Notes

Temperature as a risk factor for outbreaks of Amoebic Gill Disease in farmed Atlantic salmon (Salmo salar)

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Abstract

Amoebic Gill Disease (AGD) outbreaks in Atlantic salmon have recently occurred below the lower temperature limit previously recognised for Neoparamoeba pemaquidensis. This observation challenges the role of ambient water temperature as one of the prime risk factors for AGD.

Amoebic Gill Disease affects cultured salmonids in Australia (Munday et al. 1990, Clark and Nowak 1999), Ireland (Rodger and McArdle 1996, Palmer et al. 1997), France (Carson pers. comm.), Spain (Carson pers. comm.), Chile (Groman and Bustos pers. comm.) and the USA (Kent et al. 1988). In Tasmania Australia, AGD is the prime health concern affecting Atlantic salmon culture (Roubal et al. 1989, Munday et al. 1990, Nowak 2001). Temperature has been identified previously as one of the main risk factors for AGD (Nowak 2001) and in Tasmania, this environmental factor is considered second only to salinity as a significant environmental factor affecting AGD outbreaks (Clark and Nowak 1999). Clinical AGD was documented in Atlantic salmon at temperature ranges from 15 to 20°C in Tasmania (Munday et al. 1990) and from 12 to 21°C in Ireland (Rodger and McArdle 1996, Palmer et al. 1997). During a histological survey of salmon cultured in Tasmania AGD lesions were recorded at the minimum temperature of 10.6°C (Clark and Nowak 1999). Amoebae were observed on the gills of cultured Atlantic salmon in winter in Tasmania (Munday 1990, Howard and Carson 1993), with no attendant clinical disease or histological lesion. Experimental tank infections suggested that temperatures above 16°C drastically increased fish mortalities and that temperature below 13°C precluded mortalities (D. Zilberg pers. comm.). The optimum temperature for in vitro culture of Neoparamoeba pemaquidensis was 15°C (Kent et al. 1988). This indicated that temperature
played an important role in AGD outbreaks which were reported to occur only at higher water temperatures.

Recently we observed AGD outbreaks and AGD associated mortalities requiring treatment at lower temperatures than previously expected. These cases took place both in the Northern (USA) and Southern (Australia) hemispheres. Mortalities of Atlantic salmon, at a net pen facility in Puget Sound, Washington State, USA were observed at temperatures sustained below 10°C. The mortalities occurred from September through to November when the mean water temperature was 9.2°C (Table 1). The presence of *Neoparamoeba pemaquidensis* was confirmed by species-specific polyclonal antibodies employed in an indirect IFAT (Howard and Carson 1993) of branchial sections of the gills from moribound fish.

While AGD is seen as a summer problem in Atlantic salmon cultured in Tasmania (Clark and Nowak 1999), recent winter outbreaks of clinical disease have been observed. Fifty percent of out of season smolt (100-220 g) exhibited variable (light or greater) AGD infection (as determined by gross gill checks and confirmed by IFAT on gill smears) approximately 3 months after transfers to sea water. This outbreak occurred when the average water temperatures were about 10°C (maximum 13°C) and the stock required fresh water treatment to limit mortality (M. Hortle pers. comm.).

Isolates of *Neoparamoeba pemaquidensis* from outbreaks in Tasmania, Ireland and USA were shown to have near identical DNA sequence for the 18S rDNA gene (F. Wong and N. Elliott pers.comm.). This finding supports previous morphological and immunological observations that the same species (*Neoparamoeba pemaquidensis*) is responsible for AGD outbreaks worldwide (Wong and Elliott pers.comm.). Thus, temperature differences during outbreaks between Washington State and Tasmania or Ireland cannot be explained simply by inherent species-specific differences and potential alterations in optimal temperature for isolation of this pathogen from disparate geographical locations should be further examined. Furthermore, Kent et al (1988) suggested that factors other than temperature such as abundance of food organism in the

<table>
<thead>
<tr>
<th>Month</th>
<th>Mortality (%)</th>
<th>Cumulative mortality (%)</th>
<th>Water temperature (°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>August</td>
<td>0.02</td>
<td>0.02</td>
<td>10.3</td>
</tr>
<tr>
<td>September</td>
<td>10.87</td>
<td>10.89</td>
<td>9.9</td>
</tr>
<tr>
<td>October</td>
<td>21.85</td>
<td>32.75</td>
<td>9.2</td>
</tr>
<tr>
<td>November</td>
<td>5.31</td>
<td>38.05</td>
<td>9.1</td>
</tr>
<tr>
<td>December</td>
<td>0.15</td>
<td>38.21</td>
<td>8.9</td>
</tr>
</tbody>
</table>

Table 1. Mortalities and water temperature at affected salmon farm in Washington state, USA.
water, may cause bloom of *Neoparamoeba pemaquidensis* and result in a disease outbreak.

The epizootiological observations described here question the role of high temperature as one of the main environmental risk factors with epizootics and reveal that clinical disease can occur at temperatures below 10°C. The potential role of other stressors predisposing the fish to AGD outbreaks and environmental factors favouring the pathogen should be further investigated.

We would like to thank salmon farms for providing information, in particular Mr M. Hortle, Tassal Ltd. We are grateful to Dr F. Wong and Dr N. Elliott, CSIRO Marine Research for the information on DNA sequencing of different *Neoparamoeba pemaquidensis* isolates. MH was supported by funding from Cooperative Research Centre for Aquaculture.

**References**


