

Taura Syndrome Virus in shrimp aquaculture: A comprehensive review of biology, epidemiology, and control strategies

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ARTICLE INFO

Received: 01 July 2026

Accepted: 31 July 2026

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Keywords:

Aquaculture, Biosecurity, *Penaeus vannamei*, Shrimp, Taura Syndrome Virus.

ABSTRACT

A prominent virus in the shrimp aquaculture sector, Taura Syndrome Virus (TSV) mainly affects *Penaeus vannamei* and causes large financial losses in a number of international production regions. The positive-sense single-stranded RNA (+ssRNA) genome of this virus, which is a member of the Dicistroviridae family, allows for effective replication and quick horizontal dissemination. A wide range of symptoms, including altered tail color, reduced eating activity, growth retardation, and abrupt mortality during the outbreak phase, are indicative of TSV infection. Without the assistance of laboratory techniques, non-specific illness symptoms can make early identification at the field level more difficult. Environmental circumstances, culture density, seed quality, and inadequate biosecurity procedures are some of the factors that affect TSV transmission. This virus causes gradual cellular destruction through necrosis and systemic physiological failure, with a pathobiological preference for ectodermal and mesodermal tissues. Despite not being zoonotic, TSV is a crucial problem for global food security and fisheries economics due to its effects on decreased aquaculture output and supply chain stability. Since specific treatments are still unavailable, current TSV control still depends on preventive measures such as the adoption of pathogen-free seed, environmental management, and stringent biosecurity procedures. Therefore, maintaining the viability of contemporary aquaculture systems requires a thorough understanding of the biological traits, epidemiology, and management techniques of TSV.

Introduction

The productivity and sustainability of culture systems globally are greatly impacted by Taura Syndrome Virus (TSV), a major virus in the penaeid shrimp aquaculture sector (Pinheiro *et al.*, 2007). Taura syndrome, the illness brought on by this virus, mostly affects *Penaeus vannamei*, or Pacific white shrimp, a crucial species in the busy global shrimp production industry (Audelo-del-Valle *et al.*, 2003). Since its first discovery in the early 1990s, TSV has grown to be a significant health risk for aquaculture because of its high mortality rate and quick dissemination in crowded culture settings (Nielsen *et al.*, 2005).

The spread of TSV across nations and continents has accelerated due to the growing intensification and globalization of aquaculture commerce (Wilisiani *et al.*, 2013). One of the main ways that the virus enters new culture systems is through the transfer of fry between areas without thorough health screening (Wertheim *et al.*, 2009). Furthermore, TSV's persistence and spread are facilitated by its stable biological traits in aquatic settings and its capacity to endure under specific circumstances (Ochoa *et al.*, 2020). Because of this, TSV is a significant illustration of a viral disease that affects aquaculture and is heavily impacted by the interplay of biological, environmental, and culture management factors (Vergel *et al.*, 2019).

Clinically, TSV infection can cause a wide range of symptoms, from little discomfort to sudden, severe death (Nielsen *et al.*, 2005). Early signs of infection in the field may include distinctive alterations in tail pigmentation, reduced eating activity, and stunted growth (Dhar *et al.*, 2004). However, without the use of laboratory techniques, early diagnosis is frequently challenging due to the vague character of these symptoms (Surfianti *et al.*, 2010). This circumstance highlights the necessity of an integrated diagnostic strategy for illness prevention (Fadilah and Fasya, 2021).

From a pathobiological standpoint, TSV is a member of the Dicistro-

viridae family of positive-sense, single-stranded RNA (+ssRNA) viruses (Ochoa *et al.*, 2020). This virus demonstrates tropism for ectodermal and mesodermal tissues in shrimp, and despite having a relatively basic genome, it multiplies effectively (Walker and Mohan, 2009). Through processes of necrosis and apoptosis, infection progressively damages cells, causing systemic physiological abnormalities in the host (Audelo-del-Valle *et al.*, 2003).

TSV has a substantial economic impact, especially in intensive aquaculture systems that depend on the production of *P. vannamei* (Taufiqurrahman *et al.*, 2026). Significant yield decreases, higher production costs as a result of prevention measures, and interruptions to the shrimp supply chain in both domestic and foreign markets are all possible outcomes of this disease (Seibert and Pinto, 2012). Additionally, the overall economic impact is made worse by indirect losses in the form of decreased business efficiency and pricing volatility (Dhar *et al.*, 2004).

TSV has indirect effects on global food security even though it is not zoonotic and does not directly endanger human health (Stentiford *et al.*, 2012). Since shrimp are a valuable source of animal protein, production disruptions brought on by this illness may have an effect on the availability of food and the financial stability of communities that depend on the fishing industry (Walker and Mohan, 2009).

Approaches based on biosecurity, environmental management, and the creation of early detection and mitigation techniques are becoming more crucial as the difficulties in controlling TSVs grow (Teng *et al.*, 2006). Supporting the sustainability of the aquaculture sector requires a thorough understanding of this virus, including its etiology, epidemiology, pathophysiology, and control methods (Dhar *et al.*, 2004).

Therefore, the purpose of this review was to provide a comprehensive overview of TSV, encompassing its biological characteristics, transmission dynamics, clinical and economic impacts, and the control approaches that have been and are being developed in modern shrimp farming systems.

Etiology

TSV, a positive-sense, single-stranded RNA (+ssRNA) virus belonging to the family Dicistroviridae, genus Cripavirus, is the cause of Taura syndrome in penaeid shrimp, especially *P. vannamei* (Ochoa et al., 2020). The icosahedral symmetry of this virus's non-enveloped virion helps it remain stable in aquatic conditions and prolongs its ability to survive outside of a host (Dhar et al., 2002).

The two primary open reading frames (ORFs) that encode structural and non-structural proteins make up the about 10–12 kb TSV genome (Robles-Sikisaka et al., 2001). While the second ORF encodes the capsid protein, which is involved in virion production and first interaction with the host cell, the first ORF is typically linked to the replication and processing of the non-structural polyprotein (Lee et al., 2022). This genome structure is comparable to that of other Dicistroviridae family members, which typically use an internal ribosome entry site (IRES) translation method to generate viral proteins (Koh et al., 2014).

There have been reports of molecular genetic heterogeneity among TSV isolates, including variations in pathogenicity and virulence (Tang and Lightner, 2005). It is believed that the virus's capacity to infect host cells and elude the innate immune response in shrimp is impacted by nucleotide alterations in particular genomic areas, especially those producing the capsid protein (Audelo-del-Valle et al., 2003). Geographical variations in the severity of outbreaks are a result of this diversity (Dhar et al., 2004).

The cuticular epithelium, hematological tissue, and peripheral nerve tissue are among the ectodermal and mesodermal tissues in shrimp where TSV exhibits a particular cellular tropism (Ochoa et al., 2020). After the virion attaches itself to the surface of the host cell, it penetrates and releases its RNA genome into the cytoplasm, where it replicates (Dhar et al., 2004).

Temperature, salt, and water quality are examples of environmental elements that influence viral stability in the environment and host vulnerability but do not directly induce infection (Walker and Mohan, 2009). Shrimp under physiological stress may express more cellular components that facilitate viral multiplication, hastening the course of the illness (Lu and Sun, 2005).

Epidemiology

With a global distribution pattern, TSV is a serious viral disease of penaeid shrimp, especially in light of the growth of the international shrimp fry trade and the intensification of aquaculture systems (Limsuwan and Chuchird, 2007). Since its first discovery in Latin America in 1992, TSV has proven to be very adaptable to a variety of aquaculture environmental conditions, hastening its geographic spread into different shrimp-producing locations across the globe (Cheng et al., 2011).

In the past, TSV epidemics were first documented in *P. vannamei* in South America, especially Ecuador, and then moved to Central and North America via the circulation of shrimp fry (Lightner, 2011). The mobility of infected post-larvae that did not exhibit clinical symptoms (subclinical carriers) was the main factor in this transatlantic spread, enabling the virus to enter new aquaculture systems unnoticed at an early stage (Nielsen et al., 2005). Figure 1 illustrates the global epidemiological spread of TSV from its initial emergence in Latin America to other major aquaculture regions worldwide.

Following its discovery in the Americas, TSV was later discovered in a number of Asian nations with significant aquaculture sectors, such as China, Vietnam, Thailand, and Indonesia (Dhar et al., 2004; Phalitakul et al., 2006; Wilisiani et al., 2013; Li et al., 2024). The import of fry and the transfer of biological material between hatcheries that have not put strict pathogen screening in place are intimately associated with the expansion in Asia (Fadilah and Fasya, 2021). TSV has occasionally spread to regions with open culture systems and inadequate biosecurity (Intriago et al., 2024).

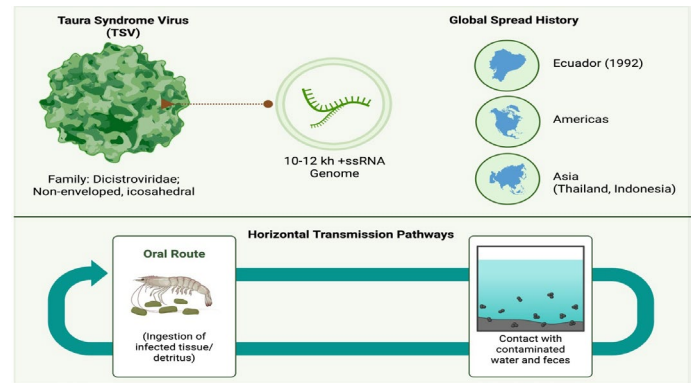


Figure 1. The pathogen and global spread of TSV.

According to epidemiology, intensive culture systems with high densities and erratic environmental variations typically have higher TSV prevalence (Cheng et al., 2011). Increased disease incidence has also been linked to some high-temperature seasons, albeit this trend varies based on production system and geographic region (Iftehimul et al., 2025). These environmental elements contribute to a decrease in host physiological resistance and an increase in viral multiplication (Tumburu et al., 2012).

The persistence of TSV in aquatic habitats is believed to be influenced by environmental reservoirs and the existence of substitute hosts (Nielsen et al., 2005). While penaeid shrimp are the primary site of active reproduction, the virus has also been found in other aquatic creatures, which may serve as mechanical carriers that sustain the virus's natural circulation (Stentiford et al., 2009).

At the moment, TSV's worldwide distribution shows an endemic pattern in a number of important shrimp production areas, with occasional outbreaks that could result in large losses (Cheng et al., 2011). The interplay between seed movement, culture techniques, and the efficiency of biosecurity measures in each location has a significant impact on these epidemiological processes (Moss et al., 2012).

Pathogenesis

The pathogenesis of TSV in penaeid shrimp, especially *P. vannamei*, starts with oral infection or direct contact with contaminated tissue or virus particles found in the aquatic environment (Srisuvan et al., 2006). The virus enters the host, passes through the digestive tract's epithelium, and then travels throughout the body through the hemolymph to reach different target regions (Cao et al., 2010).

The cuticular epithelium, hematopoietic tissue, antennal glands, and peripheral nerve tissue are among the ectodermal and mesodermal tissues that TSV favors (Fadilah and Fasya, 2021). In line with the traits of positive-stranded RNA viruses belonging to the Dicistroviridae family, viral replication takes place in the cytoplasm of the host cell (Srisuvan et al., 2006). This replication activity contributes to gradual tissue damage by disrupting cell metabolism, disorganizing organelles, and inducing necrosis and death (Luo et al., 2004).

The shrimp's innate immune response, which includes hemocyte activation of phagocytosis mechanisms and the synthesis of antiviral compounds such RNA interference (RNAi), coincides with high viral replication during the early phase of infection (acute phase) (Liu et al., 2009). Nevertheless, TSV's capacity to avoid or inhibit these immune reactions permits the virus to endure and proliferate, enabling the infection to advance to the transitional and chronic stages (Audelo-del-Valle et al., 2003).

Epithelial cell degeneration, multifocal necrosis, and pigmentation changes linked to melanin deposition as part of the host's melanization response are typical histopathological damage (Ruangsri et al., 2007). Red tail syndrome is one of the clinical manifestations caused by a combination of tissue injury and a local inflammatory response that impacts pigment distribution and cuticle integrity (Dhar et al., 2004).

Uncontrolled systemic infection can cause substantial mortality in cultured populations, poor osmoregulation, and multisystem physiological failure in advanced stages (Wilisiani *et al.*, 2013). The rate of illness progression and lesion severity are also influenced by environmental factors like high densities and poor water quality, as well as variations in virulence across TSV strains (Walker and Mohan, 2009).

Immune response

Since crustaceans do not have an adaptive immune system like vertebrates do, innate immunity mechanisms dominate the shrimp immune response to TSV infection (Wang *et al.*, 2014). Pathogen-associated molecular patterns (PAMPs), which are identified by pattern recognition receptors (PRRs) on hemocytes and epithelial tissue, initiate the first line of defense (Cox *et al.*, 2024). When this system is activated, a number of humoral and cellular reactions are triggered, which help to restrict viral replication (Lightner, 2011).

Hemocyte activity, which includes phagocytosis, encapsulation, and nodulation of viral particles or infected cells, is a crucial part of the cellular response (Wang *et al.*, 2019). By activating the prophenoloxidase (proPO) pathway, which causes melanin to accumulate around the infection site to isolate the pathogen, hemocytes also contribute to melanization (Ananya *et al.*, 2026). However, overactivation of this system may result in oxidative stress-induced host tissue damage (Kumar *et al.*, 2023).

RNA interference (RNAi) is a crucial antiviral mechanism against TSV at the molecular level (Fajardo *et al.*, 2024). In order to prevent translation and viral genome replication, the RNA-induced silencing complex (RISC) directs the Dicer enzyme's production of small interfering RNA (siRNA), which degrades viral RNA (Ongvarrasopone *et al.*, 2011). Although its efficacy varies based on the virulence of the virus strain and the physiological state of the host, RNAi activation has been demonstrated to have a substantial role in reducing TSV reproduction (Meng *et al.*, 2026).

Moreover, effector molecules such lectins, antimicrobial peptides (AMPs), and reactive oxygen species (ROS) are produced as part of the shrimp humoral response (Tassanakajon *et al.*, 2013). While AMPs have direct antiviral effect through destruction of membrane integrity or suppression of the infection process, lectins contribute to the agglutination of viral particles (Lu and Sun, 2005). Conversely, ROS generation aids in the removal of pathogens but, if left unchecked, can worsen tissue damage (Pantoja *et al.*, 2004).

By altering immune gene expression and inhibiting particular antiviral responses, TSV can upset host immunological homeostasis even when these different defense systems are activated (Fadilah and Fasya, 2021). Because of this interaction, the virus can survive and multiply for a while before the immune system either successfully or unsuccessfully manages the infection, resulting in more serious clinical symptoms (Ochoa *et al.*, 2020).

Pathology

A wide range of pathological changes, from macroscopic lesions to distinctive histological changes in target tissues, are displayed by TSV infection in penaeid shrimp (Lightner, 2011). In line with the virus's preference for cuticular epithelium, hematological tissue, and peripheral organs, the majority of pathogenic alterations often take place in ectodermal and mesodermal tissues (Lightner *et al.*, 1995).

Macroscopically, infected shrimp in the acute phase frequently show diffuse lesions on the exoskeleton that might develop into cuticular necrosis along with crimson staining of the tail (red tail syndrome) (Dhar *et al.*, 2004). In high-density cultivated populations, this situation is frequently followed within a short time by decreased activity, anorexia, and higher mortality (Lu and Sun, 2005). Figure 2 illustrates the progression of TSV infection and its associated clinical manifestations, including characteristic external lesions and pathological changes observed during dis-

ease development.

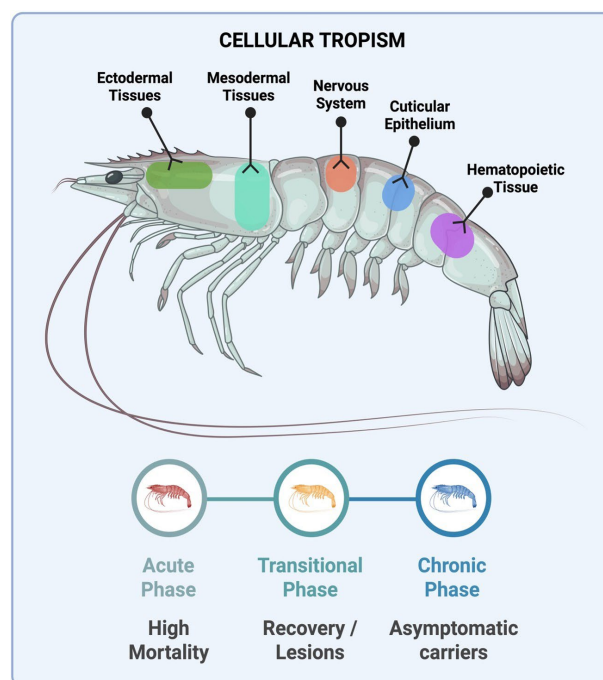


Figure 2. Disease progression and symptoms.

Histopathologically, TSV causes cytoplasmic vacuolization, nuclear pyknosis, and karyorrhexis, which are signs of increasing cellular necrosis in epithelial cells (Escobedo-Bonilla *et al.*, 2025). Infection impairs the host's innate immune system in hematopoietic tissue by disrupting cellular structure and reducing the hemocyte number (George *et al.*, 2011). Moreover, cytoplasmic inclusions linked to viral replication activity in target cells may occasionally be observed (Srisuvan *et al.*, 2006).

A local inflammatory response in the form of hemocyte infiltration and melanization process activation, which leads to the deposition of melanin pigment surrounding the lesion location, is another aspect of pathological alterations (Dhar *et al.*, 2004). Although this method is a component of the pathogen isolation effort, increased oxidative stress and compromised structural integrity may worsen tissue injury (Wilisiani *et al.*, 2013).

Lesions tend to be more limited with less necrosis during the transitional and chronic phases, but tissue function remains impaired (Lightner, 2011). Shrimp that survive the acute phase often only partially recover, but they can continue to spread the virus throughout the community by acting as carriers (Dhar *et al.*, 2004).

Clinical manifestations

Depending on the stage of infection, the environment, and the severity of the virus strain, TSV infection in penaeid shrimp can present with a variety of clinical symptoms (Lightner *et al.*, 1995). Clinical symptoms typically appear quickly during the acute phase, progress to a transitional period, and ultimately result in a chronic phase in those who survive (Lotz *et al.*, 2005).

The most distinctive clinical indication during the acute phase is a reddish staining of the tail, sometimes known as the "red tail sign," which is frequently the first evidence of infection in the field (Lightner, 2011). In more severe cases, this pigmentation change may spread throughout the body, accompanied by atypical or dull skin as a result of integument disturbance (Dhar *et al.*, 2004). Additionally, infected shrimp show reduced movement, a diminished reaction to stimuli, and a propensity to spend time at the pond's bottom (Wilisiani *et al.*, 2013).

Anorexia, or decreased appetite, and poor molting are two physiological abnormalities that accompany illness and affect growth and feed conversion efficiency (Tumburu *et al.*, 2012). Shrimp may occasionally

have soft shell syndrome as a result of abnormal exoskeleton production (Audelo-del-Valle *et al.*, 2003). Particularly in high-density culture systems and unpredictable climatic conditions, mortality during this phase can be swift and severe (Anwar and Safitri, 2024).

Clinical symptoms are usually less severe during the transition phase, but uneven pigmentation changes and poor growth performance are still evident (Lightner *et al.*, 1995). Although the virus remains present in some tissues, shrimp entering this phase often appear to partially recover (Lightner, 2011). Shrimp can become carriers of the disease, contributing to continued transmission of the virus within the population (Wertheim *et al.*, 2009).

In contrast, clinical symptoms are typically nonspecific or even sub-clinical during the chronic period (Dhar *et al.*, 2004). However, by decreasing growth rates and raising vulnerability to environmental stress or secondary infections, latent infections can have an adverse effect on aquaculture productivity (Lotz *et al.*, 2005).

Diagnosis

To achieve precise infection identification, TSV diagnosis necessitates an integrated method that combines clinical observation, histological examination, and molecular-based confirmation (Poulos *et al.*, 2008). This strategy is essential since TSV's clinical signs frequently coincide with those of other viral illnesses in penaeid shrimp (Vergel *et al.*, 2019). Table 1 summarizes various diagnostic methods for TSV in penaeid shrimp, encompassing clinical, histopathological, molecular, and immunodiagnostic approaches.

Clinical symptoms, especially changes in tail pigmentation to redness (red tail syndrome), decreased activity, and an abrupt spike in mortality, are typically seen in the field to make the initial diagnosis (Cheng *et al.*, 2011). Although these signs can raise an initial suspicion, their general character necessitates additional validation (Dhar *et al.*, 2004).

An essential technique in the traditional diagnosis of TSV is histopathological investigation (Vergel *et al.*, 2019). Cell necrosis, cytoplasmic vacuolation, and karyorrhexis are examples of degenerative alterations that can be seen when target tissues including cuticular epithelium, hematopoietic tissue, and gill tissue are examined (Yu and Song, 2000). Although they are not totally specific to TSV, these results are frequently

used to corroborate the suspicion of viral infection, particularly in the acute phase (Dhar *et al.*, 2004).

Molecular techniques based on nucleic acid amplification are typically used for definitive confirmation (Teng *et al.*, 2006). The most popular methods for identifying TSV genomic RNA are reverse transcription polymerase chain reaction (RT-PCR) and quantitative real-time PCR (qPCR) because of their excellent sensitivity and specificity (Dhar *et al.*, 2002). This method enables early infection diagnosis, even prior to the onset of overt clinical signs (Tang *et al.*, 2004). Additionally, because it is quicker and requires simpler equipment, loop-mediated isothermal amplification (LAMP) is starting to be used as a substitute field diagnostic technique (Kiatpathomchai *et al.*, 2008).

Viral antigens in infected tissues are found using immunodiagnostic procedures like immunohistochemistry (IHC) and enzyme-linked immunosorbent assay (ELISA) (Chaivisuthangkura *et al.*, 2014). Although this method's sensitivity is typically lower than that of molecular techniques, it is helpful in examining the distribution of antigens and assessing the degree of infection in certain tissues (Navarro *et al.*, 2009).

An integrated diagnostic strategy, which combines clinical data, histopathology, and molecular results to increase diagnostic accuracy, is the recommended standard in contemporary diagnostic practice (Dhar *et al.*, 2002). Additionally, this method aids in distinguishing TSV from other viral diseases with comparable clinical symptoms, such as Yellow Head Virus (YHV) and White Spot Syndrome Virus (WSSV).

Differential diagnosis

Since the clinical signs of penaeid shrimp sickness are ambiguous and can resemble different viral infections and other pathological states in intense culture systems, differential diagnosis of TSV is essential to diagnosing the disease (Audelo-del-Valle *et al.*, 2003). Without scientific confirmation, similar symptoms like body color changes, tiredness, and increased mortality frequently result in misidentification (Dhar *et al.*, 2004).

White Spot Syndrome Virus (WSSV) is one of the primary illnesses that has to be distinguished from TSV (Sánchez-Paz, 2010). In contrast to TSV infections, WSSV typically shows more fast and severe death along with the formation of white spots on the exoskeleton (Pradeep *et al.*,

Table 1. Diagnostic approaches for TSV in penaeid shrimp.

Diagnostic stage / approach	Method / Technique	Observed parameter	Advantages	Limitations	References
Clinical observation (field diagnosis)	Visual examination	Red tail syndrome, reduced activity, sudden increase in mortality	Rapid and easy to perform under field conditions	Non-specific; clinical signs may overlap with other shrimp viral diseases	Cheng <i>et al.</i> (2011); Dhar <i>et al.</i> (2004)
Histopathological examination	Tissue section analysis (cuticular epithelium, hematopoietic tissue, gills)	Cell necrosis, cytoplasmic vacuolation, karyorrhexis	Provides evidence of characteristic tissue damage associated with viral infection	Not exclusive to TSV; requires confirmatory testing	Vergel <i>et al.</i> (2019); Yu and Song (2000); Dhar <i>et al.</i> (2004)
Molecular diagnosis (primary confirmation)	Reverse transcription polymerase chain reaction (RT-PCR)	Detection of TSV genomic RNA	High sensitivity and specificity	Requires laboratory facilities and specialized equipment	Teng <i>et al.</i> (2006); Dhar <i>et al.</i> (2002)
Quantitative molecular diagnosis	Quantitative real-time PCR (qPCR)	Viral load quantification	Enables early detection before clinical signs appear	Relatively high operational cost	Dhar <i>et al.</i> (2002); Tang <i>et al.</i> (2004)
Isothermal amplification	Loop-mediated isothermal amplification (LAMP) assay	Detection of TSV nucleic acids	Rapid, simple equipment requirements, suitable for field application	Limited validation compared with qPCR	Kiatpathomchai <i>et al.</i> (2008)
Immunodiagnostic approach	Enzyme-linked immunosorbent assay (ELISA)	Detection of viral antigens	Relatively simple and applicable for large-scale screening	Lower sensitivity than molecular methods	Chaivisuthangkura <i>et al.</i> (2014); Navarro <i>et al.</i> (2009)
Immunohistochemistry (IHC)	Specific tissue staining	Distribution and localization of viral antigens within tissues	Enables visualization of infection sites and tissue involvement	Requires complex tissue preparation and technical expertise	Chaivisuthangkura <i>et al.</i> (2014)
Integrated diagnostic approach	Combination of clinical assessment, histopathology, and molecular testing	Integration of all diagnostic findings	High diagnostic accuracy and improved differentiation from WSSV and YHV	Requires complete facilities and multidisciplinary expertise	Poulos <i>et al.</i> (2008); Dhar <i>et al.</i> (2002)

2012). In contrast to TSV, which mostly induces cytoplasmic degeneration and necrosis without consistent nuclear inclusions, WSSV also shows distinctive intranuclear inclusions in ectodermal and mesodermal tissues histopathologically (Verbruggen *et al.*, 2016).

Apart from WSSV, Yellow Head Virus (YHV) is also a significant distinction because it can cause quick mortality and changes in body color (Senapin *et al.*, 2010). However, more noticeable hepatopancreatic tissue damage and yellow staining of the cephalothorax are frequently associated with YHV (Sittidilokratna *et al.*, 2006). YHV's target tissues and lesion distribution are very different from TSV's, which mainly affects cuticular epithelium and peripheral tissues (Dhar *et al.*, 2004).

Early-stage TSV infection can similarly resemble infectious myonecrosis virus (IMNV) infection, especially in terms of reduced activity and death (Saravanan *et al.*, 2025). However, IMNV is more commonly associated with alterations in muscle tissue opacity and significant skeletal muscle necrosis, which are not typical of TSV (da Silva *et al.*, 2015).

Similar clinical signs, such as decreased appetite and lethargy, can also be caused by non-infectious disorders such environmental stress brought on by warmth, salt, or poor water quality (Ochoa *et al.*, 2020). Nevertheless, neither the organized mortality pattern nor the distinctive histological abnormalities of TSV infection are typically seen in these circumstances (Nielsen *et al.*, 2005).

Transmission

In intensive shrimp farming methods, TSV is mainly transmitted horizontally, which is quite effective (Escobedo-Bonilla *et al.*, 2025). When virus particles from infected shrimp reach the aquatic environment and build up in organic materials including exuviae, excrement, and decomposing tissues, transmission takes place (Dhar *et al.*, 2004). These contagious particles can linger in the water column and infect susceptible shrimp orally (Surfianti *et al.*, 2010).

The main method of transmission is ingestion, when healthy shrimp contract the infection by eating feed or detritus tainted with TSV (Audelo-del-Valle *et al.*, 2003). The shrimp's detritivorous eating habits facilitate this process and increase the likelihood that the infectious agent will enter the digestive tract (Kumar *et al.*, 2026). The virus then infects epithelial cells and travels throughout the body after passing through the gastrointestinal barrier (Luo *et al.*, 2004).

Particularly in areas with dense populations, direct contact between people also plays a role in the spread of viruses (Tang *et al.*, 2012). Viral particles can spread across contaminated body surfaces or exoskeletons through frequent physical contact (Lightner *et al.*, 1995). Additionally, the infectious pathogen may have extra entry points due to minute cuticle lesions (Intriago *et al.*, 2025).

One significant passive vector in the spread of TSV is water (Dhar *et al.*, 2002). The virus can spread at pond sizes and between hydrologically related aquaculture systems when it is free or attached to suspended particles (Verdegem *et al.*, 2023). The efficacy of this transmission is influenced by TSV's stability in the aquatic environment (Oidtmann *et al.*, 2018).

An significant introduction route in the geographic spread of TSV is the migration of infected larvae and early-stage shrimp that do not exhibit clinical symptoms (Vergel *et al.*, 2019). One of the main causes of the proliferation across nations and regions has been found to be the distribution of fry without pathogen-free (SPF) screening (Anwar and Safitri, 2024). Additionally, automobiles, people, and aquaculture equipment can all serve as fomites, making it easier for the virus to mechanically spread between production facilities (Nielsen *et al.*, 2005).

Although the role of active virus replication in non-target hosts has not been fully established, a number of additional aquatic creatures are thought to serve as reservoirs or mechanical carriers (Nielsen *et al.*, 2023). Even in the absence of clinical outbreaks in the primary population, the existence of these organisms may sustain virus circulation in the environ-

ment (Ochoa *et al.*, 2020).

Risk factors

A number of interrelated risk variables, including as biological, environmental, and managerial elements, affect the success of TSV infection and transmission in shrimp farming systems (Walker and Mohan, 2009). The degree of sensitivity of shrimp populations and the dynamics of outbreaks in the field are determined by the combination of these elements (Dhar *et al.*, 2002).

High stocking density in intensive agricultural systems is one of the main danger factors (Escobedo-Bonilla *et al.*, 2025). This circumstance raises the likelihood of horizontal virus transmission by increasing the frequency of interpersonal contact and the buildup of organic material in the rearing conditions (Audelo-del-Valle *et al.*, 2003). In intensive culture systems, stocking densities exceeding 150–300 shrimp/m² are frequently associated with progressive deterioration of water quality, particularly when accompanied by inadequate water exchange and aeration (Jiang *et al.*, 2005). Under these conditions, total ammonia nitrogen (TAN) concentrations above 1.0 mg/L or un-ionized ammonia (NH₃) levels exceeding 0.05 mg/L, together with dissolved oxygen concentrations below 4.0 mg/L for several consecutive days, have been associated with physiological stress, immunosuppression, and increased susceptibility to viral diseases, including TSV (Lin and Chen, 2001; Liu and Chen, 2004). Moreover, lower environmental quality, such as elevated ammonia, lower dissolved oxygen, and imbalances in the aquatic microbiota, is frequently linked to excessive stocking density (Xiong *et al.*, 2024).

The onset of TSV infection is also significantly influenced by the quality of the fry (post-larvae) (Walker and Winton, 2010). The risk of viruses entering the farming system is increased when fry are used that are not from specified pathogen-free (SPF) sources (Murray *et al.*, 2021). The dispersion of carriers who appear clinically healthy but have latent virus is more likely when molecular screening is not done at the hatchery stage (Nielsen *et al.*, 2005).

Host susceptibility is greatly influenced by environmental conditions (Ødegård *et al.*, 2011). Pacific white shrimp (*Litopenaeus vannamei*) generally perform optimally at salinities of 15–25 ppt and water temperatures between 28–32°C. Abrupt salinity fluctuations exceeding 5–10 ppt within 24 hours or temperature shifts greater than 3–5°C over a short period can induce significant physiological stress (Lin and Chen, 2001). Likewise, prolonged exposure (48–72 hours) to suboptimal water quality, including elevated ammonia or reduced dissolved oxygen, impairs innate immune responses, facilitating the progression of latent or subclinical TSV infection to overt clinical disease (Dhar *et al.*, 2004). Shrimp can experience physiological stress from abrupt changes in salinity, extreme temperature swings, and poor water quality, which lowers the innate immune system's efficacy (Lemaire *et al.*, 2002). These circumstances hasten the infection's transition from the subclinical to the symptomatic stage (Dhar *et al.*, 2004).

Another important risk element is inadequate biosecurity procedures (Ahmadivand *et al.*, 2025). The virus can spread through fomite mechanisms when unsterilized equipment is used, water is managed without the best filtering or disinfection, and human access to production areas is uncontrolled (Fadilah and Fasya, 2021). Additionally, the likelihood of disease introduction from external reservoirs is increased in open culture systems with direct linkages to the natural environment (Bhassu *et al.*, 2024).

The TSV infection cycle may also be sustained by the presence of carrier organisms in the aquatic environment, whether they come from wild shrimp species or other aquatic organisms (Ochoa *et al.*, 2020). These creatures can serve as reservoirs, allowing the virus to remain in the ecosystem even though they don't usually exhibit clinical symptoms (Nielsen *et al.*, 2005).

Public health importance

TSV cannot directly infect humans and is not categorized as a zoonotic agent (Dhar *et al.*, 2004). However, through food security, economic stability, and the safety of fishery-based food production systems, its presence in shrimp aquaculture systems has indirect effects on public health (Walker and Mohan, 2009).

Shrimp is a valuable source of animal protein that helps populations satisfy their nutritional demands in the context of food security, especially in coastal regions and developing nations where aquaculture is the main source of food production (Lee *et al.*, 2022). The availability of protein sources may be disrupted by a TSV outbreak that significantly reduces productivity, which might have an immediate and long-term effect on the accessibility of nutrient-dense food (Iftheimul *et al.*, 2025).

Additionally, the stability of shrimp commodity prices in both domestic and foreign markets may be impacted by production disruptions brought on by TSV (Akte *et al.*, 2026). Price volatility may rise as a result of supply fluctuations brought on by crop failures or reduced production, which may eventually affect the distribution of food derived from fisheries and public purchasing power (Dhar *et al.*, 2004).

From the standpoint of food safety, disease conditions in aquaculture systems can impact the quality of the finished product by degrading the physiological state of the shrimp, raising oxidative stress, and increasing the possibility of secondary infections by opportunistic microorganisms, even though TSV does not present a risk of infection to humans (Lightner, 2011). Thus, one of the most important components of a fishery food quality assurance system is the management of viral infections in aquaculture (Zarain-Herzberg and Ascencio-Valle, 2001).

The One Health approach, which highlights the connections between human, animal, and environmental health, also makes TSV pertinent (Nwokedi *et al.*, 2025). As part of a sustainable food production system, disease control highlights the significance of a balanced aquaculture ecosystem, sound biosecurity procedures, and fish and shrimp health monitoring (Assefa and Abunna, 2018).

Economic impact

The shrimp aquaculture industry is significantly impacted economically by TSV infection, especially in intensive farming systems where *P. vannamei* is the main product (Lightner, 2011). Reduced output as a result of increased mortality during the acute phase, which can result in partial or complete crop failure within a single farming cycle, is the main cause of economic losses (Walker and Mohan, 2009).

In addition to mass mortality, impaired growth and decreased feed conversion efficiency in surviving individuals also contribute to the decline in production (Lu and Sun, 2005). Shrimp size quality and harvest biomass are decreased as a result of this condition, which immediately reduces market value (Valderrama and Engle, 2004). Additionally, the farming system's infection cycle may be prolonged by the existence of carrier individuals, extending the time of subpar output (Dhar *et al.*, 2004).

Farmers must pay more for disease prevention and control measures, such as buying pathogen-free (SPF) fish, enhancing biosecurity systems, cleaning facilities, and putting in place routine health monitoring (Lotz, 1997). These extra expenditures lower profit margins by raising production expenses per unit of output (Shinn *et al.*, 2018).

The supply chain and the stability of the shrimp export market are also impacted by TSV disruptions (Fadilah and Fasya, 2021). The reputation of producing nations and the competitiveness of commodities in the global market can both be impacted by unstable production swings that affect the continuity of supply to international markets (Karunasagar and Ababouch, 2012). Major epidemics may cause importers to lose faith in the provenance of goods from impacted regions (Stentiford *et al.*, 2012).

At the socioeconomic level, small and medium-sized fish farmers are more affected by TSV because they lack the resources to implement thor-

ough risk mitigation (Walker and Mohan, 2009). Crop failure losses can impair the sustainability of aquaculture enterprises in coastal areas, lower household incomes, and raise economic vulnerability (Janmaimool *et al.*, 2025).

Treatment

There is currently no particular treatment that will completely eradicate TSV in penaeid shrimp (Lightner, 2011). This is because the host immune system only uses innate immune processes, while RNA viruses are intracellular (Alam *et al.*, 2023). Therefore, rather than directly eliminating the virus, therapeutic approaches to TSV concentrate more on infection control, reducing clinical effects, and boosting host resistance (Lu and Sun, 2005).

Supportive interventions are frequently employed in aquaculture procedures with the goal of lowering the shrimp's physiological stress levels (Lotz *et al.*, 2005). Maintaining water temperature near the optimal range (approximately 28–32°C), salinity around 15–25 ppt, dissolved oxygen above 5 mg/L, and minimizing ammonia accumulation are fundamental components of supportive management (Liu and Chen, 2004). Prolonged exposure to suboptimal water quality impairs hemocyte function and innate immune responses, thereby increasing the severity of TSV outbreaks (Lightner, 2011). Reducing the spread of infection requires improving environmental quality, which includes regulating the buildup of dissolved nitrogen compounds and stabilizing temperature, salinity, and dissolved oxygen levels (Taufiqurrahman *et al.*, 2026). The innate immune system is known to be strengthened and disease development slowed under ideal environmental conditions (Dhar *et al.*, 2004).

Another crucial strategy for managing TSV is the use of immunostimulants (Sookruksawong *et al.*, 2013). Probiotics, plant extracts, decreased lipopolysaccharide, and β -glucan have all been shown to boost hemocyte function, fortify the prophenoloxidase system, and improve the synthesis of endogenous antiviral compounds (Taufiqurrahman *et al.*, 2026). This method lowers mortality and increases shrimp resilience to secondary infections, but it is not therapeutic (Lu and Sun, 2005).

Additionally, maintaining the physiological state of shrimp throughout infection is aided by appropriate dietary control (Audelo-del-Valle *et al.*, 2003). Vitamin C, vitamin E, critical fatty acids, and certain amino acids can be added to feed formulations to help repair tissue integrity impacted by viral infection and lessen oxidative stress (Taufiqurrahman *et al.*, 2026).

RNA interference (RNAi), which targets the TSV genome to prevent viral replication, has been investigated as a possible antiviral technique in a number of experimental methods (Alam *et al.*, 2023). Despite demonstrating encouraging outcomes in the lab, its practical use is still restricted because of issues with molecular stability and delivery effectiveness (Nielsen *et al.*, 2005).

Vaccination

The biological traits of penaeid shrimp, which lack an adaptive immune system like vertebrates, provide major obstacles to current TSV vaccination efforts (Bhassu *et al.*, 2024). The development of traditional vaccinations that can offer long-term protection is significantly hampered by the absence of an antibody-based immune response and immunological memory (Dhar *et al.*, 2004).

While several immunoprophylactic strategies have been investigated, the majority are still in the experimental stage (Audelo-del-Valle *et al.*, 2003). Inactivated vaccines, recombinant vaccines based on viral structural proteins, and nucleic acid-based methods including DNA vaccines and RNA interference (RNAi)-based vaccines are among the most extensively researched methodologies (Giese, 2015). These strategies seek to stimulate RNA silencing mechanisms, boost antiviral gene expression, and activate hemocytes in order to strengthen the innate immune response

(Alam *et al.*, 2023).

Shrimp resistance to infection may be increased by inactivated TSV vaccines made by physical or chemical inactivation, however the resulting degree of protection is typically erratic and fleeting (Lu and Sun, 2005). Although their effectiveness varies between investigations and under growing conditions, recombinant vaccines that target the viral capsid protein have been demonstrated to elicit a cellular immune response (Hajimasalaeh *et al.*, 2013).

Approaches based on RNA interference (RNAi) are currently thought to be among the most promising methods for controlling TSV (Fajardo *et al.*, 2024). This method inhibits viral replication early in the infection process by directly targeting viral RNA breakdown using small interfering RNA (siRNA) (Ongvarrasopone *et al.*, 2011). The RNA molecules' stability in the aquatic environment and the efficiency of transport to the target tissues, however, are the biggest obstacles to this strategy (Dhar *et al.*, 2004).

Immunostimulation techniques are frequently classified as a supplemental strategy to TSV prevention in addition to traditional immunization (Li *et al.*, 2024). It is well established that using bioactive substances like β -glucans, immunomodulatory peptides, and plant extracts can boost the shrimp's innate immune system's activity and increase their resistance to viral infections (Nolasco-Alzaga *et al.*, 2025).

Control

An integrated strategy that prioritizes preventing introduction, severing the chain of transmission, and boosting the production system's resilience is needed to control TSV in shrimp farming systems (Nielsen *et al.*, 2005). Control techniques are essential to lessening the effects of this condition because there are no effective specific medicines (Iftehimul *et al.*, 2025). Figure 3 illustrates the integrated management strategies for TSV control in shrimp aquaculture, including biosecurity measures, the use of pathogen-free seed, environmental management, and health monitoring.

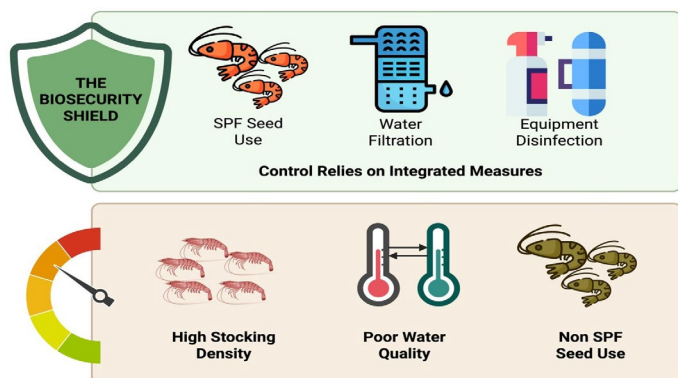


Figure 3. Management of TSV.

Implementing stringent biosecurity at every level of production is the most basic step in TSV control (Wilisiani *et al.*, 2013). This comprises managing incoming and exiting water through filtration and chemical or physical treatment, sterilizing equipment, limiting personnel access, and disinfecting culture facilities (Audelo-del-Valle *et al.*, 2003). To reduce contact with pathogen sources from the outside world, closed or semi-closed culture methods are also advised (Dwinanti and Yuhana, 2009).

One of the most important ways to stop TSV from entering aquaculture systems is to utilize specific pathogen-free (SPF) seed (Moss *et al.*, 2012). Early identification of carrier individuals is made possible by molecular screening at the hatchery stage, especially with RT-PCR or qPCR techniques (Tang *et al.*, 2004). This stops the virus from spreading to the grow-out stage (Wilisiani *et al.*, 2013).

A major factor in reducing the occurrence of disease is aquaculture environmental control (Munang'andu, 2016). To lessen physiological

stress on shrimp, stable water quality parameters such temperature, salinity, dissolved oxygen, and ammonia levels must be kept within ideal ranges (Lotz *et al.*, 2005). It is well established that stable environmental circumstances lower the rate of viral replication and increase the efficacy of the innate immune response (Fajardo *et al.*, 2024).

Another important component of mitigating techniques is controlling stocking density (Gbaoondo *et al.*, 2026). Reducing infection pressure requires modifying stocking densities according to the aquaculture system's capabilities since excessively high densities raise the risk of horizontal transmission through direct contact and water media (Iftehimul *et al.*, 2025).

Additionally, early viral discovery prior to widespread epidemics is made possible by the implementation of routine health monitoring systems by clinical and molecular surveillance (Wertheim *et al.*, 2009). This strategy makes it possible to take quick action, like separating contaminated units or harvesting early to reduce losses (Phalitakul *et al.*, 2006).

Probiotics and biocontrol agents are also starting to be applied as part of a support strategy, especially to improve the physiological resilience of shrimp and preserve the equilibrium of the aquaculture microbiota (Lakshmi *et al.*, 2013). This strategy helps lower host vulnerability to infection even if it does not directly target TSV (Nolasco-Alzaga *et al.*, 2025).

Conclusion

TSV is an important RNA disease of penaeid shrimp that mostly affects *P. vannamei*. It has been a major cause of losses in aquaculture productivity worldwide. Due to intensive farming methods, seed mobility, and variable environmental circumstances, the virus has a high capacity for horizontal transmission. Early detection without laboratory confirmation is frequently complicated by a variety of clinical symptoms, from rapid death to alterations in pigmentation.

Pathobiologically, TSV causes systemic physiological disruptions in the host by causing tissue damage through intracellular replication that targets ectodermal and mesodermal tissues. Despite not being zoonotic, its effects on shrimp supply chains and aquaculture productivity make it a crucial problem for fisheries economics and food security.

Currently, biosecurity, the use of pathogen-free seed, and environmental management are among the preventative measures used in TSV control. Since there is now no effective specific treatment, prevention-based mitigation techniques are still essential to lessening the effects of this illness.

Acknowledgement

The author thanks to Badan Riset dan Inovasi Nasional for the funding support for this study.

Conflict of interest

The authors declare that there is no conflict of interest.

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