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Title: THE HERITABILITY OF RESISTANCE TO GAS BUBBLE DISEASE OF COLUMBIA RIVER FALL CHINOOK SALMON, Oncorhynchus tshawytscha

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Dr. John D. McIntyre

A nested mating experiment in which 20 males were each mated to four different females was used to obtain an estimate of the heritability of resistance to death from gas bubble disease in Columbia River fall chinook salmon (Oncorhynchus tshawytscha). Heritability estimates ranged from 0.037 to 0.038.

Bioassays in 127 percent air supersaturated water were used to compare the inherent resistance to gas bubble disease of selected Columbia and Trask River stocks of juvenile fall chinook salmon. In the first of two comparative bioassays, time to 50 percent mortality in offspring from adults obtained at Little Goose Dam on the Snake River was more than twice as great as that in offspring from adults obtained from the Trask River. In the second experiment, no significant difference in resistance to death from gas bubble

disease was found among several lower Columbia River stocks of fall chinook; however, the average resistance of these stocks was significantly greater than that of Trask River fall chinook.

A population model which incorporated response to selection for increased resistance to gas bubble disease was used to predict the numbers of fall chinook returning in the future to Kalama Hatchery. It was found that smolt survival can be expected to show a maximum increase of five to ten percent after 30 years of selection. These results indicated that gas bubble disease will remain as a major source of mortality of fall chinook smolts in the Columbia River until lethal levels of air supersaturation are eliminated.

The Heritability of Resistance to Gas Bubble Disease of Columbia River Fall Chinook Salmon, (*Oncorhynchus tshawytscha*)

by

Steven Putman Cramer

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Assistant Professor of Fisheries
in charge of major

Redacted for Privacy

Head of Department of Fisheries and Wildlife

Redacted for Privacy

Dean of Graduate School

Date thesis is presented [illegible]

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INTRODUCTION

Construction of a series of dams completely inundating the Columbia River from tidewater up to its confluence with the Snake River has resulted in annual periods of air supersaturation of the Columbia River during spring and early summer (Beiningen and Ebel, 1971). Air supersaturation is caused by the entrainment of air in water at depths up to 30 ft in the plunge basins of the spillways below each dam. This has been substantiated by the finding that the level of air supersaturation in the Columbia River varies according to the flow of water over spillways of dams (Ebel, 1969).

Supersaturation levels which are known to be fatal to salmonid fishes (Rucker and Hodgeboom, 1953; Westgard, 1964; Ebel, 1969; and Blahm *et al.*, 1973) are often sustained in the Columbia River from April through July. This period corresponds to the peak migration time of juvenile salmonids down the Columbia to the ocean. This coincidence has resulted in substantial mortalities of migratory juvenile salmonids due to air embolisms within their circulatory systems (personal communication with Howard Raymond, fishery biologist at the National Marine Fisheries Service Biological

Laboratory, Seattle). The effects of air supersaturation on fish are known collectively as gas bubble disease.

Salmonids have shown a great amount of variability in their ability to tolerate supersaturation (Ebel, 1969; and personal communication with Dr. Gerald Bouck, Environmental Protection Agency, Western Fish Toxicology Station, Corvallis). If a portion of this variability is related to additive genetic factors, then the levels of mortality due to gas bubble disease in the Columbia River may represent significant selection for inherently more resistant fish. If this is true, a change in the genetic make-up and an increase in the average tolerance of salmon populations to air supersaturation can be expected. It was the purpose of this study to examine the genetic effects of air supersaturated water on fall chinook salmon (Oncorhynchus tshawytscha).

The specific objectives of this study were:

1. To determine the heritability of resistance to death from gas bubble disease of Columbia River fall chinook salmon.

2. To determine the inherent level of resistance of several Columbia River fall chinook salmon stocks to gas bubble disease by the comparison of bioassay results.

3. To predict, through the use of a population simulation model, the effect natural selection for increased resistance to gas bubble disease may have on future runs of fall chinook in the Columbia River.

METHODS

Heritability Estimation

Experimental Fish

Juvenile fall chinook salmon produced in a nested breeding experiment were obtained from Abernathy Salmon Cultural Development Center, near Longview, Washington. Progeny from 80 families were obtained when they were four months old. These families were produced by mating a series of 20 males, each to four separate females. This mating design was similar to Experiment 1 of Comstock and Robinson (1948). At the time the matings were made, the eggs from each female were randomly assigned to separate incubator trays and incubated at 12° C. When the larvae reached the "eyed stage", the number from each cross was reduced to 3100. Three weeks after hatching, the fry from each family were placed in separate 60 in x 15 in x 12 in rearing tanks supplied with 4 gal/min of water at 14.5° C, and were fed measured amounts of Abernathy dry pellet diet (Fowler and Burrows, 1971).

Experimental Methods

To estimate the contribution of additive genetic factors to differences in resistance to gas bubble disease, it was necessary to

define a variable that could be used to determine the relative resistance of the experimental fishes. Two such variables, time to death for each fish after exposure to air supersaturated water and the percentage survival for each family after a set exposure time, were studied in this experiment. These data were obtained by exposing the experimental fish to a lethal level of air supersaturated water in an experimental apparatus constructed at the Abernathy Salmon Cultural Development Center.

One hundred fish from each of the previously described families were marked by cold branding (Everest and Edmundson, 1967). Combinations of numbers and letters were used so that each family had a unique mark. Fish were allowed to recover for seven days before being subjected to air supersaturated water. At the time of testing, the fish weighed an average of 2 g/fish.

Thirty marked fish from each family were put into each of three circular tanks 6 ft in diameter and 1 ft in depth. These groups of 30 marked fish will be referred to as tank families. The remaining ten marked fish from each family were put into a similar tank and served as a control. The three test tanks were supplied with 5 gal/min of water which was air supersaturated to 130 ± 1.5 percent.

Air supersaturated water was produced by the apparatus shown in Figure 1. Before being supersaturated, the water was aerated to

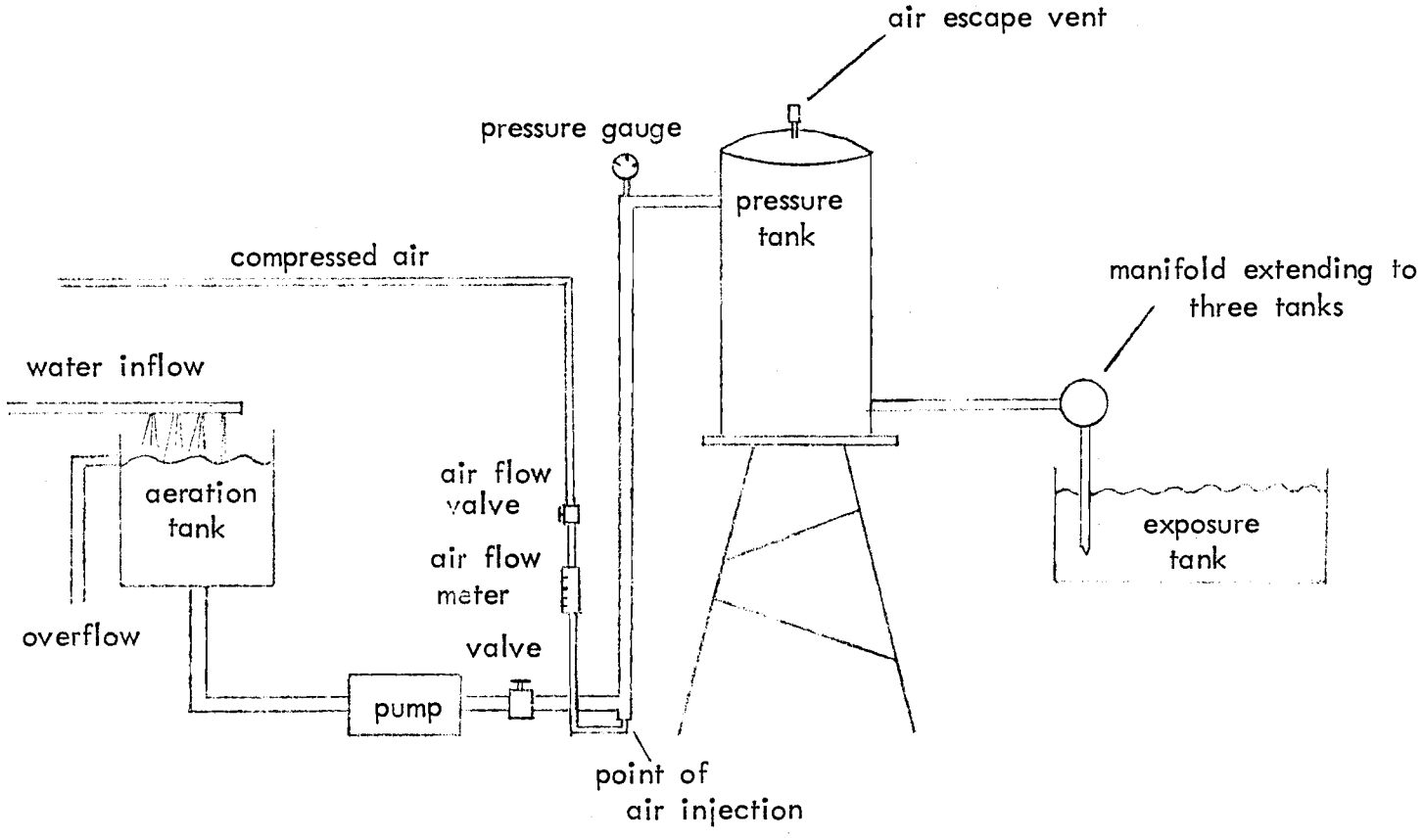

Figure 1. Pump-type supersaturation generator used in Abernathy heritability experiment.

reduce any differences in initial saturation of dissolved gases. A pump was used to create a pressure of 20 lbs/in^2 within the apparatus. In the line from the pump, a controlled amount of air was injected through an air stone inserted at a joint in the line. Aeration under pressure occurred in a 5 ft vertical section of line so that maximum dispersion of air bubbles was maintained. A pressurized tank was used to allow venting of excess air. Water was jetted below surface level into the test tanks. The jet in each tank was aimed directly at the bottom to reduce the flow velocity.

Supersaturation in the test tanks was allowed to stabilize overnight before the test fish were added. The progression of mortality was continually monitored, with fish being removed from the tanks as they died. The identifying mark and time of death of each fish was recorded.

Comparative Bioassays. Experiment 1

To test for genetic differences among stocks of fish for their resistance to gas bubble disease, it is necessary to separate variation arising from genetic and environmental factors. An attempt was made to rear the test fish under constant and identical environmental conditions, so that differences between test fish due to environment would be minimal. With the environments of all stocks

as homogeneous as possible, differences which were found in the bioassays were assumed to have genetic basis.

Test Fish

In the fall of 1972, eggs were taken from mature fall chinook at Abernathy Salmon Cultural Development Center, Little White Salmon Hatchery, and Little Goose Dam, all on the Columbia River drainage system. These stocks represented a wide range in distance of migration up the Columbia River. To reach Little Goose Dam, fish must pass over seven dams and cover approximately 490 miles. Fish returning to Little White Salmon Hatchery must cover 180 miles and pass over one dam. There are no dams below Abernathy Salmon Cultural Development Center, which is about 60 miles from the mouth of the Columbia. For comparison, fall chinook eggs were also obtained from Trask River Salmon Hatchery. The Trask River enters the Pacific Ocean approximately 50 miles south of the Columbia River. It is believed that the Trask River fish have never been exposed to lethal levels of supersaturated water. The eggs from each hatchery were taken from as large a number of random crosses as possible, so that the offspring would be a representative sample of the population. Due to difficulties in obtaining spawning fish at Little Goose Dam, crosses at this location could only be made between two males and two females.

Eggs from each hatchery were fertilized, water-hardened and transported to Oregon State University where they were placed in separate incubator trays. The incubator was supplied with dechlorinated water at a temperature of 9.5 ± 1°C. Egg mortalities during incubation did not differ appreciably between stocks. Eggs hatched in about 75 days and the larvae were free-swimming in an additional 45 days. Each group was allowed the same number of days after hatching before they were fed. The fry were fed with Oregon Moist Pellet (Hublou, 1963) for two months, at the end of which time, they were exposed to 127 percent air supersaturated water. At the time of testing, the fish averaged between 1.3 and 1.5 grams per fish. Due to difficulties in the timing of the bioassays, only fish from Little Goose Dam and the Trask River could be used for comparison.

Test Conditions and Apparatus

Supersaturated water was produced by aerating water under a hydrostatic head of 10 ft (Figure 2). This was accomplished in a 10 ft vertical column of 6 in PVC pipe. A metered amount of air was supplied to the base of the column through four air stones. Two bags made from plastic mesh material were filled with glass marbles and suspended at different heights within the column to minimize mixing of water due to aeration. Water drawn from the bottom of the column and into a fish holding tank was 123 percent

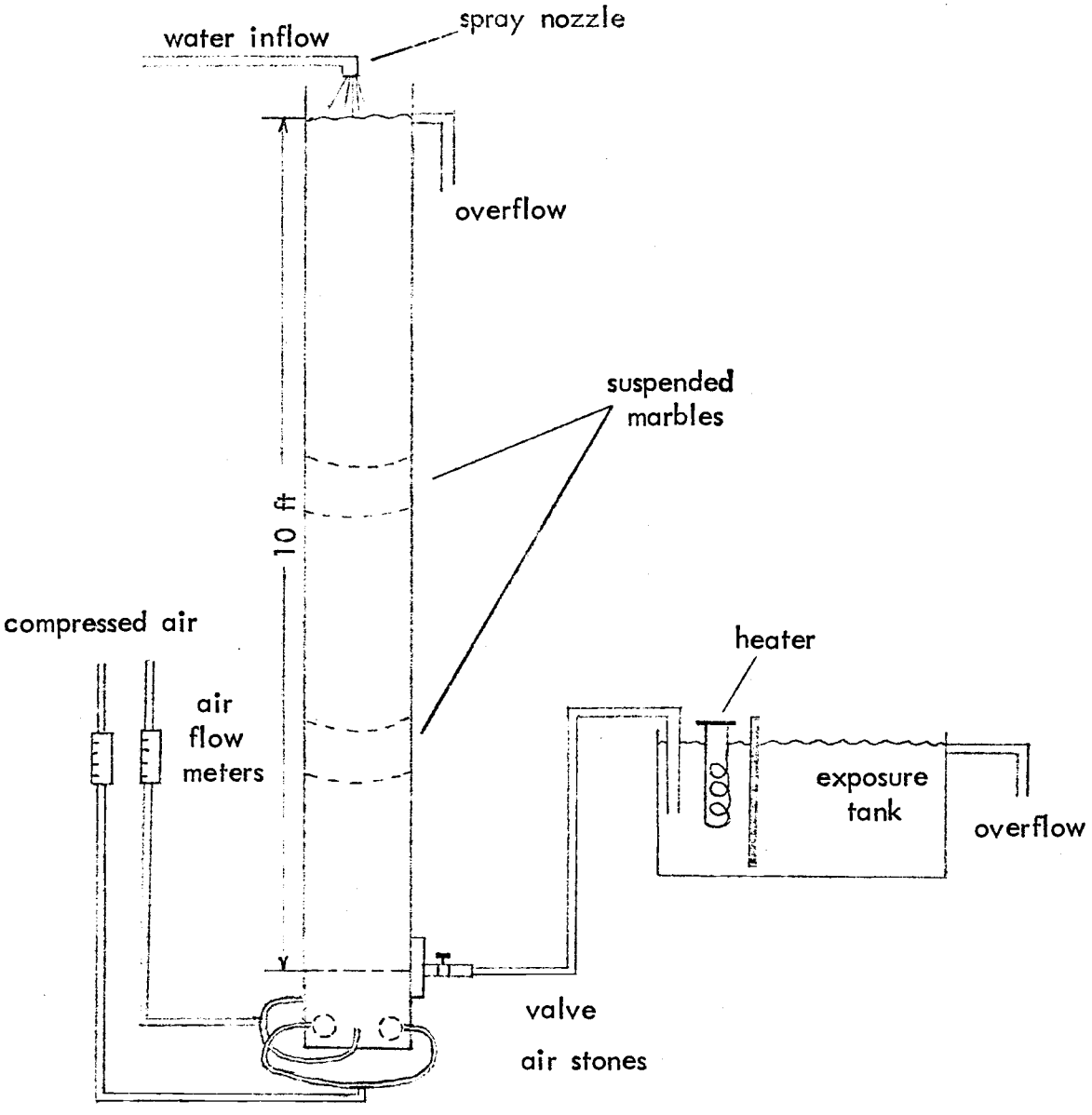

Figure 2. Column-type supersaturation generator used in comparative bioassay experiment 2.

air supersaturated. It was necessary to heat this water an additional two degrees centigrade to 13.5°C, to obtain the test level of 127 ± 2 percent supersaturation.

The test chamber was a 15 gal aquarium partitioned into three sections, one in which the water was heated and two for holding fish. The two fish holding sections were separated by a perforated plastic sheet to allow exchange of water. Dissolved oxygen measurements showed that supersaturation was equal in each section. Thirty fish were placed in each section for each bioassay.

Comparative Bioassays. Experiment 2

Test Fish

In the fall of 1973, fall chinook eggs were obtained in the same manner as 1972 from Abernathy, Little White Salmon, and Kalama hatcheries on the Columbia River and from the coastal Trask River Salmon Hatchery. All eggs from Columbia River hatcheries were taken on October 2, and Trask River eggs were taken on November 28. Unfortunately, eggs could not be obtained again from fish at Little Goose Dam. To accelerate growth, the eggs were incubated at 12.5°C. Eggs hatched in an average of 41 days and feeding began 28 days later.

The fingerlings were reared in a 17 ft x 1 ft x 1 ft plexiglass

tank which was divided into 20 separate sections, each section holding about 5 gal of water. Dechlorinated water was heated in a head box to 13.5° C and delivered to each section at approximately 150 ml per minute. The sections were arbitrarily divided into four blocks of five tanks each, according to their distance from the head box to account for any gradient in environment within the rearing apparatus. Fifty fish from each of the four hatcheries were randomly assigned to one section in each of the four blocks so that the material constituted a randomized block design.

Fish were fed measured amounts of Abernathy dry pellet for 50 days at the end of which time they weighed from 1.0 to 1.7 grams per fish.

Seven days before testing, all fish were marked by cold branding. Because of the small size of the fish, the end of a steel rod of 0.125 in diameter was used to apply a circular or dot mark. Each group of fish was marked with a different combination of dots. These marks darkened and were easily detectable within a few days.

Test Conditions and Apparatus

After 50 days of feeding, the fish were exposed to 127 ± 1 percent air supersaturated water at 11° C. The bioassay took place in a 4 ft x 3 ft x 6 in tank with supersaturated water supplied at a rate of 3 L/min.

Supersaturation was generated by a pump type apparatus similar to that described for the heritability experiment. An aeration tank prior to pressurization was not used with this apparatus, because dissolved gases in the incoming water were always at 100 percent saturation. Pressure was maintained at 9 lbs/in^2 within the apparatus.

Measurement of Dissolved Gases

The concentration of dissolved gases in all tests was monitored at regular intervals by the measurement of dissolved oxygen using the Azide modification of the Winkler method (American Public Health Association, 1965). Oxygen and nitrogen dissolve in water in constant proportions and at nearly equal rates so that dissolved oxygen is a good index of the amount of total dissolved gases (Adeny and Becker, 1920; and Benson and Parker, 1961). The final proportions of dissolved oxygen and nitrogen will differ from equilibrium only if the proportions are not in equilibrium in the intake water of the supersaturation generator.

To check the accuracy of dissolved oxygen as a measure of total dissolved gases, a Weiss saturometer was used to determine the total uncompensated hyperbaric dissolved gas pressure in the test tanks. The saturometer is composed of a gas sensor (250 ft coil of 0.25 in OD 0.012 in ID dimethyl silicone tubing) connected

to a pressure gauge. The sensor is placed under water where pressure in the tubing comes into equilibrium with the uncompensated dissolved gas pressure in the water. Readings of total dissolved gas pressure taken with a saturometer were comparable with the percent supersaturation estimated from levels of dissolved oxygen. In the heritability experiment carried out at Abernathy Salmon Cultural Development Center, total dissolved gases saturation averaged 130 percent, while dissolved oxygen saturation averaged 120 percent.

RESULTS

Heritability Experiment

Fish in the 80 families tested began dying 3 hrs after being placed in 130 percent air supersaturated water, with 100 percent mortality occurring after 132 hrs. The grand means of time of death were 22.62 hrs in tank 1, 24.66 hrs in tank 2, and 25.04 hrs in tank 3. The mean time to death for each tank-family is given in Appendix 1. Only two fish died in the control tank during the course of the experiment.

The number of individuals per tank-family varied unexpectedly from 5 to 54, with most tank-families having from 26 to 30 individuals per family. One hundred and twenty-three fish died after marking and before testing, however, these mortalities were fairly evenly distributed over all tank families. The large discrepancies in the number of individuals in some tank-families may be attributed to three sources of error. In the few groups showing large deviations from 30, it is likely that fish were counted incorrectly at the time of marking. Additional error may have arisen, because the marks of approximately 300 fish which were included in the bioassay could not be identified. These fish may not have been distributed evenly over all families. A small amount of error probably was due to incorrect identification of marks.

Time to Death

The distribution of time to death for fish in each tank appeared to approximate a poisson distribution (Figure 3). To remove the dependence of the variance on the mean, a square root transformation of time to death was applied before the analysis of variance was carried out (see Bartlett, 1947).

Because of the large number of observations and the unequal numbers per tank-family, an unweighted means analysis of variance was used (Kempthorne, 1957). By this method, two separate analyses were done. First, the unweighted means of each family in each tank were subjected to an analysis of variance (Table 1). Second, all observations were used in a one-way analysis of variance between and within tank-families. The within mean square from the second analysis is the true within progenies mean square for the complete analysis.

The mean squares obtained in this analysis are comprised of portions of several variance components (Table 2). Each variance component was estimated by comparison of the appropriate mean squares.

The additive genetic variance, V_A, was estimated as $4\sigma^2_m$ and the total phenotypic variance, V_P, was estimated as:

$$V_P = \sigma^2_m + \sigma^2_f + \sigma^2_{mr} + \sigma^2_{fr} + \sigma^2_w$$

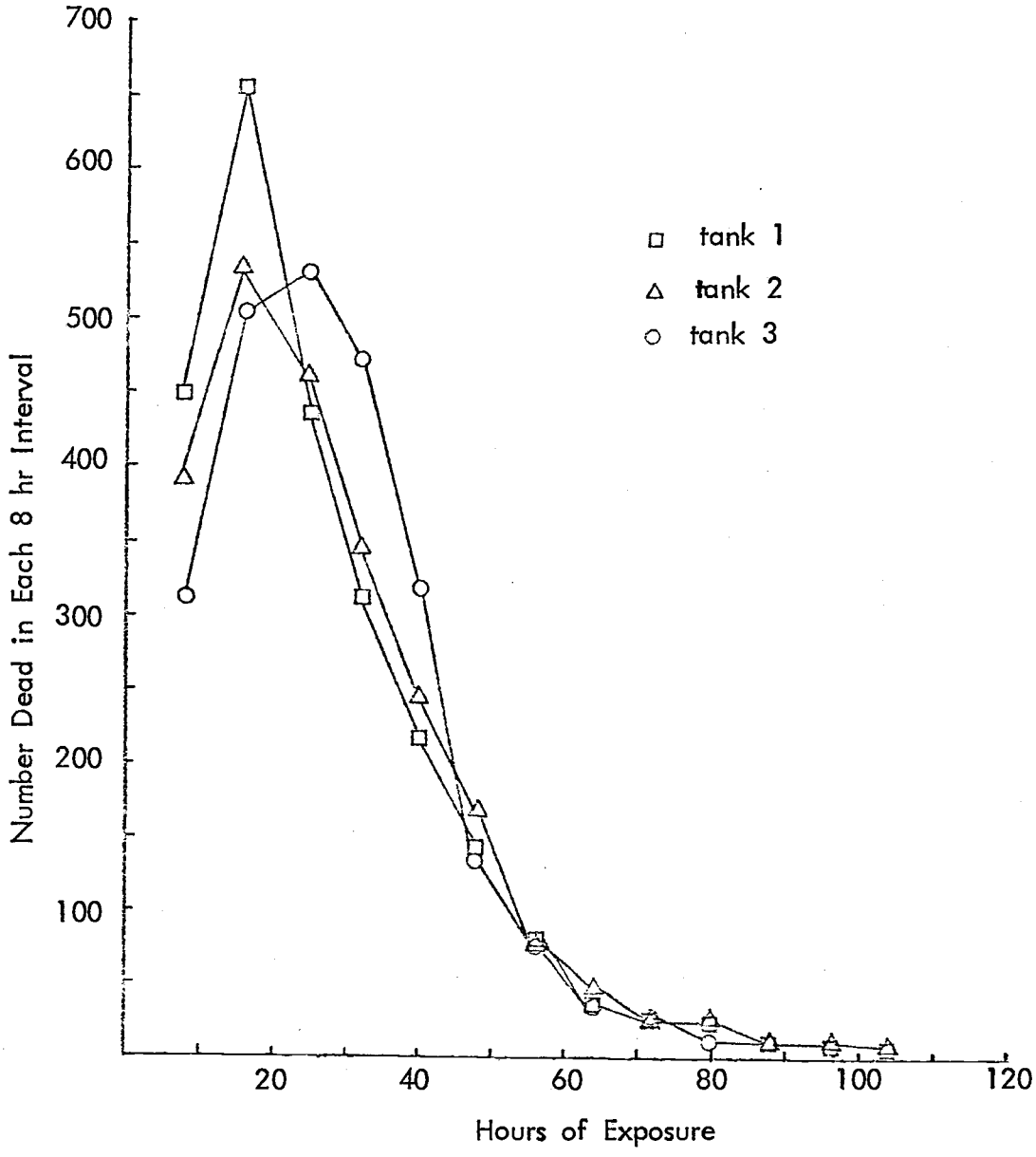

Figure 3. Mortality frequency distributions of juvenile fall chinook salmon exposed to 130 percent air supersaturated water in each of three test tanks (heritability experiment).

Table 1. Analysis of variance of the square root of time to death for juvenile fall chinook salmon exposed to air supersaturated water.

Source of Variation	Degrees of Freedom	Sums of Squares	Mean Squares	F
Tanks	2	3.768		
Males	19	10.925	0.575	2.30**
Females/Males	60	15.015	0.250	1.34
Males x Tanks	38	7.074	0.186	1.62
Females/Males x Tanks	120	13.809	0.115	1.11
Error[a/]	6566		2.800	

$\overline{n}_h$ = 26.820

a/ Error mean square obtained in separate analysis.

** Statistical significance at the 0.01 level.

Table 2. Expected mean squares for the nested analysis of variance of means and computational formulas for the variance components.

Source of Variation	Degrees of Freedom	Mean Square	Expected Mean Squares
Replicates	r-1	MS_R	
Males	m-1	MS_M	$(1/\bar{n}_h)\,\sigma^2_w + \sigma^2_{fr} + f\sigma^2_{mr} + r\sigma^2_f + rf\sigma^2_m$
Females/Males	m(f-1)	MS_F	$(1/\bar{n}_h)\,\sigma^2_w + \sigma^2_{fr} + f\sigma^2_{mr} + r\sigma^2_f$
Males x Replicates	(r-1)(m-1)	MS_{MR}	$(1/\bar{n}_h)\,\sigma^2_w + \sigma^2_{fr} + f\sigma^2_{mr}$
Females/Males x Replicates	s(r-1)(f-1)	MS_{FR}	$(1/\bar{n}_h)\,\sigma^2_w + \sigma^2_{fr}$
Error[a/]	N... - mfr	MS_W	σ^2_w

where:
- m = number of males
- f = number of females
- r = number of replicates
- N... = total number of individuals
- $\bar{n}_h$ = harmonic mean number of observations per tank-family

Variance components were estimated as:

$\sigma^2_m = (MS_M - MS_F)/rd = 0.027$

$\sigma^2_f = (MS_F - MS_{MR})/r = 0.021$

$\sigma^2_{mr} = (MS_{MR} - MS_{FR})/f = 0.018$

$\sigma^2_{fr} = (MS_{FR} - (1/\bar{n}_h)(MS_W) = 0.011$

$\sigma^2_w = MS_W = 2.800$

[a/] Error term calculated in separate analysis.

(Falconer, 1960). Heritability (h^2), or the ratio of V_A to V_P, was:

$$h^2 = \frac{4(0.027)}{2.877} = 0.038$$

with standard error, S.E. (h^2) = 0.022 (Dickerson, 1959).

Percentage Survival

The proportion of fish surviving after 36 hrs was determined. The range of survivors after 36 hrs was 53.8 percent to 0 survivors. The proportions surviving after 36 hrs in each tank-family are listed in Appendix 2.

To remove the dependence of the variance on the mean in these binomial data, the percentage survival in each group was transformed to the arcsin of its square root. With this transformation, the variance, σ_b^2, is stabilized, and is approximately equal to 821/n, where n is the number of observations in each proportion (Snedecor and Cochran, 1967). The analysis of variance of the transformed proportion of survivors after 36 hrs is presented in Table 3. This analysis shows variation between males to be highly significant. None of the other mean squares were significant.

The expected mean squares for this analysis are the same as those presented in Table 2. An error sum of squares cannot be calculated, because there is only one observation on each

Table 3. Analysis of variance of the transformed proportion of families of juvenile chinook salmon surviving after 36 hrs (heritability experiment).

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Tanks	2	336.34		
Males	19	2782.23	146.43	2.69**
Females/Males	60	3265.27	54.42	1.07
Males x Tanks	38	1927.63	50.73	1.55
Females/Males x Tanks	120	3919.62	32.66	1.07

$\bar{n}_h = 26.82$ $\sigma^2_m = 7.67$ $\sigma^2_f = 1.23$ $\sigma^2_{mr} = 4.52$[a/] $\sigma^2_{fr} = 2.05$

$\sigma^2_b = 821$

[a/] σ^2_{mr} obtained by using $(1/\bar{n}_n)$ 821 as an estimate of $(1/\bar{n}_h)\ \sigma^2_w$.

**Statistical significance at the 0.01 level.

tank-family. There are two possible methods by which the value of σ^2_w can be estimated.

First, use of the arcsin square root transformation causes binomial variance to become stable and approximately equal to 821/n. The binomial variance for individuals, then, is 821. This value was used to estimate the error variance, σ^2_w. Error variance is composed of the binomial variation plus any extraneous variation (Cochran, 1943). The extraneous error in this case would be composed of environmental and genetic variation. Variation in the data from which the above analysis was made appeared to be largely binomial in nature, so it was assumed that extraneous variation was small in proportion to the binomial variation. To estimate σ^2_{fr}, $821/\overline{n}_h$ was subtracted from MS_{FR}. By substituting 821 for the value of σ^2_w, the heritability of the proportion surviving after 36 hrs was estimated as:

$$h^2 = \frac{4(\sigma^2_m)}{\sigma^2_m + \sigma^2_f + \sigma^2_{mr} + \sigma^2_{fr} + 821} = 0.037 \pm 0.019$$

The 95 percent confidence interval for this heritability is 0 to 0.075.

A second method of estimating σ^2_w is to assume that interaction of females within males by tanks, σ^2_{fr}, is negligible so that the mean square for this interaction, MS_{FR}, can be used as an estimate of error variance. This method may overestimate error

variance. The value of MS_{FR} was obtained in a means analysis, so this value must be multiplied by the harmonic mean number of individuals per cell, 26.82, to obtain the error variance for individuals. Heritability can then be estimated as,

$$h^2 = \frac{4'(\sigma^2_m)}{\sigma^2_m + \sigma^2_f + \sigma^2_{mr} + \bar{n}_h (MS_{FR})} = 0.035 \pm 0.019$$

The similarity of the heritabilities estimated indicates that use of either estimate of error variance is acceptable.

Comparative Bioassays

Experiment 1

Cumulative mortality curves for fall chinook from Little Goose Dam and Trask River Salmon Hatchery exposed to 127 percent supersaturation were quite different (Figure 4). Time to 50 percent mortality, averaged from two replicates, was 73.5 hrs for Trask River fish compared to 154 hrs for fish from Little Goose Dam, producing a highly significant difference of 80.5 ± 3.39 hrs (Table 4).

Table 4. Analysis of variance in time to 50 percent mortality for Trask and Snake River chinook salmon (Experiment 1).

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Total	3	6503		
Between stocks	1	6480	648	56.35**
Error	2	23	11.5	

**Statistical significance at the 0.01 level.

Experiment 2

Data recorded were time to 50 percent mortality, proportion dead after 96 hrs, and the proportion dead after 150 hrs (Table 5).

In all comparisons, Abernathy fish were most resistant and Trask fish were least resistant. Cumulative mortality curves for all Columbia River fish combined and Trask River fish are presented in Figure 5. Columbia River fish were combined because mortality curves for the separate Columbia River stocks could not be clearly separated.

Differences in time to 50 percent mortality between stocks were significant (Table 6). The between stocks sum of squares was partitioned into two components to compare stocks within the Columbia River. The difference in time to 50 percent mortality between Columbia River and Trask River stocks (22.25 ± 6.37 hrs) was significant.

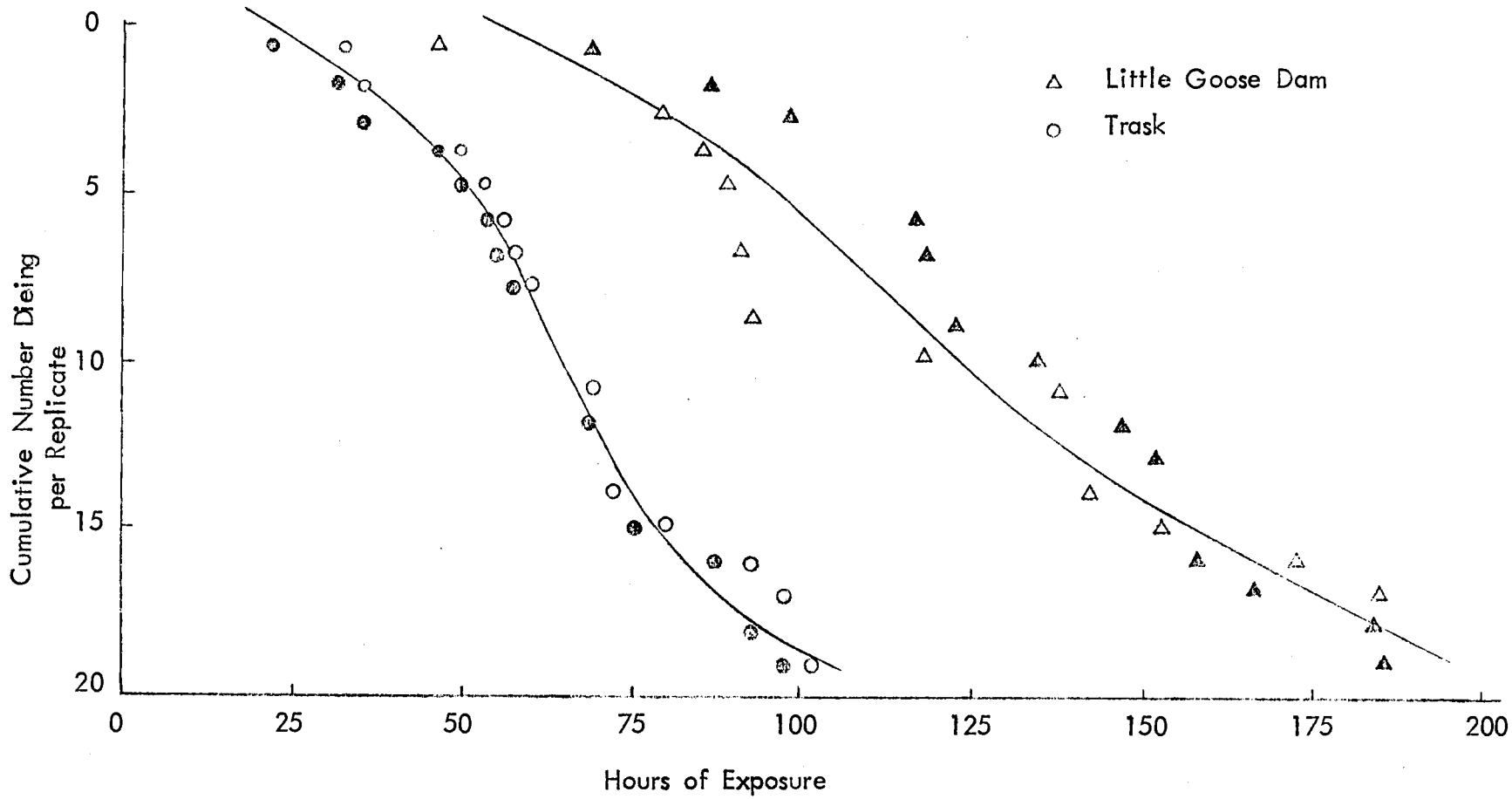

Figure 4. Cumulative mortality of juvenile fall chinook from Little Goose Dam and Trask River exposed to 127 percent air supersaturated water (Experiment 1). **Closed and open symbols represent values of replicates.**

Table 5. Comparative bioassay data for juvenile chinook salmon exposed to air supersaturated water in Experiment 2.

Stock	Tank	No.	$\bar{w}$	ET50	ND96	P96	ND150	P150
Abernathy	1	25	1.62	70	16	.64	20	.80
	2	31	1.56	76	18	.58	21	.68
	3	30	1.54	116	13	.43	19	.63
	4	30	.65	108	14	.47	19	.63
Little White Salmon	1	28	1.74	82	16	.57	21	.75
	2	30	1.39	88	17	.57	23	.77
	3	30	1.31	82	20	.67	24	.80
	4	30	1.24	94	16	.63	23	.77
Kalama	1	28	1.55	73	21	.70	25	.83
	2	30	1.06	64	19	.63	28	.93
	3	19	1.19	79	12	.63	18	.95
	4	30	.95	79	18	.60	22	.73
Trask	1	22	1.00	48	14	.64	21	.96
	2	30	1.00	65	21	.70	25	.83
	3	27	1.00	60	22	.82	25	.93
	4	28	1.10	75	18	.64	21	.75

Notation:

$\bar{w}$ = average weight
ET50 = elapsed time to 50 percent mortality (hours)
ND96 = number dead after 96 hours
P96 = proportion dead after 96 hours
ND150 = number dead after 150 hours
P150 = proportion dead after 150 hours

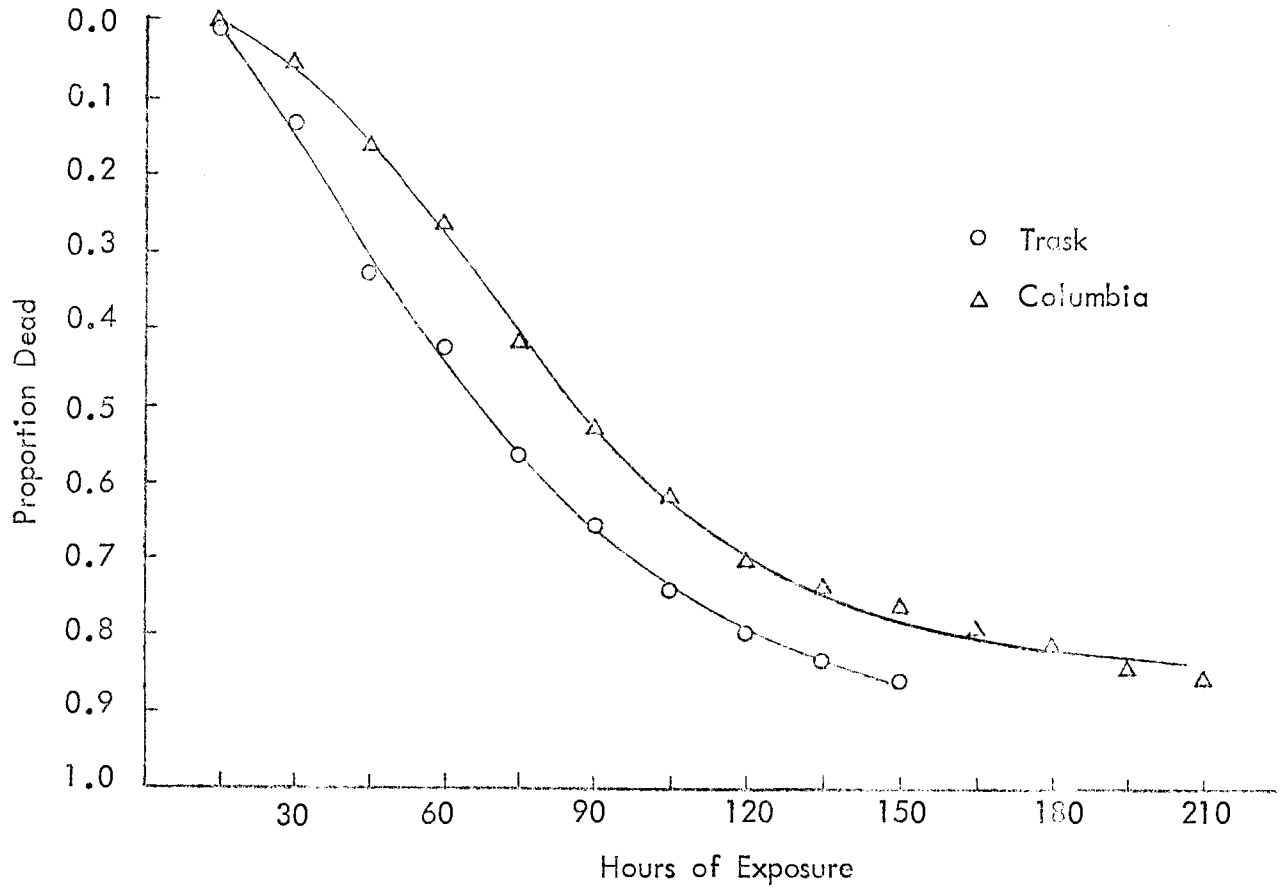

Figure 5. Cumulative mortality of Trask River and combined Columbia River juvenile fall chinook salmon exposed to 127 percent air supersaturated water (Experiment 2).

Table 6. Analysis of variance in time to 50 percent mortality for Trask and Columbia River chinook salmon (Experiment 2).

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Squares	F
Total	15	4417.4		
Blocks	3	1103.2	367.7	3.02
Between Stocks	3	2218.4	739.5	6.07*
Columbia vs. Trask	1	1485.2	1485.2	12.19**
Within Columbia	2	733.2	366.6	3.01
Error	9	1095.8	121.8	

*Statistical significance at the 0.05 level.

**Statistical significance at the 0.01 level.

After 150 hrs of exposure, 80 percent of all fish were dead. The proportion dead in each group of 30 fish varied from 63 to 96 percent. This variation made it necessary to stabilize the variation with the arcsin square root transformation.

Variation in the proportion dead after 150 hrs of exposure was also significant (Table 7). Partitioning of the treatment sum of squares had no effect on the significance of the results. Only in the comparisons of this analysis was variation between Columbia River stocks significant. The Abernathy stock was the most resistant, so comparisons were made between the Abernathy stock and the other Columbia River stocks. Only the difference between Abernathy and

Kalama, 13.0, was significant. The difference between Trask and Abernathy stocks, 13.7, was also significant.

Table 7. Analysis of variance in the transformed proportion dead after 150 hrs for Trask and Columbia River chinook salmon (Experiment 2).

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Squares	F
Total	15	1026.0		
Blocks	3	205.7	68.6	2.01
Between Stocks	3	513.1	171.0	5.01*
Columbia vs. Trask	1	171.0	171.0	5.01*
Within Columbia	2	342.0	171.0	5.01*
Error	9	307.3	34.1	

Least significant difference = 7.29

*Statistical significance at the 0.05 level.

DISCUSSION

Significance of Results

The estimates of heritabilities and of stock differences in resistance to gas bubble disease were made in this study from bioassays of juvenile fall chinook. It will be assumed for the purposes of discussion that results of this study are also valid for fall chinook smolts.

Much of the variability in the resistance of juvenile chinook salmon to air supersaturated water was random, and therefore gave relatively large values of error variance (σ^2_w). In spite of the large amount of variation within progenies, the effect of males in the heritability experiment was found to be highly significant, indicating that additive genetic factors influence resistance to gas bubble disease. This means that selection will cause an increase in the average resistance of fall chinook salmon to gas bubble disease.

The value estimated for the female effect (σ^2_f) was low in comparison to the male effect (σ^2_m) in the analysis of data from the mating experiment. Several unexplained large deviations in the proportion surviving after 36 hrs were recorded for single families in different tanks. These deviations would tend to inflate σ^2_{mr}, σ^2_{fr}, and σ^2_w; thereby reducing σ^2_f. This, in turn, implies the

heritabilities are underestimated; however, the amount of this underestimation would be small.

The highly significant differences in resistance to gas bubble disease found between the stocks of fall chinook salmon tested indicates that resistance to gas bubble disease is a heritable trait, and that selection for more resistant fish to gas bubble disease has occurred. These conclusions are supported by the bioassay data in that the graded levels of resistance to air supersaturation for each stock corresponded with their history of exposure to air supersaturation.

Variation in resistance to gas bubble disease between Abernathy, Kalama, and Little White Salmon stocks of fall chinook was not significant, suggesting that the smolts released from these hatcheries have been equally exposed to lethal levels of air supersaturated water. This conclusion cannot be made, however, because Abernathy Creek brood stock were originally taken from Spring Creek and Willard Hatcheries, both above Bonneville Dam, in 1961 and 1962 (personal communication with Laurie Fowler, fishery research biologist, Abernathy Salmon Cultural Development Center).

One would expect that fall chinook from the Snake River above Little Goose Dam, which migrate through at least 490 miles of air supersaturated water would have greater resistance to gas bubble disease than fall chinook stocks from the lower Columbia. Any

conclusions regarding this point are difficult to draw from the comparative bioassays, because rearing and testing conditions in Experiment 1 and Experiment 2 were somewhat different. The much greater difference in time to 50 percent mortality between fish taken from Little Goose Dam and fish from the Trask River (80.5 ± 3.39 hrs) does indicate, however, that fall chinook migrating as far as Little Goose Dam are more resistant to gas bubble disease than lower Columbia River stocks. This comparison may be valid, because the time to 50 percent mortality for Trask River fish in the two Experiments was similar (73.5 hrs in Experiment 1 and 62 hrs in Experiment 2).

Conditions on the Columbia River have changed considerably since gas bubble disease mortality began. For this reason, a precise estimate of the level of selection required to have caused the observed differences between stocks in their resistance to gas bubble disease cannot be obtained. A rough answer to this question can be obtained, however, if several simplifying assumptions are made. It will first be assumed that resistance to gas bubble disease in all Columbia River fall chinook was originally equal to the observed resistance of Trask River fall chinook. The beginning of annual periods of air supersaturation in the Columbia River will be taken as 1938 when Bonneville Dam was completed. It will be assumed further that an average proportion of the smolts from each

generation since that time have died from gas bubble disease, and that the average generation time is 4 yrs. This means that nine generations have been subjected to selection for resistance to gas bubble disease. Response to selection is given by the equation (Falconer, 1960):

$$R = h^2 (z/p)\sigma_p$$

where R = response to selection, h^2 = heritability, z = height of the ordinate on the normal distribution curve at proportion p (proportion selected) on the abcissa, and σ_p = phenotypic standard deviation. Values were then assigned to the variables in this equation so that p could be calculated. The increase in resistance to gas bubble disease of lower Columbia River fall chinook was taken from the difference in the proportion dead after 96 hrs between lower Columbia River stocks and the Trask River stock as 0.2. The value of heritability is 0.037 and the phenotypic standard deviation is 0.24. These values were obtained from the heritability experiment analysis of the proportion surviving after 36 hrs. The above equation can now be written:

$$R = 0.2 = ((0.037)\ (0.24)z/p) \times 9$$

The resulting of z/p is 2.57. This corresponds to 98.5 percent mortality due to gas bubble disease each generation. This level of

mortality is obviously too high. This finding suggests that 0.037 may be a low estimate of heritability.

The above calculations were repeated using the upper limit of the 95 percent confidence interval of heritability, 0.075. The value of z/p was calculated as 1.234. This corresponds to an average gas bubble disease mortality of 73.5 percent each generation. This value is only an average, because mortality was initially higher and decreased by 20 percent. As can be seen in Figure 6, the values of z/p have a fairly linear relationship to the proportion selected over the range of 0.55 to 0.75, so the above calculations can be assumed to mean that the proportion selected began at 83 percent and has decreased to 63 percent.

Estimates of actual gas bubble disease mortality of fall chinook smolts below Bonneville Dam were obtained from unpublished data collected by Howard Raymond and Carl Sims of the National Marine Service. Raymond and Sims marked and released fall chinook smolts at Spring Creek, Little White Salmon, Oxbow, and Bonneville hatcheries. Additional smolts from each hatchery were marked and released near Rainier, a point approximately 90 miles below Bonneville Dam. Recapture efforts were then made near the mouth of the Columbia, and mortalities were compared between fish released from the hatchery and fish released at Rainier. Fish released at the hatcheries in June, when supersaturation was high,

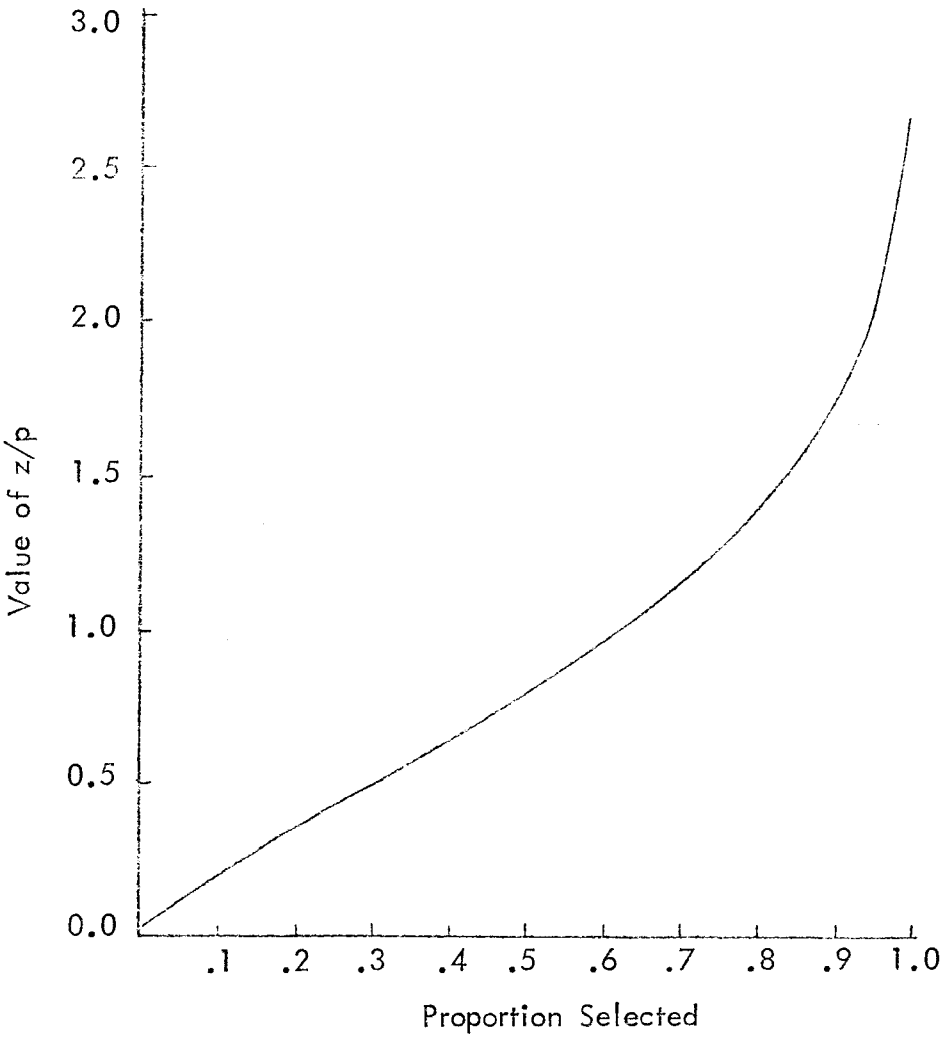

Figure 6. Relationship of selection intensity (z/p) with different proportions selected.

averaged 40-60 percent survival to Rainier. Smolts from a single release at Spring Creek Hatchery in April, when supersaturation was not present, showed a survival of 91 percent to Rainier. This data suggests that smolt mortality from gas bubble disease between Bonneville Dam and Rainier is between 30 and 50 percent. This mortality would undoubtedly be higher for the full distance from the hatchery to the ocean. Still, the true gas bubble disease mortality rates are probably lower than those calculated as necessary to explain the difference in gas bubble disease resistance between lower Columbia River stocks and the Trask River stock.

The above conclusion is again reached if the same type of calculations are made for fall chinook from Little Goose Dam. The results from Experiment 1 indicate that the change in proportion surviving a set level of exposure to supersaturation has been about 0.4. This change would require an average selection intensity of 2.469 which is equivalent to mortalities of 98 percent. This level of gas bubble disease mortality is within the upper range of recent mark-recapture estimates made on Snake River fall chinook by Raymond (personal communication). However, this level of mortality has not existed since 1938 because most of the dams between Bonneville and Little Goose have been constructed since 1960. This fact implies that selection for gas bubble disease resistance has probably increased sharply in recent years on Snake River fall

chinook smolts. Again it must be concluded that a heritability of 0.075 is not high enough to explain the difference in resistance to gas bubble disease that was found between fish from Little Goose Dam and fish from the Trask River.

The differences which were found between stocks of fall chinook have at least two possible explanations. It is possible that fish from the Columbia River drainage system were inherently more resistant to gas bubble disease than fish from the Trask River before gas bubble disease mortalities ever occurred. Secondly, it is possible that the amount of additive genetic variability which originally influenced resistance to gas bubble disease was caused by a small number of gene loci, so that selection has caused a significant reduction in that variability. This would mean that heritability, and therefore response to selection, is now less than before these fish were subjected to selection for gas bubble disease resistance. There may also have been some dominance or epistatic genetic variance before selection began, which has now been nearly eliminated.

Population Modeling of the Future Effects of Increasing Resistance to Gas Bubble Disease

To determine the effects of selection for resistance to gas bubble diseaseon fall chinook, it was necessary to develop a model

which would predict, (1) the number of fish returning to the hatchery each year, and (2) the expected average resistance of the offspring of these fish. Such a model required the input of age specific mortality and maturity rates. Because these rates are likely to differ for fish returning to different sections of the Columbia River, the model was restricted to the Kalama River stock of fall chinook. The values used in this study were obtained by averaging the values obtained by Cleaver (1969) for the 1961 brood year and Henry (1971) for the 1962 brood year when the instantaneous rate of natural ocean mortality was assumed to be 0.45 (Table 8).

Table 8. Mortality and maturity schedules of Kalama River fall chinook used in the population model.

	Year of life			
	2	3	4	5
Instantaneous natural ocean mortality	--	0.45	0.45	0.45
Instantaneous ocean fishing mortality	--	0.3	0.62	0.8
Proportion maturing	0.006	0.04	0.57	1.0
Instantaneous river fishing mortality	0.69	1.2	1.2	1.2

The population model was developed to follow the line of events occurring in the life of a Columbia River fall chinook salmon, with mortality being an exponential function (Figure 7). The model begins with the spawners returning to the hatchery in 1965. The number of

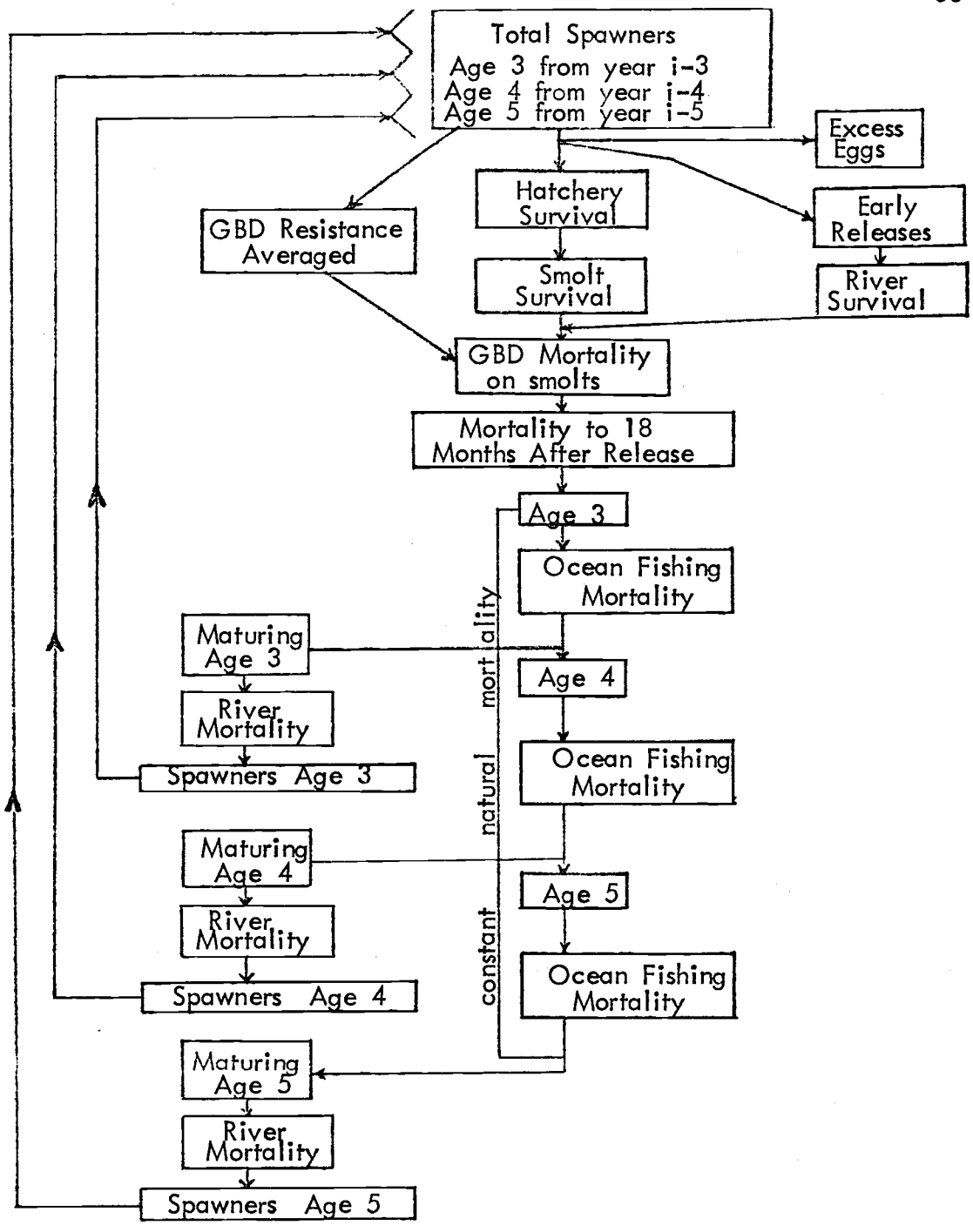

Figure 7. Flow diagram of Columbia River fall chinook salmon population model. Gas bubble disease is denoted by GBD.

returning spawners in each age group to Kalama River Hatchery from 1965 to 1969 are given in Table 9. Because of the controlled environmental conditions found within a hatchery, the fecundity of the fish can be expressed as the average number of smolts produced per spawner. John Clayton, superintendent of the Kalama River Hatchery, has found that the average number of eggs per female is 4, 800, and that the average survival of eggs to smolts is 85 percent (personal communication). The average proportion of females among the spawners, determined from 5 yrs of Kalama Hatchery data presented in the Data Reports of the U. S. Bureau of Commercial Fisheries' Columbia River Hatchery Contribution Study (1965; 1966; 1967; 1968; and, 1969) was 55 percent. The average number of smolts produced per spawner is then 2, 244.

Table 9. Mature fall chinook salmon returning to Kalama Hatchery from 1965 to 1969.

	Year of spawning				
Age	1965	1966	1967	1968	1969
5	126	498	181	216	614
4	2679	1016	2218	1087	3116
3	375	1379	143	361	916
Total	3180	2893	2542	1664	4646

The capacity of the hatchery places a ceiling on the total possible number of smolts produced. John Clayton reports that Kalama Hatchery can hold 11 million eggs and has a rearing capacity of eight million fry (personal communication). This amount of fertilized eggs is produced by 4,167 spawners. Fry in excess of eight million are assumed to be released into the stream adjacent to the hatchery. Survival to the age of smolting for fry released at this age has been estimated at one percent for fall chinook from Spring Creek Hatchery on the Columbia River (Junge and Phinney, 1963).

Smolts produced in the hatchery are released in June at six months of age. The smolts are exposed to a high rate of mortality during their downstream migration and during their first year of life in the ocean. Cleaver (1969) estimated the proportion surviving in the first 18 months after liberation at 0.011 for 1961 brood fall chinook, while Henry (1971) estimated survival over the same period to be 0.003 for the 1962 brood. The value assumed in this study was 0.012, because lower values resulted in continually declining population numbers. The model was run on a computer using values of 0.3, 0.5, and 0.7 for the proportion dying due to gas bubble disease. This mortality was assumed to be a part of the 98.8 percent mortality suffered in the first 18 months after liberation. It was therefore necessary to use different values for natural smolt mortality for each value of gas bubble disease mortality (Table 10).

Table 10. Combinations of the proportion of fall chinook salmon smolts surviving gas bubble disease and the instantaneous rate of natural smolt mortality used in the population model.

Proportion surviving gas bubble disease	0.3	0.5	0.7
Instantaneous rate of natural smolt mortality	3.219	3.730	4.075

Eighteen months after liberation, the fish conclude their second year of life. At this time, a proportion will mature and enter the river, so that in the third year, a proportionately decreased number of fish remain at sea.

During the third year of life, fish are subjected to substantial fishing mortality, and mortality due to natural causes. Natural mortality is assumed to remain constant throughout the remaining years of life at sea. At the end of the third year, a predictable proportion again mature and enter the Columbia River. These fish are also permanently removed from the ocean. The remaining fish follow a similar pattern of mortality and maturity in their fourth and fifth years of life. At the end of the fifth year of life, all remaining fish are assumed to spawn.

When fish enter the river as maturing fish, a large portion are caught in the river fishery. Those not caught are assumed to spawn. The proportion of Kalama River fish caught each year was averaged by age-group over four years of data presented in the reports of the economic contributions of Columbia River hatcheries for the 1961,

1962, 1963, and 1964 brood years (Worlund et al., 1969; Rose and Arp, 1970; Arp et al., 1971; and Wahle et al., 1972). The instantaneous rates of river mortality due to fishing determined from these proportions are given in Table 8.

Fish reaching the hatchery and being spawned each year will be made up of three age-groups, 3, 4, and 5 year olds. Each age group of fish will pass to its offspring a different inherent resistance to gas bubble disease, depending on the average resistance that age-group inherited from its parents and the increase in that average subsequent to selection on smolts. The average resistance of the smolts produced by the current set of spawners from all age-groups must then be computed as the sum of the weighted averages of each of the parent groups. This introduces the important assumption that there is complete mixing of the spawn of all age-groups.

Modeling of the population can now take place in mathematical terms. The letters and subscripts used are defined as follows:

SP_{ij} = total spawners at year i in their j^{th} year of life

TSP_i = total spawners of all ages at year i

S_i = total smolts produced in year i

GBD_i = proportion surviving gas bubble disease in year i

S/SP = average number of smolts per spawner

R_i = recruits (fish surviving 18 months after liberation)

SM = instantaneous natural mortality for smolts up to

18 months after release

P_j = proportion maturing at end of j^{th} year of life

E_{ij} = number of fish entering the river at year i in j^{th} year of life

M = instantaneous natural mortality in ocean during years 3, 4, and 5

F = instantaneous ocean fishing mortality

RF = instantaneous river fishing mortality

RM = instantaneous river natural mortality

C_{ij} = ocean catch during year i of fish in their j^{th} year of life

RC_{ij} = river catch during year i of fish in their j^{th} year of life

The model proceeds as follows:

$$S_i = TSP_i \times S/SP$$

$$SS_i = S_i \times GBD_i$$

$$R_i = SS_i \times e^{-(SM)}$$

$$E_{i2} = P_2 \times R_i$$

$$E_{i3} = R_i \times (1-P_2) \times e^{-(F_3 + M)} \times P_3$$

$$E_{i4} = R_i \times (1-P_2) \times (1-P_3) \times e^{-(F_3 + F_4 + 2M)} \times P4$$

$$E_{i5} = R_i \times (1-P_2) \times (1-P_3) \times (1-P_4) \times e^{-(F_3 + F_4 + F_5 + 3M)}$$

$$SP_{ij} = E_{ij} \times e^{-(RF_j + RM)}$$

$$TSP_{i+1} = SP_{i-2,3} + SP_{i-3,4} + SP_{i-4,5}$$

$$C_{i3} = R_{i-3} \times (1-p_2) \times F_3/(F_3 + M) \times (1-e^{-(F_3 + M)})$$

$$C_{i4} = R_{i-4} \times (1-P_2) \times (1-P_3) \times e^{-(F_3 + M)} \times F_4/(F_4 + M) \times (1-e^{-(F_4 + M)})$$

$$C_{i5} = R_{i=5} \times (1-P_2) \times (1-P_3) \times (1-P_4) \times e^{-(F_3 + F_4 + 2M)} \times F_5/(F_5 + M) \times (1-3^{-(F_5 + M)})$$

$$RC_{ij} = E_{i-j,j} \times RF_j/(RF_j + RM) \times (1-e^{-(RF_j + RM)})$$

$$GBD_{i+1} = \frac{SP_{i-2,3}}{TSP_{i+1}} \times GBD_{i-2} + \frac{SP_{i-3,4}}{TSP_{i+1}} \times GBD_{i-3} + \frac{SP_{i-4,5}}{TSP_{i+1}} \times GBD_{i-4}$$

With the beginning of each new cycle the proportion of fish surviving gas bubble disease (GBD_i), will be slightly increased due to selection. This response to selection can be calculated by the following equation.

$$\text{Response} = h^2 \times (s/p) \times \sigma_p$$

where h^2 = heritability

σ_p = phenotypic standard deviation

z = height of ordinate of the normal curve at proportion p

p = proportion selected

Response = difference between mean of original population and mean of the offspring of the selected group.

In this model the mean and phenotypic standard deviation are expressed as percentages of fish surviving gas bubble disease. This causes the response to selection to be given as the additional proportion that will survive in the following generation. The ability of the offspring of the selected fish to survive gas bubble disease will then be $GBD_{i+1} = GBD_i + \text{Response}$.

If it is assumed that fishing mortalities and natural mortalities will not change over a period of time, this model can be used to predict future catches and escapements, and the future proportion of Columbia River fall chinook dieing from gas bubble disease. The assumption that mortalities will remain constant is only within reason for a short span of time, so computer runs using the model were limited to 30 years, beginning in 1965 and ending in 1995. The Fortran program used to run this model in the computer is given in Appendix 3.

The effects of the different combinations of heritability and initial selection intensity on the return of spawners to the hatchery after 30 years are shown in Figure 8. It should be noted that the model began with 3,180 spawners. It can be seen that only when the initial proportion dieing from gas bubble disease was set at 50 percent and heritability was 0.060 or greater did the run reach the

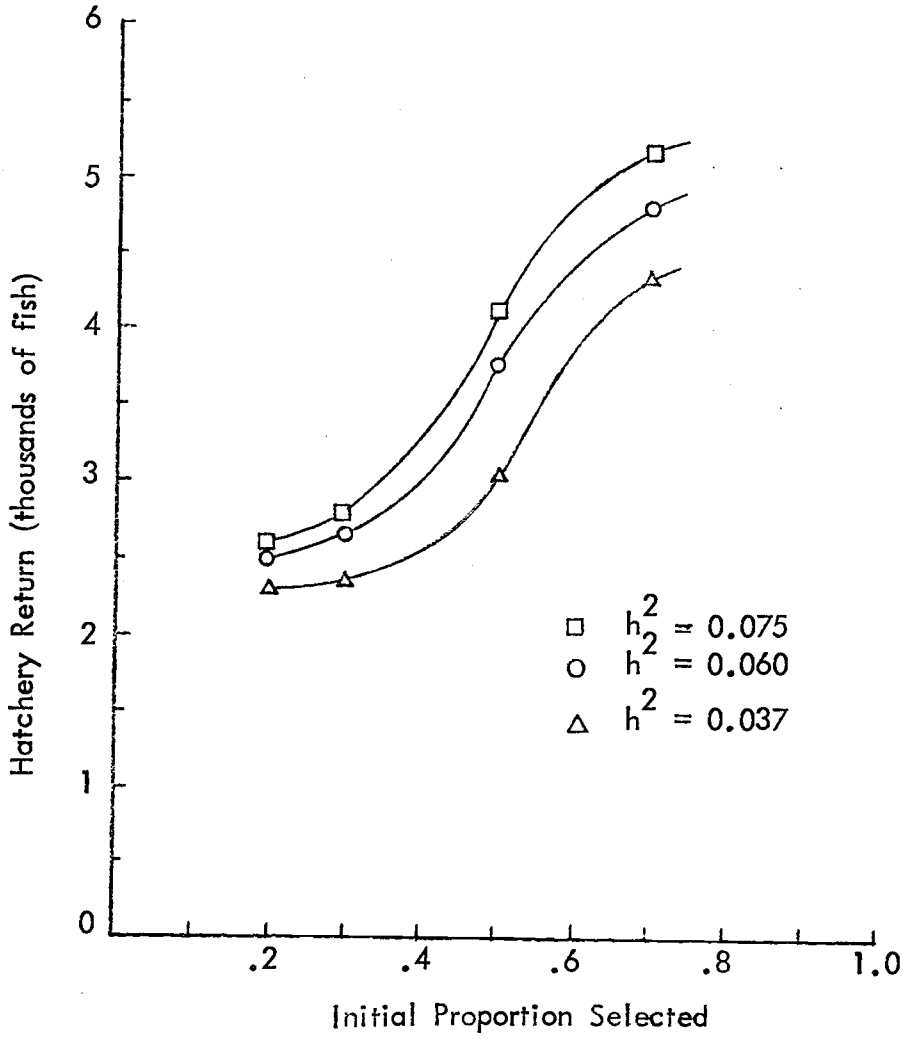

Figure 8. Relationship between heritability, initial proportion selected, and expected return of fall chinook salmon to Kalama Hatchery after 30 years.

numbers at which it started. The computer runs always caused the number of spawners returning to the hatchery to drop and stabilize around 2500 within the first 10 years. Following this period, the increased survival, due to the response to selection for resistance to gas bubble disease caused the returns to gradually increase. This increase is reflected in the ocean catch as shown in Figure 9.

The increases in the proportions surviving gas bubble disease at different values of heritability and initial proportion surviving gas bubble disease are given in Figure 10. These figures show that the proportion surviving gas bubble disease after 30 years of selection cannot be expected to increase greatly. The maximum increase in survival of gas bubble disease was from 0.3 to 0.45 when heritability was set at 0.075. This is a significant increase in survival, but sizable mortalities due to gas bubble disease will still occur. The computer runs which were probably most representative of conditions for Kalama Hatchery fish were those in which initial survival of gas bubble disease was 0.7. In these runs, survival increases ranged from 0.033 when $h^2 = 0.037$ to 0.065 when $h^2 = 0.075$.

The above values stress the extreme importance of current efforts to reduce levels of air supersaturation in the Columbia River. Genetic changes will not greatly reduce gas bubble disease mortality, so it is up to man to reduce the level of air supersaturation.

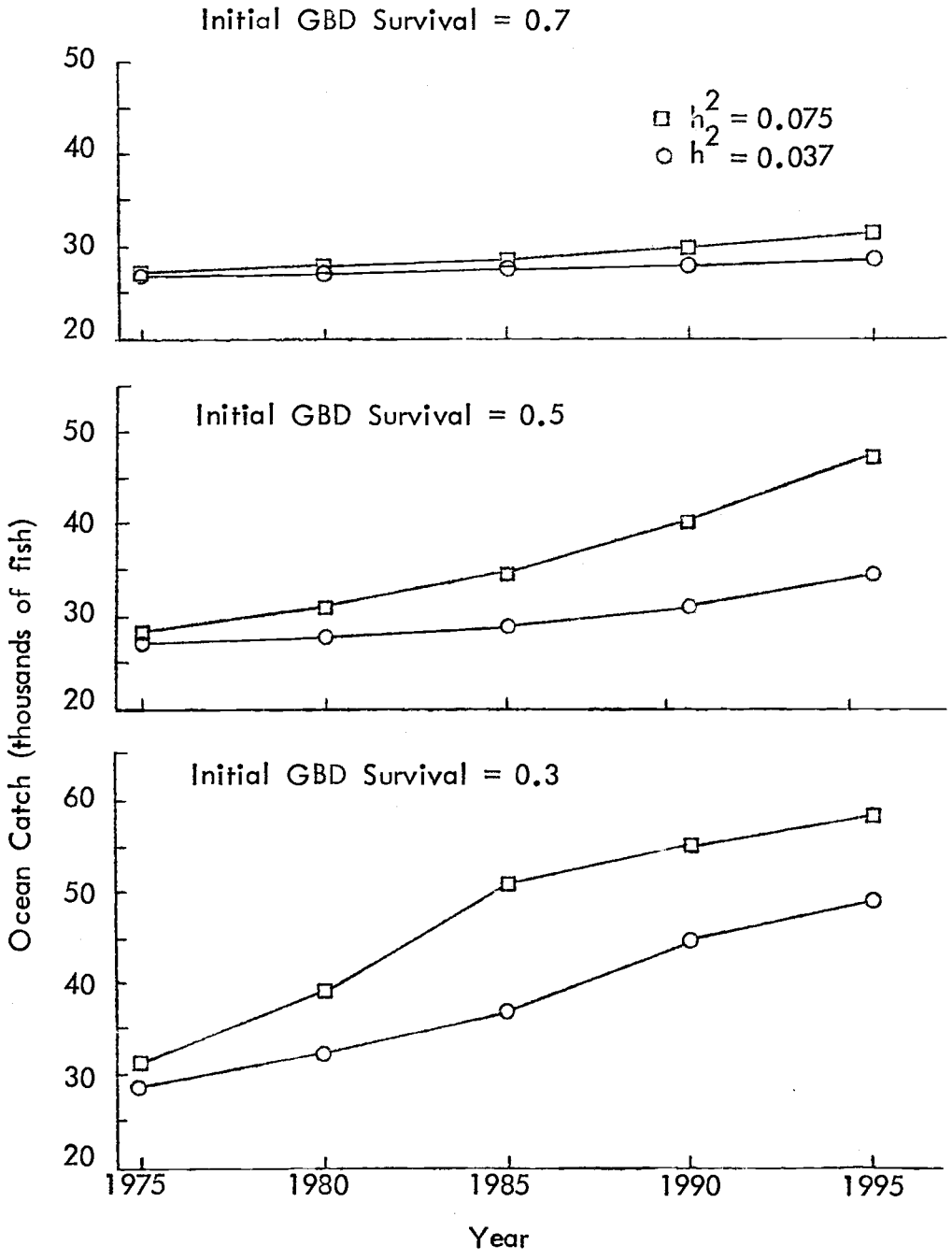

Figure 9. Expected increase in ocean catch of Kalama Hatchery fall chinook at different heritabilities and proportions selected. Gas bubble disease is denoted by GBD.

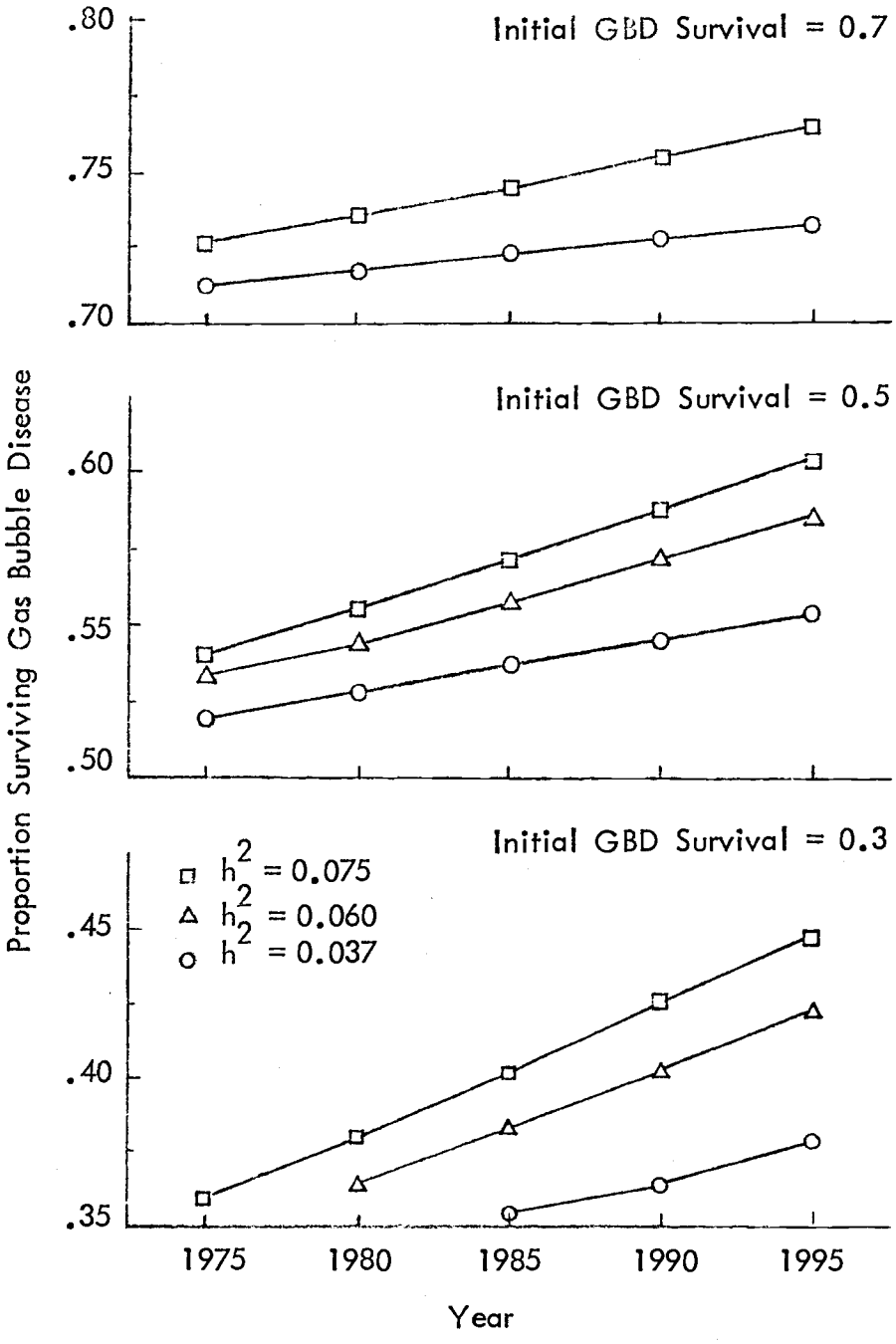

Figure 10. Expected increase in the proportion of Kalama River fall chinook surviving gas bubble disease through time. Gas bubble disease is denoted by GBD.

The model can be used to determine the effect that elimination of lethal levels of air supersaturation would have on future returns of fall chinook salmon to Kalama Hatchery. Computer runs were made using each of the instantaneous natural smolt mortality rates from Table 10, but assuming that no fish died from gas bubble disease. Under these conditions, catches and escapements rapidly increased to a maximum which was determined by the survival rate of smolts. These maximum returns were reached within 8 to 14 yrs. The ocean catch and hatchery returns after 15 years when lethal levels of supersaturation were eliminated are compared to the returns when supersaturation was not altered in Table 11.

Table 11. Returns of fall chinook salmon to Kalama Hatchery after 15 yrs when lethal levels of supersaturation remain the same and when they are eliminated. Heritability was set at 0.037. The value on the left is the number of fish in the ocean catch and the second value is the number of fish returning to the hatchery.

	Initial mortality due to gas bubble disease					
	0.3		0.5		0.7	
Supersaturation eliminated	49,812	4,399	80,930	7,074	134,908	11,792
Supersaturation unchanged	26,651	2,260	27,900	2,494	32,622	2,745

If the average mortality of smolts due to gas bubble disease is now 30 percent, then elimination of lethal levels of supersaturation will result in a 195 percent increase in the number of adults returning to the hatchery in 15 yrs. For the elimination of mortality levels of 50 to 70 percent on smolts, the number of adults returning to the hatchery in 15 yrs can be expected to increase by 284 percent and 430 percent respectively. This indicates that the return of adult chinook salmon to the hatchery is an exponential function of smolt survival. The same was found to be true for ocean and river catches.

It can be seen that elimination of lethal levels of air supersaturation resulted in large increases in catches and escapements. The magnitude of this increase rose sharply with the level of mortality from gas bubble disease currently being suffered by the stock. These results emphasize the great economic benefit that can be realized if lethal levels of air supersaturation are eliminated from the Columbia River.

CONCLUSIONS

1. Resistance to death from gas bubble disease in Columbia River fall chinook is significantly influenced by additive genetic factors.

2. The heritability of resistance to gas bubble disease is probably 10 percent or less.

3. Gas bubble disease mortalities in the Columbia River appear to have reduced the genetic variability influencing gas bubble disease resistance of fall chinook, so that heritability is now probably lower than it initially was.

4. Natural selection in the lower Columbia River for increased resistance to gas bubble disease for the next 30 years will result in a maximum increase in smolt survival of five to ten percent. This means that gas bubble disease will remain as a major source of smolt mortality until lethal levels of air supersaturation are eliminated.

5. Population modeling has shown that catch and escapement are exponential functions of smolt survival, so that any reduction of current levels of mortality due to gas bubble disease would have a great impact on the production of Columbia River fall chinook.

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APPENDICES

APPENDIX 1

Average Time to Death for Each Tank-Family (Heritability Experiment)

	Average Time to Death (Females)											
	1			2			3			4		
Males	A[a]	B	C	A	B	C	A	B	C	A	B	C
1	24.47	29.03	25.27	19.24	25.12	24.22	25.35	24.98	22.37	22.78	17.95	21.87
2	21.03	23.43	17.29	23.46	24.15	21.18	21.39	23.93	32.13	22.60	27.40	22.40
3	18.89	24.13	22.57	21.60	25.13	31.71	24.68	26.96	28.5	24.13	25.43	25.02
4	19.63	28.83	19.68	34.76	24.37	30.25	21.64	25.37	18.86	18.39	30.49	25.28
5	21.125	24.60	18.98	20.15	36.17	29.54	22.18	27.83	26.95	23.6	28.77	24.31
6	21.18	25.25	26.06	16.68	26.12	27.10	13.58	18.77	19.64	18.71	25.03	24.46
7	17.66	25.92	25.37	29.80	21.86	38.15	21.67	24.80	29.02	21.66	24.21	21.55
8	24.02	22.29	35.78	17.13	19.35	18.95	14.98	28.02	21.16	26.40	19.19	22.52
9	32.40	22.85	27.33	23.60	25.54	27.13	27.33	27.88	21.71	24.70	25.80	27.60
10	26.31	26.14	25.69	19.65	28.00	28.36	23.55	25.80	28.27	27.89	33.18	22.00
11	20.50	28.10	30.68	27.20	30.91	30.92	17.98	24.98	30.52	18.90	21.29	17.26
12	21.13	23.77	24.17	28.80	24.19	28.19	22.81	27.61	20.0	24.74	26.58	25.21
13	18.74	29.72	20.38	20.19	20.52	19.16	21.81	23.53	26.72	18.91	29.65	24.11
14	23.83	21.55	20.98	18.27	21.15	22.00	16.89	16.34	22.93	14.98	15.75	17.62
15	21.02	22.58	21.03	22.80	24.57	23.02	25.60	19.92	21.27	14.95	23.32	15.64
16	24.23	27.32	30.07	32.47	24.78	32.78	28.5	32.21	48.71	26.80	23.06	26.98
17	20.31	18.17	21.05	20.72	24.10	22.77	17.31	19.88	24.45	23.07	29.92	32.27
18	22.68	25.10	22.57	21.07	23.63	32.93	21.69	19.77	17.7	25.38	33.86	31.95
19	41.39	30.58	26.35	30.15	23.31	23.58	25.84	18.65	27.84	36.73	21.25	25.17
20	26.97	21.13	26.37	17.95	27.61	29.33	19.12	24.07	20.15	17.17	15.85	26.3

[a] A, B, C, designate tanks.

APPENDIX II

Proportion Alive After 36 Hours in Each Tank-Family (Heritability Experiment)

		Females											
		1			2			3			4		
		A[a]	B	C	A	B	C	A	B	C	A	B	C
Males	1	.226	.226	.229	.121	.182	.167	.233	.25	.116	.207	.103	.088
	2	.200	.088	.094	.222	.161	.148	.214	.185	.323	.172	.167	.154
	3	.065	.115	.194	.207	.200	.387	.24	.255	.276	.179	.138	.267
	4	.068	.285	.143	.211	.200	.167	.200	.293	.182	.065	.285	.172
	5	.143	.111	.143	.129	.483	.286	.214	.200	.200	.280	.219	.259
	6	.176	.214	.231	.036	.172	.276	.033	.067	.136	.032	.138	.115
	7	.065	.212	.143	.321	.207	.300	.205	.148	.207	.107	.138	.138
	8	.233	.103	.440	.071	.037	.103	.071	.259	.069	.300	.074	.107
	9	.345	.083	.267	.226	.214	.214	.233	.25	.194	.233	.107	.36
	10	.258	.192	.172	.077	.333	.250	.241	.172	.250	.250	.280	.93
	11	.136	.240	.250	.185	.286	.267	.071	.261	.409	.087	.069	.069
	12	.217	.143	.300	.367	.185	.346	.185	.242	.200	.200	.323	.229
	13	.069	.241	.107	.138	.069	.159	.231	.182	.200	.103	.204	.125
	14	.154	.067	.071	.067	.077	.172	.094	.034	.182	0.0	.007	.033
	15	.103	.128	.207	.179	.172	.214	.233	.143	.115	.036	.250	.034
	16	.167	.321	.310	.296	.200	.370	.400	.286	.538	.286	.154	.250
	17	.176	.037	.129	.103	.207	.207	.138	.100	.200	.222	.192	.929
	18	.158	.172	.222	.143	.250	.296	.269	.143	.133	.192	.286	.333
	19	.435	.346	.304	.346	.038	.167	.286	.167	.241	.423	.111	.172
	20	.25	.042	.200	.107	.208	.333	.103	.107	.160	.226	.045	.200

[a] A, B, C, designate tanks.

Appendix 3. Fortran program used for Kalama Hatchery fall chinook population model.

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      PROGRAM COLUMBIA
      DIMENSION Z(100),SPAWN(35,5),XMATURE(35,5),RECRUIT(35),
     1SMOLT(35),SSMOLT(35),TSPAWN(35),F(5),P(5),RF(5),
     1RCATCH(5),TRCATCH(35),OCATCH(5),TOCATCH(35),RESIST(35),
     1OWEIGHT(5),RWEIGHT(5)
      DATA (Z=2.665,2.421,2.209,2.155,2.063,1.986,
     11.913,1.856,1.804,1.755,1.709,1.667,1.627,1.589,
     11.554,1.521,1.489,1.459,1.428,1.400,1.372,1.346,1.320,
     11.295,1.271,1.248,1.225,1.202,1.180,1.159,1.138,1.118,1.097,
     11.078,1.058,1.039,1.020,1.00,.984,.966,.948,.931,.913,.903,
     1.880,.863,.846,.830,.814,.798,.782,.766,.751,.735,
     1.720,.704,.689,.674,.659,.644,.629,.614,.599,.585,
     1.570,.555,.541,.530,.513,.497,.482,.468,.453,.438,.424,
     1.409,.394,.380,.365,.350,.335,.320,.305,.290,.274,.260,
     1.243,.227,.211,.195,.180,.162,.144,.127,.109,.089,
     1.0701,.049,.027)
      READ(1,10)SO,D,M,V,RC,TSPAWN(5),(SPAWN(I,5),I=1,5),
     1(SPAWN(I,4),I=2,5),
     1(SPAWN(I,3),I=3,5),(RESIST(I),I=1,5),
     1(P(I),F(I),RF(I),I=2,5)
10    FORMAT(5F5.3/F6.0,5F5.0,4F6.0/3F6.0,5F5.3/12F5.3)
      WRITE(2,11)
11    FORMAT(5X,*RESIST*,5X,*TSPAWN*,5X,*INCRS*,5X,*EXCESS*,
     15X,*OCATCH*,5X,*INCRS*,5X,*RCATCH*,5X,*INCRS*,
     15X,*TOWEIGHT*,5X,*TRWEIGHT*)
      DO 50 I=5,35
      SMOLT(I)=TSPAWN(I)*2244.
      IF(SMOLT(I).GT.8000000.)SMOLT(I)=8000000.+(SMOLT(I)-8000000.)*0.05
      SSMOLT(I)=SMOLT(I)*RESIST(I)
      RECRUIT(I)=SSMOLT(I)*EXP(-SO)
      XMATURE(I,2)=RECRUIT(I)*P(2)
      XMATURE(I,3)=RECRUIT(I)*EXP(-D-F(3))*(1-P(2))*F(3)
      XMATURE(I,4)=RECRUIT(I)*EXP(-2*D-F(3)-F(4))*(1-P(2))*(1-F(3))*
     1P(4)
      XMATURE(I,5)=RECRUIT(I)*EXP(-3*D-F(3)-F(4)-F(5))*(1-P(2))*
     1(1-P(3))*(1-F(4))
      DO 20 L=3,5
20    SPAWN(I,L)=XMATURE(I,L)*EXP(-RD-RF(L))
      TSPAWN(I+1)=SPAWN(I-2,3)+SPAWN(I-3,4)+SPAWN(I-4,5)
      RESIST(I+1)=SPAWN(I-2,3)/TSPAWN(I+1)*RESIST(I-2)
     1+SPAWN(I-3,4)/TSPAWN(I+1)*RESIST(I-3)+
     1SPAWN(I-4,5)/TSPAWN(I+1)*RESIST(I-4)
      K=(RESIST(I+1)+.005)*100
      S=Z(K)
      R=H*S*V
      RESIST(I+1)=RESIST(I+1)+R
      IF(RESIST(I+1).GE.1.00)GO TO 18
      GO TO 19
18    RESIST(I+1)=1.000
19    EXCESS=0.0
      IF(TSPAWN(I+1).GT.4167)EXCESS=TSPAWN(I+1)-4167
      TSPAWN(I+1)=TSPAWN(I+1)-EXCESS
      SGAIN=(TSPAWN(I+1)-TSPAWN(I))/TSPAWN(I)
      OCATCH(3)=RECRUIT(I-3)*(1-P(2))*F(3)/(F(3)
     1+D)*(1-EXP(-F(3)-D))
      OCATCH(4)=RECRUIT(I-4)*EXP(-F(3)-D)*(1-P(2))*
     1(1-P(3))*F(4)/(F(4)+D)*(1-EXP(-F(4)-D))
      OCATCH(5)=RECRUIT(I-5)*EXP(-F(3)-F(4)-2*D)*(1-P(3))*(1-
     1P(4))*(1-P(2))*F(5)/(F(5)+D)*(1-EXP(-F(5)-D))
      TOCATCH(I)=OCATCH(3)+OCATCH(4)+OCATCH(5)
      OGAIN=(TOCATCH(I)-TOCATCH(I-1))/TOCATCH(I-1)
      OWEIGHT(3)=OCATCH(3)*9.205
      OWEIGHT(4)=OCATCH(4)*13.71
      OWEIGHT(5)=OCATCH(5)*20.77
      TOWEIGHT=OWEIGHT(3)+OWEIGHT(4)+OWEIGHT(5)
      RWEIGHT(2)=RCATCH(2)*6.177
      RWEIGHT(3)=RCATCH(3)*15.39
      RWEIGHT(4)=RCATCH(4)*24.21
      RWEIGHT(5)=RCATCH(5)*27.93
      TRWEIGHT=RWEIGHT(2)+RWEIGHT(3)+RWEIGHT(4)+RWEIGHT(5)
      DO 25 M=2,5
      JJ=I-M
25    RCATCH(M)=XMATURE(JJ,M)*RF(M)/(RF(M)+D)*
     1(1-EXP(-RF(M)-D))
      TRCATCH(I)=RCATCH(2)+RCATCH(3)+RCATCH(4)+RCATCH(5)
      RGAIN=(TRCATCH(I)-TRCATCH(I-1))/TRCATCH(I-1)
50    WRITE(2,13)RESIST(I),TSPAWN(I),SGAIN,EXCESS,TOCATCH(I),
     1OGAIN,TRCATCH(I),RGAIN,TOWEIGHT,TRWEIGHT
13    FORMAT(6X,F5.3,F11.0,F11.3,4F11.3,F10.0,F11.0,F10.3,4F13.3)
      CALL EXIT
      END
```