A modern view of the risk of mycotoxins in animal health is that they are ubiquitous environmental contaminants rather than rare pollutants of feed. It is therefore paramount in any risk assessment of mycotoxins in dairy health and production to know the clinical manifestations of mycotoxicoses, the toxic dosages of each toxin, and which toxins are present at what range of concentrations in the various feedstuffs used in a particular country. Even with these data, diagnosis of toxicoses is not straightforward, as despite the plethora of scientific data on the occurrence of mycotoxins and their effects on biological systems in controlled studies, there exist sparse data on proven (non-endophyte) mycotoxicoses actually recorded in ruminants. This may be due to a variety of factors:-

1. One cannot easily detect exposure on the farm as the more common mycotoxins cause non-specific clinical manifestations in farm animals, usually with decreased productivity (e.g. milk yield, increase in body weight) being the most evident sign. Such manifestations are of course seen with infection with numerous diseases, problems with feed, and even changes in husbandry or in the environment;

2. Aflatoxin, T-2 toxin and fumonisins have been shown to be immunosuppressive, whereby exposure may be manifested as an increased incidence of diseases on the farm, the primary cause being a toxicosis. In such cases, mycotoxicosis is rarely even suspected;

3. The effects of mycotoxins, in concentrations in feed likely to be found in developed countries, are typically produced in a sub-acute or chronic timeframe, which may also confound the diagnosis by making it difficult to determine when the problem started and so identifying the suspect feed (for mycotoxin analysis), and which may have been already totally consumed;

4. Protocols do not exist for sampling these less productive animals to determine exposure biomarkers (apart from aflatoxin in fully productive dairy cows) such as is the norm for diagnosis of diseases and many toxicoses;

5. Mycotoxins are usually produced as an arsenal of defensive toxins, and scientific appraisal of potential interactions between these toxins in farm animals has been barely started, mainly due to the very high cost of conducting the necessary comprehensive studies with numerous toxins at various concentrations and combinations. Such work has been done to some extent only in poultry, where additive and synergistic deleterious effects have been demonstrated;

6. It has been shown that experimental data (usually gleaned from studies conducted in conditions of optimal husbandry) often grossly underestimate the lowest toxic concentration under farm conditions. These data were found for instance with aflatoxin in poultry, where the feed concentration determined from field cases causing a reduction in weight gain was about 50 ppb, compared with 2500 ppb found in the laboratory. It is not known whether this discrepancy is manifested in other farm animals and with other toxins;

7. While most laboratories that analyze for mycotoxins quantitate aflatoxin, ochratoxin, T-2 toxin, zearalenone, deoxynivalenol and zearalenone, fewer examine for less commonly found mycotoxins such as diacetoxyscirpenol, which for instance caused numerous toxicoses in Israel in poultry, and whose effects are similar to T-2 toxin.

8. Another important aspect of laboratory inadequacy may be with silage, the feeding of which in a total mixed ration is the standard for the modern dairy herd, and which contains specific toxins not included in general screens. These include cyclopiazonic acid, gliotoxin, mycophenolic acid, penitrem, patulin and roquefortine, mycotoxins frequently found in silage, with largely unknown effects in cattle, although their toxicity has been demonstrated in other species.

With all these factors in the background, 3 important questions often posed should be answered decisively. Firstly, is it a practical threat that mycotoxins in intensive dairy herd feeds may affect production and health? Secondly, if so, do they actually cause problems? Thirdly, be there positive answers to the former questions, what steps can be taken to prevent or ameliorate such contamination? Unfortunately, in many countries, and particularly in Israel, it is assumed, without any risk assessment, that mycotoxins do harm cows, and so the use of feed additives, that are claimed to neutralize the effects of mycotoxins, has become widespread.

While the use of some such additives may to some extent help negate contamination, mainly with aflatoxin, they certainly cannot be efficacious against all mycotoxins, and even may lull the farmer into a false belief that the herd is "protected" from mycotoxins. The main risk with mycotoxins in dairy cow feed remains the finding of aflatoxin M1 residues in the milk after ingestion of aflatoxin B1. Recent research in Israel showed a high carry-over of aflatoxin in poultry, where the feed concentration determined from field cases causing a reduction in weight gain was about 50 ppb, compared with 2500 ppb found in the laboratory. It is not known whether this discrepancy is manifested in other farm animals and with other toxins.

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It is therefore recommended that in any national intensive dairy feed concern, a risk assessment be conducted, initially effected by comprehensive analyses for aflatoxin, cyclopiazonic acid, deoxynivalenol, diacetoxyscirpenol, gliotoxin, mycophenolic
acid, ochratoxin, patulin, penicillic acid, penitrem, roquefortine, T-2 toxin and zearalenone in various feedstuffs, particularly in silages. Recently developed multi-mycotoxin analytical methods make this more feasible than in the past.

REFERENCES