

coviruses Mutualism, parasitism and competition in the evolution of

Sean Nee

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Mutualism, parasitism and competition
 Mutualism, parasitism and competition in the evolution of competition
in the evolution of coviruses

Sean Nee

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 $\langle \text{sean}.\text{nee}(\mathcal{Q}\text{ed.ac.uk})$
Coviruses are viruses with the property that their genetic information is divided up among two or more
different viral particles. I model the evolution of coviruses using information on both Coviruses are viruses with the property that their genetic information is divided up among two or more different viral particles. I model the evolution of coviruses using information on both viral virulence and the interac Coviruses are viruses with the property that their genetic information is divided up among two or more different viral particles. I model the evolution of coviruses using information on both viral virulence and the interac different viral particles. I model the evolution of coviruses using information on both viral virulence and
the interactions between viruses and molecules that parasitize them: satellite viruses, satellite RNAs and
defecti the interactions between viruses and molecules that parasitize them: satellite viruses, satellite RNAs and defective interfering viruses. The model ultimately, and inevitably, contains within it single-species dynamics as defective interfering viruses. The model ultimately, and inevitably, contains within it single-species
dynamics as well as mutualistic, parasitic, cooperative and competitive relationships. The model shows
that coexistence dynamics as well as mutualistic, parasitic, cooperative and competitive relationships. The model shows
that coexistence between coviruses and the self-sufficient viruses that spawned them is unlikely, in the
sense that the that coexistence between coviruses and the self-sufficient viruses that spawned them is unlikely, in the sense that the quantitative conditions for coexistence are not easy to satisfy. I also describe an abrupt transition sense that the quantitative conditions for coexistence are not easy to satisfy. I transition from mutualistic two-species to single-species dynamics, showing questions such as 'Is a lichen one species or two?' can be given transition from mutualistic two-species to single-species dynamics, showing a new sense in which
questions such as 'Is a lichen one species or two?' can be given a definite answer.
Keywords: coviruses; metapopulation; mu

1. INTRODUCTION

strange parasites of plants. In a covirus, no single virus A number of plant viruses are 'coviruses', which are very
strange parasites of plants. In a covirus, no single virus
particle contains all the genetic information required for
a complete cycle of infection: instead, the ge strange parasites of plants. In a covirus, no single virus
particle contains all the genetic information required for
a complete cycle of infection: instead, the genome is split
into two or more senarately encansidated nac particle contains all the genetic information required for
a complete cycle of infection: instead, the genome is split
into two or more separately encapsidated, packaged
components (Fraenkel-Conrat & Wagner, 1977; Van a complete cycle of infection: instead, the genome is split
into two or more separately encapsidated, packaged
components (Fraenkel-Conrat & Wagner 1977; Van
Regenmortel & Fraenkel-Conrat 1986) They are all RNA into two or more separately encapsidated, packaged numponents (Fraenkel-Conrat & Wagner 1977; Van
Regenmortel & Fraenkel-Conrat 1986). They are all RNA by
invess but so are most plant viruses With one interesting components (Fraenkel-Conrat & Wagner 1977; Van forward—shorter molecules may be replicated faster-
Regenmortel & Fraenkel-Conrat 1986). They are all RNA but more interesting trade-offs have been found (for
viruses, but so Regenmortel & Fraenkel-Conrat 1986). They are all RNA
viruses, but so are most plant viruses. With one interesting
exception, discussed below, there are no known animal
coviruses coviruses. Example, the same of the care in shown animal
For example, tobacco rattle virus (TRV), which infects
For example, tobacco rattle virus (TRV), which infects

particles, a long and a short one (for a picture see http:// many species in addition to tobacco, consists of two
particles, a long and a short one (for a picture see http://
helios.bto.ed.ac.uk/icapb/research/evolecol/evolecol.html).
The long particle contains the so-called RNA1 th helios. bto.ed.ac.uk/icapb/research/evolecol/evolecol.html).
The long particle contains the so-called RNA1 that
encodes the replicase, while the short particle contains
RNA2 encoding the soat protein. The long particle can The long particle contains the so-called RNA1 that evident here: Are the encodes the replicase, while the short particle contains or mutual parasites?
RNA2, encoding the coat protein. The long particle can We do not need e encodes the replicase, while the short particle contains RNA1 is trapped in the plant in the absence of coat protein. A full cycle of infection ultimately requires infec-RNA1 is trapped in the plant in the absence of coat
protein. A full cycle of infection ultimately requires infec-
tion by both particles. In addition to the tobraviruses (e.g.
TRV) the comoviruses and penouiruses also have protein. A full cycle of infection ultimately requires infection by both particles. In addition to the tobraviruses (e.g. TRV), the comoviruses and nepoviruses also have this biology. On the other hand other coviruses such tion by both particles. In addition to the tobraviruses (e.g. TRV), the comoviruses and nepoviruses also have this biology. On the other hand, other coviruses, such as dianthoviruses, require all components in order to est TRV), the comoviruses and nepoviruses also have this biology. On the other hand, other coviruses, such as dianthoviruses, require all components in order to estabbiology. On the other hand, other coviruses, such as dianthoviruses, require all components in order to establish infection of the plant (Mayo *et al.* 1999). We will refer repeatedly to the biology of TRV dianthoviruses, require all compon
lish infection of the plant (Mayo et
repeatedly to the biology of TRV.
Nee & Maynard Smith (1990) h infection of the plant (Mayo *et al.* 1999). We will refer
beatedly to the biology of TRV.
Nee & Maynard Smith (1990) suggested that this
ange situation arose as a result of trade-offs between

repeatedly to the biology of TRV.
Nee & Maynard Smith (1990) suggested that this
strange situation arose as a result of trade-offs between
self.sufficiency—having a complete genome—and repli-Nee & Maynard Smith (1990) suggested that this
strange situation arose as a result of trade-offs between
self-sufficiency—having a complete genome—and repli-
cative advantages accruing to shorter genomes when their strange situation arose as a result of trade-offs between
self-sufficiency—having a complete genome—and repli-
cative advantages accruing to shorter genomes when their
deficiencies are complemented by other virus genomes i the plant. The phenomena of defective interfering (DI) deficiencies are complemented by other virus genomes in
the plant. The phenomena of defective interfering (DI)
viruses (Graves *et al.* 1996; White & Morris 1999), which
are shorter parasites of the viruses from which they the plant. The phenomena of defective interfering (DI) viruses (Graves *et al.* 1996; White & Morris 1999), which are shorter parasites of the viruses from which they are

A number of plant viruses are 'coviruses', which are very related to the host viruses they parasitize, provide much
strange parasites of plants. In a covirus, no single virus evidence for this trade-off (a satellite virus derived, and satellite RNAs and viruses (Garcia-Arenal derived, and satellite RNAs and viruses (Garcia-Arenal & Palukaitis 1999; Scholthof *et al.* 1999), which are underived, and satellite RNAs and viruses (Garcia-Arenal & Palukaitis 1999; Scholthof *et al.* 1999), which are unrelated to the host viruses they parasitize, provide much evidence for this trade-off (a satellite virus encod & Palukaitis 1999; Scholthof *et al.* 1999), which are un-
related to the host viruses they parasitize, provide much
evidence for this trade-off (a satellite virus encodes its
own coat protein whereas a satellite RNA does related to the host viruses they parasitize, provide much evidence for this trade-off (a satellite virus encodes its own coat protein whereas a satellite RNA does not). DI evidence for this trade-off (a satellite virus encodes its
own coat protein whereas a satellite RNA does not). DI
viruses and satellites are effectively viruses of viruses. The
molecular biology of the trade-offs can be st own coat protein whereas a satellite RNA does not). DI
viruses and satellites are effectively viruses of viruses. The
molecular biology of the trade-offs can be straight-
forward—shorter molecules may be replicated faster viruses and satellites are effectively viruses of viruses. The
molecular biology of the trade-offs can be straight-
forward—shorter molecules may be replicated faster—
but more interesting trade-offs have been found (for a molecular biology of the trade-offs can be straight-
forward—shorter molecules may be replicated faster—
but more interesting trade-offs have been found (for a
review see Nee & Maynard Smith 1990) review, see Nee & Maynard Smith 1990). t more interesting trade-offs have been found (for a
view, see Nee & Maynard Smith 1990).
Figure 1 illustrates the scenario for the evolution of
viruses considered by Nee & Maynard Smith (1990) in

For example, tobacco rattle virus (TRV), which infects which mutually complementing defective molecules many species in addition to tobacco, consists of two outcompete and possibly eliminate a complete virus. The coviruses considered by Nee & Maynard Smith (1990), in Figure 1 illustrates the scenario for the evolution of coviruses considered by Nee & Maynard Smith (1990) , in which mutually complementing defective molecules outcompete and possibly eliminate a complete virus. The coviruses considered by Nee & Maynard Smith (1990), in
which mutually complementing defective molecules
outcompete and possibly eliminate a complete virus. The
wery fuzzy distinction between mutualism and parasitism which mutually complementing defective molecules
outcompete and possibly eliminate a complete virus. The
very fuzzy distinction between mutualism and parasitism
(Maynard Smith & Szathmáry 1995; Beson et al. 1996) is outcompete and possibly eliminate a complete virus. The
very fuzzy distinction between mutualism and parasitism
(Maynard Smith & Szathmáry 1995; Begon *et al.* 1996) is
evident here: Are the partners in the covirus mutuali very fuzzy distinction between mutualism and parasitism
(Maynard Smith & Szathmáry 1995; Begon *et al.* 1996) is
evident here: Are the partners in the covirus mutualists
or mutual parasites? (Maynard Smith & Sz.
evident here: Are the $\frac{1}{100}$
or mutual parasites? evident here: Are the partners in the covirus mutualists
or mutual parasites?
We do not need to commit ourselves to whether the

self-sufficiency—having a complete genome—and repli-
cative advantages accruing to shorter genomes when their
deficiencies are complemented by other virus genomes in genome is encapsidated in luteovirus coat protein or mutual parasites?
We do not need to commit ourselves to whether the
incomplete but complementing viruses arose from
different deletions of the same complete virus (the We do not need to commit ourselves to whether the
incomplete but complementing viruses arose from
different deletions of the same complete virus (the
defective interfering virus route) or two different ones incomplete but complementing viruses arose from
different deletions of the same complete virus (the
defective interfering virus route) or two different ones
(the satellite virus or RNA route) Pea enation mosaic different deletions of the same complete virus (the defective interfering virus route) or two different ones (the satellite virus or RNA route). Pea enation mosaic virus (PEMV) is an example of the latter; it is a twodefective interfering virus route) or two different ones
(the satellite virus or RNA route). Pea enation mosaic
virus (PEMV) is an example of the latter: it is a two-
component virus whose components are clearly derived (the satellite virus or RNA route). Pea enation mosaic
virus (PEMV) is an example of the latter: it is a two-
component virus whose components are clearly derived
from unrelated viruses and each encodes its own replicase virus (PEMV) is an example of the latter: it is a two-component virus whose components are clearly derived from unrelated viruses and each encodes its own replicase component virus whose components are clearly derived
from unrelated viruses and each encodes its own replicase
(de Zoeten *et al.* 1995). Movement within and between
plants requires both components (Falk *et al.* 1999). On from unrelated viruses and each encodes its own replicase
(de Zoeten *et al.* 1995). Movement within and between
plants requires both components (Falk *et al.* 1999). One of
the components has a luteovirus origin, which is (de Zoeten *et al.* 1995). Movement within and between
plants requires both components (Falk *et al.* 1999). One of
the components has a luteovirus origin, which is inter-
esting as many members of the luteovirus group ar plants requires both components (Falk $et al.$ 1999). One of the components has a luteovirus origin, which is interesting as many members of the luteovirus group are the components has a luteovirus origin, which is interesting as many members of the luteovirus group are
known to be regularly 'helpful' to other viruses in nature,
rendering, them, applied, transmissable, when the alien esting as many members of the luteovirus group are
known to be regularly 'helpful' to other viruses in nature,
rendering them aphid transmissable when the alien
genome is encapsidated in luteovirus coat protein known to be regularly 'helpful' to other viruses in nature,
rendering them aphid transmissable when the alien
genome is encapsidated in luteovirus coat protein
(Falk et al. 1999). PEMV also illustrates the ontological rendering them aphid transmissable when the alien
genome is encapsidated in luteovirus coat protein
(Falk *et al.* 1999). PEMV also illustrates the ontological
conundrum coviruses pose us: it has recently been reclasgenome is encapsidated in luteovirus coat protein (Falk *et al.* 1999). PEMV also illustrates the ontological conundrum coviruses pose us: it has recently been reclassified by viral taxonomists as being two distinct viruse (Falk *et al.* 1999). PEMV also illustrates the ontological conundrum coviruses pose us: it has recently been reclassified by viral taxonomists as being two distinct viruses

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Figure 1. A simple system consisting of a complete viral Figure 1. A simple system consisting of a complete viral
genome that encodes a replicase and a coat p rotein and
two deletion mutants that encode one or the other. A solid Figure 1. A simple system consisting of a complete viral
genome that encodes a replicase and a coat protein and
two deletion mutants that encode one or the other. A solid
arrow indicates that the molecule both encodes and genome that encodes a replicase and a coat protein and
two deletion mutants that encode one or the other. A solid
arrow indicates that the molecule both encodes and uses the
indicated protein. A broken arrow indicates that two deletion mutants that encode one or the other. A solid
arrow indicates that the molecule both encodes and uses the
indicated protein. A broken arrow indicates that the molecule
inst uses the protein. The deletion mutan arrow indicates that the molecule both encodes and uses the
indicated protein. A broken arrow indicates that the molecule
just uses the protein. The deletion mutants can have their indicated protein. A broken arrow indicates that the molecule
just uses the protein. The deletion mutants can have their
deficiencies complemented by either the complete molecule or
each other and have a fitness advantage just uses the protein. The deletion mutants can have their
deficiencies complemented by either the complete molecule or
each other and have a fitness advantage over the complete
molecule when complemented | each other and have a fitness advantage over the complete
| molecule when complemented.

that are mutually dependent (M. Mayo, personal communication). at are mutually dependent (M. Mayo, personal
mmunication).
DI animal viruses are ubiquitous, at least in the labora-

DI animal viruses are ubiquitous, at least in the laboratory. An example of the DI route to covirus evolution is provided by simian virus 40 (SV40). Mutually completory. An example of the DI route to covirus evolution is
provided by simian virus 40 (SV40). Mutually comple-
menting DI viruses of SV40 were observed to eliminate
completely the wild-type virus in one study (O'Neill *et* provided by simian virus 40 (SV40). Mutually complementing DI viruses of SV40 were observed to eliminate completely the wild-type virus in one study (O'Neill *et al.* 1982), i.e. the evolution of a covirus by this route h completely the wild-type virus in one study (O'Neill *et al.* 1982), i.e. the evolution of a covirus by this route has actually been observed in an *in vitro* study of an animal virus.

ally been observed in an *in vitro* study of an animal virus.
However, DI viruses are not as widely observed in
plant as in animal viruses. And possibly the only study to
look for them in nature (tomato and aubergine plant However, DI viruses are not as widely observed in
plant as in animal viruses. And possibly the only study to
look for them in nature (tomato and aubergine plants in
Spanish greenhouses) failed to find any although the plant as in animal viruses. And possibly the only study to look for them in nature (tomato and aubergine plants in Spanish greenhouses) failed to find any, although the virus in question, tomato bushy stunt virus (TBSV) look for them in nature (tomato and aubergine plants in
Spanish greenhouses) failed to find any, although the
virus in question, tomato bushy stunt virus (TBSV),
generates them readily in the laboratory (Celix et al. Spanish greenhouses) failed to find any, although the
virus in question, tomato bushy stunt virus (TBSV),
generates them readily in the laboratory (Celix *et al.*
1997) On the other hand, the handful of studies looking virus in question, tomato bushy stunt virus (TBSV), generates them readily in the laboratory (Celix *et al.* 1997). On the other hand, the handful of studies looking generates them readily in the laboratory (Celix *et al.* 1997). On the other hand, the handful of studies looking for satellite RNAs of plant viruses in nature have all found them (Celix *et al.* 1997: Grieco *et al.* 199 1997). On the other hand, the handful of studies looking
for satellite RNAs of plant viruses in nature have all
found them (Celix *et al.* 1997; Grieco *et al.* 1997; Aranda
et al. 1997). Finally, the accumulating natura for satellite RNAs of plant viruses in nature have all
found them (Celix *et al.* 1997; Grieco *et al.* 1997; Aranda
et al. 1997). Finally, the accumulating natural history
from studies of large satellite RNAs reveals a found them (Celix *et al.* 1997; Grieco *et al.* 1997; Aranda *et al.* 1997). Finally, the accumulating natural history \rightarrow from studies of large satellite RNAs reveals a spectrum of relationships from obvious parasitism *et al.* 1997). Finally, the accumulating natural history
from studies of large satellite RNAs reveals a spectrum of
relationships from obvious parasitism to obvious mutu-
alism (Mayo *et al.* 1999). For all these reasons from studies of large satellite RNAs reveals a spectrum of partitionships from obvious parasitism to obvious mutualism (Mayo *et al.* 1999). For all these reasons, I am lis inclined to think that coviruses evolved via the in alism (Mayo *et al.* 1999). For all these reasons, I am inclined to think that coviruses evolved via the satellite alism (Mayo *et al.* 1999). For all these reasons, I am inclined to think that coviruses evolved via the satellite rather than the DI route: however, the actual modelling is simplified with no great violence done to the c inclined to think that coviruses evolved via the satellite
rather than the DI route: however, the actual modelling
is simplified, with no great violence done to the conclu-
sions by assuming a DI route i.e., one complete rather than the DI route: however, the actual modelling
is simplified, with no great violence done to the conclu-
sions, by assuming a DI route, i.e. one complete
progenitor virus is simplified, with
sions, by assumi
progenitor virus.
The quantitative **Phil. The Society Controllary and Society and Society and Service of Berlin Service Digital two deletion mutants that encodering interval in the pole indicated protein. The deletionizate protein. The deletionizate protein**

Im the quantitative model of the evolution of coviruses
The quantitative model of the evolution of coviruses
secussed in Nee & Maynard Smith (1990) is neculiar progenitor virus.
The quantitative model of the evolution of coviruses
discussed in Nee & Maynard Smith (1990) is peculiar
and seems to bave in mind some sort of well-mixed cell The quantitative model of the evolution of coviruses
discussed in Nee & Maynard Smith (1990) is peculiar
and seems to have in mind some sort of well-mixed cell
culture as the environment in which evolution is occurdiscussed in Nee & Maynard Smith (1990) is peculiar
and seems to have in mind some sort of well-mixed cell
culture as the environment in which evolution is occur-
ring. In particular, it is completely lacking any epiu culture as the environment in which evolution is occurdemiology, which is a curious feature for a model of virus evolution. miology, which is a curious feature for a model of virus
olution.
I will remedy this omission in this paper with a new
odel of covirus evolution I will begin with a description

evolution.
I will remedy this omission in this paper with a new
model of covirus evolution. I will begin with a description
of the modelling framework—metapopulation theory— I will remedy this omission in this paper with a new
model of covirus evolution. I will begin with a description (19)
of the modelling framework—metapopulation theory— thre

in the simple context of the population dynamics of a
single species I will then introduce a model of two mutuin the simple context of the population dynamics of a
single species. I will then introduce a model of two mutu-
alists studied elsewhere (Nee et al. 1997) which will be in the simple context of the population dynamics of a
single species. I will then introduce a model of two mutu-
alists studied elsewhere (Nee *et al.* 1997) which will be
modified to make it more meaningful for coviruses. single species. I will then introduce a model of two mutualists studied elsewhere (Nee *et al.* 1997) which will be modified to make it more meaningful for coviruses. Then, the qualitative behaviour of these two models as alists studied elsewhere (Nee *et al.* 1997) which will be modified to make it more meaningful for coviruses. Then, the qualitative behaviour of these two models as a varying parameter will be used to address the question modified to make it more meaningful for coviruses. Then, the qualitative behaviour of these two models as a varying parameter will be used to address the question: As a mutualistic association becomes more intimate, is the qualitative behaviour of these two models as a there a clear transition from a two-species system to a As a mutualistic association becomes more intimate, is
there a clear transition from a two-species system to a
single-species system? The answer is yes. This analysis
will provide us with some useful results, as well as us there a clear transition from a two-species system to a single-species system? The answer is yes. This analysis will provide us with some useful results, as well as useful special cases to facilitate understanding of the f single-species system? The answer is yes. This analysis
will provide us with some useful results, as well as useful
special cases, to facilitate understanding of the full
covirus evolution model Theoringinal question I wil will provide us with some useful results, as well as useful special cases, to facilitate understanding of the full covirus evolution model. The principal question I will pose the model is this: Can coviruses readily coexis special cases, to facilitate understanding of the full covirus evolution model. The principal question I will pose
the model is this: Can coviruses readily coexist with the
complete progenitor viruses from which they are derived?
The answer is no which is consistent with the f the model is this: Can coviruses readily coexist with the complete progenitor viruses from which they are derived?
The answer is no, which is consistent with the fact that such coexistence has not been observed in nature complete progenitor viruses from which they are d
The answer is no, which is consistent with the fa
such coexistence has not been observed in nature. **2. METAPOPULATION MODELLING FRAMEWORK**

Our modelling framework is that of Levins' metapopu-2. METAPOPULATION MODELLING FRAMEWORK
Our modelling framework is that of Levins' metapopulation model (Hanski & Gilpin 1997; Hanski 1999). The
Levins model is a useful abstraction that allows many Our modelling framework is that of Levins' metapopulation model (Hanski & Gilpin 1997; Hanski 1999). The Levins model is a useful abstraction that allows many systems that are biologically very dimarate to be lation model (Hanski & Gilpin 1997; Hanski 1999). The
Levins model is a useful abstraction that allows many
systems that are biologically very disparate to be
described in a common framework. The model is as Levins model is a useful abstraction that allows many
systems that are biologically very disparate to be
described in a common framework. The model is as
follows where we use inverted commas to emphasize that systems that are biologically very disparate to be
described in a common framework. The model is as
follows, where we use inverted commas to emphasize that
the interpretation of the words can be very broad We described in a common framework. The model is as
follows, where we use inverted commas to emphasize that
the interpretation of the words can be very broad. We
imagine patches' of suitable habitat for a species distribfollows, where we use inverted commas to emphasize that
the interpretation of the words can be very broad. We
imagine 'patches' of suitable habitat for a species distribthe interpretation of the words can be very broad. We
imagine patches' of suitable habitat for a species distributed in a 'landscape': empty patches may be 'colonized' by
a species and 'local populations' on occupied patch imagine `patches' of suitable habitat for a species distributed in a 'landscape': empty patches may be 'colonized' by
a species and 'local populations' on occupied patches may 're extinct', leaving an empty patch or in som uted in a 'landscape': empty patches may be 'colonized' by
a species and 'local populations' on occupied patches may
'go extinct', leaving an empty patch or, in some interpreta-
tions, the patch itself may disappear causin a species and 'local populations' on occupied patches may
'go extinct', leaving an empty patch or, in some interpreta-
tions, the patch itself may disappear causing local extinc-'go extinct', leaving an empty patch or, in some interpretations, the patch itself may disappear causing local extintion, with new, empty patches being created over time.
Let x be the fraction of empty patches and y t Im the patch itself may disappear causing local extinction, with new, empty patches being created over time.
Let x be the fraction of empty patches and y the frac-

(82), i.e. the evolution of a covirus by this route has actu-

ly been observed in an *in vitro* study of an animal virus.

However, DI viruses are not as widely observed in the metapopulation model, modified to allow var tion, with new, empty patches being created over time.
Let x be the fraction of empty patches and y the fraction of occupied patches. With colonization and extinction
rates denoted by ϵ and ϵ respectively, the b Let x be the fraction of empty patches and y the fraction of occupied patches. With colonization and extinction
rates denoted by ϵ and ϵ , respectively, the basic Levins
metapopulation model, modified to allow variation in the
total number of patches is rates denoted by c and e , redependicular model, modified
total number of patches, is

$$
\frac{dx}{dt} = -cxy + ey,
$$

\n
$$
\frac{dy}{dt} = cxy - ey,
$$

\n
$$
x + y = h,
$$
\n(1)

where *^h* is the fraction of habitat that is suitable for occuwhere *h* is the fraction of habitat that is suitable for occupation, with $h = 1$ in the 'pristine' world. (There is an important truth contained in Jeremy Jackson's (uppubwhere h is the fraction of habitat that is suitable for occu-
pation, with $h=1$ in the 'pristine' world. (There is an
important truth contained in Jeremy Jackson's (unpub-
lished) definition of 'pristine' as 'what the wo pation, with $h=1$ in the 'pristine' world. (There is an important truth contained in Jeremy Jackson's (unpub-
lished) definition of 'pristine' as 'what the world looked
like when you were growing up'). We can model the important truth contained in Jeremy Jackson's (unpub-
lished) definition of 'pristine' as 'what the world looked
like when you were growing up'.) We can model the lished) definition of 'pristine' as 'what the world looked
like when you were growing up'.) We can model the
effects of 'habitat destruction' (one topical interpretation)
by decreasing h like when you were growing up'.) We can model the effects of 'habitat destruction' (one topical interpretation) by decreasing *h*.
At a balance between colonization and extinction, equi-
librium patch occupancy v^* is gi

by decreasing *h*.
At a balance between colon
librium patch occupancy, y^* , i $*$: At a balance between colonization and extinction, equi-

$$
y^* = h - \frac{e}{c}.\tag{2}
$$

 $= h - \frac{3}{c}$. (2)
With declining *h*, the equilibrium frequency of occu-With declining *h*, the equilibrium frequency of occu-
pied patches declines continuously to zero. Extinction
occurs at a threshold level of destruction $h = e/c$; hence it With declining *h*, the equilibrium frequency of occu-
pied patches declines continuously to zero. Extinction
occurs at a threshold level of destruction, $h = e/c$: hence, it
is not necessary to destroy all suitable habitat pied patches declines continuously to zero. Extinction occurs at a threshold level of destruction, $h = e/c$: hence, it is not necessary to destroy all suitable habitat in order to occurs at a threshold level of destruction, $h = e/c$: hence, it
is not necessary to destroy all suitable habitat in order to
eradicate a species. In two influential papers, Lande
(1987–1988) seems to have been the first to is not necessary to destroy all suitable habitat in order to eradicate a species. In two influential papers, Lande (1987, 1988) seems to have been the first to note this threshold behaviour in the conservation context. In eradicate a species. In two influential papers, Lande
(1987, 1988) seems to have been the first to note this
threshold behaviour in the conservation context. In his

models, a 'patch' was a territory for a 'local population' of a single breeding female who would eventually die, or `go models, a 'patch' was a territory for a 'local population' of
a single breeding female who would eventually die, or 'go
extinct', leaving the territory open for 'colonization' by
another female a single breeding
extinct', leaving
another female.
The threshold tinct', leaving the territory open for 'colonization' by
other female.
The threshold result has long been known in epidemi-
pay which is simply another biological specification of

another female.
The threshold result has long been known in epidemi-
ology, which is simply another biological specification of
metapopulation theory: babitat patches are bost indivi-The threshold result has long been known in epidemi-
ology, which is simply another biological specification of
metapopulation theory: habitat patches are host indivi-
duals colonization is infection extinction is bost rec ology, which is simply another biological specification of
metapopulation theory: habitat patches are host indivi-
duals, colonization is infection, extinction is host recovery
or death, and vaccination programmes are want metapopulation theory: habitat patches are host individuals, colonization is infection, extinction is host recovery
or death, and vaccination programmes are wanton acts of duals, colonization is infection, extinction is host recovery
or death, and vaccination programmes are wanton acts of
habitat destruction, from the point of view of the infectious
disease organism (Nee et al. 1997). The th or death, and vaccination programmes are wanton acts of
habitat destruction, from the point of view of the infectious
disease organism (Nee *et al.* 1997). The threshold result in
epidemiology is the fact that you do not n habitat destruction, from the point of view of the infectious
disease organism (Nee *et al.* 1997). The threshold result in
epidemiology is the fact that you do not need 100% vacci-
nation coverage to eradicate a disea disease organism (Nee *et al.* 1997). The threshold result in epidemiology is the fact that you do not need 100% vaccination coverage to eradicate a disease (Anderson & May epidemiology is the fact that you do not need 100% vaccination coverage to eradicate a disease (Anderson & May
1991). Model (1) is a very simple epidemiological model in
which for example, the disease is fatal but the popu nation coverage to eradicate a disease (Anderson & May
1991). Model (1) is a very simple epidemiological model in
which, for example, the disease is fatal but the population
is regulated by other factors, so that births ex 1991). Model (1) is a very simple epidemiological model in which, for example, the disease is fatal but the population is regulated by other factors, so that births exactly balance deaths. Our subsequent models are genera which, for example, the disease is fatal but the population is regulated by other factors, so that births exactly balance is regulated by other factors, so that births exactly balance
deaths. Our subsequent models are generalizations of it. (I
note in passing that the threshold behaviour of model (1)
is found in a much broader ecological cont deaths. Our subsequent models are generalizations of it. (I
note in passing that the threshold behaviour of model (1)
is found in a much broader ecological context than that
discussed here: see Nee 1994) note in passing that the threshold behaviour of model (1) is found in a much broader ecological context than that discussed here; see Nee 1994.)

3. MUTUALISM MODEL

S. MUTUALISM MODEL
Nee *et al.* (1997) presented a simple metapopulation
model of mutualism. They envisaged a situation in which
the first partner can survive in a patch on its own but Nee *et al.* (1997) presented a simple metapopulation model of mutualism. They envisaged a situation in which the first partner can survive in a patch on its own but needs the second for dimersal to new patches whereas model of mutualism. They envisaged a situation in which
the first partner can survive in a patch on its own but
needs the second for dispersal to new patches, whereas
the second depends on the first for both survival and the first partner can survive in a patch on its own but
needs the second for dispersal to new patches, whereas
the second depends on the first for both survival and
reproduction. They had in mind the relationship between reeds the second for dispersal to new patches, whereas
the second depends on the first for both survival and
reproduction. They had in mind the relationship between
a plant species and its specialized seed disperser or pol the second depends on the first for both survival and
reproduction. They had in mind the relationship between
a plant species and its specialized seed disperser or pollin-
ator. They also suggested that the model may provi reproduction. They had in mind the relationship between
a plant species and its specialized seed disperser or pollin-
ator. They also suggested that the model may provide a
description of TRV dynamics as well. I will first a plant species and its specialized seed disperser or pollinator. They also suggested that the model may provide a description of TRV dynamics as well. I will first present this mutualism model and then extend it to correc ator. They also suggested that the model may provide a description of TRV dynamics as well. I will first present this mutualism model and then extend it to correct one of its obvious inadequacies as a description of TRV description of TRV dynamics as well. I will first present this mutualism model and then extend it to correct one of its obvious inadequacies as a description of TRV.

Patches may be either empty, occupied by the plant its obvious inadequacies as a description of TRV.
Patches may be either empty, occupied by the plant
only, or occupied by both plant and disperser. As in
equations (1) , x refers to the proportion of empty patches Patches may be either empty, occupied by the plant
only, or occupied by both plant and disperser. As in
equations (1), x refers to the proportion of empty patches,
 x is now the proportion of plant-only patches and z only, or occupied by both plant and disperser. As in equations (1), x refers to the proportion of empty patches, y is now the proportion of plant-only patches and z is the proportion of plant plus disperser patches. equations (1), x refers to the proportion of empty patches,
 y is now the proportion of plant-only patches and z is the
proportion of plant plus disperser patches. The original
model allowed for plant propagales and *y* is now the proportion of plant-only patches and z is the proportion of plant plus disperser patches. The original model allowed for plant propagules and disperser propagules to bave different colonization parameters proportion of plant plus disperser patches. The original
model allowed for plant propagules and disperser
propagules to have different colonization parameters and
for x and z patches to have different local extinction rate model allowed for plant propagules and disperser
propagules to have different colonization parameters and
for *y* and *z* patches to have different local extinction rates.
We do not need this extra complexity here and so a propagules to have different colonization parameters and
for y and z patches to have different local extinction rates.
We do not need this extra complexity here, and so assume
single-colonization, and extinction rates for y and z patches to have different local extinction rates.
We do not need this extra complexity here, and so assume
single colonization and extinction rates. Incorporating
the assumptions of the previous paragraph, We do not need this extra complexity here, and so assume
single colonization and extinction rates. Incorporating
the assumptions of the previous paragraph, the natural extension of equations (1) is

$$
\begin{cases}\n\frac{dx}{dt} = ey + ez - czx, \\
\frac{dy}{dt} = czx - ey - czy, \\
\frac{dz}{dt} = czy - ez, \\
\frac{dz}{dt} = \frac{1}{z - e} \\
\frac{dz}{dt} = \frac{1}{z - e} \\
\frac{dz}{dt} = \frac{1}{z - e} \\
\frac{1}{z - e} = \frac{1}{z}\n\end{cases}
$$
\n(3)

 $x + y + z = h$, which has the solution

$$
x^* = h - y^* - z^*,
$$

\n
$$
y^* = \frac{e}{c},
$$

\n
$$
z^* = \frac{1}{2} \left(h - \frac{2e}{c} \pm \sqrt{h^2 - \frac{4eh}{c}} \right).
$$
\n(4)

3. MUTUALISM MODEL vicinity of z^* . As h declines, these equilibria approach each other and their collision results in mutual annihilation—a mathematical catastrophe. The only equilibrium for h belo odel of mutualism. Figure 2. For any h above the extinction threshold there are
two equilibria. The upper one is stable and the lower one is
unstable: the arrows show the direction of motion of z in the Figure 2. For any *h* above the extinction threshold there are
two equilibria. The upper one is stable and the lower one is
unstable: the arrows show the direction of motion of *z* in the
vicinity of z^* . As *h* decline two equilibria. The upper one is stable and the lower one is ia. The upper one is stable and the lower one is
 c arrows show the direction of motion of z in the
 As h declines, these equilibria approach each
 e collision results in mutual approach each unstable: the arrows show the direction of motion of z in the
vicinity of z^* . As *h* declines, these equilibria approach each
other and their collision results in mutual annihilation—a
mathematical catastrophe. The onl other and their collision results in mutual annihilation—a
mathematical catastrophe. The only equilibrium for *h* below
the threshold is 0. This figure was constructed with $c/e = 5$.

Like equations (1), this also exhibits threshold be-
viour with extinction occurring at $h = 4e/c$. However Like equations (1), this also exhibits threshold behaviour, with extinction occurring at $h = 4e/c$. However, the hebaviour of the model in the vicinity of this Like equations (1), this also exhibits threshold behaviour, with extinction occurring at $h = 4e/c$. However, the behaviour of the model in the vicinity of this threshold is very different and is illustrated in figure 2 the behaviour of the model in the vicinity of this threshold is very different and is illustrated in figure 2. the behaviour of the model in the vicinity of this
threshold is very different and is illustrated in figure 2.
For *h* slightly larger than $4e/c$, there may be a substantial
level of patch occupancy with equilibrium patch threshold is very different and is illustrated in figure 2.
For h slightly larger than $4e/c$, there may be a substantial
level of patch occupancy, with equilibrium patch occu-
pancy plunging catastrophically to zero with For *h* slightly larger than $4e/c$, there may be a substantial
level of patch occupancy, with equilibrium patch occu-
pancy plunging catastrophically to zero with a tiny
increase in habitat destruction. As Nee *et al.* (1 level of patch occupancy, with equilibrium patch occupancy plunging catastrophically to zero with a tiny increase in habitat destruction. As Nee *et al.* (1997, p. 136) put it, 'a perfectly viable association of mutualists pancy plunging catastrophically to zero with a tiny increase in habitat destruction. As Nee *et al.* (1997, p. 136) put it, 'a perfectly viable association of mutualists living in great abundance across a large region can increase in habitat destruction. As Nee et al. $(1997, p. 136)$ put it, 'a perfectly viable association of mutualists living in
great abundance across a large region can be completely
destroyed by the construction of just one more shopping
mall' mall'. stroyed by the construction of just one more shopping
all'.
Model (3) can be viewed as a model of TRV dynamics
we consider v to refer to plants infected by RNA1 and z

Model (3) can be viewed as a model of TRV dynamics
if we consider *y* to refer to plants infected by RNA1 and *z* to refer to plants infected by both RNA1 and RNA2. A possible major inadequacy of model (3), if we want it as a to refer to plants infected by both RNA1 and RNA2. A
possible major inadequacy of model (3), if we want it as a
strategic description of TRV, is that it supposes (in terms
relevant to TRV infection of plants) that plants a possible major inadequacy of model (3), if we want it as a
strategic description of TRV, is that it supposes (in terms
relevant to TRV infection of plants) that plants are always
initially infected by only the long particl strategic description of TRV, is that it supposes (in terms
relevant to TRV infection of plants) that plants are always
initially infected by only the long particle, with possible
subsequent infection by the short particle relevant to TRV infection of plants) that plants are always
initially infected by only the long particle, with possible
subsequent infection by the short particle as well, whereas
we have no reason to think that a fully pr initially infected by only the long particle, with possible taneous infection does not also occur. In fact, perhaps we have no reason to think that a fully productive simultaneous infection does not also occur. In fact, perhaps simultaneous infection is the rule—it would certainly facilitate the evolution of the covirus in the first pla taneous infection does not also occur. In fact, perha
simultaneous infection is the rule—it would certain
facilitate the evolution of the covirus in the first place.
So, does the nematode vector of TRV typical multaneous infection is the rule—it would certainly
cilitate the evolution of the covirus in the first place.
So, does the nematode vector of TRV typically
numerit heth particles or is it a common occurrence that

facilitate the evolution of the covirus in the first place.
So, does the nematode vector of TRV typically
transmit *both* particles, or is it a common occurrence that So, does the nematode vector of TRV typically
transmit *both* particles, or is it a common occurrence that
plants are successfully infected with only the long particle
and perhaps subsequently infected with the short transmit *both* particles, or is it a common occurrence that
plants are successfully infected with only the long particle
and, perhaps, subsequently infected with the short
particle as well? We do not know the answer to th plants are successfully infected with only the long particle
and, perhaps, subsequently infected with the short
particle as well? We do not know the answer to this basic
natural history question, which simply reflects the and, perhaps, subsequently infected with the short particle as well? We do not know the answer to this basic natural history question, which simply reflects the fact particle as well? We do not know the answer to this basic
natural history question, which simply reflects the fact
that research into TRV is concerned entirely with its
molecular biology and not its natural history matural history question, which simply reflect
that research into TRV is concerned entirel
molecular biology and not its natural history.
In the field we do know that potatoes In the field, we do know that potatoes exhibiting
In the field, we do know that potatoes exhibiting
matoms of TRV infection are commonly infected only

molecular biology and not its natural history.
In the field, we do know that potatoes exhibiting
symptoms of TRV infection are commonly infected only
by RNA1 This suggests that infection by single particles In the field, we do know that potatoes exhibiting
symptoms of TRV infection are commonly infected only
by RNA1. This suggests that infection by single particles
does indeed occur in nature, but it could also be the case symptoms of TRV infection are commonly infected only
by RNA1. This suggests that infection by single particles
does indeed occur in nature, but it could also be the case

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Figure 3. Illustrating the qualitative behavior
solutions of model (3). See § 3 for discussion.

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that some potatoes are resistant to RNA2: we simply do
not know (S. Macfarlane, personal communication). In that some potatoes are resistant to RNA2: we simply do
not know (S. Macfarlane, personal communication). In
other crops like *Narrisus* (Amaryllidaceae) only infections that some potatoes are resistant to RNA2: we simply do
not know (S. Macfarlane, personal communication). In
other crops, like *Narcissus* (Amaryllidaceae), only infections
by both partners have been reported not know (S. Macfarlane, personal cother crops, like *Narcissus* (Amaryllidace
by both partners have been reported.
In any case, we will now general her crops, like *Narcissus* (Amaryllidaceae), only infections
both partners have been reported.
In any case, we will now generalize model (3) to
clude the possibility of transmission of both particles

by both partners have been reported.
In any case, we will now generalize model (3) to
include the possibility of transmission of both particles.
Of the new infections occurring in a time interval In any case, we will now generalize model (3) to include the possibility of transmission of both particles.
Of the new infections occurring in a time interval, a fraction f consist only of the long particle: include the possibility of transmission of both particles.
Of the new infections occurring in a time interval, a fraction f consist only of the long particle:

$$
\frac{dx}{dt} = ey + ez - czx,\n\frac{dy}{dt} = fczx - ey - czy,\n\frac{dz}{dt} = (1 - f)czx + czy - ez,\nx + y + z = h.
$$
\n(5)

For $f = 1$, we recover the previous model of two mutualists. For $f = 0$, so the partners are always transmitted region is defined by For $f = 1$, we recover the previous model of two mutualists. For $f = 0$, so the partners are always transmitted together, we recover the single-species metapopulation model (1) from the point of view of population dynamic alists. For $f = 0$, so the partners are always transmitted together, we recover the single-species metapopulation model (1): from the point of view of population dynamics, we are effectively dealing with a single entity. together, we recover the single-species metapopulation
model (1): from the point of view of population dynamics,
we are effectively dealing with a single entity. The ques-
tion we will pose of this model has an obvious ins model (1): from the point of view of population dynamics,
we are effectively dealing with a single entity. The question we will pose of this model has an obvious inspiration we are effectively dealing with a single entity. The question we will pose of this model has an obvious inspiration (Maynard Smith & Szathmáry 1995): Is there a clear houndary between these two situations and if so what is tion we will pose of this model has an obvious inspiration (Maynard Smith & Szathmáry 1995): Is there a clear
boundary between these two situations and, if so, what is
it? To put it another way is there an identifiable mi (Maynard Smith & Szathmáry 1995): Is there a clear
boundary between these two situations and, if so, what is
it? To put it another way, is there an identifiable minor
transition in population dynamics? boundary between these two situation
it? To put it another way, is there at
transition in population dynamics?
The answer is ves as we will now se To put it another way, is there an identifiable minor
unsition in population dynamics?
The answer is yes, as we will now see. The behaviour of
e single-pecies model in the face of babitat destruction

C transition in population dynamics?

The answer is yes, as we will now see. The behaviour of to zero with increasing habitat destruction.

the single-species model in the face of habitat destruction $\frac{\text{For } f > 1/2}{\text{For } f$ The answer is yes, as we will now see. The behaviour of
the single-species model in the face of habitat destruction
is qualitatively different from the behaviour of the mutu-
pliet system. In the former case, for increasin \sim the single-species model in the face of habitat destruction
is qualitatively different from the behaviour of the mutu-
alist system. In the former case, for increasing levels of
habitat destruction we have a continuous dec is qualitatively different from the behaviour of the mutualist system. In the former case, for increasing levels of habitat destruction we have a continuous decline in equili-
hrium population size to zero. In the latter c alist system. In the former case, for increasing levels of
habitat destruction we have a continuous decline in equili-
brium population size to zero. In the latter case, we have
a discontinuity: at the critical level of de habitat destruction we have a continuous decline in equili-
brium population size to zero. In the latter case, we have
a discontinuity: at the critical level of destruction, equili-
hrium population size jumps to zero from brium population size to zero. In the latter case, we have
a discontinuity: at the critical level of destruction, equili-
brium population size jumps to zero from a possibly large
distance We wish to identify a critical l a discontinuity: at the critical level of destruction, equilibrium population size jumps to zero from a possibly large distance. We wish to identify a critical level of f , f_{crit} , that brium population size jumps to zero from a possibly large
distance. We wish to identify a critical level of *f*, f_{crit} , that
has the following properties: for $f < f_{\text{crit}}$, the model
hehaves qualitatively like the sing distance. We wish to identify a critical level of f , f_{crit} , that has the following properties: for $f < f_{\text{crit}}$, the model behaves qualitatively like the single-species model and, for $f \setminus f$, the model behaves like has the following properties: for $f < f_{\text{crit}}$, the model
behaves qualitatively like the single-species model and,
for $f > f_{\text{crit}}$, the model behaves like the mutualist model. for $f > f_{\text{crit}}$, the model behaves like the mutualist model.
Phil. Trans. R. Soc. Lond. B (2000)

(If $f_{\text{crit}} = 1$, the behaviour of the mutualist model (3) is
structurally unstable) (If $f_{\text{crit}} = 1$, the behavior
structurally unstable.)
Model (5) has two po

Model (5) has two possible non-trivial equilibria in *^z*, given by the formulae

$$
z^* = \frac{1}{2} \left(h - \frac{2e}{c} \pm \sqrt{h^2 - \frac{4feh}{c}} \right). \tag{6}
$$

We will confine our attention to the larger, stable solution. As before, if the smaller solution is feasible, it is We will confine our attention to the lation. As before, if the smaller solution unstable. We will no longer consider y^* , as class of patches depends entirely on z^* . * a larger, stable solu-
on is feasible, it is
, as the fate of this tion. As before, if the smaller solution
unstable. We will no longer consider y^* ,
class of patches depends entirely on z^* .
To locate the transition in the qual class of patches depends entirely on z^* . Example. We will no longer consider y^* , as the fate of this uss of patches depends entirely on z^* .
To locate the transition in the qualitative dynamical haviour of the model we first note that as intuition

class of patches depends entirely on z^* .
To locate the transition in the qualitative dynamical
behaviour of the model, we first note that, as intuition
would suggest z^* is a monotonically increasing function To locate the tra
behaviour of the m
would suggest, z^* is
of h or equivalently would suggest, z^* is a monotonically increasing function Framerical in the qualitative dynamical
model, we first note that, as intuition
is a monotonically increasing function
by z^* declines with declining h. For the behaviour of the model, we first note that, as intuition
would suggest, z^* is a monotonically increasing function
of *h* or, equivalently, z^* declines with declining *h*. For the
model to exhibit mutualist dynamical would suggest, z^* is a monotonically increasing function
of h or, equivalently, z^* declines with declining h. For the
model to exhibit mutualist dynamical behaviour, it must
be the case that as h declines, there is of *h* or, equivalently, z^* declines with declining *h*. For the model to exhibit mutualist dynamical behaviour, it must
be the case that, as *h* declines, there is an *h*, $0 < h < 1$, for which (i) $h = 2e/c$ is positive; model to exhibit mutualist dynamical behaviour, it must
be the case that, as *h* declines, there is an *h*, $0 < h < 1$, for
which (i) $h-2e/c$ is positive; and (ii) the square-root
term in z^* goes from real to complex (wh be the case that, as *h* declines, there is an *h*, $0 < h < 1$, for which (i) $h-2e/c$ is positive; and (ii) the square-root term in z^* goes from real to complex (which happens when the two equilibria collide). Together, which (i) $h-2e/c$ is positive; and (ii) the square-root
term in z^* goes from real to complex (which happens
when the two equilibria collide). Together, these two
conditions vield term in z^* goes from
when the two equ
conditions yield

$$
\frac{2e}{c} < h < \frac{4fe}{c}.\tag{7}
$$

 $\frac{1}{c} < h < \frac{3}{c}$. (7)
This gives us $f = 1/2$ as the line dividing mutualistic
behaviour from single-species behaviour This gives us $f = 1/2$ as the line divider behaviour from single-species behaviour.
The story is not complete. In what regio is gives us $f = 1/2$ as the line dividing mutualistic
haviour from single-species behaviour.
The story is not complete. In what region of parameter
ace can the virus exist ('existence' meaning $z^* > 0$ when

behaviour from single-species behaviour.
The story is not complete. In what region of pa
space can the virus exist ('existence' meaning $z^* >$
 $h = 1$)? It can readily be seen from equation (6) f parameter
 $^* > 0$ when
6) that the The story is not complete. In what region of parameter space can the virus exist ('existence' meaning $z^* > 0$ when $h = 1$)? It can readily be seen from equation (6) that the region is given by space can the virus ex
 $h = 1$)? It can readily
region is given by

$$
f < 1 - \frac{e}{c}, \qquad \frac{c}{e} < 2,
$$

$$
f < \frac{c}{4e}, \qquad \frac{c}{e} > 2.
$$
 (8)

 $f < \frac{1}{4e}$, $\frac{1}{e} > 2$.
Finally, we also wish to note the region of parameter
space in which in a world with $h-1$ the virus can Finally, we also wish to note the region of parameter
space in which, in a world with $h = 1$, the virus can
increase when very rare. This requires that the lower space in which, in a world with $h = 1$, the virus can increase when very rare. This requires that the lower, space in which, in a world with $h=1$, the virus can
increase when very rare. This requires that the lower,
unstable equilibrium is unfeasible, i.e. it is negative. This
region is defined by increase when very ra
unstable equilibrium is
region is defined by

$$
f < 1 - \frac{e}{c}.\tag{9}
$$

Figure 3 presents a summary of this analysis.

As we can see, for $f < 1/2$, the qualitative behaviour of the system is entirely like that of the single-species model, As we can see, for $f < 1/2$, the qualitative behaviour of
the system is entirely like that of the single-species model,
regardless of the colonization rate: there is a single, stable
equilibrium population abundance that the system is entirely like that of the single-species model,
regardless of the colonization rate: there is a single, stable
equilibrium population abundance that declines smoothly
to zero with increasing habitat destructi regardless of the colonization rate: there is a
equilibrium population abundance that decli
to zero with increasing habitat destruction.
For $f > 1/2$, the situation is more complic equilibrium population abundance that declines smoothly
to zero with increasing habitat destruction.
For $f > 1/2$, the situation is more complicated. In this

to zero with increasing habitat destruction.
For $f > 1/2$, the situation is more complicated. In this
region, for $f > 1 - e/c$ we have unambiguous mutualists
whose qualitative behaviour is the same as in our original For $f > 1/2$, the situation is more complicated. In this region, for $f > 1 - e/c$ we have unambiguous mutualists whose qualitative behaviour is the same as in our original mutualism model. For $f < 1-e/c$ habitat destruction region, for $f > 1 - e/c$ we have unambiguous mutualists
whose qualitative behaviour is the same as in our original
mutualism model. For $f < 1-e/c$, habitat destruction
creates mutualistic dynamics out of single-species whose qualitative behaviour is the same as in our original
mutualism model. For $f < 1 - e/c$, habitat destruction
creates mutualistic dynamics out of single-species
dynamics In this region when $h-1$ the model is qualitamutualism model. For $f < 1-e/c$, habitat destruction
creates mutualistic dynamics out of single-species
dynamics. In this region, when $h = 1$, the model is qualita-
tively a single-species model with a single stable equilicreates mutualistic dynamics out of single-species dynamics. In this region, when $h = 1$, the model is qualitatively a single-species model, with a single, stable equilidynamics. In this region, when $h = 1$, the model is qualitatively a single-species model, with a single, stable equilibrium abundance. But as h declines, a new, lower, unstable equilibrium appears and with decreasing h tively a single-species model, with a single, stable equili-
brium abundance. But as h declines, a new, lower,
unstable equilibrium appears and, with decreasing h , rises
until it annihilates the stable equilibrium res brium abundance. But as h declines, a new, lower, unstable equilibrium appears and, with decreasing h , rises until it annihilates the stable equilibrium, resulting in a mathematical and real catastrophe for the popula unstable equilibrium appears and, with decreasing h , rises until it annihilates the stable equilibrium, resulting in a mathematical and real catastrophe for the population.

As *c*/*e* gets larger, the mutualist region is squeezed out by this 'induced' mutualist region. Perhaps a better way of As c/e gets larger, the mutualist region is squeezed out
by this 'induced' mutualist region. Perhaps a better way of
thinking about it is that for larger c/e , mutualistic
dynamics are masked i.e. the lower unstable equi by this 'induced' mutualist region. Perhaps a better way of
thinking about it is that for larger c/e , mutualistic vi
dynamics are masked, i.e. the lower, unstable equilibrium
is swamped out of existence by a high coloniz thinking about it is that for larger c/e , mutualistic dynamics are masked, i.e. the lower, unstable equilibrium is swamped out of existence by a high colonization rate in the pristine environment dynamics are masked, i.e. the lower, unstable equilibrium
is swamped out of existence by a high colonization rate in
the pristine environment.

In any case, the fact that the qualitative behaviour of the pristine environment.
In any case, the fact that the qualitative behaviour of
the model depends on both f and c/e is not surprising.
What is surprising is that the behaviour is independent of In any case, the fact that the qualitative behaviour of
the model depends on both f and c/e is not surprising.
What is surprising is that the behaviour is independent of
 c/e for $f < 1/2$ c/e for $f < 1/2$.

4. EVOLUTION OF COVIRUSES

We will model the population dynamics of a virus^ 4. **EVOLUTION OF COVIRUSES**
We will model the population dynamics of a virus-
covirus system in a metapopulation of plants by general-
izing model (5) where the word 'virus' is reserved for the We will model the population dynamics of a virus-
covirus system in a metapopulation of plants by general-
izing model (5), where the word 'virus' is reserved for the
complete autonomous virus We are interested in the covirus system in a metapopulation of plants by general-
izing model (5), where the word 'virus' is reserved for the
complete, autonomous virus. We are interested in the
circumstances under which the covirus completely ous izing model (5), where the word 'virus' is reserved for the complete, autonomous virus. We are interested in the circumstances under which the covirus completely ousts the virus from the population. As far as I am aware complete, autonomous virus. We are interested in the circumstances under which the covirus completely ousts the virus from the population. As far as I am aware, there is no example of virus-covirus coexistence: for the circumstances under which the covirus completely ousts
the virus from the population. As far as I am aware,
there is no example of virus-covirus coexistence: for the
theoretical framework presented here to be plausible it the virus from the population. As far as I am aware,
there is no example of virus-covirus coexistence: for the
theoretical framework presented here to be plausible it
must at the very least be consistent with this fact there is no example of virus–covirus coexistence: for
theoretical framework presented here to be plausi
must, at the very least, be consistent with this fact.
We will suppose that in a plant containing bot theoretical framework presented here to be plausible it
must, at the very least, be consistent with this fact.
We will suppose that, in a plant containing both the

must, at the very least, be consistent with this fact.
We will suppose that, in a plant containing both the
virus and replicating covirus (either a single component
of the covirus like the RNA1 of TRV or both) the virus We will suppose that, in a plant containing both the
virus and replicating covirus (either a single component
of the covirus, like the RNA1 of TRV, or both), the virus
concentration is reduced to such low levels that it is virus and replicating covirus (either a single component
of the covirus, like the RNA1 of TRV, or both), the virus
concentration is reduced to such low levels that it is not
transmissible This is a reasonable simplificatio of the covirus, like the RNA1 of TRV, or both), the virus
concentration is reduced to such low levels that it is not
transmissible. This is a reasonable simplification: DI viruses concentration is reduced to such low levels that it is not
transmissible. This is a reasonable simplification: DI viruses
by definition interfere with the production of virus, and
satellite RNAs can be so effective at redu transmissible. This is a reasonable simplification: DI viruses
by definition interfere with the production of virus, and
satellite RNAs can be so effective at reducing virus yield
that crop plants have been genetically eng by definition interfere with the production of virus, and
satellite RNAs can be so effective at reducing virus yield
that crop plants have been genetically engineered to
produce them themselves e.g. Harrison et al. (1987) satellite RNAs can be so effective at reducing virus yield
that crop plants have been genetically engineered to
produce them themselves, e.g. Harrison *et al.* (1987) that crop plants have been genetically engineered to produce them themselves, e.g. Harrison *et al.* (1987) (although this strategy is not without risk). A closely related and realistic assumption is that covirus can produce them themselves, e.g. Harrison *et al.* (1987) (although this strategy is not without risk). A closely related, and realistic, assumption is that covirus can infect plants infected with virus but not vice versa. (although this strategy is not without risk). A cl
related, and realistic, assumption is that covirus
infect plants infected with virus, but not vice versa.
We will be interested mainly in the quantitative related, and realistic, assumption is that covirus can
infect plants infected with virus, but not vice versa.
We will be interested mainly in the quantitative rela-

tionships between the infection rates (parameter ϵ) that determine the dynamical outcome. But what will we tionships between the infection rates (parameter c) that
determine the dynamical outcome. But what will we
assume about the virulence (parameter e) of the virus and
covirus? covirus? Figure about the virulence (parameter e) of the virus and
virus?
Remarkably little is understood about virulence in
neral beyond the generalization that disease symptoms

Remarkably little is understood about virulence in general beyond the generalization that disease symptoms Examination that is an
areal determined by properties of the host, the virus and
 ϵ (c)² general beyond the generalization that disease symptoms
are determined by properties of the host, the virus and
the interaction between the two—which is more an
admission of ignorance than anything else. There was are determined by properties of the host, the virus and
the interaction between the two—which is more an
admission of ignorance than anything else. There was
great bone that viroids would shed light on this area the interaction between the two—which is more an admission of ignorance than anything else. There was great hope that viroids would shed light on this area.
These plant viruses consist of a few hundred bases of admission of ignorance than anything else. There was
great hope that viroids would shed light on this area.
These plant viruses consist of a few hundred bases of
RNA that code for absolutely nothing at all and induce great hope that viroids would shed light on this area.
These plant viruses consist of a few hundred bases of
RNA that code for absolutely nothing at all and induce
the full spectrum of infections from asymptomatic to These plant viruses consist of a few hundred bases of RNA that code for absolutely nothing at all and induce
the full spectrum of infections from asymptomatic to
lethal (Matthews 1991: Singh et al. 1995). Hence, any RNA that code for absolutely nothing at all and induce
the full spectrum of infections from asymptomatic to
lethal (Matthews 1991; Singh *et al.* 1995). Hence, any
symptoms are being produced by bost proteins. In spite of the full spectrum of infections from asymptomatic to
lethal (Matthews 1991; Singh *et al.* 1995). Hence, any
symptoms are being produced by host proteins. In spite of
the simplicity of the system as well as its substantial lethal (Matthews 1991; Singh *et al.* 1995). Hence, any
symptoms are being produced by host proteins. In spite of
the simplicity of the system, as well as its substantial
economic importance, there has been very little pro symptoms are being produced by host proteins. In spite of
the simplicity of the system, as well as its substantial
economic importance, there has been very little progress
in understanding pathogenesis. The reason for this the simplicity of the system, as well as its substantial economic importance, there has been very little progress in understanding pathogenesis. The reason for this failure economic importance, there has been very little progress
in understanding pathogenesis. The reason for this failure
seems to be simply the difficulty of identifying which host
proteins are responding to the viroid sequence in understanding pathogenesis. The reason for this failure
seems to be simply the difficulty of identifying which host
proteins are responding to the viroid sequence (Matthews
1991) (A satellite of benatitie B benatitie de seems to be simply the difficulty of identifying which host
proteins are responding to the viroid sequence (Matthews
1991). (A satellite of hepatitis B, hepatitis delta virus, is
remarkably similar to plant viroids (Taylor proteins are responding to the viroid sequence (Matthews 1991). (A satellite of hepatitis B, hepatitis delta virus, is remarkably similar to plant viroids (Taylor 1999).)

Satellite virus and DI virus systems also provide us remarkably similar to plant viroids (Taylor 1999).)
Satellite virus and DI virus systems also provide us
with no useful generalizations. For example, different
strains of the satellite RNA CARNA 5 which parasitizes Satellite virus and DI virus systems also provide us
with no useful generalizations. For example, different
strains of the satellite RNA CARNA 5, which parasitizes
cucumber mosaic virus (CMV) can attenuate or exacerwith no useful generalizations. For example, different
strains of the satellite RNA CARNA 5, which parasitizes
cucumber mosaic virus (CMV), can attenuate or exacer-
hate disease symptoms. Furthermore, the same strain of strains of the satellite RNA CARNA 5, which parasitizes
cucumber mosaic virus (CMV), can attenuate or exacer-
bate disease symptoms. Furthermore, the same strain of
CARNA 5 will attenuate symptoms of CMV infection in cucumber mosaic virus (CMV), can attenuate or exacerbate disease symptoms. Furthermore, the same strain of CARNA 5 will attenuate symptoms of CMV infection in CARNA 5 will attenuate symptoms of CMV infection in
Phil. Trans. R. Soc. Lond. B (2000)

asitism and competition in the evolution of coviruses S. Nee 1611
tabasco pepper plants while inducing lethal necrosis in tomato (Matthews 1991). Similarly, although the first DI tabasco pepper plants while inducing lethal necrosis in
tomato (Matthews 1991). Similarly, although the first DI
virus found in plants, derived from TBSV, attenuates
disease symptoms (Hillman et al. 1987). DI turnin crink tomato (Matthews 1991). Similarly, although the first DI
virus found in plants, derived from TBSV, attenuates
disease symptoms (Hillman *et al.* 1987), DI turnip crinkle
viruses increase symptom severity (Li *et al.* 1989; virus found in plants, derived from TBSV, attenuates
disease symptoms (Hillman *et al.* 1987), DI turnip crinkle
viruses increase symptom severity (Li *et al.* 1989; Simon
1999) 1999). The sum of the severity (Li *et al.* 1989; Simon (99).
Lacking guidance one way or the other, we will assume at both virus and covirus have the same parameter ℓ

1999). Lacking guidance one way or the other, we will assume
that both virus and covirus have the same parameter e .
This is, in fact, a satisfactory simplification: inequalities
in e change the outcome of the model in that both virus and covirus have the same parameter e .
This is, in fact, a satisfactory simplification: inequalities
in e change the outcome of the model in an intuitive
fashion so we do not need the extra symbolic cl This is, in fact, a satisfactory simplification: inequali
in e change the outcome of the model in an intuit
fashion, so we do not need the extra symbolic clutter.
The variables x , y and z have the same meaning ϵ change the outcome of the model in an intuitive shion, so we do not need the extra symbolic clutter.
The variables x , y and z have the same meaning as fore We subscript the colonization parameters to iden-

fashion, so we do not need the extra symbolic clutter.
The variables x , y and z have the same meaning as before. We subscript the colonization parameters to iden-The variables x , y and z have the same meaning as
before. We subscript the colonization parameters to iden-
tify them as belonging to the virus, c_v , or covirus, c_c . The
variable w refers to the frequency of pl before. We subscript the colonization parameters to identify them as belonging to the virus, c_v , or covirus, c_c . The variable *w* refers to the frequency of plants infected by virus. The full model is tify them as belonging to
variable w refers to the
virus. The full model is

$$
\frac{dx}{dt} = ey + ez + ew - c_c zx - c_v wx,
$$
\n
$$
\frac{dy}{dt} = fc_c x + fc_c zw - ey - c_c zy,
$$
\n
$$
\frac{dz}{dt} = (1 - f)c_c zw + (1 - f)c_c zw + c_c zy - ez,
$$
\n
$$
\frac{dw}{dt} = c_v wx - c_c zw - ew.
$$
\n(10)

The full equilibrium solutions of this model will not be different discussed here. Instead we will consider two extreme
special cases The full equ
discussed here
special cases.

$f(a)$ $f = 0$

This case describes a situation in which both components of the covirus must simultaneously infect the plant This case describes a situation in which both components of the covirus must simultaneously infect the plant to establish infection. For the covirus to exist, it must be the case that nents of the co
to establish inf
the case that

$$
\frac{c_{\rm c}}{e} > 1,\tag{11}
$$

 $\frac{a_c}{e} > 1,$ (11)
and, if this is satisfied, for the virus to persist it must be
the case that ϵ and, if this is s
the case that

$$
\frac{\epsilon_{\rm v}}{e} > \left(\frac{\epsilon_{\rm c}}{e}\right)^2. \tag{12}
$$

 $\frac{1}{e} > \left(\frac{2}{e}\right)$. (12)
Notice that *c*/*e* corresponds to the *R*₀ of the epidemiol-
orist: it is the number of new infected plants produced by Notice that c/e corresponds to the R_0 of the epidemiologist: it is the number of new infected plants produced by
a single infected plant introduced into a healthy plant Notice that c/e corresponds to the R_0 of the epidemiologist: it is the number of new infected plants produced by a single infected plant introduced into a healthy plant population population. In a single infected plant introduced into a healthy plant population.
If both these conditions are satisfied, then there is an interior stable equilibrium

If both these conditions are satisfied, then there is an

interior stable equilibrium
\n
$$
(x^*, y^*, z^*, w^*) = \left(1 - z^* - w^*, 0, 1 - \frac{e}{c_c}, \frac{c_v e - c_c^2}{c_v c_c}\right).
$$
\n(13)

This case has at least two interesting features.

(i) It is identical to a model of two competing species
(i) It is identical to a model of two competing species
that coexist as metapopulations Coexistence is that case has a least two interesting leatures.
It is identical to a model of two competing species
that coexist as metapopulations. Coexistence is
achieved when the superior competitor—which can It is identical to a model of two competing species
that coexist as metapopulations. Coexistence is
achieved when the superior competitor—which can
exclude the inferior from a natch—has a lower that coexist as metapopulations. Coexistence is achieved when the superior competitor—which can exclude the inferior from a patch—has a lower colonization rate. So the inferior competitor survives achieved when the superior competitor—which can exclude the inferior from a patch—has a lower
colonization rate. So the inferior competitor survives
as a weedy, fugitive species. This model was first
studied by Hastings (1980) in the context of the colonization rate. So the inferior competitor survives
as a weedy, fugitive species. This model was first
studied by Hastings (1980) in the context of the
coexistence of coral mecies and later independently as a weedy, fugitive species. This model was first
studied by Hastings (1980) in the context of the
coexistence of coral species and later, independently,

BIOLOGICAL
SCIENCES

THE ROYAL

PHILOSOPHICAL
TRANSACTIONS

 $\mathbf S$

 $\frac{b^2 + 1}{2}$ by Nee & May (1992), who were interested in the effects of babitat destruction on the competitors by Nee & May (1992), who were interested in the effects of habitat destruction on the competitors. by Nee & May (1992), who were interested in the effects of habitat destruction on the competitors.
(They found that habitat destruction works to the advantage of inferior competitors). The same model effects of habitat destruction on the competitors.
(They found that habitat destruction works to the
advantage of inferior competitors.) The same model
reappeared with an epidemiological interpretation in (They found that habitat destruction works to the advantage of inferior competitors.) The same model
reappeared with an epidemiological interpretation in
a study of the evolution of virulence (Nowak & May advantage of inferior competitors.) The same model
reappeared with an epidemiological interpretation in
a study of the evolution of virulence (Nowak & May
1994). Here it appears as a model of mutualists reappeared with an epidemiological interpretation in
a study of the evolution of virulence (Nowak & May
1994). Here it appears as a model of mutualists
competing with a single species a study of the evolution of virulen
1994). Here it appears as a m
competing with a single species.
It is not going to be easy for the y competing with a single species.
It is not going to be easy for the virus to persist after

competing with a single species.
It is not going to be easy for the virus to persist after
the covirus has arisen. The condition in equation
(12) is demanding and requires that the transmission It is not going to be easy for the virus to persist after
the covirus has arisen. The condition in equation
(12) is demanding and requires that the transmission
of the covirus is very seriously impaired by its the covirus has arisen. The condition in equation (12) is demanding and requires that the transmission of the covirus is very seriously impaired by its divided nature. The fact that as far as I am aware (12) is demanding and requires that the transmission
of the covirus is very seriously impaired by its
divided nature. The fact that, as far as I am aware, of the covirus is very seriously impaired by its
divided nature. The fact that, as far as I am aware,
there is no example of virus-covirus coexistence
suggests that vector transmission is effective divided nature. The fact that, as far as I am
there is no example of virus–covirus coex
suggests that vector transmission is effective.

$f(b)$ $f = 1$

OCIETY (b) $f=1$
This is a version of the TRV scenario where we assume
that co-infection never occurs. The necessary condition
for the existence of the covirus which we have previously This is a version of the TRV scenario where we assume This is a version of the TRV scenario where we assume
that co-infection never occurs. The necessary condition
for the existence of the covirus, which we have previously
found to be $c/e > 4$ (forme 3) is more demanding than that co-infection never occurs. The necessary condition
for the existence of the covirus, which we have previously
found to be $c_c/e > 4$ (figure 3), is more demanding than in
the previous case. The reason for this is that for the existence of the covirus, which we have previously
found to be $c_c/e > 4$ (figure 3), is more demanding than in
the previous case. The reason for this is that newly
infected plants are not themselves infectious. But found to be $c_c/e > 4$ (figure 3), is more demanding than in
the previous case. The reason for this is that newly
infected plants are not, themselves, infectious. But, given
that the covirus can exist, the conditions for th the previous case. The reason for this is that newly
infected plants are not, themselves, infectious. But, given
that the covirus can exist, the conditions for the co-
existence of the virus are difficult to satisfy in thi infected plants are not, themselves, infectious. But, given
that the covirus can exist, the conditions for the co-
existence of the virus are difficult to satisfy in this case as
well existence of the virus are difficult to satisfy in this case as
well.
The relevant equilibrium solution of equations (10) is well.

$$
x^* = \frac{c_c + \sqrt{c_c^2 - 4c_c e}}{2c_v},
$$
\n(14*a*)

$$
y^* = \frac{1}{c_c},\tag{14b}
$$

$$
z^* = \frac{c_c + \sqrt{c_c^2 - 4c_c e - 2e}}{2c_c},
$$
\n(14c)

$$
w^* = \frac{c_v - \sqrt{c_v^2 - 4c_v^2 e/c_c} - c_c - \sqrt{c_c^2 - 4c_c e}}{2c_v}.
$$
 (14*d*)

From equation (14*d*), there is a critical value of c_v , c_v
low which $w^* = 0$ i.e. the virus cannot persist with From equation (14*d*), there is a critical value of c_v , $c_{v,\text{crit}}$, below which $w^* = 0$, i.e. the virus cannot persist with the covirus given by From equation (14
below which $w^* = 0$,
covirus, given by

$$
c_{v,\text{crit.}} = \frac{1}{2} (c_{\text{c}}^2 + c_{\text{c}} \sqrt{c_{\text{c}}^2 - 4c_{\text{c}}} - 2c_{\text{c}}).
$$
 (15)

In the previous special case, the virus needed a coloni-In the previous special case, the virus needed a colonization parameter that was the square of that of the square in order to persit. That is still the case here at In the previous special case, the virus needed a colonization parameter that was the square of that of the covirus in order to persist. That is still the case here, at least approximately for large ϵ . For smaller value zation parameter that was the square of that of the covirus in order to persist. That is still the case here, at least approximately for large c_c . For smaller values of c_c , the threshold increases with increasing c_c **S** least approximately for large c_c . For smaller values of c_c , covirus in order to persist. That is still the case here, at
least approximately for large c_c . For smaller values of c_c ,
the threshold increases with increasing c_c by ever larger
factors ultimately converging on a f

least approximately for large c_c . For smaller values of c_c ,
the threshold increases with increasing c_c by ever larger
factors, ultimately converging on a factor of c_c : for $c_c = 5$,
6. 7 the threshold is greater th values of
ever larged:
: for $c_c =$
 $d \Delta x$ the threshold increases with increasing c_c by ever large
factors, ultimately converging on a factor of c_c : for $c_c = \frac{5}{6}$, 7 the threshold is greater than $2 \times$, $3 \times$ and $4 \times c_c$. So $\begin{aligned} \text{ larger} \\ \text{.} = 5, \\ \text{. So,} \\ \text{trial} \end{aligned}$ factors, ultimately converging on a factor of c_c : for $c_c = 5$, 6, 7 the threshold is greater than $2 \times$, $3 \times$ and $4 \times c_c$. So, as before, the covirus needs to have a very substantial transmission disadvantage if it is 6, 7 the threshold is greater than $2 \times$, $3 \times$ and $4 \times c_c$. So, as before, the covirus needs to have a very substantial transmission disadvantage if it is not to supplant the virus entirely. Taken together, these two spe as before, the covirus needs to have a very substantial
transmission disadvantage if it is not to supplant the virus
entirely. Taken together, these two special cases suggest
that it may be true for all f that the virus ne transmission disadvantage if it is not to supplant the virus
entirely. Taken together, these two special cases suggest
that it may be true for all *f* that the virus needs a
colonization parameter that is the square of tha entirely. Taken together, these two special cases suggest
that it may be true for all f that the virus needs a
colonization parameter that is the square of that of the
covirus in order for persistence. This is the case, that it may be true for all f that the virus needs a colonization parameter that is the square of that of the covirus in order for persistence. This is the case, as will be shown when the full behaviour of the model is colonization parameter that is the square of that of the covirus in order for persistence. This is the case, as will
be shown when the full behaviour of the model is
described elembere (S. Nee unpublished) covirus in order for persistence. This is the
be shown when the full behaviour of t
described elsewhere (S. Nee, unpublished).

5. CONCLUSIONS

5. CONCLUSIONS
Models as simple as equations (10) are often called
enchmark' or 'strategic' models the adjective action as a \bullet Models as simple as equations (10) are often called

'benchmark' or 'strategic' models, the adjective acting as a

figules to protect the model's composer from glib accusa-Models as simple as equations (10) are often called

"benchmark' or 'strategic' models, the adjective acting as a

fig leaf to protect the model's composer from glib accusa-

tions of oversimplification. Minimalist it m 'benchmark' or 'strategic' models, the adjective acting as a
fig leaf to protect the model's composer from glib accusa-
tions of oversimplification. Minimalist it may be as a
model of covirus evolution—and I do not have th fig leaf to protect the model's composer from glib accusations of oversimplification. Minimalist it may be as a model of covirus evolution—and I do not have the know-
ledge to justify rococo elaborations—yet it contains
within it pon-trivially single-species dynamics mutumodel of covirus evolution—and I do not have the know-
ledge to justify rococo elaborations—yet it contains
within it, non-trivially, single-species dynamics, mutu-
alism connetition and parasitism—an entire soap opera ledge to justify rococo elaborations—yet it contains
within it, non-trivially, single-species dynamics, mutu-
alism, competition and parasitism—an entire soap opera
of ecological relationships This can be understood in two within it, non-trivially, single-species dynamics, mutualism, competition and parasitism—an entire soap opera
of ecological relationships. This can be understood in two
wave First, from a hiological point of view it is to alism, competition and parasitism—an entire soap opera
of ecological relationships. This can be understood in two
ways. First, from a biological point of view, it is to be
expected in a system of replicating entities that of ecological relationships. This can be understood in two ways. First, from a biological point of view, it is to be expected in a system of replicating entities that do not have exclusive access to their own gene products ways. First, from a biological point of view, it is to be expected in a system of replicating entities that do not
have exclusive access to their own gene products (Nee &
Maynard Smith 1990), and where population dynamics is
occurring at the two distinct levels of within and betw have exclusive access to their own gene products (Nee & Maynard Smith 1990), and where population dynamics is
occurring at the two distinct levels of within and between
plants. Second, from a theoretical point of view as I Maynard Smith 1990), and where population dynamics is
occurring at the two distinct levels of within and between
plants. Second, from a theoretical point of view, as I hope
I have shown, it comes about as a result of a new occurring at the two distinct levels of within and between
plants. Second, from a theoretical point of view, as I hope
I have shown, it comes about as a result of a new plants. Second, from a theoretical point of view, as I hope
I have shown, it comes about as a result of a new
confidence in using the same models in seemingly quite
dimarate biological contexts I have shown, it comes abconfidence in using the same
disparate biological contexts.
The particular emphasis of mfidence in using the same models in seemingly quite
parate biological contexts.
The particular emphasis of this paper has been on
ethodically building up to model (10) from the simplest

disparate biological contexts.
The particular emphasis of this paper has been on
methodically building up to model (10) from the simplest
starting place the Levins' metapopulation model The particular emphasis of this paper has been on
methodically building up to model (10) from the simplest
starting place, the Levins' metapopulation model,
equations (1) Giving only a partial treatment of the full methodically building up to model (10) from the simplest
starting place, the Levins' metapopulation model,
equations (1). Giving only a partial treatment of the full
model equations (10) has one somewhat misleading starting place, the Levins' metapopulation model, equations (1). Giving only a partial treatment of the full equations (1). Giving only a partial treatment of the full
model, equations (10), has one somewhat misleading
consequence. I have emphasized the coexistence difficul-
ties faced by the progenitor virus(es) when the covirus model, equations (10), has one somewhat misleading
consequence. I have emphasized the coexistence difficul-
ties faced by the progenitor virus(es) when the covirus
has arisen, but downplayed the problems faced by the consequence. I have emphasized the coexistence difficul-
ties faced by the progenitor virus(es) when the covirus
has arisen, but downplayed the problems faced by the
covirus itself—in particular, the threshold density it m ties faced by the progenitor virus(es) when the covirus has arisen, but downplayed the problems faced by the
covirus itself—in particular, the threshold density it may
first need to attain before it can spread. This was
mentioned opaquely by reference to the unstable smaller covirus itself—in particular, the threshold density it may
first need to attain before it can spread. This was
mentioned opaquely by reference to the unstable smaller
equilibrium in $\&$ 3 and will be discussed fully else first need to attain before it can spread. This was
mentioned opaquely by reference to the unstable smaller
equilibrium in $\S 3$ and will be discussed fully elsewhere.
Still once the covirus has jumped any initial hurdle mentioned opaquely by reference to the unstable smaller
equilibrium in $\S 3$ and will be discussed fully elsewhere.
Still, once the covirus has jumped any initial hurdle, the
future is bleak for the progenitor. equilibrium in § 3 and will be dis
Still, once the covirus has jumped
future is bleak for the progenitor.
 $\frac{4s}{\pi}$ in many areas of biology Still, once the covirus has jumped any initial hurdle, the
future is bleak for the progenitor.
As in many areas of biology, most of the recent

future is bleak for the progenitor.
As in many areas of biology, most of the recent
progress in covirus research has been in working out the
fine details of their molecular biology rather than filling fine details of their molecular biology rather than filling
in our knowledge of the natural history of these fasciprogress in covirus research has been in working out the
fine details of their molecular biology rather than filling
in our knowledge of the natural history of these fasci-
pating entities. That one of the clearest overvie fine details of their molecular biology rather than filling
in our knowledge of the natural history of these fasci-
nating entities. That one of the clearest overviews of
coviruses is now nearly one-quarter of a century ol in our knowledge of the natural history of these fascinating entities. That one of the clearest overviews of coviruses is now nearly one-quarter of a century old reflects this fact (Fraenkel-Conrat $\&$ Wagner 1977) But nating entities. That one of the clearest overviews of
coviruses is now nearly one-quarter of a century old
reflects this fact (Fraenkel-Conrat & Wagner 1977). But
without the natural history we cannot be confident in our coviruses is now nearly one-quarter of a century old
reflects this fact (Fraenkel-Conrat & Wagner 1977). But
without the natural history we cannot be confident in our understanding of the origins or maintenance of coviruses. without the natural history we cannot be confident in our
understanding of the origins or maintenance of coviruses.
It is to be hoped that the post-genomic world will see
halance return understanding of
It is to be hopee
balance return.

balance return.
I thank Brian Charlesworth for his careful reading of the manu-
script Mike Mayo and Stuart Macfarlane for very helpful statance recurn.
I thank Brian Charlesworth for his careful reading of the manuscript, Mike Mayo and Stuart Macfarlane for very helpful
comments and the University of Edinburgh for providing a posi-I thank Brian Charlesworth for his careful reading of the manuscript, Mike Mayo and Stuart Macfarlane for very helpful
comments and the University of Edinburgh for providing a posi-
tive research environment I thank John M script, Mike Mayo and Stuart Macfarlane for very helpful comments and the University of Edinburgh for providing a positive research environment. I thank John Maynard Smith for being a constant reminder of what the point ac tive research environment. I thank John Maynard Smith for

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