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SOCIO-ECONOMIC ANALYSIS OF IMPACT OF WATER PROJECTS ON SCHISTOSOMIASIS

Final Report

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by

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Responsibility for any errors lies, of course, with the authors.

Abstract

A methodology for integrating a schistosomiasis transmission model and associated economic analyses into water resources project planning is described and applied to an area of small-scale water activities (furrow irrigation, cattle watering in ponds) in Misungwi, Tanzania where schistosomiasis control efforts were underway from 1967 to 1973. The methodology builds upon previous work in developing and testing the basic transmission model in an area of large-scale water activities (extensive irrigation) in Iran and in an area of rain-fed agriculture and protected domestic water supplies in St. Lucia. In the model, incidence is estimated as a non-linear, interaction function of epidemiological (number of infected individuals/age) and environmental variables (feet of snail habitats/age within one-half mile of household) by regression analysis. The incidence estimates combined with a worm loss rate are used to predict the fraction of the population infected (prevalence) with schistosomiasis the following year. Costs and effectiveness of control measures are estimated through changes in the independent variables in the regression model.

Because of data requirements, the transmission model was only able to be applied to the 2-to-9-year old population in the one sector of the control project where habitats could be linked to individual households. The use of mollusciciding, the only control measure used in this sector, was incorporated through changes in the habitat term. The sample size (32 or fewer individuals

per age group) along with only two years of available data for comparison led to difficulties in validating model predictions by comparing them with observed data. An interesting result, however, was that the regression parameters obtained from use of number infected/age and feet of snail habitat/age were almost identical to those obtained in Iran with similar independent variables [incidence = 1.6×10^{-6} ($V^{0.91} \times P^{0.36}$) for Tanzania compared with incidence = 5.7×10^{-6} ($H^{1.1} \times P^{0.45}$) for Iran].

The methodology was successfully expanded to include other variables: intensity of infection (total eggs passed/age), migration, and seasonal variation. Intensity of infection was substituted for number infected in the incidence equation by 1) estimating the arithmetic mean of eggs passed per age as a function of predicted prevalance and then, 2) multiplying the arithmetic mean egg/age times the total population number/age to obtain total eggs passed, which was then regressed with the habitat term against incidence. Only slight differences in predicted prevalence levels resulted when intensity of infection was used instead of number infected.

Migration was accounted for in the model by correcting the total population term and prevalence predictions for immigrants and emigrants based on project collected data. Migration affected prevalence predictions to the greatest extent when control measures were used over the long term (after five years).

Seasonal variation was incorporated into the model by changes in the habitat term based on data in an unmolluscicided sector in Misungwi. Short-run results indicated only slight differences when this variable was included although it would assume greater importance if more detailed snail population data were used.

Results from the hypothetical cost-effectiveness analysis indicated that combined use of chemotherapy and molluscicide was most effective in preventing case-years of infection. Chemotherapy use, by itself, was the most cost-effective measure. Again, there were only slight differences in case-years of infection prevented between use of number infected or of total eggs in the incidence equation.

It was concluded that priority data items for integrating a schistosomiasis predictive methodology into water resources project planning were (on an age- and, if possible, sex-specific basis): prevalence, incidence, snail species, human contact with snail habitats, migration, total population, and control measure costs. More detailed data collection efforts would be needed in large-scale control programs. It was stressed that data should be collected and reported on an individual basis to facilitate their use in the analyses.

Recommendations for future research stressed the need for detailed studies on migration, human water contact with snail habitats, and estimating control measure cost functions. A more general migration model incorporated as a subroutine transmission model would enable the methodology to be applied in a variety of economic development projects. It was also suggested that the appropriate test for the medietive methodology was to apply it at the planning stages of a water resources project. By collecting the necessary data on a pre-project basis from an adequate sample size, prevalence predictions could be made. The predictions could then be compared over time with actual events. The combination of longitudinal and cross-sectional verification would demonstrate more conclusively the usefulness of the methodology for schistosomiasis preventive planning associated with water resources development projects.

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Chapter 1

SCHISTOSOMIASIS TRANSMISSION: BACKGROUND ON MODEL DEVELOPMENT AND DATA AVAILABILITY

Introduction

Schistosomiasis is a parasitic disease that involves both human and snail hosts. Because the snail host is usually aquatic, water plays a critical role in transmission of schistosomiasis, as may be seen from the life cycle diagrammed in figure 1. Transmission from man to snail and back to man may be influenced by a number of factors. The snail host may live in a variety of habitats ranging from canals and dams to ponds, lakes, and rivers. Seasonal variation, floods, droughts, water quality, and food supply are natural influences on the snail's ability to survive. Human contact with snail habitats may be a function of age, economic and domestic activities, housing conditions, and availability of protected water sources.

Both snails and humans are affected by changes in water availability in an area: large-scale projects that channel water for food production or dam water for power production; small-scale activities that create ponds for water storage, livestock water supply or human water supplies; and, activities of either large or small-scale that bring protected water for domestic purposes to a village. Each of these activities influences schistosomiasis transmission. In the first two cases, snail habitats and human contact with these habitats increase and, in the latter case, human contact decreases. If it were possible to predict the degree to which changes in water availability affect the level of schistosomiasis in the project population, then it

would be possible either to change the design of the project or, at the very least, to plan a control program to minimize the adverse health impacts.

The purpose of this report is to test a previously developed predictive schistosomiasis transmission model with a new set of field data from the second kind of situation described above, that of small-scale water activities. The data are from Misungwi, Tanzania (see figure 2), where a schistosomiasis control project was in operation from 1967 to 1973. The model, developed for use in large-scale irrigated areas, was tested and shown with modifications to be useful in simulating the effects of domestic water supply projects. In using the Tanzania data, the model will then have been successfully applied to all three types of water projects.

The new set of data also permitted us to examine in detail serious concerns in modeling: what is the appropriate measure of infection in the human population; what are the trade-offs between greater detail and cost of data collection in relation to reliability of predictions? In addition, the role of migration in transmission was examined. The predictive model was then used with the same data to examine costs and effectiveness of control measures.

Using the experiences gained in working with various data sets from different projects, we have suggested data collection priorities to assist in future modeling studies. The objectives of a control project often do not include data collection for modeling purposes and, as a result, assumptions must later be made that may limit either the scope of the model or the modeler's ability to test hypotheses.

The remainder of this chapter focuses on background information for the analyses of the Tanzania data followed by a review of that data. The review includes a general discussion of the schistosomiasis situation in Tanzania and specific details of the schistosomiasis control project in Misungwi,

Discussion of Model

The type and degree of sophistication of the methodology used are directly related to specific objectives. In this case, the main objective is to develop a predictive methodology to assist with development project planning and control program management. The objective is not to quantify all of the relationships in the schistosome life cycle but rather to reduce data collection efforts and costs by finding the minimum number of fundamental elements needed to predict changes in schistosomiasis transmission over time. The fundamental elements, infected persons and snail populations,are directly affected by development projects and control measures. By relating the elements in a predictive model, one can estimate changes in incidence rates and prevalence in the project populations which result from the project. The focus of the modeling effort described here is on the human population elements since "evaluation of the efficiency of the control programs must be made in terms of its impact on human schistosome infection" (1). Snails, of course, play a critical role in transmitting infection, but it is the reduction of human suffering which concerns health and development planners. It is recognized, of course, that water projects and control efforts affect snail populations, so it is necessary to include some information on snails or their habitats.

The desire to keep the variables in the model to a minimum may become more understandable when one considers in detail the variety of measures of human infection and snail populations. Human infection encompasses variables such as intensity of infection, prevalence, incidence,

reversion, and processes such as immunity.[1] Snail population information includes numbers of infected snails, population density measures, habitat characteristics, and climatic variation.

A second measure of human infection is prevalence. Prevalence levels may not adequately reflect short-term changes resulting from control programs; however, they do reflect changes over the long-term. Prevalence is, however, the easiest variable to measure. Moreover, the unit of measure is directly related to the population size (or sample of same). Since development project analyses are based on population size, prevalence could be more easily used by planners. Prevalence provides a useful, simple term for demonstrating project-induced changes.

Many medical researchers believe that human infection should be expressed in units of eggs passed. Egg output, however, "should be determined by a constant, reliable, and quantitative technique which must not vary from year to year" (2). This is often not the case and even more importantly, the reasons for variance in eggs passed by a given individual from year to year are not yet understood. The variance leads to complications in the statistical handling of egg counts, requiring transformation of the values to either the arithmetic mean or geometric mean for each age group. Even so, it is possible that this problematic indicator of severity of infection could be a better predictor of transmission intensity than prevalence because egg

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[1] Incidence is the rate at which uninfected persons become infected over a given period of time. Prevalence is the number or fraction of infected persons in a population at any point in time. Intensity of infection is measured by the number of eggs passed per unit volume of urine or feces. Reversion is the rate of infected persons spontaneously (not due to treatment) becoming negative over a given period of time. We use the word "infection" to describe the fact that there are positive cases of schistosomiasis in a population. The word "disease" is reserved for use when clinical symptoms from the infection are observed.

counts are a measure of intensity of infection. For these reasons, egg counts can be considered in the modeling to show if estimates of control measure effectiveness vary when one uses egg counts instead of prevalence levels.

Incidence provides a time dimension to transmission. Although difficult to observe without a carefully controlled study, incidence rates, like egg counts, provide a sensitive measure of changes in transmission. In the model we are interested in predicting changes of infection levels over time and incidence rates reflect changes in transmission over time.

Two aspects of human loss of infection (besides losses exogenously induced by chemotherapy) may also be considered, reversion rate and immunity. Reversion rate, or the natural death rate of the worm has infrequently been measured in the field (3). S. haematobium is thought to have an exceedingly short life span, under three years, and light human infection with no re-exposure could die out without any treatment. S. mansoni and S. japonicum live longer in man; in extreme cases, S. mansoni worms have been found in infected immigrants in New York City and California 20 years after their arrival. Any modeling attempt should account for the worm death rate.

The second loss over time is due to immunity. The process of immunity to new schistosome infections is still not understood. It is thought to occur because observations of the age prevalence curves for schistosomiasis infections, especially S. haematobium, repeatedly show a decline after ages 15 through 25. The reasons for this decline have been the subject of much debate (4). It is generally agreed that natural immunity to schistosomiasis does not exist; however, immunity acquired from continual re-infection or

high levels of infection is thought to be a significant possibility. Acquired immunity has, however, not yet been identified in the field, for it must be separated from changes in water contact patterns, seasonal changes in egg output patterns, and other variables which might account for changes in the shape of the age-prevalence or age-egg output curves.

Snail habitats are used in the model instead of density of infected snails for a variety of reasons. Despite the fact that the snail as the intermediate host in schistosomiasis transmission has often been thought of as the weak link in the life-cycle and thus highly susceptible to control measures (5), it has been difficult to control snail populations in any but the most limited environments. So many factors influence snail populations that a separate model is needed to account for the snail's role in transmission. Yet, collecting information on food, light, density and other requirements is time consuming and may not be feasible as part of the planning of large scale water projects. It may be sufficient to know which bodies of water are likely to harbor snails, which schistosome-bearing species of snails are in the area, and what role seasonal variation may play. In most areas, only a small proportion of snails are ever infected (6). One may be able to assume that any habitat with the appropriate snail species and used by humans is a likely transmission site. The knowledge of habitat dimensions and human water contact patterns may provide the requisite amount of information needed for predictive modeling. This assumption was the basis of earlier work and is examined here in the section on model modifications.

Thus, the following considerations influenced what terms we used in the model:

1. Egg counts would be most desirable to use but, if not available, prevalence levels are sufficiently reliable measures of human infection.
2. Incidence and reversion rates are critical to include since they are assumed to be sensitive measures of transmission changes.
3. Snail habitats that are transmission sites or estimates of human contact with snail habitats are satisfactory surrogates for infected snail population data.

Model Review

Implicit throughout the above discussion is the desire to maintain simplicity in the model and its data requirements. Models have been developed that encompass a range of variables. Some variables may be easily collected in the field while others require detailed data collection.

The expression that we chose to use defines straightforwardly the rate in change in prevalence levels (y) over time (t) as a function of uninfected persons become infected at some rate (A) and infected persons losing the infection at some rate (B) (7.):

$$\frac{dy}{dt} = A\,(1 - y) - B(y) \qquad [1]$$

This equation may be solved to give the following difference equation (12):

$$y_{t + \Delta t} = \left(y_t - \frac{A}{A + B}\right) e^{-(A + B)\Delta t} + \frac{A}{A + B} \qquad [2]$$

This equation is used to predict changes in levels of infection from one time period (t) to the next (t + Δt).

As mentioned above, it is assumed that the incidence rate (A) is a function of infected persons or egg output, and snail habitats or human contact with the habitats. Incidence rates may be estimated by use of regression analyses if field observations are available.[2] Regression analysis results indicate the relative importance of the independent variables in estimating the variance in incidence. Although the relative importance of the independent variables in predicting incidence rates may change when applied to a new site, the same independent variables may still be appropriate. In addition, the form of the equation relating them may remain constant. To test the hypothesis of generality of the variables and equation form, the results from this study will be compared with two previous analyses. These earlier studies indicated that the most significant form of the equation was the non-linear product of an environmental/behavioral term (H) times an epidemiological term (P) (8):

$$A = \beta_0 \left(H_t^{\beta_2} \times P_t^{\beta 1} \right) \qquad [3]$$

where A was the incidence rate, t the time unit, and β_0, β_1, β_2 estimated regression parameters.

In one study in Iran, an area of large-scale irrigation, H was measured as meters of accessible snail habitat per village per year and P was the number of infected persons per village per year. In the second study in St. Lucia—an area of domestic water supply provision, H was a human water

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[2]Incidence rate studies are carried out as follows: A group of a particular age or range of ages is determined to be definitely negative by repeated urine or feces examination. This group, with no replacement for dropouts, is followed over time to determine who becomes positive after, for example, one year. The same group minus the positive is followed and examined the next year. The study may continue as long as the sample size is sufficiently large. The incidence rate is measured by dividing the new positives in the next year by the total number of negatives the year before.

contact parameter per age group (a measure of frequency and duration of human water contact with snail habitat) called WC in that study and P was the number of infected persons per age group.

The expression for the loss rate B has been assumed to be related to the natural death rate of the worm, physiological characteristics of the human host, and immunity. Two studies have attempted to observe loss rates in the field (9).[3] These results compare favorably with mathematical estimates (obtained by solving equation [2] for b): In Iran, B = 0.2 and in St. Lucia, age-specific estimates of B = 0.3 for 0-to-9-year-olds and B = 0.2 for older ages (10). Sensitivity analyses showed this term to be highly important in prevalence predictions. Site-specific field studies, however, are needed to define more reliably the values of B for use in modeling.

Data Requirements. Epidemiological and environmental data for model testing are listed in table 1 along with control cost data required for an analysis of cost and effectiveness of control measures. We believe these data are sufficient for using a predictive model although we recognize certain items will vary with the project under consideration. For example, it is time consuming to quantify the extent of human contact with transmission sites. In either a large- or small-scale water resources project, one may assume that the canals and ponds are used for domestic purposes if no protected domestic water supplies exist. Casual observation or discussion with villagers may provide a firmer basis for the assumptions. For these

[3] The loss rate or reversion rate is measured in the field in a manner similar to incidence studies. A group of positives is followed over time, usually one year, to determine the number who lose the infection. The positive group must not be treated so that the loss may be attributed to natural conditions.

projects, however, one would want information on the size of transmission sites and on whether or not snails thrive in them. In contrast, when examining the effects of protected domestic water supplies, more detailed water contact studies would be needed but the size of the habitats would be less relevant. In all situations, the location of habitats in relationship to households must be mapped. One other important consideration in reporting data is the degree of data aggregation. So that the user of data is not limited in his analyses, information should be given on an individual person or habitat basis. For example, it is preferable to report egg counts for each individual (as was done in Tanzania) rather than aggregating egg count data by age group.

Schistosomiasis in Tanzania

Background Information. Schistosomiasis has been studied in Tanzania since the 1950's with the East African Medical Research Institute, Mwanza acting as the focal point for the research (see figure 2). Researchers at the Institute have measured prevalence levels of the two species in Tanzania, S. haematobium and S. mansoni. They have also instituted control programs, the most extensive being the project under discussion (11). In addition, there have been a number of transmission studies specifically in large-scale irrigated areas with the Arusha Chini scheme in the North as the focus for control, economic and epidemiological studies (12).

S. haematobium is found throughout Tanzania, with prevalence levels as high as 80 percent common to the north and northeast of Lake Victoria (13). Although S. mansoni is less generally widespread in Tanzania, prevalence levels may be as high as 50 percent around Lake Victoria (14).

A variety of snail hosts are found in the country. Ideal habitats occur in Lake Victoria, irrigation systems, small ponds, dams, and permanent and seasonal water courses (15).

The public health importance of schistosomiasis infections in Tanzania has been summarized by Jordan in 1966 (16). He described several studies which showed a variety of severe clinical effects from the infection, especially in children. Widespread urological changes from S. haematobium were identified, but S. mansoni infection at that time appeared to be less severe in Tanzania than in other parts of the world.

In the 1970's, Tanzanian development has been influenced by President Julius Nyerere's "villagization" program, especially by the establishment of Ujamaa villages, which stresses cooperative activity (17). The movement and settlement of large numbers of persons is bound to have a dramatic impact on schistosomiasis transmission, especially since the expected improvements in sanitary conditions (by the provision of water supplies, latrines, and health education) have not yet taken place (18). Moreover, small-scale irrigation projects continue to play a role in providing water for crop production. Schistosomiasis prevalence may be spreading as a result of too limited control activities and environmental changes brought about by new living patterns.

Schistosomiasis in Misungwi, Tanzania. The data for this study comes from the World Health Organization/Tanzania Schistosomiasis Pilot Control and Training Project, Mwanza District, Tanzania, run by the East African Medical Research Institute under the direction of D. V. M. Eyakuze, National Project Director, and Dr. Fergus McCullough, WHO Project Leader. The project began in April 1967 and ended in December 1973. Two contrasting areas were

chosen for the project site, an urban situation at Mwanza and a rural area around Misungwi (figure 3). The aim of the project was "to determine the feasibility of schistosomiasis control within present resources, and to evolve methods which may be applicable, with suitable modifications, to other parts of East Africa" (19). In Misungwi, the rural area, S. haematobium infections predominated, "with an average prevalence of over 60 percent" (20). The major snail host was B. (P) nasutus which was found in "small man-made water bodies" (21). In Mwanza, the urban area, S. mansoni was the main species of the parasite, with prevalence levels reaching 30 percent (22). The intermediate hosts, various Biomphalaria snail species, were found "in streams, lakeshore waterbodies and Lake Victoria itself" (23).

The Misungwi situation, like much of this part of Tanzania, represents a "complex transmission pattern" (24) where in a relatively uniform environment, "the infection, in both the definitive and intermediate hosts, is characteristically widespread and relatively non-focal" (25). The pilot area was to the northwest of Misungwi Town covering an area of 76 km^2 and a population of 4000 (see figure 4). Although S. haematobium was the major species present, pockets of S. mansoni infections did occur with prevalence levels of 10 percent.

The Misungwi area was chosen as the focus for this study because the data were extensive and recorded on punch cards so that a computer tape could be easily made. The area had been divided into five sectors so that different control measures could be tested in different sectors, thus providing a basis for comparison of control measure effectiveness. The work plan and results of the Project in Misungwi were stated as follows in the Tanzania 001 Final Report:

> At Misungwi the work was planned in three interconnecting phases: pre-control, control and assessment. During the baseline phase detailed information was obtained on population, prevalence, snail hosts and their habitats. The pilot area was divided into five sectors. In Sectors I to IV, those habitats which were found to be important transmission sites were molluscicided with three applications of Bayluscide (1 p.p.m.) per year. In Sectors II and III mass chemotherapy using niridazole, in addition to the routine molluscicide operations, was given. Sector V was used for comparative purposes. The pre-control phase, which began in July 1967, was complete in April 1970; molluscicide was started in May 1970 and completed in June 1970. The results were very encouraging, and indicated that S. haematobium infection was substantially reduced by the control measures undertaken at Misungwi (26).

The project data sources available to us included the following: 1) project quarterly and annual reports from 1967 to 1973; 2) published reports on the project; 3) computerized data on the human population and snail habitats; and 4) maps indicating habitat and household locations. The data covered a wide range of variables.

For each individual, the following data were obtained: study number; sector number; household number; individual number; number living in household; type of habitat used for bathing/washing or swimming; number examined in household; sex, age; length of stay in area; religion; previous treatment for schistosomiasis; education; 1968, 1970, 1972 urine examination results (number of eggs passed per 10 ml. of urine); head of household; year of registration; population movements (in and out of area or sector, or movement within sector); year of movements; and treatment history.

Snail habitat information included the following: study number; habitat number; date of survey; type of habitat; character of habitat; vegetation and vegetation clearance; measurements when full; frequency of water use; type of water use; snails found in habitat; number of houses within 300 yards of habitat; transmission potential; area and survey number.

Map data included information on the project area as a whole and on the separate sectors.[4] For the project area, there were maps showing the location of households and different types of habitats (dams, ponds, bunded fields), and maps of snail species distribution. However, only in Sector IV had both households and habitats been identified by number. Another map gave household numbers for Sector I with habitats shown but not numbered. A third map gave habitat numbers for Sectors II, III and IV and showed households without household numbers. In addition, there were more detailed maps for the Masawe indicator area, a section set aside in Sector II for special studies and as a statistical control area. The Mitando chemotherapy area in Sector II was also mapped carefully. In both these special areas, numbers were not given for household or habitats.

As may be seen from the above listing, the data available were extensive. The project determined that the age-sex structure for the sectors were similar and overall compared favorably to that for rural Tanzania (27) (see figure 5). Household distributions were similar, as were water contact patterns and socio-economic conditions. Moreover, studies indicated that age-specific prevalence and egg output were also similar. The distribution of snail habitat types varied, with some sectors having more dams. Schistosome-bearing snail species, however, were fairly evenly distributed among the sectors.

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[4] Additional map data have recently been obtained from Mwanza, Tanzania by the Ross Institute of Tropical Hygiene, London. It is possible that more detailed maps than were used in this study will thus be soon available for analysis. This will be discussed further in chapters 2 and 4.

Although population mobility was high, much of the movement was within a radius of 100 km. Thus, "S. haematobium infection at Misungwi is very highly endemic and perhaps its most remarkable feature is its homogenous, relatively non-focal distribution which stems from the widespread distribution of the snail hosts . . . and evenly scattered populace . . ." (28).

Habitat Data from WHO Project. It should be emphasized that modeling schistosomiasis transmission was not one of the objectives of the Tanzania project. Thus, in reviewing the data for modeling purposes, a number of concerns arose that led us to reorganize the data as given.

The most critical gap in the data was the difficulty in comparing houses and habitats. Water use patterns are now accepted as crucial indicators of transmission (29). It would be highly desirable to know age-specific and household-specific water contact activities. With the background material and data available to us, it was possible to make some assumptions about these patterns for Sector IV (the only sector where households and habitats were both mapped and numbered). In the other sectors, more assumptions will be necessary to make such connections. For this reason, Sector IV served as the basis for the present study (see figure 6).

In Sector IV, we considered different assumptions about the distance walked by individuals to use the different water sites. The sector, approximately 13.4 square miles, divided into three sectors — Ibilibishi, Igokelo, and Nange, contained 121 households, 660 persons, and 102 habitats. Information was not available on which persons used which habitat for a given purpose, duration, or quantitative frequency. Thus, we decided to use as the habitat variable one comparable to that in the Iran study, feet of accessible snail habitat

We considered two different assumptions of accessibility. The first was that people did not move too far from the household for the bulk of domestic activity so that they had access to all habitats within a radius of one-eighth mile. The second was that movement was really more extensive so that an outer limit of one-half mile radius would be more accurate. In rural areas, people are known to walk great distances to obtain water; in some parts of Sukumaland, people walk nine miles a day to water their cattle (30). However, in this particular sector, almost every household was close to a number of different habitats, and it is possible that most of the water contact took place close to home. In fact, the project staff did record number of households within one-fifth of a mile of each habitat, an indication that this assumption might be acceptable.

Another problem with using the data from the maps was that topographic information was limited (only edges of valleys were mapped without altitude contour lines). We assumed the area was one of gently rolling hills and that the hills offered no obstacles to habitat accessibility.

Organization of Report

The organization of the report is as follows:

In the next chapter, the model related analyses are detailed with special emphasis given to the role of intensity of infection and migration in transmission. The modifications made to the model and the results of testing the model with the Tanzania data are given. In chapter 3 the use of the model for cost-effectiveness analysis of control measures is demonstrated. The modeling and economic analyses are then combined in chapter 4 into a predictive methodology for use in project evaluation. The conclusions in chapter 5 discuss the future of modeling and economic analyses, and their role in project development and management. The methods used in calculating model related variables are given

Chapter 1

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Chapter 2

MODIFICATIONS OF SCHISTOSOMIASIS TRANSMISSION MODEL WITH TANZANIA DATA

In the first section of this chapter, we examine three groups of modifications to the transmission model. First, incidence was estimated using a variety of epidemiological and environmental terms. Second, the use of egg counts in the incidence regression equation necessitated a modification of the procedure by which prevalence in predicted from year to year. We discuss this procedure and the theoretical issues involved. Finally, the other, non-incidence related modifications to the model are discussed.

We close the chapter by discussing the predictions made by the alternative versions of the complete transmission model.

Regression Analysis for Use in the Model

The purpose of the regression analysis is to estimate incidence rates for use in the transmission equation. Incidence rates have been assumed to be a function of the non-linear interaction of environmental (snail habitat) and behavioral (water contact and epidemiological (number positive, egg counts) terms (1). With the Tanzania data, it has been possible to experiment with a number of different formulations based on environmental and epidemiological assumptions. We could only indirectly include the behavioral term because no separate quantitative study had been conducted on human water contact patterns.

Incidence was estimated using a variety of epidemiological and environmental terms (model equations are given in table 2). Each data item was collected at two-year intervals. Therefore, the regression and model analyses used a two-year prediction cycle.[1]

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[1] Our reasons for selecting the incidence data we used are outlined in appendix I.

The data were grouped by age in three ways: by each age class; 0 through 4 and 5 through 9; and 2, 3, and 4, 5 through 8, and 9-year-olds, a grouping warranted by the prevalence and incidence data.[2] The following age-specific combinations were examined:

1. Number of positive persons per age group (P_i) times volume (in cubic feet) of accessible snail habitats at the given distance (miles) from each household per age group (V_i).
2. Number of positive persons per age group (P_i) times perimeter (in feet) of accessible snail habitats at the given distance (miles) from each household per age group (PM_i).
3. Arithmetic mean egg counts per age group (E_i) times V_i.
4. Arithmetic mean egg counts per age group (E_i) times PM_i.
5. Geometric mean egg counts per age group (GE_i) times V_i.
6. Geometric mean egg counts per age group (GE_i) times PM_i.

where i indicates age. All were run for the one-eighth mile estimates. In addition, regressions 1 and 2 were run for one-half mile habitat estimates. The methods for calculating the different terms are given in appendix I along with the decision criteria for choosing accessible snail habitats.

The regression results are given in table 3, and the comparison of predicted versus observed incidence graphed in figure 7 for one-eighth and one-half miles. As mentioned before, these are two-year incidence rates for ages 2 through 9. The results indicate that the equations using perimeter and number positive, and perimeter and arithmetic mean egg counts are the most significant estimates of incidence with ages grouped as follows: 2, 3 through 4, 5 through 8, and 9-year-olds. However, equations containing volume terms predict incidence almost as well as those with perimeter terms

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and better with the one-half mile measure of accessibility. This is probably to be expected since volume is a function of perimeter. More surprising is the near absence of a difference between one-eighth and one-half mile results. This indicates that people probably make frequent use of habitats over as great a distance as one-half mile.

Role of Egg Counts in Regression Analyses and in the Model

The results from the egg count regression analyses encouraged us to delve into the issue of how best to incorporate egg counts into the transmission model. We initially attempted to relate egg counts and prevalence through the addition of an intermediate term, schistosome worm burden. The following discussion examines the value of using egg counts and worm burden estimates, and the theoretical and practical questions involved.

From an epidemiological perspective as discussed earlier, there are several reasons why egg counts should be examined for their usefulness in predictive modeling. Eggs are essential for transmission. If snails susceptible to schistosome miracidia are in the area, they are harmless unless schistosome eggs reach water and hatch into miracidia. The more eggs passed, the more likely snails vill become infected with resulting greater likelihood for human infection.

A critical aspect in determining when transmission could take place is the breakpoint, a prevalence level below which transmission cannot be maintained. It has been postulated that if the mean worm burden becomes low enough, the probability of male-female pairing will be too low to continue transmission. If such a breakpoint could be estimated, the consequences for control policies would be significant (2). One would know the target level of prevalence for breaking the transmission cycle.

These important reasons combined with the availability of egg count data prompted us to attempt to use egg counts and worm load in our modeling attempts. We were, however, interested in preserving the recursive nature of the model, that is, the prediction of incidence and prevalence in one year from incidence and prevalence the previous year. In addition, we wanted to continue to predict prevalence values since project planning involves the size of the population affected. Therefore, in order to incorporate egg counts and worm burden in the model, they must be related to prevalence. These two relationships need to be estimated for use in the model: 1) infected persons (prevalence) and worm burden; and 2) worm burden and eggs.

Theoretical Background: Prevalence and Worm Burden. Macdonald was the first to assume a statistical distribution of worm pairs (3). He assumed that worm pairs were distributed randomly in a human population and chose the Poisson distribution to describe field situations. The Poisson distribution is expressed as follows:

$$y\ (i/m,\infty) = e^{-m} m^{i}/i \qquad [4]$$

where y is the fraction of the host population infected with at least one pair of worms, i is the number of objects, m is the mean worm load, and ∞ is the limit which k, the clumping parameter, approaches. This distribution has two requirements: 1) each case is independent from the next; and 2) the probability of success in finding a positive case is constant. The population of worms, eggs, and infected persons needs to be independently, randomly distributed to fit the requirements for use of the Poisson distribution.

With animal and autopsy data measured after Macdonald's pioneering work, it was observed that worms were distributed in a clumped, not independently random, fashion. Together with the assumption that human water contact

does not tend toward randomness, it has been assumed that the distribution of schistosomes is not random but aggregated or clumped (4).[3] The clumping affects the fraction of eggs expected to be passed and therefore influences how one would model transmission processes. The distribution chosen to describe this status is the negative binomial distribution:

$$y(i/m,k) = (1 - a)^k \left[\frac{(k + 1)!\,(i+k)}{(k - 1)!\ i!}\right] a^i \qquad [5]$$

where y is the fraction of the host population infected with at least one pair of worms, i is the number of objects, m is the mean worm load, a is $m/(m + k)$ and k is the clumping parameter. The smaller the value of k, the more clumped the worms are. As k decreases, the variance increases, and the worm load in a population may be characterized by most people having zero worms and a few people having very many worms. In the case of a Poisson distribution, the clumping parameter, k, approaches infinity (∞). We have attempted to decide which distribution is more appropriate for the Tanzanian situation and if it is possible to use other distributions to relate egg counts to prevalence.

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[3]Unfortunately, the only population field data directly measuring worm burden are from autopsies although a few cases have been measured in hospitals (5). However, these data could not indicate what the distribution would be in a live human population. It is of course difficult, if not impossible, to obtain such data from live human subjects on a large enough scale.

Bradley and May (7) have derived the following equation for the fraction of the host population infected with at least one pair of worms (y) under the assumption that male and female worms are distributed together in a negative binomial fashion:

if $k \to \infty$ (Poisson distribution)

$$y_t(m,\infty) = (1 - e^{-m/2})^2 \qquad [6]$$

if $k \to 1$ (approaching high overdispersion)

$$y_t(1,1) = m^2/[(1 + m)\ (2 + m)] \qquad [7]$$

if $k \to 0$ (negative binomial)

$$y_t(m,k \to 0) \simeq k \ln\left[\frac{(m + 2k)^2}{4k(m + k)}\right] \qquad [8]$$

where m is the mean worm load per person, and k is the dispersion or clumping parameter.

We have found that as k approaches 0, it gives unrealistic results; for example, when k = 0.05, for a prevalence of 50 percent, m equals 1,000; for a prevalence of 54 percent, m equals 10,000. For this reason, in the following analyses, we used: $K \to \infty$, k = 1, and k = 0.25.

Use of Tanzania Data: Worm Burden and Prevalence, Worm Burden and Egg Counts. As outlined above, in order to incorporate egg counts into the recursive model, it is necessary to relate prevalence (or infected persons) to worm burden and worm burden to egg counts. We first estimated mean worm burden from prevalence data by using the theoretical relationships discussed earlier. For each sector, we substituted actual values of baseline prevalence y_t into equations [6], [7], and [8] and solved for m. For equation [8], k was set equal to 0.25.

Then we plotted for each sector the mean worm burden estimated in this way versus the corresponding mean egg counts. We hoped that for one of the theoretical relationships the graphs for all the sectors would have a consistent shape. This would allow us to choose the appropriate theoretical relationship between prevalence and mean worm burden and the empirical relationship between mean worm burden and mean egg counts.

Unfortunately, in looking at the graphs (figures 8 and 9), it is hard to determine which distribution is the most appropriate because there is no consistency between sections.

Since the theoretical relationship between prevalence and worm burden was not immediately apparent, we next examined the age-specific egg count frequency distribution. If one assumes that egg counts are proportional to worm loads, then the function describing the worm distribution in the population of a particular age should have the same functional form as the egg count distribution.

These results are given in figure 10. It should be noticed that there is a similar pattern to these plots, that is the greatest heights of the histograms are at the low egg count values close to zero, while there is a tailing off process that increases with age. It is difficult, however, to determine from the graphs whether the distribution is either Poisson or negative binomial. That is, with low mean values, as we have here, it is difficult to distinguish between a negative binomial or Poisson distribution. In addition, given the sample size, there are not enough individuals with high egg counts to define accurately that end of the distribution.

In sum, we decided that the extra information that could be added by worm burden would probably not be reliable enough for use in any modeling process. Further empirical and theoretical work is necessary before worm burden can be included in transmission models.

Using Tanzania Data: Prevalence and Eggs. Since we were unable to include worm burden in the model, we related prevalence and egg counts empirically. However, it was not immediately obvious what measure of egg counts should be used (see figure 11). Previous theoretical and experimental studies have reported both the geometric or arithmetic mean as well as total egg counts. When geometric mean egg counts were used in the regression analysis described earlier, geometric mean egg count had negative exponents which have no explanation cable. In addition, it seems desirable to retain egg count variability, especially the high egg counts. By using the geometric mean, we eliminate that variation. As a result, we regressed prevalence on arithmetic mean and obtained the following relationship:

$$Y = 0.0049\ E^{0.907} \qquad [9]$$

Total egg counts would be a better measure of environmental contamination than mean egg count because arithmetic mean egg count does not reflect any population increases. In making model predictions, we used equation [9] to predict mean egg count and then multiplied this value by the population size.

The results of using this empirical relationship in the model are described under model testing results.

Incidence Regression Equations Used in Modeling Analyses

Because number infected and egg counts predict incidence almost equally well, we decided to compare model predictions using number infected with those using total egg counts. Results varied little between using perimeter or volume. Volume is probably more realistic if one wants to use the model results for a cost-effectiveness analysis of molluscicide so we decided to use this unit of measurement. The results using the one-half mile measure of habitat accessibility were more significant than for using one-eighth mile. In addition, the significance of the regression differed little for perimeter and volume. We thus decided to use the one-half mile measure of habitat accessibility. The following regression equations were used in separate runs of the model:

$$A = B_0(V^{B1} \times P^{B2}) = 1.6 \times 10^{-6}(V^{0.91} \times P^{0.36}) \qquad [10]$$

$$A = \gamma_0(V^{\gamma 1} \times TE^{\gamma 2}) = 1.2 \times 10^{-8}(V^{0.07} \times P^{0.36}) \qquad [11]$$

where A is incidence, V is volume of accessible snail habitat in feet, P is number infected, TE is total eggs paired, and B_0, B_1, B_2, γ_0, γ_1, γ_2 are regression estimated parameters. The ages included are 2 through 9 year

It should be noted that the regression parameters are close to those estimated with the Iran data from an irrigated area. The exponents differ, however, from those obtained in St. Lucia where frequency of human contact with habitats was substituted for the habitat term.[4]

Additional Modifications to the Model

The model or parameters of the model were modified in several other ways to reflect the actual or likely situation in Tanzania. First, we needed to correct habitats for control measures used so that model predictions could reflect the epidemiological situation. Moreover, we needed to calculate a loss rate (B) from the data and include a natural rate of population increase. Then, in order to reflect as closely as possible the demographic situation in Misungwi, we attempted to account for migration in the model. These latter efforts are reported in greater detail.

Control Operations. In order to take control operations into account at appropriate times, we developed a chart to summarize project activities on a quarterly basis (see figure 12). With the data available from the project quarterly reports, we were able to identify on a sectoral basis which kinds of habitats were treated, the date of treatment, and the success of treatment (measured by whether or not snails were found in the habitat before the next cycle of molluscicide) for each type of habitat. It was not possible to identify by number which habitats were actually treated. Chemotherapy

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[4]The regression equation for:

a) Iran: $A = 5.7 \times 10^{-6} (H^{1.1} \times P^{0.45}$

b) St. Lucia: $A = 2.7 \times 10^{-2} (W^{0.36} \times P^{0.31})$

where A is incidence, H is meters of accessible snail habitats, P is number infected, and W is a water contact parameter. The Iran values of H and P are over ages 0-14 year olds for specific villages; the St. Lucia values for W and P are age-specific ones over all villages.

trials were also entered in the chart (see figure 12). The quarterly reports and the computer data file included information on who was treated, and egg counts after treatment. Although no chemotherapy treatment was given in Sector IV, we included this information for later use in the cost-effectiveness analyses.

For each type of habitat, the project measured the percentage of habitats found with schistosomiasis snail hosts before the beginning of a new molluscicide cycle. The cycles were as follows:

Cycle 1	16 June - 23 June 1970
Cycle 2	14 September - 16 September 1970
Cycle 3	13 January - March 1971
Cycle 4	17 May - 16 June 1971
Cycle 5	16 August - 3 September 1971
Cycle 6	15 - 20 March 1972
Cycle 7	17 - 18 July 1972

Because not all molluscicide fell within one 2-year cycle of the model, the corrections for molluscicide were incorporated into two successive cycles. The first reduction in habitat volume equaled the percentage of habitats with snails (pre-cycle 3) divided by the percentage of habitats with snails (pre-cycle 1); we corrected for molluscicide by multiplying the original volume by this fraction. The second series of molluscicide applications were simulated by multiplying this reduced volume by the fraction: pre-cycle 7/ pre-cycle 3. Table 4 contains these two fractions for each type as estimated from data in the Tanzania project reports. It should be pointed out that in a given year only treated habitats are considered in computing the percentage of habitats with snails. Ideally, this percentage should be based on all habitats treated the previous year irrespective of treatment

the following year. We did not use this value because we could not separate the habitats treated and not treated with the data available. Since all of the habitats we used were considered high transmission potential sites, it is likely they were treated. We thus believe we may be only slightly underestimating snail habitat volume as used in the model.

Estimation of Loss Rate. The age-specific loss rate was set equal to the rate at which those positive in 1968 became negative in 1970. There was no chemotherapy in Sector IV so the rate of losing the infection is likely to approximate worm death rate. The calculated value of B used in the model for all ages was 0.09. It should be remembered that this is a biennial loss rate.

Population Increases: Natural Rate. Age-specific population growth rates were estimated from information given in Ruyssenaars, et al. (8), which indicated that annual population growth rate in Sector II for the 2 through 9-year-olds was three-fourths of a percent. Working with the data from Sector IV, we also estimated an annual population growth rate for the 2 through 9-year-olds of three-fourths of a percent or, over a two-year period, 1.015. We used this as an estimate of population increases for a two-year period.

Population Increases: Migration. The role of migration in the spread of disease has been discussed at length in the literature (9). Too infrequently has the impacts of migration been discussed or considered in the implementation of disease control projects. The schistosomiasis control project in Misungwi, Tanzania, however, recognized from the beginning the importance of migration in disease control:

> A close or even broad understanding of the behavior and activities including the mobility patterns, of persons living in bilharziasis endemic areas can contribute substantially to a knowledge of the diverse factors governing the epidemiology of the disease and thus possibly contribute to the elucidation of more rational plans for controlling transmission of the infection.
>
> The principal, if not the exclusive, method for evaluating the efficacy of the control operations implemented in the Misungwi pilot area is to measure the incidence of infection in children, aged 2 to 9 years old, who were uninfected at the beginning of control operations. It is evident that mobility could be an important (if not the most important) factor confounding the evaluation results and for this reason it will have to be given the most careful consideration in any subsequent analysis (10).

Migration influences the transmission of disease by exposing people to a different set of disease conditions. Moreover, the environment may be exposed to new diseases from immigrants (11). Movements in and out of an area are known to have played a role in the spread of malaria and trypanosomiasis (12). More general health influences of population movements in relationship to new economic development projects have also been discussed in the literature (13).

The majority of studies, however, have focused on why people migrate. It is, of course critically important to understand the motives behind migration in order to predict where people move from and to, and why. These studies can shed light on the attraction of different locales and indicate what policies will achieve a more balanced distribution of population. However, even if one cannot pinpoint the reasons for moving in a given area, it is of equally critical importance to determine the impact of migration of living conditions. The few studies that consider the consequences of migration tend to emphasize the economic impact of wage rates, income levels, and labor markets in general (14).

In this instance, we are concerned about the impact of both immigration and emigration in the Tanzania 001 control project on health conditions: namely, the transmission of schistosomiasis. A detailed analysis of the impacts of migration on one of the project sectors (see figure 12) has already been reported in the literature (15). In that study, population movements were analyzed for their impact on transmission in Sector II, with an aim towards understanding the reasons for migration. In this paper, we analyze in detail migration data for Sector IV.

Migration affects the size of the resident population and the number of infected persons in a given area. Our objective in incorporating a migration component in the model was to develop a general framework that could be used in any schistosomiasis project. We planned to apply the framework to the Tanzania data and incorporate it as a sub-routine in the model. We briefly examine below the statistical technologies most commonly used in migration analyses.

Since the process of movement could be thought of as random (given no significant attractive force) and as taking place at discrete times, we considered the use of a Markov chain process for migration (16). A Markov process could give the probability of migration in and out of infecteds and uninfecteds, but the estimation requires one critical value: the size and prevalence of the population from which the immigrants come. We had no access to that term since the potential population for immigrants in this area is vast. We decided that although the Markov chain process could be a valuable tool in modeling, it was not feasible to use in this situation. For much the same reason, a gravity model often used in geography for migration was also thought inappropriate (17).

If we were simply accounting for the likelihood of new infections in an uninfected area, it might be possible to add a random variable to the regression equation we used to predict incidence rates. Random number computer programs exist and yield normally distributed random variables with mean 0 and variance 1. It may be possible to add this random variable (R) to each variable in the regression equation:

$$\text{(Incidence)} \quad = (B_0 + R_0)\left[V^{(B_1 + R_1)} \times P^{(B_2 + R_2)}\right]$$

(Reversion) $B = .2 + (R_3 \times$ standard deviation of B if estimated)

This may, however, oversmiplify the stochastic process involved in transmission.

Neither Markov chains nor gravity models nor the random component seemed to be satisfactory methods. We did not have sufficient data for the first two techniques, but since we did have some data, the random component method was not necessary. We thus decided that for the purpose of these analyses, the most satisfactory method would be to estimate an age-specific average rate of migration of infected and uninfected persons for direct use in the model.

The Method Used to Incorporate Migration into the Model. We assumed that migrants enter or leave the area after being counted in the 1968 census. In the following table of symbols, N represents the number of people in the indicated category.

Migration Status

Infection Status	Population in area, emigration (e)	1968 Census no migration (n)	immigration (im)
Infected (i)	$N_{i,e}$	$N_{i,n}$	$N_{i,im}$
Uninfected (i)	$N_{u,e}$	$N_{u,n}$	$N_{u,im}$

$$\text{1968 prevalence uncorrected for migration} = \frac{N_{i,e} + N_{i,n}}{N_{i,e} + N_{i,n} + N_{u,e} + N_{u,n}} \qquad [12]$$

The prevalence must then be corrected for those who immigrate and emigrate after the 1968 census. The population size after migration = $N_{i,n} + N_{u,n} + N_{i,im} + N_{u,im}$. The number infected = $N_{i,n} + N_{i,im}$.

$$\text{1968 prevalence corrected for migration} = \frac{N_{i,n} + N_{i,im}}{N_{i,n} + N_{i,im} + N_{u,n} + N_{u,im}} \qquad [13]$$

The corrected 1968 prevalence is used in the transmission equation [2] to predict the 1970 prevalence.

To calculate the various terms in the numerator and denominator of equations [12] and [13], certain assumptions were made:

1. The fraction of the sector that emigrates stays constant.
2. The prevalence in the emigrant group stays constant.
3. The size of the population pool from which the immigrants come will increase at the same rate as the population in the sector. This is equivalent to having the fraction of the pool that immigrates remain constant.
4. The prevalence in the immigrant group will remain constant.

The relationship between prevalence and eggs is somewhat different for Sector IV than for the other sectors. By adding immigrants to those already residing in the sector, we assume that the same relationship holds for the immigrants as for those in Sector IV and thus is different from the other sectors.

Misungwi, Tanzania, Migration Data. Because the project staff recognized the importance of migration in influencing control efforts, the following migration information was recorded for each individual:

I. First Movement

A. Location

1. Moved in from outside pilot area
2. Moved in from other group sectors in pilot area
3. Moved within one group of sectors
4. Moved to other group of sectors in pilot area
5. Moved to outside pilot area
6. Died
7. No previous record but claims to have been in area at first registration
8. Persons not traceable although previously registered
9. Moved in from area not stated
10. Moved out to area not stated

II. Second Movement

A. Items 1 through 10

B. Year of second movement

Immigrants were those people coded with a 1 or 2 in their data file; emigrants were those people coded with a 3 or 4. We tabulated the number of 2 through 9-year-olds in the following groups: in-movement of infected persons, out-movement of infected persons, infected residents, in-movement of uninfected persons, out-movement of uninfected persons, and uninfected residents. The calculated values are listed in table 5 .

The results of including migration data in the model are described in the following section.

Results of Model Testing

The basic model encompasses the estimated incidence equation [10] and transmission equation [2]. In this section, we describe the results of incorporating model modifications and offer some conclusions about their relative importance.

Modifications made possible by the availability of the Tanzania data include:

1. Modifying equation [10] to use total eggs instead of number infected as an independent variable
2. Modifying the prevalence and population to reflect migration in and out of the area by infected and uninfected persons.
3. Modifying the habitat term in the equation [10] to allow for seasonal variation

The different analyses are listed in table 6 .

Comparisons of these modifications were made by observing the differences in prevalence predictions obtained by 1) use of the modification and 2) without the modification. To assist the reader in understanding where

the modifications are made in the model, we have developed a complex flow diagram (figure 13) which will be referred to in the discussion. The results of these comparisons are given in tables 7-10 and figures 14-16 and will also be referred to throughout the discussion in this section. We present the results in a number of different methods to discuss the variation in predictions. In tables 7, 8, and 10, we give the predicted fraction of positive for each age group under different modifications for 1970, 1972, and 2004, the year where prevalence started to level off. In figures 14, 15, and 16, we graphed the age prevalence curves for the different modifications in 1970, 1972, and 2004, respectively. In figure 17, we graphed the age-prevalence curves for observed data and the different predictions for each modification separately. In figure 18, we graphed over time observed data versus predictions from the final version of the model, which reflected most closely the actual activities.

Results from the different modifications are discussed in the following order: 1) number infected versus total eggs; 2) migration; and 3) seasonal variation. Then we compare observed data versus predicted results from the final version of the model.

Comparison of Prevalence Predictions: Number Infected and Total Eggs. The original incidence equation [10] using number infected as the independent epidemiological term, was modified to use total eggs. As described earlier, this was accomplished by estimating a relationship between prevalence and arithmetic mean eggs (equation [9]). Predicted prevalence values were substituted into the equation to obtain an arithmetic mean egg value which was then multiplied by the total population to obtain total eggs. This replaced number infected as the independent epidemiological term used in equation [11] to estimate incidence.

The resulting prevalence predictions using 1) number infected and 2) total eggs are given in separate columns in tables 7, 8, and 10. For each run of the model, ranging from "no modification" to "migration and molluscicide," we made a set of age-specific predictions for the two different epidemiological variables. By looking at the last column, the following results were obtained. For 1970, predictions varied by 0.03 or less, with the majority of predictions differing by only 0.01. For 1972, predictions varied slightly more, with total eggs generally giving lower results. The differences, however, were never more than 0.05. In looking at the results for 2004, greater differences may be seen when controls are used.[5] Under the molluscicide examples, the differences range up to 0.22 (this may be due to sample size) in one instance and 0.10 through 0.06 in four cases. Except for these five points in 2004, there are, for practical purposes, no differences in results.

One may therefore conclude that the use of egg counts in predictive modeling does not significantly alter predictions, unless one is making long-run predictions (20 years).[6]

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[5] It is interesting to note, however, by reference to table (in the cost-effectiveness analysis section), that when we hypothetically include chemotherapy as a control measure (described in that section) along with molluscicide use, the use of total eggs considerably lowers results than use of number infected. This may, however, be related to the changes made in population numbers for migration and natural rate of increase and not the use of total eggs. Nonetheless, under chemotherapy and molluscicide use together, with the same population changes made for both terms, total egg predictions differ from number positive results. Under sole use of molluscicides or chemotherapy, the two terms do not yield dramatically different results.

[6] Print-out results available from Rosenfield.

Comparison of Prevalence Predictions: Migration Included and Migration Not Included. In the model, migration influences the size of the susceptible and infected population. As described at length under the migration section, we accounted for the movement in and out of an area of infected and uninfected persons by using migration rates from the Tanzania data. The flow diagram (figure 13) shows how migration is included in the model./ The 1970 and 1972 rates of immigration and emigration were used to run the model over time. This assumption may be crude, but it is the only one we could justifiably make.

The results of including migration and not including migration are given in tables 7, 8, and 10, and may be seen more clearly in figures 14 and 16. To assess the impacts of including migration, it must be realized that there is a dynamic relationship between 1) infected and uninfected people immigrating, and 2) infected and uninfected people emigrating, each group influenced by population size changes. The greatest impact of migration on changing predictions occurs under use of control measures, especially as one extends the time period (table 10). The other categories show smaller shifts in predictions due to migration.

The slight differences in migration results over time may be due to the fact that we kept emigration and immigration rates constant. Thus, the relative importance of contribution to prevalence levels from migrants would decrease as population size and resident prevalence levels increase over time. This may also explain why the effects of migration are greatest when control measures are used.

Migration, in combination with natural population increases, could cause control program planning to go astray. Migration may be an obvious concern but only a few control projects, such as the one in Tanzania, have

explicitly accounted for immigration and emigration in their planning. Since under control measure use, migration affects prevalence and thus transmission, this activity should be recorded in control program data collection.

Comparison of Prevalence Predictions: Seasonal Variation. Seasonal variation is included in the model through changes in the habitat term. We substituted habitat values for Sector V where no control activity had taken place for those in Sector IV to see how seasonal variation would offset prevalence predictions (tables 7, 8, and 10).

For 1970 results, one should compare predictions made with no modifications to those made with migration and seasonal variation. The results are not consistently greater or smaller but do differ except for the 4-year-olds. For 1972 and 2004, one should compare migration results with migration and seasonal variation. In both instances, the migration prevalence levels are greater than those predicted under migration and seasonal variation. The differences become greater over time. Seasonal variations, especially in periods of drought, could limit transmission. Yet, it is interesting to note that seasonal variation and migration consistently yield higher prevalence values than molluscicide and migration. Seasonal variation, although considerable in this area, is not sufficiently high to influence control measure results. Indeed, seasonal variation causes only slight reductions in prevalence predictions in 1972, and somewhat greater reductions in 2004. Seasonal variation, although important in understanding snail population dynamics, does not significantly influence human prevalence predictions over time.

Comparison of Prevalence Predictions: Predicted Versus Observed Values. The test of a model's reliability is how closely model predictions fit reality. To demonstrate the fit of model predictions with field observations, it is necessary to compare the prediction with the appropriate corresponding set of observations.

The baseline data we used as input to model predictions were obtained from those households which were on the Sector IV maps and in the computer data file.[7] We used 114 households out of 221. Furthermore, we focused on the 2-through-9-year-olds because there were two years of observed data in addition to the baseline observations so that incidence and reversion rates could be estimated. The older-than-9-years data were only given for 1968 and 1972. This one sample was quite special and small: There were a total of 660 for all age groups; the numbers in the 2-through-9 age groups are given in table 11.

Because of the decision criteria we used to determine baseline data, it was necessary to correct Sector IV data for all years by eliminating seven households not on the maps. Then we had to decide what age each individual was in a given year. We assumed, for example, for someone to be 2 years old in 1970, he had to be listed without 1968 data and as 2 years old. We assumed 4-year-olds in 1970 to be those individuals listed as 2 years old and having 1968 data (assuming the age listed is for the first census survey) plus those listed as 4-year-olds with no 1968 data. We did not use the registration number given as the basis for age determination because it did not jibe with egg count data dates. Thus, the observed data contained the group of 2-through-9-year-olds corrected for age and household.

[7] We could only use Sector IV data for the reasons outlined in Chapter 1.

In addition, for 1972, we had to determine which was the appropriate set of observed data. We have listed in table 9 monitor group data, all household data corrected for age based on date of urine examination, households used in model predictions corrected for age based on date of registration, and households used in model predictions corrected for age based on date of urine examinations. To be consistent with our 1970 decision, we used the last measure.

The results from the different model analyses for 1970 and 1972 are plotted in age-prevalence graphs where predicted prevalence values are compared with observed data (figures 14-18), note especially the 1970 graph, figure 14. The data for these graphs are given in tables 7, 8, and 10. Model modification that reflects the actual situation in Tanzania includes both molluscicidiing and migration. It is interesting to note, however, that other comparisons, for example 1970 values under seasonal variation and migration, seem to give a closer fit. It should be mentioned that we assumed the same migration rates for 1972 as for 1970 which could be a major source of error in the 1972 predictions. We also noted the large decrease in observed prevalence for 1970 to 1972, which could not be explained by either molluscicidiing or migration.

Several sources of error may be responsible for the problem with replicating the situation in Tanzania. One source may be the small sample size. For example, the dramatic drop in 1970 prevalence for the 6-year-olds could not be explained by the field situation. The irregular shape in the observed age-prevalence curve contrasts with plots we made of egg counts which showed a consistent increase over age until age 15 (figure 19). These, however, were made for all sectors, that is, the overall sample size was 4,000, not 208. A second possible source of error may

be the technique we used to estimate incidence. Our grouping the data resulted in highly-significant incidence equations, but this same grouping could have been misleading for the overall model. We feel that both the sample size and incidence grouping could have combined to throw awry model predictions. The results show how important it is to include all the data in model testing. It is hoped this will be accomplished in the future (see conclusions below).

The inconclusive results of model testing with observed data led us to regard the results under different model modifications as a form of sensitivity analysis. The different assumptions about the real-world situation led us to modify the input data by different events. We were able to observe how prevalence predictions would vary under different assumptions related to units of the epidemiological term, migration, seasonal variation, and control use. This is not the usual form of sensitivity analysis where, for example, the regression exponents might be changed in a consistent way to see how prevalence predictions vary and to which term they are especially sensitive.

The modification results enabled us to examine to which assumptions model predictions are sensitive. The results already discussed above are in figures 14-16 and tables 7, 8, and 10. It appears from figures 14 and 15 that the model is fairly robust to the different assumptions used. Only slight changes are observed between any of the assumptions for 1970 and 1972. Over time, however, in looking at the graph for the year 2004, prevalence predictions under use of controls and of migration and controls are lower than under separate runs for migration and seasonal variation. As pointed out earlier, there are no significant differences in predictions obtained from use of 1) number infected and 2) total egg counts.

Conclusions

The different analyses indicated the sensitivity of the model to different assumptions. Results from each analysis demonstrated that predictions were highly sensitive to the role of control measures alone and in combination with migration. Seasonal variation appeared to be less important in short-run modeling that was expected. Egg counts and number infected showed only slight differences in predicted values except over the very long term.

The results from the modeling analaysis may be summarized as follows:

1. In the short run, differences in predictions between use of total eggs or of number infected are slight. For predictive modeling, either term may be used; the choice should reflect scarcity of financial and manpower resources.
2. In long run predictions, differences between predictions from total eggs or from number infected appear to be greater in the short run. In this instance, data on total eggs may be useful to collect.
3. In both the short and long run, migration is a critical factor to consider when planning a control program.
4. Seasonal variation is of limited importance in model predictions when the emphasis is on predicting changes in the human population over time. It is, of course, critical when snail populations play a larger role in modeling or planning analyses.

The emphasis for future research will be to try to use newly available cross-sectional data information torun the model over all sectors. It is expected this will be undertaken the next year.[11] The cross-sectional

[11] Arrangements are already being made for Rosenfield to work with the new set of data unearthed at Mwanza.

data should enable the validity of the model to be tested more definitely since all ages and all sectors will be included, and the sample size will be significantly increased.

One aspect that cannot be tested and which is of critical importance is the longitudinal validity of the model. The project ran from 1967 to 1973 yet data, except for the monitor group where there were identification problems, were only available for all ages in the pre-control year 1968 and then 1972, four years after control measures started in some sectors. Future projects should strongly consider reporting on all ages each year so that control measures can be evaluated on an annual basis. Furthermore, such data reporting could greatly assist in the verification of the validity of model predictions.

Control programs may be evaluated on both their epidemiological results and their costs. In the next chapter, the transmission model is used as a framework to evaluate costs and effectiveness of control measures. The combined use of epidemiological and economic analyses, along with measuring future work, are discussed in the concluding chapter.

Chapter 2

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Chapter 3

USE OF SCHISTOSOMIASIS TRANSMISSION MODEL TO ANALYZE COSTS AND EFFECTIVENESS OF CONTROL MEASURES

Introduction

Decisions about which control strategy to use for preventing or controlling schistosomiasis transmission should involve consideration of demiological and economic conditions. The use of a transmission model would enable the decision maker to decide which single control measure, or combination, would be most effective in reducing prevalence in the population, thus limiting transmission. Control measures, however, involve costs of manpower, materials, and money. How these costs vary for each control measure needs to be considered when deciding which control measure to use. Control measures may then be compared for their costs and effectiveness in reducing schistosomiasis prevalence. It should be noted, however, that "while cost-effectiveness provides the necessary criterion to choose between alternatives to achieve a specified goal or between different degrees of goal achievement for a specified input, it provides no information about the desirability of goals" (1).

It is assumed in this report that the goal of reducing schistosomiasis prevalence or transmission has already been set. Thus, cost-effectiveness analyses may be used to define "the efficiency of different input combinations to achieve a goven goal or set of goals." (2)

In this section, by means of the transmission model and Tanzania data, we compare the costs and effectiveness of three control strategies with use of no controls: molluscicide, chemotherapy, and molluscicide plus chemotherapy. Because the Tanzania control project was experimental, we are not evaluating the actual situation in Misungwi. The aim of this section is to use the data

to show how the model provides an analytical framework for project evaluation. With cost data available from the project, the cost-effectiveness of the strategies may then be compared.

The chapter is organized as follows. After a description of our measure of effectiveness, the model is used to evaluate control measure effectiveness. We then compare the cost-effectiveness of each strategy. The chapter concludes with a consideration of research needs for new techniques and additional data requirements.

Measures of Effectiveness

The prediction of incidence involves variables that are affected by control activities: feet of snail habitats and number of infected persons or total eggs passed. Thus, the effectiveness of molluscicide in reducing snail habitats and of chemotherapy in reducing number infected or total eggs may be examined through changes in prevalence predictions. From the changes in number infected as predicted by the model, the control strategies may be compared for their effectiveness.

The measure of effectiveness used in this analysis is case-years of infection prevented (3). Although other measures may be used, such as percent reduction in prevalence from one time period to another, case year of infection prevented is more desirable because it includes both an infection and a time component. Effectiveness is measured as follows: the number of cases occurring with use of controls are subtracted from those occurring without, to obtain cases prevented for each year; the sum of the differences gives the case-years of infection prevented over the total period of analysis.

The control strategy which prevents the greatest number of case-years of infection is the most effective. The period of analysis we chose was twenty

years or ten cycles from the baseline years 1968 through 1978. Again, only the two through nine year olds were considered. Additionally, we compared case-years prevented between model runs using number infected versus those using total eggs.

For the calculation of the no controls situation, we changed the habitat term to account for seasonal variation in transmission. We used the data from Sector V, where no controls of any kind were used, to estimate how the percent of habitats with snails would have changed due to seasonal influences only. The procedure for calculating these percentages is similar to that for molluscicide given in Appendix 1. The percents used are given in table 12.

We first looked at the effectiveness of six cycles of molluscicide. As described in chapter 2, we calculated the percentage of snail habitats where snails were still found and used that to reduce accordingly each type of habitat for the use of control measures (4). In the model, we changed the volume of habitat used to estimate incidence in both 1970 and 1972 as described in Appendix I. The percentage reductions in habitat are given in table 4. The results from the molluscicide are given in table 13.

For chemotherapy we calculated the achieved percent reduction in prevalence by using results from Sectors II and III where chemotherapy was used. Molluscicide was also used but the data showed that most of the reduction came from chemotherapy, with a 40% reduction in prevalence occurring after the first treatment (5). Since there was no second round of treatment, we used the results from the project chemotherapy study area (Masawe). The second year of treatment in Masawe resulted in only a 25% reduction in prevalence due to infected immigrants (6). Again, the results are given in table 13.

We compared results from the three control strategies with results from not using controls. For two different durations of model predictions. The control program lasted from 1968-1973; but in order to show the effects of chemotherapy, we added one additional cycle. To discover what long term effects a short term control usage would have, a longer sequence of predictions--10 cycles and 20 years--was used. Results for the shorter time period show that with both number infected and total eggs, the combined strategy is more effective in preventing case-years of infection (table 13) than the others. Chemotherapy is itself more effective than molluscicide. The numbers from which these calculations were made are given in table 14.

The long term results are similar, although the combined control strategy prevents more case-years of infection than the other two, especially when using total eggs (see table 15).

Cost Calculations

The project estimated total costs for both molluscicide and chemotherapy. The molluscicide costs were calculated for the entire project area of four sectors; the chemotherapy costs were for 1,000 persons who were diagnosed and treated, if positive (7). We corrected these costs for Sector IV data by dividing the molluscicide total costs by four to get an approximate per sector cost. For chemotherapy, we corrected the total costs by our population size of two through nine year olds: 212 in 1970 and 223 in 1972.

The total costs we used were:

Molluscicide	Chemotherapy	Molluscicide plus chemotherapy
US$5,223	US$1,465.95	US$6,688.95

Many assumptions were made in using these costs because of the lack of more detailed cost information. Our stated objective, moreover, is mainly to demonstrate model use for cost-effectiveness; the data were sufficient for this purpose.

Cost-Effectiveness Comparisons

To determine the cost-effectiveness of the different strategies, we divided the total control costs by the appropriate case-years of infection prevented to get cost per case year of infection prevented. We determined these values only for the years 1968-1974, since cost data applied to that time frame.

Results given in table 16 indicate that chemotherapy is the most cost-effective strategy. The combined effort is most effective in reducing case-years of infection, yet its costs are on a per case basis three times more expensive than chemotherapy. Mollusciciding is considerably more expensive and less effective. There are no significant differences in cost-effectiveness results between use of number infected and total eggs.

It should be mentioned that these analyses do not include usual components of project analyses, such as budget constraints, discount rates and sequencing. The optimal control strategy from cost and effectiveness perspectives may be determined within the framework of an optimization procedure that accounts for such aspects. To apply optimization techniques involves detailed knowledge of investment requirements for the control measures, resource capacity constraints, labor supply, wage rates, interest rates, and salvage costs. A study at the World Bank is now underway using a dynamic programming framework for optimization (8). The results from this study will yield information on the optimal control strategy under budgetary or capacity constraints. The cost data

available to us, however, were not extensive enough to warrant using sophisticated analytical techniques.

Conclusions

The results from this study indicate that a transmission model may be used to demonstrate the effectiveness of control measures. The necessary input data are the number of habitats treated or habitats where snails remain, and the number of people treated or fraction of the population treated.

The effectiveness measurement showed the combined controls were most effective in reducing case-years of infection, but the cost-effectiveness analysis indicated that chemotherapy was the most cost-effective treatment. Molluscicide might be even more costly over time because of the need to maintain snail-free habitats. In addition, drug costs will decline over time as the number of individuals needing treatment decreases.

This analysis has not included other controls such as water supplies to limit human contact with snail habitats and engineering measures to reduce snail habitats. Further work is needed to evaluate their role in reducing schistosomiasis transmission.

An additional conclusion from these analyses is the lack of difference between use of total eggs and number infected to estimate prevalence in the short run. Over time, the differences appear to be greater but it is unclear whether that is due to our assumptions or the differences in the terms. Nonetheless, in the short-run, it is likely a project planner may base his decisions to use either one of these terms on cost considerations and not worry about the degree of information he may lose by his choice.

Further work in estimating more accurate cost functions for the range of control strategies available is needed. It will be necessary to consider

additional terms, e.g., discount rates, for long-term cost analyses. The main conclusion is that a transmission model may be used in conjunction with cost analysis to evaluate different control strategies.

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Chapter 4

CONCLUSIONS: INTEGRATING EPIDEMIOLOGICAL AND ECONOMIC ANALYSES

The primary focus of this report has been to expand a methodological approach to answering these questions concerning schistosomiasis transmission associated with water resources development projects:

1) How will the project affect schistosomiasis prevalence levels in the population at risk?
2) What is the most cost-effective mechanism for a) limiting the project's impact on schistosomiasis prevalence levels and b) preventing transmission from starting or increasing?

These questions relate to one of the objectives of the report which is to assist project managers in designing projects with minimal adverse health impacts.

It should be stressed that the methodology does not include consideration of the benefits of disease prevention and control activities. Evaluation of the benefits requires more information on how schistosomiasis affects human productivity than is currently available (1). The first step in any project evaluation, however, is usually to assess benefits. Until a quantitative measure of benefits is available, the impacts of a water resources project on schistosomiasis transmission will not be able to be incorporated in project planning in a way truly compatible with other aspects of project analyses. The emphasis of this discussion is the next stage in project planning. That is, after concern about schistosomiasis is sufficiently high so that project managers decide to practice preventive planning, the methodology described here provides a way to implement that decision.

A question associated with the earlier two arises at this stage. Should a manager be concerned about prevalence levels or transmission? Prevalence levels, whether measured by number or fraction of infected persons or by total eggs passed by individuals, indicate the extent of the problem in the project population at a given point of time: pre-project, during construction, and in the operational stage. Prevalence may be measured by a population-based epidemiological survey. Transmission, that is, the rate of change in prevalence levels over time or the incidence rate, is a significant measure of rapidity of spread of infection. It could be used to assess the impact of the project on changing epidemiological conditions. To measure incidence rates, however, requires a special, time-consuming study of noninfected persons; such a study if conducted first under pre-project conditions should then be repeated, on a regular basis, after construction to measure the impact on schistosomiasis. Often the pre-planning phase of a project takes many years so that it may be realistic to initiate such a study. Incidence studies involve a well-defined sample of the population so they do not indicate the level of infection in the population as prevalence does. We thus must conclude that both terms are necessary to predict the full impact of a water resources project on the health of the human population.

In the remainder of this chapter, data needs for using the schistosomiasis transmission methodology are discussed along with suggestions on the method of reporting. We describe how the epidemiological and economic analyses may be integrated into project planning through choice of appropriate physical control measures, incentives to implement the measures, and institutional arrangements to ensure long term meaintenance of control efforts. These latter three aspects combine to form a schistosomiasis management strategy for use in water resources

project development. The concluding discussion raises some issues for future research.

Data Requirements

In Chapter 1, basic data requirements for use of the methodologies described in Chapters 2 and 3 were presented. Examining modifications to the methodologies has resulted in a reordering of priorities for data collection for the predictive model (see Table 17).

The epidemiological, environmental and demographic information assists in analyzing which combination of control measures would be most effective in reducing or preventing transmission. The economic information enables a project manager to determine the most cost-effective strategy in the context of the particular project. A sample of the project population could be surveyed but the data should ideally be reported for each individual on an age and sex specific basis, as the Tanzania project did giving each individual a number and coding all information by reference to that number to facilitate evaluation studies. Even the habitat information should be coded in reference to individuals so that who uses which habitats may be identified. To predict post-project conditions, information on where new snail habitats may develop (e.g., side pools associated with irrigation canals) and the proximity of human settlements to those habitats should be utilized.

In organizing a full scale control program, additional data and more detailed preliminary surveys would be needed. The data discussion in Chapter 1 is perhaps more appropriate for control programs. It should be stressed that reporting data on an individual, not aggregate, basis ensures the usefulness of the data for a variety of purposes.

We have presented in Table 1 what we consider to be a satisfactory minimal amount of information needed for predictive analyses. However, in practice, few projects collect the same data items and report them in a similar way (2). Comparison of results from different projects is a difficult task. Although each situation may require site-specific modification, in the future thought should be given to the comparability problem so that scarce resources are not wasted. Ideally, with reporting of common items for each individual in the project or study, a data bank containing the information from schistosomiasis control projects could be instituted. If such information were easily available to project managers and evaluators, preventive decisions could be made on a more rational basis.

Integrating Epidemiological and Economic Analyses

Data for predicting the impacts of water resources projects are incorporated into the transmission model as follows. The first step is to estimate incidence as a function of habitat size and number infected. The habitats used are those with which the project population has contact. The data should be collected on an age-specific basis (at least) for all ages. The sample should be large enough so that estimation of regression parameters will yield statistically useful results.[1]

The next step is to estimate the natural rate of population growth, migration rates, and a schistosomiasis loss rate. The population growth and migration rates, probably based on previously collected data, would be used to modify prevalence predictions. As mentioned in the migration section, this part of the modeling effort requires further research to attempt to develop a more general method for incorporating migration into the model. A loss rate may be approximated; this term also needs more field information.

[1]Sample size is an important question discussed at length in both statistical and public health publications (3). Small sample size combined with high variance in the data frequently yield statistically insignificant results which increase the uncertainty in planning.

The initial predictions are thus made on the basis of baseline prevalence, migration, total population, incidence rate, and loss rate. The prediction process continues over time with changes made in the habitat term as necessitated by project activities. The habitat term, total population, and migration rates would reflect changes estimated by the water resources project planning staff.

The ensuing predictions should approximate the magnitude of the project's impacts. If sufficient longitudinal or cross-sectional data are available, the error in such estimates may be measured by the differences between predicted and observed prevalences levels. As a form of sensitivity analysis, different assumptions about habitat changes and migration could be made to estimate a new set of predictions. Estimates of prevalence from different initial assumptions would indicate how sensitive prevalence is to items over which the project staff has some control, such as length of irrigation canals or their proximity to human settlements.

If the schistosomiasis problem is thought to be serious enough to warrant incorporating preventive measures into project design, then the costs and effectiveness of alternative methods may be estimated by use of the model as described in Chapter 3 and elsewhere (4). Control measures as described in Chapters 1, 2, and 3 affect the number infected and habitat terms. The effectiveness of alternative control strategy may be estimated and then compared on a cost basis. The most cost-effective strategy may then be chosen.

After determining which control strategy to use, the health project planner needs to consider how to implement the strategy and which institutional arrangements would be the most appropriate. This subject has been discussed at length elsewhere (5). Unless implementation incentives and institutional arrangements

are part of preventive planning, the physical control measures might not be effective.

Use of economic, legal and social incentives ensure implementation of control measures. Economic incentives could include requirements such as tying water resources project loan agreements to control of schistosomiasis, imposing fines on the water authority for improper construction of canals. or providing free molluscicides for snail control and drugs for chemotherapy. Legal incentives could include establishing and enforcing regulations to limit human contact with snail habitats. Social incentives could include health education for the project population so that they understand the need for control measures.

Institutional arrantements need to be considered in the context of the specific project. Cooperation between the relevant different levels of government is essential. Local health centers could be linked with local representatives of the water ministry; indeed, the ministries of health, water, power, agriculture and development (finance) should all cooperate in the implementation of control measures. Since many water projects, especially large ones, are planned and managed by a central but locally based authority, such cooperation could possibly be realized by establishing a health section of the water project authority. If financial backing is sufficient, long-term maintenance of control measures should then be accomplished.

Future Research Recommendations

The most convincing test of the methodology described in this report will be to apply it in a new situation where the appropriate data may initially be collected. Impacts on prevalence can then be predicted, and the results compared over time with the actual events. Longitudinal verification, difficult to

achieve because of associated costs and efforts, can most effectively be accomplished in the context of a new project so that resources are not wasted in trying to compare incompatible data from projects established at different times. Longitudinal testing is of critical importance to determine the model's usefulness in assisting the planning process.

Further work with the Tanzania data is already planned. This will involve using the data from the other sectors and older ages to verify the model [a similar procedure to what was done in an earlier study in Iran where the transmission model was developed for 2-to-14 year olds in 14 villages and then tested with data from all ages and 52 villages (6)]. Since the newly available map data will, it is hoped, make it possible to match habitats and households in all sectors, this future effort should result in a cross-sectional verification of the model. Of course, the longitudinal questions can not be resolved with this set of data.

Migration presents another important area for future research. Developing a general model to account for the impacts of migration on schistosomiasis transmission would assist in increasing the applicability of the transmission model to a variety of economic development projects.

Water contact studies are needed to determine to what extent snail habitats influence transmission. Several studies have been carried out in different areas but the methodology requires further development and testing for routine use (7).

Conclusions

In this report, an expanded version of a previously developed methodology for predicting the impact of water resources projects on schistosomiasis transmission has been presented. It has been shown how the methodology may be used

to estimate costs and effectiveness of alternative control measures. The transmission model has been expanded to include migration rates as part of the predictive methodology. It was also shown how egg counts could be used in predictive modeling, although it was concluded that the differences in prevalence predictions from use of egg counts and from use of number infected were not significant. It was noted that the regression parameters from the Tanzania data, using similar independent variables, were almost identical with those obtained in an earlier study with data from a project in Iran. Further work with the modeling and economic analysis were suggested.

Through the use of methodologies such as the one described in this report, schistosomiasis can be a quantatively predictable result from water resources projects in developing countries. Schistosomiasis transmission may be prevented if these predictions along with appropriate health management strategies are incorporated in the design and operation of future water resources projects.

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TABLES

Table 1. -- Preliminary Priority Listing of Data Needed for Predictive Modeling and Project Evaluation: A Modeler's Viewpoint

Epidemiological	Environmental	Controls	Economic
Total numbers in each age group Number examined/age/sex on an individual basis Number infected/age/sex on an individual basis Number eggs passed/unit urine or feces/age/sex on an individual basis Map of household location in relation to type of snail habitats Human-snail contact pattern Population migration data Transmission sites identified for each individual Incidence and reversion rates/age/sex	Topographic maps Characteristics of snail habitats (e.g., dimensions, vegetation, seasonal variation)	Date of chemotherapy reduction in egg counts/age/sex on an individual basis Date of molluscicide of individual habitat basis	Employment activities/age/sex Domestic activities/age/sex Wage rates for control activities (professional and other labor) Equipment and material costs for controls

Table 2. List of Equations Referred to in Chapters 1 and 2 and Definitions of Variables

	Equation number
Chapter 1	
$dy/dt = A(1 - y) - By$	[1]
$y_{t+\Delta t} = \left(y_t - \frac{A}{A+B}\right) e^{-(A+B)\Delta t} + \frac{A}{A+B}$	[2]
$A = \beta_0 \left(H^{\beta_1} \times P^{\beta_2}\right)$	[3]
Chapter 2	
$y(i/m, \infty) = e^{-m} m^i / i$	[4]
$y(i/m,k) = (1 - a)^k \left[\frac{(k-1)!\ (i+k)}{(k-1)!\,i!}\right] a^i$	[5]
$y_t(m, \infty) = (1 - e^{-m/2})^2$	[6]
$y_t(1, 1) = m^2/[(1 + m)(2 + m)]$	[7]
$y_t(m, k \to 0) \simeq k \ln \left[\frac{(m + 2k)^2}{4k(m + k)}\right]$	[8]
$y = 0.0049E^{0.907}$	[9]
$A = \beta_0 \left(V^{\beta_1} \times P^{\beta_2}\right) = 1.6 \times 10^{-6} \left(V^{0.91} \times P^{0.36}\right)$	[10]
$A = \gamma_0 \left(V^{\gamma_1} \times TE^{\gamma_2}\right) = 1.2 \times 10^{-8} \left(V^{0.07} \times TE^{0.45}\right)$	[11]
1968 prevalence uncorrected for migration = $\frac{N_{i,e} + N_{i,n}}{N_{i,e} + N_{i,n} + N_{u,e} + N_{u,n}}$	[12]
1968 prevalence corrected for migration = $\frac{N_{i,n} + N_{i,im}}{N_{i,n} + N_{i,im} + N_{u,n} + N_{u,im}}$	[13]

Table 2 (continued)

List of Variables

y = Prevalence or fraction of host population infected with at least one worm pair

A = incidence rate

B = loss rate

t = time

H = environmental/behavioral term

P = epidemiological term, numbers of infected persons

m = mean worm load/person

i = number of objects

k = clumping or dispersion parameter

a = m/(m + k)

E = arithmetic mean egg/age group

V = feet of accessible snail habitats/age group/Sector IV

TE = total eggs paired/age group/Sector IV

β_0 β_1 β_2 γ_0 γ_1 γ_2 = regression estimated parameter

$N_{i,e}$ = population in area, emigrating and infected

$N_{i,n}$ = resident population, infected

$N_{u,e}$ = population in area emigrating and uninfected

$N_{u,n}$ = resident population, uninfected

$n_{i,im}$ = population in area, immigrating and infected

$N_{u,im}$ = population in area, emigrating and uninfected

Table 3. Results from the different regressions are presented in this table. The regressions were run for distances of 1/2 and 1/8 mile (assumptions of habitat accessibility) for different age groupings. The variables in the regressions were: A, incidence; V, volume of accessible habitats (cu.ft.); P, number of infected persons; E, arithmetic mean egg count; GE, geometric mean egg count; PM, perimeter of accessible habitats (ft.); β_0, β_1, β_2, the regression coefficients and exponents specific for each equation. R^2 is the percentage of variance in the dependent variable (I) explained in the equation; F is the F-statistic which indicates the significance of the R^2, the numbers in parentheses (2,5) are the degrees of freedom; "t" is the statistic which indicates significance of the β's at 5 degrees of freedom (which may be somewhat unreliable at small sample sizes). Table 1 A. Results for 1/8 mile runs. Table 2 B. Results for 1/2 mile runs.

TABLE 1 A.

Age Groupings	$A=\beta_0(V^{\beta_1}\times P^{\beta_2})$ $t=\ t=\ t=$ $R^2=$	$A=\beta_0(V^{\beta_1}\times E^{\beta_2})$ $t=\ t=\ t=$ $R^2=$	$A=\beta_0(V^{\beta_1}\times GE^{\beta_2})$ $t=\ t=\ t=$ R^2	$A=\beta_0(PM^{\beta_1}\times P^{\beta_2})$ $t=\ t=\ t=$ R^2	$A=\beta_0(PM^{\beta_1}\times E^{\beta_2})$ $t=\ t=\ t=$ R^2	$A=\beta_0(PM^{\beta_1}\times GE^{\beta_2})$ $t=\ t=\ t=$ R^2
2,3,4,5, 6,7,8,9	$=.261(V^{-.09}\times P^{.513})$ $-.45\ -.29\ 1.58$ $R^2=.355$ $F(2,5)=1.37$	$=.003(V^{-.1}\times E^{.75})$ $-1.70\ .36\ 1.82$ $.392$ $F(2,5)=1.61$	$=.812(V^{-.17}\times GE^{.93})$ $-.069\ -.53\ 1.81$ $.415$ $F(2,5)=1.78$			
2, 3-4, 5-8, 9	$=.054(V^{.07}\times P^{.99})$ $-2.19\ .50\ 3.38^*$ $R^2=.772^*$ $F(2,5)=8.473$	$=.001(V^{.25}\times E^{.71})$ $-5.16^{**}\ 2.29\ 4.05^*$ $.822^*$ $F(2,5)=11.515$	$=.164(V^{-.55}\times GE^{.93})$ $-1.77\ -.05\ 5.13^{**}$ $.882^{**}$ $F(2,5)=18.736$	$=.009(PM^{.32}\times P^{.99})$ $-2.37\ 1.23\ 3.38^*$ $.816^*$ $F(2,5)=11.106$	$=.003(PM^{.98}\times E^{.69})$ $-3.65^*\ 1.71\ 3.27^*$ $.771^*$ $F(2,5)=8.434$	$=.114(PM^{.04}\times GE^{.43})$ $-1.253\ 0.18\ 5.10^{**}$ $.883^{**}$ $F(2,5)=18.860^{**}$
0-4, 5-9	$=.166(V^{-.02}\times P^{.42})$ $-1.11\ -.13\ 2.41$ $R^2=.585$ $F(2,5)=3.52$	$=.006(V^{.13}\times E^{.55})$ $-2.56^*\ .85\ 2.12$ $.528$ $F(2,5)=2.798$	$=.406(V^{-.08}\times GE^{.36})$ $-.573\ -.492\ 2.83^*$ $.65$ $F(2,5)=4.71$			

* significant at $P<.05$
** significant at $P<.01$

TABLE 1 B.

2,3-4, 5-8, 9

$$A = 3.09 \times 10^{-6}\left(PM^{.83} \times P^{.38}\right)$$

$t = -1.82 \quad t = 1.42 \quad t = 2.58^{*}$

$R^2 = .830^{*}$

$F(2, 5) = 12.187$

$$A = 1.6 \times 10^{-6}\left(V^{.91} \times P^{.36}\right)$$

$t = -2.23 \quad t = 1.85 \quad t = 2.67^{*}$

$R^2 = .858^{**}$

$F(2,5) = 15.133$

Table 4. For Sector IV, Fraction of Habitatswith Snails before Next Treatment Cycle, a Measure of Success of Molluscicidingto be Used in Reducing the Amount of Habitat Affecting Transmission (Based on Project Reports from 1971 and 1972)

Habitat type	% with snails/% with snails pre-cycle 3	pre-cycle 1	% with snails/% with snails pre-cycle 7	pre-cycle 3
Pond	32/56	0.571	0/32	0.0
Dam	12/30	0.40	16.7/12	1.39
Furrow	100/100	1.00	0/100	0.0
Drinking Pond	33/50	0.66	0/33	0.0
Residual Pool	0/0	0.0	0/0	0.0
Other	0/0	0.0	0/0	0.0

Table 5. Migration Data Sector IV: 2-to-9-year olds, Misungwi, Tanzania 1968-1970

Age	Immigrants		Emigrants	
	No. infected	No. uninfected	No. infected	No uninfected
2	0	7	0	2
3	0	2	1	3
4	1	2	1	2
5	4	1	1	2
6	3	0	0	1
7	2	1	4	1
8	3	0	1	0
9	1	0	4	0
Total	14	13	12	10

Table 6. Model Analyses Tried with Tanzania Data, Sector IV, 2-to-9-year olds. An 'X' indicates that the corresponding modification was included in the model analysis.

Model analyses	Regression Equation (10)	Regression Equation (11)	Population increase 3/4% per yr.	Immigration and Emigration included	Controls included	Seasonal Variates
1	X		X			X
2		X	X			X
3	X		X	X		X
4		X	X	X		X
5	X		X		X	X
6		X	X		X	X
7	X		X	X		X
8		X	X	X		X
9	X		X	X	X	
10		X	X	X	X	

Table 7. 1970 Prevalence as Predicted by Different Runs of the Model: Number Infected, Total Eggs, Migration, Seasonal Variation, and Controls

Age	Observed	# Infected	Total Eggs	Differences
		Without Modifications		
2	.21	.23	.20	.03
3	.25	.45	.44	.01
4	.27	.51	.50	.01
5	.68	.64	.65	-.01
6	.44	.67	.69	-.02
7	.65	.67	.68	-.01
8	.74	.70	.71	-.01
9	.60	.81	.82	-.01
		With Migration		
2	.21	.21	.18	.03
3	.25	.43	.42	.01
4	.27	.51	.50	.01
5	.68	.67	.69	-.02
6	.44	.72	.73	-.01
7	.65	.65	.65	-
8	.74	.72	.73	-.01
9	.60	.79	.80	-.01
		With Migration & Seasonal Variation		
2	.21	.21	.19	.02
3	.25	.43	.42	.01
4	.27	.51	.50	.01
5	.68	.67	.69	-.02
6	.44	.72	.73	-.01
7	.65	.65	.65	-
8	.74	.72	.73	-.01
9	.60	.79	.80	-.01
		With Molluscicide		
2	.21	.16	.14	.02
3	.25	.37	.36	.01
4	.27	.42	.41	.01
5	.68	.54	.53	.01
6	.44	.59	.58	.01
7	.65	.61	.61	-
8	.74	.62	.62	-
9	.60	.76	.76	-
		With Molluscicide & Migration		
2	.21	.14	.12	.02
3	.25	.36	.34	.02
4	.27	.42	.41	.01
5	.68	.58	.58	-
6	.44	.64	.63	.01
7	.65	.58	.58	-
8	.74	.65	.64	.01
9	.60	.74	.73	.01

Table 8. 1972 Prevalence as Predicted by Different Runs of the Model: Number Infected, Total Eggs, Migration, Seasonal Variation, and Controls

Age	Observed*	# Infected	Total Eggs	Differences
		Without Modifications		
2	0.0	.36	.31	.05
3	0.0	.54	.53	.01
4	0.12	.61	.61	-
5	0.10	.75	.77	-.02
6	0.18	.75	.77	-.02
7	0.56	.72	.73	-.01
8	0.32	.77	.78	-.01
9	0.42	.83	.85	-.02
		With Migration		
2	0.0	.33	.28	.05
3	0.0	.52	.50	.02
4	0.12	.61	.61	-
5	0.10	.79	.81	-.02
6	0.18	.81	.83	-.02
7	0.56	.68	.68	-
8	0.32	.79	.81	-.02
9	0.42	.80	.81	-.01
		With Migration & Seasonal Variation		
2	0.0	.29	.24	.05
3	0.0	.49	.46	.03
4	0.12	.58	.58	-
5	0.10	.76	.78	-.02
6	0.18	.79	.80	-.01
7	0.56	.66	.66	-
8	0.32	.77	.78	-.01
9	0.42	.78	.79	-.01
		With Molluscicide		
2	0.0	.21	.17	.04
3	0.0	.39	.37	.02
4	0.12	.45	.43	.02
5	0.10	.58	.57	.01
6	0.18	.61	.60	.01
7	0.56	.61	.60	.01
8	0.32	.64	.63	.01
9	0.42	.75	.74	.01
		With Molluscicide & Migration		
22	0.0	.17	.14	.03
33	0.0	.36	.34	.02
44	0.12	.45	.43	.02
55	0.10	.64	.64	-
66	0.18	.69	.69	-
7	0.56	.57	.54	.03
8	0.32	.68	.67	.01
9	0.42	.70	.69	.01

*Refer to table 9, column B.

Table 9. Different Prevalence Values for 1972 Obtained Under Different Interpretations of Observed Data for Sector IV

Age	A Monitor group	B All households corrected for age (date of examination based age)	C Model households corrected for age	D Model households corrected for age (registration based age)
2	0.0	0.0	0.0	0.0
3	0.0	0.0	0.0	0.0
4	0.125	0.120	0.107	0.094
5	0.053	0.10	0.091	0.214
6	0.400	0.182	0.25	0.409
7	0.450	0.556	0.556	0.400
8	0.300	0.323	0.333	0.500
9	0.0	0.417	0.417	0.565

Table 10. 2004 (Asymptotic Year) Prevalence as Predicted by Different Runs of the Model: Number Infected, Total Eggs, Migration, Seasonal Variation, and Controls

Age	#infected	Total Eggs	Difference
	Without Modifications		
2	.80	.81	-.01
3	.80	.81	-.01
4	.84	.85	-.01
5	.88	.90	-.02
6	.87	.89	-.02
7	.83	.85	-.02
8	.87	.88	-.01
9	.87	.89	-.02
	With Migration		
2	.85	.87	-.02
3	.68	.67	-.01
4	.84	.87	-.03
5	.92	.94	-.02
6	.92	.93	-.01
7	.75	.76	-.01
8	.90	.91	-.01
9	.80	.81	-.01
	With Migration & Seasonal Variation		
2	.80	.82	-.02
3	.58	.54	.04
4	.80	.81	-.01
5	.90	.92	-.02
6	.90	.91	-.01
7	.69	.68	.01
8	.86	.88	-.02
9	.75	.75	-
	With Molluscicidding		
2	.56	.48	.08
3	.55	.49	.06
4	.63	.59	.04
5	.73	.73	-
6	.72	.71	.01
7	.63	.60	.03
8	.70	.69	.01
9	.72	.71	.01
	With Molluscicidding & Migration		
2	.63	.60	.03
3	.24	.02	.22
4	.63	.60	.03
5	.82	.83	-.01
6	.85	.86	-.01
7	.43	.33	.10
8	.77	.78	-.01
9	.53	.45	.08

Table 11. 1968 Baseline data on number infected in each age group and that number in each age group for Sector IV, Misungwi, Tanzania

Age	1968 Baseline Data	
	No. infected in age group	No. in age group
2	7	18
3	8	24
4	11	29
5	15	32
6	16	29
7	15	25
8	15	25
9	20	26
Total	107	208

Table 12. Percentage of Habitats Where Snails were Found in Sector V (No Controls Used).

Habitat type	% with snails/% with snails pre-cycle 3	pre-cycle 1	% with snails/% with snail pre-cycle 7	pre-cycle 1
Pond	39/56	0.696	41.7/39	1.069
Lawn	0/43	0.0	28.5/43	0.663
Furrow	14/71	0.197	50/71	0.704
Drinking Pond	32/34	0.941	46.8/34	1.376
Residual Pond	70/62	1.129	40/62	0.645
Other	0/1	0.0	0/1	0.1

Table 13. Case-years of Infection Prevented as Estimated by Model Runs Over Years of Project and One Additional Cycle (1968-1974). Number Infected and Total Eggs were run as Separate Examples for 2-through-9-Year-Olds in Sector IV

Years	Estimated case-years of infection (no control)	Case-years of infection prevented		
		Molluscicide	Chemotherapy	Molluscicide plus chemotherapy
Number infected				
1968 to 1974	552	86	162	232
Total eggs				
1968 to 1974	551	95	153	250

Table 14. Predicted Case-years of Infection Prevented Over Years of Project Plus One Additional Cycle: 1968-1974. Results given for use of Molluscicides, Chemotherapy, Molluscicides Plus Chemotherapy. Results are given for Separate use of Number Infected and Total Eggs

	Number infected			Total eggs		
	1	2	3	1	2	3
Year	Estimated case-years no control	Estimated case-years with molluscicide	Estimated case-years of infection prevented	Estimated case-years no controls	Estimated case-years with molluscicide	Estimated case-years of infection prevented
1968	101	101	0	101	101	0
1970	129	113	16	128	100	18
1972	152	122	30	151	118	33
1974	170	130	40	171	127	44
Total	552	466	86	551	456	95
	with chemotherapy			with chemotherapy		
1968	101	101	0	101	101	0
1970	129	77	52	128	78	50
1972	152	87	65	151	85	56
1974	170	125	45	171	124	47
	552	390	162	551	388	153
	with chemotherapy & molluscicide			with chemotherapy & molluscicide		
1968	101	101	0	101	101	0
1970	129	67	62	128	67	61
1972	152	63	79	151	59	92
1974	170	79	91	171	74	97
	552	411	232	551	301	250

Table 15. Case-years of Infection Prevented as Estimated by Model for 10 Cycles - Twenty Years After Start of Control Activities (1968-1988). Number Infected and Total Eggs Run as Reported Examples, for 2-through-9-Year-Olds, Based on Sector IV Data

Years	Estimated Case Years of Infection (no control)	Case-years of infection prevented		
		Molluscicide	Chemotherapy	Molluscicide plus chemotherapy
Number infected				
1968 to 1978	2115	465	242	793
Total eggs				
1968 to 1978	2132	542	240	931

Table 16. Estimated Costs per Case-year of Infection Prevented for Three Different Control Strategies Over Years of Analysis: 1968-1974 (U. S. Dollars)

Years	Molluscicide	Chemotherapy	Molluscicide plus chemotherapy
1968 to 1974			
# infected	$ 60.73	9.05	28.83
1968 to 1974			
Total Eggs	$ 54.98	9.58	26.76

Table 17. High Priority Data Items Required for Pre-Project Predictions of Water Resources Project Impacts on Schistosomiasis Transmission

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Epidemiological

- Prevalence (number or fraction infected/age/sex) in human population
- Incidence (rate of change in infection/age/sex) in human population

Environmental

- Snail species
- Age/sex specific human contact by snail habitats (pre-project)
- Post-project location of human settlements
- Post-project changes in size of high frequency contact habitats: length, width, depth for irrigation and small ponds or shoreline for larger water bodies

Demographic

- Total population in area
- Total population expected to be attracted to area or migration predictions (based on regional information)

Economic

- Control measure costs: equipment, material, personnel, transport, facilities

FIGURES

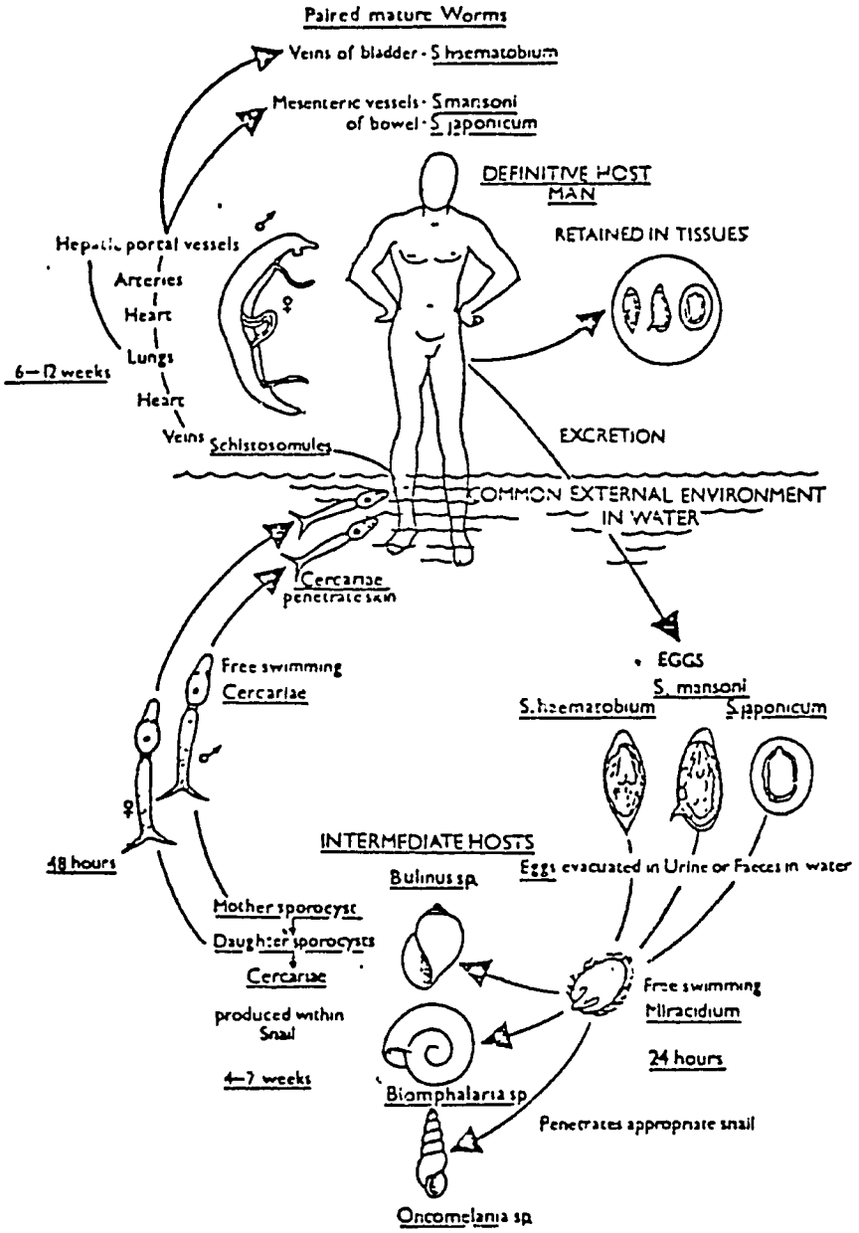


Figure 1. The life cycle of schistosomiasis. Source: P. Jordan and G. Webbe, Human Schistosomiasis, Springfield, Ill., Charles C. Thomas Publishers (1969), p. 7.

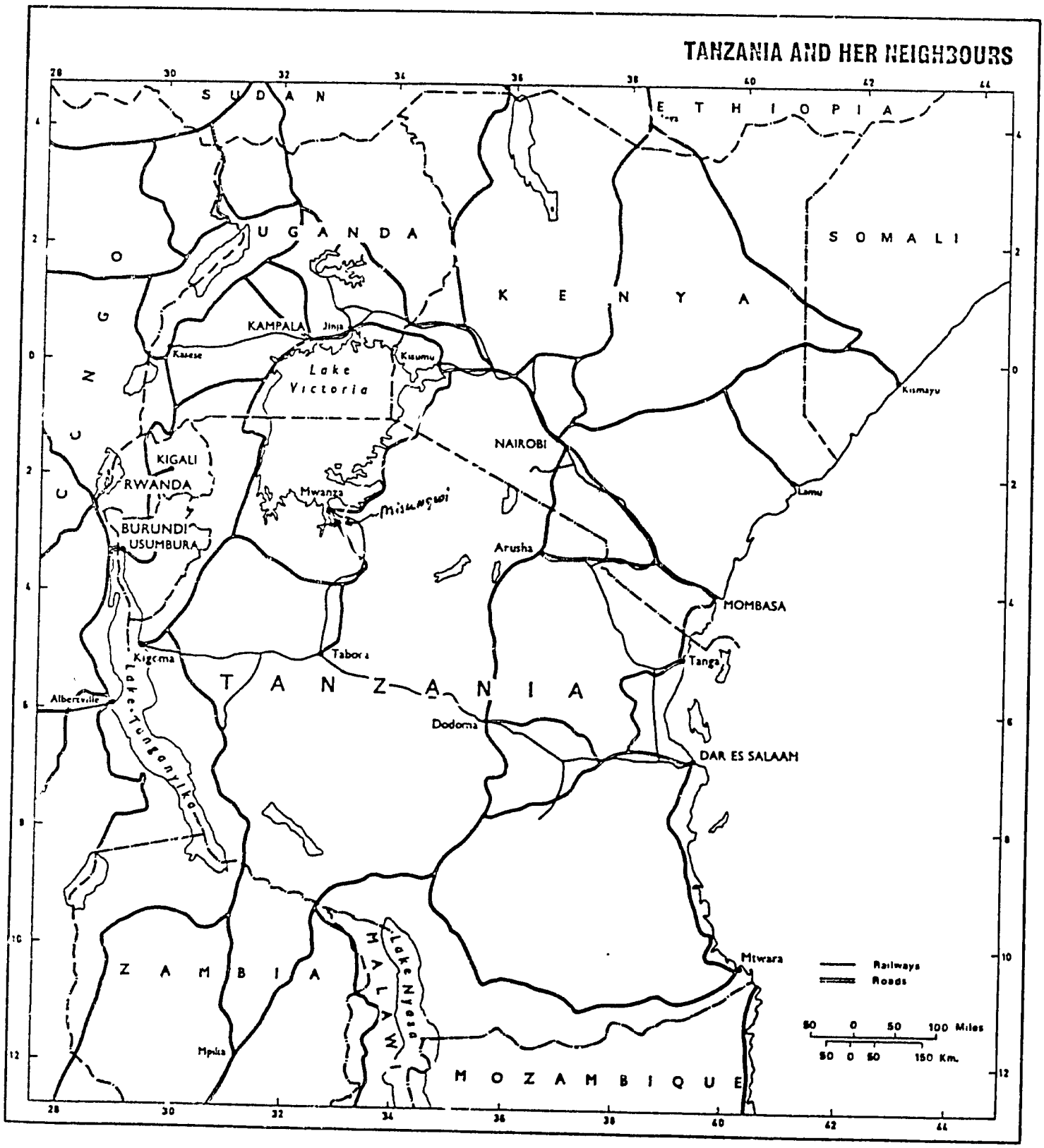


Figure 2. Map of Tanzania. From Berry, L., ed., 1971. Tanzania in Maps (London, University of London Press), p. 13.

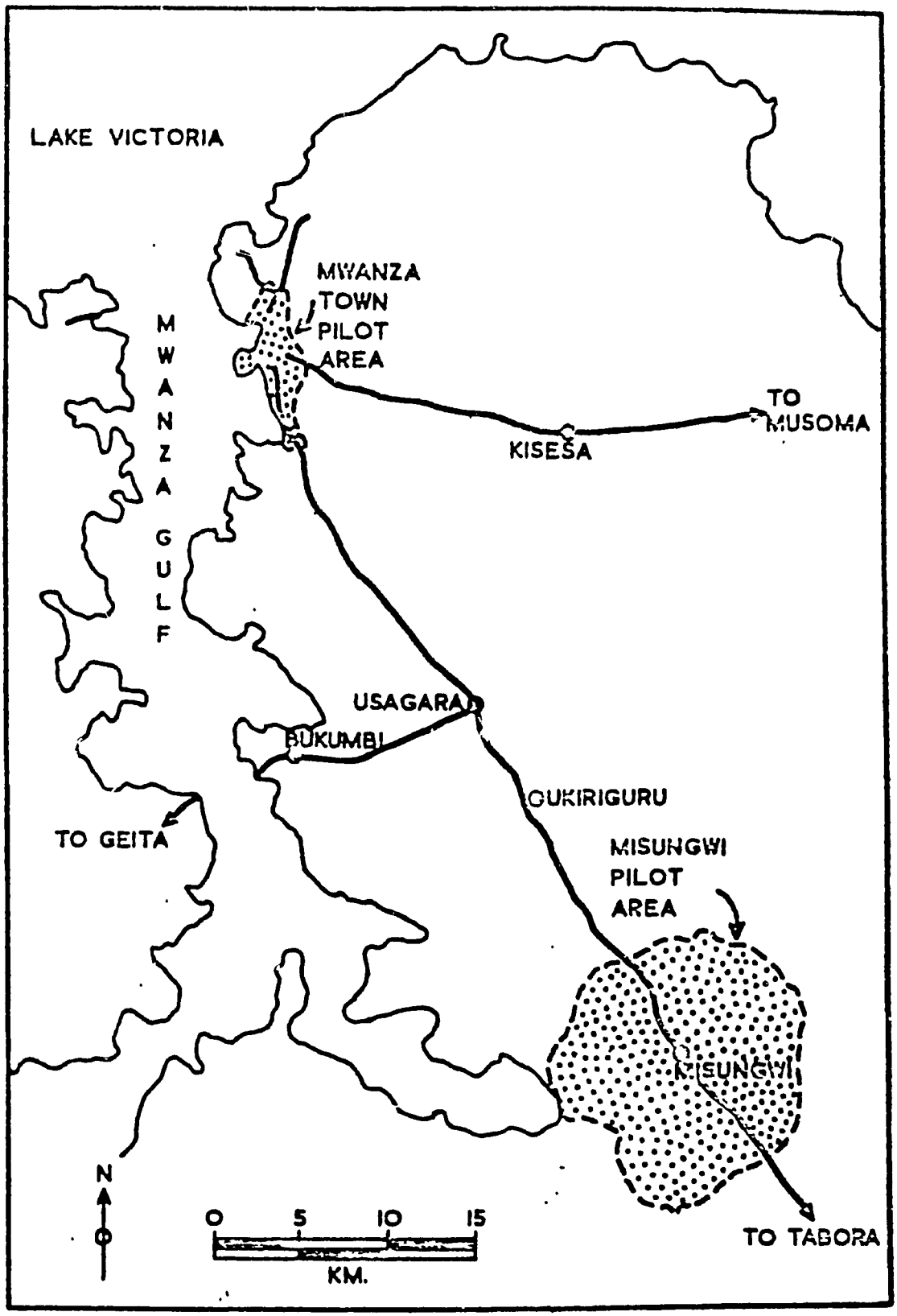


Figure 3, The location of the Misungwi and Mwanza pilot areas, (Source: McCullough, F. and V, Eyakuze, 1973. WHO/Tanzania Schistosomiasis Pilot Control and Training Project, Mwanza District, Tanzania, Final Report, AFR/SCHIST/29, WHO, Geneva).

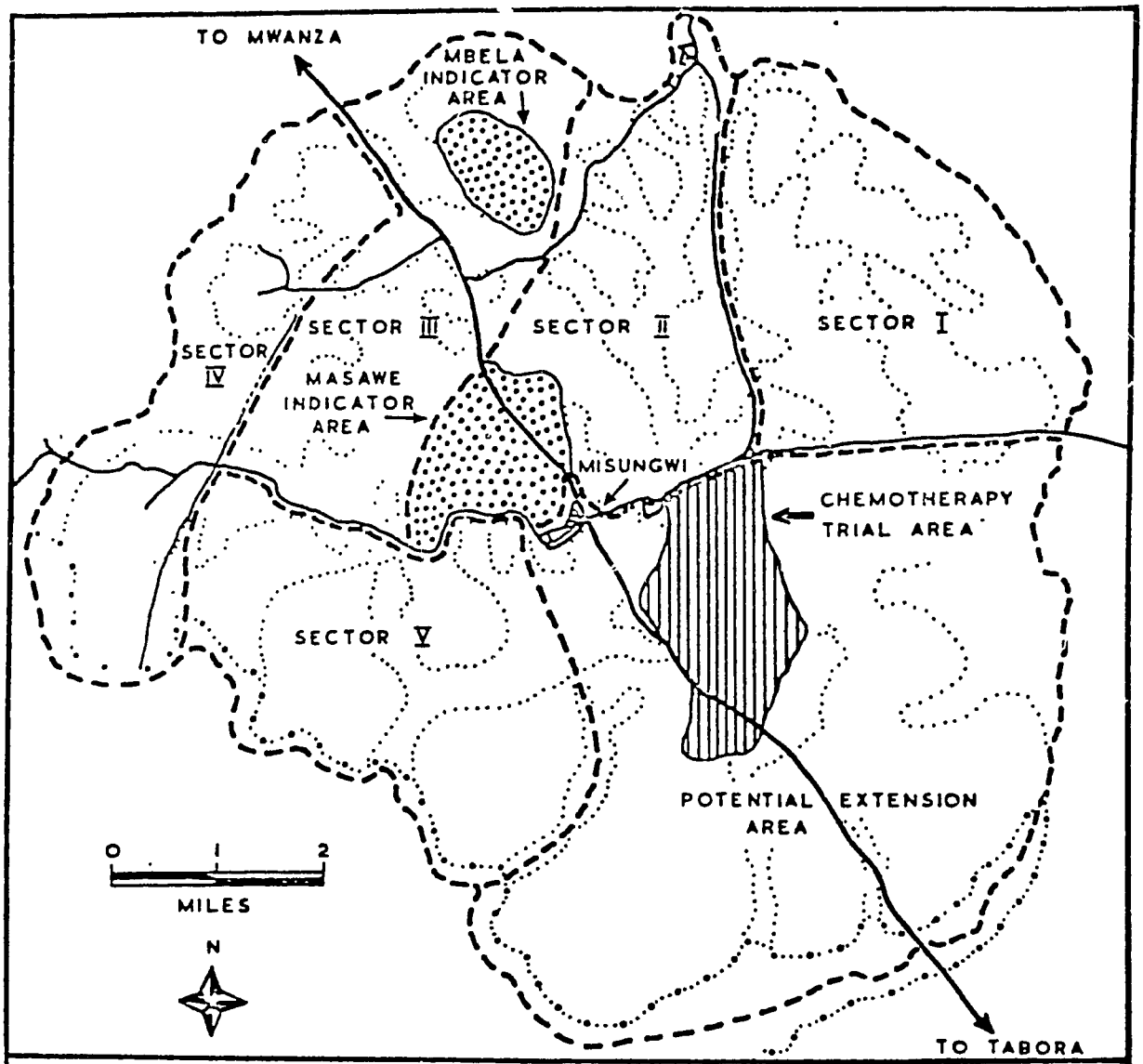


Figure 4. Misungwi pilot area showing Sectors, Mbela and Masawe Indicator Areas and The Mitindo/Mwamanga chemotherapy trial area.

From: McCullough,F. and V. Eyakuze, 1973. WHO/Tanzania Schistosomiasis Pilot Control and Training Project, Mwanza District, Tanzania, Final Report, AFR/SCHIST/29,WHO, Geneva.

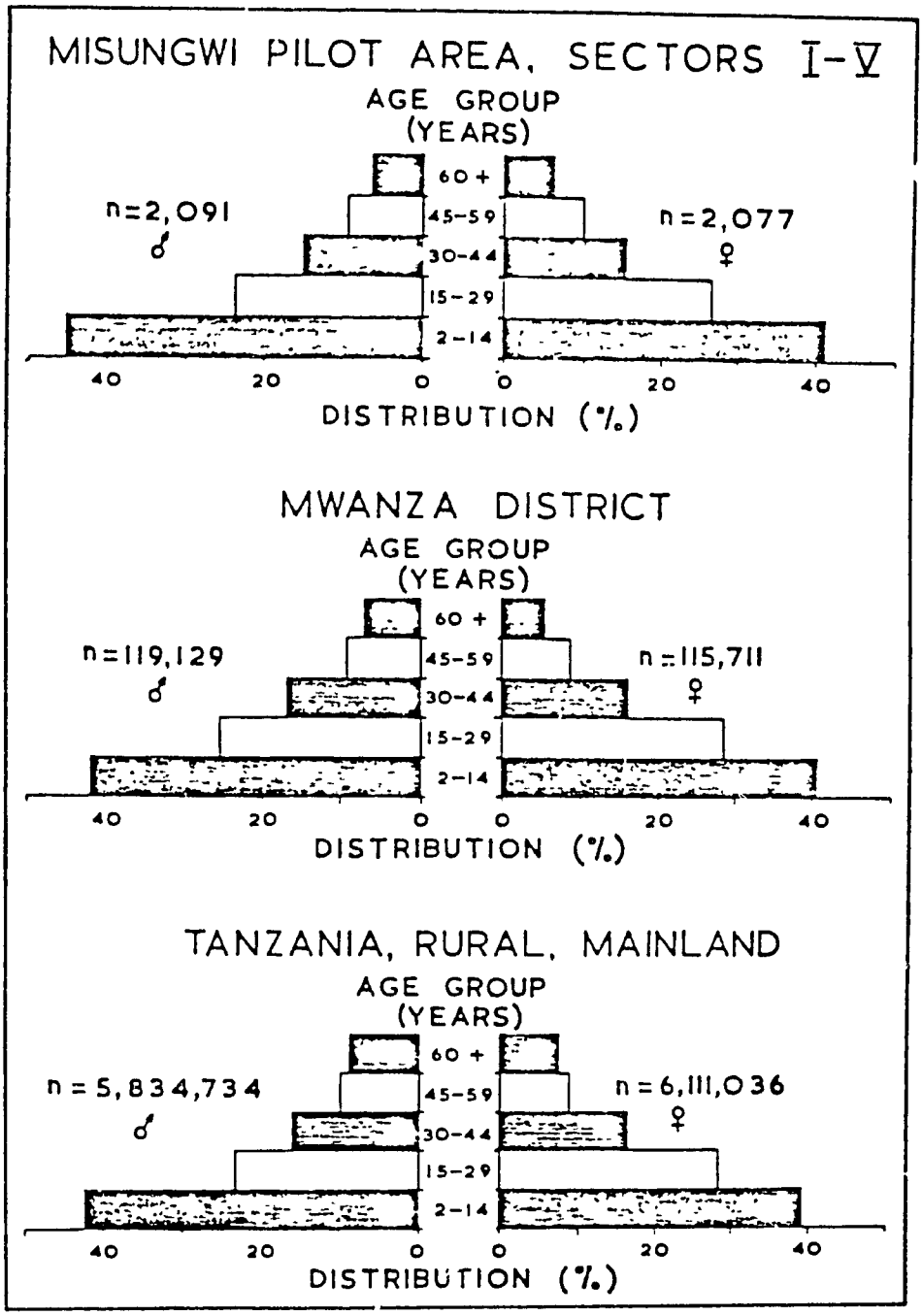


Figure 5A. Comparison of the age/sex structure of the populations in the Misungwi pilot area, Mwanza District and mainland Tanzania. From: McCullough, F. and V. Eyakuze, 1973. WHO/Tanzania Schistosomiasis Pilot Control and Training Project, Mwanza District, Tanzania, Final Report, AFR/SCHIST/29, WHO, Geneva.

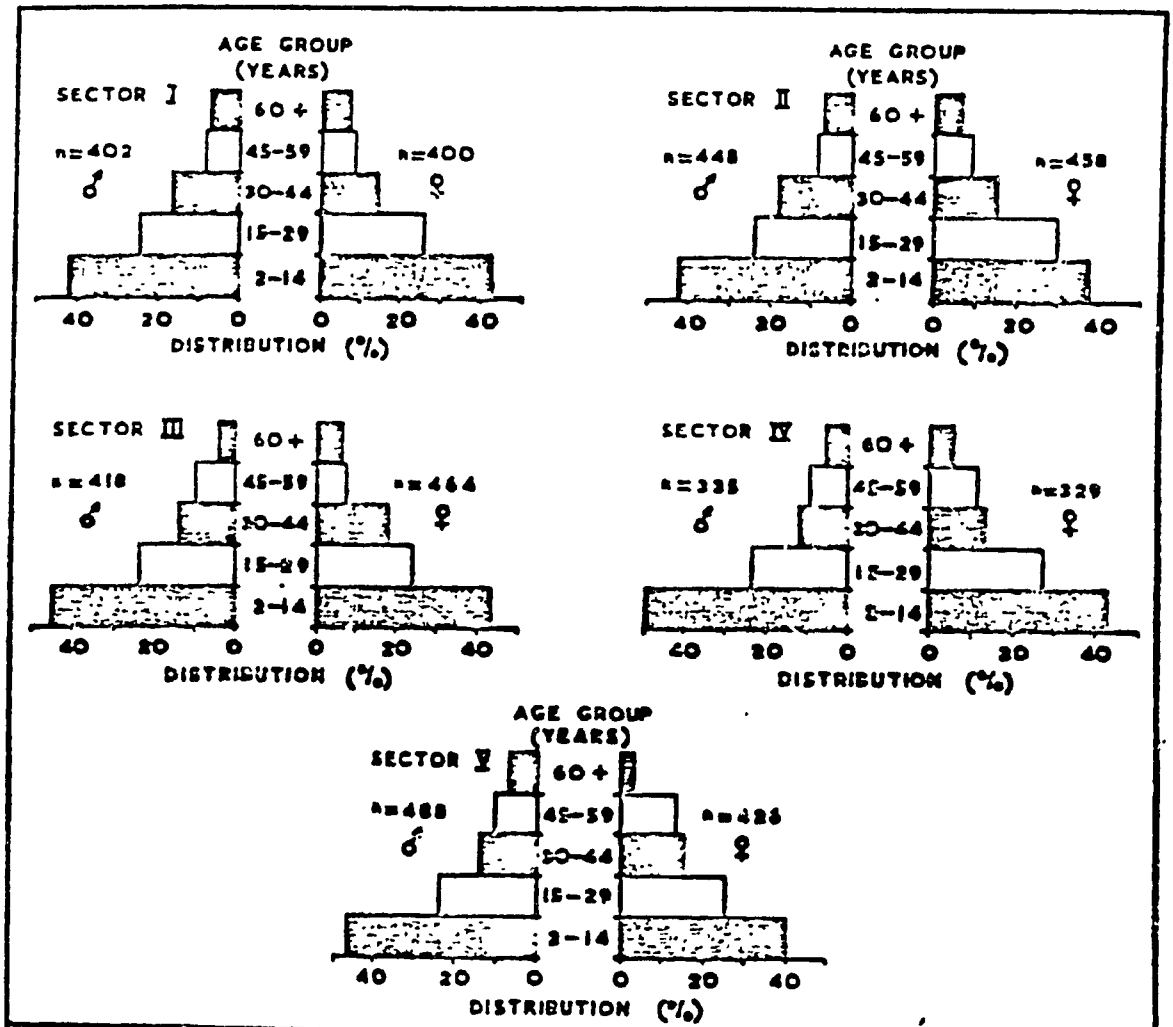


Figure 5B. **POPULATION STRUCTURE BY AGE, SEX AND SECTOR IN THE MISUNGWI PILOT AREA.**

From: McCullough, F. and V. Eyakuze, 1973. WHO/Tanzania Schistosomiasis Pilot Control and Training Project, Mwanza District, Tanzania, Final Report, AFR/SCHIST/29, WHO, Geneva.

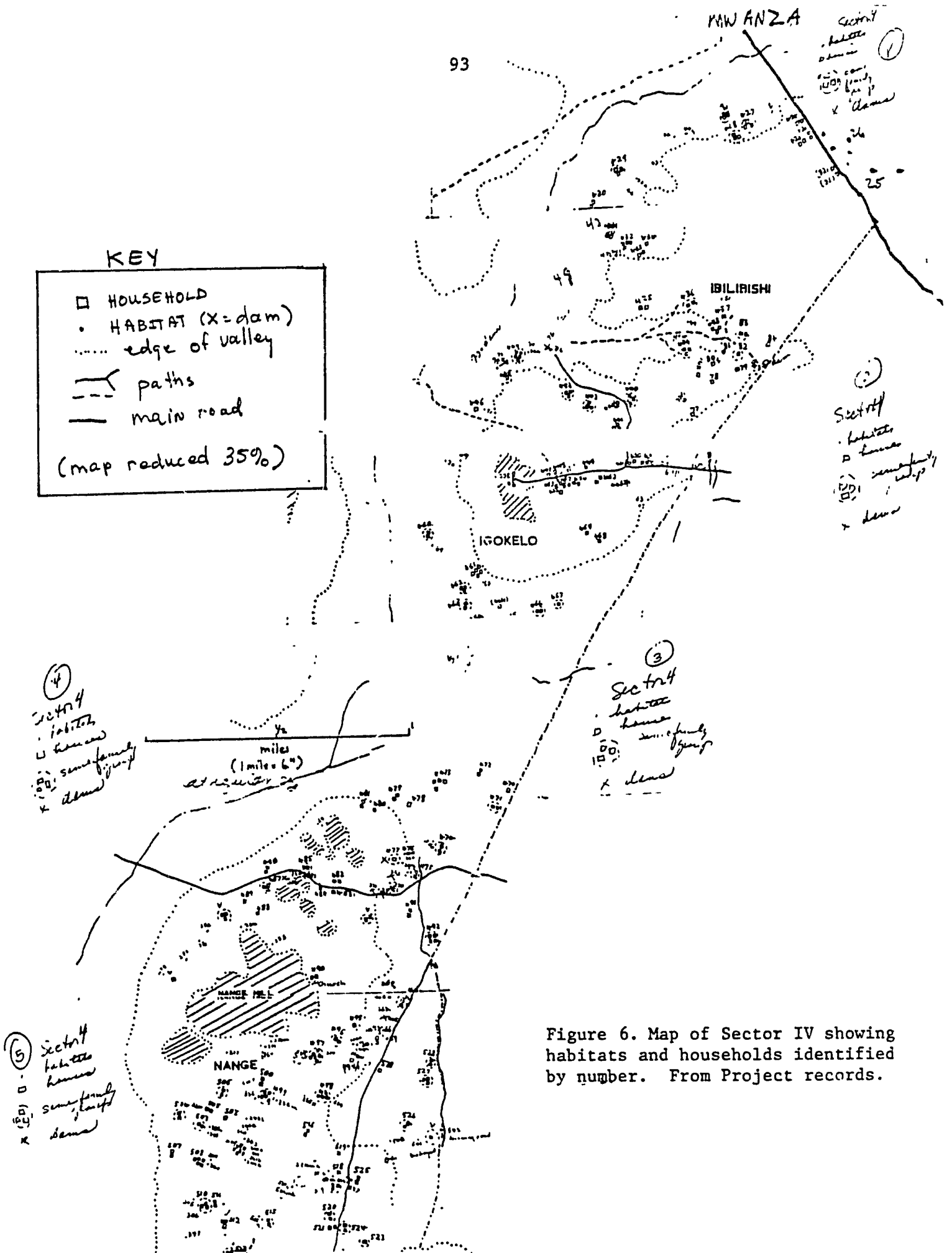


Figure 6. Map of Sector IV showing habitats and households identified by number. From Project records.

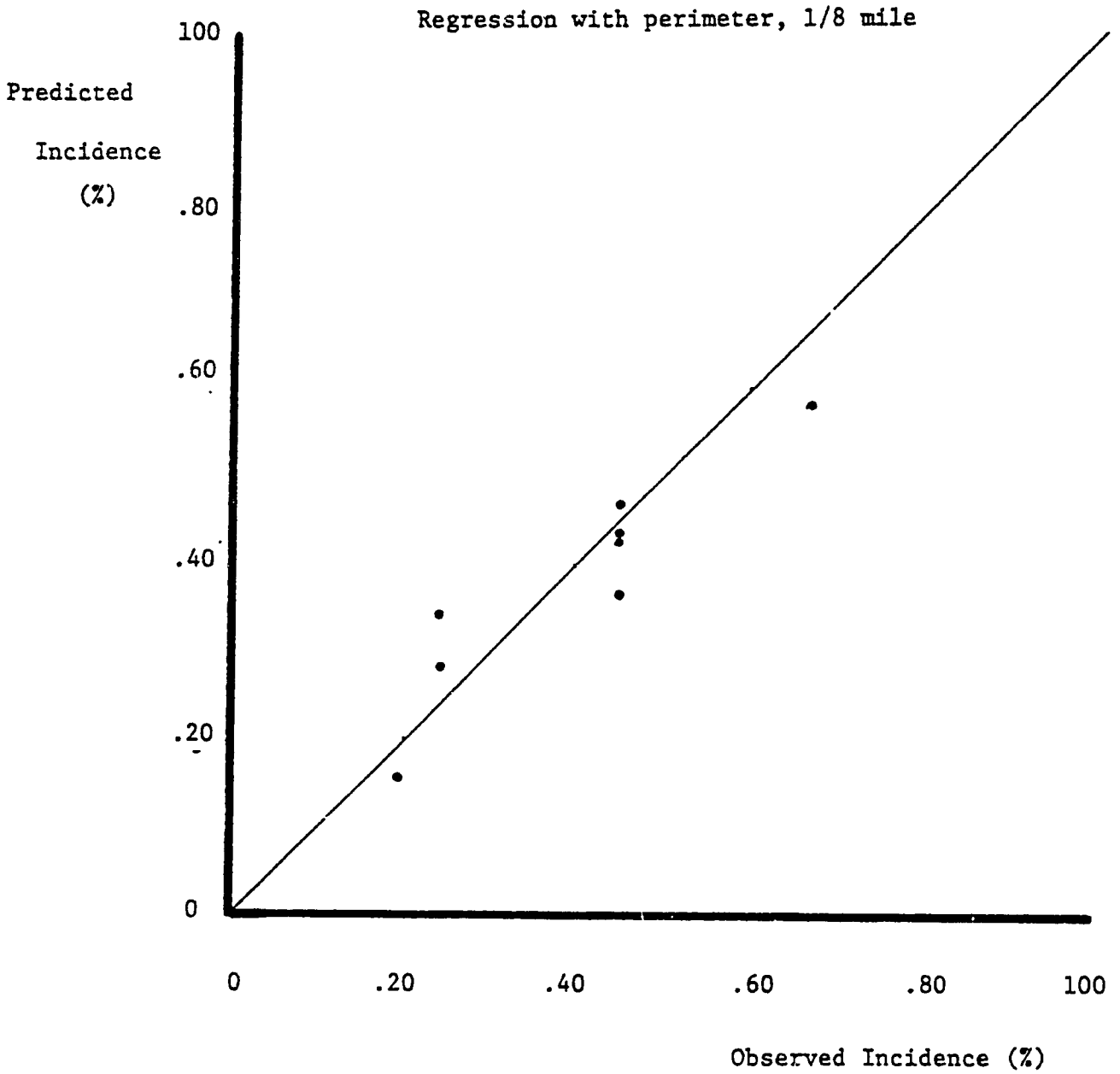


Figure 7. Results of regression analyses for perimeter and volume under 1/8 and 1/2 mile accessibility assumptions. On the graph. predicted incidence values are plotted against observed values to show the scatter from the 45 line (indication of closeness of fit of prediction: the less scatter from the line, the closer the fit).

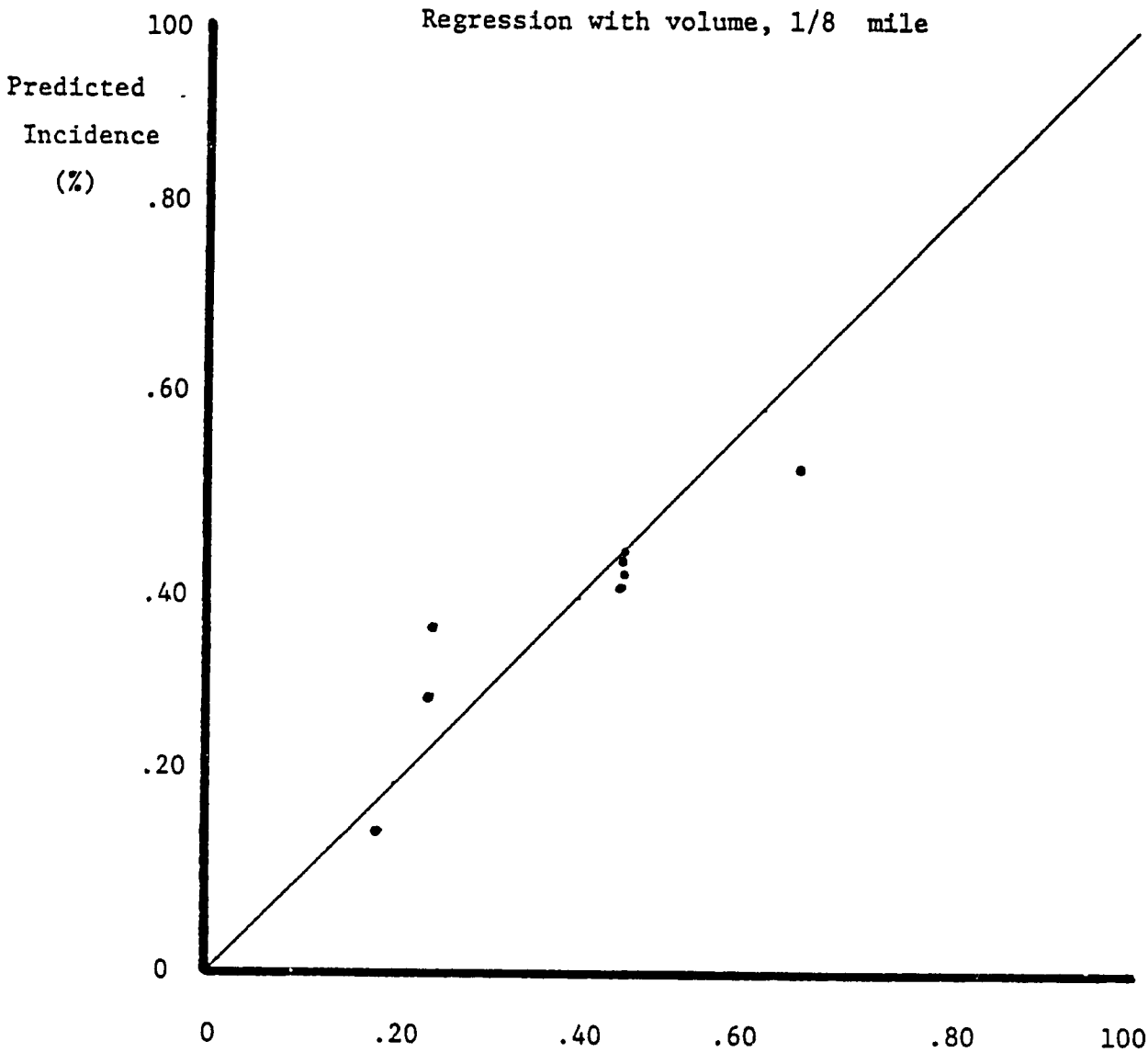
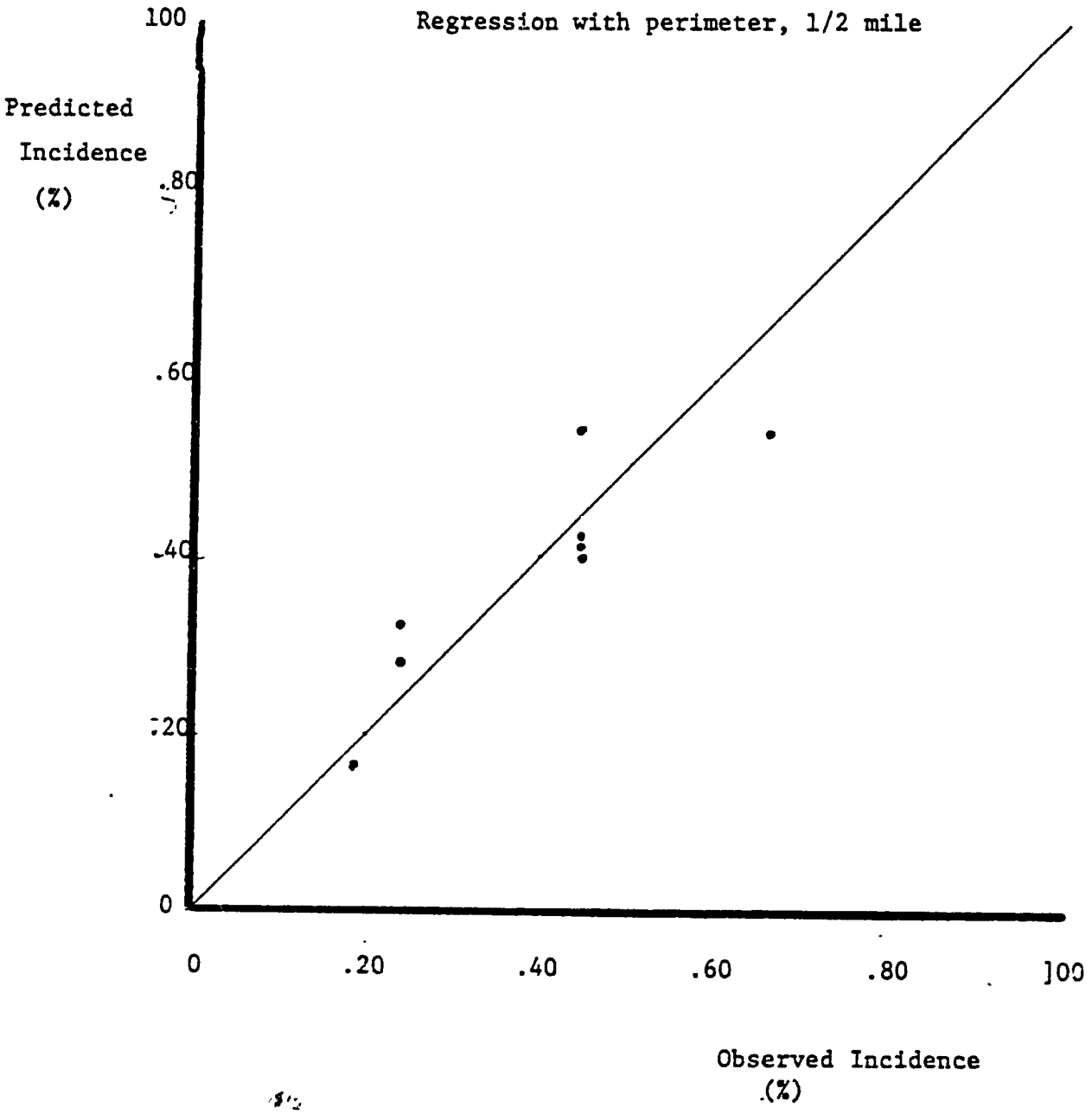


Figure 7 . cont.



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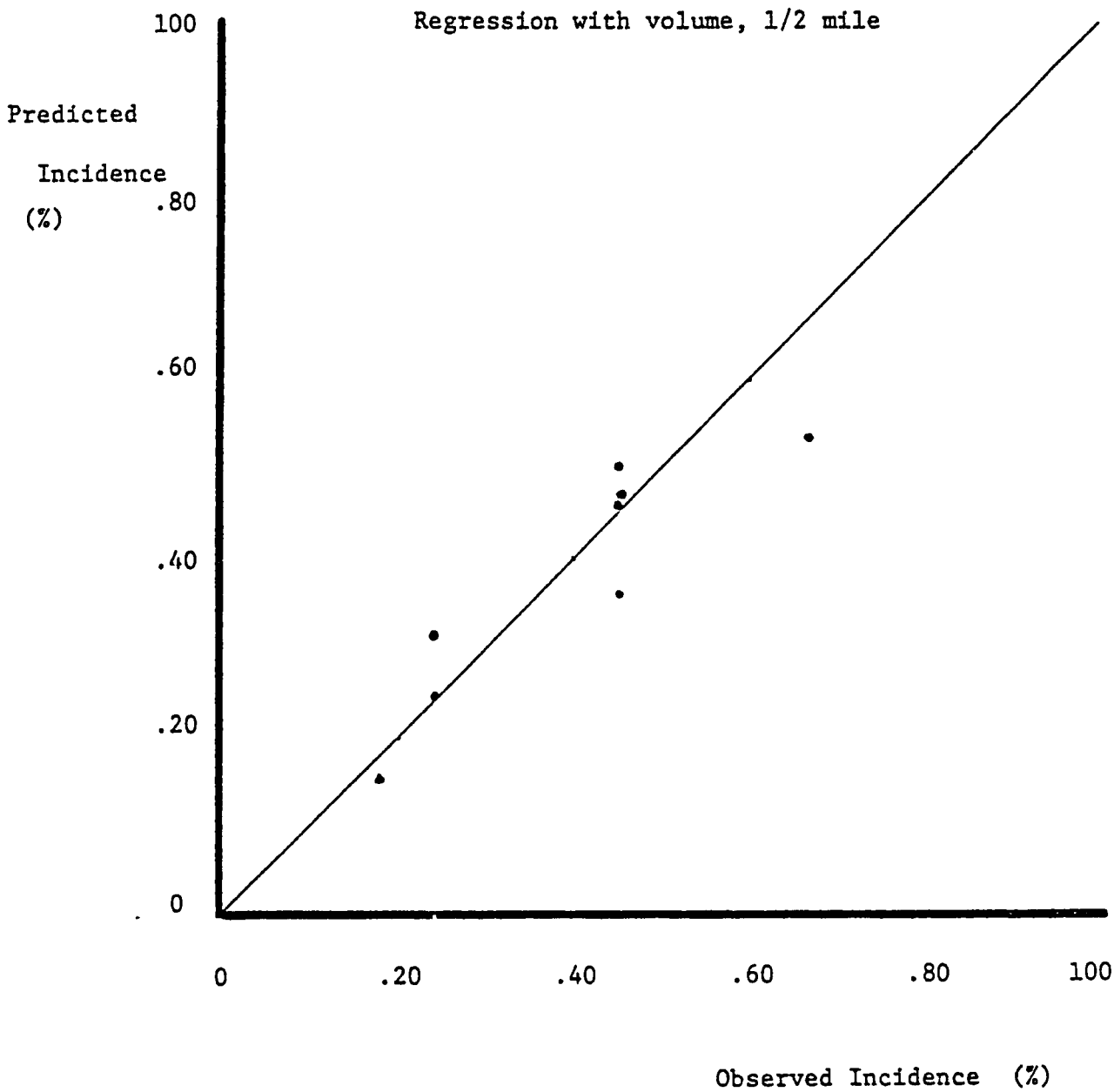

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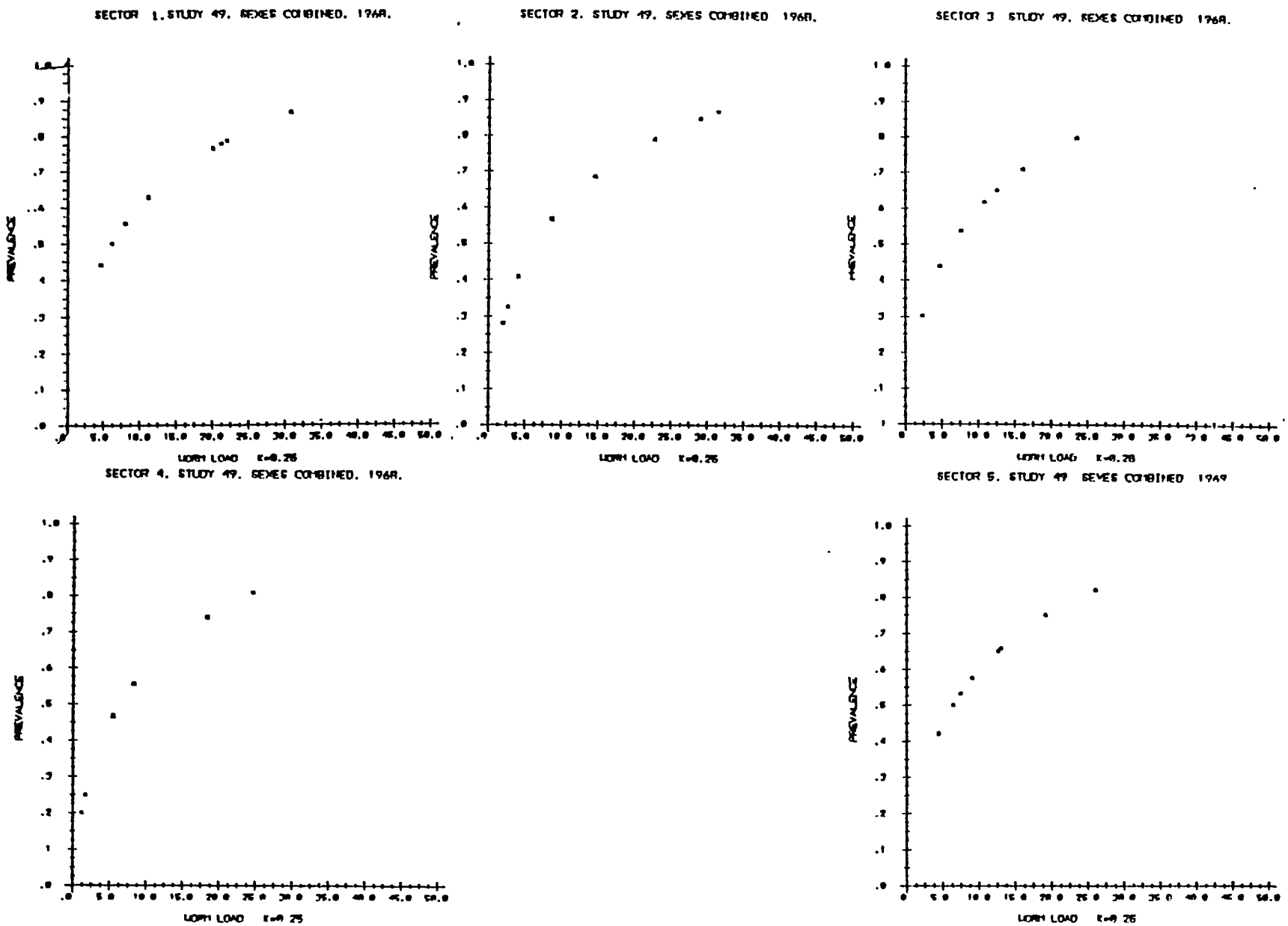


Figure 8. These plots show the results of calculating mean worm load from prevalence values under three different statistical distributions; $k \to \infty$ (Poisson); $k = 1$; $k = 0.25$ (approaching the negative binomial) for the Misungwi, Tanzania data (2-to-9-year-olds). The results show similar tendencies over the sector for each distribution, but it is not immediately apparent which distribution is the most appropriate one.

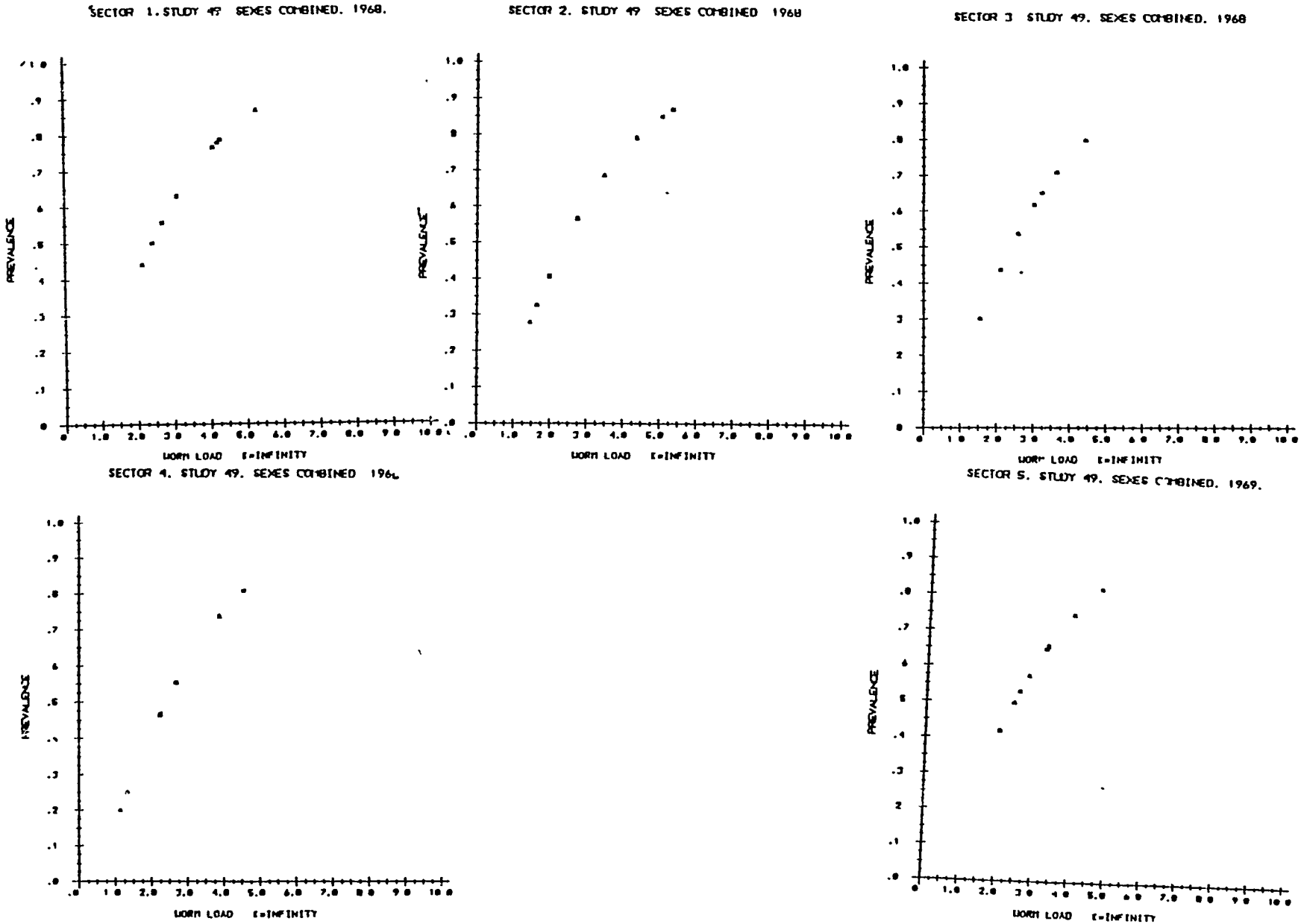
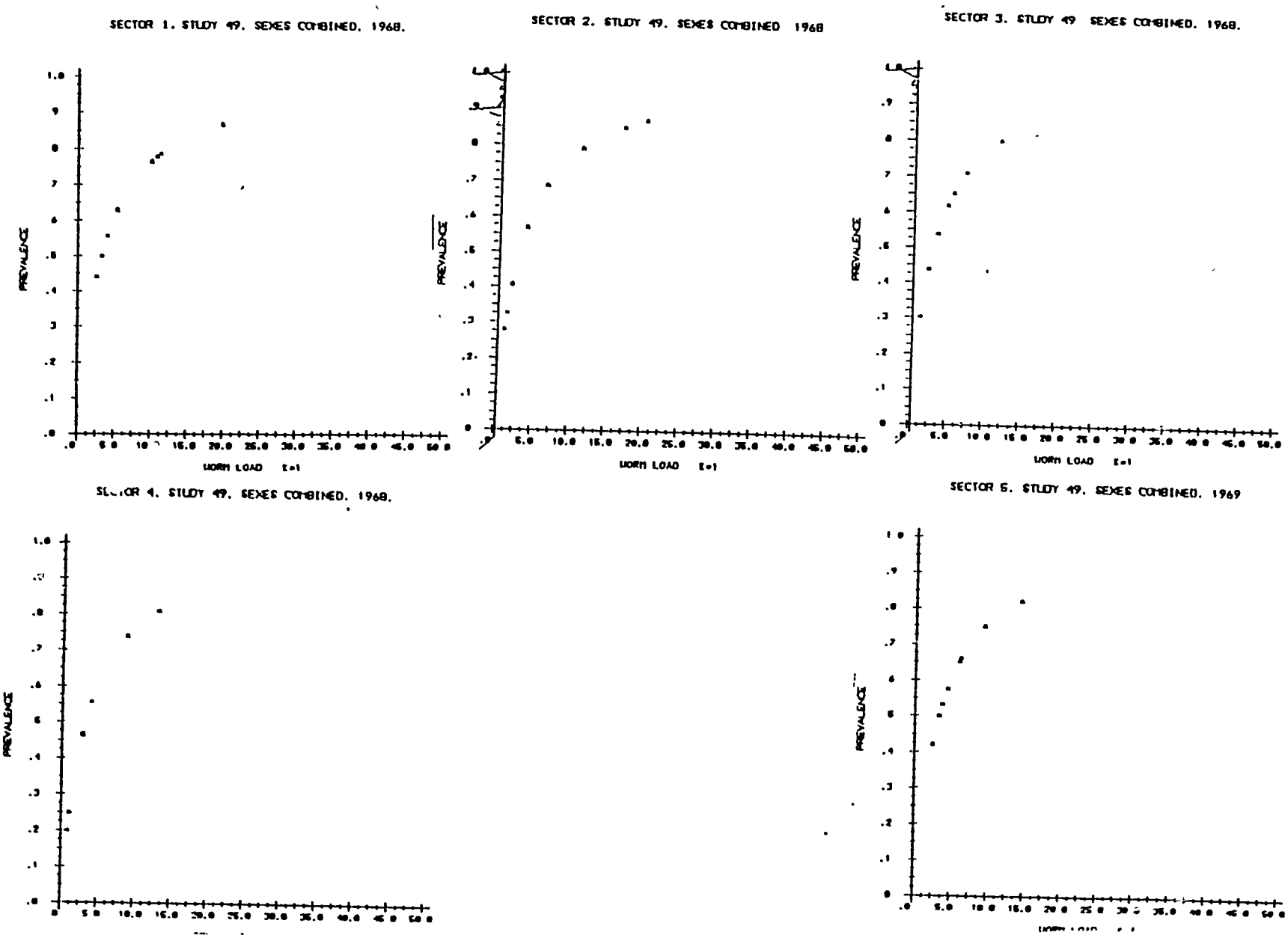


Figure 8 (continued)



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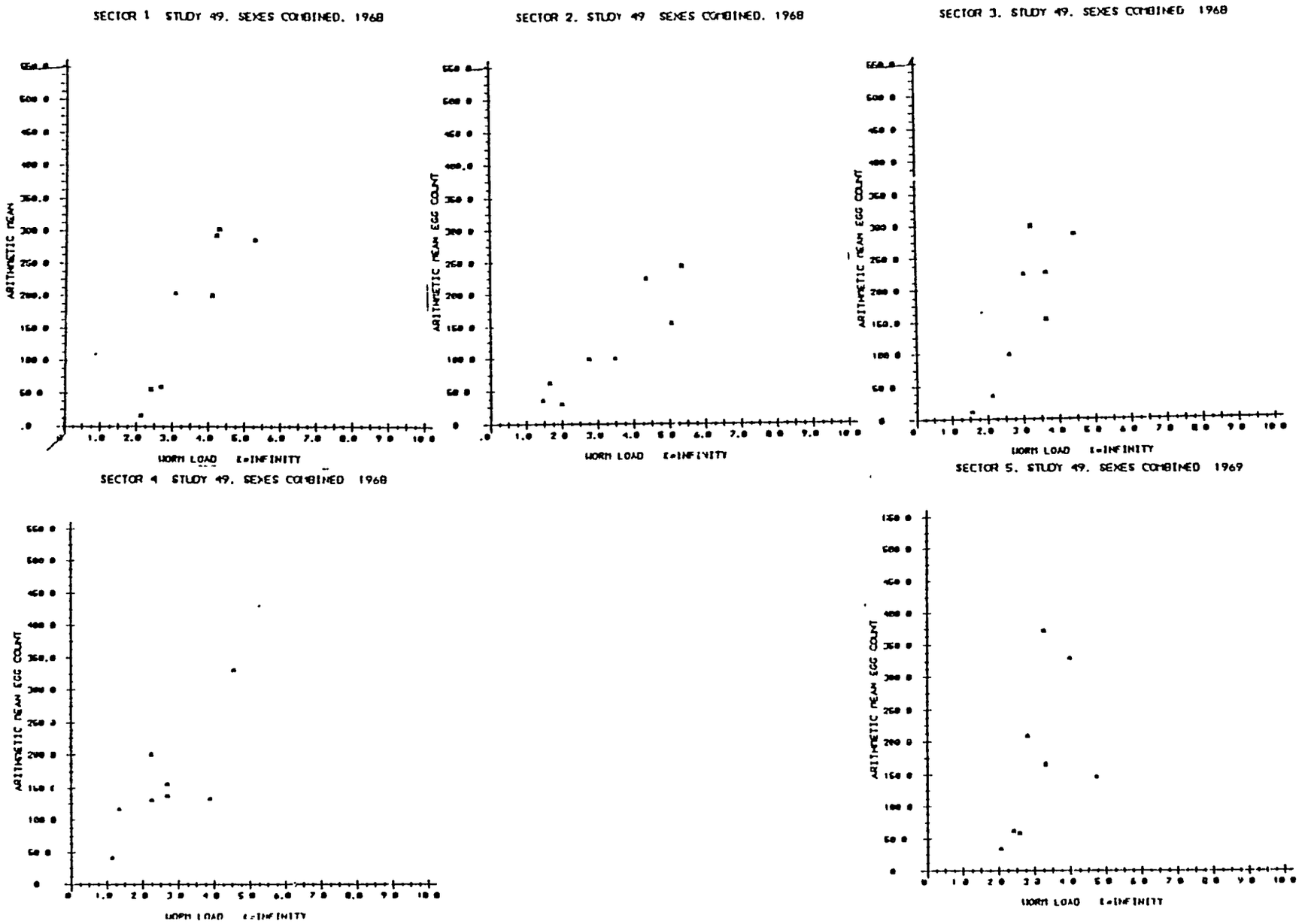
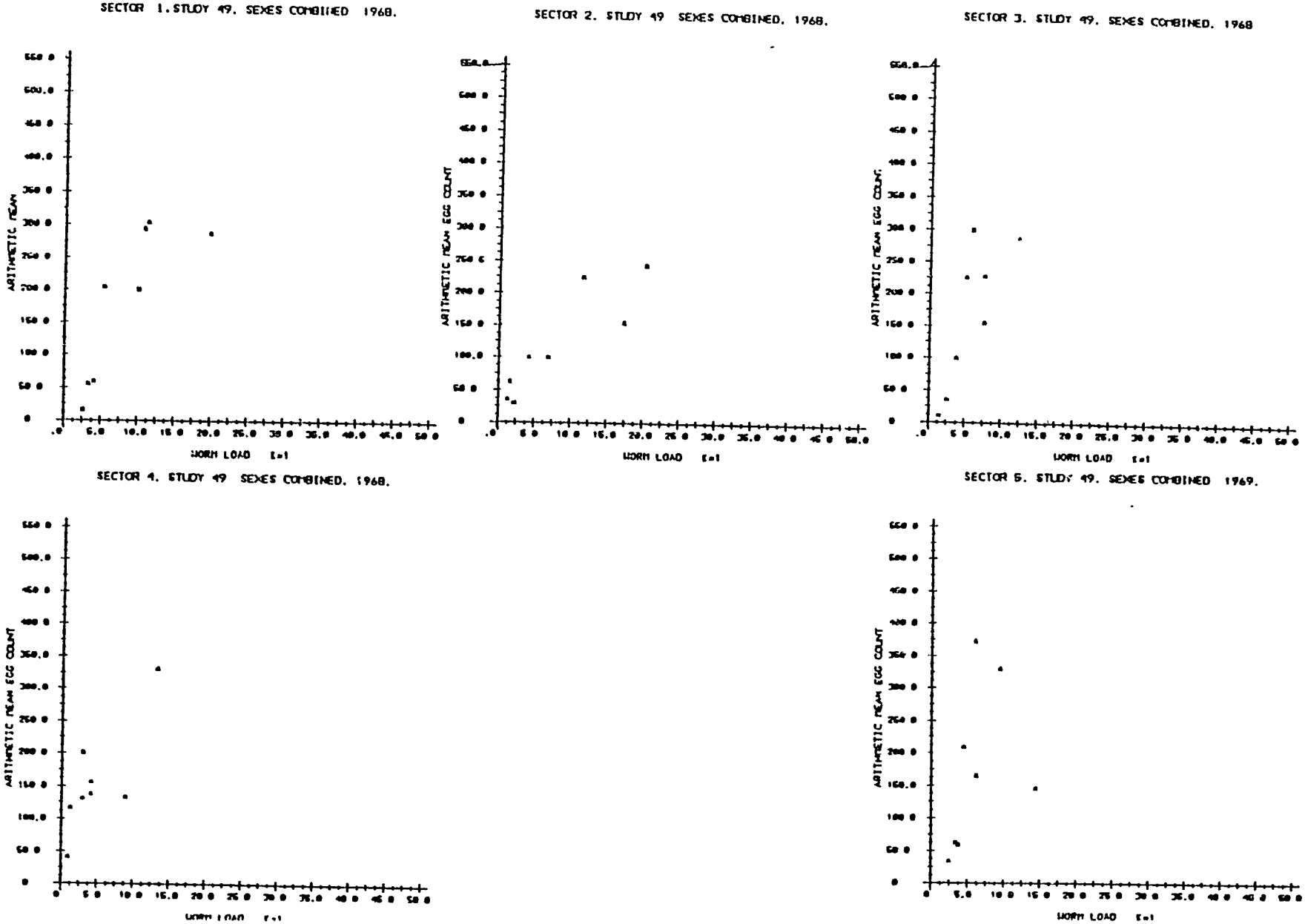


Figure 9. These plots demonstrate the relationship between geometric mean egg count (transformed to account for the large number of zero values) or arithmetic mean eggs and mean worm load for the different sectors (2-to-9-year-olds) and under different statistical distributions for the Misungwi, Tanzania data: $k \to \infty$ (Poisson); h = 1; k = 0.25 (approaching negative binomial). The results for both sets of graphs are not fitted by a line but show similar tendencies over the sectors for each distribution. Where $k \to \infty$, the line of best fit would pass through the x-axis, indicating that there may be some worms in humans before eggs are passed. This result is of interest since the worm load was estimated by prevalence, a measure determined by the presence or absence of eggs. Where k=1 or k = 0.25, the line of best fit would pass through or close to the origin, indicating that at 0 worms there are 0 eggs passed. It is again not apparent which distribution measure of egg counts to use in the



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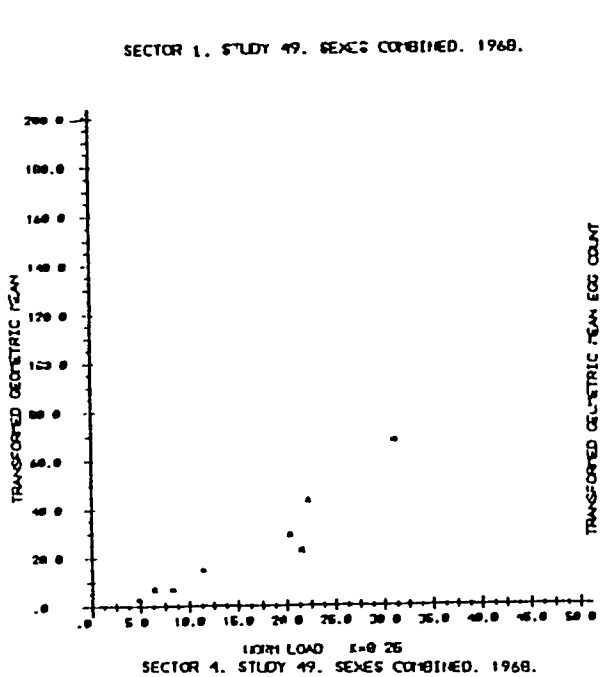

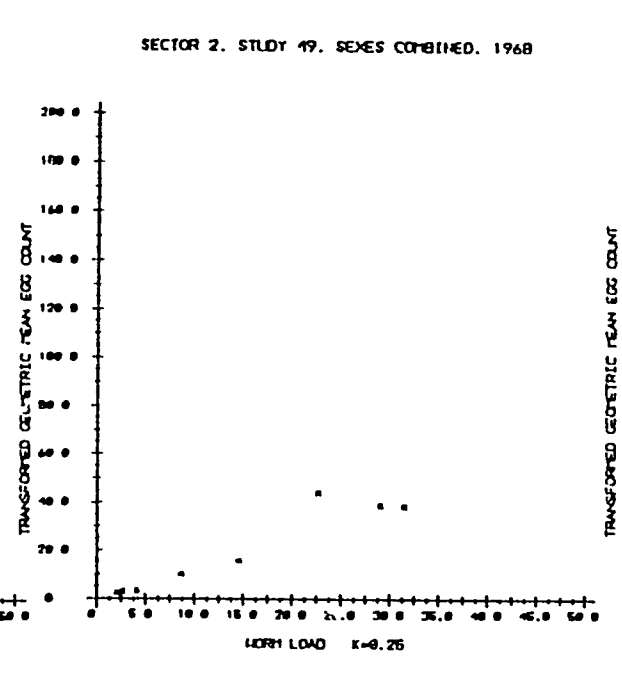

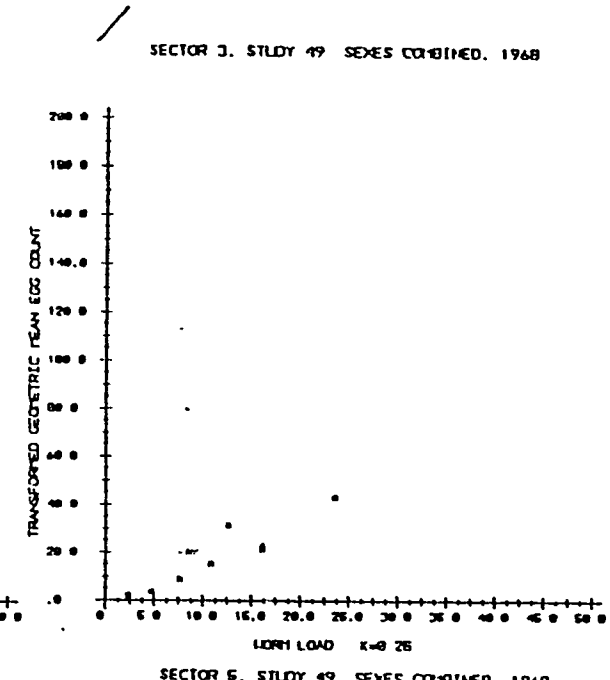

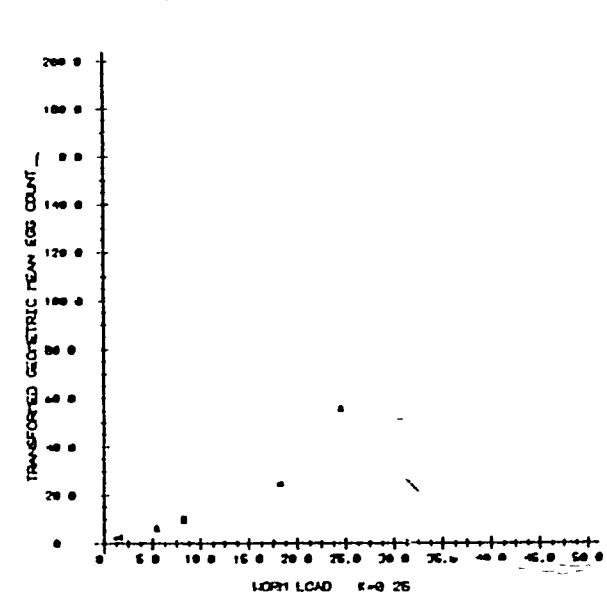

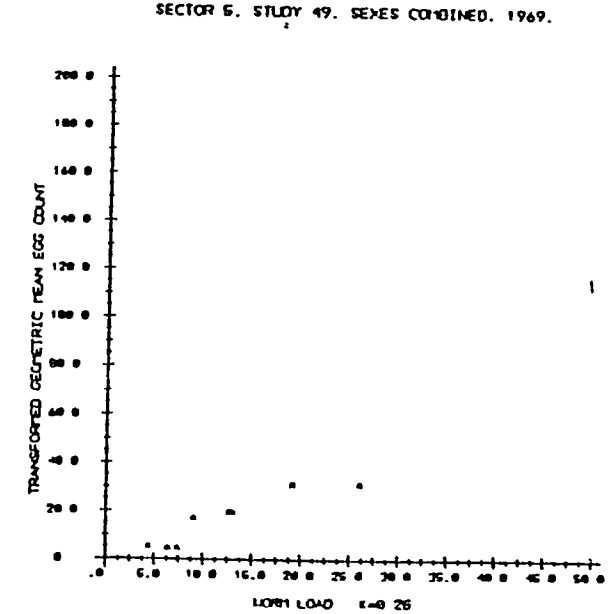

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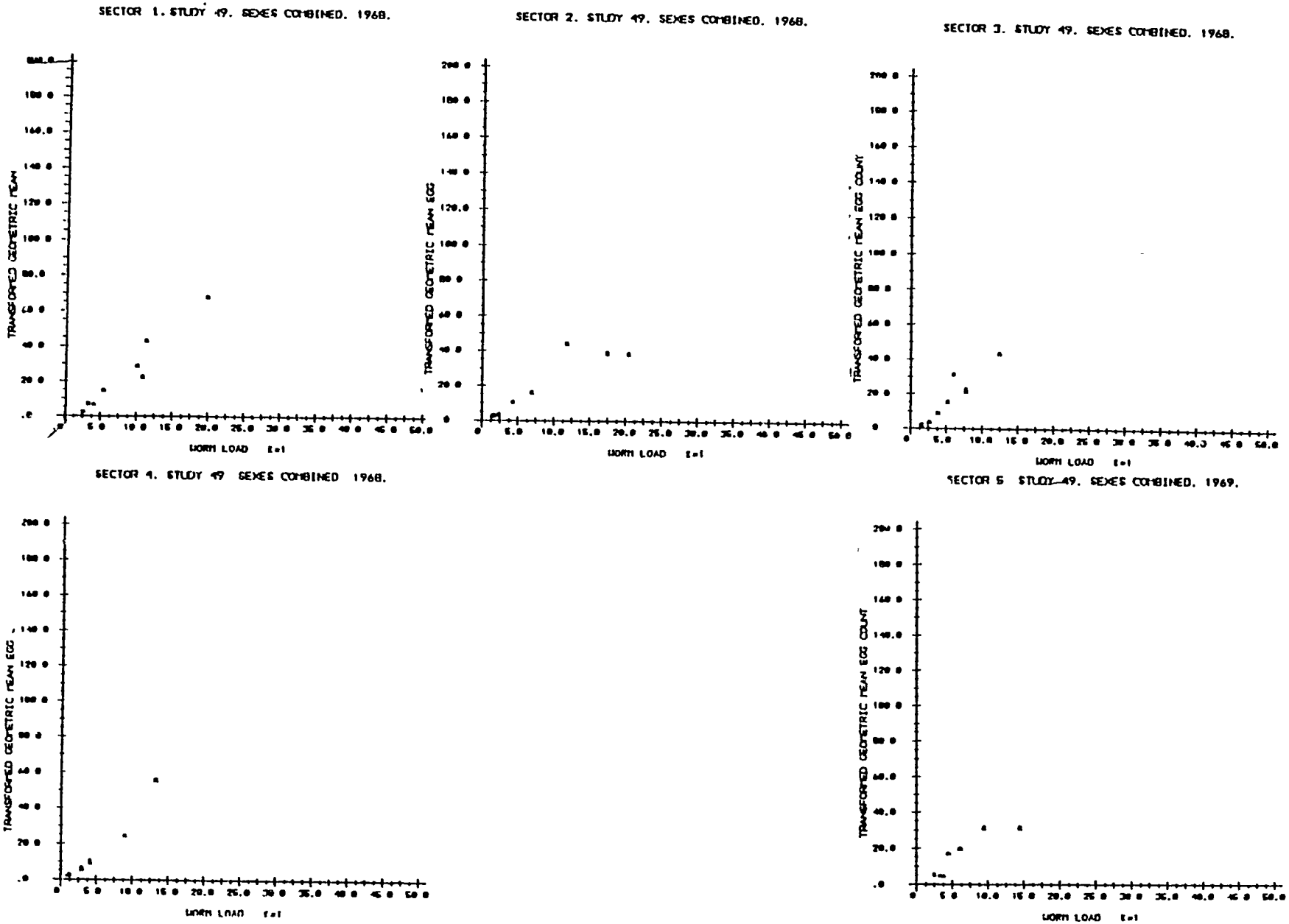

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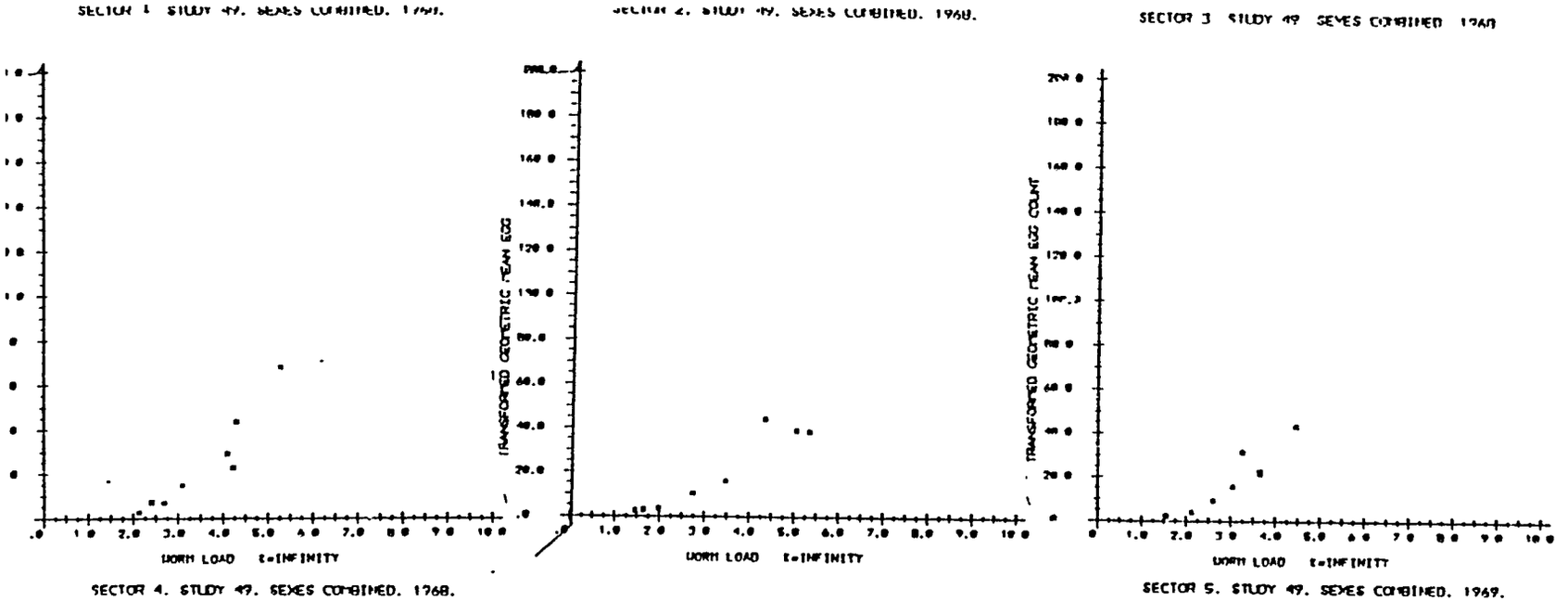


igure 9 (continued)

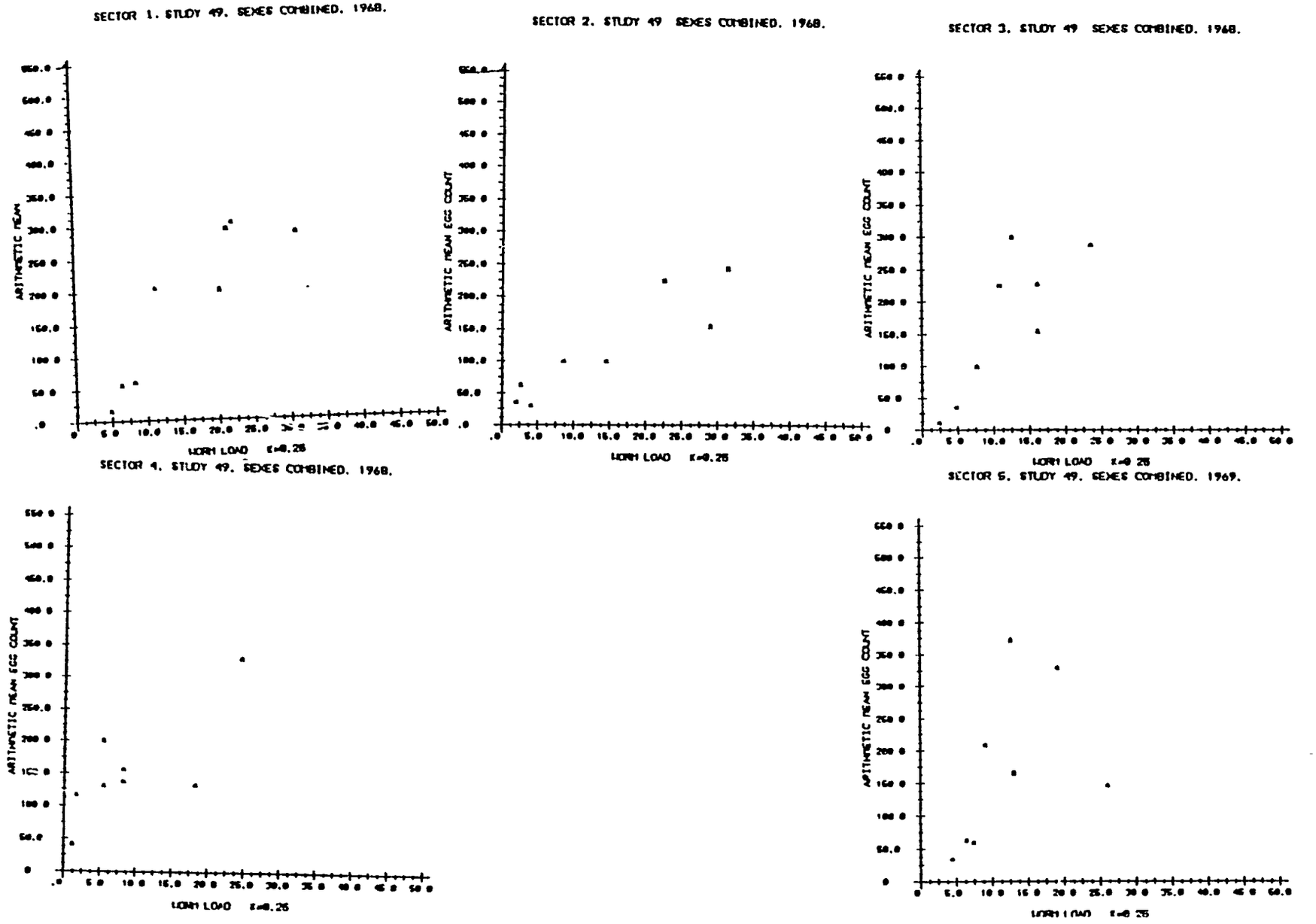


Figure 9 (continued)

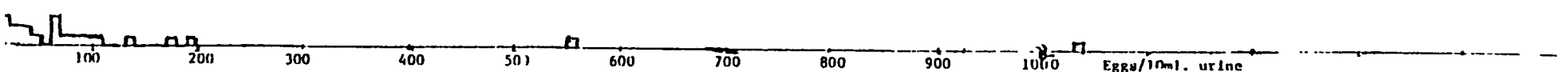


Figure 10. Plots of egg count frequency for 1) 2- and 7-year-olds and 2) 5- and 6-year-olds from all sectors, Misungwi, Tanzania. These are representative of the plots for the other ages up to 9-year-olds showing large numbers close to zero and a few high counts at the tail.

7 year olds, all sectors, Misungwi, Tanzania (N=113)

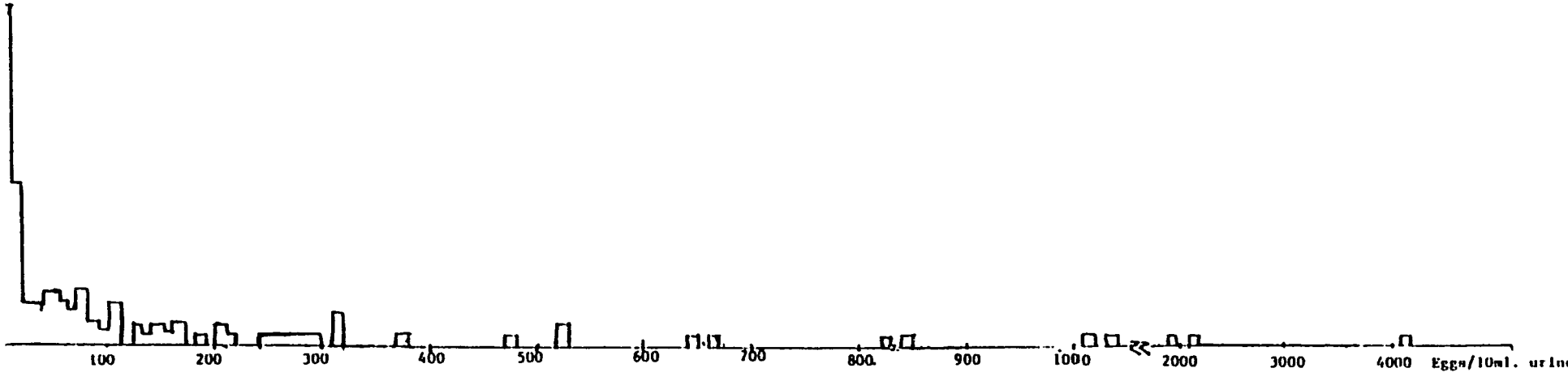
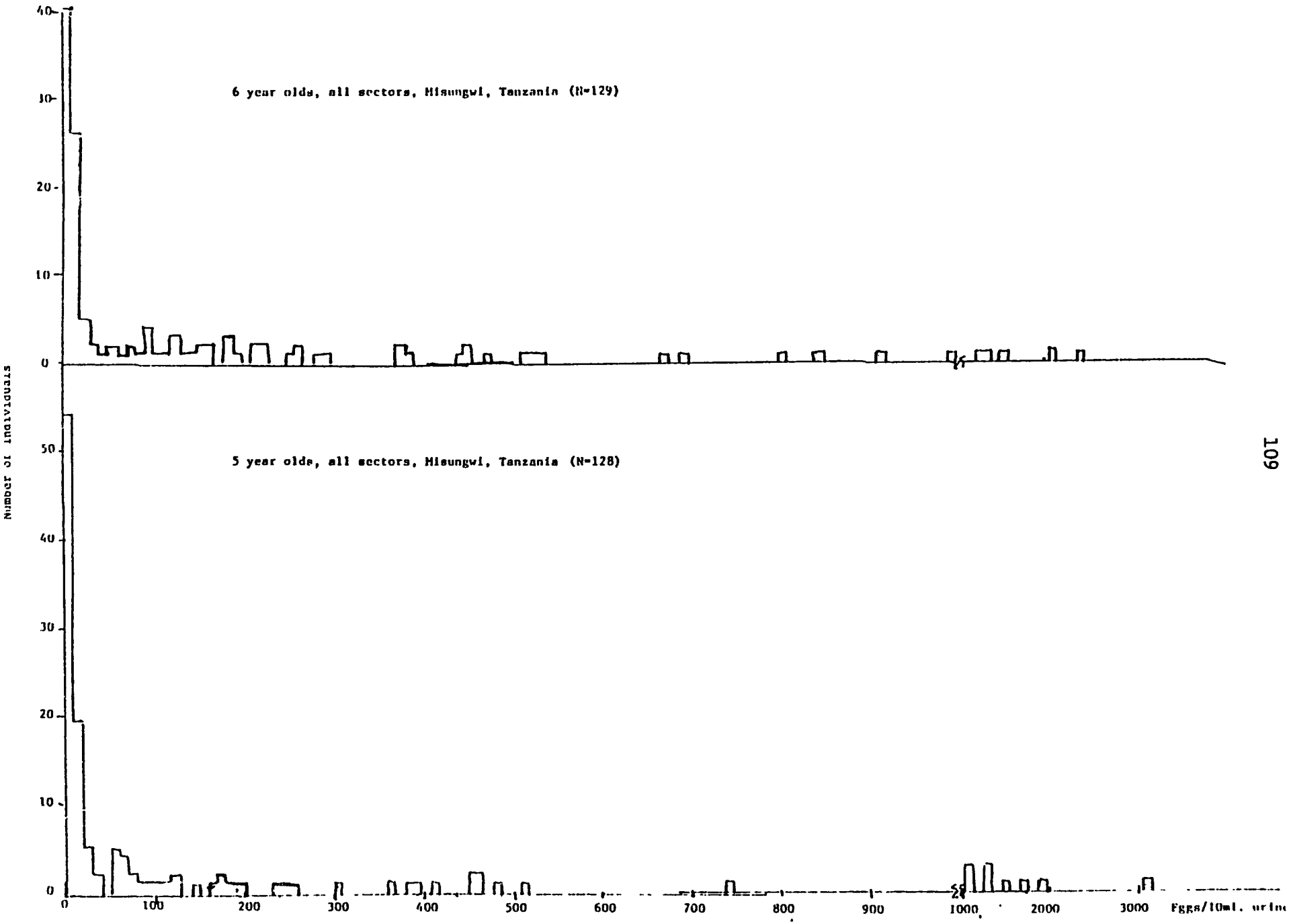


Figure 10 (continued)



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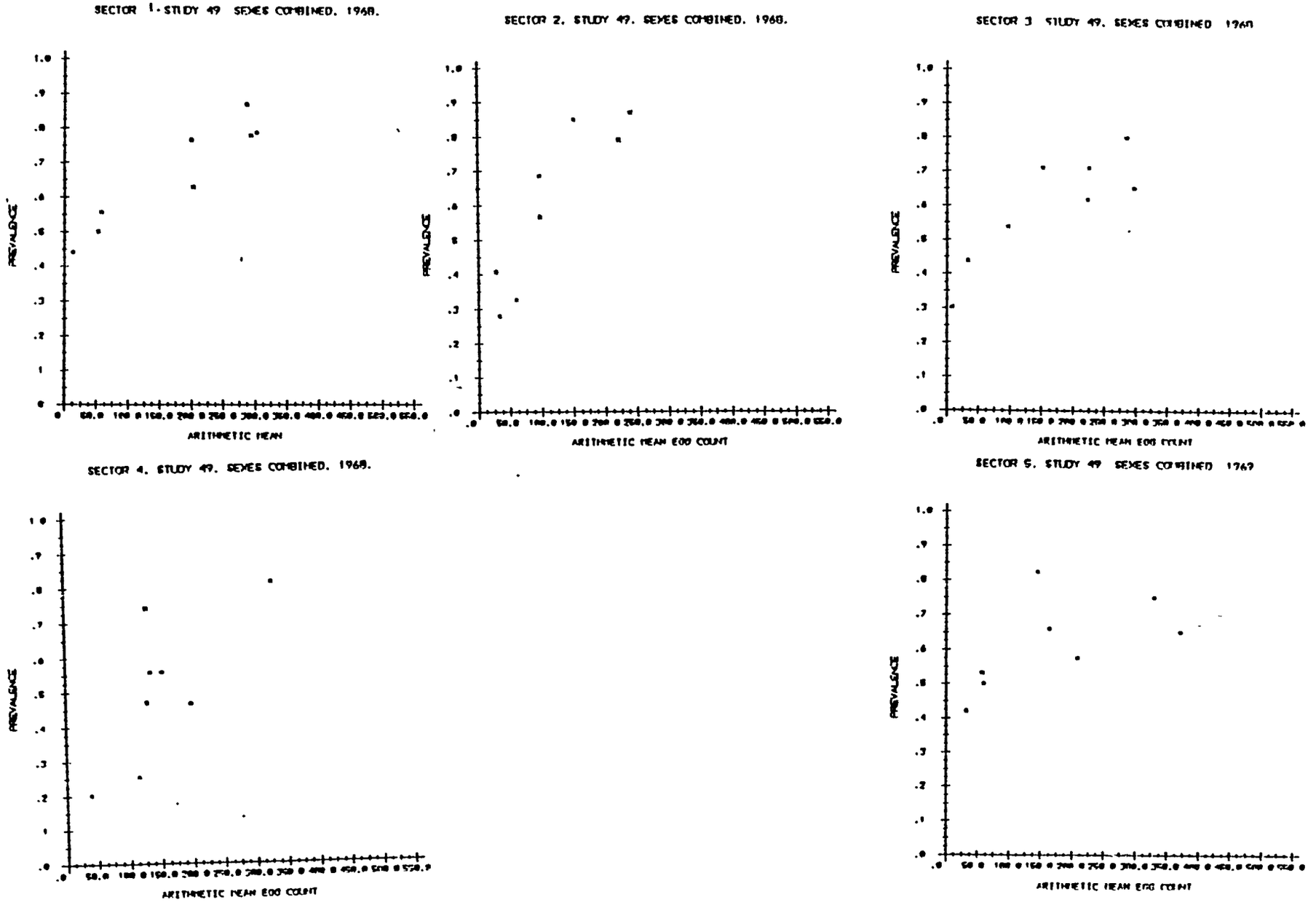


Figure 11. Plots of arithmetic mean egg count or geometric mean egg count versus prevalence for 2-to-9-year-olds in different sectors, Misungwi, Tanzania

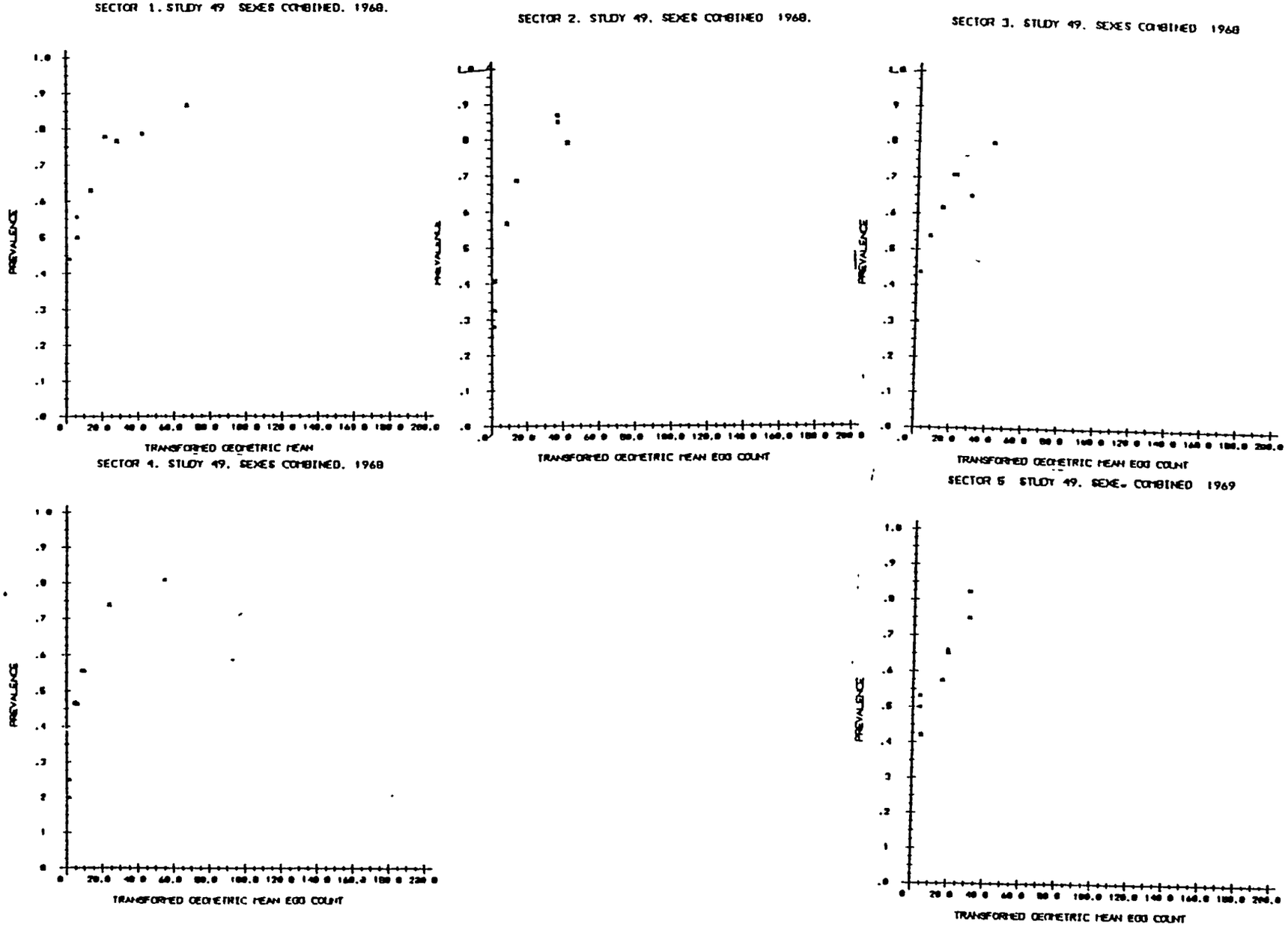


Figure 11 (continued)

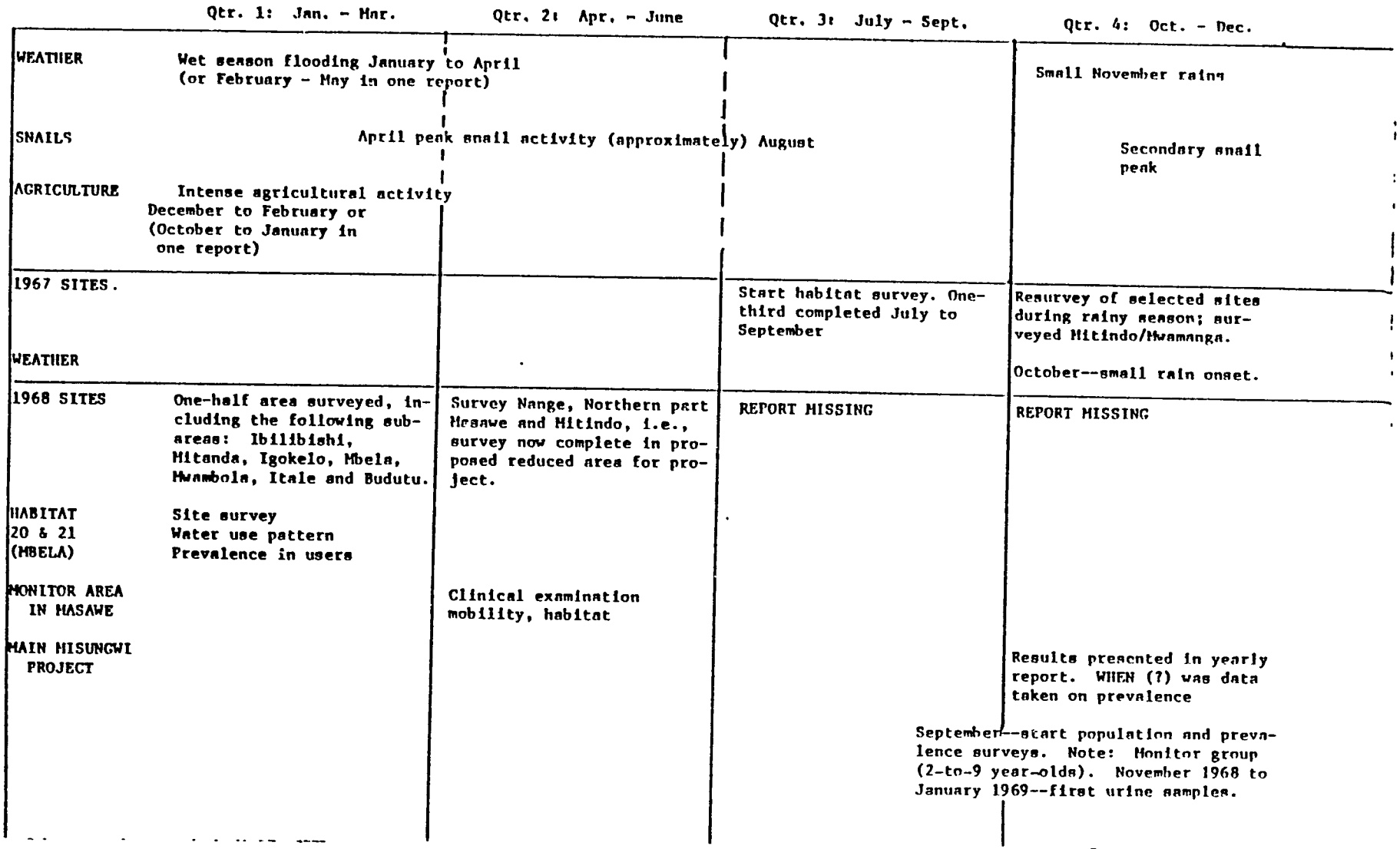


Figure 12. The Tanzania Schistosomiasis Control Project activities are outlined in the above chart by quarter and sector. Special studies are noted along with routine activities. In some cases, it was difficult to obtain the precise timing of certain activities. This is indicated on the chart.

	Qtr. 1: Jan. - Mar.	Qtr. 2: Apr. - June	Qtr. 3: July - Sept.	Qtr. 4: Oct. - Dec.
1969				
HABITAT 20 & 21 MBELA & MONITOR AREA (MASAWE)	Sometime since second quarter 1968 they performed snail density vs. season and habitat studies and incidence (2 to 13 years)		Continue habitat surveys Further urine tests (2-to-13-year-olds)	
MAIN MISUNGWI PROJECT	Continue and complete in March the population and prevalence surveys. Note: Sector V added and surveyed in 1970.			
MITINDO/ MWAMANGA CHEMOTHERAPY TRIAL AREA		Population and prevalence survey started and completed in April. Clinical survey--May assumed chemotherapy trial occurred in late May--not specifically stated.	Habitat and water use survey June and July (previously done also in October - November 1967). Follow up urine sample (after 3 months) end August.	
SECTOR I & II (OLD DESIGNATION)			Habitat resurvey.	
MONITOR GROUP 2-TO-9 YEAR-OLDS			Second urine sample September to October.	
ALL SECTORS				Modification of sector plans
MWANZA				Start prevalence and egg survey in a monitor group (2 to 14 years)

Figure 12 (continued)

	Qtr. 1: Jan. - Mar.	Qtr. 2: Apr. - June	Qtr. 3: July - Sept.	Qtr. 4: Oct. - Dec.
1970				
SECTOR V	Sector V. Habitat, prevalence and census surveys		REPORT MISSING	
MASAWE	Mobility study--Van Etten			
SECTORS I-IV		First molluscicide--blanket treatment 7 May to 23 June.	Second molluscicide treatment--August-September.	Post chemotherapy follow-up urine analysis--previous egg count. (was everyone surveyed? When was survey done? December?)
SECTORS II & III			Chemotherapy--July 13 to August 3. Urine sampled. (Was urine before or after chemotherapy?)	
SECTORS I-V				Population census I, IV, V then II & III. Initiated in August 1970; completed toward end of year.
MITANDO-MWAMANGA CHEMOTHERAPY TRIAL		12-month follow-up. Urine collected June. Treat positives		
MONITOR GROUP 2 TO 9 YEARS				Prevalence/Incidence survey

Figure 12 (continued)

	Qtr. 1: Jan. - Mar.	Qtr. 2: Apr. - June	Qtr. 3: July - Sept.	Qtr. 4: Oct. - Dec.
1971				
SECTORS I-V	Third molluscicide cycle 13 January to 5 March	Fourth molluscicide cycle 17 May to 16 June	Fifth molluscicide cycle 16 August to 3 September	
MITANDO/ MWAMANGA CHEMOTHERAPY		Second annual census and urine exams--May 1971		
SECTOR II				Migration study October 1971 - January 1972
MONITOR GROUP 2-TO-9-YEAR-OLDS				Prevalence/incidence November
1972				
SECTORS I-IV	Sixth molluscicide cycle February and March	Seventh molluscicide cycle June 12 - July 18		
SECTOR II	Finish population movement study October 1971			
MITINDO/ MWAMANGA		Prevalence/incidence surveyed. All positives again treated.		
SECTORS I-V			Prevalence/incidence/egg output/census of Misungwi begun in August--completed in November	
				Further observation on mobility, socio-economic conditions and water use completed--area unspecified.

Figure 12 (continued)

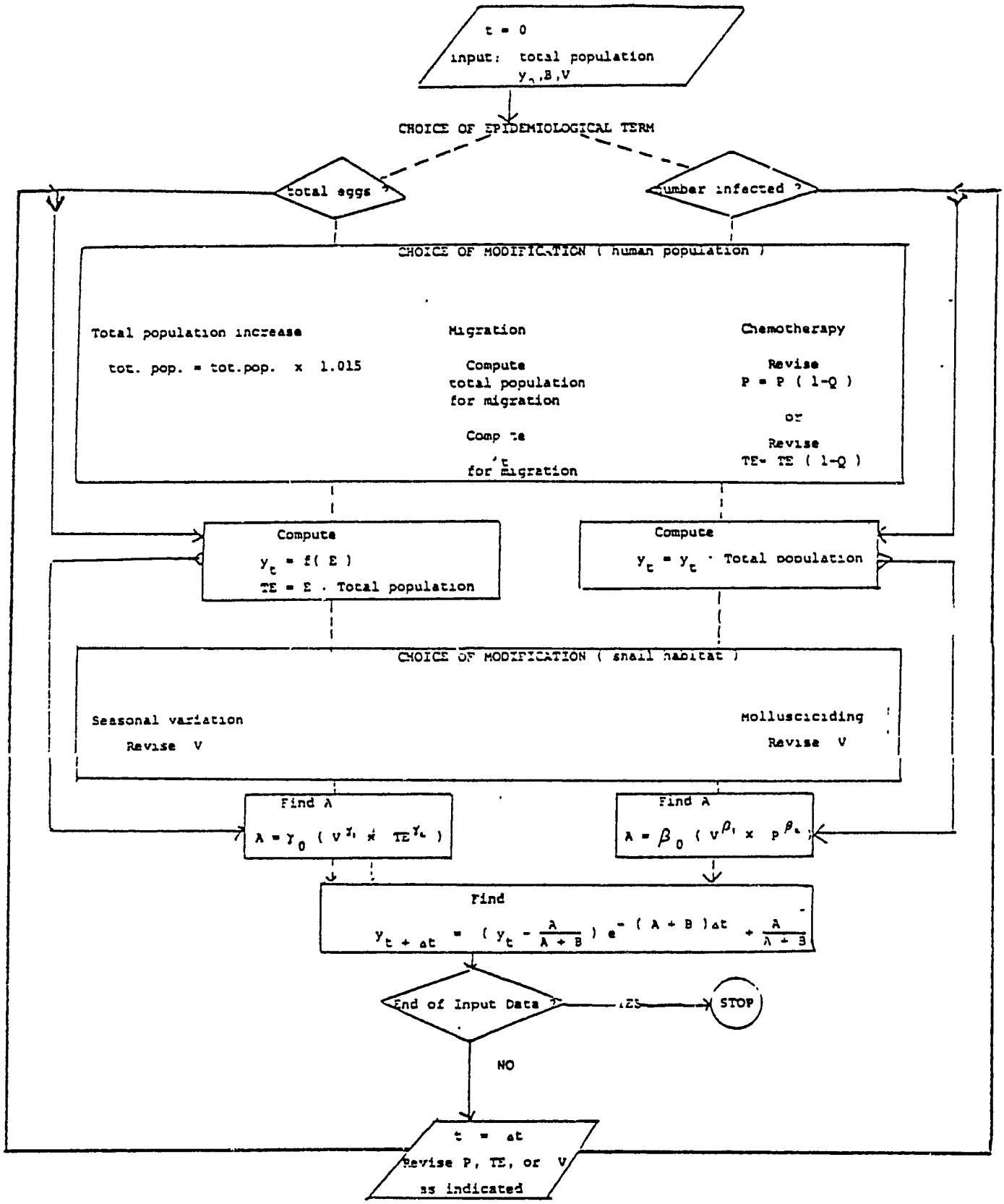


Figure 13. The logic of the model is diagrammatically represented in the flow chart. Straight lines (——) indicate the basic version of the model. Possible modifications are linked to the model via dashed lines (---). The variables used are: y = baseline prevalence or fraction positive; A = incidence rate; B = loss rate; V = feet of snail habitats within one-half mile of households/ age group; P = number of infected persons/age group; E = arithmetic mean/age

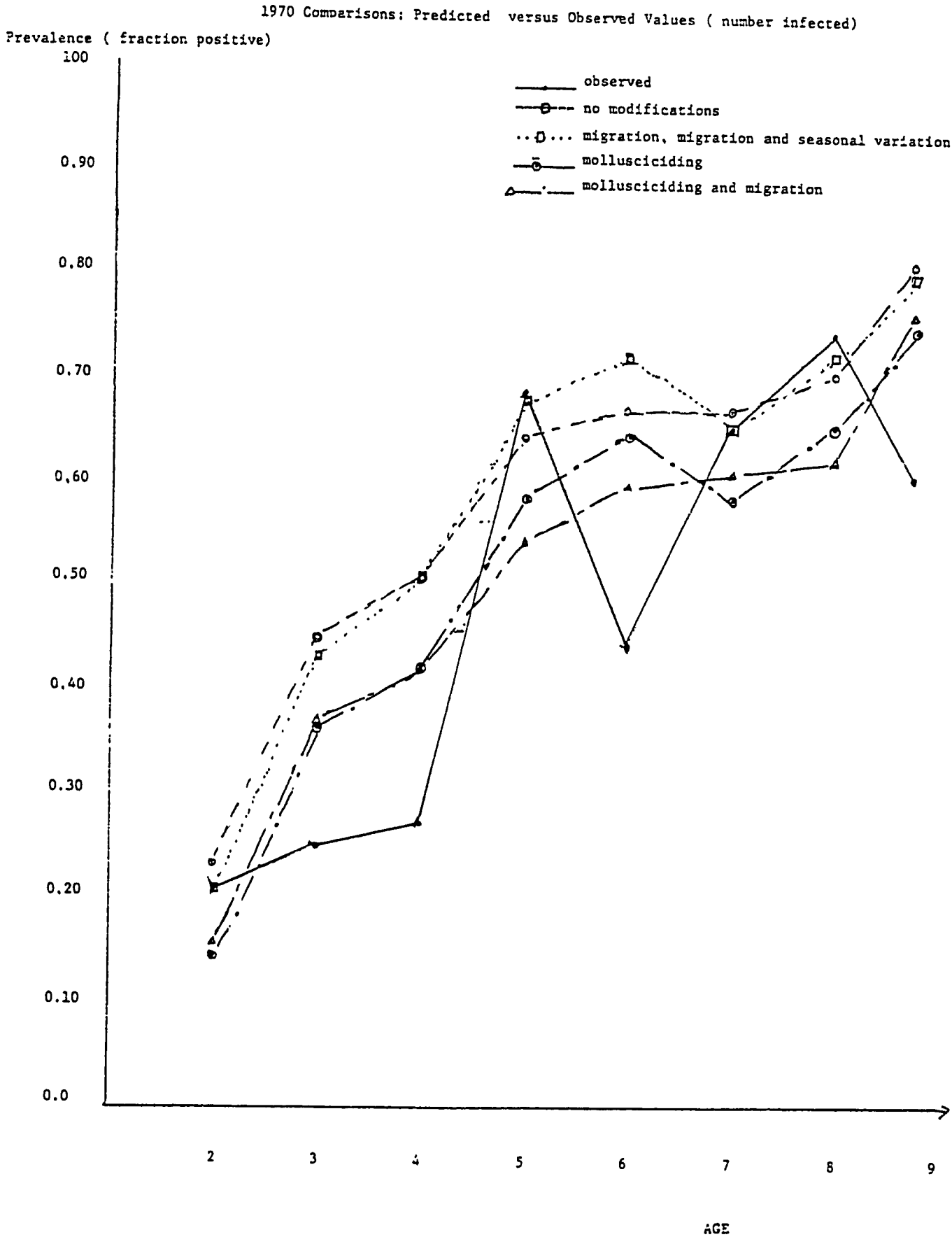


Figure 14. For 1970, age-specific comparison of predicted prevalence levels from model modifications with observed data from Sector IV, Misungwi Tanzania. 1970 represents the first year of schistosomiasis

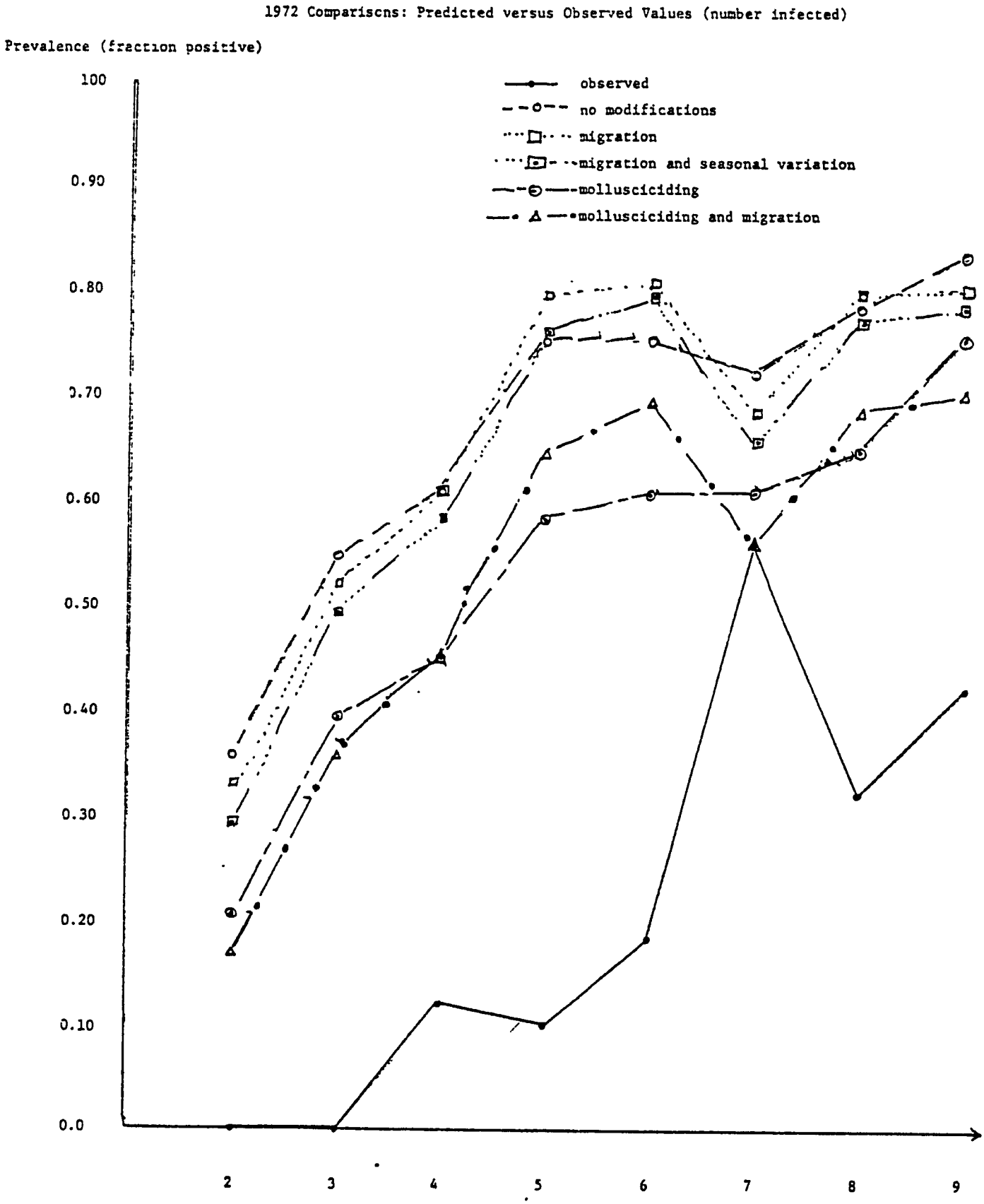


Figure 15. For 1972, age-specific comparison of predicted prevalence levels from model modifications with observed data from Sector IV, Misungwi, Tanzania. 1972 represents the third year of schistosomiasis control activities (seven cycles of molluscicide). The prevalence results are predicted

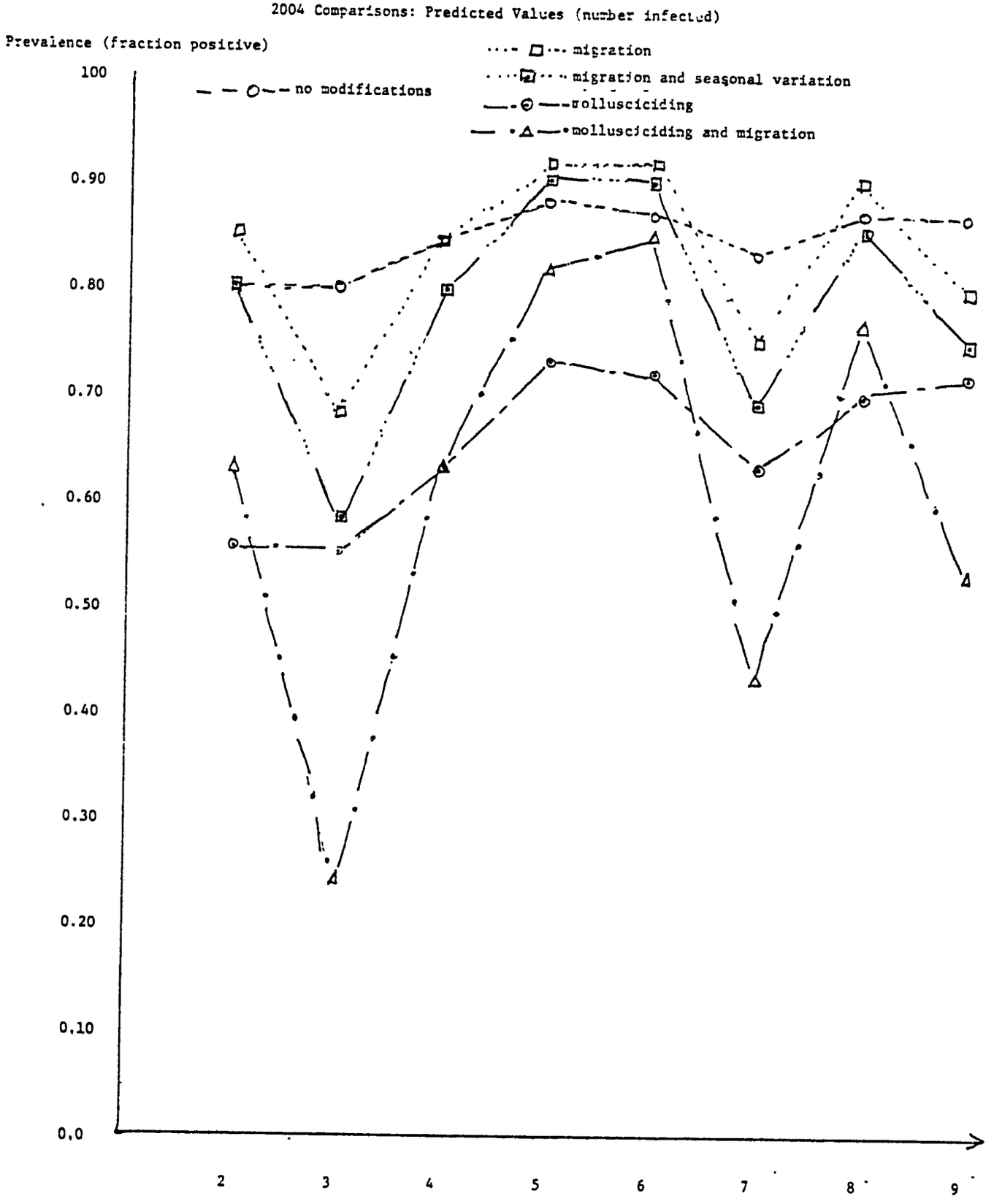


Figure 16. For 2004, age-specific predicted prevalence levels from model modification for Sector IV, Misungwi Tanzania. The prevalence results are predicted from incidence equation [10] using number infected which differ only slightly from those obtained by use of equation [11] (total eggs).

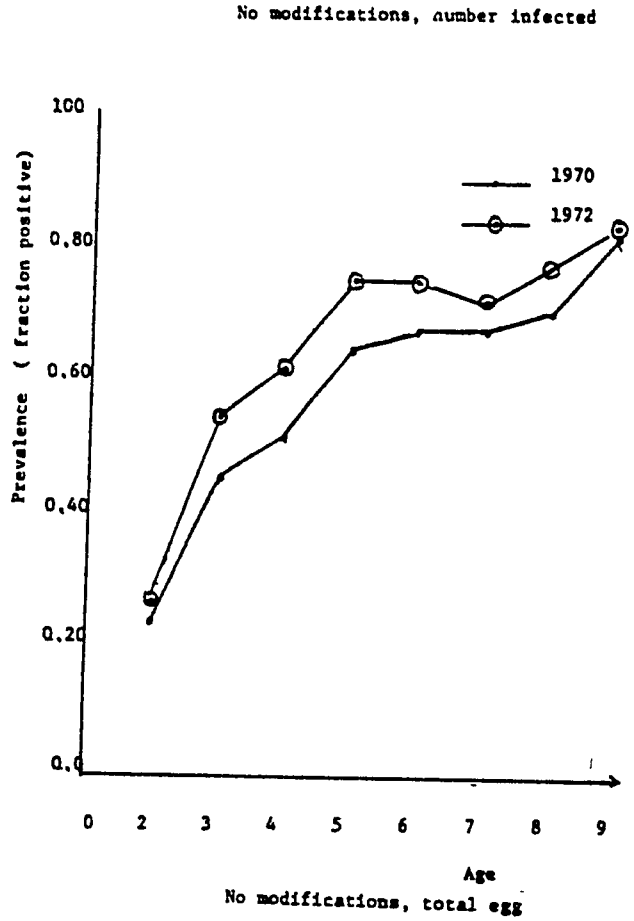
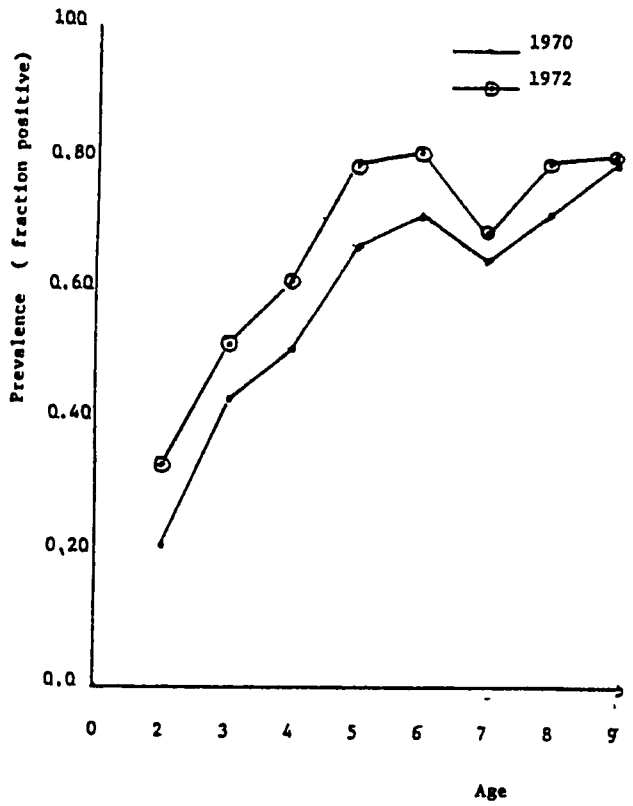
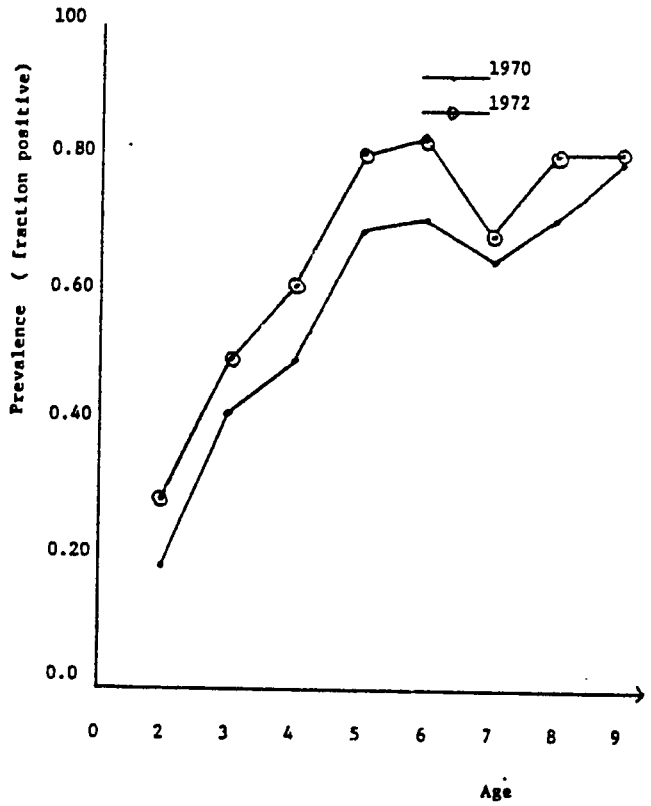


Figure 17. Age-specific predictions of prevalence made with various modifications to the model, based on data from Sector IV, Misungwi, Tanzania. Each graph shows the results for 1970 and 1972. In addition, prevalence results from each modification are given for use of number infected or total

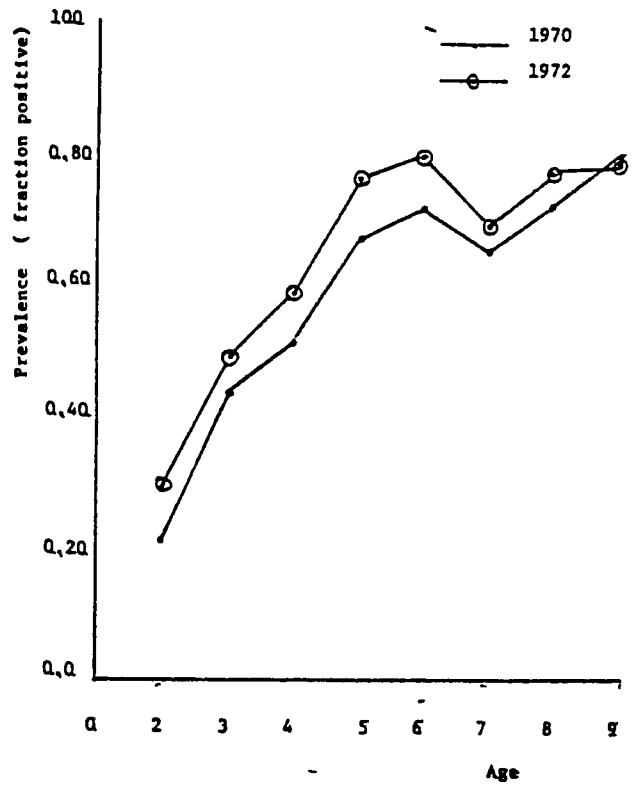
Migration, number infected



Migration, total egg



Migration and Seasonal Variation, number infected



Migration and Seasonal Variation, total egg

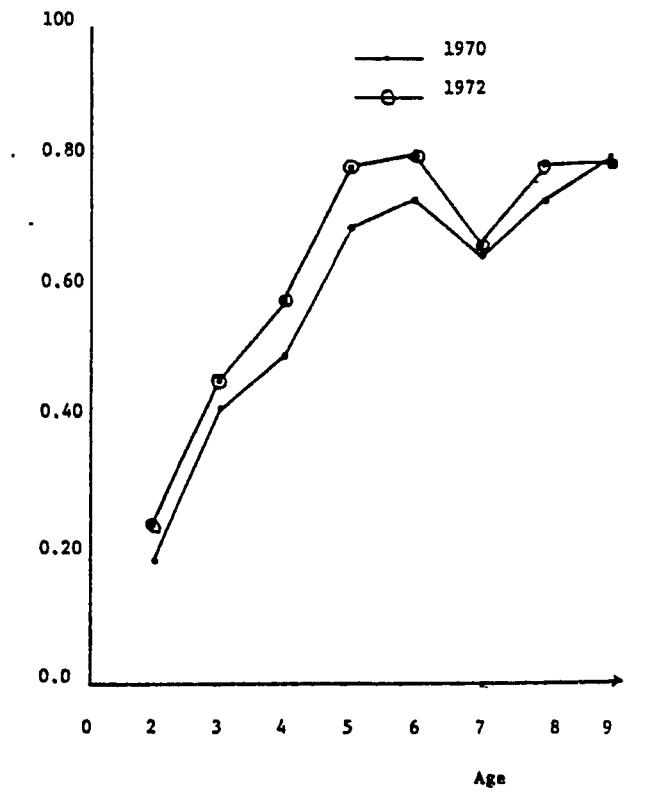


Figure 17 (continued)

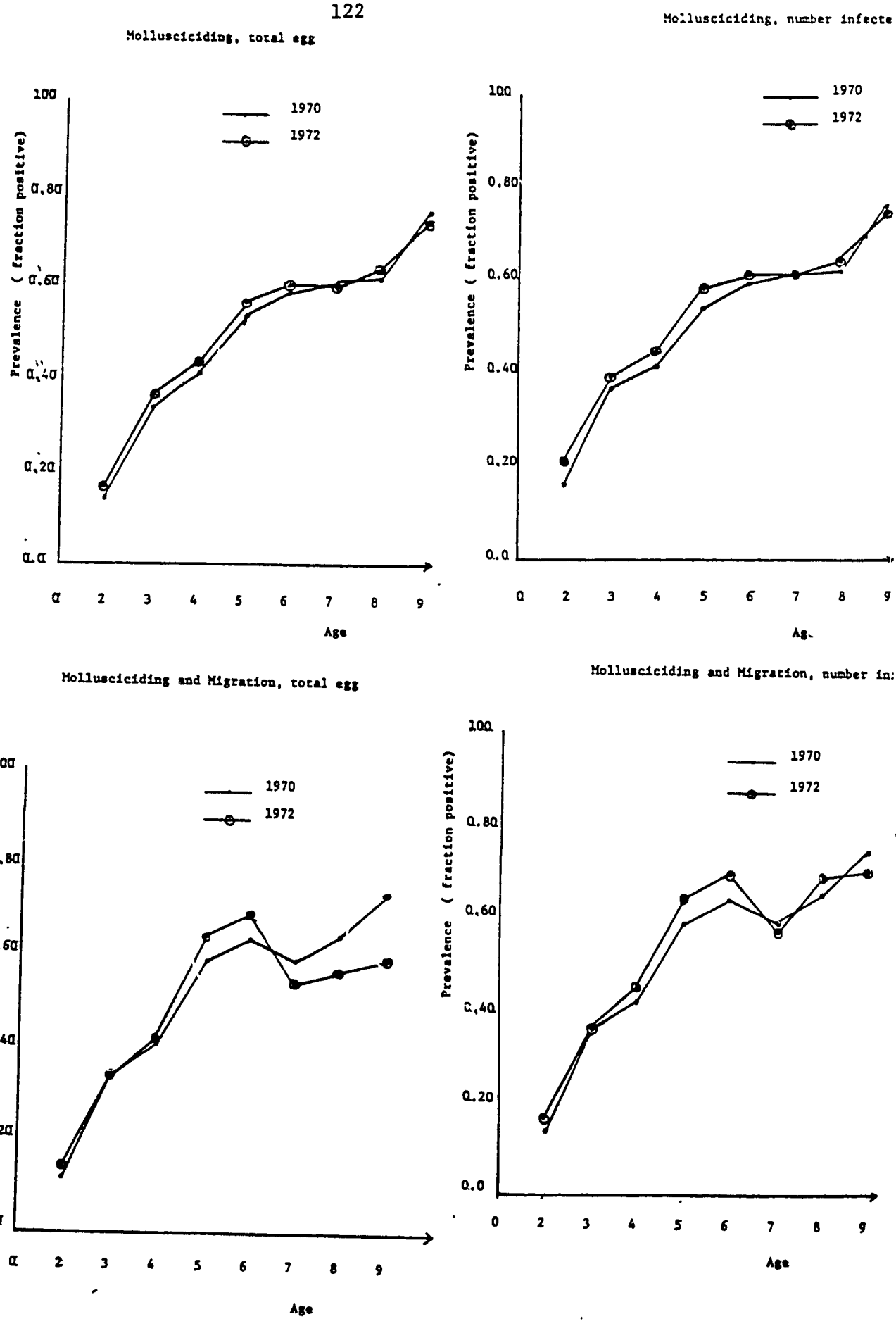


Figure 17 (continued)

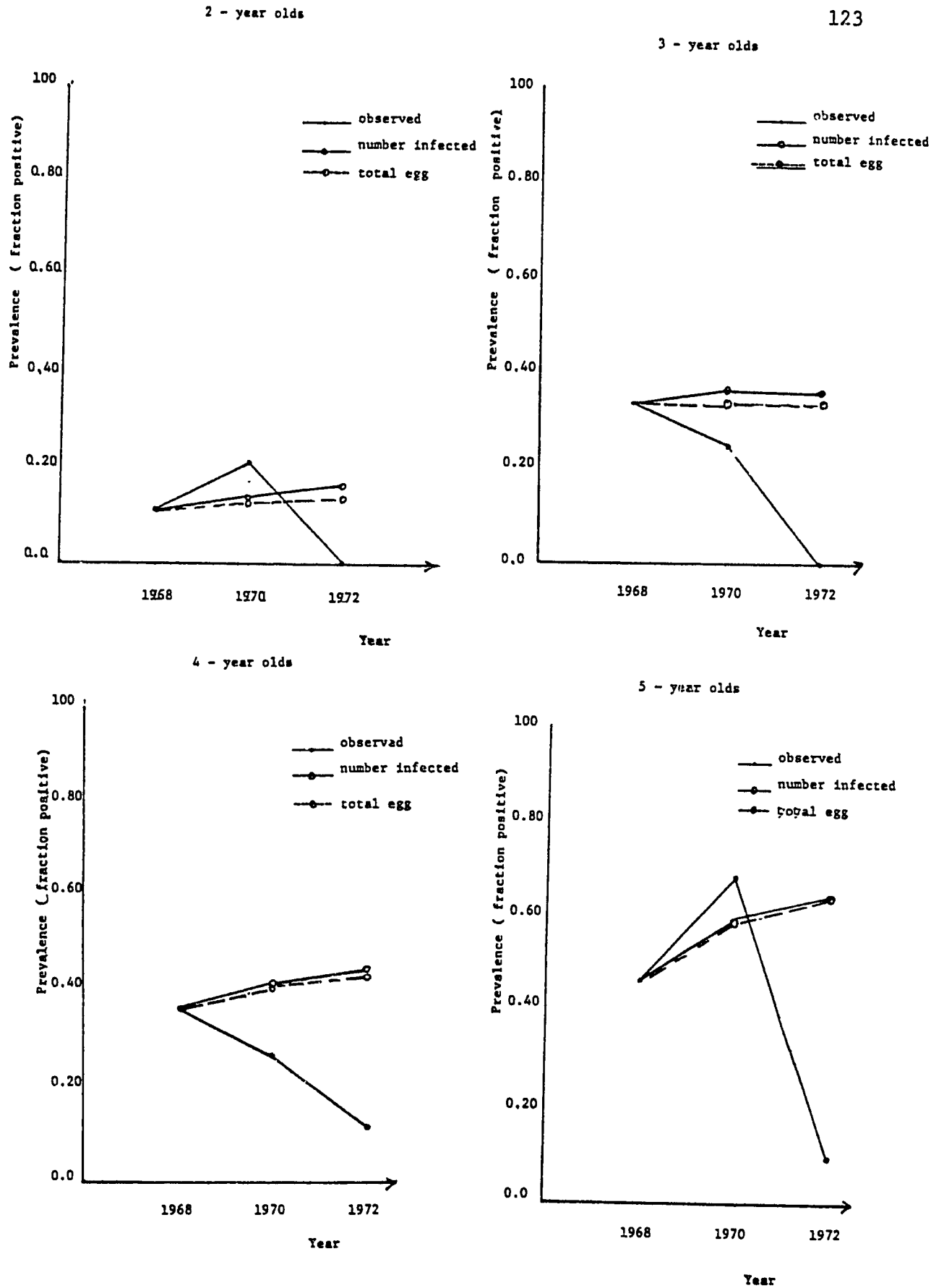
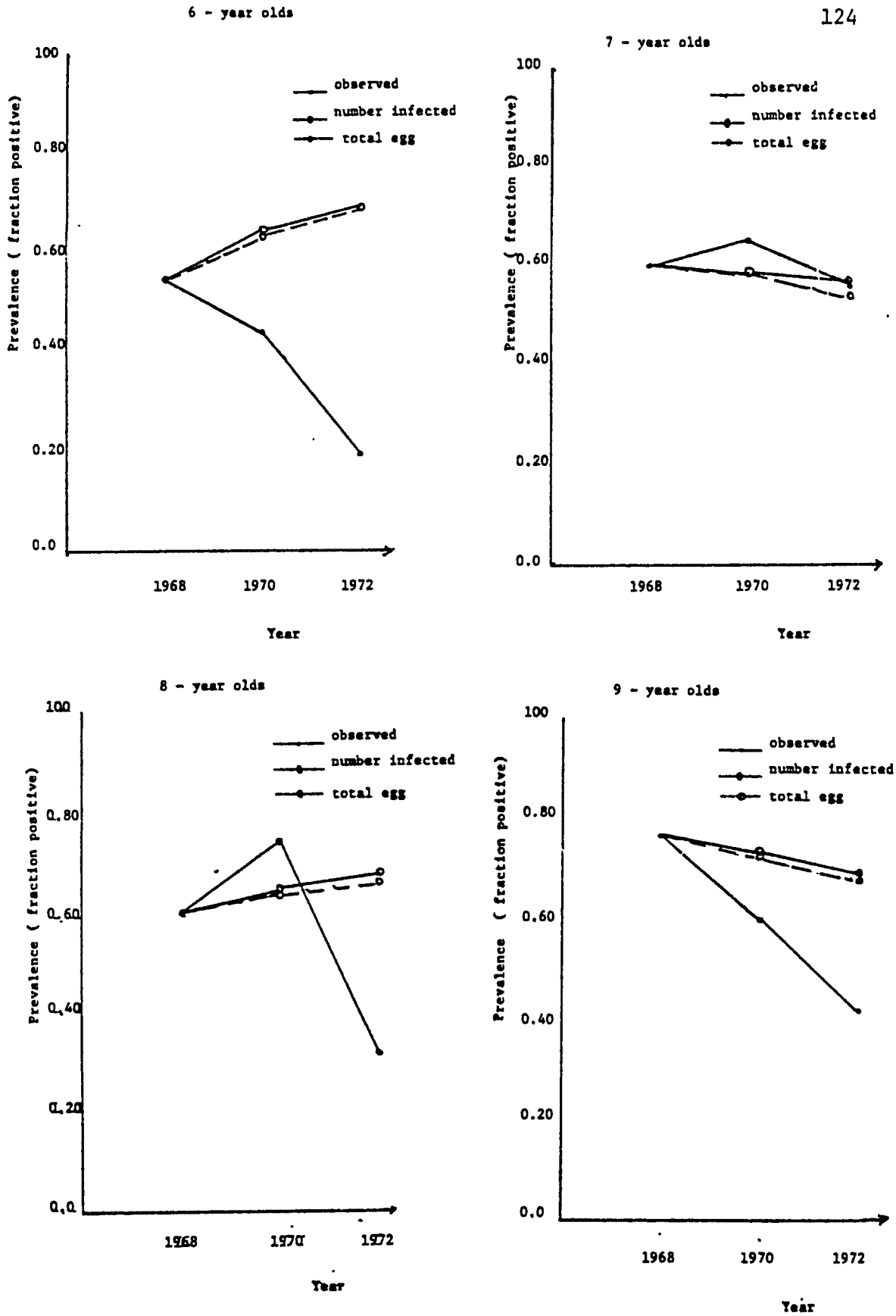


Figure 18. Comparisons of Prevalence Predictions with Observed Data from Misungwi, Tanzania over time (1968-1972) for each age. The prevalence predictions are compared also for use of number infected or total eggs in the incidence equation.



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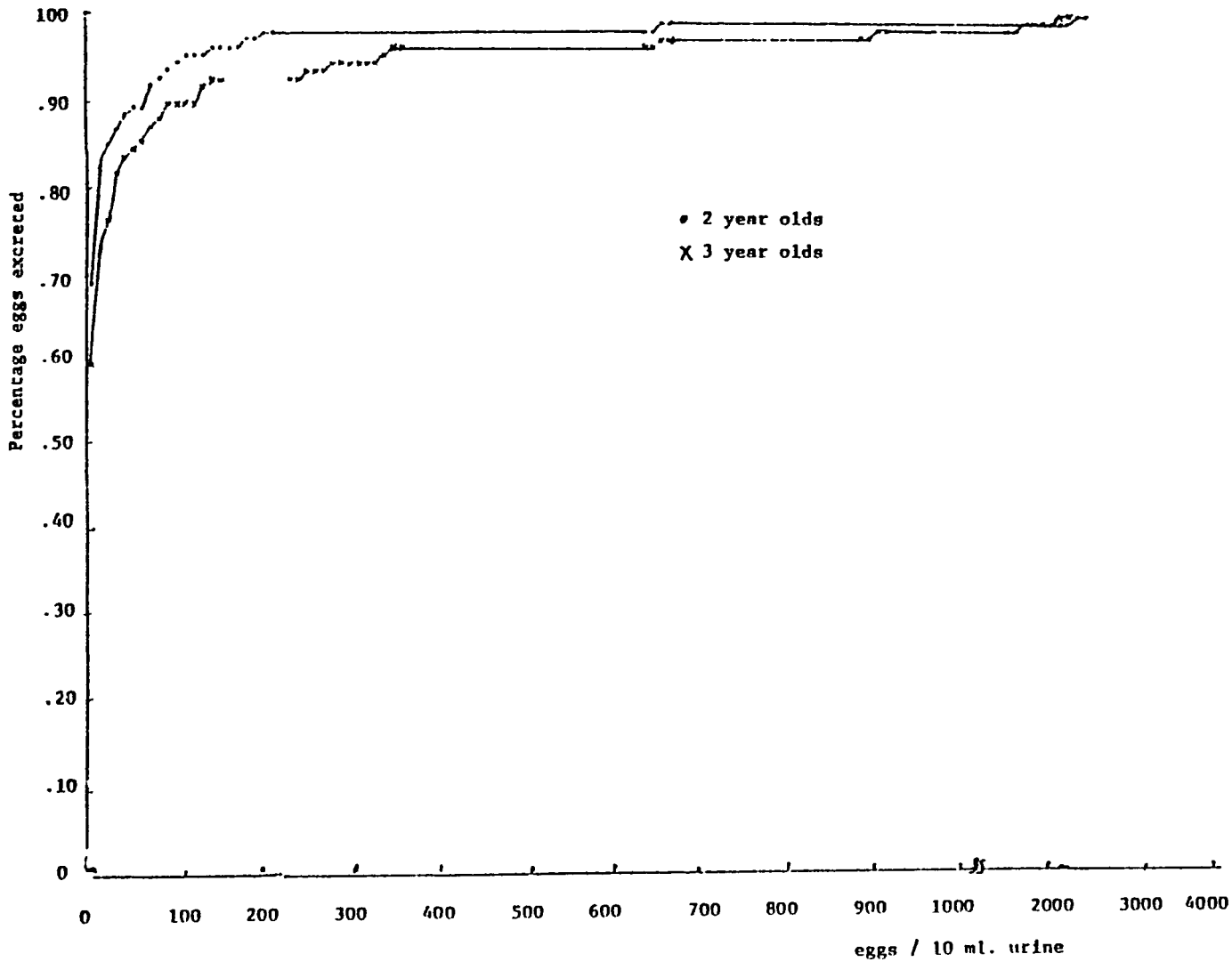


Figure 19. Cumulative frequency plots of egg counts for all ages and over all sectors from 1968 egg count data. These are included only to show the pattern of egg counts over age. The percentage of eggs excreted rises consistently with age until age 15 at which point the percentage of eggs excreted declines.

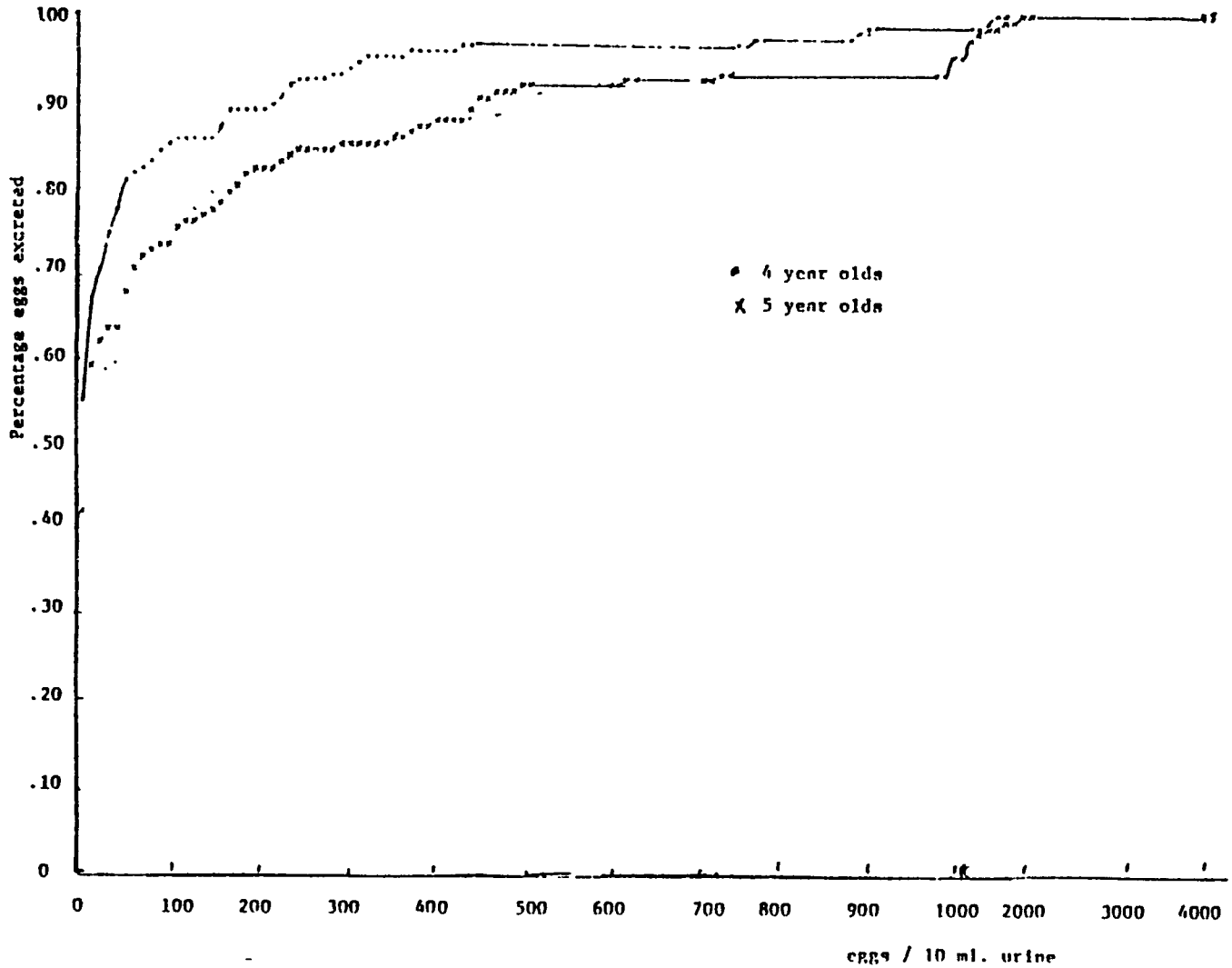


Figure 19 (continued)

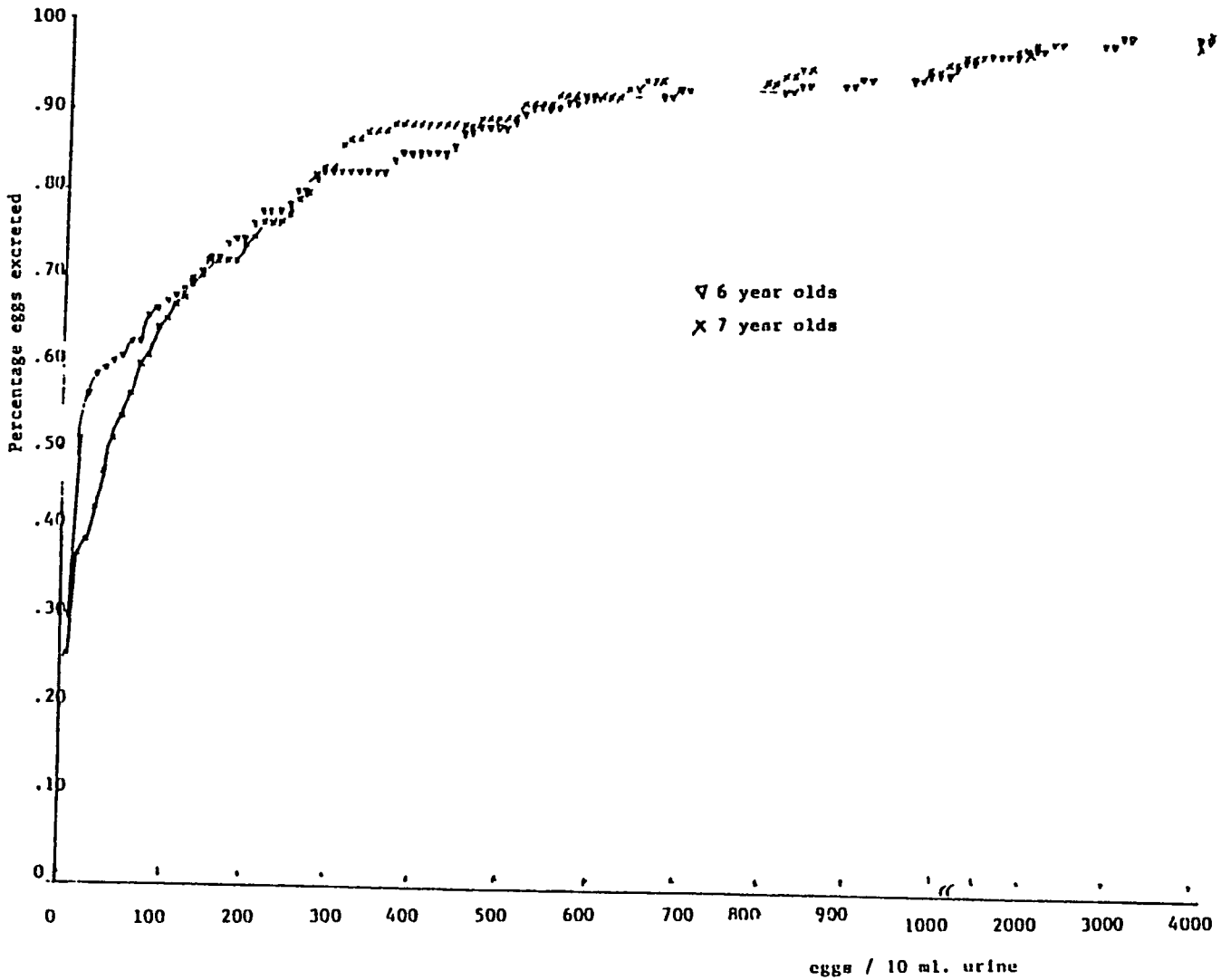
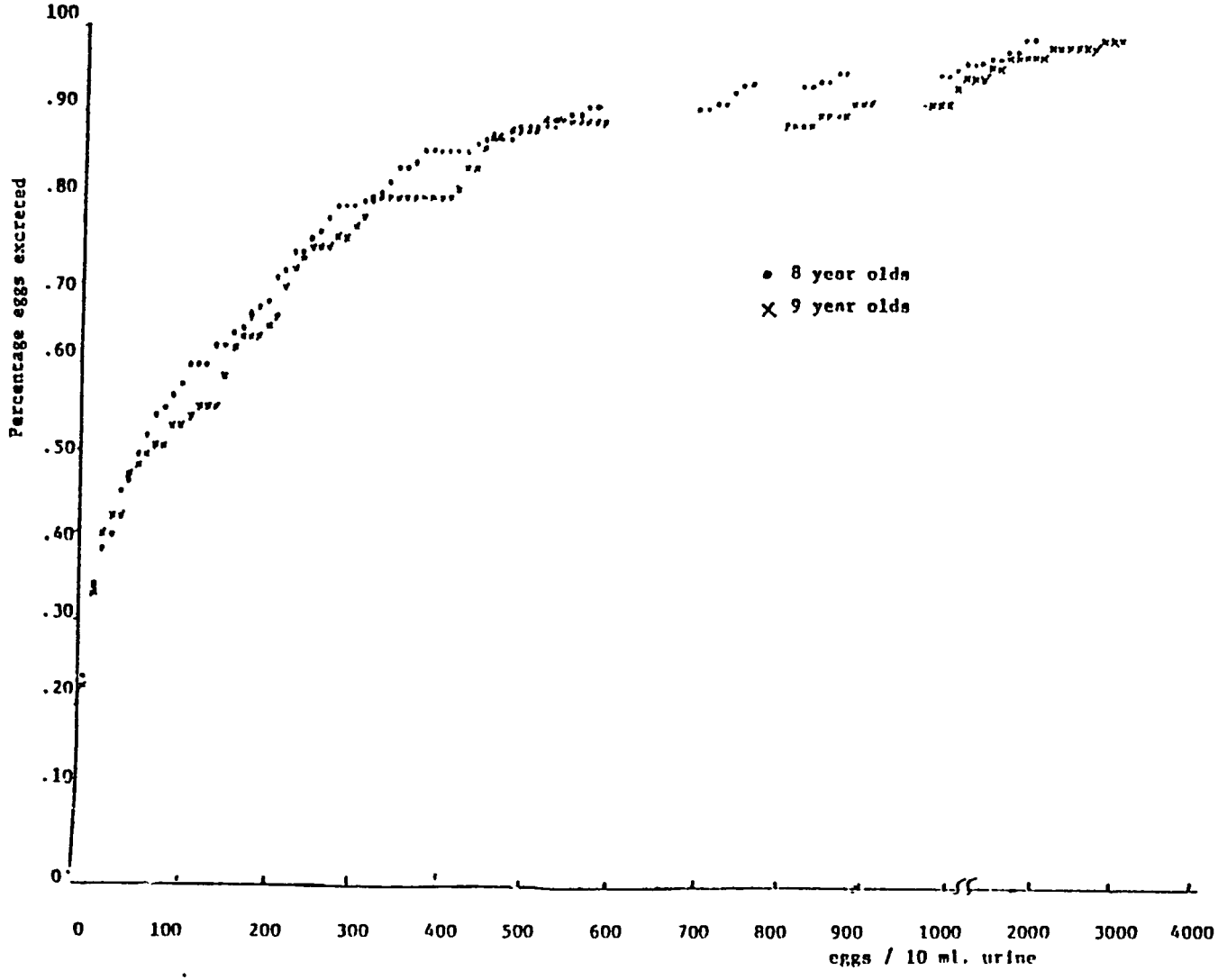


Figure 19 (continued)



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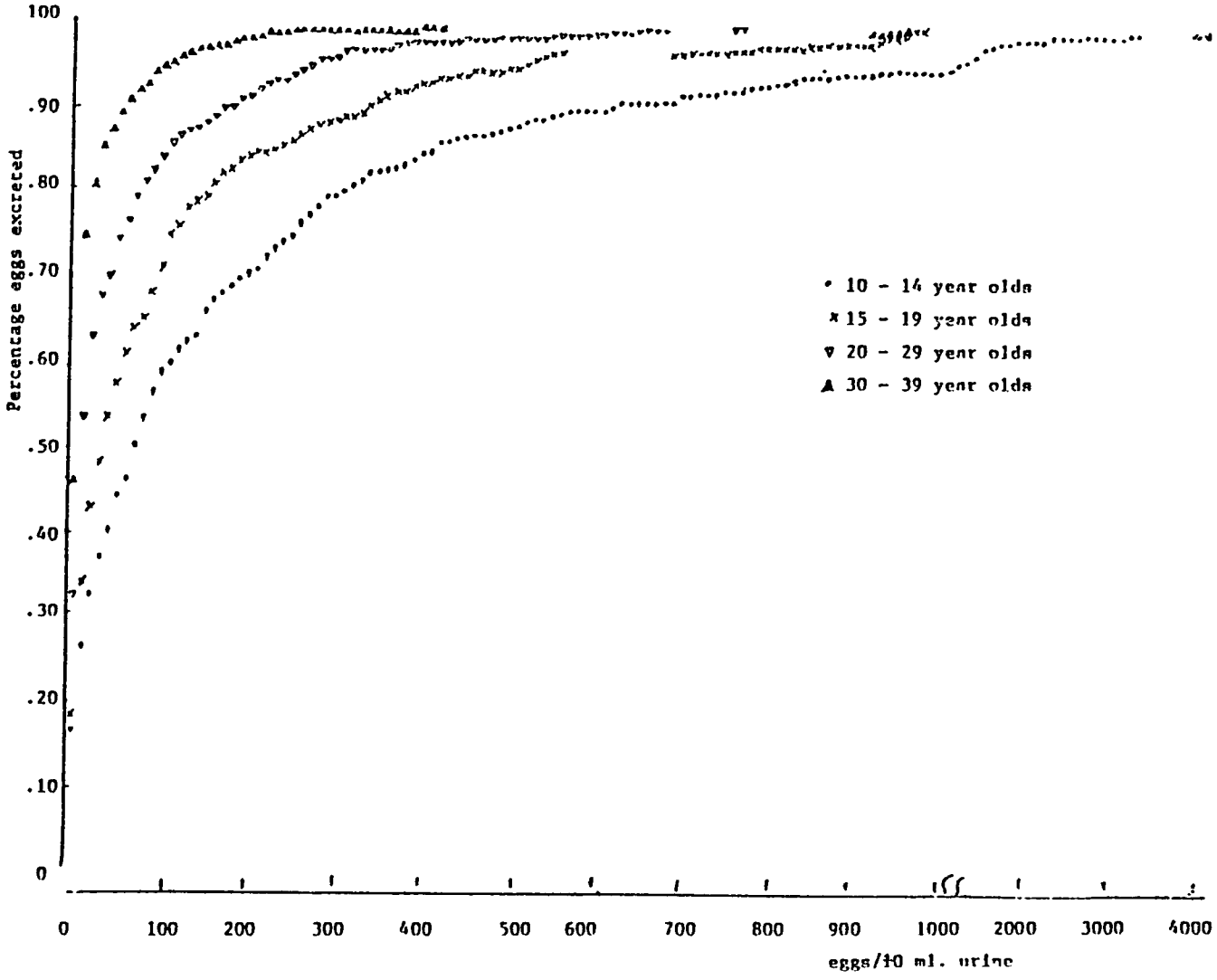

Figure 19 (continued)

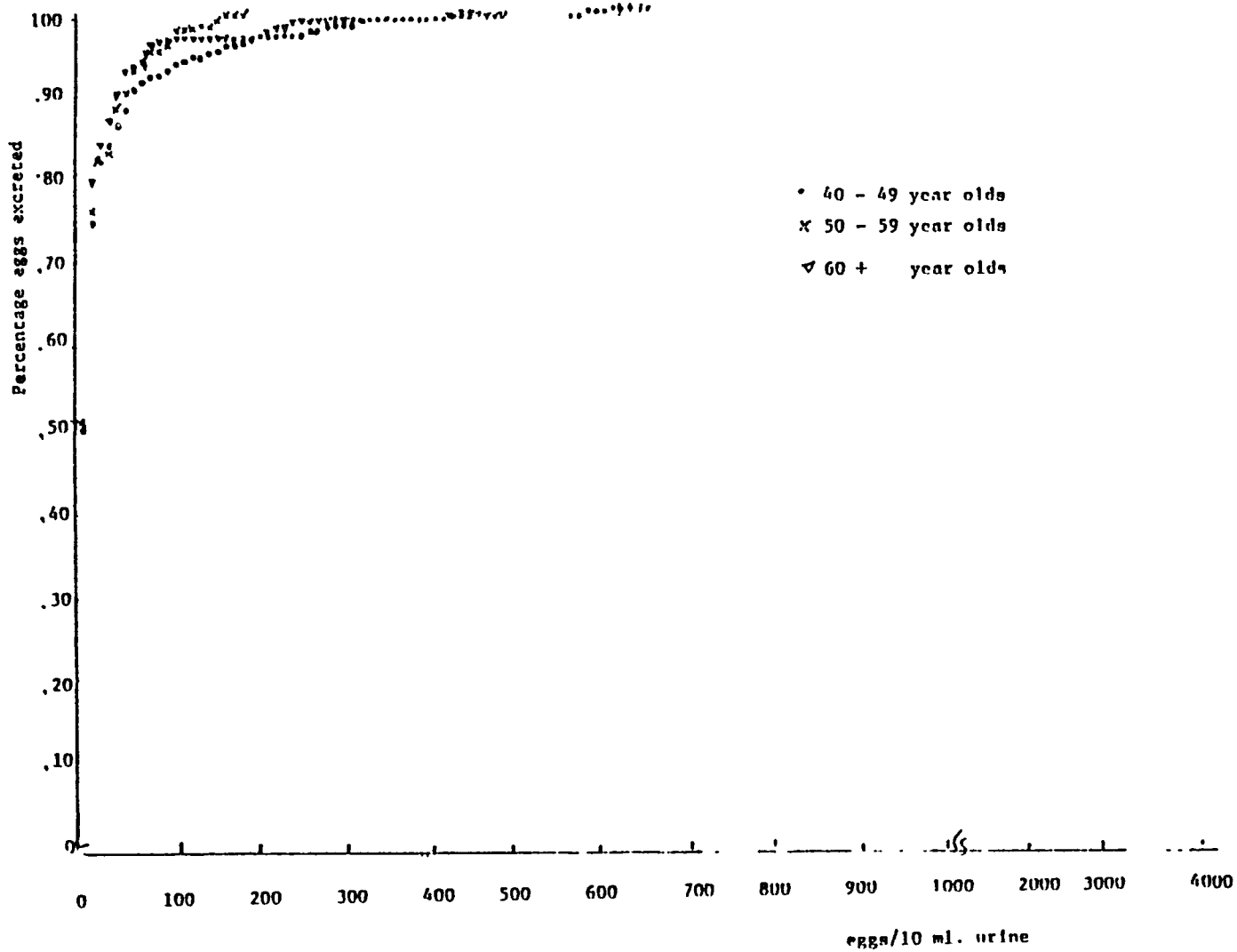

Figure 19 (continued)

APPENDICES

Appendix I

CALCULATIONS AND ASSUMPTIONS FOR INCLUDING DIFFERENT TERMS IN THE MODEL

In this appendix we give the methods we used for calculating the terms in the model. In addition, we describe the decision criteria for inclusion of households and habitats. Data for individuals are used in the calculation of reversion and incidence rates only if the computer file includes egg counts for both 1968 and 1970:

$$\text{Incidence} = \frac{\text{Number of people uninfected in 1968 but infected in 1970}}{\text{Number of people uninfected in 1968}}$$

$$\text{Reversion} = \frac{\text{Number of people infected in 1968 but uninfected in 1970}}{\text{Number of people infected in 1968}}$$

$$\text{Arithmetic mean of egg counts} = \frac{\sum_{i=1}^{N} \text{egg count of individual } i}{N}$$

$$\text{Antilog (Geometric mean of egg counts)} = \frac{\frac{1}{N} \sum_{i=1}^{N} \log (\text{egg count of individual } i)}{N}$$

Population increases were calculated assuming three-fourths of a percent increase per year. Therefore, for a two-year period, population (year 2) = 1.015 x population (year 0).

Migration (See discussion in text.)

Snail habitats

Volume (V)

When computing the volume of habitat, the habitat was treated as a sector of a sphere if length equaled width and as a sector of an ellipsoid otherwise. Letting L = length, W = width and D = depth, then:

If $L = W$, let $R = D^2 + L/4$ Then $V = \Pi R^2 D - \frac{\Pi R^3}{3} + \Pi\frac{(R-D)^3}{3}$

If $L = W$, $V = \frac{\Pi\ LWD}{4}$

To compute the volume of accessible habitat for each household, the volume of all the habitats at the given distance from the household were summed. To compute the age-specific habitat volume, each person in a particular age group was assigned the volume of his household, and the assigned volumes for all members of the age group were summed.

Perimeter (P)

When computing perimeter, the habitat was treated as a circle if length equaled width and as an ellipse otherwise:

If $L = W$, $P = \Pi\ L$

If $L = W$, $P = \Pi \sqrt{\frac{L^2 + W^2}{2}}$

The computation of the perimeter of accessible habitat for each household and each age group was similar to the corresponding volume measures.

Decision Criteria with Reference to Households and Habitats

Households

All households which could be located by number on the Sector IV map and for which data were available in the computer file were used. Out of the computer data file of 124 households, we used 117.

Habitats

The decision criteria for habitats were more complicated. Not only did we need to use habitats located on the map by number and also in the computer data file, but we also had to decide which habitats were really transmission sites. The distance criterion described in the text was an assumption on our part about habitat accessibility. Next, we determined: 1) whether the habitat contained schistosomiasis carrying snails (in at least one of two surveys when the site was resurveyed); 2) whether the habitat was in fact dry (as indicated by the project under the item "is the water used"); and 3) the transmission potential of the habitat as determined by the project. Since there were many habitats that were originally surveyed by the project during 1967 and resurveyed in a different season in 1968, we could check a) to see if the habitat were snail-free consistently or if B.(P.) nasutus or other hosts ever appeared and b) to see if the habitat remained dry all year. The project had identified drinking ponds as unlikely transmission sites because users usually kept them

quite clean and clear of vegetation. Nonetheless, many drinking ponds were both used frequently and had B.(P.) nasutus so they were included. The decision criteria for determining which snail habitats to include may be summarized as follows:

1. Habitat must be numbered on map and have data in computer file.
2. Snails which transmit S. haematobium must be present or, if two years of data, they must be present at least in one year.
3. Transmission potential must be considered "probable" or "possible" by project. If considered "unlikely" or "equivocal" by project but appropriate snail species were present, the habitat was included.
4. Frequency of use must be "frequently," "very rarely," or "occasionally" with appropriate snail species in at least one survey.

The main criteria were presence or absence of data, presence or absence of S. haematobium bearing snails, transmission potential, frequency of use, and resurvey data.

Processing of Tanzania 001 Data

Below, we have listed the different stages in data processing undertaken for this study. In addition, we briefly refer to some questions associated with the data that relate to our use of them.

1. State 1 looked at raw data.

   a. Printed data in readable form. In order to use computer tape efficiently, the data were coded on the tape by the project in a very compact form. Before using the data in computations or listing it in tables, it was put into a less compact, more usable form via several computer programs.

   b. tried to interpret the meaning of each item of data.

   c. Resolved inconsistencies or vagueness of meanings of data. Coding sheets were a problem because of their incompleteness.

   d. Decided the relationship between separate files of data. Different files supposedly coded different data, but in one case, the 2-to-9-year-olds, the epidemiological data were in two separate files. One file contained data for all individuals in the project and the other file contained data for only 2-to-9-year-olds. However, there were discrepancies between supposedly identical data items for the same individual listed in the two different files. Moreover, some data were present in one file that were not present in the other file. The 2-to-9-year-old

file listed yearly egg counts for each individual, the entire population file listed such data at two-year intervals. A decision was later made to use the file containing the data for all individuals in the project; as a result, incidence for the 2-to 9-year-olds had to be computed over a two-year period.

e. Made a list of unresolvable problems in interpreting raw data. Requested clarification from individuals involved in the original project.

f. Eventually, it became clear that there was no way to resolve the basic problem of the relationship of habitat to household without a trip to Geneva to view the project maps.

2. Stage 2 summarized raw data.

a. Prevalence, egg count, migration distributions were calculated by sector, age, and sex for all individuals.

b. Incidence for 2-to-9-year-olds was calculated on the basis of the yearly data. The reexamination of individuals was not made at a fixed interval from the first one. Therefore, in calculating the incidence, a correction had to be made to standardize the interval.

c. Calculated geometric mean egg counts, and the worm loads predicted by various statistical distributions such as the negative binomial (with various coefficients) and the Poisson. These were plotted in various combinations to see whether the obtained relationships were similar to what would be predicted by theory. This was a preliminary step to using data other than the number of infected people in the regression equations which predict the incidence.

3. Stage 3 examined and duplicated maps in Geneva
4. State 4 read maps (time consuming). For each house we had to draw radii of various sizes. For each distance we read from the map which habitats were within the specified distance from each household. Looking at the computer output, we selected those habitats which were possible transmission sites. This was difficult to do because of the incomplete explanation on the coding sheets. These data were then keypunched on cards for input to the computer.
5. Stage 5 predicted incidence. Wrote data program to predict incidence from habitat volume, prevalence and egg count data. This program selects from the data on the tape the appropriate habitat data for each house, selects the individuals whose houses were plotted on the map (some data on the computer was for individuals living in houses not plotted on the maps), selects the 2-to-9-year-olds (since these are the only ones for which we can calculate incidence) and then matches individuals to the appropriate habitat data. From this we calculated for each age group in the 2-to-9-year category the total volume of habitat to which the group was exposed, the number infected, the mean egg counts and the geometric mean egg counts. This was used as input to the regression program which predicts incidence.
6. Stage 6 predicted prevalence. A computer program simulating the recursive prevalence equations was written. The input to this program was the age-specific incidence values calculated in Stage 5, the size of each age group and the age-specific volume or habitat.

For comparing prevalence predictions to observed data and also to summarize baseline data, we had to make some assumptions about the group in which to place individuals. There was some question in our minds from the data as to when "age" was recorded, at year of registration or at treatment. We assumed that age was recorded at treatment. Therefore, if someone had egg count data first reported for 1970, and was listed as 3 years old, we assumed that the individual was 3 years old in 1970. We feel comfortable with this assumption because in a number of instances we have 1968 egg count data but the individual was registered in 1969.

Appendix II

COMPUTER PROGRAM FOR USE OF MODEL AND EXAMPLES OF PRINT-OUT

The following computer program is written in Fortran IV for use on a DecSystem 10 Computer. It was run at the Brookings Institution's Computer Center. The program and model modifications are on tape and are available from the principal investigator. The following print-outs show the program and output for two cases: 1) using total eggs in the incidence equation, with migration and molluscicide; and 2) using number infected in the incidence equation with migration, molluscicide, and chemotherapy. The basic equation and variables are given in table 2 in the text. The flow diagram of the model logic is given in figure 13.

EXAMPLE 1.

```
C  PROGRAM USES TOTAL EGG COUNTS AND VOLUME IN INCIDENCE ESTIMATION.
C THIS MODIF INCORPORATES MIGRATION INTO THE MODEL
C THIS VERSION ASSUMES 2 CYCLES FOLLOWS. PRIOR TO 1970 PREDICTIONS.
C ALL REMAINING FOLLOWS (3 THRU 4) PRIOR TO 1972 PREDICTIONS
      DIMENSION PREV(9,25),VOL(9),POPUL(8),TIMF(8),PERIM(8),AGE(8),EGG(9
     1)
      DIMENSION VOLUM(8),POPULL(8),TIMFI(8),PREVI(8,45),EMIG(8),AI,PE(8)
      DIMENSION AI"FI(8),XIMIG(8),FRACE(8),PREVE(8),PPE/I(8),PCT1(8)
      DATA AGE /' 2 ',' 3 ',' 4 ',' 5 ',' 6 ',' 7 ',' 8 ',' 9 '/
      CALL IFYL(20,'TIMEIV.DAT')
      CALL OFIL(21,'TIMEOV.DAT')
      READ(20,1)((PREV(I,L),I=1,8)
      READ(20,1)(POPUL(I),I=1,8)
      READ(20,1)(VOLUM(I),I=1,8)
      READ(20,1)(FRACE(I),I=1,8)
      READ(20,1)(PREVE(I),I=1,8)
      READ(20,1)(XIMIG(I),I=1,8)
      READ(20,1)(PREVI(I),I=1,8)
      WRITE(5,24)
24    FORMAT(/' NOTE--UNCORRECTED VALUES CALCULATED PERMIT TIMING MIGRAT
     LION INTO ACCOUNT.  THAT IS, IT IS THE PREVALENCE RATES BASED ON THE PREVIOU
     2S YEARS PREDICTIONS--CORRECTED VALUES USED FOR NEXT CYCLE PREDICT')
      WRITE(5,82)(FRACE(I),I=1,8)
82    FORMAT(/' AGE SPECIFIC FRACTION OF POPULATION THAT EMIGRATES'/1X,8
     1F8.5)
      WRITE(5,83)(PREVE(I),I=1,8)
83    FORMAT(/' PREVALENCE IN THE EMIGRANTS--AGE SPECIFIC'/1X,8F8.5)
      WRITE(5,84)(XIMIG(I),I=1,8)
84    FORMAT(/' AGE SPECIFIC SIZE OF POPULATION THE EMIGRATES'/1X,8F8.1
     1)
      WRITE(5,85)(PREVI(I),I=1,8)
85    FORMAT(/' PREVALENCE IN EMMIGRANTS--AGE SPECIFIC'/1X,8F8.5)
1     FORMAT(8F)
2     FORMAT(4F)
      WRITE(5,80)
      WRITE(21,24).
80    FORMAT(/' SECTOR 4. USING VOLUME OF MALIGNS WITHIN 1/2 MILE    */
     1" AND TOTAL EGG COUNT TO PREDICT A"/" EQUATION" EQUALS:
     2is=18.213+0.45137*LOG TOTAL EGG-1.0731*LOG VOLUME"/"       SQT FOR
     3M'S PREDICT-D FROM THE EQUA  LOG EGG = (LOG PREV+5.3158)/0.9096")
      WRITE(5,81)VOLUM
      WRITE(21,81)VOLUM
81    FORMAT(/' VOLUME VALUES'/(1X,4M14.4))
      E=0.30
      W=1966
      DO 50 J=1,20
      W=W+2   0
      GO TO (1/,11)J
      GO TO 1M
11    READ(20,2)(VOLUM(I),I=1,8)
      WRITE(5,87)W
87    FORMAT(/' VOLUSCICIDING ASSUMED TO OCCUR BEFORE USING',I5,' PREVA
     1LENCE VALUES TO MAKE PREDICTION FOR TWO YEARS HENCE.')
      WRITE(21,87)W
      WRITE(21,81)(VOLUM(I),I=1,8)
10    WRITE(21,5)W
      WRITE(21,6)
6     FORMAT(29X,"UNCORRECTED",6X,"CORRECTED")
      WRITE(21,4)
5     FORMAT(//'1',I5)
4     FORMAT(1X,"AGE",5X,"NUMBER SICK",7X,"POPULATION",1X,"PREVALENCE",4
     1X,"TOTAL EGG",11X,"NUMBER SICK",7X,"POPULATION",4X,"PREVALENCE",4X
     2,"TOTAL EGG")
      DO 40 I=1,8
      XIMF(I)=POPUL(I)*PREV(I,J)
      POPULL(I)=POPUL(I)
      XIMFI(I)=XIMF(I)
      PREVI(I,J)=PREV(I,J)
      EMIG(I)=FRACE(I)*POPUL(I)
      XIMFE(I)=PREVE(I)*EMIG(I)
      XIMFI(I)=PREVI(I)*XIMIG(I)
      POPUL(I)=POPUL(I)-EMIG(I)+XIMIG(I)
      XIMF(I)=XIMF(I)-XIMFE(I)+XIMFI(I)
      PREV(I,J)=XIMF(I)/POPUL(I)
      XIMEAN=(ALOG(PREV(I,J))+5.3158)/0.9096
      EGG1(I)=EXP(XIMEAN)
      EGG1(I)=EGG1(I)*POPULL(I)
      AIMEAN=(ALOG(PREV(I,J))+5.3158)/0.9096
      EGG(I)=EXP(XIMEAN)
      EGG(I)=EGG(I)*POPUL(I)
      WRITE(21,3)AGE(I),XIMF1(I),PUPULL(I),PREVI(I,J),EGG1(I),XIMF(I),PO
     1PUL(I),PREV(I,J),EGG(I)
3     FORMAT(1X,A3,7X,F8.4,7X,F8.4,7X,F8.4,7X,F12.4,4X,F8.4,7X,F8.4,7X,
     1F8.4,7X,F12.4)
      A=-18.213+(0.45137*ALOG(EGG(I)))+(1.0731*ALOG(VOLUM(I)))
      (F(J.EQ.1)ASTORE=A
      POPUL(I)=1.01*POPUL(I)
      XIMIG(I)=1.01*XIMIG(I)
      CONTINUE
      C=A/(1+E)
      T=EXP(-(A+C))
      PREV(I,J+1)=((PREV(I,J)-C)*T)+C
40    CONTINUE
50    CONTINUE
      WRITE(5,9)ASTORE
9     FORMAT(/' THE LOG OF THE VALUE OF A FOR THE OLDEST AGE IN THE FIRST
     1 YEAR IS'/1X,F10.4/)
      DO 60 I=1,8
60    WRITE(5,8)(PREV(I,J),J=1,5)
8     FORMAT(7F11.4)
      END FILE 21
      CALL EXIT
      END
```

EXAMPLE 1.

```
00190    C  PROGRAM USES TOTAL EGG COUNTS AND VOLUME IN INCIDENCE ESTIMATION.
00200    C THIS MODIF INCORPORATES MIGRATION INTO THE MODEL
00250    C THIS VERSION ASSUMES 2 CYCLES FOLLOWS PRIOR TO 1970 PREDICTIONS.
00255    C ALL REMAINING FOLLOWS (3 THRU 4) PRIOR TO 1972 PREDICTIONS
00300         DIMENSION PREV(8,25),VOL(8),POPUL(8),XIMF(8),PERI(8),ACK(8),EGG(8
                1)
00400         DIMENSION VOLUM(8),POPULL(8),XIMFI(8),PREV1(8,45),EMIG(8),XIMFE(8)
00500         DIMENSION AIMFI(8),XIMIG(8),FRACE(8),PREVE(8),PREVI(8),FCTL(8)
00600         DATA AGE /' 2 ',' 3 ',' 4 ',' 5 ',' 6 ',' 7 ',' 8 ',' 9 '/
00700         CALL IFIL(20,'TIMZIV.DAT')
00800         CALL OFIL(21,'TIMZOV.DAT')
00900         READ(20,1)((PREV(I,L),I=1,8)
01000         READ(20,1)(POPUL(I),I=1,8)
01100         READ(20,2)(VOLUM(I),I=1,8)
01105         READ(20,1)(FRACE(I),I=1,8)
01110         READ(20,1)(PREVE(I),I=1,8)
01115         READ(20,1)(XIMIG(I),I=1,8)
01120         READ(20,1)(PREVI(I),I=1,8)
01125         WRITE(5,24)
01130    36   FORMAT(/' NOTE--UNCORRECTED VALUES CALCULATED BEFORE TAKING MIGRAT
01135         LION INTO ACCOUNT.  THAT IS, IT IS THE RESULTS BASED ON THE PREVIOU
01140         IS YEARS PREDICTIONS--CORRECTED VALUES USED FOR NEXT CYCLE PREDICT
01145         WRITE(5,62)(FRACE(I),I=1,8)
01150    62   FORMAT(/' AGE SPECIFIC FRACTION OF POPULATION THAT EMIGRATES'/1X,8
01155         1F8.5)
01160         WRITE(5,83)(PREVE(I),I=1,8)
01165    83   FORMAT(/' PREVALENCE IN THE EMIGRANTS--AGE SPECIFIC'/1X,8F8.5)
01170         WRITE(5,84)(XIMIG(I),I=1,8)
01175    84   FORMAT(/' AGE SPECIFIC SIZE OF POPULATION THE IMMIGRATES'/1X,8F8.1
01180         1)
01185         WRITE(5,85)(PREVI(I),I=1,8)
01190    85   FORMAT(/' PREVALENCE IN IMMIGRANTS--AGE SPECIFIC'/1X,8F8.5)
01200    1    FORMAT(8F)
01300    2    FORMAT(8F)
01400         WRITE(5,80)
01420         WRITE(21,80)
01500    80   FORMAT(/' SETUP 4. USING VOLUME OF HABITATS WITHIN 1/2 'CLF    '/
01600         1' AND TOTAL EGG COUNT TO PREDICT #'/' EQUATION EQUALS:
01700         2ii=-18.213+0.45137*LOG TOTAL EGG-1.6731*LOG VOLUME'/'       EGG COM
01800         3ES PREDICTED FROM THE EQN:  LOG EGG = (LOG PREV-5.3168)/0.9056'/)
01900         WRITE(5,81)VOLUM
01970         WRITE(21,81)VOLUM
02000    81   FORMAT(/' VOLUME VALUES'/(1X,4F14.4))
02100         E=0.00
02200         M=1966
02300         DO 50 J=1,20
02400         M=M+2
02410         GO TO (10,11)J
02415         GO TO 10
02420    11   READ(20,2)(VOLUM(I),I=1,8)
02425         WRITE(5,87)M
02430    87   FORMAT(/' VOLLUSCICIDING ASSUMED TO OCCUR BEFORE USING',I5,' PREVA
02435         1LENCE VALUES TO MAKE PREDICTION FOR TWO YEARS HENCE.')
02440         WRITE(21,87)M
02445         WRITE(21,81)(VOLUM(I),I=1,8)
02500    10   WRITE(21,5)M
02525         WRITE(21,6)
02530    6    FORMAT(29X,'UNCORRECTED',6X,'CORRECTED')
02600         WRITE(21,6)
02700    5    FORMAT(///7(,I5))
02800    4    FORMAT(1X,'AGE',5X,'NUMBER SICK',7X,'POPULATION',1X,'PREVALENCE',4
02900         1X,'TOTAL EGG',11X,'NUMBER SICK',3X,'POPULATION',4X,'PREVALENCE',4
02905         2,'TOTAL EGG')
03000         DO 40 I=1,8
03100         XIMF(I)=POPUL(I)*PREV(I,J)
03105         POPUL1(I)=POPUL(I)
03110         AIMFI(I)=XIMF(I)
03115         PREVI(I,J)=PREV(I,J)
03120         EMIG(I)=FRACE(I)*POPUL(I)
03125         XIMFE(I)=PREVE(I)*EMIG(I)
03130         XIMFI(I)=PREVI(I)*XIMIG(I)
03135         POPUL(I)=POPUL(I)-EMIG(I)-XIMIG(I)
03140         XIMF(I)=XIMF(I)-XIMFE(I)+XIMFI(I)
03145         PREV(I,J)=XIMF(I)/POPUL(I)
03150         XIMEAN=(ALOG(PREV(I,J)))-5.3168)/0.9056
03155         EGG1(I)=EXP(XIMEAN)
03160         EGG1(I)=EGG1(I)*POPUL1(I)
03200         XIMEAN=ALOG(PREV(I,J))-5.3168)/0.9056
03300         EGG(I)=EXP(XIMEAN)
03305         EGG(I)=EGG(I)*POPUL(I)
03400         WRITE(21,3)AGE(I),AIMFI(I),POPUL1(I),PREVI(I,J),EGG1(I),XIMF(I),PO
03405         1PUL(I),PREV(I,J),EGG(I)
03500    3    FORMAT(1X,A2,7X,F8.4,7X,F8.1,7X,F8.4,7X,F12.4,4X,F8.4,7X,F8.1,7X,
03505         1F8.4,7X,F12.4)
03600         A=-18.213+(0.45137*ALOG(EGG(I)))-(1.6731*ALOG(VOLUM(I)))
03700         IF(J.EQ.1)ASTORE=A
03800         POPUL(I)=1.04*POPUL(I)
03805         AI(IG(I)=1.015*XIMIG(I)
03900         A=EXP(A)
04000         C=A/(1+A)
04100         T=EXP(-(A+B))
04200         PREV(I,J+1)=((PREV(I,J)-C)**T)+C
04300    40   CONTINUE
04400    50   CONTINUE
04500         WRITE(5,9)ASTORE
04600    9    FORMAT(/' THE LOG OF THE VALUE OF A FOR THE OLDEST AGE IN THE FIRST
04605         1 YEAR IS'/1X,F10.4/)
04700         DO 60 I=1,8
04800    60   WRITE(5,8)(PREV(I,J),J=1,5)
04900    8    FORMAT(7F11.4)
05000         END FILE 21
05100         CALL EXIT
05200         END
```

SECTOR 4: USING VOLUME OF HABITATS WITHIN 1/2 MILE
AND TOTAL EGG COUNT TO PREDICT A
EQUATION EQUALS:A=-10.212+0.45137*LOG TOTAL EGG+1.0731*LOG VOLUME
EGG COUNTS PREDICTED FROM THE FORM: LOG EGG = (LOG PREV+5.3168)/0.99600

VOLUME VALUES
243056.5510 221320.9610 264836.7120 374121.1220
336056.5900 261211.7310 343975.8720 361419.5190

MOLLUSCICIDING ASSUMED TO OCCUR BEFORE USING 1968 PREVALENCE VALUES TO MAKE PREDICTION FOR TWO YEARS HENCE.

VOLUME VALUES
100722.3750 87237.8603 109955.2740 143991.5090
130596.3920 114025.9010 136402.6000 156759.5010

1968

AGE	UNCORRECTED NUMBER EXP.	UNCORRECTED POPULATION	UNCORRECTED PREVALENCE	UNCORRECTED TOTAL EGG	CORRECTED NUMBER EXP.	CORRECTED POPULATION	CORRECTED PREVALENCE	CORRECTED TOTAL EGG
2	1.9998	18.0000	0.1111	561.4169	1.9999	23.0002	0.0869	547.4
490								
3	7.0992	24.0000	0.3333	2514.1055	6.9990	21.9994	0.3181	2189.2
541								
4	10.0008	27.0000	0.3704	3177.4788	10.0701	27.2082	0.3701	3199.2
526								
5	15.0016	32.0000	0.4688	4683.1661	18.0016	35.0000	0.5143	5915.7
856								
6	15.9993	29.0000	0.5517	5295.8167	18.9993	30.9995	0.6129	6357.1
800								
7	15.0000	25.0000	0.6000	5008.0579	13.0010	23.0000	0.5652	4714.1
240								
8	15.0000	25.0000	0.6000	5008.0579	17.0000	27.0000	0.6296	5703.9
875								
9	19.9992	26.0000	0.7692	6819.8226	17.0004	23.0012	0.7391	5799.9
867								

MOLLUSCICIDING ASSUMED TO OCCUR BEFORE USING 1970 PREVALENCE VALUES TO MAKE PREDICTION FOR TWO YEARS HENCE.

VOLUME VALUES
88467.1926 71787.5237 91321.5747 132124.6800
127365.8130 92425.9014 126554.7720 134149.4490

1970

AGE	UNCORRECTED NUMBER EXP.	UNCORRECTED POPULATION	UNCORRECTED PREVALENCE	UNCORRECTED TOTAL EGG	CORRECTED NUMBER EXP.	CORRECTED POPULATION	CORRECTED PREVALENCE	CORRECTED TOTAL EGG
2	2.8451	23.3452	0.1219	696.3585	2.8451	27.9556	0.1021	791.7
486								
3	7.6278	22.3292	0.3416	2403.4379	6.6977	20.6359	0.3245	2999.1
290								
4	11.2861	27.6163	0.4087	3622.2731	11.3492	27.8953	0.4067	3642.0
595								
5	20.4633	35.5250	0.5760	6503.5237	23.4132	38.3777	0.6100	7030.3
074								
6	19.8723	31.4645	0.6316	6769.0353	22.9173	37.1210	0.6057	7757.0
663								
7	13.4474	23.3450	0.5760	4470.9165	11.7432	21.7210	0.5406	3978.7
573								
8	17.6485	27.4050	0.6440	5935.3017	19.5973	29.7534	0.6587	6615.1

Example 1 (continued)

C-11

507								
9	17.1312	23.3462	0.7338	5029.1101	14.5556	20.7706	0.7008	4037.9
444								

1973

AGE	UNCORRECTED NUMBER EXP.	POPULATION	PREVALENCE	TOTAL EGG	CORRECTED NUMBER EXP.	POPULATION	PREVALENCE	TOTAL EGG
2	3.8513	28.2744	0.1362	1104.0721	3.8511	32.3417	0.1191	1368.9
154								
3	7.0594	20.9465	0.3370	2221.2818	6.1965	19.5151	0.3170	1934.3
975								
4	12.1698	28.2229	0.4312	3927.5027	12.2772	29.7953	0.4120	3945.4
719								
5	24.8030	38.9554	0.6367	9331.6789	27.7095	41.6718	0.6649	9748.5
285								
6	23.3956	33.9253	0.6896	7923.6665	26.4063	35.8456	0.7368	9034.2
835								
7	11.0508	22.0469	0.5421	3948.6073	10.4848	20.7291	0.5058	3439.6
815								
8	20.0134	29.7941	0.6717	6759.8639	21.9121	31.6930	0.6914	7423.2
469								
9	14.6141	21.0921	0.6932	4952.1900	12.4019	18.8699	0.6572	4179.5
959								

1974

AGE	UNCORRECTED NUMBER EXP.	POPULATION	PREVALENCE	TOTAL EGG	CORRECTED NUMBER EXP.	POPULATION	PREVALENCE	TOTAL EGG
2	5.1623	32.8294	0.1572	1501.9141	5.1523	36.5072	0.1414	1495.6
350								
3	6.5196	19.8079	0.3291	2040.4221	5.6941	18.5973	0.3062	1774.9
736								
4	13.0166	28.8212	0.4516	4220.8275	13.0589	29.9782	0.4358	4237.1
516								
5	29.8484	42.2969	0.6820	9759.3313	31.7033	44.9817	0.7049	10766.1
026								
6	26.6494	36.3933	0.7325	9091.7449	29.7864	38.2651	0.7784	10214.1
954								
7	10.7504	21.6390	0.5110	3530.4718	9.4765	19.9683	0.4746	3098.5
917								
8	22.2730	32.1684	0.6924	7546.5752	24.1233	34.0197	0.7091	6191.5
789								
9	12.5654	19.1530	0.6561	4233.9197	10.6554	17.7529	0.6182	3571.7
995								

Example 1 (continued)

EXAMPLE 2.

```
00100    C  PROGRAM USES A INFECTEDAND VOLUME IN INCIDENCE ESTIMATION.
00200    C THIS MODIF ALSO INCORPORATES MIGRATION INTO THE MODEL.
00300    C THIS VERSION ALSO ASSUMES CHEMOTHERAPY OCCURS BEFORE MIGRATION.
00400          DIMENSION PREV(8,25),VOL(8),POPUL(8),XINF(8),PERIM(8),AGE(8)
00500          DIMENSION VOLUM(8),POPUL1(8),XINF1(8),PREV1(8,25),EMIG(8),XINFL(8)
00600          DIMENSION XINF1(8),XIMIG(8),FRACE(8),PREVE(8),PREV1(8),XINF2(8)
00700          DATA AGE /' 2 ',' 3 ',' 4 ',' 5 ',' 6 ',' 7 ',' 8 ',' 9 '/
00800          CALL IFIL(20,"TANZIV.DAT")
00900          CALL OFIL(21,"TAMZOV.DAT")
01000          READ(20,1)(PREV(I,1),I=1,8)
01100          READ(20,1)(POPUL(I),I=1,8)
01200          READ(20,2)(VOLUM(I),I=1,8)
01300          READ(20,1)(FRACE(I),I=1,8)
01400          READ(20,1)(PREVE(I),I=1,8)
01500          READ(20,1)(XIMIG(I),I=1,8)
01600          READ(20,1)(PREV1(I),I=1,8)
01700          WRITE(5,86)
01800    86    FORMAT(/' NOTE--UNCORRECTED VALUES CALCULATED BEFORE TAKING MIGRAT
01900         1ION INTO ACCOUNT.  THAT IS, IT IS THE RESULTS BASED ON THE PREVIOU
02000         2S YEARS PREDICTIONS--CORRECTED VALUES USED FOR NEXT CYCLE PREDIC')
02100          WRITE(5,82)(FRACE(I),I=1,8)
02200    82    FORMAT(/' AGE SPECIFIC FRACTION OF POPULATION THAT 'MIGRATES'/1X,8
02300         1F8.5)
02400          WRITE(5,83)(PREVE(I),I=1,8)
02500    83    FORMAT(/' PREVALENCE IN THE EMIGRANTS--AGE SPECIFIC'/1X,8F8.5)
02600          WRITE(5,84)(XIMIG(I),I=1,8)
02700    84    FORMAT(/' AGE SPECIFIC SIZE OF POPULATION THE IMMIGRATES'/1X,8F8.1
02800         1)
02900          WRITE(5,85)(PREV1(I),I=1,8)
03000    85    FORMAT(/' PREVALENCE IN IMMIGRANTS--AGE SPECIFIC'/1X,8F8.5)
03100    1     FORMAT(8F)
03200    2     FORMAT(4F)
03300          WRITE(5,80)
03400          WRITE(21,80)
03500    80    FORMAT(/' SECTOR 4. USING VOLUME OF HABITATS WITHIN 1/2 MILE    '/
03600         1' AND NO. INFECTED TO PREDICT A'/' EQUATION FOR A=-13.374+(0.35957
03700         2*ALOG(XINF(I)))+(0.91309*ALOG(VOLUM(I)))')
03800          WRITE(5,81)VOLUM
03900          WRITE(21,81)VOLUM
04000    81    FORMAT(/' VOLUME VALUES'/(1X,4F14.4))
04100          B=0.09
04200          M=1966
04300          DO 50 J=1,20
04400          MM=2
04500          GO TO (10,16,17)J
04600          GO TO 10
04700    16    WRITE(21,89)M
04800    89    FORMAT(//' # INFECTED MULTIPLIED BY 0.6 FOR CHEMOTHERAPY IN YEAR',
04900         1I5,' BEFORE CORRECTING FOR MIGRATION. I.E. CURE RATE=40%')
05000          GO TO 10
05100    17    WRITE(21,88)M
05200    88    FORMAT(' # INFECTED MULTIPLIED BY 0.75 FOR CHEMO. IN YEAR',I5,' BE
05300         1FORE CORRECTING FOR MIGRATION. I.E. EFFECTIVE CURE RATE=0.25')
05400    10    GO TO (11,11)J
05500          GO TO 15
05700    11    READ(20,2)(VOLUM(I),I=1,8)
05800          WRITE(5,87)M
05900    87    FORMAT(//' RECIRCULATING. CHANGES ASSUMED TO OCCUR BEFORE USING',
06000         1I5,' PREV. VALUES TO PREDICT PREVALENCE FOR TWO YEARS HENCE')
06200          WRITE(21,87)M
06300          WRITE(21,81)(VOLUM(I),I=1,8)
06400       15    WRITE(21,5)M
06500          WRITE(21,6)
06600    6     FORMAT(29X,"UNCORRECTED",15X,"CHEMOTHERAPY",10X,"CHEMOTHERAPY AND
06700         1MIGRATION CORRECTION"/A0X,"USE TO MAKE NEXT CYCLE PREDIC.")
06800          WRITE(21,4)
06900    5     FORMAT(//7X,I5)
07000    4     FORMAT(1X,"AGE",5X,"NUMBER SICK",3X,"POPULATION",4X,"PREVALENCE",7
07100         1X,"NUMBER SICK",8X,"NUMBER SICK",3X,"POPULATION",4X,"PREVALENCE")
07200          DO 40 I=1,8
07300          XINF(I)=POPUL(I)*PREV(I,J)
07400          POPUL1(I)=POPUL(I)
07500          XINF1(I)=XINF(I)
07525          XINF2(I)=XINF(I)
07600          PREV1(I,J)=PREV(I,J)
07700          GO TO (12,13,14)J
07750          GO TO 12
07800    13    XINF2(I)=XINF(I)*0.6
07900          GO TO 12
08000    14    XINF2(I)=XINF(I)*0.75
08100    12    EMIG(I)=FRACE(I)*POPUL(I)
08200          XINFE(I)=PREVE(I)*EMIG(I)
08300          XINF1(I)=PREV1(I)*XIMIG(I)
08400          POPUL(I)=POPUL(I)-EMIG(I)+XIMIG(I)
08500          XINF(I)=XINF2(I)-XINFE(I)+XINF1(I)
08600          PREV(I,J)=XINF(I)/POPUL(I)
08700          WRITE(21,7)AGE(I),XINF1(I),POPUL1(I),PREV1(I,J),XINF2(I),XINF(I),P
08800         1OPUL(I),PREV(I,J)
08900    3     FORMAT(1X,A3,6X,F9.4,5X,F9.4,4X,F9.4,10X,F8.4,10X,F9.4,7X,F9.4,7X,
09000         1F9.4)
09100          A=-13.374+(0.35957*ALOG(XINF(I)))+(0.91309*ALOG(VOLUM(I)))
09200          IF(J.EQ.1)ASTORE=A
09300          POPUL(I)=1.015*POPUL(I)
09400          XIMIG(I)=1.015*XIMIG(I)
09500          A=EXP(A)
09600          C=A/(B+A)
09700          T=EXP(-(A+B))
09800          PREV(I,J+1)=((PREV(I,J)-C)*T)+C
09900    40    CONTINUE
10000    50    CONTINUE
10100          WRITE(5,9)ASTORE
10200    9     FORMAT(/' THE LOG OF THE VALUE OF A FOR THE OLDEST AGE IN THE FIRST
10300         1 YEAR IS'/1X,F10.4/)
10400          DO 60 I=1,8
10500    60    WRITE(5,8)(PREV(I,J),J=1,6)
10600    8     FORMAT(7F11.4)
10700          END FILE 21
10800          CALL EXIT
10900          END
```

Example 2 (continued)

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SECTOR 4. USING VOLUME OF HABITATS WITHIN 1/2 MILE
AND NO. INFECTED TO PREDICT 4
EQUATION FOR 4=-13.374+(0.35957*ALOG(XINF(I)))+(0.9130**ALOG(VOLUM(I)))

VOLUME VALUES
   243056.5510   221328.9610   264830.7120   374121.1220
   336056.5900   261211.7310   343975.8720   361418.5190


MOLLUSCICIDING. CHANGES ASSUMED TO OCCUR BEFORE USING 1968 PREV. VALUES TO PREDICT PREVALENCE FOR TWO YEARS HENCE

VOLUME VALUES
   100722.1750    87237.8603   109955.2740   163991.5890
   130598.3920   114825.9010   136402.6800   156759.5810
```

1968

AGE	UNCORRECTED NUMBER INF.	POPULATION	PREVALENCE	CHEMOTHERAPY NUMBER INF	CHEMOTHERAPY AND MIGRATION CORRECTION USE TO MAKE NEXT CYCLE PREDIC. NUMBER INF.	POPULATION	PREVALENCE
2	1.9998	18.0000	0.1111	1.9998	1.9998	23.0002	0.0869
3	7.9992	24.0000	0.3333	7.9992	6.9990	21.9992	0.3181
4	10.0008	27.0000	0.3704	10.0008	10.0701	27.2082	0.3701
5	15.0016	32.0000	0.4688	15.0016	18.0016	35.0000	0.5143
6	15.9993	29.0000	0.5517	15.9993	18.9993	30.9995	0.6129
7	15.0000	25.0000	0.6000	15.0000	13.0010	23.0000	0.5653
8	15.0000	25.0000	0.6000	15.0000	17.0000	27.0000	0.6296
9	19.9992	26.0000	0.7692	19.9992	17.0004	23.0012	0.7391

8 INFECTED MULTIPLIED BY 0.6 FOR CHEMOTHERAPY IN YEAR 1970 BEFORE CORRECTING FOR MIGRATION. I.E. CURE RATE=40%

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MOLLUSCICIDING. CHANGES ASSUMED TO OCCUR BEFORE USING 1970 PREV. VALUES TO PREDICT PREVALENCE FOR TWO YEARS HENCE

VOLUME VALUES
    88467.1926    71737.5227    91321.6747   132124.8800
   127365.8130   92425.9014   126554.7720   134140.4490
```

1970

AGE	UNCORRECTED NUMBER INF	POPULATION	PREVALENCE	CHEMOTHERAPY NUMBER INF.	CHEMOTHERAPY AND MIGRATION CORRECTION USE TO MAKE NEXT CYCLE PREDIC. NUMBER INF	POPULATION	PREVALENCE
2	3.3130	23.3452	0.1419	1.9878	1.9878	27.8566	0.0714
3	7.9299	22.3292	0.3551	4.7579	3.8274	20.6169	0.1855
4	11.6203	27.6163	0.4208	6.9722	7.0353	27.8058	0.2530
5	20.5874	35.5250	0.5795	12.3524	15.3023	38.3797	0.3987
6	19.9978	31.4645	0.6356	11.9987	15.0437	33.4240	0.4501
7	13.6096	23.3450	0.5830	8.1657	8.4616	21.7210	0.2975
8	17.7570	27.4050	0.6479	10.6542	12.6030	29.3538	0.4293
9	17.1711	23.3462	0.7355	10.3026	7.7270	20.7704	0.3720

8 INFECTED MULTIPLIED BY 0.75 FOR CHEMO. IN YEAR 1972 BEFORE CORRECTING FOR MIGRATION. I.E. EFFECTIVE CURE RATE=0.75

1972

AGE	UNCORRECTED NUMBER INF	POPULATION	PREVALENCE	CHEMOTHERAPY NUMBER INF	CHEMOTHERAPY AND MIGRATION CORRECTION USE TO MAKE NEXT CYCLE PREDIC. NUMBER INF	POPULATION	PREVALENCE
2	3.4410	28.2744	0.1217	2.5807	2.5807	32.3447	0.0798
3	4.6414	20.9465	0.2216	3.4811	2.6091	19.5151	0.1336
4	8.5890	28.2229	0.3043	6.4417	6.4972	28.3953	0.2289
5	18.3211	38.9554	0.4703	13.7408	16.6444	41.6718	0.1994
6	17.1469	33.9253	0.5054	12.8602	15.9509	35.8456	0.4450
7	7.4871	22.0468	0.3396	5.6153	4.1493	20.7281	0.2002
8	14.4130	29.7941	0.4838	10.8097	12.7087	31.6930	0.4010
9	9.0474	21.0821	0.4292	6.7856	4.5734	18.8499	0.2424

1974

AGE	UNCORRECTED NUMBER INF	POPULATION	PREVALENCE	CHEMOTHERAPY NUMBER INF.	CHEMOTHERAPY AND MIGRATION CORRECTION USE TO MAKE NEXT CYCLE PREDIC. NUMBER INF	POPULATION	PREVALENCE
2	4.4069	32.8299	0.1342	4.4069	4.4069	36.5022	0.1207
3	3.3764	19.8079	0.1705	3.3764	2.5509	18.5973	0.1372
4	8.1415	28.8212	0.2825	8.1415	8.1937	29.9782	0.2828
5	20.0393	42.2969	0.4738	20.0393	22.9003	44.8917	0.5102
6	18.3158	36.3933	0.5034	18.3158	21.4528	38.2551	0.5606
7	5.2320	21.0390	0.2487	5.2320	3.9582	19.9683	0.1982
8	14.8712	32.1684	0.4623	14.8712	14.7215	34.0187	0.4915
9	5.9497	19.1530	0.3106	5.9497	4.0496	17.2529	0.2347