operation. The analysis focusses on the costs which are borne by, and the benefits which accrue to, individuals involved in the program and on the effects of agricultural modernization and government subsidies on the scale and viability of the operation.

CONTENTS

The anthropologist M.N. Srinivas has stressed "the necessity of finding a solution to the problem of environmental sanitation [which is] integrally related to the main occupation of villagers"(Government of India [1956a]). This chapter focusses on the relationships between excreta disposal and agriculture in India as a whole and in a village of one thousand inhabitants in Singur thana of West Bengal.

3.1. ACTUAL AND POTENTIAL USE OF ORGANIC MANURES IN INDIA

In this section we will estimate the "absolute" and "realizable" potential nitrogen availability from organic manures and compare these figures with estimates of the present use of these fertilizers. Gotaas (1956) has warned of the dangers inherent in generalization in this area:

> The multiplicity and complexity of the factors affecting the quality and quantity of compostable refuse prohibits the use of any formula or rule of thumb method for determining the amount of waste material to be expected at any given place.... The quantity, characteristics and composition of wastes...vary widely with the season and in different localities.

The analysis which follows draws on data of varying reliability from different areas of India and Asia. The results should be interpreted cautiously.

3.1.1. Absolute Potential in Organic Wastes

a) Night Soil:

On Table 3.1 various estimates of the quantity and composition of human excreta in India are presented.

With respect to the nitrogen content of feces and urine the only relatively recent and reliable data appear to be those furnished by Gotaas. The total nitrogen excretion of 14 gms per capita per day given by Gotaas, seems to exceed that which may be expected under Indian conditions. Gavan and Dixon (1975) estimate the present average protein consumption in India to be about 60 gms. per capita per day. For West Bengal the appropriate figure is likely to be more of the order of 50 gms/cap/day as estimated for East Pakistan in 1965 by Revelle and Thomas (1970). The World Health Organization/Food and Agriculture Organization Expert Committee on Energy and Protein Requirements (World Health Organization [1973], Table 32) indicates that the appropriate factor for converting protein into nitrogen is 5.95 for rice. Since rice is the staple food in West Bengal and since the conversion factor is similar for the other foods commonly consumed, the daily per capita intake of nitrogen is about 8.4 grams. We assume that all of this nitrogen is passed in the excreta and that the fecal nitrogen to urinary nitrogen ratio is as given by Gotaas. We therefore assume that 1.92 gms. of nitrogen in feces and 6.46 gms of nitrogen in urine are excreted per capita per day by Bengalis. For Indians as a whole these figures are 2.30 and 7.75 respectively.

Source	Daily Grams per Capita					
			Nitrogen		Carbon	
	Feces	Urine	Feces	Urine	Feces	Urine
Gotaas (1956)	204(wet) 54(dry)	950(wet) 64(dry)	3.2	10.8	27.2	8.9
Wagner & Lanoix (1958)	200-400	600-1130				
Bell (1973)	540					
Nawalinski (1975)	250					
Subrahmanyan (1975)	450-500 (wet) 50(dry)	830				
Garg (1971)	133(wet) 30(dry)	1200(wet) 64(dry)	2.1	12.1		
Mohanrao (1973)	82(dry)		3.3			
Acharya (1951)			1.5	8.6		
Snell (1938)	72(wet) 20(dry)	935(wet) 51(dry)	1.2	8.7	9.5	7.1

Table 3.1. Estimates of Quantity and Composition of Human Wastes in India

In estimating the carbon content of human excreta we follow a similar procedure. Watt and Merrill (1963, Table 8) indicate that for a diet consisting primarily of cereals the metabolic and digestive losses are about 10.2% of the gross energy value of the diet or 11.4% of the available energy value of the diet. Gavan and Dixon (1975) estimate the total available calories in India to be about 2050 kcals per capita per day while Revelle and Thomas (1970) found the availability to the average East Bengali to be 2020 kcals/person/day in 1965. On this basis the caloric content of the excreta of the average Bengali (and Indian) may be about 230 kcals daily. If we assume that each kilogram of foodgrains contains 3500 kcals of energy, then an equivalent of about 65.6 grams of foodgrains are passed in the feces. If the energy is available in the form of carbohydrates and if we assume the molecular structure to be that of glucose ($C_6H_{12}O_6$) then the proportion of carbon to total carbohydrate is 0.4, since the atomic weights of carbon, hydrogen and oxygen are 12, 1 and 16 respectively. We may expect, on this basis, that Indians excrete 26.3 grams of carbon per person per day. Again this is, as in the case of nitrogen, somewhat lower than the figures given by Gotaas. Assuming the fecal/urinary carbon ratio to be that given by Gotaas we estimate that 19.8 grams of carbon are passed in fecal form and 6.5 grams of carbon are excreted in the urine.

b) Animal Wastes:

Some published figures on the quantity and composition of

cattle dung and urine are given on Table 3.2 (overleaf). While the estimates on the total weight of dung voided are consistent, the estimates of the nitrogen content of these wastes vary widely. We will use two different approaches to estimate the nitrogen intake (and thus excretion) of an average Indian head of cattle.

An average Indian animal weighs 250 kg. (Swaminathan [1975]). Using data given by Gavan and Dixon (1974) we find that the average Indian person weighs 35.8 kg. We know that the average Indian consumes 60 grams of protein daily (Gavan and Dixon [1975]) and if we assume that Indian animals and people consume the same weight of protein per unit of body weight, then we estimate that an animal ingests 56 lbs of nitrogen per year. Since we have data on the caloric intake of an average animal in West Bengal (from Odend'hal [1972]) we can check the validity of the above assumption on the constancy of food intake per unit weight for the case of calories. Since Indians consume about 2050 kcals per capita daily (Gavan and Dixon [1975]) the implied intake for animals is 14,300 kcals per head per day. Since Odend'hal estimates the average caloric intake to be 14,441 kcals/head/day, we see that the assumption is, for the case of calories at least, appropriate.

A second approach is to estimate the nitrogen content in the diet of an animal. Odend'hal gives the daily intake of a particular six year old cow as 0.650 kg of rice straw, 9.820 kg of sugar cane tops, 0.205 kg of mustard oil cake and 0.405 kg of

	Dung			Urine		
Source	Total lbs/day	Dry Weight lbs/day	Nitrogen lbs/day	Total lbs/day	Dry Weight lbs/day	Nitrogen lbs/day
Gotaas(1956)			15			
Garg (1971)	36		40	10		33
Acharya(1949)		6.0	27		1.4	54
New Alchemy Institute (1973)	52	10	62	20		
Prasad(1974)	22	7	46			
Mohanrao (1974a)	21					
Parikh(1963)	22	4.5				
Harris(1974)	20					
Scott (1956)	22		32	11		35
KVIC (1975)	22					
Swaminathan (1975)	22		16	8		18
Henderson (1975)	38	7.6				

Table 3.2. Estimates of Quantity and Composition of Animal Wastes per Head of Cattle

rice hulls. The nitrogen content of rice straw is about 0.5% (Odend'hal [1975] and Jalal [1968]), and we assume this figure to pertain to sugar tops, too. Dawson (1970) gives the N_2O content of oilseed cakes as 7% which implies a nitrogen content of 4.5%. We assume that this figure holds for mustard oil cakes. Watt and Merrill (1963) estimate the protein content of rice hulls to be 13.1%, implying a nitrogen content of about 2%. Applying these factors to the above diet, we find that the yearly intake of this cow was about 60 lbs. of nitrogen. Since this particular cow was consuming 3.2% more calories than the average animal, we estimate that the average nitrogen intake is 58 lbs. of nitrogen per animal per year. This figure is remarkably close to the previous estimate (of 56 lbs N/year). While Acharya estimates that twice as much nitrogen is voided in urine as in dung, Garg, Scott and Swaminathan independently assess the nitrogen content of dung and urine to be approximately equal.

We will assume that the average head of cattle in India excretes 29 lbs. of nitrogen per year in dung and an equal amount in urine.

c) Crop Residues:

Data from China (Scott [1956]), Bangladesh (Jalal [1968]) and West Bengal (Odend'hal [1972]) suggest that crop residues for wheat and rice amount to about 1400 lbs.per acre annually and contain about 7 lbs.of nitrogen per acre.

d) Refuse:

On the basis of a recent survey of the quantity and composi-

tion of refuse (by the National Environmental Engineering Research Institute - see Central Public Health Engineering Research Institute [1970a]) and data collected by Acharya (see Gotaas[1956]), we assume that about 366 grams of refuse per capita per day is collected. The moisture content of this refuse is 20% while carbon comprises about 22% and nitrogen nearly 0.6% of the dry weight. Village refuse, therefore, contributes 66 grams per capita per day of carbon and 1.72 grams of nitrogen per capita per day.

e) Total:

If we take the 1960 population to be about 440 million people (Keyfitz and Flieger [1968]) and 227 million cattle and the cultivated area to be 324 million acres (Agarwal [1967]), we have a total annual production of 9.2 million tons of nitrogen and:

"Absolute" Potential (lbN/acre)	=	2.5 (human feces)	+	8.5 (human urine)	+	20.3 (animal dung)	+	20.3 (animal urine)	+	7.0 (crop wastes)	+	1.9 (refuse)

= 60.9 lbs nitrogen per acre per year.

Check Stevens Allison (19..) [Annexure 7.9]
BD → average irrigated
Card → 107 mds/acre
Unirrigated 55 mds/acre

3.1.2. Present Use

Acharya (1951) estimates that the amount of cattle manure actually used for fertilizer in India in 1951, when the bovine population was 150 million, was equivalent to 0.45 million tons of nitrogen or 6.7 lb N/head/year. Acharya thus implicitly assumes that 25% of the dung produced by cattle is used as fertilizer.

Estimates of the percentage use of total dung production for fuel and fertilizer vary considerably: Parikh (1963) estimates that fuel burning accounts for 36% of dung use; Singh (1973) assumes this figure to be 75%; Harris (1974) estimates that 50% of the dung is used for fuel and 50% for fertilizers, and Garg (1971) has estimated that 33% of the total dung produced is used as manure. Odend'hal (1972) quotes two recent Indian studies which respectively estimated that 40% and 75% of dung was used as fuel. For Singur thana in West Bengal, Odend'hal accepts the Ministry of Food and Agriculture estimate of 66% fuel use and 34% fertilizer use. In this analysis we will accept Acharya's estimate and assume that 25% of total dung production is used as fertilizer.

For 1960, then, with 227 million head of cattle, the annual nitrogen input is estimated to be 0.73 million tons.

The Department of Agriculture of the Government of India has estimated that in 1965-66 about 3 million tons of compost were produced (Cheema [1968a]) while in 1960-61 about 66 million tons

of village compost were produced (Cheema [1968a]). Town compost is likely to contain about 1% of nitrogen (Acharya [1951], Gotaas [1956], Parikh [1974]), while the nitrogen content of village compost, with a lower proportion of night soil, is estimated to average 0.5% (Agarwal [1967]). We therefore estimate that in 1960-61 about 0.03 and 0.33 million tons of nitrogen were added to the soil through urban and rural composting.

Kochar (1975) found that 43% of the stools passed in rural West Bengal were passed in fields. We follow Jalal (1968) who estimated that "the effective fertilizing value of [human excreta] under existing practice [in Bangladesh] is only 10.5% of the total fertilizer value of raw night soil," and assume that Kochar's data are representative of India as a whole. Using our estimate for the amount of nitrogen passed daily, we therefore have 0.37 lbs. of nitrogen per capita, or a total of 0.07 million tons per year available to the crops.

The total addition through organic manures in 1960-61 was, therefore, 1.16 million tons, or:

Total use =	5.0	+	0.2	+	2.3	+	0.5
	(cattle manure)		(town compost)		(village compost)		(field defecation)

= 8.0 lbs. N/acre/year

We note that these estimates are similar to those presented by the National Committee on Science and Technology (NCST) as the achievements of the Fourth Five Year Plan (Government of

India [1968]). NCST estimated that 0.06 million tons of nitrogen from urban wastes and 1.27 million tons from rural wastes, were used in 1965-66. The comparable estimates above are 0.03 and 1.13 million tons, respectively, for the year 1960-61.

3.1.3. A Comparison between China and India

The agricultural effects of excreta disposal practices in India are often contrasted with those in China where "use is made of every conceivable source of manure "(Grist [1953]). Richardson (1956) has discussed the ecological basis of the farms which have been operating for "four thousand years"(King [1927]):

> For many centuries before the present time the methods of manuring described in Chinese agricultural literature have resembled those of today: the most important source of plant-food, especially of readily available nitrogen, is night-soil; ashes, cattle and pig manure, composts, and green manures, as well as other local materials like oilseed cakes and crushed bones, are also employed.

Dawson (1966), in an analysis of fertilizer and food supply in The People's Republic of China, has estimated the contribution of organic fertilizers to Chinese agriculture on the following basis:

Animal Manure: Based on "various sources" it is assumed that each animal produces 7 metric tons of manure. (For Indian cattle Parikh's [1963] figure is 6.5 tons.) Dawson assumes that nitrogen constitutes about 0.4% of this production (Parikh [1963]

assumes 0.65%; Agarwal [1967], 0.5%.), and that 70% of the animal manure is available (Prasad [1974] and Parikh assume that 75% of the dung is collected in India.) The annual contribution (in 1956) from the 116.5 million animals in China is, therefore, about 2300 thousand metric tons of nitrogen.

Night Soil: Dawson assumes the per capita production of night soil is 0.25 metric tons per year and that 60% of the night soil is used for fertilizing purposes. He further assumes that the night soil contains 0.5% nitrogen. Since the human population in 1956 was 625 million the nitrogenous contribution of human wastes was 468 thousand metric tons.

Compost, Plant Residues etc.: Dawson estimates (on the basis of crop yield data) that one metric ton of wastes, 70% of which is available and 0.4% of which is nitrogen, is produced per animal unit. The contribution is,therefore, 324 thousand metric tons of nitrogen. The 1956 production of food (cereals plus potatoes) was 182.5 million tons.

The above three sources account for 87% of the organic fertilizer (the remainder coming primarily from oil cakes and mud). Organics account for 91% of the total fertilizer applied in 1956 in China.

These statistics and the implicit proportionalities are summarized on Table 3.3, next page.

	Nitrogen (1000 metric tons)	Proportionalities	
Animal manure	2,283	(lb.N/animal)	43.5
Night soil	468	(lb.N/capita)	1.66
"Compost"	324	(lb.N/tons of food)	3.9

Table 3.3. Organic Fertilizer Use in China

Given the form in which the earlier Indian statistics were presented, it is interesting to estimate what the 1961 availability of organic fertilizer in India would have been if the proportionalities which have been calculated for China were to pertain. We would have:

Animal manure	$= 43.5 \times 227 \times 10^6 / 324 \times 10^6$	= 30.5 lb.N/acre
Night soil	$= 1.66 \times 440 \times 10^6 / 324 \times 10^6$	= 2.3 lb.N/acre
"Compost"	$= 3.9 \times 100 \times 10^6 / 324 \times 10^6$	= 1.2 lb.N/acre
	TOTAL	34.0 lb.N/acre

It is tempting to think of these figures as estimates of the "realizable" potential of organic wastes and to compare these with similar estimates, presumably made in quite a different fashion, by the National Committee on Science and Technology (1974b). The NCST figures are remarkably similar, with the potential amounting to 1.1 and 34.0 lb.N/acre/year from urban and rural sources respectively.

These data tentatively suggest, therefore, that about 25% of the utilizable nitrogen available in human, animal and crop wastes and village and town refuse is utilized under present

Indian practice. What the optimal level may be is unclear. Many commentators have suggested, for instance, that all cow-dung should be returned to the field and not used as a fuel, while Roger Revelle has suggested that more nitrogen fertilizer would be produced if cow dung were collected and used as a feed-stock for large inorganic fertilizer plants. What is clear is that the use of organic wastes as fertilizers cannot be considered in isolation from the rural, non-commercial energy system. Scott's (1956) comment that "part of the nitrogen famine in China is really because of a fuel shortage, and any attempt to solve the problem of the need for increased fertilizers must therefore take this into account" is applicable for India, too.

The analysis of fertilizer use of organic wastes which is presented in this thesis is, then, partial. Henderson (1975) has pointed out that "in relation to villages as well as urban areas, attention has been almost entirely concentrated on commercial sources of energy." A complete analysis of the role of organic wastes in Indian agriculture awaits the development of adequate models of the rural non-commercial energy sector.

3.2. NITROGEN CYCLE IN A RICE PADDY

In this section the nitrogen cycle will be schematically illustrated, and an attempt will be made to determine the strength of the nitrogen inputs to, and outputs from, the ecosystem in a particular agricultural regime: a rice paddy in West Bengal. The data for this hypothetical village are taken from the work of the Johns Hopkins Center for Medical Research and Training (1970) team in Singur thana of West Bengal. The village is assumed to have a human population of 1000, an animal population of 230 cattle and 156 acres under aman paddy. The primary objective of this exercise is to develop an understanding of the role of night soil in the nitrogen cycle of rice. This knowledge will subsequently be used in evaluating the agricultural effects of different sanitation programs and, conversely, in evaluating the sanitation significance of changes in the sources, and the price of sources, of nitrogen.

The nitrogen cycle, following Buckman and Brady (1966) is illustrated in Figure 3.1 on the next page.

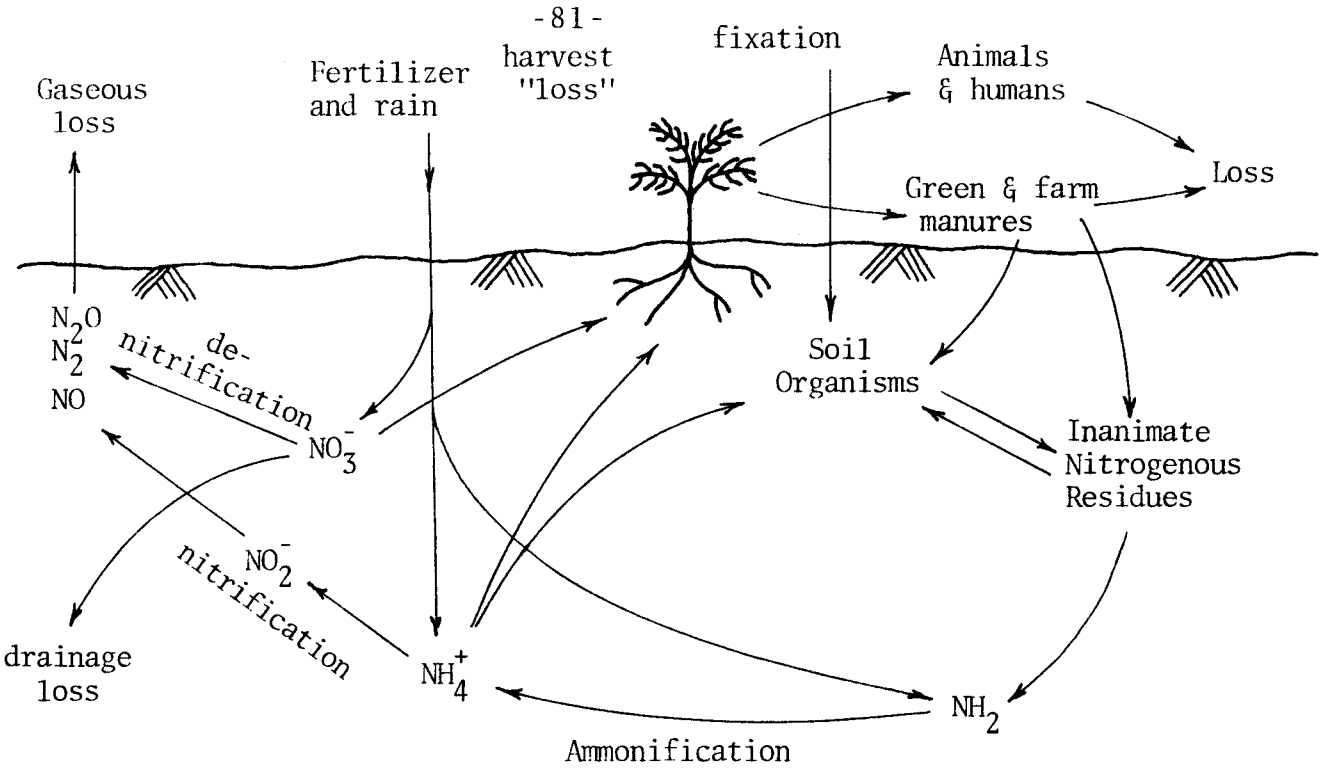


Figure 3.1. The Nitrogen Cycle

A complete quantitative analysis of the above cycle is beyond the scope of the present study. Instead, we will consider the soil-plant system to be a "black box" and concentrate on estimating the strength of the input and output arrows for the system presented in Figure 3.2 below:

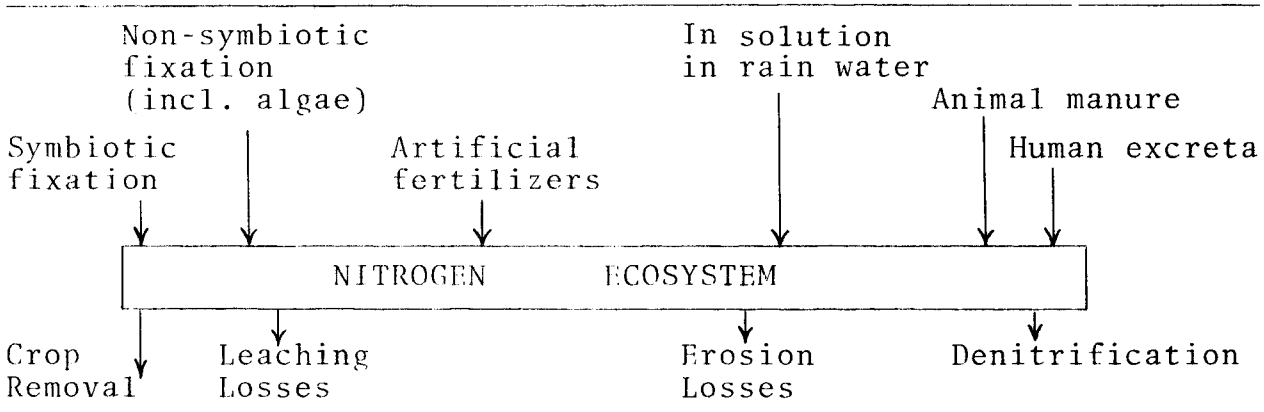


Figure 3.2. Simplified Representation of Nitrogen Flows through an Ecosystem

Grist (1953) has pointed out that "the manuring of paddy is such a complicated problem... that it is unwise to be too definite on the subject." Some of the data used in this analysis are reliable and appropriate while other figures are little more than guesses. The nitrogen "balance" presented here should be regarded as tentative.

3.2.1. Inputs

i) Rural Compost:

The Department of Agriculture data presented earlier suggest that about 1.7 lbs. of nitrogen per capita per year is made available to crops from rural compost. Assuming this figure to pertain to Singur thana we have 11 lbs.N/acre per year from rural compost. This figure is remarkable high, due primarily to the high man to land ratio.

ii) From Field Defecation:

The data of Kochar (1975) and Jalal (1968) pertain to Bengal and suggest that 0.36 lbs. of nitrogen per capita become available through defecation in the fields. The contribution is thus 2.3 lb.N/acre/year from this source.

iii) Animal Manure:

The data examined earlier indicate that about 7.3 lbs. of nitrogen per head of cattle per year become available from animal manure. The 230 head of cattle in the village supply 1668 lbs. of nitrogen annually, or about 11 lbs. per acre per year. The contribution of cattle relative to man in this area is a great deal lower than it would be for India as a whole: in Singur thana the cattle to man ratio is about 0.23 while the comparable all-India figure is 0.41.

iv) Precipitation Input:

Buckman and Brady (1966) indicate that the nitrogen input

through precipitation in the tropics is greater than the 5 lbs. of nitrogen which is estimated to enter the soil through this mechanism each year under a humid-temperate climate. Odum (1969) reports the annual rainfall input of nitrogen to be as much as 3 gm/m^2 (27 lbs./acre) in the wet tropics. Agarwal (1967) found the addition through rain water to vary from 3 to 8 lbs. per annum per acre in India. In West Bengal we assume the annual input through rainfall to be 7 lbs. of nitrogen per acre per year.

v) Fixation by organisms:

Kochar (1975) indicates that few pulses are grown in this area. We therefore assume that symbiotic fixation is insignificant.

a) Fixation by Bacteria

In ecosystems where plants with nitrogen-fixing symbionts are absent, free-living bacteria are a source of nitrogen fixation. The level of fixation is affected by a multitude of factors, including the addition of large quantities of carbon-rich substances such as straw, which may considerably increase the level of nitrogen accumulation by free-living nitrogen fixers, and the introduction of nitrogen-containing mineral substances which usually suppress the assimilation by free-living bacteria (Mishustin [1971]). Mishustin estimates that fixation through this mechanism probably does not exceed a few kilograms of nitrogen per hectare per year. Delwiche (1970)

found a figure of 2 or 3 kgms per hectare per year to be reasonable. In rice soils, azotobacter, one of the more important of the nitrogen fixing bacteria, are found in large quantities (Grist [1953]) and we therefore assume that the level of nitrogen fixation is to the upper end of the range given above. We assume that free-living bacteria fix 3 lbs. of nitrogen per acre per year in the rice fields of West Bengal.

b) Fixation by Blue-Green Algae:

Blue-green algae seem to be an important source of fixed nitrogen under conditions such as those prevailing in rice paddies, which favour their development. Odum (1969) ascribes the maintenance of fertility in rice paddies under extensive cropping primarily to this source of fixation. While much remains to be learnt of the effects of different conditions on algae nitrogen fixation (the evidence concerning the effect of the introduction of nitrogen-containing mineral substances, for instance, in conflicting), the results of many Japanese investigations suggest that the mean level of accumulation is between 20 and 25 kgN/hectare per year (Mishustin [1971]).

We assume that blue-green algae fix about 20 lbs. of nitrogen per acre annually.

vi) Input through Green Manuring:

Green manuring of paddy has given good results in several countries, including India (Food and Agriculture Organization [1966]). Dhanchia (Sesbania aculeata) is used as a green manure

crop with paddy in India. It is ploughed in when it is about five weeks old and paddy transplanted a week or two thereafter. Agarwal (1967) has estimated the nitrogen addition to be about 30 lbs. per acre. Kochar's (1975) description of agricultural activities in Haripal thana, however, suggest that green manuring is not practiced in this area and it is this assumption which we follow here.

vii) Artificial Fertilizer Input:

In India in 1970 the average application of nitrogen through chemical fertilizer was about 10 lbs. per acre of rice (Gavan [1974]). We assume this to be the application in Singur thana.

3.2.2. Losses

i) Removal by Crop:

The average yield of irrigated aman (winter) paddy in West Bengal is about 1940 lbs/acre (Government of India [1975]). We assume this to be the yield in the field considered.

Grist (1953) estimates that a production of 1550 lbs. of paddy per acre removes from the soil 20 lbs. of nitrogen in the form of grain and 11 lbs. in straw. The total nitrogen removed thus amounts to 2% of the paddy yield. Mishustin (1971) indicates that for a grain yield of 1000 kg/ha about 30 kg of nitrogen is removed from the field with the paddy and chaff. If we assume that the husk accounts for one third and the grain two thirds of the paddy weight (Revelle - personal communication), then the nitrogen removed from the field again comes to 2% of the paddy yield.

Odend'hal (1972) estimates that in Singur thana of West Bengal between 2300 and 4250 lbs. of rice straw are produced per acre annually. If we assume that the rice straw yield is twice the paddy yield and if we take Jalal's (1968) estimate of 0.5% nitrogen in the straw along with the U.S.D.A. (Watt and Merrill]1963]) estimates of about 1.2% and 2% nitrogen in the grain and husk, then for 100 lbs. of grain we have:

	Total Weight (lbs.)	% Nitrogen	Weight Nitrogen (lbs)
Grain	100	1.2	1.2
Husk	50	2.0	1.0
Straw	200	0.5	1.0

The nitrogen removed from the field (3.2 lbs.) amounts to 2.1% of the amount of paddy removed (150 lbs.).

We assume, then, that for a paddy yield of 1940 lbs./acre, 39 lbs. of nitrogen per acre is removed from the soil.

ii) Volatilization Losses

The amount of nitrogen lost in denitrification depends on the amount of nitrate nitrogen introduced and the soil conditions. The denitrification reaction is strongly favoured in those areas where anaerobic conditions prevail. While the soil in paddy fields may be partially aerated "through the gentle movement of the water through the terrace"(Geertz [1966]), conditions are likely to be largely anaerobic in wet paddy soils (Food and Agriculture Organization [1966]). Buckman and Brady (1966) suggest that perhaps 20% of the nitrogen added to the soils is lost by volatilization, while Mishustin (1971) found the losses to be between 10 and 35% of the nitrogen additions. We assume that 20% of the nitrogen input (of 66 lbs./acre/year) or 13 lbs./acre/year, is lost through volatilization.

iii) Leaching Losses:

Buckman and Brady (1966) estimate that about 5 lbs. of nitrogen may be lost annually from a representative humid mineral soil by leaching. Agarwal (1967) reports the results of an experiment under Bihar conditions which indicated that the average loss of nitrogen due to leaching was 9 to 12 lbs. per acre per

year on 6 feet depth columns. We assume that leaching accounts for 6 lbs. of nitrogen loss per acre per year.

iv) Erosion:

The only estimate of losses due to erosion which was found is that of Buckman and Brady (1966) who estimate that lands with a moderate slope may lose as much as 25 lbs. of nitrogen per acre annually. Grist (1953) points out that, more than any other cereal crop, wet rice helps check erosion. We arbitrarily assume that 4 lbs. of nitrogen are lost from each acre annually through erosion.

3.2.3. Nitrogen "Balance"

"The most striking feature of the flooded paddy terrace as an ecosystem is its extraordinary stability or durability, the degree to which it can produce with virtually undiminished yield."(Geertz [1966]) On this basis we assume that the nitrogen cycle will be in strict balance and we have adjusted the available data to ensure this balance. The annual flows through this balanced ecosystem are represented on Figure 3.3 below:

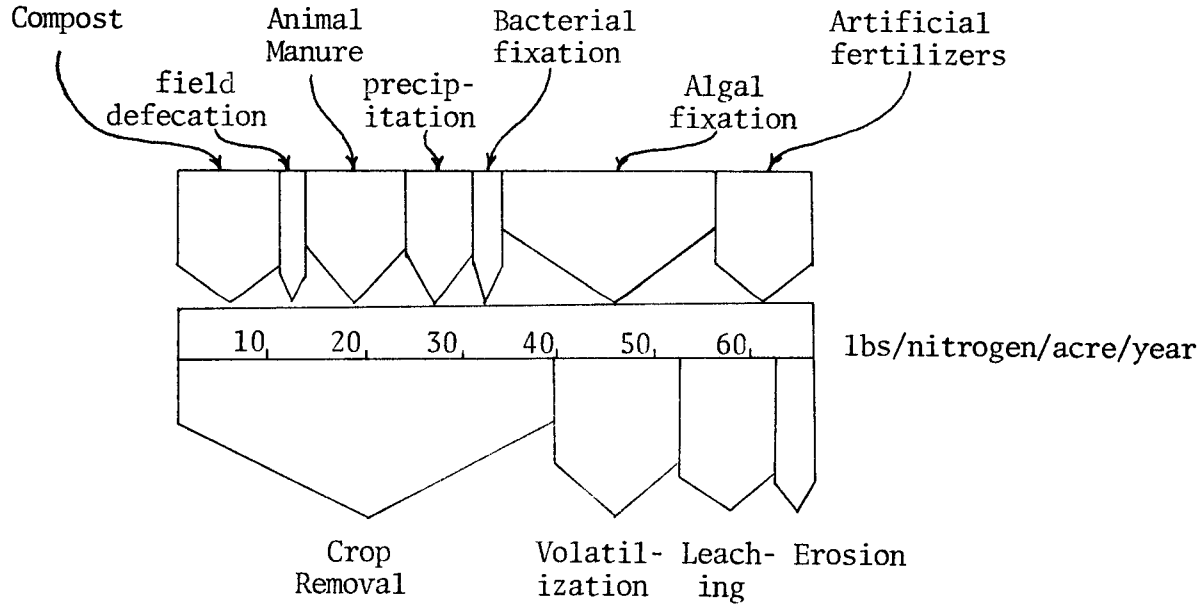


Figure 3.3: Estimates of Nitrogen Flows through a West Bengal Rice Paddy

3.3. AN ASSESSMENT OF THE ECONOMICS OF VILLAGE-LEVEL COMPOSTING

3.3.1. Theoretical Basis of the Analysis

In this analysis of the economics of a village composting operation we will assume that the primary value of compost is as a source of nitrogen to the plants and that there are two alternative sources, namely animal manure and artificial fertilizers, for augmenting the available nitrogen.

3.3.1.1. Inclusion of Animal Manure

A supplier of animal manure is assumed to operate under conditions in which he has insufficient power to be able to affect the market price of manure. We assume that the assumptions underlying the perfect competition model (homogeneous product, perfect knowledge, utility and profit maximization, atomistic competition, free entry and exit of resources) are reasonably approximated for this product. The "animal manure industry" supply curve represents the relation between market prices and the quantities that the producers are willing to supply.

The production of compost is intimately related to excreta and refuse disposal practices in the village and is likely to be subject to village health ordinances. We assume that public health considerations specify that this operation is to be centralized: we assume a single composting operation in the village of one thousand inhabitants in West Bengal. The compost supplier

then, is in an entirely different position from the animal manure suppliers: he is a monopolist for whom price and quantity are not independent variables. The monopolist's demand curve is not a horizontal line at a given price, but is the market demand curve. Since the compost supplier is able to set the market price rather than accepting it as a given datum, it is meaningless to ask what quantity the compost supplier would produce if market prices were given and there is no "supply curve" for the composter.

From the point of view of the compost supplier the market demand curve for compost is the total demand curve for nitrogen less the supply curve for animal manure. At a price p (in Figure 3.4), for example, the compost supplier knows that the demand for nitrogen will be q_d, while the supply, at that price, of animal manure will be q_s. The demand for compost at price p will, then, be $q_d - q_s$ (for $q_d > q_s$) or zero (for $q_d \leq q_s$).

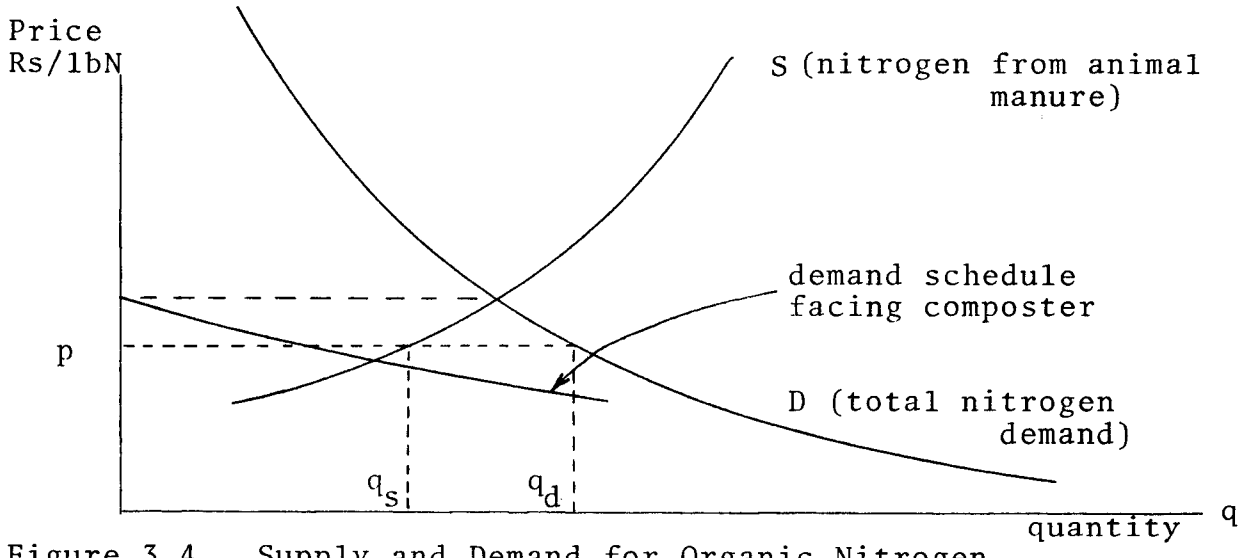


Figure 3.4. Supply and Demand for Organic Nitrogen

This demand schedule is, from the point of view of the compost supplier, the market demand schedule.

We assume that the compost supplier maximizes profit. His profits are $\pi(q) = pq - TC(q)$ (Where TC = total cost [annualized] and q = annual demand).

To maximize profits we have:

$$\frac{d\pi}{dq} = \frac{d}{dq}(pq) - \frac{d}{dq}[TC(q)] = 0$$

or, $MR(q) = MC(q)$ (with $d[MR(q)]/dq < d[MC(q)]/dq$ as the second order condition).

Graphically, the compost supplier's price and output decision can be represented as follows, where MC = marginal cost, MR = marginal revenue, AC = average cost and AR = average revenue.

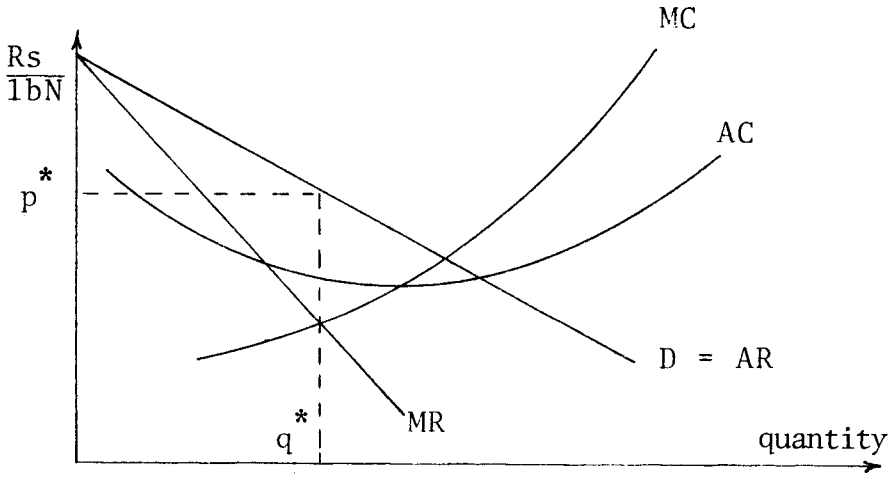


Figure 3.5. Graphical Determination of Composter's Optimal Decision

Since the plant size in this analysis is a decision variable, we examine the long run case. The most profitable short run and long run decisions of the firm are identical when long run equilibrium is attained (Cohen and Cyert [1965]).

3.3.1.2. Inclusion of Artificial Fertilizers

In this section we will outline a method whereby the availability of artificial fertilizers can be included in these models.

Two major assumptions in this section concern (a) the nature of the fertilizing action of organic and inorganic fertilizers, and (b) the nature of the supply of artificial fertilizers in the village.

With respect to the nature of the fertilizing action there are competing hypotheses on which a model can be built:

(i) We can assume (as is implicitly done in presenting the value of organic fertilizers in terms of nutrients only) that organic and inorganic fertilizers are simply two means to the same end, namely, nutrient provision. This assumption is implicit in most work in which the benefits to be derived from these two fertilizer sources are compared.

(ii) We can assume that the relationship between organic and inorganic fertilizers is more complex than that of alternative nutrient sources. One aspect of this hypothesis is that humus improves tilth and provides trace elements and nutrients while artificial fertilizers provide nutrients alone. This, in itself,

does not invalidate the assumption of independence. It is, instead, an argument for the inclusion of all the benefits of organic fertilizers in technological evaluations.

(iii) The above hypothesis forms the basis for the assertion (made by, among others, Sir Albert Howard) that artificial fertilizers should not be used to the exclusion of organic fertilizers. The notion is that the maintenance of a desirable physical structure in the soil (affecting the water retention capacity of the soil, the availability of air and the accessibility to the plants of the nutrients in the soil) is essential if nutrient provision is to be effective in increasing crop yields. This hypothesis suggests the use of a function which relates "agricultural utility" to provision of organic and inorganic fertilizers. That is, we would like a function of the form $U = f(I,O)$ where U = agricultural utility, I = quantity of inorganic fertilizer and O = quantity of organic fertilizer. ("Agricultural utility" rather than "crop yield" is used here since proponents of this hypothesis suggest that the benefits of soil improvement through the addition of humus are more comprehensive than a simple increase in current crop yield. They would argue that improved soil quality has spatial and temporal effects which are not accounted for by short-term crop yields on the specific plot on which the organics are applied.)

The data required for estimating the functional relationships in the third hypothesis are not available and are unlikely to

be collected.

The second hypothesis could be incorporated by comparing the sustained yields on fields with equivalent nutrient applications through chemical and organic fertilizers and then using a "penalty function" approach. Scientists at the National Environmental Engineering Research Institute in Nagpur have used this penalty function approach in evaluating the feasibility of alternative refuse disposal methods in Calcutta (Central Public Health Engineering Research Institute [1970a]) with the basic data being interviews with farmers rather than long term experiments. They estimated the "non-nutrient" benefits of compost by determining the equivalent amount of lime which would be used as a soil conditioner if organic fertilizers were not used. The data available for use of this method are insufficient for our purposes and we therefore revert to the conventional "nutrients only" hypothesis in this analysis. Where possible the results will be compared with those available from long-term experiments.

With respect to the nature of the artificial fertilizer supply in the village,we will assume that the prices in the village are set by the government and will investigate the effect of a constraint on the total availability of inorganic fertilizers in the village. In this analysis it is assumed that there is no "black market" in fertilizers.

The government distributor of artificial fertilizer is the source of the following supply of nutrients:

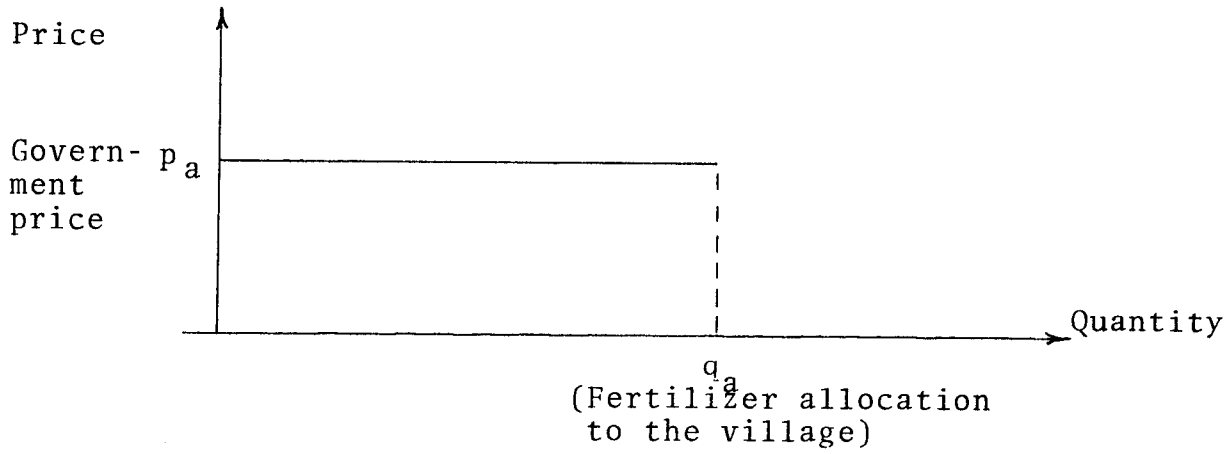


Figure 3.6. Supply Curve for Artificial Fertilizers

The provision of this alternative nutrient source affects the composting decision by altering the average and marginal revenue curves facing the composter. This effect is determined both by the quantity of fertilizer available (q_a) and the relationships between p_a, the artificial fertilizer price, p_o, the selling price of compost (in Rs/lbN) under pure monopolistic conditions, p', the price at the intersection of the average revenue and marginal cost curves, and p_{01}, which is specified by the intersection of the marginal revenue and marginal cost curves. We examine a few cases graphically to illustrate the method to be followed in evaluating these effects.

Case I: q_a large, $p_a > p_o$

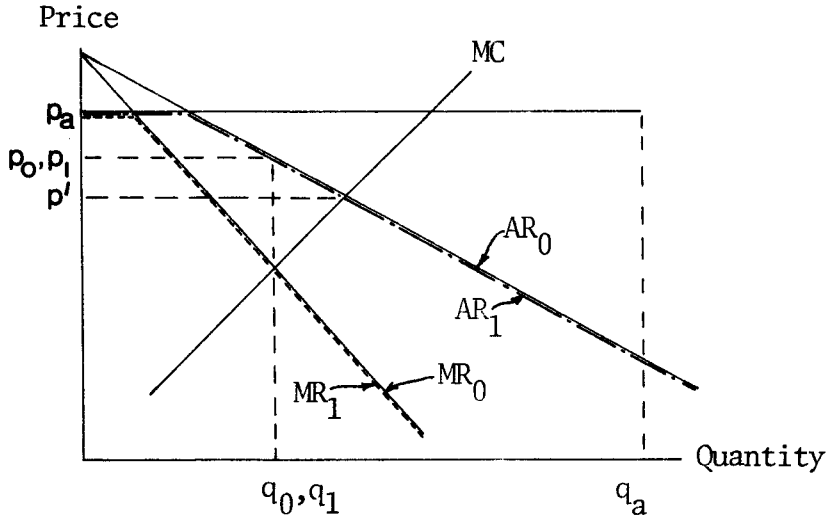


Figure 3.7: Graphical Analysis of the Effect of Artificial Fertilizers: Case I

In this case we see that the monopolist is unaffected by the availability of artificial fertilizers.

Case II: q_a large, $p' < p_a < p_0$

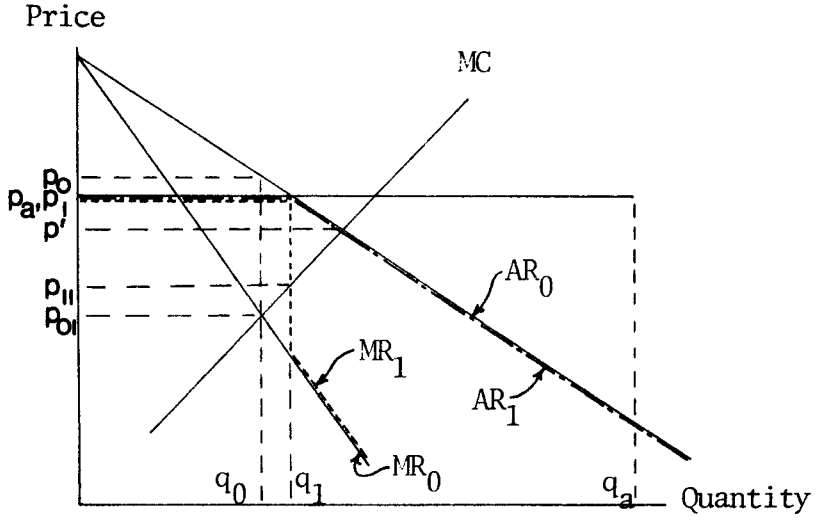


Figure 3.8: Graphical Analysis of the Effect of Artificial Fertilizers: Case II

In Case II the quantity of organic fertilizers produced is increased to q_1 and the price is reduced to p_1 (where $p_1 + \varepsilon = p_a$ for small ε). The monopolist's profit is reduced from $(p_0-p_{01})q_0$ to $(p_1-p_{11})q_1$. No artificial fertilizers would be used.

Case III: q_a large, $p_a<p'$

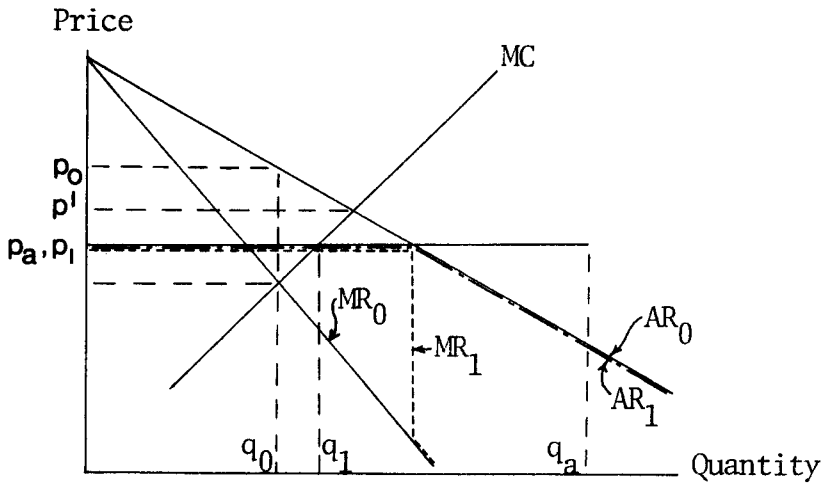


Figure 3.9: Graphical Analysis of the Effect of Artificial Fertilizers: Case III

At $p_a=p'$, q_1 reaches a maximum. In this case the "monopolist profit" is zero. In the above case the output is q_1 units and the price is p_1. The zero profit point, at which it is no longer economical for the composter to operate, is specified by the total cost function.

In the analysis which follows we attempt to quantify the various supply, demand and cost curves and investigate the

effects of the agricultural regime and government subsidies on the economic viability of a village composting scheme.

3.3.2. Marginal Cost Relationship

3.3.2.1. Technological Relationships

i) Nitrogen Retention:

For the reasons suggested earlier in this chapter (see also section 3.3.3)we will analyze compost exclusively in terms of nutrient provision. The ratio of utilizable carbon to nitrogen (the C/N ratio) in the raw materials "is an important determinant of [the] immediate utility [of a compost] in crop production"(Goluecke [1972]). The bacteria which convert the organic wastes utilize carbon for energy and nitrogen for building cell structures. These bacteria use up carbon about 30 times faster than they use nitrogen and the"optimal" C/N ratio is thus about 30.

If the C/N ratio is too high, decomposition decreases when the nitrogen is used up. Some of the organisms die and their stored nitrogen is then used by other organisms to form new cell material with the concomitant burning of more carbon. This process of reducing the C/N ratio takes time, however, since several cycles of organisms may be required to burn up most of the carbon. The proportion of nitrogen which is available for plant growth (in the oxidixed nitrite and nitrate forms) in a given digestion time would be lower than that available at the optimal C/N ratio.

When the C/N ratio is too low, on the other hand, the carbon soon becomes exhausted and the organisms get rid of the excess

nitrogen as ammonia, thereby decreasing the nitrogen availability to the plant.

Jalal (1968) has examined Indian data on aerobic composting and derived the following relationship:

Percent nitrogen conserved = $-19.22 + 5.57\ (C/N) - 0.07(C/N)^2$

(for values for C/N between 10 and 40).

ii) Raw Materials

Human wastes: We assume that 204 gms wet weight (54 gms dry weight) of feces, containing 2.3 gms of nitrogen and 19.8 gms of carbon, and 950 gms of urine (64 gms dry weight) containing 7.7 gms of nitrogen and 6.5 gms of carbon, are passed per capita per day in rural India.

The quantities of wastes available for composting may be considerably less than indicated above. Wagner and Lanoix (1958) estimate that 1000 gms/cap/day are collected in a conservancy system, while the Indian National Committee on Science and Technology (1974) has estimated this quantity to be only 450 gms/cap/day. If we assume that all fecal matter is collected the former figure implies that 84% of the urine is collected while the latter implies that only 26% is collected. We have no further data on which to base an estimate of this figure. In this analysis we will assume that 40% of the urine is collected. This figure seems reasonable but should be recognised for what it is, namely a qualified guess.

Village refuse: Indian data (presented earlier in this

chapter) indicate that village refuse contributes 66 gms per capita per day of carbon and 1.72 gms of nitrogen per capita per day.

Rice straw: In this analysis we will be examining a hypothetical village which may be considered typical of Bengal. We follow Jalal (1968) in assuming that the only suitable carbon-rich additive available is rice straw. (In any specific case this assumption would have to be reconsidered in the light of local resource availability. In many of the humid areas of India water hyacinth is a serious problem. This aquatic weed has spread over large areas of canal, tank and river surfaces. Indications are (National Committee on Science and Technology [1974b]) that composting with this material may be both remunerative and a practical approach to reducing a serious environmental problem.)

Jalal (1968) has estimated that the annual yield of rice straw in Bengal is 780 lbs. per capita. Odend'hal (1972) found that per capita rice straw production in Singur thana in West Bengal is between 390 and 720 lbs./capita/year.

In this analysis we will examine the effect of uncertainty in the estimate of the available rice straw by assuming that the per capita availability is either 780 or 390 pounds per capita per year. We will use Jalal's estimates of the moisture content (5%) and carbon and nitrogen contents (38.22% and 0.5%, or 1011 gms C/day and 13.2 gms N/day for each ton of

straw per year).

iii) Nitrogen Availability:

If we let:

P = Population size

Z1 = proportion of population contributing feces and urine to compost

W1 = proportion of feces available from those who contribute

W2 = proportion of urine available from those who contribute

Z2 = proportion of village refuse added to compost

and S = tons rice straw purchased annually for use in compost,

then:

$$\text{Initial C/N} = \frac{(19.8W1 + 6.5W2)Z1P + 66.0Z2P + 1011S}{(2.3W1 + 7.7W2)Z1P + 1.72Z2P + 13.2S}$$

and

$$\text{Available Nitrogen} = [(1.85W1 + 6.19W2)Z1P + 1.38Z2P + 10.62S] \times \left[\frac{-19.22 + 5.57(C/N) - 0.07\ (C/N)^2}{100}\right] \text{ lb.N/year.}$$

iv) Quantity of Compost:

After Gotaas (1956) and Agarwal (1967) we assume that the dry weight loss during composting is 30% and that the moisture content of the final product is 40%. The quantity of compost produced is thus:

$$Q = \frac{0.7}{0.6}\ [(.0194W1 + .0230W2)Z1F + .1078Z2F + 0.95S] \text{ tons/year.}$$

3.3.2.2. Cost Relationships

In this analysis we will consider two alternatives regarding night soil collection. We will investigate systems in which we have: (i) individual latrines constructed at the owner's expense with the night soil collected by sweepers who are paid partially by the village public health authorities and partly by those whose latrines are serviced; and (ii) communal latrines which are financed entirely by the public body.

i) Costs of Night Soil Collection from Private Latrines

The most common form of rural latrine is the box-and-can privy or conservancy system. Some data are available on the costs incurred by the public authority in servicing such latrines. Wagner and Lanoix (1955) characterise these costs as "staggering" and estimate that the cost per pail (serving about 10 people) ranges from Rs. 20 to Rs. 70 per annum.

The following data were collected in the town of Chinnallapatti in Tamil Nadu (personal communication from public health official) in February 1975. The village council services the latrines of 250 families. They employ 3 "scavengers" and one night soil cart driver at salaries of Rs. 180 per month. The night soil cart, costing Rs. 1500, and the pair of bullocks, costing Rs. 900 each, were expected to transport the night soil for a period of 5 years before new equipment and animals were required. Estimates of the operation and maintenance costs involved were not available but these were not considered significant expenditures. Amortizing the capital costs at 6% per

annum (the interest rate for Government securities [Central Public Health Engineering Research Institute (1970a)]), the total cost incurred by the municipality is about Rs. 785 per month of which Rs. 720 is for the labour involved. Assuming that each latrine services about 10 people (Wagner and Lanoix [1958]), the per capita cost incurred by the municipality is about Rs. 3.77 per annum.

The city of Madurai in Tamil Nadu incurs a cost of about Rs. 12.5 lakhs per annum for the servicing of the latrines for 180,000 people. This amounts to Rs. 7 per capita per year. The majority of this expenditure is in the form of wages to the scavengers who service about 40 households of 10 people each and are paid Rs. 200 per month by the Madurai Corporation (personal communication, Chief Public Health Engineer, Madurai Corporation [1975]). (These costs do not take into account either the capital costs incurred by the household for the construction of latrines or the payments made by individual households to the sweepers serving them. The latter costs have been estimated to be between Rs.2[Subrahmanyam (1975)] and Rs. 10 [CPHERI (1964)] per household per month. While these costs are not borne by the public authority they are of significance in that they provide a powerful disincentive against changing traditional defecation practices.)

In his analysis of regionalization of composting in Bangladesh, Jalal (1968) assumed that night soil collection costs are related to population density and the size of the collection area.

If we let P = population size, n = the number of composting "regions" within the city, and d = population density, and if we assume that $d = \alpha P^{\beta}$ where $0 \leq \beta \leq 1$, then the relative per capita collection costs in two cities are:

$$\left(\frac{P_1}{P_2}\right)^{-\frac{1-\beta}{2}} \left(\frac{n_2}{n_1}\right)^{\frac{1}{2}}$$

The per capita collection costs in large and small cities may be similar, then, through the process of regionalization. Our data are too few to test the relationship between per capita costs and population size and density. Wagner and Lanoix (1959) have examined a more extensive set of data and do not suggest any such relationship. In this analysis we will assume that a per capita collection cost of Rs 5 per annum is incurred irrespective of population size and density.

ii) Costs of Communal Latrines:

In considering private facilities we assume that the latrine cost is borne by the family and, therefore, do not include this item in the public portion of the cost of the system. When considering communal latrines all costs are borne by the public authority and are to be included in the total cost formulation.

Reliable data on the costs of latrines are few. Bhaskaran (1962) found the cost of latrines to vary from Rs 30 to Rs 100. Makhijani (1975) reports an average cost of Rs 50 per latrine while the P.R.A.I.(1969) found the total cost of an eight-seated communal latrine to be about Rs 2500. In this

analysis we will use the National Environmental Engineering Research Institute figure of Rs.250 per unit (Handa [1975]) as an estimate of the total cost of a latrine.

Several estimates have been made of the number of persons who can make use of a unit in a communal latrine. Wagner and Lanoix (1958) assume that each squatting plate can service ten people, while Makhijani (1975) estimates this figure to be 50 people per unit. Data collected by the Planning Research and Action Institute (1969) indicates that 10 people per unit is a reasonable figure while Patel's (1970) empirical figure is 20. A World Health Organization study in the Philippines (Azurin and Alvero [1974]) found that each communal latrine unit could serve 25 to 30 people. In this analysis we will assume a use factor of 20 persons per unit.

The Planning Research and Action Institute (1969) also reports data on the cost of maintenance of a communal facility. The sweeper who cleans the latrines is paid ten paise per month for each family (of five) using the facility. For a population of 1000 this would amount to Rs.20 per month. In 1974 prices this figure is about Rs.30 per month. If the salary for a sweeper in this area of U.P. is similar to that paid in Tamil Nadu, this implies that this job alone would not fully employ one sweeper. This seems reasonable given that one sweeper can service 40 to 80 family conservancy units.

In the village of Chinnallapatti in Tamil Nadu the per capita

cost of transportation of the collected night soil is Rs. 1.09 per annum. In the present case the night soil "collection" is far more centralized and an average cost of Rs.1 per capita per annum for transportation of night soil to the compost plant, which we will use in this analysis, appears to be a generous estimate.

Amortizing the capital costs at 6% per annum over a thirty year period, the total annual per capita cost of collecting the night soil and delivering it to the compost plant is about Rs.2.30 (in 1974 prices).

iii) Refuse Collection Costs:

The town of Chinnallapatti employs 24 bhangis (sweepers) and has 6 collection units, comprising a driver, a cart and a pair of bullocks, for collection of refuse in this town of 25,000 inhabitants. The annual per capita cost is Rs.2.80.

The city of Madurai spends about Rs. 12.5 lakhs on refuse collection for 600,000 people. The per capita cost is thus Rs. 2.1 per annum.

These few data suggest that per capita collection costs may be similar over a large range of city sizes. As in the case of night soil collection, the absence of any relationship between per capita collection costs and population size and density is the result of a set of counterbalancing factors. In this analysis we assume per capita refuse collection costs to be constant at Rs. 2.20 per capita per year (in 1974 prices).

iv) Costs of the Composting Operation

Data on the costs of manual composting operations are few. We investigate two cost/quantity relationships:

a) Determining economy of scale factors from European and American data and calibrating the functions on the basis of data from Asian cities.

In his analysis of composting in Bangladesh, Jalal (1968) analysed data on mechanical composting plants in Europe collected by Kupchik (1966) and found that the cost of treatment is $29.2\ Q^{-.317}$ Rs./ton, where Q is the quantity of waste in 10^4 tons/year. A similar exponent (-0.42) emerges from an analysis of American data collected by the Environmental Protection Agency (see Goluecke [1972]) and data on a mechanical composting plant in Bangkok indicate a similar relationship (McConnell [1968]).

The Central Public Health Engineering Research Institute [1971] has given estimates of the costs of mechanical and manual composting plants to treat 100 tons per day in India. These are Rs. 15 and Rs. 6 (in 1970 prices) or Rs.21 and Rs. 8.4 in 1974 prices per ton of compost respectively.

Jalal (1968) implies that the above economies of scale would characterise this cost/quantity relationship for composting operations in urban and rural Bangladesh. If we accept this hypothesis and calibrate the function on the basis of a cost of Rs. 8.4/ton for a 100 tons-per-day plant, we find:

$$\text{Cost} = 235\ Q^{-0.317} \quad \text{(Rs./ton)} \quad ,$$

where Q is the quantity of compost in tons per year.

The common method of composting in India, the Indore method, involves stacking layers of refuse, organic matter and night soil and turning these stacks at specified intervals. A priori it seems unlikely that there are significant economies of scale in such a process, and analysis of an extremely limited sample of Indian composting operations tends to confirm this hypothesis. A 100 tons/day plant (Calcutta - CPHERI [1971]) had a unit cost of Rs 8.4 per ton, a 55 tons/day plant (Madurai - personal communication, compost plant superintendent) had a unit cost of Rs 7 per ton, and an 8 ton per day plant (Chinnallapatti - personal communication, Town Public Health Engineer) had a unit cost of Rs 5 per ton. (In the latter plant the low cost is partly accounted for by the fact that the stacks were not turned.) Acharya (1949) has estimated that the hand labour requirement is about one man day per ton. At a wage of Rs 180 per month, this amounts to about Rs9.00 per ton. (Note that land amortization costs are not included in this cost.)

In this analysis we accept the hypothesis that there are no significant economies of scale in the composting operation and assume the cost of composting to be Rs 10 per ton.

v) Cost of Rice Straw:

Rice straw is a valuable resource in rural Asia, having utility as a construction material, fodder and fuel. Jalal (1968) has estimated that the opportunity cost of the rice straw is related

to the amount used in the composting process by the function:

$$w = 15 + 125(S/P)^2 \quad \text{(in 1968 prices)}$$

which is $w = 22.8 + 189.8(S/P)^2$ (in 1974 prices),

where w is the price in rupees per ton, S is the quantity of straw in tons per year, and P is the population size.

We will use this relationship when assuming that the per capita availability of rice straw is 780 lbs/cap/year and will use the function

$$w = 22.8 + 759.1(S/P)^2$$

when assuming that the per capita availability is 390 lbs/cap/year.

If the maximum available rice straw is used this function indicates that the straw costs about Rs 45 per ton. Recent data (Parikh [1974]) indicate that cow dung cakes sell for Rs 55 per ton. Since the caloric values and burning efficiencies of rice straw and cow dung cakes are similar, Jalal's estimates seem reasonable.

vi) Cost of Transportation to Fields

In this analysis the cost of transporting the ripe compost from the heap to the field will be included in the costs borne by the composter. The composter's marginal revenue curve is based on the price of compost at the field and can be used directly with the marginal revenue curves (derived later) which are based on crop response data.

If the village land area were circular (of radius r), and if the compost plant were in the centre of such an area, if there were direct access from the compost plant to each field, and if

the demand for compost were not systematically related to the distance from the centre, then the average distance over which the compost would have to be carried would be 2r/3. The configuration of the fields and the access roads is likely to be rather different. We arbitrarily assume that the average travel is r.

The total area occupied by the 1000 inhabitants and their fields is about 0.35 square miles and the effective haulage distance is about one third of a mile. The Central Public Health Engineering Research Institute (1970a) has estimated that the cost of refuse haulage in Calcutta is about Rs 1.84 per ton per mile; Popenoe (1975) has estimated transportation costs in developing countries to be between Rs 1.7 and 2.5 per ton per mile in 1970; Jalal (1968) implies a mixed refuse transportation cost of Rs. 2.29 per ton mile; and Dave (1966) found the cost of hauling refuse in Nagpur to be Rs 1.29 per ton mile. In this analysis we will assume the cost of transporting compost to be Rs 2 per ton mile in 1970 or Rs 3 per ton mile in 1974 prices.

3.3.2.3. Decision Problem

We solve the following programming problem (which arises in the case of pure competition):

Maximize NR, where NR = pq - TC(q), and NR is net revenue,
p is the price of nitrogen, q is the quantity of nitrogen,
TC is the annualized total cost, and p is independent of q.

The solution is p = dTC/dq. The plot of p versus q will give the marginal cost curve for the firm.

The decision problem is to determine Z1, Z2 and S to maximize net revenue where:

Net Revenue =	Revenue from fertilizer sale	$PN[(1.85W_1+6.19W_2)Z1\cdot P+1.38\cdot Z2\cdot P+10.62\cdot S) \times \left(\frac{-19.22+5.57(C/N)-0.07(C/N)^2}{100}\right)$
less	Cost of night soil collection	$- 5.0\cdot Z1\cdot P$ (or $2.3\cdot Z1\cdot P$)
less	Cost of refuse collection	$- 2.2\cdot Z2\cdot P$
less	Cost of composting	$- 10\cdot Q$
less	Cost of rice straw	$- (22.8+189.8(S/P)^2)\cdot S$ or $(22.8+759.1(S/P)^2)\cdot S$
less	Cost of transportation to field	$- 3\cdot 0.33\cdot Q$

and where $$C/N = \frac{(19.8W1+6.5W2)Z1\cdot P + 66.0\cdot Z2\cdot P + 1011S}{(2.3W1 +7.7W2)Z1\cdot P + 1.72\cdot Z2\cdot P + 13.2S}$$

and $$Q = [(.0194W1 + 0.0230W2)\cdot Z1\cdot F + 0.1078\cdot Z2\cdot F + 0.95S$$

and the constraints are:

$$S \leq 0.35P \quad (\text{or } S \leq 0.175P)$$
$$0 \leq Z1 \leq 1$$
$$0 \leq Z2 \leq 1$$

In this analysis we will consider the night soil and refuse collection costs to be public health expenditures and not, therefore, expenses to be charged to the composting operation. In

accordance with the available data we will assume that all of the fecal matter and 40% of the urine are available from those whose excreta is collected (i.e. $W_1 = 1.0$ and $W_2 = 0.40$). Since it is common paractice to collect all of the refuse in a village at present, we will assume that all of the village refuse is available ($Z_2 = 1.0$). We will also assume that the panchayat (or other responsible body) will not establish the composting operation unless it yields a profit. (If a public authority is operating the plant, the "break-even point" should reflect the cost of alternative disposal systems. Data are available for the cost of uncontrolled dumping in Calcutta (CPHERI [1971]), but in this analysis we will assume that in this village there will be no cost associated with uncontrolled dumping. In the case of private ownership of the plant these alternative options need not be considered.)

The programming problem was solved using the Newton-Raphson Iterative Method under both rice straw availability assumptions. The optimal levels of the decision variables are the same for both individual and communal latrines. The total cost curves, and thus the zero profit points, are different in the two cases.

	Marginal Cost, p, in Rs/1b.N					
	1.0	1.3	1.5	2.0	3.0	4.0
Proportion of night soil used, Z1	1	1	1	1	1	1
Rice Straw, S in tons/year	0	28	58	108	171	214
Carbon/Nitrogen Ratio	12.4	15.6	18.7	23.2	27.9	30.7
Annual Production, q, in 1b.N	2231	3048	3812	4960	6155	6852
Annual Production, in tons	159	190	223	279	349	397
Total Costs, Rs/year	1749	2733	3812	5769	8593	11098

Table 3.6. Level and Cost of Optimal Composting Operation with 780 lbs. per capita per year Straw Availability

	Marginal Cost, p, in Rs/1b.N					
	1.0	1.3	1.5	2.0	3.0	4.0
Proportion night soil used, Z1	1	1	1	1	1	1
Rice Straw, S, in tons/year	0	23	42	72	109	135
C/N Ratio	12.4	15.1	17.1	20.0	23.3	25.3
Annual Production, q, in 1b.N	2231	2895	3411	4160	4983	5499
Annual Production, in tons	159	184	205	239	280	309

Table 3.7. Level and Cost of Optimal Composting Operation with 390 lbs. per capita per year Straw Availability

The cost of refuse collection in all cases is Rs 2200 per annum; if individual latrines are used the cost of night soil collection is Rs 5000; if communal latrines are used the cost is Rs 2300 per year.

We note the following:

i) The maximum rice straw use is 479 lbs/cap/year in the first case and 302 lbs/cap/year in the second.

ii) The average nitrogen composition of the compost (1.32% of dry weight) is in the range (1.2% to 1.5%) given by Acharya (1951) for town compost in India.

iii) The value of nitrogen has to rise to about Rs 1.25 per pound before it becomes profitable to start adding rice straw.

iv) In no case is the carbon-to-nitrogen ratio below 10, which is the lower limit of applicability of the nitrogen retention function.

These data, as plotted on Figures 3.10 and 3.11, will be used in conjunction with the marginal revenue curves to be derived in the next section to determine the optimal level of the village composting operation.

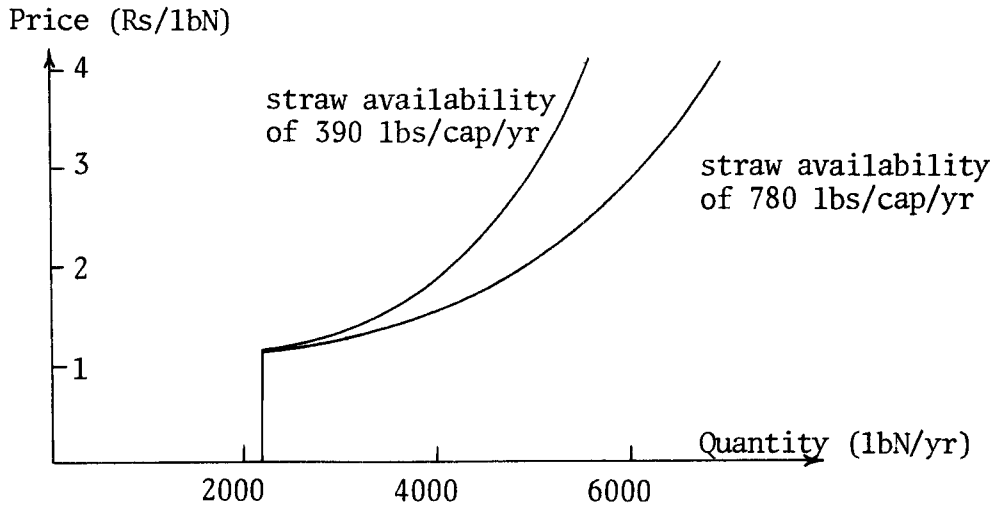


Figure 3.10: Marginal Cost of Composting Curves

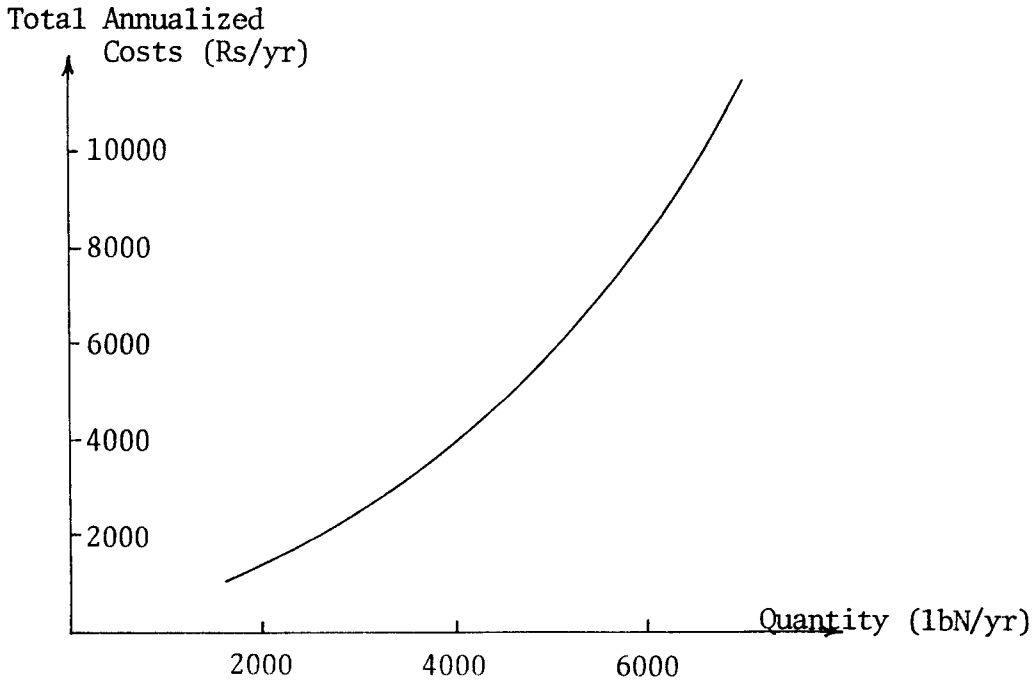


Figure 3.11: Total Cost Curve (for 780 lbs/cap/yr straw availability)

3.3.3. Marginal Revenue Relationships

To understand the potential role of composting in a village it is necessary to know both the costs involved in producing the compost and the agricultural demand for the product. In this section we will attempt to derive a realistic compost demand curve for a village of one thousand inhabitants in the Singur area of West Bengal. We will also try to determine the effect on this demand of a shift to high yielding crop varieties.

The mechanisms relating crop response to organic fertilizer dose are complex and only partially understood. Organic manures introduce a variety of plant nutrients into the soil, improve the structure, water holding capacity and base exchange capacity of the soil, and improve the microbial population and its activity (Agarwal [1967], Garg [1971]). In interpreting the results of the analysis which follows, the distinction between the simple model and the complex reality should be borne in mind.

Although some quantitative information is available on the relationship between crop yields and organic fertilizer doses, an adequately specified response function was not found in the literature. Those data which are available (Agarwal [1967], Garg [1971], Acharya [1951]) give the ratio of increased grain yield to increased nitrogen input in organic manures. The use of the nitrogen content of the manure as a measure of fertilizer value emanates from the fact that nitrogen is the principal deficiency in all Indian soils (Agarwal [1967]). Adoption of this measure facilitates use of a much broader base of data on

crop responses to nitrogen and simplifies the link between the compost "supply" and "demand" models.

In this analysis we will use data on the response of crops to nitrogen supplied in the form of chemical fertilizers to derive the demand curve for nitrogen in organic manures under different agricultural regimes. The response ratios emerging from this analysis will be compared with the available data on crop response to organic manures.

While this approach seems to be the least unattractive given the sparcity of suitable data, the underlying justification is weak. The rationale for using total nitrogen as a measure is based on the notion that the effect of low availability of nitrogen in organic fertilizers (Raheja [1967] estimates this to be between 25 and 40%, while Jalal [1968] assumes that 85% is available) will be "balanced" by not taking into account the other beneficial effects of organic manures.

3.3.3.1. Total nitrogen demand

The village has 1000 inhabitants and only 156 acres of cultivated land. In West Bengal as a whole the per capita cultivated area is 0.39 acres per capita (Government of India [1970a]): this is indeed "one of the most densely populated rural areas in the world"(Johns Hopkins Center for Medical Research and Training [1970]). Virtually all of the cultivated area is planted in aman paddy (which is harvested in the winter).

3.3.3.1.1. Before Introduction of High Yielding Varieties

Abraham (1965) has collected and compiled the results of a series of fertilizer tests carried out by the Government of India in the mid-1950's. The cultivator's fields for the trials were selected by a multi-stage random sampling procedure and the fertilizer treatments superimposed on the normal practices of the farmer.

> The responses obtained in a series of trials with a fertilizer were first averaged over all the trials conducted in a year in a district. Where more than one series of experiments was being conducted in a district, the averages over the different series were taken by weighting by the number of trials in each series. A simple average of the responses in districts falling in each zone was taken to get the average response in the zone. A quadratic response curve, $y = a + bz - cz^2$, where y is the yield corresponding to a dose of z units of nutrient applied, was fitted to the mean responses in a zone.

In the northeastern region, comprising Assam, West Bengal, South Bihar and Orissa,nearly 2000 experiments were conducted. The fitted relationship is $y = 0.16153x - .00197x^2$ where the units of y and x are maunds (one maund = 82 lbs.) per acre and pounds nitrogen per acre, respectively. (Abraham's data show these to be the lowest incremental yields in India.)

We can compare the implicit response ratios with these pertaining to organic manures. Garg (1971), using Indian Council on Agricultural Research data, indicates that an application of 12.6 tonnes per hectare of farm yard manure results in an increase

of 168 kg/ha in rice yield in West Bengal. The average nitrogen content of the farmyard manure is assumed to be 0.5% (wet weight basis) and the implied response ratio is thus 3.1:1. On the basis of the function used in this analysis we would expect a response ratio of 4.1:1 at a nitrogen application of 56.25 lb.N/acre. Agarwal (1967) cites a series of experiments on paddy in West Bengal and shows that the efficiency of nitrogen in the two sources (ammonium sulphate and town compost) was virtually identical. It would appear, then, that the response function used in this analysis gives results which are consistent with the available organic fertilizer data.

The optimal nitrogen dose (x^*)

$x^* = (bp_1 - p_2)/2cp_1$ where p_1 is the price of one unit of produce, p_2 is the price of one unit of nutrient and $y = bx - cx^2$ is the response function. At the 1973/74 procurement price of Rs 70 per quintal of rice (IBRD [1974] we find:

	Price of Nitrogen (Rs/lb. N)				
	0.5	1.0	2.0	3.0	4.0
Demand for Nitrogen (lb.N/acre)	36.1	31.3	21.6	11.9	2.2

Table 3.8. Optimal Nitrogen Doses for Traditional Rice Varieties

3.3.3.1.2. With High Yielding Varieties

Singh et al.(1970) used the data on the response of high yielding varieties of rice to graded doses of nitrogen which were collected under the All-India Coordinated Agronomic Experiments Scheme during 1967-68 and 1968-69. In the Indo-Gangetic region 159 fields were involved in the rice experiments. Fields were selected by the multi-stage stratified random sampling method. Each selected field was subdivided into 3 plots. Nitrogen applications were then: zero on one plot; 60 kgN/ha on one and 120 kgN/ha on the remaining plot. The study was confined to nitrogen since "it is the most commonly used fertilizer for foodgrains"(Singh et al. [1970]).

The response results are reported in Table 3.9 in the form of cumulative frequency tables, in which the percentage of fields which had a response of less than or equal to x kg/ha were plotted against x for the two different fertilizer applications. These results are plotted on Figure 3.12 and the effect of a dose of 120 kgN/ha obtained by adding the effects of doses 1 and 2 (of 60 kgN/ha each)horizontally on the cumulative frequency diagram. (The assumption implicit in this, namely that the percentiles on the first dose cumulative frequency diagram represent the same percentiles on the second dose cumulative frequency diagram, is unlikely to be precisely valid but would certainly seem to be a reasonable approximation.)

Response (kg/ha)	Indo-Gangetic region			
	First Dose*		Second Dose	
	No. of fields	Percent	No. of fields	Percent
less than 50	2	1	9	6
50-100	4	2	18	11
100-200	11	7	27	17
200-300	24	15	49	31
300-400	39	25	58	37
400-500	53	33	76	48
500-600	74	47	95	60
600-700	95	60	114	72
700-800	109	69	123	77
800-900	122	77	130	82
900-1,000	130	82	138	87
1,000-1,100	134	84	142	89
1,100-1,200	140	88	150	94
1,200-1,300	143	90	152	96
1,300-1,400	146	92	154	97
1,400-1,500	147	93	156	98
1,500-1,600	150	94	157	98
1,600-1,700	153	96	158	99
1,700-1,800	153	96	158	99
1,800-1,900	156	98	158	99
1,900-2,000	156	98	158	99
2,000-2,100	157	98	158	99
2,100-2,200	158	99	159	99
2,200-2,300	158	99	159	100
2,300-2,400	158	99	159	100
2,400-3,000	158	99	159	100
over 3,000	159	100	159	100

*consists of 60 kg/ha of nitrogen

Table 3.9. Cumulative Frequency Distribution of Fields According to Response of Rice to Nitrogen (from Singh [1970])

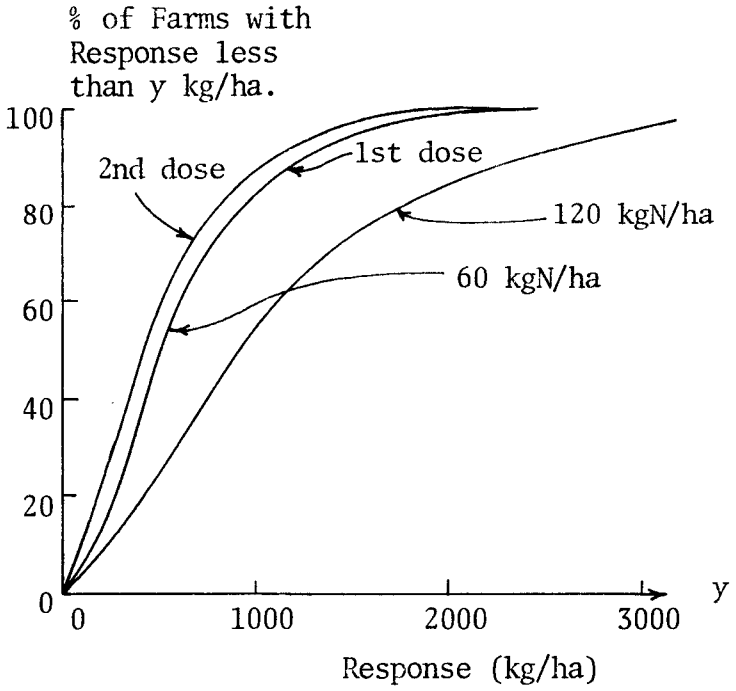


Figure 3.12: Cumulative Frequency Distribution of Fields according to Response to Nitrogen (for high yielding varieties)

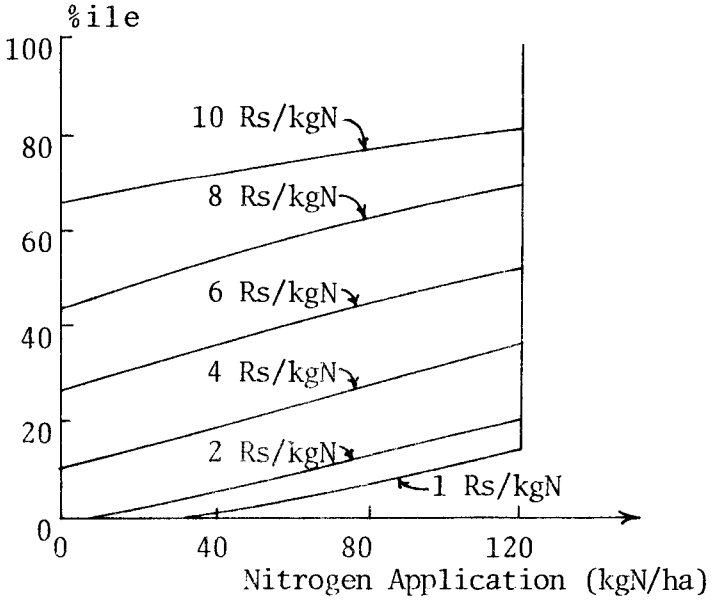


Figure 3.13: Distribution Function of Optimum Nitrogen Dose for High Yielding Rice at Different Fertilizer Prices

	Yield (kg.ha)		Response Parameters		Interpolated Yields			Optimal Nitrogen Doses for Nitrogen Price (Rs/kgN) of:					
%ile	y_{60}	y_{120}	b	c	y_{10}	y_{20}	y_{90}	1	2	4	6	8	10
95	1650	2900	30.84	.0556	302.84	594.6	2325.2	120	120	120	120	120	120
90	1260	2400	22.00	.0167	218.3	433.3	1844.7	120	120	120	120	120	120
80	960	1800	17.00	.0167	168.3	333.3	1394.7	120	120	120	120	120	110
60	700	1300	12.50	.0139	123.6	244.4	1012.4	120	120	120	120	66	0
40	550	970	10.26	.0181	100.8	198.0	978.4	120	120	120	62	0	0
20	326	590	7.09	.0181	69.1	134.6	491.6	120	120	48	0	0	0
10	250	350	5.41	.0208	52.0	99.9	318.4	98	66	1.8	0	0	0

Table 3.10. Response of High Yielding Varieties of Rice to Nitrogen and Optimal Nitrogen Applications

For the analysis which follows we require the cumulative frequency diagrams for all nitrogen doses (less than 120 kgN/ha). We interpolate for nitrogen doses other than 60 or 120 kgN/ha by assuming (see Herdt [1964] and Abraham [1968]) a quadratic response curve $y = a + bz - cz^2$, where is the yield corresponding to a dose of z units of nutrient. For any chosen percentile the parameters b and c are derived and the cumulative frequency distribution for any chosen nutrient application level determined. These interpolated results and the corresponding optimal nitrogen applications (determined as in the analysis of traditional varieties and with a maximum dose of 120 hgN/ha) are listed on Table 3.10 and the latter are plotted on Figure 3.13. The average optimal nitrogen doses are estimated graphically from Figure 3.13 and presented on Table 3.11.

	% with optimal application of (kgN/ha)				Converting:	
Rs/kgN	120	60	0	Avg kgN/ha	Rs/1bN	optimal 1bN/acre
10	19%	15%	66%	32	4.54 (4.00)	28.6 (38)
8	30	27	43	52	3.63 (3.00)	46.4 (60)
6	48	26	26	73	2.72 (2.00)	65.2 (78)
4	64	26	10	92	1.81 (1.00)	82.1 (94)
2	80	20	0	108	0.91 (0.50)	96.4 (100)
1	86	14	0	112	0.45	100.0

Table 3.11. Average Optimal Nitrogen Doses for High Yielding Varieties

3.3.3.2. Use of Animal Manure

A complete micro-economic analysis of organic manures in a village would incorporate "supply" models of both village compost and animal manure. In this analysis we are assuming monopolistic behaviour by the composter. The assumptions underlying the perfect competition model (homogeneous product, perfect knowledge, utility and profit maximization, atomistic competition, free entry and exit of resources) would appear to be reasonably approximated in the animal manure situation. In particular, the requirement of atomistic competition, namely that no buyer nor seller possesses sufficient market power to affect price, appears to be met. As far as the monopolistic composter is concerned, then, the demand which is of interest to him is not the total demand, but the demand which remains after the animal manure supply is subtracted from total demand.

Earlier in this chapter various estimates of the quantity and composition of cattle dung and its use as a fertilizer were examined. The use of dung as a manure was estimated to account for 1668 pounds of nitrogen annually in the village (see section 3.2.1.).

An important parameter in this analysis is the price elasticity of supply of animal manure for fertilizer purposes. In this analysis we will make the simplest possible assumption, namely that the supply of dung for fertilizer use is perfectly inelastic. This assumption would almost certainly be modified were any data available.

3.3.3.3. Marginal Revenue Curve

By 1973-74 the percentage of the total area under rice which was under high yielding varieties varied from 90.1% in the Punjab to 10.3% in Bihar, with the All India average being 26%. In West Bengal the percentage was 17.4% (United States Department of Agriculture [1975]). We use the data of Tables 3.8 and 3.11 to estimate the demand for nitrogen, net of the supply through animal manure, for the West Bengal village of one thousand inhabitants for different assumptions concerning the share of land under high yielding varieties.

% under High Yielding Varieties	Price of Nitrogen (Rs/lbN) 0.5	1	2	3	4
0	3964	3215	1702	188	0
20	5957	5171	3462	1689	0
40	7951	7128	5221	3190	909
60	9945	9083	6981	4690	2026
80	11938	11040	8740	6191	3143
100	13932	12996	10518	7692	4260

Table 3.12. Demand for Nitrogen in LbN/year (Net of Supply from Animal Manure) in the Hypothetical West Bengal Village

To determine the marginal revenue curve from the average revenue (or demand) curve we make use of the following relationships:

$TR = pq$ where $AR = TR/q = p$, and $MR = dTR/dq = p+q \cdot dp/dq$.

(The slope dp/dq can be measured graphically on the AR curve.) It can be easily shown that, for a straight line AR curve which intersects the ordinate at k and has a slope of -b, the MR curve

also intersects the ordinate at k and has a slope of -2b. Following Baumol (1971) we are able to sketch the MR curve from the AR curve by the following construct:

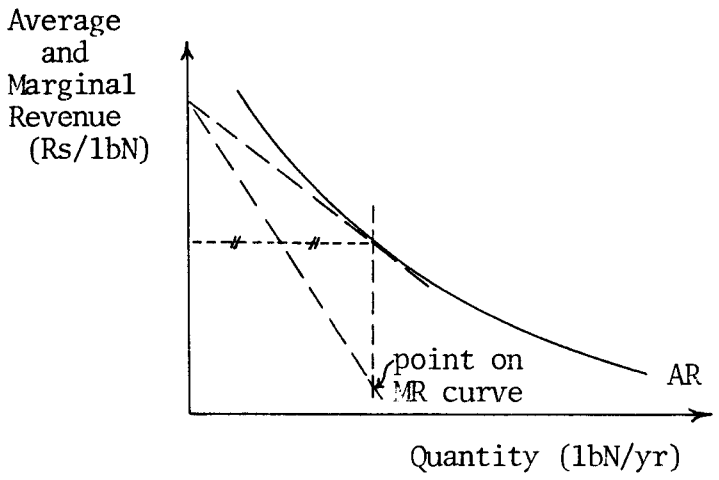


Figure 3.14. Graphical Determination of Marginal Revenue from Average Revenue

This process is followed (see Figure 3.15) for deriving the marginal revenue curve of relevance to the compost supplier under different assumptions concerning the share planted to high yielding varieties.

3.3.4. Economic Viability of Composting

The level at which the composter will choose to operate the composting plant is determined by equating the marginal cost and marginal revenue curves. The price at which this output will be offered is determined by the average revenue curve. The marginal cost curves for rice straw availability assumptions are plotted against the marginal revenue curves on Figure 3.15. The composter's profit is determined by subtracting the total cost associated with the optimal level of production (see Figure 3.11) from the total revenue (price x quantity of nitrogen produced). The optimum quantity of nitrogen produced in the compost, the price which the monopolist would charge for this compost and the profit made of such an operation are plotted against the percentage under high yielding varieties on Figure 3.16. We note that the decisions concerning the profitability of a composting operation, the level of the operation and the price of the compost are not highly sensitive to the assumption concerning the availability of rice straw. The viability of such an operation is highly dependent on the assumption concerning the inclusion or exclusion of the night soil and refuse collection costs: If these collection costs are regarded as necessary public health expenditures,the composting operation is a viable economic proposition for all crop mixes; if those costs are to be borne by the composting authority, it is necessary to have nearly 50%, for the case of collection from private latrines, or 25%, when we

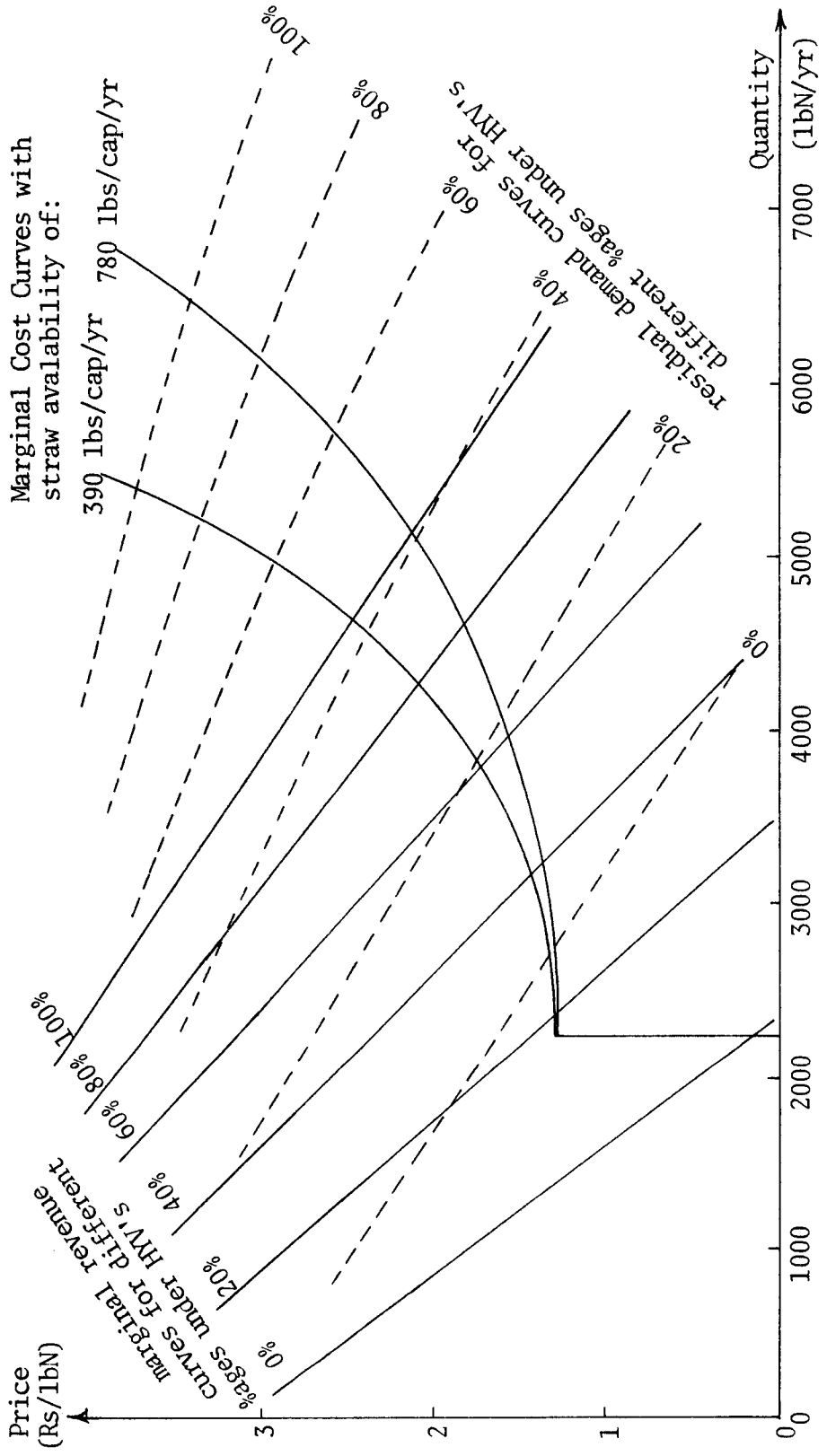


Figure 3.15: Marginal Cost, Marginal Revenue and Average Revenue Curves under Different Agricultural Regimes

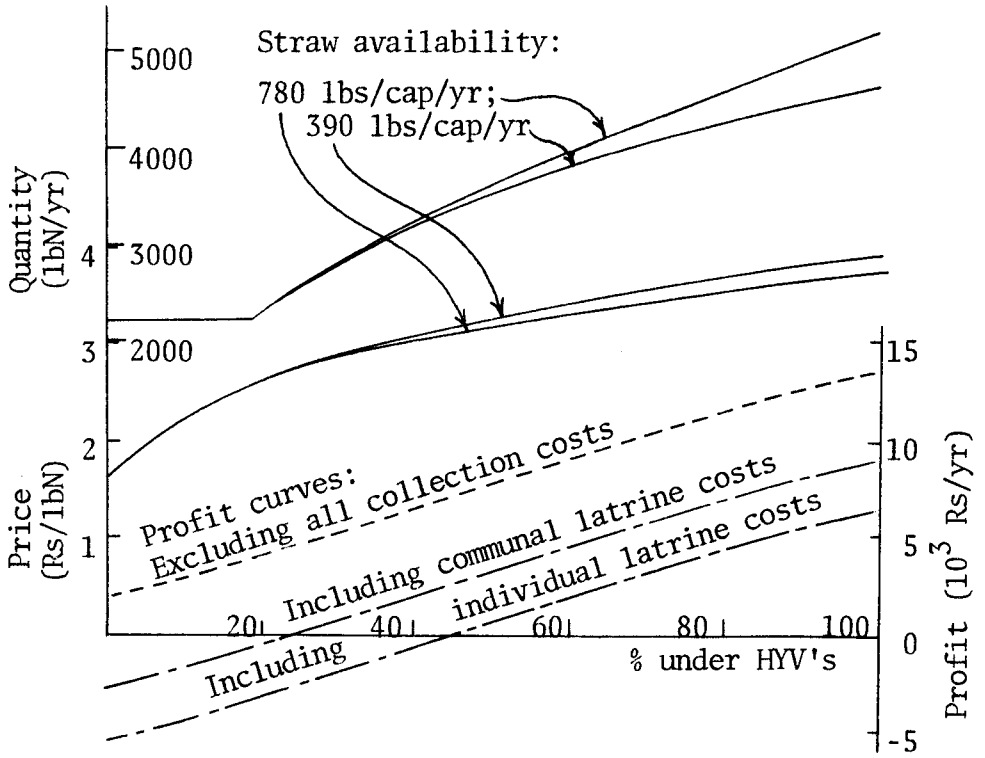


Figure 3.16: Quantity and Price of Compost and Composter's Profit for Different Agricultural Regimes

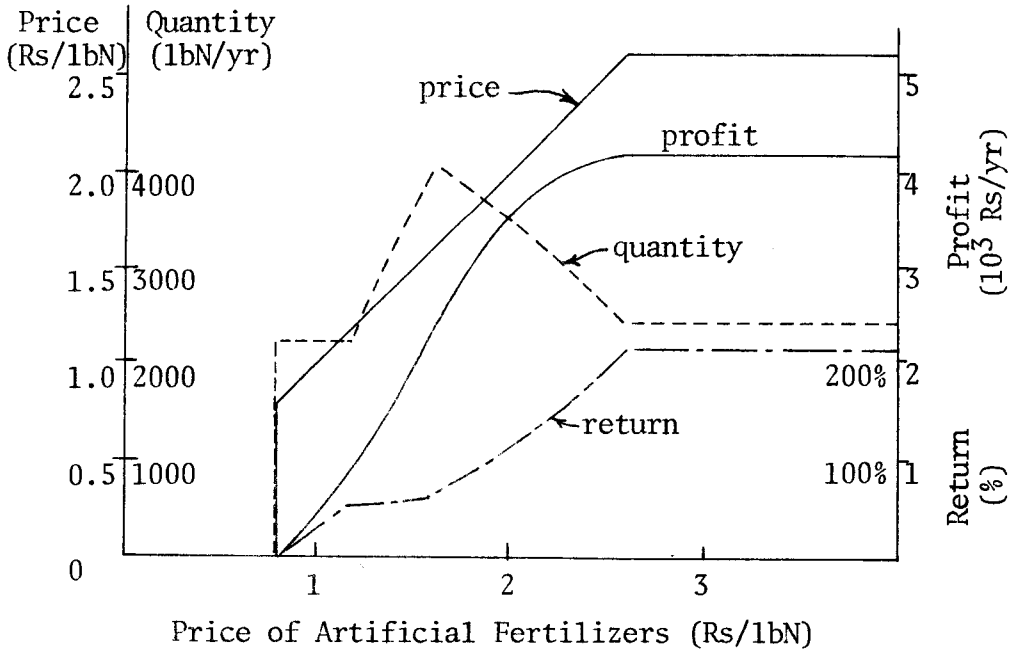


Figure 3.17: The Effect of Artificial Fertilizers on Composting

have communal latrines, of the area under high yielding varities before a profit could be envisioned. From Figure 3.16 we note that the monopolist price for compost is quite high, varying from Rs 1.7 to 3.9 per lbN.

The above results have been derived for the case in which only animal manure and compost are available as nitrogen sources. The method by which the availability of artificial fertilizers is taken into account was outlined in Section 3.3.1. For the case of composting in a village of 1000 people in West Bengal, we assume that the supply of artificial fertilizers is unlimited, that 20% of the area is under high yielding varieties and that 780 lbs of rice straw per capita are available annually. The relationships between the artificial fertilizer price which has been set, and the price and quantity of compost which the monopolist would produce, are derived (using the methods outlined in Section 3.3.1) from Figure 3.15 and presented on Table 3.13 and Figure 3.17. The relationship between profitability of the composting operation and the artificial fertilizer price, too, is indicated.

Under the present conditions (about 20% under high yielding varieties and artificial fertilizer price of Rs 2 per lbN) about 3000 lbN (or 3 lbN per capita) are produced in the village annually. If this per capita figure is applied on an all-India basis, it amounts to about 4 lbN/acre/year which is consistent with the estimate of "realizable potential" of

3.5 lbN per acre per year (human plus crop wastes) given in Section 3.1.3. Since animal manure use is taken to be constant the animal manure figure used in this analysis is consistent with that under "present conditions" given in Section 3.1.2.

From Figure 3.17 we see that if no payments have to be made to induce villagers to use the facility, and if the costs of the latrine construction and refuse collection are fully subsidized, this investment would be very attractive to an entrepreneur, yielding a profit of about Rs 3600 and a return of 108% at the 1974 fertilizer price of Rs 2/lbN. If the facility is owned by the community, we see that the per capita profit of the operation is quite low, being Rs 3.6 per capita per year. Alternatively, this figure could be considered the per capita incentive payment which could be made to villagers,at the zero-profit point, if the facility were owned by an entrepreneur.

Conclusions

The model which has been developed in this section is intended to illustrate a more sophisticated approach than was taken in Appendix 2.1, for instance, to the assessment of different excreta disposal programs. The specific advances in this analysis include an optimization approach to the choice of the decision variables and the embedding of the analysis in a "general" rather than a "partial" (in which, for instance, the demand for organic fertilizers is considered infinitely elastic) equilibrium framework.

CHAPTER FOUR

EPIDEMIOLOGICAL MODELS FOR HOOKWORM

Summary

This chapter represents the first published attempt at building a mathematical epidemiological model of a soil-transmitted helminthic disease. The first section of this chapter is an investigation into the nature of equilibrating mechanisms in mathematical models of a number of different diseases. Analytic models which are useful in interpreting the results of the complex simulation models are developed. A major finding of this section is the necessity for including non-linearities (such as immunity or density dependent effects) in epidemiological models of soil-transmitted helminths. It is suggested that the exclusion of these factors in the other models examined constitutes a major shortcoming of these models. The bulk of this chapter is devoted to the development of a set of deterministic analytic and simulation models for hookworm infestation in rural Bengal. The models are used to assess the effects of uncertainty in the parameter estimates and to quantify the response to changes in the sanitation regime. The effect of stochastic variations in weather on the operation of the model is examined.

CONTENTS

4.1: INTRODUCTION

Mathematical formalization in a field which has been dominated by less formal models serves several purposes. Quantitative models provide a useful framework in which to organize ideas on matters of considerable complexity and in which to investigate the epidemiological implications of research hypotheses (such as the hypothesis concerning delayed maturation of *Onchocerca* larvae in the human host).

As didactic tools, the models developed in this paper elucidate a number of basic and not necessarily obvious epidemiological differences between a diverse assortment of diseases. The models also serve to provide a logical approach for organizing the data relevant to understanding the relationship between different control decisions and the health implications. In the process we come to understand which data deficiencies are crucial and are thus able to make rational decisions on further data-gathering activities. As part of the above functions the models make an important contribution in forcing the analyst to be explicit about assumptions which may otherwise not be apparent to either the analyst or the person who calls upon his or her expertise. Given that the health implications of environmental changes is an area in which a wide range of assertions is made, this may be a particularly useful characteristic of the technique used.

These models contribute to an analysis of rural sanitation by: i) Specifying the expected form of the relationships between various disease measures and independent variables which may affect these measures, thus ensuring that statistical (e.g. multiple regression) models reflect the etiological realities of the different diseases; and, ii) providing a mechanism for mapping sanitation changes into incidence and prevalence changes, thus facilitating the translation of the effects of environmental programs into demographic and economic terms.

4.2. EQUILIBRATING MECHANISMS IN MATHEMATICAL EPIDEMIOLOGICAL MODELS

In this section I will discuss the mechanisms by which several other mathematical models ensure that given values of the exogenous flow variables lead to equilibrium values of the state variables. This discussion will elucidate several important issues pertaining to the models examined, in particular, and to epidemiological models in general. In the context of a hookworm model this discussion is germane to an understanding of the uniqueness of the equilibrating mechanism in infestation with soil transmitted helminths. (It should be noted that there are other, equally significant differences between the present model and those which will be discussed here. An examination of some of these other differences is undertaken elsewhere in this chapter.)

The epidemiological models analysed are:

(i) Briscoe's (1974) model of gastro-intestinal diseases in which superinfection does not take place;

(ii) Cvjetanovic's (1971) model of typhoid fever;

(iii) Uemera's (1971) model of cholera;

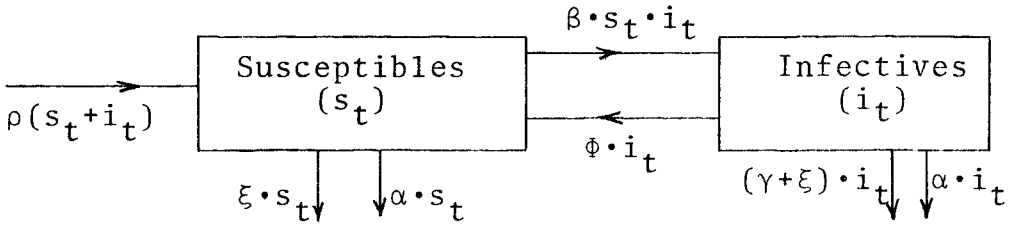
(iv) Cvjetanovic's (1972) model of tetanus;

(v) McDonald's (1965) model of schistosomiasis.

I) Models of diseases with no superinfection, no explicit inclusion of immunity and force of infection determined by

human environmental contamination:

The flow diagrams and the emerging set of difference (or differential) equations for the first three models are given on Figures 4.1, 4.2, and 4.3, overleaf. While these models vary considerably in complexity, the underlying structure and the nature of the mechanism which limits the spread of the disease under natural conditions is common to all three models.



The difference equations emerging from this model are:

$$s_{t+\delta t} = s_t + \rho(s_t+i_t) - \xi \cdot s_t - \beta s_t i_t + \Phi i_t - \alpha \cdot s_t$$

and

$$i_{t+\delta t} = i_t + \beta s_t i_t - (\gamma+\xi) i_t - \Phi \cdot i_t - \alpha \cdot i_t$$

Figure 4.1.: Briscoe's Model for a Fecal-borne Infectious Disease in which Superinfection is not Possible

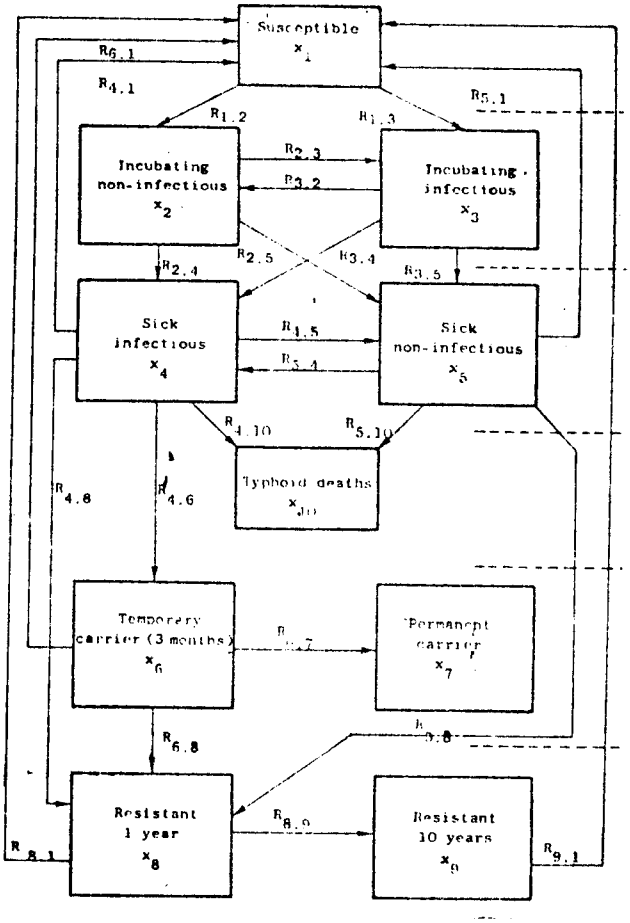


Figure 4.2: Cvjetanovic's Model for Typhoid Fever

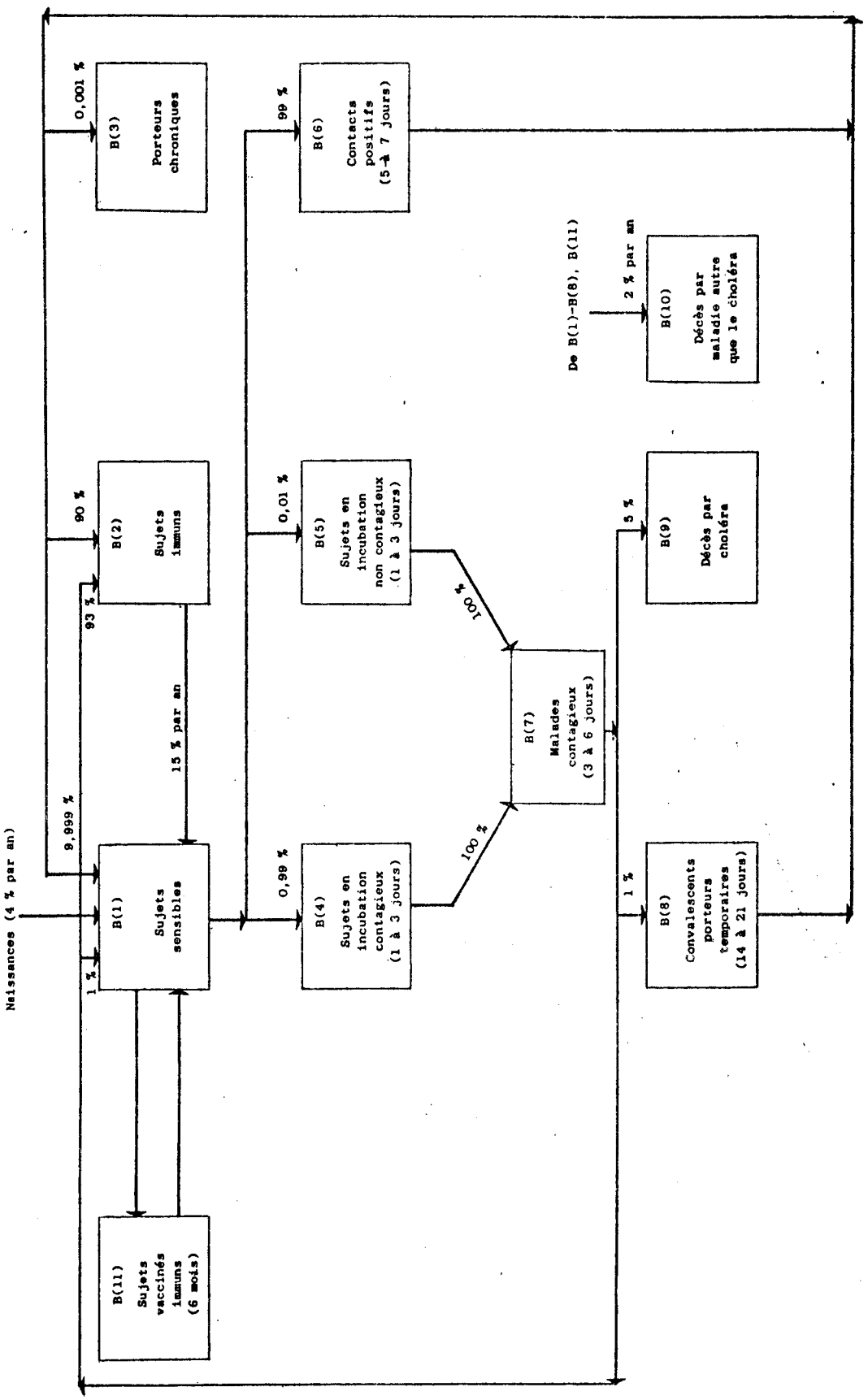


Figure 4.3: Uemera's Model for Cholera

We use the following simple, didactic model to elucidate the nature of the equilibrating mechanism.

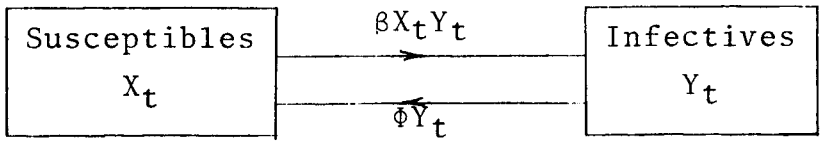


Figure 4.4.: Simplified Model of Disease Type I

We note a few assumptions incorporated in the three models and in the above diagram:

i) The infectivity of the environment is related linearly to the number of infective human hosts. [In Cvjetanovic's model the incubation differential is linearly related to the product of the number of susceptibles (X_1) and the number of "dischargers" ($X_3+X_4+X_6+X_7$).] Therefore, the number of new infectives in each time period is related to the product of the number of susceptibles and infectives.

ii) Natural immunity to infection is not explicitly taken into account.

iii) Superinfection, the simultaneous presence of multiple infections in the host, is not permitted.

The models referred to were analysed using simulation techniques. Here we will examine the above, greatly simplified, model analytically.

If we consider X_t to be the proportion of the population

which is susceptible and define Φ, the rate of recovery parameter, and β, the infectivity parameter, accordingly, the differential equation describing the system is (see Appendix 4.1 for details):

$$\frac{dX_t}{dt} = \Phi - (\Phi+\beta)\ X_t + \beta X_t^2$$

which gives equilibrium values of X_t of $\frac{\Phi}{\beta}$ and 1.

The meaning of the second equilibrium value is clear: If there are no people infected, the force of infection is zero and the disease is eradicated (in the closed system).

The approach to equilibrium over time is given in Appendix 4.2.

In two of the above models (Briscoe's fecal-borne disease model and Cvjetanovic's typhoid model) the proportions of the population free from disease are given for a range of values of the "infectivity parameter." On Figure 4.5 over-leaf, the proportion free from fecal-borne disease (from Briscoe's data) is plotted against a linear function of $1/\beta$, while on Figure 4.6 Cvjetanovic's data are plotted in a similar fashion. The results are striking: despite the considerable elaborations of the basic structure which these models (particularly the latter) represent, the straight line relationship is precise! As an aside, I believe that the above gives an analytic justification for the empirical observations made by Cvjetanovic in the following paragraph from his typhoid paper:

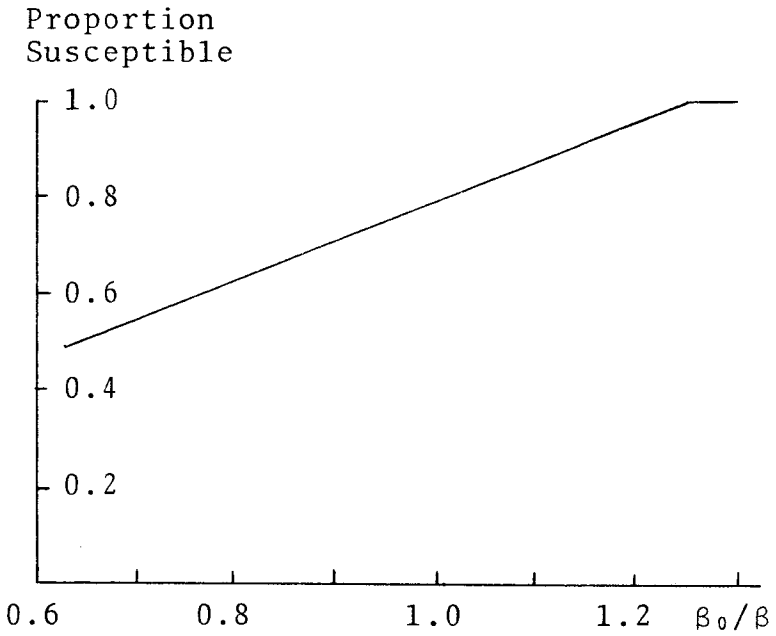


Figure 4.5: Effect of β on Prevalence in Briscoe's "Fecal-borne Disease" Model

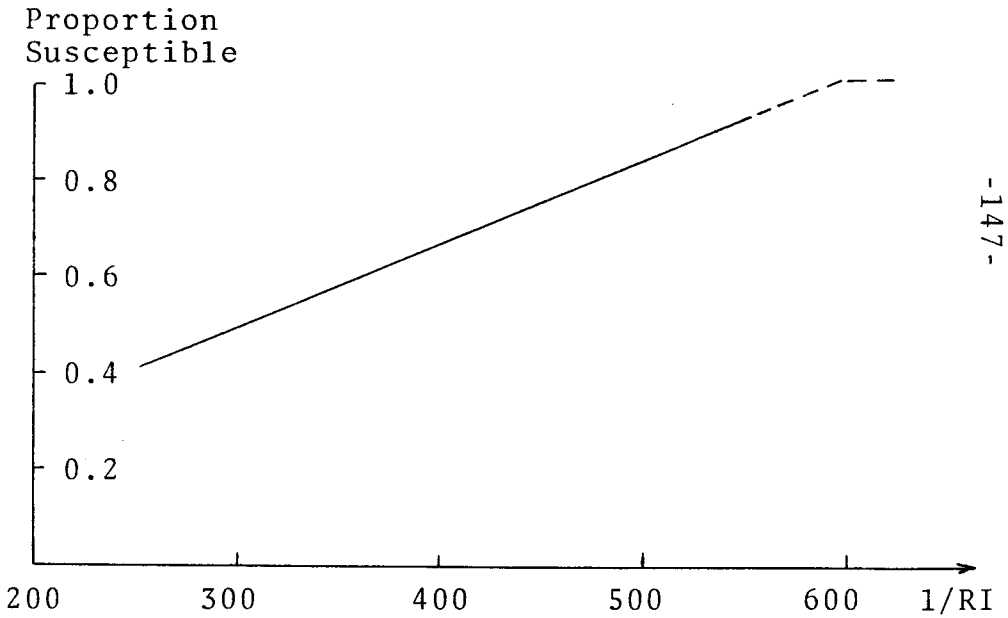


Figure 4.6: Effect of β in Cvjetanovic's Typhoid Model

> It was found that the size of the epidemiological classes was almost linearly related to the reciprocal of the force of infection RI.... It is thought that a stable level of endemicity can establish itself only if the rate RI remains above a certain critical value and that this value is a function of the birth and death rates. Further study in this direction might be fruitful.

This relationship has considerable utility in analysing model changes under different environmental conditions: from a given prevalence rate we are able to infer the implied value of the "force of infection"; we can determine (this will usually be done graphically) the value of β at which the disease will be entirely eradicated. (It sould be noted that β need <u>not</u> be reduced to zero for eradication of the disease in this model.)

While this result is apparently in accordance with epidemiological experience with this category of diseases (Cvjetanovic, personal communication), a great deal of caution should be exercised in drawing inferences concerning eradication of these diseases under field conditions. In this regard the approach of the system to equilibrium is of interest.

The difference equation (see Appendix 4.3) may be written as:

$$\frac{\Delta X_t}{\Delta t} = \beta[(X_\infty - X_t)(1-X_t)].$$

When the equilibrium value ($X_\infty = \frac{\Phi}{\beta}$) is small, $(1-X_t)$ will be approximately constant as X_t approaches X_∞ and the restoring force will be approximately linearly related to $(X_\infty - X_t)$.

When the equilibrium value is large (X_∞ is slightly less than

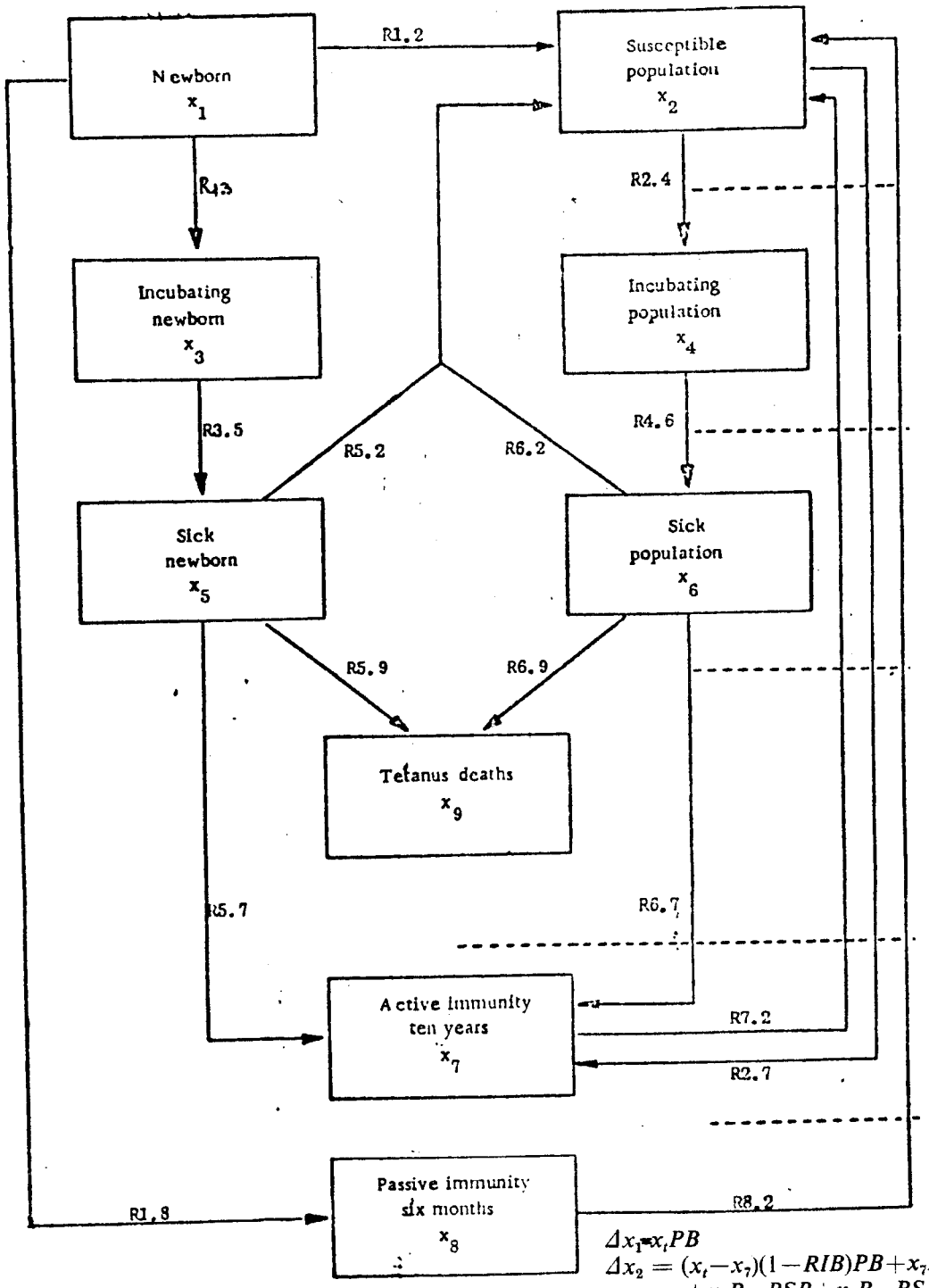
or equal to unity), the restoring force becomes extremely weak as equilibrium is approached. This has important stability implications when considering eradication of the disease in a biological system subject to incessant perturbations. If the mean value of β is less than Φ , the deterministic model indicated eradication of the disease. If β is stochastic, however, we would have : i) a slow approach to the state $X_t = 1$ for those $\beta < \Phi$; and ii) a rapid approach to $X_t = \frac{\Phi}{\beta}$ for those $\beta > \Phi$. Thus when $\overline{\beta} < \Phi$ but β is considered to be a stochastic rather than a deterministic variable, eradication may never be attained.

To reiterate the major finding germane to an analysis of the equilibrative mechanisms in epidemiological models: We have found that the complementary nature of the size of the infective and susceptible populations, and the relationship between these population sizes and the rate of infection,ensure the establishment of an equilibrium in the case of a disease in which superinfection does not occur and in the absence of any explicit invocation of an immune response or other density dependent factors.

II) Models of diseases with no superinfection, no explicit inclusion of immunity, and force of infection not primarily determined by human environmental contamination:

Epidemiological models of cholera and typhoid, developed by Cvjetanovic and his colleagues at WHO, have been discussed above. In this section, we wish to discuss a model which this group has developed for tetanus (Cvjetanovic [1972]). The essential difference between this disease and the communicable diseases in (I) above is that tetanus is a zoonotic disease in which the primary host is the horse (American Public Health Association [1960], Marshall [1972]). "The bacteria, which are anaerobic, live in the intestinal tract of horses and men and reach the soil in fecal matter. Once in soil, tetanus spores endure for long periods. Man becomes infected when spores gain entry through a puncture wound or an animal bite"(Marshall[1972]). The flow diagram and the emerging set of difference equations for Cvjetanovic's model are presented on Figure 4.7. In particular we note that the force of infection is independent of the sizes of the human epidemiological classes. This independence is a function of the dominant role of the equine host.

As was done for the previous set of models, we will analyse the simplest possible manifestation of this class of models to elucidate the nature of the equilibrating mechanism.



$$\Delta x_1 = x_t PB$$
$$\Delta x_2 = (x_t - x_7)(1 - RIB)PB + x_7 PR1 + x_8 PR2 + + x_5 R_{5\cdot 2} PSB + x_6 R_{6\cdot 2} PSA - x_2(RIA + + PD - \Delta x_9/x_t)$$
$$\Delta x_3 = (x_t - x_7)RIB.PB - x_3(PIB + PD - \Delta x_9/x_t)$$
$$\Delta x_4 = x_2 RIA - x_4(PIA + PD - \Delta x_9/x_t)$$
$$\Delta x_5 = x_3 PIB - x_5(PSB + PD - \Delta x_9/x_t)$$
$$\Delta x_6 = x_4 PIA - x_6(PSA + PD - \Delta x_9/x_t)$$
$$\Delta x_7 = x_5 R_{5\cdot 7} PSB + x_6 R_{6\cdot 7} PSA - x_7(PR1 + PD - - \Delta x_9/x_t)$$
$$\Delta x_8 = x_7 PB - x_8(PR2 + PD - \Delta x_9/x_t)$$
$$\Delta x_9 = x_5 R_{5\cdot 9} PSB + x_6 R_{6\cdot 9} PSA$$
where $x_t = x_2 + x_3 + \ldots\ldots + x_8$

Figure 4.7: Cvjetanovic's Model for Tetanus

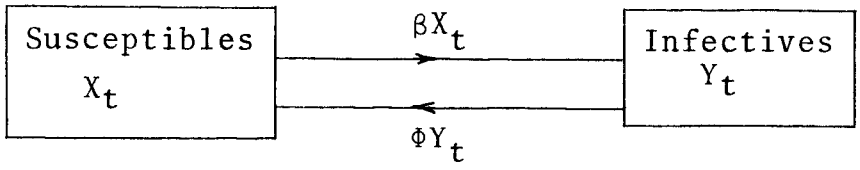


Figure 4.8.: Simplified Model of Disease Type II

The differential equation is:

$$\frac{dX_t}{dt} = \Phi - X_t(\Phi+\beta)$$

whence

$$X_\infty = \frac{\Phi}{\Phi+\beta}$$

(The approach to the equilibrium from a given starting point may be of interest in determining the optimal intervals at which mass vaccination campaigns may be conducted. Since these equations are not relevant to this discussion of equilibrating mechanisms, they are relegated to Appendix 4.4.)

The nature of the equilibrium condition is quite different from that of the conditions pertaining to typhoid and cholera. The differential equation is linear in X_t and we therefore have only one equilibrium point (which is never an end point).

It is important to note that, despite the linearity of the model, equilibrium is established without the acquisition of acquired immunity. [The relevance of this observation will be clear later when we see that where we have a linear model with superinfection the rule, there is no equilibrium in the absence of acquired immunity.]

There are interesting distinctions between the results for the typhoid and cholera models on the one hand, and the tetanus model on the other.

There is a critical minimum value, below which we have eradication of the diseases, in the cholera and typhoid models. In the case of tetanus there is no such critical value and there will be a positive stable level of endemicity for all positive β values.

With respect to the approach to equilibrium, the case of tetanus is less complicated than that of cholera. In this case the difference equation can be written as:

$$\frac{\Delta X_t}{\Delta t} = (\Phi+\beta)[X_\infty - X_t]$$

The restoring force is linearly related to $(X_\infty - X_t)$ and the disease prevalence is likely to be stable in the vincinity of the equilibrium point.

III) Models of diseases with superinfection, no explicit inclusion of immunity, and force of infection not determined by environmental contamination.

In this section I will examine the form of the mathematical epidemiological models developed for malaria (an anthropod-transmitted protozoal infection) and schistosomiasis (a zoonotic helminthic infection). The selection of these particular models from a large set of such models (Cohen, 1973a, has referenced

many of the schistosomiasis models) is arbitrary - these are merely two models with which I have become acquainted. The purpose here is not to develop new models for these diseases but to place these model types into the context of this examination of the principles of construction of epidemiological models, and, in particular, to understand the equilibrating mechanisms in models of these diseases.

The first model to be examined here is Macdonald's (1950) malaria model. Macdonald's primary purpose in constructing this model was to demonstrate the importance of including the phenomenon of superinfection in epidemiological models.

The two basic assumptions which Macdonald made are that the amount of infective material to which the population is exposed remains unchanged (and, in particular, is not affected by the proportion of the population harbouring malaria), and that the existence of infection is no barrier to superinfection.

In our simple model we have, then:

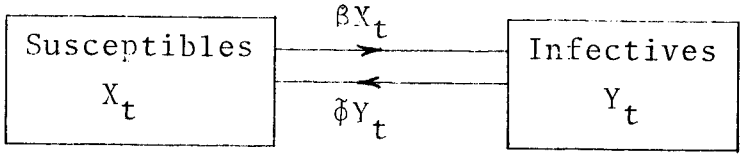


Figure 4.9: Simplified Model of Disease Type III

The essential point made by Macdonald is that the "effective recovery rate", ϕ, is not equivalent to the recovery rate from a single infection. As in the model of tetanus, we have

$$X_\infty = \frac{\tilde{\phi}}{(\tilde{\phi}+\beta)} \quad .$$

In Appendix 4.5 we consider the above process as a classic immigration-death process and find that the equilibrium value of X_t is $X_\infty = 1 - e^{-\frac{\beta}{\phi}}$ or $X_\infty = 1 - \frac{\beta}{\phi}$ when the possibility of the host carrying two or more infections simultaneously is neglected. Equating the equilibrium solutions in the two approaches, we find $\tilde{\phi} = \phi - \beta$, which is Macdonald's solution. (Appendix 4.5 also outlines Macdonald's approach and the limitations of his analysis.)

The second paper to be examined is Macdonald's (1965) publication on "The Dynamics of Helminth Infections, with Special Reference to Schistosomes." The assumptions of central importance in the dynamics of this model pertain to the quantitative relationships between the number of hatched larvae (miracidium), the snail population, and the number of free-swimming larvae (cercariae). Macdonald assumes that the possibility of superinfection of the alternative (snail) host may be ignored,"...with the result that the infectivity of the alternative host cannot for long remain proportional to the number of infections it has received". The second crucial factor is that the mean load is generally such that the mericidia are much more numerous than the susceptible snails. The result is that "...modification in the number [of mercidia] within a very wide range produces an insignificant change in the ultimate number of infective snails."

Under the above assumption the force of infection is unrelated

to the worm load in the community and the resulting catalytic model is that presented earlier in this section (for malaria).

In contrast to malaria, superinfection is the rule in schistosomiasis. The "susceptible" - "infected" dichotomy is no longer useful since the relevant measure of infestation is now the number of worms harboured by the individual. If w is the average number of worms carried by an individual,

$$\frac{dw}{dt} = \beta - \phi w$$

whence

$$w^* = \frac{\beta}{\phi}$$

[This can also be derived from the equilibrium distribution - see Appendix 4.5.]

This can be seen to be consistent with Macdonald's conclusion, emerging from his simulation model, that "...the ultimate level of endemicity attained,...is almost exclusively dependent on the number of snails, the frequency of entry to water and the longevity of the worm." Under the assumptions of the model outlined above, it is the first two factors which are the primary determinants of β , while the longevity of the worm is represented by ϕ.

In Appendix 4.6 the approach of the system to equilibrium is determined in both the deterministic and stochastic cases. The difference equation can be written as

$$\frac{\Delta w}{\Delta t} = \frac{1}{\phi} (w_\infty - w_t) \quad ;$$

the system is likely to be stable in the vicinity of the equilibrium point.

It should be noted that while account is not taken of the way in which immunity may interfere with the process, it is possible to obtain intuitively plausible results. In particular we note that the relationships are such that equilibrium is attained without the invocation of immunity.

IV) Models of diseases with superinfection and with force of infection determined by environmental contamination.

A search of the literature has revealed no models of this sort, which would pertain to helminthic infections which are transmitted primarily from person to person (in Rosenau's (1951) classification, such as ancylostomiasis (hookworm infection), strongylodiasis (a roundworm), trichuriasis (whipworm), and ascariasis (large roundworm). Inferences concerning the dynamics of this class of diseases have been made on the basis of epidemiological models of other diseases (e.g. schistosomiasis). The important differences in the nature of the relationships between different epidemiological classes make these extrapolations inappropriate.

In common with other helminthic diseases such as schistosomiasis, the unit of infectivity of the host is the number of parasites harboured, whereas in most other infections infectivity is a "zero-one" phenomenon. The crucial systemic distinction between

the infections examined here (e.g., hookworm) and those of Section III above (e.g., bilharzia) is that in hookworm the force of infection is directly related to the egg output in the feces and thus to the worm load in the population. (For schistosomiasis, we recall, the force of infection was effectively unrelated to the egg output.)

In the models which will be developed in this chapter various sources of non-linearity in both the soil and host stages of the ecological cycle of hookworm will be investigated.

Soil Non-Linearities:

Linearity is the most obvious form of the relationship between the number of infective larvae (and thus the force of infection) and the acquisition of worms. For the present we assume that the capacity of the intermediate host (soil) is virtually infinite (in contrast to the number of snails in the schistosmiasis model). It could be argued that the food supply during the larval development stage is obtained from the feces and that at a high larval density this supply would be constraining. We have no data with which to test this hypothesis, but it seems unlikely that this or any other "crowding" effect would markedly alter the linear relationship. [In discussions of the life cycle of hookworm, exhaustion of food supply is considered only in the context of third stage larval destruction. It should be borne in mind that after shedding the second larval cuticle the larva takes no more food from the environment. The storage of granules

in the larval body appears to be limited by larval capacity rather than by scarcity of food supply and thus "larval death through exhaustion of food supply" is quite consistent with the existence of a large food supply in the environment (and more particularly in the feces).]

Another possible sort of non-linearity in the soil which bears consideration is related to soil heterogeneity. This could become a factor if a certain segment of the population start using latrines and if this results in an increased proportion of the remaining traditional defecators using the preferred moist shaded sights. While this effect will be explicitly introduced into the models which will be developed, we will not consider it further in the present context.

If non-linearities in the host are ignored, we have the following differential equation describing the change in the average number of worms harboured:

$$\frac{dw}{dt} = \beta w - \phi w$$

$$= 0 \quad \text{only for } \beta = \phi \text{ and then at any (the initial) worm load;}$$

$$\text{or } w = 0, \text{ for all } \phi \text{ and } \beta .$$

For $\beta = \phi + \varepsilon$ we have an infinite increase in the worm load for $\varepsilon > 0$ and complete elimination of infection with $\varepsilon < 0$.

In short, the nature of the equilibrium conditions makes it clear that the above model does not represent the realities of hookworm infection adequately. Non-linearities in those stages

of the ecological cycle which occur in the vertebrate host have to be considered.

Host Non-Linearities:

In the epidemiological models to be developed we will assume that an increased worm burden in the host is related to: an increase in the rate of expulsion of mature worms; a decrease in the proportion of penetrated larvae which develop to maturity; and a decrease in the egg production per worm. These effects may be related to both an immune response mobilized by the host and the heterogeneity of niches within the human host. (The assumption in the latter case is that at high loads less favourable niches within the intestine may be inhabited by the worms.)

If we consider immunity to manifest itself by modifying the proportion [I(w)] of penetrated larvae which will not develop to maturity and if we assume the form of I(w) to be logistic we have:

$$\frac{dw}{dt} = \beta[1-I(w)]w - \phi w$$

where

$$I(w) = \frac{\psi_1 e^{\psi_2 w}}{1+\psi_1(e^{\psi_2 w}-1)}$$

whence

$$w_\infty = \frac{1}{\psi_2} \ln \left(\frac{1-\psi_1}{\psi_1}\right)\left(\frac{\beta}{\phi} - 1\right)$$

If we consider immunity to affect the rate of worm expulsion and if we assume that this rate is related to the worm load by the function:

$$r(w) = \psi_3 e^{\psi_4 w}$$

then $$\frac{dw}{dt} = \beta w - \phi r(w) w$$

$$= \beta w - \phi \psi_3 e^{\psi_4 w} w$$

whence $$w_\infty = \frac{1}{\psi_4} \ln \left(\frac{\beta}{\phi \psi_3} \right)$$

As in the case of the cholera model (and again this result has to be interpreted with caution) we find critical values of β (viz. $\beta_c = \frac{\phi}{1-\psi_1}$ and $\beta_c = \phi\psi_3$, respectively) below which we have, in this deterministic model, eradication of the disease.

Thus from theoretical considerations arising out of the mathematics of a simple epidemiological model we concur entirely with Darling who "...was accustomed to stress the discrepancies between the level of hookworm infection in the population groups he had studied and their very great exposure to infection," and who "...repeatedly stated in conversation that the development of an acquired immunity in these populations seemed to him the only explanation of the failure of their worm burdens to increase until they were all killed by the hookworms." (Cort [1929]).

Conclusion:

In this section I have examined the nature of epidemiological models for different classes of diseases. The simple analytic models developed in this section provide useful insights into the dynamics of more complex simulation models and insights into the nature of the equilibrating mechanisms of these models. The classification of the models reflects the primary importance of the dichotomy between those diseases in which multiplication takes place within the vertebrate host and in which the individual can be simply described as "infected" or "uninfected," and those diseases in which superinfection takes place. The importance of the relationship between the unit of infectivity and the force of infection is basic, too. The classification system is potentially useful in suggesting which infectious diseases may respond similarly to, say, changes in water supply or excreta disposal conditions. Thus in a study of the effect of environmental changes the classification system may be used for selecting "indicator diseases" from the wide range of diseases of interest.

Perhaps the most important contribution of this section is the demonstration that the linear type of epidemiological model which has been the norm to date is inadequate, at least for soil transmitted helminths. Ecologists examining the distribution of species have moved beyond linear models. MacArthur (1972) has summarized this improvement by stating that

"The population geneticist is content to say: 'Let fitness $= \frac{1}{x}\frac{dx}{dt}$', and then he proceeds in a purely mathematical program. The ecologist can do slightly better because he believes that $\frac{1}{x}\frac{dx}{dt}$ begins at a high level r and, as x grows to some level K (the carrying capacity), $\frac{1}{x}\frac{dx}{dt}$ declines to zero."

In the models which follow I attempt to incorporate these density dependent and immune response factors into epidemiological models for hookworm.

4.3. MODEL STRUCTURE

A fundamental difference between helminths and other parasitic agents such as protozoa, bacteria and viruses is that the helminths do not multiply within the vertebrate host. In cholera, typhoid and tetanus, for example, the unit of infectivity is the number of infected hosts, while in helminthic infections the average number of parasites harboured by an individual becomes the unit of infectivity. This distinction leads to a fundamental difference in the nature of the data available for, say, typhoid and ancylostomiasis and in the model structures which are useful in analysing these diseases. For typhoid the data available pertain to the rates of transition between different human states (such as infected, susceptible, immune). In hookworm the nature of the data is "helminthocentric": we have data on worm maturation, egg production, larval survival, etc. In modelling a helminthic disease, then, we cannot take account of the ecology of the agent implicitly (as was done for typhoid) but have to explicitly include both man and worm in the model.

The ecological cycle of A. duodenale is depicted in Figure 4.10 overleaf (from Hunter [1960]).

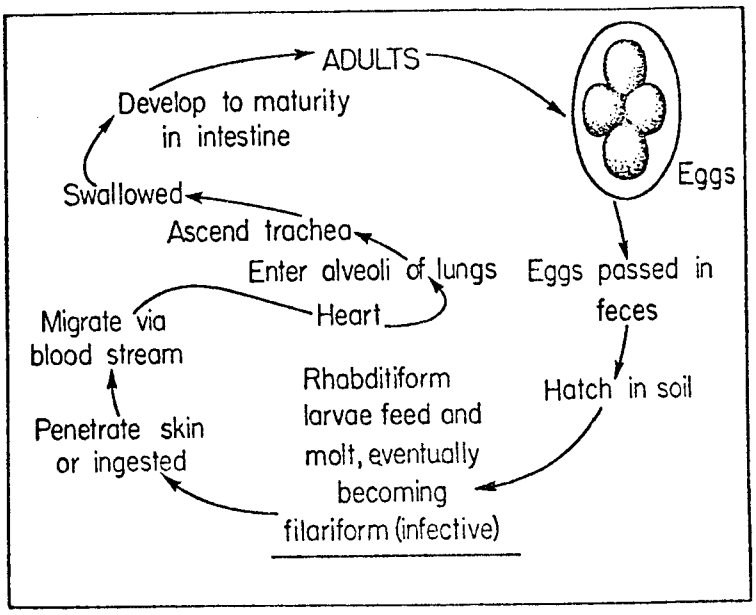


Figure 4.10: Nematode Cycle - Hookworm Type

Bearing in mind the nature of the data available and the purposes to which we wish to put the model, we may consider transmission to take place as follows: eggs are passed into the soil; eggs are hatched and developed through the first and second larval stages; man is subject to inoculation; and the worms mature and mate in the host. The schematic representation of the hookworm cycle which forms the basis of the mathematical epidemiological models is, then:

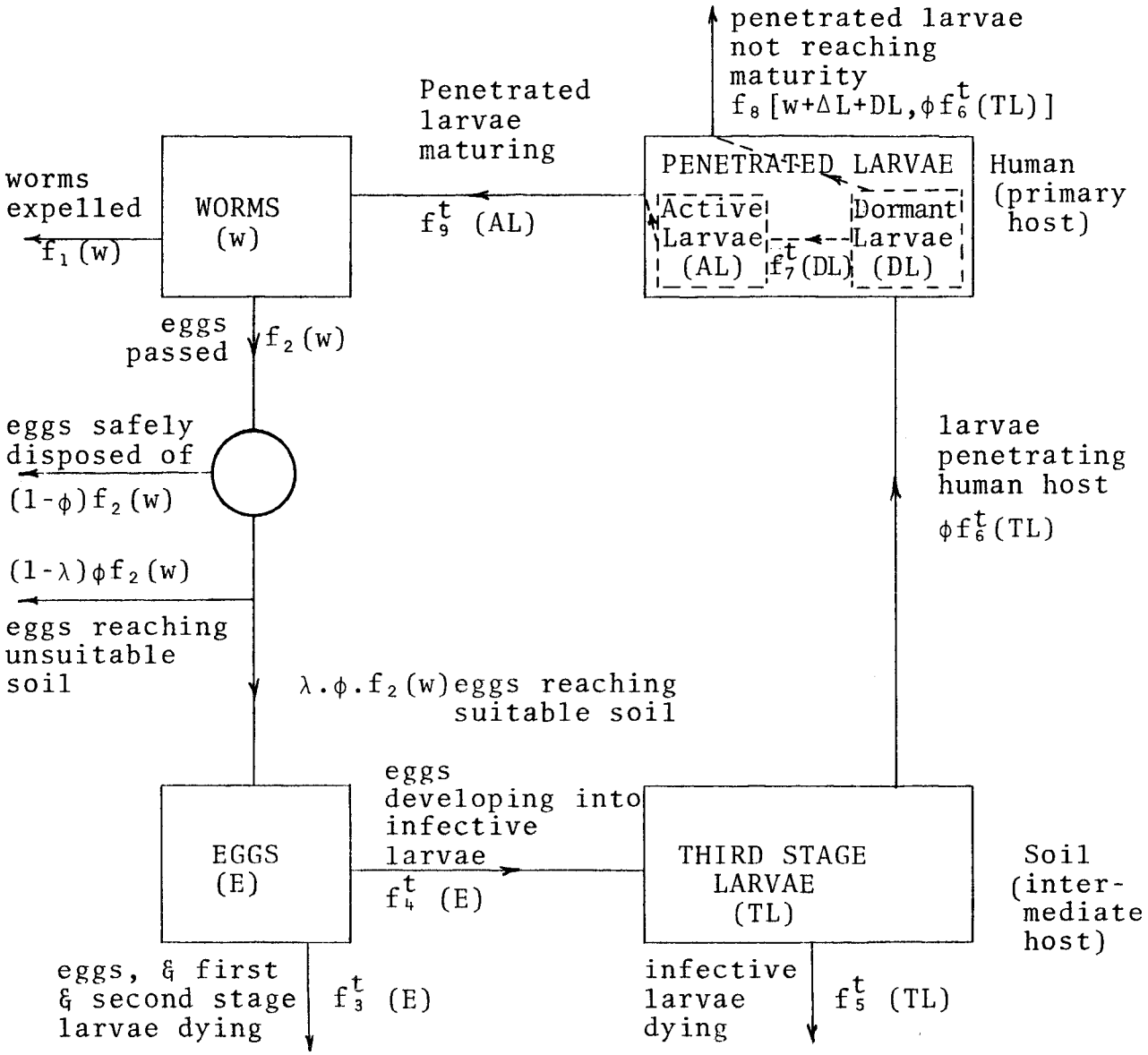


Figure 4.11: Schematic Representation of Hookworm Cycle

(In Figure 4.11, ϕ represents the proportion of the human hosts [using latrines] who neither contribute to the egg pool nor are exposed to the infective larvae in the traditional defecation areas; λ represents the proportion of the traditional defecation area which is suitable for the development of the hookworm ova.)

In the later sections of this paper on parameter estimation the nature of the relationships in this diagram will be evaluated in some detail. Here we wish to indicate briefly the types of considerations which underlie the "flow" specifications in this model.

i) The number of worms expelled daily is taken to be a function of the worm load of the community.

ii) The daily number of eggs reaching the soil depends on two sets of variables: firstly, on the populations of mature worms in the community, the mean worm load, the proportion of worms which are paired and the egg-output per fertilized worm; and secondly, on the proportion of ova which reach the soil. The first group are a function of the infection status of the community and the second a function of sanitation habits.

iii) The rate at which eggs and first and second stage larvae are destroyed is a function of the soil type, vegetation, coprophagus beetle activity, moisture and temperature in the place of fecal deposition. This rate thus varies with micro-geography, micro-climate and season.

iv) The rate at which infective (third stage) larvae develop is dependent on the number of eggs and on the environmental conditions in (iii) above.

v) The rate of infective larval destruction is a function of the number of third stage larvae and the above environmental conditions.

vi) The probability of an infective larva penetrating a human host has two constituents: a biological factor, dependent on the season, representing the efficiency of the larva in this respect; and a social factor which depends on the number of available human hosts and the nature of the exposure of these hosts to the contaminated soil.

vii) The proportion of penetrated larvae which do not develop to maturity in the human host is dependent on the degree to which the immune response of the host is mobilized. This proportion is dependent both on passive factors, which are related to race, age and diet, and on active immunity which is related to the parasite load of the host.

viii) The rate at which the "viable" penetrated larvae develop into mature intestinal worms is a function of the length of time for which these larvae may remain dormant in the body.

The system of difference equations describing the dynamics of the system is:

$$W_{t+\Delta t} = W_t + \{f_9(AL) - f_1(W)\}\Delta t$$

$$E_{t+\Delta t} = E_t + \{\lambda.\phi.f_2(W) - f_3^t(E) - f_4^t(E)\}\Delta t$$

$$TL_{t+\Delta t} = TL_t + \{f_4^t(E) - f_5^t(TL) - \phi.f_6^t(TL)\}\Delta t$$

$$DL_{t+\Delta t} = DL_t + \{\phi.f_6^t(TL) - f_7^t(DL) - f_8^t\ (W+AL+DL, \phi.f_6^t(TL))\}\Delta t$$

$$AL_{t+\Delta t} = AL_t + \{f_7^t(DL) - f_9^t(AL)\}\Delta t$$

where the variables and functions are "defined" on Figure 4.11. (Note that the superscript t denotes a relationship which varies according to the time of year.)

The models which are developed later in this section are derivatives, or varying complexity, of the above model.

4.4. TYPES OF HEALTH IMPROVEMENT PROGRAMS

Revelle and Thomas (1971) have presented a framework in which to consider environmental control methods.

> There are four general categories of methods of physical control that regulate the flux of residuals and ameliorate their effects. The first type consists of modifications or adjustments within the economic system itself to reduce or eliminate the generation of certain residuals ... The second type consists of the diverse processes of waste treatment and disposal that are introduced at the physical interface of the two systems (the environment and the economic system) at places where residuals are collected and released... The third type of control techniques treat the environment directly to remove pollutants or to reduce their adverse effects. The fourth and last category of control techniques consists of processes for purifying, cleaning or refining materials taken from the environment for use in the economic system, and of desensitization devices for direct physiological protection.

Adapting the above scheme to deal with the flow of fecal-borne pathogens between the population and the environment clarifies the ways in which disease control mechanisms can act.

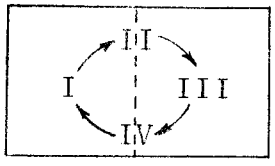


Figure 4.12: Schematic Representation of Health Controls

The first type of control mechanism consists of methods whereby the human carriers of the pathogen are treated directly. This

category of control mechanism includes curative treatment of patients suffering from disease symptoms. The second type of control mechanism consists of the diverse processes of excreta treatment and disposal that are introduced at the interface between the two systems. In treatment processes (e.g. sewage treatment) the physical, chemical and biological properties of wastes are altered to make them more innocuous. "Disposal processes include dispersion, dilution, detention and diversion. The first two of these reduce the concentrations of pollutants (pathogens) so as to lessen their effects. Dispersion is the distribution of residuals (pathogens) throughout greater volumes of the receiving air, water or land. Detention is useful for regulating releases at uniform rates or at times and seasons when the absorbtive capacity is adequate. Diversion is the transport of wastes to localities where absorbtive capacity is large or where environmental impairment is not likely to be serious." (Revelle and Thomas [1971]) The third type of control technique would treat the environment directly to destroy pathogens or vectors (e.g. swamp spraying with DDT). The fourth category of control techniques consists of methods of direct physiological protection (e.g. vaccination).

With respect to hookworm, examples of these control mechanisms are:

Type I administration of anthelmintics;

Type II use of sanitary latrines ("treatment"), expansion of traditional defecation areas ("dispersion"),

use of holding tanks (part "treatment", part "detention");

Type III use of biological controls, e.g. cockroaches, in defecation areas.

Type IV wearing shoes.

In the analysis which follows the emphasis will be on changed excreta disposal practices (Type II controls). The framework developed is quite capable of accounting for other types of control, as the simulation of the effects of anthelmintic drugs illustrates.

4.5. PARAMETER ESTIMATION

4.5.1. WORM LOSS

In this section we summarize the available direct and indirect data pertaining to the loss of Ancylostoma duodenale from the human host and then attempt to derive a reasonable functional form for describing this relationship.

In the models which are developed later, two different worm loss functions will be tried - in the first case we will assume a constant worm loss rate, while in the second case we will assume that the primary manifestation of acquired immunity is that the worm expulsion rate is dependent on the worm load in the host. We will derive appropriate parameter values for each of these assumptions.

In developing the required relationship from the available data, several major caveats should be borne in mind:

i) Recognizing that the hookworms of man are very different from those of dogs (Beaver [1961]), it nevertheless has been necessary to draw on studies (e.g. Sarles' study [1929]) of A. caninum for information.

ii) As pointed out by Kendrick (1934), the variability of the length of life of dog hookworm, even in puppies from the same litter, suggest that variability in the longevity of A. duodenale and N. americanus may be an important factor. This variability is not introduced

into the present analysis due to both the lack of data and the consequent complication in the specification of the model.

iii) All of the available data refer to the loss of worms under conditions in which reinfection is absent. Stoll (1962) doubts that inferences drawn from these data are applicable under endemic conditions, but Schad (personal communication) suggests that the decline in egg counts in the field is quite similar to that found in prisoners who are removed from the sources of infection.

Implicit in the analysis which follows is the inclusion of those factors which mobilize an immune response but which are not contingent on the presence of reinfection. These factors (e.g. racial factors) would be adequately taken into account in the data used.

a) Linear Rate of Worm Loss

The first attempt to mathematically define the loss of worms over time was published by Sarles (1929) for A. caninum. The logarithm of the egg output (y) (which was assumed to be linearly related to the number of patent worms), for dogs with a single exposure to a large number of larvae, was found to be approximately linearly related to time (t):

$$y = ab^t \quad \text{(where b is less than 1)}$$

A minor mutation of this exponential function is implied in

the descriptions of Sarles' contemporaries, Chandler (1929) and Kendrick (1934), of their experiments with human hookworm in India. They observed that prisoners who were removed from sources of further infestation exhibited a rapid loss of worms in the first half year followed by a progressive but slower drop thereafter.

Stoll has found that the form of the relationship emerging from all published work on worm expulsion is suggestive of the exponential decay curve derived by Sarles. It is this form which will be used, notwithstanding Stoll's stern warning of the inapplicability of these data under endemic conditions.

This formulation is convenient for several reasons: the process is Markovian (with the state at time t+1 dependent on the state at t and no prior state); the number of variables required to define the state at time t is small (to be precise, only one variable, the worm load, is required); the community load only, and not the distribution of this load among members of the community, is of interest (due to the rate of loss being independent of the worm load).

In the literature on hookworm, data on the loss of worms are sparse. Many of these data pertain to the length of life of hookworm (given variously as 8-15 years [Beaver,1967] 6 or 7 years [Chandler,1929] and 76 months [Kendrick, 1934]). These figures refer to the period for which some worms are not expelled and give little indication as to the moments of the length of life of a worm. The numbers are of little value but the data from which

they were derived will form the basis of the analysis which follows.

Several distinct types of data will be used in deriving a set of parameters for the worm loss function. Mhaskar and Chandler (1929) recorded the egg outputs of prisoners at Trichinopoly, South India, and Calcutta who were naturally infected with hookworm and who were not exposed to sources of reinfection (see Table 4.1 below). (It should be noted that N. americanus accounts for over 95% of the hookworm in South India and about 80% in Bengal [Patel, 1954].)

	Time in years								
	1/4	1/2	1	2	3	4	6	8	9
Calcutta	50%	60	70	80	86	90	93	95	96
Trichinopoly	41%	51	64	76	82	84	87	89	

Table 4.1. Percent Loss of Worms (Eggs) over Time

Kendrick (1934) inoculated volunteers in an Andaman Island jail, who were neither infested with hookworm nor exposed to sources of infection, with hookworm (both A. duodenale and N. americanus) larvae and observed the trend in egg counts. The relevant inoculation and egg count data are listed in Tables 4.2 and 4.3 overleaf.

The final source of Indian data of use in this context is Maplestone (1932) whose interest was focussed on the phenomenon of the seasonal variation in hookworm infestation. Figure 4.13

A. - Ancylostome Group

Case Number	Dates of Infection			Method of Infection	Approximate Number of Larvae Used		
	First	Second	Third		First Infection	Second Infection	Third Infection
	1926	1926	1926				
A-3	April 3	June 1		Skin	200	200	
A-8	May 20			Skin	200		
A-11	May 17	Sept. 7		Skin	200	300	
A-18	May 17			Skin	200		
A-20	May 17	Sept. 7		Skin	200	300	

N. - Necator Group

Case Number	Date of Infection	Method of Infection	Approximate Number of Larvae Used
	1927		
N-1	September 23	Skin	200
N-2	September 23	Skin	200
N-3	September 23	Skin	200
N-5	October 5	Skin	200

Table 4.2. Dates and Quantities of Experimental Hookworm Infections

Year	Quarter	Average Counts								
		Case A-18	Case A-20	Case A-11	Case A-8	Case A-3	Case N-2	Case N-3	Case N-5	Case N-1
1926	1									
	2									
	3									
	4	100		3,200	270	1,600				
1927	1	570	6,130	4,830	1,830	2,630				
	2	1,200	5,070	4,470	2,500	2,170				
	3	1,530	7,370	9,470	3,670	2,600				
	4	1,870	7,570	10,150	1,800	2,000				
1928	1	700	4,030	4,370	1,220	2,000	470	500	270	366
	2	570	3,630	2,070	1,270	1,800	470	670	300	233
	3	600	3,970	1,870	950	1,900	770	730	400	116
	4	620	4,270	2,730	380	2,170	670	830	380	116
1929	1	620	3,830	3,470		1,370	800	850	320	
	2	730	3,720	2,650		1,630	620	970	380	
	3	730	4,420	2,120		1,380	470	470		
	4	750	3,720	1,280		1,260	540	630		
1930	1	700	3,730	1,370		1,450	530	640		
	2	530	1,830	720		950	410	470		
	3	570	1,600	800			530	570		
	4	530	1,170	870			430	530		
1931	1	580	950				450	620		
	2	680	780				560	630		
	3	580	460							
	4	580								
1932	1	330								
	2	130								
	3	100								
	4									

Table 4.3. Egg Counts from Experimental Infections

(as presented in Chandler [1935]) gives Maplestone's monthly observations of prisoners in Calcutta who were no longer exposed to hookworm infection, and tea workers in Assam and North Bengal and Bengali villagers who lived under endemic conditions. These data (Chandler [1935]) are presented on Figure 4.13 below.

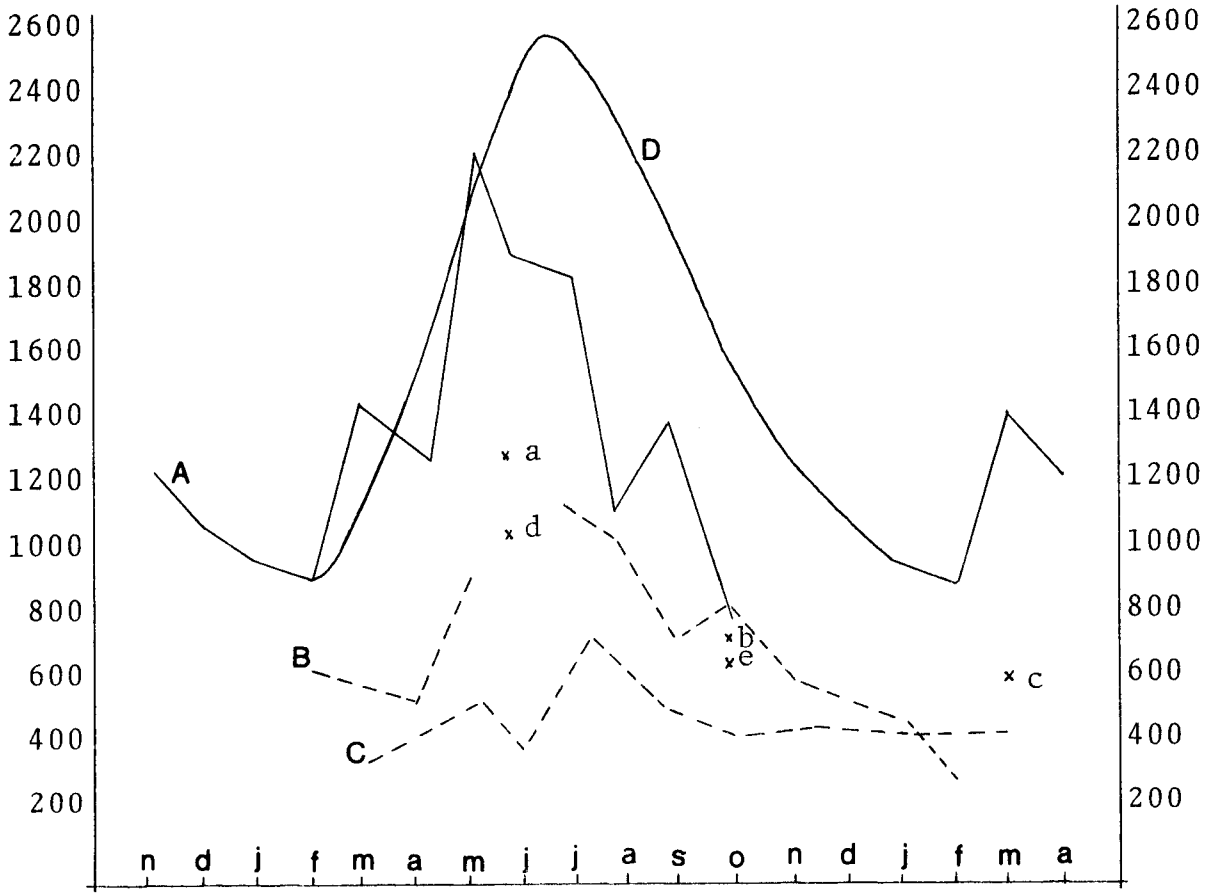


Figure 4.13: Seasonal Variation in Hookworm Loads (Maplestone's Data)

A: prisoners in Calcutta jail

B: workers in Sylhet tea gardens

a,b,c,d,e: workers in North Bengal tea gardens

C: Bengali villagers and jute mill workers

D: Hypothetical version of A if reinfection had not been stopped in November.

It should be borne in mind that in using the above data to estimate the worm expulsion rate several complicating factors are ignored.

i) With the exception of Kendrick's experiments, these data do not distinguish between Ancylostoma duodenale and Necator americanus. (This distinction can not be drawn on the basis of egg examination alone.) Relative to the subject of loss and reinfection rates, Roche and Layrisse (1966) state:

> As for turn over, the studies done in India, with mixed infections, would indicate that it is rapid, requiring approximately three to six months for a decrease of 50 per cent from the original level, while all the studies done in the Western hemisphere, in infections predominately or exclusively with Necator, would indicate a slow turnover, of the order of years.
> (quoted in Choudhury [1968])

I will follow, however, other contemporary authorities, (specifically Beaver [1961] and Otto [1965]), in not distinguishing between these two varieties in this respect.

ii) Kendrick (1934) has drawn attention to a distinction between his data and those of Chandler and Mhaskar. In the latter (we include Maplestone's data in this group) existing worms in an individual originated in larval penetrations which were distributed over time, whereas Kendrick's volunteers were subjected to a single innoculum (in some cases there were two inoculations) of infective larvae. The age distributions of the worms in these two cases therefore would be different.

iii) Until recently it was believed that the period between larval infection and worm maturation was a constant. Kendrick

(1934), for instance, believed that his data were for "worms [of] uniform age" since inoculation was a one shot process. As will be discussed in detail later, the matter of maturation is rather more complicated.

The parameters of an exponential worm loss curve, $W_t=W_o e^{-\alpha t}$, are derived for each of the above data sets (see details in Appendix 4.7), and presented on Table 4.4 below:

		α (with 95% confidence intervals)
Chandler	(108 months)	.0351 ± .0080
Mhaskar	(96 months)	.0299 ± .0090
Kendrick: A.duodenale:		
Avg. for completed cases:		.0623 ± .0081
Complete infections:		
A-8	(18 months)	.1842 ± .0610
A-18	(63 months)	.0437 ± .0091
A-20	(51 months)	.0575 ± .0111
Incomplete infections:		
A-3	(39 months)	.0210 ± .0031
A-11	(39 months)	.0805 ± .0142
Maplestone (5 months' observations)		.2678 ± .0272

Table 4.4. Worm Loss Parameters

From the table of α values and the plots of the group (as opposed to individual) curves on Figure 4.14 we note the following:

i) The variation in α values is wide, particularly among individuals.

ii) In accordance with Chandler's (1935) observation, the rate of loss of worms is markedly greater during the first

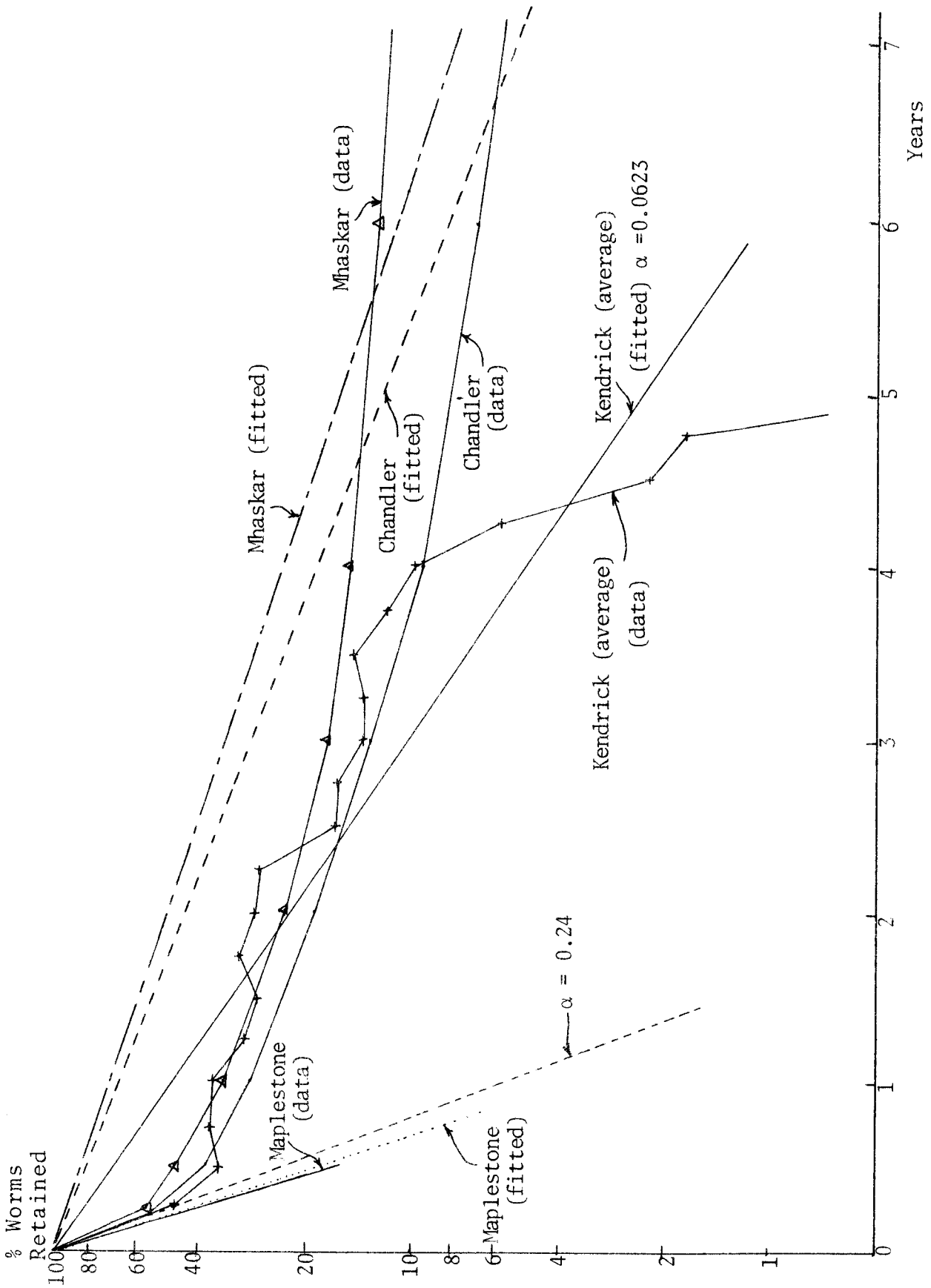


Figure 4.14: Worm Loss Curves

six months than it is thereafter. As a result of this there is a tendency for shorter observation periods to yield higher α values.

This latter observation, while not in accordance with the postulated constant rate of loss relationship, is not entirely unexpected. Stoll's brief discussion of the differences in worm expulsion under endemic and non-endemic conditions suggests the following plausible, albeit not rigorously documented, explanation: During the first few months of observation the removal of reinfection sources may not cause a serious change in the immune response of the host. After this initial period, however, the absence of reinfection is recognised, the immune response is lowered, and the rate of expulsion drops.

An alternative explanation is that while human immune response is nearly constant, the worms may be heterogeneous in resistance (a polygenetic trait). The truth probably lies somewhere in between the two explanations.

The intention of this paper is to derive a model for hookworm infestation under endemic conditions. It therefore seems appropriate to utilize a value of α which may be relevant under conditions of reinfection. From Figure 4.14 it may be seen that the losses found by Chandler, Mhaskar, Kendrick and Maplestone for the first several months are similar. A value of $\alpha = 0.24$ has been chosen as realistic under endemic conditions. This value implies a loss of approximately three quarters of the worms in six months, thus according with Otto's (1965) general-

ization that "most of the worms are lost in six months."

In the numerical analysis of the epidemiological model the accounting period will be taken as one day. The proportion of worms lost daily will be 0.7864%.

In conclusion we note that the assumption of a linear daily loss function enables us to consider only the community worm load (and not the distribution of this load) in determination of the change of the worm load. This property does not pertain when we introduce a function relating the worm expulsion rate to the worm load in the individual through an immune reaction.

b) Acquired Immunity Affecting Rate of Worm Expulsion

In the literature there is no attempt to specify the likely form of the relationship between the rate of worm expulsion and the worm load. We will assume this relationship to be of the form $d_2(w) = \alpha_2 e^{\beta_2 w}$ and will use Chandler's loss data to estimate the parameters. Chandler(1929)gives the average EPG (eggs per gram of feces) of the prisoners examined to be 758. If we assume that these prisoners had 80% N. americanus and 20% A. duodenale worms, the implied mean worm load is about 50. Patel (1954) has suggested that Chandler's data represented considerable under-counts. Contemporary evidence (Schad - personal communication) indicates that the mean life span for A. duodenale is of the order of one year in West Bengal. Chandler's data have been examined at several initial worm loads, and that worm load chosen (200 worm per capita) which gives a mean residence time of one year.

The relationship emerging is:

$$d_2(w) = 0.000449e^{0.0186w} \text{ per day.}$$

The relationship between worm load and time is given by the equation:

$$\int_{w_o}^{w_t} \left(\frac{1}{\alpha w e^{\beta w}} \right) dw = t + c \ .$$

This integral is evaluated using Simpson's Rule (see Appendix 4.8) (a simple form for the integral does not exist) and the resulting fitted curve plotted, along with Chandler's data, on Figure 4.15 below.

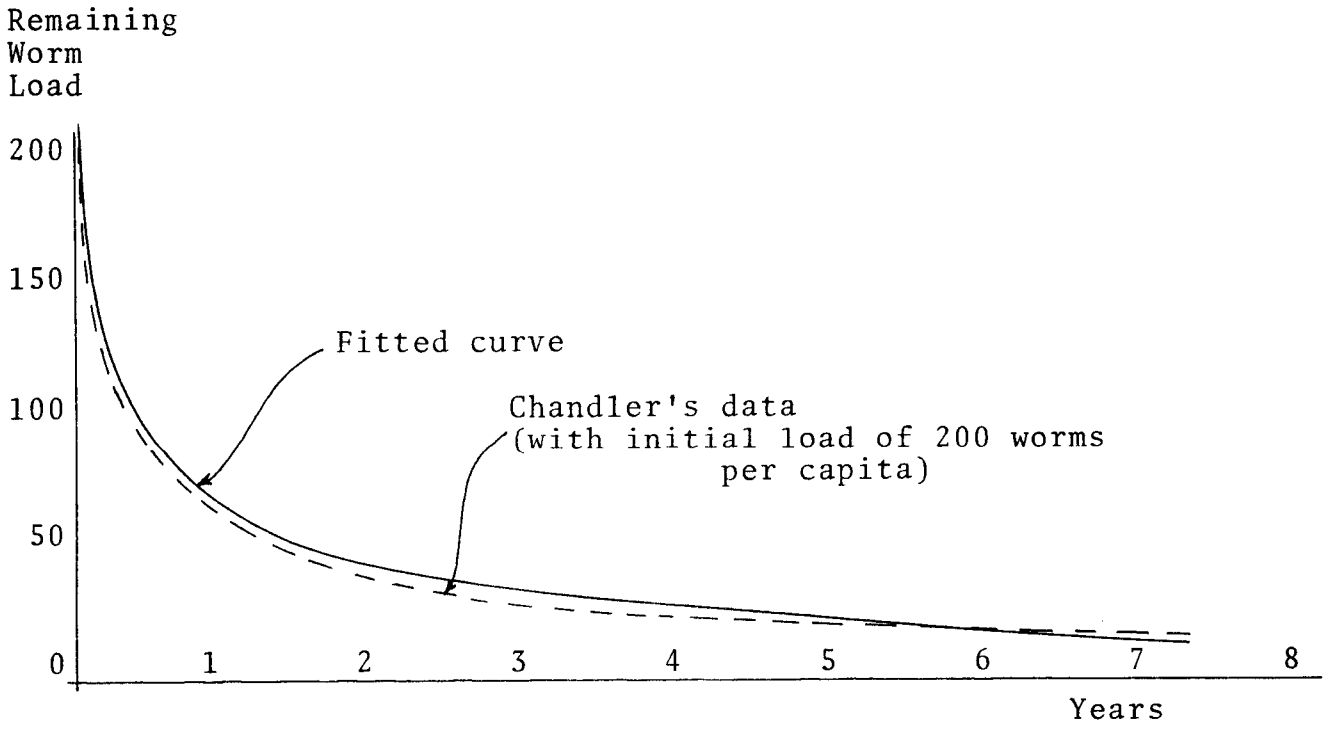


Figure 4.15: Worm Loss Curves with Immunity

4.5.2 EGG DISCHARGE

Given the number of mature worms carried by a host, we wish to determine the output of hookworm ova.

Beaver (1961) has drawn attention to the statistical character of egg counts as estimates of egg output and as related to worm burden. Sarles (1929) has shown that the size of infestations, as well as their age, affects the egg production of A. caninum. Otto (1965) has stated that the egg production of human helminths is influenced by factors such as age and number of worms, nutrition and the immunological status of the host.

The above facts notwithstanding, data availability and model simplicity suggest that we follow convention (see, for instance, Chandler [1929] and Patel [1954]) in assuming that the egg output is linearly related to the worm load.

We are interested in two different measures of egg output: in terms of the life cycle of the disease we are interested in the total daily output of eggs per ova-depositing worm; when classifying the severity of the infestation, recourse is frequently made to the EPG (eggs per gram of feces) count. It is necessary to consider the various (usually implicit) estimates of the daily feces output to ensure that the above measures are consistent.

In the hookworm literature Chandler (1929) implicitly takes the average fecal mass per capita to be 150 grams per day worldwide and 210 grams per day in India. Otto (1965) has an implicit figure of 180 grams per day in his discussion, which is

not specific to any country, of hookworm. Other sources would suggest that these figures are low: Gotaas (1956) gives a general estimate of 200 gms/cap/day; Wagner and Lanoix (1958) give a figure of 200 to 400 gms/cap/day for Asia; Subrahmanyam (1975) uses 450 to 500 gms/cap/day as a representative Indian figure; and Bell (1973) has a typical Indian vegetarian producing 540 gms/day. The most recent and reliable data available are from the Johns Hopkins (1970) team in rural West Bengal where the per capita daily fecal mass was found to average 200 to 290 grams. In this analysis we will assume the average fecal output to be 300 grams per capita per day.

The following estimates of egg output per worm appear in the literature: N. americanus: i) Chandler (1929) estimated that each necator female is represented by about 25 EPG in "mushy Indian stools". Apparently the EPG count is the primary figure from which Chandler works and we will thus take this, and not his total daily egg production figure of 3000 to 10,000 per female necator, as his estimate. ii) Otto (1965) estimates that a necator female lays about 8000 to 10,000 eggs per day and that "a reasonable estimate of the number of N. americanus present may be obtained by dividing the number of eggs per gram of feces by 25 (implicitly assuming a daily fecal output of about 180 grams per capita). iii) Patel (1954), on the basis of examination of results from Ceylon, Calcutta, Madras, Australia and China, states that roughly 100 eggs per gram in a stool represents 8 necators. iv) Beaver (1961) on the basis of experimental

induced infections with necator, states that "the average stool from an individual harboring a single pair of hookworms might contain 50,000 eggs." This figure is extremely high. It is possible (Schad, personal communication) that the subject might have had a previous inapparent (unisexual) infection.

A.duodenale: i) Chandler (1929) found the egg count per A. duodenale to be 2 - 3 times as great as that per N. americanus. If we take Chandler's estimate for necator, we get 50 to 75 EPG per female ancylostoma, or about 25 to 37 EPG per anclostoma worm (male or female).

iii) Stoll (1962) gives a figure of 26 EPG per resident hookworm without being explicit about the species of reference. An earlier figure in this paper of Stoll's refers to data from the Southern USA and this would imply that this estimate pertains to necator. An earlier paper of Stoll's (quoted in Patel [1954]), however, gives a figure of 26 EPG per female necator and it therefore is assumed that the above figure refers to A. duodenale worms.

It was expected that there would be significant differences in the various estimates of egg outputs. With the exception of the estimate given by Beaver for A. duodenale, the estimates, 12 EPG per necator and 26 EPG per ancylostoma, are remarkably consistent. These estimates will be used in the subsequent analysis.

In the epidemiological models which will be developed we

will assume that all infestation takes place in the traditional defecation areas. We will attempt to assess the influence of year to year climatic variability on the development of larvae and thus on the community hookworm burden by correlating this variability with variation in the proportion of the defecation area which is deemed suitable for egg and larval development. We arbitrarily assume that this proportion (λ) is equal to 0.5 during the average year. [Note that choice of a different factor λ will not make any difference to most of the analysis which follows, since density-dependent effects in the soil are not included, and since the value of the penetration factor is chosen to give the required worm load.]

The number of eggs reaching "suitable" soil is taken, therefore, to be λ times the number of eggs discharged into the environment.

In the sections which follow we should, on consideration of the ecology of the helminth, like to deal with (i) egg development, (ii) first to third (infective) stage larval development, and (iii) infective larval development, and (iv) infective larval survival. The nature of the data is such that these distinctions cannot be clearly drawn in this analysis. The section on "egg to infective larva development" encompasses most of the analysis which would logically fall under (i) and (ii) above while section 4.5.4. deals largely with (iii) above. In several instances the stage to which the given data pertained was not precisely specified and this introduces a further degree of

"fuzziness" into the analysis.

What is ultimately needed here is clearly a lot of better data. For the present we will make what appear to be realistic estimates of the parameter values on the basis of the data which do exist. The sensitivity of the model results to a range of parametric values will be tested.

4.5.3. EGG DEVELOPMENT

In this section we wish to determine the proportion of the eggs which may be considered capable of development and the rates at which these fertile eggs either die or develop to the third (infective) larval stage. Following Cvjetanovic (1972), these rates of transition "can be considered as the product of two components, namely the rate of exit per unit of time from an epidemiological class, and the coefficient of transfer which represents the fraction of those leaving their former class and going into another at any time."

We wish to determine, therefore, the rates α and β in Figure 4.16.

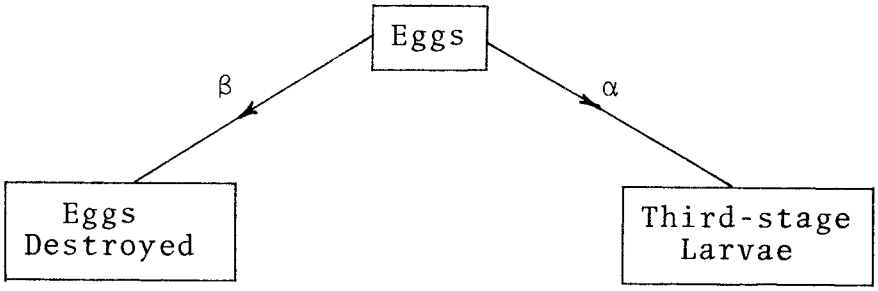


Figure 4.16. Schematic Representation of Egg Development

Environmental conditions greatly affect the development of viable hookworm eggs. The optimal temperature for egg-hatching lies between 70° and 85°F. Below 70°F development becomes slower, while at about 50°F development ceases entirely. As Chandler (1929) further points out, "the eggs are not injured by these [low] temperatures, at least not for many days, and proceed with their normal development when the temperature

rises." High temperatures destroy the eggs (Beaver [1961]). Eggs will not survive long without moisture (Otto [1965]) and are destroyed by direct sunlight (Beaver [1961]). The action of coprophagous beetles, which are highly active in India in those seasons when there is any moisture in the upper layers of ground (Chandler [1929]), produces excellent cultural conditions for egg development, since "their burying activity offers the only means by which the hookworm eggs deposited in stools in open sites can escape lethal temperatures on clear days." (Johns Hopkins [1970])

While the Indian meteorological service divides the year into four seasons, we will consider only a "favourable" (June through September) and "unfavourable" (October through May) season each year. The data available for the analysis of egg and larval development are so few that any finer "tuning" than this would be inappropriate at this stage.

a) Rate of egg development

In this section we will follow Cvjetanovic (1972) in assuming that the rate of transition out of the "ova to second stage larva" state is the inverse of the average duration in this state. It should be recognised that this implicitly assumes a Poisson process (with the probability density function of the time to the first "event" being the exponential distribution). For a Poisson process of rate α the mean development time (see Appendix 4.9) is $1/\alpha$.

Several estimates of the average time taken between ova

deposition and infective larva development under favourable conditions exist. Otto (1965) estimates the average time to be a week, Imperato (1974) gives a period of 7 to 10 days and Chandler (1929) gives a period of five days under "optimal conditions."

Under unfavourable conditions still fewer data are available. No data on the number of ova perishing under natural unfavourable conditions were found. We know that the time required to reach the infective stage is extended from five days to 13 days as the temperature falls from 70°F to 60°F and that at 50°F development ceases entirely (Chandler [1929]). Otto gives a more general statement, namely that development at sub-optimal temperatures may require 10 days to two weeks.

On the basis of these admittedly sparse data we assume that under favourable conditions the mean development period is 8 days, while under unfavourable conditions this period rises to 14 days. The corresponding rates of the transition are: α (favourable) = 0.125/day

α (unfavourable) = 0.0715/day.

ii) Rate of egg destruction (β)

Beaver measured the rate of development of infective larvae from a given number of eggs under optimal conditions. Six days after depositing feces containing 50,000 eggs in favourable conditions, Beaver (1961) picked up 25,000 hookworm larvae in a few minutes on a damp cotton pad. During these 6 days three

processes have been operative: egg and first and second stage larval destruction, egg and infective larval development, and infective larval death. In this section we will use the infective larval death rates which prevail under favourable and unfavourable conditions (these are derived in the next section) and the egg to infective larval development rates which were derived above. We will then determine the egg and first and second stage larval destruction rates which are necessary to give the results found above by Beaver.

It should be emphasized that this is squeezing a great deal more out of the data than can be justified. This is regrettable but it was considered worthwhile to lay out a method by which these parameters could be derived if and when an adequate data base becomes available. This procedure can also be justified as being the most logical basis on which to "guesstimate" the relevant parameters.

If we let α = the egg/second stage larval development rate, β = the egg/second stage larval destruction rate, and γ= infective larval death rate, we find the number of larvae living at day n to be (see Appendix 4.9):

$$100\alpha\left(\sum_{i=0}^{n-1}(1-\gamma)^{(n-1+i)}[(1-\beta)(1-\alpha)]^{i}\right) \qquad (1)$$

Under favourable natural conditions the values of α and γ are 0.125 (see above) and 0.15 (see next section) per day.

We will assume here that under "optimal" conditions $\alpha = 0.20$ (as given by Chandler) and $\gamma = 0.100$. Using these values in (1) above we wish to determine β such that:

$$50 = 100(0.200)\left(\sum_{i=0}^{5} (0.900)^{5+i}[0.75(1-\beta)]^{i}\right)$$

This can be solved most elegantly using the Newton Paphson iterative technique. We have determined the value of the right hand side for a few values of β and found that $(1-\beta) \simeq 0.965$ under optimal conditions.

There are a few direct data available on ova destruction in stored feces. [If β is the daily ovum destruction rate, the proportion of ova surviving a period of n days is $(1-\beta)^{n}$.]

Patel (1954) found that 55% of ova stored in feces are lost after a period of ten days. The implied value of β is 0.077/day. Stoll (1926), in China, found that under unfavourable temperatures "...ova (in feces stored in water) gradually die off at such a rate that in 4 to 5 weeks over 95% fail to develop when put into cultures at optimal conditions and over 99% in 6 weeks," while under favourable temperatures the periods are approximately doubled. The implied β values are 0.10 under unfavourable conditions and 0.05 under favourable conditions.

It is clear that more directly applicable data are required for the accurate estimation of the above parameters. In the absence of such data the values used in the development of the epidemiological model are:

Rates (per day)	Conditions		
	Optimal	Natural Favourable (June-Sept.)	Natural Unfavourable (Oct.-May)
Egg-infective larval development (α)	0.20	0.125	0.0715
Egg-infective larval destruction (β)	0.035	0.050	0.100
Larval destruction (γ)	0.10	0.15	0.45

Table 4.5: Egg Development Parameters

4.5.4. LARVAL SURVIVAL IN SOIL

After shedding the second larval cuticle, the larva is mature and ready for penetration of the human host. "There is no further growth and development and the larva takes no more food. The energy required for life and movement is obtained from granules stored in the body. As long as the nutritive material lasts the larva can live." (Chandler [1929])

The length of life of the larva (which we wish to describe mathematically in this section) is highly dependent on climatic, soil and vegetative conditions. "Experienced observers should be able, by a glance at the soil of an area, provided [they] know the mean temperature and rainfall of the region, to determine whether there ever can be a real hookworm problem." (Patel [1954])

The role of soil in the ecology of hookworm is similar to that played by intermediate hosts for other helminths (e.g. snails in the life cycle of schistosomiasis). Soil-transmitted helminths tolerate relatively narrow limits in the range of physical conditions within the soil (Beaver [1961]) and thus there is, ceteris paribus, a striking correlation between the amount of hookworm present and the type of soil (Patel [1954]). For the development and survival of hookworm larvae an open, light type of soil is essential (Beaver [1961]). (Ascaris and trichuris demand heavy colloidal soils.) Heavy clay soils such as the "black cotton soil" of Central India are highly unfavourable due to their tendencies to dry into hard clumps and the

consequent resistance to vertical infraction. Very light sandy or porous soils are also unfavourable unless there is a very abundant and evenly distributed rainfall. Sandy loams appear to be the most favourable soils(Chandler [1929]).

Exhaustion of food supply and dessication are believed to be the principal factors accounting for larval death (Beaver [1961]). Dessication is a function partly of the soil type and the vegetative cover but is primarily determined by the rainfall regime. There is little likelihood of significant hookworm disease where there is less than 40 inches of rainfall per year (Chandler [1929], Otto [1965]). Alternate wetting and drying of the soil, a function of the rainfall distribution and the soil type, hastens the death of infective larvae (Beaver [1953]).

Temperature asserts an important effect on larval survival. Patel (1954) reports that "over 9% will perish at 27°C after 9 weeks and at 35°C all were dead within 3 weeks. At 0°C larvae perish within one week." Anwikar (undated) asserts the optimal temperature for larval development to be 77° to 86°F.

Larval development is affected, too, by the biological environment: fungi, bacteria or protozoa hasten larval death (Patel [1954]).

There are several sources of data on which to base estimates of the survival of hookworm larvae under both favourable and unfavourable conditions. The limitations in the applicability of the emerging equations are serious: the environmental con-

ditions on which the data are based are narrow and the qualifications discussed previously regarding the applicability of an exponential decay function pertain in this case, too.

The results of two studies (by Beaver and Augustine) on the survival of hookworm larvae are presented by Beaver (1953). The results of Beaver's study of the development and survival of infective larvae in shaded Georgia soils are presented in Figure 4.17 below.

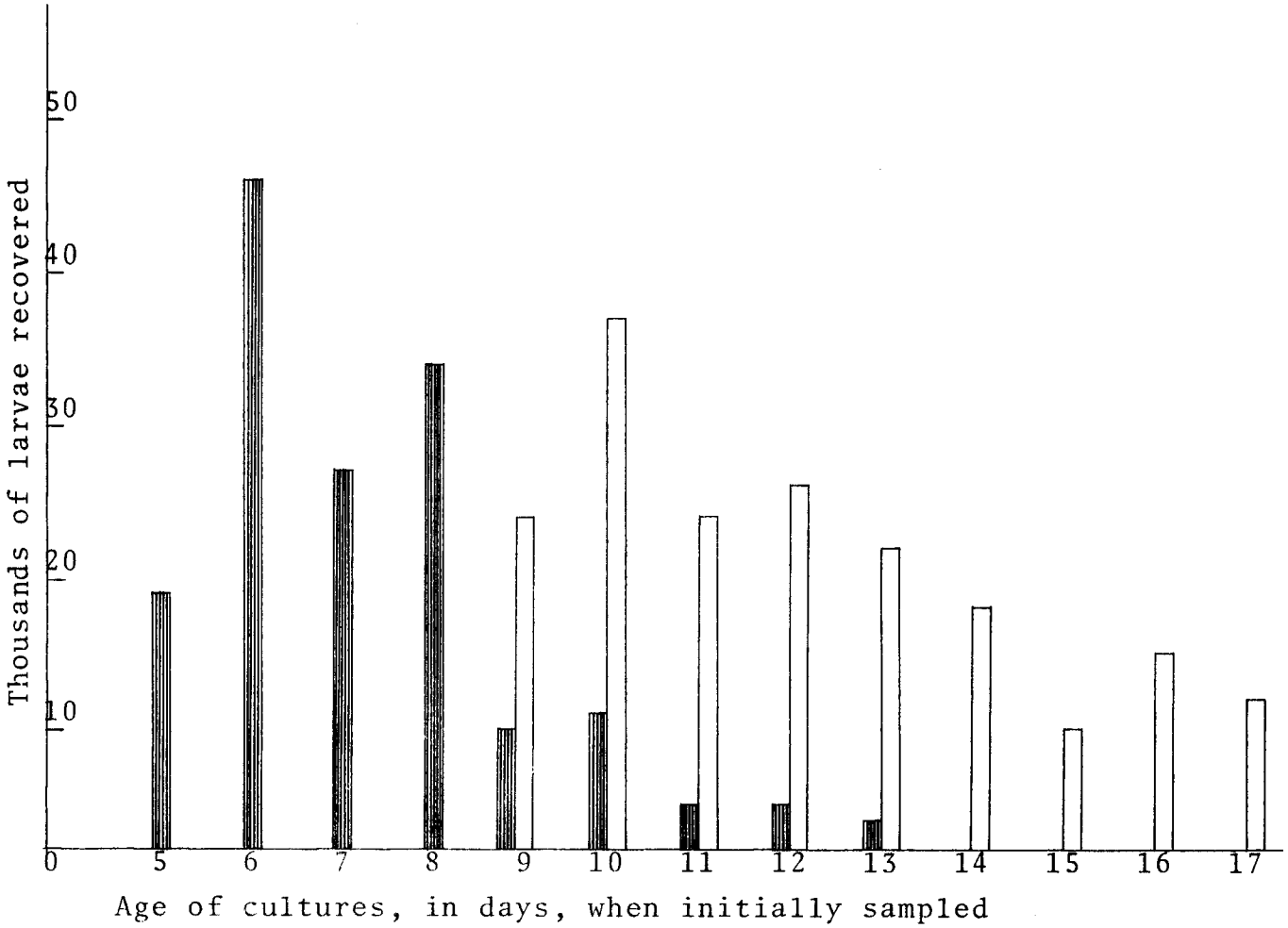


Figure 4.17. Total Yields in Hookworm from Soil Cultures Inoculated with 50,000 Eggs Each

The "open bar" data (on Figure 4.17) pertaining to conditions in which alternate wetting and drying were absent but moisture was satisfactory, are used in this analysis.

In analysing these results we face the same circumstances that were present in analysing the rate of loss of worms from the human host (see 4.5.1. above). In this case we have measurements over time of the number of infective larvae which are alive, but do not know when the eggs hatched. As we did in the worm loss case we will examine the decline in the number of larvae after the peak was reached. Postulating a Poisson process and then regressing the log of the number of larvae against time (forcing the curve through the initial point) we get the following relationship:

$y = 100e^{\alpha t}$ where y = % larvae surviving and t = time in days, and α is in $days^{-1}$. We find $\alpha = -.1880 \pm .0447$ (95% confidence interval) or $y = 100(.8286)^{t}$.

Augustine's experiments were of a slightly different nature - he did not start with eggs but with infective larvae. In analysing his data on the survival of these larvae in moist earth we take the time datum as the initial point. Augustine found that "there was 67% reduction in 5 days, 89% in 10 days and 92% in 15 days. Only one (taken here as 1%) surviving larva was found at 44 days and none at 7 weeks" (Beaver [1953]). The equation describing these results is

$y = 100e^{\alpha t}$ where the 95% confidence interval on α is

-0.11731 ± 0.0436 ; i.e. $y = 100(0.8893)^t$.

These results are in substantial agreement with other fragmentary data available:Chandler (1929) found 99% of the larvae to be dead in 6 weeks; Otto (1961) suggested that larvae may survive weeks under favourable circumstances.

The 95% confidence interval on the estimates of α from Beaver's and Augustine's data overlap in the range of -0.1433 to -0.1609. We will assume that $\alpha = -0.15$ is a reasonable value under favourable environmental conditions.

Patel (1954) has given estimates of the survival of larvae under different environmental conditions. While he is not precise about what his figures pertain to (from the above they would seem to be the maximum lengths of survival for particular samples), these will be used in estimating the survival curves under conditions less favourable than those pertaining above. Patel gives "survival times" of 8 weeks in deep shade, 6 weeks in light shade, 5 days in direct light and gives an unspecified figure of 14 days from Mhaskar which presumably applies to natural but unfavourable conditions. Assuming that "survival" in each of the above cases implies the same percentage larval survival we have:

$$y_{\text{light shade}} = y_{\text{unfavourable}}$$

and, therefore, $(e^{-\alpha}_{\text{light shade}})^{42} = (e^{-\alpha}_{\text{unfavourable}})^{14}$

If we choose a value of $e^{-\alpha}_{\text{light shade}} = e^{-0.15} = 0.8607$ (from

our earlier results) we have:

$$e^{-\alpha}_{\text{unfavourable}} = (e^{-\alpha}_{\text{light shade}})^{42/14} = 0.6376$$

In the analysis which follows we will use, therefore, a value of $e^{-\alpha}$ equal to 0.8607 in the four months of the year in which conditions may be considered favourable for larval survival and a value of $e^{-\alpha}$ equal to 0.6376 in the remaining eight "unfavourable" months. The corresponding α values are -0.15 and -0.45 respectively.

4.5.5. LARVAL PENETRATION

There are very few data indicating the magnitude of larval penetration under natural conditions. Specification of this relationship will primarily be on the basis of ensuring a reasonable outcome of the overall model.

Gooday (1925) has reported that, under ideal conditions, 76% of N. americanus unsheathed larvae penetrated the skin in one hour. In ideal conditions Nawalinski (1974) found that 40% of A. duodenale infective larvae penetrated into his arm in 40 minutes. Since feet are likely to be a good deal more resistant to larval penetration than arms, since people are unlikely to be in a defecation area for as long as 40 minutes a day, and since "ideal" conditions are unlikely to be replicated by even "favourable" natural conditions, these experimental data provide no more than an extreme upper limit to any estimate of larval penetration. It should be noted, too, that defecation areas in endemic hookworm areas may not be entirely covered with infective larvae. Jan et al. (1956) inspected a loamy defecation field, "full of human excreta, heavily manured and cultivated" outside Lahore, where "...it is easy to spot cases of ankylostomiasis." They found that only 12.7% of the 25 square yard plots, chosen at random and inspected during the months of January through May, were infected with hookworm. (These results are, however, hardly conclusive. Despite the authors' assertion concerning the

importance of hookworm in this area it is generally believed to be an area of very low infestation. Schad's data (1973), furthermore, indicate that the period January through May is likely to coincide with minimal egg transmission.)

Although evidence to this effect is lacking, it seems likely that the percentage of larvae penetrating the skin would vary from individual to individual. Kendrick (1934) found that the percentage of larvae that penetrated the skin and finally reached maturity varied greatly in different individuals.

We assume that the number of infective larvae penetrating human hosts will be proportional to the product of the number of human hosts visiting the traditional defecation sites and the number of infective larvae present in these sites. Since ancylostosome larvae require a shallow layer of liquid to afford them the necessary physical conditions to effect skin entry (Gooday [1925]), the factor of proportionality (ξ) chosen will be quite different (a ratio of one to four is assumed) in the wet and dry seasons. (ξ will vary considerably with community defecation habits.)

Assuming that the majority of infestation takes place during squatting and not in passage to the defecation spot (Johns Hopkins [1970]), we take the number of larvae penetrating an individual to be a linear function of the areal density of infective larvae.

The total number of larvae penetrating hosts daily is, then, $\xi\ (\lambda P).L.$, where L is the areal density of infective larvae in

the soil, λP is the population defecating in "suitable soil", and ξ is the penetration factor, which gives the number of larvae which would penetrate one person who uses the "suitable" area in one day if the areal density of infective larvae is one per square foot.

4.5.6. WORM MATURATION

After penetration of the skin, the infective hookworm larvae "pass from the skin via the lymphatics and blood stream to the lungs, enter the alveoli, migrate up the trachea to the pharynx, are swallowed, and reach the small intestine where they attach to the intestinal wall [and] develop to maturity " (American Public Health Association [1960]) It is this process of migration and maturation which we wish to examine here.

A large number of the larvae which penetrate the skin do not attain maturity due to both passive and acquired host antagonisms. Further discussion of the role of acquired immunity is deferred to a later section of this paper. In this section we wish to present those data which are available on worm maturation.

We wish to estimate what fraction of the larvae which penetrate the skin will develop to maturity. Sarles has shown that under loads of about 100 worms per dog, an average of 49% (with considerable individual variability) of the larvae mature (Sarles [1929]), while Chandler (1929) quotes results showing that less than 4% mature in dogs subjected to a dose of 27,000 infective larvae. Otto's experiments (quoted in Stoll [1962])on human hookworm indicate that for a total innoculation of 10,000 larvae about 3% of the worms establish themselves, while with a gradually applied dose of 1000 larvae, 30% of these develop to maturity. In this latter case (which still gives

a fairly high established infestation of 300 worms) no immunity was in evidence. An estimate of the percentage maturation can also be derived from Kendrick's data (presented earlier in Tables 4.2 and 4.3). If we assume that 12 EPG and 26 EPG represent, respectively, one N. americanus and one A. duodenale worm, and if we assume that the peak worm load gives an estimate of the maximum number of worms established, then Table 4.6 below emerges:

	Case Number								
	Ancylostosome Group					Necator Group			
	A18	A20	A11	A8	A3	N2	N3	N5	N1
# Larvae	200	500	500	200	400	200	200	200	200
Peak EPG	1870	7570	10150	3670	2630	800	970	400	366
Peak Worm Load	72	344	391	141	99	67	81	33	30
% Maturation	35	69	78	70	25	33	40	16	15

Table 4.6. Percentage Worm Maturation from Kendall's Data

The mean ($\bar{x} = \frac{1}{n}\Sigma x_i$) percentage maturation is 42%. The standard deviation ($\sigma = \sqrt{\Sigma(x_i - \bar{x})^2/(n-1)}$) of 24% indicates that the individual variability is large.

On this basis we assume that at a worm load of about 100 worms, about 40% of the infective larvae which penetrate the skin will develop to maturity. (In a later section of this paper on immunity we will examine the possible effects of increased worm loads on the percentage of penetrated larvae which will

reach maturation.)

In much of the literature on larval development the time to maturation is seen as a constant. Chandler, in his major work (1929) and Otto, much later (1965), assumed that reproduction begins about 6 weeks after infection, while Beaver (1961) estimated the time lag to be about 60 days. From empirical studies, such as that of Kendrick (1934), it is abundantly clear that this constant lag is too simple an explanation. In a later work Chandler (1935) noted the inconsistency of the data emerging from the empirical work of Kendrick (1934) and Maplestone (1952) with the above hypothesis and expressed the belief that "a longer time than was hitherto believed is required for hookworms to reach the height of their egg laying powers."

Egg counts in areas where A. duodenale is endemic (Maplestone [1932], Roche [1966], Schad [1973]) have exhibited a marked seasonal variation. Roche and Layrisse (1966) have pointed out that this seasonal phenomenon is apparently restricted to areas where either "Old World" or "Old World"&"New World" hookworm occur, but that it has not been observed in endemic areas where necator is the exclusive hookworm species. Consideration of this seasonal phenomenon in West Bengal led Schad and his co-workers at the Calcutta School of Tropical Hygiene and Johns Hopkins to examine the process of larval maturation more closely. The results of their work, which is not yet complete, are of fundamental importance in understanding the life cycle of

A. duodenale.

In their epidemiological study of hookworm in West Bengal, Schad et. al. (1973) observed that "..larvae acquired during the rainy season of one year appear to remain dormant until just before the monsoon of the following year when they resume their development and mature." This seasonal dormancy, which is known to exist for some other families of nematodes, is seen as an adaptation to an environment which is seasonally unfavourable for the development of the free- living stages.

In a later paper an alternative explanation is posited. Nawalinski's (1974) examination of a self-induced hookworm infection corroborated the above evidence. He pointed out that the data gleaned from this infection did not enable him to distinguish between the hypotheses i) that the strain had an abnormal prepatent period, and ii) that the prepatent period is environmentally sensitive. While the former hypothesis would be easier to incorporate into a model, the earlier evidence (Schad [1973]) suggests that the latter is the more likely explanation and it is this relationship which will be used here.

Schad et al. (1973) briefly report the course of a self-induced A. duodenale infection. In Figure 4.18 overleaf, these data are plotted and a straight line drawn through the four data points.

The course of the egg output, up to peak output, from Nawalinski's (1974) self-induced infection, is presented on Figure 4.19 overleaf. The data unfortunately do not indicate in what season the increase in egg output took place. A regression line has been fitted to the data recorded after the egg count became positive (see Appendix 4.3 and Figure 4.19). From this regression line the time lag between the onset of ovideposition and the peak egg load is a little over 4 months.

Kendrick's (1934) interest was in the loss of hookworms and, in most cases, he did not record egg counts for the full period prior to the attaining of peak output. The data for the two cases in which monitoring was apparently instituted shortly after the onset of egg deposition are reproduced on Figure 4.20 overleaf.

It appears that in both cases eggs were first passed in about November 1926, about six months after inoculation, and that egg production increased more or less linearly thereafter, with a peak output being reached eight to twelve months after deposition of the first eggs. It should be noted that during this period of increasing egg output we would expect a significant number of worms to be expelled from the host. Complete

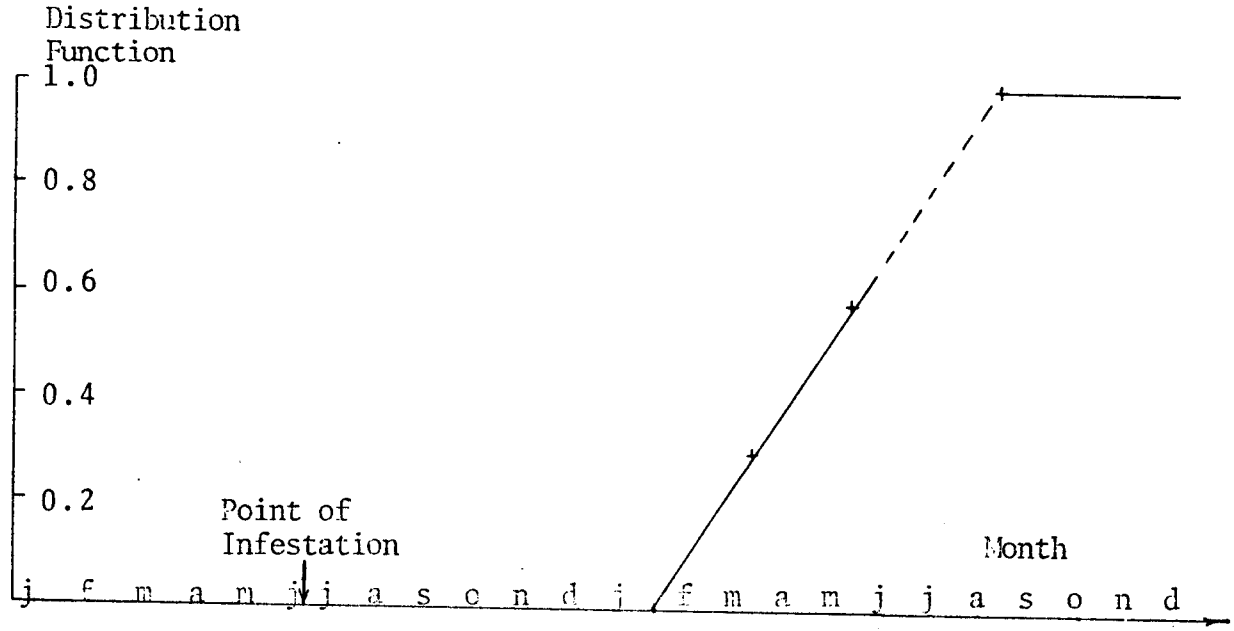


Figure 4.18: Worm Maturation Curve from Schad's Data

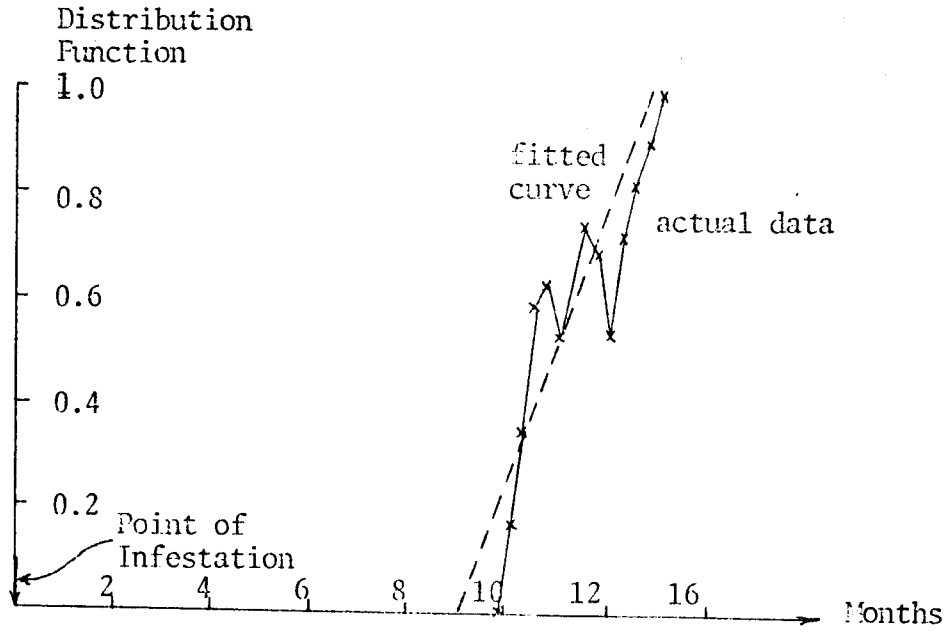


Figure 4.19: Worm Maturation Curve from Nawalinski's Data

Figure 4.20: Egg Counts from Kendrick's Data

maturation, therefore, may take an even longer time.

On the basis of the above data we cannot add much to the work of Schad and his colleagues except to affirm that this analysis gives results consistent with their hypothesis on delayed maturation.

For purposes of model calibration,we will use Schad's data (Figure 4.18 above) since these results give the most precise onset of maturation date and give a pace of maturation less than that emerging from Nawalinski's data, but greater than that implicit in Kendrick's analysis. Specifically we will assume the following: There is a minimum prepatent period of 60 days. If this period ends before February first, then linear maturation starts on February 1 and ends six months later on July 31; if the minimum prepatency period ends after February 1, the larva will remain dormant until the following February 1, when maturation of the susceptible larvae will take place as described above.

The above formulation is a simple deterministic formulation of the maturation phenomenon and seems appropriate given the nature of the epidemiological model to be developed. To indicate the directions in which we would think in formulating a more sophisticated model we have developed a more interesting stochastic, composite maturation - worm loss function in Appendix 4.10.

4.5.7. COMMUNITY HOOKWORM LOAD

A fundamental concept in helminthology is that the measure of infectivity for helminthic diseases is the number of parasites harboured, whereas in most other infections, it is the number of infected hosts (Macdonald [1965]). This distinction has not always been apparent to epidemiologists and thus many of the studies on the community hookworm load focus on the traditional measures of incidence and prevalence.

It has been estimated that nearly a quarter of the world's population is infected with hookworm (Otto [1965]). A few representative estimates of hookworm prevalence rate on the Indian subcontinent are given on Table 4.7, below:

POPULATION	PREVALENCE	DATE	INTENSITY	SOURCE
Two districts in West Pakistan	30%	1961	no counts above 2000 EPG	Beaver (1961)
Central UP villages	30.7%	1972	--	Prasad (1972)
Rural UP	59%	1965	--	PRAI (1969)
Rural West Bengal	78%	1968	Infection Index = 247	Choudhury (1968)
Rural Bengal	89%	1928	Avg EPG=244	Chandler (1929)
Bengal tea estates	95%	1928	Avg EPG=2300	Chandler (1929)
Rural Tamil Nadu	93%	1928	Avg EPG=1760	Chandler (1929)
Rural Tamil Nadu General Population	56%	1968	--	Daniel (1968)
Scavengers	90%	1968	--	"
Rural Tamil Nadu	60%	1967	--	Pisharoti (1967)
Rural West Bengal	90%	1968	Avg. Worm Load about 110/cap	Johns Hopkins CMRT (1970)

Table 4.7. Prevalence of Hookworm on the Indian Sub-Continent

In the analysis which follows we will use the data given by the Johns Hopkins team since this seems to be both the most recent and the most reliable, and gives the community measure - the mean worm load - which we wish to use.

(In Appendix 4.11, the relationships between the different measures, viz. the infection index, the prevalence and the mean worm load, are investigated in some detail. The objective of this analysis was to be able to infer the mean worm load from the other statistics which are more frequently reported. The outcome is that the confidence limits on the estimates are likely to be wide and it has been decided to use only the Johns Hopkins mean worm load data in the remainder of the present analysis.)

4.5.8. IMMUNITY TO HOOKWORM INFESTATION

In infections in which superinfection is the rule and in which the force of infection is related to the level of infestation in the community, endemicity is dependent on the capacity of the host to resist the continuous exposure to infection. In this paper the emphasis is on the mathematical demonstration of the necessity of acquired immunity. We wish to draw attention here to similar conclusions on the central epidemiological role of hookworm immunity which have been arrived at in a less formal, more empirical way by experienced helminthologists.

Cort and Otto (1940) opined that "specific host immunity seems to be the condition which limits the level of infection;" Stoll (1962) has inferred that "the host must biologically deal with (this reinfection) protectively, or it is in trouble from the pathogenic effects of the cumulative load of worms;" Schad (1975) has hypothesized that the low worm burden in the average Bengali villager is a consequence of an effective host response to "trickle infection;" Soulsby (1975) has suggested that "the ability of an individual to control his hookworm infection by immunological means may be a determinant of the hookworm burden that is established in an endemic area;" and Foy (1963) concludes that "epidemiological evidence suggests that the hookworm disease would be even more serious in hyperendemic areas if it were not for some degree of immunity preventing

overwhelming infections."

In view of this central epidemiological role, it is surprising to find that the study of immunity in helminthiases has, relative to diseases of bacterial origin, been comparatively neglected. While inferences on human helminthic immunity have been made from studies of comparable nematodes in dogs, sheep and rats, there is "no clear evidence that man develops an immunity, although circumstantial evidence would tend to suggest that he does " (Miller [1968]) This lack of direct evidence should be borne in mind in using the acquired immunity relationship which will be hypothesized.

The effects possible of the immune response to hookworms include:

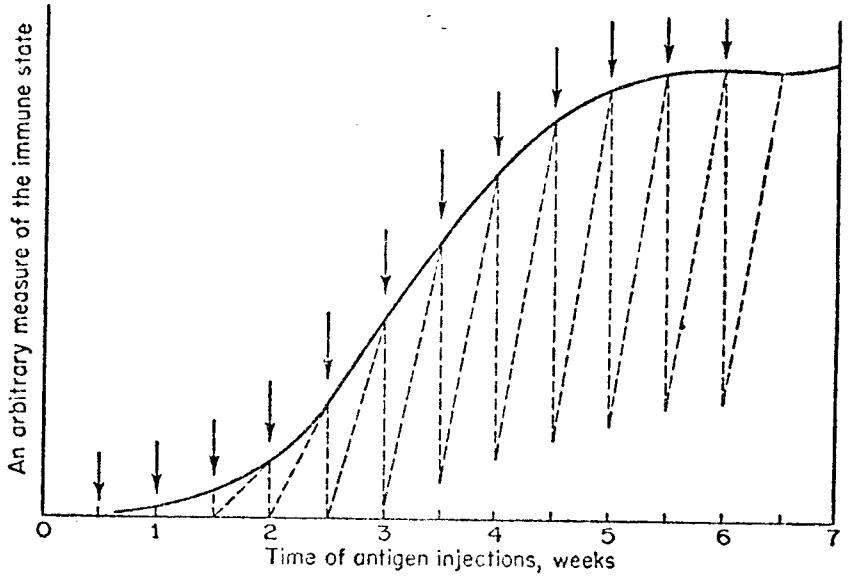
i) Reducing the invasive potential of infective larvae: Immobilization and destruction of migrating larvae in repeatedly infected cases is accomplished by the blocking of the body orifices of the larvae by immune precipitates (Chandler [1929]).

ii) Interference with the functioning of the worms in the intestine: This phenomenon includes simple expulsion of worms and changes in feeding, metabolism, behaviour and reproduction of the parasite (Dobson [1972]).

iii) Resistance to the injurious effects of the worms: This resistance may include walling off of the parasites, neutralization of toxic products, repair of mechanical damage and replacement of blood and other substances extracted by

the parasites (Chandler [1948]).

We may consider the immune response to intestinal helminths to consist of both passive and active antagonisms. Passive antagonism is related to intrinsic population characteristics such as the genetic structure and aging physiology (Dobson[1942]). The form of the acquired immunity function is assumed to be that suggested by Cushing and Campbell (1957) in Figure 4.21 below.



The general immune response of an animal to multiple injections of a foreign antigen. (Arrows represent injections.) Solid line represents the trend in the increase of the general immune response. Dotted lines show the usual transitory fall in the immune state due to the presence of antigen for a few hours after injection.

Figure 4.21: Immune Response Function (from Cushing and Campbell)

In the models which will be developed we will consider acquired immunity to manifest itself by affecting either the rate at which worms are expelled from the intestine, or the proportion of invading larvae which will develop to maturity in the human host.

The former relationship has been examined earlier in this paper. The proportion of worm load w which is expelled daily is:

$$\frac{\Delta w}{w} = d_2(w) = 0.000449e^{.0186w}$$

We will now examine the second hypothesized relationship.

The literature suggests that the antigens responsible for the mobilization of an immune response to migrating larvae (though the stimulation of antibody formation) are related to the presence both of worms in the intestine and of the excretions of the larval stages (see Cort and Otto [1940] and Dobson [1972]). Helminthologists (see Stoll [1962], Cort and Otto [1940], Dobson [1972]) stress the necessity of the continued incoming of worms for the continuous mobilization of the immune response which is necessary to secure the full spectrum of antagonistic effects.

In developing a mathematical formulation for the immune response to migrating larvae in the host, these factors have been specifically included by assuming that: (i) The immune response determines the proportion of invading larvae which will develop into mature, normally-functioning worms; (ii) The form

of the function (suggested by the shape of Figure 4.21) adopted in this analysis is the logistic curve (familiar to demographers); (iii) A surrogate for the antigen level in the body is the sum of the larvae which are migrating in the host and the intestinal worms.

On the basis of Beaver's data on worm maturation presented earlier in this chapter we assume that at an endemic mean load of about 100 worms about 40% of the infective larvae which penetrate the skin develop to maturity. We will further assume (and this is no more than a guess which is plausible both in terms of the data presented in the section on worm maturation and in terms of the immune function emerging) that due to passive antagonism, 70% (or 90% - both cases will be examined) of the infective larvae will develop to maturity when there are no other larvae or worms in the host. In analysing the epidemiological model later we find that the ratio of mature worms to infective larvae in the host averages about 1:2.25. One hundred worms, therefore, corresponds to about 325 worms in either the mature or larval stage.

The logistic curve is the solution of the differential equation

$$\frac{dN}{dt} = r_o N(1-\frac{N}{K}) \qquad \text{with initial condition } N = N_o$$

at t=0. The solution is:

$$N = \frac{N_o e^{r_o t}}{[1+\frac{N_o}{K}(e^{r_o t}-1)]}$$

Under the above assumptions and the assumption that K = 1 (total

immunity at high worm load) we have:

$$I(w+\ell) = \frac{0.3e^{.00385(w+\ell)}}{1+0.3(e^{.00385(w+\ell)}-1)} \qquad \text{(for 70\% case)}$$

and

$$I(w+\ell) = \frac{0.1e^{.00800(w+\ell)}}{1+0.1(e^{.00800(w+\ell)}-1)} \qquad \text{(for 90\% case)}$$

where $I(w+\ell)$ is the proportion of viable larvae not reaching maturity,

w = the number of mature worms,

and ℓ = the number of infective larvae.

These hypothesized immunity functions are plotted on Figure 4.22 below.

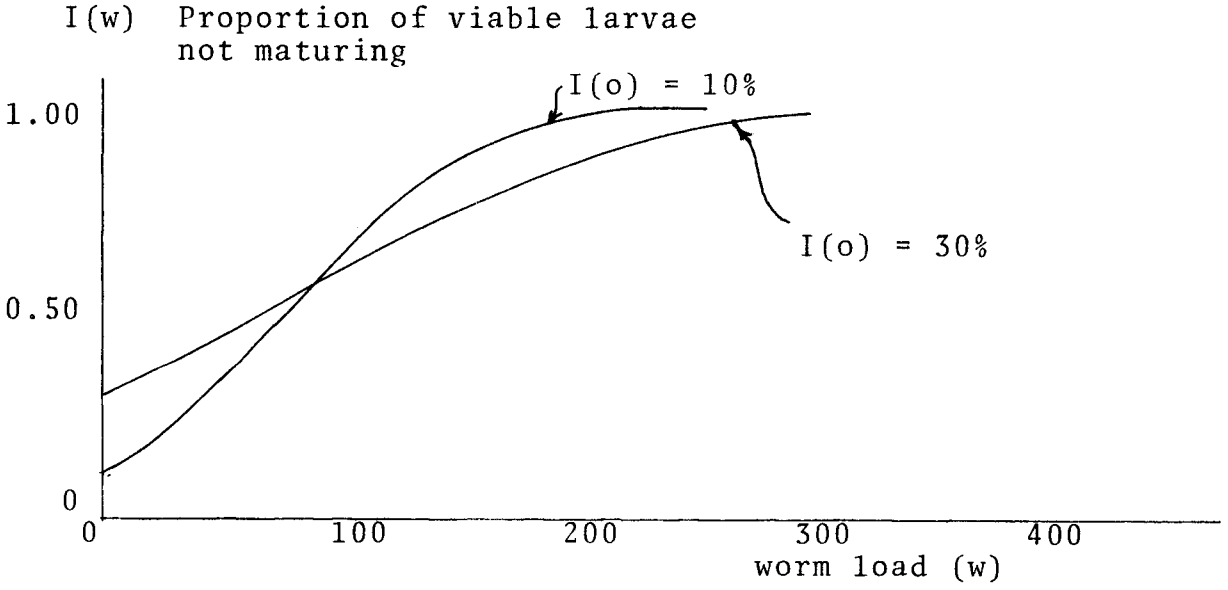


Figure 4.22. Assumed Immunity Function

In conclusion we note that any severe stress to which the host is subjected may have a profoundly beneficial effect on these helminths by interfering with the mobilization of the immune

response (Chandler [1948]). The most obvious of these in the context of hookworm are dietary conditions and anemia (Cort and Otto [1940]).

4.6. RESULTS

4.6.1. MODEL 1

4.6.1.1. Simulation Model 1

In this first model we assume that the proportion of penetrated larvae which develop to maturity in the human host is dependent on the degree to which the immune response of the host is mobilized. We further assume that this is the only way in which the immune response is manifested. We assume, too, that the size of the defecation area remains constant and that the number of larvae penetrating the host is a function of the areal density of infective larvae. (This accords with the assumption made by the Johns Hopkins team which investigated hookworm in West Bengal that "most transmission occurs when man becomes stationary in polluted areas."(Johns Hopkins [1970])

On the basis of the model structure and difference equations presented earlier, a Fortran program was written to simulate the epidemiological consequences of the hypothesized functional forms and parameter values.

i) Endemic Levels

In the first runs of this model the parameter representing the rate at which infective larvae penetrate the human host was varied over a wide range. For all of the parameter values above a certain critical value (the significance of which will be

discussed later), a positive equilibrium hookworm load was attained. A plot of the endemic mean hookworm load versus the value of the "penetration factor" in the favourable season is given on Figure 4.23 below:

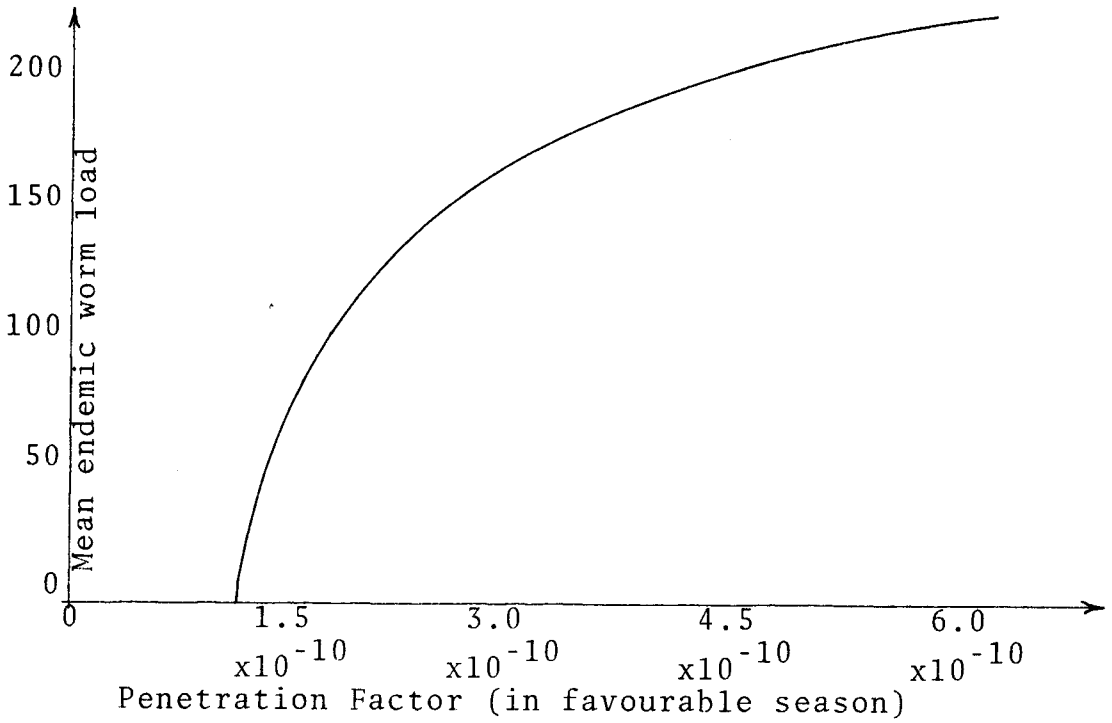


Figure 4.23. Effect of Penetration Factor of Endemic Worm Load

In the analysis which follows we will assume that the value of the penetration factor is 6.6×10^{-10} in the four favourable months and 1.65×10^{-10} in other months. The endemic mean load of about 100 worms is the same as that given as a "moderate world average" by Stoll (1962) and is approximately equal to the mean worm load found by Schad, et al. (1975) in rural West Bengal.

ii) Examination of Analytic Results:

In an earlier section of this chapter we hypothesized an immune reaction function of:

$$I(w+\ell) = \frac{0.3e^{\alpha(w+\ell)}}{1+0.3(e^{\alpha(w+\ell)}-1)}$$

whence

$$w^* = \frac{1}{3.25\alpha} \ln \frac{0.7}{0.3} \left(\frac{\beta}{\phi/3.25} - 1\right) \qquad (1)$$

We will now examine the results of the simulation model in the light of the much simpler analytic model. For any endemic mean worm load there is an implicit "equivalent β"(β̃) which may be calculated from equation (1) above. The β̃'s for some of the penetration factors used in Figure 4.19 are plotted against these factors in Figure 4.24 below.

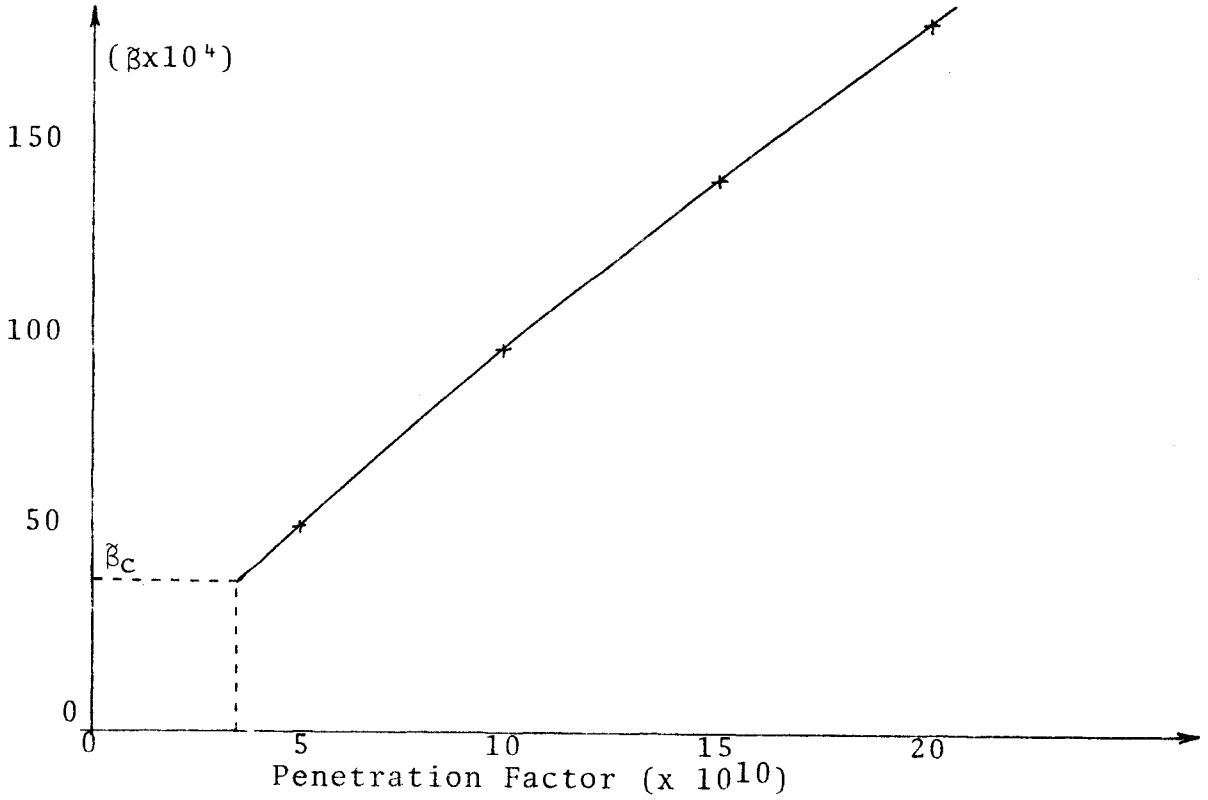


Figure 4.24. Relationship between Penetration Factor and "Equivalent β" Parameter

We see that although the complexity and seasonality introduced into the simulation model have altered the linearity of the relationship somewhat, the analytic form gives an approximation which may generally be satisfactory.

Implicit in the analytic form (see equation (1)) is a critical value of β - if β is reduced to or below β_c hookworm will eventually be eliminated. [Note that the time taken for effective elimination will depend on $\beta_c - \beta$. Unfortunately, the integral equation which would give the approach to equilibrium over time, viz.

$$\int \frac{(0.7+0.3e^{\alpha w})\ dw}{w(0.7[\beta-\phi] - 0.3\phi e^{\alpha w})} - \int dt = C$$

with boundary conditions $w = w_o$ at $t = 0$, has no analytic solution, and an "approach function" such as that given earlier for the other classes of diseases is not available. It is possible to solve the differential equation numerically for different values of w_o, but this was not deemed to be worthwhile.]

The critical value of β is defined by:

$$\frac{0.7}{0.3}\left(\frac{\beta_c}{\phi/3.25} - 1\right) = 1$$

whence $\beta_c = \frac{\phi}{2.275} = 35 \times 10^{-4}$ for the daily worm loss rate ($\phi = 0.008$) used in this analysis. (We see that if we plot the critical value of the penetration factor from Figure 4.23 onto the extrapolation of the curve of Figure 4.24 we get, as we should, $\beta_c \simeq 35 \times 10^{-4}$.)

We conclude, then, that the relationship between β and w in the simple analytic model is substantially the same as that relating β and w in the more complex simulation model. The analytic model can be useful, therefore, in interpreting the results of the simulation model.

iii) The Effect of Excreta Disposal Practices

a) Brief Literature Review:

Choudhury and Schiller (1968) have given a description of the environmental sanitation situation in West Bengal:

> Facilities for the disposal of human excreta were almost non-existent. The members of each household habitually visited the same defecation sites, which usually were small thickets of bamboo or other trees and shrubs near the compound. Other defecation areas, such as the sloping banks of tanks or ditches, dry stream-beds, corners of cultivated fields, or sides of roads or paths also were commonly used. Because of the practice of ablution, defecation sites were determined, in part, by the availability of water for this purpose.

The Johns Hopkins Center for Medical Research and Training (1970) team has related defecation practices to hookworm infestation.

> Most fecal pollution occurs in recognised areas, which are considered unclean and are avoided except during defecation... Activities associated with defecation provide the maximum regular exposure to hookworm infested soil since polluted areas are rarely visited for other purposes...(We assume) that most transmission occurs when man becomes stationary in polluted areas.

This relationship between unsanitary conditions in traditional

defecation areas and hookworm infestation is accepted by other authorities. Otto (1965) speaks of the "sharp correlation (of hookworm infection) with those human habits which concentrate feces in moist shaded places which are repeatedly visited." Beaver (1961) has studied the age, sex and occupational correlates of hookworm infestation and has concluded that "in places, such as India, where the fields are of minor importance,... the infestations are derived primarily from regularly visited, heavily polluted defecation sites near the villages." Most helminthologists who have worked in India (eg. Chandler (1929), Choudhury and Schiller (1968), Rao and Sen (1969), Schad (1975)) have come to similar conclusions.

Despite the unanimity on the role of unsanitary defecation habits, data on the effect of improved excreta disposal practices on hookworm infestation are poor. Some of these data are summarized in Table 4.8 overleaf.

Other data relevant to this issue are:

India: Anwikar (undated) found that in two groups which were identical in all measures other than occupation, 64% of sewage farmers tested positive for A. duodenale while 40% of other agricultural workers tested positive.

India: Beaver (1961) claims that in Assam a family using a heavily polluted defecation area had an average of 4200 EPG while a similar isolated family which defecated in a different area each day had an average count of 260 EPG.

STATE	SOURCE	PREVALENCE without latrines	PREVALENCE with latrines	REMARKS
Peru	WHO (1973)	99%	58%	Intestinal parasite in children, four years after start of excreta disposal program.
Bengal	Patel (1954)	29%	9.5%	In urban areas.
Puerto Rico	Cort in Patel (1954)	?	0%	Total reduction in 20 years in small area due to sanitary measures.
U.P. India	P.R.A.I. (1969)	100%	79% (after 2 years) 48% (after 3 years)	Only those who tested positive (59% of the sample) and adopted latrine use (50% of population) were followed.
U.P. India	P.R.A.I. (1969)	80%	22% (latrine users) 31% (non-users)	Seven years after start of latrine program which covered 35% of village.
Egypt	Chandler (1954)	17% (females) 42% (males)	7% (females) 12% (males)	Experimental village had improved excreta disposal and water supply.
Panama	Cort (1929)	P%	0.30P%	After anthelmintic treatment in populations with good and bad sanitation.
Panama	Cort (1929)	P% Q%	0.70P% (males) 0.40Q% (females)	Another village in above study.
Bengal, India	Hare (1940)	82%	73%	One year after installing one latrine per family.

Table 4.8. Empirical Data on Effect of Latrine Use on Hookworm Prevalence

b) Simulation Results:

In attempting to simulate the effects of altered defecation patterns on hookworm endemicity we assume that those using latrines will neither contribute any ova to the "egg pool" nor be exposed to the infective larvae emanating from this pool. The exposure of those continuing to use the traditional defecation areas is thus reduced as a result of latrine use by their fellow villagers. Thus if we consider a village with one thousand inhabitants and if we assume that 25% of the population start using latrines, we have effectively divided the population into one group of 250 who will lose their worms in accordance with the specified expulsion function, and another group of 750 who will continue to be exposed to those larvae which develop in the traditional defecation areas. The helminth dynamics in the traditional group are precisely those which would pertain if the total population were exposed to a penetration factor 25% lower.

(This equivalence arises from the assumptions that defecation area remains constant and that there are no density dependent effects in the soil. If we assume that the population is reduced by 25%, the number of larvae to which an individual (in the 750) is exposed is reduced by 25%. This is the only effect due to the nature of the assumptions. Another way of reducing the exposure of an individual by 25% is to reduce the penetration factor by 25%.)

On Figure 4.25 (overleaf) the time stream of the worm load in

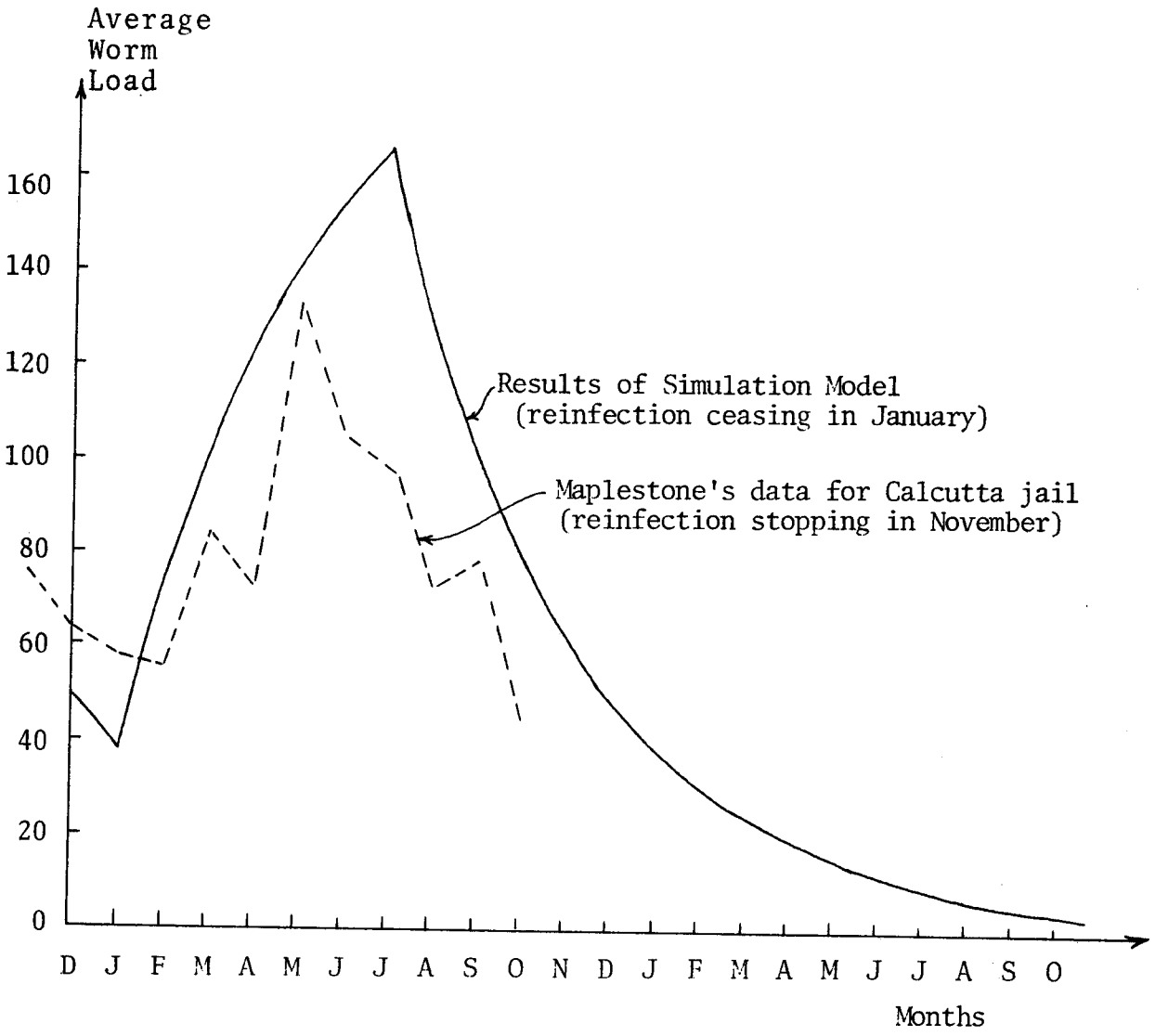


Figure 4.25: Average Worm Load for those Using Latrines

the latrine-using community is given. We note the similarity of the form of this function to the worm loss functions in Indian jails (as presented by Chandler [1935]) and plotted on Figure 4.25 for a period of one year after cessation of infection.

On Figure 4.26 (overleaf) the time stream of the worm load for those who do not use latrines is plotted. (Note that the data for Figure 4.26 are annual averages and thus the figure should present histograms rather than continuous curves. An idea of the seasonal variation masked in Figure 4.26 may be gleaned by comparing the worm loss curve on Figure 4.25 with the equivalent curve on Figure 4.26.)

A few points to note from Figure 4.26 are: There are considerable externalities accruing to non-users; the mean endemic load in the non-users falls faster than the percentage using latrines (see also Figure 4.23); if more than about 43% of the population use latrines regularly, this model suggests that hookworm will, ceteris paribus, ultimately be eradicated; the greater the number of latrine users the more rapid the eradication (for more than 43% users); for those using latrines (and never contacting the infective larval sources) hookworm is effectively eradicated in a couple of years; those who use latrines for a while and then return to traditional defecation habits will derive but a transitory benefit from latrine use.

These results must be interpreted with caution. From Table

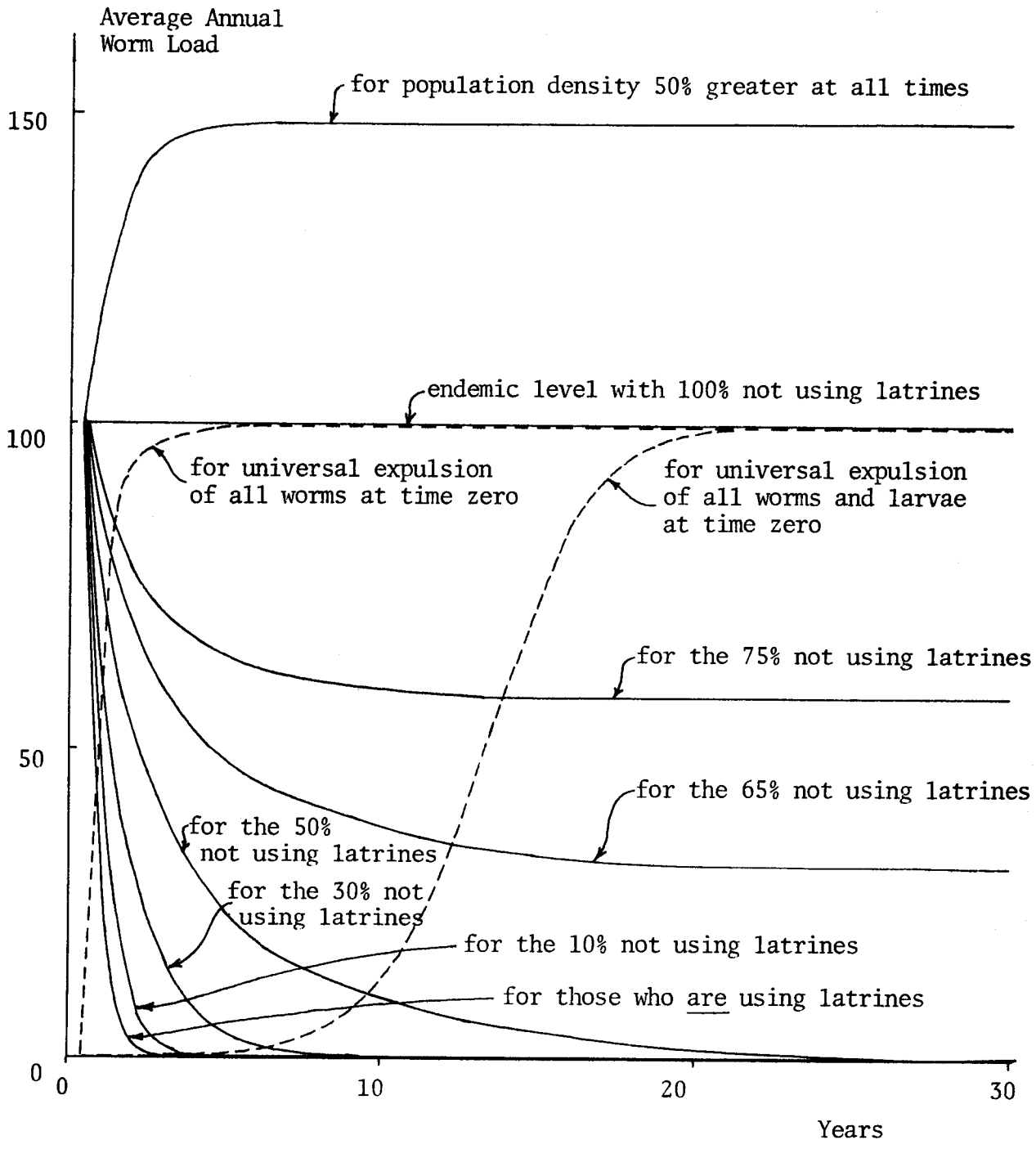


Figure 4.26: The Epidemiological Effects of Different Hookworm Control Programs (for Model I)

4.8 it is evident that the data required to calibrate the model for a situation in which latrine use led to reduced hookworm infestation were not found. Particular care should be exercised in drawing inferences about eradication from this and the other models presented. Migration - an important factor mitigating against species eradication in mainland ecosystems - is not included, the values of some parameters are probably not applicable under low endemic loads (see, especially, the derivation of the worm loss constant earlier), and the omission of consideration of infestation sources other than the focal sources is likely to be more serious at low endemic loads.

iv) Effect of Anthelmintics

The model was used to simulate the effect of the universal administration of a totally effective anthelmintic (the new anthelmintics are actually likely to be about 90% effective - Schad, personal communication). The resulting annual mean load is plotted on Figure 4.26. The endemic level is unaffected and is attained within about four years of administration of the drug. (The drug is admittedly administered in January, which is the time of lowest mean load. Just before the monsoon would be a more auspicious time for such a program to be undertaken.) These results accord with those of Cort et al. in Panama (1929) where they found little residual effect in two villages without adequate excreta disposal programs three and four years after universal treatment.

The administration of an anthelmintic does little more, then, than put a brief episode of low infestation in the lives of those in endemic areas.

The village dynamics after administration of a (hypothetical) drug which eliminates both the established worms and the migrating larvae in the human host were also investigated. In this case (see Figure 4.26) "recovery" is a great deal less rapid.

v) Seasonality

A characteristic of hookworm infestation which has been a subject of interest to parasitologists is the observed seasonal variation of the worm load. It is of interest to compare the seasonal variation emerging from the model with observed variations under endemic conditions. In the simulation model we distinguished between the probabilities of destruction and survival of ova and larvae, and the penetration and maturation of infective larvae in different seasons. The resultant seasonal variation in the community worm load is compared with seasonal variations given by Chandler (1954) and Schad (1975) on Figure 4.27 (overleaf). The model appears to simulate the seasonal variation in infestation adequately.

On Figure 4.28 the variation over the year in the number of infective larvae in the soil is compared using Schad's data and those arising from the simulation model. Although annual variation is clearly wide, the model seems to reproduce the broad features of the seasonal effect adequately.

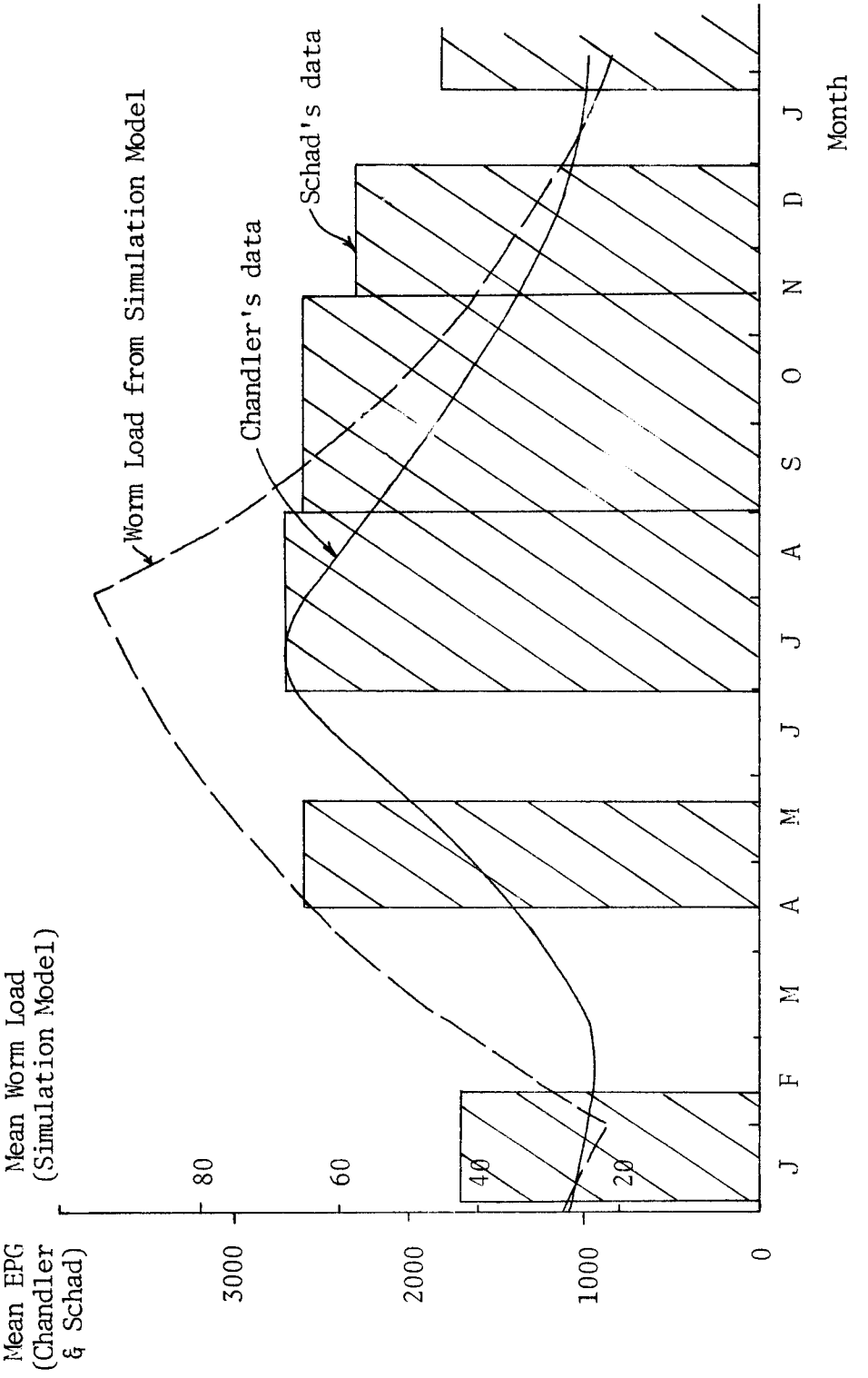


Figure 4.27: Seasonal Variation in Hookworm Infestation

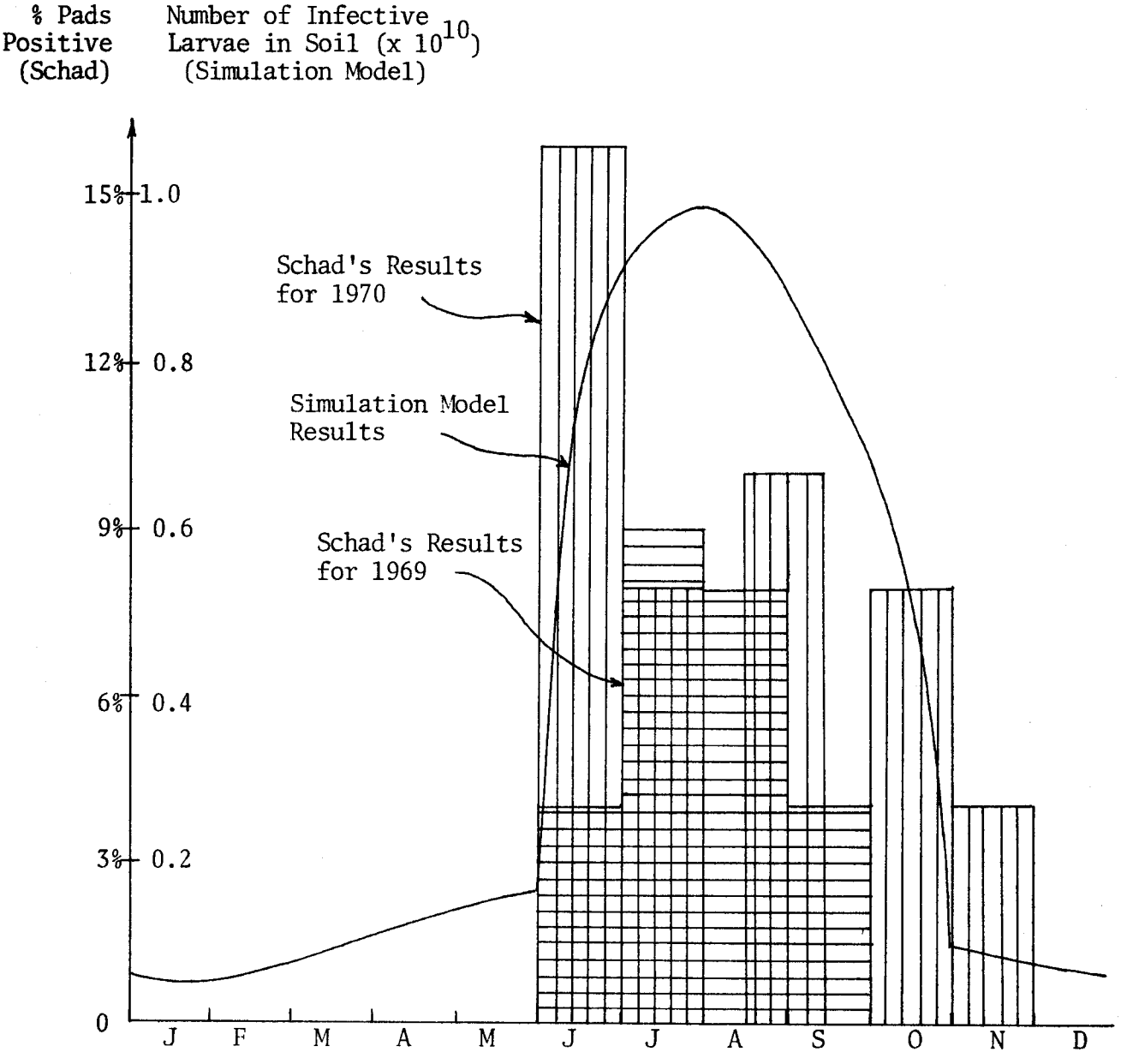


Figure 4.28: Seasonal Variation in Larvae in Soil

4.6.1.2. Overyear Model and Sensitivity Analysis of Model I

Considerable uncertainty is associated with the estimates of many of the parameters in the model. A one-season, deterministic, two-state model (see Figure 4.29 below) was developed for examining the sensitivity of the system to changes in the parameters, and for assessing the effects of stochastic variation.

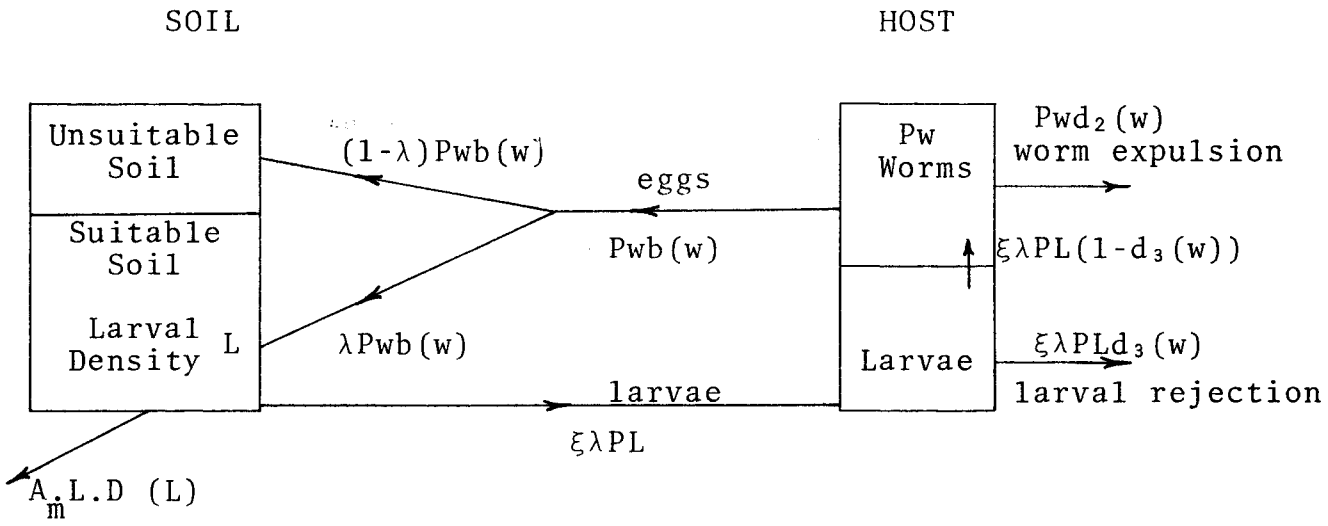


Figure 4.29. One-Season, Two-State Deterministic Hookworm Model I

[Note that in the above figure we have assumed that the number of larvae penetrating the human host is proportional to the product of the number of people using that part of the traditional defecation area which has "suitable soil" (λP) and the density of infective larvae (L). This assumption is consistent with that made in the simulation model.]

In structural terms the differences between this model and the simulation model are: seasonality and dormancy are not considered,

the distinction between eggs and infective larvae is not drawn; the size of the defecation area is explicitly included. More important is the inclusion of functional forms which facilitate incorporation of more sophisticated biological relationships. Instead of assuming that "fitness" $(\frac{1}{z}\frac{dz}{dt})$ is constant we assume that the rate of increase of an organism responds to the population density (or food density). The following density-dependent relationships are proposed for inclusion in the model:

$d_1(L) = \alpha_1 e^{\beta_1 L}$ -the death rate of eggs and larvae in the defecation area. The non-linearity may be due to soil heterogeneity or competition for food.

$d_2(w) = \alpha_2 d^{\beta_2 w}$ -rate of expulsion of larvae from the host is a function of the worm load in the host.

$d_3(w) = \frac{\alpha_3 e^{\beta_3 w}}{1+\alpha_3(e^{\beta_3 w}-1)}$ -proportion of penetrated larvae which develop to maturity is a function of the host worm load.

$b_2(w) = \gamma e^{-\delta w}$ the egg production per worm is a decreasing function of the worm load.

The equations describing the system are:

Soil: $A\frac{dL}{dt} = \lambda.P.w.b_2(w) - A.L.d_1(L) - \xi\lambda PL$ (1)

Host: $P \frac{dw}{dt} = \xi.\lambda.PL[1-d_2(w)] - Pwd_2(w)$ (2)

Setting the above equations equal to zero (the steady state condition) and substituting in the overyear parameter values (see Appendix 4.12 for determination of these), we find:

$$d_3(w) = \frac{400}{P}$$

The critical value of P is $P_c = \frac{\alpha_1 \alpha_2 Am}{[\xi \lambda^2 \gamma (1-\alpha_3])}$.

For $\alpha_3 = 0.30$ and $\alpha_3 = 0.10$ the relationship between the population size, P and the mean endemic worm load, w, is given in Figure 4.30 below. (We note that the curve for $\alpha_3 = 0.30$ is equivalent to that given earlier of Figure 4.23.)

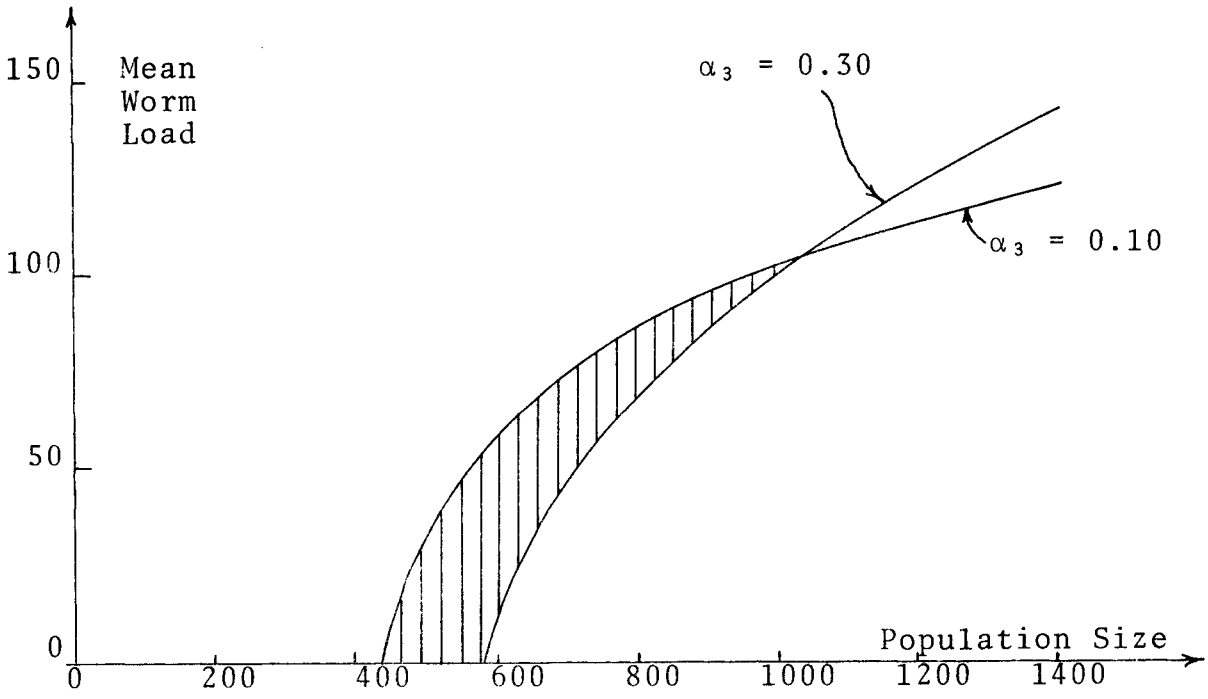


Figure 4.30. Relation Between Population using Traditional Defecation Area and Mean Worm Load in that Population (for Model I)

The vertical lines indicate that the differences in community worm load for cases in which less than 55% of the population uses latrines are considerable. The immune response function has to be specified more reliably before this model can be usefully used to predict changes in hookworm infestations resulting from different public health interventions.

The mean worm load changes would be less pronounced than those given above if:

i) "A" were reduced as a result of a smaller population using the traditional defecation area;

ii) The mean residence time in the soil were increased as a result of lower organic loading; and

iii) The rate of egg production per worm increases as worm load decreases.

4.6.2. Model 2

4.6.2.1. Simulation Model 2

The model structure is that of the first model and the parameters used are the same. In this case, however, host immunity is assumed to manifest itself by more rapid expulsion of worms from the intestine (rather than by prevention of larval maturation). This is more satisfactory in that better data are available for estimating β_2 than were available for estimating β_3 .

The output from this model is summarized in Figures 4.31, 4.32 and 4.33 (overleaf).

In deriving the worm loss function, β_2 is directly proportional to the inverse of the assumed initial worm load (w_0) in Chandler's data. (α_2 is unaffected by the initial load.) Since there is considerable uncertainty in the initial worm load in Chandler's population, we have examined the effect of changes in this load on the relationship between mean worm load and population size (see Appendix 4.13). The results are presented on Figure 4.32. We see that the effect of doubling β_2 is considerable; if we are to have any confidence in the results of the analysis, better data must be used for estimating the relationship between the worm expulsion rate and the worm load.

Comparing the results for Model 2 (Figures 4.31 and 4.32) with those for Model I (Figures 4.26 and 4.28) we note that:

i) The population size which will bring about eradication

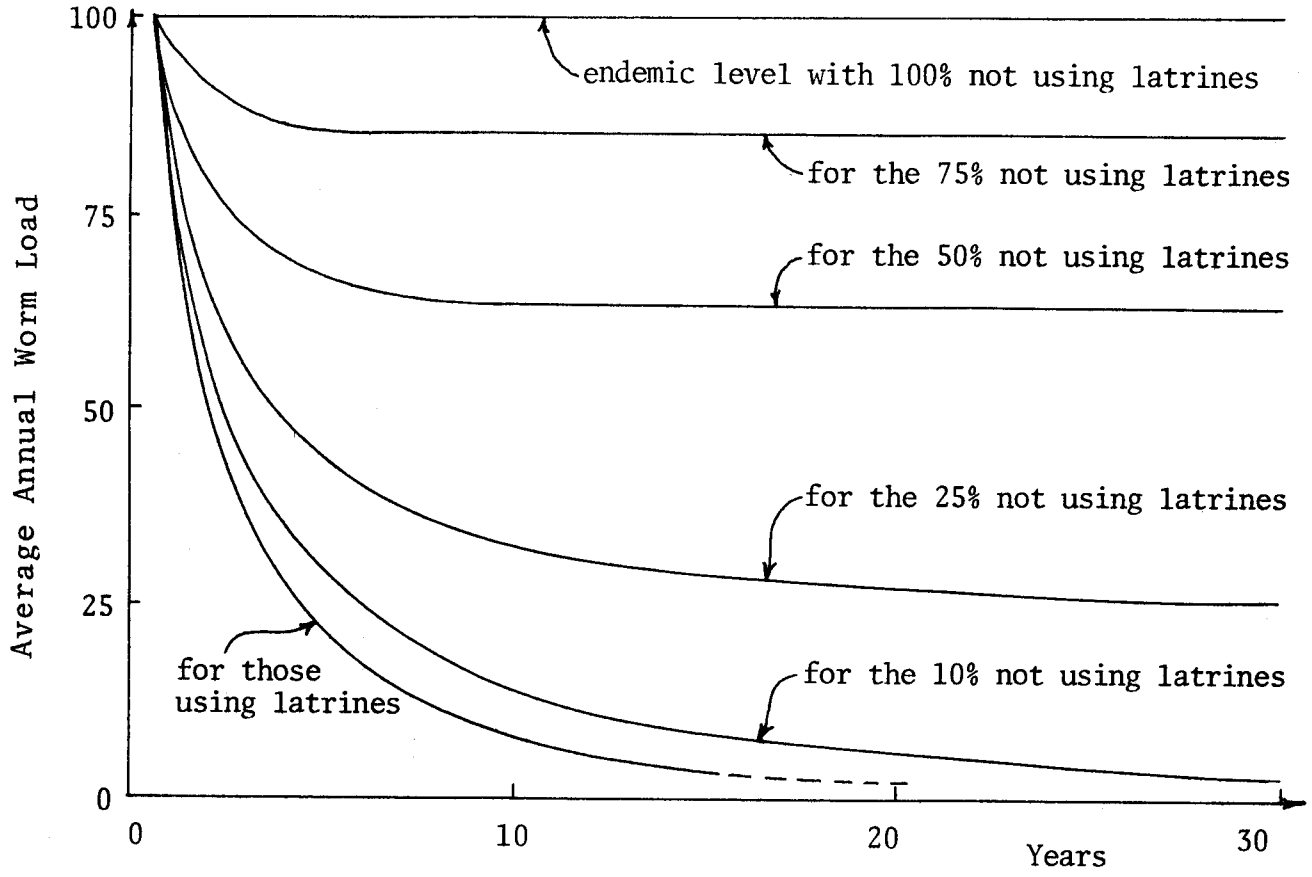


Figure 4.31: Effects of Different Hookworm Control Programs (for Model 2)

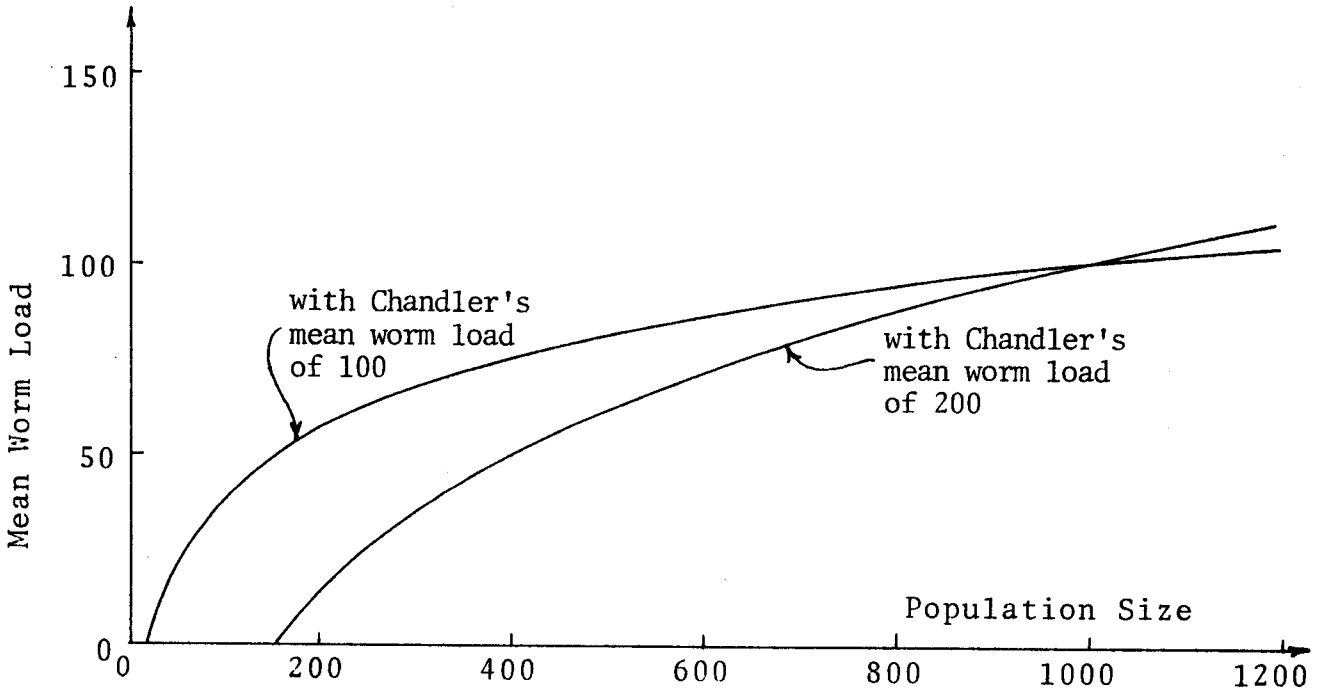


Figure 4.32: Relation between Population Size and Mean Hookworm Load (for Model 2)

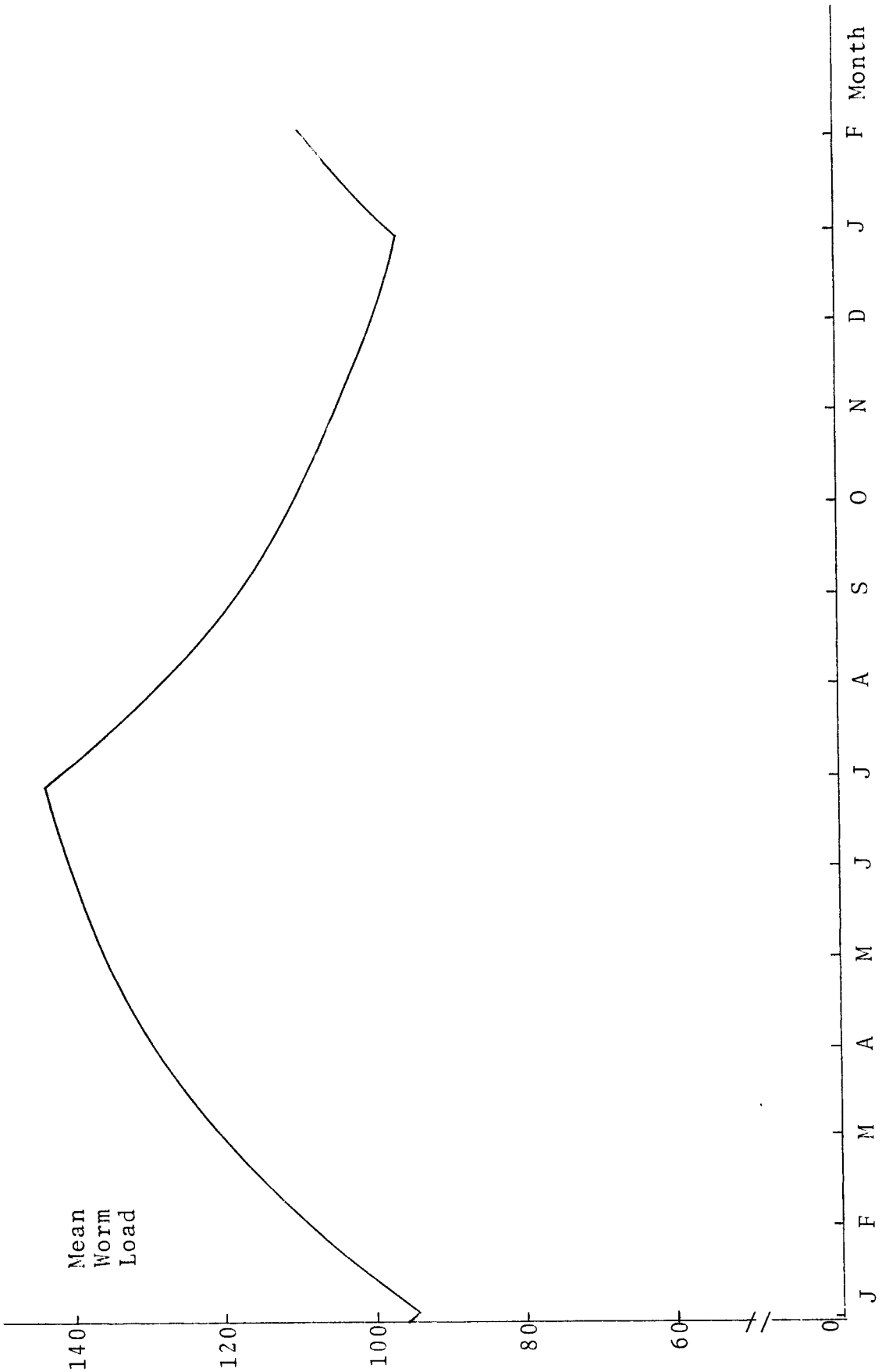


Figure 4.33. Seasonal Effects in Model 2

in the model (the critical population size) is considerably smaller in the second case;

ii) If 10% of the population use latrines, the reduction in worm load for the remaining 90% is about 6% for w_o=200, and 3% for w_o = 100, in the second model, and is 5% for α_3 = 0.10, and 18% for α_3 = 0.30, in the first model;

iii) The rate at which the equilibrium levels are approached is more gradual in the second case due to the increased persistence of worms at lower mean loads. The behaviour of the second model more closely approximates reality in this regard, and is on this basis considered the more useful of the two models.

Throughout this analysis we have assumed the system to be closed to migration. Biogeographers have shown species extinction to be much more probable on an island than on the mainland: on the mainland species migration is an important factor mitigating against extinction.

From Figure 4.31 we see that it takes about 0.67 years for the worm count to decline from 8 to 7. An annual increment of worm load due to chance infection of about 1.5 worms per year is thus sufficient to maintain this low level infestation. If we have movement of infested hosts into the area and exposure of the population to the feces of these migrants, it seems possible that low level infestation will be persistent despite good domestic sanitation.

4.6.2.2. Overyear Model 2 and Sensitivity Analysis

The one-season model in this case is:

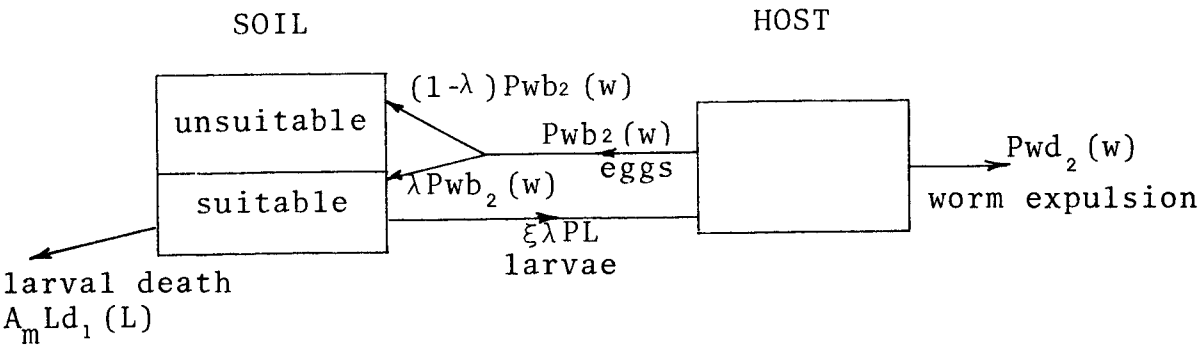


Figure 4.34. One-season, Two-state Deterministic Hookworm Model 2

The system equations are:

$$A_m \frac{dL}{dt} = \lambda P w \gamma - AL\alpha_1$$

$$P \frac{dw}{dt} = \xi\lambda PL - Pw\alpha_2 e^{\beta_2 w}$$

The equilibrium mean worm load is:

$$w_\infty = \frac{1}{\beta_2} \ell n \left(\frac{\xi \lambda^2 P \gamma}{A_m \alpha_2 \alpha_1} \right) \quad .$$

As in the case of Model I, this model reproduces the results of the simulation model.

In the above models we have assumed that the size of the defecation area remains constant as population varies. We wish to examine the effect of the extreme opposite assumption, namely that the size of the defecation area varies linearly with the size of the population using it.

At population P = 1000, λ = 0.5 we have $A_m = 10^4 ft.^2$. There-

fore, $\frac{\lambda P}{A_m} = \frac{1}{20} \frac{\text{persons}}{\text{sq. ft.}}$.

Therefore,

$$w_\infty = \frac{1}{\beta_2} \ell n \left(\frac{\xi \lambda^2 P \gamma}{A_m \alpha_2 \alpha_1} \right)$$

$$= \frac{1}{\beta_1} \ell n \left(\frac{\xi \lambda \gamma}{20 \alpha_2 \alpha_1} \right) = 100$$

In this case, then, the mean worm load in the community which does not use latrines is unaffected by the number who do use latrines -- there are no external effects accruing to the non-users. For a community behaving in this manner, low level hookworm endemicity could persist if we had the majority using latrines and if we had the few (high worm load) non-latrine users occasionally defecating in places where the latrine users would be exposed to the larvae.

We do not have any data on the relationship between the number of people using a defecation area and the size of such areas. From Kochar's (1975) study of the human factors in hookworm in rural West Bengal it appears that the defecation areas are on land which is unlikely to be agriculturally productive (e.g., bamboo groves). If this is true, the area available for defecation purposes is unlikely to be closely linked to the size of the population not using latrines.

On the other hand the view that the area <u>used</u> for defecation remains constant may not be valid. If certain communities use certain defecation areas which are under no circumstances used by other communities, and if latrine acceptance is high in certain communities and low in others (as seems likely), then it

may well turn out that the defecation density remains more or less constant for those in the low acceptance communities. In this case we would expect a considerable reduction in the worm loads of the non-users in the high acceptance community while the reduction in worm loads of non-users in the low acceptance community may be minimal. Information on the behavioual characteristics of the particular village under consideration is thus essential in understanding the effects of latrine use on hookworm infestation.

4.6.2.3. Stochastic Version of Model 2

A check on the stability of the deterministic models may be made by observing the operation of the model under a stochastic regime. Possible sources of noise in this system include variations in the proportion of the defecation area which is suitable for larval development (due to climatic variation) and in the diet of the population and thus the strength of the immune response (this, too, is likely to be correlated with weather).

In this analysis we will simulate the changes in the mean worm load in the community holding all parameters, other than the proportion of the defecation area which is wet, constant.

Professor Harold Thomas has developed a five-state lag-one Markov model for simulating the annual weather pattern in Bengal. The transition matrix is given in Figure 4.35.

		ν_{t+1}				
		0.50	0.75	1.00	1.25	1.50
ν_t	0.50	.1667	.1667	.5000	.1667	0
	0.75	.1667	.1667	.5000	.1667	0
	1.00	.0833	.1667	.5000	.1667	.0833
	1.25	0	.1667	.5000	.1667	.1667
	1.50	0	.1667	.5000	.1667	.1667

Figure 4.35. Markov Matrix for Annual Rainfall in West Bengal

The stationary probabilities are given on Figure 4.36.

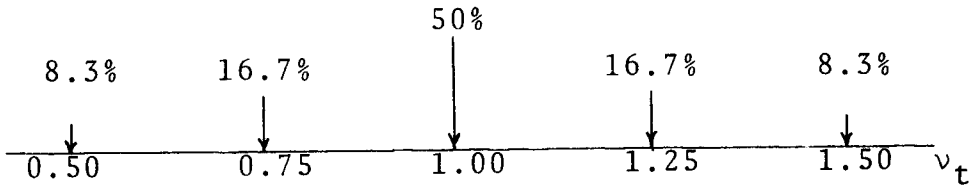


Figure 4.36. Stationary Rainfall Probabilities

where ν_t represents the rainfall in year t. The coefficient of variation is 0.25 and the serial correlation coefficient is 0.22. Assuming that sites within the defecation area are chosen randomly, the weather parameter affects both the proportion who will squat in moist areas ($\lambda_t = \bar{\lambda}\frac{\nu_t}{\bar{\nu}}$) and the size of the moist area ($A_t = \bar{A}\ \frac{\nu_t}{\bar{\nu}}$).

The difference equations are, then:

$$L_{t+\Delta t} = L_t + \left(\frac{\bar{\lambda} P W_t \gamma}{\bar{A}} - L_t \alpha_1\right) \Delta t$$

and

$$W_{t+\Delta t} = W_t + (\xi \bar{\lambda} \nu_t L_t - W_t \alpha_2 e^{\beta_2 W_t}) \Delta t \quad .$$

The computer routine generates a synthetic rainfall record using a rectangular random number generator and the transition matrix, and uses these values to simulate the mean community worm load. (The accounting period was taken to be 1/100th of a year; annual accounting incorporates too large an inertial effect.)

On Figure 4.37 a 50-year mean community worm load trace is reproduced. The model appears to function satisfactorily under

a stochastic regime.

In the above model it is assumed that infestation takes place when the host is stationary (squatting). It seems likely, however, that some infestation occurs in passage to the defecation spot. Since the distance travelled to the defecation spot is proportional to the square root of the area, assumption of exclusive infestation in this manner leads to the following set of difference equations:

$$L_{t+\Delta t} = L_t + \left(\frac{\bar{\lambda} P W_t \gamma}{\bar{A}_m} - L_t \alpha_1 \right) \Delta t$$

and

$$W_{t+\Delta t} = W_t + \left(\xi \sqrt{\bar{A}_m} \; \bar{\lambda} \nu_t^{1.5} L_t - W_t \alpha_2 e^{\beta_2 W_t} \right) \Delta t$$

The 50 year mean worm load trace under this assumption, too, is presented on Figure 4.37. We note that the standard deviation of the mean worm load when all infestation takes place during squatting is less than the standard deviation when infestation is assumed to occur in passage to the defecation spot. In the first case the rate of infestation increases linearly with the increased proportion wet while in the latter case the rate of infestation is further increased by the fact that the length of travel to the defecation spot rises in proportion to the square root of the rise in area.

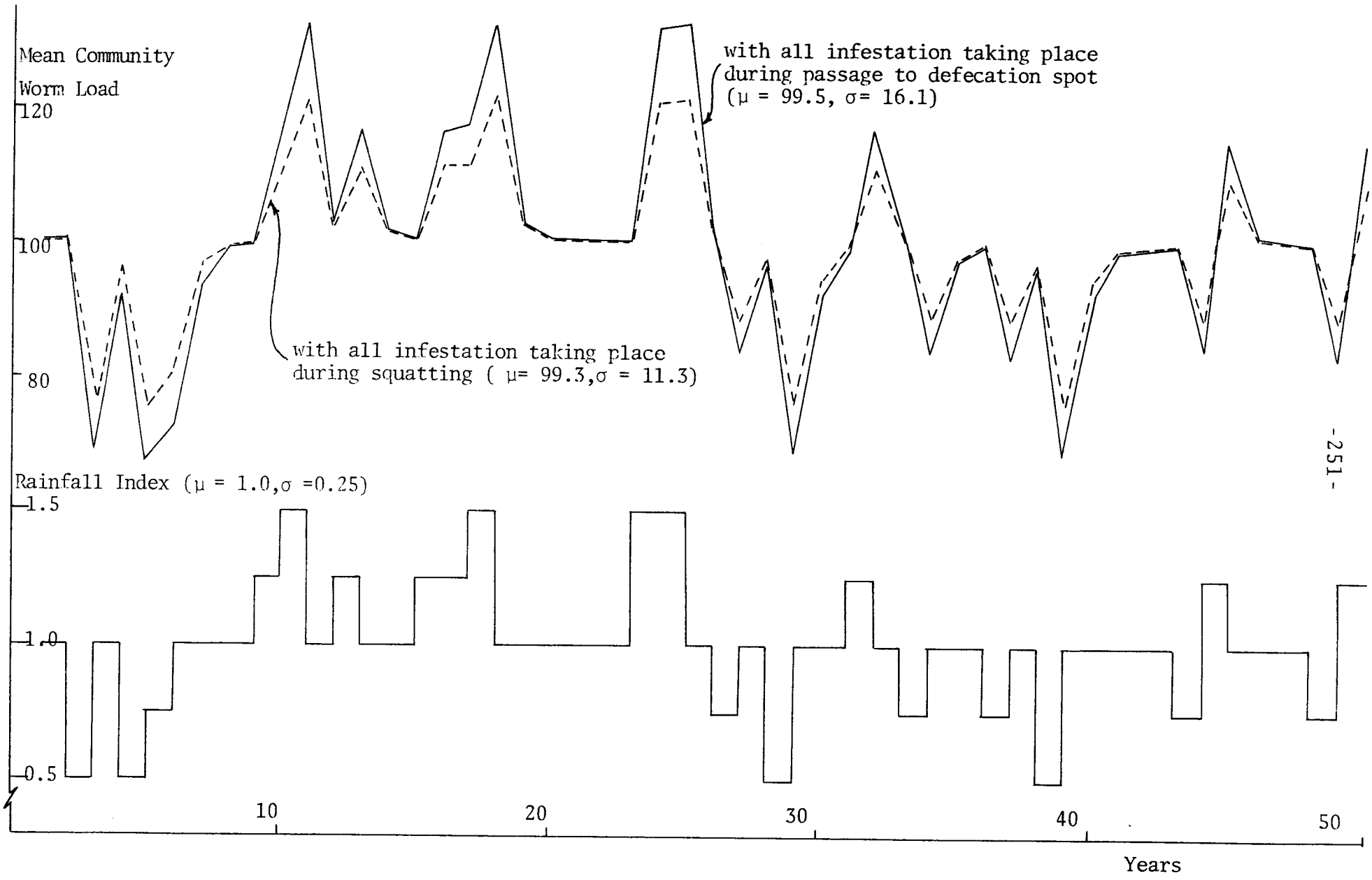


Figure 4.37: Mean Community Worm Load with Stochastic Climate

4.6.3. Conclusion

Due to structural limitations in the models and deficiencies in the data base, care should be taken in interpreting the results of the models which have been presented. Many of these problems have been addressed in the text; here we would like to comment on a few of the more prominent of these.

"Death from hookworm is infrequent in either acute or chronic stages, and then usually in association with other infections." (American Public Health Association [1960]) For this reason mortality differentials based on the degree of hookworm infestation were not included in this model. A simple numerical example of the effect of interactions among various diseases is presented in Appendix 4.14. This example shows that the distortions introduced by not considering the complicating effects of hookworm on mortality due to the primary causes of death are not likely to be significant.

In Model 2 (which appears to provide the better representation of reality) the outstanding uncertainties are those regarding the penetration parameter and the worm expulsion rate. Sensitivity analysis indicates that uncertainty in these parameters introduces considerable uncertainty in the model output. In collecting more data, attention should be focussed on these parameters and on the mean worm load changes emanating from changed environmental conditions. The latter are essential for adequate calibration of the model.

An unsatisfactory feature of the present models is that distributional measures within the community are not explicitly included. Changes in the distribution of infestation within the community over time may have important consequences in the model. The effects of these distributional factors on the specification of the immunity function are investigated in Appendix 4.15.

While we believe that the models developed here will prove useful in evaluating changes arising from environmental improvement in areas where hookworm is endemic, the assumptions (such as the exclusive acquisition of larvae from focal pollution sources) need to be critically examined before inferences concerning low level endemicity and eradication can be drawn.

CHAPTER FIVE

A MODEL FOR QUANTIFYING SOME EFFECTS OF A SANITATION PROGRAM

Summary

Improved excreta disposal practices will affect the interactions of man and a wide range of disease organisms. Given the paucity of adequate epidemiological models, this chapter evaluates the effects of only typhoid fever and hookworm infestation on a population living under different environmental conditions. The epidemiological models for these diseases are coupled with a demographic model for simulating the changes in the morbidity and mortality time streams of the presently existing village population under different sanitation regimes. The health effects are summarized by examining changes in life expectancy, expected days of sickness and degree of helminthic infestation. These health changes are translated into economic effects: the effect of increased working lives, the effect of decreased absenteeism, and the effect of improved on-the-job efficiency. The effects of improved health on some measures of nutrition are also assessed.

This model is in essence, then, a production function whereby "inputs" (changes in the sanitation regime) are mapped into a multi-dimensional "output" (mortality, morbidity, economic, nutritional) vector.

CONTENTS

5.1. INTRODUCTION

Traditionally, public expenditures on health have been justified as contributing directly to human well-being ("consumption"). The observation that the historical rate of economic growth could not be entirely explained in terms of the inputs of land, capital and labour gave rise to human capital theory and the concept of health care as an "investment". In this chapter both of these aspects will be addressed in the development of a methodology for translating environmental changes into demographic, economic and nutrition measures.

The relationships between public health interventions and the measures of interests to planners are extremely complex. In terms of this complexity the goals of this chapter are modest. The methodology to be outlined represents a considerable simplification of reality and the analysis which is carried out builds but a few of the many blocks which are needed if the implications of environmental interventions are to be fully traced.

The methodology proposed consists of:

i) Developing a set of epidemiological models, specific to each of the diseases which may be affected by the intervention, for determining the changes in the incidence of each of these diseases over time;

ii) Specifying a demographic model for translating these disease-

specific changes into age-specific mortality and morbidity changes and thus changes in the size and age structure of the presently existing population;

iii) Developing a partial equilibrium model for determining the economic implications of these demographic changes; and

iv) Constructing a model for determining the nutritional implications.

White, Bradley and White (1972) have examined the literature pertaining to the effects of improved domestic water supply on health:

> Those who wish to promote public works to improve domestic water supply invariably employ as their chief justification the expected gains to the public health. These confident and vigorous claims have often been extrapolated beyond the evidence on which they are based and, although there is no doubt improved water supplies usually do improve public health and often do so dramatically, any attempt to be quantitative must be based on a careful evaluation of the health consequences of unimproved supplies. There are three main objections to much available discussion on this topic: first, a tendency to select from the available studies only those data supporting the writer's viewpoint; second, extrapolation of results from one area to another without regard for epidemiological differences between them; and third, the fallacy of misplaced aggregation: lumping together the health problems resulting from poor water supplies as "water borne diseases" in such a way as to imply that only one type of improvement is relevant and necessary.

These objections are equally valid with respect to studies on the consequences of improved excreta disposal practices. In this analysis we have attempted to present the results of either all, or a representative subset of, studies pertinent to the estimation

of a particular parameter. Bias of the first sort has probably entered, nevertheless, through "publication bias", whereby hypothesis-supporting results are more likely to be published than hypothesis-rejecting results, and in the choice of which of the available studies is most appropriate for the purpose at hand.

A conscious effort has been made to avoid errors of the second sort by using, where possible, data for Bengal, and by specifying that the analysis is informative only under Bengal conditions. Some extrapolation is inevitable, however, given the variety of sources on which this analysis has drawn. Inappropriate use of data from different areas is probably more serious with respect to the choice of the economic parameters than it is in specifying the epidemiological parameters.

In the present context the objective is to trace the implications of a change in the system of excreta disposal in a village in India. The incidence of each gastrointestinal disease is likely to be affected by this change. While there is some attraction in the concept of dealing with these diseases as a syndrome and building a single epidemiological model for this syndrome, the etiological differences in these diseases are such that the validity of such an approach is highly dubious. There seems to be no alternative to developing epidemiological models for each of the diseases involved. Since sufficiently detailed models are available only for typhoid (Cvjetanovic [1971]) and hookworm (see Chapter Four) the analysis which follows is

confined to tracing the effects of changed excreta disposal practices on the transmission of these two diseases within a community and outlining the demographic and economic implications. Thus the fallacy of "misplaced [epidemiological] aggregation" referred to by White et al. is avoided. A complete analysis within the framework outlined above is contingent on the development of a complete set of epidemiological models.

The difficulties which arise in this simplified and proscribed treatment are indicative of the rudimentary state of the art. The analysis presented here is tentative; if these models are to have any value it is essential to constantly re-examine the assumptions as more information becomes available and to change the parameter values and expand the model structure accordingly.

5.2. EPIDEMIOLOGICAL MODELS

5.2.1. Typhoid Fever Model

A simple analytic analog to Cvjetanovic's simulation model for typhoid fever (1971) was derived in Chapter Four. We will calibrate this analytic model with representative Indian data and use it to simulate the effects of improved excreta disposal practices on the incidence of typhoid.

The differential equation describing the simplified version of Cvjetanovic's model is:

$$\frac{dx_t}{dt} = \phi - (\phi+\beta)x_t + \beta x_t^2$$

where x_t is the proportion of the population which is not infected, ϕ is the parameter representing the rate of recovery, and β is the infectivity parameter.

The equilibrium value of x_t (for $x_t \leq 1$) is $x_t = \phi/\beta$. On Figure 4.6 the relationship between x_t and $1/\beta$ (or 1/RI in Cvjetanovic's terms) is presented. The data presented by Cvjetanovic (who used the mean values) which are germane to the present analysis are: Duration of sickness: range 14-35 days; mean 28 days. Case fatality rate: range 1-10%; mean 3%. Incidence in endemic areas: range 10-150 per 10,000 population.

The stable percentage distribution in population classes for different levels of the force of infection are given in Table 5.1 overleaf (from Cvjetanovic [1971]).

Population Class	Daily Force of Infection (RI)			
	0.0018	0.0020	0.0025	0.0040
Susceptible	94.5	84.9	67.3	41.7
Incubating Non-infectious	0.0244	0.0661	0.143	0.254
Incubating infectious	0.00122	0.00331	0.00716	0.0127
Sick infectious	0.0511	0.139	0.300	0.534
Sick non-infectious	0.00583	0.0158	0.0342	0.0609
Temporary carriers	0.0158	0.0430	0.0930	0.166
Permanent carriers	0.966	2.62	5.73	10.3
Short resistant	0.527	1.43	3.09	5.51
Long resistant	3.96	10.7	23.3	41.5
Total	100	100	100	100
Annual typhoid incidence rate per 10,000 pop.	12.8	34.8	75.2	133.9
Annual typhoid death rate per 100,000 pop.	4.3	11.7	25.3	45.1

Table 5.1. Stable Percentage Distribution in Population Classes for Different levels of Force of Infection (Birth rate and crude death rate are both equal to 20 per thousand population.)

From Table 5.1 we note the following:

i) If we define:

$$\text{"Effective duration of sickness" (days)} = \frac{\text{prevalence of sickness (\%)} \times 365 \times 100}{\text{incidence per year per 10,000}}$$

we note that this is constant at about 162 days for the population considered. This is much greater than the "initial duration of

sickness", due to the phenomenon of relapse.

ii) The percentage of non-susceptibles who are actually sick is low (about 1.025%) and approximately constant for the range of infective parameters considered.

We use several sources of data to estimate reasonable parameter values under Indian conditions. In an analysis of rural health services in India, Dutt (1962) gives annual typhoid fever sickness rates of 2219, 832, 796 and 102 per lakh (100,000) in areas of West Bengal, Madhya Pradesh, Bihar and Rajasthan, respectively. In an analysis of causes of death in the rural Punjab, Gordon, Singh and Wyon (1965) found an average typhoid death rate (for all ages and both sexes) of 71.6 per 100,000.

In this analysis we will assume that the annual incidence of typhoid in the community considered is 1000 per 100,000. This estimate is consistent with the range given by Dutt and lies in the upper part of the world-wide range given by Cvjetanovic. For this incidence to be consistent with the typhoid death rate given by Gordon, Singh and Wyon, the fatality rate would be about 7.2%. This is on the high side of the range given by Cvjetanovic and seems reasonable given the absence of diagnostic and treatment facilities for typhoid fever in rural areas of India (Dutt [1962]).

Dutt (1962) found that the duration of sickness varied between four and six weeks, while Seal (1968) found an average duration of 36 days among government employees in Delhi. In this analysis we will assume that the average duration of illness

after an attack is 28 days. This is the value used by Cvjetanovic and seems to accord with Indian data.

From Figure 4.6 we have elimination of typhoid at $1/\beta = 580$. Since at eradication $\phi = \beta$, the effective value of ϕ is 0.00173 per day. For the duration of illness assumed, an annual incidence of 100 per 10,000 implies that 57% of the population is susceptible ($x_t = 0.57$). Figure 4.6 specifies a value of $1/\beta = 336$ for $x_t = 0.57$. The value of β under present conditions is, therefore, $\beta = 0.00298$.

Typhoid is transmitted by "direct or indirect contact with the feces or urine of a patient or carrier" (American Public Health Association [1970]). The World Health Organization data used by Cvjetanovic suggest that the transmission rate or force of infection is likely to be reduced to half of its former value by the use of latrines by the entire population. We will assume the relationship between latrine use and transmission reduction to be linear (i.e. if Z% use latrines, the force of infection will be reduced by 0.5Z%) and that all of the health benefits are "external". This latter assumption is equivalent to assuming that the probability of contracting typhoid is reduced by the same proportion for those who use and those who do not use latrines.

In Appendix 4.3 the equation describing the approach of the above system to equilibrium is given as:

$$x_t = \frac{\phi(x_o-1) - (\beta x_o-\phi)e^{-t(\beta-\phi)}}{\beta(x_o-1) - (\beta x_o-\phi)e^{-t(\beta-\phi)}} \qquad (1)$$

where x_t is the proportion of the population susceptible at time t.

For an initial incidence of 1000 per 100,000 (x_o = 0.57) the changes in incidence over time for various percentages of latrine use are computed using equation (1) above and the relationship between proportion susceptible and incidence implicit in Table 5.1, and given in Table 5.2 below:

Time (Years)	Annual Incidence per 100,000 for Latrine Use Percentage of:			
	0%	20%	60%	100%
0	1000.0	1000.0	1000.0	1000.0
1	1000.0	941.1	844.2	748.6
2	1000.0	903.7	741.9	547.4
3	1000.0	878.8	669.8	475.6
5	1000.0	850.7	579.1	331.4
10	1000.0	828.8	474.4	163.3
15	1000.0	825.1	434.9	-
20	1000.0	824.4	416.3	54.0
40	1000.0	-	-	8.4
80	1000.0	824.4	402.3	0

Table 5.2. Typhoid Incidence Over Time for Different Sanitation Regimes

Gordon et al. (1965) have given the following age-specific death rates for males in the rural Punjab during a period in which there were no epidemics:

	Age Group					
	0-1	1-4	5-15	15-44	45-64	65+
Typhoid Death Rate/1000	2.8	2.3	0.5	0.3	0.0	0.6
Total Death Rate/1000	144.6	19.4	1.8	2.2	14.3	77.4
Percentage Due to Typhoid	1.94	11.86	27.78	13.64	0.0	0.78

Table 5.3. Age-Specific Typhoid Death Rates

We will assume that in the community under consideration typhoid accounts for the same proportion of total mortality as that given in Table 5.3. If, then, the incidence of typhoid is cut by 50% and the incidence and virulence of other causes of death remain unchanged, the death rates for infants will decline by 0.97% while the death rate for male children age 5 - 14 will drop by 13.89%.

5.2.2. Hookworm Model

The results of latrine use in the hookworm model developed in Chapter Four are indicated on Figure 4.31. In this case the benefits of latrine use are part "internal" and part "external", and we therefore have to consider the populations of "users" and "non-users" separately. The mean worm loads in these populations are given on Table 5.4 below for different latrine use patterns.

	Mean Worm Load for Latrine Use by:					
	0%	20%		60%		100%
Time (Years)	Users & Non-Users	Users	Non-Users	Users	Non-Users	Users & Non-Users
0	100	100	100	100	100	100
1	100	62	93	62	81	62
2	100	41	89	41	70	41
3	100	29	87	29	64	29
5	100	16	86	16	57	16
10	100	7	85	7	52	7
20	100	2	85	2	50	2
40	100	0	85	0	50	0

Table 5.4. Mean Hookworm Loads for Different Sanitation Regimes

Schad's data (1975) on mean egg counts and Nawalinski's data (personal communication) on fecal output suggest that the worm burden is below the community average for those under age 15 and above this average for those over 55. In this analysis we are concerned only with the worm burdens of working age males and we will assume that the figures on Table 5.4 pertain uniformly to this group.

5.3. DEMOGRAPHIC EFFECTS

5.3.1. The Model

Cohen (1973b,c) has developed a framework in which to evaluate the increase in the economic output of a population due to an extension in working lives. Cohen's demographic analysis essentially consists of:

i) Determining the age structure of the existing population;

ii) Deriving the life table for this population;

iii) Determining the changes in the mortality regime which would result from elimination (or reduction) of a specific disease type;

iv) Computing the revised life table; and

v) Using this life table and the given population age structure to determine the effect of the new mortality regime on the existing population.

Two major demographic assumptions in the model are:

i) Any demographic consequences of the shift in age composition of the people as a result of saving lives, other than the changes in the number of individuals in each class, are ignored;

ii) The causes of death whose reduction is being considered act in a prescribed age-specific fashion, and independently of other causes of death. These other causes of death are assumed to remain unchanged throughout the period of analysis. (This assumption may not be very restrictive: the population differen-

tials are determined as the difference between the present and the changed mortality regimes, with the latter being derived from the former. If, then, the "other causes" did not remain constant, this is an error in both the "before" and "after" situation. If we take such "other changes" into account, the difference between the emerging differentials and those which we have computed may not be great, since the two errors will tend to cancel out.)

Exclusive consideration of the present population simplifies the analysis since no assumption concerning future fertility is required. The problem of comparing lives saved now with lives saved in the future is avoided in this analysis by presenting the decision-maker with the life expectancies of the different age groups in the present population at five year internals. The problem of aggregation over time has to be faced at some level in the decision process, however, and we therefore discuss it briefly here. As Feldstein (1970) has pointed out, the basic justification for discounting future increments to consumption, namely that the marginal utility of consumption falls as per capita income rises, provides no justification for discounting future lives saved. The paradoxical problem of an infinite number of lives saved by a program can only be resolved by introducing pure time preference. Cohen (1973b) sees his ex ante type of analysis as being useful in that "this view avoids a potentially infinite stream of benefits to an increasing popula-

tion." Mechanically, of course, this is true, but the method provides no escape from the necessity of introducing a pure time preference if benefits of this sort are to be aggregated.

5.3.2. Existing Population Size and Age Structure

The basic population age structure assumed in this analysis is that given by Keyfitz and Flieger (1968) for India in 1961. The population size is assumed to be 1000 - a figure which is typical of an Indian village. In this model we will deal only with the male population for reasons which will become clear when the economic implications are discussed. The implicit assumptions are such that the ratio of males to females is unity and that there is no difference in the sex-specific disease and death rates. While these assumptions are clearly not valid under Indian conditions and a more complex model would include these considerations, it seems unlikely that the distortions introduced are important.

The data for Indian males are presented on Table 5.5 (overleaf).

Age at Last Birthday	Male Population (in 100's)
0	84680
1 - 4	282770
5 - 9	295260
10 - 14	255450
15 - 19	221740
20 - 24	195260
25 - 29	176200
30 - 34	157630
35 - 39	136340
40 - 44	115750
45 - 49	96810
50 - 54	78360
55 - 59	60550
60 - 64	43870
65 - 69	29180
70 - 74	17080
75 - 79	6760
80 - 84	5810
85+	3430
Total	2262930

1/23030

Table 5.5. Male Population Size in India in 1961

5.3.3. Life Table

Keyfitz and Flieger (1968) present a life table for India for the year 1961. Table 5.6 below presents the relevant columns from this life table.

x	$_nq_x$	$_nL_x$	e_x
0	0.146107	90431	46.024
1	0.075798	323512	52.840
5	0.021446	390353	53.074
10	0.015872	383070	49.183
15	0.023029	375868	44.935
20	0.032631	365398	40.932
25	0.035872	352775	37.226
30	0.041043	339279	33.517
35	0.048738	324145	29.842
40	0.060285	306576	26.240
45	0.074309	286121	22.759
50	0.098266	261682	19.378
55	0.129096	232269	16.209
60	0.180122	196835	13.230
65	0.246828	155360	10.547
70	0.342019	110351	8.209
75	0.468195	66489	6.183
80	0.637523	30757	4.496
85+	1.000000	11167	3.304

Where x is age, $_nq_x$ is the probability of dying within n years for an individual now aged x, $_nL_x$ is the number living in the life table between ages x and x+n, and e_x is the expectation of life at age x.

Table 5.6. Life Table for India (1961)

5.3.4. Changes in Mortality Regime

In this model we are considering the effect of improved excreta disposal practices on thos two diseases (typhoid and hookworm) for which we have the necessary epidemiological models. We have assumed that mortality changes are associated with typhoid incidence changes and not with changes in the community hookworm load. As outlined earlier, we use the annual typhoid incidence changes, for any percentage latrine use (Table 5.2), with the age-specific proportions of death due to typhoid to determine ${}_{n}r_{x}^{-i}$, the proportion of observed deaths due to causes other than the causes(s) eliminated, for all age groups at each of the accounting times.

5.3.5. Computing the Revised Life Tables

Spiegelman (1968) gives a method by which an existing life table may be modified to take account of a change in the mortality regime. If we let ${}_nr_x^{-i}$ be the proportion of observed death due to all causes other than the cause eliminated, then the probability of surviving n years after age x (${}_np_x^{-i}$) in a life table from which cause i is completely eliminated is closely approximated by: $\text{colog}\ {}_np_x^{-i} = {}_nr_x^{-i}\ \text{colog}\ {}_np_x$.

(For partial elimination - which is the case applicable to this analysis - the procedure and the equations are the same as above.)

We use the initial life table as the basis. We determine the first column (${}_nq_x^{-i}$) in a revised life table for each ${}_nr_x^{-i}$. The life table column of primary interest in determining the size and age structure of the population in the future is ${}_nL_x$, the number of years lived by the persons who have reached age n before they (or, more accurately, their survivors) reach age x+n (Barclay [1958]). Column ${}_nL_x$ can be interpreted, when thinking of the life table as a cohort mortality history, as the average number of persons living in the interval x to x+n. It is this interpretation of ${}_nL_x$ that is intuitively more attractive in our analysis.

We use the following life table relations to compute column ${}_nL_x$ for each mortality regime.

Assume: $l_o = 100,000$

$$ {}_nq_x^{-i} = 1.0 - {}_np_x^{-i} $$

$$ l_{x+n} = (1.0 - {}_nq_x^{-i}) \cdot l_x $$

$$ {}_nd_x = l_x - l_{x+n} $$

$$ {}_1L_0 = a \cdot l_0 + (1.0 - a) \cdot l_1 $$

$$ {}_4L_1 = b \cdot l_1 + (1.0-b) \cdot l_5 \cdot 4.0 $$

and $$ {}_5L_x = (l_x + l_{x+5}) \times 2.5 \quad \text{for all } x \geq 5. $$

(where the implied values of a and b in the given life table are: a = 0.345 and b = 0.303.)

Revised life tables are computed for the ${}_nr_x^{-i}$ values pertaining to each of the sanitation programs. For the program with 60% latrine use, the relevant life tables are listed in Table 5.7.

Table 5.7. Life Tables Used in Case Where 60% of the Population of a Village of One Thousand People Use Latrines

INCIDENCE OF TYPHOID FOR THIS LIFE TABLE = 1.0000 %
(Initial)

x	$_nq_x$	l_x	$_nD_x$	$_nL_x$
0	0.1461	100000.	14610.	90430.
1	0.0758	85390.	6473.	323514.
5	0.0214	78917.	1689.	390365.
10	0.0159	77229.	1228.	383073.
15	0.0230	76001.	1748.	375633.
20	0.0326	74253.	2421.	365212.
25	0.0359	71832.	2579.	352713.
30	0.0410	69253.	2839.	339168.
35	0.0487	66414.	3234.	323983.
40	0.0603	63180.	3810.	306373.
45	0.0743	59370.	4411.	285821.
50	0.0983	54959.	5402.	261287.
55	0.1291	49556.	6398.	231787.
60	0.1801	43158.	7773.	196360.
65	0.2468	35386.	8733.	155095.
70	0.3420	26652.	9115.	110474.
75	0.4682	17537.	8211.	67159.
80	0.6375	9326.	5946.	31768.
85	0.0000	3381.	0.	16904.

INCIDENCE OF TYPHOID FOR THIS LIFE TABLE = 0.7200 %
(Years 1 to 5)

x	$_nq_x$	l_x	$_nD_x$	$_nL_x$
0	0.1454	100000.	14537.	90478.
1	0.0734	85463.	6271.	324369.
5	0.0198	79192.	1564.	392049.
10	0.0147	77628.	1139.	385291.
15	0.0221	76489.	1693.	378212.
20	0.0314	74796.	2347.	368113.
25	0.0346	72449.	2503.	355988.
30	0.0395	69946.	2760.	342828.
35	0.0469	67185.	3150.	328052.
40	0.0581	64035.	3718.	310882.
45	0.0743	60317.	4482.	290382.
50	0.0983	55836.	5489.	265456.
55	0.1291	50347.	6500.	235485.
60	0.1801	43847.	7897.	199494.
65	0.2463	35950.	8856.	157612.
70	0.3414	27094.	9250.	112847.
75	0.4675	17944.	8342.	68367.
80	0.6367	9603.	6050.	32387.
85	0.0000	3452.	0.	17262.

(Table 5.7 continued)

INCIDENCE OF TYPHOID FOR THIS LIFE TABLE = 0.4700 %

(Years 6 to 20)

x	$_nq_x$	l_x	$_nD_x$	$_nL_x$
0	0.1447	100000.	14471.	90521.
1	0.0712	85529.	6091.	325134.
5	0.0183	79438.	1452.	393560.
10	0.0136	77986.	1059.	387283.
15	0.0214	76927.	1643.	380529.
20	0.0303	75284.	2280.	370723.
25	0.0333	73005.	2435.	358938.
30	0.0381	70570.	2688.	346130.
35	0.0453	67882.	3072.	331728.
40	0.0561	64809.	3634.	314963.
45	0.0743	61176.	4545.	294516.
50	0.0983	56630.	5567.	269235.
55	0.1291	51064.	6592.	238838.
60	0.1801	44471.	8009.	202334.
65	0.2459	36462.	8967.	159893.
70	0.3409	27495.	9372.	114046.
75	0.4668	18123.	8460.	69465.
80	0.6360	9663.	6145.	32951.
85	0.0000	3517.	0.	17587.

INCIDENCE OF TYPHOID FOR THIS LIFE TABLE = 0.4050 %

(Years 20 to 35)

x	$_nq_x$	l_x	$_nD_x$	$_nL_x$
0	0.1445	100000.	14454.	90532.
1	0.0706	85546.	6044.	325333.
5	0.0179	79502.	1423.	393953.
10	0.0133	78079.	1038.	387803.
15	0.0212	77042.	1630.	381134.
20	0.0300	75412.	2262.	371405.
25	0.0330	73150.	2417.	359709.
30	0.0377	70733.	2669.	346994.
35	0.0448	68064.	3052.	332691.
40	0.0555	65012.	3611.	316033.
45	0.0743	61401.	4562.	295600.
50	0.0983	56839.	5587.	270227.
55	0.1291	51252.	6617.	239717.
60	0.1801	44635.	8039.	203079.
65	0.2458	36596.	8996.	160492.
70	0.3407	27600.	9404.	114491.
75	0.4666	18196.	8491.	69753.
80	0.6358	9705.	6170.	33099.
85	0.0000	3535.	0.	17672.

(Table 5.7 continued)

INCIDENCE OF TYPHOID FOR THIS LIFE TABLE = 0.3980 %

(Years 35 to 80)

x	$_nq_x$	l_x	$_nD_x$	$_nL_x$
0	0.1445	100000.	14453.	90534.
1	0.0706	85547.	6038.	325354.
5	0.0179	79509.	1420.	393996.
10	0.0133	78089.	1035.	387859.
15	0.0211	77054.	1628.	381199.
20	0.0300	75426.	2260.	371478.
25	0.0330	73166.	2415.	359792.
30	0.0377	70751.	2667.	347087.
35	0.0448	68084.	3050.	332795.
40	0.0555	65034.	3609.	316149.
45	0.0743	61425.	4564.	295717.
50	0.0983	56861.	5589.	270334.
55	0.1291	51272.	6619.	239812.
60	0.1801	44653.	8042.	203159.
65	0.2458	36611.	8999.	160556.
70	0.3407	27612.	9408.	114540.
75	0.4666	18204.	8495.	69784.
80	0.6358	9709.	6173.	33115.
85	0.0000	3536.	0.	17682.

5.3.6. Mortality History of Population Subject to the Above Risks of Death

For the case examined by Cohen (total elimination of schistomiasis in Zanzibar) the change in the mortality regime implied an instantaneous shift from the existing life table to the life table which prevails under complete elimination of schistosomiasis. In that case the mortality effect of disease elimination is determined by a comparative examination of the life tables "before" and "after".

In the case under consideration our epidemiological model specifies that there was not simply an instantaneous reduction in the mortality rates, but that the mortality reduction due to sanitation improvement is a dynamic phenomenon. We have to determine, in each time period, which life table (dependent on the mortality level) is applicable. On the basis of this life table we compute the changes which each age group will undergo in that time period.

If the number of people, who were originally in age group I, alive in time period K is S(I,K), then the number alive from this age group in period K+1 (using five year periods) is, according to the commonly-used cohort survival formula,

$$S(I,K) \quad x \quad \frac{{}_5L_{5(K+I+1)}}{{}_5L_{5(K+I)}}$$

The population histories for two different base populations, (i) the stationary population modeled by Keyfitz and Flieger

(Table 5.6), and (ii) that of a village of 500 males which has the same age structure as that of India as a whole (from Table 5.5), are presented in Tables 5.8 and 5.9 respectively, for both present and changed (60% latrine use) conditions. We note that the cohort histories in Table 5.8 under existing conditions are truncated versions of the initial life table.

The mortality experiences of each cohort under the present conditions, and under the conditions prevailing when 20%, 60% and 100% of the population use latrines, are summarized in terms of their life expectancies on Table 5.10. These expectancies are determined by summing the columns in Table 5.8 - which are equivalent to ${}_nL_x^{-i}$ on a life table - for each cohort, and dividing those sums by the appropriate l_x values in the initial life table (see Table 5.7).

Table 5.8. Number of Survivors When Taking the Base Population to be that Used in Construction of Life Tables

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AVG # LIVING UNDER PRESENT CONDITIONS

	AGE GROUP								
PERIOD	0	1	2	3	4	5	6	7	8
0	413945.	390365.	383073.	375633.	365212.	352713.	339168.	323983.	306373.
1	390365.	383073.	375633.	365212.	352713.	339168.	323983.	306373.	285821.
2	383073.	375633.	365212.	352713.	339168.	323983.	306373.	285821.	261287.
3	375633.	365212.	352713.	339168.	323983.	306373.	285821.	261287.	231787.
4	365212.	352713.	339168.	323983.	306373.	285821.	261287.	231787.	196360.
5	352713.	339168.	323983.	306373.	285821.	261287.	231787.	196360.	155095.
6	339168.	323983.	306373.	285821.	261287.	231787.	196360.	155095.	110474.
7	323983.	306373.	285821.	261287.	231787.	196360.	155095.	110474.	67159.
8	306373.	285821.	261287.	231787.	196360.	155095.	110474.	67159.	31768.
9	285821.	261287.	231787.	196360.	155095.	110474.	67159.	31768.	0.
10	261287.	231787.	196360.	155095.	110474.	67159.	31768.	0.	0.
11	231787.	196360.	155095.	110474.	67159.	31768.	0.	0.	0.
12	196360.	155095.	110474.	67159.	31768.	0.	0.	0.	0.
13	155095.	110474.	67159.	31768.	0.	0.	0.	0.	0.
14	110474.	67159.	31768.	0.	0.	0.	0.	0.	0.
15	67159.	31768.	0.	0.	0.	0.	0.	0.	0
16	31768.	0.	0.	0.	0.	0.	0.	0.	0.
17	0.	0.	0.	0.	0.	0.	0.	0.	0.

	AGE GROUP							
PERIOD	9	10	11	12	13	14	15	16
0	285821.	261287.	231787.	196360.	155095.	110474.	67159.	31768.
1	261287.	231787.	196360.	155095.	110474.	67159.	31768.	0.
2	231787.	196360.	155095.	110474.	67159.	31768.	0.	0.
3	196360.	155095.	110474.	67159.	31768.	0.	0.	0.
4	155095.	110474.	67159.	31768.	0.	0.	0.	0.
5	110474.	67159.	31768.	0.	0.	0.	0.	0.
6	67159.	31768.	0.	0.	0.	0.	0.	0.
7	31768.	0.	0.	0.	0.	0.	0.	0.
8	0.	0.	0.	0.	0.	0.	0.	0.
9	0.	0.	0.	0.	0.	0.	0.	0.
10	0.	0.	0.	0.	0.	0.	0.	0.
11	0.	0.	0.	0.	0.	0.	0.	0.
12	0.	0.	0.	0.	0.	0.	0.	0.
13	0.	0.	0.	0.	0.	0.	0.	0.
14	0.	0.	0.	0.	0.	0.	0.	0.
15	0.	0.	0.	0.	0.	0.	0.	0.
16	0.	0.	0.	0.	0.	0.	0.	0.
17	0.	0.	0.	0.	0.	0.	0.	0.

(Table 5.8 continued)

AVG # LIVING UNDER CHANGED CONDITIONS

	AGE GROUP								
PERIOD	0	1	2	3	4	5	6	7	8
0	413945.	390365.	383073.	375633.	365212.	352713.	339168.	323983.	306379.
1	391197.	383636.	376034.	365603.	353182.	339675.	324549.	307026.	286171.
2	384958.	376946.	366344.	353980.	340580.	325541.	308147.	287094.	261607.
3	378245.	367232.	354698.	341350.	326409.	309089.	288142.	262450.	232070.
4	368499.	355558.	342042.	327147.	309913.	289023.	263409.	232819.	196601.
5	356893.	342990.	327943.	310767.	289875.	264214.	233669.	197235.	155372.
6	344278.	328852.	311523.	290674.	264993.	234383.	197955.	155873.	110839.
7	330087.	312386.	291381.	265724.	235075.	198560.	156443.	111197.	67528.
8	313576.	292198.	266370.	235722.	199146.	156922.	111605.	67747.	32044.
9	293311.	267116.	236296.	199694.	157385.	111947.	67996.	32148.	0.
10	268134.	236958.	200180.	157818.	112277.	68204.	32266.	0.	0.
11	237860.	200741.	158202.	112586.	68405.	32365.	0.	0.	0.
12	201506.	158646.	112860.	68594.	32461.	0.	0.	0.	0.
13	159250.	113176.	68761.	32550.	0.	0.	0.	0.	0.
14	113607.	68953.	32629.	0.	0.	0.	0.	0.	0.
15	69216.	32721.	0.	0.	0.	0.	0.	0.	0.
16	32845.	0.	0.	0.	0.	0.	0.	0.	0.
17	0.	0.	0.	0.	0.	0.	0.	0.	0.

	AGE GROUP							
PERIOD	9	10	11	12	13	14	15	16
0	285821.	261287.	231787.	196360.	155095.	110474.	67159.	31768.
1	261287.	231787.	196360.	155136.	110553.	67228.	31815.	0.
2	231787.	196360.	155173.	110653.	67337.	31890.	0.	0.
3	196360.	155173.	110679.	67398.	31942.	0.	0.	0.
4	155173.	110679.	67414.	31970.	0.	0.	0.	0.
5	110697.	67430.	31989.	0.	0.	0.	0.	0.
6	67441.	31996.	0.	0.	0.	0.	0.	0.
7	32002.	0.	0.	0.	0.	0.	0.	0.
8	0.	0.	0.	0.	0.	0.	0.	0.
9	0.	0.	0.	0.	0.	0.	0.	0.
10	0.	0.	0.	0.	0.	0.	0.	0.
11	0.	0.	0.	0.	0.	0.	0.	0.
12	0.	0.	0.	0.	0.	0.	0.	0.
13	0.	0.	0.	0.	0.	0.	0.	0.
14	0.	0.	0.	0.	0.	0.	0.	0.
15	0.	0.	0.	0.	0.	0.	0.	0.
16	0.	0.	0.	0.	0.	0.	0.	0.
17	0.	0.	0.	0.	0.	0.	0.	0.

Table 5.9. Number of Males in a Village of 1000 Initial Inhabitants under: i) Unchanged Conditions, and ii) The Mortality Regime when Typhoid is Reduced by Use of Latrines by 60% of the Population

LIFE9 4K CORE
EXECUTION

AVG # LIVING UNDER PRESENT CONDITIONS

	AGE GROUP								
PERIOD	0	1	2	3	4	5	6	7	8
0	81.	65.	56.	49.	43.	39.	35.	30.	26.
1	77.	64.	55.	48.	42.	37.	33.	28.	24.
2	75.	63.	54.	46.	40.	36.	31.	27.	22.
3	74.	61.	52.	44.	38.	34.	29.	24.	19.
4	72.	59.	50.	42.	36.	32.	27.	22.	16.
5	69.	57.	48.	40.	34.	29.	24.	18.	13.
6	67.	54.	45.	37.	31.	26.	20.	14.	9.
7	64.	51.	42.	34.	27.	22.	16.	10.	6.
8	60.	48.	38.	30.	23.	17.	11.	6.	3.
9	56.	44.	34.	26.	18.	12.	7.	3.	0.
10	51.	39.	29.	20.	13.	7.	3.	0.	0.
11	45.	33.	23.	14.	8.	4.	0.	0.	0.
12	39.	26.	16.	9.	4.	0.	0.	0.	0.
13	30.	18.	10.	4.	0.	0.	0.	0.	0.
14	22.	11.	5.	0.	0.	0.	0.	0.	0.
15	13.	5.	0.	0.	0.	0.	0.	0.	0.
16	6.	0.	0.	0.	0.	0.	0.	0.	0.
17	0.	0.	0.	0.	0.	0.	0.	0.	0.

	AGE GROUP							
PERIOD	9	10	11	12	13	14	15	16
0	21.	17.	13.	10.	6.	4.	1.	2.
1	20.	15.	11.	8.	5.	2.	1.	0.
2	17.	13.	9.	5.	3.	1.	0.	0.
3	15.	10.	6.	3.	1.	0.	0.	0.
4	12.	7.	4.	2.	0.	0.	0.	0.
5	8.	4.	2.	0.	0.	0.	0.	0.
6	5.	2.	0.	0.	0.	0.	0.	0.
7	2.	0.	0.	0.	0.	0.	0.	0.
8	0.	0.	0.	0.	0.	0.	0.	0.
9	0.	0.	0.	0.	0.	0.	0.	0.
10	0.	0.	0.	0.	0.	0.	0.	0.
11	0.	0.	0.	0.	0.	0.	0.	0.
12	0.	0.	0.	0.	0.	0.	0.	0.
13	0.	0.	0.	0.	0.	0.	0.	0.
14	0.	0.	0.	0.	0.	0.	0.	0.
15	0.	0.	0.	0.	0.	0.	0.	0.
16	0.	0.	0.	0.	0.	0.	0.	0.
17	0.	0.	0.	0.	0.	0.	0.	0.

(Table 5.9 continued)

AVG # LIVING UNDER CHANGED CONDITIONS

	AGE GROUP								
PERIOD	0	1	2	3	4	5	6	7	8
0	81.	65.	56.	49.	43.	39.	35.	30.	26.
1	77.	64.	55.	48.	42.	37.	33.	29.	24.
2	76.	63.	54.	46.	40.	36.	32.	27.	22.
3	74.	61.	52.	45.	39.	34.	30.	24.	19.
4	72.	59.	50.	43.	37.	32.	27.	22.	16.
5	70.	57.	48.	41.	34.	29.	24.	18.	13.
6	68.	55.	46.	38.	31.	26.	20.	14.	9.
7	65.	52.	43.	35.	28.	22.	16.	10.	6.
8	62.	49.	39.	31.	24.	17.	11.	6.	3.
9	58.	45.	35.	26.	19.	12.	7.	3.	0.
10	53.	40.	29.	21.	13.	8.	3.	0.	0.
11	47.	34.	23.	15.	8.	4.	0.	0.	0.
12	40.	27.	17.	9.	4.	0.	0.	0.	0.
13	31.	19.	10.	4.	0.	0.	0.	0.	0.
14	22.	12.	5.	0.	0.	0.	0.	0.	0.
15	14.	5.	0.	0.	0.	0.	0.	0.	0.
16	6.	0.	0.	0.	0.	0.	0.	0.	0.
17	0.	0.	0.	0.	0.	0.	0.	0.	0.

	AGE GROUP							
PERIOD	9	10	11	12	13	14	15	16
0	21.	17.	13.	10.	6.	4.	1.	2.
1	20.	15.	11.	8.	5.	2.	1.	0.
2	17.	13.	9.	5.	3.	1.	0.	0.
3	15.	10.	6.	3.	1.	0.	0.	0.
4	12.	7.	4.	2.	0.	0.	0.	0.
5	8.	4.	2.	0.	0.	0.	0.	0.
6	5.	2.	0.	0.	0.	0.	0.	0.
7	2.	0.	0.	0.	0.	0.	0.	0.
8	0.	0.	0.	0.	0.	0.	0.	0.
9	0.	0.	0.	0.	0.	0.	0.	0.
10	0.	0.	0.	0.	0.	0.	0.	0.
11	0.	0.	0.	0.	0.	0.	0.	0.
12	0.	0.	0.	0.	0.	0.	0.	0.
13	0.	0.	0.	0.	0.	0.	0.	0.
14	0.	0.	0.	0.	0.	0.	0.	0.
15	0.	0.	0.	0.	0.	0.	0.	0.
16	0.	0.	0.	0.	0.	0.	0.	0.
17	0.	0.	0.	0.	0.	0.	0.	0.

Initial Age	Percent of Population Using Latrines			
	0	20	60	100
0	45.90	46.11	46.57	47.00
5	52.92	53.13	53.58	54.00
10	49.02	49.10	49.57	49.92
15	44.77	44.92	45.23	45.52
20	40.77	40.89	41.14	41.37
25	37.06	37.15	37.35	37.52
30	33.35	33.41	33.55	33.67
35	29.66	29.69	29.78	29.84
40	26.05	26.06	26.09	26.11
45	22.57	22.58	22.58	22.59
50	19.18	19.19	19.19	19.21
55	15.99	16.00	16.01	16.01
60	13.00	13.00	13.01	13.02
65	10.30	10.30	10.31	10.32
70	7.86	7.86	7.86	7.87
75	5.64	5.64	5.64	5.65
80	3.41	3.41	3.41	3.41

Table 5.10. Cohort Life Expectancies under Different Environmental Conditions

Table 5.11. Number of Man-Days of Sickness to Males under
i) Unchanged Conditions, and
ii) Changed Typhoid Incidence due to 60% Use of Latrines.

MAN DAYS SICKNESS DUE TO TYPHOID IN VILLAGE MALES
UNDER PRESENT CONDITIONS

	AGE GROUP								
PERIOD	0	1	2	3	4	5	6	7	8
1	142.	202.	182.	111.	120.	122.	127.	133.	140.
2	237.	206.	125.	133.	130.	136.	147.	156.	0.
3	242.	142.	150.	144.	146.	158.	172.	0.	0.
4	166.	170.	163.	161.	169.	185.	0.	0.	0.
5	200.	184.	182.	187.	198.	0.	0.	0.	0.
6	216.	207.	211.	218.	0.	0.	0.	0.	17.
7	243.	240.	247.	0.	0.	0.	0.	19.	17.
8	281.	280.	0.	0.	0.	0.	21.	19.	14.
9	328.	0.	0.	0.	0.	23.	21.	15.	12.
10	0.	0.	0.	0.	24.	22.	17.	14.	0.
11	0.	0.	0.	27.	24.	18.	15.	0.	0.
12	0.	0.	30.	26.	19.	16.	0.	0.	0.
13	0.	35.	30.	21.	17.	0.	0.	0.	0.
14	41.	34.	24.	19.	0.	0.	0.	0.	0.
15	39.	27.	22.	0.	0.	0.	0.	0.	0.
16	32.	25.	0.	0.	0.	0.	0.	0.	0.
17	29.	0.	0.	0.	0.	0.	0.	0.	0.

	AGE GROUP							
PERIOD	9	10	11	12	13	14	15	16
1	0.	0.	0.	0.	9.	7.	4.	9.
2	0.	0.	0.	10.	8.	6.	3.	0.
3	0.	0.	12.	10.	7.	5.	0.	0.
4	0.	14.	12.	8.	6.	0.	0.	0.
5	15.	13.	9.	7.	0.	0.	0.	0.
6	15.	11.	8.	0.	0.	0.	0.	0.
7	12.	10.	0.	0.	0.	0.	0.	0.
8	11.	0.	0.	0.	0.	0.	0.	0.
9	0.	0.	0.	0.	0.	0.	0.	0.
10	0.	0.	0.	0.	0.	0.	0.	0.
11	0.	0.	0.	0.	0.	0.	0.	0.
12	0.	0.	0.	0.	0.	0.	0.	0.
13	0.	0.	0.	0.	0.	0.	0.	0.
14	0.	0.	0.	0.	0.	0.	0.	0.
15	0.	0.	0.	0.	0.	0.	0.	0.
16	0.	0.	0.	0.	0.	0.	0.	0.
17	0.	0.	0.	0.	0.	0.	0.	0.

[Note: This table was calculated using a duration of sickness of 43 rather than 162 days. All values are to be multiplied by 3.767 for the present purposes.]

(Table 5.11 continued)

UNDER CHANGED CONDITIONS

PERIOD	AGE GROUP 0	1	2	3	4	5	6	7	8
1	100.	146.	134.	80.	87.	88.	92.	96.	103.
2	112.	102.	59.	63.	61.	64.	69.	76.	0.
3	120.	67.	71.	68.	69.	75.	85.	0.	0.
4	79.	80.	77.	76.	80.	91.	0.	0.	0.
5	82.	75.	75.	77.	85.	0.	0.	0.	0.
6	89.	85.	87.	94.	0.	0.	0.	0.	7.
7	100.	99.	107.	0.	0.	0.	0.	8.	7.
8	114.	119.	0.	0.	0.	0.	9.	7.	5.
9	140.	0.	0.	0.	0.	9.	8.	6.	5.
10	0.	0.	0.	0.	10.	9.	7.	6.	0.
11	0.	0.	0.	11.	10.	7.	6.	0.	0.
12	0.	0.	12.	11.	8.	7.	0.	0.	0.
13	0.	14.	12.	9.	7.	0.	0.	0.	0.
14	17.	14.	10.	8.	0.	0.	0.	0.	0.
15	16.	11.	9.	0.	0.	0.	0.	0.	0.
16	13.	10.	0.	0.	0.	0.	0.	0.	0.
17	12.	0.	0.	0.	0.	0.	0.	0.	0.

PERIOD	AGE GROUP 9	10	11	12	13	14	15	16
1	0.	0.	0.	0.	6.	5.	3.	7.
2	0.	0.	0.	5.	4.	3.	2.	0.
3	0.	0.	6.	5.	3.	2.	0.	0.
4	0.	6.	5.	4.	3.	0.	0.	0.
5	6.	5.	4.	3.	0.	0.	0.	0.
6	6.	4.	3.	0.	0.	0.	0.	0.
7	5.	4.	0.	0	0.	0.	0.	0.
8	4.	0.	0.	0.	0.	0.	0.	0.
9	0.	0.	0.	0.	0.	0.	0.	0.
10	0.	0.	0.	0.	0.	0.	0.	0.
11	0.	0.	0.	0.	0.	0.	0.	0.
12	0.	0.	0.	0.	0.	0.	0.	0.
13	0.	0.	0.	0.	0.	0.	0.	0.
14	0.	0.	0.	0.	0.	0.	0.	0.
15	0.	0.	0.	0.	0.	0.	0.	0.
16	0.	0.	0.	0.	0.	0.	0.	0.
17	0.	0.	0.	0.	0.	0.	0.	0.

5.3.7. Morbidity History of Population

We wish to determine the change in the number of man days of sickness due to typhoid for different proportions of latrine users. From Table 5.9 we can determine the total number of deaths under each environmental regime. Since we know the proportion of deaths attributable to typhoid in each of the periods, it is possible to determine the number of typhoid deaths. We then use a fatality rate of 0.072 and an average of 43 days sickness per attack to determine the total number of man-days of sickness in each case. On Table 5.11 these time streams for present histories are summarized in terms of the average number of man days sickness from typhoid per man year lived.

	Percent of population using latrines			
	0	20	60	100
Average no. of man days sickness due to typhoid per man year lived	1.938	1.628	0.926	0.028

Table 5.12. Typhoid Morbidity

While hookworm is assumed to cause neither death nor confining sickness, we wish to have some measure of the change in the hookworm status of the community. On Table 5.13 below we list the average worm load in the community under different environmental conditions.

	Percent of Population Using Latrines			
	0	20	60	100
Average Per Capita Worm Load in Community	100.00	72.63	26.40	8.48

Table 5.13. Average Worm Load

5.4. ECONOMIC BENEFITS

A review of the literature reveals a lack of consensus on the complex relationship between health and economic development. The assumptions incorporated into this analysis will need to be constantly re-evaluated as new information becomes available.

In their study of the effects of schistosomiasis on St. Lucia, Weisbrod et al. (1973) gave the following taxonomy of output increases due to improved health:

> The output increases due to improved health services can be divided into those that stem directly from individuals enjoying better health and those that flow from the productive agents indirectly affected by improved health services. The direct effects are: (1) increases in outputs due to the decline in absenteeism from work because of illness; (2) output increases associated with the rise in efficiency because of greater physical and mental ability of children and adults; (3) output increments due to extension of working lives. Indirect effects include: (1) reduction in goods and services required to care for the sick; (2) increases in output resulting from freeing of resources previously used by healthy people to avoid sickness; (3) the output resulting from any population increase due to a rise in the birth rate, as might result from better health conditions; and (4) any net output resulting from changes in attitudes and social and political organization that might be a consequence of better health conditions.

As Feldstein (1973) has pointed out in his study of tuberculosis in Korea, "nothing short of a comprehensive economic model would make it possible to pursue this analysis exhaustively and in quantitative terms." In the present analysis we will restrict our attention to some effects of health programs on the most salient factor of production, namely labour. We will

estimate the change in economic output arising from increased working lives, decreased absenteeism and increased on-the-job efficiency. This analysis builds on the demographic model derived earlier and, therefore, evaluates the effects on the presently existing population only.

5.4.1. Economic Assumptions

We make the following economic assumptions:

i) The economic value of livelihood is reflected in the age-specific marginal product of labour.

ii) The beneficiaries of the program are defined as both the survivors (who would live irrespective of the program) and those who would otherwise die. The implication of this assumption is that the contribution to society of the potential survivor is the total monetary value of his or her output (Weisbrod [1973]).

It has been proposed that the individual's future consumption should be deducted from his income in calculating benefits. Muscat and Berg (1974) have shown this proposition to be erroneous since the objective of economic activity is future consumption and not merely the residual after personal consumption. Saunders and Warford (1974) have pointed out that if this "net-output" method is used, a program designed to eliminate elderly unproductive people would generate positive benefits.

iii) Implicit in the above partial equilibrium approach is the assumption that reduction of deaths would not substantially change the age-specific distribution of incomes, the overall rate of growth of incomes, or other important economic parameters.

5.4.2. Marginal Productivity of Labour

The time stream of benefits accruing as a result of a public health program is crucially dependent on the assumption concerning the marginal product of labour.

A number of marginal productivity assumptions have been defended in the literature:

i) Feldstein (1973) has stated that "some economists would assert that in many developing countries the marginal product of labour is nil." The rationale is that in a situation where the limited employment and work opportunities, as perceived by the population,remain unchanged, the ability of a population to work more frequently and vigorously may not be translated into changes in output and earnings (Saunders and Warford [1974]).

In his work on India,Michael Lipton (1968) has pointed out that the existence of a potential labour surplus in agriculture does not mean that the effect of improved health and nutrition on farm labourers can be ignored. In areas where there is a seasonal labour shortage at planting and harvesting times, higher man-hour inputs and increased population vigour could pay off in increased output. The assumption of zero marginal product of labour seems to be extreme and will not be used in this analysis.

ii) In advanced countries with enough land and capital and high employment, Cohen (1973b) considers average income to be a

good indication of marginal product of labour. While Feldstein (1973) assumes that the marginal product of individuals in the productive ages is equal to the average labour income in Korea, a similar assumption would appear to be dubious in Indian conditions.

iii) In Zanzibar, Cohen (1973c) defends the use of the minimum wage paid to unskilled labour as the best estimate of the marginal product of labour. Woolley (1970, 1972) has incorporated a similar assumption into studies of the economic effects of health programs in Panama and the Philippines. While this assumption may be reasonable in the absence of any other data, the minimum wage is likely to be more of a social convention than an economic measure of the marginal product of labour (Cohen [1973]) and is not, therefore, an ideal measure of the marginal product of labour under conditions obtaining in the sub-continent.

iv) Schultz (1966) analysed data from the influenza epidemic in India in 1918 and "estimated that the marginal product of labour is likely to be 30% of the average product" (quoted in Leibenstein [1969]). Since this emanates from a study of the implications of health changes and since the study pertains specifically to India, we will use this estimate in our analysis.

In 1961, the base year for this analysis, the national income was about 133 billion rupees (Shinn [1970]). We assume that all males between the ages of 15 and 60 (about 120 million) are in the

labour force and, following Feldstein (1970), that one quarter of the females aged 15 to 60 would be employed and have the same marginal product as males. The average product per labour force member is thus Rs. 880 per year and the marginal product, by assumption, is Rs. 260 per labour force member per year (in 1961 rupees).

5.4.3. Benefits Due to Extended Working Lives

The nature of sanitation improvement is such (we assume) that the epidemiological effect of improved sanitation is confined to a reduction in the infective parameter. Specifically, we assume that sanitation improvement has no effect on the death rate of those suffering from fecal-borne diseases. Under this set of assumptions, there are no "curative benefits" arising from the health program. We, therefore, need to consider only the preventive effects of the reduction in death rate.

For each time period of five years we determine the difference between the labour force size under "present" and "changed" conditions, multiply this difference by five times the annual marginal product of labour and discount this value to the present. The time stream of undiscounted benefits from the reduction in typhoid fever when 60% of the population use latrines, are listed on Table 5.14.

Table 5.14. Time Stream of Undiscounted, and Sum of Discounted (at 6% p.a.) Benefits due to Extended Working Lives when 60% of the Population Use Latrines

LIFE[illegible] FORE
EXECUTION

WORKING LIVES BENEFITS UNDISCOUNTED

	AGE GROUP								
PERIOD	0	1	2	3	4	5	6	7	8
1	0.	0.	0.	83.	90.	91.	94.	99.	47.
2	0.	0.	271.	269.	271.	279.	296.	192.	43.
3	0.	549.	475.	463.	466.	487.	387.	176.	38.
4	1047.	772.	688.	671.	679.	574.	354.	156.	33.
5	1332.	1038.	948.	931.	778.	525.	314.	132.	0.
6	1629.	1322.	1233.	1029.	711.	466.	266.	0.	0.
7	1945.	1633.	1331.	940.	631.	395.	0.	0.	0.
8	2296.	1732.	1217.	834.	535.	0.	0.	0.	0.
9	2387.	1583.	1080.	707.	0.	0.	0.	0.	0.
10	2182.	1404.	915.	0.	0.	0.	0.	0.	0.
11	1936.	1190.	0.	0.	0.	0.	0.	0.	0.
12	1640.	0.	0.	0.	0.	0.	0.	0.	0.
13	0.	0.	0.	0.	0.	0.	0.	0.	0.
14	0.	0.	0.	0.	0.	0.	0.	0.	0.
15	0.	0.	0.	0.	0.	0.	0.	0.	0.
16	0.	0.	0.	0.	0.	0.	0.	0.	0.
17	0.	0.	0.	0.	0.	0.	0.	0.	0.

	AGE GROUP							
PERIOD	9	10	11	12	13	14	15	16
1	0.	0.	0.	0.	0.	0.	0.	0.
2	-0.	0.	0.	0.	0.	0.	0.	0.
3	0.	0.	0.	0.	0.	0.	0.	0.
4	0.	0.	0.	0.	0.	0.	0.	0.
5	0.	0.	0.	0.	0.	0.	0.	0.
6	0.	0.	0.	0.	0.	0.	0.	0.
7	0.	0.	0.	0.	0.	0.	0.	0.
8	0.	0.	0.	0.	0.	0.	0.	0.
9	0.	0.	0.	0.	0.	0.	0.	0.
10	0.	0.	0.	0.	0.	0.	0.	0.
11	0.	0.	0.	0.	0.	0.	0.	0.
12	0.	0.	0.	0.	0.	0.	0.	0.
13	0.	0.	0.	0.	0.	0.	0.	0.
14	0.	0.	0.	0.	0.	0.	0.	0.
15	0.	0.	0.	0.	0.	0.	0.	0.
16	0.	0.	0.	0.	0.	0.	0.	0.
17	0.	0.	0.	0.	0.	0.	0.	0.

PRESENT VALUE OF INCREASED EARNINGS DUE TO LIFEEXTENSION IN RUPEES AT 6%DISCOUN

RATE FOR EACH AGE GROUP
.21E+04.18E+04.16E+04.15E+04.12E+04.10E+04.73E+03.39E+03.99E+02
*****.38E-03.17E-03.00E+00.00E+00.00E+00.00E+00.00E+00

DISCOUNTED TOTAL BENEFITS AT 6% OF EXTENDED LIVES DUE TO TYPHOID REDUCTIO

.1045E+05RUPEES

5.4.4. Benefits due to Reduced Absenteeism

There are no data available on the effects of hookworm on rates of absenteeism in the labour force and any assumption made in this regard should be treated as tentative.

Perhaps the only empirical study on the relationship between absenteeism and parasitic infection is that conducted by Weisbrod et al. (1973) concerning schistosomiasis on St. Lucia. The hypothesis that workers infested with schistosomiasis worked fewer days per week than those who were not infested was not supported by the data.

The characterization of the existence of schistosomiasis on St. Lucia as "an elaborate compromise between extracting sufficient nourishment to maintain and propogate itself and not impairing too much the vitality or reducing the number of its host, which is providing it with a home and a free ride" (Weisbrod [1973]), is applicable to hookworm in West Bengal, too. On the basis of this admittedly tenuous similarity we will assume that absenteeism in the labour force in West Bengal is unrelated to hookworm infestation.

From the time stream of days sickness due to typhoid under both "present" and "changed" conditions we can determine the difference in the number of man days sickness under the two environmental regimes, and hence determine the increased output emanating from the effect of latrine construction on morbidity from typhoid. For the case when 60% of the population uses la-

trines the time streams of benefits and the total discounted benefit due to reduced absenteeism are presented on Table 5.15.

Table 5.15. Benefits due to Reduced Absenteeism (due to typhoid decline when 60% use latrines)

[Note: as in Table 5.11, all values are to be multiplied by 3.767.]

DUE TO REDUCED ABSENTEEISM

	AGE GROUP								
PERIOD	0	1	2	3	4	5	6	7	8
1	0.	0.	0.	28.	30.	30.	32.	33.	33.
2	0.	0.	59.	63.	61.	64.	69.	70.	0.
3	0.	67.	70.	68.	69.	74.	78.	0.	0.
4	78.	80.	76.	76.	79.	83.	0.	0.	0.
5	105.	97.	96.	98.	100.	0.	0.	0.	0.
6	114.	108.	111.	111.	0.	0.	0.	0.	0.
7	127.	125.	125.	0.	0.	0.	0.	0.	0.
8	149.	143.	0.	0.	0.	0.	0.	0.	0.
9	167.	0.	0.	0.	0.	0.	0.	0.	0.
10	0.	0.	0.	0.	0.	0.	0.	0.	0.
11	0.	0.	0.	0.	0.	0.	0.	0.	0.
12	0.	0.	0.	0.	0.	0.	0.	0.	0.
13	0.	0.	0.	0.	0.	0.	0.	0.	0.
14	0.	0.	0.	0.	0.	0.	0.	0.	0.
15	0.	0.	0.	0.	0.	0.	0.	0.	0.
16	0.	0.	0.	0.	0.	0.	0.	0.	0.
17	0.	0.	0.	0.	0.	0.	0.	0.	0.

	AGE GROUP							
PERIOD	9	10	11	12	13	14	15	16
1	0.	0.	0.	0.	0.	0.	0.	0.
2	0.	0.	0.	0.	0.	0.	0.	0.
3	0.	0.	0.	0.	0.	0.	0.	0.
4	0.	0.	0.	0.	0.	0.	0.	0.
5	0.	0.	0.	0.	0.	0.	0.	0.
6	0.	0.	0.	0.	0.	0.	0.	0.
7	0.	0.	0.	0.	0.	0.	0.	0.
8	0.	0.	0.	0.	0.	0.	0.	0.
9	0.	0.	0.	0.	0.	0.	0.	0.
10	0.	0.	0.	0.	0.	0.	0.	0.
11	0.	0.	0.	0.	0.	0.	0.	0.
12	0.	0.	0.	0.	0.	0.	0.	0.
13	0.	0.	0.	0.	0.	0.	0.	0.
14	0.	0.	0.	0.	0.	0.	0.	0.
15	0.	0.	0.	0.	0.	0.	0.	0.
16	0.	0.	0.	0.	0.	0.	0.	0.
17	0.	0.	0.	0.	0.	0.	0.	0.

PRESENT VALUE OF INCREASED EARNINGS DUE TO REDUCED ABSENTEEISM IN RUPEE

S AT 8% FOR EACH AGE GROUP

.13E+03.14E+03.17E+03.17E+03.15E+03.13E+03.11E+03.74E+02.28E+02
.00E+00.00E+00.00E+00.00E+00.00E+00.00E+00.00E+00.00E+00.00E+00

DISCOUNTED TOTAL BENEFITS OF REDUCEDABSENTEEISM DUE TO TYPHOID REDUCTION =

.1112E+04RUPEES

5.4.5. Benefits due to Increased on-the-job Efficiency

We assume that all workers infected with typhoid are unable to work and that upon return to work their efficiency is unaffected.

Helminthic infestation has been found to affect on-the-job efficiency in empirical studies of schistosomiasis on St. Lucia (Weisbrod [1973]) and hookworm in Indonesia (Basta [1974]). In this section we will derive a relationship between hookworm infestation and on-the-job efficiency.

In a recent study in Indonesia, Basta (1974) found that the output of rubber plantation workers who were paid on an incentive basis, in an area where hookworm prevalence was about 85% and 44% suffered from iron-deficiency anemia, "...could be raised by anything from 8 to 30% by raising hemoglobin levels one or two grams per cent." We will use Basta's data on the relationships between (i) hookworm loads and hemoglobin levels, and (ii) hemoglobin levels and work capacity, to estimate the efficiency increases resulting from a reduction in the hookworm load. Basta's data indicate that for a change in the number of hookworm eggs per gram of feces from about 1000 to 10,000 the hemoglobin level will decline approximately linearly from about 13.55 to about 10.55 gms per 100 ml. If we assume that Stoll's (1962) world average figure of 26 eggs per gram per worm holds in this case, then we have:

$$Hg = 13.883 - 0.888 \times 10^{-2} W, \qquad (1)$$

where Hg is the hemoglobin level in gms/100 ml. and W is the

worm load.

Basta's data on the relationship between monthly incentive payments and hemoglobin levels indicate that these payments rise linearly from about 1500 units at a hemoglobin level of 9.5 to about 2500 units at a level of 15. The implied relationship is:

$$P = -220 + 182\ Hg \qquad (2)$$

where P is the payment.

Combining equations (1) and (2) we find:

$$P = 2330 - 2.24\ W.$$

The elasticity of wages with respect to the worm load, $\frac{\Delta P}{P} / \frac{\Delta W}{W}$, is about 0.106 at W = 100 worms.

To determine the efficiency gains due to reductions in the worm load we divide the population into those who use and those who do not use latrines. For each of these populations we determine the number of man years worked and the corresponding marginal productivity. We multiply this product by 0.106 x (100 - W)% to estimate the increased output due to increased efficiency. On Table 5.16 the time stream and discounted benefits due to increased efficiency when 60% of the population uses latrines are presented.

Table 5.16. Benefits due to Increased Efficiency due to Reduced Hookworm Loads (when 60% of the Population use Latrines)

EFFICIENCY GAINS

PERIOD	AGE GROUP 0	1	2	3	4	5	6	7	8
1	0.	0.	0.	3512	3072.	2761.	2453.	2101.	1757.
2	0.	0.	7731.	6612.	5781.	5145.	4529.	3820.	3130.
3	0.	8199.	6980.	5946.	5149.	4555.	3949.	3262.	2589.
4	9767.	8029.	6809.	5764.	4944.	4307.	3657.	2927.	2219.
5	9567.	7834.	6603.	5538.	4677.	3989.	3282.	2508.	0.
6	9298.	7567.	6318.	5217.	4314.	3565.	2801.	0.	0.
7	8914.	7187.	5908.	4776.	3827.	3020.	0.	0.	0.
8	8466.	6720.	5408.	4237.	3242.	0.	0.	0.	0.
9	7917.	6152.	4798.	3589.	0.	0.	0.	0.	0.
10	7247.	5457.	4064.	0.	0.	0.	0.	0.	0.
11	6429.	4623.	0.	0.	0.	0.	0.	0.	0.
12	5446.	0.	0.	0.	0.	0.	0.	0.	0.
13	0.	0.	0.	0.	0.	0.	0.	0.	0.
14	0.	0.	0.	0.	0.	0.	0.	0.	0.
15	0.	0.	0.	0.	0.	0.	0.	0.	0
16	0.	0.	0.	0.	0.	0.	0.	0.	0
17	0	0	0.	0.	0.	0.	0.	0.	0

PERIOD	AGE GROUP 9	10	11	12	13	14	15	16
1	1442	1130.	836.	0	0.	0.	0.	0.
2	2486.	1865.	0.	0	0.	0.	0.	0.
3	1964.	0.	0.	0.	0.	0.	0.	0.
4	0.	0.	0.	0.	0.	0.	0.	0.
5	0.	0.	0.	0.	0.	0.	0.	0.
6	0.	0.	0.	0.	0.	0.	0.	0.
7	0.	0.	0.	0.	0.	0.	0.	0.
8	0.	0.	0.	0.	0.	0.	0.	0.
9	0.	0.	0.	0.	0.	0.	0.	0.
10	0.	0.	0.	0.	0.	0.	0.	0.
11	0.	0.	0.	0.	0.	0.	0.	0.
12	0.	0.	0.	0.	0.	0.	0.	0.
13	0.	0.	0.	0.	0.	0.	0.	0.
14	0.	0.	0.	0.	0.	0.	0.	0.
15	0.	0.	0.	0.	0	0.	0.	0.
16	0.	0.	0.	0.	0	0.	0.	0.
17	0.	0.	0.	0.	0	0.	0.	0.

Discounted Total Benefits of Increased Efficiency due to Hookworm Reduction (in Rupees) = .1088E + 06.

5.4.6. Total Direct Economic Benefits

Let $D = \frac{(1 + \text{discount rate for economic benefits})}{(1 + \text{productivity growth rate})} - 1.0$

(see Feldstein [1970]);

$U(I+K-1)$ = annual marginal product of labour for workers in initial age group I in period K (where both I and K are measured in 5 year intervals), in rupees.

where:

$U(M) = 0$ for $M = 1,2,3,13,\ldots 17$

and $U(M) = 260$ for $M = 4,\ldots 12$

$S1(I,K)$ = average number of males living in period K from initial age group I under present conditions;

$S2(I,K)$ = average number of males living in period K from initial age group I under changed sanitation conditions;

$TD1(I,K)$ = number of man hours of sickness in period K for cohort I under present conditions;

$TD2(I,K)$ = number of man hours of sickness in period K for cohort I under changed conditions;

$\delta(I,K)$ = increase in efficiency due to reduced hookworm load in period K for cohort I.

For initial age group I in period K the total marginal product is:

i) under present conditions:

$$S1(I,K) \times 5.0 \times 1.25 \times U(M) - \frac{TD1(I,K)}{365} \times 1.25 \times U(M)$$

ii) under changed conditions:

$$[S2(I,K) \times 1.25 \times 5.0 - \frac{TD2(I,K)}{365} 1.25] [1 + \delta(I,K)]U(M)$$

The discounted direct economic gain is, therefore:

$$\Sigma\Sigma[S2(I,K) - S1(I,K)] \times 5.0 \times 1.25 \times U(M)$$

$$+ \quad \Sigma\Sigma\left(\frac{TD1(I,K) - TD2(I,K)}{365}\right) \times 1.25 \times U(M)$$

$$+ \quad \Sigma\Sigma\left(S2(I,K) \times 5.0 - \frac{TD2(I,K)}{365}\right) \times 1.25 \times \delta(I,K) \times U(M)$$

These three terms are the gains due to extension in working lives, due to reduced absenteeism, and due to increased on-the-job efficiency, respectively.

5.5. NUTRITIONAL IMPLICATIONS OF IMPROVED SANITATION

In Chapter One the available evidence on the relationship between malabsorbtion and infection is outlined. With respect to the effect of infection on malabsorbtion there are three major conclusions emerging from a review of the literature:

i) The existence of a relationship between infection and malabsorbtion has been conclusively proved;

ii) The public health significance is probably considerable;

iii) Little quantitative information on this relationship is available.

Few data are available on the quantitative relationship between infection and nutritional wastage. Revelle (in Berg et al. [1973]) refers to estimates (which are based on an examination of the differences between food intake and nutritional requirements) that indicate that as much as 15% of the actual food intake in many poor countries may be wasted. The FAO/WHO Committee on Energy and Protein Requirements (WHO [1973]) increases the obligatory nitrogen losses for adults by 10% to allow for periodic stress (including infection) in normal living and states that infections may also modify requirements for energy and other nutrients. This Committee cautions that "the quantitative effects of infections on the protein needs of an individual cannot be stated, since they are likely to vary with the frequency, severity and nature of the infection and other host factors."

On an Indian diet there is unlikely to be a protein deficiency

if caloric requirements are met (this is less certain for children than for adults [Scrimshaw(1975)]). Since the only suitable quantitative data available pertain to the effect of infection on caloric requirements we will deal with the estimates of the effects of improved sanitation on energy requirements only. If and when similar data become available regarding protein and other nutrients these data can be handled in a similar fashion.

5.5.1. Effect of Typhoid

In their series on the dynamics of health in developing countries (Woolley[1970,1972]), the U.S. Agency for International Development attempts to estimate the increased caloric demand arising from accelerated metabolic rates during febrile illnesses. The Institute for Defense Analysis has estimated (quoted in Wooley [1972]) that the annual basic caloric requirement rises by 5.7% for a single attack of typhoid per year.

The daily physiological requirements for calories by male Bengalis in 1961-1965 are estimated to be (Revelle and Thomas [1970]):

	AGE									
	0-1	1-4	5-9	10-14	15-19	20-29	30-39	40-49	50-59	60+
Calories per capita	950	1050	1375	1940	2820	2495	2420	2340	2130	1830

Table 5.17. Physiological Requirements for Male Bengalis

Earlier in this chapter we estimated the number of typhoid attacks under present and changed conditions. Using this information in conjunction with the above data we find (see Table 5.18) the percentage increase in male caloric requirement under different sanitary conditions.

	Percentage of Population Using Latrines			
	0	20	60	100
Increase in caloric requirement due to typhoid	.07%	.06%	.03%	.01%

Table 5.18. Effect of Typhoid on Caloric Requirements under Different Sanitary Conditions

5.5.2. Effect of Hookworm

The effect of hookworm on both caloric and iron wastage will be estimated.

i) Energy: In a highly controlled test of the effect of hookworm infestation on the nutritional status of Chinese troops, Crowley, Pollack and Brockett (1956) established a statistically significant correlation between the degree of infestation and the average loss in body weight over a period of time. The change in body weight over a 120 day period was 0.9 kilograms greater for those infested (432 subjects with a mean egg count of 1169 per gram of feces) than for those who were not infested (588 subjects). If we take Stoll's (1962) figure of 26 eggs per gram per worm the average burden of those who are infested is 45 worms.

Pollack analysed the data collected by himself, Crowley and Brockett (above) and concluded that "the heavier the hookworm infestation, the greater the loss in body weight." In estimating the caloric wastage emanating from hookworm infestation, we assume (i) that each kg of body weight is equivalent to 7700 calories (Woolley[1970]), and (ii) that the relationship between food wastage and hookworm infestation is linear.

For an adult daily requirement of 2100 cals/day we have 2.75% caloric wastage at a worm load of 45. We have no data on other age groups but assume that this percentage figure remains constant and find that under different environmental conditions the increase in food requirements due to hookworm infestation is:

	Percentage of Population Using Latrines			
	0	20	60	100
Percent increase in food requirements due to hookworm	6.11%	4.43%	1.59%	0.50%

Table 5.19. Effect of Hookworm on Caloric Requirements under Different Sanitation Conditions

ii) Iron: Iron deficiency anemia, often in association with hookworm infestation, is widespread in the tropics (Scrimshaw [1975]). In estimating the economic effects of changes in hookworm infestation we used Basta's (1974) data on the relationship between hookworm infestation and hemoglobin levels in Indonesia. As an intermediate step in the calculations of the economic effects we obtain Table 5.20 below. This table translates the effects of improved excreta disposal practices into medical or nutritional (rather than economic) terms.

	Percentage of Population Using Latrines			
	0	20	60	100
Average no. of worms	100.00	72.63	26.40	8.48
Average Hemoglobin Level: gm/100 ml blood	12.99	13.24	13.65	13.81

Table 5.20. Effect of Sanitation on Hemoglobin Levels

5.5.3. Population Increase and Food Requirements

The relatively rapid population growth in poor countries is accounted for largely by rapidly declining death rates. Concern with the adverse effects of rapid population growth has led to reservations being expressed on the value of health programs in these countries. One of the major arguments in this context is that reduced death rates may exacerbate the problems of feeding the populations of these countries. Implicit in these arguments is the assumption that, while it is possible that improved health may improve the productivity of labour and hence the supply of food, the subsistence requirements for food are proportional to the population size.

Here the "demand" side of the food equation will be examined. There are two major effects which modify the simple linear relationship between population size and food requirements:

i) the age structure of the population changes, and

ii) the degree of food malabsorbtion changes.

[Revelle et al. (1972) have drawn attention to the fact that "cross sectional studies of the States of India during 1968 show a very high degree of correlation between birth rates and death rates." In the model used in this study it is only the presently existing population which is considered. If there is a causal relationship between death rates and birth rates the (positive) effect of this relationship on the food requirements of the population is not included in this analysis.]

	Percentage of Population Using Latrines			
	0	20	60	100
Normalized Requirement with malabsorbtion not taken into account	100	100.37	101.14	101.87
Percentage increase due to: i) typhoid	.07%	.06%	.03%	.01%
ii) hookworm	6.11%	4.43%	1.59%	0.50%
iii) Total	6.18%	4.49%	1.62%	0.51%
Requirement with Malabsorbtion taken into account	106.18	104.88	102.78	102.39
Reduction in total caloric requirement	0%	1.22%	3.20%	3.57%
Percent of food intake which is malabsorbed	5.82%	4.30%	1.59%	0.50%

Table 5.21. Nutritional Consequences of Sanitation Changes

From Table 5.21 we see that if the total food requirement over the lifetime of the presently existing population is calculated without considering caloric wastage, this requirement is 1.87% higher when all of the population use latrines than when no latrines are used. If we take food wastage into account the effect of latrines use is quite different. Now we find that the total caloric requirement in the 100% latrine use case is 3.57% lower than in the case when no latrines are used.

5.5.4. Economic Evaluation of the Nutritional Changes

We can look at the economic effects of altered nutritional status in two ways: i) We can assume food intake to remain constant and estimate the output increases associated with the improved nutritional status which will arise from improved utilization of food by the body. This approach is that which was used in Section 5.4 in that the assessment of economic output increases resulting from changed health conditions implicitly included the effect of altered nutritional status on output.

ii) We can assume that nutritional status remains constant and that improved utilization of food by the body therefore results in decreased food intake and a saving in expenditures on food. In estimating this benefit it is essential to realise that this is not an economic benefit in addition to those estimated earlier, but is the result of a different approach to the assessment of the consequences of a single biological change.

If we assume that 70% of the national income is spent on food in all cases, then the percentage of national income which is lost due to typhoid and hookworm induced malabsorbtion varies from 4.07% when no use is made of latrines to 0.35% when all members of the populations use latrines.

The results of this analysis of the nutritional consequences of changes in excreta disposal practices should be interpreted cautiously. Inferences have been drawn from a sparse and possibly unreliable data set. For instance, the marked effect of hookworm

infestation on body weight in the data used above does not accord with Scrimshaw's (1968) belief that the relative effect of helminthic infections in (at least, protein) malabsorbtion has been overemphasized. It is also important to note that the effect of infection on other nutrients (particularly protein) may be more important than the effect of calories and iron.

In conclusion we stress the major point of this section, namely that an investigation of the effect of health programs on the food situation should not be limited to the supply side of the equation, but that demand (or, more strictly, requirement) considerations, too, should be explicitly analysed.

5.6. CONCLUSION

We may summarize the effects, on the presently existing population in an Indian village of one thousand inhabitants, of reductions in typhoid fever and hookworm infestation emanating from the use of latrines as follows:

	Percentage of Population Using Latrines			
	0	20	60	100
i) Economic Effects:				
Present value (at 6% discount rate) of income of villagers = Rs. 4962 x 10^3				
Present value of Benefits (in Rs.x10^3) due to:				
Extended working lives:	0	3.35	10.45	16.91
Reduced absenteeism:	0	1.32	4.18	6.86
Increased efficiency:	0	40.45	108.80	135.40
Total:	0	45.12	123.43	159.17
Benefits (as % of income)	0	0.91%	2.69%	3.21%
ii) Demographic Effects:				
Life Expectancy at age zero	45.90	46.11	46.57	47.00
Expected days sickness due to typhoid per man year lived	1.94	1.63	0.93	0.03
Average number of worms carried	100.00	72.63	26.40	8.48
iii) Nutritional Effects:				
Reduction in total caloric requirement	0	1.22%	3.20%	3.57%
% of calorie intake which is wasted	5.82%	4.30%	1.59%	0.50%

Table 5.22. Summary of Effects of Changed Excreta Disposal Practices

An important point to note when interpreting these results is that several of the measures are just different perspectives on the same underlying changes. Thus while we report changed hemoglobin levels, because these are of interest to nutritionists, and increased efficiency, which is of interest to economists, it should be recognized that these are not independent effects but that it is the hemoglobin change which induces the efficiency change. Implicit double-counting has to be avoided when evaluating these results.

We should also note that there are important effects of health changes which have not been quantified here. These are discussed in Chapter One and include reduced medical care costs, improved mental ability, fertility changes, alteration of migration patterns and improved utilization of educational facilities.

CHAPTER SIX

SUMMARY, APPRAISAL AND DIRECTIONS FOR FURTHER RESEARCH

Summary

This chapter summarizes that which has been done in each of the previous chapters, suggests what the major shortcomings of this analysis are, and indicates fruitful directions for further research in this area.

CONTENTS

6.1. SUMMARY AND APPRAISAL

It is a basic tenet of public health that the high mortality and morbidity levels prevailing in poor countries are causally related to the level of environmental sanitation in these areas. One of the more prominent of these environmental problems is that pertaining to the removal and disposal of human excreta. While technical solutions to this classic sanitary engineering problem have been devised and successfully implemented in high income countries, the problems associated with the disposal of human excreta in most poor countries and in the rural areas of these countries in particular, have yet to be adequately addressed. In undertaking this dissertation the hope was that a multi-disciplinary approach using some of the concepts and tools of "systems analysis" would provide new insights into the nature of rural excreta disposal problems and that fruitful non-traditional approaches to the practical issues involved would emerge.

The fundamental questions in this field, to which this dissertation is a response, are:

I) What is the appropriate allocation of government resources to the water supply and sanitation sector?

II) What is the appropriate intra-sectoral apportionment of the resources allocated to this sector?

&,III) What specific institutional structures and technological options should be fostered in order to initiate the desired changes in defecation habits?

Chapter One is a descriptive account of the role of sanitation in social welfare. While there is no primary research in this chapter it is useful as an effort to address some of the issues raised by Question I in that the connections between a micro problem and those macro issues which are of direct concern to decision makers are made explicit. This chapter is useful in the context of this dissertation in that it specifies the overall matrix a few of the elements of which are explored in the subsequent chapters.

In Chapters Two through Five we work our way from the micro level to the macro level, examining in depth a few of the many pertinent questions which arise.

In Chapter Two the economic concepts which have been developed for examining environmental problems in developed countries are applied to develop a framework in which to evaluate sanitation programs. Those programs which have been undertaken in rural India are examined in terms of this framework. The fundamental assumption is that the crucial factor obstructing the transformation of defecation practices in rural India is neither the absence of an appropriate technology nor the existence of a set of anachronistic beliefs but is the perception by the populace that there are no significant tangible benefits which will accrue to the individual if that person changes his or her traditional defecation habits. Particular attention is focussed on the factors upon

which individual motivations to change traditional habits are contingent. This orientation is quite different from that which has been, and is, the norm used by planners of environmental health works and gives rise to a radically different set of attractive institutional and technological options. In Chapter Two an attempt is made to quantify the orders of magnitude of the "internal" benefits for a few of the more promising options. In summary, then, Chapter Two contributes substantially to a clarification of the way in which micro-level change may be promoted (the concern of Question III).

The analysis in Chapter Two suggests that the most propitious technological options may be those in which human excreta is treated as an agricultural and energy resource rather than as an undesirable waste product. Chapter Three consists of a more detailed analysis of the potential and actual role of night soil as a fertilizer source, and an assessment of the effects of the agricultural regime on the internal benefits to be derived from a composting operation in a village in West Bengal. Perhaps the major contribution of this exercise is the collection and refining of much of the basic data on which any analysis of the economic viability of the proposed programs must be based. The chapter is also useful in developing an analytic method for appraising the tangible benefits associated with "excreta-use" programs and in assessing the effects on these benefits of policy decisions in this sector (e.g.

subsidies of communal latrines and bio-gas plants) and exogenous changes (e.g. a change in the price and availability of chemical fertilizers or a change in the crop types in the area). This type of analysis is useful, too, in identifying critical data deficiencies.

The most important single factor inhibiting the rational allocation of resources within the public health sector (the concern of Question II) is the absence of quantitative estimates of the linkages between health interventions and disease incidence. In Chapter Four this complex problem is addressed through the medium of mathematical epidemiological models. A set of simple analytic models for different diseases are developed which are useful in interpreting the results of several existing complex simulation models and in elucidating the nature of the implicit equilibrating mechanisms. An important consequence of this enquiry is the demonstration of the necessity for including non-linearities (such as density dependent effects or immunity) in models of soil-transmitted helminths. It is suggested that the exclusion of these ecological factors in the other systems examined constitutes a major limitation of these models.

Where the etiology of a disease is clearly understood these models are useful both in simulating the effects of parameter changes and in identifying paramount data deficiencies. These

models are expected to contribute to empirical investigations into the health consequences of environmental changes by specifying the expected form of the relationships between various disease measures and the independent variables which may affect these measures, thus ensuring that statistical (e.g. multiple regression) models reflect the etiological realities of different diseases. Where the etiology is unclear (as in the case of the diarrheas) the assumptions which must be built into the model are unclear and valid simulation models can not be constructed. These models can be used in this case, however, to facilitate the choice of the appropriate statistical model for an empirical analysis. This would be done by using the full range of the epidemiological models which may be appropriate to test the sensitivity of different statistical forms to the underlying structural assumptions.

The bulk of this chapter is devoted to the development of a set of deterministic simulation models for hookworm infestation in rural West Bengal. These models proved highly useful in providing a framework in which to organize the considerable body of data on hookworm which has been assembled by parasitologists and epidemiologists. In the present context the models are useful in providing a mechanism for mapping sanitation changes into changes in the level of infestation in a community. The usefulness of these models for purposes of policy formation will remain severely limited until a body of data relating

sanitation changes to changes in the incidences of different diseases becomes available.

In Chapter Five an attempt is made to quantify some of the health effects of a change in defecation practices in demographic and economic terms (the concern of Question I). Most previous analyses of this sort have taken the health changes as given; a feature of the present approach is the coupling of epidemiological and demographic models. Given the paucity of adequate epidemiological models this chapter quantifies the effects of the changes,in only typhoid fever and hookworm infestation,which may arise from changed sanitation practices. While little light is shed on the many problems which arise in assessing the economic consequences of health changes in poor countries, the latter part of this chapter indicates the ways in which existing data may be manipulated to obtain meaningful estimates of the economic and nutritional consequences of changes in defecation habits.

In concluding this summary and appraisal we wish to emphasise those areas in which the analysis attempted in this dissertation has floundered.

In assessing the internal benefits of different excreta disposal programs the validity of the analysis has been severely proscribed by the paucity of empirical data on the role of organic fertilizers in agriculture and on the valuation

of these fertilizers by farmers. A more thorough understanding of the rural, non-commercial energy sector, too, is essential.

A comprehensive analysis of the effects of altered sanitary conditions on health is not possible at this stage. This is an extremely complex question to which clear universal answers are unlikely to emerge. The approach taken in this dissertation suggests that there are several areas in which research is needed.

The art of building useful mathematical models lies in perceiving clearly the trade-off between simplicity and replication of reality. Our examination of existing mathematical epidemiological models suggests that many of these models may be both too simple and too complex. They are too complex in that the number of states and connections structured into the models is so great that the underlying dynamics of the system are obscured. The models are too simple in that the relationships are conceived of as linear and "chemical" rather than as non-linear and "ecological" in nature.

The type of data which are available for calibrating epidemiological models reflect the underlying systemic assumptions and a crucial deficiency is thus in information pertaining to the nature of the non-linearities in these systems.

The development of a complete range of disease models

awaits, too, the development of more precise knowledge on the etiology of a number of important communicable diseases.

When these models are visualized as tools which may assist in the formation of policy, the absence of reliable empirical information relating changes in public health measures to changes in the incidences of the significant diseases becomes paramount.

An attempt to translate health changes into demographic and economic terms raises many issues which will be clarified only when far more extensive empirical analyses are performed. The quantitative effect of mortality changes on fertility, the marginal productivity of labour in situations of wide-spread un- and under-employment, and the effects of socio-economic constraints are among the more substantial issues requiring clarification. The few data on the relationships between infection and malabsorbtion which are available suggest that these relationships may be of great significance in public health in poor countries. This is clearly an area in which empirical research should be given high priority.

6.2: DIRECTIONS FOR FURTHER RESEARCH

This dissertation is the first step in an ongoing study of the role of water supply and sanitation in rural public health in the Indian sub-continent. The next phase of this work will include a large component of field work and is expected to concentrate on the following three areas:

i) An Investigation of the Relationship between Changes in Domestic Water Supply and Excreta Removal and Disposal and Changes in Health:

Bradley (in White et.al.[1972]) has devised an interesting and useful system for classifying diseases associated with water as water-borne, water-washed, water-based and water-related. Taking cognizance of this categorization we will construct a simple mathematical model (where the etiology is clear) or set of models (where the etiology is unclear) for each relevant disease. Luecke and McGinn (1975) have evaluated regression analysis as a tool in educational research. Following their approach we will simulate the disease regime under a set of parameter assumptions to generate data similar to those which may be obtained through longitudinal research. We will analyse both the longitudinal and cross-sectional data using multiple regression techniques. Our ambition is to see whether the specified regression functions enable us to draw satisfactory inferences concerning the underlying causal

structure.

Assuming that the theoretical analysis reveals appropriate ways of structuring the regression models, a similar analysis would be performed on cross-sectional and longitudinal data. It is anticipated that this analysis will be performed for a rural population in Bangladesh for whom a good longitudinal data base exists.

The objective of this exercise is both to estimate the effects of different public health interventions in a particular environment and to develop a methodology for quantifying these effects through inexpensive studies in a wide variety of situations. In using an unusually rich data base we hope to be able to establish the relationships between the inferences which can be drawn from cross-sectional studies and those emerging from the time series data. This connection is important since the longitudinal data may facilitate more reliable causal analysis, while it is feasible to collect only cross-sectional data in any rapid and inexpensive survey.

A second approach to estimating these relationships in a particular situation would be to collaborate with an agency which is improving domestic water supply and/or excreta disposal facilities on a prospective study of the impact of these changes.

The above analysis focusses on Question I (section 6.1). The two following research projects concentrate on the means

to be used to improve water use patterns and excreta disposal habits (the concern of Question III).

ii) An Investigation of the Relationship between Availability and Use of Domestic Water:

In the above analysis the water supply variable which needs to be measured is actual water use, in terms of quantity used, how it is used and water quality. While the relationship between facilities and use may be close in urban areas and in arid rural areas, this relationship becomes complicated in rural areas where alternative sources of water are available. An analysis of the relationship between actual water use and the accessibility of water, the technology by which the water is acquired and distributed, and the quality as perceived by the user may be informative with respect to the means to be used to improve patterns of water use. While useful guidelines emerge from a study by White, Bradley and White (1972) in East Africa, the analytic framework in which such an analysis would be set is not clear.

iii) An Investigation of the Relationship between Provision of Excreta Disposal Facilities and Use:

This project would be a research-cum-action project involving the setting up of a pilot project using an innovative method of excreta removal and disposal, such as a communal latrine

connected to a bio-gas plant, to see whether the theoretical advantages of such a scheme can be translated into a workable solution to rural excreta removal and disposal problems. The analysis performed in Chapters Two and Three gives guidelines both for the structure of such a project and for the collection of subsequent data.

APPENDICES

APPENDIX 2. VILLAGE LATRINE-CUM-BIO-GAS SCHEMES

The design standards and data used below are based on some analysis carried out in Chapter Three of this dissertation and on the work of Parikh(1963,1974), Prasad, Prasad and Reddy (1974), the Central Public Health Engineering Research Institute (1970b), the New Alchemy Institute (1973), Jalal (1968), Singh (1972,1973), Mohanrao (1974a), Patel (1970), the Khadi and Village Industries Commission (1975), Bhatia and Mehta (1975) and Watt and Merrill (1963).

A.2.1.1: The Production of Methane from Village Wastes

a) Rice Straw:

Singh suggests that for each pound of rice straw added to the digester and additional seven cubic feet of bio-gas are generated. We wish to check the plausibility of this figure.

The New Alchemy Institute indicates that dry algae are 50% digestible. We have no data on the digestibility of rice straw and tentatively assume that 50% is a resonable figure. The molecular structure of the straw is likely to be largely $C_6H_{12}O_6$ which implies that 40% of the

dry weight is carbon (since the atomic weights of carbon, oxygen and hydrogen are 12, 16 and 1 respectively). This is exactly the empirical figure given by Jalal. The decomposition of this organic compound is represented by the equation: $C_6H_{12}O_6 = 3CH_4 + 3CO_2$
We therefore expect equal volumes of CH_4 and CO_2 to be generated. Filed data corroborate this (see, for instance, the New Alchemy Institute publication) in that the CH_4 proportion of the bio-gas is somewhat lower that the 60% which is standard for gas generated from human feces.

For each pound of straw added we therefore have 0.20 lbs. of carbon (50% in the form of methane) released. Carbon comprises 75% (12/16) of the weight of methane while in carbon dioxide this proportion is 27.3% (12/44). The specific weights of methane and carbon dioxide are 0.0424 and 0.1170 lbs/ft^3 respectively (Rouse 1950). The volume of the methane generated is thus 3.15 cubic feet (0.10/(0.75x0.0424)) and the total volume of bio-gas is 6.30 cubic feet. This calculation suggests that Singh's figure is of the right order (if the 50% digestibility assumption is appropriate). In the analysis which follows we assume that one pound of straw (dry weight) will generate six cubic feet of bio-gas, 50% of which will be methane.

In the process of digestion an energy balance, too, must be maintained. If we assume the dry straw to contain 4000 kcals./kg., one pound of straw contains 1814 kcals. The 3.15 cubic feet of methane contain 765 kcals.(the calorific value of methane is 243 kcals./ft^3). Half of the original weight remains in the digester in the form of sludge (largely bacteria). Watt and Merrill present data which suggest that a variety of substances (for instance sea bass, rice, scallops and shrimp) contain 4000 kcals/kg dry weight. We assume the sludge to have this calorific value and we therefore have 907 kcals in the half pound (dry weight) of sludge. The remaining energy is the energy used by the bacteria in the fermentation process. Our calculations suggest that this energy is quite low, specifically only 142 kcals. While I was unable to get corroborating evidence on this imputed energy use by the bacteria, we know that very little heat energy is given off in the process of anaerobic digestion, and tentatively suggest that the energy balance seems reasonable.

b) Night Soil:

Data from existing bio-gas plants suggest that the anaerobic digestion of night soil generates one cubic foot of bio-gas per capita per day and that 60% of the gas is

methane (the remaining 40% being carbon dioxide). In this process 70% of the volatile solids in the human excreta are digested.

For each pound of carbon added to the digester we have 0.70 lbs of carbon liberated. Since 60% of the volume is methane and 40% is carbon dioxide and since one mole of each of the gases occupies the same volume, 0.42 lbs of carbon are in the form of CH_4 and 0.28 lbs in the form of CO_2. The weights of methane and carbon dioxide are thus 0.560 and 1.027 lbs and we have 13.2 cubic feet and 8.78 cubic feet, respectively, of the gases.

Assuming that all of the feces (19.8 gms of carbon per capita daily) and 40% of the urine (40% of 6.5 gms of carbon per capita per day) are added to the plant, we have 22.4 gms carbon per capita per day, or 0.0493 lbs carbon per capita daily added to the digester. The expected volume of the bio-gas generated is thus 1.08 cubic feet which is almost exactly the figure given empirically.

A.2.1.2: Value of Bio-gas in Different Uses.

a) Cooking:

If the bio-gas is used for cooking this gas can be valued (in 1974 prices) in terms of three alternative fuel sources:

i) Dung cakes: Dung cakes have a calorific value of 1500 kcals per pound and are burnt at 11% efficiency in a "chullah". Given that the value of cow-dung cakes is about Rs55/ton, the cost of effective energy is about Rs 0.15 per thousand kcals.

ii) Electricity: The efficiency of electricity use is about 76%. Since one kwh is equivalent to 860 kcals and the price of electricity is between 10 and 30 paise per kwh, the cost of energy in this form is between Rs 0.15 and Rs 0.45 per thousand effective kcals.

iii)Kerosene: The calorific value of kerosene is 4900 kcals per pound, the burning efficiency 48% and the cost Rs 1.20 per kg. The cost of energy is thus Rs 0.23 per thousand effective kcals.

Since bio-gas is a clean and convenient source of energy for cooking it is likely to be valued more highly than the traditional fuels per thousand effective kcal (Richard Meier - personal communication) and we assume the

value to be Rs 0.25 per thousand effective kilocalories. One cubic foot of gas is thus worth Rs 0.020 as a cooking fuel if 55% of the gas is methane.

b) As Fuel for a Diesel Engine:

Bhatia and Mehta give the price of light diesel as Rs 0.80 per litre and indicate that 0.25 litres of diesel are required per horse power hour (an implicit efficiency of 23%). The price of this form of energy is thus Rs 0.27 per kwh or Rs 0.32 per thousand effective kcals. The Khadi and Village Industries Commission state that 15 cubic feet of bio-gas (55% methane) are required to deliver one hp.hr. when driving a diesel engine (an implicit efficiency of 32%). We thus require 20 cubic feet of bio-gas per kwh and one cubic foot of gas (55% methane) is worth Rs 0.0133.

A.2.1.3: Alternative Latrine-cum-bio-gas Schemes

In this section I will examine several alternative latrine-cum-bio-gas schemes in terms of the costs and benefits (in 1974 prices) incurred by those who may operate the schemes.

A.2.1.3.1: Providing Fuel for Cooking:

An interesting institutional framework appears to be one in which an entrepreneur constructs a communal latrine and attached bio-gas plant and pays villagers a certain amount for consistent use of his latrine. The entrepreneur sells the bio-gas and fertilizer. If the economics are such that the entrepreneur can make a reasonable profit on the operation, this arrangement could provide the basis for self-sustaining change in defecation habits. The incentive to villagers to participate is clear and there is an incentive to the operator to maintain the facility adequately since his profit is dependent on use of the facility. This arrangement was suggested to me by Dr.K.L.Rao (1975) who had learnt of such a program in a village in Maharashtra. In a recent study on energy and agriculture Makhijani (1975) has made a similar suggestion: "If the operation is conducted on a non-profit basis, an annual payment could be made to each family....to induce people to use the latrines."

We examine the case in which the facility consists of two latrine units (for males and females) each containing six squatting plates. Since the squatting plates serve about 20 people each, the population served by this arrangement is 240 people or nearly 50 households. We will examine this alternative

when human excreta only is used in the plant and when straw is added to the human wastes.

a) With human excreta only(no rice straw) added to the plant, and with the facility owned by an entrepreneur:

We assume that all fecal matter (2.3 gms N and 19.8 gms C per capita per day) and 40% of the urine (40% of 7.75 gms N and 6.5 gms C per capita daily) are added to the plant. The per capita daily gas production is one cubic foot and the total daily production (and plant capacity) is thus 240 cubic feet per day. Assuming that a person requires eight cubic feet daily for cooking purposes (K.V.I.C.) this gas plant would meet the cooking requirements of about 5 or 6 families. These families would presumably be in close proximity to the plant and we thus expect no distribution problems.

Benefits: About 4.34 lbs N are added per capita per year. Assuming all the nitrogen to be retained and the price of nitrogen to be Rs 4.40 per kg, the nitrogen value of the sludge (probably an underestimate of the total value of the sludge) is Rs 8.66 per capita per year. Since the gas is 60% methane, since the calorific value of methane is 243 kcals per cubic foot and since the fuel is burnt at 60% efficiency, the plant yields 32,000 kcals of effective energy per capita

per year. Valued at Rs 0.25 per thousand effective kcals, the value of the fuel generated is thus Rs 8 per capita annually. The total annual income from this plant would thus be Rs 4000 of which 48% is attributable to the sale of fuel and 52% to the sale of fertilizer.

Costs: The gas plant has a capacity of 240 cubic feet per day. Given that a 70 cubic foot plant costs Rs 2332 (including the costs of appurtenances) while a 5000 cubic feet plant costs Rs 58000 (in 1974 prices), we have:

$$\text{Capital Cost (Rs)} = 95.14(\text{Capacity in cub.ft./day})^{0.753}$$

The capital cost is thus Rs 5897. The maintenance cost is Rs 0.475 /yr/cu.ft/day while the labour cost is estimated to be Rs 1.10/yr/cu.ft/day. Assuming a 10% discount rate and a 30 year lifespan, the annualized bio-gas plant costs are:

Capital		Rs 625/yr	
Labour		Rs 264/yr	
Maintenance		Rs 114/yr	
Total		Rs 1003/yr	(without subsidies)
	or	Rs 847/yr	(with a 25% KVIC subsidy on capital costs)

The cost of the two units of six latrines each is Rs 3000. Planning Research and Action Institute data indicate that the monthly cost of cleaning these latrines would be only Rs 5. Since the entrepreneur will be trying to make use of his latrine attractive to villagers we assume that the quality of maintenance will be higher than that implied by the PRAI figure. We assume the monthly maintenance figure to be Rs 10 .

We assume that the entrepreneur who is operating the facility pays villagers Rs 1 per month each for consistent use of his latrines and that this sum is sufficient to induce villagers to participate in the scheme. The total annualized costs both with and without the presently available subsidies (25% on the capital cost of the gas plant and a full subsidy on the cost of the communal latrines) are given along with the benefits on the table below:

	without subsidies	with subsidies
Gas Plant	1003	847
Latrines	438	120
User Payments	2880	2880
Total Cost (Rs/yr)	4321	3847
Total Benefits (Rs/yr)	4000	4000
% Return	-7%	+4%

We thus conclude that an entrepreneur who used only human excreta in a bio-gas plant and who paid villagers one rupee a month each to use his latrine could not make a profit if he bore all of the capital costs, and could make a small return on his investment if the state provided a full subsidy on the latrine cost and a 25% subsidy of the bio-gas plant cost.

b) Using both human excreta and rice straw in the digester:

If rice straw alone were used in a bio-gas plant little digestion would take place due to the high carbon to nitrogen

ratio. When mixed in suitable quantities with night soil, however, the C/N (carbon to nitrogen) ratio can be brought down into the vicinity of the "biological optimum" of about C/N = 30.

The lowest estimate of available rice straw per capita (see Chapter Three) in Bengal is 390 lbs per annum. In this analysis we assume that one pound of rice straw per day is added for each person using the latrine. The human waste generates one cubic foot of gas per capita per day (60% CH_4) while the pound of straw generates and additional six cubic feet of gas (55% CH_4). The amount of gas generated per capita is thus almost equal to the per capita demand for cooking purposes. Distributional constraints limit the number of dwellings which can be served by a single gas plant. The number of houses which can be served depend on the settlement pattern, but will be assumed to be limited to about eight in this analysis. There is a "lumpiness" involved in latrine construction. Since we assume that twenty people can make use of a single squatting plate a single plate would mean a plant of 140 cu.ft/day capacity serving about 3 households, while two plates (one for males and one for females) and a 280 cu.ft/day plant would serve 35 people or about 7 households. The latter arrangement seems preferable in financial, space and acceptability terms and will be used in the remainder of this

Benefits: We have 146 kcals (from the feces and urine) and 729 kcals (from the straw) generated per capita per day. The effective heat supplied by the gas (at 60% burning efficiency) is thus 192,000 kcals per capita per year. Since 40 people are using the latrines and the gas is valued at Rs 0.25 per thousand effective kcals, the total value of the gas is Rs 1920 per annum. Since 365 lbs of dry straw contain 1.83 lbs of nitrogen the total annual nitrogen addition is 6.17 lbs per capita and the value of the sludge is Rs 494. The total return is thus Rs 2410 per annum (80% of which is attributable to the value of the fuel).

Costs: The two latrine units cost Rs 500. Including maintenance costs the annual expenditure amounts to Rs 73. The gas plant has a capacity of 280 cu.ft/day and therefore costs about Rs 6623. The maintenance and labour costs amount to Rs 441 per annum and the annualized capital costs of the gas plant are Rs 702. The total cost is Rs 1143 per annum with no subsidy and Rs 968 per annum with the 25% KVIC subsidy on capital costs.

On the assumption that one pound of rice straw is added per capita per day, Jalal's cost formula (see Chapter Three) suggests that nearly Rs 30 per ton (in 1968 prices) would be paid for this straw. In 1974 prices the cost of straw would

be about Rs 45 per ton on this basis. Recent data presented by Parikh indicate that Rs 55 per ton is paid for dried cow-dung cakes. Since the calorific value and burning efficiencies of these energy sources are similar but cow dung cakes are a preferred fuel, Jalal's formulation appears to be satisfactory, and we will use a value of Rs 45 per ton of rice straw in this analysis. Since we add 6.52 tons of straw annually in this case the cost is Rs 293 annually.

With this latrine-cum-bio-gas scheme we can envision either the householders grouping together and constructing a scheme for their own use or an entrepreneur setting up the scheme as in the previous example. We investigate both of these options.

i) With the facility owned by the community:

The total annualized costs and benefits are:

	without subsidies	with subsidies
Gas plant	1143	968
Latrine	73	20
Straw	293	293
Total Costs (Rs/yr)	1509	1281
Benefits (Rs/yr)	2410	2410
% Return	60%	88%
Initial Investment (Rs)	7123	5467
Years to pay back investment	4.2	3.2

This is clearly a highly profitable community investment, yielding per capita profits of over Rs 22 and Rs 28 per annum in the two cases.

ii) With the facility run by an entrepreneur:

Investigating the cases where one and two rupees respectively are paid monthly to participants for defecation in the latrines we have:

Paying Rs 1/month:	without subsidies	with subsidies
Total Costs (Rs/yr)	1989	1761
Benefits (Rs/yr)	2410	2410
% Return	21%	37%
Initial Investment (Rs)	7123	5467
Years to pay back investment	6.0	4.6
Paying Rs 2/month		
Total Costs (Rs/yr)	2469	2241
Benefits (Rs/yr)	2410	2410
% Return	-2.3%	7.5%

If the entrepreneur pays Rs 1 per month to participants this is an attractive investment, particularly if the existing subsidies are forthcoming. If it is found necessary to pay as much as Rs2 per month to induce villagers to participate in the scheme the investment is not one which would be attractive to an entrepreneur.

A.2.1.3.2: Providing Fuel for a Diesel Engine

Diesel engines are used in rural India for small scale industrial and irrigation purposes. In this analysis we examine the case where a demand for diesel exists and where

substitution of methane for diesel is feasible. Since the demand is centralized we assume that the likely institutional structure is one in which an entrepreneur runs the facility along the lines previously examined.

We consider a scheme similar to that analysed earlier in whichwe have a communal latrine serving 240 people and in which we add one pound of rice straw per capita per day. The daily gas production is 1680 cubic feet.

Benefits: Since the gas is 51.4% methane the value as a diesel substitute is Rs 0.0126 per cubic foot. If the demand for the gas is non-seasonal, such as may be the case for small scale industrial purposes, the annual value of the gas generated is Rs 7736. If we assume that the engine is used for irrigation (there would be sufficient gas to drive about a two cusec pump which was run for eight hours daily) and that there are two growing seasons of 120 days each, then the value of the gas is Rs 5157 per annum. Since the total nitrogen addition is 6.17 lbs per capita per year the value of the sludge is Rs 2961 and the total annual revenue (with a continuous fuel demand) is Rs 10697. Using the mathane for irrigation the annual revenue is Rs 8118.

Costs: The annualized capital cost of the communal latrine

is Rs 318 and the labour cost is Rs 120 per year. The capital cost of the gas plant is Rs 25528 or Rs 2707 per annum while the annual labour and maintenance cost is Rs 2646. The annual straw cost is Rs 1759. We have:

	without subsidies	with subsidies
Gas plant (Rs/yr)	5353	4676
Latrine (Rs/yr)	438	120
Straw (Rs/yr)	1759	1759
User Costs (Rs/yr)	2880	2880
Total Costs (Rs/yr)	10430	9435
Benefits: Continuous use:	10697	10697
Irrigation:	8118	8118
% Return: Continuous use:	+2.6%	13.4%
Irrigation:	-22%	-14%

If it is necessary to pay villagers one rupee a month each to participate in the scheme this would be an attractive investment only if the subsidies were available and if there were a continuous demand for the gas.

A.2.1.4: The Sources of Additional Benefits

It is interesting to look into the sources of the benefits when the traditional practice of burning rice straw in a chullah is replaced by feeding the material with human excreta into a bio-gas plant. We examine the case in which we have a community-owned facility providing cooking fuel to those families who use the latrines.

There are three sources of additional pecuniary benefit from using the straw in the plant rather than burning it: i) Assuming that the straw contains 400 kcals per kg, that it costs Rs 45 per ton and that it is burnt at 11% efficiency, the implicit value of the fuel is Rs 0.10 per thousand effective kcals. Since bio-gas, a high quality fuel, is valued at Rs 0.25 per thousand effective kcals, there is a 148% increase in the value of the fuel due to the "quality" factor alone. ii) Under the traditional practice one pound of dry straw yields 200 effective kcals while using a bio-gas plant and assuming 6 cubic feet of gas (50% methane) per pound of straw, we get 437 effective kcals, an increase of 119%. iii) The multiplicative effect when both quality and quantity increase simultaneously increases the value of the energy by a factor of 176% (148%x119%).

The additional benefits from using the night soil and rice straw in a gas plant are accounted for as follows:

	Rs/year	%
Fertilizer from excreta	347	16.4
Fertilizer from straw	147	7.0
Fertilizer Total	494	23.4
Fuel from excreta	320	15.1
Fuel from straw:		
"Quality" effect	434	20.5
"Quantity" effect	349	16.5
"Multiplicative" effect	516	24.4
Fuel Total	1619	76.6
Total Improvement in Resource Use	2113	100.0

(When adding the original fuel value of the straw, Rs 293, we get the total revenue of about Rs 2410 as required.)

An interesting point to note from the table above is that only about one-fifth of the total improvement in resource use emanates from the increased quantity of energy liberated from the straw.

A.2.1.5: Conclusion

The calculations presented in this appendix indicate that the most promising arrangement is one in which the community owns and operates a plant which is fed with both human wastes and rice straw. With presently available subsidies a community of about 6 families could meet almost all of their cooking requirements and recoup an investment of about Rs 5500 in about three and a half years. If a centralized, non-seasonal demand for bio-gas as a substitute for diesel exists (e.g. for small scale industrial purposes) an entrepreneur could construct a latrine-cum-bio-gas facility, pay villagers one rupee a month each for consistent defecation in his latrine and make about a 13% return on his investment.

APPENDIX 4: HOOKWORM MODEL APPENDICES

Appendix 4.1.

If we consider x_t to be the proportion of susceptibles and define ϕ and β accordingly, the differential equation describing the system is:

$$\frac{dx_t}{dt} = \phi y_t - \beta x_t y_t = \phi(1-x_t) - \beta x_t(1-x_t)$$

$$\frac{dx_t}{dt} = \phi - (\phi+\beta)x_t + \beta x_t^2 \qquad (1)$$

At equilibrium $\frac{dx_t}{dt} = 0$.

therefore $$x_t^* = \frac{\phi + \beta \pm \sqrt{\phi^2 + 2\phi\beta + \beta^2 - 4\phi\beta}}{2\beta}$$

whence $$x^*_t = \frac{\phi}{\beta}, 1$$

Appendix 4.2.

We may wish to know the rate at which the above system will approach equilibrium from a given initial probability distribution.

The differential equation (1) is separable:

$$\int \frac{dx}{\beta x^2-(\phi+\beta)x+\phi} - \int dt = c$$

The value of $\int \frac{dx}{ax^2+bx+c} = f(x)$ is

$$f(x) = \frac{1}{\sqrt{b^2-4ac}} \ln \frac{2ax+b-\sqrt{b^2-4ac}}{2ax+b+\sqrt{b^2-4ac}} \qquad \text{for } b^2 > 4ac$$

$$f(x) = \frac{2}{\sqrt{4ac-b^2}} \tan^{-1} \frac{2ax+b}{\sqrt{4ac-b^2}} \qquad \text{for } b^2 < 4ac$$

$$f(x) = \frac{-2}{2ax+b} \qquad \text{for } b^2 = 4ac$$

In our system $b^2 - 4ac = (\phi-\beta)^2 \geq 0$, and we therefore have, for $\phi>\beta$ and $\phi<\beta$:

$$\frac{1}{(\phi-\beta)} \ln \frac{2\beta x-(\phi+\beta)-(\phi-\beta)}{2\beta x-(\phi+\beta)+(\phi-\beta)} - t = c$$

i.e. $$\frac{1}{(\phi-\beta)} \ln \frac{(\beta x-\phi)}{(\beta x-\beta)} - t = c$$

and $$c = \frac{1}{(\phi-\beta)} \ln \frac{(\beta x_0-\phi)}{(\beta x_0-\beta)}$$

therefore, $$\ln \frac{(\beta x_t-\phi)/(\beta x_0-\phi)}{(x_t-1)/(x_0-1)} = t(\phi-\beta)$$

$$\text{whence } x_t = \frac{\phi(x_o-1) - (\beta x_o-\phi)e^{-t(\beta-\phi)}}{\beta(x_o-1) - (\beta x_o-\phi)e^{-t(\beta-\phi)}} \qquad (2)$$

For $\beta>\phi$:

$$\lim_{t\to\infty} x_t = \frac{\phi}{\beta} \qquad \text{for all } x_o < 1$$

For $x_o = 1$

$$x_t = \frac{\phi(o)-(\beta x_o-\phi)e^{-t(\beta-\phi)}}{\beta(o)-(\beta x_o-\phi)e^{-t(\beta-\phi)}} = 1 \qquad \text{for all } t$$

For $\beta<\phi$:

Let $x_t = \frac{f(t)}{g(t)}$ where $\lim_{t\to\infty} f(t) = \infty$ and $\lim_{t\to\infty} g(t) = \infty$

Using l'Hopital's Rule:

$$\lim_{t\to\infty} \frac{f(t)}{g(t)} = \lim_{t\to\infty} \frac{f'(t)}{g'(t)}$$

$$\text{therefore, } \lim_{t\to\infty} x_t = \frac{(\beta x_o-\phi)(\beta-\phi)e^{-t(\beta-\phi)}}{(\beta x_o-\phi)(\beta-\phi)e^{-t(\beta-\phi)}} = 1$$

For $\beta=\phi$

$$\frac{-2}{2\beta x_t-\phi-\beta} - t = \frac{-2}{2\beta x_o-\phi-\beta}$$

$$\text{therefore, } x_t = \frac{1}{2\beta}\left(\frac{2\beta x_o-\phi-\beta}{-4\beta x_o t+2\phi t+2\beta t+4} + \phi+\beta\right) \qquad (3)$$

$$\lim_{t\to\infty} x_t = \frac{1}{2\beta}(\phi+\beta) = 1 \qquad \text{(since } \phi=\beta.\text{)}$$

These equilibrium conditions are those which were previously derived more directly. Equations (2) and (3) facilitate computation of the approach to the equilibrium solution.

Appendix 4.3.

$$\frac{\Delta x_t}{\Delta t} = [\phi-(\phi+\beta)x_t+\beta x_t^2]$$

$$= \beta\left(\frac{\phi}{\beta} - \frac{\phi}{\beta}x_t - x_t + x_t^2\right)$$

$$= \beta[x_\infty-(x_\infty+1)x_t+x_t^2]$$

$$= \beta[x_\infty(1-x_t)-x_t(1-x_t)]$$

therefore:

$$\frac{\Delta x_t}{\Delta t} = \beta[(x_\infty-x_t)(1-x_t)]$$

Appendix 4.4.

The equilibrium distribution is of interest as is the rate at which the equilibrium distribution is approached from any given starting point. We may be interested, for instance, in the rate at which the incidence of tetanus rises after a mass vaccination program in order to determine the optimal intervals between such mass programmes. If we take an initial probability distribution of x_o and y_o ($x_t + y_t = 1$), followed later by two time points close together, we generate the so-called "forward equations" (Cox and Miller [1970]):

$$x_{t+\Delta t} = (1-\beta)x_t + \phi y_t$$

whence $p'_x(t) = -\beta x_t + \phi y_t$

and similarly: $p'_y(t) = -\phi y_t + \beta x_t$

We solve this system of differential equations as follows:

$$x'_t = -x_t(\beta+\phi) + \phi$$

therefore, $\int \frac{dx}{-(\beta+\phi)\,x+\phi} - \int dt = C$

and, $\frac{-1}{(\beta+\phi)} \ln [-(\beta+\phi)x+\phi] - t = C$

Applying the boundary conditions we find

$$C = \frac{-1}{\beta+\phi} \ln [-(\beta+\phi)x_o + \phi]$$

therefore, $\frac{-1}{(\beta+\phi)} \ln \left(\frac{-(\beta+\phi)x_t + \phi}{-(\beta+\phi)x_o + \phi} \right) = t$

i.e. $$x(t) = \frac{\phi}{\beta+\phi} + \left(x(o) - \frac{\phi}{\beta+\phi}\right) e^{-(\beta+\phi)t}$$

and $$y(t) = \frac{\beta}{\beta+\phi} + \left(y(o) - \frac{\beta}{\beta+\phi}\right) e^{-(\beta+\phi)t}$$

The equilibrium condition, $x^*(t) = \frac{\phi}{\phi+\beta}$, is reached as $t \to \infty$. The inverse exponential approach to equilibrium accords with the reuslts illustrated by Cvjetanovic.

Appendix 4.5.

Macdonald argued that, given the proportion recovering from a single infection in element of time is ϕdt, the probability of this recovery is at least partially balanced by the chance of reinfection which for each element of time is βdt. The actual recoveries will, therefore, be represented by (in our symbols): $\phi dt - \beta dt = \tilde{\phi} dt$,

whence $\tilde{\phi} = \phi - \beta$.

If we then apply this effective recovery rate we find that

$$x_t^* = \frac{\tilde{\phi}}{\tilde{\phi}+\beta} = 1 - \frac{\beta}{\phi}$$

i.e., $y_t^* = \frac{\beta}{\phi}$ when superinfection is included, whereas if superinfection is not included we have $y_t^* = \frac{\beta}{(\phi+\beta)}$.

Macdonald's perception of the significance of superinfection is certainly correct. We would like to show that his results are not exact (as he presented them to be) but are an approximation which may or may not be valid (depending on the relative values of ϕ and β).

If we approach the problem from a different perspective and examine the change in the infection load of individuals in the population over time we get a classic immigration - death stochastic process (see, for example, Cox and Miller [1970]). We will derive the relevant properties of the equilibrium distribution

of this process.

In a small element of time we may consider only unit transitions in the infectivity of the host as being possible. If p_i is the probability of carrying i infections simultaneously, the equilibrium distribution must satisfy:

$$o = \frac{\Delta p_i}{\Delta t} = \left(\begin{bmatrix}\text{prob. of one loss}\\ \text{from state i+1}\end{bmatrix} \cdot \begin{bmatrix}\text{prob. of zero gain}\\ \text{in state i+1}\end{bmatrix}\right) p_{i+1}$$

$$+ \left(\begin{bmatrix}\text{prob. of one loss}\\ \text{from state i}\end{bmatrix} \cdot \begin{bmatrix}\text{prob. of one gain}\\ \text{in state i}\end{bmatrix}\right.$$

$$\left. + \begin{bmatrix}\text{prob of zero loss}\\ \text{from state i}\end{bmatrix} \cdot \begin{bmatrix}\text{prob.of zero}\\ \text{gain in state i}\end{bmatrix}\right) p_i$$

$$+ \left(\begin{bmatrix}\text{prob. of zero loss}\\ \text{from state i-1}\end{bmatrix} \cdot \begin{bmatrix}\text{prob. of one gain}\\ \text{in state i-1}\end{bmatrix}\right) p_{i-1}$$

and, since $\phi_i = i\phi$ and $\beta_i = \beta$

$$o = [(i+1)\phi(1-\beta)]p_{i+1} + [i\phi\beta+(1-i\phi)(1-\beta)]p_i + [(1-(i-1)\phi)\beta]p_{i-1}$$

and since ϕ and β are small numbers, we have:

$$o = (i+1)\phi p_{i+1} - (i\phi+\beta)p_i + \beta p_{i-1}$$

Since negative states cannot occur, we have:

$$o = \phi p_1 - \beta p_o$$

whence $p_1 = \frac{\beta}{\phi} p_o$

Similarly, $o = 2\phi p_2 - (\phi+\beta)p_1 + \beta p_o$

therefore, $p_2 = \dfrac{(\phi+\beta)\frac{\beta}{\phi}\, p_o - \beta p_o}{2\phi} = \dfrac{\beta^2}{2\phi^2}\, p_o$

Continuing we find $p_i = \dfrac{1}{i!}\left(\dfrac{\beta}{\phi}\right)^i p_o$

Now we find $\sum_{i=0}^{\infty} p_i = 1$

and, therefore, $\sum_i \dfrac{1}{i!}\left(\dfrac{\beta}{\phi}\right)^i p_o = p_o e^{\beta/\phi} = 1$

whence $p_i = \dfrac{1}{i!}\left(\dfrac{\beta}{\phi}\right)^i e^{-\beta/\phi}$

The proportion of individuals infected is, then,

$$x_t^* = 1-p_o = 1-e^{-\beta/\phi}$$

This is the precise solution. To arrive at Macdonald's solution we expand to get

$$x_t^* = 1 - \frac{\beta}{\phi} + \frac{1}{2!}\left(\frac{\beta}{\phi}\right)^2 - \frac{1}{3!}\left(\frac{\beta}{\phi}\right)^3 + \ldots\ldots$$

which gives $x_t^* \doteqdot 1 - \dfrac{\beta}{\phi}$ for small higher order terms. What this essentially means is that Macdonald's solution is satisfactory where the probability of carrying more than two infections may be neglected. This approximation may well be satisfactory for malaria but to suggest, as Macdonald does, that the published estimate is useful in helminthic infections (in which the number

of simultaneous infestations is likely to be high) is not valid.

[This could also be calculated from the probability density function which was derived earlier:

$$w^* = \sum_{i=0}^{\infty} i\, p_i = \sum_{i=0}^{\infty} \frac{i}{i!} \left(\frac{\beta}{\phi}\right)^i e^{-\frac{\beta}{\phi}}$$

and

$$w^* = e^{-\frac{\beta}{\phi}} \frac{\beta}{\phi} \sum_{i=1}^{\infty} \frac{1}{(i-1)!} \left(\frac{\beta}{\phi}\right)^{i-1} = \frac{\beta}{\phi} \quad .]$$

Appendix 4.6.

In this class of models, too, we may be interested in the approach to equilibrium. In this appendix we will derive the equations describing this approach in both the deterministic (when we have precisely β arrivals and ϕ departures in unit time) and stochastic (when the probability of one arrival is β and the probability of one departure in unit time is ϕ) cases.

i) Deterministic:

The solution of the differential equation $\frac{dw}{dt} = \beta - \phi w$ with initial conditions $w = w_o$ is:

$$\int \frac{dw}{\beta - \phi w} - \int dt = C$$

therefore, $$-\frac{1}{\phi} \ell n \left(\frac{\beta}{\phi} - w\right) - t = -\frac{1}{\phi} \ell n \left(\frac{\beta}{\phi} - w_o\right)$$

whence $$w = \left(w_o - \frac{\beta}{\phi}\right) e^{-\phi t} + \frac{\beta}{\phi}$$

and $w_\infty = \frac{\beta}{\phi}$ (as we had in the text).

ii) Stochastic:

As derived previously the differential equation describing the system is:

$$p_i'(t) = (i+1)\phi p_{i+1} - (i\phi+\beta)p_i + \beta p_{i-1} \qquad (1)$$

The probability generating function of $p_i(t)$ is:

$$G(z,t) = \sum_{i=0}^{\infty} p_i(t) z^i$$

Multiplying (1) by z^i and summing over all i, we get:

$$\sum_{i=0}^{\infty} p_i'(t) z^i = -\beta \Sigma p_i z^i - z\phi \Sigma \frac{i}{z} p_i z^i + \phi \Sigma \left(\frac{i+1}{z}\right) p_{i+1} z^{i+1} + \beta z \Sigma p_{i-1} z^{i-1}$$

Given that $\frac{\partial G}{\partial t} = \Sigma p_i'(t) z^i$

and $\frac{\partial G}{\partial z} = \Sigma \frac{i}{z} p_i(t) z^i$,

we have $\frac{\partial G}{\partial t} = \beta(z-1)G - \phi(z-1)\frac{\partial G}{\partial z}$

or $\frac{\partial G}{\partial t} + \phi(z-1)\frac{G}{z} = \beta(z-1)G$.

Lagrange reduced the problem of finding the general solution to the linear partial differential equation of order one,

$$P(G,t,z)\frac{\partial G}{\partial t} + Q(G,t,z)\frac{\partial G}{\partial z} = R(G,t,z) ,$$

to that of solving an auxillary system of ordinary differential equations

$$\frac{dt}{P} = \frac{dz}{Q} = \frac{dG}{R}$$ (see Ayres [1950]).

We have $\int \frac{dt}{1} - \int \frac{dz}{\phi(z-1)} = a'$

whence $a = (z-1)e^{-\phi t}$

and $\int \frac{dz}{\phi} - \int \frac{dG}{G} = b'$

whence $b = Ge^{-\frac{\beta}{\phi}z}$

and the general solution is $\phi\left((z-1)e^{-\phi t}, Ge^{-\frac{\beta}{\phi}z}\right) = 0.$

At the initial point ($t = 0$), we have w_t worms and therefore $G(z,o) = z^{w_o}$.

We now find the relationship which must exist between a and b at the initial point:

$$a = z-1 \quad \text{whence } z = a+1$$

$$b = z^{w_o} e^{-\frac{\beta}{\phi}z}$$

$$b = (a+1)^{w_o} e^{-\frac{\beta}{\phi}(a+1)}$$

Substituting in the general forms of a and b:

$$Ge^{-\frac{\beta}{\phi}z} = \left(1+(z-1)e^{-\phi t}\right)^{w_o} e^{-\frac{\beta}{\phi}[1+(z-1)e^{-\phi t}-z]}$$

whence

$$G(z,t) = \left(1+(z-1)e^{-\phi t}\right)^{w_o} e^{-\frac{\beta}{\phi}[(z-1)(e^{-\phi t}-1)]}$$

We therefore have (by the power series expansion of G) the equilibrium distribution at any time t. [We note that as $t \to \infty$, $G(z,t) \to e^{\frac{\beta}{\phi}(z-1)}$, the probability generating function for a Poisson distribution with mean $\frac{\beta}{\phi}$, which is the mean worm load in the deterministic case.]

Appendix 4.7.

For a given number of penetrated larvae, the egg output per gram of feces is determined by the proportion of worms which have matured, the probability of mating and thus the proportion of ovadepositing worms, the daily output of eggs per parent worm and the daily quantity of feces. In the analysis which follows we will assume that the EPG count is a simple multiple of the number of worms. (This assumption will be discussed in detail later.)

In analysing the data of Chandler, Mhaskar, Maplestone and Kendrick, it will be assumed (as it was by Chandler and Sarles [1929]) that there will be no further maturation after the peak worm load is reached (as discussed above) and that the time datum for worm loss is at this peak.

To transform Kendrick's and Maplestone's egg count data into equivalent worm loads we assume that 26 eggs correspond to one A. duodenale worm and 12 eggs correspond to one N. americanus worm (see discussion later). Note that the values of these constants do not affect the analysis in this section.

If we assume that the proportion of worms lost over a small time interval is constant, we have

$$W_{t+\Delta t} = W_t - \alpha W_t \Delta t$$

Where W_t = number of worms at time t

α = rate of loss, and

Δt = time interval.

Therefore, we have $\frac{dw}{dt} = -\alpha w$

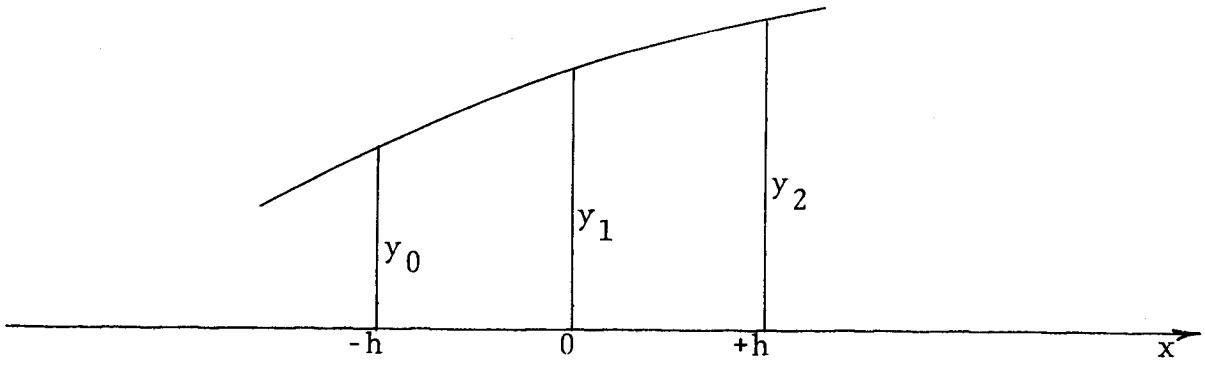
and $\int_{w_0}^{w_t} \frac{1}{w}\, dw = -\alpha \int_{t=0}^{t} dt + C$

whence $$\ell n \frac{w_t}{w_0} = -\alpha t + C$$

and $w_t = w_0 e^{-\alpha t}$ (at $t=0$, $w_t = w_0$ and therefore $C = 0$).

If we set $w_0 = 100\%$ to force the fitted curve through the initial point, and if we then regress $\ell n(w_t)$ on t (in months) we get the values of α as listed on Table 4.4 in the body of the text.

Appendix 4.8.



Simpson's Rule: $A = \frac{h}{3}[y_0 + 4y_1 + y_2]$

We have:

W	$\frac{1}{.000449e^{.0186W}}$	Δt days	$\Sigma(\Delta t)$ years
200	.2699	0	0
195	.3038		
190	.3421	3.04	.0083
180	.4350		
170	.5547	11.83	.0324
160	.7099		
150	.9120	26.19	.0718
140	1.1769		
130	1.5265	50.01	.1370
120	1.9917		
110	2.6169	90.38	.2476
100	3.4671		
90	4.6398	160.79	.4405
80	6.2869		
70	8.6538	288.87	.7914
60	12.1599		
50	17.5748	538.43	1.4752
40	26.4593		
30	42.4909	1091.44	2.9902
20	76.7655		
10	184.9162	2873.00	7.8712

Appendix 4.9.

i) The probability of one egg, which will develop fully, developing to the infective larval stage in time t is, assuming a Poisson process of rate α, equal to $e^{-\alpha t}$. The probability of developing fully in time $z<t$ is $\int_0^z \alpha e^{-\alpha t} dt = 1-e^{-\alpha t}$.

The mean hatching time $= \int_0^\infty t \cdot \alpha e^{-\alpha t} dt = E(t)$

Integrating by parts: $\int u dv = uv - \int v du$

where $u = t$ $\quad dv = \alpha e^{-\alpha t}$

$du = dt$ $\quad v = e^{-\alpha t}$

Therefore $\int_0^\infty t\alpha e^{-\alpha t} dt = -te^{-\alpha t} + \int e^{-\alpha t} dt$

$$= \left(-\frac{t}{e^{\alpha t}} - \frac{1}{\alpha} e^{-\alpha t} \right)_0^\infty$$

Using l'Hopital's Rule:

$$\lim_{t\to\infty} \frac{t}{e^{\alpha t}} = \lim_{t\to\infty} \frac{f(t)}{g(t)} = \lim_{t\to\infty} \frac{f'(t)}{g'(t)} = \frac{1}{\alpha e^{\alpha t}} = 0$$

therefore

$E(t) = \frac{1}{\alpha}$ = inverse of the development rate.

[Note that the probability density function is a skewed distribution and that the probability of full development before time $t = \frac{1}{\alpha}$ (the mean development time) is $1-e^{-1} \doteqdot 63\%$ and the median time $\doteqdot \frac{0.69}{\alpha}$.]

ii)

Let: egg-infective larval development rate = α

egg-infective larval destruction rate = β

infective larval death rate = γ

We have:

Day	% Eggs viable	% Larvae living
0	100	
1	$100(1-\beta)(1-\alpha)$	100
2	$100[(1-\beta)(1-\alpha)]^2$	$100\alpha(1-\gamma) + 100(1-\beta)(1-\alpha)\alpha$
3	$100[(1-\beta)(1-\alpha)]^3$	$100\alpha(1-\gamma)^2 + 100(1-\beta)(1-\alpha)\alpha(1-\gamma) + 100[(1-\beta)(1-\alpha)]^2\alpha$

$$= 100\alpha\left[(1-\gamma)^2[(1-\beta)(1-\alpha)]^0 + (1-\gamma)[(1-\beta)(1-\alpha)] + (1-\gamma)^0[(1-\beta)(1-\alpha)]^2\right]$$

Whence percent of larvae living at day n =

$$100\alpha\left[\sum_{i=0}^{n-1}(1-\gamma)^{(n-1+i)}[(1-\beta)(1-\alpha)]^i\right] \qquad (1)$$

Appendix 4.10.

In this section we wish to investigate the form of the stochastic relationship between the number of mature worms present in a host and the time elapsed since inoculation using functional forms which are a little more interesting that those assumed in the text.

The egg output may be regarded as the product of three factors: i) the number of larvae which have matured into worms,

ii) the proportion of the mature worms which has not yet been expelled from the host, and

iii) the number of eggs per worm.

We assume that the egg output per worm is constant, that the probability density function describing worm maturation is a normal distribution and that worm expulsion is a Poisson process.

$$p_1(\tau) = \frac{1}{\sigma\sqrt{2\pi}} e^{-\frac{1}{2}[(\tau-\mu)/\sigma]^2}$$ = probability of maturation at time τ

and $$p_2(x) = \alpha e^{-\alpha x}$$ = probability of expulsion x units of time after maturation.

If we take inoculation with a single viable larva at $\tau=0$, the probability of the host harbouring a mature worm at time T is

$$p(T) = \int_{\tau=0}^{T} \left\{ \begin{array}{l} \text{prob. of worm maturing} \\ \text{at time } \tau < T \end{array} \times \begin{array}{l} \text{prob. of worm not} \\ \text{being lost in time} \\ (T-\tau) \end{array} \right\} \, d\tau$$

$$= \int_{\tau=0}^{T} \left(\frac{1}{\sigma\sqrt{2\Pi}} e^{-\frac{1}{2}[(\tau-\mu)/\sigma]^2} \times e^{-\alpha(T-\tau)} \right) d\tau$$

$$= \left(\int_{\tau=0}^{T} \frac{1}{\sigma\sqrt{2\Pi}} e^{-\frac{1}{2}[(\tau-\mu)/\sigma]^2} d\tau \right) - \left(\int_{\tau=0}^{T} \frac{1}{\sigma\sqrt{2\Pi}} e^{-\frac{1}{2}[(\tau-\mu)/\sigma]^2-\alpha(T-\tau)} d\tau \right)$$

$$= \quad F\ (T) \qquad\qquad - \qquad\qquad B\ (T)$$

(by definition).

where $B(T) = e^{-\alpha T}\left(\int_{\tau=0}^{T} \frac{1}{\sigma\sqrt{2\Pi}} e^{-\frac{1}{2}\left[\frac{\tau^2-2\tau\mu+\sigma^2\alpha\tau+\mu^2}{\sigma^2}\right]} d\tau \right)$

$$= e^{-\alpha T} \left(\int_{\tau=0}^{T} \frac{1}{\sigma\sqrt{2\Pi}} e^{-\frac{1}{2\sigma^2}\left[\left[\tau-(\mu-\frac{1}{2}\sigma^2\alpha)\right]^2+\frac{1}{2}\sigma^2\alpha\mu-\frac{1}{4}\sigma^4\alpha^2\right]} d\tau \right)$$

$$= e^{-\alpha T-\frac{1}{2\sigma^2}\left[\frac{1}{2}\sigma^2\alpha\mu-\frac{1}{4}\sigma^4\alpha^2\right]} \cdot \int_{\tau=0}^{T} \frac{1}{\sigma\sqrt{2\Pi}} e^{-\frac{1}{2\sigma^2}\left[\tau-(\mu-\frac{1}{2}\sigma^2\alpha)\right]^2} d\tau$$

whence

$$p(T) = \left(\int_{\tau=0}^{T} \frac{1}{\sigma\sqrt{2\Pi}} e^{-\frac{1}{2\sigma^2}(\tau-\mu)^2} d\tau \right)$$

$$- \left(e^{-\alpha T-\frac{1}{4}\alpha\mu+\frac{1}{8}\sigma^2\alpha^2} \int_{\tau=0}^{T} \frac{1}{\sigma\sqrt{2\Pi}} e^{-\frac{1}{2\sigma^2}\left[\tau-(\mu-\frac{1}{2}\sigma^2\alpha)\right]^2} d\tau \right)$$

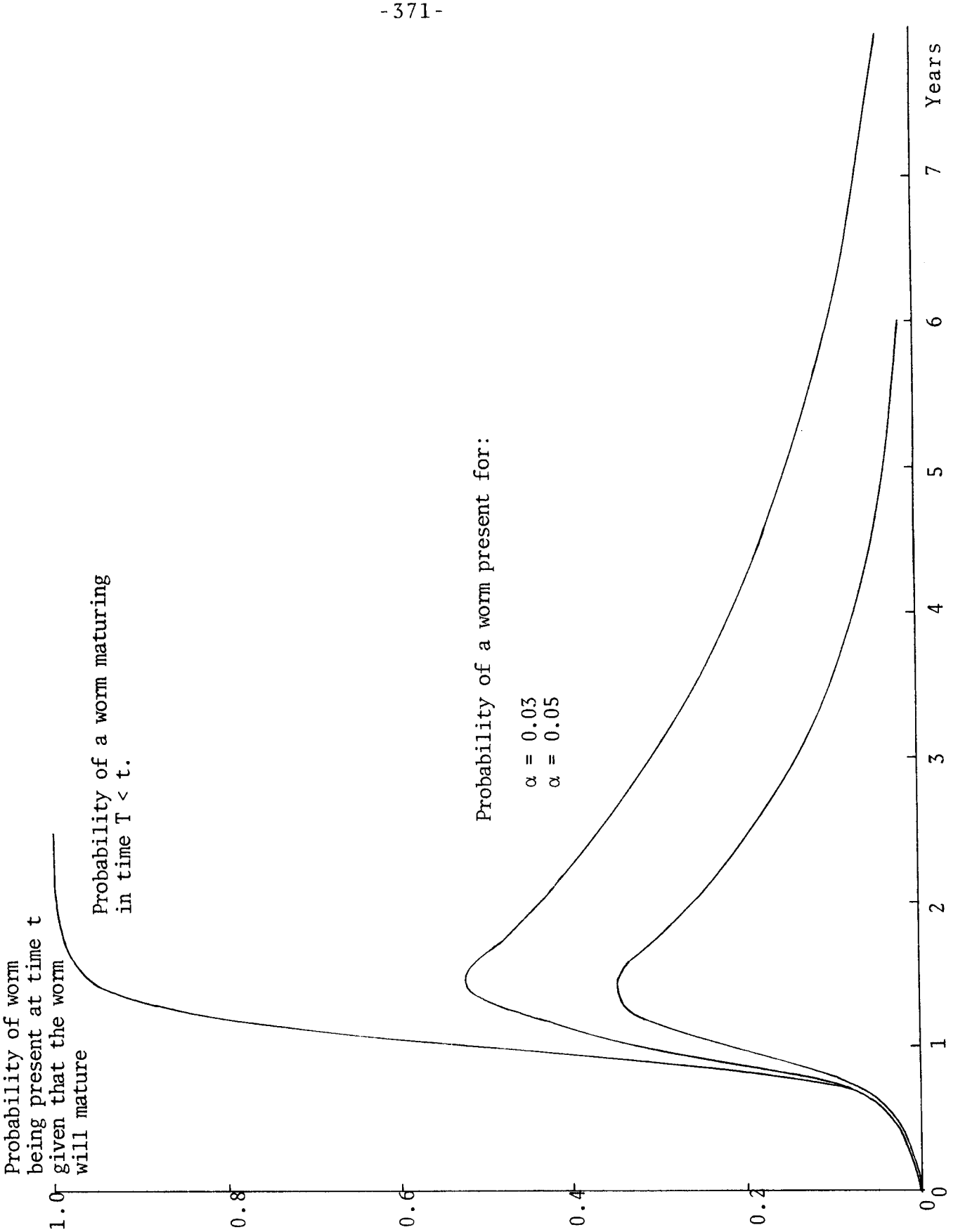
Using standardized cumulative normal tables and letting

$$v = \frac{T-\mu}{\sigma} \quad \text{and} \quad w = \frac{T-\mu+\frac{1}{2}\sigma^2\alpha}{\sigma}$$

we have: $p(T) = F(v) - e^{-\alpha T-\frac{1}{4}\alpha\mu+\frac{1}{8}\sigma^2\alpha^2} \cdot F(w)$.

Taking values of μ = 12 months and σ = 3 months (implying inoculation at about the end of April and approximately the distribution function given in Figure 4.13 - see section 4.4.6.), and values of α of 0.03 and 0.05, which are in the range of values emerging from non-endemic data (the α value is likely to be a lot higher under endemic conditions), the curves displayed on Figure A1 below emerge. It can be seen that the form of these is similar to that which Kendrick's experiments have led us to expect. The functional relationships derived here are interesting but were deemed too complicated to introduce at this stage of the modelling exercise.

Figure A1: Stochastic Worm Maturation Curves



Appendix 4.11.

Relationships Between Different Measures of Community Infestation:

The most extensive survey of hookworm infection in India was done by Chandler in the 1920's. He devised an arbitrary measure to include considerations of both degree of infection and incidence of hookworm in a community . Chandler (1928) defined the following groups:

Group 1, uninfected; group 2, those with less than 100 eggs per gram; group 3, those with 101 to 500 EPG; group 4, those with 501 to 2000 EPG; group 5, those with 2001 to 5000 EPG; group 6, those with 5001 to 10,000 EPG; and group 7, those with more than 10,000 EPG.

If we let y_i = the number of individuals in group i and $\overline{EPG}_i$ = mean egg count for individuals in the i^{th} group, then

$$\text{Infection index} = \sum_{i=1}^{7} \left(\left(\frac{\overline{EPG}_i}{100} \right)^{\frac{1}{2}} (100 y_i / \Sigma y_i) \right)$$

Chandler devised this index to compare the amount of hookworm in different areas and it has proved, and continues to prove, extremely useful for the purpose. Very lightly infected areas have infection indices of less than 100, lightly infected areas between 100 and 200, areas of moderate infection 200 to 400, and areas of heavy infection over 400 (Chandler [1928]).

In the present analysis we wish to have measures both of the mean community worm load (for construction of the epidemiological

model) and the infection index (for translating the results from this model into a meaningful index on the basis of which changes in the hookworm regime can be evaluated). The relationship between these indices is, of course, through the distribution of the worm load in the community. It seemed to be interesting to test whether the distribution in different parts of India varies widely or whether there is some consistent relationship between the mean egg load and infection index.

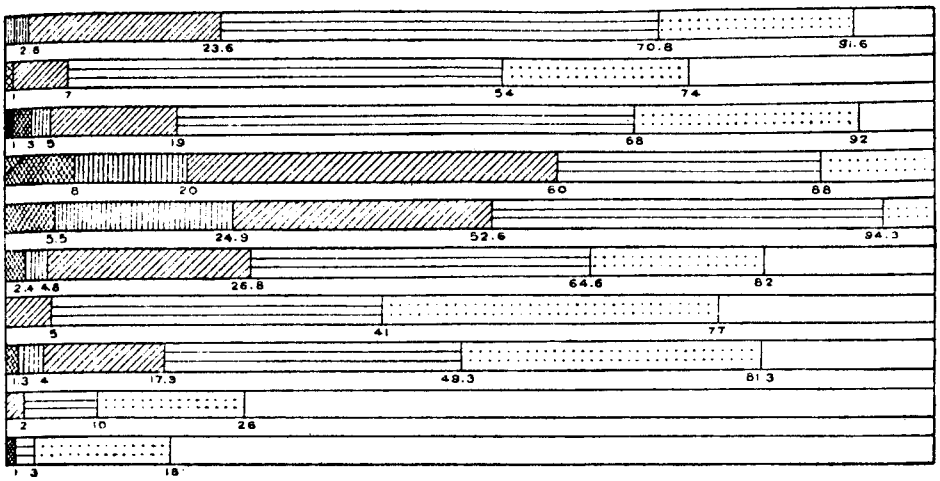
One way of approaching this problem is to: postulate a form of the function measuring the distribution of the worm load in the community; determine the parameters for different surveys; and examine whether there is some consistency in these parameters.

Another approach which is less demanding of disaggregated data, which demands considerably less data analysis and which is less restrictive in formulation , is to determine whether there is a meaningful relationship between the mean egg count and infection index in a community.

In a superb series of papers, Chandler (1928) has presented data on the amount and distribution of hookworm infestation in localities in Burma, Assam, Bengal, Bihar, Orissa, U.P., North-west India, Central India and Bombay (State), the former Madras Presidency and the other southern states. A diagrammatic presentation of Chandler's results is presented along with the infection index and mean egg count on Figures A2 overleaf. A plot of the infection index versus the mean egg count is presented in Figure A3.

Figure A2: Amount and Distribution of Hookworm in India

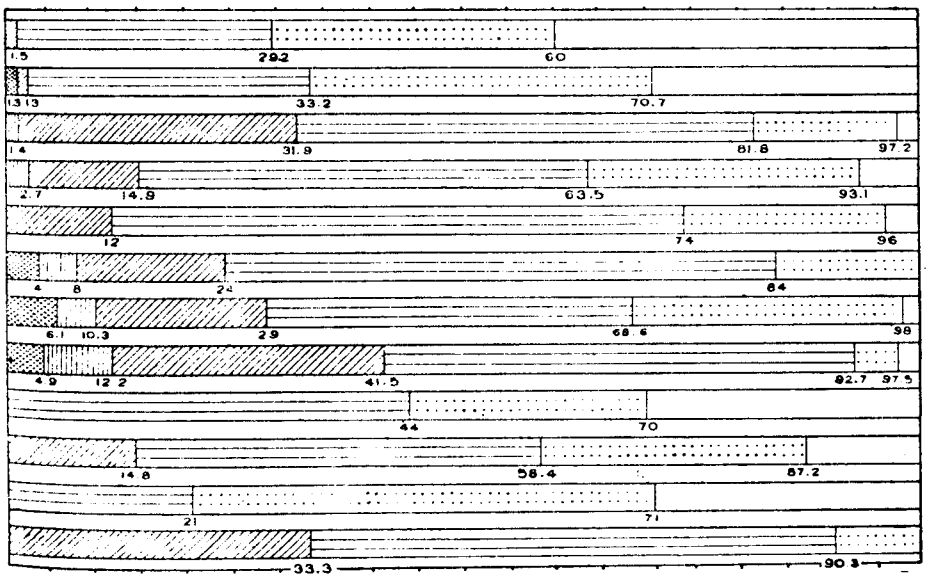
1: Burma:



	Inf. Index	Mean EPG.
Dabein	175	458
Maubin	108	219
Kamawet	176	494
Kamayut	322	1377
Prome	318	1380
Myitkyina	172	555
Hsipaw	89	137
Kalaw	131	348
Thazi	28	50
Aungbinle	16	21

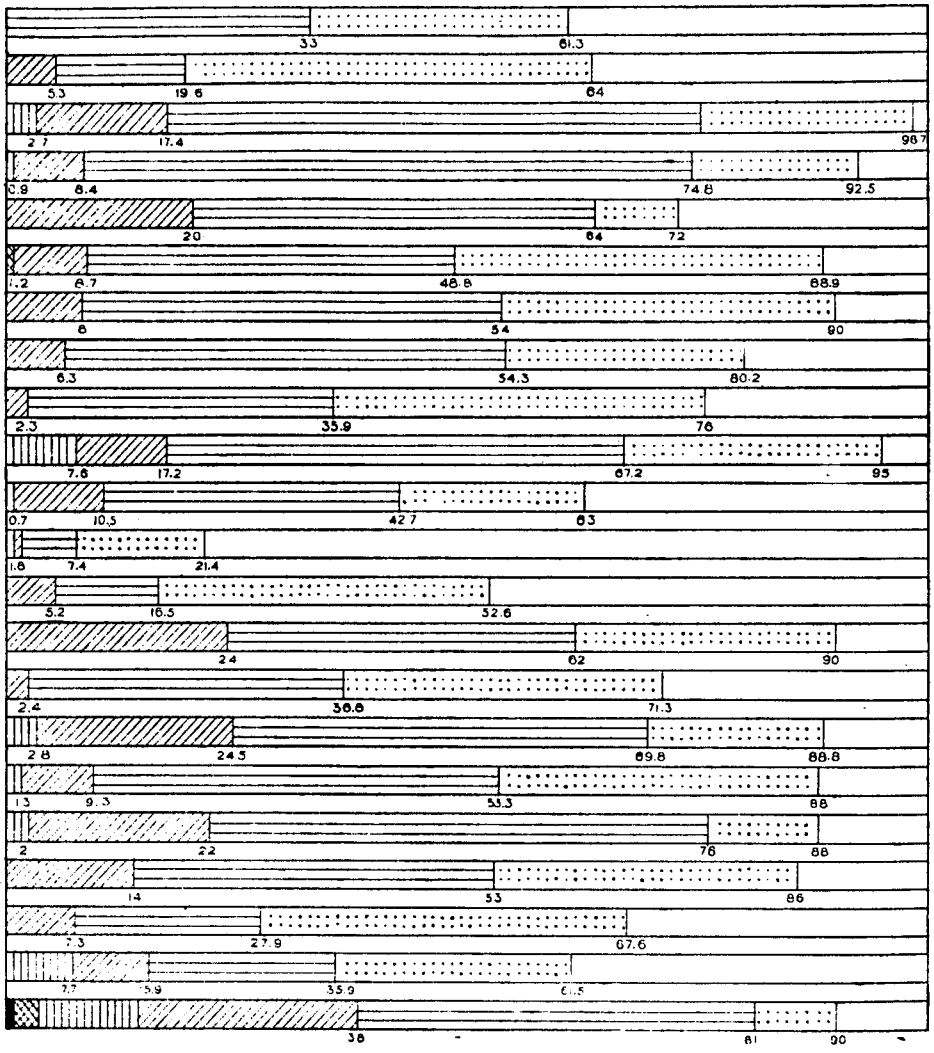
Solid black	Over 10,000 eggs per gram.
Crossed lines	5,100 to 10,000 eggs per gram.
Vertical lines	2,100 to 5,000 eggs per gram.
Diagonal lines	600 to 2,000 eggs per gram.
Horizontal lines	100 to 500 eggs per gram.
Dots	Under 100 eggs per gram.
Clear	Negative.

2: Assam and Chittagong Hills:



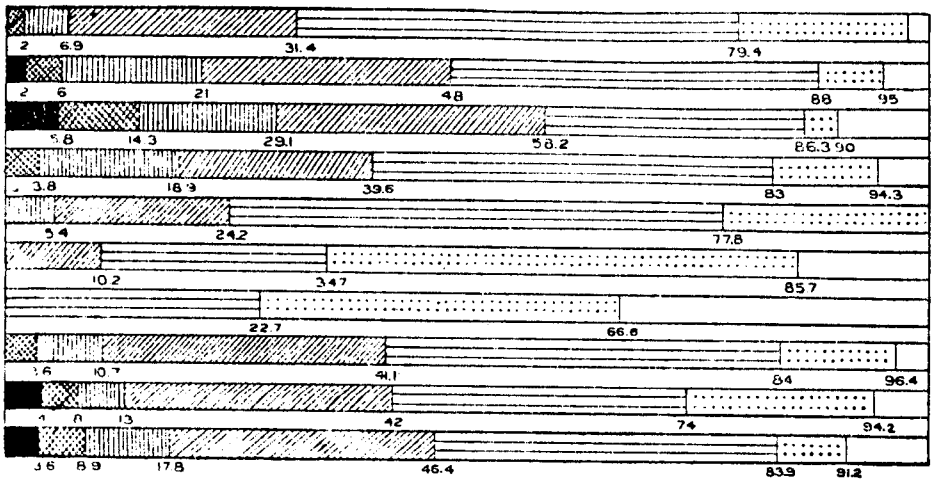
	Inf. Index	Mean EPG.
Dhubri	60	68
Gauhati	90	193
Lumding	192	485
Tinsukia	140	306
Cachar	154	305
Cachar	228	850
Sylhet	218	855
Sylhet	242	880
Shillong	71	76
Kohima	138	273
Imphal	59	54
Rangamati	193	445

3: Bengal



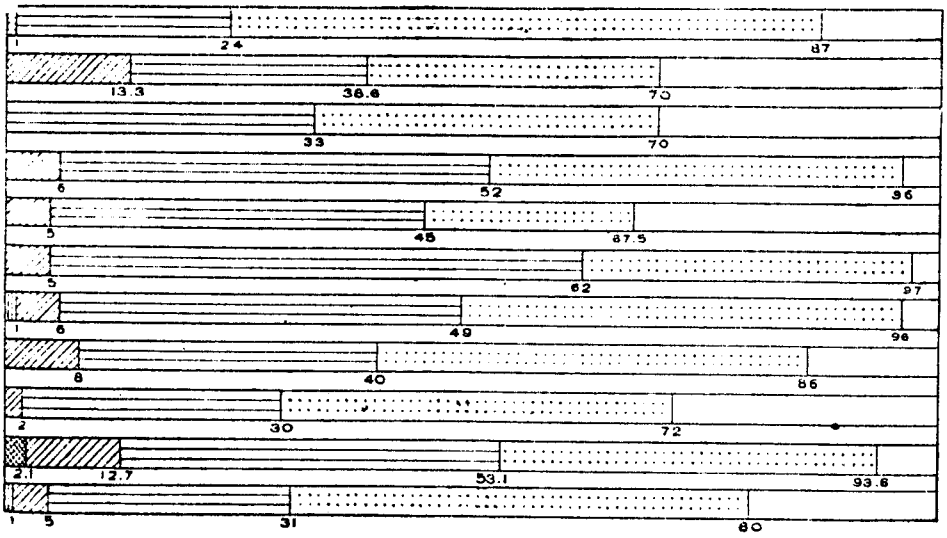
	Inf. Index	Mean EPG
Jobarpur	60	65
Sodepur	64	90
Dhala	156	340
Sitakund	128	221
Azagarah	138	279
Nurnagar	116	244
Jessore	112	173
Krishnagar	101	156
Nawadwip	72	102
Jadabpur	149	409
Kaurapukur	91	188
Gosaba	23	51
Midnapur	55	87
Bolpur	142	284
Rampur Hat	77	97
Ratibati	164	422
Ghussick	128	233
Sirajgunge	158	360
Malda	118	218
Dinajpur	71	104
Madarihat	113	354
Kalimpong	188	896

4: Tea Estates in North and South India



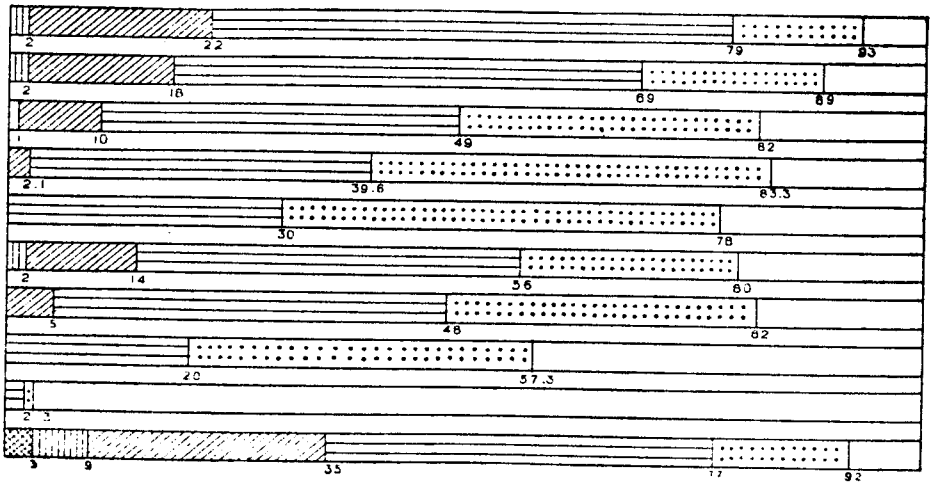
Birpara	212	665
Dumchipara	294	1153
Tukvar	312	2418
Mohima	272	1142
Labac	186	475
Langla	98	171
Terapassa	59	62
Jessie	251	890
Chundale	264	1371
Arapattu	284	1478

5: Bihar and Orissa



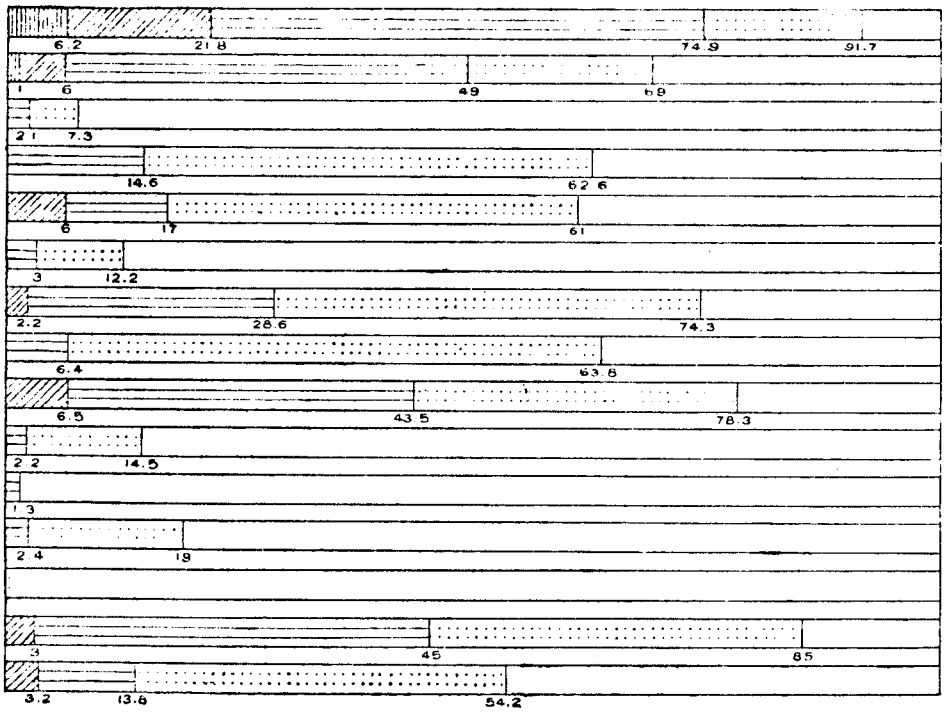
	Inf. Index	Mean EPG
Puri	72	70
Bhadrak	97	197
Chakradharpur	74	91
Camp MacClellan	114	171
Manoharpur	98	144
Ranchi	118	173
Mohulpahari	103	145
Sahebgunge	96	151
Forbesgunge	72	96
Gaya	131	363
Muzzaffurpur	86	136

6: United Provinces



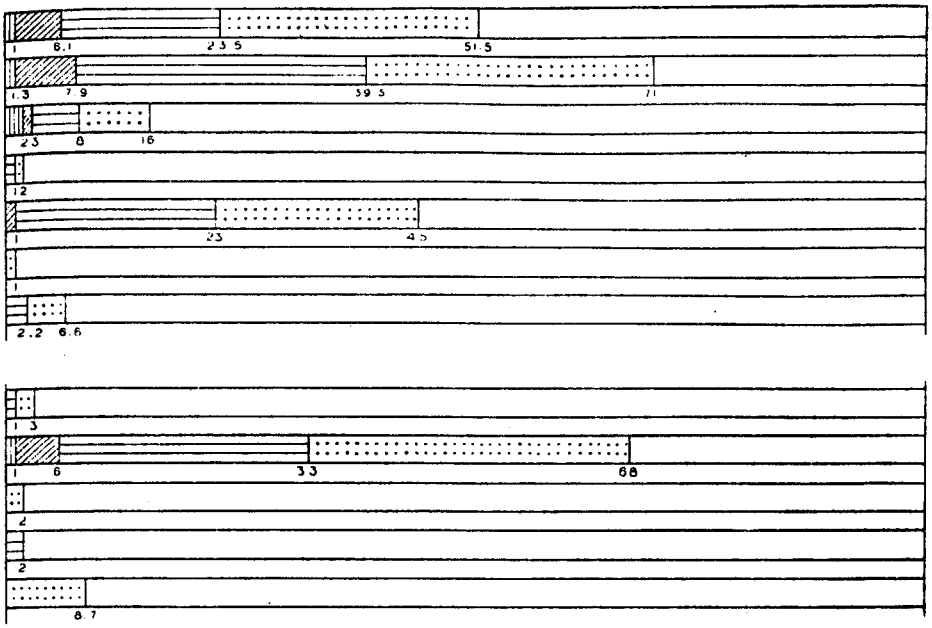
Gorakhpur	177	416
Lucknow	143	312
Kichcha	109	199
Bhimtal	80	90
Ghaziabad	65	61
Meerut	111	280
Tundla	95	138
Banda	48	45
Jhansi	3	2.5
Allahabad	219	788

7: Central and Western India



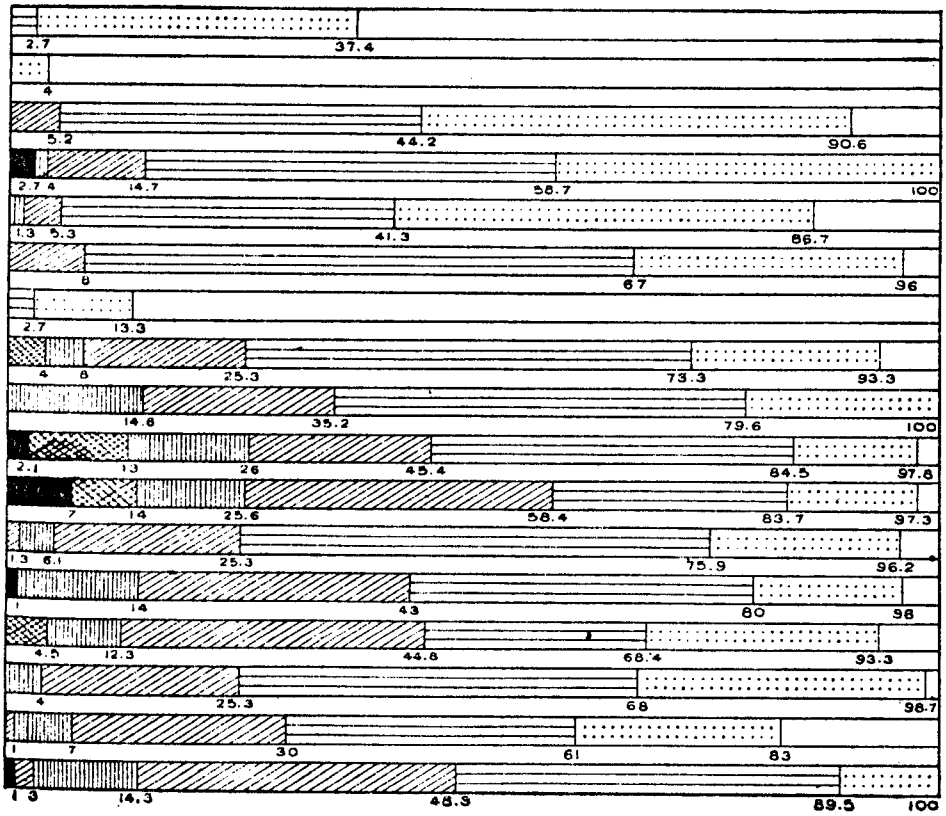
Bilaspur	177	480
Jubbulpur	102	189
Nagpur	5	5
Bina	47	40
Bhopal	46	60
Ujjain	6	7
Hyderabad	69	96
Nander	42	29
Baroda	96	157
Viramgram	10	6
Rajkot	1	1
Poona	12	7
Bombay	0	0
Dharwar	93	127
Bombay	50	75

8: North West India



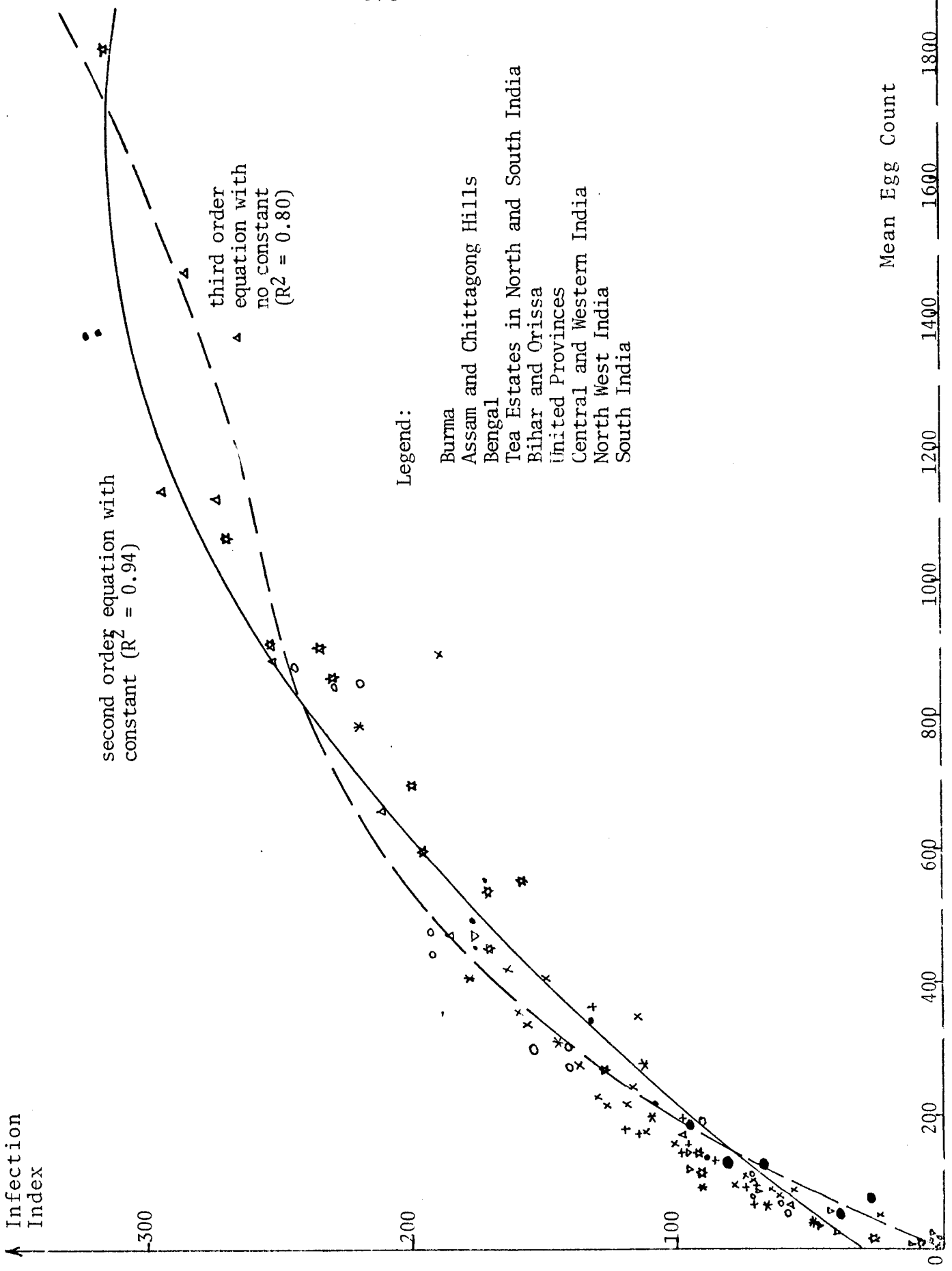
	Inf. Index	Mean EPG
Ambala	67	130
Simla	94	191
Lahore	26	77
Multan	2	2
Peshawar	38	58
Jacobabad	0	0.4
Karachi	5	1.4
Quetta	2	1.4
Jaipur	80	137
Jodhpur	1	0.8
Kotah	2	2
Udaipur	5	4

9: South India



	Inf. Index	Mean EPG
Chitaldroog	25	18
Anantapur	2	1.6
Bangalore	90	119
Hassan	157	555
Coimbatore	97	159
Salem	126	270
Ootacamund	11	10
Mangalore	200	700
Calicut	239	908
Calicut	314	1810
Ernakulam	348	2084
Trivandrum	198	603
Madras	251	913
Nellore	234	880
Bezwada	171	459
Waltair	171	554
Parlakimedi	274	1074

Figure A3: Chandler's Data for Mean Egg Count and Infection Index

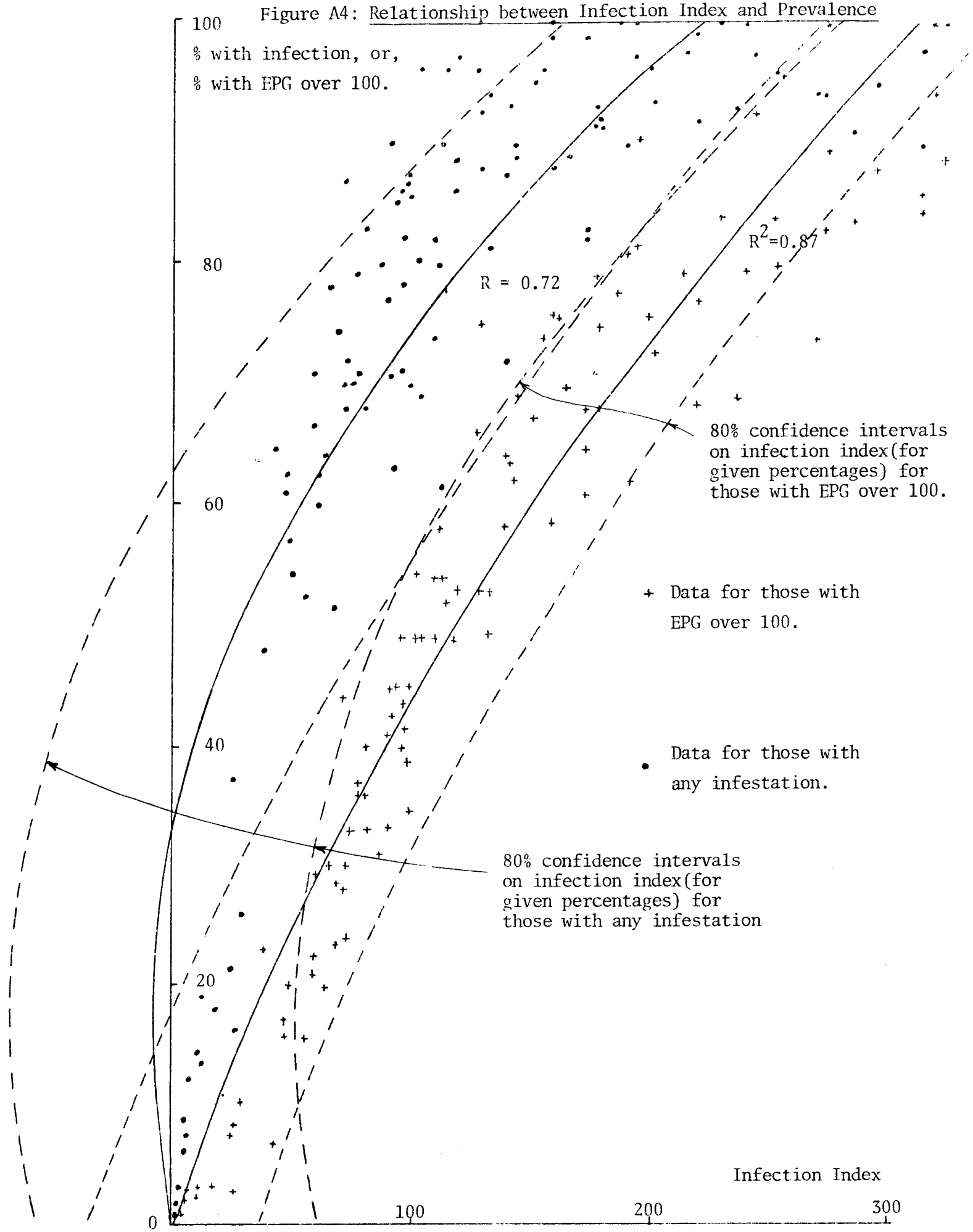


From Figure A3 there emerges a well-defined, non-linear relationship between the two indices which holds for all the regions sampled. To these data second and third order polynomials were fitted. The second order equation, with a constant term included, was chosen as the most suitable. The coefficient of determination (R^2 = 0.94) is surprisingly high. The implication is that the distribution of the hookworm load in a community is not exogenously set, but is related to the level of the infection index in the community.

In view of this result we decided to investigate the relationship between the prevalence of hookworm infestation,and the mean egg load and infection index. The hope was that a clear association would be demonstrated and that this association could be used to infer more meaningful epidemiological statistics from the widely reported prevalence data. The prevalence data given by Chandler (see Figure A2) has been regressed, in a second order polynomial form, against the mean egg count. The association is weak (R^2 = 0.41).

The lack of a clear association between the mean egg count and the prevalence data may be partially explained by a high measurement error associated with low egg counts. For this reason the percentage of the community having counts of more than 100 eggs per gram was regressed against the mean egg load. This relationship is more satisfactory, with 69% of the variance in

Figure A4: Relationship between Infection Index and Prevalence



mean egg load "explained" by the variation in the first and second order prevalence terms.

The above analyses were repeated using the infection index instead of the mean egg load as the measure of the community worm load. The regressions of infection index on prevalence and percentage with more than 100 eggs per gram (the R^2's are 0.72 and 0.87, respectively) are plotted on Figure A4.

The confidence limits for the mean of g observations given a specific set of values of the independent variables (X's) are (Draper and Smith [1966]):

$$Y \pm t(v, 1-\tfrac{1}{2}\alpha)\cdot s\cdot\sqrt{1/g + X_0' C X_0}$$

where Y is the predicted value of the independent variable,

$t(v,1-\frac{1}{2}\alpha)$ is the t statistic with v degrees of freedom and a probability level of α, s is the standard error of estimate (an estimate of the variance about the regression),

$X_0' = [\ 1\ X_1 \ldots\ldots\ldots X_p]$ where $X_1 \ldots\ldots\ldots X_p$ are the means of the observations on the independent variables;

$C = (X'X)^{-1}$ (or $\sigma^2 C$ is the variance covariance matrix of b, the vector of regression coefficients).

Using Schlaifer's (1973) AQD statistical package, the confidence intervals at various levels were computed for the regression of i) infection index on prevalence, and ii) infection index on the percent with EPG greater than 100. The 80% con-

fidence intervals are plotted on Figure A 4.

This analysis is now complete. Given the prevalence of hookworm in a community, we are able to specify the $\alpha\%$ confidence limits on the level of, say, the infection index in that community.

Appendix 4.12.

The following equivalent year-round parameter values are chosen after consideration of the seasonal values given earlier:

Egg and larvae death rate (α_1):

We assume that about 0.125 per day die as an overyear average. This implies a mean residence time in the soil of 8 days or about 0.02 years. α_1 is taken to be 50 per year.

Worm expulsion (α_2):

In the simulation model this value (of 0.24 per day) does not vary seasonally. Ths value used in 0.24 x 365 per annum.

Egg production (γ):

Assuming 300 grams per capita per day and 26 EPG per worm, we have $\gamma = 2.847 \times 10^6$ eggs/cap/year.

Kochar (1975) found the stool density in a traditional defecation area in West Bengal to be about 12.5 per 100 ft^2. Assuming these to be the accumulated stools over 60 hours, the areal "allotment" per person is 20 ft^2. In this model we have 500 people defecating in the moist area, which is taken to be 10,000 ft^2 (Am) in area. At w = 100 worms per capita, $1-d_3(w) = 0.40$ and we find that the required value of $\frac{\zeta}{A_m}$ is 15.28×10^{-6}, and therefore $\zeta = 15.38 \times 10^{-2}$.

Setting these values in equations (1) and (2), we find:

$$d_3(w) = \frac{400}{P}$$

Appendix 4.13.

$$\beta_2^{w_o=100} = 2\beta_2^{w_o=200} = 2 \times 0.0186$$

We wish to have the same calibration point, viz. $\bar{w} = 100$ for $P = 1000$, and we choose $\frac{\zeta^{100}}{A}$ (and $\frac{\zeta^{200}}{A}$) accordingly.

$$\frac{\zeta^{200}}{A} = e^{\beta_2 \bar{w}} \frac{\alpha_2 \alpha_1}{\lambda^2 P \gamma} = e^{.0186 \times 100} \times \frac{50 \times .1638}{0.5^2 \times 1000 \times 2.487 \times 10^6}$$

$$= 0.0739 \times 10^{-6}$$

and $$\frac{\zeta^{100}}{A} = e^{2 \times 0.0186 \times 100} \times \frac{50 \times .1638}{0.5^2 \times 1000 \times 2.847 \times 10^6}$$

$$= .4748 \times 10^{-6}$$

And since $\bar{w} = \frac{1}{\beta_2} \ell n \left(\frac{\zeta}{A} \times \frac{\lambda^2 \gamma P}{\alpha_2 \alpha_1} \right)$

we have $\bar{w}_{w_0=100} = -85.694 + 26.882 \ell n P$

and $\bar{w}_{w_0=200} = -271.397 + 53.763 \ell n P$

whence

P	24.23	40.00	100.00	155.50	200.00	400.00	800.00	1200.00
$\bar{w}_{w_0=100}$	0	13.47	38.10	-	56.73	75.37	94.00	104
$\bar{w}_{w_0=200}$	0	0	0	0	13.46	50.72	87.99	109

Appendix 4.14.

The purpose of constructing the present model is to use it in conjunction with similar models of other diseases, to try to simulate the health effects of a change in the environmental sanitation regime. The probability of death from, say, cholera is contingent on the health status of the community with respect to other diseases. What we wish to investigate somewhat cursorily here is the effect of excluding these interactions when combining epidemiological models for a number of different diseases.

Let us assume that we have three diseases in the community and that the disease pattern is as given below:

	Prevalence (per 100,000 population)			
	Typhoid	Tetanus	Clinical Hookworm	Total
Typhoid	144	0	6	150
Tetanus	0	360	40	400

If we assume that for:

Typhoid : Deathrate (uncomplicated) = a

Deathrate (with hookworm) = 1.30a

and that the Total Death Rate = 10%, we have

$$144a + 6(1.3a) = 0.10 \times 150$$

whence a = 9.90%

Tetanus : Deathrate (uncomplicated) = b

Deathrate (with hookworm) = 1.5b

and the Total Death Rate = 40%, we have

$$360b + 40(1.5b) = 0.40 \times 400$$

whence $b = 38.10\%$

The synergistic effect of hookworm is summarized below:

	Death Rate (%)			
	Typhoid	Tetanus	Hookworm	Total
Typhoid	9.90	-	12.87	10
Tetanus	-	38.10	57.15	40

	Total Deaths (per 100,000)			
	Typhoid	Tetanus	Hookworm	Total
Typhoid	14.23	0	0.77	15.0
Tetanus	0	137.16	22.84	160.0

If we now consider that there are reductions of 30%, 40% and 50% in the prevalences of typhoid, tetanus and hookworm, we have:

	Prevalence (per 100,000)			
	Typhoid	Tetanus	Hookworm	Total
Typhoid	102.9	0	2.1	105
Tetanus	0	228	12	240

Taking the above distribution with the death rates given earlier we get:

	Total Number of Deaths				
	Typhoid	Tetanus	Hookworm	Total	%
Typhoid	10.19	0	0.27	10.46	9.96
Tetanus	0	86.90	6.86	93.76	39.10

We see that, even though the interactions assumed here are strong and the changes in disease prevalences marked there would

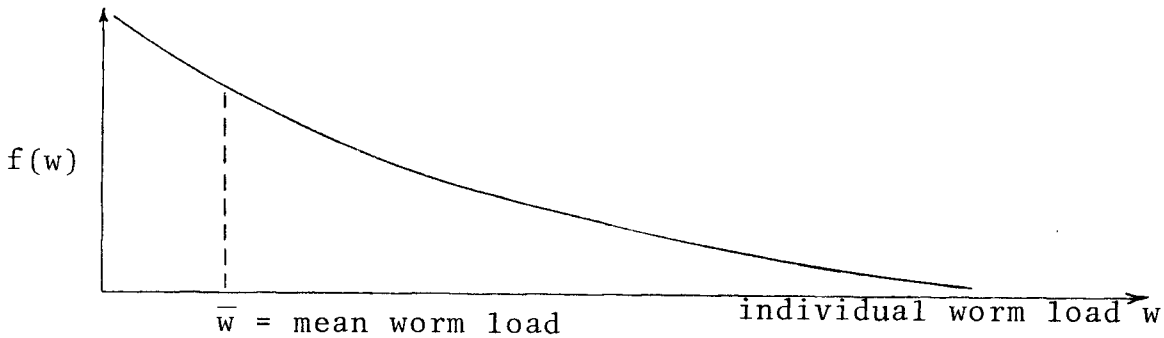
be little distortion (10% instead of 9.96%, and 40% instead of 39.10%) by excluding the complicating effects of hookworm on mortality from typhoid and tetanus.

Appendix 4.15.

An unsatisfactory aspect of the present model is the lack of consideration of distributional factors within the population. This would certainly be an area to which attention should be given in any more sophisticated model. It would be desirable to have an age structure (which may vary) built into the population and to relate the defecation practices and susceptibilities to infestation to the age groups.

By not explicitly considering the distribution of factors, such as the worm load, in the community,we may be making serious aggregation errors. This will be illustrated by investigating the relationship between individual and community immune responses given different worm load distributions.

Assume that the relationship between the proportion of "penetrating larvae" which remain viable and the number of worms harboured by the human host is known from experiments on individuals. Let the immunity function be I(w) where w represents the number of hookworms harboured by the host. Assume, further, that the probability distribution function [F(w)] of worms is known in the community under consideration. The probability density function, f(w), is the derivative of this distribution function and will look something like:



The mean worm load, $\bar{w}$, is defined as:

$$\bar{w} = \int_{\text{all } w} w\, f(w)\, dw$$

The proportion of penetrating larvae which do not develop to maturity (P) in the community as a whole is:

$$P = \int_{W} f(w)[1-I(w)]dw$$

$$= 1 - \int_{W} f(w)I(w)dw$$

If we define Ic(w) to be the immune response function which has to be applied to the whole population of mean load $\bar{w}$ such that the proportion of penetrating larvae which mature will be P, we have:

$$1 - Ic(\bar{w}) = 1 - \int_{W} f(w)I(w)dw$$

and thus $Ic\left(\int_{W} w\, f(w)dw\right) = \int_{W} f(w)I(w)dw$. (1)

This means that when considering the epidemiology of hookworm in a community we cannot simply evaluate the individual immunity response function at the mean load.

We will now outline a means by which the necessary function could be derived and then indicate the way in which the function would be used operationally.

From the analysis of data (such as Chandler's data presented earlier) we derive the distribution function F(w) for each of the sites for which data are presented. Let us assume that the

distribution function is $1-e^{-\rho w}$. From this distribution function we may derive the probability density function f(w) where

$f(w) = \frac{dF(w)}{dw}$ ($= \rho e^{-\rho w}$, the exponential distribution with parameter ρ ,in our example). Each site, then, has a mean worm load $\bar{w}$ and a parameter, ρ , describing the distribution of the worm load. Through statistical analysis we determine the relationship $\rho = \rho(\bar{w})$. Other exogenous variables which do not enter the model analysis (such as climatic variables) may be included in the specification of this relationship. (In the example the mean load $\bar{w} = \frac{1}{\rho}$, or $\rho = \rho(\bar{w}) = \frac{1}{\bar{w}}$).

By evaluating the right hand side of equation (1) above for various values of $\bar{w}$ the required function $Ic(\bar{w})$ is found.

Using the individual immunity function derived earlier with the "example" distribution function:

$$Ic(\bar{w}) = \int_{\text{all } w} \rho(\bar{w}) e^{-\rho(\bar{w})w} \times \left(\frac{0.3e^{.01253w}}{0.7+0.3e^{.01253w}}\right) dw$$

$$Ic(\bar{w}) = \int_{\text{all } w} \frac{1}{\bar{w}} \left(e^{-\bar{w}}\right)^{w} \times \left(\frac{0.3e^{.01253w}}{0.7+0.3e^{.01253w}}\right) dw \quad .$$

The determination of $Ic(\bar{w})$ in the case of a simple distribution function is clearly non-trivial. Given that the "individual immunity function" is no more than an educated guess, it was not deemed worthwhile to pursue this matter further. Since the form $Ic(\bar{w})$ is likely to be similar to that of I(w) (i.e., logistic) we will make the simplest possible assumption and assume that

$Ic(\bar{w}) = I(\bar{w})$. (Note that this equality pertains only in the case of a Dirac delta probability density function.)

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