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Gastrointestinal Nematode Parasites and the Stability and Productivity of Intensive Ruminant Grazing Systems

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Phil. Trans. R. Soc. Lond. B 1988 **321**, 541-563
doi: 10.1098/rstb.1988.0107

Phil. Trans. R. Soc. Lond. B **321**, 541–563 (1988)
Printed in Great Britain

Gastrointestinal nematode parasites and the stability and productivity of intensive ruminant grazing systems

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This paper uses mathematical models, describing the transmission dynamics of directly transmitted gastrointestinal nematode parasites of sheep and cattle, to examine the impact of these parasites on the stability and productivity of ruminant grazing systems. Current models of the ecology of grass growth under grazing, and the epidemiology of trichostrongylid nematode parasites of ruminants, are combined in a formulation that captures the general features of the plant – (ruminant) herbivore – parasite interaction. The simplest case, in which herbivore numbers are constant and not food limited (the norm for many agricultural systems) is considered in detail. The effect of gastrointestinal parasitism in reducing herbivore feeding rates is shown to act as a potential density-dependent constraint on the parasite's infection rate. The process is manifested in the model as a progressive linearization of the relation between herbivore feeding rate and plant density at the parasite equilibrium. This effect acts to stabilize the dynamics of the model grazing system and significantly affects its predictions about the impact of parasite control and the pattern of host productivity. Model predictions are discussed in the light of relevant field observations, and areas for future research are identified.

1. Introduction

The profound importance of ruminant grazing systems in world food production is reflected in a large and diverse literature examining their ecology (Coop 1982; Crawley 1983). One of the areas in which population biology has been most effectively applied to these systems is in considering the dynamics of the plant–herbivore relation and its effects on herd management and productivity (Morley 1966; Noy-Meir 1975). Broadly speaking, grazing systems may be divided between two extremes, depending on the level and variability of herbivore density. In *intensive* pasture systems, where herbivore numbers are strictly managed, the important problems are the maximization and maintenance of herd productivity and the avoidance of overgrazing (Morley 1966; Noy-Meir 1975; Coop 1982). In *extensive* range systems, where herbivore abundance is allowed to fluctuate more or less naturally, population biologists have also applied predator–prey theory in assessing the stability of the plant–herbivore interaction and, in particular, the propensity of the system to oscillate through time (Rosenzweig & MacArthur 1963; May 1973, 1981; Crawley 1983). A unifying theme throughout grazing-systems ecology is the central role of herbivore feeding rate and population density in influencing the system's behaviour (Noy-Meir 1975).

Another major effect on ruminant productivity, which has been particularly studied in intensive systems, is the impact of helminth parasites, and in particular of directly transmitted gastrointestinal nematodes of bovids (Armour & Ogbourne 1982; Williams 1983; Coop 1982). These parasites cause significant morbidity and mortality in bovid populations, and the resulting worldwide production losses due to parasitic gastroenteritis (PGE) are considerable

(Williams 1983; Smith *et al.* 1987*b*; Smith & Grenfell 1985). As a result, there is a large body of both field and experimental work on the epidemiology of PGE and, in particular, on the population biology of the abomasal roundworm of cattle *Ostertagia ostertagi* (Armour & Ogbourne 1982; Smith & Grenfell 1985). Most of the theoretical developments in this area have involved the construction of models for particular systems (Gettinby *et al.* 1979; Callinan *et al.* 1982; Paton & Grettinby 1985; Thomas *et al.* 1986). Recently, general mathematical models for the interaction of helminth parasites and their hosts (Anderson & May 1978*a*, *b*, 1985) have also been applied to the ruminant–nematode interaction (Grenfell *et al.* 1987*b*). Both types of models aim to provide a theoretical framework for understanding the epidemiology of PGE.

A number of experimental and field studies indicate that one component of production losses owing to PGE is a significant reduction in food consumption rates by the host, which may occur even at subclinical levels of infection (Coop *et al.* 1977, 1982, 1985; Randall & Gibbs 1981; Symons 1985). Because as indicated above, the level of food consumption by ruminants is also an important variable in the population biology of the plant–herbivore interaction, a parasite-induced reduction in host food consumption could have a significant effect on the overall dynamics of the system. This paper assesses the ecological properties of the plant–ruminant–nematode interaction by means of a simple mathematical model. It uses a review of existing formulations for the plant–herbivore and host–parasite interactions to construct a new model that allows for the effects of nematode parasitism on ruminant feeding rate and productivity. The simplest and most well documented case, an intensive pasture system where herbivore density is kept constant, is considered in detail. Analytical and numerical examinations of the model's behaviour are used to assess the possible effects on the stability and productivity of the grazing system of gastrointestinal nematodes, and their control by anthelmintic chemotherapy. Comparisons of these results with relevant field and experimental data are made where the latter are available.

The paper is divided into four sections. The first provides quantitative reviews of relevant work on the population biology of plant–herbivore and host–parasite interactions. The second section uses this information to derive a simple model for the plant–ruminant–nematode interaction in intensive grazing systems. It then goes on to explore the equilibrium and dynamic properties of the model. The third section compares these results with relevant field observations, and the final section provides a general discussion and suggests directions for future work.

2. Plant–herbivore and host–parasite population biology

(*a*) *The dynamics of plant – (ruminant) herbivore interactions*

The study of grazing-system dynamics in population ecology derives from the seminal work of Rosenzweig & MacArthur (1963) on the properties of simple predator–prey systems. Since then, a number of workers have developed this description of the interaction between predators and prey (Holling 1965, 1966; MacArthur & Connell 1966; May 1973, 1981; Noy-Meir 1975). The major application of this general theory to intensive grazing systems is by Noy-Meir (1975). He describes the dynamics of these systems in terms of a variable biomass V of vegetation per unit area, which is fed upon by a constant density of herbivores, H. Ignoring

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seasonal effects and other complications, the plant dynamics can be represented by a simple differential equation

$$\frac{dV}{dt} = G(V) - c(V)H, \tag{1}$$

where $G(V)$ is the net growth rate of plant biomass in the absence of herbivory and $c(V)$ is the consumption rate per head of plant biomass by herbivores; both rate processes are expressed as functions of V. In general, $G(V)$ is a convex function of V, which rises with V to a maximum and then declines, reflecting the effects of shading at high plant abundance (Crawley 1983; Johnson & Parsons 1985). Noy-Meir adopts a simple quadratic (logistic) function of V,

$$G(V) = gV(1 - V/V_{max}), \tag{2}$$

to describe this process. According to equation (2), G increases to a maximum at $V = V_m = V_{max}/2$ and then declines symmetrically to zero at $V = V_{max}$ (figure 1*a*).

The general form of the relation between herbivore consumption per head and plant biomass for ruminants is also shown in figure 1*a*. Feeding rate increases comparatively rapidly with V at low plant biomass, and then reaches a plateau at high V, corresponding to a saturation in digestion rate (Noy-Meir 1975). In the terminology of predator–prey theory, the general shape of this consumption curve is defined as the *functional response* of the herbivore to changes in plant density (Holling 1965, 1966; May 1981). For ruminants, the functional response generally shows a sigmoid increase with food abundance (the 'Type III' functional response of Holling (1966)), reflecting a peak in feeding rate per unit plant biomass (c/V) at K (Crawley 1983). The sigmoid functional response can be represented by the simple equation (Crawley 1983)

$$c(V) = c_{max}\frac{V^2}{(V^2 + K^2)}, \tag{3}$$

where c_{max} is the asymptotic maximum consumption rate per herbivore, and the constant K controls the shape of the curve (alternative but essentially similar formulations are reviewed by May (1981)).

The system's dynamics depend on the relative shapes of the net plant growth ($G(V)$) and herbivore consumption ($C = c(V)H$) curves (Rosenzweig & MacArthur 1963; Noy-Meir 1975). From equation (1), intersections of the G and C curves represent equilibria of the system. The main alternatives regarding the stability of these steady states at different levels of peak herbivore feeding rate ($c_{max}H$, and therefore stocking rate, H) is summarized in figure 1. Essentially:

(i) at low stocking rates $G > C$ up to a single stable equilibrium at high plant abundance;

(ii) at very high stocking rates, the system is overgrazed and collapses to a low, stable equilibrium, which may be above zero owing to the sigmoid functional response;

(iii) at intermediate stocking rates, both upper and lower stable steady states may exist, separated by an unstable equilibrium; the fate of the system depends on its initial condition.

Noy-Meir (1975) discusses these alternatives, and others such as the occurrence of ungrazeable plant reserves, in more detail. Much more refined grazing models, which take explicit account of photosynthesis, leaf growth and herbivore feeding rate as functions of leaf area index, have recently been developed (Johnson & Parsons 1985). Broadly speaking, these formulations exhibit similar qualitative patterns of behaviour to equations (1)–(3).

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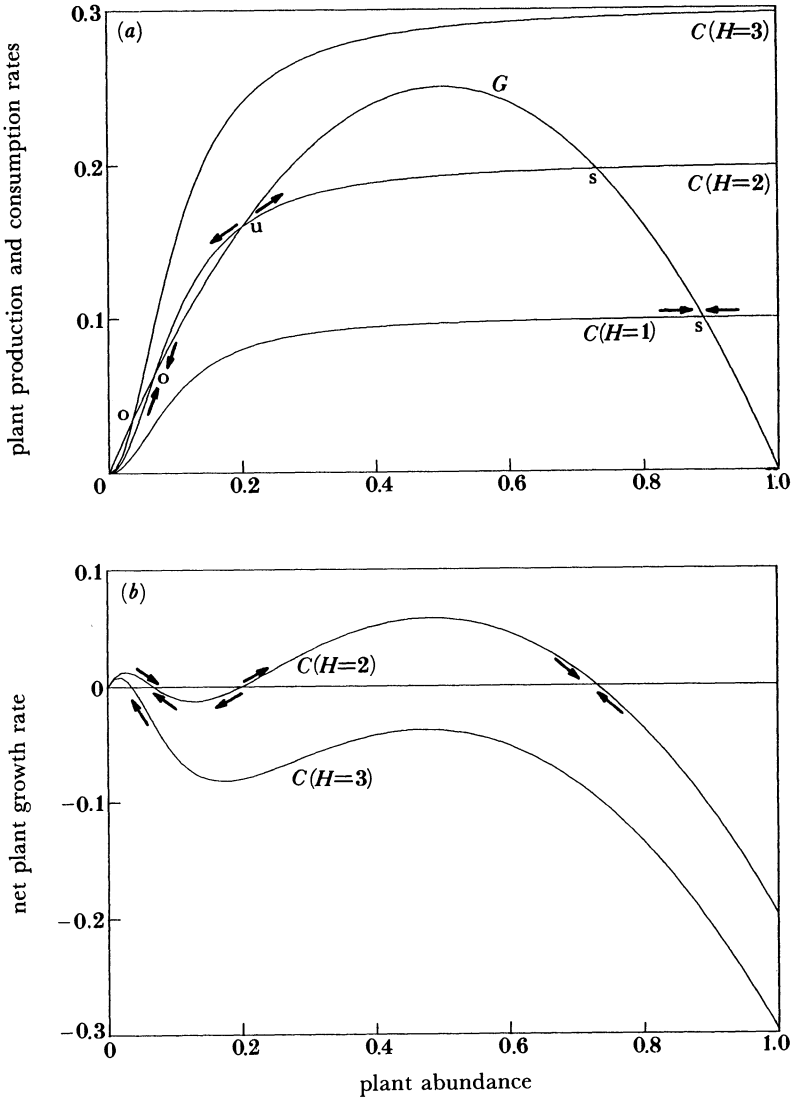

FIGURE 1. Basic dynamics of intensive grazing systems, as predicted by equations (1)–(3). (*a*) Net plant production rate, G, and herbivore consumption rates, C (at various stocking densities, H), as functions of plant abundance, V (other parameters, $V_{max} = 1$, $g = 1$, $c_{max} = 1$, $K = 0.1$; typically, $K = 0.1\,V_{max}$ for ruminant herbivores (Johnson & Parsons 1985)). The intersection of G and C curves represent various types of population equilibria: (i) stable undergrazed equilibria (s) at high plant abundance (essentially above $V_{max}/2$); (ii) stable overgrazed equilibria (o) at low plant abundance; (iii) unstable equilibria (u). The arrows summarize the dynamics around these points. (*b*) Net plant growth rate curves (G–C), corresponding to $H = 2$ and $H = 3$.

(*b*) *The host–parasite interaction*

Parasitic gastroenteritis in ruminants is caused by a variety of nematode species (Levine 1980). Among the most economically important of these, and certainly the best documented epidemiologically, are the trichostrongylids, and in particular *Ostertagia ostertagi* in cattle and *Haemonchus contortus*, *O. circumcincta* and *Trichostrongylus colubriformis* in sheep (Coop 1982; Williams 1983). The following epidemiological account is therefore based mainly on these species.

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(i) *Life cycle and population biology*

General reviews of the population biology of these parasites are given by Armour & Ogbourne (1982), Coop (1982) and Smith & Grenfell (1985). They have a comparatively simple direct life cycle.

(i) Adult parasites, in the host's abomasum or small intestine, release eggs, which are passed out in the faeces.

(ii) These hatch and develop through preinfective larval stages into infective larvae, which are progressively translated from faeces to pasture.

(iii) The infective larvae are characteristically long-lived (Grenfell *et al.* 1987*a*, *b*) and can tolerate a wide range of environmental conditions. A proportion are ingested by hosts and develop through to adult parasites, completing the life cycle. Maturation normally takes around 18 days (Grenfell *et al.* 1987*b*), but may be extended to 16–18 weeks if the larvae undergo an arrestment diapause (Armour & Bruce 1974; Grenfell *et al.* 1987*b*).

However, the available experimental and field evidence (reviewed by Grenfell *et al.* (1986, 1987*a*, *b*) and Smith *et al.* (1986, 1987*a*, *b*)) indicates that the various rate processes (developmental, reproductive, survival, etc.) that control the transitions between stages, and the stage-specific pattern of mortality, are functions of a number of other physical and ecological variables. These can be classified broadly into the following categories.

Density-independent processes, notably micrometeorological influences on the development and mortality of the free-living stages (Grenfell *et al.* 1986; Smith *et al.* 1986; Young & Anderson 1981).

Density- and infection-dependent processes, in particular a decrease in the establishment, survival and reproductive rates of adult parasites as the host's experience of infection accumulates (Michel 1969, 1970; Barger 1987; Barger *et al.* 1985; Grenfell *et al.* 1987*a*; Smith *et al.* 1987*a*).

The density-dependent processes are likely to be particularly important in regulating trichostrongylid populations (Barger 1987). Figure 2 summarizes the results of a quantitative analysis of density-dependent limitations on the survival and fecundity of adult *O. ostertagi* in calves (Grenfell *et al.* 1987*a*; Smith *et al.* 1987*a*), based on the important experimental work in this area done by Michel (1969, 1970). The results indicate a marked decrease in the survival and effective reproductive rates of adult parasites as the host's experience of infection increases; these effects have also been measured in experimental infections of sheep with *H. contortus* (Barger *et al.* 1985). Grenfell *et al.* (1987*a*) combined models for the various density-dependent and -independent rate processes in *O. ostertagi*'s life cycle in a deterministic differential equation framework, which represents a preliminary model of the epidemiology of ostertagiasis in calves. Their results illustrate the efficacy of strategic anthelmintic application in controlling the disease, and the role of density-dependent and -independent processes in generating observed epidemiological patterns. In particular, infection-dependent reductions in egg counts (figure 2) appear to have a potentially profound effect in regulating both the abundance of infective larvae on the pasture and adult worm burdens. This importance of density-dependent regulation in trichostrongylids is broadly confirmed by the results of other modelling studies (Gettinby *et al.* 1979; Callinan *et al.* 1982; Paton & Gettinby 1985; Thomas *et al.* 1986).

As with most helminth parasites (Anderson & May 1985), trichostrongylid population

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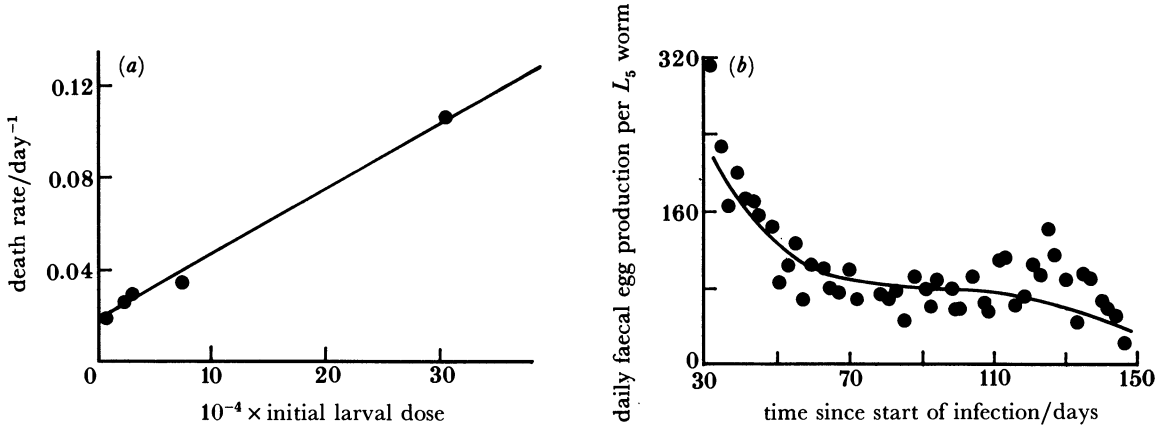

FIGURE 2. Experimental evidence for density-dependent constraints on the population biology of *O. ostertagi*. (*a*) Instantaneous per head adult death rate (μ), estimated from single infections; μ increases linearly with the size of the initial larval dose (Anderson & Michel 1977). (*b*) Average daily faecal egg production per L_5 worm (effectively per adult worm), in calves dosed with 500 infective larvae per day. The curve represents the fit of a model that expresses fecundity as a decreasing function of worm burden and infection duration (Smith *et al.* 1987*a*).

processes generate overdispersed frequency distributions for the abundance of both free-living (Raynaud & Gruner 1982) and parasitic stages (Barger 1985). The available evidence for adult distribution (negative binomial $k = 1.4$ for trichostrongylids in sheep (Barger 1985); $k > 2$ for *O. ostertagi* in cattle (Smith *et al.* 1987*a*), indicates that worm burdens are rather less overdispersed than for gastrointestinal nematode parasites of humans (Anderson & May 1985).

(ii) *Effects of parasitism on ruminant nutrition and productivity*

Because the economically important variable associated with veterinary parasitoses is production loss, the effects of parasites on the nutrition and growth rates of farm animals have been particularly well studied (Coop 1982; Williams 1983). In particular, a number of workers have measured significant effects of trichostrongylid nematodes on the productivity of ruminants, in both laboratory (Coop *et al.* 1977, 1982) and field studies (Armour & Ogbourne 1982; Coop *et al.* 1985; Randall & Gibbs 1981). These studies indicate that trichostrongylids inhibit host weight gain (particularly of young animals) by reducing both food intake (Symons 1985) and the gross efficiency of utilization of metabolizable energy for growth (EMG) (Sykes & Coop 1977). The relative influence of these two effects has been demonstrated most clearly in a series of experiments involving repeated trickle infections of sheep with various trichostrongylids (Coop *et al.* 1977, 1982; Sykes & Coop 1977; Sykes *et al.* 1979). The most detailed data were collected for *O. circumcincta* infections (Coop *et al.* 1977, 1982). These are summarized in figure 3, which depicts a significant decrease in lamb weight gain (compared with uninfected controls) with daily infection rate. This effect is caused by progressive (and prolonged) reductions in EMG and food intake with infection rate; at an infection rate of 5000 larvae per day, food intake is reduced by 12% and 20% in different experiments. These reductions appear to be due to abomasal damage caused by the emergence of larvae, although acquired immunity is a complicating effect (Sykes & Coop 1977). The authors stress that these

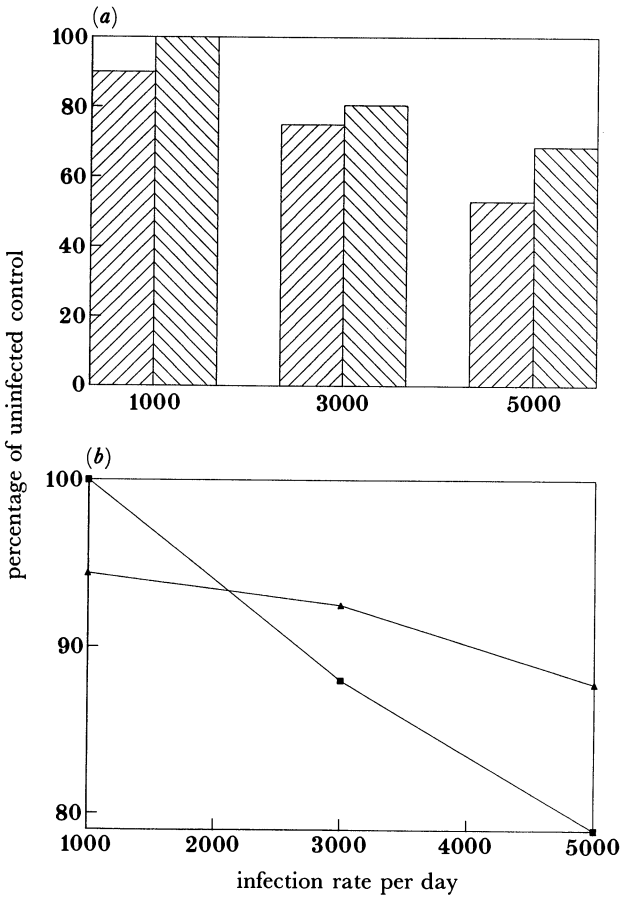

FIGURE 3. Effects of daily trickle infection with *O. circumcincta* on the productivity and feeding rate of lambs. (*a*) Weight gain and gross efficiency of use of metabolizable energy for growth of hosts at various infection rates, compared with these parameters in uninfected controls (Coop *et al.* 1982). ▨, Weight gain; ▧, efficiency of EMG. (*b*) Percentage reduction in average feeding rate, at various infection rates, compared with uninfected controls. ■, Coop *et al.* (1977); ▲, Coop *et al.* (1982).

are *subclinical* levels of infection; clinical infection can cause much more severe transient anorexia, especially in young animals (Murray *et al.* 1970; Symons 1985).

The extrapolation of these experimental results to an assessment of production losses in field infections depends on the relation between infective larval abundance on the pasture and their uptake by the host. This is a complex process, which is influenced by the patterns of larval distribution and herbivory and other variables (Crofton 1952; Kloosterman 1971; Michel 1976). In the context of grazing dynamics, the most important effect is the 'diluting' effect of grass growth on the density (and therefore availability for ingestion by hosts) of a given pasture abundance of larvae. Although several authors have noted the importance of this effect (Armour & Ogbourne 1982; Crofton 1952; Michel 1976), there does not appear to be sufficient published data to quantify it in detail (Kloosterman 1971). The implications of different models for larval dilution will be discussed below.

3. Model structure and predictions

(a) *The model*

To assess the effects of nematode parasitism on the grazing system model presented in equations (1)–(3), we require a simplified quantitative description of the epidemiological picture presented above. This is based on a general model framework for the population biology of directly transmitted macroparasites, proposed by Anderson & May (1978*a*, *b*, 1985). Dividing the parasite population into two compartments, comprising adult parasites (A), within the host population (H), and free-living transmission stages (L), the following pair of rate equations

$$\frac{\mathrm{d}L}{\mathrm{d}t} = \lambda - (\rho + \beta H)\, L, \tag{4}$$

$$\frac{\mathrm{d}A}{\mathrm{d}t} = \beta H L - \mu A, \tag{5}$$

summarize the population biology of the host–parasite interaction. Parasite mortality, transmission and reproduction in the model are controlled by the following instantaneous per head rate parameters. ρ, μ: death rates of free-living and parasitic stages, respectively; β: effective transmission rate per host; λ: effective reproductive rate per adult parasite. Density-dependent limitations on parasite population growth are expressed in the parasite reproductive function, λ, which is assumed in general to be a decreasing function of adult numbers per host. Assuming a linear decrease in the parasite's per head reproductive rate with parasite burden per host, the following expression for λ is derived in Appendix A:

$$\lambda = \lambda_0[1 - (1/A_0') - A/(HA_0)]. \tag{6}$$

Here, the maximum reproductive rate λ_0 declines with average burden A/H, depending on the parameter

$$A_0 = A_0' k/(k+1) \tag{7}$$

which allows for the observed overdispersed frequency distribution of adult parasites (described in §2), via the negative binomial dispersion parameter, k. Equation (6) (with $A < A_0$) produces essentially similar model properties to curvilinear functional forms for λ, and is much more algebraically tractable. The simplifying assumption that parasite density dependence is concentrated in adult reproduction is made because of the importance of this factor in the epidemiology of trichostrongylids, discussed in §2. Models based on density-dependent adult survival have broadly similar properties (Anderson & May 1978*a*). The effects of omitting the influence of past experience of infection and acquired immunity from equation (7) will be discussed below.

Assuming crudely that parasite-induced host mortality is offset by the replacement of hosts in intensive grazing systems (Grenfell *et al.* 1987*b*), the main link between the plant–herbivore and host–parasite interaction is the relation between infection and herbivore food consumption discussed in §2. This effect can be modelled most simply by assuming that the peak per head consumption rate of herbivores ($c_{\max}$ in equation (1)) is a decreasing function of the infection rate per host, βL (equation (5)). Over the range of infection rates shown in figure 3, food intake decreases approximately linearly with infection rate. This indicates the relation

$$c_{\max} = \gamma_1 - \gamma_2 \beta L, \tag{8}$$

i.e. c_{max} decreases linearly with βL from its maximum value, γ_1, in the absence of parasitism ($\beta L = 0$).

The 'dilution' effect of herbage growth in reducing infection rates in the field (documented in §2) is represented by

$$\beta = \beta_0 c/V$$
$$= \beta_0 c_{max}/(V^2 + K^2) \quad (9)$$

(from equation (3)). Equation (8) assumes that the infection parameter β is proportional to the ratio of c, the rate of food consumption per herbivore, and the total available plant biomass, V. β therefore increases with the proportion of herbage 'sampled' by the herbivores. The model is completely specified by combining equations (8) and (9) to obtain

$$c_{max} = \frac{\gamma_1}{1+\gamma_2 \sigma L} \quad (10)$$

and

$$\beta = \frac{\sigma \gamma_1}{1+\gamma_2 \sigma L} \quad (11)$$

with the definitions $\sigma = \beta_0 f$ and $f = V/(V^2 + K^2)$. Finally, collecting together the basic rate equations (1), (4) and (5):

$$\frac{dV}{dt} = gV(1 - V/V_{max}) - c_{max}(V, L)\, VfH, \quad (12)$$

$$\frac{dL}{dt} = \lambda_0 A[1 - (1/A_0') - A/(HA_0)] - (\rho + \beta(V, L)\, H)\, L, \quad (13)$$

$$\frac{dA}{dt} = \beta(V, L)\, HL - \mu A. \quad (14)$$

The implications of more complicated assumptions about the depression of food intake with infection (equation (8)) and the parasite 'sampling' function (equation (9)) will be discussed after an analysis of the model's general properties.

(b) *Patterns of infection and food consumption*

Equations (10) and (11) respectively define the peak herbivore feeding rate per head, c_{max}, and the infection parameter β, as functions of plant abundance (V) and the density of free-living parasites (L). The general form of these relations is shown in figure 4. For constant larval abundance, L, β increases rapidly with V to a peak and then declines at higher levels of plant abundance. This pattern in the infection coefficient with plant abundance is essentially determined by the 'sampling' function c/V of equation (9). The ratio c/V represents the herbivore feeding rate per head per unit plant abundance. For the sigmoid functional response of ruminant herbivores (equation (3)), $c/V = 0$ at $V = 0$ and increases with V to a maximum at $V = K$. β also follows this general pattern, increasing to a peak, $\beta_{max} = \gamma_1/[2K + \beta_0 \gamma_2 L]$ at $V = K$, which corresponds to the maximum encounter rate with parasites, c/V. At higher levels of plant abundance the infection coefficient declines, owing to the 'dilution' effect of herbage growth on free-living parasite density. The maximum consumption rate, c_{max}, follows the converse pattern with V, decreasing from its peak value ($c_{max} = \gamma_1$) at $V = 0$ (when $\beta = 0$ and no infection occurs), to a minimum at $V = K$; c_{max} then increases for larger V, as β declines.

Figure 4 also shows the dependence of c_{max} and β on free-living larval abundance, L, for

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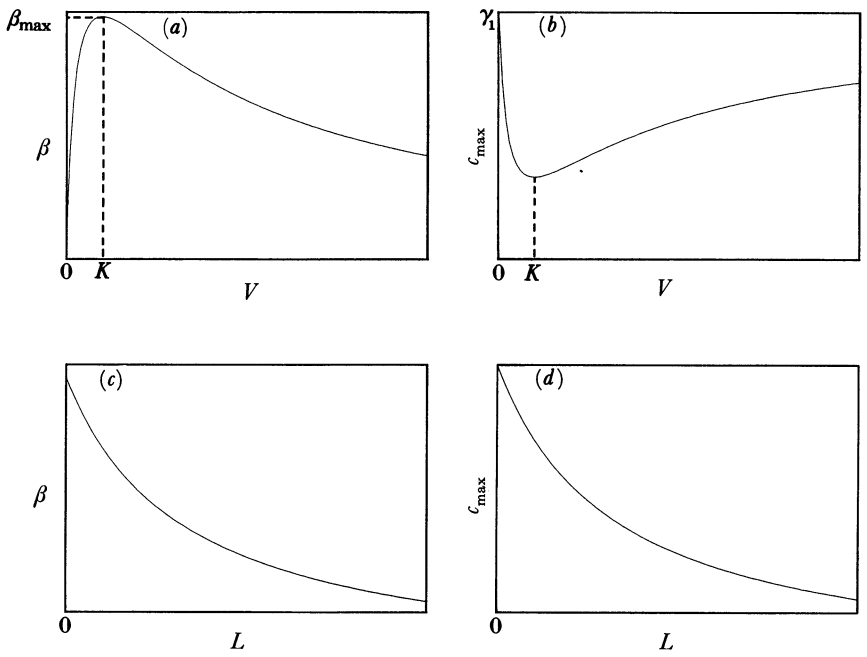

FIGURE 4. General patterns in the infection parameter (β) and peak consumption rate per herbivore (c_{max}), as functions of plant biomass (V) and the abundance of infective larvae (L) (from equations (10) and (11)).

constant V. As indicated by equations (10) and (11), both decrease hyperbolically with increasing L. The decline in β with L represents a *density-dependent decrease* in the infection probability with larval abundance. This is caused by the decrease in herbivore feeding rate per head with larval intake, which is illustrated, in terms of the effect of various constant levels of larval abundance on the herbivore's functional response, in figure 5.

(c) *Model equilibria*

The ecological implications of these patterns in the rates of infection and food consumption can be elucidated by considering the steady state (equilibrium) behaviour of the model. The equilibrium states of equations (12)–(14) (defined by $dV/dt = dL/dt = dA/dt = 0$, at which $V = V^*$, $L = L^*$, and $A = A^*$) are discussed in detail in Appendix A. The effects of parasitism on the functional response of the herbivore to changes in plant abundance can be summarized by considering the equilibrium parasite population size ($L^*(V)$, $A^*(V)$) at a given constant level of plant abundance, V. The corresponding net plant consumption rate by herbivores is

$$C(V, L^*) = c_{max}(V, L^*)\, VfH \tag{15}$$

$$= [\beta^*(V)/\beta_0]\, VH, \tag{16}$$

where $\beta V) = \beta_0 c_{max}\ (V, L^*) f$ is the infection parameter at the parasite equilibrium; $\beta^*(V)$ is defined as a function of V in Appendix A. Figure 6*a* uses equation (16) to display the effects of parasite equilibrium abundance on the functional response of the host. Three consumption curves are shown, corresponding to a zero parasite population (the normal functional response), a relatively low equilibrium parasite population (limited by a high adult death rate), and a high parasite abundance (with a low adult death rate). The results are dramatically

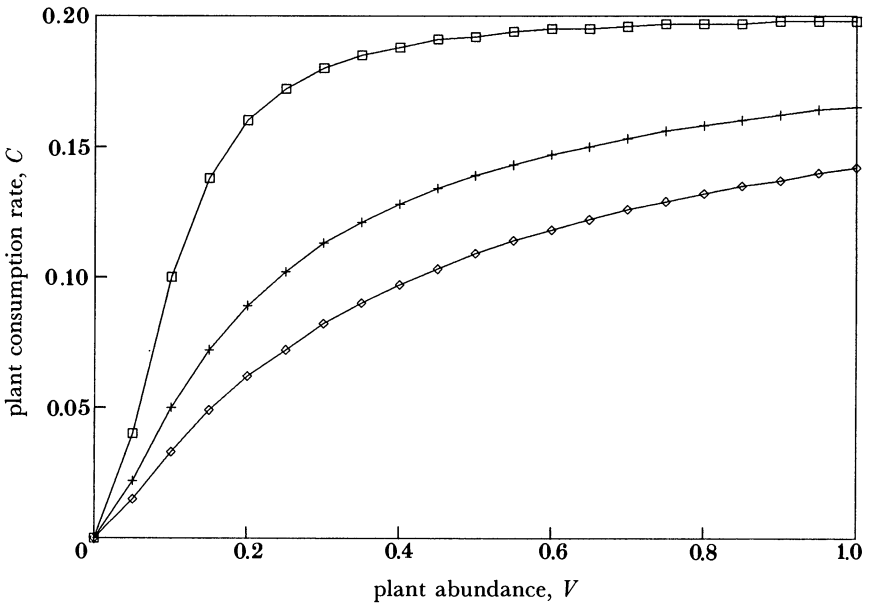

FIGURE 5. Plant consumption curve for herbivores in the absence of parasitism (as in figure 1, with $H = 2$), compared with consumption levels at two constant levels of larval abundance (L). The effect of parasitism on c_{max} is calculated from equation (10) with parameters $\gamma_1 = 1$, $\gamma_2 = 1$, $\beta_0 = 0.1$. □, $L = 0$; +, $L = 1$; ◇, $L = 2$.

different from those for constant parasite abundance shown in figure 5. Figure 6*a* indicates that increasingly high equilibrium levels of parasite abundance progressively *linearize* the host's functional response. This effect is explored in figure 6*b*, which displays the corresponding net herbivore consumption curves per unit plant biomass, C/V. At zero or low parasite abundance, the sigmoid functional response of the herbivores generates a peak in C/V at $V = K$. However, (as shown in figure 4), the infection-dependent decrease in peak host feeding rate (c_{max}) is at its maximum at this point. Increasing levels of parasite abundance therefore lower the initial peak in feeding rate, and tend to linearize the functional response at the parasite equilibrium. Note that the density-dependent decrease in $\beta^*(V)$ that causes this is also illustrated in figure 6*a* because, from equation (16), $C/V = \beta^*(V)\,H/\beta_0$.

The parasite equilibrium defined by $\beta^*(V)$ will be positive ($L^*(V) > 0$, $A^*(V) < 0$) if the corresponding basic reproductive rate of the infection

$$R_0(V) = \frac{\lambda_0(1-1/A_0')\,\beta^*(V)\,H}{\mu[\beta^*(V)\,H+\rho]} \qquad (17)$$

(the average number of mature parasites produced by a single adult parasite in the host population during its reproductive lifetime (Anderson & May 1985)) is greater than unity. In Appendix A it is demonstrated that increases in both $R_0(V)$ and γ_2 (the rate of reduction in c_{max} with infection rate) accentuate the linearization of the functional response at the parasite equilibrium.

Linear functional responses can, in general, have significant effects on both the dynamics of plant–herbivore interactions and the pattern of herbivore productivity (Holling 1965, 1966; May 1973, 1981; Noy-Meir 1975; Crawley 1983). These effects are examined for the plant–herbivore–parasite interaction in the following sections.

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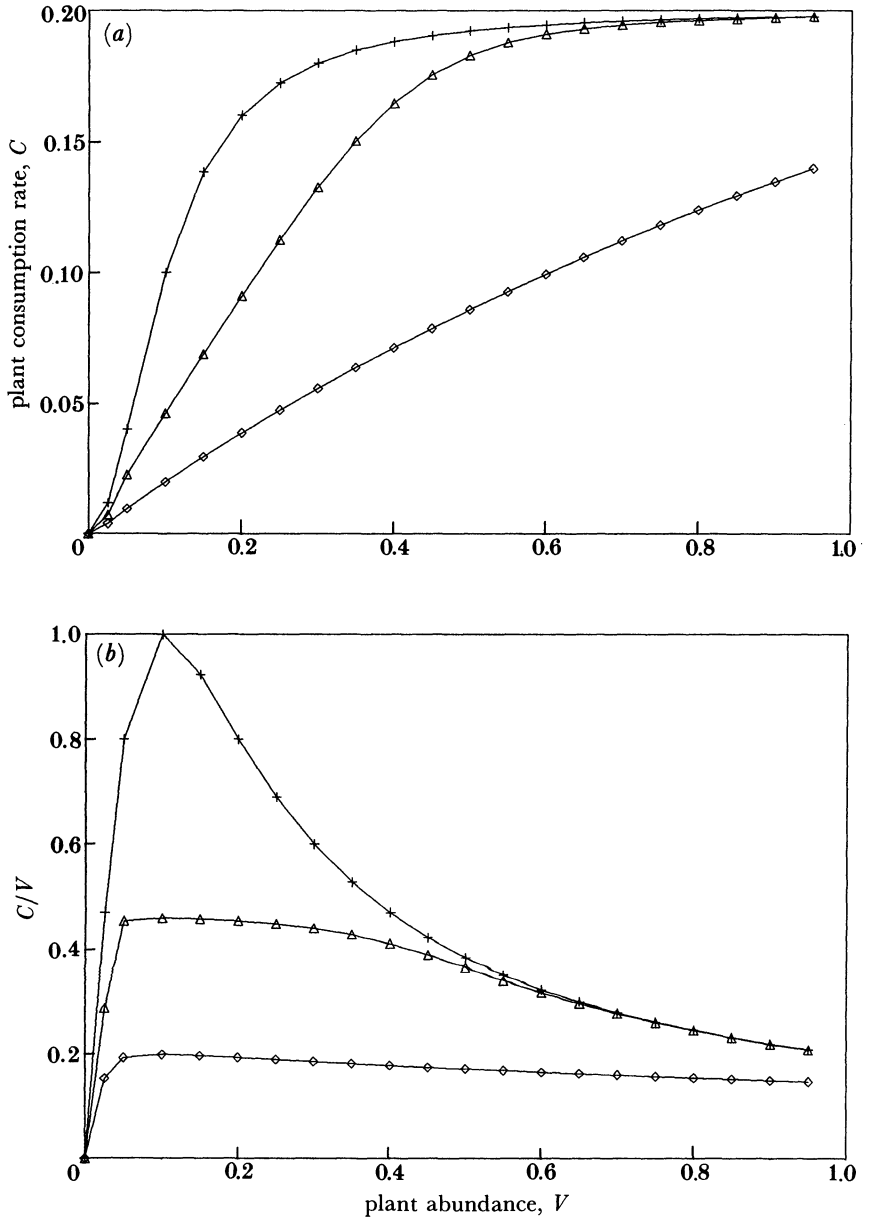

FIGURE 6. (*a*) The parasite-free herbivore consumption curve of figure 5 ($L = 0$), compared with equivalent consumption rates at two levels of parasite equilibrium (details are given in Appendix A). +, ($L = 0$). ◇, ($L(a)$), a parasite population with a relatively high basic reproductive rate, $R_0 \approx 20$ at the plant equilibrium, controlled by an adult death rate $\mu = 0.5$. □, ($L(b)$), relatively high death rate/low reproductive rate parasite population ($\mu = 1$; $R_0 \approx 2$). Other parameters are as in figure 5, with the additions $\lambda_0 = 20$, $A_0 = 2$, $\rho = 0.125$. (*b*) Corresponding net consumption rate per unit plant abundance, C/V.

(*d*) *Model dynamics*

(i) *Effects of nematode parasitism on the dynamics of overgrazing*

The most agriculturally important case to consider is the effect of nematode parasitism on the dynamics of overgrazing. In the absence of parasitism, this corresponds to the situation in figure 1*b* at the higher stocking density, $H = 3$; net plant growth rate is negative at plant abundances above an overgrazed equilibrium. The effects of a significant degree of nematode

parasitism on this system is illustrated in figure 7, which is a standard phase–space analysis for the model (Rosenzweig & MacArthur 1963). Figure 7*d* shows a series of dynamic trajectories for $V(t)$, $L(t)$ and $A(t)$, generated by numerical solution of equations (12)–(14) and plotted in (V, L, A) state space. Figure 7*a–c* shows the corresponding isosurfaces, which represent the boundaries between positive and negative population growth for V, L and A respectively.

Figure 7 shows that a significant degree of parasitism can create a single model equilibrium at a relatively high plant biomass, in an otherwise overgrazed system. This effect essentially arises because the reduction in herbivore feeding rates caused by infection generates a stable equilibrium at a plant abundance above $V_{max}/2$ (see figure 1). The dynamic processes that control the model's behaviour are apparent in the isosurfaces shown in figure 7.

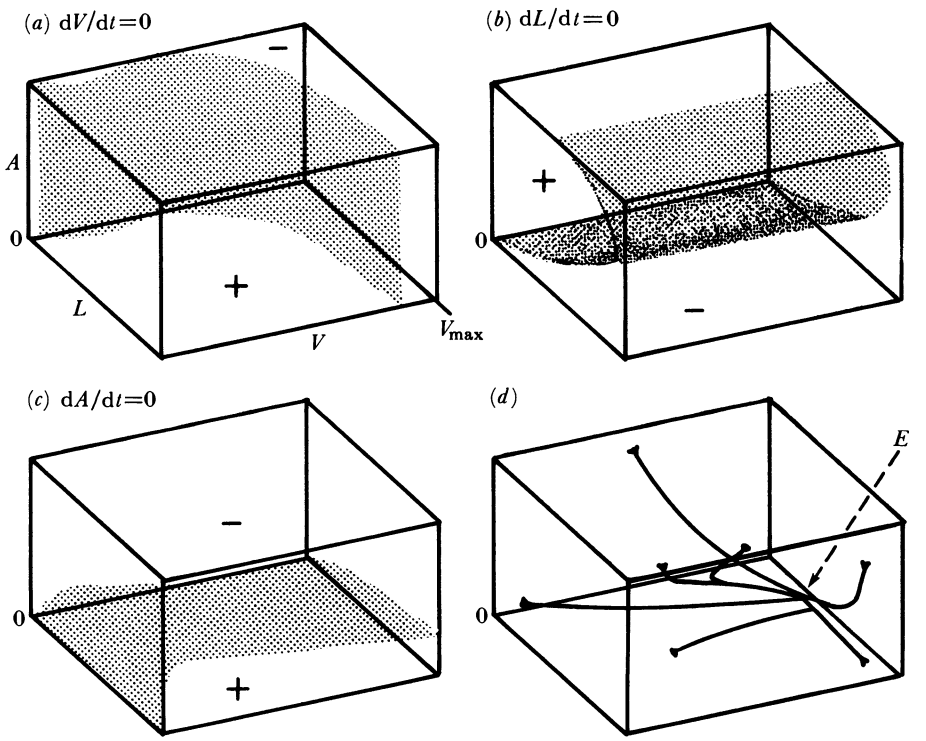

FIGURE 7. Phase–space analysis for the plant–herbivore–parasite model (equations (12)–(14)). (*a–c*) Isosurfaces, representing the boundaries, in (V, L, A) phase space, between positive and negative net growth rates for (*a*) plant abundance ($dV/dt = 0$), (*b*) larval parasite abundance ($dL/dt = 0$), and (*c*) adult parasite abundance ($dA/dt = 0$). The isosurfaces were calculated by a general numerical algorithm. (*d*) Corresponding pattern of dynamic trajectories, calculated by numerical solution of equations (12)–(14). The trajectories converge onto the single equilibrium point (*E*). (Parameter values as for figure 6, with $H = 3$, $\mu = 0.5$.)

The plant (V) *isosurface* ($dV/dt = 0$, figure 7*a*) intersects the V axis ($L = 0, A = 0$) at the stable overgrazed equilibrium in the absence of parasitism shown in figure 1. As larval parasite abundance increases, the region of positive net plant growth rate expands, reflecting a progressive decline in herbivore consumption (figure 5).

The larval parasite (L) *isosurface* ($dL/dt = 0$, figure 7*b*). As adult parasite abundance increases from zero, the region of positive population growth for the free-living parasites first expands and then shrinks. This pattern derives from the quadratic density-dependent limitation on adult parasite reproduction embodied in equation (15). An increase in the severity of density-dependent control of reproduction (or in the degree of overdispersion of adult worms) decreases

A_0 in equation (7). This sharpens the decline in the larval isosurface and lowers the larval and plant equilibria (as described in Appendix A). This pattern is almost independent of changes in plant abundance for V significantly greater than zero.

The adult parasite (A) *isosurface* ($dA/dt = 0$, figure 7*c*). The height of this surface (the A axis in figure 7*c*) is proportional to the net infection rate βHL (see Appendix A). It climbs to a plateau with V, and increases to a peak, then declines with L. The relationship of the isosurface with L clearly demonstrates the density-dependent relation between β and L shown in figure 4.

The general dynamic behaviour of the model depends on the degree of reduction in herbivore feeding rate due to parasitism (which is determined by γ_2 and the basic reproductive rate of the parasite population R_0). When the impact of parasites on host feeding rates is small (low γ_2 and/or R_0) the dynamics are essentially determined by the plant–herbivore interaction illustrated in figure 1. If, on the other hand, infection causes a large reduction in feeding rates, the lowering and effective linearization of the herbivore's functional response can have a strongly stabilizing effect, as illustrated in figure 7. The analytical details of these results, and their extension to allow for models with variable host populations, will be described elsewhere.

(ii) *The impact of parasite control*

Effective anthelmintic drugs and novel methods of drug delivery generate the possibility of strategically controlling gastrointestinal nematodes of ruminants for relatively prolonged periods of time (Smith *et al.* 1987*b*). The effects of this form of parasite control in the model defined by equations (14)–(16) can be represented simply by a significant increase in the death rate of adult parasites, μ. This modification allows an examination of the dynamic effects of strategic parasite control on the potentially overgrazed plant–herbivore–parasite system described above.

Figure 8 uses a phase–space analysis to consider the extreme case of attempted parasite eradication (only the adult isosurface is shown, as changes in μ do not effect the plant and larval rate equations, (12) and (13)). Increasing the death rate of adult parasites causes their region of positive population growth to shrink almost to zero (figure 8*a*). The resulting dynamic trajectories (figure 8*b*) indicate a rapid decrease in adult parasite abundance, followed by a more gradual collapse of the system, to a low overgrazed plant equilibrium and parasite extinction.

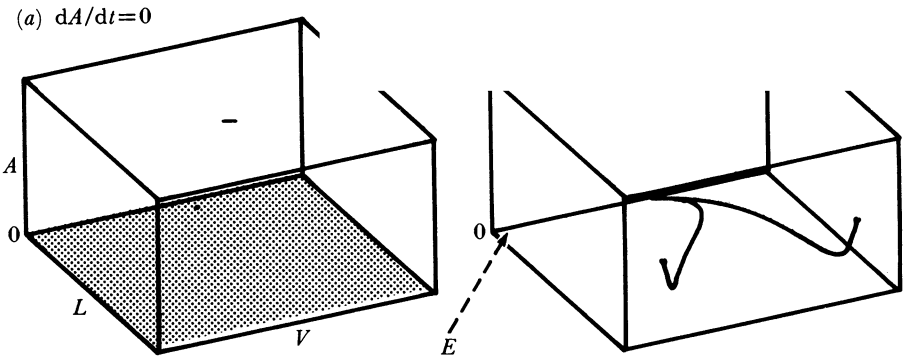

FIGURE 8. Phase–space analysis as for figure 7, with parasite eradication by strategic chemotherapy represented by $\mu = 2$ (the associated average parasite reproductive rate $R_0 < 1$). (*a*) Adult parasite isosurface ($dA/dt = 0$); (*b*), associated dynamic trajectories. The equilibrium here (E) is at the parasite-free overgrazed equilibrium of figure 1*b*, with $H = 3$.

This result reflects the intuitively sensible idea that the application of effective strategic parasite control to very marginal grazing systems could lead to overgrazing because of the improvement in feeding (and growth) rates of unparasitized animals. The likelihood of this effect in real grazing systems will be discussed below.

(*e*) *The effects of parasitism on productivity*

One of the most important factors in determining the productivity of a grazing system is the relation between the net production of grazing animals (typically per unit area) and their stocking density (Johnson & Parsons 1985; Morley 1966; Mott 1960; Noy-Meir 1975; Owen & Ridgeman 1968). This productivity curve generally shows a single maximum (at the optimum stocking rate), which reflects a tradeoff between increasing herbivore density and decreasing production per animal (Mott 1960; Owen & Ridgeman 1968). In *continuously stable* systems (Noy-Meir 1975), the herbivore functional response is relatively linear, and the equilibrium production curve decreases smoothly at stocking rates above the optimum. By contrast, in *discontinuously stable* systems, productivity decreases rapidly at high stocking rates as plant abundance 'jumps' to its overgrazed steady state (figure 1 exemplifies a discontinuous system).

The calculation of herbivore productivity is complicated by the reduction in gross production efficiency due to parasitism, reviewed in §2*b*. Assuming that this decrease is approximately linear (figure 3), the proportional decline in production efficiency (ν) can be crudely represented by

$$\nu = 1 - m\beta L, \tag{17}$$

where m is a constant. Figure 9 illustrates the effects of parasitism (as predicted by equations (12)–(14) and (17)) on the relative productivity of the discontinuously stable grazing system, defined by the functional response of Figure 1. These results indicate two possible effects of

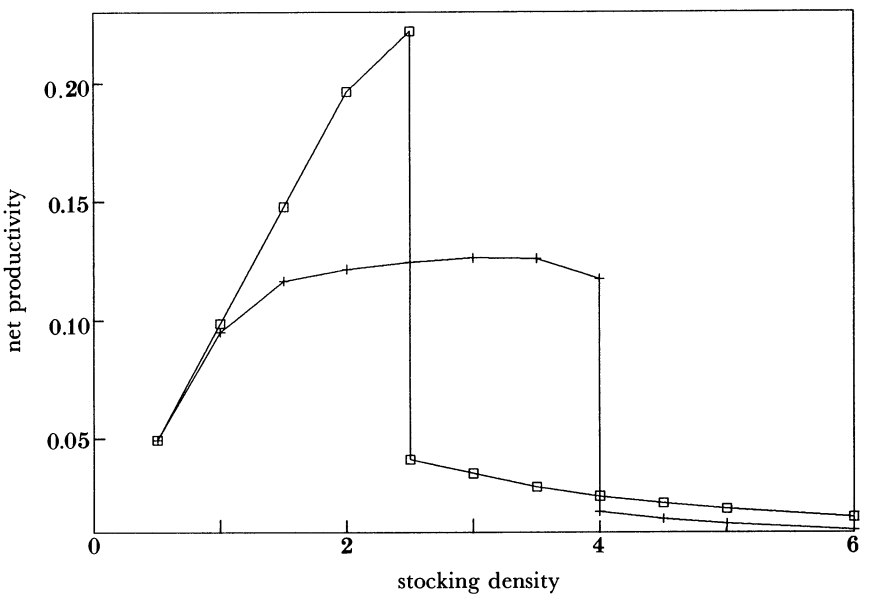

FIGURE 9. Net equilibrium productivity at a range of stocking densities, for unparasitized (□) and parasitized (+) herbivores. For comparative purposes, the productivity curves are crudely calculated as the net herbivore consumption rate at the equilibrium of equations (12)–(14). The proportional reduction in production efficiency caused by parasitism is calculated from equation (17), with $m = 1$. Other parameters are as in figure 7, with $\gamma_2 = 0.3$.

nematode infections on herbivore productivity. First, the reduction in herbivore feeding rates per head increases the stocking density at which the system collapses to a low level of productivity. This process therefore extends the region of continuous stability of the system. However, in practice, the large worm burdens and severe disease associated with uncontrolled PGE outbreaks at high stocking densities (Grenfell *et al.* 1987*b*) greatly complicate this picture. Secondly, the decline in production efficiency with infection reduces the yield curve at high host densities. The effect occurs because equilibrium larval abundance (and therefore host infection rate per head) is an increasing function of stocking density below the level of the collapse (Grenfell *et al.* 1987*b*).

4. Comparisons with field data

The ideal field experiment to examine the issues raised above would involve the construction of detailed productivity/stocking rate curves at a number of known levels of parasite challenge, with parallel estimates of herbivore feeding rate and grass availability. Although many of these variables are commonly measured in field investigations of PGE (Armour & Ogbourne 1982), parallel estimates of feeding rate and the effects of stocking density and parasite abundance appear to be comparatively rare. Figure 10 summarizes the results of a Danish study (Hansen *et al.* 1981) that sets out to examine these relations. This field experiment tested the effects of three levels of stocking density on individual feeding rate and net herd productivity through the grazing season. The experiment was done on two groups of calves: the first group was kept on pasture heavily infected with trichostrongylids throughout the grazing season, whereas the second group was moved to relatively uncontaminated pasture at mid-season (therefore experiencing a lower average level of infection).

Figure 10*a* shows the effects of stocking density and level of pasture contamination on average feeding rate per calf. It indicates a slight decrease in feeding rate with stocking density in the low infection group, and a more pronounced decline in the heavily infected animals. At all stocking densities, feeding rate was significantly more depressed at the higher level of infection. These results are in broad agreement with the predictions of the model, in that an increased density of infective larvae causes greater depression in feeding rate. This effect is accentuated at high stocking densities, because larval abundance is higher and calves are forced to feed closer to contaminated areas (Grenfell *et al.* 1987*b*; Hansen *et al.* 1981). The corresponding pattern of net productivity (figure 10*b*) indicates a peak at the intermediate stocking density for the low infection animals, which is reduced (mainly by lowered food intake) at the higher infection level. This is again in broad agreement with the predictions of the model (figure 9), although the absence of a productivity curve for completely uninfected animals in these experiments means that they cannot be used to examine the effects of parasitism on the stability of production rates.

There appear, as yet, to be no published reports to support the hypothesis, suggested in §3, that strategic anthelmintic application could act to destabilize overstocked grazing systems. This effect may be unimportant in the intensive grazing systems of developed countries, where highly productive pasture and supplementary feeding are commonly available. However, it could be more significant in the more fragile (and much less studied) systems often found in less developed countries (Jainudeen & Omar 1980). The effective control of nematode populations

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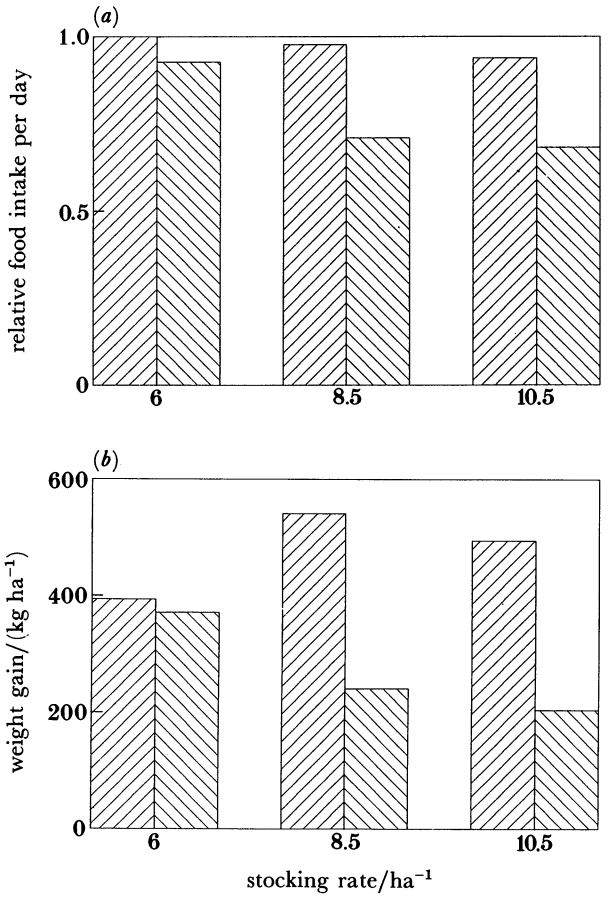

FIGURE 10. (*a*) Differences in average feeding rates of calves kept at three levels of stocking density, and two levels of pasture larval abundance (the lower level was achieved by moving animals to clean pasture at mid-season); feeding rate is expressed relative to its maximum (moved calves kept at 6 animals ha^{-1}). (*b*) Associated levels of net productivity (weight gain ha^{-1} during the grazing season). From Hansen *et al.* (1981). ▨, Moved; ▧, not moved.

by strategic anthelmintic application provides a potentially useful experimental tool for examining these issues. For example, Bell *et al.* (1988) show that protecting calves against trichostrongylid infections (by a sustained-release bolus) leads to the same reduction in feeding rate as untreated animals if pasture conditions are very poor. However, treatment does improve the *rate of increase* in weight gain and food consumption as the grazing system recovers. Further field experiments to examine these complexities are clearly required, particularly under poor grazing conditions.

5. Discussion

This paper has considered the potential impact of gastrointestinal nematode parasites on the stability and productivity of intensive ruminant grazing systems. The main result to emerge from the simple model used to represent this interaction is that significant depressions in host food intake as a result of infection could have marked effects on the population biology of the plant–herbivore interactions. These effects can be summarized via the impact of equilibrium

parasite abundance on the functional response of the herbivore to the level of plant biomass. If the depressive effect of parasitism on host consumption is sufficiently large, the density-dependent relation between feeding and infection rates effectively linearizes the functional response at the parasite equilibrium. The stabilizing influence of parasitism, and its impact on host productivity (both of which are discussed in §3 and §4) essentially follow from this. Broadly speaking, these conclusions apply equally to the impact of any helminth parasite of ruminants that is transmitted by ingestion and that depresses food intake (Symons 1985).

The model defined by equations (12)–(14) captures the essential features of the plant–herbivore–parasite relation in intensive grazing systems. However, as noted above, it omits much biological detail, and can therefore only be used to generate qualitative hypothese about the system's behaviour as a function of its basic parameters. To assess these effects quantitatively a more detailed formulation is required. The theoretical and experimental developments required to refine the model's descriptive powers can be summarized under two main headings.

(*a*) *Parasite population biology and host productivity*

The important epidemiological descriptor of PGE incidence in intensive grazing systems is the *temporal pattern* of the outbreak (Grenfell *et al.* 1987*b*). As described in §2*b*, this sequence of events is determined by a complex interaction between climatic influences and infection-dependent processes within the host. Any attempt at simulating the effects of grazing dynamics in more detail should therefore take account of these factors. The most important refinement (both in terms of model structure and experimental clarification) is the inclusion of immunological effects, which interact significantly with the reductions in host productivity arising from infection (Coop *et al.* 1982). The relation between host feeding rate and infection (represented by equation (8)) is particularly important in this context. For example, if this relation shows an initial plateau (at $c_{\max} = \gamma_1$) reflecting a lower threshold of infection for significant reductions in feeding rate (Coop *et al.* 1977), the effects of parasitism on grazing dynamics disappear at low parasite population levels.

(*b*) *Grazing behaviour and the infection process*

The relation between pasture larval abundance and the ingestion rate of larvae is a complex function of larval distribution, grazing behaviour and stocking rate (Raynaud & Gruner 1982; Hansen *et al.* 1981). A particularly important process, which requires experimental clarification, is the possible 'diluting' effect of plant growth on the infection rate. Equation (9) represents this process by assuming that the infection parameter increase with the proportion of available herbage consumed. The other extreme assumption, that β increases with the absolute quantity of herbage consumed, eliminates the dilution effect and magnifies the impact of parasitism on host food consumption. In reality, ingestion rate will be a random variable, reflecting the observed overdispersion of adult abundance. In terms of equations (12)–(14), the effects of such heterogeneities will tend to be reduced by the density-dependent regulation of feeding rate by infection. The potential role of this process in contributing to the comparatively low degree of overdispersion observed for adult trichostrongylids in intensive grazing systems will be discussed elsewhere.

The aim of constructing more detailed models for the plant–ruminant–herbivore interaction is to produce quantitative hypotheses about the behaviour of the system (Grenfell *et al.* 1987*b*). These results can then be compared with the outcome of field experiments, which assess the

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effects of parasitism and herbage dynamics on herbivore productivity (Hansen *et al.* 1981) and the impact of anthelmintics (Bell *et al.* 1988). The results presented here suggest that such experiments are a particular priority in potentially overgrazed systems.

Finally, this work has considered the relatively simple ecological interactions involved in intensive grazing systems with constant herbivore populations. In extensive rangeland systems where herbivore populations vary more or less naturally, the situation is potentially much more complex. In particular the nutritional impact of parasites described here is, in general, likely to interact with a number of other features of the host–parasite relationship. These include both dynamically stabilizing effects, such as parasite-induced host mortality, and destabilizing processes, such as limitations on host reproduction due to infection (Anderson & May 1978*a*, *b*). The impact of macroparasitic infections on extensive grazing systems will be considered in a subsequent publication.

I am particularly grateful to Dr Gary Smith for his patience and expertise in discussing these ideas. This work also benefited from discussions with Dr Marilyn Scott, Dr Roger Pritchard, Dr Mick Crawley and Mr Mervyn Jones of Pfizer Ltd. Bell *et al.* (1988) kindly provided an unpublished manuscript, and Mr W. Mosley gave expert help with the figures.

Appendix A

(*a*) *The parasite reproductive function*

If the instantaneous per head parasite reproductive rate decreases linearly with adult parasite burden, then the net reproductive rate of parasites in a host with i adult parasites can be represented by

$$\xi = \lambda_0(1-i/A_0'), \tag{A 1}$$

where λ_0 is the maximum per head reproductive rate and A_0' controls the degree of density dependence. The corresponding net reproductive rate of the total parasite population in H hosts is

$$\Lambda = H\sum_{i=0}^{\infty}\xi p(i), \tag{A 2}$$

where $p(i)$ is the probability that a host contains i parasites (Anderson & May 1978*a*). Combining (A 1) and (A 2) gives

$$\Lambda = \lambda_0 H[E(i)-E(i^2)/A_0'], \tag{A 3}$$

in which $E(i)$ and $E(i^2)$ are the expectations of i and i^2 for the frequency distribution of parasites that defines $p(i)$. Assuming an overdispersed negative distribution for $p(i)$, with parameter k, (A 3) leads to

$$\Lambda = \lambda_0 A[1-(\{A/H\}(k+1)/k+1)/A_0] \tag{A 4}$$

(Anderson & May 1978*a*), where A/H and A are the average number of parasites per host and the total adult parasite population size respectively. The corresponding average parasite reproductive rate per host (λ in equation (6)) is therefore

$$\lambda = \lambda_0[1-(1/A_0')-A/(HA_0)] \tag{A 5}$$

with the definition $A_0 = A_0'k/(k+1)$.

(b) Equilibrium results

(i) *The parasite equilibrium*

From equations (13) and (14), the equilibrium state of the parasite population ($dL/dt = dA/dt = 0$, $L = L^*(V)$ $A = A^*(V)$) at a given level of plant abundance, V is determined by

$$\lambda_0 A^*(V)[1-(1/A_0')-A^*(V)/(HA_0)]-(\beta^*(V)+\rho)\,L^*(V) = 0, \tag{A 6}$$

$$\beta^*(V)\,HL^*(V)-\mu A^*(V) = 0 \tag{A 7}$$

in which $\beta^*(V)$ is the equilibrium infection parameter, defined by equation (11). Equations (A 6) and (A 7) lead immediately to the expressions

$$L^*(V) = \frac{\bar{\lambda}_0\beta^*(V)\,H/\mu-(\beta^*(V)\,H+\rho)}{\pi H\beta^*(V)^2}, \tag{A 8}$$

$$A^*(V) = \beta^*(V)\,HL^*/\mu \tag{A 9}$$

for $L^*(V)$ and $A^*(V)$, with the definition $\pi = \lambda_0/(\mu^2 A_0)$. At the point of parasite extinction ($L^*(V) = 0$), the basic reproductive rate of infection, $R_0(V) = 1$ so that, from (A 8),

$$R_0(V) = \frac{\bar{\lambda}_0\beta^*(V)\,H}{\mu(\beta^*(V)\,H+\rho)} \tag{A 10}$$

(corresponding to equation (16) with the substitution $\bar{\lambda}_0 = \lambda_0(1-1/A_0')$). The definition of $L^*(V)$ can be completed by re-expressing equation (11) as

$$L^*(V) = (\gamma_1\sigma\epsilon-1)/(\gamma_2\sigma), \tag{A 11}$$

where $\epsilon = 1/\beta^*(V)$. Combining equations (A 8) and (A 11) leads, after some algebra, to a quadratic equation in ϵ,

$$\epsilon^2-\epsilon[\Omega-\pi\gamma_1/(\gamma_2\rho)]\,H-\pi H/(\gamma_2\sigma\rho). \tag{A 12}$$

Ω is defined here as $\Omega = (\lambda_0-\mu)/(\mu\rho)$. Equation (A 12) has one non-negative root,

$$\epsilon = H[\Omega-\pi\gamma_1/(\gamma_2\rho)]/2-\{(H[\Omega-\pi\gamma_1/(\gamma_2\rho)]/2)^2+\pi H/(\gamma_2\sigma\rho)\}^{1/2}. \tag{A 13}$$

Equations (A 9), (A 11) and (A 13) can then be used to define $L^*(V)$ and $A^*(V)$.

(ii) *The herbivore functional response at the parasite equilibrium*

From equations (16) and (A 13), the net herbivore feeding rate at the parasite equilibrium is

$$C(V, L^*) = [\beta^*(V)/\beta_0]VH = VH/\beta_0\epsilon, \tag{A 14}$$

which defines the function plotted in figure 6. The progressive linearization of the consumption curve with increasing γ_2, described in the main text, corresponds to ϵ, in equation (A 14), becoming increasingly independent of $\sigma = V/(K^2+V^2)$, and therefore of V. As γ_2 increases in equation (A 13), the term in σ becomes progressively less important, and disappears at the limit

$$\gamma_2 \xrightarrow{\lim} \infty \epsilon = H\Omega \tag{A 15}$$

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for $V > 0$. This linearization also progresses with increases in the basic reproductive rate of the parasite, $R_0(V)$ (equation (A 10)). For example, when λ_0 is large, ϵ (in equation (A 13)) can be expressed approximately as

$$\epsilon = H[\Omega - \pi\gamma_1/(\gamma_2\rho)]. \quad \text{(A 16)}$$

Equations (A 15) and (A 16) indicate that the consumption curve also becomes independent of H at these limits. Note that these results are not sensitive to the detailed shape of the functional response, which is defined by σ.

(iii) *The plant equilibrium*

The equilibrium plant abundance, V^*, can be calculated from the combination of equations (9) and (12),

$$gV^*(1 - V^*/V_{\max}) - C(V^*, L^*) = 0.$$

Given the above definition of L^*, this is a nonlinear (polynomial) function of V^*, which can be solved numerically to obtain the plant equilibrium.

The solution of equation (A 10) at the plant equilibrium was used to calculate the values of R_0 ($R_0 \approx 2$ and $R_0 \approx 20$), presented in figure 6. Calculations based on population parameters for *O. ostertagi* infections in calves (Grenfell *et al.* 1987*b*) suggest that R_0 may be at the top of this range at its seasonal maximum. These calculations will be presented in detail elsewhere.

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