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The relationship between wheat seed weight, infection by *Fusarium culmorum* or *Microdochium nivale*, germination and seedling disease

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Abstract

The distribution of seeds by weight for three lots of winter wheat cv. Avalon infected by *Fusarium culmorum* and three lots of winter wheat cv. Riband infected by *Microdochium nivale* was determined. The distribution of infected seeds within each seed lot was then determined by isolating *F. culmorum* from seeds on moist filter paper and *M. nivale* from seeds on potato dextrose agar. The distribution of *M. nivale* infected seeds between seeds of different weight was similar to that of the seed lot as a whole, whereas the distribution of *F. culmorum* was greater in light seeds than heavy seeds. The percentage germination of infected seeds decreased with seed weight. A similar situation was found with respect to seedling emergence in compost for *F. culmorum* infected seeds. However, with *M. nivale* infection, similar numbers of seedlings emerged from both light and heavy infected seeds. Seed treatment with guazatine increased seedling emergence for both light and heavy seed infected by *M. nivale*. However, seedling emergence from *F. culmorum* infected seed was poor even following treatment with guazatine. Poor emergence was most evident from light seed.

Introduction

Fusarium ear blight (FEB) of wheat, which is a frequent if sporadic disease of wheat throughout the world, is caused by four main pathogens; *Fusarium avenaceum* (Corad ex Fr) Sacc., *Fusarium culmorum* (W.G. Smith) Sacc., *Fusarium graminearum* Schwabe and *Microdochium nivale* (Fr.) Samuels and Hallet (formally *Fusarium nivale*) (Parry et al., 1995). Yield reductions in wheat following naturally occurring epidemics of FEB have been observed in many countries with estimated losses in the order of 15–50% (Mains et al., 1929; Kükedi, 1972; Tusa et al., 1981; Chaudhary et al., 1990). More precise data relating to the effects of FEB on wheat yield have been obtained from inoculated trials. For example, after inoculating wheat ears in Switzerland with *Fusarium culmorum* and *Microdochium nivale*, Häni (1981) observed grain yield reductions of 60% and 15% respectively.

There are two components of yield that could possibly be affected by FEB; the number of grains per ear and the weight of individual grains. A reduction in yield, associated with a decrease in the weight of individual grains was demonstrated by Snijders and Perkowski (1990) in field trials in the Netherlands following inoculation of wheat ears with *F. culmorum* conidia. The mean loss of yield of 9.7% was almost exclusively attributable to a reduction in grain weight. Wong et al. (1992) observed that ear infection of wheat caused by *F. graminearum* in field plots in Canada reduced the thousand grain weight (TGW) of seed by up to 36%. Most recently, Doohan et al. (1999) observed a linear relationship between spikelet infection caused by *F. culmorum* and the TGW of wheat grown in the glasshouse. Bechtel et al.(1985), working with wheat seed from the USA, demonstrated that *F. graminearum* seed infection, identified by the presence of the mycotoxin deoxynivalenol (DON) and ergosterol, was most

frequent in the lighter seeds. In addition, Nakagawa and Yamaguchi (1989) showed that the majority of seeds naturally infected by what they described as *Fusarium roseum* were lighter than non-infected seeds in a sample of wheat seed in Japan.

In contrast to *Fusarium* spp., little has been published on the effect of *M. nivale* on the components of grain yield. In a review of *Fusarium* diseases of cereals in Western Europe, Cassini (1981) proposed that reduced yield following *M. nivale* ear infection was caused solely by a reduction in TGW. However, Cassini did not offer any evidence for this claim.

In addition to reducing yield, infected seeds can provide a source of inoculum for Fusarium seedling blight which may result in a significant reduction in seedling emergence if infected seed is sown in unfavourable conditions (Hare et al., 1995). However, the relationship between seed infection, seed weight, which itself can affect seedling growth (Lafond and Baker, 1986), and Fusarium seedling blight is not fully understood.

The aims of the work reported here were to investigate the distribution by weight of *F. culmorum* and *M. nivale* infected seeds in selected seed lots of winter wheat and to investigate the relationship between infection, seed weight, seedling emergence and disease control by a fungicide seed treatment.

Materials and methods

Seed weight frequency distributions. The three seed lots of winter wheat cv. Avalon (lot Nos. 1–3, Table 1) naturally infected by *F. culmorum* were obtained from a FEB field experiment and the three seed lots of cv. Riband (Nos. 4–6, Table 1) naturally infected by *M. nivale* were selected from a range of commercially produced samples owing to the lack of other seed-borne pathogens present. The frequency distributions of seed weight for the six seed lots were produced by taking 700 seeds at random from each seed lot, weighing each seed and then placing them in one of nine weight categories; 1–9, 10–20, 21–30, 31–40, 41–50, 51–60, 61–70, 71–80 and 81–90 mg.

Seed weight frequency distributions for infected seeds. The number of seeds infected by *F. culmorum* or *M. nivale* was determined by isolating the pathogen from each of the 700 weighed seeds from each seed lot. Seeds were placed in a solution of sodium hypochlorite (NaOCl) (1% available chlorine) for 3 min. Seeds were then washed three times in sterile distilled water, placed on sterile blotting paper and then dried in a flow of sterile air. To optimize the recovery and identification of either *F. culmorum* or *M. nivale* (Limonard, 1968) the surface sterilized seeds from seed lot Nos. 1 to 3 were transferred aseptically onto sterilized filter paper moistened with 2 ml of sterile distilled water and seeds from lots 4 to 6 onto PDA supplemented with the antibiotics streptomycin sulphate (100 $\mu g\,ml^{-1}$), neomycin sulphate (50 $\mu g\,ml^{-1}$) and chloramphenicol (50 $\mu g\,ml^{-1}$). Five seeds were placed in each Petri dish. The Petri dishes were sealed with 'Parafilm' and placed in unilluminated incubators at 21 °C for moist filter paper and 15 °C for PDA. After 14 days, the resultant fungal colonies were examined and identified on the basis of their colony characteristics and spore morphology. The number of seeds in each weight category producing either *F. culmorum* or *M. nivale* colonies was counted. This allowed the production of seed weight frequency distributions for infected seeds. Data from seed lot Nos. 1 to 3 were tested for normality using Shapiro–Wilk and differences between means using ANOVA.

Germination of seeds on moist filter paper and PDA. The germination of winter wheat seeds was assessed after 14 days on either the filter paper or the PDA. Seeds were deemed to have germinated successfully if they had reached growth stage (GS) 09 (Tottman and Broad, 1987). The number of *F. culmorum* and *M. nivale* infected and non-infected seeds which had germinated was counted and expressed as a percentage of the total in each weight category.

Seedling emergence in compost. Heavy and light seeds either with or without a fungicide seed treatment Rappor (50 $g\,l^{-1}$ guazatine, DowElanco) from five of the six seed lots (Tables 2 and 3) were sown in one of two controlled environment experiments. Two experiments were performed to provide conditions conducive to either *F. culmorum* (seed lot Nos. 1 and 3) or *M. nivale* (seed lot Nos. 4–6).

For both seed lot Nos. 1 and 3, 160 heavy seeds and 160 light seeds were selected visually by seed size at random. Seeds in each of the heavy and light samples were weighed individually and the mean seed weight for each sample calculated (Table 2). Seeds from each sample were surface sterilized in NaOCl and divided into two equal sub-samples of 80 seeds at random. One of the sub-samples was treated with Rappor at the rate of 200 ml 100 kg^{-1} of seed by mixing the seed and seed

Table 1. Seed lot, pathogen and percentage infection, geographic origin and year harvested

Seed lot No.	Winter wheat cultivar	Pathogen (% infection)	Geographic origin of seed	Year of harvest
1	Avalon	*F. culmorum* (1)	Edgmond, Shropshire, UK	1996
2	Avalon	*F. culmorum* (23)	Edgmond, Shropshire, UK	1996
3	Avalon	*F. culmorum* (36)	Edgmond, Shropshire, UK	1996
4	Riband	*M. nivale* (19)	Scotland, UK	1993
5	Riband	*M. nivale* (27)	Eriswell, Suffolk, UK	1993
6	Riband	*M. nivale* (65)	Scunthorpe, Humberside, UK	1993

Table 2. Emergence of seedlings from light and heavy seed treated with or without Rappor (50 g l^{-1} guazatine) from two seed lots winter wheat cv. Avalon seed lots 1 and 3 with 1% and 39% *F. culmorum* infection respectively

Seed lot No.	Mean seed weight (mg)	Seed treatment: Rappor	Arcsine transformed % seedling emergence	Seedling emergence (%)
1	20.3	Yes	70.2	88
1	20.2	No	80.1	95
1	58.4	Yes	90.0	100
1	58.6	No	87.1	99
3	16.2	Yes	46.3	52
3	16.1	No	39.2	40
3	59.1	Yes	80.1	95
3	59.2	No	57.0	70
		SEM for treatments	3.22	
		D of F	32	
		CV	10.5	

Table 3. Emergence of seedlings from light and heavy seed treated with or without Rappor (50 g l^{-1} guazatine) from three seed lots winter wheat cv. Riband seed lots 4–6 with 19%, 27% and 65% *M. nivale* infection respectively

Seed lot No.	Mean seed weight (mg)	Seed treatment: Rappor	Arcsine transformed % seedling emergence	Seedling emergence (%)
4	21.3	Yes	80.0	97
4	21.7	No	69.0	87
4	55.8	Yes	83.0	98
4	54.1	No	59.6	74
5	15.1	Yes	70.8	89
5	15.0	No	66.0	83
5	49.9	Yes	60.0	75
5	49.3	No	49.9	58
6	18.2	Yes	77.0	95
6	18.8	No	33.1	30
6	58.7	Yes	87.1	99
6	58.1	No	29.8	25
		SEM for treatments	4.34	
		D of F	48	
		CV	15.2	

treatment together in 125 ml glass screw cap bottles until the liquid seed treatment had been evenly distributed over the seeds' surfaces. The other sub-sample was left untreated.

Sixteen seeds from each sub-sample were planted to a depth of 2 cm into 230 g of sterilized soil-based compost (John Innes No. 2) in plastic pots (70 × 70 × 80 mm). Before planting the compost was passed through a 5 mm sieve and then sterilized at 121 °C for 60 min three times with 24 h between sterilizations. Five replicates of each treatment were used giving a total of 40 pots which were placed in a controlled environment cabinet (Conviron, Controlled Environments Ltd, Winnipeg, Manitoba, Canada) according to a fully randomized design. Environmental conditions were set to a constant 20 °C for 16 h of light and 65% relative humidity. The pots were regularly watered to a mean soil moisture content of 22% (Hare and Parry, 1996).

Microdochium nivale infected seeds from seed lot Nos. 4 to 6 were treated similarly but placed in environmental conditions set to 8 °C for 8 h of light and 6 °C for 16 h of darkness and 75% relative humidity.

In each experiment the figure for final emergence was taken when no further seedlings had emerged on five successive days. Percentage seedling emergence data were arcsine transformed and analysed by ANOVA with seed lot, seed weight and treatment with guazatine as factors.

Results

Seed weight frequency distributions. The distribution of seed weight for seed lot No. 1, with little *F. culmorum* infection (1%), was normal ($P < 0.063$) and mesokurtic (0.002) (Figure 1a). The distributions of lot Nos. 2 and 3 were near normal ($P < 0.146$) and normal ($P < 0.001$) respectively but were more platykurtic (-0.352 and -0.972 respectively) than lot No. 1 (Figure 1b and c). Seed lot No. 1 had a mean seed weight of 44.6 mg (± 0.39, $n = 700$) which was significantly greater ($P < 0.0001$, LSD = 1.3, $P = 0.05$) than both lot Nos. 2 (41.0 mg, ± 0.42, $n = 700$) and 3 (35 mg, ± 0.57, $n = 700$). The percentage of infected seeds in the lighter (≤ 40 mg) weight categories increased from 0.8% to 23.8% to 43.4% for seed lot Nos. 1–3 with 1%, 23% and 36% *F. culmorum* infection respectively.

The frequency distributions of seed weight for seed lot Nos. 4–6 were similar to those for seed from each lot with *M. nivale* infection. The number of infected seeds

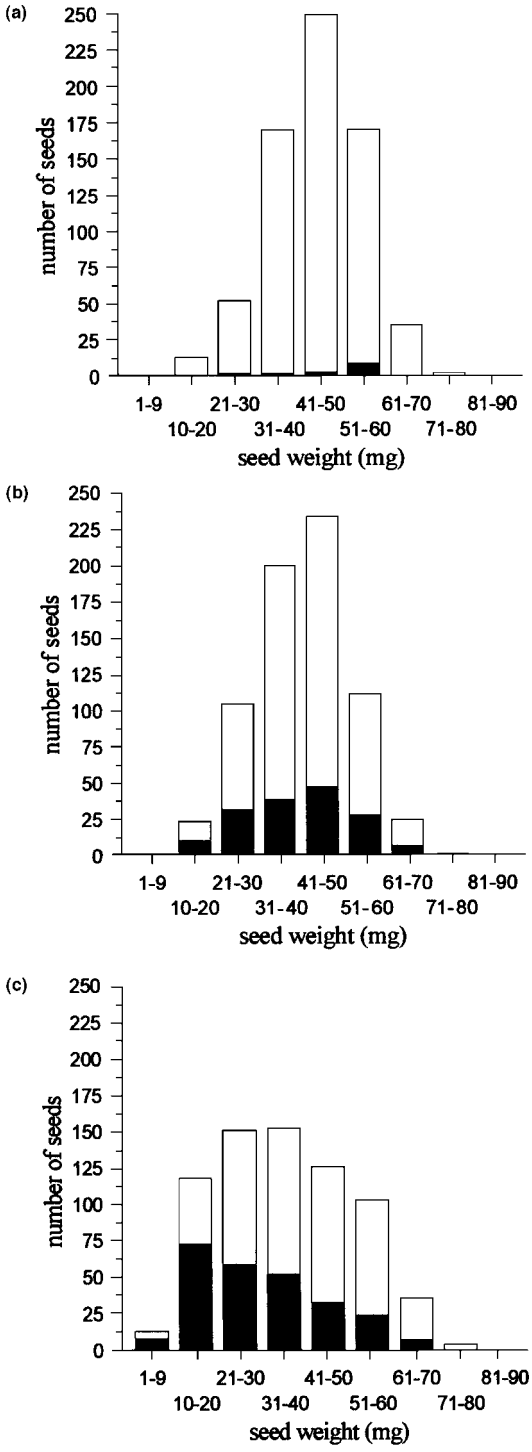

Figure 1. Frequency distributions by weight for winter wheat seed cv. Avalon. (a) Seed lot No. 1, (b) seed lot No. 2 and (c) seed lot No. 3 (Table 1). The distribution of seeds in the 700 seed sample (□) and that for *F. culmorum* infected seeds (■) are given.

occurring in each of the weight categories was similar in proportion to the total number of seeds (Figure 2a–c). There was no clear evidence that *M. nivale* infected seeds were either predominantly heavy or light seeds.

Seed germination. The gemination of seeds from seed lots infected by *F. culmorum* declined from 98% to 74% as the percentage of infected seeds increased from 1% to 36%. Fewer seeds germinated in the lighter than the heavier seed weight categories for seed lot No. 3 with the greatest *F. culmorum* infection (36%), and poor germination was correlated ($R^2 = 0.92$, $P < 0.01$) with seed infection (Figure 3a). The germination of seeds from the *M. nivale* infected seed lots also decreased from 82% to 48% as the percentage of infected seeds increased from 19% to 65%. Fewer light seeds germinated than heavy seeds for seed lot No. 6 with 65% *M. nivale* infection, but there was no apparent correlation ($R^2 = 0.03$, $P > 0.05$) between reduced germination and *M. nivale* infection across weight categories (Figure 3b).

Seedling emergence in compost. Factorial ANOVA of seedling emergence data for both *F. culmorum* infected and *M. nivale* infected seed showed significant two-way interactions between seed lot, seed weight and guazatine treatment. For this reason only means for individual treatments are presented in Tables 2 and 3.

Seedling emergence from seed lot No. 1 with little *F. culmorum* infection was good ($\geq 88\%$) for both light and heavy seeds, with and without seed treatment. *F. culmorum* infection had a significant ($P < 0.05$) effect on the emergence of untreated seedlings from seed lot No. 3. Fewer seedlings emerged from light untreated seeds than heavy untreated seeds whilst treatment with guazatine significantly ($P < 0.05$) increased seedling emergence in both light and heavy seeds although the percentage of seedlings produced from light treated seeds (52%) was significantly ($P < 0.05$) lower than that from heavy treated seeds (95%) (Table 2).

Seedling emergence decreased as *M. nivale* infection increased from 19% to 65% in untreated seed (seed lot Nos. 4–6). Seed treatment with guazatine had a significant effect ($P < 0.05$) on seedling emergence for all *M. nivale*-infected seed lots increasing the percentage of emerged seedlings from both heavy and light seed most markedly in seed lot No. 6 from 25% to 99% for heavy seed and from 30% to 95% for light seed (Table 3).

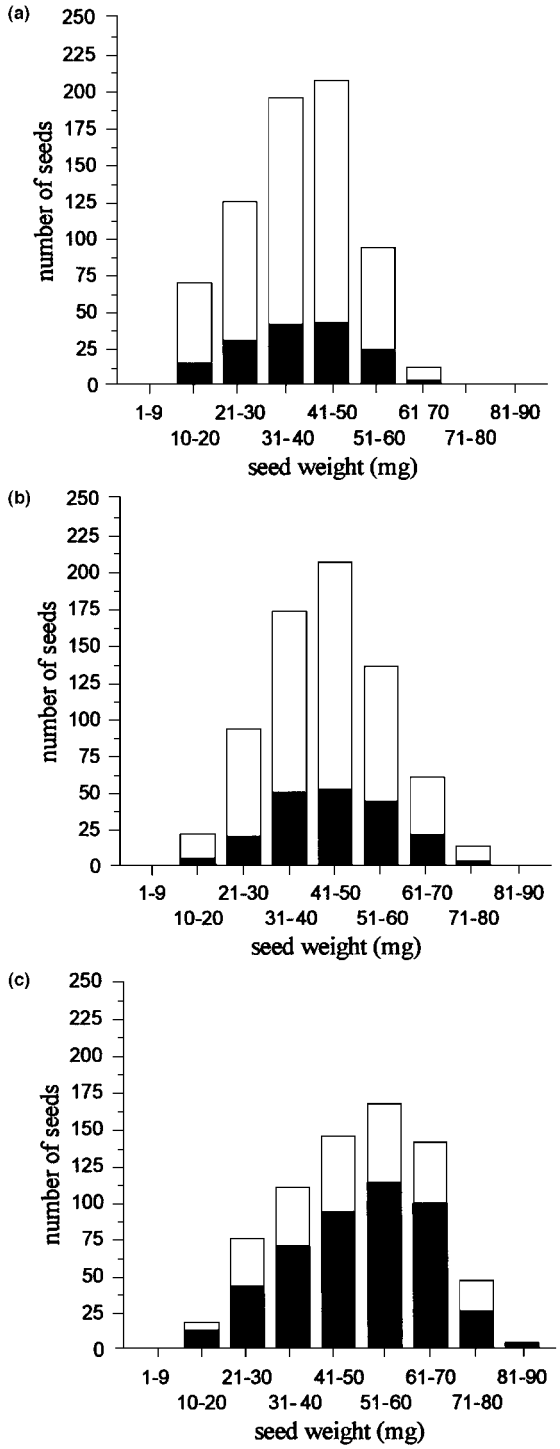

Figure 2. Frequency distributions by weight for winter wheat seed cv. Riband. (a) Seed lot No. 4, (b) seed lot No. 5 and (c) seed lot No. 6 (Table 1). The distribution of seeds in the 700 seed sample (□) and that for *M. nivale* infected seeds (■) are given.

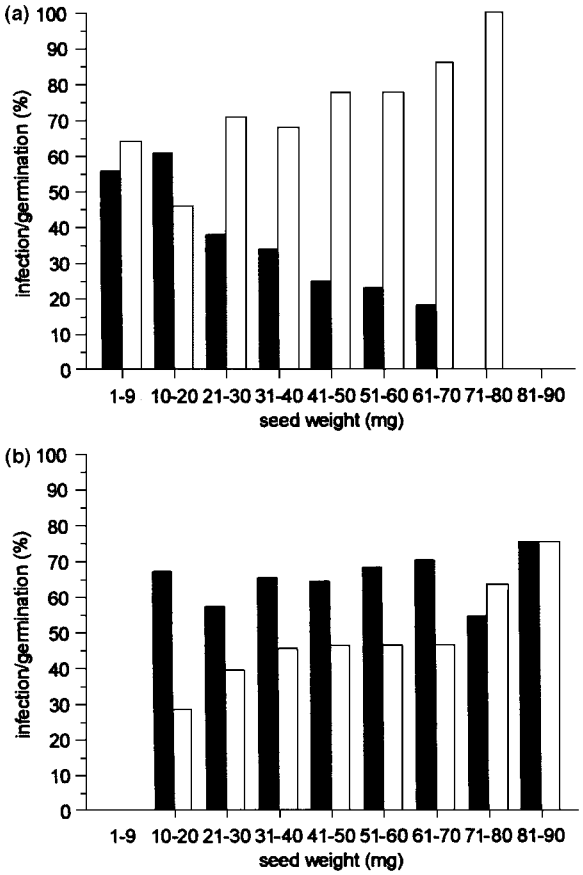

Figure 3. Percentage infection (■) and seed germination (□) for each seed weight category for seed lot No. 3 with 36% *F. culmorum* infection (a) and seed lot No. 6 with 65% *M. nivale* infection (b).

Discussion

Previous workers have highlighted the fact that within seed lots of wheat *Fusarium* spp. infected seeds are lighter than non-infected seeds (Bechtel et al., 1985; Nakagawa and Yamaguchi, 1989). This appears to be in conflict with the observations of Perkowski and Chelkowski (1991), working in Poland, who showed that the majority of *Fusarium* spp. infected wheat seeds, as identified by DON content, were the same size as non-infected seeds. However, there is evidence from the seed weight distributions of *F. culmorum* infected seeds reported in this study that both sets of workers may be correct in that the proportion of light ($\leq$ 40 mg) infected seeds increased with increasing overall percentage infection. Therefore, at low percentage infections infected seeds may be distributed evenly by weight in agreement with the observations of Perkowski and Chelkowski (1991) and at high percentage infections there may be a greater proportion of infected light seeds which would support the observations of Bechtel et al. (1985) and Nakagawa and Yamaguchi (1989).

It is often assumed, although there is little published evidence, that, like *Fusarium* spp., the *M. nivale* infected component of a seed lot is comprised mainly of smaller, lighter seeds. However, in this study, small light seeds were as likely to be infected by *M. nivale* as large heavy seeds, suggesting that this is not the case. This evidence may at first appear to contradict Cassini's previously stated assumption that reduced yield following FEB caused by *M. nivale* was attributable to a reduction in TGW. However, as no uninfected seed lots were available from the same field crops as the infected seed used in this study, comparisons between infected and uninfected seed lots are not possible. It is therefore not known if TGW has been reduced or not. In addition, as the *M. nivale*-infected seed lots used in this study were not sourced from a single crop it would not be valid to directly associate the observed difference in TGW between the seed lots with the percentage of *M. nivale*-infected seeds. The possible reduction in TGW following FEB caused by *M. nivale* may be a consequence amongst other factors of reduced grain filling owing to infection of the rachis and impediment of nutrient translocation to the grain in a similar fashion to that suggested by Doohan et al. (1998) with respect to bleaching of glumes following rachis infection rather than as a direct consequence of seed infection on the weight of individual grains.

Wheat seed infected by *M. nivale* is often associated with reduced germination (Rennie et al., 1990) and reduced emergence (Humphreys et al., 1995). In the work reported in this study the relationship between *M. nivale* infection, seed weight and wheat seed germination was different than the relationship between *M. nivale* infection, seed weight and seedling emergence, in that fewer light seeds germinated on PDA than emerged in compost. More heavy infected seeds germinated on PDA than light seeds, although more seedlings emerged in soil from light infected seeds than from heavy ones. The nutrient status of the growing medium was demonstrated by Shen (1940) to have an effect on the pre-emergence death of wheat seedlings. Fewer seedlings were killed by *F. culmorum* when grown in washed sand without nutrients than when

nutrients were added. The effect of nutrient status of the growth medium may go some way to explain the differences observed between the PDA and soil experiments. The germination tests were performed on a nutrient rich medium (PDA), whilst seedling emergence was assessed in a soil-based compost, presumed to be of lower nutrient status. The high nutrient medium in the germination experiment may have favoured the growth of the fungus. With a large supply of nutrients in the PDA, small light seeds could be colonized rapidly by the fungus. However, in a soil-based compost with a much reduced nutrient status, the fungus would be more reliant on the seed as a food source. Heavy seeds with large endosperms may provide more nutrients for fungal growth and subsequent seedling attack than small light seeds. In addition, it was noted that seedlings from small light wheat seeds emerged more quickly than from larger heavy seeds, on average reaching mean emergence 1.4 days earlier and possibly escaping pre-emergence disease symptoms (Hare and Parry, 1996).

The germination of seeds and the emergence of seedlings from the seed lots infected by *F. culmorum* were both lower for lighter seeds. This reduced germination and emergence was associated with *F. culmorum* infection, which was proportionally greater in lighter seeds. The emergence of seedlings from heavy seeds from seed lot No. 3 (heavily infected by *F. culmorum)* was significantly greater than light seeds and to some extent supports the findings of Nakagawa and Yamaguchi (1989) who showed that the 'cleaning' of naturally infected seed lots was possible by the removal of smaller seed. However, the addition of a fungicide seed treatment such as guazatine may still be required for seed to produce an acceptable number of seedlings, although it did not give good control of the disease in all situations. Where seeds were infected by *M. nivale* or where heavy seeds from the *F. culmorum* infected seed were sown, increased emergence was observed after fungicide treatment. Treatment with guazatine was, however, unable to control pre-emergence seedling death in light seeds from the *F. culmorum* infected seed lot. It is not clear from the work reported here whether guazatine was unable to control *F. culmorum* in light seeds or if the light infected seeds, owing to infection, were not viable as a germination test on treated seed was not performed. In addition, a seed viability test such as a tetrazolium test (ISTA, 1985) was not performed but the germination of *F. culmorum* infected seeds was reduced, suggesting that a reduction in seed viability owing to infection may have occurred. The presence of other fungal pathogens in seed may well affect the above relationships, but as no other pathogens were observed in the seed lots used this hypothesis was not tested.

The work presented in this study was based on a small sample of seed lots. However, it is clear that in agreement with previous work, light seeds are the main *F. culmorum* infected component of a seed lot and that removal of these lighter infected seeds will result in increased emergence. However, the addition of a fungicide seed treatment may also be required to achieve good seedling emergence. No such relationship between seed size, infection, fungicide treatment and seedling emergence was seen for *M. nivale*-infected seed, as no evidence for increased emergence following the selection of large seed was seen. For *M. nivale*-infected seed, treatment with a fungicide seed treatment appears to be the most effective option to increase seedling emergence irrespective of seed weight selection.

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