WHEAT DISEASES IN AGRICULTURAL SYSTEMS

Monograph

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OPOLE/KYIV 2020

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Opole/Kyiv 2020

Approved for publication by Academic Councils
D.K. Zabolotny Institute of Microbiology and Virology of the NASU
Protocol № 7 of October 8, 2019.
Institute of Science, Technical University of Opole, Poland
Protocol № 11 of November 12, 2019

ISBN 978-83-7395-867-8

Wydawnictwo Uniwersytetu Opolskiego, 45-365 Opole, ul. Dmowskiego 7–9.
Copies: 100 egz. Edition I.
Place an order for: tel.: 77 401 67 46, e-mail: wydawnictwo@uni.opole.pl
Printing and binding: Totem.com.pl

Content

List of conditional abbreviations

WSMV	–	wheat streak mosaic virus;
HC-Pro	–	helper-component proteinase;
VPg	–	viral protein of genome-bound proteinase;
NIa	–	nuclear inclusion of the putative protease;
NIb	–	nuclear inclusion of putative polymerase;
CP	–	coat protein
PGD	–	pale green dwarfism of wheat
HPWMoV	–	high plains wheat mosaic virus
BSMV	–	barley stripe mosaic virus
BMV	–	brome mosaic virus
WDV	–	wheat dwarf virus
BYDV	–	barley yellow dwarf virus
DNA	–	deoxyribonucleic acid
PA	–	potato agar
HR	–	hypersensitivity reaction
UCM	–	Ukrainian collection of microorganisms of D.K. Zabolotny Institute of Microbiology and Virology of the NASU

Introduction

Wheat is one of the most valuable and high-yielding crops. Due to its unique chemical composition, wheat ranks first in the world among grain crops in sown areas and use. Wheat grain is rich in protein (14–18%) and gluten (25–28%), therefore it is widely used in the baking and confectionery industries, as well as for the production of cereals, pasta, noodles and other products. Wheat is used to produce starch, alcohol, gluten, and others. Wheat bran is a highly concentrated feed, and straw is used as litter and feed in animal husbandry. Straw is also used as a building material and for the manufacture of paper. In some areas of Ukraine, winter wheat is used as green fodder.

Wheat is classified by type as ordinary soft or bread *Triticum aestivum* L. and *T. compactum* Host, and and solid *Triticum durum* Dest. [8, 10]. The first two species are hexaploids that contain three genomes AABBDD (n = 21), while the third species is tetraploid *Triticum turgidum* (L.) Thell., containing 14 chromosomes AABB (n = 14). Three species together make up about 90% of the world's wheat [31].

According to the Food and Agriculture Organization of the United Nations (FAO) in 2017–2018 years wheat was the most widely cultivated crop in the world with a harvest of 758.274 million tons, about 200 million hectares were occupied under it [6].

According to Eurostat for 2017 year, the largest wheat producer in the world is the European Union – 142.6 million tons. According to the FAO, China ranks second with the indicator 134.3 million tons, in third place – India (98.5 million tons), further – Russia (85.9 million tons), USA (47.4 million tons) and France (36.9 million tons); Ukraine produced 26.2 million tons wheat, which is about 4% of the world crop.

Wheat productivity depends on many factors, and on average in 2000 year amounted to 2.5 thousand tons/ha, and in 2015 year it increased to 3.07 thousand tons/ha. In Ukraine, 2019 year, the yield of wheat averaged 3.76 t/ha. The maximum yield of this crop was observed in 2017 year in New Zealand – 16.8 t/ha. Increasing wheat yields is a prob-

lem worldwide, since a billion tons of wheat must be produced by 2050 year to provide food.

Wheat can be affected by all types of plant disease agents: fungi, bacteria, phytoplasmas and viruses. At the same time, mixed infections are quite often observed, which can be caused by representatives of different classes of living organisms (for example, mixed bacterial-fungal infections) and various pathogens of the same class. Changes in climatic conditions and intensive farming lead to a change in the spectra of pathogens parasitizing on wheat in a particular region and require a change in the control strategy for wheat pathogens. As a separate problem, the process of transformation of unfavorable biotic factors should be considered, including an increase in the severity of certain diseases that were previously not widespread in Ukraine and occurred sporadically, as well as an increase in the resistance of pathogens to fungicides.

The monograph presented to the reader is based on many years of experience in studying pathogens of wheat diseases by specialists from three institutions: of the Department of Phytopathogenic Bacteria, the Department of Physiology and Systematics of Micromycetes of D.K. Zabolotny Institute of Microbiology and Virology of the NASU, Department of Virology, Taras Shevchenko National University of Kiev, Institute of Environmental and Biotechnology Engineering, University of Opole (Poland). It combines information on fungal, bacterial, mycoplasmal and viral pathogens of wheat diseases that are common in Ukraine and in the world, analyzes the harmfulness of diseases and identifies the possibilities for controlling their pathogens.

The authors hope that the data presented in the monograph will be of interest to plant protection specialists, plant pathologists, microbiologists, breeders, graduate students, students of biological and agronomic specialties.

Chapter 1

Bacterial diseases of wheat (*Triticum aestivum* L.)

Agents of bacterial diseases affect almost all types of wild and cultivated plants. They disrupt the normal course of physiological processes in plants, cause their partial or complete death, lead to an unripe crop, a decrease in the number of fruits and berries, as a result of which the quality of the products worsens and the yield decreases. Wheat – a grain crop on which many types of bacteria parasitize [3, 20, 40, 65].

Phytopathogenic microorganisms can persist for a long time in latent form in vegetative plants and reproductive organs. This is one of the ecological niches of phytopathogens as a primary source of infection. The causative agents of bacterial diseases of wheat are given in Table 1.1

Table 1.1 List of bacterial diseases of wheat

Bacterial diseases of wheat	The causative agent
Basal glume rot of wheat (basal bacteriosis of wheat)	*Pseudomonas syringae* pv. *atrofaciens*
Bacterial leaf blight of wheat (bacterial blotch disease)	*Pseudomonas syringae pv. syringae*
Black chaff of wheat (leaf streak, leaf stripe	*Xanthomonas translucens pv. undulosa, Xanthomonas translucens pv. cerealis*
Yellow ear rot of wheat (tundu disease)	*Rathayibacter tritici*
White blotch of wheat(tan streak)	*Bacillus megaterium pv. cerealis*
Bacterial rot of wheat	*Pectobacterium carotovorum*
Stem & head melanosis of wheat	*Pseudomonas cichorii*
Bacterial mosaic of wheat	*Clavibacter michiganensis subsp. tessellarius*
Spotted bacteriosis of wheat	*Pseudomonas fluorescens*
Brown sheath rot of wheat	*Pseudomonas fuscovaginae*
Bacterial spotting of wheat	*Pantoea agglomerans*
Pink seed of wheat (pink grain of wheat)	*Erwinia rhapontici*
Brown bacteriosis of wheat	*Pseudomonas ramonicum*

Despite numerous reports of bacterial wheat diseases worldwide, studies of the bacteria that cause these diseases remain limited, and quantitative information, such as crop loss and disease epidemiology are often not available [49, 73]. In addition, data on the spread of bacterial diseases often do not correspond to the real state of things, since they are based only on observing the symptoms without isolating the pathogen and confirming its identity [40, 49].

It must be remembered that agents of bacterial diseases can cause both mono infections and mixed infections with pathogens of micromycetic, phytoplasmic and viral origin.

Table 1.2 Differential signs of the genera of phytopathogenic bacteria that cause wheat disease [67]

Test	Phytopathogenic bacteria, genera			
	Pseudomonas	*Xanthomonas*	*Pectobacteruim Erwinia*	Clavibacter
Diseases symptoms	Leaf spots. Vascular wilts. Soft rots.	Leaf spots. Vascular wilts. Steam cankers.	Vascular wilts. Dry necroses. Leaft spots. Soft rots.	Guming of influoresces. Wilts and/or leaft spots.
Gram staining	–	–	–	+
Motility	+	+	+	variable
Flagelation	One or several polar	One polar	Peritrichous	Few polar or lateral
Colony color	White or yellow	Yellow (rarely white)	White or yellow	Orange, yellow or blue
Diffusible pigments	Fluorescent or phenazine or absent	Usually absent	Usually absent. Pink or blue in some species.	Usually absent.
Poly-ß-hydroxybutyrate	variable	–	–	–
Oxidase	variable, – (*P.syringae*)	– (or weak +)	–	–
Glucose metabolism	Oxidative	Oxidative	Fermentative	Weak oxidative or inert
Starch hydrolysis	variable	variable	–	variable
Nitrite from nitrate	–	–	+ (*P.carotovorum* and other soft-rotting species); – (most other *Erwinia* sp.)	–
3-keto lactose	–	–	–	–
G+C in the DNA	58–70	63–69	50–58	65–75

The most common bacterial pathogens of wheat in the world: *Pseudomonas syringae* pv. *atrofaciens*, *P. syringae* pv. *syringae, Xanthomonas translucens* pv. *undulosa* [49, 61, 73]. In Ukraine, most often as the causative agent of bacterial diseases of cereals occurs *P. syringae* pv.

atrofaciens [25]. The differential signs of phytopathogenic bacteria that are the causative agents of wheat diseases are shown in the Table 1.2.

1.1 Basal glume rot of wheat

Basal glume rot of wheat (basal bacteriosis of wheat), agent – *Pseudomonas syringae* pv. *atrofaciens* (**outdated pathogen name** *Pseudomonas atrofaciens*).

Bacteria *Pseudomonas syringae* are one of the most common and harmful phytopathogens. One of the main causative agents of bacterial diseases of wheat in Ukraine and the world for many years remains *Pseudomonas syringae* pv. *atrofaciens* – causative agent of basal bacteriosis [25, 49, 61, 73]. The causative agent is distributed on wheat in Russia [62], in Bulgaria [41, 75], in Central Europe [44, 72, 77], in Iran [59]. This phytopathogen affects plants in all phases of development, reduces yield and degrades grain quality. According to our data, the causative agent of basal bacteriosis is common in all studied regions of Ukraine (Fig. 1.1) and causes wheat damage in both intensive and organic methods of growing this culture.

Permanent presence of *P. syringae* pv. *atrofaciens* on cereals and non-grain crops and the threat of epiphytotics when weather conditions favorable for the development of the pathogen occur make it necessary to study all ecological niches that can be colonized by the pathogen [70].

Fig. 1.1 Map showing the identification of *Pseudomonas syringae* pv. *atrofaciens* on wheat and rye (according to employees of the department of phytopathogenic bacteria of D.K. Zabolotny Institute of Microbiology and Virology of the NASU)

The disease was first discovered by McCuloch in 1920 on plants in various regions of the United States and Canada. Later, wheat lesions in the form of browning or blackening of the lower part of glumes and grains were found in Ukraine. The disease is widespread in England, Australia, Africa, Belarus, Belgium, Bulgaria, Canada, Germany, New Zealand, Russia, Romania, Syria, the USA, Ukraine, France, Yugoslavia. The defeat with symptoms of basal bacteriosis is up to 15%, but increases in years favorable for the development of the pathogen and in some years reaches 80%. When infected at the stage of milky-wax maturity, the symptoms of the disease spread to the grain and it becomes underdeveloped, as if charred, the germ of the grain perished. With a strong degree of development of the disease, 10 to 80% of wheat ear are affected. Basal bacteriosis worsens the physical, technological and biochemical properties of wheat grains, reduces the mass of grains, germination energy and seed germination. It was revealed that the degree of development of bacteriosis depends on climatic and weather environmental conditions. The disease develops most in years with elevated temperatures in spring and summer, with high rainfall and high humidity.

Symptoms of the disease. A characteristic sign of basal bacteriosis is damage to the lower part of the glumes, less often – damage to the upper part, and spotting of various parts of vegetative plants. On plants in the sprouting and tillering phase of *P. syringae* pv. *atrofaciens* is rarely parasitizes. Elongated spots form on the leaves – transparent, watery, oily, brown, whitish or yellow, which eventually lengthen, dry out, turn brown, a brown or red-brown edging appears along their edge [5, 13]. Sometimes dark strokes are formed, which color the lower part of the stem in dark color. Shapeless brown, less often black, elongated stripes, spots and strokes can form on the leaves, stem of the ear wheat. Most often and most severely, spikelet and flower scales are affected on the surface of which free-standing small spots of black or brown color appear [30]. With a severe degree of damage, a continuous browning of almost all the glumes, as well as the awns and stem of wheat ears is formed, the spike is deformed, a flat, brown grain with a darker germ develops in it (Appendix 1.1). Occasionally, a dry rotting of the wrapping sheet occurs, with the upper leaves wrinkling and acquiring a yellow-brown color, stripes at the point of attachment of wheat ears to the stem and dwarfism of plants with the formation of raspberry-brown glumes and grains. Pathogenic strains of *P. syringae* pv. *atrofaciens* and fungi from genera *Alternaria*, *Fusarium* and *Drochslera* may coexist in lesions [41]. Symptoms similar to those formed by *P. syringae* pv. *atrofaciens* may cause the agent of bacterial leaf blight of wheat *P. syringae* pv. *syringae* [64].

P. syringae pv. *atrofaciens* can affect the same wheat plant at the same time with *Xanthomonas translucens.*

The nature of the effect of bacteria on wheat tissue depends on the sensitivity of the variety. Bacteria that enter the intercellular space of a susceptible variety multiply intensively and lead to maceration of the cells of the main parenchyma. This is manifested in the formation of brown rounded and longitudinal spots. With severe damage, bacteria populate the conducting bundles, which leads to the formation of spots on various parts of plants, at a considerable distance from the site of inoculation. In the resistant variety of bacterial reproduction in the intercellular space is delayed, from the parenchymal cells and the intercellular space around the site of the lesion formed cork tissue, which limits the further spread of the pathogen and inhibits its development [27].

P. syringae pv. *atrofaciens* persists in affected seeds, stubble, and non-decayed plant residues. Often happens as an epiphyte on healthy wheat seeds and plants [3, 24, 77]. In the soil, the pathogen quickly perished. Infection of wheat under natural conditions comes from affected plants by insects, wind, rain and irrigation water. The main source of spread of the pathogen is the seeds in which it is stored for up to three years. In non-rotten plant residues, bacteria survive up to 10 months. For a long time, the pathogen persists in wild species of cereals [9].

During artificial inoculation of a large number of wheat varieties, it was found that all varieties are sensitive to *P. syringae* pv. *atrofaciens* to varying degrees [7, 19]. In natural conditions, in addition to wheat, the causative agent of basal bacteriosis affects rye, barley, oats and weeds growing in wheat crops (trailing bindweed, field thistle, sow thistle, common horsetail, shepherd's Purse, lambsquarters, couch grass, cleavers wild radish, barnyard grass) [22].

With artificial inoculation of *P. syringae* pv. *atrofaciens* affects oats, Sudanese grass, sorghum, corn, rice, Setária itálica, from grasses – annual and pasture ryegrass, meadow bluegrass, fescue, timothy meadow and weeds.

It was established that from the legume genera soy and beans are susceptible to the pathogen, to a lesser extent – peas, fodder beans, chickpeas, all types of lupine, as well as other plants – tobacco, beets, tomatoes; green pear fruits, onions, sprigs of lilac and others.

Taxonomic position and biological properties *Pseudomonas syringae* pv. *atrofaciens.* The causative agent of basal bacteriosis of wheat belongs to the species *P. syringae*, which is a common pathogen of many plants. At first, considering the pathogenic properties of bacteria classified as *P. syringae*, they were divided into 40 pathovars [79].

Subsequently, the use of the latest research methods made it possible to establish the existence of the so-called "*P. syringae* complex", which includes up to ten species of *Pseudomonas* and 60 patovars *P. syringae* [80]. Among the patovars of *P. syringae*, three are cause to wheat damages: *Pseudomonas syringae* pv. *atrofaciens, Pseudomonas syringae* pv. *japonica, Pseudomonas syringae* pv. *syringae* [49].

P. syringae pv. *atrofaciens* is a mobile aerobic gram-negative bacteria, located singly and in short chains, do not form spores. After two or three days, on the potato agar they form gray, transparent, smooth, rounded colonies, with slightly wavy edges, sometimes with a raised center (Fig. 1.2).

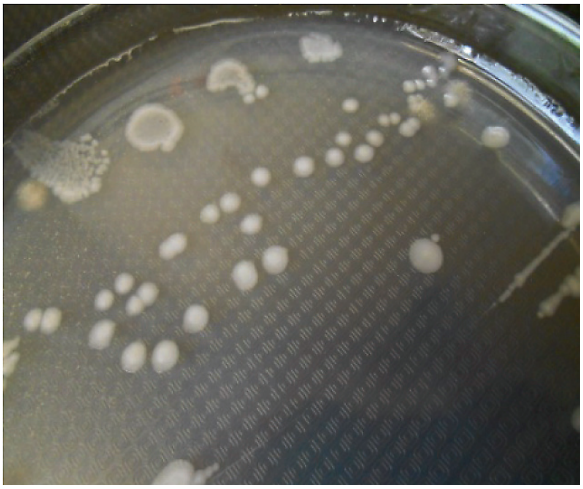

Fig.1.2 Colonies of *Pseudomonas syringae* pv. *atrofaciens*

P. syringae pv. *atrofaciens* oon the litmus serum form an alkali, is peptonize of milk, form a fluorescent pigment, do not form indole and hydrogen sulfide, most of *P. syringae* pv. *atrofaciens* strains are hydrolyse gelatin. Bacteria under aerobic conditions use glucose, mannose, arabinose, sucrose, mannitol, sorbitol, inositol as the sole source of carbon nutrition and do not ferment lactose, rhamnose, dulcitol, salicin, inulin, maltose (Table 1.3).

When studying a large number of signs of phytopathogenic bacteria of the genus *Pseudomonas*, five were identified that can be successfully used to differentiate these bacteria into groups: levan formation, oxidase activity, maceration of potato pieces, arginine dihydrolase formation and a hypersensitivity reaction on tobacco leaves (the so-called LOPAT-test). This approach is based on the definition of five features: the ability to form levan; the presence of oxidase; maceration of plant

tissues, the presence of arginine dehydrolase; induction of hypersensitivity reactions (HR) in tobacco leaves.

Table 1.3 Properties of bacteria isolated from wheat

Tests	*P. syringae* pv.		*P. viridiflava*	*P. fluorescens*	*X. translucens*
	atrofaciens	*syringae*			
Gram staining	–	–	–	–	–
Motility	+	+	+	+	+
Nitrate reduced	–	–	–	–	–
Oxidase	–	–	–	+	–
Litmus serum	Al	Al	Al	Al	n/i
Gelatin hydrolysis	+	+	+	+	–
Peptonization of milk	+	+	+	+	+
H_2S from cysteine	–	–	–	–	n/i
Indole	–	–	–	–	–
Utilization of: glucose	A	A	A	A	A
Mannitol, sorbitol	A	A	A	A	–
Xylose	A	A	A	n/i	–
Dulcitol	–	–	–	–	n/i
Fructose, arabinose, galactose	A	A	A	A	A
Lactose, rhamnose, inulin, salicin	–	–	–	–	–
Sucrose	d	A	A	d	A
Maltose	–	–	–	d	A
Raffinose	d	A	–	d	d
Inositol	A	A	A	d	–
Pectolytic activity	–	–	+	–	–

Notes: here and in the Table 1.6: – – gram negative, + – all strains are positive, – – all strains are negative, d – 11–89% of strains are positive, A – carbon consumption with acid formation, Al – fermentation of compounds with the formation of alkali, n/i – not investigated.

According to these characteristics, phytopathogenic bacteria of the genus *Pseudomonas* are divided into several species group. Species characterized by the absence of oxidase and arginine dihydrolase, the inability to potato macerate, and the ability to induce HR are assigned to group 1 LOPAT (Table 1.4). Further studies showed that these species differ in pathogenicity, but they cannot be divided according to the use of biochemical and physiological-cultural tests. These species were combined into one species *P. syringae*, with division into patovars [80].

Table 1.4 Characterization of phytopathogenic bacteria of the genus *Pseudomonas* for LOPAT-test

Representatives	Biochemical tests				
	L	O	P	A	T
Pseudomonas syringae pv. *atrofaciens*, *P. syringae* pv. *syringae*	+	–	–	–	+
P. viridiflava	–	–	+	–	+
P. cichorii	–	+	–	–	+
Pseudomonas fluorescens	+	+	–	–	–

Notes: L – levan formation; O – the presence of oxidase; P – tissue maceration; A – arginine dehydrolase; T – hypersensitivity reaction.

Many researchers point to the close relationship of *P. syringae* patovars by biochemical, physiological, and even genetic characteristics. In recent years, works have appeared in the literature that indicate that the genetic relationship within the *P. syringae* species does not coincide with its division into patovars [80].

Thus, the division of *P. syringae* into patovars does not coincide with the data from the study of the genomes of representatives of this species [80]. Today it is difficult to predict whether the taxonomy of the *P. syringae* species will be based on the study of phenotypic characters, will only take into account data on the sequence of nucleic acids in the genomes [80], and such a taxonomic unit as the pathovar within the species *P. syringae* will be preserved. It illustrated the close relationship of basal bacteriosis of wheat pathogen *P. syringae* pv. *atrofaciens* with *P. syringae* pv. *syringae* for phytopathogenic properties, toxin formation, fatty acid profile and protein profile.

Of great importance for the identification and diagnosis of the causative agent of basal wheat bacteriosis is the study of its serological properties. Despite the fact that the researchers tried to develop schemes for serotyping phytopathogenic bacteria of the genus *Pseudomonas*, to date there is no single universally accepted scheme for serogrouping this type of bacteria.

According to the scheme of serogrouping of phytopathogenic bacteria of the genus *Pseudomonas* [26], strains *P. syringae* pv. *atrofaciens,* isolated from wheat belong to the serogroups of II, IV, V and VI (Table 1.5). Strains of serogroup I and V have only antigens specific for these groups, strains of serogroup II, IV, VI, except for specific ones have common intergroup antigens. To identify the causative agent of basal wheat bacteriosis, it is necessary to use a multi-strain serum for representatives of four serogroup *P. syringae* pv. *atrofacien.*

Table 1.5 The results of the double diffusion reaction on agar of antigens of *P. syringae* pv. *atrofaciens* from wheat with antisera to *P. syringae*

P. syringae pv. *atrofaciens* strains, isolated from wheat	The number of precipitation lines with antisera to strains of *P. syringae*, serogroups			
	К-1025, II	4394, IV	948, V	7194, VI
Serogroup II	3	0	0	0–1
Serogroup IV	0	2–3	0	1
Serogroup V	0–1	0	2–3	0
Serogroup VI	0–1	0–1	0	1–2

We carried out an analysis of the serological characteristics of *P. syringae* pv. *atrofaciens* isolated from wheat agrophytocenoses under various farming systems, and *P. syringae* strains, isolated from weeds in wheat crops. Based on the results, the largest number of *P. syringae* pv. *atrofaciens* (48%), isolated from affected wheat grown under the intensive farming system, assigned to serogroup IV, less (37%) to serogroup II (Fig. 1.3). Bacteria isolated from wheat that was grown for the use of organic farming are divided into four serogroups (II, IV, V, VI) (Fig. 1.3). The largest number of strains are assigned to serogroup IV (52.4%), less to serogroup VI (23.8%). The strains that belong to serogroup II and V are 14.3% and 9.5%, respectively.

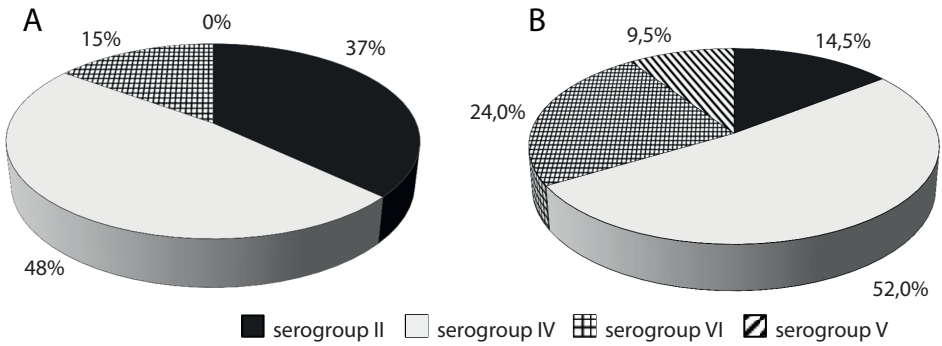

Fig. 1.3 Distribution of *P. syringae* pv. *atrofaciens* isolated from wheat grown under the intensive farming system (A) and grown according to the organic farming system (B), according to serological affiliation

1.2 Bacterial leaf blight of wheat

Bacterial leaf blight of wheat (bacterial blotch disease), agent – *Pseudomonas syringae* pv. *syringae* (outdated pathogen names: *Pseudo-*

monas syringae, P. striafaciens var. *japonica, P. syringae* pv. *japonica*). A leaf blight of wheat was first discovered in 1974 in the USA, later in Russia, Yugoslavia, Bulgaria, Bangladesh; at first it was mistakenly described like the rot of ear glumes.

Symptoms of the disease are gray-green necrosis and whitening of leaves, glumes of ear, leaf sheath and stem of wheat [64]. Leaf damage can reach 50%. *P. syringae* pv. *syringae* often causes water saturated spots. Phytopathogenic bacteria on ears of wheat caused white spots [42]. Brown oblong spots, located on the edge of the leaf blade or in its middle part, without the edging are more often formed on wheat [34]. The color of the spots and the presence of edging depends on the type and variety of plants. The leaves of the lower and middle tiers are more affected. With the strong development of the disease, individual leaves, often lower ones, can die, the whole plant rarely perished off (Appendix 1.2).

The causative agent penetrates the plant through the stomata and multiplies in the intercellular space of the parenchyma. In the tissue of the upper floral glumes bacterial cells are able to move from one side to the other. First, the bacteria infect the upper and lower flower glumes, then through the seminal cord they penetrate into the caryopsis and multiply in the intercellular space of the caryopsis parenchyma, but not in the vascular bundles [55]. Phytopathogenic bacteria move from the affected seeds to seedlings and can survive on healthy leaves, which are a source of infection bacterial leaf blight [54].

The biological properties of the causative agent of leaf blight of wheat are shown in Table 1.3.

Some strains of *P. syringae* pv. *syringae* can affect barley, rye and oats. By artificial inoculation, the pathogen affects wheat, beans, tomatoes, peppers, lemon, onions and peach twigs. When apricot was inoculated, necrosis of phloem, xylem, cambium, increase in ulcers, and perished of infected branches were observed [43].

Symptoms of wheat damage characteristic of *P. syringae* pv. *syringae* similar to the lesions that cause fungi of the genera *Helminthosporium* and *Septoria*.

1.3 Black chaff of wheat

Black chaff of wheat (black bacteriosis, leaf streak, leaf stripe), agent: *Xanthomonas translucens* pv. *undulosa*, *Xanthomonas translucens* pv. *cerealis*, *Xanthomonas translucens* pv. *translucens*.

The first information about the disease that causes blackening of wheat in Russia appeared in 1911 [39]. Black chaff of wheat was first

described by E. Smith in the USA in 1917, and its pathogen *X. translucens* pv. *undulosa* was determined, which turned out to be pathogenic for wheat and barley. In Canada, black bacteriosis was first discovered in 1922. But under natural conditions can strikes wheat *X. translucens* pv. *cerealis* [56]. Both the agents *X. translucens* pv. *undulosa* and *X. translucens* pv. *cerealis* cause black chaff of wheat, but slightly differ in the speed and intensity of biochemical reactions and aggressiveness in crops. Much later, numerous reports of black bacteriosis began to appear in other countries – France, Belgium, Sweden, Hungary, Germany, Australia, Ukraine, Georgia, and others. It was shown that *X. translucens* pv. *undulosa* affected wheat more than *P. syringae* pv. *atrofaciens* and also occurs on wheat more often than on barley and rye [56, 57]. In Russia and Ukraine, black bacteriosis was diagnose more than once, but this disease is not widespread in these regions [2, 16]. For example, in Russia (Bashkorstan) black bacteriosis predominates, and in the Kirov region basal bacteriosis prevails [15]. Black chaff of wheat is registered in all areas of wheat cultivation in most countries of America, Europe, Asia, Africa and Australia. The harmfulness of black bacteriosis is manifested in a decrease in the total number and length of ears, the number of grains in the ear of wheat. The formation of not fully developed grains causes a reduction in the mass of 1000 grains by 60–62%. The amount of water in the affected grain decreases by 3.7%, protein – by 2.7%, glucose – by 3.9%, starch – by 10.4%. Wheat yield is reduced by 15–90% depending on the variety, geographic location and climatic conditions. The development of the disease contributes to high relative humidity (70–80%), temperature (25–30°C), excessive application of nitrogen fertilizers. According to Russian researchers, crop losses did not exceed 10–15%, with the exception of years with hot summers, when crop losses are higher [18]. It was revealed that 50% of the damage to the leaf surface of the flag leaf of wheat leads to a yield loss of 8–13%, and 100% – to 13–34%, depending on the resistance of the varieties [69].

Symptoms of the disease. *X. translucens* pv. *undulosa* affects all organs of vegetative wheat plants. The disease rarely develops on plants in the seedling phase. On the leaves in the tillering phase, watery light green spots are formed, which increase in size, become chlorotic, dark yellow, brown, sometimes with a black edging, and also black. Sometimes transparent stripes appear on the leaves, which eventually turn yellow and turn brown. On mature plants, spots often develop on the lower and upper leaves. In the wheat ripening phase, the affected tissues turn yellow and are difficult to distinguish on the yellow surface of dry leaves. On the stem, the disease most often manifests itself in

the flowering phase at the base of the leaves in the form of black dots, which merge to form a black-brown spot. Sometimes black or brown longitudinal stripes appear on the stem, under the nodes of the leaves, which extend to the entire straw. Often on spring wheat with a red spike, the base of the leaves and the stem acquire a purple hue.

Most often, the symptoms of the disease appear on the ear wheat (Appendix 1.3), where black and brown spots or wide stripes elongated along the scales are formed in the upper part of the glumes. With a severe defeat, they merge at the top of the glumes and can spread to the awns, which become dark brown, pressed to the ear of wheat and 1/3 of the length move away from it to the side. With a severe defeat, browning or blackening covers the entire ear of wheat, which decreases, deforms, fewer caryopsis are formed in it, awns are bent, the grain becomes underdeveloped, sometimes with yellow stripes. In a humid chamber, the affected seeds are covered with mucus and droplets of light yellow exudate protrude on them [4]. In dry weather, the germinal part of the grain acquires a dark gray color (gray embryo), and with increased humidity – dark brown (brown embryo). The affected part of the grain is separated by a dark border from the healthy one. Sometimes underdeveloped shriveled seeds have grooves on the surface [10]. Under natural conditions, in wet weather, exudate appears on the grain in the form of whitish or yellowish mucus, which, when dried, turns into yellowish granules, beads or a gray film. Depending on the wheat variety, various types of lesions are formed on the ear wheat: blackening, often continuous, which is inherent in winter wheat varieties; the appearance of brown, which is inherent in soft spring wheat.

Under natural conditions, *X. translucens* affects weeds (sow thistle, Veronica chamaedrys and field thistle), growing in wheat crops [29].

Pathogen *X. translucens* pv. *undulosa* represents cylindrical, mobile, short, small, gram-negative rods with rounded ends, which are form on potato agar the colonies of yellow color round, convex smooth, shiny, oily, mucous, viscous with smooth edges. The biological properties of the causative agent of black chaff of wheat are shown in Table 1.3.

With artificial infection of wheat with strains of *X. translucens* pv. *undulosa* develop symptoms of the disease, which depend on the aggressiveness of the strains, varieties of wheat and weather conditions [38]. In addition to wheat *X. translucens* pv. *undulosa*, with artificial infection, affects rye and barley, strains *X. translucens* pv. *cerealis* also affects oats. Bacteria *X. translucens* pv. *undulosa* isolated from wheat in Russia is pathogenic for rye, spring barley, wheatgrass, beans, corn and is not pathogenic for oats, potatoes, beets, carrots, cabbage, radishes and pumpkins [37]. Strains isolated from wheat in Georgia are

pathogenic for corn, sorghum, beets, beans, peas, tomatoes and cucumbers [21].

Pathogens penetrate into the grain through the damaged pericarp shell, and are localized in the intercellular spaces of the grain, in parenchyma cells, and infect seedlings through the stomata of coleoptile [21]. In the affected tissues, bacteria fill the intercellular space, vascular bundles, tissue of the main parenchyma and phloem, causing their violation [5].

There are difficulties in diagnosing black chaff of wheat, since lesions in the form of tissue darkening can be caused by micromycetes, other bacteria, meteorological conditions, high temperature, high humidity, genetic changes, and can also have a physiological character. Such darkening of tissues differs from bacterial damage in the absence of bacteria in them and does not always lead to a decrease in yield.

The development of black bacteriosis depends on soil, climatic conditions, fertilizers, timing of sowing, and other agricultural techniques. High temperature and humidity create favorable conditions for the development and spread of black bacteriosis. Mild winters and heavy rains contribute to the development of the disease, which lengthen the growing season of wheat. The defeat of wheat *X. translucens* pv. *undulosa* depends on wheat varieties and their botanical forms [7]. The main source of infection is wheat grain, in which the pathogen persists for up to three years [8, 9]. The causative agent enters the grain from the affected glumes, is transferred from the affected plants to healthy by aphids, insects, rain and wind. Bacteria survive for a long time in plant residues, which serve as a reserve for the infection for the next year and wild types of cereals – *Elytrígia* and *Agropyron*. Under susceptible conditions, the pathogen in wheat grains can will remain from three to five years [52]. In wheat seeds, bacteria are preserved and multiply inside the embryo, where there are many protein substances, and move upward, affecting all above-ground organs and seeds [21]. Bacteria can be localized on the surface of healthy grains (epiphytic survival). Epiphytic populations of *X. translucens* were found on leaves of smooth brome, couch grass, bluegrass meadow, fescue and alfalfa, which grew near wheat fields and did not have disease symptoms [71].

1.4 Yellow ear rot of wheat (tundu disease)

Yellow ear rot of wheat (tundu disease), agent – *Rathayibacter tritici* (**outdated pathogen names** *Pseudomonas tritici*, *Corynebacterim tritici*, *Corynebacterim michiganense* pv. *tritici*, *Clavibacter tritici*). For the countries of the Eurasian Union, the agent of yellow mucous bacteriosis is the object of external quarantine.

The disease was first described in 1917 by C. Hutchinson in India. The disease is common in India, Egypt, China, Cyprus, Australia [76]. Infection of plants occurs with the larvae of wheat nematode *Anguina tritici*. In soil, wheat nematodes retain a viability of 5–7 years, and in dry grain – more than 20 years.

Symptoms of the disease. The disease was found in the field of wheat in the form of foci. In the seedlings, a few days after infection at the edge of the leaf and between the veins appear longitudinal white or yellowish discoloration. At first, the affected tissues are shiny, eventually mucus appears and they turn yellow. Highly infected leaves wither and die off. In the following stages of wheat development, white, rarely yellowish, narrow longitudinal strips, folds, twists and slips of leaves, stems and spikes appear at the beginning of the infectious process on leaves and leaf sheaths. The ear of wheat, along with the wrapping leaf, turns yellow, curving, forming a shapeless ugly mass covered with viscous yellow mucus. The seeds in the ear do not develop. In dry weather, the mucus hardens, dries, forms a pellicle. At high humidity a lot of mucus (exudate) is formed and it drips from the plants. Weakly affected plants of wheat lag behind in development and form dwarf ears that are covered with mucus, they rarely form grain, and if it develops, it is always underdeveloped, with dark spots. The most characteristic signs of the disease are manifested in the phase of maturation the wheat. With severe damage, the plants are often not earing and the ear wheat remains in the leaf sheath. The affected plants practically do not yield the crop [1].

R. tritici affects only wheat. The pathogen persists for more than two years in the seeds and galls of wheat nematodes.

The causative agent of *R. tritici* is short monotrichous gram-positive rods with rounded ends, not spore-forming. The cells are arranged in short chains of two or three in the shape of the letter "V". In agar medium forms round, convex, opalescent, bright yellow, shiny colonies with smooth edges.

1.5 White blotch of wheat

White blotch of wheat (tan streak), agent – *Bacillus megaterium* pv. *cerealis* Hosford 1982. The disease was discovered in 1962–1965 in Canada and in 1975 in the United States [58]. With strong plant damage, the wheat crop is greatly reduced.

Symptoms of the disease appear at the booting stage in the form of small white or yellow necrosis, which rapidly enlarge and turn into white or light brown spots and stripes of irregular shape on leaves,

leaf sheath, stems and glumes of wheat. With fever and intense light, the development of the disease symptoms increases. Young, fast-growing wheat plants are more resistant to the pathogen.

Similar symptoms of the disease are caused by the pathogen *P. syringae* pv. *syringae*. Large white spots cause both bacteria on wheat and on tobacco leaves. But the symptoms that cause these pathogens have also have their own peculiarities. *P. syringae* pv. *syringae* causes water-saturated spots, and *B. megaterium* pv. *cerealis* – spots of very large size that occupy most of the leaf [58]. The disease occurs on wheat of spring and winter wheat, and on barley and oats. With artificial infection in three days on the leaves of wheat develop chlorotic spots, which eventually increase and become white with a brown center. In susceptible varieties, visible signs of the disease begin with the formation of small white or yellowish diffuse spots, which become very large in 2–20 days after inoculation. In some cases, *B. megaterium* pv. *cerealis* was found on healthy wheat leaves, indicating epiphytic survival of the bacterium [58]. Bacteria spread through water during rain and insects and ticks.

1.6 Bacterial rot of wheat

Bacterial rot of wheat (stem rot), the agent – *Pectobacterium carotovorum* subsp. *carotovorum* (outdated names of the pathogen *Bacillus carotovorus, Erwinia carotovora* subsp. *carotovora, Pectobacterium carotovorum, Erwinia carotovora* pv. *carotovora*). The disease was detected in different regions of Ukraine and Russia in 1968 [30, 35].

Symptoms of the disease. In early spring, and sometimes in December and January, bacteriosis is manifested in the form of decay of the bush node. Initially, its tissue darkens and becomes light brown or brown. In the lower part of the stem at the level of the soil brown, black, ligth brown or yellowish with brown border are formed small longitudinal spots – areas of rotting tissue. Often yellow stains appear on the stem with a brown border, followed by light brown stripes. As a result, one to two of the first leaves at the bottom turn yellow, brown, lose shape and fade. First, the leaf parenchyma rot, then the conduction bundles, which leads to easy tearing of the aerial part of the plant. The affected plants lag behind in growth, they develop a short ear wheat, which sometimes turns brown. In the ear of wheat wrinkled grains are formed, grain is not formed at all with the strong development of bacteriosis. Wheat damage occurs depending on the conditions of the year at different times. Prolonged autumn, frequent winter thaws with air temperatures above +5 °C contribute to the development of bacterio-

sis. Therefore, the first symptoms of disease in the years with warm winters appear in December – January. These are yellow spots with a brown border, 0.1–1.0 cm in diameter on the stem. The affected areas are rotting. In cold winters with temperatures lower zero, the symptoms of the lesion appear later – in early April. With the growth of plants, the spots on the stem do not disappear, but remain until harvest [33]. Bacteria *P. carotovorum* – short, gram-negative, mobile, straight rods with rounded ends and peretrihial located flagella. Spores and capsules do not form. Colonies of bacteria of the genus *Pectobacterium* and *Erwinia* are gray-white, convex, smooth, shiny colonies with even edges. The biological properties of the causative agent of bacterial rot of wheat are given in Table. 1.6.

Table 1.6 Properties of bacteria isolated from wheat

Tests	*Pectobacterium carotovorum*	*Pantoea agglomerans*
Gram staining	–	–
Mobility	+	+
Nitrate reduction	+	+
Oxidase	–	–
Hydrogen sulfide formation	+	+
Indole	–	–
Gelatine hydrolysis	+	+
Milk coagulation	+	+
Litmus serum	A	A
Pectolytic activity	+	–
Using: Glucose (aerobic and anaerobic)	A	A
Xylose	A	+
Salicin, rhamnose	A	A
Fructose, galactose, arabinose, mannitol	A	A
Lactose	A	A
Inositol	d	A
Sucrose	A	A
Maltose	d	A
Raffinose	A	d
Dulcitol	–	–
Sorbitol	–	d
Inulin	A	–

It was found that *P. carotovorum* subsp. *carotovorum* strains, isolated in Ukraine from the affected leaves of wheat in the seedling phase, is more aggressive than isolated from dark glumes and shriveled grain [30]. The main source of infection is seeds in which *P. carotovorum* subsp. *carotovorum* persists up to 15 months, and in plant residues – up to 10 months [8].

The use of nitrogen fertilizers in combination with phosphorus and potassium reduces the damage of wheat *P. carotovorum* subsp. *carotovorum*. With increasing nitrogen dose, the percentage of affected plants also increases [33].

P. carotovorum affects the leaves of the trailing bindweed, couch grass, lambsquarters and cleavers in wheat crops.

1.7 Stem & head melanosis of wheat

Stem & head melanosis, agent – *Pseudomonas cichorii*. The causative agent was discovered in 1965 on wheat in Canada [66].

Symptoms of the disease. The affected wheat plants are characterized by a discolored empty ear, blackening of the core of the ear, pedicels and stems below the nodes. The first symptoms of the disease appear both in the early stages of growth and in the stage of milk maturity. Small, light brown necrosis of the stem develops below the two nodes. Within two weeks, the affected tissue darkens and the necrosis fuses, spreading to the stem of the ear, peduncle with subsequent spread to glumes. In the phase of milky-wax ripeness, necrosis increases and darkens. The core of ear and stem turn dark brown. The ear of wheat is discolored and the grains shriveled. In some areas, the causative agent causes small dark necrosis on wheat plants [66]. When artificially infected with wheat plants two days after inoculation, the scales, stem spikes, peduncles, and stems become light brown. The small, dark, long-stemmed lesions appear on the third day and merge on the fourth. On the fifth day after inoculation, the wheat stems enlarge to reach a large size (up to 15 cm) after inoculation. The ear of inoculated wheat plants becomes discolored, sometimes glumes are affected and underdeveloped grain is formed.

The epidemiology of wheat stem melanosis has not been elucidated. A wide range of wild-growing *P. cichorii* hosts indicate that inoculum sources may be among the weeds found in the affected wheat areas [66].

1.8 Bacterial mosaic of wheat

Bacterial mosaic of wheat, agent – *Clavibacter michiganensis* subsp. *tessellarius* (outdated name of pathogen *Corynebacterium michi-*

ganense subsp. *tessellarius*). The disease was discovered in 1990 in the United States [46]. The gram-positive coryneform bacterium was isolated from diseased tissues that were ground in sterile water and streaked on medium.

Symptoms of the disease. On the leaves of wheat small yellow lesions with uneven edges are formed, which are densely and evenly arranged on the leaf blade. Small, yellow lesions that sometimes coalesced into streaks were observed in all inoculated plants after incubation for 14 days at 25°C [46]. Pathogen *C. michiganensis* subsp. *tessellarius* by artificial inoculation doesn't affect barley, oats, corn, Sudan grass, wild rye, brome field and tomatoes. Bacteria do not cause hypersensitivity reactions in tobacco leaves that had been injected with a bacterial suspension [46]. *C. michiganensis* subsp. *tessellarius* produces typical orange, mucoid colonies with entire margins on specific media [46]. The primary inoculum is bacteria surviving in host residue, glumes and seed.

1.9 Spotted bacteriosis of wheat

Spotted bacteriosis of wheat, agent – *Pseudomonas fluorescens*. The disease was detected in Ukraine and Russia.

Pathogenic strains isolated from root neck, withered leaves in the seedling phase, affected grain and awns with brown spots have been found in Ukraine [12]. *P. fluorescens* strains have been found to affect stems and cause grain rot, which often loses germination [8]. In natural conditions, *P. fluorescens* affects rye [23] and weeds that grow in wheat crops [29]. The biological properties of the causative agent of spotted bacteriosis of wheat are shown in Table 1.3.

During the artificial inoculation with *P. fluorescens* strains, various symptoms of the disease develop depending on the variety. Chlorotic brown or oiled spots and small brown or light brown spots are formed on stems and leaves of winter wheat. On spring wheat at the site of inoculation the tissue is chlorotic, whitish oiled or beige. However, the ear is not normally developed and often deformed [12].

When artificially infected, bacteria cause barley and rye damage. *P. fluorescens* is involved in complex pathological processes that lead to decay of winter wheat stems and seed damage. In the development of bacteriosis the main role belongs to *P. syringae* pv. *atrofaciens*, and the influence of other saprotrophic bacteria depends on adverse climatic conditions, soil factors and agro-technical means that reduce the resistance of plants and make them susceptible to phytopathogenic bacteria.

1.10 Brown sheath rot of wheat

Brown sheath rot of wheat, agent – *Pseudomonas fuscovaginae*. The disease was found in wheat in 1990 in Mexico [50].

Symptoms of the disease. On the wrapping leaf of wheat, irregularly shaped angular black-brown areas with a violet-black water-saturated border, 1–2 mm wide, are formed. The lesions are necrotic and can reach 10–20 cm in length. The lesion center turns gray and perished. Under the dark field from the border of the lesion bacteria were observed. Infection begins with the adaxial side of the envelope of wrapping sheet, where water is retained in of leaf sheats. The stem and leaf plates are not affected. Old necrosis is often re-infested with fungi. With a high degree of damage observed the appearance of empty ears and sterility. Fluorescent, strictly aerobic bacteria identified as *P. fuscovaginae* (based on their positive reaction for Kovac's oxidase and arginine dihydrolase but negative for esculin hydrolysis and nitrate reduction) were isolated from these lesions [50]. The pathogenic strains from wheat behaved similarly to those isolated from rice in other countries with regard to nonproduction of 2-ketogluconate, acid production from trehalose but not from inositol, agglutination with antiserum against a reference strain, and pathogenicity on rice and wheat [50].

In addition to wheat, *P. fuscovaginae* causes a disease of rice, which is a rather harmful disease.

When artificially inoculated, the pathogen *P. fuscovaginae* causes barley disease [50].

1.11 Bacterial spotting of wheat

Bacterial spotting of wheat, agent – *Pantoea agglomerans* (outdated name of pathogen *Erwinia herbicola*).

The disease has been identified in Russia, Canada and Ukraine [35]. On healthy wheat seeds, this saprotrophic bacterium accounts for 97–100% of the total microbiota. But *P. agglomerans* can take part in the infectious process in wheat, where it causes spotting of leaves and glumes of the ear. By artificial infection of wheat leaves in the seedling phase, strains of *P. agglomerans* form brown elongated small spots. Chlorotic spots develop on the wrapping leaf, leaves and stems of adult plants before flowering, and subsequently – a brown border around them. On the glumes are formed brown or chlorotic spots – strokes [11].

Bacteria *P. agglomerans* – polymorphic mobile, short, small, gram-negative rods with rounded ends and peritrachial flagella. The colonies on potato agar are yellow, round, shiny, smooth, flat with a compacted

and elevated center and with slightly wavy edges. The biological properties of the causative agent of bacterial spotting of wheat are shown in Table 1.6.

By artificial inoculation, *P. agglomerans* turned out to be weak aggressive for various varieties of other crops – barley, rye, millet, and corn. Non-pathogenic the bacteria for *Setária itálica* subsp. *italica*, *moharicum*, oats, rice, sorghum, ryegrass, timothy, fescue [11]. Under natural conditions, the pathogen affects rye, soybeans and weeds that infest crops of wheat (trailing bindweed, lambsquarters, couch grass, field thistle, shepherd's Purse, field pansy) [29].

1.12 Pink seed of wheat

Pink seed of wheat (pink grain of wheat), agent – *Erwinia rhapontici* (outdated name of pathogen *Pectobacterium rhapontici*).

In 1966, bacteria that produce pink pigment in a culture medium were isolated from pink wheat grain in France and England. In 1974, P. Roberts [68] identified isolated bacteria in France and England as *E. rhapontici*. The disease is found in the USA, Canada, Ukraine, Russia and Belgium. Wheat grain with pink pigment is a small percentage, and the disease is not economically important.

Symptoms of the disease. A characteristic feature is reddening of the grain. Pink color covers the entire surface of the grain and softens the endosperm.

Only artificial inoculation of the ear of *E. rhapontici* does a pink-colored grain develop, which can be found in small quantities. It was found that pathogenic strains isolated in England infect only 8% of inoculated grain. At the same time, inoculation with bacterial strains from France leads to the formation of a large amount of pink wheat grain [68]. Italian scientists believe that the symptom of orange glume is associated with a complex of bacteria – *E. rhapontici* and *Xanthomonas campestris* [48]. According to other researchers, bacteria of the of family Enterobacteriaceae and genus *Serratia* are most often found in the affected grain. Some researchers have discovered three species of bacteria in pink grain – *P. syringae* pv. *atrofaciens, X. campestris* pv. *translucens* and *Serratia marcescens* [17]. They believe that such grain can be used for baking and technical purposes, if the content of grains with a pink color does not exceed 12%. There is an opinion that such damage to wheat, barley and other crops is not specific and is associated with adverse environmental conditions under which the surface microbiota of the grain can acquire pathogenic properties [5]. The pink

color of the grain may be due to the use of colored fungicides, as well as infection of plants with fungal species of the genus *Fusarium* [47].

1.13 Brown bacteriosis of wheat

Brown bacteriosis of wheat, agent – *Pseudomonas ramonicum*. The disease is described only in Russia in 1972. It has been established that on the grain of winter wheat of various varieties from Sweden, Finland, Poland, Germany, the USA, Canada, Bulgaria, the spread of brown bacteriosis is 24–34%, the development of the disease is 13–22% and grain loss is 14.9%. The number of productive stems with a strong degree of damage decreases by 72.8%, and the weight of grain from one ear of wheat – by 60.7%. The disease spreads more strongly in warm weather during the formation of seedlings and in early spring when they grow.

Symptoms of the disease. The causative agent of brown bacteriosis affects all organs and causes mainly rot of the stem and grain. In the fall, in humid warm weather or in the early spring after the growth of plants that have wintered, yellow spots with a brown border appear on the stem below, which increase in size and wrap the stem. The first two or three leaves lose turgor and brown oiled spots and stripes appear on them. The leaves turn light green, then turn yellow and dry. Sometimes the stem is evenly painted in dark brown. In the affected plants of wheat, the ear is underdeveloped, the grain is brown and not developed. In ears there are grains with a dark embryo, during the germination of which their surface turns brown and decays, and the embryo perished off. With a severe defeat, empty ear are observed. In addition to *P. ramonicum*, the authors isolated *P. carotovorum* from affected wheat samples with signs of brown bacteriosis. Fungi of the genera *Alternaria, Macrosporium, Fusarium, Cladosporium* can also cause diseases with symptoms of brown bacteriosis. Therefore, rot of the stem and roots, as well as empty ear of wheat are a complex process of brown bacteriosis, in which these fungi and bacteria P. *ramonicum* and *P. carotovorum* participate [8, 37].

The main source of bacterial infection is grain. P. *ramonicum* persists on the surface and internal tissues of the grain for up to 15 months, in non-rotted plant residues – up to 10 months [8, 9, 36]. The pathogen enters the internal tissue of the grain through the damaged pericarp shell, which covers the embryo. When germinating grain, bacteria infect plants. They actively multiply in the intercellular space and vessels.

The defeat of bacteria depends on the variety and reproduction of wheat. The spread of the disease increases with increasing reproduction. In case of artificial inoculation, the causative agent causes dam-

age to spring barley, winter rye, sorghum, sudangrass, cabbage, radish and onions [36].

In addition to the above pathogens, weeds in agrophytocenoses of wheat are affected by *Pseudomonas viridiflava*, which is potentially dangerous for wheat.

By artificial inoculation, wheat damage are caused by *Pseudomonas syringae* pv. *coronafaciens, Acidovorax avenae* subsp. *avenae, Rathayibacter rathayi.*

If bacteria isolated from wheat are capable of causing damage to wheat plants, their biological properties given in [26, 60, 63] are studied and test systems [74] are used. To determine the species affiliation of bacteriosis pathogens, the results obtained on the properties of the bacteria studied are compared with the properties of bacteria already described in the original articles and Bergey's Manual of Systematic Bacteriology [25, 45, 51].

1.14 Wheat protection management against bacterial pathogens

To effectively limit the harmfulness of wheat diseases and their pathogens, agricultural technology and the use of drugs that eliminate the causative agent of the disease, prevent its mass development, prevent and limit spread are important. It is necessary to restore crop rotation and provide cultivated plants with proper balanced nutrition systems. It is necessary to create optimal growth conditions for plants, especially those related to their overwintering, which enhances the protective reactions to pathogens and the creation of unfavorable conditions for the life of bacteriosis pathogens. It is necessary to carry out pre-sowing measures, treat plants during the growing season, and observe storage conditions for the grain crop.

Since phytopathogenic bacteria are transmitted by seed, it is necessary to check the seeds in advance for the presence of pathogens and use healthy grain for sowing. Bacteriosis-resistant wheat cultivars are recommended.

In the presence of the affected seed necessary to carry out its decontamination. Wheat seeds affected by phytopathogens by 20% or more are rejected and not allowed for sowing [6].

Vitavax is recommended for wheat disinfection, which reduces the prevalence of black wheat bacteriosis by 17–21%, the development of the disease by 10.2–12.1%, and the prevalence and development of basal bacteriosis is two times lower than untreated plants [8]. For cereals offer heat treatment of seeds, which for 11 days at a temperature

of 71, 75 or 84 °C leads to the complete destruction of *Xanthomonas translucens*, and at 72 °C for 4 days to partial destruction [53].

For the prevention of yellow ear rot of wheat, it is necessary to observe crop rotation using legumes and intermediate crops; to carry out deep autumn plowing, spring tillage, weed and nematodes control; to carry out chemical treatment of grain, to free seeds from nematode, to observe quarantine when using imported grain. If the disease is confirmed, then the lesion should be immediately eliminated, and quarantine measures should be taken to limit the spread of the disease.

To protect against pathogens of bacterial diseases of wheat, some antibiotics and antibacterial drugs are effective. In the field, the results of the positive effect of the Japanese agricultural antibiotic kasumin, a synthetic polymer of catapol and its forms on phytopathogenic bacteria of the genera *Erwinia, Pseudomonas, Xanthomonas, Clavibacter* [15, 32].

Among the fungicides studied, drugs with antibacterial activity were found, which include mancozeb – Pencoceb, Tatu, Ridomil Gold, Acidan and Champion, which can be used to protect wheat from bacterial pathogens of the genera *Pseudomonas, Pectobacterium*, *Xanthomonas*.

Since chemical preparations are often harmful not only for pathogens, but also for the host plant, they worsen the ecological situation, partially accumulating in the soil, in recent years, much attention has been paid to biological products to protect plants from diseases. Thus, *Pantoea agglomerans* strains are effective against bacteria of the genera *Pseudomonas, Erwinia, Xanthomonas, Agrobacterium* and *Corynebacterium*. Wheat plants are usually sprayed with culture fluid that contains cells of the *P. agglomerans* strain [78]. Against the main diseases of leaves and root rot of spring wheat and spring barley, the biological preparation Agate 25 K is used to spray plants during the vegetation stage [28]. To protect against bacterial root rot of cereals, the drug Bactofit, the basis of which is the culture of *Bacillus subtilis*, is recommended [31]. To protect against the most harmful bacterial diseases of wheat that cause P. *syringae* pv. *atrofaciens* and *X. translucens* use complex preparations with thiram in combination with phytolavin 300. Phytolavin 300 is almost the only effective drug for protecting a number of cultures from bacterial pathogens [14]. Its greatest efficiency is observed when spraying plants at early booting stage of wheat. When treatment seeds, the drug minimizes infection, increases yield (in the case of bacteriosis – up to 69–80%), and also significantly improves grain quality.

To eliminate the infection that is stored in the plant residues, it is recommended to clear the straw from the fields, burn and the remains of plants, planning an effective crop rotation and to prevent the re-sow-

ing of one crop in the same field earlier than three to four years. To destroy weeds, which are the reserves of infection and insect pests of crops involved in the spread of bacterial infection. It is necessary to introduce a balanced amount of mineral fertilizers and microelements in quantities corresponding to the characteristics of each soil zone. The introduction of high doses of nitrogen, phosphorus and potassium fertilizers reduces plant resistance and contributes to increased wheat damage by phytopathogens [25].

So, the observance of all the above methods in the complex can contribute significantly to a reduction in infection with agents of bacterioses, increase the yield of wheat grain and improve its quality.

Chapter 2

Fungal diseases of wheat

Among the various pathogens of wheat diseases, fungi are the biggest threat in the world. Pathogenic fungi are widespread in wheat-growing regions, and can infect various plant organs. Depending on the type of pathogen, wheat variety, and weather conditions, pathogenic fungal infections can cause a significant inhibition and reduction in yields. Unlike viruses and bacteria, fungi can be easily visualized and, in most cases, their presence can be detected using the naked eye.

It is worth noting an increase in the severity of certain diseases that were previously rare in Ukraine and occurred episodically (septoriosis, pyrenophorosis, fusariosis, yellow rust), as well as an increase in the resistance of pathogens to fungicides.

Fungal diseases of wheat are caused by obligate parasites, facultative saprotrophs and facultative parasites, which under adverse conditions survive on plant residues [15]. Causative agents such harmful diseases as smut, powdery mildew belong to the group of obligate parasites.

These parasites are highly specialized and within the species are divided into physiological races, that are cultivar-specific at plant variety. All obligate phytopathogens, which form sporulation on the aerial parts of plants, are able to form several generations of spores, aerial dispersal over long-range. Therefore, obligate parasites and similar facultative saprotrophs can cause epiphytoxics and, as a result – yield losses. Pathogens of various types of cereal rust belong to this group of parasites. The infection process when fungal diseases occur includes the following stages: germination of the pathogen on the plant surface, invasion and spread in the internal tissues of plants, the occurrence of symptoms of damage, the formation of sporulation. Obligatory parasites gently affect plants and ensure the preservation of the viability of their cells at the stage of spread of mycelium. In contrast, the spread of facultative saprotrophs in living plant tissues is accompanied by the for-

mation of a zone of perishing cells. Facultative parasite affect the plant cell with its micotoxins before penetration, therefore, the colonization pathway of the host plant also has a zone of perished cells, however, it precedes the spread of the pathogen. At this stage, the pathological process will depend on the general physiological state of the plant and weather conditions. The onset of symptoms of the disease is a visualization of the disturbance of the host plant with the fungal pathogen. Numerous species of fungi are conditionally pathogenic to plants, animals and humans.

Fungal pathogens, as a rule, reproduce by spores that are resistant to temperature and humidity. They can tolerate adverse climatic conditions in the form of mycelium in the soil or perished plant tissues. Most fungal pathogens are facultative parasites. The type of disease and its causative agent can be identified based on symptoms, morphological and pathological characteristics, including studies of virulence for various varieties of host plants. In Ukraine, the most common wheat diseases that damage the leaf surface are snow mold, septoriosis, powdery mildew, pyrenophorosis, brown leaf rust [27].

2.1 Snow mould

Causative pathogens: *Fusarium nivale* Ces. ex Berl. & Voglino – Fungi, Dikarya, Ascomycota, Pezizomycotina, Sordariomycetes, Hypocreomycetidae, Hypocreales, Nectriaceae;

Monographella nivalis (Schaffnit) E. Müll.; *Microdochium nivale* (Fr.) Samuels & I.C. Hallett) – Fungi, Dikarya, Ascomycota, Pezizomycotina, Sordariomycetes, Xylariomycetidae, Xylariales, Microdochiaceae;

Pythium aristosporum Vanterp., *P. iwayamai* S. Ito – Chromista, Oomycota, Oomycetes, Pythiales, Pythiaceae;

Typhula incarnata Lasch ex Fr., *T. idahoensis* Remsberg, *T. ishikariensis* S. Imai – Fungi, Dikarya, Basidiomycota, Agaricomycotina, Agaricomycetes, Agaricomycetidae, Agaricales, Typhulaceae.

Dispersion. The disease manifests itself in the presence of snow cover on unfrozen moist soil, relatively low temperature in spring and frequent thaws in winter [11, 13, 24, 37]. Spores develop on infected plant residues or on the surface of the soil. They are carried by airflow or rain spray. Cool and humid weather is favorable for the development of the disease. Similar weather conditions are typical for the territory of Ukraine. The disease is quite common in other European countries [32], in the northwestern United States (Oregon, Washington) [35], in East Africa, the highlands of Mexico, in the Andean region of South America, southern China, and northern Japan.

Over recent years, the disease has been widespread in the fields of the Republic of Belarus, often with an epiphytotic character of development. The causative agent of the disease in Ukraine is the facultative parasitic fungus *Fusarium nivale* Ces. (*Microdochium nivale* (Fr.) *Samuels* & I.C. Hallett) with well-defined saprotrophic properties [11, 13]. At the same time, *Pythium aristosporum, P. iwayamai, Typhula incarnata*, *T. idahoensis*, *T. ishikariensis*, which are quite common in northern Japan and the Pacific coast of the United States, can cause snow mould.

Symptoms. The disease develops in early spring, immediately after the snow melts. Watery spots with a white cobwebby mycelium are formed on winter wheat leaves, leading to gluing of the leaves; as a result, the affected leaves perish. With severe damage, the perish of the tillering node, leaf blade, roots and perish of the whole plant is observed. At the base of the culms and on the remains of perished plants during the entire growing season, conidial sporulation of the fungus is formed. The causative agents are stored in the soil on organic residues and in the fall begins the infection of winter crops. The beginning of the development of mycelium in winter crops, observed since autumn, is amplified in early spring, after the snow melts. The spotting caused by these fungi begins at and tillering node the early booting stage. In the juvenile stage of plant, an oval-elliptical, grayish-green mottling first appears, usually located on the bends of the leaves. The spots quickly get bigger and taking shape a large eye spot, with a whitened or light gray center. The leaves are split or torn, starting from the center of the lesion (Appendix 2.1). The fungus also causes withering of the plants, root rot and white ears, and in winter cereals – pink snow mould [24].

Optimum conditions for pathogen development. The pathogens are more aggressive at low temperatures (5 °C), which explains the predominant distribution of the fungi in years with a cold spring. Low temperatures in winter inhibit the development of the fungi, but the viability of mycelium and conidia is maintained even at a temperature of –33 °C. At natural conditions, this temperature occurs in winter only in the absence of snow cover. It happens on the types of years. Pathogens affect the genera *Triticum, Secale, Dactilis, Agrostis, Poa, Alopecurus* and other representatives of the Poaceae. Snowy winters, low temperatures, high moisture supply, extended snow melting, and increased acidity of soils determine the danger of the occurrence of infectious aging. In the years of epiphytotia (3 times in 10 years), the development of the disease is 40–50%, perished – 15–20%. In some fields, it's possible crop failure.

So, factors contributing to the development of the disease are weakening of plants as a result of adverse conditions (thaw, excessive soil moisture, relatively low temperature in spring, slow snow melting and snow falling on unfrozen soil), high air humidity at a relatively low temperature (4 °C) in spring, thick planting and no rotation, increased doses of nitrogen fertilizers, the location of winter crops in the lowlands.

Injuriousness. There is perishing of the leaves and produced imperfect grain with a low mass at multiplicity disease of wheat plantings.

2.2 Fusarium head blight (FHB)

Causative pathogens: *Fusarium graminearum* Schwabe, *F. culmorum* (W.G. Sm.) Sacc. and other *Fusarium* species – Fungi, Dikarya, Ascomycota, Pezizomycotina, Sordariomycetes, Hypocreomycetidae, Hypocreales, Nectriaceae.

Dispersion. Prevalent is ubiquitous. Pathogen is highly damaging wheat plantings in years with humid weather and moderate temperatures in period after the paniculation stage wheat. This disease affects most cereals. Fungi of the genus *Fusarium* exist in almost all soils and plant residues [2, 3, 24, 25, 32, 34, 35, 37].

Symptoms. The ear blight in wheat symptom on the whole is exhibit in form glumes of ears discoloration, clearly visible on the background of healthy green ears (Appendix 2.2). At fair weather conditions (relative humidity above 70% and temperatures above 15 °C during the period from flowering to harvesting), on the affected spike, depending on the type of pathogen, a plaque of white, pink, orange, red mycelium appears. Conidia are formed on the mycelium or in sporodochia. At the end of the growing season, some types of pathogens form black perithecia – the ascigenous stage (teleomorph). With early and severe damage, the caryopsis becomes light, wrinkled, white, loses its luster and vitreous, the endosperm is fragile, the groove is deep. At later stages of the lesion, the caryopsis does not differ in appearance from a healthy one, but carries an internal infection.

Sources of infection are plant residues, soil, cernel. Infection occurs with ascospore, conidia, which spread in the wind, rain, insects.

Optimum conditions for pathogen development. The development of the disease contributes to increased air humidity, a wide temperature range (10–28 °C), and rain. The critical period for infection is the flowering phase.

Injuriousness. The reduction in yields at the defeat of the entire ears is 82%, half – 76, thirds – 44, in addition, about 70% of the grains lose their germination. In such case, infected ears contain of shrunken kernel. Fusarium head blight impairs the baking quality of flour.

If wheat contains more than 5% of the infected kernel with species *F. sporotrichiella, F. graminearum*, the content of mycotoxins exceeds the level acceptable for humans and animals and can cause poisoning.

2.3 Fusarium root rot

Causative pathogens: fungi of the genus *Fusarium: F. oxysporum* Schlechtend.: Fr., *F. solani* (Mart.) Sacc., *F. avenaceum* (Fr.) Sacc. (synonym – *G. avenacea* R.J. Cook), *F. verticillioides* J. Sheld. (stage *Gibberella fujikuroi* var. *moniliformis* (Wineland) Kuhlman), *F. subglutinans* (Wollenweb. & Reinking) P. E. Nelson, T. A. Tousson & Marassas (synonym – *G. fujikuroi* var. *subglutinans* (Wollenw. & Reinking) E.T. Edwards), *F. acuminatum* Ellis & Everh. (synonym – *G. acuminata* Wollenw.), *F. equiseti* (Corda) Sacc. (synonym – *G. intricans* Wollenw.) – Fungi, Dikarya, Ascomycota, Pezizomycotina, Sordariomycetes, Hypocreomycetidae, Hypocreales, Nectriaceae.

Dispersion. Pathogens are prevalent ubiquitous in the regions of grain growing, especially in conditions of unbalanced use of high doses of nitrogen fertilizers.

Symptoms. In the early stage of plant development, root rot appears on seedlings in the form of brown or dark brown oblong necrotic spots or strokes (Appendix 2.3) [2, 3, 24, 25, 32, 34, 35, 37]. For growth season, the symptoms of the disease are observed mainly in the tillering zone and on the lower part of the culm. First, spotted or hatching necrosis is formed on these zones, which spread and configure significant of necrotic brown spot zones, then the stems perishing.

In the case multiplicity of disease manifestation on winter wheat plantings, there is a multiple both shriveling of grain and underdeveloped of ear, as well as a partial of seedless ear.

High-infected plants perishing prematurely and by harvest time their culm and ears are covered with a dark coating of saprotrophic fungi.

Optimum conditions for pathogen development. Contamination of plants is facilitated by soil moisture of more than 40% and temperature from +3 °C to +35 °C (optimum +15 ... +22 °C).

Injuriousness. Fusarium root rot liquefyes crops, worses wintering of winter crops, grain quality, thusand-kernel weigt, causes the seedless ear.

2.4 Septoriosis (Septoria spot)

Causative pathogens – fungi of the genus *Septoria*, the main of it are *Zymoseptoria tritici* (Desm.) Quaedvl. & Crous *(*synonym *S. tritici*

Desm.), stage – *Micosphaerella graminicola* (Fuckel) J. Schroet., parasitic mainly on the leaves – Fungi, Ascomycota, Pezizomycotina, Dothideomycetes, Dothideomycetidae, Capnodiales, Mycosphaerellaceae; *Parastagonospora nodorum* (Berk.) Quaedvl., Verkley & Crous *(*synonym *Stagonospora nodorum* (Berk.) E. Castell. & Germano; *Leptosphaeria nodorum* E. Müll.) – Fungi, Dikarya, Ascomycota, Pezizomycotina, Dothideomycetes, Pleosporomycetidae, Pleosporales, Leptosphaeriaceae.

Dispersion. Septoriosis spread rapidly at frequent rainfall and moderate air temperatures, and its pathogens are the most common pathogens of winter wheat in the forest-steppe of Ukraine [24, 26, 28, 37]. The causative agent dispersion with pycnospores, mainly with raindrops – near, and ascospores – long-rang distances. Usually, ascospores are producing scanty and their dispersion at low humidity and high air temperatureis are limited.

Symptoms. Septoriosis is a very complex infection, not only pathologically, but also in terms of culture protection. First of all, the symptoms of septoriosis are visible to the naked eye, when the pathogen mycelium develops in the intercellular spaces of the host plant tissues. In case of septoria infection, the mycelium develops in the intercellular spaces of the plant and the first symptoms of the disease – tissue lightening (small spots) – resemble a nutrient deficiency, physiological stress, etc. That is, at the beginning of the lesion, the symptoms are almost invisible, although the latent development of mycelium continues in plant tissues. Septoriosis can be true diagnosed at becoming of pycnidia. At this stage, the mycelium has already developed so much that lack nutrients to leaves. At this stage we are possible observed the disease and take action to plant protection.

Septoriosis affects all above-ground organs and seeds grains. Display of the development of septoriosis on wheat planting are observed throughout the growing season. The first signs may appear on the plants in the form of small oval, oval-elongated, light, yellow, light brown or gray-green spots, elongated along of leaves rib. On older leaves, spots are light, with a fuzzy outline, with a dark border or without it, at first they are almost invisible on the plant. The spots gradually increase in size, subsequently their center brightens, and roundish black pycnids containing fungal spores become clearly visible on it, see Appendix 2.4. Conidia (pycnospores) are filiform, colorless, somewhat curved, with rounded ends, 3–5 septate. Symptoms of the disease on susceptible varieties appear in the fall, mainly on leaves that touch the surface of the soil. The spots on the plants are round and often occupy the entire surface of leaves. There are clearly visible a black pycnids of the causative agent of the disease. The development of the disease begins with the lower leaves and spreads up, reaching a maximum

degree in milky-waxy stage of ripeness. In adult plants, depending on the stability of varieties and weather conditions, individual spots can merge and cover most of the leaf surface, they appear and increase in size mainly from the central part of the leaf blade, leading to premature perishing of leaves, ears, and sometimes the whole plant. Pycnids are formed on the upper side of the leaves. Septoriosis develops rapidly from the beginning of the spring growing season of winter wheat in the flag leaf stage [24, 37].

Optimum conditions for pathogen development. Strong spotting development is observed in the presence of high air humidity (90–100%) and frequent precipitation, cloudy weather, especially in flowering period, with light winds at a relatively high air temperature, optimally +12–25 °C. Under such conditions, picnospores can germinate within a few hours after exiting the pycnidia, but the minimum effective temperature for the disease is 5 °C. Arid periods of vegetation inhibit the development of the disease. In the conditions of the Southern Forest-Steppe and Steppe of Ukraine, the onset of drought completely inhibits the development of the leaf form of the disease. The late sowing period, the introduction of only nitrogen fertilizers, the thin planting also contribute to the development of the disease. In case of septoriosis infection, the assimilation surface of the leaves decreases, the physiological and biochemical processes in the plants as a whole are disrupted, the thousand-kernel weight decreases, its technological parameters deteriorate [24, 26, 28, 37]. Typical signs of leaf septoriosis are dependent on the variety's resistance, weather, and agricultural conditions. The conditions of August–December 2014 (Ukraine) created opportunities for the full realization of the infectious potential of infectious potential of causative agents of septoriosis both *Septoria tritici* and *Stagonospora nodorum*.

Injuriousness. Epiphytoties of septoriosis on wheat are possible on susceptible varieties in the presence of a sufficient amount of causative agents of Septoriosis and weather conditions that ensure its spread. The period from the booting to the heading-flowering stage is critical.

Features of the development of the pathogen and the manifestation of the disease were described at the end of the nineteenth century, but an economically significant decrease in the yield and its quality began to occure itself after introducing dwarf high-yielding varieties into the culture. If this disease reaches a high level of development before the harvesttime till, reduction in yelds can be significant as a result formation of feeble coarse graine. Shortfall in wheat wheat prodaction from defeat by Septoriosis can reach 20% or more. The costs of chemical protection of plants from causative pathogen in Europe alone reach several hundred million dollars annually.

Causative pathogens of this disease were previously considered secondary pathogens, but over the past decades due to climate warming, changes in treatment technologies (increase of surface tillage), no managmant of field changing, creation and introduction of varieties of wheat resistant to major leaf diseases (rust, powdery mildew), favorable conditions have developed for their annual accumulation, distribution and intensification of harmfulness.

Beginning from spring 2014 (in Ukraine), were used not only sighting of Septoria spot, but very often the presence of Stagonospora nodorum Berk was established using PCR diagnostics.

2.5 Helminthosporium root rot

Causative pathogen: *Cochliobolus sativus* (S. Ito & Kurib.) Drechsler ex Dastur; anamorphs – *Bipolaris sorokiniana* (Sacc.) Shoemaker; *Drechslera sorokiniana* (Sacc.) Subram. & B.L. Jain; *Helminthosporium sativum* Pammel, C.M. King & Bakke, *Helminthosporium sorokinianum* Sacc. – Fungi, Dikarya, Ascomycota, Pezizomycotina, Dothideomycetes, Pleosporomycetidae, Pleosporales, Pleosporaceae.

Dispersion. Prevalent is ubiquitous, but it causes the greatest harm in the steppe zone and forest-steppe in dry years, especially in spring wheat. It also affects other cereal crops and wild cereals [6, 24, 37].

Symptoms. At disease is observed, browning, deformation of seedlings, which often perish before the coleoptile emerges on the soil surface. Brown stripes and spots appear on the lower leaves, and later on the basal culm.

In the booting stage are sighting browning of the tillering node and at strong infected – damage of the first node visible (Appendix 2.5). Sources of infection are conidia, that do not lose their viability in 1.5 years on plant residues or in the soil. The fungal mycelium can persist in the infected seeds or, under adverse conditions, develop as saprotrophs.

Optimum conditions for pathogen development. The disease develops more intensively on weakened plants, its harmfulness increases in drought conditions, while the pathogen releases mycotoxins that destroy tissues and the plant perishing. In conditions of warm (temperature +20 ... +28 °C) and humid weather (air humidity more than 95%), rot of the basal nodes and lodging of cereals is observed, in this case the disease is called dark-brown stem spot. Under such conditions, the pathogen infects the ears, penetrates the pericarp and endosperm, to be caused browning of the kernel, and in this case the symptoms are called the blackpoint. In some cases, the fungus forms of ascigerous

stage – *Cochliobolus sativus*. The disease is facilitated by mild winters, first dry, then wet weather, crop rotation disturbance, frost injury of wheat planting.

Injuriousness. Depending on the level of development of the disease, it cause to thin planting, seedless ear and feeble and coarse grain.

2.6 Helminthosporium leaf blotch

Causative pathogen: *Cochliobolus sativus* (S. Ito & Kurib.) Drechsler ex Dastur; anamorphs – *Bipolaris sorokiniana* (Sacc.) Shoemaker; *Drechslera sorokiniana* (Sacc.) Subram. & B.L. Jain; *Helminthosporium sativum* Pammel, C.M. King & Bakke, *Helminthosporium sorokinianum* Sacc. – Fungi, Dikarya, Ascomycota, Pezizomycotina, Dothideomycetes, Pleosporomycetidae, Pleosporales, Pleosporaceae.

Dispersion. Helminthosporium leaf blotch affects wheat, triticale, barley, as well as most wild grasses. The disease is widespread, but it mainly prevails in areas with high rainfall and high humidity.

Symptoms. Disease caused by this pathogen manifest itself in the form of elongated oval dark brown spots, subsequently they become light brown or yellow-brown with a dark brown ring [3, 6, 24, 37]. When expanding, the spots merge, which leads to perishing of leaves. The initial infection develops on the lower leaves and appears as chlorotic spots. There are gradually increase in size, become dark brown and often merge. At sizable damage to the leaves and sheath, they perishing prematurely.

Optimum conditions for pathogen development. Optimum conditions for development of disease are high rainfall and high humidity.

Injuriousness. If the disease manifests itself in the initial of the growing season and the conditions for its development are favorable, the leaves may completely perish, the grain becomes flat, which leads to shortfall in wheat production.

2.7 Rhizoctonia root rot

Causative pathogen: *Ceratorhiza cerealis* (E.P. Hoeven) R.T. Moore (synonym *Rhizoctonia cerealis* E.P. Hoeven) – Fungi, Dikarya, Basidiomycota, Agaricomycotina, Agaricomycetes, Cantharellales, Ceratobasidiaceae.

Dispersion. *Rhizoctonia cerealis* is a pathogen with many hosts. It affects most crops and almost all species of cereals. It is widespread in soil and plant residues [3, 6, 14, 24, 37].

Symptoms. The first signs of the disease appear on the basal of culm and on the leaves, similar to the symptoms of caused by the fungus *Oculimacula acuformis*. Eyespot (Strawbreaker) affects superficial tissues and has clear contours in comparison with ordinary spectacle spotting (Appendix 2.6). The spots are dark brown with a straw-yellow center, on which is often the mycelium, which is easily removed. The roots that usually perish off are affected. In infected plants, growth retardation and a decrease of tillering are observed.

Optimum conditions for pathogen development. Plant infection depends on environmental conditions. Dry sandy soils, low temperatures and high humidity are favorable for the development of the disease. The fungus that exists in soil and in plant residues infects roots and leaves.

Injuriousness. The disease is usually more manifestation in those fields where growing the same crop for a long time, especially winter wheat. However, there is no information about the high severity and epiphytotics of this disease.

2.8 Kernel smudge (Black point)

Causative pathogens are fungi belonging of different species of the genera *Fusarium*, *Alternaria*, and *Cochliobolus sativus* – Fungi, Dikarya, Ascomycota, Pezizomycotina, Sordariomycetes, Hypocreomycetidae, Hypocreales, Nectriaceae; Fungi, Dikarya, Ascomycota, Pezizomycotina, Dothideomycetes, Pleosporomycetidae, Pleosporales, Pleosporaceae.

Dispersion. The disease is common in areas of growing fine-grained cereals. Wheat is the main host, in addition, the disease affects triticale and several species of cereal grasses.

Symptoms. The pericarp of a grain of wheat ripens and becomes dark-brown or black, the spot is usually limited to the germinal part of the grain. If the grain is affected by *Alternaria* sp., then only the pericarp darkens, and for the lesions by *Helminthosporium* and *Fusarium* spp. the embryo is damaged or perished off. Other of fungal species may also be the cause of the black point, but the above are the most common [3, 24, 25, 28, 34, 37].

Optimum conditions for pathogen development. If humid weather prevails for several days before harvest, the black point can affect many varieties. Infection of grain with these fungal species occurs at the stage of milk-wax ripeness.

Injuriousness. It leads to economic losses – the purchase price of contaminated grain is lower than healthy. If fungi of the genera *Fu-*

sarium and *Helminthosporium* cause the disease, germinating ability may also decrease.

2.9 Black head mold

Causative pathogens: complex defeat by species of the genera *Alternaria, Stemphylium*, *Cladosporium*, *Epicoccum* etc. – Fungi, Dikarya, Ascomycota, Pezizomycotina, Dothideomycetes, Pleosporomycetidae, Pleosporales, Pleosporaceae; Fungi, Dikarya, Ascomycota, Pezizomycotina, Dothideomycetes, Dothideomycetidae, Capnodiales, Cladosporiaceae; Fungi, Dikarya, Ascomycota, Pezizomycotina, Dothideomycetes, Pleosporomycetidae, Pleosporales, Didymellaceae.

Dispersion. Pathogens of black head mold are widespread (cosmopolitan), affect plant tissue, which then perish off.

Symptoms. Typical symptoms of the disease are the darkening or blackening of the ears shortly before ripening or the perished of spikelets as a result of superficial colonization with mycelium both phytopathogenic and saprotrophic of fungi (Appendix 2.7) [3, 24, 25, 28, 34, 37].

Optimum conditions for pathogen development. Before the ripening of wheat, providing, that the weather very humid, at abundant colonisation of plants by aphids, as well as premature perish off of plant – their populated one or more species of these fungi. In fact, black mold is not an infectious disease, since saprotrophic fungi populate dead plant tissues or those that perishing.

Injuriousness. Black head mold is not an economically important disease, but in high humidity or rainy weather, fungi populate mature grains, causing them to darken, blacken or black point.

2.10 Alternaria blight

Causative pathogens: *Alternaria tenuissima* (Nees) Wiltshire, *Alternaria alternata* (Fr.) Keissl. (synonyms *Alternaria tenuis* Fr.) – Fungi, Dikarya, Ascomycota, Pezizomycotina, Dothideomycetes, Pleosporomycetidae, Pleosporales, Pleosporaceae.

Dispersion. Species of the genus *Alternaria* are ubiquitous mycelial fungi that are described as saprotrophs or opportunistic microorganisms that can colonize a wide range of plants, including various crops (fine-grained cereals, fruits, vegetables) [3, 12–14, 25, 28, 34, 37].

Symptoms. Alternariosis in wheat crops is manifested in the flowering and the milk-ripe stage in the form of dark spots on glumes (“husks”) of ear. Later, during the grain maturity, blackening of the embryo

("blackpoint") is sighting. At penetrates into the seeds, the mycelium of causative agent accumulates mainly in (allanto) hull of grain, and sometimes reaches the endosperm. The attacked grain, usually, is large and well formed, than differs from that affected by helminthosporiosis.

Optimum conditions for pathogen development. A wide spread of alternariosis is observed in years with a high temperature (above 24 °C) and air humidity in the flowering and the milk-ripe stage.

Injuriousness At infect with alternariosis undeveloped kernel form in ears of wheat.. At infect with alternariosis undeveloped kernel form in ears of wheat.

In this case, they has low viable and germination. Plants grown from such grain lag behind in growth and development, as a result of which they have low productivity. Grain flour with a "blackpoint" has a darkish color and low baking quality. Critical is their ability to produce toxic compounds that are dangerous for food production – alternariol (AOH), alteriariol monomethyl ether (AME), altenuene (ALT) and tenuazonic acid (TeA) [34].

2.11 Alternaria leaf spot

Causative pathogen: *Alternaria triticina* Prasada & Prabhu – Fungi, Dikarya, Ascomycota, Pezizomycotina, Dothideomycetes, Pleosporomycetidae, Pleosporales, Pleosporaceae.

Dispersion. The disease is widespread in the eastern and central parts of the Indian subcontinent.

Symptoms. The disease manifests itself in the form of small, oval or elliptical forms of chlorotic spots, which gradually increase in size, acquiring an irregular shape [3, 25, 28, 34, 37]. The chlorotic border of the spots becomes dark brown. The symptoms of alternariosis are difficult to distinguish from the stains caused by species of the genus *Helminthosporium*. The disease usually begins with the lower leaves, but its symptoms can occur on all organs of plants.

Optimum conditions for pathogen development. The fungus is stored as conidia – on the surface or in-seeds as mycelium. As a result of sporulation, the inoculum spreads of aerial currents, contributing to the secondary infected to the leaves and other organs. The pathogen infects the grains as a result of contamination of the ears in the grain filling period. High humidity or irrigation, as well as moderate temperature (20–25 °C) are favorable for plant infection and the development of the disease.

Injuriousness. Alternating leaf spotting can cause serious damage under favorable conditions for the development of the disease. At the

cultivation of varieties sensitive to the pathogen, reduction in yields can be significant.

2.12 Take-all

Causative pathogen: *Gaeumannomyces graminis* (Sacc.) Arx & D.L. Olivier (synonym *Gaeumannomyces graminis* (Sacc.) Arx & D. Olivier var. *tritici* J. Walker; *Ophiobolus graminis* (Sacc.) Sacc.) – Fungi, Dikarya, Ascomycota, Pezizomycotina, Sordariomycetes, Sordariomycetidae, Magnaporthaceae.

Dispersion. It occurs mainly in regions with a temperate climate and sufficient moisture – in the western regions of the Polissya and forest-steppe zones of Ukraine. It affects wheat, rye, barley.

Symptoms. The roots, basal part and lower internodes of the culms, leaf sheaths, on which black spots initially appear to hatch, that are gradually covering all organs [24, 37]. Both the basal part of the culm and leaf sheaths acquire a glossy black color, on the lower internodes, under the perished sheath of leaves, a dark mycelium of the fungus is visible. The roots become black and brittle, the basal part of the culm is covered with a black coating of the mycelium of the fungus, the culms easily come off (Appendix 2.8). The disease is clearly visible starting from the heading stage: the plants lag behind in growth, have a pale gray color, ears are white, they are more directly compared to healthy ones. When disease reaches a high level develop both of "white-culms" and "white-spiked" are observed. When infection occurs in the juvenile stage of plant, a decrease in tillering and ear sterility are observed often.

Optimum conditions for pathogen development. The source of infection is the plant residues of cereals in the soil, which can be stored for up to three years. The initial infection occurs in the spring when the seedlings come into contact with mycelium or ascospores of the fungus in the soil. The pathogen can also winter with Chlamydospores, which are sprout in the spring and infect plants. Plants are infected by the disease throughout the growing season. The most favorable condition for infection of plants is the relatively low temperature of the soil (12–18 °C). Optimum conditions for the growth of the fungus are increased humidity and temperature of 19–24 °C, soil--alkaline reaction, a lack of nutrients and significant amount of nitrates, which significantly enhance the development of the disease. Ophiobolosis for growth season spreads at contact of roots healthy and infected of plants so are sick area observed. Damage contributes to wet and cool spring, warm and dry beginning of summer. The infectivity of rot increases markedly in autumn and early spring, progressing on the basal part of the culm and leaves.

Injuriousness. Ophiobious root rot during monoculture can lead to significant losses in the yield of spring and winter wheat at fall-planting, especially in areas where both the mulch planting, that min-till is practiced. Primary damage leads to inhibition and perished of plants, in a later period – to a decrease in the quality of grain, thousand-kernel weight and 40% decrease in yield. If the plants are infected in the late phase of development, then usually the losses are small.

2.13 Pythium root rot

Causative pathogens: over 300 species of the genus *Pythium* Pringsh – Chromista, Oomycota, Oomycetes, Pythiales, Pythiaceae.

Dispersion. Root rot is intensively dispersal in the fields with using direct seeding and well as min-till. In soil, the pathogen remains in the upper layer – up to 5 cm, which is another of the main reasons for the wide spread of pitium in area at direct sowing.

Symptoms. The causative agent of the disease affects the root system of sprouts and young plants [3, 12, 14, 25]. The first symptoms of the disease are the appearance of red-brown spots on the roots and root neck, dark brown spots appear in the basal part of the culm, and root hairs themselves are absent. Gradually, the roots begin to rot, perish off and dry out, starting from the tips. The plant easily divide from the root and drying out as a result. Sometimes this disease does not leave such serious consequences, but only manifests itself in a change in the color of the leaves and growth retardation. If increased humidity continues, then in basal part of the culm, an abundant white coating of mycelium may appear.

For pathogen is characterization of no septation of white cobwebby mycelium, which on spherical zoosporangia 12–21 μm in diameter are formed. They di-flagellate zoospores and oospores 8–25 μm in diameter are formed.

Settling on the perished zones of the roots, which become the gateway in the plant for infection, the parasite feeds and releases toxic substances, which, in turn, kill neighboring living cells. Thus, the parasite spreads throughout the plant. Young plant tissues are more likely to be infected by pitium. There are kept in the form of oospores on plant residues (often up to 12 years) on plant residues, they survive well in harsh environmental conditions.

Optimum conditions for pathogen development. Under conditions of maximum soil moisture and a temperature of about 6 °C they dispersion and harmfulness are spike.

Injuriousness. Injuriousness at disease of planting wheat are manifestation in in reducing seed germination, reducing the assimilation

surface of the first leaves, delaying plant growth, dying off the root system, reducing tillering and loss of yield.

2.14 Ergot

Causative pathogen: *Claviceps purpurea* (Fr.) Tul. (*Sphacelia segetum* Lév.) – Fungi, Dikarya, Ascomycota, Pezizomycotina, Sordariomycetes, Hypocreomycetidae, Hypocreales, Clavicipitaceae.

Dispersion. Causative pathogen are wiadespread on wheat planting and barley on areas with excessive moisture in the flowering period and on rye – everywhere [3, 12–14, 24, 37]. Ergot are found on all cereals with small grains, especially in the sterility of crops, for example at frost. Sterile flowers have an open type of flowering and are therefore more susceptible to infection. The disease is more often recorded in regions with a cool and humid climate.

Symptoms. The disease appears on ears, where instead of grain have been appear purple and then black sclerotia up to 2–4 cm in length (Appendix 2.9). After the winter sclerotia germinate, forming of fruit bodies with heads and stipes. Perithecia are formed in the head with asci and ascospores. The initial infection of flower originates from ascospores which are formed in fruit bodies from the sclerotia of previous year. Ascospores infect a floret of flowering cereal, where simultaneously with started pathogen fruiting phase which consists from fungai konidia and attracts insects is selected. Fungi are spreading on healthy ears wheat by spores contained in honeydew and with wind and rain. After germination of spores mycelium is formed, which at grain maturity transforms to sclerotium. The mature sclerotia are kept and survive in soil to the next year. Under drought conditions sclerotia do not lose viability during a few years. For their germination low temperatures are needed [28, 37].

Optimum conditions for pathogen development. Damp and cool weather are contributes to the development and spread of the disease. Causative pathogen are spreadig by insects.

Injuriousness. At damages of planting wheat by Ergot have been reduction in yields can make 12–17% and grain are poor of quality. A flour from grain with the admixture of sclerotia (over 0,5%) can affecting of people and anymals.

2.15 Wheat blast

Causative pathogens: *Pyricularia oryzae* Cavara (*Magnaporthe oryzae* B.C. Couch); *Pyricularia grisea* Sacc. (*Magnaporthe grisea* (Hebert)

Barr) – Fungi, Dikarya, Ascomycota, Pezizomycotina, Sordariomycetes, Sordariomycetidae, Pyriculariaceae.

Dispersion. Species *M. grisea* was found in South America. It probably comes from tropical herbs. An important role in the survival of the fungus is played by alternative hosts.

Symptoms. It affects the ears wheat, as a result of which the spikelets and also the leaves above the point of infection become white and sterile, however, strong epiphytoties are manifested by the defeat of the spikelets without any symptoms on the leaves. Damage to leaves, stems and and glumes ("husks") of ear wheat are resemble "air" rice, have an elliptical or elongated shape. The centers of damage are from white to light brown in color with dark gray to red-brown border. Sporulation is formed on the lower surface of the leaves. Grains infected in the early stages of development are very flat. This infection can later be transmitted to healthy grains, that becoming a major source of infection. The development of the disease on the ears can be quick and reach 100%. Within two weeks, the ears of wheat are becomed white, while the leaves remain green with no signs of disease. This indicates that the infection spreads by aerial dispersal. The rate of development of the lesion depends on the time of infection: in the heading stage it is higher, and in the grain maturity – lower [3]. The epidemiology of Pyricularia is still unknown.

Optimum conditions for pathogen development. Optimum conditions for pathogen development is temperature moderate to warm and continuous rains.

Injuriousness. Wheat blast is common in the warm areas of Latin America. In last time, increase in the incidence rate is observed. In dry seasons, this disease is not a serious problem, but 2009 epiphytotia caused 100% yield loss in much of Paraná, Brazil. This indicates the danger of the disease due to expected climate change.

2.16 Southern blight

Causative pathogens: *Sclerotinia graminearum* Elenev ex Solkina – Fungi, Dikarya, Ascomycota, Pezizomycotina, Leotiomycetes, Leotiomycetidae, Helotiales, Sclerotiniaceae; *Sclerotium rolfsii* Sacc. – Fungi, Dikarya, Basidiomycota, Agaricomycotina, Agaricomycetes, Agaricomycetidae, Agaricales, Typhulaceae.

Dispersion. Most cultivated and wild cereals, as well as many species of dicotyledonous plants, are susceptible to this disease. The fungus is widespread in tropical and subtropical conditions [3, 11–14, 24].

Symptoms. If infection occurs in the early phase of plant growth, before or after seed germination, then seedlings perishing. The surface

of the affected tissue is covered with a white fluffy mycelium of the fungus, which is visible on basal part of the culm at the soil level. Next, the disease develops as Stem rot, lower internode and root system, which leads to the perish off or white-spiked of plants. The disease begins to appears as wilting leaves. At the basal part of leaf blade, on the stems and especially at the tillering zone near the surface of the soil, a gray flaky coating appears, and after 4–6 days young whitish sclerotia are formed on it. Over time, they acquire brown, dark brown or black color and has dense, irregular shape – 1.5–6 × 1–3 mm. On one plant, up to 25 sclerotia are formed. Affected plants turn brown, dry out and lodging [24].

The causative agent of the disease is the fungus *Sclerotinia graminearum*, which is able to infect plants throughout the growing season; in its development cycle, it forms mycelium, sclerotia and apothecia with asci and ascospores.

Sclerotia, that was formed in spring and fallen to the ground persist until autumn. The primary source of infection may be the fungal mycelium, that wintering on plant residues, or sclerotia.

Optimum conditions for pathogen development. High temperatures (20 °C and above), high humidity and acidic soils are favorable for plant infection and the development of the disease. Usually, in september, less often in spring, they germinates, forming more or less rounded apothecia 2.5–6 mm diameter, that sitting on a stipe up to 15 mm long. On their surface are asci with spores. Asci are cylindrical, 175–300 × × 10–14 μm, ascospores are non-equilateral, 16–23 × 7–10 μm. Paraphyses are formed between the asci.

Injuriousness. Ascospores infect of plants, but usually the mycelium develops very weakly in the fall. There is not much damage therefore from the outside. However, in the spring, the development of the pathogen is enhanced, which leads to perish off plants after wintering.

2.17 Downy mildew

Causative pathogen: *Sclerophthora macrospora* (Sacc.) Thirum., C.G. Shaw & Naras. – Chromista, Oomycota, Oomycetes, Sclerosporales, Verrucalvaceae.

Dispersion. The disease is characteristic of crops growing on waterlogged or excessively irrigated fields. The fungus has many hosts, including many crops – corn, sorghum and a number of grasses. It can be found in any soil – from very waterlogged to dry.

Symptoms. Infected plants are short, unaligned, twisted with abundantly tillering and culms yellowish-green of color and leaves thick er-

icoid. Tillering stage continues till heading stage or plants premature perish. If an ear is formed, then it branches, most of the ears become like leaves [4, 6]. Symptoms of the disease are more degree of manifestation during the tillering-booting period of the host plant.

Optimum conditions for pathogen development. The development of the disease occurs in the temperature range between 10–25 °C. The infection is transmitted from the inoculum, preserved in the soil, or from the affected weeds, however, the presence of water is a prerequisite for the development of the infection.

Injuriousness. Small, local diseases can occur in regions with favorable conditions for its development. However, data on the spread and damage from this disease are not available.

2.18 Ascochyta leaf scorch

Causative pathogens: *Neoascochyta exitialis* (Morini) Qian Chen & L. Cai (synonym *Didymella exitialis* (Morini) E. Müll.; *Ascochyta tritici* Hori & Enjoji; *Sphaerellopsis filum* (Biv.) B. Sutton (syn. *A. graminicola* Sacc.); *A. sorghi* Sacc.; *Neoascochyta europaea* (Punith.) Qian Chen & L. Cai (syn. *A. hordei* var. *europaea* Punith. and *A. graminicola Sacc.*) – Fungi, Dikarya, Ascomycota, Pezizomycotina, Dothideomycetes, Pleosporomycetidae, Pleosporales.

Dispersion. Pathogen is prevalent ubiquitous. It occurs in wheat growing regions with a temperate humid and warm climate – in New Zealand, Japan, Korea, Europe, Argentina, and the USA, causing leaf abscission at end growing season [31]. In Ukraine, the so-called lead spotting (pathogen *Ascochyta agropyrina* (Fairm.) Trotter) and pale spotting (pathogen *Sphaerellopsis filum* (Biv.) are recorded B. Sutton).

Symptoms. Symptoms appear on the lower leaves at the at the early growth season and later on the upper ones [12, 19, 37]. On the leaves of wheat, the spots are initially small, rounded or irregular in shape, with a dark brown border and a lighter center. Subsequently, the spots merge, often covering the leaf blade entirly. The leaves are become dirty gray and dry. The fungus forms black dots of the pycnidia in the center of the spots (Appendix 2.10). Pycnids are spherical, dark brown, deepen, in groups, up to 165 μm in diameter. Pycnospores (conidia) are elongated, cylindrical, with rounded ends, wide, 1-(less often 2–3)-septate, 15–23 × 4.5–6 μm.

The fungus survives with pycnidia in infected plant residues. In spring, the leaves are infected with conidia (primary infection), which are formed in the pycnidia and dispersion by drops of rain and wind. The causative agent often penetrates leaf tissue through the stomata

or with help physical damage. Conidia that are formed on the affected plants during the growing season cause secondary infections. The fungal pycnospores germinate in droplets of water at a high humidity of more than 92% and a temperature of +15 to +20 °C. The incubation period is 5–7 days.

Optimum conditions for pathogen development. High intensity develops of pathogen are observed in warm weather with frequent rains. The fungus is wintering The fungus are wintering in the form of pycnidia on plant residues and in winter crops on infected perished of leaves [29]. In early spring, spots first appear on the leaves of winter wheat, and then the infection dispersed to spring wheat. The disease develops more severe in years with high snow cover, late spring and at long snow melting. The perish of winter wheat from ascochitosis is observed in individual years favorable for the development of the fungus, when winter crops sufficient undeveloped and in years with humid and warm summers. Since fungi are wintering on plant residues or in the soil, and conidia spread by raindrops, the initial infection occurs directly on the ground. *Ascochyta* species usually cause damage to both dicotyledons (soybeans, peas) and monocotyledonous crops (wheat, oats, barley, rye, triticale) [28, 37].

At the end growing season on the spots and on the underside of the leaves, perithecia of the teleomorphic stage can form. Asci are cylindrical 30–60 × 10–16 μm, in each of them 8 colorless 2-cell ascospores, 9–16 × 4–5.5 μm, are formed. The fungi are wintering with mycelium, perithecia and pycnidia on plant residues, winter crops and spore-infected seeds, in spring the infection is renewal by ascospores.

Injuriousness. The harmful effect is the premature drying of the leaves. With more degree of manifestation of disease, the leaves of wheat perish off prematurely, the infected plants are 10–15% lower than healthy ones and are reduced.

Infecting with this pathogen of cultivated plants does not lead to significant economic damage compared with other fungal diseases. It is also necessary to take into account the fact that the use of pesticides in agroecocenoses helps to suppress more aggressive pathogens, as a result of which a weak pathogen can cause serious problems. So, in favorable for the spread of the year crop losses can be 15–20% and more.

2.19 Powdery mildew

Causative pathogen: highly specialized parasite *Blumeria graminis* (DC.) Speer (synonym *Erysiphe graminis f. sp. tritici* É.J. Marchal) – Fungi, Dikarya, Ascomycota, Pezizomycotina, Leotiomycetes, Leotiomycetidae, Erysiphales, Erysiphaceae.

The fungus has both teleomorph and anamorph stages of the life cycle. It is also known about ability to crossing for different species Powdery mildew from different species of cereals plants.

Dispersion. Spread of diseases is ubiquitous [3, 7, 12, 37]. This disease is common for moderate climate, but dispersal area it is expanding following the irrigation intensive used at high unbalanced norms of nitrogen fertilizers applicating and modern semidwarf of genetically similar of varieties have been growed.

Infection of plants is occurs at a temperature of 0–20 °C and high humidity (50–100%). At an optimal temperature (15–20 °C), the life cycle of *B. graminis* can end in 10 days, but at a temperature above 25–30 °C, the development of the fungus slows down, therefore the disease does less injurious in time of hot summers.

The incubation period is 3–11 days (an average of 4–5). The main factor in the spread of powdery mildew conidia is the wind. Powdery mildew on winter wheat crops appears and develops since autumn. Infection reservoir of this pathogen is sprouting of fallen seed. Pathogen are wintering both on wheat plantings and sprouts of fallen seed in form accomulation of mycelium. There reported sometimes, that conidia can overwinter together with mycelium, while maintaining their viability.

The intensive development of powdery mildew is observed on shaded plants and in under the conditions of a short period of illumination. The early crops of spring wheat are resistent to infected, than the late ones, and winter wheat – vice versa [24, 37].

Symptoms. Infection can occur in a fairly wide range of temperatures and humidity [11, 13]. For the defeat of plants by powdery mildew, the most favorable is moderate temperature (15–22 °C), cloudy weather and high humidity (75–100%). Because of this, in Ukraine, damage to wheat plants by powdery mildew is observed almost every year. Symptoms appear on the leaves of the sheath, the wheat ears, the spikeds, glumes, spike, sometimes on the culm. On the plants, the disease is first found on the sheath of the leaves in the form of matte spots. Then often it coating spreading to upper part of leaf blade and sometimes on both sides. With the growth of plants, mycelium coating passes on to the leaves and culm, is spreads from the lower tiers of plants to the upper ones, with severe damage it covers the basal part culm and holds on the ear of wheat. It appears in the form of a white cobweb-shaped coating, consisting of mycelium, conidia and conidiophores. Later, the mycelium coating has been indurated, acquires a powdery appearance, and take one`s place on the organs of plants, forming cotton-like pads, which at the end of the growing season turn yellow-gray. Over time, small black cleistothecia appear on aging pads.

Affected plant tissue becomes chlorotic, necrotic, and perished off a few days after infection (Appendix 2.11). The pathogen forms a conidial and teleomorphic stage. The pathogens overwinters as mycelium on winter crops or as cleistothecia on plant residues.

Mycelium is superficial, apressorias are formed at the ends of hyphae in the form of flat thickenings for attachment on the surface of plants. Haustorium, which has the form of an elliptical swelling with finger-shaped outgrowths, and with the help of which the fungus absorbs nutrients from the plant, depart from them into the tissue cells of the plant. The pathogen forms a conidial sporulation (anamorphic stage) and a teleomorphic stage. Conidia are unicellular, colorless, cylindrical or barrel-shaped, 25–30 × 8–10 μm, form chains on unicellular, slightly elongated conidiophores. The fungal teleomorph is characterized by the formation of cleistothecia with asci and ascospores on mycelium. Cleistothecia are rounded, first brown, with time black, 135–180 μm in diameter, have a small amount of light, short appendages. They form several asci, 70–100 × 25–40 μm. Each asc contains 4–8 colorless elliptic ascospores, 20–23 × 11–13 μm. The pathogen can develop in a mono- or dicyclic type. The first is characterized by the appearance and development of conidial sporulation from the 3-leaf stage till waxy ripeness stage. In the booting stage of wheat, the fungus begins to form a teleomorphic stage, but asci with ascospores are formed slowly, and their maturation occurs only after overwintering of cleistothecia. The second type is characterized by the fact that the pathogen hibernates in the form of mycelium, and the formation of conidia begins with waxy ripeness stage. A teleomorph is formed from the end of tillering until the beginning of the boot stage, and the maturation and spread of spores from the last of summer and in autumn. During plant vegetation, the fungus can spread by conidia and ascospores.

Optimum conditions for pathogen development. Infection occurs with conidia and spores at a temperature of 3–31 °C (optimum 15–20 °C) and relative humidity 60–100%.

Injuriousness. The negative effect of the defeat of powdery mildew is manifested primarily in a decrease in the assimilation surface of the leaves and the destruction of pigments. With a severe defeat, tillering decreases, the heading stage and grain maturity were inhibited. A decrease in the density of the productive planting, the number and size of grains as a result of damage by powdery mildew to plants of susceptible wheat varieties leads to a decrease in yield from 10–15 to 30–35 and even 45% at epiphytoxics. Powdery mildew can cause a significant shortage of the crop if the infection occurs in the early phase of plant development under favorable conditions and a high infectious

background before the heading-flowering stage. The development of the disease is promouting by early sowing of winter crops, increased unbalanced norms of nitrogen fertilizers.

2.20 Tan spot

Causative pathogens: *Pyrenophora teres* Drechsler **(**synonym *Pyrenophora graminea* S. Ito & Kurib), anamorphic stage – *Drechslera teres* (Sacc.) Shoemaker; *Pyrenophora tritici-repentis* (Died.) Drechsler, anamorphs – *Drechslera tritici-repentis* (Died.) Shoemaker; *Helminthosporium tritici-repentis* Died. – Fungi, Dikarya, Ascomycota, Pezizomycotina, Dothideomycetes, Pleosporomycetidae, Pleosporales, Pleosporaceae.

Dispersion. The first cases of manifestation of wheat disease were recorded in the middle of the last century in Canada and the USA, although the disease has been described on other cereals earlier. Until the beginning of the 90s of the last century, tan spot was not widespread in Ukraine [16], but recently, in terms of the proportion of wheat diseases in the Forest-Steppe zone, it has become equal to leaf septoriosis and powdery mildew. For the pathogen that is retained on plant residues [18], no crop rotation and mulch planting during irrigated soil cultivation turned out to be favorable factors for its distribution. In Kansas, in particular, with no-till, leaf damage increased by spotting compared with treatment and even more so compared with plowing; the same thing have been at sowing wheat after wheat compared with sowing after sorghum.

The fungus has been infected a wide range of plants of the cereal family (wheat and some wild cereals, rarely rye and barley), which can be an additional source of infection, but the distribution of various races differs on different species [24, 28, 37]. The dispersal area of the disease, especially over long-range distances, can occur by seed contamination.

The dispersal area of the disease is very wide. These are different regions of North America, South America, India, Pakistan, Australia, Hungary, Czech Republic, western regions of Russia. Intensive spread of the disease in Europe and Asia is observed only in recent decades. In Ukraine, it is everywhere.

The fungus is characterized by multiplication and conidial reproduction. The disease is spread by conidia by airborne droplets, especially at high humidity. Wheat infection occurs over a wide temperature range, but spore germination requires leaves to be moist from 12:00 in susceptible varieties and up to 24 in medium resistant [16–18]. The sources of infections are plant residues and infected cernel. On the leaves

and stems that have wintered, a marsupial stage is formed – black perithecia with ascospores, which can infect plants in spring and are an additional sources of infection. In this stage, the causative agent of Tan spot is called *Pyrenophora graminea* S. Ho et Kuribay. For development of disease are promote both early first sowing and abundantly apply nitrogen.

Symptoms. The disease is common, but its diagnosis is difficult, because the symptoms of pyrenophorosis resemble atypical septoriosis. The initial lesion is caused by infection, that kept on plant residues in the soil or on infected of wheat plantings. As a rule, the disease first appears on the lower leaves and, under conditions favorable for the development of the disease, gradually spreads to the upper leaves and sheath. The disease appears on both sides of the leaves and leaf sheaths in the form of small single or numerous spots of oval or round configuration, yellow or light brown, with a diameter of 2–5 mm. In the center of the spot, the epidermis is slightly elevated. On some leaves, in the center of the affected area, brown necrotic spots are formed with a diameter of 1–2 mm, which over time grow in the longitudinal direction, become dark brown, 12–20 mm long, sometimes acquire a rhombic or lenticular configuration, that usually bordered by yellow or light brown chlorosis zone. During this period they do not differ in color from spots of septoriosis, but do not form pycnidia. The spots can be in the form of stripes, occupy a third or even more than half of the leaf surface. At last of growth season, on dark spots and after complete drying of the leaf, a dark-gray or olive-brown bloom of conidial sporulation is formed (Appendix 2.12). The maximum development of the disease occurs in time of the flowering stage. In places of infection, the leaves have dry and fall. On the leaves of resistant to disease plants, the spots do no significantly ratchet up in size or remain unchanged. The fungus can cause damage to the upper leaves and ears wheat, forming elongated spots, 2–4 × 1–2 mm, which do not grow. At the infection of wheat ear the mycelium penetrates in-kernel and induces in-infected of grain without external signs. In sensitive of wheat species, diffusive expand of mycelium is observed. Sources of infection are plant residues and seeds on which the mycelium is hold, asci with ascospores.

Optimum conditions for pathogen development. The causative agent of the disease develops in a wide range of temperatures, especially with a long period (18 hours) of dew or rain.

Injuriousness. Harvesting grain under the condition of epiphytic development can decrease by 4.5 times. The disease leads to the perished of leaves, a significant decrease in the density of crops, a decrease in the assimilation surface of plants, causes the loss of affected

plants, as a result, the formation feeble and coarse grain, a decrease thousand-kernel weight, that lead to reduction in yields and its quality. The disease is especially harmful in fields with min-till or mulch planting, which causes pathogen infection to accumulate on crop residues. Crop losses can be up to 50%.

2.21 Cercosporella root rot

Causative pathogen: *Ramulispora herpotrichoides* (Fron) Arx (synonym *Pseudocercosporella herpotrichoides* (Fron) Deighton) – Fungi, Ascomycota, Pezizomycotina, Dothideomycetes, Dothideomycetidae, Capnodiales, Mycosphaerellaceae.

Dispersion. The disease is common in the western and central regions of Ukraine [3].

Symptoms. The causative agent of the disease causes blackening of the roots, the underground internodes and the basal part culm of wheat. On the first, are forming oblong oval spots with a blurry brown or pink border and at severe lesion on the following internodes hed too.

A dark stroma is often forms inside these spots and bears certain similarity the "eye" (spotted spotting). The lesion can also appear on the leaf sheaths. With severe damage, when spots encircle of the culm to half or more, it breaks. This causes a chaotical lodging of wheat planting, as opposed to unidirecrtional lodging in one direction at heavy rain or wind [3].

The pathogen forms a conidial sporulation at sufficient moisture. Conidiophores are in the form of lateral branch off, colorless, short, cylindrical, 1–3-septate. Conidia are colorless, needle-shaped, in the upper part often curved, with 4–8 septa, mainly 50–70 × 2–4 μm, in the upper part – 1.0–1.5 μm. Secondary conidia are formed at germination, similar to primary, but have a cylindrical shape. The optimum temperature for the development of the fungus is 5–9 °C, but it easily withstands ten degrees of frost. In the soil on the infected residues, it remains viable for about 18 months. Infection of wheat crops occurs in early spring in the boot stage.

Optimum conditions for pathogen development. The optimum temperature for infection is about +9 °C. On winter wheat, the pathogen develops especially intensively in cold rainy autumn, warm winter with thaws as well as cold of spring in with a large number of cloudy days.

Injuriousness. Causes fragility of stems and their chaotical lodging. The reduction in yields at the disease can reach 30% or more. Varieties resistant to the causative agent of the disease are not known. Sources of infection are plant residues on which the fungus can keep pathogenic for up to three and a half years in the form of mycelium.

2.22 Eyespot (Strawbreaker)

Causative pathogens: *Oculimacula acuformis* (Nirenberg) Y. Marín & Crous, neuter: *Helgardia acuformis* (Nirenberg) Crous & W. Gams, *Pseudocercosporella herpotrichoides* var. *acuformis* Nirenberg, *Ramulispora herpotrichoides* var. *acuformis* (Nirenberg) Boerema, R. Pieters & Hamers; *Oculimacula yallundae* (Wallwork & Spooner) Crous & W. Gams, анаморфи: *Cercosporella herpotrichoides* Fron, *Helgardia herpotrichoides* (Fron) Crous & W. Gams, *Pseudocercosporella herpotrichoides* (Fron) Deighton, *Ramulispora herpotrichoides* (Fron) Arx – Fungi, Dikarya, Ascomycota, Pezizomycotina, Leotiomycetes, Leotiomycetidae, Helotiales, Dermateaceae.

Dispersion. This disease affects wheat, triticale, rye, oats and other genera of the cereals [37]. More susceptible to it is winter wheat. Strawbreaker spotting often occurs in areas with cooler climates, where cereal crops are sown mainly in the winter.

Symptoms. The most characteristic signs of this disease are elliptical spectacle spots on the lower internodes of the stem. Often they develop at the level of the soil with leaf sheaths, appear as expressive spectacle spots, in the center of straw yellow, can be coal-black, surrounded by a dark brown or dark green border (Appendix 2.13). With a strong development of the disease, feeble culm with the lesion can break at the level of the soil or lodging without symptoms of root rot [24, 37]. The initial infection is transmitted from conidia and mycelium stored on plant residues on the surface of the soil in contact with coleoptile and the lower part of the young shoot.

Optimum conditions for pathogen development. Cool, damp weather and high humidity at soil level are favorable for the development of the disease.

Injuriousness. As a result of the defeat, perished singular of culm, sometimes even whole plants. Crop loss is mainly take place a decrease in the weight and number of grains in the ears, as well as lodging.

2.23 Flag smut

Causative pathogen: *Urocystis agropyri* (Preuss) A.A. Fischer Waldheim – Fungi, Dikarya, Basidiomycota, Ustilaginomycotina, Ustilaginomycetes, Urocystidales, Urocystidaceae.

Dispersion. Soft wheat is the main host of stem smut, it occurs predominantly of focuses, and is more characteristic of the southern regions and Crimea. The causative agent of the disease is supported exclusively on soft wheat, but there are data that durum wheat and triticale may be infected with stem smut too. The disease is common

in areas of cultivation both winter as well as spring wheat in time autumn sowing [24, 28, 37].

Symptoms. The disease develops systemically, forming subepidermally bands of teliospores that are noticeable in heading stage of wheat. The teliospores of pathogens are gathered in round, oblong or ellipsoid glomeruli. Symptoms of the disease appear on the culm, leaves and sheath in the form of elongated convex strips up to several centimeters long. Over time, these stripes become lead-blue. At the sites of lesions, the epidermis cracks and the dark mass of teliospores protrudes outwardly easily. Affected plants lag behind in growth; instead of ears and grains, a distorted mass of tissues forms.

The sources of infection of sprouted grain or very young seedlings are grain and soil contaminated with teliospores, where they remain viable for a year or more.

The teliospores of the stem smut of wheat germinate after a month of pause. They form unicellular basidia, at the top of which are 2–6 cylindrical inseparable basidiospores. They are germinate and with infectious hyphae infect the embryos. The infection process is possible from the moment of seed germination to the formation of the first leaf. Mycelium spreads throughout the herb of wheat, but teliospores are formed only in certain places.

Optimum conditions for pathogen development. The optimal conditions for infection are temperature +13 to +21 °C and low soil moisture.

Injuriousness. Flag smut is a less harmful disease, but reduction in yields has been observe an average level of disease, especially when susceptible varieties are grown. The lack of grain yield in the field corresponds to the percentage of smut affected plants. With a decrease in productive tillering, the affected plants give 5 times less yield.

2.24 Common bunt

Causative pathogen: *Tilletia laevis* J.G. Kühn (*Tilletia caries* (DC.) Tul. & C. Tul.) – Fungi, Dikarya, Basidiomycota, Ustilaginomycotina, Exobasidiomycetes, Exobasidiomycetidae, Tilletiales, Tilletiaceae.

Dispersion. The disease is ubiquitous. The source of infection is contaminated grain. An additional source of infection is soil, where teliospores can be keep for up to one year. Solid smut teliospores on other crops can remain viable in the soil for no more than two to three weeks, so for these cases the soil cannot be a source of infection. The sources of grain contamination can also be containers and seeders. At gross yield, threshing yield, and clean out of grain, teliospores spray and enter on the grain and in soil.

Symptoms. Clearly, symptoms are found at the early milky stage of ripeness [24, 28, 37]. On wheat and rye, the ear is somewhat flattened, intensely green with a blue tint, the spikelets are unnaturally splited, the glumes are apart as a result of the action of a fungal infection. When crushing the affected ears, a grayish olive-brown liquid is released, which has the smell of rotten herring due to the trimethylamine content. In the firm ripe stage, the affected ears of wheat are erect head. Instead of grain, in ears are formed bags, filled with a black mass of teliospores. The teliospores are glued together in solid, durable lumps; therefore, solid smut is also called stone smut.

Infection of plants occurs during seed germination in the soil. In time sowing, teliospores have been enter on the soil where they germinate, forming basidia with basidiospores. After copulation, the latter form an infectious hypha that penetrates the sprout. Then a mycelium is formed in the plant, which diffusely spreads, reaches the growth apex, penetrates into leaves, culms and ears. Germination of teliospores and plant infection are largely depend on temperature and soil moisture [24, 28, 37].

Optimum conditions for pathogen development. The maximum infection of wheat germ occurs at a temperature of +5–10 °C and a relative soil moisture of 40–60%. Late sowing periods are contribute to the infection of winter wheat. Pathogens of solid smut are characterized by physiological specialization at the level of species and varieties of host plants. For example, more than 20 races are identified for the pathogen *T. tritici f. vulgaris*, for *U. hordei* – depending on ecological and geographical zones – from 2 to 20.

Injuriousness. Pathogen harmfulness is caused by the formation of a spore mass instead of grain and thin planting in virtue of the perished of infected. With a severe defeat, crop shortages can be 15–20% or more.

2.25 Loose smut

Causative pathogen: *Tilletia tritici* (Bjerk.) G. Winter (also *Ustilago segetum* var. *tritici* (Bjerk.) Brunaud, *Ustilago tritici* (Bjerk.) E. Rostrup) – Fungi, Dikarya, Basidiomycota, Ustilaginomycotina, Exobasidiomycetes, Exobasidiomycetidae, Tilletiales, Tilletiaceae.

Dispersion. The disease is spread in all zones of cultivation of wheat, barley, rye. The disease is especially harmful to barley crops. The disease is detected at the heading stage.

Symptoms. The disease manifests itself in the heading stage. In infected plants, ears of wheat are look out get blackened due to the

formation of a black mass of teliospores instead of flower parts and the glumes ("husks") of ear.

The spikes of the ears are very reduced, only the rachis is intact. On early heading stage of wheat at infection of plant, the spore mass is covered with a thin transparent membrane, which then quickly collapses, and the teliospores easily fly apart (Appendix 2.14) [24, 28, 37].

The teliospores of the causative agent of the disease are small, spherical, angular or oblong, light brown, 5–9 μm in diameter, their shell is slightly lumpy. Infection of wheat with smut pathogen occurs on flowering stage. Once on the stigma of a flower, the teliospores germinate, diploid hyphae are formed, which move along the path formed by the pollen tube, or independently reach the ovary and penetrate into the seed germ. Infected seed embryos do not perish, but an almost normal seed develops. The embryo of such a grain contains fungal hyphae. In addition to the embryo, the mycelium can penetrate the pericarp, seminal membranes, aleurone layer and endosperm. At the beginning of development, the fungus forms hyphae with low aggressiveness. They have a very thin shell and homogeneous protoplasm. By the time of grain maturity, the fungus changes morphologically and physiologically. Gyphae slightly swell, their walls thicken, and fat droplets separate in the protoplasm of the cells. In this form, the fungus goes into a dormant state and can remain viable in ungrown seeds for more than three years. At the beginning of seed germination, it have been activated and infects plant sprouts. The mycelium spreads diffusely along the stem, and sometimes it even appears on young leaves. With the development of an ears, hyphae grow and thicken, their walls become gelatiniform [24].

The fungus of *U. tritici* does not form basidiospores and the cells of its basidia are dioecious; copulation occurs between cells in the same basidia or between different basidia. Procopulation of basidium cells gives rise to diploid hyphae. They form a gelatiniform mass, in which teliospores are differentiated by division, and instead of parts of the ears, a spore mass is formed. Spores are viable for two hours, analogues from ears of later heading stage, have less virulence.

Optimum conditions for pathogen development. High humidity and 18–24 °C temperature are favor for infection of plantings wheat with prolonging the flowering stage of the host plant.

Injuriousness. It is happens in all areas of wheat cultivation. Loose smut is very harmful. Infected plants do not yield. The mass of the herb of the infected plant is 30–40% less than healthy. There are significant inhibition and reduction in yields. Crop losses depend on the number of spikes in ears infected and are usually around 1%, but can sometimes reach 30%. Some plants bounce back, but the quality and size of the crop is reduced. In plants, the fungus stop to grow at 7–8 °C, which

explains the lesser damage of winter wheat in late sowing and spring wheat with early.

2.26 Dwarf bunt

Causative pathogen: *Tilletia controversa* J.G. Kühn – Fungi, Dikarya, Basidiomycota, Ustilaginomycotina, Exobasidiomycetes, Exobasidiomycetidae, Tilletiales, Tilletiaceae.

Dispersion. It is widespread, develops in temperate climates, and is more common in areas with long-time of snow cover. In addition to winter wheat, it infects triticale and wild cereals (except of couch grass). The disease was not detected on spring wheat. It spreads mainly in the western regions of Ukraine.

Symptoms. Infection of winter wheat occurs on the surface of the soil at the time of emergence of seedlings and until the boot stage. The causative agent develops systemically in the infected plant and reaches the carpel. Subsequently, the mycelium in the spike is transformed into dark dense fragments, from which teliospores are subsequently formed.

Visible symptoms of the disease appear after the heading stage of wheat. In appearance, its disease is very similar to a smut, but differs from it in biological and environmental features. The infected plants exhibit symptoms of dwarfism (growth retardation), they are very intensivity tillering, forming up to 30 stems or more. The length of such stems, as a rule, is 2–4 times less than in healthy plants. Signs of the disease are noticeable in the milky-waxy stage of ripeness. The infected wheat ears are dense, shortened, in some cases branching of the wheat ears is observed. In plants infected by dwarf smut, the grain has a more spherical form. Infected ears of wheat completely retain the structure, only instead of grains smut sacs filled with teliospores of the fungus are formed. The teliospores are spherical form with 19–27 μm the shell diameter, brown colored, with a well-defined retina. Their viability is maintained up to 7–9 years. Destroyed smut sacs have an unpleasant herring smell. Ears affected by the smut are bluish-green or lead-gray and glumes ("husks") of spikelet are slightly open up (Appendix 2.15) [24, 37].

In harvest time, the smut sacs of the Dwarf bunt are dehiscent and the teliospores falled to the ground. Spores of Dwarf Bunt germinate only on the surface of the soil without a dormant period, germinate on the surface of seeds and affect seedlings of wheat. Sources of infection are infected both seeds and soil. Wild cereals can also be the reservoir of infection. Infection of plants occurs the boot stage till, especially for min-till areas.

Optimum conditions for pathogen development. For germination of spores and the formation of infectious mycelium, 0 °C to +5 °C temperatures are required for 3–5 weeks and poor lighting (100–150 lx).

Injuriousness. Dwarf bunt is more harmful than common bunt. Affected crops practically do not yield crops. Significantly large crop losses are caused by the cultivation of disease-susceptible varieties and using not treated grain.

2.27 Brown (leaf) rust

Causative pathogens: *Puccinia triticina* Erikss.; *Puccinia recondita f. sp. tritici* D.M. Hend.) – Fungi, Dikarya, Basidiomycota, Pucciniomycotina, Pucciniomycetes, Pucciniales, Pucciniaceae.

The causative agent of the disease is an obligate parasitic fungus with a complex development cycle. Brown rust can develop in incomplete and complete cycles. With an incomplete development cycle, urediomycelia of rust hibernates on winter wheat, and in the spring – urediospores on winter crops, and then spring crops.

Dispersion. The disease is common in all areas of cereal cultivation [21, 24, 28, 37, 39]. This disease is the main disease of wheat crops in Ukraine. Epiphytotic development of brown rust is observed once every three to four years. Disputes can be carried by the wind for hundreds and thousands of kilometers, which leads to the emergence of new disease genotypes in wheat growing regions. Natural barriers to propagation for its can be oceans and mountain ranges. Thus, the pathogen population from the countries of Transcaucasia, Turkmenistan, Uzbekistan, Tajikistan, and Kyrgyzstan was significantly different from the pathogen population of Kazakhstan due to the Tien Shan mountains, delimiting these regions, and both populations were significantly different from the pathogen population of North America. The formation of the fungus forms occurs due to mutations and through sexual reproduction on alternative species of host plants. An important center for the dispersal of the fungus in the European part of the former USSR is the Dagestan region. The regions with the highest prevalence of the disease are Europe, Central and South Asia, North and South America [33].

Symptoms. Vegetative the uredial stage on wheat plants gives several generations during the vegetation period of plants [10]. Infection can persist on other spesies of cereals, due to which a fungus can exist in uredostadia for several years without a full development cycle. New generations of uredospores can form every 10–14 days under favorable conditions. Before the wax ripeness of the grain, as well as under ad-

verse weather conditions, a mass of black teliospores develops on the leaves. Primary infection is insignificant and mainly develops as a result of long-distance transport of air.

With a full development cycle, the teliospores germinate and form the sexual stage – basidia with basidiospores. The latter affect the intermediate host plants, that are some species of *Thalictrum*, *Isopyrum*, *Anchusa*. On these plants, an ecidial stage develops with aecidiospores. Aecidia usually are formed on the underside of leaves. The aecidiospores are spherical or ellipsoid oblong, with a yellow or colorless shell, finely warty, affect the leaves of wheat, on which uredostadia are formed. In addition to wheat, the causative agent can also affect other cereals, such as rye, barley and cereal weeds. It is an additional source of infection. The fungus has many races that are dangerous for some varieties of wheat.

The first symptoms of the lesion appear mainly on the upper side of the leaves, sometimes on the stems and internodes of the stem. Uredopustules are round or slightly oval, somewhat smaller than in stem rust, do not merge with each other, usually contain a lot of orange, orange-brown ureodospores (Appendix 2.16). First they are covered with epidermis, later released and dispersal. After 10–15 days, black pustules are formed on the underside of the leaves. Around the uredopustules, necrotic and chlorotic rims often are formed, during the period of sporulation, they can form a lot of spores (about 35 thousand spores in the pustule). Uredopustules can germinate and create new foci of infection immediately after ripenig. With a severe defeat, the leaves prematurely are turn yellow and dry out. Symptoms may occur for all growth season, but most often appear on the heading stage.

Optimum conditions for pathogen development. Uredospores germinate only upon contact with drip–liquid moisture and at the temperature of about 20 °C, under such conditions, the disease can develop very quickly [1]. The fungus develops in a wide range of environmental factors, with a temperature of 20 °C being optimal when three hours are enough for infection, and 2–30 °C are permissible [33]. At the optimum temperature and the presence of moisture on the leaf, infection occurs in 4–5 hours. At a lower temperature, the rate of infection slows down to 7 hours, and at a temperature of 30 °C infection does not occur. The duration of uredostadia also depends on air temperature. At the optimum temperature, it ends in 6–8 days, and for its decrease – more than 20 days [24, 28, 37].

Injuriousness. Infection begins in the fall, but a period of high severity falls in the heading – wax ripeness stage. The harmfulness of the disease is to reduce the assimilation surface of the leaves, impaired

respiration and water balance (increased transpiration), which leads to metabolic disorders, causes premature perish of leaves and a decrease in the number of roots, worsens the wintering of winter crops. As a result, brown rust leads to premature ripening, a decrease in the quantity and underdevelopment of grain. Affected plants exhibit low drought tolerance. Since pathogens of cereal diseases are biotrophs, resistance against them is "vertical" and often manifests as a hypersensitivity reaction: in resistant varieties around the pustules appears chlorotic borders, and in immune samples chlorotic spots remain sterile without sporulation of the fungus.

Plant residues on which the pathogen hibernates are the main sources of brown rust infection.

2.28 Stem (black) rust

Causative pathogen: *Puccinia graminis* Pers. – Fungi, Dikarya, Basidiomycota, Pucciniomycotina, Pucciniomycetes, Pucciniales, Pucciniaceae [24, 37–39].

Dispersion. The causative agent of stem rust is infects of wheat, barley, triticale and some wild cereals [23, 28, 37, 39]. Intermediate hosts are the species *Berberis* L. and *Mahonia* Watt. About 410 species of cereals are known that are known hosts of *P. graminis*. The fungus is spread around the world from West Asia to the regions where agricultural lands are located. Local and regional dispersion is carried out by the wind. In Ukraine, it is happens almost everywhere, but harmfulness manifests itself only in some western regions.

For a growth season (depending on environmental conditions), the fungus can give several generations of uredospores, which explains the rapid spread of the disease. The causative agent forms a large number of uredospores – 5000 spores / day per uredium and up to 1000 uredinia per the culm of a plant. Aerial dispersal of spores over long-range distances are occursed with streams of hot air, when spores rise to a height of 3000 m. At this height, spores can spread hundreds of kilometers.

Symptoms. The causative agent is affects the stems, leaves sheath base, sometimes spikes and main axis or rachis of the ear. Rusty-brown spots are appeared on them, which turn black by the end of summer and become convex. Pustules containing a mass of uredospores, dark red-brown, is appear on both sides of the leaves, on the stems and ears (Appendix 2.17). With a weak infection, solitary pustules usually are formed, and with a strong one, they merge with each other. First, pustules can form the spots, then the spore mass breaks the epidermis of

the leaf. The presence of infection can be easily detected by touching the surface of the affected tissue, that is notable in inroughness and small tears in the tissues.

Primary infection (uredospores) is carried by aerial currents. The spores maybe carried over considerable distances. At the grain maturity stage, a mass of black teliospores is formed.

The fungus is dioecious: spermagonial and aecidial sporulation are formed on the species of barberry (*Berberis* L.) and magonia (*Mahonie* Watt.), and uredo- and teliostages – on winter wheat. On the leaves of barberry *P. graminis* forms single spermogonies or small groups of them on the upper side. They are spherical, dark yellow, 120–130 μm in diameter. They are formed from a large number of small, light, unicellular spores – spermacia, with the help of which fertilization of other spermogony occurs. This explains the formation of new biological forms and races of the fungus. It has been established that some fungi can grow on very complex in composition artificial media. In 2–5 days after spermogony, on the lower side of the leaves of barberry, and sometimes on the petioles and young shoots, aecia are formed, which are placed in round or oblong groups. Aecia are cylindrical-cup-shaped, with curved edges, whitish-yellow, 2–3 mm in diameter. Peridium cells are tightly connected, rectangular, have an outer wall of the membrane 7–13 μm thick with warts [24, 37].

Aeciospores are spherical, 14–22 × 12–18 μm, with yellow contents. The sporoderm is colorless, thickened at the top, covered with small warts. The ecidiospores are dispersing and, having fallen on cereal grains, germinate in the presence of drip-moisture and 5–24 °C temperature. At the zone of aeciospore germination, mycelium (uredomycelia) develops, on which uredinia are formed. Uredinia are rusty-brown, oblong, linear, fused, are formed on the culm, leaf sheaths, leaves, spike and glumes (`husks`) of spike. The uredospores of *P. graminis* are unicellular, oblong, elliptical, yellow, with 20–42 × 14–22 μm size and grow in drip-liquid moisture at 1 to 30 °C (optimum is 18–20 °C) temperature.

At the end of the growing season, teliospores are appeared. They are developing in the places of formation of uredinia and often form black stripes up to 22 mm long. The teliospores are bicellular on a long stipes, oblong-club-shaped, with a thickened sporoderm at the apex, brown, smooth, 35–60 × 12–22 μm.

Optimum conditions for pathogen development. The pathogen have been wintering on infected of plant residues, especially on stubble, and germinates in the spring after a dormant period at 9–29 °C (optimum is 18–22 °C) temperature and 95–100% air humidity. The time is required for germination of teliospores depends on their degree of ma-

turity. Mature teliospores can germinate 3–4 hours after being placed in a humid chamber. From teliospore cells, basidia with basidiospores are formed. Linear rust is more pronounced in the early crops of winter and late crops – spring wheat.

The disease is developed very quickly at the presence of drip moisture (rain or dew) and the prevalence of moderate temperature. At an average daily temperature of about 20 °C and above, a newer generation of uredospores is appeared within 10–15 days.

Injuriousness. Harmfulness is appear in distortion of the water balance of plants (increased transpiration). When the culm is damaged under the ear, the expected yield sharply decreases caused by the so-called grain outflow. At that, ears wheat are contain of shrunken kernel, with very low baking qualities. Crop losses can reach 20–25%, sometimes with significant development of linear rust, crop shortfall is 60–70%. With the early manifestation of the disease, crop losses can be high caused by a decrease in the number of grains in the ears, their weight and quality. If the infection occurs in the early stages of plant development, the consequences can be very serious – tillering is reduced, the mass and quality of the grain are reduced. Affected plants are less resistant to drought, ripen unevenly, and may lodging. Under favorable conditions for the development of the disease, the crop may be completely lost. With the advent of a very virulent strain of Ug99, the harmfulness of this disease has again increased significantly, so an intensive search is underway for new sources of resistance to this pathogen. In Ukraine, the severity of this disease is not very large.

2.29 Yellow (stripe) rust

Causative pathogen: *Puccinia striiformis* Westend. (syn. *Puccinia glumarum* Erikss. et Henning) – Fungi, Dikarya, Basidiomycota, Pucciniomycotina, Pucciniomycetes, Pucciniales, Pucciniaceae.

Using molecular and morphological studies, striped rust pathogens have been divided into four types *Puccinia striiformis sensu stricto* (on *Aegilops*, *Elymus*, *Hordeum* і *Triticum*), *Puccinia pseudostriiformis* (on *Poa*), *Puccinia striiformoides* (on *Dactylis*) and *Puccinia gansensis* (on *Achnatherum*).

Dispersion. There is a constant threat in countries where wheat is grown in winter or at high altitudes [20, 24, 37, 39]. This species is common in Europe, in recent years has become a problem in Australia. It infects wheat, rye, barley, grasses and wild cereals. It occurs everywhere, in Ukraine most often in the forest-steppe and in Polissya, as well as in the foothills and mountains. The intermediate host plant was unknown until it was discovered in 2010 in the USA on the

ordinary barberry and a number of other species of Berberis spp. *Berberis vulgaris* (European barberry), which was a historically important source of inoculum in North America and Europe, but now it has become rare thanks to the law on its destruction. In some regions, especially Eastern Europe and Western Asia, common where barberry grows, it is involved in the life cycle of *Puccinia striiformis* rust and can contribute to the evolution of new virulent combinations. The most significant yellow rust damage is observed in regions with a cool climate and higher altitudes. The development of the pathological process is delayed at +20 °C to +25 °C.

Symptoms. Typical for the early stages of epiphytotics of yellow rust is its nidus manifestation. On the surface of leaves are formed typical for the disease, dashed oblong linear stripes of uredopustula, from bright orange to lemon yellow, 7–11 cm long (Appendix 2.18). Pustules can also develop on all aerial parts – the sheath of leaves, the internodes of the stems and the ears of wheat. In pustules are formed yellow or orange-yellow uredospores. Sources of infection affect wild grasses, fallen grain, wheat plantings [20, 24].

The causative agent of yellow rust is wintering in uredostage on plants infected in autumn. The formation of spermogonial and aecidial stages has not been established.

Optimum conditions for pathogen development. The primary infection is caused by uredospores, which can be carried by the wind over considerable distances at the +11 °C to +13 °C is optimum temperature for spore germination. Infection occurs in the dark and depends on the presence of liquid droplet on plants. In the case of a warm winter or early spring, infection can begin even at temperatures above 3 °C. The minimum period for leaf infection is three hours and the optimal one is eight. The first uredospores appear at an air +10 °C to +20 °C after 11–15 days, at +5 °C – about 25 days. The optimal temperature range for epiphytotics is +9 °C to +11° C and a high level of humidity. At low winter temperatures and a low level of insolation, the latent period can last 60 days or more. High humidity and precipitation are favorable conditions for increasing infection in the form of epiphytotics on both sides of the leaf blade and ears. Epiphytotic infection occurs mainly only in May. At temperatures above 20 °C, the formation of uredospores ceases and teliospores often develop.

Injuriousness. Infected plants are stunted and weakened, form a smaller number of ears and shriveled grains. By mass destruction of crops, the number of grains in the ear, thousand-kernel weight and quality are reduced. The causative agent affects both winter and spring wheat, crop shortages can reach 40–50%, with a significant degree of damage up to – 100%.

2.30 Wheat protection management against fungal pathogens

Important measures to protect crops from fungal pathogens are agrotechnical, genetically, selective and biotechnological ones, namely, the selection of early and resistant varieties, including varieties that has been resistent to infecting with fungal pathogens Mironovska 61 and Donetska 46, and quick harvesting and drying to 14%, balanced mineral nutrition with trace elements (manganese, boron, copper), tillage (fall tillage), reclaiming. Application of fungicides for plantings should be carried out during flowering and, if possible, when weather conditions favorable for infection occur [2–4, 6, 11–14]. Chemical preparations must be used in accordance with technological schemes for growing wheat, in particular preliminary chemical disinfection of seeds (Alfa-Standard, Venzedor) [4, 6, 11–13]. Seed treatment with Kinto Plus seed dressers (BASF) are recommended.

The effectivive measures against Fusarium root rot as well as against both root rot and FHB are: timely harvesting; with a separate method, one cannot allow a long stay of the mowed wheat in the field, especially in rainy weather; planning an effective crop rotation, preliminary chemical disinfection of seeds; treatment of infected crops with fungicides in the milky-waxy stage of ripeness [2–4, 6, 11–14].

Reducing the defeat with the Ergot is facilitated by the purification of seed material from them, the cleaning of fields from plant residues, and ect.

Notably, the most fungicides are preventive rather than therapeutic, that is, early recognition of symptoms is very important. The use of biologically active drugs, in particular Trichodermin, 2–10 l/ha can also be effective; Planriz BT, 2 l/t.

Monitoring of fields during periods of possible primary manifestation of pyrenophorosis is an important technique for the timely detection of a disease and subsequent effective infection control. Given the aggressiveness of pyrenophorosis, when choosing drugs, preference should be given to drugs with two to three active substances: Soligor 425 EC ke 0.7–0.9 l/ha, Virtuoso ke 0.4–0.5 l/ha, Bumper Super 490 k.e. 0.8–1.2 l/ha, Acanto plus 28 hp 0.5–0.75 l/ha, Ikarus 250 c.u. 1.0 l/ha, Impact K HP 0.6–0.8 l/ha, Impact T k.c. 1.0 l/ha, Impera Gold 490 k.e. 0.8–1.2 l/ha, as well as Alto 400 SC, Real 200 and others.

If the degree of damage by spot spotting of normally developed stems to the end of tillering is 15%, and of the remaining stems – 50%, and in the booting stage of wheat – 15 and 30%, respectively, then applicating fungicides against the diseases of the plants (Phoenix Duo). Chemicals: Alto 400SC, Benlat, Mirage, Rex Duo, Sportak, Tango, Fundazole.

The great importance in reducing plant damage is the sowing of seeds in soil with a moisture content of not less than 60–70%, the prevention of repeated sowing in the same field (crop rotation), and using seed dressing [4, 6, 11–13].

Brown rust protection measures include: using crop rotation; early sowing; control of alternative plant hosts; cultivation of resistant varieties; thorough cultivation of the soil to remove plant residues, treatment of plants with fungicides at the first sign of illness on the lower leaves. Biological protection (in areas of poor development of the disease) includes pre-sowing seed with Fitosporin–M, F and foliar spraying in the boot– heading stage with Fitosporin–M, F, Gamair, SP or Bactofit, SP, chemical – treatment with fungicides [4, 6, 11–13]. The recommended treatment with fungicides based on Prothioconazole – Fandango (Strobilurins, triazole), for crops with a higher yield potential – SiltraXpro (Prothioconazole +Bixafen), that increases the yield better than Fandango. Also, when the first pustules appear, 3–5% of plants are effectively treated with the fungicidal preparations Alto Super (triazole) – 0.4–0.5 l/ha, Amistar Extra (triazole, strobilurins) – 0.5–0.75 l/ha; Amistar Trio (triazole, strobilurins) – 1 l/ha 30 g/l – against rust, Ditan M–45 (dithiocarbamates) 2–3 l/ha – on winter wheat against brown rust), Tilt (triazole) – 0.5 l/ha; Elatus Ria 385 CE (carboximides, triazole) – 0.4–0.6 l/ha; Tilt Turbo 575 EC, CE (phenpropidine, prothioconazole) – 0.8–1.0 l/ha.

Measures to protect against stem rust include agricultural practices such as early sowing (in autumn or spring) using early ripening varieties, which helps reduce the time that the pathogen affects crops. The effectiveness of this approach depends on a detailed knowledge of the epidemiology of rust in a particular region.

To effectively overcome the disease, it is also important to consider the presence of alternative hosts (barberry and wild herbs), which can be additional sources of infection and timely control over their disperstion in the regions where cereal crops are located.

Knowledge of endemic species of rust, their host ranges and inoculum movement is important, especially if cereals and feed are planted in the immediate vicinity. A possible biological control agent, although not very promising, may be *Darluca filum*, which is one of the most effective hyperparasites that can infect a number of rust pathogens, but its effectiveness is doubtful due to the ability of rust to quickly disperse over long distances and the difficulty for accumulate sufficient quantities of this microorganism [22]. In addition to the recommended ones [4, 6, 11–13], it is also necessary to take into account that resistance to a certain fungicide may develop in a pathogen, in which case fungicides

with a multicomponent composition of chemicals can be used, and alternation their different types.

2.31 Diagnostic methods of wheat fungal diseases

For the right choice of protective measures, it is necessary to have accurate data on the causes of the disease. Diagnosis and registration of diseases of cultivated plants and monitoring of their development is the basis for the development of these measures. When choosing methods and timing, they take into account not only the causes of the disease, but also the species composition of causative pathogens and the state of crops. Any diagnosis begins with an analysis of the symptoms of the disease, while it is necessary to sound out general of the plant health status. Then, fertilizers and pesticides rate are found, that have been used in this territory, the nearing manufacturing facilities, weather conditions. Dryness data are also important for diagnostics, spring and autumn chill, amount, intensity distribution of precipitation, hailstorm, the thickness of the snow depth, etc. However, it is possible to accurately identify the causative agent of the disease only in isolated cases. For a final conclusion on the causes of the disease using special methods for the diagnosis of pathogens [15].

The main methods of disease monitoring. General provisions

Integrated plant protection consists of 4 blocks: monitoring of causative pathogens, analysis of information, elaboration of measures of effective protection of plant. However, monitoring should provide regular collection of information on abiotic environmental factors and the population of harmful organisms [5].

At the intermediate stage of identification of the pathogen and, accordingly, the timely practices of crops protection it is necessary to constantly monitor the spread and dynamics of the development of diseases, especially rust, septoriosis, pyrenorosis and other. For this, 2–3 typical stationary of plot or fields are selected and, during the main phases of plant development, weekly or once per 10 days, observations are made under favorable weather conditions for plant infection and disease development. General monitoring of the phytosanitary condition is carried out during the period of mass spread of diseases, taking into account the varietal diversity of crops in mind of growing wheat plantings after various predecessors and different planting times [8].

A visual assessment of the intensity of the development of diseases is usually carried out on a 4-point scale: 0 points – plants are healthy; 1 point – weak damage to an organ or plant; 2 points – moderate damage, no strongly affected organs or plants; 3 points – the average lesion, some organs or plants have a severe lesion; 4 points – severe organ damage and plant perished.

In the process of accounting for diseases, two indicators are established: the distribution or number of affected plants in crops, the intensity or degree of development [8].

The spread of disease (P) is determined by the formula:

$$P = n \times 100/N$$

where: N – the total number of plants in the samples;
n – the number of affected plants.

The weighted average percentage of spread (P_{av}) of the disease is calculated by the formula:

$$P_{av} = \sum SP/S$$

where: $\sum SP$ – the sum of the products of the area of the fields by the corresponding percentage of spread of the disease;
P – the spread of the disease;
S – surveyed area, ha.

Disease development rate (R) in percentage or points is calculated by the formula:

$$R = \sum ab/KN$$

where: $\sum ab$ – the sum of the affected plants (a) by their corresponding score or percentage (b);
N – total number of reference plants in samples;
K – top score.

To obtain more accurate results, special scales are used that characterize the intensity of the development of a particular disease. Below are methods for accounting for the main groups of wheat diseases.

Types of rust

For timely of fungicidal treatment of wheat plantings for prevent significant losses in yields from rust and other diseases with aerogenic infection, it is necessary to constantly monitor their appearance and spread. In areas of growing winter wheat and rye, rust is taken into account in the 2–3 leaves stage appears. In the spring after their growth (April–May), at least 100–200 plants (leaves) are analyzed at 10 plot

of the field and the distribution of rust and the degree of its development are determined. Regular surveys are carried out on the main stages of spring wheat development and the timing of infection of crops, the spread of rust, septoriosis, ect. At signs of disease are founded, after every 25–50 steps, 5–10 samples of 10–15 plants or culms are taken. A detailed analysis is carried out in the laboratory, taking into account the number of diseased plants, the degree of damage to leaves or stems separately for each disease. The intensity or degree of development of rust is determined as a percentage on the scales: brown – for Rusakova, yellow – for Manners, stem – modified by Cobb Peterson. The results are entered into a field journal or computer program [8].

Depending on the association of disease pathogens with certain organs during the ontogenesis of plants, leaves of different layers, internodes of the colums and ears are analyzed. If the counting is carried out at the booting-heading stage, then 2 leaves of the lower and middle layer are analyzed, in the grain maturity stage – 2 upper leaves, including flag leaves. The last account of brown and yellow rust is carried out in the milky-waxy stage of ripeness, culm – in the middle dough stage or fully ripeness of grain [8].

Observation of urediniospores of rust in the surface layer of air, atmosphere and precipitation. To determine the timing of plant infection with rust species, constant monitoring of the content of their spores in the air and rainfall should be carried out. The most common and affordable method for analyzing spore entry by air flow is trapping it with a weather vane or spore traps of the PLS-71 and PLS-71M type. Such a study is carried out during the period of possible development of rust on wheat from the tillering stage until the grain-filling period. The indicators are installed in wheat plantings, preparation glass lubricated with petroleum jelly or castor oil have been changed every 2–3 days. The name or geographical coordinates of the observation point, date and month are recorded on the sticker. The preparation glasses are analyzed in the laboratory at a low magnification of the microscope (8 × 20 or 10 × 20). In the absence of spores or a small number of them, the entire surface of the preparation glass is scanned, in the presence of 2/3 in the presence of 1–2 spores; 1/2 at 3–4 spores, 1/3 at 5–10; 1/5 – more than 10 spores of its surface. The type of rust and the number of spores in 10 fields of view of the microscope are taken, as in determining the grain retention by teliospores of common bunt into account [8].

To obtain data on the presence of rust spores that settle with precipitation, rainwater is analyzed. To do this, use a device consisting of a funnel with a diameter of 15–24 cm with a glass tube 20–30 cm long and 44 mm in diameter. A microfilter is installed in its lower part,

which allows water to pass through but retains spores of fungi. After each rain are analyzation the content on the filter: of spores of rust and other pathogens by centrifugation and microscopy of sediments [8].

The number of urediniospores per 1 m^2 of area is calculated by the formula:

$$N = \frac{n \times 10^4}{S}$$

where: N – the number of spores per 1 m^2, units;
n – the number of spores on the entire surface of the filter, pcs;
S – the area of the upper horizontal part of the funnel, cm^2.

If the growth season, during within 24 hours, two or more rust spores per 1 cm^2 are captured in air and sediment during the day, that conditions are favorable for plant infection, then a massive outbreak of diseases can be expected after 7–10 days. The occurrence of rust epiphytoties is possible if 10–15 spores per 10 cm^2 are detected in the heading stage [8].

From the day of detection of the urediniospore of the fungus in the surface air layer, it is necessary to constantly monitor the relative humidity, average daily temperature and the appearance of the first uredini fungi on the leaves and stems. The presence of dew is important for infection of plant. Wheat infection with brown rust at an average temperature of 10 to 15 °C occurs over a duration of dew of more than 5 hours, and at 16 to 20 °C is reduced to 4 hours [8].

The rate of development of rust pathogens depends on air temperature. The time required for one generation of the fungus can be determined by the formula:

$$n = \frac{C}{T - t}$$

where n – duration of generation, day;
C – sum of effective temperatures for the development of one generation, °C;
T – average daily air temperature, °C;
t – lower temperature threshold for the development of fungi, °C.

The sum of effective temperatures, that is, daily average, exceeding the lower threshold for the development of the causative agent of brown rust, is 85 °C, stem rust – 125, yellow – 171, and lower thresholds – 1.9, – 2 and –0.7 °C, respectively [8]. Using special nomograms

or models, it is possible to predict the intensity of damage to wheat crops by types of rust before milky-wax ripeness of grain and loss of grain yield, as well as to determine the need for chemical protection of crops from the disease and the optimal timing of *disease* control.

Identification of urediniospores of rust pathogens. In stem rust, urediniospores are ellipsoidal in shape and have a clear colored shell; in brown and yellow rust, they are round or round-oval, about the same size. The sporoderm of yellow rust urediniospores is colorless, 1–2 μm thick, covered with very small spines, has 10–12 sprouting pores; in brown rust – 1–2 μm thick, densely covered with small spikes, with 8–10 sprouts in pores.

Leaf spot, powdery mildew

The distribution and degree of development of species of *Septoria*, yellow spotting or pyrenophorosis is taken into account simultaneously with rust. To determine the degree of leaf damage, you can use the unified James scale with indicators of 1, 5, 10, 25, 50 and 100%, the ears wheat – 5, 10, 25 and 50% [8].

In the wheat-growing regions, observations of powdery mildew are carried out in autumn, continue in the spring of next year, observations of spring wheat – from tillering till heading stage of wheat. The degree of damage to the leaves and other organs is determined by the modified Geschel scale [8].

Types of smut, root rot and other diseases

Types of smut are taken into account at the middle dough stage or fully ripe of grain at yield checking. At this, for detailed analysis is control or collects 1000 stems (25–50 from each plot) at equal distances (50–100 m) along of diagonally the field. In the analysis, approbation sheaves take into account all types of smut [8].

Root rot. The accounting for wheat disease is carried out in the 2–3 leaves stage and before harvesting: 10 samples are taken per every 25–50 meters, tearing 10–20 plants with root on each plot. They are analyzed in the laboratory, plant damage is determined in points on a scale: 0 points – there are no signs of the disease; 0, 1 points – single strokes in the coleoptile; 1 point – weak browning of coleoptile; 2 points – moderate browning of coleoptile; 3 points – strong browning, penetrating under the coleoptile; 4 points – the perish off sprout [8].

In the middle dough or fully ripe stage, a second count is carried out, samples are taken in the same manner as in the seadling stage,

or sheafes analysis can be used. In the laboratory, the root system is cleaned of leaves and the degree of development of the disease is determined on a 4–point scale [8]: 0 points – healthy plants; 0–1 to 1/5 of the affected at the basal culm or at its underground part occupy strokes or stripes; 2 points – brown streaks or spots cover from 25 to 50% of the surface of the affected organ; 3 points – strong browning of the first stem and underground internodes; 3–4 points – the absence of productive stems in the presence of symptoms corresponding to a score of 3.

When sampling plants at root rot at 10 points, soil weighing 1–2 kg is taken, its infection with the causative agent of the disease is determined by the method of Ledingham and Chin [8]. The soil is sieved through a sieve with a diameter of 1 mm, weighed 10 g and placed in a test tube of 20 × 200 mm, add 5 ml of mineral oil, 30 ml of tap water. The tube closed with a rubber stopper is placed in a horizontal position on a rotator and shaken with a constant frequency for 10–15 minutes. After settling the tubes for 1–1.5 hours, droplets of an emulsion with a volume of 0.01 ml are viewed on a glass slide under a binocular or a microscope for an increase of × 40–80 in 10–fold repetition. By appropriate conversion, the amount of conidia in 1 g of soil is determined.

Methods of phyto-examination of grain, economic thresholds of its infection

The grain is an infection source and is involved in the transmission of infections of many plant diseases. The main pathogens of wheat, which transmitted through grain, are pathogens of volatile, solid and dwarf smut, septoriosis, helminthosporiosis (*Bipolaris sorokiniana*) and fusariosis (species of the genus *Fusarium*) root rot and bacteriosis. In addition, numerous epiphytic and saprophytic fungi populate the seeds during the grain formation, harvest time, and grain storage, including species of the genera *Alternaria, Cladosporium, Trichothecium, Penicillium, Aspergillus,* and *Mucor*. The causative pathogens intensively develop at high grain moisture (15–16%) and reduce its vitality. The causative agents of the "black point" of wheat are *A. tenuissima* and *A. alternata*. The first species does not significantly affect the sowing quality of the grain and the technological properties of flour in connection with its localization in the fruit coat, and the second one penetrates deeper, affects the embryo and reduces germination [8].

Given the foregoing, it is necessary to determine the contamination of the grain by pathogens. By virtue of the fact that, in the form of impurities to the grain are present: teliospores of smut, conidia of fungi, cells and bacterial spores. The most common methods of inspect of grain

are visual, centrifugation, biological, bacteriological and anatomical analyzes, in some cases, serological and luminescent methods are used [8].

Control analysis of wheat grains for fungal and bacterial infections is carried out by appropriate inspections and laboratories. Visual inspection is carried out simultaneously with the analysis of the purity of the seed material. In this case, are analysis the presence in the grain of impurities sacs of smut and Ergot. The grain are placed on the glass with a thin layer and divided by a ruler into 4 triangles and 100 grains are selected from each and the degree of their contamination with the "black point" is determined on the Toropova scale [8]: 0 points – healthy grain; 0.5 points – traces of color of the embryo the size of a dot; 1 point – darkening of the embryo and adjacent tissues; 2 points – darkening covers up to ½ of the grain surface outside the embryo; 3 points – outside the embryo, darkening covers more than ½ of the grain surface.

To analyze the infection of grain with a complex of pathogens, additional tests are carried out: on contaminated with spore of smut – by centrifugation, Loose smut – using the histological method, infection with septoriosis and helminthosporiosis – biological [8].

Rinse sampling and centrifugation methods are analyze the content in grain of smut spores and fungal conidia of *Helminthosporium*, *Fusarium*, *Alternaria*, *Septoria*. For this purpose, 2 samples of 100 grains are taken from an average sample, they are placed in clean tubes and 10 ml of water is poured and shaken. After that, the water is poured into special tubes and centrifuged for 5 minutes at 1000–1500 rpm. Water is drained from the test tube, the precipitate is shaken, a drop is applied to a glass slide and examined under a microscope in 10 fields of view. The average number of smut spores are finded for each sample by the formula:

$$X = (A \times K) / 100$$

where X – number of spores per 1 grain;
A – average number of spores per ocular view of the microscope;
K – constant coefficient: the product of the number of ocular view of the microscope placed on a cover-slip multiplied by the number of drops in 0.5 ml.

The microscopic ocular view is finded by the formula:

$$S = \pi R^2$$

where π – constant equal to 3,14;
R – microscope ocular view.

The diameter of the ocular view of the microscope is measured using an object micrometer at a certain increase, then the number of divisions in one ocular view is calculated, which is multiplied by the magnitude of the division.

The biological method. 50 grains were taken from the initial grain sample and analyzed in a wet chamber. To do this, put 2–3 layers of filter paper or gauze circles on the sterile bottom of the Petri dish, moisten with sterile water. To detect an internal infection, the grain is pre-disinfected in a 0.5% potassium permanganate solution ($KMnO_4$) for 5 min or in a 0.1% formalin solution, then washed with sterile water. Petri dishes with grain are incubated in an incubator at 20–25 °C for 5–7 days. Then they are analyzed visually or at low magnification power of the microscope [8].

To analyze the infection of wheat grains with *Helminthosporium*, *Alternaria* and *Fusarium* pathogens, 20–25 grains let germinate in 4–5-fold replication at 25 °C temperature in Petri dishes on moistened sand or filter paper. On the seventh day, they are examined visually and at the presence of sporulation of fungi, the morphology of conidiophores and conidia of fungi is studied, their species composition is identified [8].

The causative agents of many fungal and bacterial diseases grow well on artificial nutrient media. Therefore, to determine the infection of grain with fungi or bacteria, to identify pathogens, along with a moist chamber, Chapek's agarized nutrient medium and potato-glucose agar can be used.

The histological method is used to determine the infection of wheat grains with a Loose smut. Grains are boiled in a flask in a 3% solution of KOH or NaOH (based on 100–150 ml per 100–120 grains), on sieves of 5, 3 and 1 mm in size, the embryos are separated from the endosperm and washed thoroughly with tap water. Within 2–4 minutes they are colourated with 1% aniline blue solution prepared in 40–45% acetic acid, then the embryos are washed with lactic acid to remove excess colors. In this solution, they can be left for 2–3 days [9].

The serological method is used to diagnose bacterial diseases of wheat. The drip method developed by Dunin and Popova allows the identification of pathogens of black and basal wheat bacteriosis. For this, a special serum is mixed with a pure culture of bacteria on a specimen glass. A positive reaction is precipitate, visible with a naked eye [8].

Luminescent analysis. Wheat grain is laid out in one layer on black paper, then it is placed at a distance of 15 to 30 cm under a mercury-quartz lamp. The healthy grains produce blue-blue or blue-violet fluorescence, while grains infected with Loose smut do not fluoresce and have a dull appearance. A similar reaction is observed for grain dam-

age by fungi of the genera *Helminthosporium, Fusarium* and pathogenic bacteria [8].

Based on an analysis of the results of many years of experimental research and a synthesis of literature data, the maximum permissible indicators of smut ears wheat smut infection, grain infection and soil contamination by root rot pathogens have been developed (Table 2.1) [8].

If the contamination of the grain is higher than the specified indicators, it is recommended are grain-treatment. Minimum infection indices are used when growing susceptible varieties or an unfavorable phytosanitary situation, maximum ones are used for relatively stable varieties and under favorable weather conditions for plant growth and development [8].

Table 2.1 The critical parameters of infection of grain and soil by fungal causative pathogens

Disease	Monitoring object	Analysis method	Permissible infection
Common bunt	Grain: teliospores	Centrifugation of grain washes and microscopy	100–500 pcs./grain
Loose smut	Grain: mycelium	Histological and microscopic analyzes	0,5–1,0%
Common bunt, dwarf bunt and loose smut	The infection of the ears wheat in the field	Testing of crops, visual analysis	0,1–0,5%
Helminthosporiosis	Grain	Paper rolls	5,0–10,0%
Fusariosis	Grain	Wet chamber	5,0–15,0%
Septoriosis	Grain	Paper rolls	5,0–10,0%
Mildew	Grain	Wet chamber	5,0–10,0%

A modern approach to the determination of wheat pathogens in the European Union is defined in the protocols of the European and Mediterranean Plant Protection Organization (EPPO), combining phenotypic, serological and molecular methods [30]. Modern methods for the determination of wheat fungal diseases include immunological and methods associated with the analysis of DNA of pathogens, the study of genes, are biomarkers of pathogens. If serological methods are mainly used to identify bacteria and viruses, then for microscopic fungi – methods based on DNA analysis (Nucleic acid-based methods). Among them, the most common are: fluorescence in situ hybridization (FISH) and the PCR variants (nested PCR (nPCR), cooperative PCR (Co-PCR), multiplex PCR (M-PCR), real-time PCR (RT-PCR), and DNA fingerprinting.

Chapter 3

Viral diseases of wheat

Viral diseases are often considered insignificant factors when assessing their impact on the environment, but they are an integral part of any ecosystem, and at different stages of development they are in close interaction with its various components. Viruses are currently one of the major reasons for the decline in yield and quality of cereals, which are of great economic importance in Ukraine. Due to the high concentration of cereals, non-observance of scientifically based crop rotation, the infectious pressure (load) of viral pathogens rises. Climate changes that have taken place in recent years, in particular, an increase in the continental climate, contribute to the emergence and spread of new species of harmful insects for agrobiocenoses of Ukraine, which serve as potential vectors of grain viruses [2, 4].

Recently, the problems associated with the development and spread of viral diseases of grain crops are becoming increasingly relevant. In addition to direct economic losses, there is a threat of the importation of new pathogens of viral etiology and their introduction into the agrocenoses of Ukraine. The consequences of this development are difficult to foresee.

Diseases of grain crops are caused by the causative agents of viral etiology described in Ukraine, starting from the 60s of the twentieth century by various authors. Researchers studied mainly the biological properties of pathogens, transmission, distribution, manifestation of the disease in different cultures, harmfulness, etc. [5–8, 13, 73]. Currently, attention is paid to monitoring, molecular biological and phylogenetic studies of cereal viruses [14, 62, 63, 74, 83]. According to the literature it is known that in different regions of Ukraine were distributed: *Wheat streak mosaic virus*, *Tritimovirus*, *Potyviridae* [6, 10, 13, 14, 62, 71], *Barley yellow dwarf virus*, *Luteovirus*, *Luteoviridae* [3, 7, 9, 18, 74], *Barley stripe mosaic virus*, *Hordeivirus*, *Virgaviridae*

[16, 17, 73, 74], *Brome mosaic virus*, *Bromovirus*, *Bromoviridae* [7, 8, 14, 17, 74], as well as a potentially dangerous virus for Ukraine, which is transmitted through the soil with the help of fungal organisms – *Polymyxa graminis – Barley yellow mosaic virus*, *Bymovirus*, *Potyviridae* [11, 15]. Also, there were reported cases of viral diseases transmitted by a leafhopper *Psammotettix alienus*, in particular induced by *Winter wheat (Russian) mosaic virus*, *Cytorhabdovirus*, *Rhabdoviridae* and *Wheat dwarf virus*, *Mastrevirus*, *Geminiviridae*) [1, 7, 8]. The spread of the wheat dwarf virus in the agrobiocenoses of Ukraine and its molecular biological and phylogenetic properties were investigated by the staff of the Department of Virology, Taras Shevchenko National University of Kiev [69, 74].

3.1 Wheat streak mosaic virus

Wheat streak mosaic virus, WSMV, what belongs to the genus *Tritimovirus* genera *Potiviridae*. According to morphology – filamentary fractions with a length of 700 nm, a diameter of 11–15 nm (Fig. 3.1).

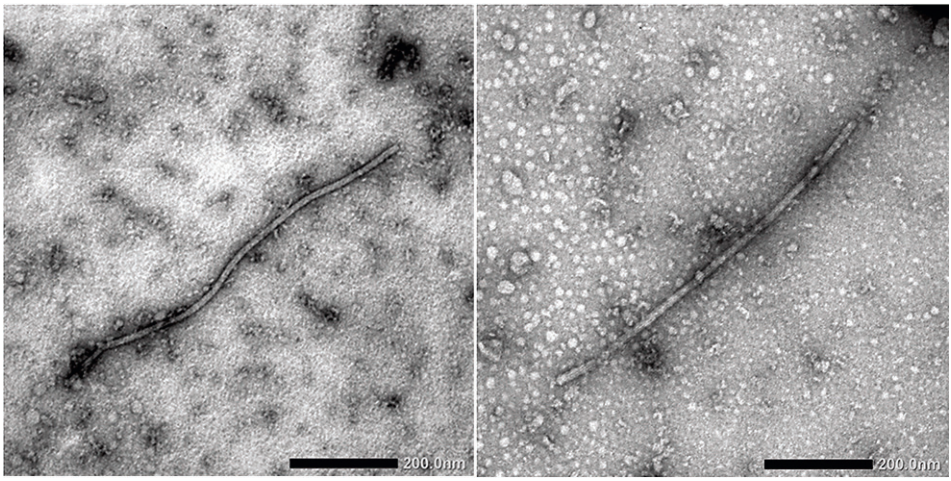

Fig. 3.1 Electron microscopic image of virions WSMV

In a purified preparation, one sedimentation component is detected; sedimentation coefficient – 165 S, the ratio of A260 / 280 – about 1.37. The unipartite (monopartid) genome represented by single-stranded (+) RNA, the total size of which is 9.3–9.4 kb. The genome expands into a single open reading frame, which is transcribed into a large polyprotein.

This polyprotein is cleaved into at least 10 mature proteins: P1 (P1 protein: 40 kDa), HC-Pro (auxiliary component of the protease: 44 kDa),

P3 (P3 protein: 32 kDa), 6K1 and 6K2 (6 kDa), CI (cytoplasmic inclusion protein: 73 kDa), VPg (viral protein of genome-bound proteinase: 23 kDa), NIa (nuclear inclusion of the putative protease: 26 kDa), NIb (nuclear inclusion of the putative polymerase 57 kDa), CP (coat protein 37 kDa) (Appendix 3.1) [79]. Virions contain one capsid protein with a molecular weight of 37,000 Da. The virus is relatively thermostable: inactivated at 54 °C for 10 min; maintains breeding up to 10^{-3}–10^{-4}; the infectivity of the virus in the sap is maintained at 20 °C for more than 4 days, and at +2 °C for more than 1 month [5, 93–95].

Virus replication can occur in the cytoplasm, nuclei, chloroplasts, Golgi apparatus, cell vacuoles, less commonly elsewhere. Viruses form ring-shaped protein inclusions in infected plant cells. These can be crystals both in the cytoplasm and in the nucleus, for example, amorphous X-bodies, membrane bodies, and viroplasmas. Inclusion may or may not contain virions [79].

WSMV is common in all regions of cereal cultivation (North America, Europe, Asia, Australia, Argentina, etc.) (Appendix 3.2). The spread of this virus in southern European countries [22, 25, 56, 84].

The disease was first noticed and described as a "yellow mosaic" in Nebraska (USA) in 1922, after which cases of VSMP began to be reported in different states of the USA, Canada, Mexico, Eastern Europe, West Asia and Australia. Under the new name, "striped mosaic of wheat", the virus was officially registered in Nebraska in 1954. Most viral epidemics in the region were associated with WSMV [37, 43].

The striped mosaic virus of wheat was detected in Europe at the end of the 30s, and its research began in the 60s of the last century. In recent years, the virus has been active in European countries, in particular, Bulgaria, Slovakia, Poland, the Czech Republic, Lithuania, etc. [21, 25, 51, 84, 87]. In Ukraine, WSMV has the greatest epidemiological significance; it was found on winter and spring wheat, winter and violent barley in Vinnitsa, Dnepropetrovsk, Donetsk, Zaporizhzhya, Kiev, Lugansk, Odessa, Poltava, Kharkov and Cherkasy regions, it should be noted that during 2017– 2019, the largest percentage of affected plants in large areas was observed in the eastern regions of the country [5, 6, 14, 74].

In Australia, WSMV was first discovered in 2002 [31]. The virus was identified using reverse transcription PCR using specific primers and genomic sequences. This virus was isolated from various varieties of wheat in Australia in the states of New South Wales, Queensland, South Australia and Victoria [31, 33].

Loss of wheat yield as a result of infection with WSMV is very significant. Kansas crop losses are reported to range from 7 to 13%. A 18%

decrease in yield was recorded during the striped wheat mosaic virus epidemic in Alberta, Canada in 1963. WSMV is reported to be the main factor limiting wheat production in Texas. Even though the average losses in the region were moderate, it was possible to find individual fields that underwent a complete loss of yield due to infection of the striped mosaic virus of wheat. In the United States for 2001, crop losses due to the striped mosaic virus of wheat exceeded 60%. Losses have been recorded for many years around the world. In Australia during 2005 and 2006. WSMV caused the destruction of crops in the amount of 5000 and 20,000 hectares, respectively. However, losses can vary from low to 100%, depending on the time of infection and the infected wheat varieties [34, 37, 92].

As a result of the defeat of WSMV plants through chlorosis and leaf necrosis, the photosynthetic ability of plants decreases, plant growth is delayed, grain weight and seed size are reduced. Grain of infected wheat has a higher protein content, but gives flour with less water absorption compared to healthy wheat [6, 20].

It was found that WSMV caused significant damage to the root system of infected plants; it can be reduced by 50 percent or more. Such a decrease in root biomass results in less water being absorbed and its efficient use. Study Price et al. [66] showed that the root systems of virus-infected plants could not effectively use soil moisture compared to healthy plants. This is especially important in areas with a dry climate, which are highly dependent on irrigation [66]. It was found that overall economic losses were large for irrigation procedures through the additional costs of paying for pumping water. Total losses quadrupled, or 29.3%. Losses are estimated at 464.5 US dollars per hectare for full irrigation [92].

Symptoms of the defeat of the striped wheat mosaic virus on individual leaves appear as small chlorotic lines along the veins of the leaves. As symptoms develop, the chlorotic lines are lengthened to form bursting yellow or pale green stripes, forming a mosaic pattern on the leaves. The leaves are often curled into an elongated tube, and the output of the next leaf is delayed, resulting in "loops". In severe cases, the strips can merge, forming large chlorotic patches, and usually this causes the progression of symptoms to necrosis of leaf tissues and death of plants. Affected leaves turn yellow, die off and lag behind in growth (Appendix 3.3) [6, 74].

The natural hosts and symptoms of viral damage are as follows: on plants *Echinochloa, Panicum, Setaria* spp. – severe mosaic, on *Triticum aestivum* – severe mosaic of leaves, shoot necrosis, growth retardation and a decrease in yield. A soft mosaic forms on infected *Zea mays*

plants, but only some varieties are sensitive. Are also systemically affected: *Agropuron, Avena sativa, Bromus, Digitaria, Eragrostis, Hordeum, Hordeum vulgare, Lolium, Phalaris, Poa compressa, Secale cereale, Setaria, Stipa* [17, 25, 37].

The striped mosaic virus of wheat is easily transmitted by mechanical damage to plants by inoculating the juice of affected plants. The main carrier of infection in nature is the tick *Aceria tosichella* (another name is *Aceria tritici, Acarina: Eriophyidae*) [60]. Eating juice, ticks weaken the plant, make it more susceptible to the virus. Sucking juice from the leaves, they cause twisting of the upper leaf and distortion of the leaf blade, making it difficult to release the colossus. This tick is tiny (0.3 mm), has a white-transparent color and an elongated shape (Appendix 3.4). Ticks are not able to fly, because they are scattered primarily by wind, but insects can help them spread over long distances. The tick is able to parasitize on the body of cereal aphids and spread them during migration. Ticks move in large numbers to the top of plants, accumulate on each other and form chains, ready for the next gust of wind, which leads to their transfer to other plants [60].

Aceria tosichella can receive the virus after eating on an infected leaf for 15 minutes. The minimum period required for transmission of the virus is also about 15 minutes. After molting, ticks do not lose their infectivity and can maintain it (at least in a greenhouse) for 6–9 days after removing them from infected plants.

The life cycle of ticks consists of an egg, two stages of a nymph and an adult. At a temperature of 25 °C, ticks complete their life cycle on average 7 days. Ticks live and feed on the green leaves of their hosts, which is critical for tick survival. High humidity is another key mite survival factor. Without food, ticks can exist for 8:00 at 24 °C and 30-40 hours at 3 °C, which indicates a weak immunity to arid conditions. Adult mites can live only 2–3 days at −15 °C. Eggs are stored longer than zero temperatures; more than 25% of the eggs studied, spent at −15 °C for 8 days, were able to hatch and extend their life cycle upon returning to room temperature. If ticks are kept at 3 °C on plants immune to the virus, they remain infectious for two months. The striped mosaic virus of wheat is transmitted by both nymphs and adult *Aceria tosichella*, but only nymphs become infected when they feed on affected plants. Checking the infectivity of individual individuals showed that about 30% of ticks transmitted the virus after feeding on affected wheat plants [60].

Under real conditions, the virus and its carriers are preserved during the winter period on crops of winter wheat varieties and other types of cereal plants. On the last ticks migrate after harvest and persist un-

til the fall. In spring and summer, ticks multiply actively and transfer the pathogen to all susceptible crops, in particular, to perennial and annual weeds and wild grasses, from which later the virophore ticks spread the infection to new crops of grain crops [60].

By direct inoculation of wheat plants with tick homogenates, as well as by electron microscopic and serological analysis of homogenates, the virus was detected in carriers. Examination of ultra-thin sections showed that the spread of the virus is limited to the intestines; the virus is present in high concentrations in the hind gut and in the back of the insect's midgut.

The carrier can infect plants both during feeding, when the virus comes back through the mouth, and when it is released through the anus, when the anal sucker and the bristles, which are used by the tick to stay on the leaf, can damage the leaf surface. Recently, viral particles have been detected in parenchymal mite cells. This suggests that the virus is able to move from the intestines to the salivary glands. However, it is not possible to determine with a single electron microscope which viral particles play a role in transmission [60].

It is not known why adults do not become infected by eating on affected wheat plants. This limitation can be associated either with some changes in the physical properties of the intestine in an adult tick, or with the appearance of enzymes in it that can destroy the virus [60].

In addition, various authors have shown the ability of the striped mosaic virus of wheat to be transmitted via seeds in different wheat genotypes at low levels from 0.03–0.06% [26, 27] to 0.22% [48, 54]. Phylogenetic analysis of WSMV isolates found in Australia suggested that the virus entered Australia due to the importation of virus-infected selective wheat seeds from the USA [31]. It was shown that seed infection with WSMV may not affect the size of the seeds, that is, there is no direct relationship between the size of the seeds and the transmission of the virus [54].

Given the epidemiological features of the striped mosaic virus of wheat, it is obvious that in addition to the use of transgenic crops, it is desirable to have different types of resistance to WSMV in the same variety, the only effective mechanism to prevent crop losses is phytosanitary measures to prevent the spread of WSMV, in particular, vector control in crops, control in the absence of cereal weeds and scavengers, which can act as virus reserves between the seeding seasons, it is also recommended that estirovanie seeds of wheat resistance to viruses. Since even a low degree of transmission by seeds in combination with transmission of the virus by insects, when the spread of the virus from the primary source of infection occurs through arthropod vectors, can lead to multiple infections and the development of viral epidemics.

3.2 High Plains wheat mosaic virus

High Plains wheat mosaic virus, **HPWMoV**), what belongs to the genus *Emaravirus*, genera *Fimoviridae*. Synonyms: *Wheat mosaic virus*, WMoV, *High Plains virus*, HPV and *Maize red stripe virus*, MRSV/MRStV) [93].

Electron microscopic studies of HPWMoV-infected wheat and maize leaves showed the presence of large double membrane particles, which are probably of viral origin, from 150 to 200 nm in diameter in the cell cytoplasm [19, 57].

The genome consists of at least 8 single stranded RNA molecules, ranging in size from 1.1 to 8 kb. Its schematic image showing the sizes of fragments and products is shown in Fig. 3.2 [36, 52].

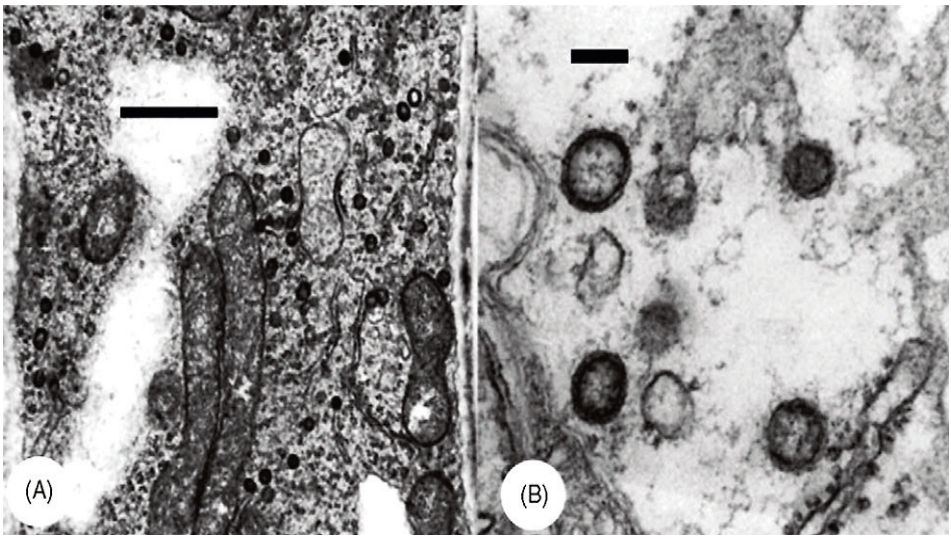

Fig. 3.2 Electron micrographs showing sub-membranous particles in areas of a leaf of corn affected by an HPWMoV (A) part of the cell, with numerous double membrane parts among normal cytoplasmic components (scale = 1 μm); (B) sub-membranous particles (scale = 0.1 μm)

The High Plains Wheat Mosaic Virus (Fig. 3.3) causes a disease that was first detected in 1993 in corn and wheat plants in the High Plains regions (Texas, Kansas, Idaho, Colorado, Nebraska and Utah) in the United States [45]. Now the virus has been identified in Florida and Ohio (USA), Israel, Brazil, Chile, New Zealand, China and Australia [22, 26, 55, 80]. In 2018, HPWMoV was first identified in Ukraine in different varieties of *Triticum aestivum* in the Dnipropetrovsk, Donetsk, Zaporizhzhya and Kharkov regions and in the *Zea mays* of the Gran 6 variety in the Vinnitsa region [75].

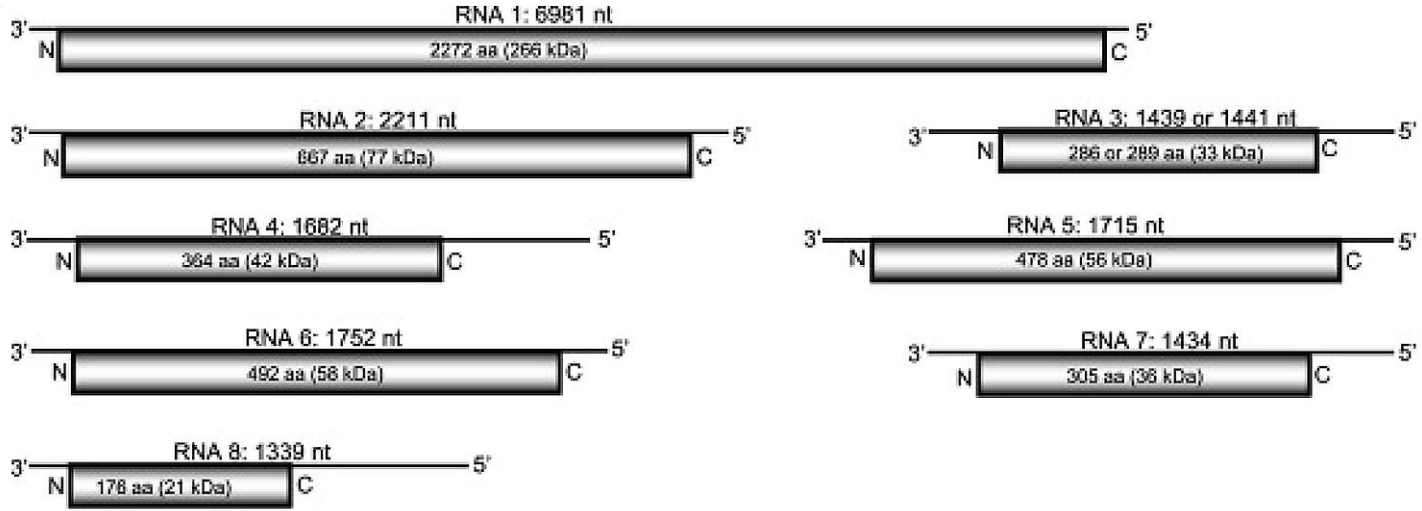

Fig. 3.3 Genetic Organization of HPWMoV

The economic losses associated with HPWMoV are unknown, but the virus has a number of economically important host plants, including wheat and corn. The virus can infect plants in a single infection, also in combination with the striped mosaic virus of wheat. With mixed infections of HPWMoV and WSMV, strong yellowing occurs at an early stage of plant development and plant death is possible.

Plant infection in the late stages of development can lead to stunted growth and reduced plant yields [23, 80]. HPWMoV affects *Triticum aestivum* L., *Hordeum vulgare* L., *Avena sativa* L., *Secale cereale* L., *Zea mays* L., *Bromus secalinus* L., *Setaria glauca* L. *Setaria viridis* [55, 68].

Symptoms of HPWMoV range from spots, chlorosis, mosaic, necrosis and severe growth retardation to rapid plant death, depending on environmental conditions, plant genotype, and time of infection. Symptoms in plants affected by the mosaic virus of the High Plains wheat are similar to those observed with other viral infections. During co-infection, plants often have pronounced symptomatic expression [23, 45, 80].

The High Plains Wheat Mosaic Virus is successfully transmitted by inoculation of infected juice into the vein. In nature, this virus has a common vector with the striped mosaic virus of wheat – the tick Aceria tritici Schev. (Other name: *Aceria tosichella*) [60].

For the mosaic virus of the wheat of the High Plains, seed transmission on corn up to 4% has been proven, but seed transmission of this virus on wheat has not been disproved [22].

Two effective directions are known to limit the spread of this virus and minimize losses from a viral infection: resistance of host plants and phytosanitary measures. At present, no sources of natural resistance to HPWMoV in wheat have been identified; therefore, genetic engineering methods should be used. Phytosanitary methods of combating viruses are to destroy the source of the pathogen, in particular – affected plants, and control the presence of the virus vector.

3.3 Barley stripe mosaic virus

Barley stripe mosaic virus (**BSMV**), what belongs to the genus *Hordeivirus*, genera *Virgaviridae*. Synonyms: false barley streak virus, yellow barley streak virus, mild barley streak virus. According to morphology, the virus is hard sticks 112–150 nm long and 18–24 nm in diameter, channel diameter 3–4 nm (Fig. 3.4). Virions contain 3.8–4% nucleic acid, 96% protein. Virions contain linear (+) – chain RNA. The total genome size is 10.289 kb. The genome is fragmented, consists of three different parts: RNA-a (4 kb), RNA-b (3.289 kb) and RNA-g

(3.164 kb or 2.5 kb) (Appendix 3.5). The main nucleotide composition: 20.3–23.5% G, 27–30.9% A, 19.4–21.5% C, 28–29.4% B. The 5 'end is methylated and is a poly-A tail. The genome has t-RNA-like activity. The envelope protein is one with a molecular weight of 21,500 Da [93–95].

A purified virus preparation consists of three sedimentation components. The main one from S_{20w} is 199 S, also 166 S and 194 S. The isoelectric point is pH 4.5. $A_{260/280}$ – 0.99. TIP – 60–68 °C, the duration of the preservation of infectivity in juice 15–22 days. Virions are found in all parts of the affected plant. The virus does not form inclusions. Peripheral vesicles can form in the chloroplasts of cells [32, 44, 93–95]. To date, several strains of BSMV are known: *Russian, Norwich* (ND), *Canadian severe, Argentine mild* (AM), *Rothamsted* (R) [38].

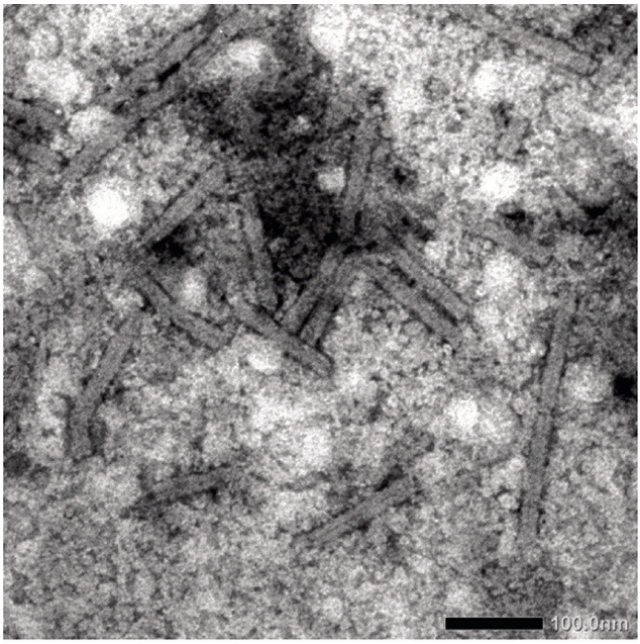

Fig. 3.4 Electron microscopic image of virions BSMV

BSMV disease was first observed in North America 1910 and still remains a potential threat to barley production in the United States. Aggressive BSMV strains seriously reduce yield and affect seed quality [24, 72]. BSMV is found in all regions of the world where barley is grown. In Europe, it is present in Poland, Russia, Romania, Greece, Turkey, Hungary, Moldova, Switzerland, Serbia, Slovakia, Slovenia and Ukraine, mainly with a limited distribution. Bulgaria, the Czech Republic, Denmark and Portugal report single identifications [35, 46, 73].

On natural hosts – common barley (*Hordeum vulgare*), soft wheat (*Triticum aestivum*) virus of striped mosaic of barley causes a weak striped mosaic, leads to necrosis (Appendix 3.6). Genera of susceptible plants to BSMV: *Chenopodiaceae, Poaceae, Solanaceae*. Affected barley plants in the exit phase into the tube have leaves with light green or yellowish stripes that stretch in parallel along the entire leaf, varying in width and often interrupted. During the formation of the colossus, they turn brown. Especially clearly the signs of the disease are noticeable before the formation of the colossus, then the leaves of barley become very yellow [17, 24].

On corn, the symptoms of BSMV appear as pale yellow and whitish solid or intermittent stripes. Sometimes there are so many that the leaves become almost white. With the development of wheat, the signs of disease on young leaves are less clearly visible. Affected sweet corn plants are very stunted. Sometimes the disease goes away without visible symptoms [35, 74].

The experimental circle of sensitive hosts includes individual representatives from 9 genera. Experimentally affected monocotyledonous plants, as a rule, give symptoms of striped mosaic, in dicotyledonous plants – local chlorotic lesions. In indicator plants, the virus causes symptoms of striped mosaic and local chlorotic lesions. So, on *Hordeum vulgare, Triticum aestivum, Avena sativa* – striped mosaic; *Chenopodium album, Chenopodium amaranticolor, Ch. quinoa, Beta vulgaris* – large chlorotic local lesions, *Zea mays* – striped mosaic, *Spinacea oleracea* – mosaic, *Nicotina tabacum* cv. *Samsun* – local chlorotic lesions. The virus can be accumulated on *Hordeum vulgare, Triticum aestivum* [93].

The virus is transmitted by mechanical inoculation, contact, seeds (up to 90%) and pollen. Almost 90% of plants grown from affected seeds can be infected, but do not have pronounced symptoms, which is very dangerous in the breeding process. BSMV is transmitted through an infected embryo; seminal transmission of this virus depends on direct and indirect invasion of the embryo at the same time [5, 33, 61]. The effectiveness of BSMV transmission by seed depends on the strain of the virus, the duration of infection, and the variety and type of crop plants. Even a small percentage of infected seeds can cause multiple infection of crops (from 105 to 107 infected plants per hectare). When polluting healthy plants with pollen from diseased plants, and vice versa, when polluting polluted plants with pollen from healthy plants, there are more diseased plants in crops in the first case than in the second [24, 72]. The conditions for embryo maturation can also affect the reproduction of the virus, decreasing or increasing the level of its seminal transmission [44, 58, 61].

The yield of seeds infected with BSMV is reduced by 20–50% depending on the time of infection, the percentage of affected plants, the pathogenicity of the strain and the variety of culture. With a 30% damage to the seed, grain loss is 30–40%. The decrease in grain mass occurs mainly due to a decrease in the number of productive stems and the mass of seeds in the ear [44]. In the United States, a natural infection of barley results in an average yield loss of 31%. With experimental inoculation, crop shortages can reach 33%. Losses are explained, first of all, by the sterility of colors. There is evidence that even after storage of infected seed for more than 19 years, BSMV remains active [58]. This to some extent complicates the selection of breeding material to create new sustainable varieties of crops. Inactivation of the virus in seeds by etching (formalin, carbon tetrachloride, etc.), cooling or heating (for 30 minutes at 130 °C) is impossible, although the point of temperature inactivation of BSMV is within 70 °C [44, 58, 61].

So, the seed transmission of the striped mosaic virus of barley plays a key role in its spread and survival, and is epidemiologically important, since seed transmission is the main source of these viruses and forms the starting point for initiating the disease in crops. Methods of protection against BSMV: (1) to avoid the virus in seeds (2) legislation (quarantine), (3) quality control of seeds by certification and (4) modern molecular and biochemical approaches to the diagnosis of the virus.

3.4 Brome mosaic virus

Brome mosaic virus (BMV) what belongs to the genus *Bromovirus* genera *Bromoviridae*. Synonym: ryegrass banding virus. Spherical virus particles with a diameter of 25–26 nm (Fig. 3.5) consist of single-stranded RNA (22%) and envelope protein (78%), sedimentation coefficient – 80 S. Isoelectric point of pH 6.8.

The inactivation temperature of various strains in the juice ranges from 67 °C to 95 °C, the final dilution from 10^{-2} to 10^{-4}. The virus remains infectious at room temperature up to 35 days, at +4 °C – up to 90 days. The genome is a single-line RNA. Total genome size: 8.216 kb. The genome consists of three parts: the first – 3.234 kb (RNA-1), the second – 2.865 kb (RNA-2), the third – 2.117 kb (RNA-3) (Fig. 3.6). Non-genomic nucleic acid is found in virions, it is a subgenomic mRNA (for surface proteins), it is called RNA-4 and is found in virions, including RNA-3. Subgenomic mRNA is also found in the affected cells. One viral protein Mr 20300, which acts as a structural protein [93].

Virions are found in the leaves and mesophyll of affected plants, in the cytoplasm, perinuclear space and in chloroplasts. Inclusions were found in the affected cells [81].

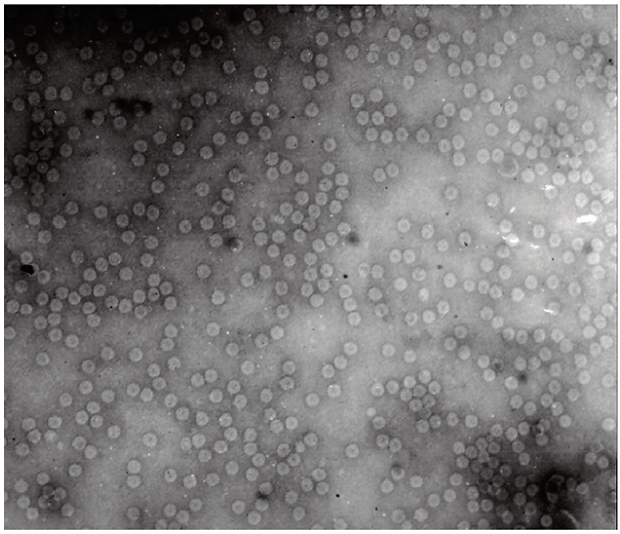

Fig. 3.5 Electron microscopic image of BMV virions

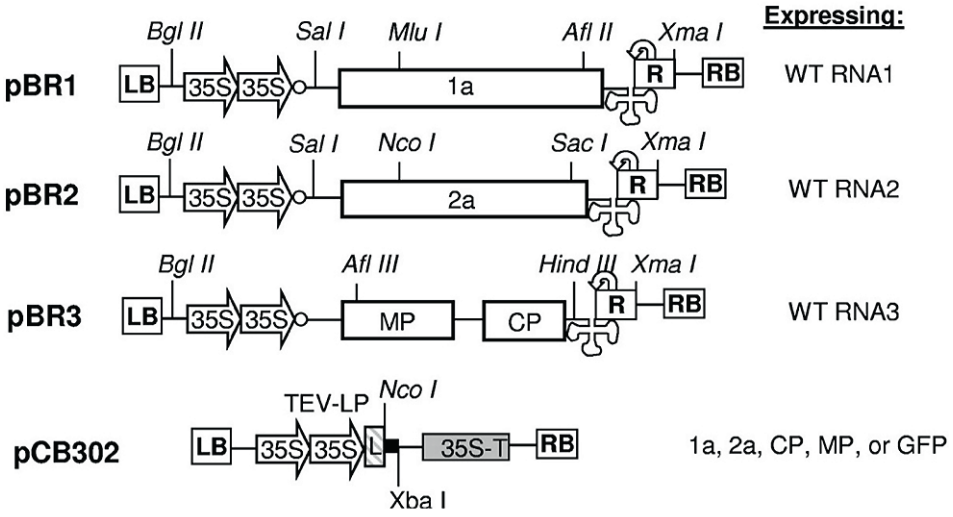

Fig. 3.6 Genomic organization of BMV

BMV occurs on all continents, prevalent in the regions of Eurasia, Australia, South Africa, the USA and Canada. In Europe, it is present in Poland, Lithuania, the Czech Republic, Italy, Estonia, Portugal, Russia, Ukraine, etc. [29, 39, 46, 68, 76, 79, 85, 86–88].

Generas that include virus-sensitive host plants *Poaceae* and *Chenopodiaceae*. This is one of the few cereal viruses that infects cere-

al plants, including beets (*Beta vulgaris*), cucumber (*Cucumis sativa*), common beans (*Phaseolus vulgaris*), cow peas (*Vigna unguiculata*), soybeans (*Glycine max*), etc. [29, 85].

Symptoms of the disease depend on the type of affected plants and growing conditions. In *Bromus* inermis plants, the virus causes mosaic symptoms (Appendix 3.7a). The experimentally infected *Zea mays* plants respond with systemic necrosis and, for the most part, with plant death (Appendix 3.7b). *Chenopodium amaranticolor*, *Ch. hybridum, Ch. quinoa* is a necrotic reaction to a viral infection. On wheat and barley, in 7–12 days after infection with BMV, mosaic, striping, pale green and yellow hatching develops. Later, the leaves become pale yellow. In oats, reddening of leaf blades is often observed [7, 17, 87, 93].

It was shown that barley and wheat infected with BMV are significantly more affected by fungal diseases: *Fusarium culmorum, Bipolaris sorokiniana* and *Septoria tritici* [7, 28].

The grain yield of virus-infected plants is reduced by 35–65% or more. The defeat of spring and winter barley, wheat, oats in the early stages of development can lead to the death of plants. The loss of wheat yield in Ohio during the growing season 2016 and 2017 in virus-infected plants compared with the control ranged from 25% to 61% depending on the variety of wheat and the period of damage to plants and was associated with a decrease in grain size and mass and plant population [39].

The virus is well transmitted by mechanical inoculation, but is not capable of seminal transmission. Testing the possibility of BMV transmission by oats and barley seeds showed a sharp negative effect of viral infection on the sowing quality of seeds, however, BMV did not show in seedlings grown from seeds that were collected from affected plants, although the seedlings were severely suppressed and some of them died. In this case, it is possible that a very peculiar and little studied phenomenon of the transmission of the effects of viral infection to the next generation appears [58, 61].

In laboratory studies, the BMV carrier has nematodes of the genus *Xiphinema*. The pathways of the spread of the virus in nature are not precisely established. There is data on the transfer of BMV by nematodes *Xiphinema coxi*, *X. diversicaudatum* and *Longidorus breviannulatus* and beetles *Diabrotica undecimpunctata, D. virgifera* and *Lema melanopus*, but these data are not always confirmed [78].

In 2019, the first report on the detection of the infectious BMV in the aquatic environment, in irrigation canals and drainage systems of Poland was published [46]. The data obtained indicate the high ability of this virus to survive outside of its host or vector and report on the risks for growing cereals in such territories.

The mechanism for preventing crop losses from the BMV is only phytosanitary measures to prevent the spread.

3.5 Wheat dwarf virus

Wheat dwarf virus (WDV) what belongs to the genus *Mastrevirus* genera *Geminiviridae*. Virions without shell 22 nm in diameter, are paired virions, typical for the genera of geminiviruses (Fig. 3.7).

There may be one or two components in a purified preparation. The first with a sedimentation coefficient – 70S, the other – 50S; A_{260} / A_{280} ratio of 1.3–1.4. Leaf juice contains several virions. Virion contains up to 20% nucleic acid. The genome consists of single-stranded circular DNA, the DNA size is 2600–2800 nt (Fig. 3.8). Replicated in the core. Replication is independent of the helper virus. The genome of the virus contains 4 open reading frames: capsid protein weighing 27 kDa, transport protein weighing 11 kDa and two parts of the protein necessary for replication with a total mass of 41 kDa. Virions are found in the nuclei of leaf and root cells. In infected cells, there are no inclusions.

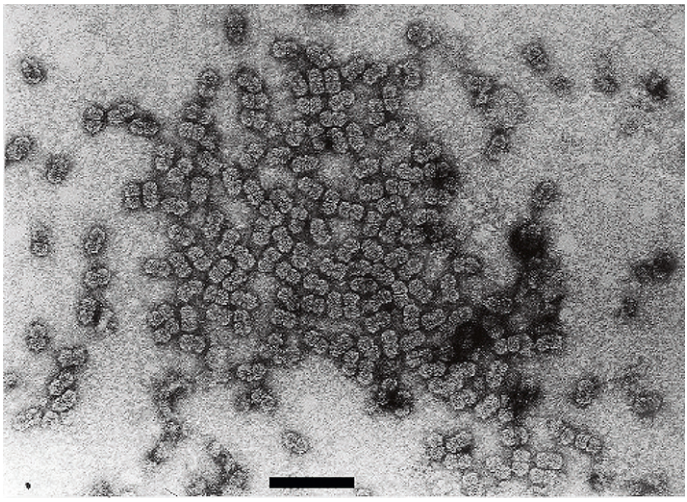

Fig. 3.7 Electron microscopic image of WDV virions

There are two main forms of WDV: a form adapted to wheat (wheat strain WDV) and to barley (barley strain WDV). Both strains infect plants of the *Poaceae* family. However, there is some conflicting evidence as to whether a wheat strain of WDV can infect barley plants and vice versa. According to the results of Kundu [53], the barley strain is limited in its specificity to barley plants, while the wheat strain is able to infect wheat plants and barley plants.

The genomes of barley and wheat strains of WDV are approximately 85% homologous; isolates within the wheat strain show a high degree of kinship (> 98%), and barley strain isolates are somewhat more diverse in genetic aspect (> 94%). According to the conclusion of the International Committee on Taxonomy of Viruses, the criterion for determining the type of mastrevirus is on the verge of 75% homology of the nucleotide sequence, and therefore both of the above strains are now considered as strains of the same species. Schubert et al. in 2007, on the basis of phylogenetic studies, he proposed isolating WDV isolates and strains into three separate types — wheat dwarf virus, barley dwarf virus, and oat dwarf virus [70].

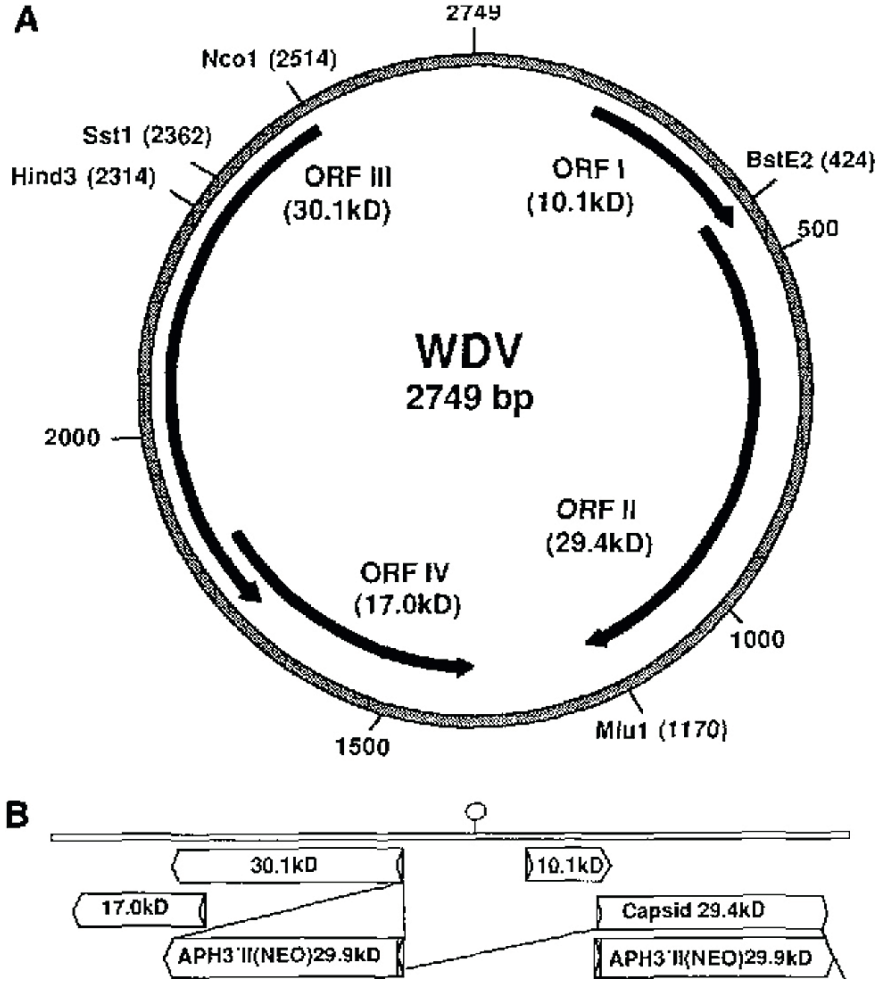

Fig. 3.8 Genomic organization of WDV

So, at least three strains adapted to wheat: *Wheat dwarf virus* (WDV), *Oat dwarf virus* (ODV), *Barley dwarf virus* (BDV), and the first strain has the ability to infect both wheat and barley, are currently identified while the other two are highly specific.

WDV was first found in *Triticum aestivum* in former Czechoslovakia in 1961. The dwarf virus of wheat is spread in various parts of the world, described in Germany, Hungary, France, Finland, Estonia, Sweden, Ukraine, Hungary, the Czech Republic, Bulgaria, Turkey, Tunisia, Zambia and China. [56, 69, 70, 77, 82, 96]. The specific strain detection of Ukrainian WDV isolates in selected samples of cultivated cereals showed that the vast majority of WDV-positive samples contained a wheat strain WDV, however, one of the samples of winter wheat taken in the Odessa region contained a barley strain WDV, which is the latest data in the biology of this pathogen [69, 83].

Vulnerable to the dwarf virus of wheat more than three families. WDV causes characteristic symptoms on plants: *Avena sativa* – yellowing and dwarfism, *Hordeum vulgare* – yellowing, dwarfism and low yields, *Lolium multiflorum, Poa annua* – reduced growth, *Secale cereale*, *Triticum aestivum* – yellowing and strong dwarfism. In indicator plants, this virus causes the following symptoms: *Bromus secalinus* – yellowing, the plant dies without ripening, *Lolium perenne* – asymptomatic, *L. remotum* – yellowing [93–95].

In laboratory conditions on winter wheat, it is shown that the first signs of the disease usually appear 18–25 days after infection. Early crops of winter wheat react symptomatically approximately 4–6 weeks after infection. The incubation period lasts 3–4 weeks. In the case of spring wheat, the first symptoms appear 10–15 days after infection. Infection in the wild is visualized after three weeks [70, 82].

At an early stage of development, affected plants are characterized by bushiness and dwarfism (Appendix 3.8). The degree of damage depends on the age of the plant at the time of infection. A strong development of damage on plants, the moment of infection of which occurred at the stage of appearance of the first leaves. Early infection of winter wheat leads to the most severe damage to plants, mostly die during the winter. Infection in the spring is reflected in the reduction of internodes and part of the colossus [56, 76, 91].

Vacke (1972) reported that during spring wheat infection (cultivar "Zlatka") no serious violations were observed if the infection occurred between the formation of shoots and the formation of a colossus. However, the infection still led to a shortening of the shoots. WDV infection also manifests itself in chlorosis and leaflet striping. These signs appear primarily on young and then on old leaves [90]. Some authors report that various wheat varieties respond to infection with either chlorosis

or leaf striation. The characteristic features of WDV are cracks or deformations of young leaves. Subsequently, the leaves turn yellow from the tip and along the edges. There may be partial redness [56, 77, 96].

The plants that reserve the infection are *Poa annua, Lolium multiflorum, Triticum aestivum, Hordeum vulgare*. It should be noted that *Agrostis stolonifera, Dactylis glomerata, Setaria italica, Lolium multiflorum* and *Lolium perenne* are asymptomatic hosts of WDV and therefore pose a significant threat as potential reservoirs of the virus in nature [69, 83].

WDV is transmitted persistently by the cicadas *Psammotettix alienus* (Appendix 3.9). Not transmitted by the following cicadas: *Javesella pellucid, Laodelphax striatellus, Macrosteles laevis*. The virus persists during molting and does not multiply in the body of the vector. In its reproduction, the cicadas does not transmit its virus to its descendants. It is not tolerated by mechanical inoculation, contact between plants, seeds and pollen [7, 93].

So, the epidemiology of this pathogen is very closely related to the geographical distribution of its unique vector, and this vector, respectively, is more common in countries of southern and eastern Europe due to the relatively warm and mild continental climate. To combat the dwarf virus of wheat, it is necessary to monitor the vector on industrial crops in autumn and spring.

3.6 Barley yellow dwarf virus

Barley yellow dwarf virus (BYDV) is one of the most common and harmful pathogens that affects crops in agrocenoses of Ukraine; for the first time this virus was detected in grain crops in the 60–70s. Twentieth century. According to the 1995 taxonomy, 6 strains of the yellow dwarf barley virus were known; they belonged to two subgroups of the *Luteovirus* genera of the *Luteoviridae* genera (subgroup I: -MAV; -PAV; -SGV and subgroup II: -RGV; -RMV; -RPV) [93]. According to the 2005 classification, these strains began to be considered separate types of viruses that belonged to different genera or without a genus belonging to the *Luteoviridae* genera. Eight types of viruses of the *Luteoviridae* genera were known that caused yellow dwarfism of the genus Luteovirus, which included 3 viruses BYDV-MAV, BYDV-PAS and BYDV-PAV, which had two strains of BYDV-PAV and BYDV-RGV. The genus *Polerovirus*, which included two types of viruses and named them – BYDV, Cereal yellow dwarf virus, (CYDV): CYDV-RPV and CYDV-RPS. Viruses BYDV-GPV, BYDV-RMV, and BYDV-SGV according to taxonomy 2005 belonged to the Luteoviridae family, but did not have a genus [93].

It is known today that genus *Luteovirus* consists of *Barley yellow dwarf virus*-PAV, *Barley yellow dwarf virus*-MAV, *Barley yellow dwarf virus*-PAS, *Barley yellow dwarf virus*-kerII and *Barley yellow dwarf virus*-kerIII. Genus *Polerovirus* includes the following viruses: *Cereal yellow dwarf virus*-RPV and *Cereal yellow dwarf virus*-RPS, and also *Maize yellow dwarf virus*-RMV (earlier known as *Barley yellow dwarf virus*-RMV). *Barley yellow dwarf virus*-GPV and *Barley yellow dwarf virus*-SGV belong to family *Luteoviridae* but not assigned to any genus [40].

These viruses cause the same symptoms on plants, but have different biological and serological properties.

Employees of the Department of Virology at Taras Shevchenko National University of Kyiv in agrocenoses of Ukraine found three species from the group of yellow dwarf viruses: *Barley yellow dwarf virus*-PAV, *Barley yellow dwarf virus*-MAV and *Sereal yellow dwarf virus*-RPV. It should be noted that it was *Barley yellow dwarf virus*-PAV that turned out to be the most common [74].

Barley yellow dwarf virus-PAV (BYDV-PAV) belongs to the genus *Luteoviridae*, the genus *Luteovirus*. The virion is spherical, with a diameter of about 25–30 nm, consisting of 180 coat protein subunits proteins (Fig. 3.9). RNA is linear monopartite (+ – chain), genome 5.3–5.7 kb in size (Appendix 3.10). Virions are isometric, with a diameter of 25–30 nm, there are no lipids and carbohydrates. The molecular weight of the virion is 65,000 Da. The shell protein is one with a molecular weight of 24,400 Da. The floating density in CsCl is 1.40 g/cm^3. TIP – 65–70 °C, withstands freezing in juice and thawing. Optimum conditions for development: t – 12 °C, lighting intensity 32400–43200 lux. Resistant to chloroform and butanol. BYDV – PAV is a strong immunogen [93–95].

Barley yellow dwarf virus-PAV is one of the most common and economically significant pathogen; it has been identified in countries where 95% of world grain production is concentrated. The annual yield loss of susceptible varieties reaches 10–15%, and in the years of epiphytoties – 60–90%. According to experts, only in the United States with a 1% level of damage to crops of grain and corn, annual losses from BYDV reach $ 250 million [7, 8, 64, 65, 86].

BYDV-PAV infects more than 150 species of plants from the Poaceae family, and can reduce the yield of subsequent crops: *Triticum aestivum* L., (*Hordeum vulgare* L., *Avena sativa* L., *Secale cereale* L., *Zea mays* L. and *Oryza sativa* L. [7, 86].

Affected plants have yellowing symptoms and reddening of leaf blades, sometimes even in the fall, in spring symptoms appear beginning in April and such plants have growth retardation (Appendix 3.11). Due

to viral damage, the flatness of the grain and the hollow-grain are formed. In plants affected by the virus, a decrease in frost resistance and an increase in susceptibility to septoria, fusarium and other diseases are observed, as well as an increase in the population of cereal aphids [3, 18, 74].

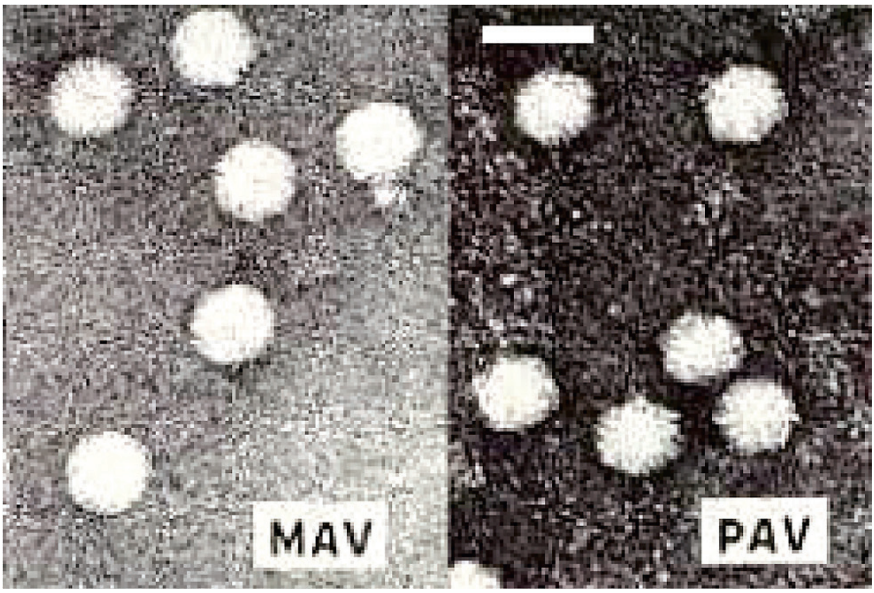

Fig. 3.9 Electron microscopic image of BYDV-PAV virions

The virus is transmitted only by aphids, a persistent method, circulates, but does not multiply in the body of the insect. It is localized in the phloem. The virus enters the insect along with the juice from the cells of the phloem. After passing through the posterior intestine, the virus enters the hemocycle, and after circulation in the hemolymph is concentrated in the salivary glands. After feeding virophoric insects on a plant, the virus with saliva enters the phloem [9, 41].

It is important that certain types of aphids can transmit only certain viruses (Table 3.1).

Interestingly, some species of aphids can transmit two or three viruses, for example, *Rhopalosiphum padi* has the ability to transmit BYDV-PAV, CYDV-RPV and CYDV-RPS; *Sitobion avenae* – BYDV-PAV and BYDV-MAV, and some only one virus. It should be noted that, according to various authors, it is *Rhopalosiphum padi* and *Sitobion avenae* that are common in Ukrainian agrobiocenoses [9, 18, 74] and both can transmit BYDV-PAV, which has the greatest epidemiological significance in our country (Appendix 3.12).

Table 3.1 Yellow dwarf viruses and their vectors

Virus	Vector
BYDV-PAV	*Rhopalosiphum padi*
	Sitobion avenae
BYDV-MAV	*Sitobion avenae*
BYDV-PAS	*Rhopalosiphum maidis*
	Metopolophium dirhodum
CYDV-RPV	*Rhopalosiphum padi*
CYDV-RPS	*Rhopalosiphum padi*
MYDV-RMV	*Rhopalosiphum maidis*
BYDV-SGV	*Schizaphis graminum*
BYDV-GPV	*Myzus persicae*

Due to the complex relationships between host plants, viruses and their vectors, and especially the strong influence of weather conditions on the activity and reproduction of the latter, the spread of these viral diseases and losses, they cause a significant fluctuation. This makes it difficult to predict the development of viral diseases and makes it difficult to make the right decision for direct control of vectors. Although insecticides are used for chemical control and insecticides for the treatment of crops, such measures are not economically feasible every year. The group of yellow dwarf viruses is transmitted in a persistent way, that is, for their absorption from the phloem, a long period of nutrition and, in addition, a long latent period of the presence of a vector (virus transmission takes several hours) are required, then chemical plant protection products most often have a good effect. Aphids die before transmission of the virus. But for effective control of insects, it is important to correctly determine the processing time. The primary infection of crops with arriving vectors, of which the virus-infected part is insignificant (usually does not exceed 4%), does not stop insecticidal treatment, however, the spread of viruses in crops can be prevented [4, 59].

3.7 Methods for laboratory diagnostics of viral diseases of cereal crops

Viruses are obligate intercellular parasites which are able to replicate exclusively inside the living cells by using cellular enzyme machinery and switching the cell for producing progeny mature virus particles

(virions). Taking into account the specifics of virus structure, size, and replication, their laboratory diagnostics requires combination of direct and indirect methods including biological, electron microscopy, serological and molecular/genetic approaches.

Biological technique, or biotesting, assumes using of susceptible bioindicators – species or groups of species of living organisms. In plant virology, these are indicator plants specifically reacting on virus challenge with visual symptoms.

Virus infection of such plants can be either systemic or local. Local symptoms develop on the infected organ and can take in the form of chlorotic lesions, necrotic lesions or ringspots. Systemic symptoms of viral disease develop on different (including distal) parts of the plant and include stunting (or dwarfing), varying sorts of leaf mosaic, discoloration of leaves and/or fruits, deformations, etc. Comparing to other organs, shoots and roots are rarely damaged by viruses [12, 49].

Selection of a panel of indicator plants depends on the virus and the task at hand. Some viruses have a wide range of indicator plants, when the others don't.

Using biotesting, it is possible:

– to establish the infectious nature of a pathogen;
– to establish the species of a pathogen;
– to determine its host range;
– to separate a virus from the mix (in case of mixed infections);
– to determine relative virus content in plants;
– to accumulate virus prep for further research.

Expectedly, most of the indicator plants used for cereal viruses belong to *Poaceae* genera. Rare exclusions are *Barley stripe mosaic virus* which is also able to infect *Chenopodiaceae* and *Solanaceae* plants, and *Brome mosaic virus* infecting *Chenopodiaceae* and also some other dicot plants including beet (*Beta vulgaris*), cucumber (*Cucumis sativa*), bean (*Phaseolus vulgaris*), cowpea (*Vigna unguiculata*), soybean (*Glycine max*), etc. [7, 12, 17, 49, 85].

Virus detection using **electron microscopy** (EM) is based on direct identification of virions by their typical morphology. As the viruses are submicroscopic, light microscopes cannot be used.

Main benefits of EM are the possibility to visualize viruses and identify them directly without preliminary research, and the speed of such diagnostics – often, the preparation can be studied minutes after made ready.

The disadvantages include the requirement of rather high virus content in the material (approx. 10^6 particles/ml), inability to study many samples in parallel, and costly equipment requiring skilled staff.

Types of electron microscopy often used for virus diagnostics include:

– *transmission electron microscopy*, based on electron scattering without energy changes when passing through a dense substance or material. Transmission electron microscopes are used for detailed study of microstructure of objects which are beyond the resolution of the light microscopy (smaller than 0.1 mcm). The objects can be analyzed as thin films or suspensions. The resolution of the typical transmission electron microscope is about 10^{-8}.

– *immunosorbent electron microscopy*, direct visualization of antigen (virus) – antibody complexes using electron microscopy.

As most of other biological objects, virus particles are electron/optically clear objects. The contrast of the electron microscope image can be increased by decreasing accelerating voltage, decreasing aperture diaphragm or increasing focal distance of the objective lenses. The most efficient approach presumes chemical contrasting – artificial increase of the electron density of the ultrastructures. By using electron-dense substances, we can increase the electron density of the virus structures as compared to their background – this is called *positive* contrasting. In positive contrasting, chemical substances bind to virus particles. Alternatively, we can increase the electron density of the background itself – this is called *negative* contrasting (in this case, chemical substances are added to the media surrounding virus particles) [50, 67].

Serological tests are rapid and useful methods for detection of plant viruses. Their main benefits include high specificity of the reaction allowing to detect viruses in cell matter and other material; small quantity of virus material required for the analysis; fast results; ability to measure virus content in the material on a wide scale; especially useful when working with viruses without known plant hosts or which are not sap-transmissible; long period of storage of antisera allowing periodic comparative testing.

In enzyme-linked immunosorbent assays (ELISAs), antigens interact with the antibodies when one of these reagents is coupled with the enzyme. The formation of antigen- antibody complex induces enzymatic reaction which is visualized by adding a specific substrate (chromogen) developing color reaction.

In plant virology, ELISA became a gold standard for virus diagnostics with many variations developed. For ELISA tests, a wide range of virus-containing material can be used: fresh plant sap, purified virus preparation, seed homogenate, soil or insect extract, etc.

Another useful advantage of ELISA is its high sensitivity which is achieved by using enzyme conjugate, increasing an incubation stage (especially for enzymatic reaction), and using a series of steps including

antigen binding by specific antibodies immobilized on a carrier (ELISA plate) with subsequent interaction with another set of specific antibodies conjugated with the enzyme.

Sap of healthy plants is typically used as an external negative control for ELISA. Also, there some internal negative controls including that for non-specific conjugate binding in wells without respective antigen [30, 42, 97].

Polymerase chain reaction (PCR) – is a widely used technique for specific increasing of the number of needed DNA fragments in a biological sample (selective enrichment) by using oligoprobes (short nucleotide sequences specific for a given virus gene), DNA polymerase, nucleotide mix, reaction buffer and thermal cycler – all mimicking real intracellular machinery of DNA synthesis *in vitro*.

Most of the plant viruses are RNA viruses and hence PCR requires an additional step called reverse transcription: prior synthesis of the first DNA strand using initial virus RNA template. For this first step an enzyme called reverse transcriptase (RT) is used, and such modification of PCR is then called RT-PCR.

Apart from simple quantitative increase of DNA copies (a process called *amplification*), PCR also allows many other manipulations with DNA (including mutation introduction, DNA ligation, etc.) and now is widely used in biology and medicine. PCR is a molecular diagnostics technique which can be used for detection of DNA originating from virtually any organism, and became an indispensable tool for every lab.

Quantitative modification of (RT-)PCR is a useful tool for determining virus content, studying expression of viral genes, virus-host and virus-vector interactions, etc. [89].

For detection and analysis of PCR products, several techniques are used including gel electrophoresis, dot blot hybridization and Southern blot hybridization. Today, gel electrophoresis in agarose gel became a typical way of rapid visualizing PCR products.

Development of PCR greatly simplified life of molecular biologists and virologists as it allows amplification of indefinite number of specific DNA. Today, any research of molecular biology of genes relies on PCR including viruses integrated into host genome, viroids, etc.

Chapter 4

Phytoplasma diseases of wheat

It is known that phytoplasmosis cause considerable damage to crop production. Yield losses from phytoplasmosis diseases under favorable conditions for pathogens reach more than 25%. Today, more than 600 plant diseases are known for which phytoplasmosis nature has been proven [1–3].

Given the significant spread of phytoplasmosis last years and their harmfulness to crops and, in particular, to cereals, the question arises of how to prevent yield losses from these pathogens [4, 5, 14, 15]. But the solution to this issue is significantly complicated by the fact that recently in Ukraine there has been a change in climatic conditions inherent in certain areas of crop production, which is considered a consequence of global warming. In addition, there is a revival of economic relations with Western countries, and at the same time, for objective reasons (privatization of land resources, restructuring of agricultural enterprises), they often do not comply with the technological conditions for growing crops. Under the pressure of different factors everywhere, there is a decrease in the resistance of cultivated plants to pathogens of already known diseases of agricultural crops, as well as the danger of the spread of microorganisms to the territory of Ukraine that were not previously registered in this region.

Mollicutes (phytoplasmas, mycoplasmas) are prokaryotic microorganisms with the least known genome at present, which are capable of independent reproduction, devoid of the cell wall and do not synthesize its biochemical precursors. Representatives of the *Mollicutes* class are ubiquitous organisms that, as optional parasites, exist inside various macroorganisms – animals, insects, humans, plants, and the like. However, their ability to saprotrophic existence on the surface tissues of plants was also established. More than 600 plant diseases from 96 families are known to be caused with the *Mollicutes*. In addition to wheat, pathogens of phytoplasmosis affect rye, barley, oats, millet, grasses.

For plant phytoplasmas, which are factors of catastrophic epiphytoties, specific virulence factors have not yet been found. The symptomatology of the disease depends on the particular plant, and phytopathogenesis is largely determined by the reactions of phytoimmunity. In general, the solution to the problem of suppressing phytoplasma diseases is possible only if a thorough study of the mechanisms of development of phytoplasmosis, the characteristics of the interaction of signal and metabolic chains in the host parasite system, as well as by combating the spread of this disease.

Phytoplasmosis of cereals is widespread not only in Western Europe, but also in the former CIS: Ukraine, Moldova, Georgia and the Krasnodar Territory. However, their pathogens are still not well understood, since their symptoms are similar to viral diseases, and diagnostic methods are complex and imperfect. Insufficient attention paid to the study of the etiology of phytoplasma diseases is the main reason for the growth of their harmfulness.

By its harmfulness, as early as the 70s of the 20th century, the defeat of the wheat of phytoplasmas was qualified as "catastrophic". So, back in 1961, the dwarfism of wheat of phytoplasmal etiology caused severe damage to the crops of ears of bread in the Krasnodar Territory, as a result of which more than 50 thousand hectares of winter bread were destroyed and plowed. During 1970–1971 in Ukraine (Odesa, Kherson, Mykolaiv and other regions), Moldova and Kuban Territory phytoplasmas were affected more than 450 thousand hectares of wheat crops. At the same time, plant damage in crops ranged from 15 to 60%, and grain yield from affected plants decreased by 80–90% compared to "healthy" ones. Almost 95–98% of the zoned and those tested wheat varieties were affected by phytoplasmas, and the lack of data on their pathogenesis inhibits a certain degree of work to develop more tolerant varieties. Therefore, one of the main tasks is the use of timely and correct diagnosis of these diseases, based on knowledge of the morphological, physiological, biochemical and serological properties of these pathogens.

Localization and pathogenicity of phytoplasmas, their factors and signs of plant damage. With the defeat cereal with phytoplasma jaundice, the manifestation of signs begins in them with enlightenment of the vessels of the leaves and the subsequent loss of green color of the entire leaf. Then the leaves turn yellow and acquire a chlorotic appearance. The appearance of such signs is associated with the fact that jaundice, like other phytoplasmosis, are systemic diseases.

Of course, phytopathogenic plant mycoplasmas are found in the sieve elements of the affected plants. Sometimes they are also found in pa-

renchyma cells, in tissue cells bordering the phloem, and in the core parenchymal tissues. They were rarely observed in xylem and mesophyll cells or in the root region. As a rule, phytoplasmas populate the cytoplasm of mature cells, however, there are reports of their presence in plant cells at an early stage of development, in meristem cells and in calluses. According to electron microscopy, the number of phytoplasma cells in a plant cell in one plane of its ultrathin section can reach 100 units, and in such cases the cell is almost completely blocked with phytoplasmas.

Naturally, a significant population of cells of various plant tissues with phytoplasmas causes a significant violation of normal processes not only in individual cells, but also in whole tissue systems. Even if plant cells are not populated with phytoplasmas, but are located close or in a systemic connection with them, then they lose their normal structure and function under the influence of various pathogenicity factors of the microorganism. Factors of pathogenicity of phytoplasmas are both their various metabolites, and their competition with host cells for certain substrates.

Localization of phytoplasmas in phloem cells affects the normal functioning of the whole plant. The physiological and biochemical activity of this microorganism in the plant's conducting system causes excessive formation and degeneration of phloem cells. As a result, the affected plants lag behind in growth, acquire dwarfism, turn yellow and fade. However, sometimes the opposite effect of phytoplasmas on plants is observed – prolongation of the vegetation period of the affected plants, in them there is a delay in the onset of reproduction and the final phase of development. This indicates that plant damage with phytoplasmas changes the normal regulatory processes in them, which also leads to a change in the whole habit of the plant: an excess of the formation of accessory buds and shoots, the dominance of the apex is disturbed. Through the quantitative formation of lateral shoots, the plant becomes a "witch's broom", looks dwarf, in such plants the internodes are shortened, the size of the leaves and the growth rate decrease. Violation of the balance of regulatory processes in a plant leads to the appearance of Philodem in plants, distorted development of generative organs and their infertility. The fruiting of the plant after the defeat of its phytoplasmas does not occur, since the generative organs turn into vegetative ones.

Phytoplasmas, the main localization of which is the phloem, are capable of disrupting normal translocation processes in affected plants and, thus, negatively affect not only the affected vascular system, but also those plant tissues that are not directly related to this system. As

a result, sometimes with phytoplasmosis tissue necrosis is observed, in most cases it is the result of hypertrophy and hyperplasia of the phloem tissue, which manifests itself in the formation of a large number of new sieve elements. Excessive starch often accumulates in the leaves of affected plants, together with growth inhibition, indicating a restriction of the function of the phloem.

Plant diseases such as jaundice caused by phytoplasmas can have various symptoms. This, in fact, is yellowing – their usual signs are an elongation of the internodes and yellowing of the leaves. In addition, there is a "witch's broom" – when there is an excessive development of additional shoots and the underdevelopment of the top of the plants. For a number of plants, characteristic features of the trunk are observed – in this case, the affected plants are characterized by underdevelopment of the apex, dwarfism, twisting of leaves, curvature of the lower part of the vegetative organs during growth, as well as greening of flowers and wilting. However, according to observations, plants affected with phytoplasmas often have a whole complex of the listed symptoms of the disease. The appearance or variety of the plants, the stage of their development during the lesion, the environmental conditions of cultivation, the time of the lesion, the type of carrier and other factors, both natural and anthropogenic, affect the manifestation of a particular type of lesion.

Among phytoplasma wheat diseases since the 60s, according to the group of signs of plant damage, dwarfism and pale green dwarfism were distinguished.

The greatest harm to crops is caused by pale green dwarfism of wheat (PGD), the causative agent of which is the representative of the *Mollicutes* class of the *Acholeplasmataceae* genus – *Acholeplasma laidlawii* var. *granulum*. PGD is one of the most harmful diseases of crops, which leads to 60–80% of crop losses. So, the dwarfism of wheat in 1961 acquired an epiphytotic character in the North Caucasus, where it caused a severe defeat of winter bread. In recent decades, this disease is also quite common in Russia - on average, the Volga region, the prevalence rate was 10–20%. At the same time, the productivity of the affected plants decreased by 92%.

Symptoms of PGD. On the territory of Ukraine, pale green dwarfism of wheat (PGD) was described in the 60s by Agarkov as a disease with characteristic signs of leaf color, characterized by the absence of classical symptoms of viral diseases. Along with these signs, mosaic leaves can also be observed. Affected plants are characterized by dwarf growth and proliferation of flowers; specific lumpy crystals are not observed during acidification of the juice. In addition, the flowers become

sterile, with proliferated films on the ears and subordinate stems, often underdeveloped spikelets with shortened scales, empty stalks (Appendix 4.1).

Characteristic signs of dwarfism is a significant lag in plant growth and the formation of dense rosettes. In most cases, the plants do not swear or form an unfilled spike and flat grain. Plants with signs of pale green dwarfism are especially noticeable in the booting stage of wheat and in the earing phase. So, for wheat plants affected by PGD, in addition to the main feature – pale green leaf color, dwarf growth and the outgrowth of sterile flowers remained characteristic.

Symptoms of wheat PGD are noted in the complete tillering phase of late autumn and early spring. At the same time, the affected plants lag behind in growth, bushes with numerous small shoots appear, that is, "rosettes" are formed. During the booting stage of wheat stem before earing, the "rosettes" die off, dwarf wheat plants with pale green leaves are lagging in growth, the number of shoots in the bushes is increased, the main shoots are often not distinguished. In the earing and grain formation phase, spikelets with growth of flower films are observed; the spike-like internodes usually do not exceed the length of the sheath of the upper leaf, which prematurely perished off (Table 4.1).

Table 4.1 Symptoms of PGD of wheat

Phytoplasma disease of wheat	Typical symptoms of the disease
Pale green dwarfism of wheat(PGD)	• Pale green color of leaves • Dwarfism • The formation of 'sockets" • Stemming of sterile flowers • Stemming of flower films

The disease is too harmful, the grain yield in the affected plants is reduced by 80–90% compared with healthy ones. The degree of damage to wheat crops significantly depends on the method of sowing, the resistance of varieties, the distribution and number of carrier cicadas, their natural infectivity (Appendix 4.1).

Employees of the D.K. Zabolotny Institute of Microbiology and Virology of the NASU found that one of the causative agents of pale green dwarf wheat is a representative of the class *Mollicutes* of the *Acholeplasmataceae* genus – the strain *Acholeplasma laidlawii* var. *granulum* 118, which, along with other phytoplasmas, isolated in a pure culture, is maintained on an artificial medium SM IMV-72 and stored in the Ukrainian collection of microorganisms (UCM) of the D.K. Zabolotny Institute of Microbiology and Virology of the NASU. It was found that

in plants affected by *A. laidlawii* var. *granulum*, the development of infection is associated with the colonization of tissues with phytoplasmal mini-bodies – ultramicroforms, the sizes of which (less than 0.2 microns).

Unlike unaffected plants, phloem cells contain bodies that in their structure and morphology do not differ from mycoplasmas isolated from humans and animals. Their number ranges from a few to the complete filling of the element, and sometimes their localization is limited to the plasmalemma of the host plant cell. In the cytoplasm of a plant cell, phytoplasmas are located along their cytoplasmic membranes. By the way, the number of phytoplasma cells is responsible for the manifestation of such a symptom of the disease as leaf chlorosis: in the case of dense filling of plant cells with pathogen cells, clogging of plant vessels occurs and more pronounced chlorosis is observed [12].

Phytoplasma cells vary significantly both in shape and size: their diameter was observed from 100–200 nm to 600–800 nm. Along with round or oval are elongated forms, but in smaller quantities. Cells of 600–800 nm in size comprise the bulk of the phytoplasmas in the cell of the host plant, regardless of the type of plant and its disease. It should be noted that it is precisely between phytoplasmas of this size that the formation of elongated "outgrowths" is observed. No inclusions similar to phytoplasmas cells were found in the cells of control sterile plants.

Significant differences in the morphology and structure of the cells of the pathogens of phytoplasmosis depending on the living conditions – different types of plants affected with different types of jaundice were not found. Both in the tissues of the host plant and in acellular nutrient media, the development of phytoplasma cells can be divided into the following phases: 1) young cells with high physiological and reproductive activity; 2) mature cells with moderate physiological activity; 3) cells grow old, in which metabolic processes freeze [12].

Young cells of all pathogens of jaundice have a clearly defined membrane with a thickness of 12–14 nm, their cytoplasm is full of ribosomes with a diameter of about 17 nm, no genetic material is observed in such cells (Fig. 4.1).

These cells are similar to those grown on an artificial nutrient medium, however, cultured cells differ in size – a larger number of forms with a diameter of 150–200 nm, especially in cultures whose age exceeds 8–10 days. They are considered mini-bodies, or nanoforms, which are formed as a result of pressure from adverse environmental conditions [13].

In this case, the destruction of chlorophyll occurs, accompanied by nonspecific changes in a number of physiological and biochemical char-

acteristics. In addition, under adverse conditions, such nanoforms can be stored for a long time, after which they are restored to a full-fledged culture of active phytoplasmas.

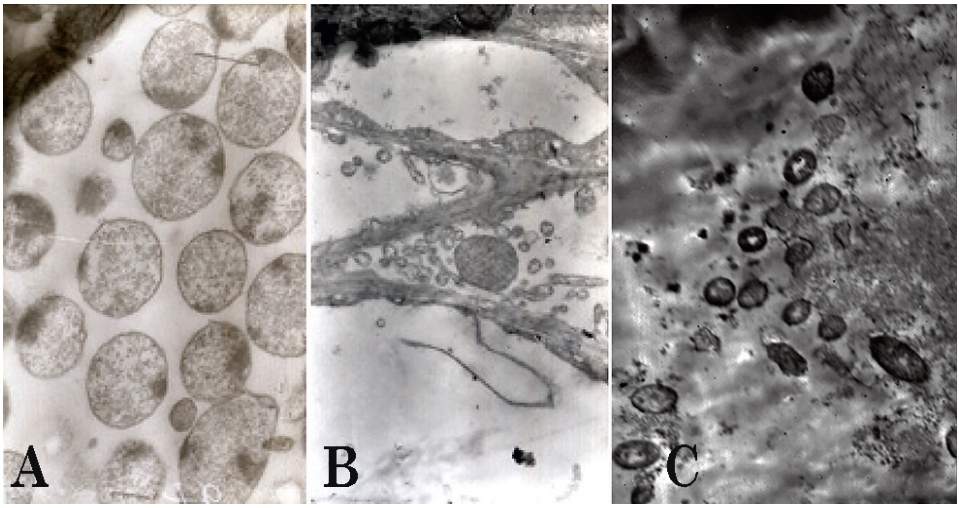

Fig. 4.1 Electron microscopy A. *laidlawii* var. *granulum* 118 – PGD pathogen of wheat: A – pure mollicutes culture; B – in a plant with signs of phytoplasmosis; C – in sugar beet calluse culture

There is evidence that, as a result of experimental infection, acholeplasma penetrate into the tissues of plants directly through the intact root system and cause morphoses characteristic of phytoplasmosis, which occur spontaneously *in vivo*. At the same time, on the sixth day after phytoplasma infection of plants, significant part of the infectious agents was represented with mini bodies, the formation of which is associated with a disruption in the entry of nucleic acid precursors into the cells of microorganisms that are unable to synthesize these substances *de novo*. Colonization of plant cells with mini bodies induces necrosis of mesophyll cells, apoptosis of phloem cells, destruction of chlorophyll and is accompanied by nonspecific changes in a number of physiological and biochemical parameters. The responses of plant to phytoplasma infections are associated with the inclusion of classical signaling mechanisms for the suppression of pathogenic microorganisms.

Carriers of phytoplasmas and causative agents of PGD of wheat – in particular, are insects. Thus, the transfer of phytoplasmosis to the cicadas of the species *Aphrodes bicinctus Schrank, Euscelis plebejus Fall*, and *Phylaenus spumarius* L. was experimentally confirmed. It was also proved that these species are carriers of phytoplasmas. Phytoplasma carriers are also insects from the *Cicadellidae* genus – the

cicade *Psammotettix striatus* L., inside which the pathogen passes an incubation period of 12 to 30 days. It was found that insects become infected at all stages of development – nymphs of all ages and adults – all the same, both males and females. At the same time, with age, the susceptibility of cicadas to the pathogen decreases (from 48.1% for the nymph and to 29.3% for the adult). *In vivo*, the infectivity of the plants with circadas is 1–2%.

An important and necessary prerequisite for preventing the spread of phytoplasma infections and further crop losses is the timely diagnosis and identification of pathogens of phytoplasma diseases [6–9].

When diagnosing wheat phytoplasmosis, it should be borne in mind that the symptoms of dwarfism of wheat are similar to the viral diseases of this culture. But phytoplasma disease of wheat with symptoms of pale green dwarfism, which belongs to the group of jaundice, which, in contrast to diseases caused by viruses of the mosaic group, has the inability to transmit the infection by inoculation with juice. Therefore, when conducting surveys of crops and selection of plants with symptoms which characteristic of this disease, this information should be taken into account. It is possible to quickly distinguish this disease by acidifying the juice of the affected plant with 0.1 N HCl – at phytoplasmosis crystals does not formed, unlike the juice of plants affected with viral mosaics. In addition, unlike viruses, in the case of dwarfism, the pathogen is not transvarially transmitted – adults, and not only nymphs, are able to carry it. When conducting a plant inspection, it is also important to pay attention to general plant chlorosis, since with phytoplasmosis of wheat, mosaic symptoms are usually absent and general plant chlorosis is observed. The phytoplasma mosaic on the leaves can appear only at the initial stage of the disease, after which it disappears and the leaves of plant infected with phytoplasmas become chlorotic in color. The first sign of phytoplasma illness of wheat plants affected by PGD is precisely the enlightenment of the vessels of the infected leaf, which gradually loses its green color. Subsequently, as the infection spreads, streaks become colorless also on other leaves. They turn yellow and become chlorotic.In the leaves of wheat plants affected by phytoplasmosis, an excess of starch often accumulates, that together with symptoms, a decrease in growth indicates a violation of the leading function of the phloem.

Phytoplasmosis can also be identified with insects, since the pathogens of phytoplasmosis are also transmitted using insects. The objects are uninfected, healthy plants on which infected with phytoplasma cicada feed. These are either healthy plants of the same species as the affected ones, or indicator plants. The value of insects as phytoplasma vectors is

considered proven if symptoms typical of the investigated phytoplasma disease appear on the experimental plant.

To confirm the infectivity of phytoplasma disease – PGD of wheat, all of the symptoms described above observed in the field on winter wheat crops can be reproduced as a result of simultaneous sowing and infection of plants in the fall. A massive manifestation of the disease of winter wheat in the form of "rosettes" in early spring and sometimes late autumn is the result of infection of plants of the PGD in the phase of two or three leaves in the second half of August – the first half of September. When plants are infected in a phase of two to three leaves during the second half of September and the first half of October, the disease manifests itself in the stem phase in the form of systemically diseased, laggingin growth plants with pale green leaves.

On the affected plants, as a rule, ears do not form. The degree of damage to wheat crops significantly depends on the method of sowing (there were cases where up to 60% of plants suffered from this disease with characteristic symptoms on broad-grown crops of winter wheat, and in narrow ones – 10–16%), as well as the stability of the variety, distribution and number of vectors cicadas, their natural contamination.

The method of indicator plants. To confirm the infectivity of phytoplasma disease, vaccinations are often used on the studied plant species and on the indicator plant, since it is not always possible to confirm the infectivity of phytoplasma by the method of inoculation with juice. This method of transferring a pathogen from plant to plant (under experimental conditions) is the most common. The infectious nature of the phytoplasma disease is considered to be proven if the lesion shows symptoms of the lesion that are identical or similar to the signs of the plant infected with the phytoplasma from which the stem for vaccination was taken.

Types of plants that are very susceptible to the pathogen are commonly used to prove infectiousness of phytoplasma disease. Among the indicator plants for phytoplasmas, it is necessary, first of all, to name the periwinkle – *Catharantnus roseus* (L) G. Don. This plant is the most convenient and suitable indicator in all respects, which responds to infection with various strains of phytoplasma isolated from wheat and on which clear, characteristic symptoms appear in response to infection of the phytoplasma. In addition, this perennial plant serves to maintain a clean infection for a long time, which is at the same time a store of pathogens in sufficient quantities, which is necessary in the future for serological studies of pathogens of wheat phytoplasmosis. In addition, periwinkle is much less likely to be exposed to accidental viral infection in a greenhouse.

In order to prove the infectivity of phytoplasma disease, in addition to vaccinations, especially when their use is difficult or practically impossible, *Cuscuta campestris, C. subinclusa* are widely used. For this purpose, the culm of plant is placed on the affected plant, and its other end, after the formation of the haustoria, is transferred to a healthy plant. When using this method, not only the transmission of infection occurs, but also the reproduction of the pathogen is recorded. At the same time, accumulations of phytoplasmas are manifested both in the cells of the conducting system and in the cells of adjacent tissues.

Molecular and genetic methods for the investigation of phytoplasmas. To identify phytoplasmas and pathogens of PGD of wheat – in particular, in recent years, molecular biology methods have been increasingly used determining the size of the genome, the composition of its nucleotides and the results of DNA hybridization from various isolates and strains [6]. The most complete information is obtained by studying the composition of proteins by electrophoresis in polyacrylamide gel with sodium dodecyl sulfate. So, using polyacrylamide gel electrophoresis, it is possible to detect differences between strains using protein composition [6].

Today, molecular-genetic methods, such as DNA hybridization and polymerase chain reaction (PCR), are based on the use of DNA probes and the principle of DNA complementarity to diagnose phytoplasma (in particular – wheat pathogens) [6, 9].

Phytoplasmas are very rarely specialized pathogens; in certain plants, they are determined by the species composition of the plants on which their carriers feed. The area of spread of the disease, as a rule, is limited to the area of distribution of the carrier. If several carriers transmit such a disease, then the area of its distribution consists of the sum of the ranges of each of them. Depending on the needs, the percentage of active vector carriers can be determined in the natural population. In this case, cicadas are caught in crops affected with phytoplasmas. At least 100–150 specimens are planted one at a time in individual isolators on healthy plants, where they are kept until perished.

The causative agents of phytoplasmosis are not inherent in specialization in certain host plants, so weeds that need to be destroyed can be their natural reserves.

The development of the disease, as well as the nature of its symptoms in plants infected with phytoplasmas, probably depends on the individual genotype of the plant. Therefore, factors of plant tolerance to phytoplasma infections are of particular interest, the identification of which can determine ways to solve the problem of controlling and suppressing phytoplasmosis in crops.

4.1 Wheat protection management against phytoplasmas

To preserve crops, one of the most important and necessary conditions is the protection of crops from phytopathogenic microorganisms. In this regard, there is a need for enhanced control over the spread of diseases, in particular – phytoplasmosis [14, 15].

Today, the main pathways for the distribution of phytopathogenic mollicutes in nature are known – these are insects and infected planting stock. To prevent the spread of phytoplasma disease of wheat, it is necessary to take into account the existence of many organizations of phytoplasma infection in nature. It is known that in nature phytoplasma infection spreads with cicadas. With regard to the ability to spread cicadas to plants, it is necessary, first of all, to develop measures to limit the number of these phytoplasma carriers, taking into account their ability to feed on certain species of host plants. At the same time, it is important to take into account the distribution of various types of cicadas – carriers of pathogens of phytoplasmosis of wheat, depending on various ecological and geographical zones of the country. In this case, general recommendations should be detailed in accordance with a specific geographical area. In addition, since pathogens of phytoplasmosis do not have specialization in certain host plants, weeds can be their natural reserves, especially perennial weeds, where phytoplasmas are first preserved and hibernated, and therefore are the source of infection for the next year. Phytoplasmas can also be stored and wintered on wild or cultivated plants and regardless of the conditions for their overwintering, next year they can all be a source of phytoplasmic infection. Therefore, an important preventive measure of protection against pathogens of phytoplasmosis is the destruction of weeds near the fields, including herbicides. They are used before or after the emergence of seedlings of culture. Spraying plants with insecticides (Aktara, Karate Zeon) is used to destroy the imago of cicadas during their appearance in the open ground. In the southern regions, the field cultivation period falls on the period from May 10 to May 30. In seedling greenhouses, plants should be sprayed with insecticides before planting them in the ground.

Therefore, it is important to promote the preservation of a healthy and partial recovery of the affected grain, pre-harvest field surveys and the priority collection of affected crops by direct combining during further drying and separation of grain.

So, to control and prevent phytoplasma diseases of wheat, it is necessary to apply such a number of complex measures – quarantine, prediction of epiphytoties, fulfillment of agrotechnical requirements for

growing crops, sowing wheat in optimal terms, compliance with scientifically based crop rotation, spatial isolation of new crops from sources of infection, removal of infected plants , destruction of weeds by reservation, introduction into production of wheat varieties resistant to phytoplasmosis, search for effective preparations for protection against phytoplasmas pathogens.

Biological methods of protection against pathogens of phytoplasmosis of wheat are also promising. In this case, it is necessary to determine the susceptibility of phytoplasmas to various biological preparations, as well as to physical factors.

Only under the condition of an integrated approach is it possible to resolve in the future the issue of phytoplasmosis of grain crops and, in particular, of such an important agricultural crop as wheat.

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To chapter 4

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Appendix

Appendix 1.1

Natural damage of *P.syringae* pv. *atrofaciens*

Basal glume rot – the agent of *P. syringae* pv. *atrofaciens*, artificial infection

Appendix 1.2

Bacterial leaf blight– the agent *P. syringae* pv. *syringae*

Appendix 1.3

Natural damage of *Xanthomonas translucens*

Appendix 2.1

Snow mould [32]

Sclerotia of *Typhula incarnata* in leaf sheath [32]

Appendix 2.2

Fusariumear blight (*FEB*) [32]

Appendix 2.3

Fusarium root rot [32]

Appendix 2.4

Septoriosis of ear and leaf – black pycnidia of *Mycosphaerella graminicola* (*Septoria tritici*) on brown necrotic spots [32]

Appendix 2.5

Helminthosporium root rot [32]

Appendix 2.6

Rhizoctonia root rot [32]

Appendix 2.7

Black head mold [32]

Appendix 2.8

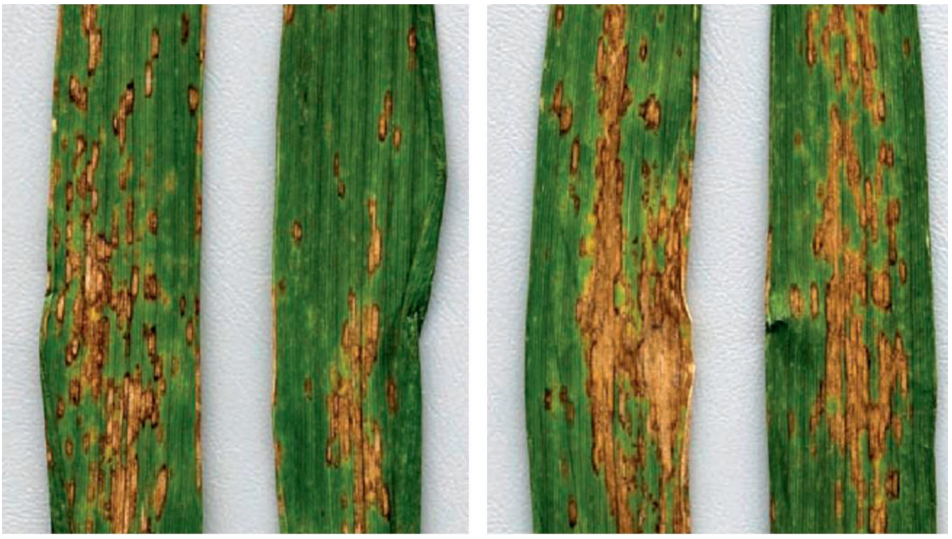
Take-all [32]

Appendix 2.9

Ergot [32]

Germinated sclerotium with fruit bodies [32]

Appendix 2.10



Ascochyta leaf scorch (*spot*) [32]

Appendix 2.11

Powdery mildew [32]

Appendix 2.12

Tan spot [32]

Appendix 2.13

Eyespot of stems [32]

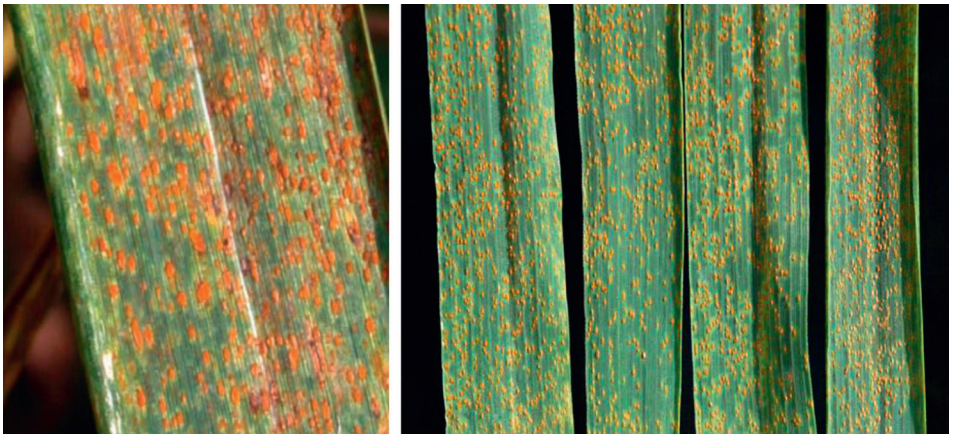
Appendix 2.14

Loose smut [32]

Appendix 2.15

Dwarf bunt [32]

Appendix 2.16



Brown (*leaf*) *rust* (author of photo: James Kolmer)

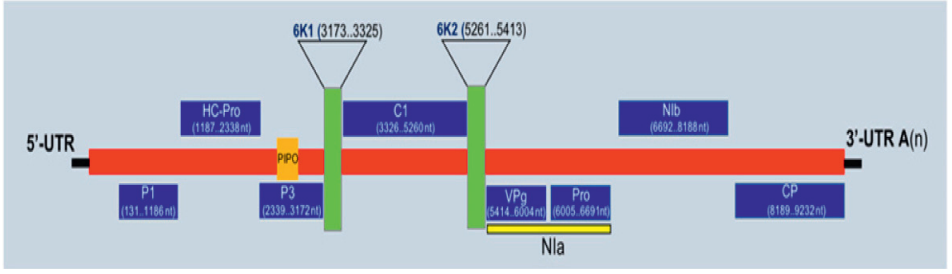
Appendix 2.17

Stem (*black*) *rust*
(author of photo Yue Jin)

Appendix 2.18

Yellow (*stripe*) *rust*
(author of photo: Yue Jin)

Appendix 3.1



Genomes structure of WSMV

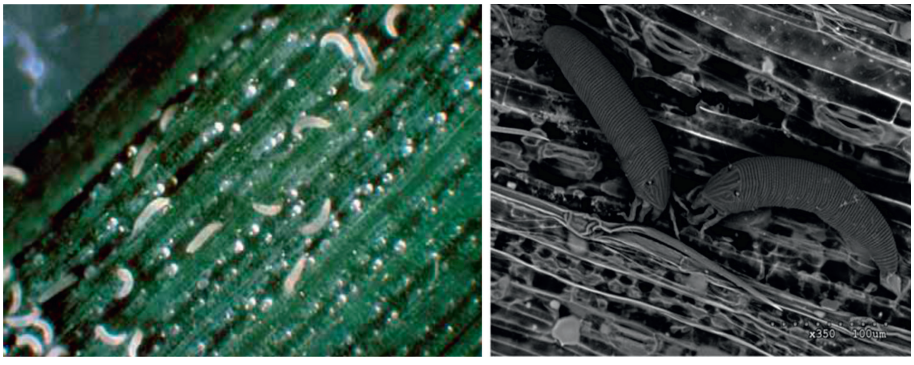
Appendix 3.2

World spread of WSMV

Appendix 3.3

Symptoms of WSM Von different varieties of winter wheat

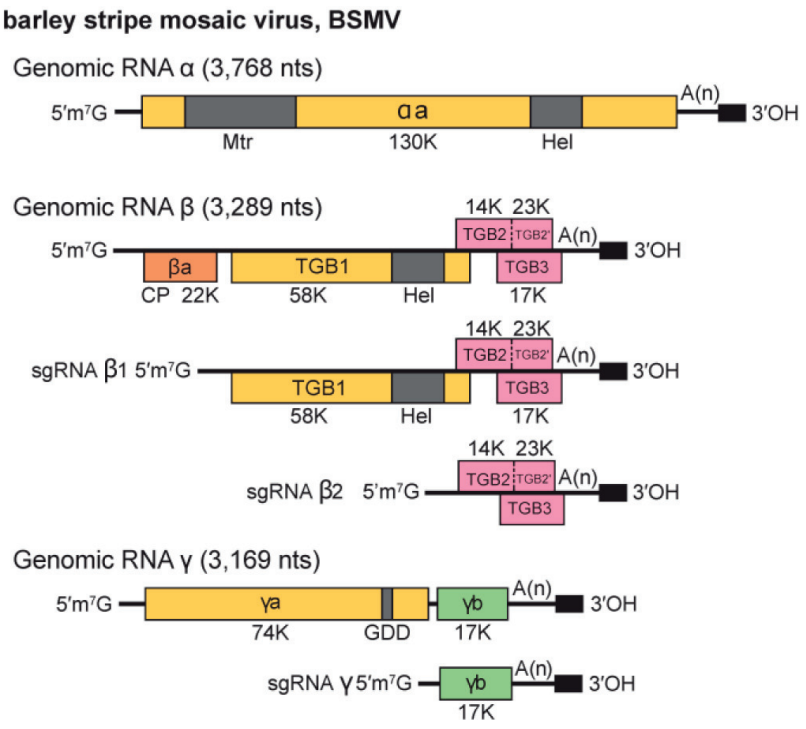
Appendix 3.4



a b

Mite *Aceria tosichella*, micrograph (a) andscanning electron microscopy (b)

Appendix 3.5



Genomic organization of BSMV

Appendix 3.6

Symptoms of BSMV on wheat

Appendix 3.7

(а) (б)

Symptoms of BM Von *Bromus inermis* (а) and *Zea mays* (б)

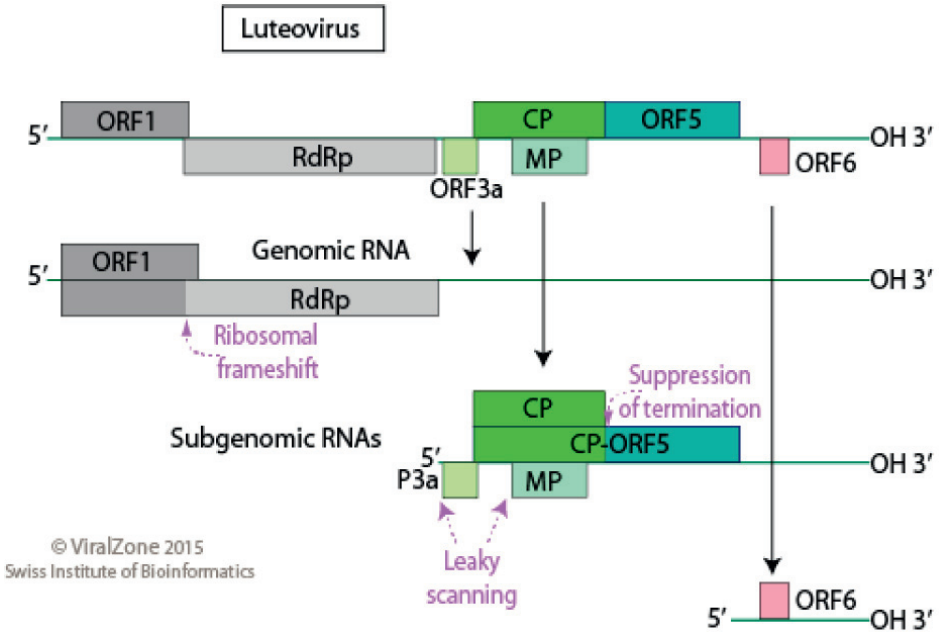
Appendix 3.8

Symptoms of WDV

Appendix 3.9

Cicadas *Psammotettix alienus*, vectors of WDV

Appendix 3.10

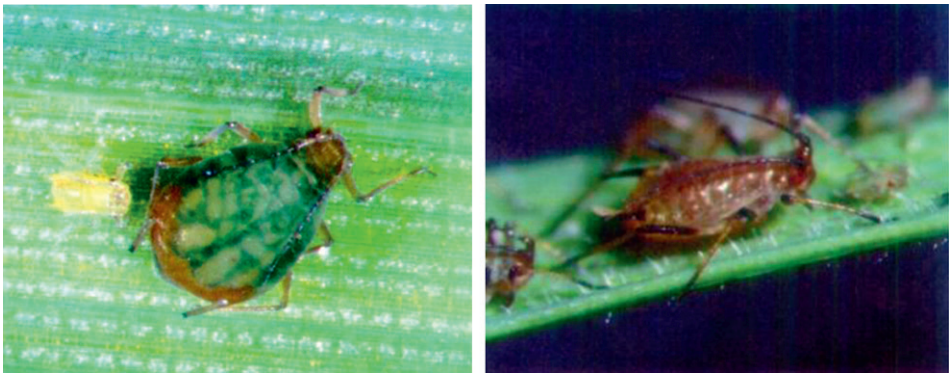


Genomic organization of BYDV-PAV

Appendix 3.11

Symptoms of yellowing and redness of wheat leaves due to damage by BYDV-PAV

Appendix 3.12



a b

Aphids, vectors of barley yellow dwarf virus (BYDV-PAV): *Rhopalosiphum padi* (a) and ***S****itobion avenae* (b)

Appendix 4.1

Stemming of flowerfilms in wheat earsaffected with pale green dwarfism (PGD)

Cicadas – carriers of phytoplasmas (genus: *Macrosteles*)