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767

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TOXIGENIC FUNGI ISOLATED FROM CEREAL AND LEGUME PRODUCTS
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Abstract
An investigation of the toxigenicity of moulds recovered from cereal and legume crops is described. 228 mould strains representing 59 species were tested by feeding Pekin ducklings on maize meal infected with pure cultures. 46 strains representing Aspergillus avenaeus, A. carneus, A. chevalieri, A. clavatus, A. flavipes, A. flavus, A. fumigatus, A. manginii, A. nidulans, Fusarium moniliforme, F. roseum, A. niveus, A. ochraceus, A. ruber, Penicillium varioli, Penicillium islandicum, P. piceum, P. rubrum, P. urticae, P. variabile and Trichothecium roseum caused death within 14 days. These 22 species were subsequently fed to weaned white mice and rats. Aspergillus avenaeus, A. flavipes, A. nidulans, A. niveus, A. ochraceus, Penicillium oxalicum and P. urticae caused the death of these animals within 14 days. Aspergillus carneus, Fusarium moniliforme, F. roseum, Penicillium islandicum, P. piceum and P. rubrum showed less severe toxic effects, while the remaining 9 species showed no overt signs of acute poisoning.

TOXIGENIC DEMATIACEAE
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Abstract
Moulds belonging to the family Dematiaceae have usually been regarded as harmless saprophytes. Recent work has indicated, however, that they may be of importance in diseases of unknown aetiology. A few mycotoxicoses caused by Dematiaceae are briefly described and a short account is given of work on toxic Dematiaceae done in this laboratory.

ACUTE LIVER INJURY IN DUCKLINGS AS A RESULT OF AFLATOXIN AND OCHRATOXIN POISONING

Abstract
In short-term experiments with ducklings aflatoxin B1, one of the toxic metabolites of the mould Aspergillus flavus, caused necrosis of the parenchymal liver cells with focal haemorrhage. Histochemical preparations showed a progressive decrease in the activity of the enzymes succinic dehydrogenase, alkaline phosphatase, adenosine triphosphatase, inosine diphosphatase and thiamine pyrophosphatase during the development of the lesions, but an increase in the activity of acid phosphatase. Ultrastructural changes in the parenchymal hepatic cells are described and it is suggested that the toxic principle (aflatoxin B1 or possibly a modified but closely related substance) was transported by the red blood cells and that at least one of its cytotoxic effects was due to a direct action on the liver cell membrane and the membranes of the various intracytoplasmic structures.

Under the same experimental conditions ochratoxin A, a toxic metabolite of the mould Aspergillus ochraceus, caused a mild fatty infiltration of the liver.

B. AGRICULTURAL ASPECTS
B. LANDBOUKUNDIGE ASPEKTE

MYCOTOXINS IN VETERINARY MEDICINE
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In recent years mycotoxins have been recognized as major aetiological factors in diseases of livestock and poultry. Intensification of the poultry industry has been followed by a fairly new complex of diseases associated with managerial deficiencies, some of which create ideal environmental conditions for fungal proliferation. The use of an important ingredient (which contained mycotoxins) in the rations of livestock led to extensive mortality which, if not recognized, could have had disastrous results, particularly for chickens, ducklings and dogs.

In veterinary medicine the mycotoxins cause 3 major disease syndromes.
1. The hepatoxins which affect only the liver, e.g. aflatoxin.
2. Those producing a haemorrhagic syndrome, e.g. haemorrhagic disease of fowls.
3. Those producing liver lesions and photosensitivity, e.g. facial eczema in sheep.

1. AFLATOXICOSES
Aflatoxin is a hepatotoxin which affects ducklings, poult's, pheasants and New Hampshire chickens up to the age of 10 weeks, the most susceptible age being 1 - 4 weeks; dogs of any age, piglets under the age of 8 weeks and calves under the age of 3 months. Sheep are not susceptible to amounts of toxin normally encountered in rations, but are affected when given two ounces of groundnut cake meal containing upwards of 60 parts per million twice a week for 4 - 6 weeks. Chickens of the Cornish Game, White Rock, White Leghorn and Rhode Island Red breeds are unaffected when given rations containing 0.75 ppm toxin. It is strange that the Rhode Island Red should be refractory since the New Hampshire breed is derived from the Rhode Island Red. Cornish Game and New Hampshire Crosses are also refractory.

There is a suspicion, yet to be confirmed, that chinchillas are also susceptible. Of the laboratory animals generally used, ferrets, white rats and guinea-pigs are susceptible, while white mice are refractory. Trout have also been found to be susceptible to very small amounts of the toxin.

In India Rhesus monkeys were found to develop acute aflatoxicosis when given high doses of the pure toxin. All the afore-mentioned susceptible animals can develop acute aflatoxicosis, the degree of which depends on the toxic level of the feed, except in rats which develop hepatomas after feeding continuously for nine months or longer on

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