Fisheries and Oceans | Pêches et Océans

MEMORANDUM **NOTE DE SERVICE**

To / À: Rick Stitt, Doug Lofthouse
Weaver Creek Spawning Channel

From / De: Pacific Biological Station
250-756-7057

Security Classification - Classification de sécurité
UNCLASSIFIED
Our file - Notre référence
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Your File - Votre référence
Date
Dec 21, 2009

Subject / Object: **PSM LOSS INVESTIGATION OCT 29, 2009 SUMMARY REPORT**

Oct 29 a team of 4 from PBS travelled to Weaver Creek Spawning Channel due to increasing levels of prespawning mortality in late run fish.

Gross clinical findings:
Only moribund fish were examined. 8 males (4 partially spawned; 4 ripe) and 12 females (9 unspawned; 3 spawned). Gill damage and enlarged spleens were the most common gross findings. White nodules (suspected granulomas) were seen in the livers of 2 fish; one fish appeared to have vascular congestion; one fish appeared pale (suspected anaemia)

Blood work:
Haematocrit tubes were filled from gill slits and spun within 30 minutes for Packed Cell Volume (PCV or Hct) evaluation. 2 samples were discarded since blood coagulation had begun; a third sample was lost as the tube broken during centrifugation.
The 17 samples ranged between 8 – 52; (normal range 30 – 50). 4 fish were considered anaemic (PCV < 20).

Microscopy:
Blood smears – stained with methylene blue
- No abnormal findings 14 or 20
- Small paired bacteria and/or fusiform bacteria 4 of 20
- *Cryptobia* 2 of 20

Kidney gram smears -
- No abnormal findings (5 of 20)
- *Parvicapsula* sp. (15 of 20 mild)
- *Cryptobia* (1 of 20 mild)
- Mixed Gram negative rods (1 of 20 heavy)

Canada

Bacteriology:
Note: Kidney smears were plated on TSA only. The growth media for *Flavobacterium* was not used, so the risk of vertical transmission of this pathogen was not examined.

No growth (3 of 19)
Growth of non significant/non fish pathogenic bacteria (15 of 20)
Brown pigment producing colonies (3 of 20) – confirmed as *Aeromonas salmonicida,* causative bacteria of furunculosis by antibody slide agglutination

Virology:
Spleen kidney and pyloric caeca samples were cultured on EPC and CHSE-214 cell lines.
The samples from fish 1 – 4 were mistakenly pooled into a single sample. All other fish were run as individual pools. The 16 single fish pools were negative for virus by cell culture; however, the 4 fish pool was positive for virus (confirmed as IHNV). The molecular group has the original samples and will be running these at some point to determine which of the 4 fish were positive.

Histology:
Gill pathology: The gills of every fish were compromised to some degree. All 20 fish had mixed bacterial infections of the gills; 14 of 20 had moderate to heavy bacterial loads on the gills. Half the fish gills examined had large areas of fungal damage. 2 of 20 fish had a severe inflammatory response (epithelial cell hyperplasia) with no inciting agent identified. Parasites noted in the gills included: *Parvicapsula* sp. (13 of 20 – mild to moderate); *Ichthyophtheirus* sp (8 of 20 - mild to moderate); unidentified myxosporean (2 of 20); *Loma* sp (1 of 20 – moderate)

Kidney pathology: *Parvicapsula* sp. was seen in the kidneys of every fish (at heavy levels in 18 of 20). Despite this infection, there was no evidence of an inflammatory response to the parasites and the clinical relevance of this infection is unknown. Additional parasites seen in the kidneys were: *Loma* sp (1 of 20) and a myxosporean, possibly *Sphaerospora* sp. (1 of 20).

Heart pathology: 2 of 20 hearts had moderate Loma infections. 1 of the 3 fish that had a systemic *A. salmonicida* infection demonstrated by bacteriology, also had consistently shaped large colonies of bacteria, presumed to be *A salmonicida*, in the heart; further demonstrating the systemic nature of this disease in this fish.

Liver and spleen pathology: findings were consistent with senescence. No pathogens were seen.

Brain pathology: 5 of 20 fish had light to moderate numbers of a myxosporean parasite within the brain stem (suspected to be *Myxobolus arcticus*), however there was no inflammatory reaction to the parasites and their clinical relevance is unknown.

Interpretation:
The findings were somewhat expected. No single pathogen or disease was identified to attribute pre-spawning loss to.

- *Parvicapsula* was the most consistent pathogen found, present in all fish, but the fish appear to be able to osmoregulate despite having heavily infected kidneys and the kidney interstitium appears normal so there is no direct evidence that this infection is harming the fish.
- Furunculosis was seen in 3 of 20 fish; this disease is common at low prevalence in most spawning stocks and is expected to contribute to pre-spawning losses in the similarly low

proportion of the population as a whole. The fish with systemic furunculosis were probably dying because of this bacteria, especially the one with large areas of the heart involved.

- Despite heavy bacterial loads on the gills of most of the fish, only two fish had a severe inflammatory response, suggesting that immune functions of the group have declined. The levels of gill compromise seen from all factors (bacterial, parasites and fungus) would definitely have a role in the pre-spawning losses, although I can't say how much of a role.
- It is not uncommon to finds the parasite in the brains when it's looked for. It's considered an incidental finding due to the lack of an inflammatory reponse to its presence. One paper has indicated that experimental infections with it can lower swimming performance of smolts, but no histology was done to examine load of infection or signs of inflammation, so it's hard to guess if it's effecting fish survival or not. We don't have a lot of historical information on this parasite as brain samples have not routinely been examined by our lab. But we'll look from now on.
- Other pathogens Cryptobia, Ich, Loma, Sphaerospora, are expected in spawning Sockeye at low prevalence rates and are normally considered to be incidental findings with low pathogen city – which was consistent with what we observed.

Senescent fish have a lowered immune reponse and pathogens that are normally not seen in high numbers at other lifestages can increase and make interpretation challenging. However, despite finding everything but the kitchen sink, there's no smoking gun. None of the pathogens seen, or proportion of fish they were seen in, or severity of infection they were seen in were unexpected. Infectious diseases and parasitism will be playing a contributory role to the pre-spawning losses, but there is no evidence that the losses are consistently due to disease or a disease agent.

MacWilliams