Fungi Associated with Common Crops and Crop Products and their Significance

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SUMMARY

Both field and storage fungi are now known to produce a wide variety of toxic metabolites. Mycotoxicoses producing acute symptoms are well documented; interest at present is centred on the possible sublethal or chronic effects of long-term ingestion of mycotoxins.

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The study of fungi on crops has gained a new dimension with the advent of mycotoxicology. Whereas formerly their interest briefly concerned plant pathology *per se*, nowadays we are interested in the effects, possibly subtle, which even common saprophytes and pathogens may exert on human physiology through the contribution of small quantities of metabolites to the crop as it develops in the field, and afterwards in storage.

Christensen[1] and other authors have defined two groups of fungi differing in one or more physiological characteristics: the field and storage fungi. The relevance of this distinction lies in the claim that the storage fungi as a group are more likely to produce mycotoxins than the field fungi, although the latter include some very important toxicogenic species. Table I lists the main differences between the two categories.

Recent work has shown that there is an intermediate group of fungi which, under varying conditions, may behave either as field fungi or as storage fungi. Thus our classification cannot be a strictly rigid one. Furthermore, many of the field and intermediate fungi have a systemic relationship with their hosts that has only recently been revealed. A good example of the intermediate group, in which a systemic relationship with the maize plant has been demonstrated, is *Fusarium moniliforme*.

The relationship of fungi to plants in general is profoundly influenced by environmental factors, the chief of which is nutrition, followed by the age of the host plant, weather conditions and competition by other organisms, including other species of fungi. It is generally accepted that the greater the degree of seed infestation, the greater the likelihood of toxin formation by the fungus concerned. Since there is now good evidence that other outbreaks of crop diseases are seasonal in incidence, a study of the factors underlying them may give a clue to the sudden outbreak of a particular mycotoxicosis. A combination of field observations and laboratory work has shown that the main factors governing the development of toxins, specifically aflatoxin, in natural products are: (*a*) compatibility of substrate; (*b*) high humidity governed by the extent of rainfall; (*c*) trauma suffered by the developing fruits and seeds at harvest or in storage. (*d*) drying treatments—rapid processing of seeds leads to low fungal infestation and little or no toxicity while slow drying gives the reverse; (*e*) method of harvesting, the method resulting in least trauma and most rapid drying being the most successful; (*f*) temperature—results indicate that aflatoxin formation takes place at relatively high temperatures

TABLE I. MAIN DIFFERENCES

Field fungi	Storage fungi
1. Persist in the seed or fruit under dry conditions. Limited by excess moisture.	1. Persist under dry conditions but require moisture contents in excess of a specific level (9% for groundnuts, 13 - 14% for cereals) for development. These moisture levels are not usually present in pre-harvest conditions.
2. Except in the case of destructive parasites (e.g. *Fusarium* scab), species do not attack the germ of the seed or contribute to rapid deterioration. Many may parasitise leaves, stems and inflorescences, and merely discolour the seed.	2. This group includes a number of species specifically invading seeds only (e.g. *Aspergillus restrictus*), that bring about deterioration and death of seeds within a short time.
3. No significant biochemical change in seeds or fruits.	3. Increase fat acidity. Increase reducing sugars. Decrease non-reducing sugars. Decrease protein content.
4. Do not cause heating.	4. May cause localised heating of grain if certain thermophilic species are present (*Penicillium cyclopium, P. funiculosum*).
5. Representative examples: **Saprophytes** — *Alternaria alternata* *Cladosporium cladosporioides* *Cochliobolus geniculatus (Curvularia geniculata)* *Epicoccum purpurascens* *Fusarium graminearum* *Penicillium oxalicum* *Rhizopus stolonifer* **Parasites** *Drechslera rostrata* *Leptosphaerulina arachidicola* *Physalospora rhodina (Botryodiplodia theobromae)* *Trichometasphaeria turcica*	5. Representative examples: *Aspergillus candidus* *A. flavus* *A. repens* *Corticium solani* *Gliocladium catenatulatum* *Macrophomina phaseolina* *Penicillium chrysogenum* *P. viridicatum*

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TABLE II. INCIDENCE OF AFLATOXIN IN AMERICAN CROPS[7,8]

Substrate	No. of samples	% frequency of *A. flavus*	% frequency of aflatoxin	Comment
Maize	**1 311**	**54**	**2,7**	**Most of the toxic samples were drawn from the poorer grades of the crop including 'sample grade' of very low quality.**
Soya beans	**866**	**50**	**0,2**	
Sorghum	**533**	**43**	**1,2**	
Wheat	**531**	**20**	**0,4**	
Oats	**304**	**14**	**1,0**	

TABLE III. INCIDENCE OF AFLATOXIN IN SWAZILAND CROPS[9]

Substrate	No. of samples	% frequency of *A. flavus*	% frequency of aflatoxin (10 g/kg)	Comments
Maize	**418**	**37,0**	**4,3**	**Mostly good quality, stored above and below ground**
Groundnuts	**180**	**49,4**	**11,1**	**Mixed quality but representative**
Groundnut meal	**238**	**78,2**	**12,6**	**Prepared by local methods**
Groundnut butter	**190**	**85,1**	**18,9**	**Prepared by local methods**
Sorghum	**39**	**33,3**	**7,7**	**Mostly good quality**
Sorghum malt	**33**	**60,6**	**0,0**	**Prepared locally for beer**
Various pulses	**46**	**54,3**	**0,0**	**Mostly good quality**

(28 - 32°C), whereas the fusarial toxins are produced at low or freezing temperatures (−7 - 25° C), and a cycle of alternate freezing and thawing greatly enhances the process.

Detailed work on the physiology of toxin formation, which will remedy the present scanty knowledge, is being undertaken at the present time. One can conclude that there is not likely to be any over-all uniformity in the conditions required to produce mycotoxins: the formation of each one will have its own specific requirements.

In terms of natural occurrence, aflatoxin would appear to lead the field. Indirect evidence shows that there is a peculiar affinity of *Aspergillus flavus* for groundnuts, both as a substrate for growth and for aflatoxin formation. A survey of isolates of *A. flavus* revealed that the most toxigenic were of groundnut origin.[2] Numerous authors[3-6] have demonstrated significant levels of aflatoxin in harvested and in stored groundnuts, concentrations being high when conditions are poor. Aflatoxin also has a high natural occurrence in groundnut cake and groundnut oil. Other crops do not apparently produce aflatoxin as readily, even though the incidence of *A. flavus* may be relatively high. Although the evidence is still scanty, it would appear that staple foodstuffs are not equally prone to aflatoxin formation, as can be seen from Tables II and III.

The natural occurrence of other toxins is much less common, but the information is also scanty. Zearalenone must obviously occur fairly frequently because of the large number of outbreaks of hyperoestrogenism in animals that have been traced to stored material. The geographical distribution of the outbreaks in those parts of the world where there is a cool temperate but a continental climate, with considerable and sudden temperature changes, is consistent with the physiological studies showing that toxin production is enhanced by sudden cold. As far as the evidence goes, however, other mycotoxins are not formed to the same extent as aflatoxin, as the surveys on harvested maize by Shotwell *et al.*[10,11] in the USA and Canada show (Table IV).

TABLE IV. YEARLY INCIDENCE OF MYCOTOXINS IN AMERICAN MAIZE

Year	No. of samples	Number with Aflatoxin	Ochratoxin	Zearalenone
1970	**283**	**6**	**1**	**2**
1971	**293**	**8**	**3**	**5**

It is significant that several investigations in the USA, Canada and Southern Africa have revealed only one isolation of sterigmatocystin, a known hepatocarcinogen in rats.[12] This apparent infrequency mitigates the likelihood of sterigmatocystins being significant in the development of human hepatoma.

One of the exciting recent discoveries has been the demonstration of zearalenone, by Dr B. D. Jones of the Tropical Products Institute, London, and the writer, in four samples of malted sorghum, pooled samples of fermented porridge and beer made from maize and sorghum, and two samples of mouldy maize off the cob from Swaziland. Other mycotoxins—aflatoxins, patulin, ochratoxin and sterigmatocystin were not found in these foodstuffs. The regular ingestion of zearalenone by the Black population, however, could explain the high incidence of disease syndromes such as cervical cancer in a way not hitherto suspected. Long-term treatment with oestradiol has been shown to induce cervical cancer in mice.[13] If naturally-occurring oestrogens were definitely implicated in human pathology, the original mycotoxin hypothesis of Oettle[14] could be dramatically extended.

The relevance of a study of common fungi infesting fruits and seeds can be appreciated by the survey of the disease syndromes known to be associated with specific fungi. The main ones are summarised in Table V.

TABLE V. DISEASE SYNDROMES ASSOCIATED WITH SPECIFIC FUNGI

Major disease category, type of illness, and fungi	Field (F) or storage (S)	Toxin	Acute toxicity symptoms	Chronic or sublethal effect
Allergic illness				
Alternaria	F	None.	Allergy resembling bronchitis (mainly humans).	Secondary lung changes, emphysema and fibrosis (human).
Aspergillus	S	Irritation thought to be directly due to mechanical action of spores.		
Candida	S			
Cladosporium	F			
Fusarium	F/S			
Helminthosporium	F			
Penicillium	F/S			—
Verticillium	F			
Mycotoxicosis: I. Mouldy corn toxicosis				
Alternaria	F	Various, acting synergistically.	Toxicosis with multiple symptoms affecting skin of face, eyes, GIT, liver, viscera, blood system and nervous system (various animals).	Not known.
Aspergillus				
flavus + *spp.*	S			
Cladosporium	F			
Mucor	S			
Penicillium				
rugulosum + *spp.*	S			
Scopulariopsis				
brevicaulis	S			
Stachybotrys				
alternans	S			
Mycotoxicosis: II. Primarily involving liver or kidney				
(a) Aflatoxicosis				
Aspergillus				
flavus	S	Aflatoxin	Centrilobular necrosis and proliferation of bile ducts and fibrosis in liver. Impairment of blood clotting system, haemorrhage, weakening of gastric motor function (animals and birds).	Hepatoma in rats, ducks and other animals. Strong circumstantial evidence for human hepatoma.[4,15-17]
A. niger	S			
A. ostianus	S			
A. parasiticus	S			
A. ruber	S			
A. wentii	S			
Penicilluim				
citrinum	S			
P. frequentans	S			
P. puberulum	S			
P. variabile	S			
(b) Sterigmatocystin toxicosis				
Aspergillus nidulans	S	Sterigmatocystin	Necrosis and peritonitis (rats).	Hepatoma, cholangiosarcoma in experimental animals only.
A. rugulosum	S			
A. versicolor	S			
Bipolaris sorokiniana	F			
(c) Luteoskyrin toxicosis				
Penicillium islandicum	S	Luteoskyrin	Centrilobular necrosis and cirrhosis of liver in many experimental animals.	Hepatoma in experimental rats only.
(d) Ochratoxicosis				
Aspergillus ochraceus	S	Ochratoxin	Necrosis and fatty infiltration of liver. Damage to renal tubules and fibrosis of kidney (animals).	Not known.
Penicillium viridicatum	S			
(e) Aspergillus fumigatus toxicosis				
Aspergillus fumigatus	S	Fumagillin, fumigatin	Multiple internal damage including liver and kidney (animals).	Not known.

TABLE V (CONTINUED)

Major disease category, type of illness and fungi	Field (F) or storage (S)	Toxin	Acute toxicity symptoms	Chronic or sublethal effect
(f) Patulin (clavacin) toxicosis				
Aspergillus clavatus	S	Patulin (clavacin)	'Maltgerm intoxication' of cattle. Necrosis and granulomata in liver and pancreas, degeneration of kidney, haemolysis of red cells.	Sarcomas in experimental rats only.
A. giganteus	S			
A. terreus	S			
Byssochlamys nivea	S?			
Penicillium claviforme	S?			
P. expansum	S			
P. urticae	S			
(g) Penicillic acidosis				
Aspergillus melleus	S	Penicillic acid	Liver damage. Loss of co-ordination.	Tumours after injection in rats only.
A. ochraceus	S			
A. quercinus	S			
A. sulphureus	S			
Penicillium baarnense	S?			
P. cyclopium	S			
P. madriti	S?			
P. martensii	S			
P. palitans	S			
P. puberulum	S			
P. stoloniferum	S			
P. suaveolens	S			
P. thomii	S			
(h) Facial eczema (sheep)				
Pithromyces chartarum	F	Sporidesmin	Biliary obstruction leading to photosensitisation of facial skin.	None reported.
Periconia minutissima	F			
(i) Diplodiosis (cattle)				
Diplodia zeae	F	*Diplodia zeae* toxin	Kidney degeneration, catarrhal enteritis, lung hyperaemia, inco-ordination.	Not known.
(j) Rubratoxicosis (camels, birds)				
Penicillium purpurogenium	S	Rubratoxin	Liver engorgement, haemorrhagia.	Not known.
P. rubrum	S			
(k) Polyuria (Sassoon Hospital syndrome)				
Absidia ramosa	S	Toxin unnamed	Glomerulonephrosis. Fatty infiltration of liver (humans).	Not known.
Aspergillus clavatus?	S	Citrinin		
Penicillium citrinum	S	Citrinin		
Rhizopus stolonifer	F	Toxin unnamed		
(l) Haematuria				
Chaetomium globosum	S	Chaetoein	Haemorrhagia in kidney (animals).	Not known.
Gliocladium fimbriatum	S	Gliotoxin		
Trichoderma viride	F	Gliotoxin		
Mycotoxicosis: II. Primarily involving organs other than liver or kidney				
(a) Drunken Bread Syndrome				
Fusarium graminearum	S	Zearalenone	Ataxia, diarrhoea (humans).	Not known.
(b) Hyperoestrogenism				
F. graminearum	S	Zearalenone	Abortion, necrosis and inflammation of genitalia. Increase in weight of uterus. GIT, blood and nervous system also affected (animals).	Not known.
F. moniliforme	S			

TABLE V (CONTINUED)

Major disease category type of illness and fungi	Field (F) or storage (S)	Toxin	Acute toxicity symptoms	Chronic or sublethal effect
(c) Alimentary toxic aleukia (alimentary septic angina)				
F. sporotrichioides	S	Sporotrichin	Haemorrhage, necrosis, inflammation of membranes, GIT disorders, disturbance of nervous system, blood system, etc. (humans).	Not known.
F. poae				
(d) Leucoencephalomalacia				
F. moniliforme	F/S	Not known	Focal necrosis of brain matter (horses).	Not known.
(e) Fescuetoxicosis				
F. equiseti	F/S	T_2 toxin (diacetoscirpenol)	Lameness and gangrene, etc. (cattle).	Not known.
F. nivale				
F. tricinctum				
(f) Stachybotryotoxicosis		T_2 toxin		
Stachybotrys alternans	F	Stachybotryotoxin	Haemorrhage, inflammation of membranes, disturbance of nervous system (cattle).	Not known.
(g) Dendrodochiotoxicosis				
Dendrodochium toxicum	F	Dendrodochiotoxin	Paralysis, generalised haemorrhage (cattle).	Not known.
(h) Ergotism				
Claviceps purpurea	F	Ergotamine	Abortion, contractile effect on uterus and circulatory system, gangrene (cattle, humans).	Not known.
(i) Tremor convulsion				
Penicillium crustosum	S	Tremorgen (tremortin)	Tremors and convulsions (animals).	Not known.
P. cyclopium	S			
P. granulatum	S			
P. palitans	S			
P. cyclopium				
(j) Haemorrhagia	S	Cyclopiazonic acid	As above (animals).	
Alternaria tenuis				
	F	Alternarin, tenuazonic acid	Haemorrhage, multiple lesions (birds).	Not known.
Cladosporium epiphyllum	F	Not named		
C. fagi	F	Not named		
(k) Ustilagotoxicosis	F	Not named	Epileptiform convulsions, salivation, other multiple effects (animals).	Not known.
Puccinia graminis	F			
Tilletia laevis	F			
Ustilago avenae	F			
U. hordei	F			
U. zeae				

It is clear from Table V that the acute effects of fungal poisoning are well documented. Interest now centres on the possible results of long-term feeding of small sublethal doses of mycotoxins. As far as aflatoxin is concerned, it is clear that hepatoma is produced in experimental animals such as the rat, and the indirect epidemiological evidence for human hepatoma is convincing. For the other mycotoxins a great gap still exists in our knowledge.

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