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Rapid growth rates are associated with increased coronary arteriosclerosis in farmed Atlantic Salmon (*Salmo salar*) under commercial production conditions

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ABSTRACT

Coronary arteriosclerosis, the narrowing and stiffening of the arteries supplying oxygenated blood to the myocardium, is gaining attention as a contributor to stress-related mortality in farmed Atlantic salmon (*Salmo salar*). Rapid growth has been proposed as a risk factor. Thus, clarifying relationships between growth and arteriosclerosis is important, as aquaculture's emphasis on accelerated growth may compromise fish cardiovascular health.

Arteriosclerotic lesions were histologically assessed in the main coronary artery of Atlantic salmon at four commercial sites in the Faroe Islands. Fish were sampled after sea transfer and before slaughter to evaluate the effects of hatchery and marine growth. Lesion prevalence (% fish affected), incidence (% artery affected), severity (mean histopathological grade, 0–5), and coronary lumen area reduction for the most severe lesions were assessed.

After sea transfer, prevalence ranged from 26 to 67%, with incidences of 5–26% across sites. Lesion severity ranged from 0.1 to 0.3, and lumen area reduction in the most severe lesions ranged from 4 to 14%. Before slaughter, prevalence increased to 90–100% and lesion incidence to 69–84%. Lesion severity ranged from 2.5 to 3, and the lumen area reduction ranged from 15 to 17%.

Growth rates were significantly associated with variation in lesion status across sites: higher hatchery growth coincided with greater severity at sea transfer, and higher marine growth was associated with increased lesion progression thereafter. These observational findings indicate that higher growth rates are associated with increased coronary arteriosclerosis in Atlantic salmon under commercial production conditions, suggesting a trade-off between rapid production and cardiovascular health.

1. Introduction

Consumption of Atlantic salmon (*Salmo salar*) and other salmonid fishes is widely promoted for its cardiovascular benefits to humans (Nousheen et al., 2026). Yet, it is less known that both wild and farmed Atlantic salmon develop cardiovascular diseases, including coronary arteriosclerosis (Farrell et al., 1990; Saunders et al., 1992; Farrell, 2002). The term coronary arteriosclerosis refers to the hardening and narrowing of the coronary arteries, which supply oxygen-rich blood to the heart muscle (myocardium), resulting in reduced blood flow (Fishbein and Fishbein, 2009). In humans, where the myocardium is one of the most aerobically active tissues, it is well established that reduced coronary blood flow can reduce myocardial perfusion and blood supply (ischemia), thereby impairing cardiac function and ultimately leading to

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myocardial infarction, heart failure, or death (Ciuca-Pană et al., 2025).

Not all fish species possess a coronary circulation, but it is present in some active, migratory species such as salmonids, and represents a critical adaptation for sustaining a high cardiac output during intense and metabolically demanding activities (Farrell, 2002; Morgenroth et al., 2021; Ekström et al., 2023). In aquaculture, farmed Atlantic salmon are routinely subjected to various physiologically demanding stressors, such as crowding, sorting, transportation, delousing, etc., which can significantly elevate cardiac workload and oxygen demand. Under these conditions, a healthy and well-functioning coronary circulation becomes particularly important for sustaining cardiac performance. However, the resilience of farmed Atlantic salmon to such stressors appears compromised, as evidenced by the alarming rise in events of stress-related mortalities across major salmon-producing countries, including Norway, Chile, the United Kingdom (Oliveira et al., 2021; Sommerset et al., 2023), and the Faroe Islands (Rúni Dam, Avrik, unpublished). Although such events are often considered multifactorial, emerging evidence suggests that coronary arteriosclerosis may play a role. For instance, in one case, over 10% of farmed Atlantic salmon died following stressful transportation (Poppe et al., 2007). Histopathological examinations revealed that these fish suffered from extensive coronary arteriosclerosis in the main coronary artery as well as myocardial necrosis. The authors hypothesized that the necrotic lesions were signs of myocardial infarctions caused by ischemia, triggered by arteriosclerosis during elevated cardiac workload and metabolic demand under the stressful transportation conditions. Similar myocardial necrotic lesions are now being reported with increasing frequency in other case studies of farmed Atlantic salmon that died in connection with stressful handling events (Poppe et al., 2021). While these observations are largely based on case reports and indirect pathological evidence rather than controlled experimental studies, they collectively support a plausible link between coronary arteriosclerosis and stress-related mortality.

Complementary experimental studies in salmonids further support a potential link between coronary arteriosclerosis and reduced stress tolerance. In farmed rainbow trout (*Oncorhynchus mykiss*), a negative correlation between coronary arteriosclerosis in the main coronary artery and stress tolerance during a standardized crowding stress test has been reported (Brijs et al., 2020). In addition, laboratory studies on rainbow trout with surgically occluded main coronary arteries, mimicking severe arteriosclerosis, demonstrate impaired cardiac and metabolic performance, as well as reduced tolerance to warming and hypoxia stress (Ekström et al., 2017, 2018; Morgenroth et al., 2021).

In salmonids, coronary arteriosclerosis is characterized by neointimal proliferation of smooth muscle cells, and sometimes disruption of the elastic lamina in the vessel wall, rather than by cholesterol-rich plaque formation as observed in humans (Farrell, 2002; Dalum et al., 2016). The coronary lesions in salmonids accumulate progressively, with older fish exhibiting more severe arteriosclerosis (Saunders et al., 1992; Farrell, 2002). In addition to age, a study conducted over thirty years ago found that accelerated growth appeared to increase smooth muscle cell proliferation in farmed Atlantic salmon (Saunders et al., 1992). More recent findings in farmed rainbow trout have supported this association (Brijs et al., 2020). However, neither of these studies directly assessed growth rate. Instead, they relied on indirect measures, such as differences in body length, as a proxy for growth (Saunders et al., 1992), or examined how growth-promoting rearing strategies, including elevated temperatures, were associated with lesion prevalence and severity (Brijs et al., 2020). Consequently, the specific impact of growth rate on the development of salmonid coronary arteriosclerosis remains unclear, as no studies have directly assessed this relationship. This knowledge is critical as the aquaculture industry continues to prioritize accelerated growth to maximize production turnover (Kristjánsson et al., 2020). Without a clear understanding of how growth influences the development of coronary arteriosclerosis, there is a risk that current rearing practices may inadvertently compromise fish cardiovascular health and their resilience to farming-related stress, thereby increasing mortality risk and ultimately undermining the long-term sustainability of the aquaculture industry.

To address this knowledge gap, the severity of coronary arteriosclerosis in the main coronary artery was assessed in farmed Atlantic salmon in the Faroe Islands experiencing different commercial growth rates rates. Atlantic salmon production typically consists of two distinct phases: a hatchery phase on land, followed by a marine phase during which the fish grow to slaughter size. The present study examined the relationship between growth rate during the hatchery phase and arteriosclerotic status at the time of sea transfer, as well as how subsequent growth rate during the marine phase influenced arteriosclerotic status just before slaughter. These two time points, the beginning and end of the marine phase, were chosen because episodes of stress-induced mortality are primarily reported during the marine production phase (Oliveira et al., 2021; Sommerset et al., 2023). It was hypothesized that accelerated hatchery growth predisposes salmon to more advanced coronary arteriosclerosis at sea transfer, and that rapid marine growth further exacerbates lesion progression.

2. Materials and methods

2.1. Fish sampling and processing

In 2021 and 2022, hearts from apparently healthy Atlantic salmon originating from the Stofnfiskur strain were collected from four sea farming sites (sites 1–4) in the Faroe Islands. The fish were sampled in connection with the mandatory count of salmon lice (*Lepeophtheirus salmonis*), which involves the random selection of fish to ensure they are representative of the overall population in the sea cage. All procedures were conducted in accordance with current ethical and legal requirements for animal research in the Faroe Islands. The study was approved by the Chief Veterinary Officer (landsdjóralæknin) in accordance with §10 of the Faroese Animal Welfare Act (Løgtingslóg nr. 49 frá 30. apríl 2018 um djóravælferð; Case no. 22/01525–2). Additionally, the study received approval from Firum's internal ethics review board (approval no. 011).

Sampling occurred one month after sea transfer and again ten months after sea transfer, close to slaughter. To minimize age-related confounding effects, given that older fish accumulate more lesions (Farrell, 2002), the sites were grouped based on the age at sea transfer. Sites 1 and 2 were stocked with fish approximately 15 months post-hatch, while sites 3 and 4 were stocked with older fish, approximately 20 months post-hatch, resulting in an approximate five-month age difference between the groups at both sampling points. Sites 1 and 2 were operated by one company, with fish originating from the same hatchery. Sites 3 and 4 were operated by another company, with fish originating from a different hatchery but shared between the two sites. This grouping ensured that fish within each age category were reared under comparable hatchery and company-specific conditions, reducing confounding effects unrelated to age or growth rate.

Hearts from 30 fish per site were sampled at each sampling point, except at site 3, where only 20 fish were available each time. This was due to the farm not fasting the fish before sampling, making them more challenging to catch. The fish were euthanized with a lethal dose (200–300 mg/L) of MS-222 before being brought to land. On land, the weight and fork length were recorded, and the condition factor (CF) was calculated for each fish as follows:

$$CF = \frac{Weight}{Length^3}\ x\ 100$$

Sex was not determined but assumed to be evenly distributed across groups due to random sampling and mixed-sex rearing.

The hearts were dissected out, and the weight of the ventricle was recorded after the tissues were blotted dry by gently dabbing them on a

paper towel while squeezing them to remove the blood. After weighing, the hearts were placed in 10% formalin (buffered 4% formaldehyde) for later histological analysis.

2.2. Growth metrics

The thermal growth coefficient (TGC), a dimensionless, normalized growth rate used to compare growth among fish, is preferable for assessing growth as it accounts for both differences in body size and temperature, making comparisons across groups more meaningful (Thorarensen and Farrell, 2011). However, detailed hatchery temperature records were not made available to the study due to company confidentiality, and TGC could therefore not be calculated for the hatchery phase. Instead, the absolute growth rate (AGR) was used to represent hatchery growth as detailed below.

As described in Section 2.1, fish at sites 1 and 2 originated from the same hatchery, while fish at sites 3 and 4 originated from another shared hatchery. In the Faroe Islands, hatchery production is conducted in recirculating aquaculture systems (RAS) due to limited freshwater resources (Ytrestølj et al., 2023), enabling controlled, year-round stable temperature with minimal fluctuations. Hatchery temperatures were therefore considered uniform within each site pair, minimizing potential temperature-related bias in AGR comparisons during the hatchery phase.

AGR was calculated as follows:

$$AGR = \left(\frac{g}{day}\right) = \frac{W2 - W1}{N_{days}}$$

Where W1 and W2 represent initial and final body weight, respectively, and N_{days} is the number of days between W1 and W2. W1 was set to 0.18 g, representing the average weight at which feeding typically begins (HM, personal communication with the participating farming companies). W2 was defined as the mean body weight at the first sampling point, approximately four weeks after sea transfer. Consequently, AGR includes a short period of early marine growth. If systematic differences in sea temperature had occurred during this early marine phase, AGR estimates could theoretically be influenced. However, sea temperatures in the Faroe Islands are relatively stable throughout the year and show limited spatial variation among fjords (approximately 6 °C in March to 11 °C in September) (ICES, 2023). Sites 1 and 2 were transferred to sea during the same period (late June–early July 2021), ensuring identical initial marine temperature exposure. Although sites 3 and 4 were transferred in different months (early December and late May 2021, respectively), the mean sea temperatures during the first month post-transfer were similar (7.7 °C and 7.1 °C). The resulting temperature difference (0.65 °C) over this short duration is unlikely to have affected relative growth rankings, particularly given that the vast majority of biomass gain occurred during the 15–20 month hatchery phase.

For the marine phase, the growth rate represented the mean growth between the two sampling points, corresponding to approximately nine months at sea. Here, TGC was used to define growth, and the farming companies provided data about sea temperature.TGC was calculated as follows:

$$TGC = \frac{W2^{\frac{1}{3}} - W1^{\frac{1}{3}}}{T\,x\,N_{days}} \times 1000$$

Where W1 and W2 represent the mean weight at sampling point 1 and 2. N_{days} is the number of days between samplings, and T is mean sea temperature (°C) between sampling points.

In addition to TGC, the mean weight of the fish just before slaughter was compared to the expected weight based on the Laksur 2.0 model. This model, developed by Rúni Dam of the Faroese benchmarking company Avrik, is based on growth data collected at sea from Faroese salmon farms between 2005 and 2022, providing a robust reference for evaluating growth performance at sea. Although unpublished, access to the model was granted, allowing assessment of whether the fish in this study were above or below the expected size just before slaughter, i.e., whether they experienced faster or slower growth than anticipated. The average weights at both sampling points, along with their corresponding sampling dates, were input into the model. The model then incorporated the daily temperature data for the period to account for temperature fluctuations throughout the year. A result of 100% means the fish reached their expected weight. Below and above 100% means they are smaller and larger than expected, respectively.

2.3. Histological processing and staining

The main coronary artery runs along the *bulbus arteriosus*, a highly elastic chamber between the ventricle and the ventral aorta. The *bulbus arteriosus*, with the main coronary artery attached, was carefully cut into three equal-sized pieces (Fig. 1), allowing lesions to be assessed at three locations along the coronary artery. The three pieces were processed in a Thermo Scientific Excelsior tissue processor (Thermo Scientific, Boston, MA, USA) and embedded in paraffin Histowax (Histolab, Askim, Sweden) using a Tissue–Tek, TEC 5 embedding center (Sakura, Alphen aan den Rijn, Netherlands).

Six serial sections (3 μm) were cut from all three pieces (18 serial sections in total) of the *bulbus arteriosus* using a Leica RM 2255 microtome (Leica Biosystems, Buffalo Grove, IL, USA) (Fig. 1). If the *bulbus arteriosus* was too small to divide into three pieces, it was divided in two, and nine serial sections were cut from each piece, giving 18 sections in total. The sections were mounted on glass slides and stained with hematoxylin and eosin (H&E). The slides were scanned in an Aperio GT-450 scanner and examined using Aperio ImageScope software (Leica Biosystems Buffalo Grove, IL, USA). All hearts were processed for histology simultaneously, meaning they were stored in formalin for varying durations, ranging from 2 to 12 months. No noticeable differences in tissue integrity or staining quality were observed in relation to fixation duration.

Considerable variation in the location of the first bifurcation of the main coronary artery was observed (Fig. 2). To ensure sufficient data, a minimum requirement of six serial sections containing the main coronary artery for inclusion was set. This threshold ensured that, despite anatomical variation in bifurcation location, at least one complete and continuous segment of the main coronary artery was captured and could be reliably assessed for arteriosclerotic lesions.

Applying this criterion reduced the initial sample size from 220 fish to 153 fish. Prior to exclusion, analyses were conducted to determine whether coronary anatomy was associated with age, size, or growth-related traits, as such associations could bias estimates of prevalence and severity. No significant differences in bifurcation number were detected between sampling points (younger vs. older fish) within any site (Wilcoxon rank-sum test; all adjusted $p \geq 0.44$). Bifurcation number was not associated with body size (Spearman's $\rho = 0.017$, $p = 0.799$) or growth rate, as neither TGC ($\rho = -0.005$, $p = 0.957$) nor AGR ($\rho = 0.159$, $p = 0.097$) showed significant correlations. Furthermore, comparisons of body length, body weight, condition factor, AGR, and TGC between included and excluded fish revealed no consistent or systematic differences across sites or sampling points.

Because excluded fish lacked sufficient serial sections for reliable lesion assessment in the main coronary artery, with some individuals represented only by sister branches, direct comparison of prevalence and severity between included and excluded fish was not methodologically appropriate. Nevertheless, given the absence of associations between coronary anatomical variation and biological traits known to influence arteriosclerosis in this study, variation in bifurcation number appears to reflect natural anatomical diversity rather than age-, size-, or growth-related remodeling. Accordingly, the applied inclusion criterion is unlikely to have introduced systematic bias into prevalence or severity estimates.

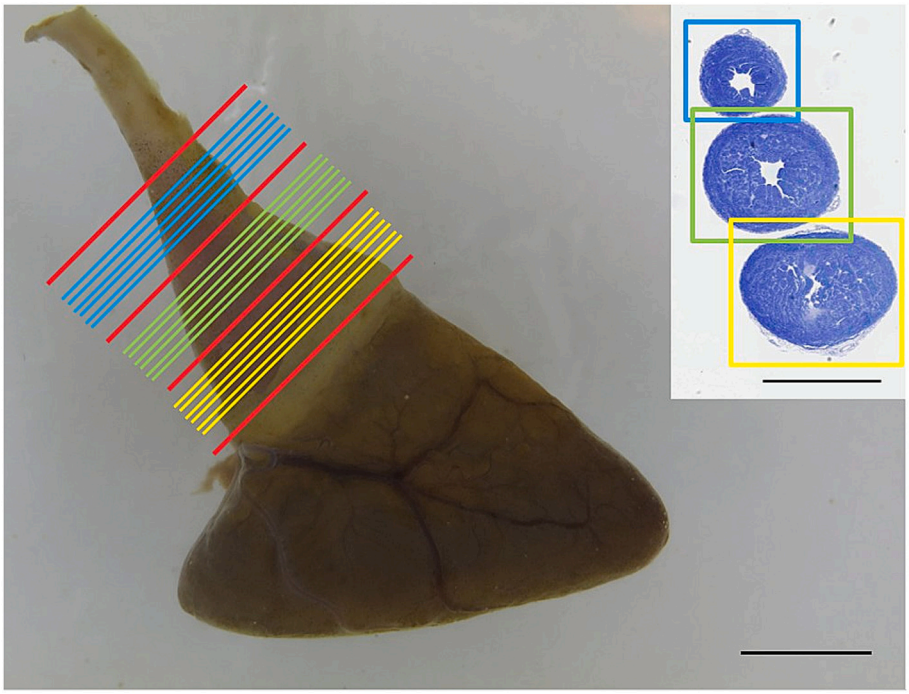

Fig. 1. The *bulbus arteriosus* was divided into three equal-sized pieces, as illustrated by the red lines. From each piece, six consecutive 3 μm serial histological sections were collected, represented by the blue, green, and yellow lines, resulting in 18 sections per heart. The black scale bar at the bottom right corner represents 1 cm. The inset in the top right corner illustrates how the three pieces of bulbus arteriosus tissue were embedded in the same paraffin block before sectioning. The black scale bar in the lower right corner of the inset represents 5 mm. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

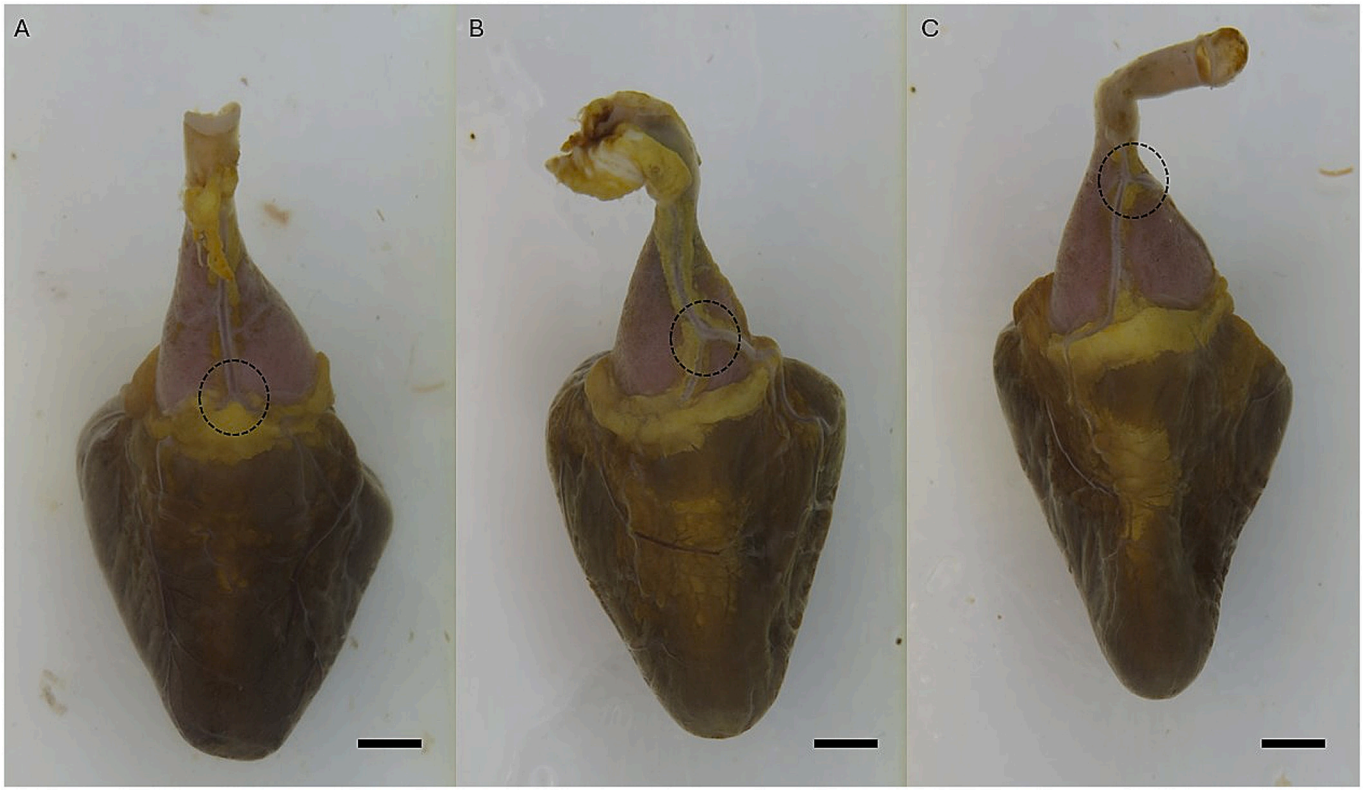

Fig. 2. Examples of anatomical variation in the location of the first bifurcation of the main coronary artery on the *bulbus arteriosus*. A. Bifurcation located caudal on the *bulbus arteriosus*, close to the base of the heart ventricle. B. Bifurcation located in the middle of the *bulbus arteriosus*, and C. Bifurcation located close to the ventral aorta. Black scale bars in the bottom right corner of each image represent 1 cm.

The final sample size from each site is summarized in supplementary data (Table S2). The supplementary data also detail the number and anatomical locations of coronary artery bifurcations on the bulbus arteriosus (Table S1) and the methodology used to determine their positions.

Although not a primary objective, the presence of arteriosclerosis in the sister branches located on the bulbus arteriosus was also evaluated. This assessment was possible for individuals from the second sampling, in which at least six serial sections included sister branches. In these individuals, lesions in the sister branches were compared with those in the main coronary artery. Table S4 in the Supplementary Data summarizes the number of individuals included in this comparative analysis at each site. The corresponding methods and results are also provided in the Supplementary Data.

2.4. Lesion prevalence and lesion incidence

The prevalence of fish that had serial sections containing lesions in the main coronary artery was calculated as follows:

$$\%Prevalence = \frac{number\ of\ fish\ with\ lesions}{total\ number\ of\ fish\ examined}\ x\ 100$$

To assess how much of the artery was affected, the lesion incidence (LI) was calculated (Farrell et al., 1986). This was done by determining the percentage of serial sections that contained arteries with lesions as follows:

$$\%LI = \frac{number\ of\ serial\ sections\ with\ a\ lesion}{total\ number\ of\ serial\ sections\ examined}\ x\ 100$$

2.5. Lesion severity

In previous salmonid studies, the severity of arteriosclerosis in the main coronary artery has been assessed using somewhat similar grading systems, albeit with variations (Farrell et al., 1986; Saunders and Farrell, 1988; Farrell et al., 1990; Seierstad et al., 2008; Dalum et al., 2016; Brijs et al., 2020). Based on these previous studies, a six-grade system was adopted to assess coronary arteriosclerosis in each serial section, as described in Fig. 3.

Farrell et al. (1986) developed a salmonid lesion severity index (LSI),

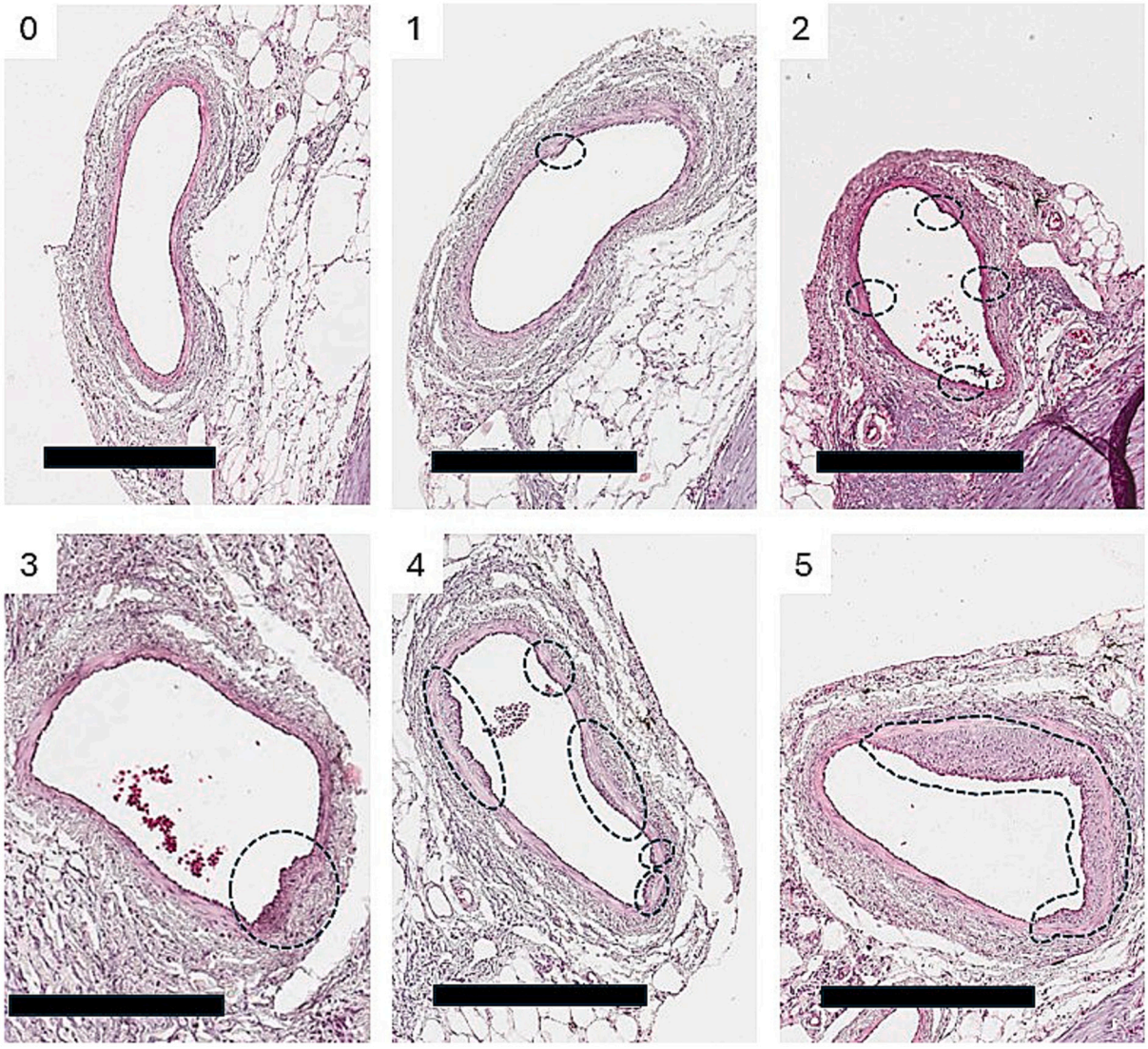

Fig. 3. Grading of the severity of arteriosclerosis in the main coronary artery of Atlantic salmon from 0 to 5. Grade 0: no arteriosclerotic lesions. Grade 1: a single small area with intima proliferation of smooth muscle cells. Grade 2: multiple small intima proliferations. Grade 3: a single large area with intima proliferation of smooth muscle cells. Grade 4: multiple small and/or large intima proliferations affecting up to 50% of the inner vessel wall., Grade 5: multiple or large intima proliferations occupying more than 50% of the inner vessel wall. Black scale bars represent 400 μm.

calculated as the mean of all grades above 0. However, this approach does not capture the overall severity landscape of the artery, as it excludes lesion-free sections. Consequently, individuals with only a few high-grade lesions may obtain a high LSI, even though most sections contain no lesions. To address this limitation, the lesion severity per fish was calculated as the mean of all grades assessed, including grade 0. By incorporating both affected and unaffected regions, this measure captures the combined extent and severity of arteriosclerotic changes and provides a more representative estimate of the overall lesion burden within the main coronary artery.

2.6. Coronary lumen reduction

In each fish with lesions present, the serial section with the most severe lesion was selected, and the extent of arterial lumen area reduction was quantified. Using ImageJ (NIH, Bethesda, MD, USA), (1) the total inner arterial lumen area excluding smooth muscle proliferation (unblocked lumen) and (2) the total inner arterial lumen area including the blockage were measured. Measurements were calibrated using scale bars from slide scans. The extent of lumen area reduction caused by smooth muscle cell proliferation was then calculated as follows:

$$\%lumen\ reduction = \frac{Total\ inner\ area\ without\ blockage - Total\ inner\ area\ with\ blockage}{Total\ inner\ area\ without\ blockage}\ x\ 100$$

Lesion-free fish were excluded from site-level mean calculations.

2.7. Statistical analysis

Statistical analyses were performed using R software (R Core Team, 2025).

The two age groups (sites 1 vs. 2, and sites 3 vs. 4) were compared for body weight, fork length, CF, ventricular weight, AGR (hatchery growth), TGC (marine growth), LI, severity (mean of grades), and arterial lumen area reduction.

Prior to analysis, homogeneity of variance was evaluated using Levene's test(car package; Fox and Weisberg, 2019), and normality was assessed visually using QQ plots(ggpubr package; Kassambara, 2020). Body weight, fork length, CF, ventricular weight, AGR, and TGC were normally distributed; however, some variables did not meet the equal variance assumption. Therefore, Welch's *t*-test (pairwise comparison) was used to assess group differences for these variables (stats package; R Core Team, 2025).

LI, severity, and arterial lumen area reduction did not consistently meet the assumptions of normality and homogeneity of variance. Accordingly, group differences were assessed using the Mann–Whitney *U* test (Wilcoxon rank-sum test) (stats package; R Core Team, 2025).

To examine associations between growth rates and lesion burden, simple linear regression analyses were conducted within each age group with growth rate (AGR or TGC) as the independent variable and LI, severity, and arterial lumen area reduction as dependent variables. Analyses were performed using the lm() function in base R. Residual plots were examined to verify assumptions of linearity and homoscedasticity.

Because multiple statistical tests were conducted, the false discovery rate (FDR) was controlled using the Benjamini–Hochberg procedure. Tests were grouped into families according to the type of statistical analysis: (i) comparisons of body size and growth traits between sites, (ii) comparisons of LI, severity, and lumen area reduction between sites, and (iii) regression analyses examining associations between growth rate and lesion metrics. FDR adjustment was applied within each age group and sampling point. Statistical significance was determined at an FDR-adjusted threshold of $p < 0.05$. Tables S2 and S3 in the supplementary data summarize all statistical outcomes.

3. Results

3.1. Age, size and growth rates at the start of the marine phase

The age, size, and growth rates at the start of the marine phase, as well as the results from the statistical analyses, are summarized in Fig. 4A and Table S2. At the start of the marine phase, at the first sampling, the fish at sites 3 and 4 were 20.7 and 21.6 months old, respectively, while the younger fish from sites 1 and 2 were 15.6 and 16.1 months old, respectively. The similarly aged fish at sites 3 and 4 exhibited significantly different growth rates during their time at the hatchery. Site 3 had a higher AGR of 1.2 ± 0.3 g/day compared to 0.8 ± 0.4 g/day at site 4. This meant that fish at site 4 were significantly smaller at the start of their marine phase, with lower body weight, fork length, and ventricular weight, than those at site 3. They also had significantly lower CF. Thus, fish at site 4 were more comparable in size to the five-month-younger fish from sites 1 and 2. Fish at sites 1 and 2 had experienced similar AGR at the hatchery of 1.1 ± 0.2 g/day and 1.0 ± 0.4 g/day, respectively, and had similar body weight, fork length, CF, and ventricular weight at the start of their marine phase.

3.2. Coronary lesions at the start of the marine phase

The prevalence, LI, severity, and reduction in arterial lumen area for the most severe lesions at the start of the marine phase are summarized in Fig. 5A and Table S3. At the start of the marine phase, the faster-growing fish from site 3 had a prevalence of arteriosclerosis of 67%, compared to 26% at site 4 with slower-growing fish. Similarly, LI was significantly higher at site 3 than at site 4 (25.6 ± 22.5% vs. 5.7 ± 13.8%). The severity was also greater at site 3 than at site 4 (mean grades of 0.8 ± 0.8 vs. 0.1 ± 0.3), and site 3 was the only site with lesions graded 5. In addition, the arterial lumen area reduction for the most severe lesions was also greater at site 3 than at site 4 (13.5 ± 11.0% vs. 4.4 ± 1.9%). At sites 3 and 4, AGR significantly explained 34% of the variation in LI ($R^2 = 0.34$, FDR-adjusted $p < 0.001$), 31% of the variation in the mean of lesion grade ($R^2 = 0.31$, FDR-adjusted $p < 0.001$), and 17% of the variation in maximum arterial lumen reduction ($R^2 = 0.17$, FDR-adjusted $p = 0.007$).

Similar to body size, the prevalence and severity of arteriosclerosis at site 4 were more comparable to those of the younger fish at sites 1 and 2, where the prevalence was 29% at both sites, with similar LI (5.4 ± 10.5% vs. 5.1 ± 9.4%), severity (mean grades of 0.1 ± 0.3 vs. 0.2 ± 0.3), and lumen area reduction (13.1 ± 12.9% vs. 7.3 ± 1.9%). At sites 1 and 2, AGR showed no association with LI, severity, or lumen area reduction.

3.3. Age, size and growth rates at the end of the marine phase

The age, size, and growth rates at the end of the marine phase are summarized in Fig. 4B and Table S2. By the end of the marine phase, at the second sampling, the fish at sites 3 and 4 were 29.4 and 30.3 months old, respectively, while the younger fish from sites 1 and 2 were 24.8 and 24.6 months old. Site 3, which initially had a higher growth rate during

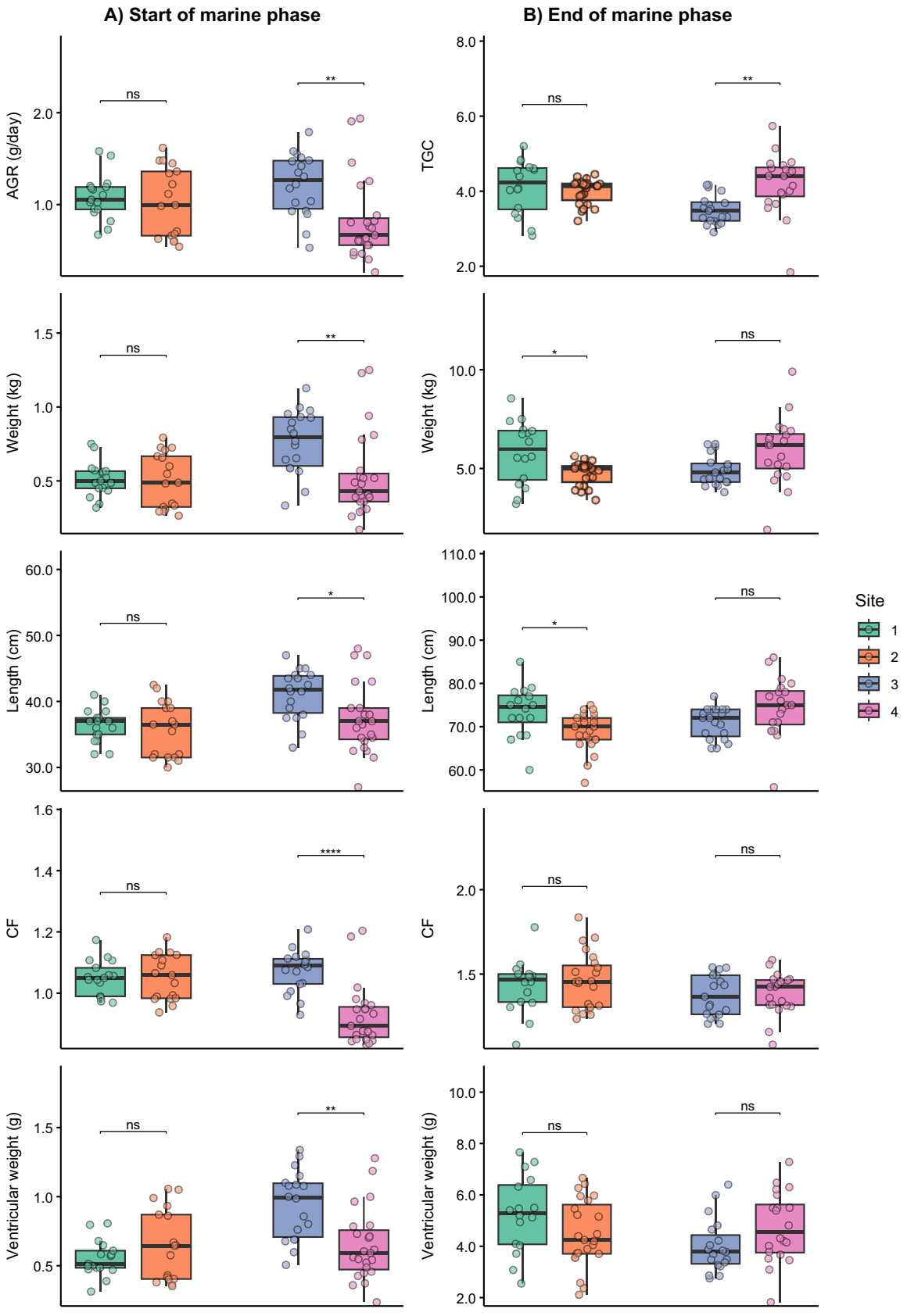

Fig. 4. Absolute growth rate (AGR), thermal growth coefficient (TGC), body weight, fork length, condition factor (CF), and ventricular weight in Atlantic salmon, grouped by age (sites 1 vs. 2 with younger fish and sites 3 vs. 4 with older fish) at the two sampling points: (A) start of marine phase and (B) end of marine phase. Each boxplot shows the median (horizontal line), interquartile range (box), and individual values (dots). Statistical significance between sites within age groups is indicated: * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; **** $p < 0.0001$; ns = not significant.

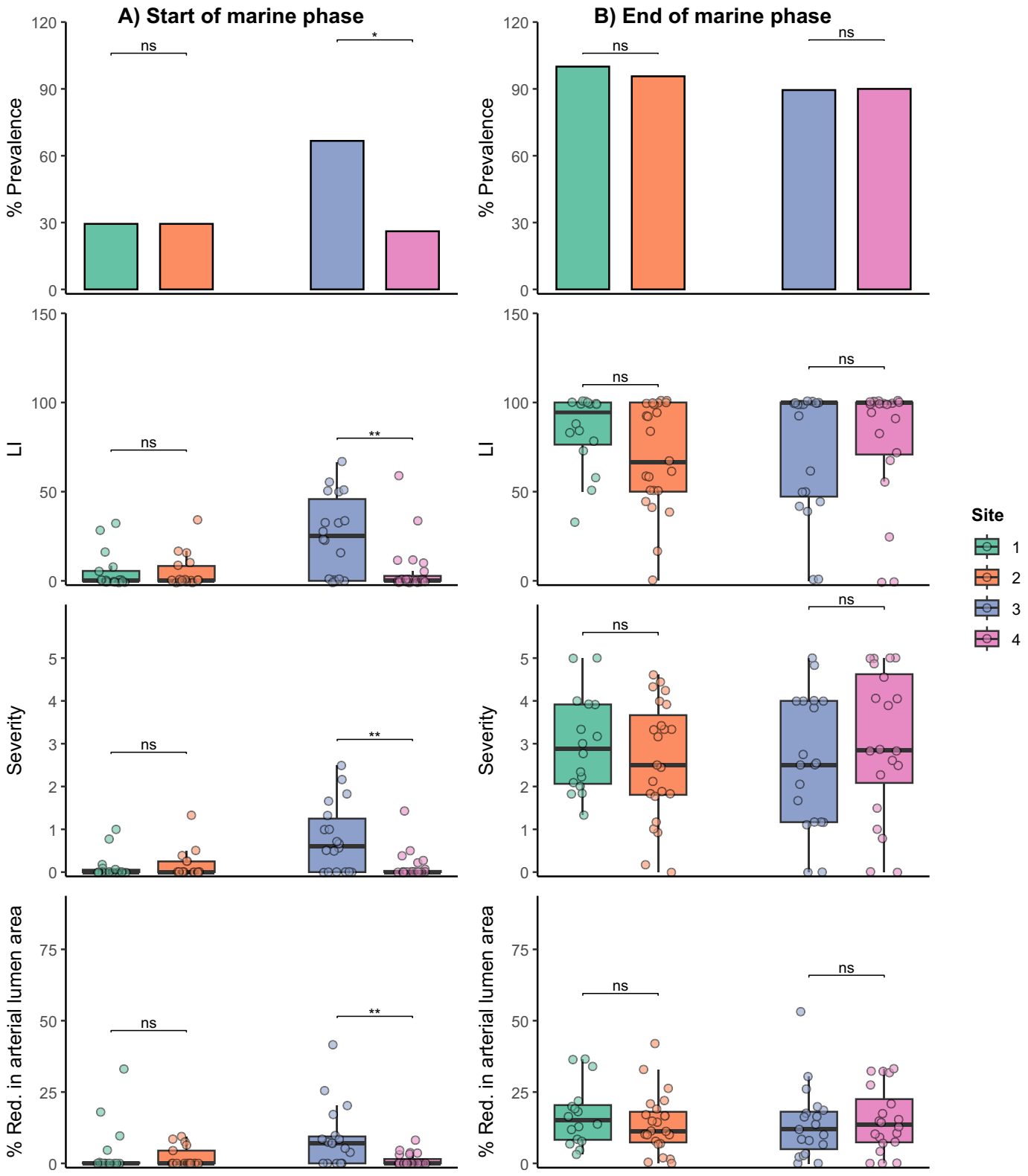

Fig. 5. Prevalence of individuals with coronary arteriosclerosis, lesion incidence (%LI), severity (mean lesion grade), maximum arterial lumen area reduction in individuals with lesions and the corresponding mean theoretical blood flow reduction, grouped by age (sites 1 vs. 2 with younger and sites 3 vs. 4 woth older fish) at two sampling points: (A) start of marine phase and (B) end of marine phase. The boxplots show the median (horizontal line), interquartile range (box), and individual values (dots). Statistical significance between sites within age groups is indicated: Statistical significance between sites within age groups is indicated: * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; **** $p < 0.0001$; ns = not significant.

the hatchery phase and were initially larger than site 4, exhibited a significantly lower growth rate during the marine phase compared with site 4 (TGC: 3.5 ± 0.4 vs. 4.2 ± 0.8). According to the Laksur 2.0 model, site 3 fish were now 14.6% below their expected size, while site 4 fish exceeded their expected weight by 22.7%, reflecting two distinct growth patterns. As a result of these changing growth patterns, no significant differences in body weight, fork length, CF, or ventricular weight were observed between sites 3 and 4 at the end of the marine phase.

Between sites 1 and 2, there was no significant difference in TGC (4.1 ± 0.7 vs. 3.9 ± 0.3), condition factor, or ventricular weight. However, fish at site 1 had significantly higher body weight and fork length, indicating faster growth. This pattern was also reflected in the Laksur 2.0 growth model, where site 1 fish were 19.7% above their expected weight, while site 2 fish were only 4.3% below their expected weight.

3.4. Coronary lesions at the end of the marine phase

Just before slaughter, the prevalence, LI, severity, and the reduction in coronary artery lumen area had increased across all sites compared to the beginning of the marine phase (Fig. 5B and Table S3). The prevalence now ranged from 90% to 100% across sites, and, in contrast to the sampling at the start of the marine phase, no statistically significant differences in LI, severity, or arterial lumen area reduction were observed between sites within either age group.

The LI values were 84.2 ± 21.0% and 69.4 ± 29.9% at sites 1 and 2, respectively, and 72.5 ± 35.3% and 79.4 ± 22.7% at sites 3 and 4. The severity was 2.9 ± 1.1 and 2.6 ± 1.4 at sites 1 and 2, and 2.5 ± 1.5 and 3.0 ± 1.7 at sites. The lumen reduction was were 19.6 ± 10.8% and 14.6 ± 10.1% at sites 1 and 2, respectively, and 15–6 ± 12.5% and 16.7 ± 10.5% at sites 3 and 4.

TGC significantly explained 16% of the variation observed with LI at sites 1 and 2 ($R^2 = 0.16$, FDR-adjusted $p = 0.03$), but no association was found between TGC and severity or arterial lumen area reduction.

3.5. Comparison of main coronary arteries and sister branches

LI did not significantly differ between the main coronary artery and sister branches, except at site 1, where LI was significantly higher in the main coronary artery (Fig. S2). No significant differences were observed in arterial lumen area reduction between the main coronary artery and sister branches (Fig. S3).

4. Discussion

4.1. Higher hatchery growth rates are associated with increased coronary lesion severity at sea transfer

Coronary arteriosclerosis is a cardiovascular disease that progresses with age in Atlantic salmon (Farrell, 2002; Saunders et al., 1992). By narrowing and stiffening the coronary arteries, the condition compromises oxygen delivery to the myocardium, potentially limiting its capacity to sustain adequate function during periods of elevated demand. This impaired capacity is implicated in stress-related mortality events in aquaculture, as evidenced by ventricular myocardial lesions consistent with ischemia observed in fish that have died in association with stressful events (Poppe et al., 2021; Poppe et al., 2007). Rapid growth has long been speculated to aggravate the condition (Saunders et al., 1992; Brijs et al., 2020), but direct evidence has been lacking. The current study provides consistent observational evidence of this association, showing that faster growth rates are associated with more severe coronary lesions in Atlantic salmon.

This was clearly illustrated when comparing sites 3 and 4, where similarly aged fish had experienced different hatchery growth rates (AGR 1.2 g/day vs. 0.8 g/day, respectively). At the beginning of their marine phase, their contrasting coronary lesion status was striking: the prevalence of fish with arteriosclerosis was more than double at site 3 (67% vs. 26%) with faster-growing fish, and the proportion of their main coronary artery affected by these lesions was four times higher (LI 25.6% vs. 5.7%). Fish at site 3 also had a greater severity (0.8 vs. 0.1), and was the only site with grade 5 lesions at this initial sampling point. The reduction in arterial lumen area in the most severe lesions was also higher (13.5% vs. 4.4%). Indeed, hatchery growth rate explained 34% of the variation in LI, 31% in severity, and 17% in arterial lumen area reduction at the beginning of the marine phase. Together, these findings suggest a strong association between hatchery growth rate and impaired coronary health at the onset of the marine phase.

Contrary to sites 3 and 4, the younger and similarly aged fish at sites 1 and 2 exhibited nearly identical hatchery growth rates (AGR 1.0 g/day vs. 1.1 g/day). Interestingly, the prevalence was identical (29%), and LI values were similar (5.4% vs. 5.1%). Additionally, they had similar severity (0.1 vs. 0.2), and the reduction in lumen area in the most severe lesions did not differ (13.1% vs. 7.3%). At sites 1 and 2, AGR showed no association with LI, severity, or lumen area reduction. Taken together, the lack of difference in AGR and lesion outcomes between sites 1 and 2 further supports the observed association between growth rate and coronary lesion development.

Notably, the slower-growing fish at site 4 were more comparable in size and lesion status to the five-month-younger fish at sites 1 and 2. However, the fish at site 4 had a low CF (0.9), close to the threshold of emaciation (Stien et al., 2013). Still, this observation supports the idea that slower growth may benefit coronary health.

4.2. Faster marine growth rates are associated with greater coronary lesion progression

Remarkably, fish at site 4 experienced a shift in growth rate during the marine phase, with site 4 exhibiting faster growth than site 3 (TGC: 4.2 vs. 3.5, respectively). This was also reflected in the Laksur 2.0 model, with site 4 fish being 22.7% above their expected weight, and site 3 fish being 14.6% below their expected weight by the end of their marine phase. This shift in growth pattern resulted in the fish at both sites having similar slaughter weight.

The reason for this change in growth during the marine phase is unclear. It may reflect environmental factors, as fish at site 4 were stocked in May during rising temperatures and favorable growth conditions in the Faroe Islands (ICES, 2023). In contrast, fish at site 3 were stocked in December, when sea temperatures were moderate but followed by progressively colder winter months, which may have influenced subsequent growth. It could also represent compensatory growth, a well-described response in Atlantic salmon whereby fish accelerate feed intake and growth following periods of restriction (Stefansson et al., 2009; Hvas et al., 2022). Even so, it has previously been shown that salmon that grow more rapidly in the hatchery can exhibit reduced growth rate during the marine phase (Frisk et al., 2020; Ytrestølj et al., 2023). This pattern may explain the lower-than-expected weight at site 3 at the end of the marine phase.

Whatever the cause, the higher marine growth rate observed at site 4 was associated with a relatively greater lesion progression than site 3, so that by slaughter, lesion severity at site 4 had converged with that of site 3, mirroring the convergence in body weight observed between the two sites. They displayed similar prevalence (90%), LI (72.5% and 79.4%), severity (2.5 and 3.0), and arterial lumen reduction (15.6% and 16.7%). This convergence in coronary artery status by the end of the marine phase may also explain why TGC was not significantly associated with LI, severity, or arterial lumen reduction at sites 3 and 4. If both sites had had the same coronary status baseline at the beginning of the marine phase, the TGC–lesion relationship would likely have been more pronounced.

Similar to the hatchery growth period, no difference in marine growth rate was found at sites 1 and 2 (TGC 4.1 and 3.9). However, site 1 fish had a larger body size by the end of the marine phase, as reflected in the Laksur 2.0 model, showing that site 1 fish were almost 20% above

their expected weight, while site 2 fish were close to their expected weight. This indicates that, despite similar TGCs, site 1 fish achieved greater overall growth. While no significant difference was found in arteriosclerosis prevalence or severity, TGC accounted for 16% of the variation in LI.

4.3. Other contributing factors beyond growth

Coronary arteriosclerosis in salmonids is characterized by intimal smooth muscle cell proliferation rather than lipid-rich plaque formation (Dalum et al., 2016; Farrell, 2002; Saunders et al., 1992). It has been proposed that these types of lesions develop as a response to vascular injury (Farrell, 2002). This hypothesis is supported by a study on rainbow trout, where mechanical abrasion of the exposed coronary artery in vivo resulted in a marked increase in vascular smooth muscle mitotic activity in subsequent explant cultures (Gong and Farrell, 1995).

One proposed source of such injury is the anatomical position of the main coronary artery along the elastic *bulbus arteriosus*. The *bulbus arteriosus* expands and recoils with each heartbeat to dampen pulsatile pressure, which may subject the coronary artery to repetitive mechanical stretching and injury, particularly during periods of elevated cardiac output (Farrell, 2002). Supporting this mechanistic injury hypothesis, Dalum et al. (2016) demonstrated a correlation between upregulation of stretch-related genes and arteriosclerotic severity in Atlantic salmon.

Consequently, if lesions are linked to mechanical injury, any condition that elevates cardiac workload and vascular strain may influence coronary health and lesion development, including intense exercise and stress. Consistent with this, studies in salmonids have shown that swimming to exhaustion at critical swimming speed significantly increased smooth muscle cell proliferation in coronary explants, whereas prolonged low-intensity swimming did not (Gong et al., 1996). Although the relationship between stress and arteriosclerosis remains unclear, Farrell (2002) suggested that faster growth could lead to a more stressful lifestyle, contributing to greater arteriosclerotic severity.

Taken together, these findings suggest that coronary lesion development in salmonids is likely influenced by multiple interacting factors. The present study was observational in nature, and therefore, it was not possible to fully control all potential confounding variables that may influence coronary health. To minimize such effects, sites were grouped in pairs with comparable genetics, hatchery origins, ages at sea transfer and sampling, and shared company management with similar feeding strategies and farming protocols. Nevertheless, fish were reared at different marine locations where environmental conditions such as temperature, hydrodynamics, and cumulative stress exposure may vary.

While residual confounding effects cannot be excluded under commercial field conditions, the consistent association across paired sites supports the observed relationship between growth rate and coronary lesion development. Future controlled experimental studies will be required to disentangle the independent effects of growth rate from other factors on lesion development and progression.

4.4. Potential consequences for stress resilience

Increasing farm mortality of Atlantic salmon is well documented (Oliveira et al., 2021; Sommerset et al., 2023), and has been linked to compromised cardiovascular health (Poppe et al., 2021; Poppe et al., 2007). However, the observed increase in mortality may also reflect the increasing frequency of stressful farming interventions. In the Faroe Islands, mortality began to rise after the 2009 executive order on lice monitoring and management, which required more frequent delousing. By 2015, many producers had shifted from chemical treatments to mechanical and thermal delousing in response to resistance, a transition that coincided with further increases in mortality (Rúni Dam, Avrik, unpublished). These procedures are highly demanding and place acute stress on the cardiovascular system. It is therefore possible that mortality increases are driven less by the severity of lesions themselves and more by the fact that fish are exposed to stressors more often and more intensely than before. At the same time, the explanation may be a combination of both factors: fish with compromised coronary status may have coped under previous conditions, but now succumb when challenged by more frequent acute stress.

A direct way to test this hypothesis would be to conduct comparative analyses of fish that succumb during acute farming stressors (e.g., delousing, crowding, pumping) and those that survive the same events. Such studies could combine detailed histological assessment of coronary lesion severity with physiological stress indicators, including plasma cortisol and blood lactate concentrations, to determine whether individuals with greater coronary compromise exhibit reduced stress tolerance. Integrating pathological and physiological endpoints under standardized challenge conditions would provide a more mechanistic understanding of how coronary arteriosclerosis may contribute to mortality risk under modern farming practices.

Beyond event-based comparisons, another complementary approach would be to investigate how different growth rates influence coronary arteriosclerosis and whether variation in lesion burden translates into differences in cardiorespiratory performance under acute stress. Cardiorespiratory performance and physiological stress responses were not assessed in the present study. However, Brijs et al. (2020) demonstrated that farmed rainbow trout unable to endure a 30-min confinement stress test exhibited coronary lumen reductions of 26–39%, whereas surviving fish had healthier coronaries. Across sites in this study, 14–28% of Atlantic salmon, just before slaughter, had lumen reductions exceeding 26% in the most severe lesions. Thus, if Atlantic salmon respond similarly to coronary narrowing, this could indicate that up to one in four fish may have reduced stress resilience under current Faroese farming conditions. Further research integrating growth history, coronary morphology, and functional stress testing is therefore needed to determine how production strategies influence the overall robustness of farmed salmon.

4.5. New insights into coronary artery anatomy

This study also provides new insights into coronary artery anatomy. Previous work has focused almost exclusively on the main coronary artery located on the *bulbus arteriosus*, partly because it was assumed that the first bifurcation occurs close to or on the ventricle, making the main artery easier to sample and analyze histologically (Farrell et al., 1986; Farrell, 2002; Seierstad et al., 2005). The findings from this study challenge this assumption, at least for the Atlantic salmon strain studied here. Among the 220 fish initially sampled, only 31.8% had a single, unbranched coronary artery along the entire length of the *bulbus arteriosus*. In most fish, the artery bifurcates more cranially, often giving rise to at least two branches near the ventricle.

Importantly, when comparing the main artery to the branching arteries located on the *bulbus arteriosus,* no significant difference in LI was found, except at site 1, where LI was slightly higher in the main artery. Likewise, no significant differences were observed in arterial lumen reduction in the most severe lesions. The coronary branches located on the ventricle itself were not examined, and so it remains uncertain whether they follow the same pattern as the vessels on the *bulbus arteriosus*. Seierstad et al. (2005), however, reported that arteriosclerosis in the smaller ventricular branches was negligible compared to the larger main branches on the *bulbus arteriosus*. This discrepancy could reflect differences in mechanical strain, supporting the idea that lesions in the main coronary artery arise from repetitive stretching due to its position on the *bulbus arteriosus* (Farrell, 2002). Seierstad et al., 2008 also reported that coronary lesions in the branching arteries were typically located toward the outer vessel walls where the artery branches, forming ‘cushions’ or ‘pads’ as they progress (Seierstad et al., 2008). The authors hypothesized that this phenomenon could be due to altered shear stress resulting from the division of blood flow. In this study, these cushions on the outer wall of the branches located at the apex of branching were not

observed; instead, lesions appeared more randomly distributed throughout the arterial intima, even in fish with severe lesions.

5. Conclusion

In conclusion, the present study demonstrates consistent associations between higher growth rates during the hatchery phase and more advanced coronary arteriosclerotic status at sea transfer, as well as between higher marine growth rates and subsequent lesion progression. These findings indicate that final coronary status prior to slaughter reflects cumulative growth across both production phases.

Although causality cannot be easily established under field conditions, the observed patterns suggest that intensified production strategies may be associated with increased cardiovascular burden. These findings highlight the importance of incorporating cardiovascular health considerations into growth management decisions. While reducing growth excessively may not be desirable from a production perspective, this study suggest that consistently high growth across both production phases may not be biologically neutral. Defining growth rates that balance production efficiency with long-term cardiovascular robustness represents an important direction for future research.

Declaration of generative AI and AI-assisted technologies in the manuscript preparation process

During the preparation of this work, the authors used ChatGPT and Grammarly in order to improve grammar, clarity, and the overall flow of already written text. After using these tools, the authors reviewed and edited the content as needed and take full responsibility for the content of the published article.

CRediT authorship contribution statement

Heidi S. Mortensen: Writing – review & editing, Writing – original draft, Visualization, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Helene Wisløff:** Writing – review & editing, Supervision, Investigation, Data curation. **Lucas A. Zena:** Writing – review & editing, Supervision. **Andreas Ekström:** Writing – review & editing, Supervision. **Albin Gräns:** Writing – review & editing, Supervision. **Rúni Dam:** Writing – review & editing, Resources. **Elin Egholm:** Investigation, Data curation. **Amanda Vang:** Writing – review & editing, Supervision. **Ida B. Johansen:** Writing – review & editing, Supervision. **Erik Sandblom:** Writing – review & editing, Supervision, Conceptualization.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.aquaculture.2026.744394.

Data availability

The data that support the findings of this study are not publicly available. These raw data were obtained from commercial aquaculture companies under confidentiality agreements and include proprietary information about the companies' farming practices. The companies have deemed the data commercially sensitive and have not granted permission for its public release. As a result, the raw datasets cannot be made available.

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