

March 17, 1937

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CHLORINE

(Continued from page 22)

toxic substance is a metabolite of chlorinated insecticide we have large doses of chlorinated acetone, chloroacetaldehyde, and chloroacetaldehyde. The chloroacetaldehyde is the one responsible for the edema of the carcasses in the chick test. The trend to date shows that chlorinated and thioxychlor produce no response.

Industrial Experiments

Similar experiments have been carried out and are still in progress at least two industrial laboratories under conditions of fatty acid production. The results so far have been largely negative or at best only suggestive but not clear cut. Dr. Arthur has reported that chlorination of naphthalene and phenanthrene substitution reactions has produced five products. The chlorinated phenanthrene is particularly promising, since preliminary fractionating experiments indicated the possibility that a highly toxic compound was produced.

Dr. Boyd O'Dell and colleagues at the University of Missouri observe hydrochloric acid and other symptoms of chick edema disease in chicks housed in freshly painted cages. They traced the responsible agent down to one of the paint ingredients, a chlorinated biphenyl sold under the name Arochlor. We have tested several Arochlores of different chlorine content. Some were not toxic; others produced the disease but only at relatively high feeding levels, e.g., 20 ppm. It may be that an impurity in these compounds is the toxic agent or that they are innately toxic at the high levels fed. Of interest is the fact that one of these Arochlores in some of its formulations. At the present time there has been no real evidence developed to implicate any specific compound. However, the circumstantial evidence is stimulating considerable speculation and activity.

The feed industry has managed a careful control of ingredients to avoid a recurrence of the 1937 epidemic. The color test developed by Brown and al., at Purina has been very useful in screening out toxic lots. This test is not specific but is useful since the presence of large amounts of steroidal compounds that give this test usually indicates still residues that may be toxic. It is useless, however, for fatty acids and other products such as triglycerides, monoacylglycerols, etc. that give no response in this test, but which sometimes are quite toxic.

Georgia Outbreak

Last year, at this time, an outbreak of the chick edema disease occurred in Georgia. Although considerable chlorinated residues were found in the feed, we do not believe they were responsible for the symptoms. Furthermore, the fact that the disease was not observed in the same feed was not observed in the same feed.

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had used in our earlier work, and had found the sample negative for chick edema. Repeat of the test in both laboratories by both procedures confirmed both findings. The sample is free of chick edema disease factor when tested on a diet of natural ingredients, and the chicks survive in apparent good health. On the casein-sucrose diet of the AOAC chick edema test, the chicks fail to grow and die early with the described symptoms but no hydropericardium. Furthermore, the dose response curve for this effect is very steep, since the ratio of the dose that gives a maximum effect to the dose that produces a minimum effect is less than two, as compared to a ratio of four to five for chick edema factor. Evidence from preliminary fractionation studies also indicates that this is an entirely different substance. There is no other information as to its characteristics at this time. There is evidence that this toxic contamination

has occurred in distillates from time to time and in several cases, fatty acid samples and distillates have occurred together with CEF. However, we must anticipate that this material may occur elsewhere independently of the fatty acid industry.

For whatever cause, we may derive, it should be noted that chick edema disease has been observed in England. In a letter to the editor of the *Veterinary Record* of June 10, 1961, C. C. Wannop of the Livingston Poultry Research Station draws attention to a condition apparently identical with the reported by Sanger, et al., and Schmitt, et al., in 1958, that has appeared in several broiler flocks. In a personal note dated Sept. 29, he says that the condition has disappeared for the time in his country.

At the present time fatty acids can be used in the manufacture of foods or food ingredients only if they are free from CEF. This requirement made necessary the development of a bioassay which has been accomplished by collaborative work and is now being adopted as official by the AOAC.

I have tried to review the history of this troublesome problem, what little is known of its toxicology and its physiological aspects, the speculations as to the origin of the contamination, and attempts to track down its sources. I have sketched quickly our own attempts at isolation and identification of the toxic factor and have alluded to the most recent developments along this line that were reported at the AOAC Section of Fats and Oils by Dr. Arman.

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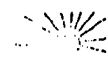
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by collaborative work and is being adopted as official by the A.O.C. I have tried to review the history of this troublesome problem, which is known of its toxicology and its physiological aspects, the relations as to the origin of the contamination, and attempts to trace down its sources. I have sketched quickly our own attempts at isolation, and identification of the toxic factor, and have alluded to the most recent developments. This line of work was reported at the A.O.C. Section of Fat and Oil, by Dr. Arpanian.

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Dr. Boyd O'Dell and colleagues at the University of Missouri have been studying the hydrochloride and other salts of the toxin of chick edema disease in chicks housed in freshly painted cages. They traced the responsible agent down to one of the paint ingredients, a chlorinated biphenyl sold under the name Arochlor. We have tested several Arochlores of different chlorine content. Some were not toxic; others produced the disease but only at relatively high feeding levels, e.g., 200 ppm. It may be that an impurity in these compounds is the toxic agent or that they are innately toxic at high levels fed. Of interest is the use of one of these Arochlores in some insecticide formulations. At the present time there has been no real evidence developed to implicate any particular compound. However, the circumstantial evidence is stimulating considerable speculation and activity.

The feed industry has managed to exercise careful control of ingredients to avoid a recurrence of the 1957 epidemic. The color test developed by Brown, et al., at Purina has been very useful in screening out toxic fats. This test is not specific but is useful since the presence of large amounts of steroidal compounds that give this test usually indicates still residues that may be toxic. It is useless, however, for fatty acids and other products such as lecithin, monoglycerides, etc., which show no response in this test. It is also sometimes the quite toxic.

Georgia Outbreak

Last year, at this time, an outbreak of the chick edema disease occurred in Georgia. Although considerable chlordane residues were found in the feed, we do not believe they were responsible for the symptoms observed. Furthermore, the best information we have indicates that only rendered fat was used in this feed and no product of the fatty acid industry was involved. With each new development the scope of the problem increased. First we were concerned only with still residues, then also with fatty acid distillates and their derivatives. For a long time it was felt that only the fat derived from animals was involved. Recent evidence from sources in the fatty acid industry and our own studies indicates that some very toxic fat sources may yield fatty acids contaminated with GFL.

Still another development has occurred to further complicate the picture. Early this year in the course of our regulatory activities, we examined a sample of oleic acid. All 11 test chicks died by the end of the second week, with symptoms of severely stunted growth, ascites, jaundice and pathology of the liver and other organs, but with no hydroperoxide. This sample had been tested by the manufacturer last year before the adoption of the present AOA test procedure. The testing laboratory had used the procedure we ourselves

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