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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

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2
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
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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*.

128

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 μm in length (mostly 35-62 μm), and 2 to 56 μm 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 μm x 3.5 – 5 μm [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/m², 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/mm³ 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 (SiO₂) 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 m² [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 (β -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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1451

1452 **References**

- 1453 1. D. Atanasoff, *Fusarium* blight (scab) of wheat and other cereals, *Journal of*
1454 *Agricultural Research*, 20(1), 1920, 1-32.
- 1455 2. J.C. Sutton, Epidemiology of wheat head blight and maize ear rot caused by *Fusarium*
1456 *graminearum*, *Canadian Journal of Plant Pathology*, 4, 1982, 195-209.
- 1457 3. Y.Z. Wang, J.D. Miller, Effects of *Fusarium graminearum* metabolites on wheat
1458 tissue in relation to *Fusarium* head blight resistance, *Journal of Phytopathology*, 122,
1459 1988, 118-125.
- 1460 4. J.D. Miller, R. Greenhalgh, Y. Wang, M. Lu, Trichothecene chemotypes of three
1461 *Fusarium* species, *Mycologia*, 83, 1991, 121-130.
- 1462 5. A. Mesterhazy, Types and components of resistance to *Fusarium* head blight of wheat,
1463 *Plant Breeding*, 114, 1995, 377-386.
- 1464 6. D.W. Parry, P. Jenkinson, L. McLeod, *Fusarium* ear blight (scab) in small grain
1465 cereals - a review, *Plant Pathology*, 44, 1995, 207-238.
- 1466 7. M. McMullen, R. Jones, D. Gallenberg, Scab of wheat and barley: a re-emerging
1467 disease of devastating impact, *Plant Disease*, 81, 1997, 1340-1348.
- 1468 8. A. Bottalico, *Fusarium* diseases of cereals: species complex and related mycotoxin
1469 profiles, in Europe, *Journal of Plant Pathology*, 80(2), 1998, 85-103.
- 1470 9. J.C. Rudd, R.D. Horsley, A.L. McKendry, E.M. Elias, Host plant resistance genes for
1471 fusarium head blight : sources, mechanisms, and utility in conventional breeding
1472 systems, *Crop Science*, 41(3), 2001, 620-627.
- 1473 10. A. Bottalico, G. Perrone, Toxigenic *Fusarium* species and mycotoxins associated with
1474 head blight in small-grain cereals in Europe, *European Journal of Plant Pathology*,
1475 108, 2002, 611-624.
- 1476 11. F.T. Bennett, *Fusarium* species on British cereals, *Annals of Applied Biology*, 20(2),
1477 1933, 272-290.
- 1478 12. G.W. Pugh, H. Johann, J.G. Dickson, Factors affecting infection of wheat heads by
1479 *Gibberella saubinetii*, *Journal of Agricultural Research*, 46, 1933, 771-797.

- 1480 13. P. Ruckebauer, H. Buerstmayr, M. Lemmens, Present strategies in resistance
1481 breeding against scab (*Fusarium* spp.), *Euphytica*, 119, 2001, 121-127.
- 1482 14. R. Cassini, Facteurs favorables ou défavorables au développement des fusarioses et
1483 septorioses du blé. In "Proceeding of meeting of sections cereals and physiology"
1484 (Eucarpia, ed.), Dijon, 1970, pp. 271-279.
- 1485 15. R.A. Daamen, C.J. Langerak, W. Stol, Surveys of cereal diseases and pests in The
1486 Netherlands. III- *Monographella nivalis* and *Fusarium* spp. in winter wheat fields and
1487 seed lots, *Netherlands Journal of Plant Pathology*, 97, 1991, 105-114.
- 1488 16. D. Caron, Les Fusarioses. In "Maladies des blés et orges", (ITCF, ed.), 1993, pp. 30-
1489 39.
- 1490 17. P. Jenkinson, D.W. Parry, Splash dispersal of conidia of *Fusarium culmorum* and
1491 *Fusarium avenaceum*, *Mycological Research*, 98, 1994a, 506-510.
- 1492 18. T. Ban, Studies on the genetics of resistance to *Fusarium* head blight caused by
1493 *Fusarium graminearum* in wheat. In "International symposium on wheat improvement
1494 for scab resistance. Section II. Genetic analysis and gene mapping for scab resistance."
1495 (Suzhou and Nanjing, eds.), Chin., 2000, pp. 5-10.
- 1496 19. R. Dill-Macky, R.K. Jones, The effect of previous crop residues and tillage on
1497 *Fusarium* head blight of wheat, *Plant Disease*, 84, 2000, 71-76.
- 1498 20. R. Ioos, B. Bakan, Study of the *Fusarium* spp. from cereal grains and their related
1499 mycotoxins. In "AFPP - 6^{ème} conférence internationale sur les maladies des plantes",
1500 Vol. 1, Tours, France, 2000, pp. 427-434.
- 1501 21. C. Maumené, M. Leuillet, D. Caron, F. Henriot, Incidence des fongicides et de la
1502 variété sur le risque de contamination du blé tendre par le déoxynivalénol (DON) en
1503 France : premiers résultats. In "AFPP - 6^{ème} conférence internationale sur les maladies
1504 des plantes", Vol. 1, Tours, France, 2000, pp. 459-466.
- 1505 22. D.R. Simpson, G.E. Weston, J.A. Turner, P. Jennings, P. Nicholson, Differential
1506 control of head blight pathogens of wheat by fungicides and consequences for
1507 mycotoxin contamination of grain, *European Journal of Plant Pathology*, 107, 2001,
1508 421-431.
- 1509 23. R.J. Cook, A.A. Christen, Growth of cereal root-rot fungi as affected by temperature-
1510 water potential interactions, *Phytopathology*, 66, 1976, 193-197.
- 1511 24. R.W. Stack, M.P. McMullen, Head blighting potential of *Fusarium* species associated
1512 with spring wheat heads, *Canadian Journal of Plant Pathology*, 7, 1985, 79-82.

- 1513 25. C.H.A. Snijders, Diallel analysis of resistance to head blight caused by *Fusarium*
 1514 *culmorum* in winter wheat, *Euphytica*, 50, 1990c, 1-9.
- 1515 26. C.H.A. Snijders, C.F. Krechting, Inhibition of deoxynivalenol translocation and fungal
 1516 colonization in *Fusarium* head blight resistant wheat, *Canadian Journal of Botany*, 70,
 1517 1992, 1570-1576.
- 1518 27. S. Homdork, H. Fehrmann, R. Beck, Effects of field application of tebuconazole on
 1519 yield, yield components and the mycotoxin content of *Fusarium*-infected wheat grain,
 1520 *Journal of Phytopathology*, 148, 2000, 1-6.
- 1521 28. L.N. Jorgensen, Control of *Fusarium* ear blight in winter wheat [Danish] (abstract). *In*
 1522 "17th Danish Plant Protection Conference II, Site-specific crop protection, decision
 1523 support, pests and diseases, ear blight", Denmark, 2000.
- 1524 29. W.G. Smith, "Diseases of field and garden crops. *Cité par Parry, 1995*," London,
 1525 MacMillan and Co, 1884, pp. 208-213.
- 1526 30. A. Mesterhazy, A.K. Rowaished, Analysis of symptoms caused by *Fusarium*
 1527 *graminearum* Schwabe and its relation to powdery mildew infection in wheat, *Acta*
 1528 *Phytopathologica Academiae Scientiarum Hungaricae*, 12(3-4), 1977, 289-301.
- 1529 31. B. Carlier, Les fusarioses : des maladies dures à combattre, *Jeunes agriculteurs*, 557,
 1530 2001.
- 1531 32. W. Ahrens, H. Fehrmann, Attack of wheat by *Septoria nodorum* and *Fusarium* ear
 1532 scab. I- crop loss analysis, *Journal of Plant Diseases and Protection*, 91(1), 1984, 42-
 1533 58.
- 1534 33. V. Le Boulc'h, S. Bedouret, V. Poyet, T. Barchietto, D. Hazouard, I. De Paepe, J.M.
 1535 Seng, Un test innovant pour contrôler la qualité sanitaire des céréales : détection des
 1536 champignons potentiellement producteurs d'une famille de mycotoxines, les
 1537 trichothécènes. *In* "AFPP - 6^{ème} conférence internationale sur les maladies des
 1538 plantes", Vol. 1, Tours, France, 2000, pp. 451-458.
- 1539 34. P. Galtier, C. Comera, I. Oswald, O. Puel, Mycotoxines : origines et toxicités. *In*
 1540 "AFPP - 6^{ème} conférence internationale sur les maladies des plantes", Vol. 1, Tours,
 1541 France, 2000, pp. 77-86.
- 1542 35. J. Gilbert, A. Tekauz, Review: Recent developments in research on *Fusarium* head
 1543 blight of wheat in Canada, *Canadian Journal of Plant Pathology*, 22, 2000, 1-8.
- 1544 36. J. Chelkowski, Fungal pathogens influencing cereal seed quality at harvest. *Cité par*
 1545 *Ruckenbauer et al. 2001*. *In* "Cereal grain: mycotoxins, fungi and quality in drying and

- 1546 storage", (Chelkowski, ed.), Developments in Food Science, 26, Elsevier, Amsterdam,
1547 1991, pp. 53-56.
- 1548 37. D. Thouvenot, Moisissure et mycotoxine. In "Mycotoxines : un prochain enjeu de
1549 sécurité alimentaire ?" (CERAAF, ed.), 2002, pp. 14-17.
- 1550 38. B. Bakan, Approche physiologique de la biosynthèse des trichothécènes par *Fusarium*,
1551 Thèse INA-PG, 1998, pp. 150.
- 1552 39. D.R. Vidal, Propriétés immunosuppressives des mycotoxines du groupe des
1553 trichothécènes, Bulletin de l'institut Pasteur, 88, 1990, 159-192.
- 1554 40. J.F. Quillien, "Les mycotoxines," Institut National de la Recherche Agronomique,
1555 France, 2002, pp. 24.
- 1556 41. D.B. Bechtel, L.A. Kaleikau, R.L. Gaines, L.M. Seitz, The effects of *Fusarium*
1557 *graminearum* infection on wheat kernels, Cereal Chemistry, 62(3), 1985, 191-197.
- 1558 42. L.M. Seitz, W.T. Yamazaki, R.L. Clements, H.E. Mohr, L. Andrews, Distribution of
1559 deoxynivalenol in soft wheat mill streams, Cereal Chemistry, 62(6), 1985, 467-469.
- 1560 43. F. Eudes, Etude de l'impact des trichothécènes de *Fusarium* chez le blé et sélection in
1561 vitro pour la résistance à la fusariose de l'épi, Université de Laval Québec, 1998, pp.
1562 83p+ annexes.
- 1563 44. IARC, Some naturally occurring substances: food items and constituents, heterocyclic
1564 aromatic amines and mycotoxins. In "Monographs on the evaluation of carcinogenic
1565 risks to humans", (IARC, ed.), Vol. 56, Lyon, 1993.
- 1566 45. W. Langseth, R. Hoie, M. Gullord, The influence of cultivars, location and climate on
1567 deoxynivalenol contamination of Norwegian oats 1985-1990, Acta Agriculturae
1568 Scandinavica, 45, 1995, 63-67.
- 1569 46. L.J. Franc, Development of *Fusarium* head blight in relation to environment and
1570 inoculum. In "National *Fusarium* Head Blight Forum. Chapter 1- Epidemiology and
1571 Disease Management", Michigan State university (USA), 1998, pp. 1-3.
- 1572 47. A.L. Andersen, The development of *Gibberella zeae* head blight of wheat,
1573 Phytopathology, 38, 1948, 595-611.
- 1574 48. R. Cassini, "A propos des dégâts provoqués par *Fusarium roseum* dans les cultures de
1575 céréales du bassin parisien," Comptes Rendus de l' Académie d'Agriculture de France,
1576 1967, pp. 858-867.
- 1577 49. G.H. Bai, R. Plattner, A.E. Desjardins, F. Kolb, Resistance to *Fusarium* head blight
1578 and deoxynivalenol accumulation in wheat, Plant Breeding, 120, 2001, 1-6.

- 1579 50. H.W. Schroeder, J.J. Christensen, Factors affecting resistance of wheat to scab caused
1580 by *Gibberella zeae*, *Phytopathology*, 53, 1963, 831-838.
- 1581 51. J. Chelkowski, H. Wisniewska, T. Adamski, P. Golinski, Z. Kaczmarek, M. Kostecki,
1582 J. Perkowski, M. Surma, Effects of *Fusarium culmorum* head blight on mycotoxin
1583 accumulation and yield traits in barley doubled haploids, *Journal of Phytopathology*,
1584 148, 2000, 541-545.
- 1585 52. B.J. Blaney, R.L. Dodman, Production of zearalenone, deoxynivalenol, nivalenol, and
1586 acetylated derivated by Australian isolates of *Fusarium graminearum* and *F.*
1587 *pseudograminearum* in relation to source and culturing conditions, *Australian Journal*
1588 *of Agricultural Research*, 53, 2002, 1317-1326.
- 1589 53. N. Magan, R. Hope, A. Colleate, E.S. Baxter, Relationship between growth and
1590 mycotoxin production by *Fusarium* species, biocides and environment, *European*
1591 *Journal of Plant Pathology*, 108, 2002, 685-690.
- 1592 54. D.C. Hooker, A.W. Schaafsma, L. Tamburic-Ilincic, Using weather variables pre- and
1593 post-heading to predict deoxynivalenol content in winter wheat, *Plant Disease*, 86,
1594 2002, 611-619.
- 1595 55. A. Champeil, J.F. Fourbet, T. Doré, Influence of cropping system on *Fusarium* head
1596 blight and mycotoxin levels in winter wheat, *Crop Protection. Sous presse*, 2004.
- 1597 56. L. Scott, S. Leath, W. Hagler, J. Murphy, Variation in *Fusarium graminearum*
1598 associated with wheat scab in north Carolina. In "National Fusarium Head Blight
1599 Forum. Chapter 1- Epidemiology and Disease Management", Michigan State
1600 University (USA), 1998, pp. 15.
- 1601 57. M. Dusabenyagasani, R.C. Hamelin, J. Collin, D. Dostaler, Importance de l'interaction
1602 entre les cultivars de blé et les souches de *Fusarium graminearum* dans l'évaluation de
1603 la résistance à la fusariose de l'épi, *Phytoprotection*, 78(2), 1997, 53-60.
- 1604 58. P. Pissinger, Fusarium infection of wheat seeds in the Vas province [Hungarian]
1605 (abstract), *Novenyvedelem*, 18, 1982, 83-85.
- 1606 59. D. Caron, Fusarioses des épis. Sait-on prévoir leur développement ?, *Perspectives*
1607 *Agricoles*, 253, 2000, 56-62.
- 1608 60. M.G. Cromeey, S.C. Shorter, D.R. Lauren, K.I. Sinclair, Cultivar and crop management
1609 influences on fusarium head blight and mycotoxins in spring wheat (*Triticum*
1610 *aestivum*) in New Zealand, *New Zealand Journal of Crop and Horticultural Science*,
1611 30, 2002, 235-247.

- 1612 61. G. Bai, G. Shaner, Scab of wheat : Prospects for control, *Plant Disease*, 78(8), 1994,
1613 760-766.
- 1614 62. P. Jenkinson, D.W. Parry, Isolation of *Fusarium* species from common broad-leaved
1615 weeds and their pathogenicity to winter wheat, *Mycological Research*, 98, 1994b, 776-
1616 780.
- 1617 63. C.M. Messiaen, R. Cassini, Recherches sue les Fusarioses. IV- La systématique des
1618 *Fusarium*, *Annales des Epiphyties*, 19(3), 1968, 387-454.
- 1619 64. F. Rapilly, J.M. Lemaire, R. Cassini, Les Fusarioses. In "Les maladies des céréales -
1620 Principales maladies cryptogamiques", (INRA-ITCF, ed.), 1973, pp. 119.
- 1621 65. G. Le Henaff, B. Oste, E. Wilhem, A. Faure, J. Moinard, P. Lepoutre, O. Pillon, Bilan
1622 sanitaire 2001-2002 des céréales. Retour au sec et au calme pour des résultats moyens,
1623 *Phytoma*, 556, 2003, 22-26.
- 1624 66. F. Trail, H. Xu, R. Loranger, D. Gadoury, Physiological and environmental aspects of
1625 ascospore discharge in *Gibberella zeae* (anamorph *Fusarium graminearum*),
1626 *Mycologia*, 94, 2002, 181-189.
- 1627 67. T. Paulitz, Diurnal release of ascospores by *Gibberella zeae* in inoculated wheat plots,
1628 *Plant Disease*, 80, 1996, 674-678.
- 1629 68. F. Trail, D. Gadoury, R. Loranger, Environmental parameters of ascospore discharge
1630 in *Gibberella zeae*. In "National Fusarium Head Blight Forum. Chapter 1-
1631 Epidemiology and Disease Management", Michigan State University (USA), 1998,
1632 pp. 11-13.
- 1633 69. W.L. Gordon, The occurrence of *Fusarium* species in Canada. VI- Taxonomy and
1634 geographic distribution of *Fusarium* species on plants, insects, and fungi. *Cité par*
1635 *Parry et al. 1995*, *Canadian Journal of Botany*, 37, 1959, 257-290.
- 1636 70. J.A. Clement, D. Parry, Stem-base disease and fungal colonisation of winter wheat
1637 grown in compost inoculated with *Fusarium culmorum*, *F. Graminearum* and
1638 *Microdochium nivale* (abstract), *European Journal of Plant Pathology*, 104, 1998, 323-
1639 330.
- 1640 71. C.S. Millar, J. Colhoun, *Fusarium* diseases of cereals. IV- Observations on *Fusarium*
1641 *nivale* on wheat, *Transactions of the British Mycological Society*, 52, 1969, 57-66.
- 1642 72. V.W.L. Jordan, E.C. Fielding, "*Fusarium* spp. on wheat. *Cité par Parry, 1995*," Long
1643 Ashton Research Station, 1988.
- 1644 73. C.H.A. Snijders, Systemic fungal growth of *Fusarium culmorum* in stems of winter
1645 wheat, *Journal of Phytopathology*, 129, 1990a, 133-140.

- 1646 74. T. Sieber, T.K. Risen, E. Müller, P.M. Fried, Endophytic fungi in four winter wheat
1647 cultivars (*Triticum aestivum* L.) differing in resistance against *Stagonospora nodorum*
1648 (Berk.) Cast. Et germ. = *Septoria nodorum* (berk.) Berk. *Cité par Jenkinson, 1994b,*
1649 *Journal of phytopathology*, 122, 1988, 289-306.
- 1650 75. W.G.D. Fernando, J.D. Miller, W.L. Seaman, K. Seifert, T.C. Paulitz, Daily and
1651 seasonal dynamics of airborne spores of *Fusarium graminearum* and other *Fusarium*
1652 species sampled over wheat plots, *Canadian Journal of Botany*, 78, 2000, 497-505.
- 1653 76. H.M. Hörberg, Patterns of splash dispersed conidia of *Fusarium poae* and *Fusarium*
1654 *culmorum*, *European Journal of Plant Pathology*, 108, 2002, 73-80.
- 1655 77. F.J. Bannon, B.M. Cooke, Studies on dispersal of *Septoria tritici* pycnidiospores in
1656 wheat-clover intercrops, *Plant Pathology*, 47, 1998, 49-56.
- 1657 78. B. Birzele, A. Meier, H. Hindorf, J. Krämer, H.W. Dehne, Epidemiology of *Fusarium*
1658 infection and deoxynivalenol content in winter wheat in the Rhineland, Germany,
1659 *European Journal of Plant Pathology*, 108, 2002, 667-673.
- 1660 79. R.W. Stack, Gradients of *Fusarium* head blight in wheat along transects away from a
1661 concentrated source of *Gibberella zeae* ascospore inoculum. In "Proceedings of the
1662 1997 National Fusarium Head Blight Foru, 10-12 nov." St. Paul, Minn, 1997, pp. 60.
- 1663 80. R. Bandyopadhyay, L.K. Mughogho, M.V. Satyanarayana, M.E. Kalisz, Occurrence
1664 of airborne spores of fungi causing grain mould over a sorghum crop, *Mycological*
1665 *Research*, 95(11), 1991, 1315-1320.
- 1666 81. F.R. Sanderson, Fusarium diseases of cereals. VIII. Discharge and dispersal of
1667 ascospores of *Calonectria nivalis*, *Transactions of the British Mycological Society*,
1668 55(1), 1970, 137-140.
- 1669 82. C.T. Ingold, "Fungal spores: Their liberation and dispersal. *Cité par Paulitz, 1996,*"
1670 Clarendon Press, Oxford, UK, 1971.
- 1671 83. A.T. Tschanz, R.K. Horst, P.E. Nelson, The effect of environnement on sexual
1672 reproduction of *Gibberella zeae*, *Mycologia*, 68, 1976, 327-340.
- 1673 84. H.Z. Ye, On the biology of the perfect stage of *Fusarium graminearum* Schw. *Cité*
1674 *par Sutton, 1982,* *Acta Phytopathologica Sinica*, 7, 1980, 35-42.
- 1675 85. www.syngenta.ca, 2002.
- 1676 86. R.N. Strange, H. Smith, A fungal growth stimulant in anthers which predisposes
1677 wheat to attack by *Fusarium graminearum*, *Physiological Plant Pathology*, 1, 1971,
1678 141-150.

- 1679 87. J. Lacey, G.L. Bateman, C.J. Mirocha, Effects of infection time and moisture on
1680 development of ear blight and deoxynivalenol production by *Fusarium* spp. in wheat,
1681 *Annals of Applied Biology*, 134, 1999, 277-283.
- 1682 88. K. Matsui, M. Yoshida, T. Ban, T. Komatsuda, N. Kawada, Role of male-sterile
1683 cytoplasm in resistance to barley yellow mosaic virus and *Fusarium* head blight in
1684 barley, *Plant Breeding*, 121, 2002, 237-240.
- 1685 89. R.N. Strange, H. Smith, Effects of choline, betaine and wheat-germ extract on growth
1686 of cereal pathogens, *Transactions of the British Mycological Society*, 70(2), 1978,
1687 193-199.
- 1688 90. A.H. Teich, Epidemiology of wheat (*Triticum aestivum* L.) scab caused by *Fusarium*
1689 spp. In "Fusarium: mycotoxins, taxonomy and pathogenicity", (Chelkowski, ed.),
1690 1989, pp. 269-282.
- 1691 91. B.D. McCallum, A. Tekauz, Influence of inoculation method and growth stage on
1692 *Fusarium* head blight in barley, *Canadian Journal of Plant Pathology*, 24, 2002, 77-80.
- 1693 92. L.M. Reid, T. Woldemariam, X. Zhu, D.W. Stewart, A.W. Schaafsma, Effect of
1694 inoculation time and point of entry on disease severity in *Fusarium graminearum*,
1695 *Fusarium verticillioides*, or *Fusarium subglutinans* inoculated maize ears, *Canadian*
1696 *Journal of Plant Pathology*, 24, 2002, 162-167.
- 1697 93. M. Urban, S. Daniels, E. Mott, K. Hammond-Kosack, *Arabidopsis* is susceptible to the
1698 cereal ear blight fungal pathogens *Fusarium graminearum* and *Fusarium culmorum*,
1699 *The Plant Journal*, 32, 2002, 961-973.
- 1700 94. Z. Kang, H. Buchenauer, Ultrastructural and immunocytochemical investigation of
1701 pathogen development and host responses in resistant and susceptible wheat spikes
1702 infected by *Fusarium culmorum*, *Physiological and Molecular Plant Pathology*, 57,
1703 2000, 255-268.
- 1704 95. W.M. Wanjiru, K. Zhensheng, H. Buchenauer, Importance of cell wall degrading
1705 enzymes produced by *Fusarium graminearum* during infection of wheat heads,
1706 *European Journal of Plant Pathology*, 108, 2002, 803-810.
- 1707 96. Z. Kang, H. Buchenauer, Studies on the infection process of *Fusarium culmorum* in
1708 wheat spikes: Degradation of host cell wall components and localization of
1709 trichothecene toxins in infected tissue, *European Journal of Plant Pathology*, 108,
1710 2002, 653-660.

- 1711 97. J.M. Sung, R.J. Cook, Effects of water potential on reproduction and spore
 1712 germination by *Fusarium roseum* 'graminearum', 'culmorum', and 'avenaceum',
 1713 Phytopathology, 71(5), 1981, 499-504.
- 1714 98. R. Cassini, Influences des techniques de culture sur le développement des maladies
 1715 des céréales. Chap. Méthodes de lutte culturale, génétique et biologique. In "La lutte
 1716 contre les maladies des céréales", (C. ITCF, ed.), Versailles, 1973, pp. 17-34.
- 1717 99. A.H. Teich, K. Nelson, Survey of *Fusarium* head blight and possible effects of
 1718 cultural practices in wheat fields in Lambton County in 1983, Canadian Plant Disease
 1719 Survey, 64(1), 1984, 11-13.
- 1720 100. A.H. Teich, J.R. Hamilton, Effect of cultural practices, soil phosphorus, potassium,
 1721 and pH on the incidence of *Fusarium* head blight and deoxynivalenol levels in wheat,
 1722 Applied and Environmental Microbiology, 79(6), 1985, 1429-1431.
- 1723 101. H. Krebs, D. Dubois, C. Kulling, H.R. Forrer, The effects of preceding crop and
 1724 tillage on the incidence of *Fusarium* spp. and mycotoxin deoxynivalenol content in
 1725 winter wheat grain [German] (abstract), Agrarforschung, 7, 2000, 264-268.
- 1726 102. L. Manuca, Competition between predominant bacteria in the wheat rhizosphere and
 1727 fusarium culmorum (Sm.) Sacc. in vitro (abstrat), I.C.C.P.T. Fundulea, 35, 1969, 765-
 1728 774.
- 1729 103. K. Trolldenier, Maladie des céréales et nutrition des plantes, Revue de la Potasse,
 1730 section 23, 1969, 1-16.
- 1731 104. I. Coulomb, J. Caneill, H. Manichon, Comportement du sol au labour : méthode
 1732 d'analyse et évaluation des conséquences de l'état initial du sol sur l'état transformé par
 1733 le labour, Agronomie, 13, 1993, 45-56.
- 1734 105. J. Guerif, G. Richard, C. Durr, J.M. Machet, S. Recous, J. Roger-Estrade, A review of
 1735 tillage effects on crop residue management, seedbed conditions and seedling
 1736 establishment, Soil & Tillage Research, 61, 2001, 13-32.
- 1737 106. D.C. Reicosky, Tillage-induced soil properties and chamber mixing effects on gas
 1738 exchange. In "International soil tillage research organisation 16th triennial conference",
 1739 Vol. 1, Erica Maddock, Acacia Computing Services Pty Ltd, The University of
 1740 Queensland Brisbane, Australia, 2003, pp. 117.
- 1741 107. Y. Ben-Yephet, M. Reuven, A. Genizi, Effects of inoculum depth and density on
 1742 *Fusarium* wilt in carnations, Phytopathology, 84(12), 1994, 1393-1398.

- 1743 108. G.L. Bateman, H. Coskun, Populations of *Fusarium* spp. in soil growing continuous
1744 winter wheat, and effects of long-term application of fertilizers and of straw
1745 incorporation, *Mycological Research*, 99(11), 1995, 1391-1394.
- 1746 109. G.L. Bateman, G. Murray, R.J. Gutteridge, H. Coskun, Effects of method of straw
1747 disposal and depth of cultivation on populations of *Fusarium* spp. in soil and on
1748 brown foot rot in continuous winter wheat, *Annals of Applied Biology*, 132, 1998, 35-
1749 47.
- 1750 110. M. Jalaluddin, J.F. Jenkyn, Effects of wheat crop debris on the sporulation and
1751 survival of *Pseudocercospora herpotrichoides*, *Plant Pathology*, 45, 1996, 1052-
1752 1064.
- 1753 111. D.J. Yarham, The effect on soil-borne diseases of changes in crop and soil
1754 management. In "Schippers B, gams Weds", *Soil-borne Plant Pathogens*, UK
1755 Academic Press, London, 1979, pp. 371-383.
- 1756 112. B.S. Rodgers-Gray, M.W. Shaw, Substantial reductions in winter wheat diseases
1757 caused by addition of straw but not manure to soil, *Plant Pathology*, 49, 2000, 590-
1758 599.
- 1759 113. T.L.W. Carver, R.J. Zeyen, G.G. Ahlstrand, The relation between insoluble silicon
1760 and success or failure of attempted primary penetration by powdery mildew (*Erysiphe*
1761 *graminis*) germlings on barley, *Physiological and Molecular Plant Pathology*, 31,
1762 1987, 133-148.
- 1763 114. R.R. Bélanger, P.A. Bowen, D.L. Ehret, J.G. Menzies, Soluble silicon: its role in crop
1764 and disease management of greenhouse crops, *Plant Disease*, 79(4), 1995, 329-336.
- 1765 115. B.A. Summerell, L.W. Burgess, T.A. Klein, A.B. Pattison, Stubble management and
1766 the site of penetration of wheat by *Fusarium graminearum* group 1, *Phytopathology*,
1767 80, 1990, 877-879.
- 1768 116. M.L. Deadman, B.M. Cooke, Wheat disease and cereal-clover bicrops, *Management*
1769 *Focus*, 2, 1997, 15-19.
- 1770 117. M.H. Leitch, P.D. Jenkins, Influence of nitrogen on the development of *Septoria*
1771 epidemics in winter wheat, *Journal of Agricultural Science*, 124, 1995, 361-368.
- 1772 118. R.A. Martin, J.A. MacLeod, C. Caldwell, Influences of production inputs on incidence
1773 of infection by *Fusarium* species on cereal seed, *Plant Disease*, 75(8), 1991, 784-788.
- 1774 119. M. Lemmens, K. Haim, H. Lew, P. Ruckebauer, The effect of nitrogen fertilization
1775 on *Fusarium* head blight development and deoxynivalenol contamination in wheat,
1776 *Journal of Phytopathology*, 152, 2004, 1-8.

- 1777 120. M.T. Fauzi, T.C. Paulitz, The effect of plant growth regulators and nitrogen on
1778 Fusarium head blight of the spring wheat cultivar max, *Plant Disease*, 78, 1994, 289-
1779 292.
- 1780 121. R.W. Smiley, R.J. Cook, P. R.I., Fusarium foot rot of wheat and peas as influenced by
1781 soil applications of anhydrous ammonia and ammonia-potassium azide solutions,
1782 *Phytopathology*, 62, 1972, 86-91.
- 1783 122. D.M. Huber, R.D. Watson, Nitrogen form and plant disease, *Annuals review of*
1784 *phytopathology*, 12, 1974, 139-165.
- 1785 123. G. Lazarovits, Management of soil-borne plant pathogens with organic soil
1786 amendments: a disease control strategy salvaged from the past, *Canadian Journal of*
1787 *Plant Pathology*, 23, 2001, 1-7.
- 1788 124. H.J.M. Loffler, E.B. Cohen, G.T. Oolbekkink, B. Schippers, Nitrite as a factor in the
1789 decline of *Fusarium oxysporum* f. sp. dianthi in soil supplemented with urea and
1790 ammonium chloride, *Netherlands Journal of Plant Pathology*, 92, 1986, 153-162.
- 1791 125. M. Ciotola, P. Neumann, Role of post harvest and spring application of urea and
1792 *Trichoderma reesii* QM9414 on the development of *Venturia inequalis*. *Cité par*
1793 *Teich, 1989. In "Mycolog. Society America and Canadian Phytopathol. Society",*
1794 *1987.*
- 1795 126. B.K. Vaidehi, Effect of foliar application of urea on the behaviour of
1796 *Helminthosporium hawaiiensis* in the rhizosphere of rice, *Indian Journal of Mycology*
1797 *and Plant Pathology*, 3, 1973, 81-85.
- 1798 127. A.K. Rowaished, The influence of different forms of nitrogen on *Fusarium* root-rot
1799 disease of winter wheat seedlings, *Phytopathology*, 100, 1981, 331-339.
- 1800 128. P.E. Onuorah, Effect of mineral nutrition on the Fusarium Brown foot-rot of wheat,
1801 *Plant and Soil*, (1), 1969, 99-104.
- 1802 129. C.M. Messiaen, Richesse en sucre des tiges de maïs et verse parasitaire, *Revue de*
1803 *Pathologie Végétale et d'Entomologie Agricole de France*, 36, 1957, 209-213.
- 1804 130. E. Kurek, J. Jaroszuk-Scisel, Rye (*Secale cereale*) growth promotion by *Pseudomonas*
1805 *fluorescens* strains and their interactions with *Fusarium culmorum* under various soil
1806 conditions, *Biological Control*, 26, 2003, 48-56.
- 1807 131. C. Alabouvette, Y. Couteaudier, J. Louvet, Recherches sur la résistance des sols aux
1808 maladies. XI- Etude comparative du comportement des *Fusarium* spp. dans un sol
1809 résistant et un sol sensible aux fusarioses vasculaires enrichis en glucose, *Agronomie*,
1810 5(1), 1985a, 63-68.

- 1811 132. C. Alabouvette, Y. Couteaudier, J. Louvet, Recherches sur la résistance des sols aux
 1812 maladies. XII- Activité respiratoire dans un sol résistant et un sol sensible aux
 1813 fusarioses vasculaires enrichis en glucose, *Agronomie*, 1985b, 69-72.
- 1814 133. J.C. Arthur, Wheat scab. *Cité par Teich, 1989 et Parry et al. 1995*, Indiana
 1815 Agricultural Experimental Station Bulletin, 36, 1891, 129-132.
- 1816 134. L. Couture, Réceptivité de cultivars de céréales de printemps à la contamination des
 1817 graines sur inflorescence par les *Fusarium* spp., *Canadian Journal of Plant Science*,
 1818 62, 1982, 29-34.
- 1819 135. M.G. Anderson, "Winter wheat disease survey 1985. *Cité par Parry et al. 1995*,"
 1820 Research Station Kentville, Nova Scotia, 1986, pp. 53-55.
- 1821 136. E.W. Hanson, E.R. Ausemus, E.C. Stakman, Varietal resistance of spring wxeats to
 1822 fusarial head blights. *Cité par Teich and Hamilton, 1989*, *Phytopathology*, 40, 1950,
 1823 902-914.
- 1824 137. I. Bujold, T.C. Paulitz, O. Carisse, Effect of *Microsphaeropsis* sp. on the production
 1825 of perithecia and ascospores of *Gibberella zeae*, *Plant Disease*, 85(9), 2001, 977-984.
- 1826 138. H. Diamond, B.M. Cooke, Preliminary studies on biological control of the *Fusarium*
 1827 ear blight complex of wheat, *Crop Protection*, 22, 2003, 99-107.
- 1828 139. I. Bujold, O. Carisse, T.C. Paulitz, Effect of *Microsphaeropsis* sp. strain P130A on the
 1829 field production of perithecia of *Gibberella zeae* on wheat residues. In "Canadian
 1830 Workshop on *Fusarium* head blight", Manitoba, 1999, pp. 112-113.
- 1831 140. M. Davanlou, A.M. Madsen, C.H. Madsen, J. Hockenhull, Parasitism of
 1832 macroconidia, chlamydospores and hyphae of *Fusarium culmorum* by mycoparasitic
 1833 *Pythium* species, *Plant Pathology*, 48, 1999, 352-359.
- 1834 141. J.C. Zadoks, T.T. Chang, C.F. Knorzak, A decimal code for the growth stages of
 1835 cereals, *Weed Research*, 14, 1974, 415-421.
- 1836 142. J. Liggitt, P. Jenkinson, D.W. Parry, The role of saprophytic microflora in the
 1837 development of *Fusarium* ear blight of winter wheat caused by *Fusarium culmorum*,
 1838 *Crop Protection*, 16(7), 1997, 679-685.
- 1839 143. A. Mesterhazy, T. Bartok, Control of *Fusarium* head blight of wheat by fungicides
 1840 and its effect on the toxin contamination of the grains, *Pflanzenschutz-Nachrichten*
 1841 Bayer, 49(2), 1996, 181-198.
- 1842 144. C.S. McLaughlin, M.H. Vaughan, L.M. Campbell, C.M. Wei, M.E. Stafford, B.S.
 1843 Hansen, Inhibition of protein synthesis by trichothecenes. *Cité par Eudes et al., 2000*.

- 1844 *In "Mycotoxins in human and animal health", (P. F. S. Pathotox, ed.), Rodricks, J.V.,*
1845 *Hesseltine, C.W., Mehlman, M.A., 1977.*
- 1846 145. F. Eudes, A. Comeau, S. Rioux, J. Collin, Phytotoxicité de huit mycotoxines associées
1847 à la fusariose de l'épi chez le blé, *Canadian Journal of Plant Pathology*, 22, 2000, 286-
1848 292.
- 1849 146. E. Young, Fusariose de l'épi sur blé tendre. Le précédent et le travail du sol sont
1850 déterminants, *Cultivar*, 506, 2001, 38-41.
- 1851 147. C.H.A. Snijders, J. Perkowski, Effects of head blight caused by *Fusarium culmorum*
1852 on toxin content and weight of wheat kernels, *Phytopathology*, 80, 1990e, 566-570.
- 1853 148. R.C. Sinha, M.E. Savard, Concentration of deoxynivalenol in single kernels and
1854 various tissues of wheat heads, *Canadian Journal of Plant Pathology*, 19, 1997, 8-12.
- 1855 149. M.E. Savard, R.C. Sinha, W.L. Seaman, G. Fedak, Sequential distribution of the
1856 mycotoxin deoxynivalenol in wheat spikes after inoculation with *Fusarium*
1857 *graminearum*, *Canadian Journal of Plant Pathology*, 22, 2000, 280-285.
- 1858 150. W.C.A. Gelderblom, K. Jaskiewicz, W.F.O. Marasas, P.G. Thiel, R.M. Horak, R.M.
1859 Vleggaar, N.P.J. Kriek, Fumonisin: novel mycotoxins with cancer-promoting activity
1860 produced by *Fusarium moniliforme*, *Applied and Environmental Microbiology*,
1861 54(7), 1988, 1806-1811.
- 1862 151. J.C. Wolf, J.R. Lieberman, C.J. Mirocha, Inhibition of F-2 (Zearalenone) biosynthesis
1863 and perithecium production of *Fusarium roseum 'Graminearum'*, *Phytopathology*, 62,
1864 1972, 937-939.
- 1865 152. J.C. Wolf, C.J. Mirocha, Regulations of sexual reproduction in *Gibberella zeae*
1866 (*Fusarium roseum 'Graminearum'*) by F-2 (Zearalenone), *Canadian Journal of*
1867 *Microbiology*, 19, 1973, 725-734.
- 1868 153. J.C. Wolf, C.J. Mirocha, Control of sexual reproduction in *Gibberella zeae (Fusarium*
1869 *roseum 'Graminearum')*, *Applied and Environmental Microbiology*, 33(3), 1977, 546-
1870 550.
- 1871 154. R. Ioos, A. Faure, Fusariose des épis, mycotoxines et traitements fongicides, *Phytoma*,
1872 539, 2001, 56-63.
- 1873 155. C.M. Placinta, J.P.F. D'Mello, A.M.C. Macdonald, A review of worldwide
1874 contamination of cereal grains and animal feed with *Fusarium* mycotoxins, *Animal*
1875 *Feed Science and Technology*, 78, 1999, 21-37.
- 1876 156. A.W. Schaafsma, L. Tamburic-Ilincic, J.D. Miller, The effect of agronomic practice
1877 on the accumulation of deoxynivalenol (DON) in winter wheat fields in Ontario,

- 1878 1996-97. In "National Fusarium Head Blight Forum. Chapter 1- Epidemiology and
 1879 Disease Management", Michigan State University (USA), 1998, pp. 7-9.
- 1880 157. E. Jenny, A. Hecker, P. Kessler, C. Kulling, H.R. Forrer, Cereal fusarioses: varietal
 1881 resistance and toxin contents [German] (Abstract), *Agrarforschung*, 7, 2000, 270-273.
- 1882 158. E. Young, Itinéraire cultural blé. Ce qui favorise les mycotoxines, *Cultivar*, 539, 2002,
 1883 52.
- 1884 159. C.J. Mirocha, H.K. Abbas, C.E. Windels, W. Xie, Variation in deoxynivalenol, 15-
 1885 acetyldeoxynivalenol, 3-acetyldeoxynivalenol and zearalenone production by
 1886 *Fusarium graminearum* isolates, *Applied and Environmental Microbiology*, 55(5),
 1887 1989, 1315-1316.
- 1888 160. J. Perkowski, A. Pavlova, A. Srobarova, J. Stachowiak, P. Golinski, Group B
 1889 trichothecenes biosynthesis in wheat cultivars after heads inoculation with *Fusarium*
 1890 *culmorum* isolates (abstract), *Biologia*, 57, 2002, 765-771.
- 1891 161. B. Bakan, C. Giraud-Deville, L. Pinson, D. Richard-Molard, E. Fournier, Y. Brygoo,
 1892 Identification by PCR of *Fusarium culmorum* strains producing large and small
 1893 amounts of deoxynivalenol, *Applied and Environmental Microbiology*, 68, 2002,
 1894 5472-5479.
- 1895 162. Y.Z. Jin, T. Yoshizawa, Relationship between the incidence of mycotoxigenic
 1896 *Gibberella zeae* (*Fusarium graminearum*) in crop fields and the occurrence of
 1897 trichothecenes in wheat [Japanese] (abstract), *Mycotoxins*, 40, 1994, 39-42.
- 1898 163. H. Hestbjerg, S. Elmholt, K.F. Nielsen, G. Felding, The ecology of toxin producing
 1899 fungi [Danish] (abstract). In "17th Danish Plant Protection Conference II, Site-specific
 1900 crop protection, decision support, pests and diseases, ear blight", Denmark, 2000.
- 1901 164. H. Hestbjerg, G. Felding, S. Elmholt, *Fusarium culmorum* infection of barley
 1902 seedlings: correlation between aggressiveness and deoxynivalenol content, *Journal of*
 1903 *Phytopathology*, 150, 2002, 308-312.
- 1904 165. P. Jennings, M. Coates, J.A. Turner, P. Nicholson, Distribution of
 1905 deoxynivalenol/nivalenol producing isolates of *Fusarium culmorum* and *F.*
 1906 *graminearum* in England and Wales (abstract), *Aspects of Applied Biology*, 2003.
- 1907 166. J.P. Carter, H.N. Rezanoor, D. Holden, A.E. Desjardins, R.D. Plattner, P. Nicholson,
 1908 Variation in pathogenicity associated with the genetic diversity of *Fusarium*
 1909 *graminearum*, *European Journal of Plant Pathology*, 108, 2002, 573-583.
- 1910 167. A. Mesterhazy, Breeding wheat for resistance to *Fusarium graminearum* and
 1911 *Fusarium culmorum*, *Z. Pflanzenzüchtg - Plant Breeding*, 91, 1983, 295-311.

- 1912 168. C. ShouXi, G. Kleijer, Effects of *Fusarium* metabolites on growth of callus and
1913 seedling in triticale (abstract), *Acta Prataculturae Sinica*, 10, 2001, 78-85.
- 1914 169. S. Marin, N. Magan, J. Serra, A.J. Ramos, R. Canela, V. Sanchis, Fumonisin B₁
1915 production and growth of *Fusarium moniliforme* and *Fusarium proliferatum* on maize,
1916 wheat, and barley grain, *Journal of Food Science*, 64(5), 1999, 921-924.
- 1917 170. W.L. Casale, L.P. Hart, Inhibition of protein synthesis in maize by trichothecene
1918 mycotoxins, Abstract, *Phytopathology*, 76, 1986, 1144.
- 1919 171. A.E. Desjardins, R.H. Proctor, G. Bai, S.P. McCormik, G. Shaner, G. Buechley, T.M.
1920 Hohn, Reduced virulence of trichothecene-nonproducing mutants of *Gibberella zeae*
1921 in wheat field tests, *Molecular Plant-Microbe Interactions*, 9(9), 1996, 775-781.
- 1922 172. L.F. Chen, Y.L. Song, Y.G. Xu, Variation in the concentrations of deoxynivalenol in
1923 the spikes of winter wheat infected by *Fusarium graminearum* Schw [Chinese]
1924 (abstract), *Acta Phytopathologica Sinica*, 26, 1996, 25-28.
- 1925 173. A. Mesterhazy, Role of deoxynivalenol in aggressiveness of *Fusarium graminearum*
1926 and *F. culmorum* and in resistance to *Fusarium* head blight, *European Journal of Plant*
1927 *Pathology*, 108, 2002, 675-684.
- 1928 174. R.H. Proctor, A.E. Desjardins, S.P. McCormick, R.D. Plattner, N.J. Alexander, D.W.
1929 Brown, Genetic analysis of the role of trichothecene and fumonisin mycotoxins in the
1930 virulence of *Fusarium*, *European Journal of Plant Pathology*, 108, 2002, 691-698.
- 1931 175. F. Eudes, A. Comeau, S. Rioux, J. Collin, Impact of trichothecenes on *Fusarium* head
1932 blight (*Fusarium graminearum*) development in spring wheat (*Triticum aestivum*),
1933 *Canadian Journal of Plant Pathology*, 23, 2001, 318-322.
- 1934 176. A.H. Teich, L. Shugar, A. Smid, Soft white winter wheat cultivar field-resistance to
1935 scab and deoxynivalenol accumulation, *Cereal Research Communications*, 15, 1987,
1936 109-114.
- 1937 177. C.H.A. Snijders, *Fusarium* head blight and mycotoxin contamination of wheat, a
1938 review, *Netherlands Journal of Plant Pathology*, 96, 1990b, 187-198.
- 1939 178. W. Liu, W. Langseth, H. Skinnes, O.N. Elen, L. Sundheim, Comparison of visual head
1940 blight ratings, seed infection levels, and deoxynivalenol production for assessment of
1941 resistance in cereals inoculated with *Fusarium culmorum*, *European Journal of Plant*
1942 *Pathology*, 103, 1997, 589-595.
- 1943 179. C. Lamper, J. Téren, T. Bartok, R. Komoroczy, A. Mesterhazy, F. Sagi, Predicting
1944 DON contamination in *Fusarium*-infected wheat grains via determination of the
1945 ergosterol content, *Cereal Research Communications*, 28(3), 2000, 337-344.

- 1946 180. J. Grabarkiewicz-Szczesna, E. Foremska, M. Kostecki, P. Golinski, J. Chelkowski,
 1947 Trichothecenes accumulation in kernels of corn inoculated with *Fusarium poae* (Peck)
 1948 Wollenw, Nahrung, 43, 1999, 330-332.
- 1949 181. J.F. Fourbet, L. Malmauret, T. Doré, P. Verger, "Impact du mode de conduite des
 1950 cultures sur la qualité agronomique et toxicologique du blé. Application à l'agriculture
 1951 biologique," Rep. No. 99-36, Convention MAP/INRA, 2001.
- 1952 182. S.L. Walker, S. Leath, W.M. Hagler, J.P. Murphy, Variation among isolates of
 1953 *Fusarium graminearum* associated with Fusarium head blight in north Carolina, Plant
 1954 Disease, 85(4), 2001, 404-410.
- 1955 183. B. Cahagnier, Les mycotoxines des céréales. In "Mycotoxines : un prochain enjeu de
 1956 sécurité alimentaire ?" (CERAAF, ed.), 2002, pp. 18-24.
- 1957 184. A. Devaux, La fusariose de l'épi du blé, Agriculture (Montréal), 44, 1988, 8 et 25.
- 1958 185. C.H.A. Snijders, The inheritance of resistance to head blight caused by *Fusarium*
 1959 *culmorum* in winter wheat, Euphytica, 50, 1990d, 11-18.
- 1960 186. Y.J. Yu, Monosomic analysis for scab resistance and yield components in wheat
 1961 cultivar Soo-mo 3, Cereal Research Communications, 10(3-4), 1982, 185-189.
- 1962 187. F.L. Kolb, G.H. Bai, G.J. Muehlbauer, J.A. Anderson, K.P. Smith, G. Fedak, Host
 1963 plant resistance genes for fusarium head blight : mapping and manipulation with
 1964 molecular markers, Crop Science, 41(3), 2001, 611-619.
- 1965 188. L. Gervais, F. Dedryver, J.Y. Morlais, V. Bodusseau, S. Negre, M. Bilous, C. Groos,
 1966 M. Trottet, Mapping of quantitative trait loci for field resistance to *Fusarium* head
 1967 blight in an European winter wheat, Theoretical and Applied Genetics, 106, 2003,
 1968 961-970.
- 1969 189. Z.Z. Liu, Z.Y. Wang, Improved scab resistance in China : sources of resistance and
 1970 problems. Cité par Ban, 2000 et Rudd, 2001. In "Wheat for the non-traditional warm
 1971 areas, international conference" (S. DA, ed.), CIMMYT publication, Mexico, 1991,
 1972 pp. 178-188.
- 1973 190. C. Pritsch, C.P. Vance, W.R. Bushnell, D.A. Somers, T.M. Hohn, G.J. Muehlbauer,
 1974 Systemic expression of defense response genes in wheat spikes as a response to
 1975 *Fusarium graminearum* infection, Physiological and Molecular Plant Pathology, 58,
 1976 2001, 1-12.
- 1977 191. C.A. Urrea, R.D. Horsley, B.J. Steffenson, P.B. Schwarz, Heritability of *Fusarium*
 1978 head blight resistance and deoxynivalenol accumulation from barley accession CIho
 1979 4196, Crop Science, 42, 2002, 1404-1408.

- 1980 192. A. Mesterhazy, T. Bartok, C.G. Mirocha, R. Komoroczy, Nature of wheat resistance
1981 to *Fusarium* head blight and the role of deoxynivalenol for breeding, *Plant Breeding*,
1982 118, 1999, 97-110.
- 1983 193. L.P. Hart, J.J. Pestka, M.T. Liu, Effect of kernel development and wet periods on
1984 production of deoxynivalenol in wheat infected with *Gibberella zeae*, *Phytopathology*,
1985 74, 1984, 1415-1418.
- 1986 194. H. Buerstmayr, M. Lemmens, G. Patschka, H. Grausgruber, P. Ruckenbauer, Head
1987 blight (*Fusarium* spp.) resistance of wheat cultivars registered in Australia. *Cité par*
1988 *Ruckenbauer, 2001, Die Bodenkultur, 47, 1996b, 183-190.*
- 1989 195. M. Nakagawa, Studies on ear-scab resistance of wheat plants. 2. genetical factors
1990 affecting on the inheritance of ear-scab disease of wheat plants. *Cité par Teich, 1989,*
1991 *Jap. J. Breeding, 5, 1955.*
- 1992 196. S. Hakimi, J. Liang, Y. Wu, C. Rosenberger, S. Castro, D. Shah, Gene therapy for
1993 plants. *Cité par Ban, 2000, Cereal Research Communications, 25, 1997, 745-747.*
- 1994 197. C. Pritsch, W.R. Bushnell, D.A. Somers, G.J. Muehlbauer, C.P. Vance, *Fusarium*
1995 *graminearum* infection on wheat spikes: early events. In "National Fusarium Head
1996 Blight Forum. Chapter 1- Epidemiology and Disease Management", Michigan State
1997 University (USA), 1998, pp. 5.
- 1998 198. W.P. Chen, P.D. Chen, D.J. Liu, R. Kynast, B. Friebe, R. Velazhahan, S.
1999 Muthukrishnan, B.S. Gill, Development of wheat scab symptoms is delayed in
2000 transgenic wheat plants that constitutively express a rice thaumatin-like protein gene,
2001 *Theoretical and Applied Genetics, 99, 1999, 755-760.*
- 2002 199. L.S. Dahleen, P.A. Okubara, A.E. Blechl, Transgenic approaches to combat *Fusarium*
2003 head blight in wheat and barley, *Crop Science, 41, 2001, 628-637.*
- 2004 200. T. Taira, T. Ohnuma, T. Yamagami, Y. Aso, M. Ishiguro, I. M., Antifungal activity of
2005 rye (*Secale cereale*) seed chitinases: the different binding manner of class I and class
2006 II chitinases to the fungal cell walls, *Bioscience Biotechnology and Biochemistry, 66,*
2007 *2002, 970-977.*
- 2008 201. G. Rebmann, F. Mauch, R. Dudler, Sequence of a wheat cDNA encoding a pathogen-
2009 induced thaumatin-like protein. *Cité par Dahleen, 2001, Plant Mol. Bio., 17, 1991,*
2010 *283-285.*
- 2011 202. A.I. Pekkarinen, B.L. Jones, Trypsin-like proteinase produced by *Fusarium culmorum*
2012 grown on grain proteins, *Journal of Agricultural and Food Chemistry, 50, 2002, 3849-*
2013 *3855.*

- 2014 203. A.I. Pekkarinen, T.H. Sarlin, A.T. Laitila, A.I. Haikara, B.L. Jones, Fusarium species
 2015 synthesize alkaline proteinases in infested barley, *Journal of Cereal Science*, 37,
 2016 2003a, 349-356.
- 2017 204. A.I. Pekkarinen, B.L. Jones, Purification and identification of barley (*Hordeum*
 2018 *vulgare* L.) proteins that inhibit the alkaline serine proteinases of *Fusarium culmorum*
 2019 (abstract), *Journal of Agricultural and Food Chemistry*, 51, 2003b, 1710-1717.
- 2020 205. M. Mohammadi, H. Kazemi, Changes in peroxidase and polyphenol oxidase activities
 2021 in susceptible and resistant wheat heads inoculated with *Fusarium graminearum* and
 2022 induced resistance, *Plant Science*, 162, 2002, 491-498.
- 2023 206. J. Duvick, Prospects for reducing fumonisin contamination of maize through genetic
 2024 modification (abstract), *Environmental Health Perspectives*, 109, 2001, 337-342.
- 2025 207. R.A.d. Maagd, D. Bosch, W. Stiekema, *Bacillus thuringiensis* toxin-mediated insect
 2026 resistance in plants, *Trends in Plant Science*, 4, 1999, 9-13.
- 2027 208. C.H. Zhong, D.J. Ellar, A. Bishop, C. Johnson, S.S. Lin, E.R. Hart, Characterization
 2028 of a *Bacillus thuringiensis* delta-endotoxin which is toxic to insects in three orders
 2029 (abstract), *Journal of Invertebrate Pathology*, 76, 2000, 131-139.
- 2030 209. R. Mitterbauer, G. Adam, *Saccharomyces cerevisiae* and *Arabidopsis thaliana*: Useful
 2031 model systems for the identification of molecular mechanisms involved in resistance
 2032 of plants to toxins, *European Journal of Plant Pathology*, 108, 2002, 699-703.
- 2033 210. E. Siranidou, Z. Kang, H. Buchenauer, Studies on symptom development, phenolic
 2034 compounds and morphological defence responses in wheat cultivars differing in
 2035 resistance to *Fusarium* head blight, *Journal of Phytopathology*, 150, 2001, 200-208.
- 2036 211. Anonyme, "Protection against trichothecene mycotoxins. *Cité par Wang and Miller,*
 2037 *1988 et Eudes, 1998,*" National Academy Press. Washington D.C. v, Washington D.C,
 2038 1983, pp. 227.
- 2039 212. Y. Castonguay, L. Couture, Epidémiologie de la contamination des grains de céréales
 2040 par les *Fusarium* spp., *Canadian Journal of Plant Pathology*, 5, 1983, 222-228.
- 2041 213. J.D. Miller, J.C. Young, H.L. Trenholm, *Fusarium* toxins in field corn. I- Time course
 2042 of fungal growth and production of deoxynivalenol and other mycotoxins, *Canadian*
 2043 *Journal of Botany*, 61, 1983, 3080-3087.
- 2044 214. J.D. Miller, J.C. Young, D.R. Sampson, Deoxynivalenol and *Fusarium* head blight
 2045 resistance in spring cereals, *Journal of Phytopathology*, 113, 1985a, 359-367.
- 2046 215. J.D. Miller, J.C. Young, Deoxynivalenol in an experimental *Fusarium graminearum*
 2047 infection of wheat, *Canadian Journal of Plant Pathology*, 7, 1985b, 132-134.

- 2048 216. J.D. Miller, P.G. Arnison, Degradation of deoxynivalenol by suspension cultures of
2049 the Fusarium head blight resistant wheat cultivar Frontana, Canadian Journal of Plant
2050 Pathology, 8, 1986, 147-150.
- 2051 217. R.W. Stack, Return of an old problem: Fusarium head blight of small grains, Plant
2052 Health Progress, 2000.
- 2053 218. X.Y. Li, Z.S. Wu, Studies on the relationship between choline concentration in
2054 flowering spikes and resistance to scab among bread wheat varieties. *Cité par Eudes,*
2055 *1998, Acta Agr. Sinica, 20, 1994, 176-185.*
- 2056 219. Y.P. Wang, Y.Q. Liu, L. Shi, N.S. Pan, Z.L. Chen, SOD activity of wheat varieties
2057 with different resistance to scab (abstract), Acta Phytophysiologica Sinica, 19(4),
2058 1993, 353-358.
- 2059 220. P.A. Okubara, A.E. Blechl, S.P. McCormick, N.J. Alexander, R. Dill-Macky, T.M.
2060 Hohn, Engineering deoxynivalenol metabolism in wheat through the expression of a
2061 fungal trichothecene acetyltransferase gene, Theoretical and Applied Genetics, 106,
2062 2002, 74-83.
- 2063 221. R.W. Stack, J.D. Rasmussen, Ferulic acid as a factor in resistance in wheat to
2064 Fusarium head blight, Phytopathology, 85, 1995, 1046.
- 2065 222. J.G. Horsfall, A.E. Dimond, Interactions of tissue sugar, growth substances and
2066 disease susceptibility, Zeitschrift fuer Pflanzenkrankheiten, 64(7-10), 1957, 415-421.
- 2067 223. T. Aoki, K. O'Donnell, Morphological and molecular characterization of *Fusarium*
2068 *pseudograminearum* sp. nov., formerly recognized as the Group 1 population of
2069 *Fusarium graminearum*, Mycologia, 91(4), 1999a, 597-609.
- 2070 224. T. Aoki, K. O'Donnell, Morphological characterization of *Gibberella coronicola* sp.
2071 nov., obtained through mating experiments of *Fusarium pseudograminearum*,
2072 Mycoscience, 40, 1999b, 443-453.
- 2073 225. A.H. Wearing, L.W. Burgess, Water potential and the saprophytic growth of *Fusarium*
2074 *roseum "graminearum"*, Soil Biology and Biochemistry, 11, 1979, 661-667.
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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].
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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) <i>F. roseum</i>	Light	Requires low intensity of UV light (300-320<390nm)		[83], with conflicting results obtained by [35].	
			Depends on the strain		[35,71]	
			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 None if T°<16°C or T°>36°C		[83]	
		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)
	<i>F. culmorum</i>	Pressure	-10 to -28 bars			
		Temperature	20 to 30°C			[23] (on osmotically adjusted agar)
	<i>F. graminearum</i> group 1 (soil) <i>F. avenaceum</i>	Pressure	-8 to -14 bars			[97] (on osmotically adjusted agar)
		Pressure	Max between -15 and -60 bars	-15 bars		[97] (on osmotically adjusted agar)
	<i>F. roseum</i>	Pressure	Max between -15 and -60 bars	-15 bars		[97] (on osmotically adjusted agar)
Temperature		> 10°C	25 to 30°C		[16]	
<i>M. nivale</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]	

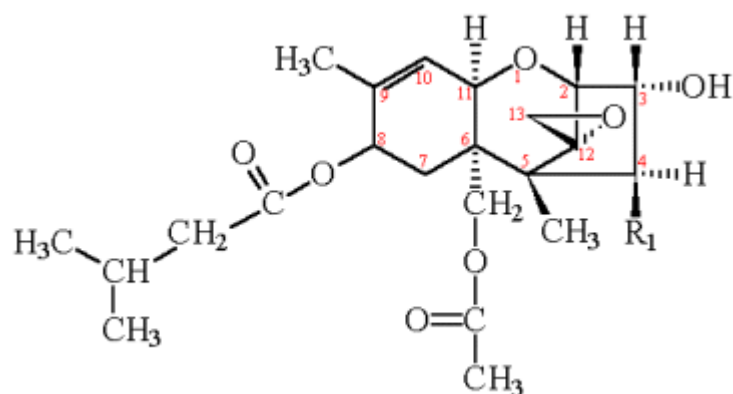
2101 Table 2: Spectrum of infection of weeds by *Fusarium* species. M: Monocotyledonous, g: grasses, D
2102 Dicotyledonous.

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]

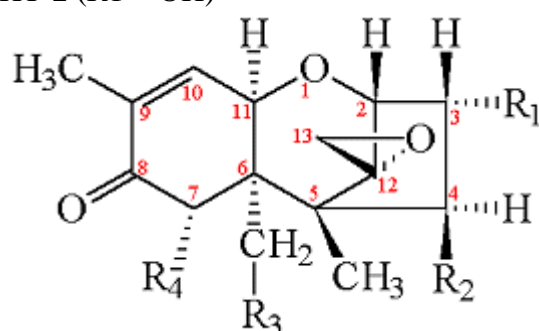
2103

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

2105 **Trichothecenes**



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

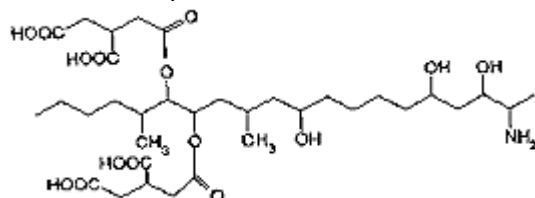


Type B trichothecenes: DON (R1 = OH, R2 = H, R3 = OH, R4 = OH)
NIV (R1 = OH, R2 = OH, R3 = OH, R4 = OH)

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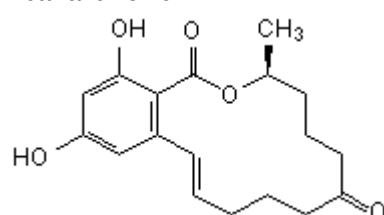
Fumonisin B₁



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Zearalenone



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