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Communication

First Detection and Seasonal Dynamics of Proliferative Kidney Disease in Northwestern Italy

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Abstract

Proliferative kidney disease (PKD), caused by the myxozoan parasite *Tetracapsuloides bryosalmonae*, represents a major threat to salmonid populations and aquaculture, particularly under warming environmental conditions. In Italy, its distribution remains poorly documented. This study aimed to update the epidemiological status of PKD by investigating its occurrence in a trout farm (Piedmont, northwestern Italy), a previously unreported area. A total of 787 individuals of *Oncorhynchus mykiss* were sampled between May and October 2024 across three seasonal periods. Fish were examined using necropsy, bacteriological, and molecular analyses. Water temperature ranged from 12.4 to 20.3 °C, showing a clear seasonal trend. Overall PKD prevalence was 42.3%, with significant temporal variation, increasing from 23.0% in early summer to 73.0% in late summer, and declining to 3.9% in autumn. A multivariable logistic regression model showed that PKD-positive fish had a lower probability of presenting macroscopic lesions, while sampling period was the main predictor of infection. These results revealed a temporal mismatch between pathological and molecular findings, highlighting the need for further histopathological validation of the observed seasonal patterns. This study provides the first evidence of PKD in southwestern Piedmont, extending the currently documented distribution of the disease in Italy and highlighting the need for enhanced surveillance under climate change.

Keywords: epidemiology; host–parasite interaction; trout farms; salmonids

Key Contribution: This study provides the first molecular evidence of proliferative kidney disease (PKD) in southwestern Piedmont (northwestern Italy), expanding the known distribution of *Tetracapsuloides bryosalmonae*. It also characterizes the seasonal dynamics of PKD in farmed rainbow trout and identifies a temporal mismatch between molecular detection and pathological findings, providing new epidemiological insights into PKD seasonal dynamics and supporting enhanced surveillance under climate change.

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1. Introduction

Proliferative kidney disease (PKD) is one of the most significant diseases affecting salmonids, with substantial impacts on both wild and farmed populations [1]. The disease is caused by the myxozoan parasite *Tetracapsuloides bryosalmonae* and is characterized by pronounced renal enlargement accompanied by a severe inflammatory response. Clinically, proliferative kidney disease (PKD) is characterized by renal enlargement and systemic alterations, including gill anemia, exophthalmos, splenomegaly, and cachexia, which may ultimately result in mortality [2]. Under favorable environmental conditions, severe outbreaks can occur in rainbow trout (*Oncorhynchus mykiss*), causing considerable economic losses in aquaculture [3].

Tetracapsuloides bryosalmonae has a two-host life cycle involving freshwater bryozoans and salmonid fish [4,5]. Following entry through the gills [6], the parasite develops primarily in the kidney and may disseminate to secondary organs such as the spleen and liver [7,8]. Host competence differs among salmonids: brown trout (*Salmo trutta*) and brook trout (*Salvelinus fontinalis*) act as carrier hosts capable of completing the parasite life cycle [9], whereas rainbow trout is considered a dead-end host because parasite development remains incomplete [10]. These species-specific differences are associated with distinct host immune responses and parasite gene expression patterns [11–13].

PKD is strongly influenced by water temperature and exhibits marked seasonal dynamics. In rainbow trout, infections remain limited below 12–13 °C but increase rapidly above 15–16 °C, with peak parasite development and kidney pathology generally occurring between 18 and 20 °C [14,15]. Elevated temperatures also promote parasite proliferation in bryozoans, increasing infection pressure [16], while accelerating disease progression and host inflammatory responses [17,18]. Consequently, climate change is expected to enhance PKD emergence by prolonging periods of favorable temperatures and facilitating the expansion of the parasite's geographic range [1,19,20]. Indeed, increasing temperatures have been associated with higher PKD prevalence, disease severity, and mortality in European salmonid populations [21,22].

Since its first formal description in the 1970s [23,24], PKD has been reported across Europe and North America. Phylogenetic studies have identified distinct European and North American lineages of *T. bryosalmonae* [10], and the parasite has recently expanded its distribution to several additional European regions, including Estonia and Norway [4,25].

In Italy, available data on PKD remain limited and geographically fragmented. The first national investigation, conducted between 2002 and 2004, confirmed the presence of the parasite as a cause of summer mortality in farmed rainbow trout across several regions of Northern Italy, except for Trentino-Alto Adige [26]. More recent studies, however, have reported a positivity rate of 11% for *T. bryosalmonae* in wild brown trout sampled from the four main rivers of South Tyrol [27].

In this context, the present study aimed to update the current epidemiological status of PKD in Italy. Specifically, fish were sampled from a trout farm located in the Piedmont region, following reports from the farmer of typical clinical signs consistent with PKD. Sampling was conducted across three distinct periods to capture seasonal dynamics: period 1 (May–June), period 2 (July–August), and Period 3 (September–October). Furthermore, this study reports the first documented case of PKD in a previously unaffected area, extending the currently documented distribution of PKD in northwestern Italy.

2. Materials and Methods

2.1. Sampling Design

Between May and October 2024, a total of 787 individuals of *Oncorhynchus mykiss* were sampled from a trout farm located on the border between the provinces of Turin and

Cuneo, in the southern sector of the Po River basin (Piedmont, Italy), as part of routine health monitoring activities within the framework of the project "RESILTROUT: aquaculture resilient to global changes: research to support the Italian trout supply chain" (see Institutional Review Board Statement). The facility is situated in a rural area characterized by a high density of agricultural and livestock activities, a semi-continental climate, and marked seasonal variations in water temperature. The farm is supplied with surface water.

Sampling was carried out across three distinct periods to account for seasonal variability: period 1 (May–June; n = 270), period 2 (July–August; n = 363), and period 3 (September–October; n = 154). During the survey period, water temperature was monitored daily using a field probe (Figure 1).

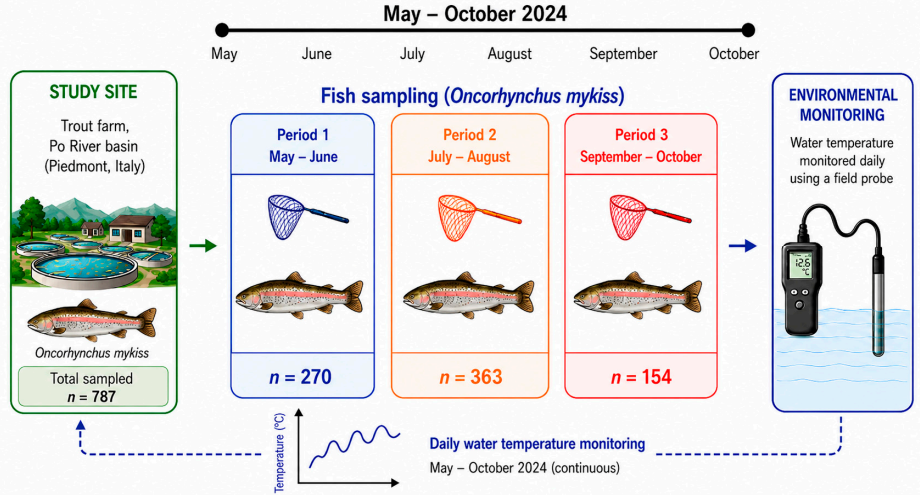

Figure 1. Schematic representation of the sampling design and environmental monitoring.

2.2. Fish Sampling and Processing

Biometric features (length and weight) were recorded for each specimen. The health status of the sampled fish was assessed through comprehensive external and internal examinations. Parasitological and bacteriological analyses were conducted following standard diagnostic procedures [28]. The fish had been frozen by the farmer immediately after collection before delivery to the Fish Diseases Laboratory of Istituto Zooprofilattico Sperimentale del Piemonte, Liguria e Valle d'Aosta (IZSPLV, Turin, Italy), precluding subsequent histopathological analyses. A pool of posterior kidney and spleen was aseptically collected from each specimen and stored at −20 °C for molecular analysis. DNA was extracted from kidney tissue using a KingFisher™ 96 Flex automated extractor and the MagMAX™ CORE Nucleic Acid Extraction Kit (Thermo Fisher Scientific, Waltham, MA, USA), following the manufacturer's instructions. Detection of *Tetracapsuloides bryosalmonae* was performed using the specific primers PKX-5F (5′-CCTATTCAATTGAGTAGGAGA-3′) and PKX-6R (5′-GGACCTTACTCGTTTCCGACC-3′), targeting a 435-bp fragment of the SSU-rDNA gene, according to [29].

To assess the presence of ectoparasites, skin mucus and gill filament scrapings were collected and mounted in a drop of water on a glass slide, covered with a coverslip to obtain fresh wet-mount preparations, and examined under an optical microscope (Olympus BX40, Olympus, Tokyo, Japan). To exclude concurrent bacterial infections, bacteriological analyses were performed on kidney samples. These were aseptically streaked onto Columbia Blood Agar (CBA) plates and incubated at 22 °C for up to 72 h. Plates were checked daily for bacterial growth and colony morphology. Dominant colonies were subcultured on CBA and identified by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS; Bruker Daltonics Inc., Billerica, MA, USA).

2.3. Statistical Analysis

The distribution map was obtained using QGIS Software (version 3.40.11) [30]. The databases used for river allocations and region boundaries were the Italian National Geographical Portal and the Infrastructure for Spatial Information in Europe website (INSPIRE). The PKD distribution range was determined based on the geographic coordinates obtained from the present analyses and reports from the literature [26]. Data were expressed as percentages for categorical variables and as mean ± standard deviation (SD) for continuous variables. Differences in PKD prevalence among sampling periods were assessed using the chi-square (χ^2) test. Pairwise comparisons between periods were performed using chi-square tests with Bonferroni correction.

To account for potential confounding effects of seasonal variation, a multivariable logistic regression model was performed, with PKD positivity (binary outcome) as the dependent variable and sampling period (categorical variable with three levels) and the occurrence of gross lesions (presence/absence) as independent variables.

An additional multivariable model was fitted including the main individual lesion types (renal enlargement, splenomegaly, hepatic anemia, enteritis, and exophthalmos) as predictors, together with sampling period.

Model results were reported as adjusted odds ratios (OR) with 95% confidence intervals (CI). All statistical tests were two-tailed, and differences were considered statistically significant at $p < 0.05$. All statistical analyses were performed using R software (v. 4.4.2) [31].

3. Results

3.1. Water Temperature and Fish Biometrics

Mean monthly water temperatures were 12.4 ± 1.2 °C (May), 14.2 ± 1.0 °C (June), 20.3 ± 1.5 °C (July), 19.0 ± 1.3 °C (August), 17.2 ± 1.1 °C (September), and 13.9 ± 0.9 °C (October), showing a clear seasonal trend with a summer peak. Mean fish weight and length were 59.93 ± 33.45 g and 16.09 ± 2.42 cm in period 1, 94.67 ± 38.89 g and 17.55 ± 2.61 cm in period 2, and 180.92 ± 34.45 g and 20.00 ± 3.54 cm in period 3.

3.2. Necropsy Examination and Parasitological Analysis

Necropsy examination identified the specimens exhibiting macroscopic lesions across the three periods, as shown in Table 1. All specimens tested negative for ectoparasites.

Table 1. Prevalence (%) of gross pathology observed in rainbow trout (*Oncorhynchus mykiss*) sampled during three distinct periods: period 1 (May–June), period 2 (July–August), and period 3 (September–October).

Lesion	Period 1	Period 2	Period 3
Kidney enlargement	78.9%	10.5%	16.9%
Splenomegaly	42.2%	7.4%	24.7%
Hepatic anemia	19.6%	1.4%	2.6%
Enteritis	38.2%	4.4%	3.9%
Exophthalmos	1.1%	0.8%	8.4%

Renal enlargement was detected in 78.9% of the fish during period 1 (May–June), decreasing to 10.5% in period 2 (July–August) and slightly increasing to 16.9% in period 3 (September–October), with an overall mean prevalence of 35.2%. Splenomegaly was observed in 42.2% of the fish in period 1, 7.4% in period 2, and 24.7% in period 3, resulting in a total prevalence of 22.7%. Hepatic anemia was recorded in 19.6% of the specimens in period 1, 1.4% in period 2, and 2.6% in period 3, for an overall prevalence of 7.9%. Enteritis was documented in 38.2% of the fish in period 1, declining to 4.4% in period 2 and 3.9% in

period 3, with an overall prevalence of 15.9%. Exophthalmos was detected in 1.1% of the fish in period 1, 0.8% in period 2, and 8.4% in period 3, resulting in a total prevalence of 2.4%. Representative photographs of the main gross pathological findings observed during the study are presented in Figure 2a–d.

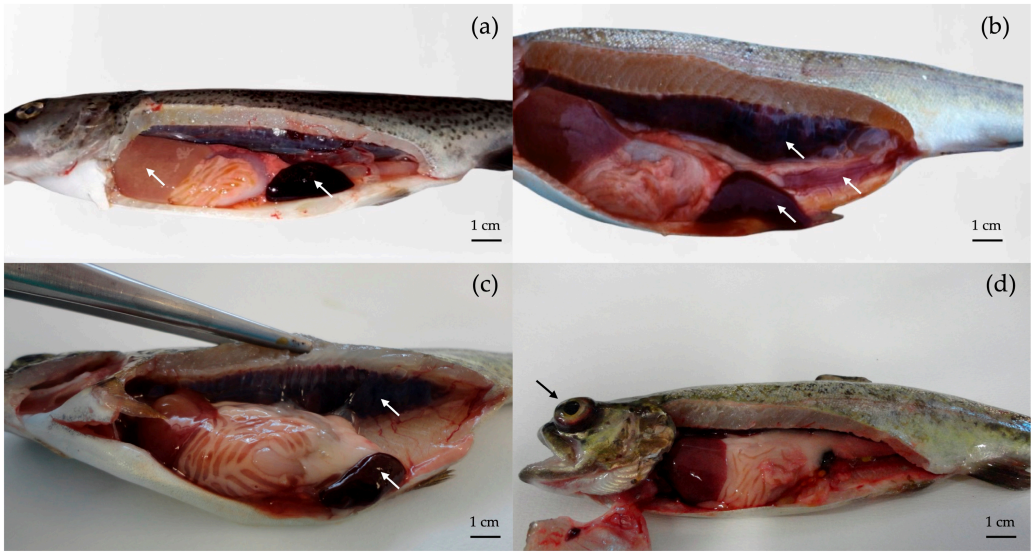

Figure 2. Representative gross pathological lesions observed in rainbow trout (*Oncorhynchus mykiss*) affected by proliferative kidney disease (PKD). (**a**) Hepatic anemia (left white arrow) and enlarged spleen (right white arrow); (**b**) enlarged kidney (upper white arrow), enteritis (right white arrow), and enlarged spleen (lower white arrow); (**c**) enlarged kidney (upper white arrow) and enlarged spleen (lower white arrow); (**d**) Exophthalmos (black arrow).

3.3. Bacteriological Analysis

Bacteriological analyses, carried out on the kidneys of each specimen subjected to necropsy, tested negative in most samples examined (70.2%). Among the positive samples, the most prevalent isolates were bacteria of limited clinical significance: *Lactococcus lactis* (3.8%), *Aeromonas* spp. (2.5%), *Hafnia alvei* (1%), *Lactococcus raffinolactis* (1.1%) and *Acinetobacter* spp. (1.02%). Coinfection between bacteria and PKD was observed in 18.6% positive cases during period 1 and 10.6% during period 2, whereas no coinfections were detected in period 3.

3.4. Molecular Analyses and Statistical Results

Molecular analyses revealed distinct temporal patterns in PKD positivity. Positive samples showed the presence of the expected 435-bp amplicon. The prevalence of PKD differed significantly among sampling periods ($\chi^2 = 272.1$, df = 2, $p < 0.001$), with a marked increase from period 1 (23.0%) to period 2 (73.0%), followed by a sharp decline in period 3 (3.9%).

A multivariable logistic regression model was applied to evaluate the association between PKD positivity, gross lesions, and sampling period. After adjusting for sampling period, PKD-positive fish showed a significantly lower probability of presenting macroscopic lesions (adjusted odds ratio = 0.52, 95% CI: 0.36–0.75, $p < 0.001$).

Sampling period was identified as the main factor influencing PKD positivity. Compared to period 1, the probability of PKD positivity was significantly higher in period 2 (OR $\approx$ 8.9, $p < 0.001$) and significantly lower in period 3 (OR $\approx$ 0.14, $p < 0.001$). In the multivariable model including individual lesion types, renal enlargement remained significantly associated with PKD positivity (OR $\approx$ 0.41, $p < 0.001$), whereas no significant associations were observed for the other lesions.

4. Discussion

From an epidemiological perspective, the detection of *Tetracapsuloides bryosalmonae* across all sampling periods indicates that PKD is well established at the study site. The identification of PKD on the border between the provinces of Turin and Cuneo extends the currently documented distribution of the disease within the Po River basin. Because no previous published surveys have investigated this specific area, the present findings should be interpreted as documenting the occurrence of PKD rather than demonstrating a recent geographic expansion. This shift is likely driven by increasing water temperatures, altered hydrological conditions, and enhanced environmental suitability for both the parasite and its bryozoan hosts, consistent with broader trends reported for PKD emergence in Europe [11].

As illustrated in Figure 3, PKD has been previously reported in several regions of northern Italy, including Lombardy, Veneto, parts of Friuli-Venezia Giulia, and limited areas of northeastern Piedmont.

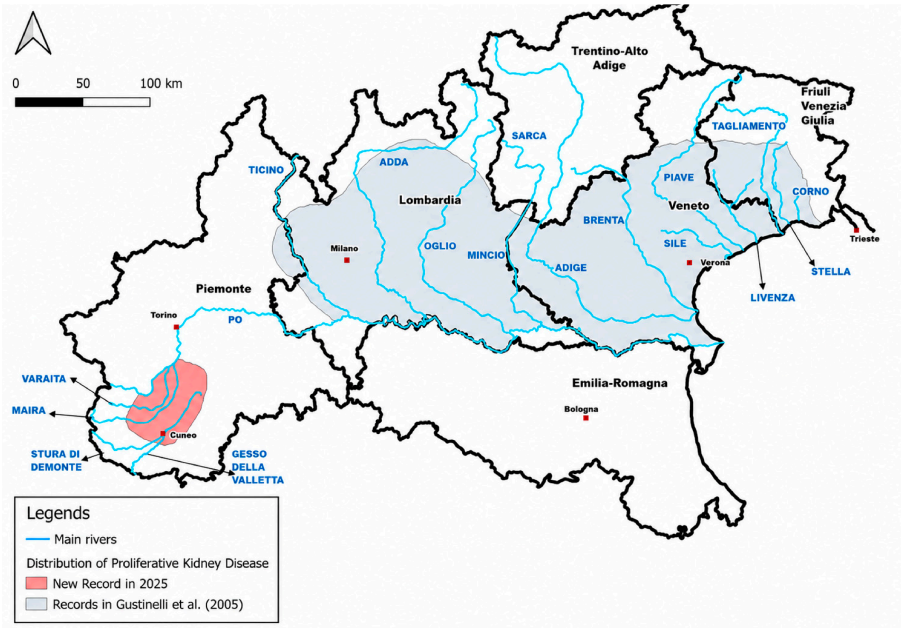

Figure 3. Geographical distribution of proliferative kidney disease (PKD) in Northern Italy, showing historical reports and the newly identified area of infection (present study).

The integration of pathological, bacteriological, and molecular findings provides evidence of a temporally structured pattern consistent with PKD, characterized by marked seasonal variability. The three sampling periods encompassed the period during which PKD typically develops under temperate environmental conditions, allowing us to infer seasonal changes in infection dynamics. However, because histopathological examination was not performed, the proposed temporal progression should be regarded as a biologically plausible interpretation rather than direct evidence of disease progression [32]. The observed temporal variation in PCR positivity is broadly consistent with the recognized influence of water temperature on PKD epidemiology [18]. However, the marked decline in PCR positivity during Period 3 despite relatively favorable water temperatures indicates that temperature alone cannot fully explain the observed seasonal dynamics. Additional factors, including host immune responses, parasite transmission dynamics, and the seasonal biology of bryozoan hosts, are also likely to contribute [32–34]. The relative importance of these factors could not be evaluated in the present study. Moreover, it is important to emphasize that the molecular approach adopted in this study was based on endpoint PCR, which provides qualitative detection of the parasite. Therefore, differences

in PCR positivity among periods should be interpreted as changes in the proportion of infected fish rather than variations in infection intensity.

An interesting pattern emerged when comparing PCR positivity with the distribution of gross lesions across the three sampling periods. During period 1, renal enlargement and splenomegaly were frequently observed despite relatively low PCR positivity, whereas period 2 showed the opposite trend, with the highest proportion of PCR-positive fish, but a markedly lower prevalence of gross lesions. This temporal mismatch indicates that gross pathological findings and molecular detection did not follow the same seasonal pattern. Although previous studies have shown that pathological alterations and parasite detection may exhibit different temporal dynamics during PKD progression [14,15], the mechanisms underlying this discrepancy could not be investigated in the present study because histopathological examination and quantitative assessment of parasite burden were not available. Therefore, the observed pattern should be interpreted as a field observation consistent with published knowledge of PKD pathogenesis rather than as direct evidence of distinct infection stages.

During period 2, PCR positivity reached its highest value (73.0%), whereas the prevalence of gross lesions remained comparatively low. This finding further supports the temporal mismatch between molecular detection and gross pathological findings observed in the present study. Previous investigations have shown that parasite detection and pathological alterations may follow different temporal dynamics during PKD progression [35,36]. Nevertheless, because histopathological examination and quantitative assessment of parasite burden were not performed, the biological significance of this discrepancy cannot be established. Therefore, these findings should be interpreted as field observations consistent with published descriptions of PKD rather than as evidence of a specific stage of infection.

In period 3, both PCR positivity and the prevalence of gross lesions declined substantially. This pattern coincided with decreasing water temperatures, which are known to reduce parasite proliferation and transmission [16,37]. Previous experimental studies have reported reductions in parasite burden and renal pathology following seasonal decreases in water temperature [38]. Although these findings are consistent with the temporal trend observed in the present study, the use of endpoint PCR and the absence of histopathological examination preclude direct comparison with those investigations. Therefore, the observed decline should be interpreted as an epidemiological observation rather than direct evidence of recovery or resolution of disease.

Although renal enlargement and splenomegaly are not pathognomonic for PKD, alternative major infectious causes were considered. No evidence of significant bacterial disease was identified, and the farm remained negative for infectious hematopoietic necrosis virus (IHNV) and viral haemorrhagic septicaemia virus (VHSV) through routine surveillance during the study period.

Overall, the lack of direct correspondence between lesion prevalence and PCR positivity suggests that gross pathology and molecular detection capture different aspects of the host–parasite interaction. The seasonal patterns observed in this study are consistent with previously described PKD dynamics. Future prospective studies integrating histopathology, quantitative PCR, and immunohistochemistry will be required to validate the biological interpretation of these seasonal patterns.

5. Limitations of the Study

One important limitation of the present study is the absence of histopathological examination. The fish were collected by the farmer during routine disease surveillance and immediately frozen before submission to our laboratory, precluding the collection of formalin-fixed tissues suitable for microscopic evaluation. Consequently, the characteristic

renal lesions associated with proliferative kidney disease, including interstitial nephritis, lymphoid hyperplasia, and granulomatous inflammation, could not be confirmed histologically, nor could lesions in secondary organs such as the spleen and liver be characterized.

A second limitation is the use of endpoint PCR, which provides qualitative detection of *T. bryosalmonae* but does not quantify parasite burden. Therefore, the present study cannot directly correlate parasite load with lesion severity or distinguish between different stages of infection based on parasite abundance.

Accordingly, the temporal framework proposed in this study should be interpreted as a biologically plausible hypothesis derived from the integration of seasonal epidemiological observations, gross pathological findings, and qualitative molecular detection, rather than as definitive evidence of disease progression. Future prospective investigations integrating histopathology, quantitative PCR, and immunohistochemical analyses on appropriately preserved tissues will be essential to validate the infection dynamics proposed here. Overall, the conclusions of the present study should primarily be interpreted from an epidemiological perspective rather than as a mechanistic description of PKD pathogenesis.

6. Conclusions

This study reports the first confirmed detection of PKD in southwestern Piedmont and extends the documented distribution of the disease in Italy. Whether this reflects a recent geographic expansion or the previous absence of surveillance in this area remains to be determined through future monitoring programmes. In the absence of vaccines or effective treatments currently available for PKD, early surveillance and prevention remain the only viable tools for mitigating disease outbreaks. Future work should implement long-term monitoring of both fish and bryozoan communities to clarify the mechanisms driving this expansion and to assess the potential for further spread. Integrating environmental data, hydrological modelling and habitat characterization will be essential to identify emerging hotspots of infection and predict future transmission dynamics. Additionally, evaluating the ecological impact on salmonid populations in newly affected areas is essential for informing conservation actions and supporting the resilience of salmonid species under changing environmental conditions.

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Institutional Review Board Statement: All animal handling procedures complied with European and Italian animal welfare regulations (Directive 2010/63/EU; Legislative Decree No. 26/2014). The study was approved by the IZSPLV Ethics Committee (protocol no. IZSTO/I/U/0005147, 16 May 2024).

Informed Consent Statement: Not applicable.

Data Availability Statement: The original contributions presented in this study are included in the article. Further inquiries can be directed to the corresponding author.

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Conflicts of Interest: The authors declare no conflicts of interest. Lucio Fariano is the owner of Azienda Agricola Canali Cavour. The remaining authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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