IS DEOXYNIVALENOL ABSORBED AT INTESTINAL LEVEL?

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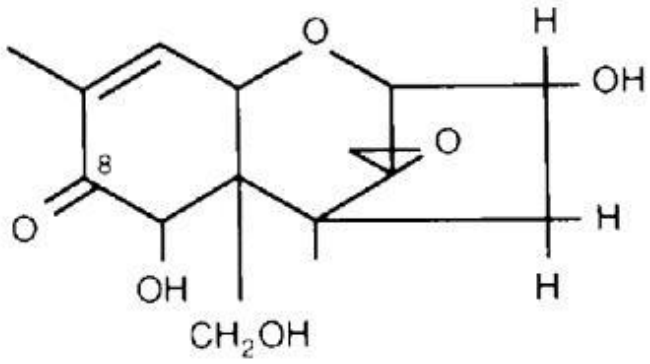
Overview on DEOXYNIVALENOL

- It is produced by several *Fusarium* genus, especially *Fusarium culmorum* and *Fusarium graminearum* (*Gibberella zeae*);
- It is not required for the growth of the producing fungus and, therefore, it is considered secondary metabolite;
- Presumably it plays some role in the ecology of the fungus, but its function has not been clearly defined.

Overview on DEOXYNIVALENOL

- Deoxynivalenol (DON , Vomitoxin) is a type B trichothecene
- DON is a tetracyclic sesquiterpenoid compound



- Molecular Formula: $C_{15}H_{20}O_6$
- DON has a great stability; it does not degrade at high temperatures

Overview on DEOXYNIVALENOL

- Deoxynivalenol can be found as a natural contaminant in:
  - cereals crops

    (maize, wheat, barley, oats, rye and rice)
  - processed grains (malt, beer, bread, cookies, cakes, pastas, breakfast cereals biscuit and croissants).

- Humans and animals are exposed to mycotoxin through consumption of contaminated food or feed.

Overview on DEOXYNIVALENOL

- **Animal Toxicity**

  - reduced growth; decreased feed consumption (**anorexia**) and emesis (**vomiting**)

- **Inhibition of protein, DNA and RNA synthesis** at ribosomal level

  - primary toxic effect

- **Genotoxic**

  - Genotoxicity (chromosomal aberrations) both *in vitro* and in *vivo*

  - The mechanism of genotoxicity is unclear, and the results are considered equivocal

- **Immunosuppressive** in a number of species
- **Embryotoxic** and **teratogenic** in mice

Overview on DEOXYNIVALENOL

- Currently in the USA from 3 to 7% of all humans are born with birth defects
- Presently the major cause of teratogenesis are unknown, but more or less the 20 % of these defects are due to:

⇒ Chromosomal aberrations;

⇒ Maternal metabolic imbalance;

⇒ Radiation exposure;

⇒ Infections;

⇒ Drugs.

- The other 80% of human defects may be due to chemical compounds present in the foods (additives, pesticides, mycotoxins, etc)

(Alberto Ritieni, 2010)

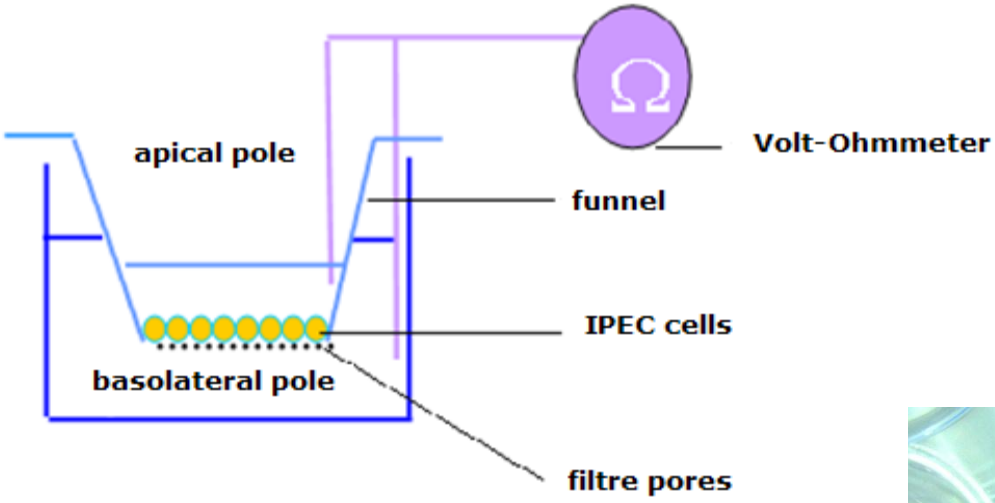
Aim of the study

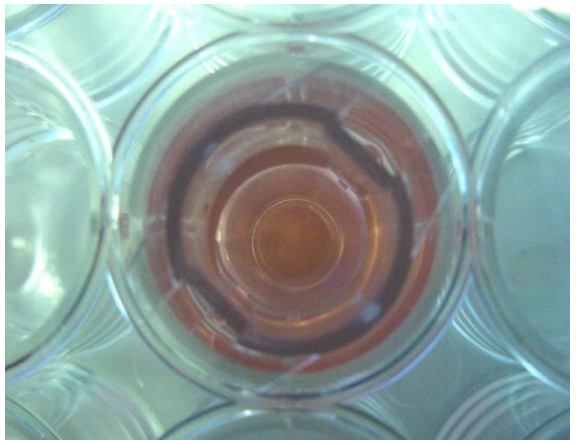
- The intestinal epithelium is an important physical barrier against ingested mycotoxin
- Is deoxynivalenol able to alter the epithelial intestinal barrier?
- The experiments aimed to find out if the mycotoxin is affecting the junction protein of the porcine intestinal epithelial cell line monolayer.

Materials and methods

- Intestinal porcine epithelial cells : IPEC 1(non-transformed intestinal cell line) were cultured in DMEM F12 medium in Transwell filters;
- The functional integrity of monolayer was determined by transepithelial electrical resistance (TEER) measurements;
- When the cells reached confluence 100 µl of 50 µM DON solution was added;
- 9 days after adding deoxynivalenol, mycotoxin determinations were done every two days, both in apical pole and in basolateral pole.

Materials and methods





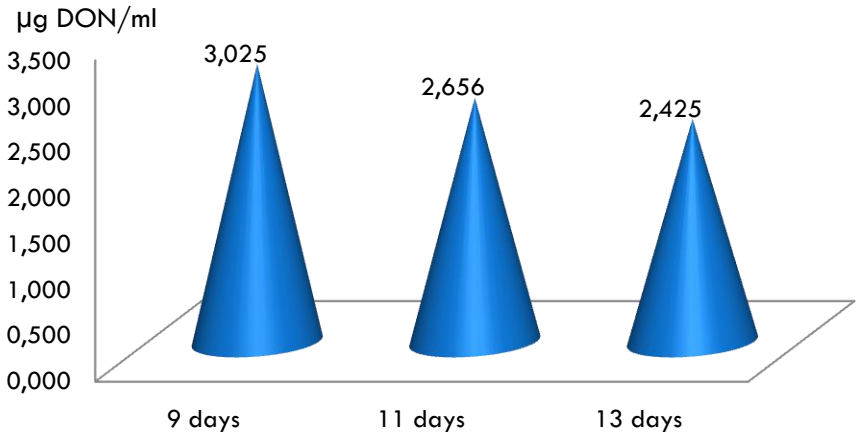
Materials and methods

- **MYCOTOXIN DETERMINATION**
- Clean-up: immunoaffinity chromatography (DONprep columns)
- HPLC-detection:

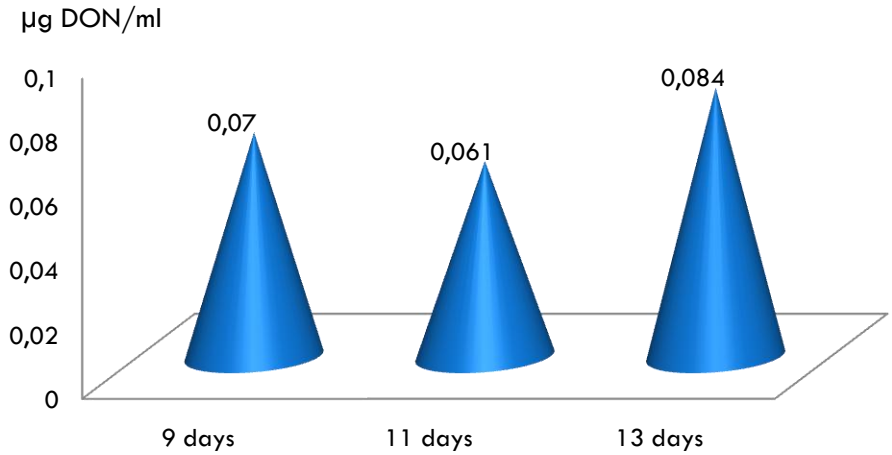
Column: C18, 150x4,6 mm, 5 µm
Mobile phase: acetonitrile:methanol:water (5:5:90)
Flow rate: 1.0 mL/min
Detection: λ = 220 nm
Injection volume: 100 µL
Temperature: 25°C
Run time: 12 minutes
Detection limit = 0.02 µg/mL

Experimental results – the 1st trial

- Apical pole

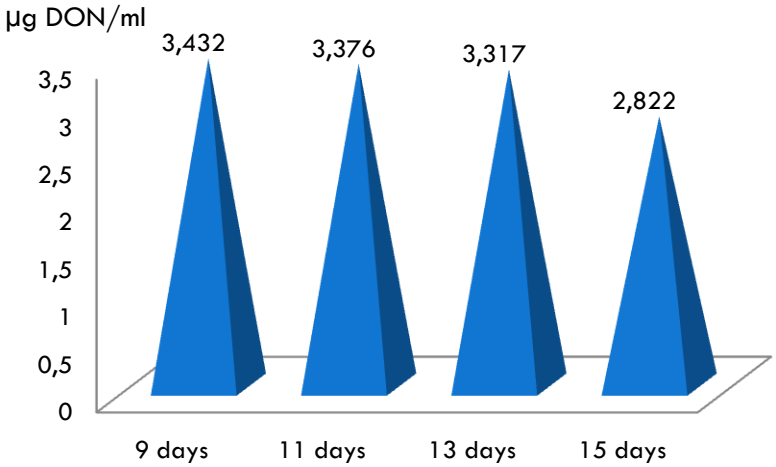


- Basolateral pole

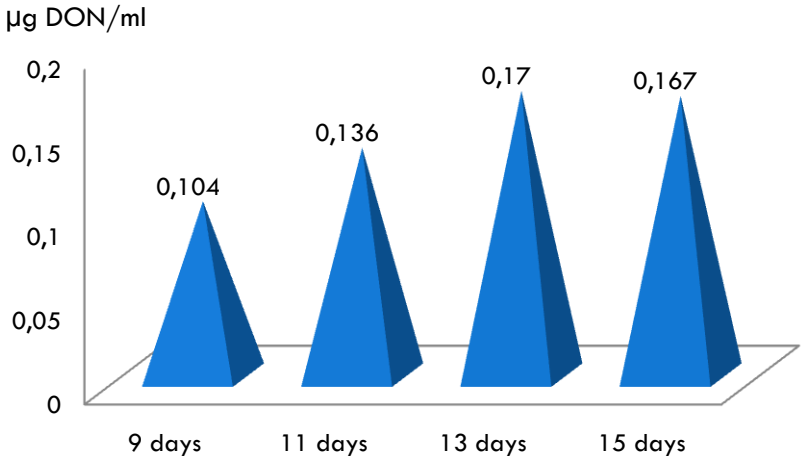


Experimental results – the 2nd trial

- Apical pole



- Basolateral pole



Conclusions

- Deoxynivalenol affects the integrity of intestinal epithelial monolayer, acting on the tight conjunction protein
- Deoxynivalenol is able to induce loss of intestinal epithelial layer barrier function, increasing permeability with consequences on the pathological bacterial infections.

THANK
YOU!