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AFLATOXIN IN RESPIRABLE AIRBORNE PEANUT DUST

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Laboratory shelling and pilot handling operations were conducted to determine if peanut dust generated by such operations contained significant amounts of aflatoxin. Air samples were collected from points of highest dust concentration. No aflatoxin B_1 was detected in dust from uncontaminated lots. Aflatoxin B_1 levels of 700 ppb and 7.6 ng/m^3 were detected from highly contaminated lots. The contamination of the dust was directly proportional to the contamination of the lots handled or shelled. In the shelling tests, the level of contamination of the dust samples was about one-ninth of the level of contamination of the peanuts. In the handling tests, the level of contamination of the dust samples was about half the contamination level of the peanuts. These results indicate that most of the contaminated dust was probably removed by handling operations prior to shelling. Although workers are not routinely exposed to such levels of contaminated dust, these findings suggest the need for a more thorough study of peanut dust during handling or processing of contaminated peanuts.

INTRODUCTION

Since its discovery in contaminated peanut meal in the 1960s, aflatoxin has been recognized as a serious problem. The aflatoxigenic fungi are found throughout the world and contribute to the deterioration of many foodstuffs (Butler, 1974). As early as 1965, aflatoxin had been found in peanut

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samples from 13 producing countries (Allcroft, 1965), and it has been found in peanuts and peanut meals in most peanut growing states in the United States (Diener and Davis, 1969). Aflatoxin B_1 is one of the most potent naturally occurring toxins known. It is not only acutely toxic, but its mutagenic, carcinogenic, and teratogenic potential have been well documented (Butler, 1974; Ciegler, 1975; Stoloff, 1977).

Although an enormous literature has been developed since the discovery of the aflatoxins, relatively little is known of the occurrence of these substances in airborne grain or other organic dust, and virtually nothing is known of the inhalation hazard to workers and others exposed to airborne grain dust. At least two studies provide circumstantial evidence for the association of cancer in humans with inhalation of aflatoxin-contaminated dust (Dvorackova, 1976; Deger, 1976), and Van Nieuwenhuize et al. (1973) have reported an epidemiological study of workers in a peanut processing plant in the Netherlands. In the latter study, rates of multiple kinds of cancer and liver cancer combined were more than three times those reported in the matched control group. In a follow-up study in the same plant, Hayes et al. (1984) demonstrated that mortality for total cancer and respiratory cancer in the aflatoxin-exposed group of peanut oil press workers was higher than expected based on standardized mortality ratio (SMR) analysis. Recently, Richard et al. (1983) reported increased morbidity in rats exposed to aerosols of aflatoxin-containing particles. The total exposure was well below the reported LD50 values. The severity of the respons was related to the concentration of aflatoxin in the aerosol. Burg et al. (1981, 1982) and Sorenson et al. (1981) have reported the presence of aflatoxins in respirable airborne corn dust, sometimes in concentrations of several hundred parts per billion. Unlike grain, the peanut fruit grows underground, and the dust associated with peanuts is mostly dirt and quite different from grain dust.

The National Institute for Occupational Safety and Health is concerned about occupational hazards of all types. Given the potent genotoxic activity of the aflatoxins and the tendency of peanut handling and processing operations (e.g., harvesting, unloading, cleaning, and shelling) to generate a dusty environment, it is possible that peanut dust from these operations contains significant amounts of airborne aflatoxin. The present report is the result of a preliminary investigation of this question using contaminated and uncontaminated peanuts at points of maximum dust concentration.

METHODS AND MATERIALS

Peanuts were grown, harvested, dried, handled, and shelled in a commercial manner. The peanuts were dug, inverted in a windrow, and then exposed to the sun to dry for 3–7 d. The peanut fruits were then picked off the vines by mechanical harvesters and the pods were placed on 4–5-

ton wagon trailers (lots) to complete drying. After drying the peanuts were sampled and graded by the Federal State Inspection Service. Samples were removed from each load and accumulated for these tests. Samples having visible *Aspergillus flavus* mold were designated segregation III and stored and processed separately from uncontaminated peanuts (segregation I). After storage, the peanuts were dumped into the dump pit for transfer to the shelling plant. Peanuts were shelled by mechanical shellers that consist of a concave steel grate enclosing a rotating cylinder. Hulls and dust were removed from the shellers by a hood and air duct exhaust system. Impact forces and dust concentrations were highest at the dump pit and in the air-duct exhaust system. Thus, these two points were chosen for sampling, because they represented the highest probability of finding aflatoxin in peanut dust. At the dump pit, the dust was confined and directed toward the sampler (samples 23A and 24A). In the shelling process (samples 1-4, 34-36, and 38-39), described by Davidson et al. (1981), all the air used to remove the shells and dust from the peanuts was directed toward the air samplers. Peanuts lots were sampled throughout the state of Georgia to obtain peanuts for these studies. These peanuts were considered representative of crop year 1980, when at least 10% of the crop was contaminated (segregation III) with aflatoxin. In most crop years, less than 1% of the crop is contaminated and aflatoxin levels are much lower than found for the 1980 peanuts. Samples 1-4 and 23A-24A were segregation III, and samples 33-36 and 38-39 were segregation I peanuts.

Thus, these tests were set up to determine if significant aflatoxin concentrations were present in peanut dust in a worst-case situation. If significant aflatoxin concentrations were not found, then there would be no potential hazard of aflatoxin in peanut dust. If significant concentrations were found, then the relative magnitude of the potential hazard could be assessed for segregation I and III peanuts, and later studies could be conducted where potential hazards exist.

Particulate samples were collected with an Andersen four-stage high-volume cascade impactor (Andersen Samplers Incorporated, Atlanta, Georgia) with a 20 X 25-cm backup filter mounted on a standard high-volume sampler (General Metal Works, Clevis, Ohio). The sampler was operated at a flow rate of 0.0094 m^3/s (20 ft^3/min, cfm) with a manometer precalibarated at the factory for the specific impactor head to provide an equivalent pressure drop corresponding to 20 cfm, and was maintained at constant flow with an electronic flow controller (Andersen Model 700). The effective cutoff diameters for the stages are 7, 3.3, 2.0, and 1.1 μm. Particles of 1.1 μm aerodynamic diameter are collected on the final backup filter. Binder-free glass-fiber collection discs and backup filters (Andersen Samplers Incorporated) were preconditioned at least 24 h before each weighing and were weighed in a constant-temperature, constant-humidity room (70°C, 50 ± 5% relative humidity, RH). Each weight value used was the average of at least 2 independent weighings to the nearest 0.1 mg. After

sampling, each collection disc and filter was folded in quarters (sample touching sample) and held in glassine envelopes to minimize loss of sample during transport and handling. Although particles were collected by impaction rather than filtration on stages 1-4, we use the term "filter" for brevity.

All five stages of the Andersen sampler were used with samples 3, 4, 23, and 24 in order to estimate mass median diameter of the particles collected. Only the backup filter was used for the remaining samples. Multiple filters from the same sample were pooled before extraction whenever the weight of dust on individual filters was less than 100 mg.

Extraction, sample cleanup, and analysis for aflatoxin B_1 by thin-layer chromatography (TLC) were performed by the method of Shotwell et al. (1981). This method was developed for application to samples of 1-10 g and is based on methods adopted by the Association of Official Analytical Chemists (AOAC) for determining aflatoxin in corn and peanuts. Briefly, individual filters or pooled filters were extracted in an explosion-proof Waring blender with 150 ml $CHCl_3$, 15 ml water, and 15 g Celite, and extracts and washes were concentrated to 2-3 ml, transferred quantitatively to vials with $CHCl_3$, and dried under nitrogen. Cleanup was performed by column chromatography on silica gel 60 (Shotwell et al., 1981). The eluates were then dried under nitrogen and redissolved in benzene-acetonitrile (98:2). A preliminary TLC plate (10 μl of extract spotted) was run to estimate the amount of extract needed per spot for quantitation. Quantitative analysis was done by two-dimensional TLC (Shotwell et al., 1981), and aflatoxin B_1 was measured densitometrically on TLC plates with a Kratos SD 3000 Spectrodensitometer, Kratos SDC 300 Density Computer, and Waters model 730 electronic integrator. The Supelco test mixture was used to generate standard calibration curves from each TLC plate. Aflatoxin contamination of the bulk peanut lots were determined by taking 4 27-kg samples, shelling, grinding, blending, and evaluating an 1100-g sample from each sample by the standard AOAC (1980) method (TLC). The four values were averaged to obtain the mean total aflatoxin level of each lot.

RESULTS

Results of the analyses of the 12 peanut dust samples are presented in Table 1. Because the total amounts of material processed were small, sampling was restricted to $\leqslant$30 min ($\leqslant$17 m^3 air). Dust concentrations ranged from 10.5 to 65.1 mg/m^3, and airborne aflatoxin B_1 concentrations reached a maximum of 7.6 ng/m^3 air. The aerodynamic diameter of dust particles was much smaller from the sheller than the unloading operation (Fig. 1). This was expected, since the larger particles had already been removed by the cyclone and only the smallest particles were collected from the cyclone effluent. The aflatoxin content of the dust was directly proportional to the aflatoxin contamination of the lot of bulk peanuts. No

TABLE 1. Aflatoxin in Airborne Peanut Dust

Sample number[a]	Source	Sampling time (min)	Weight of dust (mg)	Dust conc. (mg/m^3)	Aflatoxin B_1 conc. (ppb)	Airborne aflatoxin B_1 conc. (ng/m^3)	Total aflatoxin *in bulk peanuts* (ppb)
1	Sheller	7.0	56.3	14.3	72.4	1.0	376
2	Sheller	7.1	58.9	14.7	24.2	0.4	300
3	Sheller[b]	8.5	79.7	16.6	76.4	1.3	483
4	Sheller[b]	10.2	99.1	17.2	22.7	0.4	209
33	Sheller	7.4	80.5	19.3	ND[f]	—	~0
34	Sheller	5.7	33.8	10.5	ND	—	~0
35	Sheller	7.2	84.5	20.8	ND	—	~0
36	Sheller	7.0	102.5	26.0	ND	—	~0
38	Sheller	11.1	111.2	17.8	ND	—	~0
39	Sheller	8.0	133.2	29.5	ND	—	~0
$23A_1$	Unloading[c,e]	15.0	355.7	42.0	70.7	3.0	—
$23A_2$	Unloading	15.0	101.8	12.0	52.4	0.6	—
$23A_4$	Unloading	15.0	95.9	11.3	50.6	0.6	—
23A	(Total)	15.0	553.4	65.1	—	—	<136
$24A_1$	Unloading[d,e]	30.0	176.6	10.4	730.8	7.6	—
$24A_2$	Unloading	30.0	47.5	2.8	612.4	1.7	—
$24A_4$	Unloading	30.0	62.6	3.7	198.6	0.7	—
24A	(Total)	30.0	286.7	17.0	—	—	1020

[a]Filter sets were prelabeled and used as needed without regard to numerical sequence. All samples collected are included in the table.
[b]Stages 1-4 plus backup filter pooled before extraction.
[c]Unloading from drying trailers; 3 loads.
[d]Unloading from drying trailers; 6 loads.
[e]A_1 = stage 1; effective cutoff diameter = 7.0 μm. A_2 = stages 2 and 3 pooled; effective cutoff diameter = 2.0 μm. A_4 = stage 4 and backup filter pooled; effective cut-off diameter < 1.1 μm.
[f]*Not detected.*

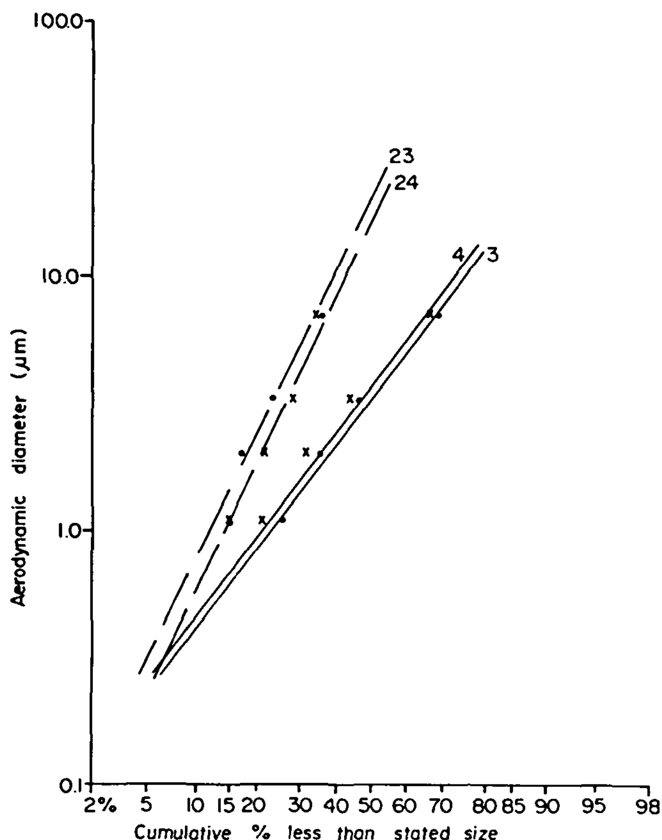

FIGURE 1. Size distribution of particles collected by the high-volume Andersen sampler: sample 3, 4, 23, and 24. The estimated mass medium diameters were 3.4, 3.9, 18.0, and 22.0 μm for samples 3, 4, 23, and 24, respectively.

aflatoxin was found in the dust from uncontaminated peanuts. The dust collected from the handling and shelling test had about half and one-ninth as much aflatoxin, respectively, as the bulk peanuts. Aflatoxin analyses of the hulls and heavy dust removed by the cyclone showed only slight traces (not reported) of aflatoxin for the highest contaminated lots. Thus it appears that most of the contaminated dust was removed prior to shelling.

DISCUSSION

The objective of this preliminary investigation was to determine whether there is reason to consider exposure to airborne peanut dust containing aflatoxin a potential hazard to workers in the United States. A previous study (Van Nieuwenhuize et al., 1973) suggested that elevated incidence of

liver cancer and other cancers occurred in Dutch workers in a peanut processing plant in the Netherlands. Our data suggest that aflatoxin in peanut dust represents a potential hazard only with a small percentage (segregation III peanuts) of the total peanut crop. There may be a hazard at the dump pit when handling segregation III peanuts, since there was a relatively high ratio of aflatoxin in the peanut dust to that in the peanuts and because the dust is discharged at ground level. The hazard in the shelling operation is probably significantly less because most modern commercial operations use negative-pressure air-duct systems with high-efficiency cyclones to discharge the dust outside the plant at 4.6 m above ground level.

The results clearly show that airborne peanut dust from contaminated lots contains aflatoxin B_1. If one assumes a breathing rate of 1 m^3/h and an airborne aflatoxin concentration of 0.2 ng/m^3 (20 ppb at a dust concentration of 10 mg/m^3), a worker would inhale 1.6 ng in an 8-h workshift and 8.0 ng in a 40-h workweek. At higher dust concentrations and higher levels of contamination, the levels of exposure would be correspondingly higher (e.g., 100 ppb at 20 mg/m^3; 2.0 ng/m^3, 16 ng/8 h, and 80 ng/40 h). At the present time, there is a paucity of information concerning the risk of inhaling such amounts of aflatoxin, but the extreme genotoxic potential of aflatoxin suggests that the risk may be real. Richard et al. (1983) exposed Wistar male weanling rats to aerosols of killed *Aspergillus fumigatus* spores alone or to killed *A. fumigatus* spores containing 1000 or 5000 ppm AFB_1. The animals were exposed for 2 h/d and 5 d/wk for 4 consecutive weeks. Necropsy was performed on a portion of the animals in each group 3 wk postexposure and on the remainder 1 yr postexposure. Hepatic lesions were only observed in rats exposed to 1000 or 5000 ppm AFB_1, and only 1 neoplasm was observed (a localized biliary adenoma in the liver parenchyma). With only 8 animals per group, it was not possible to determine whether this level of exposure resulted in an increase in the hepatic cancer rate. No lung lesions were observed 1 yr after exposure to aflatoxin-free spores or in unexposed animals, but lung lesions of varying severity were observed in 5 of the 8 rats exposed to 1000 ppm AFB_1 and were more marked in rats exposed to the higher level of aflatoxin. The authors estimate that the animals exposed to 5000 ppm AFB_1 received a total exposure of 0.006 mg/kg. This is well below the reported LD50 dose of 6.0 mg/kg (ip) for AFB_1 in the male rat (Butler, 1964). The authors believe that the general response of the exposed animals appeared to be that of a compromised host. Enna and Schanker (1972) reported that the retention time of chemicals deposited in the lung is inversely related to their partition coefficient in chloroform, i.e., the rate of uptake is greatest if the partition coefficient in chloroform is high. Aflatoxin is readily extracted from aqueous solutions with chloroform and can be expected to be absorbed rapidly from the lung. In this discussion, we have not attempted to estimate the amount of aflatoxin that would be excluded

by the respiratory system, because inhaled particles too large to reach the alveolar spaces would be transported via the mucociliary escalator to the esophagus and thus would contribute to total body exposure.

Sorenson et al. (1981) presented data on a sample of Georgia corn dust. When this sample was aerosolized in a closed system within a safety cabinet and samples were taken with an Andersen sampler (1 cfm), aflatoxins levels were much higher in the size fraction $<7\ \mu m$ than the $>7\ \mu m$ fraction. This finding suggested the possibility of size-dependent distribution of aflatoxin in corn dust particles. The data obtained in the present study, although obtained from a limited number of samples, do not support the hypothesis of size-dependent distribution. Such a distribution pattern has been documented in the case of volatile chemicals, which may condense on the surface of particles (Sorenson et al., 1982).

REFERENCES

Allcroft, R. 1965. Aspects of aflatoxicosis in farm animals. In *Mycotoxin in Foodstuffs*, ed. G. N. Wogan, pp. 153–162. Cambridge: Massachusetts Institute of Technology Press.

AOAC © 1980. *Official Methods of Analysis*, 13th ed. Secs. 26.049, 26.026–26.031. Arlington, Va.: Assoc. Offic. Anal. Chem.

Burg, W. R., Shotwell, O. L., and Saltzman, B. E. 1981. Measurements of airborne aflatoxins during the handling of contaminated corn. *Am. Ind. Hyg. Assoc. J.* 42:1–11.

Burg, W. R., Shotwell, O. L., and Saltzman, B. E. 1982. Measurements of airborne aflatoxins during the handling of 1979 contaminated corn. *Am. Ind. Hyg. Assoc. J.* 43:580–586.

Butler, W. H. 1964. Acute toxicity of aflatoxin B_1 in rats. *Br. J. Cancer* 18:756–762.

Butler, W. H. 1974. Aflatoxins. In *Mycotoxins*, ed. I. F. H. Purchase, pp. 1–28. Amsterdam: Elsevier.

Ciegler, A. 1975. Mycotoxins: Occurrence, chemistry, biological activity. *Llyodia* 38:21–35.

Davidson, J. I. Jr., Hudgins, R. F., Bennett, C. T. 1981. Some design and performance characteristics of a small peanut sheller. *Oleagineux* 36:433–438.

Deger, G. E. 1976. Aflatoxin—Human colon carcinogenesis. *Ann. Intern. Med.* 85:204.

Diener, U. L., and Davis, N. D., 1969. Aflatoxin formation by *Aspergillus flavus*. In *Aflatoxin, Scientific Background, Control and Implications*, ed. L. A. Goldblatt, pp. 18–19. New York: Academic.

Dvorackova, I. 1976. Aflatoxin and alveolar cell carcinoma. *Br. Med. J.* 3:691.

Enna, S. J., and Schanker, L. S. 1972. Absorption of drugs from the rat lung. *Am. J. Physiol.* 223: 1227–1231.

Hayes, R. B., Van Nieuwenhuize, J. P., Raatgever, J. W., and Ten Kate, F. J. W. 1984. Aflatoxin exposures in the industrial setting: An epidemiological study of mortality. *Food Chem. Toxicol.* 22:39–43.

Richard, J. L., Cheville, N. F., Songer, J. R., and Thurston, J. R. 1983. Exposure of rats to aerosols of aflatoxin-containing particles. In *Proc. 8th International Congress of Human and Animal Mycology*, ed. M. Baxter, pp. 464–468. Palmerstown, New Zealand: Massey University Press.

Shotwell, O. L., Burg, W. R., and Diller, T. 1981. Thin layer chromatographic determination of aflatoxin in corn dust. *J. Assoc. Offic. Anal. Chem.* 64:1060–1063.

Sorenson, W. G., Simpson, J. P., Peach, M. J. III, Thedell, T. D., and Olenchock, S. A. 1981. Aflatoxin in respirable corn dust particles. *J. Toxicol. Environ. Health* 7:669–672.

Sorenson, W. G., Whong, W-Z., Simpson, J. P., Hearl, F. J., and Ong, T. 1982. Studies of the mutagenic response of *Salmonella typhimurium* TA98 to size-fractionated air particles: Comparison of the fluctuation and plate incorporation tests. *Environ. Mutagen.* 4:531–541.

Stoloff, L. 1977. Aflatoxins—An overview. In *Mycotoxins in Human and Animal Health*, eds. J. V. Rodricks, C. W. Hesseltine, and M. A. Mehlman, pp. 7-28. Park Forest South, Ill.: Pathotox.

Van Nieuwenhuize, J. P., Herber, R. F. M., DeBruin, A., Meyer, I. P. B., and Duba, W. C. 1973. Aflatoxins—Epidemiological study on the carcinogenicity of prolonged exposure to low levels among workers of a plant. *Tijdschr. Soc. Geneeskd.* 51:754-759.

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