

Competitive Ability and Mortality of Growth-Enhanced Transgenic Coho Salmon Fry and Parr When Foraging for Food

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Abstract.—Coho salmon *Oncorhynchus kisutch* were genetically altered to produce growth hormone without regulation, causing them to grow 11 times larger (on average) than control fish after 14 months. This technology has potential benefits for the aquaculture industry, but the environmental risk associated with the escape of transgenic fish into the wild is not known. To partially address this issue, we experimentally investigated how well transgenic salmon survive when given a choice to consume food in a predator's presence. If transgenic salmon are to retain their growth advantage, we predict that they must also be more effective at competing for food than wild salmon and be willing to suffer higher mortality rates when foraging. The relative competitive abilities of transgenic and control salmon at two different ages were tested with an unequal competitor ideal free distribution. A larger proportion of transgenic salmon fed within the system, although they were not overrepresented at a higher-quantity food source. When feeding in the presence of a predator, there was no measurable difference in mortality rates between transgenic and control salmon at both the fry and parr stages. These data indicate that, under the limited environmental conditions we tested, transgenic coho salmon are at least competitively equal to control fish and do not suffer higher rates of mortality to acquire food resources and maintain their enhanced growth.

Pacific salmon *Oncorhynchus* spp. have been successfully genetically altered to produce growth hormone without regulation, resulting in increased growth rates (Du et al. 1992; Devlin et al. 1994, 1995b). On average, transgenic coho salmon *O. kisutch* at 14 months of age are 11 times larger than control salmon (Devlin et al. 1994). In addition to accelerated growth, transgenic coho salmon differ in their morphology, behavior, and physiology (Ostenfeld et al. 1998; Abrahams and Sutterlin 1999; Devlin et al. 1999; Stevens et al. 1999; Hill et al. 2000; Stevens and Devlin 2000). Before transgenic salmon can be used commercially, several socioeconomic issues must be addressed. One concern is the potential environmental risk posed by growth-enhanced fish should they ever escape into wild populations. Generally, trade-offs exist between growth rate and fitness, where survival

may be threatened if the heightened capacity of some physiological properties (allowing the enhanced growth) occurs at the expense of other physiological properties or at an increased risk of predation (Arendt 1997).

Since salmon have not naturally evolved to grow at maximal physiological rates, some traits mediated by an overexpression of growth hormone are probably disadvantageous. There are, in fact, several trade-offs between growth and survival that may explain the evolution of submaximal growth rates in the early life history of fish (Conover and Schultz 1997; Mangel and Stamps 2001) as well as some developmental, physiological, and behavioral features that have been revealed in transgenic coho salmon (Devlin et al. 1995a, 1995b; Farrell et al. 1997; Ostenfeld et al. 1998; Abrahams et al. 1999; Jhingan et al. 2003; Lee et al. 2003; Sundström et al. 2004).

If transgenic salmon are to retain their growth advantage under natural conditions, it is assumed that they would need to be more effective at competing for food than wild salmon to meet their increased metabolic requirements. Ideal free distribution theory can be used to test the relative

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competitive abilities of fish. In an ideal free distribution, individuals are distributed so that none would benefit by switching sites (Fretwell and Lucas 1970; Fretwell 1972), and the proportion of individuals at each site matches the proportion of resources in each site (input matching: Parker 1974). The ideal free distribution theory has been further developed by considering the situation where not all individuals are equal but instead differ in competitive ability (Sutherland and Parker 1985; Parker and Sutherland 1986). To test relative competitive ability, we measured the spatial distribution of the transgenic and control individuals in a system made up of two habitats: one with a high concentration of food and one with a low concentration of food. According to an unequal competitors ideal free distribution, the more competitive individuals are expected to occur more frequently at the higher-quantity food source (Sutherland and Parker 1985). This technique has been used to assess variation in competitive abilities among nontransgenic coho salmon (Grand 1997; Grand and Dill 1997).

To maintain increased rates of growth, fish must make trade-offs between foraging effort and risk of predation (Abrahams and Dill 1989). This suggests that transgenic fish will have higher mortality rates relative to control fish when foraging in the presence of a predator. Fish with high growth rates as a result of different growth hormone supplements have been found to be more willing to risk increased predation to forage (Johnsson et al. 1996; Jönsson et al. 1996; Abrahams and Sutterlin 1999; Dunham et al. 1999; Abrahams and Pratt 2000; Sundström et al. 2004). To maintain their increased rate of growth, transgenic salmon may have to shift the optimal tradeoff between growth and survival to a more "high-gain/high-risk" phenotype (Johnsson 1993).

Experiments were designed to assess whether transgenic fish have altered growth rates and increased mortality relative to wild fish when foraging in the presence of predators. To observe mortality rates during the fry stage, a rearing system was produced with three distinct habitats: (1) no food, no predator; (2) low food, no predator; and (3) high food, predator. At the parr stage, a two-habitat system was designed: (1) food, predator; and (2) no food, no predator. These habitats were designed to provide fish with a choice to either eat and risk predation or to hide and be safe. Transgenic and control individuals were placed together in the system and the relative mortality was observed as the individuals competed for food re-

sources in the presence of a predator. The hypothesis being tested was that the transgenic fish would choose to spend more time eating, thereby suffering higher predation relative to the control fish. As the parr were large enough to be individually tagged, their specific growth rates for the duration of the experiment could also be determined.

Methods

Experimental Subjects

Fry.—The experimental transgenic group was produced from a cross of growth-hormone-transgenic and wild coho salmon (Chehalis River strain). The transgenic fish (strain M77) contained the gene construct OnMTGH1 and were third-generation individuals derived from and maintained by backcrossing with Chehalis River wild coho salmon (Devlin et al. 1994, 2004a). Because the transgenic parent was hemizygous for the transgene and survival of both genotypes was equal to this stage, approximately 50% of the offspring possessed the transgene at fertilization (Devlin et al. 2004a). The control group consisted of 100% nontransgenic fish of wild Chehalis River strain. Both groups (transgenic and control) consisted of five single-pair crosses. The families were fertilized in January 2000, and fish in the transgenic and control groups were size matched by manipulation of rearing temperature (between 8°C and 11°C). This was necessary because the transgenic fish develop faster than the nontransgenic fish (Devlin et al. 2004a). At the beginning of the experiment (May 2000), the fry had been fed for 2 weeks to establish feeding patterns, but the mean weight of the fish in the transgenic group (mean \pm SD, 0.41 \pm 0.02 g) was not significantly different than the control group (0.42 \pm 0.02 g; $t_{118} = -0.194$; $P = 0.847$).

Parr.—The transgenic and control groups were derived from the same strains (described previously) but were fertilized in January 2003 (five single-pair crosses per group). The transgenic individuals were fed a restricted ration that maintained size matching with control animals fed a satiation ration (as in Sundström et al. 2004; Devlin et al. 2004b) until the fall when they were used in the predation experiment (September–December 2003). By the parr stage, individuals carrying the transgene could easily be distinguished from those fish not carrying the transgene on the basis of size. Therefore, all individuals in the transgenic group carried the transgene (as opposed to the fry transgenic group).

TABLE 1.—Results of a feeding competition experiment with growth-enhanced-transgenic and control coho salmon fry conducted in May 2000. Shown are mean \pm SD weights in six trials in two replicate tanks (A and B).

Trial	Tank A		Tank B	
	Transgenic	Control	Transgenic	Control
1	0.28 \pm 0.03	0.28 \pm 0.06	0.21 \pm 0.01	0.21 \pm 0.03
2	0.33 \pm 0.10	0.21 \pm 0.04	0.22 \pm 0.05	0.20 \pm 0.01
3	0.39 \pm 0.10	0.40 \pm 0.10	0.47 \pm 0.11	0.48 \pm 0.12
4	0.40 \pm 0.12	0.49 \pm 0.10	0.41 \pm 0.14	0.47 \pm 0.08
5	0.40 \pm 0.05	0.50 \pm 0.11	0.49 \pm 0.09	0.40 \pm 0.08
6	0.61 \pm 0.06	0.60 \pm 0.05	0.61 \pm 0.09	0.60 \pm 0.07

Feeding Competition Experiment

Two glass aquaria (each 60.00 cm long \times 21.75 cm high \times 18.75 cm deep) were used in this experiment. Each tank had two plastic feeding tubes placed 5 cm from each end of the tank that allowed food (NutraPlus Starter mash, Moore Clarke [now Skretting Canada], Vancouver, Canada) to be placed in the tank at a consistent 2:1 ratio between the tubes (each feeding delivered 0.10 g and 0.05 g, respectively, over a period of 20 min). For details on the design of the feeding system, see Abrahams (1989). The locations of the high and low feeders within the tank (left or right side) were assigned randomly each day and remained the same throughout the day. The photoperiod within the experimental room was maintained at a 12-h cycle; fluorescent ceiling lights came on at 0700 hours and turned off at 1900 hours. The building was completely dark at night because all windows were covered.

A total of 120 fish were used in this experiment: 60 from the transgenic group and 60 from the control group. At the start of the experiment, transgenic and control fish were the same size and selected randomly for the experiments. By day 6 the transgenic salmon demonstrated such rapid growth that the largest control individuals were selected to ensure size matching was maintained. We also attempted to size match the groups in each tank (Table 1) to avoid size-associated differences in competitive ability (Huntingford et al. 1990). Before being used in the experiment, all fish were identified with a small, numbered acetate tag painted with acrylic paint (as in Abrahams and Sutterlin 1999) and allowed a minimum recovery of 2 d. During the recovery period, both the experimental and control groups were placed together in the same tank at 15°C and fed with the same automated feeders used during the experimental trials.

Five individuals from the transgenic group and five individuals from the control group were placed into each tank in the late afternoon and allowed

to acclimate overnight. The following day, three trials with feed addition were run at 0900, 1200, and 1500 hours. Before each trial, the fish were removed from the tanks and the water was changed to remove excess food from the system. Food was placed into the glass flasks before fish were returned to the tanks. Once the fish were placed back into the tank, they were allowed to reacclimate 2 h before trials were initiated. Each trial lasted 20 min and a remote control video camera was used to film the tanks during the entire period. Two groups were filmed simultaneously each day. In total, 12 groups of fish were used for this experiment.

Since the genetic makeup of the five individuals from the transgenic group was not known, these individuals were preserved after the experiments for later polymerase chain reaction (PCR) analysis. The DNA was isolated by means of standard phenol-chloroform-isoamyl extraction (Sambrook and Russell 2001), and transgene identification was accomplished by amplification with primers MT-1 (5'-CTGATTAAGTTTGTATAGT-3') and GH-19 (5'-GTTAAATTGTATTAAATGGT-3') followed by electrophoresis in 0.8% agarose gels (Devlin et al. 2004a). Hereafter, reference to a transgenic group indicates a group consisting of 100% transgenic fish, while the sib-control group consists of nontransgenic fish (as revealed by the PCR analysis) that are siblings to the transgenic fish.

Spatial analysis of the trials was completed by viewing the videotapes. Analysis of the videotapes provided data on the location of each individual during the trials. For each trial, video clips were captured every 30 s. Each 5-s clip was used to determine the spatial distribution of competitors at the high and low food sources. Only fish that were feeding at either the high or low food source (an area 5 cm on either side of the feeding tube) were counted in the spatial distribution. Individuals that were feeding could be identified by their

movement, which included fast darts across the tank to capture a food item or a stationary position under the food supply with short darts to capture the food. Nonfeeding fish generally maintained a stationary position in the tank unless chased by another individual. One group was removed from the analysis because of problems identifying individually marked fish within the tank. This left 11 replicates for analysis.

Using the PCR data, we determined whether the transgenic fish were overrepresented at the most profitable feeder based upon the actual number of transgenic fish in the apparatus. The mean difference between the proportion of fish feeding at the high food source that were transgenic and the proportion of transgenic fish in the entire feeding population was obtained for each group, and then compared to an expected difference of zero with a *t*-test. If the transgene has no effect on feeding behavior, we would expect that the difference between the proportion of fish feeding at the high food source that were transgenic and the proportion of transgenic fish in the feeding population should be zero. The proportion of transgenic and nontransgenic fish feeding was compared with a repeated measures analysis of variance (ANOVA) to assess the impact of time of day and time within each trial.

Mortality Experiment

Fry.—Three round tanks (1 m in diameter) were connected with round pipes (5 cm in diameter). The tanks were arranged in a triangular configuration so that each tank was connected to the other two and so a fish in any one tank had equal access to the other two tanks. A 20-L pail was placed upside down in the center of each tank to form a raceway around the perimeter, and gravel was added to a depth of 10 cm. In each tank, the water depth was 30 cm with an approximated flow of 10 cm/s (maintained by adjusting the rate of water change) and a temperature of 11°C. The system followed strict measures to ensure containment of transgenic fish (with the use of screened traps). One tank was provided with high food (0.2 g of NutraPlus Starter mash three times per day) and four predators (cutthroat trout *O. mykiss*; mean weight \pm SD, 100.1 \pm 16.0 g) that were free to capture and eat any salmon in the habitat. Another tank was provided with half as much food as the high-food habitat (0.1 g of NutraPlus Starter mash, three times per day). The riskier habitat (with predators) was energetically more profitable (high food concentration) and created a conflict for the ju-

venile coho salmon on whether to risk mortality for increased food. The third tank contained neither food nor predators.

For each trial, a group of 50 individuals were removed from the mixed group of transgenic and nontransgenic sibling salmon. These individuals were weighed and frozen for later PCR analysis to estimate the initial ratio of transgenic to nontransgenic fish. Another group of 50 individuals (the experimental group) was removed, weighed, and placed into the safe habitat. After 2 d, the remaining individuals were removed from each habitat, weighed, and preserved for PCR analysis. We repeated this experiment three times.

Parr.—Under anesthetic (tricaine methane sulfonate) 40 transgenic (mean weight \pm SD, 6.9 \pm 1.7 g for trial 1 and 10.1 \pm 3.0 g for trial 2) and 40 control fish (5.6 \pm 0.9 g for trial 1 and 11.3 \pm 2.1 g for trial 2) were weighed, measured, and a passive-integrated transponder (PIT) tag was inserted into the peritoneal cavity. The fish were randomly assigned to one of four groups so that each group consisted of 20 individuals (10 transgenic and 10 control). Two groups were reared in enriched habitats consisting of large (5 m diameter) round tanks conditioned to include some algal growth and a complex substrate bottom that included gravel, rocks, and woody debris. The tanks were divided by mesh netting (7.6 cm stretched) that was large enough for the coho salmon to access both halves of the tank. Before the experiment was initiated, the fish were allowed to habituate until they were readily eating from an automatic vibrating fish feeder (Sweeney Enterprises, Inc., Boerne, Texas) that was programmed to disperse food at 0900, 1200, and 1600 hours was placed in one-half of the tank. To begin the experiment, two burbot *Lota lota* were placed in each tank on the same side as the feeder (four predators total; mean weight = 1,903 \pm 413 g; mean length = 64 \pm 4 cm) so that the coho salmon would have to risk predation to feed. The burbot were not able to cross the mesh divider. The other two groups were reared under culture conditions in 200-L tanks to provide an estimate of mortality and growth when reared in a simple environment without the presence of a predator. After 3 weeks, surviving fish were removed from all habitats, weighed, measured, and euthanized with an excess of MS-222 (tricaine methanesulfonate). All tanks received fresh aerated well water (11°C) at a rate no less than 1 L/kg and the fish were fed a commercial salmon diet (Skretting Canada) at 1.5% by weight/d (divided into three feedings). This experiment was repeated

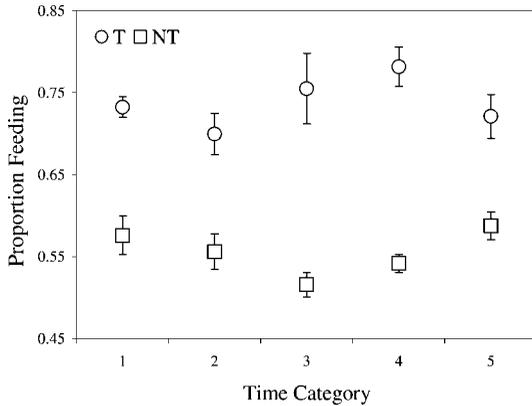


FIGURE 1.—Mean proportion of transgenic (T) and nontransgenic (NT) coho salmon fry feeding during the 20-min trials in May 2003. Each time category represents an average of a 4-min interval (see text). There was no significant effect of time category on the proportion of fish feeding.

twice. Growth and survival were compared between the cultured and enriched habitats with a two-way ANOVA that used habitat (cultured versus enriched) and genotype (transgenic versus nontransgenic) as the main factors.

Results

Feeding Competition Experiment

Using an unequal competitors ideal free distribution, we tested whether the growth hormone transgene had an impact on feeding behavior relative to the control fish. On average, $73 \pm 15\%$ of the transgenic and $55 \pm 16\%$ of the control fish fed throughout the trials. A significantly larger proportion of transgenic fish fed relative to the control fish (t -test: $t_{10} = 3.528$, $P = 0.005$). Consistent with the predictions of an ideal free distribution where we would expect twice as many individuals to occur at the high food source following the 2:1 food ratio, 69% of the feeding fish were located at the high food source.

If the growth hormone transgene has no effect on feeding behavior that is dependent on food supply level, we would expect that the difference between the proportion of fish feeding at the high-food source that were transgenic and the proportion of transgenic fish in the entire feeding population should be zero. The proportion of fish feeding at the high food source that were transgenic (41%) was not significantly higher than the proportion of transgenic fish in the feeding population (35%; t -test: $t_{10} = -0.69$, $P = 0.249$), in-

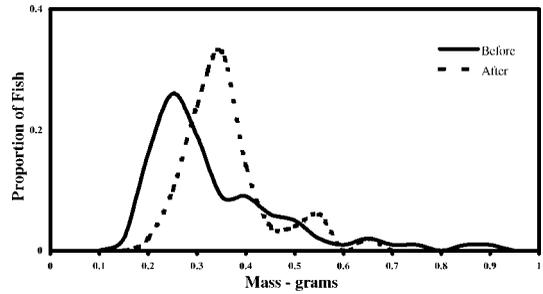


FIGURE 2.—Significant differences were observed in the size distribution of coho salmon fry before and after the three mortality experiments.

dicating no strong influence of the transgene on access to the higher-quantity food source.

To examine the proportion of fish feeding over time within each trial, the 20-min time period was divided into five 4-min categories (Figure 1). Analysis of the mean proportion of fish feeding at 0900, 1200, and 1500 hours indicated no significant effect of time of day on the proportions of fish feeding for both the transgenic (ANOVA: $F_{2,8} = 0.059$, $P = 0.943$) and control groups ($F_{2,8} = 0.379$, $P = 0.696$). However, more transgenic fish were consuming food at all time intervals, supporting an overall effect of genotype on feeding behavior.

Mortality Experiment

Fry.—Relative mortality rates of transgenic and control fry were tested when they fed in the presence of a predator in a seminatural rearing system with three distinct habitats. The proportion of transgenic fish in the six groups (the control group and experimental groups from three replicate experiments that were placed into the rearing system) was calculated with data from the PCR analysis. The proportion of transgenic fry in the three replicates that were not placed into the system (1 = 39%, 2 = 35%, 3 = 24%) was not significantly different from the proportion of surviving transgenic fry (1 = 29%, 2 = 56%, 3 = 36%) in the experimental groups (G -test = 3.48; $df = 2$; $P = 0.324$), indicating there was no differential mortality between wild and transgenic fry.

Figure 2 depicts the size distribution of the fish before and after the mortality experiments. This graph demonstrates the typical skewed size distribution of a mixed group of transgenic and nontransgenic fish; the transgenic fish are those individuals indicated by the heavier and much wider portion of the distribution. There was a significant difference in the size distributions when fish were pooled into six categories and compared with a G -

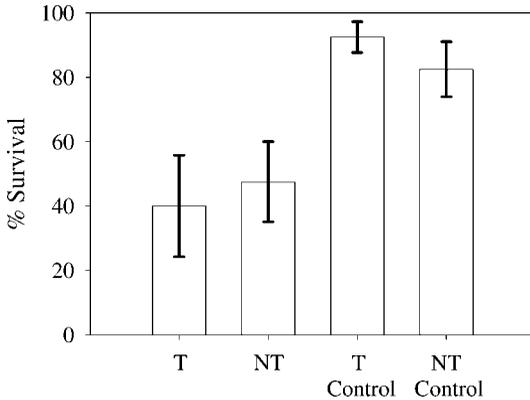


FIGURE 3.—Proportion of transgenic (T) and nontransgenic (NT) coho salmon parr that survived 3 weeks in the enriched (with predator) and cultured (control) habitats. There were no significant differences in mortality rates between the transgenic and control fish, but a significantly higher number of both transgenic and control fish died in the enriched habitat with the presence of predators.

test ($G = 11.86$; $df = 5$; $P = 0.037$). Since the experiment lasted only 2 d, it is unlikely that changes in the size-frequency distribution are a result of growth. A reduction in the variability of the size distribution after being exposed to predators indicates that the fastest- and slowest-growing individuals were those with the highest mortality rates.

Mortality Experiment

Parr.—Relative mortality rates of transgenic and control parr were compared in a two-habitat seminatural rearing system. Survival data from the four trials were pooled because there were no significant differences within or between replicates (Figure 3). There was no significant genotype effect on mortality (ANOVA: $F_{1,12} = 0.0124$, $P = 0.913$), but a significant habitat effect was detected ($F_{1,12} = 15.249$, $P = 0.002$) that indicated fewer

fish survived in the enriched habitat containing predators. Mortality was assumed to be a result of predation; however, it could not be determined whether it was the burbot or large transgenic individuals that preyed upon the fish, although the predation ability of the transgenic fish would be limited because of their smaller size relative to the burbot and their similar size to other cohorts. Although two fish in the cultured habitat of one trial had to be euthanatized because a large transgenic individual was consuming them, mortality levels were still significantly higher in the habitat with predators as indicated above. There was no significant genotype-habitat interaction (ANOVA: $F_{1,12} = 0.610$; $P = 0.450$). No significant differences in initial size of survivors to those individuals that died were detected, indicating a lack of size-selective mortality.

Specific growth rate (SGR) data (SGR_{wt} and SGR_l percentage growth [g or cm] per day) for the two trials (A and B) were not significantly different and were therefore pooled (Table 2). In both the enriched and cultured habitats, transgenics were able to maintain their growth in weight throughout the experiment, whereas the nontransgenic fish lost weight in both habitats. There was a significant genotype effect on growth in weight (ANOVA: $F_{1,101} = 162.085$, $P < 0.001$). Habitat (enriched versus cultured) did not have a significant effect on growth in weight ($F_{1,101} = 0.0210$, $P = 0.885$) and no significant genotype-habitat interaction ($F_{1,101} = 0.672$, $P = 0.0414$). A significant genotype effect was detected for growth in length ($F_{1,101} = 106.230$, $P < 0.001$). There was no significant habitat effect ($F_{1,101} = 0.644$, $P = 0.424$) or genotype-habitat interaction ($F_{1,101} = 0.222$, $P = 0.638$) for growth in length.

Discussion

The results of the competition experiment do not indicate that growth-hormone-transgenic coho

TABLE 2.—Results of a mortality experiment with coho salmon parr. Shown are the mean \pm SD specific growth rates in weight (SGR_{wt} [g·g body weight⁻¹·d⁻¹]) and length (SGR_l [cm/d]) in percentage terms for growth-enhanced-transgenic and nontransgenic parr reared in either enriched (with predators) or cultured (control) habitats. There were two replicate trials, A and B. Transgenic fish had a significantly higher SGR_{wt} ($F_{1,101} = 162.085$, $P < 0.001$) and SGR_l ($F_{1,101} = 106.230$, $P < 0.001$) than nontransgenic fish.

Habitat	Trial	Transgenic		Nontransgenic	
		SGR _{wt}	SGR _l	SGR _{wt}	SGR _l
Enriched	A	1.59 \pm 0.17	0.55 \pm 0.06	-0.18 \pm 0.11	0.04 \pm 0.03
	B	0.60 \pm 0.18	0.15 \pm 0.03	-0.35 \pm 0.03	0.03 \pm 0.01
Cultured	A	2.00 \pm 0.24	0.58 \pm 0.07	-0.06 \pm 0.07	0.04 \pm 0.02
	B	1.11 \pm 0.15	0.28 \pm 0.04	-0.54 \pm 0.05	0.01 \pm 0.01

salmon are more successful at securing higher-quantity food resources relative to size-matched nontransgenic fish. When focusing only on the individuals that were feeding, there was no difference in the proportion of transgenic fish at the high food source to the proportion of transgenic fish within the group. The statistical power of this experiment was low ($P = 0.238$), so differences between the competitive abilities of the transgenic and control fish would have had to be quite large to be detected. Devlin et al. (1999) found that transgenic coho salmon had an increased ability to compete for food, although their data were based on food acquired and eaten under competitive conditions when food was added slowly (one pellet at a time). In this experiment, food was supplied at a higher rate over a 20-min time period, which may have allowed all fish equal access. In addition, there may not have been a large enough spatial difference between the high and low food sources for them to act as two distinct habitats. Research on growth-enhanced transgenic Wami tilapia *Oreochromis hornorum* (also known as *Tilapia urolepis*; Guillén et al. 1999) produced somewhat similar results as Devlin et al. (1999), where transgenic individuals were found to be better competitors than their nontransgenic siblings. However, when compared with wild-type tilapia, the wild individuals were better competitors relative to the transgenic and nontransgenic domestic fish.

The observation in this study that more transgenic fish were feeding in the competition trials relative to the control individuals suggests that the transgenic fish have a greater overall appetite, which perhaps is needed to meet increased metabolic requirements and maintain enhanced growth. Although this would generate the prediction that transgenic fish would be less responsive to risk of predation (and, therefore, suffer increased mortality relative to nontransgenic fish), the results for both the fry and parr stages did not detect a difference in mortality when the fish had to forage under risk of predation. The limited scope of experiments possible within the contained transgenic culture facility results in relatively low statistical power for many experiments (i.e., $P = 0.13$ for fry mortality; $P = 0.05$ for parr mortality), thus warranting caution in applying a conclusion that no difference in mortality rate exists between the genotypes.

These results are in contrast to those of Sundström et al. (2004), who found that transgenic fry suffered significantly higher mortality rates than nontransgenic fry when foraging under risk of pre-

dition in a seminatural system. One important difference between these studies is our design of a system with a safe habitat that had no food and no risk of predation. This provided the fish with an option to obtain food and then hide within the safe habitat. Both studies used pair feeding as a technique for obtaining both age- and size-matched transgenic and control groups, although fry used by Sundström et al. (2004) were first-feeding fry, whereas fry used in this study had been reared under culture conditions for two weeks before use. This difference may point to a critical window of predation sensitivity during the first few days after emergence from natal redds. Of further interest in this study was the observation that the largest and smallest individuals within the fry population experienced mortality. Such an effect may arise if nontransgenic fry with poor growth potential are susceptible to predation because of a weakened physiological state, whereas the fastest growing fry (i.e., transgenic) may be susceptible because they engage in riskier foraging behavior in pursuit of resources, as found by Sundström et al. (2004). The complexity of possible interactions and responses of different phenotypes within real and simulated ecosystems makes reliable predictions of the fitness effects of different genotypes difficult at this time.

Our research indicates that transgenic fish are at least competitively equal to nontransgenic individuals (with more transgenic fish feeding at a given time) and are able to maintain significantly faster growth rates in an enriched environment under the risk of predation without incurring increased mortality. Consequently, they may have the potential to have a significant impact on the survival of wild fish populations if they entered natural ecosystems. Without increased mortality rates, transgenic salmon would have the potential to successfully survive in the wild, provided they could consume enough food to maintain their growth rates. Transgenic fish with similar competitive abilities and rates of mortality (when size matched) could lead to competitive displacement of wild fish if they were able to maintain a size advantage and low levels of mortality (Werner and Gilliam 1984).

We caution, however, that the data derived from this study represent information relating to a specific life history stage in a particular simulated natural environment. Further investigation into other complex habitats and conditions will be required for a more complete analysis of the fitness of growth-enhanced transgenic coho salmon. Con-

flicting results for experiments related to foraging and predation illustrate the profound influence of environmental–genetic interactions (Devlin et al. 2004b), underlining the complexities of conducting laboratory-based risk assessments and the necessity to refrain from extrapolating results of a single experiment to natural environments or life stages.

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