Zoonotic Transmission of *Vibrio vulnificus*: Risks Associated with Seafood Consumption

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Abstract

Vibrio vulnificus is a species of negative-staining, locomotive, arched bacterium that belonging to Vibrio genus or the Vibrionaceae family. Lethal zoonotic pathogenic *V. vulnificus* are commonly present in warm, aquaculture brackish water. The origin and routes of contamination are quite diverse for copious and aquaculture sites with the conjunction of risk factor. The expansion of virulent Vibrio bacteria in sustainable seafood farming, plant-based seafood substitutes and wild fishing, depends on the bacterial virulence level, the animal immunity level and health (a diverse range of virulence factor, acid neutralization, cytotoxicity, iron acquisition etc.), the environmental conditions. Consumption of raw seafood contaminated with *V. vulnificus* can cause serious sudden and severe infection (Vibriosis, Septicemia, Skin infections). It is fatal for immunosuppressed people with mean fatality rate surpassing in various states. Necrotizing fasciitis and inflammatory infection (sepsis) are induced by the *V. vulnificus* infections, which has a high mortality rate (64.9%) and is associated with liver dysfunction in 91.6% of cases. Extensive skin lesions, low platelet count, and advanced age are major risk factors for outcomes. Some strategies that are used for preventing *V. vulnificus* foodborne infection include post-harvest treatment, sustainable processing and depuration. In this chapter, an extensive overview of various facets of disease patterns and dynamics of *V. vulnificus*, and virulence mechanism, risk associated with consumption of contaminated seafood.

Keywords: Zoonosis, Transmission, Virulence, Foodborne pathogen, Risk factors, Preventions.

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Introduction

The genus vibrio is a part of the family *Vibrionaceae* (which includes, *Aeromonas, Plesiomonas*, and *Photobacterium*) (Onohuean and Nwodo, 2025), is widely distributed in marine environments. Most vibrio species require sodium chloride for growth purpose except two species which are *V. cholera* and *V. mimicus*. Genus Vibrio consists of 30 species; in which 13 are very harmful and detrimental to humans, that includes *V. cholerae*, *V. mimicus*, *V. fluvialis* etc. They are curved Gram-negative and they are non-spore forming rods but become straight when grown in lab. They are 0.5 to 0.8μ m in width and 1.4 to 2.6μ m in length (McLaughlin, 1995). *V. vulnificus* and *V. parahaemolyticus* both are alike by their phenotype.But different through lactose fermentation and β -D-galactosidase tests (Zhang et al., 2023). Healthcare burden is the overall impact of a health condition on individuals, communities, and healthcare systems. Which counts for direct costs of medical care, such as hospitalizations and medications, as well as indirect costs like lost productivity, transportation expenses, and reduced quality of life. The mortality rate from the infection of *V. vulnificus* approximately 40 cases reported annually in the U.S.

Life cycle of *V. vulnificus*

V. vulnificus locate nutrient-rich surfaces by using chemotaxis and motility, enhanced by overload conditions of iron and high temperatures (above 25°C). Because level of iron causes capsule and pilli production which affects formation of biofilms and dispersal (Pajuelo et al., 2016). Under iron deficiency biofilms are favored, high iron causes dispersion. Exoprotease (*V. vulnificus* protease), that drive chemotaxis, is very important for colonization by the degradation of mucin and hemoglobin, to attract bacteria for establishment and expansion. Activity of Vvp more active at 28-37°C (Valiente et al., 2008a; Elgaml and Miyoshi, 2017; Hernández-Cabanyero et al., 2020), but is inactive below the 20°C. For attachment, *V. vulnificus* firstly attached to the mucin on mucosal surfaces and then epithelial cells with their associated receptors (Goo et al., 2006; Lee et al., 2010; Jang et al., 2016) using adhesins like flagellins, pilins and proteins of outer membrane. After colonization the epithelium, *V. vulnificus* secretes several toxins or exoenzymes to use the nutrients of tissues (Reverter et al., 2018; Chernyavskikh et al., 2019) to support growth. Gene expressions pertaining to metabolic activity, nutrient carriage and ovulniobactin biosynthesis increased by high temperatures that boost colonization rates (Hernández Cabanyero et al., 2020).

Sources and Contaminated Routes of Seafood

As human pathogenic *V. vulnificus* that are normally present in warm, brackish seawater. There are many entryways for the transmission of *V. vulnificus* to seafood chain and due their consumption that further causes infection and serious illness in humans (Ndraha et al., 2020; Noorian et al., 2023). The origin and routes of contamination are quite diverse for copious fishery and aquaculture sites with the conjunction of risk factors (Ndraha et al., 2020; Noorian et al., 2023). The expansion of virulent Vibrio bacteria in sustainable seafood farming, plant-based seafood substitutes, wild fishing, depends on the bacterial virulence level, the animal immunity level and health, the and environmental conditions (Destoumieux-Garzón et al., 2020). Contamination of seafood typically initiated through two routes. First, bacteria adhere to marine organism by direct contact in contaminated water, or targeting surfaces like gills, skin, or shells. Attachment is fundamental for transmission and colonization. Research shows that *V. vulnificus* forms biofilm on these surfaces enveloping in extracellular polymeric substances (EPS) made up of polysaccharide, protein, lipids, nucleic acids etc. (Carrascosa et al., 2021). The ability of *V. vulnificus* to attach to the surface and in the formation of biofilms is facilitated by flagella and pilli on their surfaces, is fundamental in pathogenic colonization and sustained survival in oyster.

Second, *V. vulnificus* can contaminate seafood through the feeding and absorption process, through sifting or through the skin injuries via the mouth, wound and gills. It has ability to chemotactically drawn alongside the mucus over the gills, colonize their and form biofilm their and cause skin lesion or other widespread infections. It can cause the local lesion if it enters through open wound or ingestion, but if it enters into blood streams and reach internal organs, it potentially causing hemorrhagic septicemia, which can be lethal, or when enters via the ingestion *V. vulnificus* generally adhere to intestinal mucus, colonize the intestine, enter in blood stream leading to viral infection (Paranjpye et al., 2007; Fouz et al., 2010; Aagesen et al., 2013).

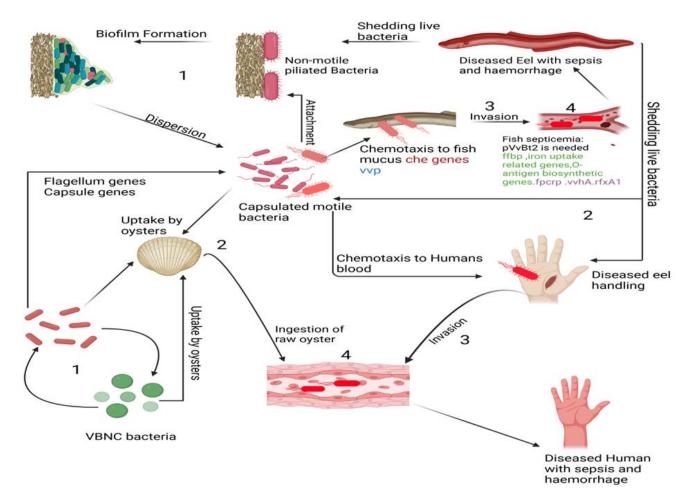


Fig. 1: *V. vulnificus* can occur as free-living and have the proficiency to make a microbial community or film over the substrate. Bacteria goes to a dormant state called VBNC at low temperature, while high temperature it becomes reactivated and leads to biofilm dispersal and resurgence. (2) Bacteria enters into blood and mucus of host and activate colonization and filtering organisms like shellfish can engulf bacteria and transmit to humans through seafood consumption, warmer temperature and iron level enhance this process. (3) Once the bacteria settled and radiated in bloodstreams leads to lesion and inflammation. (4) In order to evacuate human immunity, the *V. vulnificus* secrets protective capsule under high iron presence. In fishes, bacteria developed outer membrane to enhance resistance to immune defense system. The production of toxins leads to the sepsis a state in which body's immune response cause tissue damage. (5) Propagation to the novel carriers and the habitat occurs when infected fish causes infection and disease in *Homo sapiens* by physical exposure and consumption of infected seafood and shed live bacteria in the water as well (Retrieved from bioRender).

V. vulnificus also adhere with the chitinaceous surfaces like chitin-rich surfaces of crabs and shrimps have been discovered as optimal substrates for V. parahaemolyticus and Vibrio cholerae to adhere and establish biofilms (Roy et al., 2021). Once human pathogenic V. vulnificus adhere to the chitinous surfaces, they secrete chitinases that dismantle chitin into N-acetylglucosamine, which act as supplier for the carbon and nitrogen. This process not only provide needed nutrients for bacterial proliferation but also construct opening for colonization leads to internal infection and contamination as shown in Figure 1. V. vulnificus, like V. parahaemolyticus and V. cholera, have ability to adhere with planktonic organisms such as copepods and diatoms, which are the part of marine food web. These planktons are frequently consumed by aquatic organism in aquaculture (Roy et al., 2021; Carrascosa et al., 2021) so bacterial adherence to them may facilitate contamination of seafood, increasing chances for human infection when it is consumed raw. Filter-feeding organism, like mollusks and crustaceans, can accumulate nutrients, pollutants, and pathogenic bacteria including V. vulnificus, from the surrounding water which leads to injurious for humans when consumed raw. In fish, profound exposure of eels with V. vulnificus illustrate that can attach with gills and reach and infect the internal organs by entering and circulating through blood streams (Valiente et al., 2008b).

Zoonotic Transmission of V. vulnificus through Seafood Consumption

The virulence and invasiveness of V. vulnificus are largely linked to various factors. First, encapsulated strains are more pathogenic because of the presence of mucopolysaccharide capsule that enable the bacteria to evade the host immune system. Second, sialic acid-like molecules production that alter the lipopolysaccharide structure, allowing motility and biofilm formation leads to their survival in aquatic environment and in bloodstreams. Third, N-acetylglucosamine-binding protein A promotes adhesion to chitin (in shellfish) and to mucin in gastrointestinal track in humans. Pathogenicity also enhanced by the proteases released by the V. vulnificus that have the ability to degrade the tissues that promoting necrotizing wound infections by enhancing vascular permeability and causing edema (Phillips and Satchell, 2017; Baker-Austin and Oliver, 2018). Zoonotic transferal of V. vulnificus carried out by two ways. First, ingesting a raw seafood contaminated with bacteria leads to gastroenteritis or primary septicemia (Heng et al., 2017). Secondly, when the bacteria enter through the open wound exposed to the seawater or contaminated animals that occurs when a person have existing wound swims in contaminated water or by injury as shown in Figure 2. Minor skin breaks or ruptures can also provide a transmission route for the bacteria. Bacteria can cause severe infections leads to necrotic tissue excision and limb removal (Heng et al., 2017; Coerdt and Khachemoune, 2021).

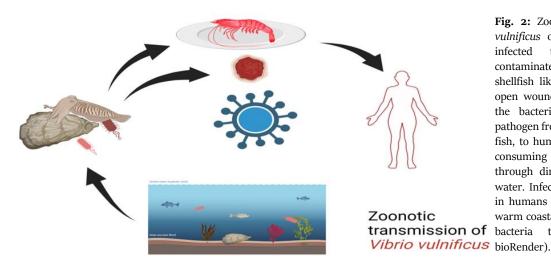


Fig. 2: Zoonotic transmission of V. vulnificus occurs when humans are infected through contact with contaminated seafood, particularly raw shellfish like oysters, or by exposing open wounds to seawater containing the bacteria. This transfer of the pathogen from marine animals, such as fish, to humans can happen either by consuming contaminated seafood or through direct contact with infected water. Infections are especially severe in humans and are more common in warm coastal environments where the transmission of bacteria thrive (Retrieved from

How host body react? Immunological Resistance in Blood

Throughout the case of fish, reduction in blood iron triggers anaerobic metabolism and nitric oxide protection. Vulnificus manufacture a secure envelop that have high molecular weight LPS and outer membrane proteins with iron regulation, like Transferrin receptor protein 1 that provide the ability to resist the phagocytosis (engulfing process). High temperatures of environment increase antioxidant enzyme production, LPS biosynthesis, iron uptake system, membrane regeneration, aiding survival (Hernández-Cabanyero et al., 2020) as shown in Figure 3. Among humans, V. vulnificus virulence is caused by iron overload in blood. The vulnificus capsule, crucial for resisting complement and phagocytosis, is upregulated under high iron conditions. Excess of iron also increase anaerobic metabolism and biosynthesis of capsule (Carda-Diguez et al., 2018) that provide structural advantages. In addition, RtxA1 toxin, overexpressed in iron-rich serum, that kills phagocytes, increasing immune resistance thus increasing risk of septicemia.

Toxins and Sepsis

Cells of Neutrophils, macrophages and endothelial play role in response, manufacturing exacerbating immune response factors (IL-1β, IL-6, IL-8, TNF-α). On other hand, patients with chronic liver diseases exhibit low cytokine levels. The toxin RtxA1, drives this dysregulation that includes cytokine storm (Murciano et al., 2017) and upregulating immune-related genes such as cytokines, interferons, inflammasomes, and transcription factors in both animal and seafood models. Other factors include, LPS, IlpA and bacterial slime layer or glycocalyx contribute to excessive cytokine production in preparation (Hernández-Cabanyero et al., 2020). Vibrio vulnificus hemolysin A (VvhA) toxin may synchronize with RtxA1, during sepsis, with VvhA lysing erythrocytes that create iron-rich environment that increase RtxA1 activity (Jeong and Satchell, 2012).

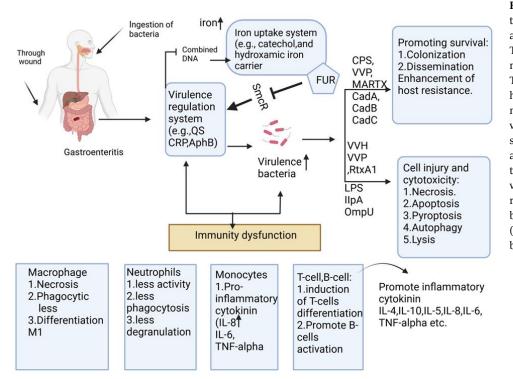


Fig. 3: Bacteria can control the amount of iron available in the host's body. This helps them become more harmful (virulent). They do this by affecting how immune cells like neutrophils and monocytes work. Bacteria use special systems to take up iron and also have ways to control their virulence genes. This weakens the immune response and allows the bacteria to survive better (Retrieved from bioRender).

Diseases caused by V. vulnificus in Humans

Infections in humans caused by this bacterium come from two different sources: eating seafood (primary septicemias) or coming into contact with seawater or seafood items (wound infections). *V. vulnificus* can lead to serious, possibly fatal infections in individuals who are susceptible. This bacterium spreads through handling or eating effected seafood, especially oysters that are eaten uncooked or undercooked. They can enter directly into the wounds of swimmers. By eating the effected seafood, gastroenteritis might arise and its symptoms are abdominal discomfort, vomiting and nausea, that usually causes fever, chills and skin-related signs as shown in Figure 4 (Gulig et al., 2003; Dechet et al., 2008; Jones et al., 2009; Daniel, 2011).

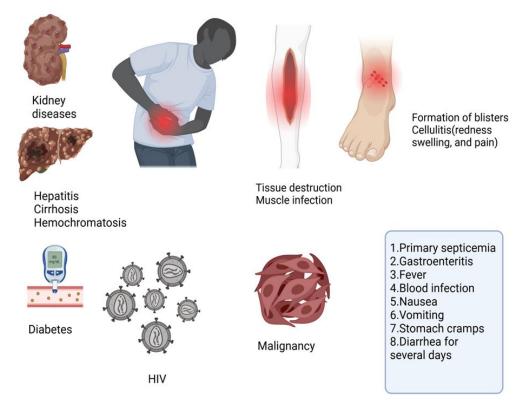


Fig. 4: This image illustrates factors risk the and symptoms associated with vulnificus infections. V_{\cdot} Conditions such as diabetes, liver diseases, and weakened immunity from HIV or malignancy heighten susceptibility. Key symptoms include tissue damage, cellulitis, septicemia, and gastrointestinal issues like nausea and diarrhea. The bacteria pose significant dangers, particularly to Immune-deficient individuals (Retrieved from bioRender).

Because these symptoms are not scary or shocking, mostly people ignore them and do not report in hospital. Sometimes *V. vulnificus* is likely to be life-threatening, especially in the case where it causes skin infection (Primary septicemia) and flesh-eating disease that leads towards invasive soft tissue infection leads to necrotic tissue excision. Septicemia has more than 50% mortality rate and is more common of aquatic infections than any others small intestine or cecum is thought to be entry point (Candelli , 2025). A specific source cannot be pinpointed for bacteria that invade in blood and causes primary septicemia. Fever, vomiting, chills, diarrhea, abdominal pain and pain in arms, legs are the specific symptoms that shows one has caught primary septicemia.

The symptoms show in 7-14 days after eating the contaminated seafood. In one Day of illness, patients may develop skin problems like redness and swelling, large fluid-filled blisters called bullae or bruises especially on the arms and legs. When swimming or while handling seafood, the direct contact of bacteria that enters open wounds leads to necrotizing fasciitis. The malady can be tame to fierce and its signs might be shown in 7 days to 14 days. If it gets even worse, it might spread to surrounding skin and tissue or the cells might start to die, which is called necrosis, but the good news is, the fatality rate is not higher than 20-30% which is less than primary septicemia. Primary septicemia spreads, but rapidly progressive soft tissue infection does not, it is remained to the specific area. Additionally, it causes some unusual symptoms like arthritis, brain infection, bone infection known as osteomyelitis, infection of eye known as endophthalmitis and keratitis (Horseman and Surani, 2011; Karunasagar, 2014).

Geographical and Seasonal Influence

Infections caused by *V. vulnificus* are uncommon, and in studies conducted in coastal regions of the United States of America, the yearly occurrence of infection caused by *V. vulnificus* is roughly 0.5 per 100,000 individuals (Candelli , 2025). Recent estimates from the CDC indicate that the yearly average occurrence of Vibrio infections rose by Forty one percent from 1996 to 2005 (CDC, 2005; Bross et al., 2007). Infections caused by *V. parahaemolyticus* are significantly more prevalent than those from *V. vulnificus* (approximately 10k cases annually in the United States of America). *V. parahaemolyticus* illness is historically linked to seasonal instances occasionally found during the hottest season of the year in both temperate and tropical regions. They are often located in estuaries, which are vulnerable to numerous alterations caused by climate and human-induced pressures. Rising temperatures and sea levels are anticipated to change the regional distribution of various wetland, marshes and estuary (amplify, diminish, and/or transition) and modify the habitat spectrum of numerous biota, involving harmful bacteria. The geographic distribution of *V. vulnificus* is expected to expand as water temperatures rise and saline waters intrude deeper into areas of coastal rivers that were previously freshwater (Deeb et al., 2018).

Environmental Factors

In the Lower-eastern United States of America, typical temperature has surge by $(2^{\circ}F)$ from the beginning of 1970s, alongside the predominant notable boost arising during the cold and snow season, coinciding with shellfish harvesting. In the 1975-1977 timeframe, the region has faced a decrease in 4-7 freezing days per year (Fernández et al., 2024). Prediction from weather patterns, atmospheric conditions, ecological conditions framework from heating ratios in Lower-eastern are over twice and those observed since the 1975-1977 timeframe, with a standard degree of warmth projected to elevation by 4.5 to 9°F by 2080s, based on discharge circumstances (Karl, 2009). Worldwide as well as domestic rise and distribution of Vibrio is believed to be linked to changes in climate and or novel ocean patterns that bring toastier aquatic bodies into cooler areas as well as modify the brininess description besides the coastal rivers' vertical or axial direction (Baker-Austin et al., 2013). The motility of *V. vulnificus* together with their relevant genus, towards cold and frigid zones (Arctic, Antarctica regions) has been observed in both hemispheres, with the recent detection in previously unreported areas (Baker-Austin et al., 2010). Expansion regarding to *V. vulnificus* has enhanced the risk of infection in the shellfish consumers especially is Southeastern United States.

V. vulnificus flourishes in brackish environments, usually where salinity varies between 10 and 30 parts per thousand (ppt). Increased salinity can hinder its growth, whereas reduced salinity levels might also restrict its survival. Elevated salinity can boost the pathogenicity of *V. vulnificus*. Studies indicate that increased salinity can enhance the production of virulence factors, resulting in the bacteria becoming more pathogenic when they infiltrate the human body via injuries or through the consumption of tainted seafood (Wong and Liu, 2008).

Diagnosis and Treatment

Patients who have watery diarrhea from raw seafood or wound infections from seawater are suspected of having vibriosis. Symptoms of *V. vulnificus* begin within 48 hours of ingestion or 16 hours of wound exposure, whereas symptoms of *V. parahaemolyticus* appear 12–24 hours after consumption. Severe cases need to be diagnosed and admitted to the hospital very away (Jamil et al., 2023). Treatment options for *V. vulnificus* infections include ceftazidime, doxycycline, or cefotaxime and ciprofloxacin. Surgery, supportive therapy, and aggressive wound care are crucial. death rises dramatically with treatment delays; 24 hours delay increases death from 33 to 53%. In one recent instance, a 61-year-old patient recovered after receiving treatment with meropenem, levofloxacin, CRRT, and several debridement's. Survival depends on early diagnosis, timely antibiotics, and surgical surgery (Ting et al., 2024).

Preventive Measures

Fish-derived zoonotic diseases (ZD) pose health risks, especially with raw seafood. Prevention includes proper handling, cooking (62°C), freezing, and strict regulations and the following measures. Harvesting curfews are a natural control method designed to limit *Vibrio* contamination and growth. By restricting harvesting to cooler morning hours and specific tidal periods, this approach minimizes exposure to conditions that promotes bacterial proliferation. Similarly; we can use the natural harvest methods such as Harvesting cessation, Submersion, Deep water suspension, Depuration etc. Post harvesting techniques or method have developed for treatment of raw or fresh of bacterial load without causing change in appearance, flavour, texture nutritional qualities and these methods includes High pressure processing, irradiation, heating, and chemical processing.

Future Prospective

Increase antimicrobial resistance and climatic changes resulting are the main factors that we are facing to control *V. vulnificus*. To cope with it following aspects will be beneficial in controlling and treating the *V. vulnificus*. Advances in molecular tools, such as advance rapid DNA sequencing, and free availability of bioinformatics resources, empower accurate tracking of the catastrophe, spread, advancement and growth of foodborne pathogens. These strategies deal with swifter, more cost-effective, and enhanced analysis in the comparison of conventional subclassing and serotyping, aiding in identifying the facilitators and agents of infectious and virulent pathogen distribution (Baker-Austin et al., 2018). Additionally, remote sensing risk evaluation aided by satellite, established over the previous two decades, shows promise for anticipating wellness risks and health concerns from bacterial infections and disease causing pathogens like *Vibrio*'s. New drug target sites identification allow use to treat pathogens strains with antimicrobial resistance. In addition, use of bacteriophages as bio-control agent such as *V. vulnificus* bacteriophage SSPoo2 belonging to *siphoviridae* family. And full utilization of AI tools and applications like AlphaFold 2 can be a game changer in field of biomedicine and developing strategies for preventions and controlling the pathogens.

Conclusion

V. vulnificus stand out as bacterial invaders that are infectious and virulent with the elevated death rate among the food-spread bacteria. Transmission routes and life cycle of *V. vulnificus* in this work discern the pinpoint sources of contamination throughout the entire seafood supply chain. The climatic changes, increase in temperature, sea levels, saline water introduction in coastal rivers, tolerance abilities, antimicrobial resistance and healthcare burdens are the risks associated with *V. vulnificus*. Therefore medical advancements and improved models, methods for better understanding and prevention of *V. Vulnificus* is required. For this we can utilize CRISPR Therapeutics, Cell and gene therapies, Noval Antigens to correct mutations, silence harmful genes and introduce new protective ones. GIS modeling, Remote sensing, AI and machine learning algorithms to for identity current and future hotspots, developing temporal data, identify seasonal patterns and long-term trendscan become helpful in making future stretegies to control the spread of disease.

References

- Aagesen, A. M., Phuvasate, S., Su, Y.-C., &Häse, C. C. (2013). Persistence of Vibrio parahaemolyticus in the Pacific oyster, Crassostreagigas, is a multifactorial process involving pili and flagella but not type III secretion systems or phase variation. *Applied and Environmental Microbiology*, 79(10), 3303–3305.https://doi.org/10.1128/aem.oo314-13
- Baker-Austin C, Oliver JD (2018) Vibrio vulnificus: new insights into a deadly opportunistic pathogen. Environ Microbiol, 20(2): 423-430. https://doi.org/10.1111/1462-2920.13955
- Baker-Austin C, Stockley L, Rangdale R, and Martinez-Urtaza J. (2010). Environmental occurrence and clinical impact of *Vibrio* vulnificus and *Vibrio parahaemolyticus*: a European perspective. *Environmental Microbiology Reports*, 2(1), 7–18.
- Baker-Austin, C., Trinanes, J. A., Taylor, N. G., Hartnell, R., Siitonen, A., & Martinez-Urtaza, J. (2013). Emerging Vibrio risk at high latitudes in response to ocean warming. *Nature Climate Change*, *3*(1), 73-77.
- Bross, M.H., Soch, K., Morales, R., and Mitchell, R.B. (2007) Vibrio vulnificus infection: diagnosis and treatment. AmFam Physician, 76, 539–544.
- Candelli M, Sacco Fernandez M, Triunfo C, Piccioni A, Ojetti V, Franceschi F, Pignataro (2025) G. *Vibrio vulnificus*-A Review with a Special Focus on Sepsis. *Microorganisms*, *13*(1), 128. doi: 10.3390/microorganisms13010128. PMID: 39858896; PMCID: PMC11768060.
- Carda-Diguez, M., Silva-Hernndez, F., Hubbard, T., Chao, M., Waldor, M., and Amaro, C. (2018) Comprehensive identification of *Vibrio* vulnificus genes required for growth in human serum. *Virulence*, *9*, 981–993.
- Carrascosa, C., Raheem, D., Ramos, F., Saraiva, A., & Raposo, A. (2021). Microbial biofilms in the food industry–Acomprehensive review. International Journal of Environmental Research and Public Health, 18(04), 2014. https://doi.org/10.3390/ijerph18042014
- Centers for Disease Control and Prevention (CDC) (2005) Preliminary FoodNet data on the incidence of infection with pathogens transmitted commonly through food-10 states, United States, 2005. MMWR Morbidity Mortality Weekly Report, 55, 392–395.
- Chernyavskikh, S.D., Borodaeva, Z., Borisovskiy, I.P., Ostapenko, S.I., and Galtseva, O.A. (2019) Blood protein spectrum in representatives of the fish superclass. *Eurasian Journal of BioSciences*, 13, 979–981.
- Coerdt, K. M., & Khachemoune, A. (2021). Vibrio vulnificus: review of mild to life-threatening skin infections. Cutis, 107(2), E12-E17.
- Dechet, A. M., Yu, P. A., Koram, N., & Painter, J. (2008). Nonfoodborne Vibrio infections: an important cause of morbidity and mortality in the United States, 1997–2006. *Clinical Infectious Diseases*, *46*(7), 970-976.
- Deeb, R., Tufford, D., Scott, G. I., Moore, J. G., & Dow, K. (2018). Impact of climate change on Vibrio vulnificus abundance and exposure risk. *Estuaries and Coasts*, *41*, 2289-2303.
- Destoumieux-Garzón, D., Canesi, L., Oyanedel, D., Travers, M. A., Charrière, G. M., Pruzzo, C., & Vezzulli, L. (2020). Vibrio-bivalve interactions in health and disease. *Environmental Microbiology*, 22(10), 4323-4341.
- Elgaml, A., and Miyoshi, S.I. (2017) Regulation systems of protease and hemolysin production in Vibrio vulnificus. MicrobiolImmunol, 61, 1-11.
- Fernández-Juárez V, Riedinger DJ, Gusmao JB, Delgado-Zambrano LF, Coll-García G, Papazachariou V, Herlemann DPR, Pansch C, Andersson AF, Labrenz M, Riemann L. (2024). Temperature, sediment resuspension, and salinity drive the prevalence of *Vibrio vulnificus* in the coastal Baltic Sea. *mBio*, 15, e01569-24 https://doi.org/10.1128/mbio.01569-24
- Fouz, B., Llorens, A., Valiente, E., & Amaro, C. (2010). A comparative epizootiologic study of the two fish-pathogenic serovars of Vibrio vulnificus biotype 2. Journal of Fish Diseases, 33(5), 383-390.
- Goo, S. Y., Lee, H. J., Kim, W. H., Han, K. L., Park, D. K., Lee, H. J., & Park, S. J. (2006). Identification of OmpU of Vibrio vulnificus as a fibronectin-binding protein and its role in bacterial pathogenesis. *Infection and Immunity*, 74(10), 5586-5594.
- Heng, S. P., Letchumanan, V., Deng, C. Y., Ab Mutalib, N. S., Khan, T. M., Chuah, L. H., & Lee, L. H. (2017). Vibrio vulnificus: an environmental

and clinical burden. Frontiers in Microbiology, 8, 997.

Hernández-Cabanyero, C., Sanjuán, E., Fouz, B., Pajuelo, D., Vallejos-Vidal, E., Reyes-López, F.E., and Amaro, C. (2020) The effect of the environmental temper ature on the adaptation to host in the zoonotic pathogen *Vibrio vulnificus*. *Frontiers in Microbiology*, *11*, 1–17.

- Horseman, M. A., & Surani, S. (2011). A comprehensive review of *Vibrio vulnificus*: an important cause of severe sepsis and skin and soft-tissue infection. *International Journal of Infectious Diseases*, 15(3), e157-e166.
- Jamil, M., Abdullah, S., Talib, F., Bashir, R., Ghafoor, N., Javed, K., & Ghafoor, A. (2023). Vibrionaceae and fish zoonosis. *Zoonosis, 4*, (pp. 468-480). Unique Scientific Publishers, Faisalabad, Pakistan.
- Jang, K. K., Gil, S. Y., Lim, J. G., & Choi, S. H. (2016). Regulatory characteristics of Vibrio vulnificus gbpA gene encoding a mucin-binding protein essential for pathogenesis. *Journal of Biological Chemistry*, 291(11), 5774-5787.
- Jeong, H. G., & Satchell, K. J. (2012). Additive function of Vibrio vulnificus MARTXVv and VvhA cytolysins promotes rapid growth and epithelial tissue necrosis during intestinal infection. *PLoS Pathogens*, *8*(3), e1002581.
- Jones, J.L., Kinsey, T.P., Johnson, L. W., Porsom R., Friedman, B., Curtis, M., Wesighan, Jones, M. K., & Oliver, J. D. (2009). Vibrio vulnificus: disease and pathogenesis. Infection and Immunity, 77(5), 1723-1733.
- Karl, T. R. (2009). Global climate change impacts in the United States, A State of Knowledge Report. New York, New York, USA.
- Karunasagar, I. (2014). Vibrio Vulnificus. In Y. Motarjemi, G. Moy, and E. Tood (Eds.), Encyclopaedia of Food Safety, 564-569. Elsevier.
- Lee, K. J., Lee, N. Y., Han, Y. S., Kim, J., Lee, K. H., & Park, S. J. (2010). Functional characterization of the IlpA protein of Vibrio vulnificus as an adhesin and its role in bacterial pathogenesis. *Infection and Immunity*, 78(6), 2408-2417.
- McLaughlin JC. 1995. Vibrio. In: JoBaron E, Pfaller MA, Tenover FC, Yolken RH, Murray PR (Eds.), *Manual of clinical microbiology*, *35*, (pp. 465–476). American Society for Microbiology (ASM) Press, Washington, D.C.
- Murciano, C., Lee, C. T., Fernandez-Bravo, A., Hsieh, T. H., Fouz, B., Hor, L. I., & Amaro, C. (2017). MARTX toxin in the zoonotic serovar of Vibrio vulnificus triggers an early cytokine storm in mice. *Frontiers in Cellular and Infection Microbiology*, *7*, 332.
- Ndraha, N., Wong, H. c., & Hsiao, H. I. (2020). Managing the risk of Vibrio parahaemolyticus infections associated with oyster con sumption: A review. *CRFSFS*, *19*(3), 1187–1217.
- Noorian, P., Hoque, M. M., Espinoza-Vergara, G., &McDougald, D. (2023). Environmental reservoirs of pathogenic Vibrio spp. and their role in disease: The list keeps expanding. *Advances in Experimental Medicine and Biology*, *1404*, 99–126.
- Onohuean, H., & Nwodo, U. U. (2025). Global systematic mapping of Vibrio species pathogenicity: A PRISMA-guided cluster-based analysis. *Medicine*, 104(9), e41664.
- Pajuelo, D., Hernández-Cabanyero, C., Sanjuan, E., Lee, C. T., Silva-Hernández, F. X., Hor, L. I., & Amaro, C. (2016). Iron and Fur in the life cycle of the zoonotic pathogen Vibrio vulnificus. *Environmental Microbiology*, 18(11), 4005-4022.
- Paranjpye, R. N., Johnson, A. B., Baxter, A. E., & Strom, M. S. (2007). Role of type IV pilins in persistence of *Vibrio vulnificus* in Crassostreavirginica oysters. *Applied and Environmental Microbiology*, 73(15), 5041–5044.
- Phillips, K. E., & Satchell, K. J. (2017). Vibrio vulnificus: from oyster colonist to human pathogen. PLoS Pathogens, 13(1), e1006053.
- Reverter, M., Tapissier-Bontemps, N., Lecchini, D., Banaigs, B., and Sasal, P. (2018) Biological and ecological roles of external fish mucus: a review. *Fishes*, *3*, 41.
- Roy, P. K., Mizan, M. F. R., Hossain, M. I., Han, N., Nahar, S., Ashrafudoulla, M., Toushik, S. H., Shim, W.-B., Kim, Y.-M., & Ha, S.-D. (2021). Elimination of Vibrio parahaemolyticus biofilms on crab and shrimp surfaces using ultraviolet C irradiation coupled with sodium hypochlorite and slightly acidic electrolyzed water. *Food Control*, *128*, 108179.
- Ting, W. A. N. G., Zehui, L. I. N., Quan, L. I., &Hanhong, Z. H. A. N. G. (2024). Successful treatment of a patient with sepsis caused by infection with *Vibrio vulnificus* following contact with seawater: a case analysis. *China Tropical Medicine*, 24(3).
- Valiente, E., Lee, C. T., Hor, L. I., Fouz, B., & Amaro, C. (2008a). Role of the metalloprotease Vvp and the virulence plasmid pR99 of Vibrio vulnificus serovar E in surface colonization and fish virulence. *Environmental Microbiology*, *10*(2), 328-338.
- Valiente, E., Padrós, F., Lamas, J., Llorens, A., &Amaro, C. (2008b). Microbial and histopathological study of the vibriosis caused by Vibrio vulnificusserovar E in eels: The metalloproteaseVvp is not an essential lesional factor. *Microbial Pathogenesis*, 45(5-6), 386 393. https://doi.org/10.1016/j.micpath.2008.09.001

Wong, H. C., & Liu, S. H. (2008). Characterization of the low-salinity stress in Vibrio vulnificus. Journal of Food Protection, 71(2), 416-419.

Zhang, J. X., Yuan, Y., Hu, Q. H., Jin, D. Z., Bai, Y., Xin, W. W., ... & Wang, J. L. (2023). Identification of potential pathogenic targets and survival strategies of Vibrio vulnificus through population genomics. *Frontiers in Cellular and Infection Microbiology*, *13*, 1254379.