Pathways of barotrauma in juvenile salmonids exposed to simulated hydroturbine passage: Boyle's law vs. Henry's law

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A B S T R A C T

On their seaward migration, juvenile salmonids commonly pass hydroelectric dams. Fish passing by the turbine blade may experience rapid decompression, the severity of which can be highly variable and may result in a number of barotraumas. The mechanisms of these injuries can be due to expansion of existing bubbles or gases coming out of solution; governed by Boyle’s law and Henry’s law, respectively. This paper combines re-analysis of published data with new experiments to gain a better understanding of the mechanisms of injury and mortality for fish experiencing rapid decompression associated with hydroturbine passage. From these data it appears that the majority of decompression related injuries are due to the expansion of existing bubbles in the fish, particularly the expansion and rupture of the swim bladder. This information is particularly useful for fisheries managers and turbine manufacturers, demonstrating that reducing the rate of swim bladder ruptures by reducing the frequency of occurrence and severity of rapid decompression during hydroturbine passage could reduce the rates of injury and mortality for hydroturbine passed juvenile salmonids.

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1. Introduction

During their seaward migrations in the Columbia River Basin (CRB), it is common for juvenile salmonids to pass hydroelectric dams. There are three possible routes of passage; over a spillway, through a juvenile fish bypass system or directly through the hydroturbines. In some cases, up to 100% of seaward-migrating salmonids may pass directly through hydroturbines. However, at many of the facilities in the Snake and Columbia River mainstem, less than a quarter of fish pass through hydroturbines due to the existence of bypass systems or spillways through which fish are intentionally guided.

Passage through a hydroturbine may expose fish to a variety of different forces (e.g., shear force and blade strike) that can lead to injury (Čada et al., 2006; Deng et al., 2005, 2007a, 2010). Perhaps of greatest concern are the rapid changes in pressure fish are exposed to as they pass by the turbine blade. As fish pass between the turbine runner blades, they are exposed to a sudden (occurring in <1 s) pressure decrease before returning to near surface pressure as they enter the downstream channel (see Stephenson et al., 2010, Fig. 2).

The magnitude of decompression during turbine passage is dependent upon the turbine runner design, the operation of the turbine, the submergence of the turbine runner (i.e., elevation of the turbine runner relative to the downstream water surface elevation), the total project head (difference between upstream and downstream water surface elevations) and the flow path (Čada, 1999; Deng et al., 2007b, 2010; Carlson et al., 2008). Pressures are generally higher near the front side of the turbine blade (upstream pressure side) and lower near the back side of a turbine blade (downstream suction side) and are typically, but not always, lower near the blade tips than they are in the mid-blade region (ENSR, 2008). The lowest pressure a fish may experience during turbine passage in the CRB can vary from approximately 200–0 kPa (Carlson et al., 2008; Deng et al., 2010; all pressure values presented are in absolute pressure), depending on turbine design, operation, head and passage route. However, typical nadir pressures (the lowest absolute pressures present during turbine passage) lower than 50 kPa are likely less common than those greater than 50 kPa.

The rapid decrease in pressure associated with hydroturbine passage may lead to barotraumas, which typically include ruptured swim bladder, exophthalmia, internal hemorrhaging and gas.
bubbles (emboli) in the gills and fins for juvenile Chinook salmon (Brown et al., 2009, in press). Barotrauma related injury can occur through two major pathways. The first is governed by Boyle’s law where damage occurs due to the expansion of preexisting free gas within the body of the fish, such as the swim bladder (Keniry et al., 1996; Pflugrath et al., in press). The second is governed by Henry’s law where gas may come out of solution due to the decompression induced reduction in solubility resulting in bubble formation.

Boyle’s law \( P_1 V_1 = P_2 V_2 \) (where \( P_1 \) and \( V_1 \) are the initial pressure and volume and \( P_2 \) and \( V_2 \) are the resultant pressure and volume) states that the volume of a gas is inversely proportional to the absolute pressure acting on the volume if the temperature is fixed within a closed system (Van Heuvelen, 1982). For a fish passing through a hydroturbine, if the pressure of its surroundings decreases by half, the volume of the preexisting gas in the body doubles. Stephenson et al. (2010) noted that for juvenile Chinook salmon acclimated to the pressures present at 7.62 m (175.8 kPa), their swim bladders began to rupture when the pressure was decreased by about half (i.e., 87.9 kPa) during simulated turbine passage (STP). We hypothesize that gas that escapes the ruptured swim bladder can also move throughout the body leading to emboli, hemorrhaging or exophthalmia.

Henry’s law states that the amount of gas that can be dissolved in a fluid, such as blood, is directly proportional to the partial pressure to which it is equilibrated. Thus, when the surrounding pressure is reduced, the dissolved gas may come out of solution into a gas phase, resulting in gas bubble formation, the basis for the “bends” in SCUBA divers. Similarly, as fish pass through a hydroturbine and experience decompression, gas bubbles may form in the blood (emboli). We hypothesized that there would be an inverse relationship between the magnitude of the decrease in pressure associated with simulated turbine passage and the amount of fish seen with emboli.

To further understand the processes that lead to barotrauma, we examined how two different gas laws, Boyle’s law and Henry’s law, influence the magnitude of barotraumas experienced by fish subjected to simulated turbine passage. We re-analyzed data collected in previous studies (described in Brown et al., 2009, in press; Stephenson et al., 2010) to identify the rates of barotraumas (e.g., emboli, hemorrhaging, exophthalmia) for fish with and without a swim bladder rupture resulting from exposure to rapid decompression. We conducted new experiments to further understand the force of swim bladder rupture and isolate pathways of gases within the body of fish as a result of the rupture. To isolate barotraumas resulting from gases coming out of solution during rapid decompression (due to Henry’s law), we also conducted new experiments that eliminated the rupture of the swim bladder while exposing fish to low pressure.

2. Methods

2.1. Barotraumas due to Boyle’s law

2.1.1. Re-analysis of data comparing injuries occurring in fish with and without a ruptured swim bladder following simulated turbine passage

To test the hypothesis that gas escaping the ruptured swim bladder can move throughout the body leading to emboli, hemorrhaging or exophthalmia, we re-analyzed data collected in previous studies (Stephenson et al., 2010; Brown et al., in press) and compared injuries in fish that had a ruptured swim bladder to fish that did not have a ruptured swim bladder. Detailed descriptions of the methods employed in these studies can be found in previous publications (Stephenson et al., 2010; Brown et al., in press).

Briefly, juvenile Chinook salmon (see Table 1 for fish sample size, length, weight and origin information for each phase of experimentation) used in these studies were acclimated to absolute pressures that would exist at three different depths in fresh water, given standard atmospheric pressure of about 101.4 kPa: 1.5 m, 4.6 m and 7.6 m (see Table 2 for sample sizes and acclimation and exposure conditions for each phase of experimentation). Fish were acclimated at elevated total dissolved gas (TDG) levels for 16–24 h prior to STP to allow fish ample time to attain neutral buoyancy and equilibration of gas tensions in bodily fluids and tissues. TDG is presented as a percent (the total dissolved gas pressure, or the sum of the partial pressures of each individual gas in the water, divided by the local barometric pressure). In previous research, elevated levels of TDG (>100%) were examined to determine their influence on barotrauma. Total dissolved gas saturation levels can be highly variable in the Columbia and Snake rivers, typically ranging from 100% to 120% (Columbia River DART, 2009) but have been observed as high as 143% (Ebel, 1969; Beiningen and Ebel 1970, 1971; Backman and Evans, 2002). These elevated TDG levels are typically associated with high rates of flow over spillways of hydro projects. Salmon are physostomous and could attain neutral buoyancy at these pressures by gulping air from a bubble maintained at the top of the chamber during the acclimation period (Stephenson et al., 2010).

Following acclimation, fish were exposed to pressure profiles representing passage through Kaplan turbine units typical of the Lower Snake and Columbia River hydropower projects (see Brown et al., 2009, Fig. 1 or Stephenson et al., 2010, Fig. 2). Exposure pressures (i.e., the nadir) during STP ranged from 6.4 to 114.8 kPa and the rate of pressure change ranged from 758.4 to 3874.9 kPa/s. The natural log of the ratio pressure change (LRP; acclimation to exposure [nadir] pressure) was used as the independent variable for statistical analysis, as it has been found to be predictive of the likelihood of mortality and injury for juvenile Chinook salmon exposed to STP (Brown et al., in press).

Following testing, a necropsy was performed to determine injuries that were present both externally and internally. These injuries included exophthalmia; hemorrhaging in the pericardium, liver, or kidney; ruptured swim bladder; blood or bile secretions from the vent; and emboli in the gills, or fins. When comparing emboli between fish with and without swim bladder rupture, we compared fish with emboli in the gills, kidney, heart or pelvic fins to those without. When comparing hemorrhaging between fish with and without swim bladder rupture, we compared fish with hemorrhaging in the pericardium, liver or kidney to those without.

2.1.2. Visual evidence of gas from swim bladder rupture traveling throughout the body

To further examine the pathways of barotrauma, X-ray images of juvenile Chinook salmon were taken between June 14 and 16, 2011. Prior to imaging, fish were acclimated to the pressures present at either 4.6 m or 40.7 m for 16–24 h. Following acclimation, fish were exposed to a rapid decompression with the lowest pressures (nadir) ranging from 12.4 to 76.5 kPa. Following exposure, fish were removed from chambers and placed on an X-ray imager. Following imaging, fish were necropsied as described previously.

2.1.3. Barotrauma due to rapid decompression without gas supersaturation

We hypothesized that emboli, hemorrhaging and exophthalmia could be linked to gas escaping from a ruptured swim bladder instead of gas coming out of saturation in the blood. To test this hypothesis, we rapidly decompressed juvenile steelhead (Oncorhynchus mykiss) from a fairly high pressure (510.1 kPa) to 117.2 kPa on July 12, 2011. Juvenile steelhead were used since, during pilot scale investigations, they were able to become...
neutral buoyancy when held at 510.1 kPa (40.7 m of depth) while juvenile Chinook salmon were not. We speculated that this additional amount of gas in the swim bladder could lead to a higher degree of barotraumas during decompression.

The pressure exposure started by acclimating juvenile steelhead to the pressure present at 40.7 m of depth. The pressure in the chambers was then reduced from this pressure to 117.2 kPa (the pressure present at 1.6 m of depth) in less than 1 s. Fish were then returned to surface pressure over 0.5 min. Following the pressure exposure, fish were euthanized using a solution of MS-222 (250 mg/L) and were necropsied as described above.

2.2. Barotraumas due to Henry’s law

### 2.2.1. Sustained decompression

To further understand the role that Henry’s law may play in barotrauma, juvenile Chinook salmon were exposed to low pressures for an extended period of time. During hydroturbine passage, fish spend less than a second below surface pressure. To determine how long it would take for bubbles to come out of suspension in the blood (governed by Henry’s law) and cause injury and mortality, we exposed fish to pressures below surface pressure for several minutes. Testing was conducted between August 2 and 4, 2011.

Fish were acclimated to the pressure present at 4.6 m of depth for 16–24 h (similar to Brown et al., in press). After acclimation, the pressure in the chambers was reduced to almost vapor pressure (13.8 kPa) over a period of 2.9–3.6 min (median = 3.3 min). The slow decompression allowed fish to expel gases from the swim bladder via the pneumatic duct, eliminating the rupture of the swim bladder as a source of barotrauma. Fish were then maintained at this low pressure for several minutes until all fish floated to the surface at which time the experiment was terminated, fish were euthanized with MS-222 (250 mg/L) and necropsied as indicated above. The time (in minutes) for each fish to float to the surface was determined by examining video of the exposure.

### 2.2.2. Slow decompression and repressurization

Another series of tests were conducted on July 27, 2011 to determine the effect of slowly decompressing juvenile Chinook salmon to a low pressure and then instantly returning them to surface pressure. This treatment was conducted to distinguish between the sources of barotraumas for fish exposed to low pressures for very short periods of time, as in rapid decompression.

The pressure exposure started by acclimating juvenile Chinook salmon to the pressure present at 4.6 m depth for 16–24 h (similar to Brown et al., in press). Following acclimation, the pressure was reduced to 13.8 kPa over 2.2–2.4 min (median = 2.3 min), which allowed gases from the swim bladder to be expelled via the pneumatic duct. Fish were then returned to surface pressure in 0.1 min. Following the pressure exposure, fish were euthanized using a solution of MS-222 (250 mg/L) and were necropsied as described above.

### 2.3. Statistical analysis

The probability of injury (i.e., hemorrhaging, emboli, exophthalmia) occurring in fish with and without a ruptured swim bladder was modeled using generalized linear models based on a logistic link function with a binomial error structure. The number of fish with embolism, hemorrhaging, or exophthalmia present and absent for each pressure trial (7 fish in each chamber) for fish with and without a ruptured swim bladder served as the binomial response variable. LR and swim bladder rupture were continuous and binary covariates, respectively. Statistical analysis was conducted using the computing program R and significance was assessed at $\alpha < 0.05$.

### 3. Results

#### 3.1. Barotraumas due to Boyle’s law

#### 3.1.1. Re-analysis of data comparing injuries occurring in fish with and without a ruptured swim bladder following simulated turbine passage

We hypothesized that gas that escapes from the ruptured swim bladder can move throughout the body leading to emboli, hemorrhaging or exophthalmia. Among fish exposed to STP, those with a ruptured swim bladder had a significantly ($P < 0.0001$ in each case) higher likelihood of emboli in their gills, kidney or heart (Fig. 1), hemorrhaging in the pericardium, kidney or liver (Fig. 2), or exophthalmia (Fig. 3) than fish that did not have a ruptured swim bladder. This provides evidence that gas from ruptured swim

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### Table 1

Summary of the species, sample size, hatchery source, and size of fish used in different experimental sections described in this article.

<table>
<thead>
<tr>
<th>Section</th>
<th>Species</th>
<th>N</th>
<th>Hatchery source</th>
<th>Fork length (mm)</th>
<th>Mass (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.1.1</td>
<td>CS</td>
<td>5713</td>
<td>Priest Rapids, Leavenworth</td>
<td>122 (71–205)</td>
<td>19.3 (3.7–134)</td>
</tr>
<tr>
<td>2.1.2</td>
<td>CS</td>
<td>11</td>
<td>Priest Rapids</td>
<td>91 (81–103)</td>
<td>8.1 (5.5–11.6)</td>
</tr>
<tr>
<td>2.1.3</td>
<td>ST</td>
<td>6</td>
<td>Lyons Ferry</td>
<td>227 (213–252)</td>
<td>125.5 (109.3–165.5)</td>
</tr>
<tr>
<td>2.2.1</td>
<td>CS</td>
<td>23</td>
<td>Priest Rapids</td>
<td>107 (88–126)</td>
<td>11.4 (8.3–21.5)</td>
</tr>
<tr>
<td>2.2.2</td>
<td>CS</td>
<td>20</td>
<td>Priest Rapids</td>
<td>103 (93–114)</td>
<td>10.9 (6.7–15.0)</td>
</tr>
</tbody>
</table>

CS = Chinook salmon and ST = steelhead.

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### Table 2

Summary of the species, sample sizes, total dissolved gas (TDG) levels, acclimation pressures, exposure pressures and rate of pressure decrease used in experimental sections outlined in this article.

<table>
<thead>
<tr>
<th>Section</th>
<th>Species</th>
<th>N</th>
<th>TDG (%)</th>
<th>Acclimation pressures (kPa)</th>
<th>Exposure pressure (kPa)</th>
<th>Rate of pressure decrease (kPa/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.1.1</td>
<td>CS</td>
<td>1193</td>
<td>112.7–127.5</td>
<td>116.5</td>
<td>44.1 (6.4–114.8)</td>
<td>1399.6 (758.4–3874.9)</td>
</tr>
<tr>
<td>2.1.1</td>
<td>CS</td>
<td>3786</td>
<td>112.7–127.5</td>
<td>146.2</td>
<td>44.1 (6.4–114.8)</td>
<td>1399.6 (758.4–3874.9)</td>
</tr>
<tr>
<td>2.1.1</td>
<td>CS</td>
<td>734</td>
<td>112.7–127.5</td>
<td>175.8</td>
<td>44.1 (6.4–114.8)</td>
<td>1399.6 (758.4–3874.9)</td>
</tr>
<tr>
<td>2.1.2</td>
<td>CS</td>
<td>10</td>
<td>140</td>
<td>146.1</td>
<td>31.2 (12.4–51.0)</td>
<td>1383.3 (1145.5–1519.5)</td>
</tr>
<tr>
<td>2.1.2</td>
<td>CS</td>
<td>1</td>
<td>140</td>
<td>510.1</td>
<td>76.5</td>
<td>1951.3</td>
</tr>
<tr>
<td>2.1.3</td>
<td>ST</td>
<td>6</td>
<td>103</td>
<td>510.1</td>
<td>117.2</td>
<td>&gt;392.9</td>
</tr>
<tr>
<td>2.2.1</td>
<td>CS</td>
<td>23</td>
<td>104</td>
<td>146.2</td>
<td>13.8</td>
<td>0.61–0.76</td>
</tr>
<tr>
<td>2.2.2</td>
<td>CS</td>
<td>20</td>
<td>104</td>
<td>146.2</td>
<td>13.8</td>
<td>0.91–1.00</td>
</tr>
</tbody>
</table>
bladders travels throughout the body of the fish leading to emboli, hemorrhaging and exophthalmia.

The equation for predicting emboli in juvenile Chinook salmon given LRP and the rupture of the swim bladder is:

$$\text{Probability of emboli} = \frac{e^{−5.68 + 1.78 \times \text{LRP} + 2.86 \times \text{Rupture}}}{1 + e^{−5.68 + 1.78 \times \text{LRP} + 2.86 \times \text{Rupture}}}$$  \hspace{1cm} (1)$$

The equation for predicting hemorrhaging in juvenile Chinook salmon given LRP and the rupture of the swim bladder is:

$$\text{Probability of hemorrhage} = \frac{e^{−6.83 + 2.24 \times \text{LRP} + 1.73 \times \text{Rupture}}}{1 + e^{−6.83 + 2.24 \times \text{LRP} + 1.73 \times \text{Rupture}}}$$  \hspace{1cm} (2)$$

The equation for predicting exophthalmia in juvenile Chinook salmon given LRP and the rupture of the swim bladder is:

$$\text{Probability of exophthalmia} = \frac{e^{−8.50 + 2.08 \times \text{LRP} + 1.50 \times \text{Rupture}}}{1 + e^{−8.50 + 2.08 \times \text{LRP} + 1.50 \times \text{Rupture}}}$$  \hspace{1cm} (3)$$

For each of these equations, rupture is either a 1 (for fish that did have a ruptured swim bladder) or a 0 (for fish that did not have a ruptured swim bladder).

### 3.1.2. Visual evidence of gas from swim bladder rupture traveling throughout the body

Further evidence of gas from ruptured swim bladders traveling throughout the body and leading to emboli, hemorrhaging and exophthalmia was noted in X-ray images of fish exposed to rapid decompression. Elongated “trails” of bubbles were visible between the body cavity and the dorsal fin of a juvenile Chinook salmon that was rapidly decompressed (Fig. 4). Images of a Chinook salmon that was not exposed to rapid decompression are provided for comparison (Fig. 5). The fish exposed to rapid decompression also had bubbles that were visible in both an X-ray and in photographs (Fig. 6).
3.1.3. Barotrauma due to rapid decompression without gas supersaturation

Large amounts of emboli, hemorrhaging and exophthalmia were observed among 6 juvenile steelhead that were rapidly (<1 s) decompressed from 510.1 to 117.2 kPa. Five of the six fish (83%) died as a result of the exposure. The surviving fish was observed expelling gas from its swim bladder during decompression and did not have any injuries. Among the five fish that died, all had a ruptured swim bladder (N = 5; 100%) and renal emboli (N = 5; 100%), while some had emboli in the gills (N = 4; 80%) or fins (pectoral or pelvic; N = 4; 50%) or exophthalmia (N = 2; 40%; Table 3).

3.2. Barotraumas due to Henry’s law

3.2.1. Solubility and emboli presence

We hypothesized that there would be an inverse relationship between the decrease in pressure and the amount of fish seen with emboli (as the change in gas pressure increases, solubility decreases and emboli form). As hypothesized, among fish that did not have a ruptured swim bladder, although the occurrence of emboli in the gills, kidney or heart was typically low, it increased as the change in total gas pressure (ΔP) increased (Fig. 7). The equation for predicting the probability of embolism to juvenile Chinook salmon given the change in total gas pressure is:

\[
\text{Probability of embolism} = \frac{e^{-8.7985 + 0.0087 \times \Delta P}}{1 + e^{-8.7985 + 0.0087 \times \Delta P}}
\]

This provides further evidence that barotrauma injuries commonly seen following rapid decompression are likely most often due to the rupturing of the swim bladder. However, little is known about circumstances that may lead to fish having emboli form when gas comes out of solution in the blood.

3.2.2. Sustained decompression

To further understand the time scale over which gas comes out of solution in the blood, juvenile Chinook salmon were exposed to low pressure for a prolonged period of time. Mortality occurred after a median of 3.0 min (range = 2.2–7.0 min) of exposure at low pressure. All of the fish had large amounts of emboli in the fins, but none had signs of exophthalmia. Several of the fish had emboli in the heart (N = 20; 83%), caudal vein (N = 8; 33%), gills (N = 5; 21%) or

Table 3

Summary of injuries observed in juvenile Chinook salmon exposed to rapid decompression without supersaturation (injuries governed by Boyle’s law) and sustained decompression (injuries governed by Henry’s law).

<table>
<thead>
<tr>
<th>Injury</th>
<th>Location</th>
<th>Rapid decompression without supersaturation</th>
<th>Sustained decompression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Swim bladder rupture</td>
<td>Heart</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Kidney</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Gills</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Fins</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Hemorrhaging</td>
<td>Heart</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Kidney</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Gills</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Fins</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Emboli</td>
<td>Heart</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Caudal vein</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Kidney</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Gills</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Fins</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Exophthalmia</td>
<td></td>
<td>X</td>
<td></td>
</tr>
</tbody>
</table>
kidney \( (N=1; 4\% ; \text{Table 3}) \). Some had hemorrhaging in the gills \( (N=1; 4\% ) \), heart \( (N=2; 8\% ) \), liver \( (N=1; 4\% ) \) or fins (pectoral or pelvic; \( N=6; 25\% ) \). None of the fish had a ruptured swim bladder since they were decompressed slowly relative to the STP treatments.

3.2.3. Slow decompression and repressurization

No mortalities were observed for juvenile Chinook salmon exposed to slow decompression and an immediate return to surface pressure. In addition, there were no injuries observed in these fishes as a result of the exposure.

4. Discussion

From the series of experiments and analysis of data in the published literature, it appears that the majority of barotrauma injuries in rapidly decompressed juvenile salmonids are associated with the expansion and rupture of the swim bladder (Brown et al., 2009, in press; Stephenson et al., 2010). This is illustrated by the higher levels of emboli, hemorrhaging and exophthalmia present among fish with a ruptured swim bladder than those without swim bladder rupture. The X-ray images that illustrate trails of bubbles within the fish also strengthen this finding. In addition, we noted emboli, hemorrhaging and exophthalmia in fish that were rapidly decompressed to a pressure above surface pressure (from 510.1 to 117.2 kPa). Since these fish were acclimated to about 100% TDG and were not decompressed below surface pressure, the \( \Delta P \) would be 0, and the emboli, hemorrhaging and exophthalmia would have had to originate from gas already present in those tissues or more likely from the ruptured swim bladder, however this needs to be investigated in more detail. These findings taken together indicate that injuries due to the expansion of preexisting gas within the body, governed by Boyle’s law, are likely the main pathway of barotrauma injuries among fish exposed to rapid decompression associated with simulated turbine passage.

The findings of this study indicate that gas coming out of suspension in the blood of fish, governed by Henry’s law, may be too slow to be a significant cause of barotrauma during STP. For juvenile Chinook salmon slowly decompressed and held at low pressures, loss of equilibrium occurred in a median time of 3.0 min by which extensive emboli were observed. While several minutes is very long in relation to the duration that fish would experience during rapid decompression associated with hydroturbine passage, further studies to characterize the time course for bubble formation associated with Henry’s law and latent effects on both lethal and sub-lethal endpoints are required to understand the degree to which this pathway may be involved in barotraumas during STP. Given that the low nadirs associated with hydroturbine passage are experienced in the order of <1 s, it is unlikely to be a major route for barotraumas. Thus, expansion and rupture of the swim bladder should be a major focus of fisheries managers trying to avoid barotrauma among fish exposed to rapid decompression. This is the first research which indicates that hemorrhaging, emboli and exophthalmia are highly associated with gas coming from the ruptured swim bladder rather than gas coming out of solution in the blood or tissues.

The force that can be present when the swim bladder ruptures during STP can be substantial. This can be illustrated by an incident that occurred during research conducted by Brown et al. (2009). A juvenile Chinook salmon gastrically implanted with a radiotransmitter (NTC-M-2, 0.42 g in air, Lotek Wireless, Inc., Newmarket, Ontario) was acclimated to the pressure present at 6.1 m of depth \( (161.0 \text{kPa}) \) and then rapidly decompressed to 11.0 kPa (ratio of pressure change = 14.6; \( \text{LRP} = 2.7) \). As a result of rapid decompression, the implanted transmitter was observed to be projected out of the mouth of the fish and video analysis determined that the transmitter was moving at a velocity of 4.5 m/s. The force of gas
moving throughout the fish was also indicated by the presence of gas throughout the body as illustrated in X-ray images. However, the damage to the fish may be highly variable depending on the amount that the gas in the swim bladder expands before and during rupture. The swim bladder of fish exposed to a higher ratio of pressure change will expand to a larger volume, leading to a higher likelihood of damage.

In contrast, some fish that have been rapidly decompressed have a ruptured swim bladder but no other injuries. Since the swim bladder of salmonids can heal within two weeks of rupture (Bellgaph et al., 2008), the rupturing of the swim bladder may not always be a source of direct mortality. However, the gas from a ruptured swim bladder could remain in the body cavity of fish following swim bladder rupture. Fish may not be able to expel the gas from the body cavity through the pneumatic duct when the swim bladder is ruptured, possibly leading to an inability to control buoyancy. Fish that are positively buoyant at the water surface would likely have an increased likelihood of being predated upon.

The pressure change needed for the swim bladder to rupture in physostomous fish is variable due to the fact that they can expel gas from their swim bladder through their pneumatic duct. A fish that expels gas from its swim bladder during decompression will be less likely to sustain injuries or mortality than one that does not. Similarly, juvenile Chinook salmon that are neutrally buoyant prior to rapid decompression have been shown to be more likely to sustain injuries or die following rapid decompression than those that are negatively buoyant (Stephenson et al., 2010). Stephenson et al. (2010) indicated that the swim bladder of juvenile Chinook salmon may need to at least double in size before it ruptures. This could happen with a fairly high pressure nadir (pressures close to surface pressure) during hydroturbine passage for fish that are neutrally buoyant at greater depths. For example, the swim bladder in a fish acclimated to 6.7 m of depth (165.4 kPa) would double in size and possibly rupture with a pressure nadir of only 82.7 kPa. The range of pressure changes that may result in swim bladder rupture may be less variable for physiostilous fish (due to the lack of pneumatic duct), however this has yet to be thoroughly investigated.

Although the majority of the barotrauma related injuries appear related to swim bladder rupture, some may be associated with compression caused by expansion of the swim bladder prior to rupture. The swim bladder can expand to the extent that it does not rupture but does still compress the internal organs enough to cause injury. Compression likely led to the presence of exophthalmia observed in fish subjected to the STP that did not possess a ruptured swim bladder (indicated in Fig. 3). Barotrauma related injuries associated with compression due to swim bladder inflation have been observed during angling or commercial fishing studies where the swim bladder is often not ruptured but barotrauma injuries are severe (Rummer and Bennett, 2005).

Commonly, fish passing through hydroelectric facilities have telemetry tags surgically implanted in the coelom to assess routes of passage and survival (McMichael et al., 2010). The expansion of the swim bladder during rapid decompression may be limited due to the presence of these tags and compression related injuries may increase in severity and potentially bias the results of survival studies (Carlson et al., in press). Deng et al. (2012) have designed a neutrally buoyant, externally attached transmitter that shows promise for assessing survival for hydroturbine passed fish (Brown et al., 2012). The attachment of the tag external to the body cavity would not influence the amount of available space for the expansion of the swim bladder during rapid decompression and warrants further investigation.

5. Future studies

Assuming that the majority of barotrauma injuries are associated with swim bladder rupture or expansion, further studies on how the rate and magnitude of pressure changes may reduce barotrauma should be conducted. For example, as demonstrated here for the first time, physostomous fish, like juvenile Chinook salmon exposed to a slower pressure change are able to expel gases via the pneumatic duct and prevent swim bladder expansion and rupture. Once thresholds are determined indicating how the rate and magnitude of pressure changes may reduce barotraumas, this information could be used to modify the design of turbines to minimize impact on seaward-migrating fish. Additionally, this information could also reduce injury and mortality of fish experiencing pressure changes when passing through bypass facilities at hydropower dams or at pump storage facilities.

Given that most of the barotraumas observed during rapid decompression were associated with the expansion and rupture of the swim bladder, a fish lacking a swim bladder, such as lamprey (genus Lampetra), may be expected to exhibit lower levels of barotrauma following rapid decompression associated with hydroturbine passage. Populations of Pacific lamprey (Entosphenus tridentatus) have declined in the CRB, and passage at hydroelectric dams has been identified as a significant factor (Schilt, 2007). There is a lack of knowledge on how lamprey respond to rapid decompression associated with hydroturbine passage and this is an area of research that clearly requires attention.

6. Conclusions and recommendations

The results of the current study indicate that injury and mortalities observed due to rapid decompression associated with STP are largely due to the rupture of the swim bladder (as governed by Boyle’s law) and are not likely due to gases coming out of solution in the blood and tissue (as governed by Henry’s law). The force of the swim bladder rupture may push gases into the surrounding musculature and result in exophthalmia and emboli and hemorrhaging in the fins and surrounding tissues. Future research should focus on identifying the rate of the ratio of pressure change needed by physostomus fishes, such as salmonids, to expel gases from their swim bladder during decompression without swim bladder rupture. It is also important to understand how physiostilous fishes, and those lacking a swim bladder, such as lamprey, may be influenced by rapid decompression associate with STP. We also recommend further research identifying the state of buoyancy of fishes prior to hydroturbine passage and the maximum depths that fish can become neutrally buoyant in the water column.

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References


