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Review

Microbial biofilms in seafood: A food-hygiene challenge

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ABSTRACT

Seafood forms a part of a healthy diet. However, seafood can be contaminated with foodborne pathogens, resulting in disease outbreaks. Because people consume large amounts of seafood, such disease outbreaks are increasing worldwide. Seafood contamination is largely due to the naturally occurring phenomenon of biofilm formation. The common seafood bacterial pathogens that form biofilms are Vibrio spp., Aeromonas hydrophila, Salmonella spp., and Listeria monocytogenes. As these organisms pose a global health threat, recent research has focused on elucidating methods to eliminate these biofilm-forming bacteria from seafood, thereby improving food hygiene. Therefore, we highlight recent advances in our understanding of the underlying molecular mechanisms of biofilm formation, the factors that regulate biofilm development and the role of quorum sensing and biofilm formation in the virulence of foodborne pathogens. Currently, several novel methods have been successfully developed for controlling biofilms present in seafood. In this review, we also discuss the epidemiology of seafood-related diseases and the novel methods that could be used for future control of biofilm formation in seafood.

1. Introduction

Food safety is a vital public-health concern that connects human health to farming and other areas of food production. Iwamoto et al. (2010) stated that foodborne pathogenicity is a major cause of the worldwide morbidity and hospitalizations that result from consuming various foods, including seafood. Currently, 31 organisms are recognized as foodborne pathogens, and recent statistics released by the United States Centers for Disease Control and Prevention (CDC) indicate that approximately 48 million foodborne illnesses occur annually in the USA alone, resulting in 128,000 hospitalizations and 3000 deaths (CDC, 2012).

Bacterial communities, such as biofilms, play a key role in the environment as approximately 99% of the microorganisms on the planet have been estimated to exist in fixed consortia (Costerton et al., 1987). Moreover, >80% of bacterial habitats have been estimated to comprise of biofilms, which are organized surface-bound communities containing bacteria and other organisms (Percival et al., 2000; Davies, 2003). Biofilm forming pathogens commonly colonize certain types of seafood, such as pacific oysters (Aagesen et al., 2013), crabs (Reguera and Kolter, 2005), shrimp (Norhana et al., 2010) etc. As a result of the concern over the safety of consuming colonized seafood species, the study of biofilm formation in these organisms has attracted increasing attention. In this review, we focus on basic concepts such as biofilm formation and the molecular mechanisms underlying bacterial-biofilm assembly. Moreover, we discuss how biofilms have emerged as a threat to the seafood industry by highlighting several organisms, including *Vibrio* spp., *Aeromonas hydrophila*, *Salmonella* spp., and *Listeria monocytogenes*, that are responsible for causing foodborne infections, and we present some of the novel techniques that can be used to prevent as well as control biofilm formation.

2. Seafood and foodborne pathogens

Eating customs vary worldwide and the types of food that people consume vary across countries. People in numerous countries consume ready-to-eat (RTE) and raw food, including seafood product, and these individuals are most vulnerable to foodborne illness. Seafood includes various species of mollusks, marine mammals, finfish, crustaceans, and fish eggs (Iwamoto et al., 2010). Because seafood is high in nutritional value, containing high-quality proteins, omega-3 fatty acids, essential micronutrients, and certain minerals and vitamins (Håstein et al., 2006; Mahaffey et al., 2008), it is increasingly incorporated as a part of a healthy diet worldwide. Recently, the Food and Agriculture Organization (FAO) estimated the amounts of seafood consumed annually: according to the FAOSTAT Database (faostat.fao.org), the largest

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amount of seafood consumed is in Japan (53.70 kg/person/year), while the consumption in China is 32.80 kg/person/year (FAO, 2014). The FAO declared the USA to be the third-largest seafood consumer in the world (21.70 kg/person/year). An annual report released by the *National Oceanic and Atmospheric Administration* (NOAA) indicated that commercial fishermen in the USA gathered 9.6 billion pounds of seafood in 2012, which was valued at US$ 5.1 billion (NOAA, 2011). Furthermore, the world's fish and seafood consumption has grown exponentially over the past few decades from 137 to 143 million metric tons, which is valued at US$ 208 billion (FAO, 2010). Globally, seafood is a major traded food product that is often shipped over long distances (FAO, 2002). As such, a large amount of seafood is imported by the USA each year (FAO, 2009). Concurrent with the increase in consumption of seafood, the disease burden associated with these products increased in the USA between 1998 and 2008. Meanwhile, general awareness of seafood-related illnesses also increased during this period (*Morbidity and Mortality Weekly Report* (MMWR), 2013). During this 10-year period, seafood-related products, such as finfish, molluscan shellfish, seafood dishes and other seafood products were reported to be responsible for 5603 cases of diseases that included 657 outbreaks of foodborne illnesses in the USA (*Center for Science in the Public Interest* (CSPI), 2013). Globally, *A. hydrophila* has been isolated from aquaculture products, including crabs and fishes (Nielson et al., 2001), and septicemia due to motile *Aeromonas* spp. has been estimated to have caused greater than US$ 12 million in losses to catfish aquaculture facilities in the USA (Hossain et al., 2014).

Although transmission of a variety of pathogens and contaminants has occurred due to seafood consumption, it is the most common factor associated with these infections (Butt et al., 2004). Furthermore, seafood can be contaminated in numerous ways. Seafood-related disease outbreaks are caused by a wide variety of bacteria, viruses, and parasites; these outbreaks are reported worldwide and can range in severity from mild gastroenteritis to life-threatening infections (Iwamoto et al., 2010). While approximately 50% of these infections are caused by viruses, most hospitalizations and deaths have been caused by bacterial agents (Butt et al., 2004). Smoked mussels, salmon, and other fish have been associated with outbreaks of *Listeria* (Thigeel et al., 2014), and *Salmonella* outbreaks have been linked to the consumption of Sushi (seafood product) (Canaven, 2013). Furthermore, there has been an increase in disease outbreaks related to *Vibrio* spp. Meanwhile, as antibiotic resistance has emerged and increased in marine organisms and in humans, interest in unconventional disease-control measures has grown (Brackman et al., 2009). Notably, despite increased awareness and novel methods for controlling growth of foodborne pathogens, outbreaks of foodborne illness have continued to increase worldwide.

3. Epidemiology of seafood-associated diseases

Huss et al. (2000) developed a scheme for categorizing the risk derived from seafood as being “high” or “low.” According to this scheme, mollusks and shellfish, raw and lightly preserved fish products, and mildly heat-processed fish are considered high-risk foods, whereas semi-preserved fish, smoke-dried fish, fresh/frozen fish and crustaceans, and heat-treated (canned) fish are considered low-risk foods. Conversely, dried and heavily salted fish are considered to pose no health risk.

Various types of bacteria, viruses, fungi, and parasites are responsible for seafood-related infections. Globally, several *Vibrio* species were identified as causative agents in foodborne diseases; however, the etiological evidence was not conclusive in the case of a few species. Seafood-borne diseases were primarily caused by *Vibrio cholerae*, *Vibrio parahaemolyticus*, or *Vibrio vulnificus*. In addition to *Vibrio* spp., *Salmonella* spp., *Clostridium perfringens*, *Clostridium botulinum*, *Campylobacter* spp., *Shigella* spp., and *Listeria* spp., have been reported to be responsible for disease outbreaks due to consumption of fish, crustaceans, and mollusks (MMWR, 2013).

Vibriosis outbreaks due to fish consumption vary with environmental changes, temperature effects, and the presence of certain virulence factors (King et al., 2004). In 2006, 96% of oysters, 94% of mussels, and 48% of shrimp consumed globally were harvested from marine habitats (World Fisheries Report; http://www.fao.org/fishery/sofia/en). Of the *Vibrio* spp., *V. parahaemolyticus* is the leading cause of seafood-associated gastroenteritis in the USA and worldwide (U.S. Food and Drug Administration (FDA), 2005). Additionally, the number of patients in China infected with *V. parahaemolyticus* has increased drastically in recent years (Deng et al., 2008; Chao et al., 2009; Liu et al., 2009). Data collected from the Korea Food and Drug Administration (KFDA, 2014) indicate that *V. parahaemolyticus* is one of the most common causes of foodborne illness in Korea, and was responsible for various outbreaks, and that it was estimated to be responsible for between 5 and 33 outbreaks nationwide between 2005 and 2013. Furthermore, in July 2004, 80 guests attending a wedding party at a restaurant in Spain were infected with *V. parahaemolyticus* (Martinez-Urtaza et al., 2005), and another outbreak of *V. parahaemolyticus* was reported in 2006 in which 177 people were infected by eating contaminated oysters that were harvested in Washington and British Columbia (CDC, 2006). Meanwhile, in Louisiana and Mississippi, *V. parahaemolyticus* caused infection in 3 injury-related cases resulting in 2 deaths after Hurricane Katrina in 2005 (CDC, 2005). According to CDC estimates, *V. parahaemolyticus* caused 104 reported infections and 6 hospitalizations in 13 states in the USA during 2013; however, no deaths were reported from these infections (CDC, 2013).

While the prevalence of infections by non-cholera *Vibrio* spp. appears to have increased recently in the USA, non-O1/non-O139 serogroups of *V. cholerae* occasionally cause outbreaks of diarrheal infection in people who consume raw or *undercooked seafood* (Ottaviani et al., 2009) or who are exposed to [unhygienic marine ecosystems (Lukinmaa et al., 2006)]. Additionally, between 1988 and 2006, approximately 900 *V. vulnificus* infections were reported in the coastal states of the USA. Consequently, the Interstate Shellfish Sanitation Conference (ISSC) planned a project to reduce the frequency of *V. vulnificus* cases by 60% through the introduction of new risk-management practices (ISSC, 2011).

Globally, *Salmonella* spp. are recognized as the principal source of seafood-associated outbreaks, and this is especially the case in the USA (CSPI, 2013) and the EU (European Food Safety Authority (EFSA), 2010). Non-typhoid *Salmonella* spp. were estimated to have caused 11% of all foodborne illnesses (1,027,561 cases), 35% of all foodborne illness resulting in hospitalization (19,336 cases), and 28% of the total number of deaths resulting from foodborne illnesses (378 deaths) in the USA in 2011 (CDC, 2014). Recently, contamination of seafood by *Salmonella* spp. has been considered the most critical public-health hazard in the seafood trade. Iwamoto et al. (2010) examined the outbreaks of *Salmonella* spp. infections related to seafood consumption in the USA, including 18 outbreaks that occurred between 1973 and 2006, which resulted in 374 reported illnesses and 28 hospitalizations. Meanwhile, the FDA reported the presence of *Salmonella* spp. in 7.2% of imported (11,312 samples) and 1.3% of domestic (768 samples) seafood products tested between 1990 and 1998 (Wafaa et al., 2011). Furthermore, in a 3-year study (2002–2005), Bouchrif et al. (2009) confirmed the presence of *Salmonella* spp. in 10 out of 526 seafood products analyzed in Morocco. Meanwhile, the FDA confirmed that RTE

seafood products are frequently contaminated by *Salmonella* spp. (Brands et al., 2005; Duran and Marshall, 2005; Heinitz et al., 2000), and a report by the CDC (CDC, 2012) stated that 425 cases, resulting in 55 hospitalizations, of salmonellosis occurred due to consumption of contaminated raw tuna in 28 states in the USA in 2012. In Asian countries, particularly those in tropical regions, there is a high prevalence of *Salmonella* spp. in seafood. Indeed, *Salmonella* serovar Weltevreden and serovar Tennessee were detected in 24.5% of shrimp samples in Vietnam (Phan et al., 2005), and *Salmonella* spp. were estimated to be responsible for destroying 58% of all seafood products through the contamination of naturally harvested shrimp and prawns alone (Allshouse et al., 2004).

Outbreaks of seafood-borne illness have also been associated with *L. monocytogenes* and *Aeromonas* spp. Indeed, listeriosis outbreaks were linked with consumption of mussels and smoked mussels, smoked cod roe, and undercooked fish (Brett et al., 1998; Facinelli et al., 1989). An average of 2.2 outbreaks was reported to the CDC each year between 1998 and 2008 (Cartwright et al., 2013). In Finland, five illnesses were reported due to the consumption of cold smoked salmon contaminated with *Listeria* (Miettinen et al., 1999), and listeriosis outbreaks occurred after contamination of raw ready-to-eat seafood products in Japan (Miya et al., 2010). Meanwhile, in Bangladesh and India, *Aeromonas* spp. were reported to cause epizootic ulcerative syndrome (EUS) resulting in significant damage to seafood products. Seafood species likely become contaminated by *Aeromonas* in marine environments through colonization of the gut (Aberoum and Jooyandeh, 2010). In developing countries, seafood-borne outbreaks, such as diarrhea related to infection with aeromonads, were reported by Ghenghesh et al. (2008). Hänninen et al. (1997) isolated *A. hydrophila* HG 2 (hybridization groups) and HG 3 from frozen shrimp, which were suspected to be the cause of foodborne outbreaks.

4. Biofilms and seafood pathogens

Bacteria frequently live in densely colonized sessile communities known as biofilms. In these sessile communities, bacteria often perform synchronized activities such as biofilm development, secretion of extracellular enzymes, luminescence production, and production of virulence determinants (Fig. 1) (Srivastava and Christopher, 2012). Biofilms play a dominant role in bacterial life on earth (Percival et al., 2000) as well as protects microorganism by anchoring a firm 3D, multicellular, complex, self-gathered structures that contain exopolymeric substances (exopolysaccharides, proteins, and exoDNA) (Costa et al., 2013; Hall-Stoodley et al., 2004) and are formed on biotic or abiotic surfaces. Moreover, biofilms are characterized by the phenotypic and genetic properties of the cells (Fig. 1) (Stewart and Franklin, 2008; Nadell et al., 2013). Bacteria, cyanobacteria, viruses, fungi, protozoa, microalgae, and invertebrate larvae can form hydrated, self-produced matrices in which microbes engage in cell–cell relationships or assemble when attached to solid surfaces (Fig. 1) (Costerton et al., 1999; Angles et al., 2007; Karatan and Watnick, 2009). Biofilms can develop on practically any surface, including animal teeth, the inner and outer surfaces of plants, rock surfaces in rivers, and the surfaces of stagnant pools and pipelines (Schwermer et al., 2008).

Most seafood-borne pathogens form biofilms on seafood, on food-contact surfaces, and in water (Shikongo-Nambabi et al., 2012) (Table 1). When microorganisms form biofilms on foods, they survive for extended periods and exhibit resistance to most antimicrobials (Jahid and Ha, 2012; Srey et al., 2013). Furthermore, recent research revealed that bacteria and viruses present in high concentrations could form biofilms that remain stable for extended periods upon exposure to the environment (Flood and Ashbolt, 2000). When these biofilms are then stimulated by certain environmental and food-related factors, they return to their planktonic state (Fig. 1) (Takahashi et al., 2009).

While seafood comprises an important component of a healthy human diet, consumption of these foods is not always safe. Seafood is contaminated by a variety of bacteria, viruses, and parasites, and contaminated seafood causes foodborne illnesses and outbreaks with a wide variety of clinical syndromes (Iwamoto et al., 2010). Here discuss several organisms that form biofilms in seafood, including *Vibrio* spp., *A. hydrophila*, *Salmonella* spp., and *L. monocytogenes*. Among these pathogens, most of the research has been dealing with *Vibrio* spp. till date which has focused on seafood biofilm formation and cross-contamination of foods.

4.1. Factors affecting biofilm formation

The preference of microbes on solid surfaces rather than in the liquid phase is a universal phenomenon; thus, most of the contamination of food by microbes is related to biofilm formation (Brooks and Flint, 2008). Biofilm growth is often associated with the production of extracellular polysaccharide materials that bind the biofilm colony together, thereby generating a firm attachment to the substrate and protecting the bacteria within the biofilm against potentially hazardous external factors. Disruptive factors, such as surfactants, which are monitored by means of quorum sensing (bacterial cell–cell communication), cause biofilm detachment; this enables the bacteria to spread and colonize other regions of the substrate, thereby resulting in the environmental contamination (Fig. 1). The assembly of such structures also confers increased resistance to disinfectants and cleaning materials to the organisms within the biofilm; thus, biofilms act as a source of contamination that is difficult to eradicate (Van Houdt and

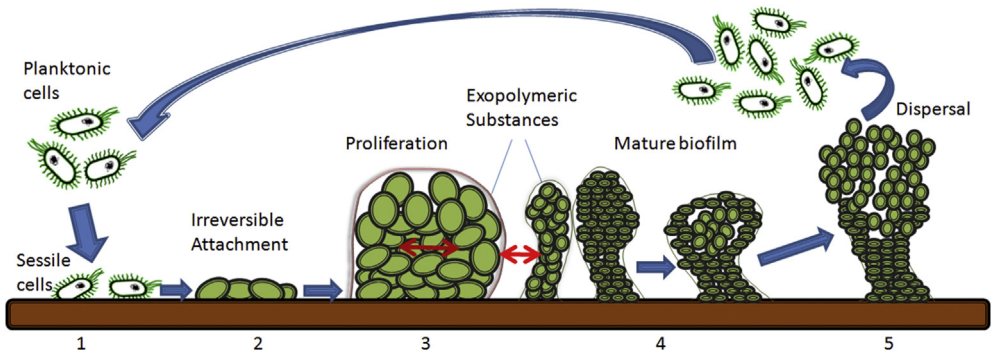

Fig. 1. Schematic representation of biofilm-formation steps. Biofilm formation involves 5 steps: (1) initial attachment, (2) irreversible attachment, (3) micro-colony development by proliferation, (4) biofilm formation, and (5) dispersal.

Table 1
Summary of research on foodborne pathogens and related foods or food-contact surfaces and key findings on seafood biofilms.

Foodborne pathogens	Foods or food-contact surfaces	Key findings	References
V. cholerae	Environmental water	Small aggregates of biofilms formed without any support in water	Faruque et al., 2006
	Continuous-flow cells covered with glass coverslips, and copepod and other zooplanktons	Motility or chemotaxis inhibited colonization	Mueller et al., 2007
	Glass coverslips, glass beads	Quorum sensing regulated biofilm formation	Hammer and Bassler, 2003
	Polyvinyl chloride microtiter plates	*vps* intergenic-region genes encode proteins that produced the biofilm matrix and maintained biofilm structure and stability.	Fong and Yildiz, 2007
	Nutrient-poor "filter-sterilized" lake water (FSLW)	FSLW inhibited cell motility but promoted biofilm Formation	Jubair et al., 2014
V. parahaemolyticus	Pacific oysters	Type I pili, Type IV pili, and both flagellar systems contributed to biofilm formation	Aagesen et al., 2013
	Diatom chitin surface	Type IV pili MshA and PilA promoted adherence to chitin	Frischkorn et al., 2013
	Polystyrene surface	Extracellular polysaccharides, MshA Type IV pili and polar (but not lateral) flagella were required for biofilm assembly	Enos-Berlage et al., 2005
V. vulnificus	Glass tubes, glass coverslips, and polystyrene	Capsular polysaccharides inhibited attachment and biofilm formation	Joseph and Wright, 2004
	Chitin	E-genotype strains attached more effectively to chitin than C-genotype strains did	Williams et al., 2014
	Polystyrene	The smcR (a LuxR homolog) mutant produced a 5-fold increase in biofilm as compared with the wild-type	McDougald et al., 2006
L. monocytogenes	Stainless-steel surface	The biofilm formed was resistant to sanitizing agents (mixture of peroxides)	Pan et al., 2006
	Glass surface	No correlation was detected between hydrophobicity and bacterial attachment	Chae et al., 2006
	Stainless-steel surface	Flagellum-mediated motility was critical for initial surface attachment and biofilm formation	Lemon et al., 2007
	Stainless steel, high-density polyethylene, Bologna and American cheese	Cross-contamination occurred between surfaces and food products through biofilm formation.	Rodríguez and McLandsborough, 2007
Salmonella spp.	Polyvinyl chloride	D-(+)-mannose did not inhibit biofilm formation	Ngwai et al., 2006
	Polystyrene (microtiter plate)	Biofilm formation was significantly correlated with persistence in fish-processing production	Vestby et al., 2009
	Beef, turkey, lettuce broth, and stainless-steel and glass surfaces	Cross-contamination occurred during handling and processing of food and very strong biofilms were formed.	Kim and Wei, 2007
	Gallstone surface	Typhoid-fever pathogenesis was prevented	Prouty et al., 2002
A. hydrophila	Microtiter plate	Glucose regulated virulence and biofilm formation	Jahid et al., 2013
	Microtiter plate	Biofilm cells were more resistant than planktonic cells to common disinfectants	Jahid and Ha, 2014
	Stainless steel	Cells attached and a thin biofilm developed that featured a complex 3D structure and covered 40%–50% of the available surface and produced large micro-colonies	Lynch et al., 2002

Michiels, 2010). The resistance of these 3D structures to physical and chemical stresses likely contributes to the biofilm-mediated cross-contamination associated with the handling and consumption of raw foods. However, the factors that are responsible for the attachment of microorganisms to surfaces have not yet been elucidated.

Biofilm formation is a dynamic process that depends on the characteristics of the growth medium and the bacterial cell surface, and this process can be affected by various environmental stimuli such as temperature and pH (Donlan and Costerton, 2002). Compared to planktonic cells, biofilm cells are more resistant to high temperature and low pH (Castro-Rosas and Escartínb, 2005). Biofilms are also highly resistant to sanitizers (Healy et al., 2009), environmental stresses such as starvation (Spector and Kenyon, 2012), desiccation (reviewed by Gandhi and Chikindas, 2007), disinfectants, dry surfaces, and antimicrobial agents. As such, decontamination of biofilms from food products, such as fresh produce, can be challenging (Jahid and Ha, 2012; Møretrø et al., 2009). Meanwhile, it is widely accepted that nearly all materials that are commonly used in food processing, including buna-n rubber (acrylonitrile butadiene) and stainless steel (Beresford et al., 2001), can support biofilm formation, and Myszka and Czaczyk (2011) reported that eliminating bacterial colonization from food-processing equipment is extremely challenging. To prevent biofilm development, food-processing equipment is therefore designed to feature highly polished and smooth surfaces that hamper bacterial adhesion, the first step in biofilm formation. Thus, a comprehensive understanding of this process is required for ensuring that food-industry products are of high quality and are free from microbial contamination. Biofilm adhesion and maturation can be enhanced by treatment with alcohol as a disinfectant, by the presence of NaCl in food substrates or it contains other bacterial species (Jensen et al., 2007; Gravesen et al., 2005). McDougald et al. (2006) demonstrated that at low and high salinities, *V. vulnificus* exhibited no marked variation in biofilm formation. In contrast, biofilm formation of *V. parahaemolyticus* is highly responsive to environmental factors such as salinity, pH, temperature differences, and the presence of organic matter (Cheng et al., 2004; Kalburge et al., 2014). The lowest temperatures at which *V. parahaemolyticus* has been reported to grow in vitro are 5 °C (Twedt, 1989) and 8.3 °C (Miles et al., 1997), indicating that these organisms can survive refrigeration temperatures. According to the

International Commission on Microbiological Specifications for Foods (ICMSF), *V. parahaemolyticus* can grow rapidly in both broth and on seafood at temperatures ranging from 18 to 40 °C and at a pH range of 7.8–8.6; however, this organism can proliferate at an overall pH range of 4.8–11 (ICMSF, 1996). Meanwhile, relative humidity, osmoadaptation, and food components such as salt and nutrients strongly affect biofilm formation of *L. monocytogenes* on stainless steel, and the transfer of these organisms to salmon products (Hansen and Vogel, 2011). Norhana et al. (2010) reported that both *Listeria* and *Salmonella* biofilm exhibited significantly higher resistance to heat by 1.5–2.0 folds at 60 °C and 1.3–2.6 folds at 70 °C respectively in shrimp surfaces.

According to Valderrama and Cutter (2013) who defined an ecological niche as a set of biotic and abiotic factors required for enduring and maintaining constant populations, biofilm development may serve as an alternative to fitness that helps an organism to boost its endurance, transmission, and spread within a particular ecological niche. This group also extensively reviewed biofilm formation and the environmental factors that affect food-processing conditions, such as temperature, NaCl concentrations, pH, nutrients, and serotypes (Valderrama and Cutter, 2013). All of these factors are closely related to biofilm formation and the cross-contamination of foodborne pathogens.

4.2. Molecular basis of biofilm formation

Biofilms represent a dynamic and complex environment; however, the mechanisms by which biofilms are formed remains unclear. Biofilm development is typically considered to comprise 3 stages: (1) adhesion of microorganisms to a surface; (2) microcolony development and proliferation, and the assembly of a mature biofilm structure; and (3) biofilm dispersal (Monroe, 2007; O'Toole et al., 2000). Recent research has provided molecular insights into all 3 stages (Joo and Otto, 2012). The adhesion process involves complex interactions between microbes and the substrate, and this process is influenced by the surrounding environment (AlAbbas et al., 2012).

Recently, increasing numbers of studies indicate that analysis of bacterial and fungal biofilm formation must involve both molecular biological genetic approaches to identify the genes and regulatory circuits that are required for initial cell-surface communication, biofilm maturation, and the dispersal of biofilm cells into their planktonic state (O'Toole et al., 2000). While our understanding of biofilm formation at the molecular level has been enhanced by genomic and proteomic approaches, such as DNA-microarray or 2D gel-electrophoresis analyses, a common gene- and/or protein-expression pattern in microorganisms that is required for biofilm formation has yet to be elucidated (Ghigo, 2003; Sauer, 2003). However, these new strategies have allowed researchers to analyze the metabolic pathways that contribute to the growth and survival of pathogens in foods, in food-processing environments, and in humans. During maturation, biofilms develop a rigid structure due to cellular production of extracellular polymeric substances (EPS) (Bogino et al., 2013). Genomic-sequence and transposon mutagenesis analyses have enabled the detection of genes that are involved in biofilm extracellular-matrix formation and in the control of biofilm development (Friedman and Kolter, 2004; Simm et al., 2004). Sauer (2003) reported that the protein-expression patterns of cells in biofilms differ from those of planktonic bacteria.

Recent reports have also shed light onto the mechanisms that regulate biofilm formation in seafood-borne pathogens. *V. parahaemolyticus* exhibits swarming motility, which is mediated by the lateral flagellar system (*laf*), and *laf* is induced by the overexpression of the *scrG* gene in the liquid phase and reduced biofilm formation (Kim et al., 2007). Moreover, this organism is capable of forming strong biofilms through the action of capsular polysaccharides (CPS) (Enos-Berlage et al., 2005). Extracellular polysaccharides, mannose-sensitive hemagglutinin (MshA) Type 4 pili, and polar (but not lateral) flagella were determined to be the primary factors that contributed to biofilm formation by *V. parahaemolyticus* (Enos-Berlage et al., 2005). In many Gram-negative bacteria, T3SSs are critical for survival, growth, and virulence (Dean, 2011). However, the requirement of the T3SS during biofilm formation has been challenged by the recent work of Aagesen et al. (2013). This group argued that Type I and Type IV pili as well as the polar and lateral flagellar systems are required for biofilm formation of *V. parahaemolyticus* on oysters, while the T3SS and phase variations are dispensable for this process. In another study, the Type IV pili proteins MshA and PilA, which are required for adherence to chitin, were demonstrated to be essential for the colonization of diatom chitin surfaces by *V. parahaemolyticus* (Frischkorn et al., 2013). Pili are multiprotein surface structures composed of pilin subunits that mediate attachment to abiotic surfaces and the subsequent development of biofilms by *V. cholerae* (Lutz et al., 2013). Type VI pili contribute to the colonization of cultured cell monolayers (Yu et al., 2011).

Two *vps* operons, containing 6 genes (*rbm A–F*) in *V. cholerae*, are also involved in the formation of biofilm cells and the maintenance of biofilm composition and constancy (Fong and Yildiz, 2007; Absalon et al., 2011; Berk et al., 2012). *Vibrio* polysaccharide synthesis (VPS), biofilm matrix assembly, and the actions of Type IV pili and flagella are considered early steps in biofilm formation by *V. cholerae* O1 (Watnick and Kolter, 1999). Lim et al. (2006) determined that the protein capsular-polysaccharide regulator protein (*Vibrio* polysaccharide regulator) is involved in regulating the formation of dimeric cGMP in biofilms of *V. cholerae*. Previous studies addressed the role of the toxin-coregulated pilus (TCP) of *V. cholerae* O1 in biofilm formation on food surfaces, such as on the chitin of shrimp and crabs (Reguera and Kolter, 2005), *and during intestinal colonization of humans* (Krebs and Taylor, 2011). Hung et al. (2006) reported that the presence of bile acids increased biofilm formation and thereby enhanced the survival capacity of *V. cholerae* (Lutz et al., 2013). Elucidating the functions of the genes involved in biofilm formation has enabled researchers to clone wild-type biofilm producers and deletion mutants.

4.3. Quorum sensing in seafood pathogens

Quorum sensing (QS) is a mechanism of bacterial intercellular communication by which bacteria coordinate individual and group behaviors, including biofilm maturation and subsequent dispersal (Liu et al., 2007; Muller et al., 2007). QS depends on the accumulation, release, and synchronized detection of extracellular signal molecules, called autoinducers (AIs), by groups of bacteria existing at high cell densities. By regulating gene expression, AIs drive various processes, including biofilm development, virulence-factor accumulation, antibiotic production, and bioluminescence (Rutherford et al., 2011; Ng and Bassler, 2009; Verma and Miyashiro, 2013). Furthermore, these systems perform a vital role in orchestrating the production of siderophores, the accumulation of virulence factors such as exotoxins, the expression of exoproteases, and the production of various secondary metabolites (Winson et al., 1995; Hentzer et al., 2003). Currently, bacterial biosensors have been used for detecting and identifying quorum-sensing molecules by a multitude of techniques that include thin-layer chromatography, gas chromatography, and liquid chromatography coupled with electrospray ionization, hybrid quadrupole linear ion trap, and Fourier-transform ion-cyclotron-resonance mass spectrometry

(Ravn et al., 2001; Morin et al., 2003; Yang et al., 2005; Steindler and Venturi, 2007; Cataldi et al., 2008).

Vibrio species are ubiquitous in aquatic habitats (Igbinosa and Okoh, 2008). QS, which is widely used and highly conserved in these organisms, mediates diverse physiological activities and controls and influences the virulence system of numerous infectious bacteria (Liu et al., 2013). The model QS regulatory systems that mediate bioluminescence have been characterized in several *Vibrio* species, particularly in *Vibrio fischeri* and *Vibrio harveyi* (Stevens and Greenberg, 1997; Freeman and Bassler, 1999; Camara et al., 2002; Kim et al., 2003). This bioluminescent phenotype is expressed when these bacteria colonize the light organs of squids and certain fish species. In contrast, these organisms repress bioluminescence during planktonic growth in aquatic environments (reviewed by Sitnikov et al., 1995 and Dunlap, 1999). In the aquatic, Gram-negative bacterium *V. fischeri*, QS was first discovered by investigating the mechanisms that control the luminescence induced within growing cultures (Nealson et al., 1970). The *V. fischeri* QS network is drastically different from those present in all other Vibrionaceae members because of higher *qrr* genes that exist in the genomes of the other members and because of the LuxI-R autoinducer (AI) system that acts downstream from the transcriptional activator LitR in *V. fischeri* (Milton, 2006; Miyashiro et al., 2010). Meanwhile, *V. harveyi* controls luminescence, colony morphology, and siderophore assembly using two specific AI-response systems (Bassler et al., 1994), and produces three AIs that are detected by membrane-bound two-component sensors (Ng and Bassler, 2009). *Salmonella typhimurium* exhibited AI-2 production using the *V. harveyi* bioassay (capable of detecting autoinducer production by other species of bacteria) (Surette et al., 1999). AI-2 is produced by the *LuxS* gene, which is responsible for the quorum sensing of *V. harveyi* and is considered a vehicle for internal communication in bacteria (Cloak et al., 2002; Belval et al., 2006).

Quorum sensing-mediated bacterial communication depends on the population density. In a previous study, Bassler (1999) demonstrated that when bacterial population densities increase, AIs accumulate and bind to specific receptors. Based on their unique cellular biology, all pathogens regulate the expression of specific phenotypes that are associated with low and high cell densities. Hammer and Bassler (2003) showed that at a low-cell density (LCD), *V. cholerae* promotes biofilm formation. Conversely, the biofilm genes in *Pseudomonas aeruginosa* are expressed in the high-cell-density (HCD) state. When *V. cholerae* is in the HCD state, the quorum-sensing-response regulator HapR induces the transcription of *hapA,* which encodes a hemagglutinin protease, and the virulence regulator *cytR,* resulting in repression of biofilm development, VPS production, and the expression of toxR (transmembrane regulatory protein) and the flagellum-biosynthesis genes (Zhu et al., 2002; Hammer and Bassler, 2003; Yildiz et al., 2004). Under LCD conditions (when the AI level is zero), luminescence expression is negatively regulated by the phosphorylated response-regulator protein LuxO (Freeman and Bassler, 1999). Lutz et al. (2013) reported that biofilms function as strong physical protectors of the *V. cholerae* under HCD conditions, and that quorum-sensing molecules are produced to regulate the expression of anti-protozoal factors. Through quorum sensing, virulence mechanisms, such as activation of toxins by serine protease, are controlled in shrimp contaminated with *A. hydrophila* (Hänninen et al., 1997). Meanwhile, Gandhi and Chikindas (2007) noted in their review article that while there has been adequate analysis of *L. monocytogenes* biofilm formation in food processing environments, there are no noteworthy studies regarding the quorum sensing system of this organism.

5. Microorganisms involved in biofilm formation on seafood

5.1. Aeromonas hydrophila

Aeromonas spp. are common contaminants of fish and seafood (Hänninen et al., 1997). *A. hydrophila*, which is the most widely recognized *Aeromonas* species, can readily infect fish, amphibians, and humans. These infections can be acquired through open wounds or through the intake of food or marine water contaminated with a sufficient infectious dose (FDA, 2013). Indeed, *A. hydrophila* is commonly isolated from unprocessed and processed seafood products (Aberoum and Jooyandeh, 2010). In various human infections, *A. hydrophila* is considered an etiologic representative of virulence and antibiotic resistance (Grim et al., 2013). Recently, it was found that *A. hydrophila* has been transmitted in the USA as an invasive bacterial species by catfish imported from China (Hossain et al., 2014). As a pathogen, *A. hydrophila* has been commonly isolated from freshly caught seafood such as finfish, prawns, and RTE seafood products bought from wholesale markets and supermarkets (Yaun and Lin, 1993; Thayumanavan et al., 2003; Illanchezian et al., 2010) and from other sources like seawater, wastewater, and freshwater environments (Edberg et al., 2007; Janda and Abbott, 2010). Aberoum and Jooyandeh (2010) suggested that processing facilities of seafood products might act as a source of contamination. Meanwhile, Thayumanavan et al. (2003) reported that along the southwestern coast of India 37% of seafood products tested were contaminated by *Aeromonas*. Tsai and Chen (1996) observed that *A. hydrophila* contaminated 50% of oysters which are purchased from local retail markets of Taiwan. The young, the immunocompromised, and patients with growth problems are highly affected by diseases caused by *Aeromonas* (Sautour et al., 2003). While fish, reptiles, and amphibians act as carriers of *Aeromonas* spp., these bacteria can also cause diseases in these animals (Aberoum and Jooyandeh, 2010).

Lynch et al. (2002) reported that *A. hydrophila* produces a thin biofilm that can cover 40%–50% of the surface of stainless steel. Fig. 2 (i) and (ii) depict the 3D-structures of *A. hydrophila* biofilms on a crab shell and on a polystyrene surface, respectively. Jahid et al. (2013) reported that glucose can regulate biofilm formation and quorum sensing of *A. hydrophila*, and the bacterial cells in biofilms have been suggested to be protected by EPS matrices and stromata that make the cells resistant to cold oxygen plasma (Jahid et al., 2014). Although no seafood product has been directly linked to biofilms of *A. hydrophila*, this organism has been shown to attach to and form biofilms on stainless steel (Lynch et al., 2002), glass (Whiteley et al., 1997), and vegetables (Jahid et al., 2014) in laboratory settings.

5.2. Vibrio parahaemolyticus

V. parahaemolyticus is a halophilic, Gram-negative marine seafood-borne pathogen that is ubiquitous in aquatic environments and is a cause of gastrointestinal and immunological disorders in humans (Blanco-Abad et al., 2009; Xie et al., 2012). Due to its pathogenicity, *V. parahaemolyticus* has attracted considerable attention as a foodborne pathogen. This organism occurs naturally in coastal marine environments, and is the principal cause of gastroenteritis resulting from the consumption of raw, lightly cooked, or mishandled seafood in the USA and in various Asian countries, including Korea, Japan, China, and Taiwan (Robert-Pillot et al., 2010; Altekruse et al., 2000; Iwamoto et al., 2010). In Korean fish markets, various types of seafood, including oysters, are sold from the same tanks containing seawater, which is a primary cause of cross-contamination (Jun et al., 2012). *V. parahaemolyticus* infections are more commonly reported in Asian countries than in

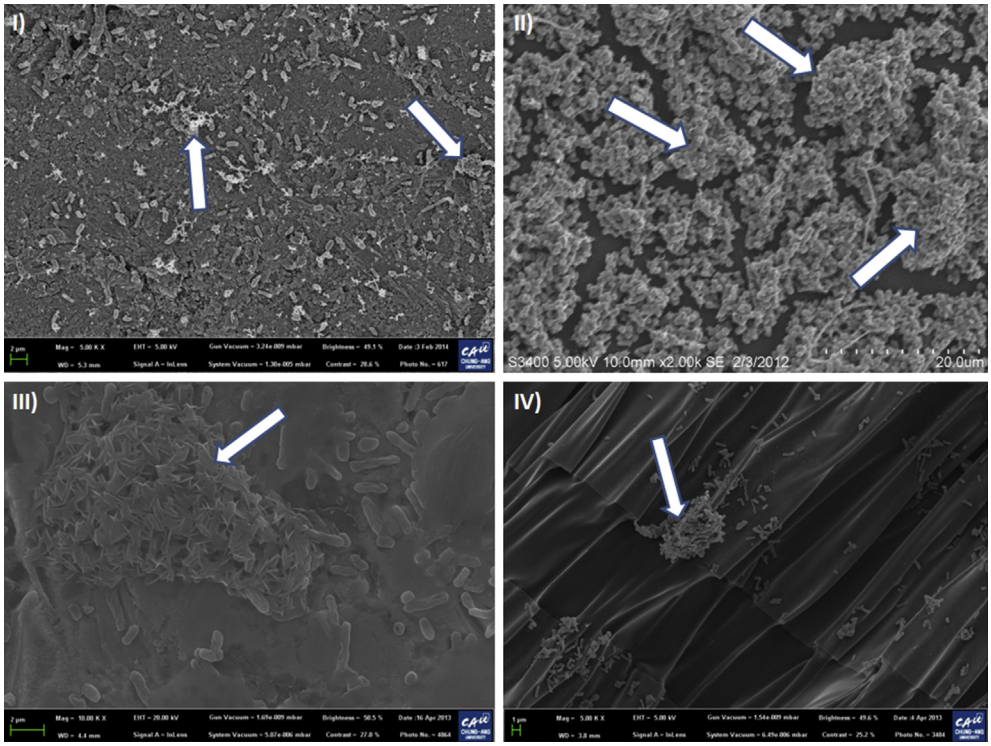

Fig. 2. Field Emission Scanning Electron Microscopy images of (I) *Aeromonas hydrophila* initial cell attachment (white arrows) on a sample of crab shell (5000× Magnification), (II) *A. hydrophila* biofilm formation (white arrows) on a polystyrene surface (2000× Magnification), (III) Detection of a *Salmonella typhimurium* biofilm (white arrow) on stainless steel (10,000× Magnification), (IV) Biofilm formation (white arrow) on polystyrene surface by *Listeria monocytogenes* (5000× Magnification).

European countries (Lin et al., 2013; Xu et al., 2013; Su and Liu, 2007). Among various types of seafood, raw fish and shellfish are the foremost sources of foodborne infections due to this organism (Sakazaki, 2002). Pu et al. (2013) reported that consumption of raw shrimp is the greatest risk factor for *V. parahaemolyticus* infection. Chao et al. (2010) reported that *V. parahaemolyticus* can be spread by both seafood and freshwater products. While the locations that are primarily responsible for cross-contamination and foodborne disease outbreaks are markets, hotels, and restaurants, contamination can also occur through bad manufacturing and poor hygienic practices (Chao et al., 2009; Wang et al., 2014b).

Like other *Vibrio* spp., *V. parahaemolyticus* can form biofilms on various surfaces, including the chitin of diatoms (Frischkorn et al., 2013) and oysters (Aagesen et al., 2013), and on other sources of organic matter (Vezzulli et al., 2008), and this process is recognized as a vital to the physiology of this microorganism (Thompson et al., 2006). *V. parahaemolyticus* is an opportunistic pathogen that causes infections in shrimp during periods of stress (Lightner, 1993). Wang et al. (2013) showed that the DNA-binding regulator AphA is vital for the development of biofilms by pathogenic *V. parahaemolyticus*. *V. parahaemolyticus* inactivation accelerated greatly after acidic electrolyzed water treatment of shrimp at refrigerated temperatures, particularly at 10 °C storage (Wang et al., 2014a).

5.3. *V. cholerae*

Seafood consumption is the largest outbreaks of food-poisoning due to *V. cholerae* (Sumner and Ross, 2002). *V. cholerae* infections are caused by the intake of unhygienic water or food. Seafood products in particular can become contaminated with toxigenic or non-toxigenic *V. cholerae* during manufacturing, handling, and processing (Blackstone et al., 2007). In humans, infections by this organism are characterized by acute diarrhea due to the secretion of cholera toxin (CT), which acts on the mucosal intestinal epithelium. The term CT was coined by Robert Koch in 1884 and was established 75 years later by Dutta et al. (1959). Cholera has been categorized as a threat to public health in developing countries worldwide (Satcher, 1995), and the CDC classified cholera as a Category B bioterrorism agent (WHO, 2008). In 2007, the World Health Organization (WHO) reported 178,677 cases of cholera and 4033 cholera-related deaths worldwide, which resulted in a substantial economic loss (Kirigia et al., 2009). Cholera has also been identified as one of the main causes of diarrhea in Southeast Asia (Siddiqui et al., 2006; Lenglet et al., 2010; Ang et al., 2010), Africa (Kirigia et al., 2009), and, recently, Haiti (Fraser, 2010). In 1992, a *V. cholerae* O139 outbreak was first reported in Bangladesh and India, and subsequently spread throughout the rest of Southeast Asia (Sack et al., 2004). Seafood-related outbreaks of cholera have also been reported in Latin America and the Philippines (Weber et al., 1992).

While *Vibrio* spp. are commonly considered to be heat sensitive (Hackney and Dicharry, 1988), *V. cholerae* was reported to be heat resistant and was detected in hot foods (Makukutu and Guthrie, 1986). Consistent with this report, Liew et al. (1998) demonstrated that mild heat-treatment for up to 25 s is not sufficient to markedly reduce the levels of *V. cholerae* 0139 strains in cockles. The contamination rate of shellfish increases when their harvest conditions combine high temperature and salinity (Ho et al., 2000) and when shellfish are stored, especially in the absence of refrigeration (Kolvin and Roberts, 1982). When harvested seafood contaminated with pathogenic *V. cholerae* is stored in water, this water can act as a reservoir for the organism and contribute to the spread of disease. Recently, handling or consuming contaminated seafood, such as oysters, clams, crabs, or other shellfish, has resulted in infection by non-cholera *Vibrio strains*. Cholera is considered endemic to several countries that export shrimp (Gopal

et al., 2005; Koonse et al., 2005). Seafood-consumption history indicates that travelers contract cholera primarily when they visit places where the disease is endemic. However, acute gastroenteritis in travelers is rarely caused by the *V. cholerae* non-O1 and non-O139 serogroups (Bhattacharya et al., 1992).

Yildiz (2007) showed that *V. cholerae* forms biofilms by attaching to the surface of phytoplankton and chitinaceous zooplankton. Meanwhile, the maturation of *V. cholerae* biofilms in shellfish is mediated by the production of exopolysaccharides (Watnick and Kolter, 1999). Furthermore, Reguera and Kolter (2005) reported that the TCP plays a role in biofilm formation by *V. cholerae* on chitinaceous surfaces and is essential for the differentiation of attached biofilms through bacterial interactions. Conversely, the *V. cholerae* El Tor strain was found to form biofilms on borosilicate in an MshA pilus-dependent but TCP-independent manner (Watnick et al., 1999). *V. cholerae*-chitin interactions have a global impact on biofilm formation (Pruzzo et al., 2008). Donlan and Costerton (2002) suggested that biofilm formation is a major feature of *V. cholerae virulence*. Consistent with this conclusion, Colwell et al. (2003) demonstrated that the infectivity of *V. cholerae* decreased substantially when *V. cholerae* biofilm-like aggregates were removed from the stool of cholera patients.

5.4. V. vulnificus

V. vulnificus is a Gram-negative, halophilic, and mesophilic bacterium that inhabits warm coastal, estuarine, and marine environments worldwide (Ji et al., 2011; Drake et al., 2007; Oliver, 2006). This opportunistic bacterial pathogen is associated with high mortality rates (Chiang and Chuang, 2003), and is the principal cause of seafood-related morbidity and mortality due to the consumption of raw or lightly cooked seafood in USA, Europe, and Asia (Han and Ge, 2010; Jones and Oliver, 2009; Cañigral et al., 2010). Similar to *A. hydrophila*, this organism can infect people through wound exposure or consumption of undercooked seafood (Phippen and Oliver, 2015), and the pathogen has been detected in various types of seafood such as crabs, oysters, shrimp, clams, and fish (Gopal et al., 2005; Warner and Oliver, 2008; Yano et al., 2004). Although *V. vulnificus* rarely causes severe disease in healthy people, wounds are infected by the pathogen during fishing injuries, seafood handling, and recreational swimming (Bisharat et al., 1999). The official statistics issued by the Korean Centers for Disease Control and Prevention (KCDC) indicate that raw fish and shellfish (particularly oysters) are the primary sources of serious foodborne infections caused by *V. vulnificus* in Korea and that these infections resulted in 24 deaths (out of 49 patients) in 2008, 11 deaths (24 patients) in 2009, and 30 deaths (73 patients) in 2010 (KCDC, 2010).

Similar to the other organisms described in this review, pathogenic *V. vulnificus* are biofilm producers (Joseph and Wright, 2004). This bacterium can form biofilms on the surfaces of polystyrene, glass tubes, and glass coverslips (Joseph and Wright, 2004). Notably, however, the expression of CPS inhibits *V. vulnificus* attachment and biofilm formation. Meanwhile, a *V. vulnificus* strain encoding a mutation in *smcR* (a *luxR* homolog) exhibited an approximately 5-fold increase in biofilm colonization on polystyrene compared to the wild-type strain (McDougald et al., 2001, 2006). McDougald et al. (2006) also showed that *V. vulnificus* biofilm formation was controlled by the LuxS/AI-2 system. Furthermore, Kim et al. (2007) reported that the protein NtrC regulates the biosynthesis of lipopolysaccharides (LPS) and EPS, and thereby modulates critical steps in *V. vulnificus* biofilm formation. The *V. vulnificus* extracellular matrix CPS content has been suggested to function as a key mediator of biofilm formation by contributing to colony assembly (Lee et al., 2013). In addition, mixed species of exogenous bacteria were recently reported to be capable of enhancing *V. vulnificus* colonization on oysters (Froelich and Oliver, 2013).

5.5. Salmonella spp

Salmonella spp. continues to be a major global cause of foodborne enteric diseases, accounting for substantial morbidity, mortality, and human suffering. Combating *Salmonella* contamination in farm animals and seafood production is a major challenge (MMWR, 2013). Salmonellosis is typically caused when people drink contaminated water or eat tainted foods such as seafood, poultry, dairy products, beef, pork, and fresh produce (Brands et al., 2005; Heinitz et al., 2000; Kumar et al., 2009). Infections by foodborne *Salmonella* spp. are the result of several environmental factors, including the use of poor sanitary measures, a lack of clean-water supply, and insufficient application of methods for food sanitization and safety. Furthermore, contamination of seafood, such as shrimp, by *Salmonella* spp. and other bacteria can occur, even under high salt conditions and elevated water temperature, due to unhygienic distribution, handling, processing, preparation practices, and retail marketing of seafood products (Zhao et al., 2003; Jay et al., 2003; Bakr et al., 2011; Amagliani et al., 2012). Panisello et al. (2000) also noted that seafood, such as shellfish and other fish, can become tainted with *Salmonella* spp. during storage, handling, and processing or through exposure to contaminated water. *Salmonella* spp. can multiply and survive for long periods in a stressful environment, and also in the ripening period of salted sardines (Arkoudelos et al., 2003; Ristori et al., 2007). *Salmonella* spp. can also survive in acidic environments. For example, *Salmonella* spp. survive in various shrimp food products, including brined shrimp, marinated shrimp, and salads mixed with shrimp (Norhana et al., 2010).

While there is little information on the presence of *Salmonella* in seafood-associated biofilms in marine environments, Vestby et al. (2009) reported a strong correlation between persistence of *Salmonella* spp. in the fish-processing industry and the biofilm-formation ability of these organisms. Stepanović et al. (2003) reported that *Salmonella* spp. feature a complex regulatory network that mediates biofilm formation. On stainless steel, *Salmonella* spp. biofilms are self-gathered and they form flat or mushroom-shaped 3D structures (Fig. 2 (III)). However, the number of biofilm cells and planktonic cells of *Salmonella* spp. formed on sterile glass slides were substantially reduced following exposure to ionizing radiation of 0.531 and 0.591 kGy, respectively (Niemira and Solomon, 2005).

5.6. Listeria monocytogenes

L. monocytogenes is considered a seafood-borne pathogen, and has been isolated from freshwater fish, catfish, crabs (Brackett and Beuchat, 1990; Jallewar et al., 2007). In the USA, *L. monocytogenes* infection has recently become a major public-health concern (Silk et al., 2012). After contamination of food products, this pathogen can multiply for days or months post-production even at refrigeration temperatures (Tompkin, 2002). *L. monocytogenes* has been detected in a variety of seafood products such as smoked seafood, raw RTE seafood, shrimp, molluscan shellfish (Jami et al., 2014). Pao et al. (2008) reported L. monocytogenes 23.5% in catfish, 5.7% in trout, 10.3% in tilapia and 10.6% in salmon purchased from online and local trade markets. González et al. (2013) detected low amounts of *L. monocytogenes* in seafood products at a retail market in Navarra, and high levels of the pathogen in certain brands of smoked salmon. The organism has also been shown to contaminate food processing apparatuses, raw materials, employees, and product trafficking (Takahashi et al., 2009; Gudbjörnsdóttir et al., 2004). Lennon et al. (1984) reported the first outbreak of listeriosis in

association with seafood from New Zealand in 1980. Infection by this pathogen mainly affects immunocompromised individuals, the elderly, newborns, and pregnant women (Painter and Slutsker, 2007). The consumption of smoked salmon contaminated with *L. monocytogenes* is considered to pose a high risk for causing foodborne illnesses (FAO/WHO, 2004). Recently, the reported incidence of listeriosis has also increased in European countries (EFSA, 2012). Although numerous studies have drawn to find out the way of *Listeria* contamination in seafood products, greater attention should be paid to understanding the mechanism and environmental factors affecting and supporting the listerial development and endurance and how seafood becomes linked with this pathogen. *L. monocytogenes* can survive and grow at low temperatures and under other stressful conditions such as low pH and high salinity (Ryser and Marth, 1999; Warriner and Namvar, 2009). Takahashi et al. (2009) suggested that *L. monocytogenes* isolated from RTE seafood can form biofilm. These biofilms are highly resistant to UV light, desiccation, and the sanitizing chemicals that are typically used for sterilization of processing equipment and surfaces, providing *L. monocytogenes* the opportunity to spread to foods (Keskinen et al., 2008; Thompson et al., 2008). *L. monocytogenes* ATCC 19111 was reported to attach efficiently to stainless-steel surfaces in rich medium (Mai and Conner, 2007; Stepanović et al., 2004). Fig. 2 (IV) presents field emission scanning electron microscopy (FESEM) images of *L. monocytogenes* biofilm formation on polystyrene surfaces. In separate study, biofilms formed to varying extents on all tested raw RTE seafood products, indicating that environmental factors strongly affect the ability of *L. monocytogenes* to form biofilms (Takahashi et al., 2009). *L. monocytogenes* isolates obtained from processing plants where mussels (*Perna canaliculus*) are processed exhibited strong, moderate, and low levels of biofilm formation: however, no correlation was noted between biofilm formation and persistence of the isolates (Cruz and Fletcher, 2011). Finally, to ensure public health novel innovative methods must be introduced to control this pathogen in seafood-processing environments.

6. Control of biofilms using novel and alternative methods

To ensure food safety, microbial-control measures are required at every step in the food ecosystem. To control biofilm formation, nutrient and water availability to the microbes must be limited, temperature must be controlled, and well-designed equipment should be fabricated (Chmielewski and Frank, 2003). Removing biofilms that have adapted to environmental stresses by routine cleaning and sanitation methods is extremely challenging. The most obvious and most effective control strategy is still recognized to be the use of sanitation by cleaning with surfactants and use of chemical disinfectants such as quaternary ammonium compounds. Numerous strategies to control biofilm are available, including the use of chitosan, bacteriophages, probiotics, organic acids, plant extracts, bacteriocins, lysozyme, ethylenediaminetetraacetic acid (EDTA), and essential oils such as cinnamon (Hermoso et al., 2007; Sandasi et al., 2010; Helander et al., 2001; Oliveira et al., 2012). Vasconcellos (2004) suggested the employment of Good Manufacturing Processes (GMP) to control bacterial pathogens in seafood. Recent progress in methods for reducing biofilm levels in seafood is summarized in Table 2. Overall, the findings indicate that a single strategy or technology is not available for controlling these seafood pathogens. Preventing biofilm formation is a more judicious approach than treating biofilms. However, no preventive method is currently available that can be used to completely eliminate unwanted biofilms without causing any side effects (Simões et al., 2010). In regards to *A. hydrophila*, Aberoum and Jooyandeh (2010) suggested that cross contamination could be controlled by maintaining distinct locations for the handling of uncooked and processed seafood products.

Certain postharvest treatments, such as ice immersion and depuration, are currently available for use with live shellfish products; however, these treatments were not highly effective in reducing *V. vulnificus* infections (Quevedo et al., 2005). To control *V. vulnificus* infections through postharvest treatment, the standard set by the ISSC is a 5-log reduction, which is equal to a 99.999% reduction in bacterial numbers and an undetectable endpoint concentration of *V. vulnificus* (Cook, 2003). Currently, high-pressure treatment is the commercial decontamination process used for controlling *V. vulnificus* levels in raw oysters, and this process has been used in the seafood industry of the USA to ensure seafood safety for more than a decade (He et al., 2002; Smiddy et al., 2005; WHO/FAO, 2005). Berlin et al. (1999) examined the effects of this treatment on several *Vibrio* spp. strains (*V. cholerae*, *V. parahaemolyticus*, *V. vulnificus*, and *Vibrio hollisae*)

Table 2
A summary of research on the effectiveness of disinfectants in inactivating microorganisms in seafood biofilms.

Treatment	Strains	Seafood	Reduction efficiency	Reference
Ice immersion, depuration	*Vibrio vulnificus*	Live shellfish and manufactured goods, particularly oysters	Weak, not very effective	Quevedo et al., 2005
High pressure		Raw eastern oysters (*Crassostrea virginica*)	>6-log reduction	Berlin et al., 1999 Cook, 2003
$scCO_2$		Oyster	Highly effective reduction	Meujo et al., 2010
Bacteriophage	*Vibrio parahaemolyticus*	Oyster	Substantial reduction	Comeau et al., 2005
High Pressure		Oyster	>5-log reduction	Cook, 2003
$scCO_2$		Oyster	Highly effective reduction	Meujo et al., 2010
Mixed vibriophages	*Vibrio harveyi*	Shrimp	80% reduction	Vinod et al., 2006
$scCO_2$	*Vibrio fischeri*	Oyster	6-log reduction	Meujo et al., 2010
Heat	*Listeria monocytogenes*	Ready-to-eat products	Effective	Huss et al., 2000
Irradiation		Frozen shrimp	4-log reduction	Rashid et al., 1992
High-pressure CO_2		Fresh shrimp (pink shrimp)	2-log reduction	Wei et al., 1991
Heat pasteurization		Cooked and peeled cold-water shrimp (*Pandalus jordani*)	Effective inactivation	Paranjpye et al., 2008
Chlorine dioxide, Ozone		Cooked and peeled cold-water shrimp (*Pandalus jordani*)	Ineffective inactivation	Paranjpye et al., 2008
Irradiation	*Salmonella enteritidis*, *Salmonella* Typhimurium	Fresh and frozen shrimp	Complete elimination at 4.0-kGy radiation	Nerkar and Bandekar, 1989
Lactic acid and cetylpyridinium chloride (CPC)	*Salmonella* Typhimurium	Cooked, peeled ready-to-eat shrimp	Reduction of 0.83-log at 18 °C 1.05-log at 45 °C	Kim, 2007

and obtained >6-log reduction by subjecting the cells to 250 MPa of pressure for 15 min or 300 MPa for 5 min at 25 °C. Calik et al. (2002) attained a 7.5-log reduction in *V. parahaemolyticus* after pressure treatment at 276 MPa for 5 min, and Cook (2003) achieved a >5-log reduction in naturally growing *V. vulnificus* in oysters after treatment at 250 MPa for 120 s. Lakshmanan and Dalgaard (2004) reported that high-pressure treatment was unable to reduce *L. monocytogenes* growth or contamination of chilled cold-smoked salmon. Meujo et al. (2010) noted another problem of high pressure is that it stimulates a release of adductor muscles from oysters' shells which reduces its shelf life. The scCO2 approach stands out in this regard because the release of adductor muscles from oysters can be monitored. Meanwhile, Meujo et al. (2010) utilized supercritical CO_2 ($scCO_2$) treatment to obtain a 6-log reduction in the levels of nonpathogenic *Vibrio* (*V. fischeri*). Moreover, the use of $scCO_2$ in the seafood industry can reduce the load of even the most resilient oyster-associated pathogens, *V. vulnificus* and *V. parahaemolyticus*.

Essential oils are considered as “generally regarded as safe” (GRAS), act as antioxidants and exhibit antibacterial activities both in vitro and in vivo (Viuda-Martos et al., 2010). These oils can be used in food industry for their fragrance, flavor and preservative properties (Burt, 2004; Oussalah et al., 2006; Goudarzi et al., 2011). A few studies have examined the effects of essential oils on bacterial biofilms. Oliveira et al. (2010) reported 100% reduction (5.64 log CFU cm^{-2}) of listerial biofilms on stainless steel surfaces using a combination of essential oils from the *Cymbopogon citratus* (D.C.) Stapf. and *Cymbopogon nardus* (L.) Rendle plants and disinfectant solutions (ethanol and Saline solution with 0.5% (v/v) of Tween 80). Furthermore, treatment with cinnamon essential oil resulted in a 2.54-log reduction in the numbers of sessile listerial cells on a stainless steel surface (Oliveira et al., 2012). Rabiey et al. (2014) found kutum fish reduced the inhibitory effects of *Carum copticum* essential oil on *L. monocytogenes* as its matrix contains fats, proteins etc. In such cases, synergistic effect could be effective to control pathogens.

In addition to mechanical and chemical methods, biological control is currently considered a promising technique of eliminating unwanted biofilms. One biological control method involves the usage of bacteriophages, which are widely recognized as natural predators of microorganisms, but are harmless to mammalian cells (Guenther et al., 2009). These organisms are ubiquitous in nature, including in food ecosystems (Chibani-Chennoufi et al., 2004), and are highly specific to bacterial hosts. The modes by which phages act against food-related bacterial pathogens are summarized elsewhere (Rees and Dodd, 2006). Andreoletti et al. (2009) reported that the food persistence of bacteriophage species varies depending on conditions such as dosage and the physical and chemical factors linked with the food matrix. While phages have been used to control bacterial pathogens since the early 1900s, few reports have been published regarding phage-based control of foodborne pathogens. However, despite enormous advantages of phage application there are still some alarming issues such as (i) release of endotoxins through rapid cell lysis of bacteria (ii) toxins may be produced by some phages. (iii) phage therapy may be failed due to neutralization of phages by the host immune system (Parisien et al., 2008).

Lu and Collins (2007) engineered a bacteriophage expressing a biofilm-degrading enzyme that reduced bacterial biofilms by >99%. Similarly, treatment of shrimp populations in shrimp hatcheries with mixed vibriophages reduced vibriosis by 80%, and resulted in infection and eradication of *V. harveyi* in vitro (Vinod et al., 2006). Furthermore, Comeau et al. (2005) demonstrated that the levels of *V. parahaemolyticus* in oysters were successfully reduced following persistent treatment with phages that exhibit a broad host range, and Guenther et al. (2009) showed that treatment with P100 and A511 *Listeria* phages lessened *L. monocytogenes* counts in smoked salmon and seafood by up to 5 log units. Meanwhile, Soni and Nannapaneni (2010) reported that phage treatment reduced the numbers of *L. monocytogenes* biofilm cells by 3.5–5.4 log CFU cm^{-2} on stainless steel surfaces. In the case of *Salmonella enteritidis*, phage SE2 eliminated greater than 97% of the biofilm-associated bacteria on glass coverslips at 37 °C within 4 h (Tiwari et al., 2013). While these findings are promising, further research is needed to assess the usage of bacteriophages for the control of seafood-borne pathogens and improve seafood safety.

Probiotics have been successfully used to treat mollusks, crustaceans, and finfish aquacultures (Verschuere et al., 2000; Tinh et al., 2008; Balcázar et al., 2006). Probiotic bacteria can inhibit pathogens by disrupting the expression of virulence genes, formation of biofilms, and quorum sensing. Mixed cultures of *Paenibacillus* spp. and *Bacillus* spp. (at 10^4–10^5 CFU/mL) produced antivibrio compounds that inhibited 70%–80% of vibriosis in trout hatcheries (Ravi et al., 2007). Therefore, the use of probiotic organisms that outcompete pathogens or block their virulence-gene expression will likely be most advantageous for ensuring safety of shellfish products. Verschuere et al. (2000) assumed in his review that these biological control agents may be very helpful for infectious diseases outbreak.

Although chitosan is rarely used as a sanitizer in the food industry, it could be used as an antimicrobial agent in association with polymeric food-packaging materials (Fernandez-Saiz et al., 2010; Friedman and Juneja, 2010). Tsai et al. (2002) reported that chitosan has antimicrobial activity in seafood against *A. hydrophila*, *L. monocytogenes*, *S. typhimurium* etc. This compound could be a promising natural, plaque-control agent for eradication of seafood-borne biofilms. Roller and Covill (1999) introduced the main obstacles for using chitosan is poor solubility in water. Lastly, Høiby et al. (2010) suggested that the use of enzymes, such as DNase and alginate lyase, might help to reduce biofilms by dissolving the exopolysaccharide matrix of biofilms and thereby enhancing the susceptibility of biofilm-associate microorganisms to antibiotics. Nonetheless, combination of antibiotics and bacteriophages application can be a valuable approach for seafood biofilm control.

7. Summary

The growing consumption of seafood, the increase in seafood-importing facilities, and the elevated levels of cross-contamination caused by biofilm formation motivated us to write this review. Most types of seafood can be contaminated in the aquatic environment. For example, *Vibrio* spp. thrive in both freshwater and seawater. Another critical concern is that most foodborne pathogens, including *Vibrio* spp., can form biofilms on seafood, particularly on chitin surfaces, through the use of flagella and pili. Biofilms that form on foods are resistant to common disinfectants, but very few studies have investigated how biofilm development can be suppressed using new, alternative techniques. The use of such novel approaches to reduce biofilms could minimize seafood contamination and enhance food safety.

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