These findings indicate that seasonal effects operate through combined influences on host physiology and environmental conditions.

Shrimp diseases also show clear regional variability. The dominant pathogen spectrum and epidemiological patterns differ among regions. Asia has experienced multiple disease outbreaks, including WSSV, YHV, AHPND, and EHP, making it one of the most complex and high-risk regions globally. In contrast, the Americas were initially dominated by TSV and WSSV, with increasing reports of AHPND in recent years. Even within a single country, significant differences may exist among farming areas. For example, studies in the east coast of India have shown varying combinations of EHP, Vibrio spp., and WSSV across different farming systems and regions. Coastal high-density farming areas generally face higher outbreak risks due to frequent seedstock movement and pathogen introduction, whereas inland low-salinity systems may experience lower disease frequency but still suffer significant losses once outbreaks occur due to limited monitoring capacity.

Furthermore, co-infection has become a prominent feature of shrimp disease epidemiology. Field observations and experimental studies indicate that multiple pathogens can coexist within the same system or host, such as EHP–WSSV and Vibrio–WSSV co-infections (Chowdhury et al., 2024). These co-infections often exhibit synergistic pathogenic effects rather than simple additive impacts. Viral infections can weaken host immune defenses and damage tissue barriers, facilitating bacterial invasion. Conversely, bacterial infections can promote viral replication through tissue damage and inflammatory responses. As a result, multi-pathogen interactions often lead to higher mortality rates, more complex clinical manifestations, and increased difficulty in diagnosis and disease control.

3 Mechanisms and Influencing Factors of Shrimp Diseases

3.1 Pathogen-host interactions and immune mechanisms

The occurrence of shrimp diseases is essentially the result of an imbalance among pathogens, the host innate immune system, and environmental factors. As invertebrates, shrimp lack a typical adaptive immune system and long-term specific immune memory; thus, their defense against infections mainly relies on innate immunity. This system consists of both cellular and humoral components, including phagocytosis, encapsulation, coagulation, and activation of the prophenoloxidase system. In addition, effector molecules such as antimicrobial peptides, lectins, and lysozymes play critical roles in immune defense. Upon pathogen invasion, the host initially recognizes pathogen-associated molecular patterns (PAMPs) via pattern recognition receptors (PRRs). This recognition subsequently activates key signaling pathways, such as NF-κB and JAK/STAT, leading to the expression of various immune effectors that contribute to pathogen clearance and restriction of their spread in hemolymph and tissues.

However, the intensity and duration of innate immune responses in shrimp are relatively limited, making it difficult to establish long-term protection similar to that observed in vertebrates. Consequently, shrimp are more susceptible to infections and disease outbreaks under conditions of high pathogen pressure or environmental stress. Transcriptomic and immunological studies have shown that multiple immune pathways are significantly modulated following infection with white spot syndrome virus (WSSV) or AHPND-causing Vibrio. Meanwhile, many pathogens have evolved sophisticated immune evasion and host manipulation strategies. For example, WSSV can interfere with host signal transduction, apoptosis, and antiviral responses, thereby creating a cellular environment favorable for viral replication and dissemination (Xiong et al., 2024). Therefore, pathogen-host interactions represent a dynamic and continuous interplay rather than a simple unidirectional process of host defense versus pathogen invasion.

In bacterial diseases, the pathogenic mechanisms of AHPND also demonstrate strong pathogen adaptability. The causative Vibrio parahaemolyticus strains typically harbor the pVA1 virulence plasmid encoding PirAB toxins, which directly damage hepatopancreatic tissues, leading to epithelial cell sloughing and necrosis. This enables rapid disruption of local immune defenses and results in high mortality (Chandran et al., 2023). Furthermore, an imbalance in host immune responses can exacerbate disease progression. Excessive inflammatory responses may