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Review of Concepts and Methods of Genetic Characterization and Breeding Wheat for Resistance to Rusts

Mesfin Kebede Gessese*

Department of plant science, college of agriculture, Wolaita Sodo University, P O BOX 138 Wolaita Sodo

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***Corresponding author:** Mesfin Kebede Gessese, department of plant science, College of Agriculture, Wolaita Sodo University, P O BOX 138 Wolaita Sodo, Ethiopia

Abstract

Rust diseases are economically the most important constraints to global wheat production. Breeding for resistance to diseases involves the understanding of genetic systems of both the host and pathogen simultaneously. Knowledge of host-pathogen interaction is essential for breeding disease resistant crops, because both host and pathogen populations are genetically diverse. Resistance to rust diseases can be classified into two broad categories; seedling/ all stage resistance (ASR) and adult plant resistance (APR). All stage resistance genes confer resistance at all growth stages against avirulent pathogen isolates. However, ASR genes often succumb to evolution in pathogen populations, whereas APR genes are expressed at later growth stages of the plant, show uniform response to all races of the pathogen and do not encourage pathotypic evolution and the nature of resistance they offer is usually durable. Characterization of resistance to rusts involves gene postulation (multi-pathotype test), genetic analyses and identifying the number and location of the gene(s) conferring resistance. Chromosomal location and mapping genes have been done using cytogenetic analysis; however, since 1990s the advancement of molecular genetics and biotechnology enabled to replace the tedious, time, labor and resource consuming cytogenetic analyses method of locating genes using molecular mapping techniques.

Keywords: Seedling resistance; Adult plant resistance; Multi-pathotype test; Genetic analysis; Genetic markers; Gene mapping

Introduction

Wheat is prone to both biotic and abiotic stresses that cause significant yield losses. Among the biotic stresses, fungal diseases are the most important production constraints to wheat [1,2]. Among the fungal diseases of wheat, rusts are economically the most important diseases [3]. Rusts of wheat might be the oldest plant parasites (pathogens) as evidenced by the excavations of Urediniospores of stem rust in Israel that dated back to 1300BC and were reported as serious diseases of cereals in Italy and Greece before 2000 years ago [3-5]. The occurrence of widespread epidemics of wheat rusts at the beginning of the 20th century initiated the need for in-depth studies in genetics of disease resistance in plants, life cycle of plant pathogens and genetics of host-parasite interactions [3,6]. Rust diseases are still the major threats of wheat plant causing significant yield losses and decreased qualities of grains [7,8] particularly, the emergence of the new race of stem rust of wheat (Ug99) brought a major anxiety in the world wheat production (McIntosh and Pretorius, 2011). Wheat is affected by three different types of rust diseases; leaf rust (caused by *P.triticina* Eriks), stripe rust or yellow rust (caused by *P.striiformis* Westend.*f. sp. tritici* Eriks) and stem rust (caused by *P.graminis* Pers. *f. sp. tritici* Eriks) [5,9-11]. Leaf rust or brown rust is the most common and widespread rust of wheat across the world as compared to stem and stripe rusts [5,11,12]. It attacks mostly the leaf blades, and under more favorable conditions can also attack leaf sheaths and glumes [5,13]. The causal agent (*P.triticina*) is an obligate parasite, which has the capacity to create infectious urediniospores only on live leaf tissues. It has primary (telial or uredinal) and secondary (pycnial or aecial) hosts to complete its full life cycle. The known primary hosts are bread wheat, durum wheat, cultivated and wild emmer wheat, *Ae.speltoides, Ae.cylindrica* and triticale; the secondary or alternate hosts are *Thalictrum speciosissimum* and *Isopyrum fumaroides* [11,14]. Under suitable environmental conditions (about 10-25°C and availability of free water on the leaf surface), the wheat plant produces dark brown, two-celled teliospores [6,14,15]. Black rust or stem rust has been one of the most devastating diseases of cereal crops across the world known to cause famines and economic as well as political crises particularly in south Asia that led to the inception of Green Revolution [16]. Stem rust incidence had been significantly reduced by eliminating its alternate host

(barberry species) particularly in North America and Western Europe, which assisted to reduce early infection of the wheat plant [11]. The distribution of semi-dwarf, high yielding and stem rust resistant varieties controlled the adverse effect of the disease [16].Pgt usually causes damage to the above ground parts of the wheat plant, and contaminated plants frequently produce smaller number of tillers and fewer kernels per spike and even total loss of yield can occur due to breakage of the stem [8,17]. The spores of Pgt can germinate from 2°C as the minimum temperature, while the optimum and maximum temperatures are 15-24°C and 30°C, respectively. Formation of spores starts from 5°C; however, sporulation optimizes at 30°C and can even go up to 40°C as the maximum temperature [5].

Puccinia striiformis f. sp. *tritici (Pst),* a causal agent of stripe rust of wheat, has a potential to be equally destructive as stem rust of wheat, though its low temperature requirement restricts its widespread attack across all wheat growing regions of the world [5,6,18,19]. Nonetheless, past evidences confirmed that stripe rust epidemics could be an important threat to the major wheat growing areas of the world such as China, USA, Southern Asia, Northern Europe and Australia [18-20]. The disease infects the wheat plant at any of the growth stages starting from single leaf stage up to maturity as long as the plant is green and causes chlorotic spots that produce yellow coloured stripes of uredinia [18,21]. The uredinia of Pst are lesser in size than uredinia of both stem and leaf rusts. They grow mostly in the upper surface of the leaf and to some extent particularly in susceptible plants, it can parasitize on the lower surface of the leaf, leaf sheaths, glumes, awns and even on immature green kernels [6,18]. The uredinial spores can germinate on the surface of the leaf with minimum requirement of 3 hours for dew formation and 0°C as the minimum, 12-16 °C the optimum and 20 °C maximum temperature [6,18,21,22].

Breeding for Rust Resistance

Resistance refers to the ability of plants to remove or minimize insect pests and pathogens by genetic and molecular mechanism [23]. Breeding for resistance to pest involves the manipulation of genetic systems of both the host and parasite simultaneously [24,25]. Knowledge of host-pathogen interaction is essential for breeding crops to be resistant against a certain pathogen because populations of both the host plant and the pathogen entail genetic diversity [26]. Particularly in rusts where the pathogens have a series of physiological races, it is imperative to use and apply the hypothesis developed by H. H. Flor for flux and flux rust disease reaction that stated "for each gene conditioning resistance in the host there is a corresponding gene conditioning pathogenicity in the fungus" [27].Flor's hypothesis was further extended as a general principle to disease reactions between a host and parasite system [28]. However, there are exceptions that the gene-for-gene concept does not hold true; such as presence of race non-specific single APR genes [22], two or three genes of the host control resistance, presence of modifiers or suppressors in major genes, epistasis and the major exception is several genes (quantitative trait loci) each with minor contribution but additive effects control resistance [29,30].Prior to planning a plant breeding project for resistance against a particular pest there should be a concrete evidence on the economic importance of the host-pathogen systems prevailing between the crop and the pest which can be justified by the significant yield losses due to the pest and knowledge on the level of genetic diversity of the pest population [31]. True disease resistance has its root in the plant's genetic material (genes) and can be manipulated using plant breeding methodologies to develop a resistant cultivar [24]. Breeding wheat for resistance to rust diseases has been and still is an economically as well as environmentally sound strategy, which is also handy for farmers to apply without additional production expenses [9,31,32].

For effective rust control program, anticipatory breeding should be in place to reduce the pathogen population and ensure a stable production by being ahead of the pathogen through a continuous germplasm development process of characterization and identification of novel resistance genes [33]. They further opined that anticipatory breeding could be realized by conducting annual country wide pathotypes surveillance program and cataloguing cultivars with resistant genes and their respective races of pathogen. There are two major categories of genetic resistances of wheat plants against rust diseases and commonly described as seedling and adult plant resistances.

Types of genetic resistance of wheat against rust pathogens

Seedling resistance/ race-specific resistance: This type of plant resistance to biotic stresses is known by different names such as specific, complete, simple, qualitative, race specific, overall, vertical, non-uniform, differential, hypersensitivity, oligogenic, major and non-durable resistance [24,31]. It is characterized by a low infection type of disease reaction in host-isolate interaction of host genotypes with different races (isolates) of a pathogen [24]. The occurrence of stable physiological races was first demonstrated by Stackman and his coworkers who tested a range of wheat genotypes and pathogen cultures of *P.graminis f.sp.tritici* during 1914-1919 cropping seasons [3,34] and laid the foundation for the knowledge of pathogenic variation and inheritance studies of resistance genes. This method was further extrapolated for leaf and stripe rusts of wheat and eventually applied for selection and breeding works for the development of rust resistant wheat cultivars [3]. Seedling resistance genes once expressed, they confer resistance to the plant throughout its growth stage [19,35]. Except in few cases, race specific resistance genes in wheat rusts follow simple or Mendelian pattern of inheritance, have complete dominant gene action and the avirulence genes in the pathogen is mostly dominant [5,19]. Hence, they are relatively easy to select and transfer using the conventional breeding procedures for crop improvement work [36-38]. Vertical or major gene resistance is usually successful in conferring resistance to the

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host up until new races of the pathogen established (about 4-5 years) and become succumbed to it, which is termed as the 'boom and bust' cycles [36,37].The boom and bust cycle operate when a cultivar with major gene resistance becomes widely adopted in a particular region, it is called the 'boom' phase. Consequently, the pathogen undergoes selection pressure against the virulent race and the other less virulent gene that the cultivar lacks major gene keeps on multiplying until it reaches epidemic level across the region which is the 'bust' phase [24,19]. Few exceptions to this are the case of ASR gene *Sr31* remained resistant for over three decades [16,39,40]. In the same manner, *Lr24* remained resistant in Australia from 1983 up to 2000 until it was broken by a new virulent race of *P.triticina* [40]. To date over 59 stem rust, 81 stripe rust and 79 leaf rust resistance genes have been identified and catalogued among which some are adult plant resistance genes [41-45].

Adult plant resistance (APR)/ Race non-specific resistance: APR genes are also called complex, quantitative, general, mature plant, horizontal, polygenic, partial, minor gene, durable, slow-rusting, field resistance, etc. [9,24,31]. This type of resistance shows uniform reaction to all isolates or races of the pathogen population hence it is called non-specific or uniform resistance [38,46]. The genes controlling one race non-specific resistance could be either single [22] or many (polygenic) and each gene has small (miner) effect individually but possesses additive gene effect where the presence of more genes indicates higher level of resistance [47]. Quantitative traits are highly influenced by the environment and segregate at many loci, which is the main reason for the continuous variation observed in their phenotypic expression [48]. An interesting characteristic of APR genes is they have high heritability as they are mostly linked genes [49,50] and hence positively respond to selection and breeding [24,31,48]. Generally, APR displays longer latent period, lower infection frequency, smaller uredinial size, shorter duration of sporulation and a smaller amount of spore production per infection site [51]. With regard to wheat rust, adult plant resistance is expressed at later growth stage of the plant, which is characterized by susceptible disease reaction but with reduced rate of disease development [9,22,31]. Some studies conducted in adult plant resistance of wheat rust diseases revealed that a few of these resistances were race specific [5]. Furthermore, greenhouse studies confirmed presence of virulent races for APR genes *Lr12, Lr13, Lr22b* [52]and quite recently for *Lr37* [13].

Unlike to race specific genes, APR genes are expressed at later growth stage of the plant, show uniform response to all races of the pathogen and do not induce mutation of avirulent genes of the pathogen; because of these reasons, the nature of resistance they offer is usually durable [22].Exhaustively studied genes with pleiotropic effects that confer resistance to leaf rust, stripe rust and powdery mildew diseases are *Lr34/Yr18/Sr57/Pm38/Ltn1*, *Lr46/Yr29/Sr58/Pm39/Ltn2* and *Lr67/Yr46/Sr55/Pm46/Ltn3* [53-56]. Presence of a single or couple of APR genes in a cultivar may not provide enough resistance levels in high disease pressure areas, however, cultivars with high levels of resistance were developed by pyramiding three to five APR genes [8,47,57-19]. To facilitate development of wheat cultivars with durable resistance to rusts, researchers continue to mine new APR genes from new and available sources of germplasm [42]. Recent additions to the already existing APR genes with complete details including their respective chromosomal locations are *Sr55, Sr56, Sr57, Sr58, Yr46, Yr48, Yr52, Yr54, Yr59, Yr62, Yr68, Yr71, Yr75, Yr76, Lr67, Lr68* and *Lr74* [42-44].

Characterization of Wheat Germplasm for Resistance to Rusts

Gene postulation (multi-pathotype test), genetic analyses and cytogenetic analyses were the general methods used for spotting rust resistant genes [5]. However, since 1990s the advancement of molecular genetics and biotechnology enabled to replace the tedious, time, labor and resource consuming cytogenetic analyses method of locating genes which involves the use of monosomics, telosomics and nullisomics using molecular mapping techniques [60].

Multi-pathotype Testing

Table 1: Responses of Arbitrary Genotypes Carrying Known Stem Rust Resistance Genes to the Different Arbitrary Pathotypes.

Resistance Gene	Pt.1	Pt.2	Pt.3	Pt.4	Pt.5	ULI *
R1	H**	H	H	H	H	X-
R2	H	L***	H	H	H	22+
R3	H	L	H	H	H	2=
R4	H	L	H	H	H	X-
R5	L	L	L	L	L	2=
R6	L	L	L	L	L	22-
R7	L	L	L	L	L	2- to 3-
R8	L	L	L	L	L	2=
R9	L	L	H	H	H	;
R10	L	H	L	L	L	X-

* =Usual low infection types; **= high infection type; ***=low infection type, Pt=pathotype.

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It is the classical application of the gene-for-gene association, which is a primary practical screening method of germplasm for identifying seedling resistance genes [36].Multi-pathotype testing entails the use of the available isolates of the rust disease, resistance sources to be evaluated and the differential cultivars for the known resistance genes. The infection types (low and high) observed in the seedling test (Table 1) could be correlated with the response of the differential cultivars included in the seedling test to postulate resistance genes that showed gene-for-gene relationship [3,5]. The genotypes that showed resistance reaction to all the virulent races (e.g. *R5, R6, R7, R8*; Table 1) may possess the new resistance gene(s) or combination of two or more R genes [37]. Those resistance genes that couldn't be recognized in the multi-pathotype test could be subjected to further genetic and cytogenetic analyses for identification and locating their specific place in the plant genome. However, multi-pathotype tests cannot help in identifying adult plant resistance genes as the test is performed in the seedling stage, hence, it should be supplemented with field screening of the same germplasm to get a complete picture of the germplasm as a source of resistance for practical plant breeding research works [36,61].

In the above table a genotype with similar phenotyping data as *R1* do not possess effective seedling resistance gene for the pathotypes used in the study, however, it may be resistant in the field due to presence of adult plant resistance genes [62]. Genotypes with similar infection types like *R2, R3* and *R4* are effective only to pathotype Pt.2; however, they can be categorized by their respective low infection types displayed (Table 1). Several authors postulated rust resistance genes in different sets of wheat genotypes. [63] postulated *Yr2, Yr3, Yr4, Yr6, Yr7, Yr9* & *YrA* either singly or in combination in a set of 42 Ethiopian bread wheat cultivars where 67% carried *Yr9* and five cultivars showed resistance reaction to all races of stripe rust used for seedling tests. A multi-pathotype test conducted on 41 emmer (*Triticum dicoccon* Schrank) and 56 durum (*T. durum* Desf.) wheat accessions from Ethiopia using 5 stem rust pathotypes resulted in the postulation of *Sr7b, 8b, 9a, 9b,10, 14, 24, 27, 28, 29, 30, 31, 32* and *Tt-3+10* in the sixteen of the emmer and five of the durum wheat germplasm accessions [64]. Randhawa, et al. [65] postulated stem rust and leaf rust resistance genes *Sr7b, Sr8a, Sr12, Sr15, Sr17, Sr23, Sr30*, & *Lr1, Lr3a, Lr13, Lr14a, Lr16* & *Lr20* either singly or in different combinations among 87 Nordic spring wheat genotypes using 8 stem rust and 7 leaf rust Australian pathotypes. They further confirmed the presence of APR genes *Yr48, Lr34/Yr18/Sr57, Lr68* & *Sr2* using molecular markers linked with these genes.

Genetic Analysis

It is conducted to determine the number and inheritance characteristics (dominant, codominant or recessive) of a gene(s) of interest in a particular genotype (cultivar, landrace, wild plant, etc.). Roelfs, et al. [5]described that genetic analyses studies may involve crossing resistant and susceptible cultivars or crossing various parents with one or more known gene(s) for resistance where the latter is called 'test of allelism'. If the resistance gene is fully expressed in the F_1 plant, it may indicate the dominance characteristics of the gene and vice versa, an intermediate response indicates partial dominance. For a self-pollinated crop like wheat the inheritance of resistance and number of genes involved in the expression of the trait can be determined using tests on F_2 populations and F_3 families, backcross/test cross (BC/TC) F_1 and F_2 families, doubled haploid (DH) or RILs of single-seed descent (SSD) populations and mixtures of more than one of these methods [5,31]. Chi-squared analyses are usually computed to estimate the number of genes by testing the goodness of fit of observed ratios to the theoretically expected values. Nearly all of the loci in wheat rust resistance genes discovered, mapped and catalogued so far have gone through the process of genetic analysis followed by chromosomal location through monosomic-cytogenetic analysis and more recently through bulked segregant analysis and/or selective genotyping [66]. Genetic analysis of the stripe rust differential cultivar Strubes Dickkopf conducted by phenotyping the backcross population Taichung 29/(Taichung 29*6/Strubes Dickkopf) using Pst pathotype CYR26 at seedling stage resulted in a single dominant gene *YrSD* located on chromosome 5B [67]. Singh, et al. [45] identified two independent APR genes for stripe rust in old American durum wheat cultivars Leeds and Wells by phenotyping Leeds/Bansi and Wells/Bansi populations using Pst pathotypes 110 E143A+ and 134 E16A+ at adult stage, respectively. Similarly, genetic analysis carried out on the durum wheat population Yavaros 79/Kingfisher for stem rust resistance at seedling stage confirmed the presence of *Sr9e* and *Sr12* in cultivar Yavaros79 [68]. Nazari & Wellings [69] identified two independent seedling stripe rust resistance genes *YrBat1* and *YrBat2* with their respective distinct infection types 12=C and 23=C in the Australian wheat cultivar Batavia by phenotyping the F_3 families of Batavia/AvS using Pst pathotypes 110 E143 A+ and 134 E16 A+.

Chromosomal location of new resistance loci

Several wheat rust resistance loci have been successfully mapped using cytogenetic analysis, which involves the use of aneuploids (particularly monosomics and monotelosomics) as one parent to be crossed with the genotype possessing the resistance gene [5,70]. In bread wheat, E.R. Sears developed the 21 complete sets of monosomic plants including the 41 possible telosomic (one chromosome arm missing plant) from Chinese Spring wheat variety [70]. The rust resistance genes mapped using cytogenetic analysis include *Yr10* on chromosome 1B [67], *Yr10vav* on 1BS [71], *Yr17/Lr37/Sr38* on chromosome 2A [72], *Yr3a* and *Yr3c* on chromosome 1B, *Yr4a* and *Yr4b* on 6B, *YrMin* and *YrND* on 4A, *YrDru* on 5B, *YrSte* on 2B, *YrH46* and *YrDru2* on 6A, *YrSte2* on 3B, *YrV23* on 2B, *Yr2* on 7B and *YrYam* on 4B [73] *Yr32* on chromosome 2A [74], *Yr27* on 2B [75], *Yr15* on 1BS [76], *Yr35/Lr53* on 6BS [77]. To date, the use of cytogenetic analysis for chromosomal location and mapping of genes is very limited or rare due to the advent of molecular markers in the 1980s [78]

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and the relatively tedious, laborious and time-consuming nature of monosomic and cytogenetic analyses. Genomic location and mapping of disease resistance genes like wheat rusts has become more simplified with a novel method called bulked segregant analysis developed by Michelmore, et al. [79]. Based on the phenotyping data of a population for a particular rust pathotype bulked segregant analysis (BSA) can be conducted by using two pooled DNA samples constituted from each of the selected homozygous resistant and homozygous susceptible genotypes of a segregating population [79]. The BSA results in identification of the genomic (chromosomal) location of the gene of interest through marker-trait association with list of markers linked to the gene(s).

Those markers that showed polymorphism between the two pooled DNA samples are used as references to identify the chromosomal location of the gene of interest [79]. After obtaining the polymorphic markers screening of the entire mapping population including the parents is done [80]. BSA is currently the most widely used technique for marker identification in the majority of plant breeding marker development programs [66]. Wheat rust resistance genes *Yr47* [81], *Sr49* [82], *Yr51* [83] & *Yr57* [84] have been successfully located using BSA.Selective genotyping of few representative resistant and susceptible lines of a population using 90K infinium assay [85]is the latest approach for locating resistance genes in the wheat genome [86,87].

Plant genetic markers

Genetic markers characterize genetic discrimination between different organisms and genotypes of a species by serving as 'signs' or 'flags. The markers tightly linked to the desirable gene(s) may be referred as gene 'tags' [80,88]. Genetic markers are basically benchmarking on chromosomes that assist as reference points to the position of genes of interest when a genetic map is created. In order to map genes using markers, knowledge of the association (linkage) of markers to genes of interest is a prerequisite. The logic behind in using markers is that "an easy to observe trait" is tightly linked to an invisible and required trait or gene. Plant genetic markers could be morphological and agronomic traits (visually assessable traits), biochemical (gene products or proteins/ isozymes) and DNA markers [1,24,31,89-91].

Plant morphological markers: Morphological markers or classical markers are phenotypic expressions of the organism as a product of the interaction of genes and the environment such as flower color, seed shape, growth habit, pigmentations, etc., which are most frequently observed in adult plants [60,80]. Morphological markers produce phenotypes which can be readily identified, but not necessarily of direct economic importance [60,92]. In breeding of wheat for disease resistance, association of phenotypic markers with low disease reaction permits indirect selection of the resistance gene [31].The use of morphological markers has been proved valuable in progeny breeding where appropriate crosses are made to transfer a gene of interest from a donor parent to an elite genotype (a recurrent parent in backcross breeding).To mention some of the phenotypic expressions of wheat associated with rust resistance are: 'brown chaff color' linked with stripe rust resistance gene *Yr10*, pseudo black chaff and seedling chlorosis linked with stem rust resistance gene *Sr2*, and leaf tip necrosis linked with genes for resistance to leaf rust (*Lr34*), stripe rust (*Yr18*), and barley yellow dwarf virus (*Bdv1*) [31,60,93]. However, their lack of stability in expression across environments and being dependent on growth stages plus their limited number is the major bottleneck to use them for marker assisted selection [80,88,89].

Biochemical (Isozyme) Markers: The most common biochemical markers are the multiple forms of the enzymes called allozymes or isozymes [60]. Isozymes are structural variants of an enzyme with variable molecular weights and electrophoretic mobility but have the same catalytic activities [94]. Since isozymes are proteins, the difference in electrophoretic mobility is caused by point mutation in the DNA leading to amino acid substitution and results in change of the shape and charge of a molecule [95]. Such polymorphic genes or allelic variations have been known to be associated with economically important traits in many agriculturally important crops including wheat [94]. In wheat, the proteins produced by the genes encoding for both the high and low molecular weight subunits of glutenin are known to be polymorphic and readily detected by polyacrylamide gel electrophoresis, making these genes useful markers for linked genes on Group 1 chromosomes. These genes are located at the *Glu-A1, Glu-B1, Glu-D1*, & *Glu-A3, Glu-B3, Glu-D3* loci of the long and short arms of chromosomes 1A, 1B, and 1D, respectively [92]. Stripe rust resistance gene *Yr10* and resistance gene to fusarium head blight of wheat has been reported to be linked with gliadin proteins [31]. Since it does not require DNA extraction, sequence information and primers, isozyme analysis is fast, cheaper and simple to use.However, the limited number of isozymes available, their uneven distribution in the genetic map and being dependent on developmental stages of plants or tissues for expression undermines their importance as genetic markers [24,60,94,95].

Molecular (DNA) markers: A molecular (DNA) marker is a specific segment of DNA with identifiable DNA sequences found at specific locations of the genome and is representative of the differences at the genome level [89]. Since molecular markers do not have any biological effect, they cannot be considered as genes; instead can be regarded as persistent landmarks in the genome transmitted by the standard laws of inheritance from one generation to the next [91,96]. The detection of polymorphisms in fragmented (at specific sites) plant genomic DNAs radically accelerated the development of molecular markers for plant breeding applications [94,96]. DNA markers are currently the most widely used markers worldwide as they have several advantages over both morphological and isozymes markers. Some of the benefits of using DNA markers are their number is nearly unlimited, they are not dependent of developmental stage of the organism and not influenced by environmental factors, they are distributed evenly across the genome of the plant and all markers

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can be detected with a single technique [89,91,94,96,97]. The likely sources of DNA markers in the genome of an organism could be point mutations and the errors occurring in replication of tandemly repeated DNA of an organism [80].

Mapping populations for DNA markers: Mapping population for a particular trait should be segregating plant population derived from a single cross between contrasting parents for one or more traits [80,91,96]. In self-pollinating species like wheat, mapping populations are developed from parents that are naturally highly homozygous in nature (such as landraces and wild genotypes of closely related species of wheat as doners) and inbred lines such as commercial cultivars or isogenic lines as susceptible or recurrent parent [88]. For preliminary genetic mapping studies, population sizes can range from about 50 to 250 individual plants [80]; however, larger populations are required for high-resolution mapping [97]. Mapping populations could be progenies from the second filial generation (F_2), BCF1/F2s, RILs, DHs, and near isogenic lines (Badea, et al.), however, BCF2s, RILs and DH are prefered for accurately identifying closely linked markers and for mapping of QTL [88]. In case of mapping of a disease resistance gene such as wheat rusts, mapping population is developed by a single cross between a resistant genotype (low infection type) and a susceptible genotype (high infection type) in order to get segregating population for a particular trait [79].

Gene mapping using DNA markers: Genetic mapping is the positioning (locating) of gene(s) to a particular region of a chromosome and determining the location and relative distances between genes on the chromosome, which is quite similar to signs or benchmarks along a highway that may be termed as a 'road map' [80]. Markers that are tightly linked with a gene of interest will be transmitted from generation to generation more often than markers or genes located distantly [98]. The relative distance between genes and/or genetic markers in the chromosomes is computed in terms of recombination frequency obtained from the segregating population as a result of crossing over during meiosis. Less number of recombinant genotypes indicates the closeness of the linkage and vice versa (http://en.wikipedia.org/wiki/Genetic_linkage). The distance between genes and/or closely linked markers is expressed in terms of centimorgans (cM), which is defined as the distance between genes for which one product of meiosis in 100 is recombinant. A recombinant frequency (RF) of 1% is equivalent to 1cM. The maximum proportion of recombinants cannot go beyond 50% that would be the condition where the two genes are unlinked and could be located either at the farthermost opposite ends of the same chromosome or possibly in different chromosomes [80].

Since markers segregate in Mendelian fashion, we compare the observed genetic ratios to the expected ones and compute chi-square tests to detect significant deviations. The final step of linkage map construction is to code the data for each DNA marker on each individual plant and conduct linkage analysis by calculating odds ratios (ratio of linkage versus no linkage) termed as logarithm of odds value or LOD score using one of the commonly used statistical software programs such as Mapmaker/EXP, MapManager QTX or Join Map [80]. So far about 210 rust resistance genes has been named and mapped [42] where closely linked markers ready to be used for marker assisted selection have been reported for the following genes *Sr2, Sr15, Sr22, Sr24/Lr24, Sr25/Lr19, Sr26, Sr31/Lr26/Yr9, Sr33,Sr36, Sr38/Lr37/Yr17, Sr39/Lr35, Sr40, Sr45, Sr50, Lr17a, Lr21, Lr34/Yr18, Lr42,Lr47, Lr51, Lr57/Yr40, Lr58, Lr67/Yr46/Sr55, Yr4, Yr10, Yr15, Yr24, Yr32, Yr35,Yr36, Yr47,* & *Yr49* [26].

Mapping QTL genes: The genomic region related to quantitative traits is termed as quantitative trait loci (QTL) [97]. The discovery of DNA technology and molecular markers realized QTL mapping [80,97]. QTL analysis is a powerful tool for identifying the genomic locations as well as estimating the number of genes involved either for simple or complex inheritance [99]. In QTL analysis, advanced backcross generations, DH, or RILs (developed by SSD method) populations are commonly used for identifying linkage between molecular markers and polygenes [91]. Markers help in dividing the mapping population into various genotypic classes based on presence or absence of a certain marker locus and conclude whether significant variation exists between phenotypic means with respect to the trait being measured [80,97]. Linkage analysis in QTL is performed using three widely used methods; single marker analysis (single point analysis), simple interval mapping (SIM) and composite interval mapping (CIM) [80]. The most efficient method of QTL detection that combines interval mapping with multiple linear regression is composite interval mapping (CIM) analysis [100,101]. CIM analysis includes additional markers apart from the adjacent pair of linked markers used in SIM; it is considered as the most precise and widely used method of detecting QTL in the genome [80,97,100,101]. So far over 80 leaf rust and 140 stripe rust resistance QTL were identified and mapped mostly using CIM analysis [51,102].

Evolution of molecular marker system: The application of restriction enzyme endonucleases to cut DNA of interest into fragments at specific sites that can be revealed by polyacrylamide gel electrophoresis [103], paved the way for application of Random Fragment Length Polymorphisms (RFLP) in genetic studies. RFLP was used for the first time in 1974 for the physical mapping of temperature sensitive mutants of adenovirus [78,88]. Consequently, construction of the first map of the human genome based on molecular markers using RFLP method launched the use of DNA markers for construction of linkage and/or genomic maps in other organisms [24,94]. The invention of polymerase chain reaction (PCR) in 1990 was another breakthrough in molecular marker technology that has introduced a new cohort of DNA markers into the modern plant breeding systems [104]. Based on their detection methods, DNA markers can be categorized in to three groups:

a) Hybridization based markers: RFLP

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b) PCR based markers: RAPD, SCAR, SSR, STS, AFLP, CAPS

c) DNA chip, sequence based and high throughput DNA markers such as SNP, DArT, DArTseq, GBS [89,98,105-107].

The choice of DNA markers is mainly based on reliability, the quantity and quality of DNA needed for analysis, simplicity and time taken for performing the assay, level of polymorphism and overall cost needed to perform the task [107,108].

Restriction fragment length polymorphism (RFLP)*:* RFLP is the first molecular marker [78] and the most widely used non-PCR or hybridization-based technique [96,98,109]. This technique employs DNA restriction enzymes which recognize specific sequences in DNA and catalyze endonucleolytic cleavages, yielding fragments of defined lengths [78]. The restriction enzymes depict a pattern of variations among DNA fragment sizes in individual plants or animals of the same species. The likely sources of these variations between individuals could be point mutations, insertion/deletion, translocation, inversion and duplication [78,88,89,104].Because of these, digestion of DNA with restriction enzymes leads to production of fragments whose number and size can vary among individuals, populations, and species [91]. RFLP analysis undergoes several steps. It includes extraction of DNA from plant, digestion of the DNA with one or more restriction enzymes (e.g. Msel, EcoRI, Pstl, etc.), separation of the restriction fragments in agarose gel using electrophoresis, transfer of separated fragments from agarose gel to a filter by southern blotting (cloning of individual fragment into a plasmid), labeling of cloned DNA sequences with radioactive (^{32}P) probe and hybridization of labeled single stranded probe to its single stranded DNA counterpart on the filter, finally autoradiography is done (washing of the filter followed by exposure to x-ray film) [91,104] Several rust resistance genes have been successfully mapped with RFLP markersincluding *Lr1, Lr9, Lr10, Lr13, Lr19, Lr23, Lr24, Lr27, Lr31, Lr34 & Lr35 [110], Rpg1 [111], & Yr15 [112].*

Random Amplified Polymorphic DNA (RAPD)*:* This technique is based on differential PCR amplification of genomic DNA that infers DNA polymorphisms produced by shifting or deletions at or between oligonucleotide primer binding sites in the genome using short arbitrary oligonucleotide sequences (often 10 bases long) [89,113]. Since RAPD technique does not require prior sequence information of the genome to be assayed, it can be applied to any species of plants or animals by using common primers.RAPD method was [89,113] popular due to its speed, high efficiency and simplicity, free from radioactivity and low cost of agarose gel electrophoresis. However, it has low reproducibility and lacks codominant markers [88,89,94]. Rust resistant genes mapped by using RAPD markers include ***Lr24, Lr28, Lr29, Lr37 [110], Yr5 [114], Lr41 [115], Rpg4 [116].***

Amplified Fragment Length Polymorphism (AFLP): AFLP is another PCR based technique that generates DNA fingerprints without prior sequence information of the genome for DNA analysis of any species [104]. According to Vos, et al. [117], AFLP has 3 major steps: i) restriction of the DNA and ligation of oligonucleotide adaptors, ii) selective amplification of sets of restriction fragments, and iii) gel analysis of the amplified fragments. The number of fragments generated depends on the recognition of the unique nucleotides flanking the restriction sites [104]. The primers so called the rare cutter and frequent cutter together can generate about 50-100 restriction fragments [89,117]. AFLP can be used in genetic and physical mapping, to distinguish closely related individuals at sub-species level. The AFLP mapping applications in plants include forming linkage groups in crosses, locating genomic regions with markers for gene pyramiding and measuring the degree of relatedness and/or diversity between cultivars [89]. Wheat rust resistance genes tagged using AFLP markers include ***Lr9 & Lr19 [118], Lr41 [115] & Lr26/Sr31/Yr9 [119].***

Microsatellites markers: Microsatellites are the smallest classes of repeated DNA sequences ranging from one to five nucleotide motifs found scattered in all eukaryotic genomes, which are commonly called simple sequence repeats (SSR), short tandem repeats (STR) or simple sequence length polymorphisms (SSLP). These markers usually arise due to strand slippage or the so called 'slipped strand mispairing' occurring during DNA replication, which leads to gain or loss of one or more repeat units [88,89]. The variation in the numbers of repeat units is the source of SSR allelic differences within the microsatellite structure. The repeated sequence usually includes two, three or four nucleotides; the familiar example of a microsatellite is a di-nucleotide repeat (CA)n, where 'n' refers to the total number of repeats that ranges between 10 and 100. In bread wheat, the microsatellites (GA)n/(GT)n, (AC)n, and (AG)n are found every 270kb, 292kb and 212kb of DNA, respectively [106]. Microsatellites are PCR based sequence specific molecular markers, which require prior sequence information of the genome of the species to be assayed. SSR markers are the most widely used and accepted DNA markers as they are codominant, plentiful, possess high degree of allelic diversity, highly reproducable, require low amount of DNA, highly transferable between populations, easy to assess their size variation by PCR with pairs of flanking primers [89,91,120]. In the last two decades SSR markers have been extensively used to map several rust resistance genes (both ASR and QTL) some of the genes linked to SSR markers include ***Sr2, Sr13, Sr17, Lr48, Lr49, Lr63 and Yr26 [121], Sr9h, Sr42, Sr49, Yr51, Yr57, Yr69 [42].***

Single Nucleotide Polymorphism (SNP)*:* SNPs are the new generation DNA markers for individual genotyping necessary for marker-assisted selection [122]. SNPs represent the most abundant DNA markers distributed across the genome and compose about 90% of genetic variation in any organism [89,91,104,105]. They are created by a single base change (insertion, deletion or substitution) in a DNA sequence, with an alternative of two possible nucleotides at a given loci, hence they are bi-allelic markers [91,104,122]. The SNPs are often found in the non-coding regions of the genome [88,89]. Their abundance in the plant genomes made them an important tool for mapping,

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marker assisted breeding and map-based cloning [91,105]. The SNPs are made available through sequencing of candidate genes/PCR products/whole genomes of more than one genotype [91,105]. Ravel C, et al. [123] reported an average of 1 SNP every 334 bases in hexaploid wheat from a sequence of 21448bp size of DNA composed of 21 genes. These authors found significant variations between the coding (1 SNP every 267 bases) and non-coding (1 SNP every 435 bases) regions. The progress in DNA marker technology has made possible for the development of high throughput genotyping by sequencing (GBS) platforms like 9K SNP array [124] and later the Illumina iSelect 90K Infinium SNP genotyping array [125] to be used for identification and mapping of genes in wheat.These microarray-based markers has become markers of choice for bulked segregant analysis construction of high density maps [57,126], quantitative trait loci (QTL) mapping [12,49,127,128] and genome wide association mapping [129-132] with a limited expense in terms of time and money [133-135].

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