Patterns of resistance of Israeli wild emmer wheat to pathogens
I. Predictive method by ecology and allozyme genotypes for powdery mildew and leaf rust

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Abstract

The association of ecological factors and allozyme markers with genotypes of tetraploid wild emmer wheat, *Triticum dicoccoides*, varying in resistance to four cultures of the pathogen *Erysiphe graminis tritici*, and to one culture of *Puccinia recondita tritici*, which incite the diseases powdery mildew and leaf rust respectively, were explored theoretically and practically. The study involved 233 accessions comprising 10 populations representing the ecological range of *T. diococcoides* in Israel. Our results indicate that genetic polymorphism for resistance to both pathogens is structured geographically, and is predictable by climatic as well as allozymic markers. Three variable combinations of water factors and temperature differentials significantly explain 0.27 and 0.14 of the spatial variance for resistance to powdery mildew and leaf rust, respectively, suggesting the involvement of natural selection. Several allozyme genotypes, singly, or in combination, are significantly associated with disease resistance. We conclude that *T. dicoccoides* populations in Israel, which grow in the center of diversity of the species, contain large amounts of unexploited disease resistant genotypes. The populations could be effectively screened and utilized for producing resistant cultivars by means of ecological factors and allozyme markers as predictive guidelines.

Introduction

The wild relatives of wheat include significant genetic variability displaying local and regional adaptive differentiation to different climates, soils and pathogens (Nevo *et al.*, 1982). Wild wheats are genetic resources that should be exploited in breeding programmes to introduce resistance to a broad range of diseases, pests, and for tolerance to poor soils and climatic extremes, so that yields will be more stable and agriculture can be extended to marginal environments. Furthermore, gains are likely to be more durable if more than one gene for resistance or tolerance is transferred to a cultivar (Plucknett *et al.*, 1983). Recently, we studied the genetic structure of wild barley (Nevo *et al.*, 1979) and of wild emmer wheat (Nevo *et al.*, 1982), to identify the potential useful genetic resources in the wild relatives of barley and wheat for breeding cultivars with agronomically important characteristics (Nevo *et al.*, 1984c), including disease resistance (Moseman *et al.*, 1983, 1984a, b; Nevo *et al.*, 1984a).

Wild emmer wheat, *Triticum dicoccoides* (genome AABB), is distributed in the Near East Fertile Crescent, and northern Israel appears to be its center of distribution. *T. dicoccoides* is the progenitor of all cultivated forms of tetraploid wheat, *T. turgidum*, and hexaploid wheat, *T. aestivum* (Feldman, 1976; Feldman & Sears, 1981). It is conspecific, and cytogenetically close to cultivated tetraploid wheats with which it is fully interfertile, and is partly fertile with hexaploid wheat. Thus, genes can be transferred from the wild to the cultivated gene pool. *T. dicoccoides* is therefore a convenient source of wild germplasm for improving cultivated wheats. Examples of the agronomically important characters

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already found in *T. dicoccoides*, and partly transferred to cultivated wheat, are large grain size and high protein content (Gerechter-Amitai & Grama, 1974; Avivi, 1979a, b; Grama *et al.*, 1984), and resistance to stripe rust (Gerechter-Amitai & Stubbs, 1970; Gerechter-Amitai & Grama, 1974; Grama & Gerechter-Amitai, 1974). Wild emmer has been also studied as a potential source of powdery mildew and leaf rust resistance (Moseman *et al.*, 1984a, b; and El-Morshidy *et al.*, 1983).

The extensive allozymic diversity found in 12 populations of *T. dicoccoides* in Israel (Nevo *et al.*, 1982) involving 110 allozyme alleles in 50 gene loci, some of which are adaptively associated with the environment, indicates that significant genetic resources for wheat improvement exist in the wild gene pool. Therefore, we explored 10 of the 12 populations studied earlier for allozymic diversity, for potential genetic sources of resistance to wheat powdery mildew, *Erysiphe graminis tritici*, and to leaf rust, *Puccinia recondita tritici*. Our results indicated that the number of sources of resistance to *E. graminis tritici* which can be obtained from wild *T. dicoccoides* in Israel is almost unlimited (Moseman *et al.*, 1984a). Almost 50% of the accessions tested were resistant to infection with 4 cultures of *E. graminis tritici* which possess the virulence genes corresponding to most of the identified resistance genes in wheat. A distinctly lower proportion of the accessions tested (about 15%) were resistant or moderately resistant to leaf rust. The proportion of resistant *T. dicoccoides* plants varied geographically. Most plants resistant to powdery mildew were found in northern Israel, whereas the single accession resistant to leaf rust was found in the western marginal population of Bat Shelomo, near the Coastal Plain. Moreover, disease resistance, at least in powdery mildew, appears as a polymorphism (resistant and susceptible phenotypes with rare intermediates occur *within* populations: Moseman *et al.*, 1984a; El-Morshidy *et al.*, 1983).

Evaluation of genetic resources through isozyme studies provide an important aid in efficient collection, storage and use (Brown & Clegg, 1983). Moreover, it should be of prime importance to locate, through allozyme markers, multiple optimal genotypes in natural populations which combine associations to various disease resistances with elite agronomic traits (Nevo *et al.*, 1984c) in order to minimize yield depression in breeding.

The objectives of this study were: (a) to analyze the correlations of climatic factors to resistance in *T. dicoccoides* to the two pathogens, *E. graminis tritici* and *P. recondita tritici*, in order to identify potential populations and genotypes with maximal resistance to both pathogens, and (b) to explore correlations of allozymic proteins to resistance to powdery mildew and leaf rust, in order to identify allozyme markers which are associated, singly or in combination, with disease resistance.

We report here the ecological correlations and allozymic associations in *T. dicoccoides* in Israel which can be utilized for identifying the best sites and genotypes of wild wheat for breeding cultivars resistant to powdery mildew and leaf rust.

Material and methods

The study involved 233 accessions of *T. dicoccoides* consisting of population samples collected at 10 sites in Israel. All populations were described geographically, ecologically, and allozymically by Nevo *et al.* (1982); for their reactions to infection with *E. graminis tritici* by Moseman *et al.* (1984a), and to infection with *P. recondita tritici* by Moseman *et al.* (1984b). The above description is summarized in Table 1. The four cultures of *E. graminis tritici*, Quincy, Mo10, 127 and ABK, possess virulence genes corresponding to known resistance genes to that pathogen in wheat. At least one of the four cultures, Quincy, Mo10, 127, and ABK, in the two composites of cultures of *E. graminis tritici*, was virulent on 9 of the 10 identified resistance genes, which confer resistance to *E. graminis tritici*, in wheat. The cultures were virulent on resistance genes *Pm1*, *Pm2*, *Pm3a*, *Pm3b*, *Pm3c*, *Pm4*, *Pm5*, *Pm6*, and *Pm7*, and avirulent on gene *Pm8* in Kavkaz. The culture PRTUS #6 of *P. recondita tritici* had the virulence characteristics of cultures most frequently isolated in the Eastern United States. The avirulent/virulent formula for culture PRTUS #6 was 2a, 9, 16, 18, 19, 24/1, 2c, 3d, 3, 10, 11, 17. The procedures and methods of inoculation and reading reactions and the variation in the reactions within and between *T. dicoccoides* accessions in the populations at the 10 sites are described in detail by Moseman *et al.*, 1984a and Moseman *et al.*, 1984b.

We determined the associations of ecological factors and allozymic genotypes with resistance to *E.*

Table 1. The climatic background* of 10 populations of wild wheat, *Triticum dicoccoides*, in Israel followed by the infection types (IT) against 4 cultures of the pathogen *E. graminis tritici*, composite 1 (Quincy, Mo10), composite 2 (127, ABK) and their average (IT-av); and the infection type (IT) against 1 culture (no. 6) of the pathogen *Puccinia recondita tritici*.

Locality	No.	Lon.	Lat.	Alt.	Tm	Ta	Tj	Td	Tdd	Rn	Rd	Hu 14	Hu an	Dw	Sh	Th	Trd	Ev	So	IT-1 ± sd.	IT-2 ± sd.	IT-av. ± sd.	IT-LR ± sd.
Mt. Hermon	14	35.73	33.30	1300	11	21	3	18	6	1400	66	48	60	60	80		0	150	1	5.0 ± 2.78	6.1 ± 2.59	5.5 ± 2.58	7.6 ± 0.85
Qazrin	35	35.67	33.02	350	18	26	10	16	12	530	50	43	58	58	50		60	155	5	2.1 ± 0.84	1.7 ± 0.63	1.9 ± 0.57	6.9 ± 0.99
Yehudiyya	13	35.70	32.93	200	19	27	11	16	12	550	47	42	58	58	50		100	160	5	5.5 ± 2.88	5.7 ± 2.95	5.6 ± 2.85	8.0 ± 0.00
Rosh Pinna	28	35.52	32.95	700	18	25	9	16	10	697	50	48	58	50	75	10	35	150	1	2.5 ± 1.73	1.8 ± 1.32	2.1 ± 1.36	7.9 ± 0.32
Tabigha	32	35.53	32.90	0	24	32	15	17	10	436	45	45	57	58	60	30	120	160	5	4.6 ± 2.64	4.5 ± 2.88	4.5 ± 2.67	8.0 ± 0.40
Bat Shelomo	32	35.02	32.60	75	20	26	13	13	10	650	55	58	68	77	40	10	30	150	2	6.8 ± 2.13	5.8 ± 2.73	6.3 ± 2.02	7.3 ± 1.33
Kokhav-Hashahar	34	35.34	31.95	600	20	28	12	16	12	400	40	45	59	30	80	30	25	165	1	3.4 ± 2.27	3.1 ± 2.38	3.2 ± 2.25	7.4 ± 0.82
Taiyiba	9	35.35	31.92	450	19	26	10	16	12	400	40	44	58	30	80	10	25	165	1	4.2 ± 2.49	4.9 ± 3.76	4.6 ± 2.93	8.0 ± 0.00
Bet Meir	18	35.03	31.80	500	19	26	11	15	9	582	44	47	60	61	70	10	100	160	1	5.5 ± 3.26	5.9 ± 3.26	5.7 ± 3.10	6.3 ± 1.33
Sanhedriyya	18	35.22	31.80	800	17	24	9	15	9	548	44	51	62	44	102	10	0	155	1	7.2 ± 1.95	6.7 ± 2.50	6.9 ± 2.18	7.5 ± 0.51
Total	233																			4.41 ± 2.79	4.17 ± 3.00	4.29 ± 2.77	7.43 ± 0.97

* Climatic values were taken from the Atlas of Israel (1970) and from multiple-year records of the Meteorological Service of Israel.

Symbols of variables:

(i) Geographical var.: Lon. = Longitude, in decimals; Lat. = Latitude, in decimals; Alt. = Altitude, in meters; (ii) Temperature var.: Tm = Mean annual temperature; Ta = Mean August temperature; Tj = Mean January temperature; Td = Mean seasonal temperature difference; Tdd = Mean day-night temperature difference; Trd = Mean number of tropical days; Sh = Mean number of Sharav days, i.e., hot and dry days; Ev = Mean annual evapoaration; (iii) Water availability: Dw = Mean number of dew nights in summer; Rn = Mean annual rainfall, in mm; Rd = Mean number of rainy days; Hu 14 = Mean humidity at 14:00; Hu an = Mean annual humidity; Th = Thornthwaite's moisture index; (iv) Edaphic variables; So = Soil type: 1 = Terra Rosa; 2 = Rendzina; 5 = Basalt.

No. = Number of accessions; IT = The infection types produced by the reactions were read on a 0 to 9 scale from immune to very susceptible: 1 – 3 = resistant, 4 – 6 = moderately resistant, 7 – 9 = susceptible (see details in Moseman *et al.*, 1984a). IT-1 = mean infection type by compound 1 of powdery mildew (*E. graminis tritici*); IT-2 = mean infection type by compound 2 of powdery mildew (*E. graminis tritici*; IT-av = mean of both infection types by powdery mildew (*E. graminis tritici*); IT-LR = mean infection type against leaf rust (*P. recondita tritici*).

graminis tritici. The statistical analysis involves uni-and multivariate computer programs described by Hull and Nie (1981). All climatic variables and the levels of immunity are defined at the bottom of Table 1. Notably, various analyses depend on different sample sizes due to two reasons: (i) For leaf rust only 233 plants were analysed corresponding to those tested for powdery mildew, and (ii) Only 232 plants were electrophoresed and in this sample some genetic systems were unscorable in some plants (n = 190–231).

Results

The results are described in the tables and figures. The climatic matrix of the 10 populations analyzed for disease resistance and their average infection types against powdery mildew and leaf rust are given in Table 1.

Correlations with ecological factors

1. Univariate analysis (Table 2)

(a) *Erysiphe graminis tritici*. Resistance to powdery mildew is significantly correlated with aridity (low humidity), and both seasonal and daily temperature differentials. Resistance also increases eastwards and northwards. The highest Spearman rank correlations are positive with daily temperature differentials ($r_s = 0.363$***), and negative with annual humidity ($r_s = -0.354$***).

(b) *Puccinia recondita tritici*. The geographic patterns of resistance to leaf rust contrast with those of powdery mildew. Only few accessions (15%) proved resistant or moderately resistant (Moseman *et al.*, 1984b), in contrast to the abundance of resistance (50%) against powdery mildew (Moseman *et al.*, 1984a). Nevertheless, moderate leaf rust resistance shows a correlation with ecological factors. Moderate leaf rust resistance is significantly and positively correlated with annual humidity ($r_s = 0.233$***), and negatively with seasonal temperature difference ($r_s = -0.243$***). Note that the direction of correlations in these parameters is opposite to that in powdery mildew. The above correlations derive primarily from the presence of moderate resistance to leaf rust in two populations: Bet Meir and Qazrin.

Table 2. Spearman rank correlations (r_s) of geographic and climatic variables with disease resistance to 4 cultures of wheat powdery mildew and 1 culture of leaf rust pathogens in *T. dicoccoides* in Israel.

Variables	Resistance to: powdery mildew Cultures:	Quincy, Mo10	127, ABK	Average	leaf rust No. 6
Geographic	Longitude	0.321 ***	0.215 ***	0.281 ***	0.131 *
	Latitude	0.337 ***	0.296 ***	0.337 ***	0.086 n.s.
	Altitude	0.107 n.s.	0.101 n.s.	0.102 n.s.	0.081 n.s.
Temperature variables	Annual	0.127 @	0.120 @	0.132 *	0.143 *
	August	0.042 n.s.	0.013 n.s.	0.025 n.s.	0.088 n.s.
	January	0.181 **	0.171 **	0.183 **	0.071 n.s.
	Season. dif.	0.281 ***	0.155 *	0.221 ***	0.243 ***
	Daily diff.	0.332 ***	0.355 ***	0.363 ***	0.025 n.s.
	Tropical d.	0.134 *	0.107 n.s.	0.124 @	0.046 n.s.
	Evaporation	0.065 n.s.	0.002 n.s.	0.023 n.s.	0.020 n.s.
	Sharav	0.032 n.s.	0.007 n.s.	0.001 n.s.	0.059 n.s.
Water availability	Rainfall	0.124 @	0.096 n.s.	0.112 @	0.032 n.s.
	Rainy days	0.025 n.s.	0.008 n.s.	0.006 n.s.	0.027 n.s.
	Humid. at 14	0.353 ***	0.273 ***	0.325 ***	0.034 n.s.
	Humidity (ann)	0.368 ***	0.319 ***	0.354 ***	0.233 ***
	Dew nights	0.240 ***	0.263 ***	0.256 ***	0.115 @
	Thornthwaite moisture ind.	0.202 **	0.111 n.s.	0.162 *	0.104 n.s.

Levels of significance: * = $p < 0.05$; ** = $p < 0.01$; *** = $p < 0.001$; @ = $p < 0.10$; n.s. = $p > 0.10$. N = 171 accessions for Thornthwaite's moisture index and N = 233 for all other correlations.

Table 3. Coefficients of multiple determination (R^2) with dependent variables of resistance to powdery mildew (IT-1, IT-2, and IT-av) and to leaf rust (IT-LR), and as independent climatic variables in 10 populations of wild wheat, *Triticum dicoccoides*, in Israel (for abbreviations see Table 1).

Stepwise model

A. Powdery mildew				
Average resistance	Hu14	Hu14 Ev	Hu14 Ev Tdd	Hu14 Ev Tdd Td
(IT-av)	0.138 ***	0.183 ***	0.266 ***	0.291 ***
Resistance to cult.	Hu14	Hu14 Ev	Hu14 Ev Tdd	Hu14 Ev Tdd Td
Quincy, Mo10 (IT-1)	0.175 ***	0.215 ***	0.263 ***	0.283 ***
Resistance to cult.	Tdd	Tdd Td	Tdd Td Ev	Tdd Td Ev Rd
127, ABK (IT-2)	0.116 ***	0.166 ***	0.262 ***	0.275 ***
B. Leaf rust				
Resistance to	Td	Td Hu14	Td Hu14 Tdd	Td Hu14 Tdd Rd
cult. no. 6 (IT-LR)	0.035 **	0.065 ***	0.129 ***	0.139 ***

Level of significance: ** = $p < 0.01$; *** = $p < 0.001$; N = 233.

2. Multivariate analysis

(a) *Multiple regression analysis (MR).* We conducted a stepwise multiple regression analysis (Hull & Nie, 1981) employing resistance values as dependent variables and climatic variables as independent variables (Table 3), to explain the variance in powdery mildew resistance by a combination of ecological factors. While single variables significantly explain only 0.12–0.18 of the variance in disease resistance, 2-variable combinations explain 0.17–0.22, and 3-variable combinations account for 0.26–0.27. All values are highly significant ($p < 0.001$). The explanation involves primarily a combination of humidity, evaporation, and temperature differences. Essentially, the same 3-variable combination that explained one fourth of the variance in resistance to powdery mildew explained 14% of the variance in resistance to leaf rust.

(b) *Smallest Space Analysis (SSA).* The multivariate pattern was analyzed by means of SSA, which is a nonmetric technique designed to plot intercorrelations of multivariate data in a space of minimal dimensionality (Guttman, 1968; Levy, 1981). The SSA treats each variable as a point in a Euclidean Space so that the higher the correlations between two variables, the closer they are in the space. The space of smallest dimensionality represents an inverse relationship between the observed correlations and the geometric distances. The results of the SSA are shown graphically in Figure 1, based on ecological and disease resistance variables discussed earlier. The following patterns are notable: (1) The climatic factors generated two poles, one involving contiguous factors of water availability (*Rn, Rd, Hu, Th,* see abbreviations in Table 1) on the right hand side, and a second, involving contiguous temperature variables (*Tm, Ta, Tj, Td, Tdd, Trd, Ev*) on the left hand side; (2) The three infection type variables of wheat powdery mildew (IT-1, IT-2, IT-av) are located in proximity to the humidity variables; (3) The three resistant variables (Res-1, Res-2 and Res-av, which are inverse to the infection types) are located in high contiguity at the opposite pole; (4) The axis of leaf rust resistance (Res-LR) is perpendicular to the axis of powdery mildew. This result indicates a correlation of leaf rust resistance with factors of water availability, as mentioned earlier, and low correlation between the resistances to two diseases.

Correlation with allozymes

1. Single allozymes

We used the breakdown programme by Hull and Nie (1981) to assess the association of resistance to *E. graminis tritici* and *P. recondita tritici* with allozymes (for all allozymic data and specifications see Nevo *et al.*, 1982). The mean infection Type (IT) of each allozyme genotype across populations (i.e. regarding all 232 genotypes as one megapopulation) is presented in Table 4. Allozyme genotypes are classified into 'resistant' (IT below average) and 'susceptible' (IT above average), respectively in the table, thereby partioning our data into genotypes displaying association and/or linkage with *tendencies* of resistance and susceptibility. The intra-locus

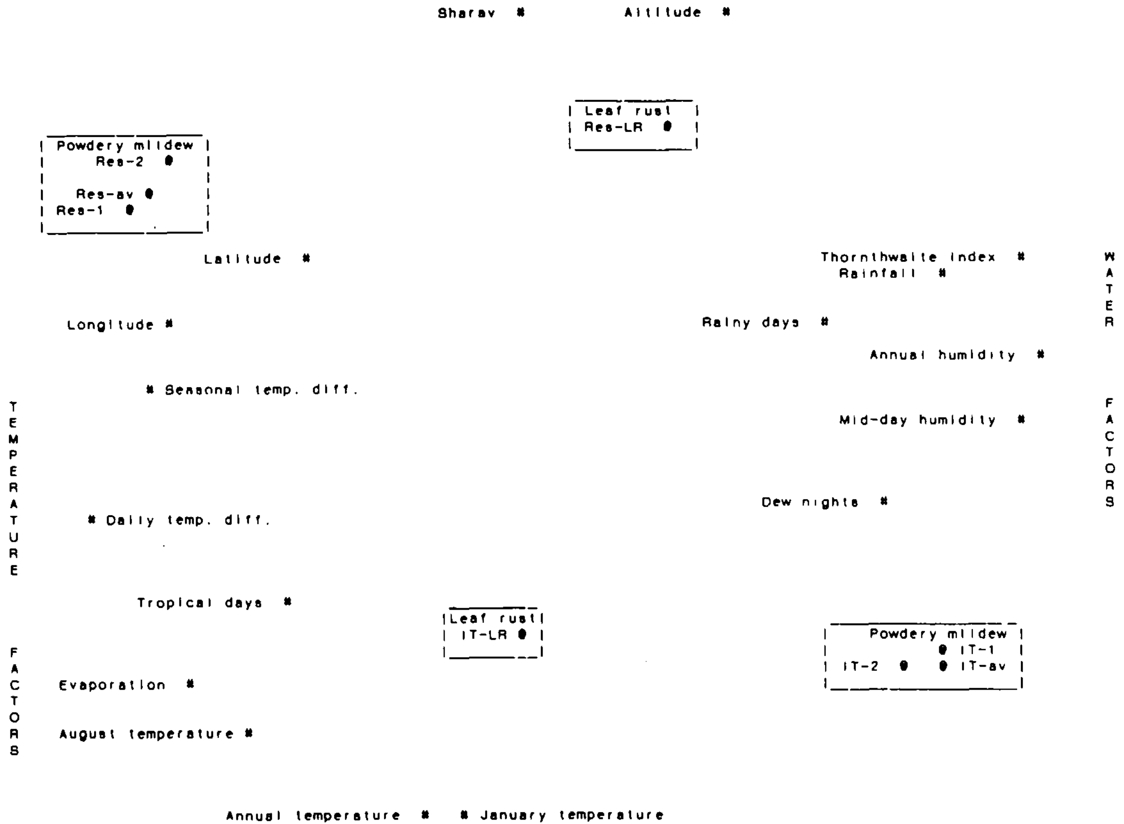

Fig. 1. Smallest Space Analysis (SSA) plotting Spearman correlations among powdery mildew and leaf rust and ecogeographic variables of *Triticum dicoccoides* in Israel. Coefficient of alienation = 0.224. # = Climatic and geographical variables; @ = Disease resistance variables; IT-av = Average infection type by powdery mildew; IT-1 = Mean infection type by cultures Quincy and Mo10 of powdery mildew; IT-2 = Mean infection type by cultures 127 and ABK of powdery mildew; IT-LR = Mean infection type by culture 6 of leaf rust; Res-av = Average resistance to powdery mildew; Res-1 = Mean resistance to cultures Quincy and Mo10 of powdery mildew (9 - IT1); Res-2 = Mean resistance to cultures 127 and ABK of powdery mildew (9 - IT2); Res-LR = Mean resistance to culture 6 of leaf rust (9 - ITLR).

differential among genotypes was tested by ANOVA and the significance is given in the last column of Table 4. For purposes of standardization, we have presented in addition to each individual mean also the average deviation of this genotype from the corresponding mean of its own population, designated 'Standardized IT' and appearing in parentheses. The resistance or susceptibility of the genotype is substantiated if its mean value is in accordance with the standardized IT. For example, a resistant genotype is substantiated if its mean IT is below the overall mean and its standardized IT is negative. This procedure differentiates between genotypes that are associated within the population with more resistance (or more susceptibility) and genotypes that appear in high frequencies in generally resistant (or susceptible) populations. As is obvious from Table 4, some allozyme genotypes are associated with resistance to *E. graminis tritici* (*e.g., Est-2Baa; Acph-1Baa; 6Pgd-2bb; Pgi-Aaa, cc*) and others are associated with susceptibility (*e.g., Ipol-Add; Adh-2Bbb; Est-1Bbb*). The search for resistance to leaf rust was less successful. However, some allozyme genotypes showed an association with relative resistance (*e.g., Est-2Baa; Est-1Abb; Est-2Aaa; Ipol-Acc, dd*).

2. *Multivariate analysis*

(a) *Multiple Regression Analysis (MR).* We con-

Table 4. Classification of allozymes of *Triticum dicoccoides* into 'resistant' (IT below average), and 'susceptible' (IT above average) genotypes.[1]
A. Powdery mildew

Locus	Resistant Genotypes					Susceptible Genotypes					Significance[2]		
	Gt[3]	N	ITav (stITav)	IT1 (stIT1)	IT2 (stIT2)	Gt	N	ITav (stITav)	IT1 (stIT1)	IT2 (stIT2)	ITav	IT1	IT2
Aat-2	aa	15	3.6 (0.18)	3.9 (0.01)	3.3 (0.35)						n.s.	n.s.	n.s.
Acph-1A	bb	18	3.5 (1.07)	3.7 (0.89)	3.3 (1.26)						n.s.	n.s.	n.s.
Acph-1B	aa	4	2.1 (1.11)	2.3 (1.13)	2.0 (1.09)						n.s.	n.s.	n.s.
Acph-2						bb	3	6.2 (0.10)	6.0 (0.75)	6.3 (+0.55)	n.s.	n.s.	n.s.
Acph-3	cc	11	4.0 (1.49)	5.0 (0.94)	3.0 (2.04)	aa	40	5.0 (+0.33)	5.0 (+0.30)	5.0 (+0.36)	n.s.	n.s.	@
Acph-X						aa	18	6.5 (+0.39)	6.4 (0.17)	6.6 (+0.96)	***	**	***
Adh-1A	bb	18	4.6 (0.75)	4.3 (0.80)	4.9 (0.69)						n.s.	n.s.	n.s.
Adh-1B	aa	27	3.6 (+0.23)	3.7 (+0.20)	3.4 (+0.26)						n.s.	n.s.	n.s.
Adh-2A						bb	3	4.8 (0.74)	4.3 (1.13)	5.3 (0.36)	n.s.	n.s.	n.s.
Adh-2B						bb	12	7.1 (+1.36)	6.8 (+1.33)	7.3 (+1.39)	***	**	***
Est-1A	aa	8	2.6 (0.36)	2.5 (0.71)	2.8 (0.01)	bb	1	8.0 (+2.28)	8.0 (+2.50)	8.0 (+2.06)	@	@	@
	cc	161	4.2 (0.21)	4.3 (0.19)	4.0 (0.24)	dd	34	5.1 (+1.11)	5.1 (+1.11)	5.1 (+1.12)			
						ff	21	5.1 (+0.06)	5.0 (0.04)	5.1 (+0.17)			
Est-1B	dd	1	1.0 (3.56)	1.0 (3.22)	1.0 (3.89)	bb	15	7.4 (+1.30)	7.1 (+0.61)	7.6 (+2.00)	***	***	***
	cc	2	1.8 (1.49)	2.0 (1.38)	1.5 (1.59)								
Est-2A	ff	4	3.8 (2.57)	4.0 (2.52)	3.5 (2.62)	aa	15	5.5 (0.27)	5.3 (0.78)	5.8 (+0.24)	n.s.	n.s.	n.s.
						bb	7	5.4 (0.45)	5.0 (0.94)	5.7 (+0.04)			
Est-2B	aa	4	2.1 (3.60)	1.8 (3.75)	2.5 (3.44)	cc	79	4.5 (+0.27)	4.7 (+0.23)	4.3 (+0.31)	n.s.	n.s.	n.s.
	dd	94	4.2 (+0.24)	4.2 (+0.19)	4.2 (+0.29)								
	bb	51	4.3 (0.57)	4.5 (0.51)	4.2 (0.63)								
Est-4A	bb	3	3.7 (+0.39)	4.0 (+0.64)	3.3 (+0.14)						n.s.	n.s.	n.s.
Est-4B	bb	1	1.5 (0.64)	2.0 (0.54)	1.0 (0.75)	aa	138	4.6 (+0.08)	4.7 (+0.05)	4.4 (+0.10)	@	n.s.	n.s.
	dd	3	2.3 (0.90)	2.7 (0.72)	2.0 (1.09)								
	cc	74	3.8 (0.38)	3.9 (0.28)	3.6 (0.47)								
Est-5A	bb	43	3.4 (+0.51)	3.6 (+0.40)	3.3 (+0.62)						**	**	*
Est-5B	cc	43	3.3 (+0.43)	3.4 (+0.25)	3.3 (+0.62)	dd	1	8.0 (+5.86)	8.0 (+5.46)	8.0 (+6.25)	***	***	***
	aa	123	4.2 (0.19)	4.4 (0.07)	4.0 (0.31)	gg	1	8.0 (+2.46)	8.0 (+3.00)	8.0 (+1.93)			
						bb	20	7.0 (+0.57)	7.0 (+0.22)	7.1 (+0.92)			
						ee	18	5.4 (0.29)	5.3 (0.18)	5.4 (0.40)			
Gdh-B	bb	6	3.2 (0.56)	3.3 (0.59)	3.0 (0.53)						n.s.	n.s.	n.s.
Gluc	bb	127	4.0 (0.16)	4.2 (0.08)	3.8 (0.24)	cc	52	5.4 (+0.28)	5.4 (+0.14)	5.4 (+0.42)	*	@	**
						aa	6	5.3 (+1.33)	5.2 (+1.25)	5.5 (+1.42)			
Hk						bb	42	4.8 (0.02)	4.8 (0.03)	4.8 (0.01)	@	n.s.	*
Ipol-A	cc	25	1.8 (0.05)	2.0 (0.02)	1.6 (0.09)	dd	10	7.7 (+1.61)	7.6 (+1.10)	7.8 (+2.11)	***	***	***
Ipor-B						bb	15	6.5 (0.01)	6.9 (+0.18)	6.1 (0.19)	**	**	*
Lap	bb	5	4.1 (1.44)	3.4 (1.60)	4.8 (1.27)	cc	7	6.8 (+1.39)	6.4 (+1.54)	7.1 (+1.24)	*	@	*
Mdh-1A						aa	42	5.1 (+0.14)	5.0 (+0.15)	5.1 (+0.12)	@	@	@
Nadh-1A	aa	7	2.4 (+0.29)	2.4 (0.11)	2.4 (+0.68)						n.s.	n.s.	n.s.
	cc	49	3.9 (+0.07)	4.1 (+0.14)	3.8 (0.01)								
Nadh-1B	bb	10	2.3 (+0.16)	2.5 (0.04)	2.1 (+0.35)						*	@	@
Nadh-2	bb	3	1.8 (0.22)	2.0 (0.38)	1.7 (0.06)						n.s.	n.s.	n.s.
Pept-1B	bb	180	4.0 (+0.02)	4.2 (+0.05)	3.9 (0.02)	aa	46	5.2 (0.15)	5.2 (0.28)	5.2 (0.01)	*	@	*
Pept-2	cc	29	3.6 (+0.09)	3.8 (+0.06)	3.5 (+0.12)	aa	8	6.3 (+0.71)	5.6 (+0.63)	6.9 (+0.80)	@	n.s.	*
Pgl-A	aa	25	2.5 (0.20)	3.0 (0.01)	2.0 (0.38)						***	**	***
	cc	21	3.2 (0.19)	3.2 (0.27)	3.1 (0.12)								
Pgm-A	aa	1	3.0 (+1.13)	3.0 (+0.94)	3.0 (+1.31)						n.s.	n.s.	n.s.
6Pgd-1B						bb	2	8.0 (+2.46)	8.0 (+3.00)	8.0 (+1.93)	@	@	@
6Pgd-2	bb	6	1.8 (0.39)	2.0 (0.54)	1.5 (0.25)	cc	33	5.1 (+0.32)	5.0 (+0.23)	5.2 (+0.42)	*	@	*

B. Leaf Rust

Locus	Resistant genotypes			Susceptible genotyp.			Significance
	Gt	N	ITLR (stITLR)	Gt	N	ITLR (stITLR)	
Aat-2				aa	15	7.7 (0.15)	n.s.
Acph-1A				bb	18	7.8 (0.14)	@
Acph-1B				aa	4	7.5 (+0.12)	n.s.
Acph-2				bb	3	8.0 (+0.72)	n.s.
Acph-3				aa	40	7.9 (0.02)	***
				cc	11	7.8 (+0.52)	
Acph-X	aa	18	7.4 (+0.05)				n.s.
Adh-1A				bb	18	7.9 (+0.23)	*
Adh-1B	aa	27	7.4 (0.01)				n.s.
Adh-2A				bb	3	8.0 (0.00)	n.s.
Adh-2B	bb	12	6.8 (+0.42)				**
Est-1A	bb	1	5.0 (1.38)	aa	8	7.8 (+0.10)	*
	ff	21	7.1 (0.58)	dd	34	7.7 (+0.03)	
Est-1B	bb	15	7.0 (0.29)	dd	1	8.0 (0.00)	n.s.
				cc	2	7.5 (+0.12)	
Est-2A	aa	15	6.6 (0.48)	ff	4	7.5 (0.12)	*
				bb	7	7.4 (-0.03)	
Est-2B	aa	4	5.5 (0.83)	bb	51	7.7 (0.05)	***
	dd	94	7.2 (+0.07)	cc	79	7.6 (0.03)	
Est-4A				bb	3	8.0 (+0.21)	n.s.
Est-4B	aa	138	7.2 (0.04)	bb	1	8.0 (+0.11)	***
				cc	74	7.8 (+0.04)	
				dd	3	7.7 (+0.28)	
Est-5A	bb	43	7.0 (0.00)				**
Est-5B	ee	18	6.8 (+0.17)	gg	1	8.0 (+0.43)	***
	dd	1	7.0 (0.89)	aa	123	7.7 (0.03)	
	cc	43	7.0 (+0.05)	bb	20	7.5 (0.01)	
Gdh-B				bb	6	7.8 (+0.45)	n.s.
Gluc	bb	127	7.3 (0.03)	cc	52	7.8 (+0.01)	*
				aa	6	7.7 (+0.22)	
Hk	aa	171	7.3 (+0.01)	bb	42	8.0 (0.00)	***
Ipol-A	cc	25	6.8 (0.09)				***
	dd	10	6.9 (0.45)				
Ipor-B				bb	15	7.5 (-0.06)	n.s.
Lap	cc	7	7.3 (0.35)	bb	5	8.0 (+0.43)	n.s.
Mdh-1A	bb	164	7.3 (0.01)	aa	42	8.0 (+0.03)	***
Nadh-1A	cc	49	7.3 (+0.09)	aa	7	7.9 (-0.04)	n.s.
Nadh-1B				bb	10	7.8 (-0.09)	n.s.
Nadh-2	bb	3	7.3 (0.22)				n.s.
Pept-1B	bb	180	7.3 (-0.02)	aa	46	7.8 (+0.04)	**
Pept-2	cc	29	7.3 (0.06)				n.s.
	aa	8	7.4 (0.20)				
Pgi-A	cc	21	7.3 (0.11)	aa	25	7.9 (+0.08)	*
Pgm-A	aa	1	7.0 (+0.11)				n.s.
6Pgd-1B	bb	2	7.0 (0.57)				n.s.
6Pgd-2				cc	33	7.9 (0.04)	**
				bb	6	7.8 (0.06)	

[1] The body of the table does not include all major genotypes, which do not deviate from the mean because of their high frequency.

[2] Significance is calculated by ANOVA, based on the differences between genotypes within a locus. Symbols: @ = $p < 0.10$; * = $p < 0.05$; ** = $p < 0.01$; *** = $p < 0.001$; n.s. = $p > 0.10$;

[3] Abbreviations: Gt = Genotypes; ITav = Average infection type by powdery mildew; stITav = Standardized average infection type (mean deviation from local mean); IT1 = Mean infection type by cultures Quincy, Mo10 of powdery mildew, stIT1 = Standardized IT1 (mean deviation from local mean); IT2 = Mean infection type by cultures 127, ABK of powdery mildew; stIT2 = Standardized IT2 (mean deviation from local mean); ITLR = Mean infection type by culture 6 of leaf rust; stITLR = Standardized ITLR (mean deviation from local mean); *Aat* = Aspartate amino transferase; *Acph* = Acid phosphate; *Adh* = Alochol dehydrogenase; *Est* = Esterase; *Gdh* = Glutamate dehydrogenase; *Gp* = General protein; *Gluc* = Glucosidase; *Hk* = Hexokinase, *Ipol* = Indophenol oxidase in leaf; *Ipor* = Indophenol oxidase in root; *Lap* = Leucine amino peptidase; *Mdh* = Malate dehydrogenase; *Nadh* = Lipoamide diaphorase; *Pept* = Peptidase; *Pgi* = Phosphoglucose isomerase; *Pgm* = Phosphogluco mutase; *6 Pgd* = 6-Phospho gluconate dehydrogenase.

Table 5. Coefficients of multiple determination (R^2) with dependent variables of resistance to powdery mildew (IT-1, IT-2 and IT-av) and to leaf rust (IT-LR) and as independent variables allozyme genotypes* of wild wheat, *Triticum dicoccoides*, in Israel.

Stepwise model				
A. Powdery mildew (N = 126)				
Average resistance	*Ipol-Acc*	+ *Pgi-Acc*	+ *Acph-1Abb*	+ *Gluc-bb*
(IT-av)	0.09 ***	0.17 ***	0.26 ***	0.32 ***
Resistance to comp. 1	*Ipol.Acc*	+ *Pgi-Acc*	+ *Acph-1Abb*	+ *Gluc-bb*
(IT-1)	0.08 **	0.17 ***	0.25 ***	0.29 ***
Resistance to comp. 2	*Ipol-Acc*	+ *Pgi-Acc*	+ *Acph-1Abb*	+ *Gluc-bb*
(IT-2)	0.09 ***	0.14 ***	0.22 ***	0.29 ***
B. Leaf rust (N = 210)				
Resistance to cult. 6	*Est-2Baa*	+ *Ipol-Acc*	+ *Est-1Aff*	+ *Est-1Bbb*
(IT-LR)	0.10 ***	0.12 ***	0.14 ***	0.15 ***

Level of significance: ** = $p < 0.01$; *** = $p < 0.001$;

* Genotypes included in the analysis: Aspartate amino transferase: *Aat-2aa*; Acid phosphatase: *Acph-1Abb*, *Acph-1Baa*, *Acph-3bb*; Esterases: *Est-1Aaa, ff*, *Est-1Bbb*, *Est-2Aaa, ff*, *Est-2Baa*, *Est-4Bcc, dd*; Glucosidase: *Gluc-bb*; Glutamate dehydrogenase: *Gdh-Bbb*; Indophenol oxidase in leaf: *Ipol-Acc, dd*; Lipamide diaphorase: *Nadh-2bb*; Phosphoglucose isomerase: *Pgi-Aaa, cc*; 6-Phospho gluco dehydrogenase: *6Pgd-2bb*.

ducted a stepwise multiple regression analysis (Hull & Nie, 1981) employing resistance values as dependent variables and allozyme genotypes as independent variables (Table 5), in an attempt to predict multilocus genotypes resistant to powdery mildew and leaf rust by a combination of allozyme variants.

A multilocus genotype consisting of 3 genotypes (*Ipol-Acc, Pgi-Acc, Acph-1Abb*) best predicts resistance to powdery mildew, and another multilocus genotype (*Est-2Baa, Ipol-Acc, Est-1Aff*) best predicts resistance to leaf rust.

(b) *Smallest Space Analysis (SSA).* The multivariate association between allozyme markers and resistance to powdery mildew and leaf rust is displayed by two SSA diagrams (Figs. 2 and 3, respectively). Fifteen allozyme genotypes that proved highly resistant to powdery mildew in the previous analysis appear in the 'resistant region' of the SSA diagram near the resistant variables. These genotypes include the following: *Aat-2aa; Acph-1Abb; Acph-1Baa; Est-1Aaa; Est-2Aff; Est-2Baa; Est-4Bdd; Est-5Baa; Gdh-Bbb; Gluc-bb; Ipol-Acc; Nadh-2bb; Pgi-Aaa, cc; 6-Pgd-2bb.* By contrast, the following genotypes appear in the 'susceptible region' (Near the IT variables): *Est-1Bbb; Est-5Bbb; Ipol-Add; Lap-cc.* The five allozyme genotypes that showed in the previous analysis (Table 4) lower susceptibility to leaf rust appear in the SSA diagram (Fig. 3) in the 'resistant region': *Est-1Bbb; Est-2Aaa; Est-2Baa; Ipol-Acc, dd.* Contrariwise, three genotypes appear in the 'susceptible region': *Adh-1Abb; Est-1Add; Est-4Bcc.* The associations displayed in the two SSA diagrams between allozyme markers and resistance represent Spearman rank correlations. The larger the distance between variables on the SSA diagram, the lower the correlation. Very large distances represent negative Spearman correlations, whereas high proximity between variables indicates positive and high correlation. Thus, allozyme markers individually, or in combination, may be useful guidelines for identifying disease resistant genotypes.

Discussion

Our results suggest that resistance of tetraploid wild wheat, *T. dicoccoides*, in Israel, to powdery mildew and leaf rust displays (a) ecogeographic variation, and (b) association with allozyme markers. Both results provide guidelines for better sampling these resources for use in wheat breeding programmes and explain the maintenance of genetic polymorphism. We will discuss these aspects and underline the practical application.

1. Ecogeographic variation of resistance to powdery mildew and leaf rust of wild wheat in Israel

Our results indicate that the resistance to *E. graminis tritici* and to *P. recondita tritici* is geographically structured and ecologically correlated. Resist-

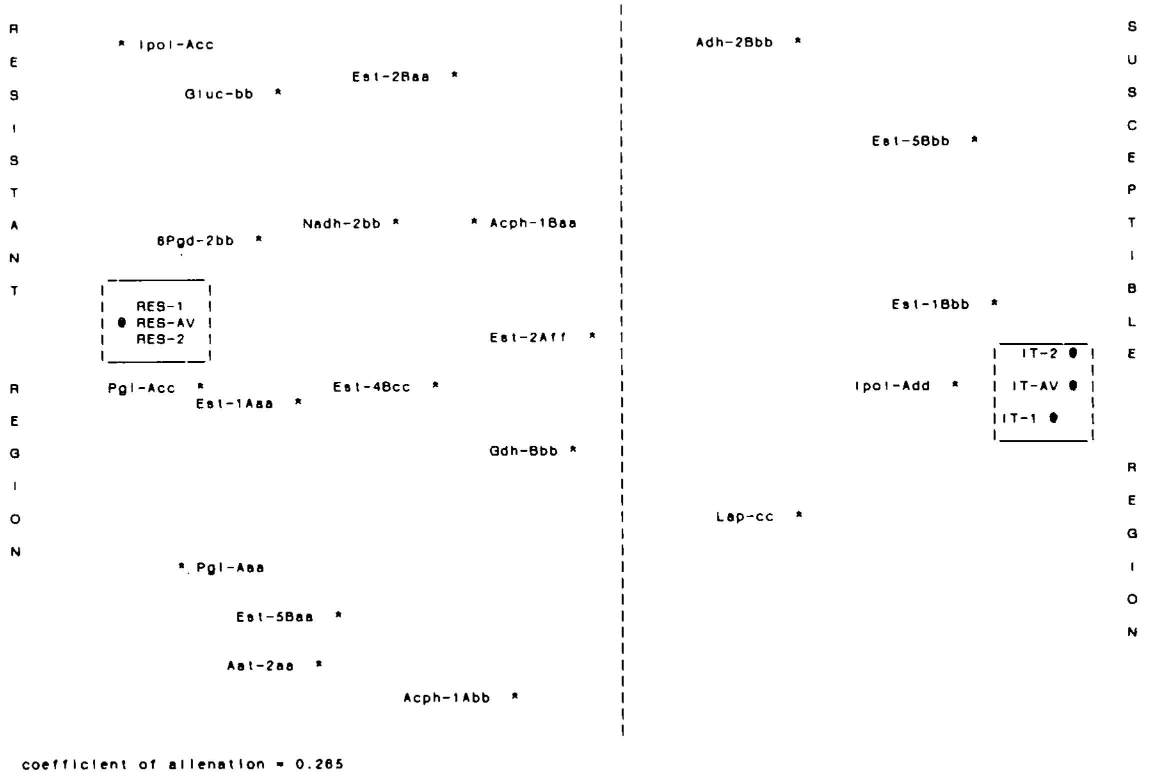

Fig. 2. Smallest Space Analysis (SSA) plotting Spearman correlations among disease resistance variables to powdery mildew and 20 allozyme markers of *Triticum dicoccoides* in Israel. * = Allozyme genotypes, @ = Disease resistance variables; IT-av = Average infection type by powdery mildew; IT-1 = Mean infection type by cultures Quincy and Mo10 of powdery mildew; IT-2 = Mean infection type by cultures 127 and ABK of powdery mildew; Res-av = Average resistance to powdery mildew; Res-1 = Mean resistance to cultures Quincy and Mo10 of powdery mildew (9 - IT1); Res-2 = Mean resistance to cultures 127 and ABK of powdery mildew (9 - IT2).

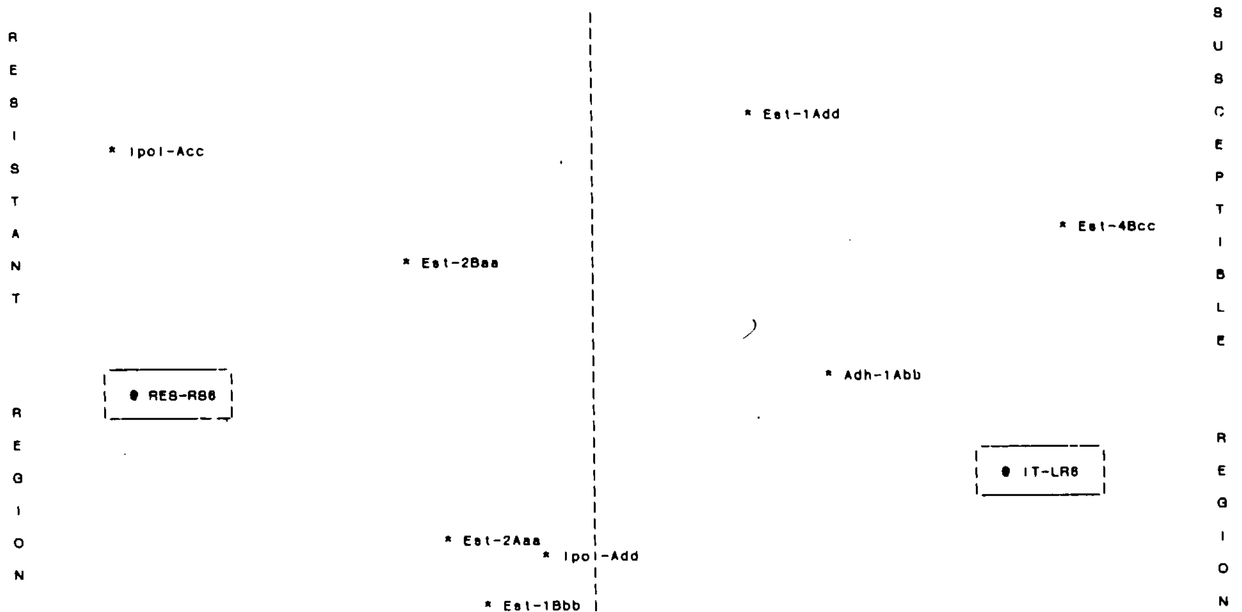

Fig. 3. Smallest Space Analysis (SSA) plotting Spearman correlations among disease resistance variables to leaf rust and 8 allozyme markers of *Triticum dicoccoides* in Israel. Coefficient of alienation = 0.125. * = Allozyme genotypes; @ = Disease resistance variables; IT-LR6 = Mean infection type by culture 6 of leaf rust; Res-RS6 = Mean resistance to culture 6 of leaf rust (9 - ITLR).

ance to powdery mildew appears to have coevolved with the pathogen. Resistance to powdery mildew appears higher in largely drier environments, both northward and eastward, characterized by high seasonal and daily temperature differentials. The most resistant populations to powdery mildew are Qazrin and Rosh Pinna, where *T. dicoccoides* appears to be at its ecological climax, as indicated by the highly dense and productive populations. While marginal populations of *T. dicoccoides* are sparse and isolated, these central populations are continuous and extensive in distribution. Similar, but not identical results, were obtained for the resistance of wild barley, *Hordeum spontaneum*, to powdery mildew (Moseman *et al.*, 1983; Nevo *et al.*, 1984a). For both cereal species the highest resistance to the pathogen was found in ecologically optimal regimes of the species range. This pattern suggests the ecogeographical regions that are most suited for sampling wild germplasm for improving resistance to the powdery mildew pathogens. Furthermore, since even resistant populations proved polymorphic to pathogen resistance (Moseman *et al.*, 1984a), not only sites, but also within sites, specific genotypes must be screened through their allozymic markers. To date 10 genes of resistance against powdery mildew are known (see Moseman *et al.*, 1984a). However, to increase the number of resistant genes, as well as, the range of new alleles in the already known loci, additional wild emmer wheat populations and additional allozyme markers should be used.

The search for resistance to leaf rust is more difficult because most populations and genotypes tested were susceptible to the pathogen. Nevertheless, ecological correlates and allozyme markers may, in principle, provide effective guidelines. While one peripheral population (Bet Meir) contained moderate resistant genotypes, the second central population (Qazrin) was also the population displaying the highest resistance to powdery mildew.

2. Allozyme markers for pathogen resistance and its application

Linkage of genetic factors controlling or associated with quantitative variation was reviewed by Thompson and Thoday (1979) following the technique by Thoday (1961). As suggested by Tanksley *et al.* (1982) in their use of naturally occurring enzyme variations to detect and map genes controlling quantitative traits in tomato, it would be desirable to have a large number of marker genes distributed throughout the genome with codominant alleles. The alleles of these marker genes should be clearly identifiable and produce little or no effect on the morphology of the organism. Enzyme marker genes are ideal candidates for such a study as was shown in *Cyprinus carpio* (Brody *et al.*, 1976). *Lycopersicon* (Tanksley *et al.*, 1982), and in *Hordeum spontaneum* (Nevo *et al.*, 1984a).

We have shown that both resistant and susceptible wild emmer wheat genotypes are associated with single allozymes or combinations of allozyme variants, and that the genetic polymorphism of disease resistance is partly associated with some allozyme polymorphism. Guided by the previous knowledge of the geographic variation in allozyme frequency (Nevo *et al.*, 1982), such associations may be utilized in quick screening of numerous wild wheat plants to choose the promising candidates for disease resistance tests. Furthermore, a multilocus profile of allozyme markers could be searched covering a maximally broad resistance to many diseases, combining them with elite agronomic traits to avoid yield depression in breeding (Nevo *et al.*, 1984c).

Since Israel is the center of diversity of *Hordeum spontaneum* and of *Triticum dicoccoides*, the genotypic arrays discovered in this region are optimal for searching multilocus genotypes with the broadest possible spectrum of disease resistance (Nevo *et al.*, 1984a). If these ideal genotypes are not identified from natural populations, they could be produced by specific crossing programmes, and later tested under multiple pathogen infections, first in the laboratory and then in the field. Finally, the rich allozymic diversity of wild emmer wheat (Nevo *et al.*, 1982) can be applied successfully in breeding highly resistant cultivars that maintain their economic status without yield depression.

As concluded by Brown and Clegg (1983). 'The genetic resources of cultivated plants consist of the genetic diversity present among developed cultivars, in the land races of primitive stocks, and in wild populations of their evolutionary relatives. Genetic evaluation of these resources through isozyme studies provides an important aid in efficient collection, storage, and use'. It is obvious that 'There are

definite limits to the numbers of samples which can be handled effectively in programmes for the conservation and utilization of crop genetic resources. Consequently, there is a need for the coordinated and systematic planning of conservation programmes to ensure the preservation of the maximum amount of useful genetic variability while keeping the total number of samples within these limits'. (Marshall & Brown, 1975). Following the above mentioned arguments, we strongly believe that (i) a predictive method is required for screening disease resistant genotypes in nature, and (ii) that the method suggested here is more efficient than the empirical methods currently used in sampling as well as in testing for resistance. The main reason is that if our method is indeed effective, it might contribute both to optimize sampling, and in identifying (and/or producing by crossings) multilocus genotypes that are resistant to *multiple* diseases and are also *agronomically superb*. Finally, (iii) association of allozyme markers with disease resistance may help us to discover *new genes and/or alleles* of resistance.

3. The maintenance of allozyme and disease resistance polymorphism

Parasites were first suggested by Haldane (1949) to be an evolutionary force which could maintain polymorphism in their hosts. The host-parasite evolutionary interactions can actively maintain genetic polymorphism in both parasite and host (Clarke, 1976). Furthermore, the large amounts of genetic diversity within natural populations at the protein and DNA levels may be explained by various forces including frequency and density dependent selection by parasites (Clarke, 1976, 1979), and by spatiotemporal heterogeneity (Nevo, 1983; Nevo *et al.*, 1984b). The theory of host-parasite coevolution (Dietel, 1974) has been extended by many workers (i.e., Anikster & Wahl, 1979). Moseman (1959) has shown that for each gene conditioning the resistance of the barley host there is a corresponding gene conditioning the virulence of the pathogen *E. graminis hordei*. Therefore, the evolutionary dynamics of the disease resistance polymorphism may be directly related to pathogen polymorphism. Indirectly, both allozyme and disease resistance polymorphisms appear to be selected by the physical (climatic) and biotic environment. Unfortunately, the direct correlation between disease occurrence and resistance can not be evaluated at the present time due to lack of pertinent information, and the sporadic nature of epidemics.

The polymorphisms of allozymes (Nevo *et al.*, 1982) and disease resistance (Moseman *et al.*, 1983a, b, 1984) in Israel are both widespread and are significantly correlated with the climatic environment. Both polymorphisms are ecologically predictable by a combination of humidity and temperature variables. Thus, the coefficient of multiple determination (R^2) of two variable combinations (of humidity and rainfall) significantly explains 0.60 of the variance in allozyme polymorphism, and a 4-variable combination including evaporation, soil, latitude, and temperature significantly explains 0.75 of the variance in expected allozyme heterozygosity, *He*, (Nevo *et al.*, 1982). In parallel, a 3-variable combination of humidity, evaporation and temperature differences explains about a fourth of the variance in resistance to powdery mildew. Both polymorphisms are highest in mild, ecologically more heterogeneous climates, rather than in marginal extreme climates, in accord with the niche-width variation hypothesis (Van Valen, 1965) and with the theoretical prediction of the multilocus theory (Karlin, 1981).

Presumably, disease resistance polymorphisms respond to the extensive pathogen variation in its ecologically optimal and heterogeneous environment. Since allozymes are involved in resistance, either indirectly (e.g. as markers) or even directly (although to date there is no supportive evidence), as suggested by the associations found here, then the maintenance of allozyme polymorphisms may be reinforced by the pathogen-host interaction. Therefore, natural selection must be involved in their pattern and differentiation.

Conclusions and prospects

Wild relatives of crops have been especially useful to breeders searching for sources of disease resistance (for examples and review see Plucknett *et al.*, 1983; for barley see Moseman *et al.*, 1983; and for wheat Moseman 1984a, b). Several disease resistance genes have already been, or are currently being transferred from *T. dicoccoides* to cultivated wheats including stripe and leaf rusts. There must be many

sources of resistance to each pathogen and each source is effective against different virulent types of the pathogen. Therefore, multiple sources of disease resistance must be used in association with agronomically elite genotypes to increase and stabilize wheat productivity. We have outlined ecological and allozymic predictive strategies which could optimize the selection of disease resistant wheat germplasm by utilizing diverse, as well as highly resistant sources in natural populations of tetraploid wild wheat in Israel.

T. dicoccoides is uniquely suitable for selecting diverse sources of resistance to many disease inciting pathogens. Its coevolution with pathogens in the Fertile Crescent at large, and in Israel, which is its center of diversity, in particular, has been proceeding for millions of years. This coevolution generated many sources of resistance against powdery mildew, rusts, and other pathogens, that have not been fully exploited. The predictive value of ecological factors and allozyme markers can greatly help in maximizing the conservation, exploitation, and utilization of the rich genetic resources of *T. dicoccoides* for improving the cultivated germplasm of wheats.

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