Journal of Fish Diseases

WILEY

Journal of Fish Diseases

RESEARCH ARTICLE

Opercular Shortening and Deformities in Farmed Atlantic Salmon (*Salmo salar*) and Rainbow Trout (*Oncorhynchus mykiss*)—A Qualitative Study

Øyvind Salvesen[1] | Grunde Heggland[1] | Ane Grøndahl[1] | Mari Viken Kjønstad[1] | Liv Østevik[2]

[1]DNV Åkerblå AS, Sistranda, Norway | [2]Department of Preclinical Sciences and Pathology, Faculty of Veterinary Medicine, Norwegian University of Life Sciences, Ås, Norway

Correspondence: Øyvind Salvesen (oyvind.salvesen@dnv.com)

Received: 20 June 2025 | **Revised:** 24 September 2025 | **Accepted:** 29 September 2025

Funding: This work was funded by the Norwegian Seafood Research Fund (grant/project number 901872) and Åkerblå AS.

Keywords: Atlantic salmon | opercular deformity | operculum | operculum shortening | production routines | rainbow trout

ABSTRACT

In the Norwegian aquaculture industry, opercular shortening has been ranked as an important cause of increased mortality, reduced welfare and growth. The aim of this study was to gather experience-based knowledge from fish health personnel and production staff involved in the commercial production of farmed Atlantic salmon and rainbow trout. A total of 29 semi-structured interviews with a predefined list of questions on the topic of opercular shortening were conducted in the period 22.04.24 to 10.12.24. Results showed that participants reported aggressive behaviour due to underfeeding, with subsequent nipping of gill lids as the main cause of shortened opercula in farmed salmonids. Inadequate feed allocation of fry was perceived to be the most important risk factor for aggression, in addition to starving fry. The prevalence of opercular shortening in the freshwater phase was reported to be between 0% and 70%. Opercular shortening was typically first detected in fry between 2 and 5 g, but sometimes already during first feeding (0.5 g) and in alevins. Damaged gill tissue, reduced growth rates and increased mortality were suggested as consequences of shortened opercula. Sufficient feed allocation in fry stages, optimization of environmental conditions, reducing fish density and culling of small fry were reported as preventive measures, while culling affected fish was the sole mitigating measure.

1 | Introduction

The operculum consists of four articulated bony plates which are essential for protecting the gills from damage and for optimal ventilation (Kryvi and Poppe 2016; Sadler 2000). Opercular shortening is a morphological abnormality that has been documented in salmonids and a wide range of other species (Beraldo et al. 2003; Blaker and Ellis 2022; Lindesjöö et al. 1994). The exact aetiology of opercular shortening and other opercular deformities is largely unknown and may vary across species, facilities and cases, but several possible causes and risk factors have been described. Generally, these can be divided into nutritional diseases, management and environmental conditions, toxins and pollution, genetic factors, infectious agents and mechanical factors such as aggression or external damage (Billiard et al. 1999; Blaker and Ellis 2022; Halver et al. 1969; Larsen et al. 2018; MacLean 1999; Sadler et al. 2001; Taylor and Haber 1974; Østevik et al. 2025).

In the Norwegian aquaculture industry, opercular shortening has been ranked as an important cause of increased mortality, reduced welfare and growth (Moldal et al. 2025). The condition may be associated with gill injuries and other negative health outcomes (Blaker and Ellis 2022; Burnley et al. 2012;

© 2025 John Wiley & Sons Ltd.

Eriksen et al. 2007). Opercular shortening is often referred to as a production-related disease in farmed salmonids, which implies that the condition can be induced by management practices. Although opercular shortening has been a challenge in the culturing of salmonids for decades, there is limited knowledge about how the aquaculture industry itself perceives and manages this problem. The aim of this study was to gather experience-based knowledge from fish health personnel and production staff involved in the production of farmed Atlantic salmon and rainbow trout.

2 | Materials and Methods

A total of 29 semi-structured interviews with a predefined list of questions regarding opercular shortening were conducted in the period 22.04.24 to 10.12.24. Topics covered included:

- Causes and risk factors of opercular shortening.
- Examination and prevalence of shortened opercula.
- Temporal development and regeneration of opercular lesion.
- Consequences of opercular shortening.
- Preventive and mitigation measures used to manage opercular shortening.

In semiquantitative interviews, follow-up questions may be biassed by the theories of the interviewee. Therefore, detailed questionnaires were made to gather the same information from each interviewee. Questionnaires and an overview of reported production routines can be found in Appendices S1 and S2, respectively. Informants included fish health personnel (veterinarian and fish health biologists) and production staff with experiences from commercial production of Atlantic salmon and rainbow trout (Table 1). Production staff include both technical personnel with responsibility for daily care and feeding, and production leaders of a department or whole facility. The informants were selected to cover most production areas in Norway as well as six other countries with significant commercial production of salmon and rainbow trout. After each interview, a detailed report was sent to the informant to confirm the content. The data were then analysed qualitatively, assigned into different themes and compiled in an Excel database. Although some quantitative data were gathered, results showed large variations in production routines and how facilities systematised the data. In addition, the selection of interviewees was not randomised, and quantitative analysis was therefore not performed.

3 | Results

3.1 | Risks and Causes of Short Opercula

The vast majority of the risk factors mentioned by the producers and fish health personnel were associated with aggressive behaviour during first feeding and the fry stage, and subsequent nipping of opercula as a direct cause of opercular shortening (Table 2). Most participants highlighted inadequate feed allocation in the fry stage as the most important risk factor for aggression, in addition to starving fry (less than 5 g) for more than 12–24 h. Several of the producers also suggested that starving or underfeeding larger fry (over 5–10 g) seemed to increase the risk of damage to the dorsal fin. The production staff reported that the technical quality of the pellet was important, especially regarding buoyancy in relation to tank depth. This means that the pellet should float long enough for sufficient spread in the tank, before it sinks at a speed that ensures that fry in the whole water column are offered feed. Some farms reported variations between different shifts in terms of care and feed allocation, in which inexperienced personnel could pose a risk of both under- and overfeeding the fry.

In addition to underfeeding, the informants suggested that different factors that may lead to a sub-optimal tank environment

TABLE 1 | Distribution of interviewees in terms of nationality, job role, production form and phase and species they have experience with. One person may have experience from production of different species and production forms.

Nationality and job role			Production form			Species	
Freshwater phase							
Nationality	Fish health	Production staff	RAS	FT	RAS and FT	Salmon	Rainbow trout
Norway	8	9	2	6	9	15	2
Iceland, Canada, Scotland, Chile and Faroe Islands.	5	0	1	0	4	5	1
Seawater phase							
Nationality	Fish health	Production staff	Open	Semi-closed	Closed	Salmon	Rainbow trout
Norway	5	0	5	3	1	5	1
Scotland and Canada	2	0	2	0	0	2	0

Abbreviations: FT, flow through system; RAS, recirculation aquaculture system.

TABLE 2 | Causes and risk factors of opercular shortening and deformities suggested by interviewees.

Phase	Cause	Risk factors
Freshwater	Biting, aggression between fish	Underfeeding Suboptimal pellet quality/buoyancy Starvation Suboptimal care and operation routines High or low fish density Tank size and shape Low water velocity High water particle load Sudden changes in light intensity or water temperature
	High water temperature during incubation of eggs	
	Calcium deficiency (also other concurrent skeletal deformities)	Water source with low calcium levels (ground water)
Seawater	Trauma sustained during handling and delousing operations	Delousing methods using flushing with water jets
	Bite injuries from cleaner fish	Suboptimal care and feeding of cleaner fish

could represent possible risk factors for shortened opercula. These included low water velocity (less than 1–1.5 fish length per second), high particle load, or sudden changes in light intensity or water temperature. Sub-optimal levels of pH, CO_2, salinity, oxygen, nitrate compounds and total gas pressure (TGP) were not associated with short opercula according to interviewees, but one case of deformed and folded opercula and generalised concurrent skeletal deformities was described due to calcium deficiency at a facility with low levels of calcium in the water source.

Both too high and low fish density were reported as risk factors for shortened opercula. However, it was suggested that the surface area of tanks was more important than tank volume, and that more than approximately 10,000 fry/m^2 could increase the risk of aggression and shortened opercula. Large tanks (> 6-m diameter) during first feeding were suggested as a risk factor because the fry appeared to clump together and were less prone to swim up to the surface in search of feed. Similarly, some experienced that the fry were distributed less evenly in octagonal tanks than in round tanks, which seemed to make them fight for the best positions in the tank. Incubation of eggs at high temperatures was suggested as a cause of deformed and folded opercula by one producer. Genetic strain was not stated as an important risk factor by the informants, but some experienced that strains modified for fast growth had a higher risk of aggression due to underfeeding.

While most participants agreed that opercular shortening primarily develops during the first feeding and fry period, some suggested that opercular injuries can occur in the sea phase as well. Damage to the opercula was described during delousing operations, especially after mechanical delousing treatments that involved flushing of the fish surface. Injuries attributed to attacks from cleaner fish, specifically wrasse, were also reported from fish health personnel in Scotland, but not from other countries.

3.2 | Examination and Prevalence of Shortened Opercula

Most producers examined opercula as a part of routine health checks during the freshwater phase. However, there were large variations in the timing of opercular assessment, how frequently it was done, and the number of fish examined per tank or fish group (range 20–100 fish). Assessment of opercular status was more intensive at sites that had a high prevalence of opercular shortening, while at sites with low prevalence, opercular status was most often only examined when assessing smolts prior to sea transfer. In Norway, opercula was primarily scored from 0 to 3 using Laksvel (Nilsson et al. 2022) or Fishwell (Noble et al. 2018) scoring systems. In other countries, different scoring systems were reported including yes/no, 0–2 and 0–3. A few farms scored gill tissue simultaneously with opercula in the freshwater phase. In Norway, the prevalence of opercular shortening in the freshwater phase was reported to be between 0% and up to 70%, with large variation across facilities and fish groups. A prevalence of around 0% and up to 40% was described by informants in other countries. At the final check before sea transfer, a prevalence of more than 5% was generally considered too high.

3.3 | Temporal Development and Regeneration of Opercular Lesion

Opercular shortening was typically first detected in fry between 2 and 5 g, but sometimes already during first feeding (0.2–0.5 g) and in alevins (Figure 1). The lesions were described as injuries with a roughened or uneven caudal opercular edge and shortening of one or both opercula. Most informants suggested that mild and moderate (score 1–2, Laksvel), but not severe (score 3), opercular shortening could heal, possibly facilitated by increasing water temperature. Regenerating tissue was described as a thin membrane expanding from the caudal edge of the damaged

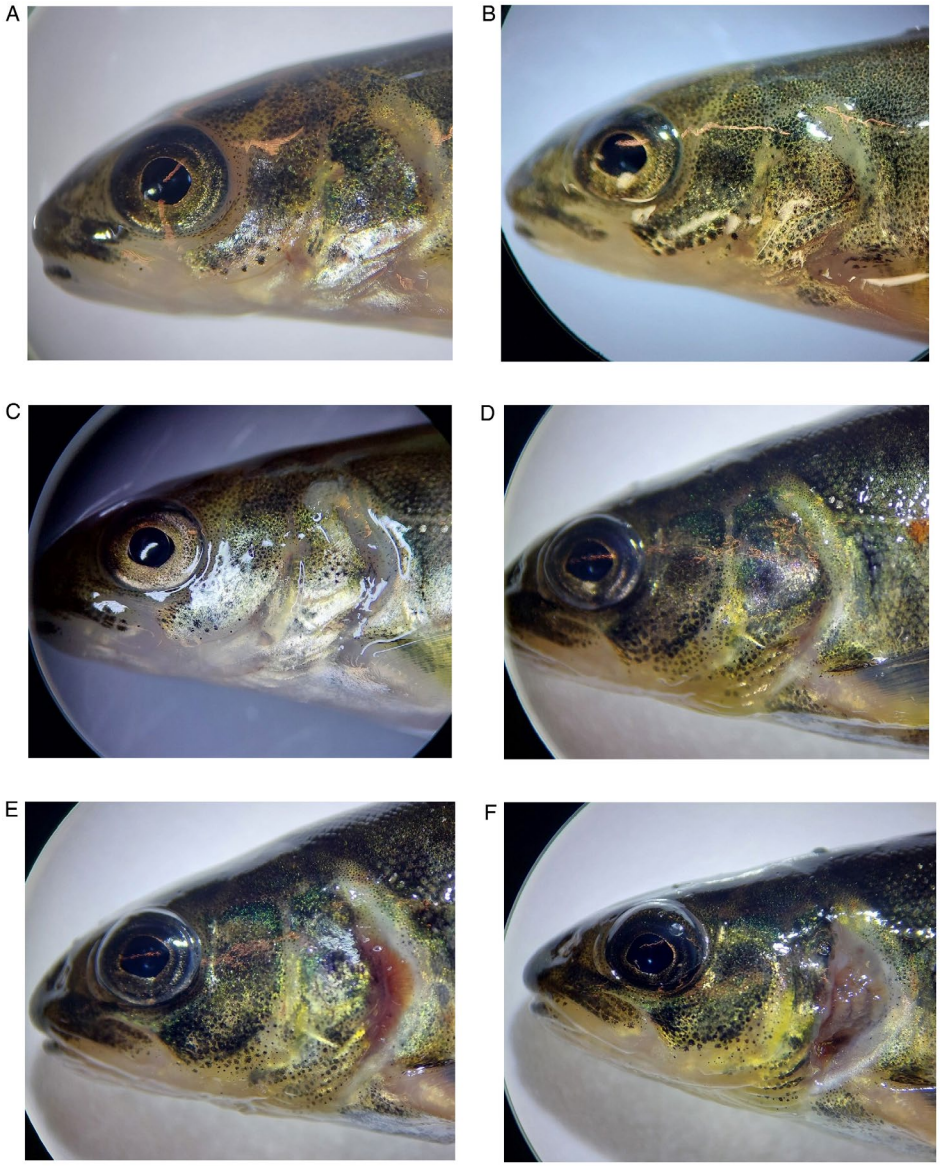

FIGURE 1 | Systematic monitoring and scoring of opercular lesions. Images show the development of opercular lesions in a group of Atlantic salmon examined repeatedly using a stereo microscope from early first feeding until 5–8 g. (A) 0.5 g—score 1, (B) 1 g—score 1, (C) 1 g—score 2, (D) 5–8 g—score 1, (E) 5–8 g—score 2, (F) 5–8 g—score 3. Fish were scored using Laksvel score (Nilsson et al. 2022) score. Photos: Kari Marie Børtveit, Mowi ASA.

opercula, which gradually covered the gill cavity and underlying gill tissue.

3.4 | Consequences of Opercular Shortening

Damage to underlying gill tissue, reduced growth rates, and increased mortality were suggested by the industry as possible consequences of shortened opercula in both the fresh and seawater phases. Gill injuries associated with opercular shortening were described as shortened and irregularly oriented gill filaments with yellow, white or grey discoloration of the gill tissue (Figure 2). Some participants thought that fish with opercular shortening were overrepresented among mortalities immediately after sea transfer and would gradually disappear during the sea phase. Others suggested that fish with opercular lesions were more likely to die during handling and delousing operations.

3.5 | Preventive and Mitigation Measures

Sufficient feed allocation during the first feeding and fry period, optimization of environmental conditions, and culling of small fry were highlighted as the most important preventive measures used by industry. Some producers also moved affected fish groups to new tanks to reduce the fish density. The most common mitigation measure was culling of fish with moderately to severely shortened opercula, primarily during vaccination prior to sea transfer. Due to predefined production plans, opercular shortening prevalence was generally not emphasised when fish groups were allocated into specific sites at sea.

FIGURE 2 | Opercular shortening and deformities associated with gill pathology. (A) Marked opercular shortening and irregular orientation of underlying gill filaments. (B) Opercular shortening and severe shortening and loss of gill filaments on the first gill arch. Affected filaments are thickened and discoloured, appearing grey to white. Gill tissue on the remaining arches is also variably disorganised and discoloured. (C) Loss or shortening of gill filaments on several gill arches. (D) Severe opercular shortening and shortened and thickened white to pink gill filaments. All images of farmed Atlantic salmon, A and B from the sea phase, C smolt and D parr. Photos: Åkerblå AS and Liv Østevik.

4 | Discussion

In this paper we describe the perceptions and experience-based knowledge on the topic of opercular shortening derived from interviews with fish health personnel and production staff involved in the production of farmed Atlantic salmon and rainbow trout. This information can be useful for scientists and fish health personnel as there is a lack of scientific studies examining the causes and risk factors, preventative and mitigation measures and effects of opercular shortening and deformities on the health and welfare of farmed salmonids. Our results may also be used to generate hypotheses for future research studies that will lead to new knowledge about this condition.

Most interviewees agreed that aggressive behaviour due to underfeeding with subsequent nipping of gill lids was the main cause of shortened opercula in farmed salmonids. Nipping has also been suggested as a probable cause in some studies (Blaker and Ellis 2022; Larsen et al. 2015; MacLean 1999), but we found no scientific studies aimed at confirming or disputing this idea. However, aggressive behaviour has been suggested as a cause of dorsal fin damage in Atlantic salmon under experimental conditions with food restriction (10 and 30 days), high stocking density ($>30\,kg/m^3$) or unpredictable food delivery (Cañon Jones et al. 2010; Turnbull et al. 1998). Notably, social network analysis of this behaviour indicated separation of fish within sub-groups of initiators of aggression and receivers of aggression (Cañon Jones et al. 2010). Nevertheless, an alternative explanation could be that damage to the opercula and fins is due to accidental nipping in mistakes for pellets rather than deliberate aggressive behaviour.

There are scientific studies examining the effect of different fish densities, feeding strategies, water temperatures and incubation temperatures on the prevalence of opercular lesions in salmonids; however, the number of studies is limited, and some studies are performed in settings that may not be directly relevant to commercially farmed fish. There is no scientific literature on the possible effects of many of the risk factors discussed by interviewees in this study, like personnel compliance, starvation, tank design and water velocities on opercular shortening and opercular deformities. Larsen et al. (2018) found no effect of different strategies for feed distribution and feeding frequency on the prevalence of opercular erosion in Atlantic salmon but reported significantly higher prevalence of opercular erosion 1 year post-hatch in fish groups kept at 7500 fry/m^2 compared to 2500 fry/m^2 (Larsen et al. 2015). An effect on incubation temperature has been shown, where incubation of Atlantic salmon eggs at 14°C was associated with a higher prevalence of opercular lesions than at 8°C (Ørnsrud et al. 2004). However, today it is routine to incubate Atlantic salmon eggs at less than 8°C to avoid deformities (Fraser et al. 2015), so this is unlikely to be a common cause of opercular deformities in commercial farming at present. Studies have not shown an effect of different

water temperatures in post-hatch fry (Amoroso et al. 2016; MacLean 1999), but the effect of rapid changes in water temperature has not been studied.

Similar to our informants, different scientific studies report a large variation in the prevalence of opercular lesions, ranging from ≤1% to >80% of examined fish (Amoroso et al. 2016; Fjelldal et al. 2007; Taylor et al. 2012). Further, in a large set of field data from 292 sites culturing Atlantic salmon in Norway, Chile, Scotland, Ireland, Canada and Chile, the prevalence of opercular lesions ranged from 0% to 60%, with a median of 4.6% (Østevik et al. 2025). As some informants in our study were selected to represent facilities with known problems with opercular shortening, the prevalence of up to 70% affected fish is probably not representative of the industry as a whole.

As suggested by the participants, most scientific studies show that spontaneously occurring opercular lesions in salmonids are first observed after first feeding (Amoroso et al. 2016; Blaker and Ellis 2022; Eriksen et al. 2007; Kazlauskienė et al. 2006; Kvernes 1990; MacLean 1999; Sadler 2000; Sadler et al. 2001), though some cases are observed in earlier life stages (Gunstrom 1973; Marty et al. 1997). In salmonids, regeneration of opercular defects has been described both after surgical creation of opercular defects for marking purposes (Pulford and Woodall 1963; Rich and Holmes 1929; Rosburg et al. 2022), and in cases with spontaneously occurring opercular erosions (MacLean 1999).

A connection between opercular lesions and gill lesions, as suggested by the interviewees, has been reported previously (Blaker and Ellis 2022; Jørgensen and Rød 2019; Kirczuk and Domagała 2009; Osburn 1911; Tchernavin 1939). Whether opercular lesions affect mortality in fresh water varies between studies (Kvernes 1990; Pulford and Woodall 1963). A single study showed that Atlantic salmon with opercular shortening were more likely to die after handling compared to normal fish (Burnley et al. 2012), supporting this notion from our interviewees. An association between lower weight and/or shorter length and opercular lesions is also described in the literature (Burnley et al. 2010; Eriksen et al. 2007; Kvernes 1990), and suggests that affected fish may have reduced growth rates. To the best of our knowledge, there are not any scientific studies or grey literature describing investigations of measures to prevent opercular lesions or resolve and mitigate the issue once it has occurred.

In conclusion, we describe insights and experiences on opercular shortening from fish health personnel and production staff working in aquaculture of Atlantic salmon and rainbow trout. Future research should focus on examining possible causes and risk factors for opercular shortening identified by interviewees and addressing the question of which preventive and mitigation measures are most effective to reduce the prevalence of opercular shortening and the subsequent negative health and welfare impacts of this condition.

Author Contributions

Øyvind Salvesen: investigation, writing – original draft, writing – review and editing, data curation, methodology. **Grunde Heggland:** investigation, writing – review and editing. **Ane Grøndahl:** investigation, writing – review and editing. **Mari Viken Kjønstad:** writing – review and editing, project administration, methodology. **Liv Østevik:** writing – original draft, writing – review and editing, project administration, methodology.

Acknowledgements

The authors would like to thank the interviewees in the study for sharing their experience and time.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

References

Amoroso, G., M. B. Adams, T. Ventura, C. G. Carter, and J. M. Cobcroft. 2016. "Skeletal Anomaly Assessment in Diploid and Triploid Juvenile Atlantic Salmon (*Salmo salar* L.) and the Effect of Temperature in Freshwater." *Journal of Fish Diseases* 39, no. 4: 449–466. https://doi.org/10.1111/jfd.12438.

Beraldo, P., M. Pinosa, E. Tibaldi, and B. Canavese. 2003. "Abnormalities of the Operculum in Gilthead Sea Bream (*Sparus aurata*): Morphological Description." *Aquaculture* 220, no. 1–4: 89–99. https://doi.org/10.1016/S0044-8486(02)00416-7.

Billiard, S. M., K. Querbach, and P. V. Hodson. 1999. "Toxicity of Retene to Early Life Stages of Two Freshwater Fish Species." *Environmental Toxicology and Chemistry* 18, no. 9: 2070–2077. https://doi.org/10.1897/1551-5028%281999%29018%3C2070:TORTEL%3E2.3.CO;2.

Blaker, E., and T. Ellis. 2022. "Assessment, Causes and Consequences of Short Opercula in Laboratoryreared Atlantic Salmon (*Salmo salar*)." *Animal Welfare* 31, no. 1: 79–89. https://doi.org/10.7120/09627286.31.1.007.

Burnley, T. A., H. Stryhn, H. J. Burnley, and K. L. Hammell. 2010. "Randomized Clinical Field Trial of a Bacterial Kidney Disease Vaccine in Atlantic Salmon, *Salmo salar* L." *Journal of Fish Diseases* 33, no. 7: 545–557. https://doi.org/10.1111/j.1365-2761.2010.01151.x.

Burnley, T. A., H. Stryhn, and K. L. Hammell. 2012. "Post-Handling Mortality During Controlled Field Trials With Marine Grow-Out Atlantic Salmon, *Salmo salar* L." *Aquaculture* 368: 55–60. https://doi.org/10.1016/j.aquaculture.2012.09.006.

Cañon Jones, H. A., L. A. Hansen, C. Noble, B. Damsgård, D. M. Broom, and G. P. Pearce. 2010. "Social Network Analysis of Behavioural Interactions Influencing Fin Damage Development in Atlantic Salmon (*Salmo salar*) During Feed-Restriction." *Applied Animal Behaviour Science* 127, no. 3: 139–151. https://doi.org/10.1016/j.applanim.2010.09.004.

Eriksen, M. S., Å. Espmark, B. O. Braastad, R. Salte, and M. Bakken. 2007. "Long-Term Effects of Maternal Cortisol Exposure and Mild Hyperthermia During Embryogeny on Survival, Growth and Morphological Anomalies in Farmed Atlantic Salmon *Salmo salar* Offspring." *Journal of Fish Biology* 70, no. 2: 462–473. https://doi.org/10.1111/j.1095-8649.2007.01317.x.

Fjelldal, P. G., T. J. Hansen, and A. E. Berg. 2007. "A Radiological Study on the Development of Vertebral Deformities in Cultured Atlantic Salmon (*Salmo salar* L.)." *Aquaculture* 273, no. 4: 721–728. https://doi.org/10.1016/j.aquaculture.2007.07.009.

Fraser, T. W., T. Hansen, M. S. Fleming, and P. G. Fjelldal. 2015. "The Prevalence of Vertebral Deformities Is Increased With Higher Egg

Incubation Temperatures and Triploidy in Atlantic Salmon *Salmo salar* L." *Journal of Fish Diseases* 38, no. 1: 75–89. https://doi.org/10.1111/jfd.1220610.1111/jfd.12206.

Gunstrom, G. K. 1973. "Gross Physical Anomalies in F1 Progeny of Irradiated and Unirradiated Coho Salmon." *Transactions of the American Fisheries Society* 102, no. 2: 438–442. https://doi.org/10.1577/1548-8659(1973)102<438:GPAIFP>2.0.CO;2.

Halver, J. E., L. M. Ashley, and R. R. Smith. 1969. "Ascorbic Acid Requirements of Coho Salmon and Rainbow Trout." *Transactions of the American Fisheries Society* 98, no. 4: 762–771. https://doi.org/10.1577/1548-8659(1969)98[762:AAROCS]2.0.CO;2.

Jørgensen, L. M., and I. H. Rød. 2019. "Temporale makroskopiske og histopatologiske forandringer etter termisk avlusing hos atlantisk laks (*Salmo salar* L.)." Doctor of Veterinary Medicine Student Paper, Others, Norwegian University of Life Sciences, Ås. http://hdl.handle.net/11250/2601456.

Kazlauskienė, N., E. Leliūna, and V. Kesminas. 2006. "Peculiarities of Opercular Malformations of Salmon (*Salmo salar* l.) Juveniles Reared in the Žeimena Salmon Hatchery." *Acta Zoologica Lituanica* 16, no. 4: 312–316. https://doi.org/10.1080/13921657.2006.10512747.

Kirczuk, L., and J. Domagała. 2009. "Deformations in Reciprocal Hybrids of Salmon (*Salmo salar* L., 1758) and Trout (*Salmo trutta m. trutta* L., 1758) Aged 0+ and 1+." *Bulletin of the European Association of Fish Pathologists* 29, no. 5: 153–162.

Kryvi, H., and T. Poppe. 2016. *Fiskeanatomi* (Vol. 1). Vigmostad & Bjørke AS.

Kvernes, E. L. 1990. "Gjellelokkforkortelse hos lakseyngel i oppdrett – eventuell sammenheng med miljøfaktorer." Hovedoppgave, Norges Landbrukshøgskole.

Larsen, M. H., J. I. Johnsson, J. Naslund, S. T. Thomassen, and K. Aarestrup. 2015. "Reduced Rearing Density Increases Postrelease Migration Success of Atlantic Salmon (*Salmo salar*) Smolts." *Canadian Journal of Fisheries and Aquatic Sciences* 73, no. 5: 804–810. https://doi.org/10.1139/cjfas-2014-0563.

Larsen, M. H., A. Nemitz, M. Steinheuer, J. Lysdal, S. T. Thomassen, and G. Holdensgaard. 2018. "Effects of Hatchery Feeding Practices on Fin and Operculum Condition of Juvenile Atlantic Salmon *Salmo salar*." https://danmarksvildlaks.dk/wp-content/uploads/2018/04/Technical-report_feeding_opercula.pdf.

Lindesjöö, E., J. Thulin, B. E. Bengtsson, and U. Tjärnlund. 1994. "Abnormalities of a Gill Cover Bone, the Operculum, in Perch *Perca fluviatilis* From a Pulp Mill Effluent Area." *Aquatic Toxicology* 28, no. 3–4: 189–207. https://doi.org/10.1016/0166-445X(94)90033-7.

MacLean, A. 1999. "Compensatory Growth, Life-History Decisions and Welfare of Farmed Atlantic Salmon (*Salmo salar* L.) Parr." Doctor of Philosophy, PhD thesis, University of Glasgow, Glasgow. https://theses.gla.ac.uk/id/eprint/76210.

Marty, G. D., R. A. Heintz, and D. E. Hinton. 1997. "Histology and Teratology of Pink Salmon Larvae Near the Time of Emergence From Gravel Substrate in the Laboratory." *Canadian Journal of Zoology* 75, no. 6: 978–988. https://doi.org/10.1139/z97-119.

Moldal, T., J. Wiik-Nielsen, V. H. S. D. Oliveira, J. Svendsen, and I. Sommerset. 2025. "Fiskehelserapporten 2024 (Veterinærinstituttets rapportserie nr. 1a/2025)." https://www.vetinst.no/rapporter-og-publikasjoner/rapporter/2025/fiskehelserapporten-2024.

Nilsson, J., K. Gismervik, K. V. Nielsen, et al. 2022. "Laksvel – Standardisert operasjonell velferdsovervåking for laks i matfiskanlegg (Rapport fra havforskningen 14)." https://imr.brage.unit.no/imr-xmlui/handle/11250/2997021.

Noble, C., J. Nilsson, L. H. Stien, M. Iversen, J. Kolarevic, and K. Gismervik. 2018. "Velferdsindikatorer for oppdrettslaks: Hvordan vurdere og dokumentere fiskevelferd." www.nofima.no/fishwell.

Ørnsrud, R., L. Gil, and R. Waagbø. 2004. "Teratogenicity of Elevated Egg Incubation Temperature and Egg Vitamin A Status in Atlantic Salmon, *Salmo salar* L." *Journal of Fish Diseases* 27, no. 4: 213–223. https://doi.org/10.1111/j.1365-2761.2004.00536.x.

Osburn, R. C. 1911. "The Effects of Exposure on the Filaments of Fishes." *Transactions of the American Fisheries Society* 40: 371–376. https://doi.org/10.1577/1548-8659(1910)40[371:TEOEOT]2.0.CO;2.

Østevik, L., M. Stormoen, T. Kortner, et al. 2025. "Gjellelokkforkortelse hos laksefisk—En kunnskapssammenstilling." https://www.fhf.no/prosjekter/prosjektbasen/901872/.

Pulford, E. F., and L. M. Woodall. 1963. "An Operculum Marking Experiment on Juvenile Chinook Salmon." http://hdl.handle.net/1957/17362.

Rich, W. H., and H. B. Holmes. 1929. *Experiments in Marking Young Chinook Salmon on the Columbia River, 1916 to 1927*. Government Printing Office. http://pi.lib.uchicago.edu/1001/cat/bib/4133676.

Rosburg, A. J., J. L. Davis, and M. E. Barnes. 2022. "Retention of Fin Clips and Fin and Operculum Punch Marks in Rainbow Trout." *Aquaculture and Fisheries* 7, no. 6: 660–663. https://doi.org/10.1016/j.aaf.2021.06.004.

Sadler, J. 2000. "Comparison of Aspects of the Physiology and Morphology of Diploid and Triploid Atlantic Salmon *Salmo salar*." PhD thesis, University of Tasmania.

Sadler, J., P. M. Pankhurst, and H. R. King. 2001. "High Prevalence of Skeletal Deformity and Reduced Gill Surface Area in Triploid Atlantic Salmon (*Salmo salar* L.)." *Aquaculture* 198, no. 3–4: 369–386. https://doi.org/10.1016/S0044-8486(01)00508-7.

Taylor, J. F., E. Leclercq, A. C. Preston, D. Guy, and H. Migaud. 2012. "Parr-Smolt Transformation in Out-Of-Season Triploid Atlantic Salmon (*Salmo salar* L.)." *Aquaculture* 362-363: 255–263. https://doi.org/10.1016/j.aquaculture.2010.12.028.

Taylor, R. E., and M. H. Haber. 1974. "Opercular Cyst Formation in Trout Infected With *Myxosoma Cerebralis*." *Journal of Wildlife Diseases* 10, no. 4: 347–351. https://doi.org/10.7589/0090-3558-10.4.347.

Tchernavin, V. 1939. "XVI.—Two Cases of Malformation of the Gill-Covers in Salmon." *Proceedings of the Royal Society of Edinburgh* 58: 233–241. https://doi.org/10.1017/S0370164600011147.

Turnbull, J. F., C. E. Adams, R. H. Richards, and D. A. Robertson. 1998. "Attack Site and Resultant Damage During Aggressive Encounters in Atlantic Salmon (*Salmo salar* L.) Parr." *Aquaculture* 159, no. 3: 345–353. https://doi.org/10.1016/S0044-8486(97)00233-0.

Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Appendix S1:** Interview questions. **Appendix S2:** Production routines.