ETIOLOGY OF SOCKEYE SALMON 'VIRUS' DISEASE

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ETIOLOGY OF SOCKEYE SALMON "VIRUS" DISEASE

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ABSTRACT

Violent epizootics among hatchery reared sockeye salmon fingerlings (Oncorhynchus nerka) caused by a filterable agent have occurred. In 1954, one source of this infectious, filterable agent was found to be adult sockeye viscera used in the diet for the fingerlings. The results of observations on an epizootic in 1958 indicate that the infection may be transmitted to fingerlings from a water supply to which adult sockeye salmon have access.

ETIOLOGY OF SOCKEYE SALMON "VIRUS" DISEASE

A virus-like disease has been recognized as the cause of heavy losses among hatchery populations of sockeye salmon fingerlings in the State of Washington. The following is a brief review of observations of this disease for the last few years and an account of the extensive experimental work conducted at two hatcheries and in the laboratory during 1954.

HISTORY

The disease was first recognized in 1950 at Winthrop, Washington, and during the ensuing years an increasing number of hatcheries were affected by the epizootic (Rucker et al 1953; Watson et al 1954). By 1953 the epizootic had appeared in sockeye salmon fingerlings at four Federal hatcheries in Washington, all located on Columbia River tributaries, and at the Washington State hatchery at Issaquah. The outbreak at Issaquah marked the first appearance of the infection at a hatchery not on a Columbia River tributary.

The disease is characterized by an explosive increase in mortality which may reach 32 percent of the original population per day with a total mortality of over 95 percent of the original population. In 1952, 85 percent of all sockeye fingerlings reared in the State of Washington succumbed to the disease and in 1953 the State-wide toll was 65 percent. Higher hatchery mortalities were prevented in 1953 by the early planting of a considerable number of fish.

ETIOLOGY

The disease is highly species specific, affecting only the fresh- and salt-water strains of Oncorhynchus nerka. Other species of salmon fingerlings reared at the same hatcheries are not affected, and it has not been possible to transmit the infection to other species of salmon or various species of trout even by intraperitoneal injection of highly infectious material obtained from diseased sockeye fingerlings.

None of many and varied chemotherapeutic treatments has stayed the course of the infection nor has a prophylactic treatment been found. Once the epizootic appears, it has been impossible to reduce the high rate of mortality in the diseased population. Not only is the disease impossible to control but it is also highly infectious, particularly in the spring of the year when the fish are very young. The disease is easily transmissible to healthy populations by the feeding of diseased material, placing the fish in a bacteria-free filtrate prepared from infected, moribund, fingerlings for a few moments, intraperitoneal or intramuscular inoculation, or by placing a dead or moribund fish in the water supply above the healthy population. In some instances infectious material obtained from diseased fish was diluted as high as 1 part in 10 billion without loss of infectivity.

SOURCE OF THE INFECTION

Until the fall of 1953 the method of entry of the infection into hatchery populations remained unexplained. As the disease spread to additional hatcheries after it was first detected in 1950 certain possibilities became remote, if not entirely eliminated. A common air-borne or aquatic vector was unlikely to be the method of entry because of the varied locations and climates of the infected hatcheries, the random distribution of infected troughs, and the occurrence of the infection in sockeye salmon being reared in well water.

Experimental work conducted late in 1953 finally produced the first step toward discovery of the source of the infection. During the spawning season of that year the livers of all returning sockeye from which eggs were taken near the Federal hatchery at Leavenworth were tested and a filterable, pathogenic agent, which caused mortalities in sockeye fingerlings, was found in a number of the adults. Since salmon viscera of mixed and unidentified species, obtained in frozen blocks from commercial canneries, were routinely added to the diets of all fish populations affected by the epizootic, it was quite possible that the agent was gaining entry to the hatcheries in the feed. It has been shown that the etiologic agent can withstand freezing for a limited time. This hypothesis could neither be proven nor disproven on the basis of past experience, however, since

the feed had, on many occasions, been used at hatcheries in which the epizootic did not appear. It was common to have diseased fish in a trough adjacent to a trough of healthy fish though both were fed the same food preparation. Although attempts to transmit the etiologic agent to other species of fish had failed, it is possible that the other species, as well as the sockeye adults, were acting as carriers of the infection.

In 1954 a large-scale experiment was undertaken at the two Federal hatcheries at Winthrop and Leavenworth, Washington, in an effort to determine the exact method of entry of the infection into the sockeye fingerling populations.

EXPERIMENTAL PROCEDURE

General hatchery procedure

Each of the two stations, Leavenworth and Winthrop, reared native fish as well as some which had been spawned at the other hatchery and, in addition, the Leavenworth station reared a group of fingerlings spawned at the Entiat station. Special arrangements were made to obtain the frozen salmon viscera on an individual species basis to determine if the sockeye viscera alone were the carriers of the virus or whether other species could transmit the infection (table 1). In addition to the viscera diets, one diet contained silver salmon eggs as the fish component and two diets had no fresh or frozen fish products; one was similar to the Cortland 6A and the other a pelleted feed of private manufacture which was discontinued shortly after it was begun because of its inadequacy to maintain the fingerlings nutritionally.

Maximum sanitary precautions were taken at both stations. Feeding and cleaning protocol was established on the assumption that all viscera were virus contaminated. Solutions of disinfectant and live steam were used liberally throughout the year and separate cleaning and feeding equipment was used for each trough and set of ponds. As an added precaution, the hatcheries themselves were divided into two sections, viz: "clean" and "contaminated" by the use of two feed-preparation rooms (at Leavenworth only) and by division of the hatchery troughs and ponds into two, physically distinct units. Even the men working at the hatcheries were divided into two groups on the basis of which side of the hatcheries they worked on -- crossing over from one side to the other was prohibited. Additional labor was hired so that this division could be maintained during weekends and holidays. Tourists and visitors were conducted through the hatchery rather than being allowed to roam and, at the Leavenworth station, special troughs were established near the main entrance to confine the tours to as small an area as possible. Personnel from other hatcheries were not allowed to enter either the hatchery proper or the feed rooms. The handles of all equipment were color-coded and separate equipment was used for infected ponds and troughs.

Both the experimental fish and the controls were handled as identically as possible. The two groups were weighed and counted at the same time, water flow was adjusted, the same amount of feed was given at the same time of the day, and the groups were moved to new troughs or into the ponds simultaneously.

In the spring, fish at the Leavenworth station had an outbreak of bacterial gill disease which was treated as it occurred. In general, the experimental fish and their controls were treated identically, but one portion of the hatchery was allowed to go untreated so that any effects of the treatment on the virus disease could be detected. After the fish had been placed in the outside rearing ponds they were given weekly prophylactic treatments to prevent the occurrence of the gill infection. Kidney disease was detected early in the year at the Winthrop hatchery but since the mortalities from this disease were low, no treatment was administered.

Leavenworth hatchery

Eggs taken at Lake Wenatchee for the Leavenworth station were spawned in groups of 12 females (about 24,000 eggs per group), hatched, and the fry reared. Representative samples of each group of eggs were tested for virus and all subsequent records of Leavenworth-reared fingerlings were maintained so that any infected trough or pond could be traced back to the original group of females from which it was obtained. No similar grouping was done with the eggs received from Winthrop and Entiat. After the eggs had hatched,

the fry were divided into two groups, one-half to act as controls and the other half used to determine if the disease is transmitted by the diet. All fingerlings received a diet of beef and hog liver until mid-March after which time the control groups continued to receive an all-meat diet without salmon products, while the experimental groups were fed a mixture of 50 percent salmon products and 50 percent hog and beef products. It was thought fairly warm water temperatures are required for the utilization of the Cortland diet so it was withheld until the latter part of July, the beef and hog liver diet being fed until this time.

Increased weight and size of fish necessitated a reduction in the number of fish per trough by early July. At this time a group of surplus experimental fingerlings was planted and 80 troughs of experimental fish were moved outside into 20 small rearing ponds (4 troughs per pond). The fish remaining in the hatchery were distributed throughout the hatchery. As in all other moves each pond and trough on the experimental diet was accompanied by the identical movement of its control. Nineteen of the 20 ponds of both groups were screened with 2-inch poultry netting to prevent birds from having access to the fish, as they previously had been observed dropping moribund and dead fish in uninfected ponds. The remaining pond in each group was covered with a fine-mesh plastic cloth to keep out insects.

Winthrop hatchery

After the eggs spawned at the Winthrop station and those received from the Leavenworth station had hatched, the young fish were divided into 56 troughs: 28 on one side of the hatchery and their 28 controls exactly opposite them on the other side. The experimental diet of 50 percent meat products and 50 percent mixed salmon viscera was begun the last week in February, 1954. These viscera were from the same lot that had been fed the previous year at the Winthrop, Entiat and Leavenworth hatcheries during which time all three hatcheries had been affected by the epizootic. On April 20, the fingerlings were moved outside into 14 small Foster-Lucas ponds -- the control fish on one side of the hatchery and the viscera-fed fish on the other side. On June 16, 1954, one pond of viscera-fed fish was planted, on June 18 three ponds of control fish were planted, and on July 7, two more ponds of healthy, viscera-fed fish were planted. There remained two ponds of fish from the Leavenworth stock and two ponds from the Winthrop stock on the viscera diet and the group of four ponds of control fish. These fish were retained for overwinter holding.

General laboratory procedure

In conjunction with the large-scale hatchery experiments, additional data were obtained in the laboratory. Of particular importance were the factors influencing the spread of the disease and the fact that the virus perpetuated itself despite the extremely high mortality of infected fingerlings.

Whenever a higher than average daily mortality occurred in any troughs or ponds at the experimental hatcheries, the fish were examined immediately in an effort to determine the cause of death. If examination failed to disclose a pathogenic agent, moribund fish and recent mortalities were brought to the Seattle laboratory for testing. They were combined for one minute in a blender and centrifuged for one minute. Dilutions were made of the supernate to give a mixture of 1 part fish in 99 parts nutrient broth; 0.05 to 0.10 ml. of this suspension was inoculated into each of ten disease-free fingerlings. The controls consisted of an equal number of fish inoculated with sterile nutrient broth. If mortalities occurred in the suspension-inoculated group, the same procedure was repeated with the remaining fish and the diluted homogenate was passed through a sterile, 7-pound Mandler filter. The filtrate was inoculated into a minimum of ten fingerlings with appropriate controls. Material from ensuing mortalities was serially transferred to exclude, by total dilution, any toxic effect of the original filtrate. The healthy fish in these experiments were obtained from the control troughs at the Leavenworth hatchery.

To check the possibility of the virus gaining entrance into the hatcheries via an air-borne or aquatic vector, plankton and bacterial counts were made periodically throughout the year at all experimental hatcheries and, in addition, insect and small mammal collections were made at the Leavenworth hatchery. Plankton samples were obtained by placing a plankton net under one of the

trough inlets, flies were obtained from flytraps placed about the feed rooms, and water insects were collected in the river above the Leavenworth hatchery water inlet by routine collection methods. The small mammals and portions of all other collections were stored frozen; plankton and insect samples were tested immediately upon collection. The samples were ground with levigated alumina, centrifuged, and the supernatant solution diluted to approximately 1:100 dilution of the original sample. Quantities of .05 ml. of this solution were then inoculated intraperitoneally into 10 healthy fingerlings reared on the control diet.

In September a small group of recovered and a small group of healthy fish were taken from the Leavenworth to the Winthrop station. Here these fish and healthy and recovered fingerlings reared at the Winthrop station were inoculated with a bacteria-free virus filtrate which varied in dilution from $1:10^2$ to $1:10^8$ and was obtained from moribund Winthrop fish. Other experiments were carried out to study the transmission of the disease in an attempt to establish the natural pattern of the virus infection. Fingerlings which had survived the disease were tested as possible carriers of the disease by placing a group of healthy fish just below recovered fish in the same trough.

In the fall of the previous year the livers of all adult fish spawned at the Leavenworth station were tested for the virus by homogenizing them with an equal quantity of nutrient broth and inoculating 0.1 ml. of the homogenate into healthy fingerlings. Thus, transmission of the virus via the fertilized egg could be detected if it occurred. Serial transfers, as previously described, were made of those fish which became moribund after inoculation with the adult liver extracts. These serial transfers were continued until the first mortalities were occurring in four days indicating the maximum degree of infectivity. At this time the inocula, and further serial transfer inocula, were filtered through Mandler filters prior to injection to demonstrate the presence of an infectious, filterable agent.

Concurrent with the laboratory studies described, an extensive hematological study was undertaken in an attempt to find a method of early diagnosis of the virus infection. The results of these studies have been reported elsewhere (Watson et al 1956).

RESULTS

Leavenworth hatchery

At Leavenworth the virus disease developed in only two troughs. The first outbreak was recognized on May 12, the second in an adjacent trough on June 18. The cumulative mortality L-5/12 and L-6/18 respectively, is shown in figure 1. The fish had a length of one inch or less and were four and one-half to five and one-half months old. These troughs, each containing about 14,000 fish, were part of the group of 23 troughs which was receiving a diet containing British Columbia sockeye salmon viscera.

Winthrop hatchery

At Winthrop the virus infection developed among the fingerlings in three of the four ponds retained on the viscera diet: the virus infection did not develop among the fingerlings in four ponds fed the control diet. Each pond was stocked with about 50,000 fish. Fish in the first pond where disease appeared were spawned at Lake Wenatchee (Leavenworth) and the first increase in mortality came on June 15; the second, stocked with Winthrop-spawned fish, showed a mortality increase on July 16; and the third on July 20 in a pond of fingerlings spawned at Lake Wenatchee. The cumulative mortality of these three ponds is shown as W-6/15, W-7/16, and W-7/20 in figure 1. All three ponds were receiving the diet containing 50 percent mixed salmon viscera. None of the infected ponds was adjacent. It is improbable, therefore, that the agent had been spread from the first pond which became infected.

At the time of the first outbreak the fish were about five and one-half months old and slightly over two inches long. By mid-July the fish were seven and one-half months old and about three inches in length. Confirmation of the virus disease as the cause of the high mortalities in the two troughs and three ponds was obtained by the laboratory procedures previously described.

Laboratory results

Hematological studies (Watson et al 1956) of the fish at the Leavenworth hatchery revealed that some of the fingerlings in those troughs receiving the sockeye viscera diet, other than the two troughs showing the typical accelerating mortality pattern, were succumbing to the virus infection though the remaining fish in these troughs failed to show symptoms of the disease. Additional hematological studies of healthy and diseased fish are necessary to establish definitely this hitherto unsuspected circumstance.

All tests of the plankton and insects in the water supply used at the Leavenworth station were negative nor were any "blooms" noted about the time the virus disease was detected. Routine bacterial counts of the water supply revealed Gram negative, motile rods as the predominant organism.

Experiments designed to demonstrate acquired immunity revealed that recovered fingerlings had complete immunity to the infection. Healthy fish from both stations which had not been previously infected succumbed when inoculated intraperitoneally with 0.10 ml. of a $1:10^5$ dilution of infectious material obtained from moribund fish while the recovered fingerlings successfully resisted a similar dose of a $1:10^2$ dilution. It was noted during the experimental work that among the healthy fish from the Leavenworth hatchery inoculated as described above, the first mortalities occurred in six days while eleven days were required before the Winthrop reared fish began to die.

The "carrier" state of recovered fish was also successfully demonstrated. In mid-July the survivors of the infected troughs at the Leavenworth station were transported to the Seattle laboratory and tested for the presence of the virus by inoculation of a bacteria-free filtrate prepared from these fish into healthy fingerlings. No mortalities occurred among the inoculated fish. Concurrent with this experiment about 50 healthy fingerlings were placed in the same trough and just below the recovered fish. The first mortality among the healthy fish occurred one month later but was considered a "normal" mortality. Two weeks later, however, two more mortalities occurred and experimental evidence showed that these two mortalities were due to the virus. By mid-September two of the formerly healthy fish developed the characteristic "bent-back" which occurs in recovered populations and by mid-December five more fish had manifested this condition.

When the adult fish returned to spawn during the fall months, it was again possible to demonstrate the "carrier" state of the adults. Unfortunately, by this time of the year, the healthy fingerlings have reached an age at which they become refractile to the pathogenic agent so the tests are comparatively insensitive. Two of the 24 adults taken at Lake Wenatchee were found to carry the agent and there was some indication that seven of ten spawning adults taken at Cultus Lake in the Fraser River drainage in Canada were carrying the virus though experimental results were inconclusive. Of the 12 sockeye adults that returned to University of Washington School of Fisheries, Seattle, only one fish was infected; ten from the Issaquah, Washington State hatchery, which is higher up in the same watershed, were found to be negative. Of ten kokanees (the landlocked variant of the sockeye salmon) from Lake Whatcom, Washington, three were carriers.

DISCUSSION AND CONCLUSIONS

The fact that the virus disease, though not as widespread as in previous years, appeared in three of seven ponds fed viscera at the Winthrop station, while at the Leavenworth hatchery the epizootic affected two of the 23 troughs being fed British Columbia sockeye viscera, is significant in view of the fact that none of the meat-fed controls contracted the infection. Because of the high mortality rate the disease may be cyclic in nature, thus accounting for the relatively low total mortality rate throughout 1954. The extensive disinfection of all equipment and the efforts made to prevent the spread of contamination in 1954 cannot be overlooked. Using normal hatchery feeding and cleaning procedures allows the spread of the infection before increased mortalities due to the disease occur. Thus, by using sterile techniques prior to the onset in the first pond or trough, there is little possibility of spreading the virus before the disease is overtly manifest.

There can be little doubt that the primary method of entry of the disease into the hatcheries

.as been via contaminated adult sockeye viscera 'ed to the young fingerlings. The random occurrence and the sporadic nature of the outbreaks of the infection in any one hatchery at first threw considerable doubt on this method of entry since it would be expected that almost all of the fish receiving the same diet would become infected simultaneously, but the hematological studies and other experimental work have shown that there are three possible beginnings to the chain of infection: the virus particles can enter the population in a comparatively large quantity at one feeding but the particles may be concentrated in one portion of the feed since not all viscera may be infected; the particles can enter the population over an extended period of time and accumulate in the fish until their natural resistance to the disease is overcome; or a small percentage of the fingerlings may be hypersensitive to the etiologic agent and the infection spread through cannibalism of the healthy fish on the dead and moribund, thus increasing the number of virus particles in the population by serial transmission.

Of the three possibilities, the last method appears to be the most probable since moribund, virus infected fingerlings, were found in troughs which never suffered a full-scale epidemic. Perhaps only those ponds or troughs with a higher than average proportion of hypersensitive fingerlings first manifest the infection. A combination of contaminated cleaning and feeding equipment and hypersensitive fish can cause the infection to become widespread throughout the hatchery. It cannot be said, however, that the other two methods never operate. During the early part of the year, when the fish are young and small, they have slight resistance to pathogenic agents. Thus, the exact method of entry of the virus into the population is usually masked by the short incubation period of the disease and the high rate of mortality.

The effect of water temperature on the virus is not noticeable until late summer or early fall of the year. During the spring and early summer any effect of water temperature is over-ridden by the high susceptibility of the fingerlings to the disease coupled with water temperatures within the optimum range of the pathogenic agent, 40-60 degrees F. The rise in water temperatures during the summer months affects the fingerlings two ways: by its effect on the size, or physiological age of the fish and by either decreasing the susceptibility of the fish or adversely affecting the virus. The decrease in water temperature in late summer and early fall re-establishes optimum temperature conditions thus accounting for the comparatively few outbreaks during the summer and the fall epidemics.

The total mortality due to the virus disease at the Winthrop hatchery has usually been proportionately lower than that of the Leavenworth hatchery. At first it was thought that a strain difference in either the sockeye salmon fingerlings or the virus was the cause of this phenomenon, but since fingerlings reared at one station from eggs spawned at another station showed the same mortality pattern of the rearing station, this excluded the possibility of strain differences. The warmer average winter and early spring water temperatures at the Winthrop station caused a more rapid growth rate and was considered the major contributing factor.

Other modes of entry of the virus disease into the hatcheries, in addition to the feeding of infected viscera may exist, but more sensitive techniques than those presently being used will be required to detect the pathogenic agent. Recovered fingerlings do not carry virus in sufficient quantity to infect seven-month-old healthy fingerlings when suspensions prepared from these recovered fish are inoculated into the healthy ones intraperitoneally. Nevertheless, when healthy fish are placed in the same water supply as the recovered fish, transmission of the infection does eventually occur even though a full-scale epidemic does not develop in the exposed fish. Perhaps there is some transmission of the virus via the eggs from "carrier" adults -- the fish hatched from these eggs may be the "hypersensitive" fingerlings. However, the virus was in so small a quantity that the inoculated one-year-old fingerlings used to test the samples of eggs were sufficiently refractile to resist the agent. Exclusion of sockeye salmon viscera from the diets of fingerlings may therefore exclude the major mode of entry of the infection in the hatcheries, but the losses in any one hatchery due to a virus epidemic are so great that all possible sources must be eliminated if sockeye salmon fingerlings are to be reared successfully.

ADDENDUM

CONTROL OF THE DISEASE BY DIET CHANGE

The reported experiments of 1954 indicated that the filterable agent causing losses among sockeye salmon fingerlings probably was introduced by the practice of incorporating sockeye salmon carcasses in the diets of young salmon. Therefore, no fresh or frozen salmon products were fed to the sockeye salmon fingerlings reared at the Leavenworth and Winthrop stations after 1954. Turbot or arrow-toothed halibut (Atheresthes stomias) was substituted for the salmon viscera formerly used.

Fish rearing has been successful at the Leavenworth station since salmon products have been eliminated from the diet. The water supply comes from wells and Icicle Creek which is not accessible to salmon.

Up to January 1958, the Winthrop station reared sockeye salmon successfully. Following normal procedure, most of the fingerlings were planted from this station into Lake Wenatchee in the fall of 1957 and a small group was retained for over-winter rearing, to be planted in the Methow River the following spring. Of this group of 125,000 fingerlings, approximately 100,000 died before April 1, 1958, when the surviving 25,000 were planted in the Methow River. Fungus (Saprolegnia parasitica) was present to some extent on the head or body surfaces of the dead fish but most were affected on the peduncle. Standard, external treatments of formalin, Roccal, Lignasan, and pyridylmercuric acetate proved of no avail as did the oral administration of the drugs sulfadiazine, sulfamethazine and terramycin. Diseased fish were taken to the Seattle laboratory where the experimental work was conducted. The disease was maintained for four months by passing millipore filtered material from moribund, inoculated fish to healthy fish. The results of bacteriological examination, including aerobic and anaerobic culturing, were negative: results of histopathologic examination were similar to the disease of former years (Wood and Yasutake, 1956); a filterable infectious agent was shown to be present (table 2). The similarity of the disease in the present study to that in past years is further borne out by its apparent specificity for sockeye salmon (table 3).

The occurrence of the disease at the Winthrop station in January 1958, in the absence of salmon products in the diet, may be attributed to infection through the water supply. Adult chinook and sockeye salmon are present in the Methow River from which the hatchery water supply is taken. Some fish enter this area in June. By preventing adult fish from entering the water supply, it may be possible to eliminate the infection from this hatchery.

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Table 1:--Distribution and diets of experimental sockeye salmon fingerlings retained at the stations after the excess fish were planted in the summer of 1954. An equal number of ponds and troughs were maintained on the all-meat diet as controls.

Hatchery	Number eggs received	Source	Diet	Number troughs or ponds retained
Leavenworth	361,000	Winthrop Canal	Pink salmon viscera	10 troughs 1 pond
	317,000	Entiat River	Pink salmon viscera	15 troughs 1 pond
	3,561,000	Lake Wenatchee	Chinook salmon viscera	10 troughs
			Alaska sockeye salmon viscera	20 troughs
			B.C. sockeye salmon viscera	15 troughs 4 ponds
			Chum salmon viscera	15 troughs 3 ponds
			Pink salmon viscera	20 troughs 4 ponds
			Pink and silver salmon viscera	1 pond
			Silver salmon eggs	15 troughs 3 ponds
			Cortland 6A diet	15 troughs 3 ponds
Winthrop	449,000	Winthrop Canal	Mixed viscera	2 ponds
	529,000	Lake Wenatchee	Mixed viscera	2 ponds

Table 2:--Weekly mortality of groups of 10 healthy sockeye salmon fingerlings inoculated with homogenates from diseased yearling sockeye salmon from the Winthrop station on January 7, 1958.

Week	Homogenate in the water	Homogenate I P	Homogenate filtered I P	Homogenate heated control
1	-	-	-	-
2	4	8	4	-
3	4	-	2	-
4	2	1	3	-
Total	10	9	9	0

Table 3:--Weekly mortality data for groups of 20 healthy salmonid fingerlings and sockeye yearlings subjected to an unfiltered homogenate from diseased sockeye fingerlings in the water. Sockeye fingerlings were tested in duplicate. Controls were subjected to an homogenate from normal sockeye salmon yearlings. Exposed on March 26, 1958.

	Diseased homogenate						Control Homogenate			
Week	Sockeye	Sockeye	Sockeye yearlings	Chinooks	Silvers	Rainbows	Sockeye	Chinooks	Silvers	Rainbows
1	1	3	0	0	0	0	0	0	0	0
2	16	16	3	0	0	0	0	0	0	0
3	1	0	5	0	0	0	0	2	0	0
4	0	1	9	0	0	0	0	0	0	0
Total	18	20	17	0	0	0	0	2	0	0

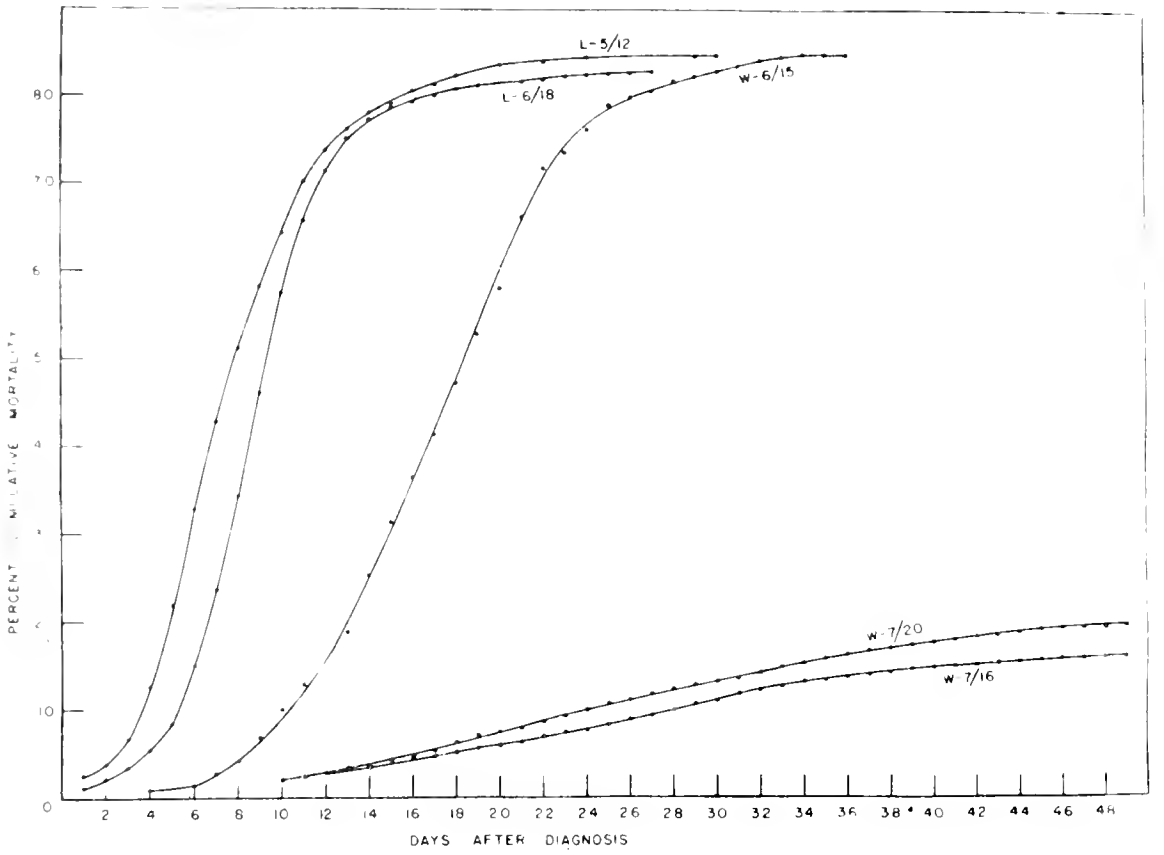

Figure 1:--Virus diagnosis dates and mortality rates of sockeye salmon fingerlings in two troughs at the Leavenworth station (L-5/12 and L-6/18) and three ponds at the Winthrop station (W-6/15, W-7/16, and W-7/20) in 1954.

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