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Effect of pancreas disease (PD) on quality attributes of raw and smoked fillets of Atlantic salmon (*Salmo salar* L.)

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ABSTRACT

The impact of pancreas disease (PD) on fillet quality of raw and cold-smoked Atlantic salmon was investigated. Commercially reared fish were sorted into six groups: (1) Control (healthy fish), (2) SAV (infection with salmonid alphavirus, without PD outbreak), (3) PD0 (PD diagnosis at slaughter), (4) PD6 and (5) PD12 (diagnosed 5-7 and 11-12 months before slaughter, respectively) and (6) PDchronic (repeated PD outbreaks). The condition factor (CF) and fillet protein content were significantly higher for the control group (1.13 and 22.1%, respectively). The CF was lowest for PDchronic (0.92), whereas the fillet protein content was lowest in PD0 (20.2%). Fillet fat content did not vary significantly between the groups, but the muscle pH was 0.2 units higher in PD12 as compared to Control. Astaxanthin (Ax) and idoxanthin (Ix) content were significantly lowest for PDO. Ax recovered six months after the outbreak, but the Ix content remained lower in the PD affected salmon. The Ax level after smoking was similar for all groups, but Ix showed a similar pattern to that of raw fillets. Results of the colorimetric analyses (L^*, a^*, b^*) indicated darkest colour for the control group and palest colour for PDO, whereas PDchronic showed highest differences between raw and smoked fillets. Firmness of raw fillets was lowest in PDchronic, but after smoking a significantly higher firmness was found in PDchronic, PD0 and PD6 (16.7–19.7 N) compared with that of Control and PD12 (14.1 N). Changes in fillet quality in the order of their appearance were decreased CF, depleted muscle glycogen, increased drip loss of raw muscle, paler colour, depleted protein and finally harder texture in smoked salmon. It is concluded that the fillet quality deteriorated after a PD outbreak, but the quality may to a large extent recover.

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1. Introduction

In Norway, pancreas disease (PD) is caused by the salmonid alphavirus subtype 3 (SAV 3) that is related to the salmonid alphavirus subtype 1 (SAV 1) which causes PD in Ireland and Scotland (McLoughlin and Graham, 2007). Currently, PD in Norway is endemic along the west coast south of the 63° latitude (Hustadvika). Fish stress plays a key role in the development of PD, with several examples of PD occurring after handling of fish (Brun et al., 2006; Raynard et al., 1992). Associations between the salmon lice (L.

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salmonis) burden and outbreaks of PD have also been reported (Ruane et al., 2005). Fish mortalities can reach 40% in PD affected pens and subsequent failure to grow is a further consequence of the disease resulting in poor condition and thin fish that are susceptible to parasitism and secondary bacterial diseases (Ruane et al., 2005). Economic losses due to PD have been estimated to reach approximately one billion NOK per year (Torrissen, 2008), and according to Aunsmo et al. (2010), a single PD outbreak on a fish farm with 500.000 smolts can result in a total loss of 14.4 million NOK.

The colour intensity is one of the most important quality parameters of Atlantic salmon fillets (Anderson, 2000). In addition, it is important that the variation among fillets from the same populations is low, and that the colour shows low variation between sections of the same fillet. Moreover, lack of dark spots or melanin is important. Firmness is another critical parameter that determines the acceptability of seafood products (Veland and Torrissen, 1999), where soft flesh leads to reduced acceptability by consumers (Ando, 1999; Hatae et al.,

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1985; Veland and Torrissen, 1999). Aunsmo et al. (2010) reported that quality downgrading contributes to the economic losses upon PD outbreaks, and anecdotal information indicates that PD may cause poor general muscle quality associated with severe discoloration (Bjerkeng, 2004). However, to our knowledge, there is no objective information available on the impact of PD on attributes of salmon fillet quality, apart from observations of grey shadows of melanin on fillets in the chronic phase of PD (McLoughlin, 2005), and decreased levels of vitamin E (Taksdal et al., 1995). Therefore the present study was undertaken to elucidate the impact of PD on quality related characteristics of commercial slaughter sized salmon. Analyses were performed on raw and cold-smoked fillets of salmon which were diagnosed with PD from 0 to 12 months prior to harvest. This is the first study of two, which is screening quality parameters of separate populations with a PD history. The second paper describes the fillet quality on an individual level based on pathological profile and gene transcriptome profiling (Larsson et al., in preparation).

2. Materials and methods

2.1. Fish material and experimental design

Slaughter ready Atlantic salmon (*Salmo salar* L.) were sampled from ten commercial fish farms, whereof nine farms participated in an epidemiological cohort study of pancreas disease (PD) in Norway, reported by Jansen et al. (2010). Fish in that cohort study were sampled for analyses with regard to salmonid alphavirus (SAV), specific antibodies and histopathological changes two and eight months following transfer to seawater and at the time of harvesting.

Salmon from seven farms were diagnosed with PD; salmon from one farm were infected with SAV without an outbreak of PD, whereas salmon from two farms had no records of PD diagnosis and worked as control farms (Table 1). These 10 farms were sorted into six different groups according to PD and SAV diagnosis; Control (number of fish (n) = 60, two farms), SAV (n = 30, one farm), PD0 (n = 19, one farm, diagnosed with PD at slaughter), PD6 (n = 50, two farms, diagnosed 5–7 months before slaughter), PD12 (n = 65, three farms, early diagnosis 11–12 months before slaughter) and PDchronic (n=30, one farm, repeated PD outbreaks during the seawater phase). The relatively high number of fish sampled from each location was based on results from a preliminary project, where a high variation in quality properties was found between individuals within the same populations (Mørkøre et al., 2011). The fish subjected to fillet quality analyses were selected randomly from each group, omitting fish below 2 kg and above 5 kg. The body weight of the fish sampled for analyses averaged 3.7 kg (range 2.5–4.9 kg).

All fish had the same age, were reared in net pens in seawater with relatively similar farming environments and they were fed commercial extruded diets. The fish were sampled randomly from the net pens for analyses, percussive killed, bled, gutted and thereafter stored on ice until sampling of tissues for virus and histopathological examination. Filleting was performed 4-6 days after slaughter. The right fillets were salted immediately after filleting, cold-smoked, vacuum packed, stored at 3 °C for three weeks and analysed for physical and chemical quality attributes. The left fillet was kept raw and subjected to quality analyses the day after filleting and muscle was sampled for subsequent chemical analyses. Raw and smoked fillets were analysed and sampled using the same procedures. The analyses included: texture, drip loss, pH (only raw fillets), gaping and colour (image analysis). Sectioning of the fillets for the various analyses is illustrated in Fig. 1. Section A (frequently termed the Norwegian Quality Cut, NQC) was stored at -20 °C, whereas section B was stored at -80 °C until chemical analyses. In addition, coldsmoked fillets of salmon originating from farm no. 1 and farm no. 10 (Table 1) were studied in separate storage trials where microbiological and chemical changes were analysed over 5 weeks at 7–8 °C (see 2.9).

2.2. PD and SAV diagnosis

Independent of the present study, all the fish farms were regularly monitored by private fish health services. When disease outbreak was suspected due to enhanced mortality and/or aberrant behaviour, standard diagnostic procedures were followed, including autopsy and submission of samples for laboratory examinations by Norwegian Veterinary Institute. PD was diagnosed when histopathological examination revealed changes characteristic for PD in Norway (Taksdal et al., 2007) combined with identification of SAV in the same individual. In short, PD pathology included significant loss of exocrine pancreatic tissue and inflammation in heart and skeletal muscle. SAV was diagnosed by real time RT-PCR using primers located in a conserved part of the genome coding for the E1 glycoprotein (Jansen et al., 2010).

For three of the farms, examinations of slaughter samples collected for the research projects confirmed PD and/or SAV infection (Table 1) although this had not previously been confirmed or identified by the diagnostic routines. In one of these farms, PD was already suspected based on histological examinations of tissues submitted by the fish health service 6 ½ month prior to slaughter. When both SAV and specific antibodies against SAV were detected at slaughter, this farm (Table 1, farm 5) was classified as PD affected for the present study. The second farm (Table 1, farm 4) was classified as PD0 because PD was diagnosed based upon the slaughter samples. The third farm (Table 1, farm 3) was diagnosed only as infected with

Table 1Groups of Atlantic salmon (*Salmo salar* L) sampled at ten different locations in Norway, number of individuals from each group, gutted body weight (mean and SD), pancreas disease (PD) diagnosis, detection of salmonid alphavirus (SAV) at different time points after sea transfer, specific antibodies to SAV determined at slaughter, and time between PD outbreaks and slaughter (months before slaughtering).

| Group | Farm | n | Gutted weight, kg | PD diagnosis ^a | SAV | | | | Time between PD |
|-----------|------|----|----------------------|------------------------------|-----------------------|-----------------------|-----------|----------|--|
| | | | | | 2 Months ^b | 8 Months ^b | Slaughter | Antibody | outbreak and slaughter ^c |
| Control | 1 | 30 | 4.90 (1.00) | No | No | No | No | No | |
| | 2 | 30 | 4.40 (1.06) | No | No | No | No | No | |
| SAV | 3 | 30 | 3.75 (0.62) | No | No | No | Yes | No | |
| PD0 | 4 | 19 | 2.54 (0.57) | Yes | No | No | Yes | Not ex | 0 month |
| PD6 | 5 | 30 | 2.66 (0.76) | Yes | No | No | Yes | Yes | 6.5 months |
| | 6 | 20 | 3.41 (0.37) | Yes | No | No | Yes | Yes | 7 months |
| PD12 | 7 | 17 | 4.13 (1.00) | Yes | No | Yes | Yes | Yes | 11.5 months |
| | 8 | 28 | 4.02 (1.20) | Yes | No | Not ex | Yes | Yes | 10.5 months |
| | 9 | 20 | 3.75 (1.49) | Yes | Not ex | Not ex | Yes | Yes | 14 months |
| PDchronic | 10 | 30 | 3.30 (1.07) | Yes | Yes | Yes | Yes | Yes | 14 and 5.5 month |

Not ex: not examined.

^a Summary of the production cycles from sea water transfer until harvest.

^b Months after sea water transfer.

^c PD diagnoses (determined by the ordinary fish health service).

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