Quantitative Disease Resistance

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Quantitative disease resistance (QDR) is the ability of the plant to reduce pathogen multiplication and is expressed on a continuous scale caused by the simultaneous segregation of many genes affected by environment.

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1. Introduction

Disease resistance has been dichotomically classified in qualitative and quantitative resistances. Qualitative resistances are based on individual resistance (*R*) genes mainly encoding *R* proteins that interact with pathogen-specific effectors triggering a complete defensive response called effector-triggered immunity (ETI). These monogenic resistances are easy to handle but prone to be overcome by the pathogen by mutation and subsequent selection of virulent populations. Quantitative disease resistances (QDR) are typically caused by many genes with moderate to small effects that are highly affected by environment, plant organ, and plant developmental stage[1]. Since the advent of genomics, we know that QDR to most pathogens is caused by hundreds of quantitative trait loci (QTLs) that are distributed across the whole genome[2]. QDR results from a restriction in the growth or development of the pathogen imposed by the host. Bascially, it slows down the rate of epidemic development by effects on latent period, infection efficiency, spore production and/or other epidemiological traits.

Theoretically, QDR should be measured by determining the amount of pathogen in the host tissues. In practice, this is not feasible for large populations and typically, QDR is determined in multi-environmental field trials by visual symptom rating with the inclusion of standard varieties of known resistance level. Each genotype that is performing significantly better than a susceptible standard is called ‘quantitatively resistant’. The degree of QDR can vary from very small to nearly full resistance. Therefore, quantitative scales of disease assessment, which are good estimators of the pathogen development, are inevitable. This could be a simple 1 to 9 scale as preferred by plant breeders or a 0–100% scale that allows a finer resolution. QDR is working against all races of a pathogen and pathogen populations cannot adjust fast to QDR due to its complex inheritance. QDR must be strictly separated from individual genes that provide partial resistant phenotypes only, because from a genetic point-of-view they are clearly monogenically inherited.

2. Genetics of Quantitative Disease Resistance (QDR)

QDR was found in all pathosystems analyzed so far and is independent from the presence of qualitative resistances. Depending on plant the growth stage, the same genotype can have different QDR to the same pathogen species. This is notorious for Fusarium diseases that can affect practically all cereal stages and organs, but the correlations among the respective QDR are always poor illustrating that different sets of resistance genes are responsible where most (but not all) are stage specific. Most important for growing of winter cereals is the adult-plant resistance. Typically, QTLs are providing a non-race specific resistance that is effective against all genotypes of a pathogen or even against different pathogens (multi-disease resistance, MDR). The molecular basis of QDR is poorly understood[1], mainly because it is a multi-facetted defense system that involves hundreds of (unspecific) cellular processes, like cell-wall thickening (lignin, callose, glycoproteins), phytoalexins, reactive oxygen species (ROS), pathogenesis-related (PR) proteins (chitinases, glucanases), inhibitors of fungal enzymes, detoxification of toxins, lack of/different toxin receptors[3]. Although some of these pathways are also used by monogenic, qualitative resistance genes, QDR typically does not comply a complete resistance and shows no symptoms of hypersensitivity.

Because of the complex inheritance and their non-race specifity, QDR is thought to be of higher durability than qualitative disease resistances that are notorious for being overcome by single mutations in the pathogen population. Although it is impossible to prove durability experimentally before the release of cultivars, there are examples where QDR lasted for many decades although it might get less effective in an evolutionary perspective.

Genetic analyses for the study of inheritance of QDR are based on biparental populations where a greater array of progenies should be analyzed. Population size depends on the number of genes and the size of their effects, but should not fall below 200. The genotype ranking among different environments could be highly different, therefore, multi-environmental trials (=locations × year combinations) are necessary. In contrast to qualitative resistances, QDR can only hardly be analyzed in climate chambers or greenhouse trials because they have to be tested in those environmental stages where they are necessary in the field, mostly the adult-plant stage. High-throughput phenotyping, for example by image analysis, offers a future perspective to improve trait assessment in QDR and makes it faster and more accurate.

Biometrical analyses of QDR must follow established quantitative-genetic theory including analyses of variance and covariance, multiple comparison tests. Since the advent of genomics, QDR are analyzed by bi- or multi-parental QTL mapping or genome-wide association mapping based on diversity panels and taking advantage from historic recombination events. Thus, much more accessions could be screened with the possibility to identify new alleles not represented in parents of bi-parental mapping populations. Both methods detect QTLs that can be used either directly for marker-assisted selection (MAS) or marker-assisted backcrossing (MABC), when the effects are high enough and reproducible in unrelated populations or for marker-assisted recurrent selection (MARS) when the effects are only small. Based on medium- to high-density marker assays, where the whole genome is scanned for QDR effects, also genomic selection is feasible[4]. The high impact in genomic research might in future result in the knowledge of more genes underlying disease-resistance QTLs with a high effect. This opens the avenue to genome editing techniques that are, however, highly challenging for manipulating whole gene networks as it is necessary for the improvement of QDR.

References

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