PLANT SCIENCE TODAY
Vol 12(sp1): 1-12
https://doi.org/10.14719/pst.8883

eISSN 2348-1900

REVIEW ARTICLE

Genetic determinants underlying major disease resistance in sunflower: A review

V Poovitha[1], R Sasikala[2*], R Kalaiyarasi[2], L Rajendran[3] & E Sumathi[4]

[1]Department of Plant Breeding and Genetics, Agricultural College and Research Institute, Tamil Nadu Agricultural University, Madurai 625 104, Tamil Nadu, India

[2]Department of Oilseeds, Centre for Plant Breeding and Genetics, Tamil Nadu Agricultural University, Coimbatore 641 003, Tamil Nadu, India

[3]Department of Plant pathology, Tamil Nadu Agricultural University, Coimbatore 641 003, Tamil Nadu, India

[4]Department of Agricultural Entomology, Tamil Nadu Agricultural University, Coimbatore 641 003, Tamil Nadu, India

*Correspondence email - sasikalacpbg@gmail.com

Received: 15 April 2025; Accepted: 24 June 2025; Available online: Version 1.0: 31 July 2025; Version 2.0: 15 August 2025

Cite this article: Poovitha V, Sasikala R, Kalaiyarasi R, Rajendran L, Sumathi E. Genetic determinants underlying major disease resistance in sunflower: A review. Plant Science Today. 2025; 12(sp1): 1-12. https://doi.org/10.14719/pst.8883

Abstract

Sunflower (*Helianthus annuus* L.) is an important oilseed crop worldwide, valued for its high-quality edible oil which is a rich source of unsaturated fatty acids and rich in vitamin E. It is widely preferred due to its adaptability to diverse agro-climatic conditions and short growing period. Statistical reports from Indiastat revealed that the sunflower cultivation in India covers 1.25 % of the global area and contributes around 0.58 % of the world's production. However, its productivity is often hindered by many biotic stresses, especially diseases ranging from 30 % to 70 %. Although pest infestations do occur, diseases are considered more critical due to their rapid spread, persistent nature and potential for severe crop losses across various growing seasons. Considering all the factors, effective disease management isthus essential to sustain and enhance sunflower production, ensuring food security and economic stability for farmers. It is essential to comprehend the genetic factors that influence disease resistance to create cultivars that are high-yielding and durable and to improve crop sustainability along with reduced fungicide need. The promise of developing sunflower cultivars with broad-spectrum and long-lasting resistance lies in the integration of conventional and molecular approaches such as detection of resistant quantitative trait loci (QTLs), utilisation of wild species and the application of genomic selection strategies. This review consolidates existing insights into the genetic keystones ofdisease resistance in sunflower and highlights how wild *Helianthus* species serve as important reservoirs of resistance genes.

Keywords: diseases; omics; resistant QTLs; sunflower; wild species

Introduction

Sunflower (*Helianthus annuus* L.), is an annual, monoecious, dicotyledonous plant that belongs to the family of *Asteraceae* (1), with chromosome number 2n = 34 (2) and has an estimated genome size of 3000 Mbp (3). The term "*Helianthus*" has originated from the Greek words "*Helios*," which means sun and "*Anthus*," which signifies flower. The sunflower exhibits an astonishing behaviour of following the sun's movement hence it is called "Girasol" in Spanish and "Tournesol" in French (4). Sunflower ranks fourth among the oil-producing crops after oilpalm, soybean and canola (5). Sunflower is one of the important oil crops and contributes to about 12 % of the global vegetable oils and 10 % of the total edible oil (FAOSTAT, 2018). Its utility can be seen in its farming regions from the sub-tropical to sub-arctic regions amid other areas where it is exploited for the preparation of high-quality consumable oils (6). Sunflower oil has been viewed as a healthy vegetable oil for ages and it is considered premium oil for salad, cooking and margarine production and is also used as a source of biodiesel in recent times. Sunflower is primarily an oil crop, with high protein meal as a by-product. Sunflower pellet production is also important among the nations, as it serves as the principal grinding sub product. With the increasing demand for oil production next to food production, sunflower plays a vital role in meeting the demand and supply of edible oil and satisfying economic and dietary needs. Sunflower oil is rich in polyunsaturated linoleic acid by nature which contributes about 70 % of the total sunflower oil content and this is followed by monounsaturated oleic acid which makes up about 20 % of total sunflower oil content (7). High-oleic acid content of sunflower oil lays the foundation for higher demand in the market along with health benefits of heart-healthy properties (8).

Sunflower is one of the fastest growing oilseed crop and India ranks second in sunflower production in Asia after China. The total area under sunflower cultivation in the world is about 26.42 million hectares, the production is about 54.45 million tons and the average yield is about 1.93 Mt/ha (USDA 2022-2023). India occupies an area of about 3.64 lakh hectares with the production of 3.63 lakh tonnes (Indiastat 2022-2023). In India, the major sunflower-growing states are Karnataka, Maharashtra, Andhra Pradesh, Tamil Nadu and Gujarat (9).

However, its production is frequently threatened by various fungal, bacterial and viral diseases, which can significantly reduce yield, oil content and overall crop quality. Among various diseases affecting sunflower, most devastating diseases are downy mildew (*Plasmopara halstedii*), powdery mildew (*Golovinomyces cichoracearum*), rust (*Puccinia helianthi*), Sclerotinia head rot (*Sclerotinia sclerotiorum*), Alternaria leaf spot (*Alternaria helianthi*) and Sunflower Necrosis Virus (SFNV).

For an instance Sunflower Necrosis Disease (SND) was noted as an epidemic disease in southern states of India during 1997-1999, with incidence ranged from 10 % to 80 % and yield loss upto 90 %. SND had caused a steep decline in cultivation of sunflower in India from 2.34 million hectares during 2005-06 to less than 2.5 lakh hectares (DOR Annual report, 2001). Sunflower yield is also affected to nearly 50 % by downy mildew, 30-74 % by powdery mildew, nearly 80 % by rust, nearly 60 % by head rot and 27-80 % by leaf spot (4). Hence, knowledge on controlling of sunflower diseases under threshold level becomes a key area of concern.

Managing these diseases effectively requires a combination of cultural practices, chemical control and most importantly, the development of disease-resistant sunflower varieties. Traditional breeding, genetic modification and molecular marker-assisted selection have played a crucial role in enhancing sunflower resistance to major pathogens. Resistance genes (R-genes) have been identified for key diseases such as downy mildew and rust, enabling breeders to develop hybrids with improved tolerance. This article explores the major diseases affecting sunflower, their impact on production, the genetic basis of resistance and recent advancements in breeding strategies that contribute to improving disease resistance in this economically important crop.

Major diseases of sunflower and their impacts

Major diseases that affect sunflower and their causal organisms are listed in Table 1. Mode of spread of disease pathogens can be broadly classified into soil, air and vector borne. Different diseases are found to be spread by various modes which are mentioned in Fig. 1.

Downy mildew

Symptoms

The most severe symptoms in the field are caused by root infections in immature sunflower plants, which can significantly affect yield (10). Damping off of sunflower seedlings, short stature of plants, leaf bleaching from veins and observable white sporulation starting on the underside of cotyledons and leaves are some of the other major symptoms (11).

Effects and losses

Sunflower downy mildew, which is spreading in all major sunflower-growing countries of the world except Australia, has been considered to be the worst foliar disease that ever occurred (12). It often damages nearly 50 % of the plants and is responsible for more than 80 % loss of potential production (13).

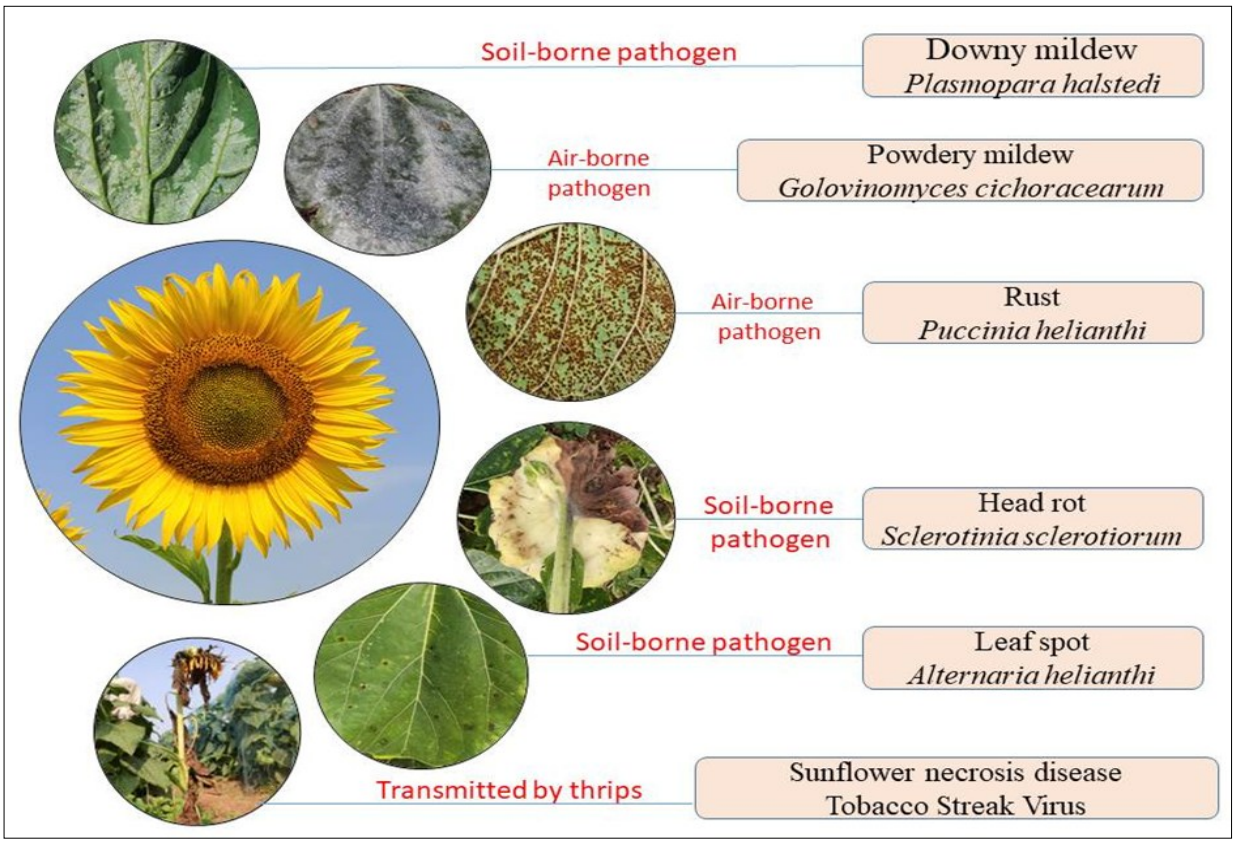

Fig. 1. Major diseases of sunflower with their causal organism and mode of spread.

Table 1. Major diseases of sunflower and their causal organism

Sl.No	Disease	Causal organism
1.	Downy Mildew	*Plasmopara halstedi*
2.	Powdery Mildew	*Golovinomyces cichoracearum*
3.	Rust	*Puccinia helianthi*
4.	Head Rot	*Sclerotinia sclerotiorum*
5.	Leaf Spot	*Alternaria helianthi*
6.	Sunflower Necrosis Disease	Tobacco Streak Virus

Disease cycle

The life cycle begins with overwintering oospores in the soil. In spring, these oospores germinate to produce zoosporangia, which release motile biflagellate zoospores into free water in the soil (14). Upon contacting sunflower roots, zoospores encyst and germinate within a few hours, infecting roots either by directly penetrating root cells (sometimes through the formation of appressoria or by entering through wounds), especially at the base of root hairs. Inside the root, the pathogen forms infection vesicles and spreads through the intercellular spaces of the cortex to colonize shoot tissues, where it develops haustoria to extract nutrients (11). During humid and moderately warm conditions in late spring and summer, the pathogen emerges on the undersides of leaves and cotyledons through stomata and produces branched zoosporangiophores that bear zoosporangia (10). These zoosporangia can disperse to nearby plants, releasing zoospores that encyst and penetrate leaf tissues, mainly at cell junctions using appressoria and more rarely through stomata. The progression of the pathogen within infected leaves is not yet fully understood. Secondary infections, which occur under cool and humid conditions, typically result in milder symptoms than those caused by primary root infections (11).

Disease diversity

Downy mildew pathogen was originally classified into two groups: the European race, considered the least virulent and the more aggressive North American or Red River Valley race (15). These were later designated as Race 1 and Race 2, respectively and were initially believed to be limited to their respective continents. In 1980, a third race emerged in the U.S., described and was sometimes called the “new” Red River Valley race (16). Subsequent discoveries included Race 4 in 1984 (17) and a highly virulent Race 5 in 1987 (18), both seemingly restricted to North America. By using a set of 12 public differential lines, researchers had identified at least 12 distinct races or virulence patterns by early 1996 (19). Interestingly, Races 4 and 5 were also detected in Germany (20). In Hungary, six races of *Plasmopara halstedii* had been identified, including Races 2, 4, 8 and 9. Globally, Race 1 is typically the most widespread. In the Indian state of Maharashtra, Race 1 has been reported (21).

Control measures

Choice of planting sites and disposal of infected crop residues also give a fairly good control. Maintaining a minimum four-year crop rotation is essential because the oospores remain viable for many years in the soil. Effective seed treatments include oxadixyl and benalaxyl, analogs of metalaxyl and to a lesser extent, acetamide and triadiazole compounds. Seed treatment with metalaxyl at the rate of 3 g per kg of seed has been found to give effective control (22). While metalaxyl completely suppresses aboveground symptoms and fungal sporulation, *P. halstedii* is still able to colonize portions of the seedling root. Metalaxyl applied as a soil drench was effective as an eradicant, but the rates would similarly be cost prohibitive for large-scale field applications. Both mycoparasitic fungi such as *Trichoderma* spp., *Gliocladium* spp. and *Pythium* spp. and bacteria, such as *Bacillus* spp., *Enterobacter* spp. and *Pseudomonas* spp., have been shown to control diseases caused by various *Pythium* spp. and *Phytophthora* spp. (14).

Powdery mildew

Symptoms

The symptoms of powdery mildew by *Erysiphe* and other genera during the early stages of the crop are little, dull-white, spherical spots on the leaf surfaces which later turn into black spots, or cleistothecia, may start to show up on the leaf toward the conclusion of the growing season. Eventually, white mycelium will cover the leaves. Larger, blended white to grey spots cover much of the plant. As the season progresses, the powdery, hazy mildew leaves turn brown. It has been shown that when leaves ripen, the infection shifts from the more infected, lower leaves to the more diseased, upper leaves. These patches of white fungal mycelium that are easily removed from both the upper and lower leaf surfaces (23).

Effects and losses

Over the past ten years, powdery mildew has become a serious issue and has quickly spread to all of India's sunflower-growing regions (15, 16). Although powdery mildew is found all over the world, depending on the severity of the disease, higher intensity is observed on sunflower in tropical regions and 30-74 % in India. One of the main issues with sunflower farming in the tropics and subtropics nowadays is powdery mildew (23, 24).

Disease cycle

The fungus *Golovinomyces cichoracearum* causes powdery mildew in sunflower and survives between seasons as conidia or cleistothecia (resting spores) on plant debris or alternative hosts. In favourable conditions - typically warm, dry days with cool, humid nights, the airborne conidia are released and land on sunflower surfaces, where they germinate without the need for free water (23). The fungus forms a superficial mycelium on the leaf surface and allows haustoria into epidermal cells to draw nutrients. It continues to reproduce asexually by producing more conidia, leading to rapid spread across the crop. The disease appears as white, powdery spots on leaves, stems and eventually flower parts, reducing photosynthesis and yield (24).

Control measures

Powdery mildew is controlled by applying protective fungicides such as sulfur, triazoles, or strobilurins at the early onset of symptoms. Avoiding excessive nitrogen fertilization can reduce the density of susceptible plant tissues. Additionally, ensuring adequate spacing between plants promotes airflow and reduces humidity, which can help suppress fungal development (22).

Rust

Symptoms

Rust symptoms include reddish-brown pustules that can merge and significantly reduce plant productivity. With a few notable exceptions, rust was frequently more common than 50 % in the locations under study and in some years, it was much more common (4).

Effects and losses

In a year with a rust pandemic, yield losses can reach 80 % when the atmosphere is conducive to the growth of rust diseases (4). One of the rare illnesses frequently found on sunflower is sunflower rust.

Disease cycle

Puccinia helianthi, the rust pathogen of sunflower, is an autoecious fungus that completes its life cycle on sunflower and overwinters as teliospores on crop residue. In spring, teliospores germinate to form basidiospores, which infect sunflower tissues and produce pycnia (yellow-orange humps on upper leaves) and aecia (orange cup-like structures on the underside). Aeciospores lead to the formation of urediniospores, which drive repeated infections throughout the season. Urediniospores appear as cinnamon-brown pustules (1-2 mm), commonly on lower leaves and are surrounded by a yellow, non-removable halo. They are wind-dispersed, germinate under moist conditions and enter through stomata. Late in the season, the cycle is completed by forming firm black telia (4, 22).

Disease diversity

Four races of cultivated sunflower in Canada were identified using three lines created by the Canadian Department of Agriculture (25). These were named Races 1, 2, 3 and 4. Nearly two thirds of the rust gathered in Manitoba in the 1950s was from Race 1, the least virulent race. The most virulent race at the time, Race 4, made up 2 % of the isolates, followed by Races 2 and 3. Using seven differentials, ten races were categorized in Argentina, which were tried to be related to races in North America (26).

Control measures

Fungicides such as triazoles or strobilurins should be applied at the first sign of disease, especially during periods conducive to rust development. Removing and destroying infected crop debris after harvest can significantly reduce the overwintering inoculum. Crop rotation with non-host plants also helps in breaking the disease cycle (22).

Head rot

Symptoms

Sclerotinia species can cause head rot and basal stem rot at any time, although they usually show symptoms right before flowering. A *sclerotinia* infection swiftly kills the plants, causing the crop to fail. Internally colonized, the fungus eats away at the internal tissues. During flowering, the fungus typically infects the back of the head. The tissues turn pale brown, spongy and squishy as the condition worsens. Eventually, only the fibrous strands at the back of the head and upper stalk remain due to this infection, which also spreads into the developing head and down the stalk. The sheer weight of the diseased seeds at the front of the head causes them to finally fall out of the head (4).

Effects and losses

Head rot affects both the yield and quality of seeds by directly producing severe effects on the capitulum. One of the most common diseases affecting sunflowers worldwide is *Sclerotinia* head rot. For *S. sclerotiorum* in particular, complete resistance is not found but there has been a 60 % reduction in susceptibility since 1970 and, as no isolate-variety interactions exist, this resistance should be durable (4).

Disease cycle

Sclerotinia sclerotiorum, the fungus causing head rot in sunflower, survives in soil as hard, black resting bodies called sclerotia. Under cool, moist conditions, especially during flowering, sclerotia germinate to form small mushroom-like structures (apothecia) that release airborne ascospores. These spores land on sunflower heads, especially on senescing tissues or wounds and germinate in the presence of moisture. The fungus invades through florets and spreads through the head tissue, forming soft, white, cottony mycelium and producing new sclerotia inside or on the sunflower head. These sclerotia return to the soil at harvest and can remain viable for many years, repeating the cycle (4).

Control measures

Control measures include crop rotation with cereals or other non-hosts for at least three years to reduce soil borne sclerotia. Avoiding overhead irrigation or excessive moisture during flowering is crucial, as the disease favours humid conditions. Fungicides such as mancozeb or chlorothalonil can be applied. Biological control agents such as *Coniothyrium minitans* may help reduce sclerotia viability. Timely fungicide applications during flowering can also be effective, especially in high-risk weather conditions (22).

Leaf spot

Symptoms

The distinctive feature of *Alternaria* leaf spot disease is the appearance of dark brown to black, round to oval spots on leaves, stems and the calyx of flowers. These spots are surrounded by necrotic chlorotic zones with a gray-white necrotic center surrounded by concentric rings and a yellow halo (4). Severe infections cause lesions to coalesce and become irregular, which causes blight and the plant's mortality.

Effects and losses

In India, yield losses range from 27 % to 80 % because of sunflower leaf spot and affect the seed quality and its germination rate (4).

Disease cycle

Alternaria helianthi, the causal agent of sunflower leaf spot, survives in infected plant debris or seeds as conidia or mycelium. In favourable conditions (warm temperatures and high humidity) the conidia are wind- or rain-splashed onto sunflower foliage. The spores germinate and directly penetrate leaf tissue, forming lesions that expand and produce concentric rings. The fungus sporulates again from these lesions, releasing more conidia for secondary infections. Repeated cycles during the season can lead to severe defoliation, especially under prolonged wet conditions. Infected residues left in the field or infected seeds serve as the primary source of inoculum for the next season (4).

Control measures

Foliar fungicides such as mancozeb or chlorothalonil should be applied when symptoms first appear. Rotating with non-host crops and removing infected plant debris at the end of the season are essential practices to reduce inoculum carryover and disease incidence in subsequent seasons (22).

Necrosis

Symptoms

The characteristic field symptoms include mosaic pattern, extensive necrosis of leaf lamina, petiole, stem, floral calyx and complete death of plants ultimately. Early infection either kills or causes serve stunting of plants, malformation of heads and chaffy seeds. Necrosis at bud stage makes the head to bend and twist and results in complete failure of seed setting.

Necrosis leads to complete death of the plant by affecting all the parts of the plant (27).

Effects and losses

The majority of hybrid sunflowers being grown in India have demonstrated varying degrees of disease vulnerability for sunflower necrosis (27). The production of sunflower has been practically threatened in later years by outbreaks of this disease in key sunflower-growing states of India, particularly Andhra, Karnataka and Maharashtra. Reports of yield losses ranging from 30 % to 100 % have been made (28).

Disease cycle

Sunflower necrosis is observed at all growth stages initiating from seedlings to harvest. The disease is caused by tobacco streak virus (TSV) (29) and it was found to be transmitted by thrips (30). The condition is caused by a virus, making it difficult to treat with a single strategy. Tobacco streak virus (TSV), which causes necrosis in sunflower, is primarily transmitted by thrips in a non-persistent manner, meaning the insects acquire and transmit the virus quickly while feeding on infected pollen or plant sap (31). Once inside the plant, TSV spreads systemically through the vascular tissues, leading to symptoms such as necrotic lesions on leaves, stems and flower heads, stunted growth and complete plant death. Thrips moving between infected weeds and sunflowers help spread the virus during the growing season. Weed hosts and volunteer sunflower plants often act as reservoirs of the virus between cropping cycles (30).

Control measures

Necrosis is mainly spread by thrips, hence vector control is crucial. This includes monitoring and reducing thrips populations using systemic insecticides, such as imidacloprid which can be effective against thrips that burrow into plant tissue. Avoiding the proximity of sunflower fields to alternate host crops like cotton and groundnut can reduce the risk of virus spread. Using virus-free seeds and resistant hybrids, as well as maintaining field hygiene.

Majority of the sunflower production worldwide is affected by these diseases and hence development of resistant lines is essential to improve the efficient sunflower cultivation.

Breeding strategies to improve disease resistance

Breeding strategies for disease resistance in sunflower (*Helianthus annuus L.*) have undergone significant advancement, integrating classical methods with modern genomic tools. Initially, conventional breeding relied on phenotypic selection focusing on visual assessment of disease symptoms in field trials. From this a breakthrough came with the utilization of wild *Helianthus* species, which serve as a rich source of resistance genes. For example, *Helianthus argophyllus* and *H. tuberosus* have been exploited to introgress resistance genes against *Plasmopara halstedii* and *Sclerotinia sclerotiorum* (32). This method, though foundational, was limited by environmental influence and the time consumed to develop resistant lines by conventional breeding is longer. Hence, scientists adopted molecular breeding techniques to overcome these limitations. The development of quantitative trait loci (QTLs) has significantly improved our understanding of disease resistance genetics. For instance, the identification of *Pl* resistance genes from wild relatives has played a vital role in downy mildew management. Coupled with MAS, the application of molecular markers such as SSRs, SNPs and In/Dels has enabled breeders to select for resistance alleles with greater precision and efficiency (33-35). To enhance mapping resolution and dissect complex traits, multi-parent advanced generation inter-cross (MAGIC) populations have been developed. These populations capture greater allelic diversity and recombination events compared to biparental crosses (36).

Furthermore, omics approaches including genomics, transcriptomics, proteomics and metabolomics have deepened our insights into the molecular mechanisms of resistance. Proteomic profiling has highlighted stress-responsive proteins, while metabolomics has uncovered phytohormones associated with resistance responses (37). Advancements in next-generation sequencing (NGS) and genome-wide association studies (GWAS) have further accelerated the identification of candidate genes across diverse germplasm collections. Recently, gene editing tools such as CRISPR/Cas9 have been proposed for precise manipulation of resistance genes, although their application in sunflower is still in early stages. Nonetheless, these combined strategies are shaping a new era of precision breeding, aiming for durable, broad-spectrum resistance against evolving pathogen threats in sunflower (38). Various breeding strategies employed in sunflower are depicted in Fig. 2.

Resistance mechanisms offered by host

It has been found that INA (dichloroisonicotinic acid) and BABA (β-aminobutyric acid) might cause systemic resistance to downy mildew in sunflower, also BTH (benzo(1,2,3)-thiadiazole-7-carbothioic acid S-methyl ester) could cause resistance. According to findings, induced resistance is a practical strategy for managing sunflower downy mildew (39).

The antioxidant enzyme activities of resistant genotypes are substantially higher than those of susceptible genotypes when it comes to host defense response mechanisms against powdery mildew in sunflower. Transcripts of *NPR1* gene, the master regulator of the salicylic acid-based resistance signalling that contributes to plants' resistance against biotrophic pathogens and *hsr203J* gene, which has been implicated in cell death, were found in greater amounts in the resistant genotypes (40).

It was discovered that pathogen infection in sunflower changed the activities of several antioxidant enzymes. Furthermore, head rot (*S. sclerotiorum*) infection significantly increased the concentration of two stress-inducible phyto-hormones, such as salicylic acid and abscisic acid. Similarly, after infection, the resistant plants' leaves had considerably higher levels of total protein, carbohydrates and chlorogenic acid than the susceptible plants', suggesting a positive relationship between resistance and these suggestive indicators (41).

Resistant genes identified for major disease resistance

Understanding the genetic basis of disease resistance in sunflower is the key to effective breeding strategies. Resistance genes in sunflower can be identified using a range of genetic and biotechnological methods. One of the most widely used approaches is QTL mapping, which involves crossing resistant and susceptible genotypes to develop mapping populations, followed by phenotyping for disease response and genotyping using molecular markers to locate genomic regions associated

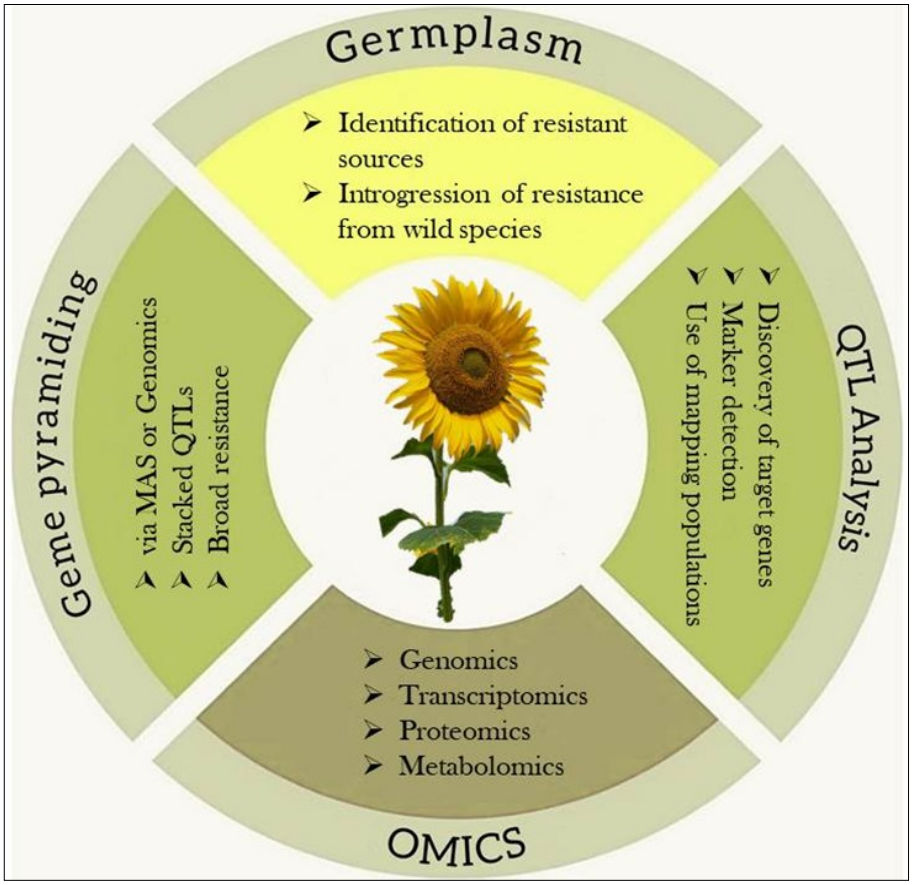

Fig. 2. Breeding strategies and tools for disease resistance in sunflower.

with resistance traits. GWAS offer another powerful tool, particularly for traits controlled by multiple genes, by examining natural populations to establish associations between phenotypic variation and specific genetic markers such as single nucleotide polymorphisms (SNPs) (38). Comparative genomics further aids in resistance gene identification by analyzing conserved regions between resistant and susceptible genotypes or related species. Transcriptomics, particularly RNA sequencing (RNA-Seq), provides insights into genes that are differentially expressed during pathogen attack, helping to pinpoint those involved in defense responses (42). Additionally, mutagenesis followed by Targeting Induced Local Lesions IN Genomes (TILLING) can be used to generate and identify functional mutations in candidate genes (43). Functional validation of resistance genes is often achieved through genome editing tools such as CRISPR-Cas, allowing direct assessment of gene function. Once identified, these resistance genes can be efficiently incorporated into elite cultivars using marker-assisted selection or marker-assisted backcross breeding and even pyramided to develop broad-spectrum and durable disease resistance in sunflower.

Several resistance genes have been mapped to specific linkage groups in the sunflower genome. Molecular markers linked to these genes aid in precise and efficient selection of genotypes during breeding process. For an instance, in case of downy mildew resistance, nearly 27 resistant genes have been identified and majority of them are associated with linkage group 8 and 13 (44). Similarly powdery mildew resistant genes were identified in linkage group 10 (9, 23) and in head rot resistance, 16 genomic regions were majorly identified in 8 chromosomes (45). These genetic tools are essential for developing resistant varieties through marker-assisted selection. Various genes conferring resistance against major diseases along with their linkage group and markers associated with them are mentioned in Table 2.

Resistant sources for major disease resistance in wild populations

Wild species of sunflower possess diverse traits that are often absent in cultivated varieties. Hence, they serve as valuable genetic resources for improving disease resistance in advanced sunflower varieties/hybrids. Identifying these species helps in broadening the genetic base of sunflower breeding programs. Among various wild *Helianthus* spp., *Helianthus argophyllus* (46) and *Helianthus debilis* (47) contribute resistance to majority of the diseases such as downy mildew, powdery mildew, rust and were widely utilised in pre-breeding programme by possessing relatively good compatibility. Various wild species possessing resistance against major diseases are listed in Table 3.

Molecular markers and mapping approaches

Using RAPD markers, the first map of wild sunflower was created during 1993 (48). A few years later, maps were created and released utilizing RFLP markers that were not based on PCR in various cultivated sunflower crosses (49).

The maps were then updated to include AFLP markers (50). The number of haploid chromosomes in sunflowers is represented by the 17 linkage groups (LG) found in most sunflower linkage maps. Genetic maps based on SSR markers came after these maps (51).

The first composite genetic SSR map covered 1423 cM and included 379 public and proprietary markers in addition to 278 single-locus SSR markers. In order to investigate three new

Table 2. Genes conferring resistance against major diseases of sunflower

Sl. No	Linkage group	Genes identified	Markers associated	Reference
			Downy Mildew	
1.	LG1	*Pl35*	NSA_006938, NSA_005423, NSA_009758 & NSA_000630,	(76)
		Pl arg	NSA_007595, NSA_001835	(74)
2.	LG2	Pl_5	ORS-316, ORS-630	(44)
		Pl18	CRT214 & ORS203	(70)
			SFW03013 & SFW03060	(76)
3.	LG4	*Pl19*	ORS963 & HT298	
		Pl_{33}	ORS644, ORS963, SFW04901 & SFW04052	(71)
			NSA_006089, NSA_008496	
4.	LG 8	*Pl20*	SFW02745, SFW09076, S8_11272025,	
			S8_11272046	(74)
		Pl20	SFW01920 & S8_100385559	(76)
		Pl_6	ORS328	(66)
		Pl_8	ORS781	(44)
		Pl_1	SCT06 (950 b)	(69)
		*Pl*15	SFW01920, SFW00128, SFW05824 NSA_008457	
5.	LG9	Pl_2	ORS-333	(44)
			SFW-00211 & SFW-01272	(65)
6.	LG11	Pl_{12}	CRT-275 & ZVG-53	(68)
7.	LG13	*Pl*13a, *Pl*13b	ORS-316	
		Pl_4	ORS-316	
		Pl_4u	ORS-799, ORS-45	(44)
		Pl_{11}	ORS-728 & ORS-45	(67)
		Pl har6	ZVG-61 & ORS-581	(75)
		Pl arg	ORS662, ORS509, ORS-316, NSA-001392, NSA-002798	
		Pl8	SFW01497, SFW06597	
		Pl8	SFW05743	
			Powdery Mildew	
8.	LG 5		ORS 1028, ORS 538	(23)
9.	LG 10		ORS 691, ORS 853, ORS 78	(23)
			ORS 1110, ORS 684	(9)
			Rust	
10.	LG 2	R_5	SFW01272, NSA_007071, NSA_001605, SFW03654	(67)
11.	LG 8	R_1		(65)
		R_{15}		(69)
12.	LG 11	R_{12}		(79)
		R_{14}		(80)
13.	LG 13	R_4	NSA_000267, ORS316, ZVG61, SFW05240, HT382	
		R_3		(65)
		R_2/R_{10}		(67)
		R_{11}		(71)
		R_{13a}		(77)
		R_{13b}		(78)
		R_{16}		(79)
		P_{u6}		(81)
		R_{adv}		(82)
		R_{17}		
		R_{18}		
14.	LG 14	R_2	HT567, SFW00211, SFW01272	(65)
			Head Rot	
15.	LG 1	*Qhr-1.1*	C1_4397272, C1_4397312, C1_4777966	(45)
		Qhr-1.2	C1_52669431, C1_52709188, C1_53853108, C1_58607030	
		Qhr-1.3	C1_77649943, C1_76872191, C1_77560891, C1_85024024	
16.	LG 2	*Qhr-2.1*	C2_13204329, C2_23136760	
17.	LG 10	*Qhr-10.1*	C10_134531054, C10_138804279	
18.	LG 12	*Qhr-12.1*	C12_25882328, C12_25210925, C12_27633722, C12_29909279	
19.	LG 13	*Qhr-13.1*	C13_163298005, C13_164438892, C13_164412093	
20.	LG 14	*Qhr-14.1*	C14_28826540, C14_28826567	
		Qhr-14.2	C14_131942451, C14_131764128	
21.	LG 16	*Qhr-16.1*	C16_21796525, C16_23076152, C16_23084558	
		Qhr-16.2	C16_200693781, C16_201545537	
22.	LG 17	*Qhr-17.1*	C17_3382790, C17_4443495	
		Qhr-17.2	C17_17987938, C17_18206805	
		Qhr-17.3	C17_128483584, C17_136177433	
		Qhr-17.4	C17_147584722, C17_149360561	
		Qhr-17.5	C17_169747479, C17_191024502	

mapping populations, this map which is currently used as the standard genetic map for sunflower (52) was further saturated with more SSR markers (51).

More than 2000 SSR markers are currently available for mapping and genotyping after being derived from genomic sequences (gSSR) and EST (EST-SSR) (53-56). Ultimately, SNP-based markers enabled the transition to high-density maps. Next-generation sequencing technology enabled whole genome sequencing and genotype-by-sequencing, which enabled genomic selection and GWAS in sunflower and the fabrication of vast quantities of SNP markers for high density maps and SNP arrays. This SNP-based map was used to fine-map the rust resistance gene *R12*. To further address and orient the linkage groupings in accordance with the sunflower reference genetic map, 118 SSR markers were added to the SNP map (37).

Forty-two SSR and/or SNP markers that were found using a 384 Illumina SNP-oligo pool array were used to compare the two marker types' estimated and observed heterozygosity as well as clustering using STRUCTURE and Discriminant Analysis of Principle Components (DAPC). The maintainer/restorer characteristic dominated the population structure, as in previous investigations (57, 58).

The newly created high-density maps in sunflower have a high resolution, which makes it easier to identify and clone genes for a variety of pertinent features in the future for the regions of interest. Furthermore, SNP-based maps provide markers that are closely associated with, for example, resistance genes. These markers can be used in extensive marker-assisted breeding initiatives or included into SNP arrays.

Genomic selection

For association mapping two approaches have been explored:

(1) Genome-wide association studies (GWAS) and

(2) Candidate gene approaches.

Genomic selection (GS) selects the individuals based on genomic breeding values (GEBVs) (38). Establishing a genotyped and phenotyped training population is the initial stage in GS. Three key distinctions between GS and regular MAS are readily apparent:

(1)MAS identifies markers associated with a gene of interest and quantitative traits during the training phase, while GS models are created to predict GEBVs,

(2)Traditional MAS uses only a small number of markers for genotyping during the breeding phase, while GS gathers genome-wide data and

(3)Traditional MAS uses only the identified markers to select individuals by genotype during the breeding phase, while GS selection is based on the GEBV (59).

If nothing is known about one or both parental lines, GS can predict the unknown parental GCA by using the model trained using both phenotyped and genotyped hybrids and can significantly increase breeding efficiency when compared to traditional GCA modelling (60).

The introduction of monogenic characteristics, particularly disease and herbicide resistance, into the breeding material has been accomplished with success using traditional MAS. The limitations of using identified molecular markers in different genetic backgrounds have also been demonstrated by validation of these markers across genotypes. Regarding disease responses of susceptible and resistant genotypes to pathogens such as *Plasmopara halstedii* (61), *S. sclerotiorum* (62) and *Verticillium dahlia* (63), transcriptional profiling has been carried out.

RNASeq or microarray studies have been used to examine the response of sunflower genotypes to drought using transcriptome analysis in water-limited environments compared to well-watered plants (42).

Sunflower has been mutagenized in order to enhance the genetic heterogeneity that is naturally present (64). Recently a TILLING (Targeted Induced Local Lesion In Genomes) population was created for high throughput screening of EMS (ethyl methane sulfonate)-induced mutations in sunflower, which is utilized for research on genes related in the synthesis of fatty acids (43).

A solid foundation for the localization and mapping of just inherited traits was established by developed linkage maps. Due to their dominant inheritance, the majority of the downy mildew resistance genes that confer resistance to the oomycete *Plasmopara halstedii* are reasonably easy to map using molecular markers. Finding markers that are closely related serves as a solid foundation for map- based gene cloning.

Table 3. Wild species possessing resistance against major diseases of sunflower

Sl. No	Source of resistance	Reference
	Downy Mildew	
1.	*H. annuus*, *H. petiolaris*, *H. tuberosus* and *H. praecox ssp. runyonii*, *H. argophyllus*, *H. pumilus*, *H. salicifolius*	(46, 83-86)
	Powdery Mildew	
2.	*H. agrestis*, *H. angustifolius*, *H. annuus*, *H. argophyllus*, *H. atrorubens*, *H. bolanderi*, *H. californicus*, *H. ciliaris*, *H. debilis ssp. debilis*, *H. debilis ssp. vestitus*, *H. decapetalus*, *H. divaricatus*, *H. eggertii*, *H. giganteus*, *H. glaucophyllus*, *H. hirsutus*, *H. laciniatus*, *H. laetiflorus*, *H. laevigatus*, *H. microcephalus*, *H. multiflorus*, *H. pauciflorus*, *H. praecox subsp. praecox*, *H. resinosus*, *H. rigidus*, *H. salicifolius*, *H. silphoides*, *H. simulans*, *H. smithii*, *H. strumosus*, *H. tuberosus*	(16, 47, 87-91)
	Rust	
3.	*H. annuus*, *H. argophyllus*, *H. grosseserratus*, *H. maximiliani*, *H. nuttallii*, *H. pauciflorus*, *H. petiolaris*, *H. tuberosus*	(16, 67, 92)
	Head Rot	
4.	*Helianthus angustifolius*, *H. argophyllus*, *H. californicus*, *H. debilis*, *H. decapetalus*, *H. divaricatus*, *H. giganteus*, *H. grosseserratus*, *H. maximiliani*, *H. neglectus*, *H. niveus*, *H. nuttallii*, *H. petiolaris*, *H. praecox ssp. Runyonii*, *H. resinosus*, *H. salicifolius*, *H. strumosus*, *H. tuberosus*	(93-98)
	Leaf Spot	
5.	*H. debilis*, *H. decapetalus*, *H. divaricatus*, *H. grossesseratus*, *H. hirsutus*, *H. mollis*, *H. maximiliani*, *H. occidentalis*, *H. pauciflorus*, *H. resinosus*, *H. simulans*, *H. strumosus*, *H. tuberosus*	(99)

The new high-throughput technologies combined with new genomic-based breeding strategies give us the opportunity, as never before, to understand and mine genetic variation and to use it for improvement of sunflower cultivars.

Conclusion

Genetic studies on major disease resistance in sunflower advances our understanding in the areas of complex interactions between host genome and pathogen. Conventional breeding has been crucial to the development of new sunflower varieties and hybrids over decades. However, the selection process and the difficulty in choosing appropriate genotypes owing to the quantitative nature of traits pose significant challenges like lengthy breeding procedures, mis-prediction, mis-selection, unfavourable environment etc. This has led breeders to increasingly adopt molecular technology (molecular breeding) to enhance precision and efficiency. In molecular breeding, the development of QTL mapping and association mapping facilitate the development of molecular markers that can be utilised in marker assisted selection in resistance breeding programme. Further development was achieved with the help of omics. This enables the successful introgression of resistant genes from wild species into high yielding cultivars. Although, advanced research in identification and validation of resistant QTLs by combining biotechnological tools and breeding approaches on resistant germplasm pools can be carried out to accelerate the development of broad-spectrum disease resistant and high yielding cultivars in sunflower.

Acknowledgements

The authors thank the DST-SERB and ICAR-DAC projects on sunflower being operated at the Department of Oilseeds, TNAU, Coimbatore for providing financial assistance and fellowship for conducting the experimental/research trials on sunflower.

Authors' contributions

VP involved in concept and writing of the review article; RS helped in concept and idea; RK arranged resources; LR and ES has contributed in editing the manuscript. All the authors were read and approved the final manuscript.

Compliance with ethical standards

Conflict of interest: The authors declare no conflict of interest.

Ethical issues: None

References

1. Nabipour A, Darvishzadeh R, Sarrafi AJCB. Conventional and molecular breeding of sunflower (*Helianthus annuus* L.). Crop Biotechnology. 2021;10(33):35-78. https://doi.org/10.30473/cb.2021.58978.1838
2. Darvishzadeh R, Pirzad A, Hatami-Maleki H, Kiani SP, Sarrafi A. Evaluation of the reaction of sunflower inbred lines and their F1 hybrids to drought conditions using various stress tolerance indices. Spanish Journal of Agricultural Research. 2010;8(4):1037-46. https://doi.org/10.5424/sjar/2010084-1398
3. Kolkman JM, Berry ST, Leon AJ, Slabaugh MB, Tang S, Gao W, et al. Single nucleotide polymorphisms and linkage disequilibrium in sunflower. Genetics. 2007;177(1):457-68. https://doi.org/10.1534/genetics.107.074054
4. Parkash V, Gaur A, Chauhan AJV. Sunflower: head rot, rust and powdery mildew. In: Diseases of Nationally Important Field Crops. 2014;30(100):491-500.
5. Rauf S, Jamil N, Tariq SA, Khan M, Kausar M, Kaya YJJotSoF, et al. Progress in modification of sunflower oil to expand its industrial value. Journal of the Science of Food and Agriculture. 2017;97(7):1997-2006. https://doi.org/10.1002/jsfa.8214
6. Lawson WR, Goulter KC, Henry RJ, Kong GA, Kochman JKJMB. Marker-assisted selection for two rust resistance genes in sunflower. Molecular Breeding. 1998;4:227-34. https://doi.org/10.1023/A:1009667112088
7. Cvejić S, Jocić S, Dedić B, Radeka I, Imerovski I, Miladinović D, editors. Determination of resistance to broomrape in newly developed sunflower inbred lines. In: Third international symposium on broomrape (*Orobanche* spp) in sunflower. Cordoba, Spain; 2014.
8. Dimitrijević A, Imerovski I, Miladinović D, Cvejić S, Jocić S, Zeremski T, et al. Oleic acid variation and marker-assisted detection of Pervenets mutation in high-and low-oleic sunflower cross. Crop Breeding and Applied Biotechnology. 2017;17:235-41. https://doi.org/10.1590/1984-70332017v17n3a36
9. Rajeshwaran D, Narayana M, Palaniappan V, Ramasamy S, Lingan R, Muniyandi SJE. Detection and validation of novel QTL associated with powdery mildew (*Golovinomyces cichoracearum* (DC.) VP *Heluta*.) resistance in sunflower (*Helianthus annuus* L.). Euphytica. 2022;218(10):143. https://doi.org/10.1007/s10681-022-03098-6
10. Zimmer D. Some biotic and climatic factors influencing sporadic occurrence of sunflower downy mildew. Phytopathology. 1975;65:751-4. https://doi.org/10.1094/Phyto-65-751
11. Gascuel Q, Martinez Y, Boniface MC, Vear F, Pichon M, Godiard L. The sunflower downy mildew pathogen *Plasmopara halstedii*. Molecular Plant Pathology. 2015;16(2):109-22. https://doi.org/10.1111/mpp.12164
12. Singh B, Dev U, Agarwal PC, Khetarpal RK. Downy mildew of sunflower (*Plasmopara haistedii*), a disease introduced and established in India-status report in relation to quarantine. Indian Journal of Plant Genetic Resources. 2006;19(2):262-70.
13. Molinero-Ruiz M, Melero-Vara JM, Domínguez JJE. Inheritance of resistance to two races of sunflower downy mildew (*Plasmopara halstedii*) in two *Helianthus annuus* L. lines. Euphytica. 2003;131:47-51. https://doi.org/10.1023/A:1023063726185
14. Ioos R, Laugustin L, Rose S, Tourvieille J, Tourvieille de Labrouhe D. Development of a PCR test to detect the downy mildew causal agent *Plasmopara halstedii* in sunflower seeds. Plant Pathology. 2007;56(2):209-18. https://doi.org/10.1111/j.1365-3059.2006.01500.x
15. Zimmer D. Physiological specialization between races of *Plasmopara halstedii* in America and Europe. Phytopathology. 1974;64(11):1465-7. https://doi.org/10.1094/Phyto-64-1465
16. Carson M. New race of *Plasmopara halstedii* virulent on resistant sunflowers in South Dakota. Plant Diseases. 1981;65:842-3. https://doi.org/10.1094/PD-65-842
17. Gulya T, Raje N. A new race of sunflower downy mildew; 1985. https://www.cabidigitallibrary.org/doi/full/10.5555/19861656095
18. Ljubich A, Gulya T, Miller J. A new race of sunflower downy mildew in North America. Phytopathology. 1988;78:1508.

19. Gulya T, editor Proposal of a revised system of classifying races of sunflower downy mildew. In: Proceedings of the 17th Sunflower Research Workshop; 1995.
20. Spring O, Miltner F, Gulya T. New races of sunflower downy mildew (*Plasmopara halstedii*) in Germany. Journal of Phytopathology. 1994;142(4):241-4. https://doi.org/10.1111/j.1439-0434.1994.tb04535.x
21. Patil M, Mayee C. Race identity of Indian *Plasmopara halstedii*, cause of downy mildew of sunflower; 1991. https://www.cabidigitallibrary.org/doi/full/10.5555/19922314065
22. Gulya T, Rashid K, Maširević S. Sunflower diseases, Sunflower technology and production. American Society of Agronomy, Madison, Wisconsin, USA; 1997. p. 834. https://doi.org/10.2134/agronmonogr35.c6
23. Kallamadi PR, Mulpuri SJB. Inheritance and molecular mapping of powdery mildew (*Golovinomyces orontii*) resistance gene (s) in sunflower (*Helianthus annuus* L.). 3 Biotech. 2020;10(5):234. https://doi.org/10.1007/s13205-020-02224-2
24. Rao SC, Sujatha M, Karuna K, Varaprasad K. Powdery mildew disease in sunflower: A review. The Indian Society of Oilseeds Reseach. 2006;32:111. https://doi.org/10.56739/jor.v32i1.141781
25. Putt ED, Sackston W. Studies on sunflower rust: I. Some sources of rust resistance. Canadian Journal of Plant Science. 1957;37(1):43-54. https://doi.org/10.4141/cjps57-005
26. Bushland T, Antonelli E. Reactions of Argentine and Australian sunflower rust differentials to four North American cultures of *Puccinia helianthi* from North Dakota. Plant Disease. 1986;70:883-6. https://doi.org/10.1094/PD-70-883
27. Shirshikar SJH. Integrated management of sunflower necrosis disease/manejo integrado de la necrosis del girasol/controle integral de la necrose dutournesol. Integrated Management of Sunflower Necrosis Disease. 2008;31(49):27-34. https://doi.org/10.2298/HEL0849027S
28. Shirshikar S. Sunflower necrosis disease management with thiomethoxam. Helia. 2010;33(53):63-8. https://doi.org/10.2298/HEL1053063S
29. Prasada Rao R, Reddy A, Chander Rao S, Varaprasad K, Thirumala -Devi K, Nagaraju MV, et al. Tobacco streak ilarvirus as causal agent of sunflower necrosis disease in India. Journal of Oilseeds Research. 2000;17(2):400-1.
30. Harvir Singh HS. Thrips incidence and necrosis disease in sunflower, *Helianthus annuus* L. Journal of Oilseeds Research. 2005;22(1):90-2.
31. Bhat A, Jain R, Kumar A, Ramiah M, Varma AJAov. Serological and coat protein sequence studies suggest that necrosis disease on sunflower in India is caused by a strain of Tobacco streak ilarvirus. Archives of Virology. 2002;147:651-8. https://doi.org/10.1007/s007050200015
32. Škorić D. Sunflower breeding for resistance to abiotic and biotic stresses. In: Abiotic and biotic stress in plants-recent advances and future perspectives: IntechOpen; 2016. https://doi.org/10.5772/62159
33. Livaja M, Unterseer S, Erath W, Lehermeier C, Wieseke R, Plieske J, et al. Diversity analysis and genomic prediction of *Sclerotinia* resistance in sunflower using a new 25 K SNP genotyping array. Theoretical and Applied Genetics. 2016;129:317-29. https://doi.org/10.1007/s00122-015-2629-3
34. Qi L, Long Y, Talukder ZI, Seiler GJ, Block CC, Gulya TJ. Genotyping-by-sequencing uncovers the introgression alien segments associated with sclerotinia basal stalk rot resistance from wild species-I. *Helianthus argophyllus* and *H. petiolaris*. Frontiers in Genetics. 2016;7:219. https://doi.org/10.3389/fgene.2016.00219
35. Talukder ZI, Seiler GJ, Song Q, Ma G, Qi L. SNP discovery and QTL mapping of *Sclerotinia* basal stalk rot resistance in sunflower using genotyping‐by‐sequencing. The Plant Genome. 2016;9(3):plantgenome2016.03.0035. https://doi.org/10.3835/plantgenome2016.03.0035
36. Cavanagh C, Morell M, Mackay I, Powell W. From mutations to MAGIC: resources for gene discovery, validation and delivery in crop plants. Current Opinion in Plant Biology. 2008;11(2):215-21. https://doi.org/10.1016/j.pbi.2008.01.002
37. Dimitrijevic A, Horn R. Sunflower hybrid breeding: from markers to genomic selection. Frontiers in Plant Science. 2018;8:2238. https://doi.org/10.3389/fpls.2017.02238
38. Meuwissen TH, Hayes BJ, Goddard M. Prediction of total genetic value using genome-wide dense marker maps. Genetics. 2001;157(4):1819-29. https://doi.org/10.1093/genetics/157.4.1819
39. Körösi K, Lázár N, Virányi F. Resistance to downy mildew in sunflower induced by chemical activators. Acta Phytopathologica et Entomologica Hungarica. 2009;44(1):1-9. https://doi.org/10.1556/APhyt.44.2009.1.1
40. Kallamadi PR, Yadav P, Dandu K, Soni PK, Sankaraneni CR, Bharadwaja KP, et al. Host defense responses during powdery mildew (*Golovinomyces latisporus* comb. nov.) infection in sunflower (Helianthus annuus L.). Tropical Plant Pathology. 2022;47(4):495-508. https://doi.org/10.1007/s40858-022-00501-4
41. Liu J, Zhang Y, Meng Q, Shi F, Ma L, Li Y. Physiological and biochemical responses in sunflower leaves infected by *Sclerotinia sclerotiorum*. Physiological and Molecular Plant Pathology. 2017;100:41-8. https://doi.org/10.1016/j.pmpp.2017.06.001
42. Liang C, Wang W, Wang J, Ma J, Li C, Zhou F, et al. Identification of differentially expressed genes in sunflower (*Helianthus annuus*) leaves and roots under drought stress by RNA sequencing. Botanical Studies. 2017;58:1-11. https://doi.org/10.1186/s40529-017-0197-3
43. Sabetta W, Alba V, Blanco A, Montemurro C. sunTILL: a TILLING resource for gene function analysis in sunflower. Plant Methods. 2011;7:1-13. https://doi.org/10.1186/1746-4811-7-20
44. Qi L, Gulya T, Seiler GJ, Hulke BS, Vick BA. Identification of resistance to new virulent races of rust in sunflowers and validation of DNA markers in the gene pool. Phytopathology. 2011;101(2):241-9. https://doi.org/10.1094/PHYTO-06-10-0162
45. Talukder ZI, Underwood W, Misar CG, Seiler GJ, Cai X, Li X, et al. A quantitative genetic study of sclerotinia head rot resistance introgressed from the wild perennial *Helianthus maximiliani* into cultivated sunflower (*Helianthus annuus* L.). International Journal of Molecular Sciences. 2022;23(14):7727. https://doi.org/10.3390/ijms23147727
46. Seiler G. Registration of 13 downy mildew tolerant interspecific sunflower germplasm lines derived from wild annual species. Crop Science. 1991;31(6):1714-6. https://doi.org/10.2135/cropsci1991.0011183X003100060093x
47. Rojas-Barros P, Jan C-C, Gulya TJ, editors. Transferring powdery mildew resistance genes from wild *Helianthus* into cultivated sunflower. In: Proceedings of the 27th Sunflower Research Workshop Fargo National Sunflower Association; 2005.
48. Rieseberg LH, Choi H, Chan R, Spore C. Genomic map of a diploid hybrid species. Heredity. 1993;70(3):285-93. https://doi.org/10.1038/hdy.1993.41
49. Jan C, Vick B, Miller J, Kahler A, Butler III E. Construction of an RFLP linkage map for cultivated sunflower. Theoretical and Applied Genetics. 1998;96:15-22. https://doi.org/10.1007/s001220050703
50. Gedil MA, Wye C, Berry S, Segers B, Peleman J, Jones R, et al. An integrated restriction fragment length polymorphism-amplified fragment length polymorphism linkage map for cultivated sunflower. Genome. 2001;44(2):213-21. https://doi.org/10.1139/g00-111

51. Yu JK, Tang S, Slabaugh MB, Heesacker A, Cole G, Herring M, et al. Towards a saturated molecular genetic linkage map for cultivated sunflower. Crop Science. 2003;43(1):367-87. https://doi.org/10.2135/cropsci2003.3670

52. Tang S, Kishore VK, Knapp SJ. PCR-multiplexes for a genome-wide framework of simple sequence repeat marker loci in cultivated sunflower. Theoretical and Applied Genetics. 2003;107:6-19. https://doi.org/10.1007/s00122-003-1233-0

53. Brunel D. A microsatellite marker in *Helianthus annuus* L. Plant Molecular Biology. 1994;24:397-400. https://doi.org/10.1007/BF00020177

54. Dehmer K, Friedt W. Development of molecular markers for high oleic acid content in sunflower (*Helianthus annuus* L.). Industrial Crops and Products. 1998;7(2-3):311-5. https://doi.org/10.1016/S0926-6690(97)00063-0

55. Paniego N, Echaide M, Muñoz M, Fernández L, Torales S, Faccio P, et al. Microsatellite isolation and characterization in sunflower (*Helianthus annuus* L.). Genome. 2002;45(1):34-43.https://doi.org/10.1139/g01-120

56. Kiani SP, Talia P, Maury P, Grieu P, Heinz R, Perrault A, et al. Genetic analysis of plant water status and osmotic adjustment in recombinant inbred lines of sunflower under two water treatments. Plant Science. 2007;172(4):773-87. https://doi.org/10.1016/j.plantsci.2006.12.007

57. Mandel JR, Nambeesan S, Bowers JE, Marek LF, Ebert D, Rieseberg LH, et al. Association mapping and the genomic consequences of selection in sunflower. PLoS Genetics. 2013;9(3):e1003378. https://doi.org/10.1371/journal.pgen.10033

58. Filippi CV, Aguirre N, Rivas JG, Zubrzycki J, Puebla A, Cordes D, et al. Population structure and genetic diversity characterization of a sunflower association mapping population using SSR and SNP markers. BMC Plant Biology. 2015;15:1-12. https://doi.org/10.1186/s12870-014-0360-x

59. Nakaya A, Isobe SN. Will genomic selection be a practical method for plant breeding? Annals of Botany. 2012;110(6):1303-16. https://doi.org/10.1093/aob/mcs109

60. Mangin B, Bonnafous F, Blanchet N, Boniface M-C, Bret-Mestries E, Carrère S, et al. Genomic prediction of sunflower hybrids oil content. Frontiers in Plant Science. 2017;8:1633. https://doi.org/10.3389/fpls.2017.01633

61. Livaja M, Wang Y, Wieckhorst S, Haseneyer G, Seidel M, Hahn V, et al. BSTA: a targeted approach combines bulked segregant analysis with next-generation sequencing and de novo transcriptome assembly for SNP discovery in sunflower. BMC Genomics. 2013;14:1-10. https://doi.org/10.1186/1471-2164-14-628

62. Muellenborn C, Krause J-H, Cerboncini C. Analysis of differential transcript expression reveals time-dependent leaf responses to *Sclerotinia sclerotiorum* in wild and cultivated sunflower. Plant Molecular Biology Reporter. 2011;29:597-608. https://doi.org/10.1007/s11105-010-0265-2

63. Guo S, Zuo Y, Zhang Y, Wu C, Su W, Jin W, et al. Large-scale transcriptome comparison of sunflower genes responsive to *Verticillium dahliae*. BMC Genomics. 2017;18:1-13. https://doi.org/10.1186/s12864-016-3386-7

64. Zambelli A, León A, Garcés R. Mutagenesis in sunflower. In: Sunflower. Elsevier; 2015. p. 27-52. https://doi.org/10.1016/B978-1-893997-94-3.50008-8

65. Qi L, Long Y, Jan C, Ma G, Gulya T. *Pl* 17 is a novel gene independent of known downy mildew resistance genes in the cultivated sunflower (*Helianthus annuus* L.). Theoretical and Applied Genetics. 2015;128:757-67. https://doi.org/10.1007/s00122-015-2470-8

66. Ramazanova SA, Badyanov EV, Guchetl SZ. Validation of microsatellite markers to identify *Pl*6, *Pl*8 and *Plarg* genes that control resistance to *Plasmopara halstedii* in sunflower. Caspian Journal of Environmental Sciences. 2021;19(5):915-20. https://doi.org/10.22124/CJES.2021.5266

67. Qi L, Seiler G, Vick B, Gulya T. Genetics and mapping of the *R 11* gene conferring resistance to recently emerged rust races, tightly linked to male fertility restoration, in sunflower (*Helianthus annuus* L.). Theoretical and Applied Genetics. 2012;125:921-32.https://doi.org/10.1007/s00122-012-1883-x

68. Talukder ZI, Gong L, Hulke BS, Pegadaraju V, Song Q, Schultz Q, et al. A high-density SNP map of sunflower derived from RAD-sequencing facilitating fine-mapping of the rust resistance gene *R12*. PLoS One. 2014;9(7):e98628. https://doi.org/10.1371/journal.pone.0098628

69. Ma G, Song Q, Markell S, Qi L. High-throughput genotyping-by-sequencing facilitates molecular tagging of a novel rust resistance gene, R 15, in sunflower (*Helianthus annuus* L.). Theoretical and Applied Genetics. 2018;131:1423-32. https://doi.org/10.1007/s00122-018-3087-5

70. Qi L, Foley M, Cai X, Gulya T. Genetics and mapping of a novel downy mildew resistance gene, *Pl* 18, introgressed from wild *Helianthus argophyllus* into cultivated sunflower (*Helianthus annuus* L.). Theoretical and Applied Genetics. 2016;129:741-52.https://doi.org/10.1007/s00122-015-2662-2

71. Liu Z, Zhang L, Ma G, Seiler G, Jan C, Qi L. Molecular mapping of the downy mildew and rust resistance genes in a sunflower germplasm line TX16R. Molecular Breeding. 2019;39:1-11. https://doi.org/10.1007/s11032-018-0921-z

72. Boulos C, Salameh P, Barberger-Gateau P. The AMEL study, a cross sectional population-based survey on aging and malnutrition in 1200 elderly Lebanese living in rural settings: protocol and sample characteristics. BMC Public Health. 2013;13:1-13. https://doi.org/10.1186/1471-2458-13-573

73. Qi L, Ma G, Seiler G. Registration of two confection sunflower germplasms, HA-DM5 and HA-DM6, resistant to sunflower downy mildew. Journal of Plant Registrations. 2020;14(1):87-91. https://doi.org/10.1002/plr2.20014

74. Ma G, Markell S, Song Q, Qi L. Genotyping-by-sequencing targeting of a novel downy mildew resistance gene *Pl* 20 from wild *Helianthus argophyllus* for sunflower (*Helianthus annuus* L.). Theoretical and Applied Genetics. 2017;130:1519-29. https://doi.org/10.1007/s00122-017-2906-4

75. Qi L, Cai X. Characterization and mapping of a downy mildew resistance gene, *Pl* 36, in sunflower (*Helianthus annuus* L.). Molecular Breeding. 2022;42(2):8.https://doi.org/10.1007/s11032-022-01280-1

76. Ma G, Song Q, Li X, Qi L. High-density mapping and candidate gene analysis of *Pl* 18 and *Pl* 20 in sunflower by whole-genome resequencing. International Journal of Molecular Sciences. 2020;21(24):9571. https://doi.org/10.3390/ijms21249571

77. Mohase L. Biochemical events associated with rust resistance in sunflower. University of the Free State; 2003.

78. Lambrides C, Miller J. Inheritance of rust resistance in a source of MC29 sunflower germplasm. Crop Science. 1994;34(5):1225-30. https://doi.org/10.2135/cropsci1994.0011183X003400050015x

79. Gong L, Hulke B, Gulya T, Markell S, Qi L. Molecular tagging of a novel rust resistance gene *R 12* in sunflower (*Helianthus annuus* L.). Theoretical and Applied Genetics. 2013;126:93-9. https://doi.org/10.1007/s00122-012-1962-z

80. Zhang M, Liu Z, Jan C-C. Molecular mapping of a rust resistance gene *R 14* in cultivated sunflower line PH 3. Molecular Breeding. 2016;36:1-12. https://doi.org/10.1007/s11032-016-0456-0

81. Bulos M, Vergani PN, Altieri E. Genetic mapping, marker assisted selection and allelic relationships for the Pu6 gene conferring rust resistance in sunflower. Breeding Science. 2014;64(3):206-12. https://doi.org/10.1270/jsbbs.64.206

82. Qi L, Talukder Z, Ma G, Li X. Discovery and mapping of two new rust resistance genes, R 17 and R 18, in sunflower using genotyping by sequencing. Theoretical and Applied Genetics. 2021;134:2291-301. https://doi.org/10.1007/s00122-021-03826-x

83. Tan AS, Jan CC, Gulya TJ. Inheritance of resistance to race 4 of sunflower downy mildew in wild sunflower accessions. Crop Science. 1992;32(4):949-52. https://doi.org/10.2135/cropsci1992.0011183X003200040022x

84. Dussle CM, Hahn V, Knapp SJ, Bauer E. *Pl Arg* from *Helianthus argophyllus* is unlinked to other known downy mildew resistance genes in sunflower. Theoretical and Applied Genetics. 2004;109:1083-6. https://doi.org/10.1007/s00122-004-1722-9

85. Nikolova L, Shindrova P, Entcheva V. Resistance to diseases, obtained through interspecific hybridization/Resistance a las enfermedades obtenida por la hibridizacion interspecies/Resistance a la malade obtenue par hybridation interspecies. Helia. 2000;23(33):57-64. https://doi.org/10.1515/helia.2000.23.33.57

86. Encheva J, Köhler H, Friedt W. RAPD analysis of lines derived from interspecific cross *Helianthus annuus* L.× *Helianthus salicifolius*. Helia. 2012;35(57):9-18. https://doi.org/10.2298/HEL1257009E

87. Saliman M, Yang S, Wilson L. Reaction of *Helianthus* species to Erysiphe cichoracearum. Plant Disease. 1982;66:572-3. https://doi.org/10.1094/PD-66-572

88. Skoric D. Sunflower breeding. Uljarstvo (Yugoslavia). 1988;25(1).

89. McCarter S. Reactions of Jerusalem artichoke genotypes to two rusts and powdery mildew. Plant Disease. 1993;77:242-5. https://doi.org/10.1094/PD-77-0242

90. Dauguet S, Labalette F, Lecomte V, Leflon M, Mestries E. Account of the 17th International Sunflower Conference at Cordoba (Spain), 8-12 June 2008.

91. Dedić B, Terzić S, Atlagić J, Miladinović D, Mrđa J, Tančić S, et al., editors. Screening perennial *Helianthus* species for powdery mildew. In: Proceedings, 18th International Sunflower Conference, Mar del Plata & Balcare, Argentina, 27 February-1 March 2012. Paris: International Sunflower Association; 2012.

92. Seiler G, Marek LF. Germplasm resources for increasing the genetic diversity of global cultivated sunflower. Helia. 2011;34(55):1-20. https://doi.org/10.2298/HEL1155001S

93. Hahn V. Genetic variation for resistance to Sclerotinia head rot in sunflower inbred lines. Field Crops Research. 2002;77(2-3):153-9. https://doi.org/10.1016/S0378-4290(02)00082-5

94. Block C, Gulya T, Marek L. Evaluation of wild sunflower species for resistance to Sclerotinia stalk rot. Phytopathology. 2009;99:S13.

95. Tikhomirov VT, Chiryaev PV. Sources of resistance to diseases in original material of sunflower/Fuentes de resistance a las enfermedades en el material original de girasol/Les sources de resistance aux maladies dans le materie 'origine du tournesol. Helia. 2005;28(42):101-6. https://doi.org/10.2298/HEL0542101T

96. Seiler GJ, Gulya TJ, editors. Exploration for wild *Helianthus* species in North America: challenges and opportunities in the search for global treasures. International Sunflower Conference Proceedings; 2004.

97. Henn H, Steiner U, Wingender R, Schnabl H. Wildtype sunflower clones: source for resistance against *Sclerotinia sclerotiorum* (Lib.) de Bary stem infection; 1997.

98. Block C, Marek L, Gulya T. Identifying resistance to Sclerotinia stalk and root rot in perennial sunflower germplasm. Phytopathology. 2012;102:S4.

99. Sujatha M, Prabakaran A, Chattopadhyay C. Reaction of wild sunflowers and certain interspecific hybrids to *Alternaria helianthin*; 1997.

Additional information

Peer review: Publisher thanks Sectional Editor and the other anonymous reviewers for their contribution to the peer review of this work.

Reprints & permissions information is available at https://horizonepublishing.com/journals/index.php/PST/open_access_policy

Publisher's Note: Horizon e-Publishing Group remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Indexing: Plant Science Today, published by Horizon e-Publishing Group, is covered by Scopus, Web of Science, BIOSIS Previews, Clarivate Analytics, NAAS, UGC Care, etc
See https://horizonepublishing.com/journals/index.php/PST/indexing_abstracting

Publisher information: Plant Science Today is published by HORIZON e-Publishing Group with support from Empirion Publishers Private Limited, Thiruvananthapuram, India.