

**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**

Agnès Champeil, Thierry Doré, Jean-Francois Fourbet

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3 **Review — *Fusarium* head blight: epidemiological origin of the effects of cultural**  
4 **practices on head blight attacks and the production of mycotoxins by *Fusarium* in wheat**  
5 **grains**

6  
7 Champeil A., Doré T. and Fourbet J.F.

8 UMR of Agronomy INRA INA-PG, 78850 Thiverval-Grignon, France

9 [champeil@grignon.inra.fr](mailto:champeil@grignon.inra.fr), [dore@inapg.inra.fr](mailto:dore@inapg.inra.fr), [fourbet@grignon.inra.fr](mailto:fourbet@grignon.inra.fr)

10  
11 **Abstract**

12 *Fusarium* head blight is an ancient disease and is very common throughout the world. In this  
13 article, we review current knowledge concerning the effects of cultural practices on the  
14 development of head blight and the production of toxins in the field. The qualitative effects of  
15 these practices on the severity of the disease and/or the production of toxins are in the process  
16 of being elucidated but, in many cases, detailed studies have not yet been carried out or  
17 conflicting results have been obtained. However, it should be noted that these effects have not  
18 yet been quantified. Three different cultural practices are today considered to be of prime  
19 importance for combating this disease and the production of mycotoxins: deep tillage, the  
20 choice of the preceding crop in the rotation and the choice of appropriate cultivar, as varietal  
21 effects exist.

22  
23 **Keywords: head blight, *Fusarium*, *Microdochium*, wheat, cropping systems, mycotoxins**

24  
25 **Contents**

26 Introduction ..... 2  
27 1- Epidemiological study of the disease ..... 4  
28 2- Effects of various cultural practices on the disease..... 13  
29 3- Effects of various cultural practices on the production of mycotoxins by *Fusarium* ..... 24  
30 4- Relationship between the disease and toxin production..... 28  
31 Conclusion..... 39  
32 References ..... 44

## 35 **Introduction**

36 Head blight is a fungal disease affecting many small-grain plant species worldwide [1-10].  
37 Affected wheat grains are small, light (the kernel is degraded), wrinkled and sometimes  
38 covered with a white or pink down. Rings or oval stains with brown edges and clear centres  
39 may be visible on the back of the grain [1,2,6,11-13] and on the external surface of the glumes  
40 (Figure 1). Head blight results from the development of a complex of two genera of  
41 pathogenic fungi: *Microdochium* and *Fusarium* [8,14-22]. *Microdochium* consists of a single  
42 species, *M. nivale*, made up of two subspecies: *M. nivale nivale* and *M. nivale majus*.  
43 *Fusarium* consists of five main species (*F. graminearum*, *F. culmorum*, *F. avenaceum*, *F.*  
44 *poae* and *F. triticum*), with several strains per species. The most common of these species are  
45 *F. graminearum* and *F. culmorum* [2,4,5,23-27], which are also the most pathogenic, reducing  
46 the size of the grains and the final yield [24,28].

47

### 48 ***The consequences of head blight contamination***

49 This disease was first described at the end of the 19th Century in England [29] and has  
50 economic consequences. Yield losses (flower abortion, decrease in grain weight, highly  
51 damaged grains eliminated during threshing) of wheat may reach 15 stooks (180 sheaths) per  
52 hectare in cases of severe disease (according to the technical organisation dealing with cereals  
53 in France), or 50-60% [1,6,7,30] about once every three to five years [6,16]. In 2000, the year  
54 with the highest levels of disease in the last ten years, up to 100% of ears were affected on  
55 certain French plots [31]. There is a non-linear relationship between the percentage of ears  
56 attacked by *M. nivale* and thousand-grain weight [19,32]. Head blight also decreases the  
57 nutritive and technological quality of the grains (degradation of proteins, baking quality,  
58 nutritive value etc.).

59

60 Since the 1990's, interest in food health and safety has increased. For cereal products, one of  
61 the most important elements of alimentary risk is the possibility of mycotoxin accumulation  
62 on or in the grains [33-35]. Mycotoxins are the products of fungal secondary metabolism  
63 [34,36], which occurs when growth stops altogether or slows markedly [37]. These toxins  
64 result from adaptation of the growth of the fungus to stressful situations [37]. Head blight is  
65 the principal means by which mycotoxins develop in the grains before harvest. The  
66 mycotoxins produced in crops suffering from head blight are produced by fungi of the genus  
67 *Fusarium*. Fungi of the genus *Microdochium* are thought not to produce toxins [8]. Levels of

68 the mycotoxins produced by *Fusarium* cannot be reduced after harvest by means of classical  
69 transformation procedures [38]. These toxins are chemically and thermally stable [39,40].  
70 They may therefore be found in the raw material (grains) and in transformed products (flour,  
71 bread) or in products from animals fed with contaminated grain (meat, milk, eggs) [35].

72

73 Mycotoxins cause two types of problem. They may generate technological problems, such as  
74 negative effects on malting for beer (inhibition of enzyme synthesis) or on fermentation  
75 (inhibition of yeast growth), or even on the production of high-quality bread [38]. Indeed, *F.*  
76 *graminearum* modifies a protease that acts on gluten. Damage to this enzyme prevents the  
77 dough from retaining the gas produced during fermentation, resulting in heavier, less  
78 voluminous bread. Furthermore, *F. graminearum* also destroys starch grains, protein stores  
79 and cell walls [6,41,42].

80

81 Mycotoxins may also cause poisoning incidents, the principal symptoms of which are nausea,  
82 lethargy, fatty and cancerous infiltrations and possibly even death [38,43]. Six main families  
83 of mycotoxins have been studied to date: aflatoxins (B1, B2 and M1), ochratoxin A,  
84 trichothecenes (T2 toxin, deoxynivalenol etc.), zearalenone and fumonisins. *Fusarium*  
85 produces toxins of the last three groups, which may attack the liver, the kidneys, the nervous  
86 system, the circulatory system, the endocrine system, the skin, the digestive tract and the  
87 blood [44]. These toxins are thought to be highly carcinogenic, although this assertion  
88 remains unproven in most cases. However, T2 toxin has been shown to be carcinogenic [44].  
89 The absorption of trichothecenes has resulted in the poisoning of animals and humans in the  
90 United States, Canada, the Soviet Union (as it was at the time) and Japan [39]. The massive  
91 absorption of T2 toxin was responsible for toxic alimentary aleukia in several regions of the  
92 USSR between 1942 and 1947 [34,39]. Pigs are the animals most reactive to deoxynivalenol,  
93 whereas poultry seem to be particularly resistant. Effects on humans have been observed after  
94 the absorption of 300 to 8000 µg/kg deoxynivalenol in India [8]. This led the public  
95 authorities to establish norms fixing maximum deoxynivalenol levels in food destined for  
96 human consumption. The European Commission has proposed setting limits of 750 µg/kg  
97 (750 ppb) in cereals and 500 µg/kg in cereal-based products such as flour. A major problem in  
98 trichothecene toxicology is the current lack of knowledge concerning the risk of food  
99 contamination by several of these toxins at the same time, as *Fusarium* can produce a  
100 multitude of toxins simultaneously [34].

101

## 102 ***Factors triggering attacks***

103 The climate is known to have an effect, and is said to be the principal factor affecting the  
104 development of head blight on oat in Norway [45]: humidity determines the severity and  
105 intensity of the disease whereas precipitation and total radiation determine inoculum levels  
106 (number of spores per ear) [46]. The climate plays a role at all stages of development of the  
107 fungus [47-49]. The climate during winter affects the survival of the fungus on crop residues:  
108 during mild periods of the winter, the fungus is capable of sporulating on the cereal stubble  
109 debris [48]. In spring, the climate determines the type and quality of spores produced,  
110 together with the date of their dispersal and thus the intensity of the disease. Indeed,  
111 coincidence in the dates of spore dispersal and crop flowering, marking the start of the critical  
112 phase of infection for plants [2,6,16,50], is of prime importance in determining the intensity  
113 of the attack. In summer, together with the virulence characteristics of the fungal strain and  
114 the intrinsic resistance of the plant, climate controls competition between pathogen species,  
115 thereby controlling the rate of mycelium growth in the plant [47]. The climate may also affect  
116 the production of fungal toxins in the field [2,51-54]. The effects of climate are such that the  
117 levels of deoxynivalenol recorded on diseased crops in the field and in the glasshouse are not  
118 correlated [49]. The effects of climate are of course uncontrollable, but also difficult to predict  
119 due to their complexity.

120

121 However, in any given year, two neighbouring plots may display different levels of infection  
122 [19,55]. It must therefore be possible, to some extent, to control the production of mycotoxins  
123 by modifying the cropping system.

124

125 The literature review below aims to identify and to elucidate the relationships linking  
126 cropping systems to grain contamination by *Fusarium*, *Microdochium* or to mycotoxin  
127 production by *Fusarium*.

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129

## 130 **1- Epidemiological study of the disease**

### 131 **1-1. Impact of climate on the species complex**

132 The species of fungi making up the pathogenic complex responsible for head blight may  
133 differ from year to year and from one region to another, particularly as a function of climate  
134 [6,11,56] and wheat variety [56,57]. Indeed, climate partly controls competition between the

135 various species. Thus, at flowering, the development of *Microdochium* is favoured by lower  
136 temperatures [6,58] and rainfall [59], whereas that of *Fusarium* is favoured by high  
137 temperatures [58] and storms [59]. *F. graminearum* is generally the predominant *Fusarium*  
138 species in warmer regions, whereas the predominant species in cooler regions are *F.*  
139 *culmorum* [6,16,28,60], *F. avenaceum* [6] and *M. nivale* [6,16]. Furthermore, in spring  
140 (before ear emergence), drought favours the development of symptoms of *F. graminearum*  
141 and *F. culmorum* infection at the base of the plant, at the expense of other species of  
142 *Fusarium* [59].

143

### 144 **1-2. Sources of inoculum**

145 Several reservoirs of the parasite complex responsible for head blight are known. The primary  
146 reservoir of inoculum is debris from the previous crop [2,61]. All species of *Fusarium* and  
147 *Microdochium* can survive as saprophytes [6]. However, the pathogens survive longer on  
148 residues that do not degrade easily, such as stem nodes [2].

149

150 Other sources of inoculum include numerous plant hosts. These may be cultivated plants  
151 and/or weeds, such as grasses and evergreen weeds [6]. However, the roles of weeds,  
152 inoculum source (site at which the fungi develop) and supports for survival have not yet been  
153 determined [62]. Seeds are the other major source of inoculum, making it possible for the  
154 disease to begin in autumn in the case of grains contaminated with mycelium [14,16]. The  
155 further into the grain the mycelium has penetrated, the lower is the chance of the grain  
156 germinating. The soil may also be contaminated [1,2]. Soil-borne infections take hold less  
157 rapidly than seed-borne infections, resulting attacks affecting essentially the collar and the  
158 upper parts of the roots [2,14]. Soil humidity, particularly during winter, decreases the  
159 pathogen survival rate [2].

160

161 The flag leaf is the principal site of spore production and source of ear infection in the plant  
162 [32].

163

### 164 **1-3. Production and maturation of inoculum**

165 During the winter, the fungi survive as chlamydo spores, mycelium or propagules [2,6].  
166 Sporulation, which involves the production of various types of spore, occurs during mild  
167 periods [48]. Four types of asexual inoculum may be produced, depending on the species  
168 concerned: macroconidia, microconidia, chlamydo spores and hyphal fragments, the size and

169 form of which depend on the species of pathogen [63]. The macroconidia of *F. graminearum*  
170 may contain one to seven septa — most frequently three to seven — and measure 20 to 105  
171  $\mu\text{m}$  in length (mostly 35-62  $\mu\text{m}$ ), and 2 to 56  $\mu\text{m}$  in width [2]. This type of inoculum may be  
172 available during the entire crop cycle [16]. Sexual spores, known as ascospores, may also be  
173 produced by reproductive organs called perithecia. Only three of the species most frequently  
174 responsible for head blight appear to be capable of producing ascospores in the natural state:  
175 *M. nivale*, *F. graminearum* and *F. avenaceum* [6,16,64]. These three species produce  
176 ascospores that are similar in size and form. They generally have three septa and measure  
177 about 17.5 – 26  $\mu\text{m}$  x 3.5 – 5  $\mu\text{m}$  [2]. They take a long time to mature and therefore  
178 contamination cannot occur before ear emergence [16]. Caron reported that *M. nivale*  
179 produces ascospores from May onwards in France and that *F. graminearum* produces its  
180 ascospores later in the year.

181

182 In the laboratory, the optimal environmental conditions for production of the principal forms  
183 of inoculum — macroconidia and ascospores — depend on the species and the environment.  
184 These results probably cannot be directly transposed to natural conditions. Table 1 sums up  
185 the principal results reported in the literature.

186

187 The maturation of the inoculum has been studied in less detail and depends on interactions  
188 between environmental factors, both in the laboratory and in the field. The maturation of the  
189 inoculum is thought to be hindered by drought and cold in autumn and winter [65]. In the  
190 laboratory, perithecia have been reported to mature in six to nine days [66], or nine to ten  
191 days [2] after their initiation in ideal conditions. In contrast, perithecium maturation takes two  
192 to three weeks in the field whereas conidiospores can be produced in large quantities within a  
193 few hours [59]. According to another study, macroconidia reach maturity in three weeks in  
194 the field [67]. The rate of maturation of perithecia depends on light [66,68].

195

196

#### 197 **1-4. Dispersal of the inoculum**

198 The inoculum is dispersed by various animal vectors. Mites (*Siteroptes graminum*) transport  
199 *F. poae* spores [6]. Insects are parasitised by certain species of *Fusarium*, *F. episphaeria* (SN.  
200 and H) in particular [63]. *F. avenaceum*, *F. culmorum* and *F. poae* have been isolated on  
201 various insects including *Musca domestica* (housefly), *Hypera punctata* (clover leaf weevil)  
202 and *Melanoplus bivittatus* [69].

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The possibility of inoculum dispersal by the systemic route has long been debated; after cutting the peduncle of a wheat ear into segments, Atanasoff (1920)[1] observed *F. graminearum* in the segments close to the ear but not in those close to the flag leaf. Following inoculation of the base of the wheat stem, only 3% of plants display colonisation beyond the second node and no fungus is detected beyond the fifth node [70]. In another study [71], the tops of plants produced from seeds inoculated with *M. nivale* presented similar numbers of perithecia to those of plants grown from healthy seeds, even though the plants grown from contaminated seeds had more perithecia at the base of the stem. This finding confirms the lack of relationship between head blight and foot rot due to *Fusarium* [6,16]. To date, these two infections have been considered to be essentially independent [1,16]. However, it should be noted that the presence of the parasite at the base of the stem (below the second node) may disturb the water supply to the ear sufficiently to cause shrivelling at the slightest increase in temperature [14]. Shrivelling of part of the ear is one of the symptoms of head blight. A confusion of symptoms is possible: in some cases, shrivelling of the ears may be due to the presence of the parasite at the base of the stem rather than on the ears. In addition, following the artificial inoculation of spring wheat seeds, sown in pots, with *F. culmorum*, the pathogen is isolated from all nodes and from the ears [72]. Similarly, Snijders (1990a)[73] observed the pathogen in stem tissue 70 cm above soil level after inoculating plants at soil level. Many cases of infection with various species of *Fusarium* have been observed after the sterilisation of weeds [62] and wheat [74] with sodium hypochlorite, which suggests that these infestations are endophytic. Evidence that this is indeed the case is provided by the observation that the fungal mycelium is capable of infecting both the parenchyma and the vascular tissue, in which it is able to travel more rapidly [50]. Indeed, after the injection of spores into the rachis, these authors showed that the pathogen was able to migrate within the plant and that it propagated more rapidly longitudinally than transversely. However, it is possible that this route of contamination is only possible in a few species of plant, for a few species of fungus or for a few plant-fungus interactions.

Dispersal by leaf-to-leaf contact also seems likely as the pathogens (*Microdochium* and *Fusarium*) are found on the leaves [15]. Atanasoff (1920)[1] also observed infection by contact. This mode of dispersal should be considered to be a specific case of aerial contamination.



237 Aerial contamination by ascospores and conidia is thought to be the principal source of ear  
238 contamination [1]. This means of contamination has been studied by many groups and  
239 involves two possible modes of dispersal: splashing and wind [2,6,7,16,64]. The relative  
240 importance of these two modes of dispersal depends on the climate and the species making up  
241 the pathogenic complex in the year and region studied and the capacity of these species to  
242 produce ascospores.

243

244 Splashing transports spores, macroconidia in particular, that are too heavy to be transported  
245 by wind [35]. The density of *F. graminearum* macroconidia in the air above the canopy has  
246 been estimated to be only one twentieth that of ascospores from the same species [75].

247

248 Splashing is the only means of dispersal of *M. nivale* conidia [71]. Millar and Colhoun were  
249 able to trap conidia only during simulated rain conditions, whereas Fernando *et al.* (2000)[75]  
250 observed a peak in the release of macroconidia of *F. graminearum* in the air one to two days  
251 after rainfall following a long period of drought. These two studies demonstrate the major role  
252 played by rainfall in the dispersal of spores. In the laboratory, a conidium of *F. graminearum*,  
253 produced in a sporodochium (the asexual fruiting body bearing the conidiophores) from wheat  
254 straw, receiving a single drop of water 5 mm in diameter falling from a height of 6 m, was  
255 displaced by up to 45 cm in the vertical plane and 90 cm in the horizontal plane [17]. In the  
256 same conditions, a conidium of *F. culmorum* was displaced 60 cm vertically and at least 1 m  
257 horizontally [17,76]. The spores of *F. poae* may reach a height of 58 cm and may travel 70  
258 cm horizontally in rebounding raindrops [76]. Splashing alone is therefore sufficient to  
259 transfer a conidium from crop residues or the stem base to the ear (in one or several rebounds,  
260 relayed by the leaves), assuming there is no obstacle. In the canopy, the leaves of the  
261 neighbouring plants form an obstacle. Under simulated rainfall, the wheat canopy reduces the  
262 dispersal of *Septoria tritici* spores by 33%, to 15 cm, in the horizontal plane from the source  
263 of infection, and by 63% in the vertical plane [77]. A leaf positioned low in the canopy is  
264 principally infected at its base whereas leaves higher up in the canopy are principally affected  
265 at their tips, from which the spore can rebound, resulting in their transfer to another leaf [76].  
266 Splashing is considered to be the most likely means of dispersal because *F. avenaceum*, *F.*  
267 *culmorum* and *M. nivale* have been observed on the flag leaf [15]. Rainfall plays an important  
268 role in the development of the disease. In years with major epidemics of head blight or rotting  
269 of the ear due to *F. graminearum*, precipitation levels are generally high [2,7,15,78].  
270 Similarly, in spray irrigation trials, up to 89% of ears may be infected, versus 0% in non-

271 irrigated controls [6]. Furthermore, humidity and rainfall in spring favour the formation of  
272 perithecia [59].

273

274 In natural conditions, perithecia are formed in only the three species that generate ascospores:  
275 *F. graminearum*, *F. avenaceum* and *M. nivale* [6,16,64]. Ascospores are a form of inoculum  
276 that can be transported by the wind [6,16]. The wind has long been considered the principal  
277 vector for spore dispersal, and damage tends to be greatest in the direction of the prevailing  
278 wind [1]. Following the artificial inoculation of a zone of field with maize grains infected  
279 with *F. graminearum*, *F. graminearum* spores travel less than 5 m [75]. However, Stack  
280 (1997)[79] indicated that the dispersal distance of *F. graminearum* spores is proportional to  
281 the size of the area inoculated. According to this model, head blight levels halve as the  
282 distance to be travelled by the inoculum doubles. The maximum density of ascospores  
283 observed is 1500 spores/m<sup>2</sup>, at 1.5 m from the source of inoculum [75]. The dispersal of *F.*  
284 *graminearum* ascospores in the field occurs at temperatures of 13 to 22 °C, with a relative  
285 humidity of 95 to 100% [2]. Ascospore release is maximal at a relative humidity of 100%, but  
286 also occurs at lower levels of relative humidity [66].

287

288 Despite the importance of humidity, studies on *F. moniliforme* [67,80] and *F. graminearum*  
289 [75] have shown that the release of ascospores is not directly linked to rainfall. Indeed,  
290 ascospore release peaks one to four days after rainfall [67,75] of at least 5 mm or a relative  
291 humidity exceeding 80% [67]. Paulitz found that a shower of rain in the evening did not  
292 inhibit spore release but that heavier rain (at least 5 mm), a relative humidity exceeding 80%  
293 continuously throughout the day or the alternation of rain and high humidity inhibited the  
294 release of ascospores during the night. This result contrasts with another study showing the  
295 release of ascospores during the night following a day on which 8 mm of rain fell [81]. The  
296 correlation between spore release and humidity suggests that the maturation of perithecia  
297 requires a certain level of humidity. This would account for the results of Fernando *et al.*  
298 (2000)[75], who observed four periods of spore release over a period of 20 to 30 days.

299

300 The process of ascospore release may be described as follows [71]: after a period of drought  
301 (air with low water vapour content), free water (rain or heavy dew) triggers the release of  
302 ascospores. Paulitz (1996)[67] speculated that the increase in relative humidity during the  
303 evening after a dry day might increase the turgor pressure of the asci, which contain a vacuole  
304 at high osmotic pressure [82]. This high osmotic pressure is generated by the accumulation of

305 mannitol and the flux of potassium and calcium ions [66]. Calcium ions are also involved in  
306 signalling for ascospore discharge [66]. This increase in pressure triggers the release of  
307 ascospores, which are expelled from the perithecia in a gelatinous substance, which then dries  
308 out, releasing the ascospores into dry air. This would account for the finding that a trap placed  
309 above the canopy captures several *M. nivale* ascospores in dry conditions and that the  
310 concentration of these spores increases by a factor of 20 in the 10 minutes following the  
311 application of a fine simulated dew [71]. A similar phenomenon has also been observed for  
312 the ascospores of *F. graminearum* [6]. Similarly, in control conditions, the maximal release of  
313 *F. graminearum* ascospores is regulated by dehydration of the perithecia in non-saturated  
314 humidity conditions [83]. Furthermore, the time at which the spores are released is strongly  
315 correlated with the increase in humidity following the decrease in temperature that occurs at  
316 the end of the afternoon [67]. Paulitz observed that ascospore release began before the leaves  
317 became humid at the base of the canopy (which occurred at 22-24 h), towards 17 or 18 h for  
318 temperatures varying between 11 and 30 °C and values of relative humidity of between 60  
319 and 95%. These results have been confirmed by the observation, in control conditions, that  
320 the maximal release of *F. graminearum* spores occurs at temperatures between 11 and 23 °C,  
321 with 16 °C the optimal temperature [83].

322  
323 After temperature and humidity, the next most important environmental factor affecting  
324 ascospore release is light. Light is required for the production and maturation of perithecia  
325 [68] and ascospores [83]. In contrast, the process of ascospore release does not directly  
326 require light, as it has been observed during the night: 4 to 5 h before sunrise [67]. This result  
327 has been corroborated by studies in control conditions, which also showed that light had no  
328 effect on ascospore release [66,68].

329  
330 Finally, the periodicity of aerial dispersion has been studied. The release of conidia is not  
331 periodic, whereas that of ascospores is, particularly in *F. graminearum* [75]. Indeed, Fernando  
332 *et al.* detected no ascospores in the air between 12 and 16 h, whereas ascospore density was  
333 maximal between 20 h and 08 h (1.5 times higher than at other periods of the day). Other  
334 authors have reported that ascospore density peaks between 21 h and 08 h [84]. Similarly,  
335 Paulitz reported that spore release often occurs between 18 h and early morning (04 h – 08 h),  
336 mostly before midnight, with a peak at around 23 h [67]. For *F. graminearum*, the maximum  
337 ascospore concentration observed is of the order of 4333 ascospores/mm<sup>3</sup> in one hour. Paulitz  
338 also reported spores of other species of *Fusarium* to be continuously present in the air, but

339 with densities varying according to the period of the day. Paulitz identified four distinct  
340 periods in the day, and found that spore density was low in the morning. For *F. monoliforme*,  
341 a large number of ascospores was found to be released between 17 h and 08 h, with a peak  
342 towards 02 h [80].

343

344 Although dispersal over large distances seems to be possible, local aerial contamination  
345 appears to predominate, in that two neighbouring plots of land planted with the same variety  
346 may display very different levels of contamination [19,55].

347

#### 348 **1-5. Infection and colonisation of the ear**

349 In wheat, the most critical period corresponds to the moment at which the degree of resistance  
350 to primary infection and to the propagation of fungal hyphae in the plant is lowest. This  
351 period has been defined differently by different authors, but the first half of the grain-filling  
352 period is the most critical. Several authors have identified ear emergence [64,85] or the mid-  
353 grain-filling stage [47] as the most critical, but most authors consider anthesis to be the most  
354 susceptible to attack in wheat [12,16,64,86,87], with susceptibility decreasing strongly after  
355 the start of the dough stage [12,16,47,64,86,87]. Elimination of the male organs from wheat  
356 ears decreases the frequency of infection by *F. graminearum* [47,86]. Similarly, sterile wheat  
357 lines are less susceptible to head blight than fertile lines [88]. This, together with extensive  
358 colonisation of the anthers by this fungus [86] indicates that the growth of *F. graminearum* is  
359 stimulated in these structures, suggesting that entry into the anthers during anthesis has major  
360 consequences for the grain (degradation). Two substances — choline chloride and betaine  
361 hydrochloride — are much more concentrated in the anthers than elsewhere [89]. These  
362 substances favour the extension of conidial hyphae, but not the germination of spores of *F.*  
363 *avenaceum*, *F. culmorum* and *F. graminearum* [89]. Once the spores have germinated, the  
364 propagation of the hyphae is therefore more strongly favoured in the flower parts than in the  
365 other organs. However, the difference in susceptibility of different stages is probably due to  
366 the fact that the critical receptivity peak observed (degree of resistance to primary infection  
367 and propagation of fungal hyphae) is dependent on many factors. These factors include the  
368 wheat cultivar [12,47,50,90], air temperature and humidity [12,47] at the critical stage for the  
369 plant and, before this stage, development of the inoculum, and the stage of maturity of the  
370 plant at the time of hyphal penetration [12]. The testa (seed coat) is more resistant to  
371 penetration when the grains are mature [12]. However, this finding remains open to debate  
372 because a recent study [91] showed that the date of inoculation has no effect on the final level

373 of *F. graminearum* colonisation. In maize, susceptibility to the disease is maximal at the start  
374 of silk development and decreases throughout the grain-filling period [92]. In *Arabidopsis*  
375 *thaliana*, tobacco, tomato and soybean, the floral tissues are also the most heavily infected  
376 [93].

377

378 The infection process is very similar in susceptible and resistant varieties [94]. The pathogen  
379 first penetrates host tissues 36 to 48 h after inoculation [94]. The first organs affected are the  
380 lemmae and the tip of the ovary [94,95], the anthers [12] and/or the spikelets, glumes and the  
381 rachis [50]. It remains unclear which of these organs is most frequently the initial zone  
382 infected, but this probably depends on the date of infection and possibly also on the infecting  
383 species. The penetration of the fungus into the ear is favoured by relatively low temperatures  
384 and high humidity [64]. The presence of and colonisation by a large number of hyphae are  
385 required for infection [50]. The hyphae of *F. graminearum* and/or *F. culmorum* invade the  
386 host tissue directly [94] or via the stomata [50]. They then propagate into the ear, passing  
387 through and around the cells in their path [12,94-96], degrading the cells that they  
388 contaminate [50,94,96]. They move principally towards the rachis [94,95] or towards the  
389 young grains, which they invade via the parenchyma of the pericarp close to the embryo [50].  
390 A short time after flowering, the parenchyma of the pericarp begins to break down, the nuclei  
391 and cytoplasm of the cells disappear and the walls of the cell break [12]. *F. graminearum* is  
392 then able to enter this tissue and propagate throughout the grain [12]. Indeed, *F. graminearum*  
393 hyphae penetrate the thinner cell walls of the parenchyma tissue more easily than the thicker  
394 cell walls of other more specialised tissues [12]. Birds also facilitate infection by creating  
395 lesions on the grains that favour the penetration of fungi [2].

396

397 Infection of the ears by macroconidia of *F. graminearum* [47], *F. avenaceum*, *F. culmorum*,  
398 *F. poae* and *M. nivale* [6] is optimal at 100% relative humidity and 25 °C, and takes place  
399 over 24 to 60 hours. Infection may occur at temperatures of 20 to 30 °C, but is negligible at  
400 temperatures below 15 °C [47]. Below 18 °C, the conidia of *M. nivale* are more competitive  
401 than those of *Fusarium*, resulting in higher levels of *M. nivale* contamination. Furthermore, a  
402 period of four to eight days of low humidity reduces the incidence of infection, but does not  
403 entirely eliminate it [47]. The germination of macroconidia, ascospores and chlamydospores  
404 is maximal between 0 and -20 bars and is inhibited between -60 and -80 bars [97].  
405 Ascospore germination is inhibited beyond a threshold of -30 bars (-3 MPa) of water

406 potential after eight hours of drought [67]. Free water or a low water potential also favours  
407 infection, reducing the length of the incubation period [67].

408

### 409 **1-6. Incubation and sporulation**

410 Perithecia and conidia develop on the surface of spikelets and of the rachis in humid climatic  
411 conditions [2]. The duration of the incubation period required decreases with increasing  
412 humidity [16]. In conditions of saturating humidity, the duration of incubation required for the  
413 appearance of macroconidia of *F. culmorum* and *F. graminearum* on the ear was 12 days at  
414 14 °C, less than five days at 20 °C and less than three days at between 25 and 30 °C [2,16].  
415 More spores are formed after a long period of high humidity. This may then result in the  
416 infection of later crops, such as maize.

417

418

## 419 **2- Effects of various cultural practices on the disease**

420 Various studies have identified different elements concerning the effects of agricultural  
421 practices (crop rotation, crop management) on head blight attacks.

422

423

### 424 **2-1. Effects of the crop succession history of the field**

425 The effects of crop rotation have been studied in detail. They depend on the preceding crop,  
426 whether that crop is a potential host for the pathogens responsible for head blight, and the  
427 frequency of the crop concerned in the rotation. The shorter the rotation, the higher the  
428 frequency of head blight. Thus, head blight is most frequent when the susceptible crop occurs  
429 frequently within the rotation [7]. The density of crop residues left in the field [19], their  
430 nutritional value [19] and pathogen competition may also modify the effect of crop rotation  
431 [19,62].

432

433 Head blight contamination is more severe if the preceding crop is maize, durum wheat or oats,  
434 rather than wheat or barley [14,19,48,64,98-101], and even less contamination is observed  
435 following other crops [16,48]. For example, the frequency (%necrotic ears) and the severity  
436 (%necrotic spikelets) of the disease on wheat are lower following soybean than following  
437 another wheat crop, or worse still, maize [19]. This may be accounted for in two ways.  
438 Firstly, soybean crops leave fewer residues than wheat crops, which in turn leave fewer

439 residues than maize crops [7,19,100]. Secondly, the principal *Fusarium* species infecting  
440 soybean is *F. sporotrichioides*, whereas wheat and maize are more frequently affected by *F.*  
441 *graminearum* [19], like sorghum [59]. Thus, in addition to the density of residues left by the  
442 preceding crop, the nature of the preceding crop is important in determining the pathogen  
443 species likely to infect the next crop in the rotation. The preceding crop affects the  
444 composition of the pathogen complex throughout the following year. Thus, a preceding crop  
445 of potato will allow the development of only *F. culmorum* and *F. sambucinum*, whereas a  
446 preceding wheat crop will allow the development of all *Fusarium* species [62]. It should also  
447 be noted that the highest levels of *F. graminearum* contamination are recorded on grains  
448 harvested from wheat crops following maize in the rotation, whereas *F. avenaceum* and *F.*  
449 *poae* are the most common species found in grain samples harvested from wheat crops  
450 following a crop other than maize [60]. It is therefore advisable to introduce non-host plants  
451 into crop rotations, to limit the disease [7]. Indeed, the use of oats as the preceding crop  
452 results in a doubling of head blight inoculum in the soil [64,98] even though this crop is  
453 unaffected by the disease [14] whereas a preceding crop of sugarbeet, which is hardly affected  
454 by *Fusarium*, halves the incidence of the disease in the subsequent wheat crop [14,64,98].  
455 Similarly, flax can be used as a clean-up crop [16], as can alfalfa, after which no disease is  
456 observed [102]. However, the quantity of crop residues and the development of pathogens on  
457 the preceding crop are not the only explanations of the effect of preceding crop. Wheat crops  
458 present different levels of infection depending on whether they follow wheat or durum wheat  
459 in the rotation, even though these two preceding crops produce similar amounts of residues  
460 and are equally permissive for *Fusarium* development.

461

462 Among the other elements possibly involved, the amount of nitrogen in crop residues may  
463 affect the possible duration of colonisation by *Fusarium*. Indeed, the high nitrogen content of  
464 maize residues may result in a longer period of colonisation of those residues by the fungi,  
465 strengthening these fungal populations [2] and favouring disease.

466

467 It is also likely that proteolytic soil bacteria, favoured by the low carbon to nitrogen ratio  
468 under alfalfa, compete with *Fusarium* species, limiting their development and thus their  
469 ability to attack the subsequent crop [62,102]. It should also be noted that, at the end of the  
470 cropping cycle, buried cereal stubble provides an ideal substrate for saprophytes, increasing  
471 the carbon to nitrogen ratio, possibly resulting in the reappearance of the disease [98].

472

473 **2-2. Effects of soil tillage**

474 The effects of soil tillage are also well known, at least from a qualitative point of view. As for  
475 yellow and brown rusts [103], limited soil tillage increases the frequency of head blight  
476 [7,19,100], whereas deep tillage (ploughing) decreases it [7,19,64,90,101]. Similarly, limited  
477 tillage systems increase the number of *Fusarium* propagules in the soil [14] whereas  
478 ploughing decreases inoculum levels [98].

479

480 This effect of ploughing may be accounted for in several ways. Ploughing has several direct  
481 and indirect effects on the structure [104,105] and microclimate of the soil [105,106] and  
482 therefore on the development of fungi [105], notably those of the genus *Fusarium*. Limited  
483 tillage and direct drilling systems make it harder to deliver the seed to the required position  
484 during sowing, decrease soil porosity, increase structural stability and litter on the surface.  
485 The litter layer protects the soil from rain (battering and erosion), and increases surface  
486 humidity (by limiting evaporation), soil temperature and inertia. This layer breaks down into  
487 organic matter, enriching the surface soil in carbon and organic forms of nitrogen, and  
488 modifying its chemical and biological characteristics. Stores of inorganic carbon increase, the  
489 supply of soil mineral nitrogen decreases very slowly and the mineralisation process is  
490 delayed. The microbial biomass (bacteria and fungi) increases at the surface and its turnover  
491 rate increases in the first 10 cm of soil. Humus is thus less well degraded in this zone, which  
492 rapidly becomes more acidic, favouring the development of fungi over that of bacteria. Thus,  
493 about 90% of the *Fusarium roseum* population is located in the first 10 cm of soil [98].  
494 Furthermore, the development of this fungus depends on soil aeration [14]. Although this  
495 pathogen can survive for four years [16] at a depth of 20 to 25 cm, it is only active and able to  
496 develop on plant debris in the first 5 cm of soil [14]. Beyond a depth of 15 cm, the incidence  
497 of *Fusarium oxysporum* attacks on carnation decreases as the depth of propagule burial  
498 increases [107]. However, this result cannot be readily transposed to other species because  
499 *Microdochium nivale* and *Fusarium avenaceum* do not produce chlamydospores [108] and the  
500 number of propagules in the soil does not necessarily affect *Fusarium* foot rot levels due to  
501 *Fusarium culmorum* [109]. Thus, for certain species, propagules may simply ensure the  
502 survival of the fungus rather than serving as an inoculum [98,109].

503

504 The development of the *Fusarium roseum* population also depends on the quantity of  
505 substrate (crop residues) available [14]. Thus, a decrease in the density of residues on the  
506 surface of the soil (after ploughing for example) helps to decrease the production of inoculum



507 [100,110] and the quantity of spores available for dispersal [109]. This hypothesis seems to be  
508 validated by the observation that there are fewer *Fusarium culmorum* propagules if the  
509 stubble from the preceding crop is burnt than if it is buried [109]. However, an opposite effect  
510 on the disease is observed. Thus, in years in which there are high levels of *Fusarium* infection  
511 at the base of the stem, disease levels have been found to be higher if the stubble from the  
512 previous crop was burnt than if it was incorporated into the soil [109]. A similar result was  
513 previously reported for eyespot [111]. Similarly, the supplementary incorporation of infected  
514 stubble not originating from the preceding crop decreases the incidence of *Fusarium* attacks  
515 resulting in wheat foot rot to levels below those observed in soils in which the density of  
516 residues has not been modified [112]. However, these results may be explained if stubble  
517 limits conidium dispersal by splashing, favours growth of the canopy [111], or modifies the  
518 infection process. In Yarham's study [111], the addition of stubble did not affect leaf area  
519 index, photosynthetic potential or the number of leaves. It also resulted in no decrease in the  
520 density of wheat or weeds and had no effect on the size of the plants. All these factors might  
521 have facilitated spore dispersal. In the infectious process, four aspects may be affected by the  
522 presence or absence of stubble. Firstly, the resistance of the plants may be affected by  
523 increases in the amount of silica (SiO<sub>2</sub>) in the leaves of the wheat plants following the  
524 incorporation of additional stubble [112]. The hypothesis that silicon plays a protective role  
525 arises from work [103] indicating that this element strengthens cell walls and, particularly, the  
526 outer membrane of epidermal cells, increasing resistance to the penetration of pathogen  
527 germination tubes in leaves containing large amounts of silicates (shown experimentally for  
528 the penetration of *Erysiphe graminis*, which causes powdery mildew on barley) [113].  
529 Similarly, adding silicon decreases *Fusarium* infections and powdery mildew in cucumber  
530 [114]. All changes in the structure and microclimate of the soil, the date, method and depth of  
531 stubble incorporation also influence the extent and dynamics of crop residue decomposition  
532 [105], which may in turn affect head blight levels. Indeed, in optimal conditions of  
533 microorganism colonisation and water and nutrient exchange, the area of contact between the  
534 soil and crop residues (affected by the size of the residues and soil porosity) is maximal,  
535 accelerating residue decomposition [105]. Consequently, despite providing a larger source of  
536 inoculum, stubble incorporation increases the rate of degradation of crop residues, decreasing  
537 the duration of colonisation of these residues by pathogens such as *Pseudocercospora*  
538 *herpotrichoides* [110]. These pathogens are thus less well developed, resulting in lower attack  
539 rates, which in turn results in slower spore maturation following changes in humidity, light  
540 levels or temperature, or lower levels of spore production [111]. Indeed, it is possible that the

541 decomposition of stubble results in the production of substances toxic to fungi or that favour  
542 the microorganisms engaged in stubble decomposition, thereby creating competition between  
543 microorganisms [111], limiting the production of spores. Finally, a study of *Fusarium* foot rot  
544 showed that the distribution of crop residues is the primary factor influencing the site of  
545 penetration of the fungi [115].

546

### 547 **2-3. Effects of irrigation**

548 The irrigation of a field influences its microclimate and may encourage the development of  
549 the pathogen. Regardless of whether the climate is favourable for the disease in a given year,  
550 irrigation increases the frequency (% necrotic ears) and severity (% necrotic spikelets) of the  
551 disease over that in non-irrigated plots [19,90].

552

553 Uncertainties concerning the survival of the fungus in the soil and its capacity to sporulate in  
554 the soil as a function of soil aeration, porosity and light levels, together with the role played  
555 by the rate of residue degradation and that of the compounds generated by this process, and  
556 their mineral composition (nitrogen, silicon) limit our ability to make quantitative predictions  
557 of the effects of the preceding crop, soil tillage or irrigation.

558

### 559 **2-4. Mineral nutrition**

560 The effects of mineral nutrition on head blight attacks are unclear. As nitrogen applications  
561 favour the development of the plant, making the canopy more humid, it has often been  
562 suggested that such applications are likely to favour the appearance of diseases [103,116].  
563 This has been demonstrated for bunt [103] and blotch or *Septoria* disease [117]. In fact, the  
564 severity of *Septoria* attacks on wheat is increased by 11%, which corresponds to a yield loss  
565 of 8-9%, following treatment with 100 kg of nitrogen per hectare [117]. Nitrogen application  
566 significantly increases the incidence of *Fusarium* infection in grains of wheat, barley and  
567 triticale [118,119] and the predisposition of wheat to attacks by *F. avenaceum* and *M. nivale*  
568 [116]. Similarly, mineral nitrogen applications increase the number of *F. culmorum*  
569 propagules [108]. However, in a two-year study, Teich (1989)[90] showed that nitrogen  
570 application limited the disease in the first year [99], although this decrease was not confirmed  
571 in the second year [100]. Fauzi and Paulitz (1994)[120] also demonstrated that disease levels  
572 were similar in the presence and absence of applications of 140 kg/ha ammonium nitrate.

573

574 There are two possible reasons for the uncertainties concerning the effect of nitrogen  
575 applications on the disease. Firstly, in certain cases, the applied nitrogen may be poorly  
576 distributed in the soil. *Fusarium* foot rot levels have been shown to decrease if ammoniacal  
577 nitrogen is applied with a syringe to the tilled layer but not if it is mechanically applied to the  
578 field [121]. Secondly, there may be an as yet unidentified link between attacks of *Fusarium*  
579 foot rot and head blight [6]. Thus, nitrogen-containing fertilisers may have a different effect  
580 on the *Fusarium* head blight and thus affect the production of head blight inoculum. This link  
581 may involve competition between the species of *Fusarium* more specific to foot rot (such as  
582 *F. solani*) and those more specific to head blight (such as the species of the *F. roseum* group).  
583 Indeed, whereas germination of the conidia of *F. solani* is decreased by nitrate application,  
584 spores of the species of the *F. roseum* group use all forms of nitrogen [122].

585

586 Different forms of nitrogen have different effects. Urea appears to reduce head blight levels  
587 more than ammonium [90,123]. Teich (1989)[90] suggested three hypotheses to account for  
588 this difference between urea and ammonium: i) the urea may decrease the size of the  
589 *Fusarium* population because nitrite blocks reproduction in these fungi and inhibits the  
590 formation of chlamydospores [124], ii) urea may prevent the maturation of *Fusarium*  
591 ascospores, as it does for those of *Venturia inequalis* [125], iii) urea may increase the number  
592 of actinomycetes in the soil [122,126], these microbes being antagonists of *Fusarium*  
593 *graminearum* [126] and *F. oxysporum* [122]. Furthermore, rotting of the base of the stem and  
594 of the roots is more severe if nitrogen is applied in an ammoniacal form than as nitrate  
595 [121,122,127]. This may also result from the higher rate of germination of *F. solani* (which  
596 may cause foot rot) in the presence of ammoniacal nitrogen than in the presence of nitrate  
597 [122].

598

599 In contrast to what was observed for bunt [103], the application of phosphorus has been  
600 shown to limit foot rot [128] and head blight [99]. However, the result obtained for head  
601 blight was not confirmed in the second year of the study [100].

602

603 Potassium application decreases transpiration by causing stomatal closure, thereby helping to  
604 decrease the humidity of the air. Thus, the application of potassium limits the germination of  
605 rust spores [103]. Furthermore, high levels of potassium favour the synthesis of high-  
606 molecular weight compounds (such as cellulose), decreasing the availability of nutrients to

607 the pathogens and increasing mechanical resistance to parasite penetration [103]. However,  
608 potassium applications do not appear to affect the incidence of head blight [100].

609

610 To understand the meaning of these contradictory results, we need to bear in mind that the  
611 effect of fertiliser applications depends on two factors: the age of the plant and the balance of  
612 mineral elements. Indeed, foot rot levels are decreased by early applications of nitrate but  
613 increased by late nitrate applications [128]. Conversely, foot rot levels are increased by early  
614 potassium applications and decreased by late potassium applications [128]. Moreover, the  
615 application of manures with a poor nutrient balance leads to more severe foot rot symptoms  
616 [14,64]. Antagonism between nitrogen and potassium is generally reported [14,103]: excess  
617 potassium and nitrogen deficiency render the plant more resistant whereas potassium  
618 deficiency and excess nitrogen render it more susceptible. This antagonism results from the  
619 fact that potassium increases nitrate absorption [122]. The nitrate absorbed activates nitrate  
620 reductase, leading to the accumulation of amino acids [127]. These amino acids are used in  
621 the synthesis of organic nitrogenous compounds [122] and of high-molecular weight  
622 compounds [103]. These compounds, like cellulose, contribute to the resistance of plants to  
623 diseases by increasing mechanical resistance to parasite penetration and by decreasing the  
624 quantities of soluble amino acids and low-molecular weight carbohydrates, which serve as  
625 nutrients for these pathogens [103]. Soluble amino acids and low-molecular weight  
626 carbohydrates are produced by increasing the activation of nitrate reductase in the presence of  
627 excess nitrate, by activating enzymes such as amylase, proteases and glucosidases, and by  
628 decreasing phosphorylation in conditions of potassium deficiency [103]. This increases the  
629 intracellular concentration of these compounds [103], favouring the development of  
630 pathogens.

631

632 Maize plants are most resistant to diseases when sugar concentrations are high [129]. Sugar  
633 levels are inversely proportional to nitrogen supply and directly proportional to potassium  
634 supply [14]. Conversely, if we consider three, rather than two, mineral elements, the mineral  
635 balance least favourable for the development of foot rot in wheat, both in seedlings and in  
636 adult plants, is high levels of nitrogen and potassium and low levels of phosphorus [128].

637

638 Several studies have demonstrated the existence of a relationship between the mineral and  
639 organic composition of the soil and the development of soil microorganisms, which may  
640 antagonise or stimulate the development of *Fusarium*. Thus, applications of organic fertiliser

641 rich in nitrogen, particularly the ammoniacal and nitrous acid forms, significantly reduce (by  
642 a factor of 1000) the size of the populations of many soil pathogens and markedly increase the  
643 size of populations of soil microorganisms [123]. Furthermore, a mixture of ammoniacal  
644 nitrogen and ammonium is recommended to increase the capacities of *Pseudomonas*  
645 *fluorescens* populations, thereby favouring the development of the plant (as this bacterium is a  
646 rhizobial bacterium) and inhibiting the development of *Fusarium* [130]. An application of  
647 glucose (simulating root exudates) to soils containing chlamydospores of *F. oxysporum* and *F.*  
648 *solani* favours the germination of these spores and the development of their germination tubes  
649 [131]. A similar result was also reported in a previous study: wheat root exudates increase the  
650 number and length of germination tubes arising from the spores of *F. roseum* and *M. nivale*  
651 [98]. The effect of glucose application is even greater if the soil is nutrient-poor, especially for  
652 slow-growing species such as those of the genus *Fusarium* [132].

653

654 The mineral nutrition of plants and the development of microorganisms in the soil also  
655 depend on soil pH. The conidia of *M. nivale* do not germinate in culture *in vitro* if the pH of  
656 the medium is lower than about 5 [11]. However, pH has been found not to affect the  
657 incidence of the disease [99,100].

658

## 659 **2-5. The effect of sowing date**

660 Sowing date is another element of crop management that has an indirect effect on the  
661 production of, and infection by spores, because it partly determines flowering date, together  
662 with the variety sown and climate. If the sowing date is such that flowering coincides with  
663 spore release, then more frequent and severe attacks are likely. This hypothesis is based on  
664 the fact that the anthesis is the stage in wheat at which the consequences of pathogen attack  
665 are thought to be the most severe [12,16,86,87].

666 The effect of sowing date should therefore be considered with respect to the level of attack as  
667 a function of the earliness of the variety. Early-maturing wheat cultivars tend to be more  
668 resistant to head blight than cultivars that mature later [133]. The duration of the growth  
669 period in wheat has a significant positive effect: the longer the cycle of the variety, the lower  
670 the degree of contamination observed [134]. As this effect is not observed in barley and oats,  
671 Couture [134] suggested that the threshold number of days exceeds the length of the cycle for  
672 wheat, but not for barley and oats. However, conflicting results were obtained in other  
673 studies: resistance to head blight has been shown to be independent of maturity factors [135],  
674 and late sowing has been shown to favour the development of head blight whereas early

675 sowing favoured the development of foot rot [16]. The results obtained almost certainly  
676 depend on the climate of the region concerned and the variety grown. Flowering period and  
677 the duration of the growth period for a given variety probably depend essentially on the year  
678 and region concerned: the only valid way to determine the characteristics (period and duration  
679 of flowering and growth duration) of varieties adapted to a given region is to carry out a  
680 frequency analysis of the climate, which requires the monitoring of head blight attacks in a  
681 given region over many years. Sowing on several dates (to extend the flowering period)  
682 should make it possible to limit the risk of heavy contamination in all the fields. A frequency  
683 analysis in a given region may also provide useful information for the optimal choice of  
684 sowing date.

685

686

## 687 **2-6. Effects of canopy density**

688 If the inoculum is dispersed primarily by splashing, the density of the canopy is an important  
689 factor as it may place obstacles in the way of this dispersal. Thus, a low canopy density can  
690 favour spore dispersal (by creating fewer obstacles). In the case of high canopy density, due  
691 to nitrogen fertilisation and/or higher sowing density and/or smaller spaces between rows of  
692 wheat, two opposing effects may be observed. Firstly, the high density is likely to increase the  
693 humidity of the canopy [116], favouring spore germination, as has been observed for rusts  
694 [103]. Alternatively, the high density of the canopy may increase the number of obstacles,  
695 limiting the vertical dispersal of spores towards the ear. However, for *Fusarium* infections,  
696 more severe attacks of foot rot [16] and head blight [5] have been reported in cases of high  
697 population density: more than 350 grains sown per m<sup>2</sup> [16].

698

699 Plant population density may also be increased by weed infestations. In the presence of a  
700 large weed population, the number of species of *Fusarium* causing head blight in wheat  
701 increases, as does the intensity of the disease [1]. This may be accounted for by the number of  
702 common field weeds (both dicotyledonous and monocotyledonous weeds) on which  
703 numerous species of *Fusarium* have been detected [62]. Table 2 lists the various weeds  
704 known to serve as hosts for *Fusarium*, and the species of *Fusarium* found on them.

705

706 The "host-plant" nature of these weeds [14,48,62,98] suggests that they constitute a potential  
707 source of inoculum. Since 1950, grass weeds have been suspected to act as a source of

708 inoculum whereas other weeds have been thought to favour infection in a different way, by  
709 modifying the microclimate, increasing heat and humidity [136].

710

711 Whatever the role played by the various weed families, the decrease in the incidence of the  
712 disease following the use of herbicides suggests that weeds do have an effect [99]. However,  
713 different results were obtained in the following year of the same experiment [100]. This  
714 difference probably results from interactions between cultural practices and weeds. For  
715 example, the withering of forage grasses (fescue, cocksfoot, ryegrass) due to *F. roseum*  
716 increases with the dose of nitrogen applied [14].

717

## 718 **2-7. Effects of infestation of the canopy by bioaggressors others than fungi responsible** 719 **for the head blight**

720 The term "canopy" is often used to describe the green canopy, consisting of healthy plants.  
721 Diseases, particularly foliar diseases, decrease the area of the plants capturing light, thereby  
722 limiting the development of the crop. Very few studies have focused on the competition  
723 between pathogens or between diseases. Only two authors have analysed the effect of a  
724 powdery mildew attack on head blight, and the results of these two studies conflicted. In the  
725 first study, powdery mildew attacks were found to have a positive effect on head blight  
726 attacks [30] whereas in the second, no effect was observed [99]. These conflicting results may  
727 be accounted for by several factors, such as climatic conditions, the varieties cultivated and  
728 the intensity of the diseases in these studies.

729

730 In addition to soil microorganisms (discussed in section 2-2), the ear microorganisms play a  
731 role, notably in biological control [137]. Indeed, the plant may be protected against pathogen  
732 attacks by prior inoculation with pathogen isolates of various degrees of virulence, which is  
733 known to induce resistance in cases in which protection requires activation of the host's  
734 defence responses [138]. Thus, inoculation with *Microsphaeropsis* spp. significantly  
735 decreases the production of *F. graminearum* ascospores on wheat and maize ears and, to a  
736 lesser extent, on wheat and maize residues [137,139]. This decrease is particularly large if the  
737 residues are inoculated early with *Microsphaeropsis* (test with inoculation two weeks before  
738 the *F. graminearum* attack and 4 to 6 weeks after it) [137]. Prior inoculation with *Phoma*  
739 *betae* or *Pythium ultimum* decreases the severity of head blight on wheat and extends the  
740 incubation and latent periods of the disease due to *F. culmorum*, *F. avenaceum*, *F. poae* and  
741 *M. nivale* [138]. Such prior inoculation also significantly increases the number of grains per

742 ear and decreases the number of *Fusarium* isolates obtained from the harvested grains [138].  
743 A previous study [140] showed that contact between *Pythium oligandrum* and *F. culmorum*  
744 macroconidia led to coagulation and a loss of cytoplasm, followed by complete degradation of  
745 the walls of the macroconidia and the production of *Pythium oligandrum* oogonia in  
746 abundance on the parasitised macroconidia. Inoculation of wheat ears with *Alternaria*  
747 *alternata*, *Botrytis cinerea* or *Cladosporium herbarum* at GS69 (Zadoks growth scale, Zadoks  
748 *et al.*,1974[141]) favours the infection of ears inoculated with *F. culmorum* at GS65, whereas  
749 prior inoculation, at GS59, decreases infection of the ears by *F. culmorum* [142]. Following  
750 the demonstration of antagonism between these saprophytes, Liggitt *et al.* [142] suggested  
751 that *Alternaria alternata*, *Botrytis cinerea* and *Cladosporium herbarum* produce volatile  
752 antibiotic substances that limit the growth of *F. culmorum*, consistent with the decrease in size  
753 of *F. culmorum* colonies observed following prior inoculation with other saprophytes.

754

755 Such antagonism is also observed between *Fusarium* species [22,27] and between species of  
756 *Fusarium* and *Microdochium* [22]. This is the case, in particular, for fungicide treatments  
757 acting against only one of these genera, resulting in the development of the unaffected genus  
758 to a greater extent than in the absence of treatment, when the two genera compete [21,22].  
759 Similarly, prior inoculation with germination fluid from *F. avenaceum* predisposes wheat ears  
760 to infection by conidia of *F. avenaceum* or *M. nivale*, but not by conidia of *F. culmorum*, or *F.*  
761 *poae*. Other germination fluids were found to have no such effect [138]. This suggests that  
762 diffuse substances present in the germination fluid of *F. avenaceum* promote the production in  
763 competitive host tissues of resistance factors specific for *F. avenaceum* and *M. nivale* [138].

764

## 765 **2-8. Effects of chemical treatments**

766 The efficacy of fungicide treatments for head blight is variable, and difficult to predict. It  
767 depends not only on the active ingredient but also on the method and date of application of  
768 the fungicide [6,27]. If a fungicide treatment is to be effective, it must be applied several days  
769 before the attack and entirely cover all the ears [142]. Efficacy also depends on the interaction  
770 between fungal development and climate [143], the virulence of the pathogenic strains [143]  
771 and above all, the parasite complex present on the plot of land concerned [6]. Each of the  
772 fungal species implicated in head blight has a specific fungicide susceptibility profile: *F.*  
773 *graminearum* is particularly susceptible to triazoles whereas *F. avenaceum* is more  
774 susceptible to strobilurins. *F. roseum*, a complex consisting of *F. graminearum*, *F. culmorum*  
775 and *F. avenaceum*, is much more susceptible to triazoles than is *M. nivale*, which is



776 essentially susceptible to strobilurins [22]. Triazole fungicides containing tebuconazole are  
777 currently the most effective [21,27,143].

778

779 Chemical treatments may have an effect by modifying the height of the canopy. For a single  
780 variety grown in a single year, in a single region, the use of plant growth regulators (primarily  
781 gibberellin inhibitors) results in more severe head blight attacks [14,64,118,120]. This is  
782 presumably because plant growth regulator treatment results in the ears being closer to the  
783 soil, and therefore to the crop residues (source of inoculum), facilitating the dispersal of the  
784 spores to the ear (by splashing in particular). Indeed, the distance of the ear from the soil is a  
785 factor for resistance by avoidance in cultivars. Plant growth regulators also affect the  
786 microclimate of the crop residues on the soil [120], possibly increasing the production of  
787 perithecia and ascospores.

788

### 789 **3- Effects of various cultural practices on the production of mycotoxins by** 790 ***Fusarium***

791 The effects of cultural practices on the intensity and severity of head blight symptoms are  
792 well documented, at least for certain practices, but far fewer studies have considered the  
793 effects of these practices on mycotoxin production.

794

#### 795 **3-1. Mycotoxins produced by *Fusarium***

796 Fungi of the genus *Microdochium* are currently considered not to produce toxins [8] and will  
797 therefore not be dealt with in this section. It should also be noted that studies on the  
798 mycotoxins produced by *Fusarium* have generally focused on deoxynivalenol (DON), even  
799 though *Fusarium* also produces other types of mycotoxin. Figure 2 presents the chemical  
800 structures of the main mycotoxins produced by *Fusarium*.

801

802 Deoxynivalenol belongs to the trichothecene family, which contains two types of toxin: type  
803 A and type B. Type A toxins include toxins T2, HT2, diacetoxyscirpenol (DAS) and  
804 scirpenol [39], which act on the initiation of protein elongation [144]. Type B corresponds to  
805 deoxynivalenol (DON or vomitoxin) and its acetyl derivatives — 3-acetyldeoxynivalenol  
806 (3acDON) and 14-O-acetylDON-4 — nivalenol (NIV) and verrucarol [39]. The toxins of this  
807 group act on the elongation and termination steps of protein synthesis [144]. Trichothecenes  
808 are toxic to all organisms. In mammals, the absorption of trichothecenes leads to blood and

809 digestive disorders. In humans [145], burning sensations in the mouth and stomach,  
810 headaches, a decrease in red blood cell count, bleeding, necrosis of the throat and stomach  
811 and, in some cases, death, were observed in a humanitarian catastrophe in the USSR between  
812 1942 and 1947 caused by the ingestion of wheat that had suffered foot rot all winter [38,39].  
813 The production of trichothecenes is favoured by cold and humidity [146] and studies on  
814 various organisms have demonstrated the toxicity of these substances. In animals, vomiting is  
815 observed after the ingestion and absorption of at least 10 mg deoxynivalenol per kg, together  
816 with irritation of the mucous membrane lining the mouth and the oesophagus, with animals  
817 refusing to eat after ingesting 2 mg deoxynivalenol per kg.

818  
819 In plants, deoxynivalenol retards the germination and growth of wheat, and inhibits the  
820 growth of the grain and the coleoptile tissues [147]. The phytotoxicity of mycotoxins is  
821 generally estimated by means of coleoptile elongation tests [43,145]. In adult plants,  
822 deoxynivalenol seems to circulate in the phloem, with the concentration of this molecule in  
823 the plant following a descending gradient from the rachis, through lemmas and grains to the  
824 peduncle [148]. In addition, from the fourth day after inoculation, the flower parts, rachis and  
825 peduncle contain larger amounts of deoxynivalenol below the point of infection than above it  
826 [149].

827  
828 *Fusarium* can also produce mycotoxins of two other families: fumonisins, which are  
829 recognised carcinogens [13,150] and zearalenone. Zearalenone belongs to the oestrogen  
830 family. It perturbs reproductive hormone equilibrium and alters secondary sexual characters  
831 [145]. Pigs are particularly sensitive to this toxin [145]. In the laboratory, it has been shown  
832 that this compound also controls reproduction in the fungus that produces it, by regulating  
833 perithecium production [151-153]. No phytotoxic effect of zearalenone has been observed in  
834 coleoptile elongation tests [145].

835  
836 To account for the small number of studies dealing with the mycotoxins produced by  
837 *Fusarium* on wheat, it should be borne in mind that most of these studies have been carried  
838 out in Europe and North America, where type A trichothecenes are rarely detected [154].  
839 Furthermore, fumonisins and zearalenone are found essentially on maize [8,155]. In addition,  
840 zearalenone is considered to be only mildly toxic to humans [155].

841

842 **3-2. Effect of soil tillage**

843 Soil tillage seems to have similar effects on the production of mycotoxins and on the disease  
844 itself (see part 2-1). Unlike chisel ploughing and direct drilling systems, mouldboard  
845 ploughing decreases the concentration of deoxynivalenol in the grains [19,101,156]. In one  
846 study, a significant positive correlation between deoxynivalenol concentration and the  
847 intensity of soil tillage (ploughing or direct drilling) after a maize crop was observed [100].  
848 Similarly, another study comparing three cropping systems involving ploughing (productive,  
849 integrated and organic) with a direct drilling system over two years reported that levels of  
850 deoxynivalenol, zearalenone and nivalenol contamination were highest for the direct drilling  
851 system [55].

852

853 **3-3. Effect of the preceding crop**

854 The effect of the preceding crop is also similar for disease symptoms and deoxynivalenol  
855 concentration. Deoxynivalenol concentration in wheat is 25% lower after a soybean crop than  
856 after a wheat crop and 49% lower after soybean than after maize [19]. Wheat following maize  
857 in the rotation has been found to have deoxynivalenol concentrations six times higher than  
858 those in wheat following another cereal (wheat, barley) or soybean [100]. In direct drilling  
859 systems, wheat crops following an oilseed rape crop contain 90% less deoxynivalenol than  
860 wheat crops following a maize crop [101]. As for soil tillage, only few studies have produced  
861 useful results.

862

863 **3-4. Effect of mineral nutrition**

864 We saw in section 2-3 that the effects of mineral nutrition on the disease are unclear. In  
865 contrast, there are fewer strong contradictions concerning the effect of mineral nutrition on  
866 mycotoxin production. This is probably because very few studies have focused on this  
867 question. Increases in nitrogen input from 0 to 80 kg/ha, result in increases in grain  
868 deoxynivalenol content; at levels above 80 kg/ha, a small but significant decrease was  
869 observed [119]. Urea-based fertilisation resulted in lower levels of deoxynivalenol in wheat  
870 grains than did ammonium nitrate in 1997, but this result was not observed the preceding  
871 year, in which no effect had been demonstrated [156]. Applications of ammonium-nitrate-  
872 urea solution and nitramoncal did not give significantly different results [119]. Potassium,  
873 phosphorus and pH did not appear to affect deoxynivalenol concentration significantly [100].  
874 A laboratory study showed that  $Mg^{2+}$  ions inhibit the elements responsible for inducing  
875 trichothecene synthesis [38].

876

877 These studies were empirical, and were not based on any particular assumption concerning  
878 the mechanisms involved. It therefore remains difficult to extrapolate the results obtained.

879

### 880 **3-5. Effect of variety earliness**

881 Very few studies have focused on the effect of wheat variety earliness on the production of  
882 mycotoxins. The earliest varieties seem to accumulate more deoxynivalenol than do late-  
883 flowering varieties [157], possibly due to varietal differences but more probably due to  
884 greater coincidence of the phase of maximum susceptibility in plants with the period most  
885 favourable for spore dispersion. This trend appears to be based on the relationship between  
886 the severity of the attack and the intensity of toxin contamination. The choice of variety is  
887 therefore of key importance for combating the accumulation of toxins in the grains [158].

888

### 889 **3-6. Effect of fungicide treatments**

890 The application of a triazole fungicide, such as tebuconazole, decreases levels of  
891 deoxynivalenol contamination in artificially inoculated crops with respect to those observed  
892 in untreated controls [21,27,28,143]. However, conflicting results have been obtained [22].  
893 More generally, in conditions of natural contamination, the application of fungicides does not  
894 significantly decrease the concentrations of deoxynivalenol and nivalenol and there is not  
895 necessarily a correlation between the application of fungicides and the quantity of  
896 deoxynivalenol and/or nivalenol found in the grains [6,55]. This may be accounted for by the  
897 multitude of species present in fields and the relative specificity of fungicide treatments for  
898 only one or a few species. Another hypothesis has also been put forward: too low a dose of  
899 fungicide may stimulate the production of deoxynivalenol by *Fusarium* in wheat grains [53].

900

901 This analysis shows not only that few studies have investigated the effects of cropping  
902 systems, but also that the characterisation of the disease in these studies is often severely  
903 flawed. This makes it difficult to extrapolate and to interpret the results. To understand the  
904 relationship between cropping systems and mycotoxin production, we therefore need to break  
905 down this relationship into two parts: the effects of cropping systems on the disease  
906 (symptoms, nature of the pathogenic agent) (section 2) and the relationship between the  
907 disease and the production of mycotoxins, which may itself vary as a function of cropping  
908 system.

909

910

## 911 **4- Relationship between the disease and toxin production**

912

### 913 **4-1. Toxin production by the fungi**

914 Toxin production in fungi is controlled by many factors. One of the most important factors  
915 intrinsic to the fungus is the genetic capacity of the pathogenic strain to produce toxins, in  
916 terms of the quantity and type of toxins produced [34,159,160,161]. Thus, strains of *F.*  
917 *graminearum* may, like *F. culmorum*, produce isomers of monoacetyldeoxynivalenol or  
918 alternatively, like *F. crookwellense*, may produce acetyl derivatives of nivalenol [4]. Strains  
919 of *F. sambucinum* may produce the T2 toxin whereas those of *F. sporotrichioides* produce  
920 diacetoxyscirpenol (DAS) [4]. It should be noted that strains of *F. graminearum* [60,159,162]  
921 and of *F. culmorum* [163,164] tend to produce either deoxynivalenol or nivalenol, but not  
922 both. Thus, the two toxins are generally not produced by the same strains, although the two  
923 types of strain often coexist in the same field [165]. Moreover, regionalisation of the types of  
924 strain at world level is observed for *F. graminearum* strains producing 3-acetyldeoxynivalenol  
925 and those producing 15-acetyldeoxynivalenol, with strains producing 3-acetyldeoxynivalenol  
926 predominating in Europe, China, Australia and New Zealand whereas those producing 15-  
927 acetyldeoxynivalenol predominate in the United States [159]. Data for a collection of 188  
928 strains of *F. graminearum* indicated that European and American strains produce essentially  
929 deoxynivalenol and are more aggressive than Nepalese strains, which produce either  
930 deoxynivalenol or nivalenol [166]. A negative correlation has been found between the  
931 production of zearalenone and that of trichothecenes (deoxynivalenol or nivalenol) by the  
932 spores of *F. graminearum* [52]. This appears to indicate that the production of these toxins  
933 shares a common control process, which may play an important role in the ecology of *F.*  
934 *graminearum* and *F. pseudograminearum* [52].

935

936 Other factors may also cause variations in toxin formation. These factors include the  
937 substrate, the period of colonisation of the substrate by the strain, competition between  
938 microorganisms [2,34,52,53], interaction between the pathogenicity of the strains and the  
939 wheat variety [167,168] overall climatic conditions [51] and, more particularly, temperature  
940 [2,52-54], humidity [2,52,53] and rainfall [54].

941

942 Other poorly understood factors extrinsic to the fungus also operate. For example, the protein  
943 to carbohydrate ratio, which is important in mycotoxin synthesis [169], may play a role.  
944 However, Bakan (1998)[38] showed that toxin formation is not necessarily controlled by food  
945 stresses and is not necessarily linked to the growth of the fungus.

946

947 This last point is particularly important because many authors have suggested that the  
948 trichothecenes (deoxynivalenol in particular) produced by *Fusarium* species play a role in the  
949 aggressiveness or virulence of certain isolates [164,170-174]. Indeed, certain mutants  
950 deficient in deoxynivalenol are less aggressive on wheat than the original strain [175].  
951 Similarly, the least virulent mutants of *F. graminearum* in terms of plant colonisation are  
952 those unable to produce deoxynivalenol or nivalenol (mutations in the Tri5 gene, involved in  
953 trichothecene production) [171]. Thus, trichothecenes are considered to be virulence factors in  
954 *F. graminearum*, during the infection of wheat [145,175], and in *F. culmorum*, during the  
955 infection of barley [164].

956

957 In conditions of artificial contamination with a given pathogenic strain or in natural  
958 contamination conditions, several authors [19,27,90,100,147,164,176,177] have observed a  
959 significant, positive correlation between the incidence and/or severity of the disease and  
960 deoxynivalenol concentration (also reported by Bai *et al.*, 2001[49] for 116 wheat lines).  
961 Others [26,49,178,179] have demonstrated a significant correlation between deoxynivalenol  
962 concentration and the fungal biomass of the grains (measured by assessing the amount of  
963 ergosterol). These results suggest that new cultivars could be selected on the basis of  
964 symptoms to ensure low levels of deoxynivalenol. However, exceptions were found among  
965 these lines: certain cultivars present severe symptoms with low deoxynivalenol levels,  
966 particularly those with moderate resistance to propagation of the pathogenic agent [49]. Thus,  
967 the production of deoxynivalenol is not essential for the infection of grains [164].  
968 Furthermore, after the inoculation of five wheat varieties with a strain of *F. graminearum*, no  
969 correlation was observed between deoxynivalenol concentration and the severity of infection  
970 [172]. Similarly, after inoculation with a complex of species, no correlation was observed  
971 between deoxynivalenol concentration and infection on wheat, barley or oats [178]. In  
972 conditions of natural contamination, no correlation has been found between the intensity of  
973 the disease on wheat and the concentration of deoxynivalenol [53,55,78], or between the  
974 presence of *Fusarium* and the concentration of deoxynivalenol [154]. This lack of relationship  
975 is also observed with other toxins, such as nivalenol [55]. A similar result was obtained with

976 maize: after inoculation of the ear with *F. poae*, no relationship was found between the  
977 severity of the disease and toxin (nivalenol and fusarenon X) production [180]. These  
978 differences in results may be due to the ranges of variation tested and the comparisons made  
979 not being the same. Some comparisons were made in the field and others in the laboratory;  
980 some were made after artificial inoculation with one or several species and others were made  
981 in natural contamination conditions.

982

983 The date of the attack and the species involved also affects the relationship between  
984 symptoms and toxin levels. Indeed, it is only to be expected that as the number of affected  
985 grains increases, so does the risk of major contamination by mycotoxins. However, a late  
986 attack may prevent the disease from reaching a high level of severity, even if very high levels  
987 of mycotoxins are produced. Thus, fungi and mycotoxins are detected before any visual  
988 symptoms of disease [148,181]. In addition, not all the species of the parasite complex  
989 responsible for head blight produce mycotoxins: *Microdochium*, for example, does not  
990 produce mycotoxins. Similarly, even among *Fusarium* species potentially capable of  
991 producing toxins, strains unable to produce toxins have been identified [2,38,162,182]. Other  
992 strains have been identified that produce far more toxin than the mean amount for all strains  
993 [183]. In addition to climate (particularly rainfall levels and temperature before and at  
994 flowering), the species profile of the fungi responsible for head blight also depends on  
995 agronomic factors such as soil tillage, nitrogen fertilisation, fungicide use, crop rotation and  
996 host genotypes [10]. Furthermore, the species of *Fusarium* potentially able to produce toxins  
997 cannot necessarily produce trichothecenes (the most studied mycotoxins) [183]. These  
998 observations indicate that it is possible for the plant to present symptoms in the absence of  
999 toxin production. However, the conditions of toxin productions in the field remain mostly  
1000 unknown.

1001

1002 The relationship between the intensity or severity of symptoms and toxin production also  
1003 depends heavily on the type of resistance carried by the plant. The severity of symptoms is  
1004 known to depend heavily on plant resistance: certain genotypes limit the development of the  
1005 mycelium in the grain, protecting the grain against degradation and limiting the visual signs  
1006 of attack, but are not very tolerant of mycotoxins, with very high mycotoxin levels being  
1007 recorded [49]. Conversely, other types of cultivar may present severe symptoms with only  
1008 low mycotoxin levels [49].

1009

1010 **4-2. Different mechanisms of genetic resistance to *Fusarium* head blight in plants**

1011 *Non-specific resistance*

1012 It is not easy to identify cultivar resistances because the species of *Fusarium* responsible for  
1013 head blight are saprophytes with a broad host-range and low levels of intraspecific variation,  
1014 which is not the case for pathogens in general [6,18]. It should also be noted that all cultivars  
1015 are susceptible to some extent, with even the least susceptible cultivars displaying only  
1016 moderate resistance [16,49,50]. Resistance to the development of the fungus and to the  
1017 accumulation of trichothecenes is probably controlled by different genes [43].

1018

1019 *Heritability of the resistance*

1020 The low specificity of *Fusarium* and *Microdochium* generates polygenic resistance in the  
1021 plant, involving genes with various degrees of dominance [43,184]. It is the combination of  
1022 these genes, controlled by the environment [176] that results in genetic resistance in the plant  
1023 [13].

1024

1025 The inheritance of resistance can be described by a dominance-additive effect model, in  
1026 which the additive effect is the essential factor (results based on diallel analyses) [9,25,185].  
1027 Several studies [18,25,186-188] have demonstrated the existence of resistance genes on 18 of  
1028 the 21 chromosomes (1B, 2A, 3, 3B, 4B, 5A, 6B, 6D, 7A, 7B etc.). Three pools of genes are  
1029 responsible for three different genetic constitutions conferring different types of resistance to  
1030 head blight in wheat: in Eastern European winter wheats, in Japanese and Chinese spring  
1031 wheats and in Brazilian and Italian spring wheats [189]. The most commonly used source of  
1032 resistance is undoubtedly Sumai 3 (in China), in which stable resistance is combined with  
1033 valuable agronomic characteristics (yield potential and resistance to rusts and powdery  
1034 mildew) [18,43,190]. The resistance of this genotype, like that of Frontana (a Brazilian  
1035 variety) and Nobeokabouzu-komugi (a Japanese variety) — the two other major sources of  
1036 resistance — is based on two or three genes with additive effects, the sensitivity of which  
1037 varies with a multitude of minor genes [9,18,191]. The crossing of Frontana and Sumai 3  
1038 resulted in the detection of two major quantitative trait loci (QTLs) [13]. These sources of  
1039 resistance are of great potential value for future crosses and the selection of new cultivars  
1040 because they are stable.

1041

1042 Resistance to head blight in wheat is horizontal (non-specific) and thus protects against all  
1043 species of *Fusarium* and *Microdochium* [6,18,43,192].



1044

1045 No correlation has been found between seedling resistance and resistance in the ear. Thus, a  
1046 given cultivar may be susceptible at the seedling stage and resistant at the adult stage or vice  
1047 versa. Alternatively, it may be susceptible throughout the crop cycle [5]. The activation of  
1048 resistance genes during an attack depends on the developmental stage of the wheat, varying  
1049 throughout the crop cycle [5,47,49,50,90]. Peak toxin accumulation also depends on the  
1050 cultivar grown [193]. Finally, it should be noted that the duration of the induction period and  
1051 the intensity of the response to the defence mechanisms induced also determine the defence of  
1052 the plant [43]. Tetraploid wheats are more resistance to head blight than are diploid wheats  
1053 [194].

1054

1055 The defence or resistance mechanisms of the plant depend not only on growth stage, but also  
1056 on the humidity and temperature of the air [47] and on the genetic capacity of the wheat  
1057 cultivar [5,18,49,50,90]. However, no plant host defence reaction has ever been detected  
1058 macroscopically or histologically on the outside of the glumes [12]. In addition, no cellular or  
1059 anatomical characteristics [13,50] or histological features [50] have been found to be  
1060 associated with resistance or susceptibility. However, the techniques currently used, and  
1061 ultrastructural studies in particular, have shown that susceptible and resistant wheat varieties  
1062 react differently to infection and to the propagation of pathogens [94].

1063

1064 This implies the existence of multiple mechanisms of defence or resistance in plants, both  
1065 active (including physiological processes) and passive (including morphological  
1066 characteristics such as avoidance), and/or tolerance [5]. Five types of cultivar resistance are  
1067 currently known and described.

1068

### 1069 *Types of resistance*

1070 **Type I: Resistance to initial infection** [50]. Many cultivars may be resistant to initial  
1071 infection. According to Nakagawa (1955) [195], this type of resistance is controlled by  
1072 dominant genes at three loci subject to epistasis. This type of resistance may be passive or  
1073 active.

1074

1075 *Passive mechanisms* involve morphological characteristics facilitating avoidance, making it  
1076 possible for the plant to decrease the severity of the disease. The receptiveness of cultivars to  
1077 the disease is lower in cultivars with awns [5,14,134], regardless of varietal differences in

1078 resistance [134]. Similar results have been obtained with wheat, barley [134] and oats  
1079 [45,134]. Similarly, the height of the ear and its angle with respect to the stem are negatively  
1080 correlated with the severity of head blight and the accumulation of deoxynivalenol [191]. The  
1081 receptiveness of cultivars to *Fusarium* and/or *Microdochium* is also lower for ears with a  
1082 large peduncle — at least 15 cm between the flag leaf and the ear [5] — and/or without  
1083 growth arrest, reducing the area in contact with the conidia and the duration of grain humidity  
1084 [5].

1085

1086 A cultivar well adapted to its environment is also a criterion for resistance to head blight  
1087 [18,43]. Indeed, a canopy with too many ears [5,16] or a flowering season that lasts too long  
1088 [2,90] is likely to result in slightly higher susceptibility to the disease. The resistance of a  
1089 variety may be evaluated as a function of the duration of time for which the flowers remain  
1090 open and of the percentage of flowers presenting autogamous flowering (unopened anthers)  
1091 and allogamous flowering (opened anthers) [50]. Studies have shown either that resistance to  
1092 head blight may be maximised by the use of early varieties [133] or that earliness has no  
1093 importance [135]. However, the importance of this factor seems to depend on the date of the  
1094 attack and, thus, on the year and region studied. Similarly, only one study has investigated the  
1095 effect of flowering date on mycotoxin contamination: the time of infection by *F. culmorum*  
1096 that produces the greatest amount of deoxynivalenol contamination is restricted to a short  
1097 period during anthesis [87]. However, like disease, flowering period and variety growth  
1098 duration probably depend essentially on the year and region concerned: the only valid way to  
1099 determine the characteristics (period and duration of flowering and growth duration) of  
1100 varieties adapted to a given region is to carry out frequency analysis of the climate, which  
1101 requires the monitoring of mycotoxin contamination in a given region over many years.

1102

1103 It is widely accepted that morphological characteristics are less important than the possible  
1104 physiological resistance of cultivars [9]. This physiological resistance includes all the other  
1105 possible mechanisms. *Active mechanisms* include defence reactions concerning the  
1106 physiological qualities of the cell that limit colonisation of the plant by the fungus, such as the  
1107 activation of enzymes degrading the fungal cell wall. Such a mechanism has been proposed  
1108 for pathogenesis-related proteins (PR proteins) and hordothionins [196]. Mechanisms of this  
1109 type may account for the induction of several defence genes 48 hours after inoculation,  
1110 including genes encoding chitinases, glucanases, peroxidases and thaumatin-like proteins  
1111 (TLP) [197,198].

1112

1113 The endo- and exochitinases in plant cell walls help to improve plant resistance by  
1114 hydrolysing chitin, a protective polysaccharide present in the cell walls of the pathogens  
1115 [18,199]. The various types of chitinase known do not have the same effects [200]: the class I  
1116 chitinase-a of rice strongly inhibits re-extension of the hyphae and is present in mature cells in  
1117 particular. It collects at the tip of the hyphae, on the lateral walls and in the septa and has a  
1118 very high affinity for fungal cell walls. This enzyme releases a large amount of reducing sugar  
1119 from the fungal cell walls. It also ligates the lateral walls and the septa, is part of the walls of  
1120 mature cells and degrades mature chitin fibres. In contrast, the class II chitinase-c of rice is  
1121 found essentially at the tip of the hyphae, which it ligates before degrading young chitin  
1122 fibres. Thus, chitinase-a is more effective than chitinase-c at inhibiting the growth of the  
1123 fungus [200].

1124

1125 The antimicrobial activity of thionins is attributed to their capacity to create pores in the  
1126 membranes of fungi by means of interactions between the phospholipids and  
1127 phosphoinositides of the membrane or by decreasing the activity of enzymes by reducing  
1128 disulphide bonds [199].

1129

1130 Thaumatin-like proteins may have two modes of action: they may be specifically produced in  
1131 response to the presence of pathogenic fungi in a certain number of plants, including wheat  
1132 [201] or they may disturb the signal transduction cascade in the cell, increasing tolerance to  
1133 trichothecenes and favouring the development of fungi in the plant [198]. A trypsin-like  
1134 protease produced by *F. culmorum* may be one of the key enzymes in the colonisation of  
1135 plants by the fungus [202]. Indeed, the presence of this enzyme in barley has been shown to  
1136 be correlated with the degradation of specific buffer proteins in infected grains [203]. The  
1137 alkaline protease produced by *F. culmorum* is also involved in the colonisation of barley and  
1138 wheat grains [204]. Peroxidases (POX) are oxidoreductive enzymes involved in the  
1139 construction of the cell walls of plant hosts (phenol oxidation, suberisation, lignification)  
1140 during defence reactions against pathogenic agents [205]. The concentration of POX increases  
1141 significantly during the milk stage in wheat anthers inoculated with *F. graminearum*, whereas  
1142 this is not the case in healthy wheat [205].

1143

1144 Polyphenol oxidases (PPO) are involved in the oxidation of quinone polyphenols (a microbial  
1145 component) and in the lignification of plant cells during microbial invasion [205]. The

1146 specific activity of PPO is maximal during the milk stage in wheat and declines rapidly  
1147 thereafter [205]. Levels of PPO activity are higher in resistant than in susceptible varieties and  
1148 increase following inoculation with *F. graminearum* [205].

1149

1150 One possible strategies for reducing the risk of mycotoxin contaminations is the development  
1151 of more resistant transgenic crops. Three approaches are possible [206]: (i) transgene-  
1152 mediated control of the ability of *Fusarium* to infect and colonise the ear, through the  
1153 overproduction of specific antifungal proteins and metabolites, or by increasing the plant's  
1154 own defense systems in kernel tissues; (ii) the prevention of mycotoxin biosynthesis, or the  
1155 detoxification of mycotoxins in plants and (iii) the development of more resistant plants based  
1156 on transgenic crops engineered to produce a *Bacillus thuringiensis* (Bt) toxin. Some Bt maize  
1157 hybrids have the potential to reduce the level of fumonisin B produced by *F. verticillioides*  
1158 [206]. This effect probably results from a decrease in sensitivity to one of the possible means  
1159 of ear tissue contamination: attack by insects [206,207]. Indeed, *Bacillus thuringiensis* (BT) is  
1160 known to produce proteins toxic to insects [208]. This strategy has not yet been applied to  
1161 wheat.

1162

1163 Type I resistance is generally estimated by determining the percentage of spikelets infected  
1164 seven to 21 days after inoculation or at maturity [9]. However, the number of infected  
1165 spikelets does not necessarily reflect the total damage caused by the pathogen. The symptoms  
1166 also depend on the severity and rapidity of rachis invasion by the pathogen.

1167

1168 **Type II: resistance to (kinetic) propagation of the pathogenic agent** in the tissues [50].

1169 The mechanisms involved in this type of resistance are purely active, such as inhibition of the  
1170 translocation of deoxynivalenol [18] by an ABC (ATP-binding cassette) transporter protein,  
1171 the Pdr5p (pleiotropic drug resistance) [209] or increasing the stability of cell membranes  
1172 [18]. This process limits the propagation of *Fusarium culmorum* and *F. graminearum* [95]  
1173 from the glumes [18,26] and/or ovaries and glumes to the rachis and the pedicel [94-96]. The  
1174 endo- or exocellular migration [94-96] of the fungus leads to a series of changes in host cells,  
1175 including degeneration of the cytoplasm, organelles (e.g. chloroplasts) and cell wall [95,96]  
1176 by enzymes such as cellulases, xylanases and pectinases [95,96] produced during the  
1177 penetration and colonisation of the tissues of the wheat ear [95,96]. Once attacked, susceptible  
1178 varieties respond to infection only by forming a very thin wall in the periplasmic space of the  
1179 infected tissues [94]. In contrast, resistant varieties react strongly: extensive formation of a

1180 very thick wall located close to the infected cells and of a large papilla formed by the rapid  
1181 and intense deposition of callose ( $\beta$ -1,3-glucan), lignin and other compounds [94]. Three days  
1182 after inoculation, defence structures are more solid, with denser cell walls, particularly in cells  
1183 adjoining contaminated cells, in the envelopes of cultivar Frontana (resistant) than in those of  
1184 cultivar Agent (susceptible) [210]. The process of lignification may be involved in plant  
1185 defences, establishing mechanical barriers to pathogen invasion [94,210], modifying the  
1186 structure of cell walls and making them more resistant to the degradative enzymes produced  
1187 by the pathogen [210]. Furthermore, lignification may inhibit or reduce the diffusion of small  
1188 molecules or ions, including the mycotoxins secreted by the fungal hyphae in the host cells  
1189 [94,210], and reduce the movement of nutrients from the host cell to the pathogen [210]. In  
1190 addition to the lower permeability of the cell membranes in resistant genotypes, the basis of  
1191 the inhibition of deoxynivalenol translocation may also involve a lower affinity of the  
1192 membrane for deoxynivalenol [26]. This hypothesis is based largely on observations in two  
1193 types of mutant yeast [144]. One of these types of mutant yeast presents a low affinity and  
1194 low cell membrane permeability for trichothecenes, and in the other, the 60S ribosomal  
1195 subunit has a lower than normal affinity for these toxins. The target of deoxynivalenol is a  
1196 cytoplasmic peptidyl transferase [211]. Mammalian and fungal cells have a modified peptidyl  
1197 transferase that is tolerant to trichothecenes [211]. In cases of type II resistance, the  
1198 trichothecenes seem to bind to the ribosomal 60S subunit, by methylation, thereby blocking  
1199 the translation of the RNA and inhibiting protein synthesis [38,43,145]. This inhibition leads  
1200 to the inhibition of peptidyl transferase activity, opposing resistance to trichothecenes. In  
1201 cases in which the membrane is not permeable to deoxynivalenol, a virulence factor  
1202 promoting fungal growth [26] the propagation of the pathogen agent is restricted. Thus, three  
1203 to five days after inoculation, susceptible varieties display more extensive propagation of the  
1204 fungus, higher levels of deoxynivalenol accumulation and fewer pathological changes in  
1205 infected tissues [94]. A hypersensitive reaction is also possible [18].

1206

1207 This type of resistance can be identified visually by observing the symptoms, which reflect  
1208 the propagation of the disease from the point of infection [9]. It is also characterised by low  
1209 levels of ergosterol (an indicator of fungal biomass) [3]. The most well known variety  
1210 displaying this type of resistance is Sumai 3 [189].

1211

1212 Type I and II resistances may be combined in a given genotype [18,50]. This results in a  
1213 variety tolerating the invasion of grains that displays no symptoms on the grains [50].

1214 Similarly, the number of infected spikelets does not necessarily reflect the total amount of  
1215 damage done by the pathogen. The amount of damage also depends on the severity and  
1216 rapidity of the invasion of the rachis by the fungus. Thus, the three principal sources of  
1217 variation in the expression of symptoms are the environment [57], the species and/or strain of  
1218 *Fusarium* [57,212] and factors involved in the maturation of cereals that also play an  
1219 important role in the epidemiological profile of the disease [212]. Wheat contamination is  
1220 favoured by high levels of precipitation in the ten days preceding grain maturity, but only if  
1221 the minimum temperature in the ten days following ear emergence is sufficiently high [212].  
1222 It should be noted that these factors are varietal and also depend on the fungal species  
1223 concerned [212].

1224

1225 It seems likely that, in certain cases, the production of trichothecenes (deoxynivalenol in  
1226 particular) by fungi activates their development (hyphal growth: type II resistance) and  
1227 colonisation (type I resistance), by interfering with the defence response of the plants  
1228 [18,26,175,213]. The quantity of trichothecenes in the grains is controlled by the degradation  
1229 of deoxynivalenol and by the tolerance of the plants to this toxin [18].

1230

1231 Two other types of resistance have recently been discovered. Both involve defence reactions  
1232 directed against trichothecenes, in which the synthesis of this toxin is blocked or its  
1233 degradation enhanced [147,214].

1234

1235 **Type III: resistance involving the capacity to degrade** deoxynivalenol [214-216].

1236 In some varieties (e.g. Fredrick), enzymatic systems for the detoxification (degradation) of  
1237 trichothecenes, involving the acetyltransferase Ayt1p [209] for example, have been identified  
1238 based on a decrease in the amount of deoxynivalenol six weeks after inoculation [215].  
1239 Deoxynivalenol begins to accumulate three days after inoculation (four days according to  
1240 Savard, 2000[149]) of an ear. The concentration of this toxin increases, peaking after six  
1241 weeks, and then decreases naturally to a concentration that remains constant thereafter at  
1242 maturity, harvest and during grain storage [90].

1243

1244 This resistance was identified by inoculating grains of wheat, rice and triticale with a single  
1245 strain of *F. graminearum* [214]. This study showed that the varieties most resistant to  
1246 pathogen development (low *F. graminearum* biomass, as estimated from ergosterol content)  
1247 have a higher ergosterol/deoxynivalenol ratio than do varieties more susceptible to the

1248 development of this pathogen. This implies that the susceptible varieties are able to degrade  
1249 the deoxynivalenol produced by the large number of fungi present.

1250

1251 This type III resistance, characterised by a high ergosterol/deoxynivalenol ratio [213],  
1252 protects against symptoms due to the propagation of the fungus. However, there may  
1253 nonetheless be a decrease in the number or weight of grains and their size [9]. This resistance  
1254 is estimated visually by measuring yield and by comparing the values obtained for ears with  
1255 and without symptoms [9] but this estimation also depends on resistance to grain infection.

1256

1257 **Type IV: resistance involving tolerance to high concentrations** of deoxynivalenol  
1258 (apparent insensitivity to trichothecenes) [3].

1259 Like cultivars with type II resistance linked to the modification of peptidyl transferase, the  
1260 target of deoxynivalenol, cultivars with type IV resistance also have a modified peptidyl  
1261 transferase [3]. This enzyme stabilises the membrane, conferring greater tolerance to  
1262 trichothecenes [43]. It has also been suggested that changes in the permeability of the cell  
1263 wall or in the signal transduction cascade in the cell inducing greater tolerance to  
1264 trichothecenes by thaumatin-like proteins can account for this type of resistance [198].

1265

1266 For a given severity of disease, this resistance is generally estimated by a higher level of  
1267 deoxynivalenol compared to other genotypes [9].

1268

1269 **Type V: resistance to grain infection** [5].

1270 This type of resistance results in differences in yield despite similar levels of attack or,  
1271 conversely, little effect on yield despite the evident presence of the disease after artificial  
1272 inoculation [5,9,217]. In contrast to the other types of resistance, this type of resistance may  
1273 not correspond to a physiological process, but rather to the “morphological” expression of an  
1274 interaction between various complex processes: resistance of types II, III and IV.

1275

#### 1276 **4-3. Resistance to head blight according to the composition of plant tissues**

1277 In addition to genetic resistance, the composition of plant tissues is also a criterion of  
1278 resistance to the development and/or propagation of the fungus and its toxins. The  
1279 susceptibility of wheat cultivars is linked to the concentration of choline in the ear at anthesis  
1280 [218], superoxide dismutase activity in the ears in cases of contamination by deoxynivalenol  
1281 or *F. graminearum* [219] and the concentration of p-coumaric acid, one of the phenolic acid

1282 precursors of lignin [210]. The quantity of this compound in the ear is much higher after  
1283 inoculation of the glumes, envelopes and rachis with *F. culmorum* than in uninoculated  
1284 tissues [210]; this is thus a very important factor determining the susceptibility of crops. The  
1285 accumulation of an acetyltransferase encoded by *FsTri101* in the endosperm and glumes of  
1286 wheat confers partial protection against *F. graminearum* [220]. Similarly, feluric acid in the  
1287 ears seems to be involved in resistance to ear blight in maize [221], although healthy and  
1288 inoculated ears contain similar amounts of this compound [210].

1289

1290 The composition of wheat and barley grains may also affect contamination levels [134].  
1291 Couture [134] suggested that a high gluten content in hard wheats and a high starch/protein  
1292 ratio in malting barley could protect the crop against head blight.

1293

1294 The relative sugar content of leaves or stems has been proposed as an indicator of  
1295 susceptibility to diseases [222]. Unlike powdery mildew, head blight is favoured by a low  
1296 sugar content of the tissues [222]. Similar results were obtained in studies of stem blight in  
1297 maize, involving a parasitic complex consisting of 70% *Fusarium graminearum* [129]. The  
1298 refraction index of the sap of a section of pressed stem is used to assess the sugar content of  
1299 stem bases (Bertrand's method) and it has been shown that the higher the sugar content of  
1300 maize stems, the more resistant to stem blight the maize is likely to be [129]. Messiaen [129]  
1301 also indicated that *F. graminearum* is equally able to use sugars, starch and cellulose as  
1302 sources of carbon, and proteins, ammonium salts or nitrates as sources of nitrogen. Thus,  
1303 *Fusarium* makes use of storage proteins, and degrades the cell walls and starch grains of  
1304 infected grains [41,42].

1305

## 1306 **Conclusion**

1307 Head blight is a widespread disease with major consequences for health. However, it has been  
1308 little studied in the open field. Indeed, field studies of the epidemiological cycle of the disease  
1309 are hindered by a major problem: identification of the species and strains making up the  
1310 parasitic complex of the inoculum. A mean of five to seven species, and up to nine in total  
1311 [8], may be present in the same field in the same year. These species can only be identified  
1312 after culture in Petri dishes and the observation of a collection of morphological criteria  
1313 (colour, macroconidia, microconidia, ascospores, chlamydospores of a strain), which requires



1314 the conservation of living spores, or molecular genotyping (which remains difficult for these  
1315 species).

1316

1317 In the laboratory, the various steps in the plant infection process and in mycotoxin production  
1318 are essentially studied from an environmental viewpoint. Uncertainties remain concerning, in  
1319 particular, the necessity of inducing the development of perithecia by exposure to ultraviolet  
1320 light, the natural climatic conditions in which spores are released (macroconidia and  
1321 ascospores) and the sensitivity of these mechanisms to environmental factors.

1322

1323 In the field, spore dispersal depends on climatic factors in two ways: climatic factors affect  
1324 spore production (see section 1-2) and strongly regulate spore release. However, the climatic  
1325 conditions required for spore release are unclear. This is probably due to the difficulties  
1326 involved in identifying and isolating the various types of spore (ascospores or macroconidia).  
1327 Furthermore, the results obtained probably depend on the maturity of the spores: four periods  
1328 of release over a period of 20 to 30 days have been observed.

1329

1330 However, it seems that macroconidia are dispersed by means of splashing during rainfall. In  
1331 contrast, the release of ascospores is only triggered by rainfall (or high humidity), resulting in  
1332 the rehydration of the perithecia after a dry period. The true release of the ascospores occurs  
1333 several days later, depending on air humidity. Ascospore release, in *F. graminearum* in  
1334 particular, appears to be periodic.

1335

1336 Although the dispersal pathways of the spores are known (splashing for the macroconidia  
1337 and/or ascospores and wind dispersal for ascospores only) and long-distance dispersal appears  
1338 to be possible, the maximum dispersal distance and the contaminating potential of splashing  
1339 and wind dispersal have yet to be determined. Furthermore, local contamination seems to  
1340 predominate in the epidemiological profile of the disease in that two neighbouring plots  
1341 planted with the same variety may present very different levels of contamination.

1342

1343 The importance of local contamination raises the question of the hypothetical contamination  
1344 of spikelets via the systemic route. This question remains unanswered because as far as we  
1345 know no study has shown, microscopically, the systemic colonisation of the spikelets and the  
1346 growth of the fungus in plant tissues (or the impossibility of such colonisation). Similarly, the  
1347 existence, conditions of existence and potential importance of such contamination have not

1348 been precisely determined. Conflicting results have been obtained and it is possible that this  
1349 route of contamination is possible only for a few plant genotypes, for a few fungal species or  
1350 strains, or for a few plant-fungus interactions. Indeed, interactions between plants and  
1351 pathogens have been observed during infection. We can therefore presume that the virulence  
1352 characteristics of the infecting fungal strain, the resistance of the plant and the virulence-  
1353 avirulence interaction between plants and pathogens determines the capacity of the plant to be  
1354 infected by mycelium and/or spores, the speed of infection (colonisation) of the ear by the  
1355 fungal mycelium and the amount of mycotoxins present in the grains at harvest. These  
1356 phenomena, like the production of inoculum, also depend on climate.

1357

1358 The choice of variety is a practical consideration that may affect plant infection: the date and  
1359 site of fungal penetration, the propagation rate and the intensity of the consequences of the  
1360 attack (severity of symptoms and/or amount of toxins). Indeed, the choice of variety  
1361 determines the major mechanisms of varietal resistance activated. However, it should be  
1362 stressed that although various types of resistance have been identified, they have not yet been  
1363 entirely elucidated. Furthermore, the types of varietal resistance have not yet been  
1364 characterised for most wheat varieties, which makes it difficult to compare varieties in a  
1365 particular area. Together with genetic characteristics, sowing date and climate determine the  
1366 date at which the crop is most susceptible to infection.

1367

1368 The extent of infection depends directly on the quality of inoculum, the primary source of  
1369 which is crop residues. In the epidemiological cycle of the disease, residues probably modify  
1370 the conservation and development of the fungus and spore dispersal, with effects depending  
1371 on the amount of residue present (which depends on soil tillage methods and the preceding  
1372 crop) and their nutritive value for the pathogen and for the plant (which also depends  
1373 essentially on the preceding crop). The high levels of nitrogen in maize residues may result in  
1374 a longer period of residue colonisation by *Fusarium*, strengthening pathogen populations, and  
1375 stimulating their development. If too many crop residues are present, spore dispersal by  
1376 splashing may be physically limited by obstacles. Conversely, if the fungus has access to too  
1377 few residues or residues insufficiently rich for it to complete its life cycle and to develop, then  
1378 the fungus is likely to produce fewer spores and to disperse less effectively. There is also an  
1379 interaction between these factors. Limited soil tillage methods increase the density of residues  
1380 on the surface of the soil and hence increase the quantity of inoculum available, particularly if  
1381 the preceding crop is a potential host of the fungus (e.g. maize or durum wheat). Mycelium

1382 development and the production and dispersal of spores therefore depend on climate and  
1383 irrigation.

1384

1385 Although there is broad agreement among scientists and advisers concerning the effects of  
1386 soil tillage, irrigation and preceding crop on both disease severity and contamination by  
1387 mycotoxins, the cause of these effects remains unclear. In addition, uncertainties remain  
1388 concerning the survival of the fungus and its capacity to sporulate in soil, according to soil  
1389 aeration, porosity and light penetration, all of which are affected by soil tillage. Similarly, the  
1390 role of the compounds generated by the degradation of crop residues and the rate of residue  
1391 degradation — which depend on soil tillage, the nutritive value of the preceding crop and the  
1392 microclimate of the residues, which itself depends on irrigation and its relationship to mineral  
1393 composition (nitrogen, silicon, depending on the preceding crop) — make it difficult to  
1394 predict the quantitative effects of soil tillage, preceding crop and irrigation.

1395

1396 Other unanswered questions remain concerning the effect of residue degradation on  
1397 competition between microorganisms and/or plant resistance (notably by means of differences  
1398 in mineral nutrition).

1399

1400 The effect of mineral nutrition on disease severity and mycotoxin contamination, if indeed  
1401 there is one, remains unclear. The mineral balance of the soil influences pathogen populations  
1402 and should therefore have repercussions for the development of *Fusarium*. The composition  
1403 of plants, in terms of nitrogen and silicon, for example, probably affects the growth and  
1404 development of the fungus, although this has not been explicitly demonstrated in wheat for  
1405 several reasons: equilibrium between the various elements, fertilisation date, link with  
1406 *Fusarium* foot rot etc. A canopy with unlimited mineral nutrition develops well. This  
1407 development slightly modifies the environmental conditions of the residues, which may in  
1408 turn modify the rate of residue degradation. If this is indeed the case, then the capacity of the  
1409 fungus to sporulate and of the spores to disperse may be affected.

1410

1411 The role played by canopy density, in terms of crop plant and weed densities, and in the  
1412 development of epidemics of the disease remains to be determined. Weeds may carry  
1413 *Fusarium*, but is their role limited to that of an intermediate host (during splashing) or do they  
1414 act as a source of inoculum? If they serve as a source of inoculum, how many spores are  
1415 produced on weeds? Weeds probably also play an indirect role, modifying mineral nutrition

1416 and/or microclimate by increasing plant population density. The overall density of plants in  
1417 the canopy also depends on sowing density and the tillering capacity of the variety. Thus,  
1418 weeds may affect the number of spores produced, spore maturation and/or spore dispersal.  
1419 The uncertainties that remain probably arise at least in part from variations in the role played  
1420 by weeds according to the type or family of weeds concerned and competition with the crop:  
1421 date of emergence, density etc. (which also depends on climate). Although canopy density  
1422 (crop plus weeds) seems likely to play a role, we cannot be sure of the nature or magnitude of  
1423 this effect.

1424

1425 It also seems clear that competition between microorganisms on the leaves and ears is likely  
1426 to have an effect, but this is difficult to study in the field and necessarily depends on the  
1427 fungicides used and on canopy development.

1428

1429 Five types of varietal resistance have been identified to date. They affect the penetration of  
1430 the fungus into the plant (type I), infection kinetics (type II), the expression of the infection  
1431 (symptoms; type III), the consequences of infection (quantity of mycotoxins produced, yield;  
1432 type IV) and grain infection (type V). This complexity of plant resistance complicates any  
1433 study of several varieties. In such studies, caution is required when interpreting results  
1434 analysing the relationships between cultural practices and symptoms, between symptoms and  
1435 the quantities of mycotoxins produced and between cultural practices and the mycotoxins  
1436 produced.

1437

1438 In conclusion, the relationship between disease symptoms and mycotoxin contamination is of  
1439 key importance. The conditions in which this relationship is purely qualitative and those in  
1440 which it also has a quantitative element are unclear. If we are to propose effective methods for  
1441 the prevention of grain contamination by toxins, we must improve our understanding of this  
1442 relationship, especially as concerns the role of the cropping system. Investigation of the  
1443 effects of crop management and crop rotation on the profile of *Fusarium* species and strains  
1444 obtained within a cereal field, and the conditions in which the potential for toxin production is  
1445 expressed in agricultural environments, should be a research priority.

1446

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2076 Figure 1: Symptoms of *Fusarium* head blight on the external surface of wheat ear glumes.

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2092 Table 1: Data on environmental conditions for the production of macroconidia and ascospores in the laboratory,  
2093 according to species. Two natural populations of *F. graminearum* can be identified: Group 1 (or *F.*  
2094 *pseudograminearum*), normally associated with the diseased crowns of host plants, which do not form perithecia  
2095 in culture and form such structures only rarely in nature, and Group 2, associated with diseased of aerial plant  
2096 parts, which do form perithecia- [2,223,224].  
2097

Stage	Species	Determinant factors	Range	Optimal value	References	
Mycelial growth	<i>F. graminearum</i> group 2 (aerial)	Pressure	> -9 bars		[75] (on maize grains)	
		Humidity	> 94%RH			
		Pressure	> -12 bars	-2 bars		[225] (on agar)
		Temperature	4 to 32°C	28°C		[47]
		Pressure, Temperature	At -10 bars, T. optim 20°C At -28 bars, T. optim 30°C At -55 bars, T. optim 35°C			[16]
	Temperature	20 to 30°C			[23] (on osmotically adjusted agar)	
	Pressure	-10 to -28 bars				
			Accelerated by alternate periods of rainfall and drought before ear emergence			[59]
	<i>F. culmorum</i>	Temperature	20 to 30°C			[23] (on osmotically adjusted agar)
Pressure		-8 to -14 bars				
Pressure		At -8 bars, T. optim 20°C At -14 bars T. optim 30°C At -28 bars, T. optim 38°C Maximum growth at 25°C			[16]	
<i>M. nivale</i>	Temperature	0 to 28-32°C	18-20°C		[11, 16]	
Initiation of perithecia	<i>F. graminearum</i> group 2 (aerial)	Light	Requires low intensity of UV light (300-320<390nm)		[83], with conflicting results obtained by [35].	
			Depends on the strain		[35,71]	
	<i>F. roseum</i>	Depends on humidity and rain in spring			[59]	
Production de perithecia <i>F. graminearum</i> group 2 (aerial)	Temperature	5 to 35°C		29°C	[2,16]	
	Temperature	15 to 31°C		29°C	[83]	
	Pressure	< -50 bars, poor from -5 bars		-1.5 bars	[97] (on osmotically adjusted agar)	
	Depends on light				[68]	
Production of ascospores	<i>F. graminearum</i> group 2 (aerial)	Temperature	13 to 33°C	25-28°C	[2,16]	
Production of spores	<i>M. nivale</i>	Temperature	< 16-18 °C	6 à 8 °C	[64]	
	<i>Fusarium</i>	Temperature	Around 10°C		[64]	
Production of macroconidia	<i>F. graminearum</i> group 2 (aerial)	Temperature	28 to 32°C		[83]	
		None if T°<16°C or T°>36°C				
		Temperature	16-36°C	28-32°C		[47]
		Pressure	max < -50 bars	-1.4 to -3 bars		[97] (on osmotically adjusted agar)
	Temperature	20 to 30°C			[23] (on osmotically adjusted agar)	
	Pressure	-10 to -28 bars				
	<i>F. culmorum</i>	Temperature	20 to 30°C			[97] (on osmotically adjusted agar)
		Pressure	-8 to -14 bars			
	<i>F. graminearum</i> group 1 (soil) <i>F. avenaceum</i>	Pressure	Max between -15 and -60 bars		-15 bars	[97] (on osmotically adjusted agar)
Temperature		> 10°C		25 to 30°C	[16]	
<i>F. roseum</i>	Light	UV light required				
	Humidity	RH of about 100% Favoured by water stress				
<i>M. nivale</i>	Temperature	1 to 16 °C		6 to 8 °C	[16]	

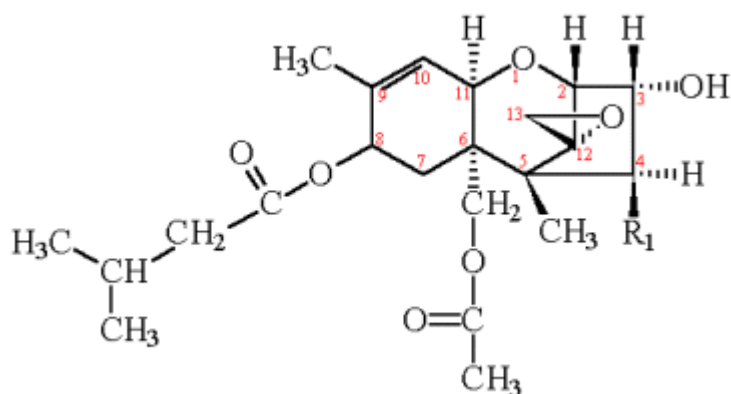
2101 Table 2: Spectrum of infection of weeds by *Fusarium* species. M: Monocotyledonous, g: grasses, D  
2102 Dicotyledonous.

Weed host			<i>Fusarium</i>	Site and form	Reference
Family	Latin name	Common name	Species		
Poaceae Mg	<i>Agropyron</i>	Wheatgrass	Not indicated	Not indicated	[85]
Liliaceae M	<i>Allium</i>	Garlic	<i>F. avenaceum</i> , <i>F. culmorum</i> , <i>F. graminearum</i>	Not indicated	[64]
Poaceae Mg	<i>Alopecurus myosuroides</i> Huds.	Blackgrass	<i>F. roseum</i>	In the seed	[48]
Poaceae Mg	<i>Avena fatua</i> L.	Wild oats	<i>F. roseum</i>	Lesion, spores on leaves and seeds	[48]
Chenopodiaceae D	<i>Beta vulgaris</i>	Wild beet	<i>F. culmorum</i>	Stem base	[62]
Cruciferae D	<i>Capsella bursa-pastoris</i> L.	Shepherd's purse	<i>F. avenaceum</i> , <i>F. culmorum</i> , <i>F. poae</i> , <i>F. sambucinum</i>	Stem base	[62]
Compositae D	<i>Cirsium arvense</i> L.	Thistle	<i>F. avenaceum</i>	Stem base	[62]
Poaceae Mg	<i>Dactylis</i> L.	Cocksfoot	<i>F. roseum</i>	Lesion on straw, stem base, increase with N	[14,48]
Poaceae Mg	<i>Echinochloa crus-galli</i> L.	Barnyardgrass	Not indicated	Not indicated	[85]
Poaceae Mg	<i>Festuca</i>	Fescue	<i>F. roseum</i>	Stem base, increase with N	[14]
Rubiaceae D	<i>Galium aparine</i> L.	Cleavers	<i>F. avenaceum</i> , <i>F. culmorum</i> , <i>F. poae</i>	Stem base	[62]
Poaceae Mg	<i>Lolium multiflorum</i> L.	Ryegrass	<i>F. roseum</i>	Lesion on straw, stem base, increase with N	[14,48]
Compositae D	<i>Matricaria spp.</i>	Mayweed	<i>F. avenaceum</i> , <i>F. culmorum</i> , <i>F. graminearum</i> , <i>F. poae</i> , <i>F. sambucinum</i>	Stem base	[62]
Ranunculaceae D	<i>Ranunculus acris</i>	Common buttercup	<i>F. avenaceum</i> , <i>F. culmorum</i> , <i>F. poae</i>	Stem base	[62]
Ranunculaceae D	<i>Ranunculus repens</i> L.	Creeping buttercup	<i>F. avenaceum</i> , <i>F. culmorum</i>	Stem base	[62]
Polygonaceae D	<i>Rumex obtusifolius</i>	Dock	<i>F. avenaceum</i> , <i>F. culmorum</i>	Stem base	[62]
Compositae D	<i>Senecio vulgaris</i> L.	Groundsel	<i>F. avenaceum</i> , <i>F. culmorum</i>	Stem base	[62]
Caryophyllaceae D	<i>Spergula arvensis</i> L.	Corn spurrey	<i>F. sambucinum</i>	Stem base	[62]
Carophyllaceae D	<i>Stellaria media</i> L.	Chickweed	<i>F. avenaceum</i>	Stem base	[62]
Fabaceae D	<i>Trifolium</i>	Clover/trefoil	<i>F. avenaceum</i> , <i>F. culmorum</i> , <i>F. graminearum</i>	Not indicated	[64]
Urticaceae D	<i>Urtica dioica</i> L.	Nettle	<i>F. avenaceum</i> , <i>F. culmorum</i> , <i>F. sambucinum</i>	Stem base	[62]
Scrophylariaceae D	<i>Veronica persica</i>	Speedwell	<i>F. graminearum</i>	Stem base	[62]
Violaceae D	<i>Viola arvensis</i> Murray	Field violet	<i>F. avenaceum</i> , <i>F. poae</i> , <i>F. culmorum</i> , <i>F. graminearum</i> , <i>F. sambucinum</i>	Stem base	[62]
Cryophyllaceae D	<i>Dianthus</i>	Carnation	<i>F. avenaceum</i> , <i>F. culmorum</i> , <i>F. poae</i> , <i>F. graminearum</i>	Not indicated	[64]

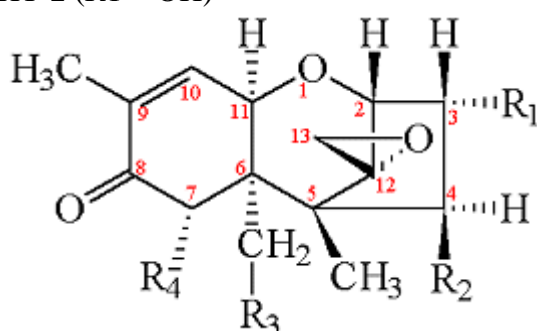
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2104 Figure 2: Chemical structures of trichothecenes, fumonisin B1 and zearalenone.

2105 **Trichothecenes**



Type A trichothecenes: T-2 (R1 = OAc)  
HT-2 (R1 = OH)

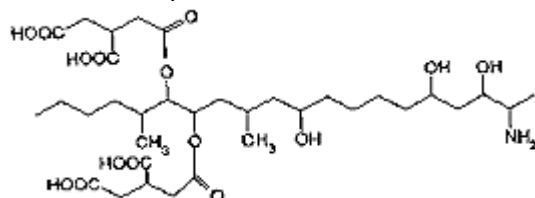


Type B trichothecenes: DON (R1 = OH, R2 = H, R3 = OH, R4 = OH)  
NIV (R1 = OH, R2 = OH, R3 = OH, R4 = OH)

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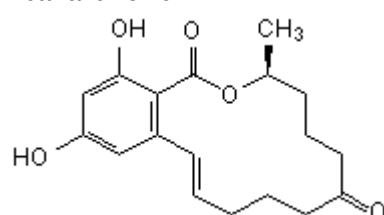
**Fumonisin B<sub>1</sub>**



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**Zearalenone**



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