Letter to the Editor

Response to Comments by Peet and Hunt on the van der Kamp-Tait Susceptibility Model for Resistance Selection

Bart J. van der Kamp

Department of Forest Sciences, University of British Columbia, 270-2357 Main Mall, Vancouver, BC, Canada V6T 1Z4.
Accepted for publication 17 July 1996.

In 1990, van der Kamp and Tait (5) presented a model that accounted for the observed variation in disease severity of western gall rust (*Endocronartium harknessii* (J.P. Moore) Hirat.) on lodgepole pine (*Pinus contorta* Douglas) in terms of the variation in relative susceptibility of pine to the rust. Later, van der Kamp (4) used this model to explore the limits to selection for resistance when the number of discrete infections per plant is low. Peet and Hunt (3) recently questioned some of the arguments as well as the practical significance of the results of van der Kamp (4). In responding to Peet and Hunt's comments I want to address three issues raised by them. These concern the meaning of "perfect knowledge" as used in van der Kamp (4); the magnitude of the loss of resistance arising from the lack of perfect knowledge; and the manner in which the model of van der Kamp and Tait (5) can be used to compare host populations. Finally, I also want to correct an error in the appendix of van der Kamp and Tait (5) called to my attention by F. Peet.

As part of their discussion of breeding for resistance in forest trees, Carson and Carson (1) produced a figure (their Fig. 2) that illustrates selection of the most resistant trees from a population. In that figure, the selected trees are all more resistant than any tree not selected. Similar illustrations of selection can be found in Falconer (2). In van der Kamp (4), I argue that in pathosystems in which the number of discrete infections per host plant is small, selection in this manner is not possible. Examples of such pathosystems include all the Cronartium blister rusts, most cases of bole cankers of trees, and, in fact, almost any pathosystem in which pathogen propagules are distributed randomly, if they are examined during the early stages of epidemics when the number of lesions per plant is still low. Although in theory the phenomenon holds for all pathosystems, the effect becomes more marked as the number of infections per plant declines.

Starting from the model of distribution of susceptibility to western gall rust in a lodgepole pine population developed by van der Kamp and Tait (5), which assigns trees to susceptibility classes, I show that the more resistant classes contain both infected and disease-free trees. I then calculate the average susceptibility of uninfected trees as a function of average disease severity in the population and compare that to the average susceptibility that would be obtained if an identical number of trees were selected with "perfect knowledge." Selection with perfect knowledge is defined as selection such that all trees chosen are more resistant than any tree not chosen and is implied by Figure 2 in Carson and Carson (1). In the context of van der Kamp and Tait (5), perfect knowledge requires that the susceptibility class to which each tree belongs is known. I concluded that the ability to identify resistant trees is significantly compromised by the random nature of infection when the number of infections per tree is low.

In their comment, Peet and Hunt (3) begin by describing in some detail the methodology of van der Kamp and Tait (5), leading to the development of Figure 1 in Peet and Hunt (3), which is identical to Figure 2 in van der Kamp and Tait (5), except that susceptibility classes are numbered instead of identified by their relative susceptibility. Then, following the procedures in van der Kamp (4), they derive their Table 1 (3). Their table provides the information described in Figure 1 in van der Kamp (4).

At this point, the argument of Peet and Hunt (3) diverges from that of van der Kamp (4). I argue that selection of resistant individuals, such as that portrayed by Carson and Carson (1) or Falconer (2), requires perfect knowledge. In the case of pathosystems in which the number of discrete infections per individual is small, perfect knowledge is not attainable. Thus, in van der Kamp (4, Fig. 3) I compare the average relative susceptibility of disease-free individuals with that of an equal number of individuals selected with perfect knowledge. Using the parameters derived for the gall rust-lodgepole pine pathosystem in van der Kamp and Tait (5) and an average disease severity of five galls per tree, this yields an average relative susceptibility of 0.084 for selection of disease-free trees and 0.050 for selection with perfect knowledge (4, Fig. 3).

Peet and Hunt (3) appear to misunderstand the use of perfect knowledge in van der Kamp (4), which leads them to calculate average susceptibility of various sets of uninfected trees selected with perfect knowledge based on their susceptibility class membership (3, Fig. 2). Given their assumptions, their calculations are correct, but they have no bearing on the arguments of van der Kamp (4).

The main argument of Peet and Hunt (3) is that selection is not significantly compromised, because in either case the relative susceptibility of selected trees is very small compared to the population mean (unity). For instance, for the pine-gall rust pathosystem as described by van der Kamp and Tait (5) and under conditions in which the average number of galls per tree is five, the difference in average infection severity between trees selected with perfect knowledge and as disease-free trees is only 0.17 galls per tree (5(0.084 – 0.050); van der Kamp [4], Fig. 3).

I will respond to this central point in two ways. First, the usual purpose of selection is to obtain a new population of resistant trees. The average resistance of such a population is a function of the resistance of the parents and the heritability of that resistance. Therefore, I would argue that the relevant comparison is between the expected disease severity of the respective F_1 populations, which will likely be considerably higher than that of the selected parents. Assuming that heritability is the same, whether the parents are disease-free individuals or they are selected with perfect knowledge, the expected disease severity of the F_1 population derived from parents selected as disease-free individuals can be expressed as a percentage of the F_1 population obtained with selection by perfect knowledge. In van der Kamp (4) Figure 3, this is approximately 170% (0.084/0.050), and it remains nearly constant

Corresponding author: B. J. van der Kamp
E-mail address: bart.john.van.der.kamp@mtsg.ubc.ca

Publication no. P-1996-0903-01O

over disease severity. I chose to call this "significantly compromised," and of course, one might debate how significant such a difference in susceptibility might be.

A second response to the main concern of Peet and Hunt (3) is to note that in the particular example used, that of western gall rust on lodgepole pine as described by van der Kamp and Tait (5), the range in susceptibility in the wild population is very large. In that example, the most susceptible 5% of the population is predicted to have >2,000 times as many galls per tree as the most resistant 5%. Wild conifer populations are among the most variable plant populations found in nature. In many agricultural crop systems, variability in susceptibility is generally much smaller, because such systems usually consist of inbred lines or even clones. In van der Kamp and Tait (5), the range in susceptibility is largely described by parameter B, which is the ratio of susceptibility of successive classes. Figure 1 shows (for the case in which average disease severity is five galls per tree) the mean susceptibility of uninfected trees and the percentage of trees that remain disease-free as a function of B.

Two patterns are evident. First, as the range of susceptibility increases, the percentage of trees that remain disease-free also increases. This arises because more and more infections are concentrated on the more susceptible trees. It is universally understood that greater variability in susceptibility leads to greater gains in resistance in each selection cycle. This is partially counterbalanced by the fact that more trees remain disease-free and, hence, the ability to identify the most resistant individuals decreases as variability in susceptibility increases. Second, the mean susceptibility of uninfected trees increases with decreasing range in susceptibility. The mean susceptibility of disease-free trees is again about 170% of that of trees selected with perfect knowledge over the range of variation portrayed (results not shown). Thus, if the results of van der Kamp (4) were extended to other populations or pathosystems, the variation in susceptibility in such systems also would have to be considered, and the mean susceptibility of selected individuals (and their offspring) might lie much closer to the population mean than in the example used.

Finally, Peet and Hunt (3) point out, correctly, that the model of van der Kamp and Tait is not designed to make comparisons between population means. The term "dosage" is used by van der Kamp and Tait as an inclusive term describing the set of conditions that together results in a particular outcome, i.e., a particular average disease severity. Different samples (plots) of the same population are exposed to different dosages and, therefore, exhibit different infection severity, yet all are used simultaneously to estimate the distribution of relative susceptibility. Our methodology does not allow for the standardization of dosage in the manner suggested by Peet and Hunt (3) (i.e., "dividing the number of infections per tree by the average number of infections per tree in the population"). Rather, an iterative process is used in which a series of possible distributions of relative susceptibility (defined in terms of p and B in the case of van der Kamp and Tait [5], but in theory by any set of parameters defining a distribution) are used, in turn, to calculate a set of distributions of expected disease severity (i.e., number of trees with 0, 1, 2... galls per tree), one for each sample of the population. These distributions are compared to the actual field observations to find values for p and B that minimize χ^2. The model can be used to compare variation in susceptibility of different populations (e.g., a local population and the open-pollinated, half- and full-sib families derived from it). This is done by deriving optimum values for p and B for a set of samples (plots) of each of the populations and determining whether p or B differs significantly between populations.

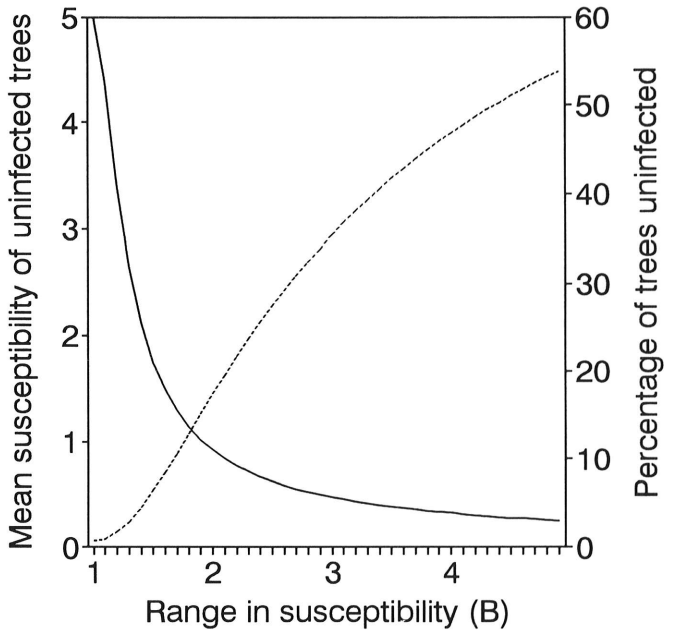

Fig. 1. Mean susceptibility (galls per tree) of uninfected trees (solid line, left axis) and percentage of trees with no galls (dotted line, right axis) when the average population disease severity is five galls per tree, as a function of variation in susceptibility (B), predicted by the model of variation in susceptibility proposed by van der Kamp and Tait (5). B is the ratio of susceptibility of adjacent classes in the model in van der Kamp and Tait (5); $B = 1$, indicating there is no variation in susceptibility.

Comparisons between population means should be made in the normal way, namely by an experimental design that ensures equal exposure (uniform dosage) to the rust and a comparison of means. The number of galls per tree is unlikely to be normally distributed. Hence, the best design will compare estimates derived from small subplots whose mean disease severity will more closely approach a normal distribution.

Finally, on a peripherally related matter, I wish to point out that the value of S_o in equation 17 in the appendix of van der Kamp and Tait (5) is incorrect and misrepresents the calculations performed in that study. The correct value is the reciprocal of the one given:

$$S_o = 1/\sum_{i=0}^{N} B^i b_i(N, p)$$

LITERATURE CITED

1. Carson, S. D., and Carson, M. J. 1989. Breeding for resistance in forest trees—A quantitative genetic approach. Annu. Rev. Phytopathol. 27:373-395.
2. Falconer, D. S. 1960. Introduction to Quantitative Genetics. Ronald Press Co., New York.
3. Peet, F. G., and Hunt, R. S. 1995 Comments on the van der Kamp-Tait susceptibility model for resistance selection. Phytopathology 85:1332-1334.
4. van der Kamp, B. J. 1993. Limits to selection for disease resistance from natural tree populations. Can. J. For. Res. 23:1944-1947.
5. 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.