# Krill-derived nutrients as a risk management strategy

Under challenging farming conditions, they support hepatopancreatic function and recovery of shrimp growth performance from an EHP infection

**By Alberto J .P. Nunes, Daranee Seguin and Kiran Kaur**

The hepatopancreas is a central organ in shrimp physiology, integrating digestive, absorptive, metabolic and detoxification functions. Structurally, it is part of the midgut and is ventrally connected to the posterior stomach, where digestive secretions are released and mixed with ingested food.

This organ is composed of numerous blind-ending tubules lined with specialised epithelial cells and containing digestive enzymes and lipid-emulsifying compounds essential for nutrient utilisation. Each tubule comprises four main epithelial cell types with distinct but complementary roles (Figure 1).

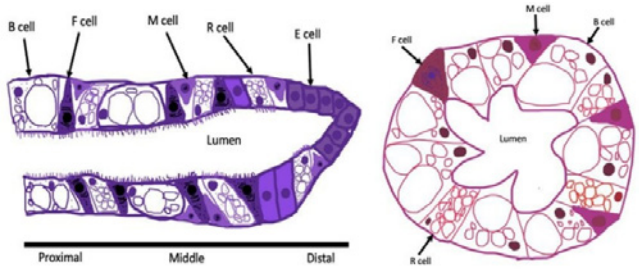


**Figure 1.** Illustration of hepatopancreatic tubules. Credit: Goh et al., 2023.

- E-cells (embryonic or regenerative cells) are responsible for cell proliferation, renewal and differentiation,
- F-cells (fibrillar cells) synthesise and secrete digestive enzymes, including proteases, lipases and amylases. They directly influence digestive efficiency,
- R-cells (resorptive cells) are primarily involved in nutrient absorption, intracellular digestion, and storage of lipids and glycogen. They play a key role in energy metabolism,
- B-cells are associated with intracellular digestion, waste processing and detoxification through holocrine secretion mechanisms.

The structural and functional integrity of these cells are critical for maintaining optimal growth, feed efficiency and resilience to stress.

Given this central role, hepatopancreatic health is highly sensitive to both nutritional quality and disease challenges. Histopathological assessment of this organ, such as evaluation of R-cell abundance and vacuolisation, B-cell hypertrophy or epithelial degeneration, is widely used in shrimp nutrition and health studies as a functional indicator of how dietary nutrients support hepatopancreatic health, physiological status and performance outcomes.

## A primary target under stress and pathogen challenges

Multiple pathogens target shrimp hepatopancreas, directly impairing its function and leading to reduced growth performance. Bacterial infections are particularly detrimental. *Vibrio parahaemolyticus*, the causative agent of acute hepatopancreatic necrosis disease (AHPND), produces toxins that destroy hepatopancreatic cells, leading to severe digestive dysfunction, poor growth and high mortality. Other *Vibrio* species (e.g., *Vibrio harveyi* and *Vibrio alginolyticus*) cause chronic infections that impair organ function and reduce feed intake.

Today, the most economically significant endemic pathogen is the microsporidian *Enterocytozoon hepatopenaei* (EHP) which specifically infects hepatopancreatic cells, significantly reducing digestion and absorption capacity and representing a major cause of slow growth in shrimp farming. Parasitic gregarines (e.g., *Nematopsis* spp., *Cephaloisporidium* spp.) further compromise nutrient absorption by colonising the digestive tract and hepatopancreas.

Together, these pathogens highlight the critical importance of hepatopancreatic health in optimising nutrient absorption, improving disease resistance and supporting overall performance. Targeted nutrition for hepatopancreatic function offers a proactive, farm-level strategy to strengthen shrimp resilience and productivity.

## From least-cost to precision nutrition in shrimp feed formulation

Modern shrimp farming operates under continuous stress conditions, including fluctuating water quality, high stocking density and pathogen pressure. Under these scenarios, traditional nutrition approaches based solely on meeting minimal nutrient requirements are insufficient. Instead, precision (or adaptive) nutrition is required, focusing on the strategic inclusion of functional nutrients that actively support metabolic capacity, tissue integrity and resilience.

Shrimp feed formulation has evolved from empirical fishmeal-based recipes through least-cost and digestible-nutrient approaches, towards precision nutrition, where diets are adapted to animal's developmental stage and genetics, production season and pathogen challenges. Within this framework, the hepatopancreas is the primary organ-level target. Nutrients that enhance membrane stability, digestive efficiency, lipid transport and cellular turnover directly determine whether animals can sustain productive function under the stresses that define modern intensive production.

## Functional nutrients from krill meal for hepatopancreatic support

Krill-derived ingredients carry a highly integrated package of functional nutrients that directly support hepatopancreatic structure, metabolism and resilience, particularly during nutritional and pathological stress. Unlike conventional ingredients that primarily provide protein and energy, krill meal and krill oil deliver a combination of bioactive compounds, including phospholipids, long-chain n-3 polyunsaturated fatty acids (EPA and DHA), astaxanthin, cholesterol, free amino acids, nucleotides, small peptides and chitin. These act synergistically to enhance digestive efficiency, cellular integrity and metabolic capacity.

Krill lipids have a predominance of phospholipid-bound fatty acids, distinguishing them from fish oil and other marine lipids. In most terrestrial and marine lipid sources, fatty acids are mainly present as triglycerides. This structural feature enhances the bioavailability and incorporation of essential lipids into cell membranes, particularly in hepatopancreatic tissues where lipid transport, storage and metabolism are critical.

Phospholipids play a central role in maintaining membrane fluidity and integrity, facilitating lipid digestion and absorption and supporting hepatopancreatic energy reserves. In addition, they improve the transport and utilisation of cholesterol and triglycerides, thereby contributing to more efficient metabolic regulation and improved resilience under stress conditions.

The lipid fraction of krill is also rich in EPA (20:5n-3) and DHA (22:6n-3), which are essential for maintaining cellular membrane structure and function. These fatty acids modulate membrane fluidity and permeability, directly influencing ion transport processes and reducing the energetic cost of osmoregulation.

This role is particularly relevant for the hepatopancreas, which is highly sensitive to environmental fluctuations such as salinity and temperature. Experimental evidence shows that diets enriched with krill-derived lipids improve growth performance, feed efficiency and stress tolerance, while also enhancing the deposition of EPA and DHA in shrimp tissues.

Cholesterol from krill meal further contributes by supporting membrane stability and serving as a precursor for moulting hormones. Importantly, the association of cholesterol with phospholipids enhances its absorption and transport efficiency, allowing krill meal to partially spare supplemental cholesterol in formulated diets without compromising performance. This is particularly relevant in low fishmeal formulations, where endogenous cholesterol supply may be limiting.

Astaxanthin, a key carotenoid present in krill meal, adds a strong antioxidant and immunomodulatory dimension to its functional profile. Astaxanthin contributes to the stabilisation of cellular membranes and protects hepatopancreatic cells from oxidative damage associated with stress and infection. Its antioxidant activity supports immune competence and reduces the impact of reactive oxygen species generated during environmental or pathological challenges. These combined effects support overall physiological robustness and immune competence during high-risk production periods.

Krill meal contains high levels of natural feeding stimulants, including free amino acids (e.g., arginine, glycine and proline), nucleotides and low-molecular-weight peptides. These compounds are potent chemo-attractants, enhancing feed detection, ingestion and palatability. Nucleotide concentrations in krill meal are particularly high, comprising predominantly monophosphates (e.g., AMP, IMP, GMP), known to stimulate feeding responses in crustaceans. Small peptides, largely below 1kDa, facilitate rapid diffusion in water and enhance sensory detection, contributing to improved feed intake and nutrient utilisation. These effects are especially important under disease or stress conditions, where feed intake is often compromised.

The functional benefits of chitin, present in low levels and finely distributed within krill meal, are related to immune stimulation and gut health. Unlike more recalcitrant chitin sources, krill-derived chitin is more bioavailable and exerts immunomodulatory effects, including the activation of innate immune responses and the enhancement of antimicrobial defenses. It may also contribute to the modulation of gut microbiota, indirectly supporting digestive efficiency and disease resistance.

Collectively, these functional nutrients contribute to maintaining integrity of the hepatopancreas by supporting key processes such as membrane stability, lipid transport, intracellular digestion and cellular turnover. This integrated mode of action is particularly relevant under challenging conditions, where hepatopancreatic dysfunction is often associated with reduced R-cell activity, impaired nutrient absorption and compromised energy metabolism. By enhancing both structural and functional aspects of the hepatopancreas, krill-derived nutrients help sustain metabolic efficiency and support recovery of growth performance even under disease pressure (Figure 2).

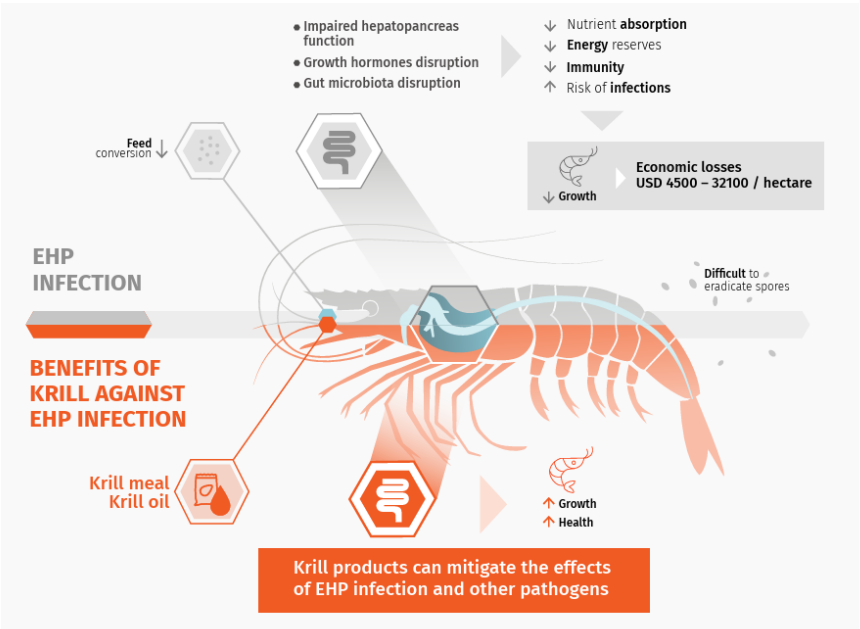


**Figure 2.** Functional nutrients from krill meal for shrimp hepatopancreatic support.

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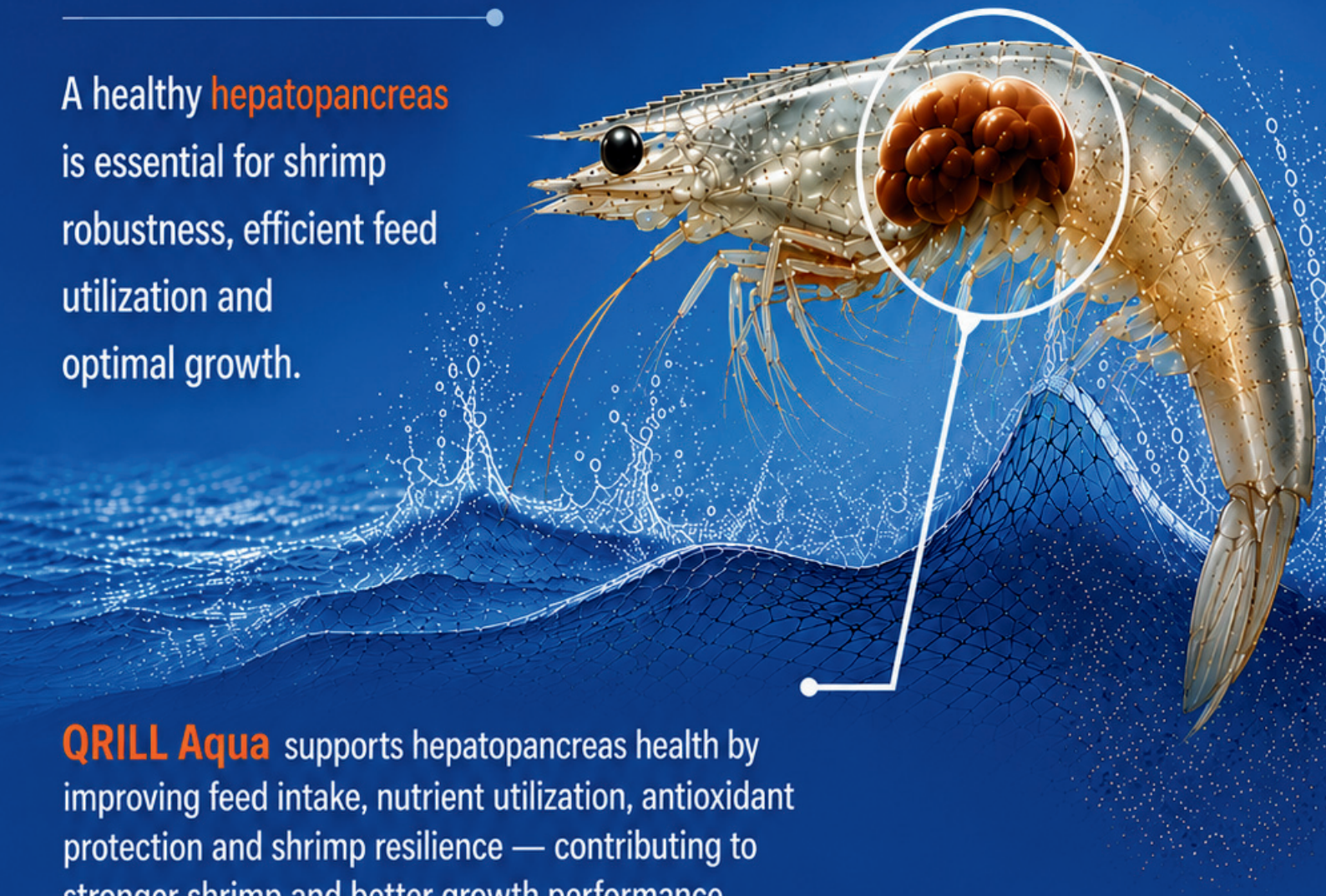

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A series of recent studies highlights the nutritional and functional benefits of incorporating krill-derived nutrients into shrimp diets to boost hepatopancreatic health under disease challenge.

## Supporting hepatopancreatic function when challenged by EHP

Two independent EHP challenge trials provide consistent quantitative evidence. In the first trial conducted at the Aquaculture Pathology Laboratory, University of Arizona, USA, 400 *Penaeus vannamei* were stocked in 20 independent 90L tanks. There were four replicates per treatment. Five diets were tested:

- a non-infected negative control (NEG),
- an infected positive control (POS),
- three treatment diets containing krill-derived ingredients- 3% krill meal (KM3), 10% krill meal (KM10), and 2% krill oil (KO2).

Shrimp were fed their respective diets for 35 days prior to challenge, followed by experimental infection with EHP via a single feeding of infected hepatopancreatic tissue. The trial lasted 73 days, including 38 days post-infection. Growth performance, survival and EHP load (via qPCR) were assessed, while hepatopancreatic health was evaluated through histopathological analysis, focusing on R-cell vacuolisation as an indicator of nutrient absorption and metabolic activity.

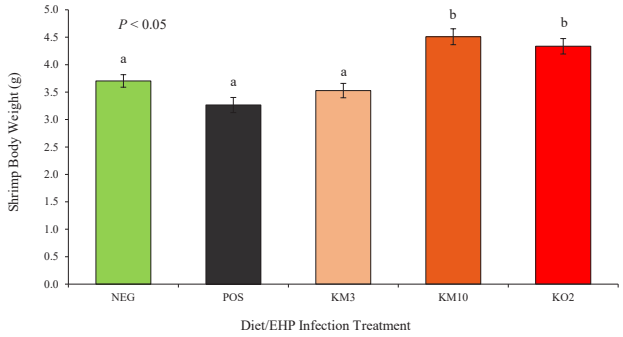


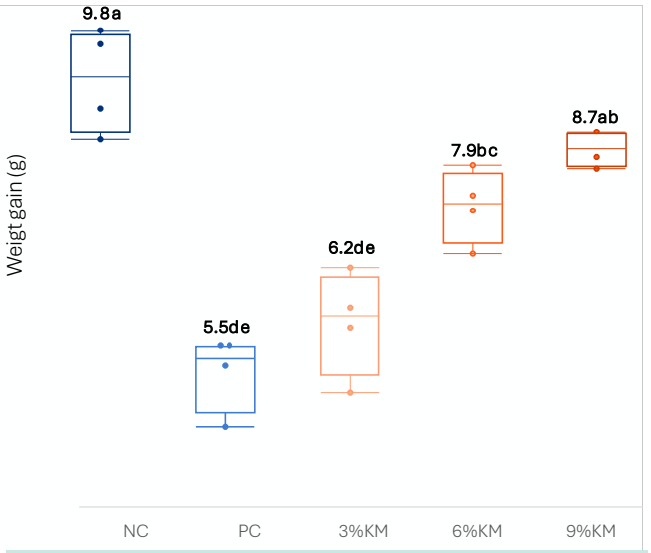

**Figure 3.** Shrimp mean body weight following EHP challenge (± standard error) at a trial conducted at the Aquaculture Pathology Laboratory, University of Arizona, USA. All shrimp were subjected to EHP challenge, except those in the negative control (NEG-CTL) treatment. Data values with same letters indicate no significant differences among dietary treatments according to Tukey's HSD test at p < 0.05. Burri et al., 2024.

This trial demonstrated that dietary inclusion of krill meal (KM) or krill oil (KO) can partially mitigate the negative effects of EHP infection on shrimp growth performance. Higher inclusion levels, 10% KM (4.6 ± 0.4g) and 2% KO (4.4 ± 0.4g), resulted in significantly greater final body weights compared to the infected control (POS: 3.3 ± 0.3g), and also outperformed the lower inclusion level (KM3: 3.5 ± 0.3g) and the non-infected control (NEG: 3.7 ± 0.2g), indicating a recovery of growth potential under disease pressure (Figure 2).

Histological analysis showed that EHP infection reduced hepatopancreatic R-cell vacuolisation, reflecting impaired nutrient absorption, digestion and energy storage, whereas krill-based diets counteracted this effect, suggesting improved cellular functionality. These findings highlight that krill-derived nutrients help maintain functionality of the hepatopancreas even under adverse conditions, supporting growth performance through improved metabolic efficiency.

## Validation under applied conditions

Further validation was obtained under controlled experimental conditions at the Directorate of Incubation and Vocational Training in Aquaculture, Tamil Nadu Dr J. Jayalalithaa Fisheries University (DIVA–TNJFU) in India. Apparently healthy and retarded growth juvenile shrimp (~4 g initial body weight) were obtained from commercial farms and were stocked in a tank system, with four replicates per treatment and 60 shrimp per tank. Four diets with graded krill meal inclusion levels (3%, 6% and 9%) were tested over a 56-day feeding period to assess growth performance, survival, hepatopancreas histology and EHP load. Treatments included a negative control (NC; non-infected shrimp) and a positive control (PC; EHP-infected shrimp fed a control diet without krill meal).




NC: negative control without EHP fed on control diet ;
PC: positive control with EHP fed on control diet;
3%KM: EHP infected shrimp fed with 3% krill meal
6%KM: EHP infected shrimp fed with 6% krill meal
9%KM: EHP infected shrimp fed with 9% krill meal
Source: Uma et al., 2025 in press

**Figure 4.** Body weight gain of *Penaeus vannamei*, mean± standard error) following EHP challenge at DIVA–TNJFU, India. All treatments, except the NC (negative control), were infected with EHP. Data values with same letters indicate no significant differences among dietary treatments according to Tukey's HSD test at $p < 0.001$ (Burri et al., 2024).

A clear dose-dependent pattern of improvement was observed across weight gain, specific growth rate (SGR) and survival. EHP infection significantly reduced growth and survival in positive control (PC), whereas dietary inclusion of krill meal progressively improved outcomes (Table 1). Survival increased from 62.5% in the PC group to 80.9% in the 9%KM group, comparable to the non-infected control (87.6%). Similarly, body weight gain improved from 5.46 ± 0.28g (PC) to 8.74 ± 0.13g in 9%KM, approaching the performance of non-infected shrimp (9.78 ± 0.39g). Growth metrics followed a similar trend, with specific growth rate (SGR) increasing from 9.75% in PC to 15.61% in 9%KM. Feed efficiency also improved markedly, as reflected by a reduction in FCR from 2.61 (PC) to 1.53 in the highest krill meal inclusion group.

Importantly, these performance improvements were supported by both histopathological evidence of preserved R-cell vacuolisation and EHP count in hepatopancreas. EHP infection resulted in complete

| Performance parameters | | | Treatment diets with krill meal (KM) | | |
|---|---|---|---|---|---|
| | Negative control (NC) | Positive control (PC) | 3% KM | 6% KM | 9% KM |
| Survival (%) | 87.6 ± 1.5$^{a}$ | 62.5 ± 2.5$^{c}$ | 67.5 ± 1.1$^{bc}$ | 72.5 ± 1.7$^{b}$ | 80.9 ± 1.7$^{a}$ |
| Body weight gain (g/shrimp) | 9.78 ± 0.39$^{a}$ | 5.46 ± 0.28$^{de}$ | 6.20 ± 0.39$^{de}$ | 7.90 ± 0.28$^{bc}$ | 8.74 ± 0.13$^{ab}$ |
| Growth (g/day) | 0.175 ± 0.007 | 0.098 ± 0.005 | 0.111 ± 0.007 | 0.141 ± 0.005 | 0.156 ± 0.009 |
| Specific growth rate (%) | 17.46 ± 0.70$^{a}$ | 9.75 ± 0.51$^{de}$ | 11.07 ± 0.70$^{de}$ | 14.11 ± 0.49$^{bc}$ | 15.61 ± 0.24$^{ab}$ |
| FCR | 1.29 | 2.61 | 2.28 | 1.78 | 1.53 |
| R cell(count/10Hp cells) | 215 | 0 | 55 | 95 | 139 |
| EHP copies | 0 | 20,011 ± 2,148 | 16,973 ± 1,725 | 12,293 ± 1,250 | 9,743 ± 1,123 |

**Table 1.** Growth performance and histopathological evidence of EHP infected shrimp (~4g, initial weight) fed different krill meal levels in diets in comparison to non-infected shrimp over 56 days. There were four replicates for each treatment and 60 shrimp per tank. Values are mean ± standard error (n = 4). Data with different letters is significantly different ($p < 0.001$). Source: Uma et al., 2025 (in press).

loss of hepatopancreatic R-cell vacuolisation in the positive control, indicating severe impairment in nutrient absorption and metabolic activity. In contrast, krill meal inclusion preserved R-cell integrity in a dose-dependent manner, with substantial recovery observed at higher inclusion levels (up to 139 R-cells per 10 HP cells in 9%KM).

Consistent with this observation, EHP load decreased progressively with increasing krill meal inclusion, from 20,011 ± 2,148 copies in the PC group to 9,743 ± 1,123 copies in 9%KM. This reduction suggests improved resilience to infection and/or enhanced capacity to limit pathogen proliferation.

## Conclusion

The findings demonstrate that krill-derived nutrients play a functional role beyond basic nutrition by directly supporting hepatopancreas integrity and performance under disease challenge. Across these independent studies, dietary inclusion of krill meal consistently improved growth performance, survival and feed efficiency while preserving hepatopancreatic structure, notably through the maintenance or recovery of R-cell abundance and vacuolisation, which are key indicators of nutrient absorption and metabolic activity.

In parallel, reductions in EHP load suggest that such nutrients may enhance host resilience not only by improving digestion and energy status but also by modulating physiological and possibly immune responses. These effects are likely driven by the unique composition of krill, which is rich in phospholipid-bound omega-3 fatty acids, choline, astaxanthin and other bioactive compounds that support membrane integrity, lipid metabolism and cellular function.

Trials demonstrated that krill-derived functional ingredients serve as a preventive, risk-management tool, with strongest benefits at 6–10% krill meal or 2% krill oil inclusion. These interventions are most valuable during high-risk phases, including EHP-endemic production cycles, periods of environmental instability and diet transitions when hepatopancreatic reserve capacity is under greatest pressure. By supporting hepatopancreatic functions during these critical windows, krill supplementation helps deliver more consistent production, reduced crop variability, improved economic returns and ultimately strengthen adoption confidence across the industry.

At a time when global resources are under strain, the responsibly managed krill fishery provides essential proteins and omega-3s, which are vital nutrients for animal and human health. The krill fishery is regulated by the Commission for the Conservation of Antarctic Marine Living Resources (CCAMLR). It is a fishery with the lowest by-catch. Scientific studies conclude that the by-catch in the Antarctic krill fishery, ranging from 0.1%-2.2%, is significantly lower than other fisheries. The latter has a range of 10% to 55%.

Aker QRILL Company is certified by Marine Stewardship Council (MSC) with 100% sustainable and traceable records. Certified by Friend of the Sea, the fishery as a whole has received an 'A' rating by Sustainable Fisheries Partnership.

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