Zoonotic Infections: The Role of Biofilms

Ana B. García and Steven L. Percival

Abstract A zoonosis or zoonose is any infectious disease that can be transmitted from non-human animals, both wild and domestic, to humans. Infectious diseases transmitted from humans to non-human animals is sometimes called reverse zoonosis or anthroponosis. Sixty one percent of the pathogens known to affect humans are zoonotic. Biofilm formation is used as a mechanism by zoonotic and environmental pathogens to infect animals and humans. It has been suggested that biofilms are involved in 65–80% of infections treated by doctors in developed countries. Microorganisms can resist extreme temperatures, antibiotic treatments and low levels of nutrients by forming biofilms. Therefore the selection of the right antibiotics to treat human and animal infections caused by biofilms is paramount. It is apparent that more research into biofilm infections in humans and animals, biofilm resistance mechanisms and new strategies for effective treatment need to be developed.

1 Introduction

As mentioned throughout this book, biofilm is a term used to refer to a "vast number of microbial aggregates" as reported by Julian Wimpenny in (2000). However, through the last decade different definitions of biofilms have been reported in the literature as new discoveries in biofilmology are being made. Today biofilms can be defined as a community of microbial organisms which become adherent to each other to form microcolonies. The presence of microcolonies is used as a

A.B. García (✉)
Division of Microbiology and Risk Assessment, National Food Institute, Technical University of Denmark, Mørkhøj Bygade 19, Building H, 2860 Søborg, Denmark
e-mail: agar@food.dtu.dk

S.L. Percival
Department of Pathology, Medical School, West Virginia University, Morgantown, WV, USA
e-mail: biofilms@hotmail.co.uk

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biomarker for the existence of biofilms. Microcolonies are encased in a matrix of extracellular polymeric substances (EPS) and have been identified in the sputum of cystic fibrosis patients (Bjarnsholt et al. 2009), in chronic wounds (Cochrane et al. 2009) and on catheters (Percival et al. 2005).

Biofilm aggregates have also been found in anthropogenic and natural aquatic environments and are known to harbour pathogenic organisms that may be transmitted to humans (Jennings et al. 2003; Knulst et al. 2003). Consequently, the dissemination of a biofilm known to harbour pathogens is considered to be a public health hazard, particularly in hospital environments. In fact, recent findings have suggested that biofilm dissemination is akin to metastasis of a tumour and as such the detached biofilm fragments might cause serious problems when they detach within the circulatory system of both humans and animals (Stoodley et al. 2001). Detached biofilm fragments once in the circulatory system are free to colonise new niches and as such have been linked to conditions such as endocarditis.

From an evolutionary and natural selection process, the ability of a microorganism to form a biofilm is very important since this phenotypic state will aid in and promote a greater flexibility in microbial adaptation (O'Toole and Kolter 1998; Van Loosdrecht et al. 2002; Costerton et al. 2003).

2 Biofilm Formation and Composition

Bacterial components such as flagella, membrane proteins, pili and fimbriae have been shown to have a role to play in attachment to surface and therefore in biofilm formation (Cloete et al. 1989; Prakash et al. 2003; Lelieveld 2005). The presence of flagella and fimbriae is very important in adhesion as they are able to overcome the repulsive forces bacteria encounter when they first attach to a surface (Corpe 1980; Korber et al. 1989; O'Toole and Kolter 1998; Pratt and Kolter 1998; Giron et al. 2002).

Proteomic studies involving *Pseudomonas aeruginosa* biofilms have enabled identification of the stages bacteria go through during adhesion and biofilm formation, a commonality shared by most if not all prokaryotes. The non-specific binding of bacteria to a surface followed then by multiplication of the adherent microorganisms is the first steps to biofilm formation, as discussed in Chap. 2. Following microbial adhesion the microorganisms multiply, produce EPS culminating in the formation of microcolonies. These microcolonies are maintained and supported at the surface by fluid flow and overtime result in the formation of a "mature" biofilm. The architecture of the "mature" biofilm is affected by the abundance, or limited availability, of nutrients and pH. Biofilms are highly heterogeneous and within in vitro models they have been shown to be complex, being composed of features such as stacks, mushrooms, water channels and streamers. Within a natural biofilm a similar architecture to that found in the in vitro biofilm has been hypothesised to occur. However, within these natural biofilms a more diverse microflora composed of fungi, bacteria, algae and protozoa have been reported which have displayed features significantly different to those observed in pure culture models. A biofilm

is able to modulate its internal environment significantly aiming to add stability and protection for the inherent microorganisms present. Whilst the biofilm matrix is in a constant nutritional and biochemical flux a balance between the inherent microbiota of the biofilm is achieved through microbial commensalism, antagonism, mutualism and competition (Lelieveld et al. 2001; Sauer et al. 2002; Prakash et al. 2003; Bartram 2007).

Microbial biofilms which are formed on the surfaces of normal human tissues are purported to offer protection to the host. This phenomenon has been named as the "mucus blanket". It covers the trachea and intestine aiding protection from invasive pathogens (Lambe et al. 1991; Costerton et al. 2003). Biofilms formed by commensal bacteria attached to gut epithelial cells represent a barrier against foodborne pathogens by preventing their attachment (Lee et al. 2000).

Dental plaque on teeth, in both healthy and diseased mouths, is a known biofilm. The microbial composition of these biofilms has been shown to have important implications to health and disease in both animals and humans (Bradshaw et al. 1996; Parsek and Fuqua 2004). In fact, it has been shown that intrauterine infections can be caused by microorganisms initially present in the oral cavity (Fardini et al. 2010). Preterm birth can be caused by infection of the intrauterine environment. Traditionally it has been stated that intrauterine infections predominantly originate from the vaginal tract. However, thanks to technological advances, microbial species that do not belong to the vaginal microflora have been identified in intrauterine infections. Fardini et al. (2010) systemically examined what proportion of the oral microbiome could translocate to the placenta using pregnant mice. Several bacterial species that have been associated with intrauterine infections in humans were identified and the majority of these species were oral commensal organisms. Interestingly, some bacterial species were present with a higher prevalence in the placenta than in oral cavity samples and therefore it was concluded that the placental translocation was species specific.

Many areas of the human body constitute anatomical barriers for preventing the formation of biofilms such as epithelial cells found within the bladder (Uehling 1991). Some organs like the liver need to be maintained in aseptic conditions and as such biofilm development is rare on these organs (Sung et al. 1992).

The formation of biofilms is a dynamic and complex process generating an architecture which aims to protect the inherent microorganisms from chemical or physical removal in some areas and allowing them to colonise new niches (Wimpenny et al. 2000; Allison 2003; Hall-Stoodley et al. 2004; Clutterbuck et al. 2007). Studies have shown that adverse conditions promote biofilm formation and also dispersal (van der Wende and Characklis 1990; Stoodley et al. 2002). It has been suggested that biofilm formation might select in favour of virulent microbial strains (Declerck 2010). It is well documented that some bacteria might help other microorganisms to resist adverse conditions. For example, in biofilms composed of *Pseudomonas fluorescens* their presence has been shown to increase the resistance of *Salmonella typhimurium* to chlorine (Leriche and Carpentier 1995). In addition to this, *Staphylococcus aureus*, a common causative agent of community-acquired and hospital-acquired infections, has been shown to be able to survive and colonise a

range of environments due to its metabolic versatility and potential to form a recalcitrant biofilm (O'Neill et al. 2007).

Genetic and environmental factors influence biofilm formation. The genes expressed in bacteria present in biofilms are reported to differ significantly from the genes expressed in the same bacteria in free-living form. In fact, expression of particular genes such as those that regulate flagella, surface-adhesion proteins and the formation of the extracellular matrix seems essential for biofilm formation compared to planktonic phenotypes (Cramton et al. 1999; Whitchurch et al. 2002; Valle et al. 2003; Hall-Stoodley et al. 2004). Advances in molecular technologies such as microarrays and proteomics have been used to investigate gene expression in cells forming biofilms advancing further our understanding of biofilms (Sauer et al. 2002; Tremoulet et al. 2002; Wagner et al. 2003).

3 Exploring Public Health Aspects and Zoonotic Potential of Biofilms

Biofilms have been present since prehistoric times, especially in hydrothermal environments (Hall-Stoodley et al. 2004). They have a role to play in nutrient cycling and from an environmental perspective they can be beneficial in different ways. In addition, biofilms are useful for water treatment, bioremediation and providing colonisation resistance against pathogens on natural mucosal surfaces (Lebeer et al. 2007; McBain 2009). However, they also have adverse effects by causing infections of humans and animals. In fact it has been suggested that biofilms are involved in 65–80% of infections treated by doctors in developed countries (Ghannoum and O'Toole 2004). Zoonotic and environmental pathogens use biofilm formation as a mechanism to infect animals and humans.

Biofilm formation is one of the most important virulence factors for the development of staphylococcal infections. A number of factors are known to induce biofilm formation in staphylococci, including MRSA and MSSA. The presence of glucose is an example, while sodium chloride seems to induce biofilm formation in MSSA isolates. Sodium chloride is known to activate the transcription of *ica* operon and MSSA biofilm formation is dependent on *icaADBC* operon while MRSA biofilm formation is *icaADBC* independent (Fitzpatrick et al. 2005; O'Neill et al. 2007).

The formation of biofilms allows microorganisms to survive hostile environments and to resist conventional treatments. In fact, the formation of biofilms seems to be the preferred method of growth for microbial organisms (Costerton et al. 1999; Watnick and Kolter 2000; Webb et al. 2003; Parsek and Fuqua 2004). By forming biofilms microorganisms can resist the constraints of extreme temperatures, antibiotic treatments and low levels of nutrients (Prakash et al. 2003; Bartram 2007). They are also highly resistant to acid treatments, dehydration exposure to UV light and phagocytosis (Jefferson 2004; Hall-Stoodley and Stoodley 2005). Whilst UV light has been successfully used against pathogenic microorganisms such as *Giardia*

muris, *Bacillus subtilis*, *Cryptosporidium parvum* and *Legionella pneumophila* in experimental conditions, it has shown to be ineffective against biofilms in water systems (Zhang et al. 2006).

Human infections associated with biofilm formation frequently involve microbial colonisation of medical devices (Lynch and Robertson 2008). Microorganisms can form biofilms in hospital water and medical equipment used to treat humans and animals. In fact, biofilm formation has been associated with persistent infections in medical tools such as *Mycobacterium avium* infection in an intravascular catheter (Schelonka et al. 1994). Changes induced by biofilm formation in intravascular catheters are known to result in total obstruction of the catheter which has led to the development of novel anti-biofilm agents (Stickler et al. 1993; Percival et al. 2009). Biofilm infections of medical devices seem more commonly associated with urinary catheters (Darouiche 2001; Donlan and Costerton 2002; Kite et al. 2004). Several pathogens of public health importance such as *E. coli*, *S. aureus*, *Clostridium perfringens* and *Candida* have been isolated from biofilms contaminating medical devices. In particular, *S. aureus* infections tend to be more acute producing an acute immune response and significant tissue damage (Lynch and Robertson 2008). Biofilm infections of *Candida albicans* have been associated with high mortality (Kojic and Darouiche 2004: Ramage et al. 2005). Biofilm-forming *S. aureus* and *S. epidermidis* have been isolated from humans (Krepsky et al. 2003), dialysis medical devices (Chaieb et al. 2005), bovine mastitis (Vasudevan et al. 2003) and food processing environments (Moretro et al. 2003).

Biofilm formation has been suggested as a risk factor for chronic bovine intramammary infections caused by *S. aureus*. In fact, chronic mastitis caused by *S. aureus* can be very difficult to treat due to antibiotic resistance (Taponen et al. 2003; Cucarella et al. 2004; Fox et al. 2005). Furthermore, *S. aureus* could be present in milk and dairy products derived from animals with clinical or subclinical mastitis. Dairy cattle suffering from clinical mastitis should be milked last and/or using separate milking equipment if possible. In fact, animals suffering from mastitis should be treated and their milk should not be used for human or animal consumption. However, subclinical mastitis might not be diagnosed posing a risk to public health; the use of pasteurisation and decontamination technologies has been recommended as part of food safety assurance systems applied to the production of milk and dairy products.

Staphylococcus epidermidis present on human skin and *P. aeruginosa*, an environmental microorganism, can cause serious chronic infections in compromised hosts (Costerton et al. 1995). *S. aureus* and *S. epidermidis* are responsible for medical device-associated biofilm infections. *S. aureus* (like *P. aeruginosa*) can regulate virulence factors via two quorum-sensing (QS) systems (Yarwood et al. 2004). The production of adhesins by Staphlococci has been considered as the best-understood mechanism of biofilm development (McKenney et al. 1998). *S. epidermidis* has been recognised as a nosocomial pathogen causing human and medical device-related biofilm infections. Tormo et al. (2005) demonstrated that SarA represents an important regulatory element for *S. epidermidis* virulence factors including biofilm formation. Genetic expression profiling of a *S. epidermidis* biofilm

proved that this microorganism exhibited a varied range of genes expressed to increase protection from antibiotics and from the host immune system during biofilm infections (Yao et al. 2005). Regulatory genes such as *agr* and *sarA* are responsible for the production of virulence factors by *S. aureus* and for the expression of specific genes during different stages of infection and biofilm formation (Dunman et al. 2001). In fact, genes can be expressed or repressed during biofilm formation and the use of transcriptome analysis identified 84 genes that were repressed and 48 genes that were induced for *S. aureus* biofilm growth (Beenken et al. 2004). Furthermore, mutations affecting sarA can inhibit *S. aureus* biofilm formation while mutations affecting agr seemed to have a neutral or even increasing effect on *S. aureus* biofilm formation (Beenken et al. 2003). Vectors called staphylococcal cassette chromosome (SCC) contain the mecA gene responsible for methicillin resistance that gets integrated into Staphylococci genetic material to produce the MRSA phenotype (Baba et al. 2002; Boyle-Vavra et al. 2005; Jemili-Ben Jomaa et al. 2006). It has been recognised that mecA genes may spread between humans via *S. epidermidis* (Silva et al. 2001) and 40% of healthcare workers may carry the mecA gene on their hands (Klingenberg et al. 2001). Furthermore, the zoonotic potential of the mecA mobile genetic element responsible for methicillin resistance exhibited by some microorganisms such as Staphylococci has been recognised (Epstein et al. 2009) although more consideration should be given to this zoonotic potential (Guardabassi et al. 2004; Morris et al. 2006; Vengust et al. 2006). In fact, the types of SSCmec found in pets are similar to those found in humans (Malik et al. 2006). MRSA can be embedded in biofilms conferring antimicrobial resistance properties. Furthermore, MRSA may form part of biofilm infections in humans.

Biofilms are also responsible for chronic infections in the urinary tract. Management of urinary infections and the use of a combination of antibiotics (fluoroquinolone and macrolide or fluoroquinolone and fosfomycin) should be considered to treat biofilm infections in the urinary tract (Kumon 2000). Raad et al. (2007) investigated the efficacy of different antibiotics against MRSA present in biofilms. These authors concluded that newer antibiotics such as daptomycin, minocycline and tigecycline should be used in combination with rifampin for antibiotic treatment against MRSA infections. The incidence of MRSA infections is increasing and the emergence of human MRSA infections in hospitals represents a major public health concern (Panlilio et al. 1992; O'Neill et al. 2007). In fact, cross-infection of MRSA between animals and humans has been recognised (Baptiste et al. 2005; Witte et al. 2007) indicating the possibility that mecA genes could spread across species (Boost et al. 2007).

Staphylococcus intermedius has been isolated from pigeons, dogs, dog bites-wound sites (Talan et al. 1989; Futagawa-Saito et al. 2006), from human patients after invasive procedures (Vandenesch et al. 1995) and a human patient with otitis externa (Tanner et al. 2000). *S. intermedius* produces many virulence factors such as coagulase, clumping factor, leukotoxin, enterotoxins and biofilm formation (Raus and Love 1983; Futagawa-Saito et al. 2006). The production of extracellular proteases has been recognised as an important virulence factor in *S. aureus* and

S. intermedius (Karlsson and Arvidson 2002). Meticillin-resistant *S. intermedius* (MRSI) is emerging as a pathogen of concern due to mecA gene acquisitions (Bannoehr et al. 2007). In fact, the zoonotic potential of MRSI and biofilm-forming *S. intermedius* from dogs to humans has also been recognised (Tanner et al. 2000; Futagawa-Saito et al. 2006; van Duijkeren et al. 2008).

Animals might serve as reservoirs for pathogens causing periodontal disease in humans. *Porphyromonas gingivalis* and *Tannerella forsythia* are highly prevalent in humans with periodontitis. Interestingly, *Porphyromonas* and *Tannerella* spp. can be present in the oral cavity of cats. A recent study conducted by Booij-Vrieling et al. (2010) determined the presence and prevalence of *Porphyromonas gulae, P. gingivalis* and *Tannerella forsythia*, and in the oral microflora of cats and their owners by culture and polymerase chain reaction (PCR). This study suggested that the isolates the owners were *P. ingivali* and those isolated from cats were *P. gulae*. However, in one cat/owner couple the *T. forsythia* isolates were identical. Therefore, it was hypothesised that transmission of *T. forsythia* from cats to owners is possible and that cats may be a reservoir for this pathogen (Booij-Vrieling et al. 2010).

Periodontal disease in humans has been associated with chronic obstructive pulmonary disease (COPD). Leuckfeld et al. (2010) found high amounts of Veillonella in the subgingival microflora of COPD subjects. These authors identified subgingival Veillonella isolates by phenotypic and genetic methods in order to assess if Veillonella strain properties correlated with periodontal disease or COPD. The majority of the subgingival Veillonella isolates examined were identified as *Veillonella parvula*. Furthermore, a subgingival and pulmonary isolate obtained from one COPD subject was found to be genetically identical strains of *V. parvula*. Small cocci co-aggregating with larger cocci on the airway epithelium of this patient were observed by electron microscopy. However, no correlation between the subgingival Veillonella strains and the presence of periodontitis or COPD was found. These authors concluded that subgingival *V. parvula* can translocate to the lungs but no particular Veillonella genotype could be associated with periodontal disease or COPD.

L. pneumophila is considered responsible for 90% of human legionellosis cases causing pneumonic and non-pneumonic disease (Yu et al. 2002). *Legionella* grows at high temperatures and hot springs represent natural habitats for this microorganism (Mashiba et al. 1993). However, *L. pneumophila* can transfer from natural habitats to other environments where it can form biofilms and parasitise protozoans as survival mechanisms (Declerck et al. 2005, 2007; Bartram 2007). *Legionella* forms associations with other microorganisms already forming part of biofilms (Watnick and Kolter 2000). *L. pneumophila* can form algal-bacterial biofilms (Hume and Hann 1984). Furthermore, *L. pneumophila* shows necrotrophic growth as another survival mechanism (Temmerman et al. 2006). The survival of *Legionella* forming biofilms also depends on relationships and interference with other microorganisms. In fact, *P. fluorescens* SSD, known as the best bacteriocin producer, had the ability to inhibit *L. pneumophila* present in biofilms (Guerrieri et al. 2008). *L. pneumophila* can survive, replicate and detach from biofilms using

infected amoebae such as *Naegleria* spp. which possess an additional flagellate stage. Furthermore, infected amoebae can release very small vesicles containing *L. pneumophila* that get transported over considerable distances posing a risk to human health (Berk et al. 1998; Greub and Raoult 2004; Weissenberger et al. 2007). *Legionella* can spread by aerosols and human infection may result from inhalation. In fact, this organism can colonise hot and cold water systems (at temperatures of 20–50°C), humidifiers, whirlpool spas and other water-containing devices. *L. pneumophila* can survive in air-conditioned systems and has caused fatal casualties in hotels and hospitals (Wright et al. 1989). Biofilms including *Legionella* can be found in poorly maintained buildings and water systems representing a risk for public health (WHO 2005).

Burkholderia pseudomallei (also known as *Pseudomonas pseudomallei*) causes melioidosis, a disease affecting humans mainly in Southeast Asia and Australia. This microorganism can be very persistent in the environment (Stone 2007). Humans can be infected through a break in the skin or through the inhalation of aerosolised *B. pseudomallei* cells. *B. pseudomallei* can be transmitted from infected rodents to humans through biofilms contaminating soil and water causing persisting chronic disease. The mean incubation period for acute melioidosis is 9 days (with a range between 1 and 21 days). Human infection can present a wide range of symptoms; however, the bacteria can hide in the body and some patients may remain symptom free for a very long time (Stone 2007). However, bacteria might detach from biofilms and cause acute infections and bacteraemias. In fact, this seemed to be the cause when hundreds of people stressed by seasonal starvation died of acute melioidosis in northeast Thailand (Vorachit et al. 1995). Glanders is primarily a disease of animals such as horses, mules and donkeys and occasionally cats, dogs and goats and is caused by *Burkholderia mallei*. Glanders disease and melioidosis can cause similar symptoms in humans. These microorganisms are being studied at laboratories worldwide due to their potential use in biological warfare (Wheelis 1998). However, Glanders is rare in humans; it is sporadic and usually an occupational disease affecting people in frequent contact with infected animals such as animal caretakers, abattoir workers, veterinarians and laboratory personnel (Al-Ani and Roberson 2007). In 2000, a human case of Glanders disease was reported in a laboratory worker in the USA (Srinivasan et al. 2001). *B. mallei* is usually sensitive to most common antibiotics (tetracyclines, novobiocin, ciprofloxacin, gentamicin, streptomycin and the sulphonamides) although resistance to chloramphenicol has been reported.

Immune compromised individuals and people with medical devices are more at risk of suffering infections from biofilm formation. However, host defences are usually not effective against biofilms (Khoury et al. 1992). In fact, cells involved in immune defence mechanisms can actually aid in the formation and maintenance of biofilms when emigrating to the injured body area (Walker et al. 2005). *P. aeruginosa* biofilm infection in children suffering from cystic fibrosis has shown to increase mortality rates (Ghannoum and O'Toole 2004).

Some bacteria such as *S. aureus* might even be able to use the immune reaction as a virulence mechanism (Gresham et al. 2000). Staphylococci bacteria growing

in biofilms have been associated with resistant infections in humans (Vuong and Otto 2002). In fact, in vitro experiments have shown that bacteria present in biofilms can be 10–100 times more resistant to treatments in comparison with the same strain free floating bacteria (Amorena et al. 1999; Olson et al. 2002). Observational studies have been employed to investigate the role of biofilms in causing infections by studying biofilms extracted from infected tissues or contaminated materials recovered from patients. However, in many cases of biofilm chronic infections, it was very difficult to culture bacteria or to recover bacteria from biofilms by using traditional microbiological methods. Therefore, diagnosis and treatment of these infections may prove difficult (Costerton et al. 2003; Lynch and Robertson 2008).

4 Biofilms in Veterinary Medicine and Zoonotic Infections

Microorganisms of veterinary and medical importance are frequently found in biofilms. In animals, biofilm infections might be caused by environmental and even commensal microorganisms such as *S. aureus*. *S. aureus* has been reported to be a concern in postoperative wound biofilm infections (Galuppo et al. 1999) and mastitis (Melchior et al. 2006a, b). In some cases, the same microorganisms can be responsible for biofilm infections in animals and humans. Such bacteria have included *Acinetobacter baumannii* which have been reported to be responsible for wound infections in humans, dogs, cats and horses (Boerlin et al. 2001; Tomaras et al. 2003). *A. baumannii* has also been isolated and the cause of catheter-related infection in horses (Vaneechoutte et al. 2000). *Actinobacillus equuli* is a Gram-negative bacterium commensal of equine oral cavity and upper respiratory tract found to be responsible for sleepy foal disease (Rycroft and Garside 2000) and postoperative wound infections in horses resistant to antibiotics (Smith and Ross 2002). *Actinobacillus lignieresii*, *A. equuli* and *Actinobacillus suis* can be present in the oropharyngeal flora of cattle, horses and pigs, respectively, and therefore may cause bite wound infections in humans (Weyant et al. 1996). In fact, *A. equuli* was isolated from a 53-year-old butcher affected with septicaemia and presented at hospital suffering acute septic shock (Ashhurst-Smith et al. 1998).

Aeromonas hydrophila is a Gram-negative organism causing septicaemia and pneumonia in humans, pigs, cattle and horses (Lallier and Higgins 1988; Zong et al. 2002). Pasteurellosis is considered one of the most important zoonosis; *Pasteurella haemolytica* has been involved in human infections (Takeda et al. 2003). Pasteurella can cause infections in animals. Aspiration of *P. haemolytica* biofilms by feedlot cattle results in severe respiratory infections that might be lethal in 2% of the animals (Morck et al. 1987; Morck et al. 1990).

Aspergillosis is a term used to define a range of infections caused by the fungus of the *Aspergillus* spp. that mainly affects mainly birds but also other animal species and man. It is considered a zoonosis although transmission to humans usually occurs through contaminated soil or organic material in the environment.

Aspergillosis infections in humans can have serious consequences especially in inmunocompromised patients (Seidler et al. 2008). Aspergillus biofilms has been reported on cardiac devices resulting in endocarditis in numerous immunocompromised patients (Lynch and Robertson 2008).

Candida spp. has been recovered from humans, animals and the environment (Beran and Steele 1994) and zoonotic concerns have been raised (Marshall 2003). *Candida* spp. have been responsible for hospital-acquired infections and associated with catheter-related infections (Hawser and Islam 1999; Chandra and Ghannoum 2004). *C. albicans*, in particular, has also been associated with biofilm formation in medical devices and high mortality (Kojic and Darouiche 2004). Kuhn et al. (2002) suggested that *C. albicans* produces quantitatively more biofilm than other *Candida* species. *Candida* forms complex three-dimensional biofilms offering optimal spatial construction for the establishment of microniches throughout the biofilm. *Candida* biofilms present multiple resistance mechanisms making the organism resistant to a range of antifungal products (Douglas 2003; Ramage et al. 2005). Candida and Aspergillus are emerging as dangerous pathogens (patient survival rate as low as 50%) although their prevalence of implant infections has been shown to be only around 8% (Anderson and Marchant 2000).

M. avium and *M. intracellulare* (*M. avium-intracellulare* complex, MAIC) are slow-growing, atypical mycobacteria, ubiquitous in the environment. *M. avium* produces granulomatous lesions that are indistinguishable from the tubercular lesions produced by *M. tuberculosis* and *M. bovis* (Greene and Gunn-Moore 1990; Zeiss et al. 1994). *M. avium* and *M. intracellulare* are potentially zoonotic and have been found growing in biofilms in drinking water systems (Falkinham et al. 2001). Therefore, samples should be collected from pipe biofilms when testing water systems. *M. avium* adheres to surfaces, grows at low levels of oxygen and is resistant to heavy metals forming biofilms on metallic surfaces (Falkinham et al. 2004). *Mycobacterium ulcerans* causes necrotising skin lesions in humans, a disease known as Buruli ulcer (BU) considered an emerging disease (Walsh et al. 2009). *M. ulcerans* has been found in biofilms attached to aquatic plants (Marsollier et al. 2002). It has been suggested that insect bites may play a role in transmitting *M. ulcerans* (Marsollier et al. 2005). *Mycobacterium marinum* and *M. ulcerans* sequences for the 16S rRNA gene are highly similar (Portaels et al. 1996). *M. marinum* causes disease in fish usually called "fish tank granuloma" (Walsh et al. 2009). *M. marinum* can infect humans through skin lesions and produce superficial and self-limiting lesions in hands, forearms, elbows and knees (Steitz et al. 1997). Demangel et al. (2009) propose that *M. ulcerans* originated from *M. marinum* by transfer of a virulence plasmid carrying genes for mycolactone production. Recently, some closely related mycolactone-producing mycobacteria have been discovered causing public health concern (Chemlal et al. 2002; Gauthier and Rhodes 2009). *Mycobacterium haemophilum* causes bone, joint, skin and pulmonary infections in immunocompromised adult humans and lymphadenitis in children (Kiehn and White 1994; Samra et al. 1999). *M. haemophilum* infections in animals have been reported including a case of pulmonary infection in a royal python (Hernandez-Divers and Shearer 2002) and zebrafish infections (Kent et al.

2004; Whipps et al. 2007). *M. haemophilum* was isolated from infected fish at a research facility (University of Georgia) where biofilm samples obtained were also positive for *M. haemophilum* (Whipps et al. 2007). The possibility of the biofilm acting as a reservoir for infection was considered. *M. haemophilum* has been isolated from biofilms growing in water distribution systems (Falkinham et al. 2001). Overall, aquatic Mycobacteria can affect many species of fish and represent a potential zoonotic risk to humans. Water and associated biofilms have been recognised as natural habitats for *Mycobacterium* spp. (Pedley et al. 2004). Therefore, the implementation of programmes for the prevention, reduction or elimination of these pathogens (living free or as part of biofilms) in aquatic facilities is paramount. *M. avium paratuberculosis* (MAP) causes Johne's disease in animals including primates; however, its role in Chrones disease in humans is still controversial. The identification of MAP in blood extracted from patients suffering from Chrones disease in a case-control study suggested that MAP has a role in the pathogenesis of this disease although more research seems necessary to clarify its role (Naser et al. 2004). Bull et al. (2003) observed that the identification of MAP in the majority of tested individuals with chronic intestinal inflammation suggested causality and the fact that MAP causes chronic inflammation of the intestine of a broad range of animal species made this organism the ideal suspect for Chrones disease. Animals can be infected with MAP for years without showing clinical symptoms and shed MAP in their milk and in faeces contaminating the environment. MAP may survive in the environment for a very long time and possible amplify within environmental protozoa (Grant et al. 2002; Ayele et al. 2005). In this way, MAP may be transmitted to humans through drinking water or aerosols (Hermon-Taylor et al. 2000; Fazakerley et al. 2001; Percival et al. 2000; Bull et al. 2003). Aerosol droplets can concentrate bacteria and in that way mycobacteria can spread via aerosols (Pickup et al. 1999; Beard et al. 2001; Whittington et al. 2004). *M. avium* subsp. *paratuberculosis* infection in livestock is endemic in areas of Wales; furthermore, this microorganism has been isolated from the river Taff in Wales. Epidemiological data suggested that the inhalation of *M. avium* subsp. *paratuberculosis* from the river Taff might be responsible for the clustering pattern of Crohn's disease observed in Cardiff (Pickup et al. 2005). *M. avium* subspecies has been isolated from environments worldwide (Falkinham 1996); they are known to persist in biofilms in drinking water distribution systems (Falkinham et al. 2001; Torvinen et al. 2007). It has also been suggested that potable hot water systems may contain *M. avium* concentrations greater than expected (du Moulin et al. 1988). In fact, *M. avium* may survive traditional water disinfection treatments and can be more resistant to chlorine when growing in biofilms (Taylor et al. 2000; Steed and Falkinham 2006). MAP ability to form biofilms seems to vary between isolates and under different conditions (Carter et al. 2003; Johansen et al. 2009). MAP has also been found to survive in biofilms in food-producing animals watering systems. Therefore, the control of biofilms on farms should be included in any farm management programmes (Cook et al. 2010).

Commensal and biofilm-forming environmental microorganisms such as *Listeria monocytogenes*, *Campylobacter*, *E. coli*, *Salmonella*, *S. aureus*, *S. epidermidi*,

Pasteurella multocida, *P. haemolytica*, *Streptococcus suis*, *S. agalactiae*, *Actinobacillus pleuropneumoniae* and Mycoplasmas cause more than half of the infections in mildly compromised individuals (Costerton et al. 1999; Donlan 2001; Prakash et al. 2003).

Treatment of biofilm infections can prove difficult. Antibiotics can be effective against planktonic cells released from biofilms but cannot eliminate biofilms (Marrie et al. 1982). This fact could explain recurring symptoms, after cycles of antibiotic treatments, until the sessile bacterial population is removed or eliminated (Costerton et al. 1985). Studies of polymicrobic biofilms revealed symbiotic interactions and enhanced transfer of antimicrobial resistance genes (Hansen et al. 2007; Seidler et al. 2008).

Some of the major zoonotic microorganisms that are known to be avid biofilm formers and therefore of public health importance are presented below.

4.1 Listeria monocytogenes

L. monocytogenes is a Gram-positive pathogen that can cause severe infections among pregnant women and immunocompromised patients (Farber and Peterkin 1991). *L. monocytogenes* can form biofilms on a variety of surfaces, such as stainless steel and rubber (Blackman and Frank 1996; Meyer 2003). Borucki et al. (2003) demonstrated that *L. monocytogenes* are able to form biofilms in static conditions. Rieu et al. (2008) using laser-scanning confocal microscopy (LSCM) compared *L. monocytogenes* biofilms grown under two different environmental conditions (static growth media and flow conditions). They reported that *L. monocytogenes* formed biofilms under flow conditions which appeared to be more organised with rounded microcolonies surrounded by a network of knitted chains compared to static conditions. Furthermore, biofilms formed by *L. monocytogenes* under different conditions depended on different patterns of gene expression (Hefford et al. 2005; Rieu et al. 2008).

4.2 Helicobacter

Helicobacter species have been isolated from the stomachs of several animals such as cats, dogs, pigs, birds, mice, chickens, ferrets and monkeys (Mirkin 2009). *Helicobacter* spp. DNA has been detected in the oral cavity of dogs representing a risk factor for *Helicobacter* spp. infections in humans (Recordati et al. 2007). Vertical faecal-oral transmission of *H. pylori* infection of Mongolian gerbil pups from an infected mother has been demonstrated (Oshio et al. 2009).

Helicobacter spp. cause stomach problems in humans and may also cause other conditions such as liver disease, clotting, heart attacks and certain skin conditions (Meining et al. 1998). A large number of different species of *Helicobacter* have been isolated from animals with transmission likely to human. These have included

H. helmannii (Mention et al. 1999), *H. rappini*, *H. felis*, *H. cinaedi*, *Helicobacter* sp. strain Mainz (Vandamme et al. 2000), *H. fennelliae*, *H. pullorum*, *H. hills*, *H. hepaticus*, *H. billis* and *H. canis* (Ferenci 2000). One of the most researched *Helicobacter* spp. in both animals and humans is *Helicobacter pylori* a bacterium well known to form biofilms both within in vivo and within in vitro conditions.

H. pylori is a Gram-negative microaerobic rod of public health importance because it causes gastric ulcers, gastritis and contributes to the development of gastric cancer (Amieva & El-Omar 2008; Kandulski et al. 2008). In fact, *H. pylori* has been considered as carcinogenic since 1994 and can be found all over the world but seems more prevalent (90% prevalence) in developing countries (van Duynhoven and de Jonge 2001). Human infection by *H. pylori* can occur through multiple pathways.

Cole et al. (2004) observed that the presence of mucin increased the number of planktonic *H. pylori* and suggested that *H. pylori* biofilm formation might be more difficult in the mucus-rich stomach of humans and animals. However, Carron et al. (2006) photographically documented the presence of mature dense *H. pylori* biofilms on samples of human gastric mucosa obtained from patients undergoing esophagogastroduodenoscopies. Animals constitute a reservoir for *H. pylori* and the possibility of transmission through the food chain has been considered. In fact, *H. pylori* might survive in foods in a viable but non-culturable form (VBNC) and therefore difficult to isolate from foods leading to underestimation of its prevalence (Dimola and Caruso 1999; van Duynhoven and de Jonge 2001). Quaglia et al. (2008) have detected *H. pylori* DNA in raw goat, sheep and cow milk by using a Nested Polymerase Chain Reaction (Nested-PCR) assay. In addition these flies have been shown to potentially transmit *H. pylori* mechanically through excreta. It has been suggested therefore that flies might contaminate food with *H. pylori* (Grübel et al. 1997).

H. pylori can survive up to 1 year in fresh water as viable coccoid forms that are non-culturable but represent a public health hazard (Shahamat et al. 1989; Adams et al. 2003; Konishi et al. 2007). It has been demonstrated that the consumption of environmental water or dirty water is a risk factor for human infections with *H. pylori* (Goodman et al. 1996; Ahmed et al. 2007). In fact, *H. pylori* infections in humans seem to be correlated with biofilm formation and access to contaminated water (Percival and Thomas 2009). Biofilms can provide a protective environment for *H. pylori* to survive in water and even to reach concentrations that could cause harm to humans (Gião et al. 2008). They have also been documented to survive with amoeba and as such its transmission could be similar to that of *M. avium*.

4.3 Campylobacter

Campylobacter spp. are Gram negative, microaerophilic but reported to be unable to grow in air and outside an animal or human host (Park 2002). However, *Campylobacter* are very robust and can survive environmental stresses. In fact

Campylobacter sp. are considered to be the main pathogen causing human gastrointestinal infections in developed countries (Kalmokoff et al. 2006; Murphy et al. 2006). The majority of human *Campylobacter* infections are caused by *C. jejuni* and *C. coli*, with *C. jejuni* being the more common species isolated from human cases of campylobacteriosis (Skirrow 1994).

Trachoo et al. (2002) suggested that *C. jejuni* might form VBNC forms within biofilms isolated from chicken houses. As with all microorganisms, biofilm formation protects *Campylobacter* organisms from the environment and therefore enhances their survival. Trachoo and Frank (2002) demonstrated that the effectiveness of sanitizers against *C. jejuni* was decreased by the presence of biofilms. However, *C. jejuni* biofilms are inactivated by chlorine (Dykes et al. 2003).

Campylobacter jejuni can form different types of biofilms when surviving in different environmental conditions. This variability in biofilm formation could partly explain the survival of *Campylobacter* in the environment and the high incidence of *Campylobacter*-related infections (Gaynor et al. 2005; Joshua et al. 2006). *C. jejuni* biofilms have been isolated from water and in fact, *C. jejuni* can form biofilms in a variety of materials used in animal production watering systems (Reeser et al. 2007).

Biofilms with low levels of oxygen will promote the growth of microaerophilic bacteria such as *Campylobacter*. Furthermore, the presence of organic material will also assist on the survival and growth of *Campylobacter* in environmental biofilms (Humphrey et al. 1995; Kusamaningrum et al. 2003).

4.4 Salmonella

Salmonella causes abdominal pain, diarrhoea and fever. Most individuals infected with *Salmonella* recover without treatment and require oral fluid replacement in 4–7 days. However, some patients need to be hospitalised and treated with antibiotics when suffering from persistent diarrhoea and sometimes bacteraemia or septicaemia can develop. Salmonellosis can be fatal in immunocompromised patients (CDC 2008).

Salmonella species are known to colonise the intestines of mammals, birds and reptiles and when shed into the environment they have been reported to survive for long periods in water, soil and foods. Most human infections with *Salmonella* in developed countries are related to the consumption of contaminated foods (Angulo et al. 1999). *Salmonella* serotype *typhimurium* and *Salmonella* serotype *enteritidis* seem to be the most common serotypes involved in human infections (Rodrigue et al. 1990; Rubino et al. 1998; Herikstad et al. 2002; Galanis et al. 2006).

Salmonella enteritidis are known to form biofilms under a number of conditions and on different materials (Austin et al. 1998; Bradshaw and Marsh 1999). Interestingly *Salmonella* have been reported to form biofilms on gallstones which is known to enhance their proliferation in these organs. The use of antibiotics has been shown not to eradicate *Salmonella* from infected gallstones (Lai et al. 1992) and

surgery is considered an unfeasible solution for individuals with *Salmonella* infected gallstones in developing countries (Crawford et al. 2008).

It has been demonstrated that *S. enterica* serovar typhimurium can compete and outgrow even displace *E. coli* when forming biofilms on HEp-2 epithelial cells (Esteves et al. 2005). Research directed to the study of "biofilm genes" has been conducted with a number of potentially pathogenic Gram-negative bacteria including *Salmonella* (Solano et al. 2002), *E. coli* (Pratt and Kolter 1998) and *Vibrio cholera* (Watnick and Kolter 1999).

4.5 Shigella

Shigella is a Gram-negative, rod-shaped bacteria usually transmitted via faecal contamination through humans, food, water and flies and therefore associated with poor hygienic conditions (Troller 1993; ICMSF 1996). Poor staff hygiene in food processing establishments may lead to food contamination. Gunduz and Tuncel (2006) have isolated *Shigella* from a feeding unit and an aging tank in an ice-cream processing plant which were shown to pose a risk to public health.

4.6 Giardia *and* Cryptosporidium

Giardia and *Cryptosporidium* are parasitic protozoa causing disease in humans and animals frequently transmitted through contaminated water (Brandonisio 2006; Cheng et al. 2009). Wildlife and livestock can contribute to the maintenance of these parasites in the environment (Paziewska et al. 2007) although in a study conducted by Heitman et al. (2002) genetic differences that may indicate host specificity were discovered by genetic characterisation of *Cryptosporidium* isolates collected from humans, dogs, cats, pigs, steer, calfs and beaver hosts.

Cryptosporidium has been responsible for disease outbreaks related to contaminated water in developed countries. The interaction of *Cryptosporidium* oocysts with biofilms present in water distribution systems has been reported but further investigation is required for this (Angles et al. 2007). Biofilms can provide a protective environment for the accumulation of *C. parvum* oocysts assisting on the propagation of this pathogen and the contamination of the environment and water systems (Searcy et al. 2006; Wolyniak et al. 2009). Epidemiological studies revealed that water contamination with animal faeces was the main suspected source of an outbreak of *Cryptosporidium* in Lancashire (UK) in 2000. Furthermore, the persistence of *Cryptosporidium* oocysts after switching to another water source indicated the possibility of oocysts being protected by biofilm formation (Howe et al. 2002). Ecological studies, epidemiological data and risk assessment form the basis for the implementation of effective water treatment to protect public health (Szewzyk et al. 2000). *Giardia* are known to attach to the intestinal epithelial

surface forming part of biofilms. Pathogens need to attach to the intestinal lumen and overcome the forces produced by intestinal mucus to remove not properly attached microorganisms (Costerton et al. 2003).

4.7 E. coli

Reisner et al. (2006) conducted an extensive analysis of *E. coli* biofilm formation and reported very different responses to various environmental conditions and great variation between diverse *E. coli* isolates to form biofilms in vitro. In fact, an association between pathogenic *E. coli* strains and increased biofilm formation capabilities was not observed like it has been reported in other microorganisms such as *Enterococcus faecalis* (Mohamed et al. 2004).

4.8 E. coli *0157*

E. coli 0157 infections in humans can be severe in immunocompromised patients and children. Animals are known to act as reservoirs for *E. coli* 0157 contaminating the environment. Humans can become ill through direct contact with carrier animals or contaminated food or water. Investigations into an outbreak of *E. coli 0157* in people visiting a farm in Pennsylvania revealed that 13% of the farmed cattle were carrying *E. coli 0157* and these isolates had the same distinct molecular pattern that was found in isolates from 51 patients tested in the case-control study (Crump et al. 2002). The possibility of *E. coli 0157* requiring another microorganism such as *Pseudomonas aeuroginosa* to assist in its ability to form biofilms has been investigated (Klayman et al. 2009).

4.9 Yersinia

Yersinia can cause illness in humans through the consumption of contaminated raw or undercooked meat, seafood, tofu and contaminated water. *Y. pseudotuberculosis* can cause lymphadenitis and septicaemia and *Y. enterocolitica* causes gastrointestinal syndromes (Naktin and Beavis 1999; Gerald 2009). *Yersinia entercolitica* can form biofilms and its flagella play an important role in biofilm formation. *Y. enterocolitica* and *Y. pseudotuberculosis* are motile but *Y. pestis* is non-motile (Kim et al. 2008).

Yersinia pestis is responsible for plague syndromes in humans. *Y. pestis* infects rodents and is transmitted to humans by fleas. *Y. pestis* produces dense biofilms on the hydrophobic surface of spines inside the proventriculus in the flea's foregut (Jarrett et al. 2004). However, it has been suggested that *Y. pestis* strains that are unable to form biofilms can also cause plague (Eisen et al. 2007).

4.10 Clostridium botulinum

C. botulinum is a Gram-positive, anaerobic, spore-forming rod responsible for botulism. This microorganism produces a potent neurotoxin which causes flaccid paralysis. Different *Clostridium* species are known to produce seven types of toxins (A–G). *Clostridium* produce spores and these are known to be resistant to antimicrobials and aids survival in the environment and in foods (Hirsh and Birbenstein 2004). Foodborne botulism is a severe condition (high mortality rate if not treated properly) caused by ingestion of contaminated foods. The botulinum toxin is only destroyed at high temperatures (80°C for 10 or more minutes) and therefore represents a highly significant public health concern. *C. botulinum* type C forms biofilms and survives in biofilms within grass; this has been suggested to be the cause of equine dysautonomia (Grass Sickness) (Hirsh and Birbenstein 2004).

Clostridium spores have been implicated in food poisoning cases. The hydrophobicity of *Clostridium* spores plays a key role in their adhesion to surfaces, biofilm formation and increased resistance to sterilisation treatments (Wiencek et al. 1990). Hydrophobic interactions have been associated with the adhesion of bacteria to surfaces and biofilm formation (Rosenberg and Kjelleberg 1986).

4.11 Clostridium perfringens

C. perfringens is an ubiquitous bacteria, Gram-positive, anaerobic that can be found in the environment, in animals (Narayan 1982; Songer 1997), as part of the microbiota in human intestine (Carman et al. 2008) and has the ability to form biofilms enabling it to adapt to different environments (Varga et al. 2008). *C. perfringens* biofilms have been shown to provide recalcitrance to antibiotics and may contribute to antibiotic-associated diarrhoea (Asha et al. 2006; Varga et al. 2008). Some strains of *C. perfringens* such as type C, D and E can colonise the guts of mammals and cause enteric infections in livestock (Songer 1997) and can also form biofilms (Varga et al. 2008).

C. perfringens biofilms exhibited a dense extracellular matrix containing carbohydrates and type IV pilin proteins (Varga et al. 2008). These authors observed that biofilm formation increased in absence of glucose or carbohydrates which could represent a survival mechanism for *C. perfringens* in low nutrient or starvation conditions. However, other survival mechanisms have been observed in stressful conditions such as endospore production and enhanced motility (Varga et al. 2004; Mendez et al. 2008).

4.12 Streptococcus *sp.*

Some members of the genus *Streptococcus* are part of the normal microflora in the human body but sometimes they might produce opportunistic infections such as

dental caries. However, exogenous pathogens of the genus Streptococcus can cause a wide range of infections from mild conditions to life-threatening illnesses (Cvitkovitch et al. 2003).

Streptococcus-related infections are considered zoonotic with some *Streptococcus* sp. more commonly associated with human infections than others (Acha and Szyfres 2003). The most frequently detected Streptococci in human infections have included *Streptococcus equi* subsp. zooepidemicus and *S. equi* subsp. *equi* (Krauss et al. 2003). Other Streptococci of human significance have included *S. pyogenes* which are known to produce several illnesses including a toxic shock-like syndrome (Demers et al. 1993). *S. pyogenes* are known to form biofilms which assits in their tolerance to antimicrobial treatments (Baldassarri et al. 2006).

Streptococcus pneumoniae and *Enterococcus* (*Streptococcus*) *faecalis* have been shown to have increased resistance to antibiotics and are known to cause serious problems in immunocompromised and hospitalised people (Appelbaum 1992). Raw milk and eggs are considered a source for *Streptococcus* infections in humans (Gerald 2009).

4.13 Streptococcus suis

S. suis type 2 can be isolated from carrier adult pigs' upper respiratory tract and tonsils and may cause disease in young pigs and humans. *S. suis* is usually isolated from infected pig carcases but it can also be found in the faeces of infected herds (Huang et al. 2005). *S. suis* type 2 has been isolated from human patients (associated to the pig industry) which suggests an occupational zoonosis route. However, a few cases have been detected in humans with no known contact with the swine industry (Zanen and Engel 1975; Clifton-Hadley 1983; Sriskandan and Slater 2006). *S. suis* human infections have been reported in New Zealand, Australia, Argentina, several Asian and European countries and Canada (Lun et al. 2007). In fact, *S. suis* is one of the most common causes of bacterial meningitis in Hong Kong (Hui et al. 2005). The possibility of the emergence of a new more virulent *S. suis* strain has been raised based on epidemiological studies of outbreaks caused by this particular strain in China (Sriskandan and Slater 2006).

S. suis can form biofilms as reported by Grenier et al. (2009). Grenier et al. (2009) characterised a biofilm formed by *S. suis* type 2, isolated from a pig with meningitis, and observed that *S. suis* strain 95–8242 produced a dense biofilm when sucrose, glucose or fructose was used as the primary nutrient source. Within this study *S. suis* 95–8242 was shown to be more resistant to penicillin G and ampicillin when grown as a biofilm compared to its planktonic counterpart.

Fittipaldi et al. (2007) have used selective capture of transcribed sequences (SCOTS) and identified 28 genes preferentially expressed by *S. suis* when interacting with cells of the porcine brain microvascular endothelial cells, some of these genes were considered potential new virulence factors.

4.14 Vibrio

V. cholera can form biofilms on crustacean shells, aquatic organisms and aquatic plants to reach an infective dose. Consequently, these biofilms have been reported to act as reservoir for *V. cholera* in a non-culturable coccoid form (Hall-Stoodley and Stoodley 2005; Alam et al. 2007). In addition to *V. cholera*, *Vibrio parahaemolyticus* has been isolated from seafood and associated with foodborne illness (D'Mello 2003).

4.15 Aeromonas

Aeromonas sp. are Gram-negative and rod shaped. They are motile aquatic bacteria considered important pathogens in reptiles, amphibians and fish. In particular, they are known to be a major problem in fish farming. Fish are thought to act as a reservoir of *A. hydrophila* possibly leading to infection in mammals (Lallier and Higgins 1988; Lynch et al. 2002).

In humans, *Aeromonas* sp. are known to cause gastroenteritis (from mild to cholera-like symptoms) and other infections such as endocarditis, septicaemia, haemolytic uraemic syndrome, peritonitis, respiratory infections, myonecrosis, osteomyelitis, ocular infections and meningitis. *A. hydrophila* and *E. coli* have been reported to grow well in biofilms detected in drinking water systems (Walker et al. 2000). As well as water *Aeromonas* sp. have also been isolated from foods (seafood, raw milk, meat and vegetables) as they are well known to form biofilms utilising QS mechanisms. By using an N-acylhomoserine lactone (AHL)-dependent QS system (bacterial communication system) the ability of Aeromonas to form biofilm has been shown to be significantly enhanced.

5 Control of Food-Borne and Water-Borne Biofilms of Zoonotic Importance

Biofilms are known to provide a protective environment for pathogenic bacteria, parasites and viruses aiding their dissemination in water systems leading to disease in animals and humans (Howe et al. 2002; Helmi et al. 2008). Microbial cells in biofilms can easily detach voluntary or involuntary from biofilms to aid their dispersal which represents a very important survival strategy (Sauer et al. 2002). Consequently, bacterial cells which reside in the planktonic phase are thought to be in a phase of moving from one surface to another (Parsek and Fuqua 2004). It is plausible to suggest that these dispersal strategies are therefore the cause of food and water contamination and therefore animal and human infection/disease (Zottola and Sasahara 1994; Piriou et al. 1997).

A dynamic equilibrium between attachment and detachment processes occurs in wastewater biofilms which are composed of bacteria, viruses and parasites. Therefore, the microbial concentrations within a biofilm may be regulated purposely to enhance the survival of the biofilm community (Skraber et al. 2007). In fact biofilms have been compared to tissues of higher evolutioned organisms. If pathogenic bacteria detach from biofilms this is potentially dangerous particularly if the infective dose reaches immunocompromised hosts (Storey et al. 2004). Understanding better the particular phases of biofilm formation, proliferation and detachment could prove very useful for investigating the control of biofilms (Ghannoum and O'Toole 2004; Hall-Stoodley et al. 2004; Sawhney and Berry 2009).

As mentioned previously, biofilm formation causes public health problems in food processing and water systems (Lelieveld 2005; Alakomi et al. 2002). However, there are positive applications for biofilms, in particular, they may be used for water treatment (Bryers 2000; Wuertz et al. 2003). However, water-borne pathogens can form part of biofilms and exchange genetic material with other microorganisms present. The use of markers to detect biofilms in water has been suggested (Committee on Indicators for Waterborne Pathogens 2004). The use of chlorine dioxide for biofilm control and general disinfection in water distribution systems has been proposed (O'Leary et al. 2002).

The formation of biofilms has been observed in various food processing industries such as raw and cooked/fermented meats, raw and smoked fish, seafood, chicken and turkey, milk and dairy products and yeast (Sharma and Anand 2002; Bagge-Ravn et al. 2003; Carpentier and Chassaing 2004; O'Brien et al. 2004).

Pathogenic organisms such as *Listeria* and *Shigella* can form biofilms in food processing establishments (Gunduz and Tuncel 2006). These authors isolated a wide range of microorganisms such as *Proteus*, *Enterobacter*, *Shigella*, *Escherichia*, *Edwardsiella*, *Aeromonas*, *Pseudomonas*, *Staphyloccus*, *Bacillus*, *Listeria* and others from an ice-cream processing plant. These authors also observed that the microbiological burden decreased after the cleaning of food producing areas. However, they neglected to remove biofilms and *Shigella* was still present in one of the tanks after cleaning and disinfection, proving this constitutes a significant public health issue. In this same study, *L. monocytogenes* was found on the conveyor belt of the packaging machine indicating the possibility of food contamination. Again despite cleaning in place because of the existence of recalcitrant biofilm the effectiveness of these cleaning agents was clearly proved.

Important public health pathogens such as *S. typhimurium*, *C. jejuni*, *E. coli O157:H7*, *L. monocytogenes* and *Yersinia enterocolitica* have been found in biofilms causing severe problems in the food industry (Griffiths 2003). In fact, some pathogens of zoonotic importance such as *L. monocytogenes* and *S. aureus* are difficult to remove from biofilms (Lelieveld 2005). *L. monocytogenes* has been isolated from dairy and meat processing plants (Wong 1998). *Listeria* has been found to be particularly resistant to disinfectants and temperatures (Wirtanen and Salo 2004). In fact, *Listeria* can survive for up to 10 years in food processing establishments due to the presence of persistent strains with enhanced attachment properties (Lundén et al. 2002). A dramatic and lethal foodborne epidemic caused

by *Listeria* forming biofilms in a food plant and contaminating food products occurred in the USA in 1998. In 2000, USDA required *Listeria* testing in food processing establishments (Drexler 2002). Bjerklie (2003) tested 41 meat processing establishments and found *L. monocytogenes* on or in 39% of the floors tested, 29% of floor drains, 34% of cleaning equipment, 24% of wash areas and 20% of food-contact surfaces. *Listeria* and other pathogens of public health importance forming part of biofilms can survive sanitation procedures in food premises (Arnold 1998). In fact, resistance to chlorine and heat treatments increases when microorganisms such as *Listeria* and *Salmonella* attach to surfaces (Wirtanen and Mattila-Sandholm 1992; Dhir and Dodd 1995). *C. jejuni*, *E. coli O157:H7*, *L. monocytogenes* and *S. typhimurium* forming biofilms were more resistant to trisodium phosphate treatment (Somers et al. 1994). Some sanitizers have been shown to be ineffective against bacteria in biofilms formed during milk processing (Mosteller and Bishop 1993). Biofilms may also form in pasteurisation equipment contaminating pasteurised product (Langeveld et al. 1995).

The presence of biofilms on materials and equipment used in the food industry poses a risk to human health. In fact, biofilms may recontaminate previously treated and/or cooked food products (Lelieveld 2005). Food contamination may result in human foodborne illness and lowers product shelf-life producing economic losses (Criado et al. 1994).

The presence of biofilms in crates used to transport poultry has been documented. The biofilms in crates are difficult to remove by pressure washing, even from well-designed smooth crates (Mead 2005). It seems very important to understand biofilm formation conditions and properties in order to successfully implement sanitation strategies in the poultry industry and protect public health. Poultry production can be an intensive and heavily contaminated operation due to the high number of animals being processed in commercial processing establishments. Furthermore, the use of water at several stages of the process will facilitate the formation of biofilms. Therefore, biofilms can be very prevalent and efforts should be directed to prevent and/or eliminate biofilms in poultry processing. Biofilm control and elimination strategies should be considered as part of HACCP implementation and food safety systems in the food industry. Several factors such as plant and equipment design, maintenance, cleaning and disinfection products and techniques will influence the formation and/or elimination of biofilms in food establishments. Control measures to prevent and eliminate biofilms in the food industry involve adequate design, good manufacturing practices including effective cleanliness of surfaces and proper staff training (Husu et al. 1990; Eklund et al. 1995; Autio et al. 1999; Miettinen et al. 1999, 2001; Lyytikäinen et al. 2000; Wirtanen 2002; Aarnisalo et al. 2003; Lundén 2004; Miettinen and Wirtanen 2006; Wirtanen and Salo 2004).

Some surfaces are preferred by microorganisms to form biofilms. The identification of factors that make different surfaces resistant or susceptible to biofilm formation seems crucial to control biofilms in the food industry. Some types of rubber materials seem to inhibit microbial attachment. Although the presence of some elements such as zinc and sulphur can partially explain this fact, there are other factors associated with rubber that contribute to the inhibitory effect (Arnold

and Silvers 2000). However, proper maintenance of rubber material will also influence the effect on biofilm formation (Arnold and Bailey 2000). The effects of corrosion on metallic surfaces and biofilm formation have also been investigated. Attachment of bacteria to metallic surfaces is facilitated by corrosive treatments (Arnold and Suzuki 2003). Therefore, proper maintenance of surfaces and equipment used in food processing and the use of materials resistant to corrosion seem important measures to reduce and control biofilm formation. Effective sanitation strategies are crucial for the control of biofilms in the food industry. Some materials such as Teflon seem easier to effectively clear of biofilms than others like stainless steel (Marriott 1999). Thorough cleaning before disinfection of surfaces and equipment is crucial to removed attached microorganisms and for the effectiveness of sanitation procedures in the food industry. The use of mechanical energy has been shown to be most effective against biofilms (Gibson et al. 1999). Food producers should use mechanical equipments which do not create aerosols. The use of non-aerosol generating detergents and sanitizers will be more effective when used together with mechanical methods (Meyer 2003). The use of sanitizers such as acidic quaternary ammonia, chlorine dioxide and peracetic acid has been found more effective to remove attached microorganisms (Krysinski et al. 1992). The use of antimicrobial coatings on equipment surfaces, such as bacteriocins and silver ions, will help in controlling the formation of biofilms (Kumar and Anand 1998; Meyer 2003). Special treatments such as the use of specific enzymes might be necessary to eliminate pathogens from biofilms in the food industry (Oulahal-Lagsir et al. 2003). However, some decontamination technologies used in or on foods might potentially provide stressful conditions promoting biofilm formation and increasing pathogens' virulence (Samelis and Sofos 2003). It has also been suggested that the use of chemical sanitizers may exert a selective pressure on attached pathogens selecting for resistant strains (Langsrud et al. 2003).

Furthermore, a steady increase in the prevalence of antibiotic-resistant strains isolated from food processing environments has been observed (Teuber et al. 2003; Nayak et al. 2004) and antibiotic-resistant strains also exhibited cross-resistance to other antimicrobial agents, such as sanitizers (Langsrud et al. 2003; Lundén et al. 2003). This phenomenon could be linked to the transfer of genetic material such as plasmids coding for antibiotic resistance between microorganisms present in biofilms. Biofilms might then represent a reservoir for antimicrobial-resistance genes (Watnick and Kolter 2000; Jefferson 2004; Parsek and Fuqua 2004; Weigel et al. 2007). Human infections with antibiotic-resistant pathogens represent a serious public health problem. *Salmonella enterica* serovars are increasingly resistant to commonly administered antibiotics (Boyle et al. 2007). Recommendations have been made to limit the use of antibiotics in farm animals and to adopt non-antimicrobial farm management strategies (Angulo et al. 1999).

More research seems necessary in the control and prevention of biofilm formation including a balance between benefits and risks of using particular decontamination technologies in order to select optimum treatments and improve food safety. In general, decontamination technologies currently used reduce pathogen prevalence on fresh meat (Sofos 2005).

6 Further Research and Biofilm Control Strategies

The use of new technologies for the study of biofilms can provide interesting and useful information for the control of biofilm infections. Pathogens such as *L. pneumophila*, *C. jejuni* and *C. parvum* have been identified in biofilms by using episcopic differential interference contrast (EDIC) microscopy together with epi-fluorescence using gold nanoparticles or fluorophores (EDIC/EF) revealing 3D biofilm structure (Keevil 2003). LSCM is also a powerful technology to study biofilm architecture and 3D structure and used to study *P. aeruginosa* and *S. aureus* biofilms (Klausen et al. 2003; Jefferson et al. 2005; Pamp and Tolker-Nielsen 2007). However, biofilm structure can be diverse in response to environmental conditions (Wimpenny et al. 2000).

New molecular technologies such as the SCOTS can offer advantages for the study of genetic regulation of biofilm formation (Osaki et al. 2002).

Bioengineering has also been successfully used to produce bacteriophages that can induce the production of biofilm-degrading enzymes during infection to act against the biofilm matrix and cells (Lu and Collins 2007). The use of artificially engineered biofilms has been proposed to capture pathogens present in water to monitor the hygienic status of drinking water (Bauman et al. 2009). Biofilm engineering can contribute to the elimination of biofilms and to the control of biofilm infections. Nanotechnologies such as the use of fluoride nanomaterials might present a solution to inhibit biofilm formation (Lellouche et al. 2009). The use of current electric fields and ultrasonic radiation can render biofilms more susceptible to conventional antibiotic treatments (Costerton et al. 1994; Rediske et al. 1998).

The use of microemulsions and nanoemulsions has also been tested to control biofilms. Teixeira et al. (2007) used emulsions to inactivate biofilms formed by *P. aeruginosa*, *Salmonella* spp., *S. aureus*, *E. coli* 0157:H7 (VT-) and *L. monocytogenes*. All biofilms were inhibited by the used emulsions except the one formed by *L. monocytogenes*. Essential oil compounds have been proved successful against biofilms of *L. monocytogenes* and *E. coli* 0157:H7 representing a potential solution for the treatment of biofilms in the food industry (Pérez-Conesa et al. 2006).

The study of biofilms' structure, architecture, cellular spatial arrangement (Davey and O'Toole 2000) and synergistic and antagonistic or inhibitory interactions between biofilm cells will provide new insights into biofilm infections and the development of effective treatments (Reisner et al. 2006). A synergistic effect was observed in mixed biofilms of *P. aeruginosa* and MRSA involved in catheter-associated urinary tract infections (CAUTI). In fact, *P. aeruginosa* was producing more exotoxin A when forming biofilms with MRSA (Goldsworthy 2008).

Adhesion to surfaces is the first necessary step for the production of biofilms (Klemm and Schembri 2004) and therefore actions taken to prevent adhesion of microorganisms could provide interesting solutions for the prevention of biofilms. Several factors play a role in biofilm formation and bacterial adhesion such as environmental conditions, the capacity of microorganisms to adhere to surfaces and

also the nature of the surface (Katsikogianni and Missirlis 2004). Bacterial attachment to inert surfaces can be reduced by using aqueous extract of fish muscle proteins (FMPs) to precondition the surface (Bernbom et al. 2006). This type of proteinaceous coating from fish muscle was used against biofilm formation by UTI *E. coli* and *Klebsiella* strains on different materials. A 100-fold reduction in biofilm formation was observed although the effect depended also on other variables such as the growth medium, the particular bacterial strain and the extract (Vejborg and Klemm 2008).

Antibiotic resistance is one of the main global public health concerns (Croft et al. 2007). Antibiotic resistance associated with biofilm infections can be explained by several factors associated with biofilm formation such as the different growth phases of cells forming biofilms and the presence of the extracellular matrix (Stewart and Franklin 2008).

Further research into genetic control of MRSA biofilm formation will increase our understanding of the pathogenesis of *MRSA* infections involving biofilms and will be useful for the development of novel therapies to treat persistent infections (O'Neill et al. 2007).

Oritavancin is a semisynthetic lipoglycopeptide with bactericidal properties against Gram-positive microorganisms such as methicillin-resistant *S. aureus* (MRSA), vancomycin-resistant *S. aureus* (VRSA) and others (Belley et al. 2009). Oritavacin can produce loss of membrane integrity in susceptible biofilm cells (McKay et al. 2006) being this its principal antibacterial mechanism against stationary phase *S. aureus* cells (Belley et al. 2009).

The selection of the right antibiotics to treat human and animal infections caused by biofilms is paramount (Clutterbuck et al. 2007). Furthermore, more research into biofilm infections in humans and animals, biofilm resistance mechanisms and new strategies for effective treatment should be developed. New therapies need to overcome significant difficulties encountered when treating biofilm infections (Belley et al. 2009). It has been suggested that prophylactic use of microbicides in medical devices can be effective against biofilm formation and associated infections.

The use of antibiotics as part of any prophylactic strategy is controversial but they are routinely used in immunocompromised patients. It has been suggested that the most effective treatment of biofilm infections associated with medical devices combines removal of an infected material and systemic antibiotic and/or antifungal therapy. However, fungal infections can be very difficult to treat. New experimental therapies to prevent and/or treat biofilm-related infections of medical devices are being developed such as new materials and intelligent implants. Future research into biofilm-related infections and the use of new imaging technologies and biomarkers will assist on the fight against biofilm infections (Lynch and Robertson 2008). Compounds that can interfere with QS systems or cellular communication systems represent an important alternative for the treatment of biofilm infections. One of the main advantages is that these products are very unlikely to create selective pressure for more virulent or resistant microbial strains (Morten et al. 2003).

7 The Future

Future research should include genetic investigations into regulatory genes involved in biofilm formation and survival mechanisms, bioengineering and new technologies for the study of biofilms. Biofilm control strategies should be based on risk assessment and assessment of the effectiveness of control methods. Furthermore, new treatments and therapies should be developed and their effectiveness on the fight against biofilms adequately assessed.

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