mango fruits (cv. Banarasi Langra, about 1 kg), infected with *A. niger* van. Tiegh (CMI-IMI 205879), were macerated with acetone in a high-speed blender. After 4 h, the mixture was filtered and the solvent was removed under reduced pressure. The extract was poured into water (200 mL) and the suspension was successively extracted with diethyl ether, ethyl acetate, and *n*-butyl alcohol (three 100-mL portions each). The three extracts were combined since their TLC behavior was similar. The combined extract was processed for neutral, phenolic, and carboxylic fractions in the usual fashion (Ghosal et al., 1978b). The phenolic fraction was shaken with a mixture of hot hexane–benzene (1:1). Evaporation of the solvent from the hexane–benzene soluble fraction afforded a yellow solid (74 mg) from which the six naphtho-γ-pyrones were obtained as described above. The yields of the compounds are recorded in Table I. The hexane–benzene insoluble fraction contained mainly gallic acid, 4-*O*-methylgallic acid, protocatechuic acid, and catechins. The identity of these compounds was established as described before (Ghosal et al., 1978a).

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Aflatoxin Residues in the Tissues of Pigs Fed a Contaminated Diet

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Pigs fed aflatoxins for 21 days had 36% heavier livers, gained 25% less weight, and ate 18% less feed than controls, but did not differ in efficiency of feed utilization or show any gross pathological lesions on postmortem examination. Assay of liver, heart, kidney, spleen, and muscle showed that there was some carry-over of aflatoxins B_1 and B_2 to all tissues, but G_1 and G_2 were not present. Residues of B_1 metabolites, M_1 and B_{2a}, were also found in all tissues of the pigs fed aflatoxins. This is the first time that B_{2a} has been identified as a tissue residue in the pig. The average retention of the aflatoxin dosage was calculated to be 0.015 and 0.005% for B_1 and B_2, respectively.

There is strong epidemiological evidence that aflatoxins are carcinogenic (Shank et al., 1972; Campbell and Stoloff, 1974). Since aflatoxins have been found to be widely distributed in common livestock feeds and produce aflatoxicosis in farm animals (Keyl and Booth, 1971), it is possible that tissue carry-over into meat may contribute to dietary carcinogenesis in man. Ingested aflatoxins may be deposited in the tissues of animals fed contaminated rations as either the original compound or as one of its metabolites (Purchase, 1972).

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One of the problems in assessing the seriousness of aflatoxins in animal products has been the availability of suitable methodology for determining tissue carry-over. Most of the early work was based on the methodology used for assaying contamination in plant materials and failed to detect aflatoxins, even in animals showing confirmed signs of aflatoxicosis (Allcroft and Carnaghan, 1963; Platanow, 1965; Kratzer et al., 1969; Keyl and Booth, 1971). Although improvements in methodology have demonstrated aflatoxin carry-over from feed to animal tissues (Brown et al., 1973; Jemmali and Murthy, 1976), the methods still lack sensitivity and accuracy so that much of the information in the literature on aflatoxins in animal products is inconclusive. Recently, Trucksess et al. (1977) reported development of an improved method for determination of aflatoxins in eggs, which has since been mod-

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Table I. Composition of the Ration[a,b]

ingredients	percentage
corn-ground	75.35
soybean meal	21.85
mineral mixture[c]	2.30
vitamin premix[d]	0.50

[a] Feed analysis: protein, 16.5%; lysine, 0.80%; methionine + cystine, 0.55%; tryptophan, 0.19%; calcium, 0.67%, and phosphorus, 0.505%. [b] Digestible energy, 3436 kcal/kg. [c] Composition of mineral mixture as percentage of diet: sodium chloride, 0.50; limestone, 1.00; dicalcium phosphate, 1.00; and the following in ppm: Se, 0.1; Zn, 74.8; Mn, 37.4; I, 2.7; Cu, 9.9; and Fe, 59.4. [d] The vitamin premix supplied the following per kilogram of ration: vitamin A, 3300 IU; vitamin D, 660 IU; vitamin E, 5.5 IU; vitamin K compound, 2.2 mg; riboflavin, 3.3 mg; niacin, 17.6 mg; D-pantothenic acid, 13.2 mg; choline, 110.0 mg; and vitamin B_{12}, 19.8 μg.

ified for quantitation of aflatoxins B_1 and M_1 in liver (Trucksess and Stoloff, 1979).

The present investigation was undertaken to ascertain the extent of aflatoxin carry-over from the ration to the tissues of the pig. This was accomplished by using a ration spiked with known quantities of aflatoxins B_1, B_2, G_1, and G_2 and assaying various tissues following slaughter.

MATERIALS AND METHODS

Feeding Trial. Eight crossbred (Duroc × Yorkshire) barrows, representing two litters and weighing from 24.5 to 26.3 kg, were randomly assigned to two groups. One group (I) served as the control and was fed a basal corn–soybean meal ration supplemented with minerals and vitamins (Table I), while the other group (II) was fed the same diet spiked with 662, 273, 300, and 285 μg/kg of aflatoxins B_1, B_2, G_1, and G_2 (Cal Biochem), respectively. The animals were individually fed ad libitum and given free access to water. They were weighed weekly and feed consumption data were recorded over a 21-day feeding period.

Tissue Samples. At the conclusion of the feeding trial, the pigs were confined without feed overnight (approximately 16 h) and slaughtered the following morning. All tissues were examined for gross lesions by a Michigan State Department of Agriculture meat inspector. Samples of heart, kidney, liver, spleen, and muscle were collected, weighed, packaged, frozen, and stored at −20 °C for aflatoxin analysis.

Extraction and Quantitation of Aflatoxins. Extraction of the tissues by the method of Jemmali and Murthy (1976) was carried out, but liver extracts were always oily and resulted in an unresolved streak without spot differentiation on the TLC plates. The procedure of Brown et al. (1973) was also investigated, but showed the same difficulties, in addition to being more complicated and time consuming. Therefore, a modification of the method of Trucksess and Stoloff (1979), which was provided to us before publication, was utilized. Although specifically designed for analysis of aflatoxins B_1 and M_1 in liver, the method proved satisfactory for all tissues. The method makes use of saturated NaCl–acetone extraction, followed by filtration. Water (150 mL), $(NH_4)_2SO_4$ (8.5 g), and $Pb(OAC)_2$ solution (35 mL) were added to the filtrate and mixed with diatomaceous earth, which was then filtered to remove the interfering substances. The filtrate containing the aflatoxin extract was purified using liquid–liquid partition by adding petroleum ether (bp 30–60 °C), followed by chloroform–acetone. The aflatoxins were located in the chloroform–acetone layer, which was drawn off and evaporated to dryness on a Rotavapor R (Buchi, Switzerland). The extract was redissolved in chloroform (10 mL) and placed on a silica gel column. Interfering substances were sequentially eluted from the column with hexane (150 mL) and anhydrous diethyl ether (100 mL), and the aflatoxins were eluted with chloroform–methanol (250 mL, 97:3). The chloroform–methanol eluate was evaporated to dryness and the aflatoxin extract was dissolved in 100 μL of chloroform.

TLC glass plates (10 × 10 cm) coated with 0.5 mm of Adsorbosil-1 (Applied Science Labs) were spotted with 20 μL of the tissue extracts and with 3, 3, and 10 μL of aflatoxin B_1, B_2, and M_1 standards, respectively. The concentration of the standards was 1.25 μg/mL for B_1, 0.25 μg/mL for B_2, and 0.5 μg/mL for M_1. The plates were developed in the first direction with chloroform–acetone–2-propanol (85:10:5) and in the second direction with anhydrous diethyl ether–methanol–water (96:3:1). The plates were dried and the spots were quantitated by densitometric analysis with a double-beam scanning-recording-integrating Schoeffel SD 3000-3 spectrodensitometer. Concentrations of aflatoxin B_1, B_2, and M_1 were calculated according to the following formula: $\mu g/kg = (B \times Y \times S \times V)/Z \times X \times W$, where B = area in sample spot, Y = concentration of standard in μg/kg, S = microliters of standard, V = dilution of sample extract in microliters, Z = area of standard spot (average 3 replications); X = microliters of sample spotted on plate, and W = weight of sample in extract in grams.

Aflatoxin standards were prepared according to the official AOAC method (1975). Confirmatory tests for aflatoxins were carried out according to the methods of Przybylski (1975) and Trucksess and Stoloff (1979). Aflatoxin B_{2a} was synthesized on the plates from B_1 according to the procedure of Trucksess and Stoloff (1979). This was used as a confirmatory test for aflatoxin B_1 and also to identify B_{2a} in the sample by superimposing it over the sample plate.

Table II. Summary of Feeding Trial: Mean Values[a]

treatment	group I controls	group II fed aflatoxins
starting wt, kg	25.8	24.3
final wt, kg	43.9[d]	37.7[e]
total gain, kg	18.1[f]	13.4[g]
total feed intake, kg	49.5[h]	36.5[i]
feed efficiency[b]	2.73	2.73
av daily intake, mg		
B_1	0[j]	1.15[k]
B_2	0[l]	0.48[m]
G_1	0[n]	0.52[o]
G_2	0[p]	0.49[q]
total daily intake, mg	0[r]	2.64[s]
daily dosage ratio[c]	0[t]	85[u]

[a] Mean values having different superscript letters on the same line differed significantly at $P < 0.01$. [b] Feed efficiency = feed/unit body weight gain. [c] Daily dosage ratio = $Wa/0.5(Ws + We)\,t$, where Wa = μg of aflatoxins ingested; t = time in days; Ws = starting wt, and We = final weight in kilograms.

RESULTS AND DISCUSSION

Results of the feeding trial are summarized in Table II. The pigs fed the ration spiked with aflatoxins had an average daily intake of 2.64 mg of aflatoxins and an average daily dosage ratio of 85 μg/kg. This level of intake has been reported to be toxic and killed pigs during a 16-week feeding period according to Armbrecht et al. (1971).

The pigs fed aflatoxins exhibited depressed growth, on average gaining 25% less over the 21-day feeding period than controls (Table II). Feeding the pigs aflatoxins also

Table III. Summary of Organ Weights in Grams and as Percentage of Body Weight[a]

	group I, control		group II, fed aflatoxins	
treatment	wt, g	%	wt, g	%
liver	990	2.26[b]	1152	3.07[c]
heart	159	0.36	156	0.42
kidneys	195	0.44	156	0.42
spleen	70	0.16	64	0.17

[a] Means followed by different superscripts are significantly different at $P = <0.01$.

resulted in an 18% reduction in feed intake, although there was no difference in feed efficiency as compared to the controls. The failure to find a difference in feed efficiency in the present study is in contrast to the results of other workers (Armbrecht et al., 1971; Keyl and Booth, 1971; Krogh et al., 1973) and is probably due to the short period of exposure as compared to the effects of the long-term feeding trials.

There was no evidence of any gross pathological lesions upon postmortem examination of the viscera and carcasses. Thus, the carcasses showed no evidence that would have justified condemnation for human food, indicating that tissues from pigs eating naturally contaminated rations would probably be approved for human consumption.

The livers of the pigs fed the aflatoxin spiked diet were 36% heavier than controls (Table III). However, there was no significant difference in the weight of the other internal organs (hearts, kidneys, and spleens). Enlargement of the livers of pigs fed aflatoxins is in agreement with reports by Armbrecht et al. (1971) and Keyl and Booth (1971). These same investigators have also observed kidney enlargement during long-time feeding, but it appeared to be less severe than that in the liver. Results of the present study confirm the suggestion by Jacobson et al. (1978) that the liver is the best internal organ for monitoring the pig's response to aflatoxins.

Results of the aflatoxin analysis for the tissues are presented in Table IV. None of the internal organs nor the muscle samples of the control pigs contained detectable aflatoxin residues. Similarly, the basal ration contained no detectable levels of aflatoxins upon analysis. However, measurable amounts of aflatoxins B_1, B_2, M_1, and B_{2a} were present in all tissues from the pigs fed the spiked ration (Table IV), except for several samples containing traces below the limit of detection (<0.05 μg/kg). Results verify the report of Jacobson et al. (1978) showing that aflatoxins B_1 and M_1 were deposited in the livers, kidneys, and muscles of pigs fed a ration containing aflatoxins.

Neither G_1 nor G_2 were detected in any of the tissue samples. Results agree with Armbrecht et al. (1972), Murthy et al. (1975), and Jemmali and Murthy (1976), all of whom were unable to find any carry-over of G_1 or G_2 in the tissues of pigs. Results indicate that aflatoxins G_1 and G_2 are readily metabolized and either eliminated or else deposited in the tissues as unidentified metabolites or as unextractable conjugates.

Aflatoxin B_{2a}, which is a metabolite of B_1, was found in appreciable amounts in heart, kidney, liver, spleen, and muscle. However, we did not quantitate it since B_{2a} standards were not available. This is the first time that aflatoxin B_{2a} has been identified in the tissues of the pig, although Chipley et al. (1974) identified B_{2a} in tissues of chickens after enzymatic digestion. B_{2a} had been previously shown to be produced in vitro on incubation of aflatoxin B_1 with hepatic tissue subfractions (Gurtoo and Campbell, 1974). It has been reported that aflatoxin B_{2a} rearranges itself to form dialdehydic phenolate resonance hybrid ions at physiological and alkaline pH values, and in this form can react with amino acids, peptides, and proteins to form Schiff bases (Gurtoo and Campbell, 1974; Ashoor and Chu, 1975). The removal of B_{2a} through transformation to the phenolate form, which then rapidly reacts with different cellular components and becomes unextractable, may explain why it has been difficult to identify B_{2a} in animal tissues. The use of strong protein and nucleic acid precipitants (ammonium sulfate and lead acetate) in combination with acidulating agents and competitive reactants (citric acid and concentrated sodium chloride solutions) in the method of Trucksess and Stoloff (1979) may be responsible for dissociation and release of aflatoxin B_{2a} by releasing the covalent bound form.

It was demonstrated herein that there is carry-over of aflatoxins B_1 and B_2 from the ration to the tissues of the pig. Furthermore, the metabolites (M_1 and B_{2a}) are formed and deposited in the various tissues. The mean percentage retention of the aflatoxin dosage was calculated to be 0.015 and 0.005% for B_1 and B_2, respectively. Although retention is low, there was carry-over into the tissues, so that meat from pigs fed aflatoxins provides another dietary source of aflatoxins. However, the amounts found in the tissue are extremely low (maximum of 1.41 μg/kg) as compared to the upper limit of 15–20 μg/kg allowed in other foods in the United States (Krogh, 1977).

SAFETY PROCEDURES

All glassware and vials in contact with aflatoxins were soaked in ammonia solution and washed with 5–6% NaOCl solution (household bleach) to destroy any residual aflatoxins. Plastic disposable gloves were worn routinely during all work with the aflatoxins. Respirator masks were worn when mixing and handling the spiked rations. Surface work areas were routinely scanned with a UV lamp and any contaminated areas were treated by washing thoroughly with a 5–6% NaOCl solution. All TLC plates and other material suspected to be contaminated were thoroughly soaked with household bleach before discarding.

Table IV. Aflatoxin Residues in Tissues of Pigs Fed Aflatoxins (μg/kg) (Group II)[a,b]

	B_1		B_2		M_1	
tissues	range	mean	range	mean	range	mean
liver	0.05-0.10	0.07	Tr-0.06	0.04	Tr-0.20	0.12
heart	0.05-1.41	0.41	Tr-0.14	0.07	0.05-0.54	0.18
spleen	Tr-0.15	0.07	Tr-0.05	0.02	Tr-Tr	Tr
muscle	Tr-0.10	0.07	Tr-0.05	0.02	Tr-0.24	0.07
kidney	0.05-0.75	0.27	Tr-0.55	0.17	NR	NR

[a] No detectable levels of any of the aflatoxins were found in any tissues from the group I (control) pigs. All tissues from group II pigs contained B_{2a} but it was not quantitated due to the unavailability of a suitable standard. [b] Tr = trace amounts, visible but too small of an amount to quantitate (<0.05 μg/kg). NR = spots could not be resolved due to the presence of interfering material.

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Volatile Nitrosamines in Various Cured Meat Products: Effect of Cooking and Recent Trends

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A study was carried out to determine the effect of cooking on the levels of volatile nitrosamines in various cured meat products. Of 64 samples tested, 39 were negative (<0.1 ppb) both before and after cooking. The majority of the positive samples contained extremely low levels (<1 ppb) of nitrosamines; the highest level detected was 8.6 ppb NPIP in a sample of spiced smoked beef. Only in two cases were significant increases in the levels of nitrosamines observed after cooking. Ten samples of baby foods containing various meat products were all found to be negative. In addition, 12 samples of fried bacon and 10 of cooked-out bacon fats were analyzed; all were positive for *N*-nitrosopyrrolidine, *N*-nitrosodimethylamine, and in some cases *N*-nitrosodiethylamine. The results indicate a continuously lowering trend in the levels of volatile nitrosamines in all types of cured meat products, except fried bacon.

The formation and occurrence of traces of volatile *N*-nitrosamines in various cured meat products have been well established (Eisenbrand et al., 1977; Fazio et al., 1973; Gough et al., 1977; Groenen et al., 1976, 1977; Sen, 1972; Sen et al., 1976a, Wasserman et al., 1972). The most commonly occurring nitrosamines in these foods are *N*-nitrosodimethylamine (NDMA), *N*-nitrosodiethylamine (NDEA), *N*-nitrosopyrrolidine (NPYR), and *N*-nitrosopiperidine (NPIP), all of which are potent carcinogens (Magee et al., 1976). Although the levels of various nitrosamines detected in most cases are in the low parts per billion range, fairly high levels (50–300 ppb) have occasionally been detected in some products, especially those prepared with the addition of spice–nitrite premixes (Sen and McKinley, 1974; Wasserman, 1978) and fried bacon and cooked-out bacon fats (Fazio et al., 1973; Gough and Walters, 1976). Among cured meat products bacon is unique in the sense that it is generally free of nitrosamines in the raw stage; nitrosamines are formed only during the high-heat frying.

Previous studies from this and other laboratories suggest that although the levels of these nitrosamines in cured meat products have been decreasing steadily during the past few years, traces are still detected (Gough, 1978; Havery et al., 1978a,b; Eisenbrand et al., 1978; Sen et al., 1977).

Fried bacon still remains a problem item which consistently contains NPYR and NDMA. Various studies have shown that the addition of nitrite-scavenging chemicals such as ascorbate, ascorbyl palmitate, α-tocopherol, etc. to bacon can significantly reduce the concentration of nitrosamines in fried bacon, but these studies are still at

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