Nature and Inheritance of Resistance to Gymnosporangium juniperi-virginianae in Apple Cultivars

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ABSTRACT

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Fifty-eight apple (*Malus pumila*) cultivars and numbered selections were inoculated artificially with a population of *Gymnosporangium juniperi-virginianae* (the incitant of cedar apple rust) of wide-host-range. The most advanced reaction (chlorotic mottle, flecks, pycnia, or aecia) was recorded for each cultivar, and maximum and mean infection ratings for each cultivar were determined. Analysis of 15 progenies from crosses of selections derived from *M. pumila*, *M. floribunda*, *M. prunifolia*, and *M. toringo* supports the hypothesis that resistance (absence of pycnia) is controlled by two genes. The genes, designated *Gy-a* and *Gy-b* are phenotypically indistinguishable. Both genes are required in dominant form for suppression of pycnia. Leaf damage, expressed as infection ratings, is not predicted reliably by the dual-gene hypothesis and is probably controlled by *Gy-a*, *Gy-b*, and several modifying genes. A virulence formula similar to those used for wheat rusts is proposed to describe the pathogenicity of a given population of *G. juniperi-virginianae*. A list of sequential numbers for current differential cultivars is presented. Virulence formulae already observed have been assigned formula numbers.

Additional key words: apple cultivars, apple breeding, *Malus sylvestris*.

Thomas and Mills (27) and Miller (17) showed that conflicting reports on the susceptibility of apple [*Malus pumila* Miller (also called *M. sylvestris*)] cultivars to cedar apple rust (*Gymnosporangium juniperi-virginianae* Schw.) could be due to confusion with infections incited by hawthorn rust (*G. globosum*) or quince rust (*G. clavipes*). Many early lists of cultivar susceptibility (6, 10, 23), therefore, must be regarded with caution. Furthermore, Aldwinckle (2) demonstrated that the frequency and type of symptoms were affected by inoculum concentration and age of apple leaves. The existence of populations of *G. juniperi-virginianae*, differing in pathogenicity, was suggested by Bliss (4), Crowell (7), and McNew (16) and was confirmed by Aldwinckle (3). The confusion with other apple rusts and the existence of populations differing in pathogenicity limit the value of observations of natural infections (8, 21) and experimental inoculations where inoculum concentration was neglected (4, 16).

Resistance of apple cultivars to *G. juniperi-virginianae* has been characterized as absence of pycnia and aecia (22, 23), absence of aecia (4, 18, 20), or a limit of one to five aecia per sorus (7). Bliss (4) and Niederhauser and Whetzel (21) used five groups: immune (no symptoms); very resistant (flecks only); resistant (pycnia, but no aecia); susceptible (few aecia); and very susceptible (many aecia). Aldwinckle (3) differentiated pathogenic races of *G. juniperi-virginianae* by the apple cultivars on which they incited aecia. To rate infections from artificial inoculation, Aldwinckle (2) developed a formula based on the number and diameter of lesions on any leaf. This infection rating correlated well with field observations and could be measured objectively and easily.

In this paper, lesion types and infection ratings resulting from the inoculation of 58 cultivars and numbered selections with a wide-host-range population of *G. juniperi virginianae* are reported.

Moore (19) and Shay and Hough (24) postulated that inheritance of resistance was conditioned by a single dominant gene for which Knight (14) suggested the symbol *Gy*. Mowry (20), however, concluded that susceptibility (production of aecia) was governed by two recessive genes, *g* and *j*, with "duplicate recessive interaction between gene pairs". Either of the genes in the double recessive form would condition aecial production. He reinterpreted the data of Moore (19) and Bradford (5) to fit this hypothesis. No information is available on the pathogenicity of the population of the fungus in any of these studies.

It was of interest to determine whether rust resistance (absence of pycnia) was inherited similarly in progenies from the Geneva disease-resistant apple breeding program.

Pathogenicity of Gymnosporangium juniperi-virginianae.—In an earlier paper, the race concept in the

apple/*G. juniperi-virginianae* system was based on the sum of pathogenicities of the segregates (basidiospores) in the population (3). "Races" in this sense cannot be maintained or reproduced. To clarify this situation, we propose using the virulence formula method, which was introduced by Green (11, 12) for classifying *Puccinia graminis tritici* races, as modified by Loegering and Browder (15) for *Puccinia recondita tritici*, to characterize the pathogenicity of populations of *G. juniperi-virginianae*. Each differential apple cultivar will be given a sequential number (SN). The SN's of those cultivars used to date are given in Table 1; SN's can be assigned to additional cultivars as necessary. The SN's are used to construct virulence formulae. The SN's of the apple cultivars that are effective in conditioning an incompatible host-parasite interaction (absence of aecia) with a given population of *G. juniperi-virginianae* form the numerator of the formula, and the SN's of the ineffective cultivars the denominator (Table 2). A given *G. juniperi-virginianae* population receives a virulence formula according to its interaction with the differential cultivars. Virulence formulae are assigned formula numbers for convenience (Table 2). Formula numbers 1 to 5 correspond with the "race" numbers previously used (3).

MATERIALS AND METHODS

Apple cultivars.—Apple cultivars and numbered selections were bench-grafted on seedling rootstocks and stored at 2 C until required. They were potted in a loam:sand:peat mix (1:1:1, v/v) in 13-cm diameter plastic pots, grown in a 23 ±3 C greenhouse and trained to single shoots, which were inoculated when approximately 30 cm long using a modification of the method of Szkolnik (26).

Inoculum.—Galls of *G. juniperi-virginianae* were collected from eastern red cedar near Dresden, New York after partially extended telial horns had dried naturally, and were stored at −17 C. When required for inoculation, galls were thawed, soaked in water for 30 min and kept in an 18.5 ±2 C chamber at 100% relative humidity for 15 hr. Re-extruded horns were rinsed with distilled water to obtain a suspension of basidiospores standardized to 267,000 spores/ml with a hemacytometer. This concentration was adequate to eliminate any influence of low inoculum level on symptom type (2).

Inoculation.—Freshly prepared inoculum suspension was sprayed on the six youngest leaves of each plant to produce a uniform coating of droplets about 2 mm in diameter, and on glass slides coated with 'Parlodion' (Mallinckrodt Chemicals, St. Louis, Missouri). Plants and slides were placed in an illuminated (1,600 lux) 15.5 ±2 C chamber at 100% relative humidity for 48 hr. The plants then were returned to the 23 C greenhouse. Basidiospore germination on glass slides was determined microscopically.

Symptoms.—Symptoms began to appear on leaves approximately 10 days after inoculation. In a fully compatible host-pathogen interaction, disease development progressed through three stages: (i) invasion and hyphal proliferation resulting in a nonsporulating lesion that was either small and discrete (N) or a more diffuse, chlorotic mottle (Mc), (ii) formation of pycnia (P) within the lesions; and (iii) formation of aecia (A). In incompatible interactions, no macroscopic symptoms (0) or only the first or second stages appeared. The presence of nonsporulating or pycnial lesions was scored at 3 wk and of aecial lesions at 10 wk after inoculation. An infection rating of $\log_{10}(10nd^2)$, where n is the highest number of lesions per leaf and d is the largest mean diameter of lesions on any leaf was calculated for each plant (2).

Cultivar tests.—Using these techniques from 1972 to 1974, three-to-five plants of each cultivar or numbered selection were inoculated in two to seven independent tests. The most advanced reaction, the maximum infection rating of any plant in any test, and the mean infection rating from all tests were determined.

Seedling tests.—Crosses were made among some of the cultivars and numbered selections in 1972 and 1973. Seedlings from the crosses were grown in the greenhouse and inoculated in the same way as the parents. The 1972 and 1973 seedlings were inoculated when 12 and 8-10 wk old, respectively; the incidence of pycnia and infection ratings were determined.

Statistical comparison of segregation of 1972 seedlings for resistance (absence of pycnia) with hypothetical ratios were made using the chi-square test with Yates correction for continuity.

TABLE 1. Sequential numbers assigned to apple cultivars as differentials to identify populations of *Gymnosporangium Gymnosporangium juniperi-virginianae*

Sequential number	Apple cultivar
1	McIntosh
2	Delicious
3	Arkansas Black
4	Empire
5	Cortland
6	Golden Delicious
7	Turley
8	Tolman Sweet
9	York Imperial
10	Yellow Newtown
11	Ben Davis
12	Jonathan
13	Prima
14	Rome Beauty

TABLE 2. Formula codes for virulence formulae of populations of *Gymnosporangium juniperi-virginianae*

Formula number	Virulence formula[a]
1	1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11/12, 13, 14
2	1, 2, 3, 4, 5, 6, 7, 8, 9, 10/11, 12, 13, 14
3	1, 2, 3, 4, 5, 6, 7, 8/9, 10, 11, 12, 13, 14
4	1, 2, 3, 4, 5, 6, 7/8, 9, 10, 11, 12, 13, 14
5	1, 2, 3, 4, 5, 6/7, 8, 9, 10, 11, 12, 13, 14
6	1, 2, 3, 4, 5, 7/6, 8, 9, 10, 11, 12, 13, 14

[a]Sequential numbers of apple cultivars effective/ineffective in conditioning an incompatible host-parasite interaction (absence of aecial) with a given population of *G. juniperi-virginianae*.

TABLE 3. Reaction of apple cultivars and numbered selections to artificial inoculation with *Gymnosporangium juniperi-virginianae*[a]

Cultivars	No. of tests[b]	Most advanced reaction[c]	Infection rating[d]	
			max.[e]	mean[f]
Arkansas Black	3	P	3.8	2.9
Baldwin	3	N	3.6	1.2
Ben Davis	6	A	4.1	3.4
Britemac	2	P	3.3	2.2
Burgundy	2	A	3.9	3.8
Carroll	2	Mc	-[g]	-[g]
Cortland	5	P	4.1	2.8
Delicious	6	P	3.2	1.2
Duchess of Oldenburg	2	A	2.0	1.7
Empire	6	P	4.0	2.3
Golden Delicious	5	A	4.1	3.7
Holly	2	Mc	-	-
Idared	3	A	2.8	2.5
Jerseymac	2	O		
Jonagold	2	A	3.2	3.0
Jonamac	2	Mc	-	-
Jonathan	7	A	4.4	3.7
Julyred	2	A	3.3	3.1
Kola	1	A	3.8	-
Lodi	2	A	4.2	4.0
Magnolia Gold	2	A	3.9	3.7
McIntosh	6	Mc	-	-
Milton	2	Mc	-	-
Mollie's Delicious	2	P	2.4	1.2
Monroe	4	A	3.9	3.1
Mutsu	3	A	3.4	3.3
NY 151	3	A	3.7	3.3
NY 53708-23	3	Mc	-	-
NY 53710-95	2	A	3.6	3.1
NY 55140-19	4	Mc	-	-
NY 56601-3	3	Mc	-	-
NY 61345-2	4	P	3.2	1.6
NY 61356-22	3	A	3.6	2.9
NY 62319-22	3	A	4.0	3.1
Northern Spy	2	A	3.9	3.5
Northwestern Greening	2	P	2.0	1.7
Ottawa 523	3	A	4.2	2.9
Ozark Gold	2	A	3.3	3.0
Prima	6	A	4.6	4.0
Priscilla	3	P	2.2	1.2
Puritan	2	Mc	-	-
Quinte	3	A	3.2	3.0
Rome Beauty	5	A	4.4	4.0
Scotia	2	P	2.0	1.0
Spartan	3	P	1.4	0.7
Spigold	4	A	4.0	3.2
Spijon	2	A	3.2	3.0
Stayman	2	P	3.2	3.2
Summerred	2	A	3.9	3.8
Tolman Sweet	4	A	4.1	4.0
Turley	4	P	3.8	3.3
Twenty Ounce	2	A	4.3	3.8
Tydeman's Early	3	Mc	-	-
Wayne	3	A	4.0	3.5
Wealthy	3	A	4.2	3.7
Winesap	4	P	3.2	1.9
Yellow Newtown	6	A	4.3	3.7
York Imperial	5	A	4.4	4.0

[a]Cultivars grown as single shoot trees on seedling rootstocks in pots in greenhouse. The six youngest leaves were sprayed with a suspension of 267,000 basidiospores/ml from *G. juniperi-virginianae* galls collected in western New York State (population with virulence formula no. 6).

[b]Three-to-five plants inoculated in each test.

[c]In order of increasing compatibility of host-parasite interactions: O = no macroscopic symptom; N = small, discrete, nonsporulating lesion; Mc = indiscrete, chlorotic mottle, no sporulation; P = pycnia; A = aecia.

[d]Infection rating = $\log_{10}(10nd^2)$, where n is highest number of lesions on any leaf, d is largest mean diameter of lesions on any leaf.

[e]Highest infection rating on any plant in any test.

[f]Mean of mean infection ratings for all tests.

[g]Reaction too diffuse to quantify as an infection rating.

RESULTS

Cultivar reactions.—The most advanced reaction and the maximum and mean infection ratings for every cultivar tested are shown in Table 3. Also shown, are the same determinations for numbered selections from Geneva, New York (NY) and Ottawa, Canada, that were involved in the inheritance studies.

Since aecia were produced on Rome Beauty, Prima, Jonathan, Ben Davis, Yellow Newtown, York Imperial, Tolman Sweet, and Golden Delicious, the collection of rust galls was assigned virulence formula no. 6 (Table 2) (3).

In cultivars with the Mc reaction, symptoms were too diffuse to quantify as an infection rating. This reaction appeared less injurious than the N reaction.

Seedling reactions.—The frequency of seedlings with pycnia in progenies from the 1972 crosses is shown in Table 4. Genotypes were assigned to the parents on the basis of their own reaction and the segregation for pycnia

TABLE 4. Reaction of apple seedlings from 1972 crosses to artificial inoculation with *Gymnosporangium juniperi-virginianae*[v]

Parents and genotypes[w]			Seedlings −Pycnia (no.)	Seedlings +pycnia (no.)	Expected ratio	Goodness of fit (P)
NY 53710-95[x] (*gy-a/gy-a, Gy-b/gy-b*)	×	NY 53708-23 (*Gy-a/gy-a, Gy-b/gy-b*)	11	15	3:5	0.85
NY 53710-95 (*gy-a/gy-a, Gy-b/gy-b*)	×	NY 55140-19 (*Gy-a/gy-a, Gy-b/gy-b*)	21	40	3:5	0.69
NY 53710-95 (*gy-a/gy-a, Gy-b/gy-b*)	×	NY 61345-2 (*Gy-a/gy-a, Gy-b/ Gy-b*)	37	52	1:1	0.15
NY 53710-95 (*gy-a/gy-a, Gy-b/gy-b*)	×	NY 61356-22 (*Gy-a/gy-a, gy-b/gy-b*)[w]	11	48	1:3	0.30
NY 53710-95 (*gy-a/gy-a, Gy-b/gy-b*)	×	Ottawa 523[y] (*gy-a/gy-a, Gy-b/gy-b*)[w]	8	90	0:1	
NY 53710-95 (*gy-a/gy-a, Gy-b/gy-b*)	×	NY 56601-3 (*Gy-a/gy-a, Gy-b/gy-b*)	34	36	3:5	0.03
Ottawa 523 (*gy-a/gy-a, Gy-b/gy-b*	×	NY 55140-19 (*Gy-a/gy-a, Gy-b/gy-b*)	64	92	3:5	0.43
Ottawa 523 (*gy-a/gy-a, Gy-b/gy-b*)	×	NY 53708-23 (*Gy-a/ gy-a, Gy-b/gy-b*)	18	59	3:5	0.014
Ottawa 523 (*gy-a/gy-a, Gy-b/gy-b*)	×	NY 56601-3 (*Gy-a/gy-a, Gy-b/gy-b*)	55	100	3:5	0.68
Spartan (*Gy-a/ Gy-a, Gy-b/ Gy-b*)	×	NY 55140-19 (*Gy-a/gy-a, Gy-b/gy-b*)[w]	18	0	1:0	
Spartan (*Gy-a/ Gy-a, Gy-b/ Gy-b*)	×	NY 61345-2 (*Gy-a/gy-a, Gy-b/ Gy-b*)	35	2[z]	1:0	
Spartan (*Gy-a/ Gy-a, Gy-b/ Gy-b*)	×	NY 62319-22 (*gy-a/gy-a, Gy-b/ Gy-b*)	52	10[z]	1:0	
NY 151 (*gy-a/gy-a, Gy-b/gy-b*)	×	NY 61356-22 (*Gy-a/gy-a, gy-b/gy-b*)[w]	4	13	1:3	1.00
NY 151 (*gy-a/gy-a, Gy-b/gy-b*)	×	NY 62319-22 (*gy-a/gy-a, Gy-b/ Gy-b*)	0	22	0:1	
NY 61356-22 (*Gy-a/gy-a, gy-b/gy-b*)	×	NY 53708-23 (*Gy-a/gy-a, Gy-b/gy-b*)	18	24	3:5	0.65

[v]Seedlings 8-10 wk old were sprayed with a suspension of 267,000 basidiospores/ml from *G. juniperi-virginianae* galls from population with virulence formula no. 6.

[w]Seed parent is shown first except for crosses marked.

[x]Apple selections from New York State Agricultural Experiment Station, Geneva, are designated NY.

[y]Selection from Canada Department of Agriculture, Ottawa.

[z]Very small pycnial lesions; no aecia observed.

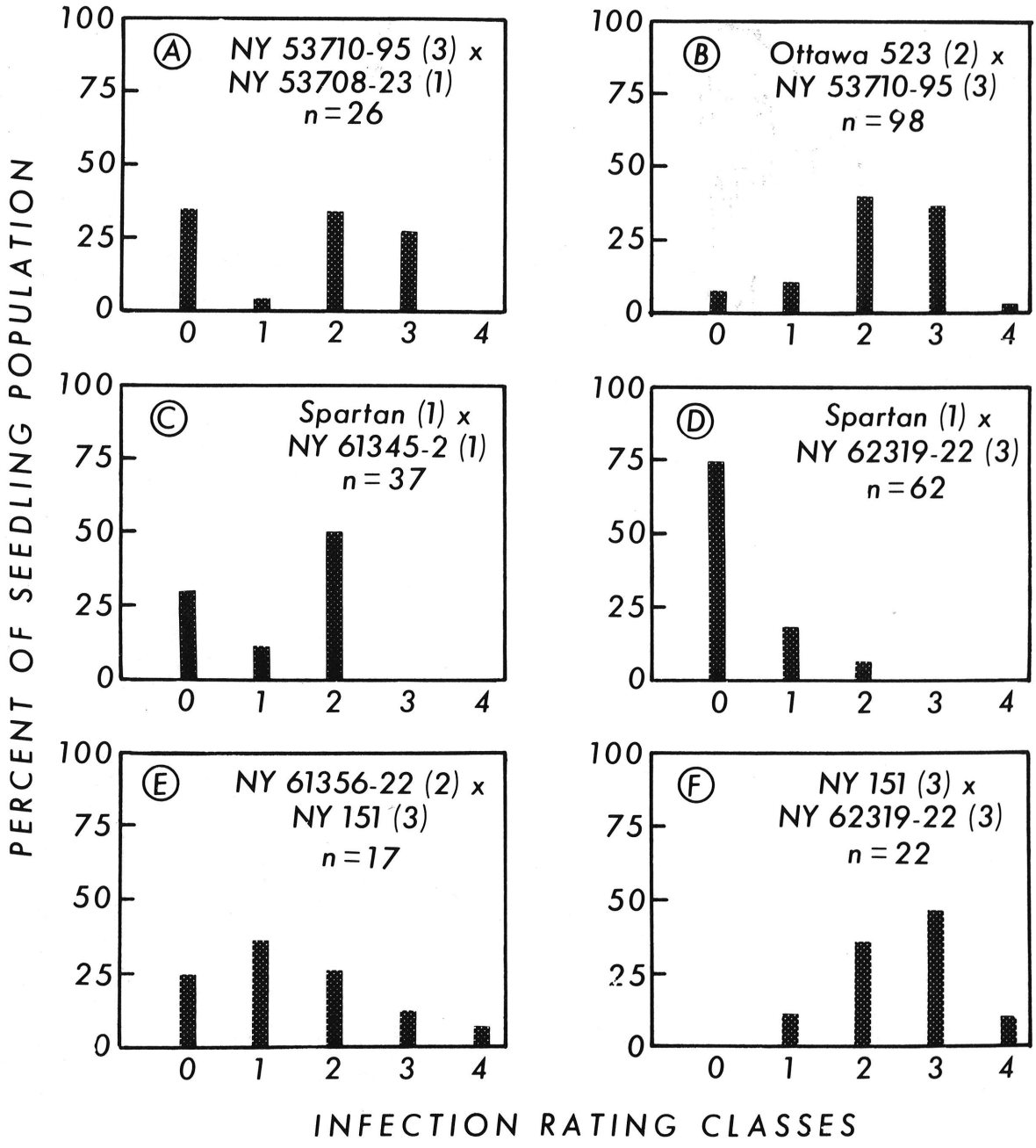

Fig. 1. Frequency distribution of cedar apple rust infection rating in 1972 progenies inoculated as 12-week-old seedlings in 1973 with a population of *Gymnosporangium juniperi-virginianae* with virulence formula no. 6. Infection rating class 0 was $\log_{10}(10nd^2) = 0$; class 1, $\log_{10}(10nd^2) = 0.1$ to 1.9; class 2, $\log_{10}(10nd^2) = 2.0$ to 2.9; class 3, $\log_{10}(10nd^2) = 3.0$ to 3.9; class 4, $\log_{10}(10nd^2) = \geq 4.0$. Infection rating classes of parents are shown in parentheses.

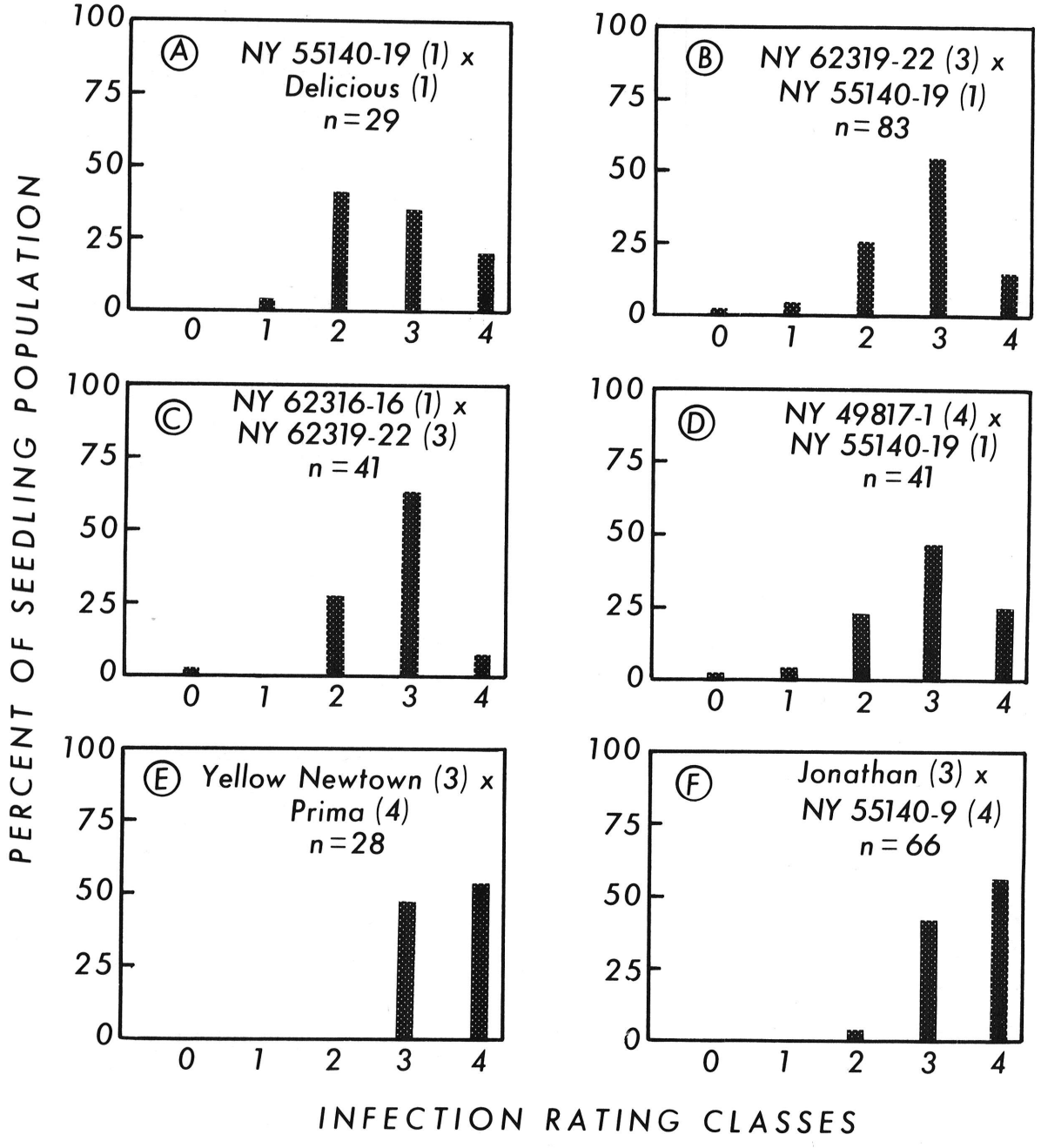

Fig. 2. Frequency distribution of cedar apple rust infection rating in 1973 progenies inoculated as 8-to 10-wk-old seedlings in 1974 with a population of *Gymnosporangium juniperi-virginianae* with virulence formula no. 6. Infection rating class 0 was $\log_{10}(10nd^2) = 0$; class 1, $\log_{10}(10nd^2) = 0.1$ to 1.9; class 2, $\log_{10}(10nd^2) = 2.0$ to 2.9; class 3, $\log_{10}(10nd^2) = 3.0$ to 3.9; class 4, $\log_{10}(10nd^2) = 4.0$. Infection rating classes of parents are shown in parentheses.

production in their progenies according to the hypothesis of Mowry (20). The two genes, *Gy-a* and *Gy-b*, must both be in dominant form to confer resistance to pycnia production. Only two progenies out of fifteen had a goodness of fit to the predicted ratio with $P < 0.05$ and none with $P < 0.01$. One cross, NY 53710-95 × Ottawa 523, was expected to produce all susceptible seedlings, but it produced eight that bore no pycnia. These symptomless seedlings were interpreted as escaping infection. Two crosses with Spartan, × NY 61345-2 and × NY 62319-22, had progenies with 5% and 16%, respectively, seedlings with pycnia where none was predicted. The pycnia were all ≤1mm in diameter and no aecium was observed subsequently.

The infection ratings of selected progenies from the 1972 crosses are summarized in Fig. 1. Comparison with Table 1 illustrates that segregation for pycnia production and frequency distribution of infection ratings are loosely correlated; e. g., Fig. 1 (a) and (b). Several progenies, however, contained a larger proportion of seedlings with high infection ratings than would be predicted by the dual gene hypothesis for pycnia production; e. g., Fig. 1 (c). A few progenies; e. g., Fig. 1 (e), had a smaller proportion of seedlings with high infection ratings than predicted.

In some progenies the frequency distribution of infection ratings was related more to the parents' infection ratings than to their postulated genotypes; e. g., Fig. 1 (f).

The seedlings from 1973 crosses were inoculated when 8-10 wk old, at which age resistance to pycnia formation appeared poorly developed. Nearly all seedlings bore pycnia, and genotypes were not postulated. However, infection ratings were determined and selected frequency distributions are presented in Fig. 2. The ratings were higher overall than for the older 1972 seedlings. Nevertheless, the distributions were related to the parents' infection ratings, cf. Fig. 2 (a) with Fig. 2 (f).

DISCUSSION

Most of the NY numbered selections were resistant to apple scab [*Venturia inaequalis* (Cke.) Wint.] with resistance derived from *M. pumila* cultivar 'Antonovka' (NY 53708-23), *M. floribunda* 821 (13) (NY 55140-19, NY 61345-2, NY 61356-22, NY 62319-22), *M. prunifolia* 19651 (13) (NY 53710-95) and *M. pumila* R12740-7A (25) (NY 56601-3). There was no evidence for linkage between genes for scab resistance and rust resistance (Aldwinckle and Lamb, *unpublished*). The scab-resistant selections showed a broad range of reaction to *G. juniperi-virginianae*.

Although virulence of the pathogen may have changed, the reactions of many of the older apple cultivars to artificial inoculation with the population of *G. juniperi-virginianae* with virulence formula no. 6 agree with their reactions reported in numerous published lists. In the field those cultivars with a mean infection rating<2.5 will usually suffer insignificant injury from cedar apple rust although visible symptoms may be seen, whereas infection ratings >3.0 indicate the possibility of serious damage in the field.

Among cultivars that have only recently achieved recognition in North America, Britemac, Carroll, Empire, Jerseymac, Mollie's Delicious, Scotia, and Spartan had low ratings. Holly, Jonamac, Puritan, and Tydeman's Early developed diffuse, mildly chlorotic lesions (Mc). Burgundy, Jonagold, Julyred, Magnolia Gold, Monroe, Mutsu, Ozark Gold, Spigold, Summerred, and Wayne had high infection ratings.

Our data (Table 3) are generally compatible with the genotypes proposed by Mowry (20) for resistance based on absence of aecia. However, Arkansas Black and Cortland, which produced no aecia and were given resistant genotypes by Mowry (20), nevertheless had infection ratings of 2.9 and 2.8, respectively. Delicious was given a susceptible genotype by Mowry (20) although we know of no reports of aecia on Delicious leaves and no aecia were produced after artificial inoculation with any race of *G. juniperi-virginianae* (3). Delicious has, however, been reported as very susceptible to quince rust (1, 9). Starking, a sport of Delicious, was given a resistant genotype by Mowry (20). Kola, given a resistant genotype by Mowry (20), produced abundant aecia in our tests (Table 3). Rome Beauty, in agreement with data here, was given a susceptible genotype, but one of its sports, Gallia, received a resistant genotype, although aecia have been observed following artificial inoculation (M. Szkolnik, *personal communication*). It is unlikely that these differences were due to differences in virulence of the pathogen (3). Correction of the parental genotypes would strengthen Mowry's (20) data since Delicious, Kola, and Gallia were parents in five of the eleven crosses that produced significant chi-square values under his hypothesis.

The bad fits may be due to modifying genes, or to gene dosage. Apples are secondary polyploids (28) that behave as diploids in some genetic characters but not in others; e. g., fruit color (29). It also is possible that variability in the pathogen population could cause bad fits.

The good fit of this hypothesis to most of our data (Table 2) on pycnia production in crosses between parents with diverse origins supports its utility. Because of the priority of Knight's (14) suggestion that one of the genes be designated *Gy* and the fact that the genes are phenotypically indistinguishable, they are assigned the symbols *Gy-a* and *Gy-b* here.

The reaction of apple seedlings to artificial inoculation appears to change during the first few weeks after germination, independently of leaf age. Seedlings 3 to 6 wk old appeared uniformly susceptible and all produced pycnia even from crosses involving different resistant parents (Aldwinckle, *unpublished*). Similar seedlings inoculated when older segregated for pycnia production. Hamilton and Lamb (*unpublished*) found the same situation in crosses where no susceptible (with pycnia) seedlings were expected. Several susceptible seedlings did occur, but too few to fit any possible duplicate-gene ratio.

The distribution of infection ratings could not be predicted by the duplicate-gene hypothesis alone. The parents' infection ratings were also influential and must be taken into account in breeding for resistance. The infection rating is probably controlled by the two major genes plus several modifying genes plus the genetic capacity of the pathogen, interacting together and with the environment.

Information on the resistance of apple cultivars to *G. juniperi-virginianae* will facilitate more economical

disease control. Where apples are growing near red cedars, fungicides are the only practical method of controlling rust. The only fungicides presently registered for this use in the United States are carbamates. These chemicals often are not preferred for control of apple scab and do not control powdery mildew. Rust-resistant cultivars can be sprayed with chemicals that are more effective against scab and powdery mildew. Where rust is a severe annual problem, growers should avoid, if possible, planting very susceptible cultivars. Knowledge of sources and inheritance of resistance, and of fungal pathogenicity will expedite the production of rust-resistant cultivars in the future.

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