Chapter 10
Fusarium Diseases of Canadian Grain Crops: Impact and Disease Management Strategies

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10.1 *Fusarium* Species Classification and Genetics

Species of the genus *Fusarium* are ascomycetes that are characterized by their typical conidia, which are often fusiform to sickle-shaped with an elongated apical cell and pedicellate basal (foot) cell. Several important *Fusarium* species, including *F. avenaceum*, *F. graminearum* and *F. pseudograminearum*, are known to produce a teleomorph state that was formerly classified in the genus *Gibberella*. In other species, such as *F. culmorum*, *F. oxysporum* and *F. sporotrichioides*, no teleomorph has been reported, so far. Geiser et al. (2013) proposed to recognize the genus *Fusarium* as the sole name for a group that includes virtually all important saprophytic, plant pathogenic, and mycotoxigenic species. *Fusarium* spp. can be identified by morphological features and also by genetic analysis. Morphological species identification can be based on microscopic and/or macroscopic characters, such as conidia, phialides, chlamydospores, ascospores and colony characteristics of pure cultures (see Figs. 10.1 and 10.2). Macroconidia, a form of asexual spores most often aggregated to sporodochia, are usually 'banana'-shaped in *Fusarium* species, which can also produce microconidia in the aerial mycelium and/or chlamydospores in hyphae. The size, shape and number of septa in the macroconidia are often used to differentiate

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A. Goyal and C. Manoharachary (eds.), *Future Challenges in Crop Protection Against Fungal Pathogens*, Fungal Biology, DOI 10.1007/978-1-4939-1188-2_10,

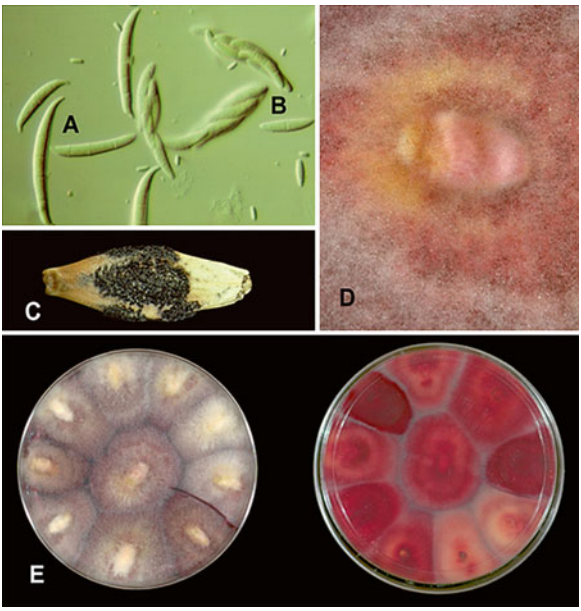

Fig. 10.1 *Fusarium graminearum*: (**a**) Conidia of a culture grown on potato dextrose agar (PDA); (**b**) asci and ascospores of the teleomorph; (**c**) purplish-black perithecia (teleomorph) on barley seed; (**d**) close-up of typically red mycelia with a yellow tint; (**e**) colonies growing from wheat seed as seen from above (*left*) and below (*right*)

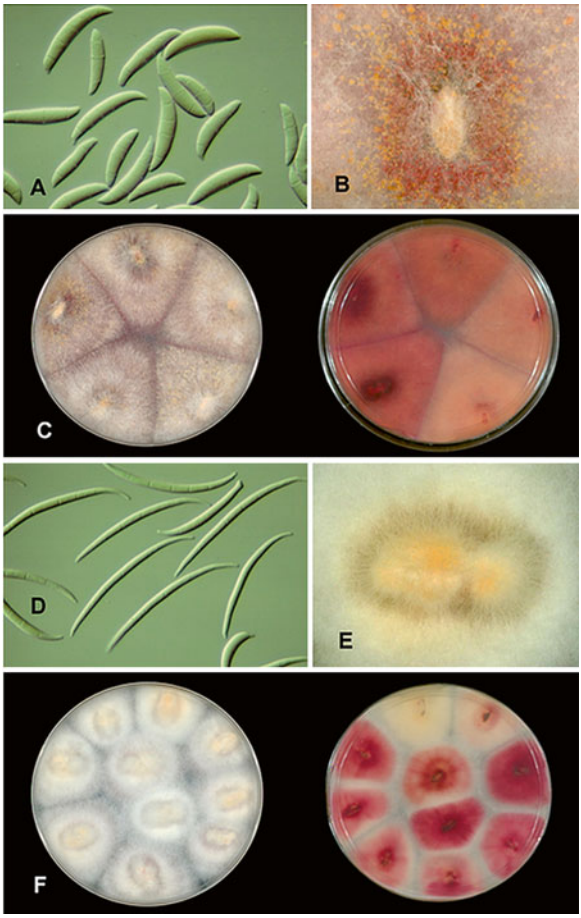

Fig. 10.2 *Fusarium culmorum*: (**a**) short and broad conidia of a culture grown on PDA; (**b**) close-up of loose mycelium with abundant orange and red sporodochia; (**c**) fast growing colonies from wheat seed as seen from above (*left*) and below (*right*). *Fusarium avenaceum*: (**d**) long and slender conidia of a culture grown on PDA; (**e**) close-up of dense white mycelium predominantly without sporodochia; (**f**) colonies often with white margin growing from durum seed as seen from above (*left*) and below (*right*)

between species (Leslie and Summerell 2006). The teleomorph state is characterized by black, flask-shaped perithecia with a single ostiole (narrow opening) at the top, from which the asci (containing the ascospores) are released. The asci of *F. graminearum* (formerly *Gibberella zeae*) have been shown to be forcibly ejected through the ostiole under conditions of high humidity (Trail et al. 2002).

Genetic analysis is a more accurate approach for species identification and is enhanced by an increase in available *Fusarium* genome sequences. The genomes of several important *Fusarium* species such as *F. graminearum*, *F. oxysporum*, *F. pseudograminearum*, *F. solani* and *F. verticillioides* are publicly available through various data portals (e.g. Broad Institute of Harvard and MIT; DOE Joint Genome Institute; NCBI GenBank). The *F. graminearum* strain Ph1 was the first complete *Fusarium* genome to be published and annotated (Cuomo et al. 2007). The *F. graminearum* genome is 36 megabases (Mb) with over 13,000 genes. A comparison of the Ph1 strain with the partially completed genome of *F. graminearum* strain GZ3639 revealed over 10,000 single-nucleotide polymorphisms found along all four chromosomes (Cuomo et al. 2007). The complete genome sequence and assembly of *F. graminearum* GZ3639 and seven other *F. graminearum* strains were recently prepared by R. Subramaniam and colleagues (personal communications). *F. graminearum* has a small number of chromosomes compared to other *Fusarium* spp., and this is believed to be a result of ancestral chromosome fusion (Cuomo et al. 2007). *F. verticillioides* strain 7600 and *F. oxysporum* f. sp. *lycopersici* strain 4287 were sequenced and compared to *F. graminearum* Ph1 by Ma et al. (2010). *F. verticillioides* is 42 Mb with over 14,000 genes found on 11 chromosomes. *F. oxysporum* f. sp. *lycopersici* genome is 60 Mb, the largest of the three genomes, with over 17,000 genes encoded on a total of 15 chromosomes. Four of the 15 chromosomes are lineage specific and are composed primarily of transposable elements. Ma et al. (2010) also observed that the lineage specific regions differ in sequence among different formae speciales of *F. oxysporum*, and some of these regions encode virulence factors specific to the host range of a given strain. They were able to demonstrate that chromosome 14, which encodes host-specific virulence factors, is able to undergo horizontal transfer between different *F. oxysporum* f. sp., and that this transfer leads to a change in host-specificity. *F. solani* (*N. haematococca* MPVI isolate 77-13-4) has also been sequenced and has 17 chromosomes with a genome size of 54.43 Mb. Of the 17 chromosomes, three (14, 15 and 17) are non-essential, and at least one of these (chromosome 14) is involved in host-specificity (Coleman et al. 2009).

A subset of *Fusarium* spp. produces a series of mycotoxins, including trichothecenes and fumonisins. Both of these classes of toxins are secondary metabolites and their production is controlled by a specific set of genes found in clusters on the genome. Fumonisins are produced by a number of *Fusarium* spp., including *F. verticillioides*, an important pathogen of maize. Over 28 fumonisins have been identified to date, and are divided into four groups (Rheeder et al. 2002): A-, B-, C- and P-series. Fumonisins are composed of a 19- to 20-carbon aminopolyhydroxyalkyl backbone, similar in structure to sphingosine (Shier 1992). The B-series is the most abundant of the four series (where fumonisin B_1 is the major fumonisin found in *Fusarium*-infected grain) and forms the basic fumonisin structure.

The A-series is characterized by the acetylation of the amino group (Abbax et al. 1993); members of the C-series do not have a terminal methyl group (Branham and Plattner 1993); members of the P-series have a 3-hydroxypyridinium in place of the carbon-2 amine (Musser et al. 1996). Fumonisin toxicity is related to its ability to disrupt sphingolipid metabolism through inhibition of ceramide synthase activity (Voss et al. 2007). These toxins have been shown to affect mitochondrial respiration (Domijan and Abramov 2011) and have been associated with various cancers in humans and animals (Gelderblom et al. 1988; Müller et al. 2012). The fumonisin biosynthetic genes are referred to as FUM genes and are found in the FUM cluster (Proctor et al. 2003, 2006).

Trichothecenes are potent inhibitors of eukaryotic protein biosynthesis and are expressed by *Fusarium* pathogens that affect cereal crops, including *F. culmorum*, *F. graminearum* and *F. sporotrichioides*. Over 200 trichothecenes have been identified from a variety of fungal species (Cole and Cox 1981; Schollenberger et al. 2007). The trichothecenes are divided into four groups based on specific structural features (reviewed in Shank et al. 2011): Types A, B, C and D. Trichothecene-producing *Fusarium* spp. produce either Type A trichothecenes (such as T-2 toxin and HT-2 toxin), or Type B trichothecene (such as nivalenol (NIV), 4-deoxynivalenol (DON; also known as vomitoxin) and acetylated derivatives). The genes encoding trichothecene biosynthesis and metabolism (*TRI* genes) are mainly found in the TRI cluster (Hohn et al. 1993; Ward et al. 2002; Brown et al. 2004), and the specific trichothecenes produced by a given species are determined by the sequences of the TRI genes within this cluster. A summary of TRI genes and their functions are reviewed in Foroud and Eudes (2009). Sequence differences among specific TRI genes, which define their trichothecene genotype (Desjardins 2008), have been used to predict the trichothecene chemotype of a given *Fusarium* strain (Lee et al. 2001; Ward et al. 2008; Alexander et al. 2011; Boutigny et al. 2011; Reynoso et al. 2011). For example, NIV chemotypes are determined by the presence of functional sequences of the Tri13 and Tri7 genes for NIV and 4-acetylnivalenol production, respectively (Lee et al. 2002; Kim et al. 2003). DON producers, which do not express functional Tri13/Tri7 genes, are divided into two chemotypes (3-acetyldeoxynivalenol (3-ADON) and 15-acetyldeoxynivalenol (15-ADON)) determined by the esterase specificity encoded in the *TRI8* gene (Alexander et al. 2011).

From the *Fusarium* TRI cluster, DNA sequences of the TRI12 gene encoding a trichothecene efflux pump have been used to develop real-time PCR assays for quantitative diagnostics of the 3ADON, 15ADON and NIV genotypes of *F. graminearum* and *F. culmorum* (Kulik 2011; Nielsen et al. 2012). These assays utilize genetic markers directly involved in the production of trichothecenes and provide a powerful, cost effective tool to monitor the genotype composition and shifts in pathogen populations of mycotoxigenic *Fusarium* species. Over the past decade, a number of quantitative assays for the detection and quantification of other toxin producing *Fusarium* species have been published (reviewed by Morcia et al. 2013). Applications of these quantitative diagnostic tools included traceability studies of different *Fusarium* species on small cereals (Waalwijk et al. 2004, 2008; Yli-Mattila et al. 2006; Fredlund et al. 2010, 2013; Lindblad et al. 2013), grain dust (Halstensen et al. 2006), and along wheat processing chains (Terzi et al. 2007; Tittlemier et al. 2014).

For the development of species-specific detection assays, DNA sequences of barcode regions, including genes encoding the translation elongation factor 1α, β-tubulin, or mating type (MAT) were successfully used for the detection and quantification of *Fusarium* species (Nicolaisen et al. 2009; Demeke et al. 2010). In *Fusarium*, however, universal barcode markers, such as the internal transcribed spacer (ITS) regions of the ribosomal DNA and the cytochrome oxidase 1 (COX1) gene, commonly used for identification of other groups of fungi have been reported to be non-orthologous and paralogous, respectively (O'Donnell and Cigelnik 1997; Gilmore et al. 2009). Multiple copies of these barcode regions are present in *Fusarium* genomes, showing a rather low degree of divergence among homologous sequences with a number of closely related species sharing identical sequences. In metagenomic profiling of microbial communities employing next generation sequencing (NGS), universal barcode markers often provide insufficient resolution at or below species level when used for identification and semi-quantification of plant-associated *Fusarium* species. Other universal barcodes, such as the protein coding chaperonin-60 (*cpn*60) gene have been reported to be a robust target for species-level characterization in bacteria (Links et al. 2012). The potential of *cpn*60 to identify and differentiate species of *Fusarium* is currently being explored by the authors.

Studies on population dynamics based on single-nucleotide polymorphisms (SNP) have proven to provide sufficient resolution for population- and individual-level analyses of *Fusarium* species. They are adaptable to high-throughput DNA chip-based methods, but finding markers to characterize and distinguish populations is often problematic. Multilocus genotyping (MLGT) assays are a powerful tool that can facilitate accurate identification of species and trichothecene chemotype for large numbers of *Fusarium* isolates. Population dynamics behind adaptive shifts observed for mycotoxin chemotypes and newly emerging pathotypes in *Fusarium graminearum* were studied employing multiplex PCR assays to enable simultaneous determination of species identity and trichothecene chemotype (Ward et al. 2008; Gale et al. 2011). For other population studies (Suga et al. 2004), the genome of *Fusarium graminearum* was mined for repeat sequences to analyze genotypes based on variable number of tandem repeats (VNTR). A number of VNTR markers were selected based on length polymorphisms and used to analyze population genetics in *F. graminearum* and closely related taxa (Ward et al. 2008; Gale et al. 2011; Zhang et al. 2012).

10.2 Fusarium Diseases of Pulse Crops

10.2.1 Pathogens

Canada is a major world exporter of pulse crops. In particular, lentil (*Lens culinaris*) and field pea (*Pisum sativum*) production in Canada has been rising due to the benefits of crop diversification, nitrogen inputs into the soil, and increased

worldwide demand for pulse crops (Graham and Vance 2003). In 2012, 2.5 million hectares of pulse crops were planted in Canada, with the majority planted to lentil (1.0 million hectares), and peas (1.35 million hectares) (Statistics Canada). Dry beans (*Phaseolus vulgaris*) and chickpeas (*Cicer arietinum*) comprise the remainder of pulse crops with 120,000 and 80,000 hectares planted, respectively, in 2012. The goal of the Canadian pulse industry is to realize an increase of pulse acreage to 15 % of total planted area. Currently, pulse acreage accounts for an average of 5–10 % total crop area over the three main pulse-growing provinces of Manitoba, Saskatchewan and Alberta.

As pulse acreage increases, the prevalence and incidence of root rots, caused by *Fusarium* spp., have also been increasing. Most pulse crops are subject to root rot pathogens that build up in the soil over several years and reduce plant stands and yields (Persson et al. 1997; Bailey et al. 2003; Infantino et al. 2006; Naseri and Marefat 2011). Root rot is a general term that describes disease symptoms which include reddish-brown-black lesions on the hypocotyl and tap root, often accompanied by vascular discolouration, foliar chlorosis and wilt (Agrios 1997; Bailey et al. 2003; Infantino et al. 2006). Yield losses of 10–30 % are commonly observed in pulse crops affected by moderate to severe root rot, but yield loss potential can be even higher under favourable environmental conditions (Oyarzun 1993; Schneider et al. 2001; Schwartz et al. 2005; Cichy et al. 2007). Root rots, causing wilt and death of mature plants, are reported throughout pulse-growing regions in Canada, and experienced growers are increasingly challenged with yield loss due to stand death. Annual disease surveys indicate that root rot incidence is now widespread in most pulse-growing regions of Canada.

Root rots can be caused by a number of fungi, including *Pythium*, *Rhizoctonia* and/or *Fusarium* spp. (Bailey et al. 2003). However, Fusarium root rot is considered the most prevalent root disease in field peas, dry beans and lentils (Henriquez et al. 2012b; McLaren et al. 2012; Miller et al. 2012). In recent years, 80–100 % of pea fields surveyed in Saskatchewan and Manitoba had plants with root rot symptoms, with severity usually occurring at a moderate level, or 30–40 % of roots and lower stem with symptoms (McLaren et al. 2010, 2011, 2012; Dokken-Bouchard et al. 2011). *Fusarium solani* f. sp. *pisi* and *F. avenaceum* (teleomorph formerly *Giberella avenacea*) are the most prevalent pathogens in field pea crops, with *F. avenaceum* becoming the predominant species isolated from rotted roots in recent years (McLaren et al. 2012). *Fusarium avenaceum* is a common soil saprophyte in temperate regions, and has traditionally been associated with crown rot and Fusarium head blight (FHB) of cereals (Leslie and Summerell 2006; Fernandez 2009). However, it is also very aggressive on all pulse crops, including lentils, dry bean and field peas, and is now the principal species associated with root rot of field pea across the prairie region (Feng et al. 2010; Chittem et al. 2012). The '*F. solani* species complex' comprises over 50 phylogenetic species, of which many members are common soil-dwelling fungi, and act as saprophytes and/or plant pathogens (Coleman et al. 2009). Pathogenic isolates in this group are further characterized by formae speciales to indicate the specific host plants to which they are restricted, such that *F. solani* f. sp. *pisi* is only pathogenic to pea, and *F. solani* f. sp. *phaseoli* is only pathogenic to beans (Oyarzun et al. 1993).

F. solani f. sp. *phaseoli* is generally considered to be the most common pathogen causing dry bean root rots, but recent surveys conducted in Manitoba indicate that there is a complex of *Fusarium* spp. associated with root rot symptoms (Henriquez et al. 2012a). Lentil root rot can also be caused by a number of different pathogenic species including *F. avenaceum*, *F. acuminatum* and *F. redolens* (Hwang et al. 1994; Bailey et al. 2000; Esmaeili Taheri et al. 2011). The vascular wilt pathogens *F. oxysporum* ff. spp. *pisi* and f. sp. *phaseoli* are frequently found in pea and dry bean fields, respectively, but are generally associated with low disease severity indexes (Henriquez et al. 2012b; McLaren et al. 2012). *Fusarium graminearum*, the primary causal agent of FHB in Canada, has also been associated with root rot of legumes (Chongo et al. 2001; Goswami et al. 2008; Bilgi et al. 2011; Esmaeili Taheri et al. 2011; Henriquez et al. 2012a). *Fusarium redolens*, a *Fusarium* species closely related to *F. oxysporum* (Bogale et al. 2007), is also frequently isolated from diseased roots of pulse crops, including field pea, lentil and chickpea (Esmaeili Taheri et al. 2011; Jiménez-Fernández et al. 2011). *Fusarium redolens* has a broad-host range, and has also been shown to induce root rot symptoms in durum wheat in Saskatchewan (Esmaeili Taheri et al. 2011).

10.2.2 Infection Pathways and Symptoms

Most of the *Fusarium* spp. capable of causing root rots on pulse crops produce the same disease symptoms, making it difficult to distinguish between causal agents (Hwang et al. 1995, 2000; Bailey et al. 2000; Bilgi et al. 2008; Feng et al. 2010). Symptoms first appear as small reddish-brown lesions at the base of the hypocotyl and taproot (Fig. 10.3a) (Stahl et al. 1994; Schwartz et al. 2005). As the disease advances, lesions coalesce to form large necrotic areas which encircle the stem and expand vertically (Fig. 10.3c) (Bailey et al. 2003). A reduction in root mass also becomes evident at this stage. In the final stages of root rots, root mass will be reduced by 80–100 %, the hypocotyl becomes pithy and lesions can extend vertically upwards of 2 cm (Bilgi et al. 2008). At this point, the plant is functionally dead with obvious signs of yellowing, wilting and collapse. Infection with *F. solani* and *F. avenaceum* also causes red or brown streaking of the vascular system, indicating that these pathogens can enter the xylem (Fig. 10.3b) (Bailey et al. 2000, 2003; Feng et al. 2010). A major impact of root-rotting fungi on pulse crops is the reduction in the number of nodules on the roots, primarily because secondary root growth is severely impacted (Hwang et al. 1994, 1995). This then results in a reduction in nitrogen fixation, thus reducing the benefit of pulses to subsequent crops in a rotation.

Infection pathways of *F. solani* f. sp. *pisi* on field pea and *F. solani* f. sp. *phaseoli* on dry bean have been well characterized. *Fusarium solani* survives in crop residues and in soil as chlamydospores, which serve as the primary inoculum source (Leslie and Summerell 2006). Chlamydospores are produced in infected tissues of host crops at the end of the growing season, and can survive in the soil for extended periods of time (Bailey et al. 2003; Schwartz et al. 2005). Germination of chlamydospores are stimulated by the presence of nutrients exuded from germinating

Fig. 10.3 Symptoms of root rot on field pea: (**a**) early symptoms with small brown lesions at point of seed attachment; (**b**) red streaking of the vascular system characteristic of *Fusarium* infection; (**c**) extended brown/black lesions on tap root, and loss of secondary root mass and root nodules

seeds of host crops, and thus use of rotations with non-host crops is essential to reduce survival of chlamydospores (Mondal et al. 1996; Oyarzun et al. 1998). Chlamydospores germinate to produce hyphae which can directly infect the developing hypocotyl and epicotyl of seedlings, or the hyphae produces macroconidia which then infect the seedling (Nelson 2004). In vitro studies have shown that macroconidia of *F. solani* invade root tissues by primarily colonizing the zone of elongation. The remainder of the root zones appear to be resistant to primary infection, even in the presence of large numbers of macroconidia and fungal mycelia (Gunawardena et al. 2005). However, Stahl et al. (1994) describe direct penetration of the epidermis of the epicotyl. Production of cutinases by *F. solani* f. sp. *pisi* has also been implicated in initial infection and penetration of the cuticle barrier (Li et al. 2002; Hadwiger 2008). After penetrating the epidermis, mycelium then advances through the cortex both inter- and intra-cellularly until it reaches the Casparian strips present in the endodermis of epicotyl stems. At this point, degradation of the vascular parenchyma is visible in advance of the invading hyphae, suggesting that cell wall degrading enzymes aid in breaking down the barrier to the vascular system, and resulting in colonization of the vascular bundles. Studies of *F. solani* on peas have shown that this pathogen will exclusively colonize the xylem stem tissues beyond the epicotyl, while external lesions on the stem abruptly stop on the epicotyl 1–2 cm above ground (Stahl et al. 1994). It is unknown whether the other root-rotting fungi, such as *F. avenaceum* and *F. graminearum*, colonize and infect tissues of all host pulse crops in a similar manner.

Unlike the other Fusarium root rot pathogens of pulses, *F. avenaceum* is unable to produce chlamydospores, and thus survives in crop residues of susceptible host crops (Leslie and Summerell 2006). Modern agronomic practices, such as reduced

tillage, increased glyphosate use and crop rotation with susceptible hosts, have likely allowed pathogenic *Fusarium* spp. to accumulate to damaging levels (Fernandez et al. 2008, 2009, 2011). The increasing prevalence of *F. avenaceum* associated with both broad-leaf pulse and cereal crops in Saskatchewan suggests that *Fusarium* inoculum is being maintained or even increasing on residues of these host crops (Bailey et al. 2001; Fernandez 2007; Abdellatif et al. 2010; Feng et al. 2010). *Fusarium avenaceum* isolates display genetic and ecological plasticity, allowing this fungus to occupy several ecological niches, such as root tissues of pulses, head and root tissues of cereals and residues of host crops (Abdellatif et al. 2010). *Fusarium avenaceum* survived in colonized stem bases of winter wheat over a period of 10 months in the Netherlands (Köhl et al. 2007) with DNA levels decreasing by only 50 % over the winter months. *F. graminearum* survived in standing wheat stubble for up to 20 months, and provided sufficient inoculum levels to serve as a primary inoculum for subsequent crops (Hogg et al. 2010).

10.2.3 Resistance Mechanisms

No commercial cultivars of field pea, lentil or dry beans are completely resistant to Fusarium root rot (Bailey et al. 2003; Grünwald et al. 2003; Xue 2003). Partial resistance to Fusarium root rot caused by both *F. solani* and *F. avenaceum* has been reported in one commercial cultivar, 'Franklin' (Chittem et al. 2012). The mechanism of resistance is not known, although generally genotypes of dry beans and field peas with large, robust root systems show better resistance than those with small root systems (Kraft and Boge 2001; Cichy et al. 2007). Partial resistance is present in several field pea accessions, but these have not yet been transferred into lines with desirable commercial attributes (Grünwald et al. 2003). Quantitative trait locus/loci (QTL) that confer partial resistance to *F. solani* and *F. avenaceum* have been described in field pea, however these QTL did not account for 60 % and 80 %, respectively, of the observed phenotypic variation in root rot resistance (Feng et al. 2011; Li et al. 2012). This suggests that additional resistance genes or QTL are associated with root rot resistance (Feng et al. 2011; Li et al. 2012).

In general, large-seeded Andean dry beans (e.g. kidney beans) tend to be more susceptible to Fusarium root rot than the small-seeded Mesoamerican type beans (e.g. black beans) (Bilgi et al. 2008). Cultivars with partial resistance to *F. solani* f. sp. *phaseoli* also appear to have resistance to other Fusarium root rot pathogens, such as *F. graminearum* (Bilgi et al. 2011). Similar to the situation in field peas, QTL have been identified in dry bean from bean lines with different root rot resistance sources, but these QTL generally account for a small proportion of root rot variation (Schneider et al. 2001; Román-Avilés and Kelly 2005; Ronquillo-López et al. 2010). Most of these QTL are present in regions of the bean genome where resistance genes, such as pathogenesis-related proteins (*PVPR-2*), polygalacturonase-inhibiting protein (*Pgip*) and chalcone synthase (*ChS*) are located (Schneider et al. 2001). This would indicate that partial physiological resistance to Fusarium root rot is associated with generalized host defense responses that are induced upon host attack

(Schneider et al. 2001; Román-Avilés and Kelly 2005). However, markers associated with field root rot resistance often do not correlate with greenhouse root rot screening experiments (Román-Avilés and Kelly 2005). This lack of association suggests that environmental variation is the most important factor contributing to disease development and resistance responses to Fusarium root rot. As a result, breeding for resistance and elucidation of genetic resistance to Fusarium root rots has been challenging, and limited progress has been made in identifying sources of genetic resistance (Singh and Schwartz 2010).

The pea-*F. solani* interaction has been studied as a model system to understand the biochemical and molecular components of non-host resistance by comparing the difference in responses of pea to infection with *F. solani* ff. spp. *pisi* and *phaseoli*. This topic has been reviewed extensively in a recent article (Hadwiger 2008), and thus will not be reviewed again here.

10.3 *Fusarium* Diseases of Cereals

10.3.1 Pathogens and Associated Mycotoxins

The two main *Fusarium* diseases of cereal crops are FHB and Fusarium crown rot (FCR), both of which have been observed in wheat, barley, rye, oats and triticale. FHB is reviewed here in greater detail since it is the main *Fusarium* disease of cereal crops in Canada and worldwide. *Fusarium culmorum*, *F. graminearum* and *F. pseudograminearum* (teleomorph aka *Gibberella coronicola*) are the major species responsible for FHB and/or FCR (O'Donnell et al. 2000; Liddell 2003; Backhouse et al. 2004; Smiley et al. 2005; Tóth et al. 2005); although, *F. pseudograminearum*, primarily responsible for FCR, is only found occasionally in Canada, as this species prefers warmer and drier climates. The majority of *Fusarium* spp. involved in FHB and FCR produce mycotoxins belonging to the trichothecene class, although other *Fusarium* mycotoxins including fumonisins, moniliformin (MON) and zearalenone (ZEA), have also been found in FHB-infected cereals worldwide (Golrnski et al. 1996; Palacios et al. 2011). Trichothecenes contaminate the kernels of FHB-infected spikes, and can also accumulate in the kernels of FCR-infected cereals when the fungus moves up the stem and into the spike (Mudge et al. 2006). Trichothecenes are harmful for human and animal consumers (Eriksen and Pettersson 2004; Godfray et al. 2010), and also interfere with downstream processing including malting (Wolf-Hall 2007). Various cytotoxic effects of trichothecenes have been observed in mammalian and plant systems (Ueno 1983; Rocha et al. 2005; Pestka 2010; Arunachalam and Doohan 2013), although inhibition of eukaryotic protein synthesis machinery is the main mechanism of toxicity (Ueno et al. 1968; McLaughlin et al. 1977). Consumption of contaminated grain can lead to a condition known as alimentary toxic aleukia (ATA), where symptoms of ingestion include gastroenteritis, abdominal and oesophageal pain, ataxia, dyspenia, and subcutaneous haemorrhaging (Lutsky et al. 1978; Peraica et al. 1999). The main potential source of trichothecene contamination of food is from FHB-infected cereals, and

DON is the main trichothecene detected in grain. For this reason, limits are in place to manage DON in food and feed, as described in Sect. 10.6.

The *Fusarium* spp. belonging to the *F. graminearum* (*Fg*) complex, responsible for FHB in North America, are Type B trichothecene producers (Ward et al. 2002; Starkey et al. 2007). Historically, *Fg* populations dominated by 15-ADON chemotypes were responsible for FHB in North America. Some 3-ADON producers were identified more frequently on the continent over 10 years ago, and have since been replacing the 15-ADON populations (Ward et al. 2008). The 3-ADON producers were shown to be the predominant genotype representing more than 90 % of the *Fg* populations in the Canadian Maritimes (R. Clear, unpublished). Since the 1990s, the 3-ADON populations have been moving from the Red River valley in Manitoba to eastern Saskatchewan, and currently represent up to 60 % of the *Fg* population in central Alberta. The 3-ADON producers tend to be more aggressive (Foroud et al. 2012a) and produce higher levels of toxins both in culture and in planta (Ward et al. 2008; Puri and Zhong 2010; von der Ohe et al. 2010; Yli-Mattila and Gagkaeva 2010; Foroud et al. 2012a; Clear et al. 2013). The other important Type B trichothecene producing species, *F. culmorum* (*Fc*), can be associated with FHB and FCR of cereals. Its distribution in western Canada appears to depend partly on environmental factors. Especially in cooler and wet years, *F. culmorum* can be more frequently detected (Fig. 10.4) and contribute significantly to DON contamination in cereal grains (Clear et al. 1993). Similarly to *F. graminearum*, the 3-ADON chemotype of *F. culmorum* is reported to be the more aggressive and toxigenic genotype (Miedaner et al. 2004). In Canada, the 3-ADON genotype represents 100 % of the *Fc* populations found on cereals.

While DON producers are the main species in North America, other chemotypes have also been identified in cereal crops. NIV producers, for example, encompass 79 % of the *F. graminearum* strains identified in Louisiana (Gale et al. 2011), although, this is not representative of the entire population in the United States. In Canada, the NIV chemotypes represent less than 1 % of the *F. graminearum* population (Gräfenhan, unpublished). NIV has been shown to be less phytotoxic than DON, and accumulates in lower quantities in the kernels of infected spikes in cereal crops (Muthomi et al. 2000; Miedaner et al. 2001; Foroud et al. 2012a). However, to animal and human health NIV is more acutely toxic than DON, with one tenth the emetic potential compared to that of DON (Wu et al. 2013). In Canada, occasional contamination of barley with NIV is often caused by infections with *F. poae*. The Type A trichothecenes, produced by species such as *F. sporotrichioides* and *F. poae*, tend to be more toxic in mammalian systems than Type B trichothecenes and have also been identified in FHB-infected crops. In western Canada, trichothecene Type A producing *Fusarium* species are more frequently recovered from FHB-diseased oat and barley seeds (Gilbert and Tekauz 2011). On durum wheat, the predominant *Fusarium* species found in Canada are *F. graminearum* and *F. avenaceum* (Clear et al. 2005; Gräfenhan et al. 2013; Tittlemier et al. 2013b). Depending on the year, the latter species can cause significant damage on heads and seed of durum, especially in the main growing areas of western Canada (Fig. 10.5). Tittlemier et al. (2013b) demonstrated that *F. avenaceum* is the main producer of emerging mycotoxins, including MON and enniatins (ENNs), on durum wheat in Canada.

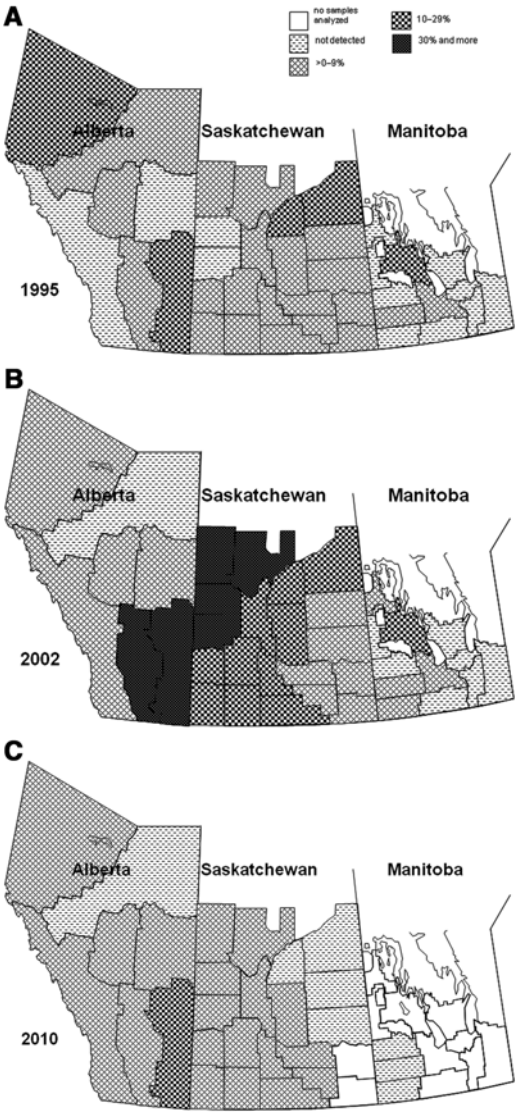

Fig. 10.4 Frequency of occurrence of *Fusarium culmorum* on Fusarium damaged kernels (FDK) of wheat from western Canada in the years 1995 (**a**), 2002 (**b**), and 2010 (**c**)

10.3.2 Infection Pathways and Symptoms

Fusarium infection of cereals is caused by inoculum build up in the soil on crop residues. Initial FCR infection, which can be caused by *Fusarium* mycelium or spores, occurs on emerging shoots, or at the crown or stem base of cereals (Burgess et al. 2001). It has been shown that the trichothecene biosynthesis is initiated during early stages of infection, and while trichothecene accumulation is not necessary for symptoms to develop, a higher infection rate is observed in the presence of the toxin

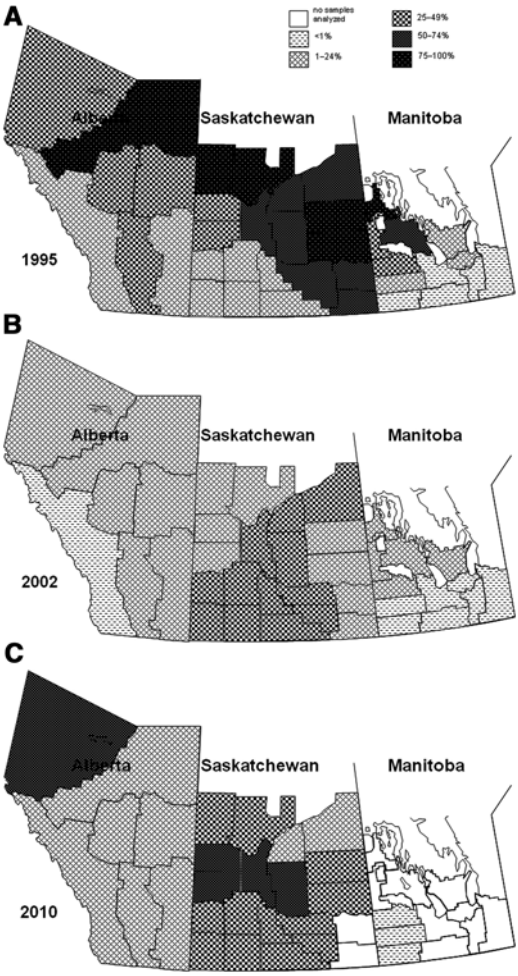

Fig. 10.5 Frequency of occurrence of *Fusarium avenaceum* on Fusarium damaged kernels (FDK) of wheat from western Canada in the years 1995 (**a**), 2002 (**b**), and 2010 (**c**)

(Mudge et al. 2006). The fungus can be isolated from the stem base, the flag leaf node, mature heads and kernels of FCR-infected plants (Mudge et al. 2006). FCR has been observed in cereal crops worldwide, including Canada and the United States (Smiley et al. 2005; Fernandez et al. 2011), and tends to be a major problem in Australia (Backhouse et al. 2004; Obanor et al. 2013).

While FHB can be caused by macroconidia or chlamydospores, ascospores released from the perithecium under humid conditions (reviewed in Bai and Shaner 1994; Parry et al. 1995; Gilbert and Haber 2013) provide the main source of inoculum under field conditions (Sutton 1982; Fernando et al. 2000; Markell and Francl 2003). Infection of cereal inflorescence occurs during anthesis and grain development. Initial symptoms appear as brown water spots on individual spikelets, typically near the base of the glume. As infection progresses, the whole spikelet shows signs of necrosis or premature senescence (Fig. 10.6), and sometimes white or

Fig. 10.6 Symptoms of FHB: (**a**) browning/discoloration of infected spikelets in wheat; (**b**) premature senescence of FHB-infected wheat spike; (**c**) browning/discoloration of infected spikelets in barley and (**d**) oat

pinkish mycelium is visible on the surface of the spikelet (Parry et al. 1995). Detailed studies of *Fusarium* invasion of the wheat spike have been conducted using sophisticated microscopy techniques (Kang and Buchenauer 1999, 2000a, b, c, 2002a, b, 2003; Siranidou et al. 2002; Wanjiru et al. 2002; Jansen et al. 2005; Kang et al. 2005). *Fusarium* can gain access to the host cell through the stomata; however, the primary mode of invasion is by direct penetration of the adaxial epidermal cell walls of the spikelet (Kang and Buchenauer 2000a; Pritsch et al. 2000). Penetration could be facilitated by cutinases and lipases which may lead to cuticle degradation, and expression of a *Fusarium* gene encoding the latter has been implicated in FHB aggressiveness (Voigt et al. 2005). While cuticle degradation has not been experimentally observed, degradation of cell wall components has been observed during *Fusarium* infection of wheat (Kang and Buchenauer 2000b; Wanjiru et al. 2002; Kang et al. 2005). Furthermore, expression or accumulation of cell wall degrading enzymes, including cellulases and pectate esterases, has been observed in the *F. graminearum* secretome and upon exposure to plants or cell wall components (Phalip et al. 2005; Paper et al. 2007; Carapito et al. 2013; Rampitsch et al. 2013). Once established within the spikelet, hyphae can spread to other spikelets within the head through the rachis (Parry et al. 1995). Disease spread typically occurs below the infected spikelets, and premature senescence, or wilt, is sometimes observed above the infected spikelets. Trichothecene biosynthesis is induced upon colonization of the developing kernel, and again at the rachis node (Ilgen et al. 2009). DON accumulates ahead of the growing hyphae, and by 4–6 days after inoculation the hyphae can be found at the rachis both inside and outside of the vascular bundles (Kang and Buchenauer 1999). The production of trichothecenes has been shown to be necessary for *Fusarium* disease spread in *Triticeae* (Proctor et al. 1995; Eudes

et al. 2001; Bai et al. 2002; Langevin et al. 2004; Jansen et al. 2005; Maier et al. 2006). By contrast trichothecenes are not required for the establishment of initial infection (Bai et al. 2002; Jansen et al. 2005). FHB infection of the wheat spike leads to yield losses when infection occurs during anthesis and early stages of kernel development, and the kernels that do develop are often contaminated with mycotoxins (Bushnell et al. 2003; Steffenson 2003; Del Ponte et al. 2007).

10.3.3 Physiological Mechanisms of Resistance

The most effective means to prevent *Fusarium*-related damage is to cultivate crops with high levels of resistance (Foroud and Eudes 2009). The mechanisms of FCR resistance are not well characterized. FHB resistance mechanisms are well described, with two major forms of resistance initially defined by Schroeder and Christensen (1963): Type I, resistance to initial infection; and Type II, resistance to disease spread within an infected spike. Other forms of resistance, as summarized by Mesterházy (2003a), include: Type III, resistance to kernel infection (Mesterházy 1995); Type IV, tolerance to FHB and trichothecenes (Mesterházy 1995); and Type V, resistance to trichothecene accumulation (Miller et al. 1985). Type V resistance can be further subdivided into two classes based on the method of resistance, as defined by Boutigny et al. (2008): Type V class 1 is defined as resistance to trichothecene accumulation by chemical modification, and Type V class 2 is defined as resistance to trichothecene accumulation by inhibition of its biosynthesis.

Cell wall lignification or thickening of the rachis node, accompanied with delayed hyphal colonization of the rachis, has been implicated in Type II resistance in wheat (Kang and Buchenauer 2000c). Jansen et al. (2005) also observed cell wall thickening at the rachis node of susceptible wheat inoculated with a trichothecene non-producing mutant (Proctor et al. 1995) of *F. graminearum* that is unable to spread in otherwise susceptible cultivars. As previously mentioned, trichothecene biosynthesis is induced when the hyphae reaches the rachis node (Ilgen et al. 2009), and trichothecene production is required for disease spread to occur (Proctor et al. 1995). Thus, it is likely that accumulation of trichothecenes is involved in weakening the barrier at the rachis node, and that Type II resistant genotypes are able to prevent and/or slow this process through enhanced cell wall thickening compared with susceptible genotypes.

Among the major cereal crops, wheat is the most susceptible and the most heavily FHB-affected crop, where tetraploid (AABB) durum wheat is more susceptible than hexaploid (AABBDD) bread wheat (Langevin et al. 2004). Barley is the second most affected cereal crop—although 6-row barley is nearly as susceptible as wheat, whereas 2-row barley is more resistant. Barley has inherent Type II resistance, and while unconventional mycelial spread by external routes has been observed (Langevin et al. 2004), disease spread does not occur through the rachis (Langevin et al. 2004; Jansen et al. 2005). Rye and oats are the most FHB resistant among the major cereals (Langevin et al. 2004), although disease symptoms are not

as clearly discernible in standing oats as they are in other cereals (Fig. 10.6) (Tekauz et al. 2004, 2008). Furthermore, oats tend to accumulate more DON and T-2 toxin than wheat (Langseth and Rundberget 1999; Tekauz et al. 2004). Triticale, a hybrid of wheat and rye which has generally been shown to have higher disease resistances than wheat, is shown to have similar FHB-susceptibility as hexaploid wheat (Langevin et al. 2004).

10.3.4 Genetics of Resistance

FHB resistance is polygenic and also tends to be associated with poor agronomics, making it challenging for breeders to incorporate high levels of resistance into favourable cultivars. Over 100 QTL have been identified in FHB resistance of hexaploid wheat, and 22 of these have been reported in multiple mapping populations (reviewed in Bürstmayr et al. 2009). One of the most widely used and best characterized sources of resistance is 'Sumai3', a Chinese cultivar with very strong Type II resistance. Three major QTL have been identified in 'Sumai3': 3BS (also known as Fhb1), which is the best source of Type II resistance; 5A, which is associated with Type I resistance and is found in different germplasm from different regions worldwide; and 6BS (Fhb2) (Bürstmayr et al. 2009). It has been proposed that the 3BS QTL encodes or regulates expression of a UDP-glycosyltransferase (Lemmens et al. 2005) or a pectin methyl esterase inhibitor (Zhuang et al. 2012). UDP-glycosyltransferases can detoxify DON through condensation of glucose with the C-3 hydroxyl group (Poppenberger et al. 2003). Glycosylated-DON and derivatives thereof have been observed in *Fusarium*-infected cereals (Berthiller et al. 2005; Dall'Asta et al. 2005; Lemmens et al. 2005). Pectin methylesterase inhibitors interfere with the activity of pectin methylesterase, a key enzyme involved in pectin biosynthesis. Pectin is a major component of the plant cell wall, and its degradation has been observed during *Fusarium* infection of wheat (Kang and Buchenauer 2000b). Transgenic expression of an *Actinidia chinensis* pectin methylesterase inhibitor has been shown to improve resistance to fungal diseases (including FHB) in durum wheat (Volpi et al. 2011).

Despite the higher susceptibility and lower genome complexity of durum wheat, only five QTL mapping studies on FHB resistance have been reported in tetraploid wheat, and some of these were conducted in wild relatives of *Triticum turgidum* subsp. *durum* (Ban and Watanabe 2001; Stack et al. 2002; Somers et al. 2006; Bürstmayr et al. 2012). Several FHB-resistance QTL identified in tetraploid wheat correspond to genomic regions of resistance QTL from hexaploid wheat, including 3B from *T. turgidum* subsp. *durum* and 6B from *T. turgidum* subsp. *dicoccum*, corresponding to the hexaploid 3BS and 6BS QTL, respectively (Bürstmayr et al. 2012).

In barley, FHB-resistance QTL have been identified on all seven chromosomes (de la Pena et al. 1999; Zhu et al. 1999; Ma et al. 2000; Yu et al. 2010). The Vrs1 locus, which confers row-type, is associated with a QTL that confers the higher FHB resistance observed in 2-row barley compared with 6-row. It is not known whether this resistance is result of a pleiotropy or if it is directly linked to Vrs1 (reviewed in Massman et al. 2011).

In addition to QTL studies, a series of functional genomic experiments have been conducted in wheat and barley to identify genes and/or molecular pathways involved in mediating FHB resistance (Pritsch et al. 2000, 2001; Wang et al. 2005; Zhou et al. 2005, 2006; Boddu et al. 2006, 2007; Bernardo et al. 2007; Golkari et al. 2007, 2009; Geddes et al. 2008; Li and Yen 2008; Jia et al. 2009; Steiner et al. 2009; Cho et al. 2012; Foroud et al. 2012b). In these studies, *Fusarium*-induced up-regulation of pathogenesis-related (PR) proteins and antioxidants has been observed, and in some cases this up-regulation was higher and/or sooner in resistant lines compared with susceptible ones (Pritsch et al. 2000; Geddes et al. 2008; Golkari et al. 2009; Foroud et al. 2012b). Changes in expression of genes involved in regulating plant hormone biosynthesis and responses have also been observed. In microarray studies, complemented by hormone treatment experiments, Li and Yen (2008) reported that the hormones jasmonic acid (JA) and ethylene (ET) are involved in mediating FHB resistance. Similarly, Desmond et al. (2005) reported a role for JA signalling in FCR resistance in wheat; although, it should be noted that, a different set of host genes is believed to be responsible for FHB and FCR resistances (Li et al. 2010). Virus-induced gene silencing experiments in wheat, where suppression of the ET signalling pathway leads to increased FHB-susceptibility (Gillespie et al. 2012), support results presented by Li and Yen (2008). By contrast, genetic silencing of *ET-INSENSITIVE 2* (*EIN2*; involved in ET signalling) by RNA-interference led to reduced FHB-susceptibility in wheat cv. 'Bobwhite' (Chen et al. 2009). Furthermore, exogenous applications of an ethylene precursor or inhibitor demonstrated that ET signalling can enhance FHB-susceptibility in wheat and barley (Chen et al. 2009). This discrepancy in results was also observed in a separate hormone silencing experiment, where *EIN2* was silenced by RNA-interference in three wheat genotypes (Foroud 2011). In this study, ET silencing led to increased susceptibility in the susceptible genotype, had no impact on the Type I resistant genotype, and led to increased resistance in the Type II resistant genotype. Different outcomes were also observed in different genetic backgrounds silenced in the JA- and salicylic acid (SA)-signalling pathways (Foroud 2011). In the dicot plant *Arabidopsis*, Makandar et al. (2010) observed crosstalk between SA and JA signalling pathways in *Fusarium* resistance, and proposed that the timing of SA and JA signalling is critical in differentiating between resistant and susceptible outcomes. Together, these studies suggest that the role of plant hormones in mediating disease outcomes is genotype-dependent, and may be dependent on crosstalk among different signalling pathways.

10.4 Fusarium Diseases of Maize

10.4.1 Pathogens and Associated Mycotoxins

Several species of *Fusarium* infect maize with infection of the ear and the stalk being the most commonly found diseases. The predominant species causing ear and stalk rot in Canada is *F. graminearum* (Koehler 1957, 1959; Sutton 1982; Reid 1996). A less predominant species is *F. verticillioides* (previously referred to

as *F. moniliforme*, Seifert et al. 2003). A comprehensive list of other *Fusarium* species responsible for ear and stalk rots is provided by Mesterházy et al. (2012). The optimum temperature for *F. graminearum* development is 26–28 °C, while *F. verticillioides* tends to grow best at higher temperatures (Reid et al. 1999); therefore, *F. graminearum* is predominantly found in northern regions worldwide and *F. verticillioides* in the southern regions or in dryer years in a northern area (Reid et al. 1999). A single ear or grain can occasionally be infected by different *Fusarium* spp. (Logrieco et al. 2002). Pathogenicity between *Fusarium* spp. and aggressiveness within a species is quite variable and highly dependent on the environmental conditions in a given field season (Reid et al. 2002; Garcia et al. 2009; Iglesias et al. 2010; Miedaner et al. 2010).

Fusarium spp. produce a large number of chemically very different mycotoxins (Logrieco et al. 2002). *Fusarium graminearum* infected ears are usually contaminated with the trichothecene toxin DON before harvest and ZEA during storage. If contaminated grain is fed to livestock, especially swine, DON results in vomiting, feed refusal, decreased weight gain and reproductive problems (Vesonder et al. 1981; Prelusky et al. 1994). This toxin is also an immunosuppressant and thus predisposes animals to other diseases and masks underlying toxicoses (Pestka and Bondy 1994). ZEA causes reproductive problems including reduced litter size, swine estrogenic syndrome and male infertility (Prelusky et al. 1994). Grain contaminated with the polyketide fumonisin mycotoxins produced by *F. verticillioides* can result in equine leukoencephalomalacia (Kellerman et al. 1990), porcine pulmonary edema (Harrison et al. 1990), liver cancer in rats (Gelderblom et al. 1988) and neural tube defects in mice (Voss et al. 2006). Fumonisins have also been associated with human esophageal cancer (International Agency for Research on Cancer (ICARC) 1993). These fungal contaminations cause both direct and indirect economic losses to the maize and livestock industry but they also affect the health of grain handlers and processors.

10.4.2 Infection Pathways and Symptoms

There are many infection pathways by which *Fusarium* spp. can enter maize plants. Stalk rot is often initiated from root infection, through stalk nodes or through holes in the stalk often created by insects and sometimes mechanical damage from cultural practices after planting. There are three potential fungal entry points for ear infection: (1) by fungal spores landing on the silks of the flowering ears, germinating and then the fungal mycelia grow down the silks to infect the kernels and cob (rachis) (Koehler 1942); (2) through wounds created by insects, hail, or birds on the ear (Sutton 1982); and (3) from systemic stalk infections of *F. verticillioides* (Foley 1959; Munkvold et al. 1997b). Which infection pathway is more important depends on the *Fusarium* spp. that is predominant, the insect pressures in a given geographical location and the environmental conditions. For example, Munkvold et al. (1997a) reported less ear rot on maize hybrids with the Bt trait which considerably lowers European corn borer

Fig. 10.7 Symptoms of Fusarium ear blight in maize

populations. Larger populations of thrips, especially on ears with looser husks, were correlated to ear rot (Farrar and Davis 1991; Parsons and Munkvold 2010a, b).

The symptoms of ear rot are depicted in Fig. 10.7. Ear rot caused by *F. graminearum* is characterized by a pinkish coloured mold (White 1999). Infection from the silk commonly begins as white mycelium moving down from the ear tip. This mycelium later turns reddish-pink on infected kernels. In some cases, pinkish fungal growth can be found on the exterior husk leaves and in severe infections it is impossible to separate the husks from the kernels as the entire ear becomes a tightly bound mass of fungal and plant tissue that appears 'mummified'. When infection occurs through kernel wounds, a similar fungal growth pattern is seen but it starts from the initial wound site and tends to spread to the tip of the ear faster than to the butt of the ear (Reid and Sinha 1998). Once the kernels reach 22–23 % moisture it is difficult for the fungus to further infect (Christensen and Kaufmann 1969; Xiang et al. 2010a); however cob (rachis) moisture can be 15–25 % higher than kernel moisture, so the infection may spread in the cobs and can enter younger kernels via the pedicel (Reid and Sinha 1998). In some cases the ear may appear to be symptomless but when squeezed by hand it will feel quite spongy and the cob will be wet and often pink/red in colour. Symptoms of *F. verticillioides* infection on maize ears are quite different from that of *F. graminearum*. Depending on the mode of fungal entry, the symptoms often occur on individual kernels or on a limited area of the ear (White 1999). Infected kernels develop a cottony white growth or may develop white streaks on the pericarp and fungal growth on the cob. How fast symptoms develop in a given year is highly dependent on the environment which, not only influences ear development and subsequent kernel drydown, but also fungal growth. Infection through the silks cannot proceed once the silks have dried out (Reid et al. 1992a; Reid and Sinha 1998) and there is a relationship between kernel drydown rates and ear rot severity symptoms (Xiang et al. 2010a).

Maize plants with *Fusarium* stalk infections often wilt and the leaves may change from a light to a dull green colour while the lower stalk becomes dry and the pith tissue disintegrates to a shredded appearance. For *F. graminearum*, distinctive symptoms are a tan to dark brown discolouration of the lower internodes and pink to reddish discolouration of the pith tissue. Bluish-black coloured perithecia or reddish-white asexual spores may form on the stalk surface. For *F. verticillioides*, brown streaks appear on the lower internodes and the rotted pith tissue may be whitish-pink to salmon in colour. For both pathogens, symptoms usually appear late in the season and plants may lodge if infection is severe. Plants that are stressed, such as from an early frost, are more susceptible to stalk rot.

For *F. graminearum* ear rot, visual symptoms are highly correlated to DON levels (Reid et al. 1996; Perkowski et al. 1997; Reid and Sinha 1998; Bolduan et al. 2009). Correlations between symptoms and fumonisin levels are less reliable for *F. verticillioides* infections possibly in part due to systemic infections from the stalk leading to more asymptomatic infections (Pascale et al. 1997; Murillo-Williams and Munkvold 2008).

10.4.3 Resistance Mechanisms

There is variability within the maize gene pool for levels of resistance to Fusarium ear and stalk rots and breeders have successfully developed genotypes with high levels of resistance to some of these diseases (Reid et al. 2001a, b, 2003); however, it is not clear what the mechanism of this resistance is. Phenotypically, two forms of resistance have been described in maize that are somewhat similar to resistance to initial infection and disease spread, respectively, in cereals: (1) 'silk resistance', where the fungus does not penetrate the silk channel, and thus does not infect the kernels (Reid et al. 1992b); and (2) 'kernel resistance', where the fungus does not penetrate the cob, and thus does not spread from kernel to kernel (Chungu et al. 1996). Studies have indicated that the resistance mechanisms may be associated with flavone content in the silks, stalks and kernels (Reid et al. 1992a; Sekhon et al. 2006; Santiago et al. 2007), (E)-ferulic acid content and dehydrodimers of ferulic acid in kernels (Assabgui et al. 1993; Bily et al. 2003), and 4-acetylbenzoxazolin-2-one (4-ABOA) in kernels (Miller et al. 1997). Recently, Cao et al. (2011) researched the role of hydroxycinnamic acids and reported that several changes in cell wall bound compounds of silk tissues were observed after inoculation with *F. graminearum*. It has been postulated that the *An2* gene which encodes an ent-copalyl synthase gene which has a role in gibberellin synthesis might play a role in silk resistance as this gene is strongly up-regulated after maize silk is inoculated with *F. graminearum* (Harris et al. 2005).

Hoenisch and Davis (1994) observed a correlation between higher pericarp thickness and resistance to *F. verticillioides*. The thicker pericarp may inhibit fungal growth as well as act as a barrier to insect feeding. Sampietro et al. (2009) identified various properties of the pericarp and its wax layer as resistance factors.

Sweet corn, which has been bred to have a thin pericarp, is extremely susceptible to both *F. graminearum* and *F. verticillioides* (Reid et al. 2000). Long chain alkanes on the surface of maize silks have also been implicated in resistance to *F. graminearum* (Miller et al. 2003).

Genotypes developed with selection for *F. graminearum* ear rot resistance also exhibit high levels of resistance to *F. verticillioides* and common smut (*Ustilago zeae*) in inoculated trials (Reid et al. 2009) indicating that there may be an associated resistance mechanism to multiple ear diseases. Resistance to ear rot and stalk rot do not correlate (Mesterházy and Kovács 1988).

10.4.4 Genetics of Resistance

The inheritance of resistance to *Fusarium* spp. in maize is complex and maize genotypes possess different resistance levels as regards to kernel and silk channel resistance (Lemmens et al. 2005). Resistance to *F. graminearum* ear rot through kernel infection is under both simple (additive and dominance) and digenic (dominance x dominance) effects (Chungu et al. 1996). Estimates of the number of factors affecting kernel resistance ranged from 4.6 to 13.7. For *F. verticillioides*, Boling and Grogan (1965) estimated several additive, dominant and additive x dominant digenic epistatic gene effects. They estimated an average dominance of approximately 0.5 and the number of participating genes was estimated at 1.47. Eller et al. (2008) established that resistance to *F. verticillioides* ear rot is determined by polygenes. Maternal effects for both species have also been reported (Headrick and Pataky 1991; Kovács et al. 1994).

Several studies have found QTL associated with resistance to *Fusarium* in maize. Robertson-Hoyt et al. (2006) found 7 QTL that explained 47 % of the phenotypic variation for *F. verticillioides* ear rot and nine were found for fumonisin content explaining 67 % of the variation. Working with two maize populations, they found that three QTL for ear rot and two for fumonisin were mapped in similar positions. Two QTL, localized on chromosome 4 and 5, appeared to be consistent in both populations. Ding et al. (2008) reported two QTL on chromosome 3. Pérez-Brito et al. (2001) identified nine and seven QTL in two populations, three of which were co-located. Recently, Martin et al. (2011) identified co-localized QTL for both *F. graminearum* ear rot resistance and reduced levels of DON in different mapping populations. Reinprecht et al. (2008) identified about 100 genes behind the QTL, among them chitinase and protein kinase. A meta-analysis of QTL associated with ear rot resistance (Xiang et al. 2010b) from the data of 14 studies representing *F. graminearum*, *F. verticillioides* and *Aspergillus flavus* QTL studies found that resistance QTL against the three fungi were clustered on the same chromosomes. These data seem to support the idea of common resistance. Various other studies have reported the identification of possible genes and genetic resistance mechanisms related to ear rot resistance (Jenczmionka and Schäfer 2005; Igawa et al. 2007; Yuan et al. 2008; Lanubile et al. 2010; Zhang et al. 2011).

10.5 Management of Fusarium Head Blight Caused by *Fusarium graminearum*

The occurrence of plant disease depends on the interaction of three factors that 'have often been visualized as a triangle…' (Agrios 1988); a virulent pathogen, a susceptible host and a favourable environment are needed for disease to occur. Variation in any one factor will influence the ultimate level and severity of disease. For example, disease severity may be low if the host has some resistance even though the environment is conducive and a pathogen is present at sufficient levels. Disease severity may also be low if weather conditions are too hot, too cold or too dry, even with a susceptible host and a source of disease inoculum. Where a virulent pathogen is not present or is at low levels, disease may either not occur or be at low levels even when the host is susceptible and there is a favourable environment. Disease management strategies employed by farmers rely on manipulation of one or more components of the disease triangle. The ultimate goal is to create cropping conditions that do not favour pathogen survival and/or disease development. Unfortunately, effective management of FHB, while limiting its impact, cannot be achieved by simply manipulating a single component of the disease triangle (e.g. host resistance). As McMullen et al. (2008, 2012) suggests, effective management of FHB and its impacts on crop production and quality require the use of a combination of strategies.

10.5.1 Crop Rotation

Fusarium graminearum overwinters mainly on infected crop residue, but can also be seed-borne (Wiese 1987; Mathre 1997; Gilbert and Tekauz 2000). Survival in crop residue is highest in plant tissues that are resistant to decay, especially the node tissues of small grain cereals (Burgess and Griffin 1968; Sutton 1982). Gilbert and Tekauz (2000) suggested that *F. graminearum* was unlikely to survive in soil without crop residues. Sutton (1982) also indicated that soil is not likely a 'major inoculum source' and referred to work by Gordon (1954, 1956) in Canada where *F. graminearum* was not isolated from soil samples collected from cereal fields. In Australia, Wearing and Burgess (1977) were able to isolate *F. graminearum* from soil, but it was mainly associated with small pieces of debris.

Given the key role of infested crop residues as a source of inoculum, crop rotation to nongramineous hosts and avoiding corn in rotations, or in close proximity, have been suggested as methods of reducing the risk from FHB or ear/stalk rot in corn (Seaman 1982; Wiese 1987; Parry et al. 1995; Mathre 1997; White 1999; Gilbert and Tekauz 2000; Stack 2000). Corn is an important host of *F. graminearum* (White 1999) and can support extensive colonization of not only infected ears, but also of stalks (Windels and Kommedahl 1984; Kommedahl and Windels 1985; Windels et al. 1988). Although Wiese (1987) and Mathre (1997) recommended at least 1 year between grass or cereal production, rotations with at least 2 years between susceptible crops are needed to reduce the risk of FHB (Burgess and Griffin 1968; Warren and Kommedahl 1973; Khonga and Sutton 1988).

For example, Khonga and Sutton (1988) placed infested corn and wheat residue in the field for up to 3 years and found production of perithecia and ascospores by *Gibberella zeae* (perfect state of *F. graminearum*) occurred primarily in the first and second years. Inch and Gilbert (1999) also found that *F. graminearum* could survive in infected seed for up to 2 years regardless of whether it was on the soil surface or buried up to 10 cm deep in the soil.

Inclusion of highly susceptible crop types either directly in the rotation or in adjacent fields can exacerbate FHB issues. Mathre (1997) reported that barley production in the first half of the 1900s was more or less eliminated when corn was grown in rotation with barley in the eastern and central corn belt of the United States, because the level of FHB became so severe. Other research has highlighted the risk of FHB associated with corn in rotation with small grain cereals. The first report of significant levels of FHB and DON contamination in Manitoba wheat, caused by *F. graminearum*, was associated with two fields that were previously cropped to corn (Clear and Abramson 1986). In Ontario, Teich and Nelson (1984) and Teich and Hamilton (1985) found that FHB levels were lower in wheat when it was not sown after corn. More recently, Schaafsma et al. (2001) conducted a survey of hand-harvested grain from commercial wheat fields in Ontario under a range of agronomic practices. They found that in 2 of 4 years (1996–1999), DON levels in wheat were significantly higher when planted on corn residue compared with wheat or soybean residue. In 1996, Schaafsma et al. (2001) found that levels of DON were similar when corn or wheat had been planted 2 years previously and were significantly higher than when soybean was the previous crop. In a Minnesota trial, Dill-Macky and Jones (2000) found that FHB and DON were higher when wheat followed corn, lowest when wheat followed soybean and intermediate with wheat on wheat. In contrast, Yi et al. (2001) found that FHB and DON levels were similar when winter wheat was grown after maize or spring wheat, whether it had been harvested for grain or silage. However, Yi et al. (2001) stated that inoculation of the pre-crop treatments with infested oat grain may have precluded treatment differences. A spore trapping study by Francl et al. (1999) found that inoculum of *G. zeae* was significantly higher on wheat spikes exposed in fields with corn residues than wheat residues. Wheat heads exposed in fields with corn residue had an average number of colony forming units (CFU) of *G. zeae* per wheat spike (head) per day of 126 versus 13 CFU for wheat heads sampled next to wheat residue. Khonga and Sutton (1988) found that corn residue, including kernels and stalk pieces, tended to be more abundant producers of both conidiospores and ascospores than wheat stems, but not wheat kernels or spikelets.

10.5.2 Tillage

Tillage is a traditional strategy that has been recommended for managing FHB, while conservation tillage has often been implicated as a risk factor for FHB caused by *F. graminearum*, as crop residues are the most important source of inoculum (Parry et al. 1995; McMullen et al. 1997; Stack 2000). Teich and Hamilton (1985)

found that FHB was lower in ploughed fields than fields with 'light tillage', but in an earlier study, Teich and Nelson (1984) found that FHB levels were similar with or without ploughing. In a separate study, FHB levels were found to be lower in ploughed treatments versus treatments that had been 'disc-cultivated' (Teich unpublished) (Teich 1989). Dill-Macky and Jones (2000) found that tillage regime did have a significant effect on FHB and DON levels in wheat. When averaged over tillage systems, the incidence of FHB was 63.5 %, 71.8 % and 70.8 % for moldboard ploughing, chisel plough, and no-till, respectively. Small, but significant differences in DON level were observed for the tillage treatments, with moldboard ploughing having 8.1 ppm, compared to chisel plough (10.6 ppm), and no-till (11.1 ppm) which were not significantly different. However, other work indicates that FHB and DON may not always be reduced with tillage. Clear and Abramson (1986) found that the initial appearance of significant levels of Fusarium damaged kernel (FDK) and DON in Manitoba occurred in two wheat fields that had been disced in the previous fall and in the following spring, a tillage regime that would be considered to be conventional and fairly aggressive in western Canada. In a subsequent survey, Gilbert and Tekauz (1993) found no difference between tillage practices during the 1993 FHB epidemic in Manitoba. In Ontario, Miller et al. (1998) found that tillage system (moldboard ploughing versus no-till) did not have a significant influence on the level of FHB or kernel infection. The authors suggested that under weather conditions favourable for disease, other factors such as variety resistance, rotation and previous history of disease would likely be more critical for FHB than the tillage system used. Schaafsma et al. (2001) found similar results from a survey of hand-harvested grain from commercial fields in Ontario from 1996 to 1999. DON levels tended to be slightly higher under minimum tillage versus no-till or conventional, which had similar levels. Overall, Schaafsma et al. (2001) found that tillage system accounted for very little of the variation in DON levels from 1996 to 1999. Other factors such as year, cultivar, and rotation accounted for more variation in DON compared with tillage system. Fernandez et al. (2001) found zero tillage did not result in more FHB compared with conventional tillage in eastern Saskatchewan. FHB severity tended to be highest under minimum tillage, but was lower under both zero and conventional tillage. Khonga and Sutton (1988) suggested that complete burial of infested residue by moldboard ploughing may help to prevent spore production, if residues are not brought back to the soil surface by subsequent tillage. Earthworm activity, which is enhanced under conservation tillage practices (House and Parmelee 1985; Wardle 1995; Kladivko et al. 1997; Chan 2001; Chan and Heenan 2006; Eriksen-Hamel et al. 2009), may help to reduce the amount of *F. graminearum*-infested crop residue under direct seeding (Oldenburg et al. 2008; Schrader et al. 2009; Wolfarth et al. 2011) and perhaps this has contributed to the variable effect of conservation tillage in relation to FHB. In areas where *F. graminearum* is commonly found on crop residues, a general background level of inoculum may preclude any differences in disease risk among tillage systems. Ascospore dispersal from one field to another would introduce the pathogen into fields where infested residues were not present either as a result of burial by tillage or extended crop rotation to non-host crops.

10.5.3 Field Location

Head infections in wheat typically arise from wind-borne ascospores released from fruiting bodies (perithecia) produced by the sexual stage of *F. graminearum*, *G. zeae*, and are formed on old crop residue and infected seed left on the soil surface. Although production of perithecia typically occurs in the spring, these fruiting structures can also be found on harvested grain, especially barley, and can be produced in the fall depending on the location (Paulitz 1996; Mathre 1997). Old crop residues including vegetative and reproductive plant tissues and infected seed are the main sources of inoculum (Sutton 1982).

Dispersal of ascospores appears to occur over relatively short distances. Gilbert and Tekauz (2000) have suggested that the appearance of FHB in eastern Saskatchewan is not likely the result of long-distance (300 km) transport of ascospores, based on results from Fernando et al. (1997). Fernando et al. (1997) demonstrated gradients of head and seed infection resulting from ascospores of *G. zeae*, over distances of at least 22 m. Gilbert and Tekauz (2000) cited reports by Stack (1997) who suggested, based on analysis of spore dispersal gradients, that ascospores could be dispersed and result in head blight symptoms in fields up to 1 mile away from the source of inoculum. Francl et al. (1999) suggested that dispersal of ascospores produced by *G. zeae*, may occur over 'kilometers to tens of kilometers or more….' Based on current research, immediately adjacent fields or areas would be most at risk from air-borne ascospores. Maldonado-Ramirez et al. (2005) and Schmale III and Bergstrom (2007) demonstrated the presence of viable ascospores in the planetary boundary layer suggesting the occurrence of long-distance transport of *G. zeae* ascospores. Recent work using clonal sources of *G. zeae* inoculum identified using microsatellite markers demonstrated dispersal of a released clone up to 750 m, with the majority being collected within 100–250 m of the source (Prussin 2013). Keller et al. (2010) also used clones to study inoculum dispersal of *G. zeae* and found that head infections resulting from a local source of inoculum decreased by 90 % within 6 m of the source. Overall, research suggests that an FHB epidemic within an individual field would largely originate from inoculum produced within the field itself or in adjacent fields. However, as Schmale III and Bergstrom (2007) suggest, long-distant transport of viable ascospores of *G. zeae* may result in the introduction of novel strains into regions where they were not previously present.

Long-distance transport of ascospores into Alberta from eastern Saskatchewan and Manitoba is unlikely. Moreover, there would be a greater potential for a significant reduction in ascospore viability during long-distance dispersal as the ascospores would be exposed to greater periods of ultraviolet (UV) radiation (Waggoner et al. 1983; Rotem and Aust 1991). Radiation has been shown to influence spore survival for many fungi (Leach and Anderson 1982; Caesar and Pearson 1983; Boland 1984; Rotem et al. 1985). Caesar and Pearson (1983) found that average ascospore survival for *Sclerotinia sclerotiorum* was 51 and 22 % after 2 and 4 days field exposure on the upper leaves of a bean canopy. Survival rates of <10 % were observed after 6 days exposure. Ascospore survival also decreased rapidly at relative

humidities of >35 % and temperatures of ≥25 °C. Boland (1984) also demonstrated decreased *S. sclerotiorum* ascospore viability, with average survival rates of <50 % after 2 days and <1 % after 3 days field exposure of ascospores on Millipore filter paper. Higher ascospore survival was observed by Boland (1984) and Caesar and Pearson (1983) when ascospores were shielded from UV radiation. Caesar and Pearson (1983) also found that survival was increased on shaded leaves in the lower part of a bean canopy. Rotem and Aust (1991) found that exposure to UV radiation reduced spore viability from up to several days to less than 50 min for various pathogens including *Aspergillus macrospora*, *A. niger* and *Mycosphaerella pinodes*.

10.5.3.1 Integration of Strategies to Limit Inoculum Availability and Host Infection

Development of less susceptible, and eventually more FHB-resistant cultivars, has been a key focus of Canadian cereal breeding programmes. However, unlike resistance to many of the cereal rusts, high levels of resistance to FHB have been elusive, although substantial improvements in reducing the level of susceptibility and moving towards FHB resistance have been made since the early 1990s in western Canada. Extensive reviews of the topic of host resistance have been published by numerous authors (Parry et al. 1995; Gilbert and Tekauz 2000; Tekauz et al. 2000; Mesterházy 2003a; Steffenson 2003; McMullen et al. 2012).

Like host resistance, fungicides have not provided high levels of FHB control and DON suppression, but depending on the level of host resistance can provide moderate reductions in FHB severity, Fusarium damaged kernel levels, and DON contamination (Mesterházy 2003b; Paul et al. 2008; McMullen et al. 2012). The other major approach to limiting inoculum availability is crop rotation, which if sufficient time is given between host crops, substantial reductions in pathogen viability and inoculum availability can be achieved. However, given the ability of the pathogen to produce wind-borne ascospores, which readily move to adjacent fields and the potential for regional epidemics of FHB to occur as consequence of inoculum dispersal over tens of kilometres, crop rotation in itself may not provide a high level of FHB management where the pathogen is well established on crop residues. McMullen et al. (2008, 2012) emphasized that effective FHB management cannot rely on individual strategies, but rather an integration of multiple disease management strategies that limit inoculum availability and host infection. The combination of growing small grain cereals on residue of non-host crops, use of a moderately resistance host genotype, and application of effective fungicides has been found to greatly reduce the level of disease and DON contamination, while significantly increasing crop yield (McMullen et al. 2008). The combination of host resistance, rotation and fungicide represents a foundation on which other strategies can be added to further reduce inoculum availability and disease development. For example, producers growing small grain cereals under irrigation may be able to reduce the risk of head and seed infection by careful water management (McLaren et al. 2003) In Washington State, FHB or scab, caused by various *Fusarium* spp. including

F. graminearum, was found in irrigated fields, but not in dryland wheat fields (Strausbaugh and Maloy 1986). More recently in Idaho (Marshall et al. 2012) and southern Alberta (Turkington et al. 2005, 2006) irrigation was an important contributing factor to FHB outbreaks in these areas. However, the most difficult aspect of irrigation management for FHB control in the irrigated dry regions such as southern Alberta will be trying to balance the water requirements of the crop during flowering versus the need to reduce the risk of FHB. Efetha (2008) has produced a set of recommendations to help producers meet the water needs of their cereal crops, but at the same time reduce the risk of FHB and potential DON contamination of harvested grain.

Harvest management can be an important consideration when dealing with an infected crop. In areas where the disease is severe, producers are advised to adjust their combines to blow out scabby wheat kernels, FDK, (which are lighter than the other seeds) and infected chaff as a way of improving the grade and reducing toxin levels in harvested grain (Tkachuk et al. 1991; Anonymous 1996; Gilbert and Tekauz 2000; Salgado et al. 2011; McMullen et al. 2012). However, this will not completely eliminate problems in wheat, especially when wet harvest conditions allow for continued fungal growth on the maturing crop and potential DON contamination issues even though FHB and FDK levels appear to be low. Removing severely infected kernels during harvesting is not very effective with barley and oat, although removing the hull in hulless barley is an effective way of reducing DON levels (Clear et al. 1997). The downside to harvest management is that it will typically return highly infected wheat kernels and chaff back into the field where this material can act as a source of inoculum in future growing seasons.

McMullen et al. (2012) also suggest that effective chopping and distribution of straw may help to encourage decomposition of infested residue, thereby reducing the availability of inoculum for subsequent epidemics. Chopping of crop residues into smaller pieces, which exposes a greater surface area to microbial activity increases the rate of decomposition of crop residues (Sims and Frederick 1970; Bremer et al. 1991; Angers and Recous 1997; Jensen and Ambus 1998; Gunnar 2001), thereby removing a potential source of FHB inoculum. Moreover, retention of crop residues under conservation tillage can enhance soil flora and fauna activity (House and Parmelee 1985; Chan 2001; Chan and Heenan 2006), which can result in enhanced residue decomposition, especially where residues are chopped into smaller pieces (Boström and Lofs-Holmin 1986; Lowe and Butt 2003). Ultimately, enhanced activity of soil fauna such as earthworms may help to reduce FHB inoculum availability (Schrader et al. 2009; Wolfarth et al. 2011).

Integration of irrigation and residue management with the combination of rotation, host resistance and fungicide may help to further reduce the impact of FHB. Moreover, strategies such as irrigation management may also help to reduce the amount of infested residue, thereby reducing inoculum availability. The use of more resistance crop varieties has been shown to reduce the amount of infested residue thereby reducing the amount of inoculum available to initiate subsequent epidemics (Salas and Dill-Macky 2003, 2004, 2005). Fungicide application may also help to reduce the amount of infested residue and thus the level of inoculum.

There may be a synergistic effect of using crop rotation, host resistance, residue and irrigation management, and fungicides in relation to the availability of inoculum to initiate FHB. Further research is needed to study these interactive effects and whether they have the potential to further reduce the impact of FHB. Ultimately, for FHB management strategies employed by farmers to be more effective, they must incorporate practices that influence all components of the disease triangle, with the goal to create cropping conditions that do not favour pathogen survival, inoculum production and/or disease development.

10.6 Modern Detection Methods for *Fusarium*-Related Mycotoxins

Many jurisdictions, including Canada, have established regulations and guidelines for the presence of *Fusarium* mycotoxins in grains that are used in the production of food and feed in order to protect consumers. Health Canada has set maximum limits of 1.0 and 2.0 mg/kg for DON in soft wheat used in baby foods and non-staple foods, respectively. These limits are currently under review by Health Canada (2011). The Canadian Food Inspection Agency (CFIA) has guidelines and recommended tolerances for a wider range of *Fusarium* mycotoxins in feed, including diacetoxyscirpenol, T-2 and HT-2 toxins, ZEA, and DON. These values range from 0.025 mg/kg for T-2 toxin in diets for dairy animals up to 5 mg/kg in diets for cattle and poultry (CFIA 2012b). The Canadian regulatory limits for food and guidance and recommended levels for feed are consistent with those in other countries.

In Canada, the analysis of grain and grain products is performed along the grain handling and processing chains in order to demonstrate compliance with established regulatory and guidance levels. Domestic and export shipments of bulk grain are monitored by the Canadian Grain Commission (Tittlemier et al. 2014), feed components are monitored by the Canadian Food Inspection Agency, and grain-based foods are monitored by the Canadian Food Inspection Agency (CFIA 2012a) and Health Canada (Scott 1997).

10.6.1 Sampling and Sample Preparation

The determination of any analyte in a given material involves the following general steps: sampling of the material, processing of the sample and a chemical test to detect and quantify the analyte. The initial sampling step is the basis of the entire analysis—without proper sampling, the final analytical result will be meaningless if it does not relate back to the original material of interest.

Proper sampling is especially important for the analysis of mycotoxins in particulate material such as grains because mycotoxins are heterogeneously

distributed in this type of material. Consequently, small portions of the larger sample can contain very different concentrations of mycotoxins such as DON (Biselli et al. 2008) because kernel to kernel concentrations can vary over two orders of magnitude (Sinha and Savard 1997). This effect of sampling is magnified for samples composed of larger particles such as maize kernels. Concentrations of mycotoxins in cereal kernels can also significantly differ from concentrations in chaff and other non-kernel segments. DON concentrations were approximately 2–10 times higher in chaff, peduncle and rachis tissues from wheat heads (Sinha and Savard 1997); ZEA was similarly elevated in chaff versus *Fusarium* damaged kernels (Golinski et al. 2010).

Sample preparation can help to minimize the heterogeneous distribution of mycotoxins in materials and reduce the variability in analytical results. For example, grinding of whole grain samples reduces the variability of DON and NIV measurements (Champeil et al. 2004). Increasing the size of the sample analyzed also reduces the variance of the entire analysis (Whitaker et al. 2002).

10.6.2 Screening Methods for the Detection and Quantification of Mycotoxins

There are a number of methods in use to detect and quantify *Fusarium*-related mycotoxins based on a variety of technologies; Shephard et al. (2012) provide an overview of recent advances. Methods can be classified and organized based on the different technologies they are based upon, however a more user-friendly way to classify methods is to place them along a continuum from screening to confirmatory methods.

Screening methods generally emphasize ease of use, speed and an overall reduced cost of analysis. Development of many screening methods is aimed towards use in settings outside of the traditional laboratory, such as in-field assessment or monitoring of incoming deliveries at processing facilities. However, screening methods can still be useful in laboratory settings where large numbers of samples need analysis. Screening methods often incorporate quick sample clean-up and rapid detection, but depending on their scope, they may still require access to fume hoods and other laboratory safety equipment due to the use of solvents for extraction.

The most basic screening method is visual inspection. Due to the physical damage that can be produced by *Fusarium* infection, visual inspection of wheat may be used as a screening method in order to estimate *Fusarium* mycotoxin concentrations. Such visual inspection is feasible for wheat, since FDK can be distinguished from healthy kernels due their shriveled and discoloured appearance. However, visual inspection is not feasible for other grains because the physical damage is not as easily discerned. It has been shown that FDK can serve as an estimate of DON (Miedaner et al. 2001; Mesterházy 2002) in wheat, and that FDK are associated with MON in durum wheat (Tittlemier et al. 2014). In order

to manage DON concentrations in wheat, FDK is used as a grading factor in Canada where tolerances for FDK in various wheat classes have been established by the Canadian Grain Commission.

In addition to a restriction to wheat, visual inspection of *Fusarium* damage has other limitations that confine its use to a screening tool. Asymptomatic wheat kernels can still contain mycotoxins (Sinha and Savard 1997). As well, later stage infection can also affect the presence of visual *Fusarium* damage. Reduced damage can be observed from spikes infected past the soft dough stages of kernel development (Del Ponte et al. 2007).

Commercially available chemistry-based screening methods are available for a limited number of *Fusarium*-related mycotoxins in grain. The majority of kits are for the analysis of DON, but some are available for ZEA and T-2/HT-2 (Meneely et al. 2011). Technologies currently available are predominantly immuno-based and include lateral flow devices, enzyme linked immunosorbent assays (ELISA) (Meneely et al. 2011), and planar waveguide-based methods (Tittlemier et al. 2013a).

The performance of commercially available screening methods has been recently reviewed by different groups. The Grain Inspection, Packers & Stockyards Administration (GIPSA) of the United States Department of Agriculture evaluates submitted screening methods against criteria for the quantitative determination of mycotoxins in grains, oilseeds and processed-grain products. GIPSA has evaluated a number of quantitative and qualitative screening methods for the analysis of DON or ZEA, and has posted the results on their website (http://www.gipsa.usda.gov/fgis/insp_weigh/raptestkit.html). Aamot et al. (2012) and Tangni et al. (2011) also report on the performance of commercially available ELISA and lateral flow devices for the analysis of DON in oats and wheat.

Screening methods are useful tools to gauge mycotoxin concentrations in grain outside of a laboratory, or to process a large number of grain samples without requiring complex laboratory equipment. However, there are limitations to screening methods. One very important limitation is the potential for cross reactivity in immuno-based methods. Methods that are based upon the recognition of the mycotoxin analyte by an antibody or other receptor can return inaccurate results when another molecule interacts with the antibody (Tangni et al. 2010). For example, mean results from ELISA tests for DON submitted to an interlaboratory study were approximately 2–3 times higher than those obtained from confirmatory methods that had analyzed the same test material. The difference was attributed to the cross reactivity of the ELISA tests towards 15-ADON, a metabolite of DON (Josephs et al. 2001). There is no confirmation of analyte identity in these screening methods that would avoid issues caused by cross reactivity.

Until recently, screening methods have also focused on single analytes, thus additional tests would need to be run to obtain data on additional analytes. However, multi-mycotoxin screening methods are being developed and commercialized for use in laboratory (Dorokhin et al. 2011; Meneely et al. 2012; Tittlemier et al. 2013a) and non-laboratory settings (He et al. 2012; Lattanzio et al. 2012).

10.6.3 Confirmatory Methods for the Detection and Quantification of Mycotoxins

Confirmatory methods are more complex than screening methods, and require expensive instrumentation and associated technical expertise to operate and maintain the instrumentation. These methods mainly employ chromatography with mass spectrometry, and with advances in fast chromatography, can generate results as quickly as screening methods.

The strength of modern confirmatory methods lies with their ability to confirm analyte identity as well as perform a simultaneous sensitive analysis of many mycotoxins. For example, methods that use liquid chromatography with tandem mass spectrometry have been developed to analyze over 150 various fungal and bacterial metabolites, including many *Fusarium*-related compounds (Vishwanath et al. 2009). The co-occurrence of multiple mycotoxins drives the need for multi-mycotoxin methods, and almost all new methods incorporate multiple mycotoxin analytes (Shephard et al. 2012).

Advances in confirmatory methods include a move towards 'dilute and shoot' methods that minimize sample clean-up in order to decrease analysis time and increase sample throughput (van der Fels-Klerx et al. 2012; Warth et al. 2012). Simple clean-up of sample extracts may also be used to minimize matrix effects and increase sensitivity further. Methods incorporating simple clean-up predominantly use solid phase extraction in cartridge (Schenzel et al. 2012) or dispersive format (Rubert et al. 2012). Sorbents used for the clean-up of sample extracts containing *Fusarium*-related mycotoxins include immunoaffinity materials (Tang et al. 2012) as well as conventional abiotic materials such as silica or C_{18} (Rubert et al. 2012; Schenzel et al. 2012).

10.6.4 General Considerations for the Detection of Fusarium-Related Mycotoxins

Many chemical test methods are available for mycotoxin analysis, and as with any tool, users need to ensure they are properly used in order to generate accurate and precise data. As described above, proper sampling and sample preparation must be performed for the results of the chemical test method to meaningfully relate back to the original sample of interest.

Consumers of chemical test method data must also ensure the methods used follow general proper analytical chemistry practices. All methods used to generate data must be validated so that accurate and precise data are obtained. Validation should be performed for different matrices of interest, because a method that works for one matrix may not work for another (Malachova et al. 2012). There must be

routine monitoring of method performance as well. There are many commercially available certified reference materials containing characterized amounts of the more commonly analyzed *Fusarium*-related mycotoxins (DON, HT-2, T-2), as well as many proficiency tests that have been established for laboratories to verify and monitor their performance.

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