Seed-associated Fungal Pathogens and their Management in Safflower

Dr. N. A. Musmade[1] Dr. P. B. Khaire[2] Dr. S. D. Magar[3], Dr. S. V. Kolase[4] and Dr. D. H. Sarnobat[5]

1 Seed Technology Research Unit, Mahatma Phule Krishi Vidyapeeth Rahuri 413 722 (Dist. Ahmednagar) (Maharashtra State)

2 Department of Plant Pathology and Microbiology, Mahatma Phule Krishi Vidyapeeth Rahuri 413 722 (Dist. Ahmednagar) (Maharashtra State)

3. Department of Horticulture, Mahatma Phule Krishi Vidyapeeth Rahuri 413 722 (Dist. Ahmednagar) (Maharashtra State)

Email: musmadenarayan@gmail.com; Mo (9420452667)

---

1. Introduction

Safflower is a cultivated annual oilseed crop that belongs to the tribe *Cardueae* (thistles), family *Asteraceae* (Compositae) and subtribe *Centaureinae* (Berville *et al*., 2005). The *Asteraceae* family, which includes annual herbs and woody shrubs, is acknowledged as the biggest group of flowering plants. It has more than 1500 genera and 22,000 species. There are numerous more names for safflower, including kusum, kasunmba, kusumbo, kusubi, kabri, ma, sufir, kar/karar, sendurgam, agnisikha, hebu, su, and suban. In addition to affore, asfiore, asfrole, astifore, asfiori, zaffrole or zaffrone, saffiore, and finally, safflower, it is also known as hung-hua or "red flower" in the People's Republic of China (hereinafter referred to as "China"), as well as by numerous other names throughout the world as compiled by Smith (1996).

Safflower is one of the oldest crops grown by humans, yet it is still a small crop in comparison to other oilseeds (FAOSTAT, 2019). Vegetable oil is now the primary reason that safflower is grown (Kumar *et al*., 2015). According to Kumar and Kumari (2011), safflower is an annual plant that is upright, herbaceous, densely branched, prickly, and thistle-like and can reach heights of 30 to 150 cm. Safflower seedlings develop a rosette and spend a number of weeks in this vegetative state, during which time leaves and a substantial taproot system appear. Due to its extensive taproot system and numerous thin horizontal roots, this plant is able to access deeper soil strata for water and nutrients than a number of agricultural plants (GRDC, 2017). After the rosette stage, the stem quickly lengthens, there is significant branching, and then there is flowering with leaves grouped on both sides of the stem (Singh and Nimbkar, 2006). Typically, farmed safflower has beautiful orange flowers. The size of a leaf varies depending on the variety and where it is located on the plant, but average leaves are 2.5–5 cm wide and 10-15 cm long. According to Teotia *et al*. (2017), the ovate-lanceolate, alternating, sessile leaf shape. Lower leaves typically lack spines, but those higher up the stem frequently grow stiff spines. While these spines make the crop challenging to traverse, they serve as a barrier to larger animals like pigs and kangaroos (GRDC, 2010). Growing older makes plants stiffer and more resistant to environmental stressors like wind and hail.

According to Pearl *et al*. (2014), the domestication of the ancient crop known as safflower dates back to the Fertile Crescent around 4,000 years ago. According to Chapman *et al.* (2010), this area stretches from western Iraq to southern Israel. Safflower has been grown for many years in northern Africa, China, and India.

Throughout spite of the fact that safflower is a dryland oilseed crop, it was once planted throughout the Mediterranean, southern and central Asia, and the Middle East to extract colours for food and textiles (Zohary et al., 2012; Li and Mundel, 1996). Nowadays, safflower is produced in semi-arid and arid anywhere plants can tolerate heat and dryness. A number of safflower cultivars is depicted in Figure 1.

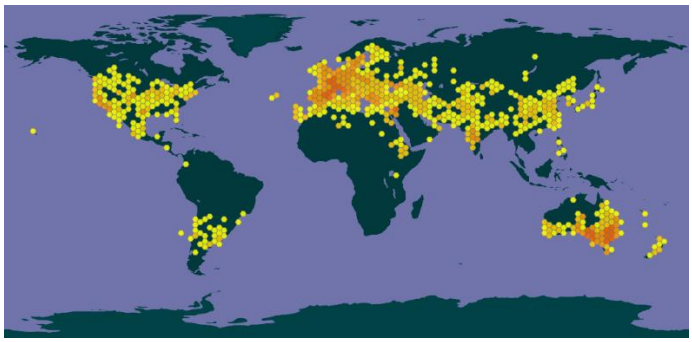

Figure 1: The cultivated safflower (*Carthamus tinctorius* L.) is found across the world. Georeferenced occurrences are indicated by yellow (or light grey) dots.
Source: GBIF Backbone Taxonomy (2023).

1.1 Production – productivity

1.2 Uses of safflower

Safflower is a long-cultivated crop with several applications. It is a versatile crop grown for a variety of reasons, including its high-quality oil, medical advantages, feed value, or orange-red dye that is derived from the petals (Dwivedi, 2005). Before more affordable aniline dyes became available, safflower was traditionally produced for its seeds, for food colouring, as a medicinal, and for creating red and yellow colours.

1.1.1 Food Uses

Safflower oil is made from safflower seeds, and because it contains some saturated fatty acids than olive, canola, and sunflower oils (Dajue and Mundel, 1996), it is frequently seen as a healthier alternative. According to Arslan *et al*. (2003), safflower oil with a higher concentration of linoleic acid contains tocopherols, which are known to have antioxidant properties and contain a lot of vitamin E. Because of its significant value for lowering cholesterol, safflower oil is included in the diets of people with cardiovascular disorders (Pongracze *et al.*, 1995). Safflower has traditionally been farmed in India for its high-quality edible oil, which is having polyunsaturated fatty acids, and the orange-red dye that is derived from its beautiful florets.

1.1.2 Livestock feed

Safflower is beneficial for both human and animal consumption, as well as storage as hay or silage (Bar-Tal *et al*., 2008). The yields of its forage are comparable to or better than those of oats and lucerne, and it is palatable with feed value (Smith, 1996; Wichman, 1996). A valuable source of animal feed is safflower oil cake. Safflower meal has a significant amount of fibre and roughly 24% protein. According to a recent analysis (Chidoh, 2012), values of 21.8% protein, 67.4% nitrogen detergent fibre (NDF), 38.5% acid detergent fibre (ADF), 15–20% acid detergent lignin, and 3.3% ash are appropriate for cattle feed or supplements.

1.1.3 Textile industry

Natural colours are made by extracting them from dried flower petals. Plant-based natural dyes are growing popularity nowdays due to their naturalness and current fashion trends. Carthamin, a benzoquinone-based pigment, is what gives safflower its colour (Garcia, 2009). In Eastern Europe and the Indian subcontinent, the carpet weaving business uses the insoluble red dye (carthamine) and the water-soluble yellow dye (carthamidin) (Weiss, 1983).

1.1.4 The cut flower business

Western Europe, Japan, Latin America, and Kenya are among the regions where spineless varieties of safflower grow for the domestic and export markets (Kizil et al., 2008; Emongor, 2010).

1.1.5 Medical and Clinical Applications

Safflower is also employed medicinally. According to More *et al*. (2005), safflower seeds can be utilised to cure urinary calculi. Safflower petals are said to be effective in cure large nu,mber of chronic illness such as hypertension, coronary heart disease, rheumatism, male and female fertility issues, and hypertension. Since safflower oil has no allergic reactions, it is appropriate for use in injectable treatments (Smith, 1996).

2. Seed-associated pathogens of safflower

Pathogen cause	Disease	Yield losses	References
Fungi			
Alternaria carthami	*Alternaria* leaf spot	25–60%	Borkar and Shinde (1989); Awadhiya (1991); Singh and Prasad (2005); Prasad *et al*., (2008)
Fusarium oxysporum f. sp. *carthami*	Fusarium wilt	40–80%	Awadhiya (1991); Kalpana Sastry (1996); Kalpana Sastry and Chattopadhyay (2003); Rao *et al*., (2014)
A. nigar, A. flavus, Chaetomium sps., *Rhizopus* sps *Curvularia*sps, *Macrophomina phaseolina* and *Fusarium* sps	Fusarium wilt, Macrophomina root rot, Leaf spot	40–80%, 1%–10%	Padaganur and Anil kumar (1976); Raghuwanshi *et al*., (2002); Rajendraprasad *et al*, (2021)
M. phaseolina	Macrophomina root rot	1%–10%	Awadhiya (1991); Prasad and Suresh (2012), Rajeswari *et al*., (2012)
Bacteria			
Pseudomonas syringae	Bacterial Leaf Spot and Stem Blight		Jacobs (1982)
Viruses			
Cucumber Mosaic Virus (CMV)	Cucumber Mosaic Disease		Klisiewiez (1965), Tomas (1981); Milosevic *et al* (2020)

2.1 Seed-Associated Mycoflora of Safflower

Safflower seed mycoflora was researched by Padaganur and Anil Kumar in 1976, who observed *Curvularia* sp., *Alternaria* sp., *A. flavus*, *A. niger*, and *Fusarium* sp. on several seed lots of two kinds. *A. carthami* was isolated from surface-sterilised safflower seeds by Rajagopalan and Shanmugam in 1983. They noted that the infection is outwardly seed borne and rarely transported within. *Along with Alternaria alternata, Alternaria carthami was discovered as well in the safflower pericarp (Zazzerini et al. 1985).* Prasad (1985) used the standard blotter method to evaluate 35 different safflower types for the presence of *A. carthami*. A 4-42 percent connection with the fungus, which caused prior and after-emergence seedling death, was found in 27 cultivars. Raghuwanshi et al., (2002) examined seed fungi of safflower varieties and noticed that the seed germination and vigour had been adversely affected by Alternaria, Fusarium, Aspergillus sp. in varieties viz., A-1, Manjira, APRR-3, CO-1, Bhima, HUS-305, A-300, S-144, K-1, JSF-1, NRS-209 and Gima. Singh et al. (1987) studied seed mycoflora corresponding to 13 varieties of safflower and reported 11 species of fungi attributed to the seeds. *Rhizoctonia* spp. and *Alternaria* spp. were observed to be prevalent among the species discovered, with occurrence rates of 40% and 30%, respectively. According to Borkar and Shinde (1989), *A. carthamii* in safflower, which is externally seed-borne, not only causes seed rot, which lowers seed quality but also seedling degradation and pre- and post-emergence mortality of seedlings. The findings showed that 48 to 100% of safflower seeds had externally transmitted *A. carthami* infection. *A. carthami* was identified to be prevalent (100%) in Awadhiya's study of the seed mycoflora linked with fifty types of safflower in 1991. She also noted the presence of *Fusarium* and *Macrophomina* sp. using the component plating technique, Prasad *et al.* (2008) investigated the seed-borne nature of *A. carthami* in safflower. *A. carthami* infection was highest in the seed coat (76.6%), endosperm (38.3%), and embryo (20.4%). *A. carthami*, *A. alternata*, *M. phaseolina*, *F. oxysporum*, *A. flavus*, *A. niger*, *Curvularia lunata*, and *Rhizopus* sp. were all noted as being present.

3. Measurement of diseases

3.1 Fusarium wilt and leaf spot disease severity scale.

***Fusarium* wilt**		**Leaf spot diseases**	
Disease scale	**Leaf/Stem symptoms**	**Disease scale**	**Leaf symptoms**
0	No yellowing/stem discolouration	0	No discolouration
1	Yellowing of leaf with slight necrosis (10–20%)/reddish-brown discolouration of the stem	1	Small irregular spots covering <10% of leaf area
2	Leaf with >50% necrotic areas/stem decay and stunted plant.	2	Lesions coalesce to form larger spots with 50% of the plant infected with the disease
3	Leaf/plant dead	3	Lesions are observed up to bracts with infected seeds

* On the basis of symptoms in the leaves and lower stem vascular discolouration, a disease rating methodology for wilt (0–3) was developed (adapted from Thies, 2000).
*Leaf spot malady scale of assessment adapted from Gud *et al*. 2008.

3.2 Scale for assessing the severity of Macrophomina disease.

***Macrophomina* disease**			
Disease scale	**Leaf/Stem symptoms**	**Disease scale**	**Leaf symptoms**
0	No symptoms on the leaf	7	Inconsistent brown patches having circular rings are formed when lesions combine. Occupying 26–50% of the leaf's surface. Both the stem and petioles have the same lesion.
1	Small, irregular brown spots covering 1 per cent or less of the leaf area.	9	Eventually, lesions will create uneven, dark brown spots with ring-like structures that will cover at least 51% of the leaf surface. Petioles and the stem have lesions.
3	1 to 10% of the leaf's surface is covered in tiny, erratic brown dots with rings that are concentric		
5	Expanding, erratic, brown lesions with concentric circles that encompass 11–25% of the total leaf surface.		

*Standard 0-9 grade disease ratting scale (Mayee and Datar, 1986)

4. Important seed-borne diseases

4. 1 Leaf spot/ blight disease

4.1. 1 Geographical Distribution, Economic Importance, and Losses

The Alternaria leaf blight (ALB) on safflowers was initially identified in India by Chowdhury (1944). Kenya, Argentina, Portugal, Australia, Israel, Italy, Pakistan, Russia, Spain, Tanzania, the US, Ethiopia and Zambia are safflower-growing nations that have all recently reported occurrences of the illness. According to Bergman and Jacobsen (2005), the northern part of the Great Plains territory of the US, as well as the Indian regions of Bihar and Madhya Pradesh, are all thought to be facing particularly severe malady-related effects. There have been reports of serious disease harm to experimental safflower crops from Kenya and Tanzania in East Africa. During flowering but before maturity, a week of humid conditions can reduce safflower yields by 50% to 90% in cultivars that are especially sensitive to the disease. There is a considerable negative correlation between the severity of the disease and yield (Chattopadhyay 2001). Seeds from affected plants become discoloured, contain less oil, and have a noticeable increase in the quantity of free fatty acids, all of which are detrimental to seed germination.

4.1.2 Symptomatology

According to Chowdhury (1944), *A. carthami* originally generated little brown patches with concentric rings that eventually grew larger and consolidated. Additionally, he noted markings on the stem and petiole. A. carthami was reported to have developed an unevenly shaped small, dispersed leaf spot, a turned yellow halo lacking resemblance of a target board, and a light brown dot in the centre of the spot by Krishna Prasad and Basuchaudhary (1989). *A. carthami* previously caused similar symptoms in safflower, which were described by numerous researchers (Prabhakar *et al*., 2012; Taware *et al*., 2014 and Gholve *et al.,* 2015). When seedlings were 30 days old, tiny, solitary, light brown to deep brown round spots (1-2 mm dia.) first appeared on the bottom foliage before moving to the upper leaves. These patches grew larger and merged into larger spots and/or leaf blight as the virus spread. In rare instances, a brown dot encircled by numerous dark, alternating concentric rings also emerged in the core of these areas. Shot holes typically emerged in the diseased area in mature locations.

Under field conditions, the disease often began to manifest in November and reached its peak prevalence by the middle of February. In the sensitive safflower kinds, the disease propagated quickly; as a result, plants that were harshly infected became dark and dried out without producing any seeds. Other varieties of safflower displayed signs of the disease on both the top and bottom leaves, which encompassed the stem's underside. In some instances, the symptoms also manifested themselves as elongated, dark brown to black discolourations on the stem, which led to the spliting of the infected stems. On floral portions, the infected capitula stayed closed, shrivelled, and dried out (Wagh *et al*., 2020). The condition manifested as small, deep brown patches that initially emerged at the bottom of the involucral bracteoles before expanding and reaching different parts of the capitulum (Plate 1).

4.1.3 Pathogen- *Alternaria carthami*

The mycelium has small constrictions at the septa and is septate both intracellularly and between cells. When young, it has a subhyaline colour, but as it ages, it takes on a black hue. The conidiophores may show up alone or together via the stomata or epidermis. They are dense, erect, rigid, free of branches, septate, straight or flexuous, rarely geniculate, brown or olivaceous brown, and paler near the apex. The conidiophores are 15–85 min long and 6–10 min wide, with the base occasionally enlarged. The conidia are solitary or found in extremely short chains and are carried on conidiophores. They have a large beak and are smooth, straight or curved, obclavate, light brown and translucent in colour. Constrictions at the septa of the conidia can occasionally be seen. They are 12-28 millimetres wide and 36-171 millimetres long (with a beak) and 36-99 millimetres long (without a beak). The spores have up to 7 longitudinal or oblique septa and 3-11 transverse septa. The spores' beaks range in length from 25 to 160 m, are 4-6 m thick at the base, tapering to 2-3 m, and have up to 5 transverse septa. Near the base, the beak turns light brown, becoming practically hyaline at the tip. It's possible to view some spores without beaks. In culture, conidial beaks can produce chlamydospores. The ideal temperature range for the fungus to grow is between 25°C and 30°C. Additionally, it can endure a wide pH range; however, development is greatest around pH 6.0 (Choudhary, 1944). The provisional mounts constructed from Alternaria-affected safflower foliage tissues and pure A. carthami culture included olivaceous brown-colored septate mycelium.

4.1.4 Epidemiology Role and Disease Cycle

According to Prasad *et al*. (2009) and Gayathri and Madhuri (2014), the disease can persist through seeds as well as alive conidia of *A. carthami* on detritus on naturally infected sensitive safflower cultivars. With the aid of relatively easy procedures, *A. carthami* is easily separated from seeds. The most reliable method for determining the presence of A. carthami in seeds appears to be the isolation procedures used in alongside seed planting to evaluate seedling health (Awadhiya 2000). The primary infection is seen in the seeds collected from infected plants. On the leaf margin, the infectious agent infects the spines (Borkar 1997). Each spine's apex needs to have an entrance with a diameter of 120 m for infection to pass through. By altering the location of the spine on the outer edge of the leaf and the connection among the positions of the spines on the leaf margin, it is possible to assess the amount of infection by measuring the diameter of the holes at the spine apex. Because spores are released on lesions that emerge on plants grown by contaminated seeds, the disease harms the crop all during the growing season. Brefeldin A (BFA) and 7-dehydrobrefeldin A (7-oxo-BFA), two macrolide antibiotics from A. carthami, have been identified as phytotoxins and pathogenicity factors; the toxins are known to obstruct endoplasmic reticulum-Golgi

flow and also inhibit processing (Kneusel 1994; Driouich et al. 1997). Rains, elevated relative humidity above 80%, irrigated temperatures around 21°C and 32°C, severe fog or regular showers, cyclonic storms, particularly during the seedling and grain production stages, and rain are all risk factors for the disease (Sastry and Chattopadhyay 2005; Murumkar *et al*. 2008a). In the sensitive safflower cv. Manjira, the incubation duration was altered. Based on the incubation time (day from the first symptom's expression) (Wagh *et al*., 2020).

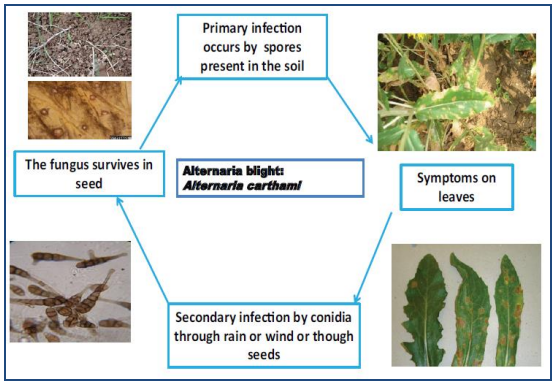

Fig 1. Disease cycle of ALB disease

4.1.5 Management

4.1.5.1 Cultural practices

Utilising seeds that are without disease will inhibit the disease from spreading. Rather than from areas with irrigation, these seeds may originate from previously planted dry land crops. Fungicides may also be utilised to treat the diseased seeds, as was previously described. Rotating crops and maintaining strict cleanliness standards for agricultural detritus are efficient ways to control the disease. By adding KCl to the soil at a rate of 67 kg/ha, safflower seed production is improved and disease severity is significantly decreased (Chattopadhyay 2001). This practice can be paired with the spraying of effective fungicides and the use of the right planting dates for improved disease management.

4.1.5.2 Fungicides

Treating seeds with difenoconazole + mefenoxam or mefenoxam + thiram can lessen the primary cause (Jacobsen et al. 2008). To avoid secondary infection, treat the crop using any aerial fungicide, like fosetyl at 0.1% (Bramhankar et al. 2001), difenoconazole at 0.5%, or AAF (carbendazim 12% + mancozeb 63%) at 0.2% (Sumitha and Nimbkar 2009). For efficient and cost-effective disease management, carbendazim at a concentration of 0.1% should be applied as soon as the disease manifests (typically at the rosette stage, or 25 days after sowing), followed by need-based second and third applications at intervals of 15 days and during the flowering and seed-setting stages, accordingly (Murumkar *et al*. 2008a, 2009a). The combination seed treatment and foliar spray of *T. viride* ST @ 10 g/kg + Garlic clove extract ST @ 10 ml/kg + Hexaconazole FS @ 0.1% resulted in the lowest average disease intensity (14.34%) and the largest decrease (74.36%) (Wagh *et al*, 2020). Treatment with *Trichoderma harzianum* Th4d sc at 2 ml/kg resulted in low disease incidence and severity measurements of 41.66% in *Fusarium* sp, 08.33% in *Rhizoctonia* sp, 06.66% in *Phytophthora* sp, 04.33% in *Alternaria* leaf spot, and 03.33% in *Cercospora* leaf spot (Pawar *et al*., 2013). The highest percentage of seedling vigour was found in Thirum + Mancozeb (16%), and there was no seedling mortality found in 24 or 48 hr against *A. carthami* (Gholve *et al.*, 2017).

4.1.5.3 Bioagents

After 9 days of incubation, the *Trichoderma viride* was particularly efficient against *A. carthami* and *F. oxysporium* f.sp. *carthami* (Shinde and Hallale, 2013).

The *T. harzianum* was discovered to be the best efficient, and it considerably inhibited *A. carthami* and *A. alternata* mycelial growth by about 81.48% and 83.70%, respectively (Zanjare *et al*., 2020).

4.1.5.4 Botanicals

The antifungal effects of eucalyptus, nerium, onion, garlic, lantana, datura, neem, and ocimum species extracts against A. carthami have been demonstrated, and they can be further

utilised for efficient disease treatment (Shinde et al. 2008, Ranaware et al. 2010, Taware et al. 2014). At both 15% and 25% concentrations, a combination of neem, chilli, garlic, eucalyptus, and menthol extracts was identified to be extremely efficient against pathogens; at the latter concentration, mycelial inhibition was found at 62.16% (Upadhyay *et al*., 2019).

4.1.5.5 Resistance sources

A variety of safflower cultivars respond to *A. carthami* infection in different ways (Munoz-Valenzuela *et al*. 2007, Thomas *et al*. 2008). Some genotypes such as EC 32012, NS 133, CTS-7218, HUS 524, and CTV 251 (Desai 1998); GMV 1175, GMV-1199, and GMV-1585 (Indi *et al*. 2004); GMV-5097, GMV-5133, and GMV-7017 (Murumkar *et al*. 2009a); and Ellite Line 21-33. The most intriguing genotypes to be employed in the programme of breeding for incorporating resistance to the Safflower genotypes that vary from partially spiny to non-spiny have been found to display different levels of tolerance to A. carthami infection indicating an elevated level of tolerance to A. carthami under excessive disease pressure (Pawar *et al*. 2013).

High yield and elevated levels of disease tolerance are compatible (Mundel and Chang 2003; Harish Babu *et al*. 2005). According to laboratory and field testing, four wild *C. Carthamus* species, palaestinus, *C. lanatus*, *C. creticus*, and *C. turkestanicus*—are resistant to Alternaria leaf spot. The crossings between *C. tinctorius*, *C. creticus*, *C. oxyacantha*, *C. tinctorius*, *C. turkestanicus*, *C. tinctorius*, *C. lanatus*, *C. palaestinus*, and *C. oxyacantha* resulted in twenty-four F1s. *A. carthami* infection (immunity) has not been found in *C. tinctorius* after screening. In order to molecularly label the resistant genes for marker-assisted field selections for ALB resistance, these disease-resistant lines would be used as the starting point for disease-resistance breeding (Prasad and Anjani 2008a). According to reports, some safflower genotypes have homozygous recessive defence against A. carthami in their seedlings. Still, resistance in adult plants is governed by two identical loci, with a minimum of one of these loci providing adult plant resistance. Safflower can now be bred to be resistant to Alternaria blight by using transgenic safflower plants, thanks to the discovery of plants that are resistant to A. carthami through organogenesis, somatic embryogenesis, and molecular breeding. The cloned esterase gene that degrades the BFA (phytotoxin and pathogenic factor) provides the foundation for the creation of transgenic safflower plants (Kneusel et al. 1994).

Table 2: Responses of safflower cultivars and germplasm lines to the ALB disease

Cultivar/germplasm line	Reaction	References
HUS-305, DSH-242 (IHT), A-1	Tolerant	Wagh *et al*, 2020
PBNS-125, SSF-1109, PBNS-124, DSI-116, DSI-114, SSF-1201, ASF-1302, AKS-326, NARI-95, DSH-250, NARI-H-15, DSH-249, PBNS-12 and PBNS-120	Susceptible	
JSI-120, NARI-198, PBNS-12, DSI-118, AKS/GMU-4576, JSI-118, SSF-1102, DSI-117, NARI-97, ASF-1301, JSI-119, PBNS-123, SSF-1215, NARI-96, AKS-327, DSI-115, PBNS-122, DSI-113 and JSI-117	Highly susceptible	
Nari-P-26, W-521-3, Nari-P-22, Nari-P-25, GMU-7396, Nari-P-27, Nari-P-24, SAF-15-21, Nari-P-21, DSI-116,Nari-P-23, GMU-3705/6, GMU-3705/6 (12)	Tolerant	Pawar *et al*, 2017
Nari-P-28, SAF-15-07 (02)	Susceptible	
GMU-3705, DSI-108 (02)	Highly susceptible	

4.2 *Fusarium* Wilt

4.2.1 Economic Importance, geographical distribution and Losses

Safflower fusarium wilt was first noted in India in 1975 (Singh *et al*. 1975) and the Sacramento Valley of California, USA, in 1962 (Klisiewiez and Houston 1962). Egypt has also been reported to have the disease (Zayed *et al*. 1980). In all of India's safflower-growing regions, it is now recognised as the most dangerous disease (Murumkar and Deshpande 2009). When infected seed is sowed, losses in crops in the stand may result because the affected plants hardly survive past the stage of seedlings. Reports state that the prevalence of disease is between 10% and 20% in most areas, and up to 50% in a select few. Yield losses from sensitive types may be 100% if they are grown in fields that have already experienced severe Fusarium wilt (Sastry and Chattopadhyay,

2005). It poses an important risk to India's sunflower crop, destroying up to 25% of the plants and severely reducing productivity in the Gangetic Valley. According to reports, infected safflower seeds in storage are producing enough fusarium mycotoxins, including diacetoxyscirpenol, T-2 toxin, and 12,13-epoxytrichothecene, to be able to cause mycotoxicosis.

4.2.2 Symptomatology

The condition shows symptoms at every stage of development. Cotyledonary leaves may develop little dark spots, either randomly or in a ring, on the inner surface of the leaf during the seedling stage. They may also become shrivelled, and brittle, and occasionally tend to roll and curve. When the seeds germinate, the seedlings that survived the fungus attack regain their health during the first phases of blooming and recur as a disease. The symptoms start to become very obvious when plants are approximately 15 cm and 20 cm in height and in the sixth to tenth leaf stage. Knowing four essential characteristics of the symptoms at this phase may help identify the disease. This includes unilateral infection on leaves and branches, epinasty, and golden-yellow foliage browning that occurs followed by death. Another instance is vascular browning that appears solely on a single side of the roots and stems of plants that have unilateral top symptoms. One symptom after another emerges quickly. The degree of reddish-brown vascular darkening on the petiole flesh, stem, and roots of plants infected varies greatly based on the circumstances, degree of infection, and reaction of the variety. When a plant is more developed, the disease can only start to harm the lower branches on a single side of the plant, leaving the other parts of the plant intact. These kinds of plants may partially recover between bud development and early flowering, though the symptoms may subsequently reappear. Plants that were severely afflicted generate little, partially opened flower heads. Many ovaries are incapable of producing seeds, or when they do, they may be black, small, malformed, chaffy, or abortive.

4.2.3 Pathogen-*Fusarium oxysporum*

F. oxysporum Schlecht, f. sp. *carthami* Klisiewicz, and Houston are the pathogen. On potato dextrose agar (PDA), the fungus is easily isolated from damaged plant sections. Mycelium can range from being sparse to being numerous, branching, and septate. It is typically white with a purple tint or a delicate pink colour. Microconidia are plentiful, oval to elliptical, one-celled, and slightly curved, measuring 5-16. 2.2-3.5 m, and they are carried on simple phialids emerging laterally on the hypha or short, sparsely branched conidiophores. The macroconidia are hyaline, can have up to five septa but typically have only three, are constricted at the septa, are borne in sporodochia, can be straight or curved, frequently have a point at the tip with a rounded base, and measure 10-36. 3-6 m mostly 28. 4-5 m. One-celled, smooth, subduedly coloured chlamydospores range in size from 5 to 10 microns. They are both terminal and intercalary, frequently solitary but occasionally could form in chains, and they are abundantly created (Sastry and Chattopadhyay 2005).

4.2.4 Epidemiology Role and Disease Cycle

The fungus spreads by seeds as well as by soil. Mycelium and spores remain contaminate the seed surface of the diseased seed, despite the discovery that hyphae continue to exist in the cells that are parenchymatous of the seed coat. Chlamydospores from plant debris are the main way that the fungus thrives in the soil. The organism that causes malady can more readily enter the host cells by mechanical means when the crops are still in the growth phase of seedlings and their tissues are still pliable. When plants are afflicted, cortical cells start to shrink. Production of the enzymes polygalacturonase, pectin methyl esterase, cellulase, and protease appears to aid in the spread of the infection. Safflower plants with the illness have been found to contain the mycotoxins diacetoxyscirpenol and T-2. Additionally, the pathogen is said to release T-2 toxin, fusaric acid, diacetoxyscirpenol, and lycomarasmin in culture filtrate. However, as mentioned previously, the precise function of either enzymes or toxins is poorly understood or unknown. However, it has been claimed that the quantity of fusaric acid produced by *F. oxysporum* f. sp. *carthami* strongly correlates with its virulence. It has also been asserted that the pathogenicity is removed by preventing the production of fusaric acid. The amount of fungus dispersed throughout the vascular tissue found on the stem, side branch, as well as seed cap may limit the amount of diseased seeds per head. The vascular thread that runs through the pericarp to the receptacle connection is thought to be the route by which the fungus enters the seed. The pericarp and seed-coating tissue are affected by intra- and intercellular fungus. Different isolates of *F. oxysporum* f. sp. *carthami* have been reported to exhibit different morphology, culture traits, and pathogenicity (Sastry and

Chattopadhyay 2003; Murumkar and Deshpande 2009; Raghuwanshi and Dake 2009; Somwanshi *et al.* 2009).

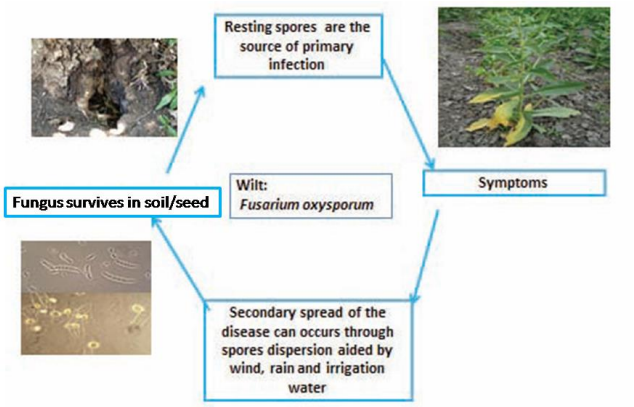

Fig 2. Disease cycle of *Fusarium oxysporum* disease

High nitrogen levels and warm, humid weather are also favourable to the sickness. According to reports, the wilt is less severe where paddy or millets are grown before safflower on fallow land. The incidence of the disease is said to be low in uplands with neutral to alkaline, clay-like soil (Kolte, 1985). Stress from high temperatures, poor drainage, and compacted soil all contribute to disease severity. The plant is more vulnerable to Fusarium wilt due to any cause that slows down root growth. The dense planting also makes plants more stressed and makes infections more likely. The impact of F. oxysporum f. sp. carthami tends to be apparent in flowering, as crops including their ability to produce are most susceptible to stress. The disease intensity decreases as the temperature drops (from 21°C to 15°C) between the last weekend of December to the beginning of February, while under Indian conditions, it may increase as the temperature rises (23.6°C). Depending on the variety and inoculum density, seedlings grow less prone to disease as they mature (Sastry and Chattopadhyay 1999a).

4.2.5 Management

4.2.5.1 Resistance sources

Numerous safflower genotypes have been tested for resistance utilising the water culture approach employing pathogen culture filtrate at a concentration of 3.5% (Shinde and Hallale 2009; Waghmare and Datar 2010). Thus, the causes of Fusarium wilt disease resistance in natural and domesticated *Carthamus* species have been discovered. *C. oxyacantha*, *C. lanatus*, *C. glaucus*, *C. creticus* and *C. turkestanicus* are examples of wild safflower species that are resistant to wilt.

By selecting and reselecting from superior lines of breeding developed from crossings of C. tinctorius, C. oxyacantha and *C. turkestanicus* resistant cultivars are being produced. The likely-to-succeed safflower genotypes with the greatest wilt resistance are GMU-1553 (Gadekar and Jambhale 2002b); 86-93-36A, 237550, VI-92-4-2, and II-13-2A (Sastry and Chattopadhyay 2003); GMU-1702, GMU-1706, and GMU1818 (Chavan *et al.* 2004); 96-508-2-90 (Anjani *et al.* 2005); HUS 305 (Sastry and Chattopadhyay 2003, Raghuwanshi *et al*. 2008, Singh *et al*. 2008b); WR-11-4-6, WR-8-24-12, WR8-19-10, WR-46- 5, WR-5-20-10, and WR-8-17-9 (Singh *et al.* 2008b); released hybrids DSH-129, NARI-NH-1, and NARI-H-15; and released cultivars A-1, PBNS-40, and NARI-6 (Murumkar *et al*. 2008b, 2009b, Prasad and Suresh 2012). Sehgal and Raina (2005) and Johnson *et al*. (2007) both produced information on the utilised of molecular markers for safflower germplasm characterization and for genotyping safflower cultivars. High levels of disease resistance can be identified in *C. lanatus* (2n = 22) and the alloploid that results from treating seedlings from a hybrid between *C. lanatus* and *C. tinctorius* (2n = 24) with colchicine. The dominant genes provided by the *C. lanatus* genome appear to minimize the malady resistance in the alloploid. It has been found that the accumulation of the antifungal chemical carthamidin (4, 5, 7, 8-tetrahydoxy flavone) in sick plants is what makes the plants resistant to infection.

Two dominant genes with complementary forms of gene action control some genotypes resistance to *F. oxysporum* f. sp. *carthami*, whereas inhibitory types of gene action control other genotypes' resistance (Shivani *et al*. 2011). And in yet other cases, resistance in seedlings is found to be straightforwardly monogenic dominant, whereas resistance in adult plants is discovered to be regulated by epistatic nonallelic interactions (Gadekar and Jambhale 2002b). However, the

emergence of novel races in the *F. oxysporum* f. sp. *carthami* natural population may hinder the development of long-lasting wilt-resistant cultivars (Kolte 1985).

4.2.5.2 Chemical Control

Fungicides like captan, thiram, carboxin, or a mixture of carboxin + thiram, benomyl, and carbendazim + mancozeb @ 0.1% or 0.2% can be used in seed treatment to minimise outermost layer infection by F. oxysporum f. sp. carthami and to effectively eradicate the fungus inside the seed. However, the efficacy of such fungicides is enhanced when used in conjunction with wilt-tolerant varieties or cultural methods (Sastry and Jayashree 1993, Govindappa et al. 2011b).

4.2.5.3 Cultural Control

In India, nonhost plants that are typically grown in succession with safflower, such as lentil, chickpea pea, and wheat, have been found to increase safflower yield and decrease the occurrence of wilt by secreting compounds that prevent the pathogen from growing (Kolte 1985; Sastry and Chattopadhyay 1999a; Sastry *et al.* 1993). Wheat and chickpea both increase the amount of antagonistic microorganisms in the rhizosphere, which significantly reduces the pathogen's ability to develop. Through exudates and extractives, the root system of *Ruellia tuberosa* L. displays potent therapeutic and protective benefits against the safflower wilt. The aerial fungicide potential of the root extractive is revealed. *R. tuberosa* is believed to have an inhibitory effect on *F. oxysporum* f. sp. *carthami* due to the quantities of 2,6-dimethoxy quinone, acacetin, and C16-quinone in the root exudates and extractives. *R. tuberosa*, a typical weed prevalent in India, is said to be able to grow in safflower farms to avoid wilt (Kolte 1985).

4.2.5.4 Biological Control

Trichoderma viride (Patibanda and Prasad 2004, Singh Saroj *et al*. 2006), *A. fumigatus* (Gaikwad and Behere 2001) and *Bacillus subtilis* have all been found to be antagonistic against *F. oxysporum* f. sp. *carthami*, showing their possible benefit for the managing of the malady. More encouraging findings are seen in local isolates of *Trichoderma* species (Waghmare and Kurundkar 2011). The employment of various disease control strategies in conjunction with integrated disease management has always been beneficial (Sastry *et al*. 2002). For instance, combining the use of NSKE at 5% and *T. harzianum* or *T. viride* at 4–10 g/kg seed on the moderately susceptible safflower variety A-1 or both (Prasad and Anjani 2008b) leads to substantial suppression of the infection along with the boost in safflower yield (Singh Saroj *et al*. 2006).

4.2.5.5 Botanicals

It has been identified that several plants extracts of leaf, including *Parthenium hysterophorus*, *Leucaena leucocephala*, *Vinca rosea*, *Gliricidia maculata*, *Ocimum basilicum*, *Eucalyptus globulus*, *Azardica indica*, *Datura metel*, and *Bougainvillaea spectabilis* can suppress the mycelial growth of *F. oxysporum* f. sp. However, when it comes to lowering the per cent wilt incidence of safflower, all of the examined leaf extracts fall short of Thiram (Kolase *et al*. 2000).

4.3 RUST

4.3.1 Economic Importance, geographical distribution and Losses

Rust, which is brought on by *Puccinia carthami* is the most prevalent disease affecting safflower. In Bohemia in 1840, Corda for the first time described it while he was battling *C. tinctorius* L. (Arthur and Mains 1922). This malady has been identified in all is growing regions of safflower and is widespread throughout the plant's native range (Kolte 1985). Recent reports of it include Oman (Deadman *et al*. 2005), China's snow lotus (Saussurea involucrata (Kar. & Kir.) (Zhao *et al*. 2007), and cross-border regional areas between Romania and Bulgaria (Anonymous 2014). In nations where the crop is farmed year after year, the malady is more severe. Safflower monoculture is so prohibited. Severe epiphytotic's of this rust were noted there following the arrival of the safflower crop in 1949 and 1950 (Schuster and Christiansen 1952). Because it now happens too late in the cultivating season to affect yields in the Great Plains of the United States, it is rarely a concern there (Lyon *et al*. 2007). Before 1990, the sickness appears to have caused large yield losses in India, but over the last 10 to 15 years, it has not been acknowledged as having a significant impact in diminishing safflower production (Prasad *et al*. 2006; Singh and Prasad 2007). However, it is also believed that seed and seedling contamination is economically significant because it is the origin of inoculum for beginning leaf infection. Furthermore, heavily polluted seeds won't grow well if they are saved for future plantings (Lyon *et al*. 2007). The mean yearly loss from safflower rust in the US is estimated to be roughly 5% and costs about $1 million, based on

calculations by Kolte (1985). Safflower rust's primary loss is the reduction in stand caused by sowing untreated teliospore-infested seed or seed where there are still viable soil-borne teliospores present. Only about 20% stand loss has been documented when using naturally rust-infected seed, compared to 98% stand loss when using artificially contaminated seed. Rust-infected yet rust-resistant safflower kinds display a stand loss of 26%, according to field tests with rust-resistant and rust-susceptible cultivars. But because these resistant kinds of surviving plants have the capacity for growth compensation, any yield loss is insignificant in comparison to the stand losses of susceptible types that range from 55% to 97% despite their substantially lower yield.

4.3.2 Symptomatology

Safflower rust has two distinct pathological phases: (1) root and foot maladies, which appear as rust signs on cotyledons, hypocotyls, etc.; and (2) leaf phase illness, which appears as rust indications on the leaves, blossoms, fruits, etc. later in the plant's development. The primary cause of rust in the seedling phase is the infection of developing seedlings by basidiospores brought on by the germination of soil- or seed-borne teliospores. Pycnia, which are initially represented as orange-yellow spots on cotyledons, may also cause the seedlings to droop and wilt. These patches later produce primary uredia, a uredinoid aecidia, which causes colour shifts. Many of these uredia become pustules, and subsequent pustules join together to form enormous rust pustules. Taproots and lateral root systems are examples of subterranean structures that have rust pustules. The epidermal and cortical layers of the affected area frequently display longitudinal cracking, as described by Schuster and Christiansen (1952). The primary cause of part of the cracks is the accidental roots that are distributed at the infection sites. Wilted plants may be able to survive thanks to their roots. Seedlings that are 8 to 10 weeks old may have an infected stem and develop orange-yellow pycnia. Girdling of the invaded area as a result of tissue collapse is a particularly distinctive sign on comparatively older plants. Due to their sturdy stems, these plants can stand upright, but their leaves are typically wilted. Due to wind or rain, these kinds of plants frequently break where they are girdled. The disease's foliar phase is indicated by uredial pustules that develop on flowers, leaves, and fruits. On the leaves, the chestnut-brown uredia remains scattered and wilted. Teleutospores develop in the uredopustules as the safflower plant matures, giving the corroded parts of the plant a dark-brownish tint.

4.3.3 Pathogen- *Puccinia carthami*

P. carthami (Hutz.) Corda is the pathogen. *P. carthami* is an obligate pathogen that lives on the Carthamus species and has an autoecious life cycle. Since real aeciospores are inadvertently left out of the life cycle of macrocyclic rust, the rust is said to be of the brachy-form type. The uredosori are sporadic and are typically located close to the pycnia on both sides of the leaves. Uredia can occasionally occur between two pycnia that are quite close to one another. Many globoids or broadly ellipsoid uredospores ranging 21-27. 21-24 m in size are seen in uredosori. The spore wall is 1.5–2.0 m thick. The uredospores are light chestnut brown, echinulate, and have three to four equatorial germ pores. Uredosori produce teleutosori. The teliospores are bicelled, ellipsoid, 36–44, 24–30 m, slightly or not constricted at septa, chestnut-brown, rounded or slightly obtuse at both ends, coarsely verrucose, 2.5–3.5 m thick at the side, and the spores are typically depressed from the apical position. The teliospores have a 10 lx-long pedicel, and are hyaline, delicate, and mainly deciduous. Pycnia are subepidermal, flask-shaped or spherical, and typically occur in groups. They have a diameter of 80–100 min. Numerous flexuous hyphae are visible sticking out, and the ostiole is filled with a lot of pycniospores.

4.3.4 Epidemiology Role and Disease Cycle

P. carthami is mostly kept alive during the uncropped season by teleutospores that lazily hang to seeds or hidden crop waste. Two categories have been established for teliospores. One of the two types is known to have a rapid germination ability, while the other shows a 5- to 6-week duration of dormancy. In the outdoors, teliospores—which signal dormancy—can survive for 12 months, yet only for 21. It was found that the contaminated safflower straw still had live teliospores despite 45 months of retention at these temperatures. Uredospores are unable to survive in the wild. They can, however, reportedly endure for more than a year in dry conditions at 8°C–10°C. After three weeks at room temperature, the uredospores become inactive. On infected plants, uredospores can survive for three weeks at 30°C to 31°C and for three days at 52°C to 55°C. Intriguingly, rust likes to directly form teliospores at temperatures above 40°C (Kolte 1985). Numerous wild species of Carthamus act as collateral hosts for P. carthami for it to survive (Sastry

and Chattopadhyay, 2005). In India, the wild C. oxyacantha variety of safflower is frequently infected with this rust, and it seems that this host develops an infection one month sooner than the farmed type. Additionally, during the off-season, live teliospores were observed on this wild safflower, suggesting a potential source for the pathogen's survival. Other Carthamus species, such as C. glaucus MB, C. lanatus L, C. syriacus (Boiss) Dinsm., and C. tenuis (Boiss) Bornm., also seem to be collateral hosts for P. carthami. The resting teliospore, which is one of two types of teliospores, overwinters and is still viable the following season, claim Prasad and Chothia (1950). However, it's possible that the first disease in the safflower plant was brought on by teliospores generated by native safflower species, especially those that don't need a dormant period after production. These may attack the wild variety first and create uredospores, which are then blown into the farmed safflower to begin the infection process. Alternatively, they may infect the safflower crop. Based on reports, the polyacetylenes in particular, found in crop residue from safflower, help teliospores germinate. Between 12 and 18 degrees Celsius is the optimum temperature range for teliospore germination. During typical germination, teliospores generate a four-celled promycelium containing a cell containing a tiny sterigma and a kidney-like sporidium. Infected roots and feet originate from this gametophytic reproduction, which appears as the formation of sporidia and occurs while seedlings are buried during the germination of the seeds period and before to plant emergence. While a temperature around 30°C and 35°C prevents such an infection, a lower temperature range of 5°C–15°C encourages a greater number of seedlings showing the root and foot stage of the disease. Changes in soil moisture from 35% and 80% of its capacity for holding water have not been shown to affect seedling rust infection. The elongation and hypertrophy of the afflicted seedlings are one of the key signs of *P. carthami* infection in seedlings. Orange patches made up of spermogonia occur on cotyledons a week after the main infection by sporidia, and primary uredosori form around them after 2 or 3 days. These infect the earliest leaves, causing the first foci of infection. Late in the growing season, secondary uredospores, the fungus's sporophytic generation, attack leaves. Uredospores can form a germ canal at temperatures ranging from 8°C to 35°C, however 18°C and 20°C are optimum. The germ tube develops an appressorium in the substomatal vesicle to aid in the penetration of leaf tissues through the stomata.

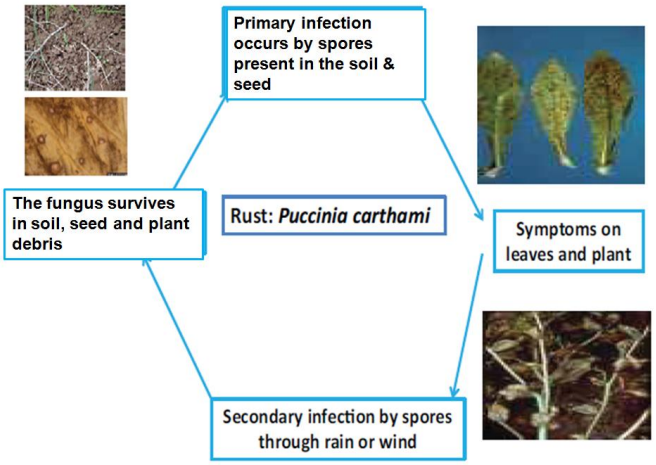

Fig 3. Disease cycle of *Puccinia carthami* disease

The infection benefits from high humidity levels and a chilly environment. Depending on the temperature, the incubation time is said to span 10 to 14 days. The incubation period is 10 days at the ideal temperature range of 18°C to 20°C; however, above 35°C, the rust uredospores may partially emerge and the disease might not manifest. The uredospores don't even start to germinate at 40°C. Applying uredospore treatment on seed mechanically to leaves only produces P. carthami uredo- and teliospores; seedlings are not infected. Recombination has the most potential because P. carthami is an autoecious macrocyclic rust that completes the sexual cycle quickly. In the US, there were several different rust races. To distinguish the races, several rust gradient hosts were created in the US (Kolte 1985).

4.3.5 Management

4.3.5.1 Host Plant Resistance

The rust-resistant safflower hypocotyls have been shown to be resistant to *P. carthami*. The resistant seedlings, on the other hand, do not show hypocotyl expansion; sporulation only occurs on the cotyledons, but the seedlings survive. The extremely vulnerable seedlings fail to thrive and

show widespread sporulation on the hypocotyl. Most of the time, it appears that foliage rust resistance and seedling rust resistance are physiologically and genetically connected. The disease seedling phase is likewise resistant to lines that have resistance to the leaf phase. Less than 5% of seedlings die from the rust's seedling phase in plants with a high level of leaf resistance. An effective way to screen for foliar rust resistance is closely correlated with the seedling rust resistance test. Thus, it is argued that the seedling test can be a useful tool for identifying foliar rust resistance. The microliter drop approach (a defined quantity of teliospores suspended within 1 mL) can be used to screen many different genotypes for resistance (Bruckart 1999). Researchers have examined how different safflower introductions and selections for rust resistance respond (Zimmer and Leininger 1965; Kalafat *et al*. 2009). Some safflower varieties have reportedly shown resistance to both the seedling and leaf stages of the disease. PI 170274-100, 193764-66, 19988282, 220647-98, 220647-55, 250601-109, 250721-93, 253759-62, 253911-25, 253912-9, 253913-5-72, 253914-5-108, 253914-7-9, and 257291-68 are among them. Other genotypes, including No. 1 and Tayan No. 1 in China and No. 30 and No. 26 in Turkey (Kalafat *et al*. 2009), are rust-resistant (Liu *et al*. 2009).

The safflower line N-l-1-5 has been discovered to be incredibly resistant to the rust's seedling phase, despite being just somewhat vulnerable to the disease leaf stage. PCA, PI 195895, and 6458-5 are further such lines that have a high level of seedling resistance. One dominant gene (N) controls the N-l-1-5 seedling resistance. This source should occasionally be given top priority when breeding for resistance to seedling rust. The successful application of seedling rust resistance on P. carthami may theoretically exert an identical impact on race establishment in a heteroecious species of the exact same genus as the removal of a different host. Reducing the quantity of primary inoculum through the utilisation of seedling rust-resistant cultivars would lessen the likelihood that new pathogenic strains will develop through vegetative recombination. The predominant source of disease in the foliar phase is seedling sickness.

4.3.5.2 Chemical Control

According to reports, safflower rust disease in seedlings is able to be avoided by applying a seed coating with fungicides such maneb, mancozeb, captafol, and thiram (each at a concentration of 0.2%–0.3%). Systemic fungicides, like oxycarboxin treatment of seeds, have been shown to work best when applied at an amount of 24-48 ounces per 100 kg of seeds to attempt to control the condition and stop the growth of disease-causing spores. To manage the foliar phase of the rust on safflower, two treatments of systemic fungicides, such as calnexin at 0.05% spaced 15 days apart, are helpful (Prasad and Suresh 2012; Varaprasad 2012).

4.3.5.3 Cultural Control

Safflower rust can be managed through cultural practices including avoiding low-lying locations for safflower cultivation, avoiding monocropping safflower, and delaying irrigation until the crop shows symptoms of moisture stress (Varaprasad 2012).

4.4 *Cercospora* Leaf Spot

4.4.1 Economic Importance, geographical distribution and losses

Cercospora sp caused safflower leaf spots to be widespread throughout the world, particularly when safflower is grown extensively as a single crop. It reportedly also occurs in Ethiopia, India, Iran, Israel, Kenya, the Philippines, and the former Soviet Union, in addition to the western Great Plains and Northern Plains of the United States (Mundel and Huang 2003). The epiphytotic incidence of the disease was recorded in the Coimbatore region of southern India in 1921, 1924, and 1925. Still, there is not much data on the stated monetary losses caused by the disease.

Research conducted in Montana, USA, between 2006 and 2007 reveals that safflower acts as an extra host for Cercospora beticola, an infection that infects sugar beetroot. If both crops are grown within four years of each other, this could result in novel disease threats affecting both crops (Lyon et al. 2007). This offers more proof that safflower serves as a secondary host for C. beticola. This is important because, in Montana, USA, wherein two crops may be cultivated next to one another, irrigated safflower is being explored more and more for use in rotation alongside sugar beetroot (Lartey et al. 2005, 2007).

4.4.2 Symptomatology

Plants of the sunflower family are impacted either a few weeks following sowing or during the flowering stage. The symptoms appear as 3–10 mm diameter brown sunken patches that are uneven or round in shape on leaves. The symptoms begin to show up on the lower leaves before gradually moving up to the middle and top leaves. Patches occasionally have zoning and have a golden colour to their boundary. The leaves may become curled and black with internal necrosis as the disease advances. When the patches are damp, the sporulation of the fungus gives them a velvety greyish-white appearance. On the upper and lower surfaces of the lesions on the impacted leaves, a little black fructification of the pathogen may be observed. Nodes and stems might also sustain damage. In severe cases, the infection also affects the bracts, which exhibit reddish-brown patches. Flower buds that are affected perish and turn brown. Without seed growth, the capitulum as a whole could also be harmed.

4.4.3 Pathogen-*Cercospora carthami*

The pathogen is *C. carthami* (H. and P. Sydow) Sundararaman and Ramakrishnan. Mycelium that is hyaline, smoky brown, septate, and branching collects in the stomatal areas where stromata are produced. Conidiophores can develop singly or as fascicles (tufts of 12–20 conidiophores) on both leaf surfaces. They immediately emerge from the epidermis in moist situations (Kolte 1985). The conidiophores are simple, septate, erect, and occasionally branched, and they range in size from 104.74 to 209.56. The characteristics of the conidia include hyaline, linear, 2-20 septate, and borne acrogeneously on the conidiophores. They are broad at the base and taper in a whip-like fashion towards the end, measuring 2.5–5.5–300 m. according to the length of the conidia, the amount of septavary, and the prevailing environmental conditions. In water, the conidia readily germinate and produce growth tubes on both the ends and sides. Each cell can produce a germ tube (Sastry and Chattopadhyay, 2005).

4.4.4 Epidemiology Role and Disease Cycle

C. carthami has a small host range and solely infects *Carthamus* sp., according to reports. The pathogen reproduces by embedding living stromata in agricultural waste and employing a vegetative saprobic mycelium. The pathogen's stromata, which are microscopic black specks organised in concentric rings, are visible on ill leaves. Conidia carried by the wind or water settle on safflower and develop when there is available moisture, initiating the disease cycle. The fungal organism enters plant components directly, via wounds or natural holes, or a combination of both. Warm, humid weather makes the disease worse, and infection needs massive, continuous early-morning dew or similar free moisture. According to Lyon et al. (2007), the Cercospora foliage spot pathogen can proliferate through wind, water splashing, and the motion of infected plant debris.

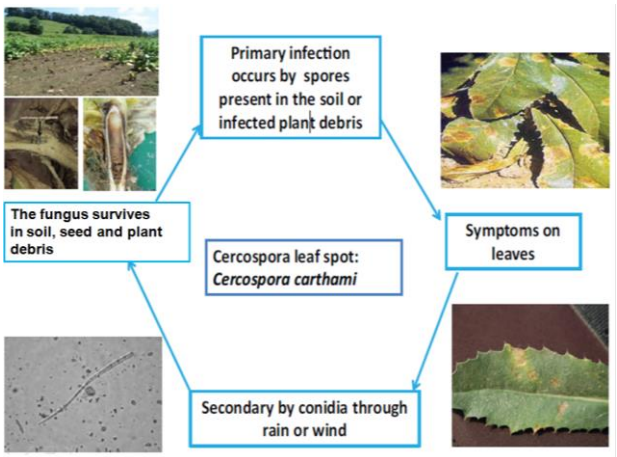

Fig 4. Disease cycle of *Cercospora carthami*

4.4.5 Management

4.4.5.1 Host Plant Resistance

Despite the high level of host plant resistance sources that are known, only five genotypes namely, 8-12-1, SSF-650, 2-10-2, 4-13-1, and 2-11-2—are resistant to both *Cercospora* leaf spot and aphid attack (Akashe *et al*. 2004). Spraying a 1% Bordeaux mixture on the diseased region will help manage it. Dithiocarbamate fungicides (0.25%) and copper oxychloride (0.3%), according to Prasad and Suresh (2012), may also be helpful in the treatment of the condition.

4.4.5.1 Chemical control

The management of the disease may benefit from a seed treatment with thiram 3 g/kg and spraying with mancozeb 2.5 g or carbendazim 1 g/L of water. When micro mobilized with safflower seeds, four rhizobacterial strains (GBO-3, INR937a, INR937b, and IPC11) were discovered to be inducers of systemic resistance in safflower, avoiding infection caused by *C. carthami* (Govindappa *et al*. 2013). For the *Cercospora* leaf spot, few specialised cultural management methods have been discovered. Crop switching lasting a minimum of three years to crops that are not host crops (such as tiny grains or corn), the addition of crop waste, and refraining from overhead watering and over-irrigation are likely to reduce the incidence and extent of Cercospora leaf spot (Lyon et al. 2007).

4.5 *Macrophomina* Root Rot – *M. phaseolina*

4.5.1 Economic Importance, geographical distribution and losses

The disease has, however, become widely prevalent in warm temperate and tropical regions of the world as a result of recent climate change. The illness is typically regarded as being of less importance during the regular crop-producing season in the winter months. According to Mahdizadeh *et al*. (2011) and Lotfalinezhad *et al*. (2013), it considerably lowers yields in Iran, especially during the dry seasons. According to Prasad and Suresh (2012), India often experiences yield losses of 1%–10% due to the sporadic *Rhizoctonia* disease phase. The production and height of the crop are inversely correlated with disease incidence, claim Chattopadhyay *et al*. (2003).

4.5.2 Symptomatology

The lesions start out as dark-brown to black on the roots. The epidermal as well as subepidermal membranes at the bottom of the stem along with the root of plants that are infected can ultimately take on a distinctive ash appearance. As the fungus advances up to the vascular and pith tissues of the stem, the infected tissues turn greyish-black and eventually form numerous tiny sclerotia that resemble finely ground charcoal (charcoal rot). Around the pith cavity and along the vascular components are sclerotia. Plants harmed develop stuntedly and mature too soon. Recent investigations of 30-day-old safflower plants have shown a novel type of distinctive stem-split symptom: tiny holes that grow both downward and upward, approximately 2-3 cm above the soil surface, resulting in a big split. A white to grey fungal mycelia mat develops inside the broken part, which turns brown and hollow (Govindappa *et al*. 2005). Such plants are not resilient.

4.5.3 Pathogen- *M. phaseolina* (Tassi) Goid

M. phaseolina (Tassi) Goid is the name of the pathogen, which is *R. bataticola* (Taub) Butler in its pycnidial stage. The details of the pathogen's characteristics and the disease cycle have been described under the titles of peanut and sunflower diseases. Using RAPD markers and UPGMA cluster analysis, the genetic diversity of isolates common in safflower-growing regions may be classified into two major types. There are various levels of genetic similarity, ranging from 50% to 55%, according to the dendrogram created by cluster analysis (Prasad *et al*. 2011, Navgire *et al*. 2014).

4.5.5 Management

4.5.5.1 Host Plant Resistance

The Indian Institute of Oilseeds in Hyderabad, India, has devised methodologies for seed germination utilising towel paper and infected soil cups to test safflower germplasm lines for disease resistance (Prasad and Navneetha 2010). However, neither in cultivated nor in wild safflower have sources of resistance been identified. The length and width of the necrotic lesion on the stem of safflower have been observed to positively and significantly correlate with the diameter of the lower stem (DLS) of safflower; as a result, the DLS trait needs to be utilised as an indication for the indirect selection of resistant genotypes in safflower (Pahlavani *et al*. 2007). IUT-k 115, GUA-va 16, CW-74, AC-Stirling, AKS-152, AKS-68, NARI-6, SSF-658, A-2, PBNS 12, and PBNS 40 are a few of the disease-tolerant genotypes (Pahlavani *et al*. 2007, Ingle *et al*. 2004, Prasad and Suresh 2012). Three genotypes, GMU-3265, GMU-3285, and GMU-3297, are found to be resistant with only up to 1–10 per cent seedling mortality, while four genotypes, GMU-3259, GMU-3262, GMU-3306, and GMU-3316, are identified as being extremely resistant with no seedling infection (Salunkhe 2014). These can be incorporated into breeding programmes to increase safflower resistance to *M. phaseolin*a-caused root rot and charcoal rot.

4.5.5.2 Chemical Control

There is no recommended chemical treatment that is both practically beneficial and affordable for the disease. fortunately the pathogen's seed-borne inoculum can be decreased by treating the

safflower seeds using thiram or carbendazim (Subeej25 DS) at a rate of 2 g/kg seed for prevention of disease and improved plant stand development in the growing environment (Prashanti et al. 2000a, Prasad and Suresh 2012).

4.5.5.3 Cultural Control

Prasad and Suresh (2012) proposed a number of cultural practises for disease control, such as the utilisation of neat seed, the usage of organic matter, long rotations alongside non-host crops, refusing overly thick plant populations, and hygiene practises like hand- or plough-interred debris throughout the summer.

4.5.5.4 Biological Control

Safflower rhizosphere soil-derived biocontrol agents like *Trichoderma harzianum*, fluorescent Pseudomonads (*P. fluorescens*), and *Bacillus subtilis* are utilised as seed treatments before being manufactured as talc-based formulations. In addition to controlling the disease, these biocontrol agents at 10 g/kg also demonstrate their efficacy in inducing systemic resistance by activating defence-related enzymes engaged in phenylpropanoid pathways. High activity of peroxidase, PAL, chitinase, polyphenol oxidase and beta-1,3-glucanase could be observed in *P. fluorescens* and *T. harzianum* treated safflower plants after challenge inoculation with *M. phaseolina* (Prashanti *et al*. 2000b, Kaswate *et al*. 2003, Singh *et al*. 2008a, Govindappa *et al*. 2010, 2011a). The lowest preemergence mortality caused by *M. phaseolina* is shown when a seed is treated with *T. harzianum* at a rate of 4 g/kg seed and sawdust + soil is added to the soil at a ratio of 1:10 (Deshmukh *et al*. 2003).

Refrances

Agrios GN (1978) Plant Pathology 2nd Ed, Academic PressInc., New York, New York. 703 p.

Akashe VB, Patil AJ, Indi DV and Mummkar DR (2004.) Response of different genotypes of safflower to major insect pests and leaf spot diseases. Sesame Safflower Newsl. 19: unpaginated.

Alosi MC, Hildebrand DC. and Schroth MN (1978) Entrapment of bacteria in bean leaves by films formed at air-water interfaces. (Abstr.) Phytopathol. News 12:197.

Anjani K, Singh H and Prasad RD (2005) Performance of multiple resistant line of safflower (*Carthamus tinctorius*). Indian J. Agric. Sci. 75: 178–179.

Anonymous (2014) Romania-Bulgaria, Safflower (*Carthamus tinctorius* L.). Cross-Border Cooperation Program-European Regional Development Fund, Bucharest, Romania.

Arthur JC and Mains EB (1922) Bullaria. N. Am. Flora 7: 482.

Awadhiya GK (1992) Seed borne pathogenic mycoflora of safflower. Crop Research 5(2):344-347.

Awadhiya GK (2000) Study of detection methods and location of *Alternaria carthami* Chowdhury in safflower seed. Gujarat Agric. Univ. Res. J. 26: 67–68.

Bar-Tal A, Landau S, Li-Xin Z, Markovitz T, Keinan M, Dvash L, Brener S and Weinberg ZG (2008) Fodder quality of safflower across an irrigation gradient and with varied nitrogen rates. Agronomy Journal, 100: 1499-1505.

Bergman JW and Jacobsen BJ (2005) Control of Alternaria blight (*Alternaria carthami* Choud.) of safflower (*Carthamus tinctorius* L.) in the United States Northern Great Plains region. In: Proceedings of the Sixth International Safflower Conference—SAFFLOWER: A Unique Crop for Oil Spices and Health Consequently, a Better Life for You, Istanbul, Turkey, June 6–10, pp. 215–221.

Berville A, Breton C, Cunliffe K, Darmency H, Good AG, Gressel J, HallLM, McPherson MA, Medail F, Pinatel C, Vaughan DA and Warwick SI (2005) Issues of ferality or potential ferality in oats, olives, the pigeon–peagroup, ryegrass species, safflower, and sugarcane. In: Gressel J (Editor)–Crop ferality and volunteerism: a threat to food security in the transgenicera? Taylor and Francis Books, CRC Press, Boca Raton, pp. 231–255.

Borkar SG, Shinde R (1988). Alternaria leaf spot of safflower. Seed Res. 16:126-127

Borkar, SG (1997) Spines, a new avenue for infection by *Alternaria carthami* on safflower. J. Mycol. Plant Pathol. 27: 311–316.

Bramhankar SB, Asalmol MN, Pardey VP and Ingle YV (2002) Effect of foliar spray of different fungicides on transmission of *Alternaria carthami* (Chowdhury) from plant to seed and seed to plant. Ann. Plant Physiol. 16: 112–114.

Bramhankar SB, Asalmol MN, Pardey VP, Ingle YV and Burgoni EB (2001) Efficacy of fungicides against Alternaria leaf blight of safflower. PKV Res. J. 25: 99–101.
Bruckart WL (1999) A simple quantitative procedure for inoculation of safflower with teliospores of the rust fungus, *Puccinia carthami*. Plant Dis. 83: 181.
Carson ET, Richardson PE, Essenberg JK, Brinder-hoff LA, Johnson WM and Venere RJ (1978) Ultrastructural cell wall alterations in immune cotton leaves inoculated with *Xanthomonas malvacearum*. Phytopathol. 68:1015- 1021.
Chand JN and Walker JC (1964) Inheritance of resistance to angular leafspot of cucumber. Phytopathol, 54:51-53.
Chapman MA, John Hvala, Jason Strever and John M. Burke (2010) Population genetic analysis of safflower (*Carthamus tinctorius*; Asteraceae) reveals a Near Eastern origin and five centers of diversity", American Journal of Botany, Vol. 97, pp. 831-840
Chattopadhyay C (2001) Yield losses attributable to Alternaria blight of safflower (*Carthamus tinctorius* L.) and some effective control measures. J. Mycol. Plant Pathol. 31: 298–392.
Chattopadhyay C, Meena PD, Sastryand RK, and Meena RL (2003) Relationship among pathological and agronomic attributes for soilborne diseases of three oilseed crops. Indian J. Plant Prot. 31: 127–128.
Chauhan LS and Singh DR (1979) Occurrence of mosaic on safflower in northern India. Indian Phytopathology, 32: 301-302.
Chavan RA, Bharose AA, Pawar RB and Patil VD (2004) Screening of safflower germplasm lines and penetration mechanism of *T. viride* against Fusarium wilt. J. Soils Crops 14: 79–82.
Chen G (2009) Induced resistance in safflower against safflower rust disease by exogenous chemicas. Master's dissertation, Sichuan Agricultural University, Ya'an, China.
Chenulu VV, Sachchidanandaand J and Prakash N (1971) Safflower mosaic. Phytopathology, 71: 129-134. https://doi.org/10.1111/j.1439-0434.1971.tb03148.x
Chowdhury S (1944) An Alternaria disease of safflower. J Indian Bot. Sci. 23:59-65.
Crosse JE and Garrett CME (1963) Studies on the bacteriology of Pseudomonas mors-prunorum, *P. syringae*, and related organisms. J. Appl. Bact. 26:159-177.
Dajue L and Mundel HH (1996) Safflower (*Carthamus tinctorius* L.). Promoting the conservation and use of underutilized and neglected crops. 7. International Plant
Darrel Lee Jacobs (1982) Bacterial leaf and stem blight of safflower in Montana: its epidemiology, sources of resistance and inheritance of resistance. Master of Science in Plant Pathology, Montana State University.
Daub ME and Hagedorn DJ (1980) Growth kinetics and interactions of *Pseudomonas syringae* with susceptible and resistant bean tissues. Phytopathol. 70:429-436.
Deadman ML, Al-Sadi AM, and Al-Jahdhami S (2005) First report of rust caused by *Puccinia carthami* on safflower in Oman. Plant Dis. 89: 208.
Desai SA (1998.) A note on the preliminary screening of safflower genotypes against Alternaria leaf spot in northern Karnataka. Karnataka J. Agric. Sci. 11: 824.
Deshmukh VV, Chore NS and Jiotode DJ (2003) Effect of soil amendments with saw dust and viability of Trichoderma harzianum in different carriers. Crop Res. (Hisar) 26: 508–511.
Dordas C (2008) Role of nutrients in controlling plant diseases in sustainable agriculture: A review. Agron. Sustain. Dev. 28: 33–46. doi:10.1051/agro.2007051
Doudoroff M and Palleroni NJ 1974) Genus Pseudomonas. Pages 217-243 in: R.E. Buchanan and N.E'. Gibbons, eds.
Driouich A, Jauneau A, and Staehelin LA (1997) 7-Dehydrobrefelin A, a naturally occurring brefeldin A derivative, inhibits secretion and causes a cis-to-trans breakdown of Golgi Stocks in plant cells. Plant Physiol. 113: 487–492.
Dubey V, Aminuddin K and Singh VP (2010) Molecular characterization of Cucumber mosaic virus infecting Gladiolus, revealing its phylogeny distinct from the Indian isolate and alike the Fny strain of CMV. Virus Genes, 41: 126-134. DOI: 10.1007/s11262-010-0483-6
Dwivedi A (2005) Handbook of Research on Information Technology Management and Clinical Data Administration Health Care. Meical Information Science Reference, Hershey-NewYork, 1027p.
Dye DW, Bradbury JF, Dickey RS, Goto M, Hale CN, Hayward AC, Kelman A, Lelliott RA, Patel PN, Sands DC, Schroth MN, Watson DRW and Young JM (1975) Proposals for a reappraisal of

the status of the names of plant pathogenic Pseudomonas species. Int. J. Syst. Bact. 25:252-257.
Emongor VE (2010) Safflower (*Carthamus tinctorius* L.) the underutilized and neglected crop: A review. Asian Journal of Plant Sciences, 9 (6): 299- 306
English H and Davis JR (1960) The source of inoculum for bacterial canker and blast of stone fruit-trees. Phyto- pathol. 50:634 (Abstr.).
Ercolani GL (1969) Epiphytic survival of Pseudomonas mors-prunorum Wormald from cherry and *P. syringae* van Hall from pear on the host and on the non-host plant. Phytopathol. Mediterr. 8:197-206.
Erwin DC, Starr MP and Desjardins PR (1964) Bacterial leaf spot and stem blight of safflower caused by *Pseudomonas syringae*. Phytopathol. 54:1247-1250.
FAOSTAT (2019) Crop production data, Food and Agriculture Organization of the United Nations, Statistics Division online database, http:/www.fao.org/faostat/
Fletcher JD (2001) New hosts of Alfalfa mosaic virus, Cucumber mosaic virus, Potato virus Y, Soybean dwarf virus, and Tomato spotted wilt virus in New Zealand. New Zealand Journal of Crop and Horticultural Science 3: 213-217. DOI: 10.1080/01140671.2001.9514180
Gadekar DA and Jambhale ND (2002a) Inheritance of seedling and adult plant resistance to Alternaria leaf spot in safflower (*Carthamus tinctorius* L.). Indian J. Genet. Plant Breed. 62: 238–239.
Gadekar DA and Jambhale ND (2002b) Genetics of seedlings and adult plants resistant to wilt caused by *Fusarium oxysporum* f. sp. *carthami* in safflower. Sesame Safflower Newsl. 17: 81–84.
Gaikwad SJ and Behere GT (2001) Biocontrol of wilt of safflower caused by *Fusarium oxysporum* f. sp. *carthami*. In: Proceedings of the Fifth International Safflower Conference—Safflower: A Multipurpose Species with Unexploited Potential and World Adaptability, Williston, ND, July 23–27, 2001, pp. 63–66.
Garcia A (2009) Insulin production from transgenic safflower. http://www.sembiosys.ca/.
Garrett CME, Panagopoulos CG and Crosse JE (1966) Comparison of plant pathogenic psuedomonads from fruit trees. J. Appl. Bacteriol. 29:342-356.
Gayathri DA and Madhuri V (2014) Seed mycoflora of safflower and its control by using otanicals, bioagents and fungicides—A review. Int. J. Appl. Biol. Pharm. Technol. 5: 209–215.
GBIF (2023) Global Biodiversity Information Facility, Denmark, https://www.gbif.org/ (accessed 7th April 2023)Genetic Resources Institute, pp.1-83.
Gholve VM, Ghuge SB, Pawar SV and Barde PT (2017) In vitro efficacy ofvarious seed dresser on seed mycoflora of safflower. Agric. Update, 12(TECHSEAR-5): 1440-1445;
Gholve VM, Taware MR, Wagh SS. (2015) Symptomatology, isolation, identification and pathogenicity test of Alternaria blight of safflower. Trends Biosci. 8(1):57-60.
Gnanamanickam SS and Kandaswamy TK (1978) Bacterial leaf spot and stem blight - A new disease of safflower in India caused By *Pseudomonas syringae*. Current Sci. 47: 506-508.
Govindappa M, Lokesh S, and Rai VR (2005) A new stem-splitting symptom in safflower caused by *Macrophomina phaseolina*. J. Phytopathol. 153: 560–561.
Govindappa M, Lokesh S, Rai VR, Naik VR, and Raju SG (2010) Induction of systemic resistance and management of safflower Macrophomina phaseolina root-rot disease by biocontrol agents. Arch. Phytopathol. Plant Prot. 43: 26–40.
Govindappa M, Rai VR and Lokesh S (2011a) Screening of *Pseudomonas fluorescens* isolates for biological control of *Macrophomina phaseolina* root-rot of safflower. Afr. J. Agric. Res. 6: 6256–6266.
Govindappa M, Rai VR and Lokesh S (2011b) *In vitro* and *in vivo* responses of different treating agents against wilt disease of safflower. J. Cereals Oilseeds 2: 16–25.
Govindappa M, Rai VR, and Lokesh S (2013) Induction of resistance against Cercospora leaf spot in safflower by seed treatment with plant growth-promoting rhizobacteria. Arch. Phytopathol. Plant Prot. 36: 1–14. doi:10.1080/03235408.2014.880574.
GRDC (2010) Raising the bar with better safflower agronomy, ACT, Australia, Grains Research and Development Corporation.
GRDC (2017) GrowNotes Safflower Northern, Grains Research and Development Corporation, Australia.

Gud MA, Murumkar DR, Shinde SK and Kadam JR (2008) Correlation of weather parameters with development of leaf spot of safflower caused by *Alternaria carthami*. In S. E. Knight and T. D. Potter (Eds.), Safflower: Unexploited potential and world adaptability. Proceedings of 7th International Safflower Conference, Wagga Wagga, New South Wales, Australia. ISBN: 978-0-646-50329-5.

Harish Babu BN, Naik VR, Hanumantharaya L, Raju SG and Yaragoppa SD (2005) Evaluation of 6 promising breeding lines of safflower for Alternaria tolerance, seed yield and its components. Karnataka J. Agric. Sci. 186: 803–806.

Hildebrand DC (1973) Identification of species within the syringae group of Pseudomonas using nutritional screening. Pages 5-9 in Synopsis. Int. Soc. Plant Pathol. Working Party on Pseudomonas syringae group. Inst. Nat. Rech.Agron, Angers, France, April 1973. 118p.

Hoitink HAJ, Hagedorn DJ and McCoy E (1968) Survival,transmission, and taxonomy of *Pseudomonas syringae* van Hall, the causal organism of bacterial brown spot of bean (*Phaseolus vulgaris* L.). Can. J. Microbiol. 14:437-441.

Indi DV, Murumkar DR, Patil AJ and Akashe VB (2004) Screening of safflower germplasm against Alternaria leaf spot under field conditions. J. Maharashtra Agric. Univ. 29: 344–346.

Ingle VN, Deshmukh VV, Jiotode DJ, Chore NS and Dhawad CS (2004) Screening of safflower varieties against root rot (*Rhizoctonia bataticola*). Res. Crops 5: 113–114.

Jalender P, Bharati NB and Anitha K (2015) Studies on host range of cucumber mosaic virus in Tomato (*Solanum lycopersicum* L.). Ecology, Environment and Conservation, 21. Suppl. Issue; 417-420.

Johnson RC, Kisha TJ and Evans MA (2007) Characterizing safflower germplasm with AFLP molecular markers. Crop Sci. 47: 1728–1736.

Kalafat S, Karakaya A, Kaya MD and Bayramin S (2009) A preliminary study on the reactions of some safflower genotypes to rust disease. Bitki Koruma Bulteni 49: 183–187.

Kalpana Sastry R (1996) Symptoms of wilt disease—clues for use in resistance breeding. In: Hegde DM, Raghavaiah CV, Pati D (Eds.), Proceedings of Training Programme on Breeding Approaches for Improving Productivity of Safflower and Group Meeting on Heterosis Breeding in Safflower. Directorate of Oilseeds Research, Rajendranagar, Hyderabad, pp. 25–32.

Kalpana Sastry R, Chattopadhyay C (2003) Development of Fusarium wilt resistant genotypes in safflower (*Carthamus tinctorius*). Eur. J. Plant Pathol. 109, 147–151. https://doi.org/10.1023/A:1022502618887.

Kaswate NS, Shinde SS and Rathod RR (2003) Effect of biological agents against different isolates of *Rhizoctonia bataticola* (Taub.) Butler in vitro. Ann. Plant Physiol. 17: 167–168.

Kizil S, Cakmak O, Kirici S and Inan M (2008) A comprehensive study on safflower (*Carthamus tinctorius* L.) in semi-arid conditions. Biotechnology and Biotechnological Equation, 22(4): 947-953.

Klement Z and Goodman RN (1967) The hypersensitive re action to infection by bacterial plant pathogens. Ann. Rev. Phytopathol. 17-44.

Klisiewicz JM (1975) Race 4 of *Fusarium oxysporum* f. sp. *carthami*. Plant Dis Rep. 59: 712.

Klisiewicz JM and Houston BR (1962) Fusarium wilt of safflower. Plant Dis. Rep. 46: 748–749.

Klisiewicz JM, Houston BR and Petersen LJ (1963) Bacterial blight of safflower. Plant Pis. Reptr. 47: 964-966.

Kneusel RE, Schiltzg E and Maternhl U (1994) Molecular characterization and cloning of an esterase which inactivates the macrolide toxin brefeldin A*. J. Biol. Chem. 269: 3449–3456.

Kolase SV, Deokar CD and Sawant DM (2000) Effect of plant extracts on the incidence of safflower wilt. Sesame Safflower Newsl. 15: 92–94.

Kolte SJ (1985) Diseases of Annual Edible Oilseed Crops, Vol. III: Sunflower, Safflower and Nigerseed Diseases. CRC Press, Boca Raton, FL.

Krishna Prasad NVK, Basuchaudhary KC (1989) Symptomatology and epidemiology of Alternaria leaf blight of safflower in India. Second Intern. Conf., 9-13 January, ISOR, DOR, Hyderabad. 271-275.

Kulkarni VR and Byadgi AS (2004) Incidence of safflower mosaic disease in North Karnataka. Karnataka Journal of Agricultural Sciences. 17(4): 836-837

Kumar S, Heena Ambreen, Murali TV, Sapinder Bali, Manu Agarwal, Amar Kumar, Shailendra Goel and Arun Jagannath (2015) Assessment of genetic diversity and population structure in a

global reference collection of 531 accessions of *Carthamus tinctorius* L. (safflower) using AFLP markers. Plant Molecular Biology Reporter, Vol. 33, pp. 1299-1313.
Lalit Mahatma, Singh SD and Lodha PC (2001) Pathogenic seed mycoflora of Sesame (*Sesamum indicum* L). Journal of Mycology and Plant Pathology. 31(3):377-379.
Lartey RT, Caesar-TonThat TC, Caesar AJ, Shelver WL, Sol NI and Bergman JW (2005) Safflower: A new host of *Cercospora beticola*. Plant Dis. 89: 797–801.
Lartey RT, Lenssen AW, Evans RG and Ghoshroy S (2007) Comparative structural study of leaf spot disease of safflower and sugar beet by *Cercospora beticola*. Plant Pathol. J. (Faisalabad) 6: 37–43.
Latorre BA and Jones AL (1979) Evaluation of weeds and plant refuse as potential sources of inoculum of *Pseudomonas syringae* in bacterial canker of cherry. Phytopathol. 69:1122-1125.
Li D and Mundel HH (1996) Safflower *Carthamus tinctorius* L., Vol. 7, Institute of Plant Genetics and Crop Plant Research, Gatersleben/International Plant Genetic Resources Institute, Rome.
Liu TT, Gao F, Ren YZ, Li GY and Tan Y (2009) Identification of safflower varieties rust-resistance. Xinjiang Agric. Sci. 46: 719–723.
Lotfalinezhad E, Mehri Z and Sanei SJ (2013) Temperature response of *Macrophomina phaseolina* isolates from different climatic in Iran. Annu. Rev. Res. Biol. 3(4): 724–734.
Lyon D, Burgener P, Harveson R, Hein G and Hergert G (2007) Growing safflower in Nebraska. NebGiude. University of Nebraska-Lincoln, Extension Institute of Agriculture and Natural Resources, Lincoln, NE.
Madhu MR, Basavarajappa MP, ChidanandLokapur and Anand ID (2018) Efficacy of fungicides in inhibition of *Alternaria carthamic a* using leaf spot of safflower *In vitro*. International Journal of Chemical Studies 2018; 6(1): 1051-1053.
Mahdizadeh V, Safaie N and Goltapeh EM (2011) Diversity of Macrophomina phaseolina based on morphological and genotypic characteristics in Iran. Plant Pathol. J. 27: 128–137.
Manu Upadhyay, Ram Chandra and Ajad Patel (2019) Bio-efficacy of plants extracts against *Alternaria carthmic a* used Alternaria Leaf spot of safflower. Journal of Pharmacognosy and Phytochemistry 2019; 8(2): 1615-1618
Mayee CD, Datar VV (1986) Phytopathometry. Technical Bull-I, MAU, Parbhani. pp. 88-89.
Milosevic D, Ignjatov M, Marjanovic Jeromela A, Nikolic Z, Tamindzic G, Miljakovic D, Stankovic I (2020) Presence and molecular characterization of cucumber mosaic virus on safflower in Serbia. Ratar. Povrt., 57 (2), 49-54.
More SD, Raghavaiah CV, Hangarge DS, Joshi BM and Dhawan A S (2005) In: Esendal, E. (Ed.), VIth International Safflower Conference, Instanbul, Turkey, 6-10th June, pp. 180-186.
Mundel HH and Huang HC (2003) Control of major diseases of safflower by breeding for resistance and using cultural practices. In: Advances in Plant Disease Management, eds., H.C. Huang and S.N. Acharya. Research Signpost, Trivandrum, India, pp. 293–310.
Munoz-Valenzuela S, Musa GLC, Montoya-Coronado L and Rivera-Rojas VM (2007) Evaluation of safflower genotypes in Northwest Mexico. In: New Crops and New Uses, eds., J. Janick and A. Whipkey. ASHS Press, Alexandria, VA, pp. 193–195.
Murumkar DR and Deshpande AN (2009) Variability among isolates of *Fusarium oxysporum* f.sp. *carthami*. J.Maharashtra Agric. Univ. 34:178–180.
Murumkar DR, Gud MA and Deshpande AN (2008b) Screening of released varieties and hybrids of safflower against wilt (*Fusarium oxysporum* f. sp. *carthami*). J. Plant Dis. Sci. 3: 114–115.
Murumkar DR, Indi DV, Akashe VB, Patil AJ and Gud MA (2009b) Multiple resistance sources against major diseases and pests of safflower, *Carthamus tinctorius* L. J. Oilseeds Res. 26: 175–176.
Murumkar DR, Indi DV, Gud MA and Shinde SK (2008a) Field evaluation of some newer fungicides against leaf spot of safflower caused by *Alternaria carthami*. In: Seventh International Safflower Conference—Safflower: Unexploited Potential and World Adaptability, Wagga Wagga, New South Wales, Australia, November 3–6, 2008.
Murumkar DR, Indi DV, Gud MA, Shinde SK and Deshpande AN (2009a) Fungicidal management of leaf spot of safflower caused by *Alternaria carthami*. J. Maharashtra Agric. Univ. 34: 54–56.
Navgire KD, Prasad RD and Wadhave AV (2014) Genetic diversity of *Macrophomina phaseolina* causing root rot of safflower. In: “Abstract-cum-Souvenir” National Symposium on Plant

Pathology in Genomic Era, Indian Phytopathological Society, New Delhi, India, May 26–28, 2014, p. 45.
Neergaard P (1977) Seed Pathology, Vol I and II, MacMillan Press, London,U.K, p.1187.
Padaganur GM and Anilkumar TB (1976) seed mycoflora of safflower and its control. Pesticides, 10 (7): 39-41.
Pahlavani MH, Razavi SE, Mirizadeh I and Vakili S (2007) Field screening of safflower genotypes for resistance to charcoal rot disease. Int. J. Plant Prod. 1: 45–52.
Patibanda AK and Prasad RD (2004) Screening Trichoderma isolates against wilt pathogens of safflower, *Carthamus tinctorius* L. J. Biol. Control 18: 103–105.
Pawar SV, Ghuge SB, Gholve VM and Sutar DS (2017) Screening of safflower germplasm/advanced material /parental lines against major disease Alternaria leaf spot. Agric. Update,12(TECHSEAR-5) : 1208-1212; DOI: 10.15740/HAS/AU/12.TECHSEAR(5)2017/1208-1212.
Pawar SV, Utpal Dey, Munde VG,. Sutar DS and Dibakar Pal (2013)Management of seed/soil borne diseases of safflower by chemical and biocontrol agents. African Journal of Microbiology Research
Prabhakar M, Padmavati P, Prasad RD, Prayaga L (2012) Retrieval of biophysical parameters and assessment of Alternaria disease severity in safflower using ground based hyper spectral radiometry. J Oilseeds Res. 29:377-381.
Prasad KVV (1985) Seed borne *Alternaria carthamiin* safflower and its control. Indian Phytopathology. 38:602.
Prasad R and Chothia H (1950) Studies on safflower rust in India. Phytopathology 40: 363.
Prasad RD and Anjani K (2008a) Sources of resistance to Alternaria leaf spot among Carthamus wild species. In: Seventh International Safflower Conference—Safflower: Unexploited Potential and World Adaptability, Wagga Wagga, New South Wales, Australia, November 3–6, 2008, pp. 1–2.
Prasad RD and Anjani K (2008b) Exploiting a combination of host plant resistance and Trichoderma species for the management of safflower wilt caused by *Fusaruim oxysporum* f. sp. *carthami* Klisiewicz and Houston. J. Biol. Control 22: 449–454.
Prasad RD and Navneetha T (2010) Techniques for pathogenicity testing and screening of safflower cultivars against *Macrophomina phaseolina*. J. Oilseeds Res. 27: 199–200.
Prasad RD and Suresh M (2012) Diseases of safflower and their management. In: Safflower Research and Development in the World: Status and Strategies, eds., I.Y.L.N. Murthy, H.
Prasad RD, Navaneetha T and Rao N.N (2011) Cultural, morphological, pathogenic and molecular diversity in *Macrophomina phaseolina* isolates of safflower from southern India. Indian Phytopathol. 64: 247–253.
Prasad RD, Sharma TR and Prameela Devi T (2007) Molecular variability and detection of Fusarium species by PCR based RAPD, ISSR and ITS-RFLP analysis. J. Mol. Plant Pathol. 37: 311–318.
Prasad RD, Sharma TR, Jana TK, Prameela Devi T, Singh NK and Koudal KR (2004) Molecular analysis of genetic variability in Fusarium species using microsatellite markers. Indian Phytopathol. 57: 272–279.
Prasad RD, Singh V and Singh H (2006) Crop protection. In: Research Achievements in Safflower, ed., D.M. Hegde. All India Coordinated Research Project on Safflower, Directorate of Oilseeds Research, Hyderabad, India, pp. 52–77.
Prashanthi SK, Kulkarani S, Sangam VS and Kulkarni MS (2000a) Chemical control of *Rhizoctonia bataticola* (Taub.) Butler, the causal agent of root rot of safflower. Plant Dis. Res. 15: 186–190.
Prashanthi SK, Kulkarni S and Anahosur KH (2000b) Management of safflower root rot caused by *Rhizoctonia bataticola* by antagonistic microorganisms. Plant Dis. Res. 15: 146–150.
Raghuwanshi KS and Dake GN (2009) Fusarium wilt of safflower (*Carthamus tictorius* L.): A review. J. Plant Dis. Sci. 4: 110–117.
Raghuwanshi KS, Dake GN, Mate SN and Naik RM (2008) Pathogenic variations of *Fusarium oxysporum* f. sp. *carthami*. J. Plant Dis. Sci. 3: 241–242.
Raghuwanshi KS, Deokar CD (2002) Studies seed borne mycoflora of safflower. Sesame and safflower newsletter. 17.

Rajagopalan T and Shanmugam N. (1993) Studies on host range and seed borne nature of Alternaria carthemi incitant of leaf spot of safflower. Madras Agriculture Journal. 70: 832.

Rajendraprasad M, B Vidya Sagar, Uma Devi G, Narendra Reddy C and Triveni S (2021) Detection and identification of the seed mycoflora associated with safflower seed samples. The Pharma Innovation Journal; 10(4): 51-56.

Rajeswari B, Keshavulu K, Krishna Rao V. (2012) Management of seed mycoflora of safflower. Journal of Oil Seeds Research, 29:332-335.

Ranaware A, Singh V and Nimbkar N (2010) *In vitro* antifungal study of the efficacy of some plant extracts for inhibition of *Alternaria carthami* fungus. Indian J. Nat. Prod. Resour. 1: 384–386.

Rao VN, Sastry RK, Craufurd P, Meinke H, Parsons D., Rego TJ, Rathore A (2014) Cropping systems strategy for effective management of Fusarium wilt in safflower. Field Crop. Res. 156, 191–198. https://doi.org/10.1016/j. fcr.2013.11.013

Ravinder T, Govinda Rao N and Sastry KS (1992) Effect of homeopathic drugs on Cucumber mosaic virus infection in Safflower. Indian Journal of Virology. 8(1): 40-44.

Saad SM and Hagedorn DJ (1972) Relationship of isolatesource to virulence of *Pseudomonas syringae* on Phaseolus Vulgaris, Phytopathol. 62;678-680.

Salunkhe VN (2014) Screening of safflower germplasm accessions for resistance source against Macrophomina root rot. Bioscan 9: 689–690.

Sands DC, Schroth MN and Hildebrand DC (1970) Taxonomyof phytopathogenic pseudomonads. J. Bacteriol. 10l:9-23.

Sastry KR and Chattopadhyay C (1999a) Influence of non-host crops on the survival of *Fusarium oxysporum* f. sp. *carthami*. J. Mycol. Plant Pathol. 29: 70–74.

Sastry KR and Chattopadhyay C (1999b) Effect of soil solarization on *Fusarium oxysporum* f. sp. *Carthami* population in endemic soils. Indian Phytopathol. 52: 51–55.

Sastry KR and Chattopadhyay C (2005) Diseases of safflower. In: Diseases of Oilseed Crops, eds., Saharan GS, Mehta N and Sangwan MS. Indus Publishing Co., New Delhi, India, pp. 236–267.

Sastry KR and Chattopdhyay C (2001) Effect of soil solarization on wilt incidence and seed yield of safflower (*Carthamus tinctorius* L.). Indian J. Plant Prot. 29: 47–52.

Sastry KR and Jayashree J (1993) Eradication of wilt fungus from heavily infected safflower seed. J. Oilseeds Res. 10: 227–231.

Sastry KR, Rego RTJ and Burford J.R (1993) Effects of cropping systems on wilt of safflower. In: Proceedings of the Third International Safflower Conference, eds., L. Dajue and H. Yunzhou. Beijing, China, June 14–18, 1993, pp. 636–645.

Sastry RK, Chattopadhyay C, Singh V and Hegde DM (2002) Integrated management of safflower wilt using host resistance, cultural and chemical measures. J. Mycol. Plant Pathol. 32: 189–193.

Scharen AL, Bergman JW and Burns EE (1976) Leaf diseases of Winter wheat in Montana and losses from them in 1975. Plant Pis, Reptr9 JO;686-690.

Schneider RW and Grogan RG (1977) Bacterial speck oftomato: sources of inoculum and establishment of a resident population. Phytopatho1. 67:388-3.94.

Schuster ML and Christiansen DW (1952) A foot and root disease of safflower caused by *Puccinia carthami* Cda. Phytopathology 42: 211.

Sehgal D and Raina SN (2005) Genotyping safflower (*Carthamus tinctorius*) cultivars by DNA fingerprints. Euphytica 146: 67–76.

Sequeira L, Gaard G and De Zoeten GA (1977) Interaction of bacteria and host cell wall St its relation to the mechanism of induced resistance. Physiol, Plant Pathol, IQ: 43-50.

Shinde AB and Hallale BV (2009) Standardization of water culture technique for Fusarium wilt of safflower. Int. J. Plant Sci. 4: 248–249.

Shinde AB, Hallale BV and Vaidya AP (2008) Antifungal activity of leaf extracts against Alternaria blight of safflower. Natl. J. Life Sci. 5: 203–206.

Shinde Anjali B and Hallale BV (2013) Biological management of seed pathogen of safflower. Internat. J. Plant Sci., 8 (2): 458-460.

Shivani D, Sreelakshmi C and Kumar CVS (2011) Inheritance of resistance to Fusarium wilt in safflower (*Carthamus tinctorius* L.) genotypes. J. Res. ANGRAU 39: 25–27.

Singh AK, Chakrabarti DK and Basuchaudhary KC (1975) Two new diseases of safflower from India. Curr. Sci. (India) 44: 397.

Singh Saroj, Kumar S and Suryawanshi DS (2006) Evaluation of different treatments for the management of safflower insect pests and diseases under rainfed conditions. Indian J. Entomol. 68: 286–289.

Singh SN, Agarwas SC and Khare MN (1987) Seedborne mycoflora of vand de Faculteit L and bouwwetensxhappen, Universiteit Gen., 57:161-163

Singh V and Nimbkar N (2006) Safflower (*Carthamus tinctorius* L.), in: Genetic Resources, Chromosome Engineering, and Crop Improvement: Oilseed Crops, CRC Press, pp. 167-194.

Singh V and Prasad RD (2007) Integrated management of pests and diseases in safflower. Directorate of Oilseeds Research, Hyderabad, India, p. 49.

Singh V, Prasad RD (2005) Integrated Management of Pests and Diseases in Safflower. Directorate of Oilseeds Research, Hyderabad, India.

Singh V, Ranaware AM and Nimbkar N (2008a) Safflower: Unexploited potential and world adaptability. In: Seventh International Safflower Conference, Wagga Wagga, New South Wales, Australia, November 3–6, 2008, pp. 1–4.

Singh V, Ranaware AM and Nimbkar N (2008b) Breeding for Fusarium wilt resistance in safflower. In: Seventh International Safflower Conference—Safflower: Unexploited Potential and World Adaptability, Wagga Wagga, New South Wales, Australia, November 3–6, 2008, pp. 1–5.

Smith JR (1996) Safflower, Champaign, Illinois, AOCS Press.

Smith JR (1996) Safflower. AOCS Press, Champaign, IL, p. 624.

Somwanshi SD, Deshpande GD and Magar JS (2009) Differential resistance of safflower genotypes to three isolates of *Fusarium oxysporum* f. sp. *carthami* Klisiewicz and Houston. Adv. Plant Sci. 22: 343–346.

Stankovic I (2020) Presence and molecular characterization of cucumber mosaic virus on safflower in Serbia. Ratar. Povrt., 57 (2), 49-54.

Sumitha R and Nimbkar N (2009) Efficacy of fungicides against Alternaria leaf spot disease in safflower. Ann. Plant Prot. Sci. 17: 507–508.

Taware MR, Gholve VM, Utpal Dey (2014) Bio-efficacy of fungicides, bioagents and plant extracts/botanicals against *Alternaria carthami*, the causal agent of Alternaria blight of safflower (*Carthamus tinctorius* L.). Aftrican J Microbio. Res. 2014; 8(13):1400-1412.

Thies JA (2000) Reactions of regional sweet potato breeding clones and standard check cultivars to southern root-knot nematode race 3 and Fusarium Wilt. Biol. Cult. Tests 15, 194.

Thomas BJ (1981) Virology: hardy nursery stock. In: Annual report Glass House Crops Research Institute, Littlehampton, West Sussex, pp 143–146

Thomas V, Norton R and Wachsmann N (2008) Variation in response to *Alternaria carthami* infection by a range of safflower (*Carthamus tinctorius*) varieties. In: Seventh International Safflower Conference— Safflower: Unexploited Potential and World Adaptability, Wagga Wagga, New South Wales, Australia, November 3–6, 2008, pp. 1–3.

Valand GB, Gaikwad NY and Patel AJ (1983).Seed borne fungi of some Sesame varieties. Indian Journal ofMycology and Plant Pathology. 13(3): 363-365.

Valleau WD, Johnson EM and Diachun S (1944) Root infection of crop plants and weeds by tobacco leaf-spot bacteria. Phytopathol. 34:163-174.

Varaprasad KS (2012) Safflower scenario: Status and future strategies. In: Souvenir, Eighth International Safflower Conference, Safflower Research and Development in the World: Status and Strategies. Indian Society of Oilseeds Research, Hyderabad, India, January 19–23, 2012, pp. 1–9.

Wagh SS, Suryawanshi AP, Ambadkar CV and Badgujar SL (2020) Symptomatology and pathogenic variability of *Alternaria carthami* isolates from Maharashtra state infecting safflower crop. International Journal of Chemical Studies 2020; 8(2): 1533-1538

Wagh SS, Suryawanshi AP, Badgujar SL and Ambadkar CV (2020) Integrated management of Alternaria blight of safflower caused by *Alternaria carthami* under field conditions. International Journal of Chemical Studies. 8(2): 1957-1962

Wagh SS, Suryawanshi AP, Badgujar SL and Ambadkar CV (2020) Screening of safflower varieties/cultivars, genotypes and germplasm lines against *Alternaria carthami*. International Journal of Chemical Studies. 8(2): 1929-1931

Waghmare SJ and Datar VV (2010) Studies on seed borne nature of *Fusarium oxysporum* f. sp. *carthami* causing wilt of safflower. J. Maharashtra Agric. Univ. 35: 491–492.

Waghmare SJ and Kurundkar BP (2011) Efficacy of local isolates of Trichoderma spp. against *Fusarium oxysporum* f. sp. *carthami* causing wilt of safflower. Adv. Plant Sci. 24(1): 37–38.

Walker JC and Patel PN (1964) Inheritance of resistanceto halo blight of bean. Phytopathol. 54:952-954.

Weiss EA (1971) Castor, Sesame and Safflower. Barnes and Noble, Inc., New York, pp: 529-744.

Weiss EA (1983) Oilseed crops: Safflower. Longman Group Limited, Longman House, London, UK, pp: 216-281.

Wichman DM (1996). Safflower for forage. In: Mundel HH, Braun J and Daniels C (Eds.). Proceedings of North American Safflower Conference, Great Falls, Montana, Lethbridge, AB, Canada, pp: 56-60.

Wunsch M, Schafer M and Kraft B (2013) Field evaluation of fungicides for management of rust and Alternaria blight on safflower. Carrington Research and Extension Center, North Dakota State University, Carrington, ND.

Zanjare Snehal S, Suryawanshi AV, Zanjare SR and Shelar VR (2020) *In vitro* evaluation of bio control agents against seed borne *Alternaria carthami* and *Alternaria alternate* causing leaf spot disease of safflower. International Journal of Chemical Studies. 8(1): 2994-2997.

Zayed MA, Yehia AH, El-Sebaey MA and Gowily AM (1980) Studies on the host–parasite relationships of safflower root rot disease caused by *Fusarium oxysporum* S Clecht. Egypt. J. Phytopathol.12: 63–70.

Zhao S, Xie GL, Li H and Li C (2007) Occurrence of rust caused by *Puccinia carthami* on snow lotus in China. Plant Dis. 91: 772.

Zimmer DE and Leininger LN (1965) Sources of rust resistance in safflower. Plant Dis. Rep. 49: 440.

Zohary D, Hopf M and Weiss E (2012) "Dye crops", in: Domestication of Plants in the Old World: The origin and spread of domesticated plants in Southwest Asia, Europe, and the Mediterranean Basin, Oxford University Press, pp. 166-168