The acute and subacute oral toxicity of Fusarium-mycotoxin moniliformin

Introduction

The plant pathogen Fusarium is the most prevalent fungus, infecting small-grain cereals in the temperate regions of the world. Fusarium-species produce a range of secondary metabolites, responsible for a wide variety of adverse effects on plants, animals and humans. The major Fusarium mycotoxins include trichothecenes, zearalenone and fumonisins. However, most Fusarium-species also produce a variety of less prevalent mycotoxins, such as moniliformin (MON). The natural occurrence of MON has been demonstrated throughout the world. It has been frequently found in cereals in northern European countries, but in rather low concentrations (up to 810 µg/kg wheat, Finland). The highest levels have been found in Fusarium contaminant maize (425–530 µg/kg) (1). In this study, the acute and subacute oral toxicity of MON in rats was investigated, as only limited data existed regarding the in vivo toxicity of MON.

Experimental and results

The acute oral toxicity study 4

The acute oral toxicity study of MON was performed by adapting OECD Guideline 423, as a dose-finding trial for following subacute toxicity studies. The level of acute toxicity, possible target organs, clinical symptoms, histopathological changes and MON excretion into urine and feces were examined. A single high dose of 50 mg/kg b.w. synthetic MON and a single low dose of 5 mg/kg b.w. were administered to three, 9-week-old Sprague Dawley male rats /dose. The low dose was tested twice.

The subacute oral toxicity study 5

The subacute (repeated dose, 28-day) oral toxicity of MON was studied by adapting OECD-guideline 407. Sprague-Dawley rats (5 male rats/group) were exposed to synthetic MON by gavage at 6 different exposure levels (0-15 mg/kg b.w.). The exposure levels were determined based on the preceding acute oral toxicity study. In addition, two 14-day satellite groups were included in the study to assess the reversibility, persistence or delayed occurrence of toxic effects. Three animals per group were housed in metabolic cages. During the follow-up period and was only approx. 24% compared to control. These results suggest that the innate immunity of the rats was heavily affected and not recovered.

Conclusions

Our results indicate that MON is acutely toxic to rats with a LD₅₀ cut-off value of 25 mg/kg, causing acute heart failure, respiratory stress and muscle weakness. The urinary excretion is the main route for elimination and is fast (< 24 h) and independent of the exposure level. The toxin does not seem to accumulate or cause late signs of toxicity, however, the innate immunity was affected already by low doses.

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References

3. Logrieco et al., Occurrence and toxigenicity of Fusarium proliferatum from preharvest maize ear rot, and associated mycotoxins, in Italy Plant Disease 74 (1990) 72-73.