Effects of Copper or Zinc in Fresh Water on the Adaptation to Sea Water and ATPase Activity, and the Effects of Copper on Migratory Disposition of Coho Salmon (*Oncorhynchus kisutch*)[1]

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The 96-h LC50 of copper ($CuCl_2$) for yearling coho salmon (*Oncorhynchus kisutch*) was found to decrease from 74 μg/liter Cu in November to 60 μg/liter in May as the fish became smolts. The 96-h LC50 of zinc ($ZnCl_2$) for yearling coho in April was 4600 μg/liter Zn. All tests were conducted at 10–12 C in water with alkalinity and hardness ranging from 68 to 78 and 89 to 99 mg/liter as $CaCO_3$, respectively.

Exposures of yearling coho for 144 h to sublethal concentrations of zinc in fresh water had little effect on the enzyme activity of Na^+, K^+-activated ATPase in gill microsomes or on the survival of fish transferred to sea water. Acute and chronic exposures (maximum of 4128 h) of yearling coho salmon to sublethal concentrations of copper (5–30 μg/liter) in fresh water had deleterious effects on downstream migration after the fish were placed in a natural stream, gill ATPase activity, and survival in sea water. More severe effects were produced in the chronic exposures than in 144-h exposures on downstream migration and survival in sea water, but not on gill ATPase. Effects of copper began to occur within 24–72 h and were often maximized within 120–144 h of exposure. Concentrations of copper (5–20 μg/liter) were 0.09–0.35 of the 96-h LC50 for coho smolts.

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La CL50 après 96 h du cuivre ($CuCl_2$) pour des saumons coho (*Oncorhynchus kisutch*) de l'année diminue de 74 μg/litre de Cu en novembre à 60 μg/litre en mai au moment où les poissons se transforment en smolts. La CL50 après 96 h de zinc ($ZnCl_2$) pour des saumons coho de l'année en avril est de 4600 μg/litre de Zn. Tous les essais ont été effectués à 10–12 C, dans de l'eau d'alcalinité et de dureté allant de 68–78 et 89–99 mg/litre de $CaCO_3$ respectivement.

Des expositions de saumons coho de l'année durant 144 h à des concentrations sublétales de zinc en eau douce ont peu d'effet sur l'activité enzymique de l'ATPase activée au Na^+ et au K^+ dans les microsomes branchiaux, ou sur la survie de poissons transférés dans l'eau de mer. Des expositions aiguës et chroniques (maximum de 4128 h) de saumons coho de l'année à des concentrations sublétales de cuivre (5–30 μg/litre) en eau douce ont des effets nuisibles sur la migration d'avalaison une fois les poissons placés dans un cours d'eau naturel, de même que sur l'activité de l'ATPase branchiale et sur la survie dans l'eau de mer. Les expositions chroniques ont des effets plus graves que les expositions de 144 h sur la migration d'avalaison et sur la survie en eau de mer, mais non sur l'ATPase branchiale. Les effets du cuivre commencent à se faire sentir en dedans de 24–72 h et atteignent souvent un maximum en dedans de 120–144 h d'exposition. Les concentrations de cuivre (5–20 μg/litre) sont de 0.09–0.35 de la CL50 après 96 h pour les smolts de saumon coho.

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THE seaward migration of juvenile coho salmon (*Oncorhynchus kisutch*) normally occurs during the spring of their second year of life. They are euryhaline several months earlier provided that they achieve a threshold size of 9 cm (Conte et al. 1966). The natural movement or experimental transfer of juvenile anadromous salmonids from fresh water to sea water is followed by a transient but marked disturbance of water–electrolyte balance (Conte et al. 1966; Miles and Smith 1968).

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These disturbances are minimized at the time of normal seaward migration or "parr–smolt transformation."

One of the physiological factors correlated with migratory behavior in steelhead trout (*Salmo gairdneri*) is an elevation of activity in Na^+, K^+-stimulated adenosine triphosphatase (ATPase) in gill microsomes (Zaugg and Wagner 1973). This enzyme activity increases twofold during parr–smolt transformation of coho salmon and steelhead trout (Zaugg and McLain 1970, 1972; Zaugg and Wagner 1973). Activity in salmonids and other euryhaline fishes continues to increase after the first few days in sea water, reaching a maximum after about 30 days, and is thought to be important in maintaining their salt and water balance (Epstein et al. 1967; Zaugg and McLain 1970).

Considerable research has been done to determine the effects of copper or zinc on the survival and growth of juvenile salmonids in fresh water (Chapman 1973; Hodson and Sprague 1975; Lloyd 1960; McKim and Benoit 1971; Sprague 1964; Sprague and Ramsay 1965). However, there is a paucity of information available for establishing water quality criteria as they relate to the migration of fish into sea water following toxicant exposure. There is a particular need for this type of data in the Pacific northwest where large runs of anadromous salmonids are a valuable sport and commercial fishery resource and whose well-being is a significant factor in environmental impact considerations. This study was designed to determine the effects of copper or zinc (in fresh water) on yearling coho salmon's ability to adapt to sea water, ATPase activity, and effects of copper on downstream migration.

Materials and Methods

Experimental Fish

Coho salmon of the 1972 and 1973 year-classes from Fall Creek Salmon Hatchery (Alsea River, Oregon) were used. Fertilized eggs were incubated at temperatures that ranged from 7.8 to 12.3 C. The fish were reared under natural light or simulated natural photoperiod in well water at a constant temperature of 12.3 C. In 1974 the experimental fish (1972 brood year) were reared and tested at a constant temperature (12.3 C). In 1975 the experimental fish (1973 brood year) were reared and exposed to toxicant at 12.3 C until February 1. Fish were then acclimated to 8.6 C over a 6-day period and exposed to a simulated natural temperature; 8.6 C on February 7 to 12.3 C on May 19. The temperature pattern used was based on mean biweekly maximal and minimal temperatures for the North Fork of the Alsea River.

Fish were fed a commercially prepared moist pellet generally to repletion. Fish were not fed during the 24 h preceding exposures to toxicant, during acute ($\leqslant$144 h) toxicant experiments, nor during exposures to sea water. Feeding of fish was resumed in the chronic experiments after the first 144 h.

Exposures to Toxicant

All exposures were conducted on coho salmon between the ages of 10 and 18 mo posthatching. Fish were exposed to copper ($CuCl_2$) for periods ranging from 24 to 4128 h. Four 144-h exposure tests were also conducted with zinc ($ZnCl_2$). All 96-h LC50s were determined from static water bioassays in 61-cm diam fiberglass tanks. The water was continuously aerated and 85% of the 120 liters of test water was exchanged daily. Toxicant solutions were mixed in a separate container prior to introduction to the tanks. Total metal concentrations in daily water samples were assayed by atomic absorption spectrophotometry. Loss of metal from the test water during any 24-h period was undetectable. The tanks were maintained at 12 $\pm$ 1 C in 1974 and 10 $\pm$ 1 C in 1975, with simulated natural photoperiod. The above procedure was used for the static 144-h copper exposures in 1974, and all zinc exposures.

Exposures of more than 144 h, as well as some $\leqslant$144 h, were conducted in a flowing water system. This system consisted of a gravity flow dilutor that delivered 12 liters/min to each exposure tank. Nominal concentrations of copper were 30, 20, 10, 5 and 0 μg/liter and were duplicated. The background concentration of copper in the well water was less than 2 μg/liter. A volume of 1000 liters was maintained in each of the ten 1.5-m diam fiberglass tanks. At the measured flow rate, 99% of the water was replaced every 5.5 h. A submersible pump was used in each tank to provide additional mixing.

Smaller groups of "stock" fish were exposed to the flowing toxicant in 61-cm diam fiberglass tanks to provide test fish with shorter toxicant exposures. Flows to these 120-liter tanks were provided by siphons from mid-depth in the 1.5-m tanks, with no change in water quality observed. All tanks were covered with nets to prevent loss of fish. In 1975 a curtain was installed around the periphery of the tanks to minimize disturbance from external sources.

Concentrations of copper used were chosen on the basis of prior static water bioassays. Total copper concentration was analyzed at least once per week in water from each exposure tank by atomic absorption spectrophotometry. Water and toxicant flows in the dilutor were checked at least once daily. Only minor adjustments were required. Nominal concentrations of toxicant will be used in the Results and Discussion since measured concentrations were within $\pm$10% of these values. A summary of routinely measured water quality is listed in Table 1.

Tolerance to Sea Water

Tests of tolerance to sea water were conducted on samples of 10–20 fish from each toxicant exposure tank. Survival was used as a measure of osmoregulatory ability. Fish were placed into the 61-cm diam

TABLE 1. Range of values for water quality assays in test tanks during exposures to toxicant.

Parameter and units	Exposure system	
	Static	Flowing
Dissolved oxygen: mg/liter	6.0–10.8	7.3–10.5
Alkalinity: mg/liter $CaCO_3$	68–78	72–75
EDTA hardness: mg/liter $CaCO_3$	89–99	84–99
pH	6.8–7.9[a]	7.2–7.5
Total ammonia: mg/liter NH_3-N	0.04–0.44[a]	0.12–0.57

[a]Reflects diel fluctuations caused by daily exchange of 85% of the water in the test tanks.

fiberglass tanks containing 120 liters of natural sea water. Salinity of the water ranged from 29 to 33‰ in 1974 but was maintained at 30.0 ± 0.5‰ in 1975. The tanks were maintained at 12 ± 1 C in 1974 and 10 ± 1 C in 1975, under simulated natural photoperiod. The seawater exposure period was 15–20 days in 1974 but was reduced to 10 days in 1975 because all deaths usually occurred before the eighth day.

During the tests in sea water, dissolved oxygen in the water was maintained at 6.4–8.5 mg/liter and pH at 7.4–8.1. The total ammonia ranged from 0.04 to 1.8 mg/liter NH_3-N due to the daily exchange of 85% of the water in the test tanks.

Downstream Migration

Effects of copper on migration were assessed by releasing fish into a tributary to the North Fork of the Alsea River and enumerating migrants entering a trap 6.4 km downstream. The trap was built into a permanent weir and usually was checked daily. On the day prior to release, 50–100 fish from each copper test tank were anesthetized with MS-222®, weighed, measured, and identified by freeze branding and fin-excision. Parr–smolt transformation is markedly size dependent and seasonal in this stock. Wild coho juveniles normally spend 1 yr in the natural stream before migrating seaward provided they have reached the size of 7 cm. Under artificial propagation most fish reach a size of 11–15 cm in less than 1 yr. No fish less than 10 cm were released. Seaward migration normally begins in April, peaks in late May, and ceases by late June. Releases of copper intoxicated fish and their controls were made between April 8 and June 4, 1975. trapping was terminated in early July, 1 mo after the last release.

Gill ATPase Activity

The activity of Na^+, K^+-activated ATPase in microsomes isolated from gill filaments was assayed by the procedures of Zaugg and McLain (1970). The activity was measured in three to six fish per toxicant concentration prior to release into the stream and before most tests in sea water. Enzyme assays were also made from gill tissues of survivors of two tests in sea water.

No inhibition of the ATPase activity occurred when $CuCl_2$ was added to homogenized gill tissue from control fish. Final concentrations of copper in the spiked homogenates (0.04 gm of tissue/ml of homogenate) were up to 10 μg/ml Cu, which was over 200 times the concentration found in the gills of coho salmon exposed to 30 μg/liter Cu in fresh water. This suggests that the decreased ATPase activity observed in gills from fish exposed to copper was not a result of copper ions liberated during the homogenization procedure. Free copper ions are probably bound by the EDTA (5mM) in the homogenizing solution (W. S. Zaugg unpublished data).

Results and Discussion

In the first 20 mo of study, six static water bioassays of copper and four of zinc were completed. In addition, 16 tests of tolerance to sea water were run using fish exposed to copper in the flowing toxicant system. Only a few of the tests will be discussed.

96-H LC50 Experiments

The 96-h LC50 of copper for juvenile coho salmon was determined at 3 different times. In late November it was 74, in early March it was 70, and by late May it dropped to 60 μg/liter Cu. The greater sensitivity to acutely lethal effects of copper in May was probably due to the onset of smoltification rather than increasing age or size of fish. This physiological transformation, which preadapts coho salmon to a seawater existence, apparently increased their susceptibility to lethal effects of copper in fresh water. The observed increase may have been modified slightly in either direction because the earlier tests were conducted at a higher temperature (12 C) than the May test (10 C) and the interactions between temperature and metal toxicity are complex (Hodson and Sprague 1975). Water hardness, alkalinity, pH, ammonia, and dissolved oxygen values were similar in all tests.

Our 96-h LC50 values of copper for coho

TABLE 2. Survival and gill ATPase activity of yearling coho salmon (*Oncorhynchus kisutch*) exposed to copper in static fresh water and their subsequent survival, and gill ATPase activity after transfer to sea water.

Nominal concn (*μg/liter Cu*)	% Survival in toxicant (*144-h exposure*)[a]	ATPase activity in toxicant[b]	% Survival in sea water	ATPase activity in sea water[b]
		Mar. 20–Apr. 8, 1974[c]		
0	100	12.9	94	51.4
20	100	7.1	6	63.4[d]
30	100	5.6	0	
50	95	5.0	0	
60	85	—	0	
80	25	—	0	
		Apr. 16–May 6, 1974[e]		
0	100	25.5	100	57.3
5	100	16.3[f]	94	58.6
10	100	9.8[f]	59	57.8
20	100	6.6[f]	24	53.7[f]
30	100	6.5[f]	6	70.2[d]
60	30	4.0[g]	0	

[a]Twenty fish exposed per concentration.
[b]Gill microsomal Na^+, K^+-activated ATPase; micromoles ATP hydrolyzed per milligram protein per hour; mean of four fish at the end of exposure.
[c]312-h seawater exposure.
[d]Only one fish.
[e]336-h seawater exposure.
[f]Mean of three fish.
[g]Mean of two fish.

salmon are higher than those reported by Sprague (1964) for Atlantic salmon (*S. salar*) and Lloyd and Herbert (1962) for rainbow trout (*S. gairdneri*). However, the alkalinity and hardness of our water (66 and 95 mg/liter $CaCO_3$, respectively) were about 4 times greater, and our fish were larger and of a different genus than those used in the cited studies. Chapman (unpublished data) found that the 96-h LC50 of copper for juvenile coho salmon (same stock as used in our studies) was 28–38 μg/liter Cu at the Western Fish Toxicological Station, Corvallis, Oregon, with a water hardness and alkalinity of 20–25 mg/liter $CaCO_3$.

The 96-h LC50 of zinc for yearling coho salmon was 4600 μg/liter in April at 12 C. Our 96-h LC50 value for zinc is higher than that reported by other investigators for salmonids (Chapman 1973; Hodson and Sprague 1975; Sprague 1964). However, the greater alkalinity and hardness of the water in our studies may account for the difference.

SURVIVAL IN SEA WATER AND GILL ATPASE

The survival and gill microsomal Na^+, K^+-activated ATPase activity of yearling coho salmon exposed to copper for 144 h in static fresh water and their survival following transfer to sea water in two 1974 tests is presented in Table 2. The ATPase activity in fresh water and the percent survival in sea water were decreased in proportion to the concentration of copper. The decreased ATPase activity probably was one of the factors leading to loss of osmoregulatory ability and death in sea water. The ATPase activity in fish that survived exposure to sea water for 312 h was increased two- to threefold over the values obtained for coho in fresh water, corroborating the results of Zaugg and McLain (1970).

Exposures of yearling coho for 144 h to sublethal levels of zinc in static fresh water ($\leqslant$2000 μg/liter) did not seriously affect subsequent survival in sea water (Table 3). The percent survival in sea water was not consistently related to the zinc concentrations, reflecting the tendency of fish to become hypersensitive and hyperactive to stimuli during and after exposures of $\geqslant$1000 μg/liter Zn. These responses were quite variable and were sometimes followed by tetanic spasms and death of the fish.

Gill microsomal Na^+, K^+-activated ATPase activity appears unaffected by zinc (Table 3). The apparent stimulation in the March test is probably an artifact of the fish analyzed as controls as these fish were subjected to a low copper

TABLE 3. Survival and gill ATPase activity of yearling coho exposed to zinc in static fresh water and their subsequent survival, and gill ATPase activity after transfer to sea water.

Nominal concn (*μg/liter Zn*)	% Survival in toxicant (*144-h exposure*)[a]	ATPase activity in toxicant[b]	% Survival in sea water	ATPase activity in sea water[b]
		Mar. 20–Apr. 8, 1974[c]		
0	100	12.9[d]	100	51.4
100	100	22.2	94	70.1
300	100	—	100	65.7
600	100	26.6	100	76.9
1000	100	24.4	81	95.0
2000	100	29.8	81	70.5
2500	100	25.8	87	66.2
		Apr. 16–May 6, 1974[e]		
0	100	25.5[f]	100	57.3
2500	85	21.6[f]	100	48.6[f]
4000	75	21.8[f]	67	46.5[f]
5000	65	20.2[f]	80	58.3[f]
6000	10	—	50	—

[a]Twenty fish exposed per concentration.
[b]Gill microsomal Na^+, K^+-activated ATPase; micromoles ATP hydrolyzed per milligram protein per hour; mean of four fish at the end of exposure.
[c]312-h seawater exposure.
[d]Control also served for copper exposed fish concurrently being run (see Table 2).
[e]336-h seawater exposure.
[f]Mean of three fish.

TABLE 4. Gill ATPase activity and survival in sea water of yearling coho exposed to copper in fresh water for ≤144 h (tests conducted from March 6 to June 9, 1975). Number of fish is in parentheses.

Nominal concn (*μg/liter Cu*)	Exposure time (*h*)	ATPase activity in toxicant (*mean ± SD*)[a]	% Survival in sea water (*240–312-h exposure*)
0	144	46.3±8.8 (18)	100.0 (103)
10	144	39.3±8.0 (4)	98.8 (83)
20	144	—	60.7 (107)
30	144	13.4±3.1 (8)	10.1 (99)
30	120	15.4±1.5 (4)	9.5 (21)
30	96	17.2±4.6 (4)	45.0 (20)
30	69	—	61.0 (59)
30	56	30.2±13.5 (4)	—
30	41	—	78.0 (41)
30	24	—	75.6 (41)

[a]Gill microsomal Na^+, K^+-activated ATPase; micromoles ATP hydrolyzed per milligram protein per hour.

contamination (13.7 μg/liter). The acute toxicity of zinc to yearling coho salmon appears to involve a different mechanism than that of copper, as indicated by the difference in effects of the two metals on gill ATPase and subsequent survival in sea water.

Yearling coho salmon subjected to 30 μg/liter Cu in fresh water for only 24 h suffered a 25% mortality upon transfer to sea water (Table 4). The number of deaths in sea water increased with longer exposures to toxicant but reached a maximum at 96–120 h of copper exposure (Table 4). Inhibition of gill ATPase was also observed in these exposures (30 μg/liter Cu), although data on exposures of less than 56 h were not obtained.

The effects of copper on seawater tolerance

were greater after exposures to toxicant in the static water system (Table 2) than after exposures in the flowing water system (Table 4). This was probably due to the manual water changes (once per day) which caused greater excitation of the fish and fluctuations in water chemistry (Table 1) during the static exposures. The values for gill ATPase activity in Table 2 (1974) are lower than those in Table 4 (1975) but probably do not represent biological differences. The discrepancies may be due partially to the use of a different centrifuge for enzyme isolation in 1975.

The survival of juvenile coho salmon in sea water following exposure to copper in fresh water for 144 h at different times of the year is shown in Fig. 1. The effects were dependent upon copper concentration during tests on any given date, but there was a trend toward reduced effect with the onset of smoltification in the fish. Disturbance of osmotic balance in the fish upon transfer to sea water is minimized during smolting process (Conte et al. 1966). This physiological process reduced the deleterious effects of copper on survival of smolts in sea water, although increasing size and age may have helped.

In June to early August 1974, four survival tests in sea water were run on coho salmon from the flowing toxicant system after 144–880 h of copper exposure. From mid-December to early June 1975, five survival tests in sea water were carried out with coho salmon exposed to copper from 144 to 4128 h. Data showing the survival in sea water of exposure groups in 1975 are shown in Fig. 2. A noticeable increase in tolerance to sea water was seen as the season progressed, very similar to the pattern observed for coho salmon exposed for only 144 h (Fig. 1). However, the chronically exposed fish generally had less tolerance for sea water than did the 144-h exposed fish. This was due partly to poorer condition and

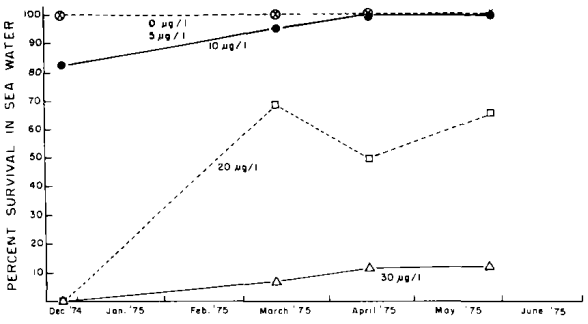

Fig 1. Survival of juvenile coho (*Oncorhynchus kisutch*) in sea water following exposure to copper in fresh water for 144 h at different times of the year (each point represents 16–45 fish).

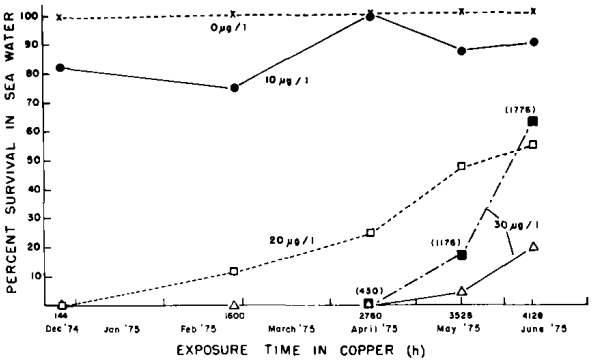

FIG. 2. Survival of juvenile coho in sea water at different times of the year following exposure to copper in fresh water starting on December 20, 1974 (each point represents 20–45 fish). Numbers in parentheses indicate exposure hours of an additional group of fish placed in copper on March 27, 1975 (first two points represent 40 fish, last point represents 11 fish).

nutritional state and smaller size of the chronically exposed fish resulting from a suppression of feeding in the copper toxicant, in a concentration dependent manner. These same factors precipitated an earlier onset of mortality in sea water (Fig. 3B).

A marked recovery of ability to survive in sea water during May and June was observed in the survivors of groups chronically exposed to 30 µg/liter Cu (Fig. 2). The fish that remained were copper-resistant and subsequently began to recover their appetite. Recovery of feeding improved their condition and nutritional state, and consequently improved their tolerance for sea water. In both groups chronically exposed to 30 µg/liter Cu, recovery of feeding responses began to occur in May, although one group had been exposed for over 3500 h and the other for less than 1200 h. Therefore, during the April, May, and June tests of survival in sea water, fish in the group exposed for the shorter time to copper were larger and in better condition as well as having begun parr–smolt transformation before their exposure to copper started on March 27. It must be a combination of these factors that resulted in the quicker and greater recovery of tolerance to sea water in this group.

No deaths occurred during the 144-h freshwater copper exposures (Fig. 3A) but upon transfer to sea water the coho previously exposed to 20 and 30 µg/liter began to die following 72 h in sea water. In the chronically exposed groups deaths in the 30µg/liter Cu (freshwater) began to occur after 144 h of exposure and reached an asymptote after 1800 h (Fig. 3B). Coho salmon yearlings, previously exposed to 20 and 30 µg/

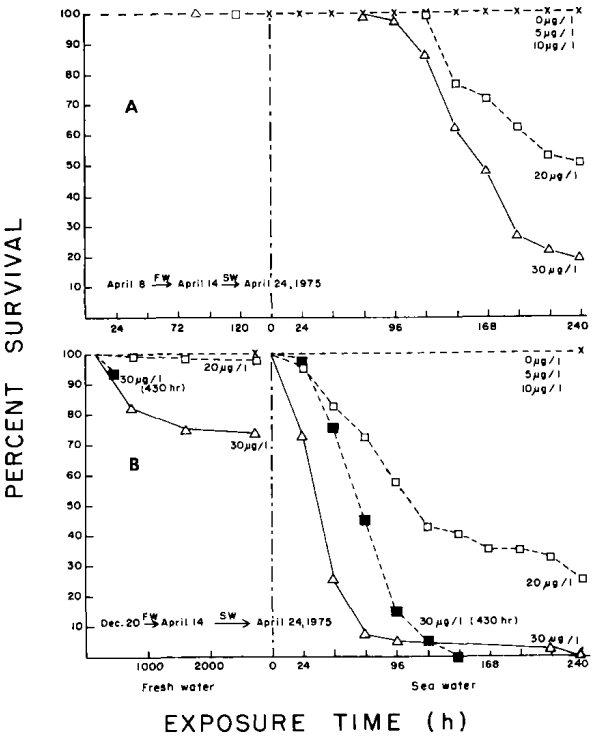

FIG. 3. Survival curves of yearling coho during exposures to copper in fresh water and subsequent seawater exposure in April. A, Copper exposure for 144 h and then transferred to sea water for 240 h; B, Copper exposure of 2760 h (also includes a group exposed to 30 µg/liter for 430 h) and then transferred to sea water for 240 h.

liter Cu for over 2700 h, began to die within 12 h following transfer to sea water (Fig. 3B). This lower tolerance for sea water of the chronically exposed fish is due partially to their poorer condition.

The gill microsomal Na^+, K^+-activated ATPase activity of juvenile coho salmon chronically exposed to copper starting on December 20, 1974 is shown in Fig. 4. A peak in activity occurred in the control fish during April and May, corroborating the data of Zaugg and McLain (1970). Gill ATPase activity was suppressed by copper, but did not reflect the increasing tolerance of coho salmon to sea water that occurred as the season progressed (Fig. 2). This lack of correlation and the large variability in activity levels between fish in the same group limits the use of gill ATPase activity as an indicator of tolerance.

Downstream Migration

Four releases of coho salmon exposed to copper since late December and two releases of coho exposed to copper for 144 h were made into

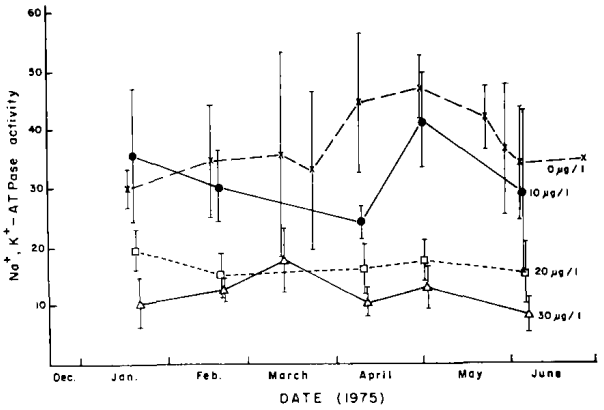

FIG. 4. Influence of exposure to copper in fresh water on gill microsomal Na^+, K^+-activated ATPase activity of juvenile coho chronically exposed to copper starting on December 20, 1974. Each point represents four to six fish (micromoles ATP hydrolyzed per milligram protein per hour ± SD).

Crooked Creek, a tributary of the Alsea River. The releases were made from early April to June 1975, during the normal migration period. The control fish showed the best movement with fewer fish migrating with increasing copper concentration (Fig. 5). Similar trends of movement occurred in the various exposure groups released earlier. Even the lowest concentration used (5 µg/liter), which had no measurable effect on gill ATPase activity or survival in sea water in the 1975 experiments, reduced the percentage of downstream migrants. As noted in the seawater tolerance tests, survivors of a group chronically exposed to 30 µg/liter (initiated March 27) had recovered some of their migrational abilities as the season progressed (Fig. 5).

A greater percentage of fish from the 144-h

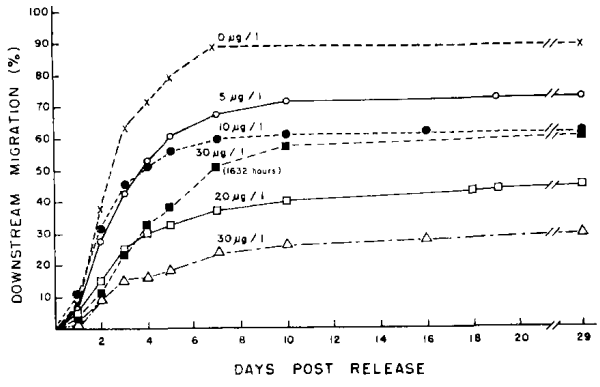

FIG. 5. Influence of exposure to copper for 3960 h in fresh water on percent downstream migration of yearling coho (includes coho exposed to 30 µg/liter Cu for 1632 h). Each line represents 78–172 fish released on June 4, 1975.

exposure groups migrated than did the chronically copper exposed fish at each respective concentration. Exposure to 30 μg/liter Cu for as little as 72 h caused a considerable reduction in migration as compared to control fish (52 and 93% movement, respectively).

Most of the fish that moved downstream to the weir did so within the 1st wk following release (Fig. 5). Only a few fish showed delayed migration arriving at the trap between 8 and 29 days after release. A large percentage of the fish that did not migrate by early July, including control fish, are presumed to have died. In migration studies in prior years, electrofishing the stream above the weir following the normal migrational period accounted for 5% or less of the fish that did not migrate.

These results indicate that concentrations of copper that were sublethal to yearling coho salmon in fresh water reduced the chances of successful migration to the ocean and adaptation to sea water. These concentrations (5–20 μg/liter Cu) were 0.09–0.35 of the 96-h LC50 of copper for coho salmon during their normal seaward migration in May. Direct effects of copper on ATPase activity and adaptation to sea water began to occur within 24–72 h and were maximized within 120–144 h. Secondary effects related to suppression of appetite in the copper toxicant magnified the effects on downstream migration and adaptation to sea water in exposures >144 h.

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