https://doi.org/10.17221/99/2019-PPS

# Characteristics of powdery mildew and its importance for wheat grown in Poland

*Aleksandra Pietrusińska[1]*, Anna Tratwal[2]*

*[1]National Centre for Plant Genetic Resources: Polish Genebank, Plant Breeding and Acclimatization Institute – National Research Institute, Radzików, Poland*
*[2]Department of Pests Methods Forecasting and Economy of Plant Control, Institute of Plant Protection – National Research Institute, Poznań, Poland*

**Corresponding author: a.pietrusinska@ihar.edu.pl*

**Citation:** Pietrusińska A., Tratwal A. (2020): Characteristics of powdery mildew and its importance for wheat grown in Poland. Plant Protect. Sci., 56: 141–153.

**Abstract:** Powdery mildew of grasses and cereals (*Blumeria graminis*) is a fungal plant disease which is caused by species of fungi from the *Erysiphaceae* order. *B. graminis* is a biotrophic parasite, biologically diverse parasite with a high degree of specialization in certain host species and with numerous physiological breeds adapted to different varieties of a particular host species. In Poland, powdery mildew of cereals and grasses is recorded every year, and its greatest intensity is in south-eastern and south-western regions. The degree of infestation by *B. graminis* varies every year. This means that the disease occurs every year, in greater or lesser severity. Therefore, it requires monitoring (harmfulness thresholds) and chemical control practically in every vegetation season. Nowadays, an important role is played by immunological breeding. In breeding programs, resistance genes from wild crop forms, primitive and indigenous varieties are an effective tool. The introduction of effective resistance genes into cultivated varieties is a common procedure used in breeding program.

The aim of this study was to describe the fungal disease of plants from the group of powdery mildews caused by *B. graminis* as an overview.

**Key words:** *Blumeria graminis*; cereals; resistance genes; incidence; characteristics; resistance genes

Powdery mildew is a very important disease of crops with distinctive symptoms and epidemiology. Powdery mildew is widespread and affects various plant species in different climatic zones. The disease is common in cereals and many species of grasses, and is one of the most dangerous fungal disease of wheat and barley, every year causing losses in yield volume and quality. It is less damaging to oats and rye, and until recently did not affect triticale. Powdery mildew occurs throughout Poland, but is most damaging in the coastal regions, and south-eastern and central parts of Poland.

Powdery mildew of cereals and grasses is becoming more and more important in the times of climate change. Global warming and prolonged periods of drought may reduce the incidence of powdery mildew of cereals and grasses in some regions where cereals are grown. The disease may attack crops that have not been seriously affected before. Considering this, new virulent races of *Blumeria graminis* overcoming the resistance of cereal cultivars may evolve.

The aim of this paper is to present the problems related to powdery mildew of cereals and grasses and the use of effective sources of resistance to *B. graminis* f. sp. *tritici* for wheat grown in Poland.

**Description of *Blumeria graminis*.** Powdery mildew is caused by fungus *Blumeria graminis* DC. Speer (synonym *Erysiphe graminis* DC.) from the family *Erysiphaceae*, order *Erysiphales*, class *Leotiomycetes*, phylum *Ascomycota*, kingdom *Fungi*,

https://doi.org/10.17221/99/2019-PPS

domain *Eukaryota* (Kirk et al. 2001; https://www.cabi.org/isc/datasheet/22075).

*B. graminis* is a biologically diverse obligate parasite, highly specialized in parasitism on specific host species (special forms) and exists in numerous physiological races adapted to different cultivars of a particular host species.

A special form is identified based on the ability of a given single-spore culture of *B. graminis* to infect a specific plant species, and the physiological race based on the ability to infect cultivars of a given host species with specific resistance genes (Gacek 1983). Eight special forms of *B. graminis* have been reported based on host specialization (Marchal 1902; Oku et al. 1985). These forms of the pathogen attack wild grasses of the genera *Dactylis*, *Agropyron*, *Poa* and *Bromus*, and cereals from the genera *Triticum, Hordeum*, *Secale* and *Avena* (Dewey 1983; Troch et al. 2014). It can be assumed based on this classification that *B. graminis* f. sp. *tritici* mainly infects wheat, *B. graminis* f. sp. *hordei* infects barley, *B. graminis* f. sp. *avenae* infects oats, *B. graminis* f. sp. *secalis* infects rye, and *B. graminis* f. sp. *triticale* infects triticale (Troch et al. 2012; Walker et al. 2011).

**Life cycle and symptoms of infection caused by *Blumeria graminis.*** The *B. graminis* life cycle has two stages – ascosporal and conidial. In the ascosporal stage the fungus produces dark brown or black ascocarps (perithecia or cleistothecia or chasmothecia) 135 to 224 µm in diameter. Chasmothecia are covered with filamentous appendages and contain 8–25 asci 70–100 × 25–40 µm in size. Ascospores formed in asci in late summer or early autumn are ovoid, single-celled and 20–23 × 10–13 µm in size. After rainfall, ripe ascocarps break open and release ascospores that infect grass, volunteer cereal plants and germinating winter crops (Gołębniak 1993; Braun et al. 2002; Legler et al. 2012). A white scurf on the infected plant is formed by mycelium, conidiophores and conidia. The pathogen overwinters as mycelium on winter cereals, volunteer plants and wild grasses and can survive until the next growing season (Hacquard et al. 2013; Wicker et al. 2013; Shi et al. 2016). In the spring, the growing mycelium produces conidiophores and conidia that spread infection to new plants. Conidia are colourless, ellipsoid, 24–35 × 12–17 µm in size and arranged in chains on conidiophores formed by hyphae growing on the surface of leaves. The dense mycelium of greyish colour is gradually formed (Wiese 1987). Sporing is favoured by dry and warm weather (Fiedorow et al. 2004). *B. graminis* is an ectoparasite. The fungus assimilates nutrients necessary for its growth and development using haustoria penetrating epithelial cells of the host plant. Plants are infected in a wide temperature range of 5 to 30 °C and air humidity of 50–100%, but the optimal conditions are 12–20 °C and high humidity. Powdery mildew produces conidia as often as every 7 to 10 days (Piarulli et al. 2012; Esmail & Draz 2017; Draz et al. 2019).

In regions where chasmothecium are an important source of infection (e.g. southern Europe, USA), asci are released from the *perithecium* under favourable weather conditions and give rise to primary infections. Experimental studies have demonstrated that fungal spores can be dispersed by wind for several hundred kilometres, initiating many successive cycles of secondary infections during the growing season (Zadoks 1967; Hermansen et al. 1978; Götz et al. 1996). This is extremely important from an epidemiological point of view, and in this respect many researchers have recognized Europe as a reservoir for powdery mildew of cereals and grasses (Limpert et al. 1984).

The first symptoms of powdery mildew of cereals and grasses develop on winter cereals in late autumn or early spring. The most severe symptoms of powdery mildew are observed on the lower leaves. Infection spreads gradually from the lower leaves to higher parts of the stem. Initially, small, fluffy white-grey pustules develop on the surface of leaves or underneath leaves (Daamen 1989). Over time, the disease progresses, and under favourable weather conditions, white fluffy pustules cover increasingly larger area of leaves, sheaths, stems and ears. The scurf on infected parts of plants is white, later turning grey and farinose. Severely infected leaves become chlorotic and gradually die back. In a darkening scurf, small dark brown or black chasmothecia can be seen. The first symptoms of powdery mildew on ears occur on the inner side of the spikelets. Later, the scurf can cover the whole ear. Kernels in infected ears are poorly developed, small, and sometimes malformed (Fiedorow et al. 2004).

**Physiological races of *Blumeria graminis.*** Cultivars of cereal species vary in their degree of disease resistance, which is determined by specific genes. Resistance types are denoted with symbols, for example R1, R2, R3... R*n* (type 1, 2, ..., $n^{th}$ resistance). Some cultivars may also have genotypes ensuring two or more types of resistance, denoted R(1, 2), R(1, 3), R(2, 3), etc.

https://doi.org/10.17221/99/2019-PPS

Virulence types (pathogenicity) of individual physiological races are denoted P1, P2, P3,..., P*n* (type 1, 2, ..., $n^{th}$ pathogenicity). Races with more than one type of pathogenicity in their genotype are denoted P (1, 2), P (1, 3), P (2, 3) etc. (Gacek & Czembor 1983).

The interaction between cereal cultivars and physiological races of obligate pathogens, including *B. graminis,* is consistent with the gene-for-gene hypothesis proposed by Flor (1956). According to this hypothesis, cultivars with a given type of resistance can only be infected by races of pathogen with the corresponding type of virulence. For example, cultivars with resistance type R1 are infected by races P1, P (1, 2), P (1, 3), P (1, 2, 3), i.e. those that have genotypes of a corresponding type of pathogenicity (virulence) marked with number 1. However, cultivars with resistance type R1 will not be infected by physiological races characterised by virulence types P2, P3, or P(2, 3). The resistance type of these cultivars creates a genetic barrier that effectively protects them from infection by these physiological races. Generally speaking, there is no cultivar resistant to all physiological races, and there is no race of pathogen able to infect all cultivars (Czembor 1981; Gacek 1983; Gacek & Czembor 1983, 1988).

The discovery of host specialization in *B. graminis* initiated studies on the identification of physiological races. Initially, the virulence of *B. graminis* f. sp. *hordei* was explored (between the 1930s and 1980s) with the qualitative approach, which involved the identification of physiological races (Mains & Dietz 1930). Race identification was performed using standard test systems of host cultivars with different major resistance genes. Using a differentiating system where *n* is the number of differentiating cultivars, 2*n* number of races can be identified (Day 1974). Following this principle, a system of 10 differentiating cultivars was used to identify 1024 physiological races. This system for describing the virulence of *B. graminis* f. sp. *hordei* served its purpose for several decades until it was considered overly complicated (Gacek 1990). Identification of physiological races (qualitative system) had become overcomplicated because both the number of resistance genes and the number of identified physiological races increased significantly (Gacek & Czembor 1995).

Considering the above problems, Wolfe and Schwarzbach (1975) proposed investigating the virulence of obligate pathogens with virulence analysis using quantitative methods that provide a more detailed description of the pathogen-host interaction. Host specialization in *B. graminis* f. sp. *hordei* in this system is defined by virulence frequency (Wolfe & Schwarzbach 1975; Scott et al. 1980; Gacek 1990). Studies on virulence frequency in the population of *B. graminis* in Poland have been carried out using three methods – analysis of leaf samples infected with powdery mildew and collected from fields, use of mobile nurseries, and the use of the Jet Spore Trap mounted on the roof of a car (Gacek 1987; Gacek & Czembor 1983, 1984, 1988, 1993). Studies carried out in 1993–1996 on the pathogenicity of barley powdery mildew using the Jet Spore Trap revealed an increase in the frequencies of virulences *Va9*, *Va13*, *Va6*, *Va7* + *k* and *Va7* + *Ab*. A strong pathogenic effect of *B. graminis* f. sp. *hordei* populations from neighbouring countries on barley fields in Poland was also reported (Gacek et al. 2004).

A quantitative method of virulence analysis was used in the studies carried out by Czembor and Czembor (Czembor & Czembor 2005). Phenotypic evaluation consisted in determining the frequency of individual virulence on the basis of true powdery mildew colonies on the first leaf of seedlings. For this purpose, a set of cultivars with different resistance genes was used and the evaluation was performed with reference to the susceptible cultivar (Czembor & Czembor 2005). On the basis of the obtained results it turned out that virulence *Vg* + *CP* (93.2%), *Va7* + *Ab* (100%), *Vh* (98.6%), *V*(*La*) (100%), *V(Ru2)* (100%) and *Vra* (100%) were characterized by the highest occurrence frequency. On the other hand, the low frequency of occurrence was indicated by virulence *Va1* + ? (24.3%), *V*(*St1)* + *St2* (27.2%), *Va9* + *k* (28.3%) and *Va3* + (*Tu2*) (10.1%) (Czembor & Czembor 2005).

Monitoring of the dynamics of virulence changes in the population of *B. graminis* f. sp. *avenae* in Poland allowed to determine the dynamics of changes taking place in the population in Poland. The study showed that the diversity of true powdery mildew population in Poland is small, but an increase in virulence level is observed. In 2010, 2 pathotypes were identified, in 2011 five pathotypes, while in 2012 and 2013, six pathotypes were identified (Okoń & Ociepa 2017).

**Incidence of powdery mildew of cereals and grasses on the word include Europe.** Powdery mildew does not spread between cereal species. Until recently, this disease did not affect triticale. However, in recent years there have been an increasing number of reports on infections caused by *B. graminis* in this hybrid cereal. It has been assumed that triticale

https://doi.org/10.17221/99/2019-PPS

powdery mildew was first noted in early 2000 in many regions of Europe where this cereal was grown, probably as a result of the spread of pathogen types specialized in wheat, and the increasing acreage of this cereal (Bouguennec et al. 2014; Troch et al. 2014). Powdery mildew of cereals and grasses rarely infects rye and oats, and does not infect rice, millet, sorghum or maize (Fiedorow et al. 2004).

The highest incidence of this disease is reported from humid and relatively cold areas characterised by high rainfall at the beginning of the growing season, typical for oceanic and continental climates (Bennett 1984). Globally, the highest incidence of powdery mildew of cereals and grasses has been reported from China, the United Kingdom, Germany, Japan, North and East Africa, and the South-Eastern United States. In these regions the disease causes significant damage to the quality and volume of yield (Saari & Wilcoxson 1974; Roelfs 1977; Merchan & Kranz 1986; Cao et al. 2013; Li et al. 2013; Peng et al. 2014; Mandal et al. 2015; Wang et al. 2015). Considering all continents, in 1969–2010 the incidence of powdery mildew was highest in Western and Southern Europe (93%), and in Central and Eastern Europe (72%) (Morgounov et al. 2012). In 1991–2008 powdery mildew of cereals and grasses occurred in all regions of Poland where winter wheat was grown. The multiannual mean for Poland in this period was 25.6% of infected winter wheat stems (Walczak et al. 2009).

Powdery mildew of cereals and grasses has been reported throughout Poland, but it usually causes the most serious losses in the south-east, south-west, in coastal regions, and in central Poland. Its incidence depends on the type and acreage of grown cultivars, the frequency of corresponding virulences in the pathogen populations, and the environmental factors influencing powdery mildew of cereals and grasses. Air humidity in the range of 50 to 100% during rainfall or persistent dew, adequate insolation, and a wide range of temperatures create optimal conditions for the development of the pathogen, germination of spores, and formation of the next generations of conidia. Winter cultivars infected by the pathogen in early spring have poorly developed stems and root systems (Grzeszczuk 2017; https://www.farmer.pl/produkcja-roslinna/ochrona-roslin/progi-szkodliwosci-maczniaka-prawdziwego-zboz-i-traw,63696.html)

**Harmfulness, monitoring and factors influencing of *Blumeria graminis*.** The severity of infection with *B. graminis* varies year to year depending on the weather conditions, but the disease occurs each year, with weaker or stronger effects, and therefore has to be monitored and chemically controlled in almost every growing season in both Europe and Poland.

In European crops production powdery mildew of cereals and grasses causes a significant decrease in the volume and quality of yields and leads to economic losses, both because of reduced productivity and increased costs of production (Everts et al. 2001; Conner et al. 2003). This disease has a devastating effect on grain yield and quality (Everts et al. 2001; Conner et al. 2003; Asad et al. 2014). Under weather conditions particularly favourable for the development of *B. graminis* powdery mildew can cause a 13–30% reduction in grain yield of spring and winter wheat (Kochman & Węgorek 1997; Tratwal & Jakubowska 2004; Jańczak & Pawlak 2006; Lackermann et al. 2011).

For example in Poland, under weather conditions particularly favourable for the development of fungal pathogen *B. graminis* f. sp. *tritici* can cause up in to 30% drop in yield of winter wheat (Kochman & Wegorek 1997; Tratwal & Jakubowska 2004; Jańczak & Pawlak 2006). Powdery mildew of cereals and grasses causes the most serious loss when it affects heads in wheat, and flag leaves and leaves F-1 in barley. In winter barley cultivars infected by powdery mildew of cereals and grasses in early spring, poor development of shoots and root system is observed at subsequent stages of growth. Finally, the yield of barley is reduced. In winter barley the infection with *B. graminis* f. sp. *hordei* limits photosynthesis and increases the intensity of transpiration, which, along with the infection of spikelets, causes a decrease in the number and mass of kernels. Changes in the chemical composition of kernels were also reported, including reduced content of carbohydrates and a slightly increased content of protein. These changes lower the quality of barley grain, which is particularly important in the production of malting barley (Korbas & Mrówczyński 2012; Packa et al. 2018).

In order to effectively limit losses caused by *B. graminis*, the field should be systematically checked. The illustration of plantations or experimental plots is an extremely important element in order to identify the main culprit of the disease together with a description of the condition of plants infected with other fungi. The field should be evaluated, preferably by the same people, at intervals of several days. Depending on the size of the plantation, between 100 and 150 plants are assessed visually.

https://doi.org/10.17221/99/2019-PPS

Assessment of the degree of infestation by the pathogen or pathogens should be carried out from the edges of the field by at least 2 m.

Economic injury levels (EILs) for powdery mildew of cereals and grasses have been established based on popular science literature and weed atlases of plant protection. Depending on the cereal species grown, the threshold of harmfulness varies. Three harmfulness thresholds have been determined for wheat. In the tillering phase, it is 50–70% of infected leaves on which single white clusters of fungus structures are visible. In the shooting phase, the stem is 10% of the plants with the first symptoms of paralysis and in the nesting phase, when the symptoms of pathogen paralysis are visible on the leaf under the flag leaf, flag leaf and spike. For barley, the harmfulness threshold includes the tillering phase up to the earing phase when the disease symptoms are present on the upper leaves and the number of infected stems by powdery mildew and grass is at least 10%. For rye and triticale, the harmfulness threshold includes the stage of shooting into the stalk or at the beginning of earing. Then the disease symptoms are visible on the sub flag leaf and the number of infected stalks is over 20%. There are two economic harmfulness thresholds for oats. The first one is the end of the stemming or at the beginning of ear-topping. During these phases, the clear symptoms of *B. graminis* occur on 15 to 20% of plants. The second threshold is valid for the stalk shooting phase up to the panicle shooting phase. Then the disease symptoms are already visible on the third leaf and appear on the subflag leaf. Then more than 20% of the stalks are infected (http://www.cdr.gov.pl/images/Radom/IOR/pliki/PROGI%20SZKODLIWOCI.pdf; /http://www.minrol.gov.pl/Informacje-branzowe/Produkcja-roslinna/Ochrona-roslin/Integrowana-ochrona-roslin/Metodyki-integrowanej-ochrony-roslin; http;//www.agrofgai.com.pl; https://www.ior.poznan.pl/plik,1749,metodyka-integrowanej-ochrony-pszenicy-ozimej-i-jarej-dla-producentow-pdf.pdf).

Crops can be protected against powdery mildew of cereals and grasses using methods limiting the sources of primary infections. The spread and progression of the disease is promoted by the overwintering mycelium on winter crops, perennial grasses, volunteer plants and live tissues of plants. The disease can be prevented by avoiding too dense sowing and sowing spring crops near the winter crops. The incidence of powdery mildew of cereals and grasses is characteristic for areas with increased use of nitrogen fertilizers in agricultural production, which is why rational fertilization with nitrogen, potassium or phosphorus is an important measure limiting the disease (Cunfer 2002; Felczak 2016). Of note is that climate change may increase or decrease the activity/aggressiveness of fungal pathogens, which in turn may contribute to increase, decrease or shift of the geographical range of fungal pathogen(s) responsible for economically significant fungal diseases of crops (Wójtowicz et al. 2017).

**Assessment of disease caused by *Blumeria graminis* and rating systems.** There are several methods/scales used for the assessment of infection with *B. graminis.* All these scales measure the severity of disease on the analysed plants. Disease symptoms caused by powdery mildew of cereals and grasses are assessed by visual inspection. Most field experiments rely on a 1 to 9 scale in which 9 means no disease, 5 means moderate disease and 1 means disease affecting the whole plant, the most severe disease stage. This graphical/phenotypic scale was first proposed by Saari and Prescott (1975), who investigated diseases of wheat (Saari & Prescott 1975). The scale categorizes resistant and susceptible genotypes, including more and less susceptible/intermediate forms, and the assessment is made using a susceptible and/or resistant reference cultivar. The huge advantage of using this scale is its wide range, allowing for the assessment of disease severity expressed as the percentage of leaves damaged by *B. graminis*. On a 1 to 9 scale the rates correspond with the % range of leaves damaged by the fungal pathogen, in which 1 – 1% of damaged leaves, 2 – 3%, 3 – 9%, 4 – 16%, 5 – 26%, 6 – 37%, 7 – 55%, 8 – 70%, 9 – 89%. In field experiments it is necessary to inspect at least ten leaves/samples on the whole field for good accuracy and reliable assessment. The 1 to 9 scale is commonly used for the assessment of disease severity caused by *B. graminis* in wheat (Paillard et al. 2000; Singh et al. 2016) and barley (Tratwal & Bocianowski 2014; Bengtsson et al. 2017).

Effects of infections, either experimentally induced under controlled conditions in growth chambers, or naturally occurring in the field, are also assessed using the 5-degree (0–4) scale proposed by Mains and Daetz (1930), where 0 – no visible symptoms; 1 – minute necrotic flecks; 2 – larger necrotic flecks and sparse sporulation; 3 – chlorosis, moderate mycelial growth and moderate sporulation; 4 – profuse sporulation of well developed mycelium. Plant lines categorised 0–2 are resistant,

https://doi.org/10.17221/99/2019-PPS

and those categorised 3–4 are susceptible. This rating scale has also been used in controlled trials aimed at the selection of inbred rye lines resistant to *B. graminis* (Bujak & Jurkowski 2013), in studies on new rye-wheat chromosome translocation lines resistant to powdery mildew (An et al. 2013), in studies on linseed, common wheat and triticale naturally infected with *B. graminis* f. sp. (Kowalczyk et al. 2011; Singh et al. 2016; Dhirhi et al. 2017), in assessment of plants experimentally inoculated with isolates of *B. graminis* f. sp. *tritici* from Chinese wheat cultivars (Liu et al. 1999; Yang et al. 2017), and for barley lines (Gupta et al. 2014).

Under controlled conditions plant material can be assessed for susceptibility/resistance to fungal diseases, including powdery mildew of cereals and grasses, using a 1–0 rating scale, where 0 – resistant plants, and 1 – susceptible plants. This system allows for a quick differentiation between genotypes that are completely resistant to the pathogen and those affected, even to a minor degree. This strategy is very popular when searching for new and effective sources of resistance to economically significant fungal diseases, including powdery mildew of cereals and grasses (unpublished data).

A scale for the assessment of fungal disease severity in plants, regardless of the model, is a very important and sometimes indispensable diagnostic tool for the selection of resistant and desirable genotypes.

**Breeding wheat for disease resistance in Poland.** Breeding for disease resistance is a method of protecting crops against fungal pathogens. It is a vital aspect of modern environmentally-friendly plant production since it helps prevent losses in the quality and volume of yield while reducing the use of chemical pesticides. Moreover, breeding for disease resistance gains particular significance in view of ongoing debates in the EU regarding the ban on the use of certain chemical plant protection products. Therefore, the identification of new and effective sources of resistance to fungal diseases of cereals and their further translocation into crops may be a significant contribution to solving the problem of low resistance of crops to powdery mildew of cereals and grasses, and other fungal diseases (Pietrusińska et al. 2019).

An important source of genes that determine resistance to *B. graminis* are wild forms of cultivated plants and their relatives, primitive cultivars, and local cultivars. Sometimes a population of infected plants includes specimens with genotypes making them resistant to one or more pathogens. Such genotypes of plants can be mapped for the presence of a specific gene or a combination of resistance genes. This is especially important because of adaptation to climatic and geographical conditions in which the cultivar is currently grown. It is also noteworthy that such cultivars have an advantage over wild species because of greater productivity, resistance to lodging and better hardiness (Pietrusińska et al. 2018).

For wheat species the most important source of genes determining resistance to powdery mildew are wild species, exotic species, and their relatives, including rye.

Wheat powdery mildew resistance genes mainly originate from ancestors and wild species related to hexaploid wheat (AABBDD), i.e. *Triticum tauschii* Coss. (syn. *Aegilops tauschii*, *Aegilops squarrosa*), *Aegilops speltoides* Tausch (syn. *Triticum speltoides*, *Aegilops aucheri*), *Aegilops ventricosa* Tausch (syn. *Triticum ventricosum*), *Aegilops opata*, *Haynaldia villosa* L. Schur. (syn. *Dasypyrum villosum* L. Candargy), *Agropyron intermedium* (Hausskn.) Roshev, *Triticum timopfeevii* (Zhuk.) ssp. *armeniacum* (syn. *Triticum araraticum*), *Aegilops triuncialis* Linnaeus (syn. *Triticum triunciale*), *Triticum monococcum* Linnaeus (syn. *Triticum aegilopoides*, *Triticum boeoticum*). Wild forms and currently grown cultivars are evolutionarily distant. Because of different levels of ploidy and lack of chromosomal homology, the convergence crossing between such genotypes is less effective and sometimes impossible. Four gene pools have been identified based on the level of genetic (sexual) relatedness: primary, secondary, tertiary and quaternary (Harlan & de Wet 1971; Gepts 2000; Gepts & Papa 2003; Pietrusińska et al. 2018). Detailed characteristics of gene pools have been presented in review papers by Alam et al. (2011) and Pietrusińska et al. (2018).

In the breeding of cereals there are several ways for controlling the susceptibility/resistance of plants to *B. graminis*. One strategy relies on the use of chemical plant protection products. However, findings from genetic studies on resistance are utilized first of all. Cereal plants have two types of resistance to *B. graminis*. The first type is quantitative (horizontal or polygenic) and the second type (complete, vertical and race-specific) is determined by major resistance genes R (Leath & Bowen 1989; Leath & Heun 1990; Niewoehner & Leath 1998; Pietrusińska & Czembor 2015). Both horizontal and vertical resistance are characterized by different chronological response to emerging new races of one or more pathogens. Breeding wheat for disease resistance mainly relies

https://doi.org/10.17221/99/2019-PPS

on race-specific strategies determining monogenic resistance consistent with the gene-for-gene theory coined by Flor, which states that the products of virulence genes of the pathogen interact directly or indirectly with products of major resistance genes of the host plant (Flor 1955; Tyrka & Chełkowski 2003; Górny 2004; Chełkowski & Koczyka 2005; Pietrusińska & Czembor 2015). This strategy leads to complete but usually short-term disease resistance, because new races with corresponding virulence genes evolve in the pathogen population (Pietrusińska & Czembor 2017). Therefore, pyramidization of different resistance genes is important to create cultivars with durable resistance to fungal pathogens. Reportedly, this strategy is more desirable when breeding for disease resistance. Effectively combined genes may complement each other, while resistance attributed to a major resistance gene may prove to be ineffective because of new races with corresponding virulence genes evolving in a population of the pathogen (Pietrusińska & Czembor 2017). Effective gene pyramids are particularly valuable in resistance genetics because they create cultivars with long-lasting resistance to one or more pathogens (Rubiales & Niks 2000; Ribeiro do Vale et al. 2001; Pietrusińska & Czembor 2017).

Wheat powdery mildew resistance genes are denoted *Pm* (Powdery mildew – *Pm* genes). It is difficult to conclude from literature reports how many *Pm* genes have been identified to date. According to McIntosh et al. (2004), at the early stage of the studies on the identification and characterization of resistance genes to powdery mildew of wheat there were 39 *Pm* genes known. Their number increased as the studies progressed. Some reports indicate that there are 41 *Pm* genes and 60 alleles (He et al. 2009; Hua et al. 2009; Li et al. 2009; Luo et al. 2009; Alam et al. 2011; Ma et al. 2011). Hao et al. (2015) reported 49 wheat powdery mildew resistance genes and 77 alleles (Hao et al. 2015). Liu et al. (2017) mentioned 55 genes (*Pm1–Pm55*) of wheat powdery mildew resistance (Liu et al. 2017). Ullah et al. (2018) reported 57 *Pm* genes and 31 alleles of wheat powdery mildew resistance genes (Ullah et al. 2018). Differences in the number of identified and characterized Pm genes may result from the fact that in the gene catalogue some *Pm* genes were in fact the alleles of other *Pm* genes, new resistance genes have been identified, or new *Pm* genes in subsequent studies turned out to be already known *Pm* genes. This refers to studies on the following wheat powdery mildew resistance genes: *Pm18* = *Pm1c*, *Pm22* = *Pm1e*, *Pm23* = *Pm4b* and *Pm4c*, *Pm31* = *Pm21* (Duan et al. 2001; Huang et al. 2004; Hao et al. 2008; Sun et al. 2018; Tan et al. 2018).

*Pm* genes in plant material are identified by mapping one or more genes at very specific *loci* or *locus* corresponding with resistance. This method allows for the very accurate detection of molecular marker(s) closely linked to a particular utility feature, and for high-density genetic mapping. It is commonly used for the detection of markers closely linked to the resistance to fungal pathogens, including *B. graminis.* High resolution mapping for the *Mlm2033* and *Mlm80* loci allowed for the determination of their position with respect to the *Pm1* gene. It was suggested that *Mlm2033* and *Mlm80* genes are probably the alleles of *Pm1a* (Yao et al. 2007). In addition, the gene with the provisional name *Pm5055* is linked to *MlIW170* and may be linked to the *Pm42* gene. However, the authors of the reported study indicated the need to continue research in order to verify these findings (Saidou et al. 2016). Genetic mapping studies by Sun et al. (2006) suggest that two resistance genes with the provisional names *PmY201* and *PmY212* are two new sources of wheat powdery mildew resistance and are not linked to the *Pm2* and *Pm-M53* genes (Sun et al. 2006). There have also been four other *Pm* resistance genes reported, with provisional symbols *MlRD30* (Singrün et al. 2004), *MlZec1* (Moher et al. 2005), *MlIW72* (Ji et al. 2008), and *PmU* (Qiu et al. 2005). The genetic analysis showed that a single recessive *PmQ* gene, is resistant to Bgt isolates currently found in China. The collective segregation analysis of RNA-Seq (BSR-Seq) was carried out on the collective lines where 57 SNP were detected on the 2BL chromosome. Based on further research, 485 microsatellite markers were designed and used to construct the genetic map. On the basis of the obtained results two markers flanking the *PmQ* gene were determined: Xicsq405 and WGGBH913. Probably the *PmQ* gene can be the allele of the *Pm63* gene (Li et al. 2019).

Resistance to *B. graminis* f. sp. *tritici* in wheat cultivars can be improved by introducing one or more effective resistance genes into the wheat genome. The most common way of enriching gene pools of wheat is convergence crossing along with the parallel selection of plant material using molecular markers MAS (marker-assisted selection) and phenotypic selection. Host-pathogen tests are also an important tool in studies on the identification of resistance genes.

https://doi.org/10.17221/99/2019-PPS

Phytopathological tests are used both for the detection and identification of *Pm* genes. The great advantage of phenotypic selection is that it can be carried out both at the seedling stage and at the adult plant stage. The combination of these selection methods is the simplest and most effective breeding strategy by means of which it is possible to transfer and identify many useful traits to progeny genotypes.

Wheat cultivars currently grown in Poland, and those for which preliminary/registration studies are pending in respect of resistance to *B. graminis* f. sp. *tritici* are rated as moderately susceptible or susceptible at the seedling stage, and less or more susceptible in field conditions, depending on weather conditions during the growing season. Therefore, studies aimed at the detection and effectiveness of new genes of resistance to powdery mildew of cereals and grasses are extremely important for the breeding process (Riar et al. 2012). In addition, the appropriate selection of effective sources of resistance to be used as components for crossing determines the success of the whole breeding process. This is especially important when new races evolve overcoming the resistance determined by *Pm* genes, both in single and multiple segments of resistance genes (Boyd et al. 2012). Therefore, all aspects of studies, including the identification of individual *Pm* genes, analysis of their susceptibility/resistance in relation to races of pathogens currently present in a given area, and pathogen monitoring, provide the most effective method of disease control.

In Poland studies on breeding for disease resistance aimed at the identification of *Pm* genes in different combinations are conducted on cultivars and breeding material. Breeding for disease resistance in Poland relies on wheat cultivars with resistance profiles *Pm*, *Pm* + *Pm* or *Pm* + *Pm* + *Pm* (Pietrusińska & Czembor 2017). Although the *Pm2* gene cannot be regarded as an effective source of resistance to powdery mildew of cereals and grasses, it can be used in combination with other *Pm* genes (Ma et al. 2011; Tomkowiak et al. 2017). Tomkowiak et al. (2017) used three molecular markers and identified the *Pm2* gene in the Polish cultivar Tonacja. Kowalczyk et al. (1998) identified the combination of two genes (*Pm2* + *Pm6*) in winter and spring wheat cultivars: Alba, Arda, Korweta, Oda, Olcha, Rada, Roma, Sakwa and Weneda (Kowalczyk et al. 1998). In addition, the same pyramid of resistance genes (*Pm2* + *Pm6*) was characterised for Polish wheat cultivars: Tonacja, Clever and Finezja (Kowalczyk et al. 2011). The combination of resistance genes *Pm2* + *Pm6* in these cultivars was inherited from the English cultivar Maris Huntsman, which was widely used in European breeding programmes of wheat as a source of resistance to powdery mildew of cereals and grasses (Zeller et al. 1993; Pietrusińska & Czembor 2015). Kowalczyk et al. (1998) also identified the gene combination *Pm3d* + *Pm4b* in Gracja and Mona cultivars, *Pm3d* + *Pm8* in Turnia cultivar, *Pm4b* + *Pm8* in Wilga cultivar, *Pm5* + *Pm8* in Aleta cultivar, *Pm1* + *Pm3d* + *Pm4b* in Hesja and Omega cultivars and *Pm2* + *Pm3d* + *Pm4b* in Polna cultivar (Kowalczyk et al. 1998). Polish winter wheat cultivars Bogatka and Nadobna were improved with the combination of two effective resistance genes, *Pm21* + *Pm34*. Homozygous lines obtained in experiments are used as an effective source of resistance to powdery mildew of cereals and grasses for wheat cultivars grown in Poland and at the same time maintain good utility traits (Pietrusińska & Czembor 2017). A detailed list of genes of resistance to powdery mildew of cereals and grasses identified in Polish wheat cultivars was presented by Pietrusińska and Czembor (2015).

## SUMMARY

This paper attempts to briefly describe the fungal disease caused by *B. graminis*. Powdery mildew of cereals and grasses occurs each year, in different intensity, causing year-to-year lower or higher yield losses. However, it should be remembered that mildew from cereals and grasses does not have to destroy crops. Current knowledge of the pathogen(s) in question guarantees good effectiveness in combating this disease. Chemical treatments can be used for this purpose, as well as the objectives of immunological breeding. Furthermore, in an era of global warming, true cereal and grass mealybugs can play a significant role in agricultural crops and should not be underestimated in any way. As a result of climate change, it is believed that true grain and grass mealybugs are becoming more aggressive and resistant to fungicides from year to year. Therefore, current knowledge is an extremely important element in the fight against this disease.

## REFERENCES

Alam M., Xue F., Wang C., Ji W. (2011): Powdery mildew resistance genes in wheat: identification and genetic analysis. Journal of Molecular Biology, 1: 20–39.

https://doi.org/10.17221/99/2019-PPS

An D., Zheng Q., Zhou Y., Ma P., Lv Z., Li L., Li B., Luo Q., Xu H., Xu Y. (2013): Molecular cytogenetic characterization of a new wheat-rye 4R chromosome translocation line resistance to powdery mildew. Chromsome Research, 21: 419–432.

Asad M., Bai B., Lan C., Yan J., Xia X., Zhang Y., He Z. (2013): QTL mapping for adult plant resistance to powdery mildew in Italian wheat cv. Strampelli. Journal of Integrative Agriculture, 5: 756–764.

Bengtsson T., Ahman I., Mannien O., Reitan L., Christerson T., Jensen J., Krusell L., Jahoor A., Orabi J. (2017): A novel QTL for powdery mildew resistance in Nordic spring barley (*Hordeum vulgare* L. ssp. *vulgare*) revealed by genome-wide association study. Frontiers in Plant Science, 8: 1–11.

Bennett F. (1984): Resistance to powdery mildew in wheat: a review of its use in agriculture and breeding programmes. Plant Pathology, 33: 279–300.

Bouguennec A., Trottet M., du Cheyron P., Lonnet P. (2014): Triticale powdery mildew: population characterization and wheat gene efficiency. Communications in Agricultural and Applied Biological Science, 79: 106–21.

Boyd L., Ridout C., O'Sullivan D., Leach J., Leung H. (2012): Plant–pathogen interactions: disease resistance in modern agriculture. Trends in Genetics, 29: 1–8.

Braun U., Cook R., Inman A., Shin H. (2002). The taxonomy of the powdery mildew fungi. In: Bélanger R., Bushnell W., Dik A., Carver T.L.W. (eds): The Powdery Mildews: A Comprehensive Treatise. St. Paul, APSPress: 13–15.

Bujak H., Jurkowski A. (2013): Estimation of winter rye (*Secale cereal* L.) susceptibility to infection by powdery mildew (*Blumeria graminis* f. sp. *secalis*). Acta Agrobotanica, 66: 49–54.

Cao X., Luo Y., Zhou Y., Duan X., Cheng D. (2013): Detection of powdery mildew in two winter wheat cultivars using canopy hyperspectral reflectance. Crop Protection, 45: 124–131.

Centrum Doradztwa Rolniczego w Brwinowie (2018): https://cdr.gov.pl/aktualnosci/57-cdr-informuje/2642-relacja-ze-szkolenia-rozpoznawanie-agrofagow-w-uprawach-rolniczych?highlight=WyJwcm9naSIsInN6a29kbGl3b1x1MDE1YmNpIiwicHJvZ2kgc3prb2RsaXdvXHUwMTViY2kiXQ== (accessed May 30, 2018).

Chełkowski J., Koczyka G. (2005): Genomika i Bioinformatyka Roślin. Rozprawy i Monografie. Poznań, IGR PAN: 139–157.

Conner R., Kuzyk A., Su H. (2003): Impact of powdery mildew on the yield of soft white spring wheat cultivars. Canadian Journal of Plant Science, 83: 725–728.

Cunfer B.M. (2002): Powdery mildew. Available at http://www.fao.org/3/y4011e0l.htm

Czembor H. (1981): Rasy fizjologiczne mączniaka jęczmienia (*Erysiphe graminis* DC ex Merat f. sp. *hordei*) występujące w Polsce w latach 1975–1979. Hodowla Roślin, Aklimatyzacja i Nasiennictwo, 25: 215–225.

Czembor H., Czembor J. (2005): Pathogenicity of barley powdery mildew (*Blumeria graminis* f. sp. *hordei*) in Poland in 2001. Biuletyn IHAR, 236: 183–192.

Daamen R. (1989): Assessment of the profile of powdery mildew and its damage function at low disease intensities in field experiments with winter wheat. Netherlands Journal of Plant Pathology, 95: 85–105.

Day P. (1974): Genetics of host-parasite interaction. San Francisco, W.H. Freeman and Company.

Dewey D. (1983): Historical and current taxonomic perspectives of *Agropyron, Elymus*, and related genera. Crop Science, 23: 637–642.

Dhirhi N., Mehta N., Singh. S (2017): Screening of powdery mildew tolerance in linseed (*Linum usitatisimum* L.). Journal of Plant Development Sciences, 9: 153–156.

Draz I., Esmail S., Abou-Zeid M., Essa T. (2019): Powdery mildew susceptibility of spring wheat cultivars as a major constraint on grain yield. Annals of Agricultural Sciences, 64: 39–45.

Duan X., Xiang Q., Zhou Y., Sheng B., Zhao Z. (2001): Allelic analysis of powdery mildew resistance genes in four Chinese landraces. Acta Phytopathologica Sinica, 31: 32–35.

Esmail S., Draz I. (2017): Fungal morphogenesis tracking of *Blumeria graminis* f. sp. *tritici* on leaf freed of epiculticular wax using scanning electron microscopy. International Journal of Microbiology and Biotechnology, 2: 181–188.

Everts K., Leath S., Finney P. (2001): Impact of powdery mildew and leaf rust on milling and baking quality of soft red winter wheat. Plant Disease, 85: 423–429.

Felczak K. (2016): Mączniak prawdziwy zbóż. Available at https://doradca-rolniczy.pl/maczniak-prawdziwy-zboz/

Fiedorow Z., Gołębniak B., Weber Z. (2004): Choroby Roślin Rolniczych. Poznań, AR.

Flor H. (1956): The complementary genetic systems in flax and flax rust. Advances in Genetics, 8: 29–54.

Gacek E. (1983): Analiza patogeniczności grzybów porażających jęczmienia. Biuletyn IHAR, 150: 11–15.

Gacek E. (1987): Distribution of barley powdery mildew resistance and virulence in Poland in 1984–1986. In: Wolfe W.S., Limpert E. (eds): Advances in Agricultural Biotechnology. Dordrecht, Martinus Nijhoff Publishers: 93–98.

Gacek E. (1990): Studia nad sposobami wykorzystania odporności genetycznej jęczmienia w zwalczaniu mączniaka prawdziwego (*Erysiphe graminis* DC f. sp. *hordei* Marchal). Hodowla Roślin i Nasiennictwo, 34: 3–49.

Gacek E., Biliński Z., Czembor H., Czembor J. (2004): Chorobotwórczość mączniaka prawdziwego jęczmienia (*Blumeria graminis* f. sp. *hordei*) w Polsce w latach 1993–1996. Biuletyn IHAR, 231: 365–376.

https://doi.org/10.17221/99/2019-PPS

Gacek E., Czembor H. (1983): Problem wykorzystania genetycznej odporności w hodowli i uprawie mieszanin zbóż ze szczególnym uwzględnieniem jęczmienia. Biuletyn IHAR, 151: 37–45.

Gacek E., Czembor H. (1984): Prawidłowe wykorzystanie genetycznej odporności jęczmienia na choroby. Zeszyty Problemowe Postępów Nauk Rolniczych, 305: 163–169.

Gacek E., Czembor H. (1988): Analiza ilościowa struktury populacji mączniaka jęczmienia (*Erysiphe graminis* f. sp. *hordei*). Biuletyn IHAR, 167: 13–19.

Gacek E., Czembor H. (1993): Evolution of barley powdery mildew population in Poland. Polnohospodarstwo, Bratislava: 39: 373–379.

Gacek E., Czembor H. (1995): Metody badań chorobotwórczości patogenów obligatoryjnych zbóż. Krajowe Sympozjum "Odporność Roślin Na Choroby, Szkodniki i Niesprzyjające Czynniki Środowiska". IHAR Radzików, 169–179.

Gepts P. (2000): A phylogenetic and genomic analysis of crop germplasm: A necessary condition for its rational conservation and utilization. In: Proc Stadler Genetics Symposium PGJ. New York, Plenum: 163–181.

Gepts P., Papa R. (2003): Possible effects of (trans)gene flow from crops on the genetic diversity from landraces and wild relatives. Environmental Biosafety Research, 2: 89–103.

Gołębniak B. (1993): Wybrane zagadnienia z biologii i epidemiologii mączniaka prawdziwego zbóż i traw (*Erysiphe graminis* DC). In: Roczniki AR w Poznaniu, Rozprawy Naukowe, Zeszyt. Poznań, Akademii Rolniczej w Poznaniu.

Górny A. (2004): Zarys Genetyki Zbóż. Tom 1. Jęczmień, pszenica i żyto. Poznań, Instytut Genetyki Roślin PAN: 181–327.

Götz M., Friedrich S., Boyle C. (1996): Development of cleistothecia and early ascospore release of *Erysiphe graminis* DC. f. sp. *tritici* in winter wheat in relation to host age and climatic conditions. Journal of Plant Diseases and Protection, 103: 134–141.

Gupta S., D'Antuono M., Bradley J., Li Ch, Loughman R. (2014): Identification and expression of adult plant resistance in barley to powdery mildew (*Blumeria graminis* f. sp. *hordei*) in Australia. Euphytica, 203: 595–605.

Hacquard S., Kracher B., Maekawa T., Vernaldi S., Schulze-Lefert P., Ver Loren van Themaat E. (2013): Mosaic genome structure of the barley powdery mildew pathogen and conservation of transcriptional programs in divergent hosts. Proceedings of the National Academy of Sciences, USA, 110(24): e2219-28. doi:10.1073/pnas.1306807110

Hao Y., Liu A., Wang Y., Feng D., Gao J., Li X., Liu S., Wang H. (2008): *Pm23*: a new allele of *Pm*4 located on chromosome 2AL in wheat. Theoretical and Applied Genetics, 117: 1205–1212.

Hao Y., Parks R., Cowger C., Chen Z., Wang Y., Bland D., Johnson J., Murphy J., Guedira M, Brown-Guedira G. (2015): Molecular characterization of a new powdery mildew resistance gene *Pm54* in soft red winter wheat. Theoretical and Applied Genetics, 128: 465–476.

Harlan J., de Wet J. (1971): Towards a rational classification of cultivated plants. Taxon, 20: 509–517.

He R., Chang Z., Yang Z., Yuan Z., Zhan H., Zhang X., Liu J. (2009): Inheritance and mapping of powdery mildew resistance gene *Pm43* introgressed from *Thinopyrum intermedium* into wheat. Theoretical and Applied Genetics, 118: 1173–1180.

Hermansen J., Torp U., Prahm L. (1978): Studies of transport of live spores of cereal mildew and rust fungi across the North Sea. Grana, 17: 41–46.

Hua W, Liu Z, Zhu J, Xie C, Yang T, Zhou Y, Duan X, Sun Q, Liu Z (2009): Identification and genetic mapping of *Pm42*, a new recessive wheat powdery mildew resistance gene derived from wild emmer (*Triticum turgidum* var. *dicoccoides*). Theoretical and Applied Genetics, 119: 223–230.

Huang X., Röder M. (2004): Molecular mapping of powdery mildew resistance genes in wheat: A review. Euphytica, 137: 203–223.

Instytut Ochrony Roślin (2008): Avalable at https://www.ior.poznan.pl/plik,263,stan-fitosanitarny-2007.pdf

Jańczak C., Pawlak A. (2006): Występowanie i szkodliwość mącznika prawdziwego (*Blumeria graminis*) w pszenicy ozimej w latach 2003–2005. Postępy w Ochronie Roślin, 46: 538–542.

Ji X., Xie C., Ni Z., Yang S., Nevo E., Fahima T., Liu Z., Sun Q. (2008): Identification and genetic mapping of a powdery mildew resistance gene in wild emmer (*Triticum dicoccoides*) accession IW72 from Israel. Euphytica, 159: 385–390.

Kirk P., Cannon P., David J., Stalpers J. (2001): Ainsworth & Bisby's Dictionary of the Fungi. : 9th edition. Wallingford. CABI Publishing

Kochman J., Węgorek W. (1997): Ochrona Roślin. Choroby infekcyjne, 4th Ed. Kraków, Plantpress: 445–447.

Kowalczyk K., Gruszecka D., Nowak M., Leśniowska-Nowak J. (2011): Resistance of *Triticale* hybrids with *Pm4b* and *Pm6* genes to powdery mildew. Acta Biologica Cracoviensia, Series Botanica, 53: 57–62.

Kowalczyk K., Hsam S., Zeller F. (1998): Identification of powdery mildew resistance genes in common wheat (*Triticum aestivum* L. em. Thell.). XI. Cultivars grown in Poland. Journal of Applied Genetics, 39: 225–236.

Lackermann K., Conley S., Gaska J., Martinka M., Esker P. (2011): Effect of location, cultivar, and diseases on grain yield of soft red winter wheat in Wisconsin. Plant Disease, 95: 1401–1406.

https://doi.org/10.17221/99/2019-PPS

Leath S., Bowen K. (1989): Effects of powdery mildew, triadimenol seedtreatment, and triadimefon foliar sprays on yield of winter wheat in North Carolina. Phytopathology, 79: 152–155.

Leath S., Heun M. (1990): Identification of powdery mildew resistance genes in cultivars of soft red winter wheat. Plant Disease, 74: 747–752.

Legler S., Caffi T., Rossi V. (2012): A nonlinear model for temperature-dependent development of *Erysiphe necator* chasmothecia on grapevine leaves. Plant Pathology, 61: 96–105.

Li B., Cao X., Chen L., Zhou Y., Duan X., Luo Y., Fitt B., Xu X., Song Y., Wang B., Cao S. (2013): Application of geographic information systems to identify the over summering regions of *Blumeria graminis* f. sp. *tritici* in China. Plant Disease, 97: 1168–1174.

Li G., Fang T., Zhang H., Xie Ch., Li H., Yang T., Nevo E., Fahima T., Sun Q., Liu Z. (2009): Molecular identification of a new powdery mildew resistance gene *Pm41* on chromosome 3BL derived from wild emmer (*Triticum turgidum* var. *dicoccoides*). Theoretical and Applied Genetics, 119: 531–539.

Li Y., Shi X., Hu J., Wu P., Qiu D., Qu Y., Xie J., Wu Q., Zhang H., Yang L., Liu H., Zhou Y., Liu Z., Li H. (2020): Identification of a recessive gene *PmQ* conferring resistance to powdery mildew in wheat landrace Qingxinmai using BSR-Seq Analysis. Plant Disease, 104(3). doi: 10.1094/PDIS-08-19-1745-RE

Liu N., Bai G., Lin M., Xu X., Zheng W. (2017): Genome-wide association analysis of powdery mildew resistance in U.S. winter wheat. Scientific Reports, 7: 1–11.

Liu Z., Sun Q., Ni Z., Yang T. (1999): Development of SCAR markers linked to the *Pm21* gene conferring resistance to powdery mildew in common wheat. Plant Breeding, 118: 215–219.

Luo P., Luo H., Chang Z., Zhang H., Zhang M., Ren Z. (2009): Characterization and chromosomal location of *Pm40* in common wheat: a new gene for resistance to powdery mildew derived from *Elytrigia intermedium*. Theoretical and Applied Genetics, 18: 1059–1064.

Ma H., Kong Z., Fu B., Li N., Zhang L., Jia H., Ma Z. (2011): Identification and mapping of a new powdery mildew resistance gene on chromosome 6D of common wheat. Theoretical and Applied Genetics, 123: 1099–1106.

Mains E., Dietz S. (1930): Physiologic forms of barley mildew, *Erysiphe graminis hordei* Marchal. Phytopathology, 20: 229–239.

Mandal M., Fu Y., Zhang S., Ji W. (2015): Proteomic analysis of the defense response of wheat to the powdery mildew fungus, *Blumeria graminis* f. sp. *tritici*. Protein Journal, 33: 513–524.

Marschal E. (1902): De la specialization du parasitisme chez *l'Eysiphe graminis*. Comptes Rendus Academie des Sciences, Paris, 135: 210–212.

McIntosh R., Devos K., Dubkovsky J., Rogers W. (2004): Catalogue of gene symbols for wheat: 2004 Supplement. Available at https://wheat.pw.usda.gov/ggpages/wgc/2004upd.html

Merchan V., Kranz J. (1986): Die Wirkung des Regens auf die Entwicklung des Weizenmehltaus (*Erysiphe graminis* DC. f. sp. *tritici* Marchal). Journal of Plant Disease and Protection, 93: 262–270.

Ministerstwo Rolnistwa i Rowvoju Wsi. Available at http://www.minrol.gov.pl/Informacje-branzowe/Produkcja-roslinna/Ochrona-roslin/Integrowana-ochrona-roslin/Metodyki-integrowanej-ochrony-roslin

Moher V., Zeller F., Wenzel G., Hsam S. (2005): Chromosomal location of genes for resistance to powdery mildew in common wheat (*Triticum aestivum* L. em Thell). 9. Gene *MlZec1* from *Triticum dicoccoides*-derived wheat line Zecoi-1. Euphytica, 142: 161–167.

Morgounov A., Tufan H., Sharma R., Akin B., Akin B., Bagci A., Braun H., Kaya Y., Keser M., Payne T., Sonder K., McIntosh R. (2012): Global incidence of wheat rusts and powdery mildew during 1969–2010 and durability of resistance of winter wheat variety Bezostaya 1. European Journal of Plant Pathology, 132: 323–340.

Mucha S. (2018): Integrowana ochrona roślin. MRiRW. Avalilable at https://www.gov.pl/web/rolnictwo/integrowana-ochrona-roslin (accessed April 5, 2018).

Niewoehner A.S., Leath S. (1998): Virulence of *Blumeria graminis* f. sp. *tritici* on winter wheat in the Eastern United States. Plant Disease, 82: 64–68.

Okoń S., Ociepa T. (2017): Virulence structure of the *Blumeria graminis* DC.f. sp. *avenae* populations occurring in Poland across 2010–2013. European Journal of Plant Pathology, 149: 711–718.

Oku T., Yamashita S., Doi Y., Nishihara N. (1985): Host range and forma specialis of cocksfoot powdery mildew (*Erysiphe graminis* DC) found in Japan. Annals of the Phytopathological Society of Japan, 51: 613–615.

Packa D., Kuraczyk A., Tworkowski J. (2018): Available at http://www.uwm.edu.pl/eurequa/pl/I.3.htm

Paillard S., Goldringer I., Enjalbert J., Trottet M., David J., de Vallavieille-Pope C., Brabant P. (2000): Evolution of resistance against powdery mildew in winter wheat populations conducted under dynamic management. II. Adult plant resistance. Theoretical and Applied Genetics, 101: 457–462.

Peng F., Song N., Shen H., Wu H., Dong H., Zhang J., Li Y., Peng H., Ni Z., Liu Z., Yang T., Li B., Xie C., Sun Q. (2014): Molecular mapping of a recessive powdery mildew resistance gene in spelt wheat cultivar Hubel. Molecular Breeding, 34: 491–500.

Piarulli L., Agata G., Giacomo M., Signorile M., Pasquini M., Blanco A., Simeone R. (2012): Molecular identification of a new powdery mildew resistance gene on chromosome

https://doi.org/10.17221/99/2019-PPS

2BS from *Triticum turgidum* ssp. *Dicoccum*. Plant Science, 196: 101–106.

Pietrusińska A., Czembor H. (2017): Pyramiding of resistance genes (*Pm21* + *Pm34*) of powdery mildew of cereale and grasses (*Blumeria graminis* f. sp. *tritici*). Progress in Plant Protection, 57: 41–46.

Pietrusińska A., Czembor J. (2015): Gene pyramiding – a tool commonly used in breeding programs. Biuletyn IHAR, 278: 3–16.

Pietrusińska A., Żurek M., Mańkowski D. (2019): The search for sources of biotic stress resistance in old varieties and landraces of wheat and triticale. Biuletyn IHAR, 287: 25–28.

Pietrusińska A., Żurek M., Piechota U., Słowacki P., Smolińska K. (2018): Searching for diseases resistance sources in old cultivars, landraces and wild relatives of cereals. A review. Agronomy Science, 73: 45–60.

Platforma Sygnalizacji Agrofagów (2018): Available at https://www.agrofagi.com.pl/ (accessed May 14, 2018).

Qiu Y., Zhou R., Kong X., Zhang S., Jia J. (2005): Microsatellite mapping of a *Triticum urartu* Tum. derived powdery mildew resistance gene transferred to common wheat (*Triticum aestivum* L.). Theoretical and Applied Genetics, 111: 1524–1531.

Riar A., Kaur S., Dhaliwal H., Singh K., Chhuneja P. (2012): Introgression of a leaf rust resistance gene from *Aegilops caudata* to bread wheat. Journal of Genetics, 91: 155–161.

Ribeiro do Vale F., Parlevliet J., Zambolim L. (2001): Concepts in plant disease resistance. Fitopatologia Brasileira, 26: 577–589.

Rubiales D., Niks R. (2000): Combination of mechanisms of resistance to rust fungi as a strategy to increase durability. Options Mediterraneenes Series A, 40: 333–339.

Saari E., Prescott J. (1975): Scale for appraising the foliar intensity of wheat diseases. Plant Disease Management Reports, 59: 377–380.

Saari E.,Wilcoxson R. (1974): Plant disease situation of high-yielding dwarf wheats in Asia and Africa. Annual Review of Phytopathology, 12: 49–68.

Saidou M., Wang Ch., Alam A., Chen Ch., Ji W. (2016): Genetic analysis of powdery mildew resistance gene using SSR markers in common wheat originated from wild emmer (*Triticum dicooccoides* Thell). Turkish Journal of Field Crops, 21: 10–15.

Scott P., Johnson R., Wolfe M., Lowe H., Bennett F. (1980): Host-specificity in cereal parasites in relation to their control. Applied Biology, 5: 349–393.

Shi W., Gong S., Zeng F., Xue M., Yang L., Yu D. (2016): Sexual reproduction and detection of mating-type of *Blumeria graminis* f. sp. *tritici* populations. Acta Phytopathologica Sinica, 46: 645–652.

Singh D., Sharma A., Sharma I., Singh D., Rana S., Upadhyay S., Singh K., Kumar J., Srivastava K., Bhardwaj S., Prashar M., Gangwar O., Jain S., Pant S., Brahma R., Kumar J., Singh K., Devlash R., Prasad A., Dodan D. (2016): Identification of resistance sources against powdery mildew (*Blumeria graminis*) of wheat. Indian Phytopathology, 69: 413–415.

Singrün Ch., Hsam S., Zeller F., Wenzel G. (2004): Localization of a novel recessive powdery mildew resistance gene from common wheat line RD30 in the terminal region of chromosome 7AL. Theoretical and Applied Genetics, 109: 210–214.

Sun H., Hu J., Song W., Qiu D., Cui L., Wu P., Zhang H., Liu H., Yang L., Qu Y., Li T., Li T., Cheng W., Zhou Y., Liu Z., Li J., Li H. (2018): *Pm61*: a recessive gene for resistance to powdery mildew in wheat landrace Xuxusanyuehuang identified by comparative genomics analysis. Theoretical and Applied Genetics, 131: 2085–2097.

Sun X., Liu D., Zhang H., Huo N., Zhou R., Jia J. (2006): Identification and mapping of two new genes conferring resistance to powdery mildew from *Aegilops tauschii* (Coss.) Schmal. Journal of Integrative Plant Biology, 48: 1204–1209.

Tan Ch., Li G., Cowger Ch., Carver B., Xu X. (2018): Characterization of *Pm59*, a novel powdery mildew resistance gene in Afghanistan wheat landrace PI 181356. Theoretical and Applied Genetics, 131: 1145–1152.

Tomkowiak A., Kurasiak-Popowska D., Grynia J., Nawracała J., Mikołajczyk S., Weigt D., Niemann J., Kiel A. (2017): Evaluation of the usefulness of molecular markers *Xgwm205*, *Xcfd81*, *Whs350* for the identification of resistance gene *Pm2* to powdery mildew (*Blumeria graminis* f. sp. *tritici*) in wheat cultivars of different origins. Progress in Plant Protection, 57: 146–152.

Tratwal A., Bocianowski J. (2014): *Blumeria graminis* f. sp. *hordei* virulence frequency and the powdery mildew incidence on spring barley in the Wielkopolska province. Journal of Plant Protection Research, 54: 28–35.

Tratwal A., Jakubowska M. (2004): Ocena przydatności systemów wspomagania decyzji o ochronie pszenicy ozimej przed mączniakiem prawdziwym na terenie Wielkopolski. Postępy w Ochronie Roślin, 44: 1169–1172.

Troch V., Audenaert K., Bekaert B., Höfte M., Haesaert G. (2012): Phylogeography and virulence structure of the powdery mildew population on its 'new' host triticale. BMC Evolutionary Biology, 12: 76. doi:10.1186/1471-2148-12-76

Troch V., Audenaert K., Wyand R., Haesaert G., Höfte M., Brown J. (2014): Formae speciales of cereal powdery mildew: close or distant relatives? Molecular Plant Pathology, 15: 304–314.

Tyrka M., Chełkowski J. (2003): Enhancing the resistance of triticale by using genes from wheat and rye. Journal of Applied Genetics, 45: 283–295.

Ullah K., Li N., Shen T., Wang P., Tang W., Ma S., Zhang Z., Jia H., Kong Z., Ma Z. (2018): Fine mapping of powdery mildew resistance gene *Pm4e* in bread wheat (*Triticum aestivum* L.). Planta, 248: 1319–1328.

https://doi.org/10.17221/99/2019-PPS

Walczak F., Gałęzewski M., Jakubowska M., Skorupska A., Tratwal A., Wojtowicz A., Złotowski J. (2009): Zespół Zakładu Metod Prognozowania i Rejestracji Agrofagów oraz Zakład Badania Gryzoni Polnych IOR w Poznaniu. Available at http://www.ior.poznan.pl/aktualizacja/data/pliki/263_Stan_fitosanitarny_2007.pdf

Walker A., Bouguennec A., Confais J., Morgant G., Leroux P. (2011): Evidence of host-range expansion from new powdery mildew (*Blumeria graminis*) infections of triticale (× *Triticosecale*) in France. The Plant Pathology Journal, 60: 207–220.

Wang Z., Li H., Zhang D., Guo L., Chen J., Chen Y., Wu Q., Xie J., Zhang Y., Sun Q., Dvorak J., Luo M., Liu Z. (2015): Genetic and physical mapping of powdery mildew resistance gene *MlHLT* in Chinese wheat landrace Hulutou. Theoretical and Applied Genetics, 128: 365–373.

Wicker T., Oberhaensli S., Parlange F., Buchmann J., Shatalina M., Roffler S., Ben-David R., Doležel J., Šimkova H., Schulze-Lefert P., Spanu P., Bruggmann R., Amselem J., Quesneville H., Ver Loren van Themaat E., Paape T., Shimizu K., Keller B. (2013): The wheat powdery mildew genome shows the unique evolution of an obligate biotroph. Nature Genetics, 45: 1092–1096.

Wiese M. (1987): Compendium of Wheat Diseases. 2nd Ed., St. Paul, APS Press.

Wójtowicz A., Wójtowicz M., Ratajkiewicz H., Pasternak M. (2017): Prognoza zmian czasu inkubacji sprawcy rdzy brunatnej pszenicy w reakcji na przewidywane ocieplenie klimatu. Fragmenta Agronomica, 34: 197–207.

Wolfe M., Schwarzbach E. (1975): The use of virulence analysis in cereal mildews. Phytopathologische Zeit, 82: 297–307.

Yang L., Zhang X., Zhang X., Wang J., Luo M., Yang M., Wang H., Xiang L., Zeng F., Yu D., Fu D., Rosewarne G. (2017): Identification and evaluation of resistance to powdery mildew and yellow rust in a wheat mapping population. PLoS ONE 12(5): e0177905. doi:10.1371/journal.pone.0177905

Yao G., Zhang J., Yang L., Xu H., Jiang Y., Xiong L., Zhang C., Zhang Z., Ma Z., Sorrells M. (2007): Genetic mapping of two powdery mildew resistance genes in einkorn (*Triticum monococcum* L.) accessions. Theoretical and Applied Genetics, 114: 351–358.

Zadoks J. (1967): International dispersal of funöi. Netherlands Journal of Plant Pathology, 73: 61–80.

Zeller F., Lutz J., Reimlein E., Limpert E., Koening J. (1993): Identification of powdery mildew resistance genes in common wheat (*Triticum aestivum* L.) II. French cultivars. Agronomie, 13: 201–207.

Received: September 13, 2019
Accepted: April 2, 2020
Published Online: June 11, 2020