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COMPARISONS OF TRAITS OF AGGRESSIVENESS IN SEXUAL AND ASEXUAL POPULATIONS OF PUCCINIA CORONATA

Iowa State University PH.D. 1983

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Comparisons of traits of aggressiveness in sexual and asexual populations of *Puccinia coronata*

by

James Harold Oard

A Dissertation Submitted to the
Graduate Faculty in Partial Fulfillment of the
Requirements for the Degree of
DOCTOR OF PHILOSOPHY

Department: Plant Pathology,
Seed and Weed Sciences
Major: Plant Pathology

Approved:

Signature was redacted for privacy.

In Charge of Major Work

Signature was redacted for privacy.

For the Major Department

Signature was redacted for privacy.

For the Graduate College

Iowa State University
Ames, Iowa

1983

TABLE OF CONTENTS

INTRODUCTION

The origin and maintenance of variation in pathogen populations has been a constant if unknowing concern to plant pathologists. This concern is based on the potential advantages of gene recombination at the population or "group selection" level, as originally proposed by Fisher (16) and Muller (30). In this model, the benefits of different gene combinations apply particularly to characters that exhibit simple Mendelian inheritance in small or finite populations (15, 18, 27). However, the advantage of recombination for individual loci has been reported in certain pathogen populations of large or infinite size (39, 41). Whether or not this advantage will occur under the effects of multiple loci, linkage, changing selection pressures, or particular generation structures is not clear (27, 46).

I have, therefore, attempted to test the potential advantages of recombination for certain traits of aggressiveness in a Minnesota population of Puccinia coronata Cda. var. avenae Fraser and Led. where genes are recombined on the alternate host, Rhamnus cathartica. The Minnesota population was compared to an asexual Texas population in field and greenhouse studies in order to obtain the following objectives:

(i) Determine the role of gene recombination for traits of aggressiveness in a sexual population of P. coronata.

(ii) Estimate and compare the level of genetic control or heritability between the sexual and asexual populations of P. coronata.

(iii) Estimate the number of effective factors for traits of aggressiveness in the sexual population.

LITERATURE REVIEW

Theoretical Considerations

Introduction

In a survey of the current literature, Felsenstein (15) wrote that "any review...of the evolution of recombination must necessarily be vague and impressionistic." This conclusion is borne out in the complexities that surround the role of recombination in natural populations. Therefore, two basic concepts are briefly presented here that play central roles in the adaptive strategies of mating systems. Free genetic recombination can be considered the result of genes segregating and assorting in a random fashion during meiosis. In contrast, the nonrandom association of genes that results from epistatic (nonadditive) interactions or the effects of genetic drift has been described as "linkage disequilibrium" by Lewontin and Kojima (21). The lack of interaction among loci is then defined as "linkage equilibrium." The consequences of gene distribution will vary depending upon the particular characteristics of a population and which components of natural selection are operating. The challenge has been, therefore, to interpret the interplay among gene recombination, linkage disequilibrium, natural selection, and other factors.

The consequences of gene recombination

Fisher (16) proposed without proof over 50 years ago that gene recombination would confer an evolutionary advantage by accelerating the accumulation of favorable alleles in a population over that of a

population that reproduces asexually. Since that time, several models have been developed which attempt to verify or refute the conclusions of Fisher (16) and Muller (30). Felsenstein (15) has proposed that the consequences of gene recombination described in the various models are dependent primarily upon population size. Accordingly, a principal advantage of gene recombination in finite populations (i.e., populations smaller than the reciprocal of the mutation rate) lies in the fact that chance events will generate random linkage disequilibrium (15, 18). This means that selection of individual genes in different genetic backgrounds will increase the variance of offspring number and the amount of genetic drift which accompanies selection. The result is that the response to selection at one locus is dependent upon changes in gene frequences at other loci. Gene recombination would have the effect of reestablishing a random association among the loci and the response to selection would increase over that of a population in linkage disequilibrium.

Muller (31) proposed an additional advantage of gene recombination in finite populations. If we assume a haploid asexual population will accumulate slightly deleterious mutations over time, there is no individual that can arise with less than the current minimum number of harmful substitutions. That is, natural selection can never reduce the number of unfavorable mutants below a fixed level in an asexual population. In addition, the minimum number of harmful mutations in asexual individuals may increase over time if the number of highly adapted individuals is small. This is contrasted with the ability of two individuals to produce

through gene recombination some offspring with fewer than the current minimum number of deleterious mutations. Gene recombination in finite populations can, therefore, be considered as a mechanism to retard the accumation of harmful alleles, but these beneficial effects of gene reshuffling would not be felt in large populations (15).

Two models developed by Crow and Kimura (8) and Maynard-Smith (25) appear to contradict Felsenstein's effect of population size. The advantage of gene recombination would occur in populations of infinite size, small selective advantages per locus, and large numbers of favorable segregating loci as predicted by Crow and Kimura. The computer simulations by Maynard-Smith suggested that an increased rate of adaptation with gene recombination would occur in large populations with many favorable segregating loci.

In natural populations, the offspring of a single parent will compete with one another when food resources become limiting in a single environment. Competition among genetically identical asexual offspring is greater in this situation than among offspring produced by sexual reproduction according to Williams (46). Sib competition under these conditions was considered to provide an immediate advantage for gene recombination. Computer simulations by Maynard Smith (26) suggested, however, that if more than one locus with no heterosis is associated with a single adaptive environmental feature (e.g., the genotype AABB is associated with adaptation to high temperature and aabb to low temperature), then gene recombination will provide no advantage under sib competition.

Maynard Smith (25) used this conclusion to challenge the widely held belief that high levels of recombination are favored in a variable environment. The model predicts that even when the environment is variable, the associations between environmental "states" (e.g. hot associated with dry or cold with wet) must change signs from one generation to the next (hot now associated with wet or cold with dry) for selection to favor gene recombination. That is, a certain combination of genes of a genotype in one generation must be poorly adapted in the next for gene recombination to be favored over asexual reproduction. Similar conclusions were reached in a more general model by Charlesworth (4).

The models presented up to now have attempted to specify the conditions under which gene recombination is favored over asexual reproduction, but they have failed to describe under which situations would high levels of gene recombination be favored over low levels and vice versa. Fisher (16) again set the stage to answer this question when he proposed that natural selection would favor a reduction in gene recombination (i.e. closer linkage) between two favorable interactive loci.

Mather (24) expanded this view by noting that individuals in a population have the dual problem of maintaining fitness in both stable and fluctuating environments. Given the assumption that natural selection favors "intermediate genotypes" or heterozygotes, both problems are solved simultaneously by favoring a reduction in gene recombination (closer linkage) between factors occurring in highly "balanced" or adapted combinations with the ability to release variability by gene

recombination under changing environments.

Numerical examples from Lewontin (22) have shown that in two locus heterotic models the mean population fitness is generally greater when linkage is present than in a completely random situation. This occurs whenever linked deleterious alleles are removed from the population at a faster rate than when the loci are randomly associated. If heterosis and epistasis are present, permanent linkage disequilibrium will occur under moderate rates of recombination. Selection for favorable alleles in the repulsion phase will, therefore, favor an increase in the variance and the rate of increase in mean fitness of the population (22). Felsenstein (14) considered the effects of linkage on response to selection in four models with two additive, nonoverdominant loci under random mating. The results suggested that if coupling phase linkage is favored, the response to selection will increase under tight linkage while the reverse is true with repulsion phase linkage.

Several models have been developed to describe an adaptive mechanism for a reduction in gene recombination. Nei (32) has shown that intensity of linkage among interactive loci can fluctuate through selection of modifier genes affecting recombination frequencies. Bodmer and Parsons (3), Charlesworth (4), and Feldman (13) demonstrated that genes at a selectively neutral modifier locus that determines recombination levels at other loci will increase in frequency when there is linkage disequilibrium among the selected loci. Nei (33) proposed that the rate of change in modifier gene frequency is proportional to the degree of linkage between the modifier and selected loci.

Several theoretical models have concentrated on the effect of gene recombination and selection toward the stability and position of populations in equilibrium. Lewontin and Kojima (21) proposed that the equilibrium reached by a population is not affected by linkage if fitnesses between loci are additive. This equilibrium state will be altered, however, if linkage values are greater than the magnitude of the epistasis present. Other stable equilibria can occur under certain conditions only when linkage is tight. Kimura (19) has shown that if alleles at two loosely-linked loci maintain constant fitnesses in a large random mating population, the ratio of coupling to repulsion phases remains relatively constant when gene frequencies change slowly under natural selection. Turner (44) concluded that the various two locus equilibrium models may be inappropriate for higher order interactions. Equations involving three loci predicted that fitness can decrease as linkage becomes tighter with the result that recombination levels reach some optimum level. It was also shown that generation structure affects the outcome of the various types of epistasis. For example, loci under epistasis that create a departure from multiplicativeness do not generate linkage disequilibrium if generations overlap while the reverse is true with discrete generations when selection is strong and linkage is tight.

Experimental Evidence

Only a limited number of experiments has been conducted to specifically test the potential advantages of gene recombination. Simons

et al. (41) compared phenotypic variation at 24 virulence loci in a population of P. coronata that had undergone sexual reproduction in a nursery in Minnesota to that of an asexual population in southern Texas. Although the average number of identified virulence genes was similar in both populations, the percentage of distinct phenotypes was greater in the sexual (64.5%) than in the asexual (17.5%) population. Similar results were obtained by Roelfs and Groth (39) with a comparison of virulence phenotypes in sexual and asexual populations of Puccinia graminis f. sp. tritici.

Considerably more experiments have been conducted to determine the level of genetic control on gene recombination. Emara (9) estimated the inheritance of aggressiveness in Ustilago hordei by inoculating susceptible barley seed with dikaryons derived from 13 different mating combinations of the fungus. The infected seeds were planted in the field where aggressiveness was recorded as a percentage of smutted spikes. Significant differences for aggressiveness were detected among all dikaryons. Estimates for epistatic variance indicated low levels of linkage disequilibria while most of the genetic variation was due to additive variance. In a subsequent study by Emara and Sidhu (11), ordered tetrads from two teliospores of U. hordei were crossed in all possible combinations and the resulting 16 dikaryons were used to inoculate barley seed and determine levels of aggressiveness as in the previous study. The authors concluded that an appreciable amount of gene interaction (21.3% for combined dominance and epistatic variance) contributed to the polygenic control of aggressiveness. However, the

existence of gene interaction at more than one locus is dependent solely upon the presence or absence of epistasis and not upon the combined dominance and epistatic variances (12). Therefore, the level of gene interaction for those loci controlling aggressiveness in U. hordei in this study may be minimal or nonexistent.

The level of genetic control of infection frequency, sporulation efficiency, and lesion size in Helminthosporium maydis was studied by Hill and Nelson (17). Five isolates of H. maydis race T of diverse origin were crossed in four combinations and the resulting ascospore progeny were isolated and used individually to inoculate Texas male sterile corn seedlings. Estimated narrow-sense heritability values for infection frequency and sporulation efficiency ranged from 21-70% while the range for lesion size was 0-13%. Estimates of epistatic variance were generally low which suggested selection for random association among the loci controlling aggressiveness and presumably for high levels of recombination.

In a review of data obtained in experiments with large natural populations of Drosophila melanogaster, Langley (20) concluded that high levels of linkage disequilibrium are not selected for as predicted by Lewontin (23). Yamaguchi et al. (47) tested the effects of gene recombination on the level of linkage disequilibrium in four finite populations of D. melanogaster. Cytological and electrophoretic results from these and other populations (29) indicate a gradual decay in the level of linkage disequilibrium in the presence of gene recombination and genetic drift. Additional studies by Charlesworth and Charlesworth (5)

using D. melanogaster have indicated selection for reduced levels of recombination, but the levels did not drop to zero.

MATERIALS AND METHODS

Description of Pathogen Populations

Growth chamber and field studies were conducted to compare traits of aggressiveness between a sexually propagated and an asexually propagated population of P. coronata. Aggressiveness is defined in this study as the relative rate at which a virulent isolate produces a given amount of disease. To obtain the sexual pathogen population, 40 aecial field collections were randomly selected in 1979 from the University of Minnesota Buckthorn Nursery in cooperation with P. G. Rothman, St. Paul, Minn. Twenty of the collections were taken at random to comprise the final sample of the sexual population for the growth chamber studies. Individual leaves of Rhamnus cathartica bearing aecia were placed in petri dishes containing moistened filter paper. The dishes were then covered for 24 hr to enhance sporulation, after which aeciospores from a single aecial cluster were transferred with a sterile needle onto leaves of a susceptible oat cultivar. The plants were then held in a dew chamber for 18 hr at 25 C. The uredia, or pustules, that developed from the aecial inoculations were purified once by "single pustuling" to obtain pure isolates. Spores of each isolate were increased to permit inoculation of 24 host differential cultivars and lines of oats. Each host cultivar and line possessed a single crown rust-resistance gene (41). Infection type of each isolate-differential combination was rated susceptible, moderately susceptible, moderately resistant, or resistant according to guidelines of Stakman et al. (42). Those

isolates exhibiting susceptible or moderately susceptible infection types were classified as virulent while those with resistant or moderately resistant infection types were classified avirulent.

Except for the aecial inoculations, a similar procedure was carried out for 116 field collections originating on oats from 7 nursery sites in southern Texas in 1979. Twenty of these collections, which had been furnished by M. E. McDaniel, College Station, Texas, were taken at random to form a second sample population for the growth chamber studies. Because gene recombination on the alternate host (Rhamnus spp.) is minimal or nonexistent for P. coronata in southern Texas, the second population will be referred to as the asexual population. Five isolates were taken at random from each of the sample populations described above for field studies in 1980 and the number was increased to 15 per population in 1981.

Growth Chamber Study

The experimental design consisted of a randomized complete block design with three replications, with individual growth chambers serving as complete blocks. The expected mean squares for traits measured in the growth chamber studies are given in Table 1. One experimental unit consisted of 10 seeds of the susceptible cultivar, 'Markton', planted in a linear row in a 4-inch clay pot. When secondary leaves of the seedlings were fully expanded (approximately 14 days after planting), the primary and secondary leaves were inoculated with individual isolates, using a quantitative inoculator, at a concentration of 2 mg

Table 1. Expected mean squares for traits of aggressiveness measured in the growth chamber

Source	df	Expected mean squares
Reps	2	$\sigma^2_e + 3\sigma^2_{L/R*I/P} + 9\sigma^2_{R*I/P} + 180\sigma^2_{RP} + 360\sigma^2_R$
Popn	1	$\sigma^2_e + 3\sigma^2_{L/R*I/P} + 9\sigma^2_{R*I/P} + 180\sigma^2_{RP} + 27\sigma^2_{IP} + 540\,\sigma^2_P$
Iso(Popn)	38	$\sigma^2_e + 3\sigma^2_{L/R*I/P} + 9\sigma^2_{R*I/P} + 27\sigma^2_{I/P}$
Reps*Popn	2	$\sigma^2_e + 3\sigma^2_{L/R*I/P} + 9\sigma^2_{R*I/P} + 180\sigma^2_{R/P}$
Reps*Iso(Popn)	76	$\sigma^2_e + 3\sigma^2_{L/R*I/P} + 9\sigma^2_{R*I/P}$
Leaf(Reps*Iso)Popn	240	$\sigma^2_e + 3\sigma^2_{L/R*I/P}$
Det(Leaf/Reps*Iso)Popn	720	σ^2_e
Total	1079	

spores/1 ml 'Soltrol' oil. Only fresh spores with a minimum of 80% germination on 2% water agar were used. After inoculation, the seedlings were held in a dew chamber for 18 hr at 25C and then placed in the growth chambers as described above. An optimum environment for host and pathogen was provided by standard greenhouse fertilizer, sterilized soil, 21C $\pm$ 1C air temperature, 10,000 lux illumination, and a 14-hr daylength.

The 15 traits of aggressiveness measured in the growth chamber fell into four categories. The "visual" category consisted of the Latent Period, defined as the time in days from inoculation to first pustule eruption of the host epidermis, Telia(1) as the time from inoculation to first telia formation, and the time difference between the above traits or Telia(1)-Latent Period. The "pustule measurement" category included the length, width, and area of three randomly selected mature isolated pustules per leaf from three secondary leaves per replication (a total of 9 pustules per replication). Data were collected by detaching leaves and photographing them adjacent to a metric ruler. Slides produced from the photographs were used to project a picture of the leaves, pustules, and ruler onto a screen for estimation of pustule dimensions to the nearest 13.3 microns. Pustule area was estimated by the formula, 3.14 [Pustule length/2 x Pustule width/2] (43). A ratio of the pustule length to width was also calculated. "Pustule density" traits (number of pustules/cm^2 leaf tissue) for the primary and secondary leaves were calculated separately and in combination to determine if physiological or biochemical factors affect pustule densities for the two leaves.

To estimate values of four "spore weight" traits, spores produced by each isolate were collected every three days by suspending inoculated leaves over the mouth of a large glass funnel. The leaves were gently tapped to release spores that passed through the funnel and into a preweighed glass vial. At the end of the spore production period, the vial and spores were weighed to the nearest 0.1 mg. Data for total

pustule number and total area of inoculated leaves were also collected and used with the spore weights to generate those traits given in Table 2.

Table 2. "Spore weight" traits measured in the growth chamber studies

Trait	Method of calculation
SPOREWT/PUSTULE	$\frac{\text{Spore weight in mg}}{\text{Total pustule number}}$
SPOREWT/ TOTAL LEAF AREA	$\frac{\text{Spore weight in mg}}{\text{Leaf area in cm}^2\text{ of primary and secondary leaves}}$
SPOREWT/ PUSTULE SIZE	$\frac{\text{Spore weight in mg}}{\text{Pustule size in } \mu^2}$
SPOREWT/ TOTAL PUSTULE AREA	$\frac{\text{Spore weight in mg}}{\text{Total pustule number x Pustule size in } \mu^2}$
SPOREWT/ PUSTULE L-W RATIO	$\frac{\text{Spore weight in mg}}{\text{Ratio of pustule length to width}}$

Component analyses of variance to estimate narrow sense heritability for each trait on an isolate means basis is given in Table 3.

Table 3. Component analysis of variance for the estimation of narrow sense heritability for traits of aggressiveness measured in the growth chamber

Source	df	Expected mean squares
Iso	19	$\sigma_e^2 + 3\sigma_I^2$
Rep(Iso)	40	σ_e^2
Total	59	

Heritability estimates were calculated from the analyses in the following manner:

$$\sigma_A^2 = \sigma_I^2$$

$$\sigma_P^2 = \sigma_I^2 + 1/3\ \sigma_e^2$$

$$h^2 = \frac{\sigma_A^2}{\sigma_P^2} \times 100$$

where: σ_A^2 = additive genetic variance

σ_I^2 = variance among isolates

σ_P^2 = total phenotypic variance

σ_e^2 = environmental + non-additive variance

h^2 = narrow sense heritability.

A procedure proposed by Croft and Simchen (7) was used to estimate the number of effective factors for traits in the sexual population where:

$$K = \frac{(\text{progeny extreme differences})^2}{4\sigma_A^2}$$

and

K = number of effective factors

σ_A^2 = additive genetic variance.

Assumptions for the model are: (1) allelic pairs at each locus have an equal effect, (2) all alleles of "positive" effect are in one parent and all alleles of "negative" effect are in the other, and (3) linkage between loci is absent.

Field Study

Field plots were planted at two locations in 1980 and 1981 using a split-plot design with four replications. Studies at the Hinds Farm (trickle irrigation) and Curtiss Farm (natural rainfall) were planted 12 and 15 April, 1980, and 7 and 10 April, 1981, respectively. A corn jab-planter was used to plant individual hill plots in one square-foot spacings at 30 seeds per plot. The physical layout of the plots is shown in Figure 1. Ten randomly selected susceptible oat cultivars (see Table 4) were planted in blocks which were separated from all others by a minimum distance of 10 m. The blocks of cultivars to be inoculated were surrounded by a two-row border of the susceptible cultivar, Richland. A "disease free" block of the 10 cultivars was planted in the center of the four inoculated blocks and maintained free of rust by spraying with maneb fungicide every 5-7 days. To reduce interplot interference, the cultivar Stout, resistant to all isolates tested, was planted between the inoculated and disease-free blocks. The field lay-out, shown in Figure 1, was repeated to accommodate the total number of isolates tested.

Table 4. Oat pure line host cultivars used in 1980 and 1981 field studies

Richland	Bates
Clintford	Nodaway 70
Lang	Allen
Noble	Spear
Otee	Larry

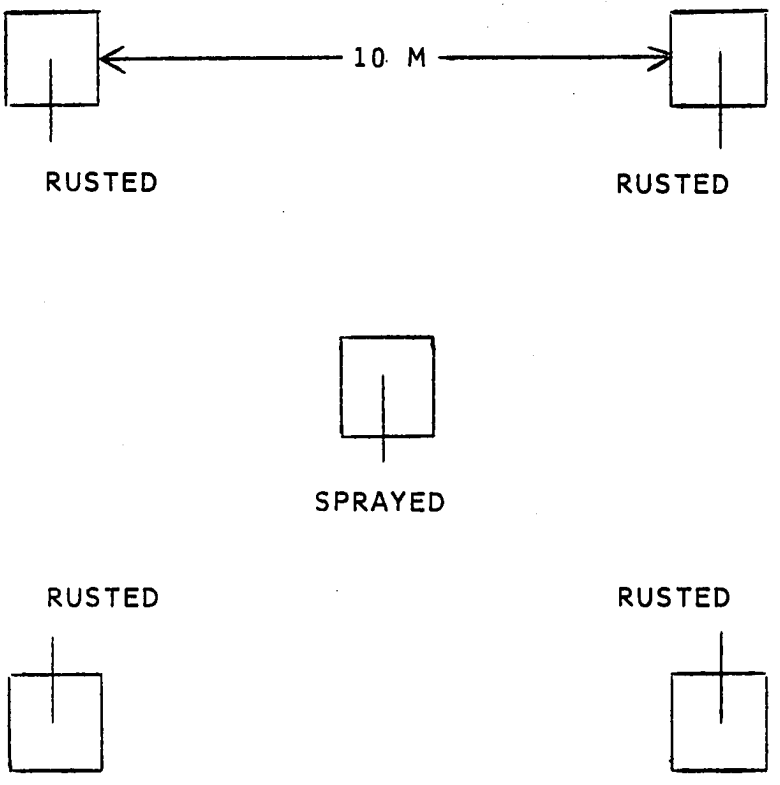

Figure 1. Physical layout of inoculated (RUSTED) and "disease-free" (SPRAYED) plots in the 1980 and 1981 field studies

When the host plants reached the mid-to-late tillering stage (approximately 20 May each year), one tiller per plot was hypodermically-inoculated with approximately 0.5 ml of an inoculum suspension of 1 mg spores per 1 ml water plus 'Tween 20'. Individual isolates were used to inoculate separate blocks of 10 cultivars. To reduce statistical bias in the data, two isolates per population were randomly assigned to the four inoculated blocks as shown in Figure 1. A mixture of equal parts of all isolates was used to inoculate a single block of 10 cultivars in 1980, while isolates from each population were mixed separately in equal proportions and used to inoculate two blocks in 1981. Those traits of aggressiveness measured in the field are defined in Table 5 and their expected mean squares are given in Tables 6 and 7. Expected mean squares for combined years and locations are given in Tables 8 and 9.

The component analyses of variance of heritability estimates for the traits in the individual and combined locations are given in Tables 10-13. Estimates of all traits for combined locations and cultivars were calculated from the analysis in Table 10 in the following manner:

$$\sigma_A^2 = \sigma_I^2$$

$$\sigma_P^2 = \sigma_I^2 + \frac{\sigma_{R(I)}^2}{4} + \frac{\sigma_{LI}^2}{2} + \frac{\sigma_{CI}^2}{5} + \frac{\sigma_{CIL}^2}{10} + \frac{\sigma_e^2}{40}$$

$$h^2 = \frac{\sigma_A^2}{\sigma_P^2} \times 100$$

where: σ_A^2 = additive genetic variance

σ_I^2 = variance among isolates

Table 5. Traits of aggressiveness measured in the 1980 and 1981 field studies

Trait	How defined
Latent Period (LP)	Time from inoculation to first pustule eruption of host epidermis
Telia(1)	Time from inoculation to first telia formation
Telia(90)	Time from inoculation to 90% telia formation
Telia(1)-LP	Time between LP and Telia(1)
Telia(90)-LP	Time between LP and Telia(90)
Telia(90)-Telia(1)	Time between Telia(1) and Telia(90)
Yield	Grain yield in grams of rusted cultivar
Yield Index	$\frac{\text{Grain yield of rusted cultivar}}{\text{Grain yield of "sprayed" cultivar}}$
Seed Weight	Weight in grams of 200 seeds of rusted cultivar
Seed Weight Index	$\frac{\text{200 seed weight of rusted cultivar}}{\text{200 seed weight of "sprayed" cultivar}}$
Coefficient of Infection (1981 only)	Percentage of infection using modified Cobb scale x Infection Type[a]

[a]The following numerical values assigned to the infection types: S = 1.0, MS = 0.8, MR = 0.5, and R = 0.3.

Table 6. Expected mean squares for traits of aggressiveness except yield and seed weight indices in the individual 1980 and 1981 field studies

Source	df	Expected mean squares
Popn	1	$\sigma_e^2 + 4\sigma_{CI/P}^2 + 48\sigma_{PC}^2 + 5\sigma_{R/I/P}^2 + 20\sigma_{I/P}^2 + \sigma_P^2$
Iso(Popn)	22	$\sigma_e^2 + 4\sigma_{CI/P}^2 + 96\sigma_C^2 + 5\sigma_{R/I/P}^2 + 20\sigma_{I/P}^2$
Reps(Iso*Popn)	72	$\sigma_e^2 + 5\sigma_{R/I/P}^2$
Cultivar	4	$\sigma_e^2 + 4\sigma_{CI/P}^2 + 48\sigma_{PC}^2 + 96\sigma_C^2$
Popn*Cv.	4	$\sigma_e^2 + 4\sigma_{CI/P}^2 + 48\sigma_{PC}^2$
Cv.*Iso(Popn)	88	$\sigma_e^2 + 4\sigma_{CI/P}^2$
Cv.*Reps(Iso*Popn)	288	σ_e^2
Total	479	

Table 7. Espected mean squares for yield and seed weight indices in the individual 1980 and 1981 field studies

Source	df	Expected mean squares
Popn	1	$\sigma_e^2 + 11\sigma_{CP}^2 + 5\sigma_{I/P}^2 + \sigma_P^2$
Iso(Popn)	20	$\sigma_e^2 + 5\sigma_{I/P}^2$
Cultivar	4	$\sigma_e^2 + 11\sigma_{CP}^2 + 22\sigma_C^2$
Cv.*Popn	4	$\sigma_e^2 + 11\sigma_{CP}^2$
Cv.*Iso(Popn)	80	σ_e^2
Total	109	

Table 8. Expected mean squares of traits of aggressiveness for combined locations and cultivars in the 1980 and 1981 field studies

Source	df	Expected mean squares
Location	1	$\sigma_e^2 + 4\sigma_{CLI/P}^2 + 8\sigma_{CI/P}^2 + 48\sigma_{CLP}^2 + 96\sigma_{CP}^2 + 96\sigma_{CL}^2 + 192\sigma_C^2 + 5\sigma_{LR/IP}^2 + 10\sigma_{R/IP}^2 + 20\sigma_{LI/P}^2 + 40\sigma_{IP}^2 + 240\sigma_{LP}^2 + 480\sigma_P^2 + 480\sigma_L^2$
Popn	1	$\sigma_e^2 + 4\sigma_{CLI/P}^2 + 8\sigma_{CIP}^2 + 48\sigma_{CLP}^2 + 96\sigma_{CP}^2 + 5\sigma_{LR/IP}^2 + 10\sigma_{R/IP}^2 + 20\sigma_{LI/P}^2 + 40\sigma_{I/P}^2 + 240\sigma_{LP}^2 + 480\sigma_P^2$
Loc*Popn	1	$\sigma_e^2 + 4\sigma_{CLI/P}^2 + 48\sigma_{CLP}^2 + 5\sigma_{LR/IP}^2 + 20\sigma_{LI/P}^2 + 240\sigma_{LP}^2$
Iso(Popn)	8	$\sigma_e^2 + 4\sigma_{CLI/P}^2 + 8\sigma_{CI/P}^2 + 5\sigma_{LR/IP}^2 + 10\sigma_{R/IP}^2 + 20\sigma_{LI/P}^2 + 20\sigma_{I/P}^2$
Loc*Iso(Popn)	8	$\sigma_e^2 + 4\sigma_{CLI/P}^2 + 5\sigma_{LR/IP}^2 + 20\sigma_{LI/P}^2$
Rep(Iso*Popn)	30	$\sigma_e^2 + 5\sigma_{LR/IP}^2 + 10\sigma_{R/IP}^2$
Loc*Rep(Iso*Popn)	30	$\sigma_e^2 + 5\sigma_{LR/IP}^2$
Cultivar	4	$\sigma_e^2 + 4\sigma_{CLI/P}^2 + 8\sigma_{CI/P}^2 + 48\sigma_{CLP}^2 + 96\sigma_{CP}^2 + 96\sigma_{CL}^2 + 192\sigma_C^2$
Cv.*Loc	4	$\sigma_e^2 + 4\sigma_{CLI/P}^2 + 48\sigma_{CLP}^2 + 96\sigma_{CL}^2$

Table 8. (Continued)

Source	df	Expected mean squares
Cv.*Popn	4	$\sigma_e^2 + 4\sigma_{CLI/P}^2 + 8\sigma_{CI/P}^2 + 48\sigma_{CLP}^2 + 96\sigma_{CP}^2$
Cv.*Loc*Popn	4	$\sigma_e^2 + 4\sigma_{CLI/P}^2 + 48\sigma_{CLP}^2$
Cv.*Iso(Popn)	32	$\sigma_e^2 + 4\sigma_{CLI/P}^2 + 8\sigma_{CI/P}^2$
Cv.*Loc*Iso(Popn)	32	$\sigma_e^2 + 4\sigma_{CLI/P}^2$
Loc*Cv.*Rep(Iso*Popn)	240	σ_e^2
Total	399	

Table 9. Expected mean squares of traits of aggressiveness for combined locations and individual cultivars in the 1980 and 1981 field studies

Location	df	Expected mean squares
Location	1	$\sigma_e^2 + 5\sigma_{LR/IP}^2 + 10\sigma_{R/IP}^2 + 20\sigma_{LI/P}^2 + 40\sigma_{IP}^2 + 240\sigma_{LP}^2 + 480\sigma_P^2 + 480\sigma_L^2$
Popn	1	$\sigma_e^2 + 5\sigma_{LR/IP}^2 + 10\sigma_{R/IP}^2 + 20\sigma_{LI/P}^2 + 40\sigma_{I/P}^2 + 240\sigma_{LP}^2 + 480\sigma_P^2$
Loc*Popn	1	$\sigma_e^2 + 5\sigma_{LR/IP}^2 + 20\sigma_{LI/P}^2 + 20\sigma_{LP}^2$
Iso(Popn)	8	$\sigma_e^2 + 5\sigma_{LR/IP}^2 + 20\sigma_{LI/P}^2 + 10\sigma_{R/IP}^2 + 40\sigma_{I/P}^2$
Loc*Iso(Popn)	8	$\sigma_e^2 + 5\sigma_{LR/IP}^2 + 20\sigma_{LI/P}^2$
Rep(Iso*Popn)	30	$\sigma_e^2 + 5\sigma_{LR/IP}^2 + 10\sigma_{R/IP}^2$
Loc*Rep(Iso*Popn)	30	$\sigma_e^2 + 5\sigma_{LR/IP}^2$
Total	79	

Table 10. Component analysis of variance of all traits of aggressiveness for combined locations and cultivars in the 1980 and 1981 field studies

Source	df	Expected mean squares
Location	1	$\sigma_e^2 + 4\sigma_{CLI}^2 + 20\sigma_{LI}^2 + 20\sigma_{LC}^2 + 100\sigma_L^2$
Cultivar	4	$\sigma_e^2 + 4\sigma_{CLI}^2 + 8\sigma_{IC}^2 + 20\sigma_{LC}^2 + 40\sigma_E^2$
Loc*Cv.	4	$\sigma_e^2 + 4\sigma_{CLI}^2 + 20\sigma_{LC}^2$
Iso	4	$\sigma_e^2 + 4\sigma_{CLI}^2 + 8\sigma_{IC}^2 + 20\sigma_{LI}^2 + 10\sigma_{R/I}^2 + 40\sigma_I^2$
Rep(Iso)	15	$\sigma_e^2 + 10\sigma_{R/I}^2$
Loc*Iso	4	$\sigma_e^2 + 4\sigma_{CLI}^2 + 20\sigma_{LI}^2$
Cv.*Iso	16	$\sigma_e^2 + 4\sigma_{CLI}^2 + 8\sigma_{IC}^2$
Cv.*Iso*Loc	16	$\sigma_e^2 + 4\sigma_{CLI}^2$
Error	135	σ_e^2
Total	199	

Table 11. Component analysis of variance of all traits of aggressiveness for combined locations and individual cultivars in the 1980 and 1981 field studies

Source	df	Expected mean squares
Location	1	$\sigma_e^2 + 4\sigma_{LI}^2 + 20\sigma_L^2$
Iso	4	$\sigma_e^2 + 4\sigma_{LI}^2 + 2\sigma_{R/I}^2 + 8\sigma_I^2$
Rep(Iso)	15	$\sigma_e^2 + 2\sigma_{R/I}^2$
Loc*Iso	4	$\sigma_e^2 + 4\sigma_{LI}^2$
Error	15	σ_e^2
Total	39	

Table 12. Component analysis of variance for "visual" traits of aggressiveness for combined cultivars in the individual 1980 and 1981 field studies

Source	df	Expected mean squares
Cultivar	4	$\sigma_e^2 + 4\sigma_{IC}^2 + 20\sigma_C^2$
Iso	4	$\sigma_e^2 + 4\sigma_{IC}^2 + 5\sigma_{R/I}^2 + 20\sigma_I^2$
Rep(Iso)	15	$\sigma_e^2 + 5\sigma_{R/I}^2$
Cv.*Iso	16	$\sigma_e^2 + 4\sigma_{IC}^2$
Error	60	σ_e^2
Total	99	

Table 13. Component analysis of variance of Yield Index and Seed Weight Index for combined cultivars in the individual 1980 and 1981 field studies

Source	df	Expected mean squares
Cultivar	4	$\sigma_e^2 + 20\sigma_C^2$
Iso	4	$\sigma_e^2 + 5\sigma_I^2$
Error	16	σ_e^2
Total	24	

σ_P^2 = total phenotypic variance

$\sigma_{R(I)}^2$ = variance of replications within isolates

σ_{LI}^2 = variance of Loc*Iso interaction

σ_{CI}^2 = variance of Cv.*Iso interaction

σ_{CIL}^2 = variance of Cv.*Iso*Loc interaction

σ_e^2 = variance of experimental error.

Estimates of all traits for combined locations and individual cultivars were calculated from the analysis in Table 11 in the following manner:

$$\sigma_A^2 = \sigma_I^2$$

$$\sigma_P^2 = \sigma_I^2 + \frac{\sigma_{R/I}^2}{4} + \frac{\sigma_{LI}^2}{2} + \frac{\sigma_e^2}{8}$$

$$h^2 = \frac{\sigma_A^2}{\sigma_P^2} \times 100$$

Symbols used are the same as the analysis in Table 10.

Estimates of "visual" traits for individual locations and combined cultivars were calculated from the analysis in Table 12 in the following manner:

$$\sigma_A^2 = \sigma_I^2$$

$$\sigma_P^2 = \sigma_I^2 + \frac{\sigma_{R/I}^2}{4} + \frac{\sigma_{CI}^2}{5} + \frac{\sigma_e^2}{20}$$

$$h^2 = \frac{\sigma_A^2}{\sigma_P^2} \times 100$$

Symbols used are the same as the analysis in Table 10.

Estimates of Yield Index and Seed Weight Index for individual locations and combined cultivars were calculated from the analysis in Table 13 in the following manner:

$$\sigma_A^2 = \sigma_I^2$$

$$\sigma_P^2 = \sigma_I^2 + \frac{\sigma_e^2}{5}$$

Symbols used are the same as before. Estimates of "visual" traits for individual locations and cultivars were calculated as in the previous analysis above. A lack of proper error terms prevented calculations of heritability estimates of Yield Index and Seed Weight Index for individual locations and cultivars.

RESULTS

Growth Chamber Study

The analyses of variance for "Pustule Dimension" traits are given in Tables 14-17. Significant differences between populations were detected for Pustule Length, Pustule Area, and Pustule L-W Ratio. Highly significant differences were found within populations for all measured traits. The interactions of Rep*Iso(Popn) and Leaf(Rep*Iso/Popn) were also significant in each analysis which would suggest that Popn and Iso(Popn) should not be considered as independent sources of variation. However, I believe this is not the case due to the relatively large number of degrees of freedom for the interactions and the relatively smaller sizes of their mean squares and F values compared to those of Popn and Iso(Popn).

No differences were detected at the population level for any of the "Pustule Density" traits shown in Tables 18-20. Highly significant differences were observed for all traits within populations and the Rep*Iso(Popn) term was again significant for each trait. The analyses of variance for "Spore weight" traits are given in Tables 21-25. Sporewt/Pustule and Sporewt/Total Pustule Area exhibited significant differences between populations while the same trend of variation within populations continued for these five traits. The number of degrees of freedom for Iso(Popn) was not constant for the different analyses due to missing data for some isolates.

The "visual assessment" traits exhibited discrete data and, therefore, a "log-linear analysis for categorical variables" (2) was used with

Table 14. Analysis of variance for "Pustule Area" measured in the growth chamber

Source	df	Sum of squares	Mean squares	F value	p value
Rep	2	223601.04	111800.52	0.99	0.4761
Popn	1	7683907.48	7683907.48	6.84	0.0127
Iso(Popn)	38	42663310.47	1122718.70	6.07	0.0001
Rep*Popn	2	224059.89	112029.94	0.61	0.5481
Rep*Iso(Popn)	76	14046767.39	184825.89	2.94	0.0001
Leaf(Rep*Iso/Popn)	240	15085410.55	62855.88	1.77	0.0001
Det(Leaf/Rep*Iso/Popn)	720	25500318.54	35417.11		
Total	1079				

Table 15. Analysis of variance for "Pustule L-W Ratio" measured in growth chamber

Source	df	Sum of squares	Mean squares	F value	p value
Rep	2	0.49	0.24	12.00	0.1319
Popn	1	28.88	28.88	6.90	0.0124
Iso(Popn)	38	159.03	4.18	3.67	0.0001
Rep*Popn	2	0.05	0.02	0.02	0.9782
Rep*Iso(Popn)	76	86.59	1.14	2.47	0.0001
Leaf(Rep*Iso/Popn)	240	110.53	0.46	2.09	0.0001
Det(Leaf/Rep*Iso/Popn)	720	159.77	0.22		
Total	1079				

Table 16. Analysis of variance for "Pustule Length" measured in the growth chamber

Source	df	Sum of squares	Mean squares	F value	p value
Rep	2	423.16	211.58	2.64	0.2437
Popn	1	18007.50	18007.50	14.08	0.0006
Iso(Popn)	38	48609.99	1279.21	4.55	0.0001
Rep*Popn	2	159.75	79.87	0.28	0.7534
Rep*Iso(Popn)	76	21357.52	281.02	3.73	0.0001
Leaf(Rep*Iso/Popn)	240	18080.44	75.33	2.31	0.0001
Det(Leaf/Rep*Iso/Popn)	720	23392.00			
Total	1079				

Table 17. Analysis of variance for "Pustule Width" measured in the growth chamber

Source	df	Sum of squares	Mean squares	F value	p value
Rep	2	5.81	2.90	0.38	0.6148
Popn	1	28.03	28.03	0.32	0.5729
Iso(Popn)	38	3293.20	86.63	5.46	0.0001
Rep*Popn	2	15.23	7.61	0.48	0.6205
Rep*Iso(Popn)	76	1205.83	15.86	2.00	0.0001
Leaf(Rep*Iso/Popn)	240	1902.88	7.92	1.79	0.0001
Det(Leaf/Rep*Iso/Popn)	720	3221.33	4.47		
Total	1079				

Table 18. Analysis of variance for "Pustule Density (primary leaf)" measured in the growth chamber

Source	df	Sum of squares	Mean squares	F value	p value
Rep	2	231.78	115.89	5.98	0.2347
Popn	1	848.61	848.61	0.76	0.3889
Iso(Popn)	32	35591.37	1112.23	34.71	0.0001
Rep*Popn	2	38.70	19.35	0.60	0.5497
Rep*Iso(Popn)	64	2025.49	31.64	4.99	0.0001
Leaf(Rep*Iso/Popn)	204	1292.59	6.33		
Total	350				

Table 19. Analysis of variance for "Pustule Density (secondary leaf)" measured in the growth chamber

Source	df	Sum of squares	Mean squares	F value	p value
Rep	2	495.94	247.97	4.97	0.2812
Popn	1	774.00	774.00	1.04	0.3163
Iso(Popn)	32	23889.15	746.53	18.34	0.0001
Rep*Popn	2	99.63	49.82	1.22	0.3009
Rep*Iso(Popn)	64	2605.57	40.71	7.52	0.0001
Leaf(Rep*Iso/Popn)	204	1103.93	5.41		
Total	305				

Table 20. Analysis of variance for "Pustule Density (primary and secondary leaves)" measured in the growth chamber

Source	df	Sum of squares	Mean squares	F value	p value
Rep	2	388.22	194.11	8.88	0.1986
Popn	1	800.77	800.77	0.95	0.3364
Iso(Popn)	32	26899.51	840.60	29.19	0.0001
Rep*Popn	2	43.69	21.84	0.76	0.4725
Rep*Iso(Popn)	64	1843.34	28.80	8.69	0.0001
Leaf(Rep*Iso/Popn)	204	675.71	3.31		
Total	305				

Table 21. Analysis of variance for "Sporewt/Pustule" measured in the growth chamber

Source	df	Sum of squares	Mean squares	F value	p value
Rep	2	0.1833	0.0916	1.96	0.1602
Popn	1	1.8322	1.8322	5.66	0.0321
Iso(Popn)	14	4.5287	0.3235	6.90	0.0001
Rep*Popn	2	0.0347	0.0173	0.37	0.6933
Rep*Iso(Popn)	28	1.3122	0.0468		
Total	47				

Table 22. Analysis of variance for "Sporewt/Average Pustule Size" measured in the growth chamber

Source	df	Sum of squares	Mean squares	F value	p value
Rep	2	0.001	0.0005	0.00	0.9974
Popn	1	0.023	0.0230	3.59	0.0791
Iso(Popn)	14	0.090	0.0060	13.83	0.0001
Rep*Popn	2	0.001	0.0005	1.62	0.2155
Rep*Iso(Popn)	28	0.013	0.0004		
Total	47				

Table 23. Analysis of variance for "Sporewt/Total Pustule Area" measured in the growth chamber

Source	df	Sum of squares	Mean squares	F value	p value
Rep	2	0.1460	0.0730	2.92	0.0705
Popn	1	0.7654	0.7654	4.70	0.0479
Iso(Popn)	14	2.2794	0.1628	6.51	0.0001
Rep*Popn	2	0.0393	0.0196	0.79	0.4651
Rep*Iso(Popn)	28	0.7003			
Total	47				

Table 24. Analysis of variance for "Sporewt/Total Leaf Area" measured in the growth chamber

Source	df	Sum of squares	Mean squares	F value	p value
Rep	2	33.43	16.71	3.16	0.0482
Popn	1	47.12	47.12	0.42	0.5222
Iso(Popn)	38	4292.84	112.96	21.33	0.0001
Rep*Popn	2	5.54	2.77	0.52	0.5944
Rep*Iso(Popn)	76	402.59	5.29		
Total	119				

Table 25. Analysis of variance for "Sporewt/Pustule L-W Ratio" measured in the growth chamber

Source	df	Sum of squares	Mean squares	F value	p value
Rep	2	117.97	58.98	0.86	0.4325
Popn	1	1368.44	1368.44	2.77	0.1180
Iso(Popn)	14	6907.24	479.08	7.23	0.0001
Rep*Popn	2	54.22	27.11	0.40	0.6760
Rep*Iso(Popn)	28	1911.95			
Total	47				

chi-square values given in Table 26. All "visual" traits exhibited significant differences between the two populations. Population means, standard errors and R square values for all traits are given in Tables 27-30. Pustule densities on the secondary leaves were less than on the primary leaves because a portion of the inoculum to be sprayed onto the secondary leaves was blocked by the primary leaves. Inoculation of the secondary leaves alone, however, can produce pustule densities equivalent to that of the primary leaves.

Correlation coefficients between traits in both populations are given in Tables 31-46. Similar trends in association between these traits have been previously reported in a review of components for horizontal resistance (37). Different correlations did occur between the two populations for some traits. For example, Latent Period (LP) was positively correlated with Telia(1) and Telia(1)-LP in the asexual population while no such correlation occurred in the sexual population. Telia(1)-LP was positively correlated with Pustule Density in the asexual population while no such correlation occurred in the sexual population. Telia(1)-LP was positively correlated with Pustule Density in the asexual population, but the association did not hold in the sexual population. "Spore weight" traits were in general negatively correlated with other traits in both populations, probably due in part to high inoculum concentrations (37).

The frequency distribution of identified virulence genes for both populations is given in Figures 2 and 3. Both populations approached a normal or Poisson distribution, and we have assumed that virulence genes

Table 26. Log linear analyses for Latent Period, Telia(1), and Telia(1)-LP measured in the growth chamber

Source	df	Chi square	p value
Trait: Latent Period (LP)			
Intercept	1	19579.14	0.0001
Rep	2	0.02	0.9876
Popn	1	4.35	0.0370
Residual	2	0.35	0.8377
Trait: Telia(1)			
Intercept	1	2833.13	0.0001
Rep	2	0.63	0.7283
Popn	1	17.10	0.0001
Residual	2	0.63	0.9384
Trait: Telia(1)-LP			
Intercept	1	1313.39	0.0001
Rep	2	2.16	0.3400
Popn	1	27.02	0.0001
Residual	2	0.87	0.6473

Table 27. Population means, standard errors, and R square values for "pustule measurement" traits measured in the growth chamber

Trait	Mean[a] of Asex Popn	Standard error of Asex Popn	Mean of Sex Popn	Standard error of Sex Popn	R square[b]
Pustule Length	86.81	1.38	95.00	1.67	0.82
Pustule Width	21.73	0.36	22.00	0.43	0.67
Pustule Area	1481.34	36.37	1649.22	53.03	0.75
Pustule L-W Ratio	4.01	0.09	4.32	0.08	0.71

[a]Data from pustules on secondary leaves only.

[b]Values for combined data of asexual and sexual populations.

Table 28. Population means, standard errors, and R square values for "pustule density" traits measured in the growth chamber

Trait	Mean[a] of Asex Popn	Standard error of Asex Popn	Mean of Sex Popn	Standard error of Sex Popn	R square[b]
Pustule Density (primary leaf)	29.49	2.70	32.26	2.59	0.96
Pustule Density (secondary leaf)	18.80	6.30	21.56	7.68	0.96
Pustule Density (primary and secondary leaves)	22.69	2.30	25.45	2.30	0.96

[a]Mean values in number of pustules/cm^2 leaf tissue.

[b]Values for combined data of asexual and sexual populations.

Table 29. Population means, standard errors, and R square values for "Spore Weight" traits measured in the growth chamber

Trait	Mean of Asex Popn	Standard error of Asex Popn	Mean of Sex Popn	Standard error of Sex Popn	R square[a]
Sporewt/Pustule	0.73	0.13	1.19	0.14	0.83
Sporewt/Total Pustule Area	0.11	0.01	0.15	0.02	0.90
Sporewt/Ave. Pustule Size	0.56	0.10	0.81	0.10	0.82
Sporewt/Total Leaf Area	8.51	1.68	8.30	2.06	0.91
Sporewt/Pustule L-W Ratio	37.60	4.50	48.30	5.27	0.81

[a]Values for combined data of asexual and sexual populations.

Table 30. Population means and standard errors for "visual assessment" traits measured in the growth chamber

Trait	Mean of Asex Popn	Standard error of Asex Popn	Mean of Sex Popn	Standard error of Sex Popn
Latent Period (LP)	7.32	0.10	7.10	0.13
Telia(1)	28.72[a]	3.24	20.68	2.88
Telia(1)-LP	21.40[a]	3.15	13.58	2.87

[a]Estimated from "log linear analysis" (2).

Table 31. Correlation coefficients for traits of aggressiveness in the asexual poulation measured in the growth chamber

Characters	Telia(1)	Telia(1) -LP	Pus. Length	Pus. Width	Pus. Area	Pus. L-W Ratio
Latent Period	0.83**	0.82**	0.29	0.41	0.47*	-0.11
Telia(1)		0.99*	0.41	0.66**	0.74**	-0.23
Telia(1)-LP			0.41	0.67**	0.75**	-0.23
Pus. Length				0.06	0.72**	0.65**
Pus. Width					0.74**	-0.71**
Pus. Area						-0.06

*,**Statistically significant at the 0.05 and 0.01 levels of probability, respectively.

Table 32. Correlation coefficients for traits of aggressiveness in the asexual population measured in the growth chamber

Characters	Telia(1)	Telia(1) -LP	Pus. Den. (pri. leaf)	Pus. Den. (sec. leaf)	Pus. Den. (pri. + sec.)
Latent Period	0.83**	0.82**	0.61**	0.66**	0.65**
Telia(1)		0.99**	0.65**	0.73**	0.71**
Telia(1)-LP			0.65**	0.74**	0.72**
Pus. Den. (pri. leaf)				0.91**	0.97**
Pus. Den. (sec. leaf)					0.98**

**Statistically significant at the 0.01 level of probability.

Table 33. Correlation coefficients for traits of aggressiveness in the asexual population measured in the growth chamber

Characters	Pus. Den. (sec. leaf)	Pus. Den. (pri. + sec.)	Pus. Length	Pus. Width	Pus. Area	Pus. L-W Ratio
Pus. Den. (pri. leaf)	0.91**	0.97**	0.34	0.40	0.59**	-0.03
Pus. Den. (sec. leaf)		0.98**	0.18	0.62**	0.62**	-0.27
Pus. Den. (pri. + sec.)			0.25	0.54*	0.62**	-0.18
Pus. Length				0.06	0.72**	0.65**
Pus. Width					0.74**	-0.71**
Pus. Area						-0.06

*,**Statistically significant at the 0.05 and 0.01 levels of probability, respectively.

Table 34. Correlation coefficients for traits of aggressiveness in the asexual population measured in the growth chamber

Characters	Telia(1)	Telia-(1)-LP	Sporewt/ Pus.	Sporewt/ Ave. Pus. Size	Sporewt/ Tot. Pus. Area	Sporewt/ Tot. Leaf Area	Sporewt/ Pus. L-W Ratio
Latent Period	0.83*	0.82*	-0.69**	-0.68**	-0.67**	-0.68**	-0.72**
Telia(1)		0.99*	-0.77**	-0.78**	-0.75**	-0.78**	-0.80**
Telia(1)-LP			-0.78**	-0.78**	-0.75**	-0.78**	0.77**
Sporewt/Pus.				0.83**	0.99**	0.82**	0.77**
Sporewt/Ave. Pus. Size					0.82**	0.99**	0.98**
Sporewt/Tot. Pus. Area						0.79**	0.75**
Sporewt/Tot. Leaf Area							0.98**

*,**Statistically significant at the 0.05 and 0.01 levels of probability, respectively.

Table 35. Correlation coefficients for traits of aggressiveness in the asexual population measured in the growth chamber

Characters	Pus. Den. (sec. leaf)	Pus. Den. (pri. + sec.)	Sporewt/ Pus.	Sporewt/ Ave. Pus. Size	Sporewt/ Total Pus. Area	Sporewt/ Total Leaf Area	Sporewt/ Pus. L-W Ratio
Pus. Den. (pri. leaf)	0.91**	0.97**	-0.67**	-0.40	-0.68**	-0.38	-0.39
Pus. Den. (sec. leaf)		0.98**	-0.80**	-0.64*	-0.80**	-0.62**	-0.60**
Pus. Den. (pri. + sec.)			-0.76**	-0.55*	-0.76**	-0.53*	-0.52*
Sporewt/Pus.				0.84**	0.99**	0.82**	0.77**
Sporewt/Ave. Pus. Size					0.82**	0.99**	0.98**
Sporewt/Tot. Pus. Area						0.79**	0.98**
Sporewt/Tot. Leaf Area							0.79**

*,**Statistically significant at the 0.05 and 0.01 levels of probability, respectively.

Table 36. Correlation coefficients for the traits of aggressiveness in the asexual population measured in the growth chamber

Characters	Pus. Width	Pus. Area	Pus. L-W Ratio	Sporewt/ Pus.	Sporewt/ Ave. Pus. Size	Sporewt/ Total Pus. Area	Sporewt/ Total Leaf Area	Sporewt/ Pus. L-W Ratio
Pus. Length	0.06	0.72**	0.65**	-0.06	-0.25	-0.08	-0.23	-0.36
Pus. Width		0.74*	-0.71**	-0.84**	-0.71**	-0.84**	-0.69*	-0.61**
Pus. Area			-0.06	-0.69**	-0.67**	-0.70**	-0.64**	-0.67**
Pus. L-W Ratio				0.50*	0.39	0.49*	0.39	0.23
Sporewt/Pus.					0.84**	0.99**	0.82**	0.77**
Sporewt/Ave. Pus. Size						0.82**	0.99**	0.98**
Sporewt/Tot. Pus. Area							0.79**	0.98**
Sporewt/Totl Leaf Area								0.79**

*,**Statistically significant at the 0.05 and 0.01 levels of probability, respectively.

Table 37. Correlation coefficients for traits of aggressiveness in the sexual population measured in the growth chamber

Characters	Telia(1)	Telia(1) -LP	Pus. Length	Pus. Width	Pus. Area	Pus. L-W Ratio
Latent Period	0.09	0.05	0.14	0.73**	0.52*	-0.62**
Telia(1)		0.99*	0.27	0.52*	0.48**	-0.30
Telia(1)-LP			0.26	0.49*	0.44*	-0.27
Pus. Length				0.46*	0.85**	0.41
Pus. Width					0.86**	-0.62**
Pus. Area						-0.13

*,**Statistically significant at the 0.05 and 0.01 levels of probability, respectively.

Table 38. Correlation coefficients for traits of aggressiveness in the sexual population measured in the growth chamber

Characters	Telia(1)	Telia(1) -LP	Pus. Den. (pri. leaf)	Pus. Den. (sec. leaf)	Pus. Den. (pri. + sec.)
Latent Period	0.09	0.05	0.47*	0.49*	0.49**
Telia(1)		0.99**	0.45	0.45	0.46
Telia(1)-LP			0.43	0.43	0.44
Pus. Den. (pri. leaf)				0.94**	0.98**
Pus. Den. (sec. leaf)					0.99**

*,**Statistically significant at the 0.05 and 0.01 levels of probability, respectively.

Table 39. Correlation coefficients for traits of aggressiveness in the sexual population measured in the growth chamber

Characters	Pus. Den. (sec. leaf)	Pus. Den. (pri. + sec.)	Pus. Length	Pus. Width	Pus. Area	Pus. L-W Ratio
Pus. Den. (pri. leaf)	0.94**	0.98**	0.06	0.59**	0.35	-0.58**
Pus. Den. (sec. leaf)		0.99**	0.05	0.58**	0.36	-0.53**
Pus. Den. (pri. + sec.)			0.03	0.60**	0.36	-0.56*
Pus. Length				0.46*	0.85*	0.41
Pus. Width					0.86**	-0.62**
Pus. Area						-0.13

*,**Statistically significant at the 0.05 and 0.01 levels of probability, respectively.

Table 40. Correlation coefficients for traits of aggressiveness in the sexual population measured in the growth chamber

Characters	Telia(1)	Telia-(1)-LP	Sporewt/ Pus.	Sporewt/ Ave. Pus. Size	Sporewt/ Total Pus. Area	Sporewt/ Total Leaf Area	Sporewt/ Pus. L-W Ratio
Latent Period	0.09	0.05	-0.61**	-0.72**	-0.61**	-0.72**	-0.73**
Telia(1)		0.99**	-0.53*	-0.47*	-0.53*	-0.47*	-0.47*
Telia(1)-LP			-0.50*	-0.45*	-0.51*	-0.44	-0.44
Sporewt/Pus.				0.89**	0.99**	0.91**	0.90**
Sporewt/Ave. Pus. Size					0.91**	0.99**	0.99**
Sporewt/Tot. Pus. Area						0.91**	0.91**
Sporewt/Tot. Leaf Area							0.99**

*,**Statistically significant at the 0.05 and 0.01 levels of probability, respectively.

Table 41. Correlation coefficients for traits of aggressiveness in the sexual population measured in the growth chamber

Characters	Pus. Width	Pus. Area	Pus. L-W Ratio	Sporewt/ Pus.	Sporewt/ Ave. Pus. Size	Sporewt/ Tot. Pus. Area	Sporewt/ Total Leaf Area	Sporewt/ Pus. L-W Ratio
Pus. Length	0.46*	0.85**	0.41	-0.33	-0.45*	-0.42	-0.42	-0.45*
Pus. Width		0.86**	-0.62**	-0.82**	-0.87**	-0.85*	-0.85**	-0.85**
Pus. Area			0.13	-0.69**	-0.77**	-0.74**	-0.74**	-0.76**
Pus. L-W Ratio				0.53*	0.49*	0.51**	0.51*	0.48*
Sporewt/Pus.					0.89**	0.99**	0.91**	0.90**
Spotewt/Ave. Pus. Size						0.91**	0.99**	0.99**
Sporewt/Tot. Pus. Area							0.91**	0.91**
Sporewt/Tot. Leaf Area								0.99**

*,**Statistically significant at the 0.05 and 0.01 levels of probability, respectively.

Table 42. Estimated heritability values from the component analyses of variance for traits of aggressiveness measured in the growth chamber

Trait	h^2 Asex Popn	h^2 Sex Popn
Pustule Length	65%	86%
Pustule Width	83%	80%
Pustule Area	79%	75%
Pustule L-W Ratio	71%	75%
Pustule Density (pri. leaf)	96%	98%
Pustule Density (sec. leaf)	92%	97%
Pustule Density (pri. + sec.)	95%	98%

Table 43. Estimated heritability values from the component analysis of variance for traits of aggressiveness measured in the growth chamber

Trait	h^2 Asex Popn	h^2 Sex Popn
Sporewt/Pustule	90%	81%
Sporewt/Average Pustule Size	93%	92%
Sporewt/Total Pustule Area	86%	82%
Sporewt/Total Leaf Area	94%	96%
Sporewt/L-W Ratio	90%	69%

Table 44. Estimation of the number of effective factors, K, for traits of aggressiveness in the sexual population

Trait	K value
Pustule Length	7.56
Pustule Width	9.88
Pustule L-W Ratio	10.66
Pustule Area	8.59
Pustule Density (pri. leaf)	4.06
Pustule Density (sec. leaf)	3.25
Pustule Density (pri. + sec. leaf)	3.99

Table 45. Estimation of the number of effective factors, K, for traits of aggressiveness in the sexual population

Trait	K value
Sporewt/Pustule	6.40
Sporewt/Average Pustule Size	3.10
Sporewt/Total Pustule Area	4.94
Sporewt/Total Leaf Area	3.99
Sporewt/Pustule L-W Ratio	8.24

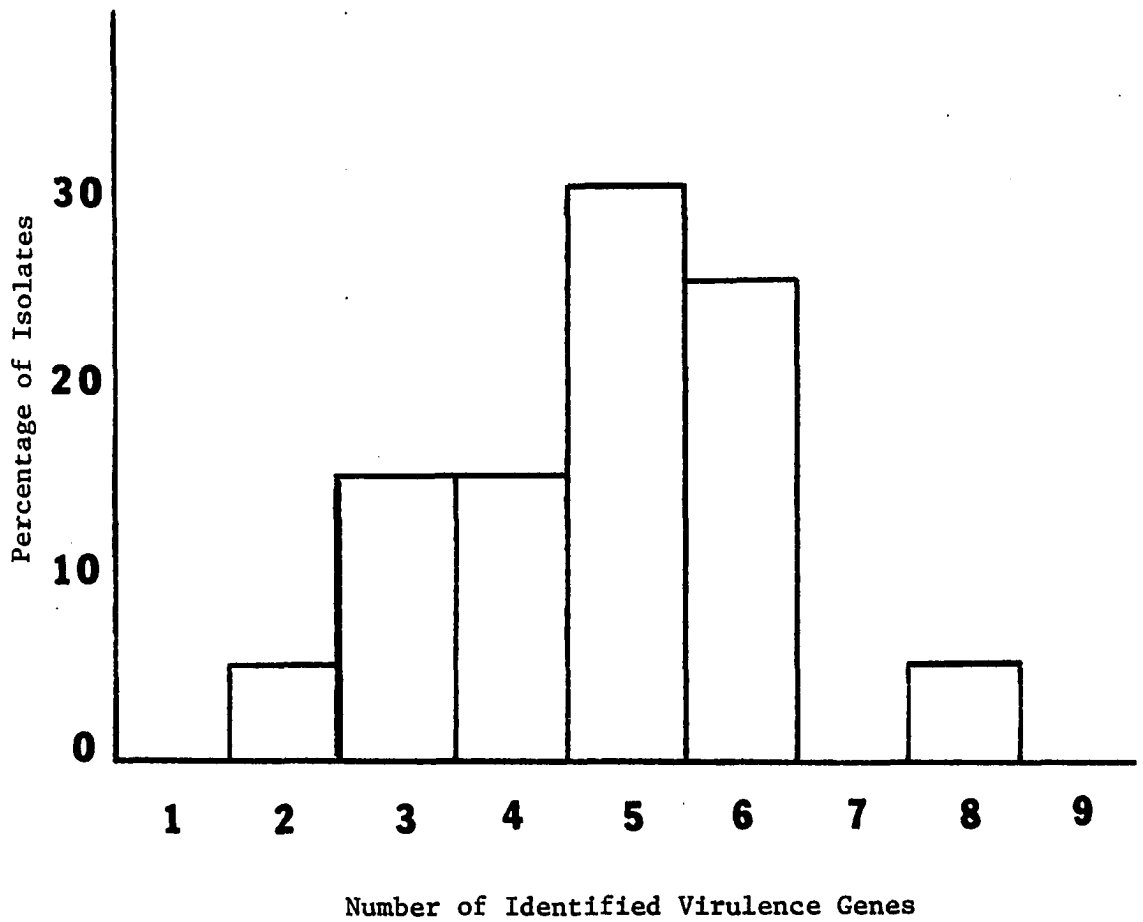

Figure 2. Frequency distribution of virulence genes in the asexual population of P. coronata

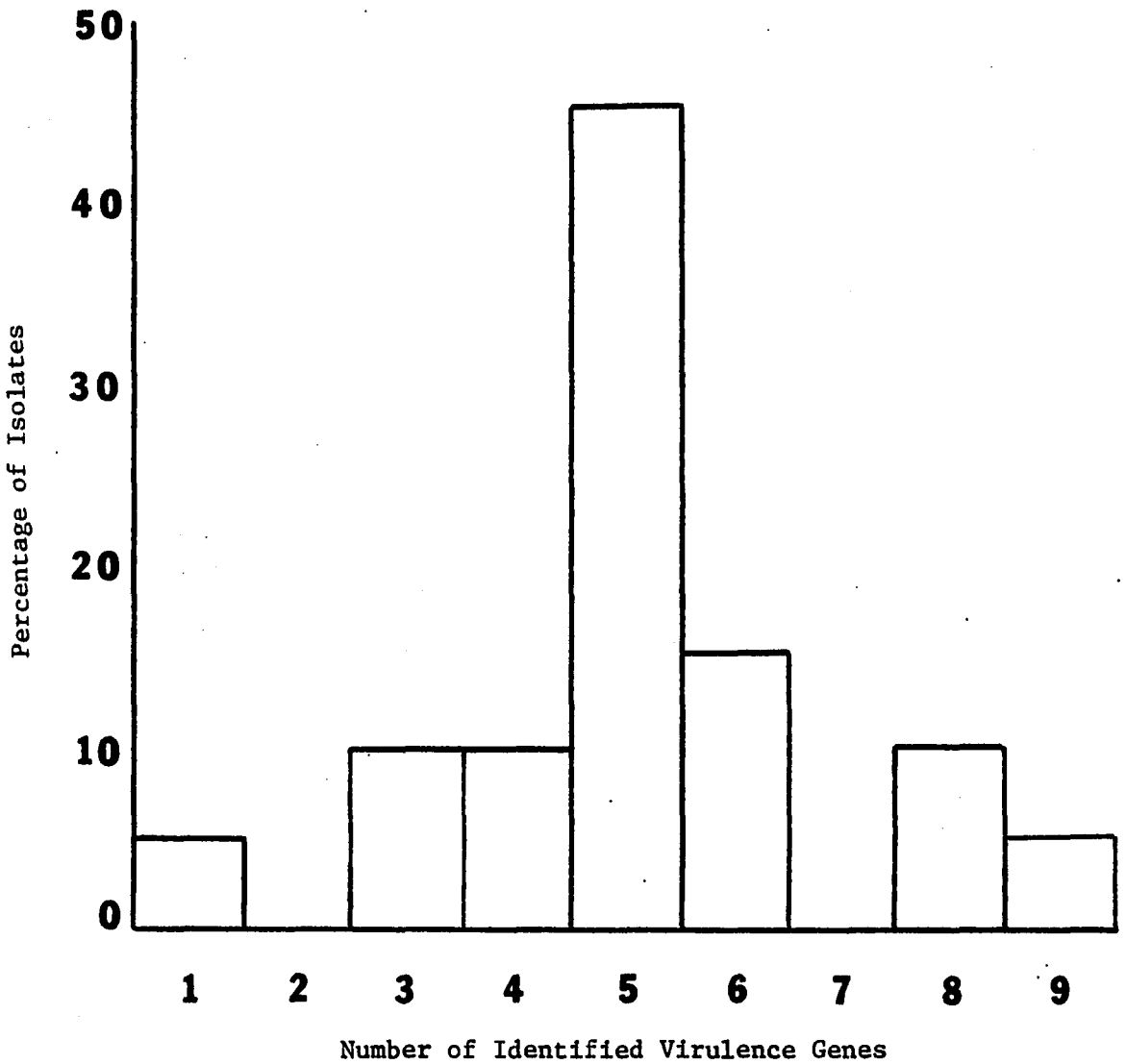

Figure 3. Frequency distribution of virulence genes in the sexual population of P. coronata

act independently of each other such that the total average effect of the genes in each population cancel each other out. This means that the effects of virulence genes at other loci are minimal or nonexistent. The distribution of individual virulence genes provides no explanation of the phenotypic variability at the virulence loci between the two populations. A simple comparison of variability is the percentage of distinct phenotypes for these loci, which was 85% for the sexual population and 35% for the asexual. A second measure of variation is "Simpson's Measure of Diversity" (38) defined as:

$$D = 1 - \sum_{j} \frac{N_j(N_j-1)}{N(N-1)} \text{ , where}$$

N_j = the number collected of the j^{th} phenotype

N = sample size.

For the sexual population, D = 0.984 and D = 0.726 for the asexual.

Heritability values for each trait are given in Tables 42 and 43. Heritabilities for Latent Period, Telia(1), and Telia(1)-LP were not estimated because values for σ_A^2 were not greater than zero. Estimated numbers of effective factors for traits in the sexual population are given in Tables 44 and 45.

1980 Field Study

The analyses of variance of traits of aggressiveness for combined cultivars in the 1980 field study are given in Tables 46-55. The cultivars Bates, Nodaway 70, Allen, Spear, and Larry displayed moderate resistant or resistant infection types and are not included in the study.

Table 46. Analysis of variance of Latent Period for combined cultivars in the 1980 field study

Source	df	Sum of squares	Mean squares	F value	p value
Popn	1	99.00	99.000	5.56	0.0461
Iso(Popn)	8	142.42	17.800	42.42	0.0001
Rep(Popn*Iso)	70	0.17	0.002	1.00	0.4849
Cultivar	4	0.01	0.002	1.00	0.5000
Popn*Cv.	4	0.01	0.002	1.00	0.4219
Cv.*Iso(Popn)	32	0.08	0.002	1.00	0.4219
Cv.*Rep(Popn*Iso)	280	0.70	0.002		
Total	399				

Table 47. Analysis of variance of Telia(1) for combined cultivars in the 1980 field study

Source	df	Sum of squares	Mean squares	F value	p value
Popn	1	3534.30	3534.30	1.27	0.2929
Iso(Popn)	8	22304.96	2788.12	2349.30	0.0001
Rep(Popn*Iso)	70	83.07	1.19	1.54	0.0078
Cultivar	4	261.60	65.40	2.80	0.1710
Popn*Cv.	4	93.31	23.32	0.38	0.8227
Cv.*Iso(Popn)	32	1975.14	61.72	80.18	0.0001
Cv.*Rep(Popn*Iso)	280	215.55	0.77		
Total	399				

Table 48. Analysis of variance of Telia(90) for combined cultivars in the 1980 field study

Source	df	Sum of squares	Mean squares	F value	p value
Popn	1	18130.62	18130.62	13.64	0.0061
Iso(Popn)	8	10634.35	1329.29	3508.03	0.0001
Rep(Popn*Iso)	70	26.52	0.37	0.53	0.9991
Cultivar	4	142.53	35.63	1.84	0.2844
Popn*Cv.	4	77.14	19.28	0.50	0.7326
Cv.*Iso(Popn)	32	1227.25	38.35	53.27	0.0001
Cv.*Rep(Popn*Iso)	280	201.60	0.72		
Total	399				

Table 49. Analysis of variance of Telia(90)-Telia(1) for combined cultivars in the 1980 field study

Source	df	Sum of squares	Mean squares	F value	p value
Popn	1	5655.04	5655.04	2.96	0.1235
Iso(Popn)	8	15266.07	1908.26	1151.54	0.0001
Rep(Popn*Iso)	70	116.00	1.66	3.03	0.0001
Cultivar	4	201.78	50.44	3.54	0.1241
Popn*Cv.	4	56.98	14.24	0.20	0.9376
Cv.*Iso(Popn)	32	2302.23	71.94	131.66	0.0001
Cv.*Rep(Popn*Iso)	280	153.00	0.55		
Total	399				

Table 50. Analysis of variance of Telia(1)-LP for combined cultivars in the 1980 field study

Source	df	Sum of squares	Mean squares	F value	p value
Popn	1	4816.36	4816.36	1.80	0.2171
Iso(Popn)	8	21465.50	2683.19	2254.78	0.0001
Rep(Popn*Iso)	70	83.30	1.19	1.54	0.0079
Cultivar	4	259.93	64.98	2.76	0.1749
Popn*Cv.	4	94.31	23.58	0.38	0.8194
Cv.*Iso(Popn)	32	1972.10	61.63	79.72	0.0001
Cv.*Rep(Popn*Iso)	280	216.45	0.77		
Total	399				

Table 51. Analysis of variance Telia(90)-LP for combined cultivars in the 1980 field study

Source	df	Sum of squares	Mean squares	F value	p value
Popn	1	20909.16	20909.16	17.62	0.0030
Iso(Popn)	8	9490.69	1186.34	3110.24	0.0001
Rep(Popn*Iso)	70	26.70	0.38	0.53	0.9991
Cultivar	4	141.05	35.26	1.81	0.2902
Popn*Cv.	4	78.04	19.51	0.51	0.7286
Cv.*Iso(Popn)	32	1223.81	38.24	52.93	0.0001
Cv.*Rep(Popn*Iso)	280	202.30	0.72		
Total	399				

Table 52. Analysis of variance of Yield for combined cultivars in the 1980 field study

Source	df	Sum of squares	Mean squares	F value	p value
Popn	1	1505.44	1505.44	3.51	0.0980
Iso(Popn)	8	3433.27	429.16	11.55	0.0001
Rep(Popn*Iso)	70	2600.85	37.15	1.46	0.0173
Cultivar	4	1016.41	254.10	3.62	0.1202
Popn*Cv.	4	280.61	70.15	1.32	0.2849
Cv.*Iso(Popn)	32	1704.93	53.28	2.09	0.0008
Cv.*Rep(Popn*Iso)	280	7121.65	25.43		
Total	399				

Table 53. Analysis of variance of Yield Index for combined cultivars in the 1980 field study

Source	df	Sum of squares	Mean squares	F value	p value
Popn	1	0.34	0.340	2.55	0.1487
Iso(Popn)	8	1.08	0.130	11.99	0.0001
Cultivar	4	0.11	0.030	5.42	0.0652
Cv.*Popn	4	0.02	0.005	0.46	0.7609
Cv.*Iso(Popn)	32	0.36	0.011		
Total	49				

Table 54. Analysis of variance of Seed Weight for combined cultivars in the 1980 field study

Source	df	Sum of squares	Mean squares	F value	p value
Popn	1	0.42	0.42	0.09	0.7600
Iso(Popn)	8	35.80	0.05	20.03	0.0001
Rep(Pop*Iso)	70	15.64	0.22	1.57	0.0056
Cultivar	4	119.30	29.82	153.11	0.0001
Popn*Cv.	4	0.78	0.19	0.29	0.8838
Cv.*Iso(Popn)	32	21.66	0.68	4.76	0.0001
Cv.*Rep(Popn*Iso)	280	39.79	0.14		
Total	399				

Table 55. Analysis of variance of Seed Weight Index for combined cultivars in the 1980 field study

Source	df	Sum of squares	Mean squares	F value	p value
Popn	1	0.002	0.002	0.13	0.7290
Iso(Popn)	8	0.161	0.002	5.87	0.0001
Cultivar	4	0.043	0.011	5.42	0.0653
Cv.*Popn	4	0.008	0.002	0.58	0.6820
Cv.*Iso(Popn)	32	0.110	0.003		
Total	49				

Significant population differences were detected for Latent Period, Telia(90), and Telia(90)-LP. In addition, a highly significant Iso(Popn) term was present for each trait in both populations. However, a significant Cv.*Iso interaction was exhibited by all traits except Latent Period. Significance of the interaction for Yield Index and Seed Index was not testable since an appropriate error term did not exist. This interaction may be statistically significant due to the relatively large degrees of freedom (240) for the error term. Analyses of variance of the traits were also conducted for individual cultivars. Population means, standard errors, and R square values of traits for combined cultivars are given in Tables 56 and 57. The results of the analysis for each cultivar were similar to those of the combined cultivar analyses, except for Yield and Yield Index in the cultivar, Clintford. Significant population differences were detected for the two traits, as shown in Tables 58 and 59. Population means, standard errors, and R square values for Clintford are given in Table 60.

Correlation coefficients for the sexual population are given in Table 61. Latent Period exhibited a weak association with Yield (r=0.54**), Yield Index (r=0.49*), and Seed Weight Index (r=0.41*), which suggested that long latent periods were correlated to a slight degree with reduced levels of aggressiveness. Telia(1)-LP showed a weak negative correlation with Seed Weight Index (r=-0.41*) which implied long periods between Latent Period and Telia(1) were weakly associated with increased levels of aggressiveness. Telia(90)-Telia(1) displayed a weak association with Yield Index (r=0.42*) which

Table 56. Population means, standard errors, and R square values of "visual" traits of aggressiveness for combined cultivars in the 1980 field study

Trait	Mean of Asex Popn	Standard error of Asex Popn	Mean of Sex Popn	Standard error of Sex Popn	R square[a]
Latent Period	6.80	0.37	7.80	0.20	0.98
Telia(1)	26.28	5.31	20.34	1.44	0.98
Telia(90)	41.96	3.60	28.49	1.32	0.98
Telia(1)-LP	19.48	5.19	12.54	1.49	0.99
Telia(90)-LP	35.15	3.41	20.69	1.29	0.98
Telia(90)-Telia(1)	15.67	4.48	8.15	1.19	0.99

[a]Values for combined data of asexual and sexual populations.

Table 57. Population means, standard errors, and R square values of "yield" traits of aggressiveness for combined cultivars in the 1980 field study

Trait	Mean of Asex Popn	Standard error of Asex Popn	Mean of Sex Popn	Standard error of Sex Popn	R square[a]
Yield	18.45	1.77	22.33	1.67	0.60
Yield Index	0.59	0.06	0.76	0.09	0.81
Seed Weight	4.30	0.20	4.37	0.15	0.83
Seed Weight Index	0.80	0.04	0.81	0.04	0.66

[a]Values for combined data of asexual and sexual populations.

Table 58. Analysis of variance of Yield Index for the cultivar Clintford in the 1980 field study

Source	df	Sum of squares	Mean squares	F value	p value
Popn	1	0.0882	0.088	11.82	0.0088
Iso(Popn)	8	0.0591	0.007		
Total	9				

Table 59. Analysis of variance of Yield for the cultivar Clintford in the 1980 field study

Source	df	Sum of squares	Mean squares	F value	p value
Popn	1	812.81	812.81	6.68	0.0324
Iso(Popn)	8	973.45	121.68		
Total	9				

Table 60. Population means, standard errors, and R square values of traits of aggressiveness for the cultivar Clintford in the 1980 field study

Trait	Mean of Asex Popn	Standard error of Asex Popn	Mean of Sex Popn	Standard error of Sex Popn	R square[a]
Latent Period	6.82	0.36	7.80	0.20	0.98
Telia(1)	26.62	5.16	21.67	1.22	0.99
Telia(90)	42.40	3.60	29.55	1.01	0.99
Telia(1)-LP	19.80	5.04	13.87	1.32	0.99
Telia(90)-LP	35.57	3.41	21.75	1.06	0.99
Telia(90)-Telia(1)	15.77	4.32	7.87	1.98	0.98
Yield	17.35	1.11	23.72	2.20	0.48
Yield Index	0.52	0.03	0.71	0.05	0.60
Seed Weight	4.49	0.20	4.56	0.15	0.57
Seed Weight Index	0.79	0.03	0.80	0.03	0.01

[a]Values given are for combined sexual and asexual populations.

Table 61. Correlation coefficients for traits of aggressiveness in the sexual population of the 1980 field study

Characters	Telia-(1)	Telia-(90)	Telia-(1)-LP	Telia-(90)-LP	Telia(90)-Telia(1)	Yield	Yield Index	Seed Weight	Seed Weight Index
Latent Period	-0.23	-0.02	-0.34	-0.17	0.23	0.54**	0.49*	0.26	0.41**
Telia(1)		0.51**	0.99**	0.54**	-0.61**	-0.13	-0.07	0.01	-0.37
Telia(90)			0.50*	0.99**	0.37	-0.05	0.37	-0.01	-0.22
Telia(1)-LP				0.54**	-0.61**	-0.18	-0.13	-0.03	-0.41*
Telia(90)-LP					0.33	-0.14	0.29	-0.05	-0.27
Telia(90)-Telia(1)						0.08	0.42*	-0.02	0.21
Yield							0.59*	0.67*	0.72**
Yield Index								0.25	0.37
Seed Weight									0.67**

*,**Statistically significant at the 0.05 and 0.01 levels of probability, respectively.

suggested that rate of telia formation was correlated with reduced levels of aggressiveness. Correlation coefficients for the asexual population are given in Table 62. Latent Period exhibited similar associations for Yield (r=0.45*) and Seed Weight Index (r=-.41*), as in the sexual population. Telia(1), Telia(90), Telia(1)-LP, and Telia(90)-LP, however, showed positive associations with Yield Index, Seed Weight Index and a moderate association with Seed Weight, all of which were in contrast to those correlations in the sexual population. These correlations in the asexual population suggested that longer periods to telia formation were associated with reduced levels of aggressiveness. An example of this was seen with an isolate of 264B, a predominant race in the asexual population which has exhibited relatively short times for telia formation.

Heritability estimates of traits for combined cultivars are given in Table 63. High heritability values for all traits occurred in each population except for low estimates of Telia(1) and Telia(1)-LP in the sexual population. Heritability estimates of the "yield" traits for the cultivars Clintford, Lang, and Otee are given in Table 64. The asexual population exhibited higher estimates on Lang and Otee, while the sexual population displayed higher estimates on Clintford.

1981 Field Study

The analyses of variance of traits of aggressiveness for combined cultivars in the 1981 field study are given in Tables 65-74. No population differences were detected for any trait except Coefficient of Infection. Again, a significant Iso(Popn) term occurred for each trait

Table 62. Correlation coefficients for traits of aggressiveness in the asexual population of the 1980 field study

Characters	Telia-(1)	Telia-(90)	Telia-(1)-LP	Telia-(90)-LP	Telia(90)-Telia(1)	Yield	Yield Index	Seed Weight	Seed Weight Index
Latent Period	0.35	0.60	0.28	0.53**	0.07	0.45*	0.32	0.26	0.41*
Telia(1)		0.55**	0.99**	0.55**	-0.73**	0.17	0.63**	0.51*	0.73**
Telia(90)			0.52*	0.99**	0.16	0.62**	0.62**	0.41*	0.64**
Telia(1)-LP				0.52*	-0.76**	0.14	0.62**	0.50*	0.71**
Telia(90)-LP					0.15	0.61**	0.63**	0.41*	0.63**
Telia(90)-Telia(1)						0.30	0.24	-0.26	-0.34
Yield							0.76**	0.57**	0.58**
Yield Index								0.59**	0.76**
Seed Weight									0.84**

*,**Statistically significant at the 0.05 and 0.01 levels of probability, respectively.

Table 63. Estimated heritability values from the component analysis of variance of traits of aggressiveness for combined cultivars in the 1980 field study

Trait	Asex Popn	Sex Popn
Latent Period	98%	99%
Telia(1)	99%	69%
Telia(90)	98%	80%
Telia(1)-LP	98%	34%
Telia(90)-LP	98%	80%
Telia(90)-Telia(1)	97%	61%
Yield	89%	78%
Yield Index	95%	97%
Seed Weight	92%	86%
Seed Weight Index	92%	56%

Table 64. Estimated heritability values from the component analysis of variance of yield for selected cultivars in the 1980 field study

Cultivar	Asex Popn	Sex Popn
Clintford	16%	86%
Lang	52%	23%
Otee	89%	45%

Table 65. Analysis of variance of Latent Period for combined cultivars in the 1981 field study

Source	df	Sum of squares	Mean squares	F value	p value
Popn	1	9.56	9.56	2.13	0.1570
Iso(Popn)	24	107.51	4.48	8.71	0.0001
Rep(Popn*Iso)	78	40.10	0.51	1.96	0.0001
Cultivar	4	10.20	2.55	14.86	0.0114
Popn*Cv.	4	0.68	0.17	0.32	0.8644
Cv.*Iso(Popn)	96	51.61	0.54	2.05	0.0001
Cv.*Rep(Popn*Iso)	312	81.90	0.26		
Total	519				

Table 66. Analysis of variance of Telia(1) for combined cultivars in the 1981 field study

Source	df	Sum of squares	Mean squares	F value	p value
Popn	1	101.11	101.11	1.21	0.2815
Iso(Popn)	24	1998.99	83.29	8.05	0.0001
Rep(Popn*Iso)	78	806.65	10.34	2.31	0.0001
Cultivar	4	51.51	12.88	3.07	0.1517
Popn*Cv.	4	16.80	4.20	0.41	0.8033
Cv.*Iso(Popn)	96	991.09	10.32	2.30	0.0001
Cv.*Rep(Popn*Iso)	312	1398.60	4.48		
Total	519				

Table 67. Analysis of variance of Telia(1)-LP for combined cultivars in the 1981 field study

Source	df	Sum of squares	Mean squares	F value	p value
Popn	1	172.86	172.86	2.09	0.1615
Iso(Popn)	24	1998.19	83.25	8.22	0.0001
Rep(Popn*Iso)	78	785.95	10.07	2.14	0.0001
Cultivar	4	39.10	9.98	1.80	0.2916
Popn*Cv.	4	20.05	5.01	0.50	0.7385
Cv.*Iso(Popn)	96	969.85	10.10	2.15	0.0001
Cv.*Rep(Popn*Iso)	312	1466.80	4.70		
Total	519				

Table 68. Analysis of variance of Telia(90)-LP for combined cultivars in the 1981 field study

Source	df	Sum of squares	Mean squares	F value	p value
Popn	1	90.12	90.12	0.29	0.5940
Iso(Popn)	24	7411.86	308.83	600.71	0.0001
Rep(Popn*Iso)	78	40.10	0.51	1.96	0.0001
Cultivar	4	10.20	2.55	14.86	0.0114
Popn*Cv.	4	0.69	0.17	0.32	0.8644
Cv.*Iso(Popn)	96	51.61	0.54	2.05	0.0001
Cv.*Rep(Popn*Iso)	312	81.90	0.26		
Total	519				

Table 69. Analysis of variance of Telia(90)-Telia(1) for combined cultivars in the 1981 field study

Source	df	Sum of squares	Mean squares	F value	p value
Popn	1	512.62	512.62	1.90	0.1812
Iso(Popn)	24	6486.71	270.27	26.14	0.0001
Rep(Popn*Iso)	78	806.65	10.34	2.31	0.0001
Cultivar	4	51.51	12.88	3.07	0.1517
Popn*Cv.	4	16.80	4.20	0.41	0.8033
Cv.*Iso(Popn)	96	991.09	10.32	2.30	0.0001
Cv.*Rep(Popn*Iso)	312				
Total	519				

Table 70. Analysis of variance of Coefficient of Infection for combined cultivars in the 1981 field study

Source	df	Sum of squares	Mean squares	F value	p value
Popn	1	3711.26	3711.26	4.37	0.0474
Iso(Popn)	24	20394.78	849.78	7.34	0.0001
Rep(Popn*Iso)	78	9027.75	115.74	1.51	0.0076
Cultivar	4	5963.33	1490.83	16.11	0.0098
Popn*Cv.	4	370.08	92.52	1.21	0.3077
Cv.*Iso(Popn)	96	13956.66	145.38	1.91	0.0001
Cv.*Rep(Popn*Iso)	312	23914.62	76.65		
Total	519				

Table 71. Analysis of variance of Seed Weight for combined cultivars in the 1981 field study

Source	df	Sum of squares	Mean squares	F value	p value
Popn	1	1.50	1.50	0.42	0.5258
Iso(Popn)	22	78.80	3.58	10.95	0.0001
Rep(Popn*Iso)	168	54.99	0.32	133.56	0.0001
Cultivar	4	348.31	0.01	1379.88	0.0001
Popn*Cv.	4	0.25	0.06	9.51	0.7284
Cv.*Iso(Popn)	88	10.88	0.12	1.35	0.0246
Cv.*Rep(Popn*Iso)	672	61.63	0.09		
Total	959				

Table 72. Analysis of variance of Seed Weight Index for combined cultivars in the 1981 field study

Source	df	Sum of squares	Mean squares	F value	p value
Popn	1	0.001	1.540	0.09	0.9104
Iso(Popn)	22	0.342	0.155	16.42	0.0001
Cultivar	4	0.143	0.036	33.80	0.0024
Popn*Cv.	4	0.004	0.001	1.12	0.3541
Cv.*Iso(Popn)	88	0.813	0.009		
Total	119				

Table 73. Analysis of variance of Yield for combined cultivars in the 1981 field study

Source	df	Sum of squares	Mean squares	F value	p value
Popn	1	0.25	0.25	0.01	0.9801
Iso(Popn)	22	8791.47	366.31	7.95	0.0001
Rep(Popn*Iso)	168	8443.17	108.24	2.29	0.0001
Cultivar	4	12210.09	3052.52	360.75	0.0001
Popn*Cv.	4	33.85	8.46	0.25	0.9112
Cv.*Iso(Popn)	88	3023.91	31.49	1.56	0.0014
Cv.*Rep(Popn*Iso)	672	14774.95	21.98		
Total	959				

Table 74. Analysis of variance of Yield Index for combined cultivars in the 1981 field study

Source	df	Sum of squares	Mean squares	F value	p value
Popn	1	0.01	0.010	0.09	0.7610
Iso(Popn)	22	1.46	0.070	7.19	0.0001
Cultivar	4	1.27	0.320	68.35	0.0001
Popn*Cv.	4	0.01	0.012	0.50	0.7734
Cv.*Iso(Popn)	88	0.01	0.024		
Total	119				

in both populations accompanied by a significant Cv.*Iso interaction. The analyses of variance of individual cultivars gave results similar to those from the combined cultivar analyses. Population means, standard errors and R square values are given in Table 75.

Correlation coefficients of the sexual population are given in Table 76. As in the 1980 study, Latent Period displayed a weak association with Yield Index (r=0.41**). Telia(1) was weakly associated with Seed Weight Index (r=0.34**). In contrast to results of 1980, Telia(1)-LP exhibited a weak positive association with Seed Weight Index (r= 0.33**). Correlation coefficients of the asexual population are given in Table 77. Latent Period again showed a weak correlation with Yield Index (r=0.30*) and Telia(90)-Telia(1) displayed a weak negative association with Yield Index (r=-0.30*). Estimated heritability values of traits for combined cultivars are given in Table 78, and selected cultivars showing large population differences in certain traits are given in Table 79.

1980 and 1981 Combined Field Studies

The analyses of variance of traits of aggressiveness for combined locations and cultivars are given in Tables 80-87. Ten randomly selected isolates from each population of the 1981 study were omitted from the study to obtain balanced data for the combined location analyses. Significant differences between populations were again detected for Latent Period and significant Location effects were present for Latent Period and Seed Weight Index. The Loc*Iso effect was highly significant for each trait, while the Cv.*Iso interaction was nonsignificant in any

Table 75. Population means, standard errors, and R square values of traits of aggressiveness for combined varieites in the 1981 field study

Trait	Mean of Asex Popn	Standard error of Asex Popn	Mean of Sex Popn	Standard error Sex Popn	R square[a]
Latent Period	10.94	0.14	11.22	0.17	0.73
Telia(1)	20.62	0.73	19.73	0.64	0.74
Telia(90)	35.25	1.14	36.36	1.07	0.98
Telia(1)-LP	9.67	0.72	8.51	0.65	0.73
Telia(90)-LP	24.30	1.15	25.14	1.04	0.99
Telia(90)-Telia(1)	14.63	1.12	16.62	1.09	0.86
Yield	22.50	0.97	22.53	1.29	0.69
Yield Index	0.71	0.05	0.71	0.06	0.77
Seed Weight	3.87	0.09	3.79	0.09	0.89
Seed Weight Index	0.79	0.02	0.78	0.02	0.85
Coeff. of Infect.	48.04	2.25	42.67	2.37	0.69

[a]Values given for combined sexual and asexual populations.

Table 76. Correlation coefficients of traits of aggressiveness in the sexual population of the 1981 field study

Traits	Telia-(1)	Telia-(90)	Telia-(1)-LP	Telia-(90)-LP	Telia(90)-Telia(1)	Yield Index	Seed Wt. Index	Coeff. of Infect.
Latent Period	0.03	0.17	-0.23	0.01	0.15	0.41**	0.01	0.08
Telia(1)		0.29*	0.96**	0.30*	-0.31*	0.26*	0.34**	-0.04
Telia(90)			0.23	0.99**	0.81**	0.03	-0.02	0.01
Telia(1)-LP				0.28*	-0.35**	0.15	0.33**	-0.06
Telia(90)-LP					0.80**	-0.04	-0.02	-0.01
Telia(90)-Telia(1)						-0.14	-0.24	0.03
Yield Index							0.62**	-0.35**
Seed Weight Index								-0.49**

*,**Statistically significant at the 0.05 and 0.01 levels of probability, respectively.

Table 77. Correlation coefficients of traits of aggressiveness in the asexual population of the 1981 field study

Traits	Telia-(1)	Telia-(90)	Telia-(1)-LP	Telia-(90)-LP	Telia(90)-Telia(1)	Yield Index	Seed Wt. Index	Coeff. of Infect.
Latent Period	0.30*	0.09	0.11	-0.03	-0.11	0.30*	-0.01	-0.17
Telia(1)		0.36**	0.98**	0.33*	-0.30*	0.02	0.09	0.11
Telia(90)			0.36**	0.99**	0.77**	-0.02	-0.08	0.01
Telia(1)-LP				0.35**	-0.30*	-0.03	0.10	0.15
Telia(90)-LP					0.79**	-0.28*	-0.08	0.03
Telia(90)-Telia(1)						-0.30*	-0.15	-0.07
Yield Index							0.47**	-0.48**
Seed Weight Index								-0.48**

*,**Statistically significant at the 0.05 and 0.05 levels of probability, respectively.

Table 78. Estimated heritability values from the component analysis of variance of traits of aggressiveness for combined cultivars in the 1981 field study

Trait	Asex Popn	Sex Popn
Latent Period	76%	86%
Telia(1)	57%	80%
Telia(90)	98%	99%
Telia(1)-LP	77%	83%
Telia(90)-LP	98%	97%
Telia(90)-Telia(1)	95%	96%
Yield	70%	78%
Yield Index	70%	75%
Seed Weight	78%	85%
Seed Weight Index	71%	80%
Coeff. of Infect.	81%	76%

Table 79. Estimated heritability values from the component analysis of variance of certain traits of aggressiveness for selected cultivars in the 1981 field study

Cultivar	Trait	Asex Popn	Sex Popn
Richland	Seed Weight	33%	74%
Clintford	Latent Period	27%	92%
	Coeff. of Infection	22%	65%
Noble	Telia(1)	91%	52%
	Telia(1)-LP	90%	54%
	Latent Period	18%	77%

Table 80. Analysis of variance of Latent Period for combined cultivars of the 1980 and 1981 field studies

Source	df	Sum of squares	Mean squares	F value	p value
Loc	1	1421.29	1421.29	286.67	0.0331
Popn	1	57.76	57.76	7.08	0.0288
Loc*Popn	1	5.29	5.29	0.80	0.3973
Iso(Popn)	8	65.53	8.19	1.24	0.2764
Loc*Iso(Popn)	8	52.92	6.61	18.88	0.0001
Rep(Popn*Iso)	30	10.50	0.35	1.00	0.5000
Loc*Rep(Popn*Iso)	30	10.50	0.35	2.33	0.0471
Cultivar	4	1.08	0.27	5.40	0.0973
Loc*Cv.	4	0.78	0.19	0.86	0.5234
Popn*Cv.	4	0.21	0.05	0.25	0.9091
Loc*Popn*Cv.	4	0.33	0.08	0.38	0.8241
Iso*Cv.(Popn)	32	6.95	0.21	0.95	0.4801
Loc*Iso*Cv.(Popn)	32	7.13	0.22	1.46	0.0627
Loc*Rep*Cv.(Iso*Popn)	240	35.50	0.15		
Total	399				

Table 81. Analysis of variance of Telia(90)-Telia(1) for combined cultivars of the 1980 and 1981 field studies

Source	df	Sum of squares	Mean squares	F value	p value
Loc	1	2761.50	2761.50	1.33	0.7348
Popn	1	803.72	803.72	1.95	0.2003
Loc*Popn	1	2065.70	2065.70	2.37	0.1619
Iso(Popn)	8	3300.03	412.50	0.47	0.7850
Loc*Iso(Popn)	8	6960.07	870.00	91.97	0.0001
Rep(Popn*Iso)	30	169.32	5.64	0.60	0.6308
Loc*Rep(Popn*Iso)	30	283.82	9.46	4.07	0.0001
Cultivar	4	34.91	8.73	1.57	0.3077
Loc*Cv.	4	90.88	22.72	1.00	0.3126
Popn*Cv.	4	22.21	5.55	0.24	0.9128
Loc*Popn*Cv.	4	33.78	8.44	0.37	0.8273
Iso*Cv.(Popn)	32	736.37	23.01	1.01	0.3108
Loc*Iso*Cv.(Popn)	32	727.93	22.75	9.81	0.0001
Loc*Rep*Cv.(Iso*Popn)	240	557.10	2.32		
Total	399				

Table 82. Analysis of variance of Telia(1) for combined cultivars of the 1980 and 1981 field studies

Source	df	Sum of squares	Mean squares	F value	p value
Loc	1	1218.01	1218.01	1.58	0.3762
Popn	1	1082.41	1082.41	1.23	0.2998
Loc*Popn	1	772.84	772.84	1.16	0.3124
Iso(Popn)	8	7044.35	880.54	1.32	0.2473
Loc*Iso(Popn)	8	5319.45	664.93	78.50	0.0001
Rep(Popn*Iso)	30	172.40	5.74	0.68	0.4806
Loc*Rep(Popn*Iso)	30	254.10	8.47	3.34	0.0001
Cultivar	4	61.03	15.25	1.07	0.3825
Loc*Cv.	4	76.61	19.15	1.35	0.3104
Popn*Cv.	4	56.61	14.15	0.75	0.5625
Loc*Popn*Cv.	4	27.03	6.76	0.33	0.8545
Iso*Cv.(Popn)	32	600.30	18.76	0.92	0.4137
Loc*Iso*Cv.(Popn)	32	651.80	20.36	8.05	0.0001
Loc*Rep*Cv.(Iso*Popn)	240	607.00	2.53		
Total	399				

Table 83. Analysis of variance of Telia(1)-LP for combined cultivars of the 1980 and 1981 field studies

Source	df	Sum of squares	Mean squares	F value	p value
Loc	1	5270.76	5270.76	5.82	0.2418
Popn	1	1640.25	1640.25	2.01	0.1944
Loc*Popn	1	906.01	906.01	1.32	0.2839
Iso(Popn)	8	6541.11	817.64	1.19	0.3137
Loc*Iso(Popn)	8	5493.23	686.65	87.69	0.0001
Loc*Rep(Pop*Iso)	30	235.00	7.83	2.86	0.0001
Cultivar	4	57.74	14.43	1.01	0.3814
Loc*Cv.	4	81.04	20.26	1.11	0.3762
Popn*Cv.	4	57.70	14.25	0.78	0.5482
Loc*Popn*Cv.	4	29.14	7.28	0.35	0.8399
Iso*Cv.(Popn)	32	583.61	18.24	0.88	0.5014
Loc*Iso*Cv.(Popn)	32	660.07	20.63	7.53	0.0001
Loc*Rep*Cv.(Iso*Popn)	240	658.50	2.74		
Total	399				

Table 84. Analysis of variance of Yield for combined cultivars of the 1980 and 1981 field studies

Source	df	Sum of squares	Mean squares	F value	p value
Loc	1	1068.64	1068.64	5.37	0.7353
Popn	1	1068.64	1068.64	3.65	0.1048
Loc*Popn	1	199.14	199.14	0.51	0.5016
Iso(Popn)	6	1758.21	293.03	0.75	0.4204
Loc*Iso(Popn)	6	2338.58	389.76	8.00	0.0001
Rep(Popn*Iso)	56	2316.24	41.36	0.85	0.3879
Loc*Rep(Popn*Iso)	56	2725.54	48.67	2.04	0.0001
Cultivar	4	4467.99	1116.99	20.52	0.0032
Loc*Cv.	4	1238.10	309.52	6.17	0.0001
Popn*Cv.	4	217.73	54.43	1.20	0.3351
Loc*Popn*Cv.	4	248.98	62.24	1.24	0.3202
Iso*Cv.(Popn)	24	1085.68	45.24	0.90	0.3925
Loc.*Iso*Cv.(Popn)	24	1203.62	50.15	2.10	0.0001
Loc*Rep*Cv.(Iso*Popn)	448	10671.10	23.82		
Total	639				

Table 85. Analysis of variance of Yield Index for combined cultivars of the 1980 and 1981 field studies

Source	df	Sum of squares	Mean squares	F value	p value
Loc	1	0.726	0.726	6.20	0.6239
Popn	1	1.049	1.049	2.16	0.1920
Loc*Popn	1	0.117	0.117	0.19	0.6758
Iso(Popn)	6	2.915	0.486	0.80	0.4237
Loc*Iso(Popn)	6	3.642	0.607	15.17	0.0001
Rep(Popn*Iso)	56	2.220	0.040	0.78	0.4300
Loc*Rep(Popn*Iso)	56	2.874	0.051	2.12	0.0001
Cultivar	4	2.951	0.738	5.72	0.0785
Loc*Cv.	4	2.047	0.512	6.16	0.0001
Popn*Cv.	4	0.516	0.129	1.59	0.2087
Loc*Popn*Cv.	4	0.097	0.024	0.29	0.8806
Iso*Cv.(Popn)	24	1.945	0.081	0.97	0.3618
Loc*Iso*Cv.(Popn)	24	2.004	0.083	3.46	0.0001
Loc*Rep*Cv.(Iso*Popn)	448	10.891	0.024		
Total	639				

Table 86. Analysis of variance of Seed Weight Index for combined cultivars of the 1980 and 1981 field studies

Source	df	Sum of squares	Mean squares	F value	p value
Loc	1	0.0117	0.0117	117.00	0.0328
Popn	1	0.0047	0.0047	0.04	0.8525
Loc*Popn	1	0.0001	0.0001	0.01	0.9922
Iso(Popn)	6	0.7500	0.1250	0.69	0.6049
Loc*Iso(Popn)	6	1.0895	0.1816	15.38	0.0001
Rep(Popn*Iso)	56	0.7983	0.0142	1.20	0.2902
Loc*Rep(Popn*Iso)	56	0.6599	0.0118	2.14	0.0001
Cultivar	4	0.4151	0.1038	4.31	0.2316
Loc*Cv.	4	0.0563	0.0141	0.50	0.6113
Popn*Cv.	4	0.0966	0.0241	1.39	0.2685
Loc*Popn*Cv.	4	0.0623	0.0156	0.68	0.6111
Iso*Cv.(Popn)	24	0.4181	0.0174	0.62	0.5834
Loc*Iso*Cv.(Popn)	24	0.5477	0.0279	5.07	0.0001
Loc*Rep*Cv.(Iso*Popn)	448	2.4621	0.0055		
Total	639				

Table 87. Analysis of variance of Seed Weight for combined cultivars of the 1980 and 1981 field studies

Source	df	Sum of squares	Mean squares	F value	p value
Loc	1	35.36	35.36	1768.00	0.0001
Popn	1	0.01	0.01	0.01	0.9468
Loc*Popn	1	0.02	0.02	0.01	0.9577
Iso(Popn)	6	10.60	1.76	0.28	0.4873
Loc*Iso(Popn)	6	38.00	6.33	22.61	0.0001
Rep(Popn*Iso)	56	18.33	0.32	1.14	0.3958
Loc*Rep(Popn*Iso)	56	15.87	0.28	2.33	0.0001
Cultivar	4	201.94	50.48	98.98	0.0001
Loc*Cv.	4	2.53	0.63	1.75	0.2177
Popn*Cv.	4	2.03	0.51	1.16	0.3536
Loc*Popn*Cv.	4	1.47	0.37	1.03	0.4123
Iso*Cv.(Popn)	24	10.53	0.44	1.22	0.3825
Loc*Iso*Cv.(Popn)	24	8.57	0.36	3.00	0.0001
Loc*Rep*Cv.(Iso*Popn)	448	56.48	0.12		
Total	639				

analysis. The Loc*Rep(Popn*Iso) and Loc*Iso*Cv. interactions were significant, but this again may have been due to the relatively large degrees of freedom (240) for the error term. Population means, standard deviations, and R square values for the combined analyses are given in Table 88. The individual cultivar analyses gave results similar to those from the combined analyses except for Yield and Yield Index in the cultivar Clintford, as shown in Tables 89 and 90. Yield and Yield Index exhibited significant population differences while both traits continued the trend of significant Loc*Iso interactions. Population means, standard deviations, and R square values of Yield and Yield Index for Clintford are given in Table 91.

Correlation coefficients of the traits for combined locations and cultivars in the asexual population are given in Table 92. Latent Period and Telia(1) exhibited weak associations with Yield Index (r=0.43** and r=0.31*, respectively), while Telia(1), Telia(90), and Telia(1)-LP showed weak to moderate correlations with Seed Weight Index (r=0.58**, r=0.36*, and r=0.51**, respectively). Correlation coefficients of the traits for combined locations and cultivars in the sexual population are given in Table 93. Latent Period, Telia(90), and Telia(90)-Telia(1) were weakly associated with Seed Weight (r=-0.34*, r=-0.34*, and r=-0.37*, respectively). Correlation coefficients of traits for combined locations of the cultivar Clintford in the asexual populations are given in Table 94. Latent Period displayed a positive association with Yield (r=0.74**) and Yield Index (r=0.66**) and Telia(1) and Telia(1)-LP exhibited positive associations with Seed Weight (r=0.79*

Table 88. Population means, standard deviations, and R square values of traits of aggressiveness for combined cultivars in the 1980 and 1981 field studies

Trait	Mean of Asex Popn	Standard deviation	Mean of Sex Popn	Standard deviation	R square[a]
Latent Period	8.81	2.12	9.57	1.87	0.98
Telia(1)	23.15	8.51	19.92	2.92	0.97
Telia(90)	39.18	6.86	33.05	5.85	0.99
Telia(1)-LP	14.34	9.27	10.36	3.85	0.97
Telia(90)-LP	30.37	7.67	23.48	4.63	0.99
Telia(90)-Telia(1)	16.02	6.92	13.12	6.18	0.97
Seed Weight	4.11	0.73	4.14	0.66	0.86
Seed Weight Index	0.79	0.07	0.81	0.07	0.67
Yield	19.92	4.78	22.73	4.22	0.65
Yield Index	0.64	0.16	0.75	0.19	0.68

[a]Values for combined asexual and sexual populations.

Table 89. Analysis of variance of Yield for the cultivar Clintford of the 1980 and 1981 field studies

Source	df	Sum of squares	Mean squares	F value	p value
Loc	1	325.12	325.12	1.62	0.6327
Popn	1	639.03	639.03	6.40	0.0447
Loc*Popn	1	200.00	200.00	1.67	0.2444
Iso(Popn)	6	599.31	99.88	0.83	0.7116
Loc*Iso(Popn)	6	720.50	120.08	4.45	0.0009
Rep(Iso*Popn)	56	1479.37	26.42	0.98	0.5308
Loc*Rep(Iso*Popn)	56	1510.37	26.97		
Total	127				

Table 90. Analysis of variance of Yield Index for the cultivar Clintford of the 1980 and 1981 field studies

Source	df	Sum of squares	Mean squares	F value	p value
Loc	1	1.6423	1.6423	2346.14	0.0001
Popn	1	0.9262	0.9262	10.44	0.0179
Loc*Popn	1	0.0007	0.0007	0.01	0.9503
Iso(Popn)	6	0.5325	0.0887	0.57	0.4325
Loc*Iso(Popn)	6	0.9340	0.1557	6.62	0.0001
Rep(Iso*Popn)	56	1.3969	0.0229	0.94	0.5860
Loc*Rep(Iso*Popn)	56	6.9193	0.0235		
Total	127				

Table 91. Population means of Yield and Yield Index for the cultivar Clintford of the 1980 and 1981 field studies

Trait	Population	Isolate means	Standard deviation	R square[a]
Yield	Asexual	19.72	4.05	0.72
	Sexual	24.19	3.54	
Yield Index	Asexual	0.62	0.16	0.79
	Sexual	0.79	0.13	

[a]Values given for combined sexual and asexual populations.

Table 92. Correlation coefficients of traits of aggressiveness for combined cultivars in the asexual population of the 1980 and 1981 field studies

Traits	Telia-(1)	Telia-(90)	Telia-(1)-LP	Telia-(90)-LP	Telia(90)-Telia(1)	Seed Wt.	Seed Wt. Index	Yield	Yield Index
Latent Period	-0.25	-0.25	-0.46**	-0.50**	0.06	-0.24	0.07	0.43**	0.34*
Telia(1)		0.61**	0.97**	0.62**	-0.62*	0.38**	0.58*	-0.01	0.31*
Telia(90)			0.62**	0.96**	0.24	0.23	0.36*	0.19	0.20
Telia(1)-LP				0.68**	-0.58**	0.41**	0.51**	-0.10	0.21
Telia(90)-LP					0.19	0.27	0.30*	0.05	0.09
Telia(90)-Telia(1)						0.23	-0.34*	0.19	-0.17
Seed Weight							0.79**	0.46**	0.49*
Seed Weight Index								0.49**	0.62**
Yield									0.85**

*,**Statistically significant at the 0.05 and 0.01 levels of probability, respectively.

Table 93. Correlation coefficients of traits of aggressiveness for combined cultivars in the sexual population of the 1980 and 1981 field studies

Traits	Telia-(1)	Telia-(90)	Telia-(1)-LP	Telia-(90)-LP	Telia(90)-Telia(1)	Seed Wt.	Weed Wt. Index	Yield	Yield Index
Latent Period	-0.26	0.74**	-0.68**	0.54**	0.82**	-0.34*	-0.14	0.16	0.01
Telia(1)		0.13	0.88**	0.27	-0.34*	0.11	0.07	0.01	0.01
Telia(90)			-0.26	0.96**	0.88**	-0.34*	-0.06	0.11	0.04
Telia(1)-LP				0.05	-0.66**	0.25	0.11	-0.07	0.01
Telia(90)-LP					0.78**	-0.29	-0.02	0.09	0.05
Telia(90)-Telia(1)						-0.37*	-0.09	0.11	0.03
Seed Weight							0.57*	0.68**	0.43**
Seed Weight Index								0.55**	0.37**
Yield									0.64**

*,**Statistically significant at the 0.05 and 0.01 levels of probabilty, respectively.

Table 94. Correlation coefficients of traits of aggressiveness for the cultivar Clintford in the asexual population of the 1980 and 1981 field studies

Traits	Telia-(1)	Telia-(90)	Telia-(1)-LP	Telia-(90)-LP	Telia(90)-Telia(1)	Seed Wt.	Weed Wt. Index	Yield	Yield Index
Latent Period	-0.26	-0.30	-0.46	-0.53	0.02	-0.23	0.48	0.74*	0.66*
Telia(1)		0.61*	0.97**	0.61*	-0.61*	0.79**	0.69*	-0.14	0.01
Telia(90)			0.63*	0.96**	0.25	0.30	0.29	-0.08	-0.16
Telia(1)-LP				0.68*	-0.57*	0.78*	0.52	-0.31	-0.15
Telia(90)-LP					0.21	0.33	0.13	-0.28	-0.32
Telia(90)-Telia(1)						-0.64*	-0.52	0.08	-0.17
Seed Weight							0.68*	0.06	0.07
Seed Weight Index								0.30	0.38
Yield									0.93**

*,**Statistically significant at the 0.05 and 0.01 levels of probability, respectively.

and r=0.78**, respectively). Telia(1) was positively associated with Seed Weight Index (r=0.69*), while Telia(90)-Telia(1) showed a negative association with Seed Weight (r=-0.64*). Correlation coefficients of traits for combined locations of the cultivar Clintford in the sexual population are given in Table 95. Latent Period was again positively associated with Yield Index (r=0.88**). In contrast to the asexual population, Telia(90) was negatively correlated with Seed Weight (r=-0.69**) and positively associated with Yield Index (r=0.63**). Telia(1)-LP also showed differences from the sexual population by a negative correlation with Yield Index (r=-0.83**). As in the sexual population, Telia(90)-Telia(1) exhibited a negative association with Seed Weight (r=-0.66*), but showed a contrast by a positive correlation with Yield Index (r=0.72*).

Heritability estimates of the "visual" traits for combined locations of individual cultivars are given in Tables 96 and 97. Estimates for most traits were much lower than in the individual year analyses. The sharp reduction in estimated values was attributed to the strong Location and Loc*Iso effects, as seen in the combined analyses of variance and the combined variance component analyses (data not presented). Some traits exhibited negative values for the variance component, Iso(Popn), and estimates of heritability were, therefore, less than zero in these cases. We have used zero values in place of these negative estimates of Iso(Popn) as one alternative suggested by Searle (40), which resulted in zero values for heritability of several traits in the sexual population and for Telia(90)-Telia(1) in the asexual population. A maximum

Table 95. Correlation coefficients of traits of aggressiveness for the cultivar Clintford in the sexual population of the 1980 and 1981 field studies

Traits	Telia-(1)	Telia-(90)	Telia-(1)-LP	Telia-(90)-LP	Telia(90)-Telia(1)	Seed Wt.	Weed Wt. Index	Yield	Yield Index
Latent Period	-0.47	0.76**	-0.80**	0.52	0.77**	-0.50	0.16	0.22	0.88**
Telia(1)		-0.40	0.90**	-0.30	-0.71*	0.33	0.26	-0.09	-0.60
Telia(90)			-0.64*	0.95**	0.93**	-0.69*	-0.03	-0.06	0.63*
Telia(1)-LP				-0.45	-0.85**	0.47	0.10	-0.16	-0.83**
Telia(90)-LP					0.85**	-0.70*	-0.11	-0.20	0.43
Telia(90)-Telia(1)						-0.66*	-0.12	-0.01	0.72*
Seed Weight							0.61*	0.68*	-0.18
Seed Weight Index								0.79**	0.33
Yield									0.44

*,**Statistically significant at the 0.05 and 0.01 levels of probability, respectively.

Table 96. Estimated heritability values for "visual" traits of aggressiveness of individual cultivars in the 1980 and 1981 field studies

Cultivar	Trait	Asex Popn	Sex Popn
Richland	Latent Period	7.3%	0%[a]
	Telia(1)	19.3%	0%
	Telia(90)	63.8%	0%
	Telia(1)-LP	11.6%	0%
	Telia(90)-LP	46.3%	0%
	Telia(90)-Telia(1)	0%	0%
Clintford	Latent Period	6.5%	0%
	Telia(1)	8.7%	0%
	Telia(90)	63.8%	0.9%
	Telia(1)-LP	0.9%	0%
	Telia(90)-LP	45.2%	11.8%
	Telia(90)-Telia(1)	0%	0%
Lang	Latent Period	1.2%	0%
	Telia(1)	23.9%	66.5%
	Telia(90)	64.0%	0%
	Telia(1)-LP	11.4%	28.3%
	Telia(90)-LP	46.2%	0%
	Telia(90)-Telia(1)	0%	8.7%

[a]Zero values indicate the variance component for Iso(Popn) was negative.

Table 97. Estimated heritability values for "visual" traits of aggressiveness of individual cultivars in the 1980 and 1981 field studies

Cultivar	Trait	Asex Popn	Sex Popn
Noble	Latent Period	6.3%	0%[a]
	Telia(1)	41.6%	53.1%
	Telia(90)	63.6%	0%
	Telia(1)-LP	25.8%	31.6%
	Telia(90)-LP	44.9%	0%
	Telia(90)-Telia(1)	0%	0%
Otee	Latent Period	6.4%	0%
	Telia(1)	0%	25.6%
	Telia(90)	76.1%	0%
	Telia(1)-LP	0%	0%
	Telia(90)-LP	57.2%	0%
	Telia(90)-Telia(1)	0%	0%

[a]Zero values indicate the variance component for Iso(Popn) was negative.

likelihood procedure for estimating variance components was also attempted with a computer whereby components were calculated by an iterative scheme that tried to minimize an "objective function". These estimates were unstable over several iterations and the procedure was discontinued since additional computer time would have been cost prohibitive.

Heritability estimates for "yield" traits in the combined locations of individual cultivars are given in Table 98. The estimates were again much lower than the single year analyses. Given the very approximate nature of the estimation procedure for heritabilities, no strong population differences were detected for any trait. Two possible exceptions were Seed Weight Index (60%, sexual population vs. 0%, asexual population) with Lang and Yield Index (59%, sexual population vs. 0%, asexual population) with Otee.

Table 98. Estimated heritability values for "yield" traits of aggressiveness of individual cultivars in the 1980 and 1981 field studies

Cultivar	Trait	Asex Popn	Sex Popn
Richland	Seed Weight	0%[a]	0%
	Seed Weight Index	0%	0%
	Yield	0%	23.6%
	Yield Index	0%	37.1%
Clintford	Seed Weight	0%	0%
	Seed Weight Index	0%	0.9%
	Yield	7.7%	0%
	Yield Index	0%	10.0%
Lang	Seed Weight	0%	23.6%
	Seed Weight Index	0%	60.3%
	Yield	19.9%	0%
	Yield Index	55.6%	75.0%
Noble	Seed Weight	0%	33.5%
	Seed Weight Index	0%	46.8%
	Yield	43.9%	0%
	Yield Index	0%	0%
Otee	Seed Weight	0%	0%
	Seed Weight Index	0%	39.8%
	Yield	0%	0%
	Yield Index	0%	59.2%

[a]Zero values indicate the variance component for Iso(Popn) was negative.

DISCUSSION

Growth Chamber Study

Significantly higher mean square values for several traits in the sexual population suggest that gene recombination provides an "immediate" potential advantage in an optimum environment by producing a wider range of phenotypes and some offspring with higher levels of aggressiveness than would occur with asexual reproduction. In addition, increased aggressiveness through recombination in the sexual population appears to overcome the potential disadvantage of additional energy expenditure and reduced population size on the alternate host, R. cathartica. The advantage of recombination detected in the large (in the uredial spore stage) sexual population conforms to the predictive models of Crow and Kimura (8) and/or Maynard Smith (25). However, large reductions in population size in the spermatial and aecial spore stages may confer an advantage to recombination in a finite sexual population under random genetic drift (15).

If we assume that loci in the asexual population are tightly linked, then the model by Lewontin (23) of maximum fitness under tight linkage does not hold for the sexual population. A "balanced" or optimum level of recombination in the sexual population probably occurs to satisfy the needs of immediate and long-term adaptation (24). That the level of recombination for traits of aggressiveness is largely under genetic control in the growth chamber is evidenced by the heritability estimates in both populations. These estimates suggest that the rate of response for

increased levels of aggressiveness is potentially high for both populations and that the longevity of "horizontal resistance" in the host population is not permanent under a stable physical environment.

Estimated numbers of effective factors suggest the quantitative nature of traits of aggressiveness which coincides with data in a review by Nelson (34). Possible exceptions are Latent Period, Telia(1), and Telia(1)-LP, which exhibit discrete data and are probably simply inherited.

Frequency distributions shown in Figures 2 and 3 indicate selection for an intermediate number of virulence genes in both populations. This type of data would seem to provide evidence against Vanderplank's theory of stabilizing selection. The evidence is inadequate, however, since complementary gene frequencies in the primary and alternate hosts are unknown and different host gene frequencies associated with each pathogen population may bias the comparison (39). Appropriate data to help explain the evolution of virulence genes may, therefore, be difficult to obtain solely from race surveys.

Different correlations between populations for the same trait suggest the possibility of different strategies and selection pressures for adaptation to the Minnesota and Texas environments through accumulation of different gene complexes. In this regard, the direct comparison of the two populations is weakened. Another drawback is that although phenotypes at the virulence loci suggest minimal or non-existent levels of recombination in the Texas isolates, small undetected amounts of recombination may maintain certain levels of polymorphism in

the "asexual" population (6).

Field Study

In contrast to the general results obtained in the growth chamber studies, the asexual population exhibited significantly higher levels of aggressiveness than the sexual population for certain traits. The potential advantages of gene recombination for all traits in the sexual population were not expressed under the particular plot conditions in the field studies. Fluctuating environments may at first seem to favor high levels of gene recombination, but this potential advantage will be expressed only when fitnesses of genotypes in a population change drastically from one generation to the next (4, 25). None of the isolates in either population behaved, at least from visual observation, in this manner. All of the data support the general conclusion that gene recombination provided no significant advantage to the sexual population in the origin and maintenance of aggressiveness sometime prior to 1979. This lack of an advantage to recombination in the large pathogen populations conforms most closely to the model of Felsenstein (15). Whether or not the potential advantages to recombination in the sexual population will occur in the future is discussed below. The contrasting results of the growth chamber and field studies cannot be properly compared, since no cultivar was common to both experiments. The cultivar Markton, used in the growth chamber, is unadapted to Iowa growing conditions.

The higher levels of aggressiveness detected for some traits in the asexual population are not conclusive evidence for any general

disadvantage to recombination in the sexual population. The high levels, however, were associated with specific cultivars and suggest the instability to some degree of levels of aggressiveness under changing susceptible host populations. Cultivar specificity for levels of aggressiveness can be seen in Clintford, which exhibited significant population differences for Yield and Yield Index, but such differences did not exist for the other four cultivars. The absence of a significant Cv.*Iso interaction may seem to weaken this conclusion, but its importance, discussed in the Results section, is strongly influenced by the statistical design and size of the experiment. The significant Loc*Iso interaction present for all traits indicates the dependence of levels of aggressiveness on the physical environment.

The sample size of each pathogen population was quite small, but was large enough to detect population differences for some traits. There are two points, however, that may weaken the comparisons between populations of these types. Of most importance is whether the sample size is indeed representative of the two pathogen populations. That is, could a larger sample reveal drastically different population structures? Another aspect is that the pathogen populations occur in distinct environments and different selection pressures may bias the results. Lack of appropriate markers in the host and pathogen make it difficult to accurately determine how this affects the comparisons.

Correlations between most traits varied from one year to the next and were clearly dependent upon the different location environments. Lack of consistent associations between the traits will make it difficult

to better characterize the mechanisms of aggressiveness in pathogen populations. Similar results were reported in a review of components of horizontal resistance (37). One fairly consistent correlation did occur, however, with Latent Period and Yield Index in all analyses. Parlevliet (36) has reported Latent Period to be one of the most effective traits for estimating the level of horizontal resistance in a Puccinia hordei-barley system. It is interesting to note that as an estimate of the amount of disease present near the end of the season in 1981, the Coefficient of Infection was not associated with any "visual" trait which was the case with Latent Period and Yield Index as noted above. If we assume that the "visual" traits are associated with aggressiveness of an isolate, then Coefficient of Infection was no better an estimator of fitness than Yield Index in 1981. Early telia formation has been commonly considered a sign of shorter sporulation periods and subsequent reductions in levels of aggressiveness. Yet no telia trait in either population of this study exhibited consistent correlations with aggressiveness (i.e. with Yield Index, Seed Weight Index, or Coefficient of Infection). Indeed, some telia traits exhibited positive associations with aggressiveness, most notably in the asexual population, which suggests that in some instances "late" formation of telia is correlated with reduced levels of aggressiveness.

Heritability estimates were consistently high for several traits with each year, but the estimates varied between years for certain traits and cultivars. For example, heritability of Latent Period in Noble was nearly 98% in 1980 but dropped to 18% in 1981. In contrast, heritability

of Latent Period in Otee remained high in both years. More importantly, large population differences for heritability estimates were not consistent for any trait from one year to the next.

The combined analyses for heritability estimates provide a clearer picture. If we consider the two field locations as different environments ("dry" in 1980 and "wet" in 1981), then the genotype x environment interaction significantly contributes to the much lower and more variable heritability estimates in the combined analyses. The genotype x environment interaction was reported as significant in the heritability of aggressiveness in Ustilago hordei (10). Low heritability estimates for aggressiveness follow the pattern of estimates for reproductive fitness in various organisms (12).

The wide range of heritability values for most traits in the combined analyses suggests that response to increased levels of aggressiveness in both populations will vary for each trait and cultivar. While no consistent advantage in heritability was detected for either population, the sexual isolates exhibited a potentially greater response for increased aggressiveness in Seed Weight Index on Lang and Yield Index on Otee. This suggests that the sexual population has a greater future potential to exhibit significantly higher levels of aggressiveness than the asexual population on host genotypes similar to Lang and Otee. Heritability values of the "visual" traits were not always indicative of estimates of the "yield" traits. For example, the relatively high estimates of Seed Weight Index and Yield Index in the sexual population for Otee did not correspond with the low "visual" estimates. This probably

occurred because the response of any one trait could not account for the overall heritability of aggressiveness. How the interaction of traits affects their individual relative contribution to aggressiveness is also unknown. If trait response, cultivar specificity, and genotype x environment interactions do indeed contribute to reduce levels of aggressiveness, then deployment of genes for "horizontal resistance" in different cultivars and locations may prove effective in lowering fitness in the asexual population. This would be most easily accomplished by cultivar mixtures. Other factors not considered in this study may weaken this conclusion (1). In view of relatively higher estimated rates of increased levels of aggressiveness in the sexual population, the longevity of "horizontal resistance" in Lang, Otee, and other cultivars with similar responses may be reduced when high levels of gene recombination occur in the crown rust pathogen.

SUMMARY

The following conclusions were made from the study.

(1) The potential advantages of gene recombination to produce significantly higher levels of aggressiveness were expressed by the sexual population of P. coronata in the growth chamber.

(2) Gene recombination provided no consistent advantage, however, to the sexual population in two field plot locations. This may be due to the absence of the effects of genetic drift in the two large pathogen populations.

(3) Low heritability values for most traits over combined locations in the field study were primarily due to significant genotype x environment interactions.

(4) The general response to selection for increased levels of aggressiveness in the asexual population was not large.

(5) Potentially higher levels of aggressiveness were detected in the sexual poulation on the cultivars Lang and Otee which suggests that the advantage of gene recombination will vary over changing host populations and fluctuating environments.

LITERATURE CITED

1. Barrett, J. A. 1980. Pathogen evolution in multilines and variety mixtures. J. Plant Dis. and Protect. 87:383-386.

2. Bishop, Y. M., Fienberg, S. E., and Holland, P. W. 1975. Discrete Multivariate Analysis. MIT Press, Cambridge, Mass. 238 pp.

3. Bodmer, W. F. and Parsons, P. A. 1962. Linkage and recombination in evolution. Adv. Genet. 11:1-100.

4. Charlesworth, B. 1976. Recombination modification in a fluctuating environment. Genetics 83:181-185.

5. Charlesworth, B., and Charlesworth, D. 1976. An experiment on recombination load in Drosophila melanogaster. Genet. Res. 25: 267-274.

6. Clegg, M. T., Allard, R. W., and Kahler, A. L. 1972. Is the gene the unit of selection? Evidence from two experimental plant populations. Proc. Natl. Acad. Sci. 69:2474-2478.

7. Croft, J. H., and Simchen, G. 1965. Natural variation among monokaryons of Collybia velutipes. Am. Nat. 99:451-462.

8. Crow, J. F., and Kimura, M. 1965. Evolution in sexual and asexual populations. Am. Nat. 99:439-450.

9. Emara, Y. A. 1972. Genetic control of aggressiveness in Ustilago hordei. I. Natural variability among physiological races. Can. J. Genet. Cytol. 14:919-924.

10. Emara, Y. A., and Freake, G. W. 1981. Effect of environment and genotype and their interaction on pathogenicity of Ustilago hordei. J. Hered. 72:261-263.

11. Emara, Y. A., and Sidhu, G. 1974. Polygenic inheritance of aggressiveness in Ustilago hordei. Heredity 32:219-224.

12. Falconer, D. S. 1960. Introduction to Quantitative Genetics. Ronald Press, New York, N.Y. 365 pp.

13. Feldman, M. W. 1972. Selection for linkage modification: Random mating populations. Theoret. Pop. Biol. 3:324-346.

14. Felsenstein, J. 1965. The effect of linkage on directional selection. Genetics 52:349-363.

15. Felsenstein, J. 1974. The evolutionary advantage of recombination. Genetics 78:737-756.

16. Fisher, R. A. 1958. The Genetical Theory of Natural Selection. Second Revised Edition. Dover Pub., New York, N.Y. 291 pp.

17. Hill, J. P., and Nelson, R. R. 1982. The heritability of three parasitic fitness attributes of Helminthosporium maydis race T. Phytopathology 72:525-528.

18. Hill, W. G., and Robertson, A. 1966. The effect of linkage on limits to artificial selection. Genet. Res. Camb. 8:269-294.

19. Kimura, M. 1965. Attainment of quasi linkage equilibrium when gene frequencies are changing by natural selection. Genetics 52: 875-890.

20. Langley, C. H. 1977. Nonrandom associations between allozymes in natural populations of Drosophila melanogaster. pp. 265-273 in F. B. Christiansen and T. M. Fenchel, eds. Measuring Selection in Natural Populations. Springer-Verlag, Berlin.

21. Lewontin, R. C., and Kojima, M. 1960. The evolutionary dynamics of complex polymorphisms. Evolution 14:458-472.

22. Lewontin, R. C. 1964. The interaction of selection and linkage. I. General considerations. Heterotic models. Genetics 49:49-67.

23. Lewontin, R. C. 1974. The Genetic Basis of Evolutionary Change. Columbia Univ. Press, New York, N.Y. 346 pp.

24. Mather, K. 1943. Polygenic inheritance and natural selection. Biol. Rev. 18:32-64.

25. Maynard-Smith, J. 1971. What use is sex? J. Theoret. Biol. 30: 319-335.

26. Maynard Smith, J. 1976. A short term advantage for sex and recombination through sib competition. J. Theoret. Biol. 63:245-258.

27. Maynard Smith, J. 1978. The Evolution of Sex. Cambridge Univ. Press, Cambridge. 222 pp.

28. Mortenson, K., and Green, G. J. 1978. Assessment of receptivity and urediospore production as components of wheat stem rust resistance. Can J. Botany 56:1827-1839.

29. Mukai, T., Mettler, E., and Chigusa, S. I. 1970. On the linkage equilibrium of isozymes in a Raleigh, N.C. population of Drosophila melanogaster. Drosophila Inform. Serv. 45:77.

30. Muller, H. J. 1932. Some genetic aspects of sex. Am. Nat. 66: 118-138.

31. Muller, H. J. 1964. The relation of recombination to mutational advance. Mutat. Res. 1:2-9.

32. Nei, M. 1967. Modification of linkage intensity by natural selection. Genetics 57:625-641.

33. Nei, M. 1969. Linkage modification and sex difference in recombination. Genetics 63:681-699.

34. Nelson, R. R. 1978. Genetics of horizontal resistance to plant diseases. Ann. Rev. Phytopathol. 16:359-378.

35. Parlevliet, J. E. 1977. Evidence of differential interaction in the polygenic Hordeum vulgare-Puccinia hordei relation during epidemic development. Phytopathology 67:776-778.

36. Parlevliet, J. E. 1978. Race specific aspects of polygenic resistance of barley to leaf rust, Puccinia hordei. Neth. J. Plant Pathol. 84:121-126.

37. Parlevliet, J. E. 1979. Components of resistance that reduce the rate of epidemic development. Ann. Rev. Phytopathol. 17:203-222.

38. Pielou, E. C. 1969. An Introduction to Mathematical Ecology. Wiley Science, New York., N.Y. 286 pp.

39. Roelfs, A. P., and Groth, J. V. 1980. A comparison of virulence phenotypes in wheat stem rust populations reproducing sexually and asexually. Phytopathology 70:855-862.

40. Searle, S. R. 1971. Linear Models. Wiley Science, New York, N.Y. 532 pp.

41. Simons, M. D., Rothman, P. G., and Michel, L. J. 1979. Pathogenicity of Puccinia coronata from buckthorn and from oats adjacent to and distant from buckthorn. Phytopathology 69:156-158.

42. Stakman, E. C., Stewart, D. M., and Loegering, W. Q. 1962. Identification of physiological races of Puccinia graminis var. tritici. U.S. Dept. Agric. Special Pub. E617. 53 pp.

43. Statler, G. D., and Jones, D. A. 1981. Inheritance of virulence and uredial color and size in Puccinia recondita tritici. Phytopathology 71:652-655.

44. Turner, J. R. 1967. Why does the genome not congeal? Evolution 21:645-656.

45. Wahl, I., Dinoor, A., Halperin, J., and Schreiter, S. 1960. The effect of Rhamnus palestina on the origin and persistence of oat crown rust races. Phytopathology 50:562-567.

46. Williams, G. C. 1975. Sex and Evolution. Princeton Univ. Press, Princeton, N.J. 200 pp.

47. Yamaguchi, O., Motoshi, I., Matsuda, M., and Mukai, T. 1980. Linkage disequilibrium in isolated populations of Drosophila melanogaster. Genetics 96:507-522.

48. Yarwood, C. E. 1961. Uredospore production by Uromyces phaseoli. Phytopathology 51:22-27.

ACKNOWLEDGMENTS

The author wishes to express his thanks and appreciation to:

Dr. Marr Simons, my major advisor, for his efforts and consideration in the last three years and his helpful suggestions for improvement of the manuscript.

Leonard Michel for his training, suggestions, and support in the field and greenhouse.

Pete Christiansen for his interest in rusts and assistance in the statistical modeling and analysis.

The Quaker Oats Company, represented by Don Schrickel and Sam Weaver, for their encouragement and financial support.

J.H.O.