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REVIEW

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Components of slow disease development: a key to enhance resistance in crops

Kumari Shikha[1,6], R. Chand[2], N. K. Mishra[3], S. Singh[4], B. R. Sayiprathap[5], R. M. Nair[5] and A. K. Singh[6*]

Abstract

Plant resistance is a result of interaction between host, pathogen, environment and temporal factors. Major or "R" gene resistance may break down following the emergence of virulent isolates of a pathogen. Limited durability of R genes has opened the door for the utilization of slow disease resistance in crop breeding. Plant pathogens with high reproduction ability exhibit greater genetic diversity leading to loss of major gene-based resistance. Consequently, minor genes-based resistance can be effectively employed against all the available virulent isolates within a pathogen population, including non-elicitor producing pathogens. Several researchers have identified valuable genetic sources by screening germplasm collections and characterizing genes conferring slow disease development. The identification and possible cloning or tagging of such genes obtained from crop wild relatives will create better opportunities for their use in crop improvement. Nevertheless, very little information is available about the nature of individual genes responsible for slow disease development. A thorough understanding of the nature of inheritance of slow disease resistance, interactions, and the possible breeding strategies to enhance resistance governed by slow disease components will help in breeding or developing resistant cultivars with enhanced yield. This review discusses the components of SDD in terms of identification, characterization, factors influencing it, and breeding strategies to enhance resistance governed by SDD components. Furthermore it emphasizes the importance of targeted breeding strategies to exploit the potential of SSD in developing cultivars with enhanced resistance and maintaining a good yield.

Keywords Gene pyramiding, Germplasm, Multiline, Diseases resistance

Introduction

The initiation of plant disease results from the interplay between the host (plant) and pathogen in a disease favourable environment. Tolerance and resistance are two terms most frequently used in context to plant disease. Tolerance is defined as the host's ability to reduce the negative effects of infection. However, *resistance* is referred as host's ability to limit pathogen multiplication (Pagan and Garcia-Arenal 2020). Various plant pathogens including, bacteria, fungi, viruses, nematodes, mycoplasma, and parasitic phanerogams significantly impact the crop production posing severe threat to agriculture (Nazarov et al. 2020). In addition to these pathogenic agents, abiotic factors, genetic uniformity, and the area of the cultivated varieties also play a critical role

*Correspondence:
A. K. Singh
aksingh25@gmail.com
[1] Department of Genetics and Plant Breeding, Institute of Agriculture and Natural Sciences, Deen Dayal Gorakhpur University, Gorakhpur 273 009, India
[2] Department of Mycology and Plant Pathology, Institute of Agricultural Sciences, Banaras Hindu University, Varanasi 221 005, India
[3] Late Dr. RCSD College of Agriculture and Research Station, Korea, Baikunthpur, Chhattisgarh 497 335, India
[4] Department of Horticulture, Institute of Agriculture and Natural Sciences, Deen Dayal Upadhyay Gorakhpur University, Gorakhpur 273 009, India
[5] World Vegetable Center South/Central Asia, ICRISAT Campus, Patancheru, Hyderabad, Telangana 502 324, India
[6] Department of Genetics and Plant Breeding, Institute of Agricultural Sciences, Banaras Hindu University, Varanasi 221 005, India

in determining the disease spread (Keneni et al. 2012). Plants exhibit three primary responses to pathogens (Fig. 1): (i) disease, (ii) a hypersensitive response (HR) leading to no disease development and (iii) a non-hypersensitive response resulting in slow disease development (SDD) (Hoglund et al. 2005; Bhardwaj et al. 2021). The HR is pathogen race-specific, and is governed by major resistant gene often nucleotide-binding site leucine-rich repeat (NLR) proteins that interact with the pathogen in a gene-for-gene manner (Basnet et al. 2022; Waheed et al. 2022). Many molecularly described major genes for HR belong to the nucleotide-binding leucine-rich repeat (NB-LRR) class, particularly against biotrophic pathogens (Nimchuk et al. 2003). While major genes for HR can confer complete resistance, instances of incomplete resistance have also been known (Gonzalez et al. 2012). Notably the effectiveness of major gene resistance is short-lived, as the pathogens can adapt by losing avirulence factors that trigger host defense. Consequently, major genes contribute to a low durability resistance (Plissonneau et al. 2016). In a recent study, resistance to oat powdery mildew (*Blumeria graminis* f. sp. *avenae*) governed by three major genes (*Pm1*, *Pm3* and *Pm8*) show broke down due to the over-reliance on a single cultivar Barra grown in Ireland since 1985 (Reilly et al. 2024).

SDD can be interchangeably referred to as quantitative resistance, race non-specific, incomplete resistance, partial resistance, polygenic resistance, complex resistance, horizontal resistance, field resistance, and durable resistance (Gonzalez et al. 2012). Its worth nothing that each of these terms holds specific meaning in different context. The term 'SDD' itself is not novel and has often been used as a substitute for slow rusting, slow mildewing, slow blighting, slow blasting and slow wilting. SDD has been proved effective in reducing disease spread by interfering pathogen's reproduction capabilities. The concept of resistance was initially proposed by Vanderplank based on epidemiological evidence (Shaner et al. 1978). SDD in plants, especially against multiple pathogen races, has gained significant interest in crop improvement for disease management, particularly in low-input cropping systems. It can serve as a valuable guide for breeders aiming to achieve sustainable crop production with stable yield. However, aggregating SDD contributing alleles is challenging due to its complex polygenic inheritance conferred by quantitative trait loci (QTL). Understanding the nature of the inheritance of slow disease, its components, and possible associations among them is crucial for utilizing the durable resistance offered by SDD.

The detection of QTLs governing SDD has become feasible in recent times due to the availability of whole-genome covering molecular markers, such as Single Nucleotide Polymorphisms. To enhance the durability of plant resistance against pathogens, huge numbers of progeny will be required if the effect of any one gene is small and various breeding approaches such as, multiline development, gene pyramiding, rotation of resistant genotypes have been employed to deploy major resistance genes/QTLs. Combining resistant genes with QTLs in a cultivar can contribute to maintaining the durability and effectiveness of the resistant genes for a prolonged period of time. This review aims to discuss the components of SDD, their identification, characterization, and the breeding strategies employed to achieve enhanced disease resistance.

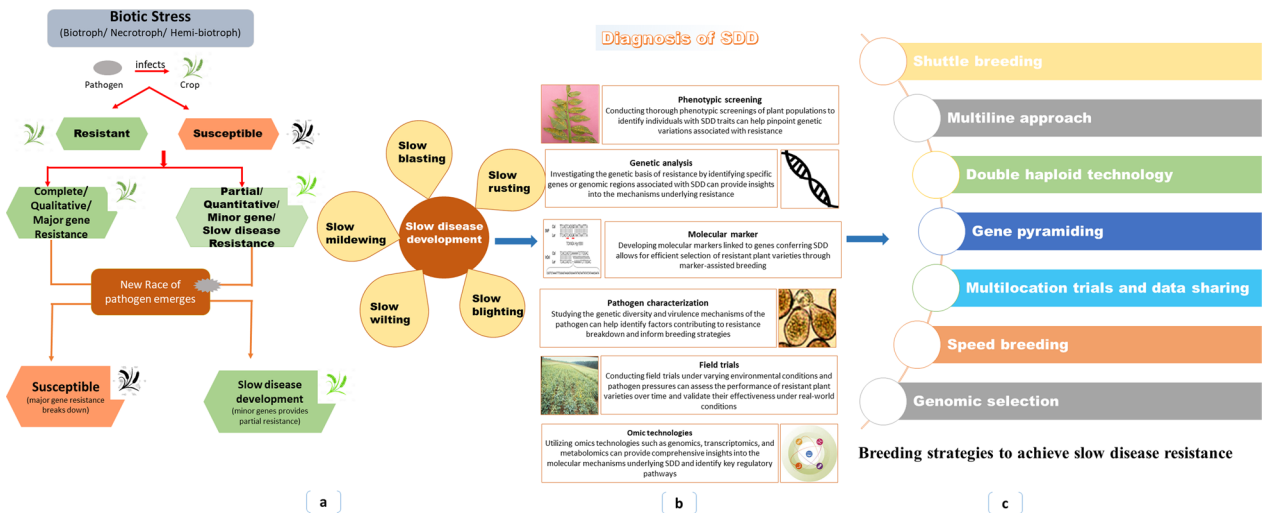

Fig. 1 **a** Flow chart depicting the nature of slow disease development, **b** approaches for identifying several components of SDD and **c** breeding strategies for SDD

Components of slow disease development (SDD)

The processes associated with slow disease development (SDD) have an invariable impact on different stages of the pathogen's infection cycle such as, spore germination, penetration, and colonization of host tissues, as well as the duration of incubation (the length of time between infection and first symptoms to appear), latent period ('the length of time between the start of the infection process by a unit of inoculum and the start of production of infectious units' defined by Madden et al. 2007).), and sporulation. These processes referred to as "*components of slow disease development*," which plays an important role in regulating the epidemic that can arise from a chain of infection cycles during the host-growing season (Bove and Rossi 2020). Here, we emphasize the model of SDD in which the level of inoculum is directly proportional to the disease. But in most cases this relationship is logarithmic/exponential, reaching an asymptote. When there is no inoculum, no disease occurs; if the inoculum is low, the disease is also less severe. Conversely, as the inoculum increases, the disease intensifies. Thus, we can conclude that in SDD, all the resistant components work collectively or individually to limit inoculum production. The components of SDD have been extensively studied in a number of crops (*Supplementary table*). The individual SDD components such as incubation period, latent period, number of diseased symptoms per unit plant area and pustule size or amount of infectious spores produced exhibit continuous variation during segregation, suggesting their influence by oligogenes where the effect of individual gene(s) is very small. Furthermore, several researchers reported moderate to high heritability estimates for these components, therefore, these components could serve as valuable criteria for screening slow disease-resistant lines. Additional research is needed to decipher the genetics and transmission of individual SDD components to facilitate the development of varieties with enhanced level of resistance.

Area under disease progress curve (AUDPC)

The AUDPC serves as a quantitative measurement of total resistance, as it combines all measurable SDD components of resistance, including latent period, infection efficiency, size of uredia/pustule, and rate of sporulation into a single value (Sakr 2019). It is calculated by using the following function (Shaner and Finney 1977):

$$AUDPC = \sum_{i}^{n-1} \left[\left\{ \frac{y_i + y(i+1)}{2} \right\} x(t(i+1) - t_i) \right]$$

here y_i is the disease severity on the ith date; t_i is the ith day; n is the number of dates on which disease symptom was recorded.

AUDPC recognised as one of the most efficient measures for assessing SDD (Kushwaha et al. 2007, 2010; Aktas and Zencirci 2016). AUDPC has been proven effective in differentiating various test genotypes into the category of fast (susceptible) and slow (resistant) disease developer (Afzal et al. 2022 in wheat; Singh et al. 2015 in pea; Negussie et al. 2005 in lentil; Sinha and Biswas 2010 in pigeonpea). Utilizing AUDPC as an index for evaluating SDD and partial resistance offers a more precise differentiation among test genotypes compared to final disease severity percentage. This precision is particularly valuable as all genotypes tend to display susceptible disease reactions in the later stages of the crop growth.

Slow disease genotypes can be identified in the early segregating generations resulting from the crosses between slow and fast developing AUDPC genotypes, either based components of resistance or, preferably on the basis of AUDPC calculated under field conditions (Das et al. 1993). As AUDPC is directly associated with yield loss, it serves as a reliable indicator of SDD under field conditions. El-Orabey et al. (2019) categorized 39 wheat genotypes into 03 distinct groups (complete resistant, slow rusting, and fast rusting) based on AUDPC estimates. Furthermore, based on the AUDPC values, QTLs associated with partial resistance have been identified using linked molecular markers (*CFD71*, *csLV34*, *csGSR* and *Xgwm259*). AUDPC measurement is recognised as an important indicator for slow mildewing genotypes and has been highly correlated with per cent diseases index in bitter gourd (Prasanth et al. 2019). Through AUDPC, we can measure the progress of disease development in terms of the combined effect of various SDD components. Some of the important SDD components are discussed below:

Disease severity/final disease severity

Disease severity can be assessed as percentage (%) of the total green plant parts covered by diseased symptoms such as rust pustules or mildew (Belan et al. 2020). Visual assessment of disease severity is subjective and somewhat imprecise due to bias from the data observer (Pujari et al. 2016; Gallego-Sanchez et al. 2020). Ren et al. (2017) proposed recording of disease severities (DS) on the host using a modified Cobb's Scale (Belan et al. 2020). Moreover, optical techniques, such as thermography, multi- and hyperspectral sensors, or chlorophyll fluorescence, RGB imaging, are proven to be useful in revealing primary disease foci and areas varying in disease severity in fields (Mahlein 2016). Despite the presence of the virulent pathogen and favourable environmental conditions,

variations are observed in the rate of disease development among different cultivars. Cultivars exhibiting consistently low final disease severity considered as partial or complete resistant cultivars (Meena et al. 2011).

The evaluation of disease infection frequency is more accurate, as counting symptoms is less subjective than estimating disease severity as a whole, although it is time-consuming and tedious. For precise assessment, the disease scoring scale should adopt a decimal system, and standard deviation equal between class intervals is essential to ensure normal distribution of data. Given its high correlation with AUDPC, selection for slow disease genotypes can be effectively carried out based on Field Disease Score (FDS) estimates in the field. Consequently, FDS data proves to be efficient in the selection of genotypes exhibiting SDD, particularly in scenarios where resources are limited, and obtaining three or more readings for AUDPC estimation is not feasible (Das et al. 1993). The final reading date should be determined based on the susceptible check, ensuring similar days to maturity, and FDS must reach to > 80%. FDS may offer more insightful information for genotypes of similar maturity group with variable resistance. A low frequency of smut infected plants usually results in a lower AUDPC, assuming the disease does not spread significantly over time or the severity is not extreme. However, AUDPC provides a more comprehensive picture by integrating the disease's progression and severity over time, not just a snapshot of infection frequency (Jeger 2004).

Infection frequency/no. of symptoms per unit area

The most accurate method for scoring damages is infection frequency, involving the counting of pustules per unit area after infection and the number of leaflets infected per plant (Pujari et al. 2016; Chaudhary and Banyal 2017). Diaz-Lago et al. (2003) emphasized the significance of a lower relative infection frequency in the selection of a slow rusting oat cultivar for crown rust. A previous finding demonstrated an inverse linear relationship between temperature and infection efficiency; infection efficiency was approximately 20 times greater at 15 °C than at 32 °C in sugarcane cultivar for rust (Barrera et al. 2012).

The reduced number of pustules per unit leaf area may results from a lower percentage of infection sites (Mabrouk et al. 2019). In some slow rusting cultivars, a lower number of aecial cups per pustule (Singh et al. 2015), or conidia per colony, or mildew colonies per leaflet, may be associated with mechanism that interfere with fungus penetration or growth before visible symptoms occur (Chaudhary and Banyal 2017). For example, wheat accessions production of fewer uredinia per square centimetre of leaf and lower pustule density (0.01 to 9.59 cm^2) indicated the presence of uncharacterised slow rusting adult plant resistance (Desai et al. 2018).

Infection rate and apparent infection rate (r)

The magnitude of the infection rate is influenced by number of spores produced per unit area of the sporulation body and the proportion of spores that infect and give rise to lesions (Oerke et al. 2019). Counting pustules is less subjective as compared to estimating disease severity but time-consuming and tedious. Chilasa et al. (2016) observed that slow mildewing pumpkin cultivars had a lesser apparent infection rate (0.02–0.16/day) compared to fast mildewers (0.28–0.31/day). Similarly, based on an infection rate 0.038 to 0.050/day, pea cultivars IC-219028 (A), DPP-54, EC-292166 and VRP-12 were considered slow mildewers compared to Lincoln (0.073/day) (Chaudhary and Banyal 2017).

The apparent infection rate (r) serves as a measure of the speed at which an epidemic develops. It is assessed by recording disease symptoms at multiple intervals, starting from disease initiation to the end of the epidemic. Vanderplank (1978) proposed a formula for estimating the apparent infection rate (r), later modified by Kushalappa (1982). Estimates of r values are widely used to understand, predict, and compare the progression of infection rate as observed in studies where slow mildewing host genotypes exhibited lower apparent infections rates (Chilasa et al. 2016). Similarly, Jain et al. (2021) assessed the apparent infection rate of leaf blast fungus among different rice cultivars to identify slow-blasting cultivars, recognizing the crucial role of conducive weather conditions in infection rate and disease development. Therefore, apparent infection rates using weather data involve predicting the likelihood of pathogen infection based on environmental conditions. This approach helps in detecting partial resistance to pathogens and, importantly, contributes to saving time, labor, and economic resources.

Coefficient of infection (CI)

The Coefficient of Infection (CI) integrates the disease severity and the host reaction, thus providing more comprehensive information. This is calculated by multiplying values of disease severity by host disease reaction (Shabana et al. 2017). Host disease reaction refers to the intensity of infection in percent (Mahmoud et al. 2015). The Average Coefficient of Infection (ACI) is determined by adding CI values of each entry dividing the sum by number of tested years/seasons. Mitiku et al. (2018) appraised slow rusting resistance to wheat stem rust using CI (< 20%) and reported the presence of different partial resistance conferring genes in wheat lines. Several investigators have also suggested the importance of CI in

categorising slow rusting resistance against wheat stem or leaf rust (Pathan and Park 2007; Draz et al. 2015; Hei et al. 2015). Safavi et al. (2010) reported a positive association of CI with FDS and relative AUDPC (rAUDPC) with a strong R^2 value of 0.98 and 0.91, respectively.

Longer incubation and latent periods

The incubation period is defined as the time between inoculation and the initial appearance of visual disease symptoms such as rust, wilt or mildew, while the latent period is the number of days from inoculation until the onset of spore production. In the case of wheat rust, LP 50 refers to the number of days from inoculation to the appearance of 50% urediniospores. Omrani et al. (2019) find that latent period is influenced by both additive and dominance genetic effects. The latent period also varies in slow to late wilting. In late wilting, the first appearance of symptoms is delayed but once initiated, disease progresses rapidly and reaches 100% incidence. Disease incidence can be referred to as the number of plant units that are (visibly) diseased, typically in relation to the total number assessed. However, the appearance of symptoms occurs simultaneously with other lines, but the disease progresses at a slower rate (Hei et al. 2015). The period is also influenced by environment; e.g., in wheat brown rust, increased temperature prolongs the latent period, resulting in a significant increase in the latent period of late rusting cultivars (Nopsa and Pfender 2014). Bove and Rossi (2020) emphasized that slow rusting cultivars with a longer latent period would be effective in reducing the rate of disease development. Negussie et al. (2005) considered latent period as an important component of partial resistance for determining the rate of the rust (*Uromyces viciae-fabae*) epidemic. However, effect on yield loss is not demonstrated in the mentioned study. As per the findings of other researchers (Herrera-Foessel et al. 2006; Safavi 2015; Srinivas et al. 2023a, b), SDD can positively influence grain yield by reducing yield losses, extending the grain filling period, improving grain quality, minimizing stress effects, and enhancing crop resilience.

Small size and delayed pustule rupture

Small pustule size has been reported to be associated with slow rusting in wheat (Skowronska et al. 2020), beans (Souza et al. 2013), lentil (Negussie et al. 2005) and peas (Singh et al. 2023). Additionally, pustule size has been found associated with the sporulation capacity, with larger pustules producing more spores compare to smaller ones (Singh et al. 2015). Mabrouk et al. (2019) suggested that the pustule size varies depending on the differences in the latent period of the testing cultivars. Ramirez-Cabral et al. (2017) explained that fewer and smaller pustules cause damage to smaller areas of the leaf, indirectly resulting in the production of fewer urediniospores. Moreover, delayed pustule rupturing enables slow rusting cultivars to cope with disease infection by delaying dispersion of uredinospores, thereby lowering disease spread.

Reduced lesion size and reduced localization

Van der Plank (1978) stated that the expansion of lesion growth contributes to the total severity of the disease in an epidemic. Hence, researchers potentially utilized lesion size as a component trait for selection of resistant cultivar to various pathogens (Suffert et al. 2013; Bove and Rossi 2020). Counting the number of lesions and determining the pustule size by visual scale is not precise and may lead to errors. Several image analysis tools now are available that can be used for the counts and size of the lesions. Image J (Adobe Photoshop Version: 12.0) is the most commonly used tool for the measurement of number and size. During the assessment of pustule size, sampling time and size are the most important factors as they are highly influence by the environment.

The localization ability of a pathogen is estimated by counting the number of plants colonized by the pathogen (Jendoubi et al. 2017). The higher number of colonized plants indicates the susceptibility of the host. Sinha and Biswas (2010) revealed that slow-wilting pigeonpea genotypes delay the progress of the wilt epidemic (*Fusarium udum*) due to reduced root colonization as well as slowing the growth of the mycelium in xylem vessels. Hence, localization ability could be considered as a component trait for the rapid selection of resistant genotypes in a breeding program. This can be measured as proportion of plants infected out of total plants in a particular area or by the comparison of test genotypes with infector rows where more than > 80% mortality is observed. The resistant materials screened here always show less disease symptoms as compared susceptible genotypes.

Factors influencing slow disease development

The expression of SDD is significantly influenced by various factors, which are described under the following headings.

Genetic diversity of pathogen and gene flow

Stam and McDonald (2018) provided a comprehensive overview of, how the evolutionary potential, mating system, gene flow and population size of the pathogen affect the durability of disease resistance in the host. High evolutionary potential coupled with substantial gene flow and a large population size, empowers pathogen to overcome host genetic resistance. The continuous selection for aggressiveness within the pathogen population

is a dynamic process. Over time many resistant varieties become susceptible e.g., slow blighting cultivar of wheat 'Shatabdi' become susceptible few years after release in Bangladesh. This shift could be attributed to emergence of a new aggressive population of the causal fungal pathogen *Bipolaris sorokiniana*. Similarly, the Bianca cultivar of grape initially exhibits partial resistance to most *Plasmopara viticola* strains but becomes susceptible when infected with the 'L' strain collected in the Czech Republic (Bellin et al. 2009). Additionally virulence factors of the pathogen, play a crucial role in determining the durability of corresponding host resistance genes at the host–pathogen interactions (Sakr 2022).

Genetic background

In a specific host plant, the expression and impact of various components, including incubation period, latent period, pustule size and pustule/spore/colony production all significantly contribute to the genotype effect. Several studies demonstrated that resistance genes are also influenced by cultivar background. For instance, '*Lr2*' alleles exhibited the highest level of resistance in the wheat cultivar 'Thatcher', intermediate resistance in 'Prelude,' and the least resistance in 'Red Bobs' against leaf rust (Chu et al. 2009). A recent study revealed that *Pm7* powdery mildew resistance gene varies in resistance to *Blumeria graminis* f. sp. *avenae* in different oat cultivars (Reilly et al. 2024). Importantly, the genetic background of the host plays a crucial role in influencing the intensity of phenotypic expression of various resistant genes (or QTLs) due to the presence of frequent epistatic interactions among them. A list of different genes/QTLs reported for SDD in various crops has been compiled in Table 1.

Plant's growth stage

The expression of SDD is also influenced by plant growth stages. Experimental evidence has revealed the impact of plant age on partial resistance to *Ascochyta rabiei* in chickpea, showing a decrease in resistance as the plant ages (Elliott et al. 2013). In young leaves, the presence of small and compact cells prevents penetration and spread of pathogen as compared to old leaves (Mabrouk et al. 2021; Azzimonti et al. 2022). However, this theory is not applicable in the *Botrytis fabae*—faba bean interaction system, where the pathogen destroys cells as it progresses through the host (Beyene et al. 2018). The availability of limited nutrients at podding stage due to lower photosynthesis in plants retards growth of *B. fabae*. Additionally, a smaller lesion size and a longer incubation period were observed at the podding stage in faba bean (Beyene et al. 2018). In a previous study (Ficke et al. 2002), grape berries showed the maximum severity to powdery mildew, caused by *Uncinula necator* unless inoculated late in their development. Such age-related or ontogenic resistance describes the ability of whole plants or plant parts to resist or tolerate disease as they age and mature. Thus, it can be concluded that some cultivars often become either more susceptible or more resistant with respect to different plant developmental stages. Many hemibiotroph/necrotrophs mostly spread in succeed the plants from lower leaves towards upper leaves. Such epidemiological information reveals that older leaves are more prone to *Septoria* blight than younger leaves (Odilbekov et al. 2014). The disease severity is generally reported to be higher on the lower leaves compared to the upper new leaves while, obligate biotroph such as powdery and downy mildew prefer the young leaves.

Environment

Environment plays a crucial role in SDD, especially temperature and humidity. The spores of most of the pathogens germinate in the free water, therefore slow resistant-gene express more effectively in a dry environment due to poor dew formation on the leaf and its rapid evaporation during day time. Certain genes require a specific environmental condition for their expression (Rodriguez-Algaba et al. 2019). Many researchers have observed that the expression of certain resistance genes is responsive to a particular temperature range e.g., *Lr11*, *Lr15*, *Lr18* and *Lr30* genes exhibit a lower leaf rust infection at low temperature, while *Lr16*, *Lr17* and *Lr23* resulted in lower infection at higher temperatures (Martinez-Moreno et al. 2021; Srinivas et al. 2021). Katsantonis et al. (2017) reported that the components contributing to the slow blasting resistance in rice genotypes are influenced by factors such as conidia concentration, nitrogen levels and light conditions. Understanding these thresholds can facilitate the identification of desirable genotypes. Thus, the environment, particularly temperature, is believed to plays a crucial role in SDD. Some resistance genes are thermolabile and become ineffective after reaching a certain temperature. Therefore, identifying such genes before deployment is essential. Multi-location testing and screening at different temperature regime are useful to sorting out such genes. Further, assessment of SDD in the field can be subject to high experimental error, due to the effects of environmental factors like field heterogeneity and the presence of other pathogens or pests. On the other hand, measuring individual components of resistance in a controlled environment results in a much smaller experimental error values. If a single component showed a high correlation with field partial resistance, it would be feasible to evaluate and select specifically for that component. This indirect approach could lead to a more efficient enhancement of resistance. SDD resistance

Table 1 List of various genes/QTLs associated with slow disease development (SDD) in different crops

SDD component	Crop name	Genes/QTLs	Chromosome location	References
Slow rusting in wheat	Leaf + stripe rust in wheat	*Lr34/Yr18*	7DS	Singh et al. (2000)
	Powdery mildew + leaf rust + stripe rust in wheat	*Lr34/Yr18/Pm38*	7DS	Spielmeyer et al. (2005)
	Powdery mildew + leaf rust + stripe rust in wheat	*Lr34/Yr18/Pm38*	7D	Lagudah et al. (2009)
	Leaf + stripe rust in wheat	*Lr67/Yr46*	4DL	Herrera-Foessel et al. (2011)
	Stem + stripe rust in wheat	*Sr2/Yr30*	3BS	Singh et al. (2011)
	Leaf rust in wheat	*Lr68*	7BL	Herrera-Foessel et al. (2012)
	Powdery mildew + stripe rust + leaf rust in wheat	*QPm.caas-4DL*, *QPm.caas-6BS*, and *QPm.caas-2BL*	4DL, 6BS and 2BL, respectively	Liu et al. (2014)
	Stem rust + powdery mildew in wheat	*Lr67/Yr46/Sr55/Pm46/Ltn3*	4DL	Herrera-Foessel et al. (2014)
	Stem rust in wheat	*Sr56* (*QSr.sun-5BL*)	5BL	Bansal et al. (2013)
	Leaf rust in wheat	*QLr.hwwgru-2DS*, *QLr.hwwgru-7BL* and *QLr.hwwgru-7AL*	2DS, 7BL and 7AL, respectively	Lu (2016)
	Leaf + stem + stripe rust in wheat	*Lr34/Yr18/Sr57* + *Sr2/Yr30*	7DS	Randhawa et al. (2018)
	Leaf + stem + stripe rust in wheat	*Lr46/Yr29/Sr58*	1B	Randhawa et al. (2018)
Slow mildewing	Powdery mildew in barley	*er1*	LG 6	Humphry et al. (2011)
		Rbgnq2/Rbghq1	–	Romero et al. (2018)
	Downy mildew in cucumber	*QTL dm5.2*	Chr 5	Zhang et al. (2018)
Slow blighting	*Ascochyta* blight in pea	*mpIII-1*, *mpIII-3*, *mpVa-1* and *mpVII-1*	First two QTLs on LG III, next one QTL on V and VII	Prioul et al. (2014)
	Septoria nodorum blotch in wheat	*QSnb.nmbu-2A.1* and *QSnb.nmbu-5A.1*	2 A & 5A	Lin et al. (2021)
Slow root rotting	*Aphanomyces* root rot in pea	*Ae-7.6*, *Ae-Ps4.1*, *Ae-Ps3.1*, *Ae-Ps2.2*, and *Ae-Ps1.2*	–	Hamon et al. (2011)
		IV.11, *IV.12* and *VII.18*	–	Desgroux et al. (2016)
Slow wilting	*Fusarium* wilt in chickpea	*H1* (syn *foc-1*) *H2* *H3*	–	Singh et al. (1987); Sharma and Muehlbauer (2007)
		GSSR 18-TC14801	–	Jingade and Ravikumar (2015)
Slow blasting	Blast in rice	*Pi9*, *Pi2* and *Piz-t*	–	Jiang et al. (2012)
		Pigm locus	–	Deng et al. (2017)
		Pi21, *Pi35*, *Pi63*, *Pb1* and *Pid3-I1*	–	Ning et al. (2020)

could possibly erode to some extent over time due to interactions among the components of SDD and the parasitic fitness. Rosewarne et al. (2008) suggested that the factors determining SDD resistance and epidemiological fitness are inherited quantitatively. To strengthen confidence in this conclusion, a larger number of families could be required for more accurate estimations of the genetic correlations (Bus et al. 2006).

Modifier genes

Modifier genes can either enhance or suppress the expression of another resistant gene e.g., the *Lr34* gene acts as a modifier for various genes such as *Lr12*, *Lr13* and *Lr16*. Such modifiers are able to interact with other genes to foster effective resistance against different races of the leaf rust (Cristina et al. 2015). Similarly, Huckelhoven et al. (2000) revealed that two additional genes viz., *Ror1* and *Ror2* are needed for expression of race nonspecific resistance (conferred by recessive *mlo* alleles) against the barley powdery mildew. Genetic interaction between more than two genes is known to provide higher resistance as compared to resistance conferred by the individual genes (McCallum and Hiebert 2022). Analysing the role of modifier genes can be achieved by comparing transformed isogenic lines with resistance genes, along with modifier genes and another line lacking modifier genes.

In some hosts the expression of a strong resistance gene has been found to be suppressed by the presence of partial or complete inhibitor genes present in either homozygous dominant/heterozygous condition. For instance in the wheat cultivar Thatcher a suppressor gene was observed to inhibit the expression of the resistance gene '*Lr23*' when infected with isolates of *Puccinia recondita* in Canada. However it only partially inhibited the *Lr23* gene when infected with Australian isolates (McCallum et al. 2016). It's worth noting that a resistance gene under the influence of a modifier gene can affect other traits than resistance. Different combinations of modifier genes can affect SDD by influencing the expression and effectiveness of SDD genes. These combinations can enhance or suppress resistance, interact in complex ways, and lead to varying levels of disease progression. Therefore, discovering and understanding the role of modifier genes is crucial due to their fundamental and economic significance.

Co-localized genetic loci

Multiple disease resistance can contribute to fitness in host plants infected with multiple diseases. Wisser et al. (2011) identified a candidate gene 'GST' in maize that confers resistance against three pathogens: grey leaf spot, southern leaf blight and northern leaf blight. QTLs present within 20 cM intervals are considered to be co-localized. Furthermore, these co-localizing genomic regions contain specific functional groups of genes involved in other biotic/abiotic stress tolerance mechanism in various crops e.g., Schweizer and Stein (2011) identified 20 meta-QTLs, including eight hot spots conferring resistance to different diseases in barley. Vatter et al. (2018) suggested the importance of nested association mapping (NAM) population in meta-QTLs analysis conferring resistance against stripe and leaf rust diseases in barley.

It is now realized that co-occurring resistance loci result in enhanced host resistance e.g., in wheat, the 2A + 2D QTL combination contributed more durable resistance against different races of stripe rust pathogen (Maree et al. 2019). Ye et al. (2022) suggested three co-located pleiotropic genes (*Yr30, Yr17 and Lr46/Yr29*) conferred durable resistance to yellow rust and leaf rust in a wheat variety 'Borlaug 100'. Different resistance phenotypes were conferred by distinct and different interactions between QTL combinations. Hence, it is very necessary to identify the best QTL combinations providing multiple race resistance. The knowledge of co-occurring resistance loci will help breeders to deploy potent and manifold disease resistance profiles. In linseed, five loci (K, L, M, N and P) were found to confer resistance against flax rust (Lawrence et al. 2010). Genes at four of the loci (L, M, N and P) are translated into resistance proteins of the Toll interleukin 1 receptor–nucleotide binding site–leucine-rich repeat (TIR-NBS-LRR) class. Besides, the P locus encodes a protein with an additional C-terminal domain present at 150 amino acids downstream of the LRR region. Co-localized QTLs providing multiple disease resistance will not only intensify resistance, but also extend their effectiveness (Maree et al. 2019).

Breeding strategies to enhance resistance through SDD components

Identifying quantitative trait loci (QTLs) with small effects, even with whole genome sequences available, can be challenging. Additionally, achieving transferable combinations with consistent effects across different genetic backgrounds adds another layer of complexity. To propose a route for slowing disease development, a multi-faceted approach combining traditional breeding techniques with modern genomic tools and integrated disease management strategies could be effective: genomic selection, marker-assisted selection (MAS) pyramiding of QTLs, genome editing, integrated disease management and continuous monitoring and adaptation. Considering the multigenic nature of SDD components and its inconvenience to phenotype, improved breeding methods and strategies for SDD could be useful. The incorporation of enhanced components to SDD can lead to more resilient and productive agricultural systems, benefiting both farmers and the environment in the long run. The successful use of resistance through SDD came from the strategic use of different slow rusting genes e.g. Lr68, Lr34, Lr67, Lr46, and Sr2 in wheat. These genes are considered to act as backbone genes for imparting durable leaf rust resistance in wheat cultivars since the green revolution and still function in the current Mexican wheat germplasm, especially when present in combination with other major genes and/or with minor genes (QTLs) or modifiers (Ellis et al. 2014; Huerta-Espino et al. 2020).

Stability in disease resistance is of utmost importance to attaining stable yield, especially in epidemic prone regions. Minor-gene resistance has no or minimal gene-for-gene interaction (Bekele et al. 2019). Consequently, breeding for durable minor-gene resistance can help in reducing Genotype × Environment (G × E) interactions of resistance and improve breeding efficiency. A number of studies have shown that no single component of SDD alone could effectively provide the desired level of resistance in a genotype, rather a combination of SDD components would be desirable for an efficient and effective disease resistance breeding program (Parlevliet 2002; Singh et al. 2015). Although, improvement in resistance level through a single SDD component can

reduce AUDPC (Habtu and Zadoks 1995; Negussie et al. 2005), more components may be used as selection criteria for selecting a particular resistant genotype (s). The selected genotypes may be utilized in resistance breeding programs as parents to obtain high yielding lines with durable resistance. The following breeding methods have been applied:

Shuttle breeding

Shuttle breeding programs were originated by N. Borlaug to evaluate the segregating materials with the aim to cut short the time required to develop a variety from 10–12 years to 5–6 years (Tadesse et al. 2019). The success of any shuttle breeding program depends upon the prevailing disease, availability of resistant genotypes, screening methodology under field conditions at seedling or adult plant stage, replicated field trials or the use of molecular markers. The inclusion of check varieties for resistance/susceptibility is important to assess the level of disease and degree of resistance as well. The selection of field sites with appropriate environmental conditions is crucial for success of any shuttle breeding program under field conditions. The wheat breeding program carried out by CIMMYT is a success story that brought about the green revolution in Mexico, India, Pakistan and Turkey in 1960s. Another example is the shuttle breeding in mungbean between the World Vegetable Center in Taiwan and the Nuclear Institute for Agriculture and Biology (NIAB) in Pakistan, which enabled scientists to develop improved Mungbean Yellow Mosaic Disease (MYMD) resistant varieties for South Asia region (Shanmugasundaram et al. 2009). Similarly, Patocchi et al. (2020) accentuated the need of shuttle breeding in identification of most desirable scab resistant gene combinations for different geographic regions in apple.

Multiline approach

Multiline is a mixture of agronomically superior near-isogenic lines (NILs) differing for the single gene (resistant gene) at the corresponding loci. However, it doesn't confer resistance against all prevailing races of the pathogen. Nevertheless, a multiline cultivar provides higher level of resistance than an 'isoline' cultivar, as multiline contains resistance to some of the races and is more adaptive to environmental fluctuations than the individual component lines e.g., multilines of transgenic wheat *Pm3a*, *Pm3b* and *Pm3d* lines showed higher resistant to powdery mildew than their individual component lines (Brunner et al. 2012). In the event of disease outbreak, only few of the component lines get invaded, while others remain resistant. The heterogeneity of partial resistant genes helps stabilize the spread of pathogen inoculum, thus reducing on-farm yield losses. Furthermore, it is realised that epidemic development has been reduced as majority of CIMMYT wheat germplasm carries one to four slow rusting genes. The combination of such SDD gene complexes with additive effects needs to be incorporated into a cultivar. Currently, availability of molecular markers enables the rapid and efficient introgression of the gene of interest into the adaptable genotypes. However, some flaws are associated with use of multiline varieties as all the lines constituting a multiline variety may be attacked by the new race of a pathogen, must mature at the same time and it is also not suitable for cross pollinated crops.

Gene pyramiding

Gene pyramiding is an efficient approach to enhance durability of resistance by introgressing multiple resistant genes or QTLs into a single genotype creating a genetic pyramid. Pyramiding resistant genes with high or low magnitude from multiple sources acting different stages of host–pathogen interaction is expected to be long lasting over time than relying on single sources (Hu et al. 2008; Luo et al. 2020). Conventionally, transferring desirable resistant genes in the recurrent parent requires a minimum of six generations to recover approximately 99.2% of the recipient parent genome. However, the advent of molecular marker use allows early generation selection of QTL-linked markers with similar phenotypic expression in less time as compare to conventional methods (Dormatey et al. 2020). For instance, Imam et al. (2014) demonstrated the presence of multiple genes (*Piz-t*, *Pita*/*Pita-2* and *Pi9* genes) in rice accessions from N-E and Eastern India related to slow blasting using SNP markers. Identifying effective SDD genes is a prerequisite for initiating an SDD gene pyramiding program. Various researchers dissected genes from the newly observed QTLs to strengthen SDD genetic resources for slow rusting in wheat (Muhammad et al. 2020) and slow blasting in rice (Imam et al. 2014). Marker-aided gene pyramiding is also cost-effective and technical collaboration makes this more affordable for developing countries to use for local germplasm improvement. Various durable resistance pyramids have been developed mainly introgressing major resistance genes. In some instances, a combination of major and minor genes has been introgressed e.g., transfer of major gene *Lr24* along with the slow rusting gene *Lr48* via marker-assisted breeding in wheat (Samsampour et al. 2009; Pal et al. 2022). Similarly, stem rust (*Yr15*) and two leaf rust resistance (*Lr19/Sr25* and *Lr24/Sr24*) genes were introgressed into wheat cultivar 'UP 2338' background to provide durable resistance against the stem rust pathogen (Singh et al. 2018). Thus, identified and cloned slow disease genes or QTLs can be used for gene pyramiding to attain sustainable crop

production. For the isolation of minor disease resistance genes or QTLs, candidate gene method has proven to be an efficient and rapid approach (Hu et al. 2008).

Kaushal et al. (2024) suggested that pyramiding resistance of two or three minor QTLs can achieve the same or even a higher level of resistance than that governed by a major resistance gene. The importance of pyramiding of minor genes for disease resistance has been reviewed by Mundt (2018). In resistance pyramids, minor genes result in less selection pressure on pathogen populations as resistance is incomplete and expressed only during a particular part of the host's life cycle. Furthermore, different selection pressures against the pathogen may be also due to difference in biochemical characteristics of major and minor genes and their involvement in different defense signal transduction pathways. In fact, some minor genes do not belong to the NB-LRR class that is more common for major genes (Ellis et al. 2014).

Genomic selection

Genomic selection (GS) is one of the promising approaches to improve prediction accuracy and genetic gain for complex traits, such as quantitative disease resistance, as it does not required preliminary specification of genes/QTLs governing desirable attributes. It assists in predicting breeding value of untested genotypes by using genome-dense markers (Bekele et al. 2019). Rutkoski et al. (2015) implemented GS to reduce time of breeding cycle by up to twofold during the introgression of genomic regions associated with wheat stem rust resistance governed by both major and minor genes. GS enables the selection of slow disease resistance genes even in the presence of major resistance gene and can evaluate large number of genotypes with a higher rate of selection intensity, thus resulting in increased genetic gain (Olatoye et al. 2019). The finding of Juliana et al. (2022) indicated moderately high mean genomic prediction accuracies of 0.53 and 0.40 within and across breeding panels, respectively which were on average 177.6% and 60.4% higher than the mean accuracies from fixed effects models using selected spot blotch loci.

In GS, a training population (consisting of individuals with well documented phenotypic and genotypic data) is needed to appraise breeding value of the breeding population (consisting of only genotyped individuals). Moreover, the accuracy of genomic prediction can be enhanced by adding new lines derived from the current germplasm to the training population. Various models and algorithms have been employed to improve accuracy of genomic prediction such as genomic best linear unbiased prediction (GBLUP), Bayes and machine learning (Wang et al. 2018). Juliana et al. (2017) compared three different genomic prediction models to reckon the breeding value for APR to leaf rust, stem rust and stripe rust in wheat. They further stated that, application of genome-wide marker-based models maximizes the genetic gain as compared to Marker Assisted Selection. Thus, GS is a new prospective approach for combining favourable alleles to improvement quantitative disease resistance and can be executed in the advanced breeding genotypes.

Multilocation trials

The success of any resistant breeding program relies on the assessment of environmental variability and the adaptability of the cultivars to cope up with adverse climatic conditions. Various international research institutes, such as CIMMYT, ICRISAT, IRRI, World Vegetable Center etc., as well as national breeding institutions and regional cooperative agricultural research units, conduct multi-location trials (MLTs) to identify cultivars adapted to the increasingly variable environmental conditions (Braun et al. 2010). These networks are based on free access and exchange of germplasm worldwide for crop improvement. The genetic response of the cultivars to aberrant climatic conditions limits the accuracy of yield estimations. Vazquez et al. (2012) suggested that epistasis and genetic background are the major influences on expression of complex traits, including slow disease development. Therefore environmental factors play a crucial role in aggravating the disease severity. Vazquez et al. (2015) assessed the genetic variability to slow rusting to stripe rust in wheat RILs population across eight locations. Analysis across locations needs to be done to estimate genotype × environment (GE) interactions. Alternatively, the factor regression model is also used to split the GE interaction in order to provide some elements of a biological explanation of GE interaction for yield. Recently, Sankar et al. (2021) unravelled GE interactions to identify and validate stable resistant pearl millet genotypes against blast disease via multi-location testing. Also Srinivas et al. (2023b) identified wheat varieties with durable and broad-spectrum rust resistance using multi-environment phenotyping. Hence multi-environment testing is considered as a potent tool in selection of cultivars with SDD before releasing it for farmer's practice.

Future perspectives

Accurate and detailed information is crucial to understand SDD components to impart durable disease resistance against major diseases in globally important major crops. Moreover, it is also important to explore the inheritance of SDD for minor diseases of major crops e.g., *Septoria tritici* blotch, head blight, tan spot in wheat. Several studies successfully demonstrated the great potential of the SDD components in predicting and selecting for quantitative disease resistance. Most findings suggest

a preponderance of additive gene action for conferring resistance through slow disease development. Therefore, selection for SDD components can enable improvement in breeding lines. Since, the effect of an individual SDD component may be small but most of the components are governed by additive gene, they may be used in combinations to obtain a desirable level of resistance. In the current scenario, breeders target development of cultivars conferring resistance to multiple diseases. This can be possible by identifying meta-QTLs. Wild accessions act as great reservoir of allelic richness that can be exploited to identify various SDD-QTLs or genes using various mapping strategies. This can help broaden the genetic base of susceptible genotypes through gene pyramiding, multiline development or genome editing technologies. Furthermore, researchers need to identify co-localized resistance loci to sustain the effectiveness of SDD genes or QTL over time for durable resistance. Thus, SDD components proved to be a valuable tool for crop resistance breeding programs in changing climatic conditions.

Supplementary Information

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Supplementary Material 1

Author contributions

R.C., B.R.S., R.M.N. and A.K.S. conceived the outline of the review article. K.S., S.S., N.K.M. and A.K.S. helped in writing the article. K.S., S.S. and N.K.M. prepared the tables. R.C., B.R.S., R.M.N. and A.K.S. finalized the article. All authors reviewed and approved the article.

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Data availability

It is a generalised statement based upon the studies on slow disease development components but any speciic data is not available.

Declarations

Competing interests

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