l c a , – a g ac , a c b a

Bret D. Elderd[a], Brian J. Rehill[b], Kyle J. Haynes[c]

the soil-moisture differences that determine forest composition have no direct effect on the gypsy moth (12). Meanwhile, simple natural-enemy models that include a specialist baculovirus pathogen (14) and a generalist predator (5) can reproduce qualitative features of gypsy moth cycles (6), but standard models do not produce a subharmonic. Bjornstad et al. (13) therefore extended a natural-enemy model to allow for spatial variability in generalist-predator attack rates. Their work suggests that the subharmonic requires some kind of spatial structure, but their model only produces a subharmonic if infected larvae are allowed to disperse and uninfected larvae are not allowed to disperse. In nature both infected and uninfected larvae disperse (15), so spatial variability in generalist predators does not appear to be a sufficient explanation.

We therefore considered whether the observed differences in outbreak dynamics between forest types could instead be due to differences in inducibility between genera of gypsy moth host trees. In the range of the gypsy moth in North America, defoliation induces hydrolyzable tannins in most oak species (16), including red oak ( ) (17), black oak ( ) (17), and chestnut oak ( ) (18), whereas the effects of white-oak defoliation ( ) on gypsy moths are also likely due to increases in hydrolyzable tannins (19). Meanwhile, pines do not contain hydrolyzable tannins at all (20), whereas levels of hydrolyzable tannins in hickories are close to or equal to zero (21). The effects of induced hydrolyzable tannins on baculovirus transmission are therefore likely to be stronger in oak–hickory forests than in oak–pine forests because of the higher fraction of oaks in oak–hickory forests.

Direct toxic effects of induced defenses on gypsy moths are known to be relatively weak (22), but like many baculoviruses (23), the gypsy moth virus is transmitted when host larvae consume virus-contaminated foliage. Induced hydrolyzable tannins in foliage can therefore alter a gypsy moth larva's risk of infection, but as we will discuss, previous laboratory evidence for such effects (24) was not consistent with field data (14). Induced birch defenses (*B* ssp.) can similarly alter the responses of autumnal moth (*E*) larvae to artificially implanted plastic filaments in the laboratory (25), but efforts to detect induction effects on autumnal moths in the field were likewise unsuccessful. Also, there is no obvious signature of induced defenses in time series of autumnal moth defoliation (26).

Accordingly, for differences in host-plant inducibility to explain the disparate dynamics of gypsy moth outbreaks in oak–hickory and oak–pine forests, induced hydrolyzable tannins in oaks must first of all affect baculovirus transmission in nature. We therefore carried out an experiment to test whether induced hydrolyzable tannins modulate baculovirus transmission under field conditions. Second, spatial variability in tree-species composition must explain the differences in outbreak dynamics between the two forest types. We therefore used a mathematical model to test whether the mechanism revealed by our field experiment produces alternating severe and mild outbreaks in simulated oak–hickory forests and consistently moderate outbreaks in oak–pine forests, as seen in the data for each forest type.

Results

A previous effort to induce hydrolyzable tannins using artificial defoliation was unsuccessful (27). We therefore induced hydrolyzable tannins by spraying foliage with the plant-signaling compound jasmonic acid (JA) (28), which had previously been used to induce hydrolyzable tannins in red-oak seedlings in the greenhouse (29). Hydrolyzable tannin levels in oak foliage in nature increase after budburst in defoliated trees and decline in undefoliated trees (17). We thus expected that induction effects would be manifest through statistically significant effects of week

Because we explicitly accounted for variability in infection risk, our results instead imply that virus mortality will increase with host density, for reasons that are explained by epidemiological theory. In our field experiment, the decline in variability in infection risk was much more dramatic than the decline in the

spatial scales of the data to which we compare our model predictions. We therefore note that the model parameters that determine the dynamics of the induced defenses were first fit to the experimental data, but then were adjusted to give a better fit to the defoliation data (*IA* , section 2.3). The model output thus does not depend on the accuracy of our experimental measurements, and so our experimental data and the predictions of our spatial model serve as stand-alone arguments that nevertheless complement each other.

Variability in plant quality similarly modifies the effects of predation on population cycles of herbivorous mammals (37),