Race Diversity and Complexity in Selected Populations of Fungal Biotrophic Pathogens of Cereals

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We thank M. S. Wolfe, Swiss Federal Polytechnic Institute, Zürich, for his many valuable comments and suggestions on an early draft of the manuscript.

Accepted for publication 9 November 1994.

ABSTRACT

Andrivon, D., and de Vallavieille-Pope, C. 1995. Race diversity and complexity in selected populations of fungal biotrophic pathogens of cereals. Phytopathology 85:897-905.

The relationships between several variables (sample size, measures of race number and frequency, number of discriminant differential hosts) and mathematical estimates of race diversity and complexity were studied by means of linear correlation and principal component analysis in various populations of cereal biotrophic pathogens, using race survey data from the literature. No constant relationships between the variables studied were found in geographically distinct populations of several rust and powdery mildew pathogens of cereals, suggesting a large local effect of selection by resistance genes on race diversity and complexity in these populations. Diversity estimates based on race abundance, race dominance, or synthetic indexes were generally correlated, irrespective of the population considered; no clear correlation was found between estimates of diversity and complexity. The increase in virulence complexity in rust pathogens and stability of complexity at a low level in powdery mildew fungi as new genes are introduced in the host population are possibly related to the biology of the pathogens considered, and particularly the presence or absence of a sexual phase. The large incidence of the characteristics of the differential set used on recognized diversity and complexity patterns is shown and discussed.

The efficacy of strategies to control plant diseases depends largely on their ability to manage variability in local pathogen populations, and the need to adapt control measures to ever-changing strains prompted the setting-up of numerous surveys of races and fungicide sensitivity.

Several mathematical indexes are available to estimate phenotypic diversity (15,27). Using annual race survey data for *Puccinia graminis* Pers.:Pers. f. sp. *tritici* Eriks. & E. Henn. from 1918 to 1982 in the United States, Groth and Roelfs (15) showed that diversity in its broadest sense, as estimated by the Shannon index, was dependent on both richness (number of different races in the sample) and evenness (similarity of race frequencies). Furthermore, three diversity estimates (Shannon, Simpson, and Gleason indexes) were correlated with sample size, sample richness, and frequencies of the single or the three major races. Similar conclusions were obtained from the analysis of race surveys of *Puccinia recondita* Roberge ex Desmaz. f. sp. *tritici* (Eriks. & E. Henn.) Henderson in Canada over a 56-year period (21). However, it is not known whether these observations apply also to these two fungi outside North America or over shorter periods, or to other biotrophic fungal plant pathogens.

Besides diversity, virulence complexity is also of major concern for the understanding of patterns of race evolution. Observations of the changes in race structure of *Phytophthora infestans* (Mont.) de Bary and *P. g. tritici* led Vanderplank (57) to postulate the existence of what he called "stabilizing selection against unnecessary virulence genes," and most further debate on the validity of this hypothesis centered on virulence complexity data as related to fitness (41). Recently, a 5-yr survey of three French populations of the barley powdery mildew pathogen *Erysiphe graminis* DC f. sp. *hordei* Ém. Marchal revealed a pattern of changes in virulence complexity quite different from that observed in cereal rust fungi (1). While a stepwise increase of virulence number per race is usually observed in rust agents such as *P. r. tritici* or *Puccinia striiformis* Westend. as new resistance genes are sequentially introduced in the host crop (2,12,21,29,56,59), a dramatic modification of the predominant races, but with the mean virulence complexity remaining almost constant, was induced in French *E. g. hordei* populations by sequential changes in selection pressures (1). The replacement of simple races by other simple races with different virulence spectra explained this pattern of structural change, as well as the increase in race diversity observed during the first years of the survey and its subsequent decrease. Maximal diversity corresponded to the presence in almost equal amounts of "old" and "new" races. The data therefore suggested divergent evolutions of race complexity and diversity in the barley mildew fungus (1), but little information on diversity-complexity relationships in other pathosystems is available in the literature.

However, a better understanding of diversity and complexity evolution in plant pathogens is required to provide guidelines for resistance breeding programmes (e.g., to decide whether pyramiding resistance genes might or might not be successful), as well as for using with greatest benefit the possibilities of resistance gene recycling and of spatial diversification of resistance (12, 31,61). From a more fundamental standpoint, such data are also needed to compare and explain microevolutionary patterns and strategies in plant pathogens.

This paper compares data on diversity and complexity of pathogenic races in cereal rust and powdery mildew populations, using published surveys of *P. graminis, P. r. tritici, P. striiformis, P. coronata, P. hordei,* and *E. graminis* from Europe, North America, South Africa, and Israel. Earlier papers have compared diversity between populations of some cereal rust species (e.g., 5–8,10,18–20, 22,28,42–50,52,53,59) or between sexual and asexual populations of a species (14,32,51), while others documented changes in diversity in some rusts over many years (9,15,21,29,38). This paper extends these comparisons to a wider scope. Comparisons of race diversity and complexity between geographically separated populations of the same fungi were performed to assess the influence of local conditions on their race evolution. Comparisons were also made between different pathogens, in order to estimate the incidence of fungus identity on race patterns and their changes. The present study had three major goals: *(1)* to test the correlations

between several variables (sample size, measures of race number and frequency, number of discriminant differentials) and race diversity estimates (mathematical indexes) in populations of cereal biotrophic pathogens from different locations; *(2)* to evaluate the relationships between race complexity and diversity in some of those pathosystems; and *(3)* to determine whether changes in race diversity and complexity mostly depend on the taxonomic identity of the pathogen considered or on local variation for pathogen life cycle or resistance gene deployment.

MATERIALS AND METHODS

Selection of data. Analyses were based on published race survey data (selected populations and corresponding references listed in Table 1). Emphasis was given to cereal rust and powdery mildew fungi, reflecting the abundance of published reports available and the worldwide economic incidence of those pathogens.

Eleven populations (populations marked *, Table 1) of wheat, oat, and barley pathogens surveyed over a short- to medium-term period were selected to determine common features and peculiarities of changes in diversity and complexity in pathogens characterized by similar host range and biology (cereal biotrophs). To allow comparisons, data selection was guided by the following criteria: *(1)* data were available over at least five consecutive years for each population with sufficient sample size (at least 30 individuals), and *(2)* separation of the different subpopulations, each being characterized by a different history of resistance genes deployment or by biological differences (e.g., presence or absence of the sexual stage), was possible from the original data. The period considered was limited to a maximum of 15 yr for each population to keep the time scale similar. Each population was described using annual values of 10 race diversity and complexity variables (see infra) for each of the years considered. Whenever possible, geographically distinct populations of a single pathogen were used, in order to evaluate the influence of local factors on diversity and complexity changes and relationships. Rust populations were typically obtained from field samples of infected leaves, as were *E. g. tritici* samples, whereas *E. g. hordei* populations were collected directly from the air using a jet spore trap mounted on the roof of a car.

Simultaneous comparison of a greater number of populations was also performed on all yearly samples previously studied plus data from pathosystems for which only limited information (i.e., 1 yr–one location) was available (Table 1). This allowed the inclusion of sexual populations of fungi usually reproducing clonally such as *P. g. tritici* in the U.S., and of populations that have been surveyed over a limited period of time, e.g., *P. g. tritici* in China (17) or *P. hordei* in Israel (30). In this analysis, each population sample (i.e., each pathogen-year-location combination) was considered as a single unit. Overall, 101 units from 21 populations were retained for the analysis.

Variables. For each pathogen-year-location combination, four groups of variables were recorded or calculated:

(1) Sample size (N_i), i.e., the number of isolates tested. Sample size varied considerably between pathosystems and surveys, the large number of isolates (> 2,000) included in the *P. g. tritici* surveys in the U.S. in most years contrasting with sample sizes of 30–300 isolates in the remaining populations. Variation for N_i was much greater than for any other variable; therefore, a transformation to natural logarithms ($\ln N_i$) was done before statistical analysis.

(2) Diversity variables, consisting of the number of distinct

TABLE 1. Race diversity and complexity in populations of biotrophic fungal pathogens of cereals, with average annual values of the variables[a] calculated from annual data concerning years stated

Pathogen/location[b]	Years	Refs.	$\ln N_i$	$\ln N_p$	R_1	R_3	H_G	H_{GR}	H_S	H_{SR}	C_i	C_p	NDD	NDDR
Puccinia spp.														
1 *P. recondita tritici*/RSA*[c]	1983–1988	42–44	5.21	2.00	0.44	0.78	1.51	0.05	1.51	0.30	7.26	6.23	14.8	0.99
2 *P. recondita tritici*/ Manitoba-Sask., CAN	1986–1989	17–19, 33	5.44	2.91	0.28	0.67	3.31	0.08	2.00	0.37	4.99	4.88	12.5	0.77
3 *P. recondita tritici*/D	1986–1988	24	4.63	4.14	0.11	0.21	15.36	0.63	3.79	0.83	9.61	9.62	18.3	0.66
4 *P. striiformis*/F*	1987–1990	57[d]	4.01	2.10	0.43	0.83	1.87	0.16	1.51	0.39	7.03	7.64	11.7	0.80
5 *P. striiformis*/BU*	1981–1986	35–36	4.94	1.89	0.32	0.75	1.15	0.04	1.64	0.33	5.93	6.11	11.2	0.73
6 *P. graminis tritici*/USA*	1973–1988	38, 45–50, 52–53	6.62	2.00	0.69	0.86	1.15	0.01	1.08	0.16	7.47	6.41	9.4	0.77
7 *P. graminis tritici*/USA[e]	1984	46	4.44	2.48	0.67	0.88	2.48	0.12	1.27	0.27	4.41	5.91	11.0	0.69
8 *P. graminis tritici*/USA[e]	1986	48	5.10	1.95	0.57	0.91	1.78	0.04	1.23	0.24	2.10	3.14	5.0	0.42
9 *P. graminis tritici*/CHI	1987	16	4.84	2.08	0.33	0.50	1.44	0.06	1.60	0.34	7.93	7.50	4.0	0.33
10 *P. graminis avenae*/ Manitoba, CAN	1980–1982	32	4.91	1.56	0.75	0.95	0.96	0.04	0.78	0.16	4.88	4.56	5.7	0.57
11 *P. graminis avenae*/ Ontario, CAN[e]	1980–1982	32	4.68	1.95	0.37	0.79	2.59	0.06	1.50	0.32	5.60	5.39	4.0	0.40
12 *P. coronata*/Manitoba-Sask., CAN*	1984–1989	5–8, 10–11	5.16	3.92	0.16	0.37	9.44	0.29	3.24	0.62	2.00	2.84	12.5	0.75
13 *P. coronata*/Ontario-Quebec, CAN*	1984–1989	5–8, 10–11	4.38	3.23	0.24	0.52	5.53	0.33	2.63	0.60	2.00	2.70	12.3	0.75
14 *P. hordei*/ISR	1983–1985	30	4.59	3.56	0.23	0.44	7.46	0.35	2.93	0.64	6.49	5.61	8.0	0.89
Erysiphe graminis														
15 *E. g. tritici*/PL*	1973–1981	25	3.63	2.52	0.25	0.52	3.17	0.33	2.20	0.60	2.87	2.80	6.5	0.94
16 *E. g. tritici*/YU*	1978–1987	22–23	5.50	2.35	0.50	0.81	1.91	0.04	1.51	0.27	5.18	4.36	5.4	0.77
17 *E. g. tritici*/Ontario, CAN	1985–1987	34	4.76	3.36	0.17	0.44	5.89	0.24	2.87	0.60	2.32	2.55	7.7	0.96
18 *E. g. tritici*/New York, USA	1984	39	4.75	3.29	0.27	0.47	5.47	0.23	1.21	0.56	1.18	2.19	7.0	0.78
19 *E. g. hordei*/Northern F*	1986–1990	1	3.62	2.90	0.18	0.40	5.32	0.49	2.67	0.74	4.07	4.36	7.2	0.61
20 *E. g. hordei*/Paris area, F*	1986–1990	1	3.84	3.00	0.21	0.42	5.02	0.43	2.67	0.69	4.37	4.37	7.4	0.63
21 *E. g. hordei*/Eastern F*	1986–1990	1	3.78	2.77	0.24	0.53	4.12	0.39	2.36	0.64	4.44	4.48	6.4	0.54

[a] Codes for variables: $\ln N_i$ = natural logarithm of isolate number; $\ln N_p$ = natural logarithm of pathotype number; R_1 = frequency of the most frequent pathotype; R_3 = cumulated frequencies of the 3 most frequent pathotypes; H_G = Gleason index of diversity; H_{GR} = relative Gleason index of diversity; H_S = Shannon index of diversity; H_{SR} = relative Shannon index of diversity; C_i = average isolate virulence complexity; C_p = average pathotype virulence complexity; NDD = number of discriminant differentials; NDDR = relative number of discriminant differentials.

[b] Numbers preceding each population are given for referencing in the text. Regional locations are given only when local populations could be separated within national data. BU = Bulgaria; CAN = Canada; CHI = People's Republic of China; D = Federal Republic of Germany; F = France; ISR = Israel; PL = Poland; RSA = Republic of South Africa; USA = United States of America; YU = Federal Republic of Yugoslavia.

[c] Asterisk (*) denotes populations included in both correlation and principal component analyses.

[d] Data for 1989 and 1990, de Vallavieille-Pope et al (*unpublished*).

[e] Sexual populations.

races in the sample (N_p) as an estimate of richness, and of the frequency of the single (R_1) or three (R_3) dominant races as estimates of evenness (15). When three or more races were found at equal frequencies for the second place, only two were considered for the calculation of R_3. Similarly, if two or more races ranked third with equal frequency, only one was retained for the calculation of R_3. If fewer than three races were detected in a sample, R_3 was set at 1. Because of large variations in race number among populations, N_p was also ln-transformed.

Estimates of phenotypic diversity were obtained from both the Gleason and Shannon indexes, calculated respectively as

$$H_G = \frac{N_p - 1}{\ln N_i} \text{ and } H_S = -\Sigma_j [p_j \ln(p_j)], j = 1 \ldots N_p,$$

where N_i and N_p are as described above and p_j is the frequency of the jth race in the sample (15). Groth and Roelfs (15) showed that H_G is especially sensitive to the richness aspect of diversity while H_S accounts for both richness and evenness.

Since H_G and H_S are often dependent on sample size (15,21,27), the large differences in N_i among the 101 population samples might have biased the estimates of diversity provided by these indexes. Therefore, relative indexes were also calculated as

$$H_{GR} = \frac{H_G}{H_{Gmax}} = \frac{N_p - 1}{N_i - 1} \text{ and } H_{SR} = \frac{H_S}{H_{Smax}} = \frac{H_S}{\ln N_i}.$$

H_{Gmax} and H_{Smax}, the maximum theoretical value of H_G and H_S in a sample of N_i individuals, are reached for $N_i = N_p$, and equal $(N_i - 1)/\ln N_i$ and $\ln N_i$, respectively.

(3) Complexity variables. Mean race complexity was estimated as both

$$C_i = \Sigma_j (p_j \cdot v_j) \text{ and } C_p = \frac{1}{N_p} \Sigma_j v_j, j = 1 \ldots N_p,$$

with N_p and p_j as described above and v_j representing the number of virulences carried by the jth race (1). C_j, therefore, represents the mean number of virulences per isolate, while C_p is the mean number of virulences per race.

(4) Differential set variables were included in the analysis because the total number of genes available to research groups for race analysis (NDT) was highly variable from one pathogen to the next, and between groups studying the same pathogen. Furthermore, the number of discriminant differentials (NDD) usually varied between populations screened with the same host sets. NDD was assessed as the number of differential lines or cultivars in the set for which the pathogen population was polymorphic. NDD was therefore 0 if only one race was found, and 1 if only two races differing by a single virulence gene were recorded in a sample. NDD being obviously dependent, among other factors, on NDT, and since NDT varied noticeably between pathogens and research groups, a relative index NDDR (= NDD/NDT) was built. A low value of NDDR may reflect either the inadequacy of the set used to describe pathogenic variation within a given population, or a low race diversity in this population. NDD and NDDR were both used for the joint analysis of the 101 population samples; they were not used for the separate analysis of the 11 pathosystems over time, since they varied little among yearly samples in these populations.

Statistical methods. Descriptive statistical methods were used to estimate relationships between either variables or individuals (population units). Linear correlation coefficients between the variables chosen were calculated. Correlation was used primarily to investigate the extent of simultaneous variation between diversity or complexity variables, either in time for single pathosystems or among pathosystems. Furthermore, principal component analysis (PCA) was performed on all 101 population samples. PCA is a descriptive, multivariate method intended to illustrate relationships or relatedness between individuals described by a set of variables. An extensive description of the method and its conditions of use is given by Lebart et al (26).

RESULTS

Analysis of single-pathosystem data. Linear correlation coefficients between pairs of variables are listed in Table 2 for the 11 pathosystems investigated. No significant correlation ($P > 0.05$) was usually found between sample size and the remaining variables. In particular, no correlations were apparent between $\ln N_i$ and virulence complexity parameters, except in one of the three French populations of *E. g. hordei* (population 20, Paris area, $P = 0.048$ and $P = 0.025$ for C_i and C_p, respectively) and in the South African *P. r. tritici* population (population 1, $P = 0.021$). Correlations between sample size and diversity estimates were also rare. A positive correlation between $\ln N_i$ and H_S was found only in population 5 (*P. striiformis*-Bulgaria, $P = 0.013$), while $\ln N_i$ and H_G were negatively correlated in population 1 ($P = 0.027$) and positively correlated in populations 5 ($P = 0.017$) and 12 (*Puccinia coronata* Corda f. sp. *avenae* Eriks-Manitoba, $P = 0.026$). $\ln N_i$ was significantly, positively correlated with sample richness ($\ln N_p$) in populations 4 (*P. striiformis*-France, $P = 0.016$), 5 ($P = 0.005$), 12 ($P = 0.003$), and 19 (*E. g. hordei*-northern France, $P = 0.039$), and with the frequency of the dominant race (R_1) only in population 16 (*Erysiphe graminis* DC f. sp. *tritici* Êm. Marchal-Yugoslavia, $P = 0.035$).

The richness variable ($\ln N_p$) was positively correlated ($P < 0.05$) with H_G in all populations but one (population 1), in accordance with the description of H_G essentially as a measure of richness (15). In five of the 11 cases studied, $\ln N_p$ was correlated with H_S. Race dominance, as estimated by R_1, was negatively correlated with H_S in all cases, except the two *P. coronata* and two *E. g. hordei* populations (populations 12, 13, 19, and 20). Correlations with H_S were equally frequent (seven out of 11 cases) with R_1 or R_3 as a measure of dominance, but did not always involve the same populations (Table 2). R_1 and R_3 were rarely significantly correlated with H_G (two and four cases, respectively); the same occurred when considering R_1 / R_3 and $\ln N_p$ (two and three cases, respectively), suggesting that richness and evenness of race frequencies were largely independent components of diversity in the majority of the populations studied.

The correlations between the two diversity indexes seemed to depend to a large extent on local characteristics, as shown by the data for *P. striiformis*, *P. coronata*, *E. g. tritici*, and *E. g. hordei* for which several locations were available. In all four pathogens, the correlation between H_G and H_S never reached significance at the 0.05 level in geographically distinct populations available for the comparison (Table 2).

No consistent trend was apparent concerning the relationships between diversity and complexity estimates. $\ln N_p$ and C_p were correlated positively only in the French *P. striiformis* population (population 4, $P = 0.020$), while a negative correlation between these two variables was found for populations 5 ($P = 0.041$), 6 (*P. g. tritici*-USA, $P = 0.003$) and 21 (*E. g. hordei*-eastern France, $P = 0.003$). An identical pattern was observed with H_G and C_p. $\ln N_p$ and C_i were correlated positively only in population 4 ($P = 0.016$), and negatively in populations 6 ($P = 0.003$) and 16 ($P = 0.045$). Negative correlations between H_G and C_i were found in populations 1 ($P = 0.017$), 6 ($P < 0.001$), and 16 ($P = 0.046$). R_1 was correlated with C_i and C_p negatively only in population 4 ($P < 0.01$) and positively in populations 6 and 16 ($P < 0.05$). R_3 was significantly correlated with C_i in only two of the 11 populations (populations 6, $P < 0.001$ and 16, $P = 0.040$; negative correlations), and with C_p in only one (population 6, $P = 0.039$, positive correlation).

Multivariate analysis of the 21 selected pathogen populations. Coefficients of variation among the 101 population units selected ranged between 22.5 ($\ln N_i$) and 59.6% (R_1), except for H_G (95.6%) and H_{GR} (99.1%). Variation for the two latter variables was mainly attributable to the *P. r. tritici* samples from the Federal Republic of Germany collected in 1987 and 1988 (H_G and H_{GR} much greater

than the mean) and to the 1986 and 1987 *P. g. tritici* populations from the U.S. (H_G and H_{GR} much lower than the mean).

Sample size was positively correlated ($P < 0.001$) with R_1, R_3, C_p and C_i, and negatively ($P < 0.001$) with H_S, H_{GR} and H_{SR} (Table 3). No significant correlation ($P > 0.05$) was found between $\ln N_i$ and either NDD or NDDR. A loose, albeit significant negative correlation ($P < 0.05$) was observed between $\ln N_i$ and $\ln N_p$, as well as between $\ln N_i$ and H_G.

As expected, strong correlations ($P < 0.01$) were found between the different diversity variables and indexes. Positive correlations were observed between diversity indexes (absolute or relative) and $\ln N_p$, as well as between H_G, H_S, H_{GR} and H_{SR}. Negative

TABLE 2. Correlation coefficients between pairs of diversity and complexity variables in 11 pathogen populations[a]

Pairs of variables[b]	Pathogen population[c]										
	P. r. tritici	*P. striiformis*		*P. g. tritici*	*P. coronata*		*E. g. tritici*		*E. g. hordei*		
	1	4	5	6	12	13	15	16	19	20	21
$\ln N_p / \ln N_i$	0.02	0.85*[d]	0.95	−0.11	0.93**	0.77	0.23	−0.15	0.89*	0.77	0.11
$R_1 / \ln N_i$	0.35	−0.53	−0.56	−0.01	−0.49	−0.43	0.26	0.67*	−0.33	0.47	0.79
$R_3 / \ln N_i$	0.46	−0.45	−0.52	−0.08	−0.37	0.09	0.43	0.40	−0.47	0.24	0.70
$H_G / \ln N_i$	−0.87*	0.62	0.89*	−0.18	0.87*	0.35	−0.47	−0.12	0.80	0.40	−0.31
$H_S / \ln N_i$	−0.41	0.60	0.91*	0.09	0.51	0.57	−0.26	−0.38	0.80	0.31	−0.54
$C_i / \ln N_i$	0.89*	0.74	0.32	0.21	0.33	0.38	0.37	0.41	−0.30	−0.91*	−0.46
$C_p / \ln N_i$	0.37	0.77	−0.68	0.31	0.46	0.54	0.33	0.44	0.01	−0.94*	−0.09
$R_1 / \ln N_p$	−0.79	−0.86*	−0.36	−0.81***	−0.57	−0.56	−0.18	−0.56	−0.68	0.16	−0.45
$R_3 / \ln N_p$	−0.70	−0.82*	−0.33	−0.85***	−0.51	−0.41	−0.13	−0.84**	−0.80	0.01	−0.62
$H_G / \ln N_p$	0.30	0.93**	0.99***	0.92***	0.97**	0.87*	0.75*	0.98***	0.97**	0.89*	0.91*
$H_S / \ln N_p$	0.79	0.92**	0.81	0.93***	0.59	0.90*	0.42	0.86**	0.98**	0.71	0.78
$C_i / \ln N_p$	0.00	0.85*	0.25	−0.67**	0.30	−0.10	−0.41	−0.63*	−0.33	−0.45	−0.86
$C_p / \ln N_p$	−0.07	0.94**	−0.83*	−0.79***	0.40	−0.01	−0.29	−0.46	0.01	−0.51	−0.98**
R_3 / R_1	0.88*	0.79*	0.81*	0.89***	0.91*	0.74	0.90***	0.82**	0.81	0.92*	0.96**
H_G / R_1	−0.49	−0.87*	−0.26	−0.82***	−0.59	−0.44	−0.34	−0.59	−0.78	−0.14	−0.77
H_S / R_1	−0.99***	−0.97***	−0.83*	−0.90***	−0.60	−0.69	−0.83*	−0.85*	−0.79	−0.56	−0.88*
C_i / R_1	0.26	−0.88**	−0.14	0.85***	−0.09	−0.10	0.12	0.72*	−0.11	−0.66	−0.02
C_p / R_1	−0.21	−0.93**	0.11	0.71*	−0.07	0.04	−0.20	0.66*	−0.41	−0.55	0.39
H_G / R_3	−0.62	−0.97***	−0.22	−0.87***	−0.60	−0.64	−0.41	−0.88***	−0.82	−0.18	−0.88*
H_S / R_3	−0.92**	−0.90**	−0.75	−0.95***	−0.39	−0.71	−0.86**	−0.98***	−0.88*	−0.69	−0.97**
C_i / R_3	0.55	−0.52	0.14	0.78***	−0.32	−0.22	0.26	0.65*	0.35	−0.46	0.21
C_p / R_3	0.07	−0.73	0.22	0.61**	−0.22	0.43	0.05	0.51	0.19	−0.28	0.60
H_S / H_G	0.53	0.96***	0.74	0.92***	0.61	0.87*	0.55	0.90***	0.98***	0.81	0.96**
C_i / H_G	−0.90*	0.70	−0.19	−0.79***	0.26	−0.44	−0.61	−0.64*	−0.33	0.00	−0.62
C_p / H_G	−0.59	0.85*	−0.88*	−0.78***	0.32	−0.41	−0.49	−0.46	0.12	−0.07	−0.88*
C_i / H_S	−0.34	0.83*	0.17	−0.76***	−0.43	−0.07	−0.49	−0.67*	−0.31	0.09	−0.43
C_p / H_S	0.14	0.93**	0.59	−0.71**	−0.33	−0.06	−0.04	−0.52	0.04	−0.07	−0.77
C_i / C_p	0.61	0.92**	0.28	0.65**	0.97**	0.94**	0.76*	0.95***	0.72	0.98**	0.91*

[a] *Puccinia recondita tritici*/RSA; *P. striiformis*/F; *P. striiformis*/BU; *P. graminis tritici*/USA; *P. coronata*/Manitoba-Sask., CAN; *P. coronata*/Ontario-Quebec, CAN; *E. g. tritici*/PL; *E. g. tritici*/YU; *E. g. hordei*/Northern F; *E. g. hordei*/Paris area, F; *E. g. hordei*/Eastern F. See footnote C below for regional location codes.

[b] Codes for variables: $\ln N_i$ = natural logarithm of isolate number; $\ln N_p$ = natural logarithm of pathotype number; R_1 = frequency of the most frequent pathotype; R_3 = cumulated frequencies of the 3 most frequent pathotypes; H_G = Gleason index of diversity; H_{GR} = relative Gleason index of diversity; H_S = Shannon index of diversity; H_{SR} = relative Shannon index of diversity; C_i = average isolate virulence complexity; C_p = average pathotype virulence complexity; NDD = number of discriminant differentials; NDDR = relative number of discriminant differentials.

[c] Numbers preceding each population are given for referencing in the text. Regional locations are given only when local populations could be separated within national data. BU = Bulgaria; CAN = Canada; CHI = People's Republic of China; D = Federal Republic of Germany; F = France; ISR = Israel; PL = Poland; RSA = Republic of South Africa; USA = United States of America; YU = Federal Republic of Yugoslavia.

[d] *, **, and *** indicate significance at the 0.05, 0.01 and 0.001 probability levels, respectively.

TABLE 3. Correlation coefficients of race diversity and complexity variables[a], calculated for the 101 pathosystem units (pathogen-year-location combinations) selected for performing principal component analysis

	$\ln N_i$	$\ln N_p$	R_1	R_3	H_G	H_{GR}	H_S	H_{SR}	C_p	C_i	NDD
$\ln N_p$	−0.191*[b]										
R_1	0.579	−0.731									
R_3	0.499	−0.832	0.874								
H_G	−0.197*	0.869	−0.612	−0.780							
H_{GR}	−0.708	0.594	−0.675	−0.827	0.634						
H_S	−0.410	0.928	−0.884	−0.951	0.862	0.744					
H_{SR}	−0.723	0.725	−0.870	−0.933	0.682	0.930	0.889				
C_p	0.353	−0.318	0.352	0.316**	0.092 NS	−0.234*	−0.323	−0.359			
C_i	0.456	−0.399	0.488	0.417	−0.208*	−0.332	−0.440	−0.472	0.936		
NDD	0.166 NS	0.405	−0.217*	−0.206*	0.408	0.070 NS	0.326	0.122 NS	0.327	0.220*	
NDDR	0.101 NS	0.279**	−0.152 NS	−0.096 NS	0.056 NS	0.067 NS	0.177*	0.059 NS	−0.176 NS	−0.095 NS	0.486

[a] Codes for variables: $\ln N_i$ = natural logarithm of isolate number; $\ln N_p$ = natural logarithm of pathotype number; R_1 = frequency of the most frequent pathotype; R_3 = cumulated frequencies of the 3 most frequent pathotypes; H_G = Gleason index of diversity; H_{GR} = relative Gleason index of diversity; H_S = Shannon index of diversity; H_{SR} = relative Shannon index of diversity; C_i = average isolate virulence complexity; C_p = average pathotype virulence complexity; NDD = number of discriminant differentials; NDDR = relative number of discriminant differentials.

[b] All coefficients significant at $P = 0.001$, except those marked ** (significant at $P = 0.01$), * (significant at $P = 0.05$) and NS (non significant, $P > 0.05$).

correlations occurred between richness (H_G, $\ln N_p$) and dominance (R_1, R_3) estimates. Redundant information on the set of population samples examined was thus provided by $\ln N_p$, R_1, R_3, H_G, H_S, H_{GR}, and H_{SR}.

As a general trend, C_i and C_p were positively correlated with dominance estimates, and negatively with richness and diversity indexes. However, no significant correlation ($P > 0.05$) was found between C_p and H_G, and only weak correlations ($0.01 < P < 0.05$) arose between C_i and H_G on one hand, C_p and H_{GR} on the other.

NDD was positively correlated with $\ln N_p$, H_G, H_S, C_p and NDDR, and negatively with R_1 and R_3. NDDR was positively correlated only with $\ln N_p$ ($p < 0.001$) and H_S ($p < 0.05$), and negatively with the dominance estimates R_1 and R_3 ($p < 0.05$). Correlations with NDDR were apparent neither for complexity variables nor for relative diversity indexes.

Due to the above-mentioned correlations between variables, PCA was performed using $\ln N_i$, H_G, C_p, NDD, and NDDR as variables. These were chosen because they showed the least correlations with one another and displayed maximum variability.

The first plane of principal components represented 63.4% of total variation, with almost equal repartition betwen the two axes (34.4 and 29.0% for the first and second principal axes, respectively). The third, fourth, and fifth axes accounted for 20.5, 11.8, and 4.3% of total variance. The first axis was mainly determined by differential set variables (loadings of −0.924 and −0.612 for NDD and NDDR, respectively), while the remaining three variables were the main contributors to the second axis (negative loadings for $\ln N_i$ and C_p, positive loading for H_G; absolute values ranging from 0.597 to 0.703). The third axis mostly accounted for variation in H_G on one hand (loading +0.561) and in NDDR on the other hand (loading −0.670). Due to the low percentage of total variance explained by the fourth and fifth axes, interpretation was restricted to the first three principal components.

The repartition of the 101 population units over the first factorial plane showed extensive scattering of the *P. r. tritici,*

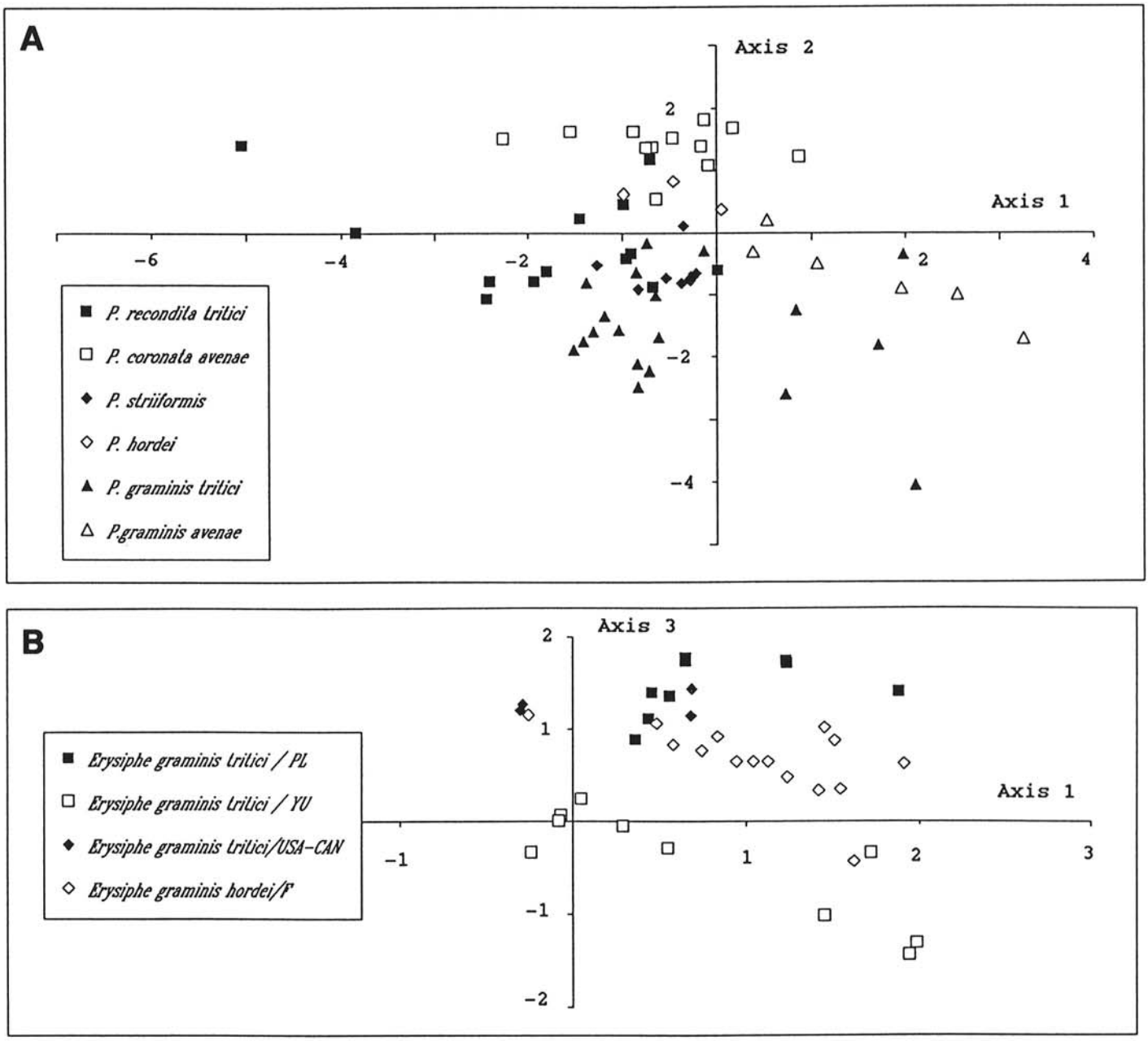

Fig. 1. Repartition of the 101 yearly samples from Table 1 over first factorial plane (axes 1 and 2), computed by principal component analysis using $\ln N_p$, H_G, C_S, number of discriminant differentials and relative number of discriminant differentials as variables. For the sake of clarity only, data for the rust pathogens were grouped on Fig. 1A and those for the other pathogens on Fig. 1B.

P. g. tritici, *P. g. avenae,* and, to a lesser extent, *P. coronata* samples (Fig. 1A). The point cloud corresponding to *P. coronata* was characterized by high positive coordinates on the second principal axis (high diversity, low complexity) and mostly negative coordinates on the first one (high number of discriminant differentials). On the other hand, *P. g. tritici* samples lay in the lower two quadrats (corresponding to high complexity and limited diversity), but formed a cloud extending along the first axis. *P. r. tritici* was a very heterogeneous group, with singular points corresponding to the German populations. *Puccinia striiformis* and *P. hordei* samples clustered in the vicinity of the origin; French and Bulgarian populations of *P. striiformis* were not separated. *Erysiphe g. tritici* samples split into two groups, one corresponding to the Yugoslavian populations and the second to the Polish and North American samples. These two groups were separated mainly on the second axis, reflecting higher diversity and lower complexity in the Polish/North American populations than in the Yugoslavian samples (Fig. 1B). *Erysiphe g. hordei* populations clustered in an intermediate position between both *E. g. tritici* groups (Fig. 1B).

Point clouds corresponding to *P. g. avenae, P. g. tritici, P. hordei,* and *E. g. tritici* were orientated in parallel lines along the first bissector of the second principal plane (first and third axes) (Fig 2A and 2B), indicating simultaneous variation for differential set and diversity variables within each of these groups. On the other hand, the clouds corresponding to *E. g. hordei* and *P. coronata* extended mainly parallel to the first principal axis, indicating predominant variation for differential set variables. Again, *P. r. tritici* formed an extremely scattered group, with the three German samples lying on the upper left side of the plane and the South African ones on the lower left quadrat, indicating high discriminance of the host set used and limited race diversity. The two *P. striiformis* populations again were grouped close to the origin. The two groups of *E. g. tritici* populations identified on the first factorial plane were less clearly separated, a consequence of the lower contribution of diversity and complexity variables to this plane and of the greater contribution of differential set variables for which the two groups had similar characteristics. However, *E. g. tritici* samples clustered away from *E. g. hordei* (negative coordinates on the third axis

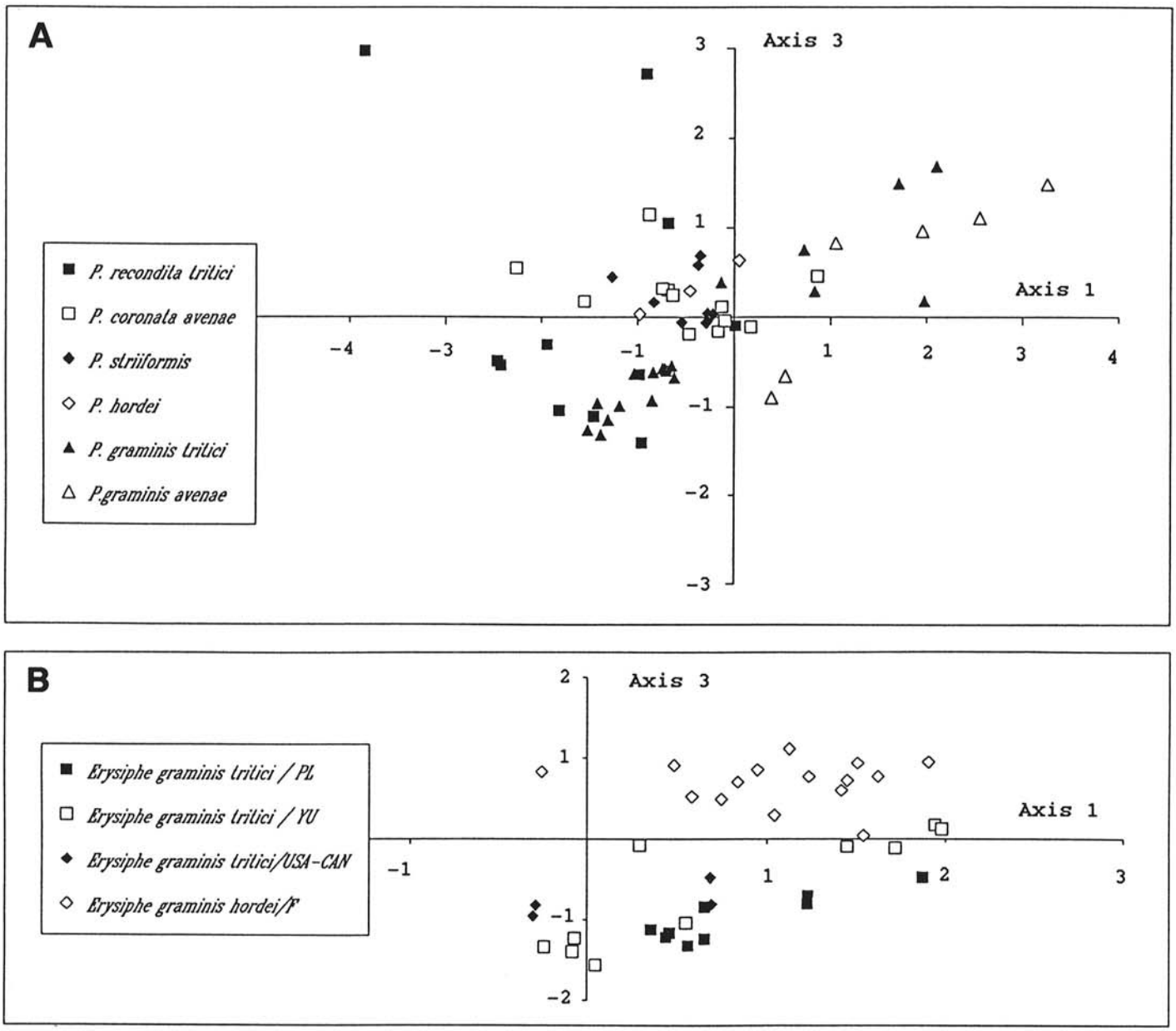

Fig. 2. Repartition of the 101 yearly samples from Table 1 over the second factorial plane (axes 1 and 3), computed by principal component analysis using $\ln N_p$, H_G, C_S, number of discriminant differentials and relative number of discriminant differentials as variables. For the sake of clarity only, data for the rust pathogens were grouped on Fig. 2A and those for the other pathogens on Fig. 2B.

for *E. g. tritici*, positive coordinates for *E. g. hordei*), a consequence of the higher diversity and lower proportion of discriminant differentials in *E. g. hordei* samples. Similar conclusions were obtained from the examination of the third factorial plane (second and third principal axes; data not shown).

DISCUSSION

Methodological aspects of pathosystem comparison. This study compared race features of different populations of fungal cereal pathogens using descriptive statistical methods. These methods are only indicative of the extent to which variables vary simultaneously in the same or opposite directions, or to which populations had similar race diversity and complexity patterns. They, however, do not suppose or provide any explanation for the relationships (or for their absence). Therefore, most of this discussion will be devoted to formulating explanatory hypotheses concerning the results obtained and to analyzing their consequences.

Simultaneous comparison of different pathogens or populations by means of PCA should be interpreted with care, because not all pathosystems were analyzed with the same number of differential hosts and not all differentials were equally discriminant. The first principal axis, representing over one-third of the total variability of the data from the 101 units, was almost exclusively determined by the two variables describing the differential sets (NDD and NDDR), a fact to be kept in mind when analyzing groupings on the first factorial plane. However, populations surveyed with the same sets of differentials were markedly dissimilar on the second principal axis (e.g., *E. g. tritici* samples from Poland and Yugoslavia), thus stressing the influence of local factors (such as the history of selection exerted by resistance genes, or the annual number of pathogen generations) on diversity and complexity. The large part of total variance accounted for by differential set variables makes comparisons of pathosystems investigated with different numbers of discriminant hosts difficult, but also shows that these variables should be included whenever populations or pathosystems are compared for their race patterns.

It should also be emphasized that sampling procedures are obviously a major bias in the description of population structures. While relatively small samples may reflect with reasonable accuracy the diversity present in a population, they may fail to reveal the overall diversity present in the area. This is particularly true with samples collected from few sites and with pathogens structured as metapopulations, i.e., as populations composed of groups of individuals colonizing different sites, each group having a non-zero probability of extinction and having the possibility of being restored through migration from other sites (40). This structure, probably common among important plant pathogens, such as *P. infestans* (13), makes evaluation of diversity difficult, since sampling has to be performed in ways allowing all groups to be taken into account. The extent of the biases caused by the two levels of uncertainty associated with sampling (representativity of the overall population by the population sampled, and representativity of the population sampled by the sample itself) are a function of both the sampling scheme retained and the biology of the pathosystem, and should not be overlooked when comparisons between pathosystems are attempted.

Diversity patterns. Few consistent trends could be recognized concerning the mutual relationships of the several diversity estimates and parameters over the different populations examined. This may be partly due to the often low number of yearly samples considered for each population (5–6 in most cases), which reduced the possibility of recognizing highly significant correlations. Of particular interest are the correlations including sample size as one of the variables. Sample size of at least 30 isolates has been one of the criteria in the selection of the populations studied, in order to avoid excessive biases in estimates of richness and evenness of the samples. Under these conditions, sample size was generally not strongly correlated with diversity and complexity estimates, at variance with some previous reports (15,27) but consistent with observations concerning *P. coronata* in Canada over a 17-yr period (9). With a few exceptions, sample size was not significantly correlated with any of the variables retained for the description of the 11 populations considered individually. This was particularly apparent for the 15-yr samples of *P. g. tritici* in the U.S., an observation departing from calculations made over a longer period (15). However, it can be noted that correlation coefficients calculated over the 1918–1982 period from the data of Groth and Roelfs (15), although significant, are usually low when sample size is taken as one of the variables (from 0.133 for N_i/N_p to 0.590 for N_i/R_3). Their statistical significance could, therefore, be mainly a consequence of the large number of yearly samples included in the calculation. The lack of correlation between sample size and the number of discriminant differentials and the weak correlation between $\ln N_i$ and $\ln N_p$ both suggest that increasing sample size to several thousands of isolates does not necessarily improve consistently the qualitative description of the population studied. This is true, of course, provided that sample sizes are not too small (at least 30–50 individuals per sample). This observation particularly applies to very homogeneous populations, such as those of *P. g. tritici* in the U.S. H_G, usually considered to be more sensitive to sample size than H_S (15), proved not to be so with the present set of data.

Relevant data for estimating the contribution of the several components to diversity are rare, mainly because measures of richness or evenness alone are not available. Therefore, all indexes or variables related to phenotypic diversity incorporate both of its components to a varying extent. Hence, positive correlations between synthetic diversity estimates (H_G, H_S, H_{GR}, H_{SR}), and negative correlations between dominance and richness estimates are to be expected. However, the frequent lack of correlation between dominance estimates (R_1/R_3) and H_G among the 11 pathosystems for which data were available over several years tends to indicate that richness and evenness varied independently in these populations. The general value of this independence should now be confirmed in other race-forming plant pathogens.

The major problem with long-term comparisons of race diversity is that the differential set has to be identical for the entire period. If the original set was restricted to a few resistance genes, only part of the virulence variability will be detected. As resistance genes absent from the set are introduced into commercially successful cultivars, the variability detected in the pathogen population using the original set of differentials might be very far from the actual diversity present. Therefore, trends toward decreasing diversity, commonly observed during long-term surveys (15,21), may be largely artificial and reflect only the progressive loss in discrimination capacity of old sets. For instance, *P. r. tritici* in Canada shows a very high diversity when screened with a modern, extended set of differential lines, but only limited variation with the old international set (21). A similar example can be found with the same pathogen in western Europe (58).

Diversity-complexity relationships. In the populations investigated, the relationships between changes in race diversity and complexity depend more on the population considered than on the taxonomic identity of the pathogen. However, the simultaneous analysis of the 101 yearly samples showed that complexity estimates were generally positively correlated with dominance variables and negatively with diversity. Such correlations are not easy to interpret. It is possible that the statistical significance of the correlations is mainly caused by the weight of populations, such as the U.S. *P. g. tritici* samples, in which diversity was low and complexity was high. However, this hypothesis does not provide any explanation for the cause of this structure in the populations concerned. Two mechanisms may lead to such a structure. It can be supposed that, because of the relatively low number of complex virulence combinations possible, populations dominated by complex races should be globally less diverse than those composed predominantly of simpler races. The relevance of this explanation is greater when predominant virulence combinations included almost all genes present in the differential set, as in the U.S. population of *P. g. tritici*. However, this situation was rather unusual among the other populations studied, for which C_i was generally much lower than NDT, and even than NDD. An alternative hypothesis is that complex races are often at a

selective advantage over simple ones, and consequently tend to predominate in populations. If this is the case, once complex races appear, complexity would increase and diversity decrease. This could happen either in populations in which selection for flexibility (60,61) is important (i.e., resistance genes are separate but common in the host crops), or in pathogens faced with hosts with combined resistance genes.

The different behavior of geographically distinct populations of the same fungus suggests that local factors are of utmost importance in the determination of race diversity and complexity patterns. Therefore, experience with one population of a pathogen is not sufficient to enable a reliable prediction of the structure and evolution of other populations of the same organism, even those that are geographically close.

Three main factors may account for these structures: the dynamics and history of the use of resistance genes in popular cultivars; the biology of the pathogen and particularly its breeding system; and stochastic effects. It is no surprise that races with the highest complexity were found in clonally reproducing pathogens long faced with race-specific host resistance genes, such as *P. g. tritici* in the U.S. or *P. r. tritici* and *P. striiformis* in Europe. Conversely, fungi that may undergo sexual reproduction at frequent intervals, such as *E. graminis,* or that have only recently been combatted by means of resistant cultivars, such as *P. coronata* in western Canada (5,6), had the least complex races and often a greater diversity of virulence phenotypes. Therefore, it may be hypothesized that most races of the first group of pathogens carry "fossil" virulences, once selected for and that have been maintained in the population because no mechanism existed to remove them—a phenomenon akin to Muller's ratchet (37). Examples of such "fossil virulences" can be found with the genes matching *Lr2a, Lr2c,* or *Lr3* in German populations of *P. recondita.* These virulences have been present since the 1930s (16,54,55), and are still detected at a high frequency (58), although current German cultivars are thought to carry only *Lr3, Lr4a,* or *Lr26* (58).

Stochastic events (i.e., genetic drift) have been shown to influence the evolution of *E. g. hordei* (4). They probably act in the same manner in most biotrophic pathogens of annual crops, because of the severe demographic gaps that these pathogens suffer after crop harvest. Genetic drift may alter very rapidly the composition of pathogen populations, especially sexual ones in which recombination provides variability, and hence contribute to generate divergent race structures among populations of a given pathogen.

The influence of fungal breeding systems on diversity and complexity patterns can be further illustrated by the comparison between *E. g. hordei* and *P. striiformis* in Europe. In both cases, specific resistance genes have been used at least since the 1930s and introduced one at a time into commercial cultivars (56,62). This trend is still apparent today, as most cultivars carry only 2–3 resistance genes (3,12). However, diversity and complexity patterns are clearly divergent in these two pathogens. One major difference is the occurrence of a yearly sexual cycle in *E. graminis* and the absence of sexual mating in *P. striiformis*. Table 1 shows that *P. r. tritici* in South Africa and *P. g. tritici* in the central and eastern U.S., two pathogens with no or extremely limited sexual reproduction, also display populations composed mainly of a few complex races.

Sexual populations of *P. g. tritici* and *P. g. avenae* from North America have been shown to be generally more diverse and composed of simpler races than their clonal counterparts (14, 32,51). However, the distinction between sexual and asexual populations of the two fungi was not clearly apparent in the present analysis. This may be due in part to the reduced number of sexual populations available, but also to common characteristics in the differential sets used for screening both sexual and asexual samples and to the extensive variation in diversity and complexity among asexual samples from different years. Under these conditions, a clear-cut separation between sexual and asexual populations could hardly be expected. A general comparison of diversity and complexity patterns in sexual and asexual populations of plant pathogens is still lacking. It might be attempted using multivariate analysis in a way similar to that used for the present work, provided that both groups are represented in almost equal proportions and that large biases, such as obviously different sample sizes or differential host numbers, are avoided. Unfortunately, these prerequisites could not be met with the data available to date.

The four major conclusions of this study may thus be summarized as follows. In populations of obligate fungal pathogens of cereals (rusts and powdery mildews), the race diversity and complexity recognized depended mainly on the number and discriminating capacity of the differential set used, and on local factors such as the history of selection induced by resistance gene deployment, the presence or absence of a sexual stage, and the incidence of genetic drift. Race diversity and complexity were generally negatively correlated, and some possible explanations for this have been proposed. Finally, sample size was not usually correlated with diversity or complexity estimates. Altogether, these findings indicate that experience with one population of a pathogen cannot be readily extrapolated to other populations of the same or related organisms, and that comparisons should take into consideration both technical and biological biases. Further work is now needed to investigate the possibility of extending these conclusions to other race-forming pathogens, and to test some of their possible explanations.

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