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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Review — *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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Abstract

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

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

Contents

Introduction

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

The consequences of head blight contamination

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

Since the 1990's, interest in food health and safety has increased. For cereal products, one of the most important elements of alimentary risk is the possibility of mycotoxin accumulation on or in the grains [33-35]. Mycotoxins are the products of fungal secondary metabolism [34,36], which occurs when growth stops altogether or slows markedly [37]. These toxins result from adaptation of the growth of the fungus to stressful situations [37]. Head blight is the principal means by which mycotoxins develop in the grains before harvest. The mycotoxins produced in crops suffering from head blight are produced by fungi of the genus *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

1- Epidemiological study of the disease

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 chlamydospores, 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, chlamydospores 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].

203

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.

231

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.

236

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. monoliforme* [67,80] and *F. graminearum*

289   [75] have shown that the release of ascospores is not directly linked to rainfall. Indeed,

290   ascospore release peaks one to four days after rainfall [67,75] of at least 5 mm or a relative

291   humidity exceeding 80% [67]. Paulitz found that a shower of rain in the evening did not

292   inhibit spore release but that heavier rain (at least 5 mm), a relative humidity exceeding 80%

293   continuously throughout the day or the alternation of rain and high humidity inhibited the

294   release of ascospores during the night. This result contrasts with another study showing the

295   release of ascospores during the night following a day on which 8 mm of rain fell [81]. The

296   correlation between spore release and humidity suggests that the maturation of perithecia

297   requires a certain level of humidity. This would account for the results of Fernando *et al.*

298   (2000)[75], who observed four periods of spore release over a period of 20 to 30 days.

299

300   The process of ascospore release may be described as follows [71]: after a period of drought

301   (air with low water vapour content), free water (rain or heavy dew) triggers the release of

302   ascospores. Paulitz (1996)[67] speculated that the increase in relative humidity during the

303   evening after a dry day might increase the turgor pressure of the asci, which contain a vacuole

304   at high osmotic pressure [82]. This high osmotic pressure is generated by the accumulation of

305   mannitol and the flux of potassium and calcium ions [66]. Calcium ions are also involved in

306   signalling for ascospore discharge [66]. This increase in pressure triggers the release of

307   ascospores, which are expelled from the perithecia in a gelatinous substance, which then dries

308   out, releasing the ascospores into dry air. This would account for the finding that a trap placed

309   above the canopy captures several *M. nivale* ascospores in dry conditions and that the

310   concentration of these spores increases by a factor of 20 in the 10 minutes following the

311   application of a fine simulated dew [71]. A similar phenomenon has also been observed for

312   the ascospores of *F. graminearum* [6]. Similarly, in control conditions, the maximal release of

313   *F. graminearum* ascospores is regulated by dehydration of the perithecia in non-saturated

314   humidity conditions [83]. Furthermore, the time at which the spores are released is strongly

315   correlated with the increase in humidity following the decrease in temperature that occurs at

316   the end of the afternoon [67]. Paulitz observed that ascospore release began before the leaves

317   became humid at the base of the canopy (which occurred at 22-24 h), towards 17 or 18 h for

318   temperatures varying between 11 and 30 °C and values of relative humidity of between 60

319   and 95%. These results have been confirmed by the observation, in control conditions, that

320   the maximal release of *F. graminearum* spores occurs at temperatures between 11 and 23 °C,

321   with 16 °C the optimal temperature [83].

322

323   After temperature and humidity, the next most important environmental factor affecting

324   ascospore release is light. Light is required for the production and maturation of perithecia

325   [68] and ascospores [83]. In contrast, the process of ascospore release does not directly

326   require light, as it has been observed during the night: 4 to 5 h before sunrise [67]. This result

327   has been corroborated by studies in control conditions, which also showed that light had no

328   effect on ascospore release [66,68].

329

330   Finally, the periodicity of aerial dispersion has been studied. The release of conidia is not

331   periodic, whereas that of ascospores is, particularly in *F. graminearum* [75]. Indeed, Fernando

332   *et al.* detected no ascospores in the air between 12 and 16 h, whereas ascospore density was

333   maximal between 20 h and 08 h (1.5 times higher than at other periods of the day). Other

334   authors have reported that ascospore density peaks between 21 h and 08 h [84]. Similarly,

335   Paulitz reported that spore release often occurs between 18 h and early morning (04 h – 08 h),

336   mostly before midnight, with a peak at around 23 h [67]. For *F. graminearum*, the maximum

337   ascospore concentration observed is of the order of 4333 ascospores/mm^3 in one hour. Paulitz

338   also reported spores of other species of *Fusarium* to be continuously present in the air, but

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

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

1-5.   Infection and colonisation of the ear

In wheat, the most critical period corresponds to the moment at which the degree of resistance to primary infection and to the propagation of fungal hyphae in the plant is lowest. This period has been defined differently by different authors, but the first half of the grain-filling period is the most critical. Several authors have identified ear emergence [64,85] or the mid-grain-filling stage [47] as the most critical, but most authors consider anthesis to be the most susceptible to attack in wheat [12,16,64,86,87], with susceptibility decreasing strongly after the start of the dough stage [12,16,47,64,86,87]. Elimination of the male organs from wheat ears decreases the frequency of infection by *F. graminearum* [47,86]. Similarly, sterile wheat lines are less susceptible to head blight than fertile lines [88]. This, together with extensive colonisation of the anthers by this fungus [86] indicates that the growth of *F. graminearum* is stimulated in these structures, suggesting that entry into the anthers during anthesis has major consequences for the grain (degradation). Two substances — choline chloride and betaine hydrochloride — are much more concentrated in the anthers than elsewhere [89]. These substances favour the extension of conidial hyphae, but not the germination of spores of *F. avenaceum, F. culmorum* and *F. graminacearum* [89]. Once the spores have germinated, the propagation of the hyphae is therefore more strongly favoured in the flower parts than in the other organs. However, the difference in susceptibility of different stages is probably due to the fact that the critical receptivity peak observed (degree of resistance to primary infection and propagation of fungal hyphae) is dependent on many factors. These factors include the wheat cultivar [12,47,50,90], air temperature and humidity [12,47] at the critical stage for the plant and, before this stage, development of the inoculum, and the stage of maturity of the plant at the time of hyphal penetration [12]. The testa (seed coat) is more resistant to penetration when the grains are mature [12]. However, this finding remains open to debate 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

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

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

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

2-2. Effects of soil tillage

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

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

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

507   [100,110] and the quantity of spores available for dispersal [109]. This hypothesis seems to be
508   validated by the observation that there are fewer *Fusarium culmorum* propagules if the
509   stubble from the preceding crop is burnt than if it is buried [109]. However, an opposite effect
510   on the disease is observed. Thus, in years in which there are high levels of *Fusarium* infection
511   at the base of the stem, disease levels have been found to be higher if the stubble from the
512   previous crop was burnt than if it was incorporated into the soil [109]. A similar result was
513   previously reported for eyespot [111]. Similarly, the supplementary incorporation of infected
514   stubble not originating from the preceding crop decreases the incidence of *Fusarium* attacks
515   resulting in wheat foot rot to levels below those observed in soils in which the density of
516   residues has not been modified [112]. However, these results may be explained if stubble
517   limits conidium dispersal by splashing, favours growth of the canopy [111], or modifies the
518   infection process. In Yarham's study [111], the addition of stubble did not affect leaf area
519   index, photosynthetic potential or the number of leaves. It also resulted in no decrease in the
520   density of wheat or weeds and had no effect on the size of the plants. All these factors might
521   have facilitated spore dispersal. In the infectious process, four aspects may be affected by the
522   presence or absence of stubble. Firstly, the resistance of the plants may be affected by
523   increases in the amount of silica (SiO_2) 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 *Pseudocercosporella*
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* ascopspores 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, diacetoxy-scirpenol (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

3-2. Effect of soil tillage

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

3-3. Effect of the preceding crop

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

3-4. Effect of mineral nutrition

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

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

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

4- Relationship between the disease and toxin production

911

912

4-1. Toxin production by the fungi

913

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

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

Non-specific resistance

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

Heritability of the resistance

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

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

Resistance to head blight in wheat is horizontal (non-specific) and thus protects against all 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 dexoynivalenol
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

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

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

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

Conclusion

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

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

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

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

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

The role played by canopy density, in terms of crop plant and weed densities, and in the development of epidemics of the disease remains to be determined. Weeds may carry *Fusarium*, but is their role limited to that of an intermediate host (during splashing) or do they act as a source of inoculum? If they serve as a source of inoculum, how many spores are 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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References

1. D. Atanasoff, *Fusarium* blight (scab) of wheat and other cereals, Journal of Agricultural Research, 20(1), 1920, 1-32.

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

3. Y.Z. Wang, J.D. Miller, Effects of *Fusarium graminearum* metabolites on wheat tissue in relation to *Fusarium* head blight resistance, Journal of Phytopathology, 122, 1988, 118-125.

4. J.D. Miller, R. Greenhalgh, Y. Wang, M. Lu, Trichothecene chemotypes of three *Fusarium* species, Mycologia, 83, 1991, 121-130.

5. A. Mesterhazy, Types and components of resistance to Fusarium head blight of wheat, Plant Breeding, 114, 1995, 377-386.

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

7. M. McMullen, R. Jones, D. Gallenberg, Scab of wheat and barley: a re-emerging disease of devastating impact, Plant Disease, 81, 1997, 1340-1348.

8. A. Bottalico, *Fusarium* diseases of cereals: species complex and related mycotoxin profiles, in Europe, Journal of Plant Pathology, 80(2), 1998, 85-103.

9. J.C. Rudd, R.D. Horsley, A.L. McKendry, E.M. Elias, Host plant resistance genes for fusarium head blight : sources, mechanisms, and utility in conventional breeding systems, Crop Science, 41(3), 2001, 620-627.

10. A. Bottalico, G. Perrone, Toxigenic *Fusarium* species and mycotoxins associated with head blight in small-grain cereals in Europe, European Journal of Plant Pathology, 108, 2002, 611-624.

11. F.T. Bennett, Fusarium species on British cereals, Annals of Applied Biology, 20(2), 1933, 272-290.

12. G.W. Pugh, H. Johann, J.G. Dickson, Factors affecting infection of wheat heads by *Gibberella saubinetii*, Journal of Agricultural Research, 46, 1933, 771-797.

1480   13.   P. Ruckenbauer, 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, Nertherland 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   (Suzhau and Naujing, 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 - 6ème 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 - 6ème 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 contôler la qualité sanitaire des céréales : détection des
1536   champignons potentiellement producteurs d'une famille de mycotoxines, les
1537   trichothécènes. *In* "AFPP - 6$^{\text{ème}}$ 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$^{\text{ème}}$ 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. Francl, 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. Cromey, 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 Phytopatholigica 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   inoculaiton 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 suceptible 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 *Pseudocercosporella 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 barbley, 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. Ruckenbauer, 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, Nertherland 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 rhizophere 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. Knozak, 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, Fumonisins: novel mycotoxins with cancer-promoting activity

1860   producted 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 occurence 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 B_1
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 Phytopatholigica 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 (*Titicum 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, Nertherland 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 bligh in north Carolinia, 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 cerevisae* 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.

216. J.D. Miller, P.G. Arnison, Degradation of deoxynivalenol by suspension cultures of the Fusarium head blight resistant wheat cultivar Frontana, Canadian Journal of Plant Pathology, 8, 1986, 147-150.

217. R.W. Stack, Return of an old problem: Fusarium head blight of small grains, Plant Health Progress, 2000.

218. X.Y. Li, Z.S. Wu, Studies on the relationship between choline concentration in flowering spikes and resistance to scab among bread wheat varieties. *Cité par Eudes, 1998*, Acta Agr. Sinica, 20, 1994, 176-185.

219. Y.P. Wang, Y.Q. Liu, L. Shi, N.S. Pan, Z.L. Chen, SOD activity of wheat varieties with different resistance to scab (abstract), Acta Phytophysiologica Sinica, 19(4), 1993, 353-358.

220. P.A. Okubara, A.E. Blechl, S.P. McCormick, N.J. Alexander, R. Dill-Macky, T.M. Hohn, Engineering deoxynivalenol metabolism in wheat through the expression of a fungal trichothecene acetyltransferase gene, Theoretical and Applied Genetics, 106, 2002, 74-83.

221. R.W. Stack, J.D. Rasmussen, Ferulic acid as a factor in resistance in wheat to Fusarium head blight, Phytopathology, 85, 1995, 1046.

222. J.G. Horsfall, A.E. Dimond, Interactions of tissue sugar, growth substances and disease susceptibility, Zeitschrift fuer Pflanzenkrankheiten, 64(7-10), 1957, 415-421.

223. T. Aoki, K. O'Donnell, Morphological and molecular characterization of *Fusarium pseudograminearum* sp. nov., formerly recognized as the Group 1 population of *Fusarium graminearum*, Mycologia, 91(4), 1999a, 597-609.

224. T. Aoki, K. O'Donnell, Morphological characterization of *Gibberella coronicola* sp. nov., obtained through mating experiments of *Fusarium pseudograminearum*, Mycoscience, 40, 1999b, 443-453.

225. A.H. Wearing, L.W. Burgess, Water potential and the saprophytic growth of *Fusarium roseum "graminearum"*, Soil Biology and Biochemistry, 11, 1979, 661-667.

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	*F. graminearum* group 2 (aerial)	Pressure Humidity	> -9 bars > 94%RH		[75] (on maize grains)
		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 Pressure	20 to 30°C –10 to –28 bars		[23] (on osmotically adjusted agar)
	F. culmorum	Accelerated by alternate periods of rainfall and drought before ear emergence			[59]
		Temperature Pressure	20 to 30°C -8 to –14 bars		[23] (on osmotically adjusted agar)
		Pressure Temperature	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]
	M. nivale	Temperature	0 to 28-32°C	18-20°C	[11, 16]
Initiation of perithecia	*F. graminearum* group 2 (aerial) *F. roseum*	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 *F. graminearum* 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	*F. graminearum* group 2 (aerial)	Temperature Light	13 to 33°C UV light required	25-28°C	[2,16]
Production of spores	*M. nivale*	Temperature	< 16-18 °C	6 à 8 °C	[64]
	Fusarium	Temperature Humidity	Around 10°C Around 80% humidity		[64]
Production of macroconidia	*F. graminearum* 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 Pressure	20 to 30°C –10 to –28 bars		[23] (on osmotically adjusted agar)
	F. culmorum	Temperature Pressure	20 to 30°C -8 to –14 bars		
		Pressure	Max between -15 and –60 bars	-15 bars	[97] (on osmotically adjusted agar)
	F. graminearum group 1 (soil) *F. avenaceum*	Pressure	Max between -15 and –60 bars	-15 bars	[97] (on osmotically adjusted agar)
	F. roseum	Temperature	> 10°C	25 to 30°C	[16]
		Light Humidity	UV light required RH of about 100% Favoured by water stress		
	M. nivale	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			*Fusarium*	Site and form	Reference
Family	Latin name	Common name	Species		
Poaceae Mg	*Agropyron*	Wheatgrass	Not indicated	Not indicated	[85]
Liliaceae M	*Allium*	Garlic	*F. avenaceum, F. culmorum, F. graminearum*	Not indicated	[64]
Poaceae Mg	*Alopecurus myosuroides* Huds.	Blackgrass	*F. roseum*	In the seed	[48]
Poaceae Mg	*Avena fatua* L.	Wild oats	*F. roseum*	Lesion, spores on leaves and seeds	[48]
Chenopodiaceae D	*Beta vulgaris*	Wild beet	*F. culmorum*	Stem base	[62]
Cruciferae D	*Capsella bursa-pastoris* L.	Shepherd's purse	*F. avenaceum, F. culmorum, F. poae, F. sambucinum*	Stem base	[62]
Compositae D	*Cirsium arvense* L.	Thistle	*F. avenaceum*	Stem base	[62]
Poaceae Mg	*Dactylis* L.	Cocksfoot	*F. roseum*	Lesion on straw, stem base, increase with N	[14,48]
Poaceae Mg	*Echinochloa crus-galli* L.	Barnyardgrass	Not indicated	Not indicated	[85]
Poaceae Mg	*Festuca*	Fescue	*F. roseum*	Stem base, increase with N	[14]
Rubiaceae D	*Galium aparine* L.	Cleavers	*F. avenaceum, F. culmorum, F. poae*	Stem base	[62]
Poaceae Mg	*Lolium multiflorum* L.	Ryegrass	*F. roseum*	Lesion on straw, stem base, increase with N	[14,48]
Compositae D	*Matricaria spp.*	Mayweed	*F. avenaceum, F. culmorum, F. graminearum, F. poae, F. sambucinum*	Stem base	[62]
Ranunculaceae D	*Ranunculus acris*	Common buttercup	*F. avenaceum, F. culmorum, F. poae*	Stem base	[62]
Ranunculaceae D	*Ranonculus repens* L.	Creeping buttercup	*F. avenaceum, F. culmorum*	Stem base	[62]
Polygonaceae D	*Rumex obtusifolius*	Dock	*F. avenaceum, F. culmorum*	Stem base	[62]
Compositae D	*Senecio vulgaris* L.	Groundsel	*F. avenaceum, F. culmorum*	Stem base	[62]
Caryophyllaceae D	*Spergula arvensis* L.	Corn spurrey	*F. sambucinum*	Stem base	[62]
Carophyllaceae D	*Stellaria media* L.	Chickweed	*F. avenaceum*	Stem base	[62]
Fabaceae D	*Trifolium*	Clover/trefoil	*F. avenaceum, F. culmorum, F. graminearum*	Not indicated	[64]
Urticaceae D	*Urtica dioica* L.	Nettle	*F. avenaceum, F. culmorum, F. sambucinum*	Stem base	[62]
Scrophylariaceae D	*Veronica persica*	Speedwell	*F. graminearum*	Stem base	[62]
Violaceae D	*Viola arvensis* Murray	Field violet	*F. avenaceum, F. poae, F. culmorum, F. graminearum, F. sambucinum*	Stem base	[62]
Cryophyllaceae D	*Dianthus*	Carnation	*F. avenaceum, F. culmorum, F. poae, F. graminearum*	Not indicated	[64]

2103

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

2105   **Trichothecenes**
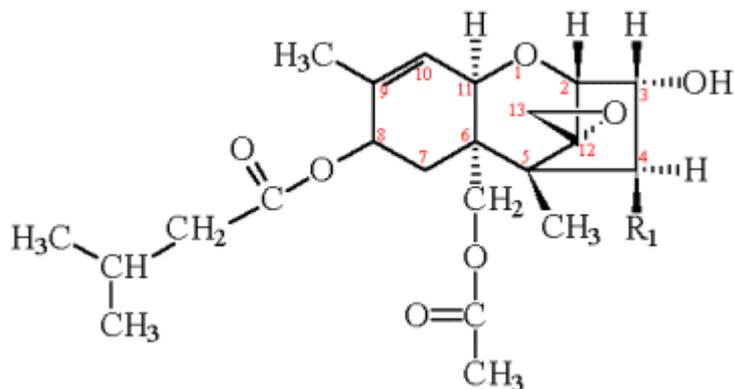
Type A trichothecenes: T-2 (R1 = OAc)
                         HT-2 (R1 = OH)
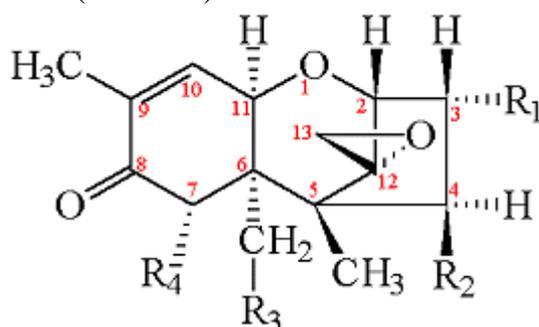
Type B trichothecenes: DON (R1 = OH, R2 = H, R3 = OH, R4 = OH)
                         NIV (R1 = OH, R2 = OH, R3 = OH, R4 = OH)

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2108   **Fumonisin B$_1$**
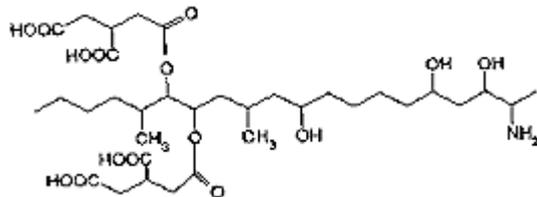
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2110   **Zearalenone**

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