# A PATHOLOGICAL STUDY ON TETRACHLOROBIPHENYL POISONING IN CHICKENS

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About two million chickens – including broilers and layers – from all over the western part of Japan were infected with an acute, highly fatal, but unknown disease in 1968. In the beginning, some kind of feed poisoning in the field was suspected. Subsequent epizootiological observations revealed that tetrachlorobiphenyl (TCB) was a strongly suspected causal agent of this poisoning.

Clinical and pathological studies were performed on 38 field cases, 90 cases of experimentally reproduced disease, and 326 cases having in experimental TCB poisoning, or 454 cases in total. The following results were obtained.

1) In the field cases, reduced appetite, gasping, and abdominal distension were noted clinical symptoms. Postmortem examinations revealed subcutaneous edema, hydropericardium, ascites and swelling; the liver showed a mottled appearance and the kidneys were faded and swollen. Histopathologically, there were edemas in the skin, skeletal muscle, epicardium, pulmonary interstitium, and the serous membrane of the alimentary tract. Blood vessels close to these lesions often showed edematous alteration of the walls. Centrilobular necrosis of the liver, dilatation of the renal tubules and involution of follicles of the bursa of Fabricius were also recognized in those field cases.

2) All the above-mentioned clinical signs and pathological changes observed in the field cases were also found both in the reproduced and in the TCB-poisoned cases. From this result, the disease occurring in the field was confirmed pathologically to be poisoning caused by TCB.

The pathogenesis of TCB poisoning in chickens is discussed as follows:

1) Liver lesions were most often produced by administration of a moderate concentration (100~300 ppm) of TCB. These lesions were assumed to have been produced by angiopathic injuries persisting for a relatively long time.

2) In the kidneys, the collecting tubules and ureters exhibited severe dilatation when a higher concentration of TCB was administered. It seems that the intake of TCB may have caused the obstruction of the ureter, which possibly brings about early death in chickens.

3) Distinct lesions were produced in the bursa of the Fabricius by the administration of a higher or moderate concentration of TCB. These lesions

were quite similar to those obtained from several infectious diseases and vitamin A deficiency, and followed ACTH administration.

4) It was presumed that ingested TCB might cause an increase in vascular permeability throughout the body. Consequently, a large amount of fluid might leak out of the blood vessels, giving rise to a remarkable anasarca.