The Freshwater Fish Epidemics in Queensland Rivers.

By Professor T. Harvey Johnston, M.A., D.Sc., F.L.S.,

and

M. J. Bancroft, B.Sc., formerly Walter and Eliza Hall Fellow in Economic Biology, University, Brisbane.

(*Read before the Royal Society of Queensland, 28th Nov., 1921.*)

At irregular intervals very widespread and deadly epidemics have appeared amongst freshwater fish in Queensland rivers, more especially those in the western portions of the State. We have endeavoured to ascertain the cause of the mortality but have not as yet succeeded. The outbreaks occur usually in localities that are not readily accessible and moreover generally last for a short time. These facts, together with tardy arrival of information as to the presence of the malady in any particular district, have prevented either of us from being present during an actual outbreak, though on one occasion a visit was paid to a locality just as an epidemic had subsided.

Since one of us has now left the State and the other has undertaken additional duties, it seems unlikely that either will be able, for some time at least, to give further attention to the matter now under consideration. We have therefore thought it advisable to bring together the information which we have collected, so that it may form a basis for some future worker.

We take this opportunity to express our indebtedness to the following for their kind assistance :—Mr. W. Hamilton, Chief Inspector of Fisheries, Brisbane ; Mr. R. Caldwell (Charleville) ; Messrs. M. J. Bergin (Goondiwindi), W. H. Ryan (Charleville), J. McKinley (Goondiwindi), and J. Hogan (Inglewood) of the Police Department ; Messrs H. A. Longman and J. D. Ogilby, Queensland Museum ; Messrs. F. Mills, T. Woulfe, and members of the Longreach Shire Council ; R. Varney (Brisbane) ; A. V. Stretton (Rankine River) ; J. F. Colbert (Lake Nash) ; W. H. Rudd (Austral Downs) ; the last-mentioned three localities being situated in the Northern

Territory. We are also indebted to the Commonwealth Bureau of Meteorology for furnishing full particulars regarding temperatures and rainfall recorded at various Queensland stations.

A preliminary report was published in 1917 by the senior author, but the information and material then available were very scanty. In that report it was regarded as likely that, prolonged dry weather having converted the rivers into a chain of stagnant waterholes, an unhealthy environment for fish had been created, such leading to weakness which gave the fungus *Saprolegnia* an opportunity to exchange its saprophytic life for a parasitic mode of existence, the invasion of the gills leading to death. Decomposing fish would cause a still further reduction of the oxygen supply and thus aggravate the condition. It was believed that the arrival of good rains would remove the stagnation, improve the aeration of the water, and establish a suitable environment for healthy fish life (Johnston, 1917, p. 131).

OCCURRENCE OF EPIDEMICS ALREADY RECORDED.

1892.—An officer of the Fisheries Department, N.S.W., mentioned the occurrence in 1892 of an epidemic causing mortality among fish in a tributary of the Barcoo near Lammermoor Station, in the vicinity of Winton, Queensland (Johnston, 1917, p. 126). A Longreach resident (Mr. Coleman) informed us that a similar outbreak happened in the Thomson River in that year.

No other record of such an occurrence was made until 1917, when widespread mortality appeared among the fishes of the western rivers of Queensland, while milder outbreaks occurred in certain rivers in the south-eastern portion of this State.

1917.—In July, Mr. F. Mills, Clerk of Longreach Shire, reported that fish were dying in the Thomson River and that similar conditions prevailed right out to the MacKinley River, nearly all species being affected.

In August of the same year Mr. A. Sugden sent down a catfish (*Neosilurus hyrtlii* Steind.), taken from the Bulloo River near Quilpie, and reported that fish were dying in large numbers in that river, and that similar conditions had occurred in Cooper's Creek.

In August and September 1917, Dr J. S. Elkington saw

fish (perch and catfish) floating down the Brisbane River. Mr. C. Booker reported that during the same months a widespread mortality had occurred among the fish in Wide Bay Creek and Mary River.

In the September number of the "Scientific Australian" (1917, p. 17) there appeared the following paragraph :—

"Mr. C. A. Baker writes from Kapunda that the proposed trip from Adelaide to the Gulf (of Carpentaria), which was to start on 17th of this month, has now been postponed until the end of March next year for the following reasons :—

"Mr. Kidman has been advised by some of his back-station managers that a most extraordinary and unique fish epidemic has occurred in the following rivers :—Diamantina, Bulloo, Cooper's Creek, and Wilson Creek. The fish have died in such quantities that the water in these rivers has become so polluted that it is not only unfit for human consumption but also for stock. The most extraordinary thing about the death of the fish in these rivers is that the epidemic has occurred in rivers, the headquarters of which are remote from each other and have different sheds and exits."

THOMSON RIVER, COOPER'S CREEK SYSTEM OF DRAINAGE.

Information regarding the earlier outbreak at Longreach (1917) is contained in the previous report (Johnston, 1917).

Early in April 1918 a letter, dated 30th March 1918, was received from Mr. F. Mills stating that fish were again dying in the Thomson River in the neighbourhood of that town. The following notes are taken from his letter :—

The river was in high flood during the months of January and February of this year and large numbers of fish were to be seen after the floods, apparently in a healthy condition but seemed to have a most voracious appetite as plenty were caught with the least of trouble. *The river was still running strong and there was no stagnant water in this locality*[1] as was the case during the previous epidemic. The fish principally affected were what are commonly known as yellow-belly, black bream, bony bream, and perch. They came to the surface of the water in an inert state, suddenly appeared to

[1] Italics ours.

take a fit and swam towards the shallow water or bank of the river, where they died. Their eyes bulged out and they appeared to be sightless. Inside the mouth was of a bluish colour. Their gills and scales appeared to be normal. The fish upon being opened up were found to be very fat. Neither the jewfish nor the freshwater tortoises, which were plentiful in the river, were affected. The epidemic, which commenced shortly after the heavy rains, was still present at the time of writing, *i.e.*, for a period of from three to four weeks. On 22nd March this outbreak was reported in the " Brisbane Courier."

On 23rd April Mr. Mills again wrote reporting that the mortality was not so pronounced and that difficulty had been experienced in obtaining for us a fish in the moribund state. Eventually a specimen, identified by Mr. J. D. Ogilby as *Therapon carbo* Ogilby & McCulloch, was sent down.

In the middle of May a rise in the river was occasioned by rains up country, sweeping away all signs of the outbreak, which had apparently lasted for almost two months, being at its height at the end of March.

On 7th August Mr. Mills informed us that the mortality had reappeared at the end of July, fish dying in large numbers. The senior author was away from town at the time and the junior author was not able to arrive at Longreach until 17th August. On the following day, although prolonged search was made up and down the large lagoon adjacent to the town, only two moribund fish were obtained, one being a *Therapon hillii* Castln., the other a bony bream, *Nematalosa elongata* Macleay. Dead and rotting fish were exceedingly abundant, especially at the lower end of the lagoon, where they were piled up against the crossing. Although close search was made, no more dying fish were obtained. On several days hauls were made with a small-meshed net in the hope of obtaining diseased specimens. An astonishingly large number of fish were caught in each haul but none appeared diseased, so the majority were returned to the water, a few being kept for examination. Three species, *Plectroplites ambiguus* Richardson (golden perch or yellow-belly), *Therapon hillii* (" black bream " or grunter), and *Nematalosa elongata* (slender bony bream) were by far the most abundant. No catfish were caught in these hauls.

During the week previous to the 18th, men had been employed raking the fish out of the river and burning them.

Heaps of fish could be seen along the bank for about two miles. A large proportion of those seen in the unburnt heaps were small jewfish (resembling *Neosilurus*), but specimens of black bream, golden perch, and bony bream could also be recognised. The mortality apparently had affected old and young fish alike, as large and small specimens of perch and bream were found.

Flocks of water-birds had also assisted in clearing the river of dying fish. Cormorants (*Phalacrocorax sulcirostris* and *P. melanoleucus*), snake-birds (*Plotus novæhollandiæ*), white egrets (*Herodias timoriensis*), blue cranes (*Notophoyx novæhollandiæ*), nankeen herons—so-called bitterns (*Nycticorax caledonicus*)—and kites (*Milvus affinis*) were all present in large numbers. Black ibis (*Plegadis falcinellus*) and white ibis (*Ibis molucca*) were also numerous, but pelicans (*Pelecanus conspicillatus*), though present, were not common. In the upper reaches of the lagoon wild pigs had been seen feeding on the dead fish.

Mr. W. Woulfe wrote from Longreach (26th December 1918) stating that the epidemic had reappeared in the Thomson River, affecting chiefly fish of from four to six pounds in weight. He counted 111 dead fish that morning along a length of only fifty yards of the bank. Cormorants were present in countless thousands, while pelicans, herons, blue cranes, &c., were in great numbers.

In the "Courier" of 7th January, 1919, it was stated that during the preceding three weeks great mortality of fish had occurred in the Thomson River, deaths being more numerous than on the previous occasion, large fish especially being the victims.

As an outbreak was reported in the Brisbane daily press (27th August 1919) as having occurred in the Bulloo and Wilson Rivers, we wrote to Mr. Mills who informed us that the epidemic had broken out at Longreach during the winter and had lasted about six weeks, terminating in early August.

McINTYRE AND SEVERN RIVERS.

Through the kindness of Mr. W. Hamilton, Fisheries Department, Brisbane, we had access to reports from the police officers at Goondiwindi (Messrs. M. J. Bergin, J. McKinley) and Inglewood (Mr. J. Hogan) relating to an epidemic during the late winter of 1918 in these two rivers.

Mr. Hogan reported (10th August 1918) that large numbers of fish, principally jewfish and golden perch, had died recently in the McIntyre and Severn (Dumaresq) Rivers, the outbreak being locally regarded as due to one or other of the following :— (1) intense cold and continual heavy frosts destroying fish, especially in shallow water ; (2) the prevalence of a disease ; (3) the low state of the river. Death of the fish was not due to the use of dynamite though this had been put forward as a possible explanation.

Mr. Bergin reported (7th August) that dead fish were coming down the river past Bengalla Station. On 28th August he kindly forwarded to us additional information. The McIntyre River had been rather dry and stagnant, as little rain had fallen from Christmas 1917 until August 1918, when rain caused a fresh in the river and the epidemic ceased. Murray cod, yellow-belly, and jewfish were especially affected, and diseased specimens were all found to be fat. During his ten years' residence in the district he had only once previously noted a similar epidemic, viz., during the great drought of 1915. He also stated that he remembered fish dying in the Condamine River some years ago, but believed that it was due in that particular case to the pollution of the water by an adjacent wool-scour.[2]

Mr. J. McKinley referred (2nd September 1918) to the mortality in the McIntyre in the Goondiwindi district affecting chiefly the Murray cod and golden perch, mainly small specimens. Such fish when opened were found to be very fat, though otherwise they looked normal. Since the recent heavy rains the disease had disappeared.

BURNETT RIVER.

In July 1918 fish died in the lagoon at the junction of the Nogoa and Burnett Rivers. When visited by Dr. T. L. Bancroft some weeks later only a few dead fish were to be seen and these were all in the shallow water.

---

[2] H. B. Ward has drawn attention to the effect of industrial wastes on fish life in his paper on "The Elimination of Stream Pollution in New York State" (Trans. Amer. Fisheries Soc. 48, 1918, pp. 1-25). *See also* Shelford 1917, 1918a, 1918b, 1919a; Shelford and Powers 1919; Hofer 1906, pp. 83-86.

BULLOO RIVER.

On 4th September 1918 a paragraph appeared in the Brisbane "Daily Mail" announcing that a mysterious disease was attacking fish in the Bulloo River from Adavale to the vicinity of Toompine, where they were dying in countless numbers, rendering the water unfit for human consumption. A local theory attributed the disease to the extraordinary season and to overbreeding.

The Commissioner of Police was approached on this matter, and communicated with one of his officials in the Bulloo district to find out whether the epidemic had ceased. Subsequently the text of Sub-inspector W. H. Ryan's reply and, later, a copy of his report (dated 12th Sep ember, 1918) on the matter were forwarded. This officer stated that some weeks previously the fish had been dying in great numbers in the Bulloo, but since then rain had fallen in places and as a result of the fresh in the river the mortality had ceased. All species of fish in the stagnant waterholes were affected but golden perch appeared most susceptible, being the first to die. It was considered locally that the mortality was caused by lack of oxygen in the stagnant water. It was noticed at Quilpie that fish were not killed in Hoodrum Lake, though this contained Bulloo River water and was only a short distance from the smaller stagnant river-waterholes where fish were dying in hundreds.

In the "Daily Mail" of 27th August 1919, mention was made that the epidemic had reappeared in the Bulloo and in the Wilson River, fish of three pounds and upwards dying in extraordinary numbers, smaller specimens apparently escaping the disease.

GEORGINA AND DIAMANTINA RIVERS.

In the Brisbane "Daily Mail" of 14th September 1918, the observations of Mr. E. R. Caldwell on the condition of fish in the Georgina River were given. Evidences of an epidemic were first seen by him in the Georgina near Lake Nash (Northern Territory), but on following the river southward dead fish could be seen piled up along the banks. Mr. Ogilby had suggested this might be due to the salmon disease which affected fish when the water was low. There were many large pools in the Georgina, however, and no sign of contaminating influence, the only places in which fish were not dying being the "kopai"

(mineralised) holes. All kinds of fish were affected and in each case the disease showed the same symptoms—" a blue spot on the side, upon the bursting of which the fish died." Mr. Ogilby subsequently supplied information to the Press that *Saprolegnia* would not live in water which contained any degree of salinity, as was the case with the " kopai " holes.

Mr. Caldwell informed us that the epidemic had appeared in November 1917 and June and July 1918, in the upper reaches of the Georgina, in the vicinity of Lake Nash. Tons of dead fish were to be seen and plenty of sick fish were being caught near the surface and along the edge of the lake by aboriginals. The species represented were yellow-bellies, catfish (jewfish), bony bream, and another kind. The stretch of water in which the mortality occurred was five or six miles long and in places between 20 and 30 feet in depth. Commonly associated with the disease was the presence on attacked fish of a bluish swelling about the size of a sixpence or shilling, at the side in the abdominal region, the aboriginals stating that when these " boils " burst the fish turn over and die. In " kopai " holes the water was clear and brackish owing to abundance of calcium sulphate (" kopai ") and the fish were normal, whereas in the adjacent waterholes containing clayey or muddy water, even though somewhat brackish, the fish were dying. This happened between May and September 1918 while Mr. Caldwell was on the Georgina. There was no drought at the time and cattle were fat.

Mr. Caldwell also stated that in large waterholes in the Austral Downs district (Northern Territory) near Camooweal, though fish were plentiful, no dead ones were seen by him during his visit in May 1918.

In subsequent communications (October and November 1919) he informed us that attacked fish came to the surface and were very sluggish in their movements. An old aboriginal had informed him that fish had died periodically in the Georgina River as long as he could remember. Mr. Caldwell stated that one view as to the cause was that it was due to over-stocking and consequent shortage of food; another, that it was due to cold weather, or to the prevalence of " umbrella grass " which blocked up the gills of the fish. Cormorants were especially abundant, and these, together with the large numbers of pelicans present, were in his opinion sufficient to prevent any overstocking.

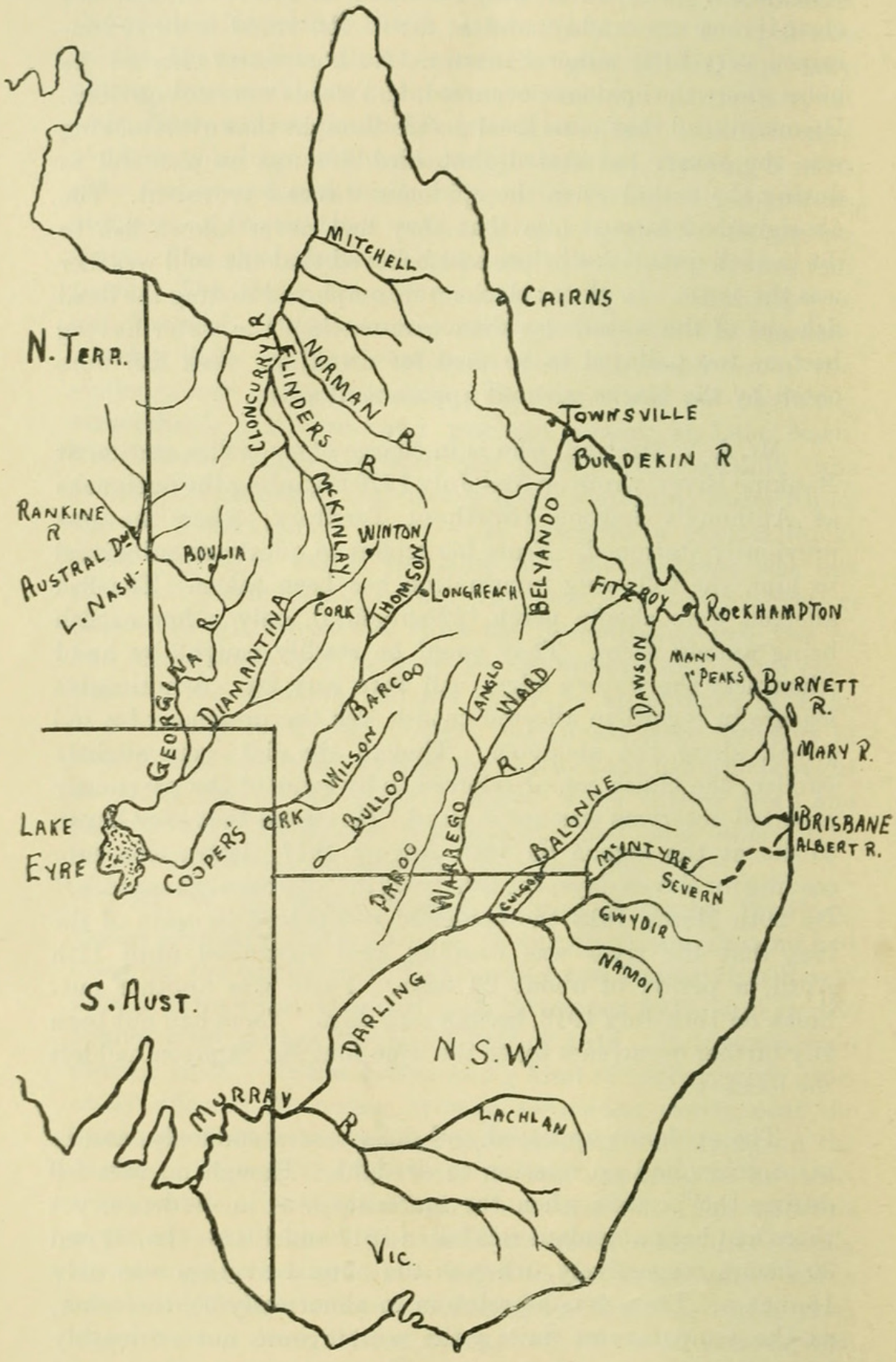
Mr. J. F. Colbert, of Lake Nash Station, Northern Territory, in reply to a series of questions, gave the following information (September-November 1919). He had met with the epidemic on the Diamantina, Bourke, and Georgina Rivers, especially during June and July. In these rivers and particularly at Lake Nash and at Boulia (Bourke River), the dead fish were at times piled up on the banks by the wind, forming a mass some feet across, and this in spite of the presence of enormous numbers of water-birds which were engaged in devouring them. The chief kinds affected were yellow-belly, bream, and perch. The bluish "boil" mentioned by Mr. Caldwell was not observed. Diseased specimens were fat. He was unaware whether there was any relation between the occurrence of the epidemic and drought or cold. The water was not obviously mineralised, and bore-water was not present at Lake Nash. The disease, which appeared and disappeared suddenly, was found both in shallow and in deep holes containing water which was of a dark-green colour—"as green as a typical duck-pond"—whereas during the time that the epidemic was not present it was muddy or milky. In places, *e.g.* Old Cork Station (Diamantina), the stench from the decomposing fish was so bad that people had to leave the homestead and camp elsewhere. The epidemic did not make its appearance at Lake Nash during 1919. Mr. Colbert questioned large numbers of aboriginals, who believed that the death of the fish was brought about by one of two causes—(*a*) the water turned green and killed them; (*b*) the fish fought and killed each other. The latter is obviously an insufficient explanation.

Mr. W. H. Rudd, Austral Downs, Northern Territory, stated (January 1920), in reply to our questions, that he had observed the condition in the Georgina and Diamantina Rivers during September, October, and November 1917, and in the former river during the latter half of 1918. It was not seen during 1919. Yellow-bellies and a kind of catfish were especially affected, becoming drowsy, swimming slowly near the banks in shallow water, and then floating and dying on the surface. Though the fish were fat and appeared to be somewhat swollen, no discolouration was noticed. The epidemic appeared each time rather suddenly about midwinter, finally disappearing when the rivers began to flow as a result of heavy rainfall. Though it occurred during the dry time of the year, there was no drought, but there were very cold periods with ice on the water occasionally. Good rains had fallen each

year prior to the outbreak. Fish died chiefly in the large waterholes. The water was described as good; though not clear it was not muddy, and as far as Mr. Rudd knew it contained very little mineral matter. No bore-water entered he holes where the epidemic occurred, and weeds were not obvious. He mentioned that some local people thought that overstocking was the cause, but stated that bird life was as plentiful as during the period when the epidemic was not prevalent. The aboriginals informed him that they had never known fish to die in such quantities before and believed that the cold weather was the cause. In places blacks were employed to drag the dead fish out of the waterholes with wire-netting, the water having become too polluted to be used for drinking. Sick fish were eaten by the blacks without apparent ill effect.

Mr. A. V. Stretton, who is in charge of the police station at Rankine River, wrote on 31st July 1920 regarding the outbreaks at Anthony's Lagoon (Northern Territory) where he was previously stationed. From his replies to questions submitted to him, the following information has been taken. The fish affected were chiefly perch (*Plectroplites*), only a few catfish being among them. They could be readily caught by hand when near the water's edge. All were very fat. A noticeable feature in regard to affected catfish was the presence of a red streak along the abdomen. Though the fish were slightly swollen, the bluish colour referred to by some of the previously reported observers was not noticed. He stated that they began to die at the lagoon on 10th August 1917, destruction proceeding for eleven days, the fish dying in "countless thousands." On 20th March 1918 the epidemic reappeared in spite of the fact that the river was running, and continued until 12th April, a period of about 23 days. There was another outbreak on 16th July 1918, lasting nine days. There had not been any further occurrence up to the time that Mr. Stretton had left the locality.

The epidemic appeared and disappeared suddenly, and in his opinion had no relation to drought. Though no rain fell during the periods when the mortality was in evidence, yet there had been abundant rainfall in 1917 and 1918—viz., 32 and 30 inches respectively, whereas the annual average was only 18 inches. There was no relation to abnormally cold weather, as the temperatures during the winter were not noticeably lower than during other years. The water was not charged


MITCHELL
CAIRNS
N. TERR.
CLONCURRY R
FLINDERS R
NORMAN R
TOWNSVILLE
BURDEKIN R
RANKINE R
AUSTRAL DMP
L. NASH
BOULIA
McKINLAY
WINTON
BELYANDO
LONGREACH
THOMSON
CORK
FITZROY
ROCKHAMPTON
GEORGINA R
DIAMANTINA R
BARCOO
LANGLO
WARD R
DAWSON
MANY PEAKS
BURNETT R.
MARY R.
WILSON
BULLOO
BALONNE
BRISBANE
ALBERT R.
LAKE EYRE
COOPERS CRK
McINTYRE
SEVERN
PAROO
WARREGO
CULGOA
GWYDIR
NAMOI
S. AUST.
DARLING
N.S.W.
MURRAY R
LACHLAN
VIC.

with mineral. Artesian bore water did not enter the creeks. All permanent waterholes were affected irrespective of depth. The colour of the water during the epidemic was milky or muddy, which was the normal appearance in the district.

He concurred in the opinion generally held in the locality that the cause of the mortality was "overstocking," since fish were present in enormous numbers in the rivers and creeks. Birds such as ibises, spoonbills, pelicans, jabirus, herons, cormorants, were extremely abundant at the time.

Owing to the amount of pollution which had occurred, Mr. Stre ton forwarded a sample of the water to the Health Department, Darwin, with a view to ascertaining whether it was fit for human consumption. The report of the analysis is referred to later.

WARREGO RIVER.

Mr. Caldwell, in a letter to the Brisbane "Daily Mail" of 7th December 1918, stated that, though the epidemic was in evidence in the more northerly situated rivers in this State, it had not appeared in the Warrego and its tributaries, *e.g.* Langlo and Ward Rivers, during 1918, whereas it had caused heavy mortality in these rivers during 1918. He mentioned the current belief that it was due to overstocking in stagnant pools, and stated that though fish might be dying in the main streams, yet in the billabongs or lagoons only a short distance away, and fed by the flood-waters of these streams, fish life was healthy and plentiful.

He informed us by letter dated November 1919 that he had been told that the epidemic had made its appearance in the Warrego River near Cunnamulla and that the decomposing fish were constituting a nuisance to the townspeople (October). Conditions were hot and very dry, stock dying from drought. At Dillalah, also on the Warrego but some distance to the north, a very large waterhole was at the time apparently free from the disease.

OTHER LOCALITIES.

Mr. R. Varney reported (April 1920) that he had observed the epidemic amongst yellow-bellies and black bream, particularly in the muddy water of lagoons in the Longreach and Winton districts, during very dry weather in 1918 and 1919. The condition was noticed in the Cork Lagoon near Winton late in 1919. [Thomson and Diamantina Rivers.]

He also gave an account of some other observations. The heavy thunderstorm which ended the long drought in 1902 caused an enormous quantity of vegetable débris, dead leaves and grass, to come down Enoggera Creek, Brisbane, this forming a blanket about six inches in thickness covering the surface. Fish, mainly mullet, died in hundreds but only a few eels seem to have been affected. Although the water was clear and the bottom was sandy and rocky, the fish, he believed, had been suffocated.

In midwinter 1909 very cold weather was experienced in the Mount Tambourine district, and large numbers of mullet were killed in the Albert River. He believed this to have been due to poisoning by the Moreton Bay chestnut, since large numbers of these trees which were growing along the banks were killed by the cold, their leaves and fruit falling into the water.

He also mentioned that at Cania, 80 miles west of Many Peaks, large numbers of fish—chiefly bony bream and mullet—came to the surface of the water in Three Moon Creek (a tributary of the Upper Burnett) and died, as did also the eels which fed on them. In this case the water was clear and running over a sandy and rocky bottom with plenty of weed present. He believed the occurrence to be due to some form of poisoning.

We are not in a position to comment on any of these three occurrences, which appear to be isolated.

It will thus be seen that during 1917 and 1918 fish epidemics occurred in many rivers widely remote from one another and belonging to different drainage systems. It is reported that an outbreak occurred in July 1917 at McKinley on a tributary of the Cloncurry, itself a tributary of the Flinders, flowing into the Gulf of Carpentaria. This is the northernmost locality known to us. The most seriously affected was that system of rivers which flow inland towards Lake Eyre—the Georgina, Eyre's Creek, the Diamantina, Cooper's Creek or Barcoo with its tributaries, the Thomson and Wilson Creeks. All exhibited the same phenomena at one time or another. Four outbreaks have been recorded from the Thomson. The mortality occurred during the winter of each year in the Bulloo, another inland river. An epidemic was reported in July 1918

in the Severn (Dumaresq) and McIntyre Rivers on the southern border of Queensland. These rivers form part of the Barwon or Darling River system. Three rivers flowing east from the Great Dividing Range were affected though not very seriously—viz., the Brisbane and Mary in the winter of 1917 and the Burnett in 1918.

We may note that the outstanding features of the epidemic were as follows :—

(*a*) The species especially affected were the golden perch or yellow-belly (*Plectroplites ambiguus*), the freshwater black bream or grunters (*Therapon* spp.), Murray cod or perch (*Oligorus macquariæ*), and bony bream (*Nematalosa elongata*), and jew-fish or catfish (various species of *Siluridæ*).

(*b*) It usually occurred during the colder and drier portion of the year, July and August, though sometimes earlier and often persisting later.

(*c*) The water was nearly always stagnant and the epidemic ceased suddenly, after heavy rains had caused the rivers to flow.

(*d*) The affected fish were always fat ; they became lethargic, swam slowly at the surface of the water, and died. A bluish colour was commonly seen in the mouth region.

POSSIBLE CAUSES.

1. The use of dynamite or other explosive.

2. Climatic—
   (*a*) Dry weather ;
   (*b*) Low temperature.

3. State of the water—
   (*a*) Physically, *i.e.* presence of suspended matter or weeds which might clog fish-gills ;
   (*b*) Chemically, *e.g.* excess of carbon dioxide, deficiency of oxygen, acidity or alkalinity, etc.

4. Poisoning due to the presence of some toxic substance in the water.

5. Overstocking and consequent starvation.

6. Disease caused by parasites which may be—
   (*a*) Helminths,
   (*b*) Protozoa—
      (i.) Myxosporidia,
      (ii.) Flagellata, Infusoria,
   (*c*) Fungi,
   (*d*) Bacteria.
7. Two or more of the foregoing acting at the same time.

(1) DYNAMITE THEORY.

It has been suggested that the use of dynamite as an illegal means for obtaining fish might be an explanation of the widespread mortality.

The reports from the Severn and McIntyre Rivers are opposed to such an opinion. We think that the presence of dead fish floating down the Brisbane River in 1918 was, at least in part if not entirely, due to this cause. Specimens from the locality submitted to us by Mr. H. A. Longman, Director of the Queensland Museum, were found to have the swim-bladder burst and the viscera disorganised, an effect such as one might expect from the use of some high explosive.

Mr. Ogilby of the Queensland Museum, in a letter to the Brisbane "Sunday Times" of 18th September 1918, referred to the matter and stated definitely that boating parties in the vicinity of Ipswich were in the habit of using dynamite, and since they probably obtained not more than one in five of the fish killed by the explosion the remainder would float downstream, such fish as perch (*Sciæna australis*), sea mullet (*Mugil* spp.), and catfish (*Tandanus* and *Neosilurus*) being recognised. Mayer[3] has recently referred to the effects of high explosives on fish, especially on those possessing a swim-bladder.

We believe that we may then rule out the Brisbane River reports regarding the epidemic, but there is no justification for attributing the widespread mortality elsewhere to this cause.

(2) EFFECT OF CLIMATE.

This should be treated under two headings—(*a*) the influence of dry weather and (*b*) the effect of temperature—but we have not sufficient data to allow us to consider them separately.

[3] A. G. Mayer, Yearbook, Carnegie Institution 1917, No. 16 pp 185-6; abstract in J.R.M.S., 1919 (3), p. 239.

Queensland is a country in which heavy rain normally falls during the summer, December to April, while the winters are dry. Thus cold and dry conditions commonly go together, although in the early summer the weather may be hot and dry.

The Commonwealth Meteorologist, in a letter dated 31st January 1918, drew our attention to the following statement by Mr. J. B. Henderson, Government Analyst, Brisbane, in a report on a sample of water from Cooper's Creek at Windorah :—

" With reference to your letter . . . and sample of water, no poisons were found in the water. A small fish placed in the water for 48 hours was quite normal at the end of that time.

" The enormous number of dead fish referred to in your letter points either to suffocation by mud or to a more common cause, a sudden drop in temperature. Nothing in your letter indicates or contra-indicates the presence of either of these causes."

The Meteorologist went on to state that an investigation of the temperature records for June 1917 showed rather remarkable departures from the normal and appeared to bear out Mr. Henderson's theory. A copy of minima records for a number of inland stations in Queensland was enclosed.

The localities were Urandangie, Boulia, Winton, Longreach, Isisford, Windorah, Tambo, Adavale, Thargomindah, and Cunnamulla. From the 1st to the 15th of that month there was a warm period in which the averages of daily minimum temperatures for the fifteen days and the number of degrees *above* June normal—given in brackets—for each locality were as follows, respectively :—52·3 (6·8) ; 51·2 (6·5) ; 51·3 (1·6) ; 50·5 (2·8) ; 48·4 (1·4) ; 48·8 (4·0) ; 47·2 (3·8) ; 49·5 (5·7) ; 50·0 (4·9) ; 49·4 (7·2). During the remainder of the month there was a sudden drop experienced at all these stations, commencing on 16th June. The mean of the daily minima during the cold period and the number of degrees *below* the June normal, for each of the ten localities were respectively as follows :—37·8 (7·7) ; 38·6 (7·1) ; 40·5 (9·2) ; 38·2 (9·5) ; 37·9 (9·1) ; 35·8 (9·0) ; 32·6 (10·8) ; 35·3 (8·5) ; 38·5 (6·6) ; 38·0 (4·2). The mean of daily minima for June 1917 at each locality as compared with the normal (calculated from twelve years' records) were given as follows :—45·1 (45·5) ; 44·9 (45·7) ; 45·9 (49·7) ; 44·4 (47·7) ; 43·2 (47·0) ; 42·3 (44·8) ; 39·9 (43·4) ; 42·4 (43·8) ; 42·2 (45·1) ; 43·7 (42·2).

Thanks to the kindness of the Commonwealth Meteorologist we have been able to attempt the correlation of weather records and outbreaks of the epidemic.

Detailed climatological data from several localities for certain specified months were supplied and the following particulars have been abstracted and presented for convenience of reference in tabular form :—

1. The average minimum for the month for all years in which records were taken, *i.e.* the normal minimum.
2. The minimum recorded for the actual month under review.
3. The mean of the minima for that month.
4. The average of total rainfall (in points) for the month for previous years.
5. The actual rainfall recorded for the month.

—	1917.				1918.				
	June.	July.	Aug.	Sept.	Mar.	June.	July.	Aug.	Sept.
				THOMSON RIVER, LONGREACH.					
1	47·1	45·9	46·8	55·0	66·3	46·8	45·2	46·9	..
2	32·2	35·5	32·2	39·4	51·0	35·2	27·0	37·0	36·0
3	44·4	44·8	47·0	53·8	61·6	44·5	40·9	47·5	49·4
4	88	81	32	58	237	85	78	32	..
5	21	4	149	193	97	0	0	51	0
				BULLOO RIVER, ADAVALE.					
1	43·5	42·3	43·3	52·0	..	43·1	41·5	43·8	..
2	30·8	31·5	32·2	36·8	..	31·2	26·1	34·2	26·0
3	42·4	42·2	44·4	51·5	..	41·2	36·7	46·0	49·4
4	121	74	55	64	..	117	73	57	..
5	88	25	91	167	..	15	32	91	0
				BULLOO RIVER, THARGOMINDAH.					
1	45·0	42·1	44·8	50·6	..	45·0	42·0	45·0	50·5
2	33·0	37·5	35·0	39·0	..	34·1	31·0	40·3	39·1
3	44·2	44·6	45·5	51·2	..	44·0	40·4	48·9	49·8
4	84	51	56	50	..	82	51	59	49
5	90	27	6	75	..	0	85	164	0
				MCINTYRE RIVER, GOONDIWINDI.					
1	43·7	41·1	42·1	48·7	..	43·3	40·6	42·4	48·6
2	28·4	29·0	30·8	34·2	..	27·8	23·2	37·2	39·0
3	40·5	39·8	42·4	49·8	..	39·1	35·4	46·3	47·5
4	178	179	133	162	..	174	177	136	159
5	113	77	64	440	..	22	94	252	15
				MARY RIVER, GYMPIE.					
1	46·0	43·9	44·1	52·2	..	45·1	42·9	44·7	51·6
2	29·0	29·0	31·0	33·0	..	30·0	29·5	39·0	35·2
3	42·4	39·9	46·8	50·4	..	40·8	38·1	47·6	48·8
4	252	218	193	218	..	247	214	194	219
5	56	114	175	363	..	2	39	215	295

(i) *Thomson River.*—Longreach records for 1917 shew that June and July of that year were both colder and drier than in the average year (2·7° and 67 points less for June; 1·1° and 77 points less for July). An outbreak occurred in July. The lowest minima recorded during June and July were 32·2° on 22nd June and 35·5 on 5th July. Thus, although both months were colder and drier than usual, the thermometer reached freezing point only once. We are not aware of the condition of the river prior to the outbreak.

The outbreak which occurred in March 1918 can scarcely have had any dependence on the temperature. The records again shew that it was colder (by 4·7°) and drier (140 points less) than the average, but the lowest minimum recorded was only 51°, viz., on 18th March.

The rainy season had begun early in November 1917. The Thomson was in high flood during part of January and February 1918, and was still running when the outbreak began in March (*vide* Mr. Mill's letter of 30th March), there being no stagnant water at the time.

June 1918 was again colder and drier (2·3° and 85 points less) than the average, while the lowest minimum (35·2°) occurred on the 28th. In July, however, a more decided cold snap was experienced; for the nine days following 8th July the minimum records were consistently low, culminating in three nights of frost—28·5° on the 15th, 27° (lowest recorded) on the 16th, and 31·7° on the 17th. After this no further frosts were experienced.

No rain fell in the district during May, June, or July, so that the river was fairly low when the outbreak began at the end of July. The epidemic was at its height during the first week in August and had abated completely by the middle of the third week. Showers of rain yielding 45 points fell on 3rd and 4th of August. During September 1918 no rain fell. The minimum temperature fell to 36° on the 5th, this being the lowest for the month, though the three succeeding days experienced low minima.

An outbreak occurred during the latter half of December 1918, extending into early January 1919 (midsummer), when the temperatures were certainly not low. It was a very dry period and the water was stagnant. Another made its appearance in July and early August 1919, but particulars as to the weather are not in our possession.

(ii) *Bulloo River.*—Weather records for Adavale shew that June and July 1917 were drier and colder than the normal, being, however, only very slightly colder for July. The outbreak was reported in August but probably began in July. June 1918 was again colder and drier than normal. The epidemic began about the end of July and ceased after a fresh had occurred in the river in August. It reappeared in August 1919.

(iii) *McIntyre River.*—With regard to the epidemic occurring in the McIntyre and Severn Rivers during July and August 1918, the records shew that during June and July both minimum and rainfall records were considerably below the normal. The river was very low until rain fell in August.

(iv) *Mary River.*—Gympie weather records shew that June 1917 was colder and drier, and July 1917 much colder and drier, than the normal. An epidemic was reported to have occurred in the Mary River in August and September of that year.

June and July 1918 were both still colder and drier than in the preceding year, but no epidemic followed.

(v) *Burnett River.*—For the last ten days of June 1918 and the first eighteen days of July frosts were experienced practically every night (twenty nights) at Eidsvold. We are indebted to Dr. T. L. Bancroft for allowing us to use his records.

(vi) *Georgina and Diamantina Rivers.*—The outbreaks took place in September, October, and November 1917 and from June to September 1918, but none occurred during 1919. Though the epidemic made its appearance during the dry weather, conditions were not those of drought. Many local people thought that cold was the cause of the trouble.

From the foregoing it will appear that cold is not a necessary factor though it was a very common concomitant. Neither is drought a necessary condition, though dry weather appeared to be common to nearly all the outbreaks.

One can, however, state that dry cold conditions, and especially abnormally dry weather, favour the epidemic, and that the advent of sufficient rain to set the rivers in motion terminates it.

Heath (1883) found that certain species of fish were able to survive after having been frozen in blocks of ice for a few

hours, provided they were slowly thawed out. Bumpus (1898) suggested that the heavy mortality of tile-fish off Florida in 1878 might be due to a sudden diminution of temperature as a result of an alteration of the Gulf Stream. Hofer, in his valuable work on fish diseases (1906, pp. 87-93), gave an account of the effects of cold on the skin of freshwater fish in Europe.

Wells, in one of his many papers dealing with the relation of fish to their environment, stated that many species can detect and react to temperature differences as small as 1 to 2 degrees Centigrade (1913, p. 339). Next year he published a paper giving an account of his investigations regarding the resistance and reactions to temperature (1914). He found that, in the case of freshwater fish, the degree of resistance varied with the species and with the size of the individual, large specimens being more resistant to high temperatures than small fish of the same species, while small individuals were able to adapt themselves more successfully to sudden changes from warm to cold. He also reported that in no case did death result from sudden change from a higher to a lower temperature, though the widest range—viz., from the maximum for the species down to freezing point—was tried. He admitted that it was possible that a sudden and great lowering of temperature might cause death in the case of certain species. His experiments showed that fish can detect and react to variations of temperature amounting to only 0·1 degree Centigrade.

Shelford and Powers (1915, p. 325) ascertained that marine fish were capable of detecting differences of ·5° to ·6° C., and probably as low as ·2° C.

3. STATE OF THE WATER.

This may be considered under various headings—viz., alterations in regard to amount of suspended matter or weeds, the amount of gases (oxygen and carbon dioxide) present, in the degree of alkalinity or acidity, and in the amount of salts in solution.

(*a*) Suspended Matter.

We know that the amount of suspended matter in stagnant pools depends mainly on the chemical composition of the water, since the presence of certain substances leads to the precipitation of finely divided and colloidal material. One might draw attention to the muddy water of a stream and the clear water of adjacent "kopai" holes rich in sulphate of lime.

Under this heading one may refer to the presence of organisms, whether plant or animal, which could act mechanically by interfering with the passage of water through the fish-gills.

Reference was made to the suggestion that overgrowth of water-weeds, including "umbrella grass," might cause trouble. It is to be pointed out that weeds grow only under certain conditions of light, depth, etc., and in the case of rooted plants form only a fringe around deep lagoons. Some of the waterholes in which the epidemic occurred were many miles long and up to 30 or more feet in maximum depth, the greater part of the lagoons being too deep for rooted plant growth. Abundance of green water-vegetation improves aeration, though one must admit that organic decomposition results in the using up of oxygen and the liberation of carbon dioxide and other gases. Moreover, the presence of abundant decomposing matter is associated with abundance of saprophytic and saprozoic organisms—*e.g.* fungi, bacteria, and certain protozoa—all of which are using up oxygen instead of liberating it.

Mr. Colbert referred to the deep-green colour of the water in his locality, Lake Nash, during the periods in which the fish epidemic occurred. The colour suggests that phytoflagellates were present in enormous numbers. It is not impossible that they might set up some irritation of the gills and become entangled in the mucus produced, and thus lead to partial or complete suffocation. But the presence of this intense colouration does not seem to have been a constant feature of the outbreaks, and suggests to us that the stagnant conditions allowed the organisms to grow at a much more rapid rate than they were being devoured by the various other organisms present. As a rapid decomposition of these might cause the liberation of toxic substances, the matter will be referred to later, under the heading of poisons as possible causes.

(*b*) Effect of Alterations in Chemical Composition of Water.

An analysis of samples of Longreach water, one from the Thomson River and the other from an adjacent billabong, was made by the Government Analyst, Mr. J. B. Henderson,

his report to the Longreach Shire Council, dated 29th August 1918, being as follows :—

——	(1) From River.	(2) From Billabong.
Odor	Earthy and fœtid	Same but much more pronounced
Total solids	41·0	36·8 parts per 100,000
Chlorine	2·0	2·0
Alkalinity	6·0	6·0
Sulphates	2·1	2·1
Nitrates as nitrogen	·048	trace
Free ammonia	·01	·044
Albuminoid ammonia	·06	·094
Oxygen consumed in 15 minutes at 90° F.	·281	·462
Oxygen consumed in 4 hours at 90° F.	·536	·782
Hardness	5·5	5·5

The samples are highly contaminated with organic matter and are unfit for drinking.

We do not know what was the composition of the water from either situation during periods when the epidemic was not present.

When discussing the above analysis of the Longreach water with Mr. Henderson, he informed us that the sample was milky and was taken at a time when the Thomson River was covered with dead or dying fish, the water being then green and stagnant. He said that the results relating to the presence of ammonia were of no value as indicating the constituents of the original sample. The amount of total solids was distinctly low and the organic matter unduly high.

On the occasion of our visit the water in the billabong was found to be very darkly coloured, with a greenish tint, apparently on account of an exaggerated growth of algæ. The escape of fat or oil from the mass of decomposing fish present caused the water to be fœtid and to possess a greasy feel. Ciliates and flagellates were in abundance, as were tiny crustaceans.

A report on a sample of water from Anthony's Lagoon, Northern Territory, sent by Mr. Stretton to the Health Department, Darwin, during the 1918 epidemic, was issued by Mr. M. A. Kelly for the Chief Health Officer, and contained the following information :—Colour, dirty greenish white ; odour,

sulphuretted hydrogen; reaction, none, neither acid nor alkaline; residue on evaporation, ash and a slight charring; free ammonia in considerable amount; chlorides, equivalent to 16 grs. per gallon; sodium chloride 26·3 grs. per gallon; hardness, 8 degrees of temporary hardness; nitrates, none; metallic impurities—iron, strong trace; zinc, lead, copper, and arsenic absent; oxygen absorbed in 15 minutes at 212° F., 1·30 grs. per gallon; microscopic examination, grass and weeds in all stages of putrefaction; bacteriological examination, innumerable colonies.

These last two findings might have been expected owing to the time which would necessarily elapse between the collecting of the sample and its examination. The analysis suggests that the sample originally contained a considerable amount of organic matter.

Marsh (1908, p. 905-6) has pointed out that there is as yet no sure method of determining by chemical tests whether water is suitable for fish-life. The ordinary "sanitary analysis" determines whether water is fit for drinking and for domestic use, but water which may be passed as suitable for such purpose may kill fish in a short time, and we know that fish can thrive in waters which on routine examination would be pronounced unfit for human use. Both Marsh (1908) and Shelford (1918c, p. 39 footnote) point out that, in this connection, it is important that such additional items as acidity or alkalinity, the amount of hydrogen sulphide, carbonaceous material capable of being utilised as food, unusual metals, dissolved air, etc., should be known.

The most important items in the above analyses seem to be those relating to the amounts of oxygen and carbon dioxide present. The blue colour inside the mouths of affected fishes suggests a deficient oxygenation of the blood, and this may be due either to a diminished amount of oxygen or to a greatly increased amount of carbon dioxide, or to both.

Wells (1913) has studied the resistance of fishes to different concentrations and combinations of oxygen and carbon dioxide. In regard to the latter he pointed (p. 329) out that the presence of a high and low concentration of CO_2 is affected by many factors, such as the amount of vegetation in the water, character of the surrounding soil and incoming water, depth of water, season of the year, daily temperature, animals present,

amount of decaying organic matter, rainfall, and exposure of the surface to winds. Hence great variation may occur within the same body of water at different times. He stated (p. 344) that small variations (*e.g.* 5 to 10 cc. CO_2 per litre) from the normal in regard to the amount of CO_2 present apparently produce ultimately effects similar to those caused by greater variations (25 cc. CO_2 per litre) in relatively short periods. Certain species are more sensitive than others in this respect and would therefore react first—*i.e.* they would endeavour to move away from the adverse conditions. Resistance comes into play when organisms cannot move away from unsuitable surroundings but must adapt themselves to the unfavourable environment. We know that fish are able to withstand stagnant water during dry seasons by gulping air at the surface. We also know that some fish are less affected than others. Wells found—(1) that the presence of oxygen in large amounts (10 cc. per litre) counteracted the detrimental effect of high CO_2 content (50 cc. per litre) ; (2) that low oxygen content (0·1 cc. per litre) in alkaline water caused death sooner than when it occurred in slightly acid water ; (3) that the resistance of fishes to fatal concentrations and combinations of oxygen and carbon dioxide varied with the individual, with the species, and with the weight, small fish being more resistant per unit weight than were large ones.

Wells (1916) investigated the seasonal resistance of fishes in the United States, and stated that as a result of several years' observations i had been noted that in nature their resistance to detrimental factors in general was lowest in late summer (July to October) and highest in spring (February to May or June). They were found to be least resistant just after the breeding season.

Shelford and Allee had previously (1913) pointed out that young fish were more sensitive to changes in regard to the amounts of these two gases than older fish were, and that some species reacted to a concentration of CO_2 as low as 5 to 7 cc. per litre, and of oxygen as high as ·7 to 1 cc. per litre.

Powers (1914) found that freshwater crayfishes reacted to very weak concentrations of CO_2.

Wells (1918) stated that at a concentration of 10 cc. per litre CO_2 soon proved fatal to more sensitive species, and that it was doubtful if there were any freshwater fish which could

continue to live in water where the CO_2 content averaged as high as 6 cc. per litre throughout the year, but that it was still to be demonstrated whether there were any species of truly freshwater fish which could reproduce successfully in water that was decidedly alkaline to phenolphthalein throughout the year. Shelford (1918c, pp. 45-6) pointed out that since CO_2 results from the decomposition of organic matter, in the process of which oxygen is consumed, so the presence of any large quantity of CO_2 nearly always indicates a lack of oxygen. He thinks it probable that the CO_2 content should not average more than 3 cc. per litre over breeding grounds, and more than 6 cc. per litre during the summer, as such quantities are not usually accompanied by lack of oxygen. He suggested that the amount of CO_2 might be taken as an index of the suitability of the water for fish-life.[4] Wells (1915) found that the CO_2 optimum for the various species of freshwater fish experimented upon under summer conditions varied from the acid side of neutrality to 6 cc. per litre. Marine fish behaved differently as they preferred slight alkalinity to acidity (Shelford and Powers, 1915; Shelford 1918c, p. 40; 1919). The time taken to kill freshwater fish, using higher concentrations of acid, was found by Wells to be proportional to the hydrogen ion concentration.[5] This author gave considerable attention to the reactions of fish to the ions of H and OH.[6]

---

[4] *See also* Birge and Juday (1914, pp. 583-7) regarding the distribution of CO_2 in lakes. *Also* Shelford, 1918c, pp. 40-1; 1914.

[5] "The theory of solution explains acidity in water by the occurrence of hydrogen ions, formed from dissolved electrolytes, in excess of hydroxyl ions; and alkalinity by a similar excess of hydroxyl over hydrogen ions. Neutrality is, then, the condition when, as in pure water, the two concentrations are equal." (L. J. Henderson, The fitness of the environment. MacMillan, New York, 1913, p. 142.)

[6] By titration, using phenolphthalein and methyl orange as indicators, Wells (1915) determined the amount of CO_2 present in a fixed condition (as carbonates), "half bound" (*i.e.* bicarbonates), and free (as CO_2), since methyl orange remains unaffected by carbonic acid so that the bases present as carbonates or bicarbonates can be titrated with an acid; while carbonates are alkaline to phenolphthalein, bicarbonates neutral, and free CO_2 acid. Methyl orange is very sensitive to OH ion whereas the latter indicator reacts to the H ion instead and consequently gives an acid reaction with CO_2. In the presence of CO_2, methyl orange will give an alkaline reaction though the water may still be acid owing to the presence of a higher concentration of H than OH ion.—*See also* Shelford, 1919b.

Distilled water has been shown to be toxic to various organisms owing to its influence on the permeability of the gill membranes, leading to the loss of salts by the animal and the absorption of water by osmosis (Abbot, 1913). Such water was found by Wells (1915, pp. 241, 254) to be not toxic if rendered slightly acid, but remained so if made slightly alkaline.

The following extract from Wells's paper (1915, pp. 243-4) is of interest :—" The fact that in natural bodies of water the chemical reactions of the water may vary from alkalinity through neutrality to acidity, or the reverse, makes he practical importance of a knowledge of the reactions and resistance of fishes and other organisms to such chemical conditions an obvious one. From the experiments (referred to in his paper) it is clear that water which gives an alkaline reaction to phenolphthalein for any length of time during the year is undesirable as a home for most freshwater fishes. On the other hand, marine fishes with the exception of anadromous species would probably not survive in water which was even faintly acid. Since algæ or other phytoplancton forms may cause a body of water to become wholly or partly alkaline, through their ability to dissociate the bicarbonates, vegetation in fish waters assumes a line of importance heretofore little considered. The effects of sewage upon the acidity or alkalinity of natural bodies of water must be reconsidered in the light of its possible injurious or beneficial effects due to its chemical action. . . . Henderson's work (1913)[7] on the mechanism which maintains a constant proportion of H and OH ions in the blood of animals, suggests the physiological reason for this extreme sensitiveness of the fishes. Very small variations in the proportions of these two ions in the blood of the organism are of grave importance and we find in the blood a combination of gases and salts that makes such variations impossible as long as the animal is normal. The blood will maintain its normal chemical reaction (just on the alkaline side of neutrality) in the face of relatively large changes in the environment, yet we know that the mechanism breaks down when the change is either too gr at or too long continued. . . . The hypersensitiveness of the animals to the chemical reaction of the water in the case of aquatic organisms is another important factor in preserving the normal reaction of the blood, as the reactions

[7] L. J. Henderson, The fitness of environment. McMillan, New York, 1913.

of the organisms work in a way that causes them to turn back from concentrations of H and OH ions that would be detrimental. . . . The physiological effect of the acid, neutral, and alkaline water upon the organism very probably has to do with decrease or increase in the pe meability of the exposed tissue cells (especially the gills in the case of fishes)." Alkalinity increased and acidity at first caused a decrease in permeability, but acidity if increased caused an increase in permeability, so that, as in the case of alkalinity, death was ultimately the result. In regard to marine fishes the results of Shelford and Powers indicated that the action of alkaline water produced a normal permeability of the membranes, and it is likely that an acid condition of the water would kill such fish by diminishing the permeability (Wells, 1915, p. 245).

As already stated, the appearance of the affected Queensland fish suggested suffocation, while the bulging of the eyes noted by some observers strengthens the suggestion that the water contained excess of carbon dioxide.[8]

Carbon monoxide is very poisonous to freshwater fish (Shelford, 1917). Wells (1918, p. 562) ascertained that a concentration of from 75 to 100 cc. per litre CO_2 would be required to produce as deadly results as 1 cc. per litre CO, and that a saturated solution of CO in water did not lose its toxic effects even after two weeks' exposure to the air (p. 563).

Another gas which is formed as a result of organic decomposition, and may be added to water supplies as a result of pollution by industrial waste, is sulphuretted hydrogen. Shelford and Powers (1915 ; Shelford, 1918b) drew attention to the extreme sensitiveness of fish to this gas, as they endeavoured to avoid the presence of even a fraction of a cubic centimetre per litre. Fish died in a few minutes in water containing 7·6 cc. H_2S per litre, and a combination of this gas with CO_2 was reported to be "exceedingly deadly." "Since decomposition yields CO_2 and consumes oxygen and is accompanied by the production of hydrogen sulphide which is also accompanied by the consumption of oxygen, it is reasonable to suppose that on a bottom from which vegetation is absent and decomposition actively takes place, a fatal combination of lack of oxygen and presence of hydrogen sulphide and probably

[8] For references to "pop-eye" of fishes *see* C. C. Farr, Rep. Austr. Assoc. Adv. Sci. 13, 1911 (1912), p. 354 ; Ogilby and McCulloch, Mem. Q'land Museum, 5, 1916, p. 112.

carbon dioxide can quickly develop" (Shelford and Powers, 1915, p. 322). No doubt, in the case of the Queensland epidemics H_2S has played an important rôle in aggravating the condition, though it probably did not cause it in the first place.

Ammonia is very toxic to fish and is apparently not recognised by them, as they do not react in such a way as to avoid it when given the opportunity (Wells, 1915a).

The reactions and resistance of fishes in their natural environment to salts were studied by Wells (1915b), who used the chlorides, nitrates, and sulphates of the commoner bases—*e.g.* sodium, ammonium, potassium, calcium, and magnesium.[9] He found—(1) that freshwater fish reacted to their presence in solution but were not as sensitive to salt ions as to H and OH ions; (2) that they reacted to combinations of antagonistic salts or salt and acids in a manner which tended to bring them into a region of optimum stimulation; and (3) that rhythmic alterations in metabolic activity in the case of anadromous fish (such as salmon) were correlated with their migrations.

Powers (1917) has studied the relative toxicity of the chlorides and nitrates of the alkalies and alkaline earths, as well as various other substances, on goldfish. He pointed out that it is improbable that toxicity of a substance is due to osmotic pressure.

Reduction in salinity as a result of flood-waters being turned aside to cover certain Japanese reefs[10] led to very great destruction of marine algæ and the associated fauna.

4. Poisons as a Possible Cause of the Epidemic.

Apart from the toxic effects likely to be caused by the agencies referred to in the last section, one can probably dismiss the possibility of the mortality being caused by a poison—*e.g.* one of plant or mineral origin—on account of the wide area involved, the different flora in each region, the different types of water, and the character of the outbreaks.

---

[9] Whetmore (1918) in his investigations regarding the epidemic amongst wild ducks in Utah, U.S.A., proved that it was due to alkali poisoning, especially by the chlorides of calcium and magnesium which are brought to the surface of the soil of the swamps by capillary attraction.

[10] Yendo, Econ. Proc. Roy. Dublin Soc., 2, 1914, pp. 105-122 (not available). *See also* papers by Sumner (Bull. Bur. Fisheries 25, 1905 (1906) pp. 53-108) and Scott (*Id.* 28, 1908 (1910), pp. 1145-1150) regarding the effects on the blood of fishes of changes in salinity and density of water.

In a report Mr. J. B. Henderson stated that toxic effects were not produced by a sample of the water in which the epidemic had occurred, when a fish was kept in it for 48 hours. It should be pointed out, however, that all species are not equally susceptible, and that it was possible that gaseous poisons, if such were the cause, may have escaped or have become altered.

The possibility of some toxic substance being liberated as a result of decomposition of myriads of dinoflagellates has been mentioned. It is known that certain phytoflagellates, especially *Peridinium*, *Gonyaulax*, and allied forms, have caused very serious epidemics amongst various organisms, the result being brought about by the death of immense numbers of these tiny organisms, the decomposition products destroying fish, molluscs, etc., In fact, such water, which is generally coloured reddish by these flagellates, is often spoken of as "poison water." The animals so killed, on decomposing, aggravate the condition so that widespread mortality has been caused.

In the "Sydney Morning Herald" of 27th December 1918, attention was drawn to the "red weed pest" destroying fish and oysters owing to its extraordinary abundance in Port Macquarie, N.S.W. Mr. A. H. Lucas[11] described this dull-red seaweed as *Falkenbergia olens*. He stated that it probably lived on plants in deeper water, being brought inshore in great masses at irregular intervals, sometimes collecting on oyster-beds with disastrous results, owing to its rapid decomposition and putrefaction, a great deal of gas being evolved.

Hedley (1915, p. 29) referred to two sudden and widespread epidemics which occurred amongst sedentary intertidal organisms in Port Jackson in 1866 and 1891. In regard to the latter Whitelegge (1891) reported that immense numbers of the dinoflagellate *Glenodinium rubrum* caused the clogging of the gills of various molluscs and led to their death and ultimately, through their decomposition, to the destruction of great numbers of other organisms.

Other accounts of heavy mortality amongst marine fish, caused by flagellates, have been published by Torrey, 1902 [*Gonyaulax*—Californian Coast in 1901]; Gilchrist, 1914 [*Noctiluca*, *Peridinium*—South Africa]; Hornell, 1917 [Eugle-

[11] A. H. Lucas, Notes on Australian Marine Algæ, P.L.S. N.S.W., 44, 1919 (pp. 175-6).

noids—South India]; Nishikawa, 1901 [*Gonyaulax*—Japan]. Taylor (1917) rejects these as being a likely cause of certain epidemics in Florida waters, one being previously reported by Ingersoll (1882).

5. OVERSTOCKING.

Overstocking is commonly regarded as being the cause. Owing to dry weather, the rivers and waterholes shrink considerably, and as a consequence there is much less water and food for the fish which come to occupy the restricted areas. It seems likely that overstocking may be a contributing factor to the epidemic. The increased number of fish would use up more food and oxygen and liberate more CO_2, which would lead to ill effects unless there were increased plant activity to preserve the balance and so prevent the water from becoming more and more acid. Besides, should the real cause of the epidemic be some protozoan, fungoid, or bacterial organism, then the greater density of the fish population would be favourable to the rapid spread of the disease.

6. PARASITISM AS A POSSIBLE CAUSE.

It is well known that organisms may cause serious epidemics. Animal parasites likely to be incriminated may be (*a*) Helminths, and (*b*) Protozoa such as (i) Sporozoa (Myxosporidia and Microsporidia), or (ii) Flagellates, perhaps Ciliates. Amongst plant parasites one must consider (*c*) Fungi and (*d*) Bacteria.

(A) Helminth Infection.

The cyanosed appearance of infected fish suggested the possibility of gill parasites, especially Heterocotylean Trematodes, being a cause, through their interference with normal gill function. It is known that infection is sometimes extremely heavy, and Ward (1918, p. 374) has stated that *Gyrodactylus* may be in sufficient numbers to cause death. McCallum (1915, p. 410) has reported that infestation by *Diplectanum* may be so intense as to bring about the death of its host. Pratt (1919, p. 2) has also referred to gill-infesting trematodes as a cause of considerable mortality, especially in enclosed bodies of water. Magath (1917, p. 59) in a paper dealing with a fluke, *Lissorchis*, which was regarded as causing heavy losses amongst certain fish in Iowa, U.S.A., mentioned that some parasites so weaken their hosts that the latter may die from some cause which would otherwise not have so affected them.

A careful examination of a considerable number of affected and healthy fish, chiefly from the Thomson River, revealed the presence of many species of Gyrodactyloid trematodes infesting the gills in both cases, so that invasion by the minute parasites can at most be only a contributing factor and not the cause of the outbreaks.

Though an examination of the viscera revealed the presence of digenetic trematodes, various nematodes and echinorhynchs, as well as occasional cestode larvæ, none of these can be incriminated. The marked fatty degeneration of the viscera has already been mentioned.

(B) Protozoa as a Cause.

(i) *Sporozoa.*

Of the Sporozoa the most important groups in the present connection are the Myxosporidia and the Microsporidia. It is well known that some species give rise to epidemics amongst freshwater fish, while many produce lesions resulting in the death of the host, though an epidemic may not follow. The best known is *Myxobolus pfeifferi* Thel., which at times brings about a tremendous destruction of barbels in the Moselle, Rhine, Meuse, Marne, Aisne, and Seine. Full accounts of this Myxoboliasis are given by Hofer (1906, p. 71) and Gurley (1894, p. 227), in whose works further references to literature are to be found. The fatty degeneration which takes place as a result of that disease reminds one somewhat of the condition commonly associated with the Australian outbreaks, but the "boil formation" so common in the barbel disease is not manifested in our epidemics. Though some observers have referred to the presence of a bluish swelling on the under side of affected fish, it is not a constant feature and is certainly not of the type associated with the European epidemics. A disintegration of muscular tissue of fish is also caused by certain other Sporozoa—*e.g.* some species of *Chloromyxum* and *Glugea* (*G. destruens* Thel.).

Though some of the diseased fish first examined by us were found to be parasitized by Myxosporidia, belonging to the genera *Myxobolus*, *Myxosoma*, *Myxidium*, and *Henneguya*, and occurring in various organs such as the gills, gall-bladder, and kidneys, yet a search through healthy material showed the

presence of similar organisms, so they may be disregarded as direct causes of the mortality. Those met with have been already described by us (J. & B. 1918).

Lymphosporidium truttæ, a member of the Haplosporidia and a parasite of the lymph system, was described by Calkins (1900) as a cause of an epidemic amongst trout. (*See also* Hofer, p. 60 ; Doflein, p. 934.) Another member of this Sporozoon group, *Ichthyosporidium* sp., near *I. gasterophilum* Caull. & Mesnil, has been recorded by Robertson (1909) as fatal to sea-trout.

(ii) *Protozoa—Infusoria and Flagellata.*

The chief ciliate parasites harmful to freshwater fish are *Ichthyophthirius*, *Chilodon*, and *Cyclochæta*. The first-named causes epidemics in aquaria (Hofer, pp. 122-7). The other two appear to be of less importance.

Of the Flagellata, *Costia* is said to cause at times heavy mortality amongst salmon fry in Austria (Franke, 1910 ; Hofer, p. 115).

None of these protozoa except an occasional *Cyclochæta* was detected during our examinations of Queensland material, whether diseased or not.

(c) Fungi as a Possible Cause.

As a result of our examination of fish dead or dying from the disease, we found the fungus *Saprolegnia* constantly present, either on the general body-surface (including fins and tail) or on the gills, or even on both situations. A similar finding was reported by one of us (Johnston, 1917) when dealing with a specimen previously sent down. In addition to the records of Australian occurrences contained in that paper, there is a short one by Waite (1894), who mentioned finding the fungus on *Ctenolates ambiguus* Richdsn. (= *Plectroplites*). Further references to the disease and to literature relating to it can be found in papers by Hofer (1906), Clinton (1893), and Johnston (1917).

It was previously regarded as the cause of the salmon disease, but it is now recognised that the fungus is secondary and is capable of readily exchanging a saprophytic mode of existence for a parasitic one, should it have an opportunity. Such would be given by injuries as well as by the presence of external conditions or diseases which interfere with normal

healthy fish-life. Bacterial maladies of fish are commonly associated with *Saprolegnia* attack, and Shelford (1918c, p. 46) has stated that excessive acidity due to CO_2 probably favours the development of this destructive fungus.

(d) Bacteria as a Possible Cause.

Almost the whole of the diseased material was already dead at the time of our examination, and as putrefactive changes had taken place a bacterial exploration under field conditions would probably have been of little value. Though bacteria were found in various tissues, the possibility of a bacterial disease being the cause of the epidemic had not been provided for by us, and as a consequence cultures were not able to be made on the occasion of our only visit to a locality in which the epidemic was present.

Several bacterial diseases of fish are known, the most destructive being probably that which has at different times caused heavy mortality amongst salmon. The so-called "salmon disease," which was formerly attributed to *Saprolegnia*, has been shown by J. H. Patterson in his Parliamentary Report, Fishery Board for Scotland (1903), to be due to a diplobacillus, *Bacillus salmonis pestis*. Additional information was given by Drew (1909). [*See also* Hofer, pp. 19-22]. Marsh had previously (1902, 1904) described *Bacterium truttæ*, a pleomorphic organism, sometimes assuming the form of a coccus, or a bacillus, or a diplobacillus, which was found to be fatal to various kinds of trout in the United States.

Hofer (1906) gave a summarised account of the various bacterial diseases described as occurring in freshwater fish in Central Europe, the causative organisms being *Bacillus salmonicida, B. pestis astaci, B. cyprinicida, B. anguillarum, B. vulgaris, B. piscicidus, B. piscicidus agilis*, and a few others.

Grieg Smith (1900a) described one as *B. piscicidus bipolaris*, which was found to be fatal to certain marine fish in New South Wales ; and also another (1900b) called by him *Vibrio bresimæ*, which destroyed marine bream in that State.

7. IS IT DUE TO MORE THAN ONE CAUSE?

The epidemic nature of the Queensland disease, the apparently rapid course which it runs, and the particular susceptibility of certain species, all suggest that the malady is

of bacterial origin, and that various local and climatic conditions favour it. We then offer the suggestion that the epidemic may be due to a high acidity on account of excess of CO_2 in stagnant water, favouring the spread of a virulent bacterial disease amongst the weakened fish. The presence of *Saprolegnia* aggravates the condition, as probably also does hydrogen sulphide, which is itself a decomposition product resulting from the effects of the disease.

SUMMARY.

A very destructive epidemic makes its appearance amongst freshwater fish in Queensland and Northern Territory at very irregular intervals, usually during dry and cold conditions, ceasing when the rivers run freely. The affected streams belong to various watersheds—*e.g.*, Lake Eyre basin (Cooper's Creek, Diamantina, Georgina, Thomson, and Wilson Rivers); Bulloo River; Warrego River; Darling system (McIntyre and Severn Rivers); Flinders system (McKinley River); Burnett. It is apparently not due to the following:—Use of explosives; dry weather; low temperature; overstocking; animal parasites (helminth or protozoon).

It is suggested that the prime cause will be found to be a bacterial organism, whose spread is favoured by a high acidity of the water due to excess of CO_2. The presence of the fungus *Saprolegnia* aggravates the disease, as no doubt does hydrogen sulphide also.

ADDENDUM.

Dr. J. Shirley informed us that a series of epidemics causing considerable mortality occurred in the Logan River, in South-eastern Queensland, during the drought years just prior to 1902, but further information regarding these outbreaks is not yet available.

Mr. W. B. Alexander supplied us with the following statement which is of interest. Early in July 1921 a steamer arrived at Fremantle with its hold on fire, and large quantities of water were pumped into it. Among the cargo was a consignment of cyanide for the Kalgoorlie mines, which partly dissolved and entered the harbour water, causing the death of a large number of fish belonging to many different species. The occurrence was reported in the Perth daily Press.

LITERATURE.

1913. Abbott, J. F.—Effect of distilled water upon the fiddler crab. Biol. Bull. 24, 1913, pp. 169-174.

1914. Birge, E. A., & Juday, C.—A limnological study of the Finger Lakes of New York. Bull. Bur. Fisheries U.S.A. 32, 1912 (1914), pp. 529-609.

1898. Bumpus, H. C.—The reappearance of the tile-fish. Bull. U.S. Fish Commiss. 1898.

1900. Calkins, G.—*Lymphosporidium trutiæ*, the cause of a recent epidemic among brook trout, *Salvelinus fontinalis*. Zool. Anz. 23, 1900, pp. 513-20.

1894. Clinton, G. P.—Observations and experiments on *Saprolegnia* infesting fish. Bull. U.S. Fish Commiss. 13, 1893 (1894), pp. 163-172.

1911. Doflein, F.—Lehrbuch der Protozoenkunde. Edit. 3.

1909. Drew, G. H.—Some notes on parasitic and other diseases of fish. Parasitology 2, 1909, pp. 193-201.

1910. Franke, J.—Radical prevention of *Costia necatrix* in Salmonoid fry. Bull. Bur. Fisheries, U.S.A. 28, 1908 (1910), pp. 919-928.

1914. Gilchrist, J. D.—An inquiry into the fluctuations in fish supply on the South African Coast. Marine Biol. Rep. 2, Cape of Good Hope, 1914, pp. 8-35.

1894. Gurley, R. R.—The Myxosporidia or psorosperms of fishes and the epidemics produced by them. Rep. U.S. Fish Commis. 1892 (1894), pp. 65-304.

1917. Hornell, J.—A new protozoan cause of widespread mortality among marine fishes. Rep. No. 2, Madras Fisheries Bull. 11, 1917, pp. 53-66.

1883. Heath, N.—Effect of cold on fishes. Tr. N.Z. Instit. 16, pp. 275-8; Bull. U.S. Fish Commiss. 4, pp. 369-371.

1915. Hedley, C.—An ecological sketch of the Sydney Beaches. P.R.S. N.S.W. 49, 1915, pp. 15-77.

1906. Hofer, B.—Handbuch der Fischkrankheiten, Stuttgart.

1882. Ingersoll, E.—On the fish mortality in the Gulf of Mexico. Pr. U.S. Nat. Mus. 4, 1881 (1882), pp. 74-80.

1917. Johnston, T. Harvey.—Notes on a *Saprolegnia* epidemic amongst Queensland fish. P.R.S. Q'land 29, 1917, pp. 125-131.

1918. Johnston, T. H., & Bancroft, M. J.—Some new sporozoon parasites of Queensland freshwater fish. P.R.S. N.S.W 42, 1918, pp. 520-8.

1903. Marsh, M. C.—A more complete description of *Bacterium truttæ*. Bull. U.S. Fish Commiss. 1902 (1903), pp. 411-415. (*See also* Science 16, 1902, p. 706.)

1917. Magath, T. B.—The morphology and life-history of a new trematode parasite, *Lissorchis fairporti*. Jour. Parasit. 4, 1917, pp. 58-69.

1915. McCallum, G. A.—Some new species of ectoparasite trematodes. Zoologica 1, 1915, pp. 395-410.

1901. Mishikawa, T.—*Gonyaulax* and the discoloured water in the Bay of Agu. Annot. Zool. Jap. 4, 1901, pp. 31-34.

1914. Powers, E.B.—The reactions of crayfishes to gradients of dissolved carbon dioxide and acetic and hydrochloric acids. Biol. Bull. 27, 1914, pp. 177-200.

1917. Powers, E. B.—The goldfish as a test animal in the study of toxicity. Illinois Biol. Monogr. 4 (2), 1917 ; 73 pp.

1919. Pratt, H. S.—Parasites of freshwater fishes. U.S. Bur. Fisheries ; Circular 42, 8 pp.

1909. Robertson, M.—Notes on an Ichthyosporidian causing a fatal disease in sea-trout. P.Z.S. 1909, pp. 399-402.

1908. Rosenberg, A.—Experience in abating disease among brook trout. Bull. U.S. Bur. Fisheries 28, 1908, pp. 941-5.

1913. Shelford, V. E., & Allee, W. C.—The reactions of fishes to gradients of atmospheric gases. Jour. Exp. Zool. 14, 1913, pp. 207-266.

1914. Shelford, V. E.—Suggestions as to indices of suitability of bodies of water for fishes. Trans. Amer. Fisheries Soc. Dec. 1914, pp. 1-14.

1915. Shelford, V. E., & Powers, E. B.—An experimental study of the movements of herring and other marine fishes. Biol. Bull. 28, 1915, pp. 315-334.

1917. Shelford, V. E.—An experimental study of the effects of gas waste upon fishes, with special reference to stream pollution. Bull. Illinois State Lab. Nat. Hist. 11 (6), 1917, pp. 381-410.

1918a. Shelford, V.E.—Ways and means of measuring the dangers of pollution to fisheries. Illinois Nat. Hist. Survey, Bull. 13.

1918b. Shelford, V. E.—The relation of marine fishes to acids with particular reference to the Miles acid process of sewage treatment. Puget Sound Biol. Station, 2 (39), 1918, pp. 97-111.

1918c. Shelford, V. E.—The conditions of existence. Chapter 2 in "Freshwater Biology" by Ward and Whipple, pp. 21-60.

1919a. Shelford, V. E.—Fortunes in wastes and fortunes in fish. Scientific Monthly, August 1919, pp. 97-124.

1919b. Shelford, V. E.—Reaction of fishes to H ions. Proc. Amer. Soc. Zool. in Anat. Rec. 1919, 15, p. 347 (Summary in J.R.M.S. 1919, p. 33).

1900a. Smith, R. Grieg.—A new bacillus pathogenic to fish. P.L.S. N.S.W., 1900, pp. 122-130.

1900B. SMITH, R. GRIEG.—A fish disease from George's River. P.L.S. N.S.W. 1900, pp. 605-9.

1917. TAYLOR, H. F.—Mortality of fishes on the west coast of Florida. Bur. Fisheries, U.S.A. ; Document 848, 1917, 22 pp.

1902. TORREY, H. B.—An unusual occurrence of Dinoflagellata on the Californian Coast. Amer. Nat. 36, 1902, pp. 187-192.

1894. WAITE, E. R.—[Exhibit of a golden perch attacked by *Saprolegnia ferax.*] P.L.S. N.S.W. 9 (2), 1894, p. 740.

1918. WARD, H. B.—The elimination of stream pollution in New York State. Trans. Amer. Fisheries Soc. 48, 1918, pp. 1-25.

1918B. WARD, H. B.—Parasitic flatworms. Chapter in "Freshwater Biology" by Ward and Whipple (Wiley, New York).

1913. WELLS, M. M.—The resistance of fishes to different concentrations and combinations of carbon dioxide and oxygen. Biol. Bull. 25, 1913, pp. 323-347.

1914. WELLS, M. M.—Resistance and reactions of fishes to temperature. Trans. Illinois Acad. Sci. 7, 1914 ; Reprint, 11 pp.

1915A. WELLS, M. M.—The reactions and resistance of fishes in their natural environment to acidity, alkalinity, and neutrality. Biol. Bull. 29, 1915, pp. 221-257.

1915B. WELLS, M. M.—The reactions and resistance of fishes in their natural environment to salts. Jour. Exp. Zool. 19 (3), 1915, pp. 243-283.

1916. ELLS, M. M.—Starvation and the resistance of fishes to lack of oxygen and to KCN. Biol. Bull. 31, 1916, pp. 441-452.

1918. WELLS, M. M.—The reactions and resistance of fishes to carbon dioxide and carbon monoxide. Bull. Ill. State Lab. Nat. Hist 11 (8), 1918, pp. 557-569.

1918. WHETMORE, A.—The duck sickness in Utah. U.S. D.A. Bur. Biol. Survey, Bull. 672, June 1918, 25 pp. A preliminary report by this author was published as "Mortality among Waterfowl around Great Salt Lake, Utah," Bull. 217, U.S. D.A. 1915, 10 pp.

1891. WHITELEGGE, T.—On the recent discolouration of the waters of Port Jackson. Rec. Austr. Mus. 1 (9), 1891, pp 179-192.