FURUNCULOSIS IN TROUT AND THE IMPORTANCE OF CARRIERS IN THE SPREAD OF THE DISEASE[1].

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(With a Map.)

Introduction.

Under the authority of the Kennet Valley Fisheries Association, an inquiry into the cause of the prevalence of furunculosis among trout in the River Kennet was commenced in May 1926.

A bad epidemic of this disease had occurred in the river in 1924. It again broke out in the following year to a less but still serious extent. A grant was accordingly obtained from the Ministry of Agriculture and Fisheries to conduct a research in the next disease season (1926) as to the source of infection, its method of spread and the conditions favouring the latter.

With regard to the source of infection, a review of the literature on the subject suggested three chief possibilities: (1) Trout farms; (2) Carriers (*i.e.* infected fish with no signs of disease) in the river; and (3) Sewage pollution. These three sources were investigated in turn in the course of the summer as recorded below.

The method of spread was not so easy a matter to tackle, particularly as in the year under survey only a mild epidemic occurred. Laboratory experiments under necessarily artificial conditions were, therefore, carried out to test infectivity by wounds, water infection and contact with carriers. Others were planned to test transmission by feeding and the production of immunity by injection of dead cultures, but, owing to illness, these latter were not carried out.

The question of sewage pollution was investigated by analyses of the river water at certain infected and non-infected points between Marlborough and Newbury, and by laboratory experiments devised to show the action of sewage on the growth of *Bac. salmonicida*.

The area of the River Kennet under survey extended some 20 miles from Marlborough to Newbury and included practically all of the valuable trouting water. Throughout this part it is a shallow slow-flowing stream, coursing through a valley of rich pasture meadow and water-cress land.

Two sewage farms discharge their effluent into this area, the one below Marlborough, the other below Hungerford. Some years ago the former was responsible for crude pollution of the river sufficient to destroy practically

[1] From a Report to the Kennet Valley Fisheries Association on Work Carried Out in 1926–27.

all the fish in that section, and at that time steps were at once taken to stop such fouling.

Furunculosis is said to have first appeared in the Kennet shortly after 1911. In that year the disease was rampant in several rivers in S.W. England (Exe, Wye, Trent, etc.) and the infection of the Kennet may have been due to the importation of hatchery fish from that area. Since then the disease seems to have maintained its hold, but did not cause alarm till the severe epidemic of 1924.

The disease tends to commence towards the end of May or beginning of June, following, it is said, the mayfly on its course up stream, and to drop off and disappear in October with the onset of cold weather. The fish attacked are as a rule good-sized and well-conditioned trout. Local observers all agree that they have never seen a "black fish" taken out dead with obvious signs of furunculosis.

The river is largely stocked from year to year either from trout farms or from local "stews" ("native" trout hatcheries) on its banks. The stocking is usually done in March after cutting the river weed and netting pike.

The river bed provides abundance of natural food supply in the way of caddis worm, mayfly larvae, shrimps and snails. At one time fresh-water cray-fish were equally abundant, but these were wiped out some years ago by some unknown disease.

Local anglers complain that the average weight of Kennet trout has fallen off considerably of recent years. This may possibly be due to increase in numbers consequent on stocking. But another change for the worse, more difficult to account for, is the prevalence of "black fish" in the river. The typical Kennet trout is a light-coloured, stocky, deep-shouldered fish of beautiful proportions, and the "black fish," lean, lank and ugly as they often are, are naturally regarded as unsound and undesirable intruders. That there is additional reason for this view will appear later.

Course of Investigation.

The work of investigation was begun on May 11th. At that time there was no ascertainable disease in the river, and it was decided to devote the first few weeks of the inquiry to visiting trout farms in S. England and to examining samples of the farm trout of various sizes for evidence of furunculosis overt or latent. These farms send trout for stocking purposes all over the country in the winter and spring months, and the importance of ascertaining the presence or absence of the disease in them is obvious.

The farms visited were:

	Date	No. examined
Berks Trout Farm (Hungerford)	May 21	20 trout
Exe Valley Trout Farm (Dulverton)	May 26	36 ,,
Surrey Valley Trout Farm (Haslemere)	May 28	34 ,,
Upper Dun Trout Farm (Hungerford)	June 1	20 ,,

The fish examined were all Brown Trout (*S. fario*) with the exception of six "Rainbows," and varied in size from one-year-olds to "Breeders" of

5 lb. or more in weight. They were sectioned on the spot. Cultures were made in every case from the blood. The body cavity was opened and after sterilising the area selected for puncture with a searing-iron, a capillary pipette was driven through the kidney in its posterior third into the dorsal vein. The blood so obtained was at once transferred to culture tubes of broth and blood- or serum-agar. Cultures were also made from gut contents but only in the case of fish showing marked gut congestion. These cultures were then taken back to the laboratory the same day or the next, kept at room temperature for ten days, and any suspicious colonies picked off and subcultured on appropriate media. All cultures (110 blood and 7 gut) proved negative for *B. salmonicida*.

The great majority of the fish examined at farms looked perfectly healthy, though gut congestion was not uncommon. A few backward, ill-conditioned and recently spawned fish were obtained from one farm, but these also gave negative cultures.

It may here be mentioned that in 1911 one of the above-mentioned farms was found to be infected, one dead and one living trout being received from its ponds in June, both of which yielded cultures of *B. salmonicida*. However, no evidence of disease could be found among its fish at this time (May 1926).

These results appeared to indicate that the trout farms were not harbouring the disease nor disseminating it by the supply of infected trout. It was, therefore, decided not to visit any more trout farms and attention was turned to the Kennet, where the disease was now beginning to appear.

Early in June the finding of an occasional dead fish in the river was reported, and on June 14th a dead grayling (from the Chilton Park water) and three days later a dead trout (from the Ramsbury water) were received, both of which gave typical cultures of *B. salmonicida*. Attention was now concentrated on one infected section (Ramsbury), the proprietor of which was kind enough to allow the taking of live fish for examination for carriers.

These were not easy to secure in spite of the use of a net and shot-gun. There was much weed in the way of the net, and shooting usually failed unless the fish was within a few inches of the surface.

The river was visited for this purpose about once a week, and any dead fish found at the time were also removed and taken back for examination.

The first carrier came from the Chilton Park water on June 18th and was sent to the laboratory alive. It was a "black fish," lean and in poor condition, but free from all signs of furunculosis. Its blood gave a pure culture of *B. salmonicida*, but the culture from the gut was negative.

Another "black fish" was received from the same water on July 3rd in much the same condition as the first. It had, however, a small cavity about the size of a lemon seed in the muscle of the left flank. There was no congestion in and around its wall, and it appeared to be a furuncle in a chronic or healing condition. Cultures from the blood and the furuncle were positive. The gut was not examined as it had been shattered by the bullet wound.

Another fish which proved to be a carrier was shot in the Ramsbury water on July 14th. This was a light-coloured, recently spawned fish with a congested gut, but no other signs of disease. Culture from the blood was positive, that from the gut negative.

In all, seven dead fish, all light-coloured, and eighteen living fish were taken from the Kennet (see Appendices I and II). Of the latter eight were black and ten light-coloured. Six of the dead fish showed signs of the disease and cultures of *B. salmonicida* were obtained from them, one had no lesions and was not infected. Of the eighteen living fish from the Kennet three proved to be infected, of which two were black and had no signs of furunculosis, whilst the third was light-coloured and in good health except for the presence of a very small and apparently chronic furuncle.

By way of comparison nineteen living fish (fifteen black, three light trout, one grayling) from the Test at Whitchurch (Hants) and the Lambourne, a tributary of the Kennet, rivers believed to be free from disease, were also examined for *B. salmonicida* with negative results.

Appearances at Autopsy.

Abscesses or pinkish areas of congestion in the muscle were present in eight out of eleven infected dead fish. These varied in size from a five-shilling piece to a pin's head, in number from one to four, and in position, from the flank to the under surface close to the gills.

Four fish showed marked congestion of the gut, with blood at the vent. This figure is probably too low, for in four other fish decomposition made it impossible to say what the condition of the gut was at death. This sign, however, was not uncommon in healthy fish, especially in trout farms.

Worm infection (Nematodes) of the gut, and to a less extent of the swim-bladder, was the rule in the river fish. Occasionally the infection was very severe and was then associated with great loss of condition.

Bacteriology.

The *Bacillus salmonicida* was found in eleven out of thirteen dead fish sent for examination from different rivers (see Appendix I), viz. seven trout, three salmon and one grayling.

Its distribution in the body appeared to be general, for it was possible to recover it from abscesses, blood, liver, kidney, gut and stomach. As a rule cultures were made from abscesses (when present), blood, gut, liver or kidney. If the fish was fairly fresh the cultures from blood, muscle (abscess wall) and internal organs were fairly pure and showed the characteristic pigment of *B. salmonicida* in three or four days. In those from the gut, however, and in others from extensively decomposed fish, the colonies of *B. salmonicida* were as a rule greatly outnumbered by other organisms and not easy to isolate. The development of pigment was then inhibited and it was only by searching out and subculturing small round colonies with a friable consistency that the

presence of *B. salmonicida* was detected. After a little experience, the peculiar consistency of the colonies on blood agar plates proved a very useful character by which to detect the *B. salmonicida* on plates containing a mixture of bacteria. The *B. salmonicida* colony was found to split under pressure from the platinum loop and could be pushed along the culture medium in two separate parts.

No exhaustive cultural study was made of the bacillus, as that had already been done by Arkwright (1912, 1913). Three characters were taken as essential for a positive diagnosis:

(1) Non-motility in broth.
(2) Liquefaction of gelatin.
(3) Production of chocolate-brown pigment.

All the strains of *B. salmonicida* met with during the inquiry (viz. Kennet, Lyon, Lledr, Bourne (Test) and a culture from the Coquet) satisfied these three tests.

The liquefaction of gelatin and the production of pigment were found to vary considerably in time of onset. As a rule the time was three to four days for pigment and two for liquefaction. But occasional colonies picked from a plate yielding normal cultures of *B. salmonicida* required much longer to develop pigment or to show liquefaction.

An additional character which was observed and proved of some assistance in isolating the bacillus was the halo of translucency and decoloration which surrounds the colonies on a blood-agar plate. This medium made with rabbit's blood was, therefore, almost invariably used for isolation, since both the pigment formation and the clear halo were well seen on it.

SEWAGE POLLUTION.

The question of Sewage Pollution as a factor in the production of furunculosis was dealt with in August.

Water samples were taken for analysis from eleven points on the River Kennet from Marlborough to Newbury (see Map), six on August 13th and five on August 28th. Two sewage farms, at Marlborough and Hungerford, discharge into this section, and the points were selected above and below these, as well as above, in, and below the towns of Marlborough, Hungerford and Newbury, so as to reveal possible sources of pollution, and afford comparisons between waters infected and non-infected with furunculosis.

The samples were collected in a leaded sterile bottle cast from a bridge when available or, when not, from the bank. The bottle was then transferred to an ice-box carrier, taken back to the laboratory, placed for the night in the cold room and examined the following day.

Counts of colonies were made after growth at room temperature and at 37° C. and the *B. coli* content estimated in the usual way. Cultures in lactose, peptone water, litmus milk and in broth for indol were used as confirmatory tests.

The results are given in Appendix III.

The analysis shows fairly high counts of saprophytic organisms in the Ramsbury and Hungerford areas and below the Marlborough sewage farm.

B. coli was present in all samples in quantities varying from 1 c.c. to 0·01 c.c., being most numerous in Marlborough (below sewage farm) and Ramsbury (below some riverside cottages).

There is, however, no indication of serious pollution at any of the eleven points chosen. The Ramsbury counts are, it is true, rather higher than most of the others, but the difference is hardly distinctive enough to indicate any connection with the prevalence of disease.

B. salmonicida was not recovered from any of the samples.

The following experiment was made in the laboratory to test the effect of foul as contrasted with pure water on the growth of *B. salmonicida*.

Samples of crude sewage and sewage effluent were obtained from Hungerford, and 100 c.c. of each placed in a separate test-tube, with two controls of tap and distilled water. 0·1 c.c. of a broth culture of *B. salmonicida* was then sown into each, and the tubes were incubated at room temperature and examined daily for the presence of the bacillus by sowing a loopful from each on agar plates.

From the sewage tubes it was not recovered after 48 hours, whereas it persisted in the tap water for three to four days, and in the distilled for fourteen days and over.

This experiment was repeated on two other occasions with similar results.

Pathogenicity.

The following experiments were performed to test possible modes of infection and spread. The fish used were goldfish and gobies.

(1) 27. iv. 1926. *Infection by external wound.*

One goldfish was scarified on the left shoulder and the wound rubbed with a culture of *B. salmonicida*. It remained alive and well.

(2) 3. v. 1926. *Infection by intramuscular inoculation.*

0·2 c.c. of a broth culture of *B. salmonicida* was injected into the left shoulder of a goldfish. The fish died on the third day. The bacillus was isolated from blood and muscle.

(3) 11. xi. 1926. *Infection by water.*

Twelve gobies were placed in a sink of circulating water (about three litres) in the laboratory, with sandy bottom and stones. Three broth cultures were poured in on Nov. 11th and the water circulation stopped for six hours to increase the chance of infection. This was repeated on two subsequent days (Nov. 12th and 14th).

Five fish died between Nov. 29th and Dec. 11th (15–26 days later), all of which gave cultures of *B. salmonicida* from liver and vein but no furuncles were present. No more deaths occurred in the next ten days and the survivors

were presumed to be ready for use as potential carriers in the next experiments.

(4) 23. xii. 1926. *Infection by carriers*[1].

Three of the survivors from Exp. 3 were removed from the sink on Dec. 23rd, marked by a nick in the left labial fin, and placed in a bowl of circulating water with six fresh healthy gobies.

The first death occurred about seven weeks later (Feb. 14th). This was one of those previously exposed to infection. It was not examined.

Five more died in March (10th, 16th, 19th, 25th, 28th), of which three, on examination, proved to be infected with *B. salmonicida*; and three (the remainder) in April (1st, 4th, 11th) also all yielded positive cultures.

It was not noted at the time which of the last eight fish which died were possible carriers. Since, however, only two possible carriers survived till March, four at least of the six, found to be infected post mortem in March and April, must have acquired their infection by contact with the carriers.

This experiment shows that fish (gobies) which were the apparently healthy survivors of the previous experiment, in which some of their companions had become infected and died, passed on the infection to other fish with which they were placed in contact in a clean bowl of running water. These latter fish died after not less than 77 days after their first contact with the carriers. In none were any furuncles found, but the blood and organs were shown to be infected.

To summarise these two experiments:

Exp. 3 showed that out of twelve healthy fish (gobies) kept in water infected with cultures of *B. salmonicida*, five died 18–30 days after first exposure to infection and seven were still apparently healthy 42 days after first exposure.

Exp. 4. Three apparently healthy survivors (possible carriers) of Exp. 3, 42 days after first exposure and 12 days after the last death in the tank, were placed in contact in an uninfected bowl of water with six healthy fish (gobies). These nine fish all died in the course of the next 109 days. Of these nine fish, eight were examined and *B. salmonicida* was recovered from six. Of the six which died and proved to be infected, at least four had become infected by contact with the carriers. These died between 77 and 109 days after first exposure to carrier infection.

Discussion.

Whatever the original source of infection may have been, there is no evidence to show that the persistence of furunculosis in the Kennet is due to the introduction from year to year of trout from infected trout farms. Our observations are against this assumption, for out of over 100 farm fish of various sizes from four different farms not one fish was found infected.

[1] The working out of this experiment is entirely due to the kindness of Mrs M. M. Barratt, who sectioned all the fish during the writer's absence.

In the river, on the contrary, three out of eighteen live trout were found to be harbouring *B. salmonicida* in their blood. All three were ill-conditioned fish, one almost blind, one recently spawned, and one with a small punched-out ulcer in one flank. But they were alive and active, and were, therefore, in all probability a chronic source of infection to healthy fish in the river. The bacilli, it is true, were not recovered from their gut contents but this may have been due to some fault in technique, such as insufficient plates. According to Plehn (1909), a German worker of long experience, the bacillus is met with "fairly frequently" in the gut of outwardly healthy fish in infected waters. Accepting that and the finding of three carriers out of eighteen live fish in the Kennet, it seems reasonable to infer that carriers are now the source of the infection in the Kennet, and the cause of its persistence from year to year.

The extent of the investigation both in regard to the number of fish examined and period of time during which examinations were made is perhaps too small to warrant a final conclusion. But it strongly suggests the advisability of a systematic examination of a large number of trout in a selected area at suitable times, *e.g.* before, during and after the furunculosis season (May, August and November), in a subsequent year.

A knowledge of the "carrier rate" on a sufficiently large scale at such different seasons would be of great epidemiological value. It would throw light on such obscure points in the spread and persistence of the disease as duration of infectivity of carriers. The effect of a dry summer and of the winter season on such carriers, and the difference in the "carrier rate" in healthy years and in years during which the disease is prevalent, would also be valuable information. It might then be possible to gauge the probable course of the disease in a given season, and test the effect of such preventive measures as avoidance of stocking and the removal of ill-conditioned fish from the river.

The danger of carriers spreading the disease was further emphasised by laboratory experiments on gobies. These appear to show:

(1) That pollution of water with *B. salmonicida* may lead to the infection and death of fish (gobies) without the existence of obvious surface wounds.

(2) That fish surviving such an infection can and do infect healthy fish placed among them in uninfected water.

The part played by "black fish" is also worth further study. They appear to be a type of fish not normally inhabiting the Kennet. They are as a rule ill-conditioned, and yet they seem never to have been found dead with obvious signs of furunculosis. Two out of three carriers found in the Kennet were "black fish," while all of the six dead trout were "light."

As regards the question of sewage pollution, no evidence was obtained that this is responsible for disease in the Kennet. Analyses of river water done in August revealed no serious state of pollution at any of the eleven points selected. Nor was there any distinct relationship between high bacterial content and presence of furunculosis infection.

Further, a few experiments done in the laboratory to test the action of sewage on *B. salmonicida*, suggested that sewage and sewage effluent are not specially favourable to its growth. The bacillus could not be recovered from the sewage after 48 hours, and yet persisted in distilled water for more than 14 days. This finding, however, is completely at variance with recent German work (Plehn, 1924), which goes to show that *B. salmonicida* thrives in foul water but dies off rapidly in pure. The explanation of such contradictory results possibly lies in the use of crude sewage and sewage effluent. But more experimental work is certainly needed.

In this connection the case of the River Lyon in Perthshire is of particular interest as an example of a heavily infected river with apparently negligible sewage pollution. It is a swift, clear Highland stream, flowing through a secluded mountain valley. Small communities of 10–60 people are dotted along its banks (above Fortingall), and the sewage is disposed of by dry closets or water closets with small septic tanks discharging into the river. This has been the system in vogue for about 20 years. Yet epidemic furunculosis was reported for the first time in 1926 (260 salmon being taken from the river dead by August 7th), a year with a dry summer, low water, and fish crowded in the pools. The dry season probably intensified the pollution due to sewage, but, under the conditions as stated, carriers are much more likely to have been the main cause of the outbreak.

Again, the recent record of the finding of an Exe salmon in May 1925 with unmistakable signs of furunculosis, which had that morning been caught in the estuary nets, confirms the view that salmon carry infection with them from the sea. The recent discovery of the bacillus in a sea louse from a salmon, by James, of the Ministry of Agriculture and Fisheries (information privately communicated), points to the same conclusion.

Carriers then would appear to be the main reservoir of infection, and the chief obstacle to its removal. It is, of course, not possible to deal with them as such. But a great deal can probably be done to stop disease and prevent its recurrence by maintaining a healthy native stock of fish under conditions as favourable as possible, *e.g.* (1) by avoidance of over-stocking and over-crowding, (2) by bacteriological scrutiny from time to time of the source from which fresh stock is derived, (3) by removal of sick and ill-conditioned fish, (4) by sewage control.

By such measures the danger of severe epidemics will be greatly reduced and the disease will tend to disappear as a natural resistance is established and the carriers die off.

SUMMARY AND CONCLUSIONS.

1. No evidence was found of the presence of furunculosis in trout farms. Over 100 trout from four farms in S. England were examined with entirely negative results.

2. Of eighteen live trout taken from the Kennet, three were carrying

the *B. salmonicida* in their blood. All three were in poor condition but alive and active, and two of them were so-called "black fish."

3. Bacteriological analyses of Kennet water at eleven selected points were carried out in the month of August. They revealed no serious pollution nor any definite relationship between high bacterial content and the disease.

4. Laboratory experiments to test pathogenicity of *B. salmonicida* appear to show:

(*a*) That the bacillus is lethal to goldfish when injected into the muscle but not necessarily so when rubbed into a skin wound.

(*b*) That in the case of gobies infection from water polluted with pure broth cultures of *B. salmonicida* is possible and may cause death in about fifteen days after the introduction of the culture.

(*c*) That fish (gobies) which had survived the last experiment for a period of six weeks were capable of infecting healthy "contacts," when kept with them in fresh running water.

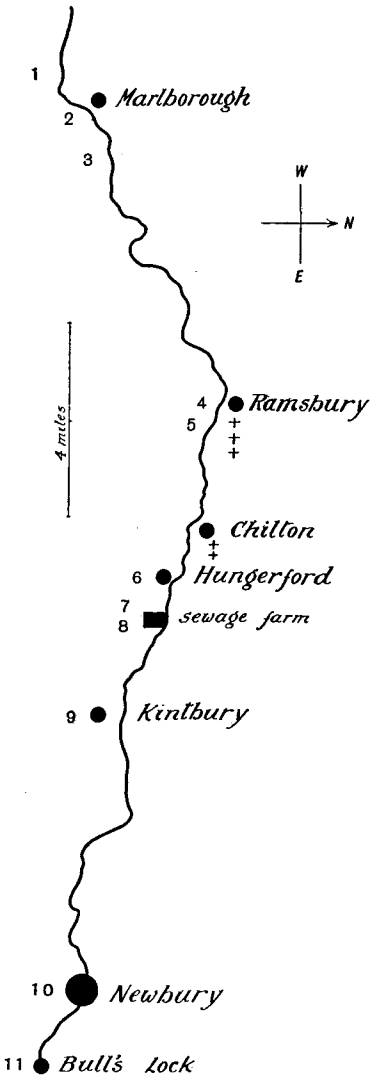
5. A few laboratory experiments on the behaviour of *B. salmonicida* in sewage appeared to indicate that the bacillus persists longer in tap and distilled water than in crude sewage and sewage effluent.

6. Specimens of fish (salmon and trout) infected with furunculosis were also received during the summer from the Rivers Lledr (Wales), Lyon (Scotland) and a private hatchery on the Test at Whitchurch. The conditions in the two latter cases suggested that the outbreak was mainly due to carriers.

7. Measures suggested in the case of infected streams are: avoidance of over-stocking, bacteriological scrutiny of source of fresh stocks, removal of sick and ill-conditioned fish, and sewage control.

APPENDIX I. *Examination of dead fish.*

No.	Fish	Date	Source	P.M. findings	Cultures	Result
1.	Grayling ...	June 14	Chilton Park	Decomposed. Open sloughing sore on flank	Muscles	+
2.	Trout ...	June 17	Ramsbury	Congested gut	Blood, gut	+
3.	„	„ 25	„	Congested gut, red areas in muscle	Blood, gut, muscle	+
4.	„	„ 25	„	Abscess	Blood, gut, muscle	+
5.	„	„ 30	„	„	Blood, gut, abscess	+
6.	„	July 14	„	Abscesses, decomp.	Blood, abscesses	+
7.	„	Aug. 27	„	Nil	Blood, gut	−
8.	Grayling ...	June 28	Lambourne	Bruise on flank	Blood, gut, muscle	−
9.	Salmon ...	Aug. 5	Lyon	Decomposed	Muscle, kidney	+
10.	„	„ 5	Lledr	Abscesses	Abscesses, blood, liver	+
11.	„	„ 5	„	„	Abscesses, blood, liver	+
12.	Trout ...	Sept. 14	Bourne (Test) Stews	„ decomp.	Abscesses, blood, liver	+
13.	„	„ 14	Bourne (Test) Stews	Red areas in muscle, decomp.	Muscle, liver, blood	+



Map of River Kennet. The numbers 1–9 indicate the sources of water samples. The crosses indicate the areas where Furunculosis occurs.

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APPENDIX II. *Bacteriological examination of live trout in search for carriers.*

Kennet.

Area	No. of fish	Black	Light	Positive	Negative
Ramsbury ...	8	4	4	1	7
Chilton Park ...	3	2	1	2	1
Hungerford ...	5	2	3	–	5
Avington	2	–	2	–	2
Totals ...	18	8	10	3	15

Other Rivers.

Rivers					
Lambourne ...	2 trout	1	1	–	2
„	1 grayling	–	–	–	1
Test	16	14	2	–	16
Lledr	2 salmon	–	–	–	2

Details as to carriers from the Kennet.

Date	Fish	Source	Colour	Condition	P.M. signs	
June 18	Trout	Chilton Park	Black	Blind, long and lean	—	Blood +
July 3 ...	„	„	„	Long and lean	Small abscess on left flank	Blood and abscess +
July 14...	„	Ramsbury	Light	Recently spawned, congested gut	—	Blood +

+ = presence of *B. salmonicida.*

APPENDIX III. *Analyses of water samples from the Kennet, Aug.* 13 *and* 28.

No.	Source of sample	Colonies per c.c. at room temp.	Colonies per c.c. at 37° C.	*B. coli* present in
1.	Marlborough footbridge to St George's Church	326	110	0·5
2.	„ Town Bridge (Sewage farm) ...	398	118	0·01
3.	„ Elcot Mill Bridge	1350	102	0·01
4.	Ramsbury—Fraser's Bridge	2000	55	1 c.c.
5.	„ Chamberlain's Cottage	3500	65	0·01
6.	Hungerford—Eddington Mill	352	164	1 c.c.
7.	„ Donford Mill (Sewage farm) ...	956	164	0·05
8.	„ Footbridge below Commoners' water	1300	200	0·5
9.	Kintbury—Station Road Bridge	160	45	0·5
10.	Newbury—Hovis Mill	120	70	0·05
11.	„ Bull's Lock	790	100	0·5

It remains to be noted that during September and October twenty-six samples of water from various points on the Kennet were bacteriologically examined by Messrs Rideal and Sciver (London), who reported that the bacterial condition of the stream was on the whole good, although there was some evidence of pollution in a few side-streams.

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