

Review

Evolution of Fusarium Head Blight Management in Wheat: Scientific Perspectives on Biological Control Agents and Crop Genotypes Protooperation

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Abstract: Over the past century, the economically devastating Fusarium Head Blight (FHB) disease has persistently ravished small grain cereal crops worldwide. Annually, losses globally are in the billions of United States dollars (USD), with common bread wheat and durum wheat accounting for a major portion of these losses. Since the unforgettable FHB epidemics of the 1990s and early 2000s in North America, different management strategies have been employed to treat this disease. However, even with some of the best practices including chemical fungicides and innovative breeding technological advances that have given rise to a spectrum of moderately resistant cultivars, FHB still remains an obstinate problem in cereal farms globally. This is in part due to several constraints such as the Fusarium complex of species and the struggle to develop and employ methods that can effectively combat more than one pathogenic line or species simultaneously. This review highlights the last 100 years of major FHB epidemics in the US and Canada, as well as the evolution of different management strategies, and recent progress in resistance and cultivar development. It also takes a look at protooperation between specific biocontrol agents and cereal genotypes as a promising tool for combatting FHB.

Keywords: fusarium head blight; scab; biocontrol; breeding; protooperation; epidemics; common wheat; durum wheat; resistance

1. Introduction

The ongoing global intensification of wheat production will likely be accompanied by rising pressure of Fusarium disease [1]. The fungal genus *Fusarium* that was introduced by German mycologist Link in 1809 is the single major cause of several plant diseases, including Fusarium Head Blight (FHB). This plague has become one of the most important cereal diseases worldwide and evidence suggests that the likelihood of FHB epidemics has increased over the past century. Pandemic lines of *Fusarium graminearum* have augmented disease development at an accelerated rate, thus inducing large-scale outbreaks and widespread mycotoxin contamination [2–6]. Although significant effort has been made to control FHB and its persistent mycotoxins (e.g., deoxynivalenol- DON, zearalenone-ZEN) in staple cereal crops, food and feed, a holistic effective management of FHB remains a challenging task for numerous innovative investigations [7]. Nowadays, assembling FHB damage data, using algorithms, and correlating predictive computational models are being explored to achieve a predictive accuracy of FHB epidemics [8] and to develop strategies for reducing risk of mycotoxins in wheat and other small grain cereals [9]. The ultimate goal is to achieve genetic gains in agronomic and selected end-use quality traits in wheat worldwide [10] to feed the growing human population.

Since Fusarium Head Blight, commonly called scab, is a major pest to wheat crops, breeders strive to find achievable solutions by producing wheat varieties with resistance genes. Indeed, the plant defense responses to *Fusarium* have been thoroughly explored,

thus genomic selection in cereals has resulted in improved crop resistance which has now surpassed the efficiency of the traditional phenotypic selection in controlling FHB [1,11]. However, breeding against FHB seems to be hampered by the limited variation in the elite gene pool and difficulties in efficiently combining the numerous small-effect resistance quantitative trait loci (QTL) in the same crop line [11]. In addition to this, while historic breeding panel enables high-resolution mapping of FHB resistance QTLs [12], the *Fusarium* infection-induced metabolic changes in different cereal varieties associate with different physiological, metabolic and phytohormonal network-related resistance [13–15]. Even with all this information, the shifts in crop defense responses have not yet been exploited for an integrated scientific strategy or management approach in controlling FHB. Furthermore, despite multiple genomics and functional traits [16,17] and their relation to the evolution of disease resistance in cereals [18], it is still unclear how resistance works.

Even though it is not clearly known if other plant associated traits such as microbiome-based wheat characteristics are connected to host resistance, it is believed that the microbiome as the plant's second genome can reduce *Fusarium* virulence [19]. Studies have found that the microbiome of wheat can disable *Fusarium* pathogen infection [20] or can confer resistance [21] and so without genetic modifications. In addition, *Fusarium* whole genome and specifically its pathogenome features have been targeted to reveal gene sequences, gene order, gene clusters, regulatory sequences, and other genomic organisational landmarks. It is possible that these revelations can provide a better understanding of the complex dynamics of host-microbe interactions that lead to diseased versus healthy plants [22]. From the pathogen perspective, virulence mechanisms, such as the production of toxins and virulence proteins, as well as pathogen reproduction and survival, have also been studied in connection to climate change [23]. On the other hand, microbiologists and plant pathologists argue that a better understanding of cereal host genetics-*Fusarium* genetics-beneficial microbes interactions is possible by looking more closely at FHB from a microbiome perspective [24]. They propose that findings from the latter perspective can eventually lead to the development of new control options for this global disease. So far, contemporary science has observed the differences in resistance between wheat genotypes with different *Fusarium* pandemic lines and associated protooperative microbial populations, such as *Fusarium*-specific biocontrol agents (BCAs) for developing more resistant crop varieties. Can we take advantage of the BCA-based resistance in elite wheat genotypes without gene modification or plant gene editing in combatting FHB?

2. *Fusarium* Head Blight (FHB)

2.1. FHB and Crops

FHB was first described by Worthington G. Smith of England in his 1884 book, *Diseases of Field and Garden Crops* [25]. Since then, this disease that mainly affects small cereal grain crops has been better characterized from a brief but detailed description of the symptomology in W.G. Smith's book to a complex disease that has had a devastating effect on the cereal crop industry worldwide over the last century. With increased human migration around the globe, crops such as wheat grew in prominence and so did FHB colonisation of wheat and other crops. This disease is now endemic to certain parts of Africa, Asia mainly China, Europe, South and North America leading to major outbreaks in North America in the early 20th and 21st centuries [26–33]. The first Canadian case of an FHB outbreak was reported in 1919 [34]. Losses in eastern Canada date back to at least the early 1940s. Western Canadian fields across Manitoba were first affected in 1923, although no serious outbreaks were reported until 1984 [35]. After these events, the contemporary survey data of FHB prevalence and severity in wheat crop started in 1987 throughout Canada [34], a few years after reports of serious outbreaks were made. Since then, survey data has been continuously updated to reflect all FHB outbreaks and epidemics in Canada.

However, data collection and breeding for resistance remain complicated because the FHB disease in cereals is caused by several *Fusarium* species. Some of the more commonly known species include, *F. chlamydosporum*, *F. boothii*, *F. scirpi*, *F. arthrosporioides*, *F.*

poae, *F. avenaceum*, *F. culmorum*, *F. graminearum*, *F. verticillioides*, *F. asiaticum*, and *F. cortaderiae* [36]. Over the years, the complex has been regularly updated with the use of current technological advances such as multilocus genotyping (MLGT), polymerase chain reaction (PCR) and next-generation DNA sequencing platforms [3,37–40]. Recent molecular advancements revealed the existence of *Fusarium graminearum* species complex (FGSC), also known as *F. graminearum sensu lato* with 16 formally described phylogenetically distinct species [3,4,39,40]. These are among the most important FHB causative species on a global scale. North and South America (Argentina, Brazil and Uruguay) registered *F. graminearum sensu stricto* as the primary aetiological agent of FHB outbreaks over the last decades [26–33,41]. Other important *Fusarium* species in North America includes *F. culmorum* and *F. poae* [37,42]. In other world agroecological zones, the most abundant species strongly correlate to geographical location. In Eastern Asia, in the cooler regions of North Japan, *F. graminearum* is the predominant species in diseased crops, while in the more arid southern region *F. asiaticum* dominates [43]. A similar trend is also seen in China, in which *F. graminearum* is the most abundant pathogen in diseased cereal crops in the North and *F. asiaticum* dominates the South of China [44]. In Europe, a complex of species, *F. graminearum*, *F. avenaceum*, *F. culmorum*, and *F. poae*, are mainly associated with FHB; however, in most cases, it was reported that *F. graminearum* was the primary cause of FHB in many European countries [45]. On the African continent, in countries such as Kenya, *F. graminearum*, *F. poae*, and *F. chlamydosporum* [46] are the prevalent species, while a novel species *F. aethiopicum* that is frequently the cause of FHB, is registered in Ethiopia [3].

2.2. FHB in the US

Fusarium graminearum literally ‘fusarium of the grasses’ [26] has been named among the most threatening plant pathogenic fungus in the world because of its aggressiveness and its economic importance [47,48]. It threatens some of the major global producers of wheat that are coincidentally in some of the most FHB prone areas. In these regions severe FHB epidemics occur at a minimum of every fourth or fifth year [49] resulting in great losses. A concise history of major epidemics in North America is shown in (Figure 1). Dating back to the early 20th century, the US saw a steady increase in FHB epidemics that was mostly concentrated in the Eastern, Northern, and Central Midwest states of Iowa, Ohio, Indiana, and Illinois [28,29,33]; however, the swift spread of FHB epidemics of 1917 and 1919 did not serve to many as a harbinger of the indomitable and imperceptible nature of this disease. At the turn of the new decade, in the 1920s, the disease then quickly raced its way into many wheat fields of the Upper Midwest states of Minnesota, North and South Dakota, and the East South-Central state of Kentucky before resting comfortably but disastrously in the Midwest [33]. The mid of the 20th century saw a well needed reprieve from the devastating epidemics of the last decades; however, this reprieve was short lived and frequent FHB epidemics returned in the 1970s and extended its grip into the Northeastern and South Atlantic provinces of Pennsylvania, Maryland, and Virginia [33].

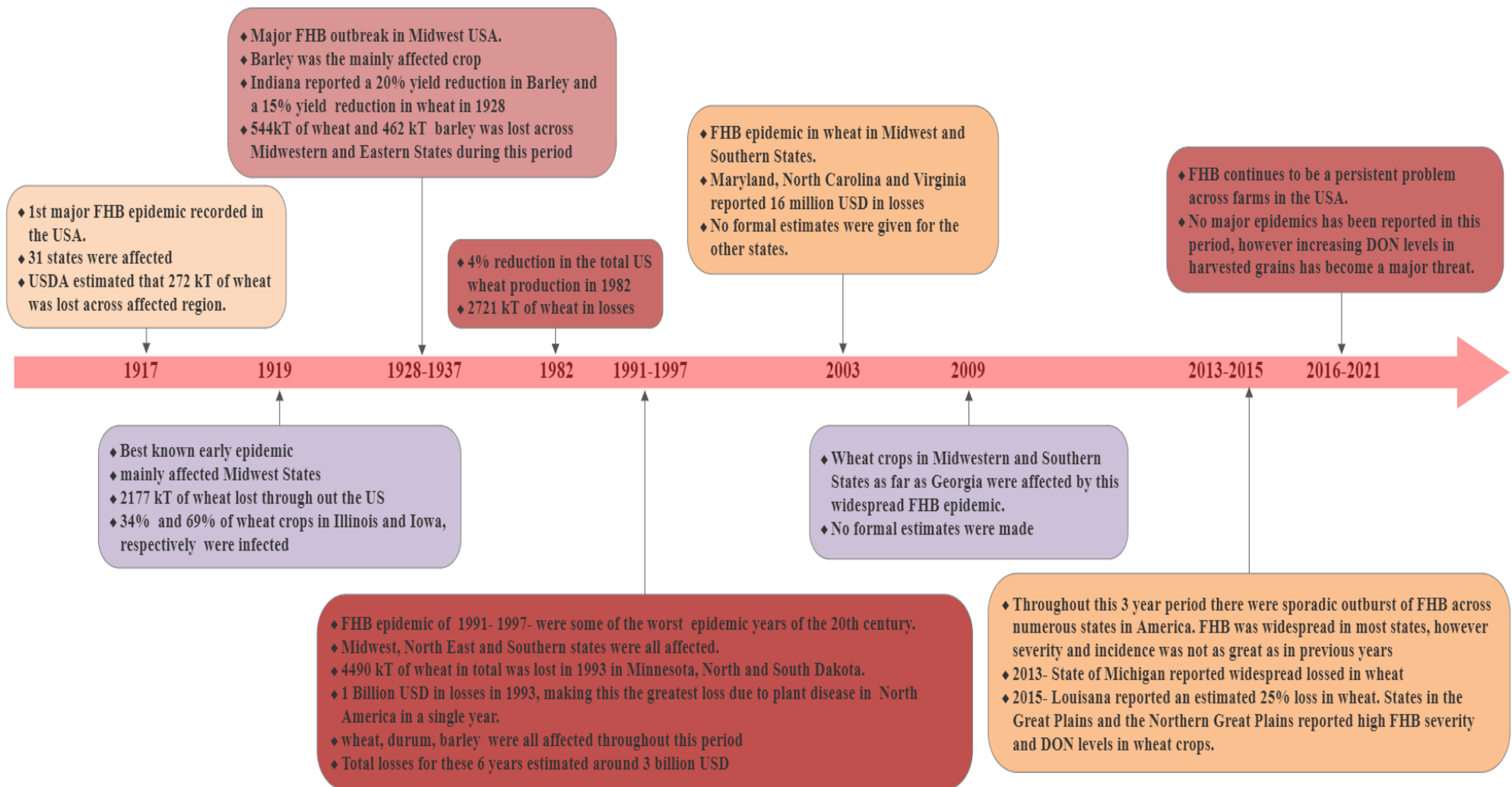


Figure 1. A timeline of the major Fusarium Head Blight (FHB) epidemics in the USA over the last 100 years. A brief description of the major FHB epidemics in the USA covering a period of 107 years. The diagram above is a condensed version showing FHB epidemics from 1917 to 2007.

By the 1980s FHB epidemics were reported in as far as New York. The epidemic of 1982 resulted in a 4% reduction in the total US wheat production equivalent to 2.72 million metric tonnes or 100 million bushels [28]. This was by far the worst epidemic the US had experienced since the epidemics of 1919 and 1928, the latter of which mostly affected barley crop resulting in significant international trade disturbances [28,33]. As the curtains closed on the 20th century the frequent outbreaks and epidemics had scarcely left any wheat-producing states unscathed. FHB in the 1990s would go on record to be some of the worst epidemic years both in terms of crop damage and economic losses in North America and the global wheat industry [26,28,29,32]. By this time FHB outbreaks had touched most if not all the cereal crop producing states in the US. The unparalleled and social impacts caused by the 1993 epidemic has been described and documented in numerous articles [26,28,33,50] to mention a few. The 1993 epidemic resulted in an estimated \$1 billion USD loss to producers in the tri-state areas of Minnesota, North and South Dakota, making it the greatest loss compared to any other plant disease in North America in a single year [28]. From then on at the close of the 20th century, FHB wreaked havoc on wheat, durum wheat and barley in the US which also extended to the Canadian prairie province of Manitoba. At the end of this prolonged epidemic period, it was estimated that over 500 million bushels or 13.6 million metric tonnes of wheat valued at \$2.5 billion was lost [28], which at present is approximately equivalent to 4 years supply for food and industrial use, based on the 2015–2019 consumption data from Statistics Canada and Agriculture and Agri-Food Canada [51].

The economic significance and cataclysmic loss caused by this disease overshadowed the disastrous epidemics just a decade earlier in 1982, in which it was estimated that 100 million bushels or 2.72 million metric tonnes of wheat was lost due to FHB [50,52]. Losses resulting from the 1991–1996 FHB epidemics were estimated to be around 7.67 billion USD, the most expensive loss to date [9,29]. At the dawn of the 21st century the science of plant pathology was better prepared to deal with FHB. Different management strategies were implemented to treat this complex disease and even though the outbreaks and epidemics did not stop and by this time had extended south to Georgia and North Carolina, farmers were not fighting FHB empty handed as they had been in previous years. Armed with new fungicides, new cultural practices, improved and more resistant cultivars as well as the promise of natural enemies of these FHB pathogens, it seemed producers would be able to put up a worthwhile effort against this highly intricate disease. In the words of Robert W. Stack “There is every reason to expect that the history of *Fusarium* Head Blight of the first decades of the 21st century will be quite different from its history in the 20th century”. This was a fair assessment, and this was the case for the first two decades of the 21st century. FHB history and particularly its control was different due to new and improved integrated management strategies. The current era of advanced technology has allowed for the discovery and greater understanding of many new FHB pathogenic species. The predominant FHB agent in North America *F. graminearum* Schwabe genome has been mapped [53], resistant cultivars have been developed [47,54], cultural practices have been changed and possibly improved, and new fungicides both chemical and more recently biological are all a part of the integrated management strategy [55]. However, irrespective of all these methods FHB and its causative agents remain a dominant threat to the world’s number three food crop. In fact, every year infection of wheat by the *Fusarium graminearum* results in losses of ~28 million metric tons of wheat grain [56], valued at \$5.6 billion [57].

2.3. FHB in Canada

The history of wheat cultivation and FHB in Canada is not as extensive as that of its neighbour America. The first grain of wheat was sown in Canada in the year 1541 and it would take a little over 200 years before wheat cultivation became an established crop in Canada [58]. Nevertheless, Canada has seen and experienced devastating losses and a steady increase in FHB incidence and outbreaks over the last decades. Fortunately, knowledge and records of the USA’s history of FHB and the relatively short cultivation history of wheat in Canada has allowed for up-to-date documentation of FHB incidence and

outbreak in wheat, durum and other cereal crops. In a 2003 article “History of Fusarium Head Blight with an emphasis in North America”, R.W. Stack mentions that the FHB epidemic of 1919 in America extended to the Canadian prairie provinces of Manitoba and Saskatchewan as reported in the Cereal Crop Disease Report by W.P. Fraiser from University of Saskatchewan [33]. However, numerous articles including the Canadian Grain Commission (CGC) and the Canadian Phytopathological Society (1923), recorded that *F. graminearum*, the main aetiological agent in most FHB cases, was first registered on corn stubble in Manitoba [58]. Since then, *F. graminearum* and other pathogenic *Fusarium* species have been isolated from diseased cereal crops, including wheat and barley. While the disease in Canadian grains has not been as devastating as it has been in America, moderate widespread FHB epidemics were reported once every 9 years in Southwestern Ontario from 1927 to 1980 [26,42]. Losses to the Canadian grain industry during the 1990s totaled US \$200 million for Ontario and Quebec and US \$300 million for Manitoba [50]. In addition to the huge economic cost, the wheat industry over the last decades has witnessed a shift and spread of FHB from East Canada to the Canadian prairie provinces, the main producers of common wheat and durum wheat in Canada.

Accompanying this shift and spread of FHB is a steady increase in mycotoxigenic *Fusarium* pathogens that have spread from the Southeastern Manitoba across to Saskatchewan, Alberta and British Columbia over the last few decades [59–61]. Figure 2 gives a brief outline of the major FHB epidemic years in Canada and the numbers of scientific papers that referenced FHB epidemics over a 35-year period from 1985 to 2020 as shown in data retrieved from the Web of Science Database (WSD, 2021). The increase or decrease in the production of scientific papers on FHB epidemics in Canada can be divided into three phases, (i) 1985–1997, the slow growth phase, (ii) 1997–2007, the exponential growth phase, and (iii) 2008–2016, the extensive growth phase, (Figure 2).

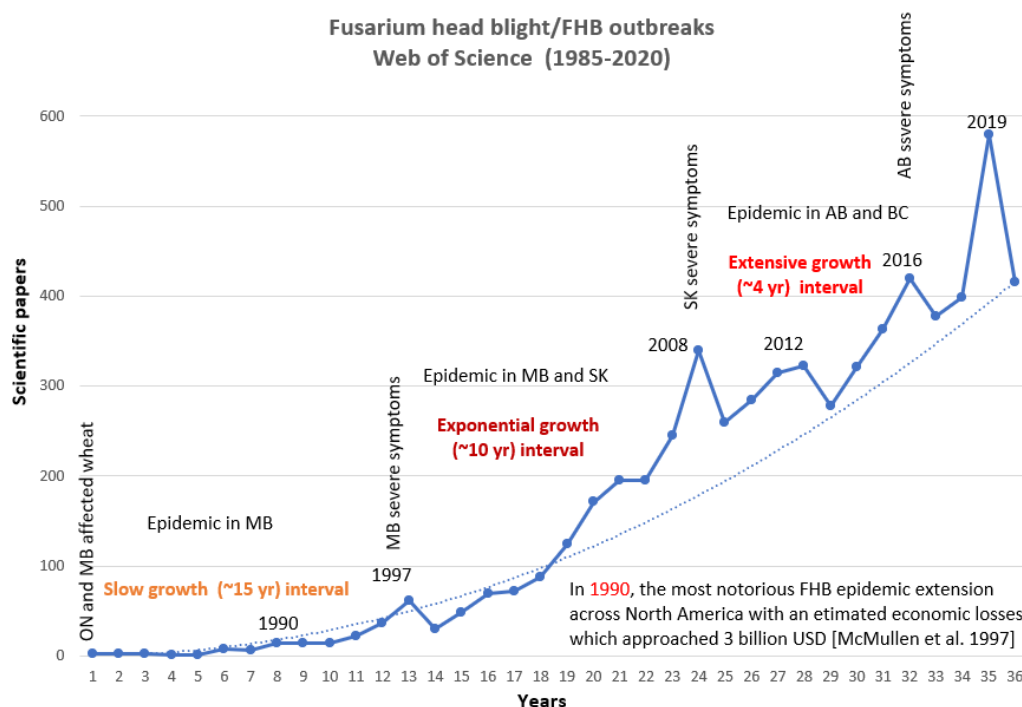


Figure 2. FHB progression and history in Canada as reflected in scientific papers from the year 1985–2020 found on the Web of Science database-WSD, (i) phase 1—slow growth years 1985–1996, (ii)—exponential growth years 1991–2007, and (iii)—extensive growth years 2008–2016.

Phase 1, from 1985–1997, corresponds to the slow growth of the output of information on FHB disease in crops in Canada. During this time FHB woes were mostly seen and experienced in Ontario and in Manitoba, with a stand-out epidemic of the 1990s that would for the first time in a decade remind cereal farmers of the dreadful nature of this disease. Over 20 million bushels of wheat was lost in Manitoba in the year 1993 [28]. Compounding this great loss was the unfortunate reality that FHB was now slowly but strongly moving from the East to the West across Canada, first to the neighbouring province of Saskatchewan. Even though this was the early stages of FHB in Canada, all this would quickly change as FHB moved into the exponential growth phase over the next decade from 1997–2007. The exponential growth, identified as phase 2, of FHB across the prairies was mostly seen in the provinces of Saskatchewan and Alberta. During the same period there was also an increase in the number of scientific articles that were being produced based on data from the Web of Science database (2021) [62] (Figure 2). A decade earlier, just over 1% of the total scientific papers available on FHB were produced in the slow growth phase compared to 27% of the current total that reported on FHB in the exponential phase. Numerous agencies including universities, private and governmental agricultural firms busied themselves finding different and effective controls for FHB. During this time FHB management in cereal crops became one of the priority research areas in plant diseases. However, while progress was being made FHB's spread and evolution was a few steps ahead and had already made its way to Alberta, thus completing its sweep across the prairie provinces and reaching British Columbia. The third phase, the extensive phase, lasted approximately 8 years from 2008–2016. A total of 70% of all scientific papers on FHB as shown by Web of Science database (Figure 2) was produced in this period. The papers covered the increasing epidemics across Canada. It could be argued that there was a better understanding of the disease and different management strategies were being investigated and deployed, which was also reflected in the massive output of scientific articles. However, there was still a great hurdle to controlling *Fusarium* spp. mycotoxins, that are hallmarks of FHB disease. As a result of this, nationwide research was aimed at understanding how to control these mycotoxins which also generated a large volume of scientific articles during this period. Since 2016 FHB has remained an important threat to common wheat, durum wheat and other cereal crops not only in Canada but globally and the search continues with the hopes of finding methods to effectively control this disease and associated mycotoxins.

3. Mycotoxigenic *Fusarium* Pathogens

In Canada and North America as well as many other regions in the world, *F. graminearum* is the predominant pathogenic species that causes FHB in wheat and barley [33,47,55]. *F. graminearum* is a highly virulent hemibiotrophic pathogen that produces type B trichothecenes, deoxynivalenol (DON) and its acetylated derivatives 3 ADON and 15 ADON, nivalenol (NIV), and zearalenone (ZEN) [56,63–66]. These are some of the most important fungal toxins that have been known to contaminate cereal grains and other crops that are prepared for human and animal consumption. DON also known as vomitoxin, is the most observed toxin in Canadian grain [60,67]. Several studies have described the adverse health effects experienced by humans and animals upon exposure to varying levels of DON [33,48,55]. DON has been linked to feed refusal and weight loss in pigs [33,67]. In humans, DON has been identified as the causal agent in vomiting, diarrhea, neural disturbances [27], liver and kidney damage, immune and nervous system impairment [27] after the consumption of contaminated food.

In addition to its adverse effects on mammals, DON has been reported to increase the virulence of *F. graminearum* on wheat and durum [67–69]. Canadian prairies, studies on wheat and durum showed that there was a significant relationship between DON levels and visible *Fusarium* Damage (FUS DMG) in Canadian Western Amber durum (CWAD) and most Canadian Western Red Spring (CWRS) [61]. The FUS DMG and DON relationship is currently used as grading factor for grains produced in Canada as implemented by the Canadian Grain Commission (CGC) [61,70]. However, conclusions on the FUS DMG

and DON relationship as a gold standard grading factor for harvested grains remain equivocal, since different studies have shown that the relationship is not always linear [71]. In the same study carried out in the Canadian prairies, Tittlemier et al. (2019) reported that the relationship between FUS DMG and DON was at times variable [60]. In one of the wheat districts, the low concentration of DON measured did not correspond to the high level of FUS DMG seen in CWRS of that district. Additional data showed that a variation in *Fusarium* species resulted in a variation of mycotoxins detected at any given time. This variation was attributed to prevailing environmental conditions such as precipitation and high temperature in the growing year which was ideal for the growth of some *Fusarium* species over others. Indeed, visible FUS DMG is not always correlated to DON concentration, signifying that species other than *F. graminearum* and *F. culmorum* can produce visible FUS DMG [61]. These findings suggest that to effectively combat or implement integrated strategies to manage FHB and *Fusarium* mycotoxins, there is a necessity for a more centralised reporting on mycotoxin surveillance in Canada [72]. Numerous reports will be able to give a clearer picture of the correlation, if any, between the diversity and abundance of *Fusarium* species across the prairies and the mycotoxins produced and their concentration in wheat and durum. This information can then be used to establish a range for the concentration of *Fusarium* toxins that can be tolerated or can be detrimental to animal and human health.

In addition to type B trichothecenes such as DON, a plethora of other mycotoxins including Type A trichothecenes and Fumonisin are prevalent *Fusarium* species mycotoxins that are observed in infected cereal grains. Deoxynivalenol- DON is mostly commonly produced secondary metabolite of (*F. graminearum* and *F. culmorum*), Fumonisin—FUM of (*F. verticillioides* and *F. proliferatum*), aurofusarin -AUS (*F. graminearum*, *F. poae* and *F. sporotrichoides*), type A- trichothecenes, T-2 and HT-2 are commonly produced by (*F. poae*), beauvericin and enniatins (*F. avenaceum*, *F. poae* and *F. sporotrichoides*), and moniliformin (*F. proliferatum* and *F. subglutinans*) [2,9,27,73]. However, the type B trichothecenes, DON and its acetylated derivatives 3 and 15 ADON produced by *F. graminearum* and *F. culmorum* are the predominant mycotoxins found in cereal grains and, hence, a better understanding of these toxins and how to reduce them in grains is of vital importance to the stability of the cereal crop production sector and agri-food industry.

In the last two decades, the highly aggressive 3 ADON chemotype has gained more prominence and has rapidly replaced the native 15 ADON chemotype in North America [6,74]. The more virulent 3 ADON chemotype showed higher levels of DON biosynthesis upon *Fusarium* infection [6,75]. Its emergence on the Canadian wheat scene coincides with reported >50% kernel infection rates for some Saskatchewan farms in 2012 [75]. The Canadian Grain Commission (CGC) in 2017, also reported that in the year 2016, several Saskatchewan districts accounted for >90% infected spring wheat samples, which is about 70% increase compared to 2010 (CGC, 2017). In addition to this, *Fusarium* is predicted to cause the downgrade of about 40% of Saskatchewan's durum, with about 25% likely to be downgraded to below food grade [76]. These factors have highlighted the severity of this disease in Canada. Therefore, understanding how and what affects the shift in type and concentration of mycotoxins produced by different fusaria can be very useful in mounting a sustainable management strategy in controlling toxigenic *Fusarium* species complex and related FHB damage.

4. FHB Management

In Canada, US, China, Europe, and other leading wheat-producing countries worldwide, the preventative measurements of FHB and *Fusarium* mycotoxins are usually categorised as pre- and post-harvest treatments. Some of the most common methods include agronomic approaches, such as crop rotation and no-till, breeding and host optimisation by means of genetic modification, chemical and biological treatments, and FHB forecasting. Additionally, in the last two decades, a handful of natural parasites of the *Fusarium* fungi have also been explored [9,19,29,47–49,54,65,76,77]. Presently, there are countless

articles, reviews, and commentaries that collectively provide an exhaustive coverage of the different FHB management strategies that have been used over the past five decades. A quick and comprehensive guide to some of the most popular and effective methods can be found in the following articles by Gilbert and Tekauz in 2011 [50], McMullen 2012 [29], Wegulo 2015 [55], Legrand 2017 [48], and Mielniczuk 2020 [9]. In these articles, most of the traditional management strategies mentioned have yielded positive results, however, none of them so far has been considered as a sustainable treatment for FHB [78–80].

4.1. Chemical and Physical Control

Chemical treatment including the widely used demethylation inhibitor (DMI) class fungicides including tebuconazole (Folicur), prothioconazole plus tebuconazole (Prosaro), and metconazole (Caramba) results in no greater than 60% control of FHB and 30% to 50% of DON in wheat, although the actual reductions are highly variable [29,81]. In addition, multiple pesticide constraints have been reported including inadequate coverage and timely application, creating significant challenges for farmers [29]. Newer fungicides such as phenamacril, a cyanoacrylate compound that asserts its antifungal effect on susceptible *Fusarium* by disrupting the activity of essential actin-motor protein have been shown to be more effective against *Fusarium* spp. than DMI class fungicides [82]. However, field-resistant *Fusarium* strains to phenamacril have been characterised [83]. There is also a newer fungicide pydiflumetofen, a novel succinate dehydrogenase fungicide that was released in 2018 [83]. It appears effective against *Fusarium* and has been used on crops in America, Canada and China [84]. As a novel fungicide, its efficacy is still being tested. However, as history has shown, overuse of these chemicals eventually results in fungicide resistance [76,85–88]. There are also concerns about toxicity and related health and environmental issues [65,78,89].

Over the years, alternatives to reduce the dependence on toxic synthetic chemicals have been explored. In terms of *Fusarium* toxins, mycotoxin adsorption agents are promising; they bind to mycotoxins in the gastrointestinal tracts of animals thereby decreasing their bioavailability and reducing any potential harm [90]. However, while this seems to be a promising alternative, a study by Zain et al. [91] reported on various types of adsorption agents with varied efficacy. Furthermore, according to Stoev et al. [92] adsorption agents are effective in preventing aflatoxicosis, but they are not very effective for other mycotoxins. Other alternatives include optimising host resistance which is favored by many scientists including Figueroa et al. [49], who states that host genetics would be the most desirable approach to controlling FHB and preventing toxins accumulation and associated health risks.

4.2. Host Resistance

Host resistance has been touted by many scientists throughout the years as the most desirable approach in controlling FHB in cereals and other crops [33,49,93–97]. However, despite screening thousands of wheat lines, little resistance to *Fusarium* has been found [98]. In addition, optimising host resistance in wheat and durum has not been an easy feat as resistance to FHB is a complex and quantitatively inherited trait that is controlled by multiple genes and affected by environmental factors [47] leading to relatively slow genetic gain per unit time [65]. Nevertheless, despite all these impediments, host or genetic resistance is considered the safest and most cost-effective tool for providing sustainable control against FHB. Following Astanoff's observation on the differences in resistance among different wheat varieties, and the conclusion that durum wheat was more susceptible than bread wheat to FHB [99], a genuine interest in FHB resistance grew worldwide. This ushered in a new era of host resistance research and cultivar improvement studies. Since then, the insights into host resistance have been forthcoming and thousands of scientific results have been published [2,7,31,93,94,97–99]. Globally, numerous governmental and private breeding, screening, and genotyping labs and organisations have also joined the fight against FHB. These efforts over the last decades utilizing different strategies have led to the

development of numerous wheat (*Triticum aestivum*) and a few durum (*Triticum turgidum* subsp *durum*) cultivars with varying levels of resistance [100].

At the start of this era in harnessing host plant, resistance mainly came through conventional breeding programs in which germplasm of *allohexaploid* common bread wheat and to a lesser extent *tetraploid* durum wheat were screened for desirable FHB resistant traits. The US is said to have been one of the earliest countries to screen for FHB resistant germplasm dating back to the 1920s with experiments of FHB severity in different wheat varieties carried out at the University of Wisconsin by Dimitr Astanoff, [99]. Other countries like China, prompted in part by FHB epidemics in the 1930s in different regions of country, conducted one of the largest nationwide screenings backed by the governmental organisation, All Corporation of Research on Wheat Scab (CCRWS) in 1974 [101]. According to Ma et al. [101] the CCRWS surveyed a total of 34,571 accessions including 23,434 domestic and introduced accessions of common wheat and 1935 accessions of wheat relatives. Among the common wheat, 1796 or 5.2% accessions were identified as resistant or moderately resistant [101]. However, there was no specific mention of durum wheat, even though it is possible that tetraploid wheat could be among the group labelled relatives of wheat. The same source also reported that 70,000 accessions of hexaploid wheat and its relatives collected worldwide have been screened, leading to the identification of about 7000 accessions with different levels of FHB resistance. In durum and wheat subspecies and its close relatives including Emmer, Oats, and Einkorn, approximately 10,500 accessions have been screened for their resistance to FHB. However, there are not many accessions identified as having or showing varying levels of FHB resistance [101].

4.3. Conventional Breeding

Conventional breeding based on phenotypic selection was one of the main tools of improving resistance for most of the 20th century. It has led to the development of some very important FHB resistant wheat cultivars such as Sumai-3. Sumai-3 is a Chinese spring wheat cultivar that has been and is still extensively used in breeding programs worldwide [29,54,89,90,101–103]. It was the first FHB resistant cultivar to be developed in China in 1947 from a cross of a moderately susceptible, Italian wheat variety, Funo and a resistant landrace (Taiwanxiomai) from Taiwan [26,54,104]. Since then, Sumai3 cultivar (cv). has been instrumental in the development of other resistant cultivars such as Ning 7840 [104]. Sumai3 is also a major source of resistance in wheat and durum breeding programs in the USA and Canada, contributing to the development and release of more than 20 modern cultivars. These include commercially available moderately resistant (MR) hard spring wheat cultivars Alsen, Glenn, Barlow, and SY Ingmar from North Dakota, Faller and Prosper from Minnesota, [54] and AAC Brandon a spring wheat cv. from Canada [54]. Another Italian cultivar Montana and Brazilian cultivar Frontana have also contributed to the improvement of FHB resistance in North America [101,103].

Conventional breeding has also played a part in the identification of one of the first major FHB resistance quantitative trait loci *Fhb1*, which was first mapped in Sumai 3 by DJ Somer's laboratory [101,105]. The *Fhb1* gene has been validated in different studies [54,81,103–105] and is located on the long arm of Chromosome 3B and has been shown to confer Type II resistance—resistance to fungal spread according to the mechanisms of resistance first described by Christensen and Schroeder, 1929 [106]. *Fhb1* resistance is widely used in spring wheat (hexaploid) cultivars and has been shown to contribute (20–50%) resistance to FHB in bread wheat compared with their parents [93,99].

At the end of the 20th century conventional breeding had established a solid foundation in the area of FHB resistance in wheat and other crops. Undoubtedly, this era in host resistance has led to a better understanding of the genetic control of FHB. It has also led to the development and release of not only, the well-known spring wheat cultivars (cvs). such as Sumai-3 and its derivatives from China, but others such as Nobeoka Bozu from Japan, and Brazilian cultivar Frontana. that was mainly used in winter wheat breeding programs in Canada in the late 20th century. Research in host optimisation has resulted in

creation of winter wheat *cvs.* such as Praag 8 and Novokrumka from Europe [31] as pioneer resistant cultivars in the fight against FHB aggressiveness. However, these advances have encountered limitations due to the lack of good germplasms and major resistance genes, polygene nature of FHB resistance, and the difficulty in disease resistance evaluation [101]. In addition, it takes up to 10 years to develop a new resistant cultivar [107]. At this stage of the research, breeders are just not able to afford the costly consequences of delays in getting more resistant cultivars on the global market [107]. The disastrous FHB epidemics of the 1990s and early 2000s decimated wheat and durum crops in the US and Canada. Indeed, the seemingly effortless way in which *Fusarium* pathogens were able to circumvent and render ineffective the most up to date control measurements of the time underscores the urgency to have improved cultivars on the market and in time to prevent the next FHB attack. As a result of this, conventional breeding alone has not and will not be able to mitigate this disease. Therefore, the race to produce more resistant cultivars has to be continuously supported with newer technological tools such as GS (genomic selection), MAS (marker assisted selection), and MARS (marker-assisted recurrent selection [108–111]. With these innovative tools and interactome research [20] that are currently being explored there is a high possibility that more resistant *cvs.* will be commercially available for both common wheat and durum wheat in the near future.

4.4. Resistance in Common Wheat

Marker-assisted selection (MAS), which is based upon the establishment of a tight linkage between a molecular marker and the chromosomal location of the gene(s) governing the trait to be selected in a particular environment, uses modern genetics to speed up the traditional process of conventional breeding. Unlike conventional breeding where the progeny must be grown and observed for the desired trait before a selection can be made, MAS can be done at any stage in the breeding programme and allows selection for and pyramiding of resistance genes at the absence of the pathogen [79]. As a result of this, the time to develop improved cultivars can be halved. Several studies have reported [111,112] the success of the genetic tools in improving FHB resistance. The first FHB resistant cultivar Sabin a hard red spring wheat developed by MAS at the University of Minnesota Experiment Station was registered in 2009 [113]. The efficacy of MAS relies on QTLs that produce large and stable effects and the availability of makers to validate these QTLs. So far studies have reported many *Fhb* resistance QTLs in wheat and durum wheat [94,105,113–116]. Of these, seven (7) with varying levels of resistance have been formally assigned with a gene name. These are *Fhb1* [105], *Fhb2* [117] *Fhb4* [118,119], *Fhb5* [120,121] all from common wheat cultivars and *Fhb3*, *Fhb6*, and *Fhb7* all from wild relatives of common wheat. *Fhb1* is the most studied QTL and confers the greatest FHB resistance as confirmed by many studies [74,94,105,116]. However, there is no single QTL that confers complete FHB resistance further underscoring that FHB resistance is a complex trait that is controlled by multiple genes. Even though breeders have had success in selecting for FHB resistance with MAS, the complex genetic architecture of FHB resistance, difficulties in precision phenotyping, and delayed availability of high-density genome-wide molecular markers in wheat have hindered progress in breeding for FHB resistance. Therefore, newer methods such as genomic selection (GS) that estimates the value of all markers or QTLs in the entire genome, has been shown to be a more effective marker-based strategy when breeding for complex quantitative traits such as FHB resistance [121–123]. Moreover, GS can be combined with MAS and MARS, and for large effect QTLs.

4.5. Resistance in Durum Wheat

It has been long established that durum wheat (*T. turgidum*) is more susceptible to FHB than the common bread (*T. aestivum*) wheat. Even though the reason(s) for this is not clearly known and is yet to be elucidated, several plausible explanations have been put forth. Firstly, durum production for a long time was limited to the fertile crescent and semi-arid environments where FHB was not a problem. Secondly, in comparison to

bread wheat, durum wheat is cultivated on a smaller scale. Gill et al. [124] reported that durum wheat accounts for about 4% of the total wheat production worldwide. Another possible explanation hints at the narrow genetic variation for FHB resistance in durum wheat elite germplasm as an important challenge [125]. It has been hypothesised that the D genome of hexaploid wheat plays a possible role in reducing FHB severity in wheat. Szabo-Hever et al. [126] reported that synthetic hexaploid wheat lines (SHW), generated by crossing tetraploid wheat (*T. turgidum*) with *Aegilops tauschii*, were more resistant than the *T. turgidum* parents.

Even though tetraploid wheat such as durum lacks a set of D chromosomes [121], researchers and breeding centers worldwide have been looking into exotic and native sources to find ways to improve FHB resistance. Thus far numerous studies have shown promising results for FHB resistance in durum [127,128], however, these studies have also confirmed that it is more challenging to improve resistance in elite durum wheat than it is to improve resistance in elite bread wheat cultivars. One of the major tools used to enhance FHB resistance in wheat and durum over the last decades, QTL mapping, has resulted in the detection of numerous QTLs for FHB resistance. However, in durum and other tetraploid wheat only a few small effects QTLs have been identified [93,125,129]. For instance, the *Fhb1* gene has been transferred to different bread wheat lines but for years introgression into elite durum lines has not been an easy feat [129]. Nevertheless, recent reports [126,129,130] have confirmed the successful introgression of *Fhb1* gene from bread wheat to durum wheat. This result is promising for the future of FHB resistance in durum, and as new sources of resistance are being discovered it is conceivable that with the advance technology such as genomic selection, genome editing, transgenics and others, improved durum cultivars is within reach. It can also be expected that commercial wheat and durum cultivars, with substantially higher defence against FHB, will be released in the near future.

In the last three decades the area of host resistance exploration has led to some great achievements especially in the development of highly resistant cultivars such as the previously mentioned Sumai-3 and Ning 4870 [131–133], predecessors to Canada's AAC Brandon. AAC Brandon is one of the most resistant spring wheat cultivars currently used in Canada and widely grown across Canadian Prairies [54]. However, Wegulo et al. [55] have shown that even these current resistant varieties are still not immune to the catastrophic effect of FHB. Furthermore, ideal resistance in elite commercial wheat has not been achieved yet [7], due to the extremely complex nature of FHB resistance traits. As a result of all these constraints with these current management strategies, FHB and *Fusarium* mycotoxins remains a recalcitrant problem worldwide. Hence, there is an ever pressing need to combine all possible strategies old, novel, or improved that can stand alone or be integrated with other current management strategies to effectively stand a chance of minimising the effects of this disastrous disease.

4.6. Biocontrol

Another appealing strategy is to incorporate the microbiome approaches in contemporary global breeding programs [134]. Currently, the importance of plant pathogens and the plant microbiome interactions have been increasingly recognised with an emphasis on pathogenic and mycotoxigenic *Fusarium* species [24]. From a microbiome perspective, there is an increasing interest in using biological control against plant pathogens. Biocontrol broadly refers to the use of living organisms to curtail the growth and proliferation of other undesirable ones [66,135]. These organisms, particularly microorganisms, reduce the survival or activity of a pathogen through suppression, inhibition, control or reduction of the pathogen and its metabolites and are aptly called biocontrol agents (BCA) [48,66,136]. Pal and Gardener [137] reported that the mechanism of biological control includes different types of interaction between fungal BCAs and their fungal hosts. According to Vujanovic, these interactions include direct interaction which can be either mycoparasitism or hyperparasitism; mixed path interaction, which includes antibiotic secretion, lytic enzymes, and

indirect interaction, which includes competition for nutrient or space and induction of host resistance [66,138]. These different interactions offer varying levels of protection, and it has been surmised that the most efficient BCA will employ a combination of these different interactions for pathogen control at any given time [66]. In addition to their protective effective against plant pathogens, BCAs are considered a more natural and environmentally acceptable alternative to current chemical fungicides.

Numerous studies have shown that BCAs control FHB development and mycotoxins, in both preharvest and postharvest agriculture systems, and sometimes with plant growth promoting (PGP) abilities to wheat crops [48,139–141]. BCAs have also been able to protect seeds from pathogens by forming a coat around the seed [142,143]. Overall, BCAs are able to offer a holistic and semi-flexible treatment approach that provides greater coverage against FHB and *Fusarium* mycotoxins throughout the life cycle of a plant, from the seed stage to end use products. This broad protective coverage cannot be achieved with the most effective fungicide due to legal limitation concerning application times and samples [144]. Over the years different BCAs including bacterial BCAs such as *Bacillus* sp., *Pseudomonas* sp., *Actinobacter* sp. as well as fungal BCAs such as *Trichoderma* spp., *Clonostachys rosea* and *Sphaerodes mycoparasitica* have been studied [145–151]. These studies have revealed tremendous potential of BCAs in reducing the effects of FHB. However, there is a shortage of commercial BCA products that can be used along with current FHB management strategies. Among BCAs explored in the last two decades, *Trichoderma* spp. and *Gliocladium roseum* have been the most extensively studied for application in FHB treatment worldwide.

Trichoderma spp. (telomorph *Hypocrea*) are rapidly growing saprophytes isolated for the first time in 1794 from soil. According to Sood et al. (2020), >60% broadspectrum biofungicides are obtained mainly based on *T. harzianum* and *T. viride* isolated from pathogen-contaminated soils [152–158]. These species have been shown to promote plant growth and induce systemic and local resistance in plants as well as to reduce DON production in different *Fusarium* species [159]. However, it is worth mentioning that numerous studies and field applications have also shown various *Trichoderma* spp. as producers of mycotoxins having undesirable effects to plants, animals, and humans [138,160,161].

Gliocladium, teleomorph *Clonostachys*, is another common soil-born saprophytic mycoparasite isolated and described in the years 1840–1939. In last decades, *C. rosea* has been reported as broadspectrum BCA with application against numerous plant pathogens, including *Fusarium* pathogens and the mycotoxin ZEN [162–164]. It has shown ability to degrade DON, as well as minimise the effect of FHB in wheat [164–166]. Recently, the *C. rosea* ACM941 strain was isolated from peas in Manitoba and its formulation ‘CLO-1’ tested against FHB in field trials. The studies confirmed that CLO-1 reduces FHB severity in wheat, is most effective on moderately resistant cultivars, and to a large extent is effective as a conventional fungicide. Hence, it is evident that biological control is a promising option that can be a part of an integrated management system for the treatment of FHB and *Fusarium* mycotoxins. However, there is a need to continue to investigate newly discovered BCAs that are *Fusarium*-host specific with potential to degrade different mycotoxins in the cereal crops.

Sphaerodes mycoparasitica has been described in 2009 as specific mycoparasite to phytopathogenic and mycotoxigenic *Fusarium* hosts [150]. It represents a next-generation BCA-effective in controlling FHB [167–169]. According to Naranjo-Ortiz and Gabaldón [170] it is among the best-studied mycoparasites within the Sordariomycetes [168,171]. It is an exemplary model system to study biotrophic mycoparasitism [172] and polyphagous lifestyle against multiple *Fusarium* species [66] and associated mycotoxins involved in FHB [66]. This BCA is particularly suitable for prenatal plant care as protecting flowers and germinating seeds from *Fusarium* infection and thus providing an early control of FHB [20]. It has been shown in numerous studies to downregulate expression of *Tri* (trichothecene) and *AUS* (aurofusarin) genes in fusaria and degrade mycotoxins such as DON, 3ADON, 15ADON and ZEN [167–171,173]. Evidence from ongoing studies has also shown that *S. mycoparasitica* alone bolsters defense in wheat and barley against FHB

and mycotoxins as well as in combination with low dose of synthetic chemical (Folicur®) pesticide in both greenhouse and field experiments [138]. These findings have received farmers' attention [174,175] particularly when *S. mycoparasitica* benefits resistant cereal varieties [173–177].

4.7. Potential of Biocontrol Agent (BCA)-Resistant Crop Genotype (RCG) Protooperation

Several studies have already demonstrated cereal hosts resistance factors as well the BCA eco-friendly control effectiveness in reducing FHB. Undoubtedly, great strides have been made using Next Generation Sequencing (NGS) technology in deciphering the whole genome of wheat, durum, and *Fusarium graminearum*. The transcriptome of *F. graminearum* during the infection of wheat [178] further revealed the fungal genes coding for metabolites and multiple virulence factors assisting the spread and progression of the FHB epidemics. However, these insights also highlighted the complexity of pathogenic *Fusarium*, suggesting that a more rigorous management system is needed to control FHB and its mycotoxins. Further underscoring the need for a more improved management strategy, is the emergence of new *Fusarium* pathogenic lines and a shift to aggressive mycotoxins chemotypes such as the acetylated DON derivatives 3 ADON over 15 ADON, and the challenge of global warming which makes FHB a greater threat to even the most resistant wheat and durum (*cvs*) presently on the market.

While the most promising cultivars provide a certain level of type I and type II resistance, there is no cultivar that is primed to effectively reduce or minimise the effects of harmful mycotoxins such as DON, NIV, FUM, AUS. It is at this juncture that protooperation between wheat and durum hosts and BCAs can possibly be a promising sustainable strategy in the management of FHB in cereal crops.

Numerous studies are available on *Fusarium*-host interaction, host improvement through cultivar optimisation, breeding for FHB resistance, fungicide effectiveness and other management strategies as uncovered by data from the Web of Science Database (Figure 3). However, at the bottom of this FHB management list sits the least explored management area BCA-*Fusarium*-crop host interaction. A quick advance search in the Web of Science Database [62] attests that a great chasm exists between studies that investigate other FHB management strategies and the tripartite interaction between BCA as crop host supporters or enhancers of resistance against FHB. Indeed, the tripartite *Sphaerodes* (BCA)-*Fusarium graminearum* (pathogenic fungal host)-wheat (plant host) experiments using Synchrotron-based Fourier-transform infrared spectroscopy (FTIR) showed that protooperative strategy is possible; it can be applied in agriculture fields as an early disease prevention and without genetic modifications in cereal crops [174]. Nonetheless, there is a huge gap of knowledge pertinent to the domain of protooperation such as (i) BCAs and breeding, (ii) BCAs and cultivars, (iii) BCAs and fungicides and (iv) BCAs and other management strategies as shown in (Figure 4). A search in the Web of Science database also revealed that there are 7% scientific papers on BCA + breeding and 22% on BCA + cultivars compared to 71% papers reported on BCA with fungicides and management over the 70-year period (1951–2021). Hence, there is a great potential in exploring the interaction of BCA and cultivars throughout breeding programs in small cereals towards more sustainable protooperative control method to reduce FHB in crops (Figure 4). This area of protooperation between BCAs and crop genotypes under current management strategies is vast and the potential for discovering new protective mechanisms using OMICS (genomics-transcriptomics-proteomics) strategies is promising. As the history of controlling FHB in wheat, durum and other crops have shown, a more effective resistance against FHB is mounted when management strategies are combined.

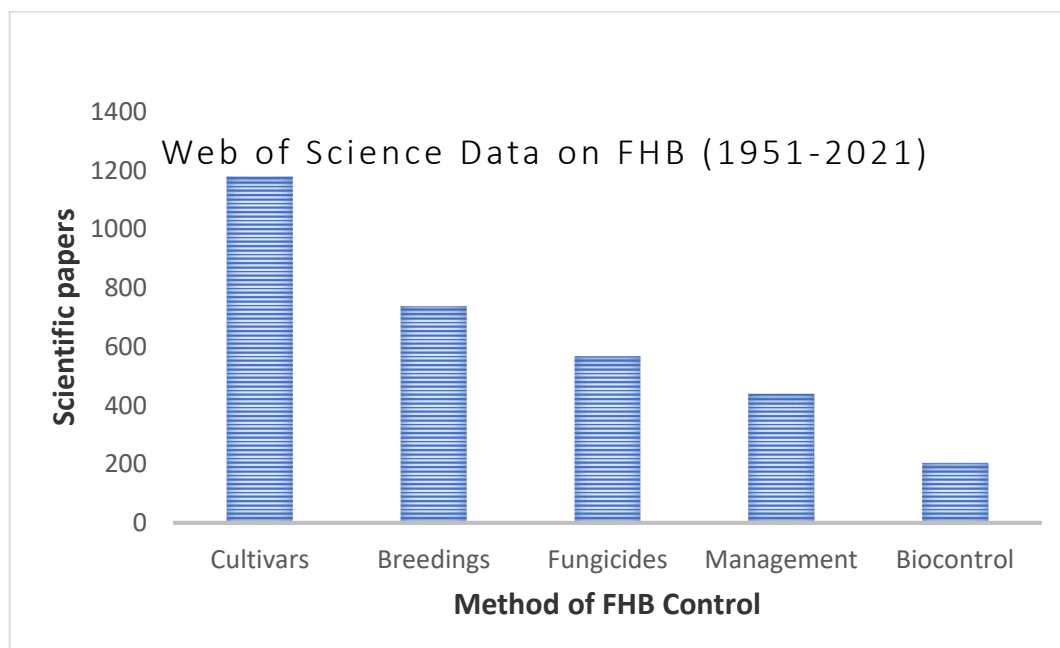


Figure 3. Scientific papers on different resistance methods (cultivars, breeding, fungicides, management and biocontrol) against FHB over the last 50 years as shown by data from Web of Science Database.

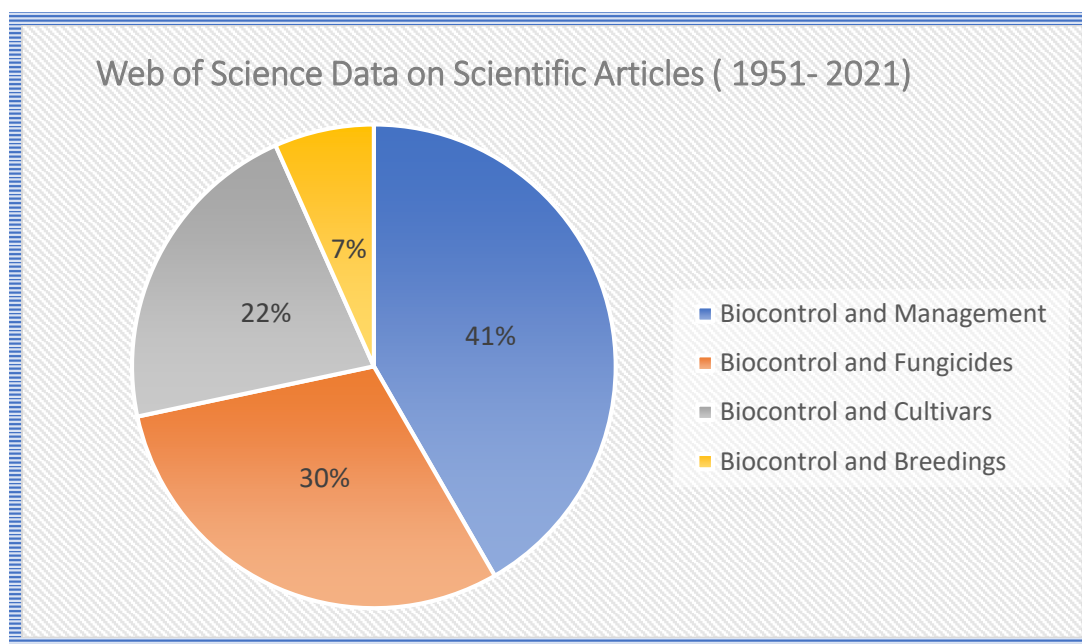


Figure 4. The diagram represents the data from the Web of Science for the years 1951–2021 that showed the proportion of scientific articles on protocoooperation between BCAs and other resistance methods including traditional management strategies, fungicides, cultivars, and breeding.

5. Conclusions and Future Work

In the last century, a plethora of information on *Fusarium* Head Blight (FHB) epidemics and wheat genetic determinants of the disease's progress have focused on crop resistance mechanisms. Although biocontrol studies lag behind genetics and FHB research in wheat, recent advances have invigorated the importance of beneficial biocontrol agents (BCAs) critical to determining the fate of disease. Therefore, it seems worthwhile making a more concerted and deliberate effort to explore the field of protocoooperation between BCAs and

resistant wheat genotypes under current management strategies and changing climate conditions. The joint breeding and biocontrol effort could produce and add another weapon to the resistance arsenal in the fight against FHB within a well-defined integrated management strategy. It can also help smart farming [179] to improve the accuracy in the prediction of FHB disease epidemics and associated mycotoxins. In particular, positive outcomes might be expected when applying BCA control measures with highly correlated resistant wheat, also, to other staple cereal crops. Special attention should be given to assembling a protective-protocooperative model system, computational analyses, imaging, phenotyping, mapping, as well as deep learning [180] in precision agriculture to protect farms globally.

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References

1. Voss-Fels, K.P.; Qian, L.; Gabur, I.; Obermeier, C.; Hickey, L.T.; Werner, C.R.; Kontowski, S.; Frisch, M.; Friedt, W.; Snowdon, R.J.; et al. Genetic insights into underground responses to *Fusarium graminearum* infection in wheat. *Sci. Rep.* **2018**, *8*, 13153. [\[CrossRef\]](#)
2. Agriopoulou, S.; Stamatelopoulou, E.; Varzakas, T. Advances in Occurrence, Importance, and Mycotoxin Control Strategies: Prevention and Detoxification in Foods. *Foods* **2020**, *9*, 137. [\[CrossRef\]](#) [\[PubMed\]](#)
3. O'Donnell, K.; Kistler, H.; Tacke, B.K.; Casper, H.H. Gene genealogies reveal global phylogeographic structure and reproductive isolation among lineages of *Fusarium graminearum*, the fungus causing wheat scab. *Proc. Natl. Acad. Sci. USA* **2000**, *97*, 7905–7910. [\[CrossRef\]](#) [\[PubMed\]](#)
4. O'Donnell, K.; Ward, T.J.; Abern, D.; Kistler, H.C.; Aoki, T.; Orwig, N.; Kimura, M.; Bjørnstad, Å.; Klemsdal, S.S. Multilocus genotyping and molecular phylogenetics resolve a novel head blight pathogen within the *Fusarium graminearum* species complex from Ethiopia. *Fungal Genet. Biol.* **2008**, *45*, 1514–1522. [\[CrossRef\]](#) [\[PubMed\]](#)
5. Summerell, B.A.; Laurence, M.H.; Liew, E.C.Y.; Leslie, J.F. Biogeography and phylogeography of *Fusarium*: A review. *Fungal Divers.* **2010**, *44*, 3–13. [\[CrossRef\]](#)
6. Ward, T.J.; Clear, R.M.; Rooney, A.; O'Donnell, K.; Gaba, D.; Patrick, S.; Starkey, D.E.; Gilbert, J.; Geiser, D.M.; Nowicki, T.W. An adaptive evolutionary shift in *Fusarium* head blight pathogen populations is driving the rapid spread of more toxigenic *Fusarium graminearum* in North America. *Fungal Genet. Biol.* **2008**, *45*, 473–484. [\[CrossRef\]](#)
7. Nešić, K.; Habschied, K.; Mastanjević, K. Possibilities for the Biological Control of Mycotoxins in Food and Feed. *Toxins* **2021**, *13*, 198. [\[CrossRef\]](#)
8. Shah, D.A.; De Wolf, E.D.; Paul, P.A.; Madden, L.V. Accuracy in the prediction of disease epidemics when ensembling simple but highly correlated models. *PLoS Comput. Biol.* **2021**, *17*, e1008831. [\[CrossRef\]](#)
9. Mielniczuk, E.; Skwaryło-Bednars, B. *Fusarium* Head Blight, Mycotoxins and Strategies for Their Reduction. *Agronomy* **2020**, *10*, 509. [\[CrossRef\]](#)
10. Hucl, P.; Briggs, C.; Graf, R.; Chibbar, R.N. Genetic Gains in Agronomic and Selected End-Use Quality Traits over a Century of Plant Breeding of Canada Western Red Spring Wheat. *Cereal Chem. J.* **2015**, *92*, 537–543. [\[CrossRef\]](#)
11. Steiner, B.; Michel, S.; Maccaferri, M.; Lemmens, M.; Tuberosa, R.; Buerstmayr, H. Exploring and exploiting the genetic variation of *Fusarium* head blight resistance for genomic-assisted breeding in the elite durum wheat gene pool. *Theor. Appl. Genet.* **2019**, *132*, 969–988. [\[CrossRef\]](#)

12. Sari, E.; Knox, R.E.; Ruan, Y.; Henriquez, M.A.; Kumar, S.; Burt, A.J.; Cuthbert, R.D.; Konkin, D.J.; Walkowiak, S.; Campbell, H.L.; et al. Historic recombination in a durum wheat breeding panel enables high-resolution mapping of Fusarium head blight resistance quantitative trait loci. *Sci. Rep.* **2020**, *10*, 7567. [\[CrossRef\]](#)
13. Bollina, V.; Kumaraswamy, G.K.; Kushalappa, A.C.; Choo, T.M.; Dion, Y.; Rioux, S.; Faubert, D.; Hamzehzarghani, H. Mass spectrometry-based metabolomics application to identify quantitative resistance-related metabolites in barley against Fusarium head blight. *Mol. Plant Pathol.* **2010**, *11*, 769–782. [\[CrossRef\]](#)
14. Cuperlovic-Culf, M.; Wang, L.; Forseille, L.; Boyle, K.; Merkley, N.; Burton, I.; Fobert, P.R. Metabolic Biomarker Panels of Response to Fusarium Head Blight Infection in Different Wheat Varieties. *PLoS ONE* **2016**, *11*, e20592. [\[CrossRef\]](#)
15. Wang, L.; Li, Q.; Liu, Z.; Surendra, A.; Pan, Y.; Li, Y.; Zaharia, L.I.; Ouellet, T.; Fobert, P.R. Integrated transcriptome and hormone profiling highlight the role of multiple phytohormone pathways in wheat resistance against fusarium head blight. *PLoS ONE* **2018**, *13*, e0207036. [\[CrossRef\]](#)
16. Maccaferri, M.; Harris, N.S.; Twardziok, S.O.; Pasam, R.K.; Gundlach, H.; Spannagl, M.; Ormanbekova, D.; Lux, T.; Prade, V.M.; Milner, S.G.; et al. Durum wheat genome highlights past domestication signatures and future improvement targets. *Nat. Genet.* **2019**, *51*, 885–895. [\[CrossRef\]](#)
17. Walkowiak, S.; Gao, L.; Monat, C.; Haberer, G.; Kassa, M.T.; Brinton, J.; Ramirez-Gonzalez, R.H.; Kolodziej, M.C.; Delorean, E.; Thambugala, D.; et al. Multiple wheat genomes reveal global variation in modern breeding. *Nat. Cell Biol.* **2020**, *588*, 277–283. [\[CrossRef\]](#)
18. Krattinger, S.; Keller, B. Molecular genetics and evolution of disease resistance in cereals. *New Phytol.* **2016**, *212*, 320–332. [\[CrossRef\]](#) [\[PubMed\]](#)
19. Chen, Y.; Wang, J.; Yang, N.; Wen, Z.; Sun, X.; Chai, Y.; Ma, Z. Wheat microbiome bacteria can reduce virulence of a plant pathogenic fungus by altering histone acetylation. *Nat. Commun.* **2018**, *9*, 3429. [\[CrossRef\]](#) [\[PubMed\]](#)
20. Kim, S.; Lahlali, R.; Karunakaran, C.; Vujanovic, V. Specific Mycoparasite-Fusarium Graminearum Molecular Signatures in Germinating Seeds Disabled Fusarium Head Blight Pathogen's Infection. *Int. J. Mol. Sci.* **2021**, *22*, 2461. [\[CrossRef\]](#) [\[PubMed\]](#)
21. Guo, X.; Shi, Q.; Yuan, J.; Wang, M.; Wang, J.; Wang, C.; Zhang, J.; Fu, S.; Su, H.; Liu, Y.; et al. Alien chromatin but not Fhb7 confers Fusarium head blight resistance in wheat breeding. *bioRxiv* **2021**. [\[CrossRef\]](#)
22. Rampersad, S.N. Pathogenomics and Management of Fusarium Diseases in Plants. *Pathogens* **2020**, *9*, 340. [\[CrossRef\]](#) [\[PubMed\]](#)
23. Velásquez, A.C.; Castroverde, C.D.; He, S.Y. Plant-Pathogen Warfare under Changing Climate Conditions. *Curr. Biol.* **2018**, *28*, R619–R634. [\[CrossRef\]](#) [\[PubMed\]](#)
24. Karlsson, I.; Persson, P.; Friberg, H. Fusarium Head Blight from a Microbiome Perspective. *Front. Microbiol.* **2021**, *12*, 628373. [\[CrossRef\]](#)
25. Smith, W.G. *Diseases of Field and Garden Crops: Chiefly Such as Caused by Fungi*; Macmillan: London, UK, 1884; pp. 208–213.
26. Bal, G. Scab of Wheat: Prospects For Control. *Plant Dis.* **1994**, *78*, 760–766. [\[CrossRef\]](#)
27. Champeil, A.; Doré, T.; Fourbet, J.F. Fusarium head blight: Epidemiological origin of the effects of cultural practices on head blight attacks and the production of mycotoxins by Fusarium in wheat grains. *Plant Sci.* **2004**, *166*, 1389–1415. [\[CrossRef\]](#)
28. McMullen, M.; Jones, R.; Gallenberg, D. Scab of Wheat and Barley: A Re-emerging Disease of Devastating Impact. *Plant Dis.* **1997**, *81*, 1340–1348. [\[CrossRef\]](#)
29. McMullen, M.; Bergstrom, G.; De Wolf, E.; Dill-Macky, R.; Hershman, D.; Shaner, G.; Van Sanford, D. A Unified Effort to Fight an Enemy of Wheat and Barley: Fusarium Head Blight. *Plant Dis.* **2012**, *96*, 1712–1728. [\[CrossRef\]](#)
30. Moschini, R.C.; Fortugno, C. Predicting wheat head blight incidence using models based on meteorological factors in Pergamino, Argentina. *Eur. J. Plant Pathol.* **1996**, *102*, 211–218. [\[CrossRef\]](#)
31. Snijders, C.H.A. Genetic variation for resistance to Fusarium head blight in bread wheat. *Euphytica* **1990**, *50*, 171–179. [\[CrossRef\]](#)
32. Stack, R.W. Return of an Old Problem: Fusarium Head Blight of Small Grains. *Plant Health Prog.* **2000**, *1*, 19. [\[CrossRef\]](#)
33. Stack, R.W. History of Fusarium Head Blight with emphasis on North America. In *Fusarium Head Blight of Wheat and Barley*; Leonard, K.J., Bushnell, W.R., Eds.; APS Press: St. Paul, MN, USA, 2003; pp. 1–34.
34. Tekauz, A. History of FHB research in (western) Canada. In Proceedings of the 9th Canadian Workshop on Fusarium Head Blight, Winnipeg, MB, Canada, 22 November 2018. Available online: <https://static1.squarespace.com/static/56be29e022482ec146a7c5b8/t/5bfed9768985834c7e47a1fb/1543428484162/Plenary+C+Tekauz.pdf> (accessed on 18 March 2021).
35. Clear, R.; Patrick, S.; Mycology, Grain Research Laboratory, Canadian Grain Commission. Fusarium Head Blight in Western Canada. 2018. Available online: <https://grainscanada.gc.ca/en/grain-research/scientific-reports/fhb-western/fhb-1.html> (accessed on 20 November 2020).
36. Khan, M.K.; Pandey, A.; Athar, T.; Athar, T.; Choudhary, S.; Deval, R.; Gezgin, S.; Hamurcu, M.; Topal, A.; Atmaca, E.; et al. Fusarium head blight in wheat: Contemporary status and molecular approaches. *Biotech* **2020**, *10*, 172. [\[CrossRef\]](#)
37. Fernando, W.D.; Oghenekaro, A.O.; Tucker, J.R.; Badea, A. Building on a foundation: Advances in epidemiology, resistance breeding, and forecasting research for reducing the impact of fusarium head blight in wheat and barley. *Can. J. Plant Pathol.* **2021**, *43*, 495–526. [\[CrossRef\]](#)
38. Starkey, D.E.; Ward, T.J.; Aoki, T.; Gale, L.R.; Kistler, H.; Geiser, D.M.; Suga, H.; Tóth, B.; Varga, J.; O'Donnell, K. Global molecular surveillance reveals novel Fusarium head blight species and trichothecene toxin diversity. *Fungal Genet. Biol.* **2007**, *44*, 1191–1204. [\[CrossRef\]](#) [\[PubMed\]](#)

39. Yli-Mattila, T.; Gagkaeva, T.; Ward, T.J.; Aoki, T.; Kistler, H.C.; O'Donnell, K. A novel Asian clade within the *Fusarium graminearum* species complex includes a newly discovered cereal head blight pathogen from the Russian Far East. *Mycologia* **2009**, *101*, 841–852. [CrossRef] [PubMed]
40. Sarver, B.A.; Ward, T.J.; Gale, L.R.; Broz, K.; Kistler, H.C.; Aoki, T.; Nicholson, P.; Carter, J.; O'Donnell, K. Novel *Fusarium* head blight pathogens from Nepal and Louisiana revealed by multilocus genealogical concordance. *Fungal Genet. Biol.* **2011**, *48*, 1096–1107. [CrossRef] [PubMed]
41. Ward, T.J.; Bielawski, J.; Kistler, H.; Sullivan, E.; O'Donnell, K. Ancestral polymorphism and adaptive evolution in the trichothecene mycotoxin gene cluster of phytopathogenic *Fusarium*. *Proc. Natl. Acad. Sci. USA* **2002**, *99*, 9278–9283. [CrossRef] [PubMed]
42. Parry, D.W.; Jenkinson, P.; McLeod, L. *Fusarium* ear blight (scab) in small grain cereals—A review. *Plant Pathol.* **1995**, *44*, 207–238. [CrossRef]
43. Suga, H.; Karugia, G.W.; Ward, T.; Gale, L.R.; Tomimura, K.; Nakajima, T.; Miyasaka, A.; Koizumi, S.; Kageyama, K.; Hyakumachi, M. Molecular Characterization of the *Fusarium graminearum* Species Complex in Japan. *Phytopathology* **2008**, *98*, 159–166. [CrossRef]
44. Qiu, J.-B.; Sun, J.-T.; Yu, M.-Z.; Xu, J.-H.; Shi, J.-R. Temporal dynamics, population characterization and mycotoxins accumulation of *Fusarium graminearum* in Eastern China. *Sci. Rep.* **2016**, *6*, 36350. [CrossRef]
45. Talas, F.; Longin, F.; Miedaner, T. Sources of resistance to *Fusarium* head blight within Syrian durum wheat landraces. *Plant Breed.* **2011**, *130*, 398–400. [CrossRef]
46. Wagacha, J.M.; Steiner, U.; Dehne, H.-W.; Zuehlke, S.; Spiteller, M.; Muthomi, J.; Oerke, E.-C. Diversity in myco-toxins and fungal species infecting wheat in Nakuru District, Kenya. *J. Phytopathol.* **2010**, *158*, 527–535. [CrossRef]
47. Bai, G.; Shaner, G. Management and resistance in wheat and barley to *fusarium* head blight. *Annu. Rev. Phytopathol.* **2004**, *42*, 135–161. [CrossRef]
48. Legrand, F.; Picot, A.; Díaz, J.F.C.; Chen, W.; Le Floch, G. Challenges facing the biological control strategies for the management of *Fusarium* Head Blight of cereals caused by *F. graminearum*. *Biol. Control* **2017**, *113*, 26–38. [CrossRef]
49. Figueroa, M.; Hammond-Kosack, K.E.; Solomon, P.S. A review of wheat diseases-A field perspective. *Mol. Plant Pathol.* **2018**, *19*, 1523–1536. [CrossRef]
50. Gilbert, J.; Tekauz, A. Strategies for management of *fusarium* head blight (FHB) in cereals. *Prairie Soils Crop J.* **2011**, *4*, 97–104. Available online: <https://prairiesoilsandcrops.ca/articles/volume-4-11-screen.pdf> (accessed on 22 March 2021).
51. Statistics Canada (STC) and Agriculture and AgriFood Canada. Canada: Historical Supply and Disposition, Crops 2017–2018 to 2018–2019. Available online: <https://aimis-simia.agr.gc.ca/rp/index-eng.cfm?action=pR&r=244&lang=EN> (accessed on 27 March 2021).
52. Windels, C.E. Economic and Social Impacts of *Fusarium* Head Blight: Changing Farms and Rural Communities in the Northern Great Plains. *Phytopathology* **2000**, *90*, 17–21. [CrossRef] [PubMed]
53. King, R.; Urban, M.; Hammond-Kosack, M.C.U.; Hassani-Pak, K.; Hammond-Kosack, K.E. The completed genome sequence of the pathogenic ascomycete fungus *Fusarium graminearum*. *BMC Genom.* **2015**, *16*, 544. [CrossRef] [PubMed]
54. Zhu, Z.; Hao, Y.; Mergoum, M.; Bai, G.; Humphreys, G.; Cloutier, S.; Xia, X.; He, Z. Breeding wheat for resistance to *Fusarium* head blight in the Global North: China, USA, and Canada. *Crop J.* **2019**, *7*, 730–738. [CrossRef]
55. Wegulo, S.N.; Baenziger, P.S.; Nopsa, J.F.H.; Bockus, W.W.; Hallen-Adams, H. Management of *Fusarium* head blight of wheat and barley. *Crop Prot.* **2015**, *73*, 100–107. [CrossRef]
56. Savary, S.; Willocquet, L.; Pethybridge, S.J.; Esker, P.; McRoberts, N.; Nelson, A. The global burden of pathogens and pests on major food crops. *Nat. Ecol. Evol.* **2019**, *3*, 430–439. [CrossRef]
57. Wang, H.; Sun, S.; Ge, W.; Zhao, L.; Hou, B.; Wang, K.; Lyu, Z.; Chen, L.; Xu, S.; Guo, J.; et al. Horizontal gene transfer of *Fhb7* from fungus underlies *Fusarium* head blight resistance in wheat. *Science* **2020**, *368*, eaba5435. [CrossRef]
58. Aboukhaddour, R.; Fetch, T.; McCallum, B.D.; Harding, M.W.; Beres, B.L.; Graf, R.J. Wheat diseases on the prairies: A Canadian story. *Plant Pathol.* **2020**, *69*, 418–432. [CrossRef]
59. Clear, R.; Patrick, S. *Fusarium* head blight pathogens isolated from *fusarium*-damaged kernels of wheat in western Canada, 1993 to 1998. *Can. J. Plant Pathol.* **2000**, *22*, 51–60. [CrossRef]
60. Tittlemier, S.A.; Roscoe, M.; Trelka, R.; Gaba, D.; Chan, J.M.; Patrick, S.K.; Sulyok, M.; Krska, R.; McKendry, T.; Gräfenhan, T. *Fusarium* Damage in Small Cereal Grains from Western Canada. 2. Occurrence of *Fusarium* Toxins and Their Source Organisms in Durum Wheat Harvested in 2010. *J. Agric. Food Chem.* **2013**, *61*, 5438–5448. [CrossRef]
61. Tittlemier, S.A.; Blagden, R.; Chan, J.; Gaba, D.; McKendry, T.; Pleskach, K.; Roscoe, M. *Fusarium* and *Alternaria* mycotoxins present in Canadian wheat and durum harvest samples. *Can. J. Plant Pathol.* **2019**, *41*, 403–414. [CrossRef]
62. Web of Science Database. Available online: <https://apps-whofofknowledge-com.cyber.usask.ca/> (accessed on 4 May 2021).
63. Miller, J.D. Epidemiology of *Fusarium* ear diseases. In *Mycotoxins in Grain: Compounds other than Aflatoxins*; Mil-ler, J.D., Trenholm, H.L., Eds.; Eagan Press: St. Paul, MN, USA, 1994; pp. 19–36.
64. Dean, R.; van Kan, J.; Pretorius, Z.A.; Hammond-Kosack, K.; Di Pietro, A.; Spanu, P.; Rudd, J.; Dickman, M.; Kahmann, R.; Ellis, J.; et al. The Top 10 fungal pathogens in molecular plant pathology. *Mol. Plant Pathol.* **2012**, *13*, 414–430. [CrossRef] [PubMed]
65. Dweba, C.; Figlan, S.; Shimelis, H.; Motaung, T.; Sydenham, S.; Mwadzingeni, L.; Tsilo, T. *Fusarium* head blight of wheat: Pathogenesis and control strategies. *Crop Prot.* **2017**, *91*, 114–122. [CrossRef]

66. Kim, S.H.; Vujanovic, V. Relationship between mycoparasites lifestyles and biocontrol behaviors against *Fusarium* spp. and mycotoxins production. *Appl. Microbiol. Biotechnol.* **2016**, *100*, 5257–5272. [CrossRef]
67. Desjardins, A.E. *Fusarium* mycotoxins: Chemistry, genetics, and biology. *Int. J. Food Microbiol.* **2006**, *119*, 47–50. [CrossRef]
68. Canadian Phytopathological Society. Canadian Plant Disease Survey. 1923. Available online: phytopath.ca/publication/cpds (accessed on 12 January 2021).
69. Canadian Grain Commission. *Fusarium Head Blight in Western Canada*. Available online: <https://grainscanada.gc.ca/en/grain-research/scientific-reports/fhb-western/fhb-2.html> (accessed on 12 January 2021).
70. Jin, F.; Bai, G.; Zhang, D.; Dong, Y.; Ma, L.; Bockus, W.; Dowell, F. *Fusarium*-Damaged Kernels and Deoxynivalenol in *Fusarium*-Infected U.S. Winter Wheat. *Phytopathology* **2014**, *104*, 472–478. [CrossRef]
71. Schaafsma, A.W.; Hooker, D. Climatic models to predict occurrence of *Fusarium* toxins in wheat and maize. *Int. J. Food Microbiol.* **2007**, *119*, 116–125. [CrossRef]
72. Amarasinghe, C.C.; Fernando, W.G.D. Comparative Analysis of Deoxynivalenol Biosynthesis Related Gene Expression among Different Chemotypes of *Fusarium graminearum* in Spring Wheat. *Front. Microbiol.* **2016**, *7*, 1229. [CrossRef]
73. Azam, M.S.; Yu, D.; Wu, A. Enzymes for degradation of *Fusarium* mycotoxins. In *Food Safety & Mycotoxins*; Wu, A., Ed.; Springer: Singapore, 2019; pp. 113–135. [CrossRef]
74. Gilbert, J.; Haber, S. Overview of some recent research developments in *Fusarium* head blight of wheat. *Can. J. Plant Pathol.* **2013**, *35*, 149–174. [CrossRef]
75. Dawson, A. *Fusarium* Concerns Reach New Heights. In *The Western Producer—SaskSeed Guide 2017*; Western Producer: Saskatoon, SK, Canada; pp. 22–24. Available online: https://static.producer.com/wp-content/uploads/2017/01/2017_SK_Seed_Guide.pdf (accessed on 16 April 2021).
76. Ji, C.; Fan, Y.; Zhao, L. Review on biological degradation of mycotoxins. *Anim. Nutr.* **2016**, *2*, 127–133. [CrossRef] [PubMed]
77. McNeil, J.N.; Cotnoir, P.-A.; Leroux, T.; Laprade, R.; Schwartz, J.-L. A Canadian national survey on the public perception of biological control. *BioControl* **2010**, *55*, 445–454. [CrossRef]
78. Paulitz, T.C.; Bélanger, R.R. Biological control in greenhouses systems. *Annu. Rev. Phytopathol.* **2001**, *39*, 103–133. [CrossRef] [PubMed]
79. Gilbert, J.; Fernando, W.G.D. Epidemiology and biological control of *Gibberella zeae*/*Fusarium graminearum*. *Can. J. Plant Pathol.* **2014**, *26*, 464–472. [CrossRef]
80. Miller, J.D.; Young, J.C.; Sampson, D.R. Deoxynivalenol and *Fusarium* Head Blight Resistance in Spring Cereals. *J. Phytopathol.* **1985**, *113*, 359–367. [CrossRef]
81. Shaner, G.E. Epidemiology of *Fusarium* head blight of small grain cereals in North America. In *Fusarium Head Blight of Wheat and Barley*; Leonard, K.J., Bushnell, W.R., Eds.; APS Press: St. Paul, MN, USA, 2003; pp. 84–119.
82. Wollenberg, R.D.; Taft, M.H.; Giese, S.; Thiel, C.; Balázs, Z.; Giese, H.; Manstein, D.J.; Sondergaard, T.E. Phenamacril is a reversible and noncompetitive inhibitor of *Fusarium* class I myosin. *J. Biol. Chem.* **2019**, *294*, 1328–1337. [CrossRef]
83. Hou, Y.-P.; Qu, X.-P.; Mao, X.-W.; Kuang, J.; Duan, Y.-B.; Song, X.-S.; Wang, J.-X.; Chen, C.-J.; Zhou, M.-G. Resistance mechanism of *Fusarium fujikuroi* to phenamacril in the field. *Pest Manag. Sci.* **2018**, *74*, 607–616. [CrossRef]
84. Hou, Y.-P.; Mao, X.-W.; Wang, J.-X.; Zhan, S.-W.; Zhou, M.-G. Sensitivity of *Fusarium asiaticum* to a novel succinate dehydrogenase inhibitor fungicide pydiflumetofen. *Crop Prot.* **2017**, *96*, 237–244. [CrossRef]
85. Nagelkirk, M.; Chilvers, M. Managing *Fusarium* Head Blight in Winter Wheat. MSU—Michigan Wheat Program and the U.S. Wheat and Barley Head Scab Initiative. 2020. Available online: https://www.canr.msu.edu/wheat/disease/FHB%20fact%20sheet%202020%20revised%20MN_MC_Final%202.pdf. (accessed on 18 March 2021).
86. Zhang, Y.-J.; Yu, J.-J.; Zhang, X.; Cheng, C.-J.; Wang, J.-X.; Hollomon, D.W.; Fan, P.-S.; Zhou, M.-G. Effect of Carbendazim Resistance on Trichothecene Production and Aggressiveness of *Fusarium graminearum*. *Mol. Plant Microbe Interact.* **2009**, *22*, 1143–1150. [CrossRef]
87. Liu, Y.; Chen, X.; Jiang, J.; Hamada, M.S.; Yin, Y.; Ma, Z. Detection and dynamics of different carbendazim-resistance conferring β -tubulin variants of *Gibberella zeae* collected from infected wheat heads and rice stubble in China. *Pest Manag. Sci.* **2014**, *70*, 1228–1236. [CrossRef]
88. Spolti, P.; Del Ponte, E.M.; Dong, Y.; Cummings, J.; Bergstrom, G.C. Triazole Sensitivity in a Contemporary Population of *Fusarium graminearum* from New York Wheat and Competitiveness of a Tebuconazole-Resistant Isolate. *Plant Dis.* **2014**, *98*, 607–613. [CrossRef]
89. Paul, P.A.; Lipps, P.E.; Hershman, D.E.; McMullen, M.P.; Draper, M.A.; Madden, L. Efficacy of Triazole-Based Fungicides for *Fusarium* Head Blight and Deoxynivalenol Control in Wheat: A Multivariate Meta-Analysis. *Phytopathology* **2008**, *98*, 999–1011. [CrossRef] [PubMed]
90. Vila-Donat, P.; Marín, S.; Sanchis, V.; Ramos, A.J. A review of the mycotoxin adsorbing agents, with an emphasis on their multi-binding capacity, for animal feed decontamination. *Food Chem. Toxicol.* **2018**, *114*, 246–259. [CrossRef] [PubMed]
91. Zain, M.E. Impact of mycotoxins on humans and animals. *J. Saudi Chem. Soc.* **2011**, *15*, 129–144. [CrossRef]
92. Stoev, S.D. Food Safety and Increasing Hazard of Mycotoxin Occurrence in Foods and Feeds. *Crit. Rev. Food Sci. Nutr.* **2013**, *53*, 887–901. [CrossRef]

93. Anderson, J.A.; Stack, R.W.; Liu, S.; Waldron, B.L.; Fjeld, A.D.; Coyne, C.; Moreno-Sevilla, B.; Fetch, J.M.; Song, Q.J.; Cregan, P.B.; et al. DNA markers for Fusarium head blight resistance QTLs in two wheat populations. *Theor. Appl. Genet.* **2001**, *102*, 1164–1168. [\[CrossRef\]](#)
94. Bai, G.; Su, Z.; Cai, J. Wheat resistance to Fusarium head blight. *Can. J. Plant Pathol.* **2018**, *40*, 336–346. [\[CrossRef\]](#)
95. Buerstmayr, H.; Ban, T.; A Anderson, J. QTL mapping and marker-assisted selection for Fusarium head blight resistance in wheat: A review. *Plant Breed.* **2009**, *128*, 1–26. [\[CrossRef\]](#)
96. Christensen, J.J.; Stakman, E.C.; Immer, F.R. Susceptibility of wheat varieties and hybrids to Fusarial Head Blight in Minnesota. *Minn. Agric. Exp. Stn Bull.* **1929**, *59*, 1–29.
97. Gilbert, J.; Tekauz, A. Review: Recent developments in research on fusarium head blight of wheat in Canada. *Can. J. Plant Pathol.* **2000**, *22*, 1–8. [\[CrossRef\]](#)
98. Wulff, B.B.H.; Jones, J.D.G. Breeding a fungal gene into wheat. *Science* **2020**, *368*, 822–823. [\[CrossRef\]](#)
99. Atanasoff, D. Fusarium blight (scab) of wheat and other cereals. *J. Agric. Res.* **1920**, *20*, 1–32.
100. Haile, J.K.; N'Diaye, A.; Walkowiak, S.; Nilsen, K.T.; Clarke, J.M.; Kutcher, H.R.; Steiner, B.; Buerstmayr, H.; Pozniak, C.J. Fusarium Head Blight in Durum Wheat: Recent Status, Breeding Directions, and Future Research Prospects. *Phytopathology* **2019**, *109*, 1664–1675. [\[CrossRef\]](#) [\[PubMed\]](#)
101. Ma, Z.; Xie, Q.; Li, G.; Jia, H.; Zhou, J.; Kong, Z.; Li, N.; Yuan, Y. Germplasms, genetics and genomics for better control of disastrous wheat Fusarium head blight. *Theor. Appl. Genet.* **2020**, *133*, 1541–1568. [\[CrossRef\]](#)
102. Mesterhazy, A. Types and components of resistance to Fusarium head blight of wheat. *Plant Breed.* **1995**, *114*, 377–386. [\[CrossRef\]](#)
103. Pirgozliev, S.R.; Edwards, S.; Hare, M.C.; Jenkinson, P. Strategies for the Control of Fusarium Head Blight in Cereals. *Eur. J. Plant Pathol.* **2003**, *109*, 731–742. [\[CrossRef\]](#)
104. Bai, G.; Kolb, F.L.; Shaner, G.; Domier, L.L. Amplified Fragment Length Polymorphism Markers Linked to a Major Quantitative Trait Locus Controlling Scab Resistance in Wheat. *Phytopathology* **1999**, *89*, 343–348. [\[CrossRef\]](#)
105. Cuthbert, P.A.; Somers, D.J.; Thomas, J.; Cloutier, S.; Brulé-Babel, A. Fine mapping Fhb1, a major gene controlling fusarium head blight resistance in bread wheat (*Triticum aestivum* L.). *Theor. Appl. Genet.* **2006**, *112*, 1465–1472. [\[CrossRef\]](#) [\[PubMed\]](#)
106. Schroeder, H.W.; Christensen, J.J. Factors affecting resistance of wheat to scab caused by *Gibberella zeae*. *Phytopathology* **1963**, *53*, 831–838.
107. Anderson, J.A. Marker-assisted selection for Fusarium head blight resistance in wheat. *Int. J. Food Microbiol.* **2007**, *119*, 51–53. [\[CrossRef\]](#) [\[PubMed\]](#)
108. Collard, B.; Mackill, D.J. Marker-assisted selection: An approach for precision plant breeding in the twenty-first century. *Philos. Trans. R. Soc. B Biol. Sci.* **2007**, *363*, 557–572. [\[CrossRef\]](#) [\[PubMed\]](#)
109. Krishnappa, G.; Savadi, S.; Tyagi, B.S.; Singh, S.K.; Mamrutha, H.M.; Kumar, S.; Mishra, C.N.; Khan, H.; Gangadhara, K.; Uday, G.; et al. Integrated genomic selection for rapid improvement of crops. *Genomics* **2021**, *113*, 1070–1086. [\[CrossRef\]](#)
110. Rai, N.; Bellundagi, A.; Kumar, P.K.C.; Thimmappa, R.K.; Rani, S.; Sinha, N.; Krishna, H.; Jain, N.; Singh, G.P.; Singh, P.K.; et al. Marker-assisted backcross breeding for improvement of drought tolerance in bread wheat (*Triticum aestivum* L. em Thell). *Plant Breed.* **2018**, *137*, 514–526. [\[CrossRef\]](#)
111. Yang, Z.; Gilbert, J.; Somers, D.; Fedak, G.; Procnier, J.; McKenzie, I. Marker Assisted Selection of Fusarium Head Blight Resistance Genes in Two Doubled Haploid Populations of Wheat. *Mol. Breed.* **2003**, *12*, 309–317. [\[CrossRef\]](#)
112. Anderson, J.A.; Chao, S.; Liu, S. Molecular Breeding Using a Major QTL for Fusarium Head Blight Resistance in Wheat. *Crop Sci.* **2007**, *47*, S-112–S-119. [\[CrossRef\]](#)
113. Pumphrey, M.; Bernardo, R.; Anderson, J.A. Validating the Fhb1 QTL for Fusarium Head Blight Resistance in Near-Isogenic Wheat Lines Developed from Breeding Populations. *Crop Sci.* **2007**, *47*, 200–206. [\[CrossRef\]](#)
114. Anderson, J.A.; Wiersma, J.J.; Linkert, G.L.; Kolmer, J.A.; Jin, Y.; Dill-Macky, R.; Wiersma, J.V.; Hareland, G.A. Registration of ‘Sabin’ Wheat. *J. Plant Regist.* **2012**, *6*, 174–179. [\[CrossRef\]](#)
115. Löffler, M.; Schön, C.-C.; Miedaner, T. Revealing the genetic architecture of FHB resistance in hexaploid wheat (*Triticum aestivum* L.) by QTL meta-analysis. *Mol. Breed.* **2009**, *23*, 473–488. [\[CrossRef\]](#)
116. Liu, S.; Zhang, X.; Pumphrey, M.; Stack, R.W.; Gill, B.S.; Anderson, J.A. Complex microcolinearity among wheat, rice, and barley revealed by fine mapping of the genomic region harboring a major QTL for resistance to Fusarium head blight in wheat. *Funct. Integr. Genom.* **2005**, *6*, 83–89. [\[CrossRef\]](#)
117. Cuthbert, P.A.; Somers, D.J.; Brulé-Babel, A. Mapping of Fhb2 on chromosome 6BS: A gene controlling Fusarium head blight field resistance in bread wheat (*Triticum aestivum* L.). *Theor. Appl. Genet.* **2007**, *114*, 429–437. [\[CrossRef\]](#)
118. Xue, S.; Li, G.; Jia, H.; Xu, F.; Lin, F.; Tang, M.; Wang, Y.; An, X.; Xu, H.; Zhang, L.; et al. Fine mapping Fhb4, a major QTL conditioning resistance to Fusarium infection in bread wheat (*Triticum aestivum* L.). *Theor. Appl. Genet.* **2010**, *121*, 147–156. [\[CrossRef\]](#)
119. Jia, H.; Zhou, J.; Xue, S.; Li, G.; Yan, H.; Ran, C.; Zhang, Y.; Shi, J.; Jia, L.; Wang, X.; et al. A journey to understand wheat Fusarium head blight resistance in the Chinese wheat landrace Wangshuibai. *Crop J.* **2018**, *6*, 48–59. [\[CrossRef\]](#)
120. Xue, S.; Xu, F.; Tang, M.; Zhou, Y.; Li, G.; An, X.; Lin, F.; Xu, H.; Jia, H.; Zhang, L.; et al. Precise mapping Fhb5, a major QTL conditioning resistance to Fusarium infection in bread wheat (*Triticum aestivum* L.). *Theor. Appl. Genet.* **2011**, *123*, 1055–1063. [\[CrossRef\]](#) [\[PubMed\]](#)

121. Dong, H.; Wang, R.; Yuan, Y.; Anderson, J.; Pumphrey, M.; Zhang, Z.; Chen, J. Evaluation of the Potential for Genomic Selection to Improve Spring Wheat Resistance to Fusarium Head Blight in the Pacific Northwest. *Front. Plant Sci.* **2018**, *9*, 911. [CrossRef] [PubMed]
122. Arruda, M.P.; Brown, P.J.; Lipka, A.E.; Krill, A.M.; Thurber, C.; Kolb, F.L. Genomic Selection for Predicting Fusarium Head Blight Resistance in a Wheat Breeding Program. *Plant Genome* **2015**, *8*, 1–12. [CrossRef] [PubMed]
123. Arruda, M.P.; Lipka, A.E.; Brown, P.J.; Krill, A.M.; Thurber, C.; Brown-Guedira, G.; Dong, Y.; Foresman, B.J.; Kolb, F.L. Comparing genomic selection and marker-assisted selection for Fusarium head blight resistance in wheat (*Triticum aestivum* L.). *Mol. Breed.* **2016**, *36*, 84. [CrossRef]
124. Gill, B.S.; Appels, R.; Botha, A.-M.; Buell, C.R.; Bennetzen, J.L.; Chalhoub, B.; Chumley, F.; Dvořák, J.; Iwanaga, M.; Keller, B.; et al. A Workshop Report on Wheat Genome Sequencing: International Genome Research on Wheat Consortium. *Genetics* **2004**, *168*, 1087–1096. [CrossRef] [PubMed]
125. Prat, N.; Guilbert, C.; Prah, U.; Wachter, E.; Steiner, B.; Langin, T.; Robert, O.; Buerstmayr, H. QTL mapping of Fusarium head blight resistance in three related durum wheat populations. *Theor. Appl. Genet.* **2017**, *130*, 13–27. [CrossRef]
126. Szabo-Hever, A.; Zhang, Q.; Friesen, T.L.; Zhong, S.; Elias, E.M.; Cai, X.; Jin, Y.; Faris, J.D.; Chao, S.; Xu, S.S. Genetic Diversity and Resistance to Fusarium Head Blight in Synthetic Hexaploid Wheat Derived From *Aegilops tauschii* and Diverse *Triticum turgidum* Subspecies. *Front. Plant Sci.* **2018**, *9*, 1829. [CrossRef] [PubMed]
127. Miedaner, T.; Wilde, F.; Steiner, B.; Buerstmayr, H.; Korzun, V.; Ebmeyer, E. Stacking quantitative trait loci (QTL) for Fusarium head blight resistance from non-adapted sources in an European elite spring wheat background and assessing their effects on deoxynivalenol (DON) content and disease severity. *Theor. Appl. Genet.* **2005**, *112*, 562–569. [CrossRef]
128. Zhao, M.; Leng, Y.; Chao, S.; Xu, S.S.; Zhong, S. Molecular mapping of QTL for Fusarium head blight resistance introgressed into durum wheat. *Theor. Appl. Genet.* **2018**, *131*, 1939–1951. [CrossRef] [PubMed]
129. Buerstmayr, M.; Matiasch, L.; Mascher, F.; Vida, G.; Ittu, M.; Robert, O.; Holdgate, S.; Flath, K.; Neumayer, A.; Buerstmayr, H. Mapping of quantitative adult plant field resistance to leaf rust and stripe rust in two European winter wheat populations reveals co-location of three QTL conferring resistance to both rust pathogens. *Theor. Appl. Genet.* **2014**, *127*, 2011–2028. [CrossRef]
130. Giancaspro, A.; Giove, S.L.; Zito, D.; Blanco, A.; Gadaleta, A. Mapping QTLs for Fusarium Head Blight Resistance in an Interspecific Wheat Population. *Front. Plant Sci.* **2016**, *7*, 1381. [CrossRef]
131. Buerstmayr, H.; Lemmens, M.; Hartl, L.; Doldi, L.; Steiner, B.; Stierschneider, M.; Ruckebauer, P. Molecular mapping of QTLs for Fusarium head blight resistance in spring wheat. I. Resistance to fungal spread (Type II resistance). *Theor. Appl. Genet.* **2002**, *104*, 84–91. [CrossRef]
132. Mesterhazy, A. Breeding wheat for Fusarium head blight resistance in Europe. In *Fusarium Head Blight of Wheat and Barley*; Leonard, K.J., Bushnell, W.R., Eds.; APS Press: St. Paul, MN, USA, 2003; pp. 211–240.
133. Poppenberger, B.; Berthiller, F.; Lucyshyn, D.; Sieberer, T.; Schuhmacher, R.; Krska, R.; Kuchler, K.; Glössl, J.; Luschign, C.; Adam, G. Detoxification of the Fusarium Mycotoxin Deoxynivalenol by a UDP-glucosyltransferase from *Arabidopsis thaliana*. *J. Biol. Chem.* **2003**, *278*, 47905–47914. [CrossRef]
134. Shude, S.P.; Yobo, K.S.; Mbili, N.C. Progress in the management of Fusarium head blight of wheat: An overview. *S. Afr. J. Sci.* **2020**, *116*, 1–7. [CrossRef]
135. Wei, Z.; Jousset, A. Plant breeding goes microbial. *Trends Plant Sci.* **2020**, *22*, 555–558. [CrossRef]
136. Gnanamanickam, S.S.; Vasudevan, P.; Reddy, M.S.; Defago, G.; Kloepper, J. Principles of biological control. In *Biological Control of Crop Diseases*; Springer: New York, NY, USA, 2002; pp. 1–9.
137. Pal, K.K.; Gardener, B.M. Biological Control of Plant Pathogens. *Plant Health Instr.* **2006**. [CrossRef]
138. Vujanovic, V. Fusarium and Fusarium Mycotoxin Biocontrol. PCT: WO2011/022809A1. 2011. Available online: <https://patentimages.storage.googleapis.com/b5/54/bf/2c20f0e0ba1f48/WO2011022809A1.pdf> (accessed on 3 May 2021).
139. Roberti, R.; Veronesi, A.; Cesari, A.; Cascone, A.; Di Berardino, I.; Bertini, L.; Caruso, C. Induction of PR proteins and resistance by the biocontrol agent *Clonostachys rosea* in wheat plants infected with *Fusarium culmorum*. *Plant Sci.* **2008**, *175*, 339–347. [CrossRef]
140. Hermosa, R.; Viterbo, A.; Chet, I.; Monte, E. Plant-beneficial effects of *Trichoderma* and of its genes. *Microbiology* **2012**, *158*, 17–25. [CrossRef] [PubMed]
141. Mukherjee, P.K.; Horwitz, B.A.; Herrera-Estrella, A.; Schmoll, M.; Kenerley, C.M. *Trichoderma* Research in the Genome Era. *Annu. Rev. Phytopathol.* **2013**, *51*, 105–129. [CrossRef] [PubMed]
142. Kim, S.H.; Vujanovic, V. Biodegradation and biodegradation of Fusarium mycotoxins by *Sphaerodes mycoparasitica*. *AMB Express* **2017**, *7*, 1–9. [CrossRef] [PubMed]
143. Vujanovic, V. Tremellomycetes Yeasts in Kernel Ecological Niche: Early Indicators of Enhanced Competitiveness of Endophytic and Mycoparasitic Symbionts against Wheat Pathobiota. *Plants* **2021**, *10*, 905. [CrossRef] [PubMed]
144. Simpson, D.R.; Weston, G.E.; Turner, J.A.; Jennings, P.; Nicholson, P. Differential Control of Head Blight Pathogens of Wheat by Fungicides and Consequences for Mycotoxin Contamination of Grain. *Eur. J. Plant Pathol.* **2001**, *107*, 421–431. [CrossRef]
145. He, J.W.; Bondy, G.S.; Zhou, T.; Caldwell, D.; Boland, G.J.; Scott, P.M. Toxicology of 3-epi-deoxynivalenol, a deoxynivalenol-transformation product by *Devosia mutans* 17-2-E-8. *Food Chem. Toxicol.* **2015**, *84*, 250–259. [CrossRef]

146. Ikunaga, Y.; Sato, I.; Grond, S.; Numaziri, N.; Yoshida, S.; Yamaya, H.; Hiradate, S.; Hasegawa, M.; Toshima, H.; Koitabashi, M.; et al. *Nocardioide* sp. strain WSN05-2, isolated from a wheat field, degrades deoxynivalenol, producing the novel intermediate 3-epi-deoxynivalenol. *Appl. Microbiol. Biotechnol.* **2011**, *89*, 419–427. [\[CrossRef\]](#)
147. Kollarczik, B.; Gareis, M.; Hanelt, M. In vitro transformation of the *Fusarium* mycotoxins deoxynivalenol and zearalenone by the normal gut microflora of pigs. *Nat. Toxins* **1994**, *2*, 105–110. [\[CrossRef\]](#)
148. Tan, H.; Hu, Y.; He, J.; Wu, L.; Liao, F.; Luo, B.; He, Y.; Zuo, Z.; Ren, Z.; Zhong, Z.; et al. Zearalenone degradation by two *Pseudomonas* strains from soil. *Mycotoxin Res.* **2014**, *30*, 191–196. [\[CrossRef\]](#)
149. Yu, Y.; Qiu, L.; Wu, H.; Tang, Y.; Yu, Y.; Li, X.; Liu, D. Degradation of zearalenone by the extracellular extracts of *Acinetobacter* sp. SM04 liquid cultures. *Biodegradation* **2011**, *22*, 613–622. [\[CrossRef\]](#)
150. Vujanovic, V.; Goh, Y.K. *Sphaerodes mycoparasitica* sp. nov., a new biotrophic mycoparasite on *Fusarium avenaceum*, *F. graminearum* and *F. oxysporum*. *Mycol. Res.* **2009**, *113*, 1172–1180. [\[CrossRef\]](#) [\[PubMed\]](#)
151. Busko, M.; Chełkowski, J.; Popiel, D.; Perkowski, J. Solid substrate bioassay to evaluate impact of *Trichoderma* on trichothecene mycotoxin production by *Fusarium* species. *J. Sci. Food Agric.* **2008**, *88*, 536–541. [\[CrossRef\]](#)
152. Sood, M.; Kapoor, D.; Kumar, V.; Sheteiwy, M.S.; Ramakrishnan, M.; Landi, M.; Araniti, F.; Sharma, A. *Trichoderma*: The “Secrets” of a Multitalented Biocontrol Agent. *Plants* **2020**, *9*, 762. [\[CrossRef\]](#)
153. Popiel, D.; Kwaśny, H.; Chełkowski, J.; Stępień, Ł.; Laskowska, M. Impact of selected antagonistic fungi on *Fusarium* species—Toxicogenic cereal pathogens. *Acta Mycol.* **2013**, *43*, 29–40. [\[CrossRef\]](#)
154. Matarese, F.; Sarrocco, S.; Gruber, S.; Seidl-Seiboth, V.; Vannacci, G. Biocontrol of *Fusarium* head blight: Interactions between *Trichoderma* and mycotoxigenic *Fusarium*. *Microbiology* **2012**, *158*, 98–106. [\[CrossRef\]](#)
155. Ferrigo, D.; Raiola, A.; Picollo, E.; Scopel, C.; Causin, R. *Trichoderma harzianum* T22 induces in maize systemic resistance against *Fusarium Verticillioide*s. *J. Plant Pathol.* **2014**, *96*, 133–142. [\[CrossRef\]](#)
156. Harman, G.E. Myths and Dogmas of Biocontrol Changes in Perceptions Derived from Research on *Trichoderma harzianum* T-22. *Plant Dis.* **2000**, *84*, 377–393. [\[CrossRef\]](#) [\[PubMed\]](#)
157. Harman, G.E.; Petzoldt, R.; Comis, A.; Chen, J. Interactions Between *Trichoderma harzianum* Strain T22 and Maize Inbred Line Mo17 and Effects of These Interactions on Diseases Caused by *Pythium ultimum* and *Colletotrichum graminicola*. *Phytopathology* **2004**, *94*, 147–153. [\[CrossRef\]](#)
158. Bigirimana, J.; Meyer, G.D.; Poppe, J.; Elad, Y.; Höfte, M. Induction of systemic resistance on bean (*Phaseolus vulgaris*) by *Trichoderma harzianum*. *Meded. Fac. Landbouwk. Toegep. Biol. Wet. Univ. Gent.* **1997**, *62*, 1001–1007.
159. Cardoza, R.E.; Malmierca, M.G.; Hermosa, M.R.; Alexander, N.J.; McCormick, S.P.; Proctor, R.H.; Tijerino, A.M.; Rumbero, A.; Monte, E.; Gutiérrez, S. Identification of Loci and Functional Characterization of Trichothecene Biosynthesis Genes in Filamentous Fungi of the Genus *Trichoderma*. *Appl. Environ. Microbiol.* **2011**, *77*, 4867–4877. [\[CrossRef\]](#) [\[PubMed\]](#)
160. Degenkolb, T.; Dieckmann, R.; Nielsen, K.F.; Gräfenhan, T.; Theis, C.; Zafari, D.; Chaverri, P.; Ismael, A.; Brückner, H.; Von Döhren, H.; et al. The *Trichoderma brevicompactum* clade: A separate lineage with new species, new peptaibiotics, and mycotoxins. *Mycol. Prog.* **2008**, *7*, 177–219. [\[CrossRef\]](#)
161. Malmierca, M.G.; Cardoza, R.E.; Alexander, N.J.; McCormick, S.P.; Hermosa, R.; Monte, E.; Gutiérrez, S. Involvement of *Trichoderma* Trichothecenes in the Biocontrol Activity and Induction of Plant Defense-Related Genes. *Appl. Environ. Microbiol.* **2012**, *78*, 4856–4868. [\[CrossRef\]](#)
162. Takahashi-Ando, N.; Ohsato, S.; Shibata, T.; Hamamoto, H.; Yamaguchi, I.; Kimura, M. Metabolism of Zearalenone by Genetically Modified Organisms Expressing the Detoxification Gene from *Clonostachys rosea*. *Appl. Environ. Microbiol.* **2004**, *70*, 3239–3245. [\[CrossRef\]](#) [\[PubMed\]](#)
163. Utermark, J.; Karlovsky, P. Role of Zearalenone Lactonase in Protection of *Gliocladium roseum* from Fungitoxic Effects of the Mycotoxin Zearalenone. *Appl. Environ. Microbiol.* **2007**, *73*, 637–642. [\[CrossRef\]](#)
164. Xue, A.G. Biological Control of Pathogens Causing Root Rot Complex in Field Pea Using *Clonostachys rosea* Strain ACM941. *Phytopathology* **2003**, *93*, 329–335. [\[CrossRef\]](#)
165. Hue, A.G.; Voldeng, H.; Savard, M.; Fedak, G.; Tian, X.; Hsiang, T. Biological control of fusarium head blight of wheat with *Clonostachys rosea* strain ACM941. *Can. J. Plant Pathol.* **2009**, *31*, 169–179. [\[CrossRef\]](#)
166. Xue, A.G.; Chen, Y.; Voldeng, H.D.; Fedak, G.; Savard, M.E.; Längle, T.; Zhang, J.; Harman, G.E. Concentration and cultivar effects on efficacy of CLO-1 biofungicide in controlling *Fusarium* head blight of wheat. *Biol. Control* **2014**, *73*, 2–7. [\[CrossRef\]](#)
167. Vujanovic, V.; Goh, Y.K. *Sphaerodes mycoparasitica* biotrophic mycoparasite of 3-acetyldioxynivalenol and 15-acetyldeoxynivalenol-producing toxigenic *Fusarium graminearum* chemotypes. *FEMS Microbiol. Lett.* **2011**, *316*, 136–143. [\[CrossRef\]](#)
168. Vujanovic, V.; Goh, Y.K. qPCR quantification of *Sphaerodes mycoparasitica* biotrophic mycoparasite interaction with *Fusarium graminearum*: In vitro and in planta assays. *Arch. Microbiol.* **2012**, *194*, 707–717. [\[CrossRef\]](#) [\[PubMed\]](#)
169. Vujanovic, V.; Kim, S.H. Adaptability of mitospore stage in *Sphaerodes mycoparasitica* towards its mycoparasitic-polyphagous lifestyle. *Mycologia* **2017**, *109*, 1–9. [\[CrossRef\]](#) [\[PubMed\]](#)
170. Naranjo-Ortiz, M.A.; Gabaldón, T. Fungal evolution: Major ecological adaptations and evolutionary transitions. *Biol. Rev.* **2019**, *94*, 1443–1476. [\[CrossRef\]](#) [\[PubMed\]](#)
171. Goh, Y.K.; Vujanovic, V. *Sphaerodes quadrangularis* biotrophic mycoparasitism on *Fusarium avenaceum*. *Mycologia* **2010**, *102*, 757–762. [\[CrossRef\]](#)

172. Chenthamara, K.; Druzhinina, I.S. 12 Ecological Genomics of Mycotrophic Fungi. In *Environmental and Microbial Relationships. The Mycota A Comprehensive treatise on Fungi as Experimental Systems for Basic and Applied Research*, 3rd ed.; Esser, K., Druzhinina, I.S., Kubicek, C.P., Eds.; Springer: Cham, Switzerland, 2016; Volume IV, pp. 215–246. Available online: https://link.springer.com/chapter/10.1007/978-3-319-29532-9_12 (accessed on 17 April 2021).
173. Vujanovic, V.; Daida, M.A.; Daida, P. qPCR assessment of aurofusarin gene expression in mycotoxigenic *Fusarium* species challenged with mycoparasitic and chemical control agents. *Biol. Control* **2017**, *109*, 51–57. [CrossRef]
174. Hoover, J. Strategies for managing fusarium head blight in western Canada: A review of literature. In Proceedings of the 7th Canadian Workshop on Fusarium Head Blight, Winnipeg, MB, Canada, 27–30 November 2011. Available online: http://www.cwfhb.org/programs/7_CWFHB_2011_Winnipeg.pdf (accessed on 15 April 2021).
175. King, C. Fighting Fungus with Fungus. *Top Crop Manager*. 2010, pp. 32–33. Available online: <https://www.mydigitalpublication.com/publication/?m=1031&i=47114&p=32&ver=html5> (accessed on 11 May 2021).
176. King, C. A Beneficial Parasite: A Fungus Native to Saskatchewan Helps Control Both Fusarium and its Toxins. *Top Crop Manager*. 2020. Available online: <https://www.topcropmanager.com/a-beneficial-parasite/> (accessed on 11 May 2021).
177. Arnason, R. Discovery of Biological Control Benefits Resistant Varieties. *The Western Producers Crops*. 2011. Available online: <https://www.producer.com/crops/discovery-of-biological-control-benefits-resistant-varieties/> (accessed on 10 May 2021).
178. Lysøe, E.; Seong, K.-Y.; Kistler, H. The Transcriptome of *Fusarium graminearum* During the Infection of Wheat. *Mol. Plant Microbe Interact.* **2011**, *24*, 995–1000. [CrossRef]
179. Udendhran, R.; Balamurugan, M. Towards secure deep learning architecture for smart farming-based applications. *Complex Intell. Syst.* **2021**, *7*, 659–666. [CrossRef]
180. Bauer, A.; Bostrom, A.G.; Ball, J.; Applegate, C.; Cheng, T.; Laycock, S.; Rojas, S.M.; Kirwan, J.; Zhou, J. Combining computer vision and deep learning to enable ultra-scale aerial phenotyping and precision agriculture: A case study of lettuce production. *Hortic. Res.* **2019**, *6*, 70. [CrossRef]