

Accumulation of Organochlorine Pesticides in Poultry: A Review

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Accumulation ratios (level of pesticide in fat or egg to its level in the feed) of hexachlorobenzene (HCB), α -, β -, and γ -hexachlorocyclohexane (HCH), heptachlor and its epoxide, DDT, dieldrin, aldrin, endrin, and methoxychlor in laying hens and broilers are discussed. The pesticides can be grouped into several categories according to accumulation ratios: highly accumulating like HCB, β -HCH, heptachlor epoxide, dieldrin, aldrin; intermediate like heptachlor, endrin; low like α - and γ -HCH; and very low like methoxychlor. Depletion of residues of the pesticides in laying hens and eggs is correlated with the accumulative properties. Depletion of residues in broilers is mostly governed by growth rate and thus by dilution in the fat.

Because of the use of organochlorine pesticides in growing and storing agricultural commodities, one can encounter residues in plant products. When present in poultry feed, there are two problems to be faced. Firstly, the pesticides can have a negative influence of the performance of poultry. This aspect has received much attention in the past, and numerous reviews have been written, e.g., Foster (1974) and Naber (1977). Secondly, the organochlorine pesticides are persistent and residues can accumulate in fat and eggs. This fact has often been demonstrated. Naber (1977) is one of the few to review this field.

As residues are undesirable in products of animal origin and thus in animal feed, legislation has set separate maximum tolerable contents for the two types of product. Unfortunately, these tolerances have been developed quite often along independent lines, mostly influenced by practical residue limits. This can lead to the undesirable situation that a tolerable level of a certain pesticide in poultry feed gives rise to an intolerable level of that pesticide in body fat or eggs. Or, in other words, both tolerances should at least be separated by the accumulation ratio of the pesticide concerned.

To discover these possible inconsistencies in pesticide legislation, we tested a mixture of several organochlorine pesticides on different types of poultry: broiler breeders, broilers, and laying hens (Kan and Tuinstra, 1976; Kan and Jonker-den Rooyen, 1978a,b; Kan et al., 1978).

Since our data apply to certain experimental conditions, it is quite probable that other conditions produce other values. Several known possibilities will be described. (1) The influence of laying percentage changed by Ca level in the diet (Cecil et al., 1973) or age of the birds (Kan and Tuinstra, 1976; Kan and Jonker-den Rooyen, 1978a) on accumulation in both eggs and fat has been clearly demonstrated. (2) Liska et al. (1964) and Stemp (1965) found an influence of amount and type of fat in the diet on accumulation of DDT. (3) Ritchey et al. (1967) detected an interaction between DDT and lindane during residue formation in broilers, although de Vos et al. (1972) could not find similar effects. (4) External factors like soil contamination (Putnam et al., 1974), HCB in woodshavings (Kan and Tuinstra, 1976), or HCB residues in a laying house (Kan and Jonker-den Rooyen, 1978b) will also influence residue found. As the necessary details are lacking in most reports, these and other possible factors cannot be completely taken into consideration in the following discussions. However, most of these factors may be present in practice too, so their influence cannot be

excluded in practical residue formation.

The aim of this review is, therefore, to examine the recent data from literature. These data can then be used to reveal possible inconsistencies between tolerances in poultry feed and poultry products. Our own results have already demonstrated several inconsistencies in current Dutch regulations (Kan, 1978). Other research has done the same in other countries, e.g., Reed et al. (1977) for HCB in the United States.

The following organochlorine pesticides will be discussed: hexachlorobenzene, α -, β -, and γ -hexachlorocyclohexane, heptachlor (epoxide), DDT, dieldrin, aldrin, endrin, and methoxychlor. The literature data are given in Tables I, II, and III. Attention will also be paid to diminution of residues after contamination has ceased.

HEXACHLOROBENZENE

Eggs. Table I shows the accumulation ratios found for HCB in eggs. The data from Avrahami and Steele (1972a) were much higher than other reported data. In this experiment there were, however, three complicating factors. Firstly, HCB was added to the feed as a commercial mixture containing other substances also, and no check on actual feed residues is reported. Secondly, the number of birds used was small, so individual variation may have played a role. Thirdly, the background contamination in the control group is rather high in relation to the lowest dosed group (highest accumulation). Thereby a correction for this contamination is rather tricky. Furthermore, actual plateauing in residues was never reached in that experiment.

Abdominal Fat. Laying Hens. Table II shows that once again the values of Avrahami and Steele (1972a) are rather high. The remarks for values in eggs apply here too.

Broilers. Table III shows high values from Avrahami and Steele (1972b). Besides the peculiarities in a commercial mixture and high background, this experiment lasted 26 weeks whereas the others lasted 6–8 weeks. This might have influenced values.

α -HEXACHLOROCYCLOHEXANE

Eggs and Abdominal Fat. The low accumulating potency of this isomer is clearly demonstrated in Tables I, II, and III.

β -HEXACHLOROCYCLOHEXANE

Eggs and Abdominal Fat. The β isomer of hexachlorocyclohexane, in which all Cl atoms are in the equatorial position of the chair conformation of the cyclohexane ring, has the highest accumulation ratio of the three isomers tested. Tables I, II, and III demonstrate this fact clearly.

γ -HEXACHLOROCYCLOHEXANE (LINDANE)

Eggs. Table I shows that in all experiments a low accumulation ratio similar to the α isomer is found.

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Table I. Accumulation Ratios in Eggs (Level of Pesticide in the Egg to Its Level in the Feed)

	HCB	α -HCH	β -HCH	γ -HCH	hepta-chlor epoxide	DDT total	dieldrin	al-drin	en-drin
A. whole egg basis									
Avrahami and Steele (1972a)	5.5								
Balasubramaniam (1971)						0.8	0.8		
Brown et al. (1965)							2		
Brown et al. (1974)							1.6-1.8		
Cecil et al. (1972)						1-1.2			
Cecil et al. (1973)						1-1.4			
Combs and Brewer (1975)					2.9	1.6	2.5		
Cummings et al. (1966)				0.2		1.0	1.1	1.1	0.6
Driver et al. (1976)					2.1		1.3	2.2	
Dy et al. (1970)							0.9		
Foster et al. (1972)				0.2			0.9		
Graves et al. (1969)							0.7-1.7		
Kan and Tuinstra (1976)	1.3	0.10	1.5	0.13	0.5	1.2	1.3		
Kan and Jonker-den (1978a)	1.9	0.16	2.3	0.2	0.7	1.6	1.7		
Liska et al. (1964)						0.8			
Noakes and Benfield (1965)						0.3-0.4			
Singh et al. (1970)						0.3-0.5			
Smith et al. (1970)						0.3			
Waldron and Naber (1974)				0.2	0.5	0.9	1.5	1.2	
Wisman et al. (1967)					0.8				
Zabik (1970)				0.3		0.8	1.2		
B. fat basis									
Kan and Jonker-den (1978a)	16	1.4	20	1.8	6	14	15		
Kan and Jonker-den (1978b)	11	2	13	2	5	10	11		
Vogt et al. (1975)	11								

Table II. Accumulation Ratios in Abdominal Fat of Laying Hens (Level of Pesticide in the Fat to Its Level in the Feed)

	HCB	α -HCH	β -HCH	γ -HCH	hepta-chlor epoxide	DDT total	dieldrin	al-drin	en-drin
Avrahami and Steele (1972a)	21-31								
Brown et al. (1965)							14	11	
Brown et al. (1974)							12-14		
Cecil et al. (1972)						13-14			
Cecil et al. (1973)						15-19			
Cummings et al. (1967)				2		11.5	9	11.5	9
Davison et al. (1970)							15		
Foster et al. (1972)				0.7		8-10			
Kan and Tuinstra (1976)	17	1.8	18	1.8	6	14	14		
Kan and Jonker-den (1978a)	19	1.8	25	2	7	18	17		
Kan and Jonker-den (1978b)	13	2	15	2	7	12	14		
Liska et al. (1964)						5			
Morgan et al. (1972)				1.5		4	3		
Noakes and Benfield (1965)						10-12			
Singh et al. (1970)						3-10			
Smith et al. (1970)						3-12			
Waldron and Naber (1974)				2-3	3-5	8	15	12	
Zabik and Funk (1971)				1		3	2.5		

Abdominal Fat. Laying Hens. Table II shows a low accumulation ratio of γ -HCH in the experiment of Foster et al. (1972). As they used few hens, this may be due to chance. The same applies to the study of Zabik and Funk

(1971). In none of those studies are data on actual feed residues reported.

Broilers. Table III shows a good agreement between published data on γ -HCH in broilers.

Table III. Accumulation Ratios (Level of Pesticide in Fat to Its Level in the Feed) in Abdominal Fat of Broilers

	HCB	α -HCH	β -HCH	γ -HCH	hepta-chlor	hepta-chlor epoxide	DDT total	dieldrin	aldrin	endrin
Avrahami and Steele (1972b)	20-30									
Combs and Brewer (1975)					30		30	47		
Donaldson et al. (1968)							7-15			
Kan et al. (1978)	11	3	14	2	5		10	11		
Liska et al. (1964)							6-8			
Olney et al. (1975)						16-20	12	15-17		7-10
Putnam et al. (1974)					40		30	70		
Reed et al. (1977)	11-18									
Ritchey et al. (1967)				2			6-15			
Ritchey et al. (1969)							10			
Ritchey et al. (1972)				2	8			13	14	7
Siegel et al. (1976)								10-13		
de Vos et al. (1972)	12			3.3		13	11	11		10

HEPTACHLOR AND ITS EPOXIDE

Although heptachlor and its epoxide differ considerably in their accumulation behavior when given as such, they will be discussed together as it is not always clear which compound was in the feed.

Eggs. Table I shows that rather high values were found by Combs and Brewer (1975) and Driver et al. (1976). It is not clear whether the former used heptachlor or its epoxide, but the latter clearly used heptachlor. There are two possible explanations: In both reports the level in the feed was low and hard to estimate accurately. There may also be a deviation from the general rule that accumulation is independent of level used.

Abdominal Fat. Laying Hens. The data in Table II clearly show the difference in accumulation between heptachlor, converted in the body to β -heptachlor epoxide (Kan et al., 1978) and heptachlor epoxide, when administered as such.

Broilers. Table III shows large differences in accumulation as determined by different authors. Again it is not clear whether Combs and Brewer (1975) used heptachlor or its epoxide. Nevertheless, the low levels used by Combs and Brewer (1975) and Putnam et al. (1974) could cause uncertainties.

The difference in accumulation of heptachlor epoxide found by de Vos et al. (1972) and Olney et al. (1975) may be caused by the use of different isomers of the epoxide. There is no indication in their papers on this point but α -heptachlor epoxide, which is commercially more easily available than the β isomer, formed in nature may have been used in one of the studies.

DDT

Eggs. Table I shows a wide range of accumulation ratios for DDT. They are all calculated (if possible) on *p,p'*-DDT in the feed and *p,p'*-DDT + *p,p'*-DDE in the product. The range is partly explicable by the influence of egg production as shown by Cecil et al. (1973) and our own experiments (Kan and Tuinstra, 1976; Kan and Jonker-den Rooyen, 1978a). The low values of Singh et al. (1970) and Smith et al. (1970) are probably due to the use of a mixture of *p,p'*-DDT and other isomers or metabolites, but no details on this point are available. It is known that *o,p'*-DDT accumulates to a much lesser extent than its *para,para'* isomer (Cummings et al., 1966). In the study of Noakes and Benfield (1965), only a few eggs at the beginning of the laying period were analyzed, so we cannot treat their data too seriously.

Abdominal Fat. Laying Hens. Also in Table II, one can see the influence of egg production on accumulation. The highest values are from those experiments where egg production was low. The low values of Liska et al. (1964), Singh et al. (1970), and Smith et al. (1970) were probably

caused by the use of crude DDT, as discussed before. Few hens were used by Zabik and Funk (1971) and Morgan et al. (1972). Another factor might be that in all these studies, plateauing of residues was not sought and therefore not demonstrated. It might be possible that in some instances the plateau was not yet reached at the time of sampling, so the value was too low.

Broilers. Table III shows a wide range of accumulation ratios for DDT. The low values found by Liska et al. (1964) and Ritchey et al. (1967) may be due to the use of crude DDT. The high value found by Combs et al. (1975) and Putnam et al. (1974) may have been due to the levels in feed being low for analysis or to greater accumulation at those low levels.

DIELDRIN

Eggs. Table I shows the tendency of dieldrin to accumulate. The highest figures are those of Brown et al. (1965), who used only one hen, and Combs et al. (1975) and Driver et al. (1976). The problems of the low levels used in the last two experiments have been discussed above.

Abdominal Fat. Laying Hens. There is good agreement in accumulation ratios shown in Table II. The exceptions are the experiments of Zabik and Funk (1971) and Morgan et al. (1972), who used few hens and did not look for plateauing of residues.

Broilers. In contrast to laying hens, rather divergent values have been found in broilers (Table III). The two extremes are Combs et al. (1975) and Putnam et al. (1974). From the paper of Combs et al. (1975), no plateauing up to 8 weeks of age was evident and they only took the last measurement, which could therefore be deviant. The values of Putnam et al. (1974) are complicated by two facts. Firstly, there was interaction in residue formation with contaminated soil. Secondly, a mixture of aldrin and dieldrin was present in the feed and aldrin being metabolized to dieldrin. No separation can be made.

ALDRIN

Eggs and Abdominal Fat. Tables I, II, and III show that the accumulating properties of aldrin are almost identical with that of dieldrin.

ENDRIN

Eggs and Abdominal Fat. Tables I, II, and III show a good agreement in accumulation data of endrin, which has a lower ratio than dieldrin or aldrin.

METHOXYCHLOR

Lillie et al. (1973), Olney et al. (1962), and Thompson et al. (1967) detected minute amounts of methoxychlor in eggs and fat after feeding on high levels. Waldron and Naber (1974) and Olney et al. (1975) did not detect any

Table IV. Half Value-Times (Weeks) in Eggs and Abdominal Fat of Laying Hens

		HCB	α -HCH	β -HCH	γ -HCH	hepta- chlor epoxide	DDT	dieldrin	endrin
Avrahami and Steele (1972a)	egg	11							
Cummings et al. (1966)	egg				1.5-2	>8		>6	4-5
Cummings et al. (1967)	fat				1.5-2	4	>6	>6	4-5
Driver et al. (1976)	egg					8			
Kan and Tuinstra (1976)	egg	11							
	fat	9							
Kan and Jonker-den Rooyen (1978a)	egg	n.m. ^a	1.5-2	n.m.	1.5-2	n.m.	n.m.	n.m.	
	fat	n.m.	1.5-2	n.m.	1.5-2	n.m.	n.m.	n.m.	
Kan and Jonker-den Rooyen (1978b)	egg	8	1.5-2	7	1.5-2	6.5	7	6	
	fat	7	1.5-2	7	1.5-2	5	6	6	
Lillard and Noles (1973)	egg						3.8-7.6		
	fat						2.5-7.3		
Mick et al. (1973)	egg							4	
Wesley et al. (1966)	egg						6-7		
	fat						6-7		
Wesley et al. (1969)	egg						6		
	fat						10		
Wisman et al. (1967)	egg					8			

^a n.m. = not measurable.

residues after feeding on low levels. The accumulation ratios calculated are of the order of a tenth to a hundredth of those of γ -HCH.

HALF-VALUE TIMES IN LAYING HENS

The assumption is made that first-order kinetics hold.

Hexachlorobenzene. Table IV shows the relatively long half-value time of hexachlorobenzene. This half-value time depends on egg production and thus on excretion through eggs as it was shortest in high-producing hens. On the contrary, in low-producing hens no real depletion was detected (Kan and Jonker-den Rooyen, 1978a).

Hexachlorocyclohexane. There are only a few data on HCH isomers, and Table IV shows that these data are nicely correlated with the accumulation behavior of the different isomers. Half-value times of α - and γ -HCH are, however, independent of egg production and thus of excretion through eggs. So metabolism is the major factor in disappearance.

Heptachlor Epoxide. Table IV shows divergent data for calculated half-value times of heptachlor epoxide. However, within one experiment data are reasonably consistent, except in the experiment of Cummings et al. (1966, 1967). There is no obvious explanation for the clear decrease in residues in fat, whereas residues in eggs decreased only marginally.

DDT. Data on DDT are consistent also. The lower values of Lillard and Noles (1973) in Table IV are attributable to special treatment like starvation or thyroxine treatments which were effective in shortening half-value times. On the other hand, Wesley et al. (1966, 1969) reached only marginal effects with starvation, androgen treatment, or different protein contents in the diet.

Dieldrin. Table IV shows rather long half-value times for dieldrin too. Mick et al. (1973) could not shorten these by phenobarbital or charcoal.

Endrin. The somewhat lower value for endrin is in line with its accumulative properties.

HALF-VALUE TIMES IN BROILERS

The diminution of pesticide residues in broilers has been studied by several authors. Donaldson et al. (1968, 1971) studied the influence of periodic starvation or iodinated casein on DDT residues. Both treatments can enhance conversion of DDT to DDE and elimination of DDT from the body. Polin and Ringer (1975) administered diphenylhydantoin to broilers while studying DDT depletion. The half-value time remained unchanged at 16 days. This

half-value time is comparable to that of dieldrin reported by Combs et al. (1975), 12 days, and Siegel et al. (1976), about 2 weeks. In all cases, dilution in the growing broiler is the main factor in diminution although some metabolism or excretion must have taken place (Combs et al., 1975; Kan et al., 1978). The influence of growth rate can also be deduced from the somewhat longer half-value times to be calculated from research by Onley et al. (1975), carried out in 1967, since growth rates in their trial could be considerably less than in recent years.

LITERATURE CITED

- Avrahami, M., Steele, R. T., *N.Z. J. Agric. Res.* **15**, 482-488 (1972a).
 Avrahami, M., Steele, R. T., *N.Z. J. Agric. Res.* **15**, 489-494 (1972b).
 Balasubramaniam, A., Ph.D. Thesis, Louisiana State University, Baton Rouge, La., 1971.
 Brown, V. K., Richardson, A., Robinson, J., Stevenson, D. E., *Food Cosmet. Toxicol.* **3**, 675-679 (1965).
 Brown, V. K. H., Robinson, J., Thorpe, E., Barrett, J. W., *Pestic. Sci.* **5**, 567-586 (1974).
 Cecil, H. C., Fries, G. F., Bitman, J., Harris, S. J., Lillie, R. J., Denton, C. A., *Poult. Sci.* **51**, 130-139 (1972).
 Cecil, H. C., Bitman, J., Fries, G. F., Harris, S. J., Lillie, R. J., *Poult. Sci.* **52**, 648-653 (1973).
 Combs, G. F., Brewer, R. N., Proceedings of the 1975 Mld. Nutrition Conference, 1975 pp 14-18.
 Combs, G. F., Brewer, R. N., Williams, N. K., Richburg, R. W., *Poult. Sci.* **54**, 1713-1716 (1975).
 Cummings, J. G., Zee, K. T., Turner, V., Quinn, F., Cook, R. E., *J. Assoc. Off. Anal. Chem.* **49**, 354-364 (1966).
 Cummings, J. G., Eidelman, M., Turner, V., Reed, D., Zee, K. T., Cook, R. E., *J. Assoc. Off. Anal. Chem.* **50**, 418-425 (1967).
 Davison, K. L., Sell, J. L., Rose, R. J., *Bull. Environ. Contam. Toxicol.* **5**, 493-501 (1970).
 Donaldson, W. E., Sheets, T. J., Jackson, M. D., *Poult. Sci.* **47**, 237-243 (1968).
 Donaldson, W. E., Jackson, M. D., Sheets, T. J., *Poult. Sci.* **50**, 1316-1320 (1971).
 Driver, D., Brewer, R. N., Cottier, G. J., *Poult. Sci.* **55**, 1544-1549 (1976).
 Dy, K. B., Mountney, G., Day, C., Green, E., *Food Technol.* **24**, 1308-1311 (1970).
 Foster, T. S., Morley, H. V., Purkayastha, R., Greenhalgh, R., Hunt, J. R., *J. Econ. Entomol.* **65**, 982-988 (1972).
 Foster, T. S., *Res. Rev.* **51**, 69-121 (1974).
 Graves, J. B., Bonner, F. L., McKnight, W. F., Watts, A. B., Epps, E. A., *Bull. Environ. Contam. Toxicol.* **4**, 375-383 (1969).
 Kan, C. A., Tuinstra, L. G. M. Th., *J. Agric. Food Chem.* **24**, 772-775 (1976).

- Kan, C. A., IVth Congr. Pest. Chem., Zürich, July 1978 (comparison of current Dutch regulations on organochlorine pesticides in poultry feed and poultry products).
- Kan, C. A., Jonker-den Rooyen, J. C., *J. Agric. Food Chem.* **26**, 465-470 (1978a).
- Kan, C. A., Jonker-den Rooyen, J. C., *J. Agric. Food Chem.* **26**, 935 (1978b).
- Kan, C. A., Jonker-den Rooyen, J. C., Tuinstra, L. G. M. Th., Roos, A. H., Traag, W., *J. Agric. Food Chem.* **26**, 618 (1978).
- Lillard, D. A., Noles, R. K., *Poult. Sci.* **52**, 222-228 (1973).
- Lillie, R. J., Cecil, H. C., Bitman, J., *Poult. Sci.* **52**, 1134-1138 (1973).
- Liska, B. J., Langlois, B. E., Mostert, G. C., Stadelman, W. J., *Poult. Sci.* **43**, 982-984 (1964).
- Mick, D. L., Long, K. R., Aldinger, S. M., *Bull. Environ. Contam. Toxicol.* **9**, 197-203 (1973).
- Morgan, K. J., Zabik, M. E., Funk, K., *Poult. Sci.* **51** 470-475 (1972).
- Naber, E. C., *Fed. Proc., Fed. Am. Soc. Exp. Biol.* **36**, 1880-1887 (1977).
- Noakes, D. N., Benfield, C. A., *J. Sci. Food Agric.* **16**, 693-697 (1965).
- Olney, C. E., Donaldson, W. E., Kerr, T. W., *J. Econ. Entomol.* **55**, 477-479 (1962).
- Onley, J. H., Giuffrida, L., Watts, R. R., Ives, N. F., Storherr, R. W., *J. Assoc. Off. Anal. Chem.* **58**, 785-792 (1975).
- Polin, D., Ringer, R. K., *Can. J. Physiol. Pharmacol.* **53**, 166-173 (1975).
- Putnam, E. M., Brewer, R. N., Cottier, G. J., *Poult. Sci.* **53**, 1695-1698 (1974).
- Reed, D. L., Booth, N. H., Bush, P. B., Goetsch, D. D., Kiker, J., *Poult. Sci.* **56**, 908-911 (1977).
- Ritchey, S. J., Young, R. W., Essary, E. O., *J. Food Sci.* **32**, 238-240 (1967).
- Ritchey, S. J., Young, R. W., Essary, E. O., *J. Food Sci.* **34**, 569-571 (1969).
- Ritchey, S. J., Young, R. W., Essary, E. O., *J. Agric. Food Chem.* **20**, 291-293 (1972).
- Siegel, H. S., Latimer, J. W., Drury, L. N., Loy, E. W., *Poult. Sci.* **55**, 2319-2326 (1976).
- Singh, S. N., Bahga, H. S., Soni, B. K., *Ind. Vet. J.* **47**, 656-660 (1970).
- Smith, S. I., Weber, C. W., Reid, B. L., *Poult. Sci.* **49**, 233-237 (1970).
- Stemp, A. R., Ph.D. Thesis, Purdue University, Lafayette, Ind., 1965.
- Thompson, E. M., Mountney, G. J., Ware, G. W., *J. Econ. Entomol.* **60**, 235-237 (1967).
- Vogt, H., Torges, H. G., Steinke, L., *Arch. Geflügelkde* **39**, 184-187 (1975).
- Vos, R. H. de, Bouwman, J., Engel, A. B., *Pestic. Sci.* **3**, 421-432 (1972).
- Waldron, A. C., Naber, E. C., *Poult. Sci.* **53**, 1428-1435 (1974).
- Wesley, R. L., Stemp, A. R., Liska, B. J., Stadelman, W. J., *Poult. Sci.* **45**, 321-324 (1966).
- Wesley, R. L., Stemp, A. R., Harrington, R. B., Liska, B. J., Adams, R. L., Stadelman, W. J., *Poult. Sci.* **48**, 1269-1275 (1969).
- Wisman, E. L., Young, R. W., Beane, W. L., *Poult. Sci.* **46**, 1606-1608 (1967).
- Zabik, M. E., Ph.D. Thesis, Michigan State University, East Lansing, Mich., 1970.
- Zabik, M. E., Funk, K., *Poult. Sci.* **50**, 1226-1227 (1971).

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Distribution of Aflatoxin and/or Zearalenone in Wet-Milled Corn Products: A Review

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Aflatoxin and zearalenone are but two of the many mycotoxins that are metabolized in grains by field and storage molds. Corn appears particularly susceptible to these mycotoxin-producing fungi, and there have been several incidences of contamination in recent years. While most of the corn produced is fed to livestock, over a half-billion bushels are used to manufacture food and industrial products. Of this quantity, some 450 million bushels of corn are converted to starch, oil, and by-product feeds by the wet-milling industry. This industry requires high-quality, clean corn and does not purchase lower grade corn, especially that infected by molds. However, when corn is purchased in large quantity from many lots of varying storage history, the possibility of inadvertently getting contaminated corn does exist. To determine the fate of aflatoxin and zearalenone during the wet-milling process, studies have been carried out on artificially and naturally contaminated corn. The extent of contamination and the distribution of the mycotoxins in the milled products are reviewed.

Occurrence of Aflatoxins in Corn. Numerous surveys to determine the incidence and level of aflatoxin in corn have been conducted by the wet-milling industry, the Agricultural Research Service, and the Food and Drug Administration. A summary of these surveys is listed in Table I. Some of the first studies on aflatoxin in corn were done on 1964, 1965, and 1967 corn (all grades) from commercial channels in the Midwest (Shotwell et al., 1969, 1970). These surveys (>1300 samples) indicated both low incidence (2.1-2.3%) and low levels (3-37 ppb) of afla-

toxins. Contaminated samples were mostly from poorest grades of corn. An extensive survey for aflatoxin by the wet-milling industry (Watson and Yahl, 1971) on grain inspection samples from 230 ears of shelled corn showed only four contaminated samples at levels of 3-5 ppb. In addition, corn lots in three plants were sampled daily for 1 year. Aflatoxin B₁ was reported in 6 of the 142 weekly samples at levels of 3-5 ppb. In 1971 and 1972, 525 samples collected from preharvest corn in Indiana showed no aflatoxin (Rambo et al., 1974). Extensive surveys were conducted by the Grain Division, AMS, USDA in 1972, 1973, and 1974 (Hunt et al., 1976). In 1972, 7913 samples were inspected for bright greenish-yellow (BGY) fluorescence, and suspect samples were assayed by CB method and minicolumn method. Only 1.1-1.5% of the

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