SPECIAL ARTICLE

TOXIC WATERBLOOM IN SASKATCHEWAN, 1959

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IN A LETTER to the editor of *Nature*, in 1878, G. Francis of Adelaide, Australia,[1] described for the first time a phenomenon which is now referred to as "algae poisoning" or "waterbloom" poisoning. Published on May 2, 1878, his letter reported on the low water level and the high temperatures which prevailed in that year on Lake Alexandria, and mentioned in some detail the heavy bloom of algae which preceded and existed concurrently with the observed mass killings of sheep and cattle along the lake shores.

Seventy-two years later, J. L. Ophel, after years of plankton studies in the Murray Valley, referred, in 1950, to reports by local inhabitants of frequent water blooms of Lake Alexandria, commonly accompanied by the death of many sheep and cattle.[2] The last mass killing had been in 1945, when the live stock died after grazing on a dense scum of grassy-smelling blue-green algae at the shore or after drinking the lake water underneath. The algae involved were described as *Microcystis flos-aquae* and *Nodularia spumigera*. When the pastures were fenced off the lake shore, no further deaths occurred.

Similar reports[2] on algae poisoning of poultry and livestock have come from many parts of the world in the years between these two observations and all incriminate certain species of Cyanophytae or blue-green algae. Those most commonly mentioned are species of the genera Polycystis (Microcystis), Anabaena, Aphanizomenon, Nodularia, Gloeotrichia and Coelosphaerium.[2] They all contain pigments in solution, and the name of blue-green algae refers to phycocyanin and to phycoerythrin, two of these pigments. They grow in microscopic and multicellular colonies of variable arrangement, often surrounded by a gelatinous sheath, and are more commonly inhabitants of fresh-water bodies.

The reports on algae poisoning of warm-blooded animals and poultry are more or less well substantiated. Less convincing is the evidence of human involvement in waterbloom disease. It has, however, often been suspected. Morton and David Schwimmer,[3] in their excellent review of the literature on the role of plankton in medicine, list nine reports of possible human algae poisoning in the United States. The symptomatology of the reported and fairly mild sicknesses was either nausea, vomiting, gastroenteritis and arthromyalgia or conjunctivitis, photosensitivity and generalized urticaria. It seems feasible that many such cases may escape medical attention or are not reported for lack of suitable explanation of their etiology.

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The following report on toxic waterbloom in Saskatchewan, occurring in spring and early summer of 1959 in Saskatchewan, adds a number of similar observations. That year brought a hitherto not experienced number of animal killings by toxic waterbloom in the province and also a fairly convincing indication of human waterbloom disease.

On June 29, 1959, a number of dogs and geese died after swimming in Echo Lake, north of Regina, Saskatchewan. Other dogs were reported sick after lapping up water from this lake. Many large fish were found dead along the lake shore.

The residents of nearby Fort Qu'Appelle became alarmed, and the R.C.M.P. detachment at Fort Qu'Appelle requested an investigation of a suspected mass poisoning, by the Provincial Laboratories at Regina. They thoughtfully had preserved several carcasses and also collected water samples from the lake at Valley Centre Beach, where three of the dogs had died.

Investigation, begun on June 30, 1959, revealed that the lake had started to "bloom" in the night of June 28-29. The surface was covered with greenish algae, and in the direction of the on-shore breeze a heavy algal scum had formed at the beaches. While June 28 had seen the first thunderstorm in the area for several weeks, June 29 was a cloudless hot day with barely a ripple of the lake-water. Some stronger winds blew in the afternoon of this day, however, and on June 30 the scum at the beaches had nearly disappeared. Nevertheless the water was still greenish with floating algae.

No animals died on June 30.

Upon autopsy at the Provincial Laboratories late on June 30, two dogs showed a swollen and spotty liver, signs of edema of the lungs, and an impressive hemorrhagic inflammation of the intestinal mucosa beginning in the lower third of the stomach and extending in increasing degree to about the cecal region. Numerous greenish flecks were seen in the mucosa of the duodenum and the jejunum. The airways were free, except for foamy slime in the lower bronchi. One goose also showed lung edema, an enlarged and congested liver, a lesser degree of inflammation of the intestine, and the same dark green flecks on the inner linings of the gut as seen in the dogs.

These findings were consistent with the reports of the dogs' owners that the animals became visibly uncomfortable shortly after leaving the water, lay down, started wretching with convulsive movements and soon showed foam at the mouth and then had diarrhea. Within half an hour to one hour, they were unable to rise and would not respond to fondling and proffered water or food. Death followed after a short period of laboured breathing, interrupted by convulsions. The owner of the geese reported that the birds had swum for 5-10 minutes, then hurriedly came ashore and died within a few minutes under craning of their necks.

The analysis of the stomach contents and liver failed to reveal the presence of strychinine, arsenicals or heavy metals. The water samples contained only 0.1 p.p.m. of copper.

Bacteriological cultures of the stomach contents and tissues of the dogs and goose and of the water yielded no known pathogens. The greenish flecks on the intestinal mucosa consisted of cells and broken-up colonies of algae, identified as Microcystis and Anabaena. The algae of the water-samples of June 29 were predominantly of the species Microcystis and Anabaena. The samples were slimy, blue-green and of a foul smell.

Mice at first were reluctant to drink diluted portions of this scum, but when forced by thirst to do so, they developed no signs of illness. However, four mice died after intraperitoneal injections of 0.1 ml. of the undiluted scum within 60-90 minutes, showing visible discomfort shortly after the injections, excitement, then convulsive twitching of the legs and high jumps. After about 20 minutes they crouched, then fell on their sides, attained a remarkable blue-grey colour of nose and tail, and died with legs stretched out. Autopsy showed in all four mice a dark-red congested liver and slight inflammation of the peritoneal serosa. The anal region was moist with a sticky slime.

From these observations it was concluded that toxic waterbloom of blue-green algae at Echo Lake had killed the dogs and geese. Public warnings were issued on July 1 by press, television and radio that algal waterbloom was a proved hazard to domestic and wild animals, and presumably might also be toxic to human bathers.

Reports also arrived from other areas in the province that cattle, horses and dogs had died after drinking algal scum. Two horses had died at Kindersley Lake, Saskatchewan, on June 29; 3 cows at the Souris-River Dam site near Weyburn on July 2; and 4 dogs at various beaches of Last Mountain Lake, 35 miles north of Regina. One 80-lb. Newfoundland dog died after drinking water from a blooming farm dug-out near Balgonie, Saskatchewan, and autopsy of this animal revealed the same pathological findings, only more pronounced than in the carcasses from Echo Lake. The accompanying water sample showed a heavy growth of *Aphanizomenon flos-aquae* and proved lethal within eight hours to mice upon intraperitoneal injection of 0.1 ml. of these algae, scooped from the layer they formed on the surface of the container. This water sample was remarkably free from foul odour and when filtered did not have a blue-green hue.

Human Involvement

On July 10 a stool specimen was received at the laboratories from a patient at Gull Lake, Saskatchewan, for examination for enteric pathogenic organisms. The clinical diagnosis was enteritis or amebic dysentery. No Salmonella or Entameba were found in the stool, but there were many tiny greenish spheres which resembled in size and morphology the cells of Microcystis. A phone call to the physician revealed that the patient, a tourist from Oregon, had swum on July 8 in one of the Katepwa lakes and had become ill with headache, nausea and gastrointestinal upset in the night afterwards. Intent on returning home, he had proceeded westward, but had been forced at Gull Lake to seek medical advice for his gastroenteritis. He was admitted to hospital but recovered within 24 hours upon administration of oral chloramphenicol (Chloromycetin), and forced his discharge from hospital. Further details have not been obtained.

On July 12, ten children at a camp near Govan, Long Lake, Saskatchewan, reported sick with diarrhea and vomiting to the local physician, after they had bathed on the previous day in algae-covered lake-water. In the stools of one of them, cells that resembled Anabaena cells were found in great numbers.

The examination of the lake-water on July 15 was negative for these species, however, and speculation on the etiology of these cases was frustrated. The area was visited on July 22 for exact reports, but by this time the camp had been dissolved and the physician at Govan was off for the day. Nevertheless, a farmer, four miles southeast of the camp site, described the bloom and reported that two of his cows had died on July 12, 12-16 hours after the lake had bloomed and his herd had watered at the beach. The cows were found lying on their sides, breathing hard and already moribund. They had froth around the nozzles and were quite blue-nosed. A third cow was found to be sick 10 hours later and the farmer decided to try penicillin injections, after which it made a remarkably quick recovery. He believed that the death of the other cows was due to "black-leg" and the injection had saved the third animal. However, this cow, while escaping closer inspection, showed on July 22 a peculiar sloughing of the skin around the nostrils and large scabs on its udder, and the farmer felt this was due to the "infection". No further evidence could be obtained, as the carcasses had been buried after burning. Dried bluish scum was found on the shore, containing cells and scant colony-fragments of Microcystis and Anabaena.

The final observation concerns a physician practising part-time at Fort Qu'Appelle. On August 3, he went for a swim after a busy hot day. Echo Lake was blooming again and he decided on the beach at B-Say-Tah-Point, where the scum was least heavy. After some crawling he climbed on the diving board, slipped and fell five feet into the water. The surprise caused him to swallow an estimated half-pint. It tasted muddy. He left for home and awoke three hours later with crampy stomach pains and a feeling of nausea. He vomited 2-3 times, and five hours after his fall from the board had an attack of painful diarrhea with 6-8 rather painful bowel movements. In the morning his temperature was 102° F., and he had a splitting headache and pains in limb muscles and joints, and felt quite weak. The stools were now a slimy green, similar to the colour of the vomitus. One stool was sent to the laboratories and checked for pathogens, including virus. Microscopic examination revealed innumerable spheres of Microcystis and 2-3 well-preserved curved chains of *Anabaena circinalis* per high-power field. A phone call on August 5 revealed the details of his incident and

also that he felt he was recovering. No other pathogens were found.

Municipal Water Supplies and Algae

On July 14 the main water supply for the cities of Regina and Moose Jaw, Buffalo Pound Lake, 14 miles north of Moose Jaw, had a heavy surface bloom and was found to show mainly species of Anabaena. The filtration plant at Buffalo Pound reported on July 15 difficulties in removing the algae, which clogged the filterbeds. A team of biologists from the University of Saskatchewan, studying the water life of Buffalo Pound and contacted for their observations, agreed to take, and preserve on ice, daily samples of water, and to record blooming and remarkable observations and rumours around the lake. The most important finding was that on July 18 the first colonies of Microcystis were seen and that on July 21 Microcystis was predominating. Up to then no deaths of animals had been reported, but on July 24, Dr. S., veterinarian of Moose Jaw, was consulted about the death of three cows and six dogs shortly after heavy scum had formed on the south shore of the lake. Again, no exact data are available, since Dr. S. reported his experience only on August 3, while visiting the Provincial Laboratories. His diagnosis was algae poisoning, but no verification by autopsy was attempted.

Samples of waterbloom collected on July 23 showed especially heavy amounts of Microcystis and for the first time issue of a blue-violet pigment into the clear water underneath the surface scum in the container. Both the scum of algae and this clear, pigmented fluid underneath were toxic to mice upon injection of 0.5 ml. intraperitoneally (i.p.), and all the mice died within 10 hours under typical signs. The livers were markedly congested. There seemed to be no difference in degree of toxicity of either the algal scum or the clear pigmented filtrate. Aliquots of the daily samples had been collected in 50% denatured ethyl alcohol for better preservation of the toxic principle, but the aqueous solutions were as lethal as the alcoholic extracts, if judged by the time of death of simultaneously injected mice.

Understandably, concern was voiced lest the drinking water for Regina and Moose Jaw be polluted with algae poison. Several series of tests with raw untreated lake-water and with the finished clear-well product were performed. This raw water, upon injection of 0.5 c.c. into mice, did not produce death, although the mice were listless for a day, while the clear-well samples did not show any sickening effect.

A similar study was made of the main water-supply of the city of Weyburn, which collects its water behind a dam of the Souris River and transports it via a 5-mile-long concrete pipeline to the filtration plant. On August 13 heavy bloom occurred at the dam site, the species Anabaena and Aphanizomenon predominating. Mice were killed by 0.5 ml. of the bloom within eight hours and Daphnia crustaceae were immobilized within 30 minutes. Aliquots of scum from raw water samples taken at plant level, i.e. after passage through five miles of pipeline, were toxic to mice after 18 hours only and did not immobilize Daphnia for 52 hours. The clear-well water, still showing some algal, though rather bleached, cells, did not exert toxicity for mice and none for Daphnia.

The concern for the water quality at a time of heavy water bloom of Microcystis rose from reports in the literature on suspected algae poisoning of municipal water supplies in West Virginia and the area of the Anacostia Reservoir near Washington, D.C., in the drought years of 1930 and 1931.[3] In these years low water levels and high temperatures preceded heavy water bloom along the Potomac and Ohio rivers and their watersheds. In Charleston, 1930, 5000-8000 people suffered from mild gastroenteritis at the height of such bloom, and the same thing occurred in several other cities drawing water from the Anacostia Reservoir, innumerable citizens becoming ill in both 1930 and 1931. There was no thorough investigation of these outbreaks, but bacterial causes of the generally mild and temporary gastroenteritis were excluded. It is still an open question whether algal toxins passed in sufficient quantity into the water supply and might have caused the illnesses.

Much research has been done on this algal toxin. In Canada, Bishop, Anet and Gorham[4] at the National Research Council have extracted and studied a toxic substance from uni-algal cultures of *Microcystis aeruginosa Kütz.* It could be identified as a cyclic polypeptide of acidic reaction and has been named "fast death factor", since it killed mice within 2-3 hours, causing pallor and convulsions before death by an intraperitoneal LD_{50} of 0.5 mg./kg. body weight.

They found this factor to be water soluble, remarkably stable to light, stable over a wide range of pH (1-10), and thermostable up to 120° C. It is distinctly neurotoxic for mice and albino rats, and regularly causes parenchymal injury of the liver. The factor is an endotoxin, emitted into the water only by leaky or decomposing cells of Microcystis.

A second substance, extracted by Gorham's[5] team from freshly harvested cells, but not yet too well defined, also proved toxic for mice, killing them in 4-14 hours with symptoms of piloerection and dyspnea. It is called "slow death factor" and seems to be present in fresh waterbloom, though its presence may be concealed by the action of the "fast death factor".

The toxins produced by decaying algae are indeed complex and may be a mixture of exotoxins and endotoxins. There is some evidence to suggest that the rate of algal decomposition influences the concentration of the toxic factors. Much evidence, both from natural and experimental observations, exists for its concentration in the liver of victimized animals.[2, 6] Predators, feeding upon carcasses of algae-poisoned ducks, have been observed to become violently ill and die when devouring the livers.

Interesting is the hypothesis that "Haff" disease, the baffling syndrome of sudden onset of fever, abdominal cramps, myalgia, myoglobinuria and frequent vomiting, which swept through tens of thousands of Europeans at the Baltic coasts in the 1930's and 1950's, was caused by algal toxins.[7] The syndrome was always connected with the con-

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sumption of fish or fish livers, cooked or fried. The lakes from which these fish were taken show blooming frequently, and the algae involved are identical with many blooms in North America. B. Z. Heddeloh injected scum from waterbloom during Haff disease into animals and caused symptoms similar to those evoked by the feeding of these animals with fish from the blooming lakes. While there is not sufficient evidence available to prove that algae were responsible, the analogy to mussel poisoning seems rather striking.

Mussel poisoning[3] is now accepted as the cause of mass deaths after the consumption of shell-fish and other plankton-feeders which were caught after or during the so-called "red tides", the "blooms" of marine plankton, especially of the group of Dinoflagellates. Innumerable reports exist on outbreaks of mussel poisoning in many coastal areas of the world, stemming from as early as 1530.

It seems that only exceptionally heavy waterblooms will emit sufficient toxic principle to prove poisonous or lethal or to cause disease via municipal water supplies. Nevertheless, it could happen. Wheeler demonstrated that the toxic agent may withstand the process of alum coagulation, filtration and ordinary chlorination of water treatment and that activated carbon will not completely absorb the toxins.[2]

Hence, the hazard of water-borne algae poisoning should be considered in years of drought and outbreaks of otherwise unexplained gastroenteritis in our communities. Greatest algal growth takes place during hot weather, with reduced water levels, and in lakes which are shallow and rich in nitrogen, phosphorus substances and loosely bound carbohydrates.[3] Heavy bloom occurs especially when excessive multiplication and deprivation of oxygen in warmed-up waterbodies lead to the starvation of the algae in their beds or when a sudden violent storm rips them up from their accustomed depths and drives them ashore. The scum is composed of decaying algae, and, should the toxin-producing blue-green species prevail, much toxin will enter the water.

Public Health Aspects of Toxic Waterbloom

The multiple killings of dogs and geese at Echo Lake on June 29, 1959, caused considerable alarm. As soon as toxic waterbloom was established as the most likely cause, the Regional Medical Officer of Health, who had shared in the investigation from its beginning, decided on precautionary measures. Within the next two days all public and private beaches in the area were surveyed, and it was observed that similar waterbloom had amassed at the shores of all main lakes in the area.

Since water sport is the principal outdoor summer recreation for the people of southern Saskatchewan and since the summer vacation was close, it was feared that human health might be at risk. Consequently the public was warned via press, radio and TV, and all public beaches were placarded with warning signs. People were advised not to bathe where waterbloom formed scum at the shore lines. On the public beaches the lifeguards were authorized to prevent violations of these regulations.

The entire health region was covered in this way and, since there were recurrences of heavy waterbloom throughout the summer, the measures were maintained through the season. Nevertheless, the above-described cases of human poisoning happened and in addition a number of further reports of gastrointestinal disturbance among many vacationing people reached the office of the M.H.O. None of these was serious enough to require medical attention or to invite laboratory investigation.

The public reaction to these measures was extensive. Many phone calls, privately and from official sources, had to be answered by the M.O.H., and of course there were some vigorous objections to the warning signs on the part of the resort operators and concessionaires in the area whose economic interests were hurt. Concern was voiced also by the Department of Travel and Information of Saskatchewan that the tourist trade might suffer.

Saskatchewan has always had a variable degree of waterbloom on its lakes and smaller water bodies, and in previous years loss of livestock, owing to blue-green algae, was a well-known although sporadic experience of farmers. The growth of algae during the warmer season has always been a nuisance for water treatment plants.

Yet, 1959 was the first year when the extent of this nuisance caused concern to the health authorities, and the number of animal deaths was certainly greater than usual. The Advisory Board of the Public Health Department of Saskatchewan reviewed the matter in the middle of July and agreed to endorse the precautionary measures taken in the Health Region. Waterbloom poisoning was accepted as a possible health hazard and a study of suitable algicides was authorized, in cooperation with the Departments of Agriculture and Natural Resources, and also the University of Saskatchewan. This co-operative study was already well under way last June.

SUMMARY

A rather startling experience is reported: blooming and scum-forming blue-green algae killed in the summer of 1959 numerous dogs, cattle and also poultry which watered at the shore of lakes in Saskatchewan where scum had amassed. Some hazard seems to exist also for humans bathing at such sites. The attention of the medical profession is drawn to blooming blue-green algae.

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REFERENCES

1. FRANCIS, G.: *Nature*, 18: 11, 1878.
2. OLSON, T. A.: Lecture on toxic plankton, Inservice Training Course for Waterworks Personnel, Ann Arbor, February 15-16, 1951.
3. SCHWIMMER, M. AND SCHWIMMER, D.: The role of algae and plankton in medicine, Grune & Stratton, Inc., New York, 1955.
4. BISHOP, C. T., ANET, E. F. AND GORHAM, P. R.: *Canad. J. Biochem. Physiol.*, 37: 453, 1959.
5. HUGHES, O. E., GORHAM, P. R. AND ZEHNDER, A.: *Canad. J. Microbiol.*, 4: 225, 1958.
6. ASHWORTH, C. T. AND MASON, M. F.: *Am. J. Path.*, 22: 369, 1946.
7. ZU JEDDELOH, B.: *Erg. Inn. Med.*, 57: 139, 1939.