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PEA CROP DISEASES - AN OVERVIEW

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Abstract

The pea (Pisum sativum L.) represents one of the most important leguminous crops worldwide, being in the top 10 vegetable crops, belonging to the Fabaceae family. Peas are grown both for human consumption, fresh or canned, and for animal feed in the dry state. The pea crop is affected by an important number of pathogens, which in favorable conditions can significantly decrease both the yield and the quality of the grains, even leading to total losses. Fungi, bacteria and viruses can cause a number of foliar diseases in peas. The most important pathogens that cause significant economic damage are: Didymella pinodes, Neocosmospora pisi, Pea enation mosaic virus¹, Peronospora pisi, Uromyces pisi, Pseudomonas pisi. Pea enation mosaic virus¹ can cause severe loss of pea harvest by up to 50% and Neocosmospora pisi leads to a decrease in yield by 15-60%. The Ascochyta blight disease complex can decrease the yield with values ranging from 10-60%. This bibliographic review provides an overview of recent studies on the main pathogens of pea crops.

Key words: *control, pathogens, Pisum sativum.*

INTRODUCTION

The pea (*Pisum sativum* L.) is native to the Middle East and is the oldest cultivated plant in the world (Zeven & Jukovski, 1975). Peas leave the soil enriched in nitrogen, having the ability to fix molecular nitrogen from the atmosphere (Popescu&Roman,2008), through nitrogen-fixing nodules following the symbiosis with bacteria of the genus Rhizobium, thus contributing to the improvement of soil fertility (Matsumiya et al., 2013). Production of dried peas has expanded in developed countries such as France, Canada and Australia, where it is used for protein supplements. Varieties of peas grown for processing shall be harvested when they are green and juicy and dried peas shall be harvested when the seeds reach a moisture content of 12% or less. Fresh peas are often grown in rotation with other vegetables. Pea is usually the first crop sown in the spring, and is therefore often planted on cool, wet soils (Grunwald et al., 2004). In Romania, the areas cultivated with peas are somewhat reduced, compared to other countries of the world. According to the latest FAO data, the area of peas cultivated in Romania increased in 2020, registering 94,360 thousand/ha compared to 2015 when 31,056 thousand/ha were cultivated. The seed is an important means of disease transmission to plants (Berca et Cristea, 2015; Dudoiu et al., 2016; Zaharia et al., 2022). Disease transmission through pathogen-bearing seeds also involves proper management of pathogen control (Couture et al., 2002). Fungi and bacteria can cause major foliar diseases of pea crops. Pathogenic fungi cause significant losses on the pea crop. Among the most important diseases caused by fungal pathogens we list: Downy mildew (*Peronospora pisi*), *Ascochyta* blight (*Didymella pinodes*) and rust (*Uromyces pisi*) (Grunwald et al., 2004).

PEA PATHOGENS

1. Ascochyta blight complex, is one of the main diseases affecting field pea production and can be caused by several pathogens of the genus *Ascochyta* (Tivoli & Banniza, 2007). The main pathogen that causes ascocytosis was initially named *Mycosphaerella pinodes* (Berk.

Et Blox) Nissel, the anamorph or conidial form *Ascochyta pinodes* (Br. Et Bl.) but Peever et al. (2007) proposed that the name *Didymella* be used because Internally Transcribed Spatial Region (ITS) DNA sequences are clustered with *Didymella* species and not with *Mycosphaerella* species. ***Didymella pinodes*** is the most widespread causative pathogen and the most damaging. In Australia, other *Phoma* species have also been shown to be pathogens of peas, including *Phoma koolunga* (Davidson et al., 2009), *Phoma herbarum* (Li et al., 2011) and *Phoma glomerata* (Tran et al., 2014), associated with the *Ascochyta* disease complex. All these pathogens can occur together in a pea crop, even on a single plant (Hare & Walker, 1944). Disease-causing fungi can be spread via seeds, but this is a minor source of infection compared to spores released from plant residues of the previous crop (Bretag et al., 2006). Spores can be carried by wind several kilometers, and once the crop is infected, the current plant lesions act as a secondary source of spores for further spread.

Symptoms. The disease manifests itself on all aerial organs of the plant: leaves, stems and pods. On plants that have just emerged, the disease makes its presence felt on the leaves, circular spots appear, dark brown in color, being basically isolated. On the stem and petiole, the spots are deep in the tissues and arranged longitudinally, showing a dark brown color with a dark and slightly raised edge. The characteristic form of manifestation of the disease appears on the pods, showing circular or irregular spots, confluent or isolated, light brown, outlined with a reddish border. If the infection occurs later, after the formation of the grains, the mycelium of the fungus also reaches the seeds, the disease manifesting itself in the form of dark or light yellow spots with a diffuse border. Next to the attacked tissues, small light brown or blackish dots appear during the vegetation period, which represent the pycnidia of the fungus. On the remains of diseased plants, the perithecia of the fungus are formed on the leaves over the winter, in the form of small black dots. *Didymella pinodes* can infect newly emerged seedlings and all above-ground parts of adult pea plants, causing seedling root rot, necrotic spots on leaves, lesions and blackening of stem base, and dark brown discoloration of seeds (Ahmed et al., 2015).

Life cycle. Transmission from one year to another occurs via infected seeds and plant debris. Dissemination of the fungus during the vegetation period is done by pycnospores and ascospores, carried by air currents. The primary inoculum (asexual conidia or ascospores) of all causative pathogens is spread by wind and rain on newly emerged crops (Carter & Moller, 1961). Only *D. pinodes* is known to produce ascospores (Punithalingham & Holliday, 1972), which develop from pseudothecia on infested stubble or senescent plant material, whereas all causative pathogens produce conidia. Regardless of the type of inoculum, the initial infection of the leaves results in small purple or black spots, which spread in wet conditions, leading to the death of the leaves (Roger & Tivoli, 1996b). Pycnidia develops in the resulting lesions and conidia is spread to neighboring plants (Schoeny et al., 2008). The conidial mode of spread increases disease severity more at the base of the plants than in the mid or upper parts of the plants (Tivoli et al., 1996). In Australia for example, conidia are considered of minor importance (Bretag et al., 2006) and ascospores are the main factor in the secondary spread of *D. pinodes*. Ascospores are produced in pseudothecia when infected leaves and stems become senescent and are forced into the air when moisture conditions are met. Ascospores are rapidly spread throughout the crop and subsequent precipitation promotes rapid infection, increasing disease severity (Roger & Tivoli, 1996). Stem infection may begin at the soil line and spread upward, with lesions often coalescing on the stem. When fungi develop on the petiole of an infected leaf, a stem lesion may begin at the base of the dead leaf, advancing above and below that point. Subsequent lesions eventually coalesce, surrounding the stem (Hare & Walker, 1944; Tivoli et al., 1996). Infection of the flowers causes them to dry up and drop, while infection of the pods causes them to become distorted and may drop. This effect may be transient and is often unobserved (Hare & Walker, 1944).

Primary air-borne inoculum. Moisture is an important factor in the release of ascospores. When the pseudothecia becomes moist, the asci enlarges and ruptures at the tip, releasing all the ascospores simultaneously into the air (Carter 1963; Hare & Walker, 1944). The timing of ascospore counts was analyzed and showed that dew is effective in causing ascospore release, with the highest numbers occurring during periods of rainfall (Carter & Moller, 1961; Carter, 1963). A diurnal spore release experiment was noted, with a peak in the late afternoon and a trough in the middle of the night (Bretag & Lindberck, 2006; Carter, 1963).

Soil-borne inoculum. Pathogens, with the exception of *A. pisi,* survive in the soil for many years, and grain yield was strongly correlated with the amount of fungus (*Ascochyta blight*) present in the soil (Bretag & Ward, 2001). This source of inoculum is particularly important in field pea growing areas, where inoculum accumulates in the soil if the pea has been grown in succession (Wallen & Jeun, 1968). Pathogens survive as chlamydospores, mycelium or sclerotia. *D. pinodes* is also a moderate saprophyte (Dickinson & Sheridan, 1968; Sheridan,1973), have shown an increase in detectable levels in soil in the 6-12 months after harvest, after an initial decrease in the first 6 months. Burying infested stubble decreased survival time below 12 months (Davidson et al., 1999; Sheriden, 1973; Zhang et al., 2005) possibly by preventing saprophytic growth by depleting oxygen and microbial activity.

Seed-borne inoculum. All pathogens can infect seeds (Kraft et al., 1998; Maude, 1996). Surface sterilization of seeds resulted in a decrease in *D. pinodes* infection from 60% to 18%, leading to the conclusion that most of the time the pathogen is carried on the seed coat (Bathgate et al., 1989). High seed infection is influenced by several factors, namely spring rainfall that disperses the inoculum, early sown crops that are exposed to large numbers of airborne ascospores, but also later harvested crops that are more likely to be exposed to the inoculum secondary (Bretag et al., 1995). Seed lots grown in areas with low rainfall, less than 350 mm per year, can be free from pathogens, making these areas suitable for seed production (Bathgate et al., 1989). Under controlled conditions transmission of pathogens from seed to the basal parts of the plant is frequent, for example 40% for *A. pisi* and 100% for *D. pinodes*, leading to death of young plants (Maude, 1966; Xue and Warkentin, 2001). However, the disease does not spread to the upper parts of the plant, suggesting that seed infection is not an important source of inoculum for an *Ascochyta blight* epidemic (Bretag et al., 1995; Moussart et al., 1998).

Secondary inoculum. During the growing season, both pycnidia and pseudothecia have been observed on the same plant organs, pycnidia are produced on both green and senescent plant organs, while pseudothecia appear only on senescent parts, appearing just before flowering. Discharge of both types of spores is initiated by precipitation or dew, so that epidemics are more severe in humid conditions (Roger & Tivoli, 1996). When ascospores are produced, the disease spreads rapidly to the top of the plant crown. The greatest damage caused by *Ascochyta blight* is produced by this secondary spread of ascospores (Bretag, 1991; Hare & Walker, 1944). In *D. pinodes* pseudothecia can appear 18 days after the appearance of lesions. Because they mostly form on senescent stems, the greatest number of pseudothecia are found on organs with senescent tissues. The number of ascospores remains relatively low until the end of the season, when pseudothecia develop on senescent material, and then the number of ascospores can triple (Roger & Tivoli, 1996). Pycnidia can form within 11 days from the onset of symptoms and increase in number to the end of the vegetative cycle of the crop. Roger and Tivoli (1996) found that pycnidia formed when lesions covered approximately 25% of the leaf surface.

Infection process. Conidia of *D.pinodes* germinate with one or more germ tubes, which frequently branch and form appressor-like structures on the leaf and cotyledon surface 6 h after inoculation (Clulow et al., 1991; Roger et al., 1999). Penetration occurs through epidermal walls 8 h after inoculation (Nasir et al., 1992; Roger et al., 1999), not through

stomata, and then an infection vesicle is formed, located partly in the epidermal wall and partly in the cell lumen. From this the penetrating hypha arises and then initiates intra and intercellular hyphae (Nasir et al., 1992). Penetration occurs within 24 hours of inoculation with cell wall degrading enzymes (Heath & Wood, 1969; Roger et al., 1999) and rapid colonization by *D. pinodes* is soon followed by tissue collapse (Heath & Wood, 1969) in both resistant and susceptible genotypes (Nasir et al., 1992). In resistant types, the formation of infection vesicles and penetration hyphae is reduced, and the development and spread of lesions is delayed (Nasir et al., 1992). Symptoms may appear within 24 hours of inoculation and consist of brown spots, 2 mm in diameter, which continue to grow and coalesce, leading to death of the entire leaf, and pycnidia may form within 3 days (Heath & Wood, 1969; Roger et al., 1999). Symptoms on the cotyledon are visible only after 4 days after inoculation (Clulow et al., 1991). Increasing inoculum concentration increases disease severity by increasing the rate of lesion expansion (Heath & Wood, 1969; Roger et al., 1999) but inexplicably does not affect lesion number (Heath & Wood, 1969). The specialized literature indicates that there has been no clarification of this aspect since the study was carried out.

Detection and Quantification. Over time, several studies have been reported on the *D. pinodes* infection process on resistant and susceptible pea lines (Heath & Wood, 1969; Clulow et al., 1991; Nasir et al., 1992). Nasir et al. (1992) showed that the infection process of D.pinodes on pea leaves started with the germination of conidia and the formation of one or more germ tubes which later ramified and formed appressor-like structures on the surface the leaf. Subsequently, vesicle-like infection structures were formed, which formed penetrating hyphae and developed a network of hyphae with intra- and intercellular growth in the pea tissues. However, Clulow et al. (1992) showed that 32 h after inoculation, infection by *D. pinodes* on the leaf could occur without apressor formation by direct penetration of the cuticle through the germ tube tips. *D. pinodes* infection on the epicotyl, for a period of 30h after inoculation on susceptible pea lines, most germ tubes produced one appressorium that penetrated the cuticle (Clulow et al., 1992). Colonies on the nutrient substrate are generally light gray to almost dark gray in color, the pseudothecia and pycnidia are distributed along mycelial rays growing from the central point. After 20-30 mm growth, the pseudothecia and pycnidia become arranged in concentric rings in response to a 12 h photoperiod (Onfroy et al., 1999). Pycnidia production is high in light and decreased at low temperatures (Hare & Walker, 1944). On the stems, the pycnidia have a diameter of 100-200 µm. Conidia are hyaline, 1 or occasionally 2 septate, slightly constricted at sep and 8-16 x 3-4.5 µm (Punithalingham & Holliday, 1972). *D. pinodes* produces dark brown, globular pseudothecia with papillate ostioles 90 x 180 µm in diameter. As a general rule, pseudothecia will develop on poor or minimal media, while pycnidia are more likely to develop on highly nutritious media, although significant variability occurs among tupins (Hare & Walker, 1944; Roger & Tivoli, 1996). *D. pinodes* can produce pseudothecia on malt agar, Mathur agar medium, oatmeal agar medium. The favorable temperature for pseudothecia development and maturation was at 16°C, decreasing at 20°C, rare at 24°C and 28°C, and nonexistent at 30°C. From 12°C to 4°C, the same number of pseudothecia, but time to maturity increased from 35 to 100 days at lower temperature. At 16°C pseudothecia developed in 25-30 days (Hare & Walker, 1944).

Management. Cultivation of resistant varieties, use of healthy seed, crop rotation (3-4 years), collection and destruction of infected plant residues, deep plowing, weed control, seed treatment with specific recommended products, specific control methods. Unfortunately, sources of resistance to *Didymella pinodes* fungi are very limited, and pea varieties highly resistant to this disease have not yet been developed. Delayed seeding by 3-4 weeks reduces *D. pinodes* severity by more than 50%, however, such measures are not suitable at higher altitudes due to the shorter growing season. Crop management is the preliminary option to control the progress of the disease by minimizing the repainting of the inoculum, as well as the survival of the inoculum on plant

residues and in the soil by avoiding reinfection. Burying infected residues also decreases the survival of pathogens. Integrating two or more control methods increases the chances of success (Jeger, 2004; Mc Donald & Peck, 2009). Jha et al. (2019) conducted research by applying foliar fungicides such as Hexaconazole @ 0.1% which was applied 2 times at 15 day intervals, this being the most effective in reducing the severity of *Ascochyta blight* up to 10.65% compared to the control (42.08%). The percent reduction in the incidence of the disease compared to the control after the application of the fungicide was 74.69%. In case of treatment with carbendazim 0.1%, the disease incidence was reduced to 12.74% compared to the control. In research conducted by Liu et al. (2016), fungicides based on tebuconazole, boscalid, iprodione, carbendazim and fludioxonil were found to be more than 80% effective in controlling the disease. Also, 3 biocontrol strains of *Bacillus* sp. and one of *Pantoea agglomerans* significantly reduced disease severity under greenhouse and field conditions (Liu et al., 2016). Other fungicides have been used to effectively control ascochyta blight but also to increase yield such as: mancozeb, chlorothalonil, thiabendazole (Xu and Warkentin, 1996; Bretang et al., 2006). However, the application of fungicides may increase production costs, and may also pose a risk to the environment due to diversion to other non-target areas. In addition, intensive application of fungicides can lead to strains that are resistant to commercial chemicals (Liu et al., 2016). Limitations on fungicide application have prompted the exploration of safer and more environmentally friendly biological control measures against *Ascochyta blight*. The bacterial antagonists *Pseudomonas fluorescens*, *Bacillus* spp., and *Serratia* spp. significantly reduced the severity of *Ascochyta blight* under greenhouse conditions (Wang et al., 2003). The mycoparasite *Clonostachys althaea* strain ACM941 represented an effective bioagent in the control of root rot complex caused by *A. pinodes* (Xue, 2003). The role of individual pathogens in the *Ascochyta* complex must also be understood in order to develop successful management practices that target all pathogens involved in this disease.

2. Downy mildew caused by the pathogen *Peronospora viciae* f. sp. *pisi* (Sydow) Boerema & Verhoeven (family Peronosporaceae, ord. Peronosporales). The pathogen exhibits long, thin, hyaline sporangiophores, ramified dichotomously in the upper third. The stegrims are short, divergent, unequal, slightly pointed at the tip. The sporangia are ovoid, yellowish, 22-27 x 1519 µm. Downy mildew leads to high yield and pod quality losses in pea crops. Peas are affected by a number of diseases, but downy mildew can be devastating in cool, wet conditions.

Symptoms. Downy mildew symptoms in peas can be local or systemic (Bathula and Singh, 2022). Systemic infection is the most severe form of the disease and usually results in stunting and stunting of plants leading to their death before flowering. Downy mildew usually appears on peas in the early stages of plant growth or when the weather is cool and wet. Diseased plants may show symptoms characterized by lack of vigor, small size, wilting and eventually death of the plants. In already developed plants, leaves appear, from the base of the plant, with chlorotic spots at first and later brown, usually located at the edges and covered on the underside with gray or purple powders (Melgarejo et al., 2010). Infected leaves turn yellow and die if the weather is cold and wet.

Life cycle. The fungus survives in the soil, on plant debris and in seeds. The disease is influenced by climatic conditions, namely continuous drizzle or days in which fog persists for more than 12 hours with temperatures between 15-20°C.
In these conditions, abundant conidia appear that infect the plants. From 20°C the disease stops evolving. The pathogen produces abundant inoculum in the form of sporangia on the surface of infected plants. Foliar infections are usually local and start on the underside of the leaf. The pathogen can infect and sporulate also on inflorescences and tendrils. Pod infection can occur in relatively high humidity conditions even in the absence of foliar infection. Infected pods become deformed and blister the surface. The optimal conditions for the development of downy mildew are cold and

wet weather. Secondary spread of the disease occurs only through sporangia (Bathula and Singh, 2022). Systemic infection is the result of direct infection of the upper meristem. This type of infection is most common in cultivars with reduced stipule size (Matthews & Dow, 1983; Taylor et al., 1990). Local infections on leaves develop from conidia present on the plant surface (Mence & Pegg, 1971). Severe infections can lead to general plant deformity, which can lead to early plant death. *Peronospora pisi* can often go undetected by remaining asymptomatic until a 12-hour period occurs with at least 90% relative humidity, favoring an environment conducive to disease expression. This can lead to sporadic outbreaks, the severity and expression of the disease depending on environmental conditions and agricultural practices specific to each area (Marr et., 2021).

Soil-borne infection. Oospores in the soil are the primary inoculum early in the season. Oospores can survive for 10-15 years in soil (Olofsson, 1966).

Air-borne inoculum. The rate of disease progression is also greatly determined by relative humidity (RH). Exposure of leaves to moisture for a period of at least 3-4 hours is necessary to initiate infection (Olofsson, 1966; Pegg & Mence, 1970). The temperature can vary between 1 and 24°C, with an optimum between 12 and 20°C (Pegg & Mence, 1970). Initiation and production of conidia require greater than 90% RH for at least 12 hours (Olofsson, 1966) and reach a maximum at 100% RH. Most conidia lose viability within 3 days of removal (Pegg & Mence, 1970). Wind-distributed conidia from neighboring fields or from more distant cultivated areas are also important sources of primary inoculum. Conidia distributed by wind or dispersed by water droplets play an important role in the spread of the disease in pea crops. Young plants are more sensitive than mature plants. Results presented by Stegmark (1988) support the hypothesis that pea downy mildew mainly infects young tissue. Mence and Pegg (1970) demonstrated that terminal embryonic leaves, not yet developed at the time of inoculation, were found to be more susceptible to the disease than more mature leaves. In addition, increased resistance was found in older seedlings. This was found when seedlings of different ages, i.e. 2-6 nodes developed, were inoculated in the same experiment (Stegmark, 1991).

Detection and quantification. Singht et al. (2020) studied downy mildew intensity and meteorological parameters in different pea cultivars noting a negative correlation with minimum, maximum temperature, relative humidity and sunshine hours and a positive correlation with wind speed both in protected space as well as in the field.

Host resistance. Resistance variation in pea cultivars has been reported by Olofsson (1966), Allard (1970), Ryan (1971) and Stegmark (1988). Some pea cultivars are fully resistant to some isolates but are fully susceptible to others. However, there are also pea genotypes that have partially stable, never complete, resistance to different isolates (Stegmark, 1990). Race-specific complete resistance has been found in several cultivars, but there is no pea genotype with complete resistance to all known pathogen races (Ester and Gerlagh, 1979; Matthews & Dow, 1983). The *Pisum* gene bank, Weibullsholm Collection, kindly provided by Stig Blixt, was tested for resistance to oospore infection of germinated seeds at Nordreco (Stegmark, 1994). One line (L1382) showed complete resistance in replicate studies when pre-germinated seeds were soaked in a conidia suspension according to a method described by Ryan (1971). This line has red flowers and brown seeds. When the seed coat was removed before sowing, the seedlings were severely infested with downy mildew. This shows that the seed coat contributes most to the resistance of this line.

Partial resistance. The cultivar "Dark Skin Perfection" (DSP) was more resistant to downy mildew than other cultivars used in canning and freezing pea production (Olofsson 1966; Stegmark, 1988). However, DSP is also affected by downy mildew under conditions favorable to the pathogen. Stegmark (1988) described a pea breeding line with a high level of partial resistance. This line showed low

susceptibility to all isolates of the fungus, but never complete resistance to any isolate (Stegmark, 1990).

Management. There are a variety of approaches to managing downy mildew, including growing resistant varieties, crop rotation, seed treatment and foliar fungicide applications. Implementing a good crop rotation is not only an economic strategy for reducing downy mildew impacts, but is also favorable from a general pest management perspective. Cultivating resistant cultivars and using fungal seed treatments are also economical options in downy mildew control. Plant infection is often caused by oospores present in the soil. Seed treatment with Aliette Super® (fosetyl-aluminium 528 g/kg + thiram 172 g/kg + thiabendazole 129 g/kg) and Wakil XL (50 g/kg fudioxonil + 175 g/kg metalaxylM + 100 g/kg cymoxanil) was also proven effective in controlling downy mildew (Pung et al., 2005). An advantage of seed treatment over foliar fungicide application is the lower cost of application when seed treatments are used. However, if downy mildew occurs in the crop, foliar fungicides can provide good control. Mixtures of phosphoric acid (2000 g a.i./ha) and chlorothalonil (1296 g a.i./ha) considerably reduced disease severity in field experiments (Pung et al., 2005). Regardless of the specific control measures used, pea crops should be routinely surveyed to prevent downy mildew occurrence and severity as part of a proactive pest management strategy. Further research by Falloon et al. (2000) showed that matalaxyl treatments applied to pea seeds were ineffective in controlling downy mildew in pea. Seed treatment with cymoxanil or fosetyl-Al provided better protection against the pathogen (Falloon et al., 2000).

3. Pea rust. Pea rust can be caused by different pathogens depending on the climatic conditions. In temperate regions of the world, pea rust is caused by *Uromyces pisi* (Pers.) Wint. (Emeram et al., 2005), while in tropical and subtropical regions, the fungus that causes the rust is *Uromyces fabae* (Pers.) de Bary (Rai et al., 2011). Based on the morphology of the telia and infection structures, these two species can be differentiated using internal transcription spatial (ITS) markers (Emeran et al., 2005; Barilli et al., 2006). *Uromyces pisi* can cause yield losses of over 30% (EPPO 2012) compared to *U. fabae* which can lead to losses of up to 50% (Kushawaha et al., 2006). Pea rust has become an important pathogen since the mid-1980s and is mainly distributed in Europe, North and South America, India, China, Australia and New Zealand, especially in regions with warm and humid weather (EPPO, 2012).

Symptoms. The attack is manifested by the appearance of discoloration spots on the leaves and stems, in the center of which small, dusty, light-brown dots appear. *Uromyces pisi* usually appears in mid-spring when the crop is in the flowering stage (Barilli et al., 2014). Later, spots appear on which groups of black spores open, more numerous on the underside of the leaves. Infected stems develop faster in the season than uninfected stems, as a result of increased concentrations of growth hormones (Pilet, 1953). Heavily attacked plants dry out prematurely and show scaly grains in the pods.

The host. *Uromyces pisi* is a heterotic macrocyclic fungus that completes its life cycle on *Euphorbia cyparissias*. The host range of *U. pisi* is wide, being able to affect plant species belonging to other genera (*Euphorbia*, *Medicago, Pisum*, etc.) (Barilli et al., 2012). *Uromyces viciae-fabae* commonly called bean rust, is reported to be a fungus that infects peas in addition to beans (Cummins, 1978).
Uromyces viciae fabae is the main causal agent of pea rust in tropical and subtropical regions such as India and China, where warm and humid weather favors the development of the fungus (Kushwaha et al., 2006). Asciospores are the infective structures of *U. fabae* (Kushawaha et al., 2006) while in *U. pisi* uredospores are the infective spores (Barilli et al., 2009). In Romania, the main pathogen that causes pea rust is represented by *Uromyces pisi*.

Life cycle. *Uromyces pisi* (fam. Pucciniaceae, order Uredinales) is a heteroeous pathogen of rust, which carries out its life cycle on two host plant species. The sexual stages are completed on *Euphorbia cyparissias*, while the asexual life cycle stages are completed on leguminous crops such as *Lathyrus, Orobus, Pisum* and

Vicia spp. *Euphorbia cyparissias* is an erect, branched perennial that usually grows up to 30 cm height. It occurs on poor and mainly dry soils, along roadsides and forests. The incubation time of the fungus on *Euphorbia* lasts a full year. Under European conditions, the fungus remains dormant during the winter in the roots of *Euphorbia cyparissias* and develops with the host as spring occurs. Infected host plants develop earlier in the season and are inhibited from flowering. The host plant is induced by the fungus to form pseudo-flowers; yellow leaves that grow in a rosette at the top of the stems and resemble true flowers in color and shape (Pfunder & Roy, 2000). The fungus produces a sweet smelling nectar on the surface of the yellow leaves, giving the appearance of a real flower. Nectar contains fungal gametes (spermatia) that are transferred by nectar-feeding insects (bees and ants) from one type of fungal mating to another.

Primary air-borne inoculum. The fungus survives on plant residues and the dispersion of the inoculum can occur from several sources, namely: infested residues, dust and soil. The number of days with precipitation during the growing season plays an important role in the spread of the disease than other meteorological parameters (Martins et al., 2022).
Ascospores produced by *U. pisi* on *E. cyparissias* are dispersed by wind to infect pea crops. Jørstad (1948) observed rust on field pea 25 km from the nearest source (*Euphorbia cyparissias*) in Norway, suggesting that long-distance wind dispersal is possible. The asexual stage begins with the release of ascospores, which are dispersed by wind and infect pea crops. Infection with ascospores results in the production of uredinia and subsequently uredospores. The primary source of uredospores may be from pea plants, infected earlier in the growing season, or from spores carried long distances by wind. As the host plant matures, telia is produced, resulting in the formation of teleutospores.

Detection and Quantification. When infected *Euphorbia cyparissias* cannot flower, but instead is induced by the fungus to form pseudoflowers. Pfunder and Roy (2000) hypothesized that fungi depend on insect visitation to achieve gamete mating. Pseudoflowers induced by *Uromyces pisi* interact with uninfected true host flowers via insects during their co-"flowering" period in early spring. Field experiments were conducted to test whether the two species (*Pisum sativum* and *Euphorbia cyparissias*) share their insects and whether they were mutually influenced by insect visitation. Following the results, real flowers received more visits from insects (Pfunder & Roy, 2000). In his work, Barilli et al. (2012) studied the response of pea to seven species of rusts that can infect related legumes, and found that indeed pea can be infected mainly by 2 pathogens, namely *Uromyces pisi* followed by *Uromyces viciae fabae.* Other pathogens that can cause rusts, such as *Uromyces striatus*, *Uromyces ciceris-arietini*, *Uromyces anthyllidis* and *Uromyces vignae,* can also infect and reproduce on peas, although to a lesser extent. Knowledge about the host range of a biotrophic fungus like *Uromyces* is of great agronomic and epidemiological importance. In fact, one of the constraints shown by the species belonging to this genus is that several rust species can infect the same host plants e.g. *U. viciae-fabae* and *U. pisi* on peas (Barilli et al., 2009). Furthermore, it is possible for a rust-causing fungus to infect a plant species that was thought to be resistant e.g. *Medicago* spp. which was recently added to the host range of *Uromyces ciceris-arietini* (Stuteville et al., 2010). These characteristics prevent a clear characterization of pathogens and, consequentl, their control.

Management. Rusts can cause significant damage to the pea crop. Sidenko (1960) reported that early tillering influences high-level occurrence of the pathogen in the Ukrainian steppe, especially if the crop is in close proximity to alternative hosts. An outbreak of pea rust can lead to a reduction in production area for a short period of time as a result of increased production costs, which would make field peas less competitive compared to other crops. According to Plant Health Australia (2009) germplasm with improved resistance to this disease has been identified in the Australian field pea breeding program but has not yet been published. The decision to apply one or more fungicides depends on the risk of rust epidemic in a given

year. The rust epidemic is determined by the interaction of three important factors namely the host, the pathogen and the most important is the favorable environment for a certain period of time. Therefore, it is necessary to know the correlation between different meteorological parameters and the severity of rust. To prevent the spread of the disease, it is recommended to collect the remains of pea plants left in the field, as well as the *Euphorbia* plants around leguminous crops, to interrupt the biological cycle of the fungus. In case of strong attack, the pea plants will be treated with the following products Polyram combi 0.30%; Plantvax 75 wp 0.20% (Pârvu, 2010).

CONCLUSIONS

The pea crop is affected by numerous biotic and abiotic stressors. Fungal diseases such as rusts, downy mildew, ascochyta blight complex fall under the most widespread biotic stress. Rusts and downy mildews cause major crop damage in both tropical and temperate regions. Using fungicides to control plant diseases is a good approach, but excessive use of fungicides can cause environmental pollution and also lead to pathogen resistance. Therefore, to eliminate these constraints, we need to grow disease-resistant pea varieties. Their cultivation represents a safe and effective alternative method in controlling plant diseases.

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