Results of a Pilot Study to Evaluate Effects of Hooking and Handling on Fall-angled Salmonids.

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Note: this version was recovered in 2010 from cd backup. It contains more images than the printed department document.

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Abstract

“Catch and Release” angling is a fisheries management technique designed to improve fishing quality by returning live fish to the water so that they may grow and mature, and be available again for multiple angling bouts. It is a management technique that is supported by many angling organizations and is utilized selectively nationwide by state and federal fisheries management agencies. “Catch and Release” is not an innocuous activity. Fishing’s adversarial nature causes some injury and mortality in fish. In this observational study, looking at lethal and sub-lethal effects of catch and release fishing on mature brook trout Salvelinus fontinalis, landlocked Atlantic salmon Salmo salar, and brown trout Salmo trutta, we attempted to identify quantitative characteristics useful in identifying what effect a “catch and release” fall angling season might have on salmonid broodfish. One hundred two fish were individually fly tagged, released into a holding area, and angled over an 8-week period. Hooking mortality for brook trout was 11%. The relative risk of death after hooking was 2.37x. Hooking mortality for salmon and brown trout was zero. Egg quality and embryo survival of hooked fish was less than 10%. There were no visible gross fish quality measurements differences between hooked fish and not hooked fish. In fact, overall fish quality improved. However, hooked fish were 3.27x more likely than not hooked fish to have skin injuries visible on histology sections. Additionally, nearly all fish had lesions in kidney, pancreas, and liver tissues. These lesions, although due to spawning stress rather than angling, make them poor candidates for survival of multiple stressful events, such as “catch and release” angling at this time of the year.

Introduction

There is a request by some Maine anglers to expand the open water fishing season for salmonids to include a late autumn fishery. This proposed fishery would be exclusively “catch and release” and further restricted to artificial lures and/or fly-fishing. This proposed autumn fishery coincides with the natural spawning period of many feral trout, salmon, and char populations in Maine (Everhart 1976; Warner and Havey 1985). Therefore, state fisheries biologists and fisheries planners are concerned about the possible negative impact of a late autumn fishery upon spawning salmonid populations. Specific concerns are raised by fisheries biologists in several areas:

A. Acute hooking mortality rates in comparison with the spring and summer fishery.
   a. Single catch and release event mortality.
   b. Multiple catch and release events mortality.

B. Post-spawning mortality in comparison to undisturbed populations.

C. Chronic winter mortality due to depletion of energy reserves during the autumn fishery.

D. Chronic winter mortality due to the effects of sub-lethal gross morphological injuries during the autumn fishery.
   a. Ovariorrhysis, damage to ovarian arteries and smaller vasculature, damage to oviducts.
   b. Orchiorrhysis, damage to testicular arteries and smaller vasculature.
   c. Dermabrasion, and subsequent dermatitis, dermatomycosis or other dermatopathy over the winter.
   d. Hooking wound healing at over wintering temperatures.
e. Ova quality, fertility, and embryo survival to hatch.
f. Sperm quality, motility and embryo survival to hatch.

E. Chronic winter mortality due to effects of sub-lethal histological injuries.
a. Histological damage to dermis and musculature
b. Histological damage to the myocardium, renal tissues, hepatic tissues, spleen, ovaries, testes, or gastrointestinal tissues.

F. Effects on Offspring
a. Impacts of disturbing gravid females from defended redds.
b. Impacts on deposition of fertilized embryos into redds.
c. Impacts of human foot traffic on incubating embryos.
d. Impacts of human foot traffic on spring emergence of fry.

G. Long-term effects of damage to spawning populations if ovaries or testes of fish are damaged during the autumn fishery.

H. Effects on catch rate during ice fishing season.

“Catch and Release” fishing has been one tool used by fishery managers nationwide to produce “quality” angling with the logic that fish will be angled multiple times eventually living to reach a large size. Under light to moderate angling pressure, this concept works well in managing for quality fishing. However, the factors of hooking mortality or sublethal hooking stress may become important considerations for the manager in programs with heavy fishing pressure or when fish are otherwise stressed (Wydoski 1977).

Stress in fish can disrupt normal metabolic and osmoregulatory functions and various stressors are cumulative in their effect. Hooking stress does not cause mortality in fish that are in good physiological condition. However, hooking stress added to fish that are already under stress from adverse environmental conditions or pollutants may cause mortality either directly or indirectly by allowing them to become more susceptible to predators, diseases, or parasites. The delayed mortality that results from stress can be more important than the initial hooking mortality that is observed and must be taken into account by fishery managers (Wyoski 1977).

To date, hooking mortality studies have included fish from a variety of species (Wyoski 1977). In fact, more than 40 published studies on hooking mortality rates and their concomitant variables have been conducted between 1930 and 2000. In 1992, Taylor and White synthesized the hooking mortality literature on nonanadromous trout. Their meta-analysis of 18 studies concluded that trout caught on bait died at higher rates than trout caught on artificial flies or lures, that fish caught on barbed hooks had higher mortality rates than fish caught on barbless hooks, that brown trout _Salmo trutta_ had lower mortality rates than other species of nonanadromous trout, and that wild trout died at higher rates than hatchery reared trout. The overall average mortality rate in these 18 studies was about 12%. Under the best condition, with barbless flies or lures, the percentage dropped to under 3% (Taylor and White 1992).

Fish that have a vital organ damaged from a hook(s) have an initial mortality that may be immediate or occur within the first 24-hours. For example, Klein (1965) demonstrated that the majority of the rainbow trout (>50%) were dead within 24-hours. However, Klein emphasized that lures with a single hook were taken farther into the fish's mouth and resulted in a more serious wound than treble hooks. As a result, almost all fish caught on single hooks died within two days while those caught on treble hooks died more gradually because their wounds were not as serious. Similar results were obtained by Stringer (1967) for rainbow trout that were caught on various types of

Figure 1. Fall fly fishing in shallow river. MDIF&W photo.
terminal gear. Bouck and Ball (1966) found that rainbow trout caught on lures and played to exhaustion did not exhibit mortality initially but died from progressive shock—about 20 percent of the fish died three days after being hooked, slightly less than 60 percent by day four, nearly 80 percent by day five, and 87 percent by day nine.

Few published studies have been conducted on hooking mortality in pre-spawning trout (or other fish) or on spawning grounds (Maramiche 1990). This may be, in part, due to the fact that “catch and release” is a fisheries management technique applied to a fish population, not a technique directed specifically at a subset of the fish population (ie., broodfish). The mortality associated with “catch and release” angling may have vastly different effects on the fish population if angler’s specifically target broodfish.

**Methods**

This is an observational study. It identifies risk factors and estimates the quantitative effects of the various component causes that contribute to the occurrence of injury or death in this group of fish. The investigation is based on analysis of injury or death occurrences in a population by comparing groups of individuals with respect to disease occurrence and exposure to hypothesized risk factors. Observational studies differ from experimental studies. In the former, the investigator is not free to randomly allocate factors (disease and hypothesized risk factors—injury/death and number of hooking events during Autumn) to the individuals, whereas in the latter the investigator is free to allocate factors to individuals at random (Thrusfield 1995).

**Subjects:** One hundred two free-living adult size salmonids residing in the water discharge outlet of the Cobb State Fish Hatchery, Enfield, Maine.

<table>
<thead>
<tr>
<th>Fish Type</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brook trout <em>Salvelinus fontinalis</em></td>
<td>75</td>
</tr>
<tr>
<td>Landlocked Atlantic salmon <em>Salmo salar</em></td>
<td>24</td>
</tr>
<tr>
<td>Brown trout <em>Salmo trutta</em></td>
<td>3</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>102</strong></td>
</tr>
</tbody>
</table>

**Marking:** Each fish was tagged through the dorsal fin base with a numbered floy-type tag.

**Fish Quality Measurements:** MDIF&W’s hatchery division has conducted semi-annual quantitative fish quality measurements on all production lots since 1979. Fish quality assessments were conducted at the beginning and end of the this study. Each fish was individually morphologically sexed, weighed (grams), measured for total length (millimeters), and scored on a DIF&W fish quality datasheet.

**Null Hypotheses (Ho):**

1. There is no relative risk (RR=1) difference in 48-hour post release survival between brook trout subjected to single or multiple “catch and release” events and brook trout not subjected to “catch and release” events.
2. There is no relative risk (RR=1) difference in gross (a) external or (b) internal pathological differences between brook trout subjected to single or multiple “catch and release” events and brook trout not subjected to “catch and release” events.
3. There is no difference in egg fertilization (a) or embryo (b) survival to hatch between brook trout subjected to single or multiple “catch and release” events and brook trout not subjected to “catch and release” events.
4. There is no relative risk (RR=1) difference in 48-hour post release survival between brook trout that hemorrhaged as a result of a hooking injury and brook trout that did not hemorrhage as a result of a hooking injury.
5. There is no relative risk (RR=1) of histological differences between brook trout subjected to single or multiple “catch and release” events and brook trout not subjected to “catch and release” events.

**Alternative Hypotheses:**

1. Not 1 above.
2. Not 2 above.
3. Not 3 above.
4. Not 4 above.
5. Not 5 above.

**Angling Equipment:** Two casting type rod and reel...
sets and one fly-fishing type rod and reel were provided at the hatchery for fishing. The following artificial barbed lures and flies were used.

Mepps No.1. Minnow       Dry fly midge (black)
Mepps No.1. Spinner       Dry fly diptera (brown)

Angling Effort: Angling began the 48 hours after fly tags were attached to the fish’s dorsal fin. Anglers used provided equipment and data sheets were located near the pond to record catch and release information (See sample data sheet). Hooks were removed manually. Deeply hooked fish were to be released after the line was cut flush with the fish’s mouth. Anglers recorded playing time, lure type, hooking location, and visible hemorrhage. A total time of angling effort was not kept.

Mortality Monitoring: Pools were checked at least twice daily for mortalities. Dead and severely moribund fish were to be removed promptly, examined immediately or refrigerated for subsequent inspection.

Results
Sixty-seven of 102 fish (66%) retained their fly tags during the eight week hooking mortality study. Tags were lost primarily from brook trout (18♂; 16♀). Only one tag was missing from a landlocked Atlantic salmon and no tags were missing from brown trout. Of the fish who lost tags, 19 had been hooked and landed at least once; while 16 were never recorded as hooked. Three untagged brook trout were found dead at the end of the study. They were found dead, stuck in the outflow grate. They did not have tags and they were to autolysed to determine if they had been hooked.

Hooking Mortality: Four fish died during the angling period of this study—one brook trout males and two brook trout females. All the dead fish had been hooked at least once. Two of the four were landed and two escaped without being landed. Fish #538 (♀) was hooked in the gill, hemorrhaged, and died immediately after being released. Fish #539 (♀) was hooked, played, escaped without being landed, but died within 20 minutes. Fish #584 (♀) was hooked, landed and released, but didn’t die until 14 days later. Fish #588 (♀) was found dead in the tank, not recorded as being hooked; however, she was found to have a hooking injury in her mouth.

Thirty-four of 75 (45%) brook trout were hooked at least once during the study. The brook trout hooking

<table>
<thead>
<tr>
<th>Acute Hooking Mortality (Percentage)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. Hooked 1+ times</td>
</tr>
<tr>
<td>---------------------</td>
</tr>
<tr>
<td>Brook Trout (males)</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Brook trout (females)</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Brook Trout (total)</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Landlocked Atl. Salmon</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Brown trout</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Total (3 species)</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

Table 1. Acute hooking mortality results
[This value includes the three untagged dead fish].

<table>
<thead>
<tr>
<th>H0(1): Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of Dead Fish</td>
</tr>
<tr>
<td>Hooked at least once</td>
</tr>
<tr>
<td>Not hooked</td>
</tr>
<tr>
<td>Total</td>
</tr>
</tbody>
</table>

Incidence exposed to hooking = 0.108
Incidence unexposed = 0.046
Relative Risk = 2.34 (RR = 6.05; assuming 1/2 of untagged dead fish had been hooked.)
Variance = 0.54
95% Confidence Interval: (-1.44;1.44)

Table 2. 2x2 Contingency table constructed to measure relative risk of mortality associated with hooking.
mortality was between 12% and 21%. Three of 24 (13%) Atlantic salmon were hooked. The salmon hooking mortality was zero. No brown trout were hooked during the study. The overall hooking mortality for all three species combined was between 11% and 19% (See Table 1).

Assuming the 3 untagged fish died of causes other than hooking mortality, the 2x2 contingency table measuring acute mortality relative risk demonstrates that there is a 2.34x greater chance of dying after being hooked than not being hooked. If we assume that 1/2 of the 3 untagged fish had been hooked the relative risk of death after hooking increases to 6.05x (See Table 2). If we assume all 3 untagged dead fish had been hooked the relative risk of death after hooking becomes an infinite number since there is no fish death other than hooking mortality in this study group. Ho1: Rejected.

**Fish Quality:** Fish quality parameters compared the 67 fish retaining their floy tags between the beginning and end of the study. Fish average total length increased by 5 mm. Fish average mass decreased by 17 grams. There was no meaningful change in the fish’s condition factor. There were increased injuries to the snout, jaw, and mouth. There were fewer fish with noticeable operculum shortening. There were no differences in body conditions of eyes, gills, thymus, shape, color, scales, or symmetry. Fin conditions either improved or were unchanged from the beginning of the study to the end (See Table 3).

There is no relative risk difference in gross (a) external or (b) internal pathological differences (as measured by fish quality exams) between brook trout subjected to single or multiple “catch and release” events and brook trout not subjected to “catch and release” events. (Ho2: Accepted).

**Egg Quality/Embryo Survival:** Approximately 7,000 eggs were collected from four hooked female brook trout at the conclusion of the study. Eggs were fertilized by brook trout males, water hardened, disinfected, and placed in incubation trays at the Enfield Fish Hatchery. After 24-hours of incubation approximately 95% of the eggs were opaque—indicating that the embryos were dead. Control eggs from hatchery brook trout broodfish average 15% 24-hour mortality. Ho3: Rejected.

**48-Hour Post Hooking Survival:** During the study 37 of 102 fish were hooked and released. Two died within 48-hours of the hooking event. One died after 14 days and the death of three other fish occurred without a clear timeframe between hooking and death. Given this data, it is not possible to calculate the relationship between angling and risk of death within 48-hours post angling. Ho4: Not testable. This may outline part of the difficulty in this type of experiment. It is easy to identify

<table>
<thead>
<tr>
<th><strong>Hooking Mortality Study: Fish Quality Results</strong></th>
<th>Before</th>
<th>After</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ave Length</td>
<td>390 mm.</td>
<td>395 mm.</td>
<td>Increase 5 mm</td>
</tr>
<tr>
<td>Ave Mass</td>
<td>766 gr.</td>
<td>749 gr.</td>
<td>Decrease 17 gr.</td>
</tr>
<tr>
<td>Ave C. Factor</td>
<td>1.22E-6</td>
<td>1.16E-6</td>
<td>No change.</td>
</tr>
<tr>
<td>% Affected Before Study</td>
<td>% Affected After Study</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Snout</td>
<td>2%</td>
<td>3%</td>
<td>Increase.</td>
</tr>
<tr>
<td>Jaw</td>
<td>1%</td>
<td>6%</td>
<td>Increase.</td>
</tr>
<tr>
<td>Mouth</td>
<td>1%</td>
<td>4%</td>
<td>Increase.</td>
</tr>
<tr>
<td>Eyes</td>
<td>0%</td>
<td>0%</td>
<td>No change.</td>
</tr>
<tr>
<td>Operculum</td>
<td>3%</td>
<td>0%</td>
<td>Decrease.</td>
</tr>
<tr>
<td>Gills</td>
<td>0%</td>
<td>0%</td>
<td>No change.</td>
</tr>
<tr>
<td>Thymus</td>
<td>0%</td>
<td>0%</td>
<td>No change.</td>
</tr>
<tr>
<td>Shape</td>
<td>0%</td>
<td>0%</td>
<td>No change.</td>
</tr>
<tr>
<td>Color</td>
<td>0%</td>
<td>0%</td>
<td>No change.</td>
</tr>
<tr>
<td>Scales</td>
<td>0%</td>
<td>0%</td>
<td>No change.</td>
</tr>
<tr>
<td>Symmetry</td>
<td>0%</td>
<td>0%</td>
<td>No change.</td>
</tr>
<tr>
<td>% Normal Fin Quality Before</td>
<td>% Normal Fin Quality After</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dorsal</td>
<td>81%</td>
<td>100%</td>
<td>Increase.</td>
</tr>
<tr>
<td>Pectoral (L)</td>
<td>99%</td>
<td>100%</td>
<td>Increase.</td>
</tr>
<tr>
<td>Pectoral (R)</td>
<td>100%</td>
<td>100%</td>
<td>No change.</td>
</tr>
<tr>
<td>Pelvic (L)</td>
<td>100%</td>
<td>100%</td>
<td>No change.</td>
</tr>
<tr>
<td>Pelvic (R)</td>
<td>100%</td>
<td>100%</td>
<td>No change.</td>
</tr>
<tr>
<td>Anal</td>
<td>100%</td>
<td>100%</td>
<td>No change.</td>
</tr>
<tr>
<td>Adipose</td>
<td>100%</td>
<td>100%</td>
<td>No change.</td>
</tr>
<tr>
<td>Caudal (upper)</td>
<td>100%</td>
<td>100%</td>
<td>No change.</td>
</tr>
<tr>
<td>Caudal (lower)</td>
<td>44%</td>
<td>100%</td>
<td>Increase.</td>
</tr>
</tbody>
</table>

Table 3. Fish size and quality measurements.
fish that die within 48-hours of angling. It becomes increasingly difficult to identify fish that die subsequently.

**Histology:** Tissue samples from 27 brook trout, 3 land-locked Atlantic salmon, and 3 brown trout from this study were collected, preserved in 10% buffered formalin (Stoskopf 1984), and sent to Path Lab, Inc. Portsmouth, NH for histological preparation. Hematoxylin and Eosin stained slides were prepared by Path Lab, Inc., and sent to the MDIF&W Fish Health Laboratory. The following tissues were submitted for histology: skin, muscle, liver, kidney, spleen, gonad (ovary/teste), heart, pancreas, pyloric caeca, intestine, bone and gill (Figures 3-11). Histological slides were numbered randomly by Path Lab, Inc. After slides were read, information regarding the fish’s species, sex, hooking history was added to the results for comparisons. There were no differences between hooked and not hooked fish for any tissues examined except skin. Fish that were hooked and released had an incidence of histological skin injury = 73%. Fish that were never hooked had an incidence of skin injury = 23%. The relative risk of skin injury in this study was 3.17x greater for hooked fish (Table 4). Figures 3a-d show examples of the types of skin injuries seen. Ho3; Rejected.

These fish tissues revealed many other very interesting facts regarding the overall health of these broodfish. Even though there were not differences between hooked and not hooked fish; these fish had significant histological lesions. Thirty of 33 fish examined had significant kidney disease, including shrunken and col-

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**Figure 3a.** Normal fish skin. Image from C. Horsch 99. USFWS National Conservation Training Center.

**Figure 3b.** Hypertrophy of epithelial mucosa. Image from MDIF&W Fish Health Laboratory.

**Figure 3c.** Hypertrophy of dermis. Image from MDIF&W Fish Health Laboratory.

**Figure 3d.** Ulceration of epidermis with loss of epithelial mucosa. Image from MDIF&W Fish Health Laboratory.
H0(5): Skin injury visible by histology

<table>
<thead>
<tr>
<th></th>
<th>Skin injury</th>
<th>No Skin Injury</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hooked at least once</td>
<td>11</td>
<td>4</td>
<td>15</td>
</tr>
<tr>
<td>Not hooked</td>
<td>3</td>
<td>10</td>
<td>13</td>
</tr>
<tr>
<td>Total</td>
<td>14</td>
<td>14</td>
<td>28</td>
</tr>
</tbody>
</table>

Incidence exposed to hooking = 0.73
Incidence unexposed = 0.23
Relative Risk = 3.17
Variance = 0.28
95% Confidence Interval: (-1.04; 1.04)

Table 4. 2x2 Contingency table constructed to measure relative risk of skin injury associated with hooking.
Figure 5a. Normal pancreatic tissue. Image from C. Horsch 1999, USFWS National Conservation Training Center.

Figure 5b. Pancreatic tissue from brook trout. Notice how acinar cells are replaced by fat cells leaving only island of pancreatic tissue. Image from MDIF&W Fish Health Laboratory. H&E x 200

Figure 5c. Pancreatic tissue from brook trout. Notice how acinar cells have been lost and replaced by fat cells. Image from MDIF&W Fish Health Laboratory. H&E x 40.

Figure 5d. Pancreatic tissue from brook trout. Notice how acinar cells have been lost and replaced by fat cells (>95%). Image from MDIF&W Fish Health Laboratory. H&E x 200

Figure 6a. Normal Liver tissues. Image from C. Horsch 1999, USFWS National Conservation Training Center.

Figure 6b. Liver tissue from brook trout with hepatic cellular swelling, lipid/glycogen accumulation. Image from MDIF&W Fish Health Laboratory. H&E x 100x.
Figure 7a. Normal gill filament tissues. Image from C. Horsch 1999. USFWS National Conservation Training Center.

Figure 7b. Hypertrophy of epithelial mucosa. Image from MDIF&W Fish Health Laboratory.

Figure 7c. Normal gill lamellae and filaments. Image from C. Horsch 1999. USFWS National Conservation Training Center.

Figure 7d. Lamellar hypertrophy. Image from MDIF&W Fish Health Laboratory. H&E x 40.

Figure 8a. Normal Pyloric caeca tissue. Image from C. Horsch 1999. USFWS National Conservation Training Center.

Figure 8b. Intestine from brook trout broodstock. Normal appearance, however, notice cestode located in center. Image from MDIF&W Fish Health Laboratory. H&E x 40.
Figure 9a. Normal intestinal musculature around two parasites. Image from MDIF&W Fish Health Laboratory. H&E x 100

Figure 9b. Image of parasite infecting smooth muscle of intestine. Image from MDIF&W Fish Health Laboratory. H&E 400.

Figure 10a. Normal ovarian tissue. Image from MDIF&W Fish Health Laboratory. H&E x 100.

Figure 10b. Normal testicular tissue. Image from MDIF&W Fish Health Laboratory.

Figure 11. Normal muscle tissue. Image from Chris Horsch, USFWS.

Figure 12. Ulceration of skin of landlocked Atlantic salmon. Image from MDIF&W Fish Health Laboratory.
lapsed glomeruli, nephron tubule degeneration, nephrocalcinosis, granulomas, and increased amounts of melanin (Figures 4a-4d). Twenty-nine of 33 fish had significant pancreatic lesions in which pancreatic tissues was replaced by adipocytes (Figures 5a-5d). Thirteen of 33 fish had significant histological liver disease, including hepatocellular swelling, excessive lipid and glycogen accumulation, and a loss of hepatic cellular detail (Figures 6a, and 6b). Twelve of 33 fish had significant gill disease; including lamellar hypertrophy, lamellar hemorrhage, increased mucosal thickening, and lamellar clubbing (Figure 7a-7d). One brook trout was infected with intestinal Cestodes (Figure 8b). Several fish were infected with a Microsporidian-type parasite in the intestinal muscle wall (Figure 9a-9b).

Discussion

This was an observational study. As such the goal was to establish some preliminary data regarding the possible effects of a “catch and release” fishery on brook trout. This was not meant to be an experimental study with controls for each possible variable. The results are observations from which experiments with controls can be constructed and tested.

“Catch and Release” fishing has been one tool used by fishery managers nationwide to produce “quality” angling with the logic that fish will be angled multiple times eventually living to reach a large size. Anglers seek to hook a fish, play it to submission, sometimes photograph it, unhook it and return it to the water alive. It is a fisheries technique based on the concept, “a good game fish is too valuable to be caught only once.”- Lee Wulff, 1938.

Under light to moderate angling pressure, this concept works well in managing for “quality” fishing. It is a management tool to provide better fishing in areas with heavy fishing pressure by preventing over harvest. Many angling associations support “catch and release” regulations.

Sometimes people like to equate “catch and release” angling with other “green” programs such as “Pack In:Pack Out, and “Leave no trace.” This is not exactly the same principle. Fishing by its confrontational nature is stressful and injurious to fish. That doesn’t mean that a fish cannot recover from the event and live on; however, there is some degree of mortality associated with all types of angling—including “catch and release.”

Under the best conditions (i.e., experienced anglers, barbless hooks, healthy fish, short playing times), hooking mortality can be very low. The average hooking mortality associated with all types of “catch and release” angling is 12% (Taylor and White 1992). Typically, techniques taught to improve fish survival include:

- Don’t play fish to exhaustion.
- Handle fish with wet hands, grabbing across the back and head.
- Don’t remove swallowed hooks, cut the line.
- Don’t keep fish out of the water more than 15 seconds.
- Return fish to the water head towards the current.

“Catch and Release” fishing is a management tool applied to a fish population. A fish population contains fish of all sizes from fry to adult. The effects of “catch and release angling on the population is to increase the number of fish in the older, larger fish group by not removing them. “Catch and Release” applied selectively to the larger fish group might not have the beneficial effects it has when applied to the overall population. For example: Imagine that the lake contains 100 brook trout. There are 30 fry, 20 1-year olds, 20-2 year olds, 10 3-year olds, 10 4-year olds and 5 5-year olds and 5 6-year olds (See Figure 12).

Scenario 1

A “catch and release” regulation is applied to
the lake and there is a 10% hooking mortality. If over the angling season 100 fish were caught and released 10 would die and 90 would survive. The hooking mortality distributed over the population would leave 27 fry, 18 1-year olds, 18 2-year olds, 9 3-year olds, 9 4-year olds and 9 fish in the age 5 and 6 group. If only fish in ages 4, 5, 6 can spawn there is a net loss of only 3 broodfish. The proportions of the population would remain unchanged (See Figure 12).

**Scenario 2**

A “catch and release” regulation is applied to the lake and there is a 10% hooking mortality, however, anglers selectively target broodfish by locating their spawning habitat. If over the season 100 fish were caught and released 10 would die and 90 would survive. This time since the anglers selectively angled only for fish in the spawning areas the mortality would be limited to age groups 4, 5, and 6 resulting in the removal of 50% of the reproductive population (See Figure 12). If brook trout of all age groups were equally vulnerable to angling pressure, as in Scenario 1, it would take 5-fold increase in overall fishing pressure to do as much damage to the broodstock population.

The selective angling for broodfish could devastate the population’s ability to reproduce. This in turn could have negative effects on the recruitment in subsequent generations and the population in the lake could decline. It is for this reason that this observational study looked at the both lethal and sub lethal effects of “catch and release” fishing upon broodfish.

**Hooking Mortality:** Fifty-nine percent of the male brook trout population was hooked at least once during the study; 35% of female brook trout were hooked at least once during the study. Overall, the hooking mortality for this observational study was between 11% and 19%. This resulted in a 5-9% decline in the brook trout broodstock population. Landlocked Atlantic salmon and brown trout proved to be more difficult to catch and consequently had much lower hooking mortalities. These fish were not subjected to an overly stressful angling event. The increase in hooking mortality would appear to be a consequence of their physical condition. Their performance as athletes or “sparring partners” with anglers is likely compromised by the physiological changes associated with spawning. The angling itself was only the “final straw.” Salmonid broodstock reared by MDIF&W have a predictable seasonal mortality correlated with the spawning period (See Figure 13). It is common for broodstock deaths to occur near or shortly after spawning. Broodstock often do not tolerate the stress of handling, spawning, and/or anesthesia. It is also sometimes necessary to treat the broodfish after spawning with therapeutics to reduce mortalities and remove epidermal fungal infections.

**Fish Quality:** Grossly fish quality improved during the course of this study. The change was rather minor given that fish quality was good even at the beginning of the study. Fish grew an average of 5 mm. The average weight of fish declined by 17 grams. Since fish were not feeding and some spawned, this decrease in weight would be normal. Interesting was the difference between the skin’s normal gross appearance and its appearance histologically. Just looking at the fish it was not possible to see the extent of damage their skin had withstood. What effects it might have on the

![Brook Trout Population](example-brook-trout-population.png)

**Figure 12.** Example brook trout population before and after “catch and release” angling scenarios. Population = 100 fish.
fish’s overall survival is open to discussion. Certainly, loss of their mucosal epithelium would expose them to *Saprolegnia* fungal infections and osmotic stress.

**Egg Quality/ Embryo Survival:** The ultimate goal of every broodstock operation (feral or aquaculture) is production of a good quality eggs and offspring (Figure 14a-c). Without offspring there are no future generations. Maintenance of broodstock in good condition is a major concern in salmonid aquaculture because of potentially high pre-spawning mortalities, large numbers of fish that do not mature when predicted, and poor incubation success from inferior gametes (Pennell and Barton 1996). The protection of broodfish and spawning habitats is also a significant concern to the State of Maine. The legislature mandates the responsibility of protecting and managing these areas to the Department of Inland Fisheries and Wildlife under Chapter 713 Section II §7673 Fish Spawning Areas; and Chapter 10 Significant Wildlife Habitat.

The normal period over which spawning occurs for many species of salmonids is three to four weeks. During this time fish are very susceptible to injury. Literature on incubation and early rearing survival confirms highly variable results. Briggs (1953) documented average hatchery survival to the eyed-egg stage of 77-78%. This is consistent with results typical for brook trout, lake trout, and landlocked Atlantic salmon reared by the Maine Department of Inland Fisheries & Wildlife: Fish Hatchery Division. Salmonid hatchery manuals warn culturists about the negative effects of handling broodfish. For example:

“Any technique that causes a sudden and violent contraction of body musculature can rupture blood vessels and result in internal bleeding” (Pennell and Barton 1996).

“It is essential to avoid [skin] wounds on which fungus will rapidly develop” (Leitritz and Lewis 1976).

“The plasticity of unfertilized eggs is greater than in water-hardened eggs. Therefore the membrane may be easily broken by pressure during stripping, especially in younger females which have more fragile eggs” (Winnicki and Bartel 1968)

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**Figure 13** Broodstock mortality estimates from three MDIF&W fish hatcheries. Notice the mortality peaks associated with fall spawning periods in these salmonids.

**Figure 14a.** Newly fertilized salmonid egg. Outer egg membrane removed showing germinal disk in upper right quadrant of the animal pole. *Image from Velsen (1980).*

**Figure 14b.** Salmonid parr. It is common for wild populations to have 90% survival of green eggs (above) to parr. *Image from Velsen (1980).*

Billard (1977) demonstrated that a small amount of egg yolk from broken eggs can decrease fertility by >90%. Yolk precipitates in water to form a network that traps spermatozoa and clogs the micropyle. Salmonids
unlike many other species have a single location, the micropyle, on their egg where a spermatozoa enters during fertilization.

Billard (1981, 1992) found that there are a number of intrinsic (e.g., genetic, physiological) and extrinsic (e.g., diet, holding conditions, handling) factors affecting egg fertility and survival that when optimized produce quality gametes that produce a high incidence of fertilized eggs with good survival potential. Wild salmonid eggs buried in redds can have a survival rate of over 90% and buried alevins can have similarly high survival rates (Elliot, 1984, Wertheimer 1981). Losses of fry in the first month after emergence can be very high and are density dependent (Wooton 1990, Warner 1984). In contrast, survival of >90% of green eggs to early parr stage is considered excellent fish culture.

Added to the intrinsic and extrinsic factors affecting fish egg fertility is a temporal factor. Egg fertility deteriorates with time after ovulation. The capacity of an egg to be fertilized depends upon completion of meiosis. This process begins before ovulation but the oocyte does not complete meiotic maturation until some time after fertilization. Final oocyte maturation involves dissolution of the nuclear membrane (germinal vesicle breakdown) upon resumption of meiosis, and achievement of the second meiotic metaphase stage (Mylonas et al. 1992). Only at this stage is an oocyte first considered fertilizable (Figure 14a).

Hatchery managers must routinely balance the goal of getting the best quality eggs with the risk of damaging eggs and broodstock by excessive handling. If they frequently check female broodfish for ripeness (i.e., ovulation of eggs from the ovary into the fish’s abdomen) they will get the best quality eggs. However, the handling process itself causes damage to both broodfish and unripe eggs. The trade-off typically means fish are checked for ripeness once weekly. Needham (1988) reported that appropriate stripping time for salmonids occurs in the four days following egg release from the ovary. Springate et al. (1984) reported that eggs stripped between 4 and 10 days following ovulation at 10°C consistently achieved high rates of fertilization. After 20 days, only a few eggs were capable of being fertilized and of those, few survived incubation. In contrast, Fitzpatrick et al., (1987) found the highest mean viability of coho salmon O. kisutch eggs 20 days after ovulation. Hynes et al. (1981) reported that ova may be kept up to 30 days in vivo for large rainbow trout females but only 15 days for younger females. Hynes et al. 1981 also reported that the maximum egg and fry survival are achieved if the ova are stripped four to six days after ovulation. Egg survival in dead females is variable, from as short as a few hours (Billard et al. 1981) to as much as 12 hours (Ingram 1985). Scott and Baynes (1980) reported that sperm taken from Pacific salmon between 1.5 and 5 hours after death were still completely viable. Ingram (1985) reported that sperm has been used with no loss of viability for up to 72 hours after death.

In this observational study it is not possible to precisely determine which of the above factors contributed to the poor egg survival of hooked female brook trout. While eggs appeared grossly normal, they could have been stripped from the fish and fertilized after the window of fertility had closed; it is also possible that their micropyles were obstructed by small amounts of yolk material from damaged eggs. Regardless, the effect was the same.

Some fish did attempt to spawn during the study. Several brook trout, landlocked Atlantic salmon and brown trout had gorged themselves on spawned eggs. These fish were in a cement enclosure. They were not able to prepare redds where eggs could incubate protected from other fish. It was not possible to determine the fertility of the eggs in the fish stomachs. It does raise an additional concern regarding angling during the spawning process, “Would angling cause fish to deposit fewer eggs into reds?”

Salmonids have a natural repertoire of stimuli that synchronize maturation to a time of year that maximizes
survival potential of offspring in their natural habitat (Brannon 1987). To the extent that “catch and release” angling interferes with this repertoire its effects can hardly be viewed as benign.

Histology: Pathology is the branch of medicine and biology given the task of determining whether cells (or parts of cells) are functioning properly, contributing to the proper function of associated tissues, and organs, organ systems, and ultimately whole organisms. It is the basic tenet of pathology that all disease is essentially a manifestation of cellular injury. Such injuries leads to changes in the structure and function of tissues and organs. The changes in function are what we recognize as symptoms and clinical signs. The changes in structure are what we recognize as morphological lesions. With rare exceptions, there are no new metabolic or biochemical pathways involved in disease, nor are new structures usually involved. Rather structures and functional pathways that already exist are altered, either accentuated, diminished, or lost altogether. It is the departure from normal day-to-day balance or steady state that produces disease.

The 102 fish in this study appeared grossly normal. They had some amount of external fin wear associated with living in a cement enclosure; but otherwise were clinically healthy fish. During the eight weeks of this study, they were exposed to the stresses of sexual maturation, two amass net capture events, two anesthesia events, fin tagging, and some of the fish were further exposed to “catch and release” angling. How the fish perceived the stresses of this study is not known. How their bodies reacted to the stresses can be interpreted by examining histological samples of various tissues. When the tissues appear normal, we can conclude the fish were able to function appropriately. When the tissues are abnormal, we must look at the biological processes leading to the observed biological abnormality and further decide whether it was caused by the identified stress or some additional etiology.

Figure 3a shows normal fish skin. The outer most slimy layer of a fish’s skin is composed of cells that secrete mucous. These cells are responsible for creating a physical barrier between the fish and its environment. Loss of this barrier makes the fish vulnerable to osmotic and ionic stress, and disease causing organisms. There is normally a thickening of the fish’s skin during spawning. This is especially true in males. The thicker skin gives them protection during spawning. Evidence of this dermal thickening can be seen in Figure 3c. It is not normal for there to be a loss of dermis or epidermis. It is likely that this loss is a consequence of human handling and since all fish were handled identically during the study, it is likely that angling contributed to this lesion.

Kidney tissues in these fish did not differ between angled and not angled fish. The kidney lesions in these fish are common to all state operated fish hatcheries with soft water. It is likely that this lesion is a consequence of living in soft water (i.e., water with Ca$^{2+} < 3.0$ mg/l). The extent of these lesions in naturally occurring populations of brook trout is unknown and under investigation.

Pancreatic tissues replaced by adipose “fat” cells means that these fish have a diminished capacity to digest food items during the spawning season. This is not surprising since many salmonid anglers report fish are difficult to catch during spawning. They must be triggered to strike many times with bright terminal gear. It also means that they have largely accumulated the necessary food stores for the fall and winter. If angling affects the amount of energy available for use during the winter, it could affect long-term broodstock survival.

Hepatic lesions were consistent with fish that are using stored body fats for energy. Foamy hepatocytes full of glycogen are cells typically utilizing stored fat. This mobilization of body reserves is a slow biochemical process. Domesticated mammalian species that cannot mobilize adequate energy during birth, lactation, or similar stressful peri-parturient events become ketotic and die with-
out immediate medical attention (Dryer 1994). It is possible that some of the post spawning mortality seen in multi-porous salmonids is due to diabetic ketoacidosis and diabetic shock. But this has not been researched adequately.

The fish also contained a couple of odd parasites not typically seen in hatchery fish. Why, I can’t explain. There presents makes these fish more like feral fish. Many populations of feral fish in Maine contain similar parasites. These organisms contribute stress to the fish. In fall 2000, landlocked Atlantic salmon in the Jordan River broodstock run of Sebago lake were infected with at least 3 different parasites. Parasite loads are so high, that I am concerned that they may be contributing significantly to the salmon’s declining population.

Finally, there are many factors involved in determining the effects of fall angling on salmonid broodstock. With the initial information gathered in this observational study, samples from “catch and release” feral populations can be compared. There is likely room for some fall angling for salmonids in the State of Maine. It would be irresponsible to open all waters to fall angling. There are too many unknowns, and the potential to substantially injure a self sustaining feral salmonid population is too great.

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Acknowledgement

Fall colors on Moose River, Maine.
Image from discovermaine.com. Staff photographer.

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Attach copy of data collection form.