Euphytica **63**: 141–152, 1992.
R. Johnson and G.J. Jellis (eds), Breeding for Disease Resistance

Discovery, characterization and exploitation of Mlo powdery mildew resistance in barley

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Key words: barley, disease resistance, *Erysiphe graminis hordei, Hordeum vulgare*

Summary

Mlo resistance to barley powdery mildew is a relatively new kind of resistance. It was originally described in a powdery mildew resistant barley mutant in 1942 and has been mutagen-induced repeatedly since then. About 1970 it was also recognized in barley landraces collected in Ethiopia in the 1930s. It is unique in that 1) Mlo resistance does not conform to the gene-for-gene system; 2) *mlo* genes originating from different mutational events map as non-complementing recessive alleles in one locus; 3) all alleles confer the same phenotype, though with small quantitative differences; 4) it is effective against all isolates of the pathogen; and 5) the resistance is caused by rapid formation of large cell wall appositions at the encounter sites preventing penetration by the fungus. Powdery mildew isolates with elevated Mlo aggressiveness have been produced on barley in the laboratory, but have not been found in nature. Mlo resistance is considered very durable. The exploitation of Mlo resistance has been hampered by pleiotropic effects of the *mlo* genes, viz. necrotic leaf spotting and reduced grain yield, but they have been overcome by recent breeding work. During the 1980s Mlo-resistant spring barley varieties have become cultivated extensively in several European countries, in 1990 on about 700,000 ha.

Introduction

The Mlo resistance of barley (*Hordeum vulgare*) to the powdery mildew fungus (*Erysiphe graminis* f. sp. *hordei*) is a relatively new kind of resistance. It has become widely utilized recently in European barley breeding and production. It combines the main advantages of the other two kinds of mildew resistance amenable for barley breeding, viz. race-specific (gene-for-gene), and partial (horizontal) resistance, by being monogenic, non-race-specific and durable.

The main characteristics of Mlo resistance have been reviewed by Jørgensen (1984, 1987, 1991) based mainly on studies of mutagen-induced *mlo* resistance genes. In the present chapter, the current knowledge on Mlo resistance derived from spontaneously arisen and mutagen-induced *mlo* resistance genes, the characteristics of Mlo resistance, and the exploitation of this resistance in European barley breeding and production are reviewed. Selected references to recent literature only are given.

Mutagen-induced Mlo resistance

The first powdery mildew resistant mutant of barley, Mutante 66 (M66), was induced by X-rays in the German variety cv. Haisa and described in 1942. Subsequently, many powdery mildew resistant mutants were described. Ten of them (Table 1)

were shown to possess independently induced mutant genes in one locus (Jørgensen, 1976). These ten alleles were designated *mlo1* to *mlo10*. In the 1970s and 1980s many more Mlo mutants were reported, e.g. from Japan (Yamaguchi & Yamashita, 1985), Sweden (Lundqvist, 1991) and Germany, East (Hentrich, 1979) as well as West (Röbbelen & Heun, 1991).

In total more than 150 *mlo* mutant genes have been reported to be induced in a variety of genetic backgrounds constituting mainly highly bred varieties of 2-rowed spring barley. Many of these mutants were described in the literature and made available for research and breeding purposes. In the three decades from the mid 1940s to the mid 1970s *mlo* powdery mildew resistance genes were known from induced mutations only (Jørgensen, 1971, 1976).

Spontaneously occurring Mlo resistance

German expeditions to Ethiopia in 1937 and 1938 collected numerous barley seed samples, which were described in detail in the subsequent years on bulk samples and single-plant progenies (designated E.P. (Einzelpflanzen nachkommenschaften)) (Table 2) at Halle-Hohenturm in eastern Germany. Some data and much of the plant material was lost during the second world war, but Giessen et al. (1956) published extensive data on 250 Ethiopian accessions. Some of them were resistant to powdery mildew in the field and to individual mildew isolates when tested in the greenhouse. Most of the resistant ones were 2-rowed, awnless or awnletted, naked spring barleys collected at locations Bulchi Gofa, Demhi Dollo and Ubamer Baco in southwest Ethiopia. The outstanding powdery mildew resistance of some of these accessions or plant progenies (Table 2) viz. 9 from the E.P. collection at Halle-Hohenturm and 30 from the Abyssinian collection at Köln-Vogelsang was described by Hoffmann & Nover (1959) and subsequently by Nover (1968, 1972) and Meyer & Lehmann (1979). A monogenic, recessive inheritance was shown for two of them (Nover, 1972). The allocation of these genes to locus *mlo,* previously known from the induced mutant genes only, was proven by Nover & Schwarzbach (1971) and Jørgensen (1971). The spontaneously occurring *mlo* allele in the Ethiopian barley line Grannenlose Zweizeilige was designated *mlo11* (Jørgensen 1976). The exact identity of several of the Ethiopian Mlo-resistant lines is dubious because many of them have been shuttled back and forth between breeding institutions and gene banks in Europe and USA, and assigned various designations over time (Table 2). One important line, a six-rowed black seeded line, designated L100 at the former Foundation for Agricultural Plant Breeding in The Netherlands (and in the European Barley Disease Nursery), is deposited at the Dutch Centre for Genetic Resources in Wageningen as accession number GGN00527. It apparently originates from Halle-Hohenturm or the gene

Table 1. Origin of ten barley mutants with Mlo powdery mildew resistance

Mother variety	Mutant	Country	Year	Mutagen	Allele
Haisa	M66	Germany	1942	X-rays	mlo1
Vollkorn	H3502	Austria	1953	X-rays	mlo2
Malteria Heda	MC20	Argentina	< 1960	γ-rays	mlo3
Foma	SR1	Sweden	1961	X-rays	mlo4
Carlsberg II	R5678	Denmark	1963	EMS	mlo5
Carlsberg II	R6018	Denmark	1963	EMS	mlo6
Carlsberg II	R7085	Denmark	1963	EMS	mlo7
Carlsberg II	R7372	Denmark	1963	EMS	mlo8
Diamant	SZ5139b	Czechoslovakia	1965	EMS	mlo9
Foma	SR7	Sweden	1966	γ-rays	mlo10

bank at Gatersleben (in the former GDR) under the name Abyssinian 1140 or HOR2556 which makes it likely that it originates from the German expedition in 1937–1938. The apparently same accession is also in the gene banks at Braunschweig (FRG) as BBA1621, and at Wageningen as CGN1121.

More recently, Mlo-resistant landrace barleys have been described by Negassa (1985a). Among 421 landrace populations, one collected in southern Ethiopia in the village of Jinka in the province of Gemugofa contained four Mlo-resistant lines, viz. 24 per cent of that population. Mlo resistance was not found at any other location. In total the *mlo* gene was found at a frequency of 0.24 per cent of the material.

Over the past 10 years the present author has screened about 4,100 spring barley accessions (*H. vulgare*) for powdery mildew resistance in the field (Jørgensen, 1988). Approximately 3,200 of them originated from Ethiopia. Twenty-four accessions were suspected to have Mlo resistance and 17 of them that were tested in the greenhouse produced the typical Mlo infection type. Among the 24 accessions, 20 originated from Ethiopia. Where specific locations are known, these were all in the southwest of Ethiopia at Bulchi Gofa, Dembi Dollo, Ubamer Baco, Sulto and Jimma. It thus appears that spontaneous *mlo* resistance gene(s) are found with a frequency of 0.2 to 0.6 per cent in Ethiopian landrace material, but predominantly at a few locations in southwest Ethiopia. This region comprises highland areas with high rainfall (up to 1,500 mm/year) and barley cultivation at altitudes from 1,600 m to above the timberline, where the diversity of barley is highest (Nagassa 1985b).

Characteristics of Mlo resistance

a. Genetics. The Mlo powdery mildew resistance in barley is conferred by a long series of recessive, non-complementing alleles in locus *mlo* of which 10 induced mutant genes are designated *mlo1* to *mlo10* (Table 1) and a spontaneously occurring gene *mlo11* (Jørgensen, 1976). There are, however, many more mutagen-induced alleles, and possibly more than one which has arisen spontaneously. Locus *mlo* is located distally on the long arm of barley chromosome 4 (Jørgensen, 1984, 1987).

The genetic fine-structure of locus *mlo* studied by interallelic recombination (Jørgensen & Jensen,

Table 2. Fourteen barley accessions from Ethiopia known to possess Mlo powdery mildew resistance

Accession designation	HOR number	Other designation	In EBDN[a] from	In IBDN[b] from	Reference[c]
Abyssinian 6	1677		1967		1, 4
Abyssinian 9	42				2
Abyssinian 1102	2551	HOR3036, L94, BBA1465	1970		1, 2
Abyssinian 1105	3280	HOR4257			1
Abyssinian 1126	2574	HOR3075			2
Abyssinian 1139	2558	L99	1970		2
Abyssinian 1140	2556	L100, BBA1621	1969		2, 5
Abyssinian 2193	4259				2
Abyssinian 2231	3210				1, 3
Abyssinian 2232	3540				1, 3
Abyssinian 6208	3025	BBA1400	1969		1, 3
E.P. 79	1504	HOR4408, L92, CI14017	1970	1970	1
Grannenlose Zweizeilige	2937	HOR3028, BBA1437	1967	1971	1, 3, 4
Duplialbum	–	CI11793		1965	4

[a] European Barley Disease Nursery.
[b] International Barley Disease Nursery.
[c] 1) Meyer & Lehmann 1979; 2) Nover 1968; 3) Nover 1972; 4) International Barley Disease Nursery Report 1965; 5) Jørgensen 1977, van Hintum pers. comm.

1979) revealed at least three mutational sites within a distance of about 0.05 per cent recombination. One site apparently comprises two X-ray induced alleles *mlo1* and *mlo4;* another site comprises three EMS-induced alleles *mlo5, mlo8* and *mlo9;* and the third site is represented by the spontaneously occurring allele *mlo11* only. Since the 11 named *mlo* alleles all confer an identical phenotypic expression, it is likely that the dominant wild-type Mlo^+ allele can be changed to the recessive state, *mlo*, by mutational events at one of at least three sites within the locus. Furthermore, the occurrence of recombination within the locus excludes the possibility that deletions, except at the molecular level, can be the cause of the mutations. Lastly, the recombination study proves that the necrotic leaf spotting is a true pleiotropic effect of the *mlo* genes because the susceptible recombinants did not show any sign of leaf necrosis. Other *mlo* mutant genes differ, however, in severity of necrotic leaf spotting and frequency of occasional mildew colonies formed on the leaves (Lundqvist, 1991; Hentrich & Habekuss, 1991; Röbbelen & Heun, 1991) suggesting that some mutant genes disrupt the plants' metabolic and defence activity more severely than others. This and related questions may be solved when the *mlo* gene is cloned and sequenced (Hinze et al., 1991) or otherwise identified at the molecular level (Yokoyama et al., 1991).

The *mlo* resistance genes interact with other genes in the genotype of barley. This interaction is seen mainly as modifications in the frequency of the occasional mildew colonies formed on Mlo-resistant barley, and as different levels of necrotic leaf spotting. The *mlo* genes do not, however, interact with other mildew resistance genes in the plant, and they can be combined with other resistance genes so that multi-resistant plants may be produced combining Mlo resistance, race-specific resistance and partial resistance.

b. Phenotype. The phenotype of Mlo-resistant barley lines is characterized mainly by 1) the occurrence of occasional mildew colonies on the leaves, 2) a more or less pronounced tendency to necrotic and/or chlorotic leaf spotting and 3) a reduced grain yield apparently through reduced grain size.

The resistance of Mlo barleys is universal in that an isolate of *E. graminis* which renders them susceptible has never been found (Jørgensen, 1977; Andersen, 1991). When Mlo-resistant barley seedlings are inoculated with *E. graminis,* the plants remain green except that occasionally there are a few small mildew colonies (Fig. 1). These arise mainly from primary infections in the subsidiary cells adjacent to the stomata (Jørgensen & Mortensen, 1977; Andersen & Jørgensen, 1991). This infection type is denoted by an '0' followed by a '4' in parenthesis i.e. 0/(4). If, however, the barley seedlings have an additional, effective resistance gene conferring infection type 2, the seedlings will exhibit infection type 0/(2). If the additional gene confers infection type 0, the seedlings will show infection type 0 because no colonies will be produced. The frequency of occasional mildew colonies may vary from very low to very high depending on several factors. One is density of inoculum; a high density results in a high frequency of colonies (Jørgensen & Mortensen, 1977). A second factor is the genotype of the powdery mildew isolate. All isolates collected in nature (Jørgensen, 1977) cause the formation of about the same low frequency of colonies (Andersen, 1991). Only an isolate selected for high aggressiveness in the laboratory causes many more colonies, i.e. about 10 per cent of the infection occurring in a compatible interaction between a virulent isolate and a susceptible barley (Andersen & Jørgensen, 1991). A third factor is the particular *mlo* allele. Studies in Germany (Hentrich & Habekuss, 1991; Röbbelen & Heun, 1991) and Sweden (Lundqvist, 1991) show that *mlo* alleles of different mutational origin may differ in the quantitative expression of the resistance i.e. the number of occasional mildew colonies. Fourthly, the host gene background may strongly affect the degree of expression of Mlo resistance in terms of the number of colonies (Jørgensen & Mortensen, 1977).

In the field a substantial amount of powdery mildew has occasionally been observed on Mlo-resistant barley. This appears to be the result of unusual environmental factors viz. large amounts of plant-available nitrogen and/or fast growth, for instance, after rainfall following upon a long dry

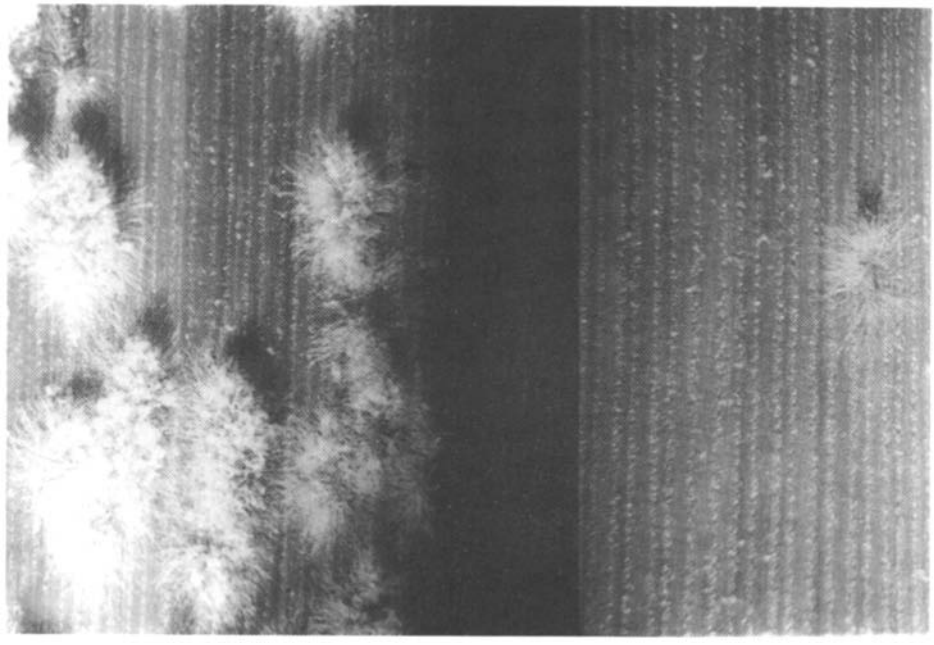

Fig. 1. Mildew colonies developed on non-Mlo-resistant (left) and Mlo-resistant (right) barley seedlings (eight days after inoculation).

period, or heavy and continuous inoculation from severely mildewed barley in neighbouring plots.

The second characteristic feature of Mlo-resistant barleys is a more or less pronounced tendency to develop necrotic (and/or chlorotic) leaf spotting (Schwarzbach, 1976). This is most easily seen at heading (Fig. 2). The necrosis is a pleiotropic effect of the *mlo* genes and is not an expression of hypersensitivity. It may be expressed to a varying degree by mutants with different *mlo* mutant genes (Hentrich & Habekuss, 1991; Röbbelen & Heun, 1991; Lundqvist, 1991). One of the most important factors affecting severity of necrosis is the overall genotype of the barley line with the *mlo* gene. Some genotypes, including lines of Mlo-resistant barley landraces from Ethiopia, may develop quite severe necroses (Fig. 2). Other genotypes, including most of the recent Mlo-resistant spring barley cultivars have only a very slight tendency to develop necrosis (Bjørnstad & Aastveit, 1990; Schwarzbach, 1976). The environment also plays a role in determining the severity and phenotype of necroses. This has been frequently observed but has not been analysed in detail.

The third characteristic trait of Mlo-resistant barley mutants is a reduced grain yield (Hänsel, 1971; Schwarzbach, 1976; Kjær et al., 1990). In many cases most of this effect may be ascribed to simultaneously induced mutant genes that generally reduce plant vigour. In a recent study, three alleles *mlo5, mlo6* and *mlo10* were evaluated in a common but heterogenous gene background (Kjær et al., 1990). All three alleles reduced grain yield and grain size equally, by about 4 per cent, whereas the remainder of the yield reduction was ascribed to other genes independent of locus *mlo*. The reduced grain yield was ascribed to the necrotic leaf spotting that reduces the effective photosynthetic leaf area and the translocation of products of photosynthesis from leaves to spikes during grain filling. There was, however, a considerable variation in grain yield and necrotic leaf spotting among the lines studied, and the best Mlo-resistant lines were equal to the two mildew-susceptible mother varieties Foma and Carlsberg II when tested in disease-free trials.

A considerable number of commercial spring barley varieties with Mlo resistance have been released in Europe. At Risø these barleys have very little or slight necrotic leaf spotting and they have

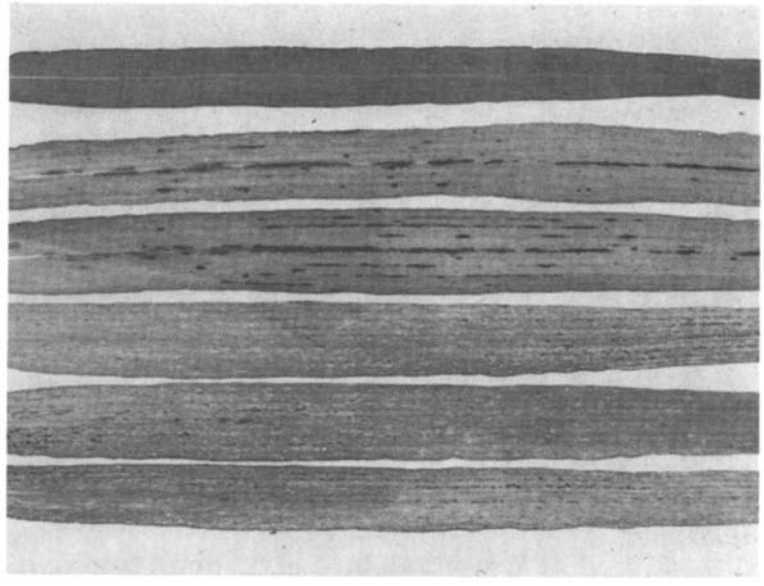

Fig. 2. Necrotic and chlorotic leaf spotting on Ethiopian Mlo-resistant barley. The leaf below the flag leaf of: Proctor (top), Abyssinian 6, Duplialbum, HOR2936, HOR2937, HOR2938 (bottom).

competed well on the European market with non-Mlo-resistant varieties with respect to agronomic traits and to quality traits such as malting quality. These observations indicate that high-yielding Mlo-resistant spring barley lines can be produced provided that appropriate adjustments are made to the genetic background. It also suggests that absence of necrotic leaf spotting may be an easy selection criterion for removing undesirable pleiotropic effects of the *mlo* resistance genes.

c. Histology. Time course studies on barley seedlings inoculated with powdery mildew have revealed that Mlo-resistant barley rapidly develop large cell wall appositions (papillae) below the encounter sites of the pathogen (Skou et al., 1984). The subsidiary cells at the stomata do not develop papillae (Skou, 1985), probably because these cells have a unique physiology and flexible cell walls enabling them to open and close stomata. It has also been shown that non-Mlo-resistant barley develops small-sized papillae when challenged by the infection peg of the germinated powdery mildew conidium, irrespective of the presence or absence of other (gene-for-gene) powdery mildew resistance genes. Other kinds of mechanical damage such as the stylet of an aphid, a microneedle or abrasion by carborundum treatment also induce a rapid formation of large papillae or papilla-like structures in epidermal cells of Mlo-resistant barley (Skou et al., 1984; Skou, 1985; Russo & Bushnell, 1989; Aist & Gold, 1987).

The papillae contain mainly callose but also basic staining material, carbohydrates, phenols and protein (Russo & Bushnell, 1989). An extensive series of studies on papillae and their formation in barley conducted at Cornell University, USA, have substantiated that early papilla formation is the mechanism of Mlo resistance, and that callose plays a decisive role (Bayles et al., 1990; Yokoyama et al., 1991). When Mlo-resistant barley tissue was treated with an inhibitor of callose formation in plants the tissue became susceptible. The papillae in a Mlo-resistant line are about 50 per cent thicker than those in a non-resistant line, 3.9 versus 2.6 μm (Aist et al., 1988) supporting the view that the large papillae constitute a barrier for the infection peg of the fungus. A light absorbing component and basic staining material in the papillae may also be molecular components of Mlo resistance (loc. cit.). Results of other experiments suggest that calcium ion level strongly affects the effectiveness of Mlo resist-

ance. When Ca^{++} chelators were added to Mlo-resistant tissue, it became susceptible, suggesting that the *mlo* mutant gene affects calcium regulation (Bayles & Aist, 1987). Callose deposition in plants occurs within minutes of mechanical pertubation. This suggests that the enzyme for callose synthesis, the plasma membrane associated 1,3-β-glucan synthase, may be present in an inactive state at the cell membrane and is activated by the influx of Ca^{++} induced by the pathogen. The mutated *mlo* resistance gene may thus allow for an early, rapid increase in the Ca^{++} level in the host cell (Bayles et al., 1990). This is supported by the finding that the specific activity of 1,3-β-glucan synthase did not differ between inoculated non-Mlo-resistant and Mlo-resistant barley (Pedersen, 1990). It has recently been reported that an aqueous extract from barley leaves induced barley and wheat plants to form large papillae in response to powdery mildew infection (Yokoyama et al., 1991), i.e. made susceptible barley and wheat Mlo-resistant. This further supports evidence that Mlo resistance is conditioned by an early, rapid formation of large cell wall papillae. It also supports the notion that this papilla-based resistance mechanism is present in plant species other than barley; it can be induced in barley and wheat by extracts from barley, cauliflower, cucumber and onion (Yokoyama et al., 1991).

d. Function. The data summarized above can be used to establish the function of Mlo resistance. One firm conclusion is that the Mlo resistance is due to a rapid formation of enlarged cell wall appositions below the fungus' encounter sites. They constitute a physical and chemical barrier that the infection peg can rarely penetrate. The mutational origin and recessiveness of the *mlo* genes suggests that the wild type Mlo^+ gene is a functional one that can be inactivated by mutation. The inactivation may be more or less effective because of the differences seen in the phenotype conferred by some mutant genes. The involvement of several complex molecules such as callose, protein and carbohydrates, and their deposition in the papillae in a layered structure, must involve many genes in the plant. Many of these compounds and processes are common in the normal metabolism in plants. The presence of single, non-functional (mutant) *mlo* genes and the involvement of many functional genes may be explained by assuming that the wild type Mlo^+ gene has a regulatory function affecting the expression of some of the genes controlling normal metabolism (Skou et al., 1984).

The tendency of Mlo-resistant barleys to exhibit necrotic leaf spotting may be the result of a number of metabolic processes being upset in the resistant plants. Any mechanical damage to the barley leaves, and probably other stress factors, may activate these processes. The absence of the genetical control mechanism implies that these processes run out of control when triggered, and that they are terminated only when the affected cell (and neighbouring ones) is exhausted of substrate(s) for the biochemical processes. The reduction in the tendency to form leaf necrosis by manipulating the gene background indicates that some of the genetic regulation missing in Mlo-resistant barleys may be compensated for by that of other genetic factors.

The Mlo resistance mechanism is limited to pathogens that infect living epidermal cells. It does not affect diseases such as leaf rust, *Puccinia hordei,* because the germ tubes of rust spores infect through open stomata and penetrate the mesophyll cells. In the case of diseases such as net blotch, *Drechslera teres,* or scald, *Rhynchosporium secalis,* the germinating spores exude toxins, which kill the epidermal cells, and thus inactivate possible effects of gene *mlo.* In practice, Mlo-resistant spring barley varieties react to diseases other than powdery mildew in the same way as non-Mlo-resistant barley varieties.

One general defence mechanism of higher plants against mechanical pertubation of the epidermal cell walls is wound-sealing by formation of a callose-rich cell wall apposition below the encounter site. When the recessive *mlo* gene is present, these defence processes occur more rapidly and excessively. Thus the Mlo resistance to powdery mildew barley is related more directly to a physical wound healing than to defence against the powdery mildew fungus. Since many other plant species possess this fundamentally identical wound-healing mechanism, this kind of powdery mildew resistance

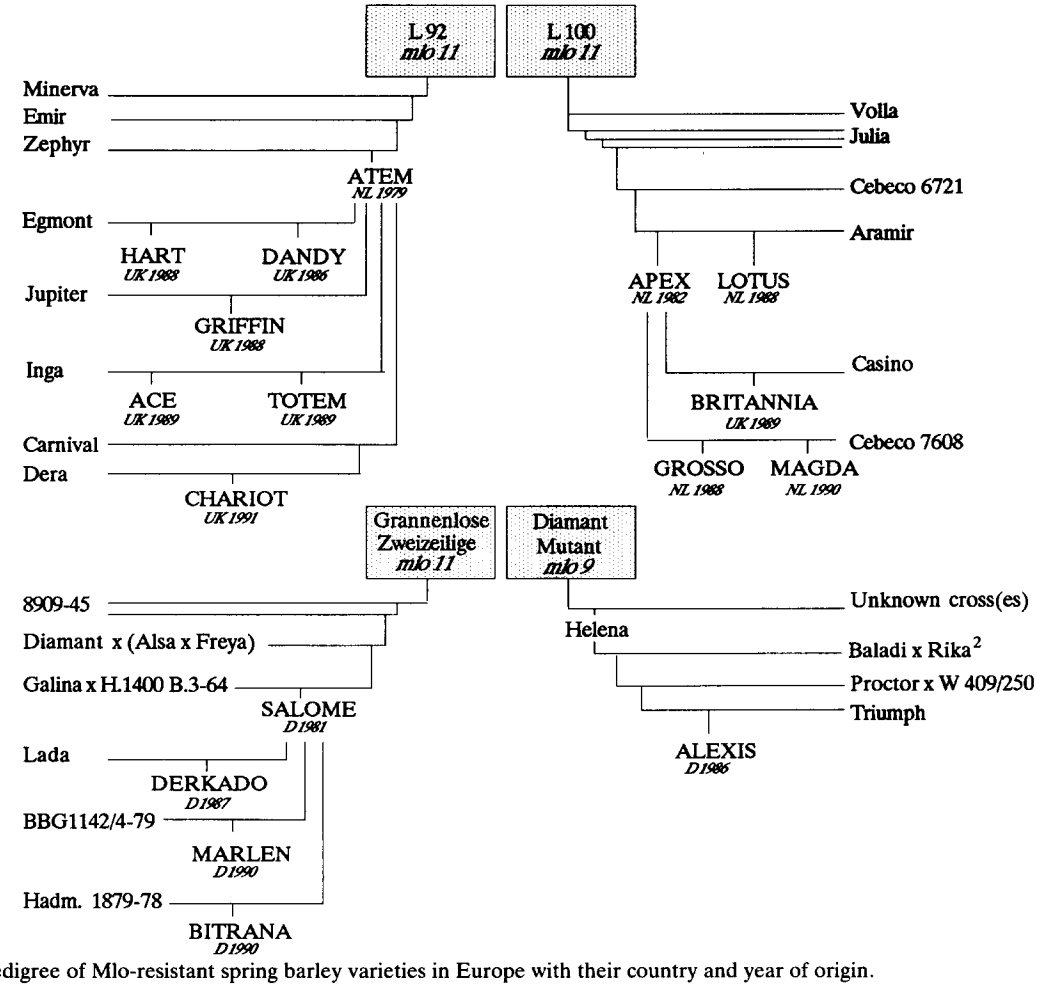

Fig. 3. Pedigree of Mlo-resistant spring barley varieties in Europe with their country and year of origin.

should also be present in other plant species. It should be borne in mind, however, that this resistance mechanism is effective in living epidermal cells only. It is not effective against pathogens producing toxins killing the host cells or against fungi that enter through the stomata and penetrate mesophyll cells.

Exploitation of Mlo resistance

Mutagen-induced powdery mildew resistance genes in locus *mlo* have been exploited in barley breeding for many years (Bjørnstad & Aastveit, 1990; Hänsel 1971; Schwarzbach, 1976; Czembor & Gacek, 1991). Due to the undesirable pleiotropic effects of the *mlo* genes viz. necrotic leaf spotting and reduced grain yield, and the availability of several other highly effective resistance genes such as *Mla7, Mla9* and *Mla12,* barley breeders tended to neglect Mlo resistance and concentrate on the other sources. During the 1970s and early 1980s, the ephemeral effectiveness of the race-specific genes became obvious and the possible durability of Mlo resistance became apparent. This probably led barley breeders to reassess the potential of Mlo resistance. In spite of the fact that the great majority of research was done on mutagen-derived *mlo* resistance genes, it was three Ethiopian sources of Mlo resistance, all probably with gene *mlo11,* that were first introduced in commercial varieties on the European market.

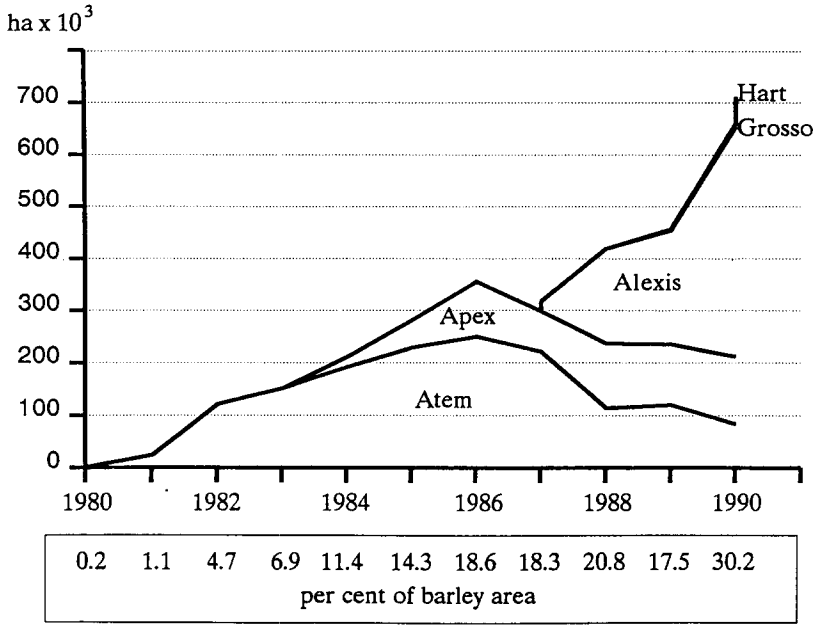

Fig. 4. Area (in ha × 10^3) cultivated with five Mlo powdery mildew resistant spring barley varieties and as percentage of the total spring barley area in the following European countries (from year): Austria (1981); Belgium (1980); West Germany (1985); Great Britain (1982); The Netherlands (1980); Denmark (1989); and Italy (1990).

The first Mlo-resistant barley variety in Europe derived its resistance from the Ethiopian accession L92 (= E.P.79 in Table 2) that gave rise to the variety Atem (released in The Netherlands in 1979) and Atem's descendants released in UK (Fig. 3). Shortly afterwards the variety Apex with Mlo resistance from the donor L100, (Table 2) was released by another breeder in The Netherlands in 1982. Another four varieties with this resistance were marketed subsequently (Fig. 3). The two donor lines L92 and L100 were selected for outstanding powdery mildew resistance in the European Barley Disease Nursery and incorporated into the prebreeding programme at the former Foundation for Agricultural Plant Breeding, The Netherlands, from where advanced breeding populations were released to Dutch barley breeders (L. Slootmaker, pers. comm.). In the former GDR, the variety Salome was marketed in 1981 with gene *mlo11* derived from Grannenlose Zweizeilige (Table 2). Salome was used extensively in variety mixtures in East Germany throughout the 1980s (Gabler & Fritsche, 1991; Wolfe, 1992). Its descendants Derkado and Krona, Bitrana and Marlen (Lau, pers. comm.) were released in 1987 and 1991, respectively (Fig. 3). The fourth source of Mlo resistance is distinctly different from the former three. It is line Helena derived from mutant SZ5139b (Table 1) carrying gene *mlo9* mutagen-induced in the variety Diamant (Schwarzbach, 1976). It gave rise to the malting barley variety Alexis (Fischbeck, 1992) marketed in West Germany in 1986.

Three of these varieties have been widely cultivated in western Europe (Fig. 4). Atem was popular in Austria, Belgium, The Netherlands and particularly in the UK. Apex was, and still is, widely grown in Austria and West Germany. During the last few years, Alexis has become very popular (Fig. 4), particularly in western Germany where it occupied around 35 per cent of the spring barley area in 1990, and in Denmark and Italy where it occupied about 20 per cent of the spring barley area in 1990. Over the past 5 years Mlo-resistant varieties have covered around 20 per cent of the spring barley area in western Europe (Fig. 4), rising to about 30 per cent or more than 700,000 ha. In 1990

two additional varieties, Grosso and Hart have become established in Austria and the UK, respectively.

Durability of Mlo resistance

The durability of Mlo resistance to powdery mildew in barley has been a subject of concern in recent years (Andersen, 1991; Andersen & Jørgensen, 1991; Jørgensen, 1984; Schwarzbach, 1979, 1987).

The rapid increase in the area of spring barley with Mlo resistance cultivated in Europe over the last decade (Fig. 4) may have served as a selective force favouring the emergence and multiplication of powdery mildew with elevated Mlo aggressiveness. Occasional reports have described finds of field samples of mildew with elevated Mlo aggressiveness (Schwarzbach, 1987), but the putative aggressiveness of some of them has not been confirmed (Schwarzbach, 1987; Andersen 1991). An analysis of the extent of cultivation of Mlo-resistant barley in Europe and its correlation with the severity of powdery mildew on Mlo-resistant and non-Mlo-resistant barley (data from the European Barley Disease Nursery 1976–1988) did not disclose any general trend towards an elevated level of Mlo aggressiveness, nor any individual location (local populations) with elevated aggressiveness (Andersen, 1991) in countries with extensive cultivation of Mlo-resistant barley.

Considering the data available, it appears safe to predict that Mlo resistance will be a very durable powdery mildew resistance of barley. If, however, Mlo-resistant spring and winter barley varieties are grown extensively, it is possible that the powdery mildew fungus will slowly but steadily evolve increased aggressiveness and gradually cause disease that may approach the threshold level for crop losses. Therefore, it has become highly relevant to initiate research on the potential of the fungus to 'overcome' Mlo resistance, to devise models and strategies that may aid in preserving the durability of Mlo resistance, and to develop and expand survey programmes for the occurrence of elevated levels of Mlo aggressiveness in European powdery mildew populations.

Conclusion

The Mlo resistance to powdery mildew in barley has been available to plant breeders for about 50 years as induced mutant genes in several high-yielding European spring barley varities, and since about 1970 from Ethiopian landrace barleys. Extensive studies mainly on induced mutants with *mlo* resistance genes disclosed the mechanism of the resistance, its world-wide effectiveness, its possible durability and its undesirable pleiotropic effects. The latter caused plant breeders to emphasize other promising sources of resistance rather than Mlo resistance. More by chance than upon deliberation Mlo resistance from three Ethiopian landraces, probably all with the spontaneously occurring gene *mlo11*, were introduced in commercial varieties around 1980. These varieties rapidly became progenitors for many new varieties. The other source of Mlo resistance is the induced mutant gene *mlo9*. During the 1980s Mlo-resistant varieties have become widely cultivated in many European countries; in 1990 on more than 700,000 ha or close to 30 per cent of the spring barley area.

Acknowledgements

The author is indebted to many colleagues for encouragement and constructive criticism over the years, and to those who have provided unpublished information for the present paper.

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