

Variation in Disease Severity in the Lodgepole Pine-Western Gall Rust Pathosystem

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This project was supported by the Natural Sciences and Engineering Research Council (NSERC) of Canada grant F-0003.

Accepted for publication 18 May 1990 (submitted for electronic processing).

ABSTRACT

van der Kamp, B. J., and Tait, D. E. N. 1990. Variation in disease severity in the lodgepole pine-western gall rust pathosystem. *Phytopathology* 80:1269-1277.

The number of western gall rust (*Endocronartium harknessii*) infections per tree was measured in a lodgepole pine (*Pinus contorta*) population consisting of 3,215 trees in 20 blocks, established from a single, large seed lot collected and planted near Prince George, B.C. Canada. Average disease incidence in these blocks varied from 0.58 to 6.07 infections per tree. A method is presented that allows the prediction of tree frequencies

by disease severity classes for each block from a single distribution of relative susceptibility. Resistance genes appear to interact multiplicatively. The most susceptible 10% of the population was estimated to have an infection rate about 800× as high as the least susceptible 10%. The implications of this wide range in susceptibility for stability of the pathosystem are discussed.

Additional keywords: additive resistance, horizontal resistance, incidence severity relationship, multiplicative resistance, natural pathosystem, nonspecific resistance, *Peridermium harknessii*, pine stem rust, stability.

Several studies of natural pathosystems report that if the same population of hosts is exposed to a pathogen under different conditions, different distributions of infection severity result. Good examples of this phenomenon are described in (5) and (12). Under conditions not very favorable to the pathogen, many or most individuals are free of infection or lightly infected, resulting in an inverse J-shaped distribution of disease severity, while under more severe disease conditions, the same pathosystem may show a normal, or even a negatively skewed distribution. This leads to a problem. The consequences of various theoretical models of the expression of resistance, pathogenicity, and their interaction on the distribution of infection have been explored in a number of papers (4,7). But which of the various distributions that may be observed in a particular pathosystem is the appropriate one to test a model prediction? In this paper we propose, using the natural pathosystem of western gall rust (*Endocronartium harknessii* (J. P. Moore) Y. Hiratsuka) on lodgepole pine (*Pinus contorta* Douglas ex Loud.) as an example, that the underlying distribution of relative susceptibility can be characterized and used to test various models of host-parasite interaction. Such a distribution also allows the prediction of the distribution of disease severity for any average level of infection.

The lodgepole pine-western gall rust pathosystem in the study area in central British Columbia can be viewed as a natural pathosystem. Large-scale harvesting of lodgepole pine in this region did not start until the mid 1960s, and large areas of both mature and immature pine remain undisturbed. Furthermore, in those areas that have been logged and regenerated, either naturally or by planting, no attempt has been made to alter the western gall rust resistance of the pine population.

Western gall rust on pine is a useful model for the study of natural pathosystems. Infection produces discrete galls that are easily counted, and the number of galls per tree is not only an indication of the susceptibility of that tree, but also a measure of the inoculum produced and the damage sustained by that tree. Furthermore, there is a good deal of variation in resistance; the rust has a simple life cycle; aeciospores are easily collected and can be stored for years; and artificial inoculation of seedlings with good control of spore deposition is possible. The major disad-

vantage of the pathosystem is the long time period necessary for many types of inoculation experiments.

Western gall rust is the most common stem rust of lodgepole pine in Canada (2,10,21,24). The rust is a native pathogen and occurs throughout the ranges of lodgepole and jack (*P. banksiana* Lamb.) pine except perhaps at their extreme northern limits, as well as on several other hard pine species. The rust has a simple life cycle. Aeciospores produced on discrete woody galls in the spring infect newly emerging pine shoots. Spermogonia are only rarely produced. Hence, if meiosis occurs at spore germination (9), it is probably preceded by the fusion of two identical nuclei. Alternatively, nuclear divisions at germination may be strictly mitotic (6,13). Isozyme analysis suggests that *E. harknessii* is homozygous (18). Thus, the offspring of a single infection will normally be genetically identical to that infection.

Two to four years elapse between infection and spore production. Thereafter, galls produce spores annually for about four years, but occasionally for several decades (19). Damage to trees occurs in two forms. Infection of the leader or of laterals close to the bole results in a stem gall. Trees with stem galls within a few meters of the ground usually die before maturity, either by breakage at the gall or when the gall bark and cambium are killed by secondary fungi or girdled by rodents. Branch infections cause some premature branch mortality which may result in reduced volume increment, although the effect on growth has not been demonstrated (8). Branch infections are much more numerous than stem infections and produce the bulk of the inoculum. Under artificial inoculation conditions using dry spores dusted on uninjured seedlings, followed by misting and moist incubation, susceptible seedlings can have as many as 150 galls per meter of internode (16). Inoculation, under similar conditions, of grafted scions derived from trees in our study area and therefore of the same physiological age as the trees described in this study, yielded an average of 15.6 infections per meter of internode (maximum 100 infections per meter) (13). Under natural conditions, the number of galls is much lower and seldom exceeds one gall per meter of internode length in the most severely infected trees (19).

There is considerable variation in the resistance of pine to western gall rust (3,11,13,14,16,23). Variation in pathogenicity among rust isolates is less clear. Single gall spore collections do vary (13,16), but while such variation may be genetic, it may

also arise from differences in spore quality resulting from host effects, aecium maturity, contamination, or other causes. Tests using single gall isolates that have been replicated by inoculation have not to our knowledge been reported. A study of the pathogenicity of five single gall spore sources replicated on 95 three-year-old trees derived from 12 open-pollinated families, in which both the isolates and the parent trees came from a single stand, failed to detect any interaction between spore source and either tree or family (16). On the other hand, a study involving the inoculation of 16 grafted pine clones with 4 single gall spore sources did show a significant interaction between clone and spore source (13). That interaction was largely attributable to variation in the infection rate, and not to the presence or absence of infection in particular isolate-clone combinations. These results suggest that while some specific resistance, conditioned by major genes for resistance in the host and complementary genes for virulence in the pathogen, may be present, it does not play a dominant role in a local pine-western gall rust pathosystem. Geographically remote pine and western gall rust spore sources do show an interaction that might be caused by major genes (20).

MATERIALS AND METHODS

The population to be described consisted of a set of 20 blocks of lodgepole pine of 102–318 trees each, all raised from a single large seed collection originating near Prince George, B.C. These

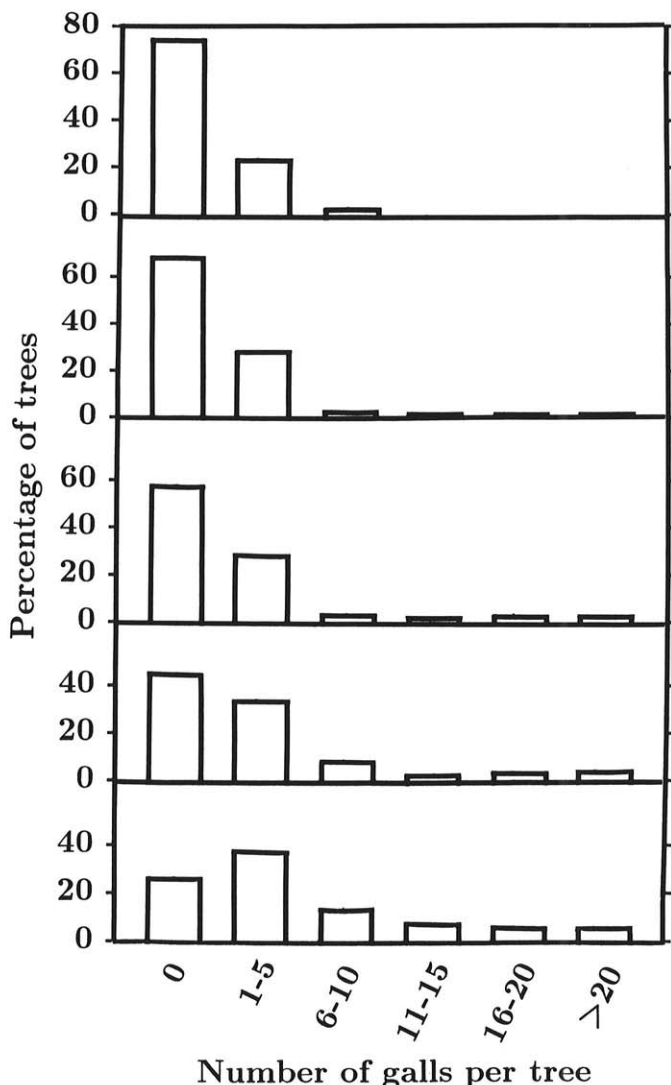


Fig. 1. The distribution of western gall rust infection by disease severity classes for five of 20 blocks of a lodgepole pine population. These blocks span the range of infection encountered.

blocks were part of a planting density study established in 1967, using three densities (640, 1,080, and 2,200 stems per ha) with two replicates at each of three locations, all within 100 km of Prince George, B.C., and separated from each other by similar blocks of Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco) and white spruce (*Picea glauca* (Moench) Voss). At one of the locations, an additional two replicates at 4,300 stems per ha were established. Block size varied from 0.09 to 0.22 ha. Natural, young pine stands infected by western gall rust surrounded all locations.

In 1980, the number of stem and branch infections per tree for each of the 3,215 trees in the total trial was recorded. In addition, a stem map was prepared for each block showing the location and number of infections of each tree. The blocks were established on uniform and carefully prepared terrain, and the variation in tree height within blocks was rather small. Crown shape and architecture, which are genetically controlled and form part of the complex set of tree characteristics that determine resistance through their effect on spore deposition and within crown microclimate, varied randomly within the blocks. The average block infection level varied from 0.58 to 6.07 infections per tree. The overall infection level was 2.26 galls per tree. Stand density was not related to disease incidence (19). For each of the twenty blocks, a frequency distribution of trees by number of galls per tree was prepared. Five of these are shown in Fig. 1.

In addition, the height, diameter at breast height (dbh), age, and number of stem and branch galls were measured for a block of 370 trees in an older, natural stand surrounding one of the three locations. These data are described in detail in (19).

THEORETICAL MODELS OF THE DISTRIBUTION OF RELATIVE SUSCEPTIBILITY

We postulate that the number of galls per tree is a function of the genetically determined susceptibility of that tree to the mixed natural inoculum to which it is exposed, the environment in which that tree has grown over time, and the nature (amount, genetic composition, and quality) of the inoculum. For the purpose of building the models of the distribution of relative susceptibility, it was assumed that neither the environment nor the inoculum made a significant contribution to the variation in number of galls per tree within blocks. These assumptions are supported by three tests (described below) that deal with variation in inoculum concentration and environment within blocks, variation in tree size, and variation in the genetic composition of the inoculum within blocks over time. The effects of the environment and of the nature of the inoculum were combined to generate an effective dosage level. The effective dosage level experienced by a block of trees is considered to be proportional to the average number of infections per tree in that block. Unit dosage is defined as that level of effective dosage that results in an average of one gall per tree in the host population. The infection rate for each tree is thus assumed to be proportional to the effective dosage experienced by the block in which it occurs and to the tree's susceptibility.

However, host susceptibility and effective dosage level as defined here do not wholly determine the actual number of galls that will appear on a tree. The source of uncertainty resides in the random processes of spore deposition and penetration. One way to visualize this is to consider that the critical events of penetration and establishment of the pathogen take place through a microscopic infection court. Such a court may be thought of as a small space which exists for a limited time determined by the duration of suitable environmental conditions and the stage of development of the host tissue. Each tree has many such infection courts; and at infection rates commonly observed in the field, only a few of these become infected. For instance, inoculation under controlled conditions of unwounded, grafted material derived from the study area, using moderate inoculum loads, yielded about 500× as many galls per meter of branch length as the natural infection level (estimated from [13] and [19]). Thus, at a particular effective dosage rate, each infection

court has a particular but small probability of becoming infected. The uncertainty may be attributed to the random placement of spores on the host (not the total number per host tree), such that only a small proportion land at times and in places where they can cause infection. The sum of the probabilities of infection of all the infection courts that have existed in the life of a tree will equal the expected infection rate of that tree. The probability of infection of individual courts, though variable, is always very small, and hence the number of courts is much larger than the expected number of infections. Under these conditions, the Poisson distribution approximates the probability of having zero, one, two, or some other whole number of galls on a tree with a particular expected infection rate. Hence, a tree with a particular number of infections should be seen as a sample of one, taken from a hypothetical population of trees with a particular but unknown expected infection rate.

Thus, in this paper, the degree of infection on a tree is described by three related terms. The relative susceptibility of a tree is defined as the expected number of infections on that tree at unit effective dosage. The expected infection rate (EIR) of a tree is the expected number of infections at a particular effective dosage. Finally, the actual number of infections observed is interpreted to be a function of the expected infection rate and of the random placement of spores, the effect of which can be approximated by the Poisson distribution. In the case of gall rust, and presumably similar diseases in which the number of discrete infections per host plant is rather low, there is a source of variation in infections per tree which is neither genetic nor environmental in the normal sense of these terms. The Poisson process presumes that individual galls develop independently and are a function only of the tree EIR. For instance, a tree with an EIR of 1.5 galls per tree (i.e., a tree for which the sum of all the probabilities of infection of individual infection courts equals 1.5) has a probability of 0.223, 0.335, 0.251, and 0.191 of having 0, 1, 2, and >2 galls, respectively. At low EIRs, the variation introduced by the random placement of spores is considerable compared to other sources of variation (the variance of a Poisson distribution is equal to its mean), so that the number of galls on a tree gives only a very rough estimate of the EIR of that tree.

For this study we assume that the distribution of susceptibility in the lodgepole pine population is a unimodal finite distribution. Various reports of the distribution of gall rust infection in various host populations (12,14, Fig. 1 in this study) all show unimodal distributions. Furthermore, virtually all trees become infected if the dosage is high enough (3, and the natural stand described in this paper). Thus, there is no ground for dividing the pine population into distinct groups of resistant and susceptible trees.

The binomial distribution is used as a discrete approximation of what is presumably an essentially continuous distribution of relative susceptibility. Using the binomial corresponds to dividing the range of susceptibility into $N+1$ classes. This does not imply that there are N genes involved, or that different genes have the same effect. Similarly, the binomial parameter p does not imply that all genes involved have a frequency of p . Rather, N and p together describe the shape of the distribution that results when many genes, each with a particular efficacy and frequency, act and interact together. Thus, the binomial is used to describe the proportion of trees in each of the $N+1$ susceptibility classes. The choice of the binomial limits the possible shapes that the model of the distribution of relative susceptibility might have. The binomial model implies that the probability of an individual tree belonging to the i th susceptibility class (classes are indexed from zero to N) is:

$$b_i(N, p) = \binom{N}{i} p^i (1-p)^{N-i} \quad (1)$$

where $b_i(N, p)$ is the probability of a tree belonging to the i th susceptibility class and N and p are the binomial parameters.

However, the binomial distribution does not specify the relative susceptibility of trees in consecutive susceptibility classes. Two alternative assumptions are explored in this paper. The first assumption is that there is a constant increase in susceptibility

and, hence, a constant increase in the EIR from one class to the next. This assumption generates what we refer to as the additive model which has the form:

$$S_{i+1} = S_i + B \quad (2)$$

where S_i is the susceptibility of the i th class and B is the difference in susceptibility between classes.

The second assumption is that there is a proportional increase in susceptibility from class to class. This generates what we refer to as the multiplicative model which has the form:

$$S_{i+1} = BS_i \quad (3)$$

where S_i is as before and B in this case is the proportional increase from class to class. Thus, the two models may be written as:

$$S_i = S_0 + iB \quad (4a)$$

$$S_i = S_0 B^i \quad (4b)$$

where S_i is the susceptibility of the i th class, S_0 the susceptibility of the 0th class (in both cases the most resistant class), and B the increment parameter.

The additive model implies that each resistance mechanism works independently of the others and, if present, reduces the EIR of a tree by a particular amount that is proportional to the effective dosage. It has the logical difficulty that with certain parameters it may predict that there are more resistance mechanisms than are required to reduce infection to zero. A prediction based on this model, thus, might give a negative number of infections. In this study, we applied the constraint that S_0 cannot be less than zero.

The multiplicative model implies that each resistance mechanism reduces the EIR by a certain proportion. Thus, a tree that carries a certain resistance mechanism will have an EIR that is lower, by a percentage characteristic of that mechanism, than a tree similar in all respects except that it does not have that resistance mechanism. The predicted EIR is never reduced to zero, and the logical difficulty in the additive model is not encountered. The multiplicative model would arise if resistance mechanisms act in sequence, such that each, if present, reduces, by a percentage characteristic of that mechanism, the number of successful penetrations that have circumvented or overcome resistance mechanisms that act earlier in the penetration process. For instance, two separate, rate-reducing resistance mechanisms might involve the cuticle and the epidermis, respectively. The multiplicative model would be appropriate if the epidermal mechanism reduced the number of penetrations that made it through the cuticle by a fixed percentage, irrespective of whether the cuticle resistance mechanism was functioning or not.

We have assumed that each tree in a block receives a block-specific effective dosage. The susceptibility of each class (S_i) is expressed as the expected number of infections per tree in that class at unit dosage. The EIR for a tree belonging to the i th susceptibility class and experiencing an effective dosage D is therefore DS_i . Thus, the distribution of galls per tree for trees belonging to the i th susceptibility class is described by the following Poisson process:

$$f(X|S_i, D) = \frac{e^{-(DS_i)} (DS_i)^x}{x!} \quad (5)$$

where $f(X|S_i, D)$ is the probability of X galls on a tree, given that the tree's susceptibility is S_i and the tree is experiencing an effective dosage D .

For trees within a block, the distribution of galls per tree is generated by combining equations 1 and 5:

$$g(X|D) = \sum_{i=0}^N b_i(N, p) \times f(X|S_i, D) \quad (6)$$

where $g(X|D)$ is the probability that a tree in a block has X

galls, given that the effective dosage experienced by trees in the block is D . The rest of the terms are as defined above. Hence $g(X|D)$ is also the predicted proportion of trees in a block having X galls.

Equation 6 represents a set of equations, one equation for each block of trees. Equation 6 is a function of the binomial parameters N and p , the block-specific dosage levels D , and $N+1$ susceptibility levels represented by S_i . However, the susceptibility levels S_i may be generated as a function of the S_o and B (equation 4). It can further be shown (Appendix) that the effective block dosage levels may be estimated as the average number of galls per tree in the block and that S_o may be expressed as a function of B . Thus, the system of equations represented by equation 6 is a function of only N , p , and B . Recognizing that the binomial distribution was only intended to provide a numerical approximation to a continuous distribution, we have arbitrarily fixed N to be 20.

Following the approach outlined in (1), equation 6 may be used to generate the expected number of trees in a block that would have a particular range of galls per tree. For example, if the k th range of galls per tree represents trees with galls in the interval ranging from a_k galls per tree to b_k galls per tree, then:

$$E_{jk} = T_j \sum_{X=a_k}^{b_k} g(X|D_j) \quad (7)$$

where E_{jk} is the expected number of trees in block j that have a number of galls per tree in the K th interval range, T_j is the total number of trees observed in block j , and D_j is the effective block dosage. (D_j is estimated as the average number of galls per tree in the block.) In this analysis we used eight gall ranges as follows: 0, 1, 2, 3, 4-5, 6-7, 8-10, and >10 galls per tree. In a few blocks, gall ranges that had an expected number of trees less than one were combined. Highest range classes were

combined with the preceding range until the expected number of trees exceeded one.

The expected number of trees in each gall range in each block of trees was compared to the observed number of trees in each gall range in each block to generate a chi-square test for the parameters to be estimated:

$$\chi_v^2(p, B) = \sum_j \sum_k \left[\frac{T_{jk} - E_{jk}}{E_{jk}} \right]^2 \quad (8)$$

where T_{jk} is the observed number of trees with galls in range k in block j . The minimum chi-square method consists of finding values for the parameters p and B which minimize equation 8. The degrees of freedom (df) represent the total number of classes (combined classes count as one) minus the number of parameters (= 2) minus two times the number of blocks. In each block, the total number of galls and the total number of trees are used in estimating the expected block frequencies.

Using the average block infection rates as estimates of effective block dosage rates (Appendix) and fixing N at 20, equation 8 is a function of two parameters: p and B . Optimum values for p and B may be determined using a nonlinear estimation technique to minimize chi-square (1). Alternatively, a combination of a range of values for p and B may be explored to identify the optimum combination.

RESULTS

An advantage of the minimum chi-square method is that it generates a chi-square value for each block and each range of gall frequency as well as for all blocks together, thus allowing each of these to be examined separately. Table 1 contains the analysis for five of the 20 blocks and all the gall frequency ranges. The selected blocks span the observed range of infection levels

TABLE 1. Observed and predicted frequency of trees by western gall rust disease severity classes for five of 20 blocks of lodgepole pine originating from the same seed lot

| N^a (inf) ^b | Model | Disease severity classes (no. galls per tree) | | | | | | | | Chi-square |
|---|-------------------|--|------|------|------|------|------|------|-------------------|------------|
| | | 0 | 1 | 2 | 3 | 4-5 | 6-7 | 8-10 | >10 | |
| 309 (6.07) | obs ^c | 85 | 56 | 25 | 18 | 18 | 22 | 21 | 64 | |
| | mult ^d | 108.7 | 48.6 | 29.0 | 19.1 | 23.0 | 17.5 | 20.0 | 43 | 19.40 |
| | add ^e | 81 | 64.1 | 25.5 | 7.2 | 5.7 | 12.4 | 33.8 | 79.2 | 59.42 |
| 121 (4.67) | obs | 54 | 21 | 6 | 6 | 9 | 8 | 2 | 15 | |
| | mult | 47.7 | 19.8 | 11.2 | 7.1 | 9.5 | 6.8 | 4.8 | 14.0 | 5.40 |
| | add | 38.1 | 23.3 | 7.4 | 2.5 | 5.7 | 10.0 | 15.1 | 18.9 | 26.68 |
| 102 (2.53) | obs | 59 | 15 | 5 | 2 | 7 | 3 | 1 | 10 | |
| | mult | 50.7 | 17.5 | 9.0 | 5.7 | 6.3 | 2.9 | 3.5 | 6.4 | 9.70 |
| | add | 42.8 | 15.8 | 6.3 | 6.1 | 12.9 | 8.7 | 6.0 | 3.4 | 32.61 |
| 117 (1.07) | obs | 80 | 14 | 4 | 6 | 8 | 1 | 2 | 2 | |
| | mult | 75.3 | 19.1 | 8.1 | 4.3 | 4.8 | 2.2 | 1.1 | 2.1 | 7.90 |
| | add | 65.0 | 20.1 | 12.6 | 8.6 | 7.8 | 2.2 | 0.6* | 0.1* ^f | 13.61 |
| 107 (0.58) | obs | 79 | 18 | 3 | 2 | 2 | 1 | 2 | 0 | |
| | mult | 78.9 | 15.4 | 5.6 | 2.7 | 2.3 | 0.9 | 0.8 | 0.3* | 2.55 |
| | add | 71.3 | 19.8 | 9.4 | 4.1 | 2.2 | 0.3* | 0.0* | 0.0* | 9.00 |
| Gall range specific total chi-square values | | | | | | | | | | |
| mult | | 14 | 26.8 | 19.9 | 18.5 | 18.4 | 14.2 | 24.6 | 16.4 | |
| n^g | | 20 | 20 | 20 | 20 | 20 | 20 | 20 | 17 | |
| add | | 82.7 | 40.8 | 48.7 | 70.4 | 79.7 | 42.5 | 86.0 | 55.0 | |
| n | | 20 | 20 | 20 | 20 | 20 | 15 | 11 | 8 | |

^aNumber of trees per block.

^bAverage number of galls per tree in the block.

^cObserved values.

^dPredicted values by the multiplicative model.

^ePredicted values by the additive model.

^fPredicted values marked with * have been added to the next lower class for the purpose of chi-square calculation, with a corresponding loss of degrees of freedom.

^gThe number of terms of which these total chi-square values are comprised.

(block average galls per tree). Table 2 summarizes the results for all 20 blocks. The overall chi-squared value is the sum of all the block-level chi-square values. The overall df are the total of the block-level df minus 2 (for p and B).

The additive model. The additive model yielded a minimum overall chi-square value of 474.7 with 93 df ($\chi^2_{0.0005}$ with 93 df = 145). An examination of the contribution of each block to the overall chi-square value (Table 2) reveals that 14 of the 20 blocks had a contribution greater than expected ($\alpha = 0.01$). The df in determining block-specific expected chi-square values was taken to be the number of gall range classes used in the calculation minus 2. This represents an approximation in that the parameters p and B are ignored, and assumes that when considering a specific block, the values of p and B are essentially determined by the 19 remaining blocks. If the additive model were an appropriate description of the pathosystem, one would expect 50% of the block-specific contributions to the overall chi-square value to be less than the $\alpha = 0.5$ chi-square value. Only one block out of the 20 had a value less than the $\alpha = 0.5$ chi-square value.

At the parameters that yield the minimum chi-square, the additive model underestimated the number of uninfected trees for all 20 blocks and the number of trees with >10 galls for all but the three most heavily infected blocks. In these three heavily infected blocks, the model underestimated most of the intermediate infection ranges, while in the 17 remaining blocks the number of trees with intermediate numbers of galls was almost always overestimated.

If the parameters of the additive model are estimated from single blocks, the minimum chi-square values are somewhat reduced, even taking the loss of two additional df (for p and B) into account. However, in that case the values for p and B vary considerably from block to block. Hence, the main failing of the additive model is its inability to predict the distribution of infection at various dosage rates simultaneously.

The multiplicative model. The multiplicative model had an overall minimum chi-square value of 152.8 with 115 df ($\chi^2_{0.01}$ with 115 df = 153.0). There was no obvious bias with respect to dosage level or tree infection level. The model overestimated the number of uninfected trees in seven of the blocks and underestimated it in 13. At the >10 infections-per-tree level, the model overestimated 11 of the blocks (seven of eight blocks with >2.0 galls per tree were underestimated in this gall range; in these eight blocks 167 trees had >10 galls versus a prediction of 134 trees). Similarly, at the 2 or 3 galls-per-tree level, the model overestimated eight of the 20 blocks, but with only one overestimation in the 11 blocks with an average of >1.35 galls per tree. The model predicted 150 trees with 2 or 3 galls in these 11 blocks compared with an observed value of 169 trees. The distribution of block-specific chi-square values followed a chi-square distribution (Table 2). For instance, nine of the 20 blocks had a chi-square value less than the 50% chi-square expected value.

Figure 2 shows the proportion of trees in each susceptibility

TABLE 2. A comparison of the ability of the additive and multiplicative models to predict the distribution of western gall rust infection in 20 blocks of lodgepole pine originating from a single seed lot

| | Additive | Multiplicative |
|--|----------|------------------------|
| Minimum total chi-square | 474.70 | 152.80 |
| Total df | 93 | 115 |
| Chi-square _{0.01} | 127.60 | 153.00 |
| Parameter estimate | | |
| p | 0.027 | 0.824 |
| B (unit load) | 1.62 | 3.21 |
| S_p (unit load) | 0.130 | 9.85×10^{-10} |
| No. blocks (out of 20) with a calculated chi-square (3-6 df) | | |
| < chi-square _{0.50} | 1 | 9 |
| < chi-square _{0.25} | 2 | 13 |
| < chi-square _{0.05} | 4 | 17 |
| < chi-square _{0.01} | 6 | 19 |

class as predicted by the multiplicative model. Only 10 out of 21 classes are shown. Trees with EIRs of less than .0004 galls per tree at unit dosage constitute less than 0.1% of the pine population. The predicted EIR of a tree in a particular resistance class is calculated as the block dosage (the average number of galls per tree in the block) \times the relative susceptibility of the class. The prediction of the distribution of disease severity in a particular block is calculated by estimating, for each class, the proportion of trees with 0, 1, ..., 10, and >10 galls per tree as predicted by the Poisson distribution for the EIR of the class, multiplying these proportions by the number of trees in that class, and summing the products across all classes.

To test the effect of the arbitrary choice of $N = 20$, the values of p and B at minimum chi-square were also determined with N equal to 3, 5, 10, 15, and 25. These values as well as the minimum chi-squares associated with them are presented in Table 3. As N decreases, the total minimum chi-square increases. This is interpreted to mean that the distribution of relative susceptibility is indeed a continuous unimodal distribution. As N decreases, p also decreases, resulting, in all but the case of $N = 3$, in a distribution with a similar negative skew. Also, as N decreases, B increases, because with fewer classes the difference between successive classes must increase to accommodate the total range of relative susceptibility.

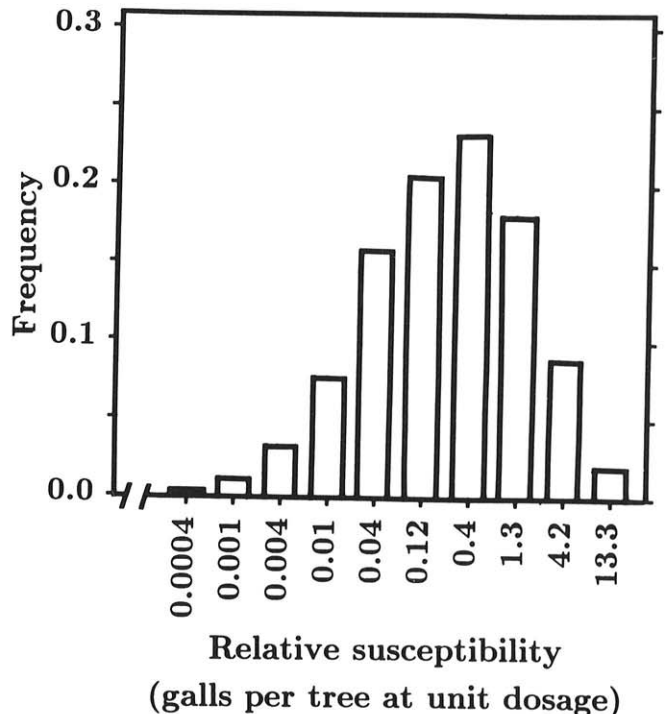


Fig. 2. The frequency distribution of trees by relative susceptibility class (logarithmic scale) as predicted by the multiplicative model. The values for the susceptibility classes are infection rates predicted when the average number of infections per tree for the population is one (unit dosage). The 11 least susceptible classes are not shown since they represent a negligible proportion of the host population.

TABLE 3. The effect of the binomial parameter N on the optimum value of the parameters of the multiplicative model and the minimum total chi-square value

| N^2 | p | B | Chi-square | df |
|-------|-------|-------|------------|-----|
| 3 | 0.470 | 5.780 | 315.1 | 104 |
| 5 | 0.633 | 5.082 | 231.5 | 104 |
| 10 | 0.668 | 3.487 | 165.6 | 115 |
| 15 | 0.770 | 3.302 | 156.3 | 115 |
| 20 | 0.824 | 3.210 | 152.8 | 115 |
| 25 | 0.857 | 3.157 | 151.0 | 115 |

²Number of classes = $N + 1$.

Other sources of variation. The above models imply that all of the variation in number of galls per tree within a block is a function of variability in susceptibility and the Poisson process. The effective dosage (the integration of inoculum load and environmental factors) is assumed to be the same for each tree within a block. This assumption was examined by testing the hypothesis that trees with large numbers of galls occur in clumps. Spatially clumped distributions would be expected if heavily infected trees increased the inoculum load in their immediate vicinity. Also, if local microsite factors varied significantly within blocks, one would expect clumps of more heavily infected trees in those sections of each block in which the environment was most favorable for infection.

The hypothesis was tested by dividing the trees in each block into three classes. The first class consisted of all trees that had more than four \times the average number of infections in the block and at least 10 galls, or the ten most heavily infected trees, whichever yielded the smallest number of trees. Such trees were present in 17 of the 20 blocks. The second class consisted of all trees immediately adjacent to these heavily infected trees. The last class consisted of the remaining trees. The average number of infections per tree in the second class was 0.08 greater than that in the last class (range: 1.01 to -0.70 for the 17 blocks). A one-tailed paired Student's *t* test yielded $t = 0.73$ ($t_{0.05}$ (16 df) = 1.75). This result is interpreted to mean that variation in effective dosage within blocks was small and masked by the variation in susceptibility and the variation introduced by the Poisson process. Hence, variation in effective dosage within blocks was apparently not a significant component of the variation in number of galls per tree within blocks. The stands surrounding the three experimental areas were older and much more heavily infected than those comprising the 20 blocks. Presumably, most of the inoculum came from surrounding trees. Nevertheless, the oldest infections in the blocks were at least 8 years old, and therefore old enough to have given rise to two generations of detectable galls. Many of the heavily infected trees had such old infections. The failure to detect higher infection rates in the immediate vicinity of such trees presumably means that dispersal gradients from such trees were so shallow that they could not be detected.

A second source of variation to be considered is variation in tree size. Within heavily infected trees, there is a direct relationship between the amount of tissue available for infection (total internode length) and the number of galls (20). Tree size data were not collected in the 20 blocks of the planting density study. In the natural stand, the height, dbh, and age of each tree were recorded. An index of crown volume was estimated from these as $\text{dbh}^2 \times \text{height}$. Correlation between number of infections per tree and height, dbh, crown volume, and age yielded $r = -0.06$, -0.028 , -0.054 , and -0.001 , respectively ($r_{0.05}$ [368 df] = 0.102). Tree size or age (duration of exposure) were not significant factors in the variation of number of infections per tree.

Crown architecture varied considerably between trees and might well represent a significant source of variation, both through its effect on the efficiency with which spores are trapped and deposited on stem surfaces and through its effect on within-crown microclimate. However, crown architecture is thought to be largely genetically determined and therefore represents one of the ways in which genetically determined susceptibility is expressed.

DISCUSSION

The description of model development and parameterization is presented as if the lodgepole pine-western gall rust pathosystem were essentially nonspecific. The results are certainly compatible with that assumption, and other studies (13,16) lend some support to it. However, the study described in this paper was not designed to determine whether the pine-gall rust system is largely specific or nonspecific. All trees were exposed to a mixed natural inoculum. Under such conditions, specific host parasite interactions are largely masked. It may be that part of the apparent variation in infection is attributable to autoinfection of some trees by rust

races which are particularly well adapted to them, resulting in severe infection on such trees.

The main purpose of this study was to search for a hypothetical distribution of the relative susceptibility of a lodgepole pine population exposed to a natural, mixed western gall rust inoculum. The distribution in Figure 2 can also be viewed as a distribution of relative disease severity, the challenge being to explain that distribution in terms of host and parasite genetics as well as environmental factors.

It is, however, possible to examine and test a consequence that would follow if specific resistance played a significant role in this pathosystem. One would then expect that some trees, which were fairly resistant to the general rust population, would experience marked increases in their tree-specific infection rate once rust races to which they were particularly susceptible became established on them. In blocks with a low overall infection level, there would have been little opportunity for such autoinfection. On the other hand, in the more heavily infected blocks, many of the more heavily infected trees might represent the result of such autoinfection. Under such circumstances, one would expect considerably more variation in relative disease severity (as measured by the value of *B* in the two models) in the more heavily infected blocks. The test, then, consisted of developing two distributions of relative susceptibility such as shown in Figure 2, one based on the 10 most heavily infected, and the other on the 10 least infected blocks. The former had block infection levels ranging from 1.35 to 6.07 infections per tree with 9.3 percent of the trees having more than 10 infections per tree, and the most heavily infected tree having 129 galls. Average infection in the latter ranged from 0.58 to 1.22 galls per tree with 0.8 percent of the trees having more than 10 infections per tree, the most heavily infected tree having 31 galls.

The test was conducted for both the additive and the multiplicative model. Using the additive model, the values of *B* were 1.59 ($P = 0.027$, total minimum $\chi^2 = 355.69$ with 57 df) for the heavily infected blocks, and 2.31 ($P = 0.019$, total minimum $\chi^2 = 76.438$ with 34 df) for the lightly infected blocks. Using the multiplicative model, the values of *B* were 3.24 ($P = 0.825$, total $\chi^2 = 94.9$ with 58 df) for the more heavily infected blocks, and 3.58 ($P = 0.850$, total $\chi^2 = 53.7$ with 52 df) for the 10 less infected blocks. In both models, the variation in relative susceptibility was slightly greater for the blocks with the lower infection levels. Hence, the results of this test do not support the interpretation of significant autoinfection at the average infection levels encountered in these blocks. The natural stand discussed below, which was much more heavily infected, did appear to show evidence of such autoinfection.

The same test also serves to eliminate another interpretation. The test for clumping of infection around heavily infected trees failed to show disease gradients around such trees. It is possible, however, that such gradients do exist within one or a few meters of individual galls. Such short-distance gradients might not be detected by considering whole tree infection levels, since the distance between individual trees varied from 1.5 to 4.2 m in the 20 blocks. If such short-distance gradients existed, they would increase the dosage to which individual trees, which happened to be infected early, would be exposed, thus violating the assumption of uniform effective dosage within blocks. Applying the same arguments that are presented above, such a phenomenon would lead to greater variation in the apparent relative susceptibility in heavily infected than in lightly infected blocks. This was not found, and hence the interpretation of significant short-distance dosage gradients is rejected.

The purpose of this study was to search for a distribution of relative susceptibility to the mixed natural inoculum that could predict the distribution of disease severity for a range of average infection levels. The additive model did not yield such a distribution. It was unable to predict the observed number of gall-free trees, trees with intermediate levels of infection, and heavily infected trees simultaneously. The common implicit assumption in the literature (4,7), that the susceptibility of an individual as measured by the number of infections on that individual is

inversely proportional to the number of minor resistance genes, appears to be inappropriate for the lodgepole pine-western gall rust pathosystem. On the other hand, the multiplicative model was able to predict most of the variation in gall frequency both within and between blocks of trees. Apart from a slight but consistent underestimation of the number of trees with >10 galls in the more heavily infected blocks, there was no apparent systematic bias in the fit of the multiplicative model to the observed distributions over the range of gall frequencies or the range of block infection levels. Thus, the distribution of relative susceptibility generated by the multiplicative model meets the objectives of this study.

The range of susceptibility predicted by the multiplicative model is very large. The most susceptible 10% of the trees are predicted to have an average EIR about 800× as high as the least susceptible 10%. Given this wide range in susceptibility, one can expect to find marked differences in the number of galls on adjacent trees. It is not necessary to invoke the hypothesis of major race-specific resistance and virulence genes to explain the wide range in numbers of galls per tree.

The wide range in susceptibility results in a significant number of gall-free trees, even in blocks experiencing a high dosage level. Table 4 compares the predicted distribution of trees by gall rust infection classes for the natural stand with the observed. The predicted values are based on the parameters p and B derived for the multiplicative model from the 20 blocks. The average infection rate in this natural stand was 17.6× higher than the overall average for the 20 blocks. The model anticipates the relatively large number of gall-free trees in this heavily infected stand, but appears to overestimate the number of lightly infected trees and underestimate the number of severely infected trees. This shift of trees from the lightly to the more heavily infected classes may be interpreted as evidence of some host specificity or short-distance inoculum gradients, leading to excessive auto-infection in some trees in this heavily infected stand. If the additive model is used to predict the distribution of infection in the natural stand, the total chi-square value exceeds 10,000 (3 df). It predicts that <1% of the trees will remain uninfected, compared with a prediction of 12.2% by the multiplicative model, and an observed value of 14.9%.

The curvilinear relationship between the percent of trees infected and the stand dosage level as predicted by the multiplicative model is consistent with the data set examined in this paper (Figure 3). It is also consistent with the curvilinear relationship between inoculum load and percent of lodgepole pine seedlings infected by western gall rust reported by Blenis and Pinnell (3). They observed 62.1, 78.1, 90.2, 97.8, and 99.5 percent infected for inoculum loads of 2, 7, 20, 60, and 180 mg of aeciospores, respectively. Assuming that the average number of infections per seedling is directly related to inoculum load at low infection levels (22) (the interest here is only in the seedlings that remain uninfected), the best fit of the multiplicative model occurs when an inoculum load of 1 mg of spores results in 2.7 infections per seedling. With this average infection rate per mg of inoculum, and using the parameters p and B as determined from the 20 blocks, the multiplicative model predicts infection levels of 63.0, 80.4, 90.1, 95.7, and 98.4 percent, respectively, for the reported inoculum levels. Although the relationship between average number of galls per tree and percent infected as predicted by the additive model was also curvilinear, that model predicted a much faster rise in percent infected with increasing average

infection level than the multiplicative model. The additive model was unable to generate the results reported by (3).

Figure 3 reflects an anticipated relationship between incidence (the percentage of trees infected) and severity (the average number of infections per tree). This relationship can be used to predict severity from incidence data, particularly at low infection levels. The multiplicative model, therefore, provides a conceptual basis for the relationship between incidence and severity, rather than the normal approach of using regression methods (17).

In Figure 2, the scale of the independent variable is logarithmic. The multiplicative relationship implies that the number of minor susceptibility genes is proportional to the log of the infection rate (assuming that all minor susceptibility genes have the same effect). Hence, in Figure 2 the independent variable may also be seen as a linear scale proportional to the number of susceptibility genes. The figure suggests that there is a slight negative skew in the pine population with respect to number of susceptibility genes. If resistance is the result of a very large number of minor genes, each with a particular frequency and efficacy, and acting independently in an additive fashion, then the central limit theorem predicts that the distribution of susceptibility should approach normality. Similarly, if resistance genes interact in a multiplicative fashion, the log of the susceptibility should approach normality. In either case, selection for resistance will increase the average resistance of the population, but the resistance genes in each new generation of trees should be randomly distributed over trees and, hence, have a near normal distribution. Thus, we expected that the distribution of relative susceptibility should be approximately normal. The binomial used in this paper could be thought of as a discrete bounded approximation to the normal distribution. The log of the resistance classes represents a linear scale so the distribution approximates a log normal distribution. It is in this sense that we refer to the distribution as a log binomial distribution. The distribution can be forced to be symmetric by setting p at 0.5. With this restriction, the minimum chi-square method can be used to estimate the only remaining parameter, B . The minimum total chi-square value with this restriction is 210.8 (115 df). Fourteen of the 20 blocks still have chi-square values less than the corresponding block chi-square ($\alpha = 0.05$). The perceived deviation from normality is not particularly significant. It may be an artifact of the estimation

TABLE 4. Predicted and observed frequency of trees in western gall rust disease severity classes in a natural stand of lodgepole pine (370 trees; average 39.9 infections per tree)

| | No. infections per tree | | | | | Chi-square |
|-----------|-------------------------|-------|------|-------|-------|------------|
| | 0 | 1-5 | 6-15 | 16-40 | >40 | |
| Predicted | 45.3 | 102.1 | 69.8 | 49.5 | 103.3 | 50.93 |
| Observed | 55 | 50 | 64 | 80 | 121 | |

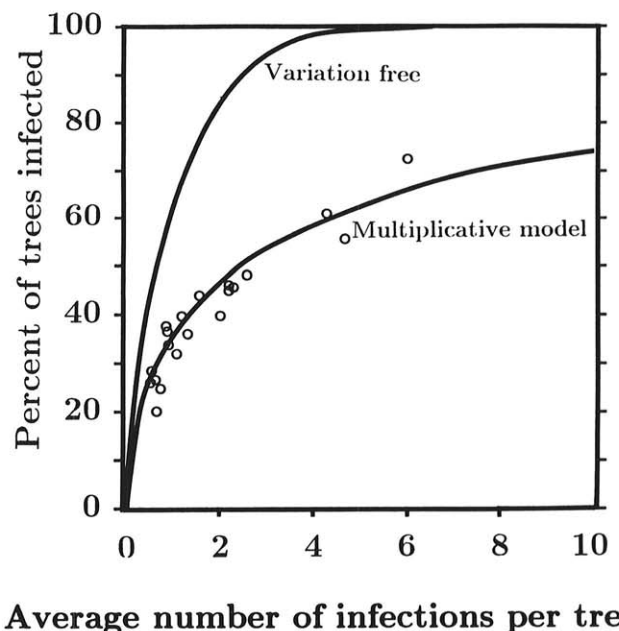


Fig. 3. Relationship between disease severity (as average number of infections per tree) and percent of trees infected by western gall rust predicted by the multiplicative model (lower line) and for the hypothetical case of no variation in resistance (upper line). The data points represent the observed values for the 20 blocks.

procedure which involved collapsing trees with large numbers of galls into a few classes. Alternatively, the slight skew may result from dominance or epistatic effects, linkage among resistance genes, or the influence of a few rather important genes. If p is set to 0.50 in the additive model (an approximation of the way in which that model is portrayed in [4] and [7]), the total chi-square value is $>10,000$. The best fit of the additive model occurred when $p = 0.027$ (with $N = 20$). Hence, the additive model predicts that 57.8% of all the trees belong to the most resistant class. The large deviation from normality in the additive model is further evidence for rejecting that model as an appropriate description of this pathosystem.

Fleming and Person (7) suggest that a multiplicative interaction between host susceptibility and pathogen aggressiveness could result in an approximately lognormal distribution of disease reaction, not unlike the one demonstrated in this paper. In deriving that distribution, however, they intrinsically assume that each host individual is exposed to only one of the pathogen races present in the pathogen population. Thus, the frequency of reaction types is calculated in the manner in which a Punnett square is used to estimate the frequency of genotypes, modified to allow for the relative frequency of the various host and pathogen types. In this paper we describe a situation in which each host individual is exposed to the whole range of pathogen races present in the local population. If that condition is applied to the multiplicative interaction model in (7), a normal distribution of disease reaction results. Hence, we do not believe that a multiplicative interaction between susceptibility and aggressiveness as described in (7) can explain the lognormal distribution of disease reaction demonstrated in this paper.

One advantage of the models presented in this paper is that they allow quantitative predictions. Thus, a consequence of the wide variation in susceptibility may be displayed by comparing the expected number of gall-free trees as predicted by the multiplicative model with the number of gall-free trees as predicted by a model free of genetic variation. The comparison, shown in Figure 3, spans a range of effective dosage levels (average number of galls per tree). The variation-free model assumes that all trees in the stand will generate galls according to the Poisson process at the effective dosage rate, and is calculated from the Poisson probability of zero galls. The difference between the two

curves is the consequence of the variation in susceptibility in the pine population. At higher dosage levels, a few trees will have a large number of galls while many trees will remain gall free. It follows that variation in susceptibility results in a buffering of the effect of environmental factors and inoculum load on the proportion of the population infected. For instance, a change in environmental conditions and/or inoculum load that results in a doubling of the average infection rate from 5 to 10 infections per tree is predicted to increase the percentage of trees infected from 61.8 to 72.3.

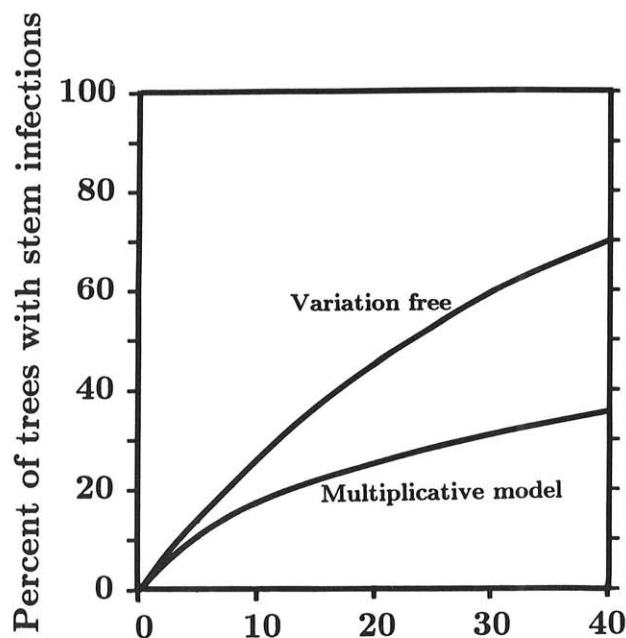
Extending the above argument, it can be shown that the wider the range of susceptibility (while holding the mean level of susceptibility constant), the less the potential mortality induced by heavy rust infestations. We will assume that tree death due to western gall rust is the result of bole infection. However, the percentage of the total number of infections located on the bole varies considerably between stands, and decreases with tree size and age. As crown size increases, the proportion of the total crown represented by the leader and the base of the upper laterals, the only infection sites which result in stem galls, decreases. Also, infection decreases with height above ground (19). In the 20 blocks of the main study area, 5.1 percent of all infections occurred on boles. In the older, natural stand, 1.2 percent of the infections on living trees were bole infections, and some trees with stem infections had died. As a conservative estimate, we assume that 3 percent of all infections are stem infections. Figure 4 shows the predicted proportion of trees with stem infections as a function of average stand infection level. That proportion was calculated by estimating the proportion of trees with 1, 2, ..., 55, and >55 infections per tree for a given stand infection level. The probability that a tree with n infections has at least one stem infection is one minus the probability that all n galls are not on the main stem ($1 - 0.97^n$). As a comparison, the expected percentage of trees with stem infections, if there was no variation in resistance, is also shown. At low levels of infection, the estimated percent of trees with stem infections is nearly the same in both cases. At higher average infection levels, however, the two curves diverge. This divergence is the result of the greater frequency of multiple stem infections on the more susceptible trees. The total number of stem infections at a particular stand average infection level is always the same for both cases. Figure 4 shows that even at substantial infection levels the survival of a predominant pine canopy is not seriously threatened.

The above implies that even at high dosage levels the host will survive. At the other extreme, low effective dosage, the high variability in susceptibility implies that there will always be a few very susceptible trees that will allow the parasite to persist. Thus, the host and parasite coexist and survive over a wide range of infection severity, a condition predicted by Robinson (15) for natural horizontal pathosystems.

Discussion of pathosystem stability usually revolves around the mechanisms of inheritance and selection that allow both the host and the pathogen to survive over many generations. In this system, it may be that the wide range in resistance of the host, coupled with what appears to be a narrower range in pathogenicity, means that selection for resistance proceeds about as fast as selection for pathogenicity, in spite of the fact that the pathogen goes through something like 25 generations for each generation of the host. In this context, we believe that the distribution of susceptibility in the pine population represents a steady state that is the result of a long period of selection for resistance and virulence. Artificial selection of the most resistant trees may result in pine populations with a narrower range of susceptibility.

The term stability is also used to refer to resistance to change in response to environmental variation. The buffering of the effect of environmental factors and inoculum load on the percent of trees infected, and, more importantly, on the percent of trees with stem galls, that is attributable to the variation in susceptibility, introduces stability of that kind into the pathosystem.

In summary, we have identified a source of substantial variation in number of infections per tree that is neither genetic nor environmental in the normal sense of these terms, but arises from



Average number of infections per tree

Fig. 4. Predicted relationship (multiplicative model) between disease severity and percent mortality (i.e., percent of trees with one or more stem infections) (lower line) and, for the hypothetical case of no variation in resistance (upper line).

the random deposition of spores on trees. Two models of the mode of action of resistance genes were examined. Only the multiplicative model gave an adequate description of the distribution of western gall rust within and between stands. The biological basis for the multiplicative model, namely that resistance mechanisms act sequentially, is appealing. Several independent lines of evidence supported the multiplicative model. Only the multiplicative model gave a reasonable prediction of the distribution of disease in a heavily infected natural stand, and of the percent of seedlings infected at various inoculum dosages in an artificial inoculation experiment. Finally, only the multiplicative model yielded the expected near-normal distribution of susceptibility genes. While the additive model could be parameterized to give a reasonable fit for some of the 20 blocks, it was insufficient to describe all blocks simultaneously, and it performed very poorly when extended to the natural stand or the inoculation experiment.

The models in this paper have been presented as having a genetic basis with the assumption that the observed number of galls is related to the inherited susceptibility of trees. We have not provided any direct evidence for this assumption beyond rejecting local site factors, local inoculum loads, and tree size factors as significant sources of variation within blocks. The genetic arguments are supported only in that the generated model performs well in accounting for the distributions of galls per tree. There may be other models which give equally good or better predictions.

APPENDIX

Equation 6 represents the probability that a tree in a block has X galls, given a block effective dosage of D . The expected or average number of galls per tree in the block is:

$$E(X) = \sum_{X=0}^{\infty} X g_X(X|D) \quad (9)$$

$$= \sum_{X=0}^{\infty} \left[X \sum_{i=0}^N b_i(N,p) \times f(X|S_i,D) \right] \quad (10)$$

$$= \sum_{i=0}^N \left[b_i(N,p) \sum_{X=0}^{\infty} X \times f(X|S_i,D) \right] \quad (11)$$

where $E(X)$ represents the expected value of X , and equation 11 represents a rearrangement of the summation order of equation 10 which is an expansion of equation 9. The second summation in equation 11 corresponds to the expected value of a Poisson process and is DS_i . Thus equation 9 may be rewritten as:

$$E(X) = \sum_{i=0}^N b_i(N,p) DS_i \quad (12)$$

$$= D \sum_{i=0}^N S_i b_i(N,p) \quad (13)$$

Equation 13 for a block receiving a unit dosage ($D = 1$) indicates that the expected value of the susceptibility (S) must be equal to 1:

$$E(S) = \sum_{i=0}^N S_i b_i(N,p) = 1 \quad (14)$$

where $E(S)$ is the expected value of susceptibility in the population.

Rewriting equation 14 with S_i expanded to represent the additive model (equation 4a) yields:

$$\begin{aligned} \sum_{i=0}^N (S_0 + iB) b_i(N,p) &= E(S_0) + BE(i) \\ &= S_0 + BNp = 1 \end{aligned} \quad (15)$$

where $E(S_0)$ and $E(i)$ are expectations of S_0 and i for a binomial

distribution. Equation 15 may be rearranged to show:

$$S_0 = 1 - BNp \quad (16)$$

Rewriting equation 14 with S_i expanded to represent the multiplicative model (equation 4b) yields:

$$S_0 = \sum_{i=0}^N B^i b_i(N,p) \quad (17)$$

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