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White Feces Disease In Liptopenaeus Vannamei Caused By Various Pathogen Agents: A Case Study Reported In Vietnam

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Abstract

White Feces Disease (WFD) presents a persistent challenge in the aquaculture of Litopenaeus vannamei and Penaeus monodon throughout Southeast Asia, manifesting as white fecal strings, diminished feeding, and stunted growth. This study examined outbreaks in Bac Lieu, Soc Trang, and Ben Tre provinces in Vietnam to elucidate the causative agents and their interactions. Bacterial cultures and molecular analyses consistently identified Vibrio parahaemolyticus, confirmed through 16S rRNA sequencing and the detection of virulence genes (toxR, tlh). All tested shrimp were also positive for the microsporidian Enterocytozoon hepatopenaei (EHP). Histopathological examinations revealed hepatopancreatic degeneration, sloughing of enterocytes, and the presence of intracytoplasmic EHP spores, while the white fecal casts consisted of shed epithelial cells heavily colonized by bacteria. Assessments of pond phytoplankton indicated a diverse algal community, with no harmful species detected, suggesting that algae are not direct causative agents of the disease. These findings indicate that WFD is a multifactorial syndrome primarily driven by EHP and V. parahaemolyticus, with pond conditions likely exacerbating the disease's manifestation. In contrast to acute hepatopancreatic necrosis disease (AHPND), the pathology associated with WFD arises from the synergistic interaction of microsporidian infection and opportunistic Vibrio overgrowth. These results underscore the necessity for integrated management strategies that combine biosecurity measures to control EHP, interventions to limit Vibrio proliferation, and ecological practices to sustain stable pond environments. Such measures are essential to reduce disease risk and promote sustainable shrimp aquaculture in Vietnam.

Keywords: *Liptopenaeus vannamei, White feces disease, V. parahaemolyticus*

1 INTRODUCTION

Vietnam ranks among the top three global shrimp exporters, significantly contributing to economic development and poverty reduction in rural and coastal communities (Nguyen et al., 2019; Nhuong, 2002; Ha et al., 2013). In 2024, shrimp farming spanned over 700,000 hectares, producing 1.29 million tons, with whiteleg shrimp (Litopenaeus vannamei) accounting for 951,000 tons and generating $3.95 billion in export revenue, a 14% increase from 2023 (Ministry of Agriculture and Rural Development, 2025). This underscores the critical role of shrimp aquaculture, particularly whiteleg shrimp, in Vietnam's socioeconomic progress, enhancing income, employment, and foreign exchange (Nhuong, 2002; Ha et al., 2013).However, the global shrimp industry faces numerous challenges, including climate change, market volatility, feed costs, broodstock quality, and diseases (FAO/Global Aquaculture Alliance, 2020; Villarreal, 2023). Major diseases affecting shrimp include White Spot Syndrome Virus (WSSV), Vibriosis, Acute Hepatopancreatic Necrosis Disease (AHPND), White Feces Syndrome (WFS), and Enterocytozoon hepatopenaei (EHP), leading to significant economic loss due to high mortality rates and yield reduction. White Feces Syndrome (WFS) in shrimp manifests through a constellation of clinical signs, including white to yellowish midguts, retarded growth, significant size variation, reduced average daily growth, elevated feed conversion ratios, loose exoskeletons, and sporadic mortality (Aranguren Caro et al., 2021; Prachumwat et al., 2021). Histopathological analyses reveal pronounced intestinal damage, characterized by diminished or absent microvilli, detachment of intestinal epithelial cells, and thinning of the intestinal wall (Huang et al., 2020). Typically emerging 50–60 days post-stocking, WFS induces chronic mortality, weakening shrimp and reducing production yields by up to 60% (Durai, 2015; Huang et al., 2020). These

effects destabilize farm revenues, undermine export competitiveness, and exert socioeconomic pressure on rural aquaculture-dependent communities.Etiological investigations indicate that WFS arises from a synergistic interaction between the microsporidian Enterocytozoon hepatopenaei (EHP) and opportunistic Vibrio species, notably Vibrio parahaemolyticus. These bacteria exploit EHP-induced epithelial damage to colonize and proliferate within the shrimp gut (Sriurairatana et al., 2014; Piamsomboon & Han, 2022). Histopathological examinations of the WFS-affected shrimp reveal severe degeneration of the hepatopancreas and midgut epithelium, including microvillar atrophy, enterocyte detachment, and the formation of aggregated microvillar bodies, which contribute to the characteristic white fecal casts (Sriurairatana et al., 2014).

Despite advancements in pathogen detection and molecular characterization, translating this knowledge into practical, field-validated management strategies remains a challenge, particularly in southern Vietnam, where shrimp farming is shaped by seasonal variability, fluctuating water quality, and diverse pond management practices. The interplay of bacterial virulence factors, microsporidian load, and environmental parameters—such as pond phytoplankton dynamics—remains inadequately elucidated. Existing research has predominantly focused on isolated etiological components, leaving a critical knowledge gap regarding their integrated dynamics in shrimp pond ecosystems. Addressing this gap is essential for developing targeted interventions that align with the ecological and socioeconomic contexts of the region.

To address these knowledge gaps, this study conducts comprehensive field investigations across three major shrimp-farming provinces in southern Vietnam—Bac Lieu, Soc Trang, and Ben Tre. By integrating etiological insights with field-tested management practices, including balanced nutrient regimes, periodic pond sanitation, and ecological restoration, this research provides a robust framework for mitigating WFS impacts. These strategies aim to reduce economic losses, enhance farm productivity, and promote the sustainable development of Vietnam's shrimp aquaculture industry. Beyond addressing immediate disease challenges, the proposed interventions contribute to broader objectives of environmental stewardship, economic resilience, and sustained global market competitiveness for Vietnamese shrimp.

2 METHODS AND MATERIALS

2.1Sample Collection and Preservation

Shrimp displaying the WFS-clinical signs, including yellow to white midguts, segmented or floating white feces, were collected from commercial aquaculture ponds in Bac Lieu, Soc Trang, and Ben Tre provinces, Vietnam. Ten shrimps per pond were aseptically sampled, immediately placed on ice, and transported to the laboratory within 4 hours. Water samples (1 L) were simultaneously collected from the corner and center of each pond, stored on ice, and processed within 4 hours. For molecular analyses, shrimp were fixed in 90% ethanol. For histopathological examination, shrimp were preserved in Davidson's fixative for 24–72 hours and subsequently transferred to 70% ethanol for storage.

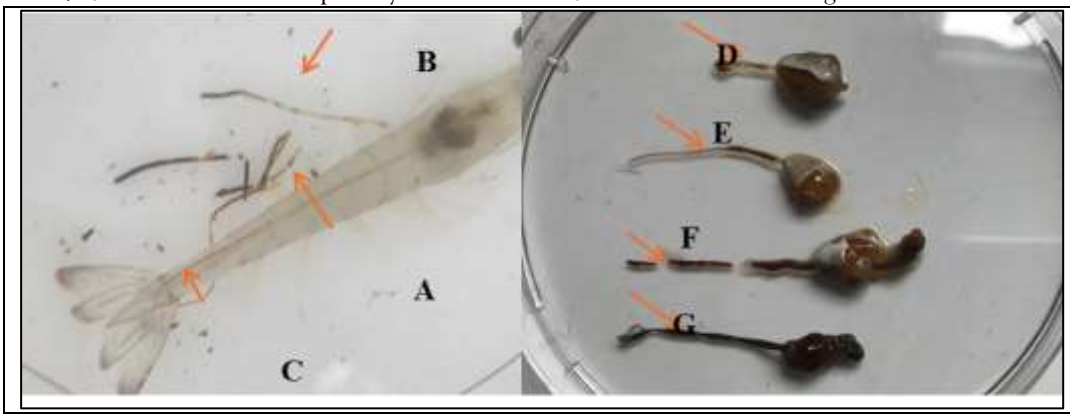

Figure 1. Clinical Presentation of naturally diseased shrimp
(A) Digestive tract exhibiting a pale yellow coloration; (B) and (C) sticky feces displaying pale yellow to white hues.

Figure 2. Comparative analysis of digestive Tracts in Shrimp Affected by WFD
(D) and (E) depict a loose intestinal structure; (D), (E), and (F) illustrate pale yellow intestinal coloration; (F) shows a segmented intestinal morphology. For comparison, (G) represents the digestive tract of a healthy shrimp.

2.2Bacterial Isolation and Identification

Hepatopancreas and intestinal tissues were aseptically excised from ethanol-fixed shrimp. Tissues were streaked onto CHROMagar™ Vibrio plates (CHROMagar, France) and incubated at 30°C for 18-24 hours. Presumptive Vibrio spp. Colonies were subcultured onto thiosulfate-citrate-bile salts-sucrose (TCBS) agar (HiMedia, India) to assess sucrose fermentation and pigment production. Representative colonies were underwent to biochemical profiling using the API 20E system (bioMérieux, France) following the manufacturer's protocol.

2.3DNA Extraction and PCR Detection of Virulence Genes

Genomic DNA was extracted from bacterial isolates using a boiling lysis method (95°C for 10 minutes). Polymerase chain reaction (PCR) assays targeted the toxR (368 bp), tlh (450 bp), pirA, and pirB genes, following established protocols (Han et al., 2015; Sirikharin et al., 2015). The pirA and pirB genes, homologues of Photorhabdus Pir toxins, serve as validated markers for identifying Acute Hepatopancreatic Necrosis Disease (AHPND)-causing Vibrio parahaemolyticus strains (Han et al., 2015; Sirikharin et al., 2015).

Table 1. Primer and specific sequence used

Gene	Primer	Primer Sequence (5' – 3')	Product Size (bp)	Reference
toxR	tR	F: GTCTTCTGACGCAATCGTTG R: ATACGAGTGGTTGCTGTCATG	368	(Kim Yung, Okuda et al. 1999)
Tlh	tl	F:AAAGCGGATTATGCAGAAGCACTG R: GCTACTTTCTAGCATTTTCTCTGC	450	(Hayat Mahmud, Kassu et al. 2006)
$pirA^{vp}$	VpPirA-284	VpPirA-284F: TGACTATTCTCACGATTGGACTG VpPirA-284R: CACGACTAGCGCCATTGTTA	284	(Han, Tang et al. 2015)
$pirB^{vp}$	VpPirB-392	VpPirB-392F: TGATGAAGTGATGGGTGCTC VpPirB-392R: TGTAAGCGCCGTTTAACTCA	392	(Han, Tang et al. 2015)
16S rRNA	27F/1492R	27F: AGAGTTTGATCMTGGCTCAG 1492R: TACGGYTACCTTGTTACGACTT	1400	(Miller, Handley et al. 2013)

2.416S rRNA Sequencing

Two representative isolates (V11 and V13) were selected for 16S rRNA gene amplification using universal primers 27F and 1492R. Amplified PCR products were purified and sequenced. Sequences were analyzed against the NCBI GenBank database using the Basic Local Alignment Search Tool (BLAST) to confirm species identity.

2.5Detection of Enterocytozoon hepatopenaei (EHP)

DNA was extracted from hepatopancreatic tissues using the Qiagen DNeasy Blood & Tissue Kit. Nested PCR targeting the small subunit rRNA gene of Enterocytozoon hepatopenaei (EHP) was performed as described by Tangprasittipap et al. (2013). Positive controls (EHP-infected shrimp) and negative controls were included in all PCR reactions to ensure assay reliability.

2.6Histopathology

Hepatopancreas, midgut, and fecal strands were fixed in Davidson's fixative for 24–48 hours, dehydrated through a graded ethanol series, embedded in paraffin, and sectioned at 5 μm thickness. Tissue sections were stained with hematoxylin and eosin (H&E) and examined under light microscopy. Histopathological analysis focused on the detection of EHP spores, epithelial integrity, and identifying gut lesions or sloughing.

2.7Phytoplankton Analysis

Water samples (500 mL), collected from each pond and preserved with Lugol's iodine solution, were allowed to settle for 48 hours in a dark chamber. After decanting the supernatant, the concentrated sediment was analyzed using an inverted microscope. Phytoplankton were identified to the genus level using standard taxonomic keys and quantified using a Sedgwick-Rafter counting chamber. Cell density was reported as cells per liter (cells/L).

3 RESULTS AND DISCUSSION

3.1Bacterial Isolation and Identification

Hepatopancreas and intestinal samples from shrimp exhibiting the WFS-clinical signs consistently yielded Vibrio parahaemolyticus when cultured on CHROMagar™ Vibrio. Two dominant colony morphotypes were observed: blue-green colonies, comprising 40–90% of isolates, and pale violet colonies, accounting for 40–60% (Figure 3). On thiosulfate-citrate-bile salts-sucrose (TCBS) agar, these isolates produced characteristic green colonies indicative of V. parahaemolyticus (Figure 4). Other bacterial species were detected infrequently, constituting less than 5% of total colonies. No gregarine parasites were observed in any of the examined samples. Molecular analysis via polymerase chain reaction (PCR) confirmed the presence of Enterocytozoon hepatopenaei (EHP) in all five sampling batches (Table 2).

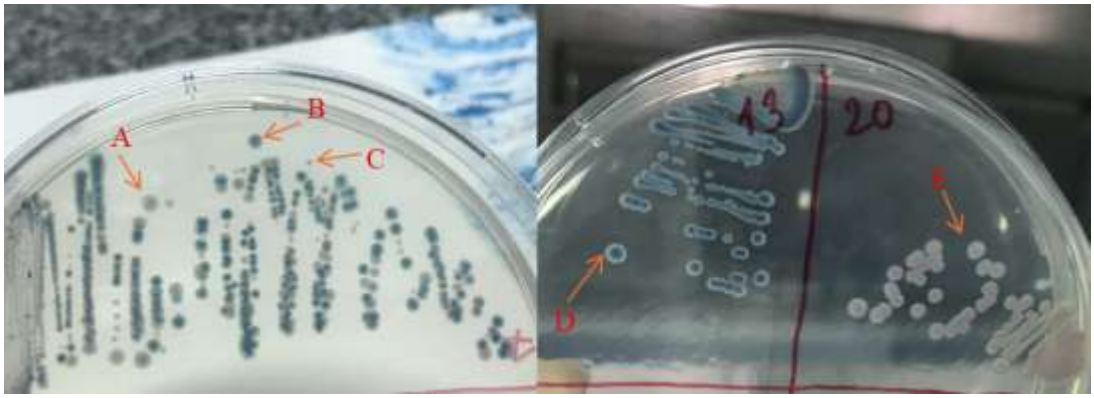

Figure 1. Isolation of bacteria from shrimp intestines affected by WFD on CHROMagar. The majority of colonies are blue-green (A) and pale purple (B), while other bacteria form smaller, scattered colonies (C). Vibrio

Table 2. Results of Pathogen Analysis in Shrimp Samples Affected by White Feces Disease (WFD)

	Batch 1		Batch 2		Batch 3		Batch 4		Batch 5	
	Frequency(%)	Rate (%)	Frequency(%)	Rate (%)	Frequency(%)	Rate (%)	Frequency(%)	Rate (%)	Frequency(%)	Rate (%)
V. parahaemolyticus blue-green	100	40-90	100	50-90	100	70-90	100	<50	100	30-40
V. parahaemolyticus Pale purple	100	5-20	100	5-40	100	20-40	100	90	100	50-70
Other bacterias	20	<5	10	<5	10	<5	20	<5	20	<5
Gregarine	Negative		Negative		Negative		Negative		Negative	
EHP	+		+		+		+		+	

Biochemical profiling of 21 representative isolates using the API 20E system identified 11 strains as V. parahaemolyticus, three as V. alginolyticus, two as Pseudomonas aeruginosa, and one as Shewanella putrefaciens; four isolates remained unidentified (likely weak-growing Enterobacteriaceae) (Table 3).

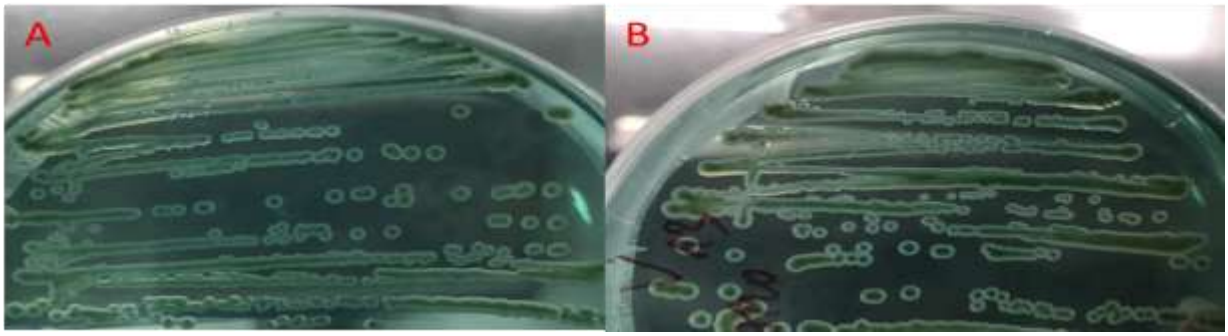

Figure 2. V. parahaemolyticus V11 on TCBS (A), V. parahaemolyticus V13 on TCBS (B)

The consistent isolation of V. parahaemolyticus from all shrimp exhibiting WFS highlights its pivotal role in the pathogenesis of WFS outbreaks. The predominance of V. parahaemolyticus over other Vibrio species and opportunistic bacteria corroborates earlier studies associating this pathogen with white feces syndromes in Southeast Asian aquaculture (Somboon et al., 2012; Cao et al., 2015). The universal detection of Enterocytozoon hepatopenaei (EHP) across all sampled batches suggests a co-infection dynamic, wherein EHP-induced damage to the gut epithelium likely facilitates the proliferation of V. parahaemolyticus, exacerbating disease severity.

Table 3. Results of Bacterial Identification by API 20E Biochemical Test

	Identification
1	V. parahaemolyticus
2	V. alginolyticus
3	Shewanella putrefaciens
4	Pseudomonas aeruginosa
5	V. alginolyticus
6	n/a
7	V. alginolyticus
8	n/a
9	n/a
10	V. parahaemolyticus
11	V. parahaemolyticus
12	V. parahaemolyticus
13	V. parahaemolyticus
14	V. parahaemolyticus
15	V. parahaemolyticus
16	V. parahaemolyticus
17	V. parahaemolyticus
18	V. parahaemolyticus
19	Pseudomonas aeruginosa
20	V. parahaemolyticus
21	n/a

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3.2Molecular Confirmation and Virulence Gene Profiling

Partial 16S rRNA gene sequences (approximately 1,400 bp) from two bacterial isolates (V11 and V13) exhibited ≥99.8% sequence identity with Vibrio parahaemolyticus reference sequences in the NCBI GenBank database (Table 4). Polymerase chain reaction (PCR) assays confirmed the presence of species-specific genes toxR (368 bp) and tlh (450 bp) in both isolates. However, the pirA and pirB genes, which are associated with Acute Hepatopancreatic Necrosis Disease (AHPND), were not detected (Figure 5).

Table 4. Gene Sequencing Results of Isolates V11 and V13

Isolates	V11	V13
Gene fragment length (bp)	1,405	1,396
Sequence identity (%)	99.93	99.86
Gaps	0	0
GenBank Accession No.	MG762012.1	CP046783.1
Reference sequence	S24P132 16S	2013V-1181 chromosome 1
Conclusion	Vibrio parahaemolyticus	Vibrio parahaemolyticus

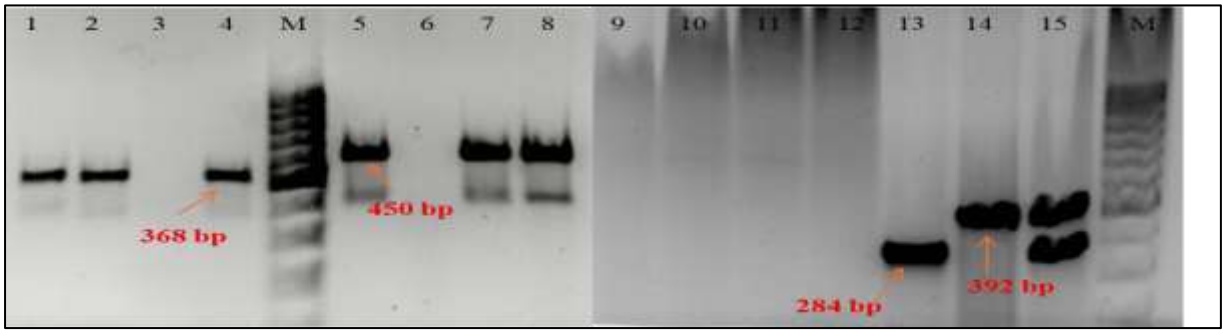

Figure 3. PCR amplification results for toxR, tlh, and pirAB genes in Vibrio parahaemolyticus isolates V11 and V13.

The presence of toxR, a global regulator of hemolysin expression, and tlh, encoding thermostable hemolysin, confirms the pathogenic potential of these strains (Kim Yung, Okuda et al. 1999, Hayat Mahmud, Kassu et al. 2006). The absence of pirAB indicates that WFD in these ponds is mechanistically distinct from AHPND, supporting the hypothesis that different virulence factors drive WFD pathology.

3.3Histopathological Observations

Histological examination of shrimp feces reveals stark contrasts between normal and White Feces Syndrome (WFS)-affected samples (Figure 6, 7a-b). Normal feces (Figure 6A, 6C) appear dark, primarily comprising food debris, as observed at 100× magnification, indicative of a healthy digestive process with intact food particles and minimal cellular damage. Conversely, white fecal casts (Figure 6B, 7A–B) retain the midgut's epithelial architecture, with luminal casts composed of sloughed enterocytes. At 40× magnification, these casts exhibit an organized outer epithelial layer, an interior containing food particles and bacteria, and a distinct luminal cavity, suggesting that WFS pathology involves shedding of gut lining cells rather than solely undigested feed (Sriurairatana et al., 2014).

Figure 4. Normal feces (A) versus white feces (B). Histological section of dark feces containing food debris fragments (arrows), viewed at 100× magnification (C)

High-magnification (100×) sections of white fecal casts (Figure 7C) reveal dense colonization by rod-shaped and vibrioid bacteria, likely Vibrio parahaemolyticus, on the mucosal surface, contributing to tissue damage and the casts' white appearance. Numerous intracytoplasmic Enterocytozoon hepatopenaei (EHP) spores, confirmed by PCR, were observed in hepatopancreatic and gut epithelial cells (Figure 7D, 8) at 40× and 100× magnifications, indicating systemic microsporidian infection. The extensive sloughing of microvilli and enterocyte detachment align with characteristic WFS lesions, where EHP-induced epithelial compromise facilitates V. parahaemolyticus proliferation, exacerbating gut pathology and forming white fecal casts (Sriurairatana et al., 2014).

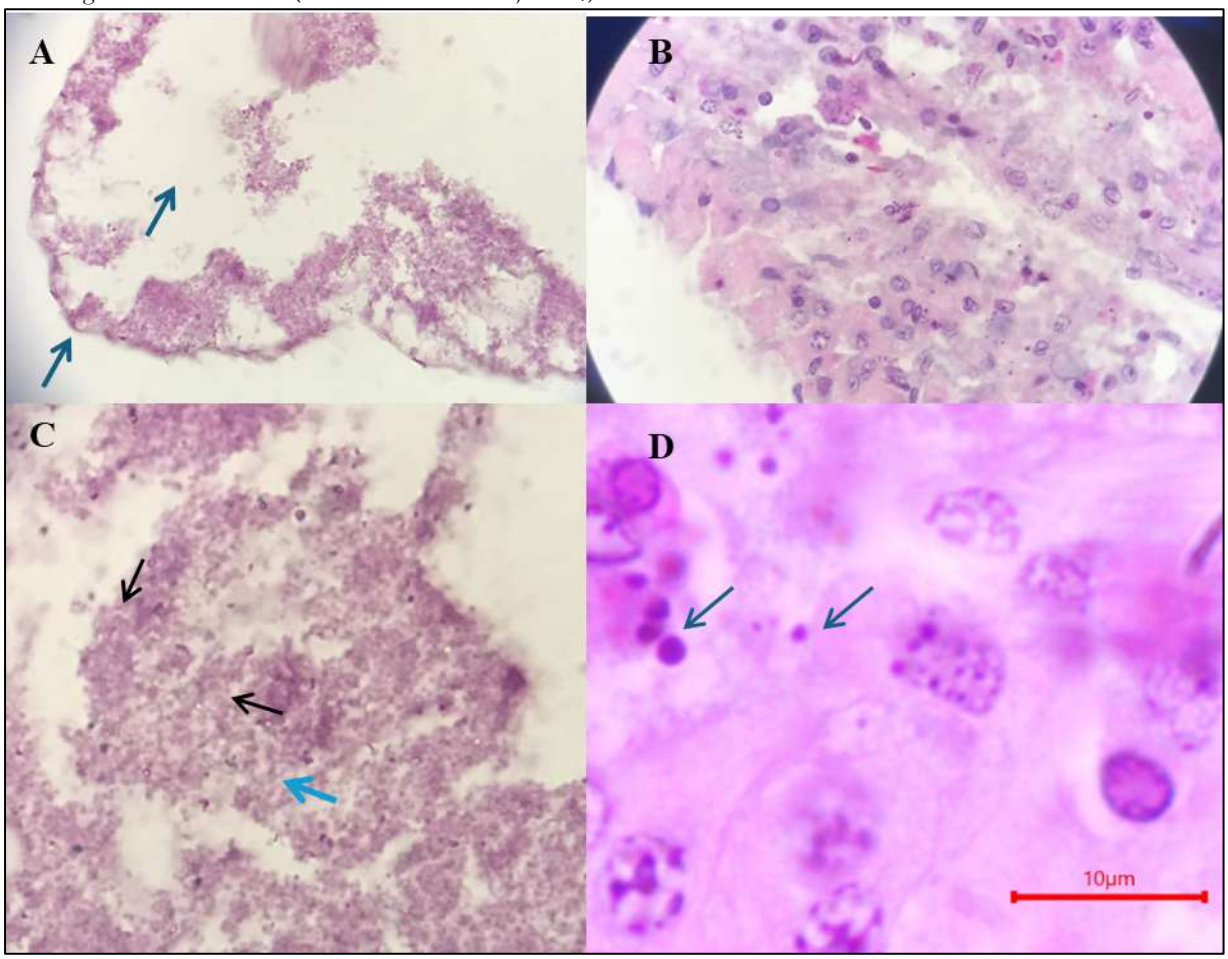

Figure 5. Histological section of white feces. (A) Outer epithelial structure visible, with interior containing food particles and bacteria, at 40× magnification. (B) White feces section showing cellular organization and luminal cavity, 40× magnification. (C) Presence of vibroid bacteria (black arrow) and rod-shaped bacteria (green arrow), 100× magnification. (D) Numerous oval granule-like structures in the gut (arrows), 100× magnification.

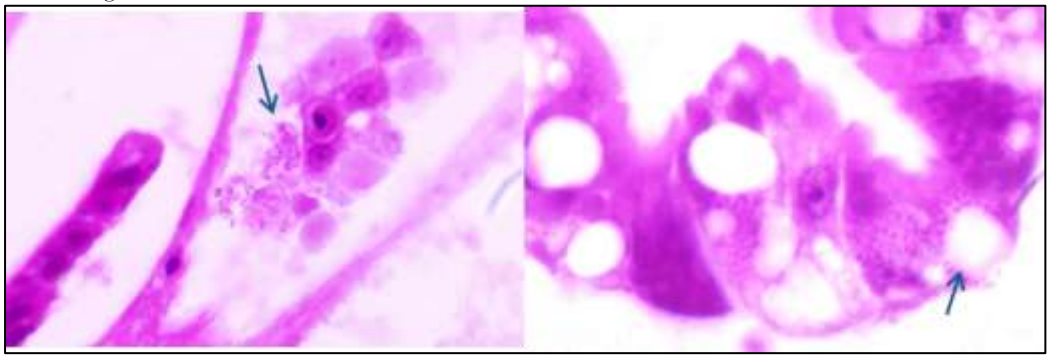

Figure 6. Histological sections of hepatopancreas from shrimp afflicted with WFD, showing numerous oval granule-like structures (arrows) within hepatopancreatic tissue, viewed at both 40× and 100× magnifications

The massive sloughing of microvilli and detachment of enterocytes observed here mirror classic WFD-associated lesions (Sriurairatana et al. 2014). Microsporidian infection likely compromises epithelial integrity, creating niches for V. parahaemolyticus adherence and proliferation, thereby exacerbating tissue damage and leading to characteristic white fecal casts.

3.4Phytoplankton Community Structure

Table 5 details a diverse phytoplankton community comprising 16 taxa in shrimp pond water samples from southern Vietnam, with cell densities ranges indicating site-specific variability. Chlorella spp. (chlorophytes) exhibited the highest maximum density at 482,000 cells L^{-1}, followed by Phormidium spp. (cyanobacteria) at 102,000 cells L^{-1}, and Pleurosigma spp. (diatoms) at 47,400 cells L^{-1}. Euglena spp. (euglenophytes) recorded the lowest minimum density at 10 cells L^{-1}. Total plankton density ranged from 17,750 to 674,180 cells L^{-1}. No harmful algal species were detected, aligning with the etiological focus on Enterocytozoon hepatopenaei (EHP) and Vibrio parahaemolyticus as primary drivers of White Feces Syndrome (WFS).

Table 5. Phytoplankton Composition in Pond Water Samples

No.	Scientific names	Population (Cells/L)				
		BL1	BL2	ST1	ST2	BT
	DIATOMS (BACILLARIOPHYCEAE)					
1	Cyclotella sp.	10,200	17,500	10	1,920	0
2	Nitzschia sp1.	1,810	510	0	0	30
3	Pleurosigma sp.	47,400	22,400	80	260	1,170
	GREEN ALGAE (CHLOROPHYCEAE)					
4	Chlorella sp.	482,000	58,000	3,500	11,210	27,100
5	Scenedesmus bijugatus Kützing, 1834	40	210	105	2,280	0
	BLUE-GREEN ALGAE (CYANOPHYCEAE)					
6	Merismopedia sp.	320	1100	0	0	0
7	Phormidium sp.	96,240	102,000	31,050	1,200	75,650
8	Aphanocapsa sp.	0	0	0	0	1,870
9	Limnothrix sp.	0	0	0	0	40
10	Oscillatoria sp.	0	0	1,480	520	0
11	Pseudanabaena sp.	8,550	1,150	0	0	10
	EUGLENOIDS (EUGLENOPHYCEAE)					
12	Euglena sp.	27,600	32,540	180	50	10
13	Euglena sp2.	10	240	520	180	0
14	Trachelomonas volvocina. Ehrenberg 1833	10	50	0	0	10
	DINOFLAGELLATES					
15	Diplosalis sp.	0	0	0	10	5,010
16	Protoperidinium sp.	0	0	0	120	2,130
	Total	674,180	235,700	36,925	17,750	113,030

The predominance of chlorophytes and cyanobacteria suggests eutrophic conditions, likely driven by nutrient inputs from shrimp feed. Wide density ranges (e.g., Chlorella spp. from 3,500 to 482,000 cells L^{-1}) indicate heterogeneity in pond environments, potentially influenced by water exchange rates or seasonal factors. High phytoplankton biomass may contribute to organic loading and hypoxia, indirectly promoting Vibrio proliferation. In contrast, the absence of toxic algae reduces the risk of acute blooms but does not negate the need for ecological management to maintain pond stability (Lemonnier et al., 2016; Sun et al., 2018).

4 CONCLUSION

The analyses confirm that White Feces Syndrome (WFS) in shrimp is not attributable to a single pathogen but arises from a complex pathobiome involving Enterocytozoon hepatopenaei (EHP) and virulent Vibrio parahaemolyticus, within eutrophic environmental conditions. From the hepatopancreas and intestine of diseased shrimp, 21 bacterial strains were isolated, including 11 V. parahaemolyticus, 3 V. alginolyticus,

2 Pseudomonas aeruginosa, and 1 Shewanella putrefaciens. Out of those strains, V. parahaemolyticus was dominant, present in 100% of samples and forming two distinct colony types (blue-green and pale purple) with over 90% prevalence.

Histopathological examinations revealed abundant oval Enterocytozoon hepatopenaei (EHP) spores within the hepatopancreas and intestinal tissues of affected shrimp. White Feces Syndrome (WFS) fecal casts retained epithelial and luminal structures, exhibiting dense colonization by vibrioid and rod-shaped bacteria. Phytoplankton assessments identified high densities of Chlorella spp. (3,500–482,000 cells L^{-1}), Phormidium spp. (1,200–102,000 cells L^{-1}), Pleurosigma spp. (80–47,400 cells L^{-1}), and Euglena spp. (10–32,540 cells L^{-1}), indicative of eutrophic pond conditions but lacking acute toxins. Molecular analyses confirmed that Vibrio parahaemolyticus isolates V11 and V13 harbored the virulence genes toxR and tlh but lacked pirA and pirB, distinguishing WFS from Acute Hepatopancreatic Necrosis Disease (AHPND). These isolates were successfully used in co-infection trials, supporting a synergistic disease mechanism.

The findings indicate that EHP-induced epithelial damage compromises gut integrity, facilitating pathogenic V. parahaemolyticus colonization, which exacerbates gut pathology and leads to the formation of characteristic white fecal casts in nutrient-rich pond environments. These observations align with experimental studies demonstrating EHP/Vibrio synergism (Aranguren Caro et al., 2021; Piamsomboon & Han, 2022), reinforcing a multifactorial etiology distinct from AHPND. Given the absence of effective treatments (Piamsomboon & Han, 2022), integrated management strategies are critical. These include implementing biosecurity measures and monitoring to control EHP prevalence, employing probiotics or phage therapy to limit Vibrio proliferation, and maintaining ecological balance in ponds to mitigate microbial dysbiosis.

To mitigate risks associated with phytoplankton dynamics:

- Implement routine monitoring using microscopy to track density shifts, with alerts for total densities exceeding 500,000 cells L^{-1}.
- Optimize feed regimes and employ biofloc systems to control nutrient loads and reduce eutrophication.
- Introduce probiotics to balance microbial communities and enhance aeration to prevent oxygen depletion.
- Implement biosecurity to screen for EHP, use probiotics or phage therapy to control Vibrio overgrowth, and optimize pond water quality (e.g., dissolved oxygen, pH) to reduce shrimp stress and infection susceptibility

Author contributions:

Van Nhan Le, Hai Anh Tran, conceived and designed the experiments; Thi Quynh Bui, Ha Minh Duc Tran performed the experiments; Van Diep Le analyzed the data; Thi Thanh Mai Nguyen contributed reagents/materials/analysis tools; Van Nhan Le, Ha Tran Minh Duc wrote the paper.

Conflicts of interest: The authors declare no conflicts of interest.

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