6

Epidemic Waves in Animal Populations: A Case Study

Preface

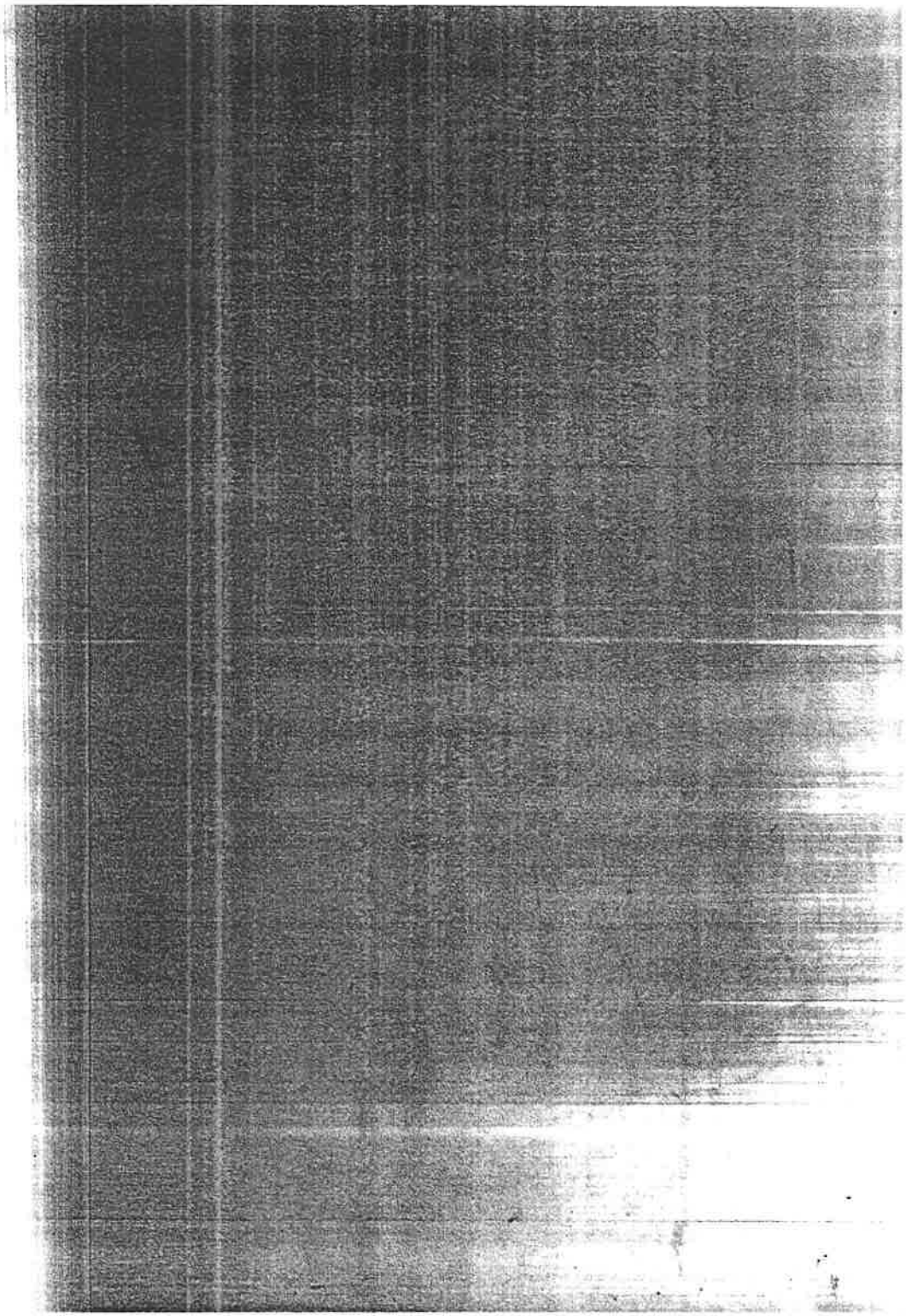
The application of mathematics to biology has led to tremendous advances in the understanding of plant and animal dynamics and growth. Paramount among these areas is the spread of diseases. The application described here motivated the adaption of diffusive wave analysis to the spread of haemorrhagic disease among rabbit populations in New Zealand. The disease was introduced as an attempt, initially illegal but subsequently legalised, to control the burgeoning rabbit population in highly productive farming areas. The conceptual basis adopted was that there is a threshold maximum value of the spatial density of healthy rabbits ("susceptibles") below which the disease will not propagate. The dependence of the wave speed on the density (when it is above the threshold) can also be evaluated.

The procedure is generic and can be applied to a wide range of modelling scenarios. The nonlinear dynamics involves a range of parameters which are determined from data: the infectivity of the disease, the dispersion constant, and the infected death rate.

The purpose of this project was to see if the predicted threshold matches that seen in practice, with a view to assisting the understanding of the disease spread and the ability of it to deal with the huge problem of the endemic rabbit population many farming areas have.

The values obtained for the threshold density are close to those observed in practice and indicate that the model used is approximately correct. Of course many questions remain about the method of disease transmission. The effect of wind is taken into account. The methods employed are a healthy mix of analytical and numerical techniques, demonstrating again the interplay that underpins many successful solutions of nonlinear systems.

A major scientific study was launched in 1998 to investigate aspects of the disease, and the modelling aspects play a key role in underpinning and auditing the overall focus. This project is part of that effort.



6.1 The History of Rabbit Haemorrhagic Disease and its Introduction into New Zealand

Rabbit Haemorrhagic Disease (RHD) is a viral disease which affects European rabbits ([29]). It first appeared in China in 1984 and has subsequently spread through most of Europe as a result of human trading of rabbits on the domestic market ([1]). Its initial appearance may have been due to the mutation of a benign strain of the virus, but this is uncertain – although there is evidence to suggest that a benign strain does exist ([3]). In 1993 the virus was introduced into Australia under quarantine by CSIRO (Commonwealth Scientific and Industrial Research Organisation) for the testing of its capabilities as a biological control of wild rabbits in both Australia and New Zealand ([17]). In 1995 a quarantined experiment began on Wardang Island but, despite precautions, the virus escaped onto the mainland and spread quickly to many other parts of Australia over the following months ([6]).

Rabbits first arrived in New Zealand with European colonists as early as 1777 ([11]). They were originally carried aboard sailing ships as a food source, but by 1866 they were regularly bred and distributed throughout parts of New Zealand's mainland and offshore islands by acclimatisation societies. They responded favourably to New Zealand's conditions. With an adequate food supply available throughout most of the year, the breeding season was lengthened; by 1889, populations had exploded, and they had become a pest.

Although it is not well quantified economically or environmentally, rabbits have a large negative impact on one of New Zealand's main industries, agriculture. Rabbit damage to New Zealand pastures has been an ongoing problem over the last century and more than $NZ600m has been spent by governments since 1950 in a bid to limit rabbit numbers ([15]). Although rabbits were previously, and in some parts of the world still are, seen as attractive pet animals, the situation of overpopulation and degradation of agricultural land had reached crisis proportions in New Zealand to the degree that desperate control measures were needed. The scientific and agricultural communities had investigated many different alternatives on how to deal with this serious problem which had potentially considerable economic threat. Biological controls were considered, and in 1987 an application to import myxomatosis virus as a biological control was rejected. Similarly, on July 2nd 1997, the application to have RHD legally introduced into New Zealand was refused by P. J. O'Hara, the Deputy Director General of MAF (Ministry of Agriculture and Forestry), ([19]). However, by August 25th 1997, there were rumours circulating that the disease was in New Zealand. This was later confirmed when the virus was found on four properties in Cromwell in the southern part of the South Island of New Zealand. After its initial release, RHD was spread mainly through the human vector. Illegally, farmers were instigating spot releases and then taking the livers of infected rabbits, grinding them in a kitchen blender and spraying this mixture over chopped carrots. The carrots were then aerially distributed over large areas. The virus was also released in the North Island and soon it appeared naturally at certain sites. It was suspected that releasing virus in this manner might be actually immunising large numbers of rabbits. It became evident that the virus was becoming established and hopes of containment or better still eradication were fading. So, in an effort to control RHD, the use of the virus as a biological control was legalised.

6.2 What is Known about the Disease

Even in the light of studies in Australia, Europe, and New Zealand, little is known about the spread of RHD. There are two possible modes of transport, the faecal/oral route ([5]) and via a possible windborne vector ([1]). The latter would explain why RHD spreads long distances in a short space of time (e.g. its escape from Wardang Island in Australia ([28])). In many Australian sites, viable RHD virus has been detected in blowflies ([24]).

Spread seems to be radial with a tendency in the prevailing wind direction in some cases ([14]). RHD epidemics occur seasonally ([29]). This is no surprise if the mechanism for the spread of the disease is via an insect vector, and also no surprise if the epidemic occurs when population densities are high, or if there is an increase of rabbit-to-rabbit contact.

6.3 What We Want to Know

From the farming perspective, the aims are:

- To minimise the adverse effects of RHD and implement management to gain full use of RHD in order to maximise the reduction of rabbit densities. For example the current percentage of kills after an epidemic of RHD is anywhere between 10% and 90% ([21]). The aim is to have a 90% kill rate in all environments.
- To find out whether RHD should be spread as a biocide (where RHD virus is spread by distribution of baits over an area) or a biocontrol (where the virus is initiated at a point source and then allowed to spread naturally).
- To determine when the virus should be released (i.e. which season), and under what conditions (temperature, rabbit density, etc.).
- If the virus is not effective, to decide on the next move.

The questions from the scientific perspective are:

- How does RHD work, and is it predictable?
- Under what conditions does it persist, and how often are epidemics? For example persistence might depend on rabbit density, time of year, temperature, or different vectors.
- Are flies the aerial vector?

Such studies are important because they may conclude that RHD as a biological control may be ineffective in certain circumstances, both in New Zealand and in other countries. Understanding a virus is crucial if it is to be used as a biological control.

6.4 The Modelling, Analytical/Numerical

We are trying to model the (natural) spread of RHD in New Zealand (and elsewhere) from a point source in order to find the critical population density threshold below which the disease will no longer persist. We can also use the model to predict the speed of the wave of infection. A specific question we addressed is how the speed of infection depends on the density of the population. A key ingredient is the need of the model outcomes to match the available data. This was the major factor in this project: that we needed to be able to predict observed outcomes and thereby have confidence in the model to predict the spread of the disease in new circumstances. This matching of model outcomes to real data is an important aspect of the modelling process.

Estimates of rabbit densities can be calculated in the field using spot light counts along a one km transect line. With this in mind we will extend an SIR (susceptibles, infectives, recovereds) model of RHD developed by [2] to incorporate a one-dimensional spatial spread of rabbits and diffusion of infection (figure 6.1). A similar approach was taken by [23], who investigated the spread of foot and mouth disease in feral pigs.

We let $I(x, t)$ be the spatial density of infectives at position x in kilometres at time t in days. We assume the point source release is at the origin $x = 0$, giving an initial condition for infectives $I(x, 0) \propto \delta(x)$, where δ is the Dirac delta function. Similarly we let $S(x, t)$ and $R(x, t)$ be the density of susceptibles and recovereds (who are immune) respectively at position x, in kilometres, and at time t, in days. We assume initially that there are no rabbits with immunity ($R(x, 0) = 0$) and that the density of rabbits is uniform ($S(x, 0) = S_0$).

We assume that susceptible and immune rabbits are essentially stationary, but we incorporate diffusion and advection, with parameters D and v respectively, in the equation for the infectives. This allows for the diffusion of the infection by rabbits moving randomly and also the possibility of the infection being spread via a wind vector. Such diffusion models are derived from a random walk approach in (among others), [20], [9], [10], [25], and [16]. [8] derives the diffusion equation using the Fokker–Planck (stochastic) approach. The random walk derivation of the diffusion equation for infectives is as follows: we assume that the number of infectives at position x at time $t + \Delta t$ (given by $I(x, t + \Delta t)$) is equal to those infectives who moved from position $x + \Delta x$ a distance Δx to the left in the time interval from t to $t + \Delta t$ (i.e. $p_1 I(x + \Delta x, t)$, where p_1 is the probability of moving a distance Δx to the left) together with those infectives who were at position $x - \Delta x$ at time t and moved a distance Δx to the right during the same time interval (i.e. $p_2 I(x - \Delta x, t)$, where p_2 is the probability of moving a distance Δx to the right) added to those infectives who did not move at all from position x (i.e $(1 - (p_1 + p_2))I(x, t)$). That is,

$$I(x, t + \Delta t) = p_1 I(x + \Delta x, t) + p_2 I(x - \Delta x, t) + (1 - (p_1 + p_2))I(x, t).$$

Taylor's series expansions in Δx and Δt of the terms are taken. The equation is then rearranged and higher-order terms are ignored. When Δx and $\Delta t \to 0$, the equation becomes

$$\frac{\partial I}{\partial t} = D\frac{\partial^2 I}{\partial x^2} - v\frac{\partial I}{\partial x} \tag{6.1}$$

where $D = \lim_{\Delta x, \Delta t \to 0}((p_1 + p_2)/2)(\Delta x^2/\Delta t)$ and $v = \lim_{\Delta x, \Delta t \to 0}(p_2 - p_1)(\Delta x/\Delta t)$ are the diffusion and advection (constant) coefficients respectively (it should be noted that, for these limits to exist, p_1 and p_2 must depend on Δx and Δt). The actual calculation of the diffusion and advection coefficients in the field is discussed in section 6.4.3. Equation (6.1) is the standard diffusion/advection equation. Adding spread of infection, mortality due to disease, and immunity gives equation (6.3) for infectives below. Our model assumes that the rabbits can die by contracting the disease which has a constant mortality rate d per capita per day, and that we have a transmission coefficient β, which represents the proportion of susceptibles that can be infected each day by contact with one infected rabbit. Thus the spread of infection is proportional to the product of the densities of infectives and susceptibles ([13]).

We are able to ignore the spatial movement of the susceptible population as a first approximation, and so equation (6.2) is a conservation equation for susceptibles, with the term βSI being the loss due to becoming infected. We assume that both susceptibles and recovereds can breed. The breeding rate

a of rabbits depends on pasture biomass availability ([4]) but, because the duration of infection is of the order of 40–80 days ([22]), we assume the breeding rate is constant over this time.

Similarly equation (6.4) is the conservation equation for the recovereds, where the rate of immunity is r per capita per day. Equations (6.5), (6.6), and (6.7) are initial conditions for susceptibles, infectives, and recovereds respectively. Thus, the system of equations and initial conditions governing the above assumptions is:

$$\frac{\partial S}{\partial t} = -\beta SI + a(S + R) \tag{6.2}$$

$$\frac{\partial I}{\partial t} = D\frac{\partial^2 I}{\partial x^2} - v\frac{\partial I}{\partial x} + \beta SI - dI - rI \tag{6.3}$$

$$\frac{\partial R}{\partial t} = rI \tag{6.4}$$

$$S(x, 0) = S_0 \tag{6.5}$$

$$I(x, 0) \propto \delta(x) \tag{6.6}$$

$$R(x, 0) = 0. \tag{6.7}$$

See figure 6.1 for descriptions of individual terms and table 6.1 for the units of parameters used in the model.

6.4.1 Case: No Immunity ($R(x, t) = 0$) and No Breeding ($a = 0$)

As a first step to understanding our system of equations, we assume that there is no immunity (i.e $R(x, t) = 0$) and that the release of the virus is not in the breeding season ($a = 0$). Our system becomes

$$\frac{\partial S}{\partial t} = -\beta SI \tag{6.8}$$

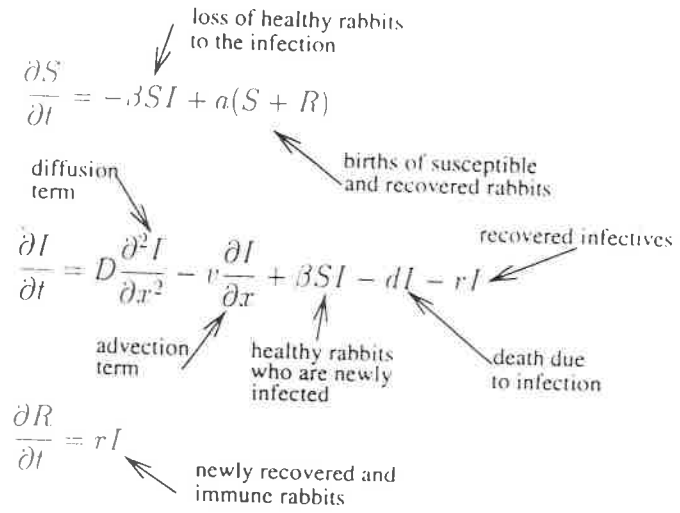
$$\frac{\partial I}{\partial t} = D\frac{\partial^2 I}{\partial x^2} - v\frac{\partial I}{\partial x} + \beta SI - dI \tag{6.9}$$

$$S(x, 0) = S_0 \tag{6.10}$$

$$I(x, 0) \propto \delta(x) \tag{6.11}$$

which is discussed in [16] (with $v = 0$) in terms of the spread of rabies in foxes. It is shown that the system has travelling wave solutions (figure 6.2) of the form $I(x, t) = f(x \pm ct)$ and $S(x, t) = g(x \pm ct)$.

In order to find the speed of the wave of infection analytically, [16] assumes that the rate of change of susceptibles is much slower than the rate of change



with initial conditions

$S(x, 0) = S_0$ ← initially there is a uniform density of susceptibles

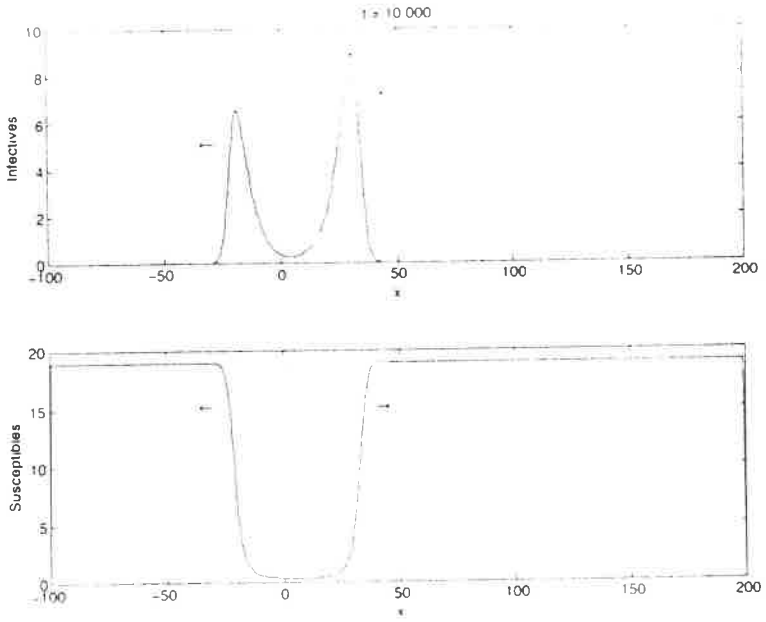
$I(x, 0) \propto \delta(x)$ ← the infection has a point source at the origin

$R(x, 0) = 0$ ← initially there are no immune rabbits

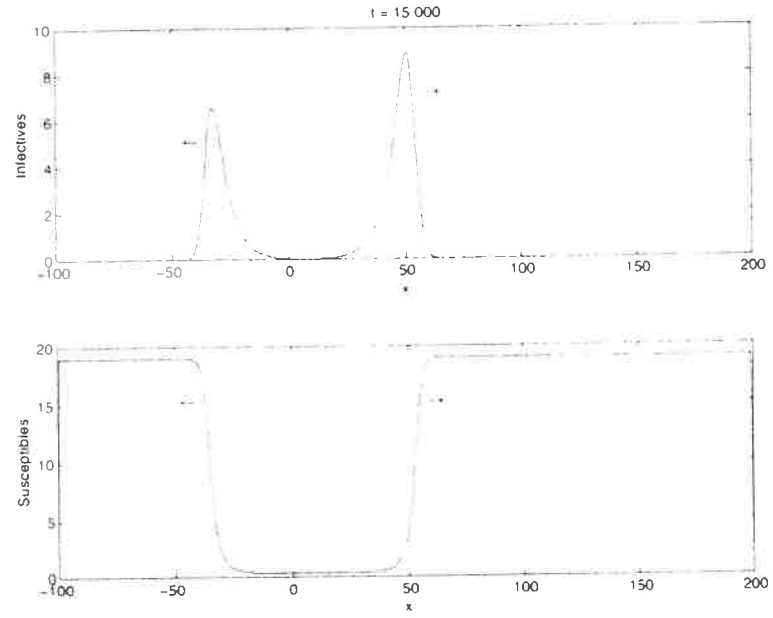
Fig. 6.1. Partial differential equations and initial conditions for susceptibles, infectives, and recovereds ($-\infty < x < \infty$)

Table 6.1. *Summary of Parameters*

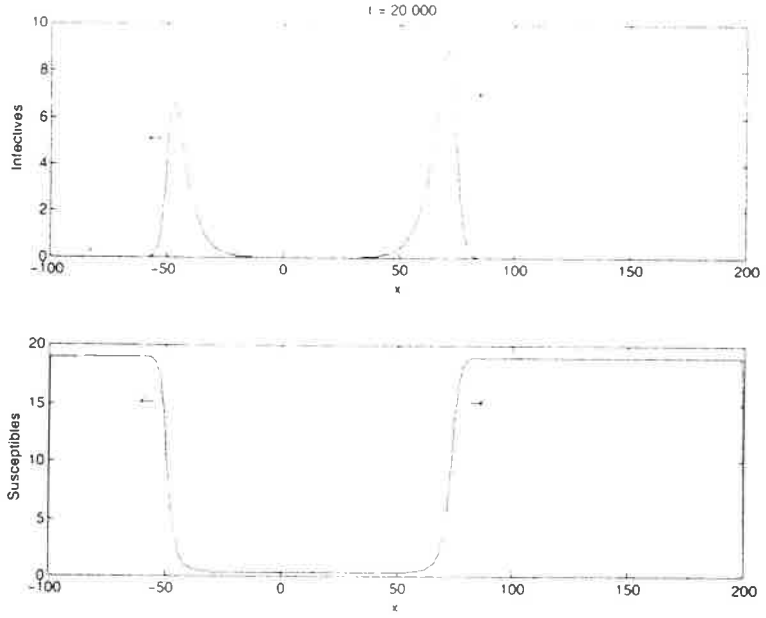
Parameter	Units	Description
D	$km^2 day^{-1}$	diffusion coefficient
v	$km\ day^{-1}$	advection coefficient
βS_0	day^{-1}	transmission coefficient scaled by initial population
r	day^{-1}	per capita recovery rate
d	day^{-1}	per capita death rate due to infection
a	day^{-1}	per capita birth rate



(a) $t = 10$ (Fig. 6.2)



(b) $t = 15$ (Fig. 6.2)



(c) $t = 20$ (Fig. 6.2)

Fig. 6.2. Travelling wave solutions of the system where there is no immunity and no breeding. The arrows indicate the direction in which the wave is travelling. Parameter values are $\beta = 0.14$, $d = 0.67$, $D = 1.5$, $v = 0.5$, $S_0 = 19$, $I(0,0) = 1$, $\Delta t = 0.1$, $\Delta x = 1.5\sqrt{2D\Delta t}$, $t_{min} = 0$, $t_{max} = 25$, $x_{min} = -100$, $x_{max} = 200$. The units of parameters are summarised in table 6.1

of infectives (i.e at the wave front, the density of susceptibles is approximately constant). It is then possible to solve the system of equations analytically by linearising about S_0. That is, we let $S(x,t) = S_0$ be a constant. Using Fourier transforms ([26]), we find

$$I(x,t) \sim \frac{1}{\sqrt{t}} e^{-(x-vt)^2/4Dt} e^{(\beta S_0 - d)t}$$

which is plotted in figure 6.3. Note that this is only an approximate solution of our system, and it does not have travelling waves, but we can still use it to find the speed of infection and then numerically verify this speed.

To find the speed of infection analytically, we note that, since $I(x,t) \to 0$ as $t \to \infty$, $I(x,t)$ cannot oscillate about 0. We therefore require

$$(\beta S_0 - d)t < \frac{(x-vt)^2}{4Dt} \quad \Rightarrow \quad c = \frac{x}{t} > |v \pm 2\sqrt{D(\beta S_0 - d)}|.$$

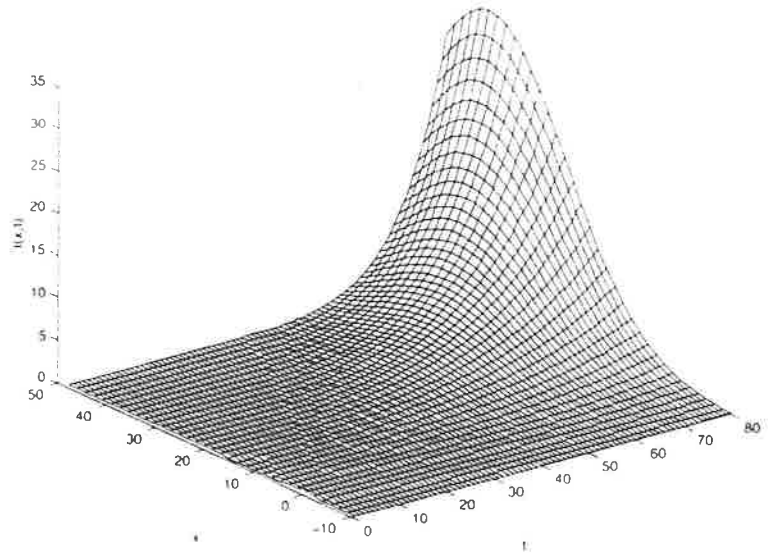


Fig. 6.3. The analytic solution for parameter values $\beta = 0.04$, $d = 0.67$, $a = 0$, $D = 1.5$, $v = 0.5$, $S_0 = 19$, $\Delta t = 1$, $\Delta x = 1.5\sqrt{2D\Delta t}$, $t_{min} = 0$, $t_{max} = 80$, $x_{min} = -100$, $x_{max} = 200$. The units of parameters are summarised in table 6.1

Therefore the centre of the wave travels with speed $c = v$, the front of the wave (travelling to the right) travels with speed $c > v + 2\sqrt{D(\beta S_0 - d)}$, and the front of the wave (travelling to the left) travels with speed $c > |v - 2\sqrt{D(\beta S_0 - d)}|$.

These analytical wave speeds ($c > v \pm |2\sqrt{D(\beta S_0 - d)}|$) have been found assuming that $\partial S/\partial t = 0$. If we drop this assumption, then we must resort to numerics to solve the system.

Explicit finite-difference numerical methods can behave pathologically when solving diffusion equations and are dependent on the step size for convergence ([7]). We therefore use an implicit method by taking the difference quotient for the second derivative and first derivatives ($\partial^2 I/\partial x^2$ and $\partial I/\partial x$) centred at (x_i, t_{j+1}) ([7], [27]), i.e

$$\frac{\partial I(x_i, t_j)}{\partial x} = \frac{I(x_{i+1}, t_{j+1}) - I(x_{i-1}, t_{j+1})}{2\Delta x},$$

$$\frac{\partial^2 I(x_i, t_j)}{\partial x^2} = \frac{I(x_{i+1}, t_{j+1}) - 2I(x_i, t_{j+1}) + I(x_{i-1}, t_{j+1})}{(\Delta x)^2}.$$

We also add boundary conditions that $I(x_{min}, t) = I(x_{max}, t) = 0$. To numerically approximate the initial condition $I(x, 0) \propto \delta(x)$, we assume that there is a density of I_0 infected rabbits in a neighbourhood of the origin.

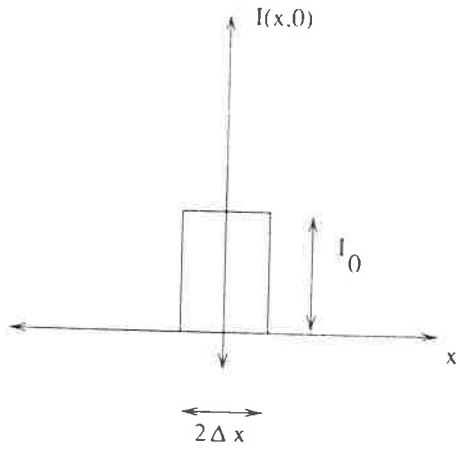


Fig. 6.4. Numerical approximation of the initial force of the infection. We assume that the density of infectives is $2\Delta x I_0$ in a neighbourhood of the origin

The force of the infection, $F(t)$, is defined as the total amount of infection present at time t; i.e. $F(t) = \int_{-\infty}^{\infty} I(x, t)dx$. Initially ($t = 0$) the force of infection is approximated numerically (using a step size Δx) by $F(0) = \int_{-\infty}^{\infty} I(x, 0)dx = 2I_0\Delta x$ (figure 6.4).

Numerically we can see that, when $\beta \neq 0$, a wave split occurs (figure 6.5) which represents waves travelling to the left and right of the origin. In figure 6.6 we can see that wave speeds to the left and right attain their minimum, i.e. the wave speed is $c = |v \pm 2\sqrt{D(\beta S_0 - d)}|$. Note that, in figure 6.6, a negative wave speed indicated that the wave is travelling to the left. (Similarly figures 6.7 and 6.8.)

Thus we have the wave speed $c = x/t$ moving faster than the wind speed v, provided that S_0 is big enough. If $S_0 < d/\beta$, then the wave of infection will not propagate. This is the same critical density as found in [2] using a nonspatially structured model.

6.4.2 Case: No Immunity ($R(x, t) = 0$) But Breeding Season ($a \neq 0$)

As soon as a becomes nonzero, we no longer have travelling wave solutions. Similarly the wave speed cannot be calculated as before, since we cannot make the assumption that $S(x, t)$ is constant at the wave front. However, we can see numerically (figure 6.7) that the infection at time t is travelling at a speed of approximately

$$v \pm 2\sqrt{D(\beta \times \max(S(x, t_0)) - d)}$$

where $\max(S(x, t_0))$ is the maximum number of susceptibles at time t_0.

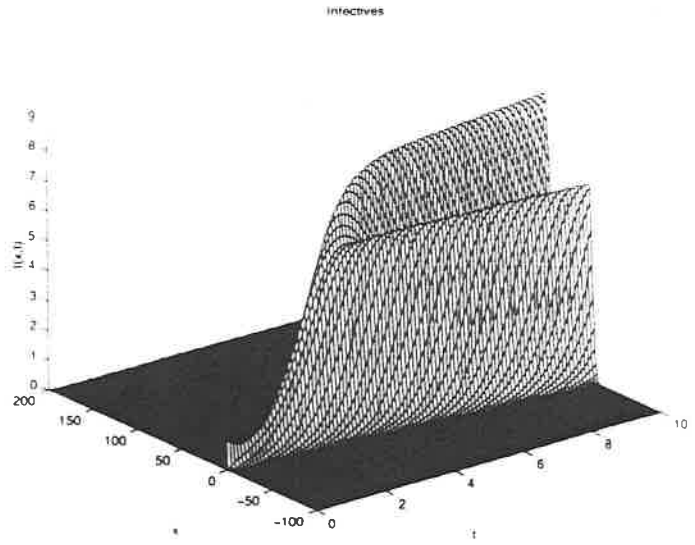


Fig. 6.5. The numerical solution of the system showing the wave split when $\beta \neq 0$. Parameter values are $\beta = 0.14$, $d = 0.67$, $a = 0$, $D = 1.5$, $v = 0.5$, $S_0 = 19$, $I(0,0) = 1$, $\Delta t = 0.1$, $\Delta x = 1.5\sqrt{2D\Delta t}$, $t_{min} = 0$, $t_{max} = 10$, $x_{min} = -100$, $x_{max} = 200$. The units of parameters are summarised in table 6.1

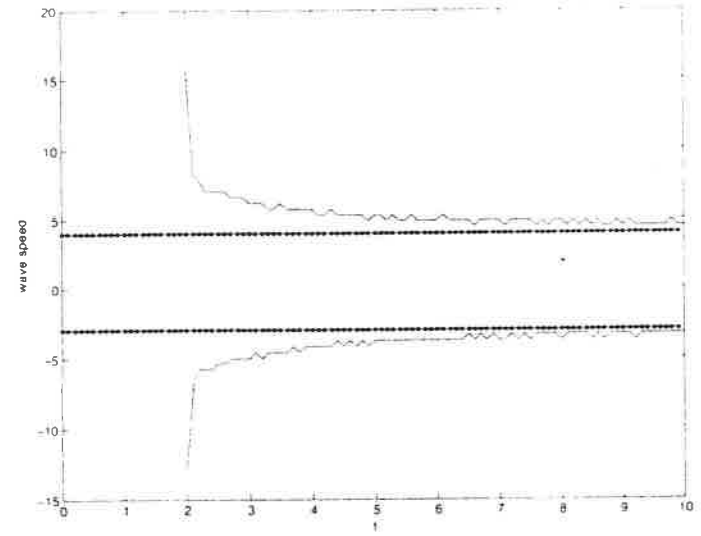


Fig. 6.6. The starred line "***" is the analytic wave speed $v \pm 2\sqrt{D(\beta S_0 - d)}$. The solid line is the numerical wave speed. The parameter values are $\beta = 0.04$, $d = 0.67$, $a = 0$, $D = 1.5$ $v = 0.5$, $S_0 = 19$, $I(0,0) = 1$, $\Delta t = 0.1$, $\Delta x = 1.5\sqrt{2D\Delta t}$, $t_{min} = 0$, $t_{max} = 10$, $x_{min} = -100$, $x_{max} = 200$. The units of parameters are summarised in table 6.1

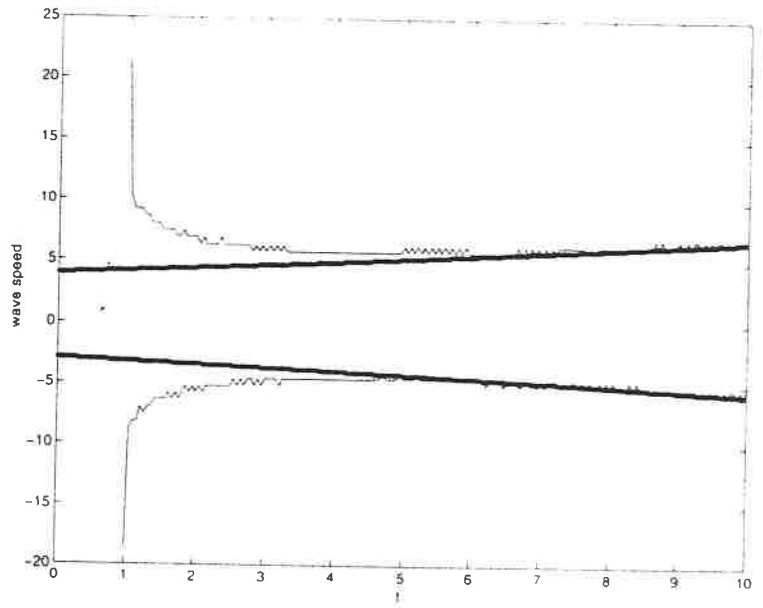


Fig. 6.7. The starred line "***" is the analytic wave speed $v \pm 2\sqrt{D(\beta \times \max(S(x,t_0)) - d)}$. The solid line is the numerical wave speed. The parameter values are $\beta = 0.14$, $d = 0.67$, $a = 0.1$, $D = 1.5$, $v = 0.5$, $S_0 = 19$, $I(0,0) = 1$, $\Delta t = 0.1$, $\Delta x = 1.5\sqrt{2D\Delta t}$, $t_{min} = 0$, $t_{max} = 10$, $x_{min} = -100$, $x_{max} = 200$. The units of parameters are summarised in table 6.1

6.4.3 Parameter Values

Because RHD has been in New Zealand only for such a short time, there are very few data available. Scarcity of data is a common problem especially in biological modelling situations. As more data become available, we can further validate and refine our model. In the meantime we have some data from Earnscleugh station in Central Otago in the South Island of New Zealand where RHD arrived naturally. Spotlight counts prior to the arrival of RHD were 35 rabbits per 1 km of transect. After the epidemic had swept through (around about 80 days with an average speed of approximately 200 metres per day), spotlight counts had reduced to 12 rabbits per 1 km of transect. This is a reduction of about 67% [18].

The life expectancy of a rabbit which has contracted the virus is $T = 1.5$ days ([2]). Therefore the value of d, the mortality rate due to the virus, is $d = 1/T = 0.667$ per day.

The transmission coefficient β is calculated in [2] using combined data from Spain and four sites in Gum Creek Australia. The initial number of susceptibles that were infected by one infected rabbit was $\beta S_0 = 2.1$ per day.

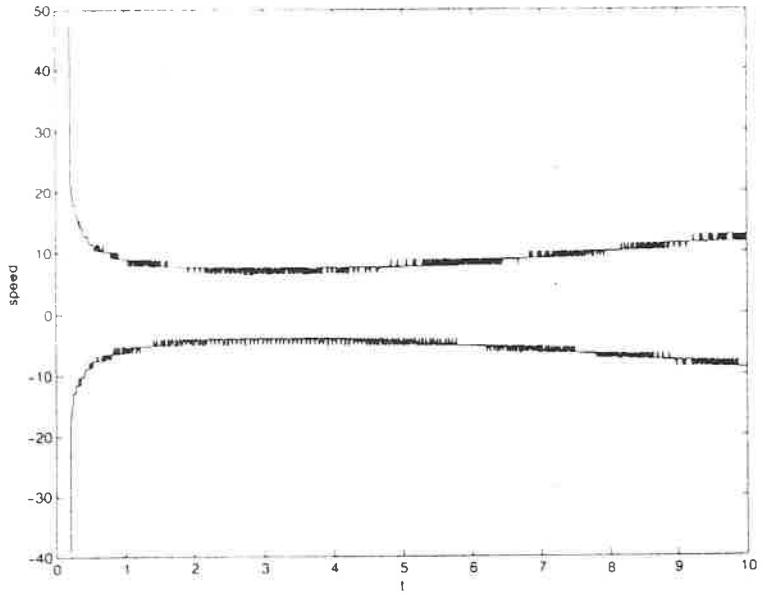


Fig. 6.8. The smooth lines are the analytic wave speeds $c(t) = v \pm 2\sqrt{D(\beta \times \max(S(x,t)) - (d+r))}$. The jagged line is the numerical wave speed. The parameter values are $\beta = 0.14$, $d = 0.67$, $a = 0.2$, $r = 0.2$, $D = 1.5$, $v = 1.5$, $S_0 = 19$, $I(0,0) = 1$, $\Delta t = 0.01$, $\Delta x = 1.5\sqrt{2D\Delta t}$, $t_{min} = 0$, $t_{max} = 10$, $x_{min} = -150$, $x_{max} = 150$. The units of parameters are summarised in table 6.1

We combine this value with the value of S_0 from the Earnscleugh site in New Zealand where densities, prior to the introduction of RHD, were 35 rabbits per km of transect. Thus $\beta = 0.06$ per day per unit density of infectives.

Using our values of d and β, we predict the critical density of susceptibles below which the disease will no longer propagate to be $d/\beta \sim 11$ rabbits per km of transect. Post-RHD densities at Earnscleugh were 12 rabbits per km of transect and there have been no further outbreaks of RHD. However d/β is extremely sensitive to values of β close to zero. Also β, which may not necessarily be constant, is sensitive to values of S_0. We clearly need further analysis of data sets in cases where RHD spread and in cases where it did not.

To find the actual speed of the wave requires the calculation of the diffusion coefficient D and the advection coefficient v. There are many ways of calculating diffusion coefficients. In [9], [20], and [10], the diffusion coefficient is calculated via the random walk derivation of the diffusion equation. [8] and [20] use the Fokker–Planck approach. Other methods are discussed (in relation to practical problems) in [20].

A way of calculating D is shown in [25] and [16] and will be discussed here. This method is practical in the field and has been used by (among others) [23], [12] and [20].

First, consider the basic 2-dimensional diffusion equation, which has a corresponding 2-dimensional diffusion coefficient D_2:

$$\frac{\partial N}{\partial t} = D_2\left(\frac{\partial^2 N}{\partial x^2} + \frac{\partial^2 N}{\partial y^2}\right)$$

with the initial condition $N(x, y, 0) \propto \delta(x, y)$. This has the solution ([26])

$$N(x, y, t) = \frac{1}{4\pi D_2 t} \exp \frac{-(x^2 + y^2)}{4D_2 t}$$

which is the 2-dimensional normal distribution of mean $\mu = (0, 0)$ and variance $\sigma^2 = 2D_2 t$. In polar coordinates (where $r^2 = x^2 + y^2$)

$$N(r, t) = \frac{1}{4\pi D_2 t} \exp \frac{-r^2}{4D_2 t}.$$

If we think of $N(r, t)$ not as a population density function but as the probability density function that an individual is at location r at time t then $\langle r^2 \rangle$, the mean square displacement by an individual's random walk during time t, is given by

$$\langle r^2 \rangle = \int_0^\infty r^2 N(r, t) 2\pi r dr = 4D_2 t.$$

This gives us a way of calculating a 2-dimensional diffusion coefficient

$$D_2 = \frac{\langle r^2 \rangle}{4t}.$$

To calculate a one-dimensional diffusion coefficient, we note that $D_1 = 2D_2$ ([20], [10]) and so

$$D_1 = \frac{\langle r^2 \rangle}{2t}.$$

To calculate the (one-dimensional) diffusion coefficient D for the RHD model, we let

$$D = \frac{\text{mean square displacement of a rabbit from its original location at time } t}{2t}.$$

Home ranges of rabbits vary depending on season, time of day or night, topography, distances to feeding grounds, and density of rabbits, but are approximately one hectare (100 m × 100 m) ([11]). We therefore assume that

the maximum distance is 140 m per day, which gives a diffusion coefficient of $D = \frac{0.14^2}{2} = 0.0098$ km^2 per day.

The advection coefficient v in diffusion models is the drift velocity or the convective flux ([9]). In our model, v is the wind speed. If we assume that there was no wind at Earnscleugh, then we predict that the infection will travel at

$$c = 2\sqrt{D(\beta S_0 - d)} = 2\sqrt{0.0098(0.06 \times 35 - 0.667)} \sim 237 \text{ m per day.}$$

It was, however, observed at Earnsleugh that the speed of infection was on average 200 m per day ([18]) and travelled in the direction of the prevailing wind. Our speed is therefore a rough estimate. D could be too high, and we have not taken into account immunity which would slow down the speed of the wave of infection.

6.5 Immunity

There are a number of different types of immunity. First, young rabbits have natural resistance because of their age (0–8 weeks.) Second, if the mother has been exposed to the disease and survived, then the maternal antibodies are passed across the placenta and the young rabbit is immune. But this maternal immunity only lasts 10 weeks, and then the rabbit becomes susceptible. Third, adult rabbits can have antigen immunity. When they are challenged with RCD, they produce antibodies and are subsequently tested as seropositive. This immunity is for life. There could be other types of immunity: for example cellular immunity. For the purposes of modelling, we will only consider life immunity. We let r be the per capita rate of immunity in the rabbit population. The system of partial differential equations and initial conditions is that shown in figure 6.1.

Using our previous results, we can numerically verify (see figure 6.8) that the analytical wave speed is

$$c(t) = |v \pm 2\sqrt{D(\beta \times \max(S(x,t)) - (d+r))}|$$

giving a threshold density of $\max(S(x,t)) = (d+r)/\beta$.

It can be seen (figure 6.9) that, once the infection has passed through, the immune rabbits breed producing further susceptibles. The density of susceptibles that remain after an RHD epidemic may be below the threshold required for the disease to persist. Predators may keep densities low, but the need to cull immune rabbits is evident.

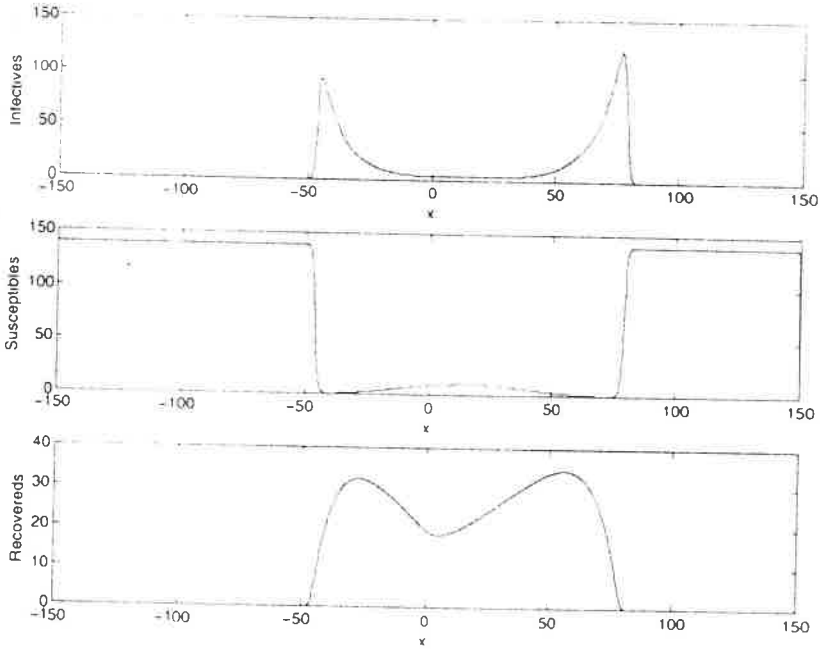


Fig. 6.9. Parameter values are the same as in figure 6.8

6.6 Results and Conclusions

This work has given a quantitative formula for the speed of the wave of infection, which depends on the density of susceptibles, the wind speed, the diffusion coefficient, the transmission coefficient, the recovery rate, and the death rate due to infection. From this it is possible to calculate the threshold density below which the infection will not spread. This threshold density is the same as that concluded in a nonspatial *SIR* model by [2]. For the calculated value of the speed to be meaningful, the parameters on which it depends must be evaluated accurately from real data. However, data adequate for this purpose are difficult to obtain in New Zealand, given the short time span that RHD has been present in the country.

The model predicts that, for the RHD virus to spread through a property, there must be a sufficiently high density of susceptibles. Thus, the best time to seed RHD would be when the entire population is challenged (i.e. when young no longer have immunity and have left the nest). After the RHD epidemic has passed through, the remaining population needs to be culled in order to eradicate any immune rabbits. The limiting factor in this study has been the paucity of data. These will take time to collect. The model developed here appears to make predictions in the range shown in the field.

Models of this structure are widely used in epidemiological studies elsewhere with considerable success. Spatial structure is best handled by partial differential (diffusion-type) models, thereby using the well-developed theory these equations have. We have communicated the outcomes for the wave speeds to the co-workers in this project and convinced them that these models have the capacity to replicate the diffusive waves seen in practice.

6.7 Further Work

There is plenty of scope for refinement in the model. Some extensions to the current work are listed:

- Further analysis of our work using different data sets as they become available is essential in verifying our model.
- We would also like to model and hence compare the case of releasing RHD as a biocide (where wide-scale baiting is used to spread RHD) and biocontrol (where the epidemic is allowed to spread naturally from small virus seeding points) ([21]). This would be done by using a distribution over position as the initial condition for the infectives rather than as a point source. It would be important to see how the initial profile affects the outcome for the wave speeds in the model.
- It would be important to re-examine the effect of the possible diffusion of the susceptible population. This would replace equation (6.3) by a diffusion equation similar to equation (6.2). The mathematical complication introduced would be considerable and would make it difficult to obtain an analytical expression for the wave speed. It is possible for some diseases that the diffusion coefficient in the susceptible cohort is different to that for the infective cohort. This needs to be considered.
- In Canterbury it was found that most of the survivors with immunity were females ([21]). It would be interesting to try and incorporate gender structure in our model to find out why this is the case. This would mean the model would have gender classes for both susceptible and infective cohorts adding two further diffusion–advection equations. The contact between these classes would need to be initially examined.
- We need to consider the reality of nonconstant parameter values reflecting different physical environments and climatic conditions.

All these refinements will lead to the same kind of analysis and outcomes, albeit complicated by the increased detail of the new models.

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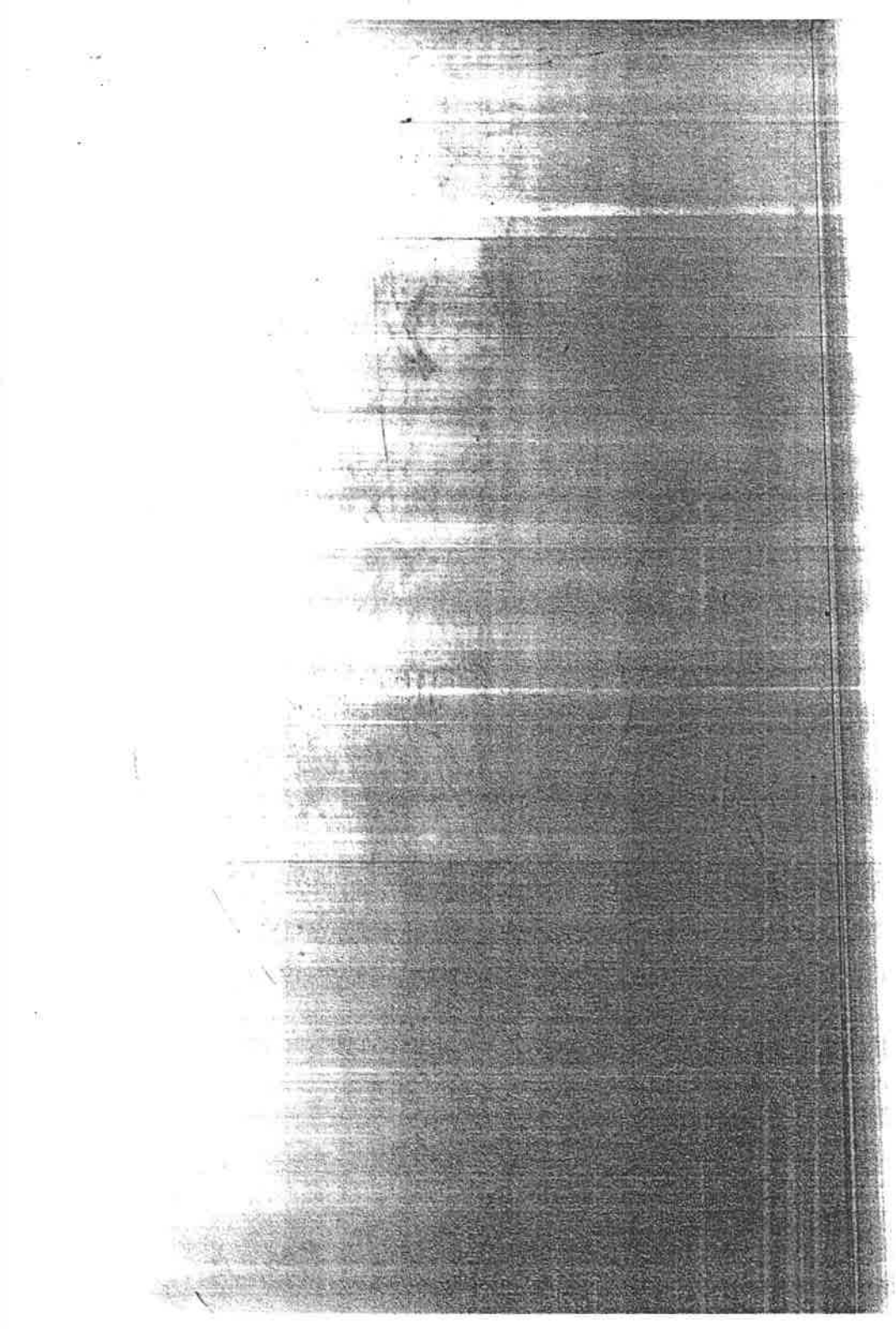

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