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The ability of a pathogen to spread in a crop influences the progress of an epidemic, depending on the effect of environment. Investigations on the use of this phenomenon to estimate quantitative resistance differences of cereals were reported by some authors as Barabas et al. (1974), Mackenzie (1976) and Matuz et al. (1979) on *Puccinia graminis tritici* and Freidt et al. (1979) on *Erysiphe graminis* in wheat, and Berger and Luke (1979) on *Puccinia coronata*. The methods reported must be estimated differently, and further research

INTRODUCTION

Infection gradients in eight varieties and breeding lines of spring barley were estimated and compared. The gradient obtained by regression analysis of the function $\log y = \log a + b \cdot \log x$ showed a trend to a greater slope with increasing resistance. Time and work saving disease assessment methods are suggested and discussed. An early infection by *Puccinia hordei* significantly decreased the thousand kernel weight (by 18.5%) and the yield (by 20.7%) only on the susceptible variety L94.

SUMMARY

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BY

THE SPREAD OF *Puccinia hordei* OTH. IN VARIETIES AND BREEDING LINES OF SPRING BARLEY WITH DIFFERENT RACE - NONSPECIFIC RESISTANCE

will be necessary on other host parasite combinations for effective use in breeding.

MATERIAL AND METHODS

Breeding lines and varieties without known resistance genes (Hartleb et al. 1983) were used for the investigations; L94 (very susceptible), Mirrena and Vada, and the lines St.32, St.116, St.115 and St.96 (moderately susceptible).

The field experiments were performed in a 4 replicate block design. The size of each plot was 1m x 30m, drilled in east-west-direction (west = main wind direction). One metre strips of oats for isolation were drilled between the plots, and along the west side of the plots five spreader rows of the very susceptible variety L94 were drilled. For artificial infection with the compatible race, R 23-4280 (Trumpf virulence), vernalised winter barley was cultivated in 15cm plots and infected in the glasshouse.

Prior to sporulation winter barley was planted on the westside of the plots when the spring barley was at growth stage 4 to 5 (Reekes scale).

The spread of *P. hordei* in the plots was estimated on the following sections, depending on the disease progress.

1st and 2nd week of assessment: 4 sections of 50cm.
3rd week of assessment: 4 sections of 100cm.
4th and 5th week of assessment: 5 sections of 100cm.
6th week of assessment: 6 sections of 200cm.
7th week of assessment: 7 sections of 200cm.

At each assessment in each section, the pustules where counted on the lower non-yellowed leaves from the same evaluation stage. As the mathematical gradients for the varieties and breeding lines are not comparable (fig.1), the equation $\log y = \log a + b \cdot \log x$ was used (Gregory 1968). The slopes of the straight lines, obtained after regression analysis, the squares of deviation and the derivation products where compared with the following t-test at $P=5\%$; $DF = N_1 + N_2 - 4$.

In order to evaluate the influence of infection by *Puccinia hordei* on the yield of spring barley every first and last section of the plots ($2m^2$) was harvested and weighed (4

replicates) and the thousand kernel weight estimated. The results were calculated by variance analysis.

RESULTS

With the exception of L94 the speed of progress of the disease in all varieties and breeding lines was almost equal, with differences in the disease severity from section to section. The whole plot of L94 showed infection on June 29th, whilst in the other varieties/lines the pathogen had advanced only fourteen metres (7th section in the last week of assessment). Beyond this section only traces of \bar{P} . hordei were found at the end of the vegetation period.

Figure 1 shows the different slopes of the gradients of five selected varieties and breeding lines. The comparison of the straight lines found by regression analysis after log-transformation depended on the results from the assessment of date. Thus for instance on June 14th significant differences were observed between L94 and St.32, Vada, St. 15, and St.96 and between St.32 and St.15, Vada and St.96 (Fig.2). On the 5th July no differences could be found (Fig.3).

Gregory (1968) noted that the slope b is suitable for characterization of the infection gradients. The equation $\log y = \log a + b \cdot \log x$ gives negative values for b after the regression analysis. On each assessment date a gradient of infection was found for the eight varieties and breeding lines. The average of the b -values over all the assessment dates shows a trend to a greater slope with increasing resistance (table 1).

Table 1: Comparison of the slope of the gradient of infection (b) with the area under the disease progress curve (A).

Varities	b	A
Breeding material		
L94	-1.31	184.0
St.32	-1.74	33.1
Mirena	-1.58	25.3

As a result of early infection all varieties suffered yield losses, possibly due to a reduction in thousand kernel weight (table 3).
 Table 3: Influence of an early infection by *Puccinia hordei* on the thousand kernel weight and the grain yield of seven resistant varieties and breeding lines of spring barley.

Assessment date	Disease in % ⁺	Slope b of the infection gradient ⁺
25 May	1.1	-2.53
1 June	2.2	-1.85
8 June	2.5	-2.65
14 June	4.4	-2.42
22 June	10.7	-1.62
29 June	16.6	-1.35
5 July	19.4	-0.99

Table 2: Relations between assessment date, disease severity and slope of infection gradient.

In plots of *Mirena* and *St.116*, *Puccinia hordei* spread better (lower b-values) than in plots of *St.32*, although *St.32* has a lower resistance (higher A-value). Due to such differences the correlation between b and A from all of the investigated varieties and breeding lines is not significant ($r = 0.61$).
 An expected flattening of the gradient was seen with increasing disease severity and in later assessments. (Table 2).

<i>St.116</i>	-1.58	18.6
<i>St.123</i>	-1.72	14.8
<i>St.15</i>	-2.25	13.5
<i>Vada</i>	-2.40	12.7
<i>St.96</i>	-2.79	2.4

Varities and breeding lines differ in their ability to influence the spread of pathogen. To use this ability in breeding systems with preselcted material the number of sections assessed is too large. In attempts to reduce the number of sections assessed it was found that the rank order between the first and second section was already fixed. This could clearly be demonstrated when the results from the first section are put equal to 100 and those from the other sections were expressed relative to this value (fig. 4). The relative value of the second section correlates to 0.91 at $P = 1\%$ significantly with the b-values found by analysis of the results from seven sections of each variety or breeding line after regression analysis.

The use of logarithmic values for the disease (number of pustules) and the distance (cm) for the regression analysis resulted in a high coefficient of determination that could be confirmed by the t-test in most cases. The logit-transformation of the relative disease severity recommended by Berger and Luke (1979) on *Puccinia coronata* was not as favourable as the log - transformation.

DISCUSSION OF THE RESULTS

+ significantly different according to t - test ($P = 0.5\%$) between the results from the first and the fifteenth section.

Varities	Thousand Kernel	yield g	breeding material	
			Section 1	Section 15
L94	38.80	47.74 ⁺	352.5	445.0 ⁺
St.33	40.40	41.44	850.0	980.0
Mirena	40.64	41.16	850.0	915.0
St.116	32.09	35.80	695.0	847.5
St.123	38.05	39.85	880.0	892.5
St.15	42.60	44.83	975.0	1022.5
Vada	39.01	40.90	725.0	842.5

These results were obtained in 1982 when there was no natural infection pressure by Puccinia hordei. If this pressure exists, an early and large amount of spores from the infection source is necessary to delay a flattening of the gradient.

On the east side of the plots no infection could be obtained, except on the standard variety L94. Therefore section fifteen was compared with the first section. Only the highly susceptible variety L94 showed a reduction in yield and thousand kernel-weight, 20.7% and 18.5% respectively. A disease value of 100% was observed on the standard variety L94 in the first section by the 29th of June leading to a premature loss of leaf area and thus assimilation. In all other varieties the influence of infection could not be confirmed statistically. Increasing resistance led to a decreasing influence on thousand kernel-weight ($r = 0.84$) and yield ($r = 0.63$).

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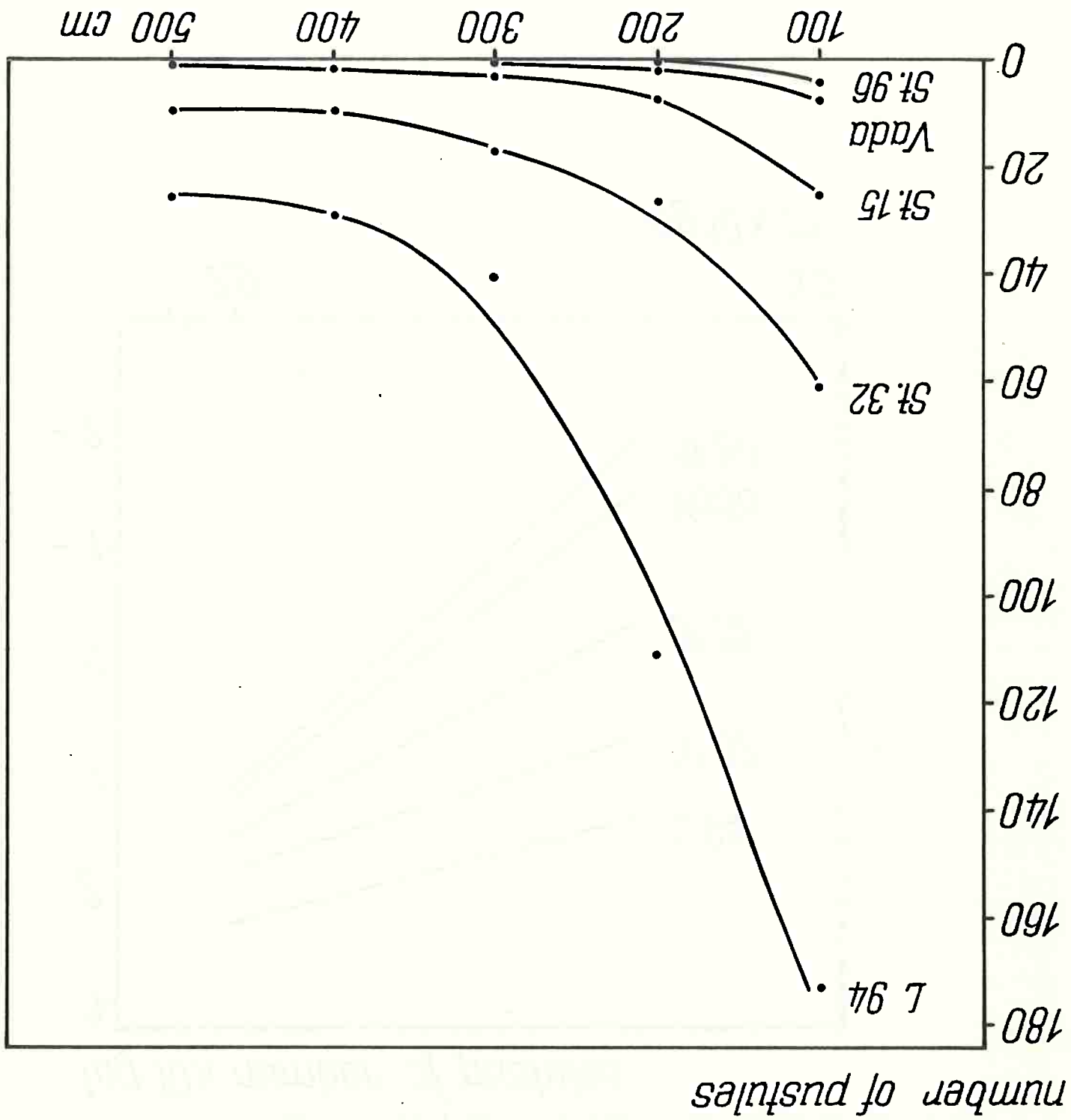


Figure 1: Infection gradients of brown rust, caused by *Puccinia hordei* in spring barley. Fourth assessment week. Abscissa: Distance from the infection source in cm. Ordinate: Number of pustules per leaf.

Figure 2: Infection gradients of brown rust, caused by *Puccinia hordei* in spring barley, as in figure 1, after linear regression analysis.
 Abscissa: Logarithm from infection source in cm.
 Ordinate: Logarithm from number of pustules per leaf.

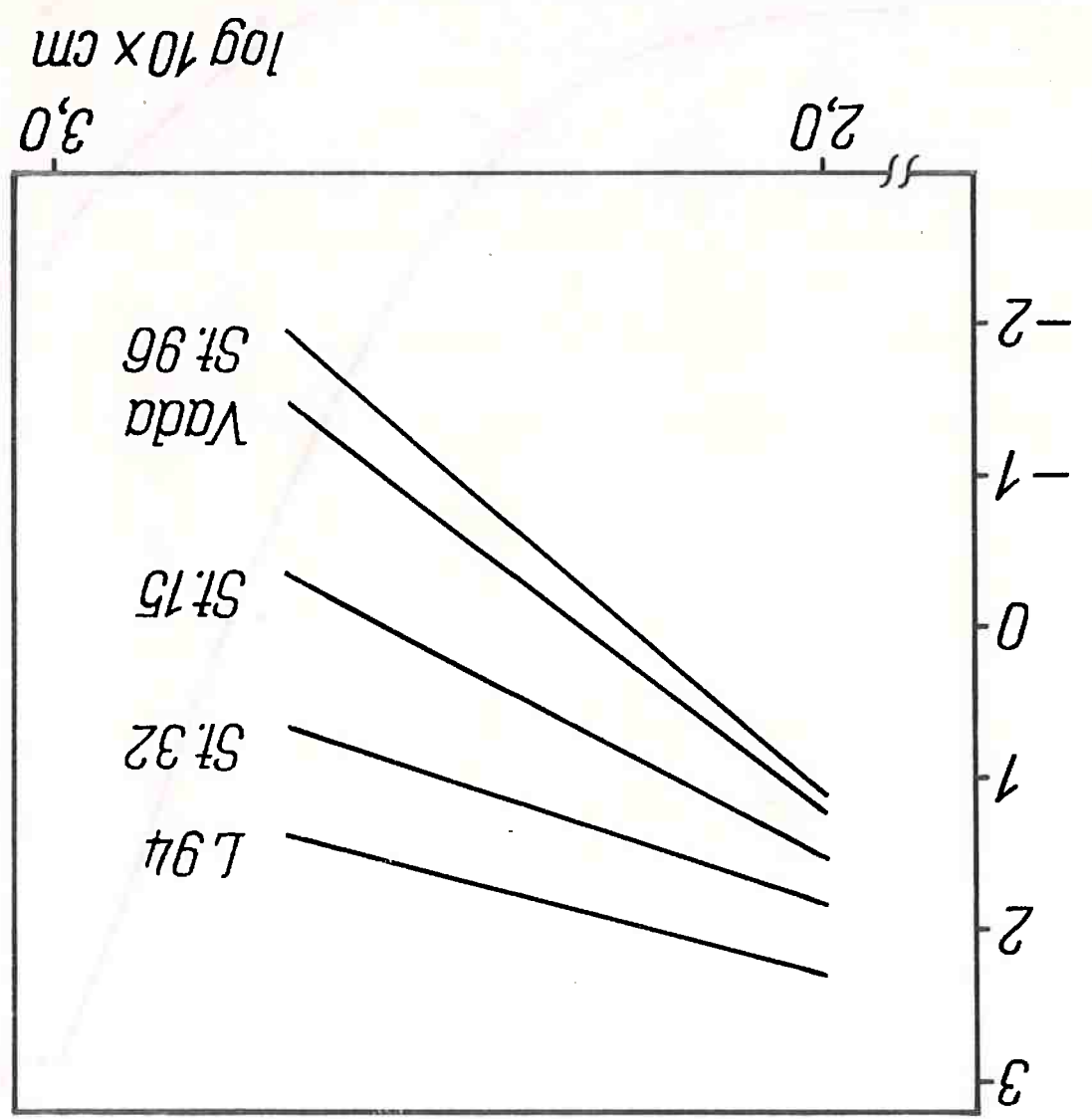


Figure 3: Infection gradients of brown rust, caused by *Puccinia hordei*, after linear regression analysis. Sixth assessment week. Abscissa: Logarithm from infection source in cm. Ordinate: Logarithm from number of pustules per leaf.

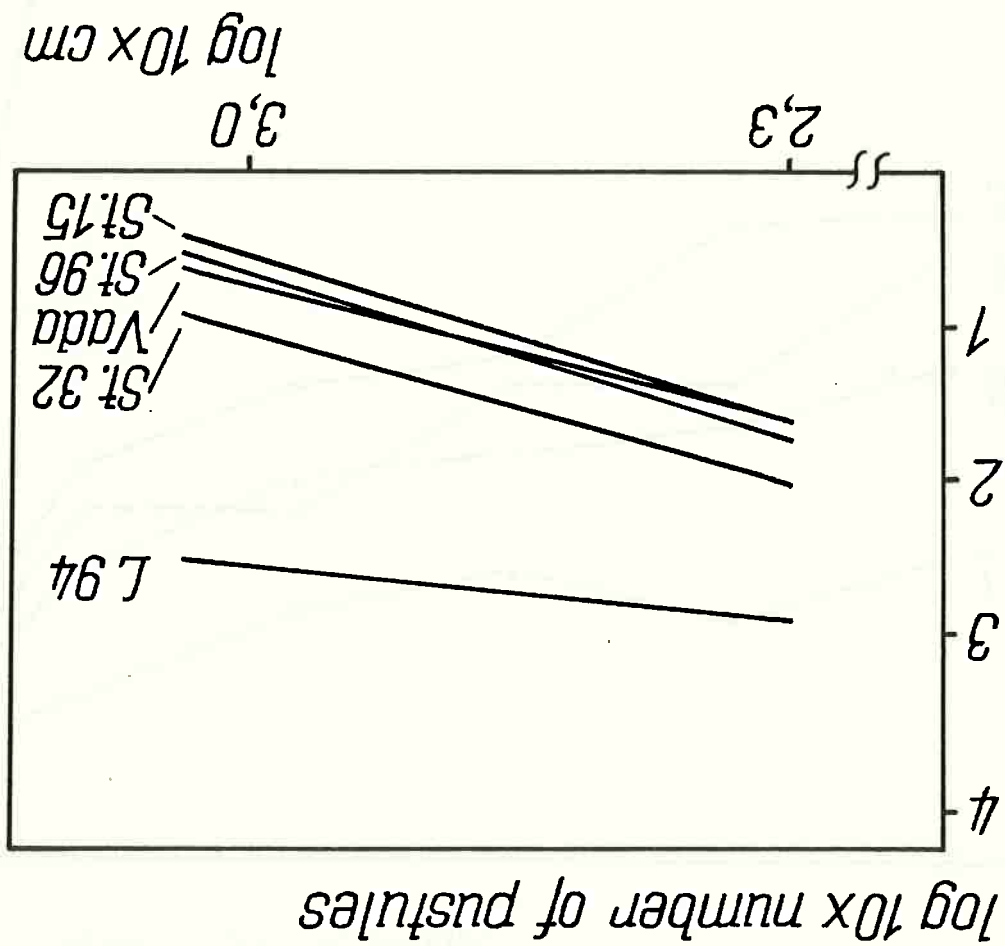
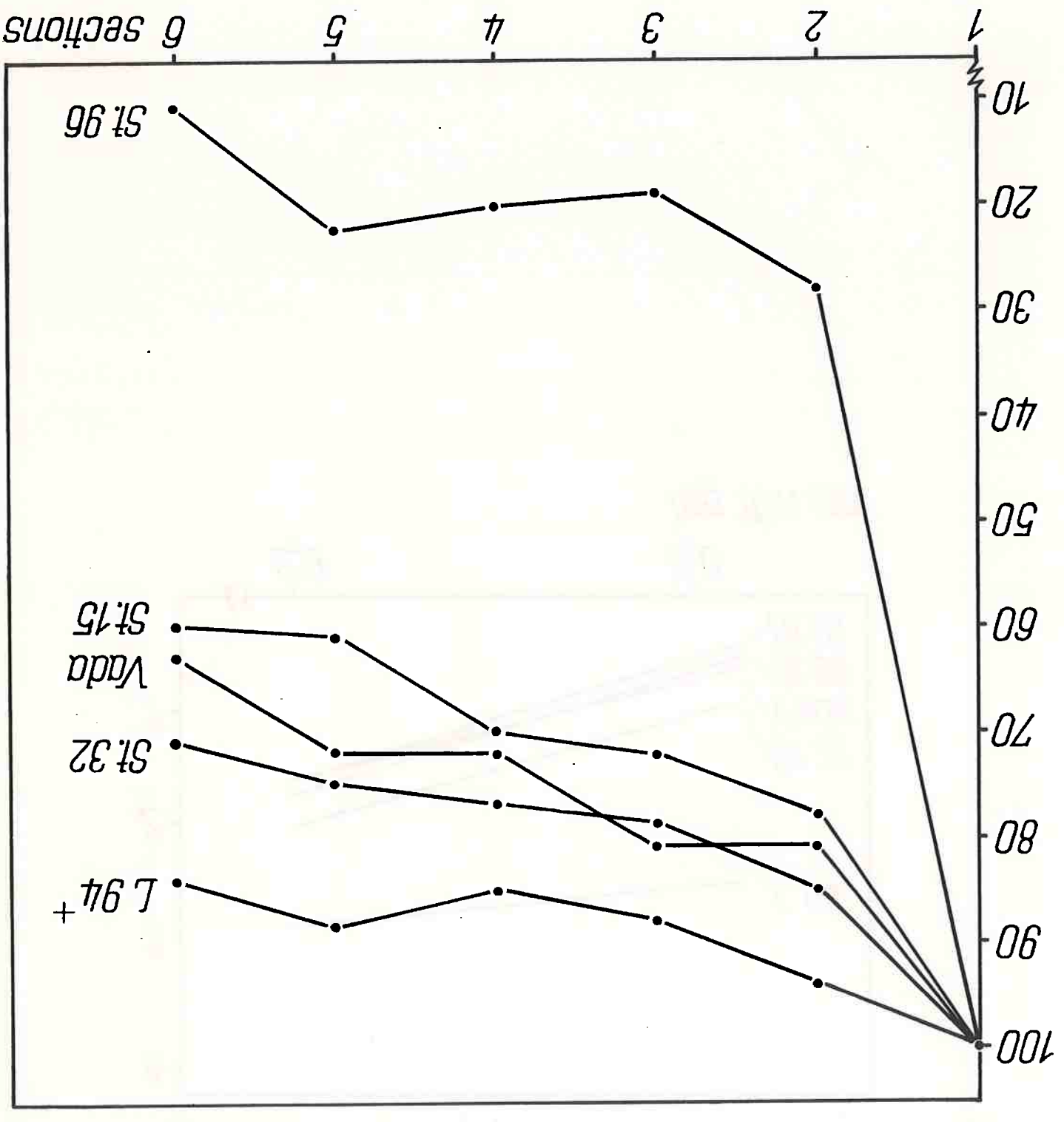


Figure 4: Brown rust, caused by *Puccinia hordei*, in the seventh assessment week in percent of infections on the first section, which is that nearest to the infection source. Sections are 200 cm long. Abscissa: Number of sections. Ordinate: Percentage of infection, expressed as rusted leaf area. *Value of 6th assessment week, the last assessment possible due to premature death of plants.



INDUCTION OF RESISTANCE TO Puccinia striiformis WEST.
IN BARLEY (Hordeum vulgare L.)

BY

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SUMMARY

Active resistance in barley plants to the yellow rust pathogen Puccinia striiformis West. (race 24) is correlated with the appearance of a terpenoid compound (internal denotation: $Sx(X_{1a}^I)$; Hofferer, 1980) - current term "activation factor" (AF) - not present in healthy plants. The metabolite - AF - is produced only in the roots of (foliar) infected resistant varieties and acts as a signal for activation of resistance mechanisms in the challenged plants. On the basis of recent work, induction of resistance to P. striiformis in susceptible barley varieties succeeded by transmission of AF from roots of infected resistant plants to susceptible plants. Resistance was induced in susceptible plants when the leaves were treated with AF prior to the challenge inoculation. The data presented show that there is a resistance potential in barley which can be activated by a natural activator.

INTRODUCTION

Yellow rust of barley (Hordeum vulgare L.), caused by Puccinia striiformis West., is a widespread and serious disease. The most typical symptoms, repeatedly described in the literature (Hassebrauk, 1970; Hassebrauk and Robbelen, 1975), are leaf chlorosis and pustules in the colonized

tissues, both indicating the nature of the infection. It is well known that pathogenic species are characterized by their host-range, or by specialization on the host-species level (Hassebrauk and Robbelen, 1974; Vanderplank, 1978). Specificity in plant diseases is connected with the problem of varietal specificity in gene for gene systems and raises the question of recognition and interaction between parasite genes for avirulence and the host genes for resistance (Wood, 1982); generally, the resistance mechanisms are unknown in detail. It is beyond doubt that the total metabolic activity of the cells is under genetic control, simultaneously influenced by environmental factors. The biochemical or physiological responses of the host plants to pathogen, also, induction resistance, or susceptibility, are therefore not necessarily specific. The establishment of a physiological change may be sufficient. Disease resistance is not a static but an elastic feature of plants (Heitefuss and Williams, 1976; Schonbeck et al., 1980; Beicht, 1981).

There are many reports on the phenomenon of induced resistance; numerous cases of induced resistance to fungal, bacterial and viral diseases are documented in the literature. Cultivar nonpathogenic races of pathogens, nonpathogens of host, pathogens, and metabolic products of hosts or infectious agents have been used to induce resistance (Beicht, 1981; Calova et al., 1976; Jenns and Kuc, 1979; Kuc, 1981, 1982; Schonbeck et al., 1980; Schonbeck et al., 1982; Sequiera, 1979).

Also various synthetic chemicals are tested for their activity as inducers (Cartwright et al., 1977; Rathmell, 1980).

The metabolic mechanisms controlling the phenomenon of induced resistance and its practical implications have received relatively little attention. In the last years, however, careful experimentation has established the validity of induced resistance, and with the establishment of its validity, progress is slowly being made on the elucidation of molecular mechanisms responsible for the phenomenon and the application of resistance induction to the practical control of disease in the field (Beicht, 1981; Calova et al., 1976;

Kuc, 1982; Schonbeck et al., 1982). The data obtained in our laboratory focused our special interest on the relationship between rust-infected leaves and the root system of barley. Studies of induced resistance in barley challenged with Puccinia striiformis West. have shown that the resistance is correlated with the appearance of a terpenoid compound (internal denotation: $Sx(X_{1a})$; Hoferek, 1980) - in this paper using the term "activation factor" (AF), the name implies the biologic activity not present in healthy plants. The formation of this metabolite occurred in the roots of infected resistant barley. AF is able to induce resistance in susceptible plants.

In this paper, the induced yellow rust resistance in barley plants and the effectiveness of the detected resistance principle are described for representative objects.

MATERIALS AND METHODS

The susceptible barley varieties Dornburger Heils Franken, Abed Binder 12, Certina, Xenia, the moderately susceptible varieties Stotz's Salmer, Grannenlos zweizeilige, and the resistant varieties Bigo, Japan 1 (Schmiedeknecht et al., 1976; Wolffgang, 1976) were used in experiments. Race 24 of Puccinia striiformis West. was used as inoculum. The plants were grown at 14 C under controlled conditions in climate chambers (Wache and Wolffgang, 1966) regulated to provide a 16h light/8h dark cycle (Opel et al., 1970), in special sand cultures (nutrient solution: 0.5 g urea, 0.5 g K_2HPO_4 , 5 drops of 5% $FeCl_3$, per litre water). The primary leaves of seedlings were infected with race 24 urediospores (inoculum: spores/talcum, g/g) 11 days after sowing. After inoculation the seedlings were incubated (3d) in dew chambers.

The plants were treated 3d prior to the challenge inoculation (P. striiformis) by spraying with the aqueous solutions of various preparations of AF, and reference preparations. Standard AF concentration was 50ppm. The treatments were started at seven o'clock a.m. and were carried out hourly (ten times daily) - in the first

Puccinia striiformis West. (race 24) causes active resistance in plants of resistant barley varieties; interconnected processes are induced. The above-ground organs and the roots function as an integrated whole with centres of parasite-host interaction in the infected leaf tissues and determinative events in the roots (root-and-shoot relationships). There must be important metabolic sites for resistance regulation and formation of active metabolite(s) in the roots (Hoferek, 1976, 1977, 1980, 1981; Schlegel and Opel, 1983). The data of experiments present strong evidence that a terpenoid compound, called "activation factor" (AF), detected in the roots of *P. striiformis* infected resistant

RESULTS AND DISCUSSION

series of tests, or later: 7h, 12h (at noon), and 17h (five p.m.) (standard condition). Control: treatment with water (aqua dest.). The success of infection (visible symptoms) was estimated on the basis of the standard infection index (Hoferek, 1980; Schmiedeknecht et al., 1976) in the same manner as previously described (Hoferek, 1977). (Tables 1 and 2, annotations a, b, and c - respectively). Terpenoid preparations from barley roots (*H. vulgare* L., var. Bigo) were obtained by extraction and fractionation on AgO_3 column (column chromatography method combined with TLC and/or HPLC) as described by Hoferek, (1980); special methods for the fine fractionation were applied. The complex procedure, combined with the cited methods, for the isolation of AF will be described in a separate paper, the detailed chemical structure (also of all other detected terpenoids) is unknown. In standard trials (Tables 1,2) the relative concentration, expressed by the extinction of the solution (U.V. absorption; Hoferek, 1980), was equal; the ultraviolet absorption spectra of the fractions (AF; AF + 1 component (terpenoid); AF + 5 components (terpenoids); Table 1) were very similar. There are evidences that the terpenoids (group of 6 compounds) are biogenetically closely related.

plants (H. vulgare var. Bigo) has the capacity and effectiveness of an inducer of resistance (Tables 1, 2).

The "activation factor" in resistant barley challenged with P. striiformis is transmissible from roots of infected resistant plants to (uninfected) susceptible plants. Resistance is induced in the susceptible plants if the leaves (shoot, all above ground parts) were treated with the factor prior the challenge inoculation with the pathogen. The induced resistance depended on a time interval (1-3 d; standard: 3d) the application of AF and the inoculation. The induced resistance acts systemic to a certain degree and is characterized by its effectiveness against the obligate parasitic fungus (P. striiformis) and its ineffectiveness against Puccinia hordei Oth., or viruses (the effectiveness of induced resistance against other diseases is being tested this year). At present it seems that in other cases protective effects occur.

Through the use of various terpenoid preparations, e.g. in series of tests with AF-preparations, differing in contents and in composition, which are tested for their activity as inducers, practically all degrees of resistance (success of infection) can be obtained from a susceptible host-parasite relationship (Table 1); in a special study the dependence of induced resistance upon the concentration of AF has been verified.

It is seen in Tables 1 and 2 that the most effective induction of resistance was obtained with the isolated, highly purified AF in the applied standard concentration 50ppm. The data indicate that there was a resistance potential in the plants (H. vulgare L. var. Abed Binder 12) which was activated by AF. The development of yellow rust was successfully inhibited. The effectiveness of induced resistance under best conditions (which has been found in preliminary experiments) in comparison with the resistance behaviour of eight barley varieties, differing in yellow rust resistance, is demonstrated by the data in Table 2. In an earlier series of tests (Hoferek, 1976; Wolfigang, 1976) an order of rank for the resistance of these varieties has been estimated. The results presented in the table confirm this

order of rank: susceptibility (eight varieties) is in a positive correlation with the degree of symptom formation (rank sequence 2-9), or otherwise resistance is in negative correlation with the degree of symptom formation. In this order of rank the variety Japan has the position of highest resistance (rank 2). The example for induced resistance (var. Abed Binder 12) expand the hitherto existing order of rank. It is seen (Table 2) that the values for induced resistance almost reached the highest possible degree of resistance (non symptoms). The data of the physiologic transformant inserted in the data of the eight varieties are equated with rank 1 in the order of rank 1-9. This demonstrates the high effectiveness of induced resistance. The mechanism of action of AF is unknown. There is the supposition that in the induced incompatibility of host and pathogen some important metabolic alterations in the plant render the host incompatible to the pathogen. We have no direct evidence to offer as yet that an inhibitory mechanism is involved in damaging the fungus in the resistant host. More research is needed to understand the molecular mechanism of resistance induction. The fact and the possibility by any way to activate an existing resistance potential in plants supports the hope that in the future this principle may be inaugurated a new way in disease control.

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Table 1: The effect of treating the above ground parts of barley with the "activation factor" (AF) detected in a terpenoid preparation on resistance efficiency and parasite development in tissues of the first leaf inoculated with *Puccinia striiformis* West., race 24. The data represent the induction of resistance to yellow rust in susceptible plants (*Hordeum vulgare* L. var. Abed Binder 12).

Treatment ^a	Induced Rust	Resistance ^(b)	Efficiency of Success of infection	Per cent of Index-number total resist- of symptom ance (100) severity	
				Development ^(c)	Resistance
Control (water-treated)		(0)		455	
Terpenoid(s) of <i>H. vulgare</i> ; Variety Bigo (resistant)				445	
- Preparation from roots of healthy plants (Reference)		(2.82)			
- Preparation from roots of plants infected with <i>P. striiformis</i> (race 24), fractionated	AF, accompanied with other terpenoids.	46.47		290	
AF, two components		62.26		234	
AF, highly purified		92.96		125	
		94.37		120	
Index number limit (highest, possible resistance)		100			100

a. Total of twenty plants per treatment in three up to fourteen separate trials (standard conditions in controlled environment chambers). The effectiveness of terpenoids prepared from barley under comparable terms were tested by the standard method.

- b. Induced resistance: susceptible plants (*H. vulgare* var. Abed Binder 12) treated with various preparations of AF and reference preparation, respectively, three days prior to the challenge inoculation (*P. striiformis* race 24) of the first leaf by spraying with aqueous solutions of the compound(s).
- c. Rust development: success of infection estimated on the basis of the standard infection index (Schmiedeknecht et al., 1975; Hoferek, 1980). Index-number 100 = highest, possible yellow rust-resistance: non symptoms at all plants of the cultivar. Duration of observation: usually, about thirty days; the data in this table are given for the twenty-first day after infection. Means of a total of twenty plants in at least three separate experiments, see annotation a and b, respectively.

Table 2: Induction of resistance to *Puccinia striiformis* West. in barley (*H. vulgare* L. var. *Abded Binder 12*) by the isolated "activation factor" (AF), and comparison of the induced resistance with the resistance behaviour of eight barley varieties (*H. vulgare* L., primary leaves of plants challenged with *P. striiformis* West.)

Rust Infection(a)	Development of symptoms (b)	Insertion(c)	Action of AF	Varities(d)	Totals Rank(e)
				Abded Binder 12	20
				Franken	
				Dornburger Heils	
				Abded Binder 12	8
Control:				Abded Binder 12	(8)
				Certina	
				Xenia	
				Stotz's Salamer	
				Grannenlose)	
				Zweizeilige)	
				Bigo	
				Japan 1	
Induced resistance:				108	
				182	
				172	
				244	
				285	
				322	
				1023	
				879	
				5	
				4	
				741	
				3	
				681	
				2	
				568	
				2	
				105	
				174	
				196	
				206	
				3	
				125	
				135	
				148	
				160	
				2	
				100	
				110	
				115	
				120	
				445	
				1	
				100	
				96.33	
				95.31	
				93.95	
				93.84	

(a) Standard conditions; Cp. Table 1: annotations a, b and c.
 (b) Symptom development on the first leaf inoculated with *Puccinia striiformis* West, race 24. Index numbers (boniture notes): 100 - lowest possible value, equal to highest resistance; 600 - highest possible value, equal to highest susceptibility; dpi = days after infection.
 (c) Data of induced resistance in *Abded Binder 12* barley inserted into the earlier well defined order of rank of resistance (see d).

(d) Order of rank of resistance of the barley varieties (Wolfgang, 1976; Hofferek, 1976) - differing in yellow rust resistance, including Abed Binder 12 barley treated with purified AF prior to the challenge inoculation (induced resistance: rank 1 - highest resistance in this order of rank).

(e) Ranks 1-9: rank 1 - lowest values of totals; rank 9 - highest value of totals (each of these totals = sum of boniture values represented for one variety).

INVESTIGATION OF STEM RUST OF WHEAT (*Puccinia graminis* Pers.
F. SP. *TRITICI* ERIKSS. ET HENN.) IN AUSTRIA WITH SPECIAL
EXAMINATION OF SLOW RUSTING OF WINTER WHEAT VARIETIES (X)

BY

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Stem rust of wheat (*Puccinia graminis* Pers. f. sp. tritici Erikss. et Henn.) is potentially a very aggressive disease in Austria. This is confirmed by epidemics of stem rust in Austria in the last 50 years, where it is usual to find that dangerous epidemics of stem rust are caused by uredospores blown from wheat growing regions. A survey of pathotypes during the years 1975 to 1980 showed the dominance of pathotypes 1, 11, 18 and 34. On the basis of virulence investigations, the gene SR11 expressed itself throughout as resistant whereas the gene SR5 (resistance gene of Thatcher and incorporated in some Austrian winter and summer wheat varieties) is highly susceptible). (The analysis was done by Mrs N. Vlahovic, M.Sc., University of Zagreb, Yugoslavia).

Slow rusting is coordinated with quantitative and horizontal resistance. The experiments to identify varieties with slow rusting resistance were done by inoculating seedlings and adult plants. Based on the type of attack and

(X) This contribution is essentially the summary of the publication "Studie über den Schwarzrost des Weizens (*Puccinia graminis* Pers. f. sp. tritici Erikss. et Henn.) in Österreich mit besonderer Untersuchung der Slow-rusting-Resistenz von Winterweizensorten, Die Bodenkultur, 33, 1982, 246-274.

number of pustules (seedling resistance) and type of attack and severity (field resistance) respectively, the method of calculating "area under disease progress curve" (A-value) was used to characterize the varieties (table 1). The curves correspond to the numbers in table 1). Varieties could be classified into the following groups based on reaction to disease attack (severity and type of attack):

1. resistant varieties (number 1, 2 and 4)
2. slow-rusting varieties (3, 5 and 14)
3. fast-rusting varieties (15-32); in figure 1 only variety 32 (Onus) is shown as an example.

The mean reduction of TKW of resistant varieties was 7.96% (s 2.30), of slow-rusting varieties was 14.07% (1.89) and of fast-rusting varieties 29.65% (s 2.41); $r = 0, 63, b = 0.025$ (fig. 2).

By testing separately the three groups of varieties the following correlation between A-values and TKW was found:

resistant varieties: $r = 0.39$ (not significant)
 $b = 0.018$

slow-rusting varieties: $r = 0.44$ (significant)
 $b = 0.014$

fast-rusting varieties: $r = 0.47$ (high significant)
 $b = 0.038$

From these results it is concluded that the methods used give the possibility of differentiating between varieties, particularly to identify slow-rusting varieties. Attacks of stem rust in Austria usually appear earliest at the flag leaf stage. Therefore the examination of susceptibility should be done at the adult plant stage only. By taking all present genetical and epidemiological knowledge into consideration the importance of slow rusting varieties could be summarized:

1. Higher security of yield.
2. Reduced risk of epidemics.
3. Lower pressure for selection of new pathotypes.

4. Longer durability of the obvious polygenic resistance.
 5. Decreased costs of chemical treatments.
 6. Progress in realization of biological and integrated plant protection.
- For a full set of references see the original paper.

Table 1 - Slow rusting as field resistance

No.	Variety's name	A-value (summarized)	Duncan-test significance of 95% to following varieties (number)
1	Atut II	135	6-32
2	Disponent	338	8-32
3	Wieselb.II	486	12-32
4	Gotz	499	12-32
5	Probst. Pokal	523	13-32
6	Eira Kolben	598	14-32
7	David	685	17-32
8	Probus	756	18-32
9	NR 63/76	781	18-32
10	Adam	815	18-32
11	Rivoli	845	18-32
12	P4258	928	26-32
13	NR400/76	983	29-32
14	Sava	1051	32
15	Probst.Karat	1081	-
16	Agron	1090	-
17	Probst. Perlo	1108	-
18	Danubius	1261	-
19	Probst. Gigant	1269	-
20	Aquila	1279	-
21	Libellula	1289	-
22	P100/73	1297	-
23	Probst.Extrem	1317	-
24	Armada	1318	-
25	Kormoran	1330	-
26	Gambrius	1345	-
27	Jubilat	1371	-
28	Probst. Regent	1390	-
29	Diplomat	1418	-
30	Multiveiss	1419	-
31	Primus	1433	-
32	Onus	1471	-

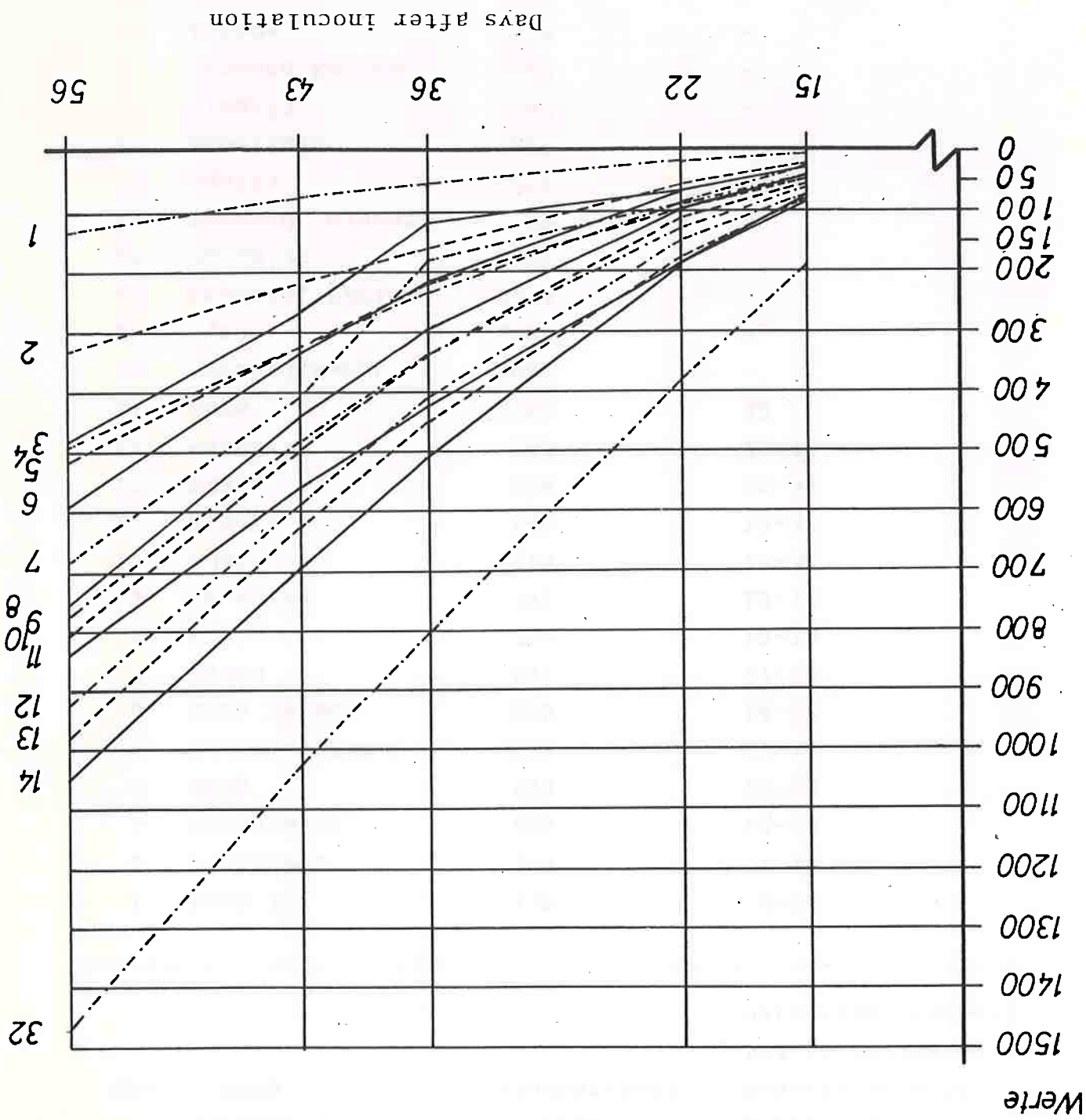
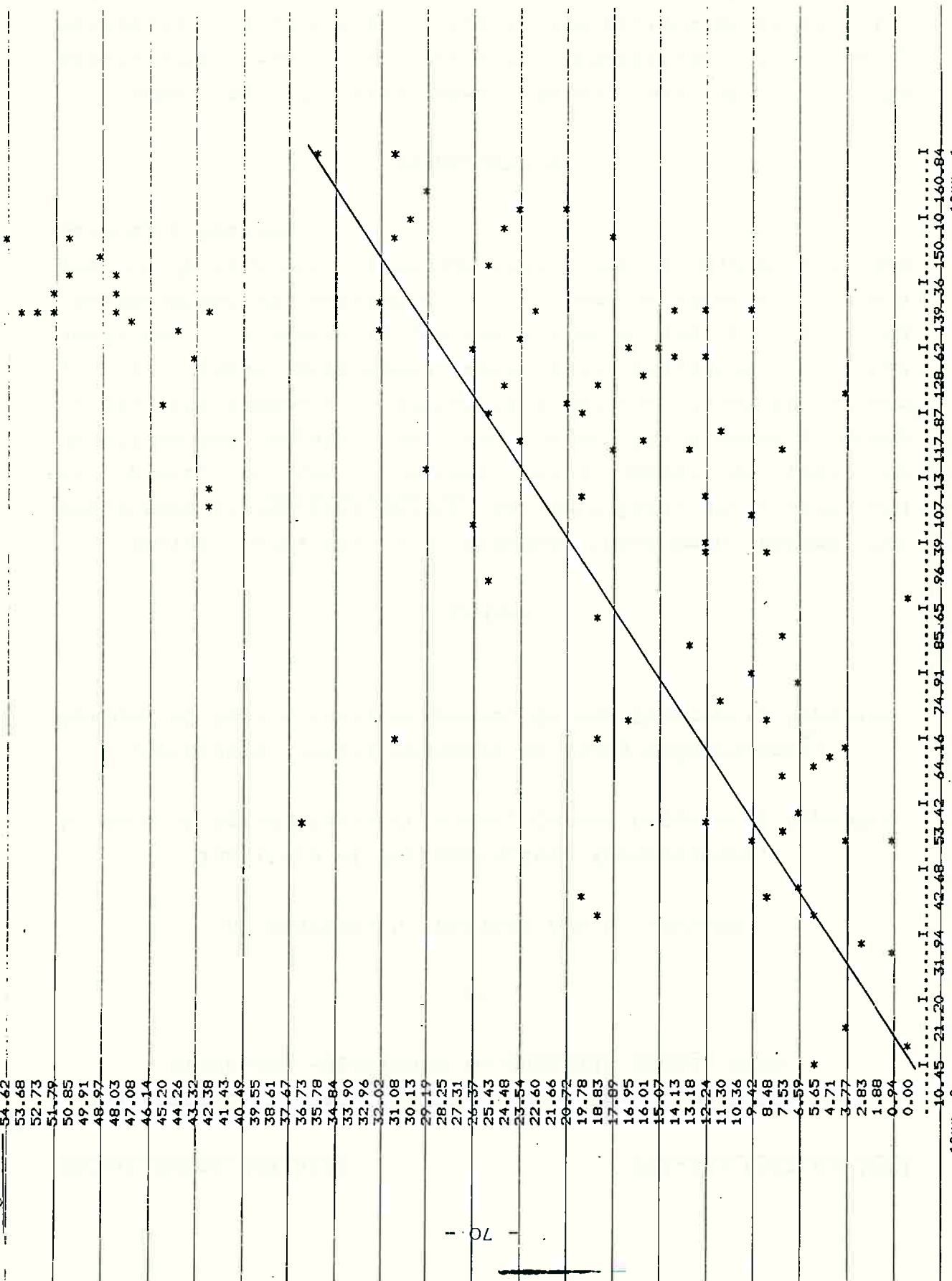


Figure 1

Figure 2 55.56



Resources of resistance genes available in the barley/leaf rust host-parasite combination are nearly exhausted. It is for this reason that world-wide efforts are made to utilize race-nonspecific resistance which can be detected in several ways (Johnson and Wilcoxon 1978, 1979; Neervoort and Parlevliet 1978; Niks 1982; Niks and Parlevliet 1979; Parlevliet 1975; 1976, Parlevliet et al. 1980).

INTRODUCTION

Twelve cultivars and breeding lines were tested for resistance to Puccinia hordei on both large scale plots and microplots, at two locations for a number of years to determine and compare the area under the disease progress curve, the apparent infection rate and the relative latent period. These parameters were also evaluated for their usefulness in assessing the resistance of the plants. The ranking order of resistance is the same on large scale plots and on microplots, although infections on microplots are generally heavier.

SUMMARY

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BY

COMPARATIVE EPIDEMIOLOGICAL STUDIES OF SPRING BARLEY WITH
DIFFERENT RESISTANCE TO PUCCINIA HORDEI OTH.

Vol. 12, Part 2, 1984

Cereal Rusts Bulletin

Although there are differences of opinion on the genetic mechanisms of race nonspecific resistance, there is almost general agreement when it comes to evaluate epidemiology: the disease proceeds slowly and is subject to environmental influences. The study described in this paper aims at a comparison and appreciation of the results achieved by various methods over a number of years for the assessment of quantitative levels of resistance. While disease progress was assessed at No.1 location by counting pustules on relatively large areas the same was done at No. 2 location under nursery conditions.

MATERIALS AND METHODS

The cultivars and breeding lines used were selected from those which had been cultivated and artificially infected in the nursery in 1979. The selection criteria applied were: latest period, and percentage of diseased leaf area. The material selected in the field in this way was tested for vertical resistance with 5 races of P. hordei in the greenhouse the following winter (Table 1).

Cultivars with known vertical resistance were included to check their response to the Trumpf races which had been detected in the pathogen population in the proportion of approximately 70% since 1977. Field tests at No.1 location were carried out on 1 x 30m plots arranged in blocks with four replications, in 1981 and 1982. On the west side of plots five spreader rows of the highly susceptible cultivar L94 were sown. For isolation, oat plots were sown 1 metre wide between plots. The long shape of plots was designed to observe the disease progress (Hartleb et al., 1984 in press). In 1981, the assessment of disease progress was performed by counting the number of pustules on the three upper leaves of 100 randomized culms per plot every week from June 24 until July 14, this procedure being followed by an estimation of percentage infection. Artificial inoculations with race R23-4280, virulent on all the cultivars and breeding lines tested, were made in 1982. Vernalized seeds of the

Table 1. Seedling test reaction of selected lines and cultivars to 5 races of *Puccinia hordei* Oth.

Line of	Races	Trumpf	Resistance
cultivar	UN23	UN54-3 races	genes
	UN23	UN23	UN23 +4280

L94	s	s	s
St.32	s	s	s
Mirena	s	s	s
St.116	s	s	s
St.123	s	s	s
St.15	s	s	s
Vada	s	s	s
St.96	s	s	s
St.76	r	mr	s Trumpf resist-
			ance (at least
			2 genes)
Lada	r	mr	s Trumpf resist-
			ance
Consta	r	mr	s Trumpf resist-
			ance
Gerlinde	r	mr	s Trumpf resist-
			ance

S = susceptible; mr = moderately resistant; r = resistant

susceptible winter barley cultivar Vogelssanger Gold were sown in 15cm pots and infected later in the greenhouse. These were planted to the western side of the various plots just before sporulation when spring barley had reached Feekes growth stage 5. From May 25 until July 5 1982, disease assessments were made every week to evaluate disease progress in an area within 3m of the source of infection, by counting

the numbers of pustules and assessing the degree of infection on 20 randomized leaves per replicate. Disease assessment was always done on the lower non-yellowed leaf to follow the development of the disease from its very beginning. This method ensured that leaves at the same growth stages were assessed for all cultivars.

Calculations were made both for the area under the disease progress curve (Wilcoxson et al., 1974) and apparent infection rate according to Vanderplank (1963). At No.2 location breeding lines and cultivars were tested under nursery conditions in blocks (microplots). For each test number a row of 90cm length was established. A highly susceptible short-stemmed winter barley line was drilled as a spreader row in the main direction of wind in autumn and infected in middle/end of May with a mixture of races corresponding to the natural population of the previous year. Disease assessments were commenced about the middle of June when the susceptible control cultivar L94 showed approximately 10% of diseased plants, and repeated 4 or 5 times at approximately weekly intervals according to weather conditions. The amount of leaf area affected by disease was estimated in percentage on each test plot on the third leaf from the top. Latent period was determined with L94 as the control for the beginning of infection and is given in terms of "x days later than L94".

Such a 'relative latency period' can not be directly compared with values obtained in the greenhouse from artificial inoculation because the actual day of infection is unknown. In addition, field No.2 was situated within a area of extensive spring and winter barley cultivation and where natural spore spread occurred. The level of resistance was indicated by the calculated area under the disease progress curve according to Wilcoxson et al., (1974).

RESULTS

No.1 location: Disease attack on L94 was as high as 7% at the beginning of disease assessment in 1981 and increased

to 100% at the end. Lada, St.32, St.123 and St.116 showed less than 1% of disease attack during the first assessment week while the remaining cultivars and lines were free from attack at that time. Analysis of variance of the calculated A-values (area under the disease progress curve) results in significant differences between all the cultivars and breeding lines from L94, but only slight differences among themselves (Table 2). There was a significant difference between Nos.2 to 12 and L94, Nos.6 to 12 and St.32, and Nos.10 to 12 and Lada at = 5% and a limit difference of (Tuckey) of 6.35.

In this test good agreement was found between the apparent infection rate r , and the level of resistance expressed by A-values (Table 3).

In 1982, the first colonies of uredospores occurred on 8 test lines twenty days after sporulating winter barley had been planted while only single pustules were observed on St.76, Gerlinde, Lada and Consista (up to 0.2 pustules per leaf) until the sixth week of disease assessment (by June 29th). Fig.1 shows the levelled disease progress curves of three selected cultivars and breeding lines which are clearly distinguished from the sigmoid curve shape of the highly susceptible cultivar L94. Analysis of variance of calculated A-values at = 5% and a limit difference (Tuckey) of 17.05 showed significant differences between L94 and numbers 2 to 12, as well as between St.32 and numbers 3, 8, 11 and 12 (Table 2). In this test differences in level of resistance were shown more clearly by the A-value than by the apparent rate of infection r (Table 3). Graduation of A-values will not allow for adequately changed r -values to be distinguished except for L94. However, differences in disease progress obviously seem to be too small than can be made visible by means of r .

The low significance of apparent infection rate in this test is reasoned by too strong a levelling over the period of time of seven weeks. Therefore r was calculated both for the beginning (r_1) and end (r_2) of the disease but none of the values turned out to be significant on its own.

No.2 location. The 1979-1981 nursery results are shown

in Table 2 and are very similar to those of No.1 location. The more susceptible cultivars L94, St. 32, Lada ad Mirrena can clearly be identified at both locations over the years. However Lada is an exception at No.1 location with artificial infection with race R23-4280 in 1982. Although this race occurs in the natural population together with 5 other Trumpf races it is able to attack Lada only very slightly. Contrary to that, this cultivar was more severely infected under natural conditions or after inoculation of a mixture of races found at both locations from 1979 to 1981. This indicates a difference between Trumpf races.

Table 2. Levels of resistance of cultivars and breeding lines expressed as the area under the disease progress curve (relative to L94 = 100) from tests continued over years, at 2 locations.

No. Cultivar/ breeding line	No.1 location		No.2 location	
	1981	1982	1979	1980 1981
1 L94	100.0	100.0	100.0	100.0
2 St.32	11.2	18.0	85.0	89.9
3 Lada	9.2	0.3	26.7	41.4
4 St.116	6.3	10.1	11.3	-+
5 Mirrena	5.1	13.8	23.3	23.0
6 St.15	4.0	7.3	5.2	-+
7 St.123	3.7	8.0	7.0	-+
8 St.76	3.3	0.9	7.0	-+
9 Vada	3.0	6.9	8.3	17.7
10 St.96	2.8	1.3	19.3	16.7
11 Gerlinde	2.3	0.2	8.6	16.3
12 Conista	2.0	0.7	7.0	16.1

+ not tested

The cultivars and breeding lines were classified according to their latent period (LP) determined at No.2 location. Barley in the first class shows no delay of attack compared with

L94. The difference between the various classes is 7 to 10 days depending on time of assessment. This means that cultivars and breeding lines of class 3 showed a delay of attack of 14 to 20 days compared with L94. Duration of latent period varies considerably over the years. In 1979 and 1980 when weather conditions were suitable for continuous development of attack, good agreement with classification was

Table 3. Comparison of values of the area under the disease progress curve (A) with apparent infection rate (r) found at various points of time during the disease.

Cultivar	1981		1982		1982	
	r	r ²	r	r ²	r	r ²
L94	0.45	0.20	0.24	0.06	0.05	0.02
St.32	0.21	0.06	0.04	0.02	0.05	0.02
Lada	0.24	0.06	0.04	0.02	0.04	0.06
St.116	0.27	0.07	0.04	0.02	0.04	0.11
Mirena	0.25	0.06	0.07	0.03	0.05	0.03
St.15	0.23	0.05	0.04	0.02	0.04	0.12
St.123	0.23	0.05	0.05	0.03	0.05	0.08
St.76	0.22	0.05	0.07	0.03	0.07	0.19
Vada	0.21	0.05	0.08	0.03	0.08	0.19
St.96	0.22	0.06	0.02	0.01	0.02	0.06
Gerlinde	0.19	0.04	0.01	0.01	0.01	0.01
Conستا	0.19	0.04	0.06	0.02	0.06	0.14

+ no attack by that time

observed (Table 4). Clammy weather in the middle of June, 1981 slowed down or stopped disease progress, and a

distinct increase became visible only at the beginning of July. Disease records on Vada and St.123 therefore could be made only as late as the fourth assessment date. Cultivars and breeding lines which could be classified into No.1 class in previous years showed delay in 1981. The longest period was found with Vada, except 1980. The pustules on this cultivar generally had been very small, frequently embedded in necroses.

Table 4. Classification of studied cultivars and breeding lines for classes according to duration of latent period.

Cultivar/ breeding line	1979 class	1980 class	1981 class
L94	1	1	1
St.32	1	1	2
Lada	1	1	2
St.116	1	-+	3
Mirena	1	2	3
St.15	2	-+	2
St.123	2	-+	4
St.76	2	-+	2
Vada	3	2	4
St.96	2	3	-++
Gerlinde	2	1	2
Consta	2	2	3

+ not tested
++ not diseased

No large differences in susceptibility to *P. hordei* were found with cultivars and breeding lines selected for these experiments. This indicates, however, that slight differences in disease progress could be identified only by means of several assessments for disease level. In 1982 for instance St.32 had at location No.1, 2.6 fold the A-value of Vada which had been caused by a short latent period and a severe attack during the first assessments while final assessment values were the same for both varieties. Calculation of the area under the disease progress curve turned out to be very well suited for estimating quantitative differences of resistance thus proving statements made in earlier publications (Wilcoxson et al 1974, Johnson and Wilcoxson 1978, Rees et al 1979, Andrejcenko et al 1982, Zwat 1982). This value will allow for statements on disease progress in time as well as on the disease level to which the disease proceeds. This will not apply to the apparent infection rate (r) as it expresses only the disease progress in time. Since the slopes of disease progress curves are very much different from each other in the various phases from the beginning to the end of disease, different r-values will result with respect to the choice of assessment dates and it will be doubtful whether the sections used for disease assessment and, for resistance level assessment are the decisive ones. Those r-values for their own in most cases. The proportion from r2 to r1 was formed. As a result those cultivars and lines which rank on the first three orders for their A-value are distinctly distinguishable from the others. It needs to be checked whether this value can be utilised for differentiation of fast-rusting and slow-rusting types.

The detection of the 'relative latency period' of breeding material in the field by contrast to a highly susceptible standard had results which largely correspond to influences on progression of disease exerted by variety resistance. With this, plant breeders are given just another piece of information useful for evaluating breeding material.

DISCUSSION

The comparison between results of resistance studies made in field plots and microplots, agrees with reports by Zadocks and Schein (1979), Ress and Thompson (1979) and Parlevliet et al. (1980) stating that the ranking of tested cultivars is more or less the same. In the comparable year 1981 a correlation of $r = 0.97$ ($= 0.18$) could be found. It was realized that a much more impetuous attack of disease under nursery conditions than on microplots can result in the underestimation of resistance. The differences between the highly susceptible L94 standard and the other cultivars and breeding lines on microplots were less significant than on isolated large plots which may be important in selecting for resistance in breeding material. In the proximity of spreader rows and highly susceptible lines it consequently can not be avoided that 'reflected diseases' (Zadocks and Schein 1979) are recorded. Microplots, however, are useful for some screening in the beginning of the breeding process.

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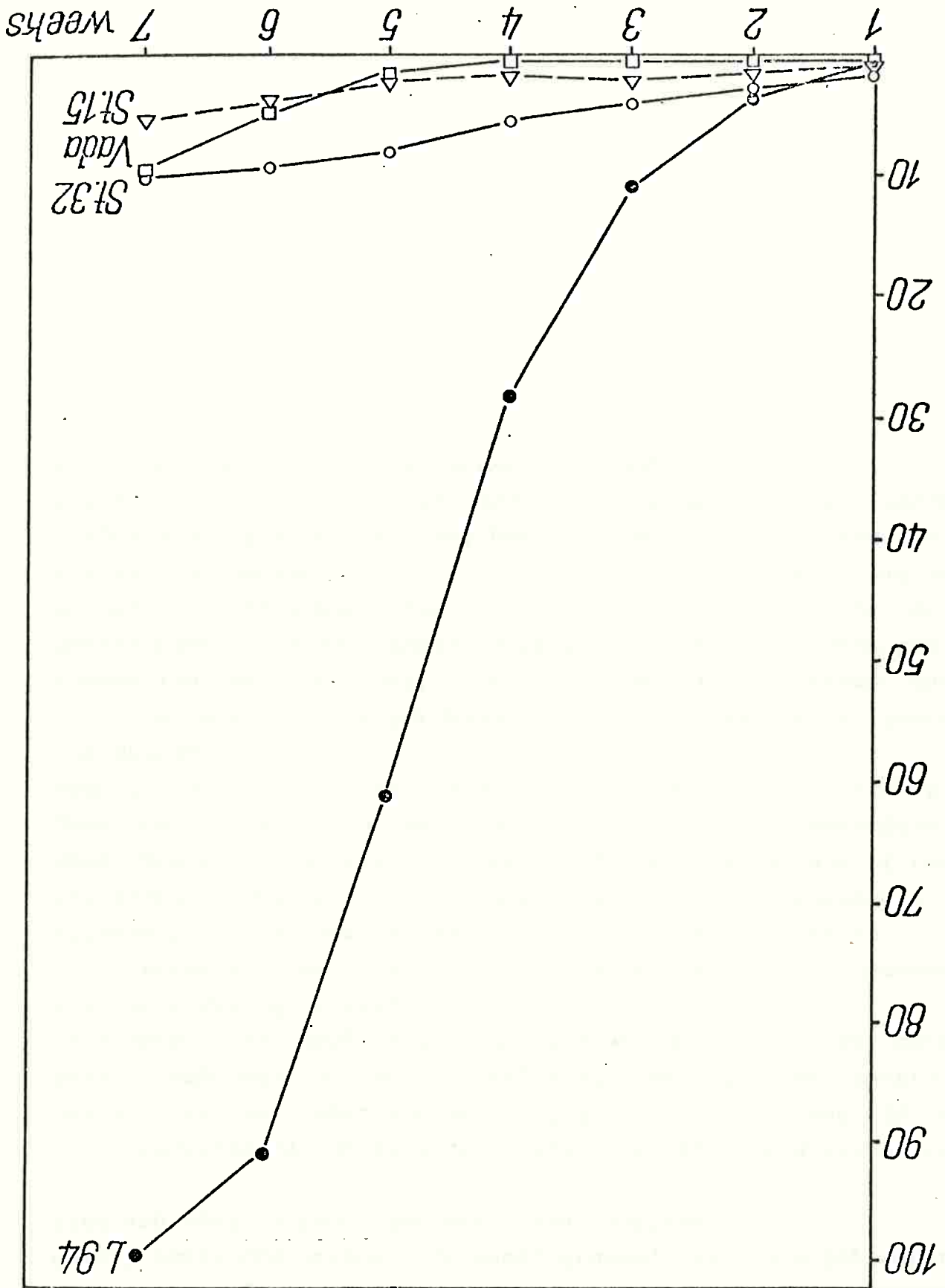


Figure 1: Disease progress curves of *Puccinia hordei* on three partial resistant varieties and breeding lines, compared with the highly susceptible standard L94.

Resolution agreed at the 11th European and Mediterranean Cereal Rusts Conference - Grignon, France, 3rd - 7th September 1984 and sent to major European organisations

Genetically controlled resistance to rust diseases in cereals is an important means of disease control and is a major component of any integrated rust disease control programme. In many areas of the world it is the only available method of control.

Breeding for disease resistance cannot be pursued efficiently without knowledge of the genetic basis of resistance. Analysis of the genetics of resistance and the development of resistant cultivars requires a knowledge of the genetical variation in pathogenicity in the rust pathogens, and the availability of the appropriate pathogen strains to the breeders.

The board of the European and Mediterranean Cereal Rusts Foundation and the participants of the 11th European and Mediterranean Cereal Rusts Conference therefore urge that surveys of such pathogens should be continued and that any trends towards reductions of international surveys should be compensated for by the development of national and regional resources for this purpose, and that such developments should be co-ordinated within and between geographic regions.

In order for the Bulletin to continue to be a means of contact between Cereal Rust workers a supply of papers, short notes, letters to the editor etc, are essential. These can be any length and on any topic concerned with cereal rusts eg virulence surveys, particularly the National surveys carried out in different countries or regions, resistance gene analysis, breeding for resistance, chemical control, techniques, information about future meetings, reports of meetings, changes in personnel at research institutes and breeding stations, comments on books etc. In fact a wide range of topics can be covered. It may be possible to produce a Bulletin consisting of formal papers and also a series of informal news letters. This can only be achieved by regular contributions from all rust workers. All contributions should be sent to Dr. N. H. Chamberlain, Nickerson RPB Ltd, JNRC, Rothwell, Lincoln, LN7 6DT, England.

PAPERS/CONTRIBUTIONS

Regrettably, increased costs have meant that the subscription rate, maintained stable for the past eight years, will need to be increased. From 1st January 1985 the annual subscription will be increased by 5 guilders. All enquiries concerning subscriptions should be sent to Professor J. E. Parlevliet, Treasurer, European and Mediterranean Cereal Rusts Foundation, P O Box 271, Wageningen, The Netherlands.

SUBSCRIPTIONS

