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ORIGINAL PAPER

Effect of the mycotoxin deoxynivalenol on grain aphid *Sitobion avenae* and its parasitic wasp *Aphidius ervi* through food chain contamination

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Abstract We investigated the influence of the *Fusarium* mycotoxin deoxynivalenol on the English grain aphid *Sitobion avenae* (Hemiptera: Aphididae) and its parasitic wasp *Aphidius ervi* (Hymenoptera: Braconidae) using in vitro laboratory experiments. In this tritrophic interaction, deoxynivalenol caused lethal (declined survival) and sublethal (prolonged nymphal development and reduced reproduction) effects on *S. avenae* aphids and consequentially led to a decreased production of parasitoid offspring resulting from parasitized deoxynivalenol-contaminated aphids. This paper highlights that the presence of mycotoxins should be considered in environmental risk assessment tests because they may alter the efficiency of biological control agents such as parasitoids through food chain contamination.

Keywords Cereal aphids · Deoxynivalenol · Food chain contamination · Parasitic wasps · Tritrophic interactions

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Introduction

The trichothecene deoxynivalenol (DON) is a mycotoxin produced by fungal species of the genus *Fusarium* that causes Fusarium head blight disease on cereals (Bottalico and Perrone 2002; Goswami and Kistler 2004). DON can be translocated inside the plant through xylem vessels and phloem sieve tubes (Kang and Buchenauer 1999). Concentrations of DON found in wheat samples from fields all over Flanders (Belgium) fluctuate around 0.1–10 mg kg^{-1} (Audenaert et al. 2009; Isebaert et al. 2009; Landschoot et al. 2013). DON has been notorious because it provokes acute and chronic disease symptoms in humans and animals that consume contaminated grains (Bennett and Klich 2003). Its toxic effects range from diarrhea, vomiting, gastro-intestinal inflammation, necrosis of the intestinal tract, the bone marrow and the lymphoid tissues. It causes inhibition of mitochondrial function and has effects on cell division and membrane integrity and induces apoptosis (Pestka 2010). Finally, it also inhibits protein, DNA and RNA synthesis (Rocha et al. 2005).

To date, little research has been done regarding the influence of trichothecenes on insects, even though it is an interesting topic to explore because trichothecenes can pose a threat for insects feeding on contaminated plant tissue and alter insect–plant interactions. It was found that trichodermin and other 12,13-epoxytrichothecenes have larvicidal activity against the mosquito *Aedes aegypti* (Grove and Hosken 1975). Deoxynivalenol and especially nivalenol have toxic effects on lepidopteran *Spodoptera frugiperda* cells (Fornelli et al. 2004). Diacetoxyscirpenol and neosolaniol are potent antifeedants against larvae of *Galleria mellonella* (Mule et al. 1992) and a novel isoquinoline type pigment from *F. moniliforme* showed larvicidal activity against *A. aegypti* and *Anopheles stephensi* (Pradeep et al. 2015).

Also the impact of mycotoxins on the survival of insect natural enemies, developing in herbivorous insects that feed on mycotoxin-contaminated plants, is not well explored. On a higher trophic level, natural enemies such as parasitic wasps developing in contaminated herbivores, can be directly exposed to these secondary metabolites or indirectly be affected by the reduced growth of the host (Bukovinszky et al. 2012; Gols 2014). Endoparasitoids can be especially vulnerable to mycotoxins because they develop for a period of time inside the mycotoxin-contaminated host. It is already known that plant secondary metabolites can pose a threat to natural enemies (Barbosa et al. 1991; Campbell and Duffey 1981; Harvey et al. 2007; Ode et al. 2004; Roth et al. 1997), but also mycotoxins are important hazards for natural enemies that should not be neglected nor underestimated. The effect of fungus-infected plants on adult female parasitoid behavior and development has been investigated (Bultman et al. 2009; Cardoza et al. 2003; Harri et al. 2008; van Nouhuys and Laine 2008). Harri et al. (2009) investigated life-history traits of the parasitoid *Aphidius ervi* when it was exposed to the endophyte-tolerant aphid *Metopolophium festucae* feeding from *Lolium perenne* infected with the mycotoxin-producing endophyte *Neotyphodium lolii*. The presence of endophytes significantly increased the development time of *A. ervi*. The authors concluded that extended parasitoid development should ultimately reduce the population growth of *A. ervi* and thus endophyte presence may represent an advantage for endophyte-tolerant aphid species. Still, to our knowledge, research remains scarce on the impact of mycotoxins against parasitoids that develop inside herbivorous insects feeding directly from those mycotoxins.

To fill this knowledge gap, we performed a case study investigating the influence of the mycotoxin DON on the phloem-feeding cereal aphids *Sitobion avenae* Fab. (Hemiptera: Aphididae). Both the mycotoxin-producing *Fusarium* spp. and the aphid *S. avenae* reside on wheat ears during anthesis, the critical fungal infection period. This can increase the chances that the cereal aphids come into contact with DON when feeding from the DON-contaminated phloem. Moreover, we examined the effect of DON-contaminated *S. avenae* on aphid parasitism by the parasitic wasp *A. ervi* Hal. (Hymenoptera: Braconidae), one of the most important parasitic wasps of *S. avenae* aphids in European cereal fields (Al Dobai et al. 1999; Barczak et al. 2014; Tomanovic et al. 2008).

Materials and methods

Insects and deoxynivalenol

A laboratory stock culture of *S. avenae* aphids was maintained on wheat seedlings at constant temperature of 22 °C, 60 % relative humidity and a photoperiod of 16 h light, stimulating parthenogenesis (De Zutter et al. 2012).

Sitobion avenae aphid mummies containing *A. ervi* were acquired from Biobest (Westerlo, Belgium). Newly emerged parasitoids were sexed and pairs consisting of one female and one male were put individually in Petri dishes to allow mating overnight in order to obtain naive females (i.e., females that have never laid an egg before) (Joseph et al. 2011; Pan and Liu 2014). All experiments were conducted in a climate chamber at 22 ± 2 °C, 60 % relative humidity and a photoperiod of 16 h light.

The mycotoxin deoxynivalenol (DON) (purity >99 %) was kindly provided by M. Lemmens (BOKU, Vienna, Austria). A stock solution (1000 mg L^{-1}) was prepared in sterile water and stored at −20 °C.

Experimental setup to examine the lethal effects of deoxynivalenol on *S. avenae* and *A. ervi*

Aphids were dietary exposed to DON by using an aphid feeding apparatus as described in Sadeghi et al. (2009) that contained 200 µL of artificial diet based on formulation A of Prosser and Douglas (1992) amended with DON to a final concentration of 0.25, 1, 3 and 5 mg L^{-1} DON or sterile water (0 mg L^{-1} DON). During the experiment, the diet was changed every other day and amended with the different concentrations of the persistent molecule DON. Adult *S. avenae* aphids were randomly selected from the stock culture and put on wheat seedlings to produce neonates (nymphal stage 1). The age of these neonates was between 0 and 24 h (day 0). Five neonates were transported from the seedlings to each aphid feeding apparatus. At day 4 the aphids were exposed to one naive *A. ervi* female per aphid feeding apparatus. At this time the aphids were in the second or third nymphal stage. Subsequently, aphids remained in the feeding apparatus until mummification. The number of mummies was counted daily. Each mummy was then put in a small Petri dish in order to evaluate the time until emergence of the parasitoid progeny. In total, 60, 30, 45, 45 and 30 aphids divided into groups of five individuals per aphid feeding apparatus were used as starting population in this experiment and exposed to 0, 0.25, 1, 3 and 5 mg L^{-1} DON, respectively. During the experiment, the percentage of aphid populations was calculated at different stages: % surviving aphids in the DON treatment at the moment of exposure to the parasitoid female and % surviving aphids at the beginning of mummification. Moreover, the percentages of aphids that turned into mummies (% mummification) and newly emerged parasitoids (% successful parasitism) were calculated.

Experimental design to examine sublethal effects of deoxynivalenol on *S. avenae* aphids

S. avenae neonates were exposed to DON in a final concentration of 0.25 or 1 mg L^{-1} DON or sterile water (0 mg L^{-1} DON) using aphid feeding apparatus as described above. Aphids were examined daily to examine the nymphal development and mortality. The presence of exuvia was used for the determination of a molt. To examine reproduction, the progeny of surviving females was counted daily and carefully removed using a fine brush. Progeny of females who died during nymphal development was considered zero. The intrinsic rate of increase (r_m) (Wyatt and White 1977) was calculated by following formula: $r_m = [0.738 \ \ln(\mathrm{Md})]/D$, with Md = numbers of nymphs produced by one female during its whole adult life (D). In total, ten aphids divided into ten aphid feeding apparatus (one per apparatus) were monitored daily for each concentration of DON (0, 0.25 and 1 mg L^{-1} DON).

Dual-choice assay to test preference of *A. ervi* for control and DON-contaminated aphids

In the preference test, naive female *A. ervi* were given the choice between aphids fed with DON and control aphids. A repeat consisted of two aphids in a small Petri dish (3.5 cm diameter), one fed with 3 mg L^{-1} DON and one control aphid (fed with 0 mg L^{-1} DON). Both aphids were synchronized to the second or third nymphal stage (Pan and Liu 2014). In the Petri dish, one naive female was introduced and allowed to choose between the DON-contaminated aphid and the control aphid. The female was monitored for a maximum of 10 min. The aphid that was first stabbed by the female and the time until this first stab (i.e., parasitoid touching the aphid with its ovipositor) occurred was recorded. Females that required more than 10 min to stab an aphid were labeled as 'no choice' (Daza-Bustamante et al. 2003). In total, the choice of 60 females was tested.

Statistical analyses

Data were analyzed using SPSS Statistics 22. Statistical differences of (sub)lethal effects by different DON concentrations against *S. avenae* and *A. ervi* (Fig. 1; Table 2) and the duration of *A. ervi* development (Table 4) were analyzed using a nonparametric Kruskal–Wallis test. In case of significant differences between the treatments, nonparametric Mann–Whitney tests were used to compare two treatments. Data in Fig. 1 were analyzed using one-sided Mann–Whitney tests corrected with the Benjamini and Hochberg False Discovery Rate (B&H FDR) at a level of 0.05 for controlling the type I error rate (Benjamini and Hochberg 2000). All tests were conducted with a significance level of $\alpha = 0.05$.

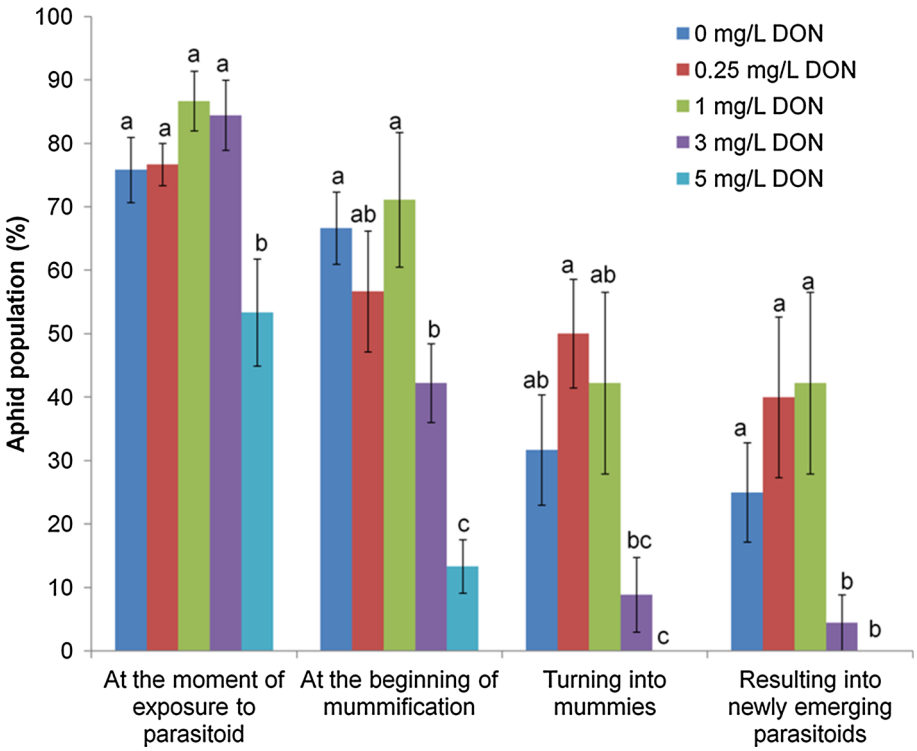

Fig. 1 The percentage of aphid populations (mean ± SE) at different stages: % surviving deoxynivalenol (DON) at the moment of exposure to the parasitoid female, % surviving at the beginning of mummification, % aphids becoming mummies and % aphids that produced newly formed parasitoid progeny. Different letters represent significant differences ($P < 0.05$) between treatments with different concentrations of DON using one-sided Mann–Whitney tests corrected with B&H FDR. Starting populations (100 %) consisted of 60, 30, 45, 45 and 30 aphids divided into groups of five individuals per aphid feeding apparatus exposed to 0, 0.25, 1, 3 and 5 mg L^{-1} DON, respectively

Table 1 *P* values according to one-sided Mann–Whitney tests associated with Fig. 1

P values	At the moment of exposure to parasitoid	At the beginning of mummification	Turning into mummies	Resulting into newly emerging parasitoids
1–2	0.456	0.384	0.090	0.145
1–3	0.075	0.071	0.316	0.232
1–4	0.135	**0.008**	0.027	**0.019**
1–5	**0.017**	**0.001**	**0.007**	**0.013**
2–3	0.061	0.076	0.382	0.476
2–4	0.135	0.098	**0.002**	**0.004**
2–5	**0.012**	**0.004**	**0.001**	**0.004**
3–4	0.406	**0.013**	0.039	**0.018**
3–5	**0.003**	**0.004**	**0.017**	**0.017**
4–5	**0.006**	**0.002**	0.116	0.207

The concentrations of 0, 0.25, 1, 3 and 5 mg L^{-1} DON are represented as numbers 1 to 5, respectively. *P* values in bold are significant after B&H FDR correction

Results

(Sub)lethal effects of deoxynivalenol on *S. avenae* and *A. ervi*

In this experiment *S. avenae* aphids were dietary exposed to five different concentrations of DON (0, 0.25, 1, 3 and 5 mg L^{-1} DON). The lethal effects of DON were measured by calculating the percentage of surviving *S. avenae*, and for *A. ervi* by calculating the percentage of aphids that turned into mummies and resulted into newly emerged parasitoid progeny. When the aphids that were feeding on the different concentrations of DON were exposed to the female parasitoid, a negative effect of 5 mg L^{-1} DON on the survival of *S. avenae* was found (Fig. 1; Table 1). The surviving aphid population feeding on 5 mg L^{-1} DON was significantly lower than the other treatments. Treatment of the aphids with 3 mg L^{-1} DON also caused a decrease in the aphid population but at a later time (when mummification started).

Sublethal effects of DON were measured by investigating the aphids nymphal development and reproduction while feeding on 0, 0.25 and 1 mg L^{-1} DON (Tables 2, 3). The duration of the last three nymphal stages (days) of aphids dietary exposed to 1 mg L^{-1} DON was significantly longer ($P < 0.05$) compared to 0 or 0.25 mg L^{-1} DON. When aphids were fed with 0, 0.25 and 1 mg L^{-1} DON, Md (i.e., the number of nymphs produced per female during its whole adult life), D (i.e., the whole adult life of the aphid in days) and r_m (i.e., the aphid population intrinsic rate of increase) were calculated. Md, *D* and r_m showed no significant differences between 0 and 0.25 mg L^{-1}, but both treatments were significantly higher ($P < 0.05$) than 1 mg L^{-1} DON, except for r_m where the effect by 0.25 and 1 mg L^{-1} DON was not significantly different ($P = 0.057$).

Table 4 presents the duration of *A. ervi* development in days inside *S. avenae* aphids contaminated with different concentrations of DON (0, 0.25, 1 and 3 mg L^{-1} DON). No significant differences were found between treatments 0, 0.25, 1 and 3 mg L^{-1} DON ($P = 0.349$ and 0.340 for oviposition until mummification and from mummification until emergence, respectively, according to Kruskal–Wallis tests). No mummies were found for the 5 mg L^{-1} DON treatment. Aphids feeding on 3 mg L^{-1} DON produced significantly fewer mummies compared to aphids feeding on 0.25 mg L^{-1} DON (Fig. 1; Table 1). Significantly less parasitoid progeny emerged from aphids fed on 3 mg L^{-1} DON compared to the lower concentrations tested.

Preference of parasitic wasp to attack control and DON-contaminated *S. avenae* aphids

In our binary-choice experiment, female *A. ervi* did not discriminate between DON-contaminated and control aphids. Out of a total of 60 wasps, 21 stabbed the control aphid first, 21 stabbed the DON-contaminated aphid first, and 18 were recorded as 'no choice'. The mean time (±SE) needed by females to make a choice was not significantly different between both treatments (2.9 ± 0.5 min for control aphids and 3.3 ± 0.6 min for DON-contaminated aphids).

Discussion

Survival, nymphal development and reproduction of *S. avenae* aphids were negatively influenced by DON. In our experiment, female *A. ervi* did not discriminate between control and DON-contaminated aphids, but successful parasitism of the aphids by *A. ervi* (i.e., emergence of

Table 2 Sublethal effects of different concentrations of deoxynivalenol (DON, mg L^{-1}) on *S. avenae* aphids nymphal development and reproduction

DON	Duration of nymphal development (days)				Reproduction		
	N1	N2	N3	N4	Md	*D*	r_m
0	1.1 ± 0.1^a	1.4 ± 0.2^a	1.4 ± 0.4^a	1.9 ± 0.1^a	16.6 ± 4.6^a	12.8 ± 3.6^a	0.108 ± 0.033^a
0.25	1.0 ± 0.1^a	1.8 ± 0.2^a	1.6 ± 0.2^a	1.9 ± 0.1^a	14.3 ± 3.3^a	14.6 ± 3.4^a	0.076 ± 0.017^{ab}
1	1.5 ± 0.3^a	2.6 ± 0.2^b	3.0 ± 0.4^b	3.5 ± 0.5^b	0.9 ± 0.7^b	1.7 ± 1.5^b	0.035 ± 0.026^b

N1–N4: aphid nymphal stage 1–4, Md: number of nymphs produced by females during their whole adult life, *D* whole adult life of the aphid in days, r_m: aphid population intrinsic rate of increase. Data are presented as mean ± SE. Levels of significance between treatments (0, 0.25 and 1 mg L^{-1} DON) were derived by comparing two treatments by using Mann–Whitney tests. Different letters indicate significant differences between treatments ($P < 0.05$)

Table 3 *P* values according to Mann–Whitney tests associated with Table 2

P values	Duration of nymphal development (days)				Reproduction		
	N1	N2	N3	N4	Md	*D*	r_m
1–2	0.563	0.195	0.677	1	0.818	0.646	0.566
1–3	0.194	**0.008**	**0.033**	**0.015**	**0.008**	**0.016**	**0.039**
2–3	0.114	**0.042**	**0.016**	**0.015**	**0.008**	**0.008**	0.057

P values in bold are smaller than the significance level of 0.05

The concentrations of 0, 0.25 and 1 mg L^{-1} DON are represented as numbers 1–3, respectively

Table 4 Duration of *A. ervi* development (mean ± SE) inside *S. avenae* aphids contaminated with different concentrations of deoxynivalenol (DON, mg L^{-1})

DON	Oviposition → mummification (days)	Mummification → emergence (days)
0	8.1 ± 0.3	5.2 ± 0.2
0.25	8.6 ± 0.3	5.5 ± 0.3
1	8.4 ± 0.3	5.4 ± 0.2
3	8.0 ± 0.7	4.5 ± 0.5

Data are presented as mean ± SE. There were no significant differences between treatments according to Kruskal–Wallis tests

parasitoid progeny) decreased. This could mean that the parasitoid eggs or larvae were directly or indirectly susceptible to DON or DON derivatives inside the aphid body. Directly, DON causes inhibition of mitochondrial function, has effects on cell division and membrane integrity, induces apoptosis and inhibits protein, DNA and RNA synthesis (Pestka 2010; Rocha et al. 2005). Possibly these toxic effects can lead to mortality of the parasitic wasp larvae inside the aphid body. Indirectly, it is possible that DON-contaminated aphids were feeding less efficiently than control ones (indeed, their nymphal development is slower), leaving the parasitoid larvae with fewer nutritional resources for its development. Nevertheless, there was a negative effect on the parasitoid developing inside DON-contaminated aphids. This could suggest that DON causes a decreased efficiency of biological control agents such as parasitoids through food chain contamination.

In our experiment we used concentrations of DON ranging from 0 to 5 mg L^{-1}. DON is a water soluble compound and is translocated in ears of cereal crops through phloem sieve tubes (Kang and Buchenauer 1999). Concentrations found in wheat samples from fields all over Flanders (Belgium) fluctuate around 0.1–10 mg kg^{-1} DON (Audenaert et al. 2009; Isebaert et al. 2009; Landschoot et al. 2013). These concentrations are present in grains at harvest and thus do not necessarily represent concentrations in the phloem during the time period that aphids are feeding from the phloem sieve tubes. Exact concentrations of DON and its derivatives in the phloem and dosages inside aphids that impair both the aphids and their parasitoids are unknown. Maybe DON can accumulate inside the aphids, but it could also be converted into other less toxic compounds such as DON-3-glucoside. These molecular mechanisms provide an interesting future research topic. To cope with xenobiotics, *S. avenae* has several enzyme families which show increased activities upon contamination. Cytochrome P450 monooxygenases (P450, phase I metabolic enzymes) and glutathione-*S*-transferases (GST, phase II metabolic enzymes) activities increased with the secondary plant compound hydroxamic acid levels in wheat (Castaneda et al. 2010) and GST activities also increased when *S. avenae* was dietary

exposed to phenol catechol, alkaloid gramine and non-protein amino acid L-ornithine-HCI (Cai et al. 2009; Zhang et al. 2013). Lu and Gao (2009) suggested the involvement of P450s and GSTs in the susceptibility to the insecticide pirimicarb. More specifically for the role of these mechanisms in detoxifying mycotoxins, studies with aphids remain scarce. In contrast to the aphids, hymenopteran insects such as honeybees, bumblebees and the parasitoid *Nasonia vitripennis*, for which the whole genomes are sequenced, are known to have substantially fewer genes coding for detoxification enzymes (Claudianos et al. 2006; Oakeshott et al. 2010; Sadd et al. 2015; Werren et al. 2010) compared to other insects, leaving them more vulnerable for xenobiotics. Exposure of Hymenoptera parasitoids, such as *A. ervi*, to mycotoxins through food chain contamination is thus another important parameter when assessing the risk against natural enemies.

In conclusion, this study emphasized the importance of mycotoxin (DON) in food chain contamination from the plant to insects (insect–plant interactions; grain aphids *S. avenae*) and their natural enemies (higher trophic interactions; parasitic wasp *A. ervi*). Based on our data, we believe that DON can cause a decreased efficiency of biological control agents such as parasitoids through food chain contamination. Therefore, it is important also to consider the presence of mycotoxins in environmental risk assessments when insect pest control systems are based on biological control with natural enemies, such as the use of parasitic wasps against aphids.

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