Climate change impacts on the ecology of *Fusarium graminearum* species complex and susceptibility of wheat to Fusarium head blight: a review

M. Vaughan[1], D. Backhouse[2], E. M. Del Ponte[3]

[1] United States Department of Agriculture, Agricultural Research Service, Mycotoxin Prevention and Applied Microbiology Unit, National Center for Agricultural Utilization Research, 1815 North University Street, Peoria, IL 61604 USA

[2] School of Environmental and Rural Science, University of New England, Armidale NSW 2351, Australia

[3] Departamento de Fitopatologia, Universidade Federal de Viçosa, 36570-000, Viçosa, MG, Brazil

Corresponding author: E. M. Del Ponte, Email: delponte@ufv.br

Running title: Climate change effects on the Fusarium graminearum-wheat interaction

Abstract

Fusarium head blight (FHB) of wheat caused mainly by a few members of the *Fusarium graminearum* species complex (FGSC) is a major threat to agricultural grain production, food safety, and animal health. The severity of disease epidemics and accumulation of associated trichothecene mycotoxins in wheat kernels is strongly driven by meteorological factors. The potential impacts of change in climate are reviewed from the perspective of the FGSC life cycle and host resistance mechanisms influenced by abiotic pressures at the ecological, physiological and molecular level. Alterations in climate patterns and cropping systems may affect the distribution, composition and load of FGSC inoculum, but quantitative information is lacking regarding the differential responses among FGSC members. In general, the coincidence of wet and warm environment during flowering enhances the risk of FHB epidemics, but the magnitude and direction of the change in FHB and mycotoxin risk will be a consequence of a multitude of effects on key processes affecting inoculum dynamics and host susceptibility. Rates of residue decomposition, inoculum production and dispersal may be significantly altered by changes in crop rotations, atmospheric carbon dioxide concentration ([CO_2]), temperature and precipitation patterns, but the impact may be much greater for regions where inoculum is more limited such as temperate climates. In regions of non-limiting inoculum, climate change effects will likely be greater on the pathogenic rather than on the saprophytic phase. Although the mechanisms by which abiotic stress influences wheat defenses against *Fusarium* species are unknown, available data would suggest that wheat may be more susceptible to *Fusarium* infection under future climate conditions. Additional research in this area should be a priority so that breeding efforts and climate resilient management strategies can be developed.

Keywords: *Triticum aestivum*, global change, wheat scab, trichothecene, deoxynivalenol

Introduction

Fusarium head blight (FHB) is a fungal disease of wheat, barley and other cereal crops which has significant negative consequences for agricultural production, food safety, and animal health. FHB epidemics may substantially decrease yields and also result in price discounts due to reduced grain quality characteristics such as low test weight (Windels, 2000). In addition, FHB pathogens contaminate grain with trichothecenes and other mycotoxins that can render grain unfit for human or animal consumption. Trichothecenes are of particular concern because they are potent inhibitors of protein synthesis that can induce a wide variety of symptoms including neurologic, gastrointestinal and immune function disorders (Pestka, 2010; Wu *et al.*, 2014). The combination of reduced yield, poor grain quality, and mycotoxin contamination makes FHB a serious threat to the economics of cereal production worldwide (Gilbert and Haber, 2013; Goswami and Kistler, 2004). For example, the direct and indirect economic losses due to FHB outbreaks in the Midwestern United States were estimated at nearly $1 billion per year (Nganje *et al.*, 2004). In South America, FHB has become an increasing problem in reduced-tillage systems, and yield losses of 50% have been reported in some regions during epidemic years (Pereyra and Lori, 2013). In some of China's provinces, acreage devoted to wheat production has been reduced by more than 50% due to recurring FHB epidemics (Yang *et al.*, 2008).

Members of the *F. graminearum* species complex (FGSC) are the primary etiological agents of FHB worldwide (O'Donnell *et al.*, 2000; Sarver *et al.*, 2011), although other complexes such as *F. culmorum*, *F. avenaceum* and *F. poae* can be found associated with FHB (*Parry* et al., 1995) but are of regional importance, especially across Europe (Xu *et al.*, 2007). FGSC consists of at least 16 species, all of which are able to incite FHB and produce trichothecenes. Collectively, members of the FGSC are found in all major cereal production areas of the globe. However, these species display significant biogeographic structure and

optimum climate conditions differ among species within this complex (Backhouse, 2014; O'Donnell *et al.*, 2004). Although members of the FGSC do not appear to be host-specific, significant structuring of FGSC diversity in relation to host has been observed, such that host distributions likely have a significant influence on regional FHB pathogen composition (Boutigny *et al.*, 2011; Gomes *et al.*, 2015; Kuhnem *et al.*, 2015; Lee *et al.*, 2009).

The FGSC also exhibits diversity in trichothecene toxin types, which has important consequences for pathogen fitness and food safety (Goswami and Kistler, 2004; Pasquali and Migheli, 2014; Ward *et al.*, 2002). Members of the FGSC typically produce one of three strain-specific profiles of trichothecene metabolites (chemotypes). These include the type B trichothecenes nivalenol (NIV), 3-acetyldeoxynivalenol (3ADON), and 15-acetyldeoxynivalenol (15ADON) (Miller *et al.*, 1991). Trichothecene chemotype diversity is biogeographically structured and correlated with species diversity (Aoki *et al.*, 2012; Boutigny *et al.*, 2011; Del Ponte *et al.*, 2015; O'Donnell *et al.*, 2004). For example, all three B trichothecene types are found among strains of *F. graminearum*, which is the most common member of the FGSC in many parts of the world. In addition, *F. graminearum* strains from the Midwestern U.S. and southern Canada were recently found to produce a novel type A trichothecene, referred to as NX-2 (Kelly *et al.*, 2015; Liang *et al.*, 2014; Varga *et al.*, 2015). In contrast, *F. meridionale* is found at greatest frequency in South America where it appears to be adapted to maize production environments, and virtually all strains of this species have the NIV chemotype (Aoki *et al.*, 2012; Del Ponte *et al.*, 2015; Gomes *et al.*, 2015). Even among the cosmopolitan *F. graminearum*, trichothecene chemotype diversity is regionally structured and can be substantially altered by the introduction of novel genetic populations (Gale *et al.*, 2011; O'Donnell *et al.*, 2004; Ward *et al.*, 2008).

FHB epidemics caused by FGSC and resulting accumulation of associated trichothecene mycotoxins in the kernels are strongly weather-dependent, and occur when viable airborne

inoculum and warm wet weather coincided with flowering and early grain filling (Sutton, 1982; Wegulo, 2012). With increases in industrial development and the untempered burning of fossil fuels, the atmospheric carbon dioxide concentration ([CO_2]) exceeded 400 ppm in 2013, and is predicted to double or even triple by the end of the century depending on anthropogenic carbon emissions (Intergovernmental Panel on Climate Change 2014). As a consequence of rising [CO_2] and other atmospheric greenhouse gases (methane, nitrous oxide and ozone), seasonal and regional climate conditions are becoming much more variable and extreme. Average temperatures are rising, and severe precipitation events, ranging from heavy downpours to intense episodes of drought, are increasing. Such conditions will impact FHB epidemiology through direct and indirect effects on the FGSC, their host plants and the host-pathogen interactions (Paterson and Lima, 2011; West *et al.*, 2012). Since the abiotic pressures of climate change are important drivers affecting the life-cycle of many pathogens and the host-pathogen interactions, the topic has been of increasing interest to the plant pathological research community during the last decade. Multiple review articles have summarized the potential impacts of anticipated climate changes on plant diseases in general (Garrett *et al.*, 2006; Pautasso *et al.*, 2012; Chakraborty, 2008; Elad and Pertot, 2014).

Due to the close association with favorable weather conditions, disease or mycotoxin risk/intensity can be predicted with reasonable accuracy and precision (> 70%) using forecasting models using within-season weather around flowering and, in some cases, agronomic factors (De Wolf *et al.*, 2003; Del Ponte *et al.*, 2005a; Hooker *et al.*, 2007; Rossi *et al.*, 2003). Some of these models have been linked to projected climate scenarios for future estimates of FHB risk. In most of the studies targeting specific locations or geographic regions in the UK, China, Brazil and Argentina, FHB incidence is predicted to increase mainly due to warmer temperatures in the early season causing wheat to flower earlier, coinciding with projected more wet conditions (Fernandes *et al.*, 2004; Madgwick *et al.*, 2011; Moschini *et al.*,

2013; Zhang *et al.*, 2014). In contrast, FHB risk is predicted to decrease in Scotland where earlier flowering coincides with projected drier conditions (Skelsey and Newton, 2015).

The impacts of climate change on potential shifts in the dominance of pathogenic species in Europe, including FGSC (Parikka *et al.*, 2012) and FHB and associated mycotoxins in the UK (West *et al.*, 2012) and globally, including risk maps, have recently been reviewed (Battilani and Logrieco, 2014). Other researchers have focused on summarizing the climate change effects on mycotoxigenic fungi in general, including FGSC (Paterson and Lima, 2010; 2011; Medina *et al.,* 2015). In this review article, the effects of climate change on FHB and mycotoxin risk with a focus on FGSC members, and not other complexes which may also be affected by climate change. We structured our review from the perspective of the disease cycle for which the final stages, that culminate in disease intensity and mycotoxin amount, result from a complex interaction where climate-related factors are major drivers of the physical, chemical and biological and ecological processes. We paid particular importance to changes in cropping systems that strengthen survival and aggressiveness of the pathogen and ecosystem stresses that weaken wheat health and resistance to FHB.

Fusarium head blight cycle: a systematic overview

A diagram for the FHB cycle was constructed based on the principles of systems analysis (Zadoks, 1971) and summarizes state variables, flows of information, relationships and key processes in the dynamics of FGSC, the wheat host and the plant-pathogen interaction. In the diagram, the main processes (valves) regulate the change from one state (boxes) to another and are driven by climate, host or pathogen-related factors (Fig. 1). The cycle of FHB in wheat caused by FGSC is well known (Schmale and Bergstrom, 2003) and can be understood as two main phases of the life cycle of a homothallic fungi that reproduces sexually. In its saprophytic

phase, the amount of within-field inoculum source is the first logical state variable regulated by the rate of residue decomposition. The type and amount of airborne inoculum available to infect the crop is a function of background inoculum and the rate of inoculum production and release from both local (within-field) and regional (distant) sources. In its pathogenic phase, disease onset occurs during early flowering when fungal propagules land on the florets and germinate in the presence of moisture and non-limiting temperatures. The precise timing of flowering depends on planting dates, host genotype and climate factors affecting crop growth rate. The rate of host infection and colonization are a function of several factors related to host susceptibility/defense mechanisms, pathogen aggressiveness and micrometeorological conditions. The amount of disease is a function of both the rate of new lesions arising from newly infected florets by airborne inocula and lesion expansion to adjacent florets. The type and the amount of mycotoxin in kernels is a function of the FGSC species/genotype, host resistance and weather factors. The potential impact of abiotic pressures of climate change on each of these processes occurring on each phase is reviewed and discussed in details in the following sections.

Saprophytic phase – inoculum source, production and release

The main state variable related to FGSC inoculum is the amount of within-field or regional sources of inoculum, such as crop residues where ascospores and macroconidia are formed. The inoculum originating from distant sources, mainly ascospores, can reach the crop via atmospheric transport (Keller *et al.*, 2014b) or be locally introduced via planting of infected seeds (Sutton, 1982). The role of infected seed in FHB epidemics is considered of nil (Xi *et al.*, 2010) or minor importance should the inoculum from infected seedlings be able to build-up and survive on the foliage up to flowering (Ali and Francl, 2001). Systemic transmission of the

pathogen from infected seed to various aerial parts of the plants, excepting heads and kernels, has been demonstrated, together with considerable amounts of deoxynivalenol (DON) and DON-3-glucoside (a product of DON glycosylation) found in the kernels (Moretti *et al.*, 2014). However, the practical significance of seed borne inoculum to FHB epidemics and further DON accumulation is yet to be clarified. Undoubtedly, the dominant form of inoculum for ear infection is the ascosporic inoculum produced from perithecia on host residues (Keller *et al.*, 2014a). Conidia can also cause infection, but they are usually trapped in much lower numbers than ascospores (Fernando *et al.*, 2000; Manstretta *et al.*, 2015). Conidia may be produced under wetter and possibly warmer conditions than ascospores, and are generally splash-dispersed over shorter distances than for wind-dispersed ascospores (Keller *et al.*, 2014a). Because conidia are generally considered less important, this discussion will focus on ascospores.

Local versus regional inoculum

There is some uncertainty as to how much local inoculum levels limit head blight epidemics (Skelsey and Newton, 2015). On one hand, previous crop can have a significant effect on FHB in wheat, with both airborne propagule density and disease incidence being higher following host crops like wheat and maize than following non-host crops (Dill-Macky and Jones, 2000; Schaafsma *et al.*, 2005). Burial of residues can also reduce FHB incidence, presumably by reducing inoculum production (Dill-Macky and Jones, 2000). In these cases, local inoculum was likely the dominant source. On the other hand, predominant random spatial patterns of FHB incidence in some areas suggest the contribution of regional sources of inoculum (Del Ponte *et al.*, 2003; Spolti *et al.*, 2015). This may be particularly important in areas like southern Brazil (Spolti *et al.*, 2015) where residue retention is the dominant practice and most crops in the

system (wheat, maize, soybean) can host multiple species of the complex (Del Ponte *et al.*, 2015).

Cropping systems and species diversity

Climate change could affect the levels of regional inoculum of FGSC species by either direct effects on the pathogens, or effects on cropping systems. It is very difficult to separate these two effects. For example, *F. asiaticum* appears to be favored by warm, wet conditions (Backhouse, 2014; Qu *et al.*, 2008) but this most likely reflects the climates under which its preferred host, rice, is grown (Lee *et al.*, 2009). The effect of climate on this species is therefore mediated through its effect on rice production. Changes in cropping system, and particularly increases in maize cultivation, have been suggested as causes for the increased prevalence of *F. graminearum* in northern Europe (Waalwijk *et al.*, 2003). Within the FGSC, *F. graminearum* is less competitive on maize than are *F. boothii*, *F. meridionale* or *F. cortaderiae* (Boutigny *et al.*, 2011; Del Ponte *et al.*, 2015). There is no clear evidence to suggest that *F. graminearum* has greater virulence or fitness on maize than *F. culmorum*, the non-FGSC species that is being displaced in Europe. For this species, in this region, it is likely that its change in distribution is due to a combination of recent introduction and better adaptation to warmer climates than indigenous species of *Fusarium* (Skelsey and Newton, 2015; van der Lee *et al.*, 2014). While both *F. culmorum* and *F. graminearum* exhibit increased growth at warmer temperatures, the optimum temperature for production of DON, which functions as a pathogenicity factor, is 20°C for *F. culmorum* and 25°C for *F. graminearum* (Schmidt-Heydt et al., 2011).

In areas where several FGSC members co-occur, a different suite of species dominates on the residues of each cereal crop (Boutigny *et al.*, 2011; Del Ponte *et al.*, 2015). There is little information on whether the total inoculum load of FGSC species differs depending on host

cereal species, and inconsistent conclusions on the implications of this for FHB. In many areas of the world it is likely that climate change-induced alterations to cropping systems may result in a change in the prevalence of individual species within the FGSC, but that this will have minor effects on the risk of head blight for most crops. An exception may be for increased maize production in areas with currently low inoculum loads. The greater biomass of C4 maize compared with C3 wheat and barley could conceivably lead to sufficient increase in infested residues to alter disease risk.

Residue decomposition

The survival of *F. graminearum* in straw is tightly linked to the rate of decomposition (Pereyra *et al.*, 2004). As well as a reduction in the quantity of residue, there is also a displacement of pathogenic species of *Fusarium* by other fungi (Pereyra *et al.*, 2004). Maize and wheat straw lose relative mass at similar rates (Wang *et al.*, 2012), while rice straw decomposes slightly faster than wheat (Viswanath *et al.*, 2010). However, the larger size of maize straw means that pieces large enough to support perithecial production are likely to persist for longer.

The main meteorological factors affecting the rate of decomposition of straw, and the displacement of *Fusarium* species from that straw, are temperature and water availability (Lakhesar *et al.*, 2010a). The decline in recovery of *F. graminearum* from straw pieces follows an exponential decay function of thermal time adjusted for rainfall. The simplest satisfactory index for this is rainday-degrees, calculated as the sum of the mean temperature above a base of 0°C for days on which rain falls (Lakhesar *et al.*, 2010a). This gives an obvious way to predict the effect of climate change on survival of FGSC inoculum in residues using daily temperature and number of raindays from climate change scenarios. Because of the temperature

dependence of residue decomposition, changes to summer climate will have greater effects than changes in winter.

Biocontrol experiments have shown that addition of specific antagonists can increase decomposition and displacement of *Fusarium* species from cereal residues relative to the indigenous soil microflora (Luong *et al*., 2005; Wong *et al*., 2002; Singh *et al*., 2009). The relative effectiveness of these antagonists is dependent on temperature and water potential (Singh *et al*., 2009). This suggests the possibility that the actual effects of climate change in a particular location will depend on the suite of antagonists that are available to compete with pathogens in the crop residues. However, not enough is known yet about interactions between FGSC species and other organisms in residues to determine how important this will be.

Effects of climate change on residue decomposition will be more complex than just those mediated by changes in temperature and rainfall. A meta-analysis study found that elevated CO_2 increased the C:N ratio of C3 non-legumes by 16%, although the effect on C4 plants was not significant (Lam *et al.*, 2012). Because rate of displacement of *Fusarium* species from straw is limited by N availability (Lakhesar *et al.*, 2010b), this may increase survival in wheat or rice straw (but possibly not maize), potentially offsetting any reduction in survival due to increased temperatures or increased number of raindays. This is consistent with experimental evidence that the rate of decomposition of rice straw is reduced at high CO_2 levels (Viswanath *et al.*, 2010). There is also evidence that elevated [CO_2] may increase the biomass of pathogenic *Fusarium* species in wheat stubble relative to plant biomass (Melloy et al., 2010), which may also offset increased decomposition rates. There is scope for more study on the effects of atmospheric composition on survival of FHB pathogens.

Local inoculum production

The production of ascospores (primary inoculum) from residues is the end point of three processes: perithecial development, perithecial (ascus) maturation, and ascospore discharge. From a climate change perspective, the key interest in perithecial development and maturation is when, and how many, ascospores will be available in the field for discharge. Several researchers over many years have studied the effect of temperature and/or water on perithecial development and maturation. In a typical experiment maize stalks artificially inoculated with *F. graminearum* were incubated at combinations of a range of constant temperatures and water potentials (Dufault *et al.*, 2006). Perithecial numbers and rates of maturation were highest at moderate temperatures (20-24°C) and higher water potentials (> -1.3 MPa). A recent study confirmed that maturation is favoured by temperatures between 20-25°C and high relative humidity (Manstretta and Rossi, 2015). From these and previous studies it is clear that environmental conditions determine the rate of production of mature perithecia. However, all of this work has been done in the laboratory.

The laboratory experiments suggest the potential for seasonal effects on the timing of perithecial maturation, but very little is known about this in the field. For obvious reasons, field-based experiments on inoculum have generally been done in areas where head blight occurs regularly and have assumed that mature perithecia will be present at anthesis. However, it is possible that in some parts of the world environmental conditions will be such that perithecial maturation is out of phase with anthesis of susceptible crops. The long-term spore trapping experiment of (Reis, 1988) in southern Brazil showed that ascospores of the FGSC were present throughout the study period (July to April), implying that mature perithecia were present throughout the year. However the study site has mild winters that would allow perithecia to develop at any time. Cold winters at higher latitudes may delay perithecial development and maturation, but there are no field data to test this. Predictive models for the effects of climate change on FHB (Madgwick *et al.*, 2011; Zhang *et al.*, 2014) have shown that anthesis dates will

alter but there is insufficient information from anywhere in the world to suggest how this will interact with the timing of perithecial maturation. The most likely effect is that higher temperatures will allow perithecial maturation earlier in the season but this would be accompanied by earlier anthesis dates, so the effect of climate change on timing of inoculum production would have a small influence on disease severity.

Inoculum release

A recent paper has rightly pointed out that most studies on the effects of weather on airborne ascospores have not differentiated effects on ascospore release from those on perithecial development and maturation (Manstretta and Rossi, 2015). However, most of the published work has been done in areas, particularly in North America, where it can be assumed that the presence of mature perithecia is not limiting. A typical pattern is that ascospore numbers in spore traps around the time of anthesis are generally low but show large peaks following rainfall events (Del Ponte *et al.*, 2005b; Fernando *et al.*, 2000; Inch *et al.*, 2005).

There is evidence that a period of drying is required to prime perithecia for ascospore release (Tschanz *et al.*, 1976) but spore discharge depends on turgor pressure so requires high relative humidity (Trail *et al.*, 2002). Heavy rain or a run of rainy days inhibits discharge (Del Ponte *et al.*, 2005b; Paulitz, 1996) possibly because the ascospores cannot escape from water films over the perithecia. Modelling of the relationship between weather variables and ascospore discharge has shown that rain events and humidity, calculated as either relative humidity or vapour pressure deficit, are the best predictors of airborne ascospore density (Del Ponte *et al.*, 2005b; Manstretta and Rossi, 2015).

Climate changes that increase the frequency of light to moderate rainfall, and the frequency and duration of periods of high relative humidity, could be expected to increase the discharge of ascospores and hence airborne inoculum levels. Conversely, drier conditions will

reduce inoculum levels. However, these changes in climate will also affect the suitability of conditions for infection. In situations where inoculum is not limiting, climate change effects on infection will be much more important than climate change effects on spore production.

Pathogenic phase: climate-pathogen perspective

Pathogen virulence, host susceptibility, favorable microclimate conditions, and timing are essential elements involved in pathogenesis. Virulence is, primarily, a function of the species/genotype composition of FGSC inoculum (originated from local and/or regional sources) reaching wheat spikes during early flowering and favorable weather conditions. As the geographical range of suitable weather conditions shift or expand, the diversity and distribution of organisms within the community will be redefined. Warmer temperatures enable the migration, introduction or increased abundance of more thermophilic/thermotolerant species (Olesen and Bindi, 2002). At the same time, the higher frequency of extreme weather events diminishes the number of species with low phenotypic plasticity (Jentsch *et al.*, 2009).

It is not entirely clear how climate changes will affect the future spatial distribution of FGSC, especially because of close associations with a preferred crop host (Del Ponte *et al.*, 2015; Gomes *et al.*, 2015; Kuhnem *et al.*, 2015), and how they currently differ in relation to infection efficiency and competitiveness mediated by the climate. Similarly to the biology of inoculum production, knowledge on factors influencing pathogenicity is mostly available for *F. graminearum* at both the species and chemotype level. Several FGSC members have been isolated from wheat kernels, suggesting their ability to cause infections, but *F. graminearum* is the most widespread and dominant species across the main wheat-growing regions of the world, suggesting its adaptation as a major FHB pathogen among FGSC in wheat (Aoki *et al.*, 2012; Backhouse, 2014; van der Lee *et al.*, 2014). However, significant regional contributors to

epidemics in wheat include *F. asiaticum* in wheat grown in typical rice-growing regions of Asia (Backhouse, 2014; van der Lee *et al.*, 2014; Zhang *et al.*, 2012) and *F. meridionale* in some regions of Brazil where maize is largely cultivated (Del Ponte *et al.*, 2015). These examples may be linked to increased inoculum of these other FGSC, as previously discussed, but pathogenic fitness advantages have also been hypothesized. In a molecular survey of species at the field level, *F. graminearum* was dominant in infected kernels obtained from wheat crops while *F. meridionale* was dominant in locally infected maize stubbles (Del Ponte *et al.*, 2015). In addition, *F. graminearum* was isolated at higher frequency from wheat spikes inoculated with an equal mixture of both species, further suggesting its increased pathogenic fitness towards wheat. Knowledge on the differential infection ability within FGSC is limited and may also depend on their complex interactions with microclimate but also with host phenology and host resistance traits.

Among the weather variables, temperature and wetness duration have been shown to be key drivers of infection. Experiments using *F. graminearum* sensu lato isolates inoculated on potted plants or detached spikes determined optimum temperatures ranging from 25 to 30 °C and increasing infection frequency with increasing hours of wetness (Rossi *et al.*, 2001; Xu *et al.*, 2007). Temperature and moisture variables from periods around flowering explained a large portion of the variation in disease intensity and mycotoxin levels in modeling studies using field data (De Wolf *et al.*, 2003; Hooker *et al.*, 2007). A recent climate envelope modeling work showed an association between temperature around flowering dates and reported distributions of *F. graminearum*, *F. asiaticum* and *F. boothii* (Backhouse, 2014). However, precise knowledge on the differential temperature and moisture sensitivity of FGSC members is limited and empirical evidence suggests *F. graminearum* as the fastest grower at 25 °C compared to *F. meridionale* and *F. cortaderiae* (Kuhnem *et al.*, 2015; Spolti *et al.*, 2014). However, these parameters link more directly to the rate of host colonization as empirically derived functions

have been used in simulation models for prediction of host invasion rate by *F. graminearum* (Rossi *et al.*, 2003).

The microclimate, differential FGSC aggressiveness, and type II host resistance (discussed in the next section) may largely affect the rate of host colonization. This trait is commonly measured based on disease severity assessment in greenhouse experiments using single-floret inoculations (Foroud *et al.*, 2012; Spolti *et al.*, 2012; Tóth *et al.*, 2008). Intriguingly, most inferences about the interaction effects of water activity (α_w) and temperature are based on mycelial growth rate determined *in vitro* using artificial substrates (Hope *et al.*, 2005; Marín *et al.*, 2010). Differential aggressiveness among FGSC members has been determined in a few studies under optimal conditions (Goswami and Kistler, 2004; Malihipour *et al.*, 2012; Spolti *et al.*, 2012; Tóth *et al.*, 2008). Brazilian *F. graminearum* isolates were more aggressive than *F. meridionale* only towards a less susceptible cultivar, but not towards a more susceptible one (Spolti *et al.*, 2014) and Canadian and Iranian *F. graminearum* isolates were more aggressive than *F. boothi* (Malihipour *et al.*, 2012). Within species, differential pathogenicity has been hypothesized between 3ADON and 15ADON chemotypes of *F. graminearum*, and although results are contradictory in relation to disease levels, DON accumulation seem to be higher in plants inoculated with 3ADON chemotypes (Foroud *et al.*, 2012; Gilbert *et al.*, 2010; Ohe *et al.*, 2010; Puri and Zhong, 2010; Spolti *et al.*, 2014; Ward *et al.*, 2008). In China, *F. asiaticum* isolates from an emergent population that produce 3ADON revealed significant pathogenic fitness advantages over isolates that produce NIV in planta (Zhang *et al.*, 2012). It is less certain whether interactions with temperature determine the rate of colonization by FGSC members, but this seems to be less explored given the higher importance of temperature effects on mycotoxin production rate.

In addition to alterations in species/population dynamics, climate change has the

potential to influence the production of fungal secondary metabolites and mycotoxins which are essential for pathogen virulence and can regulate aggressiveness. 3ADON isolates of *F. graminearum* are more resilient to extreme temperature events, and in response to heat or cold become more aggressive by producing more DON and zearalenone (ZEA) than the isolates from the 15ADON sub-population (Vujanovic *et al*., 2012). Similarly, elevated [CO_2]-acclimated *F. graminearum* is more aggressive and results a greater FHB severity and DON contamination in comparison to ambient [CO_2]-acclimated *F. graminearum* infection (Váry *et al.*, 2015; Vaughan and McCormick, unpublished data). Environmental conditions including water activity (α_w) and temperature have been shown to influence trichothecene production at the level of gene expression, and optimum conditions for thrichothecene gene cluster expression can vary between thrichothecene producers (i.e. *F. graminearum* and *F. culmorum)* (Schmidt-Heydt *et al.*, 2011). However, such studies have not been conducted for other members of the FGSC such as *F. boothi* and *F. asiaticum* nor have they included elevated [CO_2]. Based on results obtained from evaluating the combined interacting effects of α_w x temperature x [CO_2] on *Aspergillus flavus* aflatoxin production and aflatoxin biosynthetic gene expression, the combined interactive effects of three factors can significantly vary from results obtained from only examining α_w x temperature (Medina *et al.*, 2014). Additionally, the effect of climate change factors on pathogens grown, gene transcription and mycotoxin production may differ on artificial medium and in interaction with the host. For example, while *F. graminearum* radial growth is inhibited at elevated [CO_2] on artificial media (Medina *et al*., 2015), fungal biomas accumulates much faster on wheat heads at elevated [CO_2] (Vaughan and McCormick, unpublished data). Therefore, it may be essential to include the interacting factors and organisms in order to understand the potential combined effects of climate change on the FHB.

Pathogenic phase - climate-host perspective

Host resistance level is a product of genetic makeup and environmental conditions. Inherited traits encode the potential for resistance, but expression of resistance traits is largely dependent on environmental signals, which activate or suppress regulatory machinery. Wheat FHB resistance mechanisms and the influence of abiotic stress on their regulation represent significant knowledge gaps in our understanding of how climate change will impact wheat susceptibility to FHB. Despite intense screening, germplasm sources of complete resistance to FHB have not been identified in wheat (McMullen *et al.*, 2012). Even the most resistant cultivars such as Sumai 3 display only partial resistance under favorable weather conditions (Niwa *et al.*, 2014). The potential for particular weather factors to weaken inherent host resistance mechanisms is cause for concern given future climate predictions. While literature describing the host's defense response to *F. graminearum* in particular is gaining ground (Gunnaiah and Kushalappa, 2014; Gunnaiah *et al.*, 2012; Makandar *et al.*, 2012), few studies have evaluated the influence of weather/abiotic stress on wheat defenses. It has been well documented that temperature, humidity and precipitation affect FHB intensity (De Wolf *et al.*, 2003; Rossi *et al.*, 2001); however, the mechanisms by which these factors influence the host-pathogen interaction are still not well understood (Audenaert *et al.*, 2013).

Recent research demonstrated that wheat grown at elevated [CO_2] was more susceptible to *Fusarium* infection, and the highest level of FHB disease was observed when both the pathogen and host (wheat) were acclimated for elevated [CO_2] (Váry *et al.*, 2015). Even moderately resistant cultivars were more susceptible to FHB at elevated [CO_2] suggesting a reduction in the effectiveness of host defense pathways. In this section we discuss the potential of climate change to weaken wheat health and resistance to FHB by 1) imposing additional ecosystem stress, 2) inducing changes in the wheat plant's biology, and 3) reconfiguring the

host defense response.

Climate-host: Ecosystem stresses on the host

Wheat crops, attempting to cope with abiotic stress, will be confounded by additional biotic stress factors, which have the potential to increase their susceptibility to FHB. For example, aphids are predicted to migrate to more temperate regions leading to greater damage in cereal crops (Sharma, 2014), and wheat simultaneously exposed to both aphids and *F. graminearum* exhibits accelerated FHB progression, heightened disease severity, and increased mycotoxin accumulation as compared to plants suffering from *F. graminearum* alone (Drakulic *et al.*, 2015). Likewise, the severity of FHB is greater in wheat plants infected with foliar diseases such as leaf rust (*Puccinia recondita*) and leaf blotch (*Zymoseptoria tritici*) (Mantecón, 2013); both of these pathogens are also expected to increase under future climate conditions (Bebber, 2015). Large quantities of *Fusarium* inoculum are also more likely overcome the threshold of host resistance. As discussed in detail in the saprophytic phase section, agricultural practices that favor inoculum build up are more prone to FHB epidemics, and there is a positive relationship between amount of inoculum and disease severity (Wegulo, 2012).

The microbial community surrounding the plant plays a crucial role in plant health and resistance as well (Berendsen *et al.*, 2012). As studies of the phytobiome have only recently started to break ground, there is still a great deal to learn with respect to how populations of beneficial microorganism will be affected by climate changes and how these changes will influence host susceptibility. Nevertheless, results have shown that disease outbreaks and epidemics typically coincide with reduced microbial diversity which tends to occur during extreme weather events (Quinn and Alexandrov, 2014).

Plant communities will similarly exhibit climate induced demographic changes which have the potential to affect host health. Although C4 plants, such as many of the most troublesome weeds, do not exhibit a pronounced growth stimulation in response to [CO_2] enrichment, they are adapted to relatively arid regions with warmer temperatures, and may have a greater overall advantage depending on the combined conditions of climate change (Yamori *et al.*, 2014). Alberto and collaborators demonstrated that simultaneous exposure to both elevated [CO_2] and temperature increased the competitiveness of a C4 weed relative to a C3 crop (Alberto *et al.*, 1996). Additionally, it has been suggested that climate change is accelerating weed herbicide resistance (Manea *et al.*, 2011; Runion *et al.*, 2014). Increases in weed density will likely compete with the wheat crop for nutrient and water uptake further enhancing abiotic stress.

Accelerated soil erosion and loss of soil fertility is another major agricultural concern with regard to climate change (Brevik, 2013) which has the potential to influence crop productivity and health. Soil management strategies being proposed include a switch to conservation tillage or no-till systems (Garbrecht *et al.*, 2015; Zhang and Nearing, 2005), but these strategies do not consider the potential implication this will have on FHB (West *et al.*, 2012). Research results have varied but data would suggest that soil type, nitrogen source and amount of fertilizer can affect the incidence and severity of FHB (Subedi *et al.*, 2007; Yang *et al.*, 2010). Compensation for low soil fertility with fertilizer application may intensify FHB epidemics. However, adequate soil micronutrients such as copper, zinc and phosphorus also play a role in wheat resistance to FHB (Franzen *et al.*, 2008). Nevertheless, how these differences in soil mineral nutrition influence FHB remains unclear. Therefore, additional research is still needed to determine the best strategy to manage soil erosion without increasing risk of grain mycotoxin contamination.

Climate-host: changes in host biology

Plants have evolved sophisticated defense mechanisms that enable them to respond or adapt to challenges in their environment. In response to climate changes, wheat plants undergo transcriptional and metabolic modifications that directly affect their biology including growth, phenology, nutritional content, allocation of resources and defense responses. Such changes can alter wheat susceptibility to infection and wheat-*Fusarium* interactions.

The exact timing of the window of vulnerability to infection is variable and may extend to two or more weeks after flowering, likely also dictated by cultivar and environment interactions (Andersen, 1948; Schroeder and Christensen, 1963). Recent studies conducted under controlled environment and field conditions have shown that late infection can result in significant DON levels without the same degree of kernel shriveling and lowered yield as infections that occur at anthesis (Cowger and Arrellano, 2010; Yoshida and Nakajima, 2010). It is not known whether distinct FGSC species, populations, or trichothecene genotypes vary in relation to their ability to cause late infections, a trait that may be more related to intrinsic pathogenic traits irrespective of the host phenology. For example, in a study using a mix of DON and NIV-producing *F. graminearum* isolates from Japan, similar levels of these mycotoxins were found in kernels from inoculations at anthesis as well as 10 and 20 days thereafter (Yoshida and Nakajima, 2010).

Most *Fusarium* species enter the wheat husk through the stomata, tiny pores that allow gas exchange (Boenisch and Schäfer, 2011). As atmospheric [CO_2] increases the stomatal conductance and density tends to decrease (Allen *et al.*, 2011). A reduction in the number and size of epidermal openings could reduce the ability of some pathogens to successfully penetrate and cause infection. However, *F. graminearum,* can also penetrate the floret tissues directly via infection hyphae (Boenisch and Schäfer, 2011) and will not likely be significantly hindered.

Weather conditions have a direct effect on plant primary metabolism. Under conditions of elevated [CO_2], photosynthesis is enhanced and carbohydrates accumulate; however, nitrate assimilation is inhibited and tissue protein and nitrogen concentrations decline (Bloom *et al.*, 2014). Such changes in the host's primary metabolism and nutritional content can affect both plant growth and defense as well as pathogenic fungal growth and production of virulence factors (Audenaert *et al.*, 2013). In order for wheat to mount an optimal defense against *Fusarium* infection, it must redistribute energy resources from its primary carbohydrate and protein metabolism.

Carbohydrate partitioning plays an important role in establishing a sink at the site of infection for the mobilization of defenses, but increases in leaf carbohydrate concentration may disrupt the physiological balance of this process (Lemoine *et al.*, 2013). Although it is well established that environmental factors can alter source/sink relationships, the potential effects of growth at elevated [CO_2] on wheat sink establishment during pathogen infection have not been investigated. Recent research in C4 maize, however, has shown that carbohydrates do not significantly accumulate at the site of *Fusarium verticillioides* infection under conditions of elevated [CO_2] suggesting that enhanced photosynthetic efficiency may not be necessary to disrupt the physiological balance of carbohydrate partitioning (Vaughan *et al.*, 2014). Additionally, higher carbohydrate concentrations will likely stimulate DON biosynthesis. Sucrose strongly upregulates the transcription of both *Tri5* and *Tri4* which initiate trichothecene biosynthesis by converting farnesylpyrophosphate to trichodiene and trichodiene to 15-decalonectrin, respectively (Jiao *et al.*, 2008).

Nitrogen transport also plays a role in plant defense. Nitrogen based compounds can be relocated away from the site of infection to evade fungal utilization or transported to infected host cells to aid with endurance depending on the biotrophic or necrotrophic nature of the pathogen (Seifi *et al.*, 2013). However, since *Fusarium* species are hemibiotrophic pathogens,

the role of nitrogen-based compounds in the balance of the host's redox status is most crucial. Proper and timely regulation of oxidative defenses is essential to ensure the most effective strategy is utilized during the initial biotrophic phase and subsequent necrotrophic phase of pathogen invasion (Audenaert *et al.*, 2013). However, DON-producing *Fusarium* species manipulate nitrogen metabolism of the host by encouraging the formation of polyamines. Accumulation of polyamines and nitric oxide can lead to reactive oxygen species (ROS) which benefits the necrotrophic phase of *Fusarium* pathogenesis through the induction of the hypersensitive response (HR) and programmed cell death (PCD) (Gardiner *et al.*, 2010). However, the nitrogen-containing compound glutathione functions as an antioxidant to alleviate oxidative damage and glutathione-S-transferases are thought to be involved in DON detoxification in plants (Gardiner *et al.*, 2010). Due to the dynamic involvement of nitrogen in both host defense and susceptibility to pathogen manipulation, it is difficult to project how changes in host nitrogen content will influence the wheat-*Fusarium* interaction without additional research. Nevertheless, it is evident that climate induced changes in primary metabolism ultimately carry over into secondary metabolism. Both carbon and nitrogen containing skeletons from primary metabolism are utilized for diverse metabolic processes including the formation of essential secondary defense metabolites such as terpenes and benzoxazinoids, respectively (Gunnaiah and Kushalappa, 2014; Makowska *et al.*, 2015).

Climate-host: Reconfiguration of the host defense response

Both abiotic and biotic stress induced plant defenses are controlled by an interconnected network of signaling pathways (Atkinson and Urwin, 2012). Wheat defense responses against *Fusarium* are regulated by two of the main phytohormone signaling pathways which conveniently correspond with the two types of FHB resistance. Type I resistance, or resistance

to initial infection, is regulated by the salicylic acid (SA) pathway (Makandar *et al.*, 2012). SA activates changes in the cellular redox status and its downstream defense machinery include HR and PCD which isolate and deprive nutrients from the biotrophic phase of the pathogen. Jasmonic acid (JA) can have an antagonistic effect on SA signaling and presumably constrains the activity of SA during the initial stages of infection (Makandar *et al.*, 2012). Host 9-lipoxygenases (9-LOX) also appear to be engaged by the pathogen to manipulate the balance between SA and JA signaling to facilitate infection (Nalam *et al.*, 2015). The JA signaling pathway is primarily involved in promoting Type II resistance, or resistance to *Fusarium* spread throughout the wheat head, during the later necrotrophic stages of infection (Makandar *et al.*, 2012). JA signaling activates the production of antimicrobial and antioxidant defense metabolites and host cell wall fortification (Gunnaiah *et al.*, 2012). *Fusarium* circumvents Type II resistance through the production of DON which inhibits protein synthesis, triggers the formation of H_2O_2, and causes cell death (Audenaert *et al.*, 2013; Gunnaiah and Kushalappa, 2014). However, moderately resistant cultivars which carry the inherited *Fusarium head blight resistance loci*, *Fhb1*, do not appear to resist pathogen spread through the detoxification of DON into DON-3-O-glucoside (Gunnaiah and Kushalappa, 2014). Instead, *Fusarium* advance is obstructed by the accumulation of resistance related (RR) metabolites belonging to the phenylpropanoid pathway which enhance host cell wall thickening and reduce pathogen growth due to antifungal and antioxidant properties. The RR phenylpropanoids found in Sumai-3 were primarily the preformed syringyl rich monolignols and their glucosides, which are precursors of lignin biosynthesis and antimicrobial flavonoids (Gunnaiah and Kushalappa, 2014).

It is evident that a coordinated and ordered expression of diverse signaling pathway is necessary for an effective defense response against *Fusarium* (Ding *et al.*, 2011). However, these complex signaling networks are sensitive to the entire condition of the plant, and when abiotic stress is added the host will respond differently (Atkinson and Urwin, 2012). Abiotic

stress factors such as those associated with climate changes have been shown to reconfigure plant defense signaling pathways (Asselbergh *et al.*, 2008; Atkinson and Urwin, 2012; DeLucia *et al.*, 2012; Elad and Pertot, 2014). Such reconfigurations can reduce or enhance plant susceptibility to a particular biotic stress.

SA functions in the defense response against several factors of climate change including elevated ozone ([O_3]), drought and temperature stress (Khan *et al.*, 2015). Interplay between SA, ROS and glutathione appears to be involved in the reprograming that occurs during the plant defense response against biotic and abiotic stress (Herrera-VÃ squez *et al.*, 2015). Furthermore, the atmospheric environment ([O_3] and [CO_2]) surrounding the plant can alter its total antioxidant capacity (Gillespie *et al.*, 2011). Since redox status control of the wheat is an essential part of resistance to *Fusarium* and also a determining factor of DON production, abiotic stress induced redox modifications will likely disrupt the ordered coordination of wheat defenses against *Fusarium*.

Phytohormone signaling pathways appear to be particularly sensitive to elevated [CO_2]. Elevated [CO_2] enhanced SA biosynthesis and signaling but suppressed JA and lipoxygenase (LOX) pathways in both soybean and tomato (Casteel *et al.*, 2012; DeLucia *et al.*, 2012). As would be expected, these changes resulted in enhanced resistance against biotrophic pathogen infection but increased susceptibility to necrotrophic pathogen infection and insect herbivory (Zhang *et al.*, 2015). Constitutive phytohormone levels were not altered in maize grown at elevated [CO_2], but the accumulation of SA, JA, LOX transcripts, and phytoalexin defense metabolites was reduced following *Fusarium verticillioides* inoculation (Vaughan *et al.*, 2014). The weakened defense response increased maize susceptibility to pathogen proliferation but did not increase fumonisin mycotoxin levels. However, the attenuated induction of 9-LOXs, which stimulate mycotoxin biosynthesis (Christensen and Kolomiets, 2011), would explain the observed reduction in fumonisin per unit fungal biomass at elevated [CO_2] (Vaughan *et al.*,

2014). How wheat phytohormones and defense signaling pathways respond under elevated atmospheric [CO_2] is unknown, but it has been demonstrated that wheat plants are more susceptible to *F. graminearum* (Váry *et al.*, 2015).

Given that moderately resistant wheat cultivars were also more susceptible to FHB under conditions of elevated [CO_2] (Váry *et al.*, 2015), it is likely that phenylpropanoid metabolism is compromised at elevated [CO_2]. It has been demonstrated in soybean and aspen trees that elevated [CO_2] negatively affects phenylpropanoid and flavonoid metabolism (Wustman *et al.*, 2001). Moreover, transcriptomic analyses of winter wheat grown at elevated [CO_2] reported a down-regulation in the transcription of *phenylalanine ammonia-lyase (PAL)* which is the enzyme involved in the first committed step in phenylpropanoid biosynthesis (Kane *et al.*, 2013).

Concluding remarks and perspectives

It is evident that changes mediated by the future environment, especially climate, will impact the evolutionary ecology of FGSC and future FHB epidemic patterns. Abiotic pressures due to climate change influence the major processes of the saprophytic and pathogenic phases of the disease cycle . Climate change has direct effects not only on the wheat-pathogen interaction but also on aspects of the cereal cropping system and ecosystem stresses that ultimately affects inoculum potential and host susceptibility, respectively (Figure 2). The complexity of the system means that a multitude of factors and key processes should be taken into account in order to anticipate the magnitude and direction of the change in FHB and mycotoxin risk, which might vary across wheat-growing regions. This may be challenging for quantitative assessments due to gaps in knowledge . In general, warm and wet weather during flowering increase the risk of infection if inoculum is not limiting, but the several direct and

indirect effects of climate change on the inoculum dynamics and host susceptibility are not entirely known. The key rates driving inoculum dynamics such as residue decomposition, perithecial maturation and ascospore discharge are amenable to modeling but most of the data required to build weather-driven models of inoculum production are available for *F. graminearum*, and it is likely that the parameters may vary across FGSC members. Further synthetic and empirical work is required to determine whether climate change-induced effects on inoculum production are significant relative to effects on infection. For such, simulation modeling approach that combines multi-layered data on weather/climate, pathogen and host biology, disease epidemiology, geography and cropping systems may provide useful estimates of disease and mycotoxin risk under the anticipated scenarios (Battilani and Logrieco, 2014; Skelsey and Newton, 2015). Further research is needed to understand the combined influence of multiple abiotic factors on host defenses, and the mechanisms by which individual and combined factors modulate wheat defenses during *Fusarium* infection. Nevertheless, available data would indicate that wheat may be more susceptible to *Fusarium* under future climatic conditions. Therefore additional research in this area should be a priority so that breeding efforts and climate resilient management strategies can be developed.

Acknowledgements

Thanks to the National (Brazilian) Council for Scientific Research (CNPq) for a research fellowship for Dr. Emerson Del Ponte. We are thankful for the useful comments, discussion and contribution of Dr. Todd J. Ward during the preparation of the manuscript. Mention of trade names or commercial products in this article is solely for the purpose of providing specific information and does not imply recommendation or endorsement by the United States Department of Agriculture (USDA). USDA is an equal opportunity provider and employer.

References

Alberto, A., Ziska, L.H., Cervancia, C.R. and Manalo, P.A., 1996. The influence of increasing carbon dioxide and temperature on competitive interactions between a C3 crop, rice (*Oryza sativa*) and a C4 weed (*Echinochloa glabrescens*). Functional Plant Biology 23: 795–802.

Ali, S. and Francl, L., 2001. Progression of *Fusarium* species on wheat leaves from seedlings to adult stages in North Dakota. In: National Fusarium head blight forum, Erlanger, KY, USA. p. 99.

Aoki, T., Ward, T.J., Kistler, H.C. and O'Donnell, K., 2012. Systematics, phylogeny and trichothecene mycotoxin potential of Fusarium head blight cereal pathogens. Mycotoxins 62: 91–102.

Audenaert, K., Vanheule, A., Höfte, M. and Haesaert, G., 2013. Deoxynivalenol: A major player in the multifaceted response of *Fusarium* to its environment. Toxins 6: 1–19.

Backhouse, D., 2014. Global distribution of *Fusarium graminearum*, *F. asiaticuman* and *F. boothii* from wheat in relation to climate. European Journal of Plant Pathology 139: 161–173.

Battilani, P. and Logrieco, A.F., 2014. Global risk maps for mycotoxins in wheat and maize. In: Mycotoxin reduction in grain chains. John Wiley & Sons, Ltd, Chichester, UK, pp. 309–326.

Bebber, D.P., 2015. Range-expanding pests and pathogens in a warming world. Annual Review of Phytopathology 53: 335–356.

Berendsen, R.L., Pieterse, C.M.J. and Bakker, P.A.H.M., 2012. The rhizosphere microbiome and plant health. Trends in Plant Science 17: 478–486.

Boutigny, A.L., Ward, T.J., Van Coller, G.J., Flett, B., Lamprecht, S.C., O'Donnell, K. and

Viljoen, A., 2011. Analysis of the *Fusarium graminearum* species complex from wheat, barley and maize in South Africa provides evidence of species-specific differences in host preference. Fungal Genetics and Biology 48: 914–920.

Brevik, E., 2013. The potential impact of climate change on soil properties and processes and corresponding influence on food security. Agriculture 3: 398–417.

Cowger, C. and Arrellano, C., 2010. Plump kernels with high deoxynivalenol linked to late *Gibberella zeae* infection and marginal disease conditions in winter wheat. Phytopathology 100:719-728.

De Wolf, E.D., Madden, L.V. and Lipps, P.E., 2003. Risk assessment models for wheat Fusarium head blight epidemics based on within-season weather data. Phytopathology 93: 428–435.

Del Ponte, E.M., Fernandes, J.M.C. and Pavan, W., 2005a. A risk infection simulation model for Fusarium head blight of wheat. Fitopatologia Brasileira 30: 634–642.

Del Ponte, E.M., Fernandes, J.M.C. and Pierobom, C.R., 2005b. Factors affecting density of airborne *Gibberella zeae* inoculum. Fitopatologia Brasileira 30: 55–60.

Del Ponte, E.M., Shah, D.A. and Bergstrom, G.C., 2003. Spatial patterns of Fusarium head blight in New York wheat fields suggest role of airborne inoculum. Plant Health Progress. Plant Management Network, 1–8.

Del Ponte, E.M., Spolti, P., Ward, T.J., Gomes, L.B., Nicolli, C.P., Kuhnem, P.R., Silva, C.N. and Tessmann, D.J., 2015. Regional and field-specific factors affect the composition of Fusarium head blight pathogens in subtropical no-till wheat agroecosystem of Brazil. Phytopathology 105: 246–254.

Dill-Macky, R. and Jones, R.K., 2000. The effect of previous crop residues and tillage on Fusarium head blight of wheat. Plant Disease 84: 71–76.

Drakulic J., Caulfield J., Woodcock C., Jones S.P., Linforth R., Bruce T.J. & Ray R.V., 2015. Sharing a host plant (Wheat [*Triticum aestivum*]) increases the fitness of *Fusarium graminearum* and the severity of Fusarium head blight but reduces the fitness of grain aphids (*Sitobion avenae*). Applied Environmental Microbiology 81:3492-3501.

Dufault, N.S., De Wolf, E.D., Lipps, P.E. and Madden, L.V., 2006. Role of temperature and moisture in the production and maturation of *Gibberella zeae* perithecia. Plant Disease 90: 637–644.

Fernandes, J.M., Cunha, G.R. and Del Ponte, E., 2004. Modelling Fusarium head blight in wheat under climate change using linked process-based models. IN: *Proc. 2nd International Symposium on Fusarium Head Blight*,. Michigan State University, East Lansing, Michigan. pp. 441-444

Fernando, W.G.D., Miller, J.D., Seaman, W.L., Seifert, K. and Paulitz, T.C., 2000. Daily and seasonal dynamics of airborne spores of *Fusarium graminearum* and other *Fusarium* species sampled over wheat plots. Canadian Journal of Botany Canadian Science 78: 497–505.

Foroud, N.A., McCormick, S.P., MacMillan, T., Badea, A., Kendra, D.F., Ellis, B.E. and Eudes, F., 2012. Greenhouse studies reveal increased aggressiveness of emergent Canadian *Fusarium graminearum* chemotypes in wheat. Plant Disease 96: 1271–1279.

Gale, L.R., Harrison, S.A., Ward, T.J., O'Donnell, K., Milus, E.A., Gale, S.W. and Kistler, H.C., 2011. Nivalenol-type populations of *Fusarium graminearum* and *F. asiaticum* are prevalent on wheat in Southern Louisiana. Phytopathology 101: 124–134.

Garbrecht, J.D., Nearing, M.A., Steiner, J.L., Zhang, X.J. and Nichols, M.H., 2015. Can conservation trump impacts of climate change on soil erosion? An assessment from winter wheat cropland in the Southern Great Plains of the United States. Weather and Climate Extremes 10: 32–39.

Garrett, K.A., Dendy, S.P. and Frank, E.E., 2006. Climate change effects on plant disease: genomes to ecosystems. Annual Review of Phytopathology 44: 489–509.

Gilbert, J. and Haber, S., 2013. Overview of some recent research developments in Fusarium head blight of wheat. Canadian Journal of Plant Pathology 35: 149–174.

Gilbert, J., Clear, R.M., Ward, T.J., Gaba, D., Tekauz, A., Turkington, T.K., Woods, S.M., Nowicki, T. and O'Donnell, K., 2010. Relative aggressiveness and production of 3- or 15-acetyl deoxynivalenol and deoxynivalenol by *Fusarium graminearum* in spring wheat. Canadian Journal of Plant Pathology 32: 146–152.

Gomes, L.B., Ward, T.J., Badiale-Furlong, E. and Del Ponte, E.M., 2015. Species composition, toxigenic potential and pathogenicity of *Fusarium graminearum* species complex isolates from southern Brazilian rice. Plant Pathology 64: 980–987.

Goswami, R.S. and Kistler, H.C., 2004. Heading for disaster: *Fusarium graminearum* on cereal crops. Molecular Plant Pathology 5: 515–525.

Gunnaiah, R. and Kushalappa, A.C., 2014. Metabolomics deciphers the host resistance mechanisms in wheat cultivar Sumai-3, against trichothecene producing and non-producing isolates of *Fusarium graminearum*. Plant Physiology and Biochemistry 83:40-50.

Gunnaiah, R., Kushalappa, A.C., Duggavathi, R., Fox, S. and Somers, D.J., 2012. Integrated metabolo-proteomic approach to decipher the mechanisms by which wheat QTL (Fhb1) contributes to resistance against *Fusarium graminearum*. PLoS ONE 7: e40695.

Hooker, D.C., Schaafsma, A.W. and Tamburic-Ilincic, L., 2007. Using weather variables pre- and post-heading to predict deoxynivalenol content in winter wheat. Plant Disease 86: 611–619.

Hope, R., Aldred, D. and Magan, N., 2005. Comparison of environmental profiles for growth and deoxynivalenol production by *Fusarium culmorum* and *F. graminearum* on wheat grain. Letters in Applied Microbiology 40: 295–300.

Inch, S., Fernando, W.G.D. and Gilbert, J., 2005. Seasonal and daily variation in the airborne concentration of *Gibberella zeae* (Schw.) petch spores in Manitoba. Canadian Journal of Plant Pathology 27: 357–363.

Bloom, A., Burger, M., A Kimball, B. and J Pinter, J.P., 2014. Nitrate assimilation is inhibited by elevated CO2 in field-grown wheat. Nature Climate 4: 477–480.

Jentsch, A., Kreyling, J., Boettcher-Treschkow, J. and Beierkuhnlein, C., 2009. Beyond gradual warming: extreme weather events alter flower phenology of European grassland and heath species. Global Change Biology 15: 837–849.

Jiao, F., Kawakami, A. and Nakajima, T., 2008. Effects of different carbon sources on trichothecene production and Tri gene expression by *Fusarium graminearum* in liquid culture. FEMS Microbiology Letters 285: 212–219.

Kane, K., Dahal, K.P., Badawi, M.A., Houde, M., Huner, N.P. and Sarhan, F., 2013. Long-term growth under elevated CO2 suppresses biotic stress genes in non-acclimated, but not cold-acclimated winter wheat. Plant Cell Physiology 54: 1751–1768.

Keller, M., Bergstrom, G. and Shields, E., 2014a. The aerobiology of *Fusarium graminearum*. Aerobiologia 30: 123–136.

Keller, M.D., Bergstrom, G.C. and Shields, E.J., 2014b. The aerobiology of *Fusarium graminearum*. Aerobiologia 30: 123–136.

Kelly, A.C., Clear, R.M., O'Donnell, K., McCormick, S., Turkington, T.K., Tekauz, A., Gilbert, J., Kistler, H.C., Busman, M. and Ward, T.J., 2015. Diversity of Fusarium head blight populations and trichothecene toxin types reveals regional differences in pathogen composition and temporal dynamics. Fungal Genetics and Biology 82: 22–31.

Khan, M.I.R., Fatma, M., Per, T.S., Anjum, N.A. and Khan, N.A., 2015. Salicylic acid-induced abiotic stress tolerance and underlying mechanisms in plants. Frontiers in Plant Science 6: 462.

Kuhnem, P.R., Ward, T.J., Silva, C.N., Spolti, P., Ciliato, M.L., Tessmann, D.J. and Del Ponte, E.M., 2015. Composition and toxigenic potential of the *Fusarium graminearum* species complex from maize ears, stalks and stubble in Brazil. Plant Pathology. online early: 10.1111/ppa.12497

Lakhesar, D.P.S., Backhouse, D. and Kristiansen, P., 2010a. Accounting for periods of wetness in displacement of *Fusarium pseudograminearum* from cereal straw. Annals of Applied Biology 157: 91–98.

Lakhesar, D.P.S., Backhouse, D. and Kristiansen, P., 2010b. Nutritional constraints on displacement of *Fusarium pseudograminearum* from cereal straw by antagonists. Biological Control 55: 241–247.

Lam, S.K., Chen, D., Norton, R., Armstrong, R. and Mosier, A.R., 2012. Nitrogen dynamics in grain crop and legume pasture systems under elevated atmospheric carbon dioxide concentration: A meta-analysis. Global Change Biology 18: 2853–2859.

Lee, J., Chang, I.Y., Kim, H., Yun, S.H., Leslie, J.F. and Lee, Y.W., 2009. Genetic diversity and fitness of *Fusarium graminearum* populations from rice in Korea. Applied and Environmental Microbiology 75: 3289–3295.

Lemoine, R., La Camera, S., Atanassova, R., Dédaldéchamp, F., Allario, T., Pourtau, N.,

Bonnemain, J.-L., Laloi, M., Coutos-Thévenot, P., Maurousset, L., Faucher, M., Girousse, C., Lemonnier, P., Parrilla, J. and Durand, M., 2013. Source to sink transport and regulation by environmental factors. Frontiers in Plant Science 4, doi:10.3389/fpls.2013.00272.

Liang, J.M., Xayamongkhon, H., Broz, K., Dong, Y., McCormick, S.P., Abramova, S., Ward, T.J., Ma, Z.H. and Kistler, H.C., 2014. Temporal dynamics and population genetic structure of *Fusarium graminearum* in the upper Midwestern United States. Fungal Genetics and Biology 73: 83–92.

Luongo, L., Galli, M., Corazza, L., Meekes, E., De Haas, L., Van Der Plas, C. L. and Kohl, J. 2005. Potential of fungal antagonists for biocontrol of *Fusarium* spp. in wheat and maize through competition in crop debris. Biocontrol Science and Technology 15:229–242.

Madgwick, J.W., West, J.S., White, R.P., Semenov, M.A., Townsend, J.A., Turner, J.A. and Fitt, B.D.L., 2011. Impacts of climate change on wheat anthesis and Fusarium ear blight in the UK. European Journal of Plant Pathology 130: 117–131.

Makandar, R., Nalam, V.J., Lee, H., Trick, H.N., Dong, Y. and Shah, J., 2012. Salicylic acid regulates basal resistance to Fusarium head blight in wheat. Molecular Plant Microbe Interactions 25: 431–439.

Makowska, B., Bakera, B. and Rakoczy-Trojanowska, M., 2015. The genetic background of benzoxazinoid biosynthesis in cereals. Acta Physiologiae Plantarum 37: 1–12.

Malihipour, A., Gilbert, J. and Piercey-Normore, M., 2012. Molecular phylogenetic analysis, trichothecene chemotype patterns, and variation in aggressiveness of *Fusarium* isolates causing head blight in wheat. Plant Disease 96: 1016–1025.

Manea, A., Leishman, M.R. and Downey, P.O., 2011. Exotic C4 grasses have increased tolerance to glyphosate under elevated carbon dioxide. Weed Science 59: 28–36.

Manstretta, V. and Rossi, V., 2015. Effects of weather variables on ascospore discharge from *Fusarium graminearum* perithecia. PLoS ONE 10: e0138860.

Manstretta, V., Gourdain, E. and Rossi, V., 2015. Deposition patterns of *Fusarium graminearum* ascospores and conidia within a wheat canopy. European Journal of Plant Pathology 143: 873–880.

Mantecón, J.D., 2013. Effect of early foliar disease control on wheat scab severity (*Fusarium graminearum*) in Argentina. International Journal of Agronomy 2013: 6–6.

Marín, P., Jurado, M., Magan, N., Vázquez, C. and González-Jaén, M.T., 2010. Effect of solute stress and temperature on growth rate and TRI5 gene expression using real time RT–PCR in *Fusarium graminearum* from Spanish wheat. International Journal of Food Microbiology 140: 169–174.

McMullen, M., Bergstrom, G., de Wolf, E., Dill-Macky, R., Hershman, D., Shaner, G. and Van Sanford, D., 2012. A unified effort to fight an enemy of wheat and barley: Fusarium head blight. Plant Disease 96: 1712–1728.

Medina, Á., Rodríguez, A. and Magan, N. 2015. Climate change and mycotoxigenic fungi:

impacts on mycotoxin production. Current Opinion in Food Science 5:99–104.

Melloy, P., Hollaway, G., and Luck, J. O., Norton, R. and Aitken, E., Chakraborty, S. 2010. Production and fitness of *Fusarium pseudograminearum* inoculum at elevated carbon dioxide in FACE. Global Change Biology 16, 3363–3373.

Miller, J.D., Greenhalgh, R., Wang, Y.Z. and Lu, M., 1991. Trichothecene chemotypes of 3 *Fusarium* species. Mycologia 83: 121–130.

Moretti, A., Panzarini, G., Somma, S., Campagna, C., Ravaglia, S., Logrieco, A. and Solfrizzo, M., 2014. Systemic growth of *F. graminearum* in wheat plants and related accumulation of deoxynivalenol. Toxins 6: 1308–1324.

Moschini, R.C., Martínez, M.I. and Sepulcri, M.G., 2013. Modeling and forecasting systems for Fusarium head blight and deoxynivalenol content in wheat in Argentina. In: Fusarium head blight in Latin America. Springer Netherlands, Dordrecht, pp. 205–227.

Nalam, V.J., Alam, S., Keereetaweep, J., Venables, B., Burdan, D., Lee, H., Trick, H.N., Sarowar, S., Makandar, R. and Shah, J., 2015. Facilitation of *Fusarium graminearum* infection by 9-lipoxygenases in Arabidopsis and wheat. Molecular Plant Microbe Interactions 28: 1142–1152.

Nganje, W.E., Kaitibie, S., Wilson, W.W., Leistritz, F.L. and Bangsund, D.A., 2004. Economic impacts of Fusarium head blight in wheat and barley: 1993-2001. Agribusiness and Applied Economics. North Dakota State University, ND, USA.

Niwa, S., Kubo, K., Lewis, J., Kikuchi, R., Alagu, M. and Ban, T., 2014. Variations for Fusarium head blight resistance associated with genomic diversity in different sources of the resistant wheat cultivar "Sumai 3." Breeding Science Japanese Society of Breeding, 64: 90–96.

O'Donnell, K., Kistler, H.C., Tacke, B.K. and Casper, H.H., 2000. Gene genealogies reveal global phylogeographic structure and reproductive isolation among lineages of *Fusarium graminearum*, the fungus causing wheat scab. Proceedings of the National Academy of Sciences of the United States of America 97: 7905–7910.

O'Donnell, K., Ward, T.J., Geiser, D.M., Kistler, H.C. and Aoki, T., 2004. Genealogical concordance between the mating type locus and seven other nuclear genes supports formal recognition of nine phylogenetically distinct species within the *Fusarium graminearum clade*. Fungal Genetics and Biology 41: 600–623.

Ohe, von der, C., Gauthier, V., Tamburic-Ilincic, L., Brule-Babel, A., Fernando, W.G.D., Clear, R., Ward, T.J. and Miedaner, T., 2010. A comparison of aggressiveness and deoxynivalenol production between Canadian *Fusarium graminearum* isolates with 3-acetyl and 15-acetyldeoxynivalenol chemotypes in field-grown spring wheat. European Journal of Plant Pathology Springer Netherlands, 127: 407–417.

Olesen, J.E. and Bindi, M., 2002. Consequences of climate change for European agricultural productivity, land use and policy. European Journal of Agronomy 16: 239–262.

Parikka, P., Hakala, K. and Tiilikkala, K., 2012. Expected shifts in *Fusarium* species' composition on cereal grain in Northern Europe due to climatic change. Food Additives

and Contaminants Part a-Chemistry Analysis Control Exposure & Risk Assessment 29: 1543–1555.

Parry, D. W., Jenkinson, P., and McLeod, L. 1995. Fusarium ear blight (scab) in small grain cereals - a review. Plant Pathology 44:207–238.

Pasquali, M. and Migheli, Q., 2014. Genetic approaches to chemotype determination in type B-trichothecene producing *Fusaria*. International Journal of Food Microbiology 189: 164–182.

Paterson, R.R.M. and Lima, N., 2010. How will climate change affect mycotoxins in food? Food Research International 43: 1902–1914.

Paterson, R.R.M. and Lima, N., 2011. Further mycotoxin effects from climate change. Food Research International 44: 2555–2566.

Paulitz, T.C., 1996. Diurnal release of ascospores by *Gibberella zeae* in inoculated wheat plots. Plant Disease 80: 674–678.

Pautasso, M., Döring, T., Garbelotto, M., Pellis, L. and Jeger, M., 2012. Impacts of climate change on plant diseases—opinions and trends. European Journal of Plant Pathology 133: 295–313.

Pereyra, S. and Lori, G.A., 2013. Crop residues and their management in the epidemiology of Fusarium head blight. In: Magliano, T.M.A. and Chulze, S.N. (eds.) Role of temperature and moisture in the production and maturation of *Gibberella zeae* perithecia. Springer, Dordrecht,, pp. 143–156.

Pereyra, S.A., Dill-Macky, R. and Sims, A.L., 2004. Survival and inoculum production of *Gibberella zeae* in wheat residue. Plant Disease Scientific Societies 88: 724–730.

Pestka, J.J., 2010. Toxicological mechanisms and potential health effects of deoxynivalenol and nivalenol. World Mycotoxin Journal 3: 323–347.

Puri, K.D. and Zhong, S., 2010. The 3ADON population of *Fusarium graminearum* found in North Dakota is more aggressive and produces a higher level of DON than the prevalent 15ADON population in spring wheat. The American Phytopathological Society 100: 1007–1014.

Qu, B., Li, H.P., Zhang, J.B., Xu, Y.B., Huang, T., Wu, A.B., Zhao, C.S., Carter, J., Nicholson, P. and Liao, Y.C., 2008. Geographic distribution and genetic diversity of *Fusarium graminearum* and *F. asiaticum* on wheat spikes throughout China. Plant Pathology 57: 15–24.

Reis, E.M., 1988. Quantificação de propagulos de *Gibberella zeae* no ar através de armadilhas de esporos. Fitopatologia Brasileira 13: 324–327.

Rossi, V., Giosue, S., Pattori, E., Spanna, F. and Del Vecchio, A., 2003. A model estimating the risk of Fusarium head blight on wheat. EPPO Bulletin 33: 421–425.

Rossi, V., Ravanetti, A., Pattori, E. and Giosue, S., 2001. Influence of temperature and humidity on the infection of wheat spikes by some fungi causing Fusarium head blight.

Journal of Plant Pathology 83(3).

Runion, G.B., Prior, S.A., Price, A.J., McElroy, J.S. and Torbert, H.A., 2014. Effects of elevated CO2 on biomass and fungi associated with two ecotypes of ragweed (*Ambrosia artemisiifolia L.*). Frontiers in Plant Science Frontiers Media S.A., 5: 500.

Sarver, B.A.J., Ward, T.J., Gale, L.R., Broz, K., Kistler, H.C., Aoki, T., Nicholson, P., Carter, J. and O'Donnell, K., 2011. Novel Fusarium head blight pathogens from Nepal and Louisiana revealed by multilocus genealogical concordance. Fungal Genetics and Biology 48: 1096–1107.

Schaafsma, A.W., Tamburic-Ilincic, L. and Hooker, D.C., 2005. Effect of previous crop, tillage, field size, adjacent crop, and sampling direction on airborne propagules of *Gibberella zeae/Fusarium graminearum*, Fusarium head blight severity, and deoxynivalenol accumulation in winter wheat. Canadian Journal of Plant Pathology 27: 217–224.

Schmale, D.G. and Bergstrom, G.C., 2003. Fusarium head blight in wheat. The Plant Health Instructor, doi:10.1094/PHI-I-2003-0612-01.

Schmidt-Heydt, M., Parra, R., Geisen, R., and Magan, N. 2011. Modelling the relationship between environmental factors, transcriptional genes and deoxynivalenol mycotoxin production by strains of two Fusarium species. Journal of The Royal Society Interface 8:117–126.

Seifi, H.S., Van Bockhaven, J., Angenon, G. and Hofte, M., 2013. Glutamate metabolism in plant disease and defense: friend or foe? Molecular Plant Microbe Interactions 26: 475–485.

Sharma, H.C., 2014. Climate change effects on insects: Implications for crop protection and food security. Journal of Crop Improvement 28: 229–259.

Singh, D. P., Backhouse, D. and Kristiansen, P. 2009. Interactions of temperature and water potential in displacement of *Fusarium pseudograminearum* from cereal residues by fungal antagonists. Biological Control 48:188–195.

Skelsey, P. and Newton, A.C., 2015. Future environmental and geographic risks of Fusarium head blight of wheat in Scotland. European Journal of Plant Pathology 142: 133–147.

Spolti, P., Barros, N.C., Gomes, L.B., Santos, dos, J. and Del Ponte, E.M., 2012. Phenotypic and pathogenic traits of two species of the *Fusarium graminearum* complex possessing either 15-ADON or NIV genotype. European Journal of Plant Pathology 133: 621–629.

Spolti, P., Del Ponte, E.M., Cummings, J.A., Dong, Y. and Bergstrom, G.C., 2014. Fitness attributes of *Fusarium graminearum* isolates from wheat in New York Possessing a 3-ADON or 15-ADON trichothecene genotype. Phytopathology 104: 513–519.

Spolti, P., Shah, D.A., Fernandes, J.M.C., Bergstrom, G.C. and Del Ponte, E.M., 2015. Disease risk, spatial patterns, and incidence-severity relationships of Fusarium head blight in no-till spring wheat following maize or soybean. Plant Disease Scientific Societies, 99: 1360–1366.

Subedi, K.D., Ma, B.L. and Xue, A.G., 2007. Planting date and nitrogen effects on Fusarium head blight and leaf spotting diseases in spring wheat. Agronomy Journal 99: 113.

Sukumar Chakraborty, S., 2008. Impacts of global change on diseases of agricultural crops and forest trees. CAB Reviews: Perspectives in Agriculture, Veterinary Science, Nutrition and Natural Resources 3: 1–15.

Sutton, J.C., 1982. Epidemiology of wheat head blight and maize ear rot caused by *Fusarium graminearum*. Canadian Journal of Plant Pathology 4:195-209

Tóth, B., Kaszonyi, G., Bartok, T., Varga, J. and Mesterházy, Á., 2008. Common resistance of wheat to members of the *Fusarium graminearum* species complex and *F. culmorum*. Plant Breeding 127: 1–8.

Trail, F., Xu, H.X., Loranger, R. and Gadoury, D., 2002. Physiological and environmental aspects of ascospore discharge in (anamorph *Fusarium graminearum*). Mycologia 94: 181–189.

Tschanz, A.T., Horst, R.K. and Nelson, P.E., 1976. The effect of environment on sexual reproduction of *Gibberella zeae*. Mycologia Mycological Society of America, 68: 327–340.

van der Lee, T., Zhang, H., van Diepeningen, A. and Waalwijk, C., 2014. Biogeography of Fusarium graminearumspecies complex and chemotypes: a review. Food Additives and Contaminants Part a-Chemistry Analysis Control Exposure & Risk Assessment 32: 453–460.

Varga, E., Wiesenberger, G., Hametner, C., Ward, T.J., Dong, Y.H., Schofbeck, D., McCormick, S., Broz, K., Stuckler, R., Schuhmacher, R., Krska, R., Kistler, H.C., Berthiller, F. and Adam, G., 2015. New tricks of an old enemy: isolates of *Fusarium graminearum* produce a type A trichothecene mycotoxin. Environmental Microbiology 17: 2588–2600.

Vaughan, M.M., Huffaker, A., Schmelz, E.A., Dafoe, N.J., Christensen, S., Sims, J., Martins, V.F., Swerbilow, J.A.Y., Romero, M., Alborn, H.T., Allen, L.H. and Teal, P.E.A., 2014. Effects of elevated [CO2] on maize defense against mycotoxigenic *Fusarium verticillioides*. Plant, Cell & Environment 37: 2691–2706.

Váry, Z., Mullins, E., McElwain, J.C. and Doohan, F.M., 2015. The severity of wheat diseases increases when plants and pathogens are acclimatized to elevated carbon dioxide. Global Change Biology 21: 2661–2669.

Viswanath, T., Pal, D. and Purakayastha, T.J., 2010. Elevated CO2 reduces rate of decomposition of rice and wheat residues in soil. Agriculture, Ecosystems & Environment 139: 557–564.

Vujanovic, V., Goh, Y. K. and Daida, P. 2012. Heat- and cold-shock responses in *Fusarium graminearum* 3 acetyl- and 15 acetyl-deoxynivalenol chemotypes. The Journal of Microbiology 50:97–102.

Waalwijk, C., Kastelein, P., de Vries, I., Kerényi, Z., van der Lee, T., Hesselink, T., Kohl, J. and Kema, G., 2003. Major changes in *Fusarium* spp. in wheat in the Netherlands.

European Journal of Plant Pathology 109: 743–754.

Wang, X., Sun, B., Mao, J., Sui, Y. and Cao, X., 2012. Structural convergence of maize and wheat straw during two-year decomposition under different climate conditions. Environmental Science & Technology American Chemical Society, 46: 7159–7165.

Ward, T.J., Bielawski, J.P., Kistler, H.C., Sullivan, E. and O'Donnell, K., 2002. Ancestral polymorphism and adaptive evolution in the trichothecene mycotoxin gene cluster of phytopathogenic *Fusarium*. Proceedings of the National Academy of Sciences of the United States of America 99: 9278–9283.

Ward, T.J., Clear, R.M., Rooney, A.P., O'Donnell, K., Gaba, D., Patrick, S., Starkey, D.E., Gilbert, J., Geiser, D.M. and Nowicki, T.W., 2008. An adaptive evolutionary shift in Fusarium head blight pathogen populations is driving the rapid spread of more toxigenic *Fusarium graminearum* in North America. Fungal Genetics and Biology 45: 473–484.

Wegulo, S., 2012. Factors influencing deoxynivalenol accumulation in small grain cereals. Toxins 4: 1157–1180.

West, J.S., Holdgate, S., Townsend, J.A., Edwards, S.G., Jennings, P. and Fitt, B.D.L., 2012. Impacts of changing climate and agronomic factors on Fusarium ear blight of wheat in the UK. Fungal Ecology 5: 53–61.

Windels, C.E., 2000. Economic and social impacts of Fusarium head blight: Changing farms and rural communities in the Northern Great Plains. Phytopathology 90: 17–21.

Wong, P. T. W., Mead, J. A. and Croff, M. C. 2002. Effect of temperature, moisture, soil type and *Trichoderma* species on the. Australasian Plant Pathology 31:253–257.

Wu, F., Groopman, J.D. and Pestka, J.J., 2014. Public Health Impacts of Foodborne Mycotoxins. Annual Review of Food Science and Technology 5: 351–372.

Wustman, B.A., Oksanen, E., Karnosky, D.F., Noormets, A., Isebrands, J.G., Pregitzer, K.S., Hendrey, G.R., Sober, J. and Podila, G.K., 2001. Effects of elevated CO2 and O3 on aspen clones varying in O3 sensitivity: can CO2 ameliorate the harmful effects of O3? Environmental Pollution 115: 473–481.

Xi, K., Turkington, T.K. and Chen, M.H., 2010. Systemic stem infection by *Fusarium* species in barley and wheat. Canadian Journal of Plant Pathology 30: 588–594.

Xu, X.M., Monger, W., Ritieni, A. and Nicholson, P., 2007. Effect of temperature and duration of wetness during initial infection periods on disease development, fungal biomass and mycotoxin concentrations on wheat inoculated with single, or combinations of, *Fusarium* species. Plant Pathology 56: 943–956.

Yamori, W., Hikosaka, K. and Way, D., 2014. Temperature response of photosynthesis in C3, C4, and CAM plants: temperature acclimation and temperature adaptation. Photosynthesis Research 119: 101–117.

Yang, F., Jensen, J.D., Spliid, N.H., Svensson, B., Jacobsen, S., Jorgensen, L.N., Jorgensen, H.J., Collinge, D.B. and Finnie, C., 2010. Investigation of the effect of nitrogen on severity of Fusarium head blight in barley. Journal of Proteomics 73: 743–752.

Yang, L., van der Lee, T., Yang, X., Yu, D. and Waalwijk, C., 2008. *Fusarium* populations on Chinese barley show a dramatic gradient in mycotoxin profiles. Phytopathology 98: 719–727.

Yoshida, M. and Nakajima, T., 2010. Deoxynivalenol and nivalenol accumulation in wheat infected with *Fusarium graminearum* during grain development. Phytopathology 100: 763–773.

Zadoks, J.C., 1971. Systems analysis and the dynamics of epidemics. Phytopathology 61:600-610.

Zhang, H., van der Lee, T., Waalwijk, C., Chen, W., Xu, J., Xu, J., Zhang, Y. and Feng, J., 2012. Population analysis of the *Fusarium graminearum* species complex from wheat in China show a shift to more aggressive isolates. PLoS ONE 7: e31722–13.

Zhang, S., Li, X., Sun, Z., Shao, S., Hu, L., Ye, M., Zhou, Y., Xia, X., Yu, J. and Shi, K., 2015. Antagonism between phytohormone signaling underlies the variation in disease susceptibility of tomato plants under elevated CO2. Journal of Experimental Botany 66: 1951–1963.

Zhang, X., Halder, J., White, R.P., Hughes, D.J., Ye, Z., Wang, C., Xu, R., Gan, B. and Fitt, B.D.L., 2014. Climate change increases risk of Fusarium ear blight on wheat in central China. Annals of Applied Biology 164: 384–395.

Zhang, X.C. and Nearing, M.A., 2005. Impact of climate change on soil erosion, runoff, and wheat productivity in central Oklahoma. Catena 61: 185–195.

Figure legends

Figure 1 Flowchart of a simplified model for Fusarium head blight epidemics that depicts state variables (boxes), processes or rates (valves), flow of information (solid arrows), and relationship between variables (dashed arrows). Temperature, wetness (or water activity) and carbon dioxide [CO_2], are the main known climate drivers affecting individual processes as indicated based on the literature. These processes include both the saprophytic (A) and pathogenic (B) phase (wheat as model crop) of the *Fusarium graminearum* species complex (FGSC).

Figure 2 Direct (solid arrow) and indirect (dashed arrow) effects of climate change on the risk of Fusarium head blight (FHB) and mycotoxin accumulation in wheat as a model crop by members of the *F. graminearum* species complex (FGSC).

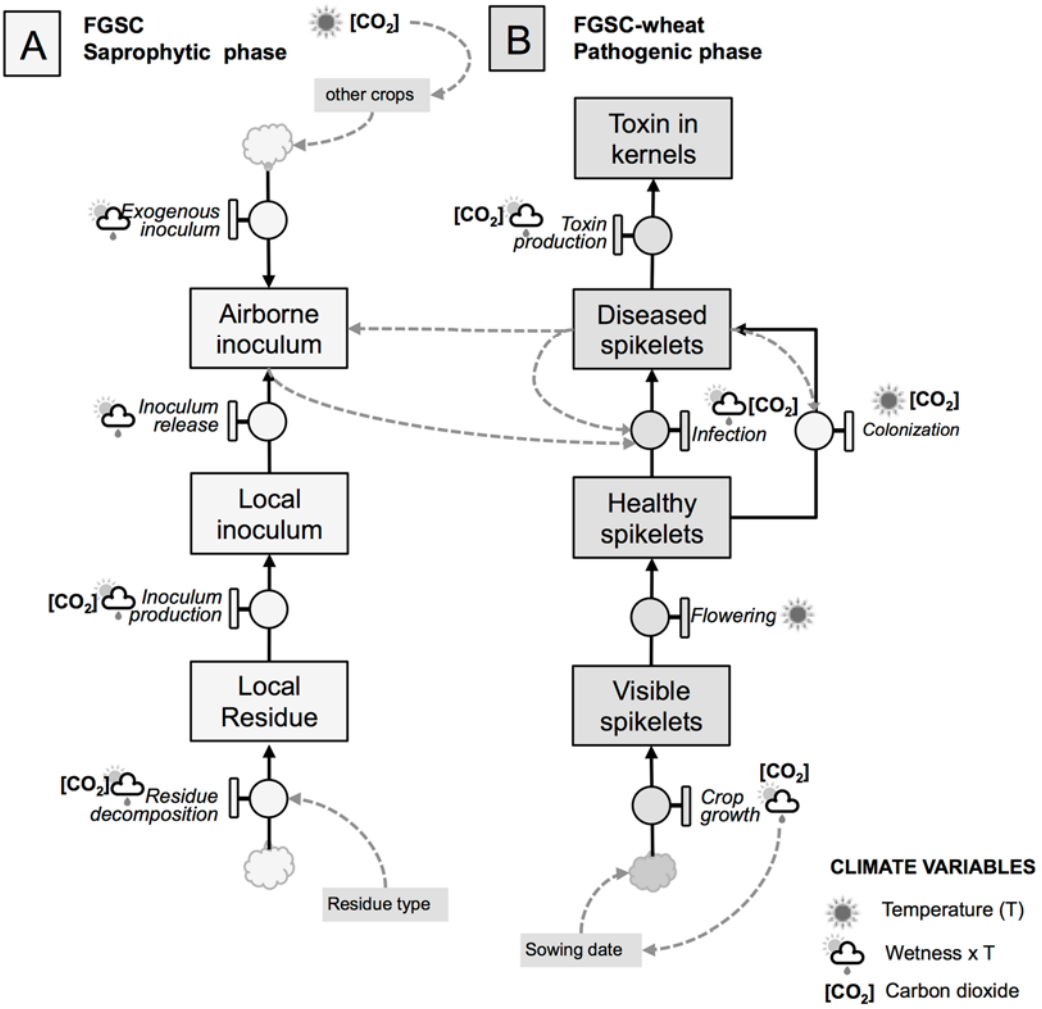
A
FGSC
Saprophytic phase
[CO2]
other crops
Exogenous inoculum
Airborne inoculum
Inoculum release
Local inoculum
[CO2] Inoculum production
Local Residue
[CO2] Residue decomposition
Residue type
B
FGSC-wheat
Pathogenic phase
Toxin in kernels
[CO2] Toxin production
Diseased spikelets
[CO2] Infection
[CO2] Colonization
Healthy spikelets
Flowering
Visible spikelets
[CO2] Crop growth
Sowing date
CLIMATE VARIABLES
Temperature (T)
Wetness x T
[CO2] Carbon dioxide

Figure 1

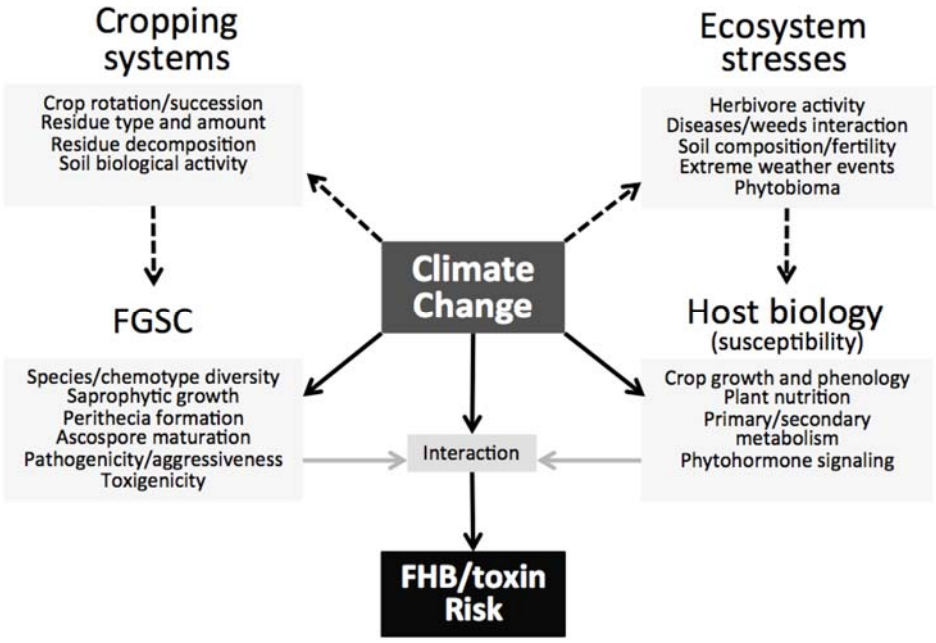
Cropping systems
Crop rotation/succession
Residue type and amount
Residue decomposition
Soil biological activity
Ecosystem stresses
Herbivore activity
Diseases/weeds interaction
Soil composition/fertility
Extreme weather events
Phytobioma
Climate Change
FGSC
Species/chemotype diversity
Saprophytic growth
Perithecia formation
Ascospore maturation
Pathogenicity/aggressiveness
Toxigenicity
Host biology
(susceptibility)
Crop growth and phenology
Plant nutrition
Primary/secondary metabolism
Phytohormone signaling
Interaction
FHB/toxin Risk

Figure 2