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Hydropericardium and Ascites in Chicks Fed a Chlorinated Hydrocarbon*

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An epoxy-resin paint† has been used in our laboratory to coat galvanized batteries employed for trace mineral studies. This paint was supplied as two separate fractions which were mixed just prior to use. One portion contained the epoxy monomer and the other the amine hardener which catalyzed polymerization. On one occasion the paint did not harden before chicks were placed in the battery and within 3 weeks the chicks showed symptoms closely resembling those of the "chick edema" or "toxic fat" syndrome described by Schmittle *et al.* (1958) and Sanger *et al.* (1958). Because of the interest in the chemical nature of the toxic factor (Brew *et al.*, 1959; Friedman *et al.*, 1959; Wooton and Alexander, 1959; Harman *et al.*, 1960) that has been observed in feed-grade fats and the lipid origin of certain components of the paint, it seemed worthwhile to investigate further the causative agent in the epoxy-resin paint.

EXPERIMENTAL

Groups of ten, straight-run, Vantress-White Rock, cross-bred chicks were placed in electrically heated batteries at hatching and allowed to consume the experimental rations *ad libitum* for a four-week period. They were weighed weekly and examined daily for gross symptoms. All chicks were autopsied at death and at least two individuals per group were selected during the third and fourth weeks for histological examination of liver, kidney, heart, lung and spleen. The tissues were processed in the routine manner for histological examination and were stained with hematoxylin and eosin.

The basal ration was a practical-type broiler ration composed of soybean oil meal 36%, yellow corn 59.5%, $CaHPO_4$ 2%, limestone 1.3%, iodized salt 0.5%, methionine 0.1%, a trace mineral mixture and a vitamin supplement. The various paint fractions were dissolved in isopropyl alcohol and added to the mixed ration. Since some of the components were slightly volatile, the feeds were stored in closed containers and kept under refrigeration. Small portions were placed in the feeders daily.

RESULTS

In a preliminary trial the two fractions of the paint, the amine hardener and the epoxy monomer, were tested at a level of 0.1%. One-half of the chicks that received the amine hardener died whereas the other group lived and grew at a normal rate. From this test it was clear that the toxic substance was associated with the amine hardener. According to the manufacturer this fraction contained isopropyl alcohol, polyamid (made from diethylenetriamine and dimerized fatty acids), tri-dimethyl-

* Contribution from the Missouri Agricultural Experiment Station, Journal Series No. 2295.

† The paint (Epo Floor Top) was obtained from the Steelcote Manufacturing Co., St. Louis, Missouri. We gratefully acknowledge the assistance of Mr. A. G. Sternberg of this company who kindly supplied the various fractions tested.

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drome such as distended abdomen and labored respiration were evident, but there was no mortality during the four-week test period. At necropsy hydropericardium was evident. The 0.04% level produced symptoms resembling those observed among chicks fed 0.1% level of the amine hardener fraction. Feeding the polyamid along with chlorinated biphenyl did not change the results appreciably.

Gross Pathology. The gross pathology produced by chlorinated biphenyl closely resembled that observed in the "chick edema" syndrome. Losses usually started during the third week and reached major proportions during the fourth week. Before death the chicks exhibited symptoms of labored respiration with rales and in some cases the abdominal cavity was distended with fluid. At necropsy the most striking pathology was hydropericardium as illustrated in Figure 1. The crop of several birds contained bloody fluid. The kidneys were swollen and pale in most chicks but in the advanced stages many were hemorrhagic. The liver was sometimes enlarged and mottled in appearance. The lungs were commonly hydropic and hemorrhagic. A yellow, jelly-like fluid was frequently found under the skin and within body cavities. A large amount of fibrin was often present in this fluid.



FIG. 1. Hydropericardium observed in a chick fed 0.02% of chlorinated biphenyl for 4 weeks.



FIG. 2. Tubular dilatation in kidney of a chick fed 0.1% of chlorinated biphenyl. H and E stain (100×).

Histopathology. Chlorinated biphenyl at the 0.1% level caused severe renal tubular dilatation with numerous casts especially near the surface of the kidneys. Figure 2 illustrates the typical kidney damage and Figure 3 is a photomicrograph from a control kidney. The majority of the casts were basophilic, but appeared to be made up of strands resembling fibrin. Many of these basophilic bodies contained smaller homogenous eosinophilic bodies suggestive of amyloid. The damage was concentrated largely in the smaller collecting tubules.

The liver had a few areas of lymphocyt-

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Harman *et al.* (1960). It is possible that the toxic component of the chlorinated biphenyl product used here is a contaminant rather than the major component.

Although the toxicity of some chlorinated hydrocarbons has been studied in poultry, the pathology involved has received little attention. Pudelkiewicz *et al.* (1958) observed that a mixture of penta- and hexa-chloronaphthalenes caused high mortality in poults but there was no accumulation of fluid in body cavities.

Hydropericardium and ascites in chicks can be caused by a variety of compounds, the most common cause under practical conditions being excessive amounts of sodium chloride. Gordon *et al.* (1959) observed that a fat-soluble substance in blood meal caused a syndrome resembling the "toxic fat" syndrome. Excess salt increased the incidence of the disease in the presence but not in the absence of the fat-soluble factor. Accumulation of fluid results when the kidney fails to maintain normal water balance either because of excess electrolytes or because of kidney damage. Chlorinated biphenyl causes extensive kidney damage and ascites is probably a secondary result.

SUMMARY

Chicks fed fractions of an epoxy-resin paint developed hydropericardium and ascites. These symptoms were similar to those observed in the "toxic-fat" syndrome. The toxicity was found to be caused by a chlorinated biphenyl product used as a plasticizer in the paint.

Chlorinated biphenyl was moderately toxic when fed at a level of 0.02% and caused high mortality and extensive pathology within 4 weeks at a dietary level of 0.04%. Gross pathology included hydropericardium, hydroperitoneum, enlarged heart, liver and kidneys, and hemorrhage of internal organs. Microscopically the kidneys showed marked tubular dilatation and numerous casts.

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