THE RELATION OF SELENIUM TO WESTERN DUCK SICKNESS

BY H. W. LAKIN, E. R. QUORTRUP AND NEIL HOTCHKISS

IN 1936 Twomey and Twomey reported that 20–50 parts per million of selenium as sodium selenite in the drinking water produced poisoning in ducks in which the syndrome was identical with that produced by *Clostridium botulinum* type C, which had been considered the principal causative agent in western duck sickness (Kalmbach and Gunderson, 1934). In a more recent paper, Twomey, Twomey and Williams (1939) reported the analyses of a number of duck livers collected at various points near Great Salt Lake where the sickness is prevalent. These analyses show the presence of 7–148 p. p. m. of selenium, based on the oven-dried weight of the livers. In contrast, no selenium was found in duck livers obtained in Pennsylvania, where sickness has not been reported. As a result of these findings, a thorough investigation of the significance of selenium in outbreaks of western duck sickness was undertaken in the summer of 1940.

In July and August, 1940, the authors made a reconnaissance survey of areas in which losses from western duck sickness were known to occur. The areas examined were on the Des Lacs, Long Lake, and Upper Souris National Wildlife Refuges in North Dakota; Bowdoin and Medicine Lake Refuges, Montana; Waubay Refuge, South Dakota; Crescent Lake Refuge, Nebraska; Bear River Refuge, Utah; Malheur Refuge and Copco Marsh on Upper Klamath Lake, Oregon; and Tule Lake Refuge, California.

The livers and gizzards of both sick and healthy waterfowl (28 Pintails, 12 Mallards, 5 Shovellers, 2 Gadwalls, 1 Baldpate, 1 Blue-winged Teal, 1 Green-winged Teal; 1 Redhead, 1 Canada Goose, and 2 Coots; gizzards only of an additional 4 Mallards, 3 Pintails, and 2 Baldpates) were collected, preserved in formalin, and analyzed for selenium. Samples of mud were collected in each area and examined for selenium. A few samples of vegetation and one sample of Cladocera were also examined for selenium.

The methods of analysis used for determining the selenium content of soils and vegetation are those of Robinson, *et al* (1934) for soils and of Williams and Lakin (1935) for plants. They are the tentative methods given in 'Official and Tentative Methods of Analysis of the Association of Official Agricultural Chemists' (1940). In determining the selenium content of livers and gizzards, 10–15 gms. of the material were cut into small cubes about 0.5 cm. on the side and covered with a mixture of nitric and sulfuric acids and treated by

the procedure described by Williams and Lakin. This essentially consists of distilling the selenium as selenium tetrabromide from a bromine-hydrobromic acid mixture, collecting in water, and reducing to selenium with hydroxylamine hydrochloride. The analyses are given on the basis of the weight of the preserved material, after the excess formalin has been drained off on an absorbent paper.

ANALYTICAL DATA AND OBSERVATIONS

The selenium content of the muds, taken from lakes in which botulism occurred, ranged from less than 0.1 p. p. m. to 2.4 p. p. m. It was more than 1 p. p. m. in only one instance, while 65 of the 79 samples of mud examined contained 0.5 p. p. m. or less.

On Lostwood National Wildlife Refuge, Burke County, North Dakota, where no outbreaks of western duck sickness have been reported, a sample of mud was found to contain 0.6 p. p. m. of selenium. On Des Lacs Refuge, Ward County, North Dakota, only a few miles east of Lostwood, the selenium situation appears to be essentially identical, but severe outbreaks of western duck sickness are common.

A composite sample of water collected at Tule Lake Refuge, California, contained seven parts per billion of selenium. Byers, *et al* (1938: 67–68) report the selenium content of a number of samples of water from areas in which western duck sickness occurs (Lacreek National Wildlife Refuge, South Dakota; Bamforth Lake Refuge, Wyoming; and Gimlet Lake on Crescent Lake Refuge, Nebraska). None of these samples contained in excess of one part per billion. The minimum amount of selenium as sodium selenite in the drinking water that Twomey and Twomey (1936) used to produce the syndrome identical with that produced by *Clostridium botulinum* type C was 300- to 2000-fold that found in natural waters where the sickness occurs.

The livers of 54 waterfowl were examined for selenium. Seven of these were normal healthy birds shot on the wing at or near the Bear River Refuge in Utah. Forty-four birds died of botulism (seven not conclusively). Of the remaining three birds, metallic poisoning was indicated in two and aspergillosis in the third.

Selenium was present in detectable quantities in the livers of all birds examined from areas in Montana, North Dakota, and Nebraska. In only five of the 44 birds which died of botulism was there no detectable amount of selenium in 10 gms. of the liver. One contained 3 p. p. m. of selenium, one had 2 p. p. m., two had 1.4 p. p. m., and the remaining 35 livers had 1 p. p. m. or less of selenium. Of the livers of the healthy birds and of those which died from causes other than

botulism, four livers contained no detectable amount of selenium, two contained 0.2 p. m. m., two contained 1 p. p. m., and two 3 p. p. m.

At Bear River Refuge, Utah, the livers of thirteen birds which died of botulism and of seven healthy birds were examined for selenium. While four of the livers of the healthy birds and three of the poisoned birds gave no detectable amounts of selenium, both sets of samples varied over the same range in selenium content, with a maximum of 3 p. p. m. in the livers of both healthy and poisoned birds.

The gizzards of 63 waterfowl were examined for selenium. They varied in selenium content from no detectable amount in 10 gms. of the preserved tissue to 1.4 p. p. m. There is no apparent correlation between the selenium content of the liver of a bird and that of the gizzard. There is also no evidence in these data of particularly high selenium content in the diet of the birds.

The amount of selenium found in the mud samples is so small in every area visited that only the abnormal absorbers of selenium (such as the indicator plants *Astragalus pectinatus, A. bisulcatus, A. racemosus,* etc.) would be toxic. *Astragalus pectinatus* and *A. bisulcatus* were observed growing near the lakes on the Des Lacs and Lostwood Refuges in North Dakota; *A. pectinatus* was found on Medicine Lake and Bowdoin Refuges in Montana; and *A. racemosus* was observed along a lake shore at Waubay Refuge in South Dakota. At no other points visited was there any evidence of the occurrence of the high absorbers of selenium. A sample of sago pondweed (*Potamogeton pectinatus*) from Bear River Refuge and a sample of algae from Tule Lake Refuge were both found to contain less than 0.1 p. p. m. of selenium. Beath, *et al* (1939) report the analyses of eighteen samples of marsh vegetation collected during the seed stage of growth by staff members of the U. S. Biological Survey. Only four of these samples contained selenium. The highest value was 1.5 p. p. m. in *Potamogeton pectinatus.* This is less than one-half the minimum amount of 4 p. p. m. of selenium necessary in the total diet to produce evidence of injury to white rats (Munsell, *et al.,* 1936).

There are no available data on the amounts of selenium that occur in the livers of healthy waterfowl exposed to low concentrations of selenium in their food. Moxon and Poley (1938) fed selenium to poultry in various concentrations from 1.25 to 10 p. p. m. in the total diet for a period of five to six weeks and then killed the hens and examined the carcasses for selenium. The livers of the seventeen birds examined contained from 25 p. p. m. to no detectable amount of

selenium on the dry basis. Assuming the livers to be 75 per cent moisture, they contained 6 p. p. m. or less of selenium on the wet basis. There was no close correlation between the amount of selenium in the diet and the amount in the liver in individual birds, although the average selenium content of the livers of four hens on a diet containing 1.25 p. p. m. of selenium was about half that of four birds on a diet containing 5 p. p. m. These birds were not killed by selenium poisoning and were presumably in good enough condition to be acceptable on the market for food purposes. The high value of 3 p. p. m. in the present investigation is only half the maximum content of chicken livers reported by Moxon and Poley.

Selenium poisoning in poultry is most apparent in chicks hatched from eggs laid by selenized hens (Moxon, 1937). Many of the eggs fail to hatch because of the high incidence of monstrosities (deformities). The most common deformities are: missing or short upper beaks, missing eyes, edema of the head and neck, and wiry down. The chicks which do hatch are usually weak and have a wiry down. Francke, *et al* (1936) demonstrated that these deformities were caused by selenium from the fact that injection of selenium salts into the air cells of hens' eggs before incubation produced monsters similar to those occurring naturally. The authors were unable to obtain any reports of monstrosities in young waterfowl in the areas examined.

In the voluminous publications concerned with the alkali disease in South Dakota there is no reference to limberneck in poultry, nor is there any report of its having developed in experiments conducted with poultry on highly seleniferous diets. Since the toxin of *Clostridium botulinum* type C causes limberneck in poultry (Kalmbach and Gunderson, 1934), one would expect limberneck to be particularly common in the seleniferous area of South Dakota if selenium regularly produced symptoms like those produced by *Clostridium botulinum* type C.

SUMMARY

The distribution of selenium in the soils in the areas visited appears to be general; but amounts found range from less than 0.1 p. p. m. to only 2.4 p. p. m. This is so little that only those plants which absorb large quantities of selenium could be expected to be toxic. There is no close correlation between the amount of selenium in the plants and the selenium content of the soil on which they were grown. Toxic quantities of selenium in plants were observed in the areas affected by western duck sickness only at Bowdoin, Medicine Lake, Des Lacs, and Waubay Refuges. In all of these cases selenium indicator plants (*Astragalus* spp.) were the offenders.

The minimum amount of selenium as sodium selenite in the drinking water that Twomey and Twomey (1936) used to produce the syndrome identical with that produced by *Clostridium botulinum* type C was 300- to 2000-fold that found in natural waters where the sickness occurs. These data indicate no probability that the disease in question is due to selenium in the water.

Selenium was present in detectable quantities in all but nine of the fifty-four livers of waterfowl examined. The selenium content of the livers of birds which died of botulism varied over the same range as healthy birds shot in flight and those dead from other causes. There is no available information on the amount of selenium that one may expect in the livers of healthy waterfowl exposed to low concentrations of selenium in their food other than those data presented here and by Twomey, Twomey and Williams (1939). However, Moxon and Poley (1938) report approximately twice as much selenium in the livers of apparently healthy hens as was found in any of the 54 livers herein reported upon.

The 63 gizzards examined were of low selenium content and gave no evidence of recent ingestion of particularly toxic food due to selenium.

The marsh vegetation which has been examined in affected areas has in all cases been too low in selenium to be toxic.

No deformities of young waterfowl in the affected areas have been reported. Since deformities in the young are one of the primary symptoms of selenium poisoning in poultry, it is of interest that this symptom is not found in western duck sickness. Further, while limberneck is the same disease in chickens as the western duck sickness in waterfowl, no reports of limberneck symptoms are given in the descriptions of selenized chickens.

Selenium, then, does not appear to be a factor in western duck sickness. Selenium has been shown to be widespread in soils, plants, and even in the dust of cities (Lakin and Byers, 1941), as well as a common constituent of human urine (Williams *et al,* 1941). It is not surprising, therefore, that ducks in areas of mildly seleniferous soils should have selenium in their livers; but that the small amounts of selenium in their diet and drinking water should cause the tremendous losses of waterfowl experienced in some of these areas is most unlikely.

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U. S. Navy, formerly U. S. Bureau of Plant Industry
Beltsville, Md.
U. S. Fish and Wildlife Service
Brigham, Utah
U. S. Fish and Wildlife Service
Bowie, Md.