Chapter 8
Genomic Designing for Biotic Stress Resistance in Carrot (*Daucus carota* L.)

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Abstract Carrot productivity may be impacted by an array of insect pests and diseases. Carrots are affected by at least 36 fungal and oomycete pathogens, five bacterial pathogens, 13 viruses, two phytoplasmas and, in addition to seven nematode species and two parasitic plant taxa. Additionally, a number of insect pest and mite infestations may result in loss. There have been significant efforts to identify wild species that are resistant to certain biotic stresses for introduce into breeding populations and viable varieties, as well as to choose carrot varieties that are partially or completely resistant to a variety of these diseases and insect pests. Significant advances have been made in identifying resistance to a range of diseases and insect pests, as well as mapping that resistance to the carrot nuclear and mitochondrial genome. However, progress in understanding the inheritance of resistance and building extremely efficient resistance to the majority of these many stresses has been slow. Due to the myriad of stresses and relations among insect pests and diseases, it may be challenging to develop hybrids or varieties that are resistant to all of the carrot growing region's key biotic stresses while still fulfilling market and consumer expectations. Novel strategies for detecting resistant varieties and speeding up conventional breeding are being developed using molecular breeding tools like as marker development and deep-coverage carrot genome libraries. These critical genetic techniques will aid researchers in identifying and developing disease, insect, and virus-resistant carrot varieties.

Keywords Carrot · Fungi · Resistance · Insect · Pest · Virus

8.1 Introduction

Diseases and insect pest or mite infestations reduce carrot production considerably in the majority of carrot-growing regions around the world (Rubatzky et al. 1999). Powdery mildew Cercospora leaf spot, Alternaria leaf blight, and bacterial blight are

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the most common foliar diseases (Davis and Raid 2002). Carrot cavity spot, white mould and root-knot nematodes are the most common soil-borne root diseases (Davis and Raid 2002).

Pests such as the carrot willow aphids, carrot rust fly, and the two-spotted spider mite pose a risk to carrot growers (Simon et al. 2008). Fusarium dry rot, Violet root rots and bacterial soft rots are other carrot diseases that cause regional losses (Davis and Raid 2002). Breeders have emphasized on primary infection in places where the disease or insect pest is quite well established in order to discover the most appropriate for genetic resistance to the majority of these biotic stresses. Biotic stress is exacerbated by interseeding the extremely sensitive cultivars or breeding lines with carrot entrants. Plants are injected with diseases or infected with insect pests in some of these insect pest and disease screening and breeding approaches. Due to the difficulties involved in establishing relatively homogeneous soil-borne disease stresses, testing for resistance to diseases and soil-borne insect pests can be difficult, even more so when screening a large number of characters and for stresses derived from multiple pathogen species or races or a pathogen-host interaction. This chapter discusses attempts to create resistance to certain carrot insect pests and diseases, as well as phenotypic screening approaches and current understanding of the genetic component of susceptibility, including inheritance and resistance gene annotation on the carrot nuclear and mitrochrondrial genome. Regrettably, the majority of biotic stresses affecting carrots are unknown, as is the recognised genetic foundation of resistance. As previously stated in this chapter, there are significant gaps in our knowledge of carrot germplasm response to a number of biotic stresses, emphasising the need for more study. This chapter is not intended to be a thorough review of all current information on carrot illnesses and insect pest resistance. The chapter gives insight into the genetic basis and genetics of tolerance for a few of the most prevalent diseases and caused by pests for which scientists have endeavored to screening for resistance. Several illnesses and insect pests that were previously mentioned in this chapter have been updated. The need of using current scientific language was emphasised. Several insect pests and diseases have acquired nicknames in recent years. This chapter differentiates between foliar pathogen and soil pathogen-caused carrot diseases, and it finishes with a discussion of insect pests and nematode.

8.2 Foliar Diseases

8.2.1 Powdery Mildew

Carrots are sensitive to *Leveillula lanuginosa* and *L. taurica* caused by *oidiopsis* and *oidiodium*, as well as *Erysiphe heraclei* caused *oidiodium* (syn. *E. polygoni* and *E. umbelliferarum*) (Aegerter 2002). *Erysiphe heraclei* is found all throughout the globe, although it is most common in warm, semi-arid climates. Powdery mildew

severity is weather dependent, crop development stage dependent, production technique dependant, and cultivar dependent (du Toit and Derie 2008; Aegerter 2002; Abercrombie and Finch 1976; Palti 1975). Powdery mildew is especially damaging to delicate cultivars and parent lines cultivated in hot, semi-arid environments with drip or furrow irrigation. Infections on the leaves may prevent mechanical harvesters from extracting roots from the ground. Disease has the potential to be lethal in greenhouses (Geary and Wall 1976). Carrots in the India, Kazakhstan, Armenia, Middle East, and other Central Asian republics, as well as the Mediterranean areas of Europe and Africa, are infested with *Leveillula* spp (Palti 1975). It is established that *Toxoplasma heraclei* produces haustoria during ectotypic development on carrot, which invade the carrot epidermal cells, resulting in infection. The fungus produces milky white mycelial growth on the petioles, leaves, bracts, umbels, flower stalks and roots of the plants (Aegerter 2002). Foliage that has been significantly affected may develop chlorotic and die prematurely. *L. lanuginosa* and *L. taurica*, on the other hand, produce the endophytic and ectotopic mycelium. During the development of conidiophores that emerge from stomata, *L. lanuginosa* and *L. taurica* generate conidia at the tips of their long conidiophores. Powdery mildew (*Leveillula spp.*) is a fungus that causes light yellow lesions on the surface of the leaves as well as white sporulation (Aegerter 2002). Infections that are contained within leaf veins cause angular lesions. Spores may form on the upper surface of the leaf, and chlorotic regions may necrotize. *Erysiphe heraclei* produces a white fungal bloom that is less noticeable. Powdery mildew fungus conidia are distributed by the air (Aegerter 2002). Unlike other fungal plant diseases, the spores survive and infect plants under conditions of high humidity and moderate temperature. Because sunlight destroys conidia and mycelium, powdery mildews flourish in shaded locations. Powdery mildew occurs on older leaves and spreads to younger plants as a result of increasing humidity and shadow levels in the canopy. Powdery mildew causes havoc on mature carrot plants (Aegerter 2002). Symptoms appear 7–14 days after infection, and sporulation occurs 7–14 days later. The disease may be more severe due to the dense cover of carrot seed fields (du Toit et al. 2009). While powdery mildew growth does not seem to infect carrot seeds, it is possible that cleistothecia (sexual fruiting structures) are affected. At least 86 distinct species of plants belonging to the genus Apiaceae have been found to be infected by the *Erysiphe heraclei* (Hammarlund 1925; Marras 1962; Braun 1987; Aegerter 2002; Glawe et al. 2005; Cunnington et al. 2008). One host species' inoculum may be incompatible with another host species' inoculum. While certain isolates may infect a broad range of plant species and genera, Apiaceae genera and species vary in their virulence (Koike and Saenz 1994, 1997; Cunnington et al. 2008). Similarly, *L. lanuginosa* has been shown to infect a wide variety of Apiaceae genera and species, with isolate specificity varying greatly (Cirulli 1975).

As a consequence, *Leveillula taurica* has a far wider host range and is much more host specific than *Leveillula taurica* (Palti 1975; Braun 1987; Aegerter 2002).

Four *Daucus* subspecies have been chosen for resistant breeding (Umiel et al. 1975; Bonnet 1977). Bonnet (1983a, b) revealed a single powdery mildew resistance gene in *D. c.* subsp. *dentatus* that conferred resistance to powdery mildew. Backcrossing with the susceptible 'Touchon' revealed that resistance is governed by a

monogenic dominant, *Eh*, which was identified during the research process. Orange roots were used to identify resistant lineages. Bonnet (1983a, b) proposed *Daucus siculus* and *Daucus carota* 'Bauers Kieler Rot' as powdery mildew resistant plants, while Lebeda and Coufal (1987) tested the resistance of 111 *D. c.* subsp. *sativus* cultivars to E. heraclei in the Czechoslovak wild. '*Gavrilovskaya*,' one cultivar, was entirely free of powdery mildew, whilst the other thirteen had considerable powdery mildew. Almost half of the 111 cultivars tested demonstrated "possible partial dominance and quantitative resistance to powdery mildew". The enzyme, as previously noted, is lytic against pathogenic fungus and bacteria. Resistance to Alternaria leaf blight was found in one of the transgenic 'Nantes Scarlet' plants (Table 8.1). Human lysozyme production increased in these lines in response to resistance. When Wally et al. (2009a) employed the *Arabidopsis thaliana* (*At*) *NPR1* gene to develop transgenic 'Nantes Coreless' carrot lines; they were the first to report on this technique (non-expressor of PR genes). A study of two transformants, *NPR1-1* and *NPR1-XI*, found that when treated with isolated *Sclerotiorum* cell membrane segments or 2, 6-dichloroisonicotinic acid, the *DcPR-1*, *DcPR-2*, and *DcPR-5* genes were expressed at higher levels than when exposed with a control. When these lines were infected with *E. heraclei*, they experienced a 90% decrease in powdery mildew relative to non-transgenic cultivar lines. *NPR1*, a master switch for systemic acquired resistance (SAR), has been shown to be overexpressed in plants, conferring resistance to powdery mildew, *X. hortorum* pv. *carotae* and necrotrophic diseases. In Czechoslovakia in 1987, Lebeda and Coufal employed spontaneous infections to test for resistant cultivars, but only one out of every three fields had adequate disease pressure. Powdery mildew pressure may be easily induced in the field or greenhouse by using highly susceptible variety as "*spreader*" plants under warm, dry conditions. In a greenhouse, inoculum may be maintained by regularly growing healthy plants alongside infected ones. Powdery mildew grows on close-up pictures of plants. du Toit et al. (2009) studied the impact of extremely high powdery mildew pressure on carrot seed rates.

8.2.2 Alternaria Leaf Blight

Alternaria leaf blight (*Alternaria dauci*) is the most common foliar disease in the majority of carrot-growing countries. *A. dauci* was found in Germany in 1855 and is a major carrot crop pest in areas with considerable precipitation and high temperatures (Farrar et al. 2004). Every day of the growth season, massive amounts of saprophytic spores are generated and disseminated aerially throughout a broad temperature and moisture range (8–28 °C) (Maude, 1966). According to Langenberg et al. (1977), little green–brown lesions appear 8–10 days following the infection. During the progression of the lesion, the sick tissue darkens to the point of being completely black, and a chlorotic haze is seen (Farrar et al. 2004). As well as infecting developing florets and seeds inside inflorescences, *A. dauci* may cause symptoms on the leaves

Table 8.1 Genetics of disease and pest in carrot

Disease/Pest	Scientific name	Resistance gene/QTL	Resistance variety	References
Alternaria leaf blight	*A. dauci*	Three QTL		Le Clerc et al. (2009)
	A. dauci	Elevan QTLs		Le Clerc et al. (2015a, b)
Cercospora leaf spot	*C. carota*	*Ce*	Wisconsin Inbred 1 (WCR-1)	Angell and Gabelman (1968)
Aster yellows	Mycoplasma like organism		Scarlet Nantes, Royal Chantenay, Gold King	Gableman et al. (1994)
Motley dwarf	Virus		CVC-14	Watson and Falk (1994)
			Autumn	Dunn (1970)
			Kurnella Strongtop, Western Red	Tomlison (1965)
Cavity spot	*Pythium* sp.		Redca, Nandor	Bonnet (1983a, b), Cofal (1987)
			Amsterdam Forcing, Nantes, Chantenay, Berlicum, Autumn King	Bonnet (1983a, b), Cofal (1987)
Powdery mildew	*Erysiphe heraceli*	*Eh*	*Daucus siculus, Bauers Kieler Rote, Gavriloskaya*	Bonnet (1983a, b), Cofal (1987)
			Daucus carota ssp. dentatus	Bonnet (1983a, b)
Lygus bug	*Lygus hesperus, Lygus elisus*		Imperida	Scott (1977)
Carrot fly	*Psila rosae*		Gelbe Rheinische St. Valery Clause's Sytan Original, Royal Chantenay Elite (Rota) No.275, Vertou LD, Long Chantenay,, and Danvers Half Long 126, Clause's Jaune Obtuse de Doubs	Ellis and Hardman (1981)
Root knot nematode	*M javanica* and *M. incognita*	*Mj-1*	Brasilia and Tropical	Ali et al. (2014), Simon et al. (2000)

(continued)

Table 8.1 (continued)

Disease/Pest	Scientific name	Resistance gene/QTL	Resistance variety	References
		Brasillia × B6274		Simon et al. (2000)
		1 or few		Yunhee et al. (2014)
			BRS Planalto	Pinheiro et al. (2011)
Root knot nematode	*M.incognita*	7 QTLs		Parsons et al. (2015)
			DR-333	Siddiqui et al. (2011)
		Mj-2	PI652188	
Root knot nematode	*M.e hapla*	*Mh-1, Mh-2*		Wang and Goldman (1996), Bridge and Starr (2007)
	M.chitwoodi, M. fallax		Berlanda, Bolero, Chantenay, Nantucket, Parmex	Wesemael and Moens (2008)
			Ingot	
			Daucus capillifolius	Ellis et al. (1991)
			Flyaway	Simon et al. (2013)
Aphids				
Carrot-willow aphid	*C. aegopodii*		Osborne Park, Autumn King	

of plants as well. Damping-off is an inoculum-induced disease that spreads via the seeds or seedlings of infected plants (Maude 1966; Farrar et al. 2004).

Certain carrot types have exhibited resistance to *A. dauci*. Despite the fact that only three cultivars are completely immune to Alternaria leaf blight, additional study is needed. A total of 90 carrot inbred lines and 241 PI lines from 31 regions were studied by Strandberg et al. (1972). After a natural infection emerged in Brazil less than a week afterwards, the variant designated 'Brasilia' was shown to be the most resistant. (Boiteux et al. 1993). Resistance stability data is useful to breeders since it displays the frequency with which a trait appears in a variety of situations. Rogers and Stevenson (2010) identified three commercial carrot cultivars that reacted differently to *A. dauci* isolates. When 11 A. dauci isolates from across the world were employed, Le Clerc et al. (2015a) discovered no significant interaction between isolates, inbred lines, and a segregating population. Certain data might be explained by genome polymorphisms, fungal isolates, or other environmental factors. Different kinds of resistance components, according to Le Clerc et al. (2015b), may impact resistance effectiveness in a variety of settings. While Rogers and Stevenson (2010) collected

samples eight and sixteen days after infection, Le Clerc et al. (2015b) collected samples twenty and thirty-five days later, with further samples collected every fifteen days. Due to the fact that various defence mechanisms are triggered at different stages after infection, disease development in carrot cultivars may vary considerably.

Simon and Strandberg (1998) identified a relationship between *A. dauci* resistance in the experiment and greenhouse resistance ratings. Although field testing is often employed, it is inefficient, costly, and difficult to maintain. To solve these challenges, experiments like as growth chambers, tunnels, and greenhouses are utilised. The bulk of field experiments focus on plant penetration. Fewer plants are utilised in controlled settings, sometimes just one specimen of a specific species or unconnected plant components. Baranski et al. studied transgenic plant resistance by inoculating detached leaves and petioles with a fungal pathogen (2007). Pathogen-treated greenhouse plants, according to Pawelec et al. (2006), are capable of effectively grading carrot varieties. Experiments with the excised leaf and hypocotyl, on the other hand, were a failure. To accelerate screening, utilise less plant material, and reduce environmental impact, a drop inoculation method was devised (Boedo et al. 2010). In addition, we investigated the sensitivity of carrot lines to A. dauci in vitro (Dugdale et al. 2000; Lecomte et al. 2014). To determine disease resistance, the chlorophyll content of damaged and excised leaves was measured in seedling hypocotyls from regenerant somaclone plants. Courtial et al. (2018) investigated A. dauci resistance in carrot embryogenic cell cultures. Because of the necessity for automated testing, these tests will help in high-throughput characterisation.

Breeders must understand the inheritance and combining capabilities of resistance sources in order to develop resistant hybrid carrot varieties. In the open-pollinated cultivar 'Brasilia,' resistance to *A. dauci* was shown to be 40% narrow-sense heritable (h^2) (Adults and their consorts.) A F_2 population of the carrot cultivars 'Kuroda' and 'Nantes,' used to investigate foliar leaf blight resistance, was reported by Vieira et al. (1991), who did not identify the most likely causal agent(s) as *X. hortorum* pv. *carotae*, but did report increased genetic variation. According to Simon and Strandberg (1998), a high amount of positive diversity, in combination with dominant genetic alterations and epistasis, may result in resistance to *A. dauci* in a plant population. Le Clerc et al. (2009) found three QTLs in an $F_{2:3}$ progeny population, demonstrating that disease resistance is polygenic. Each QTL explained between 10 and 23% of the phenotypic variation. The identification of particular QTLs in a tunnel or field experiment shows that they are environment-dependent and display expression delay after infection. Over a two-year period, two additional genetically distinct populations were studied in the field, yielding 11 QTLs. Because the advantageous alleles at each QTL are mutually exclusive, breeders may be able to raise resistance levels by mixing resistance alleles into a single genotype. In the case of carrots, certain QTLs may prevent pathogen entry into the epidermal tissue, whereas others may prevent pathogen invasion after the leaf has been pierced (Le Clerc et al. 2015b).

Understanding the processes of carrot foliar disease resistance is crucial for developing strong, highly resistant cultivars with a range of resistance mechanisms. Boedo et al. (2008) used resistant and sensitive carrot cultivars to test *A. dauci* resistance

and susceptibility in carrot leaves. Following inoculation 21 days later, SEM analysis indicated that the two cultivars grew differently (dpi). The fungus, for its part, quickly infiltrated the weak cultivar's leaf tissues. At 15 days post-infection, a quantitative real-time PCR assay established that the susceptible cultivar's leaves contained significantly more fungal biomass than the resistant cultivar's leaves, whereas by using a susceptible cultivar as a comparison, Boedo et al. (2010) revealed that two partly resistant varieties had considerably less fungal infection than one partially resistant variety. It was discovered that the two partly resistant cultivars of *A. dauci* had up to 3.42 ± 0.35% more germ tubes per conidium than the susceptible cultivar when *A. dauci* conidia were planted on carrot leaves in a laboratory setting (1.26 ± 0.18). The fungus is very infectious and spreads quickly via the skin. The spores of the resistant cultivar included several germ tubes per conidium, indicating that the fungus tried to enter the epidermis on multiple times.

Lecomte et al. (2012) studied *A. niger* resistance to falcarindiol and 6-methoxymellein (6-MM) in breeding lines infected with *A. dauci*. A statistically significant difference in 6-MM production between resistant and susceptible cultivars (Bolero vs. Presto) demonstrated that this phytoalexin helped to resistance by delaying disease transmission. In vitro, falcarindiol suppressed fungal growth and permeabilized *A. daucii* better than 6-MM. It is found in greater abundance in 'Bolero' leaves than in 'Presto' leaves, suggesting that it aids in fungal resistance. According to Lecomte et al. (2014) carrots are resistant to *A. niger* toxins. Dauci's involvement in the small resistance seems conceivable. To evaluate embryogenic cellular cultures derived from resistant carrot genotypes, fungi extracts were used. Overall plant resistance and cellular resistance to fungal exudates have a substantial association, showing that resistant and susceptible cultivars respond differently. More research is needed to determine the presence of phytotoxic chemicals in exudates. Fungal extracts were equally efficient as fungal extracts on carrot embryogenic cell cultures, but presented a reduced danger, according to Courtial et al. (2018). The fungus may produce aldaulactone, a very toxic chemical. It is necessary to identify its cellular targets. Koutouan et al. (2018) used bulk segregant analysis to examine the leaf metabolomes of four different carrot accessions with varying levels of resistance to *A. daucii*, as well as resistant and susceptible progenies. Bulk populations sensitive and resistant to camphene, caryophyllene, bisabolene, luteolin 4′-O-glucoside, and apigenin 4′-O-glucoside produced and accumulated feruloylquinic acid and luteolin 7-O-glucuronide in different ways. The relevance of those secondary metabolites in A. dauci resistance, as well as their relationship to previously identified QTLs, are being studied using metabolite QTL approaches and microarray testing to analyse gene expression in metabolic pathways.

Arbizu et al. (2017) proposed employing prediction algorithms based on the relationship between *Daucus* clades and *Alternaria* leaf blight severity ratings rather than screening wild and farmed carrot accessions for novel sources of resistance. A phylogenetic linear regression model using 106 wild and farmed *Daucus spp.* and related taxa revealed that plant height was the most important explanatory variable for disease resistance prediction. *Daucus carota* subspecies *capillifolius*, *maximus*,

and *crinitus* may have additional resistance sources. Carrots have been found to exhibit hybridization potential.

The approach was studied in order to create transgenic carrot plants that are resistant to fungal and bacterial foliar infections. Plant-derived lysozymes prevent and defend against bacterial and fungal infections. Both bacterial peptidoglycan and fungal chitin are cleaved by human lysozyme. *Agrobacterium tumefaciens* and the human lysozyme gene were used to create carrots resistant to *A. daucii* (Takaichi and Oeda 2000). Punja (2005) developed two thaumatin-like genetically modified carrot lines using *A. radiobacter*. In both lines, Sclerotiorum and A. dauci decreased sickness. In carrot transgenic plants, the *MF3* gene was studied. *Pseudomonas fluorescence i*s a plant-growth-stimulating rhizobacterium (Baranski et al. 2007, 2008). *MF3* is thought to be involved in the signalling cascade that results in induced systemic resistance due to its interaction with FKB. When compared to non-transformed plants, transgenic plants have a 20–40% boost in disease resistance. The polyethylene glycol transformation of carrot protoplast chitinase genes yielded less impressive results. Two of the clones were more resistant to *A. dauci*, whereas a third was more sensitive to the pathogen. According to researchers Wally et al. (2009a), monitoring a higher amount of induced genes was more effective than modulating gene expression in the creation of disease resistant transgenic lines. To change systemic developed tolerance, we upregulated the *NPR1* gene in a carrot cultivar. When *B. cinerea*, *A. radicina*, and *S. sclerotiorum* were used as pathogens, the transgenic lines significantly reduced disease severity by 80%, and when *X. hortorum* pv. *carotae* was used as a pathogen, the transgenic lines greatly reduced disease severity by 35–50%. Klimek-Chodacka et al. (2018) disclose the first effective site-directed mutation in the carrot genetic code, conferring resistance against foliar fungal and bacterial infection.

8.2.3 Cercospora Leaf Spot

Unlike Alternaria leaf blight, Cercospora leaf spot produces circular lesions on the leaves and petioles, and the leaves lack dark-edged borders and a lighter centre (Milosavljevic et al. 2014; Gugino et al. 2007; Raid 2002; Carisse and Kushalappa 1990; Bourgeois et al. 1998). The fungus only affects the aerial parts of carrots, not the root portions. Conditions for infection include temperatures ranging from 20 to 28 °C, followed by six hours of leaf wetness and 100% relative humidity (Carisse and Kushalappa 1992).

There is a scarcity of data on screening *C. carotae* for resistance. Lebeda et al. (1988) investigated the resistance of 142 carrot cultivars from throughout the globe to *C. carotae*. Resistance was found in just 30% of the cultivars tested in the field. Outdoor testing were carried out by Gugino et al. (2007). Resistance varied greatly amongst cultivars, although it was not constant. Cercospora leaf spot resistance is poorly understood, and little effort has been made to develop resistant plants (Table 8.9.1).

Both *X. hortorum* pv. *carotae* and *C. carotae* may infect carrot breeding lines.

Resistance to *C. carotae* may be mediated by a single gene with a range of morphological variations, according to Lebeda et al. (1988). Angel and Gabelman (1968) demonstrated that an inbred line's resistance was produced by a dominant gene using glasshouse research.

By infecting carrots with *Cercospora carotae*, Mercier and Kuć (1996) acquired systemic resistance. *C. carotae*-infected carrot leaves exhibited much fewer lesions than control leaves, showing that the foliar pathogen strengthened carrot leaf defence systems.

8.2.4 Bacterial Leaf Blight

Bacterial leaf blight is caused by *Xanthomonas hortorum* pv. *carotae*, a seed-borne pathogen. The foliar symptoms of *A. dauci* and *C. carotae* infections are identical to those of this fungus. Bacterial leaf blight creates a slimy, sticky discharge of bacteria. Petioles, umbels, and seed stalks have all been harmed (du Toit et al. 2005). In 1934, scientists in California found bacterial leaf blight. Any carrot field is at risk of being poisoned. It has been shown that the pathogen infects plant components such as stems, leaves, umbels, and seeds. According to the inquiry, tainted roots are a possibility. This is due to the fact that infection occurs exclusively at the crown, where the petioles contact the root. Certain seeds may be infected or diseased. If the pathogen is present, the seeds must be washed in hot water to kill it or dramatically reduce the degree of infection (du Toit et al. 2005; Pfleger et al. 1974).

As a consequence, little or no public study on the pathogen's genetic resistance has been conducted under these settings. There is no commercial cultivar that is blight resistant (Christianson et al. 2015). Pfleger et al. (1974) observed that the reactions of six cultivars and breeding lines to bacterial blight differed considerably from one another. They evaluated 66 carrot inbred lines, two public sector inbred lines, 17 marketable hybrids, wild or putative ancestors and land races for *X. hortorum* pv. *carotae* in a greenhouse, and they found that they were positive for the pathogen. There were eight putatively resistant PI lines found (two varieties and two carrot inbred lines), as well as five highly sensitive PI lines. To help in the production of more robust cultivars, one line from each of the three PI numbers 418967, 432,905, and 432,906 has been recognised as blight resistant. Each access point provides insufficient resistance. Only Ames 7674 and SS10 OR were identified to be bacterial blight susceptible. Infections of leaves with *X. hortorum* pv. *carotae* differed greatly amongst accessions. Visual estimates of foliar disease severity, according to Christianson and colleagues, are beneficial, but only if enough replications are performed (2015). Both investigations found a small positive association ($r = 0.52–0.62$) between sickness severity ratings and quantification of *X. hortorum* pv. *carotae*. This research highlights the significance of USDA's National Plant Germplasm System (NPGS) Daucus germplasm to plant breeders. Christianson et al. (2015)

investigated the resistance inheritance of *X. hortorum* pv. *carotae* using carrot PI lines.

C. carotae plants showed much fewer lesions than control leaves, showing that the foliar pathogen increased carrot leaf defence systems.

8.3 Soil Borne Diseases

8.3.1 Cavity Spot

A hollow patch has been noticed in almost every region where carrots are cultivated (McDonald 2002). *Pythium sulcatum* and *Pythium violae*, two species with a modest growth rate that feed on carrot roots, are the most common inhabitants in the United States (McDonald 2002). P. *intermedium, P. ultimum, P. sylvaticum* and *P. irregular* are likewise related with Cavity Spot. Surface lesions on roots make them undesirable for both fresh and processed markets (McDonald 2002). During the first four to six weeks after planting, carrot roots are more likely to remain infected with *Pythium spp* (McDonald 1994b). The hollow space at the base of the roots will be kept for storage purposes (Vivoda et al. 1991). Secondary microorganisms such as bacteria invading root lesions generate the colour surrounding the cavities. The hollow location deteriorates due to a lack of root development (Montfort and Rouxel 1988).

Despite the fact that no carrot cultivar is completely devoid of cavity spots at the time (Soroker et al. 1984; Groom and Perry 1985; Sweet et al. 1986; White 1988; Vivoda et al. 1991; McDonald 1994b, 2002). Some cultivars, according to Guba et al. (1961) are susceptible to cavity spot. 'Hutchinson' roots exhibited fewer hollow portions than 'Waltham Hicolor' roots, despite the larger diversity found across lines of 'Waltham Hicolor.' The National Institute of Agricultural Botany in the United Kingdom revealed differences in susceptibility among carrot varieties. Redca was a rougher Chantenay variety, whereas Nandor was a stronger Nantes cultivar. Furthermore, late-maturing plants were more prone to cavernous spot. Autumn King Vita Long is more resistant to delay harvesting than early harvesting (Sweet et al. 1989).

Six California Imperator cultivars were cultivated in growth chambers at 20 °C with *P. ultimum* and *P. violae* injected into the potting mix. In all six cultivars, both species were capable of causing cavity spot, with *P. violae* isolates being more virulent (Vivoda et al. 1991). 'Topak' was especially vulnerable to attacks from both species. P. violae was more resistant to the other five cultivars, although *P. ultimum* was very sensitive. The most vulnerable varieties were 'Pakmor,' and 'Caropak' followed by 'Dominator.' and 'Sierra'. Vivoda et al. (1991) found that the lack of diversity in reaction to *P. ultimum* and *P. violae* might be attributed to the cultivars' ancestors.

White et al. (1987) examined 19 commercial carrot varieties for resistance to the hollow spot lesions *P. violae, P. sulcatum* and *P. intermedium*. They found that the

varieties were resistant to all three pathogens *Pythium* species were colonised on roots produced in a greenhouse using agar plugs that had been cleaned and colonised. *P. violae* was found in 19 different carrot cultivars and in all five types of carrot. Cavity spot variations in *P. sulcatum* have been discovered in different carrot kinds, but not in different cultivars of carrot. White et al. (1987) found significant variation in *P. intermedium* only between cultivars and in one of three cavity spot tests, and only in one of three cavity spot assays. Any of the three Pythium species that were evaluated showed no signs of having a genetic advantage in terms of resistance.

According to White et al. (1988) *Pythium spp.* was found in the periderm of asymptomatic carrots from the cultivars 'Sweetheart,' 'Chantenay New Supreme, and 'Fingo,' as well as in the periderm of symptomatic carrots from the cultivars 'Sweetheart' 'Chantenay New Supreme,' and 'Fingo,'. Following infection with mycelial plugs from the pathogens *P. sulcatum*, *P. intermedium* and *P. violae*, they discovered that genetic resistance was absent in 19 carrot cultivars from five distinct groups. Mycelial plug inoculation, according to Vivoda et al. (1991) did not give a credible measure of cultivar resistance variation. They discovered that infecting 36 carrot cultivars in the lab with *P. violae* resulted in susceptibility variations that were similar with their field findings. Using a combination of field nurseries, greenhouse screening, and laboratory root injection studies, a large number of individual breeding programmes have made significant progress in creating hybrids with improved resistance to hollow spot in recent years.

McDonald (1994b) demonstrated that the partially resistant 'Six Pak' varieties was effective for cavity spot elimination in the province of Ontario. The Chanton and Huron were the most susceptible species, with Red Core Chantenay, Eagle, and SR-481, showing intermediate resistance to mortality. Six Pak elicited a greater number of negative reactions than either 'Cellobunch' or 'Chancellor.' In non-irrigated regions, 'Eagle' was as resistant to blight as 'Six Pak,' but was more susceptible in irrigated plots. The susceptibility of the cultivars to cavity spot changed only as the roots grew in size. For the first time, this study demonstrated that stored carrots are not always more susceptible to hollow spot lesion than freshly harvested carrots, as previously thought. Towards the end of the season, fewer hollow patches were seen (McDonald 1994b).

Benard and Punja (1995) used in vitro mature root inoculation to assess cavity spot reactivity in 37 carrot cultivars. The most resistant strains were E0792, Fannia, Caropride, Panther, and Navajo. "Six Pak," "Imperator," and "XPH 3507" were shown to be resistant to the pathogen despite only having been tested once. "Eagle," one of 18 cultivars evaluated in 1991 and 1992, was found to be resistant in 1991, but susceptible in 1992, despite the fact that the results for the other cultivars were equal in both years. They hypothesised that year-to-year differences in cultivars were caused by rootstock or growing conditions. They assessed the vulnerability of commercial carrot varieties Narbonne, Bolero, Bertan, and Eastern carrot gene bank variation 'Purple Turkey,' to *P. violae* inoculation under the greenhouse phenotype screening and field experiments. In compared to other commercial cultivars, 'Purple Turkey' outperformed them in terms of quality and yield. Resistance to cavity spot is suggested to be a result of the 'Purple Turkey's' tiny cell size and enhanced enzyme

levels in the tap and adventitious roots. The study examined commercial cultivars like as 'Bolero,' 'Narbonne,' and 'Bertan.'

Cooper et al. (2006) studied cavity spot resistance in carrot seedlings from 19 somoclonal derived lines as well as commercial control varieties such 'Vita Longa', 'Nando,' 'Bolero,' and 'Bertran,' Although hollow spot susceptibility differed genetically amongst somaclones, there was no association between greenhouse and outdoor data. For many years, scientists at the University of Guelph's Muck Crops Research Station in Ontario's Holland Marsh have compared USDA experimental carrot breeding lines to commercial carrot cultivars. Infection of a cavity disease by a naturally existing pathogen in the area. Hollow spots emerge in variable degrees in breeding lines and cultivars from year to year. Orange parent lines CS736 and CS732 outperform USDA parent lines viz., 5367, 6526, and 1137 in terms of cavity spot resistance (1137B-F2M5). 2205B, 2205, 5494, and CS 724, as well as additional crosses with those lines, have all shown consistent responsiveness. Despite a very consistent disease burden in this nursery, determining cavity spot resistance was difficult (McDonald et al. 2017). In the muck nursery experiments, there was no link between carrot root forking and the existence or severity of cavity regions (McDonald et al. 2017).

Screening for cavity spot resistance is challenging due to the unequal distribution of field inoculum and the intermittent character of the diseases. Because carrot roots from the same cultivar respond so differently, a significant number of marketable roots from each carrot inbred line must be tested in duplicated and randomised plots over different seasons to allow for meaningful different responses. A variety of factors (including soil microbiology) may impact the presence and severity of hollow spot when phenotypic screening procedures are applied (McDonald 1994b, 2002; Benard and Punja 1995). It grows well in wet soil (especially after a flood) and at cold temperatures (*15 °C). Extensive roots in the soil exacerbate the hollow region (Montfort and Rouxel 1988). This might be due to increasing root sensitivity, the accumulation of seasonal lesions, root diameter expansion, or an infection change (Wagenvoort et al. 1989; Vivoda et al. 1991). Despite comparable spore concentrations and environmental conditions, symptom severity and frequency differed amongst genotypes later in the season, despite same inoculum and environmental conditions (McDonald 1994b). She discovered that increasing the severity of hollow spots did not always mean that roots were more sensitive with age, but rather that the illness advanced. Carrot age (1–3 months) has no effect on the formation of cavity spots, according to Benard and Punja (1995). The number of lesions per root rose three to five months after planting, according to Vivoda et al. (1991). McDonald (1994b) found that seasonal oscillations in hollow spot were caused by climatic variables rather than plant age, implying that the timing of cavity spot exams may influence disease resistance screening activities. Several breeding programmes have used mature carrot roots injected with *Pythium spp.* agar plugs to test cavity spot resistance. Root inoculation lesions, in contrast to lesions caused by roots growing in contaminated soil or planting medium, are often shorter, discoloured, and lack distinct boundaries (Vivoda et al. 1991). Screening for cavity spot resistance using colonised agar plugs, according to Vivoda et al. (1991), may not adequately represent

cultivar or breeding line response in soil. Because of quick epidermal suberization, which precludes root infection, carrot roots may be infected with *P. violae* colonised agar plugs 24 h after harvest. Root inoculation with P. sulcatum-infected plugs, on the other hand, may be done up to a week after harvest provided the roots are maintained cold to minimise root suberization. To overcome these challenges, several root inoculation bioassays involved cutting the tips of the roots prior to collection and immersing the roots in water until infected (Cooper et al. 2004). Other methods for improving root inoculation uniformity include culturing roots in the dark for 7–10 days at low temperatures (15–20 °C) and high RH. A significant number of roots must be afflicted and examined in order to accurately determine the extent of the lesion at various inoculation locations on the same root and throughout different backgrounds of the same plant. Their value in carrot breeding is restricted because to the time required to inoculate root agar plugs. Others have grown sick roots in high relative humidity settings to assess the size of the hollow patches. According to Suffert and Montfort (2007), introducing an inoculated and diseased carrot root to the same environment as healthy carrot roots may result in the growth of hollow spot lesions in the carrots. This method resulted in a greater number of cavity spot lesions than *P. violae* inoculated soil.

Several publications have been written about cavity spot analysis methods. Each lesion's severity is determined by its amount of lesions per root, its horizontal and/or vertical lengths, as well as any combination of these two lesion parameters (McDonald 1994b), and classification of lesions as small, medium, or large have all been used to analyse large numbers of roots. When a variety of evaluation procedures are utilised, comparing outcomes may be challenging. Because the frequency and severity of cavity spots vary seasonally, evaluating incidence or severity at a certain harvest date may provide different findings. In Canadian field study, McDonald (1994b) discovered that *AUDPC* was more successful than incidence ratings in identifying treatment effects. Several exams are required to establish the *AUDPC*. The slopes and altitudes of disease emergence curves in the *insitu* may be used to estimate cultivar resistance to cavity spot (McDonald 1994b).

According to research, Cavity Hole Growths are induced by a hypersensitive reaction of carrot root core tissues to *Pythium* infections (Klisiewicz 1968; Endo and Colt 1974). According to other researchers, there was practically little variation in quantitative resistance amongst cultivars of the same species (White 1991; Johnston and Palmer 1985). In terms of published (open-access) research, there does not seem to be any on the inheritance of cavity spot resistance available. Cavity spot lesions are caused by the enzyme cellulose and the pectate lyase of *Pythium spp.* (Cooper et al. 2004). Degrading enzymes of cells are triggered during hyphal penetration of root tissue (Guérin et al. 1994; Campion et al. 1988; Campion et al. 1988). *Pythium spp.* isolates that are extremely pathogenic, according to Benard and Punja (1995), generate more pectolytic enzymes than isolates that are moderately hazardous. As the infected zone killed host cells and hyphae developed under the epidermis, a hollow was formed. When carrot roots get infected, they produce oxidised phenolics and phenylalanine-ammonia lyase, which are subsequently deposited around the site of infection. Furthermore, it is thought that the lignin that forms surrounding the

lesion functions as a physical barrier against infection. The internal distribution of pyrthium has been connected to hollow spot resistance (Endo and Colt 1974). These results reveal that in order to battle infection, root defence mechanisms are engaged in response to cell disintegration (Soroker et al. 1984; Perry and Harrison 1979). The amount of phenol in cavity lesions tissue increased with the severity of the hollow spot lesions. Lignin and suberin were found in the periderm cell membrane, and in parenchyma cells at the infected outer region of the carrot (Perry and Harrison 1979). Chemical accumulation of antifungal drugs was more essential in *Pythium* resistance than structural barriers. Falcarindiol and phytoalexin 6-methoxymellein have been isolated from healthy root tissue, while phytoalexin 6-methoxymellein has been extracted from diseased root tissue (Garrod et al. 1978). Kurosaki et al. (1985). Guérin et al. (1998) showed that more resistant cultivars had thicker cell walls, which they hypothesised was due to higher synthesis of phenolic fungitoxic compounds as a result of the infection responses. According to Cooper et al. (2004), 'Purple Turkey' has a smaller root cell width and greater levels of constitutive enzymes than commercial cultivars, which explains for its cavity spot resistance. The pace at which a carrot root reacts to infection, according to White et al., may be connected to its sensitivity to cavity spot (1988). Pythium spp. were found in juvenile tissue more often than in mature tissue eight weeks following planting. As a result, either the carrot's defence mechanisms protect it from infection by these fast growing organisms (McDonald 1994b). Slow-growing plants, such as *P. sulcatum* and *P. violae*, prpdice the cavity reactions. Slow-growing organisms entered carrot core tissue for 3–4 days, liberate minute levels of degrading enzymes of cell wall before eliciting a host reaction, according to White et al. (1988) and Zamski and Peretz (1995). Patches of carrot root cavity degrade often during cold storage (McDonald 1994b). Bolting, a physiological change from vegetative to reproductive growth caused by vernalization, might be linked to enhanced storage vulnerability. Furthermore, storage may increase the number of lesions per root, indicating that latent disorders may resurface during storage. Minor cavity spot lesions may cure on their own if treated properly (McDonald 1994b).

8.3.2 White Mold

While *Sclerotinia* soft rot, often known as white mould, does minimal damage in the field, it is harmful to cold storage and long-distance shipment. Sclerotia are black animals with a melanized surface that colonise open root zones quickly and change into mycelium, a white flocculent mycelium. Sclerotia may live in the soil for up to ten years. Three *Sclerotinia* species have been linked to the pandemic (Leyronas et al. 2018). *Sclerotium rolfsii*, a basidiomycete unrelated to white mould, causes carrot southern blight. Ascomycetes are pathogens that cause white mould. White mould may be found on roughly 500 different species, including weeds, all across the globe (Rubatzky et al. 1999; Kora et al. 2003). A phenotyping test was employed *Sclerotiorum sclerotiorum* on different carrot accessions (Ojaghian et al. 2016). After

three minutes in 2% sodium hypochlorite, the carrot roots were washed with sterile tap water and dried on sterile filter paper. Fungi grown on carrot dextrose agar were used to inoculate the roots. An agar plug with a diameter of 5 mm was constructed from the tip of a 3-day-old culture and was used to insert the root in the core of the agar plug. A damp chamber was made using 12 plastic boxes (12 carrots each). The roots were kept in humidified cotton wool trays at 21–23 °C. Lesions Serious disease was defined as 1–4 cm in length without sclerotium development, 4–8 cm in length with 1–4 mature or immature scales, and 8 cm in length with more than 4 mature or immature scales six days after inoculation. The illness index was computed as [(1.25) + (2.53) + (3.75y4)/total carrots 1/0.05, where 0.05 is a constant (Ojaghian et al. 2016).

Using detached petioles and leaflets, Punja and Chen (2004) revealed that transformants of carrot plants encoding a rice thaumatin-like protein exhibited considerably enhanced understanding the consequences when *Sclerotiorum sclerotiorum* was injected. According to Wally et al. (2009b) carrot breeding lines upregulating the peroxidase enzyme *OsPrx114* were shown to be particularly resistant to *Sclerotiorum sclerotiorum.* Pathogenesis-related (*PR*) gene transcript levels rose in tissues treated with *S. sclerotiorum* cell wall fragments (Wally and Punja 2010).

8.3.3 Gray Mold

Botrytis cinerea, sometimes known as grey mould, has the potential to devastate temperate Asia, Europe, and North America (Rubatzky et al. 1999). Spores are the principal disease vectors in crops. The development of symptoms is accelerated by cold storage. Carrot roots are often attacked by the fungus at the petiole base or crown. Watery brown lesions develop into dark brown lesions with grey mycelium and minute sclerotia as they grow. The root inoculation resistance experiments were meant to see whether carrot varieties are sensitive to *B. cinerea* during cold storage and to look into artificial resistance (Goodliffe and Heale 1975; Bowen and Heale 1987). To measure the vulnerability of carrot leaves to grey mould, Baranski et al. (2006) designed a foliar test employing colonised agar plugs. Heat-killed *B. cinerea* conidia in carrot slices, according to Mercier et al. (2000) conferred systemic resistance to *B. cinerea.* There is considerable disagreement over whether a 24-kilodalton chitinase plays a role in induced resistance. Transgenic carrot plants expressing *CHIT36*, a chitinase lytic enzyme produced by the biocontrol agent *Trichoderma harzianum*, to study the effect of chitinase on grey mould. *B. cinerea*'s assault on transgenic plants has been decreased by up to 50% (Baranski et al. 2008).

8.3.4 Fusarium Dry Rot

Fusarium dry rot has been detected in the China, United States, Canada, Japan, and France, (Zhang et al. 2014; Villeneuve 2014; Sherf and MacNab 1986; Rubatzky et al. 1999). There is a chance that some businesses may incur major financial losses (Zhang et al. 2014; Villeneuve 2014; MacNab 1986; Rubatzky et al. 1999). Losses in China's Tuo Ke Tuo County topped 80% in 2014 (Zhang et al. 2014). A dark circular lesion with a diameter of 3–4 cm covers the root surfaces. Soft rot disease is caused by lesions, rendering the roots unmarketable. Nutrient transmission between roots and leaves, on the other hand, may be influenced by root quality and production. Disease might result in significant loss during storage. The four species that cause this disease, according to the CDC, are *avenaceum*, *culmorum*, and, most recently, *Fusarium caeruleum.* In order to examine variance in variety, Zhang et al. (2014) proposed two ways for replicating frequent symptoms. To begin, 5 mm diameter plugs were carved into potato dextrose agar plates. On this side, a mycelial plug was inserted into the root. Infected roots were incubated in a humidified atmosphere at a temperature of 25 °C (90% relative humidity). White mycelium covered the root surface, forming black bruises after four days of incubation. The second step was to fill each container with 15 carrot seeds (30 cm 25 cm). The soil contained 1104 CFU/g of spore suspension. Plants grown in uninfested soil were used as the control treatment. Each risky factory was assigned a field. Dried red emerged after 13 weeks. In the absence of known resistance sources or published varietal testing, Sidorova and Miroshnichenko (2013) confirmed genetic change. When coupled with 'Nantskaya 4,' this gene was demonstrated to be resistant to *F. avenaceum* infection.

8.3.5 Black Rot

The bacteria *Alternaria radicina* is responsible for black carrot rot (formerly *Stemphylium radicinum*). Black rot was often reported as a post-harvest disease, infection of plantation seedlings, and contamination of carrot seed harvests. Radicine induces leaf, petiole, and umbel blackening (Meier et al. 1922). The first black red record was made in New York. Planting or concealing disease-related problems Radicine may remain in the soil for up to eight years, causing carrot crops to become ill (Maude 1966; Scott and Wenham 1972; Pryor et al. 1998; Farrar et al. 2004). The black red taproot and crown are divided by dark, deep necrotic lesions. When harvesters remove reproductive tips from the ground with their heads in moist settings, a coronary infection may cause petiole rot and bladder symptoms similar to *Alternaria dauci*, culminating in catastrophic plant loss (Pryor et al. 1998; Grogan and Snyder 1952; Farrar et al. 2004). The pathogen quickly spreads throughout the root system after root infection. Seed production and germination may be hampered if the umbel becomes sick. Fungicides such as azoxyestrobin, fludioxonil, Iprodon, or thiram, as well as disinfectants such as hot water or sodium chlorite, may be used to reduce

seed-borne inoculum (Pryor et al. 1994; Biniek and Tylkowska 1987; Soteros 1979; Maude 1966). Chen and Wu (1999) revealed that 229 *Burkholderia cepacia* and 224 *Bacillus amyloliquefasciens* had a substantial affect on A. radicina. Prior to *A. radicina* infection, *Candida melibiosica* yeast was shown to suppress the development of black rot (Kordowska-Wiater et al. 2012). Pryor and his colleagues After sterilisation, the teeth were cultured for five days at 28 °C with 2 ml A. radicina conidia (1 t/104 conidia/ml). The colonised toothpick tip was put into the shoulder of a ten to twelve-week-stored carrot root after nine to ten weeks. Grzebelus et al. (2013) created a protoculum for selecting plants that outperform *A. radicina* in vitro. In protoplastic cells attacked by fungus, somaclonal alterations were seen, leading in disease-resistant plants. Cwalina-Ambroziak et al. (2014) used agar discs to inoculate *A. radicina* petioles and seedlings (every 5 mm in diameter).

In 46 field-grown carrot crops, Pryor et al. (2000) observed substantial diversity in cultivar lesion frequency. While Panther and Caropak were resistant, Royal Chantenay and Nogales were quite suceptible. While cultivars were resistant to *A. radicina*, lesions occurred faster in cold storage than in the field conditions. A black-red experimental investigation with production in 2008–2009 and achieved a wide range of findings (Karkleliene et al. 2012). Magi was the most sensitive to *A. radicina* of the 13 varieties tested. According to Cwalina-Ambroziak et al. (2014) Koral exhibited more suceptible than Bolero.

Baranski et al. (2008) used transgenic *CHIT36* plants to confirm the positive effect of chitinase on *A. radina* in vitro, which had previously been documented for the grey form produced by Botrytis cinerea. The number of those infected with *A. radicina* was cut in half. The gravity of the *A. radicina* taproot (width of injuries lowered by 50%) and the quantity of necrotic foliar patches (approximately 33% reduction in the measure of severity of foliar disease) were dramatically reduced when transgenic plants expressing the *NPR1* gene were infected. *P23*, a cationic peroxidase inhibitor in rice that improved resistance to necrotrophic foliaire infections, was studied by Wally and Punja (2010). Overexpression of *OsPrx114* increased the lignin synthesis in the outer peridermal tissues and pathogenesis-related (*PR*) genes according to Wally and Punja (2010).

8.3.6 Bacterial Soft Rot

Bacteria such as *Pectobacterium carotovorum* subsp. *carotovorum*, *Dickeya dadantii*, *Pectobacterium atrosepticum* subsp. *atrosepticum*, Bacterial soft rot of carrots is a critical concern during storage because secondary invaders of damaged or diseased roots may cause significant losses. Soft rot symptoms are more common in low-lying locations and other saturated places (e.g., near broken irrigation pipes). These bacteria, as thermophilic facultative anaerobes, have been linked to major outbreaks in fields with extended wet soil conditions and high temperatures (Farrar 2002). Irrigation water and the water used to wash carrot roots after harvesting both have the potential to contain pathogens (Segall and Dow 1973). Small, water-soaked blisters

appear on carrot roots as a result of a highly contagious bacterial disease. The squishy roots of *D. dadantii* and the infected roots of *P. carotovorum* subsp. *carotovorum* become mushy and squishy when the temperature is high (30–35 °C for *D. dadantii* squishy) and 30–35 °C for *P. carotovorum* subsp. *carotovorum* and infected roots of *D. dadantii* become mushy and squishy (Phillips and Kelman 1982). If infected roots have been macerated, internal tissue may flow through cracks in the root surface, resulting in an infection (McDonald 1994a). For determining carrot resistance to soft rot, a number of different approaches are available (Michalik and Ślęczek 1997; Michalik et al. 1992; Lebeda 1985; Bedlan 1984; Skadow 1978). It was shown that if the roots were kept at 21 °C for four days after being exposed to 2 °C for three days, they would experience more soft rot than if they were exposed to 2 °C for three days followed by four days at 21 °C. According to the findings of the research, phenolic or similar compounds generated during chilling may result in less severe soft rot in infected carrots. Carrot roots harvested immediately after harvesting contained 3 methyl-6 methoxy-8 hydroxy-3, 4-dihydroxoisocoumarin, but carrot roots stored at 0 °C for 4–8 weeks did not. According to Segall and Dow (1973), this may aid in the prevention of bacterial soft rot in carrots when in cold storage.

Michalik et al. (1992) assessed the resistance of carrot germplasm collections to soft rot caused by *P. atrosepticum* and *P. carotovorum* subsp. *carotovorum* using four root inoculation procedures. The roots were cleansed in sterile water and air dried after being stored at 0–4 °C for 1–3 weeks (Michalik et al. 1992). A fungicide was applied to soil samples and they were held at 22 °C for 48–96 h. Both bacteria elevated the severity of soft rot in response to increasing inoculum concentration, although *P. carotovorum* subsp. *carotovorum* colonised more significantly than the isolate of *P. atrosepticum.* Amount of the bacterial strain carrot line had no impact. In compared to treatments with larger root pieces, both carrots cut root inoculation strategies resulted in increased rot severity and a decreased response variance. Using bacterial-soaked filter discs, the inoculum was not dried by evaporation. Individual root slices also allowed for repeated seed creation and screening. The amount of time carrot roots were stored after harvest had no influence on soft rot (2, 6, or 12 weeks). The findings were same whether the roots were utilised whole or cut; however, the root tip was more responsive. The diversity of carrot lines revealed that breeding for resistance to soft rot may be advantageous (Michalik et al. 1992).

Michalik and Ślęczek (1997) investigated resistance to *P. carotovorum* subsp. *carotovorum* in progeny by crossing carrot orange cultivars, four Uzbek Mirzoe varieties and five wild *Daucus carota* subspecies. They detected genetic variation in orange carrot cultivars susceptible to soft rot, although it was insufficient for breeding purposes. They infected carrot root discs using filter discs that had been immersed for 30 min in a bacterial solution (5 × 106 CFU/ml). Despite an increase in the severity of soft rot in the F_2 generation, one indigenous Mirzoe cultivars shown promise as a source resistance. Carrot inred lines, open-pollinated varieties and F_1 hybrids all exhibit considerable differences in their susceptibility to bacterial soft rot, according to a group of German researchers. For the purpose of avoiding misunderstandings, any laboratory screening technique must be reinforced by field evaluations during phenotypical and storage stage. It is an imperative to use roots that have been planted,

harvested, and kept in the same location (Michalik and Ślęczek 1997; Michalik et al. 1992; Lebeda 1985; Skadow 1978).

8.3.7 Crown Rot

Carrot infections have been linked to *Rhizoctonia carotae* and *Rhizoctonia crocorum* (Davis and Raid 2002). They were buried alive in their entirety. *R. solani* may be found in practically every soil type. *Rhizoctonia solani* infections cause seedling damping-off and crown rot in adult carrots (Nuñez and Westphal 2002). The most common anastomosis groups for carrot damping-off pathogen isolates are AG-2, AG-1, and AG-4 (Nuñez and Westphal 2002; Grisham and Anderson 1983). Damping-off thrives in cold, moist soils where seeds have a difficult time germinating and emerging. Damping-off results in root dieback, seed rot due to apical meristem loss, seedling mortality before to or during emergence, and stunted seedlings (Nuñez and Westphal 2002). Crown rot is a problem in muck soils with a high organic matter content, and it often manifests itself just before to harvest (Punja 2002b; Howard and Williams 1976). The disease appears late in the season, when the leaves begin to age quickly, sometimes in patches. Toxic fungus causes dark brown lesions in the crown of the plant and, in rare cases, beneath the root of the plant (Punja 2002b). Crown rot lesions and cavity spot lesions are quite similar in appearance. Lesions in the crown or taproot reduce the marketability of the roots, and bacterial penetration may result in soft rot. In wet wounds, mycelium that resembles a web may grow. Lesions form when roots are stored. Howard and Williams (1976) reported that based on varietal responses in *in-situ* with different levels of treated pathogen and disease-friendly circumstances, it has been hypothesised that certain cultivars are somewhat resistant to crown rot.

Violet root rot (*R. crocorum*), which damages a broad range of plants, including carrots, parsley, parsnips, celery, and fennel, as well as table beets and potatoes (Punja and McDonald 2002; McDonald 1994e; Cheah and Page 1999). Violet root rot affects carrots all across the globe, although it has been particularly severe in Europe, New Zealand, and Australia. The first signs of this disease are usually dead or wilting plants with dirt sticking to their roots. The roots create substantial dark purple-brown lesions that are coated in a thick mat of fungal mycelium with a leathery look that ranges in colour from violet to dark brown. Between the plants, a thick brown mycelial mat may form (McDonald 1994b). The root decomposes gradually underneath the lesions. Violet root rot symptoms occur later in the season and may last into winter. Carrot roots may get infected at temperatures ranging from 5 to 30 °C, with a predilection for temperatures between 20 and 30 °C, according to the USDA. But in places with high soil moisture content, low pH, and nitrogen scarcity, the problem is more severe than in other locations. (Garrett 1949; Cheah and Page 1999). Dalton et al. 1981 proposed that in three naturally infected *R. crocorum* sites in the United Kingdom, susceptibility testing found no change in sensitivity to violet

root rot. In New Zealand, violet root rot was reported in all commercial carrot varieties that were examined (Cheah and Page 1999).

R. carotae is a postharvest fungus that causes crater rot in carrots that have been stored for a long time (Punja 2002a). There have been no reports of other plant species being affected. Crater rot is a mould disease that infest North America and Northern Europe, causing up to 70% damage in Denmark (McDonald 1994a). With the influence of milky white mycelial lining and adhering to the root surface as well as dark brown colored sclerotia, roots form dry, deep craters or pits under humid, cold storage conditions (Punja 2002a; McDonald 1994c). When exposed to moisture, the storage fungus spreads very fast. In the case of Crater Rot, the root system has been contaminated by bacteria. In the field, latent root infections may occur, and roots with senescent foliage retain a greater amount of inoculum than healthy roots. The fungus thrives when a layer of water accumulates on the roots or when the relative humidity is high (Punja 2002a). *R. carotae* may grow at temperatures as low as 1 °C (Punja 1987). Carrot harvesting is postponed until late October, exposing the crop to disease.

Sowing carrot seeds in cool, moist, poorly drained soils, or overwatering immediately after planting, may result in damping-off, which require additional screening (Nuñez and Westphal 2002). Screening experiments have indicated that raised beds improve soil drainage and damping-off. The ability to discern between carrot varietal responses to various damping-off organisms, such as *Pythium spp*., may be difficult to determine unless carrots are tested in sterilized or pasteurised soil or the other sowing media containing specific organism, or unless seed is treated with a mefenoxam fungicide.

After four weeks of treatment with *R. solani*-infected maize kernels, Howard and Williams (1976) counted the number of atypical and normal roots at 16–20 weeks. A highly virulent *R. solani* strain was introduced to flasks containing sterilized maize grains after two weeks at 20–24 °C and spun every 2–3 days to achieve homogeneous fungal colonisation of the corn kernels. They did, however, advise that each test be carried out with "fresh" inoculum. The most successful technique, as Mildenhall and Williams (1970) had discovered, was to cut carrots three weeks after sowing and then inject pathogene inoculum 7 days later. Howard and Williams (1976) advocated growing carrots at temperatures of 20, 24, or 28 °C to minimise crown rot and maintaining a soil moisture level of 0.1 bar. Growing carrots close together to create a humid microclimate, as well as confronting the crown and petioles with filthy soil or carrot detritus, may increase the risk of crown rot (Punja 2002b; Gurkin and Jenkins 1985). In resistance screening trials, adding inoculum into colonised grain kernels and to the soil or other potting media may increase disease stresses (Breton et al. 2003).

Because high soil humidity and low soil pH promote violet rot, using acidic soils or an acidifying medium may help with screen resistance, as disease frequency and severity increase when infected soil roots remain in the soil for a long length of time (Garrett 1949; Punja and McDonald 1994e; Punja and McDonald 1994e; Cheah and Page 1999; McDonald 2002). In three naturally infected *R. crocorum* field sites in the United Kingdom, three Berlicum species, six Feonia or Imperator species, and one

unknown type) were studied. Commercial carrot varieties, according to Cheah and Page, were similarly sensitive (1999). A lack of disease pressure in one site excludes cultivar variations in violet root rot response, while a high amount of disease burden in another place prohibits cultivar differences in violet root rot response. According to Dalton et al. (1981) Western carrots were evolved by selection or intercrossing of closely related varieties such as Early Half Long, Early Scarlet Horn, and Late Half Long. Resistance should be found in anthocyanin and yellow cultivars, which are the forerunners of western cultivars. Resistance testing for violet root rot is still required.

Carrot root hyphae may quickly blanket a carrot root in the absence of appressoria or other infection structures, penetrating the root surface and causing root cell injury (McDonald 1994a). Roots may become unmarketable after three weeks. Despite the fact that crater rot is a postharvest disease, root screening should be beneficial due to the pathogen's aggressive tendency when kept in cold, moist settings. To include wounding into a screening procedure, roots are wounded, causing crater rot to form. Adopting a soil inoculation strategy may be difficult due to latent field infections.

8.3.8 Rubbery Brown Rot

In damp soils, carrot root rot (*Phytophthora* root rot) is a common disease that may be devastating. It often emerges after a period of heavy rain or irrigation (Browne 2002). All of these species, including *P. porri, P. megasperma, P. cryptogea*, and, *P. cactorum*, have been connected to disease in the past. The fungus *Phytophthora* root rot has been found in the United States, Norway, Australia, Canada, and France among other places (White 1945; Rader 1952; Stelfox and Henry 1978; Ho 1983; Browne 2002; Saude et al. 2007). During storage, the roots become black to dark brown and become rubbery. On the other hand, the symptoms usually appear after a long time of root storage. These solid lesions cause harm to the root's centre and crown (Saude et al. 2007). France has sustained severe agricultural losses this winter. On root lesions, a white mycelium may form. Soft rot develops when bacteria and fungi infiltrate wounds. Soaking carrots in water for an extended period of time during cultivation, storage or processing steps increase the zoospores production and its invasion. Cool to moderate temperatures aid in the formation of inoculum and the spread of disease.

There is a scarcity of information about testing carrots for *Phytophthora* root rot resistance. According to Stelfox and Henry (1978), the pathogen was detected in cold storage carrot variety 'Imperator II' in Alberta, Canada, in 1969–1970, and it has since spread around the world. Aside from the fact that they were gathered and washed, there was no detrimental influence on them. Saude et al. (2007) detected this disease on carrot processing farms in Michigan, however they did not include any information on specific varieties or changes in disease severity between cultivars in their findings. It should be possible to evaluate breeding lines or carrot varieties for resistance against rubbery root rot using a procedure similar to that used to screen for cavities.

Inoculation of agar plugs on the spot root (Stelfox and Henry 1978; Saude et al. 2007). The pathogen was cultivated on cleaned carrot roots for up to seven days in conditions ranging from high relative humidity to low to moderate temperature. Infected roots were examined at between 20 and 25 °C in many investigations on *Phytophthora spp.*; however, the optimal temperature varied depending on the *Phytophthora spp.*, studied. After a week at 20 °C, symptoms began to manifest, but not until seven weeks after the temperature was lowered to 0 °C (McDonald 1994d). They discovered that no damage was necessary for this kind of inoculation to cause rubbery root rot symptoms. Wounds caused a wide range of symptoms. One method for phenotypic resistance screening is to keep carrots at 20 °C with a high RH (>95%) to imitate the saturated soil conditions required for the formation of *Phytophthora* spores.

8.3.9 Common Scab

Infections with the fungal pathogen *Streptomyces scabies* are the cause of carrot scab. Although it may be found around the world, it is most widespread in Europe and Canada, notably the Netherlands and France (Villeneuve 2014; Janse 1988). Viruses and bacteria spread via lateral secondary roots or wounds, causing the death of latent epidermal cells to occur. After a few months, a corky protrusion appears on the root surface, with the most prominent protrusion appearing at the top. As a saprophyte, *Streptomyces scabies* may persist in soil for years at a time. Schoneveld (1994) observed that the most sensitive period for *S. scabies* infection was 4–5 weeks after spring planting, which corresponded to the period following spring planting. A 60-mL volume of bacterial culture (107 spores/mL) was treated with 20 L of sterilized loamy soil with a pH of 5.9. The plants were cultivated at 18 °C, 10,000 lx light, 80% relative humidity, and 50% soil saturation. Four months after seeding, roots were collected and checked for symptoms. The germplasm of carrots is sensitive to common scab.

8.4 Virus Diseases

Carrot viruses have infected around 14 individuals (Moran et al. 2002; Nuñez and Davis 2016). The economic repercussions of various ailments varied. Several viruses, such as AMV, CTLV, and TSWV, have little economic consequences (Lebeda and Coufal 1985; Stein and Nothnagel 1995; Nuñez and Davis 2016). The most common and persistent carrot virus is mottled dwarf (CRLV and CMoV) (Watson and Sarjeant 1964; Waterhouse 1985). There has been a paucity of extremely efficient viral and/or vector resistance, according to attempts to categorize it (Elnagar and Murant 1978; Van Dijk and Bos 1985). There are differences in virus susceptibility across carrot breeding lines, which may help explain why commercial cultivars are so resilient.

8.4.1 Motley Dwarf

Carrot cultivars respond to motley dwarf in various ways, and some are resistant (Koike et al. 2002). Danvers, a delicate California cultivar, was determined to be CVC-14 resistant (Watson and Falk 1994). It's difficult to tell the difference between the two cultivars when it comes to resistance to the willow aphids (Dunn 1970). Autumn was susceptible to aphids but resistant to motley dwarfs, according to Dunn (1970). Nantes, on the other hand, was more suceptible to motley dwarf and had a lower tolerance for aphids. 'Kurnella Strongtop' and 'Western Red' are motley dwarf tolerant, according to Tomlinson (1965), while only 'Western Red' is motley dwarf tolerant, according to Kinsella (1966). 'Early Market', 'rootless Cluseed Stump,' and 'Nantes,' to name a few. He saw a broad variety of dwarf symptoms. Dunn (1970) reported that both cultivars were resistant to *Aegopodii*, with 'Berlikum' being the most resistant.

8.4.2 Carrot Virus Y *(*CarVY*)*

Carrot virus Y (*CarVY*) has been found in every common carrot type in Australia, producing a range of symptoms (Latham and Jones 2004). Green peach aphids (*M. persicae*) attacked 22 Apiaceae plants in a glasshouse. Aphids were raised in canola cages at temperatures ranging from 15 to 20 °C. Rotenone, an insecticide, was applied to the aphids for two hours. The aphids were fed tainted carrot leaves after a 10-min fast and then sprayed onto healthy carrot plants. Aphids were fed for an hour before being killed. Carrot, five Apiaceae herbs (anise), and two Apiaceae native plants (native parsnip, *D. glochidiatus* and Australian carrot, *D. glochidiatus*) and were found to be *CarVY*-infected (Jones 2005). *Trachymene pilosa* is a species of Trachymene. *Trachymene pilosa* is a species of Trachymene. In the field, infection was found in seven of the 22 host plant species, with significant variation in host plant type and disease severity. In a greenhouse, the severity of symptoms differed substantially across Daucus spp. and other wild ancestors samples fed aggressive green peach aphids. A Polish collection of 21 wild germplamic accessions (seven wild carrots, six *D. muricatus*, two *D. bicolor*, and six unidentified *Daucus* species) and a UK collection of 29 wild germplamic accessions viz., seven wild carrots, two *D. bicolor*, six unidentified Daucus species six and *D. muricatus* were used to obtain systemic *CarVY*-infected plants (27 wild carrots, one *D. littoralis*, one *D. hispidifolius*,). When more lines were introduced to the collection, some were infected many times, indicating infection, while others remained infection-free, indicating *CarVY* resistance. Finally, accessions from Australian field trials were tested for a larger spectrum of symptoms than accessions from greenhouse trials.

8.4.3 Parsnips Yellow Fleck Virus (PYFV)

PYFV resistance in carrots has not been tested. The genetic resistance of the carrot line to viruses such as motley dwarf and *CarVY* is unclear. Molecular screening approaches, as shown by the wide range of symptoms seen in virus-treated carrot lines, may be helpful for discovering viral resistance genes, including QTLs.

Carrot diseases are caused by a variety of mollicutes (phytoplasmas and spiroplasmas) that are restricted to the phloem of the crop. Infections caused by Phytoplasma affect a diverse range of cultivated and wild species, including carrots and over 300 other crops, ornamental crops, and weeds (Blomquist 2002). Leafhoppers are the vectors that carry them. However, despite the fact that phytoplasma losses in carrots are rare, aster yellows have been discovered in all major carrot-producing countries, while BLTVA yellows have only been discovered in the western United States. Yellows derived from the BLTVA Phytoplasma are categorized as subgroup A of the *16SrVI* clover proliferative group, while yellows derived from asters are classified as subgroup B. Yellows derived from asters are designated as subgroup A of the *16SrVI* clover proliferative group. Phytoplasma is considered a member of subgroup I of the *16SrI* clover proliferation group, according to the *16SrI* trefoil proliferation group (Lee et al. 2006) Phytoplasmas produce symptoms related to those of infections. It is potential for leaf veins to become chlorotic, which will ultimately result in the chlorosis of the whole leaf. The leaves of infected plants are much thinner than the leaves of healthy plants. Dormant crown buds give rise to adventurous shoots. Hand gathering is required due to the fragility of golden, crimson, or purple leaves (Blomquist 2002). Infected plants have a short main root and a taproot from which numerous branch roots grow. After bolting, carrot seed harvests develop phyllody (leaf-like petals on blossoms) and virescence (flower greening). BLTVA assists in the relief of pain. Plants infected with Phytoplasma look like aster yellows, but they bloom early and have weak, woody taproots with secondary root development. "Dormant umbels" are ones that lack virescence and phyllody.

Spiroplasma citri was reported in carrot plants in Washington State by Lee et al. (2006). It was observed that the leaves of symptomatic plants had yellowing leaves, purpling, and reddening, as well as the creation of a crown arrangement, shortening of roots and shoots, fibrous secondary root growth, and an abundance of adventitious roots. It was discovered during the carrot harvesting operation in central Washington. Yellow pigments produced from *Synechocystis citri* and *BLTVA* phytoplasma has been isolated from several plant species. Citrus greening is caused by the bacterium *S. citri* in Florida and California. A class of prokaryotes known as Phytoplasmas and Spiroplasmas colonise and reproduce inside the sieve cells of plant phloem (Blomquist 2002). In addition, their leafhopper vectors are flourishing. Given the inability of these obligate organism to be grown on agar, invasion is verified by using a polymerase chain reaction (*PCR*) or an enzyme-linked immunosorbent assay (*ELISA*) using primers specific for Phytoplasma or Spiroplasma. Golden aster yellows are disseminated by the aster leafhopper (*Macrosteles fascifrons*), which is the most common vector of Aster yellows (Boivon 1994; Blomquist 2002). The

beet leafhopper (*Circulifer tennellus*) obtains and spreads Phytoplasma and Staphylococcus citri. Leafhoppers infected with Phytoplasmas and Spiroplasmas propagate the pathogens till they die. Anise aster leafhoppers disseminate aster phytoplasma throughout the Midwest each spring as they migrate from the south on infected weeds and other crops. Aster leafhoppers do not migrate throughout the winter, except in the west and east. During the summer dry season in the western United States, insect leafhoppers gather *BLTVA* yellows Phytoplasma from infected wild plants and disseminate it to irrigated regions. In carrots, Phytoplasmas and *S. citri* do not transmit seed. Female leafhoppers are incapable of infecting their offspring (Blomquist 2002).

In 1982, Gabelman et al. (1994) started breeding carrots to improve resistance to aster yellows. They were able to produce an *AYSYN* breed with four open-pollinated carrot varieties and five lines of inbreds by evaluating 200 accessions in the field. Carrot rows were interspersed with lettuce rows to keep aster leafhoppers away from each four-row bed of carrot lines. Leafhoppers infected with phytoplasma were cultured in a greenhouse in June and July and then spread evenly over the field. In order to estimate infection rates, they performed a search for aster yellows symptoms in October. For pollination, 189 roots from the top 10% of the 200 lines were verbalised and planted in a greenhouse. Five inbred lines and four open pollinated cultivars were developed from the roots of twenty flowering plants using an unknown Russian line F_1 and W33 (Nanco, Scarlet Nantes, Gold King and Royal Chantenay). The *AYSYN* population was established via crossing seed, and inbred lines were isolated using a number of approaches. Using Gabelman et al. (1994) third technique, carrot inbred lines were derived from the Wisconsin carrot breeding programme (WBP). Four WBP roots were combined and inbred over eight generations to generate the inbred W1-1. To develop inbred lines for this population, three ways were used: they were mixed with the population's inbred selections, the population was mixed with high-color inbred lines, and the population was combined with elevated inbred lines. *AYSYN* lines were utilised to generate *AYSYN* hybrids after five generations of inbreeding. Field studies were conducted in 1990, 1991, and 1993 to test the resistance of 26 chosen lines to aster yellows on six commercial carrot cultivars. According to Gabelman et al. (1994) resistant lines showed infection rates ranging from 2.5 to 35.3% per plot, while regular cultivars had infection rates ranging from 12 to 43%. A large number of resistant lines were chosen based on their lower incidence of aster yellows. The least infected plants were 'Scarlet Nantes,' 'Royal Chantenay,' and 'Gold King,' with an infection rate of 15.3% on average. In 33.3% of instances, leafhopper populations were similar across genotypes, demonstrating that resistance had minimal effect on vector feeding. Feeding preferences for carrot genotypes were not detected. Using a synthetic population in conjunction with pre-existing inbred lines seems to have been very beneficial in generating the most successful resistance breeding approach. Inbreeding, according to Gabelman et al. (1994) led in the creation of resistance-causing recessive alleles. The resistance of naturally infected and contaminated crops was tested by exposing them to high selection pressure. The data imply that aster yellow resistance is empirical, based

on morphological heterogeneity and the relevance of various exposures in disease responses.

8.5 Carrot Fly (*Psila Rosae*)

The carrot fly, sometimes known as the carrot rust fly, is a pest of carrots and other Apiaceae crops that may cause severe damage (Hardman and Ellis 1982). Carrot plants are preferred by females for egg laying. Carrot roots are unsaleable due to the damage of carrot fly larva (Ellis 1999). In the vast majority of cases, quality losses exceed yield losses (Dufault and Coaker 1987). It has been shown that antixenosis reduces early fly infestation and leads more to resistance than antibiosis towards larvae in Umbelliferae species; however, it has been found that the opposite is true in carrot cultivars (Degen et al. 1999a). Pesticide-resistant varieties in carrot have been tested (Degen et al. 1999b, c). Several carrot fly resistance trials, according to Ellis et al. (1978) have shown negative findings. As part of their investigation into the efficiency of pesticides against the carrot rust fly, the results revealed that Speed's Norfolk Giant and Royal Chantenay were at different extremities of a susceptibility resistance curve. The damage index, which was computed using root weights and quantities in four damage categories, showed good discriminating between cultivars, even when carrot rust fly infestation was dreadful. Michalik and Wiech (2000) screened carrot varieties and developed five resistant breeding lines. *P. rosae* damage was decreased by half in cultivated carrots with the highest level of resistance. Several Daucus species have been tested for carrot fly resistance, and they may hybridise with cultivated carrots to produce resistant cultivars (Ellis 1999). Ellis and Hardman were among the first to generate resistant F_3 and F_4 carrot cultivars from hybrid *D. capillifolius* (1981). In order to develop new varieties with low resistance to the carrot fly, nine carrot inbred lines were developed in 1991 from a hybrid of two carrot varieties namely Long Chantenay and Sytan (Ellis et al. 1991). Varieties, wild ancestors, putative land rances and wild accessions were used to develop resistance, giving in the partly resistant variety Flyaway, as well as lines with much higher resistance than Sytan (Simlat et al. 2013; Ellis 1999). Identifying the physiological, pharmacological, and genetic factors of carrot fly resistance may aid breeders make better accurate cross selection decisions in their breeding programmes. Guerin et al. (1983) and Städler and Buser (1984) revealed that the chemical composition of the leaf surface is complicated. Carrot leaves, on the other hand, contain a range of oviposition stimulants that are very effective in attracting the carrot fly. According to Städler and Buser (1984), propenylbenzene, coumarins, and polyacetylene are effective antibacterial and antifungal compounds. Several experiments have been carried out in order to get a better knowledge of the processes behind carrot fly resistance. Oviposition, according to one point of view, is undesirable. Guerin and Stadler (1984) looked at how foliar chemostimulants affected this variable in four cultivars. The colour of the leaves, as well as their morphological characteristics, influenced host selection and oviposition. While some plants were resistant to antixenosis, resulting in lower egg

production, the roots were the predominant source of resistance (Guerin and Ryan 1983). Carrot roots containing chlorogenic acid have been linked to an increased risk of carrot fly larval damage (Cole 1985). When this chemical was evaluated in selected lines of 'Sytan,' there was no consistent sign of resistance, showing that this was not resistance chemistry (Ellis 1999). Simlat et al. (2013) found a link between carrot resistance phenotypes and phenolic component concentrations. The expression of *PAL1* and *PAL3* was found to be higher in resistant carrot lines. As a consequence, numerous sources of carrot fly resistance in both wild and cultivated plants have been found. Unlike Ellis (1999), few researches have looked into the genetics of carrot fly resistance. This knowledge might help in the improvement of carrot genotypes that are resistant or partially resistant or immunity to carrot fly.

8.6 Aphids

Aphid saliva may induce plant disease in addition to mechanical harm (Rubatzky et al. 1999). When beetles feed on plant leaves, they produce honeydew. Honeydew is a pleasant substance that creates a protective covering on the photosynthetic surfaces of plants. Furthermore, it spreads viruses that are harmful to *C. moestum*, including as *CMoV* and *CRLV*, which cause the plant to become motley dwarf (Carrot-Willow Aphids). Carrots are a host plant for *M. persicae*, a green peach aphid. The peach green aphid (*M. persicae*) prefers carrots as a host plant. Other popular names for these insects are melons aphids (*Aphis gossypii*), purple pea aphids (*Acyrothosiphon pisum*), bean aphids (*Aphis fabae*), potato aphids (*Macrosteles fascifrons*) and carrot-willow aphids (*Myzus ornatus*),

Because of its vigour and rapid growth, Lamb (1953) hypothesised that Osborne Park, an Australian carrot cultivar, would be resistant to the insect willow aphid. The carrot cultivar 'Autumn King' was aphid resistant in the United Kingdom because to its less severe motley dwarf symptoms as compared to lesser varieties. Dunn (1970) conducted a study on the Autumn King and the tolerance of aphids. Three Australian cultivars, Berlikum, Nantes, Autumn King and Chantenay were investigated for aphatic susceptibility in cages and field testing over a three-year period at different temperatures. Aphid levels, on the other hand, were consistently high throughout the board, with very little reproductive variation. 'Osborne Park' was more susceptible to carrot-willow aphid infection than Lamb (1953). The 'Autumn King,' on the other hand, was especially susceptible. Berlikum is more sensitive to aphids and viruses than Nantes. Dunn (1970) claims that cultivar fertility is not as temperature-dependent as aphid fertility. Antibiosis, as well as preference or non-preference and tolerance, were proposed by Painter (1951) as components of aphid resistance. Dunn (1970) proposed Berlikum in the outdoors. Aphids prefer between 20 and 30% less aggressive aphids than the host. The short cultivar tested, 'Berlikum,' may have signalled aphid escape rather than resistance.

Painter (1951) distinguished three forms of aphid resistance: antibiosis, antixenosis, and tolerance. Prior to the invention of the phrase, only minor genetic

changes in plants and insects occurred. Smith and Chuang (2014) conducted a comprehensive evaluation of the known research on plant aphid defence. They examined the genes and sequencing of aphid-resistant cultivars created for a diverse variety of plant species, as well as their host selection behaviour. They investigated the pathogenicity of aphids as well as the utility of aphid resistance genes in agricultural pest and disease management. This resistance is dominant, although it might also be polygenic, recessive, or partly dominant. Despite this, at least 17 aphid species have been identified as being harmful to plant aphid resistance genes, emphasising the crucial need for the development of novel and diversified sources of protection. Using linkage maps and fluorescence in situ hybridization, researchers discovered viral resistance genes in plants that were aphid and aphid-vectored. Aphid resistance is not bred into carrot varieties.

8.7 Thrips

Damage to carrot leaves and petioles is caused by thrips' rasping mouthparts, which induce silvering and injury (Rubatzky et al. 1999). Carrots may be attacked by thrips such as *Thrips tabaci*, *Frankliniella tritici*, and *Frankliniella occidentalis*. The tomato spotted wilt virus (*TSWV*) is disseminated by the carrot-feeding western flower thrips, which is a vector for the virus. Wild, cultivated, and biofortified carrots enhanced with the antioxidant chlorogenic acid were used in a study by Leiss et al. (2013) to investigate non-specific durable resistance to the western flower thrips (*F. occidentalis*). A total of six commercial carrot varieties (Ingot, Sugarsnax, Nantes, Paris Market, and Chantenay) and four wild accessions (D3, D2, D1 and S1) were tested (four biofortified genotypes (two germplasms with high chlorogenic acid, 309-2 inbred line (purple-yellow) and B7262 inbred line (purple-orange) from the WBP as well as a purple and an orange accession from a seed source). Silvering (feeding damage) severity varies by a factor of two most resistant and sensitive carrot inbred lines. Nuclear magnetic resonance microscopy was used to analyse the three most resistant and sensitive carrots (NMR). According to the investigation, thrips were found on wild carrots. The carrot fly (*P. rosae*), was the most resistant to Ingot. There was no thrips resistance found in biofortified carrots. Three biofortified carrots were found to have thrips. Despite having the leaf area, leaf hair content and same size, the metabolic profiles of susceptible and resistant carrot cultivars differed. The leaves of resistant cultivars contained much more sinapic acid, alanine and luteolin than the leaves of susceptible cultivars. In vitro, these compounds limit thrips growth. The natural variety of these chemicals observed in growing carrots, according to Leiss et al. (2013), may be utilised to boost thrips resistance. The compounds improve the advantages of thrips resistance breeding due to their antioxidant characteristics. More sensitive metabolomics, they reasoned, would signal an increase in the number of host resistance chemicals that may infect them.

8.8 Nematodes

Root knot nematodes that feed on carrots include *Meloidogyne javanica*, *Meloidogyne incognita* and *Meloidogyne chitwoodi*. Crop output and morphological flaws such taproot forking and galling may result in 100% losses, rendering carrots unsaleable roots (Roberts and Mullens 2002). Most common nematode in the temperate areas of the countries is *M. hapla*, while *M. incognita* and *M. javanica* are also common in these zones (Parsons et al. 2015; Bridge and Starr 2007). *M. chitwoodi* and *M. fallax* are less common, although they do cause substantial infestation to carrot taproots. *M. chitwoodi* severely galls the lenticels, resulting in a tenacious taproot (Wesemael and Moens 2008). Soil nematicides, crop rotation, and floods are used to control root knot nematode (RKN). On the other hand, genetic tolerance seems to be the most efficacious and ecologically friendly approach of minimising RKN destruction. Carrot germplasm has a high level of genetic diversity, which is linked to nematode resistance. Yarger and Baker (1981) investigated the susceptibility of 21 cultivars and breeding lines to *M. hapla* in a controlled greenhouse and in the field. Nantes and Long Chantenay rootstocks were resistant in general, although Danvers rootstocks were more vulnerable. Certain cultivars indicate tolerance by parasitizing the roots but not reproducing, whilst others demonstrate tolerance by parasitizing the roots but reproducing (Wang and Goldman 1996).

Using primary root galling on carrot seedlings, Huang et al. (1986) presented a stability study for testing *M. javanica* resistance in the greenhouse. The severity of symptoms was larger in the Nantes and Kuroda groups, indicating that these two worm species have different resistance mechanisms than *M. hapla*. The cultivar Brasilia has a low worm population density because to its resistance to worm penetration, development, and egg production delays (Huang 1986). *M. incognita* race 1 resistance was assessed in 170 Korean carrot lines by Yunhee et al. (2014). As genetic resources for breeders, they have 61 resistant lines accessible. Susceptible root tissues created huge changed cells surrounding the nematodes seven weeks after infection with *M. incognita*, while resistant root tissues developed tiny modified cells (Yunhee et al. 2014). The presence of necrotic layers around altered cells may be caused by the expression of the RKN resistance gene in resistant carrot root tissues. The northern Indian cultivar DR-333 has been shown to be resistant to southern root knot nematode (Siddiqui et al. 2011). When discussing resistance, it is an important to look at the different types of nematodes. There are three races of Columbia root-knot nematode in the United States (Wesemael and Moens 2008). The sensitivity of fifteen carrot cultivars to *M. chitwoodi* varied according to the racial group that infected the seedlings in the experiment (Santo et al. 1988). There are thirteen of fifteen *M. chitwoodi* race 1 cultivars, with quality ranging from medium to excellent. Aside from Orlando Gold, none of the *M. chitwoodi* race 2 hosts were present or performed badly (moderate host). Wesemael and Moens (2008) reported *M. chitwoodi* egg masses in 19 carrot varieties produced in glasshouses. Charchar et al. (2009) identified a novel RKN race capable of parasitizing two important vegetable

crops farmed in Brazil. In the battle against RKN, finding resistant carrot cultivars to include into crop rotations is crucial.

Prior to establishing RKN-resistant carrot cultivars, scientists must conduct resistance genetics research. The species *M. javanica* and *M. incognita* were extensively used in this investigation. According to Huang et al. (1986) *M. javanica* exhibited a high degree of narrow-sense heredity when it came to root distressing and egg mass production. Resistance to *M. incognita* was also shown in field testing using a Brasilia carrot cultivar (*Mj-1*, one or two dominant genes duplicated at a single locus). RAPD markers associated with the *Mj-1* gene that might be used in conjunction with marker-aided selection to generate hybrids resistant to *M. javanica* (Boiteux et al. 2004). According to Boiteux et al. (2004), the *Mj-1* locus dosage has an effect on phenotypic resistance, and the *Mj-1* locus may be a quantitative resistance locus. Ali et al. (2014), for example, discovered a segregating population resistant to *M. javanica* and *M. incognita*, which they attribute to *Mj-2*, a single dominant gene on the same chromosome as *Mj-1*. Using three segregated populations, Parsons et al. (2015) identified five *M. incognita* resistance QTLs. QTLs have been discovered on carrot chromosomes 1, 2, 4, 8, and 9. *Mj-1* is a chromosome 8 quantitative trait locus (QTL) that is shared by all three populations. The cross of three resistance sources from Europe, South America, and Syria resulted in two carrot populations with broad-sense heritabilities of 0.33 and 0.25 against *M. incognita* (Parsons et al. 2015). In *M. hapla*, Wang and Goldman (1996) found two homozygous recessive resistance genes. Nematode infection, on the other hand, has been linked to quantitative and qualitative resistance. According to Yunhee et al., resistance to *M. incognita* is controlled by a single or a few genes. A commercial variety with *Meloidogyne* resistance genes was found in a populations generated from the resistant variety Brasilia (Vieira et al. 2003). Brasilia germplasm remains one of the most promising sources of RKN-resistant carrots on a long-term, broad-spectrum basis (Vieira et al. 2003). The BRS Planalto cultivar was developed by Embrapa Vegetables in Brazil in 2009 to be resistant to RKN (Pinheiro et al. 2011). Standard RKN resistance breeding strategies, according to Ali et al. (2014), required labor-intensive greenhouse and field phenotyping experiments. Certain kinds of nematode resistance may be produced by using RNA interference (RNAi) to target and silence nematode genes in host plants that produce *dsRNA* and *siRNA* (Roderick et al. 2018). *Pratylenchus thornei* and *Pratylenchus zeae* were subjected to dsRNA treatment in order to inhibit the expression of two genes that are important in structural stability and muscle function, respectively (Tan et al. 2013).

Singh et al. (2019) reported RKN, *Meloidogyne spp*., in carrot genotypes in vitro. To examine carrot genotypes for RKN resistance, we inserted about 20 larvae J_2 of *M. incognita* per root tip onto pluronic gel media. The larvae pierced the roots in large numbers, with 12.5 larvae per root in black carrot Pusa Asita and 1.0 J2s per root in '6526B Sun2000'. *Mj-1* resistant carrot lines ("6526 B Sun2000" and "8542B Vilmorin") have been shown to confer RKN resistance to susceptible carrot cultivars. RKN resistance breeding will be aided by the STS-SQ1 marker and in vitro screening.

8.9 Minor Pests

Pest insects and mites attack on carrot roots and leaves, preventing seed germination and root growth (Rubatzky et al. 1999). Pests include carrot leaf miners (*Lisonotus latiusculas*, *Napomyza carotae*), leafhoppers (aster and beet leafhoppers), carrot psyllids (*Trioza apicalis*), red spider mites (*Tetranychus urticae*) and carrot weevils (*L. latiusculas* and *Lisonotus oregonensis*). With the exception of anecdotal data from breeders and producers, nothing is known about the resistant origins and genetic paths of the majority of these pests. In carrot pests that transmit viruses, phytoplasmas, and spiroplasmas, it is difficult to separate vector resistance from pathogen resistance. Lygus bugs, a microscopic root crop pest, have the ability to devastate seed yield. Insects favour seed and blossom development, causing carrot seed embryos to die and become non-viable. Scott (1970) reports that 'Nantes,' 'Imperator,' and 'Royal Chantenay' have variable degrees of resistance to lygus bug feeding. The purpose of this research was to determine how immune lygus bugs are to insect attack on flowering carrot inflorescences. In Idaho, Scott (1977) used a similar technique in order to choose for lygus insect resistance. In none of his studies, Scott (1970, 1977) seems to have tested umbels for lygus insect damage to developing seeds. It's conceivable that the insects didn't die because of pest resistance-related dietary changes. For a number of reasons, comparing cultivar sensitivity to lygus bugs proved difficult. Certain carrot infloresences may be deficient in lygus insect feeding, impairing seed development and expansion. He reported that the mortality of lygus insects varied across cultivars and among cultivars. The persistent impact of lygus insect losses on several aspects of carrot seed production raises doubt on the findings.

A diversified feeding approach, according to Kainulainen et al., promotes the acceptability of sucking insect oviposition. *T. anthrisci*, an Apiaceae psyllid, was investigated in the laboratory, greenhouse, and field. The lygus was dissected. In Northern Europe, these pests induce root stunting and leaf bending. On the other hand, Lygus bugs bite off salmon seeds to feed the growing egg, resulting in seeds that are unsustainable (Scott 1977). Leaf oil was present in variable amounts in Nantes 3 Express, Splendid, Panther, Napoli, Nantura, Parano, and Flakkeer 2 (Kainulainen et al.). Egg production, on the other hand, varies greatly across varieties. Despite their proximity to the hosts, females on Nantes Express 3 lay more eggs than Panther. The fragrance test found no indication of this preference, suggesting that physical touch is more essential than usage in the selection of lygus bug hosts. The egg-laying choice of the insect lygus was shown to be unrelated to essential oil concentration in the research. Cauliflower psyllids are drawn to high concentrations of limonene oil. This carrot psyllid was particularly fond of sabinene. Previous study indicates that the carrot psyllid favours plants with high-pinene-sabinene concentrations (Valterova et al. 1997; Nehlin et al. 1996). *T. anthrisci*, an Apiaceae psyllid, was shown to have a positive relationship between egg number and myrcene. It is a chervil scavenger, a European plant (*Anthriscus sylvestris*). According to this study, compounds in the psyllid diet, but not carrot leaves or essential oils, may influence egg-laying behaviour. According to our findings, the essential oil content of carrot cultivars

seems to be more important for *T. anthrisci* than for lygus bugs. Psyllid resistance may be enhanced in cultivars with a high limonene concentration.

8.10 Conclusion

This chapter extensively discusses the disease and insect pest resistance and susceptibility of carrot germplasm. Cercospora leaf spot and powdery mildew both exhibit monogenic resistance (Bonnet 1983a, b; Angell and Gabelman 1968). Carrot leaf blight is the most prevalent disease worldwide. Numerous studies have examined the genetics of resistance, with two identifying three and eleven QTL, respectively (Le Clerc et al. 2015a, b; Le Clerc et al. 2009). Due to the critical nature of ALB resistance, breeders are searching for markers that may be used to select for it. Root knots, or RKN, wreak havoc on carrot roots worldwide. Three RKN species are resistant genetically. Genetic resistance to *Meloidogyne hapla* is determined by two genes (Wang and Goldman 1996). On chromosome 8, a single dominant gene is involved for conferring *Mj-1* resistance. The resistance gene was selected using marker-assisted selection (Boiteux et al. 2000, 2004). *Mj-2* is also present on chromosome 8, conferring further resistance on M. javanica (Ali et al. 2014). *Mj-1*, when paired with six additional QTL on chromosomes 1, 2, 4, and 9, provides resistance to *M. incognita* (Parsons et al. 2015), a prevalent RKN species found worldwide in temperate carrot-growing areas (Parsons et al. 2015). Selective markers for *Mj-1* have been found (Boiteux et al. 2004). Numerous biotic stressors have been systematically investigated for potential phenotypic resistance, candidate genes identified, and resistance introduced into commercial cultivars. Others are unaware of potential resistance sources and lack screening tools for phenotypic resistance. The scope of this research should be widened to include a broad spectrum of other carrot diseases and pests found in regional and worldwide regions. Resistance to biotic stresses has significantly benefited in the reduction of disease and insect pests when accompanied with biological, chemical and cultural management strategies (Ben-Noon et al. 2003). Because there are no interspecific barriers between wild and cultivated carrot species, resistance genes may be transferred more easily between them. The identification of resistance genes and the breeding of resistant crops have been aided by molecular markers and other technologies (Stein and Nothnagel 1995). A proprietary array of three hundred microsatellite markers was used along with a distributed, in depth coverage carrot nuclear genome library that had > 17X coverage, as reported by (Cavagnaro et al. 2009, 2011). It was discovered that the carrot nuclear genome has a structure after a recent analysis of BAC-end sequences totaling 1.74 Mb. Iorizzo et al. estimate that it accounts for *90% of the anticipated carrot genome (Iorizzo et al. 2016). Researchers will be able to identify genes associated with biotic and abiotic stress, as well as other critical characteristics. Wang et al. (2018) decoded the sequencing of the carrot cultivar 'Kurodagosun,' which was previously uncharacterized (473 Mb). These genetic resources will benefit basic and applied carrot research,

particularly in the area of insect pest and disease resistance development. Klimek-Chodacka et al. (2018) established the very first successful site-directed mutagenesis system utilising the carrot genome, opening the possibility for disease and insect resistance. Resistance to insect pests and diseases is essential for all aspects of seed formation, carrot root growth, storage, nutritional quality, flavour, and processing.

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