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"Regulation and stability of a free-living host-parasite system: Trichostrongylus tenius in red grouse. II. Population models"

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Regulation and stability of a free-living host-parasite system:

Trichostrongylus tenuis in red grouse.

II. Population models.

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Running heading: Population models of grouse parasites.

SUMMARY

(1) The population dynamics of red grouse, Lagopus lagopus scoticus and the parasitic nematode, Trichostrongylus tenuis were explored to determine whether interactions between the parasite and host were sufficient to generate cycles in grouse abundance. Two alternate models were used that explicitly consider the dynamics of either the free-living, or arrested larval, stages of the parasite.

(2) Establishment of the parasite in grouse populaations requires host densities in excess of 24 birds per km^{-2} and free-living larval life expectancy in excess of six weeks. Larval arrestment tends to reduce the intrinsic growth rate of the parasite and to increase the size of the host population required for the parasite to establish.

(3) Grouse numbers will tend to cycle when the parasites exhibit low degrees of aggregation and parasite-induced reductions in host fecundity are greater than parasite-induced reductions in host mortality. In the model that contains arrested parasitic larvae, these population cycles have the slow increase followed by a rapid decline characteristic of many grouse populations.

(4) The period of the cycles is determined by the intrinsic growth rate of the grouse population and either, larval life expectancy (Model I), or the duration of larval arrestment (Model II). Cycle periods become shorter as host population growth rate increases, and longer with decreases in either free-living larval life expectancy, or the duration of larval arrestment. However, if the duration of larval arrestment is sufficiently long (> 6 months), the cycles die out and the dynamics of the grouse-nematode system are very stable.

(5) Estimates of all of the models parameter's may be made from long term records of grouse populations. Numerical analysis of the models behaviour suggest that the model that includes arrested larval stages more closely corresponds to the grouse populations in the North of England. The four to five year cycles exhibited by these populations will be more sporadic, or absent on estates where the parasite is unable to establish. This is likely to occur in areas where grouse numbers are low, or where climatic and habitat conditions produce low levels of survival in free-living larval stages of the parasite.

(6) The analysis shows that T.tenuis is an important cause of the cycles observed in grouse populations in the North of England. It suggests that parasites may be an under appreciated, but necessary, determinant of cycles in other game populations.

INTRODUCTION

Considerable controversy exists over whether the long term cycles of abundance observed in grouse populations are caused by extrinsic interactions between grouse and their nematode parasite (Hudson, Dobson & Newborn, 1985), or by intrinsic features of grouse population biology (Wynne-Edwards 1986; Watson & Moss 1979). The population biology of red grouse and it's caecal nematode Trichostrongylus tenuis have been studied in detail at Gunnerside, North Yorkshire for 10 years. Losses from the grouse population were associated with the intensity of parasite infection, while manipulation experiments have demonstrated that the parasite is the principal cause of reductions in grouse breeding production (Hudson, Newborn & Dobson 199X). In this paper we use the data presented in the Gunnerside study to develop a mathematical model that explores the population dynamics of the grouse-T. tenuis system. The main objective of this exercise is to determine whether interactions between T.tenuis and grouse are sufficient to generate the cycles in grouse abundance. The models are then used to examine which features of the parasite host interaction are necessary for sustained cycles in parasite and host abundance.

Anderson & May (1978) and May & Anderson (1978) (henceforth A&M) derived the fundamental model for the population dynamics of parasitic helminths, P, and their hosts, H. Here we extend the A&M models to consider the dynamics of the free-living, W, and

arrested larval stages, A, of the parasite. In particular, we are concerned with the dynamic consequences of hypobiosis - the ability of larval parasites to arrest their development after infecting a suitable host (Schad 1987). This period of arrestment has the potential to put significant time delays into the parasite life cycle and may cause time-delayed density dependent effects to appear in the dynamics of the parasite-host interaction. The various birth and death rates of the grouse and T.tenuis populations and the flow rates between the different stages of the parasites life-cycle are illustrated in Figure 1.

Initially, the effects of larval arrestment are ignored and a modified three equation A&M model is described that considers the free-living larval stages of the parasite (Model I). We then assume the free-living larvae are relatively short-lived and describe a second three equation model in which the larvae enter a period of hypobiosis immediately after infecting a suitable host (Model II).

Structure of the basic model: I. Long-lived free-living larvae.

The dynamics of a host-parasite interaction that includes free-living infective larvae may be described by the following set of three coupled differential equations:

$$dH/dt = (a-b)H - (\alpha+\delta)P \qquad (1),$$

$$dW/dt=\lambda P-\gamma W-\beta WH \qquad (2),$$

$$dP/dt=\beta WH-(\mu_P+b+\alpha)P-\alpha\frac{P^2}{H}\left(\frac{\kappa+1}{\kappa}\right) \qquad (3).$$

The parameters used to describe the different processes operating are defined in Table 1. The model assumes that the parasites are distributed in an aggregated manner, best described by the negative binomial distribution, this corresponds to the situation observed in the field data (Hudson, Newborn & Dobson, 199*); The degree of aggregation is given by K, a parameter which varies inversely with the degree of aggregation. Field experiments have shown that parasites reduce host breeding production and survival (Hudson, 1986; Hudson, Newborn & Dobson, 199*), these effects tend to act linearly over several orders of magnitude, as is assumed in the standard A&M model.

Model II. The effects of hypobiosis.

In model II we consider a second modification of the basic A&M framework that contains an equation for the larvae that enter a period of arrestment in their development after infecting the definitive host and before developing to pathogenic adults. These larvae are subject to mortalities due to the natural mortality rate of the hosts and parasite-induced mortalities due to the presence of adult parasites. As metabolic activity in arrested larvae is very low they have no discernible effect on

host survival or fecundity and their intrinsic mortality rate is extremely low (thus $\mu_P>>\mu_A$). In this model the equation for host population size remains unaltered and equations 2 and 3 are replaced by

$$dA/dt=\frac{\lambda PH}{(H+H_o)}-(\mu_A+b+\theta)W-\alpha\frac{PA}{H} \qquad (4),$$

$$dP/dt=\theta A-(\mu_P+b+\alpha)P-\alpha\frac{P^2}{H}\left(\frac{\kappa+1}{\kappa}\right) \qquad (5).$$

This model assumes that free-living larvae are short-lived relative to other stages in the life cycle and transmission is governed by the single parameter Ho ($=\gamma/\beta$). Immediately after establishing in the host the larvae enter a period of hypobiosis and remain arrested for a period of $1/\theta$ years.

Basic reproductive rate of the parasite, Ro.

Before considering the model's transient and equilibrium dynamics, it is useful to derive expressions for the basic reproductive rate of the parasite, R_o, and the threshold number of hosts, H_T, required to sustain a continuous parasite infection. Maximum rates of parasite growth occur when the parasite is first introduced into the population, at this time pathogenicity and other density-dependent constraints are effectively trivial. Collapsing equations 1-3 and 4-5 down under these conditions produces two expressions for the initial

growth rate of the parasite population in models I and II respectively

$$R_o = \frac{\beta\lambda H}{(\mu_P + b + \alpha)(\gamma + \beta H)} = \frac{T_1}{M_1 M_2} \qquad (6),$$

$$R_o = \frac{\theta\beta\lambda H}{(\mu_P + b + \alpha)(\gamma + \beta H)(\mu_A + b + \theta)} = \frac{\theta T_1}{M_1 M_2 M_3} \qquad (7),$$

The two expressions are similar in form; they consist of a 'birth' term, $T_1 = \beta\lambda H$, the rate of transmission of eggs from birth through to establishment in another grouse, and the mortality rates of each stage in the life cycle: adult worms, $M_1 = (\mu_P + b + \alpha)$, free-living larvae, $M_2 = (\gamma + \beta H)$, and arrested larvae, $M_3 = (\mu_A + b + \theta)$. In both cases the basic reproductive rate of the parasite consists of the product of the mean number of new infections established by each female worm and the life expectancies of each stage of the worms life cycle. Estimates of some of these parameters are derived in the Hudson, Newborn & Dobson (199*) or have been obtained from previously published literature (Table 2).

As is usually the case with epidemiological studies, few data are available with which to estimate rates of transmission. A coarse estimate of the rate of transmission may be obtained by determining the threshold host density at which the parasite is

just able to maintain itself in the host population. Expressions for the establishment thresholds in parasites with, or without, larval arrestment are obtained by setting R_o to unity and rearranging Equation 6 and 7.

$$H_T = \frac{\gamma M_1}{\beta(\lambda - M_1)} \qquad \textbf{(8) Model I,}$$

$$H_T = \frac{\gamma M_1 M_3}{\beta(\lambda\theta - M_1 M_3)} \qquad \textbf{(9) Model II.}$$

The threshold density of grouse at which infection rate is zero will vary between populations according to a variety of environmental conditions (Hudson, Dobson & Newborn, 1985; Shaw & Moss 1989; Hudson, Newborn & Dobson 199*). In sites similar to the main Gunnerside study area parasite persistence is erratic at densities less than 24 grouse per km^{-2}. This suggests a figure for β in the order of $0.5\text{-}0.7 \times 10^{-4}$.

As with most helminth parasites, the survival of the free-living larval stages is dependent upon climatic conditions (Shaw, Pike & Moss, 1989). The experiments of Watson (1988) suggest that the free-living larvae have a life expectancy of between 2-6 months although data from other Trichostrongyles species suggest that certain conditions may allow survival rates of longer than this (Boag & Thomas, 1985). The effect of variations in the life

expectancy of the free-living stages on the ability of the parasite to establish may be examined by plotting the values of R_0 and H_T for life-cycles with and without hypobiosis (Figure 2). In both cases once larval life expectancy falls below 6 weeks the basic reproductive rate of the parasite falls below unity and it is unable to establish.

An interesting point to emerge from Figure 2 is that parasite populations that exhibit hypobiosis have reduced growth rates when compared to populations with more direct life cycles. Examination of Eqn 7 suggests that low levels of host pathogenicity and high survival rates may be important pre-requisites for the evolution of arrested development. The costs of hypobiosis may be further appreciated by considering the effect of duration of larval arrestment on Ro (Figure 3). The potential for rapid expansion of the parasite population is considerably reduced as the duration of larval arrestment increases.

Equilibrium densities of hosts and parasites.

A qualitative comparison of how larval demography affects the magnitude of host and adult parasite populations may be gained by considering the equilibrium properties of both sets of equations. By setting dH/dt = dW/dt = dP/dt = 0 in 1-3, we obtain for model I:

$$\frac{P^*}{H^*} = M_P^* = \frac{(a-b)}{(\alpha+\delta)} \qquad (10),$$

$$W^* = \frac{d'(a-b)}{\beta(\alpha+\delta)} \qquad (11),$$

$$H^* = \frac{\gamma d'}{\beta(\lambda - d')} \qquad (12).$$

Here M_p^* is the mean parasite burden of adult grouse at equilibrium and $d' = \mu_p + \alpha + b + k'(a-b)\alpha/(\alpha+\delta)$, with $k'=(k+1)/k$. In model II, where larvae enter a period of hypobiosis, an identical expression is obtained for mean burden of adult parasites, but modified expressions are obtained for equilibrium host density and the mean burden of arrested parasite larvae:

$$H^* = \frac{\gamma d' d''}{\beta(\lambda\theta - d'd'')} \qquad (13),$$

$$M_A^* = \frac{A^*}{H^*} = \frac{d'(a-b)}{\theta(\alpha+\delta)} \qquad (14).$$

Here d' is defined as above, while $d'' = \mu_A+b+\theta+(a-b)\alpha/(\alpha+\delta)$. In both cases parasite burdens vary inversely with rates of parasite

induced reductions in host survival and fecundity. Equilibrium host population density closely resembles the expression for the threshold for establishment (Eqns 8,9; Fig 2), it varies inversely with both parasite fecundity, λ, and the rate at which parasite larvae are ingested, β. The numbers of free-living larvae varies directly with mean parasite burden and inversely with their rate of acquisition by the hosts.

DYNAMIC PROPERTIES OF THE MODEL.

The dynamic properties of the model may be explored by both numerical simulation and local stability analysis of the equilibrium (the details of the latter are given in Appendix 1). Initially, we consider the effects of larval biology on the dynamics of a basic grouse-nematode system in the absence of any parasite-induced effects on host fecundity. Numerical simulations of this simplest case show two important features of the host-parasite interaction. First, neither parasite-induced host mortality nor arrested larval development lead to oscillations in the abundance of either the hosts or the parasites (Figure 4). Secondly, larval arrestment has no effect on the levels of adult parasite burdens observed in the system, instead it leads to increases in the size of the host population exploited by the parasite (Figure 5). This increase occurs as hosts will live longer if the infective larvae enter an obligatory period of inert arrestment before developing to

pathogenic adults. Increased host life expectancy will roughly correspond to the period of larval arrestment.

Parasite-induced reductions in host fecundity.

Experimental manipulations of parasite burdens in grouse have demonstrated that T.tenuis reduces the breeding production of infected female grouse (Hudson 1986; Hudson, Newborn & Dobson 199*). The effects of parasite induced reductions in host fecundity on the dynamics of Model I and II are illustrated in Figure 6; both hosts and parasites now show a pronounced tendency to oscillate. More formally, it can be shown that in both models the numbers of hosts and parasites will cycle when the following inequality is trangressed (Appendix 1):

$$\alpha/\delta > \kappa \qquad (15).$$

The cycles are thus dependent on the relationship between the parasites effects on host survival and fecundity, and the degree of parasite aggregation within the host population. The destabilizing effects of parasite-induced reductions in grouse breeding production can only be offset by either high levels of parasite induced host mortality, or high levels of parasite aggregation. Neither of these are characteristic of the grouse-T.tenuis system.

The influence of larval biology on the dynamics of the system is more subtle. In Model I, stabilty requires

$$\gamma > d'k\left(\frac{\alpha+\delta}{\alpha-\delta k}\right) \qquad (16).$$

This inequality suggests that short larval life expectancy will tend to stabilize the system provided the inequality in equation 15 is satisfied. As larval life expectancy increases, cyclic oscillations of host and parasite are likely to become more pronounced.

Model II requires slightly more complex criterion for stability, the simplest condition is that

$$\frac{\alpha}{\delta} > \kappa\left(\frac{d'+d''}{d''}\right) \qquad (17).$$

These stability criteria for the two models are compared in Figure 7; the two models have complementary properties. In model I, population cycles require long-lived free-living larval stages (> 6 months). In contrast, shorter periods of larval arrestment (1 to 3 months) produce pronounced oscillations in Model II. In both cases low degrees of parasite aggregation produce violent oscillations that may be sufficiently large to drive both the host and the parasite to extinction. While increased survival of the free living stages increases the impact of the parasite on

the host, increased arrestment reduces the impact of the parasite on the host and ultimately leads to the grouse population breaking away from the parasites regulation and growing exponentially.

Grouse territorial behaviour.

The territorial behaviour of the grouse will tend to constrain the size of the host population, this effect may be included into the model by including an additional term into Equation 1 which reduces grouse fecundity rates as density rises.

$$dH/dt = (a-b-\Delta H)\,H-(\alpha+\delta)\,P \qquad (18)$$

Inclusion of the density-dependent term mimics a fall in the availability of suitable territories to a point where no further individuals can be recruited to the population (Davies, 1978; Paterson, 1980). In the absence of the parasite, the grouse will stabilize at carrying capacity K, where

$$H^* = (a-b)/\Delta \qquad (19).$$

Inclusion of the territorial behaviour of the grouse hosts reduces the amplitude of population fluctuations and leads to weakly damped cycles of host and parasite abundance in the absence of arrestment and sustained cycles when arrestment occurs. Curiously, longer periods of arrestment (> 6 months)

lead to more stable dynamics (Figure 8). Larval arrestment would seem to provide an interesting example of a time delay first destabilizing and then stabilizing a two species interaction!

The dynamics of the model that incorporates arrested development (model II) closely match the dynamics of the population at the Gunnerside study site (Figure 9). The structure of the cycles correspond to those observed at Gunnerside with a slow period of host population growth followed by a period of rapid decline. During the initial period of population growth, mean parasite burdens initially decline, then increase as host population size peaks; note that the parasite decline does not start until the host population crash is well advanced.

Factors determining the period of host-parasite cycles.

Standard analytical techniques may be employed to determine the length of the cycles observed in the model (Appendix 1). In model I the period of the cycles is primarily determined by the life expectancy of the parasite larval stages and the intrinsic growth rate of the grouse (Figure 10a). For the range of parameter given in Table 2, Model I tends not to show cycles in host and parasite abundance. In contrast Model II produces cyles with a time period of 4-8 years for a broad range of values of (a-b) and θ (figure 10b). This is a similar period to the cycle lengths of 4.8 years recorded by Potts, Tapper & Hudson

(1984). In both models increases in the intrinsic growth rate of the host population lead to decreases in cycle length. Decreases in the duration of larval arrestment (Model II), or in the life expectancy of free-living stages of the parasite (Model I), increase the period with which the host parasite system oscillates. These differences may account for the regional variation in cycle length recorded from grouse populations in different geographic locations (Hudson & Dobson, 1990).

DISCUSSION

The models developed by Anderson & May (1978) and May & Anderson (1978) predicted that under certain conditions, parasitic helminths could cause cyclic fluctuations in the abundance of their host populations. The application of these models to the empirical and experimental data collected on the grouse-T.tenuis system has demonstrated that when the impact of the parasite on host fecundity is greater than on grouse mortality the grouse population will tend to oscillate. This initial tendency to oscillate is enhanced if the larvae enter a period of arrested development immediately after infecting the host. The relatively low levels of parasite aggregation observed in the grouse-T.tenuis system increase the tendency of the system to oscillate.

An interesting point to emerge from the simulations (Fig 6,8) and the longitudinal study (Fig 9) was that mean parasite burdens did not peak until after the host population density had

started to decline. This result may explain why previous workers (e.g. Jenkins Watson & Miller 1963, 1967; Wynne-Edwards, 1986) have inadvertently assumed the parasites were a consequence rather than a cause of population cycles. Moss and Watson (1985) have proposed that cycles are adaptive irruptions generated from undiscovered intrinsic factors that may have developed to avoid outbreaks of trichostrongylid worms. It is difficult to see how adaptive irruptions could have evolved without invoking group selection (Wynne-Edwards, 1986). A more recent suggestion is that cycles could be generated through differential aggression between kin and non-kin coupled with inverse density dependent breeding success (Mountford et al 1990). However, simulations of this model demonstrate a number of fatal flaws, in particular the parameter values necessary to generate cycles require extreme levels of deference towards kin. In contrast, the study described here suggests that cyclic fluctuations in grouse may be generated by their interactions with parasites for wide ranges of parameter values. Variations in host population size and parasite burdens at different times in the cycle may produce artifactual results which suggest that mechanisms such as differential aggression or dispersal are driving the cycles (Moss, Watson & Rothery 1984; Moss & Watson 1985). This failure to differentiate cause from effect may be at the root of the controversies surrounding discussions of tetraonid cycles.

The dynamic behaviour of a four equation model that incorporates both types of larvae has also been explored. Its properties are essentially similar to that for the model that solely considers arrested larvae (Model II). This is fairly intuitive, as the dynamics of the free-living larvae enter the model in an essentially linear fashion; no real detail is thus lost by collapsing their stage of the life cycle down to give the transmission term used in Eqn 5.

Empirical studies of T.tenuis reveal pronounced seasonal variation in both the survival of the parasite's free-living stages and their propensity to arrest (Hudson & Dobson, 1990; Hudson, Newborn & Dobson, 199*; Watson, 1988; Shaw, 1988; Shaw, Moss & Pike 1989). The survival of free-living larvae is dependent on humidity and ambient temperature, while propensity to arrest is likely to be most pronounced as temperature variations become more severe both seasonally and geographically (Schad 197*). The models discussed in this paper assume these rates remain constant. In reality Model I corresponds to the situation operating in the spring and summer months while Model II corresponds to the autumn and winter months. A full description of the more complex seasonal models that include seasonal variation in the parasite's vital rates will be published subsequently (Dobson & Hudson, in prep). Although this more complex model has a richer array of dynamic behaviour (particularly on time scales operating within each calender

year), the main features of the analyses described above are not significantly altered by the inclusion of this seasonal forcing. Essentially the longer term dynamics are dominated by the parasites density-dependent impact on host fecundity and the propensity of larvae to arrest their development. These two factors interact to produce the population cycles characteristic of regulated systems which incorporate time-delays (MacDonald 1978; May 1972; Nunney 1985a,b).

Everything in this analysis suggests that interactions between red grouse and the parasitic nematode T.tenuis provide sufficient conditions for cycles to occur in grouse abundance. The definitive falsification of this hypothesis is to stop a population from cycling by controling or eradicating parasites. This experiment is being undertaken at a variety of locations and initial results are encouraging (Hudson, Newborn & Dobson, in prep). All previous hypothesis that seek to explain cycles in grouse abundance have been falsified when rigorously tested (Bergerud 1987; Hudson & Dobson 1990; Watson, Moss & Parr, 1984; Watson, Moss, Parr, Trenholm & Robertson, 1988). This implies that the presence of T.tenuis is the primary factor causing population cycles of grouse in the North of England. The ubiquitous presence of the parasite in other areas at similar high intensities (Hudson & Dobson 1990) suggest that it may be a significant factor in a number of areas in Scotland.

The results described here encourage examination of the potential role of parasites in other species which illustrate long term cycles in abundance. In particular, we caution that the subtle roles played by hypobiotic larvae and the parasites impact on host fecundity may have caused parasites to have been overlooked in studies seeking to determine why game populations cycle.

CONCLUSIONS

Interactions with the parasitic nematode T.tenuis generate population cycles in red grouse through their time-delayed impact on fecundity. Variations in cycle period between areas can be explained through changes in the propensity of larvae to arrest and changes in the growth rate of the grouse population. The period of the cycles varies directly with the duration of larval arrestment and inversely with host birth rates. When the life expectancy of the parasite free-living larvae is low, transmission of the parasite will be sporadic and grouse numbers will not cycle regularly. Under these conditions grouse density will be regulated by territorial behaviour and its interactions with other natural enemies, such as predators (Hudson & Dobson, 199*). Although the territorial "spacing behaviour" of the grouse stabilizes the more extreme dynamical effects of the arrestment, it does not generate population cycles in grouse.

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APPENDIX 1

This appendix outlines the formal stability analysis of Models I & II. The models may be simplified by grouping together sets of related parameters, here we set $c_1 = a-b$, $c_2 = \alpha+\delta$, $c_3 = \mu_P+b+\alpha$, $c_4=\alpha(k+1)/k$ and $c_5 = \mu_A+b+\theta$. Noting that $d' = c_3 + c_1c_4/c_2$, we can consider the fate of a small perturbation about the equilibrium, H^*, W^*, P^*. Writing $H_{(t)} = H^*+x_{(t)}$, $W_{(t)} = W^*+y_{(t)}$ and $P_{(t)} = P^*+z_{(t)}$, and linearizing by neglecting higher order terms, we obtain for model I (eqns 1-3):

$$dx/dt = c_1x - c_2z \qquad \text{(A1)}$$

$$dy/dt = -d'M^*x - \gamma\left(\frac{\lambda}{\lambda-d'}\right) + \lambda z \qquad \text{(A2)}$$

$$dz/dt = M^*(M^*c_4+d') + \frac{d'\gamma}{\lambda-d'}y - (d'+M^*c_4)z \qquad \text{(A3)}$$

The temporal behaviour of $x_{(t)}$, $y_{(t)}$ and $z_{(t)}$ then goes as $\exp(\lambda t)$, where the damping rates are given from eqns A1-A3 by the quadratic equation

$$\Lambda^3 + A\Lambda^2 + \Lambda B + C = 0 \qquad \text{(A4)}$$

In Model I, A, B and C are given by

$$A \equiv \gamma \left(\frac{\lambda}{\lambda - d'}\right) + d' + (a-b)\left(\frac{\alpha k'}{\alpha+\delta} - 1\right) \qquad (A5)$$

$$B \equiv \gamma \left(\frac{\lambda}{\lambda - d'}\right)(a-b)\left(\frac{\alpha k'}{\alpha+\delta} - 1\right) \qquad (A6)$$

$$C \equiv \gamma d'(a-b) \qquad (A7)$$

The Routh-Hurvitz criteria for local stability are that A, B and C all be greater than zero and that AB>C (May 1973). A and B will only be greater than zero when $\alpha/k > \delta$, this requires either k to be small or $\alpha > \delta$. These conditions are rarely met in the grouse - T.tenuis system. The further criteria AB>C is only met when

$$\left(\frac{\lambda}{\lambda - d'}\right)\left(\frac{\alpha/k - \delta}{\alpha+\delta}\right)\left[\gamma\left(\frac{\lambda}{\lambda - d'}\right)\right] > d' \qquad (A8)$$

when $\lambda >> d'$, this simplifies to

$$\gamma > d' k \left(\frac{\alpha+\delta}{\alpha - \delta k}\right) \qquad (A9)$$

Model II

The stability analysis for Model II follows similar lines, the stability-determining damping rates are now determined by the following expressions for A,B and C:

$$A \equiv d' + d'' + (a-b)\left(\frac{\alpha k'}{\alpha+\delta} - 1\right) \qquad (A9)$$

$$B \equiv d'd'' + (a-b)\left(d''\left(\frac{\alpha k'}{\alpha+\delta} - 1\right) - d'\left(\frac{\delta}{\alpha+\delta}\right)\right) - \frac{\theta H^* \lambda}{H^* + H_o} \qquad (A10)$$

$$C \equiv (a-b)\left(\frac{\theta H^* \lambda}{(H^*+H_o)}\left(\frac{H^*+2H_o}{(H^*+H_o)}\right) - d'd''\right) \qquad (A11)$$

The Routh-Hurvitz stability criteria still require $\alpha/\delta > k$, for A>0. More complex conditions now determine the sign of the second inequality; because $d'd'' \gtrsim \theta H^* \lambda/(H^*+H_o)$, B may be simplified to $\alpha/\delta > k(d'/d''+1)$. This requires high levels of aggregation (k<<1), or short periods of larval arrestment (θ>1), these conditions are rarely found in the grouse-T.tenuis system. The same substitution suggests that C<0. Although the requirement that AB>C is not easily simplified, extensive numerical explorations suggest it is always satisfied.

Periodicity of the cycles.

When the inequalities described in this appendix are transgressed the system has a propensity to oscillate. The period of these cycles will scale with $2\pi/w$, where $w=(s1-s2)\sqrt{3}/2$. Here $s1 = [r + (q^3 + r^2)^{1/2}]^{1/3}$, and $s2 = [r - (q^3 + r^2)^{1/2}]^{1/3}$, with $q = 0.33B - 0.11A^2$, and $r = 0.17(AB - 3C) - 0.037A^3$. Figure 9 in the main text illustrates the influence of host population growth rate and duration of larval arrestment on cycle length.

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Table 1. Notation used to denote various population parameters

Parameter	Description
a	Instantaneous birth rate of grouse (/host/unit of time).
b	Instantaneous death rate of grouse due to all causes except the parasite (/host/unit of time).
α	Instantaneous death rate of host due to the parasite (/parasite/host/unit of time).
δ	Instantaneous reduction in grouse fecundity due to the parasite (/parasite/host/unit of time).
Δ	Density dependent reduction in grouse fecundity and survival (host/host/unit of time).
λ	Instantaneous birth rate of parasite eggs (/parasite/unit of time).
μ_i	Instantaneous death rate of adult, μ_p, and arrested, μ_A, parasite stages in grouse (/parasite/unit of time).
γ	Instantaneous death rate of the free-living egg and larval stages (/parasite /unit of time).
β	Instantaneous rate of ingestion of parasite infective stages (/parasite/host/unit of time).
K	Parameter of the negative binomial distribution which measures inversely the degree of aggregation of parasites within the host population.
θ	The rate at which arrested larvae develop into adult worms. (/parasite/unit of time).

Table 2. Population parameters for T. tenuis and red grouse.

Parameter	Symbol	Estimated value
Grouse fecundity	a	0.6-3.0/grouse/year.
Grouse mortality	b	0.65/grouse/year.
Parasite fecundity	λ	10^4-10^5/worm/year.
Adult worm mortality	μ_p	0.65/worm/year.
Arrested larvae mortaliry	μ_A	0.1/worm/year.
Free-living larval mortality	γ	6.5-13/larvae/year.
Duration of arrestment	$1/\theta$	2-6 months.
Parasite pathogenicity	α	10^{-5}-10^{-4}/host/worm/year.
Parasite reduction in host fecundity	δ	$4x10^{-4}$/host/worm/year.
Aggregation of parasites in hosts	k	0.5 - 1.8.
Transmission rate	β	$6x10^{-5}$/larvae/host/year.

Table 2. The parameter estimates were obtained from: 'a' Hudson (1986); 'b' Jenkins, Watson & Moss (1963), Hudson (1986); 'λ' Hudson (1986b); Shaw, Moss & Pike (1989), Hudson & Dobson (in prep); 'μ_p and μ_A' Hudson, Newborn & Dobson (199*), Shaw & Moss (1989); 'γ' Watson (1988), Shaw, Moss & Pike (1989); '$1/\theta$' Shaw, Moss & Pike (1989); 'α, δ & k' Hudson (1986 a & b), Hudson, Newborn & Dobson (199*); 'β' this study.

Figure 1. Diagram of the life cycle of Trichostrongyle tenuis in the red grouse, Lagopus scoticus scoticus.

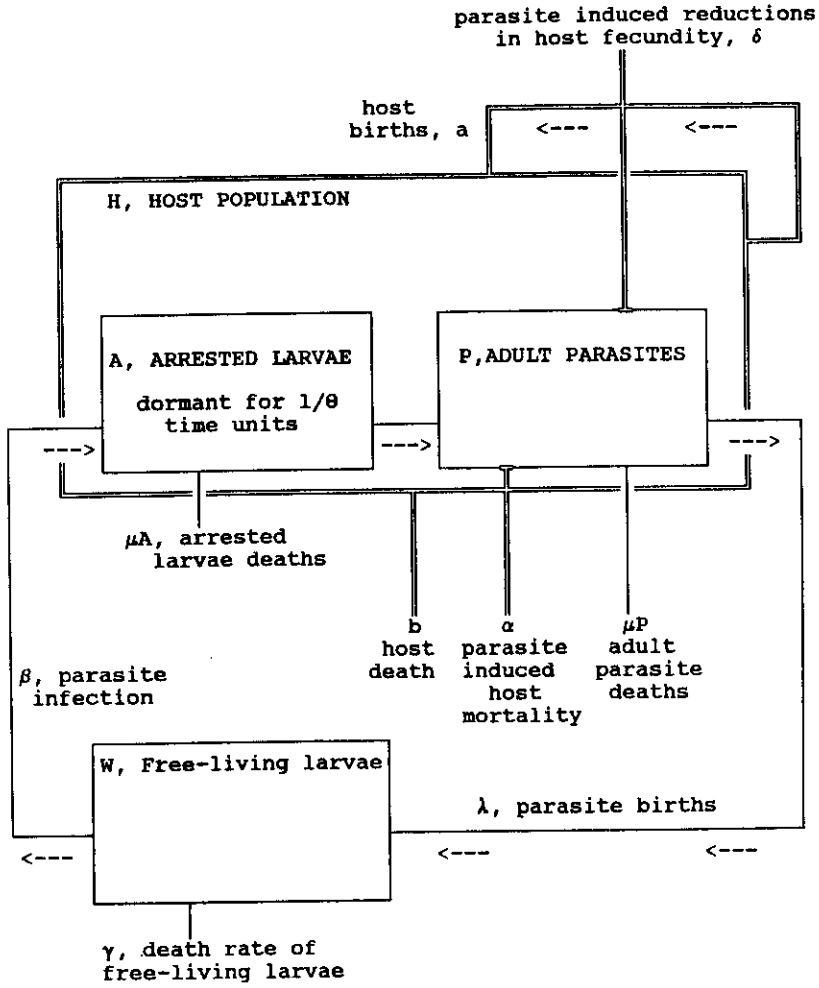


FIGURE LEGENDS

Figure 1. Schematic representation of the life cycle of T. tenuis in red grouse illustrating the different birth, death and transmission rates occuring in the life cycle and the notation used to denote each process. Note that parasites can affect both host survival and breeding production.

Figure 2. The effect of changes in the life expectancy of the free-living larvae on (a) the basic reproductive rate of the parasite and (b) the threshold host population required for parasites to establish. In both cases the results for Model 1 (no arrestment) are shown as solid lines and the results for Model 2 (arrested larvae) are shown as broken lines (all parameter values are as in Table 2).

Figure 3. The effect of duration of arrestment on the basic reproductive rate of T.tenuis (all other parameter values are as in Table 2.

Figure 4. The effect of duration of larval arrestment on host population size (parameter values as in Table 2).

Figure 5. Comparison of the dynamics of Models I & II in the absence of any effect of the parasites on host fecundity.

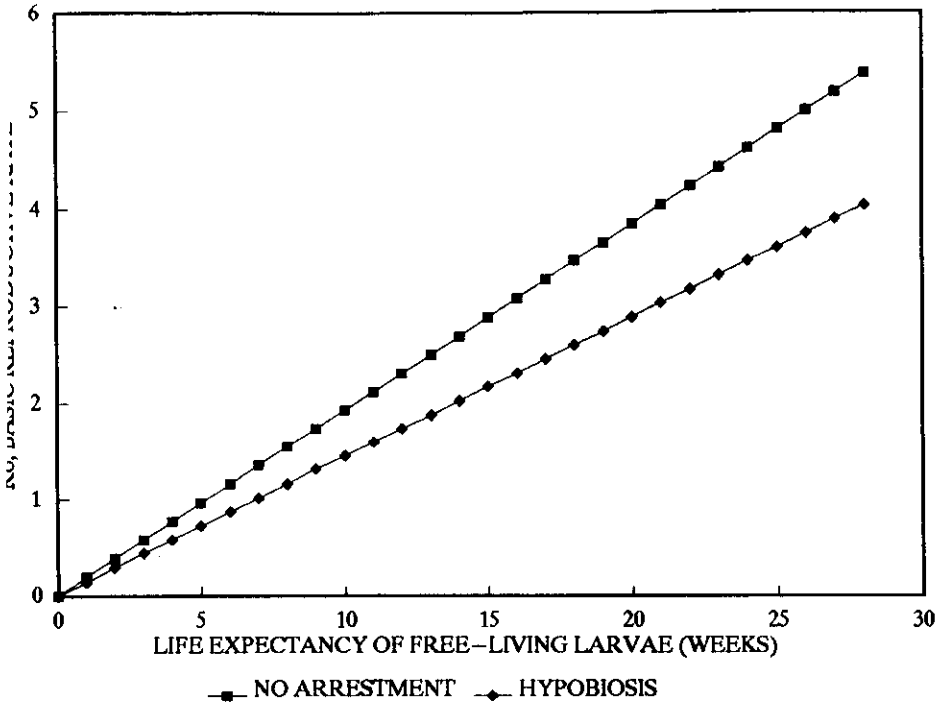
Figure 6. Comparison of the dynamics of the Models I & II when parasites affect host breeding production.

Figure 8. The effects of duration of arrestment on the dynamics of Models II. The upper figures illustrate the temporal dynamics, the lower figures illustrate the dynamic behaviour as phase diagrams.

Figure 9. (a) Changes in numbers of grouse shot and mean parasite burden over ten years at Guunerside. (b) The relationship between numbers of grouse shot and the the number of breeding hens at Gunnerside (r2 =).

Figure 10. The effect of larval life expectancy and host intrinsic population growth rate on the cycle length for (a) Model I, no arrestment, (b) Model II, arrested larvae.

Fig 2a


6
5
4
3
2
1
0
0
5
10
15
20
25
30
LIFE EXPECTANCY OF FREE–LIVING LARVAE (WEEKS)
NO ARRESTMENT
HYPOBIOSIS

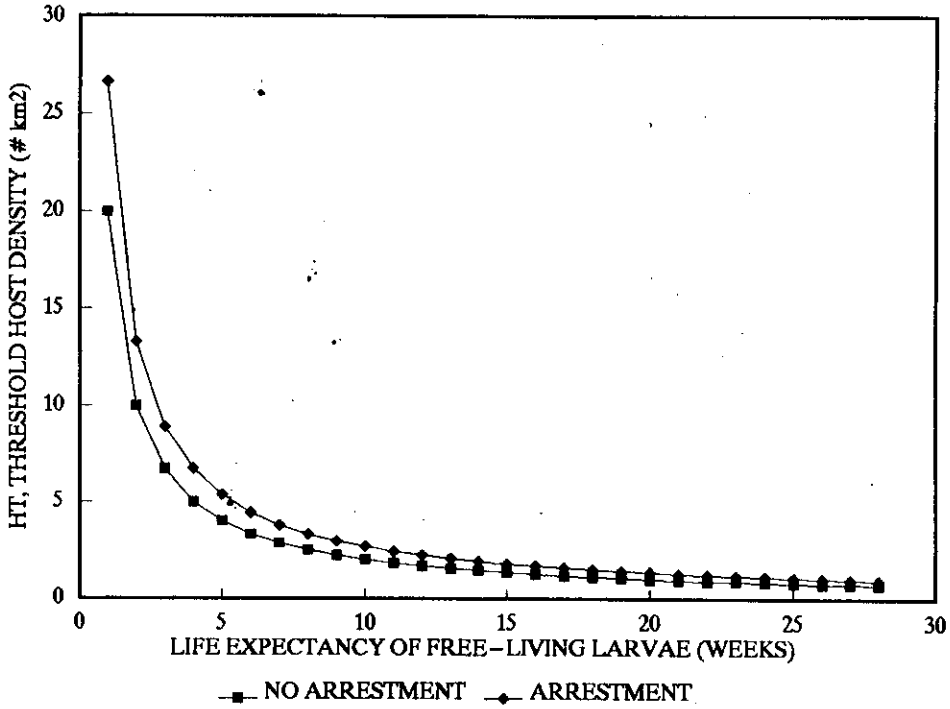
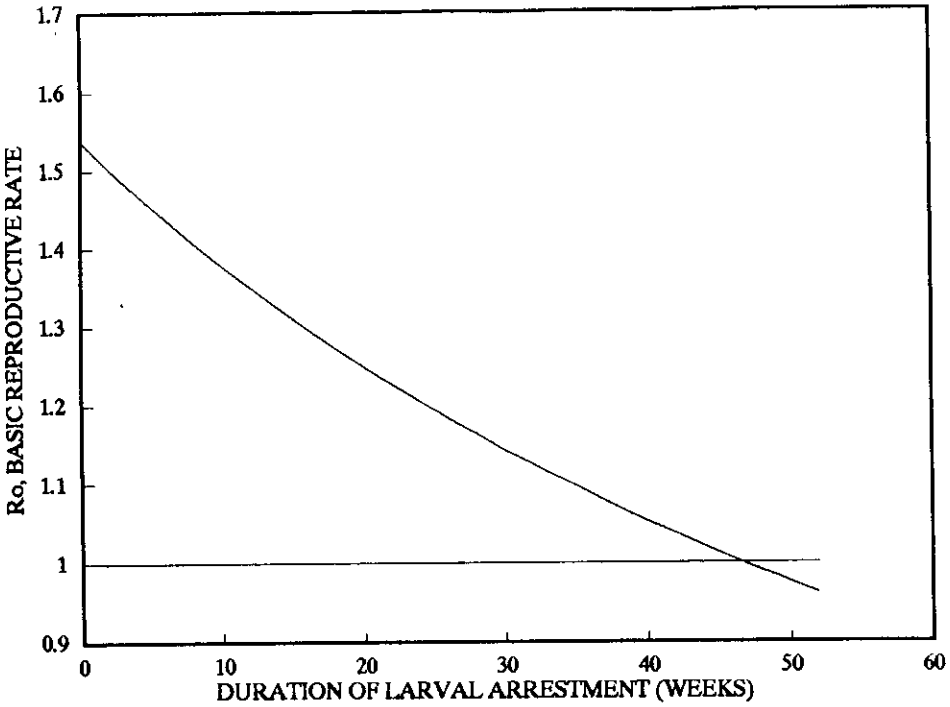
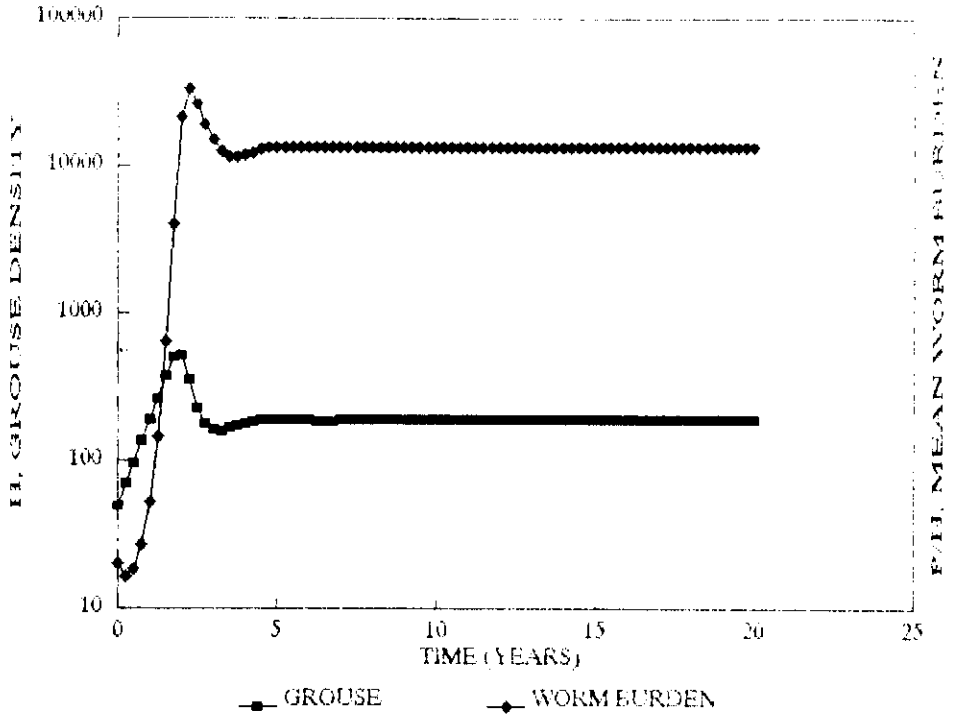
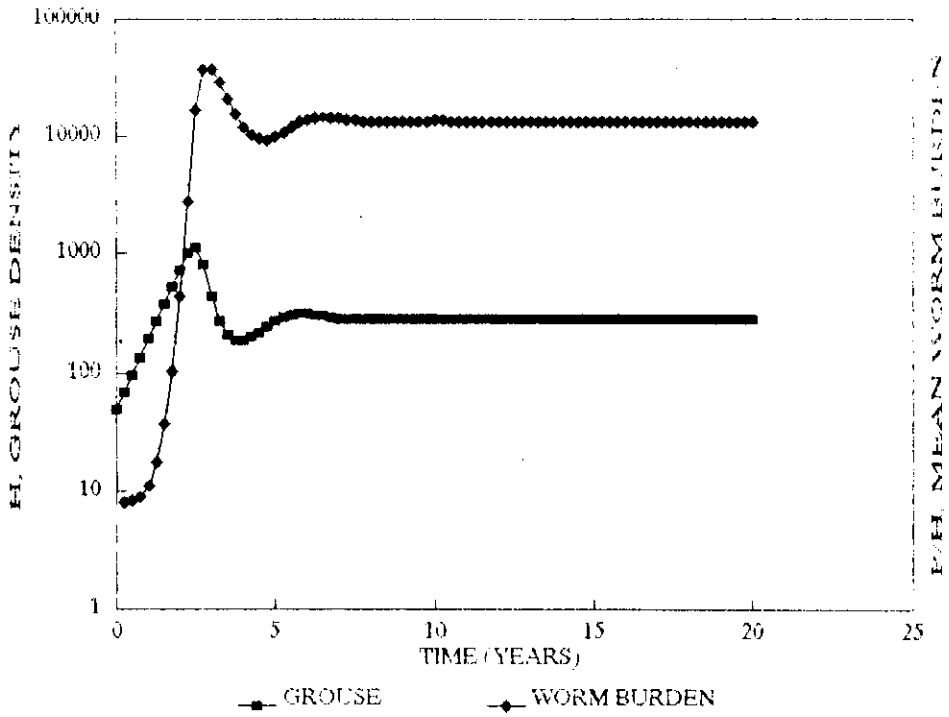

30
25
20
15
10
5
0
HT, THRESHOLD HOST DENSITY (# km2)
0
5
10
15
20
25
30
LIFE EXPECTANCY OF FREE–LIVING LARVAE (WEEKS)
NO ARRESTMENT
ARRESTMENT

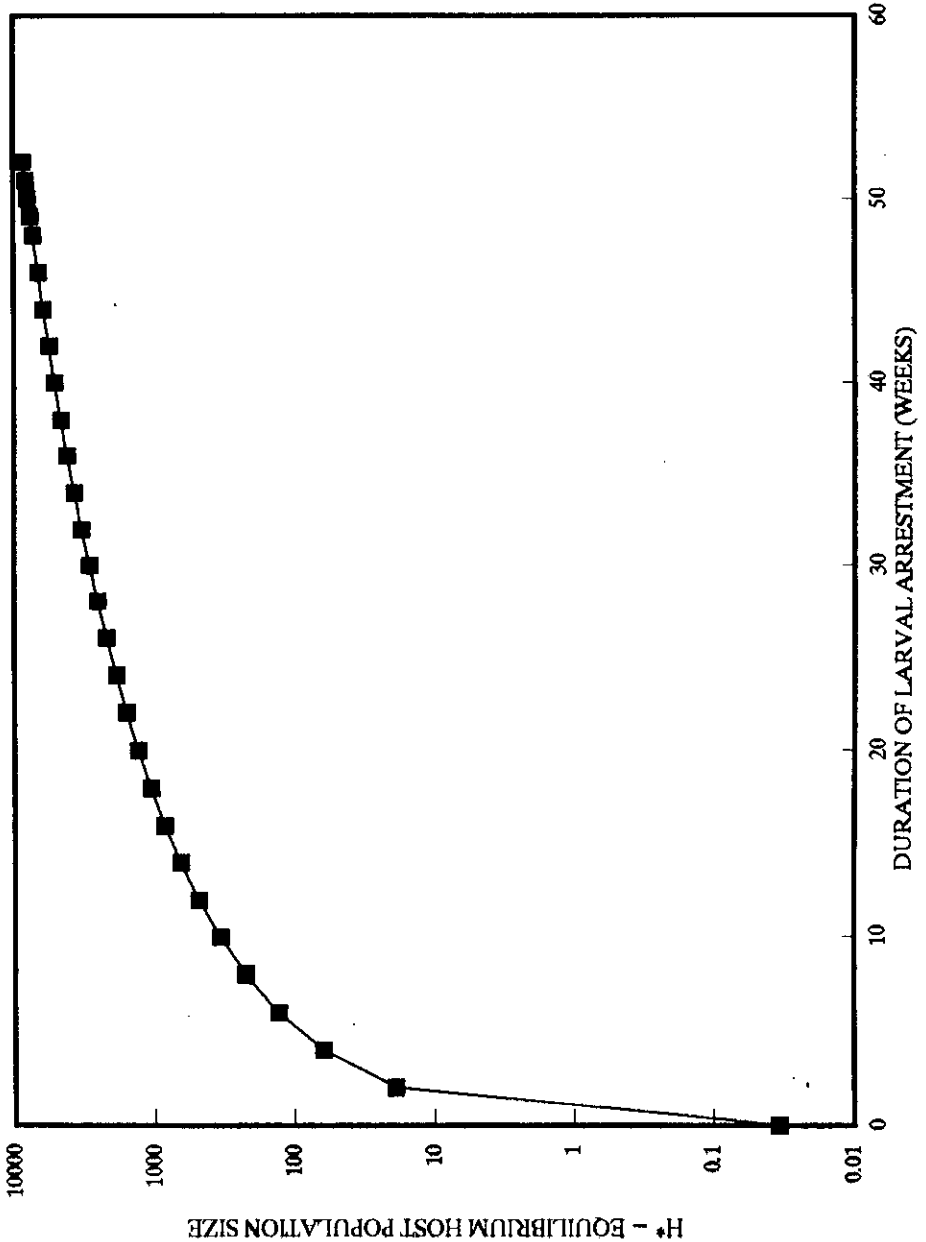
Fig 3

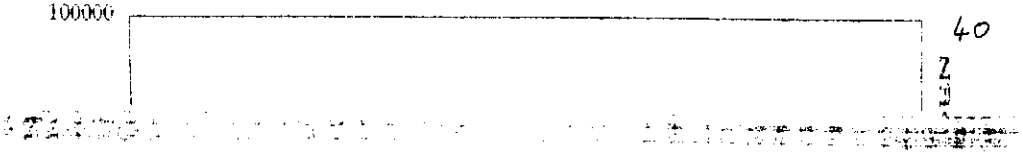

Ro, BASIC REPRODUCTIVE RATE
1.7
1.6
1.5
1.4
1.3
1.2
1.1
1
0.9
0
10
20
30
40
50
60
DURATION OF LARVAL ARRESTMENT (WEEKS)

4a


100000
10000
1000
100
10
0
5
10
15
20
25
TIME (YEARS)
GROUSE
WORM BURDEN

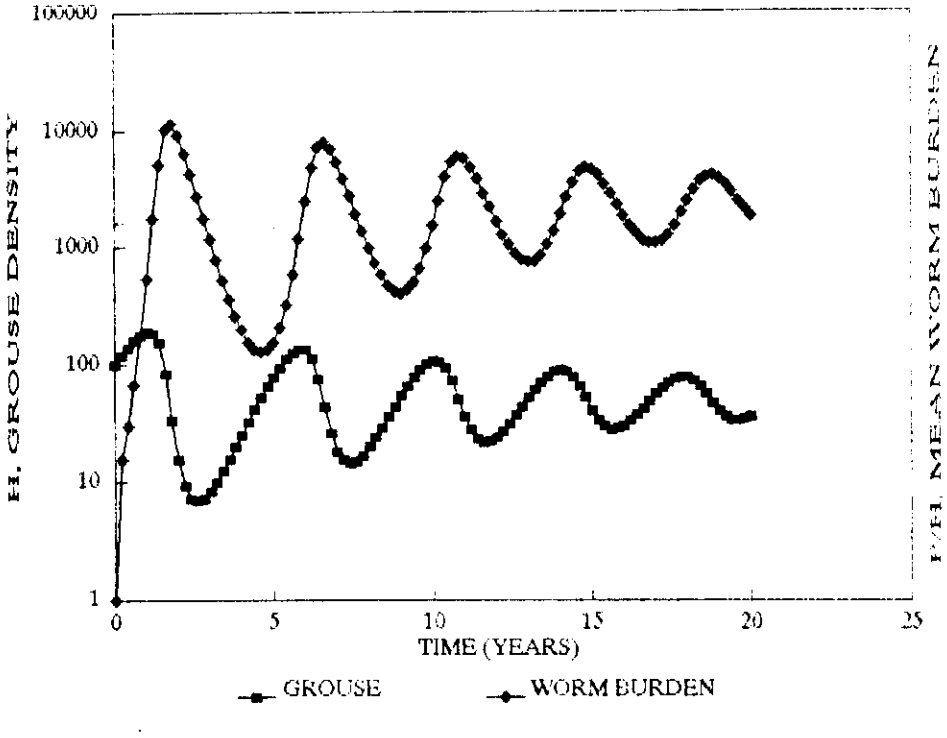

100000
10000
1000
100
10
1
0
5
10
15
20
25
TIME (YEARS)
GROUSE
WORM BURDEN


DURATION OF LARVAL ARRESTMENT (WEEKS)
0
10
20
30
40
50
60
H* – EQUILIBRIUM HOST POPULATION SIZE
10000
1000
100
10
1
0.1
0.01


100000

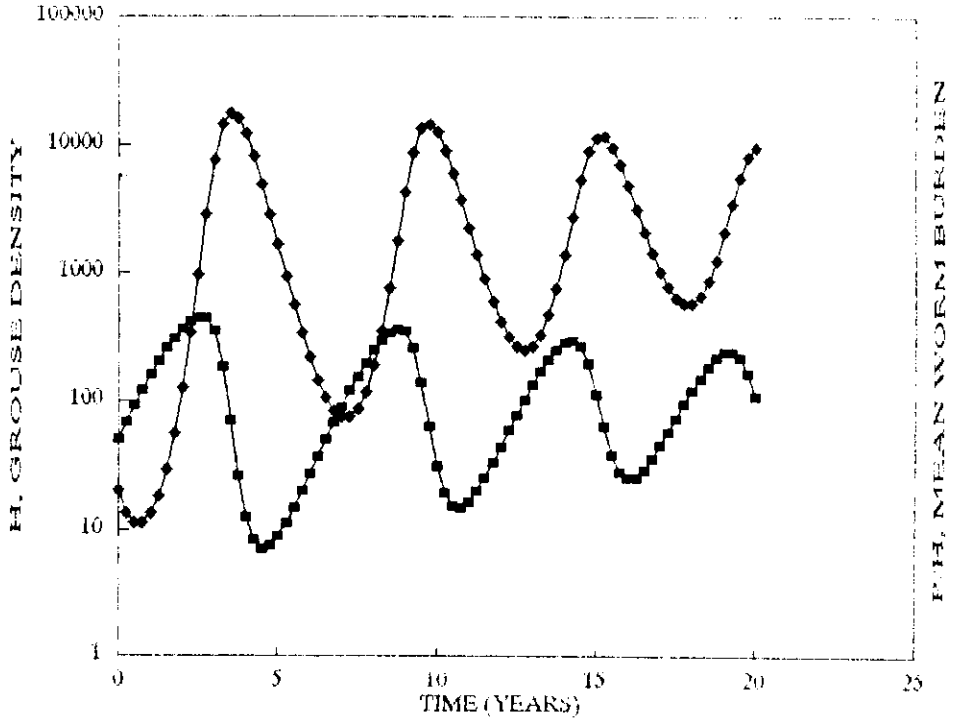
3

Fig 6a

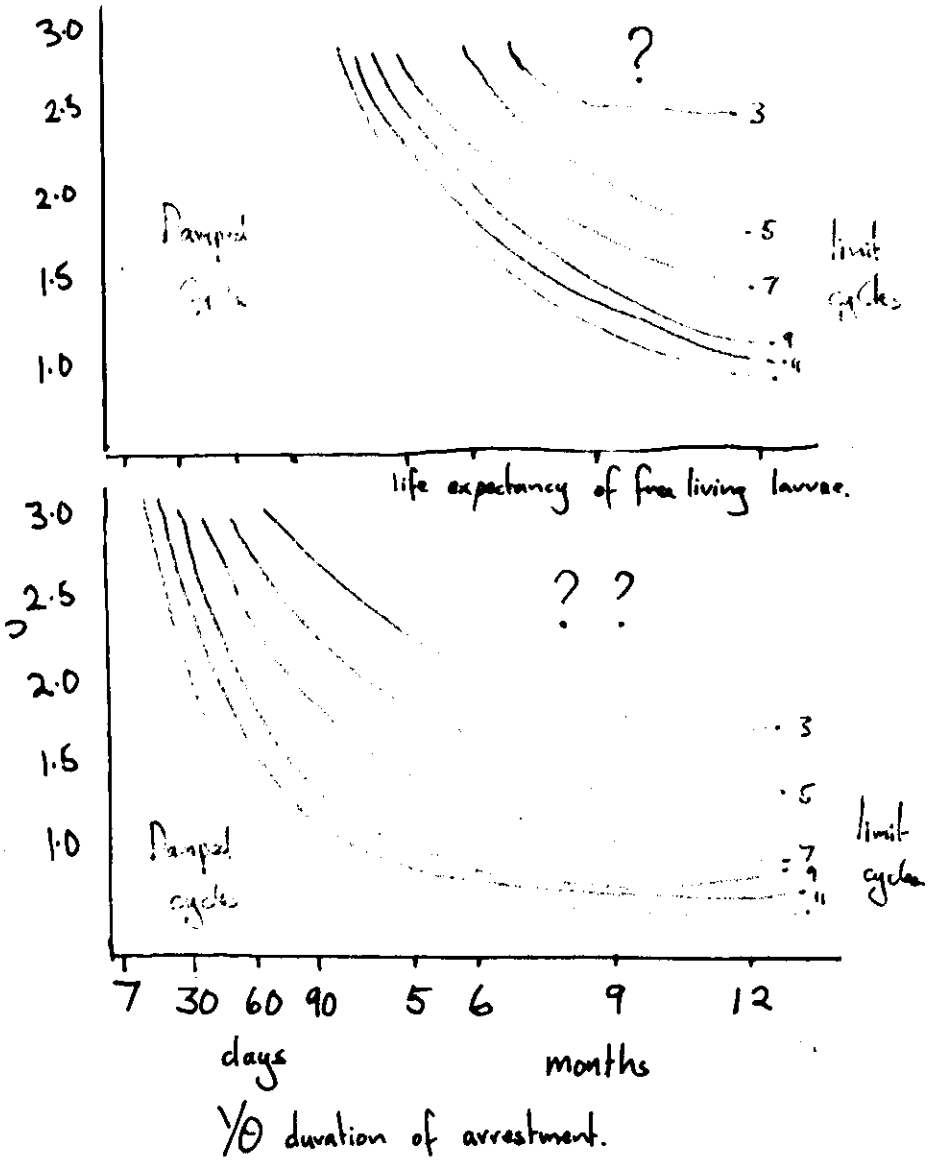


4

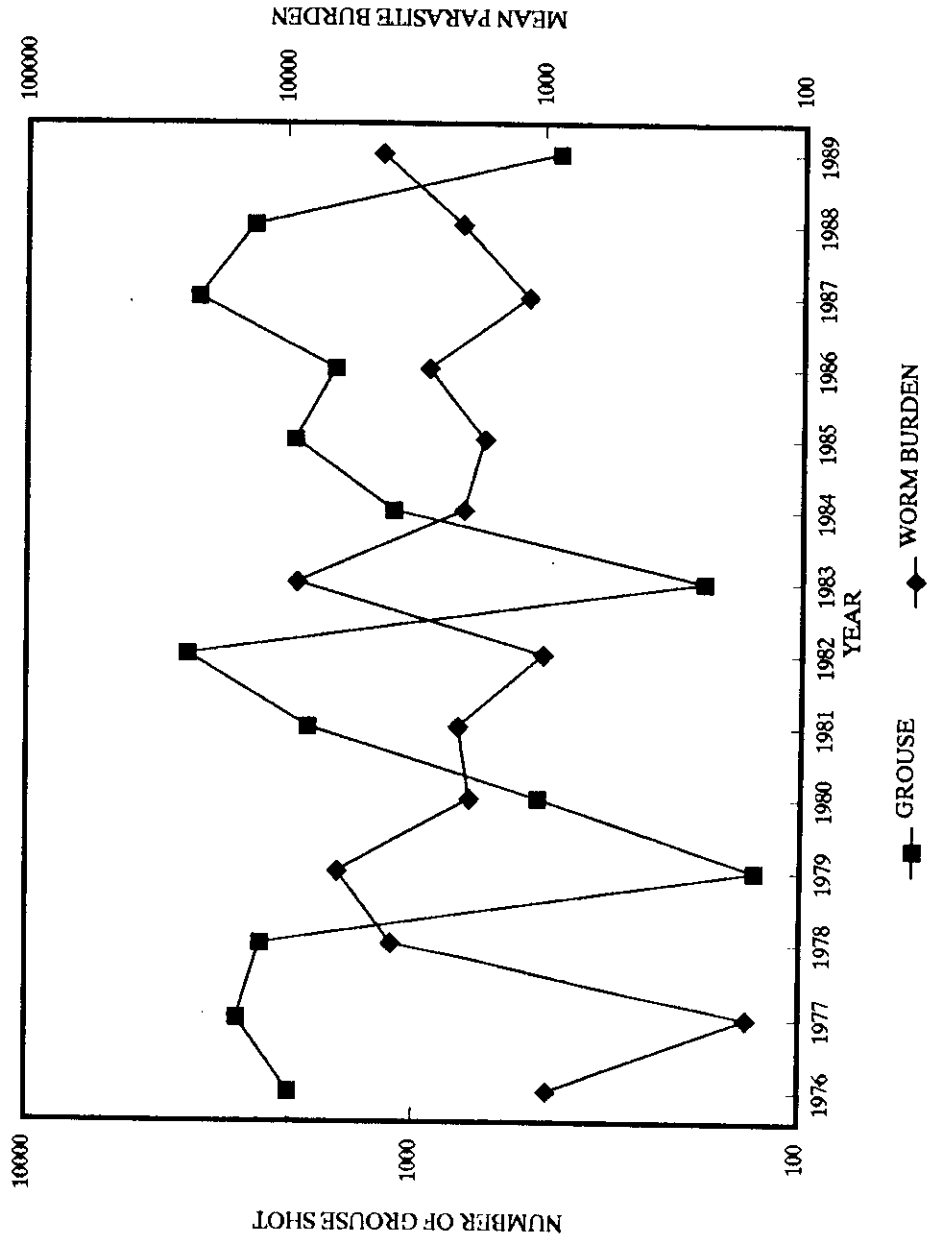
Fig 6b

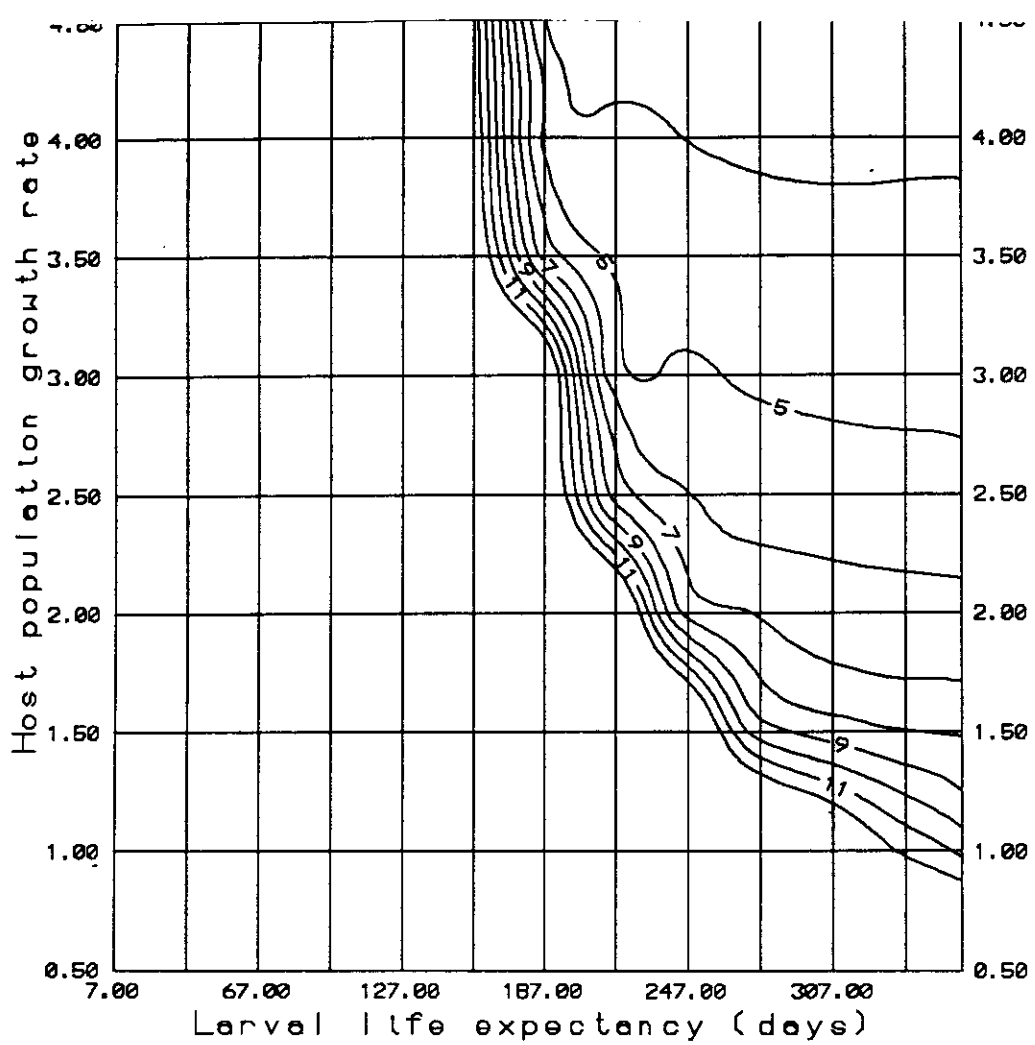


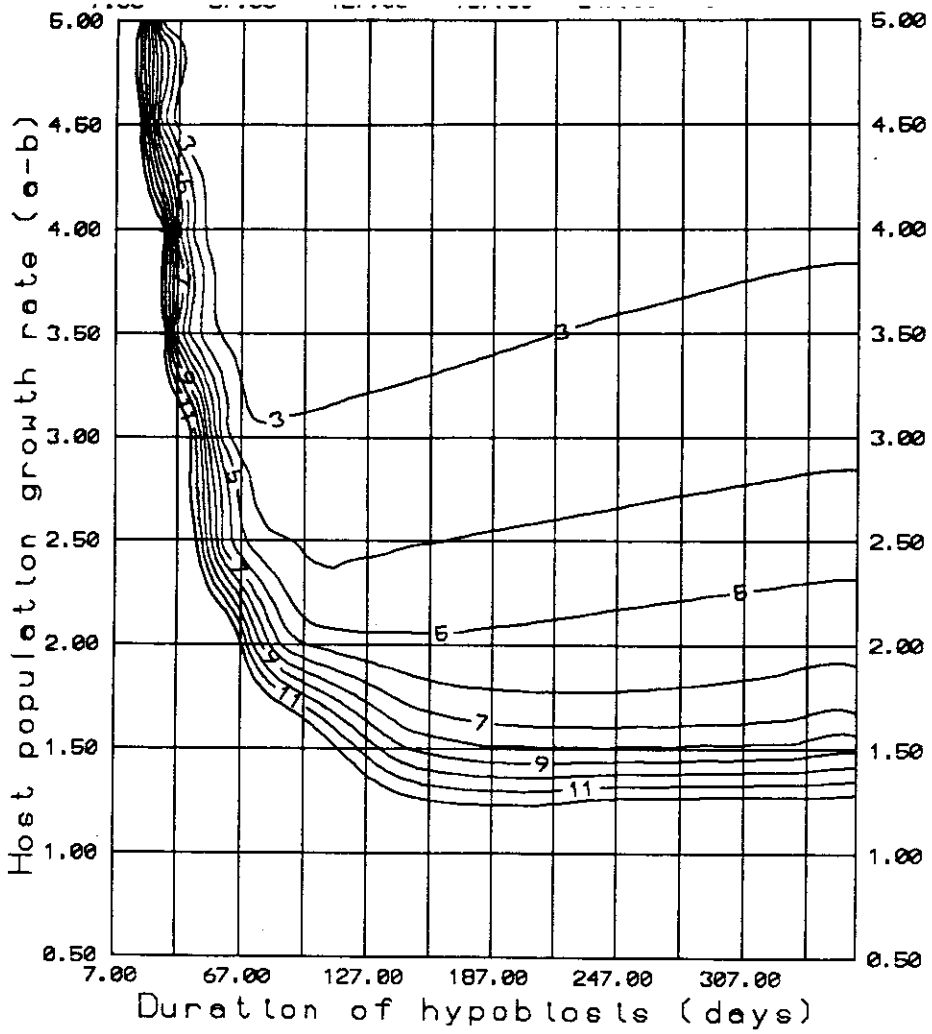
Dynamic properties of grouse-parasite model



?? ⇝ history depends on host regulation.




Host population growth rate
4.00
3.50
3.00
2.50
2.00
1.50
1.00
0.50
7.00
67.00
127.00
187.00
247.00
307.00
Larval life expectancy (days)
5
7
9
11


Host population growth rate (a-b)
5.00
4.50
4.00
3.50
3.00
2.50
2.00
1.50
1.00
0.50
7.00
67.00
127.00
187.00
247.00
307.00
Duration of hypoblosis (days)
3
5
7
9
11