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Review Article

Quantitative Trait Loci (QTLs) for Plant Disease Response

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Quantitative resistance traits are controlled by multiple genetic loci contributing to continuous allelic variation on the phenotype. Although they cannot be shown to be conditioned by individual discrete recognizable loci through classical quantitative genetics, they have been characterized into recognizable quantitative trait loci (QTLs) employing DNA-based markers. A QTL is a map position on the chromosome localized relative to the position of the genetic marker locus, and is identified through significant correlations between the segregation at a certain genetic marker locus and the variation in quantitative (trait) resistance value. Therefore, the preservation of linkage disequilibria between the genetic markers and the QTLs is the basis of marker-assisted localization of quantitative trait loci in specific chromosomal regions of the genome. The advent of QTL mapping has made it possible not only to uncover the magnitude of the effects of QTLs on plant disease response, but also to describe the roles of such specific loci in genetically complex disease resistance traits and to identify the genomic regions contributing to resistance function. With QTL mapping, it has now become possible to characterize the epistatic interactions between different resistance genes, gene × environment interactions and the relationship between quantitative resistance and race-specificity. All these developments have resulted in addressing some of the fundamental questions of plant-pathogen interactions through genetic dissection of the resistance response/ function quite unthinkable even a decade earlier. Partial resistance genes having small continuous effects on resistance function, which were classically thought to be race-nonspecific, have been shown to be race-specific by QTL mapping, suggesting that partial resistance genes might be 'defeated' major genes with residual effectiveness and race specificity. In spite of tremendous potential of QTL mapping, the usefulness of QTL-marker association for effective marker-assisted selection (MAS) is conditioned, rather limited, by epistatic interactions with other loci, variations in linkage phase and QTL × environment interactions. Although additive QTLs can significantly increase the efficiency of resistance breeding, recent studies are revealing the existence of epistatic interactions between the QTLs as well as QTL × environment. In this context, the recently introduced 'candidate' gene approach may aid in the discovery of the functions of the QTLs by linking the genetic QTL analysis with molecular biology methods- an ambitious step toward treating the QTLs as 'qualitative' loci and realizing the positional cloning of partial resistance genes underlying the QTLs conferring effective durable resistance in different crop species.

Key Words: Candidate genes, disease resistance, DNA markers, gene mapping, quantitative genetics, quantitative resistance locus, quantitative resistance traits, quantitative trait locus, plant-pathogen interaction

Introduction

After the rediscovery of Mendel's work [1] in the last century, it was recognized that disease resistance was often inherited as a single dominant or semi-dominant gene [2]. Since then a substantial amount of knowledge has been accumulated on the genetic basis of disease resistance [3-5]. The breeding for disease resistance followed by the use of resistant cultivars has become a universal strategy to control the crop diseases. Although some forms of disease resistance are genetically simple because they can be explained by simple Mendelian ratios (monogenic), genetically complex forms of disease resistance are rather poorly understood. Most complex disease resistance traits are controlled by multiple loci in contrast to a single locus involved in monogenic simple disease resistance [6]. The phenotypic variation of such a complex disease reaction is usually continuous instead of discrete, and conditioned by allelic variation at several genetic loci, each with a relatively small effect. Therefore, these complex disease resistance traits are measured quantitatively, and they are known as quantitative resistance traits (QRTs). However, lack of discrete phenotypic segregation prevents the use of classical Mendelian techniques for studying the QRTs. Moreover, gene × gene and gene × environment interactions play an important role in the phenotypic expression of QRTs resulting in lower estimates of heritability and a reduced likelihood of appearing Mendelian unless special experimental precautions are followed. Besides, QRTs can be race-specific or race-nonspecific [7]. In other words, the classical quantitative approaches describe the nature of loci involved in resistance phenotypes including the approximate number of loci affecting the resistance trait in a particular mating by studying the properties like average gene action (e.g. additive, non-additive and epistatic gene actions) and the degree to which different polygenes interact with each other and the environment in determining the ultimate phenotype (e.g. genotype × environment interaction). This does not, however, allow to dissect polygenic inheritance into discrete genetic loci or to characterize the roles of individual genes in disease response vis-à-vis resistance.

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In recent years, the availability of numerous molecular markers throughout the genome provides the opportunity to analyze the Mendelian factors determining the quantitative traits localized in quantitative trait loci (QTLs). Molecular markers are heritable entities that are associated with economically important crop traits used by the plant breeders as selection tools [8]. The individual loci controlling a quantitative trait are referred to as QTL. Different alleles at QTL cause genetic differences between individuals and families for quantitative traits [9]. Complex and polygenic forms of characters or traits can be studied by QTL mapping employing DNA markers [10]. It describes the roles of specific loci in genetically complex traits. Actually, the identification of disease resistance QTL is no different from genetic dissection of other quantitative traits vis-à-vis QTLs. Although before the advent of molecular biology some genetic experiments did predict QTL mapping, recent advances in molecular-genetic marker technology have tremendously broadened our understanding of quantitative traits, and provided a greater ability to manipulate them for crop improvement. About twenty years ago, quantitative trait loci (QTLs) were first localized to specific chromosomal regions by characterizing induced resistance mutations using alien addition and chromosome deletion lines [11]. But today, molecular marker technology has facilitated the identification and characterization of QTLs with much ease and alacrity unthinkable at that time. With QTL mapping, the researchers have begun not only to uncover the effect of individual QTLs in the disease response process, but also to identify race-specificity. In addition, QTL mapping has provided the researchers a much better recourse to characterize the interactions between disease resistance genes, plant development and the environment, and to ascertain whether homologous resistance genes exist in related plant taxa. In recent years, it has also opened up possibilities to clone partial resistance genes, which are known only by small and continuous effects on phenotype [12,13]. These all appear to have the potentials for managing complex traits like disease resistance through marker-assisted selection (MAS) and finally map-based cloning of specific genes [14]. These issues will be addressed in this article with current update of the status of QTL mapping for managing quantitative disease resistance in diverse plant taxa. The article would also go in some way to covering the conceptual frameworks of QTL analysis with special reference to their applications in plant breeding.

Quantitative Trait Locus (QTL): An Overview

The Genetic Architecture

The analysis of quantitative traits using a genetical approach rather than a statistical approach has been revolutionized at the end of the eighties [15]. By that time linkage maps were sufficiently saturated with DNA marker loci to use the segregation of the marker alleles in a progeny to correlate with the variation in a trait value in the said progeny. There were significant correlations between the segregation at a certain genetic marker locus and the variation in trait value. These correlations indicate the presence of a quantitative trait locus (QTL) in the proximity of the marker locus. Therefore, a QTL is a map position on the chromosome localized relative to the position of the genetic marker loci. It describes a region of a chromosome that has a significant effect on a quantitative trait. The inheritance and effect of this locus involved in the expression of a quantitative trait can be studied indirectly by studying the inheritance of the alleles at the marker loci. As early as in 1923, Sax [16] reported that a quantitatively inherited trait (seed size) in bean was associated with a discrete monogenic trait (seed coat colour), and this was perhaps the first report of the linkage of the single gene with one or more polygenes. Subsequently, many reports confirmed the existence of linkage between single gene markers and polygenes controlling quantitative variation [17]. These all laid the conceptual basis of QTL mapping on the supposition that if the segregation of simply inherited monogenes could be used to detect a linked polygene, it should be possible to map and characterize all the QTLs affecting a complex trait [18].

The advent of molecular markers, especially the DNA-based genetic markers, initiated the modern QTL mapping. The uniqueness of the DNA-based genetic markers is that defined sequences of DNA act as the linked monogenic markers. Using DNA-based markers, it is possible to map and characterize the polygenes underlying quantitative traits in natural populations. DNA markers can be distinguished from morphological markers in having phenotypic neutrality, much informative polymorphism, abundance, codominance and normally the absence of epistasis or pleitropy. This facilitated a virtually limitless number of segregating DNA markers for use in a single population for mapping polygenes through an entire genome. Clearly this gave the researchers more insights into the chromosomal locations, gene actions, and biological roles of specific loci involved in the expression of complex phenotypes. Modern QTL mapping involves testing DNA-based genetic markers throughout a genome for the likelihood that these markers are associated with a QTL. Individuals in a population are characterized for DNA marker genotypes and the phenotypes of interest, and accordingly they are separated into distinct classes based on marker genotypes. Marker-based localization of QTL requires the preservation of linkage disequilibrium between genetic markers and the QTL in population under investigation. Many authors have examined the theoretical basis of this association between genetic

marker and QTL [19-21]. However, the expected efficiencies of various methods of estimating QTL effects vary considerably [21,22]. In essence, the tests for QTL-trait association can involve the evaluation of one marker at a time, two marker loci simultaneously, or the consideration of all the marker loci at once. The single-marker approach, based on linear model method or one-way analysis of variance, suffers from the main limitation that it ignores the potential recombination between a marker and a QTL leading to an underestimation of QTL effects if the marker and QTL are not coincident [23]. In contrast, interval mapping strategies using maximum likelihood for the analysis of single QTLs flanked by a pair of marker loci are employed for simultaneous examination of two marker loci [20,22]. This approach permits the estimation of QTL effects at any location within a marker interval based on the means and variances observed in the marker classes and the recombination frequency between the markers bracketing a particular interval [20]. In spite of this advantage, interval mapping approach fails to test unlinked markers and to precisely locate QTLs beyond the terminal markers of a given linkage group. However, the consideration of all the marker loci at once involves the regression of trait expression on the values of multiple marker loci [24]. Even interval mapping and multiple regression have been integrated including the inclusion of co-factors to characterize QTL-trait associations more precisely [21,22,25-27]. However, since the value of a quantitative trait displays a continuous distribution, it is affected by a number of genetic factors (multiple alleles and/ or multiple loci), each making its own contribution to the trait value. Further, this trait value is modified by environmental conditions. Therefore, the practical applicability of QTL mapping in plant breeding depends on the ability to detect QTLs and the consistency of those QTLs over generations and environments [28]. The accurate estimates of QTL effects are essential if the goal is to use the information in subsequent selection programme without further validation. It requires that due consideration must be given to probabilities of both Type I (false significance of a locus) and Type II (failure to detect a significant factor) errors. Finally, the correct interpretation is dependent on having fit an appropriate genetic model and may be very complicated and difficult in the case of multiple QTLs in a genomic region [22].

The Genetic Basis of Mapping Population

The use of genetic marker loci to detect polygenes is essentially based on the assumption that there is a linkage disequilibrium (i. e. non-random association of alleles at different loci in a population) between alleles at the marker locus and alleles of the linked polygene (s) [10]. Since linkage disequilibrium due to physical linkage of loci remains at its highest value in populations derived from controlled matings, the ability to map and characterize polygenes using genetic marker loci is maximum in backcross or in F_2/F_3 populations. These populations are most commonly used for detecting linkage between DNA markers and polygenes controlling quantitative traits [15,29]. This type of population derived only from two generations contains segregating linkage blocks providing a basis for QTL mapping. However, the major drawback to F_2 and backcross populations is that they are ephemeral (i.e. seeds derived from selfing these individuals do not breed true). It is also difficult, rather impossible, to measure characters as part of QTL mapping in several locations over several years with F_2 or backcross populations [30]. The use of inbred populations is the best solution because they provide a permanent mapping population, i.e. they are not ephemeral. Recombinant inbred lines (RILs) can be used for detecting linkage between markers and quantitative traits. The RILs are derived from individual F_2 plants through single seed descent over at least five or six generations, and each of these lines contains a different combination of linkage blocks from the original parents. Since the RILs can be grown in replicated trials at several locations over several years, they are ideal for QTL mapping. However, the development of RILs is difficult in obligate outcrossing species where inbreeding is not tolerated. Moreover, the generation of RILs is quite time-consuming in addition to having the genomic regions with a propensity to stay heterozygous longer than expected from theory [31]. In QTL mapping, the size of the population is also very important because the resolution of a map and the ability to determine the order of the genetic marker is mainly dependent on population size. Multiple QTLs on a single linkage group are difficult or impossible to resolve. Therefore, the mapping population must be sufficiently large in order to uncover minor QTLs [20].

The choice of an appropriate mapping population also depends on the type of marker systems used [32]. Maximum genetic information can be obtained by using a codominant marker (i.e. RFLPs) in a classified F_2 population. In contrast, backcross populations can be used for mapping dominant markers (i.e. RAPD) if all the loci in the recurrent parent are homozygous, and the participating donor and recurrent parents have contrasting polymorphic marker alleles [33]. However, the genetic information obtained from backcross populations using either codominant or dominant markers is less than that obtained from F_2 populations. This is because in backcross populations only one recombinant gamete is sampled per plant in contrast to two gametes in F_2 populations. In RILs, the dominant markers provide as much information as codominant

markers. Using RILs or doubled haploids, the information obtained from dominant markers can be maximized because of the putative homozygosity at all the loci. But at lower marker saturation, backcross populations are more informative than the RILs because the distance between linked loci increases in the latter. The use of heterogeneous source populations as parents for marker-based QTL analysis is less informative than populations originated from a single pair of inbred parents due to ambiguous allelic sources and variable linkages between marker alleles and the alleles at an adjoining QTL within each population [34]. In complex disease reaction where the expression is controlled by QTLs, segregation data from progeny test populations derived from F_2 individuals (i.e. F_3 or F_2BC) are often used in map construction as these populations exhibit maximum linkage disequilibrium. In bulked segregant analysis [35], two bulked DNA samples are drawn from a segregating population derived from a single cross, and they are screened for DNA polymorphisms and compared against a randomized genetic background of unlinked loci. The differences between the two bulks indicate markers that are linked to a particular trait. Since all loci identified by bulked segregant analysis segregate and can be mapped, it eliminates the problem of linkage drag usually associated with nearly isogenic lines (NILs). Together with the bulked segregant analysis (BSA)-derived AFLPs, the microsatellite markers identified a major QTL for yellow leaf spot resistance in wheat contributing up to 39 % of total phenotypic variation [36]. The types of mapping populations to be employed for QTL mapping are a function of the reproductive characteristics of the crop species and the ingenuity of the researcher [10]. For example, in a self-pollinated crop the degree of inbreeding may be important in deriving the most useful QTL estimates. However, if epistasis is important, the evaluation of derived lines which have undergone more inbreeding may be desirable because it would produce few intralocus interactions and higher frequencies of interpretable additive by additive interactions than progenies which exhibit greater heterozygosity [34].

Quantitative Trait Loci (QTLs) for Disease Resistance

The Analysis of Disease Resistance QTLs

The analysis of QTL for disease resistance attempts to indicate the number and effects of genetic factors controlling quantitative resistance. The number of QTLs identified ranges from 2 to several (> 10), but usually only few loci (3-5) have been shown to control the majority of genetic variation contributing to resistance phenotypes (Table 1 [37-39]). However, in some cases, only one or two QTLs have been identified to control the expression of resistance phenotypes (Table 1 [37,40]). Michelmore [4] concluded that this type of quantitative resistance, where only one or two QTLs are involved to produce a resistant phenotype, should be considered as oligogenic rather than polygenic. For example, a major QTL such as *Grp1* that is located in the resistance hotspot on potato chromosome V might be, in fact, a single gene [41]. However, in several cases where very few QTLs have been identified, either the sizes of the populations were too small or the number of informative markers used for genome analysis (coverage) was rather limited (Table 1). As has been described earlier, a typical polygenic character like complex disease resistance assumes the involvement of many minor genes, each having approximately equal effect on phenotype. The identification of only one or two QTLs contributing significantly to the expression of a resistance phenotype may be tempting to speculate that the resistance mechanism in those cases is oligogenic [4], but it needs to be emphasized here that the QTL analysis does not necessarily exclude the possibility of the presence of minor genes that were below the threshold of significance for their accurate detection in the experiment. In addition, the borderline between a single QTL with large effect and multiple QTLs with smaller effects is rather difficult to distinguish.

Interactions between Disease Resistance QTLs

QTLs for disease resistance exhibit a variety of gene actions-additive [42], dominant or overdominant [43] and even recessive [44]. QTLs have also been shown to exhibit significant epistatic and environmental interactions. In the study of bacterial canker (*Clavibacter michiganensis* subsp. *michiganensis*) resistance in Lycopersicon hirsutum, two QTLs (*Rcm* 2.0 and *Rcm* 5.1) have been shown to exhibit epistatic interactions by ANOVA and orthogonal contrasts, suggesting that resistance was determined by additive gene action and an additive-by-additive epistatic interaction; a replicated trial using the diallel population confirmed further the presence of additive-by-additive epistasis [45]. However, genotype × environment interactions play a significant role in the stability of individual QTLs over repeated analyses. The DNA markers which can explain a significant portion of the resistance trait variance are considered to be closely linked to the QTL. But, due to possible genotype by environment interaction, the results need to be verified by repeating the experiment under more than one set of environmental conditions or in different years. Those QTLs, which cannot be detected in all years or locations indicate the presence of genotype × environment interaction. For resistance to northern leaf blight in maize, Dingerdissen *et al.* [46] showed that QTLs on chromosomes 3L, 5S, 7L and 8L were

Table 1. Characterization of quantitative trait loci (QTLs) determining polygenic pathogen and pest disease resistance in plants

Host	Disease	Pathogen/ pest	Mapping population (type)[a]	Molecular marker/ No. of markers	No. of QTL[b]/ Effects[c]	Reference (s)
Apple (*Malus* × *domestica*)	Fire blight	*Erwinia amylovora*	Fiesta × Discovery or Prima (F_1)	-/ -	1/ 34.3-46.6	[94]
	Powdery mildew	*Podosphaera leucotricha*	Idared × U211	AFLP, SSR/-	10/ 48-72	[95]
Barley (*Hordeum vulgare*)	Barley yellow dwarf	Barley yellow dwarf virus (BYDV)	Post × Vixen (Ryd 2) and Post × Nixe (DH)	AFLP, RAPD, SSR/ -	2/ 47	[96]
	Fusarium head blight	*Fusarium graminearum*	Chevron × Stander (DH)	RFLP/-	3/ -	[97]
		Fusarium graminearum	Fredrickson × Stander (SP)	SSR/ 143	3/ -	[98]
		Fusarium graminearum	Zhedar 2 × ND 9712 × Foster (DH)	RFLP, SSR, AFLP/ 214	14/ -	[90]
		Fusarium graminearum	Russia 6 × H. E. S. 4 (RI)	RGA, EST, AFLP/ -	3/ -	[99]
	Leaf rust	*Puccinia hordei*	L 94 × Vada (RIL)	AFLP/ 561	8/ 55-60	[100]
		Puccinia hordei	Vada × L 94 (RIL)	AFLP/ -	6/ -	[101]
		Puccinia recondita	HOR 1063 × Krona (DH)	RFLP/ -	4/ 96.1	[102]
		Puccinia hordei	L 94 × 116-5 (RIL)	AFLP/ -	13/ 35-42	[103]
		Puccinia hordei	Near-isogenic lines (NIL)	AFLP/ -	1/ -	[104]
		Puccinia hordei	L 94 × Vada (RIL)	AFLP/ -	3/ -	[105]
		Puccinia hordei	Sloop × Halcyon (DH)	AFLP, RFLP, SNP, SSR/ 257	8/ 9-85	[106]
		Puccinia hordei	F_2-*Hordeum chilense* accessions (SP)	AFLP, RFLP, SCAR, SSR, STS, Seed storage protein/ 437, 13, 4, 9, 1, 2	5/ -	[107]
	Leaf scald	*Rhynchosporium secalis*	Ingrid × Abyssinian (DH)	AFLP, RFLP, SSR, STS/ -	2+/ -	[108]
		Rhynchosporium secalis	Sloop × Halcyon (DH)	AFLP, RFLP, SNP, SSR/ 257	8/ 9-85	[106]
	Leaf stripe	*Pyrenophora graminea*	Proctor × Nudinka (DH)	RFLP/ -	4/ 29.3-58.5	[109]
	Net blotch	*Pyrenophora teres*	Sloop × Halcyon (DH)	AFLP, RFLP, SNP, SSR/ 257	8/ 9-85	[106]
	Powdery mildew	*Erysiphe graminis*	DH	RFLP/ 155	2/ -	[56]
		Erysiphe graminis	F_1 diallele	RFLP/ 61	5-6/ -	[43]
		Erysiphe graminis	Sloop × Halcyon (DH)	AFLP, RFLP, SNP, SSR/ 257	8/ 9-85	[106]
	Spot blotch	*Cochliobolus sativus*	Harrington × Morex (DH)	-/ -	3/ 30	[110]
	Stripe rust	*Puccinia striiformis f. sp. hordei*	DH	RFLP/ 78	2/ -	[69]
	Yellow mosaic	Barley yellow mosaic virus (BYMV)	Ko A × Mokusekko 3 (SP)	Isozyme/ 100	3/ 55-57 %	[111]
Cabbage (*Brassica oleracea*)	*Agrobacterium tumefaciens*	*Agrobacterium tumefaciens*	DH	RFLP/ -	1/ -	[112]
	Clubroot	*Plasmodiophora brassicae*	F_2/ F_3 population (SP)	RFLP/ 198	2/ 15 + 58	[113]
		Plasmodiophora brassicae	DH	AFLP, RFLP/ 92	3/ 60	[114]
		Plasmodiophora brassicae	Cabbage × Kale (HP)	RAPD, RFLP/ 99, 21	1/ 30	[115]

Host	Disease	Pathogen/ pest	Mapping population (type)[a]	Molecular marker/ No. of markers	No. of QTL[b]/ Effects[c]	Reference (s)
Cassava *(Manihot esculenta)*	Bacterial blight	*Xanthomonas axonopodis*	TMS 30572 × CM 2177-2 (SP)	RFLP/-	8/ -	[116]
Chickpea (*Cicer arietinum*)	Ascochyta blight	*Ascochyta rabiei*	FLIP 84-92C × PI 599072 (RIL)	ISSR, RAPD/ 116	2/ 45-50.3	[117]
		Ascochyta rabiei	Intraspecific population (SP)	RGA, STMS/ -	6/ -	[93]
		Ascochyta rabiei	*Cicer arietinum* × *C. reticulatum* (RIL)	STMS/ 312	3/ -	[118]
Citrus (*Citrus aurantium*)	Tristeza viral disease	*Citrus tristeza virus* (CTV)	*Citrus aurantium* × *Poncirus trifoliata* (HP)	SSR, IRAP/ 157, 63	8/ -	[80]
Cocoa (*Theobroma cacao*)	Phytophthora	*Phytophthora palmivora*, *P. megakarya*, *P. capsici*	*Theobroma cacao* (HP)	AFLP, SSR/ 190, 23	6/ 11.5-27.5	[119]
	Witches broom	*Crinipellis perniciosa*	Scavina-6 × ICS-1 (SP)	AFLP, RAPD/ 124, 69	1/ 35	[120]
		Crinipellis perniciosa	Sca-6 × ICS-1 (F_2)	SSR, RGH, WRKY genes/182	2/ -	[121]
Common bean *(Phaseolus vulgaris)*	Ashy stem blight	*Macrophomina phaseolina*	Dorado × XAN 176 (RIL)	RAPD/165	5/ 13-19	[122]
	Bacterial brown spot	*Pseudomonas syringae pv. syringae*	Belneb RR-1 × A-55 (RIL)	RAPD/ -	1/ -	[123]
	Bean golden mosaic	Bean golden mosaic bigeminivirus	Dorado × AN 176 (RIL)	RAPD/101	9/ 60	[124]
	Common bacterial blight	*Xanthomonas campestris*	F_2/ F_3 population (SP)	RFLP/ 152	4+/ 17-32	[44]
		Xanthomonas campestris	BAC6 × HT 7719 (RIL)	RAPD/ 84	6/ 14-34	[125]
		Xanthomonas campestris pv. phaseoli	Dorado × AN 176 (RIL)	RAPD/ 101	9/ 60	[124]
		Xanthomonas campestris pv. phaseoli	PC 50 × XAN 159 (RIL)	RAPD/ 181	4/ 18-53	[126]
		Xanthomonas campestris pv. phaseoli	Belneb RR-1× Black A55 (SP+RIL)	RAPD/ 87	3/ 44	[127]
		Xanthomonas campestris pv. phaseoli	BAC 6 × HT 7719 (RIL)	RAPD/ -	3/ -	[128]
		Xanthomonas campestris pv. phaseoli	PC-50 × XAN-159 (IBP) and Chase × XAN-159 (SP)	RAPD/ -	6/ 22-61	[129]
		Xanthomonas axonopodis pv. phaseoli	OAC Seaforth × OAC 95-4 (SP)	AFLP, RAPD, RFLP, SSR/ -	3/ 68.4	[130]
		Xanthomonas campestris pv. phaseoli	Montana No. 5 × Othello, Montana No. 5 × GN 1 Selection 27 (SP)	SCAR/ 3	1/ -	[131]
	Common mosaic	Bean common mosaic virus (BCMV)	Belneb RR-1× Black A55 (SP+RIL)	RAPD/ 87	3/ 44	[127]
	Fusarium wilt	*Fusarium oxysporum* f.sp. *phaseoli*	Belneb RR-1× A 55 (RIL)	RAPD/ -	1/ 63.5	[132]
	Halo blight	*Pseudomonas syringae* pv. *psaseolicola*	Belneb RR-1× Black A55 (SP+RIL)	RAPD/ 87	3/ 44	[127]
	Rust	*Uromyces appendiculatus*	BAC6 × HT 7719 (RIL)	RAPD/ 84	6/ 14-34	[125]
		Uromyces appendiculatus	Dorado × XAN 176 (RIL)	RAPD/ 165	5/ 13-19	[122]
	Web blight	*Thanatephorus cucumeris*	BAC6 × HT 7719 (RIL)	RAPD/ 84	6/ 14-34	[125]
		Thanatephorus cucumeris	Dorado × XAN 176 (RIL)	RAPD/ 165	5/ 13-19	[122]
	White mold	*Sclerotinia sclerotiorum*	PC-50 × XAN-159 (RIL)	RAPD/ -	9/ -	[133]

Host	Disease	Pathogen/ pest	Mapping population (type)[a]	Molecular marker/ No. of markers	No. of QTL[b]/ Effects[c]	Reference (s)
Cotton (*Gossypium hirsutum*)	Verticillium wilt	*Verticillium dahliae*	*Gossypium barbadense* cv. Prima S-7 × *G. hirsutum* cv. Acala 44(IC)	SSR/ 60	3/ -	[134]
Grape (*Vitis vinifera*)	Fungal disease	*Plasmopara viticola, Uncinula necator*	Regent × Lemberger (HP)	AFLP, RAPD, SSR, SCAR/ 185, 137, 85, 22	1+/ -	[135]
	Powdery mildew	*Uncinula necator*	Horizon × Illinois 547-1 (SP)	AFLP, RAPD/ 1, 1	1/ 41	[136]
Lettuce (*Lactuca sativa*)	Downy mildew	*Bremia lactucae*	*Lactuca saligna* × *L. sativa* (HP)	AFLP/ -	3/ 51	[137]
Lodge pole pine (*Pinus contorta* spp. *latifolia)*	Western gall rust	*Endocronartium harknessii*	OP	RAPD/ 148 + 77	3/ -	[138]
Maize (*Zea mays*)	Anthracnose stalk rot	*Colletotrichum graminicola*	F_2/ F_3 population	RFLP/ 113	1/ 16-75	[139]
	Common rust	*Puccinia sorghi*	(BS 11 (Fr) C7) × FrMo 17 (SP)	RFLP/ 146	11/ 22 (rust) and 14/ 13.1-16.2 (smut)	[140]
		Puccinia sorghi	IL 731a × W 6786 (SP)	RFLP/ -	3/ 5.9-18	[141]
	Ear rot	*Fusarium moniliforme*	F_2/ F_3 population (SP)	RAPD, RFLP/ 19, 95	4-5/ -	[142]
		Fusarium moniliforme	F_2 from inbred lines (SP)	RFLP/ 149 + 106	9 + 7/ 30-44 + 11-26	[143]
		Fusarium graminearum	CO387 × CG62	-/ 162	11/ 6.7-37	[144]
	European corn borer	*Ostrinia nubilalis*	F_2/ F_3 population	RFLP/ 87	7/ -	[145]
	Gray leaf spot	*Cercospora zeae-maydis*	F_2/ F_3 population	RFLP/ 87	9/ 4-26	[42]
		Cercospora zeae-maydis	B73 × Va14 (SP)	RFLP/ 78	4/ 44-68	[146]
		Cercospora zeae-maydis	FR 1141 × 061 (HP)	RFLP/ 86	11/ 51-58.7	[147]
		Cercospora zeae-maydis	RIL	AFLP, RFLP, SSR/ 11 + several	2/ 37	[49]
		Cercospora zeae-maydis	V0613Y × Pa405 (SP)	RFLP, SSR/ 50, 47	2/ 40-47	[148]
	Java downy mildew	*Peronosclerospora maydis*	Ki 3 × CML 139 (RIL)	RFLP, SSR/ -	6/ 26-57	[87]
	Maize streak	Maize streak monogeminivirus (MSV)	CML 202 × Lo 951 (SP)	RFLP/ 110	4/ 59	[149]
		Maize streak monogeminivirus (MSV)	CIRAD 390 × B 73 (SP)	RFLP/ 124	8/ 45	[150]
	Northern corn leaf blight	*Exserohilum turcicum*	F_2/ F_3 population	RFLP/ 103	7/ 7-18	[151]
		Setosphaeria turcica	Mo17 × B52 (SP)	RFLP/ 112	8/ 10-48	[46]
		Setosphaeria turcica	Lo 951 × CML 202 (SP)	RFLP/ 110	8/ 52	[152]
		Setosphaeria turcica	Lo 951 × CML 202 (SP)	RFLP/ 110	19/ 71	[153]
		Setosphaeria turcica	D32 (dent) × D145 (flint) (F_3)	RFLP, SSR/ 87, 7	13/ 48-62	[154]
		Exserohilum turcicum	IL 731a × W 6786 (SP)	RFLP/ -	3/ 5.9-18	[141]
	Rajasthan downy mildew	*Peronosclerospora heteropogoni*	Ki 3 × CML 139 (RIL)	RFLP, SSR/ -	6/ 26-57	[87]
	Smut	*Ustilago maydis*	Elite flint inbreds A, B, C and D	RFLP/ 89-151	19/ 39-58	[155]
		Ustilago maydis	(BS 11 (Fr) C7) × FrMo 17 (SP)	RFLP/ 146	11/ 22 (rust) and 14/ 13.1-16.2 (smut)	[140]

Host	Disease	Pathogen/ pest	Mapping population (type)[a]	Molecular marker/ No. of markers	No. of QTL[b]/ Effects[c]	Reference (s)
	Sorghum downy mildew	*Peronosclerospora sorghi*	G 62 × G 58 (RIL)	RFLP/ 106	3/ 12.4-23.8	[156]
		Peronosclerospora sorghi	Ki 3 × CML 139 (RIL)	RFLP, SSR/ -	6/ 26-57	[87]
	Sporisorium	*Sporisorium reiliana*	D 32 × D 145 (F_3)	RFLP, SSR/ 87, 7	11/ 13-44	[157]
	Stalk rot	*Gibberella zeae*	F_2/ F_3 population	RAPD, RFLP/ 19, 95	4-5/ -	[142]
	Stewart's wilt	*Erwinia stewartii*	Hi31 × Ki14 (RIL)	RFLP/ 127	2/ -	[158]
		Erwinia stewartii	IL 731a × W 6786 (SP)	RFLP/ -	3/ 5.9-18	[141]
	Sugarcane mosaic	Sugarcane mosaic virus	D 32 × D 145 (SP)	RFLP, SSR/ 87, 7	7/ 70-77	[159]
		Sugarcane mosaic virus	F 7 × FAP 1360A (F_3)	SSR/ 4	2/ 15 + 62	[160]
		Sugarcane mosaic virus	HangZao4 × Ye 107 (SP)	SSR/ 65	3/ 30.2	[161]
		Sugarcane mosaic virus	Huangzao 4 × Ye 107 (SP)	SSR/ 89	12/ 7.2-26.9	[162]
Melon (*Cucumis melo*)	Powdery mildew	*Sphaerotheca fuliginea*	PMAR No. 5 × Harukei 3 (SP)	AFLP, RAPD/ 21, 74	18/ -	[163]
Mungbean (*Vigna radiata*)	Powdery mildew	*Erysiphe polygoni*	F_2/ F_3 population	RFLP/ 141	3/ -	[37]
		Erysiphe polygoni	VC 1210A × TC 1966 (SP)	AFLP, RFLP/ -	1/ 64.9	[164]
		Erysiphe polygoni	Berken × ATF 3640 (RIL)	RFLP/ 51	1/ 86	[165]
Muscadine grape (*Vitis rotundifolia*)	Downy mildew	*Plasmopara viticola*	BC2-segregating population (SP)	ISSR, RAPD, SSR/ 13, 151, 208	1/ 73	[166]
Pea (*Pisum sativum*)	Blight	*Ascochyta pisi*	F_2 population	RFLP/ 56	4/ -	[167]
	Root rot	*Aphanonyces euteiches*	Puget × 90-2079 (RIL)	AFLP, ISSR, RAPD, SSR, STS/ 324	7/ 11-47	[168]
		Aphanonyces euteiches	Puget × 90-2079 (RIL)	AFLP, ISSR, RAPD, SSR, STS/ -	10/ -	[169]
Peach (*Prunus persica*)	Powdery mildew	*Sphaerotheca pannosa*	*Prunus persica* cv. Summergrand × *P. davidiana* clone 1908 (HP)	Isozyme, RAPD/ 1, 99	6 + 3/ -	[170]
Pearl millet (*Pennisetum glaucum)*	Downy mildew	*Sclerospora graminicola*	F_2/ F_4 population	RFLP/ 22	5/ 8-48	[51]
		Sclerospora graminicola	*Pennisetum glaucum* F_2 progeny (SP)	RFLP/ -	2/ 60	[171]
Pepper (*Capsicum annum*)	Cucumber mosaic	*Cucumber mosaic virus (CMV)*	Perennial × Yolo Wonder (DH)	RAPD, RFLP/ 138	3/ 57	[172]
		Cucumber mosaic virus (CMV)	Maor × Perennial (F_3)	AFLP, RFLP/ -	4/ 16-33	[173]
	Phytophthora	*Phytophthora capsici*	*Capsicum annuum* and *C. chinense*	SCAR/ 1	1/ -	[174]
Potato (*Solanum tuberosum*)	Late blight	*Phytophthora infestans*	F_1 population (HP)	RFLP/ 29	13/ -	[53]
		Phytophthora infestans	*Solanum tuberosum* × *S. phureja* (HP)	AFLP, RFLP, SSR/-	6/ 11-43	[175]
		Phytophthora infestans	*Solanum paucissectum*	Conserved sequences from potato and tomato	1/ 25	[176]

Host	Disease	Pathogen/ pest	Mapping population (type)[a]	Molecular marker/ No. of markers	No. of QTL[b]/ Effects[c]	Reference (s)
		Phytophthora infestans	*Solanum phureja* × *S. stenotomum* (full-sib progenies)	RFLP/ 162	3/ 10-23	[177]
	Potato cyst nematode	*Globodera rostochiensis*	F_1 population	RFLP/ 29	2/ 22	[40]
	Potato leaf roll	Potato leaf roll virus (PLRV)	F_1 *Erwinia* population (DG83-2025 × DG81-68)	SCAR/ -	3/ 50-60	[60]
	Stem blackleg or tuber soft rot	*Erwinia carotovora* ssp. *atroseptica*	*Solanum tuberosum* × *S. chacoense* and *S. tuberosum* × *S. yungasense* (HP)	AFLP, RFLP/ -	1+/ -	[178]
Radiata pine (*Pinus radiata*)	Needle blight	*Dothistroma septospora*	31053 or 31032 or 31053 × 31032 (OP)	RFLP, SSR/ 250	4/ 12.5	[179]
Rapeseed (*Brassica napus*)	Balckleg	*Leptosphaeria maculans*	*B. napus* cvs Cresor and Westar (DH)	RFLP/ 175	2/ -	[180]
		Leptosphaeria maculans	Darmor-bzh × Yudal (DH)	RAPD, RFLP/ 288	23/ 29-57	[181]
		Leptosphaeria maculans	Darmor × Samourai (SP)	RAPD, RFLP/ 338	10/ 36-42	[182]
	Light leaf spot	*Pyrenopeziza brassicae*	Darmor-bzh × Yudal (DH)	RAPD, RFLP/ 288	23/ 29-57	[181]
	Sclerotinia stem rot	*Sclerotinia sclerotiorum*	H 5200 × Ning RS-1 (SP)	AFLP, RAPD, RFLP, SSR/ 30, 2, 72, 3	6/ 40.7	[183]
	White rust	*Albugo candida*	Per × R 500 (RIL)	RFLP/ 144	2/ -	[184]
Rice (*Oryza sativa*)	Bacterial blight	*Xanthomonas oryzae*	Lemont × Teqing (RIL)	RFLP/ 186	10/ -	[185]
		Xanthomonas oryzae	Lemont × Teqing (RIL)	RFLP/ 182	10/ -	[55]
		Xanthomonas oryzae	IR 64 × Azucena (DH)	PCR-RGA, RFLP/ 118	11/ 11.3-20.6	[85]
	Blast	*Magnaporthe grisea*	RIL	RFLP/ 127	10/ 19-60	[57]
		Magnaporthe grisea	Nipponbare × Owarihatamochi (F_4)	RFLP, SSR/ -	4/ 66.3	[61]
		Magnaporthe grisea	Kahei × Koshihikari (SP)	RFLP/ -	2/ 71	[186]
		Magnaporthe grisea	Lemont × Teqing (RIL)	RFLP/ 175	9/ 43-53	[187]
		Magnaporthe grisea	Norin 29 × Chubu 32 (F_3)	RFLP/ 36	1/ 45.6	[188]
		Magnaporthe grisea	IR 64 × Azucena (DH)	PCR-RGA, RFLP/ 118	11/ 11.3-20.6	[85]
	Brown plant hopper	*Nilaparvata lugens*	Lemont × Teqing (RIL)	RFLP/ -	7+/ 70	[189]
		Nilaparvata lugens	IR 64 × Azucena (DH)	PCR-RGA, RFLP/ 118	11/ 11.3-20.6	[85]
	Rice yellow mottle	*Rice yellow mottle virus (RYMV)*	IR 64 × Azucena/ IRAT 177 × Apura (DH)	RAPD, RFLP/ -	1/ -	[190]
		Rice yellow mottle virus (RYMV)	Azucena × IR 64 (SP)	RFLP, SSR/ -	2/ -	[191]
	Sheath blight	*Rhizoctonia solani*	Lemont × Teqing (RIL)	RFLP/ 113	6/ 60	[192]
		Rhizoctonia solani	IR 64 × Azucena (DH)	PCR-RGA, RFLP/ 118	11/ 11.3-20.6	[85]
	Stripe	Rice stripe virus (RSV)	URK 72 × Nipponbare (SP)	RFLP, SSR/ -	2/ -	[193]
Ryegrass (*Lolium perenne* L.)	Gray leaf spot (GLS)	*Magnaporthe grisea*	Italian × perennial (HP)	-/ -	3/ 20-37	[194]
	Crown rust	*Puccinia coronata*	Susceptible × Resistant lines (HP)	AFLP, SSR, RFLP, STS/ 227	4/ 2.6-12.5	[195]

Host	Disease	Pathogen/ pest	Mapping population (type)[a]	Molecular marker/ No. of markers	No. of QTL[b]/ Effects[c]	Reference (s)
Snap bean (*Phaseolus vulgaris*)	White mold	*Sclerotinia sclerotiorum*	Benton × NY 6020-4 (RIL)	RAPD/ 27	2/ 12-38	[196]
Soybean (*Glycine max*)	Brown stem rot	*Phialophora gregata*	BSR 101 × PI 437.654 (RIL)	AFLP, RFLP/ 760, 146	2/ -	[197]
	Bud blight	Tobacco ring spot virus (TRSV)	Young × PI 416937	RFLP, SSR/ -	3/ 82	[198]
	Root rot	*Phytophthora sojae*	Conrad × Sloan, Conrad × Harosoy and Conrad × Williams (RIL)	SSR/ -	2/ 10.6-35.6	[199]
	Peanut root-knot nematode	*Meloidogyne arenaria*	PI 200538 × CNS (SP)	RFLP/ 130	2/ 51	[200]
	Soybean cyst nematode	*Heterodera glycines*	F_2/ F_3 population (SP)	RAPD, RFLP/ 7, 36	3/ 21-40	[201]
	Sudden death syndrome	*Fusarium solani*	Forrest × Essex (RIL)	RAPD/ 4	4/ 65	[202]
		Fusarium solani	Forrest × Essex (RIL)	RAPD/ 70	2/ 34	[203]
	White mold	*Sclerotinia sclerotiorum*	Williams 82 × (Vinton 81, Corsoy 79, Dassel, DSR 173, S 19-90) (RIL)	SSR/ 507	28/ 4-10	[204]
Sugar beet (*Beta vulgaris*)	Cercospora leaf spot	*Cercospora beticola*	F_2 population (SP)	AFLP, RFLP/ 221, 46	5/ 7-18	[205]
		Cercospora beticola	F_2 population (SP)	AFLP, RFLP, SCAR, SSR/ 224	4/ -	[206]
		Cercospora beticola	93164P × 95098P (SP)	AFLP, RFLP/ -	4/ -	[207]
Sugarcane (*Saccharum officineraum*)	Yellow spot	*Mycovellosiella koepkei*	M 596/ 78 × M 937/ 77 (SP)	RAPD/ 134	4/ 20-27	[208]
Sunflower (*Helianthus annuus*)	White mold	*Sclerotinia sclerotiorum*	–	RFLP, Isozyme/ -	2/ 38	[209]
	Midstalk rot	*Sclerotinia sclerotiorum*	TUB-5-3234 (IS)	SSR/ 78	3-4/ 40.8-72.7 (genotypic)	[210]
Tobacco (*Nicotiana tabacum*)	Bacterial wilt	*Ralstonia solanacearum*	W6 × Michinoku 1 (DH)	AFLP/ 117	1/ 30	[211]
Tomato (*Lycopersicon esculentum*)	Bacterial canker	*Clavibacter michiganensis* ssp. *michiganensis*	*Lycopersicon hirsutum* LA407 × *L. esculentum* (IBP)	PCR/ -	2/ 56-73	[212]
	Bacterial wilt	*Pseudomonas solanacearum*	F_2 population (SP)	RAPD, RFLP/ 12, 67	3/ 24-77	[38]
		Pseudomonas solanacearum	Hawaii 7996 × WVa 700 (SP)	RFLP/ 462	4/ 30-56	[213]
	Black mold	*Alternaria alternata*	*Lycopersicon esculentum* × *L. cheesmanii* (BP)	PCR, RFLP/ -	1/ -	[214]
	Early blight	*Alternaria solani*	*Lycopersicon esculentum* × *L. hirsutum* (BP)	RFLP, RGA/ 41, 23	10/ 8.4-25.9	[92]
		Alternaria solani	*Lycopersicon esculentum* × *L. hirsutum* (BP)	RFLP, RGA/ 145, 34	7/ 45-81	[162]
	Late blight	*Phytophthora infestans*	CLN 657 × L 3708 (SP)	AFLP, PCR, RFLP/ 120	1/ 71.4	[48]
	Powdery mildew	*Oidium lycopersici*	*Lycopersicon esculentus* cv. Money maker × *L. parviflorum* G1.1601 (SP)	AFLP/ -	3/ 68	[215]
Water yam (*Dioscorea alata*)	Anthracnose	*Colletotrichum gloeosporioides*	TDa95/ 00328 × TDa87/ 01091 (HP)	AFLP/ 469	1/ 10	[216]
Wheat (*Triticum aestivum*)	Blotch	*Stagonospora nodorum*	Liwilla × Begra (DH)	SSR/ 240	4/ 16-37	[217]

Host	Disease	Pathogen/ pest	Mapping population (type)[a]	Molecular marker/ No. of markers	No. of QTL[b]/ Effects[c]	Reference (s)
	Chlorosis	*Pyrenophora tritici-repentis*	W-7984 × Opata 85 (RIL)	RFLP/ 542	1/ 26.1	[218]
	Crown rot	*Fusarium pseudograminearum*	2-49 × Janz (DH)	-/ -	5/ 9-21	[219]
	Fusarium head blight	*Fusarium graminearum*	Fukuho-komugi × Oligo culm (DH)	RAPD/ 65	1/ -	[220]
		Fusarium culmorum	Hussar × G 16-92 and Capo × SVP 72017-17-5-10-1 (NIL)	AFLP/ 500-600	Several/ -	[221]
		Gibberella zeae	CM-82036 × Remus (DH)	AFLP, SSR/ -	3/ 60	[222]
		Gibberella zeae	LDN (Dic-3A) (RICL)	SSR/ 19	1/ 37	[223]
		Fusarium graminearum	Sumai-3 (NIL)	SSR/ -	1/ -	[224]
		Fusarium graminearum	Huapei 57-2 × Patterson (RIL)	SSR/ -	4/ -	[225]
		Gibberella zeae	CM-82036 × Remus (DH)	SSR/ -	2/ 20-29	[226]
		Fusarium culmorum	Renan × Recital (RIL)	AFLP, RFLP, SSR/ -	9/ 30-45	[227]
		Gibberella zeae	Ning 7840 × Clark (RIL)	AFLP, STS/ 6	1/ 38-50	[228]
		Gibberella zeae	Sumai 3 × Stoa (HP)	STS/ 68	1/ -	[229]
		Gibberella zeae	Sumai 3 × Alondra, 894037 × Alondra and Wangshuibai × Alondra (RIL)	SSR/ 9	7/ 47.4	[230]
		Fusarium graminearum	Ning 894037 × Alondra (RIL)	SSR/ -	3/ 51.6	[231]
		Gibberella zeae	Wuhan-1 × Maringa (DH)	SSR/ 328	4/ 32	[232]
		–	Chokwang × Clark (RIL)	SSR, TRAP/ 172	3/ -	[233]
		Fusarium graminearum and *F. culmorum*	Dream × Lynx (RIL)	AFLP, SSR/ -	4/ 11-21	[234]
	Leaf rust	*Puccinia recondita*	Parula × Siete Cerros (RIL)	RAPD/ 3	3/ -	[235]
		Puccinia recondita f. sp. tritici	Fukuho-komugi × Oligo culm (DH)	RAPD, RFLP, SSR/ 443	2/ 11.3-40.1	[236]
		Puccinia tritici	Fukuho-komugi × Oligo culm (DH)	SSR/ -	4/ -	[237]
	Powdery mildew	*Erysiphe graminis*	*Triticum aestivum* cv. Forno × *T. spelta* cv. Oberkulmer (RIL + SP)	RFLP, SSR/ 126, 9	18/ 77	[238]
		Blumeria graminis f. sp. tritici	RE 714 × Hardi (F_3)	SSR/ -	1/ 16.8-25.3	[239]
		Blumeria graminis	Massey × Becker (SP)	RFLP, SSR/ 213, 139	3/ 11-29	[240]
	Scab	*Venturia inaequalis*	Ning 7840 × Clark (RIL)	AFLP, SSR/ -, 18	3+/ -	[241]
		Venturia inaequalis	Ning 7840 × Clark (RIL)	SSR/ -	1/ -	[242]
	Septoria tritici blotch	*Mycosphaerella graminicola*	Savannah × Senat (DH)	AFLP, SSR/ 76, 244	6/ 18.2-67.9	[243]
	Stagonospora glume blotch	*Stagonospora nodorum*	Arina × Forno (SSD)	SSR/ -	2/ 31.2	[244]
	Stripe rust	*Puccinia striiformis*	Fukuho-komugi × Oligo culm (DH)	SSR/ -	4/ -	[237]
	Tan spot	*Pyrenophora tritici-repentis*	W-7984 × Opata 85, W-7976 × Trenton (RIL)	RFLP/ extensive	1/ 26-64	[245]
		Pyrenophora tritici-repentis	Grandin × BR34 (RIL)	-/ -	2/ 13-41	[246]
	Yellow leaf spot	*Pyrenophora tritici-repentis*	Krichauff × Brookton and Cranbrook × Halbred (DH)	AFLP, SSR/ 3	1/ 39	[36]

[a] BP = Backcross progenies; DH = Double haploids; HP = Hybrid progenies; IBP = Inbred backcross progenies; IS = Interspecific cross; NIL = Near-isogenic lines; OP = Open pollinated populations; RICL= Recombinant inbred chromosomal lines; RIL = Recombinant inbred lines; SP = Segregating populations; SSD = Single seed descent

[b] The number of significant QTLs above the LOD threshold value; [c] The percentage of the phenotypic variance explained by the QTLs in the experiment

significant across environments but all other QTLs were affected by a large genotype × environment interaction. However, with high LOD (i. e. logarithms of odds ratio) values, QTLs are usually stable across the environments because when the LOD threshold is raised, fewer markers are assigned to linkage groups (i.e. independent loci), and more and smaller linkage groups are identified. For example, QTLs detected using the interval mapping method at a LOD threshold of 3.0 for resistance against *Sclerotinia sclerotiorum* and *Diaporthe helianthi* in sunflower were reported to be stable over the years, i. e. they were mapped in the same chromosomal regions repeated over three years of study [47]. A major QTL with an LOD score of 18.41 for late blight resistance in tomato was found at RFLP marker TG591, which accounted for about 71.4% of the variance [48]. Similarly, a QTL on chromosome 1 in maize for gray leaf spot resistance with a LOD score of 21 was consistent in two F_2 populations over consecutive years [49].

Race-Specificity of Disease Resistance QTLs

Understanding the genetic architecture of QTLs helps not only to ascertain whether individual QTLs are race-specific or race-nonspecific, but also to test the hypothesis that the QTLs are variants of qualitative resistance loci that have been overcome by their respective pathogen [50]. Although partial resistance genes are thought to be generally race-nonspecific, QTLs can be race-specific or race-nonspecific. All the QTLs for resistance to downy mildew in pearl millet were race-specific [51]. Recently, the race specificity of QTLs for partial resistance to blast disease in rice was tested by using isolates for which no major resistance gene segregated in a mapping population [52]. RILs were repeatedly inoculated with blast isolates CD100, CM28 and PH19, and scored for lesion type, lesion size and number of lesions followed by composite interval mapping to identify the QTLs, and it was found that the majority of 18 QTLs detected were race-specific. The results also confirmed the hypothesis that partial resistance genes might be defeated major genes with residual effectiveness and race specificity [52]. Likewise, several of the QTLs for resistance to late blight were found to be race-specific [53]. In a comparative genomic studies with blast fungus in barley and rice, Chen et al. [54] observed a high degree of isolate specificity of the QTLs; four pairs of the QTL showed corresponding map positions between rice and barley, two of the four QTL pairs had complete conserved isolate specificity, and another two QTL pairs had partial conserved isolate specificity. Such corresponding locations and conserved specificity suggested a common origin and conserved functionality of the partial resistance genes underlying the QTLs for quantitative resistance. Quantitative resistance to late blight in potato, which was previously been characterized as race-nonspecific, was later shown to be race-specific by QTL analysis [53]. It is assumed that QTLs are defeated major genes (allelic versions of qualitative resistance genes with intermediate phenotypes) with residual effects, but this does not necessarily point out to a function similar to race-specific major genes [4]. For example, in rice a "defeated" resistance gene (Xa4) has been shown to act as a QTL against a virulent strain of *Xanthomonas oryzae* pv. *Oryzae* [55]. The results suggested that a high level of durable resistance to *X. oryzae* may be achieved by the cumulative effects of multiple QTLs, including the residual effects of "defeated" major resistance genes. However, there was no indication of any QTL in the barley genome at the region of powdery mildew resiatance gene, Mla*12*, indicating that the isolate used in the study completely neutralized this major resistance gene, and consequently no residual effect of this gene remained [56]. There is also the possibility that in several species QTLs for resistance have been mapped to the proximity of major resistance genes. In rice blast, three of the QTLs mapped to the same marker intervals as previously identified qualitative blast resistance genes [57]. Similarly, one QTL for late blight resistance in potato coincided in location with a dominant, race-specific gene *R1* [58] and a gene for resistance to PVX, known as *Rx2* [59]. In potato, one major and two minor QTLs have been identified for PLRV resistance; the major QTL, *PLRV.1*, mapped to potato chromosome XI in a resistance hotspot containing several genes for qualitative and quantitative resistance to viruses and other potato pathogens [60]. In this study, genes with sequence similarity to the tobacco *N* gene for resistance to tobacco mosaic virus were also found to be tightly linked to the major QTL, *PLRV.1*. The cDNA sequence of this N-like gene was used to develop the sequence characterized amplified region (SCAR) marker N1271164 that can assist in the selection of potatoes with resistance to PLRV. However, cloning of multiple alleles of major resistance genes and the generation of transgenic (truly isogenic lines) may provide conclusive evidences whether some alleles determine qualitative resistance, while others contribute to quantitative resistance.

Durable Resistance

In this context, durable resistance is beginning to be conceived due to one or more complete qualitative genes, several partial resistance genes, or a combination of both [50]. A typical example is the rice blast disease underlying the involvement of both partial and complete resistance in affecting a wide-spread durable resistance. Recently, for blast resistance in rice, two QTLs were detected on chromosome 4, and one QTL was detected

on each of chromosomes 9 and 12 [61]. The resistance gene, designated as *pi2*, was mapped on chromosome 4 as a single recessive gene between RFLP marker loci *G271* and *G317* at a distance of 5.0 cM and 8.5 cM, respectively. In South American leaf blight infecting the rubber tree, a common QTL was detected for resistance to five strains for both reaction type and lesion diameter on immature leaves, while two QTLs were common for complete resistance to four strains for reaction type and lesion diameter, respectively, suggesting the resistance determinism for complete and partial resistance [62]. For scab resistance in apple, one major resistance gene, *Vg*, and seven *QTLs* were identified for eight isolates of *Venturia inaequalis* [63]. This study further showed that a major QTL, colocalized with the major scab resistance genes *Vr* and *Vh8* on LG2, displayed alleles conferring differential specificities. QTL analysis for durable leaf rust resistance in wheat detected 8 QTLs for leaf rust resistance and 10 QTLs for the quantitative expression of leaf tip necrosis, and four QTLs for leaf rust resistance coincided with QTLs for leaf tip necrosis [64]. In *Solanum microdontum*, two different segregating QTLs for durable resistance to *Phytophthora infestans* have been mapped [65].

QTL Mapping for Disease Resistance in *Arabidopsis*: A Case Study for Powdery Mildew

QTL mapping in model plant system *Arabidopsis thaliana* is rather recent in comparison to QTL studies for other characters [66]. A minimum of eight loci controlling natural resistance to powdery mildew (caused by obligate pathogenic fungi *Erysiphe cichoracearum*) have been described including both monogenic and digenic resistance conferred by semi-dominant or recessive disease resistance genes. QTL analysis for powdery mildew in *A. thaliana* was initiated in a set of RILs derived from a cross between Kashmir-1, a highly resistant line, and accession Columbia glabrous (Col-*gl1*), a susceptible line [67]. In this study, three unlinked QTLs were identified, and for each QTL, the resistance alleles were found to be derived from Kashmir-1. The QTLs, designated as *RPW10*, *RPW11* and *RPW12* were found to act additively to confer resistance to powdery mildew, and together they explained 63 % of the total variation in powdery mildew resistance phenotype [67]. The first QTL, *RPW10*, was mapped on the bottom of chromosome III near the marker R30025 with a confidence interval of only 6.0 cM. The second QTL, *RPW11*, occurred near the marker nga139 on the top of chromosome V with a confidence interval of 12.0 cM, while the third QTL, *RPW12*, was near the marker nga1126 with a confidence interval of 11 cM. Since there were no epistatic interactions, all the three QTLs were additive in their effects on powdery mildew resistance. The demonstration that the QTL RPW10 was allelic to the cloned gene *RPW8* provided additional confirmation of its validity, and this locus having the strongest effect on powdery mildew resistance was genetically mapped to a 4 cM (500-kbp) interval defined by markers M005-S and CIC8-E1RE on chromosome III. It was also demonstrated that the QTL, *RPW10*, was allelic to *RPW7*, which confers resistance to *Erysiphe cichoracearum*, supporting the hypothesis that this locus encodes a broad-spectrum resistance mechanism [68]. QTL analysis for powdery mildew in *A. thaliana* further supports the hypothesis that QTLs are distinct from classical race-specific resistance genes [67].

QTL Mapping for Disease Resistance in Barley: A Case Study for Stripe Rust

Barley stripe rust, caused by *Puccinia striiformis* f. sp. *hordei*, is an important disease of barley (*Hordeum vulgare*) causing serious yield losses throughout the world. QTLs for barley stripe rust were mapped to barley chromosomes 4 (4H) and 7 (5H) in one accession [69] and chromosomes 2 (2H), 3 (3H), 5 (1H) and s6 (6H) in another [70]. It was hypothesized that these accessions have different QTL alleles for barley stripe rust, and accordingly a complex population was developed, which pyramided the QTL alleles on chromosome 4 (4H) and 7 (5H) sib with the QTL alleles on chromosome 5 (1H) [71]. Recently, in a study genes conferring resistance to barley stripe rust at the seedling stage after inoculation with three different isolates, viz., PSH-1, PSH-13 and PSH-14 were mapped in a double haploid population (F1-derived from cross Shyri × Galena) in which adult plant resistance genes had previously been mapped [72]. Two main-effect QTLs- one designated as *QTL5* on chromosome 5 (5H) and another as *QTL6* on chromosome 6 (6H)- were detected, and in all cases 'Shyri' contributed the resistant alleles. There was no significant QTL × race interaction, suggesting race-nonspecificity of these seedling resistance QTLs. The *QTL5* region comprised a relatively small physical part of the chromosome, but the *QTL6* region covered approximately half of the corresponding chromosome. Interestingly, however, both the QTLs coincided in their location with the two most important adult plant resistance QTLs reported earlier by Toojinda *et al.* [70]. Therefore, it became apparent that determinants of resistance to three different isolates of *P. striiformis* f. sp. *hordei* at the seedling stage, and determinants of adult plan resistance mapped to the same regions of the barley genome. This type of QTL coincidence may be due to linkage or pleiotropy. It was also observed that the *QTL5* was located in a region of intermediate recombination frequency, while *QTL6* was located in the border between high and low recombination frequency zones [73].

Multiple qualitative and quantitative resistance genes to different pathogens and different specificities of the same pathogen have been mapped to the *QTL5* and *QTL6* regions (see the references in 72). In conclusion, this QTL analysis showed that the regions of the barley (cv. Shyri) genome where adult plant QTL alleles for *P. striiformis* f. sp. *hordei* were identified could be phenotypically selected for at the seedling stage under controlled environmental conditions. This could reduce the time required to develop resistant barley varieties because multiple generations could be advanced under controlled environmental conditions simultaneously when a single generation is evaluated under field conditions.

QTL Mapping for Disease Resistance in Potato: A Case Study

Since potato (*Solanum tuberosum* L.) is a tetraploid (2n=4x=48) with complex tetrasomic inheritance and highly heterozygous due to severe inbreeding depression after repeated selfing, genetic analysis is somewhat problematic in this crop species. In potato, one to four different alleles are present per locus which results in one homozygous [quadruplex (A1 A1 A1 A1)] and four heterozygous [triplex (A1 A1 A1 A2), duplex (A1 A1 A2 A2), simplex (A1 A2 A2 A2) and nulliplex (A2 A2 A2 A2)] genotypes. Therefore, with two alleles at a tetraploid locus there are five genotypes, and with four alleles at a locus there are 35 genotypes. The profile of a simple monogenic inheritance of a dominant resistance allele (e.g. *R* gene) in a tetraploid potato plant can be in one of four allelic states: homozygous quadruplex (*RRRR*), heterozygous triplex (*RRRr*), heterozygous duplex (*RRrr*) and heterozygous simplex (*Rrrr*). In this simplest genetic model, the expected ratios in progenies of heterozygous resistant and homozygous (*rrrr*) susceptible plants would be 1: 0 resistant and susceptible plants for triplex parent, 5: 1 for duplex parent and 1:1 for simplex parent, assuming that there is chromosome segregation, not chromatid segregation. This clearly shows the complexity of the inheritance pattern of even simple qualitative (monogenic) resistance gene in potato as compared to other crop species where the inheritance pattern is disomic. This fact prevented the development of genetic linkage map in potato. But two new developments paved the way for genome-wide characterization of quantitative disease resistance in potato: the manipulation of ploidy levels and the use of DNA markers. At the diploid level, the complexity of genetic analysis in potato became simpler. Therefore, in potato the mapping population for QTLs consisted of F1 populations derived from two diploid heterozygous *S. tuberosum* subsp. *tuberosum* breeding lines and backcross progenies [74,75]. Using this type of mapping populations, over the past one decade several genetic linkage maps have been constructed in potato based on RFLP, AFLP, SSR and other PCR-based markers, and some of these maps can be aligned with the molecular maps of tomato and pepper based on common RFLP markers [41]. A list of QTLs for important pathogens (diseases) in potato is shown in Table 2.

Integration of QTLs for resistance to late blight, cyst nematode and blackleg or bacterial soft rot in the potato function map for resistance revealed several examples of linkage between *R* genes and QTLs. The most prominent genetic hotspots containing multiple genes for *R* gene resistance and QTLs for different pathogens are located on chromosomes V, XI and XII in potato. This clustering of monogenes and QTLs to diverse pathogens as observed in the potato genome may occur by chance or may be because of reduced recombination fractions due to proximity of the centromere. Some QTLs may be structurally related to *R* genes acting against the same or a different pathogen or linked QTLs to different pathogens may be similar at the molecular level [41]. Based on molecular evidences, it has been proposed that most of the single dominant genes for resistance in the potato function map are primarily encoded by *NBS/ Che Y-LRR* genes or one of the other major classes of resistance genes irrespective of their pathogen specificity [41]. For example, the clustering of genes for resistance to potato virus A (PVA), potato virus Y (PVY) and potato leaf roll virus (PLRV) suggested that some of the genes have an identical molecular basis, either being alleles of a single locus or having evolved from a common ancestor by local gene duplications with subsequent functional diversification. Marczewski *et al.*[60] have shown that a major QTL for PLRV, PLRV.1, mapped to potato chromosome XI in a resistance hotspot containing several genes for qualitative and quantitative resistance to viruses and other potato pathogens, is tightly linked to a tobacco *N*-like gene for resistance to tobacco mosaic virus. These authors further used the cDNA sequence of an *N-like* gene to develop SCAR marker N1271164 that could assist in the selection of potato with resistance to PLRV. Tightly linked to the resistance gene cluster on the long arm of potato chromosome XI are several genes with sequence similarity to the *N* gene for resistance to tobacco mosaic virus [76]. It has also been shown that the cloned potato genes for PVX [77] and root cyst nematode [78] belong to the same superfamily of resistance genes line *N*. The co-localization of N-like genes suggests that genes with sequence similarity to known *R* genes are the molecular basis for some resistance factors in the cluster on chromosome XI including *PLRV.1* [60].

Table 2. Mapping quantitative trait loci (QTLs) for important pathogenic and pest diseases in potato (*Solanum tuberosum* L.)

Chromosome	Pathogen/ pest	QTL	Reference (s)
I	*Erwinia carotovora ssp. atroseptica*	*Eca*	[179]
	Phytophthora infestans	*Pi*	[247]
II	*Erwinia carotovora ssp. atroseptica*	*Eca*	[179]
	Phytophthora infestans	*Pi*	[247]
III	*Erwinia carotovora ssp. atroseptica*	*Eca*	[179]
	Phytophthora infestans	*Pi*	[53,247,248]
	Globodera rostochiensis	*Gro1.4*	[249]
IV	*Erwinia carotovora ssp. atroseptica*	*Eca*	[179]
	Phytophthora infestans	*Pi*	[53,247,250]
	Globodera pallida	*Gpa4*	[251]
V	*Phytophthora infestans*	*Pi*	[53,247]
	Globodera pallida	*Gpa*	[252]
	Globodera pallida	*Gpa5*	[253]
	Globodera pallida, G. rostochiensis	*Grp1*	[254]
VI	*Erwinia carotovora ssp. atroseptica*	*Eca*	[179]
	Phytophthora infestans	*Pi*	[53,247]
VII	*Erwinia carotovora ssp. atroseptica*	*Eca*	[179]
VIII	*Erwinia carotovora ssp. atroseptica*	*Eca*	[179]
	Phytophthora infestans	*Pi*	[247,255]
IX	*Erwinia carotovora ssp. atroseptica*	*Eca*	[179]
	Phytophthora infestans	*Pi*	[53,247]
	Globodera pallida	*Gpa6*	[253]
X	*Erwinia carotovora ssp. atroseptica*	*Eca*	[179]
	Globodera rostochiensis	*Gro1.2*	[40]
XI	*Phytophthora infestans*	*Pi*	[53,247]
	Erwinia carotovora ssp. atroseptica	*Eca*	[179]
	Globodera rostochiensis	*Gro1.3*	[40]
	Potato leaf roll virus (PLRV)	*PLRV.1*	[60]
XII	*Erwinia carotovora ssp. Atroseptica*	Eca	[179]
	Phytophthora infestans	Pi	[176,247]

Integration of QTL Analysis and Molecular Biology: The Candidate Gene Approach

Despite tremendous progress made over the past one decade on QTL mapping in diverse plant taxa, there are several limitations of the genetic analysis of quantitative resistance. First, quantitative trait loci responsive to epistatic interactions are not easily detected by QTL mapping. Second, only those QTLs can be identified that display allelic variation, and genetic fixation at a QTL makes them unnoticeable and imperceptible. Third, many phenotypes of quantitative resistance traits are not easily defined nor can they be measured easily. Similarly, different methods for assessing resistance are likely to be controlled by overlapping sets of partial resistance genes. However, there are very few studies on QTL mapping that address these areas critically [50]. Since the life cycle of most of the pathogens requires several distinct phases of interaction with its host, different genetic interactions may occur between plant and pathogen during each of these stages. Therefore, analysis of the pathogen proliferation at each of these stages, scoring of disease symptoms by several criteria and the use of different inoculation procedures may identify the genes responsible for such differences and help in characterizing them at the functional level. The candidate gene approach intends to link the genetic QTL analysis and the molecular biological methods. The association of candidate genes with QTLs is a step toward understanding the molecular basis of quantitative resistance to an important plant disease. Candidate genes are genes that overlap QTL confidence intervals. To link quantitative resistance phenotypes to functional genes, "candidate genes" (cDNA fragments, defence gene analogues, resistant gene analogue sequences, pathogenesis-related protein genes, positional homologues, homologous sequences, expressed sequences, etc.), which are specifically expressed during disease reaction, can be used as genetic marker loci in QTL mapping studies. By mapping the specific candidate genes on the genetic map, chromosomal regions can be

detected which carry these genes. And in the same mapping population a large number of segregating resistant phenotypes can be measured resulting in the localization of QTLs on certain chromosomal regions. The genetic markers based on candidate genes or sequences involved in the expression of resistant reaction may co-segregate with certain resistant phenotypes. As a result, the presence and absence of correlations in chromosomal position between phenotypes and candidate markers would provide clues not only to understand the function of these resistant genes by their correlated phenotypes, but also to characterize the kind of functional genes involved in the realization of a certain resistant phenotype. The coincidence of a map position of a QTL on the one hand and a candidate gene on the other hand serves as a strong indication for the function of these candidate gene as well as indication of the genes involved in the QTL phenotype.

Recently, linkage disequilibrium mapping method has been employed to test for an association between a candidate gene marker and resistance to *Verticillium dahliae* in tetraploid potato [79]. In this study, a probe derived from the tomato *Verticillium* resistance gene (Ve1) identified homologous sequences (*StVe1*) in potato, which in a diploid population were mapped to chromosome IX in a position analogous to that of the tomato resistance gene. When a molecular marker closely linked to the homologues was used as a candidate gene marker on 137 tetraploid potato genotypes, the association between the marker and resistance was confirmed. Cloning of homologues indicated that the QTL comprised at least an eleven-member family, encoding plant-specific leucine-rich repeat proteins very similar to the tomato *Ve* genes; the sequence analysis showed that all homologues were uninterrupted open reading frames, and thus represented putative functional resistance genes. A very important implication of this study was that it was possible to map QTL directly on already available potato cultivars without developing a new mapping population [79]. In QTL analysis of citrus tristeza virus (CTV) in progenies derived from sour orange (*Citrus aurantium*) and *Poncirus trifoliata*, three major QTLs were detected at the position of *P. trifoliata* resistance gene, *Ctv-R*, and up to five minor QTLs were detected (Ctv-A1 to Ctv-A5) [80]. An analogue of this resistance gene was observed to be a candidate for minor QTL Ctv-A3, and two expressed sequences were candidates for minor QTLs Ctv-A1 and Ctv-A5. Recently, resistance and defence gene analogue (RGA/ DGA) sequences (as candidate genes) were isolated in cocoa with degenerate primers designed from conserved domains of nucleotide-binding-site motif present in a number of resistance genes such as the tobacco *N*, sub-domains of serine/threonine kinases such as the *Pto* tomato gene and conserved domains of two defence gene families such as pathogenesis-related proteins (PR) of classes 2 and 5 [81]. In this study, an enrichment of the genetic map with microsatellite markers resulted in several co-localisations of these candidate RGAs, DGAs and QTLs for *Phytophthora* on chromosome IV, where a cluster of *Pto*-like sequences and 4 QTLs for *Phytophthora* were also observed. DGAs and RGAs were also used as candidate genes with QTLs to anthracnose in common bean [82] and to leaf/ stem rust in wheat [83]. In pepper, a class-III chitinase gene co-localized with a major-effect QTL, and PR protein classes 2 and 5 loci such as PR4, PR2 and PR10 with minor QTLs to *Phytophthora capsici* [84]. Linkage of *Ascochyta* blight QTLs to candidate genes including disease response genes and resistance gene analogues has also been reported in pea [12]. In rice, several candidate genes involved in both recognition (RGAs) and general plant defence response (DR) were associated with QTLs for blast, bacterial blight, sheath blight and brown planthopper leading to the construction of the frame map which provided reference points to select candidate genes for co-segregation analysis using other mapping populations, isogenic lines and mutants [85]. All these studies indicate that the candidate gene markers are excellent tools when searching for universal markers for marker-assisted selection by linkage disequilibrium mapping in wide gene pools. The finding of linkage disequilibrium between a candidate gene marker and a QTL supports the hypothesis that the candidate gene is indeed the resistance gene or at least is located physically very close to the resistance gene [41].

Conclusions

No doubt QTL mapping aided by DNA markers has revolutionized the study of complex quantitative disease resistance in plants. It has become a powerful tool for marker-assisted selection (MAS) for breeding for disease resistance. However, the effectiveness of MAS is determined by the relative linkage disequilibria between the genetic marker loci and QTLs that condition disease resistance expression. It needs to be emphasized here that if a significant amount of the additive variance associated with a QTL can be accounted for by DNA markers, then MAS can increase the breeding efficiency. Further, a greater genetic gain can be made if flanking QTLs between two marker loci are used as compared to single marker, especially if the single marker are not tightly linked to the QTL, i. e. the linkage distance between the marker and the QTL is relatively high. Several factors determine the usefulness of QTL-marker association for MAS, and most important among them are epistatic interactions with other loci, variations in linkage phase and QTL × environment interactions. Very

recently only, these factors are being seriously examined in QTL analysis studies for plant disease response [86-90]. Finally, positional cloning of partial resistance genes underlying QTLs may eventually lead to their transgenic exploitation for conferring effective durable resistance. The adaptation of strategies like substitution mapping [91] and other methods to treat QTLs as qualitative loci [50] has enormous potential for realizing this goal. And in this direction, the candidate gene approach definitely offers newer perspectives; the application of candidate genes might facilitate the discovery of the functions of QTLs. It has been shown that the tight linkage of RGA markers to the major QTLs on linkage group would allow map-based cloning of the underlying resistance genes [92,93]. However, the high sequence similarity reveals potential problems for the use of RGAs as molecular markers. Their application in marker-assisted selection (MAS) and the construction of high-density genetic maps is complicated by the existence of closely linked homologues resulting in 'ghost' marker loci analogous to 'ghost' QTLs [13]. Therefore, implementation of genomic library screening, including genetic mapping of potential homologues, seems necessary for the safe application of RGA markers in QTL analysis, MAS and gene isolation. With these recent developments, complex forms of quantitative disease resistance and their underlying genes are becoming more accessible, and it is thus not unrealistic that these developments would aid in perfecting newer strategies for effective disease control and management in crop species in future.

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