Should Models of Disease Dynamics in Herbivorous Insects Include the Effects of Variability in Host-Plant Foliage Quality?

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ABSTRACT: Interactions between insects and their baculovirus pathogens are often described using simple disease models. Baculoviruses, however, are transmitted when insects consume virus-contaminated foliage, and foliage variability, whether within or between host-plant species, can affect viral infectiousness. Insect-baculovirus interactions may thus be embedded in a tritrophic interaction with the insect's host plant, but disease models include only the host and the pathogen. We tested these models by measuring the transmission of a baculovirus of gypsy moths (*Lymantria dispar*) on red oak (*Quercus rubra*) and white oak (*Quercus alba*) in the field in six experiments over four years. In all experiments, there were only weak effects of host-tree species, and in only one did the best-fitting model include tree species effects. These weak effects of foliage variability on transmission were not due to a lack of foliage variability on viral infectiousness, because when larvae were force-fed virus-contaminated foliage, infection rates were higher on white oak. Our results suggest that feeding behavior plays an important role in baculovirus transmission and that models can usefully describe baculovirus dynamics even without including foliage variability. Our work provides a clear example of how two-species models are sometimes sufficient to describe what appear to be tritrophic interactions.

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Many herbivorous insects are afflicted with baculoviruses, diseases transmitted when host larvae consume virus-contaminated foliage (Cory et al. 1997). Although baculoviruses only infect arthropods, research on baculoviruses has nevertheless made general contributions to ecology. For example, studies of the role of baculoviruses in driving forest insect outbreaks have deepened our understanding of animal outbreaks and complex population dynamics (Myers 1993; Liebhold and Kamata 2000). Studies of the potential use of genetically engineered baculoviruses for the control of crop pests have similarly improved our understanding of competition among pathogen strains and the risks of releasing transgenic organisms (Cory 2000; Hails et al. 2002). An important part of these contributions has been the construction of simple mathematical models of baculovirus population dynamics (Anderson and May 1981; Briggs et al. 1995; Dwyer et al. 2000, 2004; Dushoff and Dwyer 2001; McCallum et al. 2001).

As in any science, however, the use of simple models in ecology must always be in doubt; in the words of Box (1979), "All models are wrong, but some models are useful." Accordingly, here we use interactions between insects and their baculoviruses as a test case for the use of simple models in describing species interactions. For insect-baculovirus interactions, the use of simple models has previously been justified in two ways. First, the basic biology of baculoviruses suggests that simple models may be appropriate, because insect-baculovirus interactions generally lack the complications of vectors or host immune memory, and infections are usually fatal (Cory et al. 1997). Second and more significantly, some baculovirus models have survived testing in the field (Dwyer et al. 1997; Hails et al. 2002). Most such tests, however, have focused on population structure and other concerns of theoretical ecologists (Dwyer 1991; Goulson et al. 1995; Knell et al. 1998; Reeson et al. 1998, 2000), without considering the

effects of variability in the foliage that insects feed on. Because baculoviruses are transmitted when insects consume virus-contaminated foliage, and because secondary foliage compounds, such as tannins, can interfere with the infection process (Feldman et al. 1999), variability in the foliage of an insect's host plants can affect the chance that the insect becomes infected (Duffey et al. 1995). Most models, however, assume that this variability has no effect, even though the assumption has rarely been tested. We therefore tested for effects of interspecific variability in host-plant foliage on insect-baculovirus interactions in the field, to further our understanding of baculovirus dynamics and more generally to consider the usefulness of two-species models for describing tritrophic or multitrophic interactions. The question that we ask is, How useful are models of insect-baculovirus interactions if they do not include effects of foliage variability among host-plant species?

For the baculovirus in our experiments, the nucleopolyhedrovirus of the gypsy moth (*Lymantria dispar*), variability in foliage quality among host-plant species is especially likely to affect virus transmission, because gypsy moth larvae can complete their development on hundreds of different tree species (Barbosa 1978; Barbosa and Greenblatt 1979; Lechowicz and Jobin 1983). Many of these species differ in secondary foliage chemistry in ways that strongly affect virus infection rates in the laboratory (Keating and Yendol 1987; Keating et al. 1990*a*, 1990*b*; but see Lindroth et al. 1999). It therefore seems likely that simple models will not survive direct tests of the effects of differences between host-tree species on the transmission of the gypsy moth virus.

For simplicity, we used just two tree species, red oak (*Quercus rubra*) and white oak (*Quercus alba*). We chose these two because gypsy moth outbreaks in North America often occur in forests made up of a mixture of red and white oak (Woods and Elkinton 1987; G. Dwyer, personal observation). Existing epidemic data, however, are not detailed enough to show clearly the effects of different tree species, and so here we use experiments to test the models. Also, the two tree species have similar branch architecture and leaf shape, which should minimize differences in behavior on the part of feeding larvae, thereby maximizing our chances of detecting effects of foliage differences. Finally and most importantly, in laboratory bioassays, larvae that consume virus on red oak foliage experience lower infection rates than do larvae that consume virus on white oak foliage, apparently because red oak foliage has higher tannin levels than white oak foliage (Keating et al. 1990*b*).

Nearly all the data showing effects of foliage variability on baculoviruses come from the laboratory, however, and so may not predict what happens in the field. In laboratory experiments, larvae are fed virus in the form of small leaf disks contaminated with virus, larvae are discarded if they do not consume the virus, and for reasons of statistical power, doses are set at levels that keep infection rates well below 100% (Keating and Yendol 1987). In contrast, in the field larvae become infected by feeding on foliage contaminated with the cadavers of previously infected larvae (Dwyer 1991). Because cadavers contain thousands of infectious doses (Shapiro et al. 1986), larvae that consume the virus in the field may ingest such large amounts that they become infected irrespective of host-foliage variability. That is, the probability of infection may be determined entirely by whether or not larvae consume the virus, because it may be that all larvae that consume the virus become infected. Models that ignore the effects of host-foliage variability might therefore be sufficient.

An important difference between laboratory and field experiments is thus the difference between measuring the probability of infection given virus consumption and measuring the overall probability of infection. Because the models describe the overall probability of infection, we tested them using field experiments. As we will describe, our data showed, at most, weak effects of tree species. To make sure that the weakness of these effects was not due to a lack of foliage differences between the two tree species, we also directly tested the foliage from the field experiments by using laboratory bioassays. These bioassays confirmed that the foliage in the field varied enough between species to affect the probability of infection, given virus consumption at low and moderate doses. A key point is thus that our interests are in the consequences of host-foliage variability rather than in the mechanisms that create it. For quantifying foliage variability, bioassays of host susceptibility on different foliage types are thus more appropriate than chemical analyses of foliage. Of course, the epidemiology of this virus may also be affected by other ways in which red and white oak differ besides foliage quality, such as differences in branch architecture or in the timing of bud burst (Hunter and Elkinton 2000). Unlike other differences between red and white oak, however, foliage quality differences are known to affect infection rates (Keating et al. 1990*b*), and so it is appropriate to consider foliage quality first. Similarly, our experiments are limited in their temporal and spatial scales and so may not include some features of natural transmission. Nevertheless, estimating key model parameters from such experiments has produced accurate predictions of infection rates in natural populations (Dwyer et al. 1997), suggesting that the experiments capture important features of natural transmission processes.

Finally, there are almost certainly circumstances under which differences between red and white oak foliage lead to differences in the transmission of the gypsy moth virus, because the biological differences in the foliage of the two

tree species are too substantial for there to be no effect whatsoever. For example, it is probably true at least some of the time that virus doses in the field are small enough for differences in foliage chemistry to affect transmission. The null hypothesis that differences between red and white oak do not affect the transmission of the gypsy moth virus is therefore almost certainly untrue a priori. In our data analyses we therefore do not use a test of statistical significance to determine whether the null hypothesis can be rejected; instead, we use the Akaike Information Criterion, or AIC, to choose among competing baculovirus transmission models that do or do not include differences between red and white oak, and we use Akaike weights to quantify the relative probability that each model is the best model (Burnham and Anderson 2002). This approach is aimed at a larger goal, which is to illustrate one possible solution to the general ecological problem of multiple causation. In the words of Quinn and Dunham (1983, p. 604), "The objective of investigation … is not to determine the single cause of a pattern, as no such cause exists, but rather to assign relative importances to the contributions of … a number of processes, all known or reasonably suspected of operating to some degree." Previous work has already made clear that the epidemiology of the gypsy moth virus is strongly affected by both host and pathogen density (Woods and Elkinton 1987) and by variability among individuals in their susceptibility to the virus (Dwyer et al. 1997). Here we attempt to determine whether interspecific variability in host-plant foliage quality is also important enough to be included in models of the dynamics of this virus.

Methods

Natural History of the Gypsy Moth–Virus Interaction

Like other nucleopolyhedroviruses, the gypsy moth virus consists of occlusion bodies that enable the virus to survive outside of the host (Miller 1997). Larvae that consume a sufficiently high number of occlusion bodies become infected and generally die, which in gypsy moths occurs after 6–21 days. Infected larvae are converted almost entirely into more occlusion bodies, and viral enzymes greatly weaken the larval integument (Volkman 1997). Infected cadavers therefore break open after death to release occlusion bodies onto the foliage for consumption by other larvae (Cory et al. 1997). Because occlusion bodies must be consumed for infection to occur, the gypsy moth and other Lepidoptera that feed only as larvae can generally only become infected as larvae.

Because gypsy moths overwinter in the egg, the virus must survive between larval seasons without being transmitted among larvae. In some insects, there is evidence that baculoviruses can be transmitted vertically by surviving in the tissue of adult females and thereby being passed on to progeny in the egg (Fuxa and Richter 1992; Burden et al. 2002). For gypsy moths, however, experiments have instead shown that the most important overwintering mechanism is external contamination of eggs that occurs when females lay egg masses on contaminated bark (Murray and Elkinton 1989, 1990; Murray et al. 1991). The virus is then reintroduced into the population when larvae from contaminated egg masses hatch out infected, as a result of consuming the virus as they eat their way out of the egg. At high densities, repeated rounds of transmission during the larval period can lead to very high infection rates during the final larval stages, or "instars" (Woods and Elkinton 1987; Woods et al. 1990); in fact, this "second wave" of infection largely determines the intensity of the epidemic (Woods and Elkinton 1987). Because the amount of virus produced by an infected cadaver increases exponentially with instar (Shapiro et al. 1986), and because virus on foliage breaks down over a period of a few days, infections among final (fifth and sixth) instars are largely due to virus from fourth-instar cadavers (Woods and Elkinton 1987). Infection rates among fourth instars thus have a very strong effect on infection rates among the final instars, and so transmission to fourth instars generally has the largest effect on the intensity of the epidemic. In our experiments we therefore measured transmission to fourth instars.

The ecology of the virus has become more complicated since 1989, when the fungal pathogen *Entomophaga maimaiga* was introduced into North American populations (Elkinton et al. 1991). Wetter years are now characterized by high levels of fungal infection, and competition between the fungus and the virus can reduce the intensity of virus epidemics. Nevertheless, through 2003, the virus continued to be an important source of mortality in North American gypsy moth populations (G. Dwyer, unpublished data).

Experimental Overview

Our research used a protocol for field transmission experiments that was developed by the first author and colleagues (Dwyer 1991; Dwyer and Elkinton 1993; D'Amico et al. 1996, 1998; Dwyer et al. 1997). As we have described, this protocol allows us to measure the overall probability of infection, whereas laboratory experiments measure the probability of infection given virus consumption. Mathematically, we can express this difference as follows. If the overall probability of infection is $p(I)$, the probability of infection given virus consumption is $p(I|C)$, and the probability of virus consumption is $p(C)$, then from elementary probability we have

$$p(I) = p(I|C)p(C). \quad (1)$$

When there is no effect of host-tree foliage on the probability of infection $p(I)$ in the field, but there are strong effects of such variability on the probability of infection given virus consumption $p(I|C)$ in the laboratory, there are two obvious possible explanations. First, there may be differences in $p(I|C)$ between the laboratory and the field, due, for example, to differences in virus dose. Second, effects of foliage on $p(I|C)$ may be counterbalanced in the field by effects of foliage on the probability of consumption $p(C)$. To tease apart the relative importance of $p(I|C)$ and $p(C)$ and to make sure that foliage in our experiments varied enough to affect $p(I|C)$, we therefore carried out two additional experiments in conjunction with each of our transmission experiments. These experiments were designed to measure $p(C)$ and $p(I|C)$ independently of our measurements of $p(I)$. As a measure of the probability of consuming the virus $p(C)$, we quantified feeding rates on foliage of the two tree species. To do this, we measured the leaf area consumed by individual larvae feeding on single leaves in cups in the laboratory. To confirm that feeding rates in the laboratory were representative of feeding rates in the field, we also measured the leaf area remaining on nonvirus control branches in the field experiments. As a measure of the probability of infection given virus consumption $p(I|C)$, we quantified infection rates in the laboratory on foliage of the two tree species, using the same protocol as in previous laboratory experiments (Keating and Yendol 1987; Keating et al. 1988, 1990*a*, 1990*b*; Hunter and Schultz 1993).

An important feature of our experiments is that we consider only constitutive differences between red and white oak, rather than induced differences. Defoliation by gypsy moths famously leads to increases in tannin levels in red oak (Schultz and Baldwin 1982), and apparently as a consequence, prior defoliation of red oaks can reduce infection rates in laboratory dose-response experiments (Hunter and Schultz 1993). Nevertheless, D'Amico et al. (1998) showed that defoliation of red oaks before field transmission experiments does not affect transmission rates, in that infection rates are very similar on previously defoliated and previously undefoliated trees. Of course, in all such transmission experiments, including our own, transmission is accompanied by at least partial defoliation, but D'Amico et al. (1998) also included treatments in which additional defoliation was imposed before the start of transmission. We suspect that the reason why this prior defoliation had no effect on D'Amico et al.'s results is that larvae in the field either do not consume any virus at all or consume so much that they become infected regardless of induction.

An alternative explanation for D'Amico et al.'s (1998) results, however, is that defoliation by gypsy moths only leads to induction of tannins long after defoliation has occurred. Indeed, in D'Amico et al.'s experiments, there were no detectable rises in tannins within a larval season, while in Hunter and Schultz's (1993) study, induction occurred only in the following year. Furthermore, to our knowledge statistically significant increases in tannin levels in red and white oak due to defoliation have been observed only in late June, July, August (Schultz and Baldwin 1982; Wold and Marquis 1997; D'Amico et al. 1998), or even October (Hunter and Schultz 1995), dates that are near the end of or after the gypsy moth larval season in North America. Similarly, although Rossiter et al. (1988) also observed a statistically significant induction effect, the effect was measured over the entire season. It is therefore unclear whether induction occurs early enough to affect virus transmission within a season in gypsy moth populations. In attempting to detect a difference between red and white oak, we therefore kept our experiments simple by allowing only for defoliation that occurs in the course of an experiment. More generally, the uncertain relationship between secondary foliage chemistry and virus transmission in the field suggested that we should measure host-foliage variability directly by using bioassays, rather than indirectly by quantifying secondary foliage compounds.

A related point is that, as the season progresses, leaves of red oak show increases in toughness, decreases in nitrogen content, and decreases in tannin concentrations (Keating et al. 1988; Hunter and Lechowicz 1992; Hunter and Schultz 1995). White oak has not been studied as intensively, but existing data show trends of declines in both tannins and nitrogen (Wold and Marquis 1997). These changes are important because they could affect transmission by changing either the infectiousness of the virus inside the larval gut, and thus the probability of infection given consumption, or larval feeding rates, and thus the probability of consumption. Our first two sets of experiments, however, were carried out in August (August 2, 2000; August 3, 2001), after naturally occurring gypsy moth populations had pupated. Neither experiment showed differences in transmission between red and white oak; moreover, previous work had anecdotally suggested that transmission on red oak is the same at different times during the spring and summer (Dwyer and Elkinton 1993; Dwyer et al. 1997; D'Amico et al. 1998). Nevertheless, it was possible that phenological changes in leaf chemistry and toughness had somehow obscured differences between the two tree species in these two experiments. In 2002 and 2003, we therefore repeated our experiments in June, when fourth-instar gypsy moth larvae are normally present in naturally occurring populations. In 2002, the experiments took place on June 26, when natural populations consist mostly of fifth instars plus a small fraction of fourths,

whereas in 2003 they took place on June 10, when natural populations consist mostly of fourth instars, with a small fraction of thirds and fifths (G. Dwyer, unpublished observations). These experiments again showed very small differences in transmission on the two tree species, suggesting that our results from 2001 and 2002 were robust. Nevertheless, because of modest differences from year to year in the strain of host larvae and in the area of forest where the experiments were carried out (details are given below), it was difficult to directly compare experiments across years. In 2003, we therefore repeated our experiments on July 1 and August 7, using the same strain of insect and the same area of forest as in the June 10 experiment. These latter three experiments thus permitted a direct comparison of the effects of phenology on virus transmission on the two tree species.

Field Transmission Experiments

In our field experiments, we measured transmission from larvae infected at hatching to uninfected larvae in the fourth instar. The uninfected larvae were hatched from eggs that had been surface disinfected with 4% formalin, which deactivates the virus (Dwyer and Elkinton 1995), and were reared in the laboratory on wheat germ–based gypsy moth diet at 26°C with 16 h of light and 8 h of darkness. Larvae that are within 24 h of molting can be recognized because their head capsules slip forward. We therefore prepared larvae for our experiments by removing third instars with slipped head capsules from their diet cups and placing them in empty cups at approximately 22°C in the laboratory for 48 h. At this temperature, larvae take about 24 h to molt to the next instar and then starve for 24 h. These larvae were then newly eclosed to the fourth instar at the time of the experiments and had starved for approximately 24 h before the experiments. This period of starvation ensured that any remaining food had been eliminated from the larval gut (Keating and Yendol 1987), so that any effects of foliage on virus infection were not obscured by the effects of previous diet. In 2000, however, a lack of larvae forced us to use some fifth instars.

To produce infected larvae, we again reared larvae in the laboratory, but in this case the surface of the diet was first coated with virus solution of a concentration sufficient to ensure nearly 100% infection. To reduce variability in the virus, we used the G2 plaque-purified clone of the virus (thanks to J. P. Burand, University of Massachusetts, Amherst). The infected larvae were reared from eggs from a USDA colony of low heterogeneity (Dwyer et al. 1997). Larvae from this colony are useful for our experiments, because when they are infected and then reared at 28°C, they die almost simultaneously at 6 days and are of the same size at death. Larvae that did not rapidly become infected molted to the second instar at 5 days after exposure and were discarded. Infected first-instar larvae were therefore removed from their cups on the fifth day after infection and were placed on branches in the field. Branches were in turn enclosed within mesh bags that are fine enough to permit natural variation in moisture and temperature yet prevent the escape of the larvae and the breakdown of the virus (G. Dwyer, unpublished data).

We distributed the initially infected larvae evenly on the foliage, to mimic the high dispersal typical of hatchling larvae (Dwyer and Elkinton 1995; Hunter and Elkinton 2000). We then waited for 7 days before adding uninfected larvae, to guarantee the death of the initially infected larvae (numbers of larvae are given below). Because we only carry out experiments in areas of low gypsy moth densities, mortality in virus-free control bags was usually low, reflecting only the accidental exposure of uninfected larvae to virus in the laboratory. Uninfected larvae were allowed to feed in the bags for 6 days in 2000 and 7 days in 2001, 2002, and 2003, time periods that were short enough to prevent the death of any initially uninfected larvae that became infected in the bag. The difference in exposure time in 2000 was due to logistic considerations and had no effect on our analyses. After the feeding period, we reared uninfected larvae for 3 weeks in individual diet cups in the laboratory. Dead larvae were examined under a microscope for the presence of occlusion bodies.

Based on previous experience, we used densities of initially infected larvae of 0, 25, 50, and 75 larvae per bag in 2000 and 0, 5, 25, and 50 per bag in 2001. These treatments were organized into seven replicate blocks, where each block consisted of trees in close proximity. In 2002, we used stochastic simulations to discover that a larger number of treatments provides narrower 95% confidence intervals than does a larger number of replicates, and so in 2002 we used 0, 5, 10, 25, 50 and 75 larvae per bag, with five blocks of red oak and four blocks of white oak. In 2003, a larger field crew enabled us to use the same density treatments as in 2002, but with seven replicates in the first two experiments and six in the third. In general, the densities in all experiments gave infection rates that spanned those observed in natural populations (Woods and Elkinton 1987). In 2000, we used 20 uninfected larvae, while in 2001, 2002, and 2003, we used 25 uninfected larvae. The smaller number in 2000 reflected only the availability of larvae. These densities are well within the range of densities observed in the field (Woods et al. 1990); moreover, previous experiments have shown that the density of uninfected larvae has no effect on transmission (D'Amico et al. 1998). In 2000 and 2003, the uninfected larvae that we used were from the USDA laboratory colony. This strain is the same as that used in all previous studies of the effects of foliage on gypsy moth virus trans-

mission (Keating and Yendol 1987; Keating et al. 1988, 1989, 1990*a*, 1990*b*; Hunter and Schultz 1993; D'Amico et al. 1998). Nevertheless, to ensure that these larvae did not behave strangely as a result of many generations of being reared in the laboratory, in 2001 and 2002 we used feral insects for the uninfected larvae. In 2001, these insects were from Allegan County, Michigan, while in 2002 they were from Waupaca County, Wisconsin.

In 2000 and 2001, our experiments were carried out at the Kellogg Experimental Forest (42°22′N, 85°21′W), in a grove of artificially planted trees that were roughly uniform in height (about 5 m). To make sure that our results were not specific to this grove, in 2002 and 2003, we instead used naturally occurring trees scattered over an area of second-growth forest in the Lux Arbor Reserve of the Kellogg Biological Station (42°28′N, 85°28′W). Both sites are within the current range of the gypsy moth in North America but had negligible levels of gypsy moths or virus in the years of our study. To ensure that transmission did not differ because of a difference in leaf areas between experiments, we equalized the leaf area in each treatment by measuring 100 leaves from the trees of each species in each year and then adjusting the number of leaves so that the total leaf area per bagged branch was the same on different tree species. Across experiments, the average number of leaves per bag was 37.8, and the average total leaf area per bag was 2094 cm^2.

Foliage Consumption Experiments

Beginning in 2001, we measured the leaf area consumed by larvae in the laboratory feeding on foliage of each tree species. Larvae fed for 24 h (2001) or 72 h (2002 and 2003) in individual 16-oz wax paper cups on single, premeasured (CI202 Leaf Area Meter, CID, Vancouver, WA) leaves that had been removed from trees <4 h earlier. The difference in the leaf area before and after feeding was the area consumed. To ensure that feeding rates would be comparable to rates in the field, leaves were taken from the same trees and larvae were taken from the same rearing cups as for the corresponding transmission experiment, and each feeding trial was begun the day after the corresponding transmission experiment. To confirm that the effects of foliage variability were similar between laboratory and field, we also measured the total leaf area remaining in the control bags of the transmission experiment. Because initial leaf areas were similar across bags, this provided an inverse measure of the feeding rate in the field. The results of the two measurements generally matched, in that feeding rates were always higher on red oak than on white oak in both types of experiment. Nevertheless, it was more difficult to control the initial area per bag in the field, and so in what follows we report only the results of the laboratory experiment.

Laboratory Dose-Response Experiments

Larvae in each dose-response experiment were from the same rearing cups as in the corresponding transmission experiment and were treated identically. To further facilitate comparison, we carried out the dose-response experiments on the day after (2000, 2001, 2002) or the day before (2003) the healthy larvae were added to the bags in the corresponding transmission experiment, and we used foliage from the same trees. Our protocol followed that of previous dose-response experiments (Keating and Yendol 1987; Keating et al. 1988, 1990*a*, 1990*b*; Hunter and Schultz 1993). We used a 4-mm-diameter cork borer to produce a leaf disk small enough to be consumed within 24 h; in practice, most disks were consumed within 2 h. To reduce drying out, we placed the leaf disks on small cubes of agar, which in turn were placed on wetted filter paper inside plastic cups. Next, we added a solution of 3 μL of virus in distilled water to each leaf disk. When the solution had partially dried, we added a larva to each plastic cup, and then we sealed the cups with plastic lids. Any larva that did not completely consume its leaf disk within 24 h was discarded. We then reared the larvae in individual diet cups in the laboratory for 3 weeks. Control larvae were fed leaf disks with droplets of distilled water. Doses were quantified by counting occlusion bodies under a microscope using a hemacytometer. In 2000, low virus doses produced low infection rates, and so we do not report the results of that experiment.

Statistical Methods

To compare leaf areas consumed, we used *t*-tests. For our other data sets, we used a model selection criterion, as described below.

Field Transmission Assays. The basis of our approach is to use field transmission experiments to choose among competing models of disease dynamics. We chose among models by using the corrected Akaike Information Criterion, or AIC_c (Burnham and Anderson 2002), where the correction, symbolized by "c," allows for small sample sizes relative to the number of parameters. The AIC_c is designed to choose among competing models while allowing for uncertainty in model choice, such that the best model has the smallest AIC_c. Under least squares, the AIC_c is

$$AIC_c = n \ln \sigma^2 + 2K\left(\frac{n}{n - K - 1}\right) \qquad (2)$$

(Burnham and Anderson 2002). Here n is the number of experimental units, in our case bagged branches, K is the number of parameters in the model, and $\sigma^2 = \sum_i \sum_j \epsilon_{i,j}^2/n$, where $\epsilon_{i,j}^2$ is the squared difference between the model and the data in replicate i of treatment j. Unlike classical significance tests, the AIC_c does not involve a calculation of the probability that a model is incorrect, and so we can use it not just to compare two models but to choose the best model from a group of models. Within such a group, the minimum AIC_c is achieved through a trade-off between the poorer fit of simpler models, which have larger values of $\ln \sigma^2$, and the more uncertain predictions of more complex models, which have larger values of $2K[n/(n - K - 1)]$. For our purposes, the AIC_c has an advantage over the more traditional likelihood ratio test, in that the likelihood ratio test assumes that at least one model is correct, whereas the AIC_c allows for the possibility that all the models are incorrect (Burnham and Anderson 2002). Moreover, analyzing our data using likelihood ratio tests gave very similar results. Mixing AIC_c analyses with significance tests, however, is not recommended (Burnham and Anderson 2002), and so we report only the former.

The models that we chose from were all derived from the same underlying epidemic model. Because gypsy moths, like most outbreaking forest insects, have only one generation per year, and because the nucleopolyhedrovirus can only infect larvae, this model describes only a single epidemic. The model is

$$\frac{dS}{dt} = -\bar{\nu} P S \left[\frac{S(t)}{S(0)}\right]^V, \tag{3}$$

$$\frac{dP}{dt} = \bar{\nu} P(t-\tau) S(t-\tau) \left[\frac{S(t-\tau)}{S(0)}\right]^V - \mu P. \tag{4}$$

Here S is the density of uninfected or "susceptible" larvae, P is the density of the pathogen in the form of infectious cadavers, and μ is the rate at which cadavers are rendered uninfectious by sunlight. The model includes a realistic delay τ between infection and death and a simple form of stage structure, in that early-instar cadavers are assumed to be smaller than later-instar cadavers (Dwyer and Elkinton 1993). The model also allows for heterogeneity in the risk of infection among individuals, in the form of a distribution on the transmission rate, with average transmission $\bar{\nu}$ and squared coefficient of variation V. Previous work has suggested that such heterogeneity plays an important role in the dynamics of this virus, both in transmission experiments and in epidemics (Dwyer et al. 1997, 2000). Moreover, the model has survived extensive testing with experimental and observational data for the gypsy moth virus (Dwyer et al. 1997) and can explain a high fraction of the variability in infection rates in natural epidemics (Dwyer et al. 2002). Nevertheless, it does not include any effects of interspecific variability in foliage among host trees, and so it simplifies the biology of the virus.

To test this model, we first simplified it to match the conditions of our transmission experiments. Because the virus does not break down in the bags, and because after the initially infected larvae die there is no input of virus, the change in the density of infectious cadavers $dP/dt = 0$. With this simplification, equations (3) and (4) can be reduced to a single equation for the density of uninfected larvae S (Dwyer et al. 1997), which can be solved to give

$$\frac{S(t)}{S(0)} = (1 + \bar{\nu} V P_0 t)^{-1/V}, \tag{5}$$

where $S(t)$ and $S(0)$ are the densities of uninfected larvae at the end and the beginning of the experiment, respectively, P_0 is the density of virus in the experiment, and t is the length of the experiment. In most experiments, there were at least one or two infected larvae in control bags, and so to allow for this contamination we used Abbott's method (Morgan 1992), by which one estimates the level of control mortality from the nonvirus control treatment and then adjusts mortality levels in the other treatments accordingly. To do this, we used the average of the infection rates in the nonvirus control replicates. To demonstrate more clearly the effects of host heterogeneity, we also considered models with no heterogeneity. This is equivalent to letting $V \to 0$ in equation (5), so that instead we have

$$\frac{S(t)}{S(0)} = e^{-\bar{\nu} P_0 t}. \tag{6}$$

The point of our transmission experiments is to estimate the probability of infection $p(I)$ as a function of virus density. Because $S(t)/S(0)$ is the probability of *not* becoming infected, and because all larvae are either infected or uninfected, $p(I) = 1 - [S(t)/S(0)]$. Although neither equation (5) nor equation (6) includes effects of tree species differences on transmission, we can produce models that *do* include such effects by assuming that the model parameters $\bar{\nu}$ and V are host-tree specific. We therefore asked, Do we need versions of equations (5) and (6) that are host-tree specific, or can we use the same equation for both red and white oak? In other words, we used our data to choose between models that allowed for effects of host-tree species and models that did not. Given that the probability of infection given virus consumption is higher on white oak, we therefore expected that the best model would allow for differences in $\bar{\nu}$ between red and white oak, but we also allowed for the possibility of differences in the

heterogeneity parameter V. The complete list of models was therefore as follows: (1) average transmission rate $\bar{\nu}$ and squared coefficient of variation in transmission rate V the same for the two tree species; (2) $\bar{\nu}$ differs between the two tree species, but V is the same; (3) V differs between the two tree species, but $\bar{\nu}$ is the same; (4) both $\bar{\nu}$ and V differ between the two tree species; (5) $V = 0$ (no host heterogeneity), and $\bar{\nu}$ the same; (6) $V = 0$, and $\bar{\nu}$ differs between the two tree species. If tree species has an effect on virus transmission, then neither model 1 nor model 5 will be the best model, and we will reject models that do not allow for an effect of host foliage on the transmission of this virus.

To reduce heteroscedasticity, we used weighted least squares (Dwyer et al. 1997), with the weight at each virus density equal to the inverse of the variance in the fraction uninfected at that density. Thus, σ^2 in equation (2) represents the weighted sum of squares. We also calculated Akaike weights, such that the weight w_i for the ith model is the relative probability that model is the true model (Burnham and Anderson 2002). The ratio of the Akaike weight of model i to the Akaike weight of the best model gives the odds against the event that model i is the best.

Dose-Response Assays. To analyze the dose-response data, we used the so-called logit or logistic model, according to which the probability of infection $p(D_i)$ at dose D_i is

$$p(D_i) = \frac{1}{1 + \exp(\beta_0 - \beta_1 \log_{10} D_i)}. \quad (7)$$

Here β_0 is the logit-transformed infection rate at very low doses, while β_1 is the slope of the logit curve with respect to dose. In some experiments there was control mortality, and in such cases we again used Abbott's method (Morgan 1992). Adjusting control mortality so as to achieve the best-fitting model gave very similar results. Although nonvirus mortality was generally <5%, on white oak on July 1, 2003, it was 16.1%. We therefore suspected that most of these nonvirus larvae died from the stress of being infected, and so for that treatment of that experiment we included both nonvirus and virus mortality.

In this type of analysis, one typically assumes binomial errors, which is equivalent to assuming that all individuals are independent. If this assumption is violated due to some form of overdispersion, the variance across individuals may be larger than binomial, biasing statistics like the AIC_c that are based on maximum likelihood. Because there is no robust test for overdispersion with binomial data (McCullagh and Nelder 1989), the recommended procedure is to calculate a variance inflation factor that is equal to the goodness of fit χ^2 for the most complex model divided by the degrees of freedom (Cox and Snell 1989). If this inflation factor is larger than 4, one then adjusts the AIC_cs by dividing all log-likelihood scores by the inflation factor (Burnham and Anderson 2002). The inflation factors for our data, however, were all <3 (in chronological order, 0.232, 2.16, 1.48, 2.76, 1.59), suggesting that overdispersion was at most a minor issue.

We used these data in an AIC_c calculation to ask the statistical question, Was there an effect of host-tree species on the probability of infection given virus consumption? Again this was phrased as, Do we need a version of equation (7) that is specific to host-tree species, or can we use the same equation for both red oak and white oak? As with the transmission data, the logit model can allow for tree species differences in several ways. The models that we considered therefore included (1) no effect of host-tree species, and thus identical values of β_0 and β_1 for each host-tree species; (2) different values of β_0 for each host-tree species, but the same value of β_1; (3) different values of β_1 for each host-tree species, but the same value of β_0; (4) different values of β_0 and β_1 for each host-tree species. Because previous work suggested that larvae fed virus on white oak were more likely to become infected than were larvae fed virus on red oak, we expected that the model with no differences in β_0 or β_1 would *not* have the smallest AIC_c value.

Results

In our field transmission experiments there were only modest differences in infection rates between red and white oak (fig. 1), and in all but the July 1, 2003, experiment, the best model included no effect of host-tree species (table 1). Although on July 1, 2003, the best model predicted higher infection rates on white oak at all doses, the odds against a model with no tree species differences were only about 3 : 1 (table 1). Moreover, on that date, the bootstrapped confidence intervals for the transmission parameters $\bar{\nu}$ on the two tree species overlapped (see table A1 in the online edition of the *American Naturalist*); given that the variability in the infection rates across replicates was small, this overlap suggests that the size of the effect was small. In contrast, there were strong effects of heterogeneity in susceptibility. The best model always included heterogeneity in susceptibility, and the odds against the models with no heterogeneity in susceptibility were always high (table 1). Also, increases in heterogeneity in susceptibility cause infection rates to rise to 100% relatively slowly with virus density (Dwyer et al. 1997), and this is visually apparent in most of the data sets (fig. 1). Finally, the best-fit values of the squared coefficient of variation parameter V were generally >1, and the lower confidence bounds were usually far from 0 (see table A1).

Although differences in transmission on the two tree

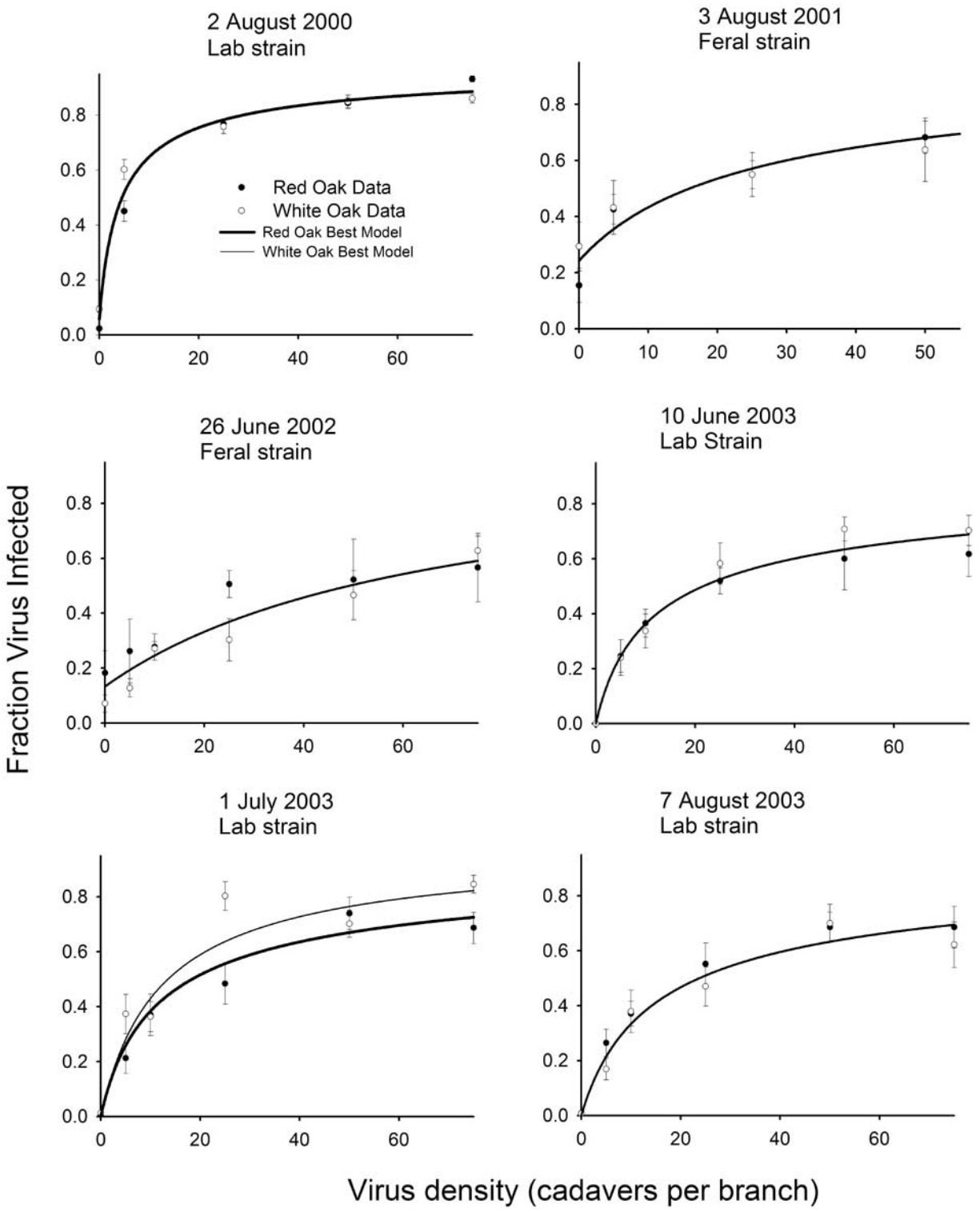

Figure 1: Results of field transmission experiments. The lines depict the best-fit version of equation (5), adjusted for control mortality. The dots represent the means of the data, and the error bars represent 1 SEM. In cases where there is only one line, the best model did not include differences between tree species.

Table 1: Corrected Akaike Information Criterion (AIC_c) analysis for field transmission data

Date and model	K	AIC_c	Rank	Weight	Odds ratio
August 2, 2000:					
$\bar{\nu}$ and V differ	9	17.88	2	.336	1.10
$\bar{\nu}$ differs	8	2.25	4	.102	3.61
V differs	8	18.97	3	.194	1.91
No host-tree effect	7	17.69	1	.369	1
$V = 0$, $\bar{\nu}$ differs	7	44.99	6	$<10^{-4}$	>1,000
$V = 0$, no effect	5	42.37	5	$<10^{-4}$	>1,000
August 3, 2001:					
$\bar{\nu}$ and V differ	8	20.84	4	.033	18.87
$\bar{\nu}$ differs	7	17.84	3	.147	4.22
V differs	7	17.81	2	.149	4.15
No host-tree effect	6	14.96	1	.618	1
$V = 0$, $\bar{\nu}$ differs	6	23.01	6	.111	55.91
$V = 0$, no effect	6	20.32	5	.0424	14.60
June 26, 2002:					
$\bar{\nu}$ and V differ	10	13.03	4	.024	15.99
$\bar{\nu}$ differs	9	7.82	2	.323	1.19
V differs	9	8.18	3	.270	1.42
No host-tree effect	8	7.48	1	.383	1
$V = 0$, $\bar{\nu}$differs	8	28.51	6	$<10^{-4}$	>1,000
$V = 0$, no effect	7	27.77	5	$<10^{-4}$	>1,000
June 10, 2003:					
$\bar{\nu}$ and V differ	10	22.56	4	.095	5.02
$\bar{\nu}$ differs	9	21.51	3	.160	2.96
V differs	9	20.46	2	.271	1.75
No host-tree effect	8	19.33	1	.475	1
$V = 0$, $\bar{\nu}$ differs	8	49.98	6	$<10^{-4}$	>1,000
$V = 0$, no effect	7	48.51	5	$<10^{-4}$	>1,000
July 1, 2003:					
$\bar{\nu}$ and V differ	10	20.36	4	.117	3.81
$\bar{\nu}$ differs	9	17.68	1	.448	1
V differs	9	18.49	2	.299	1.50
No host-tree effect	8	20.08	3	.135	3.31
$V = 0$, $\bar{\nu}$ differs	8	41.65	5	$<10^{-4}$	>1,000
$V = 0$, no effect	7	42.98	6	$<10^{-4}$	>1,000
August 7, 2003:					
$\bar{\nu}$ and V differ	10	28.16	4	.073	6.48
$\bar{\nu}$ differs	9	25.50	2	.275	1.72
V differs	9	26.33	3	.181	2.60
No host-tree effect	8	24.42	1	.471	1
$V = 0$, $\bar{\nu}$ differs	9	43.97	5	$<10^{-4}$	>1,000
$V = 0$, no effect	7	42.24	6	$<10^{-4}$	>1,000

Note: The parameter count includes the weights used in weighted least squares.

species were thus slight, larval feeding rates were always substantially higher on red oak than on white oak and these differences were always statistically significant (fig. 2). Although we did not measure tannins, this result was probably due to higher tannin levels in red oak (Keating et al. 1990*b*), coupled with the preference of gypsy moths for feeding on species of higher tannin content (Barbosa and Krischik 1987; Foss and Rieske 2003). Also, in all experiments except those of August 7, 2003, infection rates in the laboratory dose-response experiments were generally higher on white oak than on red oak (table 2), again presumably because of higher tannin levels in red oak (Keating et al. 1990*b*). The AIC_c therefore always chose a dose-response model for which there was an effect of host-tree species, and the odds against the model that assumed no host-foliage effect were never lower than 4 : 1 and were usually much higher (table 3). The results of the feeding trials and the laboratory dose-response experiments therefore suggest that the lack of a difference in transmission between red and white oak occurred at least in part because the higher probability of infection given virus consumption on white oak was counterbalanced by a higher probability of virus consumption on red oak. The August 7, 2003, experiments, however, are an exception to this trend, because on that date mortality was substantially higher on red oak than on white oak at the two higher doses, to the extent that the odds against a model with no effect of host-tree species were almost 6 : 1 (table 3).

The three sets of experiments in 2003 can also be used to assess the effects of phenology. Because our goal in this instance was simply to compare experiments qualitatively, we looked for effects of phenology by comparing transmission rates $\bar{\nu}$ across experiments rather than by carrying out an AIC_c calculation. Because the transmission rate $\bar{\nu}$ can be defined as the instantaneous, per capita, per unit time probability of infection, by analogy with the intensity parameters of stochastic birth-death models (Renshaw 1991), it can be used as an alternative measure of the probability of infection. Because the confidence intervals on $\bar{\nu}$ for the two tree species overlapped even for the July 1 experiment (see table A1), we calculated only a single value of $\bar{\nu}$ for each experiment. Figure 3*A* then shows that values of $\bar{\nu}$ were very similar in the June 10 and July 1 experiments and were only slightly lower in the August 7 experiment. Also, all the bootstrapped 95% confidence intervals strongly overlapped, suggesting that $\bar{\nu}$ was the same in all three experiments and hence that changes in phenology over the summer had no effect on the probability of infection. This pattern is the same even if we differentiate between the tree species or if we instead use the heterogeneity parameter V (see table A1).

In figure 3*B*, we have summarized the three dose-response experiments by calculating the 50% lethal dose, or LD_{50}, an inverse measure of the probability of infection given virus consumption, together with 95% confidence intervals calculated using the likelihood ratio method (Morgan 1992). Because infection rates increased slowly with dose (table 2), the confidence intervals on the LD_{50}'s for the two tree species generally overlapped, and so we again assumed no tree species effect. Figure 3*B* shows that

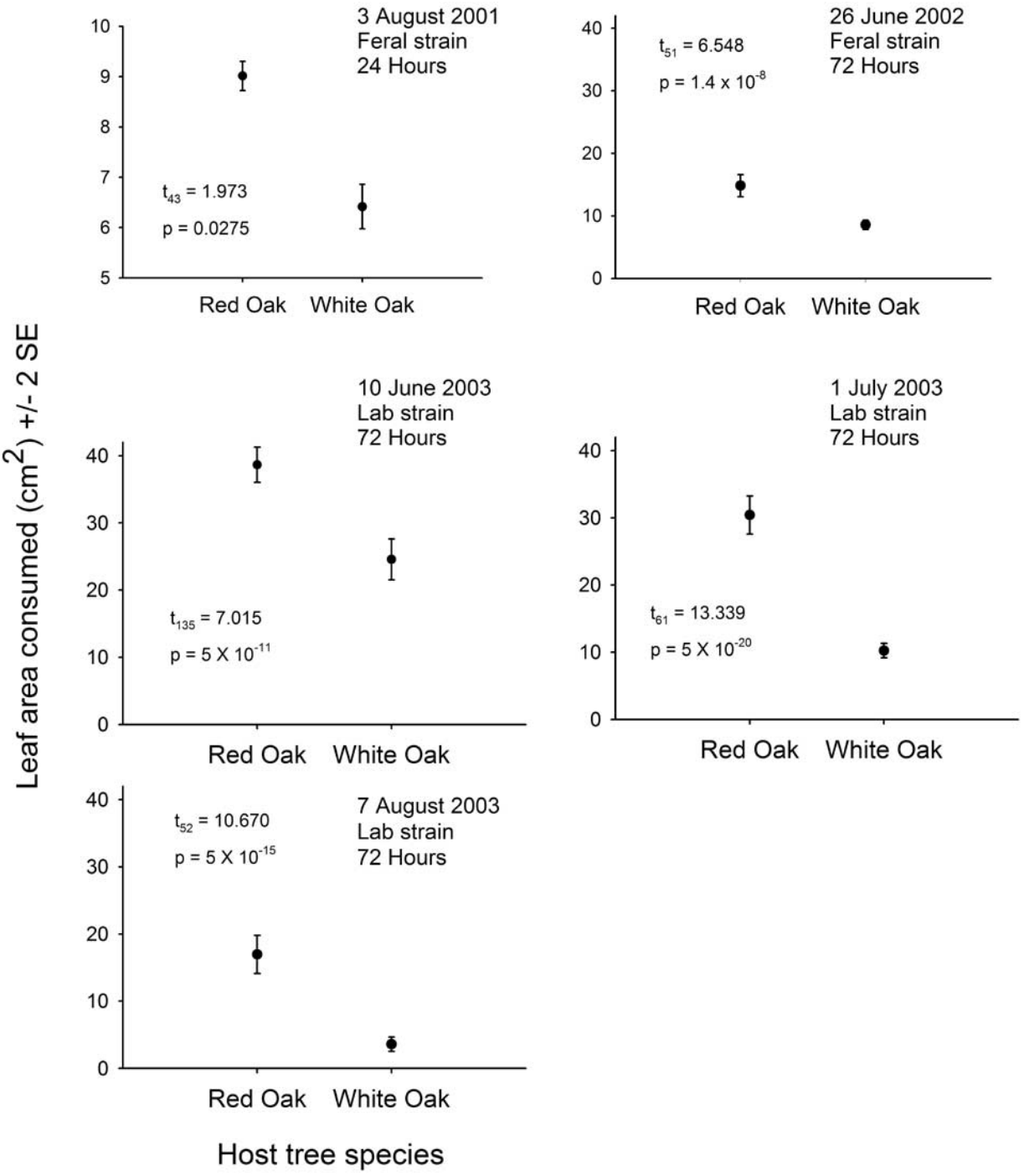

Figure 2: Area of single leaves eaten by larvae in cups in the laboratory. Larvae were fed for 24 h in 2001 and 72 h in all other experiments. Error bars represent 2 SEM.

Table 2: Results of dose-response experiments

	Doses (occlusion bodies)				
Date and tree species	0	3,000	9,000	30,000	90,000
August 2, 2001:					
Red oak	.00 (46)	.216 (37)	.372 (43)	.442 (43)	
White oak	.075 (40)	.289 (45)	.444 (45)	.688 (32)	
June 26, 2002:					
Red oak	.00 (54)		.185 (27)	.192 (52)	.511 (45)
White oak	.021 (48)		.25 (32)	.372 (43)	.711 (45)
June 10, 2003:					
Red oak	.00 (50)		.286 (49)	.615 (52)	.674 (49)
White oak	.02 (50)		.240 (50)	.680 (50)	.860 (50)
July 1, 2003:					
Red oak	.00 (48)		.140 (50)	.432 (44)	.457 (46)
White oak	.00 (22)		.294 (34)	.529 (34)	.478 (46)
August 7, 2003:					
Red oak	.00 (50)		.160 (50)	.316 (38)	.760 (50)
White oak	.00 (50)		.237 (38)	.250 (20)	.583 (12)

Note: Doses are in terms of number of viral occlusion bodies. Fractions show the fraction of larvae that became infected in each treatment, while the numbers in parentheses are the total numbers of larvae exposed.

the LD_{50} was significantly lower on June 10 than on July 1, in that confidence intervals did not overlap, and was marginally lower on June 10 than on August 7, in that confidence intervals slightly overlapped. The probability of infection given virus consumption thus declined after mid-June.

Finally, in figure 3*C*, we have summarized feeding rates by averaging the amount consumed across tree species at each date. Although rates were always significantly different between the two species, figure 3*C* shows that the decline over the summer was much larger than the difference between species on any date. Note that the error bars in figure 3*C* show 2 SEM, so that all differences between dates are significant. The probability of virus consumption thus declined dramatically over the summer.

In summary, over the course of the 2003 summer, the probability of infection was roughly constant, but the probability of infection given virus consumption and the probability of virus consumption both declined. The lack of an effect of phenology on the probability of infection therefore did not appear to be due to a balance between an increased probability of virus consumption and a reduced probability of infection given virus consumption.

Discussion

To our knowledge, our work is the first field test of the hypothesis that interspecific differences in host-foliage quality affect the transmission of baculoviruses. Our data suggest that differences between red and white oak have only weak effects on the transmission of the gypsy moth virus (fig. 1). Moreover, the lack of change in the transmission rate across the summer of 2003 suggests that phenological changes also have little effect on the transmission of this virus. The effects of host-foliage variability in our experiments were thus weak, compared, for example, to the effects of host heterogeneity in susceptibility. With enough replicates, of course, we would inevitably detect effects of interspecific or phenological variability in foliage quality on transmission. Indeed, an effect was detectable in the July 1, 2003, data, and in most other experiments the odds against one of the models that included an effect of host-tree species were $<2:1$ (table 1), suggesting that interspecific effects can sometimes be detected. Nevertheless, our data provide only weak evidence that the model in equations (3) and (4) should be modified to include such differences (fig. 1; table 1). Moreover, extending the full model in equations (3) and (4) to allow for multiple host-plant species would require at least two additional equations, significantly complicating the model. In short, we are not arguing that host-foliage variability never affects the transmission of this virus, but instead that models that do not include such effects are likely to provide just as good an approximation to virus epidemics in gypsy moth populations as models that do, and thus that such effects are of limited biological importance.

The small effects of host-foliage variability in our field experiments were not due to a lack of an effect on the probability of infection given virus consumption. In all of our dose-response experiments, the model that best described the data included effects of tree species (table 3), and in most cases infection rates on white oak were sub-

Table 3: Corrected Akaike Information Criterion (AIC_c) analysis for models fitted to dose-response data

Date and model	K	AIC_c	Rank	Weight	Odds ratio
2001:					
β_1 and β_0 differ	5	332.07	1	.543	1
β_1 differs	4	333.65	2	.206	2.20
β_0 differs	4	334.00	3	.247	2.63
No host-tree effect	3	341.96	4	.0039	140.53
2002:					
β_1 and β_0 differ	5	300.08	1	.5153	1
β_1 differs	4	300.26	2	.4705	1.10
β_0 differs	4	308.06	3	.0095	54.11
No host-tree effect	3	154.69	4	.0047	109.1
June 10, 2003:					
β_1 and β_0 differ	5	361.15	1	.826	1
β_1 differs	4	366.35	3	.0613	13.46
β_0 differs	4	365.57	2	.0906	9.10
No host-tree effect	3	368.36	4	.0225	36.69
July 1, 2003:					
β_1 and β_0 differ	5	321.62	1	.424	1
β_1 differs	4	323.04	3	.209	2.03
β_0 differs	4	322.54	2	.268	1.58
No host-tree effect	3	324.54	4	.0988	4.29
August 7, 2003:					
β_1 and β_0 differ	5	230.52	1	.638	1
β_1 differs	4	233.48	2	.126	5.06
β_0 differs	4	233.57	3	.121	5.25
No host-tree effect	3	233.69	4	.115	5.57

stantially higher than those on red oak (table 2), as in Keating et al.'s (1990*b*) dose-response experiments. Because our experiments used foliage from the same trees as in our field transmission experiments, they demonstrated that the foliage in the transmission experiments varied in a biologically meaningful way. Although we did not quantify any of the constituents of host-plant foliage, Keating et al.'s (1990*b*) work suggests that the basis of the difference in infection rates in our dose-response experiments was lower tannin levels in white oak. Moreover, infection rates in our dose-response experiments were similar to those in the literature, suggesting that host-foliage quality in our experiments was typical. The infection rates that we observed on red oak (table 2) were roughly in accord with estimates of LD_{50}'s on red oak that have ranged from 20,000 to 100,000 occlusion bodies (Keating and Yendol 1987; Keating et al. 1990*a*, 1990*b*; Hunter and Schultz 1993). Similarly, the levels of infection that we observed on white oak were comparable to Keating et al.'s (1990*b*) observation of 79% infection with 60,000 occlusion bodies.

It is important to recognize that our results only apply to two tree species and that transmission rates on other tree species may be different. For example, the probability of infection given virus consumption is moderately higher on both big-toothed aspen (*Populus grandidentata*) and quaking aspen (*Populus tremuloides*) than on red oak, again because of lower tannin levels in the aspens (Keating and Yendol 1987; Keating et al. 1990*a*, 1990*b*). Nevertheless, we suspect that transmission is unlikely to be much different on aspens, because during the August 7, 2003, experiments, we carried out a third set of treatments on quaking aspen in parallel with the treatments on red and white oak; infection rates were nearly the same on all three species. Of course, as with the effects of differences between red and white oak or with the effects of phenology, a much larger number of replicates may have allowed us to detect an effect, but the effect appears to be quite weak. This lack of an effect of aspen foliage on transmission leads us to suspect that transmission rates on other tree species may be similar to transmission rates on oak, but confirmation of this result requires further experiments. Similarly, we of course do not yet know whether interspecific variability in foliage also has little effect on the transmission of other insect viruses. Instead, we hope to have made clear that the effects of host-foliage variability on baculovirus transmission in the field cannot necessarily be predicted from the effects of such variability in the laboratory.

In particular, our results suggest that the weak effects of foliage quality on transmission were partly due to the countervailing influences of the probability of consumption $p(C)$ and the probability of infection given consumption $p(I|C)$. That is, feeding rates were always higher on red oak than on white oak (fig. 2), while infection rates in bioassays were generally higher on white oak than on red oak (table 2). Nevertheless, this explanation is clearly insufficient, in several ways. First, on August 7, 2003, feeding rates and laboratory infection rates were both higher on red oak, but transmission was again very similar on the two tree species. Second, both the probability of infection given virus consumption and the probability of virus consumption declined over the summer of 2003 (fig. 3), yet the overall probability of infection changed very little. Third, in our August 7, 2003, experiments, the probability of infection given virus consumption was higher on quaking aspen than on either oak species, and the feeding rate on aspen was similarly higher than that on either oak species, yet transmission was very similar on aspen as on the oaks.

Countervailing effects of host foliage on the probability of virus consumption and on the probability of virus infection given consumption thus cannot fully explain our transmission data. Larvae that consume the virus may therefore always receive a massive dose, so that host-foliage variability has no effect on the probability of infection given virus consumption in the field. Irrespective of the explanation, our work suggests that feeding behavior plays

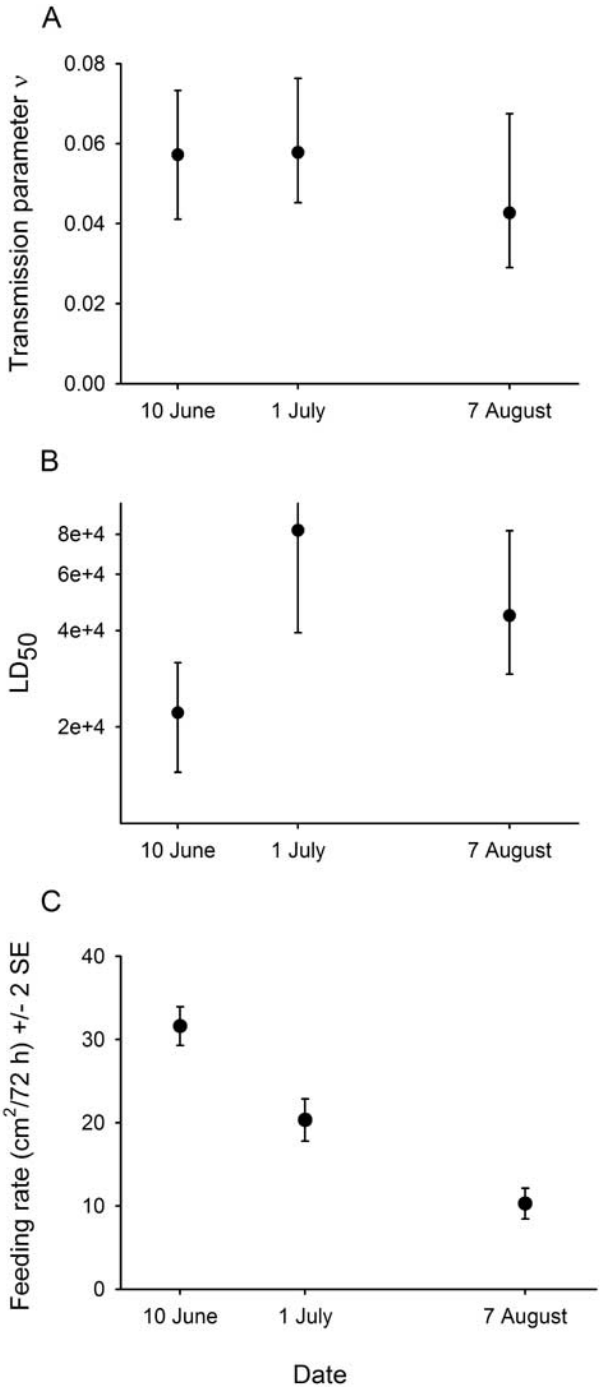

Figure 3: Trends in transmission and components of transmission over time in 2003. *A*, Transmission parameters $\bar{\nu}$ from field experiments, calculated using equation (5), assuming no effect of host-tree species. Error bars represent bootstrapped 95% confidence intervals. *B*, Doses giving 50% mortality, the LD_{50}'s, in dose-response experiments, calculated using equation (7), assuming no differences among host-tree species. The scale on the vertical axis is such that the upper error bar on July 1 is truncated, to make it easier to see that the lower error bar on that date does not overlap with the upper error bar on June 10. Error bars represent 95% confidence intervals, calculated using the likelihood ratio method (Morgan 1992). *C*, Average feeding rates, square centimeters consumed per 72 h, calculated across both tree species. Error bars represent 2 SEM.

a larger role in determining baculovirus transmission than has previously been recognized. For example, during our transmission experiments we observed that larvae on white oak move around the branch far more than do larvae on red oak and that larvae move more when the foliage is tougher later in the summer. Computer simulations carried out with a collaborator (K. Drury and G. Dwyer, unpublished manuscript) have shown that higher movement rates lead to higher infection rates. Increased movement may thus explain why the probability of infection changed very little with time in 2003, even though feeding rates declined. A key conclusion of our work, then, is that effective extrapolation from baculovirus infection rates in the laboratory to transmission in the field is likely to require a better understanding of host behavior. We therefore suggest that progress in understanding the effects of host-foliage variability on baculovirus transmission will proceed more rapidly if future work concentrates on larval behavior rather than on measurements of host-foliage chemistry. Indeed, although the decline in the feeding rate over the summer of 2003 may have been due to declining tannin levels, an equally likely explanation is that it was due instead to increases in leaf toughness (Hunter and Lechowicz 1992). The decline in infection rates in the laboratory over the summer of 2003 is even harder to explain in terms of previous observations of oak foliage chemistry. Although red and white oak tannin levels have been observed to decline over the summer (Keating et al. 1988; Hunter and Lechowicz 1992; Hunter and Schultz 1995; Wold and Marquis 1997) and such declines should lead to increases in infection rates (Keating et al. 1990*b*), in our experiments infection rates instead declined.

The basic conclusion of our research is that there are no compelling reasons to include the effects of differences between these two tree species in models of the gypsy moth virus. Simple models of the dynamics of baculoviruses may thus be useful, even though they ignore the effects of variability in host foliage. More generally, our work provides a concrete example of why more complex ecological models are not always better and suggests that two-species models may sometimes be sufficient for describing what appear to be tritrophic interactions. The previous successes of the model in equations (3) and (4) (Dwyer et al. 1997, 2002), together with the results that we present here, sug-

gest that simple disease models can indeed be useful for describing insect-baculovirus interactions.

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