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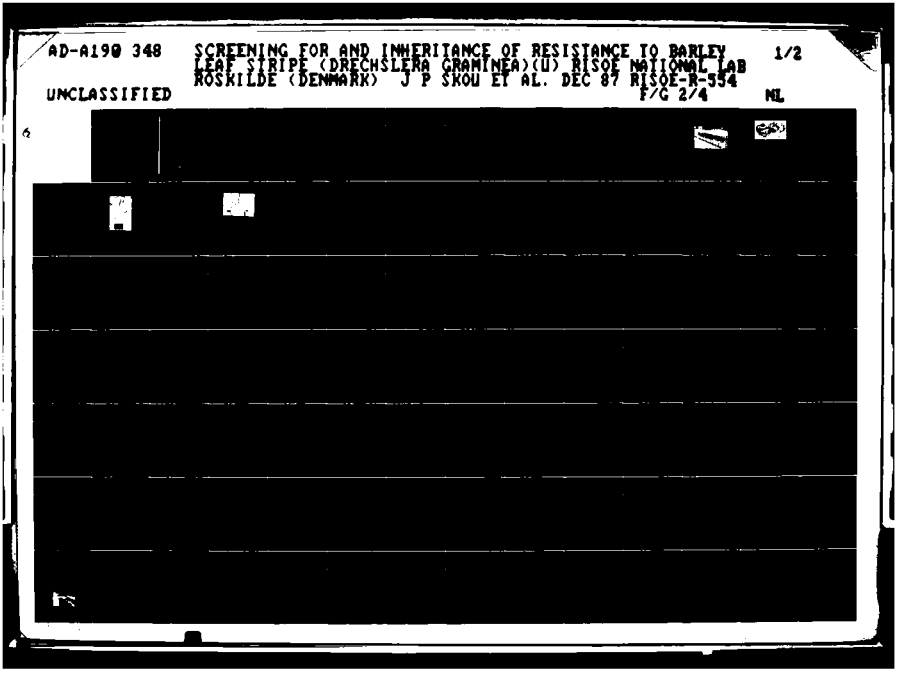

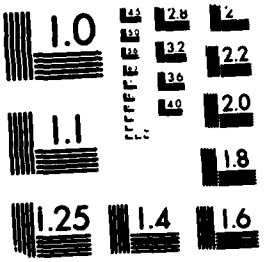

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Screening for and Inheritance of Resistance to Barley Leaf Stripe *(Drechslera graminea)*

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SCREENING FOR AND INHERITANCE OF RESISTANCE TO BARLEY LEAF STRIPE (*Drechslera graminea*)

J.P. Skou and V. Haahr

Abstract. Barley leaf stripe caused by *Drechslera graminea* (teleomorph: *Pyrenophora graminea*) gained renewed importance after the ban of seed dressing with organic mercurials. Interest rose for breeding resistant varieties in order to keep the disease down without the use of chemicals. To meet this goal 1029 varieties and lines from different Nordic barley collections were screened for reaction to leaf stripe. The monogenic and semi-dominant inheritance, named the Vada-resistance, was confirmed in an analysis of chromosome-redoubled monoploids produced with the *bulbosum*-technique. The plants were infected spontaneously from diseased plants in the field or by a monospore culture using the sandwich method with the seeds inoculated between two layers of mycelium-overgrown agar plates.

A new, uncommon symptom was uncovered. Absolute resistance or immunity in the strict sense hardly exists, as even the highly resistant barleys may now and then attract the disease. However, both host and pathogen struggle in these cases that result in dwarfing, tillering plants with weak leaf stripe symptoms.

The overall results of field inoculation revealed that 42.4% barleys had 0-10% attack and 6.7% of these had no attack at all. The reason for the low or zero attack percentage is the wide use of several relatively resistant old barleys in breeding the northwest European "family" of barleys. A subsequent test of

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selected, resistant barleys inoculated with the monospore culture separated them into different levels of resistance.

The resistance of about 50-100 barleys was traced through the pedigrees. In five series of closely related varieties levels of resistance were found that suggested the existence of different sources.

The most common leaf stripe resistance, the Vada-resistance, was found to be a one-gene conditioned semi-dominant resistance which by chance have been bred into the northwest European barleys together with - but independent of - the *Laevigatum* powdery mildew resistance via 'Vada' and 'Minerva'. This suggests this resistance to occur in many varieties descending from 'Vada' and 'Minerva'. Further, that it may easily be bred into other barleys.

Disguised behind this resistance, susceptibility modifying factors were found that suggest the existence of partial resistance.

Other highly resistant northwest European barleys, *e.g.*, 'Freja', apparently possess two additively acting genes. A few other highly resistant varieties without any known genetic background were found in the northwest European barleys - including 'Tystofte Kors', which seems to be the first barley variety in the world described as resistant.

Further, a relatively large number of barleys tested that originate from other continents and Russia were found to be highly resistant to leaf stripe.

Crossing a resistant with a susceptible variety has now and then led to a variety with an intermediary level of attack, and in other cases transgressive effects have led to varieties with either a higher level of resistance or susceptibility than in their parents.

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A series of susceptible related varieties and lines with 15-80% attack come from Finland, Norway and Sweden. They descend mainly from 'Asplund', 'Maskin', and 'Svalöf Vega'. The genes or susceptibility promoting factors in these varieties obviously have an additive effect. The infection level of more than 50 barleys may be explained by assuming they have five or six genes with one to three or four genes in each variety. Of the basic varieties only 'Maskin' is supposed to posess two genes, and no single gene conditions more than 40% attack.

On comparing the results presented with those of several others, we gain strong support for the findings of physiological specialization in the pathogen on a world-wide basis.

ISBN 87-550-1378-3
ISSN 0106-2840
Grafisk Service Risø 1987

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1. INTRODUCTION

For many years, attacks with barley leaf stripe (anamorph: *Drechslera graminea* (Rabenh.) Shoem.; teleomorph: *Pyrenophora graminea* Ito & Kuribay.) were nearly non-existent in Denmark due to an extensive use of seed treatment with organic mercurials since the 1930s. The disease gained renewed importance, however, after this treatment first became reduced and later forbidden because it frequently reaches infection levels that require chemical treatment of the seed in order to avoid a yield reduction (JØRGENSEN, 1976/77, 1979).

Previously, screening for resistance to leaf stripe to a larger extent has been performed in USA (SUNESON and SANTONI, 1943; SHANDS and ARNY, 1944; KLINE, 1971, 1972; METZ and SHAREN, 1979) and Canada (TEKAUZ, 1983; LOISELLE, 1985), and to a lesser extent in India (MOHAMMAD and MAHMOOD, 1973), Sweden (KRISTIANSSON and NILSSON, 1975; NILSSON, 1975), and Denmark (KNUDSEN, 1980, 1981; SMEDEGAARD-PETERSEN and JØRGENSEN, 1982). These investigations uncovered a markedly varying number of resistant varieties. The heredity of resistance was treated to only a limited extent in these investigations.

On this background we decided to screen a large number of barleys in Nordic collections for resistance to leaf stripe, and to analyse pedigrees of barleys in order to see how resistance might be inherited. Further, a single cross of a highly resistant and a susceptible variety was analysed genetically. Results of these investigations are presented below.

2. MATERIAL AND METHODS

The barleys tested (*Hordeum vulgare* L. *s.l.*) were provided by several Nordic barley breeders and institutions as outlined in Tab. 1. Most of these varieties and lines are now deposited in the Nordic Gene Bank, Alnarp, Sweden. Given in round figures, 900 of these barleys originate from Europe. Of these 650 are northwestern European varieties and 250 morphological variants

including "botanical barleys" of *H. vulgare* collected at many different localities by various persons and designated with old, complex epithets (*nomina synonyma, illegitima vel invalida*) pointing to their special characteristics.

Table 1. The tested barley varieties and lines as provided by Nordic barley breeders and institutes.

Name and institute	Abbre-viation	No. of entries
R. Aikasalo, Hankkija Plant Breeding Institute, Finland	Hja.	13
S. Andersen, Dept. of Crop Husbandry and Plant Breeding, The Royal Veterinary and Agricultural University, Copenhagen, Denmark	KVL	787
J.C.N. Knudsen, Axel Toft Durup A/S, Denmark	JK	23
P. Lundin, Weibulls A/B, Landskrona, Sweden	W	22
R. Manner, Institute of Plant Breeding, Jokioinen, Finland	Jo.	32
B. Nilsson, Svalöf A/B, Sweden	Sv.	38
J. Rasmussen, Government Research Station, Tystofte, Denmark	Tys.	11
E. Strand, Dept. of Farm Crops, Agricultural University, Ås, Norway	NLH	53
T. Tómasson, Agricultural Research Institute, Keldnaholt, Iceland	Ice.	8*
The Risø Collection, Roskilde, Denmark (provided by many persons)	Risø	42
Total		1029

* Only three are real Icelandic varieties, two originate from the Faroe Islands but were grown on Iceland for about 40 years, and the last three are Swedish varieties.

A hundred came from other continents and about 20 are of unknown origin. A few of the varieties were provided from more than one source, and in that way have supported the results found.

Only 'CI 6944' from Afghanistan is to be mentioned separately. It is provided by B. Nilsson, Sweden, as the most susceptible line ever seen (pers. comm.). For this reason it was used as parent for the investigation of the inheritance of the resistance in the variety 'Zita'.

The pathogen used for inoculum in the field constituted a population as it came partly from infected seeds in the collection of barleys received and partly from a heavily infected sample of 'Lami' barley (KNUDSEN, 1980). The inoculum was propagated in 'Lami' and 'CI 6944' without selection. A monospore culture was isolated from 'Carlsberg II' (Ax-48, 1977) at Risø for inoculation using the "sandwich method" in the greenhouse (see below as wellas KNUDSEN, 1986, who also used a culture from this material). This culture may not necessarily be a part of the above--mentioned population.

In 1982 and 1983 about 1,000 and 180 varieties and lines were inoculated in the field, respectively. Twenty-three kernels of each were space sown in two rows of one m. Four rows of a 1:1 mixture of the infected 'Lami' and 'CI 6944' were sown at the ends of the rows with test-barleys. The whole area was surrounded by a 2.5 m belt with infected 'Lami'. This gave a high inoculum potential and close to 100% infection would be expected in the most susceptible varieties.

Twenty-five to 30 randomly selected spikes of each variety or line were harvested. This was subsequently screened for leaf stripe reaction in the greenhouse at room temperature. The kernels were germinated in an incubator on moistened filter paper after the method of PORTER (1939), but modified to 9-10 days at 9-10°C (c.f., e.g., TEVIOTDALE and HALL, 1976) after verification of the method in order to ensure the highest possible level of attack. Thirty of the pregerminated kernels were sown in a peat-rich soil in 16 cm pots without [illegible] of

the barleys from the 1982 inoculation, and 25 kernels per pot were sown in four replicates for the test of those from the 1983 inoculation (Fig. 1).

Figure 1. A general impression of the arrangement of experiments for screening resistance to barley leaf stripe in the greenhouse.

Inoculation with the monospore culture was performed by placing 150 healthy, untreated seeds between two mycelium-over-grown agar plates (the "sandwich method" (Fig. 2)) (cf. SHANDS, 1934; HUSTON and OSWALD, 1948; KNUDSEN, 1986). After germination as mentioned above, the plants were transferred to the peat-rich soil in 28.5 x 46 cm trays without replicates. One hundred and seventy-nine barleys were selected for this test.

The plants were kept free from powdery mildew by spraying with Bayleton (25% triadimefon WP) both in the greenhouse and in the field. This fungicide does not affect the attack of leaf stripe.

Figure 2. Germinating barley seeds between two mycelial layers of a monospore culture of *Drechslera graminea*, *i.e.* the sandwich method.

The percentage of attack was measured at growth stage 10-10.5 (cf. LARGE, 1954/55) in the greenhouse, but in the last experiment with varieties and lines in the greenhouse where the attack was also measured at stage 1-2.

Several experiments have revealed an almost continuous variation from full susceptibility to full resistance to leaf stripe among the barley varieties, leaving the impression that several or many genes may be involved (cf. KNUDSEN, 1980; SMEDEGAARD-PETERSEN and JØRGENSEN, 1982). It is, however, difficult to analyse the inheritance as the disease severity can be obtained only in terms of per cent diseased plants or ability to escape the disease. This makes differentiation in f_1 and f_2 impossible. This problem may be overcome by using chromosome-redoubled monoploids from an f_1 (the *bulbosum*-technique which makes it possible to obtain completely homozygotic lines (JENSEN, 1977)). The analyses of inheritance may then be made on the necessary number of plants of each genotype and repeated under different conditions. (The details are given in Chapter 5).

As far as possible, the barley varieties and lines are set up in a number of pedigrees according to their closer relations. These are conferred with pedigree registers and outlines of inheritance (STARK, 1948; AUFHAMMER et al., 1958, 1968, 1976; HORNE, 1959 and following years; WIEBE and RIED, 1961; TORP et al., 1978; BAUM et al., 1981, 1985; ARIAS et al., 1983; MULTIMAKI and KASEWA, 1983; LARSEN, 1986) and personal communications from several breeders (Figs 6-12). These figures are formed as chessboards, and the barleys are listed alphabetically (Tab. 3) in order to make it easy to find their position in the figures. For instance, Alfa 9:1A means that this variety occurs at position 1A in Fig. 9. Underlined positions (Tab. 3) indicate where the ancestors to a variety or line is given. Varieties or lines marked with an asterisk in the table and surrounded by a dotted line in the figures were not tested in the present investigations.

Differentiation of levels of resistance or susceptibility was limited to the following rough scale due to the amount of plants tested of each variety or line:

% diseased plants	Designation
0-<5	Highly resistant when the barley had this level in both tests
0-<5	Resistant when the barley was only tested after field inoculation
6-15	Moderately resistant
16-40	Susceptible
41-100	Highly susceptible

The first two levels are taken strictly. Between the three last levels there may be some overlapping. This is taken into consideration in the pedigree analysis - especially concerning the occurring additive effects.

3. SYMPTOMS

3.1. Results

Barley leaf stripe is a one-year-cycled seed-borne disease for the reason that *D. graminea* can be transferred only from one plant to another via the seeds. Further, the disease can develop only if the fungal hyphae via coleorrhiza reaches the embryonic leaves before the seminal roots break through coleorrhiza during germination (*cf.* SKOROPAD and ARNY, 1956; PLATENKAMP, 1976). These peculiar requirements for attack are very sensitive to environmental conditions and constitute the reason why the most characteristic feature of leaf stripe is the ability to escape the disease and why resistance or susceptibility can be measured only as a percentage of attack or escape. It has been said in this connection that the resistance appeared to be a form of hypersensitivity (SKOROPAD and ARNY, 1956; SMEDEGAARD-PETERSEN, 1976).

Generally, the disease is characterized as systemic in the sense that all parts of a sick plant contain the fungus and may develop the symptoms, and further that these plants are smaller (stunted), paler green, less vigorous than the healthy ones, and sterile.

These facts were also valid in the present investigations but with characteristic exceptions, especially concerning the very resistant varieties and lines.

Regarding the resistant barleys, it is not only obvious that they are better able to escape the disease, but also that at least the most resistant varieties have the ability to get rid of a leaf stripe attack once attracted (Tab. 2). Besides this, our studies of the inheritance of the Vada-resistance (cf. Chapter 5) disclosed that the very few attacked plants in the highly resistant lines - instead of the common leaf stripe symptoms - displayed unique, characteristic symptoms under certain conditions. The leaf stripe became visible only as a single, narrow, yellow streak on the middle of the leaf blade, and this

Figure 3. The common symptoms of barley leaf stripe on the redoubled monoploid No. 97 with the growth type of 'Zita'.

streak did not always reach the brown stage. Plants with that symptom were darker green than the healthy ones and rather dwarfed than merely stunted, as was the case with plants with the common leaf stripe symptoms (Fig. 4). Further, these plants tillered in many cases in contrast to what is the normal when 25 plants are grown in 16-cm pots. The shoots did not extend unless the plants were transplanted to other pots, however. There they

either died, continued tillering or developed into normal plants either with or without weak leaf-stripe symptoms. These symptoms were later found also in other highly resistant barleys.

Table 2. Examples of barley varieties getting rid of leaf stripe once attracted.

Variety	Percentage of plants with symptoms at	
	stage 1-2	stage 10-10.5
Uffe Sejet	4.4	0
Koru	5.2	0
Modia (two samples)	5.6	0
Georgie	9.9	0
Valkie	11.1	0

Regarding the susceptible barleys, the diseased plants developed the leaf stripe symptoms (Fig. 3) with the exception of a few plants that died early in the development and of a few others where the disease was not systemic as one or two shoots extended and produced an ear. We were, however, unable to distinguish whether or not any difference occurred in this respect concerning the levels of susceptibility.

As in the case of susceptible barleys, plants in the highly resistant varieties may die early in the development, even before or soon after the seedling leaves emerge. This tendency seems even more pronounced in these cases.

Plants of the susceptible barleys may also be oppressed or dwarfed, and the result of a transfer of such plants to other pots could hardly be distinguished from that with the tillering dwarfs of the highly resistant barleys. These cases were rare exceptions and not the rule.

The dwarfing-tillering symptom was most pronounced in the pots, less in the trays, and barely distinguished in the field.

Figure 4. Comparison of the common and the dwarfing symptoms of barley leaf stripe. Left, the redoubled monoploid ('Zita' x 'CI 6944') No. 94 with common symptoms on the bunch to the right. Right, the redoubled monoploid No. 35 with the dwarfing symptoms on the bunch to the right. Type of growth is that of 'CI 6944'.

3.2. Discussion

RAVN (1900) described the barley leaf stripe symptoms so clearly and in so much detail that it has never been improved on. Concerning the susceptible barleys, our results are in complete agreement.

In the description, RAVN (1900) presented figures as well as drawings that show how much weaker and reduced in height were the diseased plants compared to the healthy ones. Several later authors use the term "stunting" for this phenomenon (*e.g.*, CHRISTENSEN and GRAHAM, 1934; ELLIS and WALLER, 1973; TEKAUZ and CHEIKO, 1980). This indicates why the specification is invalid

in connection with the dwarfing-tillering plants in the case of attack in the highly resistant barleys (not considered in SKOU and HAAHR, 1984).

Our observations of diseased plants with one or two healthy shoots are in agreement with RAVN (1900) and DRECHSLER (1923) who mention such cases in connection with a discussion of the analogy of leaf stripe with the systemic smut diseases of cereals.

Our results indicate that complete resistance or immunity to leaf stripe hardly exists, as even plants of the most resistant barleys may be attacked. However, when this happens both host and pathogen struggle because of the mutual effects. In this connection, the impression was often given that the dwarfing-tillering plants formed only lateral shoots. The explanation for this might be a killing of the main shoot at the stage of differentiation. This suggests a close agreement with SMITH (1929) who summarized his results 1) in death when meristem was invaded, 2) in leaf stripe when there was a lateral invasion, and 3) in escape. He noted in this connection that "death may come with varying speed, leaf stripe may be developed to varying degrees, and escape may be temporary or permanent". For these reasons there may be transitions between the three possibilities which may lead to the situation we observed. Further, it points to two types of escape: 1) the case where the plants appear healthy during the whole growing period, and 2) when plants get rid of the disease once attracted (cf. Tab. 2 and Chapter 5).

CHRISTENSEN and GRAHAM (1934) note that deviations from the systemic attack were due to the varieties in question rather than to the isolate of the pathogen applied. Stunting was more pronounced in 'Svansota' than in 'Minsturdi', but they found considerable evidence that the degree of stunting was influenced by the race of pathogen involved (cf. KNUDSEN, 1986). The degree of stunting could probably also be influenced by the growing conditions as we found less stunting of the infected plants under the controlled conditions in the greenhouse than in the field; furthermore, it is likely that the degree of stunting is affected

by the attack on the root system (cf. RAVN, 1900; STELZNER, 1934).

The inoculum potential and the incubation conditions influence the results strongly. The stronger the conditions, the more severe will be the seedling infection and post-emergence death (ELLIS and WALLER, 1973) and the lesser the escape. So, in our experiments, the inoculation in the field followed by testing in the greenhouse appeared less intensive than did the inoculation using the sandwich method.

Finally, it is questionable if the disease can occur to a larger extent in a latent stage and in this way cause yield loss as found by ZADE (1932). At least, we have not found these results verified anywhere.

4. SCREENING FOR RESISTANCE

4.1. Results

4.1.1. Disease screening

The barley samples received had not been seed dressed with chemicals for disease control in prior years; this gave us the first possibility for a rough screening for resistance to leaf stripe. In the following years, all 1029 barleys were screened in the greenhouse on the basis of inoculation in the field as described under Material and Methods. The most severe attacks on the barleys in these experiments were then set up in groups with 5% differences in attack (Fig. 5). This shows that a large number of the barleys had a considerable degree of resistance. Not less than 436 (42.4%) had 10% attack or less, and of these 69 (6.7%) were not attacked at all.

One hundred and sixty-six of the most resistant of these barleys (0-5% attack) and 13 others that were more or less susceptible were selected for testing with the sandwich method. Of these barleys 43.6% - mainly those without attack after the field in-

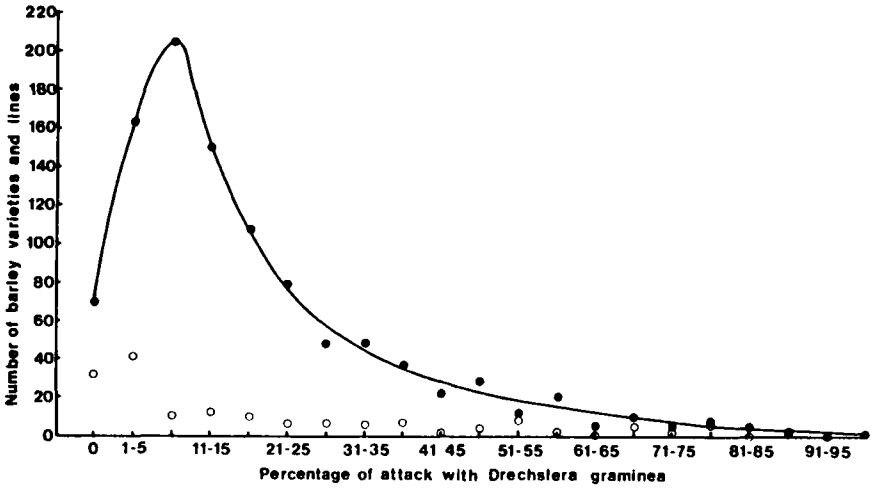

Figure 5. Distribution of 1029 and 166 barleys plotted as to level of attack with barley leaf stripe in the greenhouse, (●) upon inoculation in the field, and (O) upon inoculation with the sandwich method, respectively.

oculation - were still highly resistant. To a varying extent many were more susceptible under these conditions than after the field inoculation, and only one had significantly less attack.

4.1.2. Pedigree analyses

As hundreds of the northwest European barleys are more or less closely related we had here a good possibility to see if the results obtained exhibited any line or lines of inheritance of reaction to barley leaf stripe. Towards this aim we set up these barleys according to their closest relations in eight pedigrees formed as chess-boards (Figs 6-12). We then listed alphabetically the tested barleys as well as those untested but mentioned in the pedigrees (Tab. 3) in order to facilitate the finding of their positions. For each variety and line the percentage of attack is given after the name or number, and the figures for inoculation in the field and for inoculation in the sandwich test are separated by an inclined quoin: field inoculation/sandwich inoculation. The result presented in the pedigree is the same as that occurring in the sample that was provided directly from

the breeder in the few cases where there were significant differences between two or more samples of the same variety.

4.1.2.1. Highly resistant barleys

Only twenty-one (fifteen descend from 'Vada' or 'Minerva', two from 'Opal' and 'Seger', two from a Bohemian landvar., one from a Finnish landvar., and one as contamination in 'Kenia') of the more or less interrelated northwest European barleys may be regarded highly resistant when this term is limited to those with less than 5% (max. 4.9%) attack after both methods of inoculation. Quite a number with this level of resistance were found in other sources (Tabs 4-6), however.

A. 'Vada' derivatives. Fifteen of these highly resistant barleys may be traced back to a cross made in 1928 (DROS, 1957) in the Netherlands between 'Svalöf Gull' (Gold, Guld, Kulta) and *Hordeum laevigatum* (a so-called botanical species, taxonomically a *nomen invalidum*), the donor of the *Laevigatum* powdery mildew resistance (Figs 6:4-5A and 7:4-5A). The cross resulted in the varieties 'Vada' (Fig. 6:1-8A), and 'Minerva' (Fig. 7:1-8A) of which the former never became attacked in our experiments and the latter had 10% attack after field inoculation.

'Gull' 14/52 (Fig. 6:5A and cf. Tab. 3) was susceptible in our screenings. Besides being parent of 'Vada' and 'Minerva', it was parent of the sister varieties 'Opal', 'Kenia' and 'Maja'. Further, the pedigree analysis disclosed it as parent in several other cases but it resulted in no cases in highly resistant varieties. A line of *H. laevigatum* was also included in our screening where it came out with 15% leaf stripe after field inoculation. So, the high resistance could neither come from this line. In the pedigree analyses, *H. laevigatum* appeared further in two cases, namely in one where 'Gull' 14/52 x *H. laevigatum* (Fig. 9:2CE) ends up in an untested variety and in another where 'Jo 0758' x *H. laevigatum* gave the highly resistant 'Jo 1341' 4/1 and the significantly more susceptible 'Jo 1394'13/- (Fig. 11b:8AB). 'Jo 0758' is untested but back in the ancestors occur 'Bethge III' 7/- which is related to highly resistant varieties

Table 3. List of barley varieties and lines in the figures of pedigree and tables. Underlined positions in the pedigree figures indicate where the ancestors are presented. Untested barleys are marked with an asterisk. Barleys in brachets are mentioned only in the text. The parents of some varieties and lines of supplementary interest for the relations in the pedigrees are inserted in brachets after the varietal name. The accession number of the barleys in our collection is given in brachets.

Varieties and lines	Figures / Tables
* Abacus	6:5F
* Abed 0625 (Byg 191 x Carlsberg^3) x (Long Glumes x Emir x Lofa)	6:4DE
* Abed 3371	6:5BC 7:8CD
Abed Archer (255)	12:3CD
Abyssinian (588)	Tabs 10, 11
Abyssinian 39 (782)	Tab. 10
Adolphe Bungener, Secobra (444)	Tab. 8
Agio, Cebeco (431)	9:1C 9:3BC
Agneta Sv. (834, 1025)	11a:2D Tab. 9
(Aka Sinriki)	p. 40
Akka W (921, 1055)	9:7B
Albert W (44, 858)	8:6EF Tab. 11
Albert Busser, Secobra (443)	Tab. 8
Albion W (1059)	8:3CD Tab. 11
Alf, Carlsb. (839)	7:5B Tab. 11
Alfa, St. Kontrol. (254)	9:1A
Algerian (624)	9:7EF 10:7E
* All. 297 (Proctor x Ingrid)	7:5C
Alva Sv. (859, 1026)	6:1CD 6:3B Tab. 11
* Am.	11b:3A
* *Amer.* smooth-awned	10:4DE
Amsel Heine (779)	10:8C
* Andie	11a:2B
Anla Sejet (783)	7:1B
Anna Abed (860)	6:4E Tab. 11
Anoldium (628)	Tab. 8
Ansgar W (819, 1060)	8:3-4BC 9:5E Tab. 11
Arabian (799)	9:1B 9:3B
Aramir, Cebeco (842)	7:5AB 9:4BC Tab. 11
* Archer	12:2D 12:3D
Archer 23 (256)	12:2BC
Archer 35 (257)	12:1E
Archer 217 (258)	12:2E
Archer, stiff straw (259)	12:2BC
* Archer Goldthorpe 4-51 (Irish Archer x Goldthorpe)	12:2AB
Ariana (CI 2524) (760)	Tab. 10
Ark Royal, RPB UK (885)	6:5DE 9:7AB Tab. 11
Arla W (791, 1053)	8:6CD 8:8CD 9:7AB 10:1BC
* Arla W_1 W	8:5F
Arlington (487)	Tab. 8
* Armelle	6:5C
Arra, Jo. (Jo 1184) (944)	11b:4B Tab. 9
Asplund Sv. (13)	10:1B 11a:1AB 11a:1DE 11a:3CD 11a:4DE 11b:6C Tab. 9
Atlas (620)	Tabs 10, 11
(Australian Chevallier)	p. 54
(Australian Early)	p. 54
(Australische Fruhe)	p. 54
* B/78-1	7:1CD
* Baladi 16	8:5AB 9:6B
Balder W (260, 1066)	6:2AB 6:7DE 7:7B 8:2C 8:4BC 8:5C 9:1DE 9:3E 10:2D 11b:3F
Balkan (589)	Tab. 8
Bavaria, Ackerm. (488)	9:5AB 10:6-7DE
Bay (A64)	11b:5-6CD Tab. 11
(Bearer, cf. Plush)	
Benedicte W (1069)	8:4DE
Bente W (1051, 1075)	7:8C
Beorna, Cereal St. Eire (445)	9:1BC
* Bestehorn Diamant	10:1DE 10:4A
Bethge II (491)	12:7-8C
Bethge III (491)	11b:7A 12:7-8D
* Bethge III, mutant	12:8D
Bethge XIII (492)	12:8CD
* Betina	6:2E
Betzes (899)	12:8CD
Betzes (CI 6398)	12:8CD Tab. 11
Binder Abed (261)	6:2CD 8:4A 8:2B 8:4B 8:5B 9:4A 10:1-2A 10:6A 10:1BC
(Binder Abed 12 (481))	p. 45
(Binder Abed 35 (482))	p. 45
Bine PF (835)	7:7B
Birgitta Sv. (856, 1024)	7:6EF 8:6DE 10:3AB Tab. 11
Birka W (861)	8:6B Tab. 11
Bjørne (45, 988)	11a:3CC
Black Hull-less (625)	Tabs 8, 11
Black Hull-less (CI 2777) (893)	Tabs 8, 11
* Black kern. 2r.	7:1AB
Black Russian (763)	Tab. 4
Black smooth-awned (76?	Tab. 5
Bode, Vagønes Norway (1011)	11b:2A Tab. 9
* Bohemian Landrac.	12:7CE
Bomi Abed (784)	7:5B
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Ricardo (762)	Tab. 4
Rigel Abed (321)	9:2AB 10:2B 10:6-7CD
	11a:4EF
Rika W (400, 1049)	6:3B 8:4AB 8:5B 9:6AB
	10:7D
Ringve, Vollebek Norway (1006)	11a:4D Tab. 9
Roland W (1061, 1076)	6:5AB
Rupal Sv. (817, 1040)	7:5CD Tab. 11
• Rupee	7:4D
• Russian 2	10:8E
R 22 (select. from Crimean landvar., USSR)	10:8E
Salka PF (806)	6:1D 6:8B 9:2DE Tab. 11
Salve Sv.(1043)	8:7EF 10:3B
Sammy Sv. (1035)	9:7BC
• Scandinavian landvar.	7:6CD 10:2E
Scots Bere (592)	Tab. 10
Scots Common (593)	12:5AB
Scottische Annot (324)	Tab. 5
• Seed sample	11a:1DE
Seger Sv. (Sejr, Victory, Voitto) (323)	6:2BC 7:4C 8:1B 10:3C
	10:4C 11a:5CD 11:2BC
Sejet 51/1732 (678)	7:1CD
Senat Sv. (1034)	9:5CD Tab. 11
Seta Sejet (786)	7:1BC
(Shinriki)	p. 40
(Shiro Chinko)	p. 40
Sigur (923)	Tab. 10
Silje, Jo. (932)	11b:2B Tab. 9
Simba Sv. (1039)	7:2BC Tab. 11
Siri PF (795)	9:5C
6-rowed, naked (12)	Tab. 8
Sjåk (1000)	Tab. 10
Sold, Ås Norway (966)	7:6AB
Sort byg (423)	Tab. 8
Spain (738)	Tabs 10, 11
Spartan (697)	11b:5D Tab. 11
• Spratt	12:2B
• Spratt Archer	8:1BC 12:2C 12:6D
Spratt Archer 37-3 (416)	12:3AB
Spratt Archer 37-4 (328)	12:3BC
Spratt Archer 37-6 (432)	9:1BC 12:3CD
Standwell, Gartons (229)	12:6E
Stange, Ås Norway (960, 963)	7:6BC
Stallar W (329)	10:2D
Stallar II W (437)	9:4AB
Stella Sv. (1137)	Tab. 8
Stensgård (330)	Tab. 8
Sterling (331)	10:5D
Stewart (CI 6112) (896)	Tabs 8, 11
Stjernebyg (796)	Tab. 10
Strengs Franken (syn. of Strengs Hadostreng) (556)	8:2CD
Strengs Franken II (557)	8:2DE
Strengs Franken III (558)	8:4DE 9:3CE
Strengs Hadostreng (Hado x Streng) (559)	8:2CD
Støvring (458)	Tab. 8
• ST 1119-59	12:1A
Sultan, Cebeco (792)	9:2CD Tab. 11
Sune Sejet (1077)	7:2-3DE
Susan PF (981)	9:2D Tab. 11
Suvi, Jo. (Jo 0747) (933)	11b:2B 11b:3AB 11b:7CD
• Sv. 2148	6:2-3B
(Sv. 60210)	p. 47
• Sv. 60718	11a:5E
• Sv. 66433 (cf. p. 47)	7:3DE
Svalöf (560)	Tab. 10
Svanhals Sv. (230, 1029)	8:7BC 10:1DE 11a:7-8CD
(Svansota)	p. 17
Swallow Heine (778)	9:6B
Sørla W (1057)	11a:8B Tab. 11
(Sørum (984))	p. 56
• Ta 04369	11a:6B 11b:1E
• Ta 05864	11a:6D 11b:1F
• Ta b7990	11a:2A
• Ta b7990 (xR)	11a:2AB
Tammi, PBS Tammisto SF (80, 907)	8:7DE 10:1B
	11a:1AB 11a:2C
	11b:2A 11b:7C
Tampar (924)	Tab. 10
Taarn Sv. (1078)	7:5E
Teemu, Jo. (934)	11b:2AB
Tellus W (1064)	8:3-4DE 8:5BC 8:5DE
• Tellus D_1 W	8:6B

Continues next page

Table 3 continued

Varieties and lines	Figures / Tables
• Tellus M_1 W	8:5C
• Tellus M_1O W	6:5BC
Tern, Heine (803)	10:7-8C Tab. 11
• Tervakanges	11b:7B
Tibetania (568)	Tab. 8
Titan (438)	11b:6DE Tab. 11
Togo, Carlsb. (882)	7:5C Tab. 11
(Tokushima Mochi)	p. 40
Torkel W (883)	9:7DE Tab. 11
Trebi (CI 936) (446)	11b:6E Tab. 11
Trebi 101 (473)	Tab. 8
Trebi 628 (474)	Tab. 8
Tregal (462)	11b:6E Tab. 11
• Triple awn lemma	9:5DE
Triumph (Trumph, DDR) (884)	Tabs 10, 11
Trofimovskaja (698)	Tab. 4
Tron Sejet (829)	9:4EF 10:3EF Tab. 11
Trysil (83)	Tab. 7
(Trysil (979))	p. 41
Tunga, Vollebek Norway (1004)	Tab. 10
Tyra PF (825)	10:7D Tab. 11
Tystofte 25 (84)	Tab. 7
Tystofte Kors (62, 886)	Tabs 5, 11
Uffe Sejet (1070)	7:2-3CD Tab. 2
• Unikum Hind.	11b:7BC
• Union	6:8D 7:7-8B
• Universe	6:6B
Vada, Fonds BVL NL (731)	6:1-8AB 6:2CD 6:5E Tab. 11
Valkie (629)	Tabs 2, 4
Vanja W (1063)	Tab. 8
• Vankkuri	11a:1A 11b:2AB 11b:7B
Vantage, Brandon (442)	11b:6C Tab. 11
• Vatonga	7:3D
Varde, Vidars. Norway (606, 987)	11a:1E 11a:4B 11b:3D Tab. 9
Varunda, Fonds BVL NL (843)	6:8B Tab. 11
Vega Abed (837)	7:2E Tabs 8, 11
Vega Sv. (86)	8:6BC 10:3BC 11a:1BC 11a:5D 11b:2CD Tab. 9
Velvet (479)	Tabs 10, 11
Velvet (CI 4252) (Luth x (Manchuria x Lion)) (906)	*Tab. 11, p. 81*
(Velvet C.A.N. 1133) (411))	p. 80
(Velvet Ottawa (480))	p. 80
(Velvet 447 (478))	p. 80
Velvon, USDA (470)	11b:6E Tab. 11
Vena, As Norway (1001)	11a:5B Tab. 9
Victoria (Bot. Garden, Belfast) (335)	Tab. 8
Vigdis, Vollebek Norway (982)	11a:5BC Tab. 9
Visir Sv. (1033)	7:5EF
Vogels mildew resistant (573)	Tab. 7
(Voldaksen)	p. 48

Varieties and lines	Figures / Tables
• Volla (Breuns Wisa x Heines Haisa I)	9:8BC 9:6BC
(Vulkan)	p. 63
Vyatka 1009 (88)	Tab. 10
Vå 13002 (990)	11b:1B Tab. 9
Vå 14047 (1002)	11b:1BC Tab. 9
Vå 17128 (1012)	11b:CD Tab. 9
Vå 17165 (997)	11b:1DE Tab. 9
Vå 17236 (995)	11b:1E Tab. 9
• W 5853	9:8C 9:8F
• W 5925	12:1AB
• W 5926 (Proctor x (Balder x ((Balder x Opal) x (Proctor x Ingrid)))	9:7D
Warrior (CI 6991) (900)	11b:6EF Tab. 11
• Weihenstephan mildew resist. I CP (WMR I CP)	10:2E 10:4E
Weihenstephan mildew resist. II (WMR II) (577)	7:7-8CD 10:2E 10:6BC 10:7B
• *Weihenstephan mildew resist.*, source unknown (WMR)	8:4F
• Weihenstephaner 259 (Weih. 259)	7:7CD
Weisse Erfurter (578)	Tab. 5
Welam W (833, 1048)	9:8EF Tab. 11
White Catami (CI 920) (887)	Tabs 4, 11
White Smyrna (579)	Tabs 10, 11
Wing W (804, 1046)	8:2AB 9:5EF Tab. 11
• Wong	6:1DE
Wurla, Sweden (580)	Tab. 10
• WW 5749	7:8D
Yak Maris, PBI UK (897)	9:4B 9:6C Tab. 11
Ymer Sv. (337, 1036)	10:4C 10:5C 10:5F
Yriba, Desprez (1072)	9:6B
Yrjar, Vollebek Norway (989)	*11a:4BC Tab. 9*
Zeiners Deutschmeister (581)	Tab. 10
Zeiners Franken (582)	Tab. 10
Zeiners Frankenperle (583)	10:4DE
Zeiners Immune (585)	Tab. 7
Zeiners Universal (587)	Tab. 8
• Zephyr	6:4BC 6:5EF
Zita PF (807)	6:1B Tab. 11
Åsa Sv. (452, 1044)	10:3C 11a:2DE Tabs 9, 11
9-V-19 (717)	Tab. 6
9-v-19 (718)	Tab. 6
13-V-19 (719)	Tab. 6
• (13-v-19 (720))	p. 41
23-v-18 (730)	Tab. 6
• 57/510-44	11a:7AB
• 50-88	9:5C
• 65-44	7:3A
• 6-row barley	10:2-3A
• 6-row line	11a:5E
• 6-row, naked	Tab. 8

Figs 6-12. The inheritance of resistance to leaf stripe is outlined in eight series of pedigrees according to the closest relations of the barleys. 6. The 'Vada' series, 7. The 'Minerva' series, 8. The 'Opal' series, 9. The 'Kenia' series, 10. The 'Maja' series, 11a and 11b. The 'Alplund'/'Maskin' series, 12. Other series. The names of varieties together with the percentage of attack are placed in boxes with dot-and-dashed and full-drawn lines for untested and tested barleys, respectively. The percentage of attack after inoculation in the field and after the sandwich method is separated by an inclined quoin. The highly resistant barleys are set off in hatched boxes. Finding of the position of the single varieties or lines facelitates by referring to the coordinates.

The Vada Series

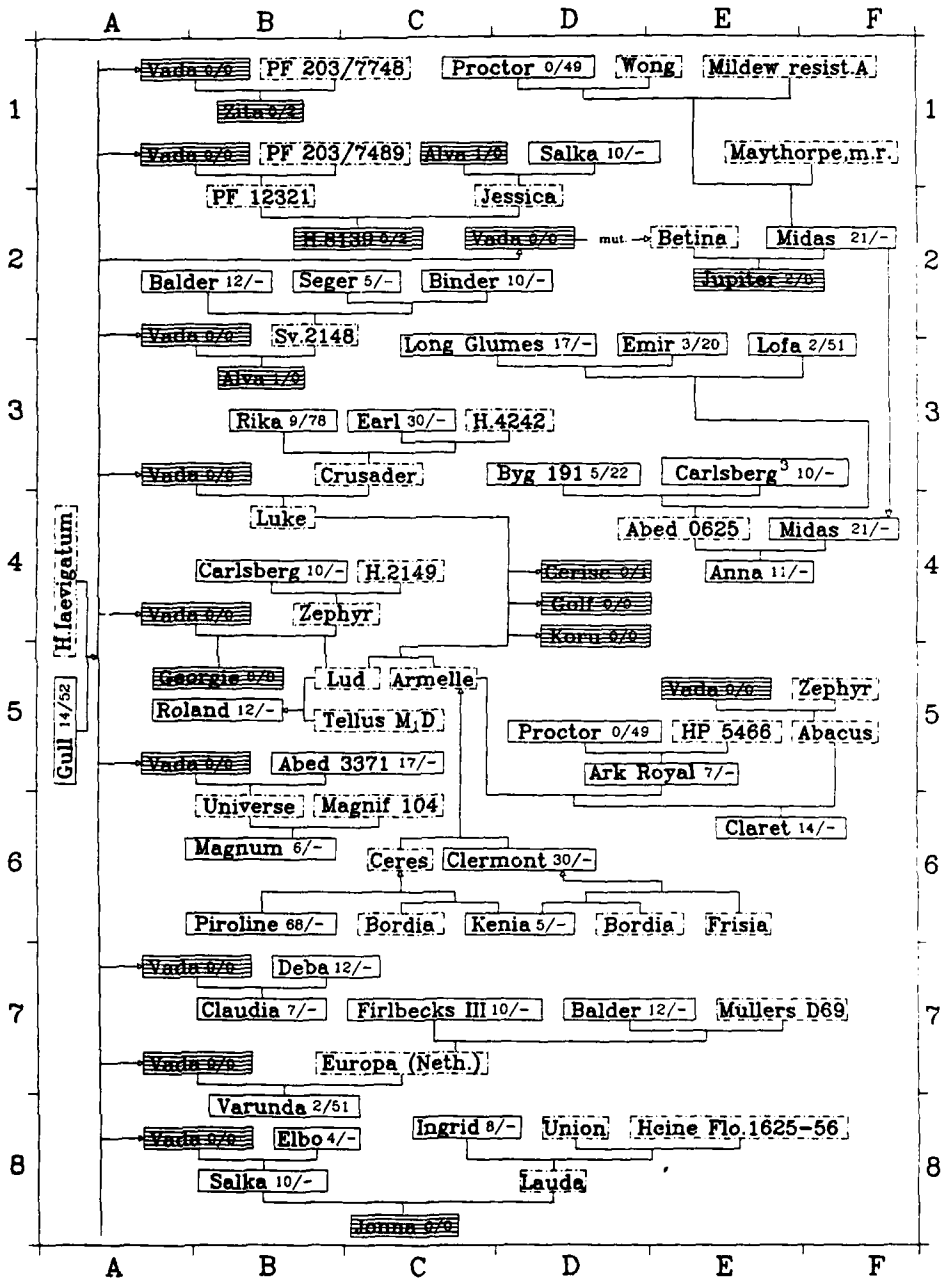


Figure 6

The Minerva Series

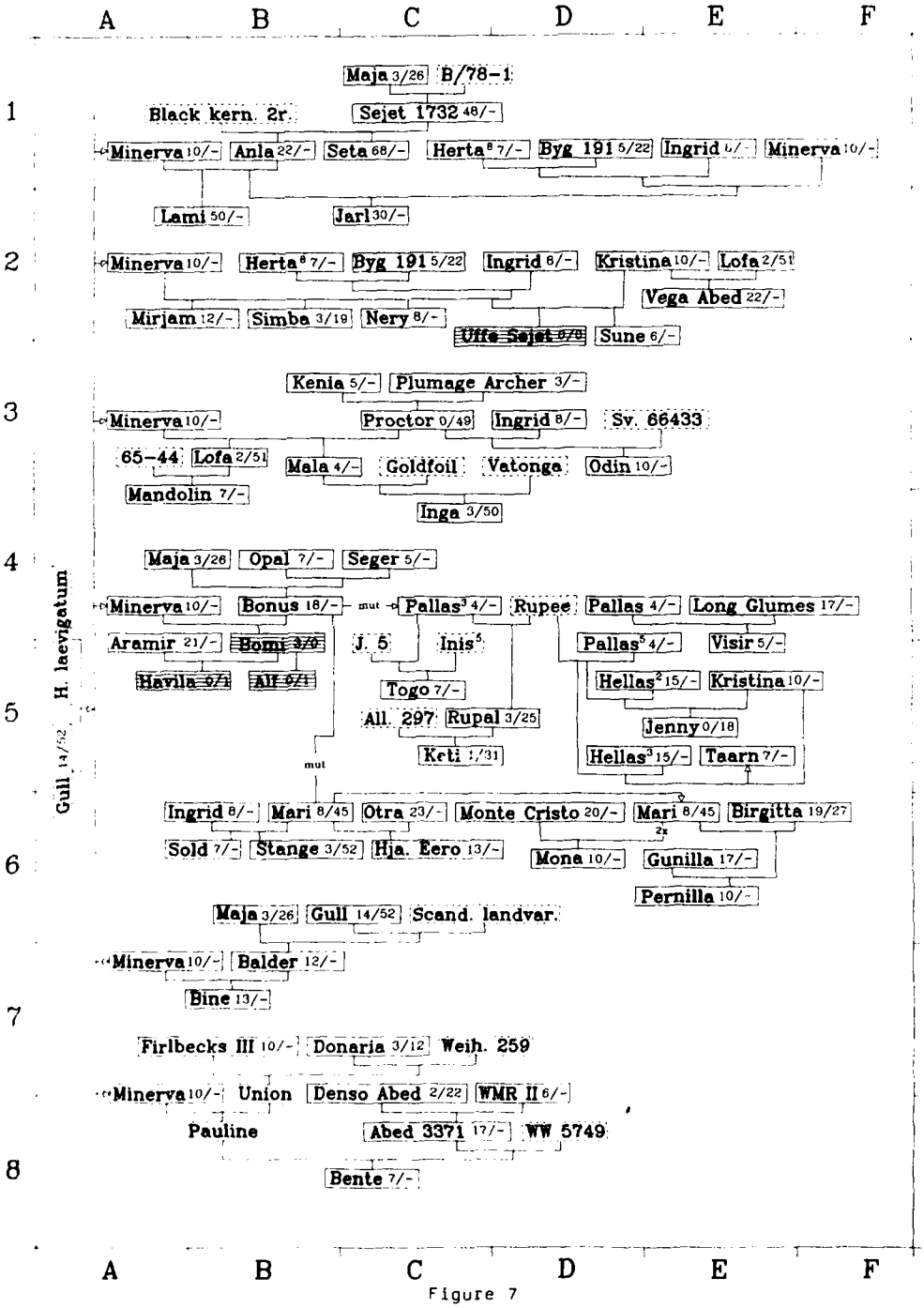


Figure 7

The Opal Series

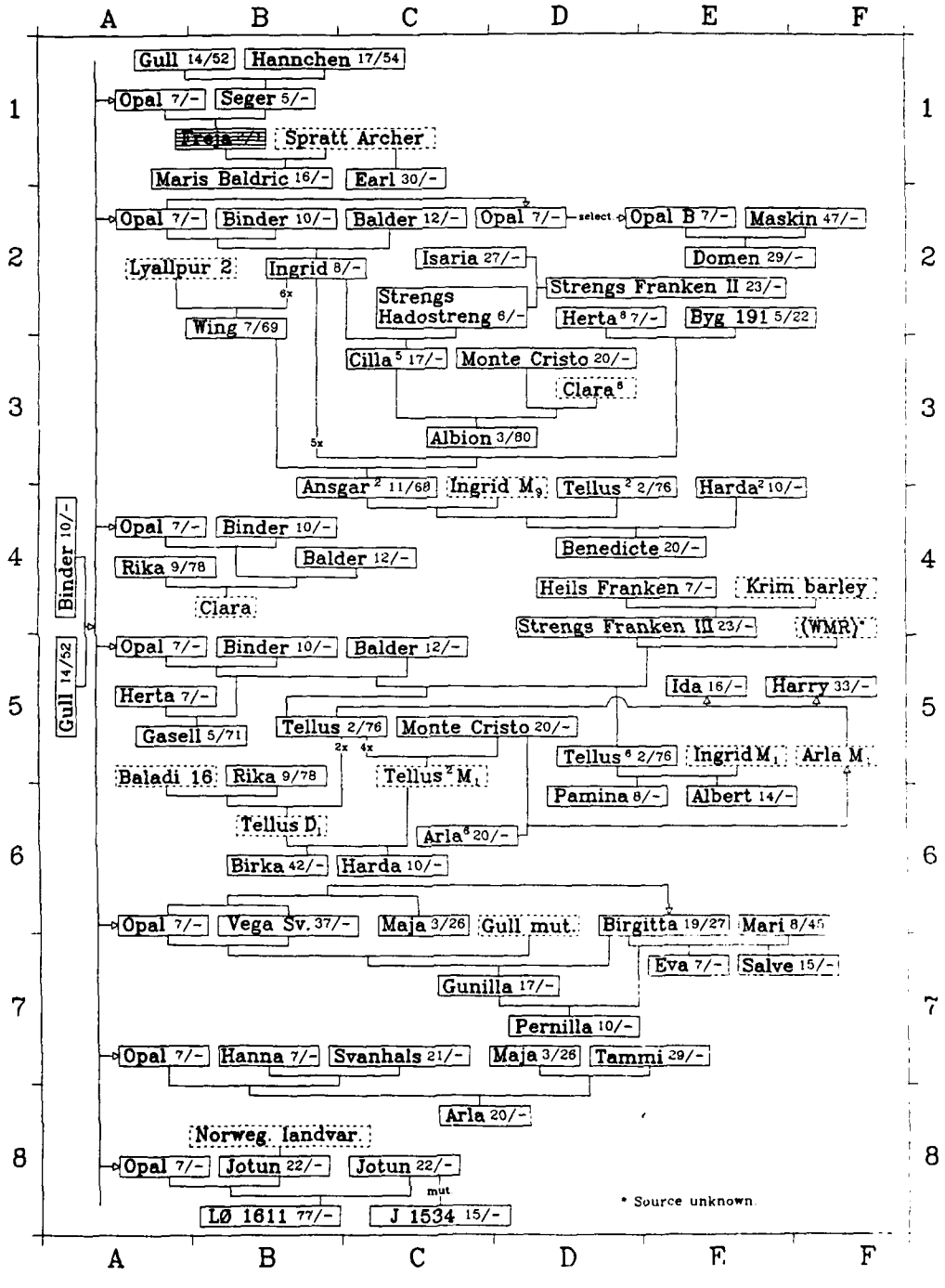


Figure 8

The Kenia Series

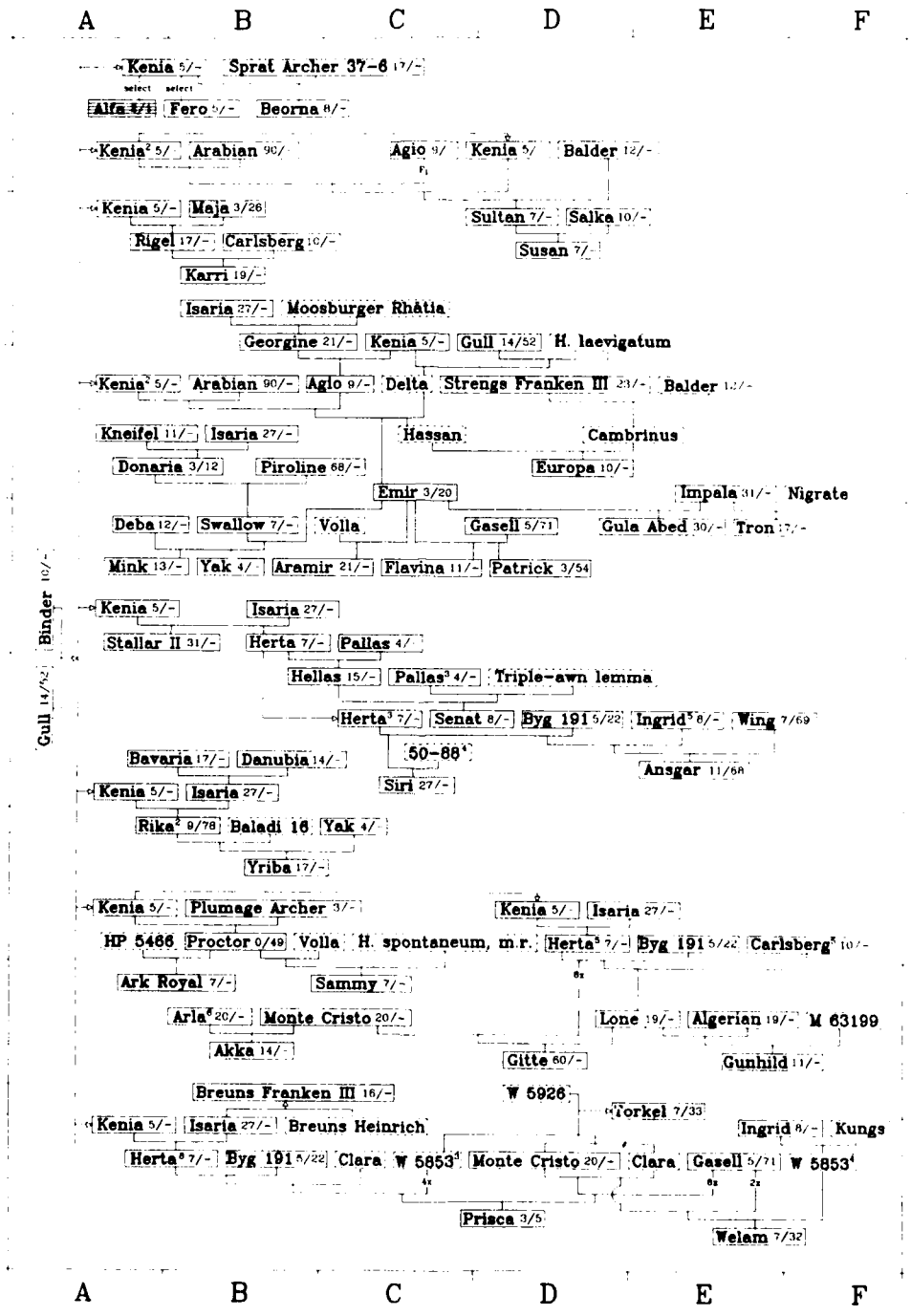


Figure 9

The Maja Series

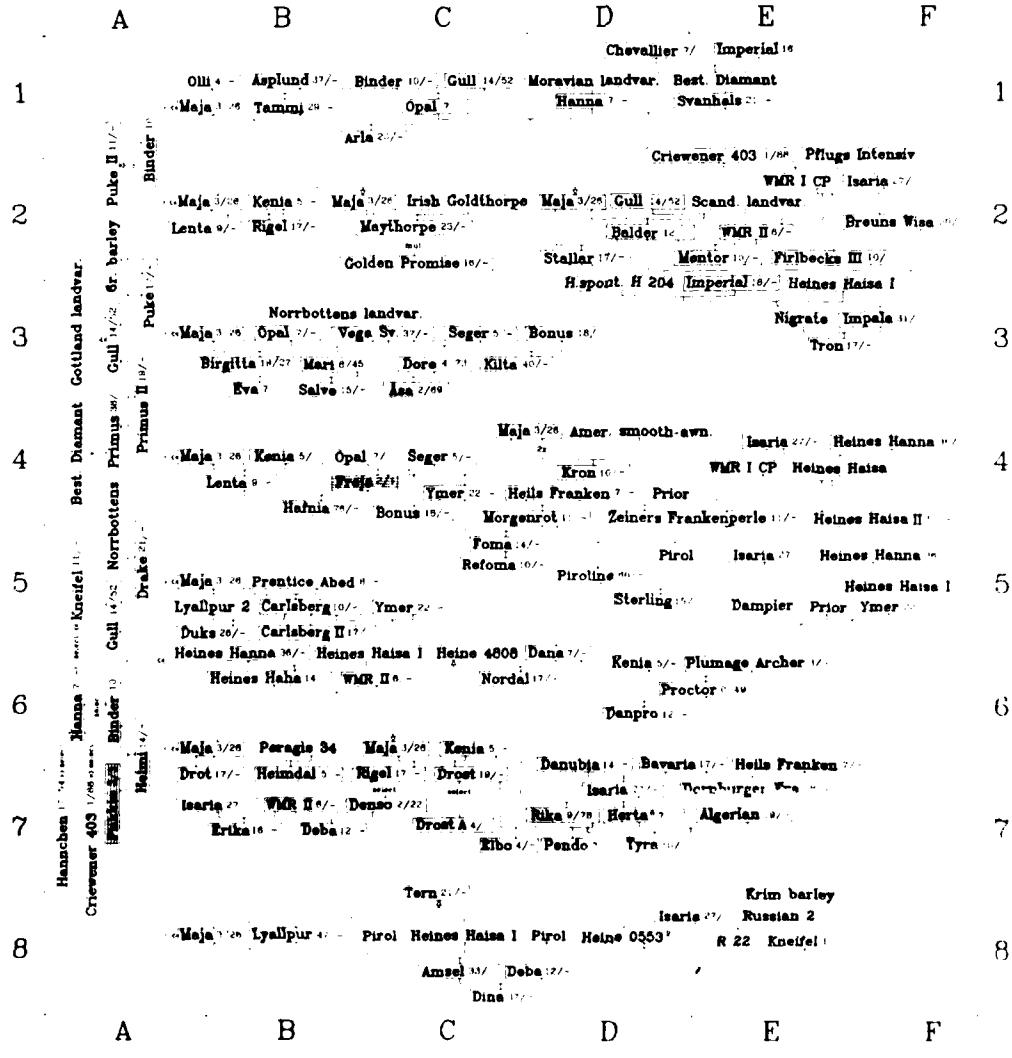


Figure 10

The Asplund/Maskin Series I

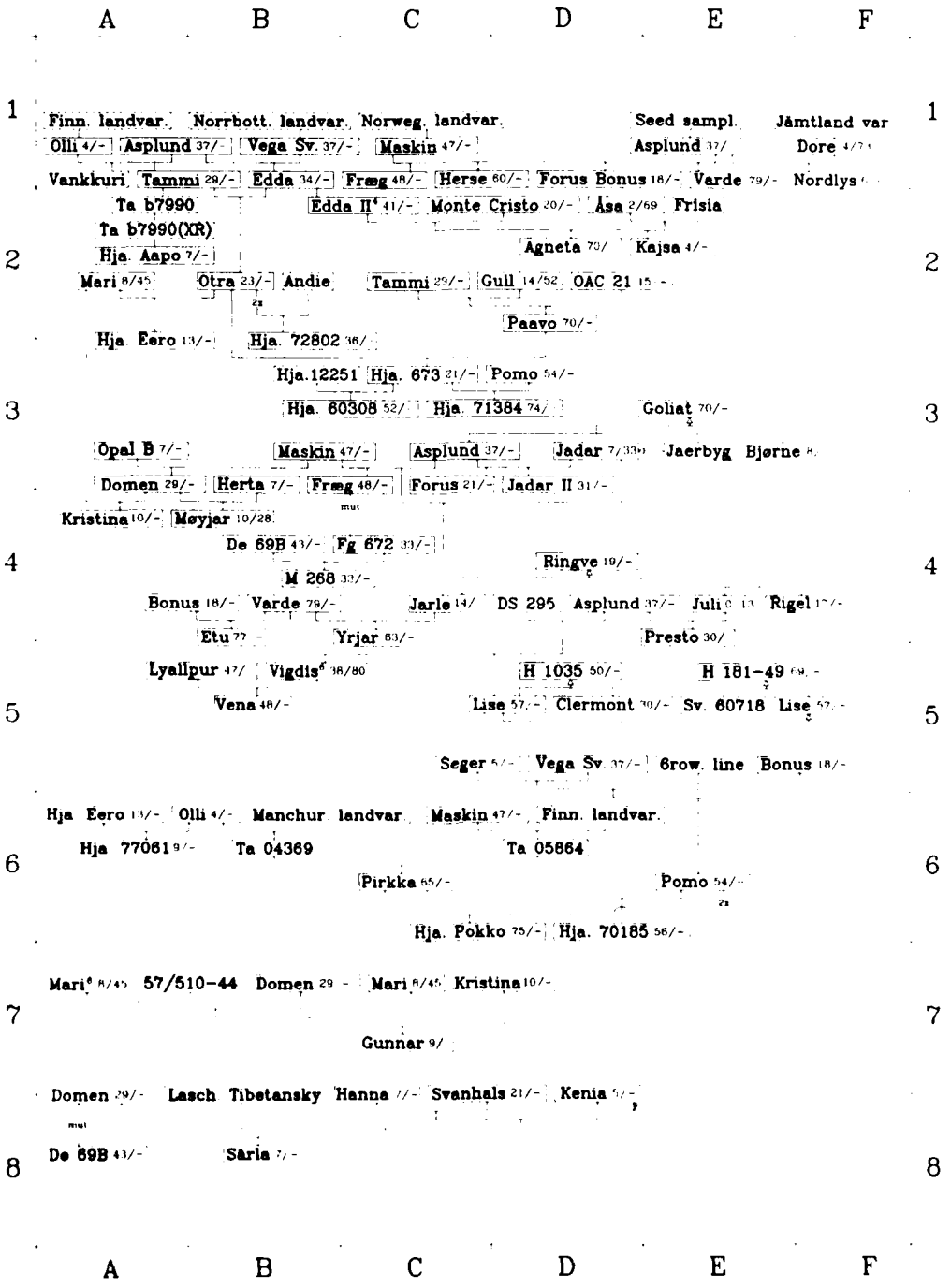
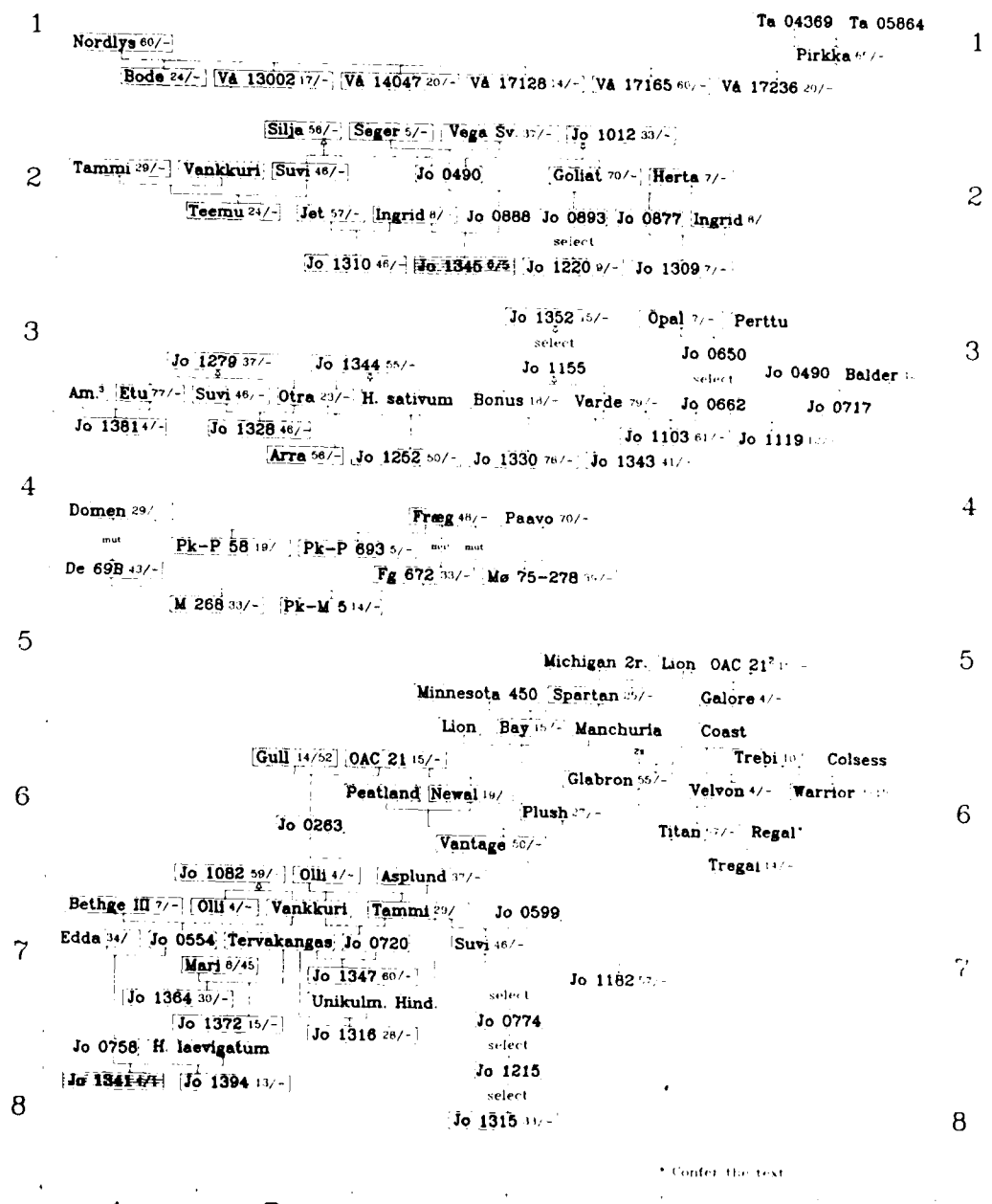


Figure 11a

The Asplund/Maskin Series II



* Confer the text

Figure 11b

Other Series

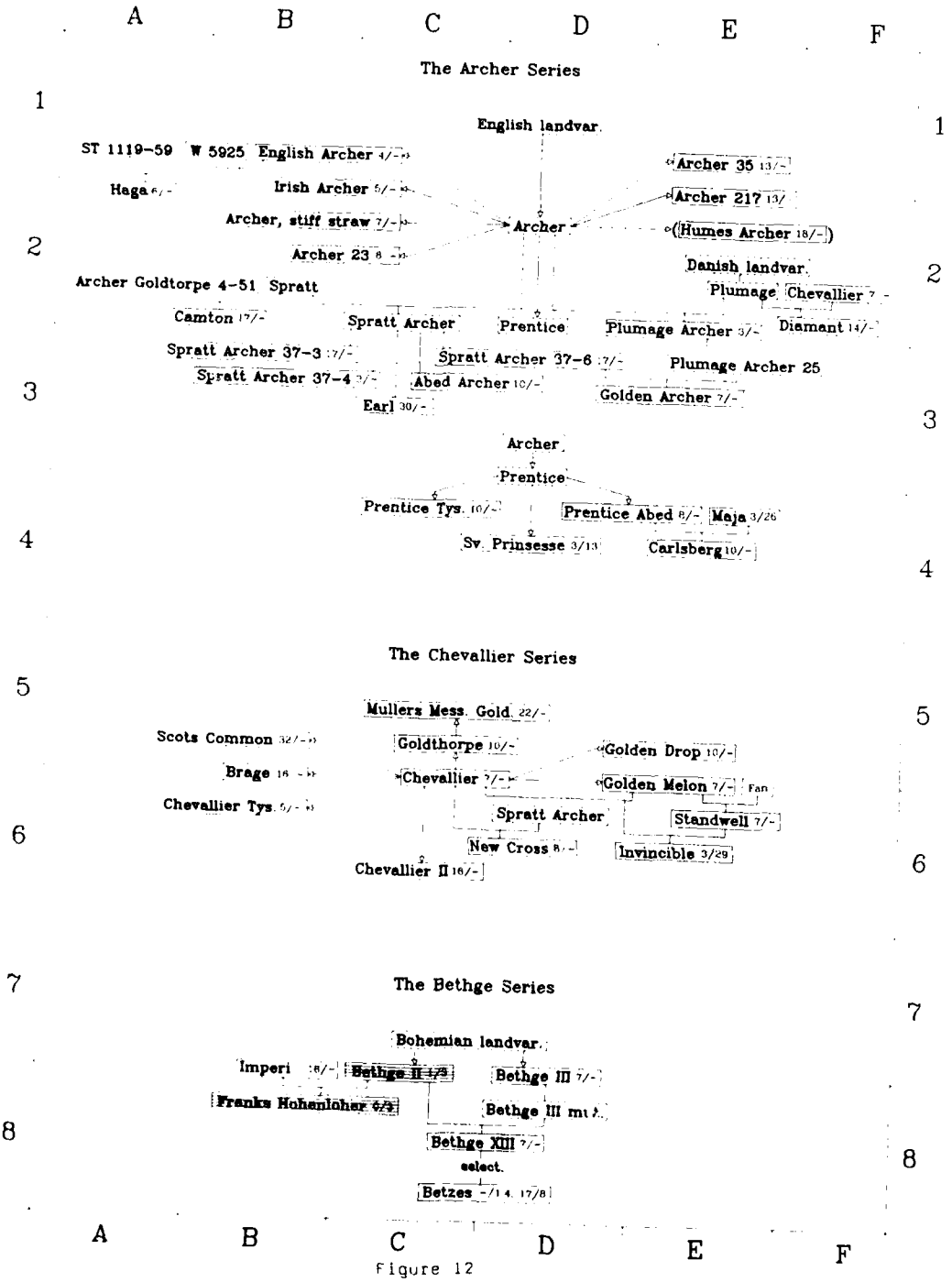


Figure 12

(cf. Fig. 12:7-8BD) and 'Olli' 4/- (Fig. 11b:6-7AC). They have both the same level of resistance as 'Jo 1341' 4/1 after inoculation in the field. So, there is a chance that one of these resistances is carried along to 'Jo 1341' 4/1 instead of the susceptibility which is equally present in the ancestors and may partly occur in 'Jo 1394' 13/-.

On the basis of this analysis, we find it most probable that this high resistance originates from *H. laevigatum*.

This leaf stripe resistance came apparently by mere chance into the barley breeding together with the *Laevigatum* resistance (gene *Ml-(La)*) to powdery mildew (*Erysiphe graminis* DC. f. sp. *hordei* Em. Marchal). The two diseases are, however, independently inherited as the resistance to leaf stripe occurs in only 15 out of 35 tested *Ml-(La)* powdery mildew resistant varieties. We propose the term "Vada-resistance" for this resistance that so often became transferred to the offspring. One Vada-resistant line ('Jo 1341', see below) was found without the *Laevigatum* resistance. Ten others have another type of resistance to leaf stripe and the last ten are susceptible or highly susceptible.

The Vada-resistance was first observed in the formerly widely grown variety 'Zita' 0/2 (KNUDSEN, 1980) (Fig. 6:1B). Other Vada-resistant varieties and lines were traced during the establishment of the pedigrees (Figs 6, 7, 11b). The Norwegian line, 'H. 8139' 0/2 (Fig. 6:2BC) came from the Pajbjerg line 12321 which is a sister line to 'Zita' (Fig. 6:2B) and 'Jessica Sv.', the parents of which both have 'Vada' as one of their ancestors (Fig. 6:1-2CD). 'Jupiter' 2/0 came from 'Betina', selected as a mutant in 'Vada', and the susceptible 'Midas' 21/- (Fig. 6:2EF). 'Vada' 0/0 x 'Sv. 2148', which is most likely less resistant, gave 'Alva' 1/0 (Figs 6:1CD, 6:3B). 'Vada' 0/0 x 'Crusader' gave 'Luke', and 'Vada' 0/0 x 'Zephyr' gave the highly resistant 'Georgie' 0/0 and 'Lud'. Neither 'Luke' nor 'Lud' were tested, but 'Luke' x ('Lud' x 'Armelle'), however, resulted in the highly resistant sister varieties 'Cerise' 0/1, 'Golf' 0/0, and 'Koru' 0/0 (Fig. 6:4-5D) which for these reasons we believe have the Vada-resistance.

'Jonna' 0/0 (Fig. 6:8BC) may have the Vada-resistance, but this requires that 'Salka' 10/- ('Vada' 0/0 x 'Elbo' 4/-) is or has been heterogenous. If this is not the case it must have got the resistance from 'Union' or the Heine line via 'Lauda' (Fig. 6:8AE).

B. 'Minerva' derivatives. A few varieties may have the Vada-resistance derived from 'Minerva' 10/-, but this requires that this variety is or has been heterogenous (cf. the discussion). 'Minerva' 10/- x 'Bonus' 18/- gave the highly resistant 'Bomi' 3/0 though none of the parents had a comparable resistance, and a cross between the susceptible 'Aramir' 21/- and 'Bomi' 3/0 resulted in the highly resistant 'Havila' 0/1, and the highly resistant 'Alf' 0/1 is a mutant in 'Bomi' 3/0 (Fig. 7:4-5AB).

Also 'Uffe Sejet' 0/0 may have got its resistance from 'Minerva' 10/- as it may have come neither from 'Kristina' nor from the complex crossing, ('Byg 191'* 5/22 x 'Herta'[8] 7/-) x 'Ingrid' 8/-. The level of resistance in the brother variety 'Sune Sejet' 6/- may have come from this complex or from 'Minerva' 10/- (Fig. 7:2AE).

C. Other highly resistant barleys I. Another six highly resistant barleys were traced in the interrelated northwest European series. 'Opal' 7/- x 'Seger' 5/- ('Victory',,'Sejr', 'Voitto') resulted in the highly resistant variety 'Freja Sv.' 2/1. However, as neither its parents nor their ancestors had that level of resistance, it may suggest an additive or transgressive effect from the parents (Figs 8:1AB, 10:4BC). An analogous situation holds for the Finnish line 'Jo 1345' 0/5 (Fig. 11b:2-3C).

The highly resistant 'Alfa' 2/1 should, according to the breeder, be a selection in the more susceptible 'Kenia' 5/-, but its

* Byg 191 = Barley 191 (Eng.) = pallidum 191 (French) = "*Hordeum pallidum cevada vulgare*" = No. 191 in the barley collection at the Royal Veterinary and Agricultural University, Copenhagen. Used as donor of nematode resistance (ANDERSEN, 1961).

real relationship is questionable and will be discussed below (Fig. 9:1A).

'Prisca' with 3 and 5.3% attack after the two methods, respectively, may or may not belong to the highly resistant barleys, but it is impossible to trace this pattern of resistance in its complex ancestry (Fig. 9:8A-E, and cf. the following section: D. The 'Opal', 'Kenia', and 'Maja' series, p. 49).

D. Other highly resistant barleys II. Forty other highly resistant barleys were not interrelated with the northwest European barleys (Tabs 4-6). Almost the only we know about them is that some of them have been tested also by others. This will be discussed below.

'Rex II' 0/0 from Denmark belongs between the ten most resistant of these barleys (Tab. 4). It was selected as especially nematode-resistant in the nematode-resistant 'Rex Abed' by Professor

Table 4. Percentage of attack of barley varieties and lines other than those of the interrelated northwest European series highly resistant to leaf stripe.

Varieties, lines and places of origin	Inoculation in the field	Inoculation with the sandwich method
Black Russian (CI 2202) (USSR)	0	0
Modia (CI 2483) (Uruguay) two samples	0	0
Näckte v. Nepal (Nepal)	0	0
Rex II (Denmark)	0	0
Ricardo (CI 6306) (Uruguay)	0	0
Trofimovskaja (Armenia)	0	0
Valkie (CI 5748) (USSR)	0	0
White Gatami (CI 920) (China)	0	0
CI 3694 (Egypt)	1.0	0
CI 4623 (Japan) three samples	0.7	0
Average	0.2	0

Table 5. Percentage of attack of barley varieties and lines other than those of the interrelated northwest European series highly resistant to leaf stripe but different from zero attack.

Varieties and places of origin	Inoculation in the field	Inoculation with the sandwich method
Cheyney (-)	2.0	0
Chirley (Iraq)	4.0	2.5
Cornutum (CI 2215) (U.S.D.A.) two samples	0.5	1.0
Frankenthaler Pfälzer (Germany)	0	3.6
Ishtar (CI 1615) (China)	0	3.4
Murasaki Mochi (CI 5899) (Japan) two samples	1.5	1.7
Nakte Hadaka (Japan)	0	3.7
Osiris (Sweden) primitive 6r.	1.0	1.4
Scottishe Annot (-)	3.0	0.7
Tystofte Kors (CI 918) (CI 6503) (Denmark) two samples	0	4.5
Weisse Erfurter (Germany)	2.0	4.9
Average	1.3	2.5

C.A. Jørgensen. 'Rex Abed' had a 10% attack after field inoculation.

Eleven other barleys that tend to be a little less resistant to leaf stripe (Tab. 5) than the above-mentioned ten originate from almost as many different localities. 'Osiris' is a primitive, nematode-resistant barley collected by Videgård in Sweden. It has neither anything to do with the French nor the German 'Osiris' (cf. ARIAS et al., 1983). 'Nakte Hadaka' originates from a cross between 'Tokuskima Mochi', selected in 'Shiro Chinko', and 'Aka Shinriki' selected in 'Shinriki' (BAUM et al., 1985).

Based on our study of the literature, the old Danish variety 'Tystofte Kors' (Tab. 5) appears to be the first variety in the world that is described as resistant to leaf stripe. It is still

resistant in our experiments as well as those in USA and Canada (cf. the discussion). According to MORTENSEN (1909) and IVERSEN (1915) 'Tystofte Kors' should be the same as or selected in 'Tystofte 25' (Tab. 7). They are both highly resistant after inoculation in the field, but the former had 4.5 and the latter 18.2% attack in the sandwich test (Tabs 5 and 7).

In 1958 J. Sandfaer (Risø, Denmark) got seeds of ten pairs of 6-rowed vs. 2-rowed isogenic barley lines from G.A Wiebe (Beltsville, USA). The lines designated 9-v-19, 13-v-19, and 23-v-18 were highly resistant to leaf stripe both as 6- and as 2-rowed. Four of them were tested by both methods (Tab. 6). These isogenic lines were made either in 'Manchuria' or in 'Betzes' (J.G. Moseman, Beltsville, USA, pers. comm.). The former were not tested in our experiments whereas 'Betzes' (CI 6398) was highly resistant in the sandwich test (Fig. 12:8CD). These isogenic lines might presumably have got their resistance from 'Betzes'.

The Latin names of the botanical barleys were given by the collector who in most cases deposited them in the Botanical Garden of Copenhagen in 1939 (Tabs 6 and 7).

Tab. 7 contains 21 barleys which were highly resistant to the inoculation in the field. A few of them were moderately resistant after inoculation with the monospore culture using the sandwich method, whereas the others were susceptible or highly susceptible.

Two samples of the old Norwegian land variety 'Trysil' were included in our screening. No. 83, which have been in the KVL collection for many years, and no. 979, which was recently provided by Professor Erling Strand. No. 83 had 1% and 11% attack after inoculation in the field and with the sandwich method (Tab. 7), respectively, whereas no. 979 had 84% attack after inoculation in the field. Therefore, it may wonder if these two samples belong to the same variety. The highly resistant varieties 'Piikkio', 'Bethge II', 'Franks Hohenloher', and 'Betzes' are delt with in the following section.

Table 6. Percentage of attack of botanical barley and experimental lines (cf. Material and Methods) highly resistant to leaf stripe.

Names or designation and places of origin	Inoculation in the field	Inoculation with the sandwich method
Black smooth-awned (-)	2.0	4.1
H. distichum L. (Dijon)	2.0	2.1
H. distichum L. (Nantes)	3.0	4.7
H. distichum L. (Pavia)	0	0.8
H. distichum L. var. *nigrum* x *trifurcatum* (Tabor)	1.0	1.4
H. distichum L. spp. *zeocrithum* L. (Vogt)	2.0	2.9
H. tetrastichum var. *agricrithum* (Delft)	0	0.7
H. tetrastichum var. *nigrum* Cev. (Lisbon)	0	2.8
H. vulgare L. (Metz)	1.0	2.2
H. vulgare L., naked (Manchester)	0	0.7
H. vulgare L. var. *nigrum* (Cluj)	0	4.3
Mærkesort 710 x 11 (Denmark)	5.0	0
Mærkesort e, v (USA)	0	0
Mærkesort Tr., V (J. 1293) (Wales)	0	0
Naked barley (Sophiehøj, Denmark)	3.0	3.5
9-v-19 (Wiebe, USA) 6r	0	0.7
9-V-19 (Wiebe, USA) 2r	4.0	1.5
13-v-19 (Wiebe, USA) 6r	0	0
23-v-18 (Wiebe, USA) 6r	4.0	0
Average	1.4	1.7

4.1.2.2. Moderately resistant barleys

A relatively large number of barleys were moderately resistant or moderately susceptible in all the series (Figs 6-12). It is possible to trace the heredity of the resistance or susceptibility from ancestors to offspring with a reasonable degree of significance for many of these barleys.

Table 7. Percentage of attack of barleys resistant to leaf stripe after inoculation in the field but susceptible when inoculated with a monospore culture by the sandwich method.

Names or designation and places of origin	Inoculation in the field	Inoculation with the sandwich method
Dorsett (CI 4821) (selected from Chinese Ottawa 60)	0	6
H. vulgare L. (Vilno) violet, naked	0	8
M. Iowa (USA)	0	9
H. vulgare L. (Oslo) violet, naked	0	11
Trysil (Norway)	1.0	11
H. vulgare L. var. *hexastichum* (Leipzig)	0	14
Harnbarley (Gatersleben)	1.0	15
Peragis (Saxonia)	0	17
H. vulgare L. var. *macrolepis* (Vogt)	1.0	17
Tystofte 25 (Denmark)	1.0	18
H. hexastichum L. (Rouen)	1.0	23
Zeiners Immune (Gatersleben)	0	26
H. distichum L. var. *deficiens* (-)	0	28
H. vulgare L. (Lisbon)	0	36
H. vulgare L. var. *macrolepis* (Kaunas)	0	37
Vogels mildew resistant 62/125 (Germany)	0	38
Koosdorfer fr. Zaya (Gatersleben)	1.0	38
Finne (Norway) two samples	0	40
Gatami (CI 1413) (Australia)	0	41
H. vulgare L. var. *polystichum* (Marbourg)	0	52
H. vulgare L. (Lyon)	0	80
Average	0.3	26.9

A. Series of old barleys. The breeding of barley was initiated in the second half of the 19th century by selection in the heterogenous local barleys or land varieties. On picking up healthy-looking plants from fields with a certain level of disease-

-pressure may now and then have resulted in selecting of resistant plants. This may be the reason why several old varieties have a reasonable level of resistance. The 'Archer', 'Chevallier', and 'Bethge' series presented in Fig. 12, and the 'Hanna' series included in Fig. 10 may constitute such examples.

Selections in 'Archer' (Fig. 12:1-4AF) have apparently resulted in different levels of resistance or partial resistance, and selections after the crosses with either 'Spratt' or 'Plumage' did not make any change in this apart from 'Earl' 30/- which is susceptible (Fig. 12:3C).

The level of attack in 'Carlsberg' 10/- ('Prentice Abed' x 'Maja') may be traced back to 'Archer' via 'Prentice Abed' 8/- and 'Prentice' as the other parent 'Maja' 3/26 had a lower level of infection after inoculation in the field.

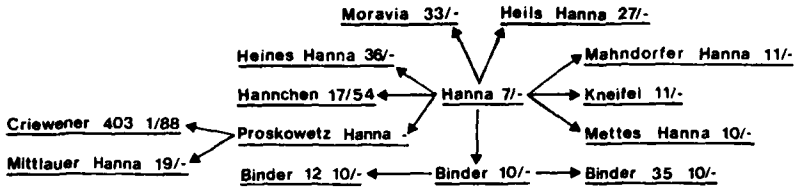
Selections in 'Chevallier' 7/- (Fig. 12:5-6AE) resulted obviously in different levels of resistance comparable to those of 'Archer'.

'Chevallier' 7/- x 'Imperial' 16/- (cf. the 'Chevallier' and 'Bethge' series in Fig. 12) gave 'Bestehorn Diamant' (Fig. 10: 1DE) from which were selected both 'Svanhals' 21/- (Fig. 10:1DE) and 'Primus' 38/- (Fig. 10:3-4A). The different levels of attack on these two varieties are hardly surprising when seen on the background of the apparent heterogenicity of 'Chevallier'.

'Bethge II' 1/3 (Fig. 12:7-8BC), which is selected from a Bohemian land variety, is highly resistant and its cross with the susceptible 'Imperial' 16/- resulted in the highly resistant 'Franks Hohenloher' 0/3. 'Betzes' (CI 6398) -/1.4 suggests to have its resistance from 'Bethge II' 1/3, too. A second sample of 'Betzes' proved susceptible. The two samples have the same isoenzyme pattern.

The 'Hanna' or 'Old Hanna' land variety, whatever it may be (Fig. 10:5-8A), must have been heterogenous in several characters. It constitutes the starting point for a large part of the

barley breeding in Europe through the mass selection in it, cf. PLARRE and HOFFMANN (1963) and the detailed outline presented below of the varieties included in our screening:



Of these selections, 'Moravia' has been mentioned as synonymous with 'Hanna' (cf. BAUM et al., 1985; and Fig. 10:1D). At least three levels of resistance were found among these selections after inoculation in the field. About half had 10% diseased plants including 'Hanna' itself as it appears today and 'Binder Abed' with 'Binder Abed 12' and 'Binder Abed 35'. Only 'Hannchen' and 'Criewener 403' were tested with the monospore culture. In this connection compare 'Criewener N.Z.' and 'Criewener 96' (Tab. 10).

The highly resistant 'Piikkio' 3/3 (Fig. 10:7A), which is synonymous with 'Pukkionohra' according to BAUM et al. (1985), is selected in a Finnish land variety (Note a typing fault: u vs. ii; -ohra = barley).

'Piikkio' 3/3 x 'Binder Abed' 10/- gave 'Helmi' 14/- with the level of attack equal to that of the latter parent. 'Puke' 17/- x 'Binder Abed' 10/- gave 'Puke II' 11/- (Fig. 10:1-3A) which had also the level of attack of 'Binder Abed' 10/-.

'Primus' 38/- x 'Gull' 14/52 (Fig. 10:2-4A) gave 'Primus II' 19/- which may have its level of susceptibility from either the heterogenous background of 'Primus' or from 'Gull'.

B. The 'Vada' series. 'Anna Abed' 11/- (Fig. 6:4E) has probably its moderate resistance from 'Carlsberg' 10/- via 'Abed 0625' as it has less likely come from 'Midas' 21/-. 'Claudia' 7/- (Fig. 6:7B) seems to be intermediary between the resistance of the parents. 'Varunda' 2/51 (Fig. 6:8B) is almost as resistant as

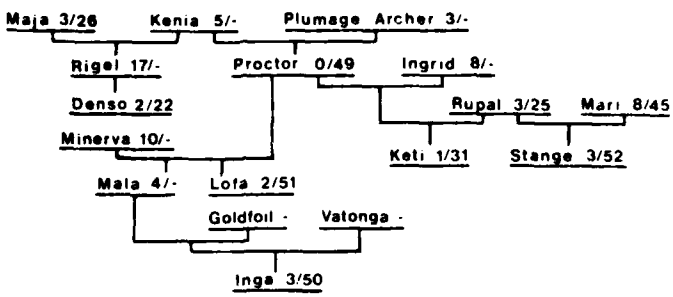
the 'Vada' parent after inoculation in the field but opposite to this it is very susceptible after inoculation with the monospore culture using the sandwich method. 'Salka' 10/- (Fig. 6:8B) seems to have been heterogenous as mentioned above. 'Salka' must have got this condition from 'Elbo' 4/-. 'Elbo' 4/- and 'Pendo' 7/- are sister varieties after 'Drost' 19/- x 'Rika' 9/78 (Fig. 10:7CD) both of which may carry the heterogenicity from 'Kenia' 5/- as discussed below. The background for the level of resistance in 'Roland' 12/- (Fig. 6:5AB), 'Claret' 14/- (Fig. 6:6EF), and 'Magnum' 6/- (Fig. 6:6B) cannot be traced on the basis of the present results.

C. The 'Minerva' series. The levels of resistance in the sister varieties 'Mirjam' 12/-, 'Simba' 3/19, and 'Nery' 8/- from Sejet (Fig. 7:2AC) may be traced back to the ancestors. 'Mirjam' and 'Nery' are equal to either 'Minerva' 10/-, 'Herta' 7/- or 'Ingrid' 8/- whereas 'Simba' 3/19 is equal to 'Byg 191' 5/22. 'Sune Sejet' 6/- (Fig. 7:3DE) may have its resistance from the same source or from 'Kristina' 10/- (Fig. 7:2DE), but it is different from the brother variety 'Uffe Sejet' 0/0 as mentioned above.

'Proctor' 0/49 (Figs 6:5DE and 9:6B) may be conditioned with resistance genes from either 'Kenia' 5/- or 'Plumage Archer' 3/-, and may be regarded as a transgressive product of these genes. If this is the case, 'Ark Royal' 7/- (Fig. 9:7AB) and 'Sammy' 7/- (Fig. 9:7BC) may have their resistance gene from 'Proctor' 0/49 though they could have got their resistance from the untested 'HP 5466' (cf. Tab. 3) and 'Volla' x '*H. spontaneum*, m.r.', respectively.

The high susceptibility in 'Proctor' 0/49 after inoculation with the monospore culture was obviously transferred to 'Lofa' 2/51 and to 'Inga' 3/50 (Fig. 7:4CD) via 'Mala' 4/- (Fig. 7:3BC). Further, the 'Proctor' genes may have been transferred to 'Keti' 1/31 (Fig. 7:5C) via 'All. 297' ('Proctor' x 'Ingrid') though it may have instead the conditions from 'Rupal' 3/25 (Fig. 7:5CD). In this connection, it should be mentioned that two samples of 'Rupal' were included in the screening, 'Rupal' 17/- (No. 817) and 'Rupal' 3/25 (No. 1040) of which the latter are provided

from the breeder. They differ, however, in isoenzyme pattern. Confer the following outline:



The larger attack in 'Mandolin' 7/- (Fig. 7:4AB) than in 'Lofa' 2/51 may have come from the unknown '65-44'. 'Odin' 10 - (Fig. 7:3DE) may have its resistance from 'Ingrid' 8/- but it might have come from 'Sv. 66433' ('Nem.' x 'Sv. 60210' x 'Long Glumes' 17/- x 'Pallas' 4/-) as well. Compare 'Visir' (Fig. 7:5EF).

The crossing between 'Maja' 3/26 and the probably resistant product of 'Seger Sv.' 5/- x 'Opal' 7 - gave 'Bonus' 18 - and 'Ymer' 22/- (Figs 7:4AC and 10:4BD) which may be explained from a probable heterogeneity in 'Maja' as discussed below. 'Mari' 8/45 and 'Pallas' 4/- are X-ray mutants in 'Bonus' (Fig. 7:4-6BC). Besides this, 'Bonus' seems to have a one-gene conditioned susceptibility where it is involved in the Asplund-Maskin series (Tab. 9), and finally, 'Bonus' 18/- x 'Minerva' 10 - gave the highly resistant variety 'Bomi' 3/0 which is referred under the Vada-resistance (Fig. 7:5B).

Four samples of 'Mari' were included in our screening, 'Mari' 22/- (No. 758, KVI), 'Mari' 43/- (No. 853, Risø), 'Mari' 27/- (No. 920, Iceland), and 'Mari' 8/45 (No. 1032, Svalöf) of which at least the Svalöf sample from the breeder is different from the others. The sister varieties 'Sold' 7/- and 'Stange' 3/52 from 'Ingrid' 8/- x 'Mari' 8/45 are equal to the parents (Fig. 7:6AB), and 'Mona' 10/- from 'Monte Cristo' 20/- backcrossed two times to 'Mari' 8/45 most likely have its resistance from the latter variety (Fig. 7:6DE), but a KVI sample of 'Mona' with 17%

diseased plants make it questionable and points back to the susceptible KVI sample of 'Mari'.

The origin of 'Gunilla' 17/- is rather complex (Fig. 8:7CD); it is so closely connected with 'Birgitta' 19/27, however, that it is likely that its susceptibility comes from this variety. 'Pernilla' 10/- (Fig. 7:6E) from the crossing of 'Gunilla' 17/- x ('Birgitta' 19/27 x 'Mari' 8/45) may likewise have the resistance of 'Mari' 8/45.

The other barleys with 'Mari' as one of the ancestors are mentioned under the series to which they belong.

Two samples of 'Pallas' were included in our screening, 'Pallas' 24/- (No. 742, KVI) and 'Pallas' 4/- (No. 1031, Svalöf), with a difference like that found in 'Mari'. 'Pallas' 4/- crossed with 'Herta' 7/- gave 'Hellas' 15/- (Fig. 9:5BC) which may hardly be explained unless 'Pallas' have been heterogenous as could be suggested on that mentioned above. 'Taarn' 7/- (Fig. 7:5E) from a product of 'Pallas' 4/- x 'Hellas' 15/- and 'Kristina' 10/- may equally well have its level of attack from 'Pallas' and 'Kristina'. 'Jenny' 0/18 has the same parents but its level of attack can hardly be explained on this basis when one takes note of the sample received from the breeder. Another sample (cf. Tab. 3) of 'Jenny' tested was on a level with 'Taarn'. The closely related 'Senat' 8/- (Fig. 9:5CD) constitutes a comparable case.

'Visir' 5/- may have its level of resistance from 'Pallas' 4/- (Fig. 7:5EF), whereas the background for the resistance in 'Togo' 7/- (Fig. 7:5C) cannot be traced as this variety is the result of 'J5' back-crossed three times into 'Pallas' 4/- and this again five times into 'Inis' (('Nora' x 'WMR II') x ('Voldagsen' x 'Ingrid')) which is not included in our screening.

'Balder' 12/- (Fig. 7:7B) has its level of resistance from either 'Gull' 14/52 or a Scandinavian land variety, and 'Bine'

13/- (Fig. 7:7B) may have its resistance from either 'Balder' 12/- or 'Minerva' 10/-.

The background for the level of resistance in 'Bente' 7/- (Fig. 7:8C) cannot be traced in the present screening. 'Balder' 12/- x 'Maja' 3/26 gave 'Stallar W' 17/- (Fig. 10:2D) with a resistance equal to the sum of those of the parents, but it might come from the more susceptible component in 'Maja', cf. the following section.

D. The 'Opal', 'Kenia', and 'Maja' series. 'Binder' 10/- x 'Gull' 14/52 gave 'Opal' 7/-, 'Kenia' 5/-, and 'Maja' 3/26 (Figs 8:1-8A, 9:1-8A and 10:1-7A) that are transgressively lower than the parents. They appear uniform but at least 'Maja' and 'Kenia' may originally have been heterogenous as a selection in 'Maja' gave 'Drot' 17/- (Fig. 10:6AB), and selections in 'Kenia' gave 'Alfa' 2/1 and 'Fero' 5/- (Fig. 9:1AB) of which the former may be regarded significantly lower than 'Kenia'. To this we note that 'Maja' 3/26 x 'Kenia' 5/- gave 'Lenta' 9/-, 'Drost' 19/- and 'Rigel' 17/- (Figs 10:2AB, 10:6-7BC) of which the last two are significantly more susceptible than the parents. Further, selection in them gave 'Drost A' 4/- and 'Denso Abed' 2/22 (Fig. 10: 6BC), respectively, and these varieties are in close accordance with the resistance of the parents of those in which they are selected. As 'Drost' and 'Rigel' are on the same level as 'Drot', it is likely that they have come from the more susceptible component in 'Maja', and that 'Drost A' and 'Denso Abed' have come from the other or are real crossings between 'Maja' and 'Kenia'. The conclusion is that, as they appear today, 'Opal', 'Kenia', and 'Maja' have most likely the same gene for resistance to leaf stripe after inoculation with a population of the pathogen in the field. At the time, however, when the tested offspring was bred, 'Maja' have had two components conditioning about 5% and 15-20% diseased plants, respectively.

'Beorna' 8/- (Fig. 9:1BC) obviously has the resistance from 'Kenia' 5/-.

'Opal B' 7/- (Fig. 8:2E), which is selected in 'Opal' 7/-, does not raise questions.

These results lead to the suggestion that 'Deba Abed' 12/- ('Denso Abed' 2/22 x 'Weihenstephan Mildew Resistant II' (WMR II) 6/-) and 'Claudia' 7/- ('Deba Abed' 12/- x 'Vada' 0/0) might have their resistance from WMR II (Figs 6:7B, and 10:7B).

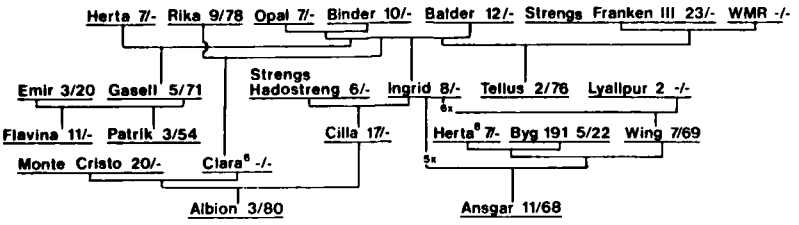
'Gull' 14/52 x 'Hannchen' 17/54 gave the resistant 'Seger' 5/- (Fig. 8:1AB) though they appear equally susceptible, which suggests that these old varieties have been heterogenous. 'Seger' 5/- x 'Opal' 7/- gave 'Freja Sv.' 2/1 (Fig. 8:1AB) (provided from three sources) which suggests the possibility that resistances may act transgressively. On the other hand, the crossing between 'Freja Sv.' 2/1 and 'Lenta' 9/- (from 'Maja' x 'Kenia') gave 'Hafnia' 76/- (Fig. 10:4AC) from which we conclude that the genes in 'Freja Sv.' and 'Lenta' are different, inherited independently, and not given to the offspring in the present case; thus, it is highly susceptible. Further, this means that the resistance gene in 'Seger Sv.' is different from that in 'Opal', 'Kenia' and 'Maja'. This statement is problematic, however, as 'Freja Sv.' has the Opal and the Seger genes, and 'Lenta' the Opal gene from both parents. For this reason the offspring should have the Opal gene. Therefore, the explanation may be that 'Lenta' has at least one gene from the more susceptible component of either 'Kenia' or 'Maja' mentioned above.

'Maris Baldric' 16/- (Fig. 8:1AB) must have its susceptibility from 'Spratt Archer' but if the level of susceptibility in 'Earl' 30/- (Fig. 8:1C) is characteristic for that old variety, 'Maris Baldric' may be regarded as intermediary between the parents.

'Balder' 12/- x (Opal' 7/- x 'Binder' 10/-) gave 'Ingrid' 8/- which is equal to any of the ancestors (Fig. 8:2BC). The reason for this equality may be that they have the same gene.

'Wing' 7/69 (Fig. 8:2AB) from 'Lyallpur 2' back-crossed six times into 'Ingrid' 8/- have most likely its resistance to leaf stripe after inoculation in the field from 'Ingrid' and probably also, on the basis of the relations outiined below, the suscept-

ibility after inoculation with the monospore culture using the sandwich method:



'Torkel' 7/33 and 'Welam' 7/32 (Fig. 9:8DF) are so closely related with 'Clara', 'Ingrid' 8/- and 'Gasell' 5/71 that they very likely belong to the outlined resistance group. Further, 'Prisca' 3/5 (Fig. 9:8CD) is on about the same level of resistance after inoculation in the field, whereas it must have got its resistance to the monospore culture from elsewhere. Also 'Elbo 4/-, 'Pendo' 7/-, and 'Tyra' 10/- (Fig. 10:7CD) may belong to this group.

This pattern of resistance and susceptibility may be traced back to old barleys. 'Herta' 7/- and 'Rika' 9/78 are sister varieties after 'Kenia' 5/- x 'Isaria' 27/- (Fig. 10:7D) and 'Kenia' 5/- is sister variety to 'Opal' 7/- and 'Maja' 3/26 (Figs 8-10) after 'Binder' 10/- x 'Gull' 14/52. Further, 'Gull' 14/52 and 'Maja' 3/26 are ancestors of 'Balder' 12/- (Fig. 7:7B). 'Emir' 3/20 (Fig. 9:3C) is closely related with this group of barleys. Crossed with 'Gasell' 5/71 it gave 'Patrik' 3/54 (Fig. 9:4D) which appear intermediary between the parents. The field resistance against powdery mildew in 'Strengs Franken III' 23/- (EWERTSON, 1974), the specific powdery mildew resistance Weihenstephan, Lyallpur and Monte Cristo, and the nematod resistance from 'Byg 191' were bred into this group of barleys but the several backcrosses made have obviously eliminated their reaction to leaf stripe from the resulting varieties. Only the susceptibility of 'Cilla' 17/- (Fig. 8:3C) appears high compared with the other varieties of the group.

An analogous and related group of varieties around 'Proctor' 0/49 and 'Ingrid' 8/- is outlined in a previous section (C. The 'Minerva' series).

'Pamina' 8/- and 'Albert' 14/-, 'Ida' 16/- and 'Harry' 33/-, and 'Harda' 10/- and 'Birka' 42/- descend from 'Tellus' 2/76 (Fig. 8:5-6AF) into which were bred Monte Cristo resistance to powdery mildew and/or to dwarf rust from 'Baladi 16'. Susceptibility from them or from 'Arla' 20/- (Fig. 8:8CD) to leaf stripe are apparently carried along to at least 'Harry' 33/- and 'Birka' 42/-.

'Maja' 3/26 x ('Opal' 7/- x 'Vega Sv.' 37/-) gave 'Birgitta' 19/27 (Fig. 8:6DE) which may be regarded as intermediary. However, as 'Vega Sv.' probably has a one-gene conditioned susceptibility (cf. Tab. 9), we must take into account that the susceptibility of 'Birgitta' is connected with the heterogenicity of 'Maja'. 'Birgitta' 19/27 x 'Mari' 8/45 gave 'Salve' 15/- and 'Eva' 7/- (Fig. 8:7EF) that is equal to each of the parents concerning the attack after inoculation in the field.

'Sultan' 7/- and 'Susan' 7/- (Fig. 9:2CD) are so closely related with 'Kenia' 5/- via 'Agio' 9/- ('Kenia' 5/- x 'Georgine' 21/-) (Fig. 9:3BC) that they are most likely conditioned by the resistance gene from this variety.

'Maja' 3/26 x 'Prentice Abed' 8/- gave 'Carlsberg' 10/- (Fig. 10:5B) which is equal to the latter parent, but it must have been somewhat heterogenous as a selection in it gave 'Carlsberg II' 17/- which has the same susceptibility as the more susceptible component in 'Maja' mentioned above. 'Carlsberg' 10/- x 'Rigel' 17/- gave 'Karri' 19/- (Fig. 9:2B), but even though its susceptibility is the same as that of 'Rigel' it may come from that part of 'Carlsberg' which led to 'Carlsberg II' (Fig. 10:5B). 'Carlsberg' was withdrawn from the market between 1952 and 1954 due to heterogenicity and replaced with the selected 'Carlsberg II' 17/-. 'Lone' 19/- (Fig. 9:7DE) is the product of 'Herta'[5] 7/- x 'Byg 191' 5/22 backcrossed five times into 'Carlsberg' 10/- why it should most likely carry the level of attack in this variety but in fact it carries that of 'Carlsberg II' 17/-. The product of 'Carlsberg' 10/- x 'Ymer' 22/- was 'Dana' 7/- (Fig. 10:6D) with 'Carlsberg''s resistance, and 'Dana' 7/- x 'Heine 4808' gave 'Nordal' 17/- (Fig. 10:6CD)

which obviously has its susceptibility from 'Heines Haha' 14/-. This variety must contain resistance genes from either 'Isaria' 27/- (of 'Danubia' 14/- x 'Bavaria' 17/- 'Fig. 9:5B)) or 'Heines Hanna' 36/- via 'Heines Haisa I' (Fig. 10:6B and 10:5EF). 'Dana' 7/- x 'Proctor' 0/49 gave 'Danpro' 12/- which is hardly different from 'Dana' (Fig. 10:6D).

'Europa' 10/- (Fig. 9:3D) may equally well carry the resistance from either 'Kenia' 5/- or 'Balder' 12/- via 'Hassan' and 'Cambrinus', respectively.

'Gunhild' 11/- is probably conditioned by a resistance gene from line 'M 63199' as it may hardly have it from the product of 'Lone' 19/- x 'Algerian' 19/- (Fig. 9:7EF). 'Europa' 10/- (Fig. 9:3D) may equally well carry the resistance from either 'Kenia' 5/- or 'Balder' 12/- via 'Hassan' and 'Cambrinus', respectively.

'Swallow' 7/- (Fig. 9:4B) must be conditioned by a resistance gene from 'Donaria' 3/12 - with a level of attack which is significantly lower than that of each of its parents ('Kneifel' 11/- x 'Isaria' 27/-) - as it is unlikely that the resistance has come from the highly susceptible 'Piroline' 68/- (Fig. 9:3BC). ('Swallow' 7/- x 'Emir' 3/20) x 'Deba Abed' 12/- gave 'Maris Yak' 4/- and 'Maris Mink' 13/- that may be conditioned by a gene from one of the parents each. On the other hand, 'Yriba' 17/- from 'Maris Yak' 4/- x ('Rika' 9/78 x 'Baladi 16' may be explained only as being conditioned by two genes unless it has a gene from the untested 'Baladi 16'.

'Isaria' 27/- ('Ackermanns Isaria') is one of the most widely used varieties in the barley breeding. Concerning the reaction to leaf stripe it is obviously conditioned by genes from each of its parents 'Danubia' 14/- and 'Bavaria' 17/- (Figs 9:5B and 10:6-7DE). The resistance level of the latter is brought along to 'Dornburger Eva' 18/- ('Bavaria' 14/- x 'Heils Franken' 7/-) (Fig. 10:7D). 'Strengs Franken II' 23/- ('Isaria' 27/- x 'Strengs Hadostreng' 6/-) may have the genes from 'Isaria' (Fig. 8:2DE). The same may be the case with 'Georgine' 21/- ('Isaria' 27/- x 'Moosburger Rhätia') (Fig. 9:2-3BC), whereas the resist-

ance of 'Donaria' 3/12 ('Ackermanns Donaria') cannot be explained from the parents ('Kneifel' 11/- x 'Isaria' 27/-) reaction to leaf stripe (Fig. 9:3AB). 'Isaria' 27/- x 'Kenia' 5/- gave 'Herta' 7/-, 'Rika' 9/78, and 'Stallar II' 31/- (Fig. 9:4B and 9: 6AB). The first two have the resistance of 'Kenia' 5/- while 'Stallar II' 31/- have that of 'Isaria' 27/- or the sum of both the parents reaction to leaf stripe.

'Isaria' 27/- x 'WMR I CP' (from 'Criewener 403' 1/88 x 'Pflugs Intensiv') gave 'WMR II' 6/-, 'Firlbecks III' 10/-, and 'Breuns Wisa' 20/- (Fig. 10:2DF). The former two may have their resistance from 'WMR I CP' but 'Firlbecks III' 10/- equally well have one of 'Isaria''s resistance genes. 'Breuns Wisa''s 27/- reaction to leaf stripe is hardly different from that of 'Isaria' 27/- but it cannot be left out of account that it has a resistance gene from each of the parents. 'Erika' 16/- ('Schweigers Erika') may have one of 'Isaria''s 27/- resistance genes or it may be intermediary between the parents 'Isaria' 27/- and 'WMR II' 6/- (Fig. 10:6AB). 'Mentor' has its level of resistance from either 'Balder' 12/- or 'WMR II' 6/- (Fig. 10:2DE). Both 'WMR I CP' and 'Isaria' 27/- may be involved in the resistance of 'Heimes Haisa II' 10/- (Fig. 10:4EF). 'Morgenrot' 11/- and 'Zeiners Frankenperle' 11/- are sister varieties. Their reaction to leaf stripe might have come from 'Heils Franken' 7/- but we cannot know if it might equally well have come from the untested 'Prior' (syns 'Australian Early', 'Australische Frühe', and 'Australian Chevallier'). 'Foma' 14/- and 'Refoma' 10/- are equal to 'Morgenrot' 11/- and not to the other parent, 'Ymer' 22/- (Fig. 10:4-5CE).

E. The 'Asplund'/'Maskin' series. 'Olli' 4/- (Fig. 11a:1A), 'Dore' 4/73 (Fig. 11a:1F), 'Jadar' 7/33 (Fig. 11a:3E), and 'Juli' 0/13 (Fig. 11a:4E) have their resistance from old landvarieties. 'Asplund' 37/- (Asp 37) x 'Olli' 4/- gave 'Tammi' 29/- with a level of attack between the parents (Fig. 11a:1AB). If this is correct, the genes from both 'Asplund' and 'Olli' should be present in 'Tammi', and nothing in the use of 'Tammi' as crossing partner conflicts with that. 'Hja. Aapo' 7/-, that is an X-ray mutant (Ta b7990 (XR)) in a selection (Ta b7990) in

'Tammi' 29/-, may then have its resistance gene from 'Olli' 4/- (Oll 4) (Fig. 11a:2AB). 'Otra' 23/- from 'Tammi' 29/- x 'Edda' 34/- is hardly different from 'Tammi' and may then have the genes Asp 37 and Oll 4. 'Hja. Eero' 13/- suggests to have the resistance from 'Mari' 8/45 as the genes conditioning the reaction to leaf stripe in 'Otra' 23/- ('Olli' 4/- and 'Asplund' 37/-) are too different from that of 'Hja. Eero' 13/- (Fig. 11a: 2-3AB). 'Hja. 77061' 9/- (Fig. 11a:6A) is intermediary between the parents 'Hja. Eero' 13/- and 'Olli' 4/-. 'Ringve' 19/- (Fig. 11a:4D) may have its level of attack from either 'Rigel' 17/- or 'Jarle' 14/-. From the former it may be traced back to the more susceptible component in 'Maja' 3/26, and from the latter back to Mas 18 - one of the suggested genes in 'Maskin' 47/- (cf. the analysis of the susceptible barleys of the 'Asplund'/'Maskin' series below). 'Gunnar' 9/- may almost equally well have its resistance - after inoculation in the field - from either 'Mari' 8/45 or 'Kristina' 10/- (Fig. 11a:7AD). 'Särla' 7/- may have the resistance from 'Opal B' 7/- via 'Domen' 29/- but the series of ancestors listed (Fig. 11a: 7-8AD) leave also other possibilities.

'Herta' 7/- x 'Jo 0490' ('Seger' 5/- x 'Vega Sv.' 37/-) gave three untested lines ('Jo 0888', 'Jo 0893', and 'Jo 0877'). 'Jo 0888' x 'Ingrid' 8/- gave the highly resistant 'Jo 1345' 0/-. The basis for this may be explained as follows: 'Herta' 7/- and 'Ingrid' 8/ have after all probability the 'Opal' resistance gene which is different from that of 'Seger'. Further, it is very likely that 'Jo 0888' is constituted with both the 'Opal' and the 'Seger' gene. This leads to the same gene-combination as that which gave the highly resistant 'Freja Sv.' 2/1 (cf. Fig. 8:1AB and 11b:2-3BE). Further, we have here the opposite case of that with 'Hafnia' 76/- where we suppose that 'Seger' 5/- x 'Lenta' 9/- gave a variety without any of the resistance genes and therefore is susceptible. A selection in 'Jo 0893' gave 'Jo 1220' 9/- and the cross 'Jo 0877' x 'Ingrid' 8/- gave 'Jo 1309' 7/-. Both these lines are in agreement with that just stated and indicate why they support the explanations given (Fig. 11b:2-3DE).

'Jo 1119' 12/- (Fig. 11b:4EF) might have the same gene combination for reaction to leaf stripe as 'Jo 1345'. The 'Opal' gene from 'Opal' 7 x 'Perttu' (Fig. 11b:3DE) and the 'Seger' gene from 'Jo 0490' x 'Balder' 12/- (Fig. 11b:3EF) but it has more likely the resistance from 'Balder' 12/-.

'Jo 1381' 4/- from 'Etu' 77/- after three backcrosses into 'Am.'. Thus, it must have its resistance from 'Am.' whatever it might be (Fig. 11b:3-4A).

F. Other moderately resistant barleys. A group of varieties from U.S.A. and Canada were bred into Finnish barleys (Fig. 11b:5-6CF). A cluster of these varieties came out of the screening with a low level of attack after inoculation in the field. 'Lion' backcrossed two times to 'OAC 21' 15/- gave 'Galore' 4/- and ('Lion' x 'Coast') x 'Trebi' 10/- gave 'Velvon' 4/-. 'Trebi' 10/- x 'Colsess' ('Coast' x 'Success') gave 'Warrior' 3/15 whereas 'Trebi' 10/- x 'Glabron' 55/- gave 'Titan' 57/- but 'Titan' 57/- x 'Regal' (CI 5030) gave 'Tregal' 14/-. 'Regal 1865' was included in our screening and came out with 15% attack equal to that of 'Tregal' 14/- but we do not know how or if 'Regal' (CI 5030) and 'Regal 1865' were related. 'Trebi 101' 4/- and 'Trebi 628' 3/- may be related to 'Trebi' 10/- (CI 936, cf. Fig. 11b:6E). They came all from Turkey.

Finally, a series of barley - primarily those which could neither be included in the pedigrees (Fig. 6-12) nor were tested by others (Tab. 11) - came out of the screening as resistant or moderately resistant after inoculation with the population of the pathogen in the field (Tab. 8). Most of these barleys are mentioned in the literature (cf. ARIAS et al., 1983 and BAUM et al., 1985). The Norwegian varieties 'Mjøs' 7/66 and 'Sørum' 4/43 belong to this group but they were also tested with the monospore culture to which they proved very susceptible. 'Goldkorn Sv. 178' 4/- is probably related to 'Gull' 14/52 (Fig. 6-10), 'Halikko' 4/- is probably the same as 'Halikongerste' 12/-. The 'Polar' 5/- (No. 1013) included in Tab. 8 is a sample provided by professor E. Strand, Norway. Another sample designated

Table 8. Percentage of attack of barleys resistant or moderately resistant to leaf stripe after inoculation with a population of the pathogen in the field.

Varieties, ancestors and places of origin	Percentage of attack
Adolphe Bungener (Comtesse x B04)	7
Albert Busser (Baronne x Bohemia 2)	3
Anoidium (Argentina)	4
Arlington (Gatersleben)	6
Balkan (Gatersleben)	4
Black Hull-less (Central Asia)	10
Brewers Favorite (America)	10
Carina ((Union x Inis) x Volla)	11
Charlottetown 80 (CI 2732) (Prince Edward Island)	8
Chinese Black (CI 1969) (Canada)	7
Colsess IV (CI 5979)	10
Duplex (Russia)	15
Erbil (Iraq)	11
Erfurter 1395 (Germany)	4
Erie (Goldfoil x Alpha)	8
Fischers Wirchenblatter II (Germany)	5
Fischers Wirchenblatter III (Germany)	9
Flynn (CI 1311) (Club Mariout x Lion)	9
Goldkorn Sv. 178 (Gatersleben)	4
Halikko (= Halikon (?)) (Finland)	4
Hörnings Sommergerste (Poland)	9
Immendorfer Cardus (Zellendorf)	13
Improved Arivat (CI 7534) (USA)	7
Kamet Mugi (CI 2253) (Japan)	5
Krafts Reid (Germany)	4
Kredlers Oberphälzer (Germany)	4
Peragis Neuzucht (Germany)	4
Polar (select. in Ørnesbygg) (Norway)	5
Rabat (Marocco)	14
6-rowed, naked (-)	4
Sort byg (black barley) (-)	4
Stella Sv. (select. in land var.) (Sweden)	11
Stensgård (Denmark)	3
Stewart (CI 6112) (select. in Coast) (USA)	3
Støvring (Denmark)	4
Tibetania (-)	5
Trebi 101 (Turkey)	4
Trebi 628 (Turkey)	3
Vanja (W25-69 x W 4-69)	5
Victoria (= Seger) (Belfast)	5
Zeiners Universal (Germany)	5
Average	6.5

'Polarbyg' (No. 746) from the KVL collection had 43% attack after inoculation in the field.

4.1.2.3. Susceptible and highly susceptible barleys

A. The 'Vada' and 'Minerva' series. The susceptibility of 'Midas' 21/- (Figs 6:2EF and 6:4EF) was neither bred into 'Jupiter' 2/0 nor to 'Anna' 11/-. 'Clermont' 30/- (Fig. 6:6CD) must have its susceptibility from either 'Bordia' or 'Fresia' and it might have been brought to 'Armelle' but not further. 'Sejet 1732' 48/- (Fig. 7:1CD) must have at least the main part of its susceptibility from 'B/78-1'. Crossed with a black-seeded two-rowed barley line it gave 'Anla' 22/- and 'Seta' 68/-. An explanation for this is that the black-seeded barley has contributed with a gene conditioning some susceptibility that acted additively with that of 'Sejet 1732' 48/-. If this is the case, 'Anla' 22/- may have got its level of attack from the black-seeded barley, whereas 'Seta' 68/- has its susceptibility from both parents (Fig. 7:1BC). If this explanation holds, it is strange that 'Anla' 22/- x 'Minerva' 10/- has given 'Lami' 50/-. However, this susceptibility was present in one of the grandparents of 'Lami' ('Sejet 1732' 48/-). This situation reminds one of that mentioned several times in connection with 'Maja'. The susceptibility of the related variety 'Jarl' 30/- may be caused by additively acting genes, *e.g.*, those of 'Minerva' 10/- and 'Anla' 22/- (Fig. 7:1-2AD).

The susceptibility of 'Vega Abed' 22/- (Fig. 7:2E) is dealt with below together with 'Kristina' 10/-.

B. The 'Opal', 'Kenia', and 'Maja' series. The susceptibility of 'Benedicte' 20/- (Fig. 8:4DE) cannot be traced in the ancestors unless it comes from the complex origin of 'Ingrid M_9' (Jørgen Löhde, pers. comm.). 'Strengs Franken III' 23/- must have most or all its susceptibility from 'Krim Barley', as it may hardly have come from 'Heils Franken' 7/- (Fig. 8:4DE).

'Arla' 20/- (Fig. 8:8CD) may have its susceptibility from 'Tammi' 29/- though this variety seems conditioned by two genes but

it may have come from 'Svanhals' 21/- or may be an additive effect of more genes. It is questionable if 'Akka''s 14/- ('Monte Cristo' 20/- x 'Arla'[6] 20/-) (Fig. 9:7B) reaction to leaf stripe is different from that of 'Arla' 20/-.

The high susceptibility of 'Lø 1611' 77/- cannot be traced in the parents (Fig. 8:8B) but it might be a result of loss of two independently acting genes as is suggested to be the case with 'Hafnia' (Fig. 10:4B, cf. above). 'Piroline' 68/- (Fig. 10:5D) may constitute an analogous case which may be supported by the fact that its susceptibility is not brought further to the tested offspring, 'Swallow' 7/- (Fig. 9:4B) and 'Sterling' 15/- (Fig. 10:5D). 'Gitte' 60/-, which most likely should have the resistance genes from 'Herta' 7/- and 'Carlsberg' 10/- (Fig. 9:6-7DF), may constitute one variety more with loss of two independently acting resistance genes.

'Gula Abed' 30/- (Fig. 9:4DE) may have its susceptibility from 'Impala' 31/- which is conditioned by genes from either 'Balder' 12/- plus 'Breuns Wisa' 20/- or 'Heines Haisa I', but it may also have it from 'Imperial' 16/- x '*H. spontaneum* H. 204' (Fig. 10:3DF). So, 'Impala' 31/- probably is conditioned by two genes which makes it reasonable to suggest that 'Tron' 16/- has its gene from this source, but it may logically also have got it from the untested 'Nigrate'.

We suggest, too, that 'Aramir' 21/- (Fig. 9:4BC) has its susceptibility from 'Breuns Wisa' 20/- or 'Heines Haisa I' via 'Volla' ('Breuns Wisa' 20/- x 'Heines Haisa I' (cf. Tab. 3)). The tested ancestors of 'Heines Haisa I' show that it must have a high degree of susceptibility after all.

The susceptibility of 'Siri' 27/- (Fig. 9:5C) must have come from the unknown line '50-88'. 'Isaria' 27/- (Fig. 9:5AB) is most likely conditioned with two genes for reaction to leaf stripe, *i.e.* one for each of 'Bavaria' 17/- and 'Danubia' 14/- (cf. the previous section). 'Breuns Franken III' 16/- (Fig. 9:7BC) might be conditioned with one of these genes.

The probability that 'Amsel Heine' 33/- (Fig. 10:8C) has its susceptibility via 'Heines Haisa 1' is higher than that from 'Lyallpur' 47/- as it can hardly have come from 'Pirol' ('WMR 1 CP' x 'Morgenrot' 11/-) - brother of 'Piroline' 68/- - if the gene conditioning suggested above is the case. 'Dina Abed' 37/- from 'Amsel Heine' 33/- x 'Deba Abed' 12/- may have the same susceptibility as the former parent. The inheritance of 'Amsel Heine' 33/- is, however, questionable as it has the *Laevigatum*--resistance to powdery mildew which does not occur in the ancestors referred in the literature. 'Tern' 21/- (Fig. 10:8C) may also have got its susceptibility via 'Heines Haisa 1' but it may also have got it from 'Kneifel' 11/- and 'Isaria' 27/- via 'Heine 0553' or from the unknown lines occurring between the ancestors. 'Duks' 26/- (Fig. 10:5AB) has probably its susceptibility from 'Lyallpur 2' which is suggested a selection in 'Lyallpur' 47/-.

C. The 'Asplund'/'Maskin' series. It proved possible with the pedigree analyses to explain the most probable gene-combinations for the inheritance of the susceptibility in a rather large number of the varieties and lines in the 'Asplund'/'Maskin' series (Fig. 11a and 11b). The basic genes for the explanations come from the following varieties with the proposed gene symbol set in brackets: 'Asplund' (Asp 37), 'Maskin' (Mas 18 + Mas 29), 'Vega Sv.' (Veg 37), 'Bonus' (Bon 18), and 'Olli' (Oll 4) (cf. the percentage of attack in Tab. 9). These genes appear monogenic inherited except in 'Maskin' which seems conditioned by the two mentioned genes and for example the case of 'Tammi' 29/- and 'Otra' 23/- (Fig. 11a:1AB) of which the former apparently is intermediary between 'Olli' 4/- and 'Asplund' 37/- and the latter either with the same genes or with those of 'Olli' 4/- and 'Edda' 34/-. 'Edda' 34/- must have its susceptibility from either 'Asplund' 37/- or 'Vega Sv.' 37/-. The various combinations of these five or six genes are seen as "additive susceptibility promoting factors" (Tab. 9) giving different levels of partial resistance and not as the abscence of resistance genes.

Used in this way, the level of susceptibility in most, if not all, cases is based on one or up to four genes which may be traced back in the ancestors.

Table 9. Proposed additive susceptibility promoting factors (ASPF) on the susceptible varieties and lines of the Asplund/Maskin series.

Variety or line	Proposed ASPF						Alternative ASPF						Pedigree position
	Asp. 37	Mas. 29	Mas. 18	Veg. 37	Bon. 18	Others	Asp. 37	Mas. 29	Mas. 18	Veg. 37	Bon. 18	Cle. 30	Others	
Asplund 37/-	•													IIa:1DE
Maskin 47/-		•	•											IIa:1C
Vega Sv. 37/-				•										IIa:1BC
Bonus 18/-					•									7:4G
Clermont 30/-												•		IIa:5DE
Agneta 70/-	•			(•)									•	IIa:2D
Arra 56/-	•		•				•	•					Oll 4	IIb:4B
Bode 24/-			•			•								IIb:2A
De 698 43/-		•	•											IIb:4-5A
Domen 29/-		•	(•)			(Opa 7)								IIa:3A
Edda 34/-	•									•				IIa:1B
Edda II 41/-	•									•				IIa:2BC
Etu 77/-	•	•	•				•	•			•			IIa:4B
Forus 21/-			•											IIa:3C
Forus Bonus 18/-			•											IIa:1DE
Frøg 48/-		•	•											IIa:1C
Fg 672 33/-		•												IIa:4BC
Goliat 70/-	•					•								IIa:3E
Herse 60/-	•	•												IIa:1CD
H 1035 50/-	•		•						•			•		IIa:5DE
H 181-49 69/-	•		•			•	•						•	IIa:5EF
Hja. Pokko 75/-	•		•		•								•	IIa:6CD
Hja. 673 21/-	(•)					(Oll 4)							(•)	IIa:3C
Hja. 70185 56/-	•				•								•	IIa:6DE
Hja. 71384 74/-	•					•				•			•	IIa:3CD
Jadar II 31/-	•													IIa:3D
Jarle 14/-			•										•	IIa:4C
Jo 1012 33/-	•					•					•		•	IIb:2D
Jo 1252 50/-	•		•			•		•					(•)	IIb:4C
Jo 1279 37/-	•									•			(•)	IIb:3AB
Jo 1330 76/-	•	•	•											IIb:4CD
Jo 1343 41/-	•								•				(•)	IIb:4DE
Jo 1352 15/-			•								•			IIb:3CD
Kilta 40/-				•										10:3CD
Kristina (10/-, 22/-)		•											Mari	IIa:4A
Lise 57/-	•		•											IIa:5CD
M 268 33/-		•												IIa:4BC
Mø 75-278 39/-		•				•	•							IIb:4-5CD
Nordlys 60/-	•					•								IIb:1F
Otra 23/-	•					(Oll 4)								IIa:2B
Pirkka 65/-		•	•			•							•	IIa:6C
Pk-M 5 14/-			•											IIb:5B
Pk-P 58 19/-			•										•	IIb:4AB
Pomo 54/-				•	•			•	•				(•)	IIa:6E
Presto 30/-	•													IIa:4E
Ringve 19/-			•										•	IIa:4D
Silja 65/-				•		•								IIb:2B
Tammi 29/-	(•)					(Oll 4)								IIa:1AB
Varde 79/-	•	•	•											IIa:1E
Vega Abed 22/-		•												7:2E
Vena 48/-	•					•							(Lyallpur 47/-)	IIa:5B
Vigdis 38/80	•													IIa:5BC
Vå 13002 17/-			•											IIb:1B
Vå 14047 20/-			•											IIb:1BC
Vå 17128 14/-			•											IIb:1CD
Vå 17165 60/-	•	•				•								IIb:1DE
Vå 17236 20/-			•											IIb:1E
Yrjar 63/-	•		•											IIa:4BC
Åsa Sv. (2/69, 32/89)				•									Dore	10:3C

The crosses 'Asplund' 37/- x 'Maskin' 47/- resulted in 'Forus Bonus' 18/-, 'Fræg' 48/-, 'Herse' 60/-, and 'Varde' 79/- that then should be conditioned by the genes as follows: 'Forus Bonus' (Mas 18), 'Fræg' (Mas 18 + Mas 29), 'Herse' (Asp 38 + Mas 29), and 'Varde' (Asp 37 + Mas 18 + Mas 29) (cf. Fig. 11a:1-2CE).

'Domen' 29/- from 'Maskin' 47/- x 'Opal B' 7/- (Fig. 11a:3A) appear intermediary between the parents which means that it has genes from both of them (*i.e.* Mas 18, Mas 29 and Opa 7). If this is correct it is neither surprising that 'PK-P 58' 19/- has the Mas 18 gene nor that 'PK-P 693' 5/- has the Opa 7 gene (Fig. 11b:4AC) but if 'De 69B' 43/- is a mutation *sensu stricto* in 'Domen' 29/- for other characters than the reaction to leaf stripe it should come out with the same level of attack as 'Domen', instead it came out on the same level as the grandparents 'Maskin' 47/-. For the same reason the mutant 'Fg 672' 33/- in 'Fræg' 48/- should be attacked on the same level as 'Fræg' (Fig. 11b:4-5C), but it came out at the level of Mas 29. Still, according to the intermediary appearence of 'Domen' 29/-, it is not surprising that 'M 268' 33/- and 'PK-M5' 14/- after the cross 'De 69B' 43/- x 'Fg 672' 33/- came out with Mas 29 and Mas 18, respectively. 'Tammi' 29/- ('Olli' 4/- x 'Asplund' 37/-) and 'Otra' 23/- ('Tammi' 29/- x 'Edda' 34/-) constitute a comparable case.

'Jo 1310' 46/- is intermediary between the parents, 'Jet' 57/- x 'Ingrid' 8/-, which means that the line is conditioned with both genes like the case with 'Tammi' 29/- from 'Asplund' 37/- x 'Olli' 4/- (Figs 11a:1AB, 11b:2BC).

'Goliat' 70/- from ('Asplund' 37/- x 'Jaerbyg') x 'Bjørne' 8/- must have about "half of its susceptibility" from the last two partners in the crossing, but this requires either that by far the most come from 'Jaerbyg' or that 'Bjørne' has been heterogeneous (Fig. 11a:3CF). 'Jo 1012' 33/- from 'Goliat' 70/- x 'Jo 0490' ('Vega Sv.' 37/- x 'Seger' 5/-) may be conditioned by Asp 37 or Veg 37 (Fig. 11b:2CD).

The supposed occurrence of three genes for the reaction to leaf stripe in 'Domen' 29/- makes it equally possible that 'Møyjar' 10/28 has its resistance from 'Domen' 29/- and 'Herta' 7/- (Fig. 11a:3-4AB), not least because the gene in 'Herta' and 'Opal B' in all probability is the same. ('Vega Sv.' 37/- x 'Seger' 5/-) x 'Bonus' 18/- (Fig. 10:3BD) gave 'Kilta' 40/- which most likely has the Veg 37 gene. 'Vega Sv.' 37/- x 'Dore Sv.' 4/73 gave 'Åsa Sv.' (Fig. 10:3C) of which two sources were tested. They were 'Åsa Sv.' 32/89 and 'Åsa Sv.' 2/69 equal to each of the parents after inoculation with the population of the pathogen in the field, but both very susceptible after inoculation with the monospore culture. This may suggest that 'Åsa Sv.' has two components like 'Maja'. 'Edda' 34/- from 'Vega Sv.' 37/- x 'Asplund' 37/- may have the gene from any of the parents (Fig.11a:1AC) and it might for this reason be the same gene.

'Agneta' 70/- (Fig. 11a:2D) from ('Monte Cristo' 20/- four times backcrossed to 'Edda II' 41/- (a selection in 'Edda') x ('Åsa Sv.' x 'Frisia' (('Granat' x 'Pisthyjarven') x ('Vulcan' x 'Kalkreuter')) might have its susceptibility from 'Edda' 34/- and the susceptible component of 'Åsa Sv.' 32/89 but in that case would Asp 37 and Veg 37 be different genes. The resistance in 'Kajsa' 4/-, sister to 'Agneta', fits surprisingly with the field resistant 'Åsa Sv.' 2/69, but in any event the whole problem may come from the complex origin of 'Frisia'.

'Paavo' 70/- (Fig. 11a:2D) is not included in Tab. 9 because the level of attack cannot be explained from the attacks on the ancestors unless the Oll 4 gene in 'Tammi' 29/- (Oll 4 + Asp 37) is lost in the crossings, and further that it contains a gene from each of the two other varieties,'Gull' 14/52 and 'OAC 21' 15/-. 'Hja. 673' 21/- (Fig. 11a:3C) may be conditioned by one or both of these genes or those of 'Otra' 23/- (Oll 4 + Asp 37). 'Pomo' 54/-, conditioned with the Veg 37 and Bon 18 genes, gave line 'Hja. 71384' 74/- after crossing with 'Hja. 673' 21/- and its level of attack is the sum of those of the parents but this would mean that it has Otr 23 (intermediary of Asp 37 and Oll 4), and for this reason 'Hja. 71384' might equally well be con-

ditioned with Veg 37 from 'Pomo' and Asp 37 without Oll 4 from 'Hja. 673' which is the other parent (Fig. 11a:2-3CD).

The untested 'Hja. 12251' must have contributed considerably to the susceptibility of 'Hja. 60308' 52/- (Fig. 11a:3BC). The same may hold for the untested 'Andie' concerning the susceptibility of 'Hja. 72802' 36/-, but it is quite as possible that it contains Asp 37 (Veg 37) from 'Otra' 23/- without the Oll 4 gene (Fig. 11a:3BC).

'Asplund' 37/- x 'Jadar' 7/33 gave 'Jadar II' 31/- and 'Forus' 21/- (Fig. 11a:3-4CD) of which the former suggests to have the Asp 37 gene and the latter Mas 18.

Two samples of 'Kristina' were tested. One with 10% and the other with 22% attack after inoculation in the field. They agree with one of the parents each, *i.e.* 'Domen' 29/- with Mas 29 and 'Mari' 8/45. The use of 'Kristina' as crossing partner may support the suggestion that it may consist of two components (cf. 'Vega Abed' 22/- (Fig. 7:2DE), 'Jenny' 0/13 (Fig. 7:5EF), and 'Gunnar' 9/- (Fig. 11a:7C)).

'Etu' 77/- (Fig. 11a:4-5B) from 'Bonus' 18/- x 'Varde' 79/- has either the same three genes as 'Varde' or these genes with Mas 18 exchanged by Bon 18. 'Etu' 77/- x 'Otra' 23/- gave 'Jo 1279' 37/- which after all must have the gene Asp 37 (Fig. 11b:3AB). 'Otra' 23/- x 'Varde' 79/- resulted in 'Arra' 56/- with the genes Asp 37 + Mas 18 or Mas 29 + Otr 23 (= Asp 37 + Oll 4), 'Jo 1252' with the same genes, 'Jo 1330' 76/- with Asp 37 + Mas 18 + Mas 29 as in 'Varde' (but not Otr 23), and 'Jo 1343' 41/- with Asp 37 or Mas 18 + Otr 23 (Fig. 11b:3-4BE). 'Jo 1352' 15/- may have either Bon 18 from 'Bonus' 18/- or Mas 18 from 'Varde' 79/- (Fig. 11b:4BE).

'Asplund' 37/- x 'Juli Abed' 0/13 gave 'Presto' 30/- with the Asp 37 gene (Fig. 11a:4-5DE). 'Varde' 79/- x 'Jarle' 14/- gave 'Yrjar' 63/- which must have Mas 18 which the two parents have in common and further Asp 37 (Fig. 11a:5C).

'Vigdis' 38/80 and 'Lise' 57/- from ('Asplund' 37/- x 'DS 295') x 'Varde' 79/- might of course have genes from the unknown 'DS 295', but it is likely that 'Vigdis' has the Asp 37 gene which the two varieties involved have in common and that 'Lise' has Asp 37 + Mas 18 (Fig. 11a:5AE). 'H 1035' 50/- from 'Lise' 57/- x 'Clermont' 30/- may have either Asp 37 + Mas 18 or Cle 30 + Mas 18 (Fig. 11a:5CE). 'Lise' 57/- x 'Sv. 60718' gave 'H. 181-49' 69/- which necessarily has some of its susceptibility from the latter parent (Fig. 11a:5EF). It is most likely that 'Vena' 48/- has Asp 37 from 'Vigdis'[6] 38/80 x 'Lyallpur' 47/-, but in any case 'Vena' must have some of its susceptibility from 'Lyallpur' (Fig. 11a:5AC).

A mutation in 'Fræg' 48/- x 'Paavo' 70/- gave 'Mø 75-278' 39/- (Fig. 11b:4-5CD) which may be explained as having a gene from each of the parents or Asp 37 back from 'Tammi'. If the untested 'Vankkuri' is conditioned like the brother variety 'Tammi' 29/- (Fig. 11a:1A) it is quite natural that 'Teemu' 24/- (Fig. 11b:2AB) (('Vankkuri' x 'Tammi' 29/-) x 'Suvi' 46/-) has the same level of attack, not least considering the close relationship of 'Suvi'. On the other hand the attack of 'Jo 1082' 59/- (Fig. 11b:5-6AB) ('Olli' 4/- x 'Vankkuri') may be explained only as a loss of genes like the case with 'Hafnia' (Fig. 10:4B).

'Suvi' 46/- x 'Otra' 23/- gave 'Jo 1328' 46/- which must have Asp 37 according to that stated above plus something else as in 'Suvi' 46/- (Fig. 11b:3-4AB).

'Jo 0490' ('Seger' 5/- x 'Vega Sv.' 37/-) x 'Suvi' 46/- produced 'Silja' 65/- which may have the gene Veg 37 + another gene or genes from 'Suvi', but certainly not the "pure" Asp 37 (Fig. 11b:2BC).

'Jo 1315' 33/- is a "third-generation" selection in 'Tammi' 29/- which have kept the same level of susceptibility (Fig. 11b:7-8CD).

'Jo 1344' 55/- from 'Otra' 23/- x *H. sativum* Jess. must in one way or another have a large part of its susceptibility from the latter of the parents (Fig. 11b:4BC).

'Pomo' 54/- from (('Seger' 5/- x 'Vega Sv.' 37/-) x a six-rowed line) x 'Bonus' 18/- might well have its susceptibility from the unknown six-rowed line, but it is very likely that it has the genes Veg 37 + Bon 18 from the known involved susceptible varieties (Fig. 11a:5-6CF). 'Pirkka' 65/- x 'Pomo' 54/- resulted in 'Hja. Pokko' 75/- and 'Hja. 70185' 56/- that may have a gene conditioning susceptibility from one parent each if single genes are involved (Fig. 11a:6CE).

'Pirkka' 65/- may have the genes from 'Maskin' 47/- + something else but it may equally well be conditioned by genes from a Manchurian or a Finnish land variety (Fig. 11a:6C).

'Nordlys' 60/- cannot be explained from the level of attack on the parents 'Asplund' 37/- x 'Dore Sv.' 4/73. It may contain Asp 37 for one part, but the other part cannot come from 'Dore Sv.' as it appears in the present experiments (Fig. 11a: 1F).

The attack on the tested offspring of 'Nordlys' 60/- x 'Pirkka' 65/- might be explained from the known genes involved (Fig. 11a:6BE), but if no backcrosses were made after crossing with the land varieties in breeding their parents, they may equally well be conditioned by something else. The varieties or lines in question are 'Bode' 24/-, 'Vå 13002' 17/-, 'Vå 14047' 20/-, 'Vå 17128' 14/-, 'Vå 17165' 60/-, and 'Vå 17236' 20/- with only one on the same level as the parents (Fig. 11b:1AE). The others might contain Mas 18 or Bon 18.

D. Other susceptible and highly susceptible barleys. A group of related American barleys occurring in the collection were screened together with the other barleys. They are moderately resistant, as indicated above, or susceptible to very susceptible after inoculation with the population of the pathogen in the field. The group was bred into 'Suvi' 46/- via 'OAC 21' 15/- (Fig. 11b:6-7BC). 'Suvi' 46/- may have part of its suscept-

Table 10. Percentage of attack of barleys susceptible or very susceptible to leaf stripe after inoculation with a population of the pathogen in the field.

Varieties, ancestors and places of origin	Percentage of attack
Abyssinian (Ethiopia)	33
Abyssinian 39 (CI 7224) (Egypt)	27
Ariana (CI 2524) (Africa)	21
Atlas (select. in Coast)	18
Brio Sv. (Sweden)	27
Caja PF (PF M-13 x PF 62 6/6-4)	50
Chevron (CI 1111) (Switzerland)	40
Compana (CI 5438) (from composite cross)	26
Criewener N. Z. (Gatersleben)	25
Criewener 96 (Gatersleben)	18
Deficiens (CI 2325) (Ethiopia)	70
Dønnes (Norway)	23
Finset (Norway)	38
Fløya (select. in Ørnesbygg) (Norway)	20
Fløya Vollebek 1954 (Norway)	75
Inka (Houtres x (Rundkorn mut. x Voldagsen))	31
Kindred (select. in Wisconsin Pedigree of Oderbrüker x Lion)	26
Klementina (Iceland)	32
Kwan (India)	53
Mianwali (Canada)	19
Morocco x Pallas[4]	23
Morocaine x Pallas[4]	47
Montcalm (USA)	26
Ochsenhauser Ria (Germany)	33
Ogalitsu (CI 7152) (U.S.D.A.)	35
Pannier (CI 1330) (China)	83
Peruvian (Peru)	48
Refsum (Norway)	46
Scots Bere (Scotland)	65
Sigur (Iceland)	21
Sjåk (Norway)	34
Spain (-)	43
Stjernebyg (Faroe Islands)	26
Svalöf (Sweden)	17
Tampar (Faroe Islands/Iceland)	47
Triumph (Trumph) (DDR)	44
Tunga (Norway)	20
Velvet ((Manchuria x Lion) x Luth)	23
White Smyrna (Turkey)	17
Wurla (Sweden)	18
Zeiners Deutschmeister (Germany)	17
Zeiners Franken (Frankonia)	21
Average	34.0

ibility from this source. Further, 'Suvi' 46/- x 'Titan' 57/- gave 'Jo 1182' 57/- (Fig. 11b:6-7CD) with a susceptibility that could come from any of the parents. 'Titan' 57/- most likely has its susceptibility from 'Glabron' 55/- which again suggests that it comes from 'Manchuria' (Fig. 11b:6DE). 'Vantage' 50/- may have its susceptibility from more sources including 'Newal' 19/- and 'Plush' 27/- (Fig. 11b:6CD, cf. Tab. 3).

Many other barleys belonging to this susceptible group were screened. Here will be mentioned only those varieties that could not be placed in the pedigrees but which occur in the used literature or are provided for the screening (Tab. 10). Relatively many of these susceptible barleys come from Nordic countries as did the susceptible and very susceptible varieties set up in the 'Asplund'/'Maskin' series of pedigree (Fig. 11a and 11b).

'Caja PF' has the *Laevigatum*-resistance against powdery mildew which suggests that it belongs to the 'Vada' or the 'Minerva' series but its background cannot be traced exactly. The two 'Criewener' barleys may be old varieties related to 'Criewener 403' 1/88 (Fig. 10:1-2DE, cf. the selections in 'Hanna' above). The two Moroccan barleys must have been very susceptible to leaf stripe since the products are still susceptible and very susceptible, respectively, after four backcrosses to the moderately resistant 'Pallas' (Fig. 7:4C). Finally, the very susceptible 'Triumph' is a widely grown variety at present.

4.2. Discussion

4.2.1. The material

The value of the screening is strongly dependent on the purity of the barleys. In order to maintain the barleys in the collections a smaller or larger number were sown side by side at years interval. Under such conditions the possibility cannot be overlooked that spontaneous crossings may occur as well as an intercontamination in the field, nor during threshing. For these reasons attacks on the level of 1-2% need not be characteristic for

a variety. In some experiments it may be difficult to distinguish among symptoms of the common leaf stripe and the dwarfing symptoms. This is why a small percentage of attack may be noted in the highly resistant varieties.

4.2.2. The significance

Much was done in order to make uniform conditions with close to 100% inoculation and maximum attack. Further, the presented results are based on the highest level of attack on each of the barleys. However, the level of significance may be questioned in this very heterogenous material with attacks varying from 0-100% after inoculation either with a population of the pathogen in the field or with a monospore culture using the sandwich method, and further because parts of the screening were made on a small number of plants. For these reasons, we differentiated only a few levels of resistance or susceptibility as indicated in the section on material and methods. This is considered in the pedigree analysis which is based on the degree of agreement between ancestors and offspring and may be regarded as the most probable pattern of inheritance. This is supported by the fact that samples of the same variety provided from different sources in most cases had the same level of attack. Further, sister varieties normally are in accordance with their parents, but possible explanations are given when deviations occur. Some unexplainable cases appear where significantly different levels of attack occurred between different samples of the same variety. In these cases we decided to use the sample in the pedigrees set-up that we received from the breeder, though in some cases it had been easier to explain the results if we had used the other sample. The most extreme differences seen were the two X-ray mutants in 'Bonus' 18/-, 'Mari' with 8% vs. 43%, and 'Pallas' with 4% vs. 24% attack in the sample from breeder and elsewhere, respectively.

It also makes it difficult to speak about differences where two or more varieties with the same level of attack are suggested to have different genes. Situations that occur both in resistant and susceptible barleys are discussed below.

4.2.3. Inoculation with a population or a monospore culture

The difference between the results after inoculation in the field and the sandwich method is obvious. The main reasons may be that in the former case it is made with a population of the pathogen spread with conidia from the attacked plants to the young seeds of the test barleys over a certain period of time, and in the latter of inoculation with mycelium from a monospore culture. Further, this culture is derived from a diseased plant collected before the experiments were initiated. for this reason, it not necessarily belongs to the population used for inoculation in the field.

The set-up of the pedigrees is based exclusively on the field inoculation because the sandwich test has not been used to a sufficient extent for setting pedigrees up in this context. Two series, however, of closely related varieties indicate that analogous inherited connections could be set up on the basis of the sandwich method (cf. p. 47 and 51). Both of them may be traced back to the 'Opal', 'Kenia', 'Maja' series and from them back to 'Binder Abed', selected in 'Hanna'.

The varieties tested in the two series are characterized as resistant or moderately resistant after the field inoculation, whereas they are susceptible or very susceptible after the sandwich method. Though selected in the susceptible 'Rigel', 'Denso Abed' appears equal to its grandparent 'Maja'. The reason for this will be explained below. The very high field resistance of 'Proctor' may be conditioned by an additive effect of a gene from each parent; it then constitutes a case analogous to 'Freja'. The two varieties are, however, extremely different after exposure to the monospore culture. 'Stange' is transgressively more resistant than its parents.

By comparing the results of the two methods as they appear in these two series and in the pedigrees that are set up in figs 6-12 it is obvious that some barleys were highly resistant after both methods, others were resistant or moderately resistant after field inoculation, but susceptible or very susceptible after inoculation with the monospore culture, and still others

were susceptible or very susceptible after both methods. On this basis we conclude that the resistances corresponding to the two methods are governed by different genes. This is most clearly seen in Tab. 7 where all the barleys are highly resistant after inoculation in the field, whereas the range of susceptibility after the sandwich method is 6-80% attack. In some highly resistant varieties the genes might be the same as no differences may be seen. Apart from this, the barleys are nearly always more susceptible after the sandwich method and in only one case more resistant.

4.2.4. Complex breeding

The reaction of the more or less closely interrelated barleys that are included in pedigrees (Figs 6-12) cannot always be explained because of random variation, the complexity of their ancestors, and because not all are tested. The most complex background occurs in the case of a number of varieties from Weibulls into which resistance to dwarf rust (*Puccinia hordei* Otth), Weihenstephan, and Monte Cristo powdery mildew (*Erysiphe graminis* DC. f.sp. *hordei* Em. Marchal) resistance are bred via 'Tellus D_1', 'Tellus M_1', 'Ingrid M_1', 'Arla M_1', and an unknown source of Weihenstephan resistance (Fig. 8:5-6BF) (cf. EWERTSON, 1974, 1979, 1983). Another example is where nematode resistance was bred into the barleys via 'Byg 191' (Barley 191 (English) = pallidum 191 (French) = *Hordeum pallidum cevada vulgare* (*nomen invalidum*, a primitive barley No. 191 in the collection at the Royal Veterinary and Agricultural University, Copenhagen)) (cf. the pedigrees and ANDERSEN, 1961), but there are several other self-explanatory examples.

4.2.5. The Vada-resistance

The main part of the highly resistant barleys found in northwest European collections has 'Vada' as one of its parents (Fig. 6). This fact was first observed by KNUDSEN (1980) without a test for the inheritance.

'Vada' came from 'Gull Sv.' 14/52 x *H. laevigatum*. The resistance may hardly have come from the former parent if it is homogenous, nor from that line of *H. laevigatum* we tested here as it had 15% attack with leaf stripe. *H. laevigatum*, however, is known to be heterogenous (cf. SHEWRY et al., 1979; LINDE-LAURSEN et al., 1982). Therefore, it is likely that this resistance came from this source. This is further supported by the origin of the highly resistant barley line 'Jo 1341' 4/1 from Finland as *H. laevigatum* x 'Jo 0758' though the chance that the latter parent may have carried the field resistance from either 'Bethge III' 7/- (12.5%) or 'Olli' 4/- (6.25%) (Fig. 11b:7-8AB) cannot be left out of account.

The probability that 'Bomi', 'Alf', Havila', and Uffe 'Sejet' carry the Vada-resistance may be explained only indirectly. DROS (1957) mentioned that 'Minerva' 10/- was brought on the market without further selection, whereas the selections for the sister variety 'Vada' were discarded in the first instance, and a better one was selected and brought on the market as 'Vada'. This may or may not indicate the possibility of more heterogenicity in 'Minerva' than in 'Vada'. Further, LINDE-LAURSEN et al. (1982) mention some heterogenicity in 'Minerva'. 'Bonus' 18/- may have been composed of a resistant and a more susceptible component as discussed below in connection with 'Maja'. Nothing else can be stated on the basis of the pedigree that could contribute to the high resistance of the three first-mentioned varieties nor in the ancestors of 'Uffe Sejet' (Figs 7:2AE, 7:4-5AC).

4.2.6. Other resistant barleys

Apart from those with the Vada-resistance, each of the few highly resistant barleys in the pedigrees has a background of its own. The resistance of 'Freja' 2/1 seems conditioned by two genes: one from 'Opal'-'Maja', which likely carries the same gene for resistance, and one from 'Seger' 5/- (Fig. 8:1AB). These are termed Opa 7 and Seg 5, respectively. 'Jo 1345' 0/5 constitutes a comparable case as 'Herta' 7/- and 'Ingrid' 8/- carry the same gene as 'Opal' 7/- (Opa 7), so it cannot have

come from 'Vega Sv.' 37/- (Veg 37) (Fig. 11b:2-3BD, cf. 'Victoria' in Tab. 8). The explanation of why we regard 'Freja' 2/1 and 'Jo 1345' 0/5 as significantly more resistant than their parents lies in the high level of resistance in 'Freja' in three samples obtained from different collections, and in that 'Opal' 7/-, 'Herta' 7/-, and 'Ingrid' 8/-, each with the same gene (Opa 7) are equally attacked.

'Freja' 2/1 x 'Lenta' 9/- gave the highly susceptible variety 'Hafnia' 76/- which should indicate that it is without any resistance genes from the parents but as 'Lenta' 9/- from 'Maja' 3/26 x 'Kenia' 5/- after all should carry the Opa 7 gene; this gene should be in both parents if the suggestions on 'Freja' are correct. Thus, 'Hafnia' 76/- should carry this resistance gene. This might invalidate our supposition unless the reaction of 'Lenta' has something to do with a previously existing heterogenicity of 'Maja' and 'Kenia' which is discussed below (Fig. 10: 4BC).

According to the breeder, 'Alfa' 2/1 should originate from a selection in 'Kenia' 5/- but there are several indications that it has not much to do with 'Kenia'. ANDERSEN (1961) supposed 'Alfa' to be a mutation in 'Kenia', but as it is different in other characters than just the nematode resistance this can hardly be the case; rather it is a question of contamination (Per Lundin, pers. comm.). As indicated by NIELSEN and FRYDENBERG (1972) 'Kenia' appears completely monomorphic today, whereas both the selected varieties 'Alfa' 2/1 and 'Fero' 5/- (Fig. 9:1AB) deviate from 'Kenia' in chromosome banding (LINDE-LAURSEN et al., 1982). These facts indicate that 'Kenia' originally may have been heterogenous.

The highly resistant 'Piikkio' 3/3 (Fig. 10:7A) is a Finnish land variety from 1922 (MULTIMÄKI and KASEWA, 1983).

It is clear that 'Franks Hohenloher' 0/3 carries the resistance from 'Bethge II' 1/3, and after all it is likely that 'Betzes' -/1.4 (CI 6398) has this resistance. The resistance of this sample of 'Betzes' is in agreement with KNUDSEN (1986). We also

tested another sample of 'Betzes' which had 17% attack after field inoculation and 8% after the sandwich method. This is the only case where the susceptibility after the sandwich method appears significantly lower than after the field inoculation (fig. 12:7-8BD).

Outside the connections with the barleys in the pedigrees, we found a relatively high number of highly resistant and resistant barleys (Tabs 4-6). Not much is known about these barleys apart from the observation that several of them also were found resistant by other authors (Tab. 11).

'Rex II' (Tab. 4) is a nematode-resistant selection made in 'Rex Abed' 10/- by Professor C.A. Jørgensen (cf. ANON., 1948, where it is designated 'K.V.A.L.', and ANDERSEN, 1961). 'Rex Abed' was selected in old Danish barley in 1918 by the breeder H.A.B. Vestergaard (IVERSEN, 1919/20; ANDERSEN, 1961). It has nothing to do with the Canadian 'Rex' ('Velvet' x 'Hannchen', WIEBE and RIED, 1961) as formerly assumed (SKOU and HAAHR,1985).

'Tystofte Kors' 0/4.5 (Tab. 5), originally designated 'Tystofte 25' 1/18 (Tab. 7), was described as a cross between 2-rowed and 6-rowed "old Danish barley" (MORTENSEN, 1909; IVERSEN, 1915). After field inoculation, the two samples behave equally well, but they might possibly be different after the sandwich method. The leaf stripe resistance in 'Tystofte Kors' was observed already in 1910 (MORTENSEN et al., 1911) and by comparing this with the resistance of other barleys it is noted that usually it is least attacked (LIND et al., 1917), or so to speak, is nearly always free of the disease (LIND and RAVN, 1918). Though, these results were based on spontaneous infection, these observations may be regarded very realistically because of the usually very high inoculum potential at that time with up to 50% attack on susceptible varieties (LIND et al., 1916; LIND and RAVN, 1918; STAPEL et al., 1976). As far as we have scanned the literature, 'Tystofte Kors' seems to be the first barley variety in the world described as resistant to leaf stripe, and it is still resistant, not only in Denmark but also in U.S.A. (SHANDS and ARNY, 1944) and Canada (LOISELLE, 1985).

Varieties with 'Maja' and/or 'Kenia' as parents and those selected in these two varieties appear with two levels of attack - apart from that of 'Alfa' mentioned above - one level equal to that of the parents and another significantly higher. Further, selections in the latter, susceptible group - 'Denso Abed' and 'Drost A' - exhibit a level of resistance equal to that of their grandparents. Analogous heterogenicities were found by NIELSEN and FRYDENBERG (1972) and LINDE-LAURSEN et al. (1982) who note that 'Opal', 'Maja', and 'Kenia' from 'Binder' x 'Gull Sv.' differ from their parents as they are known today and refer to heterogenicity in 'Binder' (NIELSEN and FRYDENBERG, 1972). 'Maja' and 'Kenia' are also mutually different (SHEWRY et al., 1979). These facts should surprise no one as it is common practice in the breeding that the best lines selected after a cross are bulked to make the new variety, and this is why it may be regarded as natural that properties not selected for may show up in the new varieties. Therefore, these results support the significance of the experiments rather than confuse them.

'Bonus' 18/- may originate from the more susceptible part of the original 'Maja', but also this variety is supposed to be heterogenous, as after X-ray treatment it produced 'Mari' 8/45 and 'Pallas' 4/- that are selected for other characters than resistance to leaf stripe. For this reason, the characters concerning this disease must have been present in the ancestor (cf. LINDE-LAURSEN, 1978; LINDE-LAURSEN et al., 1982). In this context it should be mentioned that three other samples of 'Mari' included in our tests proved susceptible with 22-43% attack (Fig. 7:4-6BC).

4.2.7. Open flowering

Formerly, infection through open flowers was widely discussed but difficult to prove (cf. FUCHS, 1930; ISENBECK, 1930). SUNESON (1946), on the other hand, got excellent results by inoculation through the open male sterile flowers. On the basis of this way of spreading and pathogenesis of the disease (cf. PLAIEN-KAMP, 1976; TEVIOTDALE and HALE, 1976) it should be expected that varieties with a pronounced tendency to open flowering were

more susceptible to leaf stripe than those that almost exclusively have closed flowers. This is not always so, however. 'Freja' 2/1 and 'Herta' 7/-, which are highly resistant and resistant, respectively, have a rather large tendency to flower with open flowers because of their large *lodiculae*, whereas 'Svanhals Sv.' 21/- (Fig. 10:1DE) with small *lodiculae* and nearly exclusively closed flowers is rather susceptible (cf. PEDERSEN and JØRGENSEN, 1965).

4.2.8. Partiel resistance and additive susceptibility promoting factors

Lining up the results after increasing susceptibility, KNUDSEN (1980) got a continuously steep increasing level of attack that made him suppose that many genes might be involved to give a range from zero to almost 100% attack. The presented results give so many examples of explainable hereditaries concerning resistance and susceptibility to leaf stripe that it cannot occur merely by chance. This allows us to suppose that only a relatively small number of genes are necessary to make the broad range of attack. This supposition is plausible because of the examples of additive effect of resistance, intermediary effect of either resistance or susceptibility, and additive effect of susceptibility that are explained on the basis of the pedigrees (Figs 6-12). The most striking evidence of "additive susceptibility promoting factors" (ASPF) or the opposite if we consider resistance is set up in Tab. 9 where the level of attack on more than 50 varieties may be explained on the basis of five or six genes in varying combinations of one to three or four genes in each variety. None of these genes condition more than 40% attack, but they are responsible for 15-80% attack. The genes are named after the basic varieties and their level of attack apart from that of 'Maskin' 47/- in which we assume two genes (Mas 29 and Mas 18) are present "in order to get the table working".

'Tammi' 29/- from 'Olli' 4/- x 'Asplund' 37/- (Fig. 11a:1AB) appears intermediary between the parents. For this reason it carry both genes (Oll 4 and Asp 37). 'Otra' 23/- from 'Tammi' 29/- x 'Edda' 34/- (Fig. 11a:1-2AB) is on the same level and then is

suggested to carry either the genes Oll 4 and Asp 37 or Oll 4 and Veg 37. These combinations were traced in the later offspring. In this context it should be mentioned that SHANDS and ARNY (1944) found 20% attack in the 'Olli' x 'Asplund' cross.

There are two problematic cases in the pedigrees because of two significantly different samples of each of 'Kristina' (10/-, 22/-) and 'Åsa Sv.' (2/69, 32/89). The former level of attack in each is that on the sample provided from the breeder. It is shown as striking, however, that both 'Kristina' 10/- and 'Kristina' 22/- could come from its parents, 'Mari' 8/45 x 'Domen' 29/- (Fig. 11a:4A). Further, that 'Vega Abed' 22/- from 'Lofa Abed' 2/51 x 'Kristina' is reasonable if its level of attack is 22%. 'Agneta' 70/- may have only the gene Veg 37 if the sample of 'Åsa Sv.' with 32% attack is one of the ancestors (Fig 11a: 2D). This causes us to assume that 'Kristina' and 'Åsa Sv.' have been heterogenous.

4.2.9. Comparison of the results with those of other outhors

Additive effects comparable with our results were observed by ARNY (1945b) who found three factors involved in the resistance of 'Oderbrücker' and that two varieties may have factors in common, by PRASAD (1974) who found a cumulative effect of genes for resistance to leaf stripe, and by SUNESON (1950) who concluded that the resistance may be dominant or recessive and involves more than six genes. Further, he found that the resistance of 'Club Mariout' may be conditioned by at least two genes in a "multiple gene action".

Several other authors have screened series of barleys for resistance to leaf stripe and in many cases the same varieties have been involved. The nine largest of these investigations are surveyed in Tab. 11 in order to facilitate comparison of similarities and differences though it may be done only in general terms because of differences in methods and inoculation potential.

Table 11. Comparison of the different author's screening of barley varieties for resistance to barley leaf stripe. Percentage of attack.

Name	I* f	I* s	II	III	IV	V	VI	VII	VIII	IX
1 Modia (CI 2483)	0	0					0	0		
2 White Gatami (CI 920)	0	0						1		
3 Vada	0	0	0.3	0.6						
4 Georgie	0	0	0	1.2						9.2
5 Alf	0	0.7	0.2							
6 Havila	0	0.7		5.8						
7 Betzes (CI 6398)		1.4							0	0
8 Zita	0	2.0	0	0	0					
9 Ishtar (CI 1615)	0	3.4	0					0		
10 Tystofte Kors (CI 918, CI 6503)	0	4.5	0.5					2-3		
11 Dorsett (CI 4821)	0	6.2						1		
12 Juli Abed	0	13.3						27-43		
13 Jenny	0	18.2		11.3						
14 Gatami (CI 1413)	0	41.2	1.4				6	0		
15 Proctor	0	49.0	0.8							
16 Cornutum (CI 2215)	0.5	1.0	2.0				0	0		
17 CI 4623	0.7	0	0.4				0	0		
18 CI 3694	1	0					0	0		
19 Alva	1	0	1.1	6.2						
20 Murasaki Mochi (CI 5899)	1.5	1.7	0							
21 Jupiter	2	0		1.8						
22 Freja Sv.	2	1.0							0.5	23.7
23 Lofa Abed	2	50.7	0.7	5.6	1.1					
24 Varunda	2	50.7	2.8							
25 Åsa Sv.	2	69.1				89				
26 Stewart (CI 6112)	3		1.0					0		
27 Prisca W	3	5.3	5.9	22.0						
28 Black Hull-less (CI 2277)	3	12.6	1.3					29		
29 Warrior	3	15							14.1	
30 Simba Sv.	3	19.4	2.2	13.9						
31 Emir	3	19.9	3.0	15.0	2.8					
32 Rupal	3	24.8	7.4		8.2					
33 Møyjar	3	28.3			17.0					
34 Albion	3	80.4		33.8						
35 Mutu Abed	4		2.2							
36 Yak Maris	4				14					
37 Velvon	4							16	9.2	
38 Galore	4								14.1	
39 Pallas Sv.	4		7.9		15.1					
40 Olli	4							37	6 0	
41 Kamet Mugi (CI 2253)	4.5	6.1	0				0	0		
42 Welam W	5	32.4	5.0	21.5						
43 Hanna	7								0	
44 Herta W	7					31			0.7	
45 Chinese Black (CI 1969)	7		1.0					0		
46 Claudia	7		4.9	12.5						
47 Ark Royal	7		7.5							
48 Sultan	7		8.9							
49 Togo	7			10.6						
50 Susan	7			11.3						
51 Mandolin	7			14.0						
52 Sårla	7					20				
53 Torkel	7	33.3	2.3	21.2						
54 Wing	7	69.0	1.5		1.9					
55 Ingrid	8					12				51.2
56 Erie (Goldfoil x Alpha)	8							0		
57 Senat	8		3.3				?			
58 Nery Sejet	8		3.7	12.5	2.7					
59 Mari Sv.	8	45.3				19				
60 Flynn (CI 1311)	9							0		
61 Heines Heisa II	10							0		
62 Trebi (CI 936)	10						5	9.0		
63 Selka PF	10		1.0	4.4	1.2					
64 Tyra PF	10		4.9	18.1	5.2					
65 Firlbecks III	10							11.8		

Continues next page

Table 11 continued

Name	I* f	I* s	II	III	IV	V	VI	VII	VIII	IX
66 Black Hull-less (see no. 28)	10						27			
67 Europa (Germany)	10			37.3						
68 Golden Drop	10						26			
69 Mona Sv.	10		1.6	14.2		9				
70 Kristina Sv.	10		10.7			16				
71 Colsess IV (CI 5979)	10							68		
72 Gunhild	11			10.3						
73 Anna Abed	11			15.3						
74 Flavina	11		6.6	18.0						
75 Ansgar W	11	68.1	1.4		15.5					
76 Mirjam Sejet	12		4.4	20.5						
77 Tregal	14								11.4	
78 Albert	14		15.5							
79 Gull Sv.	14	52.0				33				
80 Bay	15								0	
81 Regal 1865**	15								0	
82 Hellas	15		4.8			36				
83 Duplex	15							43		
84 O.A.C. 21	15							83	35.6	
85 Ida	16		5.0	16.2						
86 Carlsberg II	17				4.8					
87 White Smyrna	17							7		
88 Cilla	17		3.9			22				
89 Tron Sejet	17		9.4	15.0						
90 Nordal	17			15.6						
91 Hannchen	17	54.0						7	0	
92 Atlas	18							0	16.6	
93 Bonus Sv.	18		4.3		3.6	16				
94 Newal	19							18	2.2	
95 Primus II	19								14.1	
96 Birgitta Sv.	19	27				34				
97 Monte Cristo	20							35		
98 Tern	21		2.9		18.7					
99 Aramir	21		5.0	26.1	15.2					
100 Vega Abed	22		2.5	20.0						
101 Spartan	25						14	0	0	
102 Kindred	26							83	28.8	
103 Duks	26		0.3	15.1	21.3					
104 Compana (CI 5438)	26								0	11.0
105 Montcalm	26								29.8	
106 Plush	27							51	27.0	
107 Gula Abed	30		3.4	28.2						
108 Abyssinian	33							> 45+		
109 Harry	33		7.5	33.3						
110 Edda Sv.	34				61.0				54.6	
111 Dina Abed	37		8.9	22.7						
112 Primus	38								9.0	
113 Chevron (CI 1111)	40							7		
114 Edda II Sv.	41					62				83.0
115 Birka	42			18.3						
116 Spain	43							23		
117 Triumph	44		5.6							
118 Caja PF	50			32.2	30.1					
119 Vantage	50								3.3	
120 Lami Sejet	50		18.0	21.2						31.7
121 Kwan	53							16		
122 Glabron	55							30		
122a Glabron (CI 4577)	27									
123 Titan	57							10	0	
124 Jet	57							93		
125 Piroline	68								0	50.8
126 Pannier (CI 1330)	83		38.6					92		
127 Velvet (CI 4252)		90.5								
128 Velvet	23	76.0			16.7			45	0	

* The columns headed with Roman numbers present comparable results of different authors. I: The present experiments (f: inoculation in the field; s: inoculation with the sandwich method). II: KNUDSEN, 1980 (Denmark). III: KNUDSEN, 1981 (Denmark). IV: SMEDEGAARD-PETERSEN and JØRGENSEN, 1982 (Denmark). V: KRISTIANSSON and NILSSON, 1975 (Sweden). VI: NILSSON, 1975 (Sweden). VII: SHANDS and ARNY, 1944 (Wisconsin, U.S.A.). VIII: KLINE, 1972 (North Carolina, U.S.A.). IX: METZ and SHAREN, 1979 (Montana, U.S.A.).

** We have tested 'Regal 1865', whereas KLINE tested 'Regal' CI 5030 (cf. Fig. 11b:6E).

+ Eight lines with 45-84% diseased plants.

The most general agreement exists concerning the highly resistant and resistant varieties, though there are exceptions, e.g., 'Georgie' (Tab. 11 no. 4), 'Juli Abed' (Tab. 11 no. 12), and 'Freja Sv.' (Tab. 11 no. 22). The inoculum potential in II (KNUDSEN, 1980) has been rather low, but in spite of that these results are in close agreement with ours. The reason for this may be that both are based on inoculation in the field and in the same country - and may be the same population of the pathogen. When monospore cultures are involved, it is obvious that there are more or less pronounced differences from one author to another, and this is an indirect indication of physiological specialization in the pathogen. It is, however, in accordance with the results of analyses of physiological specialization found by several authors (CHRISTENSEN and GRAHAM, 1934; ARNY, 1945a; KLINE, 1972; NILSSON, 1975; SMEDEGAARD-PETERSEN and JØRGENSEN, 1982; TEKAUZ, 1983; KNUDSEN, 1986). In contrast, KLINE (1971) found nothing pointing to a pathogenic specialization concerning winter barley.

In connection with Tab. 11 (cf. no. 127 and 128), it should be mentioned that we tested five samples of 'Velvet':

'Velvet' (C.A.N. 1133) 3/6.7
'Velvet' 23/76
'Velvet Ottawa' 36/-
'Velvet 447' 39/-
'Velvet' (CI 4252) -/90.5

An isoenzyme test on the resistant 'C.A.N. 1133' and the highly susceptible 'CI 4252' revealed a different pattern (Gunnar Nielsen, pers. comm.). ISENBECK (1930) found 17.7% attack on 'Velvet', whereas KLINE (1972) found zero attack, and SHANDS and ARNY (1944) found 45% attack on 'Velvet' (CI 4252) which, when crossed with 'Tystofte Kors' (CI 918) with 2% attack, gave a product with zero attack which suggests a dominantly conditioned resistance (cf. Tab. 5 and the discussion above). CHRISTENSEN and GRAHAM (1934) indicated 20% attack on 'Velvet' as an average of the cultures that attacked this variety. Their level of attack, however, seems rather lower than ours, but the general

line of comparable varieties, 'Black Hull-less', 'Glabron', 'Spartan', and 'Trebi' (CI 936) follow the same pattern as our results (cf. Fig. 11b and Tab. 11). KNUDSEN (1986) who used the sandwich method found 'Velvet' (CI 4252) susceptible to seven northwest European isolates of the pathogen and resistant to five isolates from Tunesia and Morocco. 'Betzes' (CI 6398) was susceptible to an isolate from Wales and highly resistant to eleven other isolates. 'Havila' and 'Zita' with the Vada-resistance were susceptible to a Finnish and a Moroccan isolate and highly resistant to all others. 'Lofa Abed' was susceptible to a Danish isolate - the same Ax-48 isolate as it was susceptible to in our experiments - and highly resistant to all the others. 'Lami' and 'Warrior' were generally susceptible. 'Glabron' and 'Velvet' were resistant in India, whereas 'Hannchen', 'Pannier' and 'Spartan' varied from highly resistant to susceptible depending on the isolate of the pathogen (MOHAMMAD and MAHMOOD, 1973). LOISELLE (1985) found only 0.33% resistant barleys out of 6999 tested. The following ten of his resistant varieties were also tested by us (our percentage of attack in bracket): 'Bay' (15/-), 'Chevron' (40/-), 'Dorsett' (0/6.2), 'Glabron' (55/-), 'Tystofte Kors' (0/4.5), 'Modia' (0/0), 'Murasaki Mochi' (1.5/1.7), 'Titan' (57/-), 'Velvon' (4/-), 'Warrior' (3/15), and 'White Gatami' (0/0). TEKAUZ (1983) found 'Betzes' (CI 6398) highly resistant as it was in our experiments (Fig. 12:8CD), 'Birka' was susceptible to very susceptible, and 'Herta' and 'Olli' varied from 4.1 to 30.3% and from 2.0 to 51.2%, respectively, whereas in our experiments they are regarded resistant.

ISENBECK (1930) had 'Heils Franken' resistant and 'Bavaria', 'Isaria', and 'Velvet' susceptible in agreement with our results. SUNESON and SANTONI (1943) had 'Atlas', 'Hannchen', and 'Trebi' considerably more susceptible than they were in our experiments. HABGOOD (1972) found 'Anoidium' and 'Proctor' as good to moderately resistant; in agreement with this we found 4/- and 0/49% attacks, respectively. FROGNER (1978) found 'Stange' field resistant, and MAGNUS (1979) found 'Lise' very susceptible. We can agree with both results.

ARNY (1945a) tested three lines of 'Atlas' and found them all resistant to two isolates of the pathogen and susceptible to one. Finally, DICKSON (1956) notes that 'Glabron', 'Hannchen', 'Newal', 'Regal', 'Spartan', 'Trebi', and 'Velvon' were "resistant enough for practical stripe control". We can agree only in the observation concerning 'Velvon'.

5. INHERITANCE OF THE VADA-RESISTANCE

5.1. Material and method

The Vada-resistant 'Zita' (Fig. 6) was crossed with the highly susceptible 'CI 6944' (Fig. 13) and the *bulbosum*-technique was used on F_1 for production of a series of redoubled monoploids to form the basis for an analysis of the inheritance of the resistance.

This technique - described in detail by JENSEN (1977) - is summarized as follows:

1. First crossing using the varieties to be analysed.
2. Isoenzyme test on half kernels of F_1 to ensure hybridization.
3. Sowing the other half of the F_1 kernels.
4. Second crossing: F_1 plants x *Hordeum bulbosum* L.
5. Initiation of embryo formation and rejecting the bulbosum chromosome followed by culturing of the resulting monoploids (haploids).
6. Chromosome redoubling with colchicin.
7. The homozygotic chromosome redoubled plants, the doubled monoploids, were propagated separately and used for the experiments.

By this procedure, the genes from the parents should be uniformly distributed between the doubled monoploids if a fairly high number are produced.

Reciprocal crossings were made and 101 chromosome redoubled monoploids were made from the F_1 plants.

Inoculation of the 101 lines in the field was made by sowing leaf stripe infected seeds in every second row and the lines in between. The test for resistance was performed in the greenhouse with four replicates of 25 plants per pot. The 101 lines were tested also with disease-free seeds inoculated by the sandwich method as described above with 150 seeds sown per line in one tray and without replications.

A statistical analysis was made on the square-root-transformed data of the redoubled monoploids (cf. MOSTELLER and TUKEY, 1949).

5.2. Results

5.2.1. Expression of the resistance

The first crossings were made in the field in the presence of leaf-stripe-diseased plants that produced conidia. Later, when

Figure 13. Barley leaf stripe. Two plots with the Vada-resistant 'Pisa' in the rear, without any attack, and two with the highly susceptible 'CI 6944' in front, without any healthy plants. Both varieties were exposed equally to inoculum.

the resulting hybrids were grown in the greenhouse, 18.0% of them got leaf stripe which they should not have if the Vada-resistance is one gene conditioned and dominant. However, as we could neither know anything about the level of inoculation nor if reciprocal crossings would give equal results, we decided to test a series of reciprocal crossings by use of the sandwich method in order to ensure the highest possible inoculum potential. The results were 23.5% leaf stripe for 'Zita' ♂ x 'CI 6944' ♀ and 28.0% for 'Zita' ♀ x 'CI 6944' ♂ that are not significantly different. 'Zita' and 'CI 6944' had 0% and 98.1% diseased plants, respectively, in this test. This intermediate expression of the disease on the F_1 hybrids suggests that the resistance is semi-dominant. It is, however, also possible that the Vada-resistance is recessive and that the results are an expression of partial resistances disguised behind the "major" resistance gene in 'Zita', compare the analysis below.

The level of attack was measured a month before and at harvest at growth stage 10-10.5 in order to get an impression of the earliness of the symptoms. Some of the resistant redoubled monoploids, that were characterized by having a number of dwarfing plants at harvest, showed weak symptoms already a month earlier but the symptoms had disappeared in a few of them again at harvest. In five susceptible redoubled monoploids nothing was seen a month before harvest, whereas 15 had weak symptoms, 19 had clear symptoms, and four had very pronounced symptoms. The levels of the three last categories were in concordance with the severity of the attacks at harvest.

5.2.2. Disease reaction of the redoubled monoploids

The test after field inoculation included two samples of each of the original parents and was performed with four replicates of 25 kernels sown per pot. One of the two samples of 'Zita' had one diseased plant and the other none. A comparison of the isoenzyme pattern of the diseased plant and 'Zita' showed a clear deviation which made us conclude that this plant had nothing to do with 'Zita'. The two samples of 'CI 6944' had 82% and 88% diseased plants, respectively. In this test the 101 lines had

either a large number of diseased plants with the common leaf stripe symptoms or a smaller number of dwarfing, tillering plants with only weak symptoms (cf. Chapter 3). In 12 of the samples with dwarfing, tillering plants, only one or two plants were characterized as diseased, and obviously in only four had there been a considerable number with each symptom (set in brackets in Fig. 14). These results indicate that we are dealing with two fundamentally different expressions of the disease which is further explained by plotting the square root of the number of plants with each of the two symptoms in the redoubled monoploids against each other in a system of co-ordinates (cf. MOSTELLER and TUKEY, 1949) (Fig. 14). If the two sets of symptoms were related, they should spread from the mean within two times the standard deviation, but in the present case only those with no symptoms lay within this area. Apart from the 16 samples mentioned above, the redoubled monoploids had either the dwarfing symptoms or the common leaf stripe symptoms alone. This means that the redoubled monoploids are either highly resistant

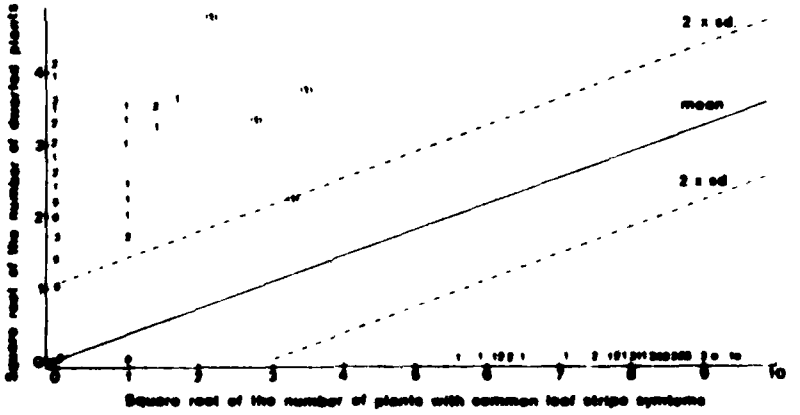


Figure 14 Computer plotted results of comparison of the common barley leaf stripe symptoms with the dwarfing, tillering symptoms in 101 chromosome redoubled monoploids produced with the [illegible] technique after cross of 'Zita' and 'CI 6944'. The amount of attack after field inoculation and test in the greenhouse. Figures represent the number of redoubled monoploids at each spot. The asterisks indicate the attack on the samples of 'Zita' (left) and 'CI 6944' (right), respectively

or highly susceptible and in both cases with mechanism(s) behind that modifies the reactions. Further, on this basis the proportion of resistant to susceptible plants 57:44 is not different from 1:1 indicating a one-gene conditioned resistance.

Two of the redoubled monoploids were lost due to a poor seed setting. The remainder 99 redoubled monoploids were tested with the sandwich method together with six replicates of each of the original parents. Of these 'Zita' had zero attack and on an average 'CI 6944' had 98.1% attack. Only very few dwarfing plants were detected. This explains why there was nearly exclusively zero attack or a high percentage of attack in a 56:43 proportion, which is not different from 1:1, in agreement with the former experiment.

The percentages of attacks in the two experiments were plotted against each other in a system of co-ordinates in order to see how well they are in agreement (Fig. 15). Again, the redoubled monoploids are clearly separated in a highly resistant group and a highly susceptible one. The former group is spread along the ordinate because the dwarfing plants in the test after field inoculation were regarded as diseased in this context. The susceptible group spread around the upper end of the axis between the results with the two original parents (marked with *). A statistical analysis of this group shows a high level of significance ($P < 0.001$) of differences between the lines, and furthermore a correlation coefficient of 0.58. This means that two thirds of the variation are due to the methods. The last third must have other causes. This third, however, is significant enough to suggest that it has a genetical basis that may be considered variations in partial resistance. This partial resistance must have come from 'Zita' because the other parent is the highly susceptible 'CI 6944'.

5.5. Discussion

The infection of the F_1 hybrid in the present investigation shows that the Vada-resistance is neither dominant nor reces-

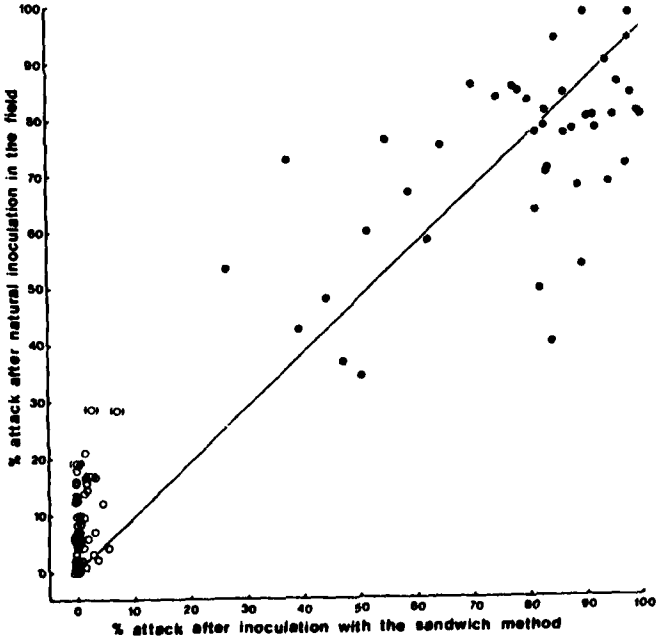


Figure 15. Field inoculation versus mycelial inoculation from a monospore culture using the sandwich method for differentiation of resistance and susceptibility to barley leaf stripe in 99 chromosome redoubled monoploids produced with the *bulbosum*-technique after the cross of 'Zita' and 'CI 6944'. The asterisks indicate the attack on 'Zita' and 'CI 6944', respectively.

sive, but may be different in other cases. ARNY (1945b) found that it depends on the varieties crossed and the number of genes involved. SUNESON (1946) crossed a susceptible male sterile barley with six varieties and found from 6 to 60% attack with leaf stripe in the F_1 produced, but as none of the pollen parents were tested nothing can be said about the level of dominance or recessiveness. Later he (SUNESON, 1950) found most frequently that incomplete dominance occurred, but partial dominance, dominance of susceptibility and "weak resistance" also were present. These findings are in agreement with our results.

At first we used the term "stunted" when we observed the small, tillering plants with weak symptoms (SKOU and HAAHR, 1984 but as this term is used in connection with the common symptoms of

barley leaf stripe, we decided instead to use the term "dwarfing, tillering" for the symptoms now and then occurring on the highly resistant plants. It was characteristic in the test of the redoubled monoploids that none of the highly susceptible lines had any dwarfing, tillering plants, whereas the highly resistant lines had either zero attack, a few dwarfing, tillering plants, or a few plants of both categories (SKOU and HAAHR, 1984; cf. Chapter 3). In the last case, it might be a question of the ability to distinguish the two sets of symptoms.

The level of resistance to barley leaf stripe can be tested only in terms of percentage of attack in occurring as an ability to escape the disease. In order to overcome this difficulty we used the *bulbosum*-technique because it leads instantly to complete homozygotic redoubled monoploid lines and because it is much less time consuming than breeding until F_3 or F_4 generations are reached. On this basis we uncovered a single-gene conditioned resistance in 'Zita' which we termed Vada-resistance because of the origin of the source.

ISENBECK (1930) tested F_3 and F_4 generations and got a large breadth in levels of attack depending on the varieties crossed. These crossings covered the whole area from zero to more than 60% attack. SHANDS and ARNY (1944) tested many crossings, but it is unclear which filial generation was in question. They selected resistant, susceptible and intermediary lines after the crossing. These experiments were continued in the extensive studies of ARNY (1945b) who tested from F_1 to F_3. Naturally, the clearest results were on F_3, with the lines spread over different levels of attack as in the case in the experiments of ISENBECK (1930). On this basis, he uncovered two types of resistance and two types of susceptibility which in some cases were conditioned by more than one gene. Further, crossing of open flowering male sterile barley with 'Trebi' pointed to a one-gene conditioned resistance (GUNESON, 1950).

The analysis of the Vada-resistance disclosed a substantial variation in partial resistance disguised in 'Zita' where it cannot be expressed because the "major gene" will condition almost zero

attack. It may be expected that these factors are also present in at least some of the highly resistant redoubled monoploids. This or these factors or genes behave like those that condition partial resistance to other diseases (cf. PARLEVLIET, 1981; KNUDSEN et al., 1986).

In itself such a partial resistance will be only of academic interest in connection with the analysis of the inheritance because the resulting disease levels will be unacceptable in any instance.

6. ACKNOWLEDGEMENTS

The authors gratefully acknowledge the financial support from Nordic Gene Bank for the screening of the barley collection. Further, the authors wish to express their gratitude to all those - listed in alphabetic order - who provided seed samples (lab. l) and/or contributed with valuable informations or proposals: Reino Aikasalo, Hankkija Plant Breeding Institute, Finland, Kirsten Andersen and Sigurd Andersen, The Royal Veterinary and Agricultural University, Copenhagen, Denmark, Ilse Bollerup, Pajbjergfonden, Denmark, J. Chr. Nørgaard Knudsen, Anton Ioft A/S, Denmark, Per Lundin and Jørgen Löhde, Weibulls AB, Sweden, Rolf Manner, Agricultural Research Center, Jokioinen, Finland, John G. Moseman, Field Crops Laboratory, Beltsville, U.S.A., Bengt Nilsson, Sveriges Utsädesförening, Skara, Sweden, Borghild Nilsson, Svalöf AB, Sweden, Jutta Rasmussen, Tystofte, Denmark, Erling Strand, Institut for Plantekultur, Norges Landbrukshøgskole, Norway, and Thorsteinn Tómasson, Agricultural Research Institute, Iceland.

Last but not least, the authors are deeply indebted to J. Helms Jørgensen, Risø, for many encouraging discussions and indispensable proposals, to Jens Jensen, Risø, for help with the statistical analyses, to Gunnar Nielsen and Hanne Johansen, Risø, for the analysis of isoenzymes, and to Ulla Lilholt, Risø, for her very skilful technical assistance.

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AD-A190 348 SCREENING FOR AND INHERITANCE OF RESISTANCE TO BARLEY LEAF STRIPE (DRECHSLERA GRAMINEA)(U) RISOE NATIONAL LAB ROSKILDE (DENMARK) J P SKOU ET AL. DEC 87 RISOE-R-554 2/2

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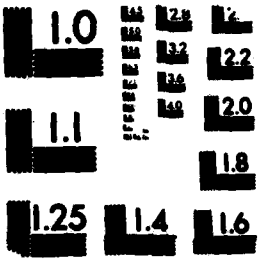

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Title and author(s)

Screening for and inheritance of resistance to barley leaf stripe (*Drechslera graminea*)

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Date December 1987

Department or group
Agricultural Research Department

Groups own registration number(s)

Project/contract no.

Pages 96 **Tables** 11 **Illustrations** 15 **References** 81

ISBN 87-550-1378-3

Abstract (Max. 2000 char.)

Screening of 1029 barleys revealed that a considerable high level of resistance to leaf stripe occurs in a large number of northwest European barleys. The resistance is present in a row of old barleys which have been widely used in the breeding. This level of resistance, however, is not high enough for avoiding chemical seed dressing for control. Almost absolute resistance is found in 'Vada' which have been widely bred into the northwest European barleys. A series of redoubled monoploids produced by the *Bulbosum*-technique were used for genetic analysis of the Vada-resistance that showed to be semidominant and monogenic. Some other highly resistant varieties were mainly found among barleys from other continents and Russia. Pedigree analyses of a large number of the barleys revealed the occurrence of transgressive, additive or intermediate effects of the resistance genes leading to levels of partial resistance. A comparison with the results of other investigations indicate physiological specialization of the pathogen on a world-wide basis.

Descriptors

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