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# Wild Wheats (*Triticum* spp.) and Relatives in Wheat Rust Diseases (*Puccinia* spp.) from a Wheat Breeder's Perspective: A General Evaluation

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## Abstract

According to the UN prediction, the human population will be more than 12 billion by the year 2050. This situation will lead to a shortage of agricultural product. Nearly all agricultural products are easily damaged by pathogens to the varying degree of disease severity if the crops are not protected from the pathogenic attack. Especially, the durability of resistance is a highly variable genetic trait and its genetical structure is still not understood completely. Wheat is a leading crop among the major food commodities of the world. In order to be more productive and to be less affected by rust diseases of wheat (*Triticum* spp.), the transfer of resistance genes "*wild germplasm(s)*" to the cultivated high yielding varieties is a viable approach. This review covers wild wheat and their relatives' and new rust resistance genes that can be used in terms of plant breeding in wheat improvement programs. With the aid of suit breeding methods, the use of "*wild germplasm diversity*" inbreeding programs opened up new horizon(s) and possibilities about the wild relatives, the possibility of their use, and the information of genetic background for the "*durable resistance*". In parallel with this, application of relevant biological approaches such as classical and modern wheat breeding programs, biotechnology, genetic engineering, molecular markers will always keep alive inevitable importance of the resistance of wild relative. © 2020 Friends Science Publishers

Keywords: Rust resistance genes; Rust diseases; Wild wheats and relatives; Wheat breeders; Diversity

## Introduction

According to Maxted and Kell (2009), there are approximately 0.4 million plants on earth. From them, only Triticeae (Pooideae, Poaceae) tribe consists of 350 annual and perennial species from 30 genera (Pradheep et al. 2019). So, quite likely the most important and strategic crop plant is wheat (Triticum spp.), which has 23 species (Anonymous 2018a). In addition, Zencirci et al. (2018) reported that the area of Eastern Anatolia (Asia Minor) and adjacent regions of Iran, Syria and Palestine and the southern Caucasus are the centers of diversity/fertile crescent, and origin for diploid, tetraploid and hexaploid wheats (Fig. 1; Anonymous 2019a). These regions were first called as "fertile crescent" by an American Orientalist, Mr. James Nery Breasted (Anonymous 2019a). On the other hand, these places are known as the bed of Western and NearEast/NearEastern/Middle or East/NearEastern/Asia Minor civilizations and agricultural activities (such as animal and plant breeding and growing) have been first started in these regions.

During the wheat cultivation and domestication processes, it has been evaluated, that it was especially naturally hybridized at the various ploidy levels with many close relatives, and it has reached its durum and common forms (Fig. 2, 3).

Wheat is the main crop plant of at least 35-40% population of the world and is expressed as 100 kg per person (Anonymous 2019b), 1.5 billion hectares of land in the world, 749 460 077 tonnes are produced and yields 34 050 kg/ha (Anonymous 2018b). It is a very nutritious crop plant and constitutes more than 45% of the energy source (as kcal capita<sup>-1</sup> day<sup>-1</sup>) and provides over 40% for the dietary protein (as g capita<sup>-1</sup> day<sup>-1</sup>) (Zhong et al. 2018). At the same time, it is obvious that (especially from the past few decades up to now) there have been many successful attempts for transferring the agronomically desired (for example wheat rust diseases genes, drought tolerance genes, etc.) traits from wild relatives (for example Aegilops tauschii Coss.) to cultivar(s)/variety(ies) (Dempewolf et al. 2017). Wild plants are important and crucial sources for the biotic and abiotic stress tolerance traits such as rust diseases, drought, light, heavy metal accumulation, salinity, etc. (Nataraj et al. 2018). In other respects, generally, agricultural activities are prone to the threat/menace/negative effect(s) of relevant factors. Wheat cultivars are also under the threat of rust diseases. It is interesting that wheat rust diseases cause the highest losses under the ideal conditions for high yield. Under these

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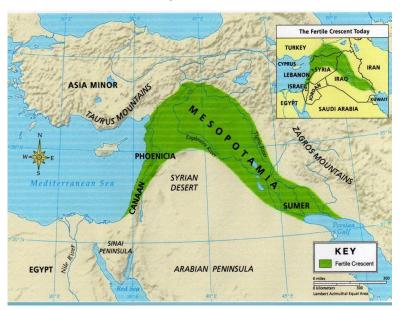


Fig. 1: Geographical location of the wheat as known Fertile Crescent (Anonymous, 2019b)

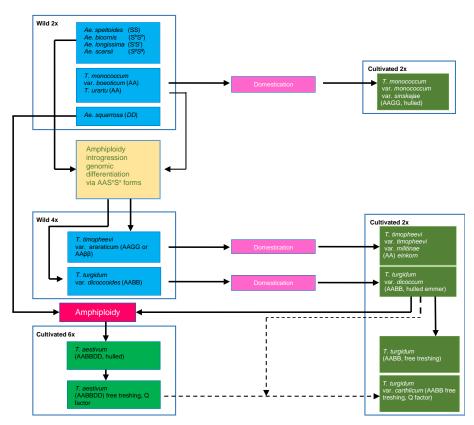


Fig. 2: Ploidy levels and species relationships during the wheat domestication (Jaradat 2012; Wang et al. 2018)

conditions, no producer or farmer is concerned with this yield loss. On the other hand, the loss of resistance of the cultivars/varieties by changing the race due to mutation or some reasons is another important threat. In this respect, the

solar radiations are the most effective mutagenic factors.

On the other hand; the leaf/brown, the stem/black and the stripe/yellow rusts are the most destructive biotic stresses and they cause major yield losses in wheat (Khan *et* 

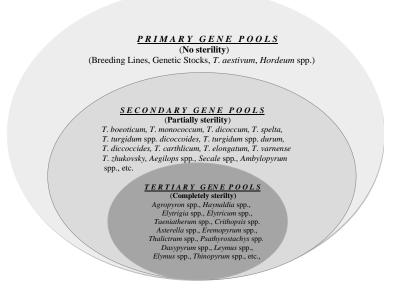


Fig. 3: Wild wheat (Triticum spp.) and relatives (Feldman and Sears 1981; Sharma and Gill 1983; Rasheed et al. 2018)

al. 2013; Muhammad et al. 2018). According to research findings, they are the most significant and devastating disease, especially for some countries of the world such as Kenya, Ethiopia, Sudan, Eritrea, Yemen, Iran, Mozambique, Zimbabwe, Tanzania, and South Africa for Black Rust (and developing other countries (Anonymous 2019c). Again, according to statistics, only 13% of global crop yields are lost from the rust diseases annually (Nelson et al. 2018), if susceptible wheat variety(ies)/cultivar(s) used, crop losses can reach up to 50% (Aktar-Uz-Zaman et al. 2017), These (crop) losses range is 0.5-90% in Turkey, 40% in the USA, 90% in the UK and 50% in the Germany and nearly 70% in the world (Özbaş 1967; Özgen and Kınacı 1985; Spitters et al. 1990; Dimov et al. 1993; Le Maitre and Botes 2013; Anonymous 2018c; Kolmer et al. 2018). Due to the rust diseases in wheat, production loses can be as high as 10-100% for black or stripe rust (Puccinia graminis), 10-70% for brown or leaf rust (P. triticina) and 20-100% for yellow or stripe rust (P. striiformis) (Riaz et al. 2016; Aktar-Uz-Zaman et al. 2017). In this paper, examples of place and importance of the wild wheats and relatives have been mentioned in order to give resistance to dry matter losses into wheat. In addition, we have tried to reach a conclusion with some hybridization barriers and (possible) solutions within them from a general perspective.

Plants are considered as *healthy* or *normal* when they carry out their metabolic function(s) without outside constraint(s); and, when plant is attacked by a pathogen, they experience any disruption or unusually different reactions in their vital functions such as cell division, differentiation, photosynthesis, respiration, etc. (Ulukan 1998). When these processes are partially expressed, deteriorate to a high degree easily they can damage one or more of their vital functions. On the other hand, the host-

pathogen relationship, humidity, prevailing temperature, the time or season of the exogenous applications, etc. plant genotype, plant pathogens and the selection of the appropriate parent(s) is/are essential for the wheat breeding program, which should be learned and practiced. Due to co-evolution of plant and pathogen(s) (Nelson *et al.* 2018), cultivated plants are easily damaged if they are not protected and quite often this damage can be reached to an extreme level(s). A major outbreak *rust epidemy*" in 2017 can be given as a major example for this topic in the world.

The mechanism of heredity, cellular and ploidy level in disease resistance are yet the subject of research. But, if it is taken as a whole, the disease resistance is determined by the interactions with related climatic factors such as humidity, temperature, etc. Indeed, if temperature, humidity, and light factors are favorable, the "epidemic(s)" appear(s) at various scales, leading to loss of quality and quantity of the product, which is undesirable, and this menace sometimes reaches significant levels. The first attempt was made by Gregor Mendel on wheat with the aim of "disease resistance" (Biffen 1905; Bartos et al. 2002). So, the resistant varieties can increase the dry matter yield to an average of 1.2-2.0 t/ha if cultivation techniques are appropriately used (Savchenko 2017). If cereals [namely, wheat (Triticum spp.), barley (Hordeum spp.), oats (Avena spp.), rye (Secale spp.) and Triticale) cannot grow in one place, no other cultivated plant can grow there. In addition, resistance to the disease is classified into vertical (qualitative, specific race resistance, mono-digenic and major gene) and horizontal (quantitative, non-specific race resistance, oligo-polygenic, minor gene) types (Nelson et al. 2018). Similarly, related phenotypic segregation rates were determined in the F<sub>2</sub> generation as 1:3, 3:7, 63: 1, 37:27, 57: 7, etc. (Vavilov 1954; Allard 1956; Knott 1990; Roelfs et al. 1992).

#### **Historical perspective**

While the history of wheat rust diseases goes back to 8,000 years ago before the domestication of crops (McIntosh 2009) or to Younger Dryas period some twelve millennia before present (Brennan 2010). Archeological record has revealed the presence of the spores of P. graminis some 1300 BC (Kislev 1982). The severity and frequency of outbreaks in wheat production vary depending on the pathogenicity of the causative agent, environmental conditions, ecology, host-pathogen relationships and their interactions. Severe epidemics of stem rust occurred in Europe in 1932 and 1951 accounting for yield losses of 5-20% in Eastern and Central Eurpoe and 9-33% in Scandanevia (Zadoks 1963). Stem rust was once thought to be the most devastating disease but now with the development of resistant cultivars, it is not now much threatening. However, its outbreak may occur when the disease resistant lines are rendered ineffective (Schumann and Leonard 2000; Rehman et al. 2013).

Nowadays, all three wheat rusts continue to be an important limiting problem in wheat production (Bashir *et al.* 2019). Especially black or stem rust disease is one of the most dangerous (Ellis *et al.* 2014). Yellow and brown rust is not as black rust, because they appear in conditions that provide higher yields and are not cared much by the farmers/producer, and it is preferred to grow/cultivated susceptible variety(ies)/cultivar(s) and this false agronomic practice is very common especially in developing countries. Similarly, interspecific cross(es) between wild wheats and their relatives with commercial variety(ies)/cultivar(s) could not be realized to the desired level due to a lot of resistance genes and (negative) linkage between the chromosomes pairs and agronomic traits.

#### Genetic source of disease resistance

Basic and important gene source is the "wild wheats (Triticum spp.) and its relatives". They are unique treasures because they carry the genes of resistance to biotic and abiotic stress factor(s), which are extremely important for plant production. In addition, wheat cultivation, which is one of the most strategic plants in the world, has gained a "sustainability" trait, with gene transfer from wild wheat relatives to cultivars/varieties. Wheat breeders have achieved successful results, especially in Aegilops spp. using wild wheat relatives (Fig. 3), which they classify as primary, secondary and tertiary based on their hybridization ability, polyploidy level and botanical affinities. Today, as mentioned before, a number of rust resistance genes have been introduced into a large number of wheat cultivars/varieties. Up to now, rust-resistant cultivars/varieties have been commercialized since both wild wheat (Triticum spp.) and goatgrass (Aegilops spp.) species have been hybridized (Table 1 and 2). The stem, stripe and leaf rusts are among the most important wheat rust diseases worldwide. In this context, as reported by Smitt (2013), more than 180 resistance genes for the wheat (state as (57) for stem rust genes, (state as 71) for leaf rust genes and (state as 54) for yellow rust genes) have been identified in wheat up to now. Nearly all of them were transferred into wheat cultivars/varieties by hybridizing the wild relatives. One of the best examples of this hybridization is given by McIntosh et al. (2007). They reported that wheat cultivar(s)/variety(ies) carrying the [T1BL.1RS] translocation arm of the rye (Secale cereale L.) chromosome was released after the mixed wheat breeding process, which comprises hybridization, mutation, and backcrosses (Fig. 3). On the other hand, Harlan and de Wet (1971) defined the Aegilops spp., as a secondary gene pool for wheat breeding. In addition, studies have shown that Ae. geniculata has resistance genes against powdery mildew disease with all three (leaf, stripe and stem rust diseases) rusts (Ohta 2017). Accordingly, primary and secondary gene sources of the Triticum spp., Aegilops spp., Amblyopyrum spp., and Dasypyrum spp., etc. are presented in Fig. 3. In this context, Turkey has wild wheat and its relatives as primary and secondary gene sources that contain wild Triticum spp., Aegilops spp., Amblyopyrum spp., Dasypyrum spp., etc. and cultivated relatives including barley (Hordeum spp.), rye (Secale spp.) and oats (Avena spp. and relatives (Fig. 1; Kün 1979; Özgen et al. 1987). They can be examined as i) modern cultivar(s)/variety(ies); ii) old cultivar(s)/variety (ies) iii) landraces, wild relatives of crops and finally iv) cytogenetic stocks and breeding lines (Zair et al. 2018).

Rust causal agents belong to the genus of *Puccinia*, phylum *Basidiomycota*, and comprise  $\approx$  7000 species. They are obligate biotrophic pathogens (Prell and Day 2001) and require a specific host to complete their life cycle. A translocation from *Thinopyrum ponticum* (syn. *Agropyron elongatum*) species for the leaf rust resistance gene *Lr19* was developed in the USA. Afterward, it was transferred into the Swedish cultivar of "Sunnan". Then, this study was carried out in Germany. Likewise, the two substitution lines [(1B/1R) and (T1BL/IRS)] were also developed there.

Rust resistance genes are of two types: a) pathotype specific resistance genes and pathotype non-specific resistance genes. Of these, pathotype non-specific genes only confer partial resistance to the Puccinia fungi but are effective against a broad range of pathotypes (Lagudah 2011). Furthermore, the rust resistance genes can be classified as either at the seedling stage and/or adult plant (APR) stage (or field resistance). According to observations, it is more commonly associated with pathotype non-specific resistance genes (Chen et al. 2013; Ellis et al. 2014). In addition, the linked rust resistance genes Lr26, Yr9, Sr31 and Pm8 are reported from rye (Secale cereale L.) plants (Bartos and Bares 1971). Other translocation lines are T. ventricosum (Ae. ventricosa), the line VPM1 (from a cross of Ae. ventricosa × T. persicum.), etc. According to an estimate, more than 640,000 accessions of Triticum spp.,

**Table 1:** Parents, species of allied genera crossed with wild wheats and relatives; *Aegilops (Ae.), Secale (S.), Agropyron (A.), Haynaldia (Ha.), Hordeum (H.), Elymus (E.), Elytrigia (Elyt.), Lophopyrum (Lo.), Leymus (Le.) and Psathyrostachys (Psa.) with their reciprocals/(as Trigeneric) (Sharma and Gill 1983; Haile <i>et al.* 2013; Edae *et al.* 2016; Curwen-MsAdams *et al.* 2017; Pradheep *et al.* 2019)

| Parent crossed           | Species of allied genera   |
|--------------------------|--|
| Diploid wheat:           | Ae. bicornis, Ae. caudata, Ae. columnaris, Ae. comosa, Ae. cylindrica,   |
| Triticum monococcum      | Ae. longissima, Ae. mutica, Ae. ovata, Ae. speltoides, Ae. squarrosa,  |
| Diploid wheat:           | Ae. triaristata, Ae. triuncialis, Ae. tripsaccoides, Ae. umbellulata,  |
|                          | Ae. uniaristata, Ae. variabilis, Ae. ventricosa  |
|                          | S. cereale   |
|                          | A. intermedium, A. elongatum   |
|                          | Ha, villosa  |
|                          | H. vulgare   |
| Fetraploid wheat:        | Ae. bicornis, Ae. biuncialis, Ae. caudata, Ae. columnaris, Ae. comosa, Ae. crossa, Ae. cylindrica, Ae. dichasians, Ae. heldreichi.                           |
| r. turgidum              | Ae. kotschvi, Ae. longissima, Ae. mutica, Ae. ovata, Ae. sharonensis, Ae. speltoides, Ae. squarrosa, Ae. triaristata, Ae                                     |
| ncludes                  | tripsaccoides, Ae. triuncialis, Ae. umbellulata, Ae. uniaristata, Ae. variabilis, Ae. ventricosa   |
| lurum, carthlicum,       | ·· T   |
| licoccum and dicoccoides |  |
| uncoccum and incoccontes | S. cereale, S. montanum, S. vavilovii, S. ancestrale, S. africanum   |
|                          | A. intermedium, A. elongatum, A. junceum, A. repens, A. campestre,   |
|                          | A. distichum, A. dasystachyum, A. distichum, A. obtusiusculum  |
|                          | Ha. villosa, Ha. hordeacea   |
|                          | H. vulgare, H. chilense, H. brevisubulatum   |
|                          |  |
| Tetraploid wheat:        | E. giganteus, E. arenarius<br>Ae. bicornis, Ae. caudata, Ae. comosa, Ae. cylindrica, Ae. dichasians, Ae. kotschyi, Ae. longissima, Ae. mutica, Ae. ovata, Ae |
| 1                        |  |
| T. timopheevi            | speltoides, Ae. squarrosa, Ae. triuncialis, Ae. umbellulata, Ae. uniaristata, Ae. ventricosa   |
|                          | S. cereale, S. vavilovii, S. africanum   |
|                          | A. cristatum, A. intermedium, A. elongatum, A. junceum, A. repens,   |
|                          | A. campestre   |
|                          | Ha. villosa  |
| Hexaploid wheat:         | H. vulgare, H. bogdanii, H. vulgare spp. distichon, H. spontaneum,   |
| T. aestivum              | H. pusillum, H. chilense, H. vulgare var. distichum  |
|                          | Ae. bicornis, Ae. biuncialis, Ae. caudata, Ae. columnaris, Ae. comosa,   |
|                          | Ae. crassa, Ae. cylindrica, Ae. dichasians, Ae. juvenalis, Ae. kotschyi,   |
|                          | Ae. longissima, Ae. mutica, Ae. ovata, Ae. sharonensis, Ae. speltoides,  |
|                          | Ae. squarrosa, Ae. triaristata, Ae. tripsaccoides, Ae. triuncialis,  |
|                          | Ae. uniaristata, Ae. umbellulata, Ae. variabilis, Ae. ventricosa   |
|                          | S.cereale, S.montanum, S.vavilovii, S.ancestrale, S.africanum, Triticosecale, Secale-Agropyron, Secale-Aegilops, Secale-Hordeum                              |
|                          | Secale-Thinopyrum, Dasypyrum-Psathyrostachys, Leymus-Thinopyrum,   |
|                          | Thinopyrum-Lophopyrum, Hordeum-Agropyron, Secale-Leymus,   |
|                          | Thinopyrum-Tritipyrum aaseae,  |
|                          | Tritipyrum aaseae, Thinopyrum intermedium  |
|                          | A. intermedium, A. elongatum, A. junceum, A. distichum, A. ciliare,  |
|                          | A. smithii, A. trachycaulum, A. scirpeum, A. yezoense, A. podperae,  |
|                          | A. caespitosum   |
|                          | E. giganteus, E. pontica, E. elongatus,  |
|                          | la villosa   |
|                          | Lo. ponticum   |
|                          | Le, mollis   |
|                          | Elyi. elongata   |
|                          | Psa. huashanica  |

Aegilops spp. and  $\times$  Triticosecale Wittmack (Triticale) are kept as *ex situ* or *in situ* collections around the world (Skovmand *et al.* 2002).

#### Wheat rust disease pathogens

Responsible pathogens from the wheat rust diseases can be generally placed into three classes as follows:

**Black or stem rust** (*Puccinia graminis* **f. sp.** *tritici* **Pers.**): *P. graminis* **f.** sp. *tritici* or black/stem rust' breeding activities were started first time in North America due to the wide epidemics in wheat fields. In Europe, stem rust on wheat lost its importance in the last decades; this was probably due to successful resistance breeding programs in the countries of South-Eastern Europe. On the contrary, due to the successful wheat disease breeding programs, it was not necessary to carry out over-breeding studies on black rust disease in the European continent.

Yellow or stripe rust (*P. striiformis* var. striiformis Westend.): Yellow rust caused by *P. striiformis* var. striiformis Westend., especially in the Western part of the European continent's wheat fields are threatened by Yrl 7 and its new variants (Meinel 1997; Chen *et al.* 2014). This disease has the potential to cause yield loss of up to 100%. However, if it is effective in the fields where yellow rust disease-resistant wheat cultivars/varieties are sown, at early stage, its losses are generally between 10–70%; but this menace is mostly dependent on weather conditions **Table 2:** Transferred some APR and seedling rust resistance genes from the relatives to wild wheats and relatives (Modified from Sharma and Gill 1983; Chelkowski and Stepien 2001; Bartos *et al.* 2002; Bulos *et al.* 2006; Purnhauser *et al.* 2011; Anonymous 2018e; Zhang *et al.* 2019)

| Gene(s)  | Parent/Donor                                      |
|--|---|
| Sr9gj/Yr7, Sr2, Sr13, Sr22, Sr31, Sr35, Sr39, Sr40   | T. aestivum/T. durum                              |
| Sr2, Sr9d, Sr9e, Sr12, Sr13, Sr14, Sr22, ST464   | T. dicoccum                                       |
| Lr19, Lr24, Lr29, Sr24, Sr25, Sr26, Sr27, Sr31   | (syn. Lophopyron elongatum) Ae. elongatum         |
| Lr9, LrU1, LrU2, YrU1, Sr51, Sr53, SrTA1662, SrTA101871, SrTA10187                               | Ae. umbellulata                                   |
| Sr5, Sr27, Sr31, Sr1RS <sup>Amigo</sup> , Sr50, Lr26, Yr9, Yr10, Pm7                             | Secale cereale and wheat introgression line       |
| Sr31, Sr36, Sr50   | Wheat-Rye (1BL.1RS) translocation line            |
| Lr26   | Wheat-Rye (T1BL/1RS) translocation line           |
| Sr4, Sr32, Sr39, Lr28, Lr35, Lr35/Sr39, Lr36, Lr47, Lr51, Lr66                                   | Ae. Speltoides                                    |
| Lr35   | Ae. speltoides/T. monococcum                      |
| Lr9, Lr28, Lr35, Lr36, Lr47, Lr51, Lr66, Sr4, Sr32, Sr39   | Ae. speltoides/T. aestivum                        |
| Sr24, Sr25, Sr26, Sr43, Sr44   | Wheat-Thinopyrum intermedium introgression line   |
| Sr52   | Wheat-Dasypyrum villosum introgression line       |
| Sr2, Sr12, Sr13, Sr21, Sr22, Sr33, Sr35, Sr49, Sr60, SrTm5, Lr53, Lr63, Sr27, Sr36, Sr38, SrSatu | T. monococcum                                     |
| Sr36, Sr37, Sr40, SrTt3, Lr18, Lr50, LrTt1, LrTt2, LrG1  | Triticale and introgression lines with wheat      |
| Lr30   | T. timopheevi                                     |
| Lr1, Lr2, Lr2a-c, Lr10, Lr12, Lr13, Lr15, Lr21, Lr22a, Lr24, Lr32, Lr34/Yr18/Bdv1,               | Ae. tauschii                                      |
| Lr34/Yr18/Pm38, Lr37, Lr37/Sr38, Lr39, Lr39/Lr41, Lr40, Lr41, Lr42, Lr43, Lr67, Lr68, LrA, LrB,  | Ae. ventricosa                                    |
| LrC, LrD, Yr28, Yr29, ALrA, ALrB, ALrC, AlrD, ALrE, ALrF, ALrG, ALrH, ALrI, ALrJ, ALrK, ALrL     | ,   |
| Sr5, Sr6, Sr18, Sr29, Sr30, SrSQ, Sr41, Sr42, Sr45, Yr1, Yr41, Yr46 Sr33, Lr37, Lr37/Sr38, Sr38  |   |
| Lr54/Yr37  | Ae. kotschyi                                      |
| Sr45   | Ae. tauscii                                       |
| Lr56/Yr38, Sr-1644-1Sh, Sr-1644-5Sh  | Ae. sharonensis                                   |
| Lr57/Yr40, LrAc, Lr76/Yr70   | (syn. Ae. markgrafii = T. dichasians) Ae. caudata |
| Lr40, Lr57, Lr40/Lr57, SR53, Yr40, Lr76/Yr70   | (syn. Ae. ovata) Ae. geniculata                   |
| Lr58   | (syn. Ae. lorentii) Ae. triuncialis               |
| Lr59   | Ae. peregrina                                     |
| Lr19, Lr24, Lr29, Lr38   | Thinopyrum elongatum                              |
| Lr62   | (syn. Ae. triaristata) Ae. neglecta               |
| Yr15, Yr30, Sr2, YrH52, Yr35, Yr35/Lr52, Lr36, Sr13, Lr53, Lr64, Yr36                            | Elymus trachycaulus                               |
| Sr2, Sr9d, Sr9e, Sr9g, Sr11, Sr12, Sr13, Sr14, Sr17, Sr24, Yr1,Yr3,                              | T. turgidum/subsp. dicoccoides/subsp. dicoccum    |
| Yr4, Yr8, Yr9, Yr10, YrCV, YrSP, Yr15, Yr26, YrH52, Yr35, Yr36,                                  | T. turgidum/subsp. dicoccoides/subsp. durum       |
| YrSM139-1B, YrTz2, Sr2/Yr30  |   |
| Lr39   | Ae. cylindrica                                    |
| Lr19/Sr25, Lr29, Sr24, Sr25, Sr26, Sr43, Sr44  | Thinopyrum ponticum                               |
| Lr24/Sr24, Lr38, YrL693, 7a  | Thinopyrum intermedium                            |
| APR: Adult plant resistance  |   |

(temperature, precipitation, light, etc.), virulence of the pathogen, genotype of the plant, host-pathogen relationship, etc. Especially, Eastern and Central Europe and many other countries including Australia, China and India are prone to this disease's effect with the 20–30% crop losses (Khan *et al.* 2013).

Brown or leaf rust (P. triticina (Erikss.) Urban et Markova = P. dispersa(l) f. sp. tritici Erikss. and Henn.= P. recondita f. sp. tritici (Roberge ex Desmaz) Erikss. and E. Henn.: P. triticina or brown rust or leaf rust is the agent of the disease. It is common in most wheat fields. The suitable temperature range for this disease agent is between 10 and 30°C. According to researchers, these diseases cause up to 30% yield losses in wheat. In arid and semi-arid regions, yield losses increase and reach a maximum level. Winzeler et al. (1995) stated that wheat rust resistance genes are modernly effective in two different stages: i) Greenhouse and ii) Field conditions. In this context, effective wheat rust disease resistance genes are of three types: i) effective at seedling resistance stage(s), ii) effective at adult plant resistance stage (APR-Under field conditions), and finally, iii) effective at both stages. First of all,

aggregating different genes that provide wheat rust various rust resistance in a single (wheat) genotype is a prerequisite for successful rust resistance breeding and this technique is applied at leading wheat breeding organizations in the world such as CIMMIYT and ICARDA. On the other hand, during hybridization of wheat to be grown with wild relatives, some important hybridization barriers can be encountered, as discussed below.

# Some of agronomic & cytological obstacles and their solutions

Jyoti (2016) reported that 94–96,000 genes are found in the A, B and D genomes of bread/common wheat (*T. aestivum*) and are linked to rust resistance. If wheat breeders do not find enough resistance to the rust, what they should do is turn to their wild ancestors and try to benefit from these sources (Feldman and Sears 1981; Savadi *et al.* 2018). The following obstacle may be encountered while transferring of characters of interest from wild relatives:

a. It is difficult to hybridize with wild genera and species, and often hybrids become partially or completely sterile,

b. At different ploidy levels, sterility phenomenon may occur,

c. Polyploidy provides an enormous versatility in terms of growth conditions, resistance to diseases and the potential to process the harvested seeds to various products; however, the domestication progress of the wheat resulted in a diversity bottleneck (Dubcovsky and Dvorak 2007),

d. Although information about taxonomy, phylogeny, and geographical distribution is frequently needed for the purpose of use of wild wheat and relatives, this information is not yet sufficient,

e. Wild wheat and its relatives have wild traits (such as hairness, thorniness, waxiness, etc.), low yield(s), insufficient flavor, and unwanted agronomic characteristic(s) that wheat breeder is concerned about.

In addition to them, these problems may arise:

a. Sterility comes from parents and generally emerges in F1,

b. Cytoplasmic and nuclear origin compatibilities,

c. Interaction(s), linkage(s) among/between traits (hairness, thorniness, quality, and quantity inadequacy, etc.),
d. Cytological, floral, structural, genomic, etc. abnormality(ies),

e. The structure of the genome is complex (esp. in bread/common wheats) in most other cereals that are polyploid (in contrast to diploid wheats),

f. Due to some biotechnological methods (such as RFLPs, AFLPs, SSRs, STSs, molecular markers), "large genome size" is a problem (Ellis *et al.* 2014). Low level of polymorphism causes cytological instability and its cellular activity has highly abundant transposable elements (TEs), jumping genes or mysterious movements in bread/common wheat, which comprise 80% of the genome (Dubcovsky and Dvorak 2007).

g. Another problem is about the largeness/volume of the wheat genome. According to researchers this quantity was found as (17.3 pg per cell-17,000 Mbp) in wheat per haploid nucleus, in barley (*Hordeum vulgare*) is  $(5 \times 10^9)$  pg per cell), in maize (*Zea mays*) is  $(5 \times 10^9)$  and in (*Oryza sativa*) is  $(4 \times 10^8 \text{ bp})$  as a member of Poaceae family (Chelkowski and Stepien 2001; Dubcovsky and Dvorak 2007).

h. The development of hybrid embryos is extremely low or non-existent, the weakness of germination, then the appearance of wrinkled-undersized or weak seeds,

i. As the level of polyploidy in the genome increases, the complexity also increases. For example, the complexity in the bread/common wheats-AABBDD genomes is much greater than diploid wheats-AA genomes,

j. The occurrence of three genomes (such as the A, B, and D) for a bread/common wheat complexity for many molecular markers (esp. In RFLPs analysis),

k. Often, wheat cultivar(s)/varietie(s) with D genome(s) is/are more stored but this genome is difficult to map.

But, the following solutions can be used to avoid the above problems (Knott 1959; Gill *et al.* 1985; Özgen *et al.* 1987; Langridge *et al.* 2001):

a. To make the reciprocal hybridizations (especially in backcrosses),

b. To utilize the hybrids (can be a cultivar, landrace, etc.) as bridge crossing material,

c. Need to make use of vegetative propagation technique(s),

d. To use materials with different cytological properties (nullisomy, tetrasomy, isogenic and/or deletion lines especially in the hybridization programs,

e. To use "Colchicine, etc." for blocking the "homoeolog" chromosomal pairing,

f. If necessary, to use some supportive and advanced methods such as mutagen usage (EMS, etc.), embryo rescue or culture(s), tissue culture(s), genetic engineering, gene transfer, biolistic, etc.

g. To use of radioactive radiations such as  $\gamma$ -rays at any stage during the breeding program(s),

h. The use of hybrid embryo development appropriate methods *in vitro* or *in situ*, etc.

i. To use *Agrobacterium tumefaciens* as a vector of transferring the target gene,

j. In case of the necessity to do as a pre-treatment imbibition, seed-coat rubbing (sanding), acid treatment, etc. for entering the water and gas via seed-coat for the germination.

In fact, the main purpose of all breeding programs (including wheat) is to cumulate the desired gene(s) into a single genotype and to make this as sustainable as possible. In this context, the most appropriate method is to transfer rust resistance genes from wild relatives through wheat breeding studies (Merker 1992). In this context, it is essential to use resistant cultivar(s)/variety(ies) for control of wheat rust disease (El Khoury and Makkouk 2010). Mostly, chemical control is used instead of gene transfer practice. But it is clear that the crop losses have not been reduced with a significant increase in pesticide use during the last century (Nelson et al. 2018). Main possible reason(s) of this trend is/are clear. The main reason is the economic impact of using them and the second one is the convenience to use the non-resistant cultivar(s)/variety(ies). As mentioned by Miedaner (2016), most of the cultivated wheats keep their rust resistance for 5-6 years and this ability mainly depends upon genotype, agronomic precautions, ecology, mutation, pathogen virulence, recombination, parasexualism, etc. (Riaz et al. 2016). In fact, the main purpose of these methods is to be able to bring together the resistance gene(s) into one genotype.

# Inducing resistance against rust diseases from wild wheat germplasm

Wheat rust resistance genes can be studied in two classes (Van der Plank 1963):

**Horizontal resistance (HR):** Multiple gene resistance, stable resistance, polygenic/race-non-specific. This means plants have resisted to all races of pathogens.

**Vertical resistance** (VR): A single-gene resistance, unstable resistance, monogenic or oligogenic/race-specific. This means resistance to various races of the pathogens.

In this regard, the best possible way is to be able to use available resistance genes in the wild wheat (*Triticum* spp.) and its relatives. Many genes that provide resistance to wheat rust diseases have been introduced by interspecific crossings made to wild wheat and its relatives. such genes are called "wild or alien" genes.

The humankind is very fortunate since wild relatives have a hypersensitive response (HR) or polygenic resistance genes, which keep durability during the rust epidemy (Simmons 1972; Browning 1974; Lindhout 2002). It is very fortunate that human beings grow or cultivate this type of wheat because their durability has a polygenic or HR inheritance pattern and it has the ability to preserve these properties for a long time in any rust epidemics. With an agronomic perspective, some beneficial and successful examples are available whereby the yield losses have been found to be reduced as, for instance, in T. aestivum, T. elongatum, T. timopheevii, T. bessarabicum, Ae. ventricosa, Ae. bicornis, Ae. speltoides, Ae. tauschii, Ae. geniculata, Ae. biuncialis, Ae. triuncialis, Ae. longissima, Ae. caudata, Ae. sharonensis, T. boeoticum, T. monococcum, T. diccoccum, T. dicoccoides, T. cartlicum, Thinopyrum intermedium, Th. ponticum, T. sarterii, Amblyopyrum muticum, Dasypyrum breviaristatum, Lophopyrum spp., Trichopyrum spp., etc. (Gustafson et al. 2009; Haider 2012; Anonymous 2018d).

#### **Conclusions and future prospects**

Wheat rust diseases are one of the most important biotic bottleneck/constraints that have always been effective in reducing the wheat production due to sunlight and other mutagens, and pathogen alteration for certain periods of time, and only because of this (*i.e.*, pathogen race change at certain periods,  $\approx 5$  years) is an ongoing effort to improve disease resistance breeding. On the other hand, wild wheats and relatives are valuable genetic resources and biological materials that should be utilized in the transfer of rust resistance genes. Therefore, each of them must utilize the wild relatives harboring very valuable durable resistance genes have been transferred from the wild wheats and relatives to cultivated wheats up to now.

There are two types of rust resistance in the wheat firstly, i) horizontal or monogenic at the seedling stage and secondly, ii) vertical/polygenic at the adult stages (field stages). Rust resistance genes have been mostly diagnosed at the seedling stage. However, vertical/polygenic rust resistance genes can protect the wheat cultivar(s)/variety(ies) at seedling and adult plant stages. So, adult plant rust reactions to rust diseases are more widely used, which give more accurate and constructive ideas in terms of resistance to these kinds of diseases in field-grown wheat. Wild relatives are already very rich in the rust resistance genes, which may protect from the rust disease when hybridizing with them if the barriers could be overcome. It should be known that this point is particularly important and valuable in terms of wheat breeding for rust resistance in the future.

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