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"Global warming and potential changes in host-parasite and disease-vector relationships"

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These are preliminary lecture notes, intended only for distribution to participants.

Global warming and potential changes in host-parasite
and disease-vector relationships.

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A. Introduction.

Parasitology has always been a discipline where purely academic studies of the evolution of parasites and their life cycles have progressed as a necessary compliment to the study of the pathology and control of the major tropical diseases of humans and their livestock. Indeed the most striking feature of parasitology is the diversity of parasites in the warm tropical regions of the world and the truly frightening levels of debilitation and misery they cause. Determining how long-term climatic changes will effect the distributions of different parasites and pathogens is at first sight a daunting task that almost defies quantification. Nevertheless as parasitologists have always been concerned with the influence of climatological effects on different parasite species, it is possible to begin to make some qualitative speculations on the ways that global warming might effect the distributions of some specific tropical diseases. Similarly, the study of parasite population dynamics has developed within a solid theoretical framework (Anderson & May 1979; May & Anderson 1979). This permits the development of quantitative speculation in more general studies concerned with how parasite-host interactions may respond to perturbation.

This paper will try to address both the more general questions about the response of parasite-host systems to long

term climatic changes and the more specific response of one particular pathogen to the changes in climate predicted for the next one hundred years. The paper initially provides a brief overview of parasite-host population dynamics with an emphasis on those features of a parasite's life history that are most susceptible to climatic change. Potential changes in the distribution of several species of *Trypanosoma*, the causitive agent of sleeping sickness in Africa, are then examined using predictions of climate change and quantitative estimates of the climatic tolerance of its vector, the tsetse fly (*Glossina* spp.). The factors determining the structure of whole communities of parasites in individual host populations are then briefly examined, while the final discussion pulls together the implications for conservation.

Macroparasites and microparasites.

Current estimates suggest that parasitism of one form or another may be the most common form of life style in at least three of the five major phylogenetic kingdoms (May 1988; Toft 1986). The enormous array of pathogens that infect humans and other animals may be conveniently divided on epidemiological grounds into microparasites and macroparasites (Anderson & May 1979; May and Anderson 1979). The former include the viruses, bacteria and fungi and are characterized by their ability to reproduce directly within individual hosts, their small size and relatively short duration of infection and the production of an

immune response in infected and recovered individuals. Mathematical models examining the dynamics of these pathogens divide the host population into susceptible, infected and recovered classes. In contrast, the macroparasites (the parasitic helminths and arthropods) do not multiply directly within an infected individual, but instead produce infective stages which usually pass out of the host before transmission to another definitive host. Macroparasites tend to produce a limited immune response in infected hosts, they are relatively long-lived and usually visible to the naked eye. Mathematical models of the population dynamics of macroparasites have to consider the statistical distribution of parasites within the host population.

Direct and indirect life cycles.

A second division of parasite life histories distinguishes between those species with monoxenic life cycles, and those with heteroxenic life cycles. The former produce infective stages which can directly infect another susceptible definitive host individual, the latter utilize a number of intermediate hosts or vectors in their transmission between definitive hosts. The evolution of complex heteroxenic life cycles permits parasite species to colonize hosts from a wide range of ephemeral and permanent environments, while also permitting them to exploit host populations at lower population densities than would be possible with simple direct transmission (Anderson 1988; Dobson

1988; Mackiewicz 1988; Shoop 1988). However, parasites with heteroxenic life cycles are essentially constrained to those areas where the distributions of all the hosts in the life cycle overlap. Changes in the distribution of the these host species due to climatic changes, will therefore be very important in determining the areas where parasites may continue to persist and areas where parasites may be able to colonize new hosts.

Aquatic and terrestrial hosts.

Climatic changes are likely to have different effects on aquatic and terrestrial environments (Harte & Torn, this symposium). The heteroxenic life cycles of some parasite species often allow them to sequentially utilize hosts from either type of habitat. It is thus important to determine the different responses of the terrestrial and aquatic stages of a parasite's life cycle to climatic change. To examine this particular question and other responses of parasites to climatic change we require a quantitative framework within which to discuss parasite life history strategies.

B. Parasite life history strategies.

The complexities of parasite host population dynamics may be reduced by the derivation of expressions which describe the most important epidemiological features of a parasite's life cycle (Anderson & May 1979; May & Anderson 1979; Dobson 1988). Three

parameters are important in describing the dynamics of a pathogen: the rate it will spread in a population, the threshold number of hosts required for the parasite to establish and the mean levels of infection of the parasite in the host population.

Basic reproductive rate of a parasite, Ro.

The basic reproductive rate, R_0, of a microparasite may be formally defined as the number of new infections that a solitary infected individual is able to produce in a population of susceptible hosts (Anderson & May 1979). In contrast, Ro for a macroparasite is defined as the number of daughters that are established in a host population following the introduction of a solitary fertilized female worm. In both cases the resultant expression for Ro usually consists of a term for the rates of parasite transmission, divided by an expression for the rate of mortality of the parasite in each stage in the life cycle (Table 1). Increases in host population size or rates of transmission tend to increase Ro, while increases in parasite virulence or other sources of parasite mortality tend to reduce the spread of the pathogen through the population.

Thresholds for establishment, H_T.

The threshold for establishment of a parasite, H_T is the minimum number of hosts required to just sustain an infection of the pathogen. An expression for H_T, may be obtained by rearranging the expression for Ro to find the population density

where Ro equals unity. This may be done for both micro- and macroparasites with either simple or complex life cycles (Table 1). The resultant expressions suggest that changes in the parameters that tend to increase Ro, tend to reduce H $_T$ and *vice versa*. Thus more pathogenic species require larger populations to sustain them, while reductions in the mortality rate of transmission stages may allow parasites to maintain infections in populations previously to small to sustain them.

Mean prevalence and burden at equilibrium.

It is also possible to derive expressions for the levels of prevalence (proportion of the hosts infected) and incidence (mean parasite burden) of parasites in the host populations. In general, parameters which tend to increase Ro, tend also to give increases in the proportion of hosts infected by a microparasite and increases in the mean levels of abundance of any particular macroparasite (Anderson & May 1979; May & Anderson 1979; Dobson 1988). Most important, increases in the size of the host population usually lead to increases in the prevalence and incidence of the parasite population (Figure 1).

Having outlined the expressions which characterize the most important features of a parasites interaction with its host at the population level, it is possible to use these expressions to ascertain how parasites with different types of life cycle will respond to long-term climatic changes. This may best be

undertaken by determining which stages of the life cycles are most susceptible to climatic variation and quantifying the response of these stages to climatic change.

C.   Effect of temperature on parasite transmission rates.

The physiology of adult parasites is intimately linked with the physiology of their hosts. Providing the hosts can withstand environmental changes, then it seems unlikely that the within-host component of the parasite life-cycle will be significantly effected. However, it is important to bear in mind that any form of increased stress upon the host may lead to increases in rates of parasite-induced host mortality (Esch, Gibbons & Bourque 1975). In the absence of data from the specific experimental studies which could throw considerable light on these relationships, this study will concentrate on the effect of changes in meteorological factors on the free-living infective stages of different groups of parasites.

Effect of temperature on parasites with aquatic transmission stages.

Several detailed laboratory studies have examined the effect of temperature on the transmission success of parasites with aquatic infective stages. The parasitic trematodes are probably the most important class of parasites to utilize an aquatic stage for at least part of their life cycle. The data presented in

figure 2 are for two different trematode groups: the echinostome species is a parasite of ducks, the schistosomes are some of the most important parasitic diseases of humans in the tropics. In both cases, increased temperature leads to increased mortality of the larval infective stages of the parasite. However, increased temperature also leads to increased infectivity of the larval stage. The interaction between larval infectivity and survival means that net transmission efficiency peaks at some intermediate temperature (Figure 2), but remains relatively efficient over a broad range of values (16 - 36 °C. for *Echinostoma liei* cercaria and 5 - 25° C. for *S.mansoni* miracidia). These synergistic interactions between the different physiological processes determining survival and infectivity allow the aquatic parasites to infect hosts at a relatively constant rate over the entire spectrum of water temperatures that they are likely to experience in their natural habitats (Evans 1985).

Effect of temperature on development rate in poikilothermic hosts.

The effect of temperature on the developmental rate of parasites in both aquatic and terrestrial hosts has been examined for several of the major parasites of humans in the tropics. In contrast to transmission efficiency, increases in temperature usually lead to reduced development times for parasites that utilize poikilothermic hosts (Figure 3). As with many physiological processes, a ten degree increase in temperature

seems to lead to a halving of the developmental time. This may allow parasite populations to build up very rapidly following increases in temperature.

Parasite populations in thermal cooling streams.

The expressions for Ro, and N_T, derived in the first part of this section suggest that increases in transmission efficiency and reductions in development time induced by temperature changes allow parasites to establish in smaller populations and grow at more rapid rates. This is observed to some extent in a pair of long-term studies which compared the parasite burdens of mosquito-fish (*Gambusia affinis*) populations in artificially heated and control sections of the Savannah river in North Carolina (Figure 4). The data for the trematode *Ornithodiplostomum ptychocheilus* show significant differences between heated and ambient sites during the earlier period of the study when temperature differences were most pronounced. Infection by the parasites start several months earlier each year in the thermally altered sites. However, infection rates decline in the summer in the artificially heated sites when populations of hosts decline in response to high water temperatures (Camp, Aho & Esch 1982). This effect may be compounded by the movement of the waterfowl that act as definitive hosts for the parasite. These birds tend to prefer the warmer water in winter and cooler water in the summer (Ref: Tracy's talk?). Similar, but less clearly defined patterns are observed in the data for *Diplostomum*

scheuringi from the same site (Aho, Camp & Esch 1982).

These studies illustrate the important role of host population density in the response of a parasite transmission to thermal stress, while also demonstrating the ability of parasites to capitilize on improved opportunities for transmission and establish whenever potential opportunities arise. Obviously these data are open to several interpretations, but they do emphasize the importance of long-term experiments in determining the possible impact of global warming on the distribution of parasites.

The effect of temperature on the parasites of terrestrial hosts.

The survival rates of the infective stages of the parasites of most terrestrial species tend to decrease with increasing temperature (Fig 5a). Although little evidence is available to determine how the infectivity of these larvae is affected by temperature, rates of larval development tend also to decrease with increasing temperature (Fig 5b). These two processes again interact synergistically allowing the parasite to establish at a broad range of environmental temperatures. However, in contrast to parasites that utilize aquatic hosts, parasites of terrestrial hosts have transmission stages that are susceptible to reduced humidity, and these stages are highly susceptible to desiccation (Wallace 1961). To compensate for reduced opportunities for transmission during periods of severely adverse climate,

parasites of terrestrial hosts have evolved adaptations such as hypobiosis. This allows them to remain in a state of arrested development within the relatively protected environment provided by their hosts until such time as transmission through the external environment proves more effective. The capacity of terrestrial nematodes to arrest their development is a heritable trait and one that seems to adapt very rapidly to different climatological and mangement regimes (Armour & Duncan 1987).

D.   Predictive models for parasites of domestic livestock.

Because interactions between temperature and humidity seem to be of major important in constraining the geographical range of many of the pathogens that infect domestic livestock, a considerable body of data exists which examines the relationship between meteorological conditions and parasite outbreaks (Gordon 1948; Kates 1965; Levine 1963; Thomas 1974; Ollerenshaw 1974; Wilson, Smith & Thomas 1982). Indeed the parasitologists of the 1950's and 1960's firmly believed that climate determined the distribution of a parasite species, while weather influenced the timing of disease outbreaks (Thomas 1974). This led to the development of large scale research programs designed to produce forecasts of when disease outbreaks were likely to occur in different areas and hence the best time to administer control measures.

Bioclimatographs.

One way of depicting the interaction between disease
outbreaks and climate was through the use of bioclimatographs
(Fig 6). The use of these diagrams for monitoring parasite
outbreaks was originally suggested by Gordon (1948) in a study of
the sheep nematode Haemonchus contortus (the barber's pole worm).
Bioclimatographs are constructed by plotting the climatological
conditions under which a parasite is able to exist and under
which outbreaks occur onto a graph of mean monthly temperature
and rainfall. When the mean weather data for each month of the
year are added to this diagram it is possible to determine at
which time of the year outbreaks of the parasite are likely to
occur. Although the initial production of these diagrams
requires a long term study of the parasite in any region, once
the data are available that describe the conditions for both
establishment and optimal development, then extrapolations may be
made to other regions for which only the climate data are
available. Thus Gordon (1948) was able to use his data for H.
contortus in Armidale, New South Wales, where outbreaks occur
from October to May, to explain why outbreaks rarely occured in
other regions such as Albury (NSW) and Deloraine (Tasmania).

Levine (1963) reviewed and extended the use of
bioclimatographs to define and explain the distribution and
seasonal incidence of a variety of gastro-intestinal parasites of

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sheep and cattle. As bioclimatographs are usually based on data
for mean temperature and rainfall, they are usually only
partially successful for predicting parasite outbreaks in any
specific year. Similarly, derivation of bioclimatographs from
laboratory determination of the parasite's minimum and optimum
development constraints is not often possible, as the climate
conditions experienced by the parasite larvae in the soil, are
often different from those measured by the local weather station
(Thomas 1974). However, bioclimatographs remain useful tools for
determining whether a parasite will establish in any region.
They may prove invaluable in determining whether long-term
climatic changes will permit specific parasites of domestic
livestock to establish in regions where they are not at present a
problem.

Effect of temperature on transmission stages of microparasites.

Up until now we have concentrated upon discussing the effect
of temperature on parasitic helminths, in part this reflects a
personal bias, but it also reflects the published literature.
Data on the effects of temperature, humidity and ultra-violet
light on the survival and infectivity of viral and bacterial
transmission stages seem relatively hard to locate. This
possibly reflects the technical difficulties in working with this
material. However, data do exist which suggest that the
development time of microparasite infections are dependent on

ambient temperature and there is some evidence to suggest that

the infectivity of some vector transmitted pathogens is

determined by the temperature at which their insect hosts are

raised (Ford 1971, p 104). Temperature may also indirectly

effect transmission rates by altering the behaviour of insect

vectors (Figure 7).

E. Effect of climate change on the distribution of

   trypanosomiasis in Africa.

Trypanosomiasis is one of the major diseases of humans and

their domestic animals in Africa (Ford 1971). The disease is of

particular importance to conservation in Africa as its presence

may exclude humans and their domestic livestock from areas where

wild animals act as a reservoir of the disease (Molyneux 1982;

Rogers & Randolph 1988). The pathogen may be classified as a

'microparasite,' it is transmitted by an insect vector, the

tsetse fly (*Glossina* spp). Rogers (1979) and Rogers & Randolph

(1986) have made an extensive study of the meteorological

conditions which determine the distribution of three species of

Tsetse flies (*Glossina morsitans, G. palpalis* and *G.* ~~*fuscipes*~~ *tachinoides*).

Their study is complimented by two models of the dynamics of the

different *Trypanosome* species, one by Rogers (1988) and one by

Milligan & Baker (1988). The former derives expressions for Ro

and H_T that provide some useful general insights into the

processes that are most important in determining the conditions

for the pathogen to establish; the latter develops a more
specific analytical model for trypanosomiasis based on detailed
parameter estimates from a study of *Trypanosoma vivax* in
Tanzania.

Rogers's (1979) analysis of the bioclimatic tolerances of
tsetse flies may be used to determine how predicted patterns of
climate change in tropical Africa might effect the distribution
of tsetse flies and trypanosomiasis. Using data from several
long term studies of two sub-species of tsetse flies in Nigeria
(*Glossina morsitans submorsitans*) and Zambia (*G. m. morsitans*),
Rogers (1979) was able to show that the mean monthly density-
independent mortality rates for these flies most closely related
to mean monthly saturation deficit (an index of humidity) and, to
a lesser extent, mean monthly temperature. These analyses
allowed Rogers to identify an environmental optimum for each
species of tsetse fly. When the data for ninety-one sites
throughout tropical Africa are examined in terms of these
climatological conditions, 94% of the sites within the present
known distribution of Tsetse flies fall within the predicted
bioclimatic limits, whilst only 50% of non-tsetse areas do so
(Fig 8). These data can be used to compare the present
distribution of Tsetse flies with the possible distribution given
a mean two degree increase in temperature for sub-Saharan Africa
(Figure 9). Here it is important to note that, because we have a
better correlation between bioclimatic data and where Tsetse

~~flies are~~ found, than where ~~they are~~ it is not found, greater confidence may be placed in the prediction for where ~~Tsetse flies~~ *G. morsitans* may decrease in abundance, than for regions where ~~they~~ it might establish. These caveats non-withstanding, the analysis suggests that ~~Tsetse flies~~ *G. morsitans* may become less common in West Africa and across the main sub-Saharan zone of central Africa. This pattern may be matched by a spread further south of the vector and its parasite in East Africa.

Although the approach we have adopted in this analysis is rather coarse, the data are available to make more sophisticated and detailed analyses for this and other pathogens. From a conservation perspective it remains very important to determine to what extent trypanosomiasis is at present maintaining areas as refuges for wild animals by excluding humans and their livestock (Molyneux 1982; Rogers & Randolph 1988). If a change of climate reduces tsetse levels, then pressure for the exploitation of these areas would increase with their subsequent loss as a wildlife refuge.

F.   The structure of parasite-host communities.

So far we have concentrated upon examining simple one host, one parasite systems. In systems that are dominated by the presence of one particularly prevalent pathogen, this approach seems fairly sensible. However, many host populations maintain a

commun... of several parasite species. The diversity of this community and the abundance of its constituent parasite species is intimately linked not only to the density of the host population, but also to the presence of other host species which act as reservoirs for other parasite species.

Dynamics of one host, many parasite species communities.

It is possible to extend the basic one host/one parasite models to examine the dynamics of more complex communities (Figure 10). Preliminary analysis of models for such communities suggests that parasite species diversity is a direct function of host density and that the relative abundance of each parasite's species is determined more by the life history attributes of the parasite that determine its transmission success, than by interactions with other parasite species (Dobson 1986, 1989). This suggests that changes in host density due to changes in meteorological conditions will be crucial in determining the diversity of the community of parasites supported by the hosts. Increases in the density of some hosts will allow them to support a more diverse parasite fauna, while decreases in the density of other hosts will reduce the diversity of their parasite community. A study comparing the effects of thermal cooling on the parasite fauna of an aquatic snail presents some corroborative evidence in support of this model.

Sankurathi and Holmes (1976a,b) studied a population of

Physa gyrina and its parasites and commensals in Lake Wabamun in Alberta, Canada. A section of the lake was used as a cooling facility by a power station and consequently was warmer than the rest of the lake and relatively free of ice in winter. The effects on the population of snails were pronounced when both density and population structure are compared for heated and control sites, with population density often several orders of magnitude higher in the heated areas (Figure 11). This, and the continual presence of the vertebrate definitive hosts in the parasites life cycle allowed a considerable increase in both the prevalence and diversity of the parasite community living in the snail population (Table 12). It is also interesting to note that the increased water temperature had a detrimental effect on the two species of commensal chaetogasters that live in the mantle of the snails. Laboratory experiments showed that these commensals act as predators which live by attacking and ingesting the infective stages of parasites that try and infect their snail host (Sankurathi & Holmes 1976b). An increase in temperature leads to the chaetogasters abandoning the snail and dying, this in turn leads to further increases in the rates of parasitism of the snail hosts.

Dynamics of two host, many parasite species communities.

A more complex pattern emerges if we consider the community structure of parasite in host species which share parasites. When parasites are able to use more than one species as a

definitive host, their ability to establish in any one host
species will be dependent upon the density of all the potential
species of definitive host present in an area. As different host
species may have different susceptibilities to the parasite and
different parasite species may reproduce at different rates in
different host species, the composition of the parasite
assemblage will dependent crucially on the density of different
host species (Fig 13). Variations in the population density of
different host species may thus lead to variations in the
parasite burdens of other host species; in some cases this may
allow pathogenic parasites to establish in populations of hosts
which would otherwise be too small to sustain them. Changes in
the composition of host communities that result from climatic
changes will lead to changes in the structure of the parasite
community that these hosts support and the possible introduction
of parasites not previously present in the host population.
Where members of the parasite community are important in
mediating competitive interactions between hosts, this may lead
to further changes in the structure of the host community and the
possible extinction of particularly susceptible hosts.

Conclusions.

     The geographical distributions of most parasite species are
constrained by either the distributions of potential host species
or by environmental constraints on the parasite's rates of

development. Although developmental rates in vertebrate hosts may be comparatively unaffected by changes in environmental temperature, the available evidence suggests that parasite free-living stages and those that live in invertebrate poikilothermic hosts are susceptible to prevailing meteorological conditions. Gillett (1974) suggests that the range boundaries of many vector transmitted diseases are determined by the development time of the parasite exceeding the average life expectancy of the insect vector. As increases in environmental temperature are likely to lead to more rapid developmental times for the stages of parasite life cycles that utilize insects as vectors, long term increases in temperature are likely to lead to increases in the ranges of many diseases transmitted by insects such as malaria and filariasis.

Up until the mid-1970's parasitologists believed that temperature and moisture were the dominant meteorological factors determining disease outbreaks. Curiously this area of parasitology has been relatively neglected for the last ten to fifteen years. In part, this may be due to the development of anthelmintic drugs which could be readily administered to livestock. It may also be due to a change of direction in models for parasites which now emphasize the previously neglected non-linear components of parasite dynamics (Anderson & May 1978, May & Anderson 1979). Finally it may also reflect the emergence of molecular immunology and the search for vaccines for parasites of

domestic livestock. However, serious levels of resistance have now emerged to many anthelmintic drugs (Anderson & Waller 1985) and the development of vaccines is progressing at a slower rate than was originally anticipated. As long-term climatic changes may lead to the introduction of parasites into new areas at a time when our ability to control them is rapidly diminishing, this may cause major disease problems for many types of domestic livestock. In some cases this will lead to the abandonment of present pasture lands which may then be set aside for nature reserves. In other regions it will lead to increased pressure by an increasingly hungry human population to utilize present reserves as grazing areas. It seems unlikely that the net result of this exchange will be in favor of wildlife.

A considerable body of literature is already available that deals with the climatic responses of a variety of parasites (Kates 1965; Levine 1963; Thomas 1974; Wilson, Smith & Thomas 1982). We now also have much better models for examining the dynamics of parasites at all stages of their life cycles (Anderson & May 1979, 1986; May & Anderson 1979). Although there are problems of scale associated with extrapolating between the physiological processes of parasites measured under controlled laboratory conditions and the coarser scale predictions available for longer term climate change, it should be possible to merge these various sources of information to produce a quantitative synthesis of the way global climate change may effect the

distribution of many parasites. It thus seems likely that global warming will give new prominence to an area of parasitology that had fallen into relative neglect.

Implications for conservation.

The examples given above are mainly from well studied species in little danger of extinction. Assessment of the potential effects of global warming on the parasites of endangered species can really only be undertaken by extrapolation from these examples and the models used to explain the more general features of parasite-host population dynamics. In this final section we consider a number of possible scenarios that are likely to arise as host populations respond to long-term climate changes.

Initially consider an endangered species whose population density has declined to such low levels that it is only present in a single nature reserve. Under these conditions it seems likely that a further decline in population size due to global warming will reduce the effects of the parasites already present in that population. However, the immigration of new host species into the area, as a response to climate change, may lead to the introduction of novel pathogens. If the endangered host has had no previous contact with these parasites, they may either fail to establish, if the host is sufficiently novel, or they may

establish and potentially produce significant levels of
mortality. Under these conditions, increases in the density of
the immigrant hosts will lead to increases in the rates of
parasite transmission and constraints may have to be placed on
interactions between the endangered species and the newly
immigrating species.

Where endangered species are tolerant to increases in
temperature and humidity, they are still likely to face increased
assault by parasites whose transmission efficiency improves due
to increases in temperature and humidity (e.g. tropical diseases
such as hookworm may become more important in temperate zones).
Furthermore, those host species that respond to increased
temperatures by increases in population size, are likely to
suffer an increase in parasite prevalence and diversity.

If the population sizes of host species decline due to
climatic changes, their rarer species of parasites and mutualists
may go extinct.   These species have their own intrinsic value,
they often perform a valuable function, such as the commensal
chaetogasters living in the snail mantles discussed above. It
should also be bourne in mind that the absence of a parasite may
be as important as its presence. Some species of hosts may grow
to become pests in the absence of pathogens which may at present
regulate their numbers.

Parasites and disease will do well out of global warming. They are, by definition, organisms that are designed to colonize and exploit. Those species of parasite that are already common will be able to spread and perhaps colonize new susceptible hosts that may have no prior genetic resistance to them, parasite species that are rare and have more specialized requirements may be driven to extinction. In general, these effects are likely to be worse in the temperate zone, where parasites from the tropics can colonize new hosts, than in the the tropics, where parasites will have to adapt or evolve. However, rare parasites that are adapted to extreme temperature may become common; changes in the ranges and sizes of some host populations may allow some hitherto unimportant pathogens to become more widely spread.

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H.   Tables

Table 1. Expression for the basic reproductive rate, Ro, and the threshold for establishment, H, for different parasite life cycles.

Life cycle	Ro	H
Monoxenic	$\dfrac{T_1}{M_1 \cdot M_2}$	$\dfrac{\delta \cdot M_1}{\beta(\lambda - M_2)}$
Heteroxenic : one free-living stage, transmission to definitive host via a predator-prey relationship.	$\dfrac{T_1 \cdot T_2}{M_1 \cdot M_2 \cdot M_3}$	$\dfrac{M_1 M_2 M_3}{\alpha\Gamma(T_2 - M_1 M_2)}$
Heteroxenic : two free-living stages, transmission to definitive host via a cercaria or free-living larvae.	$\dfrac{T_1 \cdot T_2}{M_1 \cdot M_2 M_3 M_4}$	$\dfrac{\delta M_1 M_2 M_3}{\Omega\Theta(T_2 - M_1 M_2 M_3)}$

Here T_i are the transmission rates between definitive and intermediate hosts, while the M_i are the total mortality rates for each stage in the life cycle. In parasites with heteroxenic life cycles Γ is the rate at which definitive hosts prey on intermediate hosts, α is the increased susceptibility of predation of infected intermediate hosts, while Ω is the rate of asexual reproduction of parasites that reproduce in their intermediate host. For a full definition of these expressions see Dobson (1988a).

I.   Figure legends.

Figure 1.   The relationship between mean parasite burden and
definitive host population size for a direct life cycle
macroparasite (after Dobson 1989).

Figure 2. (a) The effect of water temperature on the survival
rate of the cercaria of *Echinostoma liei* (after Evans 1985).
(b) The influence of water temperature on the infectivity of
E. liei cercariae. (c) The net effect of temperature on the
transmission efficiency of *E. liei* cercariae (Evans 1985).
(d) The results of similar interactions between survival and
infectivity on the net transmission efficiency of the
miracidia of *Schistosoma mansoni* at a range of temperatures
and at three different host densities (after Anderson et *al,*
1982).

Figure 3. The duration of the prepatent period (the time between
infection and production of infective cercariae) for two
species of Schistosome (*S. mansoni* and *S.haematobium*) in
their snail hosts (*Planorbis pfeifferi* and *Physopsis globosa*
respectively) at three different temperatures (after Gordon,
Davey & Peaston, 1934).

Figure 4.   The mean parasite burdens of (a) *Ornithodiplostomum
ptychocheilus* and (b) *Diplostomum scheuringi* in *Gambusia*

affinis from a thermally warmed and : 'control' stream
(After Camp, Aho & Esch (1982) and Aho, Camp & Esch (1982)).

Figure 5. (a) Survival of the larvae of *Trichostrongylus retortaeformis,* a parasite of rabbits, at a range of temperatures. (b) Development time of *T. retortaeformis* at these temperatures (after Levine 1963).

Figure 6. Bioclimatograph for *Haemonchus contortus* in three different regions of Australia (after Gordon 1948). The isoclines at 51° F. and 210 pts. rainfall delineate the meteorological conditions at which *H. contortus* can just establish, the isoclines at 57° F. and 260 pts. rainfall outline the conditions under which epidemic outbreaks occur. "Haemochosis season" in Armidale lasts from September through till April. In contrast, although *H. contortus* is recorded from Albury (NSW) and Deloraine (Tasmania), the climate conditions for an epidemic are rarely attained.

Figure 7. The development of the oocytes and oviposition-time in *Aedes aegypti* (a vector of malaria) (a) at 29° C and (b) at 27° C. In (a) ovarian development takes less than 48 hours and eggs may be laid during the second circadian activity period following a blood meal. In contrast, at 27° C ovarian development takes a little more than 48 hours but eggs are

not laid until the third circadian activity-period, c. 70

hours after the blood meal. This leads to a step-wise

function relating temperature to frequency of blood meals

(after Gillett 1973).

Figure 8. The predicted bioclimatic limits and annual means of

monthly meteorological conditions for (a) thirty three *G.*

morsitans areas and (b) fifty-eight non-tsetse areas

throughout tropical Africa (after Rogers 1979). 94% of

tsetse areas fall within the predicted bioclimatic limits,

whilst 50% of non-tsetse areas fall outside them.

Figure 9. (a) The distribution of the weather stations listed by

Rogers (1979) with stations where ~~tsetses are~~ *G.morsitans is* present

marked with a '+' and stations where ~~tsetse's are~~ *G.morsitans is*

absent marked with a '<>'. (b) The potential change in

the distribution of stations with and without ~~tsetse~~ *G.morsitans*

~~flies~~ following a mean two degree increase in

temperature.

Figure 10. The relative abundance of different parasite species

at a range of host densities in a simple one host, five
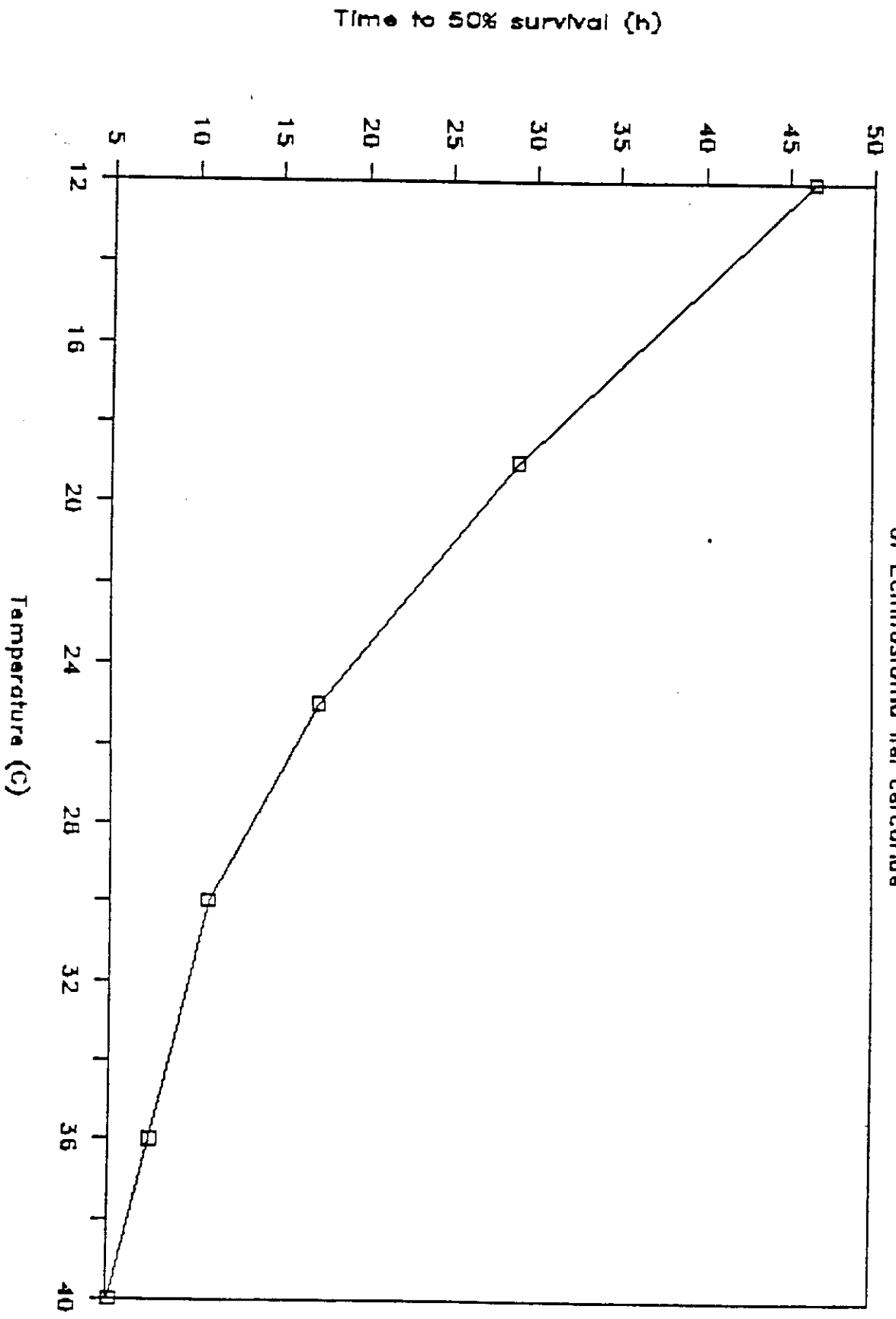
parasite species community (after Dobson 1989).

Figure 11. The effect of thermal cooling on the density of a

mollusc population and its community of parasites in a

Canadian lake (after Sankurathri and Holmes 1976 a,b). The top figure shows the surface water temperatures in the control and heated areas of the lake. The middle diagram compares the population density of the snail *Physa gyrina* in the control and heated areas. The lower diagram shows changes in the prevalence of metacercarial infections of snails from the control and heated areas.

Figure 12. The percentage of snails infected with cercariae and metacercariae in the control and heated areas of Lake Wabamum (after Sankurathi & Holmes 1976b). The abbreviations used are: E.r. - *Echinoparyphium recurvatum*, N.u. - *Notocotylus urbanensis*, C.d. - *Cercaria douglasi*, O.p.- *O. ptychocheilus*, A.g. - *Apatemon gracilis*, T.c. - *Trichobilharzia cameroni* and T.p. - *T. physellae*.

Figure 13. The thresholds for establishment and expected composition of a parasite community at a range of host densities in a simple model of a two host, five parasite species community model (after Dobson, 1989). The lines are based on parameter values a = E the pair were made up of combinations in sequence was generated and the relationship of the time taken to reach equilibrium for the density to the number of the parasite species that make up the community may change.

Influence of temperature on survival
of Echinostoma l/e/ cercariae

Influence of temp. on transmission
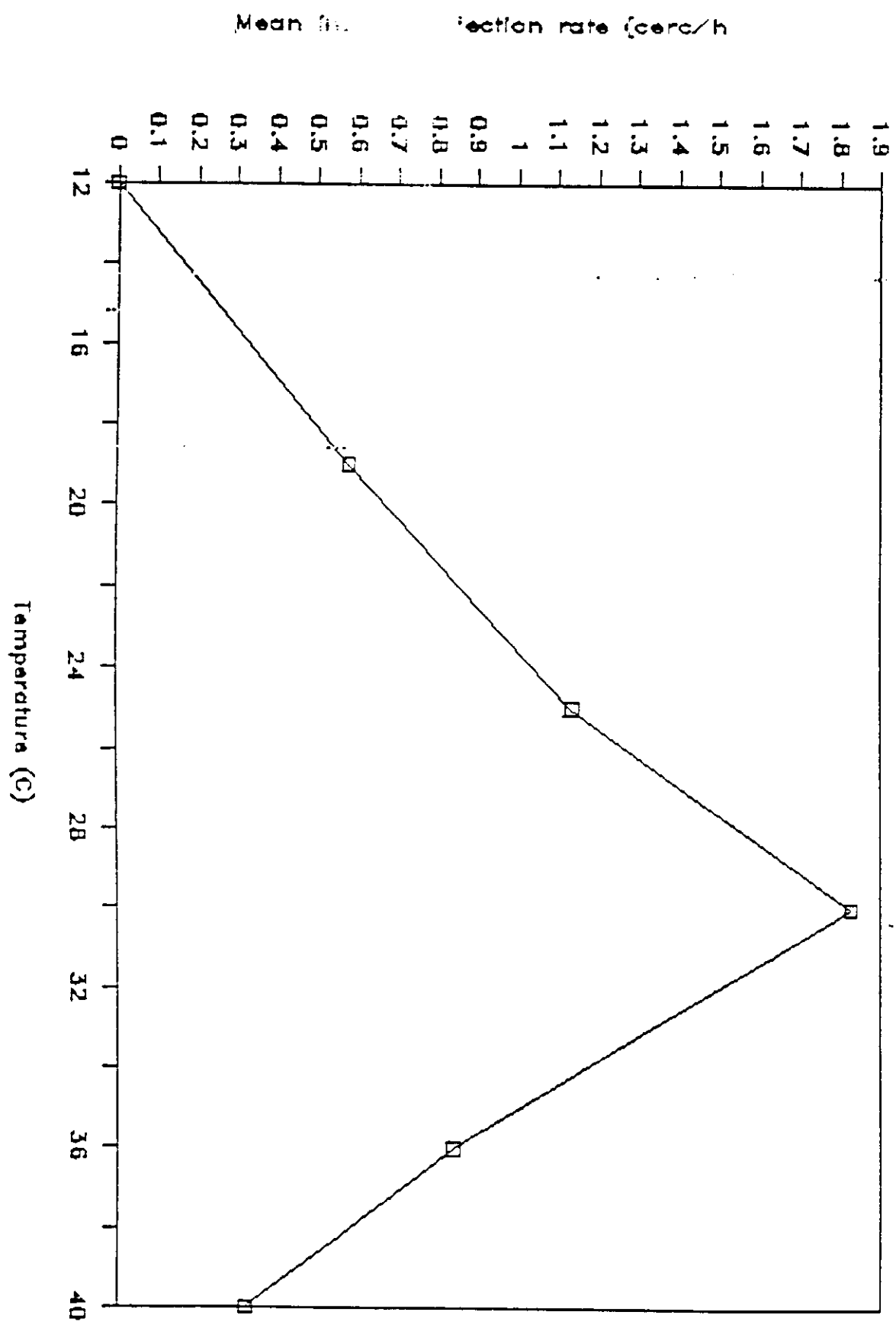### efficiency of E. Ilal cercariae

Transmission effeciency

Temperature (C)

34
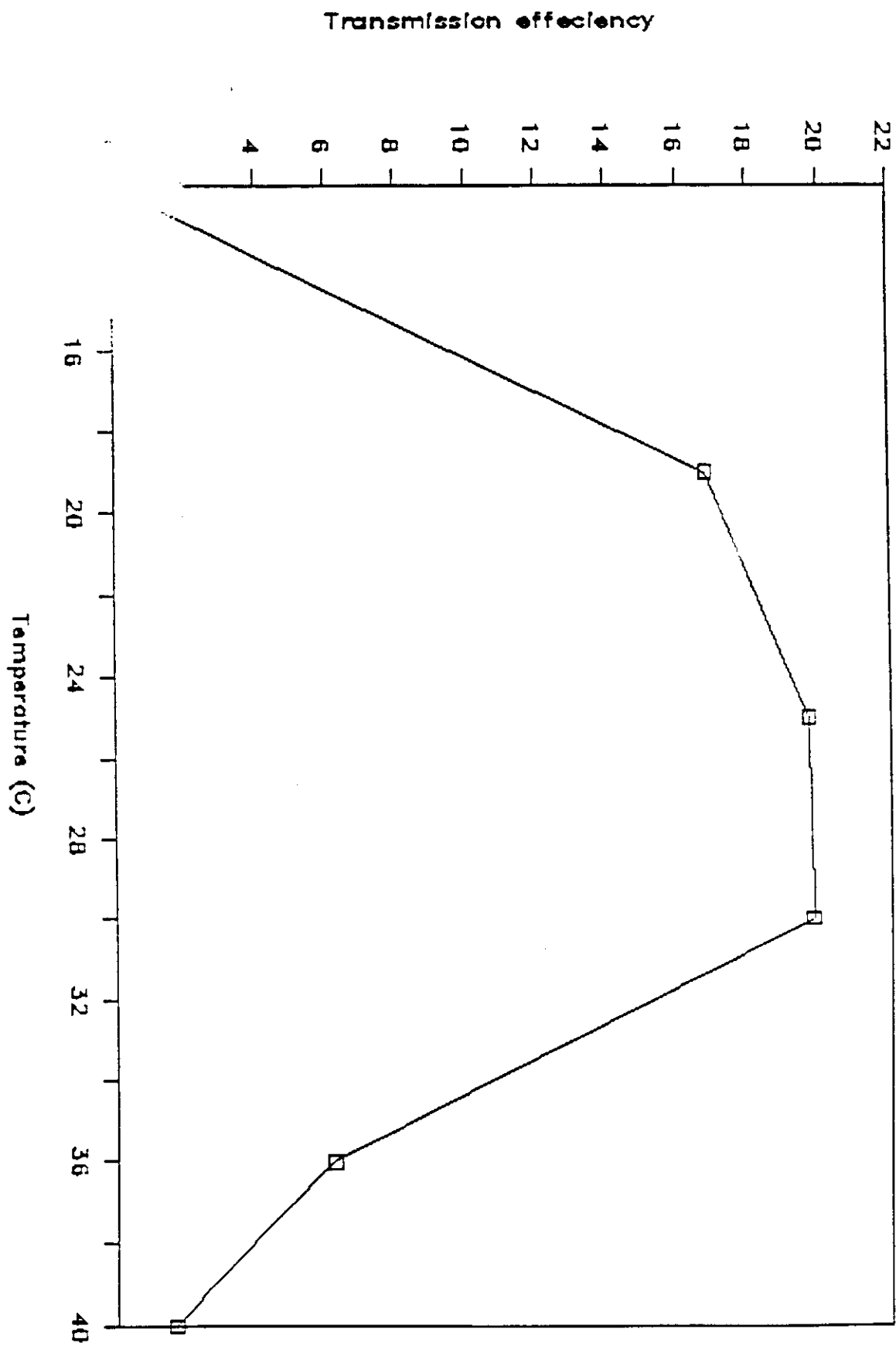
Probability of infection

Effects of density & temperature.

Probability of Infection.

Temperature (C)

□   0.04/5ml   +   0.2/5ml   ◇   1/5ml

40
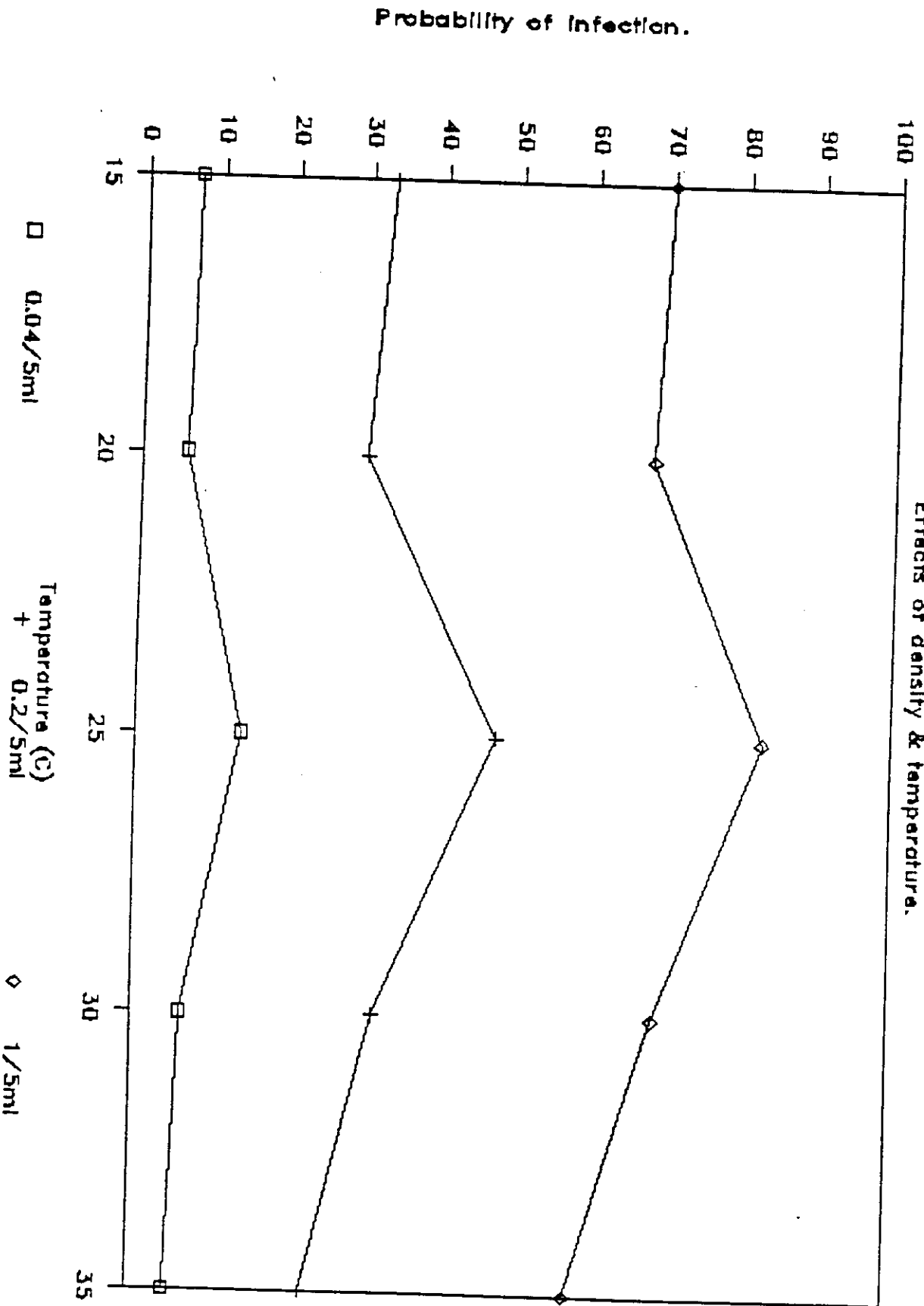
Influence of temperature on infection
rates of Echinostoma liei cercariae

Mean infection rate (cerc/h

Temperature (C)

AFRICA

Distribution of tsetse flies.

+ Tsetse

◇ No-Tsetse
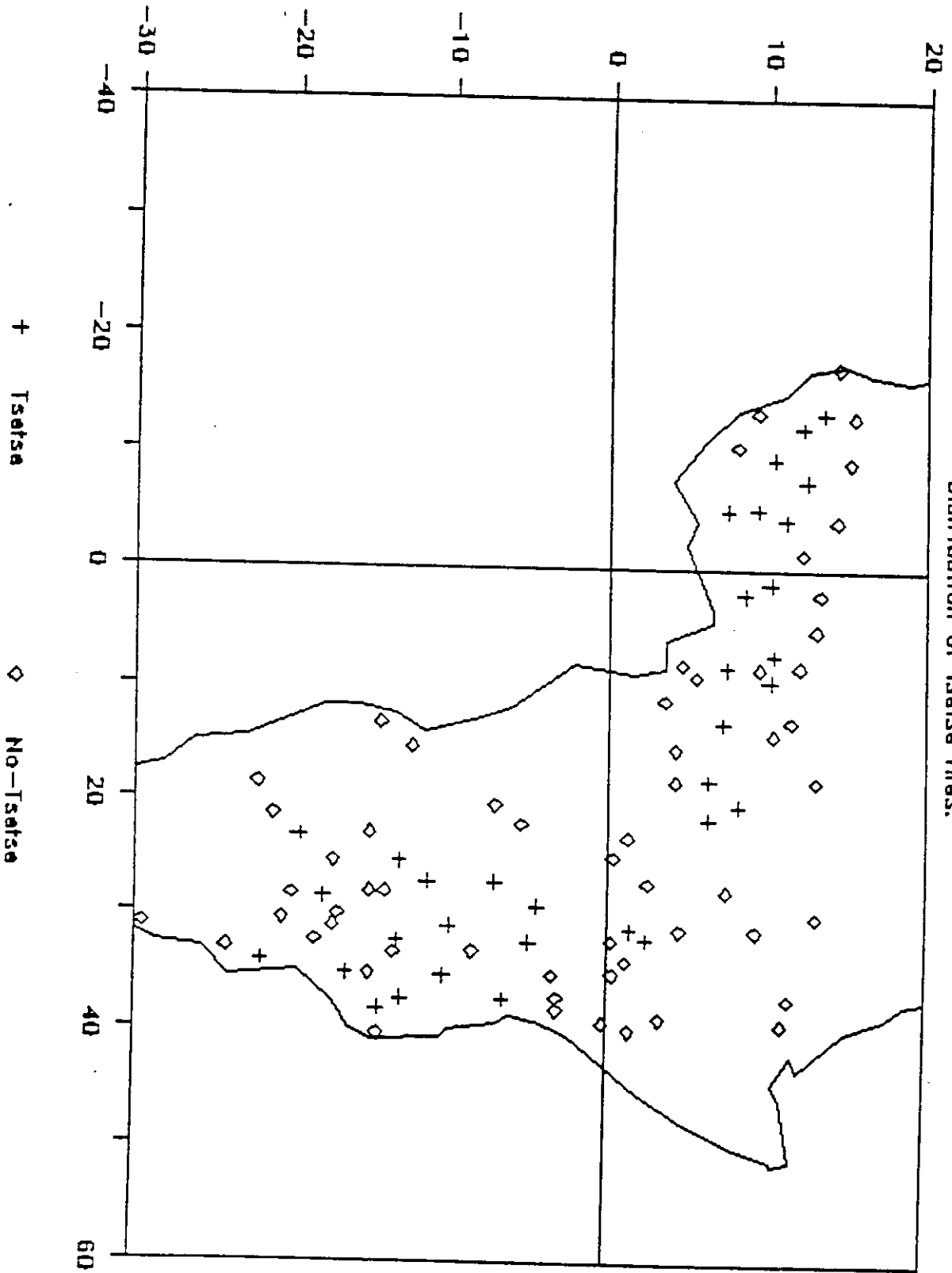
2 DEGREE INCREASE IN TEMPERATURE

Distribution of tsetse files.

+   Tsetse

◇   No-Tsetse

43
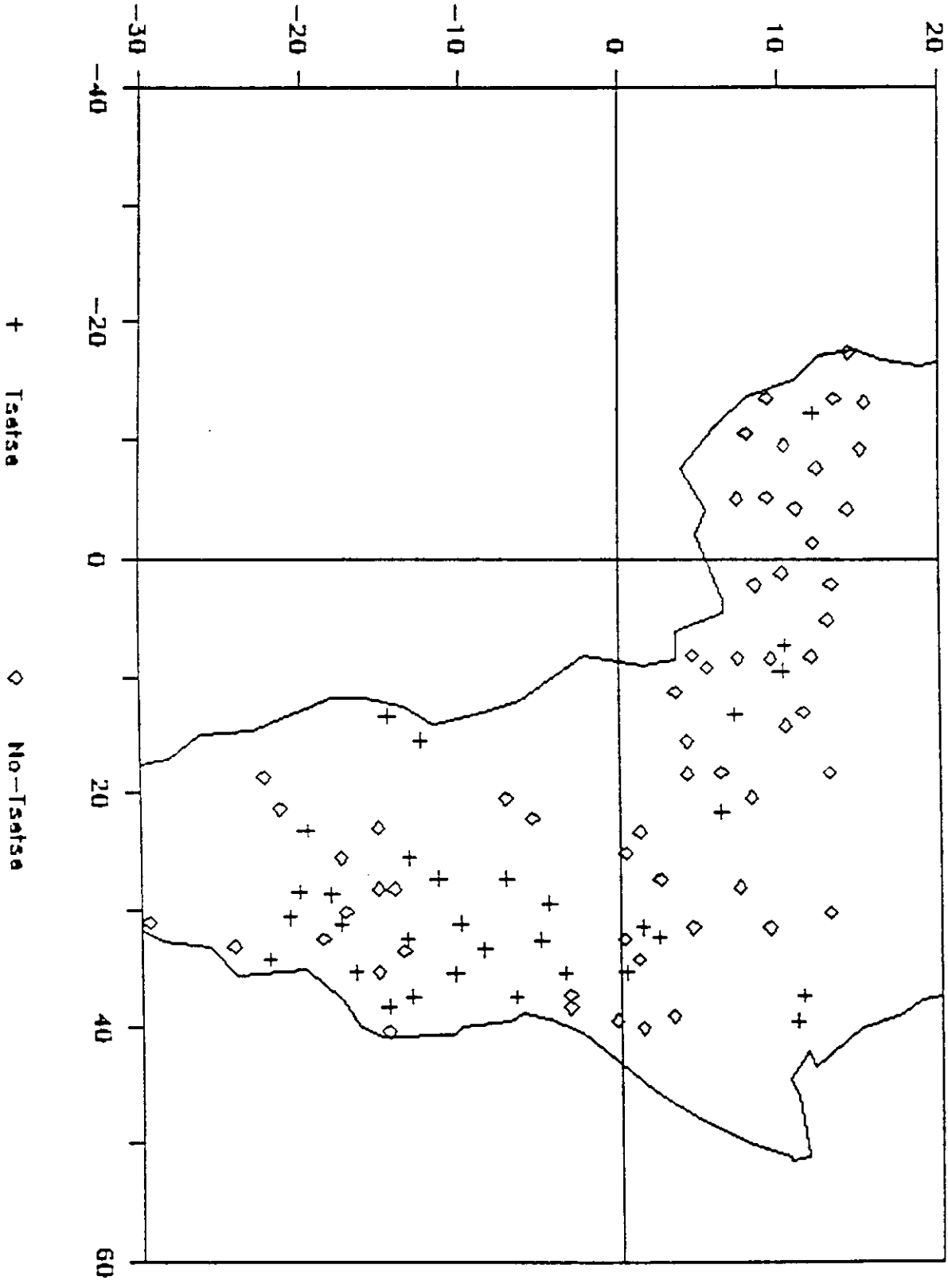
O. ptychocheilus in mosquitofish.
(Camp, Aho & Bush, 1982)

Mean Parasite Burden.

Month (Jan 1976– May 1980)

□ Ambient
+ Heated
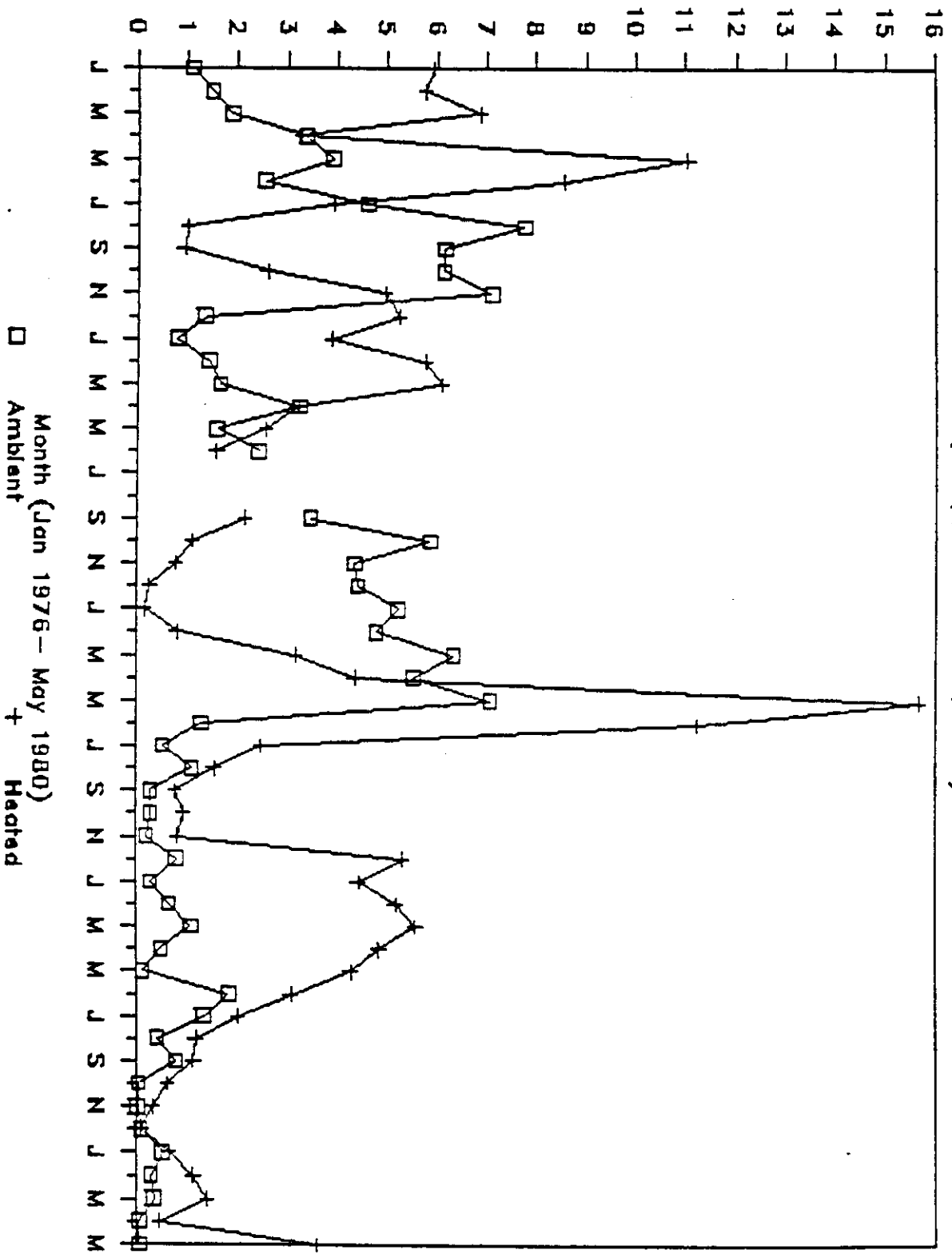
Diplostomum scheuringi in mosquitofish.

(Aho, Camp & Bush, 1982)

Mean Parasite Burden.

Month (Jan 1976– May 1980)

□   Ambient

+   Heated
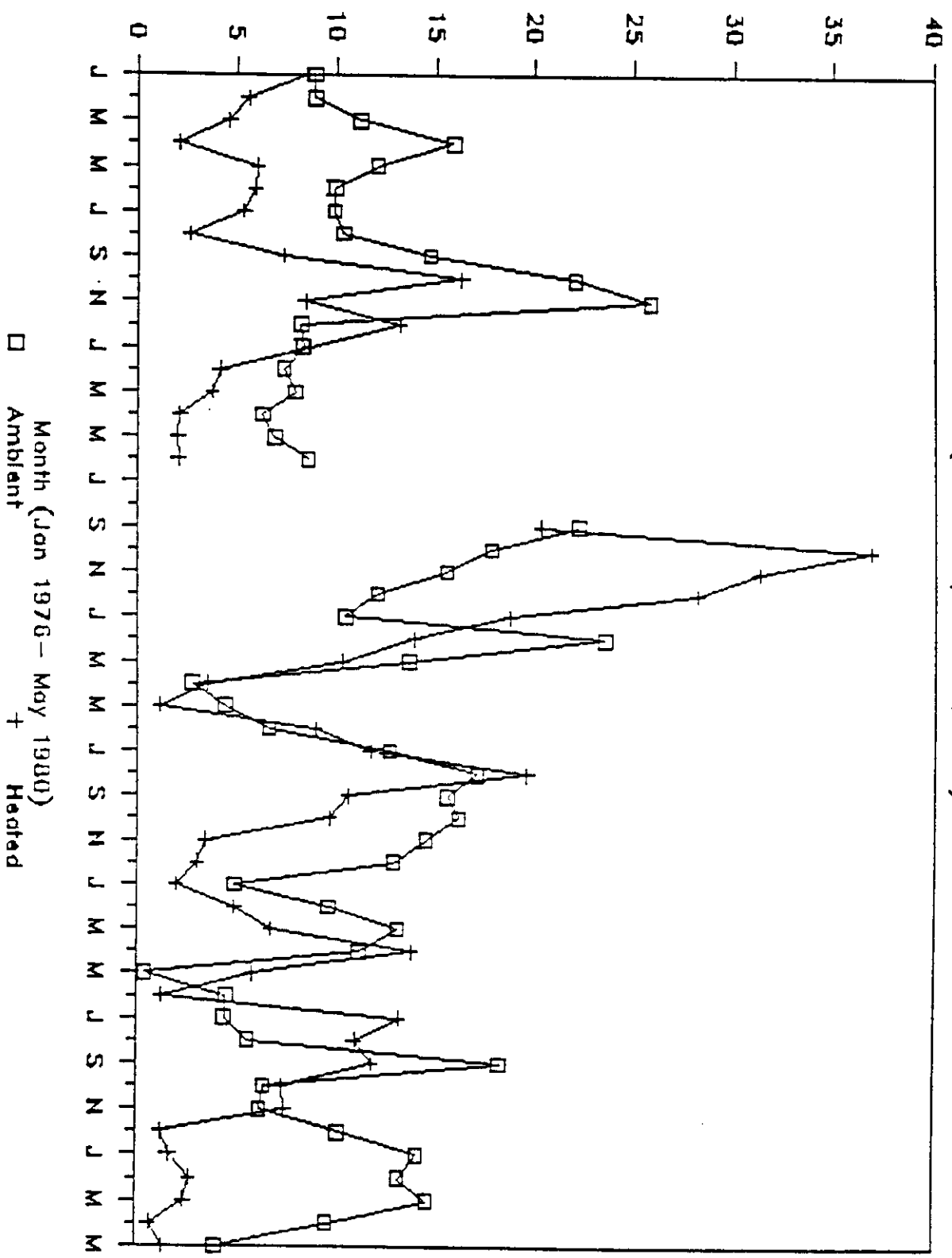
Relation of Temperature to Survival
of Trichostrongylus larvae from rabbit

Survival Time of Larvae (Days)

Temperature (Celsius)

□ 50% Survival   + 10% Survival

46
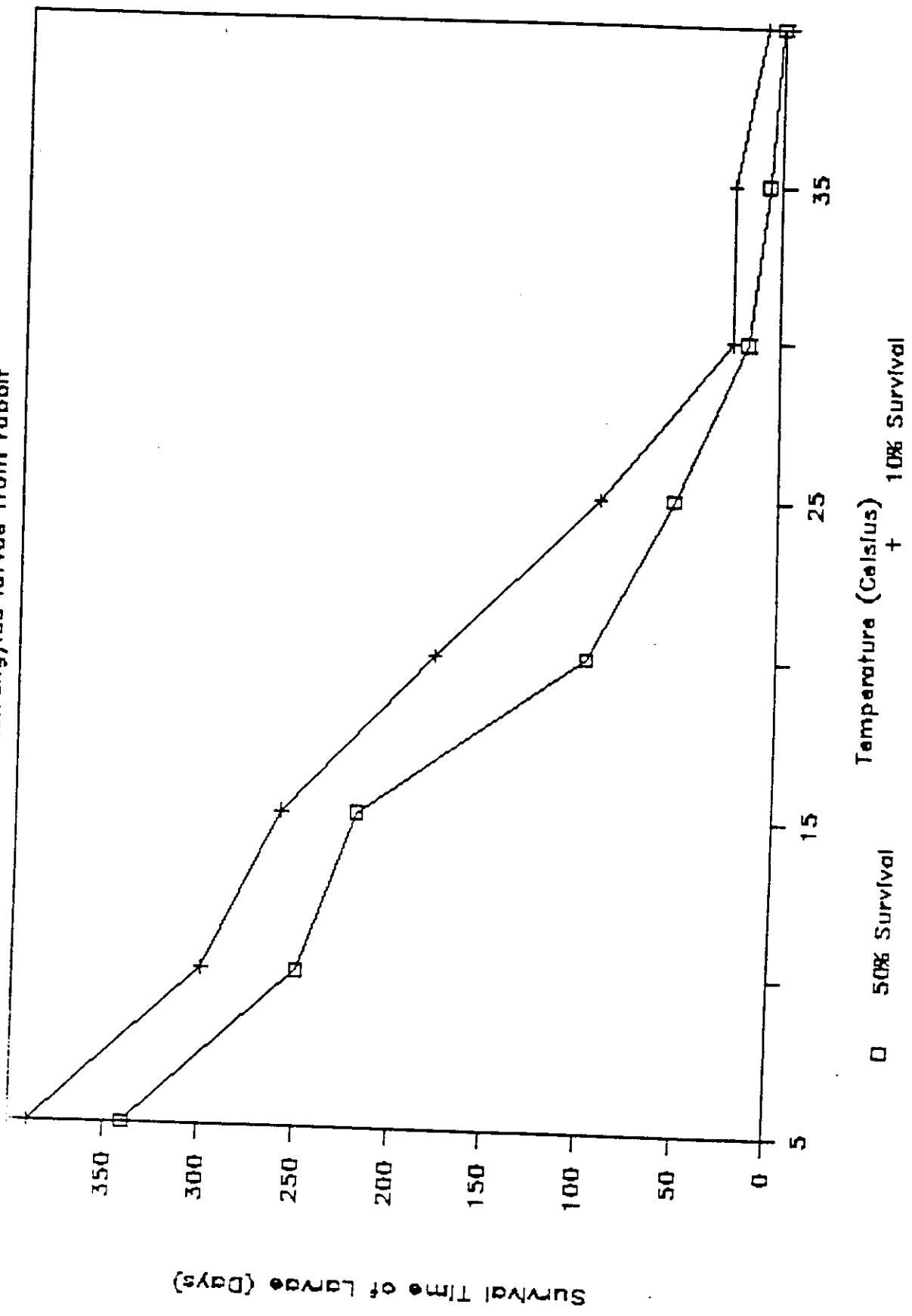
Relation of Temperature to Development

Development Time (Days)

Temperature (Celsius)

47