J. Hyg., Camb. (1980), **84**, 389
Printed in Great Britain

Simulating strategies for control of *Echinococcus granulosus*, *Taenia hydatigena* and *T. ovis*

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(*Received* 27 *July* 1979)

SUMMARY

A deterministic model to compare various control strategies for parasites having two hosts is presented. When applied to *Echinococcus granulosus*, *Taenia hydatigena* and *T. ovis*, the model shows that maximum progress would be achieved when both the definitive and the intermediate host are treated simultaneously. This type of model may prove of value in studies on the control of other two-host parasites.

INTRODUCTION

The first hundred years of attempts to control *Echinococcus granulosus* in New Zealand have been reviewed by Gemmell (1973). In 1873, *E. granulosus* was so common in humans that it was made a notifiable disease. Sporadic attempts to publicize the life cycle of the parasite and its danger to human health had no effect on prevalence. From 1937 onwards the New Zealand Government provided arecoline hydrobromide which dog owners could administer to their dogs every 3 months, on a voluntary basis. In 1940 it became illegal to feed dogs raw offal. Voluntary control committees were set up in 1947 and by 1959 436 voluntary schemes were under way. Co-ordination of these schemes followed, and with the training of 110 control officers, regular compulsory arecoline testing of about 200000 dogs began. Since 1959, over 800 new cases of human infection with *E. granulosus* have been reported, but the incidence declined from 30 per million to 7 per million from 1970 onwards, when effective anthelmintics for dogs became available. Regular treatment of dogs was then undertaken specifically to lower the incidence of *Taenia ovis* cysts in export lamb carcases. Although the control programme has now been in force for 20 years, any relaxation may well lead to rapid reversion to the precontrol situation. In addition to the canine anthelmintics already in use, potential control measures in the form of canine vaccines, ovine vaccines and ovine anthelmintics may become available as aids in the control of these parasites. In order to evaluate their probable effect, and the probable effect of varying access to potentially infective material, a model of the situation in respect to *E. granulosus*, *T. ovis* and *T. hydatigena* has been developed. The model

0022–1724/80/0075–1979 $01.00

described here attempts to compare the relative times that might be required to achieve a level of less than 0·1 % of infected animals, using various combinations of new or existing control procedures.

'Fairly simple deterministic models can be quite effective in supplying a quantitative framework for discussion of any infectious disease for which a certain amount of biological and epidemiological knowledge is available. These models have already been used to sharpen discussion of planning preventive measures and of optimising resource allocation' (Bailey, 1975).

Basic patterns in the epidemiology of *E. granulosus* infection in New Zealand have been recently analysed (Burridge & Schwabe, 1977*a*, *b*; Burridge, Schwabe & Fraser, 1977; Burridge, Schwabe & Pullum, 1977). The method of 'path analysis' involved sequential multiple regression analysis of a linear 'causal' model constructed from existing biological and epidemiological knowledge. In order to compare the efficacy of various postulated control measures for *E. granulosus*, *T. hydatigena* and *T. ovis*, a different approach is required.

The latent period between infection and appearance of the first segment in faeces is 6–8 weeks for these tapeworms in dogs. The latent period from infection to an infective cysticercus is also 6–8 weeks for *T. ovis* and *T. hydatigena* in the sheep. This has led us to select a discrete time model based on eight time periods per annum. In what follows only discrete time models are discussed.

A one-host multistate model of Firescu and Tautu (1967) (cited by Bailey, 1975) used fixed transition probabilities of an individual passing from one state to another during one time interval. This simplification is not realistic with regard to the probability of being infected, which, with most infection processes, will depend on the varying proportions of susceptible and infective individuals in both host populations.

One-host two-state models with more realistic infection probabilities, reviewed by Bailey (1975), deal mainly with a single point in time of infectiousness before isolation of the patent individual. Models where individuals have extended patent periods have so far only been amenable to finding the distribution of the 'final epidemic size', i.e. the maximum proportion of the population that will become patent during the course of an epidemic. The one-host three-state model of Cooke (1975) with time varying sub-classes, introduces more realism as it has a finite progression of infective classes.

Two-host multi-state models may not be amenable to general mathematical analysis, because of the variety of specifications of these models. The model for malaria, proposed by Dietz, Molineaux & Thomas (1974) seems nearest in specification to the deterministic situation of *E. granulosus*, *T. hydatigena* and *T. ovis* where the definitive host is the dog, and the intermediate host is the sheep. Extension to stochastic two-host multi-state models seems a more distant prospect. As a first approach the Markov chain method similar to Firescu & Tautu (1967) is applied to simulate various control measures of the endemic tapeworm situation in New Zealand.

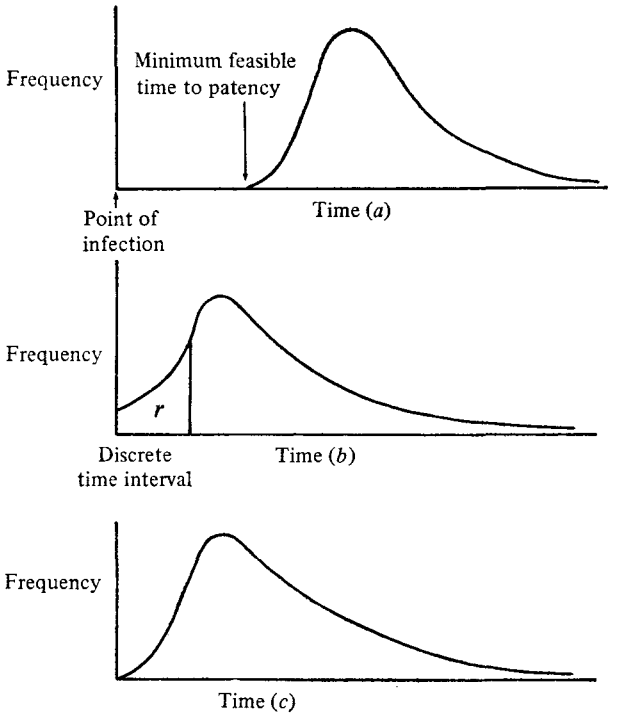

Fig. 1. Frequency distribution of (*a*) the latent period for an individual animal from point of infection, the latent period for a (*b*) group of animals with gamma-type distribution of elapsed occupancy times and (*c*) the patent period for an individual animal.

MODEL

States

The following five possible states are considered for both sheep and dogs:

U = Uninfected susceptible
L = Latent infection (prepatent)
P = Patent infection (fully infective carrier)
A = Acquired immunity (immunity stimulated by challenge infection)
V = Vaccine immunity.

The definition of these states involves several simplifying assumptions.

Uninfected susceptible [U]

In the uninfected susceptible state there is no distinction between animals which may never have been infected or those which have reverted from infected or immune states, or those which are population replacements by birth.

Latent infection [L]

For an individual animal the duration of the latent state can be thought of as having two components. Initially there is a certain interval, being the minimum time in which it is biologically possible to produce eggs or worm heads, following

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which there is added a right-hand-skewed frequency distribution of times to account for between-animal and between-cyst/worm variability – see Fig. 1(*a*).

For a group of animals representing a range of elapsed occupancy times in the latent state, the frequency distribution of residual durations will also be right-hand skewed but with a non-zero frequency near the origin. Within the next discrete time interval a fraction r of those currently in the latent state will revert as shown by the area of the frequency distribution in Fig. 1(*b*). Biological data concerning latent periods are shown in Appendix I.

Patent infection [P]

The distribution of the durations of a patent infection taking its natural course is similar to the latent state except that the minimum feasible time to reversion following attainment of patency is less definite. Resistance mechanisms stimulated during latency may cause rejection of a patent worm or cyst almost immediately after attainment of patency although most infections will last for some time (Fig. 1*c*).

The patent periods for cysts and worms are shown in Appendix I.

Acquired immunity [A]

Acquired immunity may be obtained by anthelmintic dosing of animals with either latent or patent infections, as well as by natural reversion from the patent state. Repeated infection and dosing of dogs may sometimes induce an immunity against worms of *E. granulosus* (Gemmell pers. comm.), *T. ovis* (Heath unpublished) but not *T. hydatigena* (Parmeter pers. comm.). The duration of acquired immunity may depend on whether it arose from dosing or reversion, but in this first approach it is assumed to have the same distribution of residual occupancy times.

Further, those animals lapsing from a vaccine-immune state or those that would be expected to lapse from a previously attained acquired-immune state may transfer to or re-enter the A-state by virtue of their immunity being boosted by challenge while in the previous state. For all these changes of state, attained acquired immunity is taken as the probability of having been challenged at least once while in the previous state.

For the A-state this effect would be better described by a challenge causing a shift up of a few classes on a sub-classification (Cooke, 1975) of time intervals spent in the acquired immunity state, but that is beyond the scope of a first approach.

For the L, P and A states there is a distribution of residual occupancy time which implies a certain probability of converting or reverting during the next discrete time interval. A model could be constructed from the varying numbers of animals entering the A-state during previous periods to give a *current* probability of reversion from that state during the next interval. However, in this first approach a constant probability of reversion or conversion is assumed whatever the distribution of occupancy times.

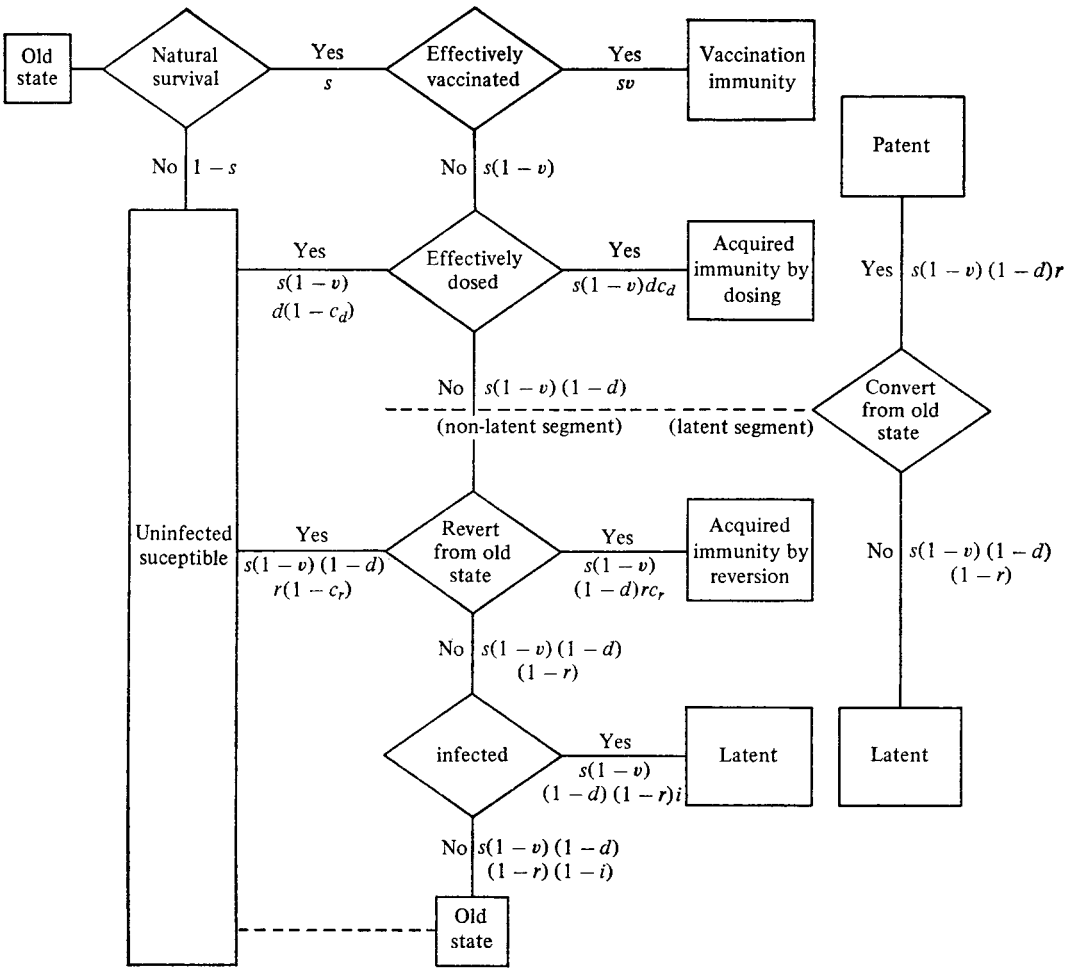

Fig. 2(*a*) Flow diagram showing the possible transitions from all states during one time interval.

Vaccine immunity [*V*]

Given that an animal is vaccinated, the effective immune response is considered to take place within one discrete time interval, followed by a decay in immunity in succeeding intervals. The frequency distribution of the duration of immunity is a property of a particular vaccine. A proportion of animals will retain sufficient immunity to withstand a challenge infection up to the time of the next vaccination. Based on a constant rate of decay of vaccine immunity, there is a fixed probability of lapsing during one discrete time interval.

Experimental sheep vaccines will not remove an existing infection. In contrast, certain postulated dog vaccines should also remove worms. Thus while stimulating an immunity to reinfection, the vaccination of an infected sheep does not cause any change of state until the animal is effectively dosed. If vaccines are developed to eliminate cysts as well, dosing would not be required. Rather than introduce a

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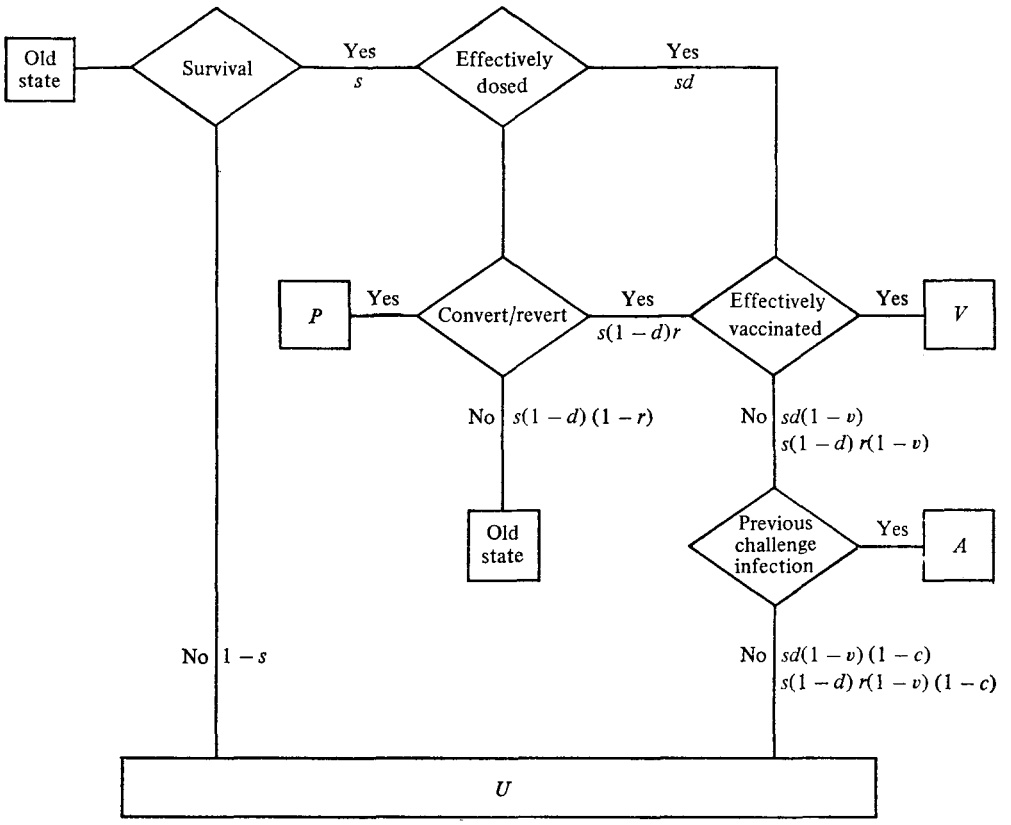

Fig. 2(*b*) Modification of flow diagram for possible transitions between states to take account of the fact that current sheep vaccines are not able to remove existing patent and latent infections.

separate state for vaccinated but infected sheep, such animals remain classified as patent or latent unless simultaneously dosed.

Transitions

Transitions between states may occur either actively or passively. Active transitions due to control strategy are by vaccination or anthelmintic dosing. Passive transitions are by reversion from P or A states, by conversion from the L state, or by infection of individuals in the U state.

Transitions may be state dependent and in particular the transition from U to L for one host will be related to the proportion in the P state of the alternate host.

Flow diagram

Given that a constant fraction s survives over one discrete time interval the possible transitions from all states may be expressed in the diagram shown in Fig. 2(*a*). Current experimental sheep vaccines impose a modified flow diagram for sheep L and P states shown in Fig. 2(*b*).

Effective dosing (d) represents the combined probability of being treated and the

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treatment being effective. Dosing and possible reversion each have three feasible outcomes. Change of state can be either to acquired immunity from prior challenge infection (c_d, c_r) or to uninfected susceptible, while no change of state is the third feasible outcome.

The possible conversion from latent to patent requires a slightly different logical pattern although the same probability symbol r has been retained and the subscript l will identify it as a state dependent 'reversion' from state L.

Several of the transition probabilities are necessarily zero, and by using the subscripts u, l, p, a, v for transition from states U, L, P, A, V the probabilities (e.g. v is the probability of being vaccinated) may be summarised as follows:

v = vaccinate; $1-v$ = not vaccinate

d = dose effectively; $1-d$ = not dose effectively ($d_u = d_a = d_v = 0$),

r = revert (convert); $1-r$ = not revert (not convert) ($r_u = 0$),

i = infect; $1-i$ = not infect ($i_l = i_p = i_a = i_v = 0$),

c = acquire immunity; $1-c$ = not acquire immunity.

Transition matrix

U	L	P	A	V
$(1-s)+s(1-v)(1-i_t)$	$(1-s)+s(1-v)\times d_l(1-c_{dl})$	$(1-s)+s(1-v)\times d_p(1-c_{dp})+s(1-v)(1-d_p)\,r_p(1-c_{rp})$	$(1-s)+s(1-v)\times r_a(1-c_{ra})$	$(1-s)+s(1-v)\times r_v(1-c_{rv})$
$s(1-v)i_t$	$s(1-v')(1-d_l)\times(1-r_l)$	—	—	—
—	$s(1-v')(1-d_l)r_l$	$s(1-v')(1-d_p)\times(1-r_p)$	—	—
—	$s(1-v)d_lc_{dl}$	$s(1-v)d_pc_{dp}+s(1-v)(1-d_p)\times r_pc_{rp}$	$s(1-v)(1-r_a)+s(1-v)r_ac_{ra}$	$s(1-v)r_vc_{rv}$
sv	sv^*	$sv^\dagger$	sv	$sv+s(1-v)\times(1-r_v)$

$$\times\begin{bmatrix}U\\L\\P\\A\\V\end{bmatrix}_t=\begin{bmatrix}U\\L\\P\\A\\V\end{bmatrix}_{t+1}$$

or $T_tS_t = S_{t+1}$

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The proportion of animals in a particular state at the end of a period is obtained by adding together the proportions in each state at the beginning of the period, multiplied by the probability of transition from that state to the particular altered state. For instance the proportion in the V state is obtained by multiplying the proportions in all states by the probability of being effectively vaccinated (assuming vaccination strategy was applied) and adding.

This can be conveniently summarized in mathematical shorthand by the matrix form where T is the transition matrix and S_t is the vector of proportions in each state at time t.

The apostrophe, asterisk and dagger superscripts on the vaccination probability cater for the modified flow diagram for sheep where $v' =$ zero, $v^* = vd$ (only effective if dosed) and $v^\dagger = v\,(d+r-rd)$ (only effective if either dosed or reverted).

Columns of the transition matrix contain probabilities of transition from a particular state and since all individuals must be accounted for, these must add to unity. For example, to obtain the proportion V_{t+1} of vaccinated animals at the end of a time period we have $V_{t+1} = svU_t + sv^*L_t + sv^\dagger P_t + svA_t + (sv + s(1-v)(1-r_v))V_t$. The first four old states are all multiplied by sv, this being the probability of surviving and being effectively vaccinated. Those surviving from the old vaccination immune state to V_{t+1} are either re-vaccinated with probability v or not re-vaccinated and do not revert with probability $(1-v)(1-r_v)$.

The elements of the matrix T are obtained directly from the flow diagram and the same form of matrix applies to each host.

Infection processes

All factors in the transition probabilities are taken as fixed except the probability of being infected, i, which is expected to vary with changing proportions in the U and P states. With homogeneous mixing of dogs and sheep it might be contended that the probability of an uninfected susceptible dog being infected is equal to the proportion of patent sheep. However, not all dogs in the same group will have access to infected sheep meat or offal and may not ingest sufficient to cause an infection when they do get access. Even then, parasite development need not automatically follow infection. So we might set $i_t = kP_t$ where k incorporates access and infectivity factors.

However, there is likely to be some lag in an effective control programme because non-foraging dogs are more likely to be fed with meat from older cull sheep. A higher proportion of these sheep harbour patent cysts than in the whole sheep population represented by P, especially for *E. granulosus*. So while k is reduced by accessibility and infectivity factors it is increased by dog feeding practice.

The transmission of infection to sheep is an even less definite process. Worm eggs can survive in pasture or soil for about one year (Sweatman & Williams, 1963*a*) so the contact level of a patent dog is greatly magnified. Further, dogs can range over territory leaving infective faeces out of proportion to their population.

For sheep we have used the same infection model as for dogs, with the current infection probability being proportional to the percentage of patent animals in the alternate host.

Table 1. *Typical latent and patent periods, the derived transition probabilities and percentage of animals with worms or cysts (latent + patent)*

	Dogs			Sheep		
	E. granulosus	*T. hydatigena*	*T. ovis*	*E. granulosus*	*T. hydatigena*	*T. ovis*
Latent period ...	7–8 weeks	7–8 weeks	5–16 weeks	1·5–2 years	6 weeks	6 weeks
Patent period ...	6–20 months	2–7 months	1–9 months	≃ 50% for many years	≃ 50% for many years	≃ 20% for many years
r_l	0·80	0·06	0·50	0·056	1·00	1·00
r_p	0·20	0·15	0·35	0·017	0·083	0·083
r_a	0·50	0·50	0·50	0·50	0·083	0·083
r_v	0·083	0·083	0·083	0·035	0·035	0·035
d_l	0·71	0·71	0·71	0·81	0·81	0·81
d_p	0·71	0·71	0·71	0·76	0·76	0·76
v	0·85	0·85	0·85	0·90	0·90	0·90
% 1958/9	6·6	12·0	0·2	—	60	—
% 1961/2	—*	—	—	37	—	—
% 1968/9	1·0	5·0	4·0	—	—	30

* — = no information from these dates used in simulation.

SIMULATION

Probability estimates

The components of the transition probabilities used are shown in Table 1.

Reversion probabilities were estimated from the somewhat sparse information available in the literature. From quoted durations the fraction of animals having not reverted (or converted) after 45 day intervals was taken as an estimate of $(1-r)^n$. Where the data covered several 45 day intervals it was possible to get more than one estimate of r from the decreasing probability of having not reverted. This information gave values of r that were more reliable. When little or no information was available the estimates for the other species were used in the case of r_l and r_p.

For reversion from the acquired immune state, the probabilities (r_a) were either 1/2, where it was thought that less than 0·5% would retain their immunity for 1 year ($(1-0{\cdot}5)^8$ i.e. 1/256 i.e. < 0·5%) or 0·083 where 50% would retain their immunity for a year ($(1-0{\cdot}083)^8 = 0{\cdot}5$, i.e. 50%). For reversion from vaccine immunity, values that seemed likely to be achieved were chosen. It was considered that between 25% and 50% of dogs might have reverted after one year while for sheep between 10 and 25% seems attainable.

The dose probability d is a combination of the dose being given and its effectiveness. A control programme would hope to achieve a 75% 'capture' for dogs (Friis, Sayers & McLean, 1967) and 90% 'capture' for sheep. The current anthelmintics were considered to be 95% effective for either latent or patent infections in dogs and 90 and 85% effective for latent and patent infections in sheep.

Similarly, a vaccine control programme would hope to reach 90% of all animals

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which is the value taken for v. The effectiveness of the vaccine once given is built into the probability of reversion. Survival probabilities s are taken from annual replenishment of the population. Currently there are 14 million hoggets out of a total flock of 50 million sheep, giving an estimate of $s = 0{\cdot}97$. Some data covering several regions of New Zealand on the age structure of dogs presented for dosing (Cook, 1967) show that 15% of 1423 dogs were less than one year old, giving an estimate of survival for one period of $s = 0{\cdot}98$.

Access and infectivity estimates

The coefficient k described previously is estimated in a different manner. With no control strategy a homogeneous mix of the two host populations should reach a steady state where the infection rates and the proportions in states U, L, P, A are constant. The available data on apparently uncontrolled infection levels in dogs and sheep is modified by the dog feeding practices of New Zealand farmers at the time when data showed maximum infection levels. Using these data, the steady state equations can be solved to give estimates of the probability of infection i which, when equated with k times the proportion of patent animals in the alternate host, gives estimates of k. The description of the steady state equations is given in Appendix II.

The data used for each species and host are shown in Table 1. *T. ovis* reached a peak in dogs in 1968/69 which corresponded with the period of cyst survey over the three killing seasons 1967 to 1969 (McNab & Robertson, 1972).

The *E. granulosus* data for sheep are a weighted proportion of the infection level as estimated by the Ewe Liver Survey (New Zealand National Hydatid Council Reports, 1963–78). This survey has run since 1961/62 and was initially based on examination of the first full mouth ewe across the killing board each day for each meat export establishment in New Zealand for the whole season.

Since 1972 the survey has been restricted to a three month autumn period and is based on the first ten sheep of up to 200 lines of sheep killed during that time. Gemmell (1961) gave an infection-by-age classification which showed that, with the lower infection levels in lambs and hoggets, overall infection is two thirds of full-mouth ewe infection when weighted by the proportion in each age group in the national flock. This 2/3 factor was applied to the survey data to give an overall infection level.

The *T. hydatigena* incidence is taken from Gemmell (1961), who estimated that 60% of older sheep were infected and from the earliest lamb liver survey data (Friis *et al.* 1967) which showed that 60% of lamb livers had cyst tracks.

Table 1 is assumed to be the measure of latent plus patent infections, and from the steady state equations these are related by $P = sr_lL/[1-s(1-r_p)]$.

Using this relation the estimates of patent infections in the uncontrolled situations were obtained as shown in Table 2.

The infection probabilities i, and access and infectivity factors (k) calculated by the steady state solution of Appendix II, are also shown in Table 2. The nature of the data and the assumption of a steady state being represented by the data suggest that for dogs the access and infectivity for *E. granulosus* (0·13) and

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Table 2. *The percentage of patent infections and estimates of* i *and* k *for the steady state model*

	Dogs		
	*E. granulosus**	*T. hydatigena**	*T. ovis*†
P%‡	6·4	12·5	2·9
i	0·016	0·026	0·012
k	0·127	0·130	0·087
	sheep		
P%	12·6§	19·8‖	13·5¶
i	0·012	0·032	0·019
k	0·180	0·260	0·648

* Derived from 1958/59 levels } Reports of the New Zealand hydatids
† Derived from 1968/69 levels } Council (1959, 1969)
‡ Latent plus patent data for dogs in Table 1 increased by 25% to account for dogs not captured and then apportioned to L and P in eqn. (1) of Appendix II.
§ Heath, unpublished; Cook, 1967.
‖ Friis *et al.* 1967; Sweatman & Plummer, 1957. Gemmell, 1968; Heath, unpublished.
¶ Gemmell, 1968; Heath, unpublished.
} Converted to an 'all sheep' figure from specific age classes of surveyed infections.

T. hydatigena (0·13) is 50% greater than for *T. ovis* (0·09). For sheep $k = 0{\cdot}18$, 0·26 and 0·65 for *E. granulosus*, *T. hydatigena* and *T. ovis* are used in the simulation.

Strategies

The strategies are no treatment or combinations of dosing or vaccination, or both treatments, for either host or both hosts. Strategies that were simulated and their probable outcomes are shown in Table 3. In all simulations the initial U, L, P and A states were the estimated percentages of the endemic situation.

DISCUSSION

The model presented here is decision-orientated. Its function is to compare the relative merits of control strategies; it should not be regarded as a definitive measure of the length of time taken for eradication. Some of the parameters in the model are little more than educated guesses, so we cannot claim that the simulation resembles the field situation in all respects. When these parameters are altered, the time necessary for eradication will vary. However, this does not materially affect the comparisons between control strategies. Their relative merits under a variety of input parameters as derived from Table 3, are as follows:

(i) Anthelmintics are most effective when the interval between treatments is not greater than the latent period for the parasite in that host. Thus, annual treatment of sheep with anthelmintics will be more effective against *E. granulosus* than *T. ovis* and *T. hydatigena*, whose latent periods are much shorter. For all three parasites, canine anthelmintics must be administered at less than 6–7

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Table 3. *A comparison of the effectiveness of various strategies* in decreasing the levels of larval cestodes in sheep and adult cestodes in dogs in New Zealand*

Strategy	Number of years to reach < 0·1% patent animals					
	Dogs			Sheep		
	E.g.	*T.h.*	*T.o.*	*E.g.*	*T.h.*	*T.o.*
Dogs dosed 6 monthly	> 10	> 10	> 10	> 10	> 10	> 10
Dogs dosed 6 weekly†	> 10	6	5	> 10	8	8
Dogs vaccinated annually‡	> 10	8	5	> 10	10	9
Sheep vaccinated annually§	> 10	> 10	8	> 10	> 10	10
Sheep dosed annually‖	6	> 10	8	7	> 10	> 10
Dogs dosed 6 weekly and vaccinated annually	> 10	5	4	> 10	7	6
Sheep dosed and vaccinated annually	5	6	3	4	7	5
Dogs dosed 6 weekly and sheep dosed annually	3	3	2	4	4	3
Dogs dosed 6 weekly and sheep dosed and vaccinated annually	2	2	2	4	3	3
Dogs dosed 6 weekly and vaccinated annually and sheep dosed annually	2	2	2	4	3	3
Dogs dosed 6 weekly and vaccinated annually and sheep dosed and vaccinated annually	2	2	2	3	3	3

* Strategies are based on a certain percentage of captured animals and on a stated degree of efficiency for each procedure, as follows:

	Capture	Effectiveness
†	75%	95%
‡	90%	50% for 1 year
§	90%	75% for 1 year
‖	90%	85%

week intervals for optimum effectiveness. In fact, it has been found by Heath, Parmeter & Lawrence (unpublished) that the latent period for *T. ovis* is 5–6 weeks in approximately 50% of dogs studied. If dogs are given access to infected sheep meat, then 6-weekly dosing in many instances will result in many thousands (estimated 1·5 million from a 6 week duration of infection) of eggs being deposited on the ground as the worm is voided. This factor could be the explanation for the increase in *T. ovis* worms in dogs and cysts in sheep in New Zealand despite dosing of dogs each 6 weeks with an effective anthelmintic. Our simulation has not taken this into account, having assumed that worms are removed before patency has been achieved, and so the simulated results in Table 3 do not fit the current New Zealand data for *T. ovis* infections.

(ii) The merits of vaccination depend upon its effectiveness on latent and patent infections and upon the duration of protection. In this simulation, anthelmintic treatment is assumed to clear latent and patent infections, whereas in practice, vaccination has little effect upon established infections in sheep. In these circumstances vaccination is more effective in populations that have a high proportion

of uninfected aninals, as compared with situations where most animals are in the latent of patent stage of infection.

(iii) In all cases the best results were obtained by treating both definitive and intermediate hosts. A single treatment, anthelmintic or vaccine, to each host was more effective than both anthelmintic and vaccine in a single host. This combined attack upon both hosts appears to offer the best prospects for eradication by reducing the overall level of infection to below some critical level that is necessary for survival of the parasite. The factor that offers the greater probability of successful eradication of *E. granulosus* appears to be its longer latent period in the intermediate host, which makes it vulnerable to anthelmintics for a longer period before patency ensues.

(iv) There are several consequences of the present New Zealand Hydatids Eradication policy which is based on dosing dogs and preventing their access to carcasses of the intermediate host. The very slow reduction of infection rates in sheep, particularly cull ewes, allows a potentially explosive situation; relaxation of control measures in dogs will very quickly result in a return to pre-treatment rates of infection. If one or all of the infections were totally eliminated from dogs, it would still be several years, equivalent to the maximum life span of the intermediate host, before the diseases could be said to have been 'eradicated'. Even with the present degree of co-operation from New Zealand dog owners, and assuming there would be no further relaxation due to a false sense of security after no cases had been found in dogs for several years, such an outcome seems unlikely.

(v) Treatment of both definitive and intermediate hosts makes eventual eradication much more feasible, although it does not render it by any means certain. Psychological and sociological considerations beyond the scope of this work must be taken into account. The relative lack of success of the present control programme in areas with a high percentage of Maoris (Burridge & Schwabe, 1977*b*) indicates a failure in effective communication. With the current regulations there may develop a similar apathy in a much wider community when overall infection levels drop. Some sort of farm certification may be needed, with financial incentives, to encourage the establishment and maintenance of infection-free farms and areas, and to exert greater peer-pressure on recalcitrant farmers and dog owners. Serological surveillance may have a useful place in such as system.

This model represents a first approximation that could be improved upon in several ways. The ultimate simulation might be performed on a stochastic or Monte Carlo model with more precise biological parameters than are currently available, but we are encouraged by Bailey's (1975) assertion that 'Great mathematical rigour or epidemiological predictive accuracy are in general probably unnecessary, though there is no reason to avoid them if they can be included without undue labour.'

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APPENDIX I. Biological data concerning the latent and patent periods of *Echinococcus granulosus*, *Taenia hydatigena* and *T. ovis* worms in dogs and cysts in sheep

Latent periods			Patent periods		
E. granulosus	*T. hydatigena*	*T. ovis*	*E. granulosus*	*T. hydatigena*	*T. ovis*
Worms					
7–8 weeks	7–8 weeks	5–16 weeks	6–20 months or longer	2–7 months or longer	1–9 months or longer
16, 24, 19, 18, 25, 26, 1, 15, 20, 17	23, 6, 4, 9	22, 10, 9, 14	24, 7, 1	23, 7, 6, 4, 9	22, 7, 9
Cysts					
1·5–2 years	≃ 6 weeks	≃ 6 weeks	≃ 50% for many years	≃ 50% for many years	≃ 20% for many years
24, 2, 13	23, 7, 12	22, 7, 3, 21, 5, 12	24, 7, 2, 13	23, 7, 8, 13	21, 11, 8

https://doi.org/10.1017/S0022172400026917 Published online by Cambridge University Press

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APPENDIX II

With no dosing and no vaccination the transition matrix gives the steady state equations

$$\begin{aligned} L &= s(iU+(1-r_l)L), \\ P &= s(r_lL+(1-r_p)P), \\ A &= s(r_pc_{rp}P+[1-r_a(1-c_{ra})]A) \end{aligned} \tag{1}$$

Where r_p is small and r_a is not small the A state may be ignored and using $U+L+P = 100$ gives a simple expression for i.

The probability of acquired immunity on reversion has been set as the probability of being challenged at least once (or twice for sheep infected with *E. granulosus*) during the average time spent in the state from which the animal is reverting. Interpreting the reversion probabilities as a rate c_r is equated to the probability of at least one challenge with infection probability i during $1/r$ time intervals. Assuming the infection rate i to be small

$$c_r = 1-(1-i)^{1/r} \simeq i/r \tag{2}$$

for most of the reversion probabilities.

Substituting eqn. (2) in eqn. (1) and using $U+L+P+A = 100$ gives a quadratic equation for i in terms of the percentage of patent animals.

For sheep, where r_p is very small, c_{rp} cannot be approximated by eqn. (2) but a similar equation for i in terms of P is derived which can be solved by simple numerical procedures.

https://doi.org/10.1017/S0022172400026917 Published online by Cambridge University Press