

## Exposure of garden birds to aflatoxins in Britain

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### Abstract

Aflatoxins are potent biological toxins that have been shown to exert a range of acute and chronic pathological effects. Multiple mortality events of waterfowl caused by acute aflatoxicosis have been documented in the USA. However, international concern has recently been expressed regarding the potential effects of chronic exposure of wildlife species to low levels of dietary aflatoxin. This study documents for the first time the presence of hepatic aflatoxin residues in British wild birds: two passerine species, the house sparrow (*Passer domesticus*) and greenfinch (*Carduelis chloris*). Further research is required to investigate the source of the dietary aflatoxins and their pathological significance, if any, for wild birds in Britain.

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**Keywords:** Aflatoxin; *Passer domesticus*; *Carduelis chloris*; Wild bird

### 1. Introduction

Aflatoxins are a group of compounds whose metabolites have been demonstrated to exert carcinogenic, immunosuppressive, hepatotoxic and other pathological effects. The ability of aflatoxins to impair protein synthesis and interact with metabolic pathways has been attributed to many of the above properties (Pier and Heddleston, 1970; Pier, 1992; Quist et al., 2000). The fungal species, *Aspergillus flavus*, *A. parasiticus* and *A. nomius* that occur commonly in the air, soil and other natural substrates, are responsible for the production of these toxins (Pitt and Hocking, 1997). Aflatoxins are considered natural contaminants of a variety of agricultural products, with corn, peanuts, cottonseed, and other

grain crops being most frequently contaminated (Gourama and Bullerman, 1995; Creekmore, 1999).

Aflatoxins were first identified when they were found to be a cause of acute toxicity in commercial turkeys in the 1960s, the condition being known as “Turkey-X disease” (Blount, 1961). Aflatoxin toxicity has been documented since in a variety of taxa (mammals, fish and birds). Although birds appear to be the most susceptible (Creekmore, 1999), there is significant variation between avian species in their susceptibility to aflatoxins. Also, susceptibility to the effects of aflatoxins varies with age, sex and nutritional status, with young birds likely to be the most significantly affected (Creekmore, 1999; Maia and Pereira Bastos de Siqueira, 2002). The acute effects of aflatoxin toxicity are characterised by hepatic injury, coagulopathy, haemorrhage, icterus and death (Pier, 1992). Chronic, low level exposure to aflatoxins has been shown to be associated with a range of more insidious effects, such as reduced weight gain, suppression of the im-

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immune system, interference with reproductive function and neoplasia (Pier, 1992; Sharma, 1993; Ortatatli et al., 2002; Verma et al., 2004).

Much research has been performed on the effect of aflatoxin exposure on domestic farmed bird species (Muller et al., 1970). Given the susceptibility of farmed turkeys to aflatoxins, concern was raised over similar potential effects on wild turkeys (*Meleagris gallopova silvestris*) in the USA. Experimental aflatoxin exposure in this species led to reduced weight gain and feed consumption, impaired cell-mediated immunity and mild liver damage (Quist et al., 2000). Feeding trials were performed on wild game bird species to examine the degree of interspecific variation in their response to aflatoxin exposure. Dietary aflatoxin concentrations of 1250, 2500 and 5000 µg/kg (ppb) were fed for three weeks, since these represent the range of concentrations in poultry that have been found to result in no adverse effects, mild effects and severe effects, respectively. In order of relative susceptibility to the effects of aflatoxin, ring neck pheasants (*Phasianus colchicus*) were most affected, followed by the domestic chicken and bobwhite quail (*Colinus virginianus*); Chukar partridge (*Alectoris chukar*) and the Japanese quail (*Coturnix coturnix japonica*) were relatively resistant. Significant mortality was experienced by ring neck pheasants and bobwhite quail at dietary aflatoxin levels exceeding 1250 µg/kg (Ruff et al., 1990, 1992; Huff et al., 1992).

Historically, the majority of aflatoxin research has been performed because of the economic implications to the poultry industry of chronic exposure (Oliveira et al., 2002) or for evaluation of potential public health risks through consumption of contaminated meat, milk or eggs (Maia and Pereira Bastos de Siqueira, 2002). However, in recent years, increasing focus has been placed on the study of aflatoxicosis in free-ranging wildlife species. Mass mortalities of several waterfowl species due to acute aflatoxicosis have been reported in Texas and Louisiana, USA. Deaths occurred during the autumn/winter season and were attributed to the birds feeding on waste crops (e.g., peanuts, corn) in agricultural fields (Robinson et al., 1982; Cornish and Nettles, 1999; Creekmere, 1999). Robinson et al. (1982) reported two mortality incidents affecting primarily snow geese (*Anser caerulescens*) and mallards (*Anas platyrhynchos*), respectively. Analysis of a pooled sample of oesophageal and proventricular contents from affected birds in the first incident yielded 500 µg/kg AFB<sub>1</sub> (dry weight) whilst the same sample types from individual birds from the second incident yielded levels from 10 to

250 µg/kg AFB<sub>1</sub> (dry weight). Robinson et al. (1982) found 110 µg/kg AFB<sub>1</sub> in a sample of peanuts taken randomly from the field of the second incident. Cornish and Nettles (1999) analysed samples of corn from fields where a variety of goose species were affected by acute aflatoxicosis and found levels as high as 8200 µg/kg aflatoxin. Recently, concern has been expressed for the significance of chronic low level exposure of aflatoxins to wild bird species either through supplementary, agricultural or wild seed sources (Schweitzer et al., 2001).

Although no evidence, either anecdotal or scientific, is available on aflatoxin exposure of birds in Britain, such exposure has been repeatedly raised as a possible cause of morbidity or mortality of garden birds provisioned with commercially available food by householders. Consequently, high standards of food quality have been set for garden bird feed retailers belonging to voluntary associations, such as the Bird Care Standards Association, with members opting to follow a code of standards whereby only peanuts that have been screened and found to contain nil detectable levels of aflatoxin (BSA, 2005) are sold. Most food purchased for garden bird provisioning in Britain is, however, bought from retailers (such as independent pet shops) outside these associations, and aflatoxin B<sub>1</sub> levels in these products are set at the legal maximum permissible level (MPL) of 20 µg/kg, as is the case for all feeding stuffs in Britain (Anon, 2004a,b,c,d).

To investigate if aflatoxin exposure is a risk for garden birds in Britain, we conducted aflatoxin analyses on liver samples collected from a subset of garden birds submitted to the Institute of Zoology, London, for postmortem examination between 1999 and 2003. The liver was selected for aflatoxin testing because this organ retains the highest levels of aflatoxins following ingestion (Madden and Stahr, 1995) and is the major target organ in terms of the toxins' pathological effects (Oliveira et al., 2002).

## 2. Materials and methods

Opportunistic reports of garden bird mortality were solicited from members of the public through an organisational network including the British Trust for Ornithology (BTO), the Royal Society for the Protection of Birds (RSPB), the Royal Society for the Prevention of Cruelty to Animals (RSPCA), the Universities Federation of Animal Welfare (UFAW) and the Zoological Society of London (ZSL). Where available, carcasses were submitted to the Institute of Zoology where they

were examined following a standardised postmortem examination protocol. Details of the date found, geographical origin and circumstances were recorded. Each bird was assigned a unique postmortem reference code. During each examination, the species, age, sex, total body weight and body condition were recorded. Systematic internal and external examination of body systems was performed and gross lesions described. Where indicated, and where the state of carcass decomposition permitted, samples were taken for parasitological and toxicological investigation. No cases were considered to be fresh enough for meaningful histopathological examination.

Liver, small intestine and any lesions observed were routinely sampled aseptically and examined for the presence of pathogenic bacteria using a standard protocol. Briefly, tissue samples were plated directly onto (1) Colombia blood agar (QCM laboratories, Unit 205–206, Greenheath Business Centre, Three Colts Lane, London, E2 6JL, UK) supplemented with 5% horse blood, and incubated under aerobic, anaerobic and carbon dioxide conditions and observed after 1, 2 and 5 days (2) Xylose–Lysine Deoxycholate (XLD) medium agar (QCM laboratories) and incubated under aerobic conditions and observed after 2 days. (3) immersed into selenite *Salmonella*-selective enrichment broth (QCM laboratories) under aerobic conditions for 48 h followed by subculture onto XLD agar aerobically, 37 °C for a further 48 h. (4) Chocolate blood agar (QCM Laboratories) in carbon dioxide at 37 °C and observed after 1, 2 and 5 days. GENbox Anaerobic and CO<sub>2</sub> gas packs in AnaeroPack rectangular jars (bioMerieux, Marcy l'Etoile, France) were used. Bacterial isolates were identified using colony and organism morphology, Gram's staining properties and biochemical properties using the API biochemical test strip method (Biomérieux BioMérieux, Marcy l'Etoile, France). Slide agglutination tests were performed for the identification of suspected *Salmonella* sp. isolates using poly-O antisera (Pro-lab diagnostics). Isolates were then placed onto microbank beads (Pro-lab diagnostics) and stored at both –25 and –70 °C. Batches of isolates were later submitted to the *Salmonella* Reference Unit, Laboratory of Enteric Pathogens, Health Protection Agency, for complete biotyping according to standardised international protocols (Anderson et al., 1977).

An archive of liver samples from garden birds was kept in frozen storage at –20 °C. Liver samples from 13 greenfinches (*Carduelis chloris*) (13 locations) and from 22 house sparrows (*Passer domesticus*) (16 locations) found dead between 1999 and 2003 (Table 1) were analysed at the Central Science Laboratory for the

major aflatoxin residues (AFB<sub>1</sub>, AFB<sub>2</sub>, AFG<sub>1</sub>, AFG<sub>2</sub>) using immunoaffinity column (IAC) clean-up and high pressure liquid chromatography. The method employed was based on published methods for aflatoxins (Sharma and Gilbert, 1991). Briefly, samples were weighed and chloroform and 0.1 M phosphoric acid solution were added before the samples were blended at high speed using an Ultra turrax blender. The samples were centrifuged and the chloroform extract (or an aliquot of it) was evaporated to dryness using a rotary evaporator. The residue was redissolved in methanol and phosphate buffered saline (PBS). This was partitioned with hexane in a separating funnel. An aliquot of the methanol/PBS extract was applied to an Easi-Extract aflatoxin immunoaffinity column (R-Biopharm Rhone Ltd., Glasgow), preconditioned with PBS. The columns were washed with distilled water (10 ml) and then eluted using high performance liquid chromatography (HPLC) grade acetonitrile (1.5 ml). An aliquot of the cleaned up acetonitrile extract was diluted with water prior to analysis by reversed phase HPLC. The IAC clean-up and HPLC injection were carried out automatically by a Gilson ASPEC system fitted with an automated Rheodyne switching valve and injection loop. Injection was by partial loop fill, using an injection volume of 400 µl. The HPLC system also comprised a Gilson HPLC pump, and a JASCO FP 1520 fluorescence detector set at excitation wavelength 364 nm and emission wavelength 440 nm. Chromatography was performed using a Spherisorb ODS1-Excel HPLC column, (25 cm × 4.6 mm i.d., 5 µm particle size). The mobile phase consisted of water:acetonitrile:methanol, (54:30:16, v/v/v) at a flow rate of 1.0 ml/min. Pyridinium bromide perbromide (PBPB, 50 mg/l, pumped at 0.3 ml/min) was used for post column derivatisation of aflatoxins B<sub>1</sub> and G<sub>1</sub>.

Quantification was by external calibration. Fresh calibration standards were prepared for each batch of samples. These were injected at the beginning and end of each run and were also interspersed between samples. All standards in a run were used to prepare four point calibration curves. Peaks were identified by matching retention times to the nearest calibration standard. The specificity of the clean-up, derivatisation method and specific excitation and emission wavelengths used were considered sufficient to ensure accurate peak identification. Duplicate spiked samples were analysed in each batch, recovery values were in the range 70–93% (acceptable range 70–110%). All results met quality assurance parameters e.g., peak asymmetry, retention time drift, resolution etc. established for UKAS accredited aflatoxin analyses.

Table 1  
Results of aflatoxin screening of liver samples from garden birds

No	Species	Season and year of death	Cause of death	Aflatoxin residues measured <sup>†</sup> µg/kg (ppb)				
				AFB1	AFB2	AFG1	AFG2	Total AF
1	Greenfinch	Winter 1999/2000	Salmonellosis	0.1	ND	ND	ND	0.1
2	House sparrow	Summer 2000	Other trauma	ND	ND	ND	ND	ND
3	House sparrow	Autumn 2000	Salmonellosis	0.8	0.1	ND	ND	0.9
4	House sparrow	Autumn 2000	Salmonellosis	0.4	ND	ND	ND	0.4
5	Greenfinch	Winter 2000/2001	Salmonellosis	ND	ND	ND	ND	ND
6	Greenfinch	Winter 2000/2001	Salmonellosis	ND	ND	ND	ND	ND
7	Greenfinch	Spring 2001	Other trauma	ND	ND	ND	ND	ND
8	Greenfinch	Winter 2001/2002	Salmonellosis	0.3	ND	ND	ND	0.3
9	Greenfinch	Winter 2001/2002	Salmonellosis	ND	ND	ND	ND	ND
10	Greenfinch	Winter 2001/2002	Not established	ND	ND	5.8	2.2	8.0
11	Greenfinch	Winter 2001/2002	Salmonellosis	0.1	ND	ND	ND	0.1
12	Greenfinch	Winter 2001/2002	Salmonellosis	0.2	ND	ND	ND	0.2
13	Greenfinch	Winter 2001/2002	Salmonellosis	0.1	ND	ND	ND	0.1
14	Greenfinch	Winter 2001/2002	Salmonellosis	0.7	0.1	ND	ND	0.8
15	House sparrow	Winter 2001/2002	Salmonellosis	ND	ND	ND	ND	ND
16	House sparrow	Winter 2001/2002	Salmonellosis	ND	ND	ND	ND	ND
17	House sparrow	Winter 2001/2002	Salmonellosis	ND	ND	ND	ND	ND
18	House sparrow	Winter 2001/2002	Salmonellosis	ND	ND	ND	ND	ND
19	House sparrow	Winter 2001/2002	Salmonellosis	ND	ND	ND	ND	ND
20	House sparrow	Winter 2001/2002	Salmonellosis	0.4	ND	ND	ND	0.4
21	House sparrow	Winter 2001/2002	Salmonellosis	0.4	ND	ND	ND	0.4
22	Greenfinch	Spring 2002	Predation	ND	ND	ND	ND	ND
23	House sparrow	Spring 2002	Predation	ND	ND	ND	ND	ND
24	House sparrow	Spring 2002	Predation	ND	ND	ND	ND	ND
25	House sparrow	Spring 2002	Predation	ND	ND	ND	ND	ND
26	House sparrow	Spring 2002	Predation	ND	ND	ND	ND	ND
27	Greenfinch	Summer 2002	Predation	ND	ND	ND	ND	ND
28	House sparrow	Summer 2002	Predation	ND	ND	ND	ND	ND
29	House sparrow	Summer 2002	Predation	ND	ND	ND	ND	ND
30	House sparrow	Summer 2002	Predation	ND	ND	ND	ND	ND
31	House sparrow	Summer 2002	Predation	ND	ND	ND	ND	ND
32	House sparrow	Summer 2002	Predation	ND	ND	ND	ND	ND
33	House sparrow	Autumn 2002	Predation	ND	ND	ND	ND	ND
34	House sparrow	Summer 2003	Predation	ND	ND	ND	ND	ND
35	House sparrow	Summer 2003	Predation	ND	ND	ND	ND	ND

†ND = not detectable.

Analyses were performed in two batches with variable residue detection limits according to sample volume and equipment calibration. Greenfinches and house sparrows were chosen for analysis as these two species are among the most common granivorous garden birds in Britain, are commonly submitted for post-mortem examination and represent species that have increasing (greenfinch) and declining (house sparrow) populations (Baillie et al., 2004).

The aflatoxin analyses results were explored by cause of death and by season of death: winter (Dec–Feb), spring (Mar–May), summer (Jun–Aug) or autumn (Sept–Nov) (Table 1). Geographical location data was explored using ArcView 3.0 software (ESRI GIS and Mapping Software, 380 New York Street, Redlands, CA 92373-8100, USA).

### 3. Results

The results of the postmortem examinations are summarised in Table 1. Cause of death was categorised as, in decreasing order of occurrence: “salmonellosis”, “predation”, “other trauma” and “not established”. All birds in this study that died of infectious disease did so of salmonellosis (nine greenfinches, nine house sparrows). No other infectious cause of death was found.

The causes of death reflect those most commonly found for greenfinches and house sparrows in a review of eleven years of garden bird postmortem examinations at the Institute of Zoology (B.L. and A.A.C., unpublished results). Salmonellosis was diagnosed on the basis of gross postmortem findings consistent with

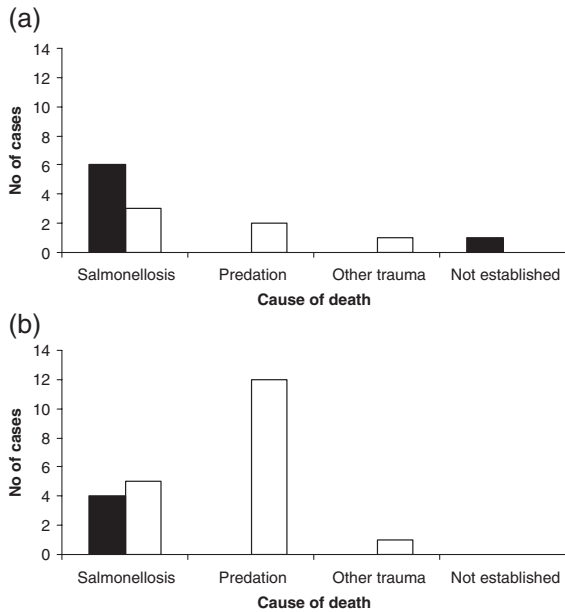


Fig. 1. Number of (a) greenfinches ( $n=13$ ) and (b) house sparrows ( $n=22$ ) screened for aflatoxin residues by cause of death. Black columns represent birds positive for residues. White columns represent birds negative for residues.

salmonellosis coupled with the isolation of *Salmonella* Typhimurium from lesions (Pennycott et al., 1998). None of the birds that died from predation or other trauma (three greenfinches, 13 house sparrows) had any evidence of infectious or other concurrent disease processes on pathological examination, including an absence of significant findings obtained on microbiological examination. These cases, therefore, have been considered together as a “non-infectious” cause of death group. The cause of death of one greenfinch could not be determined. This bird was not assigned to either the “infectious disease” or the “non-infectious” group.

Aflatoxin residues above detectable thresholds were identified in four of 22 house sparrows and in seven of 13 greenfinches examined (Table 1). Aflatoxin B<sub>1</sub> was the principal component in all but one of these eleven positive cases, with values ranging between 0.1–0.8 µg/kg. Aflatoxin B<sub>2</sub> was also detected in the two cases with the highest levels of AFB<sub>1</sub>. No gross evidence of hepatic disease consistent with either acute or chronic aflatoxicosis was observed in the birds examined.

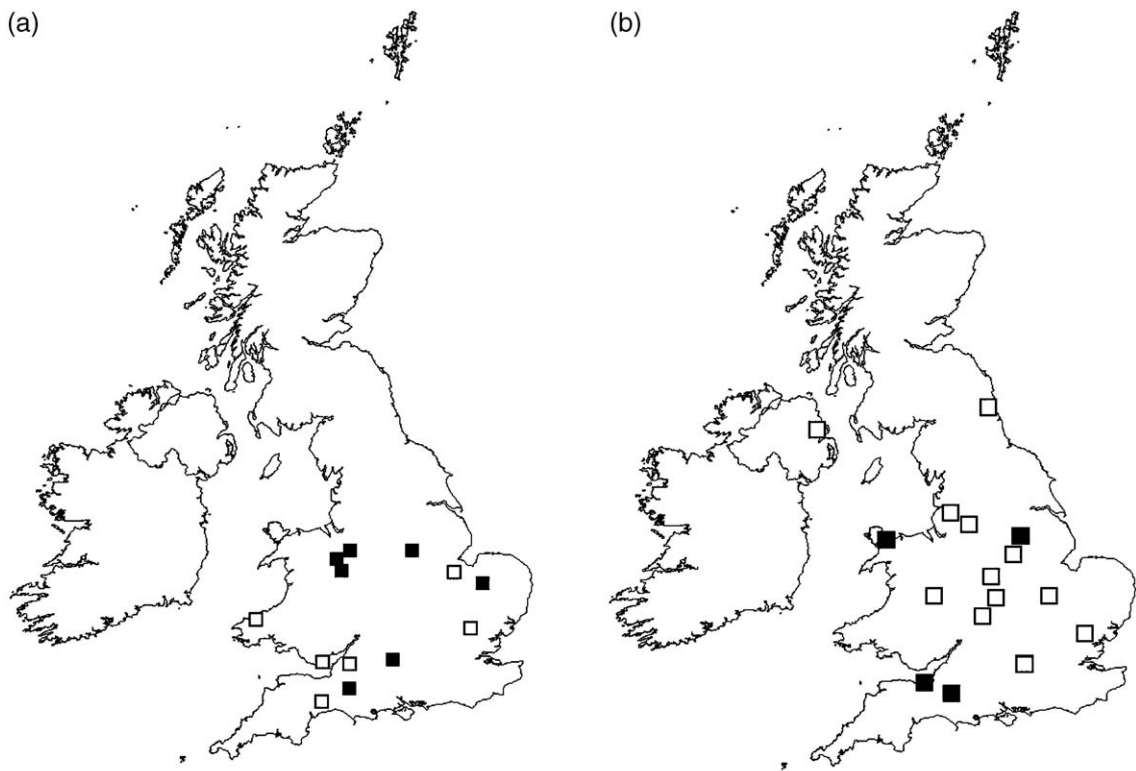


Fig. 2. Geographical distribution of (a) greenfinches ( $n=13$  locations) and (b) house sparrows ( $n=16$  locations) screened for aflatoxin residues. Each bold square represents the location of a bird that tested positive for residues. Each empty square represents the location of a bird (or birds) that tested negative for residues.

AFG<sub>1</sub> and AFG<sub>2</sub> residues were found in one greenfinch only, with a total hepatic aflatoxin level of 8.0 µg/kg. No detectable levels of AFB<sub>1</sub> or AFB<sub>2</sub> were found in this case. Unfortunately, the cause of death could not be established for this bird due to the extent of carcass decomposition; gross and microbiological examinations were negative for evidence of infectious disease, such as salmonellosis.

For both species examined, AFB<sub>1</sub> and AFB<sub>2</sub> residues were detected only in birds that had died as a result of infectious disease (salmonellosis). All tissues examined from birds in the “non-infectious disease” group (i.e., birds that had died as a result of predation or other trauma) were negative for aflatoxin residues (Fig. 1).

Only a small number of birds were examined in this study, but although there was no evidence of geographical (Fig. 2), or temporal clustering of cases with detectable aflatoxin residues there was a strong seasonal influence. Detectable levels of aflatoxin were found only in birds that died between November and February (Fig. 3). Detectable residues were found in birds that died in 1999 (1 of 1 case), 2000 (2 of 5 cases) and 2001 (8 of 15 cases), whilst no residues were detected in birds examined in 2002 (0 of 12 cases) or 2003 (0 of 2 cases).

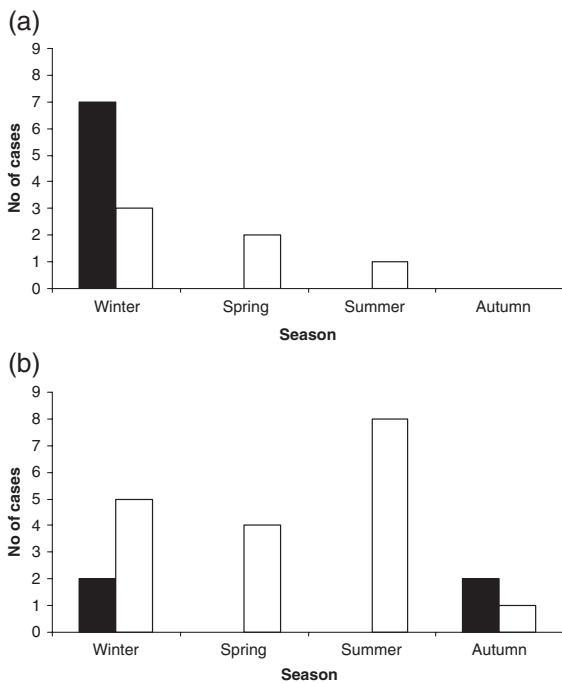


Fig. 3. Number of a) greenfinches ( $n=13$ ) and b) house sparrows ( $n=22$ ) screened for aflatoxin residues by season. Bold columns represent birds positive for residues. White columns represent birds negative for residues. Winter (Dec–Feb); Spring (Mar–May); Summer (June–Aug); Autumn (Sept–Nov).

#### 4. Discussion

Hepatic aflatoxin residues were identified in four of 22 house sparrows and in seven of 13 greenfinches examined. To the authors’ knowledge, this is the first report of wild birds being exposed to aflatoxins in Britain.

Aflatoxin B<sub>1</sub> is the most commonly occurring and the most toxic of the group of aflatoxins (Creekmore, 1999) and this was the toxin most frequently identified in the current study. The order of toxicity of the remaining aflatoxins is AFG<sub>1</sub>, greater than AFB<sub>2</sub>, and AFG<sub>2</sub> in sequence (Gourama and Bullerman, 1995). The presence of relatively high levels of AFG<sub>1</sub> and AFG<sub>2</sub> in a single greenfinch, in the absence of other residues, is of note. Aflatoxin B<sub>2</sub> was detected in the two birds with the highest levels of AFB<sub>1</sub>.

The absence of any gross evidence of toxicity and the inability to conduct meaningful histopathological examinations on the birds tested for aflatoxin exposure makes interpretation of the results difficult. There is little reported in the literature on hepatic aflatoxin levels in birds and the majority of these reports are based on experimental dosing of avian species (domestic and wild) with known dietary concentrations of aflatoxins followed by monitoring of the physiological or pathological effects observed. Little information is available regarding the relationships between dietary aflatoxin levels and resultant tissue aflatoxin residues for bird species, particularly in combination with an assessment of the aflatoxins’ pathological significance.

In some studies, however, correlations have been made between dietary aflatoxin concentrations and the subsequent concentrations of tissue residues, although often the latter are at very low levels, in the order of < several micrograms per kilogram (Gregory et al., 1983). Bintvihok et al. (2002) fed a variety of domestic poultry species with diets containing 3000 µg/kg AFB<sub>1</sub> for a 7-day period and showed that levels of AFB<sub>1</sub> and its metabolites were greater in liver than in muscle for all bird species tested. The ratio of AFB<sub>1</sub> toxin in the feed to the residual level in the liver varied between 383:1 for quail and ≥5769:1 for ducks and chickens. Madden and Stahr (1995) fed 700 µg/kg AFB<sub>1</sub> to chicks for 28 days and demonstrated that the highest levels of AFB<sub>1</sub> were found in the liver (1.29 µg/kg), followed by the crop (0.074 µg/kg) and muscle tissue (0.014 µg/kg) with an aflatoxin conversion factor from feed to liver of around 543:1. In another experiment to evaluate rates of aflatoxin residue clearance, Gregory et al. (1983) fed day-old turkey poults on a diet containing 500 µg/kg of AFB<sub>1</sub> for a period of 18 days, followed by

a variable withdrawal period on a control diet. Results showed that tissue levels of aflatoxin residue were greater in liver than in muscle tissue, although all tissue residues were at a low level (range 0.01–1.19 µg/kg). Postmortem examination of the poult on this diet revealed slight liver hypertrophy, mild petechial haemorrhages and occasional tan discolouration of the liver.

These aflatoxin feeding experiments indicate that the metabolism of dietary aflatoxin extrapolates to a much smaller corresponding level in liver tissues with a conversion ratio that is highly variable according to species and other factors. Consequently, it is likely that the levels of hepatic aflatoxin residues found in British wild birds in the current study result from a much higher dietary level of exposure.

Dietary aflatoxins are rapidly metabolised and excreted from the body, both in free and conjugated forms. For example, a study in turkey poult determined a half-life of 1.4 days for total aflatoxin residues in the liver (Gregory et al., 1983). Whilst the half-life for hepatic aflatoxins in the house sparrow and greenfinch are unknown, the rapid elimination in other avian species suggests that the birds in this study were exposed to contaminated dietary sources in the recent period prior to death. The exposure of garden birds in Britain to aflatoxins, therefore, is likely to be more frequent and widespread than our results indicate.

Chronic aflatoxin exposure has been shown to produce histopathological abnormalities such as mild biliary hyperplasia, periportal fibrosis and hepatocellular lipidosis (Pier, 1992; Ortatati et al., 2005). Appraisal of liver histopathology in tandem with aflatoxin screening would be a logical next stage in determining the significance of aflatoxin exposure in British wild birds.

It has been hypothesised that immunosuppression, predisposing to infectious disease, might be one of the most important effects of aflatoxins on wild turkeys (Quist et al., 2000). This might also be true for other free-ranging species exposed to sub-lethal doses of aflatoxins. In the current study, AFB<sub>1</sub> residues were detected only in garden birds that had died as a consequence of infectious disease (salmonellosis) and it is tempting to speculate that this could be due to aflatoxin-mediated immunosuppression. Devogowda and Murthy (2005) specifically cite increased susceptibility to salmonellosis as a consequence of subacute aflatoxicosis in poultry. However, salmonellosis has a highly seasonal occurrence in garden birds in Britain, peaking during the winter months (Pennycott et al., 1998; B.L. and A.A.C. unpublished data) and all AFB<sub>1</sub>-positive birds in the current study died between November and February.

The toxicological examination of birds that die of non-infectious causes in the winter months and of birds that die of infectious disease during the rest of the year are required in order to separate the confounding variables of infectious disease and season. Also, further research is required to determine the source of aflatoxins for garden birds in Britain. A study by Scudamore et al. (1997), for example, found evidence of aflatoxin contamination in one of fifteen samples of wild bird food screened in the UK. It might be that exposure of garden birds to aflatoxins is greater during the winter, perhaps through their increased reliance on supplementary feeding during this period. Further research is required to investigate the source of the dietary aflatoxins and their pathological significance, if any, for wild birds in Britain.

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