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A gene-for-gene model to explain interactions between cultivars of strawberry and races of *Phytophthora fragariae* var. *fragariae*

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Abstract A gene-for-gene model is postulated to explain the observed interactions between cultivars of strawberry and races of *Phytophthora fragariae*. Five interacting resistance (R1–R5) and avirulence (Avr1–Avr5) factors explain all the available data involving 15 host genotypes, including the USA and Canadian differential series, and 12 pathogen isolates from North America. Interactions between pathogen isolates and UK and German differentials are also explained by the proposed model. The model makes it possible to develop a universally applicable differential series, to present a systematic, unequivocal nomenclature of races, and to increase the efficiency of breeding programs.

Key words *Fragaria* spp. · Red stele · Red core · Resistance

Introduction

Phytophthora fragariae (Hickman 1940) var. *fragariae* (Wilcox et al. 1993) is the causal agent of red stele (red core) root rot in strawberry (*Fragaria* spp.). It is widely assumed that resistance to this soil-borne fungus is inherited polygenically (Stembridge and Scott 1959; Scott et al. 1984). However, the race specificity (e.g., Converse 1970; Montgomerie 1967; Kennedy and Duncan 1993; Nickerson and Murray 1993; Van de Weg et al. 1997a) and the observed Mendelian segregation of resistance (Van de Weg et al. 1989) suggest that genotypes of strawberry and races of *P. fragariae* exhibit a gene-for-gene (GFG) relationship as first described by Flor (1956) for flax and flax rust (*Melampsora lini*). The reports published on host-pathogen interaction data appeared at first too inconsistent (Montgomerie 1967; Van de Weg 1989b; Milholland 1994; Van de Weg et al. 1997a) to derive GFG patterns. However, a number of the inconsistencies have been determined to be due to differences in the evaluation of incompletely expressed resistance, to interchanges of cultivars which were incorrectly presumed to have the same resistance, and to the existence of different genotypes under the same cultivar name (Van de Weg et al. 1997a). In the present article data from which the inconsistencies were removed (Van de Weg et al. 1997a) have been used to derive a GFG model which satisfactorily explains the available observations.

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Materials and methods

Data

To the data of Van de Weg et al. (1997a), classifications according to Converse (1970) and Nickerson and Murray (1993) were added (Table 1) and analyzed using methods described by Person (1959). The ability of the resulting GFG model to describe interactions of other cultivar-race combinations was tested on published classifications and on classifications deduced from published disease assessments (see Tables 2 and 3). Host genotypes were classified following Van de Weg et al. (1996), being resistant if they were considerably less diseased than one of the universally susceptible cultivars 'Tennessee Beauty' and 'Senga Sengana', or if they scored 3 or less on the disease severity scale of Kennedy and Duncan (1993).

Resistance and virulence

The GFG hypothesis presumes that for each resistance factor R in the host, there is a corresponding avirulence factor in the pathogen (Newton and Andrivon 1995). The product of the resistance allele is postulated to recognize the product of the corresponding avirulence allele, which initiates the resistance response (Keen 1990). In contrast, the product of the virulence allele, if it occurs (De Wit 1992),

Table 1 Resistant (−) and susceptible (+) reactions of 12 strawberry genotypes to 9 USA (A) and 3 Canadian (NS) isolates of *Phytophthora fragariae* var. *fragariae* together with their postulated resistance and avirulence factors. Data are according to Van de Weg et al. (1997a) with additional data (encircled) from Converse (1970) and Nickerson and Murray (1993)

Host genotypes Name	Proposed resistance factors	Isolates and their proposed avirulence factors								
		A9	A1	A2[e]	A8	A7[d]	A3	A6	NS4	NS2
		1	1	1	.	.	1	1	.	1
		2	.	2	.	2	.	.	.	.
		3?[e]	3	3?	3	.	.	3	.	.
		4	4	.	4	4	4	.	4	.
		5	5	5	5	5	5	5	5	5
Blakemore[a]	0	+	+	+	+	+	+	+	+	+
Md683	1	−	−	−	+	+	−	−	+	−
Sparkle[b]	2	−	+	−	+	−	+	+	+	+
Del Norte	4	−	−	+	−	−	−	+	−	+
Yaquina A	5	−	−	⊖	⊖	−	−	−	⊖	⊖
Yaquina B	5	−	−	−	−	−	−	−	−	−
Siletz	1.2	−	−	−	+	−	−	−	+	−
Stelemaster	1.2	⊖	⊖	⊖	+	−	⊖	−	+	−
Perle de Prague	1.3	−	−	−	−	+	−	−	+	−
Aberdeen	2.3	−	−	−	−	−	+	−	+	+

[a–d] Genotypes or isolates having identical interactions: [a] 'Senga Sengana'; [b] 'Climax' and 'Redgauntlet'; [c] A4 and A10; [d] NS3
[e] Available data allow no conclusion on the presence or absence of the avirulence gene due to the absence of an R3 differential

will not be recognized by the product of the resistance allele, resulting in virulence of the pathogen and susceptibility of the host (Newton and Andrivon 1995). Thus, resistance (incompatibility of host and pathogen genotypes) occurs if a cultivar carries at least one resistance allele for which the pathogen carries the corresponding avirulence allele. Susceptibility (compatibility) requires either the absence of R-alleles or the avoidance of each R-allele in the host by the absence of the matching avirulence alleles in the pathogen.

Denotation

GFG analysis of interactions between host cultivars and pathogen isolates leads, strictly spoken, to the identification of factors for resistance and avirulence and not to genes (Person 1959). Therefore it is proposed to delay the use of the latter term until the individual identity of a factor has been substantiated by inheritance studies.

Host genotypes are denoted by their resistance factors, regardless of the number of alleles present. In this way problems arising from variations in ploidy level among *Fragaria* species and the indistinctness of their genome formula are circumvented (Bringhurst 1990; Galletta and Maas 1990). Similarly, pathogen genotypes are denoted by their avirulence factors, which circumvents the indistinctness of the ploidy level of *Phytophthora* species (Brasier 1992).

Results

The resistant and susceptible reactions listed in Table 1 can be explained by a GFG relationship involving five interacting pairs of resistance (R1–R5) and avirulence (Avr1–Avr5) factors. In this model, 'Blakemore' and 'Senga Sengana' have no resistance, while 'Md683' possesses just R1 (Table 1). Following the GFG concept, all isolates to which 'Md683' is resistant carry Avr1, while this factor is absent in all isolates to which 'Md683' is susceptible. Besides 'Md683', other single-factor genotypes are 'Climax', 'Redgauntlet', and 'Sparkle', (all carrying R2), 'Del Norte' (R4), and 'Yaquina A', and 'Yaquina B' (R5). Host genotypes with two factors are 'Siletz' (R1.2), 'Stelemaster' (R1.2), 'Perle de Prague' (R1.3), and 'Aberdeen' (R2.3) (Table 1). Among the host genotypes in Table 1 none contained R3 alone. This prevented the full assessment of isolates A2, A4, A9, and A10 (see footnotes to Table 1).

'Sparkle' (R2) and 'Stelemaster' (R1.2) are both resistant to A7, as they carry R2 for which A7 has the matching avirulence factor (Avr2). They are both susceptible to A8 (Avr3.4.5), as this isolate lacks Avr1 and Avr2.

The resistances and virulences of UK differentials and isolates can also be explained by the proposed GFG model (Table 2). The same is true for isolates from Germany and the USA, and for various North American strawberry cultivars, except for the reactions of 'Cardinal', 'Gilbert', 'Allstar', 'Midway', and 'Crimson King' (Table 3).

The genotypes '52AC18' (Table 2), 'MicMac', 'Blomidon', and 'Kent' (Table 3) are putative R3 differentials. If this holds true, A2 and A4 should possess Avr3 and A9 should not (Table 3).

Discussion

A GFG model for the interaction between cultivars of strawberry and races of *P. fragariae* var. *fragariae* is proposed. The model clarifies the genetics of resistance

Table 2 Genotypes of UK strawberry differentials and UK isolates. The incompatibilities of genotype-isolate combinations are deduced from Kennedy and Duncan (1988, 1993) and Kennedy et al. (1986)

Host genotypes	Proposed resistance factors	Isolates and their putative								
		B1 168[b]	B2 172	B4 499	B9 372	B3 169	B11A 171	B10 173	B11B 293	B11D1 452
		Avirulence factors[a]								
		1	.	1	1	1	.	.	1	.
		2	2	.	.	2	.	.	.	.
		3	3	3	3	.	3	3	.	.
		4?	4?	4?	4?	4?	4?	4?	4?	4?
		5	5	5	5	5	5	5	5	5
Fragaria vesca	0	+	+	+	+	+	+	+	+	+
Md683	1[c]	−	+	−	−	−	+		−	+
Climax	2[c]	−	−	+	+	−	+	+	+	+
52AC18	3	−	−	−	−	+	−	−	+	+
Yaquina B	5[c]	−	−	−	−	−	−		−	−
Stelemaster	1.2[c]	−	−	−	−	−	+	+	−	+
Aberdeen	2.3[c]	−	−	−	−	−	−	−	+	+
Cambridge Vigour	2.3				−	−			+	
Saladin	2.3	−	−		−	−	−		+	+
Perle de Prague	1.3[c]	−	−	−	−	−	−	−	−	+
Hood	1.2.3	−	−	−	−	−	−	−	−	+
Linn	1.3/2.3/1.2.3[d]				−	−	−			+
Tantallon	1.3/2.3/1.2.3[d]	−	−		−	−	−			+

[a] Although the isolates of this table were also tested on 'Del Norte' (see Table 1), the reported disease ratings were considered insufficiently conclusive. Consequently, no conclusions could be drawn about the presence or absence of Avr4

[b] Culture number of isolate

[c] Genotypes of these genotypes are according to Table 1 and are the basis for the assignment of genotypes for the fungal isolates and the other strawberry genotypes

[d] No final conclusion can be drawn on these genotypes as their resistance to isolates B10 and B11B is not known

in the host and the genetics of avirulence in the pathogen. It consists of five resistance (R1–R5) and five avirulence (Avr1–Avr5) factors.

Since the GFG relationship was first proposed (Flor 1942; Oort 1944), it has been helpful for the description and interpretation of the genetical relationship in many host-pathogen combinations (Thompson and Burdon 1992), including *Phytophthora* spp.; potato-*P. infestans* (Toxopeus 1956) and soybean-*P. megasperma* var. *sojae* (Ellingboe 1983). Its validity, characteristics, prospects, and limitations have been reviewed frequently (e.g., Flor 1971; Crute 1985; Keen 1990; Heath 1991; De Wit 1992; Newton and Andrivon 1995).

The reliability of the model

The reliability of the present model is supported by its consistency when related cultivars are considered. 'Climax', 'Redgauntlet', and 'Sparkle' are likely to have received R2 from their common ancestor 'Aberdeen' (R2.3) (Reid 1952; Scott et al. 1984). The resistance of 'Stelemaster' (R1.2) is also consistent with its ancestry, as it was selected from the cross ['Aberdeen' (R2.3) × 'Fairfax' (R0)] × Md683 (R1) (Scott et al. 1984). Moreover, the resistance of most North American cultivars created at, or in cooperation with, the Beltsville ARS, MD, USA, strawberry breeding program, traces back to 'Md683' (R1) and 'Aberdeen' (R2.3). These cultivars were screened for resistance to a composite of the isolates A1, A2, A3, A4, and A6 (Maas et al. 1989), and should therefore have genotypes: R1, R1.2, R1.3, or R1.2.3. This prediction is in agreement with the postulated genotypes of 'Darrow', 'Earliglow', 'Guardian', 'Lateglow', 'Lester', 'Redchief', 'Stelemaster', and 'Tristar' (Table 3).

The reliability of the model is further supported by recent studies which showed the resistance of 'Md683' to isolate NS2-25, and the resistance of 'Climax', 'Redgauntlet', 'Sparkle', and 'Siletz' to A7 was monogenically inherited (Van de Weg 1997; Van de Weg et al. 1997b). The resistance gene of 'Md683' has been called *Rpf1* and that of the other cultivars *Rpf2*. Caution in the assignment of individual genes to the other resistance factors is proposed until their individual identity has also been substantiated by inheritance studies. Inheritance studies on avirulence factors are impeded by the homothallism (Bain and Demaree 1945) and ploidy level of *P. fragariae* (Brasier 1992). Consequently, the existence of a GFG relationship in the strict sense cannot be proved (Crute 1985; Sidhu 1975).

Sources for resistance in addition to those discussed here have been reported (Reid 1952; Montgomerie 1960; Scott et al. 1962, 1984; Daubeny and Pepin 1965). The testing of these additional sources or new isolates

Table 3 Genotypes of strawberry cultivars and of isolates from North Carolina (USA) and Germany. The interactions of genotype-isolate combinations are according to Maas et al. (1988, 1989) and Table 1 or deduced from Milholland et al. (1989), Law and Milholland (1992), and Scheewe (1994)

Host genotype	Proposed resistance factors	Isolates, their origin, and putative avirulence factors												
		USA differential key							North Carolina		Maine	Germany		
		A9	A2	A8	A7	A3	A6	A5	NC1	NC2[d]	Me4K[e]	11/2	47/1	45/11[f]
		1	1	.	.	1	1	.	.	1	.	1	1	1?
		2	2	.	2	.	.	.	2	2	2	.	.	2
		.	3	3	.	.	3	.	.	3?	.	.	.	3?
		4	.	4	4	4	.	4	4?	4?	4	4?	4?	4?
		5	5	5	5	5	5	5	5	5	5	5	.	5
Blakemore[a]	0[g]	+	+	+	+	+	+	+			+			
Senga Sengana	0[g]	+	+	+	+	+	+					+	+	+
Tennessee Beauty	0	+	+	+	+	+	+	+	+					
Md683	1[g]	−	−	+	+	−	−	+	+	−	+			
Sunrise	1	−	−	+	+	−	−	+	+		+			
Climax	2[g]	−	−	+	−	+	+	+	−	−		+	+	−
Sparkle	2[g]	−	−	+	−	+	+	+	−	−	−			
MicMac[b]	3	+	−	−	+	+	−	+			+			
Del Norte	4[g]	−	+	−	−	−	+	−	?[f]	?	−			
Yaquina A	5[g]	−	−	−	−	−	−	−	−	−				
Yaquina B	5[g]	−	−	−	−	−	−	−	−		−	−	+	−
Aberdeen	2.3[g]	−	−	−	−	+	+	+			−			
Darrow	1.2	−	−	+	?	−	−	+	−					
Lester[c]	1.2	−	−	+	−	−	−	+			−			
Siletz	1.2[g]	−	−	+	−	−	−	−	−					
Stelemaster	1.2[g]	−	−	+	−	−	−	+	−	−	−			
Guardian	1.3	−	−	−	+[i]	−	−	+	+			−	−	−
Redchief	1.3	−	−	−	+	−	−	+			+			
Saladin	2.3[g]											+	+	-
Earliglow	1.2/1.2.3	−	−	±[h]	−	−	−	+	−		−			
Delite	1.2.3	−	−	−	−	−	−	+	−					
Lateglow	1.2.3	−	−	−	−	−	−	+			−			
Surecrop	1.2.3	−	±[h]	±[h]	−	−	−	+	−	−				
Tristar	1.2.3	−	−	−	−	−	−	+						
Cardinal	0?	+	+	+	+	+	+	+	⊖[j]					
Gilbert	0?	+	+	+	⊖	+	+	+			+			
Allstar	1?	−	−	+	+	−	−	+	⊖					
Midway	2?	−	−	⊖	?	+	+	+	?					
Crimson King	3?	+	−	⊕	+	+	−	+			+			

[a–e] Genotypes or isolates with identical classifications: [a]'Bounty', 'Canoga', 'Honeoye', 'Jewel', 'Raritan' and 'Vesper'; [b] 'Blomidon' and 'Kent'; [c] 'Annapolis' and 'Cornwallis'; [d]NC3; [e]Me5J and Me7F

[f] Disease ratings allow no conclusive classification

[g] Genotypes are according to Tables 1 and 2; they are used for the assignment of genotypes for the other genotypes and isolates

[h] Inconsistent data among Maas et al. (1989), Milholland et al. (1989), and Law and Milholland (1992)

[i] According to Van de Weg and Henken (unpublished)

[j] Encircled classifications are inconsistent with the suggested genotypes

may lead to the identification of more interacting resistance and avirulence factors. It is possible that the number of resistance and avirulence factors in the model will need to be increased if the data recorded for 'Cardinal', 'Gilbert', 'Allstar', 'Midway', and 'Crimson King' (Table 3) are reproducible.

Nomenclature of races

In other host-pathogen combinations, fungal races are often named according to the virulence factors they carry (De Wit 1992). This system is informative, simple, and easily applied provided the number of resistance factors is not too large, as in the case described in this paper. Since virulence is perceived to be the absence of avirulence it is not strictly correct to use the denotation 'virulence factor' or 'virulence gene'; even the deletion of an avirulence locus would result in virulence. Consequently, the concept of virulences is used (Parlevliet 1995), and a nomenclature based on this is proposed. Race 1.3 thus carries virulences 1 and 3 overcoming the resistance factors R1 and R3, isolate A7 being a representative of this race (Table 1).

It should be noted that the assignment of a given isolate to a race is never absolute since new virulences

may be identified when the isolate is tested on a larger set of differentials.

Composition of an international set of differential cultivars

The most efficient set for the testing of single-spore isolates consists of host genotypes each possessing a single, unique resistance factor and together comprising all known R-factors (Person 1959; Flor 1971). A universally susceptible cultivar should be included to identify isolates lacking any specific avirulence factors (race Avr0). The set could thus consist of genotypes such as 'Blakemore' (R0), 'Md683' (R1), 'Sparkle' (R2), 'Del Norte' (R4), 'Yaquina A' (R5), and one of the genotypes assumed to carry R3 ('Blomidon', 'Kent', 'Micmac', '52AC18'). As long as an R3 differential has not been definitively identified, 'Perle de Prague' (R1,3) and 'Aberdeen' (R2,3) can be included (Van de Weg et al. 1997a). This makes it possible to detect the presence or absence of Avr3 in races showing at least one of the virulences 1 and 2.

Current differential sets

The GFG model clarifies the similarities, differences, and shortcomings of the current, national differential sets. They consist of cultivars which have been selected for their ability to distinguish isolates, without knowledge of the number or identity of the resistance genes they carry. Therefore, it is not surprising that the composition of these sets is not optimal. The USA set consists of 'Blakemore', 'Md683', 'Aberdeen', 'Stelemaster', and 'Yaquina A' (Converse 1970); lacking 'single-factor-genotypes' for R2 and R3. The Canadian set is similar to that of the USA except that 'Sparkle' is used instead of 'Aberdeen' (Nickerson and Murray 1993); lacking an R3 differential, A6 (race 2.4) could not be distinguished from NS2 (race 2.3.4) (Nickerson and Murray 1993). The set proposed by Milholland et al. (1989) lacked a differential for R3 and R5. This set also failed a differential for R2.3 because the relative clone of 'Aberdeen' seemed not to true to type (Van de Weg et al. 1997a). Consequently, A1 (race 2) could not be distinguished from A3 (race 2.3) (Milholland et al. 1989).

In the UK, various differential sets have been used. They consist of different combinations of the genotypes *Fragaria vesca* (R0), 'Cambridge Favourite', 'Climax', 'Redgauntlet', '53Q13', 'Del Norte', 'Yaquina B', 'Siletz', 'Perle de Prague', 'Aberdeen', 'Cambridge Vigour', 'Saladin', 'Talisman', '52AC18', 'Linn', 'Hood', and 'Tantallon' (Montgomerie 1967; Kennedy et al. 1986; Kennedy and Duncan 1988, 1993). Only the paper of Kennedy and Duncan (1993) contained all of the required differentials.

Breeding for resistance

The GFG model can help breeders to specify their breeding goals for resistance accurately in terms of desired resistance genes, to decide on the host genotypes to hybridize and the isolates to screen with. For instance, R1, R2, and R3 can be pyramided by crossing 'Md683' and 'Aberdeen' and by testing the descendants for resistance to races 2.3 (A3), 1.2 (A8), and 1.3 (A7), and for susceptibility for race 1.2.3 (NS4).

Stability of resistance and distribution of races

The GFG model sheds new light on the previously reported 'breakdown' of resistance. 'Climax' was the first European cultivar originating from a specific breeding program for resistance. However, soon after its release it became severely diseased at many locations, which resulted in an assumption of the occurrence of races (Reid 1948, 1952). 'Climax' derived its resistance from 'Aberdeen', a cultivar which had remained resistant until that time. This rapid 'breakdown' of resistance may have occurred because 'Climax' carries only one of the two resistance factors present in 'Aberdeen' and because the matching virulence was already widely present prior to the introduction of 'Climax'. To date, virulent races have been found to match each of the presently identified resistance factors. Since not all races yet occur in all growing areas, certain combinations of resistance genes will still be effective in red stele-infested areas.

Resistance factor R5 seems to be effective in many growing areas since 'Yaquina B' was resistant to all 300 European isolates examined by Kennedy and Duncan (1993), and to a total of 48 North American isolates reported on by Converse (1970), Milholland et al. (1989), and Nickerson and Murray (1993). However, this factor seems to be less effective in Germany and The Netherlands as it was not effective against one out of three German isolates (Table 3) and one out of two Dutch isolates tested by Van de Weg (unpublished).

The resistance conferred by R1.2.3 seems also to be highly effective in many areas. Compatible isolates are found only occasionally. The first was isolated in 1964 in the Eastern United States as isolate A5 (Converse et al. 1966). Since then it has been isolated once more in the USA (Converse et al. 1966), Canada (isolate NS4) (Nickerson and Maas 1991; Nickerson and Murray 1993) and on three occasions in Europe (isolate B11-D) (Kennedy and Duncan 1993). Evidently, this race has never established itself widely during the past 40 years. The low zoospore-producing capacity of these isolates (Maas 1976a; Nickerson and Murray 1993), their low aggressiveness (Maas 1976b; Milholland et al. 1989; Van de Weg et al. 1997a), and instability (Kennedy and Duncan 1988, 1992) might be reasons for this.

The resistance conferred by R1.3 also seems to be effective in many areas. Race 1.3 has had a limited distribution for a long time, while more complex races are extremely rare (see above). This pathotype was not represented among the 300 European isolates tested by Kennedy and Duncan (1993). In the USA, it initially appeared on the west coast only. However, it was recently detected on the east coast of both the USA (Maas et al. 1988; Milholland et al. 1989) and Canada (Nickerson and Murray 1993), overcoming the resistance of some modern cultivars (Maas et al. 1988).

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