GENETIC CONTROL OF RESISTANCE TO HYPOXYLON INFECTION AND CANKER DEVELOPMENT IN *POPULUS TREMULOIDES* 1/

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ABSTRACT.--The responses of 24 families of *P. tremuloides* (6 groups of 4 maternal half-sibling families each) to 4 sources of *Hypoxylon mammatum* were observed. Three mechanisms of resistance to the disease were studied: (1) callus formation, (2) branch death, and (3) resistance through retardation of canker growth. Resistance by callous formation is due to a hypersensitive response of the host to the pathogen. Little variation exists in the nature or time of the host response, but the pathogen's ability to elicit the host response, expressed as incidence per inoculum, varies from about 3 to 35 percent. Evidence suggests that a few major genes control this trait and are the basis for Mendelian ratios within families and discrete differences between groups of half-sibling families. Resistance by branch death occurs at a low incidence (8.3 percent) and death is due to the canker encircling the branch. Heritability estimates are low, 0.075 or less in response to the four inocula and 0.027 for all data. These are probably underestimates because all potentially resistant phenotypes most likely have not been expressed 4 months after inoculation. The third form of resistance, retardation of the spread of the pathogen, is measured as canker length. h^2 is low for three inocula (= 0.074), but reasonably high (0.254) for the fourth source.

The best breeding methods for disease-resistant forest trees may be ;those concerned with breeding for "nonspecific" or horizontal resistance. This type of resistance functions against all pathogenic races or biotypes and is generally considered polygenic. "Specific resistance" functions against individual races of a pathogen and is also called major gene resistance, hypersensitivity, and vertical resistance. It is controlled by single or a small number of genes. A knowledge of the genetic control of resistance is necessary if we are to design an effective tree breeding program.

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Preliminary results (Valentine and Manion 1972, Manion and Valentine 1974) suggest that resistance to Hypoxylon canker in trembling aspen (Populus tremuloides Michx.) and bigtooth aspen (P. grandidentata Michx.) is polygenic. In this report we show that much genetic variation exists in factors affecting (1) infection of the host by artificial inoculation with Hypoxylon mammatum (Wahl.) Mill. (syn. H. pruinatum (Klot.) Che), (2) prevention of the establishment by rapid death of host tissue, or (3) isolation through callousing and retarding canker enlargement.

MATERIAL AND METHODS

We used 7-year-old P. tremuloides at the Tully Genetic Field Station, Tully, New York (fig. 1). Following Comstock and Robinson's Experiment 1, each of six pistillate-flowered trees was crossed to a random sample of four staminate-flowered trees to give six maternal half-sibling groups of four families each. One-year-old seedlings were planted in 1968 in a randomized complete block design with three blocks and nine trees per family per block.

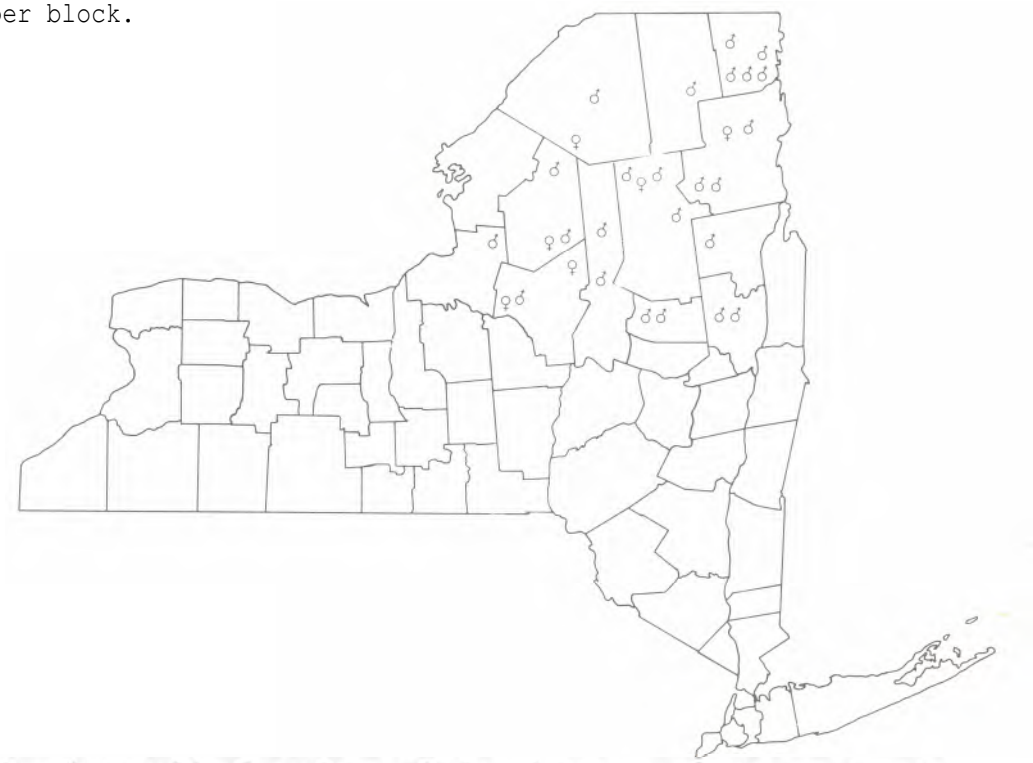

Figure 1.--Geographic locations of the six trees serving as female parents (♀) and the 24 trees as male parents (♂) in the northern New York population of P. tremuloides.

In June 1974, four 2-year-old branches, approximately 1.5 m from the ground, were inoculated with H. mammatum mycellum grown for 2 weeks at 30°C on moist sterilized wheat. Six-mm circular patches of bark were removed with a cork borer and one piece of H. mammatum-infested grain placed in each wound. The inoculations were wrapped with parafilm "M". Four pathogen sources were used: (1) a "Baldwinsville, New York" culture (French and Manion 1975, isolate 208 M), (2) a "Tully" culture originating from a single ascospore (Hsu 1975, isolate 4B19 #5 set 1), (3) a "Shiawassee County, Michigan" culture, and (4) a "Heiberg Forest" culture (French and Manion 1975, isolate 608 M). The four strains were used to inoculate branches with north, east, south, and west exposures, respectively. Some trees and branches were missing so that there were unequal numbers of inoculations in the families. Four months later the lengths of the resulting cankers were measured. Cankers surrounded or covered by callous tissues and cankers on branches that had died were noted. Cankers on broken branches (8.5 percent) were counted but not measured.

We used the method of Robertson and Lerner (1949) to estimate the heritability of resistance by callousing and by branch death (also see Dempster and Lerner 1950). The method requires the calculation of the average genetic relation (r^G) of the members of each family making up each half-sibling group. For this we used the method of Osbourne (1957).

Heritability estimates for canker length were based upon a three-way ANOVA (Veldman 1967). The methods of King and Henderson (1954) and Becker (1975) were used to calculate the coefficients for partitioning variance when there are unequal numbers of observations per family. These were then applied to the ANOVA results to estimate the covariance of maternal half-sibling groups and the phenotypic variance.

RESULTS

Resistance Through Callous Formation

Genetic factors control all three mechanisms of resistance to cankering

The results of Chi-square Tests of Homogeneity for the responses of the six half-sibling groups to each of the four inocula are statistically nonsignificant except for the Tully data, table 1, therefore shows group means only. In the Tully results, three of the six means are outside the range of the $\bar{x} \pm 2s$. Some differences were also evident in the Baldwinsville data, with two mean values outside the $\bar{x} \pm 2s$, but the probability value for the Chi-square test is greater than 0.20.

Table 1.-- Incidence of resistance by callousing among maternal half-sibling aspen families inoculated with four sources of Hypoxylon mammatum

(In percent)

Item	Source of inoculum			
	Baldwinsville, New York	Tully New York	Shiawassee County, Michigan	Heiberg Forest, New York
All families	12.6±1.4	3.3±0.8	15.7±1.5	34.9±2.0
Total number of inoculations	557	552	573	567

The heritability estimates were calculated but the values are small with large standard errors or are negative, so they have not been given. The estimate for the Tully data is the largest, but is only 0.068 and its standard error 0.067.

Male effects within maternal half-sibling groups were considered for the Heiberg data because they include the highest incidences of callousing. If segregations for one or more "major" genes for callous formation occur in the population, Mendelian ratios may be inferred from these data (table 2). The four families in each group separate into two or three different frequencies. Groups CA107, HA313, and ES107 exhibit two levels, with the lower incidence, ranging from about 20 to 30 percent, about half that of the higher incidences that range from about 40 to 50 percent. In the other three groups, one or more families occur with either a "low" or a "high" incidence of callousing, but in addition, families with a frequency intermediate to these also occur. Minor variations also can be seen among the half-sib groups, with the SL209 group lower than the others.

Resistance Through Branch Death

Responses to the Baldwinsville and Tully inocula are large, ranging from 0 to 13.4 percent for Baldwinsville and from 6.8 to 19.5 percent for the Tully isolate (table 3). In the test for homogeneity the Chi-square values are statistically significant. The variation in the other two inocula is much less, and the Chi-square results are not significant.

The mean frequency of branch death for the four inocula ranges from 4.6 percent for the Heiberg source to 11.6 percent for the Tully source. The tests for homogeneity, however, suggest that there are no real differences in the responses of the other half-sibling groups to the four pathogen sources (except for the SL209 group). The data for the four inocula for each half-sib group were then pooled and a Chi-square test used to analyze the variation in the mean incidences among the six groups. Maternal parents SL209 and LE208 appear to contribute genes

Table 2.-- Incidence of calloused cankers in response to the Heiberg inoculum in each of the four half-sibling families constituting each of the six maternal groups (percent incidence is shown by the position of the male parent tree designation along the vertical axis

Calloused cankers (percent)	Maternal groups of half-sibling families (common female parent)					
	SL209	LE208	OA107	OA201	HA313	ES107
55						
50						CL202
				HE202		
					ES111	
45						
		HA312			FU111	
	OS109	CL106				
			CL104			
40			SL112			
			HA308			
	ES201			SA113		
35		CL306				
				CL110		
30						
						HA209
	SL111	WA105			ES202	OA102
25					LE108	
			FU109	FR212		
						HE109
20						
	LE201					
15						

for resistance, especially in response to the Baldwinsville and Tully inocula, and the HA313 parent appears to lack these genes because its progeny exhibit a low incidence of branch death.

Table 3.-- Incidence and heritability of resistance by branch death among maternal half-sibling aspen families inoculated with four sources of Hypoxylon mammatum

(In percent)

Item	Source of inoculum: Baldwinsville, New York	Tully, New York	Shiawassee County, Michigan	Heiberg Forest, New York	Half-sib mean incidence	Test of homogeneity: χ^2 (d.f.=3)	Probability
Half-sib Family							
SL209	12.2±3.6	19.5±4.3	7.8	1.6	11.0±1.7	13.130	<0.01
LE208	13.4±3.8	18.9±4.1	9.8	5.0	12.4±1.9	7.193 1/	>.05
CA107	5.7±2.5	7.4±2.7	7.9	3.0	6.2±1.3	2.374 1/	>.95
CA201	8.0±2.9	8.0±2.9	8.9	8.2	8.2±1.5	0.085 1/	>.99
HA313	0	6.8±2.9	4.2	5.8	4.1±1.2	5.396 1/	>.10
ES107	7.6±3.0	9.0±2.9	7.1	4.5	7.0±1.4	1.609 1/	>.70
All families	8.0±1.2	11.6±1.4	7.7±1.2	4.6±1.1	8.3±0.6		
Test of Homogeneity value of χ^2 (d.f.=5)	11.864	15.155	1.905	4.115 1/	17.934		
Probability	<0.05	<0.01	>0.80	>0.50	<0.01		
Heritability	0.005±0.066	0.075±0.076	Negative value	Negative value	0.027±0.026		

1/ Yates adjustment was applied to these data due to expected values of 5 or less.

The variability among half-sib families could represent random sampling variation. This, however, does not appear to be the case because the callousing frequency in 20 of the 24 families is outside the range, p ± 2s (30.9 to 38.9 percent), for the Heiberg results. The three families that do occur within this range are "intermediates" in their groups, and the fourth, SL209 x ES206, is a high incidence family. The observed distribution is bimodal, with the "high" incidence families comprising one modal group and the "low" incidence families, the other.

The heritability estimates for the Baldwinsville and Tully strains are low and have large standard errors, and are negative for the Michigan and Heiberg data (table 3) .

Resistance Through Retardation of Canker Growth

The size of cankers varies with the four pathogen sources and among the six maternal half-sibling groups (tables 4, 5, and 6). The ranking of the four inocula based on canker size is very consistent. The mean is smallest for Heiberg (6.02 cm), followed by Baldwinsville (6.34 cm), Tully (7.34 cm) and Michigan (7.76 cm). With only three exceptions this ranking is also the same for the six individual half-sib groups (table 4). The differences among maternal half-sibling groups is also large and is statistically significant for all inocula except Heiberg ("Females" in tables 5 and 6).

Table 4.--Mean canker length 4 months after inoculation for each of the four sources of pathogen (each family mean for each inoculum is based on between 55 to 85 cankers)

Half-sib	Source of inoculum: Baldwinsville, New York	Tully New York	Shiawassee County Michigan	Heiberg Forest New York	Pooled data: Mean	No.
	Centimeters					
SL209	6.28	7.99	7.81	6.09	7.16	285
LE208	6.22	7.68	7.87	6.50	7.16	300
OA107	5.94	6.43	7.10	6.10	6.43	308
OA201	6.16	6.47	7.32	5.99	6.48	300
HA313	6.13	7.52	7.81	5.43	6.86	257
ES107	7.32	7.94	8.59	6.00	7.58	305
Mean	6.34	7.34	7.76	6.02	6.91	1,755
No.	443	475	457	380		

Table 5.--ANOVA for canker lengths 4 months after inoculation and the heritability estimates based on the combined data for the four sources of inoculum

Source of variation	: d.f. :	Mean square
Between families	23	22.705**
Females	5	56.565**
Males W/N females	18	13.299**
Blocks	2	16.964*
Inoculum sources	3	260.198**
Families X blocks	46	6.323
Families X inocula	69	6.723*
Blocks X inocula	6	15.965**
Families X blocks X inocula	138	4.466
Within families (error)	1,468	5.107
Weighted coefficient for variance components: t		5.92
Estimates of heritability:		
With the inocula effects included in σ^2_p		0.083
Without the inocula effects and the interactions with other main effects in σ^2_p		0.109

* Statistically significant at the 5 percent level of probability
** Statistically significant at the 1 percent level of probability

Table 6.--ANOVA results for canker lengths 4 months after inoculation and estimates of heritabilities for each of the inoculum sources

Source of variation	d.f.	Source of inoculum (mean square): Baldwinsville, New York	Tully, New York	Shiawassee County, Michigan	Heiberg Forest, New York
Blocks	2	21.162**	12.629	24.001*	7.217
Females	5	16.206**	36.002**	17.891*	6.485
Males W/N females	18	9.313**	7.969	15.474**	2.666
F-M X Blocks	46	5.398	5.661	5.719	3.306
Within families d.f.		371	403	385	308
m.s.		4.521	5.061	6.824	4.357
Weighted coefficients of variance components:					
k_1		18.168	19.513	18.840	15.611
k_2		19.274	20.597	19.604	16.461
k_3		73.574	78.993	75.967	63.159
Estimates of heritability		0.074±0.119	0.254±0.224	0.015±0.090	0.058±0.062

* Statistically significant at the 5 percent level of probability
** Statistically significant at the 1 percent level of probability

Other significant results include block effects, the interaction between blocks and inocula, and the interaction between families and inocula. Because the variation in canker sizes associated with each pathogen source is similar in each of the half-sib groups, it is not likely that a Female and Inoculum interaction is an important part of the Families X Inocula interaction.

We used the weighted coefficients of the variance components to determine the heritability of canker length (bottom tables 5 and 6) (King and Henderson 1954, Becker 1975). The difference between the two heritability estimates in table 5, $h^2 = 0.083$ and $h^2 = 0.109$, is the consequence of including the variance components due to differences in inocula sources and its interactions only in the first estimated phenotypic variance. All of the heritability values are small except for the Tully inoculum (table 6) which is 0.254. All also have large standard errors.

DISCUSSION

Three mechanisms confer resistance to hypoxylon cankering in the northern New York trembling aspen population: (1) callous formation, (2) branch death, and (3) resistance through retardation of canker growth. On the basis of individual inocula the estimates of the heritabilities for all three traits are low: 0.064 or less for callous formation, 0.076 or less for branch death, and 0.074 or less for retardation of canker growth. For the pooled data of all four inoculations on each tree, h2 are 0.027 and 0.083 for branch death and retardation of canker growth, respectively, but $h^2 = 0.109$ for canker growth retardation if variation due to inocula and its interactions with other main effects are not included in the estimate of the total phenotypic variance.

Callous formation is rapid and similar to a hypersensitive reaction, occurring before a measurable canker developed in 314 (83.5 percent) of the 376 calloused wounds or cankers. The "late" calloused cankers ranged up to 7 cm in length, with the incidence of callousing decreasing with an increase in length of the canker. The frequency distribution according to canker size when calloused, however, does not fit a Poisson distribution ($\chi 2 = \infty$, d.f. = 4., $P<0.001$). This type of early response of the host is characteristic of hypersensitive reaction, generally controlled by one or a few genes (Williams 1975).

The incidence of callousing differs markedly for the four sources of inoculum (table 1), which suggests that variation is due to the pathogen and not the host. Differences in the incidence of callousing is small and not significant (except the Tully inoculum). (Tully data are limited--only 18 calloused cankers of 552.) This suggests that the host genotypes for a gene or genes controlling the callousing response to the fungus are essentially alike. This limited variation is also responsible for the low heritability values because they are dependent upon the variance between half-sib groups.

The variations in the callousing response of each half-sib group to each of the four inocula are large and highly significant, with a high incidence exhibited by all six groups in response to the Heiberg inoculum, an intermediate incidence in response to the Michigan and Baldwinsville source, and a low incidence in response to the Tully inoculum. The greatest variation, therefore, exists in the pathogenicity of the fungus, with very little variation in the host reaction to a particular pathogen phenotype (table 1).

The sizes of calloused cankers also suggest that the host response to the pathogen is the same regardless of the source of the inoculum. The frequencies of "early" (calloused canker length = 2 cm) and "late" (>3 cm) callousing were tested for homogeneity using the Chi-square method. The "late" cankers in the 3 to 7 cm length classes were pooled. The results are nonsignificant ($X2 = 7.402$, d.f. = 3; $0.10> P> 0.05$). The Chi-square

value is large and primarily represents the contribution of the limited data for the Tully inoculum (11 "early" and 7 "late" cankers). If we include only the data for the other three inocula, the variation is small ($X^2 = 1.159$, d.f. = 2, $P > 0.50$). Therefore, the host response to the pathogen is the same regardless of the overall incidence of the callousing response, with the frequencies of "early" and "late" responses 84.6 percent and 15.4 percent, respectively.

The results support the hypothesis that callous formation is controlled by a few gene loci that are "turned on" in host tissue by the presence of pathogen genotypes with a given level of virulence. Clear-cut Mendelian ratios will not necessarily be observed, however, because this appears to be a threshold-type response. Nevertheless, sharp, distinct differences in the frequencies of callousing among the four families composing a given half-sib group would be expected. The two or three different frequencies in response to the Heiberg inoculum support this (table 2). The 11 families comprising the high incidence level of callousing in the 6 groups may represent the expectations from a testcross, namely a 1:1 Mendelian ratio. The nine families exhibiting the low level could represent an incidence of 25 percent callousing, the expectation if callousing is a recessive trait and the parents are both heterozygous. We tested the goodness of fit of the observed results to that expected for each of the 11 "high frequency" and the 9 "low frequency" families if they represent expected Mendelian ratios of 1:1 and 1:3, respectively, (calloused to noncalloused); all were nonsignificant. We also compared the observed incidence for each family to the range $p \pm 2s$, for each of the two Mendelian expectations. In all cases, the observed incidence was within the expected range. These results clearly support the hypothesis that callousing is due to a single gene in the "high" and "low" incidence families.

The intermediate frequencies exhibited by the four families in the SL209, LE208, and OA201 groups, cannot be explained as easily. It appears that resistance by callousing is not always a simple Mendelian trait, but rather, it is controlled by a more complex genetic system. Nevertheless, sharp, distinct differences that recur among related families are most easily explained by the segregation of major genes contributed by the common parent, in this case the female parent. Confirmation of this hypothesis must await further experimentation.

We expected heritability estimates to be high; instead, they were low, This is because the estimates were not based upon individuals or even family means for estimating additive genetic variance, but upon the variation among half-sibling group means around the population mean. The group mean, in turn, represented the average incidence in the four half-sibling families comprising that group. Because variation among mean values was low, heritability values were low.

Resistance by death of the infected branch does not appear to be a hypersensitive host reaction, but rather the response resulting from the spread of the canker to girdle the branch. The response is not as rapid as callousing. Only 13 (8.4 percent) of the 155 branches that died, did so before a measurable canker developed, i.e., greater than 2 cm in length. The incidence is so low that little information on the size of cankers at the time of branch death or the frequency distribution can be gleaned from the individual family data, the half-sibling group data in response to each inoculum source, or even the pooled data for each half-sib group in response to all four inocula. Data pooled for all half-sib groups for each inoculum or for all data approximate the normal distribution, with the modal class almost always including the average length of all living cankers (table 4). More branches were being girdled when these results were obtained so later field scoring should provide information on how long after infection this type of resistance can act to prevent disease development.

The variation in branch death among the six half-sibling family groups in response to the Baldwinsville and Tully inocula is statistically significant. There is little variation in response to the other two sources. Correspondingly, the largest h2 values, 0.075 and 0.055, were obtained in response to Tully and Baldwinsville sources, with negative values for both the Michigan and Heiberg sources. The incidence in response to the Heiberg source was very low, only 17 among 369 cankered branches. Therefore, even though the Yates' adjustment was applied, the results may not be reliable.

Though small, differences probably do exist among pathogen sources (table 3). This is supported by the pattern in the frequencies of branch death in response to the four inoculum sources by each of the half-sibling groups. The lowest incidence in all groups except one, HA313, is in response to the Heiberg source. The incidences in response to Baldwinsville and Michigan are generally intermediate; and to the Tully source, the highest for four of the six half-sib groups. The lack of statistically significant results in the Chi-square Tests of Homogeneity for all but SL209 probably is a consequence of three factors: (1) the low incidence of this form of resistance, (2) a small variation in response, and (3) the numbers of cankered branches in each half-sib group for each pathogen source. Though these numbers range from 52 to 100, they are not large enough to show a difference among inoculum sources. The numbers for the branch death data are smaller than for callousing because calloused cankers are not considered candidates for branch death. This also accounts for the lowest numbers of cankers for the Heiberg data because that inoculum resulted in the highest callousing frequencies. Another point regarding pathogen variation should be made: if the pathogen variation is real, the heritability estimate based upon the pooled data (table 3) would be meaningless.

Although not as convincing as the callousing data, the branch death results appear to support the hypothesis that this is a polygenic control system with a low heritability. If the trait is controlled by many loci, a small amount of variation in incidence of branch death is expected among the four half-sib families. Sharp differences, as were found in the callousing results, would not be expected. Branch death in resistant trees generally occurs in all families with the exceptions almost entirely in either the HA313 families in response to all four inocula or among the families in all six half-sib groups in response to the Heiberg inoculum. In these cases, however, the incidences of branch death are low and the absence of branch death is probably due to chance variation. The highest frequencies of branch death, such as the SL209 and LE208 in response to the Baldwinsville and Tully inocula, are evenly scattered among the four half-sib families, but they do exhibit small variations. The heritability estimates obtained in this study are probably underestimates of the true values because it is not likely that all branches had died when these observations were made at 4 months. Results from further field observations should resolve this point.

The differences in canker lengths 4 months after inoculation appear to represent true differences among the six groups of maternal half-sibling families (tables 4, 5, and 6). If short canker length can be equated to resistance by the host and long cankers to susceptibility, the HA303 groups appear the most resistant, the OA107 and 0A201 groups range from intermediate to resistant, SL209 and LE208 are generally intermediate but tend toward susceptibility and ES107 is the most susceptible. This order of ranking is similar for the pooled data for each maternal half-sib group and for the mean values for each inoculum. There is little doubt that this trait is controlled by a number of genes that exhibit a small amount of additive genetic variance. It exhibits general combining ability, but the experiment gives no information on specific combining ability. The heritability estimates suggest that slow gains could be made in the host's ability to retard spread of the pathogen by a selection program.

The variation in canker length associated with inoculum sources reflects real differences and appears to be a predictable response of the host to each inoculum (table 5). Apparently the Michigan and Tully inocula exhibit greater virulence than the other two pathogen sources. The four inocula rank from most virulent Michigan, Tully next, Baldwinsville third, and Heiberg the least virulent (table 4). The repeatability of their levels of virulence suggests that the differences are genetic. No information is available on the genetic control system.

Again, pathogen virulence is not related to resistance heritability. The Michigan inoculum exhibits the greatest virulence and results in cankers with considerable variation in size, but the heritability estimate for canker length is the smallest. Most of the variation occurs in the males within females sources of variation, with a smaller part due to females. This, combined with the large within families mean square, is the cause of the low heritability value.

As in the case of callous formation and branch death, the largest heritability for canker length ($h^2 = 0.254$) was obtained with the Tully inoculum. The most plausible explanation for the greater predictability probably is the genetic uniformity of this inoculum, which was derived from a single ascospore. The other three inocula, in contrast, were derived from mycelia extracted from diseased bark. Greater variation could occur in each of these cultures because not only could a heterokaryotic condition be present with nuclei of two or more origins, but two or more genetically different mycelia originating from multiple infections are also possible. If the pathogen as well as the host varies, the predictability of the host response would be less and heritability low. If this hypothesis is correct, the heritability estimates from the Tully data would most clearly reflect the host's ability to respond to the pathogen and would be the most reliable estimates of the predictability of the host responses. Further experiments are planned to test this hypothesis. If this is correct, one would expect more rapid improvement in resistance than our results suggest now.

The slow spread of the disease in the host is frequently referred to as a tolerance reaction of the host. If slow enough, the pathogen presumably could persist for years in a rapidly growing tree without killing the host because trees die when their stems are girdled. Selection for tolerant genotypes is feasible in an aspen breeding program, and is highly desirable because this mode of resistance would not exert strong selection pressure on the pathogen for more virulent forms. This might be especially useful in breeding trees for short rotation croppings (Einspahr 1972).

The tolerance reaction depends upon a slow growth of the fungus whereas fast canker growth is required in the branch death response because the branch has to be killed before the fungus can reach the main stem. Therefore, the characters would be expected to be negatively correlated: groups with short cankers should exhibit a low frequency of branch death, and vice versa. A comparison of mean canker length and incidence of branch death in maternal half-sib groups (tables 3 and 4), however, does not reveal any obvious relations. Rank correlations (Snedecor 1946) were also calculated, based upon maternal half-sib group means and the rank of the 24 families. Though negative correlation coefficients were obtained for the Baldwinsville and Tully results for maternal half-sib group means for the Baldwinsville, Tully, and Michigan results for the family data, none of the correlations are statistically significant. If a negative relation does exist, it may have been obscured by the effect of branch diameter (at the site of the inoculation) on the earliness of branch death. This will be taken into consideration in future studies to test this hypothesis.

Although this study considered only three mechanisms of resistance, there are undoubtedly more. Artificial inoculations with a mass of mycelial inoculum precludes genetic evaluation of resistance mechanisms affecting spore germination and initial infection. Future studies will

attempt to determine the genetic control of resistance to natural infections and the correlation of these results with that from artificial inoculations.

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