Euphytica 17 (1968): 417–438

INHERITANCE OF RESISTANCE TO POWDERY MILDEW, *SPHAEROTHECA MACULARIS* (FR.) JACZEWSKI IN THE RED RASPBERRY, *RUBUS IDAEUS* L.

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Received 21 November, 1967

SUMMARY

The varieties Lloyd George and Burnetholm (and probably Malling Promise) are heterozygous for three genes governing resistance to mildew (*S. macularis* (FR.) JACZEWSKI, syn. *S. humuli* (DC.) BURR.), Sp_1 and Sp_2 being dominant complementaries and sp_3 recessive. Resistance whether of Sp_1, Sp_2 or sp_3 sp_3 origin is epistatic to susceptibility. Sp_3 is linked with the fruit colour gene *T* with a crossover value of ca. 25%, the gene order in this linkage group being B-Sx_3-T-Sp_3. Evidence suggesting linkage between *H* (hairy canes) and resistance is presented.

INTRODUCTION

Serious attacks of powdery mildew (*Sphaerotheca macularis* (FR.) JACZEWSKI, syn. *S. humuli* (DC.) BURR.) are almost unknown on established raspberry plantations in Great Britain. In bad years, the most susceptible British commercial varieties, Norfolk Giant and Malling Jewel, may show occasional diseased cane tips and young leaves, and diseased fruits, but control sprays are rarely necessary.

In contrast, crippling attacks of powdery mildew have occurred at East Malling on young seedlings derived from a wide range of material. This suggests that present day varieties have consciously or unconsciously been selected for mildew resistance, and accordingly a study of the inheritance of resistance was undertaken. This was based mainly on progenies derived from Lloyd George and Burnetholm, raised initially for other purposes.

REVIEW OF THE LITERATURE

S. macularis occurs throughout Europe into Russia, and in Africa, Asia and North America on a wide range of host plants (SALMON, 1900). References to the relative susceptibility to mildew of raspberry varieties and other *Rubus* species and hybrids are summarized in Table 1.

From these data, it is clear that powdery mildew occurs widely in North America and Europe, affecting red, black and purple raspberries, dewberries, blackberries and other *Rubus* species. In America, Latham red raspberry (PETERSON and JOHNSON, 1928), Lucretia dewberry (YOUNG and FULTON, 1951), and Black Hawk black raspberry (FULTON, 1960) are sometimes severely attacked.

Table 1. Response of raspberry varieties and other *Rubus* cultivars and species to powdery mildew

Immune			
Loganberry	WILHELM et al. (1951)		
Resistant			
Madawaska	HUNTER and WHITE (1950)	Chief	ALDERMAN et al. (1957)
Marcy	HUNTER and WHITE (1950)	Blackberry	FULTON (1960)
Rideau	HUNTER and WHITE (1950)		
Slightly susceptible			
Baumforth B	NATTRASS (1927)	Maclaren's Prolific	NATTRASS (1927)
Lloyd George	NATTRASS (1927)	Puyallup	SCHWARTZE and MYHRE (1953)
Red Cross	NATTRASS (1927)	Cumberland	
Goliath	NATTRASS (1927)	*(R. occidentalis)*	FULTON (1960)
Hornet D	NATTRASS (1927)	Logan *(R. occidentalis)*	FULTON (1960)
Moderately susceptible			
Muskoka	SPANGELO (1955)	*R. strigosus**	ELLIS and EVERHART (1892); SALMON (1900)
Trent	SPANGELO (1955)		
Reader's Perfection	NATTRASS (1927)	*R. hispidus**	ELLIS and EVERHART (1892); SALMON (1900)
Semper Fidelis B	NATTRASS (1927)		
Blackberry	PETERSON and JOHNSON (1928)	*R. canadensis**	SALMON (1900)
*R. odoratus**	ELLIS and EVERHART (1892); SALMON (1900)	*R. spectabilis**	SALMON (1900)
		R. geoides	HASKELL and PATERSON (1966)
*R. triflorus**	ELLIS and EVERHART (1892) SALMON (1900)		
Very susceptible			
Viking	HUNTER and WHITE (1950)	Northumberland Thornless Fillbasket	NATTRASS (1927); SWARBRICK (1930)
Ottawa	HUNTER and WHITE (1950); SPANGELO (1955)	Baumforth E	NATTRASS (1927); SWARBRICK (1930)
Latham	BENNETT (1928); FULTON (1951); GIDDINGS and WOOD (1925); HUNTER and WHITE (1950); KELLY (1960); PETERSON and JOHNSON (1928); SLATE and SUIT (1944); SLATE et al. (1953)	Owasco	GIDDINGS and WOOD (1925)
		Black Hawk *(R. occidentalis)*	FULTON (1960)
		Dundee *(R. occ.)*	SLATE et al. (1953)
		Munger *(R. occ.)*	BENNETT (1928)
		Cardinal (*R. occ.* × *R. idaeus)*	BENNETT (1928)
		Lucretia dewberry	YOUNG and FULTON (1951)
		R. henryi	EPSTEIN (1965)

* Degree of susceptibility not specified

Few data are available on the inheritance of resistance to this disease. Viking transmits its susceptibility to many of its seedlings, including Ottawa (HUNTER and WHITE, 1950) which in turn gives a high proportion of susceptible seedlings in self-bred and outcrossed progenies (SPANGELO, 1955, 1961). In New York State, in a number of crosses involving Indian Summer derivatives as one or both parents, percentages of naturally infected seedlings ranged from 0.0–92.6, the highest percentages occurring in crosses with the unrelated variety Ranere, and the lowest in sib crosses of Indian Summer derivatives. Indian Summer sibs in which Milton or Taylor was the other original parent showed a particularly low proportion of susceptibles (SLATE and SUIT, 1944). SPANGELO (1955) confirmed the value of Milton in transmitting mildew resistance to its progeny.

Lloyd George, classed as slightly susceptible by NATTRASS (1927) (Table 1) was one parent of the resistant varieties Madawaska, Marcy and Rideau, of the moderately susceptible Trent, and of Indian Summer, Milton and Taylor, whose breeding behaviour has been discussed above. The other parent of Madawaska, Marcy, Rideau and Taylor was Newman, while Newburgh, the male parent of Milton, was also a Newman seedling. This suggests that both Lloyd George and Newman contribute considerable mildew resistance to their progeny.

The data of SLATE and SUIT (1944) for Indian Summer derivatives grown in Chautauqua County suggest that seedlings are relatively more susceptible to mildew than are established plantations. Severe attacks of mildew affecting up to 92.6% of the plants occurred on seedling progenies in 1943, while the parent varieties Indian Summer, Marcy, Taylor, Milton and Newburgh, growing nearby were totally unaffected.

In the published literature (as in the work reported here) there is no evidence of strain differentiation of the pathogen on *Rubus*.

ASSESSMENT OF RESISTANCE IN SEEDLING PROGENIES

Records of artificial and natural infections on first year canes of certain progenies have been kept for a number of years, grading into 5 classes. Grade 0 plants were apparently entirely free from the disease, grade 1 plants showed a very few, small, isolated mildew lesions, usually on the undersurface of the leaves, those of grade 2 showed a number of larger leaf infections, sometimes accompanied by a slight attack on one or two cane tips, those of grade 3 showed at least one severely attacked cane tip, while grade 4 plants showed several severely attacked cane tips. Plants of grade 0–1 formed an obvious and, in general, clearly defined resistant group, while those of grades 2–4 were, except for occasional grade 2 plants, equally clearly classifiable as susceptible.

The incidence of mildew varies considerably from year to year according to climatic conditions, and symptom expression also depends on the host genotype. Thus, some progenies tend to show severe infection of the cane tips and very young leaves only, while in others the mildew affects rather older leaves as well. Both upper and lower leaf surfaces may be attacked or the mildew may be more or less confined to the lower surface. Late developing 'soft' shoots are sometimes severely affected while the rest of the plant remains healthy. All these factors make grading and classification in the susceptible classes difficult. However, even in the absence of artificial inoculation, by recording over two or more years, considering only the severest grade over this period, and classifying plants with one or more grade '4', '3' or '2' attacks as susceptible and those whose grade never exceeded 'l' as resistant, progeny behaviour can be reliably assessed. Nevertheless genetic interpretation of data obtained from field records of this nature must of necessity be somewhat tentative.

The fruits of some seedlings suffer mildew attacks, but the relative susceptibility of fruits and of vegetative organs does not always appear to be correlated, and fruit susceptibility is not considered in this work.

Table 2. Segregations for mildew resistance in Lloyd George S_1 and S_2 progenies

No. of progenies	Parentage	Parental genotypes	Observed		Expected		χ^2	P
			Res.	Sus.	Res.	Sus.		approx.
1 (841)*	Lloyd George S_1	$Sp_1sp_1Sp_2sp_2Sp_3sp_3$	31	15	(43:21) 30.91	15.09	0.00	
2 (844,852)	Lloyd George S_2	$Sp_1Sp_1Sp_2sp_2Sp_3sp_3$ or $Sp_1sp_1Sp_2Sp_2Sp_3sp_3$	69	16	(13:3) 69.06	15.94	0.00	
					Heterogeneity		0.64	0.5
2 (850,853)	Lloyd George S_2	$Sp_1sp_1Sp_2Sp_2Sp_3Sp_3$ or $Sp_1Sp_1Sp_2sp_2Sp_3Sp_3$	56	19	(3:1) 56.25	18.75	0.00	
					Heterogeneity		0.14	0.7
3 (843,846,848)	Lloyd George S_2	$Sp_1sp_1Sp_2sp_2Sp_3sp_3$	86	48	(43:21) 90.03	43.97	0.55	0.9
					Heterogeneity		0.39	0.8
3 (851,855,847)	Lloyd George S_2	Sp_3sp_3	31	93	(1:3) 31.00	93.00	0.00	
					Heterogeneity		0.91	0.6
1 (849)	Lloyd George S_2	$Sp_1Sp_1Sp_2Sp_2$ or sp_3sp_3 etc.	47	2				
1 (854)	Lloyd George S_2	$Sp_1Sp_1Sp_2Sp_2$ or sp_3sp_3 etc.	41	3				

* (841) etc. = family numbers

INHERITANCE OF MILDEW RESISTANCE

Lloyd George derivatives

S_1 and S_2 progenies. Selfed progenies of Lloyd George were graded in 1961, following natural infection, and again in September 1962, one month after artificial inoculation (Table 2). Control clones of Lloyd George growing nearby were similarly inoculated and occasional canes showed 'slight' (grade 1) infections after a month.

Mildew was widespread in 1962, providing ideal conditions for demonstrating the full extent of susceptibility in these progenies.

The observed results in S_1 and S_2 progenies can be explained on the basis that Lloyd George is heterozygous for three resistance genes, Sp_1 and Sp_2 being dominant complementaries and sp_3 monogenic recessive; resistance of either origin is assumed to be epistatic to susceptibility. Parental genotypes and expected ratios in a random sample of segregating S_2 progenies would be:

4 $Sp_1sp_1Sp_2sp_2Sp_3sp_3$	43:21	1 $Sp_1Sp_1sp_2sp_2Sp_3sp_3$		
2 $Sp_1Sp_1Sp_2sp_2Sp_3sp_3$	13:3	1 $sp_1sp_1Sp_2Sp_2Sp_3sp_3$		
2 $Sp_1sp_1Sp_2Sp_2Sp_3sp_3$		2 $Sp_1sp_1sp_2sp_2Sp_3sp_3$	1:3	
1 $Sp_1Sp_1Sp_2sp_2Sp_3Sp_3$	3:1	2 $sp_1Sp_2Sp_2sp_2Sp_3sp_3$		
1 $Sp_1sp_1Sp_2Sp_2Sp_3Sp_3$		1 $sp_1sp_1sp_2sp_2Sp_3sp_3$		
2 $Sp_1sp_1Sp_2sp_2Sp_3Sp_3$	9:7			

All these ratios except the 9:7 appeared in the S_2 progenies, and all agreed well with expectation. However, genotypes assigned to parents of all families except those segregating in 1:3 ratios were necessarily tentative, as the average progeny size was too small to differentiate with certainty between 13:3, 3:1, and 43:21 ratios. The parents of Families 849 and 854 were presumably homozygous for resistance (i.e. $Sp_1Sp_1Sp_2Sp_2$ and/or sp_3sp_3), the few 'susceptible' plants probably being due to accidental recording of 'spawn' from neighbouring plants.

Lloyd George F_1 progenies. In 1966, a number of progenies derived from crossing Lloyd George with resistant (115/74, 115/117, 841/23) and susceptible (841/35) Lloyd George S_1 seedlings, with the resistant variety Malling Promise, and with two resistant self-bred selections from seedling 91/187 (341/3, 341/16), were inoculated in the field with mildew and graded as before (Table 3).

In all except two of these families, agreement with expectation was good, assuming Lloyd George to be heterozygous for all three genes. The reciprocal of one of the exceptional families (1185) also agreed well with expectation.

Segregations in Family 1477, Lloyd George × Malling Promise, agreed well with 43:21, suggesting that Malling Promise, also, is heterozygous for Sp_1, Sp_2, and Sp_3. Seedlings 341/3 and 341/16 (55/6 × 72/59 (S_1 Malling Promise) F_2) were assigned the genotype $Sp_1sp_1Sp_2sp_2sp_3sp_3$ on the basis of the excellent agreement with a 25:7 expectation in three out of their four F_1 progenies with Lloyd George (the exception being Family 1185); these genotypes were confirmed by segregations in further crosses with Lloyd George derivatives (see Table 5).

Table 3. Segregations for mildew resistance in F_1's of Lloyd George × S_1 Lloyd George, × 341/3, × 341/16 and × Malling Promise

Family	Parentage	Parental genotypes	Observed		Expected		χ^2	P approx.
			Res.	Sus.	Res.	Sus.		
					(5:3)			
1194	Lloyd George × 841/35	$Sp_1sp_1Sp_2sp_2Sp_3sp_3$ × $Sp_1Sp_1sp_2sp_2Sp_3sp_3$**	36	5	25.63	15.37	11.19	<0.001
					(13:3)			
577*	Lloyd George × 115/74	$Sp_1sp_1Sp_2sp_2Sp_3sp_3$ × $Sp_1Sp_1Sp_2sp_2Sp_3sp_3$**	77	11	71.50	16.50	2.26	0.15
1192	Lloyd George × 841/23		53	14	54.44	12.56	0.20	0.7
1193	Reciprocal		52	13	52.81	12.19	0.07	0.8
	Totals		182	38	178.75	41.25	0.32	0.6
					(9:7)			
576*	Lloyd George × 115/117	$Sp_1sp_1Sp_2sp_2Sp_3sp_3$ × $Sp_1sp_1Sp_2sp_2Sp_3Sp_3$	37	25	34.87	27.13	0.30	0.6
					(25:7)			
1182	Lloyd George × 341/3	$Sp_1sp_1Sp_2sp_2Sp_3sp_3$ × $Sp_1sp_1Sp_2sp_2sp_3sp_3$	32	10	32.81	9.19	0.09	0.8
1183	Reciprocal		39	9	37.50	10.50	0.27	0.6
1184	Lloyd George × 341/16		38	9	36.72	10.28	0.20	0.7
1185	Reciprocal		45	3	37.50	10.50	6.86	<0.01
	Totals		154	31	144.53	40.47	7.42	
					Deviation		2.84	0.09
					Heterogeneity		4.58	0.20
					(43:21)			
1477	Lloyd George × Malling Promise	$Sp_1sp_1Sp_2sp_2Sp_3sp_3$ × $Sp_1sp_1Sp_2sp_2Sp_3sp_3$	45	24	46.36	22.64	0.12	0.7

* Families 576 and 577 were not artificially inoculated in 1966; ratios are based on 4 years' records of natural infection

** An alternative genotype, with Sp_1 replacing Sp_2 and vice versa, would give the same expected ratio

First backcrosses of S_1 Lloyd George to Lloyd George, F_2's of S_1 Lloyd George × Lloyd George, and sib crosses of S_1 Lloyd George × Lloyd George. Backcrosses, F_2's, and sib crosses of resistant Lloyd George derivatives were graded after artificial inoculation in the field in 1966 (Table 4).

Agreement with expectation was good except for a deficiency of susceptibles in Families 1191 and 1221. The genotype of 577/90, the parent of Family 1191, is based on the segregation in the reciprocal (1190). In Families 1191 and 1221, a number of plants (14 and 15, respectively) died before mildew inoculations were made, and this may have affected segregation ratios.

F_1's of (Lloyd George × S_1 Lloyd George) × 341/3 and × 341/16. Nine F_1 progenies of Family 576 and 577 selections (Lloyd George × S_1 Lloyd George) crossed with 341/3 or 341/16 were inoculated in the field in 1966 and graded as before (Table 5).

All the Family 576 and 577 selections except 577/15, the parent of Family 1203, were resistant: all the Family 577 selections were bound from their origin (Table 3), to carry Sp_1 or Sp_2. Observed segregations agreed well with expectation in all progenies, confirming the genotypes $Sp_1Sp_1Sp_2sp_2Sp_3sp_3$ for 577/35 (see Table 4) and $Sp_1sp_1Sp_2sp_2sp_3sp_3$ for 341/3 and 341/16. Several alternative genotypes were possible for 577/11, the parent of the non-segregating families 1201 and 1202; two have been given in Table 5.

Burnetholm derivatives

S_1, S_2, and S_3 progenies. Segregations following natural infection in Burnetholm S_1 and S_3 progenies suggested trigenic control of resistance as in Lloyd George, Burnetholm being heterozygous for all three genes. Additional evidence supporting this hypothesis was obtained from S_2 progenies inoculated in the field in 1966 (Table 6).

The S_1 progeny of 135 plants agreed well with a 43:21 expectation, and all 18 of the small S_2 and S_3 families segregated for resistance in ratios in accord with the three-gene hypothesis. The parental genotypes in Table 6 were based on the closeness of agreement with ratios expected in S_1, S_2, and S_3, but progeny sizes in S_2 and S_3 were too small to discriminate with certainty between ratios such as 13:3 and 3:1; hence these genotypes are tentative except where confirmed in further crosses (Table 7).

The response to mildew of the parents of the S_3 progenies was not known, but that of the parents (Family 765 selections) of the S_2 progenies had been determined on natural infection in the field. All appeared to be resistant except 765/151 and 765/122, the parents of Families 1435 and 1454. Mildew attacks on 765/151 were graded as '2', and since Family 1435 segregated in a ratio of 12:3, 765/151 was presumably genotypically resistant. Seedling 765/37, the parent of Family 1444, which segregated in a ratio of 5:15, was presumably genotypically susceptible (Sp_3sp_3) and must have escaped natural infection.

Hybrid progenies. Segregations in F_1 and F_2 derivatives of Burnetholm, graded in the field following natural, or artificial (Families 1407, 1412, 1413, 1478), inoculation were consistent with the three-gene hypothesis (Table 7). The mildew responses of parents in Families 129, 239, and 332 are not known; all the remaining parents were resistant.

Table 4. Segregations for mildew resistance in first backcrosses of S_1 Lloyd George to Lloyd George, in F_2's of S_1 Lloyd George × Lloyd George and in sib crosses

Family	Parentage	Parental genotypes	Observed		Expected		χ^2	P approx.
			Res.	Sus.	Res.	Sus.		
					(7:1)			
1186	Lloyd George × 577/12	$Sp_1sp_1Sp_2sp_2Sp_3sp_3$ × $Sp_1Sp_1Sp_2sp_2sp_3sp_3$*	32	4	31.50	4.50	0.06	0.8
1187	Reciprocal	$Sp_1sp_1Sp_2sp_2Sp_2sp_3$ × $Sp_1Sp_1Sp_2sp_2sp_3sp_3$*	14	3	14.88	2.12	0.42	0.5
1190	Lloyd George × 577/90	$Sp_1sp_1Sp_2sp_2Sp_3sp_3$ × $Sp_1Sp_1Sp_2sp_2sp_3sp_3$*	47	5	45.50	6.50	0.40	0.5
1191	Reciprocal	$Sp_1sp_1Sp_2sp_2Sp_3sp_3$ × $Sp_1Sp_1Sp_2sp_2sp_3sp_3$*	57	0	49.88	7.12	8.14	<0.01
	Totals		150	12	141.76	20.24	3.84	0.05
					(3:1)			
1188	Lloyd George × 576/37	$Sp_1sp_1Sp_2sp_2Sp_3sp_3$ × $Sp_1Sp_1Sp_2sp_2Sp_3Sp_3$*	46	13	44.25	14.75	0.28	0.6
1189	Reciprocal		39	13	39.00	13.00		
	Totals		85	26	83.25	27.75	0.15	0.7
					(13:3)			
1220	577/18S	$Sp_1Sp_1Sp_2sp_2Sp_3sp_3$*	19	2	17.06	3.94	1.18	0.3
1221	577/35S	$Sp_1Sp_1Sp_2sp_2Sp_3sp_3$*	60	3	51.19	11.81	8.09	<0.01
	Totals		79	5	68.25	15.75	9.03	<0.01
					(7:1)			
1212	577/1 × 577/18	$Sp_1sp_1Sp_2sp_2sp_3sp_3$ × $Sp_1Sp_1Sp_2sp_2Sp_3sp_3$*	33	2	30.62	4.38	1.48	0.2
1214	577/35 × 577/55	$Sp_1Sp_1Sp_2sp_2Sp_3sp_3$* × $Sp_1sp_1Sp_2sp_2sp_3sp_3$	40	5	39.38	5.62	0.08	0.8
	Totals		73	7	70.00	10.00	1.03	0.3

* An alternative genotype, with Sp_1 replacing Sp_2 and vice versa, would give the same expected ratio

Table 5. Segregations for mildew resistance in F_1's of Family 576 and 577 selections × 341/3 and × 341/16

No. of progenies	Parental genotypes	Observed		Expected		χ^2	P approx.
		Res.	Sus.	Res.	Sus.		
2 (Fams 1201, 1202)	$Sp_1sp_3sp_3 \times Sp_1sp_1Sp_2sp_2sp_3sp_3$ or $Sp_1Sp_1Sp_2Sp_2 \times Sp_1sp_1Sp_2sp_2sp_3sp_3$ etc.	60	0		(7:1)		
4	$Sp_1Sp_1Sp_2sp_2Sp_3sp_3$* × $Sp_1sp_1Sp_2sp_2sp_3sp_3$	158	18	154.00	22.00	0.83	0.3
				Heterogeneity		0.72	0.9
				(25:7)			
1	$Sp_1sp_1Sp_2sp_2Sp_3sp_3 \times Sp_1sp_1Sp_2sp_2sp_3sp_3$	25	11	28.12	7.88	1.58	0.2
				(9:7)			
2	$Sp_1sp_1Sp_2sp_2Sp_3Sp_3 \times Sp_1sp_1Sp_2sp_2sp_3sp_3$	55	36	51.19	39.81	0.65	0.5
				Heterogeneity		0.11	0.7
				(3:5)			
1 (Fam. 1203)	$Sp_1sp_1sp_2sp_2Sp_3Sp_3 \times Sp_1sp_1Sp_2sp_2sp_3sp_3$	22	42	24.00	40.00	0.27	0.6

* An alternative genotype, with Sp_1 replacing Sp_2 and vice versa, would give the same expected ratio

Table 6. Segregations for mildew resistance in Burnetholm S_1, S_2, and S_3 progenies

Family	Parentage	Parental genotypes	Observed		Expected		χ^2	P approx.
			Res.	Sus.	Res.	Sus.		
					(43:21)			
765	Burnetholm S_1	$Sp_1sp_1Sp_2sp_2Sp_3sp_3$	92	43	90.70	44.30	0.06	0.8
					(13:3)			
1443	Burnetholm S_2	$Sp_1sp_1Sp_1Sp_2Sp_2sp_3$*	20	5	20.31	4.69	0.03	0.9
318	Burnetholm S_3 (= 129/5S)	$Sp_1Sp_1Sp_2sp_2Sp_3sp_3$*	16	4	16.25	3.75	0.02	0.9
1445	Burnetholm S_2	$Sp_1Sp_1Sp_2sp_2Sp_3sp_3$*	19	3	17.87	4.13	0.38	0.5
1435	Burnetholm S_2	$SP_1Sp_1Sp_2sp_2Sp_2sp_3$*	12	3	12.19	2.81	0.02	0.9
	Totals		67	15	66.62	15.38	0.01	0.9
					(3:1)			
1439	Burnetholm S_2	$Sp_1Sp_1Sp_2sp_2Sp_3Sp_3$*	11	4	11.25	3.75	0.02	0.9
					(43:21)			
322	Burnetholm S_3	$Sp_1sp_1Sp_2sp_2Sp_3sp_3$	13	7	13.44	6.56	0.04	0.8
319	Burnetholm S_3	$Sp_1sp_1Sp_2sp_2Sp_3sp_3$	13	7	13.44	6.56	0.04	0.8
	Totals		26	14	26.88	13.12	0.09	0.8
7 progenies					(9:7)			
(320, 323–4, 1442, 1447–8, 1451)	Burnetholm S_2 and S_3	$Sp_1sp_1Sp_2sp_2Sp_3Sp_3$	87	70	88.33	68.67	0.05	0.8
					Heterogeneity		0.62	0.99
					(1:3)			
1444	Burnetholm S_2	$Sp_3sp_3(\pm Sp_1$, or $Sp_2)$	5	15	5.00	15.00		
1453	Burnetholm S_2	$Sp_3sp_3(\pm Sp_1$, or $Sp_2)$	9	13	5.50	16.50	2.98	0.1
321	Burnetholm S_2	$Sp_3sp_3(\pm Sp_1$, or $Sp_2)$	5	13	4.50	13.50	0.08	0.8
1454	Burnetholm S_2	$Sp_3sp_3(+Sp_1$, or $Sp_2)$	2	22	6.00	18.00	3.56	0.06
	Totals		21	63	21.00	63.00		

* An alternative genotype, with Sp_1 replacing Sp_2, and vice versa, would give the same expected ratio

Table 7. Segregations for mildew resistance in Burnetholm derivatives

Family	Parentage	Parental genotypes	Observed		Expected		χ^2	P approx.
			Res.	Sus.	Res.	Sus.		
					(13:3)			
872	91/161 × 129/5	$Sp_1sp_1Sp_2sp_2Sp_2sp_3$ × $Sp_1Sp_1Sp_2sp_2Sp_3sp_3$	37	10	38.19	8.81	0.20	0.7
					(1:1)			
873	129/91* × 91/161*	$Sp_1Sp_1sp_2sp_2Sp_3Sp_3$ × $Sp_1sp_1Sp_2sp_2Sp_3sp_3$	23	26	24.50	24.50	0.18	0.7
1424	129/91* × 239/27	$Sp_1Sp_1sp_2sp_2Sp_3Sp_3$ × Sp_2sp_2	29	30	29.50	29.50	0.02	0.8
	Totals		52	56	54.00	54.00	0.15	0.7
					(13:3)			
583	129/5 × 349/2*	$Sp_1Sp_1Sp_2sp_2Sp_3sp_3$ × $Sp_1sp_1Sp_2sp_2Sp_3sp_3$	77	9	69.88	16.12	3.87	0.3
					(3:1)			
1412	Burnetholm × 583/100	$Sp_1sp_1Sp_2sp_2Sp_3sp_3$ × $Sp_1Sp_1Sp_2sp_2sp_3sp_3$	66	23	66.75	22.25	0.03	0.8
					(9:7)			
1413	Burnetholm × 583/3	$Sp_1sp_1Sp_2sp_2Sp_3sp_3$ × $Sp_1sp_1Sp_2sp_2Sp_3Sp_3$	21	27	27.00	21.00	3.04	0.3
					(43:21)			
1407	Lloyd George × 583/15	$Sp_1sp_1Sp_2sp_2Sp_3sp_3$ × $Sp_1sp_1Sp_2sp_2Sp_3sp_3$	58	39	65.17	31.83	2.40	0.1
					(3:5)			
1478	577/5* × 765/18*	$Sp_1sp_1Sp_2sp_2Sp_3Sp_3$ × $Sp_1sp_1sp_2sp_2sp_3sp_3$	9	16	9.37	15.63	0.02	0.9
					(1:3)			
877	332/78* S	$Sp_1Sp_3sp_3$	13	49	15.50	46.50	0.54	0.5
876	332/73* S	$Sp_1Sp_3sp_3$	12	34	11.50	34.50	0.03	0.8
	Totals		25	83	27.00	81.00	0.20	0.7

* 129/5, 129/91 = Burnetholm S_2
332/78, 332/73 = 129/91 × S_1 Lloyd George
349/2 = 91/188 × 129/5
577/5 = Lloyd George × S_1 Lloyd George
765/18 = Burnetholm S_1
91/161, 91/188 = 55/6 × 72/59 (S_1 Malling Promise)

Table 8. Segregations for mildew resistance in derivatives of Malling Promise, Newburgh and 55/6

Family	Parentage	Parental genotypes	Observed		Expected		χ^2	P approx.
			Res.	Sus.	Res.	Sus.		
					(43:21)			
1584	55/6 S	$Sp_1sp_1Sp_2sp_2Sp_3sp_3$	48	18	44.34	21.66	0.92	0.3
					(7:1)			
1331	55/6 × 72/59	$Sp_1sp_1Sp_2sp_2Sp_3sp_3 \times Sp_1Sp_1Sp_2sp_2sp_3sp_3$	81	8	77.87	11.13	1.00	0.3
1841	288/28 × 91/188	$Sp_1Sp_1Sp_2sp_2sp_3sp_3 \times Sp_1sp_1Sp_2sp_2Sp_3sp_3$	49	10	51.62	7.38	1.06	0.3
					(3:1)			
1844	288/16 × 91/166	$Sp_1Sp_1Sp_2sp_2sp_3sp_3 \times Sp_1sp_1sp_2sp_2Sp_3sp_3$	43	19	46.50	15.50	1.05	0.3
					(1:1)			
1843	288/13 × 91/166	$Sp_1Sp_1sp_2sp_2sp_3sp_3 \times Sp_1sp_1sp_2sp_2Sp_3sp_3$	12	14	13.00	13.00	0.15	0.7
					(43:21)			
869	Newburgh × 91/161	$Sp_1sp_1Sp_2sp_2Sp_3sp_3 \times Sp_1sp_1Sp_2sp_2Sp_3sp_3$	64	33	65.17	31.83	0.06	0.8

Table 9. Segregations for mildew resistance and the gene *H* in Lloyd George derivatives

Parentage	No. of prog.	Observed				Expected without linkage				χ^2			P approx.		
		H Res.	*H* Sus.	*h* Res.	*h* Sus.	*H* Res.	*H* Sus.	*h* Res.	*h* Sus.	*H*:*h*	Res:Sus.	4-class	*H*:*h*	Res:Sus.	4-class
Lloyd George S_1	1	21	11	10	3	22.67	11.07	7.56	3.69	0.36	0.07	1.11	0.5	0.8	0.8
Lloyd George S_2	9	157	118	54	21	163.78	98.72	54.59	32.91	2.38	0.66	8.36	0.1	0.5	0.04
Hybrids (Table 3)	10	258	81	195	42	274.42	94.08	155.10	52.40	6.56	5.05	15.13	0.01	0.02	<0.01
Hybrids (Table 4)	7	197	31	75	3	173.51	34.49	82.92	15.08	10.26	5.84	13.97	<0.01	0.02	<0.01
Hybrids (Table 5)	5	54	45	94	32	75.90	36.60	75.90	36.60	3.24	0.29	13.14	0.07	0.6	<0.01

Segregations in progenies derived jointly from Lloyd George and Burnetholm (Families 1407, 1478, 876, 877) confirm that these varieties are heterozygous for the same three resistance genes, Sp_1, Sp_2, Sp_3.

Malling Promise derivatives

Segregations in a cross with Lloyd George suggest that Malling Promise is heterozygous for Sp_1, Sp_2, and Sp_3 (Family 1477, Table 3), and this is confirmed by segregations in progenies derived from Malling Promise or its relatives (Table 8). These progenies comprised Family 1331 (55/6 × 72/59 (Malling Promise S_1)), a self of seedling 55/6, an F_1 of Newburgh (the seed parent of Malling Promise) crossed with a resistant dwarf seedling 91/161 (55/6 × 72/59), and three progenies from crossing Family 288 selections (72/59 S_1) with resistant (91/188) and moderately susceptible (91/166) dwarfs.

Segregations in Families 1584 and 1331 are consistent with the theory that 55/6 (S_1 Burnetholm × S_1 Preussen F_2) is heterozygous for Sp_1, Sp_2, and Sp_3, and that 72/59 is $Sp_1sp_1Sp_2sp_2sp_3sp_3$. Segregations in Families 1841, 1843 and 1844 indicated that the three S_1 seedlings of 72/59 were, as expected from the parental genotype, homozygous for Sp_1 and sp_3. Newburgh appears to be heterozygous for Sp_1, Sp_2 and Sp_3, but further evidence is needed to confirm this.

All six progenies segregated for dwarf genes as well as for mildew resistance, and five showed a higher percentage of resistant plants in the dwarf than in the normal class. In the F_1 of Newburgh × dwarf, four-class segregations differed significantly from expectation. The data are inadequate to determine whether this was due to linkage; a contributory factor could be that the more extreme dwarf segregants tend to escape infection through early cessation of growth.

LINKAGE RELATIONSHIPS OF MILDEW RESISTANCE GENES

The gene H

The Lloyd George S_1 progeny, 10 out of the 12 S_2 progenies, and 22 of the hybrid progenies classified in Table 3, 4 and 5, showed segregation for both mildew resistance and the gene *H* controlling cane pubescence (CRANE and LAWRENCE, 1931)). All the selfed progenies, and 17 of the 22 hybrid families, showed a higher percentage of resistant plants in the glabrous (*h*) than in the hairy (*H*) group. Comparison of the total observed four-class ratios with expectation for groups of families of similar origin showed poor agreement with expectation in all except the small Lloyd George S_1 progeny (Table 9).

Two of the groups of families (those included in Table 3 and 4) showed significant deviations from expectation in segregations both for *H*:*h* (due to a deficiency and an excess of *h* plants respectively) and for resistant:susceptible (due to a deficiency of susceptibles in both cases). Although these two-class deviations inevitably contributed to the deviation from expectation in the four-class segregations, in both groups of families there was a marked excess in the proportion of resistants in the *h* class, and a much smaller excess of resistants in the *H* class. In the group totals for S_2 progenies of Lloyd George and for the hybrid progenies of Table 5, two-class ratios for resistant:susceptible agreed well with expectation, and the *H*:*h* ratios did not differ significantly

Table 10. Segregations for mildew resistance and *T* in Lloyd George derivatives

Family	Parentage*	Observed				Expected**				χ^2	*P* approx.
		T Res.	*T* Sus.	*t* Res.	*t* Sus.	*T* Res.	*T* Sus.	*t* Res.	*t* Sus.		
Coupling											
841	Lloyd George S_1	23	12	8	3	21.66	12.84	9.25	2.25	0.56	0.9
846	Lloyd George S_2	18	14	9	0	19.31	11.44	8.24	2.01	2.74	0.5
	Totals	41	26	17	3	40.97	24.28	17.49	4.26	0.51	0.9
852	Lloyd George S_2	18	8	9	0	20.66	5.58	7.76	0.98	2.59	0.5
850	Lloyd George S_2	24	11	11	0	25.88	8.62	8.62	2.88	4.33	0.2
851	Lloyd George S_2	4	29	6	10	5.49	31.26	6.76	5.49	4.36	0.2
	Grand total	87	74	43	13	93.00	69.74	39.63	14.61	1.13	0.8
						100.24	62.51	33.41	20.84***	9.56	0.02
Repulsion											
844	Lloyd George S_2	38	8	4	0	31.04	6.46	9.58	2.92	8.10	0.05
847	Lloyd George S_2	10	23	0	3	8.41	18.59	0.59	8.41	5.42	0.2
	Grand total	48	31	4	3	39.45	25.05	10.17	11.33	13.13	<0.01
						37.22	27.28	12.41	9.09***	13.41	<0.01
	Grand total of hybrid progenies	412	96	155	20	317.42	88.58	218.94	58.06***		

* For parental genotypes see Table 2

** *T*–Sp_3 linked with a C.O.V. of 25.7%

*** Expected without linkage

from those expected. In these two groups of families, the excess of resistants in the *h* class was matched by a similar excess of susceptibles in the *H* class.

Thus, in all groups of families there is a tendency for glabrousness and resistance to be associated. Possible reasons for this are:

(1) The gene *h* of itself tends to confer resistance to mildew.
(2) Susceptible *hh* plants are less viable than resistant *hh* plants and than susceptible *H* plants.
(3) Linkage between *h* and one or more of the resistance genes.

As regards (1), the occurrence of 4 hybrid progenies in this material in which a slightly higher percentage of resistants occurred in the *H* than in the *hh* group and the same situation in 7 out of 10 Burnetholm progenies rules out a direct pleiotropic effect of *h* on mildew resistance. As regards (2), omitting Family 844 in which there was a marked deficit of *hh* plants, the ratio of *H*:*h* in selfbred progenies was 296:102, agreeing well with 3:1. Of the hybrid progenies in Table 3, 4 and 5 which segregated for *H*, 8 out of 12 agreed well with a 1:1 expectation, the remaining 4 differing significantly from this owing to an *excess* of *hh* plants, while 8 out of 10 agreed well with 3:1, the remaining 2 (Families 1188, 1189) showing a marked deficit of *hh* plants. Clearly, there is no evidence that *hh* plants are consistently less viable than those carrying *H*, and hypothesis (2) is untenable.

Thus it seems evident that *h* is linked with some component(s) of mildew resistance in Lloyd George, but the data are inadequate to determine which resistance gene (or genes) is so linked; 29 progenies were segregating for Sp_1 and/or Sp_2, while 24 were segregating for Sp_3, so that linkage with any of these three genes would affect most of the families.

Of Burnetholm derivatives, the S_1 progeny, six S_2 progenies and three of the hybrid progenies classified in Table 7 segregated for *H* (derived from Burnetholm) as well as for mildew resistance. In Family 765 (Burnetholm S_1), the ratio of resistant to susceptible in the *H* class was 71:30 compared with 17:14 in the *hh* class, suggesting that in Burnetholm it is the *dominant* allele, *H*, which is linked with one or more of the mildew resistance genes. However, clearcut evidence of linkage was lacking in other progenies owing to the small numbers involved, and even in total, four-class segregations did not differ significantly from expectation. This was undoubtedly partly due to three S_2 families which were probably crossovers.

The linkage group B-Sx_3-T-Ch_1

The Lloyd George S_1 progeny, 6 out of 12 S_2 progenies and 13 of the hybrid progenies included in Table 3, 4 and 5, segregated for the gene *T* (*T*, red fruit, dark spines; *t*, yellow fruit, green spines (CRANE and LAWRENCE, 1931) (Table 10).

Five of the selfed and 10 of the hybrid progenies showed a higher proportion of resistant plants in the *tt* than in the *T* class, and grand totals of four-class segregations in the selfed progenies differed significantly from expectation, assuming no linkage. However, it will be shown later from segregation ratios in Burnetholm derivatives that *T* and Sp_3 are linked with a crossover value of approximately 25.7%, and this value has been used in calculating expected ratios for selfs of Lloyd George. On this assumption, the total four-class segregation where *T* and Sp_3 were linked in coupling agreed well with expectation ($\chi^2 = 1.13$, $P = 0.8$); in this case, the totals for *T*:*t* and

Table 11. Segregations for mildew resistance and *T* in Burnetholm S_1, S_2 and S_3 progenies

Family	Parentage*	Observed				Expected**				χ^2	*P* approx.
		T Res.	*T* Sus.	*t* Res.	*t* Sus.	*T* Res.	*T* Sus.	*t* Res.	*t* Sus.		
Coupling											
765	Burnetholm S_1	70	39	22	4	63.57	37.68	27.14	6.62	2.70	0.5
318	Burnetholm S_3	15	4	1	0	11.80	3.19	4.44	0.56	4.31	0.2
1439	Burnetholm S_2	11	3	0	1	8.44	2.81	2.81	0.94	3.60	0.3
319	Burnetholm S_3	10	7	3	0	9.42	5.58	4.02	0.98	1.64	0.7
322	Burnetholm S_3	9	3	4	2	8.48	5.02	3.62	0.88	2.31	0.5
	Totals	19	10	7	2	17.90	10.60	7.64	1.86	0.17	0.99
1442	Burnetholm S_2	17	13	5	3	16.03	12.47	5.34	4.16	0.43	0.95
1447	Burnetholm S_2	5	6	6	5	9.28	7.22	3.09	2.41	7.70	0.05
1451	Burnetholm S_2	8	5	1	1	6.33	4.92	2.11	1.64	1.28	0.7
324	Burnetholm S_3	7	7	4	2	8.44	6.56	2.81	2.19	0.80	0.8
320	Burnetholm S_3	7	8	4	0	8.02	6.23	2.67	2.08	3.38	0.3
323	Burnetholm S_3	5	9	6	0	8.44	6.56	2.81	2.19	8.12	<0.05
	Totals	49	48	26	11	56.54	43.96	18.83	14.67	5.03	0.1
	Grand total	164	104	56	18	158.25	98.24	60.86	24.65	2.73	0.5
	Expected without linkage					164.35	92.15	54.76	30.74	6.83	0.1
Repulsion											
1443	Burnetholm S_2	17	4	3	1	15.52	3.23	4.79	1.46	1.14	0.8
321	Burnetholm S_3	4	8	1	5	4.20	9.30	0.30	4.20	1.98	0.5
1444	Burnetholm S_2	3	9	2	5	4.44	9.81	0.31	4.44	9.82	0.02
	Totals	7	17	3	10	8.64	19.11	0.61	8.64	10.12	0.02
	Grand total	24	21	6	11	24.16	22.34	5.40	10.10	0.23	0.95
	Expected without linkage					22.17	24.33	7.39	8.11	1.90	0.7

* For parental genotypes, see Table 6.

** *T*–Sp_3 linked with a C.O.V. of 25.7%

resistant: susceptible both agreed with those expected. However, the deficit of *tt* plants was so marked in Families 844 and 847, where *T* and Sp_3 appeared to be linked in repulsion, that the total observed four-class segregation differed significantly from expectation even assuming linkage. In the hybrid families, the deficit of the *tt* class was such, even where it was not statistically significant, as to render the calculation of ratios expected with linkage valueless. However, the grand totals for the hybrid progenies included in Table 10 show the disproportionate occurrence of resistants in the *tt* class.

LEWIS (1940) showed that *G* (referred to here as Ch_1 (KNIGHT and KEEP, 1958), normal vs. pale green leaf) is linked to *T* with a crossover value of 15%. As with the *tt* class, a disproportionate percentage of resistant ch_1ch_1 plants occurred in Lloyd George S_1, in all except one of the segregating S_2 progenies, and in 8 out of 9 of the hybrid derivatives. There was a deficit of ch_1ch_1 plants in all the S_2 and hybrid progenies, so that estimates of χ^2 values for agreement with expectation in these individual progenies are valueless. Since *T* is linked with Ch_1 (LEWIS, 1940), the resistance gene Sp_3 is presumably linked with both *T* and Ch_1, but in view of the general deficit of ch_1ch_1 plants, a crossover value for Ch_1-Sp_3 has not been calculated.

Like Lloyd George, Burnetholm is heterozygous for *t*, and also for two genes linked with *T*, viz. *B* (waxy cane) and Sx_3 (normal vs. sepaloid flower) (KEEP, 1964; LEWIS, 1939, 1940). Although four-class segregations for mildew resistance and *T* in 14 individual S_1, S_2 and S_3 progenies agreed fairly well with expectation, the combined totals for all these progenies showed, as in Lloyd George derivatives, a higher proportion of resistant plants in the *tt* than in the *T* class (Table 11). The totals for 7 hybrid progenies derived from Burnetholm (Table 12) showed a similar deviation from expectation; combined segregations for two of the progenies (Families 876, 877), which owed their resistance to heterozygosity for Sp_3 alone, differed significantly from expectation also ($\chi^2 = 14.24$, $P = 0.004$). Using the product method a crossover value of 25.7% for *T* and Sp_3 was obtained from these two families; expected values in Table 11 and 12 have been calculated on this basis. Families 321 (Burnetholm S_3) and 1444 (Burnetholm S_2), which were also segregating for Sp_3 only, were omitted when estimating linkage as being too small to be certain that *T* and Sp_3 were linked in repulsion, as the segregation ratios suggested.

Most of these progenies were too small for individual χ^2 tests of significance to be very meaningful, but the Burnetholm S_1 progeny of 135 plants agreed well with expectation assuming a 25.7% C.O.V. between *T* and Sp_3 ($\chi^2 = 2.70$, $P = 0.5$; in the absence of linkage the χ^2 value is 5.59, $P = 0.12$). The combined totals for six S_2 and S_3 progenies (whose parents were $Sp_1sp_1Sp_2sp_2Sp_3Sp_3$) agreed fairly well with the expected 27:21:9:7, and the remaining small progenies were also in accord with expectation apart from Family 1444, where the high χ^2 value (9.82) was largely due to the small size (0.31) of the minimum expected class. χ^2 values of 2.73 and 0.23 ($P = 0.5$, 0.95, respectively), were obtained for the grand totals of four-class segregations assuming linkage in coupling and in repulsion, whereas in the absence of linkage, the χ^2 values were 6.83 and 1.90 ($P = 0.14$, 0.7).

Of the hybrid derivatives of Burnetholm, segregations in all seven progenies agreed well with expectation assuming linkage between Sp_3 and *T*. The grand total for the hybrid progenies also agreed well with expectation assuming a 25.7% crossover value

Table 12. Segregations for mildew resistance and *T* in Burnetholm derivatives

Family	Parentage*	Observed				Expected**				χ^2	*P* approx.
		T Res.	*T* Sus.	*t* Res.	*t* Sus.	*T* Res.	*T* Sus.	*t* Res.	*t* Sus.		
583	129/5² × 91/188	57	8	20	1	55.13	9.37	20.12	1.38	0.37	0.95
1412	Burnetholm × 583/100	40	15	26	8	33.37	11.13	33.37	11.13	5.17	0.15
1413	Burnetholm × 583/3	8	15	13	12	13.50	10.50	13.50	10.50	4.40	0.2
1407	Lloyd George × 583/15	24	25	34	14	30.02	18.49	35.16	13.33	3.57	0.3
1478	577/5 × 765/18	6	15	3	1	7.03	11.72	2.34	3.91	3.42	0.3
877	332/78S	7	40	6	9	6.94	39.56	8.56	6.94	1.38	0.7
876	332/73S	5	31	7	3	5.15	29.35	6.35	5.15	1.06	0.8
	Totals	12	71	13	12	12.09	68.91	14.91	12.09	0.31	0.95
	Grand total	147	149	109	48	151.14	130.12	119.40	52.34	4.12	0.3
						163.17	118.08	107.35	64.40***	13.90	0.005

* For parental genotypes see Table 7

** T–Sp_3 linked with a C.O.V. of 25.7%

*** Expected without linkage

($\chi^2 = 4.12$, $P = 0.3$), and differed significantly from expectation without linkage ($\chi^2 = 13.90$, $P = 0.005$).

Clearly, in families whose parents have well authenticated resistance genotypes and in which segregations for *T* and for resistance are individually in agreement with expectation, linkage between *T* and Sp_3 accounts for the excess of resistants in the *tt* class.

Thirteen progenies derived from Burnetholm segregated for *B* as well as mildew resistance, and individual and total four-class segregations agreed reasonably well with expectation in the absence of linkage, as did the combined figures for Families 876 and 877, from which the crossover value of 25.7% for *T* and Sp_3 was obtained. Clearly the *T* locus must lie between Sp_3 and *B*.

Burnetholm S_1, seven S_2 and six hybrid progenies segregated for the gene Sx_3, which is situated between *B* and *T* (KEEP, 1964). Individual and total four-class segregations agreed with expectation. A χ^2 value of 5.35 for the combined segregations of Families 876 and 877 compares with one of 1.08 obtained from the same two families for *B* and resistance, suggesting the possibility of a loose linkage between Sx_3 and Sp_3.

The gene S

Of ten families segregating for *S* (spiny vs. spineless canes (LEWIS, 1939)), all showed a higher percentage of resistant plants in the *S* than in the *s* class. Four-class segregations in Families 765 (Burnetholm S_1), 1442 + 1447 (Burnetholm S_2) and 1412 (Burnetholm × 583/100) all differed significantly from expectation, as did the grand totals for selfed and for hybrid derivatives. Aberrant segregations for *S* in Families 765 and 1442 obviously affected χ^2 values for these families and for the grand total for selfs of Burnetholm, and the data were inadequate to determine whether the aberrant four-class segregations were due to linkage between *S* and resistance genes.

DISCUSSION

Of the three varieties, Lloyd George, Burnetholm, and Malling Promise whose breeding behaviour is discussed here, Malling Promise and its derivatives in Family 91 appear to be the best source of mildew resistance, followed by Lloyd George. Resistance genes in these varieties confer strong resistance or field immunity. The level of resistance in Burnetholm derivatives is generally lower.

All three varieties are heterozygous for resistance, and evidence for an identical three gene control in Lloyd George and Burnetholm is strong. Malling Promise appears to be heterozygous for the same three genes, but fewer progenies were available to confirm this hypothesis.

In the derivatives of Lloyd George, Burnetholm and Malling Promise discussed in this paper, resistance, whether due to $Sp_1 + Sp_2$ or to sp_3sp_3, appeared to be epistatic to susceptibility. However, segregations (KEEP, unpublished) in progenies derived in part from other varieties, although in accord with a three-gene hypothesis, suggest that susceptibility max, in some varieties, be epistatic to resistance.

Adequate data have been presented to show that the selections 341/3 and 341/16 (55/6 × 72/59 F_2) are $Sp_1sp_1Sp_2sp_2sp_3sp_3$ (Table 3 and 5), and the genotype of 129/5

(Burnetholm S_2) is fairly well authenticated as $Sp_1Sp_1Sp_2sp_2Sp_3sp_3$ (Table 6 and 7). All these selections have been widely used as parents. The genotypes assigned to other seedling selections on the basis of segregation ratios in only one or two small progenies are necessarily tentative.

The difference in severity of infection shown by susceptible Lloyd George and Malling Promise derivatives as compared with those of Burnetholm, suggests the presence of minor resistance genes causing by their segregation considerable variation in levels of susceptibility within individual progenies.

Aberrant segregations for the genes *H*, *T*, Ch_1, and *S* were relatively common in the Lloyd George progenies and, to a lesser extent, in Burnetholm derivatives. The significance of these phenomena in the determination of resistance genotypes and linkage relationships of resistance genes requires further consideration.

Segregation ratios for resistance in four Lloyd George hybrid progenies (Families 1185, 1191, 1194, and 1221) differed significantly from expectation (Table 3 and 4) owing to a deficiency of susceptible plants. Since all were artificially inoculated, this is unlikely to be due to chance failure of infection. Of these progenies, Family 1185 showed a significant deficiency of the *H* class, the other three families segregating normally for this gene, while Families 1191 and 1221 showed a deficit of the *tt* class, Family 1194 segregated normally and Family 1185 did not segregate for *T*.

All four of these progenies were segregating for Sp_3, so, assuming *t* to be linked in repulsion with Sp_3 in the parent, the deficiency of *tt* plants in Family 1221 could be a concomitant of the observed deficiency of susceptibles. Family 1221, with a four-class segregation of 56 *T* Res.: 3 *T* Sus.: 4 *t* Res.: 0 *t* Sus., clearly showed an excess of resistants in the *T* class over the maximum (13:3) to be expected in a segregating F_2 on the three-gene basis. That 577/35, the parent of this family, *is* heterozygous for resistance is shown by segregation in Families 1214 (Table 4) and 1205 (Table 5). However, in Family 1214, in which there was no significant deficit of *tt* plants, the four-class segregation of 31 *T* Res.: 5 *T* Sus.: 9 *t* Res.: 0 *t* Sus. (with an expectation of 7 Res.: 1 Sus.) suggests rather that *T* is linked in coupling with Sp_3 in 577/35, and the deficiency of susceptibles in Family 1221 cannot therefore be attributed solely and directly to deficiency of the *tt* class. In Family 1191, with no susceptible plants, the ratio of *T*:*t* was 44:28 for a 1:1 expectation. However, the reciprocal Family 1190, in which the resistant : susceptible ratio agreed well with expectation (Table 4), showed an even more marked deficit of *tt* plants (40:12). Thus, aberrant segregations for resistance are not directly associated with aberrant segregations for *T* in Families 1221 and 1191, nor in Families 1194 (normal *T*:*t* ratio) and 1185 (not segregating for T). Clearly, more or less normal segregations for resistance can occur even in progenies with a marked deficit of the *tt* class.

The occurrence of aberrant segregations for both *H* and mildew resistance in Family 1185 (Table 3) has already been mentioned. However, aberrant segregations for *H* accompanied by apparently normal segregations for resistance occurred in six progenies derived from Lloyd George: Family 844 (Table 2); Family 1183 (Table 3); Families 1188, 1189 (Table 4); and Families 1205, 1210 (Table 5). The reciprocal of Family 1183, Family 1182, with a perfect 1:1 segregation for *H*, contained 39 resistant and 9 susceptible plants, in good agreement with the 32:10 ratio in Family 1183 (Table 3). Thus, as for the gene *T*, resistance genotypes can validly be assigned to

parents whose progenies show abnormal segregation for *H*, even though linkage between *H* and one or more of the resistance genes has been postulated.

It seems likely that several factors are responsible for aberrant segregations for mildew resistance, including segregation of minor resistance genes, and slightly increased mortality of susceptible young seedlings, particularly in the inherently less viable *tt* and ch_1ch_1 classes. In addition, whatever the cause of aberrant segregations for *T* and *H* (over and above the reduced viability of the *tt* and ch_1ch_1 classes), this may influence segregations of linked resistance genes, although it has been shown that this effect appears to be slight

The excellent agreement with expectation, assuming linkage, of four-class segregations in all progenies in which two-class segregations for *T* and for resistance were in good agreement with expectation (Table 10, 11 and 12) shows that in both Lloyd George and Burnetholm, Sp_3 and *T* are linked in coupling, although the crossover value needs confirmation. Since the genes *B* and Sx_3 show no evidence of linkage with resistance it can be assumed that the gene order is $B\text{-}Sx_3\text{-}T\text{-}Sp_3$.

Linkage between *H* and one or more of the resistance genes can obviously be proven only by raising larger progenies in which each of the resistance genes is segregating on its own. Nevertheless, incidental evidence for the existence of such linkage is strong; the occurrence of an undue proportion of *hh* resistants in most Lloyd George derivatives and the reverse situation in most Burnetholm progenies is adequately accounted for on this basis.

It has already been found that the genes *H*, *T*, *B* and *S*, are associated with differences in the incidence of three other fungal diseases attacking the raspberry. The gene *H* has a marked effect in reducing the incidence of cane grey mould *(Botrytis cinerea)* and spur blight *(Didymella applanata)* probably through some direct protective effect of cane hairs (JENNINGS, 1961, 1962; KNIGHT, 1962a, b; KNIGHT and KEEP, 1958, 1966). JENNINGS (1961) also found these diseases and cane spot *(Elsinoë veneta)* to be reduced in spineless *(ss)* and, usually, in yellow-fruited *(tt)* plants. In contrast, cane spot attacks were usually worse in plants carrying *H*, and JENNINGS produced evidence suggesting the greater importance of tissue resistance against this disease. Tissue resistance under major gene control is clearly the main factor affecting the incidence of powdery mildew in this material, the association of differential resistance with the genes *H* and *T* being due to genetic linkage. Powdery mildew affects primarily young growing tissues, whereas *Botrytis* and spur blight are more prevalent on older parts of the cane, so that radically different protective mechanisms might be expected to have evolved in the host plant.

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