The Importance of *Fusarium* Fungi in Wheat Cultivation – Pathogenicity and Mycotoxins Production: A Review



1. ABSTRACT

Globally, cereals occupy about 50% of arable land. In Poland, that share is over 70%. In the structure of cereal crops, wheat is a dominant species. It occupies an area of 2.2 million hectares. The major part of this area is winter wheat, which is responsive to infection by pathogenic fungi. In recent years, the occurrence of pathogenic fungi of the genus *Fusarium* has been increasing. Their harmful effect consists of deterioration of the quality and quantity of yield and production of mycotoxins that can pose a threat to plants, animals and humans. In order to prevent the development of fusariosis, fungicides are used. However, incompetent and intensive use of fungicides may induce the resistance in strains previously sensitive to those substances. The aim of this essay is to present the problem of pathogenic *Fusarium* species occurrence in wheat crops and to discuss methods of fungicide control and related problems.

2. INTRODUCTION

Wheat has accompanied humans as a bread cereal for many decades now. Its cultivation was known as early as the Neolithic Age, about 7000 years BC, as evidenced by archaeological discoveries in the Middle East and China. To this day, this plant does not lose its importance, and among cereals cultivated in Poland it takes first place in terms of tillage area (Szempliński 2012). Cereals occupy about 50% of arable lands in the whole world. In Poland their share is over 70%, which is about 8.3 million hectares (Jaczewska-Kalicka and Krasiński 2011). The dominant species is wheat, which occupies an area of about 2.2 million hectares. Over 80% of this area is winter wheat, and the remainder is spring wheat (Statistical Yearbook GUS 2007). Wheat has high requirements in relation to climatic and environmental conditions (Rosada et al. 2010). It is also very sensitive to pests, especially pathogenic fungi that cause leaf, head, and stem diseases (Jaczewska-Kalicka 2010). Stem-base diseases in cereals may be caused by several species of pathogenic fungi. In Poland the most important are two of them: eyespot and stem-base rot, caused by fungi of the genera Tapesia and Fusarium. These diseases contribute annually to a significant loss in winter wheat production (Korbas 2004). The major economic importance among fungal infections of cereals is attributed to diseases caused by Fusarium spp. (Narkiewicz-Jodko et al. 2005). Especially stem-base rot and Fusarium head blight (FHB) constitute a serious problem wheat cultivation, contributing to a in significant reduction in yield and deterioration of its quality. Among the most dangerous fungal pathogens of wheat head are Fusarium culmorum, F. avenaceum and F. graminearum. Other

PLANT CIENCES

species of minor importance are *F. poae*, *F. oxysporum*, *F. sporotrichioides* and *F. verticillioides*. *Fusarium* head blight leads to a decrease in yield and reduces germination. Moreover, infested grain is often contaminated with mycotoxins. *Fusarium* epidemics can be a considerable problem for seed producers (Infantino *et al.* 2011; Xu *et al.* 2008).

Other frequently occurring fungal pathogens of wheat in Poland include *Blumeria graminis* that causes powdery mildew, *Puccinia recondita*causing brown rust of wheat, and *Phaeosphaeria nodorum*- causing glume blotch (Tratwal and Walczak 2012).

3. FUSARIUM SPP. – CHARACTERISTICS AND PATHOGENICITY

3.1. Description and taxonomy: The genus Fusarium contains over 70cosmopolitan species, occurring in natural conditions in different regions of the world. They are common in soil, as saprophytes. They can also grow on plant residues and other organic substrates. Some of them are facultative parasites, which, under conducive conditions, may cause plant diseases of both the underground and the aboveground parts (Leslie and Summerell 2006). According to the current taxonomy, Fusarium fungi belong to the kingdom: Fungi, phylum: Ascomycota, subphylum: Pezizomycotina, class: Sordariomycetes, subclass: Hypocreomycetidae, order: Hypocreales, family: Nectriaceae and genus: Fusarium[Index Fungorum (http://www.indexfungorum.org); Catalogue of Life (http://www.catalogueoflife.org)].The

teleomorphs of *Fusarium* species are mostly classified in the genus *Gibberella*, because they share the same species as type. In nature, *Fusarium* anamorphs occur more frequently than their *Gibberella* teleomorphs (Gräfenhan *et al.* 2011; Moretti 2009).

Complete review of the main taxonomic systems that have contributed to the defining of the modern taxonomy of *Fusarium* and various methods of their identification were described in the comprehensive manual of Leslie and Summerell (2006). This excellent and the most current work contains an updated description of 70 species within the genus.

The main approach for the *Fusarium* classification is still morphology. *Fusarium* colonies are usually fast growing, pale or brightly coloured, and may have a cottony aerial

mycelium. The tinge of the thallus varies from whitish to yellow, brownish, pink or reddish. Fusarium species can create three types of spores: macroconidia, microconidia and chlamydospores. Septated macroconidia (3-8 x11-70 µm) are colourless, fusiform to sickleshaped, often with an elongated apical cell and pedicellate basal cell; they are produced in specialized structures called sporodochia. They can also be produced on monophialides and polyphialides in the aerial mycelium. Monophialide is a conidiophore that has only one opening or pore through which endoconidia are extruded. Polyphialide is a structure with two or more openings or pores. Macroconidia tend to accumulate in ropes or balls. The different shape of macroconidia remains the most important feature for recognition the species. Microconidia (2-4 x 4-8 µm) are produced only in the aerial mycelium. Their presence or absence is a primary feature in Fusarium taxonomy. Microconidia have variable shapes and sizes. Usually they are 1celled, smooth, colourless, ovoid to cylindrical and accumulated in balls, sometimes in chains. is The third type of spores called chlamydospores. They are thick-walled, colourless and may be formed singly, in pairs, in clumps or in chains. The outer wall of chlamydospores can be smooth or rough (Nelson et al. 1994; Summerell et al. 2003, Leslie and Summerell 2006; Moretti 2009;)

3.2. Fusarium as a plant pathogen: Fusarium spp. are considered some of the most dangerous pathogens of cereals, potatoes, vegetables, Fabaceae, ornamental and forest plants, causing significant economic losses. Most of them are polyphagous, infecting different plant species. They can infect plants at any stage of growth, causing seedling blight, root rot, stem-base rot, leaf fusariosis and Fusarium head blight in cereals and (Champeil et al. 2004a; Łukanowski and Sadowski 2002). In wheat cultivation, the most considerable problem are two diseases: stem-base rot and Fusarium head blight, also known as scab. It is extremely difficult to reduce the occurrence of those diseases, because of the large share of cereals cultivation in Poland. Another factor influencing the occurrence of diseases is weather conditions that affect both plant growth and the presence of infection (Doohan et al. 2003; Narkiewicz-Jodko et al. 2005). Fungi of the genus Fusarium are present with high intensity in warm and wet years, with a large amount of precipitation, because they spread through macroconidia, which are transferred to the upper plant organs in raindrops. They can also produce forms called perithecia. After the rains, these structures throw out ascospores that are transferred with the wind directly to the heads. Infection of the head and grain occurs when the temperature during the flowering exceeds 20°C and the humidity is greater than 85-90% for at least 24-40 hours (Champeil et al. 2004a). The research of many authors shows that the main factors of stem-base rots are Fusarium culmorum and F. avenaceum. F. culmorum prefers temperature above 25°C and low precipitation, while F. avenaceum occurs more often in cooler (10-25°C) and humid growing seasons (Plaskowska 2010). A vastly important factor is the moment when the infection reaches the plant that is the most sensitive in the flowering stage or in the initial stage of milk ripeness of grain. Under favourable conditions the infection of seed can occur. Highly infected grain is characterised by decline in the quality, reduced weight of 1000 grains, low protein content, chalky, impaired ability to germinate, deterioration in gluten quality and quantity (Gilbert 1992, Boyacioglu and Hettiarachchy 1995; Dexter et al. 1997;). Such grain also contains mycotoxins, which may be dangerous

to human and animal health: zearalenone and deoxynivalenol (vomitoxin). These toxins from grain get into all of its milling products and they can be found even in baked bread (Grabarkiewicz-Szczesna et al. 2001). The presence of mycotoxins in infested grain depends on environmental factors and on the genotype of plant (Miedaner et al. 2001). A major problem in the cultivation of cereals is FHB, which contributes to significant quantitative losses and deterioration of the quality of grain and its products. Yield losses caused by Fusarium head blight can be very high and in extreme cases reach up to 70%. In European conditions the losses come to 10-20% (Sayler 1998). FHB is a pre-harvest disease, but Fusarium species can also grow post-harvest. The fungus grows if wet grain is not dried quickly and efficiently. The contagion during the flowering or at the beginning of milk ripeness of grain is called overt fusariosis. Affected wheat grains are small, light, wrinkled and sometimes covered with a pink or white down, and they have a reduced ability to germinate. Ovalor round stains with clear centres and dark edges may be visible on the back of the grain (Parry et al. 1995; Sutton 1982). When the infection occurs in the mealy ripeness or kernel hard stage, it is called covert fusariosis. Grain is normally formed and has high germination ability (Narkiewicz-Jodko 1998; Pirgozliev et al. 2003). The disease results from the development of a complex of two kinds of pathogenic fungi: Microdochium and Fusarium. The Fusarium complex consists of five main species: F. graminearum, F. culmorum, F.avenaceum, F. poae and F. tricinctum, of which the most common species in Poland and Europe are F. avenaceum, F. graminearum and F. culmorum (Champeil et al. 2004a; Chełkowski et al. 2012).Individual Fusarium species can produce a range of mycotoxins in infected grain, whereas Microdochium species are considered unable to produce mycotoxins. Moreover, Fusarium species predominate in hotter regions of Europe, while in cooler maritime areas, Microdochium species are

JOURNAL OF ANIMAL PLANT SCIENCES

favoured (Xu *et al.* 2008). The climate is the principal factor affecting the development of *Fusarium* head blight - humidity determines the intensity of the disease, while precipitation determines inoculum levels (Francl 1998). It

4. MYCOTOXINS PRODUCED BY SELECTED FUSARIUM SPECIES

For cereal products, one of the most important elements of health risk is the possibility of mycotoxins accumulation in the kernels. These toxins are the products of fungal secondary metabolism that occurs when growth stops or slows distinctly. Mycotoxins are the major problem in food safety, they are harmful to both animals and humans (Gilbert and Tekauz 2000; Ngoko et al. 2008). Some of them are produced before harvest (fumonisins, zearalenone and trichothecenes), while others are produced after harvest (aflatoxins and ochratoxin A). Fusarium species are responsible for mycotoxins production before harvest (Champeil et al. 2004b). The main toxins produced by pathogenic Fusarium species are: zearalenone, deoxynivalenol and derivatives,

may also affect the production of mycotoxins in the field (Magan *et al.* 2002). The effects of climate are uncontrollable and difficult to predict due to their complexity (Champeil *et al.* 2004a).

nivalenol, T₂-toxin and HT₂-toxin (Placinta et al. 1999). They occur on a worldwide basis in cereal grains, animal feed and forages. Other important Fusarium toxins include moniliformin and fusaric acid. Table 1 summarises the key Fusarium spp., which can be involved in head blight in cereals, and the major toxins produced by them. Contamination of feed grains is inevitable since many toxigenic Fusarium species are also common phytopathogens, causing cereal crop diseases, which are difficult to control (D'Mello et al. 1999). Excessive moisture in the field and storage, humidity, drought and temperature extremes is principal environmental factors that determine the intensity of mycotoxins contaminations (Coulombe 1993).

Table 1: Important species of Fusarium infecting cereals, their frequency of occurrence in Poland and Europe
and mycotoxins produced

Species	Frequency	Aggressiveness to head	Major mycotoxins produced
F. culmorum	+++++	high	deoxynivalenol (DON), 3-acetyl- deoxynivalenol (3ADON), 15- acetyl- deoxynivalenol (15ADON), nivalenol (NIV), fusarenone (FUS), zearalenone (ZEN)
F. graminearum	+++++	high	DON, 3 ADON, 15ADON, NIV, FUS, ZEN
F. sporotrichioides	++	low	T ₂ -toxin, HT ₂ -toxin, neosolaniol (NEO), diacetoxyscirpenol (DAS), FUS, ZEN
F. poae	+++	average	T ₂ -toxin, HT ₂ -toxin, NIV, DAS, FUS
F. verticillioides	++++	average	fumonisins, moniliformin, fusarin C
F. proliferatum	++	average	fumonisins, moniliformin, fusarin C
F. avenaceum	++++++	average	moniliformin, beauvericin
F. cerealis	++	high	NIV, FUS, ZEA
F. tricinctum	++	low	moniliformin
F. oxysporum	++	saprophyte	moniliformin, wortmannin, fusaric acid
F. equiseti	++	saprophyte	fusarochromanone, ZEN, DAS

PLANT SCIENCES

4.1. Trichothecenes: Trichothecenes (Fig. 1) are the largest group of fungal toxins. They are classified in the group of tricyclic sesquiterpenes and are characterised by the presence (type A trichothecenes) or absence (type B trichothecenes) of a keto group at the C-8 position. Type A HT₂-toxin, corresponds T_2 -toxin, to diacetoxyscirpenol (DAS) and scirpenol. Type B trichothecenes include deoxynivalenol (DON. vomitoxin) and its derivatives: 3-acetyldeoxynivalenol (3ADON) and nivalenol (NIV). Trichothecenes are potent inhibitors of eukaryotic protein synthesis and have gained particular notoriety as a group of materials highly toxic to both plants and animals. Fusarium culmorum, Fusarium graminearum and Fusarium sporotrichioidesare the main producers oftrichothecenes. These toxins are common fungal contaminants of cereals and occur naturally worldwide in the cultivation of wheat and other cereals (Brown et al. 2001; Champeil et al. 2004a; Wagacha and Muthomi 2007). Apart from their phytotoxicity, trichotecenes play an important role as virulence factors in head rots of wheat and maize caused by Fusarium graminearum (Desjardins et al. 1996; Harris et al. 1999). Increase of mycotoxingrains is caused by contaminated using monocultures and minimum tillage. The effect of these systems is the accumulation of fungal inoculum in soil and crop debris. The most Europe frequently detected toxin in is deoxynivalenol (Korbas and Horoszkiewicz-Janka 2007). DON retards the germination and growth of wheat, delays the growth of the grain and the coleoptiles tissues (Snijders and Perkowski 1990). In mature plants, deoxynivalenol seems to circulate in the phloem, with the concentration in plant following a descending gradient from the rachis, through the lemmas and grains to the peduncle. Moreover, from the fourth day after inoculation, the flower parts, rachis and peduncle contain larger amounts of DON below the point of infection than above it (Savard et al. 2000; Sinha et al. 1997). The production of deoxynivalenol by Fusarium is believed to play an important role in the

pathogenesis of head blight. The research shows that Fusarium culmorumstrains belonging to the chemotype of DON are more aggressive towards barley seedlings. Furthermore, DON-producing F. culmorum isolates are more virulent than those producing NIVin wheat. These conclusions strengthen the contribution of DON in the pathogenicity of F. culmorum in different cereal cultivations (Hestbjerg et al. 2002; Muthomi et al. 2002). Deoxynivalenol can induce complete decline of chloroplast pigments at sub-lethal concentrations (Rotter et al. 1996). Trichothecenes are also a primary determinant of Fusarium graminearum pathogenicity in most wheat cultures. It is observed that a lack of trichothecenes synthesis by F. graminearum resulted in susceptible plants being capable of slowing down and even stoppingFusarium spread (Eudes et al. 2001). Both deoxynivalenol and nivalenol are characterised by strong toxicity to plant tissues in growth. They cause yield reduction in spikes even at low concentration. It is possible that, in the presence of the toxin, plant defence mechanisms are not induced fast enough, therefore the aggressiveness of the pathogen increases (Ittu et al. 1995). In contaminated plant whole spikelets prematurely senesce, and if the rachis is infected, the head above the point of infection senesces. Kernels from infected spikelets are usually shrivelled and are chalky white or pink in colour (Edwards 2004). Fusarium infection can result not only in yield losses but also in deterioration of milling and malting quality, and in the contamination of grains with trichothecenes, which are harmful to all organisms. Consumption of food or forage contaminated with these substances causes both animal and human diseases. In mammals, the absorption of trichothecenes causes blood and digestive disorders. Emesis is observed after the ingestion and absorption of at least 10 mg DON·kg-1, together with irritation of the mouth and oesophagus mucous membrane (Champeil et al. 2004a; Edwards 2004). The adverse effects of individual trichothecenes in porcine and nonporcine livestock are summarised in Table 2.



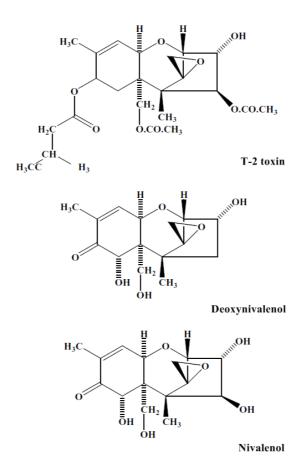


Fig. 1: Chemical structure of trichothecenes produced by Fusarium

In humans, the absorption of trichothecenes leads to burning sensations in the mouth and stomach, headaches, a reduction in red blood cell count, bleeding, necrosis of throat and stomach. In some cases, trichothecenes poisoning may even lead to death (Champeil *et al.* 2004a).

4.2. Fumonisins: Fumonisins are long-chain amino polyalcohols. They consist of a 20-carbon aliphatic chain with two ester linked hydrophilic flank chains. This structure is similar to sphingosine, an essential phospholipid within cell membranes. Although fumonisins have a relatively simple chemical structure, they may have diverse and complex effects in animals and human systems. The toxicity of fumonisins is a result of competition with sphingosine in sphingolipids metabolism (Desjardins and Proctor 2007; Riley et al. 1996). The most significant food source contaminated by these mycotoxins is maize, but the presence of

fumonisins was also found in wheat grain. A large number of commodities in the world have been analysed for fumonisins contamination, however they have mostly been reported in maize and its products (De Girolamo et al. 2011; Scott 2012). Few studies (Busman et al. 2012; Palacio et al. 2011) have been carried out on the natural contamination of wheat with fumonisins. Although, Fusarium verticillioides isolated from wheat has been reported to produce high concentrations of these toxins (Mashinini and Dutton 2006; Stanković et al. 2012). The most toxic representative of these group is fumonisin B_1 (Fig. 2) which is produced mainly by Fusarium proliferatum and F.verticillioides, commonly occurring in cereal crops(Edwards et al.2002; Pławińska-Czarnak and Zarzyńska 2010). Fumonisin B₁ is known to induce toxicity on contact in various plant tissues.



Animal species/type	Trichothecene	Effects		
Pigs	DON	emesis; reduced feed intake; decreased growth; stomach lesions; reduced blood levels of total protein, albumin, Ca and P; temporary decrease in serum protein levels; renal lesions		
	NIV	profound reduction in feed intake; increased time to consume feed		
	T ₂ -toxin	dermatitis of nose, snout and buccal commissures; reduced growth; decreased blood glucose levels; increased blood levels of inorganic P and Mg; decreased red blood cell amount; reduced corpuscular volume and haemoglobin concentration; reduced leukocyte count; decreased antibody formation		
Broiler chickens	DON	increased weight of heart, gizzard and bursa of Fabricius		
	NIV	gizzard erosions; reduced weight of liver		
	T ₂ -toxin	decreased bodyweight gain; oral lesions		
	DAS	decreased bodyweight gain; mouth lesions		
Turkey poults	T ₂ -toxin	reduced weight gain; oral lesions		
	DAS	reduced feed intake and weight gain; oral lesions		
Geese	T ₂ -toxin	decreased egg yield and hatchability; inhibition of follicle maturation in ovaries; follicle degeneration; lesions in adrenal and thyroid glands		
Cattle	DAS	anorexia; gastro-intestinal lesions; diarrhoea; reduced milk production		

Table 2: Adverse effects of individual trichothecenes in livestock

Disruption of sphingolipid metabolism, resulting in accumulation of sphingolipid bases, has been implicated in the development of disease symptoms, which include changes in the plasma membrane, disruption of the electrolyte leakage, chloroplast degeneration, chlorosis and necrosis (Abbas *et al.* 1993; Lynch *et al.* 2004).

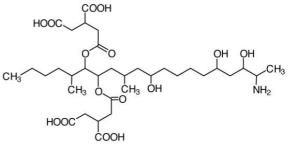


Fig. 2 Chemical structure of fumonisin B_1

The fumonisins were the most recent group of fungal toxins identified after they were found to be a causative factor of the fatal syndrome, equine leukoencephalomalacia. They have since been shown to have a number of deleterious effects in animals and humans (Kellerman et al. 1990, Edwards et al. 2001;). Fumonisins induce disorders after consumption of contaminated feed or food (Reddy et al. 2010). The diseases include porcine pulmonary edema, hydrothorax in pigs, hepatocarcinoma in rats, and immunosuppression in poultry. Absorption of fumonisins causes the formation of cancerous tumors in laboratory rodents and there is an epidemiological correlation between the consumption of infected grain and human oesophageal cancer in some regions of the world (Rheeder et al. 1992, Bucci and Howard 1996, Proctor et al. 2002;).

4.3. Zearalenone: Zearalenone (ZEN) (Fig. 3) is a non-steroidal estrogenic mycotoxin produced mainly by Fusarium culmorum, F. graminearum and F. sporotrichioides. This toxin is most frequently isolated from maize; but it also contaminates other cereals, including wheat, barley, oat and sorghum. It is associated with head rot and stalks rot in maize and with scab in wheat, moreover it is a natural contaminant of hay (Coulombe 1991; Krska et al. 2003). ZEN is one of the most common mycotoxins produced by Fusarium in the temperate regions of Europe, Asia and America (Mankevičienė et al. 2011). It belongs to a very large family of fungal metabolites derived by cyclisations and modifications of non-ketide precursors. ZEN has a chemical structure similar to estrogen, and it can produce an estrogenic response in animals. It has also a close structural similarity to antibiotic metabolites produced by many species of fungi (Desjardins and Proctor 2007). It is established that zearalenone is characterised by a relatively low toxicity, with an LD₅₀ value of 2-10 g·kg⁻¹ body weight as determined with mice (Flannigan 1991). However, its role as an endocrine disrupter is being recognised in detailed research, with effects in males and females of different species of mammals (Pfohl-Leszkowicz et al. 1995). It was discovered to be the cause of reproductive disorders in domestic animals. Fusarium fungi are able to producing both trans (α -zearalenol) and *vis* (β -zearalenol) forms of ZEN on grain. These metabolites bind to estrogen receptors varying to extents, causing

hyperestrogenism resulting in various reproductive and infertility problems, especially in pigs. Those animals are particularly susceptible to this compound because their estrogen receptors have a high affinity for α -zearalenol, which is three to four times more estrogenic than ZEN (Fitzpatrick et al. 1989; D'Mello et al. 1999; Moss 2002). Swine diseases caused by zearalenone include enlargement and swelling of the vulva in sows and gilts (vulvovaginitis), swelling of the mammary glands, atrophy of the ovaries, vaginal and rectal prolapsed. In boars ZEN reduces libido and causes a reduction in sperm quality, while in young males it may cause swelling of the prepuce, testicular atrophy and enlargement of the mammary glands (Prelusky et al. 1994).ZEN and its derivatives are capable of inhibiting mitogen-stimulated lymphocyte proliferation and can cause thymic atrophy and macrophage activation (Visconti et al. 1991). Zearalenone can be an important etiologic agent of intoxication in young children and foetuses exposed to this compound, which causes premature thelarche and breast enlargement (Mankevičienė et al. 2011). ZEN may also be one of the risk factors that influence the occurrence of mammary gland cancer in women (Kuciel-Lisieska et al. 2008). Although in west European countries ZEN in considered as a toxin, a number of studies have shown that it may also have a positive effect on plant growth. It was investigated as a regulator of the flowering process (Biesaga-Kościelniak and Filek 2010). It is established that exogenous application of ZEN in the culture of winter wheat isolated embryos strongly increases the percentage of generative plants. Research indicated that zearalenone also shows similar activity to plant hormones in some physiological processes (Biesaga-Kościelniak 2001). The application of ZEN may be useful in *in vitro* cultures. The studies have shown that ZEN stimulates the regeneration processes, growth of the callus tissue and its differentiation in wheat (Biesaga-Kościelniak et al. 2010). In view of its hormonal activity there is much information about zearalenone in the literature on growth hormones, used in animal production. Some countries, such as the USA, permit the use of zearalenone for increasing meat production. Because any compound with hormonal activity may be carcinogenic or genotoxic and there is some information that zearalenone may show both types of activity in some animal species, this practice is

banned in the countries of the European Community (Moss 2002).

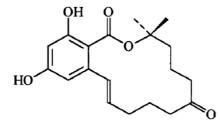


Fig. 3: Chemical structure of zearalenone

4.4. Masked mycotoxins: Recently glycosylated derivatives of deoxynivalenol and zearalenone called 'masked mycotoxins' were identified in wheat grain. These toxins are result of the glycosylation process in wheat tissues in the second phase of plant metabolism. Conjugation with glucose can convert DON into DON-3

glucoside (DON-3G) and ZEN into ZEN-14 (ZEN-14G). glucoside Chemically altered mycotoxins, as they escape analytical detection designed to DON and ZEN, because of changed polarities and masses (Vendl et al. 2009). They are recognized as potentially hazardous for animal and human because they can be transformed to DON and ZEN in the digestive tract (Chełkowski et al. 2012; Stępień and Chełkowski 2010).Occurrence of DON-3G has been proven in wheat kernels (Berthiller et al. 2009). It was also found in malt and beer (Lancova et al. 2008). Schneweis et al. (2002) proved ZEN-14G occurrence in wheat samples originating from Bavaria. There is a little information on the occurrence of this ZEN derivative in wheat and other cereal grains in Poland (Perkowski et al. 1997, Gromadzka et al. 2008;).

 Table 3:
 Maximum permissible level of mycotoxins content

Mycotoxin	Product	Maximum permissible level (µg·kg ⁻¹)
	unprocessed cereals	1 250
	unprocessed durum wheat, oat	1 750
	unprocessed corn	1 750
Deoxynivalenol	cereals intended for direct human consumption, cereal flour, bran	750
	pasta	750
	bread (include bakery products), confectionery products, biscuits, cereals	500
	processed cereal-based food, food for infants and young children, baby products	200
	unprocessed cereals	100
	unprocessed corn	350
	cereals intended for direct human consumption, cereal flour, bran	75
	refined corn oil	400
Zearalenone	bread, confectionery, biscuits, cereals	50
	processed cereal-based food , food for infants and young children, baby products	20
	corn intended for direct human consumption, corn snacks, corn flakes	100
	processed corn-based food for infants and young children,	20
	unprocessed corn	4 000
Fumonisins	corn intended for direct human consumption	1 000
	corn snacks, corn flakes	800
	processed corn-based food for infants and young children,	200

4.5. Mycotoxins content limits: Cereal products may be contaminated by mycotoxins at any stage, from the growth of the plant, through the harvest, as well as during processing, storage and transport of the end product. The maximum permissible level of contamination by selected mycotoxins (Tab. 3) is regulated by Commission

Regulation (EC) No. 466/2001 of8th March 2001 as amended (Commission Regulation(EC) No. 466/2001of 8th March 2001; Commission Regulation (EC) No. 856/2005 of 6th June 2005;Commission Regulation (EC) No. 1126/2007 of 28th September 2007).

5. FUNGICIDE CONTROL OF *FUSARIUM* HEAD BLIGHT

In recent years the occurrence of fungal infections of cereals has been increasing everywhere, which may be explained by changing climatic conditions (Ioos et al. 2005). This indicates a need to intensify protective measures. Wheat is in particular one of the crops most prone to Fusarium infections and critical mycotoxin problems. Over the last decade the quality and the safety of grain productions have increased in importance. Management of Fusarium head blight includes many effective protection procedures, especially based on agrotechnical, immunological methods biological and of controlling infections, as well as fungicide application (Blandino et al. 2006; Grishechkina et al.2012). Unfortunately, there is currently no single effective method of reducing the incidence of FHB or mycotoxin contaminationin cereals. Fungicide application is one measure available to reduce the risk; however, it does not guarantee getting rid of the pathogen(Horslev et al. 2006, Baturo-Cieśniewska et al. 2011;). The efficacy of fungicide use for the control of FHB and mycotoxins production varies from being highly effective to even increasing mycotoxin levels (Müllenborn et al. 2008). Some of the reasons of the variable effects of fungicides on FHB are probably cultivar resistance, fungicide efficacy, fungicide coverage, timing and frequency of applications, different epidemiology and the natural variation in aggressiveness of individual pathogens (Homdork*et al.* 2000: Mesterhàzy et al. 2003). Application at flowering stage seems to be the best time for the reduction of Fusarium infection risk (Chala et al. 2003).

5.1. Efficacy of various types of fungicides : Fungicide applications, pre-harvest are necessary whether in intensive or sustainable cereal production systems. Thus *Fusarium* species colonising cereals will be exposed to various groups of fungicides, such as imidazoles, benzimidazoles, triazoles, morpholines or strobilurins (Mateo *et al.* 2011). Each group is characterised by a different mechanism of action and different efficacy in

prevention of Fusarium diseases. In the European Union, many triazoles are registered for wheat. As shown in various studies, they proved to be the most active compounds against the present set of toxigenic fungal species (Matthies and Buchenauer 2000; Menniti et al. 2003). Fungicides of this type have an influence on fungal metabolism, mainly by inhibition of ergosterol biosynthesis. They often cause striking morphological deformations, irregular cell wall thickening and excessive branching of fungi. They lead to the inhibition of spore germination, initial cell growth and dry weight increase (Ramirez et al. 2004). The most widely used triazole fungicides against FHB are tebuconazole and metconazole. They show high efficiency against Fusarium culmorum and other fungal pathogens of wheat (Simpson et al. 2001, Ioos et al. 2005;). In studies by Sikora and Banachowska (2006), a group of 8 fungicides was investigated for their activity against Fusarium culmorum. Among all tested active compounds, metconazole proved to be the most effective. In many studies, triazole fungicides: tebuconazole and metconazole were shown to be effective in reducing both FHB and DON production (Jennings et al. 2000, Edwards et al. 2001;). Pirgozliev et al. (2002) showed a great reduction in FHB and DON in grain treated with metconazole, which indicates that this fungicide is very effective against both Fusarium culmorum and F. graminearum. Dardis and Walsh (2000) also reported that this compound was the most effective of a range of fungicides tested against F. culmorum. Several studies on chemical control of FHB and other Fusarium infections in grains report that the best results in artificially inoculated trials are obtained after the application of tebuconazole (Henriksen and Elen 2005; Mesterhàzy et al. 2003). According to Cromey et al. (2001), tebuconazole considerably reduces FHB incidence as well as Fusarium and mycotoxins levels in grain. Another triazole fungicide, prothioconazole, was highly effective in reducing the mycelial growth of

Journal of Animal & Plant Sciences, 2014. Vol.21, Issue 2: 3326-3343 Publication date 30/4/2014, http://www.m.elewa.org/IAPS; ISSN 2071-7024

Fusarium avenaceum, F. culmorum, F. graminearum, F. poae, F. sporotrichioidesand F. tricinctum. There was evidence that application of fungicides at flowering was more efficacious than in the other stages of growth (Müllenborn et al. 2008). Prochloraz is an imidazole derivate and its mechanism of action is similar to that of triazole fungicides. Like tebuconazole, it is extensively applied in agriculture to control fungal growth in cereals in many European countries (Ramirez et al. 2004, Mateo et al. 2011;). According to Menniti et al. (2003), its application in the field reduces the production of deoxynivalenol by an average of 43%. Prochloraz is effective especially against Fusarium graminearum, F. avenaceum and F. culmorum when infection pressure is low to medium. This compound inhibits mycelial growth of pathogen and reduces 3AcDON production (Matthies et al. 1999; Pirgozliev et al. 2003). Fungicides such as carbendazim, benomyl or thiabendazole belong to benzimidazoles group. They are specific inhibitors of microtubule assembly that act by binding to their heterodimeric subunit, the tubulin molecule. Consequently, mitotic division of the nucleus is blocked and the fungal cell loses its ability of proliferation (Davidse 1986, Cromey et al. 2001). The wide application of benzimidazoles in agriculture can lead to the development of resistance in pathogenic fungi (Steffens et al. 1996).In studies by Jones (2000), carbendazim was not as immediately effective as tebuconazole, but showed arelatively good duration of FHB control, benomyl showed levels of control equal to tebuconazole. According to Cromey (2001), carbendazim considerably reduces Fusarium disease incidence as well as mycotoxins content in grain. The moment of application has a major impact on its effectiveness. Hutcheon and Jordan (1990) reported that carbendazim was effective against Fusarium avenaceum, F. culmorum and F. graminearum by reducing their levels by about 70% in the wheat heads over the control treatment. Strobilurins, such as azoxystrobin and kresoxim-methyl, are a relatively new group of fungicides. These compounds act by blocking electron transport in the mitochondrial respiratory chain in fungi. This reduces the aerobic energy production, thereby inhibiting growth of the fungus. This new mode of action makes strobilurins an important addition to the existing fungicide range (Ypema and Gold 1999, Ramirez et al. 2004;).Different authors (Bertelsen et al. 1999; Simpson et al. 2001) reported that the

application of strobilurins, unlike the triazoles, has little effect on the reduction of Fusarium culmorum, F. graminearum and F. avenaceum, while it is highly effective in the reduction of Microdochium nivale. Lienemann et al. (2002) and Mesterhàzy et al. (2003) reported that strobilurin fungicides increase DON production, although they can partially control FHB. Contrary to these researchers, Siranidou and Buchenauer (2001) observed no effect of strobilurins on DON content, or even a decrease. In studies by Nakajima (2010), azoxystrobin was not as effective as triazole fungicides, but its efficacy was about 40%. Moreover, this compound increased the DON level significantly. As suggested by Cromey et al. (2001), early control of FHB by azoxystrobin did not translate into lower levels of Fusariumor mycotoxins in grain, it only slowed the development of FHB symptoms. The new strobilurin, fluoxastrobin, seems to be more effective in controlling mycelial growth of Fusarium species than azoxystrobin (Ioos et al. 2005).

5.2. Fungicide resistance phenomenon: A major role in suppressing crops infection is played by fungicidal treatment at optimal time. The efficacy of widely used fungicides is not higher than 40-60%, and sometimes it reaches 70%. Moreover, long use of some fungicides leads to the accumulation of Fusarium fungi in fungal community of cereals (Grishechkina et al. 2012). The effectiveness of fungicides is highly variable, even within the same group. One possible cause of these different reactions is the existence of fungicide-resistant strains within the Fusarium species (D'Mello et al. 2000). A fungal population may be resistant towards a certain fungicide from the very beginning, which is termed natural resistance (Brent and Hollomon 2007). It can also become resistant towards the fungicide during the course of its use, and this phenomenon is referred to as acquired resistance (Dubos et al. 2011). Most of systemic fungicides have a site-specific mode of action, therefore even a small biochemical change in the fungus may lead to the development of resistance. Resistant strains arise by the selection of individual specimens or as a result of single mutations. The newly created strains may be able to avoid a block in metabolism and circumvent the action of the fungicide (Isaac 1999). Fungal resistance towards benzimidazole fungicides could be correlated with changes in benzimidazoles binding to tubulin (Steffens et al. 1996). Resistance

towards strobilurins is caused by mutations in the cytochrome *b* gene. When glycine at position 143 is replaced by alanine, the fungus becomes highly resistant to these fungicides (Kaneko and Ishii 2009; Torriani *et al.* 2009).Intensive use of fungicides leads **6. CONCLUSIONS**

Fusarium species produce an extraordinary diversity of biologically active secondary metabolites, some of which are harmful to animals and humans, like mycotoxins. *Fusarium* diseases of wheat and other cereals cause significant losses worldwide, and therefore are the most important factor in quality and economy sector. The reduction of diseases and mycotoxin content in wheat is possible through the use of specific fungicides, however that does not guarantee complete elimination of the pathogen. Some of the fungicides even increase mycotoxins level. Furthermore, pathogen populations may be resistant towards certain fungicides. Because of the

7. **REFERENCES**

- Abbas HK, Duke SO, Tanaka T: 1993. Phytotoxicity of fumonisins and related compounds. *Journal of Toxicology- Toxin Reviews* 12: 225-251.
- Baturo-Cieśniewska A, Łukanowski A, Kolenda M: 2011.Effect of fungicide application on wheat head blight, occurrence of *Fusarium spp.* and mycotoxin production. *Plant Breeding and Seed Science* 63: 29-38.
- Bertelsen JR, De-Neergaard E, Smedegaard-Petersen V: 1999. Reason for improved yield when using azoxystrobin in winter wheat. In: 16th Danish Plant Protection Conference. Crop Protection in Organic Farming, Pest and Disease, Tjele, Denmark.
- Berthiller F, Dall'Asta C, Corradini R, Marchelli R, Sulyok M, Krska R, Adam G, Schuhmacher R: 2009. Occurrence of deoxynivalenol and its 3-B-D-glucoside in wheat and maize. *Food Additives and Contaminants Part A* 29: 507-511.
- Biesaga-Kościelniak J: 2001. Zearalenone as a new hypothetical regulator of plant growth and development. Monograph of Institute of Plant Physiology, Polish Academy of Sciences, Kraków, Poland (in Polish, with English summary, description of tables and figures).
- Biesaga-Kościelniak J, Filek M: 2010.Occurrence and physiology of zearalenone as a new

to the development of resistance by exerting a high selection pressure on the pathogen. Therefore it is important to apply fungicides only when they are necessary.

economic importance of Fusarium infections and difficulty in FHB prevention, it is necessary to acquire profound understanding of the action mode of both the pathogens and the protective measures. This issue requires further study on the use of innovative methods of control. From the perspective of modern agriculture, the ultimate goal of research on Fusarium, using molecular methods, is to reduce mycotoxins level in cereal grains and also to use engineering of crop plants for resistance infections to Fusarium and mycotoxins contamination.

> plant hormone. In: Lichtfouse E (ed) Sustainable agriculture reviews 3: sociology, organic farming, climate changing and soil science. Springer, Dordrecht, pp 419-437.

- Biesaga-Kościelniak J, Kościelniak J, Janeczko A: 2010.The impact of zearalenone and thidiazuron on indirect plant regeneration of oilseed rape and wheat. *Acta Physiologiae Plantarum* 32: 1047-1053.
- Blandino M, Minelli L, Reyneri A: 2006. Strategies for the chemical control of *Fusarium* head blight: effect on yield, alveographic parameters and deoxynivalenol contamination in winter wheat grain. *European Journal of Agronomy* 25: 193-201.
- Boyacioglu D, Hettiarachchy NS: 1995. Changes in some biochemical components of wheat grain that was infected with *Fusarium* graminearum. Journal of Cereal Science 21: 51-62.
- Brent KJ, Hollomon DW: 2007. Fungicide resistance in crop pathogens: How can it be managed? FRAC Monograph No. 2 (2nd ed., pp. 1-53). Brussels: Global Crop Protection Federation.
- Brown DW, McCormick SP, Alexander NJ, Proctor RH, Desjardins AE: 2001. A genetic and biochemical approach to study trichothecenes diversity in *Fusarium*

sporotrichioides and Fusarium graminearum. Fungal Genetics and Biology 32: 121-133.

- Bucci TJ, Howard PC: 1996. Effect of fumonisin mycotoxins in animals. *Toxin Reviews* 15: 293-302.
- Busman M, Desjardins AE, Proctor RH: 2012. Analysis of fumonisin contamination and the presence of *Fusarium* in wheat with kernel black point disease in the United States. *Food Additives and Contaminants Part* A 29: 1092-1100.
- Catalogue of Life. <u>http://www.catalogueoflife.org/col/brows</u> <u>e/tree/id/17093808</u>. Internet access: 10.02.2014.
- Chala A, Weinert J, Wolf GA: 2003. An integrated approach to the evaluation of the efficacy of fungicides against *Fusarium culmorum*, the cause of head blight of wheat. *Journal of Phytopathology* 151: 673-678.
- Champeil A, Doré T, Fourbet JF: 2004a.*Fusarium* head blight: epidemiological origin of the effects of cultural practices on head blight attacks and the production of mycotoxins by *Fusarium* in wheat grains. *Plant Science* 166: 1389-1415.
- Champeil A, Fourbet JF, Doré T, Rossignol L: 2004b. Influence of cropping system on *Fusarium* head blight and mycotoxins levels in winter wheat. *Crop Protection* 23:531-537.
- Chelkowski J, Gromadzka K, Stępień Ł, Lenc L, Kostecki M, Berthiller F: 2012. Fusarium species, zearalenone and deoxynivalenol content in preharvest scabby wheat heads from Poland. *World Mycotoxin Journal* 5: 133-141.
- Commission Regulation (EC) No. 466/2001of 8th March 2001setting maximum levels for certain contaminants in foodstuffs.
- Commission Regulation (EC) No. 856/2005 of 6th June 2005 amending Regulation (EC) No 466/2001 as regards *Fusarium* toxins.
- Commission Regulation (EC) No. 1126/2007 of 28th September 2007 amending Regulation (EC) No. 1881/2006 setting maximum levels for certain contaminants in foodstuffs as regards *Fusarium* toxins in maize and maize products.
- Coulombe RA: 1993. Symposium: Biological Action of Mycotoxins. *Journal of Dairy Science* 76: 880-891.

- Cromey MG, Lauren DR, Parkes RA, Sinclair KI, Shorter SC, Wallace AR: 2001. Control of *Fusarium* head blight of wheat with fungicides. *Australasian Plant Pathology* 30: 301-308.
- Dardis J, Walsh EJ: 2000. Studies of effectiveness of metconazole in controlling *Fusarium* head blight caused by *Fusarium culmorum* in spring wheat (*Triticum aestivum* L.). Cereal Research Communications 28: 443-448.
- Davidse LC: 1986. Benzimidazole fungicides: mechanism of action and biological impact. *Annual Review of Phytopathology* 24: 43-65.
- De Girolamo A, Pascale M, Visconti A: 2011. Comparison of methods and optimisation of the analysis of fumonisins B1 and B2 in masa flour, an alkaline cooked corn product. *Food Additives and Contaminants Part* A 28: 667-675.
- Desjardins AE, Proctor RH: 2007. Molecular biology of *Fusarium* mycotoxins. *International Journal of Food Microbiology* 119: 47-50.
- Desjardins AE, Proctor RH, Bai G, McCormick SP, Shaner G, Buechley G, Hohn TM: 1996. Reduced virulence of trichothecenenonproducing mutants of *Gibberella zeae* in wheat field tests. *Molecular Plant-Microbe Interactions* 9: 775-781.
- Dexter JE, Marchylo BA, Clear RM: 1997. Effect of *Fusarium* head blight on semolina milling and pasta making of durum wheat. *Cereal Chemistry* 74: 519-525.
- D'Mello JPF, Macdonald AMC, Briere L: 2000. Mycotoxin in a carbendazim-resistant strain of *Fusarium sporotrichioides*. *Mycotoxin Research* 16: 101-111.
- D'Mello JPF, Placinta CM, Macdonald AMC: 1999.*Fusarium* mycotoxins: a review of global implications for animal health, welfare and productivity. *Animal Feed Science and Technology*89:183-205.
- Doohan FM, Brennan J, Cooke BM: 2003. Influence of climatic factors on *Fusarium* species pathogenic to cereals. *European Journal of Plant Pathology* 109: 755-768.
- Dubos T, Pasquali M, Pogoda F, Hoffman L, Beyer M: 2011. Evidence for natural resistance towards trifloxystrobin in *Fusarium* graminearum. European Journal of Plant Pathology130: 239-248.

- Edwards SG: 2004. Influence of agricultural practices on *Fusarium* infection of cereals and subsequent contamination of grain by trichothecene mycotoxins. *Toxicology Letters* 153: 29-35.
- Edwards SG, O'callaghan J, Dobson ADW: 2002. PCR-based detection and quantification of mycotoxigenic fungi. *Mycological Research* 106: 1005-1025.
- Edwards SG, Pirgozliev SR, Hare MC, Jenkinson P: 2001. Quantification of trichotheceneproducing *Fusarium* species in harvested grain by competitive PCR to determine the efficacy of fungicides against *Fusarium* Head Blight of winter wheat. *Applied and Emvironmental Microbiology* 67: 1575-1580.
- Eudes F, Comeau A, Rioux S, Collin J: 2001. Impact of trichothecenes on Fusarium head blight (*Fusarium graminearum*) development in spring wheat (*Triticum aestivum*). Canadian Journal of Plant Pathology 23: 318-322.
- Fitzpatrick DW, Picken CA, Murphy LC, Buhr MM: 1989. Measurement of relative binding affinity of zearalenone, α-zearalenol and βzearalenol for uterine and oviduct estrogen receptors in swine, rats and chickens: an indicator of estrogenic potencies. *Comparative Biochemistry and Physiology* 94C: 691-694.
- Flannigan B: 1991. Mycotoxins. In: D'Mello JPF, Duffus CM, Duffus JH (Eds.), Toxic substances in crop plants, The royal society of chemistry, Cambrige, pp. 226-257.
- Francl LJ: 1998. Development of Fusarium head blight in relation to environment and inoculum, National Fusarium Head Blight Forum. Chapter 1. Epidemiology and disease management, Michigan State University, USA, 1-3.
- Gilbert J, Abramson D, Wong LD, Tekauz A: 1992. Studies of *Fusarium* head blight (*Fusarium* spp.) in Manitoba. *Hodowla* Roślin, Aklimatyzacja i Nasiennictwo 37 (3): 35-42.
- Gilbert J, Tekauz A: 2000. Recent developments in research on *Fusarium* head blight of wheat in Canada. *Canadian Journal of Plant Pathology* 22:1-8.
- Grabarkiewicz-Szczęsna J, Kostecki M, Goliński P, Kiecana I: 2001.Fusariotoxins in kernels of winter wheat cultivars field samples

collected during 1993 in Poland. *Nahrung* 45: 28-30.

- Gräfenhan T, Schroers H-J, Nirenberg HI, Seifert KA: 2011. An overview of the taxonomy, phylogeny, and typification of nectriaceous fungi in *Cosmospora, Acremonium, Fusarium, Stilbella*, and *Volutella. Studies in Mycology* 68: 79-113.
- Grishechkina LD, Volkova GV, Dolzhenko VI: 2012. Investigation of the effectiveness of fungicides for protecting cereals from *Fusarium* head blight. *Russian Agricultural Sciences* 38: 281-284.
- Gromadzka K, Waśkiewicz A, Chełkowski J, Goliński P: 2008.Zaeralenone and its metabolites: occurrence, detection, toxicity and guidelines. *World Mycotoxin Journal* 1: 209-220
- Harris LJ, Desjardins AE, Plattner RD, Nicholson P, Butler G, Young JC, Weston G, Proctor RH, Hohn TM: 1999. Possible role of trichothecene mycotoxins in virulence of *Fusarium graminearum* on maize. *Plant Disease* 83: 954-960.
- Henriksen B, Elen O: 2005. Natural *Fusarium* grain infection level in wheat, barley and oat after early application of fungicides and herbicides. *Journal of Phytopathology* 153: 214-220.
- Hestbjerg H, Felding G, Elmholt S: 2002. Fusarium culmorum infection of barley seedlings: Correlation between aggressiveness and deoxynivalenol content. Journal of Phytopathology 150: 308-316.
- Homdork S, Fehrmann H, Beck R: 2000. Effects of field application of tebuconazole on yield, yield components and the mycotoxin content of *Fusarium*-infected wheat grain. *Journal ofPhytopathology* 148: 1-6.
- Horsley RD, Pederson JD, Schwarz PB, McKay K, Hochhalter MR, McMullen MP: 2006. Integrated use of tebuconazole and *Fusarium* head blight-resistant barley genotypes. *Agronomy Journal* 98: 194-197.
- Hutcheon JA, Jordan VWL: 1990. Glasshouse evaluation of fungicides for control of *Fusarium spp.* on ears of winter wheat. *Annals of Applied Biology* 116: 50-51.
- Index Fungorum. http://www.indexfungorum.org/names/N

<u>amesRecord.asp?RecordID=8284</u>. Internet access: 10.02.2014.

- Infantino A, Santori A, Shah DA: 2011. Community structure of the *Fusarium* complex on wheat seed in Italy. *European Journal of Plant Pathology* 132: 499-510.
- Ioos R, Belhadj A, Menez M, Faure A: 2005. The effects of fungicides on *Fusariumspp*. and *Microdochium nivale* and their associated trichothecene mycotoxins in French naturally-infected cereal grains. *Crop Protection* 24: 894-902.
- Isaac S: 1999. What is the mode of action of fungicides and how do fungi develop resistance? In: *Mycology answers. Mycologist* 13: 38-39.
- Ittu M, Hagima I, Moraru I, Raducanu F: 1995.Reaction of some wheat and triticale genotypes to toxins, culture filtrates and cultures of *Fusarium*. In vivo screening and the relation between results obtained in vivo and in vitro. *Theoretical and Applied Genetics* 27: 1-13.
- Jaczewska-Kalicka A: 2010. Efekt ekonomiczny zwalczania chorób grzybowych pszenicy ozimej w latach 2000-2009. Świat Zbóż 14: 16-19.
- Jaczewska-Kalicka A, Krasiński T: 2011. Aspekty ekonomiczne i czynniki determinujące opłacalność ochrony pszenicy ozimej. *Progress in Plant Protection* 52: 565-569.
- Jennings P, Turner JA, Nicholson P: 2000. Overview of *Fusarium* ear blight in the UK – effect of fungicide treatment on disease control and mycotoxin production. In: Proceedings of The Brighton Crop Protection Conference – Pests and Diseases (pp 707-712) British Crop Protection Council, Farham, UK.
- Jones RK: 2000. Assessments of Fusarium head blight of wheat and barley in response to fungicide treatment. *Plant Disease* 84: 1021-1030.
- Kaneko I, Ishii H: 2009. Effect of azoxystrobin on activities of antioxidant enzymes and elternative oxidase in wheat head blight pathogens Fusarium graminearum and Microdochium nivale. *Journal of General Plant Pathology*75: 388-398.
- Kellerman TS, Marasas WFO, Thiel PG, Gelderblom WCA, Cawood M, Coetzer

JAW: 1990. Leukoencephalomalacia in two horses induced by oral dosing of fumonisin b1. Onderstepoort Journal of Veterinary57: 269-275.

PLANT SCIENCES

- Korbas M: 2004. Choroby podstawy źdźbła możliwości i perspektywy zwalczania. *Progress in Plant Protection*44: 147-154.
- Korbas M, Horoszkiewicz-Janka J: 2007. Znaczenie i możliwości ograniczenia szkodliwych metabolitów pochodzenia grzybowego. *Progress in Plant Protection* 47(2): 141-148.
- Krska R, Pettersson H, Josephs RD, Lemmens M, MacDonald S, Welzig E: 2003. Zearalenone in maize: stability testing and matrix characterization of a certified reference material. *Food Additives and Contaminants Part* A20: 1141-1152.
- Kuciel-Lisieska G, Obremski K, Stelmachów J, Gajęcka M, Zielonka Ł, Jakimiuk E, Gajęcki M: 2008.Presence of zearalenone in blood plasma in women with neoplastic lesions in the mammary gland. Bulletin of theVeterinary Institute in Pulany52: 671-674.
- Lancova K, Hajslova J, Poustka J, Krplova A, Zachariasova M, Dostalek P, Sachambula L: 2008. Transfer of *Fusarium*mycotoxins and 'masked' deoxynivalenol (deoxynivalenol-3-glucoside) from field barley through malt to beer. *Food Additives* and Contaminants Part A 25: 732-744.
- Leslie JF, Summerell BA: 2006. The *Fusarium* Laboratory Manual. Blackwell.,Oxford.
- Lienemann K, Meier A, Oerke EC, Steiner U, Dehne HW: 2002. Control of Fusarium head blight in wheat. In: Dehne H.W., Gisi U., Kuck K.H., Russel P.E., Lyr H. (Eds.), Modern fungicides and antifungal compounds III. 13th International Reinhardsbrunn Symposium (pp. 253-261), May 14-18, 2001 AgroConcept, Bonn.
- Lynch DV, Dunn TM: 2004. An introduction to plant sphingolipids and a review of recent advances in understanding their metabolism and function. *New Phytologist* 161: 677-702.
- Łukanowski A, Sadowski C: 2002. Occurence of *Fusarium* on grain and heads of winter wheat cultivatea in organic, integrated, conventional systems and monoculture. *Journal of Applied Genetics* 43: 73-82.
- Magan N, Hope R, Colleate A, Baxter ES: 2002. Relationship between growth and

mycotoxins production by *Fusarium* species, biocides and environment. *Journal of Plant Pathology* 108: 611-619.

- Mankevičienė A, Butkutė B, Gaurilčikienė I, Dabkevičius Z: 2011. Risk assessment of *Fusarium* mycotoxins in Lithuanian small cereal grains. *Food Control* 22: 970-976.
- Mashinini K, Dutton MF: 2006. The incidence of fungi and mycotoxins in South Africa wheat and wheat-based products. *Journal of Emvironmental Science and Health* 41: 285-296.
- Mateo EM, Valle-Algarra FM, Mateo R, Jiménez M, Magan N: 2011. Effect of fenpropimorph, prochloraz and tebuconazole on growth and production of T-2 and HT-2 toxins by *Fusarium langsethiae* in oat-based medium. *International Journal of Food Microbiology*151: 289-298.
- Matthies A, Buchenauer H: 2000. Effect of tebuconazole (Folicur) and prochloraz (Sportak) treatments on *Fusarium* head scab development, yield and deoxynivalenol (DON) content in grains of wheat following artificial inoculation with *Fusarium culmorum. Journal of Plant Disease and Protection* 107: 33-52.
- Matthies A, Walker F, Buchenauer H: 1999. Interference of selected fungicides, plant growth retardants as well as piperonyl butoxide and 1-aminobenzotriazole in trichothecene production of *Fusarium* graminearum (strain 4528) in vitro. Journal of Plant Disease and Protection 106: 198-212.
- Menniti AM, Pancaldi D, Maccaferri M, Casalini L: 2003. Effect of fungicides on *Fusarium* head blight and deoxynivalenol content in durum wheat grain. *European Journal of Plant Pathology* 109: 109-115.
- Mesterhàzy A, Bartók T, Lamper C: 2003. Influence of wheat cultivar, species of *Fusarium*, and isolate aggressiveness on the efficacy of fungicides for control of *Fusarium* head blight. *Plant Disease* 87: 1107-1115.
- Miedaner T, Reinbrecht C, Lauber U: 2001. Effects of genotype-environment interaction on deoxynivalenol accumulation and resistance to *Fusarium* head blight in rye, triticale and wheat. *Plant Breeding* 120: 97-105.
- Moretti AN: 2009. Taxonomy of *Fusarium* genus, a continuous fight between lumpers and

splitters. Zbornik Matice Srpske za Prirodne Nauke Novi Sad 117: 7-13.

- Moss M.O: 2002. Mycotoxin review 2. Fusarium. Mycologist 16: 158-161.
- Moss MO, Thrane U: 2004.*Fusarium* taxonomy with relation to trichothecene formation. *Toxicology Letters*153: 23-28.
- Müllenborn C, Steiner U, Ludwig M, Oerke E.C: 2008. Effect of fungicides on the complex of Fusarium species and saprophytic fungi colonizing wheat kernels. *European Journal of Plant Pathology* 120: 157-166.
- Nakajima T: 2010. Fungicides application against *Fusarium* head blight in wheat and barley for ensuring food safety. In: Fungicides, Odile Carisse (Ed.), InTech, pp. 139-156.
- Narkiewicz-Jodko M: 1998.Zdrowotność ziarna zbóż jako wskaźnik jego jakości. Zesz Nauk AR Wroclaw. Technologia żywności 12: 85-93.
- Narkiewicz-Jodko M, Gil Z, Urban M: 2005. Porażenie podstawy źdźbła pszenicy ozimej przez *Fusarium spp.* – przyczyny i skutki. *Acta Agrobototanica* 58: 319-328.
- Nelson PE, Dignani MC, Anaissie EJ: 1994. Taxonomy, biology and clinical aspects of *Fusarium* species. *Clinical Microbiology Reviews* 7: 479-504.
- Ngoko Z, Daoudou, Imele H, Kamga PT, Mendi S, Mwangi M, Bandyopadhyay R, Marasas WFO: 2008. Fungi and mycotoxins associated with food commodities in Cameroon.*Journal of Applied Biosciences* 6:164-168.
- Palacios SA, Ramirez ML, Zalazar MC, Farnochi MC, Zappacosta D, Chiacchiera SM, Reynoso MM, Chulze SN, Torres AM: 2011.Occurrence of *Fusarium spp.* and Fumonisin in Durum Wheat Grains. *Journal* of Agricultural and Food Chemistry 59: 12264– 12269.
- Parry DW, Jenkinson P, McLeod L: 1995. Fusarium ear blight (scab) in small grain cereals – a review. Plant Pathology 44: 207-238.
- Pfohl-Leszkowicz A, Chekir-Ghedira L, Bacha H: 1995.Genotoxicity of zearalenone, an estrogenic mycotoxin: DNA adduct formation in female mouse tissues. *Carcinogenesis* 16: 2315-2320.
- Pirgozliev SR, Edwards SG, Hare MC, Jenkinson P: 2002. Effect of dose rate of azoxystrobin and metconazole on the development of

Fusarium head blight and the accumulation of deoxynivalenol (DON) in wheat grain. *European Journal of Plant Pathology* 108: 469-478.

- Pirgozliev SR, Edwards SG, Hare MC, Jenkinson P: 2003. Strategies for the control of *Fusarium* head blight in cereals. *European Journal of Plant Pathology*109: 731-742.
- Placinta CM, D'Mello JPF, Macdonald AMC: 1999. A review of worldwide contamination of cereal grains and animal feed with *Fusarium* toxins. *Animal Feed Science and Technology* 78: 21-37.
- Pląskowska E: 2010. Charakterystyka i taksonomia grzybów z rodzaju *Fusarium*. *Mikologia Lekarska* 17: 177-176.
- Perkowski J, Stachowiak J, Kiecana I, Goliński P, Chelkowski J: 1997.Natural occurrence of *Fusarium* mycotoxins in Polish cereals. *Cereal Research Communications* 25: 379-380.
- Pławińska-Czarnak J, Zarzyńska J: 2010. Mikotoksyny w żywności pochodzenia zwierzęcego. *Mikologia Lekarska*17: 128-133.
- Prelusky DB, Rotter BA, Rotter RG: 1994. Toxycology of mycotoxins. In: Miller J.D. and Trenholm H.L. (Eds.), Mycotoxins in grain. Compounds other than aflatoxins. Eagan Press, Pt. Paul (MN) USA, pp. 359-404.
- Proctor RH, Desjardins AE, McCormick SP, Plattner RD, Alexander NJ, Brown DW: 2002. Genetic analysis of the role of trichothecene and fumonisin mycotoxins in the virulence of *Fusarium*. European Journal of Plant Pathology 108: 691-698.
- Ramirez ML, Chulze S, Magan N: 2004.Impact of environment al factors and fungicides on growth and deoxynivalenol production by *Fusarium graminearum* isolates from Argentinian wheat. *Crop Protection*23: 117-152.
- Reddy KRN, Salleh B, Saad B, Abbas HK, Abel CA, Shier WT: 2010. An overview of mycotoxin contamination in foods and its implications for human health. *Toxin Reviews*29: 3–26.
- Rheeder JP, Marasas WFO, Thiel PG, Sydenham EW, Shephard GS, van Schalwyk DJ: 1992.*Fusarium moniliforme* and fumonisins in corn in relation to human esophageal

cancer in Transkei. *Phytopathology* 82: 353-357.

- Riley RT, Wang E, Schroeder JJ, Smith ER, Plattner RD, Abbas H, Yoo H-S, Merrill AH: 1996. Evidence for disruption of sphingolipids metabolism as a contributory factor in the toxicity and carcinogenicity of fumonisins. *Natural Toxins* 4: 3-15.
- Rosada J, Dubas A, Bubniewicz P: 2010. Perspektywy agrotechnicznych metod ochrony roślin. *Progress in Plant Protection* 50: 1181-1187.
- Rotter BA, Prelusky DB, Pestka JJ: 1996. Toxicology of deoxynivalenol (vomitoxin). Journal of Toxicology Environmental and Health48: 1-34.
- Savard ME, Sinha RC, Seaman WL, Fedak G: 2000. Sequential distribution of the mycotoxin deoxynivalenol in wheat spikes after inoculation with *Fusarium graminearum*. *Canadian Journal of Plant Pathology* 22: 280-285.
- Sayler T: 1998. Study: \$ 2,6 bilion, 501 milion bushels lost to scab 1991-96. Prairie grains 11.
- Schneweis I, Meyer K, Engelhardt G, Bauer J: 2002. Occurrence of zearalenone-4-Dglucopyranoside in wheat. *Journal of Agricultural and Food Chemistry* 50: 1736-1738.
- Scott PM: 2012. Recent research of fumonisins: a review. Food Additives and Contaminants Part A 29: 242-248.
- Sikora H, Banachowska J: 2006. Wrażliwość grzyba Fusarium culmorum na substancje aktywne wybranych fungicydów. Progress in Plant Protection 46: 601-604.
- Simpson DR, Weston GE, Turner JA, Jennings P, Nicholson P: 2001. Differential control of head blight pathogens of wheat by fungicides and consequences for mycotoxin contamination of grain. *European Journal of PlantPathology*, 107: 421-432.
- Sinha RC, Savard ME: 1997. Concentration of deoxynivalenol in single kernels and various tissues of wheat heads. *Canadian Journal of Plant Pathology* 19: 8-12.
- Siranidou E, Buchenauer H: 2001. Chemical control of *Fusarium* head blight on wheat. *Journal of Plant Disease and Protection*108: 231-243.

- Snijders CHA, Perkowski J: 1990. Effects of head blight caused by *Fusarium culmorum* on toxin content and weight of wheat kernels. *Phytopathology* 80: 566-570.
- Stanković S, Lević J, Ivanović D, Krnjaja V, Stanković G, Tančić S: 2012. Fumonisin B₁ and its co-occurrence with other fusariotoxins in naturally-contaminated wheat grain. *Food Control* 23: 384-388.
- Statistical Yearbook of Agriculture and Rural Areas, 2007, GUS, Warszawa, 473 ss.
- Steffens JJ, Pell EJ, Tien M: 1996. Mechanisms of fungicide resistance in phytopathogenic fungi. *Current Opinion in Biotechnology* 7: 348-355.
- Stępień Ł, Chełkowski J: 2010. Fusarium head blight of wheat: pathogenic species and their mycotoxins. World Mycotoxin Journal 3: 107-119.
- Summerell BA, Salleh B, Leslie JF: 2003. A utilitarian approach to *Fusarium* identification. *Plant Disease* 87: 117-128.
- Sutton JC: 1982. Epidemiology of wheat head blight and maize ear rot caused by *Fusarium* graminearum. Canadian Journal of Plant Pathology 4: 195-209.
- Szempliński W: 2012.Rośliny rolnicze, Wydawnictwo UWM, Olsztyn, 69-80.
- Torriani SFF, Brunner PC, McDonald BA, Sierotzki H: 2009. QoI resistance emerged independently at least 4 times in European populations of *Mycosphaerella graminicola*. *Pests Management Science* 65: 155-162.
- Tratwal A, Walczak F: 2012. Występowanie ważnych gospodarczo chorób pszenicy ozimej w Polsce w latach 2006-2010. *Annales Universitatis Mariae Curie-Skłodowska* 67: 28-41.
- Vendl O, Berthiller F, Crews C, Krska R: 2009. Simultaneous determination of deoxynivalenol, zearalenone, and their major masked metabolites in cereal-based food by LC-MS-MS. *Analytical and Bioanalytical Chemistry* 395: 1347-1354.
- Visconti A, Minervini F, Lucivero G, Gambatesa V: 1991. Cytotoxic and immunotoxic effects of *Fusarium* mycotoxins using a rapid colorimetric bioassay. *Mycopathologia* 113: 181-186.
- Wagacha JM, Muthomi JW: 2007. Fusarium culmorum: Infection process, mechanisms of

mycotoxin production and their role in pathogenesis in wheat. *Crop Protection* 26: 877-885.

- Xu X-M, Nicholson P, Thomsett MA, Simpson D, Cooke BM, Doohan FM, Brennan J, Monaghan S, Moretti A,Mule G, Hornok L, Beki E, Tatnell J,Ritieni A, Edwards SG: 2008. Relationship between the fungal complex causing *Fusarium* head blight of wheat and environmental conditions. *Phytopathology* 98: 69-78.
- Ypema HL, Gold RE: 1999. Kresoxim-methyl, modification of naturally occurring compound to produce a new fungicide. *Plant Disease* 83: 4-19.