Main Fungal Diseases of Cereals and Legumes in Tunisia

Bouzid NASRAOUI
Professor of Phytopathology
at Higher School of Agriculture of Kef

(With Expert System for Disease Identification on CD)

Centre de Publication Universitaire
2008

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INTRODUCTION

By their grain, hay, and straw productions, cereals and legumes (or pulses) in Tunisia and many other countries in the world, are the base of human and animal food. At the beginning of the 21st century, in our country where legumes and particularly cereal cultivated areas can practically not be increased, these field crops have to be highly intensified. Such intensification needs the use of high yielding varieties which require fertilizer, water, weed control and other cultural practices. Such conditions allow cereals and legumes to grow well, making a highly shaded and humid plant canopy, often favorable to diseases and insects. Among pests of these field crops, fungal diseases are very likely the most injurious. Their installation and propagation cause losses, sometimes very heavy, in both quantity and quality of the yield. Consequently, the control of fungal diseases is highly required in order to increase field crop production in a context of modern agriculture. But, the search and proposition of control methods need the identification and characterization of the responsible fungi on the basis of their morphology and biology and the recognition of the diseases on the basis of the symptoms that they cause on infected plants. For that, this book was conceived and elaborated to help in the identification of fungal diseases of cereals (wheat, barley, and oats) and legumes (faba bean, chickpea, and pea). In addition, possibilities of chemical control are proposed for each described disease. Other possible means of control are mentioned with a general view, in a chapter preceding the description of diseases. In the precedent chapters, characteristics of fungi with their main lines of classification (with examples of cereal and legume diseases only) as well as their development as phytopathogenic agents in host plants, were presented.

In this book, we have described the symptoms of all the diseases that we have observed over more than a decade as well as the morphology of their responsible fungal species observed by light microscopy (generally through 40x10 magnification, unless mentioned differently). All observations were illustrated by photos that we have exclusively taken in Tunisia, during several years of survey. However, this does not exclude the possibility of the existence of some other fungal diseases that we had not found. These field crop diseases described in Tunisia may not be very different from those existing in countries with a similar climate. This makes the present book useful, for example, in most Mediterranean countries.

Furthermore, it is important to mention that the fungal world which is wide and complex and the approaches of mycologists and phytopathologists often different, have lead to points of view of authors not always convergent, particularly, at the level of the terminology and the recent systematic of fungi. In this book, we have tried to bring together close opinions when they converge. But, when they diverge, we have generally favored the nomenclature as well as the classification of the fungi officially used by the *International Mycological Institute* (ex-*Commonwealth Mycological Institute*) serving as world reference of mycology.

THE WORLD OF FUNGI

The term **Mycology**, coming from the greek origin (***mykes***: fungus + ***logos***: discourse), means "**Science of Fungi**". In English, the term **fungus** (plural **fungi**) is used as it comes from the latin, without change.

Usually, the **fungus** term means "particular" plants cultivated or wild encountered in meadows and forests. They are generally composed of a "foot" surmounted by a "hat". Some of these fungi are consumed by man but others are mortal for him. In reality, this category of fungi makes only a portion of the fungal world which is macroscopic, because there are also microscopic fungi which are extremely numerous, various and widespread all over the world. Commonly, we know among them, those which make mold on abandoned food or dead organic matter.

Though man did know fungi before the antiquity and was close to them over thousands of years, it is only at the 17th century that he began to observe, to study, and to classify them helped by the invention of the microscope by **Van Leeuwenhoek**. **Micheli**, the Italian botanist, is considered as the founder of the science of mycology. In 1729, he published *Nova Plantarum Genera* which included one part for fungi. Since that time, knowledge in the fungal world has been increasing as new techniques of study developed. This achievement was made possible with the contribution of fine botanists and/or mycologists as **Linne**, **Persoon**, **Fries**, **Saccardo**... The number of fungal species discovered since that time, has become too high that, mycologists, nowadays, specialize in one family, or even in one genus, of fungi.

Presently, the number of fungal species in the world is estimated (as compared with other living organisms and by extrapolation) to 1.5 millions, from which only less than 10 % are described. Among those known species, nearly 10,000 are able to cause plant diseases and only about fifty are human pathogens and another around fifty are animal pathogens. The remaining fungi, generally, live either as saprobes or less frequently in association with other living organisms.

From the 17th century to 1960's, mycology has been considered as a part of botany. This was due to the ancient concept of the division of the world of living organisms into two kingdoms, eukaryotes: animals and plants, in addition to prokaryotes. But, in 1969, **Whittaker** proposed the division of the world of living organisms into five kingdoms. In this division, the fungi are considered as an independent kingdom of ***Fungi***, no more a part of the plant kingdom (***Plantae***), because fungi live by absorption and are **chemotroph** by obtaining their energy from organic matter and are **heterotroph** by using this organic matter as carbon source for the synthesis of their own organic compounds, while plants, which also live by absorption, are **phototroph** by using light energy and **autotroph** by synthesizing their organic compounds from atmospheric carbon dioxide and water dissolved mineral substances. The animal kingdom (***Animalia***) has been limited to animals living by ingestion. The unicellular organism world has been divided into two kingdoms: ***Monera*** containing prokaryotes (bacteria) and ***Protista*** including eukaryotes (protozoa and monocellular algae). During lately 1990's, on the basis of studies and techniques specially those which are molecular, the kingdom of *Fungi* has been, itself, divided again into two groups: the group of true-fungi forming the

kingdom of ***Fungi*** and the group of pseudo-fungi which includes fungi that were previously called “lower fungi” and which are now reclassified either in the kingdom of ***Protozoa*** or in a new kingdom called ***Chromista***.

Thus, the pseudo-fungi which were previously called “lower fungi”, are now divided in two very different groups (Fig. 1):
- One group of pseudo-fungi with morphology and biology close to those of amibae, makes part of the kingdom of *Protozoa*; this group is composed of four phyla,
- Another group of pseudo-fungi with many characteristics similar to those of true-fungi, but makes part of the kingdom of *Chromista*; this group is composed of three phyla.

The true-fungi, making previously the major part of the “higher fungi”, form actually the true kingdom of ***Fungi*** which is composed of four phyla, in addition to a large group called Anamorphic Fungi or Deuteromycetes (Figure 1).

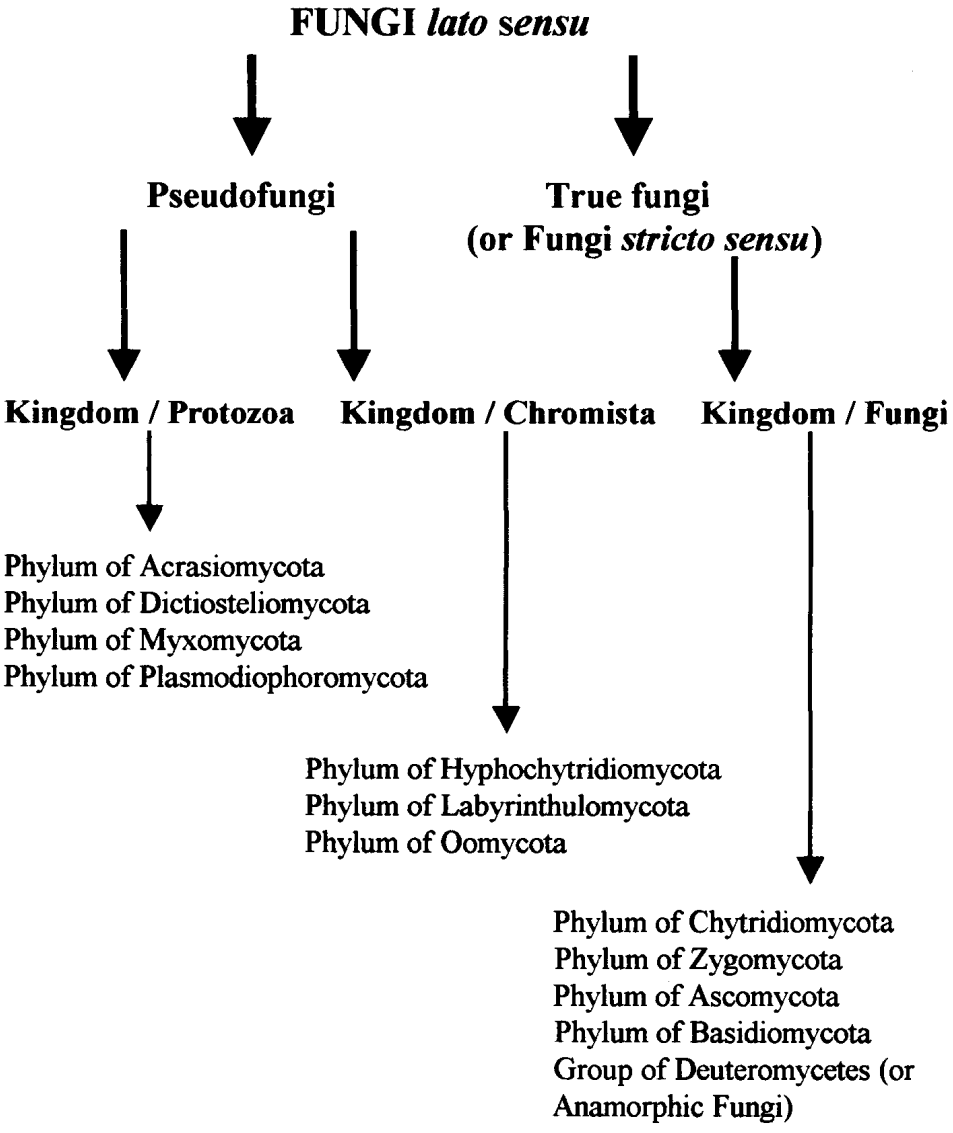

Figure 1: Main lines of the fungal world classification.

GENERAL CLASSIFICATION OF FUNGI

KINGDOM OF *PROTOZOA*

The kingdom of *Protozoa* contains, among others, pseudo-fungi which are unicellular micro-organisms, plasmodial or forming colonies, feed generally by phagocytose and without cell wall. These pseudo-fungi have been included first in fungi because of their similarities with regard to the sporal structures. Four phyla of pseudo-fungi are presently considered in the kingdom of *Protozoa* with the most important from a point of view phytopathology is that of ***Plasmodiophoromycota*** which includes the unique class of **Plasmodiophoromycetes**.

Phylum of *Plasmodiophoromycota*

Class of Plasmodiophoromycetes

Plasmodiophoromycetes are generally intracellular obligate parasites of plant underground organs. They can be vector of plant viruses. They make, within the host cells, polynucleic naked plasmodia which are neither mobile nor phagotrophic. These plasmodes produce conservation spores which release, after germination, biflagellate **zoospores** to move in liquid water. Only one order forms this class.

Order of Plasmodiophorales: This order contains two families.

Family of Plasmodiophoracae: This family is composed of numerous phytopathogenic species that belong to genera such as *Plasmodiophora*, *Polymyxa* and *Spongospora*. Example:

- *Polymyxa graminis*: cereal root parasite.

KINGDOM OF *CHROMISTA*

The kingdom of *Chromista* contains, among others, pseudo-fungi which are unicellular or coenocytic filamentous, with cellulosic wall. Three phyla of pseudo-fungi are now included in the kingdom of *Chromista*; phytopathogenic fungi exist in the phylum of ***Oomycota*** which contains one class only, class of **Oomycetes**.

Phylum of *Oomycota*

Class of Oomycetes

Oomycetes contain pseudo-fungi with morphology and nutrition by absorption similar to true-fungi, but are phylogenically distant.

In contrast with true-fungi, Oomycetes have mitochondria with tubular cristae, Golgi bodies formed by multiple flattened cisternae and dense-body vacuoles. Their cellular wall is mainly composed of cellulose, but rarely of chitin, which is, however, known to be the principal component of the wall of true-fungi. The existence of hydroxyproline in the wall of those pseudo-fungi constitutes equally a point of difference with true-fungi. Meiosis of Oomycetes takes place within the forming gametangia, which makes Oomycetes diploid during their all vegetative cycle while true-fungi are haploid, diploid and/or dikaryotic during a part or all their life cycle. Moreover, the most important synthesized sterol in Oomycetes is fucosterol and not ergosterol as in the case of true-fungi.

The thallus of Oomycetes is either unicellular or coenocytic filamentous. Asexual reproduction of Oomycetes occurs through the production of biflagellate **zoospores** released from **sporangia** but sporangia can also function as **conidia** and germinate directly. For sexual reproduction, almost all Oomycetes species are heterogametangic. Male and female gametangia are respectively called **antheridium** and **oogonium**. Following fertilization of the oogonium by the antheridium and meiosis, **oospore** is produced with a thick wall which makes it resistant to unfavorable conditions. The class of Oomycetes contains nine orders.

Order of Peronosporales: This order contains two families.

Family of Peronosporacae: Numerous species of this family are plant parasites and belong to the genera *Bremia, Peronospora, Plasmopara* and *Pseudoperonospora.* Examples:

- *Peronospora pisi*: causing downy mildew of pea,
- *Peronospora viciae*: causing downy mildew of faba bean.

Order of Pythiales: This order contains two families.

Family of Pythiacae: This family includes many species that are plant pathogens and belong to the genera *Phytophthora* and *Pythium*. Example:

- *Pythium graminicola*: cereal damping off agent.

Order of Saprolegniales: Two families exist in this order.

Family of Saprolegniacae: In this family, the most important genus is *Aphanomyces*. Example:

- *Aphanomyces euteiches*: causing root rot of pea.

Order of Sclerosporales: This order includes two families.

Family of Sclerosporacae: This family contains some phytopathogenic parasites which belong to the genera *Peronosclerospora* and *Sclerospora*. Examples:

- *Sclerospora graminicola*: graminae parasite,

Family of Verrucalvacae: Some phytopathogenic parasites exist in this family and belong to the genus *Sclerophthora*. Example:
- *Sclerophthora macrospora*: causing downy mildew of cereals.

KINGDOM OF *FUNGI*

The kingdom of *Fungi* contains the true-fungi. These organisms feed by absorption, do not form plasmodia, have their walls formed chiefly by chitin and glucans, have their mitochondria with flattened cristae, and have a generally short diploid phase. Four phyla are now considered in the kingdom of *Fungi* (in addition to the group of Anamorphiques Fungi): ***Ascomycota***, ***Basidiomycota***, ***Chytridiomycota*** and ***Zygomycota***. **Anamorphiques Fungi** (or **Deuteromycetes**) are not considered as a phylum or a class. They are a part of *Ascomycota* or *Basidiomycota*.

Phylum of *Chytridiomycota*

Class of Chytridiomycetes

Chytridiomycota (including the unique class of Chytridiomycetes) are the only true-fungi producing mobile cells which are generally uniflagellate zoospores. The thallus is unicellular or coenocytic filamentous. For their asexual reproduction, Chytridiomycetes produce **zoospores** which are released from **sporangia**. The sexual reproduction involves the union of two zoospore gametes called **planogametes**. Chytridiomycetes are divided into five orders.

Order of Chytridiales: This order includes four families.
Family of Synchytriaceae: The family of Synchytriaceae contains plant pathogens belonging to the genus *Synchytrium*. Example:
- *Synchytrium phaseoli*: parasite of legumes.

Order of Spizellomycetales: Three families exist in this order.
Family of Olpidiaceae: The most important genus in this family is *Olpidium*.
Examples:
- *Olpidium viciae*: causing warty scab of faba bean.

Phylum of *Zygomycota*

Zygomycota are characterized by the absence of mobile cells and by a thallus which is generally coenocytic filamentous. The asexual reproduction leads to the production of **sporangiospores** and rarely **conidia**. The sexual reproduction is generally realized by isogametangy. This phylum contains two classes: the class of **Zygomycetes** containing some phytopathogenic species and the class of **Trichomycetes**.

Phylum of *Ascomycota*

Ascomycota is the widest group of fungi. They are recognized by the production of **asci** which are microscopic sacks containing spores called **ascospores**. For long time, *Ascomycota* have been divided into six classes: **Hemiascomycetes**, **Plectomycetes**, **Pyrenomycetes**, **Discomycetes**, **Loculoascomycetes** and **Laboulbeniomycetes**. This classification based on the absence or presence and the nature of the sporal structure containing the asci (called **ascocarp** or **ascoma**) is presently abandoned because the phylogenic approach has shown that some of theses classes are in reality heterogeneous groups. In this book, simplified and practical systematic have been synthesized from several references. It is based on the use of the "class" term for homogenous pools of species and the "group" term for heterogenous pools of species. Hence, we propose the following height classes/groups: **Saccharomycetes**, **Taphrinomycetes**, **Erysiphomycetes**, **Plectomycetes**, **Pyrenomycetes**, **Discomycetes**, **Loculoascomycetes**, and **Laboulbeniomycetes**. Only the latters do not contain phytopathogens.

The thallus of *Ascomycota* is unicellular or septate filamentous. Asexual reproduction leads to the production of multitude kinds of **conidia** in the anamorphs (asexual stages). The sexual reproduction takes place rarely by isogametangy and generally by heterogametangy. In the latter case, the male structure (the **antheridium**) pours its content in the female structure (the **ascogonium**) via the **trichogyne**. Often, the delay of the karyogamy with regard to plasmogamy, generates a dikaryotic phase characterized by cells with two nuclei (male and female). The last stage is the production of **asci** containing **ascospores**.

To be practical and rigorous at the same time as phytopathologists, it is possible to continue considering the absence or presence and the nature of ascomata, but with a special mention to homogeneity (class) or heterogeneity (group) of the considered pool. Hence, five large pools of *Ascomycota* individualize with regard to their sporal structures:
- **Naked asci**: *Ascomycota* with no ascoma; asci develop directly on the thallus (classes of **Saccharomycetes** and **Taphrinomycetes)**,
- **Cleistothecium**: Asci in completely closed ascoma that open at maturity by dehiscence (class of **Erysiphomycetes**) or by bursting (group of **Plectomycetes**),
- **Perithecium**: Asci in more or less closed ascoma opening at maturity by ostiole (group of **Pyrenomycetes**),
- **Apothecium**: Asci in ascoma that open at maturity as a cup (group of **Discomycetes**),

- **Pseudothecium**: Asci produced directly in locules, without wall, formed in the ascoma (group of **Loculoascomycetes**).

Class of Erysiphomycetes

The class of Erysiphomycetes is characterized by cleistothecia opening at maturity by dehiscence.

Order of Erysiphales: This order contains only one family.
Family of Erysiphacae: Almost all species of this family are responsible of powdery mildew disease and belong mainly to the genera *Blumeria, Erysiphe*, *Leveillula*, *Microsphaera*, *Phyllactinia*, *Podosphaera*, *Sphaerotheca* and *Uncinula*. Anamorphs of Erysiphacae are **Hyphomycetes** and belong mainly to the genera *Oidiopsis*, *Oidium* and *Ovulariopsis*. Examples:
- *Blumeria graminis* (anamorph: *Oidium monilioides*): cereal powdery mildew agent.
- *Erysiphe pisi* (anamorph: *Oidium* sp.): causing powdery mildew of pea,
- *Erysiphe polygoni* (anamorph: *Oidium* sp): causing powdery mildew of numerous plant species including legumes,
- *Leveillula taurica* (anamorph: *Oidiopsis taurica*): causing powdery mildew of numerous plant species including legumes.

Group of Pyrenomycetes

The ascoma of Pyrenomycetes is generally perithecial type.

Order of Diaporthales: The anamorphs related to this order are various.
Family of Magnaportacae: Species of this family are generally root parasite and belong to *Gaeumannomyces* and *Magnaporthe* genera. Examples:
- *Gaeumannomyces graminis* var. *avenae*: causing take-all of oats,
- *Gaeumannomyces graminis* var. *graminis*: causing take-all of cereals,
- *Gaeumannomyces graminis* var. *tritici*: causing take-all of wheat.

Order of Hypocreales: Anamorphs in this order are generally **Hyphomycetes**.
Family of Clavicipitacae: The most important plant parasite species of this family belong to *Claviceps* and *Epichloë* genera. Anamorphs are **Hyphomycetes** such as *Acremonium* and *Sphacelia.* Example:
- *Epichloë typhina* (anamorph: *Sphacelia typhinum*): causing cat tail of graminae.
Family of Hypocreacae: Plant parasite species of this family belong mainly to the genera *Gibberella* and *Nectria*. Their anamorphs are **Hyphomycetes** belonging to genera like *Cylindrocarpon*, *Fusarium*, and *Tubercularia*. Examples:
- *Gibberella avenacea* (anamorph: *Fusarium avenaceum*): causing damping off to cereals,
- *Gibberella fujikuroi* (anamorph: *Fusarium moniliforme*): parasite of cereals,
- *Gibberella intricans* (anamorph: *Fusarium equiseti*): parasite of cereal seedlings,
- *Gibberella zeae* (anamorph: *Fusarium graminearum*): parasite of gramineae,
- *Nectria haematococca* (anamorph: *Fusarium solani*): causing root rot of numerous plant species, including cereals and legumes.

Order of Phyllacorales: Anamorphs of the Phyllacorales are usually **Coelomycetes**.

Family of Phyllacoracae: Plant parasite species of this family belong to *Glomerella* and *Phyllachora* genera. Example:
- *Phyllachora graminis*: graminae parasite.

Group of Discomycetes

The Discomycetes produce ascomata that open at maturity as cup-like form.

Order of Heliotales: The anamorphs in thie order are often **Hyphomycetes** or **Coelomycetes**.

Family of Dermateacae: Plant parasite species of this family belong mainly to the genera *Diplocarpon*, *Mollesia*, and *Pseudopeziza*. Their anamorphs are various like *Marsoninna*, *Pseudocercosporella,* and *Cylindrosporium*. Example:
- *Mollesia yallundae* (anamorph: *Pseudocercosporella herpotrichoides*): causing cereal eyespot.

Family of Sclerotiniacae: Plant parasite species of this family belong to *Botryotinia*, *Gloeotinia*, *Monilinia*, and *Sclerotinia* genera. The anamorphs are **Agonomycetes** and **Hyphomycetes** belonging to the genera *Botrytis*, *Endoconidium*, *Monilia*, and *Sclerotium.* Examples:
- *Botryotinia fabae* (anamorph: *Botrytis fabae*): causing chocolate spot of faba bean,
- *Gloeotinia granigena*: graminae seed parasite.

Group of Loculoascomycetes

The group of Loculoascomycetes produces pseudothecial ascomata.

Order of Mycosphaerellales: Anamorphs of this order are various.

Family of Mycosphaerellacae: This family contains numerous plant parasite species that belong mainly to the genus *Mycosphaerella*. Their anamorphs are in the genera *Ascochyta*, *Cercoseptoria*, *Cercospora*, *Cercosporella*, *Cladosporium*, *Paracercospora*, *Ramularia*, *Septoria*,... Examples:
- *Mycosphaerella graminicola* (anamorph: *Septoria tritici*): causing leaf blotch of wheat,
- *Mycosphaerella pinodes* (anamorph: *Ascochyta pinodes*): causing blight of pea.

Order of Pleosporales: In this order, anamorphs are various.

Family of Leptosphaeriaceae: The family of Leptosphaeriaceae includes plant pathogens belonging mainly to the genus *Leptosphaeria*. Their anamorphs are **Coelomycetes** in the genera *Coniothyrium*, *Phoma, Septoria*, and *Stagonospora*. Examples:
- *Leptosphaeria avenaria* (anamorph: *Septoria avenae*): causing speckled blotch of oats,
- *Phaeosphaeria nodorum* (anamorph: *Stagonospora nodorum*): causing glum blotch of wheat.

Family of Pleosporacae: This family contains numerous plant parasite species which belong to the genera *Cochliobolus*, *Didymella*, *Pleospora*, and *Pyrenophora*. Their anamorphs are in the genera *Ascochyta*, *Bipolaris*, *Drechslera Phoma*, and *Stemphylium*. Examples:
- *Cochliobolus sativus* (anamorph: *Bipolaris sorokiniana*): causing spot blotch of cereals,
- *Cochliobolus victoriae* (anamorph: *Bipolaris victoriae*): causing foot rot of cereals,
- *Didymella fabae* (anamorph: *Ascochyta fabae*): causing blight of faba bean,

- *Didymella rabiei* (anamorph: *Ascochyta rabiei*): causing blight of chickpea,
- *Pyrenophora chaetomioides* (anamorph: *Drechslera avenacea*): causing leaf blotch of oats,
- *Pyrenophora graminea* (anamorph: *Drechslera graminea*): causing stripe of barley,
- *Pyrenophora teres* (anamorph: *Drechslera teres*): causing net blotch of barley,
- *Pyrenophora tritici-repentis* (anamorph: *Drechslera tritici-repentis*): causing tan (or yellow leaf) spot of wheat.

Phylum of *Basidiomycota*

Basidiomycota form a wide group of fungi which most of them are usually encountered in meadows or forest and are formed by a "foot" surmounted by a "hat". *Basidiomycota* are characterized by the formation of **basidia** producing on the outside spores called **basidiospores**. Basidia may be carried by a sporal structure called **basidioma** (or **basidiocarp**). Basidioma does not exist in some *Basidiomycota* as in the case of fungi responsible of smut and rust diseases. *Basidiomycota* have been divided into three classes based on the presence or absence and the nature of the septation of the basidium, the formation or not of teliospores and the mode of basidiospore germination. These classes are **Holobasidiomycetes**, **Phragmobasidiomycetes** and **Teliomycetes**. Moreover, *Basidiomycota* have been divided into two large heterogeneous Groups: **Hymenomycetes** with basidia formed in particular layers called **hymenium** and **Gasteromycetes** without hymenium. The above classifications were abandoned and replaced by another more pragmatic classification which allows dividing *Basidiomycota* in three classes: **Basidiomycetes**, **Uredinomycetes** and **Ustilaginomycetes** depending on the absence or presence and the nature of the basidiomata as well as on the mode and the cycle of the life.

The thallus of *Basidiomycota* is generally septate filamentous. The asexual reproduction is realized by fragmentation, fission or budding leading to the production of **conidia**. The conidial production is very important in smut and rust fungi and these conidia are called **urediospores** in the case of rust fungi. The sexual reproduction culminates by the production of **basidia** releasing **basidiospores**. It starts after germination of basidiospores by cytoplasmic fusion (plasmogamy) of a **spermatium** with a **receptive hypha** or of **two hyphae**. The karyogamy does not generally occur immediately leaving the thallus dikaryotic until meiosis, just before the formation of the new basidia. Basidium stage is preceded by a **teliospore** stage in the case of rust and smut fungi.

Class of Basidiomycetes

The Basidiomycetes (sensu stricto) form a large class in which a minority is phytopathogen.

Order of Ceratobasidiales: The Ceratobasidiale order contain the unique family of Ceratobasidiacae.

Family of Ceratobasidiacae: The family of Ceratobasidiacae includes some phytopathogenic species which belong to the genera *Ceratobasidium* and *Thanatephorus*. The anamorphs are **Agonomycetes** in the genus *Rhizoctonia*. Example:
- *Ceratobasidium cereale* (anamorph: *Rhizoctonia cerealis*): parasite of cereals.

Class of Uredinomycetes

This class contains the responsible of plant rusts.

Order of Uredinales: This order is composed of 14 families. It is the order to which belong plant rust fungi.

Family of Pucciniacae: This family includes plant pathogens belonging mainly to the genera *Gymnosporangium, Hemileia, Puccinia*, and *Uromyces*. Examples:
- *Puccinia coronata*: causing crown rust of oats (stages 0 and I on *Rhamnus*),
- *Puccinia graminis*: causing stem (or black) rust of wheat (stages 0 and I on *Berberis*),
- *Puccinia hordei*: causing leaf (or brown) rust of barley (stages 0 and I on *Ornithogalum*),
- *Puccinia recondita*: causing leaf (or brown) rust of wheat (stages 0 and I on *Anchusa*),
- *Puccinia striiformis*: causing yellow (or stripe) rust of wheat,
- *Uromyces ciceris-arietini*: causing rust of chickpea,
- *Uromyces pisi-sativi*: causing rust of pea (stages 0 and I on *Euphorbia*),
- *Uromyces viciae-fabae*: causing rust of faba bean.

Class of Ustilaginomycetes

It is the class that includes agents of smut diseases.

Order of Tilletiales: The Tilletiale order contains species that cause mainly a kind of smut disease called bunt.

Family of Tilletiacae: This family contains many phytopathogenic species belonging to the genus *Tilletia*. Examples:
- *Tilletia controversa*: causing dwarf bunt of cereals,
- *Tilletia indica*: causing Karnal bunt of wheat,
- *Tilletia laevis*: causing common bunt of wheat,
- *Tilletia tritici*: causing common bunt of wheat.

Order of Urocystales: The Urocystale order contains agents of foliar smuts.

Family of Urocystacae: The family of Urocystacae includes phytopathogens which generally belong to the genus *Urocystis*. Example:
- *Urocystis agropyri*: causing foliar smut of cereals.

Order of Ustilaginales: The Ustilaginale order contains several species responsible of smut diseases.

Family of Ustilaginacae: The Ustilaginaceae family includes many plant pathogenic species belonging mainly to the genera *Moesziomyces, Sporisorium*, and *Ustilago*. Examples:
- *Ustilago hypodytes*: causing smut of gramineae,
- *Ustilago segetum*: causing covered smut of barley and oats,

- *Ustilago segetum* var. *avenae*: causing loose smut of oats,
- *Ustilago segetum* var. *nuda*: causing loose smut of barley,
- *Ustilago segetum* var. *tritici:* causing loose smut of wheat,
- *Ustilago striiformis*: causing stripe smut of gramineae.

Anamorphic Fungi

The relatively new term of Anamorphic (or Mitosporic) Fungi is attributed to fungi which have been for a long time called *Imperfect Fungi*, *Deuteromycotina*, Deuteromycetes and before that Adelomycetes. They are true-fungi *Ascomycota* and *Basidiomycota* which are in asexual stage (anamorph). Many of them have a known sexual reproduction, others not. Anamorphic Fungi which are the anamorphs in majority of *Ascomycota* and to a less degree of *Basidiomycota*, forms a large heterogeneous group of fungi whose classification used for a long time is artificial. They were considered in the past as a subphylum of *Deuteromycotina* divided into three classes: **Agonomycetes**, **Coelomycetes** and **Hyphomycetes**. The phylogenic approach having confirmed the heterogeneity of these fungi, the term *Deuteromycotina* was abandoned and replaced by the appellation of Anamorphic Fungi or Deuteromycetes. Arbitrarily, the anamorphs of yeast (*Ascomycota*) and of the agents of smuts and rusts (*Basidiomycota*) are not considered in Anamorphic Fungi. For the previously indicated classes, we prefer to use the term of “group” which is more general than the term “class” which concerns a homogenous pool.

The thallus of Anamorphic Fungi is generally septate filamentous. Their sexual reproduction was treated previously in *Ascomycota* and *Basidiomycota* chapters. Their asexual reproduction is a frequent phenomenon through which the concerned fungus actively reproduces and quickly disseminates as long as the environment conditions are favorable. Spores produced by asexual reproduction are called **conidia**. They come out directly from the pre-existent thallus or via **conidiogenous cells** brought or not by **conidiophores**. Those conidiophores with their conidia can be free or grouped on/in conidifere structures called **conidiomata**. In some cases, conidioma, as a conceptacle, wraps the conidiophores inside: **pycnidium**. In other cases, the conidiophores are grouped on the surface of the conidioma: **synnema**, **sporodochium** or **acervulus**.

Group of Agonomycetes

The Agonomycetes are characterized by the absence of conidia. They propagate only by **vegetative multiplication**. The most important genera are *Rhizoctonia* and *Sclerotium*.

Subgroup of Agonomycetales: The Agonomycetales contain phytopathogens of the genera *Rhizoctonia* and *Sclerotium* which are anamorphs of *Ascomycota* or *Basidiomycota*. Example:
- *Rhizoctonia cerealis* (teleomorph: *Ceratobasidium cerealis*): parasite of cereals.

Group of Hyphomycetes

The Hyphomycetes produce conidia on conidiophores which are either **free** or grouped in **synnemata** or in **sporodochia**.

Subgroup of Hyphomycetales: The Hyphomycetales, also called Hyphales or Moniliales, form the most important part of the Hyphomycetes. They are characterized by separated and non organized conidiophores. Many species are phytopathogens. Examples:
- *Acremonium typhinum* (teleomorph: *Epichloë typhina*): causing cat tail of gramineae,
- *Alternaria alternata*: parasite of many plant species including legumes,
- *Bipolaris sorokiniana* (teleomorph: *Cochliobolus sativus*): causing spot blotch of cereals,
- *Bipolaris victoriae* (teleomorph: *Cochliobolus victoriae*): causing foot rot of cereals,
- *Botrytis fabae* (teleomorph: *Botryotinia fabae*): causing chocolate spot of faba bean,
- *Cladosporium herbarum*: weakly parasite of cereals,
- *Drechslera avenacea* (teleomorph: *Pyrenophora chaetomioides*): causing leaf blotch of oats,
- *Drechslera graminea* (teleomorph: *Pyrenophora graminea*): causing stripe of barley,
- *Drechslera teres* (teleomorph: *Pyrenophora teres*): causing net blotch of barley,
- *Drechslera tritici-repentis* (teleomorph: *Pyrenophora tritici-repentis*): causing tan (or yellow leaf) spot of wheat,
- *Oidium monilioides* (teleomorph: *Blumeria graminis*): causing powdery mildew of cereals,
- *Pseudocercosporella herpotrichoides* (teleomorph: *Mollisia yallundae*): causing eyespot of cereals,
- *Rhynchosporium secalis*: causing scald of barley,
- *Verticillium alboatrum*: causing wilt of numerous plant species including legumes,
- *Verticillium dahliae*: causing wilt of many plant species including legumes.

Subgroup of Tubercularíales: The tubercularíales are characterized by conidiophores organized in sporodochia. Many of them are phytopathogenic species. Examples:
- *Fusarium avenaceum* (teleomorph: *Gibberella avenacea*): causing damping off to cereals and legumes,
- *Fusarium culmorum*: causing foot rot of many plant species,
- *Fusarium equiseti* (teleomorph: *Gibberella intricans*): parasite of cereal seedlings,
- *Fusarium graminearum* (teleomorph: *Gibberella zeae*): parasite of gramineae,
- *Fusarium moniliforme* (teleomorph: *Gibberella fujikuroi*): parasite of cereals,
- *Fusarium oxysporum*: vascular parasite of a large number of plant species including legumes,
- *Fusarium solani* (teleomorph: *Nectria haematococca*): causing root disease of numerous plant species including cereals and legumes.

Group of Coelomycetes

The Coelomycetes produced conidia on conidiophores which are grouped in **acervuli** or wrapped inside **pycnidia**.

Subgroup of Sphaeropsidales: The Sphaeropsidales, also called Phomales, are characterized by the production of conidia and conidiophores wrapped inside pycnidia. Many species are phytopathogenic. Examples:
- *Ascochyta avenae*: causing leaf spot of oats,
- *Ascochyta fabae* (teleomorph: *Didymella fabae*): causing blight of faba bean,
- *Ascochyta hordei*: causing leaf spot of barley,
- *Ascochyta pinodes* (teleomorph: *Mycosphaerella pinodes*): causing blight of pea,
- *Ascochyta pisi*: causing (yellow) spot of pea,
- *Ascochyta rabiei* (teleomorph: *Didymella rabiei*): causing blight of chickpea,
- *Ascochyta tritici*: causing leaf spot of wheat,
- *Phoma pinodella* (teleomorph: *Mycosphaerella*-like): causing black stem of pea,
- *Septoria avenae* (teleomorph: *Leptosphaeria avenaria*): causing speckled blotch of oats,
- *Septoria tritici* (teleomorph: *Mycosphaerella graminicola*) causing leaf blotch of wheat,
- *Stagonospora nodorum* (teleomorph: *Phaeosphaeria nodorum*): causing glum blotch of wheat.

FUNGAL DISEASE DEVELOPMENT

THE DISEASE TRIANGLE

For a plant disease to occur, three components are needed: plant and pathogen must come into contact and interact, and environment conditions must be favorable. However, each of these three components can change considerably leading to different degrees of disease severity for an individual plant and within a plant population. The host plant may be more or less resistant, Susceptible, young, old,... The pathogen may be more or less virulent, active, dormant,... The environment conditions may affect more or less the growth, susceptibility, resistance of the host plant and the growth, multiplication, virulence degree, dispersal of the pathogen,... The interaction of host plant, pathogen, and environment is generally considered as forming a triangle called “disease triangle”. Each side of this triangle represents one of the three components. The length of one side is proportional to its involvement in the severity of the disease. When the three components of the disease triangle are quantified, the area of this triangle would represent the amount of the disease in a plant or in a plant population. If any of the three components is zero, there is no disease.

STAGES OF THE DISEASE DEVELOPMENT

A series of more or less distinct events occurs successively leading to the development of the disease. This chain of events involves the changes in the plant and in the pathogen within a growing season and from one growing season to another. Those events are inoculation, penetration, infection, dissemination, and survival of the pathogen.

Inoculation

Inoculation happens when a pathogen comes into contact with a plant. Fungal pathogen propagules (spores, sclerotia, mycelial fragments,...) that land on the plant are called **inoculum**. A surviving inoculum causes a primary infection. The inoculum produced from the primary infection causes the secondary infections. The inoculum is generally found in the host plant debris, in the soil, in/on the seeds and other propagate organs,... It can also survive on weeds or alternate host plants. It can be carried from nearby plants or fields or from fields hundred kilometers away. In most cases, pathogen propagules are carried passively, generally by air, water, and insects.

Penetration

Mycelial fragment or germinating spore penetrates plant surfaces directly, through natural openings or through wounds.

Direct penetration is performed by most of fungi. It is sometimes realized by a fine hypha produced directly by the spore or the mycelium, or usually by a penetration

peg formed by an appressorium. The fine hypha or the penetration peg pierces the cuticle and the plant cell wall through mechanical force associated to an enzymatic softening.

Some fungi get into plants through natural openings such as stomata, hydathodes, and lenticels. Others can do it through a variety of wounds which may be fresh or old and may consist of a lacerated or killed tissue. These pathogens may grow briefly on such tissue before they attack healthy tissue.

Infection

Infection begins when the pathogen establishes in susceptible cells or tissues of the host plant and procures nutrients from them. Then, pathogen grows and/or multiplies so that it invades and colonizes the plant more or less quickly. When the infection succeeds, symptoms which are the visible changes due to the disease, appear. Symptoms change continuously, more or less quickly, from their apparition to the death of the entire plant. The time between inoculation and symptom apparition is called **incubation period**. During infection, some fungal pathogens, called **necrotrophs**, kill cells and utilize their contents, others, called **biotrophs**, obtain their nutrient from living cells, without killing them. Many substances such as enzymes and toxins are released by the pathogens within the host plants. These substances affect the structural integrity and the physiological processes of the host cells. In order to react against pathogens, host plants develop a variety of defense mechanisms, resulting in different degrees of protection from susceptibility to resistance. Susceptible plant variety corresponds to a virulent pathogen and resistant variety corresponds to an avirulent pathogen.

Dissemination

For some fungal species, spores are forcibly expelled and then carried away by wind, while most of fungi have their spores passively carried by different vectors.

Many fungal spores are disseminated by air currents that carry them to various distances, depending on turbulence and velocity of the air. Thus, some spores can be carried few hundreds or thousands meters while others several kilometers or even hundreds of kilometers.

Water is also an important disseminating factor of spores and mycelial fragments. These fungal propagules being in the soil can be carried by rain fall or irrigation water moving on the soil surface. When spores are on the surface of plants, they can be splashed in all directions by rain fall or sprinkler irrigation water. They can also be washed downward by rain fall and sprinkler irrigation drops when they are suspended in the air.

Many fungi are present in/on seeds and plant propagation organs and are then disseminated by them. Animals, especially insects, are also vectors that disseminate fungal propagules. Thus, most of fungal pathogens adhere to the body of animals moving among plants and from plant to plant. Fungi are also disseminated by humans through direct contact and tools used in crop production.

Survival

To overcome the hard season (winter or summer) when field is free of host plants, pathogens persist using mainly spores, in addition to mycelial fragments and sclerotia. They survive in soil, infected plant debris, seeds,... Dissemination spores (such as conidia) survive some weeks or months while conservation spores (such as oospores, chlamydospores, teliospores,...) may survive several years. Dissemination spores are actively produced by fungi during favorable season to propagate the disease while conservation spores are produced by fungi to overcome the unfavorable season.

CONTROL OF FUNGAL DISEASES

The development of methods to combat plant fungal diseases enhances the quantity and improves the quality of plant products. Methods of control vary considerably from one disease to another depending on the pathogen, the host plant, their interaction and the environmental conditions. Besides quarantines, methods of control can be cultural, genetic, biological, physical and chemical. The integration of all these methods leads to integrated management.

QUARANTINES

No disease develops if plants and pathogens are kept apart. To prevent the introduction and propagation of plant pathogens into countries where they are absent, laws were made to regulate the transfer of crops which may be grown and distributed between countries. Such regulation control is implemented through quarantine services. Thus, to keep out foreign plant pathogens and protect the country, it is necessary to prohibit the entry of plants, plant products and soil unless they are certified as disease-free material. Suspected materials may be kept in quarantines for observation and analysis.

CULTURAL CONTROL METHODS

Cultural methods of disease control mean all cultural actions of farmer that create conditions unfavorable to pathogens but favorable to plants. Among those actions, the destruction (removal or burning) of volunteer plants and alternative hosts that may behave as pathogen reservoirs during crop-free season, is an important action that reduces the amount of inoculum. The breaking of the life cycle of some pathogens is possible by eradicating their alternate hosts as it could be in the case of rusts.

Soilborne pathogens or infected host plant debris in the soil can be reduced by planting culture species with a crop rotation of three to four years. A satisfactory control is then possible with an appropriate crop rotation, especially against pathogens specific to a given host plant.

Some other practices are also used to reduce the amount of inoculum. Thus, the deep turn plowing of infected host plant debris after harvest, allows to burry the inoculum in the sol and to destroy it. Moreover, by plowing during summer, the high soil temperature due to the solar heat, inactivates many soilborne fungi and consequently reduces the amount of inoculum.

Other cultural methods consist in the avoiding of pathogens by plants. Such activities include the use of pathogen-free seeds, the choice of proper planting dates and sites, the application of a well-balanced fertilizer program, the plantation of wind break, the use of well-drained soils, the application of an appropriate insect and weed control,... Also, by disinfecting bags, containers, walls of storage houses and other tools, the amount of inoculum and subsequent infections may be reduced considerably. For

example, a presumed infested field must be plowed, treated and harvested after a pathogen-free field in order to ovoid the inoculum propagation by the heavy machinery.

BIOLOGICAL CONTROL METHODS

Pathogens can be biologically controlled by using antagonistic micro-organisms which normally exist in nature. These micro-organisms can destroy totally or partially pathogen populations. Their mode of action may be through direct parasitism, competition for food or toxic effect. To enhance the effectiveness of antagonistic micro-organisms, humans try to introduce new populations of such micro-organisms in the environment and/or add amendments to soil that stimulate the growth of existing antagonists.

GENETIC CONTROL METHODS

To induce or improve resistance of host plants to many pathogens, the genetic resistance has been introduced and increased through breeding of resistant varieties. In addition to the conventional plant breeding method used for a long time, the genetic engineering technology began to be more and more used. This technology makes possible the isolation of individual resistance genes from resistant plant species and their transfer into susceptible other species of plants in which they induce the resistance.

The use of resistant varieties is one of the most effective mean of controlling plant diseases in crops. It is also an easy, safe and relatively not expensive way of control. The cultivation of resistant varieties eliminates losses due to diseases and allows to ovoid the other methods of control which are generally expensive and polluting. It is always recommended to use the varieties that have both vertical and horizontal resistance. One to few (2 or 3) major genes control vertical resistance (which is easy to be broken down by new pathogen physiological races), while numerous minor genes control horizontal resistance. Thus, as the new pathogen physiological races develop and become widespread, the resistance of old varieties is broken down. These varieties, especially with vertical resistance, have to be replaced periodically, for example, about every ten years. For that, it is expected that genetic engineering technology helps the conventional breeding by the offer of a quick transfer of resistance genes into susceptible varieties, and consequently, by the reduction of the time required to develop a new resistant varieties compared to the only conventional breeding.

PHYSICAL CONTROL METHODS

Several physical agents can be used to control plant diseases. Those agents are temperature (high and low), dry air, unfavorable light wavelengths, radiation,…

For cereals, to control loose smut disease, seeds are soaked in hot water at 52 °C for 11 min.

CHEMICAL CONTROL METHODS

Methods of application

Fungicides are applied as sprays or dusts on plants to control fungal diseases. Numerous fungicides have to be present on the surface of plants prior to pathogens arrived in order to prevent infection (preventive control). These fungicides generally of contact are called protectant. They inhibit spore germination and kill germinating spores. Other relatively more recent fungicides are eradicant. They are partially or totally systemic because they are absorbed by plants and translocated internally toward the whole areas, mainly leaves, where they act directly against pathogens invading plant tissues (curative control). Nowadays, because of their effectiveness, persistent activity and reduced number of required treatments, the systemic fungicides are taking over many contact fungicides. Their disadvantage is however the possibility to develop resistant fungal strains. Fungicides are also used to treat seeds and soil.

Mechanisms of action

The majority of used fungicides are directly toxic to pathogens either as protectant at the entry points of pathogens or as eradicant systemic chemicals translocated into plants. They act against pathogens by solving or damaging cell membranes, by inhibiting the synthesis of certain cell wall substances, by complexing and inactivating some essential coenzymes,... For example, sulfur interferes with electron transport along cytochrome system and, consequently, deprives the cell of energy. Dithiocarbamates and ethazol inactivate -SH groups of proteins and enzymes. Chlorinated aromatic and heterocyclic compounds such as pentachloronitrobenzene, chlorothalonil, chloroneb, captan and vinclozolin react with $-NH_2$ and -SH groups of enzymes. Oxanthiins inhibit succinic dehydrogenase activity which is essential in mitochondrial respiration. Benzimidazoles interfere with nuclear division. Kitazin and edifenphos inhibit chitin synthesis. Bitertanol, fenapanil, imazalil, prochloraz, triadimefon, triadimenol, triforine and etaconazole are sterol inhibitors because they inhibit biosynthesis of ergosterol which plays a crucial role in the structure and function of fungal cell membranes.

Resistance to fungicides

For a long time, protectant fungicides such as thiram, maneb or captan were used and no resistant fungal strains were observed. This is presumably because these fungicides affect several vital processes of the pathogen and lots of gene changes would be necessary to produce a resistant strain. However, since the 1960s, resistant fungal strains against fungicides began to be observed. The apparition of such resistant strains, like in *Fusarium*, *Mycosphaerella*, *Tilletia*, *Ustilago* and others, was due to the introduction and widespread use of systemic fungicides which are generally uni- or oligo-site chemicals. Hence, they affect only one or few steps of the metabolism genetically controlled by the pathogen, and consequently, resistant strain can arise quickly by a single mutation or by selection of resistant individuals in the fungal population. Such resistance can develop in the pathogen by a decrease of cell membrane permeability to the chemical, by a detoxification of the chemical, by a decrease of conversion to the real

toxic compound, by a decrease of affinity at the reactive site in the cell, by a bypass of a blocked reaction, or by a compensation of the inhibition effect by producing more inhibited product.

To ovoid the apparition of resistant fungal strain, it is recommended to use mixtures of specific systemic fungicides and wide-spectrum contact fungicides, to alternate sprays of systemic fungicides and contact fungicides or to spray during half season with systemic fungicides and other half season with contact fungicides. Hence, systemic fungicide gives a good control of the disease while the contact fungicide reduces the probability of survival of any strains that may develop resistance to the systemic chemical.

INTEGRATED MANAGEMENT

Integrated control of fungal disease of plants is based on the use of all possible kinds of control methods. This is achieved by the elimination or reduction of the initial inoculum and its effectiveness, by the increasing of the resistance of the host, by the delaying of the onset of disease and by the slowing of the secondary cycles of the pathogen.

For field crops as many other kinds of cultures, it is recommended to use healthy seeds or, at least, treated seeds. Cropped fields, as far as possible, have to be free of soil fungi, for example by deep plowing soil in the summer. The destruction of volunteer plants, alternative and alternate host plants contributes to the reduction of the fungal inoculum and the interruption of the life cycle of the fungus. Crops other than field crops must be used in crop rotation and soils have to be well-drained. The fertilizer program should be well-balanced. When resistant varieties are not available, it is necessary to treat crops during their growth using appropriate fungicides, especially when the weather is favorable to the infections. After crop harvest, the storage houses and the tools used must be cleaned and disinfected.

Fungal Diseases of Cereals

Root and Foot Diseases

TAKE-ALL OF CEREALS

The fungus: *Gaeumannomyces graminis* var. *tritici*

G. graminis var. *tritici* belongs to the phylum of *Ascomycota* (fungi producing ascospores) and the group of Pyrenomycetes (fungi producing their asci in perithecia). Observed abroad, this species is characterized by filamentous ascospores, often slightly curved with rounded tips. These ascospores are multicellular, forming 3 to 5 or sometimes more septa. Their sizes are 80-110 x 2.5-3 µm. The anamorphic stage having no particular name is characterized by hyphopodia which are simple, oval to long cylindrical when terminal and roughly spherical when intercalary (Figure 2). In host plant, the fungus develops dark brown runner hyphae which branch in paler hyphae producing the hyphopodia. On culture medium, the anamorph often produce unicellular conidia, slightly or strongly curved, sometimes semicircular and having sizes 4-7 x 1-1.5 µm (Walker*, 1973).

Symptoms

Observed on wheat, this disease is characterized by dry rot in roots and the basal parts of stems which become dark brown to black. Roots heavily infected are mostly destroyed (Photo 1). At the heading stage, spikes of infected plants prematurely ripen and become bleached presenting white patches dispersed in the field crop. Those spikes are sterile or produce shriveled grains with low germination power.

Biology

The fungus survives in infected debris as mycelium or perithecia. Ascospores released from perithecia as well as the active mycelium cause the primary infection. Secondary infections during the cereal grown season are due to runner hyphae which develop in root from plant to plant. Ascospores produced during the growing season and disseminated by rain and wind can also play a role in the occurrence of secondary infections. A wet weather and temperatures between 10 and 20 °C are favorable to the disease. Since the infection occurs at early stages of the plant, it is recommended to treat seeds with systemic fungicides to prevent the fungal attack from the soil even though the disease is not seedborne.

Chemical control

Seed treatment: Treatment with systemic fungicides based on fluquinconazole, prochloraz, silthiofam.

***** Walker J., 1973 - *Gaeumannomyces graminis* var. *tritici*. CMI Descriptions of Pathogenic Fungi and Bacteria, N° 383.

EYESPOT (OR STAWBREAKER) OF CEREALS

The fungus: *Mollesia yallundae*
(Anamorph: *Pseudocercosporella herpotrichoides*)

The anamorph *P. herpotrichoides* belongs to the Anamorphic Fungi and the group of Hyphomycetes (free conidia). Observed abroad, conidia of *P. herpotrichoides* are multicellular, filamentous with one side wider than the other, straight or very slightly curved, smooth, with 3 to 7 septa, not constricted (Figure 3). Their sizes are 26.5-47 x 1-2 µm (Booth* & Waller, 1973).

The teleomorph *M. yallundae* belongs to the phylum of *Ascomycota* (fungi producing ascospores) and the group of Discomycetes (fungi forming their asci in apothecia).

Symptoms

Observed on wheat, this disease appears on the leaf blade as ellipsoid spots often fade with black dots on the blade internal face which are the fungal stromata. On the basis of these stromata, *P. herpotrichoides* can be differentiated from *Fusarium* spp. The infection moves up to the first internode of the stem where it causes an elliptic lesion with diffuse brown border (Photo 2). Such lesion can lead to the lodging of the mature plants. Compared to the physiologic lodging, this parasitic lodging occurs in all directions and appears as patches in the field, while physiologic lodging is generally homogenous and occurs according to the direction of the responsible wind.

Biology

The fungus, which survives in host plant debris as mycelium, infects the cereal soon after seedling plant emergence (primary infection), directly with the active mycelium or with conidia that it produces on stubbles. Secondary infections are due to conidia produced by the fungus during cereal plant growth and disseminated by wind and rain. The production of conidia is maximal when temperature is nearly 10 °C. A cool and humid weather is favorable to the disease spread. Above 25 °C, the infection seems to be blocked.

Chemical control

Foliar treatment: Treatment with fungicides based on cyprodinil, fluquinconazole, flusilazole, flutriafol, prochloraz, propiconazole, tebuconazole, tetraconazole.

* Booth C. & Waller J. M., 1973 - *Pseudocercosporella herpotrichoides*. CMI Descriptions of Pathogenic Fungi and Bacteria, N° 386.

FUSARIUM DISEASES OF CEREALS

The fungi: *Fusarium* spp.

Anamophs are species of the genus *Fusarium* which belongs to the Anamorphic Fungi and the group of Hyphomycetes (sporodochial conidiomata). Conidia of *Fusarium* spp. are generally multicellular in a crescent shape (Photo 3). The size, the shape and the cell number depend on the species among which the most recent described one is *F. pseudograminearum* (Gargouri* *et al.*, 2006).

When the teleomorphs exist, they are species of the genus *Gibberella* or *Nectria* and belong to the phylum of *Ascomycota* (fungi producing ascospores) and the group of Pyrenomycetes (fungi forming their asci in perithecia).

Symptoms

Observed on wheat and barley, the infection can lead to a damping off which makes seedlings look necrotic and die soon after seed germination or seedling emergence. Infected plants can also show rotted roots, crowns and stem basis (Photo 4). Depending on the species of *Fusarium* and the infection conditions, roots, crowns and stem basis may look brown and sometimes rotted producing within and on the first internode, a cottony layer (white, white-grey or white-pink) corresponding to the mycelium and the sporodochia of the fungus. Also, depending on the species of *Fusarium*, the infection may reach partially or totally the spike which turns white, dries before maturity and produces shriveled grains (Photo 4). The fungus infects then grains and can produce poisons called mycotoxines able to cause serious problems to the human and animal health. When infection occurs early in the season, spikes are sterile.

Biology

Some species of *Fusarium* are seedborne and all species of this genus are soilborne where they survive as mycelium and parithecia in infected debris and as chlamydospores. Infection starts from soil and infected stubbles and concerns roots and/or crowns then can move up to stems. On the infected plants, the fungus produces mycelium and releases conidia which, for some species, are carried by wind and rain to reach and infect spikes as soon as the heading stage starts. It is also possible that the fungus develops systematically from the foot of the plant to internally reach the spike, but this way of spike infection seems to be rare. Attacked spikes produce infected seeds making the disease seedborne. Optimal conditions for the development of *Fusarium* diseases depend on the *Fusarium* species, but usually low humidity and temperatures around 20 °C are conditions favorables to these diseases.

Chemical control

Seed treatment: Treatment with fungicides based on difenoconazole, fludioxonil, mancozeb, prochloraz, thiabendazole, thiram, triticonazole.

Foliar treatment: Treatment with fungicides based on azoxystrobin, epoxiconazole, fluquinconazole, metconazole, picoxystrobin, prochloraz, propiconazole, pyraclostrobin, tébuconazole, trifloxystrobin.

* Gargouri S., Hamza S., & Hajlaoui M.R., 2006 - AFLP analysis of the genetic variability and population structure of the wheat foot rot fungus *Fusarium pseudograminearum* in Tunisia. Tunisian Journal of Plant Protection, 1 (2) (*in press*).

SPOT BLOTCH OF CEREALS

The fungus: *Cochliobolus sativus*
(Anamorph: *Drechslera sorokiniana*)

The anamorph *D*. *Sorokiniana* belongs to the Anamorphic Fungi and the group of Hyphomycetes (free conidia). Observed abroad, conidia of the anamorph are multicellular, straight to curved, fusiform to broadly ellipsoidal with 3 to 12 pseudosepta (Figure 4). Their sizes are 40-120 x 17-28 µm (Sivanesan* & Holliday, 1981).

The teleomorph *C*. *sativus* belongs to the phylum of *Ascomycota* (fungi producing ascospores) and the group of Loculoascomycetes (fungi forming their asci in pseudothecia).

Symptoms

Observed on barley, this disease is a foot infection, but symptoms become generally better visible after heading. The infection begins at seedling stage when dark brown necroses appear on roots, crowns and the basal leaves. Generally, early infections of roots and crowns kill the seedlings. The disease may reach the vascular tissue of the first internode and slows down the sap movement. On the outside, the internode becomes black-grey due to the dense sporulation of the fungus. Later on, elongated to oval dark brown spots appear on leaves. Gray necroses may be formed (Photo 5).

Biology

This fungus is seedborne and survives in soil and infected plant stubbles as mycelium, conidia and rarely pseudothecia, causing the primary infection. During the cereal plant growth, secondary infections take place following conidia disseminated by wind and rain. High humidity and temperatures above 20 °C (optimum 28 °C) are favorable to this disease.

Chemical control

Seed treatment: Treatment with fungicides based on fludioxonil, mancozeb, maneb, prochloraz, thiabendazole, thiram, triticonazole.

Foliar treatment: Treatment with fungicides based on azoxystrobin, epoxiconazole, flusilazole, prochloraz, pyraclostrobin, tebuconazole.

* Sivanesan A. & Holliday P., 1981 - *Cochliobolus sativus*. CMI Descriptions of Pathogenic Fungi and Bacteria, N° 701.

Stem and Foliar Diseases

TAN (OR YELLOW LEAF) SPOT OF WHEAT

The fungus: *Pyrenophora tritici-repentis*
(Anamorph: *Drechslera tritici-repentis)*

The anamorph *D*. *tritici-repentis* belongs to the Anamorphic Fungi and the group of Hyphomycetes (free conidia). Conidia of *D*. *tritici-repentis* are multicellular, elongated, straight or slightly curved, rounded at the apex and sharp at the base. The wall is thick with cells separated by 1 to 10 pseudosepta which become constricted when conidium is aging. Their sizes are 100-240 x 10-16 µm (Photo 6).

The teleomorph *P*. *tritici-repentis* belongs to the phylum of *Ascomycota* (fungi producing ascospores) and the group of Loculoascomycetes (fungi forming their asci in pseudothecia).

Symptoms

Observed on wheat, this disease causes on leaves small but elongated spots which first appear yellow-brown, then become oval, tanned and limited by a light yellow border. Those spots develop later into lesions which coalesce making leaves yellowish with necroses spreading progressively from the top to the bottom causing their total death (Photo 7).

Biology

The fungus survives in seeds as mycelium and in infected debris as mycelium and pseudothecia. Hence, primary infection can occur from infected seeds or more frequently from infected stubbles from which the fungus disseminates conidia produced by the active mycelium and/or ascospores released by pseudothecia. Secondary infections take place when conidia, produced by the fungus on infected leaves, are disseminated by wind and rain. The infection is favored by high humidity and temperatures between 18 and 28 °C. Seedborne inoculum seems to be insignificant.

Chemical control

Foliar treatment: Treatment with fungicides based on azoxystrobin, epoxiconazole, flusilazole, picoxystrobin, prochloraz, propiconazole, pyraclostrobin, tebuconazole, trifloxystrobin.

NET BLOTCH OF BARLEY

The fungus: *Pyrenophora teres*
(Anamorph: *Drechslera teres*)

The anamorph *D*. *teres* belongs to the Anamorphic Fungi and the group of Hyphomycetes (free conidia). Conidia of *D*. *teres* are cylindrical, straight with rounded tips and smooth wall. Also, they are multicellular with cells separated by 1 to 9 pseudosepta (often 4 to 6), usually constricted. Their sizes are 50-140 x 15-25 µm (Photo 8).

The teleomorph *P*. *teres* belongs to the phylum of *Ascomycota* (fungi producing ascospores) and the group of Loculoascomycetes (fungi forming their asci in pseudothecia).

Symptoms

Observed on barley, this infection shows on leaves brown blotches which may be dot-like, irregular, or elongated but limited laterally by foliar veins. Those blotches may also be rectangular more or less elongated or become coalescent and then form a net of lines easily observable through the light (Photo 9).

Biology

The fungus survives on the infected stubbles and can be seedborne. Primary infection is caused by conidia produced by the active mycelium and/or ascospores released by pseudothecia. It can start already at the seedling stage of the host. The secondary infections are due to conidia produced by the fungus during the barley growing season and disseminated by wind and rain. This infection is favored by high humidity and temperatures between 15 and 25 °C (optimum 20 °C). The attack usually concerns leaves although the mycelium may reach the spikes.

Chemical control

Seed treatment: Treatment with fungicides based on fludioxonil, mancozeb, maneb, prochloraz, thiabendazole, triticonazole.

Foliar treatment: Treatment with fungicides based on azoxystrobin, cyprodinil, epoxiconazole, picoxystrobin, prochloraz, propiconazole, pyraclostrobin, tebuconazole, trifloxystrobin.

BARLEY STRIPE

The fungus: *Pyrenophora graminea*
(Anamorph: *Drechslera graminea*)

The anamorph *D*. *graminea* belongs to the Anamorphic Fungi and the group of Hyphomycetes (free conidia). Conidia of *D*. *graminea* are straight or rarely a little bit curved and cylindrical with one side sometimes slightly wider than the other. Those conidia are multicellular with smooth wall, rounded tips and cells separated often by 1 to 7 pseudosepta (often 4 to 5). Their sizes are 40-86 x 12-20 µm (Photo 10).

The teleomorph *P*. *graminea* belongs to the phylum of *Ascomycota* (fungi producing ascospores) and the group of Loculoascomycetes (fungi forming their asci in pseudothecia).

Symptoms

Observed on barley, this infection shows on the leaves long solitary or grouped stripes, parallel to foliar veins. These stripes appear first yellow and then turn slowly to brown while the leaf tears out in the sense of the length (Photo 11). When infection gets heavy, plants are stunted and spikes do not almost emerge from blades, but if they emerge, they produce brown grains mostly sterile.

Biology

During the growing season, the fungal conidia produced externally on infected barley leaves, are carried by wind to inflorescences of healthy barley plants. These conidia germinate and contaminate grains which keep a normal appearance. Apart from grains, the fungus can not generally infect other organs of barley during vegetation. Thus, the number of firstly infected plants in the crop does not change during one growing season. When contaminated grains are used as seeds, the fungus resumes its activities and infects barley at early stages. It invades then systemically the seedling and the growing plant by attacking mainly leaves. Hence, exclusively seedborne characteristic of this disease makes that the chemical control must be limited to seed treatment. Moreover, the transmission of the fungus inside the seeds needs the utilization of obligatorily systemic fungicides. Humid and cold weather (temperatures near 12 °C) are favorable to the disease.

Chemical control

Seed treatment: Treatment with systemic fungicides based on fludioxonil, prochloraz, tebuconazole, thiabendazole, triticonazole.

BARLEY SCALD

The fungus: *Rhynchosporium secalis*

R. secalis belongs to the Anamorphic Fungi and the group of Hyphomycetes (free conidia). Conidia of *R. secalis* are bicellular, elongated with sharp tips, straight for one cell and straight or frequently slightly curved and beaked for the other. The two cells are generally unequally sized. The conidia sizes are 8-18 x 2-4 µm (Photo 12).

Symptoms

Observed on barley, symptoms appear on the leaves and at the joint point of the sheath and the blade, as oval lesions more or less elongated. These lesions look first pale green and water-soaked inside, but soon after, they dry out and turn to whitish color with definite dark brown border. Later on, lesions increase and coalesce, invading the entire sheath (Photo 13).

Biology

This infection occurs early in mid-winter as it is favored by humid and cold weather (temperature around 15 °C). Primary infection starts from contaminated seeds by the fungal mycelium and from infected stubbles on which the fungus produces conidia from the active mycelium. Secondary infections are due to conidia produced on barley during plant growth and disseminated by rain and wind.

Chemical control

Seed treatment: Treatment with fungicides based on maneb, prochloraz, thiabendazole.

Foliar treatment: Treatment with fungicides based on azoxystrobin, cyprodinil, epoxiconazole, flusilazole, metconazole, picoxystrobin, prochloraz, propiconazole, pyraclostrobin, tebuconazole, trifloxystrobin.

POWDERY MILDEW OF CEREALS

The fungus: *Blumeria graminis*
(Anamorph: *Oidium monilioides*)

The anamorph *O. monilioides* belongs to the Anamorphic Fungi and the group of Hyphomycetes (free conidia). Conidia of *O. monilioides* observed are unicellular, often elliptical, elongated or slightly swollen. Before maturation, conidia are set in long chains. They are 22-35 x 10-14 µm in size (Photo 14).

The teleomorph *B. graminis* belongs to the phylum of *Ascomycota* (fungi producing ascospores) and the class of Erysiphomycetes (fungi forming their asci in cleistothecia that mature by dehiscence). *B. graminis* produce cleistothecia which appear in light microscopy spherical, closed, dark brown to black with long appendages. Asci can be observed once the cleistothecium is crushed (Photo 14).

Symptoms

Observed on wheat, barley and oats, this infection appears in winter as small white pustules scattered on leaf surfaces. Pustules are formed by mycelium and conidial chains of the anamorph which is an ectoparasite. As the host plant grows, the pustules extend, coalesce, turn from whitish to white-yellowish and then white-grayish color. Soon after, black scattering dots appear; they correspond to cleistothecia produced by the teleomorph (Photo 15).

Biology

Primary infection is due to fungal ascospores and mycelial fragments conserved only in the infected debris, because seeds do not transmit the disease. Secondary infections are due to conidia released by the fungus from infected plants during their growth. It develops more when the humidity is medium and no liquid water on the surface of plant because it does not make conidia germinate. Thus, very rainy winter and spring are unfavorable to the disease. Favorable temperatures are between 15 and 20 °C; that is why the infection develops early in winter. During dry weather, conidia are easily carried by wind which is the best extension vector of powdery mildew.

Chemical control

Foliar treatment: Treatment with fungicides based on azoxystrobin, cyproconazole, epoxiconazole, flusilazole, metconazole, picoxystrobin, prochloraz, propiconazole, pyraclostrobine, tebuconazole, trifloxystrobin.

SEPTORIA LEAF BLOTCH OF WHEAT

The fungus: *Mycosphaerella graminicola*
(Anamorph: *Septoria tritici*)

The anamorph *S. tritici* belongs to the Anamorphic Fungi and the group of Coelomycetes (conidia in pycnidia). Conidia are filamentous, straight or as flexible lines, more slender at the apex and usually include 1 to 4 cells separated by septa. Conidia sizes are 28-70 x 1-1.5 µm (Photo 16).

The teleomorph *M. graminicola* belongs to the phylum of *Ascomycota* (fungi producing ascospores) and the group of Loculoascomycetes (fungi forming their asci in pseudothecia).

Symptoms

Observed not only on wheat but also on oats, this disease attacks mainly the foliar part. It produces on the leaves of wheat (particularly durum wheat) brown blotches, first irregular more or less elongated, usually laterally limited by the foliar veins and surrounded by a thin chlorotic border. Then, they gradually dry and become more and more light white starting by the center. Soon after, blotches extend to cover large foliar surfaces, indeed to reach the entire leaves if meteorological conditions are favorable. Numerous little dark brown to black dots form over the blotches and correspond to the anamorph pycnidia (Photo 17). When conditions are favorable, the disease reaches also blades causing blotches similar to those of leaves and can reach spikes whose glume tips become brown; grains become spotted.

Biology

At early stages, cereals are susceptible to be infected by the disease. Primary infection is due to conidia and/or ascospores released by rain from pycnidia and/or pseudothecia (respectively) existing in the infected stubbles of the precedent crops. During the growing season, infection is favored by high humidity and an optimal temperature between 15 and 20 °C. Secondary infections are caused by conidia released from pycnidia formed in infected plants. These conidia exuded on the foliar surfaces are disseminated towards the top and laterally by rains; their dispersion, and consequently the disease extension in the crop, depends then on the precipitation number fallen during the cereal vegetation. Seeds do not seem involved in the disease transmission.

Chemical control

Foliar treatment: Treatment with fungicides based on azoxystrobin, cyproconazole, epoxiconazole, flusilazole, flutriafol, metconazole, picoxystrobin, prochloraz, propiconazole, pyraclostrobin, tebuconazole, trifloxystrobin.

ASCOCHYTA LEAF SPOT OF WHEAT

The fungus: *Ascochyta tritici*

A. tritici belongs to the Anamorphic Fungi and the group of Coelomycetes (conidia in pycnidia). Conidia of *A. tritici* are elongated, cylindrical, with rounded tips and contain two cells separated by one middle septum. Sometimes three-celled, and rarely four-celled, conidia may be observed. The sizes are 12-20 x 2,5-6 µm (Photo 18).

Symptoms

This infection, that we are the first to identify on wheat in Tunisia (Nasraoui* *et al*., 1997), shows on leaves, more or less oval, sometimes elongated, pale spots surrounded by brown border. The lesions turn then to brown necroses in which numerous dark or black dots soon appear. They are pycnidia produced by the fungus (Photo 19). Symptoms, sometimes looking like those due to *Septoria* leaf blotch, may cause confusions in the identification. In this case, sure identification needs light microscopic observations of the conidia.

Biology

The fungus survives as pycnidia in the infected debris. The primary infection is due to conidia released from those pycnidia and disseminated by rain and wind. Disseminated conidia produced on infected wheat plants during the growing season cause the secondary infections. Rainy weather is favorable to this disease.

Chemical control

Foliar treatment: Treatment with fungicides based on bromuconazole.

* Nasraoui B., Terashima T. & Hafsa M., 1997 - Maladies nouvellement observées en Tunisie: L'anthracnose de l'orge et du blé. Annales de l'INRAT, 70: 215-221.

ASCOCHYTA LEAF SPOT OF BARLEY

The fungus: *Ascochyta hordei*

A. hordei belongs to the Anamorphic Fungi and the group of Coelomycetes (conidia in pycnidia). Conidia of *A. hordei* are elongated with rounded tips, slightly thin and formed by two cells separated by one middle septum. Very rarely, a second septum formed and the conidium becomes three-celled. Sizes of conidia are 11-24 x 3-5,5 µm (Photo 20).

Symptoms

This infection, that we are the first to identify on barley in Tunisia (Nasraoui* *et al*., 1997), shows on leaves spots which are rounded, oval, to more or less elongated, firstly brown and then gradually pale, surrounded by brown border. Around these spots, tissue frequently necroses. When the infection is heavy, spots become coalescent and leaf tissue turns white-yellowish and may tears out. At the end of the attack, light brown to dark brown punctuation appears inside spots and corresponds to the pycnidia produced by the fungus (Photo 21). Before pycnidia formation, *Ascochyta* disease symptoms are very similar to those of scald making both easily confusable. No doubt persists when pycnidia are observed.

Biology

The fungus survives as pycnidia in the infected debris. Conidia liberated from those pycnidia and disseminated by rain and wind cause primary infection. Secondary infections are due to conidia released from infected barley during plant growth. This disease is favored by a rainy weather.

Chemical control

Foliar treatment: Treatment with fungicides based on bromuconazole.

* Nasraoui B., Terashima T. & Hafsa M., 1997 - Maladies nouvellement observées en Tunisie: L'anthracnose de l'orge et du blé. Annales de l'INRAT, 70: 215-221.

LEAF (OR BROWN) RUST OF WHEAT

The fungus: *Puccinia recondita*

P. recondita belongs to the phylum of *Basidiomycota* (fungi producing basidiospores) and the class of Uredinomycetes (fungi having teliospore stage). *P. recondita* is characterized by monocellular urediospores which are oval, rarely ellipsoidal and slightly echinulate. Their sizes are 20-30 x 18-25 µm (Photo 22). Teliospores are bicellular, elongated with apical cell having tip which is rounded, flat or slightly pointed at the middle or at a side, and with a basal cell generally longer than the apical one, extending by a short pedicel. Teliospores are light brown and have thick and smooth wall. Their sizes are 30-48 x 10-20 µm (Photo 22).

Symptoms

Observed on wheat (especially durum wheat), this infection shows a multitude of small brown sporal pustules, generally rounded, irregularly scattered on the two leaf sides. Blades and stems are rarely infected. Pustules are uredia produced by the fungus from which urediospores are released after the epidermis of the host plant breaks down (Photo 23). As long as wheat plants mature and become gradually yellow and dry, black pustules appear and remain generally nondehiscent. These pustules are telia containing teliospores (Photo 23).

Biology

Although wheat leaf rust can infect an alternate host (mainly *Thalictrum*, but also *Anchusa*, *Anemonella*, *Clematis*, *Isopyrum*), which transmits the disease to wheat via aeciospores, it seems that the life cycle of *P. recondita* is limited only to the wheat primary host. Hence, it is possible that the warm winter of Mediterranean regions allows the survival of the fungus as urediospores and mycelium in infected debris from one year to another. During vegetation of wheat under favorable meteorological conditions, urediospores of the precedent year or those produced *de novo* by the active mycelium in infected stubbles, would be disseminated by wind and would cause primary infection of wheat at late-winter early-spring. But, it is also not excluded that urediospores come from far regions such as South-West of Europe, carried by dominant North-West winds and disseminated in the Maghrebian regions. Secondary infections at spring are caused by urediospores produced on infected wheat plants during their growth and disseminated mainly by wind. Teliospores produced by the fungus at late-spring for its conservation until late next winter, seem to have no role to play in the occurrence of primary infection, when the alternate host is absent. High humidity and temperatures between 15 and 25 °C are favorable to the disease which is not seed-borne.

Chemical control

Foliar treatment: Treatment with fungicides based on azoxystrobin, cyproconazole, epoxiconazole, fenpropimorph, flusilazole, flutriafol, metconazole, picoxystrobin, propiconazole, pyraclostrobin, tebuconazole, tetraconazole, trifloxystrobin.

YELLOW (OR STRIPE) RUST OF WHEAT

The fungus: *Puccinia striiformis*

P. striiformis belongs to the phylum of *Basidiomycota* (fungi producing basidiospores) and the class of Uredinomycetes (fungi having teliospore stage). *P. striiformis* has monocellular urediospores which are spherical to oval, rarely ellipsoidal with a slightly echinulate wall. Their sizes are 20-30 x 10-18 µm (Photo 24). Teliospores of the fungus are bicellular, elongated with apical cell having tip which is flat, rounded or pointed at the middle or at the side. The basal cell is generally longer than the apical one, extending by a short pedicel. Teliospores are light brown, have thick smooth wall and have sizes of 25-65 x 15-25 µm (Photo 24).

Symptoms

Observed on wheat (especially bread wheat), this infection shows numerous small yellow-orange sporal pustules, arranged in long stripes, parallel to the foliar veins. Other host plant organs may be infected. Pustules are uredia releasing urediospores after the break down of host plant epidermis (Photo 25). As long as wheat plants mature and become gradually yellow and dry, new black pustules develop and remain generally nondehiscent. They are telia producing teliospores (Photo 25).

Biology

The life cycle of *P. striiformis* is limited to the wheat as sole primary host, since no aecian stage has been observed on another host. The same hypotheses presented in the leaf rust case may explain the beginning of the primary infection: urediospores causing the primary infection would come from far regions like South-West of Europe, carried by dominant North-West winds and disseminated in Maghrebian regions or the fungus would survive as urediospores and mycelium in infected stubbles in the warm winter Mediterranean regions; these urediospores or those produced *de novo* by the active mycelium cause primary infection. Secondary infections are due to urediospores produced on infected plants and disseminated by wind. The teliospores produced by the fungus at late-spring for its survival, do not seem to play a role in the starting of the disease the next year. High humidity and temperatures between 5 and 15 °C are favorable to the infection which is not seedborne.

Chemical control

Foliar treatment: Treatment with fungicides based on azoxystrobin, cyproconazole, epoxiconazole, fenpropimorph, flusilazole, flutriafol, metconazole, picoxystrobin, propiconazole, pyraclostrobin, tebuconazole, tetraconazole, trifloxystrobin.

STEM (OR BLACK) RUST OF WHEAT

The fungus: *Puccinia graminis*

P. graminis belongs to the phylum of *Basidiomycota* (fungi producing basidiospores) and the class of Uredinomycetes (fungi having teliospore stage). *P. graminis* has monocellular urediospores, generally oval to slightly ellipsoidal, rarely spherical and have echinulate wall. Their sizes are 22-37 x 12-20 µm (Photo 26). Teliospores are elongated and bicellular. The apical cell is oval to elongated with a rounded or a slightly pointed tip. The basal cell is relatively longer than the apical one and extends by a long pedicel. Teliospores are dark brown and have thick smooth wall. Their sizes are 28-75 x 15-25 µm (Photo 26).

Symptoms

Observed not only on wheat, but also on barley and oats, the stem rust shows brown long sporal pustules which develop mainly on stems and to a less degree on leaves, but can sometimes reach spikes. Pustules become coalescent when the infection is heavy. These pustules are uredia releasing urediospores after the break down of the host plant epidermis (Photo 27). As long as cereal plants mature and become gradually yellow and dry, black pustules forming telia appear and release teliospores after the break down of the host plant epidermis (Photo 27).

Biology

It is well known that the stem rust infects wheat as primary host and *Berberis* or *Mahonia* as alternate host which transmits the disease to wheat via aeciospores. Nevertheless, it is still possible that primary infection in warm winter regions is also caused by the fungus surviving in infected wheat stubbles via urediospores of the precedent year or those produced *de novo* by active mycelium. Equally, dominant North-West winds could carry urediospores from far regions (for example European South-West) and disseminate them. Secondary infections are caused by urediospores disseminated by wind from the cereal during their growth. At early-summer, the fungus produces telia releasing teliospores for survival. The stem rust is considered as a late rust which generally appears during a rainy late-spring (high humidity and temperatures between 25 and 30 °C) and it is not seedborne. Historically, this disease was most important when autochthon cultivated wheat were generally late varieties. But with the breeding during the last decades of high yield early varieties, the short life cycle of those varieties (until late-May early-June) does not give enough time to the development of stem rust which consequently has become a rather rare disease.

Chemical control

Foliar treatment: Treatment with fungicides based on azoxystrobin, cyproconazole, epoxiconazole, fenpropimorph, flusilazole, flutriafol, metconazole, picoxystrobin, propiconazole, pyraclostrobin, tebuconazole, tetraconazole, trifloxystrobin.

LEAF (OR BROWN) RUST OF BARLEY

The fungus: *Puccinia hordei*

P. hordei belongs to the phylum of *Basidiomycota* (fungi producing basidiospores) and the class of Uredinomycetes (fungi having teliospore stage). *P. hordei* have monocellular urediospores which are spherical, ovoid to slightly ellipsoidal with a slightly echinulate thick wall. Their sizes are 20-30 x 18-22 µm (Photo 28). Teliospores of *P. hordei* are not only bicellular but also monocellular. Bicellular teliospores are elongated and slightly clavate. The apical cell has tip which is rounded, flat or slightly pointed at the middle or at the side. The basal cell is slightly longer than the apical one and extends by a short pedicel. Their sizes are 36-50 x 15-25 µm. Monocellular teliospores are spherical or slightly elongated, have irregular shape and extend with short pedicel. Their sizes are 20-38 x 14-24 µm (Photo 28).

Symptoms

Observed on barley, this disease shows leaves with a multitude of small brown pustules which are rounded or slightly elongated. Pustules are uredia releasing urediospores when host plant epidermis breaks down (Photo 29). As long as barley plants mature and become gradually yellow and dry, black pustules appear. They are often nondehiscent telia producing teliospores (Photo 29).

Biology

Although the leaf rust can infect barley as primary host via aeciospores produced by the fungus on the alternate host (genus *Ornithogalum*), its life cycle may be limited to only barley. Thus, in warm winter regions, the urediospores of the precedent year or those produced *de novo* by active mycelium surviving in infected stubbles, could cause primary infection. This infection could also be due to urediospores carried from far regions (such as South-West of Europe) by dominant North-West winds. Urediospores produced on barley during spring and disseminated by wind cause secondary infections. In the absence of the alternate host, it seems that teliospores (produced in telia for the survival of the fungus) have no role to play in the primary infection the next year. Temperatures from 15 to 20 °C and high humidity are favorable to the disease which is not seedborne.

Chemical control

Foliar treatment: Treatment with fungicides based on azoxystrobin, cyproconazole, epoxiconazole, fenpropimorph, flusilazole, flutriafol, metconazole, picoxystrobin, propiconazole, pyraclostrobin, tebuconazole, trifloxystrobin.

CROWN RUST OF OATS

The fungus: *Puccinia coronata*

P. coronata belongs to the phylum of *Basidiomycota* (fungi producing basidiospores) and the class of Uredinomycetes (fungi having teliospore stage). Urediospores of *P. coronata* are monocellular, spherical to oval, sometimes ellipsoidal with slightly echinulate thick wall. Their sizes are 18-26 x 15-20 µm (Photo 30). Teliospores are bicellular and elongated. The apical cell is characterized by conical extensions arranged at random or as a crown; that is why this disease is called "crown" rust. The basal cell is longer than the apical one and extends by a short pedicel. The teliospore sizes are 25-58 x14-22 µm (Photo 30).

Symptoms

Observed on oats, the disease shows pustules light yellow-orange, often elongated, which develop on both sides of leaf sheath and on blade. Pustules are uredia releasing urediospores after the host plant epidermis breaks down (Photo 31). As long as oats plants mature and become gradually yellow and dry, black pustules which are telia producing teliospores appear; many of those pustules are nondehiscent at maturity (Photo 31).

Biology

Crown rust attacks oats as primary host via aeciospres produced by the fungus on the alternate host which is the genus *Rhamnus.* Recently, it was observed and confirmed in Tunisia that the species *Rhamnus lycioides* plays effectively the role of alternate host for this rust (Hemmami* *et al.*, 2006). The fungus develops on *R. lycioides* and produces spermatia and then aeciospores which cause primary infection on oats. Nevertheless, we may be still keep hypotheses indicated for the other cereal rusts in Tunisia as additional possibilities in the development of this disease. Hence, it is not exclude that the fungal life cycle takes place only on oats as primary host in regions where *R. lycioides* does not exist. Primary infection may be caused by urediospores carried from far regions (as South-West of Europe) and disseminated by dominant North-West winds. In warm winter regions, the fungus could also survive as urediospores or mycelium in infected stubbles; these urediospores or those produced *de novo* by active mycelium could cause primary infection. Secondary infections are due to urediospores released and disseminated by wind from infected plants during their vegetation. Teliospores of the fungus produced on oats late-spring for survival play a crucial role at the next early-spring. In fact, these teliospores surviving between early-summer and late-winter produce after germination basidiospores able to infect the alternate host *R. lycioides.* High humidity and temperature around 20 °C are favorable to this disease which is not seedborne.

Chemical control

Foliar treatment: Treatment with fungicides based on azoxystrobin, cyproconazole, epoxiconazole, fenpropimorph, flusilazole, flutriafol, metconazole, picoxystrobin, propiconazole, pyraclostrobin, tebuconazole, tetraconazole, trifloxystrobin.

* Hemmami I., Allagui M B., Chakroun M. & El Gazzeh M., 2006 - *Rhamnus lycioides* in Tunisia is a new aecial host of oat crown rust. European Journal of Plant pathology, 115: 357-361.

FLAG SMUT OF WHEAT

The fungus: *Urocystis agropyri*

U. agropyri belongs to the phylum of *Basidiomycota* (fungi producing basidiospores) and the class of Ustilaginomycetes (fungi having teliospore stage). *U. agropyri* has teliospores which are solitary or grouped as a set of 1 to 5 (often 1 to 3). They are brown, spherical with a smooth wall and have a diameter of 9-20 µm. These teliospores are systematically surrounded by sterile cells which are spherical to oval and have smaller sizes than teliospores, often 5 to 10 µm (Photo 32).

Symptoms

Observed on wheat (especially durum wheat), this infection shows long black sporal stripes parallel to foliar veins. They are the sori which release teliospores after the epidermis of host plant breaks down. Infected plants are stunted, generally fail to head and their leaves become rolled (Photo 33).

Biology

Teliospores, looking as a dark dust, are released during harvest. They pollute soil and seeds and make the fungus attacking cultivated wheat the next year (embryo infection). Infection of wheat seedlings takes place at the coleoptile level, usually during emergence, at a soil temperature between 10 and 20 °C. Teliospores germinate producing basidia that release basidiospores able to fuse developing infectious mycelium which attack wheat seedlings. The fungus develops then systemically inside the entire plant, but symptoms appear only on leaves, usually after the jointing stage. During wheat vegetation, the disease does not transmit from one plant to another.

Chemical control

Seed treatment: Treatment with fungicides based on carboxin, prochloraz, tebuconazole, triticonazole.

Spike Diseases

WHEAT (COMMON) BUNT

The fungi: *Tilletia laevis* and *Tilletia tritici*

T. laevis and *T. tritici* belong to the phylum of *Basidiomycota* (fungi producing basidiospores) and the class of Ustilaginomycetes (fungi having teliospore stage). Teliospores of *T. laevis* are brown pale, spherical or more frequently oval, sometimes angular and surrounded by a smooth wall. Their diameters are 14-24 µm (Photo 34). Teliospores of *T. tritici* are brown, spherical or a little bit oval, rarely angular and surrounded by an echinulate wall. Their diameters are 14-22 µm (Photo 34). In addition, sterile cells among the teliospores of *T. laevis* and *T. tritici*, are observed. They are spherical, with smooth wall and have sizes equal or slightly inferior to those of the teliospores (Nasraoui* *et al*., 1994).

Symptoms

Observed on wheat (especially bread wheat), this disease is difficult to identify before heading with dark green colored stems. At maturity, the bunted spike of durum wheat seems less developed than the nonbunted one and has a pale color especially with regard to its awes. However, for bread wheat, it is easier to identify the disease before plant maturity, because spikes are often dark green to blue green, then turn lighter with spikelets apart and grains frequently emerging with dark brown to black tips (Photo 35). These grains, easy to crush, are globular and full of black dust formed by the fungal teliospores which smell as rotted fish.

Biology

When wheat seeds germinate, fungal teliospores in the soil or sticking on the seed surface, germinate by basidia and produce basidiospores. These basidiospores fuse and produce conidia that germinate and infect the wheat seedling just after its germination (embryo infection). If the environment conditions are favorable to the wheat plant (available water, soil temperature above 20 °C, balanced fertilizers,...), wheat seeds germinate and seedlings develop quicker than the fungus making the majority of plants to escape to the infection, since the fungus can no more infect the seedling when it reaches 1-2 leaf stage. But, if the environment conditions are unfavorable to the plant with soil temperature between 5 and 12 °C, infection with bunt becomes heaver. When the infection succeeds, the fungus evolves, reaches the plant growing point and develops systemically in the plant as long as it grows. When the grains begin to form, the fungus invades them internally and forms its teliospores. Only the envelope of the grains persists. Hence, the disease can not propagate from one plant to another during the growing season and the number of infected plants does not change from autumn to early-summer. During harvest, crushed grains release teliospores as a black dust which pollutes the healthy grains and the soil and allows the fungus to be transmitted to the cultivated wheat of the next year.

Chemical control

Seed treatment: Treatment with fungicides based on difenoconazole, tebuconazole, thiabendazole, triticonazole.

* Nasraoui B., Yahyaoui A., Fnayou S., Khabbouchi H. & Ben-Harrath B., 1994 - La carie du blé en Tunisie: Identification des espèces fongiques responsables et essais de lutte chimique. Revue de l'INAT, 9: 19-45.

COVERED SMUT OF BARLEY AND OATS

The fungus: *Ustilago segetum*

U. segetum belongs to the phylum of *Basidiomycota* (fungi producing basidiospores) and the class of Ustilaginomycetes (fungi having teliospore stage). Teliospores of *U. segetum* are monocellular, spherical, more or less rounded, lighter at one side than at the other and have a smooth dark brown wall. Their diameter is 5-10 µm (Photo 36).

Symptoms

Observed on barley and oats, this disease invades all grains of the spike where fungal teliospores form a black and dusty mass. Sporal mass produced remains inside the floral envelopes which persist. Globally, smutted spikes are black and small, but keep intact their whole exterior structure (Photo 37).

Biology

Infection takes place during the germination of barley seeds through fungal teliospores coming from the soil or sticking on the healthy seed surface after being liberated from smutted crushed grains during harvest. These teliospores germinate forming basidia which release basidiospores able to germinate and infect barley just after seed germination (embryo infection). Fair humid soil and temperature around 15 °C are favorable to the disease. Mycelial hyphae penetrate in the seedling and then reach the vegetative point and the floral primordia whose development ends by a spike totally smutted. During harvest, smutted crushed grains release teliospores which pollute the soil and healthy grains.

Chemical control

Seed treatment: Treatment with fungicides based on difenoconazole, tebuconazole, thiabendazole, triticonazole.

LOOSE SMUT OF CEREALS

The fungi: *Ustilago segetum* var*. tritici* (on wheat)
***Ustilago segetum* var. *nuda* (on barley)**
***Ustilago segetum* var. *avenae* (on oats)**

U. segetum varieties belong to the phylum of *Basidiomycota* (fungi producing basidiospores) and the class of Ustilaginomycetes (fungi having teliospore stage). Teliospores of these varieties look alike under light microscopy. They are monocellular, spherical, more or less rounded with thick, light brown and slightly echinulate wall. Their diameter is 5-10 µm (Photos 38).

Symptoms

Observed on wheat, barley and oats, this infection invades totally the spike. Thereafter, spikelets are destroyed and transformed into a black dusty mass formed by the fungal teliospores (Photos 39). When the wind blows and carries the sporal mass, it generally remains only the rachis of the spike.

Biology

During the spring, teliospores of smutted spikes are carried by the wind and deposed on healthy inflorescence stigmata. Then, each teliospore germinates without producing basidiospores, enters as a pollen tube, infects a part of the embryo, and survive inside as a mycelium (floral infection). Infected grains keep a normal appearance. From autumn, when infected seeds germinate, the fungus resumes its activity, attacks the seedling, reaches the vegetative point, and invades the floral primordia which later become smutted. From those smutted spikes, healthy inflorescence infection occurs during the same spring season. High humidity and temperatures from 15 to 20 °C are favorable to this disease which is exclusively seedborne. In addition, internal transmission of the parasite inside seeds needs the utilization of obligatorily systemic fungicides.

Chemical control

Seed treatment: Treatment with systemic fungicides based on difenoconazole, tebuconazole, thiabendazole, triticonazole.

CLADOSPORIUM DISEASE OF WHEAT

The fungi: *Cladosporium* sp.

The genus *Cladosporium* belongs to the Anamorphic Fungi and to the group of Hyphomycetes (free conidia). This genus is characterized by a high morphological variation of conidia inside the same species. Our light microscopic observations show conidia with various shapes and sizes. Their dimensions are 5-18 x 3-7 µm. They have forms from monocellular almost spherical to tricellular thin and elongated (Photo 40). The species that we observed seems to be *C. herbarum*.

Symptoms

Observed on wheat, this disease that we report here for the first time in Tunisia (results non shown), concerns mainly the mature spikes. It is characterized by the development of black sooty which is superficial, in small tufts, covering externally the cereal spikes at maturity stage (Photo 41). This sooty is formed of mycelium and erected conidiophores of the fungus. Inside the envelops, grains already mature are usually not attacked.

Biology

This fungus is a weakly plant parasite which develops only on weakened or almost dead tissue presenting no defense reaction. Living on other weakened plants or dead tissues in the nature, this fungal species disseminated by wind and rain, develops on already mature spikes of the cereal when abundant precipitations fall during several days of early summer. The fungal attack does not normally concern grains which are hard.

Chemical control

It is useless to make chemical treatment since the grains are already at mature stage. In contrast, it is necessary to make sure that the grains will be dried after harvesting to prevent all kinds of rot during storage.

Fungal Diseases of Legumes

CHICKPEA WILT

The fungus: *Fusarium oxysporum*

F. oxysporum belongs to the Anamorphic Fungi and the group of Hyphomycetes (conidia in sporodochia). The species of *Fusarium* that we have observed, produces multicellular conidia, straight or generally arched with varied number of cells, ranging often from 5 to 6. Their sizes are 16-36 x 3-5 µm (Photo 42).

Symptoms

Observed on chickpea, this disease appears as a partial or total wilt, followed by a yellowing and a drying out. When the stem bases are split, a brown color in infected vessels is observed (Photo 43).

Biology

The fungus survives in seeds, soil, and infected debris of chickpea as mycelium or chlamydospores. The active mycelium and/or chlamydospores after germination enter in the host by roots or crown and cause the primary infection. Then, the fungus proliferates inside the xylem tissue in the stem basis causing obstruction and destruction of the attacked vessels. During vegetation, secondary infections are due to water of rain or irrigation which, when flooding on the soil, carries the fungus from one plant to another. Low to middle humidity and temperatures near or above 25 °C are favorable to this disease.

Chemical control

Seed treatment: Treatment with fungicides based on carbendazim, carboxin, thiram.

Foliar treatment: Treatment with fungicides based on carbendazim, methyl-thiophanate.

DOWNY MILDEW OF FABA BEAN

The fungus*: Peronospora viciae*

P. viciae belongs to the phylum of *Oomycota* (pseudo-fungi producing oospores). Conidia of *P. viciae* are monocellular, oval to ellipsoidal. Their sizes are 20-30 x 17-22 µm. Conidiophores end with sterigmata straight or slightly curved, quite pointed and generally grouped by couple (Photo 44).

Symptoms

Observed on faba bean, this disease appears on the leaf lower face as a cottony gray layer, resembling to mold. This cottony layer is formed of conidia and conidiophores which emerge from leaf stomata of host plant. The cottony layer begins as spots and extends progressively to cover all lower face of leaves. On the upper face of leaves, chlorotic spots appear. At the end of infection, the foliar tissue under the cottony layer becomes brown and dies (Photo 45).

Biology

The fungus survives in soil or plant debris as oospores and/or mycelium. Primary infection is caused by active mycelium or oospores after germination. Secondary infections are due to the fungal conidia produced on growing faba bean and disseminated by rain and wind. High humidity and temperatures near 15-20 °C are favorable to this disease which is not seedborne.

Chemical control

Foliar treatment: Treatment with fungicides based on fosetyl-Al, metalaxyl.

DOWNY MILDEW OF PEA

The fungus*: Peronospora pisi*

P. pisi belongs to the phylum of *Oomycota* (pseudo-fungi producing oospores). Conidia of *P. pisi* are monocellular, spherical to ovoid, with sizes 14-28 x 15-26 µm. Conidiophores end with sterigmata generally paired, quite pointed and often arched (Photo 46).

Symptoms

Observed on pea, this disease is recognized from a distance by the yellowish, shrunk and deformed upper part of the infected plant. From near, we observe on the leaf lower face a cottony gray layer similar to mold. This cottony layer corresponds to the fungal conidia and conidiophores emerging from stomata. The disease attacks mainly young organs of pea which rapidly dry out (Photo 47).

Biology

Primary infection is due to the fungus surviving in soil or plant debris as oospors and/or mycelium. Hence, active mycelium or oospores after germination start the disease development. Secondary infections are due to conidia produced on growing plants and disseminated by rain and wind. Humid weather and temperatures below or near 18 °C are favorable to this disease which is not seedborne.

Chemical control

Foliar treatment: Treatment with fungicides based on fosetyl-Al, metalaxyl.

POWDERY MILDEW OF PEA

the fungus: *Erysiphe* sp.
(Anamorph: ***Oidium* sp.)**

The anamorph *Oidium* sp. belongs to the Anamorphic Fungi and to the group of Hyphomycetes (free conidia). Several species can be the causal agent of the powdery mildew of pea. The species that we have observed seems to be *Oidium* sp. for whom the teleomorph is *E. polygoni* because their conidia are monocellular, solitary or in short chains, generally cylindrical and sometimes ellipsoidal or ovoid. Their sizes are 25-40 x 8-17 µm (Photo 48).

The teleomorph *Erysiphe* sp. belongs to the phylum of *Ascomycota* (fungi producing ascospores) and the class of Erysiphomycetes (fungi producing their asci in cleistothecia that mature by dehiscence).

Symptoms

Observed on pea, this disease appears as pustules which are powdery and white pale, covering all the aerial organs of the plant. These pustules keep extending more and more until to cover almost leaves, stems and pods. Later on, this powdery layer turns to gray yellowish and the beneath vegetal tissue becomes brown and dry out. The powdery layer corresponds to the mycelium and the conidia chains of the anamorph (Photo 49).

Biology

E. polygoni is a polyphage parasite, so in the absence of pea it can maintain on other cultivated or wild plants. It can also survive in infected debris as mycelium and/or cleistothecia. Hence, primary infection may be started by conidia released by the fungus growing on plants other than pea and/or by active mycelium or ascospores released by cleistothecia surviving on pea infected debris. The secondary infections are due to conidia produced on growing pea and carried by the wind. The extension of the disease up to late-spring indicates that the powdery mildew of pea is not disfavored by either high temperatures (above 25 °C) or low humidity. Generally, powdery mildew of legumes is not seedborne, but some exceptions exist such as powdery mildew of pea caused by *Erysiphe pisi.*

Chemical control

Foliar treatment: Treatment with fungicides based on azoxystrobin, dinocap, hexaconazole.

CHOCOLATE SPOT OF FABA BEAN

The fungus: *Botryotinia fabae*
(Anamorph: ***Botrytis fabae*)**

The anamorph *Botrytis fabae* belongs to the Anamorphic Fungi and to the group of Hyphomycetes (free conidia). Conidia of *Botrytis fabae* are monocellular, spherical or generally ovoid with smooth wall. The sizes are 10-22 x 8-17 µm (Photo 50).

The teleomorph *Botryotinia fabae* belongs to the phylum of *Ascomycota* (fungi producing ascospores) and the group of Discomycetes (fungi producing their asci in apothecia).

Symptoms

Observed on faba bean, this disease appears as red brown dots and very small circular light brown spots surrounded by red border, mainly on leaves and less frequently on stems (Photo 51). When climatic conditions are highly favorable during long time, the disease passes in an “aggressive” phase in which spots become coalescent lesions evolving later in dark brown rot.

Biology

Although the parasite is seedborne, seeds do not seem to play a major role in the beginning of primary infection. This fungus survives in infected debris as mycelium, apothecia, and sclerotia which resist for long time to unfavorable conditions. Primary infection may be caused at early stage of faba bean plants via active mycelium which resumes its development, germinating sclerotia and/or ascospores released from apothecia. Secondary infections are due to conidia produced by the fungus on the growing faba bean and disseminated mainly by wind. If climatic conditions of high humidity (around 100 %) and moderated temperatures (near 20 °C) last a certain time, they allow the disease to pass in the “aggressive” phase able to cause important damages.

Chemical control

Seed treatment: Treatment with fungicides based on vinchlozolin.

Foliar treatment: Treatment with fungicides based on mancozeb, methyl-thiophanate, vinchlozolin.

ALTERNARIA LEAF SPOT OF FABA BEAN

The fungus: *Alternaria alternata*

A. alternata belongs to the Anamorphic Fungi and the group of Hyphomycetes (free conidia). Conidia of *A. alternata* are multicellular, elongated, generally wider in one side than in the other and extend at the basis by a pedicel. The septa form simultaneously in the longitudinal and transversal direction. The sizes are 15-50 x 8-16 µm (Photo 52).

Symptoms

Observed on faba bean, this disease appears as brown gray foliar spots surrounded with a darker border and showing concentric circles inside. When the infection is heavy, spots extend on leaves and become coalescent (Photo 53).

Biology

Being polyphage, *A. alternata* is able to maintain on cultivated or wild plant species other than faba bean and then causes primary infection on faba bean via conidia. In addition, the fungus may survive in seeds and infected debris as mycelium which, after resuming its activity, can produce conidia able also to start primary infection. Secondary infections are due to conidia produced on growing faba bean plants and carried by rain and wind. A humid weather and temperatures near 20 °C are favorable to this disease which is marginally seedborne.

Chemical control

Foliar treatment: Treatment with fungicides based on azoxystrobin, chlorothalonil, mancozeb, methyl-thiophanate.

FABA BEAN BLIGHT

The fungus: *Didymella fabae*
(Anamorph: *Ascochyta fabae*)

The anamorph *A. fabae* belongs to the Anamorphic Fungi and the group of Coelomycetes (conidia in pycnidia). Conidia of *A. fabae* are straight or slightly curved, with rounded tips and generally one septum. Sometimes two or rarely three septa can be observed. The sizes are 12-24 x 3-5 µm (Photo 54).

The teleomorph *D. fabae* belongs to the phylum of *Ascomycota* (fungi producing ascospores) and the group of Loculoascomycetes (fungi producing their asci in pseudothecia).

Symptoms

Observed on faba bean, this disease appears on leaves as more or less irregular lesions which are first dark brown and then turn to light gray in the center surrounded with a darker border. When the infection is heavy, lesions become coalescent. They appear also on stems and pods and resemble to those on leaves but are generally hollowed in the tissue. Grains can also be infected. An abundant black punctuation, usually in concentric circles, appears in the lesions; they are the pycnidia produced by the anamorph (Photo 55).

Biology

The fungus, surviving in seeds and infected debris, causes primary infection at early stages of the faba bean plant. This primary infection is realized by conidia released from pycnidia and/or by ascospores released from pseudothecia. During faba bean vegetation, secondary infections are due to conidia of the fungus released from pycnidia and disseminated by wind and rain. This disease is favored by high humidity and temperatures near 20 °C. It is seedborne.

Chemical control

Seed treatment: Treatment with fungicides based on carboxin, captan, thiabendazole, thiram.

Foliar treatment: Treatment with fungicides based on azoxystrobin, chlorothalonil, metconazole.

CHICKPEA BLIGHT

The fungus: *Didymella rabiei*
(Anamorph: *Ascochyta rabiei*)

The anamorph *A. rabiei* belongs to the Anamorphic Fungi and the group of Coelomycetes (conidia in pycnidia). Conidia of *A. rabiei* are generally monocellular, rarely bicellular, elongated with rounded tips. The sizes are 5-13 x 3-5 µm. (Photo 56).

The teleomorph *D. rabiei* belongs to the phylum of *Ascomycota* (fungi producing ascospores) and the group of Loculoascomycetes (fungi producing their asci in pseudothecia). Observed for the first time in Tunisia, ascospores of *D. rabiei* are elongated, bicellular with sizes 13.5-17.5 x 6-7 µm (Rhaïem* *et al.*, 2006).

Symptoms

Observed on chickpea, this disease appears as lesions which are circular on leaves and pods and elongated on stems. They can reach even the grains. These lesions are first light brown to light gray surrounded by dark brown border, then they become necrotic. An abundant dark brown to black punctuation, usually in concentric circles, forms in the lesions and corresponds to the pycnidia produced by the anamorph (Photo 57). When the infection is heavy, lesions coalesce. On stems, they can become cankered and cause the break down of branches. Then, plant partially looses its leaves and dries out.

Biology

The fungus survives as pycnidia and pseudothia in infected debris and seeds of chickpea. Dissemination of conidia released by pycnidia and ascospores released from pseudothecia cause primary infection. Secondary infections are due to conidia liberated and disseminated by wind and rain from pycnidia produced by the fungus on growing chickpea. High humidity and temperatures around 20 °C are favorable to this disease which is seedborne.

Chemical control

Seed treatment: Treatment with fungicides based on carboxin, captan, thiabendazole, thiram.

Foliar treatment: Treatment with fungicides based on azoxystrobin, chlorothalonil, metconazole.

* Rhaïem A., Chérif M., Harrabi M. & Strange R., 2006 - First report of *Didymella rabiei* on chickpea debris in Tunisia. Tunisian Journal of Plant Protection, 1: 13-18.

PEA (YELLOW) SPOT

The fungus: *Ascochyta pisi*

A. pisi belongs to the Anamorphic Fungi and the group of Coelomycetes (conidia in pycnidia). Conidia of *A. pisi* are bicellular, elongated, straight or slightly curved with rounded tips. Their sizes are 11-16 x 3-6 µm (Photo 58) (Nasraoui* & Mlaiki, 1983).

Symptoms

Observed on pea, this disease appears as beige yellow lesions which are rounded on leaves, elongated on stems and rounded hollowed in the tissue on pods. They are surrounded by dark brown border. A dark brown to black punctuation corresponding to the pycnidia of the fungus appears in the lesions (Photo 59).

Biology

Primary infection is caused by conidia of the fungus released from pycnidia surviving in seeds and infected debris. The secondary infections are due to conidia disseminated by wind and rain. These conidia are liberated from pycnidia produced by the fungus on growing pea. Temperatures around 20 °C and high humidity are favorable to this disease. However, low temperatures (near 10 °C) seem to be more unfavorable to the host plant than to the fungus. It is a seedborne disease.

Chemical control

Seed treatment: Treatment with fungicides based on carboxin, captan, thiabendazole, thiram.

Foliar treatment: Treatment with fungicides based on azoxystrobin, chlorothalonil, metconazole.

* Nasraoui B. & Mlaiki A., 1983 - L'anthracnose du pois en Tunisie: Identification des espèces cryptogamiques responsables. Annales de l'INRAT, 56: 16 p.

PEA BLIGHT

The fungus: *Mycosphaerella pinodes*
(Anamorph: *Ascochyta pinodes*)

The anamorph *A. pinodes* belongs to the Anamorphic Fungi and the group of Coelomycetes (conidia in pycnidia). Conidia of *A. pinodes* are elongated, straight or slightly curved with tips generally rounded or sometimes slightly pointed. These conidia are generally bicellular, but conidia with three cells can be observed. Their sizes are 10-15 x 4-6 µm (Photo 60) (Nasraoui* & Mlaiki, 1983).

The teleomorph *M. pinodes* belongs to the phylum of *Ascomycota* (fungi producing ascospores) and the group of Loculoascomycetes (fungi producing their asci in pseudothecia).

Symptoms

Observed on pea, this disease causes on leaves, pods, and even grains dark brown blotches which are circular or in irregular shape, without definite border. On stems and crown, blotches are rather elongated. A black punctuation forming the pycnidia of the anamorph appears in the blotches (Photo 61).

Biology

The fungus surviving in seeds and infected debris causes the primary infection via conidia released from pycnidia and/or ascospores released from pseudothecia. During the growing of pea, conidia liberated from pycnidia (produced on infected pea) and disseminated by wind and rain, cause the secondary infections. High humidity and temperatures around 20-25 °C are favorable to this disease which is seedborne.

Chemical control

Seed treatment: Treatment with fungicides based on carboxin, captan, thiabendazole, thiram.

Foliar treatment: Treatment with fungicides based on azoxystrobin, chlorothalonil, metconazole.

* Nasraoui B. & Mlaiki A., 1983 - L'anthracnose du pois en Tunisie: Identification des espèces cryptogamiques responsables. Annales de l'INRAT, 56: 16 p.

BLACK STEM OF PEA

The fungus: *Phoma pinodella*

The anamorph *P. pinodella* (formerly *Phoma medicaginis* var. *pinodella* and more formerly *Ascochyta pinodella*) belongs to the Anamorphic Fungi and the group of Coelomycetes (conidia in pycnidia). Conidia of *P. pinodella* are elongated to ellipsoidal, monocellular, rarely bicellular. Their sizes are 3-10 x 2-5 µm (Photo 62).

The teleomorph, not named, is *Mycosphaerella*-like. It belongs to the phylum of *Ascomycota* (fungi producing ascospores) and the group of Loculoascomycetes (fungi producing their asci in pseudothecia).

Symptoms

Observed on pea, this disease that we were the first to identify in Tunisia (Nasraoui* *et al.,* 2006), is characterized by a brown color that covers all the stem basis of the plant from the crown until few centimeters higher. Irregular brown blotches appear also on leaves, mainly the basal ones, which soon after become yellow and dry out (Photo 63). If the infection is heavy, it reaches the whole aerial part including pods and grains. In the blotches, dark to black dots form and correspond to pycnidia of the anamoph.

Biology

Primary infection is caused by the fungus surviving in seeds and infected debris as pycnidia and/or pseudothecia. Conidia released from pycnidia and/or ascospores released from pseudothecia cause primary infection. Secondary infections are due to conidia liberated and disseminated by wind and rain from pycnidia produced by the fungus on growing pea. Temperatures around 10-15 °C and high humidity are favorable to this disease which is seedborne.

Chemical control

Seed treatment: Treatment with fungicides based on carboxin, captan, thiabendazole, thiram.

Foliar treatment: Treatment with fungicides based on azoxystrobin, chlorothalonil, metconazole.

* Nasraoui B., Srarfi F, Aloui S. & Kharrat M., 2006 - First Report of Pea Black Stem due to *Phoma pinodella* in Tunisia. Tunisian Journal of Plant Protection, 1 (2) (*in press*).

FABA BEAN RUST

The fungus: *Uromyces viciae-fabae*

U. viciae-fabae belongs to the phylum of *Basidiomycota* (fungi producing basidiospores) and the class of Uredinomycetes (fungi having teliospore stage). *U. viciae-fabae* produces urediospores which are monocellular, spherical, ovoid to slightly ellipsoidal, with thick and slightly echinulate wall. Their sizes are 20-30 x 18-20 µm (Photo 64). Teliospores of *U. viciae-fabae* are monocellular, ellipsoidal to ovoid, rarely spherical and sometimes cylindrical. They are extended at the basis by long pedicel and have a smooth wall which is thicker at the top. Their sizes are 25-38 x 20-25 µm (Photo 64).

Symptoms

Observed on faba bean, this disease appears on leaves as small pustules slightly elongated or more frequently rounded, firstly white pinkish and then, after the break down of the host plant epidermis, become brown reddish. They are uredia which produce urediospores. These uredia are irregularly dispersed or form concentric circles. When the infection is heavy, uredia can cover the stems and even the pods. On pods, pustules are wider with irregular shapes and are usually accompanied by fissures of various shapes which hollow in the pod tissue (Photo 65). Later on, as long as faba bean plant gets mature and dries out, dark brown to black pustules appear. They are telia which produce teliospores (Photo 65).

Biology

Faba bean rust is autoecious and macrocyclic. Its life cycle is realized completely on faba bean through characteristic stage spores of rust. Primary infection is caused by surviving teliospores which after germination release basidiospores that infect faba bean. Then, the fungus goes rapidly trough spermatium and aeciospore stages to reach urediospore stage observed on plant and which is the responsible of the damages caused by the disease on faba bean crops. In addition, it is not excluded that the mycelium survives in infected debris and resumes its activity by producing urediospsores which cause primary infection. Secondary infections are due to urediospores produced on growing faba bean and carried by the wind through long distances. At the end of the crop cycle, the fungus produces teliospores to survive. A humid weather and temperatures around 20 °C are favorable to the disease which is normally not seedborne, unless marginally.

Chemical control

Foliar treatment: Treatment with fungicides based on azoxystrobin, cyproconazole, metconazole, tebuconazole.

CHICKPEA RUST

The fungus: *Uromyces ciceris-arietini*

U. ciceris-arietini belongs to the phylum of *Basidiomycota* (fungi producing basidiospores) and to the class of Uredinomycetes (fungi having teliospore stage). Urediospores of *U. ciceris-arietini* are monocellular, spherical to ovoid and sometimes slightly elongated with a slightly echinulate thick wall. Their sizes are 17-32 x 13-24 µm (Photo 66). Teliospores are monocellular, spherical to ovoid or slightly elongated and sometimes angular with echinulate thick wall. They are generally extended by short pedicel. Their sizes are 18-30 x 12-24 µm (Photo 66).

Symptoms

Chickpea rust, that we were the first to report in Tunisia (Nasraoui* *et al.*, 1997), appears as brown pustules which form on the two sides of the leaves. Pustules are dispersed irregularly or disposed as concentric circles which can become coalescent. These pustules are uredia producing urediospores after the break down of the host plant epidermis (Photo 67). At late-spring, as long as chickpea plant gets mature and dries out, black pustules appear. They are telia producing teliospores (Photo 67).

Biology

Chickpea rust is autoecious and microcyclic. Primary infection is probably due to mycelial fragments surviving in infected chickpea debris. Active mycelium would resume its activity and produces urediospores which cause primary infection. Secondary infections are caused by urediospores produced on growing chickpea and carried by the wind through long distances. It seems that teliospores produced by the fungus for surviving do not play a role in the occurrence of primary infection. A humid weather and temperatures around 20 °C are favorable to the disease which is not seedborne.

Chemical control

Foliar treatment: Treatment with fungicides based on azoxystrobin, cyproconazole, metconazole, tebuconazole.

* Nasraoui B., Ganouni H., Achouri A. & Terashima T., 1997 - Une maladie nouvellement observée en Tunisie: La rouille du pois chiche. Revue de l'INAT, 12: 145-149.

PEA RUST

The fungus: *Uromyces pisi-sativi*

U. pisi-sativi belongs to the phylum of *Basidiomycota* (fungi producing basidia) and to the class of Uredinomycetes (fungi having teliospore stage). Urediospores of *U. pisi-sativi* are monocellular, spherical to ellipsoidal with a slightly echinulate wall. The sizes are 20-25 x 18-22 µm (Photo 68). Teliospores of *U. pisi-sativi* are monocellular, spherical to ellipsoidal with a smooth and thick wall. They are extended by generally short pedicel. Their sizes are 20-28 x 17-20 µm (Photo 68).

Symptoms

Observed on pea, this disease appears on leaves as numerous small pustules which are usually rounded and sometimes elongated with brown reddish color. Pustules are uredia which produce urediospores after the break down of the host plant epidermis (Photo 69). Later on, as long as pea plant gets mature and dries out, black brown pustules resembling to uredia appear. These pustules are telia producing teliospores (Photo 69).

Biology

Pea rust is heteroecious and macrocyclic. The aecian stage of *U. pisi-sativi* infects different plant species of the genus *Euphorbia* as alternate host. Aeciospores produced on the alternate host and carried by the wind, cause primary infection of pea as primary host. Secondary infections of growing pea are due to urediospores produced by the fungus on infected plants and carried by the wind through long distances. Teliospores are produced by the fungus to survive. The next spring, teliospors germinate and release basidiospores able to infect the alternate host. After a spermatium stage, the fungus produces on the alternate host aeciospores that infect pea. Temperatures around 20 °C and high humidity are favorable to this disease which is not seedbone.

Chemical control

Foliar treatment: Treatment with fungicides based on azoxystrobin, cyproconazole, metconazole, tebuconazole.
