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Alternative Strategies in Fungal Race-Specific Parasitism

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Summary. Based on the gene-for-gene relation in race-specific resistance versus virulence, racial complexity of a pathogen population can be revealed by using host lines each with a single gene for resistance as detector. Such inventories of cereal rusts have shown: i. Genes for virulence may have pleiotropic effects acting on general fitness and their relative prevalence. ii. Genes for virulence are, as most other genes, dependent on genetic background for their general fitness. iii. Specific and general gene erosion in a pathogen population submitted to the assortative function of a race-specific host selection pressure is proportional to the degree of existing recombination and thus ultimately upon mode of reproduction (sexual or asexual). iv. Genetic storage capacity is dependent on ploidy constitution. v. Host alternation for safe annual survival favours a genetic system able to store temporarily unnecessary genes for virulence. – Due to shifting circumstances, pathogens like rusts will even inside the same *forma specialis* show different strategies. The trend may lead to a process of stabilizing selection and dependence on immediate and provisional flexibility just as typical of true haploids. It may lead to a pattern of preparedness: i.e. accumulation of 'unnecessary' genes for virulence. In the latter case, the modern concept of gene diversification in breeding for disease resistance is less effective. In the former case, gene accumulation can also work.

Key words: Race-specific resistance strategies – Cereal rusts and mildew – Gene accumulation versus diversification

Introduction

Every organism has to adapt its evolution to others. This interdependence is especially pronounced between a host and its parasite. Genetic strokes of defence are reciprocated by counter-strokes of attack. Over time such mutual provocation will tend to develop genetic systems built like mirror images of each other. The closest possible interaction is that of a gene-for-gene interrelation (Flor 1942).

Owing to restraint in genetic capacity, true resistance must not necessarily imply an exact counter-adaptation. Nonspecific resistance, characterized by slowing down or inhibiting phases during the reproduction cycle of the pathogen, may or may not induce a gene-for-gene interrelation. If so, corresponding genes do not need to fit exactly, since they work in an additive or complementary manner and mostly inside a polygenic system.

Race-specific resistance, characterized by causing an assortative reduction of the pathogen population already at the initial infection, is an incompatibility phenomenon and appears as such always to be qualitatively gene-for-gene related. If rare observations on cytoplasmic interference and very simple additive or complementary gene actions are taken as mere variations on a theme, two basic patterns can be observed. They are tied to two different systems of parasitism, one to facultative and one to obligate parasitism.

The first, less advanced type of parasitism is based on killing the host cell by secreting substances which either dissolve the cell walls or destroy the cytoplasm. The pathogen works with toxins which are either compatible or not with the host genotype. A gene for virulence has to correspond to a gene for susceptibility in the host (Ellingboe 1976).

An obligate plant parasite does not invade cells of the host. The parasite and host cells are separated by a complicated series of membranes through which nutrients are transported. Not killed, the host cell can produce toxins for defense. These antimetabolites can either act directly or indirectly by starving the obligate parasite to death as a cause of a hypersensitivity reaction of the host cell. A virulence gene of the pathogen corresponds with a specific gene for resistance in the host by somehow inactivating the trigger mechanism of the latter. The two systems, named the *Helminthosporium* and the *Melampsora* type, behave

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Table 1. Different systems of host resistance against a pathogen, presupposing two pairs of genes involved

1. INDEPENDENT NONSPECIFIC RESISTANCE

Gene for aggressiveness in the pathogen does not match directly with any gene for resistance in the host plant.

2. INTERDEPENDENT NONSPECIFIC RESISTANCE

Gene for aggressiveness in the pathogen matches gene for resistance in the host plant.

Pathogen aggressiveness genotype	Host resistance genotype – –	*A* –	– *B*	*A B*
– –	○	◍	◍	●
a –	○	○	◍	◍
– *b*	○	◍	○	◍
a b	○	○	○	○

3. *HELMINTHOSPORIUM* TYPE RACE-SPECIFIC RESISTANCE

Gene for virulence in the pathogen matches gene for susceptibility in the host plant.

Pathogen virulence genotype	Host susceptibility genotype – –	*A* –	– *B*	*A B*
– –	●	●	●	●
a –	●	○	●	○
– *b*	●	●	○	○
a b	●	○	○	○

4. *MELAMPSORA* TYPE RACE-SPECIFIC RESISTANCE

Gene for virulence in the pathogen matches gene for resistance in the host plant.

Pathogen virulence genotype	Host resistance genotype – –	*A* –	– *B*	*A B*
– –	○	●	●	●
a –	○	○	●	●
– *b*	○	●	○	●
a b	○	○	○	○

A and *B* are genes in the host plant, *a* and *b* are genes in the pathogen. They work quantitatively in nonspecific, qualitatively in race-specific relationships.

● = resistance ◍ = low resistance
◍ = moderate resistance ○ = susceptibility

genetically as mirror images of each other (Table 1).

A basic consequence of true gene-for-gene interrelations is that genes for resistance of the host are identified only by their matching genes for attack of the pathogen and *vice versa*. This implies that a complete understanding of the genic content and arrangements in a population has to proceed by using individual genes from the other side as analytical detectors. Such an approach is essential for proper planning of race-specific resistance breeding. It is not that very important in connection with nonspecific resistance. This is not to devalue nonspecific resistance but rather its strategic complexity. Here the problems are mainly evaluation, accumulation, and transfer.

Shifting Strategies of Oat Stem Rust

In order to prove the significance and merits of gene-for-gene-based inventories, it may be suitable to use simple demonstrations. Even oligogenic systems as in race-specific resistance soon become complicated owing to the rapidly increasing number of recombinants with increasing number

Table 2. Old conventional race key for oat stem rust (*Puccinia graminis* Pers. f.sp. *avenae* E. et H.)

Differential variety	Res. gene	Reaction of differential variety to oat stem rust race no.: 1	1A	2 5	2A 5A	3	3A	4	4A	6 13	6A 13A	7 12	7A 12A	8 9 10	8A 9A 10A	11	11A
White Tartar	*D (Pg1)*	●	●	●	●	○	○	○	○	○	○	○	○	●	●	●	●
Richland	*A (Pg2)*	●	●	●	●	●	●	○	○	○	○	●	●	○	○	○	○
Sevenothree	*E (Pg3)*	●	●	○	○	●	●	●	●	○	○	○	○	○	○	●	●
Rodney	*B (Pg4)*	●	○	●	○	●	○	●	○	●	○	●	○	●	○	●	○

● = resistant ○ = mesothetic or susceptible reaction

of genes involved. The old conventional race key for oat stem rust happens to be appropriate as a basis for such a demonstration (Table 2). The only four differentials carry each one of the dominant resistance genes *A (Pg2)*, *B (Pg4)*, *D (Pg1)*, and *E (Pg3)* matched by the recessive and independently segregating virulence genes *a* (v_2), *b* (v_4), *d* (v_1), and *e* (v_3), respectively. Among altogether 26 races identified, ten (5, 5A, 9, 9A, 10, 10A, 12, 12A, 13, 13A) show mesothetic reaction on one of the differentials. This mixed and temperature-unstable syndrome is apparently due to a superimposed plasmic effect (Green and McKenzie 1967), observed in North America but not in Scandinavia. If read as susceptibility, the key will be limited to 16 races, all some time registered in both regions.

Table 3 will demonstrate that all four resistance genes are needed to control all the races observed. Those being recommended as most appropriate races for checking gene transfers in the host are only stopped by one of the four resistance genes involved. They carry single avirulent genes and are thus the analytical detectors in a host population survey. Race 6A is virulent on all four host genotypes and additional gene(s) for resistance must be searched for (Mac Key and Mattsson 1972). As to the four genes, the table makes the situation completely clear to the breeder.

The order the races are arranged in Table 3 and its middle part helps to show that the 16 observed, clearly different races represent all possible recombinations from 4 genes (or homozygous gene pairs). The gene-for-gene interrelation is verified, and the race numbers are deciphered (Mac Key 1974). The interrelation works exactly like opening a door locked by a varying number of locks (resistance genes), each one to be opened with a precise key

Table 3. Relation between the Scandinavian oat stem rust races identified by and the four genes for resistance available in the old conventional race key tester set (Mac Key 1974)

Gene for resistance	Pattern of reaction of oat stem rust race number: 1	11	1A	3	2	11A	4	8	3A	2A	7	4A	8A	6	7A	6A
A (Pg2)...	●	○	●	●	●	○	○	○	●	●	●	○	○	○	●	○
B (Pg4)...	●	●	○	●	●	○	●	●	○	○	●	○	○	●	○	○
D (Pg1)...	●	●	●	○	●	●	○	●	○	●	○	○	●	○	○	○
E (Pg3)...	●	●	●	●	○	●	●	○	●	○	○	●	○	○	○	○

Gene for virulence	Combination of virulence genes in oat stem rust race number: 1	11	1A	3	2	11A	4	8	3A	2A	7	4A	8A	6	7A	6A
a.....	–	*a*	–	–	–	*a*	*a*	*a*	–	–	–	*a*	*a*	*a*	–	*a*
b.....	–	–	*b*	–	–	*b*	–	–	*b*	*b*	–	*b*	*b*	–	*b*	*b*
d.....	–	–	–	*d*	–	–	*d*	–	*d*	–	*d*	*d*	–	*d*	*d*	*d*
e.....	–	–	–	–	*e*	–	–	*e*	–	*e*	*e*	–	*e*	*e*	*e*	*e*

Necessary genes to cover resistance against the 16 races observed in Scandinavia: *A + B + D + E + ?*

Most appropriate race for checking gene transfer: 7A 6 8A 4A 6A

● = resistant ○ = susceptible reaction

Table 4. Race spectra of oat stem rust in Sweden and the United States in 1956-59 (Mac Key 1977)

Year	n	Relative prevalence (%) of oat stem rust race number/pathotype															
		1 —	11 *a—*	1A *-b–*	3 *–d-*	2, 5 *—e*	11A *ab–*	4 *a-d-*	8, 10 *a--e*	3A *-bd-*	2A, 5A *-b-e*	7, 12 *--de*	4A *abd-*	8A, 10A *ab-e*	6, 13 *a-de*	7A, 12A *-bde*	6A, 13A *abde*
Sweden																	
1956	32	–	–	–	31	–	–	28	–	6	–	13	–	–	13	3	6
1957	61	–	–	–	7	–	–	7	–	3	–	11	3	–	13	25	31
1958	96	–	–	–	24	–	–	14	–	5	–	20	3	–	15	6	14
1959	133	–	–	–	12	–	–	9	–	7	–	16	5	–	10	21	20
1956-59	322	–	–	–	16	–	–	12	–	6	–	16	4	–	12	15	19
USA																	
1956	476	–	–	–	–	16	–	–	15	–	–	66	–	–	1	2	–
1957	522	–	–	–	–	12	–	–	21	–	0	59	–	–	2	6	0
1958	286	–	–	–	–	14	–	–	26	–	–	54	–	–	1	5	–
1959	230	–	–	–	–	7	–	–	11	–	–	59	1	–	10	10	2
1956-59	1514	–	–	–	–	13	–		19	–	0	60	0	–	3	5	0

(virulence gene). First then entrance through the door is made possible (susceptibility reaction).

Deciphered race spectra for Sweden and the United States (1956-59 is the only period for Swedish inventories), arranged not by conventional numbers but by genic constitution and degree of complexity, are given in Table 4. These two examples represent total absence and presence, respectively, of host selection pressure. No oat varieties with race-specific resistance have ever been grown commercially in Scandinavia, but they have been consciously produced in North America since the early 1940's (Mac Key 1974; Stewart and Roberts 1970).

A comparison between the two sets of race spectra for the very same pathogen but under different situations offers at least two items of important information. One deals with the autonomy of each virulence gene involved. All races vital enough to be recorded over the four years carry gene *d* in Sweden but gene *e* in the United States. Obviously, the virulence gene always present must have some influence on general fitness, which probably works by supporting the others not occurring alone. The gene *d* must be important in one situation, gene *e* in another.

The other observation to be made deals with the concept of stabilizing selection thought to be generally valid for obligate parasites (Van der Plank 1968). In the sense of this concept behind most of our modern methodological proposals in breeding for resistance, both gene *d* in Sweden and gene *e* in the United States should not be as prevalent. They are unnecessary for the ability of the pathogen to attack the concerned host population and should thus show a selective handicap. The corresponding resistance gene *E* has for clear reasons never been used in a North American oat breeding programme. In Sweden no race-specific host selection pressure whatever has been exerted.

This is, however, not enough. The prevalent races in Sweden are, contrary to expectation, complex. In North America, resistance gene *A* and *D* were separately introduced already in the late 1940's, and they were combined together or with *B* in the middle of the 1950's (Stewart and Roberts 1970). In fact, these two latter events occurred during the years selected for Table 4. With this information at hand, it becomes obvious that the oat stem rust population in the United States does not show more complex races than needed under the type of host selection pressure introduced. Thus, one population, the North American, follows the principle of stabilizing selection and the other, the Swedish or Scandinavian, accumulates apparently unnecessary genes.

The different qualifications for reproduction in Sweden and the United States probably account for the seeming contradiction (Mac Key 1974). In Sweden, the complete cycle with an alternation to barberry is necessary for safe overwintering of the stem rust. Such a passage from one host to the other calls for a storage capacity for virulence genes. In the United States and the whole North America, the more important biotypes rely on constant asexual reproduction at the uredinial stage owing to the efficiency of the so-called *Puccinia* Path (Frey et al. 1977).

In Israel (Wahl et al. 1964) and Australia (Luig and Baker 1973), cultivated oats are infected by unnecessarily complex races of oat stem rust despite the absence of barberry. Nevertheless, a host alternation is still necessary and other grasses serve this function during seasons when the annual oat plant does not grow. In Israel, oat stem rust was found on 73 different species belonging to 35 different genera of the grass family (Wahl et al. 1964).

Table 5. Race spectra of wheat leaf rust in Sweden, Portugal, and the United States in 1956-59 (deciphered from Mac Key et al. 1963; Freitas 1959, 1961, 1966; Johnston 1957, 1958, 1959, 1960)

Year	n	Relative prevalence (%) of wheat leaf rust pathotype															
		—	*a—*	*-b-*	*--c-*	*---d*	*ab--*	*a-c-*	*a--d*	*-bc-*	*-b-d*	*--cd*	*abc-*	*ab-d*	*a-cd*	*-bcd*	*abcd*
Sweden																	
1956	189	–	–	–	–	–	–	–	–	–	–	–	–	39	–	–	61
1957	84	–	–	–	–	–	–	–	–	–	–	–	–	21	–	47	32
1958	158	4	–	–	–	7	–	–	–	–	51	3	–	4	–	30	1
1959	154	2	–	–	–	4	–	–	–	–	44	5	–	14	–	31	–
1956-59	585	2	–	–	–	3	–	–	–	–	25	2	–	20	–	23	25
Portugal																	
1956	65	43	–	11	–	–	–	–	–	43	–	3	–	–	–	–	–
1957	70	43	–	–	–	–	–	–	–	57	–	–	–	–	–	–	–
1958	119	49	–	27	–	–	–	–	–	23	–	1	–	–	–	–	–
1959	231	56	–	10	7	–	–	–	–	22	1	–	–	–	–	4	–
1956-59	485	50	–	13	3	–	–	–	–	30	1	1	–	–	–	2	–
USA																	
1956	674	5	1	–	24	–	6	28	–	–	–	1	26	0	8	–	1
1957	997	2	–	–	42	–	3	26	–	0	–	1	16	2	7	–	1
1958	935	11	–	–	32	2	7	16	–	–	–	7	12	4	8	–	1
1959	1447	1	–	–	53	0	5	10	–	0	–	11	11	3	3	–	4
1956-59	4053	5	0	–	40	0	5	19	–	0	–	6	15	2	6	–	2

Symbol key: *a* = virulence gene matching *Lr-1* in 'Malakof'; *b* = virulence gene matching *Lr-2a* in 'Webster'; *c* = virulence gene matching *Lr-3* in 'Mediterranean'/'Democrat'; *d* = virulence gene matching *Lr-11* in 'Hussar'

Shifting Strategies of Wheat Leaf Rust

The different behaviour of different oat stem rust populations is far from unique. Wheat leaf rust may be chosen as a second example (Table 5).

For consistency, the demonstration is also this time limited to four gene pairs, which again is sufficient for the purpose. Sweden, and for that matter the whole eastern part of Europe, show unnecessarily complex races. In the recorded period there was no host selection pressure in Scandinavia, nor in Portugal. But in contrast, stabilizing selection is apparently operating inside the Portuguese or more appropriately called Northwest-African-Southwest-European population, causing simple races to prevail. The American leaf rust population shows the same tendency but blurred by the fact that host selection pressure here forces the rust to adapt accordingly. Virulence gene matching *Lr-11* (*d* in Table 5) appears to have a supporting function in the Swedish, gene matching *Lr-2a* (*b*) in the Portuguese, and virulence gene matching *Lr-3* (*c*) in the American population. This tendency is, however, not as sharp as found for the oat stem rust populations. The zero race does appear, in Portugal even as the leading race.

Gene Erosion and Gene Preservation in Rust Populations

The Australian approach to breeding against wheat stem rusts adds interesting observations. This programme definitely favours the idea that a pathogen is not always able to utilize its full evolutionary capacity. The stem rust in Australia is totally dependent on the uredinial stage for survival through the year, and asexual recombination is insufficient for more profound changes of once-established genotypes. The assortative effect of introducing varieties with complex race-specific resistance, therefore, implies a considerable erosion of background genes. In spite of mutation rates calculated by Parlevliet and Zadoks (1977) to be of the order of 1,000 mutants/locus/hectare/day, the Australian experience indicates an apparent restriction in evolutionary flexibility of the rust. New genes for virulence will have difficulties in becoming properly adjusted. For the same reason suddenly unnecessary and now disadvantageous virulence genes cannot be immediately eliminated. The more resistance genes it has to overcome, the less aggressive the rust becomes (Watson 1977).

There must exist a considerably greater gap between mutation rate and mutation establishment than is under-

stood. It appears likely that newly developed genes for race specificity, both on the parasite and host side, mostly require adjustment in their genetic background. As so often a second step is necessary. A macromutation must be reconciled by a subsequent microevolution. Luig (1979) found the pathogenic mutants in rust having small uredospores and/or delayed development. Watson and Luig (1968) found that rust can change in a stepwise fashion from avirulence to virulence apart from what can be reached through heterozygosity. New mutants for race-specific resistance in the host are generally recessive and often inferior in general fitness. Old resistance genes occurring in nature are generally dominant, indicating an adaptation process (Fisher 1931; Mac Key 1974; Jörgensen 1976).

All these observations point to evolutionary complications in enabling an accumulation process for race-specific genes, i.e. to evolve a preparedness. Under conditions of pronounced host selection pressure eroding the gene pool at absence of sexual recombination, this adaptation process is hampered. Virulence genes are likely to remain generally unfit and preserved only when their function makes it necessary.

Whenever mutation establishment is a problem, the organism should gain advantage by developing a storage capacity rather than rely on prompt mutation. Because of their dikaryotic constitution and the general recessiveness of virulence genes, relaxed rust populations should have the capacity to take such an advantage. Returning to our examples and expanding the number of genes involved, the realization of such a trend can very clearly be verified (Table 6; Mac Key 1974).

By using as traps the 13 genes for race-specific resistance known up to 1974, 11 of the matching virulence genes could be found in the Scandinavian oat stem rust population. Remember that no host selection pressure has been exerted. The number of virulence genes found is, in fact, very much the same as in North America where there has been conscious breeding for resistance since the 1940's. The high incidence in North America of virulence genes matching the resistance of *E* (*Pg3*), *f* (*pg8*), *h* (*pg9*), and *m* (*pg13*) but absence of the matching resistance genes in the host population (Martens et al. 1970) supports the picture of preparedness rather than mutation every time breeders introduce a new resistance gene. Remember that genes for virulence can only be detected by matching genes for resistance.

The same situation can be proved for wheat leaf rust where the East European races often hold 4-5 and even more unnecessary genes for virulence (Stewart et al. 1967; Ralski 1972; Bosković and Browder 1976; Lesovoj et al. 1976).

Table 6. Genes for race-specific resistance against oat stem rust known up to 1974 (Mac Key 1974)

Source	Carries gene
'Lanark', 'White Russian', 'Minrus'	*Pg1* (*D*)[a]
'Exeter', 'Richland', 'Ajax'	*Pg2* (*A*)[a]
'Canuch', 'Jostrain', 'Roxton'	*Pg3* (*E*)[a]
'Rodney', 'Torch'	*Pg4* (*B*)[a]
CI 2710, RL 524.1, CI 4023	*pg8* (*f*)[a]
CI 5844	*pg9* (*h*)[a]
CI 3034	*pg11*[a]
'Kyto'	*pg12*
Avena sterilis CW490-2	*pg13*(*m*)
'Garry', 'Hajira'	(*G*)[a]
CI 1575	(*G*)
'Milford', 'Winter Turf'	(*N*)[a]
'Rosen' Mutant (*pg9* + ?)	[a]
'Saia', 2x (*Avena strigosa*)	[a]

[a] Matching gene for virulence already found in the Scandinavian population of oat stem rust

Ploidy Level Governs Strategy

If restricted recombination is one cause for impaired gene accumulation, low genetic storage capacity as in true ha-

Table 7. Race spectra of wheat powdery mildew in Sweden in 1960-62 (deciphered from Leijerstam 1962, 1965)

Year	n	Relative prevalence (%) of wheat powdery mildew pathotype:																	
		----	*a---*	*-b--*	*--c-*	*---d*	*ab--*	*a-c-*	*a--d*	*-bc-*	*-b-d*	*--cd*	*abc-*	*ab-d*	*a-cd*	*-bcd*	*abcd*		
Sweden:																			
1960	161	42	23	1	6	4	2	8	5	–	–	3	1	1	4	–	–		
1961	440	16	10	3	22	3	2	26	3	2	0	7	0	0	6	0	–		
1962	358	11	14	3	20	2	3	29	2	2	–	6	2	–	5	–	1		
1960-62	959	19	14	3	19	3	2	24	3	1	0	6	1	0	5	0	0		

Symbol key: *a* = virulence gene matching *Pm1* (*Ml-t*) in 'Norka'; *b* = virulence gene matching *Pm2* (*Ml-u*) in 'Ulka'; *c* = virulence gene matching *Pm3a* (*Ml-c*) in 'Chul'; *d* = virulence gene matching *pm5* (*ml-h*) in 'Hope Selection'

Table 8. Racial composition in the Swedish wheat powdery mildew population for four virulence genes sampled through conidia and perithecia in 1960-61 (calculated from Leijerstam 1962)

Type of sampling	n	Relative prevalence (%) of virulence gene			
		Pm1(Ml-t)	*Pm2(Ml-u)*	*Pm3a(Ml-c)*	*pm5(ml-h)*
Conidia	388	38	4	44	14
Perithecia	341	37	8	42	13

Heterogeneity between types of sampling: $\chi^2 = 4.92$, $P = 0.18$

Type of sampling		Relative prevalence (%) of races carrying				
		0	1	2	3	4
		gene(s) for virulence out of the four				
Conidia	347	29	35	30	6	–
Perithecia	254	15	39	39	7	–

Heterogeneity between types of sampling: $\chi^2 = 17.64$, $P < 0.001$

ploidy should be another. Organisms with such a constitution will, however, have a more direct phenotypic exposure of mutational events increasing their success in limiting their strategy to an immediate and provisional flexibility. Wheat powdery mildew can be taken to demonstrate this situation. For consistency, again a Swedish population without race-specific selection pressure and limitation to four genes will be used as example (Table 7). The simple races prevail this time.

Another way to prove occurrence of impaired general fitness in organisms like mildew with degree of racial complexity is to compare sampling from conidia and perithecia. The former vegetatively propagated should hold vital, i.e. simple races only, the latter a wider array of recombinants. Table 8 shows that this is also the trend.

Discussion

In his important book on 'Disease Resistance in Plants' in 1968, Van der Plank added an epidemiological dimension to the genetic concept of pathogenicity. He also elaborated on the gene-for-gene model in connection with race-specificity. Mode (1958) and Person (1959, 1966) pointed earlier to the fact that the selective value of genes for resistance and their matching genes for virulence counterbalance each other towards a state of stable equilibrium, a balanced polymorphism. Van der Plank developed this model further for obligate parasites by suggesting that here unnecessary genes for virulence are somehow disadvantageous and thus steadily selected against. Leonard (1969) made an extention by assuming, Harlan (1976) by stating, that genes for resistance are also selected against in environments where they do not protect against the pathogen.

During the last decade, this principle of stabilizing selection has dominated the concept of race-specific parasitism. With such a system, the host:parasite interaction will by itself lead to a balanced *modus vivendi*, essential for the survival not only of the host but also of its dependent parasite. A diversification rather than a gene accumulation should favour such an outcome thought typical for natural systems but broken down by the earlier gene accumulation approach in breeding for race-specific resistance. The high mutant rate in the pathogen population should always guarantee enough complex races to develop and be able to overcome any resistance barrier. The resources of genes for resistance should rather be used on a single-gene basis to increase the number of components in a diversified system, thus offering less chance for every spore from correspondingly simple races to land on a compatible plant.

This model, based on stabilizing selection, was supported by practical experiments even on large scales (Frey et al. 1977) and appeared straight forward enough to call for mathematical elaborations (Groth and Person 1977; Leonard 1977; Marshall and Pryor 1978). The race-specific genes have more and more been looked upon as if they act in a nonpleiotropic and completely autonomous manner. An utmost important series of discoveries has resulted into too rigid a concept.

By an almost self-evident deciphering of conventional race numerations, old inventories have above been used to demonstrate the danger of oversimplifications. The examples chosen and for convenience summarized in Table 9 should be sufficient to prove that a strategy based on Van der Plank's concept of stabilizing selection in not the only outcome in connection with obligate parasitism. Different species in the same ecological niche as well as different populations of the same *forma specialis* in different situa-

Table 9. Some pathogen populations with different ability to preserve unnecessary genes for virulence

Pathogen	Country	Years of inventory	Host pressure, no. of genes	n	Relative prevalence (%) of races carrying				
					0	1	2	3	4
					virulence genes of four				
Pathogen population characteristic: Stabilizing Selection									
Wheat leaf rust	Portugal	1956-59	no	485	50	16	32	2	–
Wheat powdery mildew	Sweden	1960-62	no	959	19	39	36	6	0
Wheat leaf rust	USA	1956-59	3	4053	5	40	30	23	2
Oat stem rust	USA	1956-59	3	1514	–	13	79	8	0
Pathogen population characteristic: Gene Accumulation									
Oat stem rust	Sweden	1956-59	no	322	–	16	34	31	19
Wheat leaf rust	Sweden	1956-59	no	585	2	3	27	43	25

tions may differ as to ability to accumulate and preserve temporarily unnecessary genes for virulence. It even appears as if a strategy more based on preparedness rather than an immediate and provisional flexibility is preferred whenever possible. The gap between mutation rate and ability of a raw mutant to overcome general handicaps is too wide. Exploration of evolutionary gains already made by improving genetic storage capacity implies apparently a less risky alternative for an obligate parasite.

This evolutionary trend is, however, not always available. True haploidy is too primitive a genetic system to allow stepwise adaptability through heterozygosity and a hidden genetic storage capacity of recessive virulence genes. Pathogens of this constitution have to rely on flexibility enhanced by the immediate phenotypic expression of every mutational event in combination with an enormous number of individuals per population and a rapid shift between generations.

An exclusion from sexual reproduction as when cereal or grass rusts do not have access to its alternative host and thus have to survive permanently in the uredinial stage implies another restriction. It should be observed that such a restriction may very well have developed by selective advantage, since it allows opportunistically mass production of efficient genotype(s). The parallelism with evolution from allogamy towards autogamy or vegetative propagation and finally monocultures in plant, i.e. host, domestication is obvious. The rust populations migrating up and down the *Puccinia* Path in North America are good examples of such a preferential evolution.

Merely vegetative reproduction implies, however, that genes cannot combine freely and that an isolating speciation process starts to work inside the pathogen population. Whenever the assortative effect of race-specific resistance is set in against such a population, an inevitable gene erosion will take larger proportions. Not randomly distributed, genes may easier be lost. With the approach taken above, it is just as important that background genes essential for adjusting general fitness will be submitted to the same fate as virulence genes. This loss of all kinds of genes will be greater and evolutionarily thus more devastating, the more the selective advantage for any biotype(s) is enhanced by a monoculture of the host and the more genes for resistance that are simultaneously accumulated into this monoculture. The effect must of course also depend on to what extent wild and weedy plants out of control of man are able to reproduce the same asexual stage of the pathogen.

The experience from the strategy laid up against the Australian wheat stem rust (Watson 1977) suggests that this pathogen has restricted possibilities to overcome such an artificially imposed, drastic impoverishment. The rely on genetic storage capacity has apparently developed too far even for such a flexible organism as a dikaryotic fungus to meet such an adverse situation. The early North American approach could simultaneously be used as a warning against handling too few genes for resistance at a time or add them stepwise with too long intervals offering the pathogen population recovery.

An inadequate gene distribution because of insufficient ability to recombine at permanent uredinial stage will hamper any adaptation process. Genes for virulence will, therefore, likely remain generally unfit and preserved only when their special function is necessary. This implies that the strategy of gene diversification must also work against an asexually propagated rust population. It should, however, at the same time be remembered that diversification of the host automatically implies diversification of the pathogen in a gene-for-gene-related system. A pathogen capable of adjusting genetic background for more premanent preservation of virulence genes may, therefore, in the long run be able to build up such complex races by which the gene diversification strategy may be endangered. The more

general use of multi-line varieties, the sooner might this collapse occur.

Bacteria and true haploid fungi should have easier to meet a situation of a sudden and drastic gene erosion. As argued above, they are more limited to work by flexibility rather than by preparedness. Gene diversification by using multi-line varieties, varietal mixtures, or regional gene deployment ought to be a more reliable approach than is gene accumulation. There are, however, hints (Wolfe 1972; Mac Key 1974) that certain combinations of virulence genes give negative synergism as to general fitness. The corresponding genes for resistance could thus be used at least to give an epidemic retardation, i.e. they will superficially but in a different way act like nonspecific resistance.

It has above been made an attempt to outline some general tendencies in the strategy of windborne phytopathogens. It appears, therefore, appropriate to end with a warning against any too far-reaching generalization. Different pathogens, and different populations belonging to one of them, may differ too much in racial construction and overall flexibility to be handled in a breeding programme against them in the same way. Each situation must be understood sufficiently well. It is also important to observe that the two fundamental approaches, diversification and multiple gene accumulation, are never to be used simultaneously with the same set of resistance genes within a natural epidemiological region for a parasite.

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