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Review

Surface adhesins and exopolymers of selected foodborne pathogens

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The ability of bacteria to bind different compounds and to adhere to biotic and abiotic surfaces provides them with a range of advantages, such as colonization of various tissues, internalization, avoidance of an immune response, and survival and persistence in the environment. A variety of bacterial surface structures are involved in this process and these promote bacterial adhesion in a more or less specific manner. In this review, we will focus on those surface adhesins and exopolymers in selected foodborne pathogens that are involved mainly in primary adhesion. Their role in biofilm development will also be considered when appropriate. Both the clinical impact and the implications for food safety of such adhesion will be discussed.

Introduction

Foodborne diseases represent a global threat to human health. The vast majority of foodborne diseases are associated with pathogenic micro-organisms and/or their toxins, whereas other causes such as parasites, chemicals and toxins naturally present in some foods have been reported only sporadically. Besides health disorders that may vary from medium-risk to fatal, foodborne diseases can lead to very high economic losses, such as those related to medical treatments, lost wages and productivity, or recall and destruction of food products (Ray & Bhunia, 2007).

Microbiological safety of food is closely related to the quality of raw materials and hygienic practices on farms and in food-processing plants (Verran *et al.*, 2008). However, the ability of micro-organisms to persist in food-processing environments plays a crucial role in the epidemiology of

Abbreviations: ECM, extracellular matrix; EHEC, enterohaemorrhagic *E. coli*; EPEC, enteropathogenic *E. coli*; HCP, haemorrhagic coli pilus; IMP, integrated membrane protein; LEE, locus of the enterocyte effacement; LRR, leucine-rich repeat; LTA, lipoteichoic acid; MSCRAMM, microbial surface components recognizing adhesive matrix molecules; PIA, polysaccharide intercellular adhesin; SFP, Sorbitol-fermenting EHEC O157 fimbriae; SPI, *Salmonella* Pathogenicity Island; STEC, Shiga toxin-producing *E. coli*; T8SS, type VIII secretion system.

foodborne diseases. The survival and persistence of micro-organisms on food matrices and food contact surfaces including technological equipment is influenced greatly by their ability to colonize biotic and abiotic surfaces, including those conditioned with biological material (Burgess *et al.*, 2014). Colonization of surfaces consists of two successive steps: initial adhesion and biofilm formation (Götz, 2002). A range of different factors promote bacterial adhesion, namely physico-chemical properties of the surface and the bacterial cell as well as specific cell surface adhesins and the exopolymeric matrix (Chagnot *et al.*, 2013). In this review, we will primarily focus on the cell surface structures and extracellular components involved in the adhesion of selected foodborne pathogens. Particular emphasis will be placed on surface proteins that are certainly the most functionally diverse components that play a role in bacterial adhesion (Chagnot *et al.*, 2013). Interestingly, several of the surface adhesins that are involved in adhesion to various host tissues may also play a role in adhesion to abiotic surfaces. Therefore, both clinical aspects and the implications of surface adhesins for food safety will be discussed.

Salmonella enterica

Salmonella enterica comprises six subspecies and over 2500 serovars. *Sa. enterica* infects humans and animals through the faecal–oral route. Most of the human pathogenic serovars belong to the subspecies *enterica*. The serovars Typhi and Paratyphi cause typhoid and paratyphoid fever with an estimated annual global incidence of 21 million cases and a fatality rate of 1–4 % (World Health Organization, 2008). The non-typhoid serovars cause gastroenteritis with over 90 million cases estimated globally each year, leading to more than 150 000 deaths (Majowicz *et al.*, 2010). *Sa. enterica* can adhere to a variety of biotic and abiotic surfaces using fimbriae and flagella, as well as other proteinaceous and non-proteinaceous adhesins. In Fig. 1, the most important adhesion molecules of *Sa. enterica* are depicted.

Fimbria

Fimbriae are thin, proteinaceous, fibrillar surface structures. They are approximately 3–10 nm in diameter, up to several micrometres long and are generally involved in biofilm formation, colonization and invasion of cells (Allen-Vercoe & Woodward, 1999; Reid & Sobel, 1987; Ugorski *et al.*, 2001; Wagner & Hensel, 2011). Based on some morphological differences, they have also been termed pili, which can be misleading with respect to their homology and/or molecular assembly mechanisms. Some researchers reserve the term pilus for the appendage required for bacterial conjugation. In this review, the terms fimbriae and pili will be used interchangeably. Fimbriae are also immunogenic and have therefore been used as successful vaccines in animals and are important targets for diagnostic tests (Müller *et al.*, 1991). Several types of fimbria are found in *Sa. enterica*.

Curli (thin aggregative fimbriae, Tafi or SEF17 fimbriae) are involved in adhesion to solid substrates, either abiotic, such as food contact surfaces (Jain & Chen, 2007; Woodward *et al.*, 2000), or biotic, such as animal host cells (Bäumler *et al.*, 1997; Dibb-Fuller *et al.*, 1999) and plant tissues (Barak *et al.*, 2005; Lapidot & Yaron, 2009). Interestingly, bacterial curli are also recognized by specific host proteins (Toll-like receptors) resulting in an immune response (Tükel *et al.*, 2010). Curli promote cell-to-cell interactions, aggregation and biofilm formation (Austin *et al.*, 1998; Castelijn *et al.*, 2012). They belong to a growing class of fibrillar proteins known as amyloids (Blanco *et al.*, 2012). Curli are known to be assembled via the extracellular nucleation–precipitation (ENP) pathway, i.e. the type VIII secretion system (T8SS), well studied in *Escherichia coli* (Chagnot *et al.*, 2013; Hammar *et al.*, 1996; Hammer *et al.*, 2007). In *Salmonella*, the *csg* genes (previously called *agf* genes) involved in curli biogenesis are organized in two adjacent divergently transcribed operons, *csgDEFG* and *csgBAC* (Collinson *et al.*, 1996; Römling *et al.*, 1998a). CsgD is the transcriptional regulator of the *csgBAC* operon (Römling *et al.*, 2000) and its complex expression is tightly regulated by global regulatory proteins (Gerstel *et al.*, 2003) acting in hierarchical regulatory cascades (Kader *et al.*, 2006), and by several nucleotide messengers, including cyclic-di-GMP (Simm *et al.*, 2007). Curli expression is influenced by a variety of environmental stimuli, such as starvation, oxygen tension, temperature, pH and osmolarity (Gerstel & Römling, 2003). In most wild-type *Salmonella* strains, this occurs at temperatures below 30 °C, but mutations in the *csgD* promoter can lead to curli expression independently of temperature (Römling *et al.*, 1998b). Interestingly, curli synthesis has recently been shown to be controlled by small regulatory RNAs (Bordeau & Felden, 2014; Mika & Hengge, 2013).

Type 1 fimbriae are the best characterized salmonella fimbriae and are approximately 7 nm in diameter and 0.5–2.0 μm in length (Korhonen *et al.*, 1980). They have a channelled appearance due to the arrangement of subunits around a hollow core (Chu & Barnes, 2010). These subunits are composed of 17 kDa protein subunits, called pilin (Korhonen *et al.*, 1980). Type 1 fimbriae are encoded by the *fim* gene cluster and are assembled by the chaperone–usher pathway (Hultgren *et al.*, 1991). These fimbriae are termed mannose sensitive because exogenous mannose inhibits binding by the fimbriae. The *fimA* gene encodes the major structural subunit, while the *fimH* gene encodes the adhesin protein that is located at the tip of the assembled fimbrial structure and mediates binding to the receptor (Ledeboer *et al.*, 2006). Type 1 fimbriae play a role in the adhesion of salmonella to epithelial cells and aid in biofilm formation on abiotic surfaces (Chu & Barnes, 2010; Korhonen *et al.*, 1980; Ledeboer *et al.*, 2006). These fimbriae are expressed *in vitro* after static growth for 48 h at 37 °C and are repressed by high osmolarity, low pH and low temperatures (Ledeboer *et al.*, 2006; Thanassi & Hultgren, 2000).

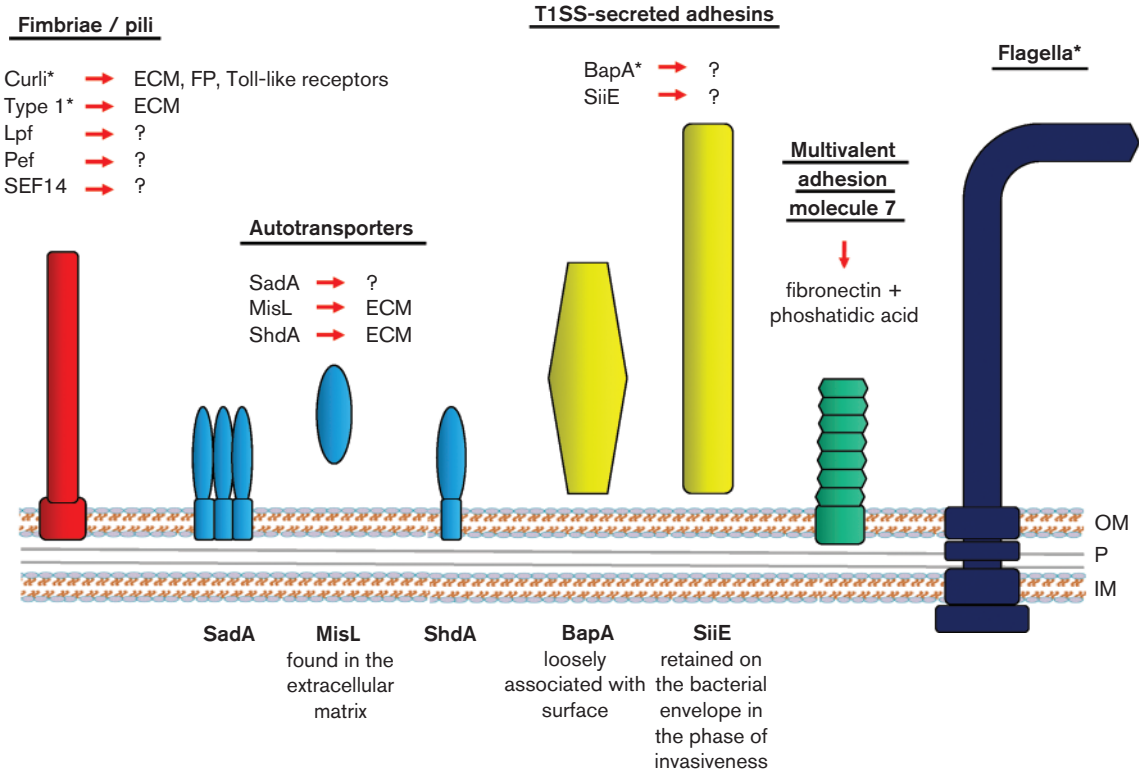

Fig. 1. Schematic drawing of the cell envelope of *Sa. enterica* (OM, outer membrane; P, periplasma with peptidoglycan; IM, inner membrane) with symbolized bacterial adhesion molecules including their receptors (?, unknown receptors). *Mediates adhesion to abiotic surfaces and biofilm formation. ECM, extracellular matrix proteins; FP, fibrinolytic proteins. The different bacterial adhesion molecule categories are symbolized and described in the text. The structures depicted do not necessarily reflect the real macromolecule structures.

To date, several other fimbriae expressing adhesive properties have been described. For instance, SEF14 fimbriae are only expressed in serovar Enteritidis and closely related serovars and may play a certain serovar-specific role in pathogenesis (Zhu *et al.*, 2013). SEF14 has been shown to be a T-cell immunogen and to contribute to adherence to murine epithelial cells (Ogunniyi *et al.*, 1994; Peralta *et al.*, 1994). Long polar fimbriae (Lpf) encoded by the *lpfABCDE* operon confer adhesion to the murine small intestine (Bäumler *et al.*, 1996b; Ledeboer *et al.*, 2006). Expression of Lpf in salmonella undergoes phase variation, such that the bacteria alternate between expressing and not expressing Lpf (Fierer & Guiney, 2001). Pef fimbriae are encoded on the 90 kb salmonella virulence plasmid by two divergently transcribed operons (Friedrich *et al.*, 1993). Pef fimbriae confer adhesion to the murine small intestine and to certain tissue culture cells (Bäumler *et al.*, 1996a).

Flagella

Recently it was established that bacterial flagella participate in many additional processes of motility including adhesion, biofilm formation, virulence factor secretion, adhesion and modulation of the immune system of eukaryotic cells (Duan *et al.*, 2013; Haiko & Westerlund-Wikström, 2013). Allen-Vercoe & Woodward (1999) concluded that a non-flagellate mutant strain, a flagellate but non-motile (paralysed) mutant strain and a smooth-swimming chemotaxis-deficient mutant strain were less adherent than the wild-type strain, but that observation depended on the assay conditions used and the fact that biofilm formation is strongly strain-dependent (Crawford *et al.*, 2010; Van Houdt & Michiels, 2010). Research data indicate that the flagellar filament, not motility, is necessary for adhesion to surfaces and biofilm formation by salmonella on gallstones (Crawford *et al.*, 2010; Prouty *et al.*, 2002). However, flagellar motility is required for biofilm formation on glass (Prouty & Gunn, 2003) and adhesion of bacteria to M cells of the appendix (Marchetti *et al.*, 2004).

Other proteinaceous adhesions

Several proteinaceous adhesins have been described in *Sa. enterica*. Biofilm-associated protein A (BapA) is a large, loosely associated surface protein that is required for biofilm formation, and also contributes to the invasion of epithelial cells (Latasa *et al.*, 2005). It is secreted by a T1SS, expression of which is coordinated with that of genes

encoding curli fimbriae and cellulose, i.e. through the action of *csgD*. SiiE is a giant protein (595 kDa) that mediates adhesion to epithelial cells (Gerlach *et al.*, 2007). It is encoded by *Salmonella* Pathogenicity Island (SPI) 4 together with its T1SS and retained on the bacterial envelope in the phase of invasiveness (Wagner *et al.*, 2011). The molecule has a highly repetitive structure of bacterial Ig-like domains, and Ca^{2+} binding confers SiiE with a rigid, rod-like habitus that is required to reach out beyond the LPS layer (Griessl *et al.*, 2013). SadA belongs to the family of trimeric autotransporter adhesins (TAAs) which are modular, highly repetitive proteins that form stable trimers on the bacterial cell surface (Hartmann *et al.*, 2012). In *Sa. enterica* serovar Typhimurium, expression of SadA resulted in cell aggregation, biofilm formation and increased adhesion to human intestinal epithelial cells when the O-antigen was removed. Thus, SadA may primarily be important under conditions where production of large surface structures is reduced, for example during macrophage infection (Raghunathan *et al.*, 2011). MisL and ShdA are fibronectin-binding autotransporter adhesins. MisL, which is found in the extracellular matrix (ECM), is involved in intestinal colonization (Dorsey *et al.*, 2005) and adhesion to plant tissue (Kroupitski *et al.*, 2013). It is encoded by SPI 3, and regulated by the transcriptional activator MarT. ShdA is surface-localized, and expressed in the intestine (Kingsley *et al.*, 2002). PagN is an outer-membrane protein which contributes to adherence and invasion in mammalian cells via interaction with proteoglycan and it is regulated by PhoP (Lambert & Smith, 2008). MAM7 is a relatively small and constitutively expressed surface adhesin that is widely distributed in Gram-negative bacteria including *S. enterica* (Krachler & Orth, 2011). It is anchored in the outer membrane and contains seven of the mammalian cell entry (mce) domains responsible for host cell binding (Chitale *et al.*, 2001; Saini *et al.*, 2008). MAM7 seems to bind to fibronectin with a low-affinity interaction, which is complemented by a second receptor, phosphatidic acid, resulting in an overall affinity that is very high.

Exopolymers

LPSs are known to be important for the initial step in biofilm formation (Williams & Fletcher, 1996) and the direct effect of LPSs on cell-surface interactions is related to the interaction between the O-antigen part and the solid surface (Jucker *et al.*, 1997). Changes in the cell surface caused by LPS alteration in the *ddhC* and *waaG* mutants of the serovar Typhimurium resulted in significant changes in the production of both curli and cellulose, as well as biofilm formation (Anriany *et al.*, 2006). Solano *et al.* (2002) concluded that cellulose is one of the main components of the biofilm produced by the serovars Enteritidis and Typhimurium and two operons, *bcsABZD* and *bcsEFG*, are required for cellulose biosynthesis. However, they found that cellulose deficiency did not affect serovar Enteritidis virulence. Furthermore, Vestby *et al.* (2009a) found no difference in biofilm formation on polystyrene when comparing wild-type strains with and without cellulose production, indicating minor differences also in adhesion. By contrast, Barak *et al.* (2007) found that mutations in bacterial cellulose synthesis (*bcsA*) and O-antigen capsule assembly and translocation (*yihO*) reduced the ability of bacteria to adhere to and colonize alfalfa sprouts whereas a colanic acid mutant was unaffected in plant adhesion or colonization. However, they noted that bacterial requirements for adhering to and colonizing plant tissue differ significantly from what is required for adherence to glass test tubes, other bacterial cells and animal cells.

Food safety impact

Salmonella can adhere to and form biofilm on various surfaces in food-processing environments, including food matrices and other organic material, and it has been shown that long-term persistence in production environments is correlated with the ability to form biofilm (Vestby *et al.*, 2009b). Fimbriae (primarily curli and type 1), flagella and BapA are all known to be involved in biofilm production, although their roles in adherence to abiotic surfaces are less clear and depend on the type of surface, as well as other environmental factors. Furthermore, LPSs are important for the initial steps of biofilm formation. Undoubtedly, the ability to recognize how salmonella attaches to raw products (e.g. meat, produce) and also food contact surfaces is an important area of focus, as a better understanding of this ability may provide valuable ways towards the elimination of this pathogenic bacterium from food-processing environments and eventually lead to reduced salmonella-associated human illness.

Enterohaemorrhagic *E. coli* (EHEC)

EHEC represent a subgroup of Shiga toxin-producing *E. coli* (STEC) that can cause serious human infectious diseases such as haemorrhagic colitis and haemolytic–uraemic syndrome (Nataro & Kaper, 1998). EHEC are zoonotic bacterial agents responsible for foodborne infections via contaminated animal food products, vegetables and watery drinks (Bavaro, 2012). By definition, all EHEC are considered as pathogenic STEC, but not all STEC are necessarily intestinal pathogenic *E. coli* (InPEC). While there are over 300 distinct STEC serotypes (Karmali *et al.*, 2010), only a limited number of serotypes have been reported to be involved in human infection, prevalently represented by serotype O157. The major non-O157 EHEC comprise the serotypes O26, O45, O103, O111, O121 and O145, the so-called 'big six' (Brooks *et al.*, 2005). Important surface adhesins of EHEC are encoded on the locus of the enterocyte effacement (LEE) pathogenicity island, other pathogenicity islands and plasmids (Farfan & Torres, 2012). Among the best-characterized adhesins are the bacterial outer-membrane protein intimin and its translocated intimin receptor Tir. In Fig. 2, the most important adhesion molecules of EHEC are depicted.

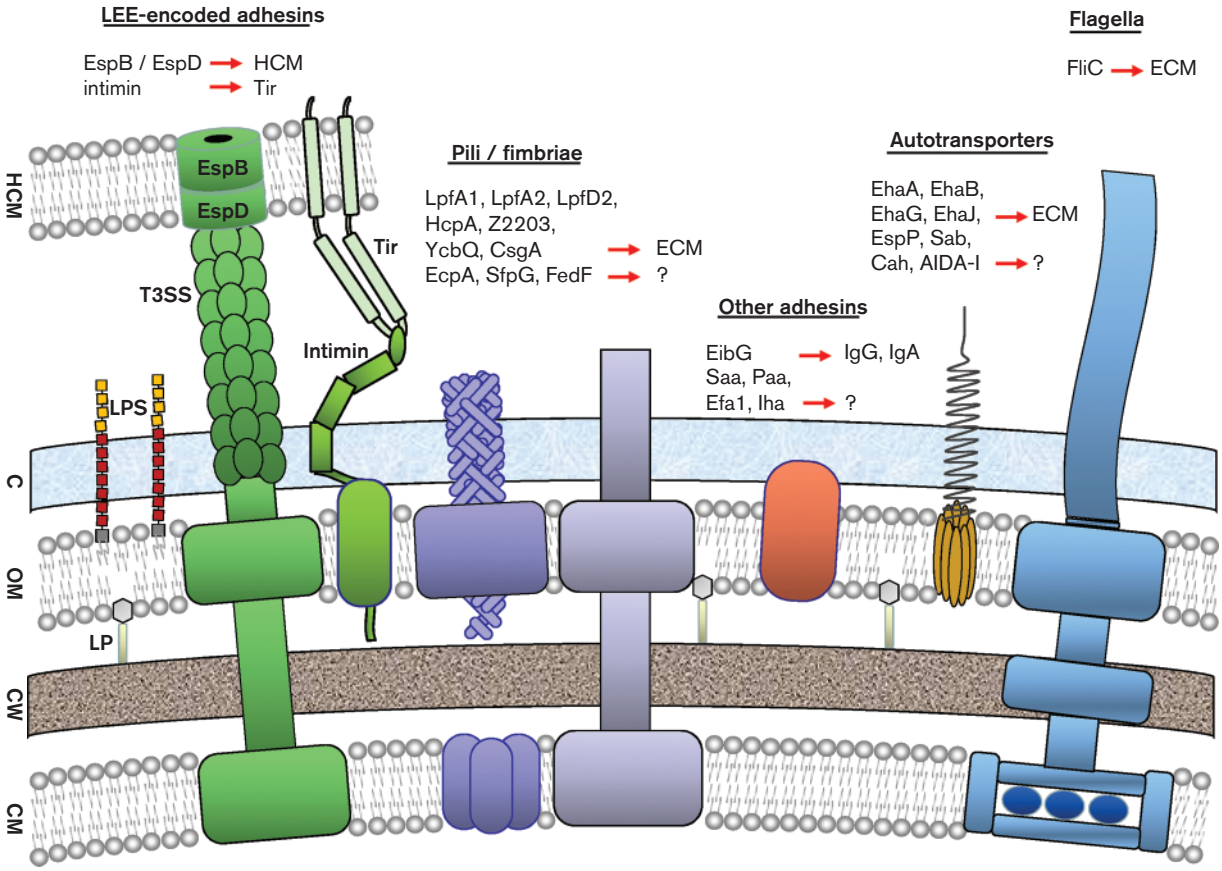

Fig. 2. Schematic drawing of the cell envelope of EHEC (CM, cytoplasmic membrane; CW, peptidoglycan cell wall; OM, outer membrane; C, capsule; LPS, lipopolysaccharide; LP, lipoprotein) with symbolized bacterial adhesion molecules including their receptors (?, unknown receptors). The interaction of EHEC with a host cytoplasmic membrane (HCM) via translocator proteins EspB and EspD is also shown. ECM, extracellular matrix proteins. The different bacterial adhesion molecule categories are symbolized and described in the text. The structures depicted do not necessarily reflect the real macromolecule structures.

LEE-encoded adhesions

A subgroup of EHEC (attaching and effacing *E. coli*, AEEC) harbour a pathogenicity island that encodes a T3SS, a number of type III effector proteins and the outer-membrane adherence protein intimin (*eae*) (Wong *et al.*, 2011). The T3SS forms a needle-like structure, called the injectisome, which connects the bacterial cell with the target host cell (Cornelis, 2010). While not generally described as a pilus, the injectisome is a cell surface supramolecular structure acting as a molecular syringe closely related to the Hrp (hypersensitive response and pathogenicity) pilus in plant pathogens. In fact, both structures belong to the T3SS, which comprises (i) subtype a, i.e. the non-flagellar T3SS (T3aSS) involved in the assembly of the injectisome as well as the Hrp pilus, and (ii) subtype b, i.e. the flagellar T3SS (T3bSS) responsible for assembly of the flagellum (Desvaux *et al.*, 2006; Journet *et al.*, 2005; Pallen *et al.*, 2005; Tampakaki *et al.*, 2004). Type III effector molecules are then transported through the needle and interact with the host actin structure to form a pedestal-like structure. The first effector molecule is the translocated intimin receptor Tir, which inserts into the host cytoplasmic membrane and comes into close contact with intimin. The resulting intimate attachment is a key feature of the interaction of LEE-positive bacteria with host cells (Wong *et al.*, 2011). The injectisome per se also mediates intimate bacterial adhesion to gut epithelial cells (Garmendia *et al.*, 2005) and was further reported to have marked tropism for the stomata during adhesion of EHEC O157 : H7 to lettuce leaves (Berger *et al.*, 2010; Saldaña *et al.*, 2011; Shaw *et al.*, 2008).

Pili

As in the case of *Salmonella* above, in this review the terms fimbriae and pili will be used interchangeably. EHEC contain numerous putative pili operons (Low *et al.*, 2006b; Rendón *et al.*, 2007). Pili are cell surface supramolecular protein complexes secreted and assembled by different secretion systems in diderm-LPS bacteria, i.e. archetypal Gram-negative bacteria (Chagnot *et al.*, 2013). Lpf are encoded by two loci in *E. coli* O157 : H7 [*lpf1* (*lpfABCC′DE*) and *lpf2* (*lpfABCDD′*)]. The LpfA proteins (encoded by both loci) as well as the LpfD2 protein of those pili mediate adhesion to

epithelial cells and intestinal colonization (Torres *et al.*, 2002a, 2007). Lpf bind to a wide variety of ECM components, e.g. fibronectin, laminin or collagen IV (Farfan *et al.*, 2011) and influence intestinal tissue tropism (Fitzhenry *et al.*, 2006; Jordan *et al.*, 2004; Torres *et al.*, 2004). Pili of the curli type (*csgBA* and *csgDEFG*) are assembled by the T8SS, i.e. the extracellular nucleation–precipitation pathway (Chagnot *et al.*, 2013; Desvaux *et al.*, 2009). While generally considered as very important for adhesion by its subunit protein CsgA, curli were shown not to contribute to intestinal colonization in *E. coli* O157 : H7 (Lloyd *et al.*, 2012). The *E. coli* common pilus (ECP, *yagZ*), also called meningitis-associated and temperature-regulated (Mat) fimbriae (Lehti *et al.*, 2013), is an important colonization factor involved not only in adhesion to epithelial cells (Rendón *et al.*, 2007) but also in the early stage of biofilm formation (Garnett *et al.*, 2012). The protein mediating this adhesion is EcpA. The haemorrhagic coli pilus (HCP, *hcpA*) is actually a type 4 pilus, which is secreted and assembled by a T2cSS (Chagnot *et al.*, 2013; Vignon *et al.*, 2003). In *E. coli* O157 : H7, HCP was shown to be multifunctional. Its main subunit protein HcpA mediates interbacterial connections conducive to biofilm formation as well as specific binding to certain ECM proteins, namely laminin and fibronectin but not collagen (Ledesma *et al.*, 2010; Xicohtencatl-Cortes *et al.*, 2009b), as well as intestinal colonization (Xicohtencatl-Cortes *et al.*, 2007). Sorbitol-fermenting EHEC O157 fimbriae (SFP, *sfpAHCDJG*, plasmid pSFO157 encoded) are a novel type of pilus identified in EHEC (Brunder *et al.*, 2001) and are most certainly assembled and secreted by the T7SS in diderm–LPS bacteria, i.e. the chaperone–usher pathway (Chagnot *et al.*, 2013). The protein acting as an adhesin is SfpG. While involved in haemagglutination, the SFP were also demonstrated to participate in adherence to epithelial cells (Müsken *et al.*, 2008). F9 pili (*f9* operon, Z2200–Z2206, chromosomally encoded on O-island 61), also secreted and assembled by the T7SS, are involved in binding to bovine fibronectin and to bovine epithelial cells (Low *et al.*, 2006a), although the expression of the T3aSS can hinder their adhesion capacities. The main subunit proteins of F9 pili are encoded by Z2203. The FedF proteins of F18 fimbriae (*fedABCEF*, plasmid encoded) mediate the adherence of EHEC to porcine enterocytes (Bardiau *et al.*, 2010). The YcbQ proteins of *E. coli* YcbQ laminin-binding fimbriae (ELF, *ycbQRST*) are responsible for specific binding to laminin but not fibronectin or collagen, and were demonstrated to contribute to adhesion to intestinal epithelial cells (Samadder *et al.*, 2009). Except for the injectisome, the respective contribution of these different pili to the colonization of food matrices remains to be evaluated.

Flagella

Motility of EHEC and other *E. coli* is mediated by flagella, and their filaments are encoded by the *fliC* gene. The heterogeneity of these flagella filaments (H-antigens) is determined by *fliC* sequence variations (Zhang *et al.*, 2014). H6 and H7 flagella, which are frequently present in EHEC, and their FliC monomers bind to mucus as well as mucins I and II (Erdem *et al.*, 2007), while no adherence was observed in deletion mutants of *E. coli* O157 : H7 strain EDL933. Likewise, *fliC* deletion mutants of this strain were significantly less adherent to leaf surfaces (Xicohtencatl-Cortes *et al.*, 2009a).

Autotransporters

Autotransporter proteins mediate adherence to eukaryotic cells and ECM proteins. While *E. coli* O157 strains mainly use curli for adhesion, non-O157 EHEC/STEC strains were reported additionally to depend on autotransporters (Biscola *et al.*, 2011). EHEC autotransporters EhaA, EhaB, EhaG and EhaJ (*ehaA*, *ehaB*, *ehaG*, *ehaJ*) are important for the formation of biofilms on biotic and abiotic surfaces and for adhesion to primary epithelial cells of the bovine terminal rectum (EhaA; Wells *et al.*, 2008), collagen I and laminin (EhaB; Wells *et al.*, 2009), laminin, fibrinogen, fibronectin and several collagen types (EhaG; Totsika *et al.*, 2012), as well as a blend of ECM compounds (EhaJ; Easton *et al.*, 2011). However, their involvement in a wild-type EHEC background has yet to be demonstrated. The extracellular serine protease autotransporter EspP (*espP*, plasmid encoded, e.g. pO157, pO113, pO26-Vir) was described by Brunder *et al.* (1997), and in the same year an isoform serine protease secreted by STEC (PssA) was characterized from a bovine isolate (Djafari *et al.*, 1997). To date, five subtypes have been identified, with EspPα being associated with the most virulent strains (Weiss & Brockmeyer, 2013). The *espP*α gene is often present in bovine isolates (Boerlin *et al.*, 1999). It increases the adherence of *E. coli* O157 : H7 strains to the intestine of calves (Dziva *et al.*, 2007), but may also downregulate the human complement system by cleavage of C3/C3b and C5 (Orth *et al.*, 2010). The STEC autotransporter mediating biofilm formation (Sab) is encoded by the *sab* gene located on a megaplasmid that is present in LEE-negative non-O157 STEC (Herold *et al.*, 2009). It mediates adherence to human epithelial cells as well as biofilm formation on polystyrene beads, but its prevalence in food isolates is low (Buvens & Piérard, 2012). The calcium-binding antigen 43 homologous protein Cah (*cah*, chromosomally encoded on O-islands 43 and 48) mediates biofilm formation under nutrient-poor conditions (Torres *et al.*, 2002b), and the induction of the *cah* gene into *E. coli* K-12 induced adhesion to alfalfa sprouts and seed coats (Torres *et al.*, 2005). The adhesion involved in diffuse adherence autotransporter AIDA-I (*aidA*) is plasmid or chromosomally (O-islands 43 and 48) encoded and contrary to the other autotransporters described, needs to be glycosylated with heptoses by the autotransporter adhesin heptosyltransferase [*aah*, plasmid or chromosomally (O-islands 43 and 48) encoded] (Benz & Schmidt, 2001). AIDA-I conveys adherence to porcine intestinal cells as well as biofilm formation on abiotic surfaces (Ravi *et al.*, 2007), and is dominant in pig isolates (Côté *et al.*, 2012). It is highly expressed under nutrient limitation (Berthiaume *et al.*, 2010).

Other proteinaceous adhesions

A number of other adhesins has been described in EHEC, the function and role of which have not yet been completely clarified. The *E. coli* immunoglobulin-binding protein EibG (*eibG*) is present in *eae*-negative STEC strains (Merkel *et al.*, 2010) and induces chain-like binding to HEp-2 cells as well as binding to human IgG and IgA (Lu *et al.*, 2006). The STEC autoagglutinating adhesin Saa (*saa*) is encoded on a large virulence plasmid of LEE-negative STEC, showing a low degree of amino acid sequence similarity to EibG and causing comparative adhesion behaviour *in vivo* (Paton *et al.*, 2001). The porcine attaching- and effacing-associated adhesin Paa is commonly detected in EHEC strains, and its sequence is highly conserved. The *paa* gene is also detected in enterotoxigenic *E. coli* (ETEC) and enteropathogenic *E. coli* (EPEC) strains (Leclerc *et al.*, 2007). In EHEC, it is thought to be involved in adhesion, but the exact mechanism still needs to be investigated. EHEC factor for adherence Efa1 (*efa1*, chromosomally encoded on O-island 122) confers haemagglutination, adherence to epithelial cells and autoaggregation (Nicholls *et al.*, 2000), and it is significant for STEC colonization of the bovine intestinal tract (Stevens *et al.*, 2002). Interestingly, the ORF of the *efa1* gene is highly homologous to that of lymphostatin (*lifA*, chromosomally encoded on O-island 122) in EPEC, which inhibits lymphocyte proliferation, interleukin production and pro-inflammatory cytokine synthesis (Klapproth *et al.*, 2000). *E. coli* O157 : H7 strains contain only a truncated *efa1* gene, but harbour the homologue *toxB* gene on the plasmid pO157 (Stevens *et al.*, 2004), and their deletion results in reduced adherence to epithelial cells. The IrgA homologue adhesin Iha (*iha*, either chromosomally encoded on O-islands 43 and 48 or plasmid encoded on megaplasmid pO113) differs from other adhesins in that it is homologous to iron-acquisition proteins (Tarr *et al.*, 2000) and is highly prevalent in human and cattle isolates (Wu *et al.*, 2010). Its expression in *E. coli* O157 : H7 on shredded iceberg lettuce during storage under near-ambient air atmospheric conditions was significantly increased in comparison with modified atmosphere packaging (Sharma *et al.*, 2011), as well as in ground beef after heating at 48 °C for 10 min (Slanec & Schmidt, 2011).

Exopolymers

Among other surface macromolecules, *E. coli* may produce two types of surface polysaccharide that are used for serotyping, namely LPSs (O-antigen, encoded by the *rfb* genes) and capsular polysaccharides (K-antigen). Their role in the attachment of EHEC to biotic and abiotic surfaces is still being investigated, and thus few general conclusions can be drawn yet. This may be due to strain-specific properties as well as the surface materials investigated.

LPSs of the *E. coli* serotypes O111 and O157 were demonstrated not to be involved in adhesion to human epithelial cells (Paton *et al.*, 1998). Furthermore, the O-side chains were shown to interfere with adherence of *E. coli* O157 : H7 strains to epithelial cells, as LPS-deficient mutants adhered more strongly than the wild-type strain (Bilge *et al.*, 1996). *E. coli* O157 : H7 LPS-deficient mutant strains attached equally well to alfalfa sprouts as their parent strains (Matthysse *et al.*, 2008). In contrast, an *E. coli* O157 : H7 mutant strain lacking the O-antigen attached significantly less well to iceberg lettuce surfaces than its parent strain (Boyer *et al.*, 2011).

Currently, *E. coli* capsules are divided into four groups while EHEC capsules are mainly assigned to groups 1 and 4 (for a review see Whitfield, 2006). Furthermore, Junkins & Doyle (1992) reported that *E. coli* O157 : H7 strains are capable of producing capsular exopolysaccharides with similar or identical structures to colanic acid. In the food matrix, the presence of capsular exopolysaccharides conveys a longer survival of EHEC strains in the acidic environment of yoghurt (Lee & Chen, 2004) and leads to a stronger attachment to fruits and vegetables (Hassan & Frank, 2004). Furthermore, cellulose and colanic acid (encoded by the *cps* gene) were found to be required in addition to poly-β-1,6-*N*-acetyl-D-glucosamine for maximum binding of *E. coli* O157 : H7 strains to alfalfa sprouts and seed coats (Matthysse *et al.*, 2008). While the production of exopolysaccharides decreased the attachment of *E. coli* O157 : H7 to stainless steel surfaces (Ryu *et al.*, 2004), it did not influence cell growth during biofilm formation on these surfaces (Ryu & Beuchat, 2005). According to Yeom *et al.* (2012), biofilm production in *E. coli* O157 : H7 is not a result of exopolysaccharide production but correlates with LPS production and increase in membrane rigidity.

Food safety impact

While certain pili, flagella (T3bSS) and the injectisome (T3aSS) participate in adhesion to vegetables (Shaw *et al.*, 2008; Xicohtencatl-Cortes *et al.*, 2009a), EHEC were also demonstrated to colonize certain meat ECM components (Chagnot *et al.*, 2012, 2013). Considering the wealth of determinants reviewed here as potentially involved in the contamination of the food chain, however, their exact and respective contribution is unknown and most certainly varies with the environmental conditions, emphasizing that much remains to be investigated.

Staphylococcus aureus

Staphylococcus aureus has been described as a causative agent of a wide spectrum of human infections ranging from minor infections to life-threatening diseases, such as osteomyelitis, endocarditis and sepsis, including several syndromes associated with the production of exotoxins and enterotoxins (Lowy, 1998). Staphylococci have also been recognized as the most frequent causative agents of infections associated with biofilm formation on catheters or prosthetic implants (Götz, 2002). Moreover, staphylococcal food poisoning (so-called staphylococcal gastroenteritis) is considered to be one of the most frequently occurring foodborne diseases worldwide (Ray & Bhunia, 2007). For instance, severe alimentary

intoxications caused by a toxigenic strain persisting on the inner surfaces of dairy plant equipment were reported in Japan (Asao *et al.*, 2003). A range of surface components expressing adhesive properties or modulating cell surface physico-chemistry have been described in *St. aureus*. These surface components enable *St. aureus* to colonize and infect various tissues as well as to adhere to and persist on abiotic surfaces. In Fig. 3, the most important adhesion molecules of *St. aureus* are depicted.

Surface proteins with specific target binding sites

St. aureus possesses a range of surface adhesins with a broad spectrum of target binding sites (Table 1). In other words, *St. aureus* can adhere to various components of the ECM to initiate colonization. Adherence to ECM is usually (but not only) mediated by protein adhesins of the microbial surface components recognizing adhesive matrix molecules (MSCRAMM) family, which are in most cases covalently anchored to the cell wall peptidoglycan via an LPXTG motif cleaved by sortase A (Clarke & Foster, 2006; Navarre & Schneewind, 1994). As summarized in Table 1, many of the surface proteins with specific target binding sites are able to bind multiple ligands; conversely, the same host component may be targeted by different adhesins. For instance, fibronectin-binding protein binds fibronectin and fibrinogen by two distinct domains. Such domain-specific binding has been comprehensively reviewed for *St. aureus* MSCRAMM proteins by other authors (Clarke & Foster, 2006; Heilmann, 2011). One of the principal functions of fibronectin-binding proteins, fibrinogen-binding proteins (clumping factors), elastin-binding protein, collagen adhesin, bone sialoprotein-binding protein, enolase, extracellular matrix-binding protein homologue and extracellular matrix protein-binding protein is to recognize and specifically bind one or more of the ECM components such as fibronectin, fibrinogen, elastin, collagen, sialoprotein, laminin and vitronectin. This allows *St. aureus* to adhere to and colonize different tissues and to cause a wide spectrum of diseases (Lowy, 1998). It has also been shown that conditioning of implant surfaces by ECM enhances their colonization by *St. aureus* (Harris *et al.*, 2004). Certain surface proteins with specific target binding sites have been shown to be involved not only in primary adhesion but also in cell–cell interaction and biofilm formation. This was observed in the case of fibronectin-binding proteins, plasmin-sensitive protein and the *St. aureus* surface proteins SasC and SasG (Corrigan *et al.*, 2007; Huesca *et al.*, 2002; O'Neill *et al.*, 2008; Schroeder *et al.*, 2009).

Besides simply adhesion to ECM, certain surface proteins may trigger the process of internalization through a fibronectin bridge to the host cell integrin $\alpha5\beta1$. As reported by Henderson *et al.* (2011), fibronectin-mediated internalization has been demonstrated for fibronectin-binding proteins but this could also be mechanistically presumed

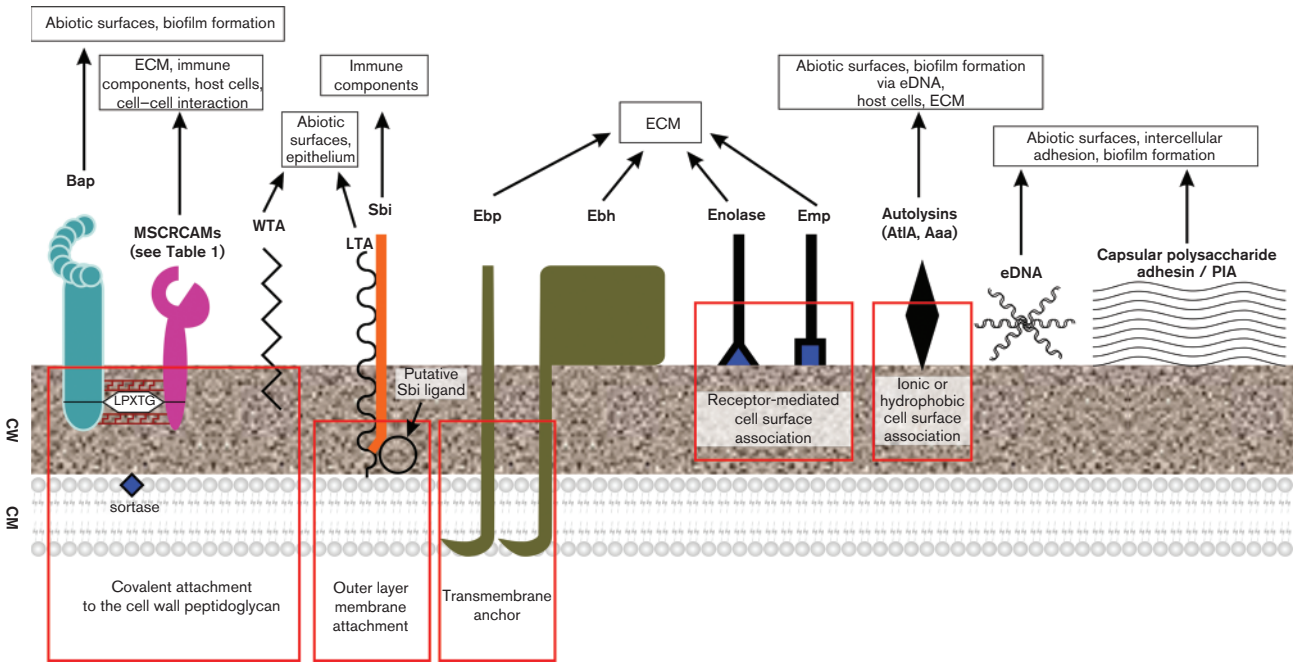

Fig. 3. Schematic drawing of the cell envelope of *St. aureus* (CM, cytoplasmic membrane; CW, peptidoglycan cell wall) with symbolized bacterial adhesion molecules including their receptors and/or functions. ECM, extracellular matrix proteins. The different bacterial adhesion molecule categories are symbolized and described in the text and Table 1. The structures depicted do not necessarily reflect the real macromolecule structures.

Table 1. *St. aureus* surface proteins with specific target binding sites

Surface protein	Gene	Target binding sites
Staphylococcal protein A (Spa)*	*spa*	Fc region of IgG (Uhlén *et al.*, 1984); IgM VH3 heavy chain (Vidal & Conde, 1985); von Willebrand factor (Hartleib *et al.*, 2000); TNFR1 (Gómez *et al.*, 2004); platelet complement receptor gC1qR/p33 (Nguyen *et al.*, 2000)
Staphylococcal protein Sbi†	*sbi*	Fc region of IgG (Zhang *et al.*, 1998); complement component C3 and complement regulators Factor H and FHR-1 (Haupt *et al.*, 2008)
Fibronectin-binding proteins (FnBPA, FnBPB)*	*fnbA*, *fnbB*	Fibronectin (Signäs *et al.*, 1989); fibrinogen (Wann *et al.*, 2000); elastin (Roche *et al.*, 2004); non-professional phagocytes through a fibronectin bridge to the host cell integrin $\alpha_5\beta_1$ (Henderson *et al.*, 2011) or direct binding to heat shock protein 60 (Dziewanowska *et al.*, 2000); platelets through a fibrinogen or fibronectin bridge to platelet integrin GPIIb/IIIa and IgG binding to the Fc gamma RIIA receptor (Fitzgerald *et al.*, 2006); cell-to-cell interaction and biofilm formation (O'Neill *et al.*, 2008)
Fibrinogen-binding proteins (clumping factors ClfA, ClfB)*	*clfA*, *clfB*	Fibrinogen (Ní Eidhin *et al.*, 1998; McDevitt *et al.*, 1994); fibrin via ClfA (Niemann *et al.*, 2004); complement regulator factor I via ClfA (Hair *et al.*, 2008); platelets through a fibrinogen bridge to platelet integrin GPIIb/IIIa and IgG binding to the Fc gamma RIIA receptor (Loughman *et al.*, 2005) or direct binding via ClfA (Siboo *et al.*, 2001); cytokeratin 10 via ClfB (O'Brien *et al.*, 2002a)
Sdr proteins (SdrC, SdrD, SdrE)*	*sdrC*, *sdrD*, *sdrE*	Platelets via SdrE (O'Brien *et al.*, 2002b); desquamated nasal epithelium via SdrC and SdrD (Corrigan *et al.*, 2009); cells expressing β-neurexin via SdrC (Barbu *et al.*, 2010)
Bone sialoprotein-binding protein (Bbp)*	*bbp*	Bone sialoprotein (Tung *et al.*, 2000)
Plasmin-sensitive protein (Pls)*	*pls*	Cellular lipids and cell–cell interaction (Huesca *et al.*, 2002); nasal epithelial cells (Roche, F.M. *et al.*, 2003)
Collagen adhesin (Cna)*	*cna*	Collagen and collagenous tissues such as cartilage (Switalski *et al.*, 1989)
Elastin-binding protein (Ebp)‡	*ebpS*	Elastin (Park *et al.*, 1999)
Enolase (laminin-binding protein)§	*eno*	Laminin (Carneiro *et al.*, 2004)
Iron-regulated surface determinants (IsdA, IsdB, IsdC, IsdH)*	*isdA*, *isdB*, *isdC*, *isdH*	Various iron-containing proteins (reviewed by Clarke & Foster, 2006); fibrinogen and fibronectin via IsdA (Clarke *et al.*, 2004); corneocyte envelope proteins via IsdA (Clarke *et al.*, 2009), platelet integrin GPIIb/IIIa via IsdB (Miajlovic *et al.*, 2010)
St. aureus surface proteins (SasB, SasC, SasD, SasF, SasG, SasK, SasH)*	*sasB*, *sasC*, *sasD*, *sasF*, *sasG*, *sasK*, *sasH*	Squamous nasal epithelium via SasG (Roche, F.M. *et al.*, 2003), cell–cell interaction and biofilm formation via SasC and SasG, attachment to polystyrene via SasC (Corrigan *et al.*, 2007; Schroeder *et al.*, 2009)
Serine-rich adhesin for platelets (SraP)*	*sraP*	Platelets (Siboo *et al.*, 2005)
Extracellular matrix-binding protein homologue (Ebh)‡	*ebh*	Fibronectin (Clarke *et al.*, 2002)
Extracellular matrix protein-binding protein (Emp)§	*emp*	ECM and plasma proteins such as vitronectin, fibronectin, fibrinogen and collagen (Hussain *et al.*, 2001)

*MSCRAMM proteins covalently anchored to the cell wall peptidoglycan via the LPXTG motif, or NPQTN and LPDTG motifs in the case of IsdC and SraP, respectively (Clarke & Foster, 2006; Heilmann, 2011; Siboo *et al.*, 2005; Tung *et al.*, 2000).
†Associated with the cytoplasmic membrane via LTA and a putative ligand (Smith *et al.*, 2012).
‡Transmembrane proteins (Heilmann, 2011).
§Proteins associated with the cell surface via unknown receptors (Bergmann *et al.*, 2001; Heitmann, 2011).

for other surface proteins that bind fibronectin, such as clumping factors, iron-regulated surface determinant A, extracellular matrix-binding protein homologue and extracellular matrix protein-binding protein. Staphylococcal surface proteins SpaA and Sbi are among those adhesins that specifically interfere with both innate and adaptive immune components (Table 1). Moreover, binding to a complement regulator factor has also been demonstrated for clumping factor A (Hair *et al.*, 2008).

Biofilm-associated protein (Bap)

Bap is a surface protein originally identified in *St. aureus* (Cucarella *et al.*, 2001). *St. aureus* strains harbouring the *bap* gene are highly adherent to inert surfaces and are strong biofilm formers (Tormo *et al.*, 2005, 2007). Bap was further identified in other Gram-positive and Gram-negative bacteria and appeared to correspond to a family of surface proteins involved in biofilm formation. Moreover, proteins of the Bap family possess the LPXTG domain

enabling their covalent anchoring to the cell wall by sortases (Lasa & Penadés, 2006; Latasa *et al.*, 2006). However, it has been demonstrated that *St. aureus* strains producing Bap exhibited a lower affinity to certain ECM proteins as well as to epithelial cells (Cucarella *et al.*, 2002). It could therefore be assumed that Bap negatively affects primary adhesion via specific receptors.

Autolysins

These non-covalently bound proteins are associated with the cell surface by ionic or hydrophobic interactions and have both enzymic and adhesive functions (Heilmann, 2011). The major autolysin of *St. aureus* (AtlA) is the most predominant peptidoglycan hydrolase in staphylococci. It is a bifunctional enzyme that undergoes proteolytic cleavage to yield two catalytically active proteins (murein hydrolases), an amidase and a glucosaminidase, both contributing to biofilm formation (Bose *et al.*, 2012). Biswas *et al.* (2006) reported that AtlA promotes adhesion of *St. aureus* to abiotic surfaces such as polystyrene and glass, and that deletion of the *atlA* gene resulted in a biofilm-negative phenotype. *St. aureus* autolysin AtlA and *Staphylococcus epidermidis* autolysin AtlE are similar in both sequence and domain organization. It was further demonstrated that *St. epidermidis* Esp protease cleaves Atl-derived murein hydrolases and prevents staphylococcal release of DNA, which serves as ECM in biofilms (Chen *et al.*, 2013).

Besides adhesion to abiotic surfaces, AtlA has recently been shown to have binding activity to fibronectin, fibrinogen, vitronectin and endothelial cells, and to be involved in the process of internalization into non-professional phagocytes via binding to the heat shock cognate protein 70 (Hirschhausen *et al.*, 2010). Similar affinity to specific biological components, namely fibronectin, fibrinogen and vitronectin, was described in another *St. aureus* autolysin Aaa, encoded by the *aaa* gene (Heilmann *et al.*, 2005).

Exopolymers

Although not covalently attached, extracellular polysaccharides and DNA (eDNA) are important adhesins in *St. aureus*. The polysaccharide intercellular adhesin (PIA) and the capsular polysaccharide adhesin are both encoded by the *icaADBC* operon. They both consist of a poly-β-1,6-*N*-acetylglucosamine backbone, but differ chemically due to different degrees of *N*-acetylation and *O*-succinylation and therefore provide different properties to the cell surface. While the capsular polysaccharide adhesin adds hydrophobic properties to the cell surface and enhances adhesion to abiotic surfaces (Maira-Litrán *et al.*, 2002), PIA is thought to be more important for the cell–cell interactions in the subsequent steps of biofilm formation. PIA possibly acts as an intercellular adhesin by electrostatically attracting the negatively charged teichoic acids (Heilmann, 2011). The enzyme dispersin B hydrolyses poly-β-1,6-*N*-acetylglucosamine, and Izano *et al.* (2008) showed that dispersin B could prevent *St. aureus* biofilm formation but could not disperse previously formed biofilm. The role of polysaccharide adhesins in cell–cell interactions must therefore be complemented by other matrix components as the biofilm matures.

Indeed, Izano *et al.* (2008) also showed that removal of eDNA could both prevent biofilm formation and remove previously formed *St. aureus* biofilms, and numerous reports point to eDNA as being critical for both adhesion and biofilm development in *St. aureus* and many other bacteria. Formation of eDNA in *St. aureus* is linked to the production of autolysins that lyse a subpopulation of cells. In addition to *atlA*, the gene encoding the major autolysin (the role for which in adhesion and biofilm formation is discussed above), there are also other genes (*cidA* and *lytS*) that are involved in cell wall remodelling and thus can act as autolysins promoting eDNA release. Knocking out any of these genes results in less eDNA and consequently less biofilm formation (Biswas *et al.*, 2006; Izano *et al.*, 2008; Mann *et al.*, 2009; Rice *et al.*, 2007; Sharma-Kuinkel *et al.*, 2009). Exactly how eDNA adsorbs to the cell surface and mediates adhesion is still somewhat unclear. Interaction with *N*-acetylglucosamine in the peptidoglycan has been suggested for other Gram-positive species (Harmsen *et al.*, 2010), and unspecific acid–base interactions from loops of eDNA strands protruding hundreds of nanometres from the cell surface have been suggested as critical for adhesion to abiotic surfaces (Das *et al.*, 2011). However, the interplay between eDNA and other adhesins has yet to be discovered.

While most surface proteins recognize specific targets in a conditioning layer or on host cells, a range of cell-surface components also facilitate strong adhesion forces between bacteria and biotic or abiotic surfaces through non-specific interactions mediated by electrostatic, acid–base and Lifshitz–Van der Waals forces. Teichoic acids are assembled by the Tar enzymes (encoded by the *tar* gene cluster) and anchored to the outer layer of the cytoplasmic membrane via a glycolipid (lipoteichoic acid, LTA) or covalently to the cell-wall peptidoglycan (wall teichoic acid, WTA) (Heilmann, 2011; Pereira *et al.*, 2008; Smith *et al.*, 2012). Mutants with reduced synthesis of LTAs completely lose their ability to form a biofilm on hydrophobic polystyrene plates, indicating changes in the physico-chemical properties of the bacterial cell surface due to the absence of LTAs (Fedtke *et al.*, 2007). While teichoic acids determine the overall negative charge of the cell surface, they also carry positive charges through linking of D-alanine to the glycerol phosphate or ribitol phosphate units. While the net cell surface charge remains negative, the local positive charges provided by D-alanine are critical for adhesion to abiotic surfaces, and Δ*dltA* mutants lacking the ability to link D-alanine to teichoic acids are therefore biofilm-deficient on several substrates (Gross *et al.*, 2001). The mutants regain adhesiveness when supplemented with $MgCl_2$ (Götz, 2002); hence, D-alanine affects adhesion simply by lowering the repulsive electrostatic forces towards negatively charged abiotic surfaces. Besides abiotic surfaces, the wall teichoic acid of *St. aureus* was also shown to mediate binding to nasal and vascular epithelium (Weidenmaier *et al.*, 2004, 2005).

Food safety impact

In *St. aureus*, surface adhesins and exopolymers are mainly responsible for the secondary contamination of food due to their involvement in colonization of food-processing surfaces and subsequent biofilm formation. From this point of view, the main role of surface adhesins with specific target binding sites lies in their involvement in primary adhesion, which is highly influenced by the presence of organic matter on food-processing surfaces. These surfaces are often conditioned with a variety of macromolecular and colloidal materials from food residues that allow microbial adhesion and subsequent biofilm formation (Poulsen, 1999; Rubio *et al.*, 2002). By contrast, some cellular components promote adhesion to abiotic surfaces in a non-specific manner by modulating cell surface properties or acting as a glue (e.g. autolysins, teichoic acids, Bap, PIA). However, their role in adhesion and biofilm formation on food-processing surfaces has not yet been studied under field conditions.

Listeria monocytogenes

L. monocytogenes is a ubiquitous soil bacterium and an opportunistic human foodborne pathogen causing listeriosis. In healthy, immunocompetent individuals, infections manifest as mild gastroenteritis or are completely asymptomatic. Indeed, severe cases of listeriosis are rare and essentially affect pregnant women, neonates, the elderly and immunocompromised patients (Farber & Peterkin, 1991). While *L. monocytogenes* is primarily a saprophytic bacterium with some strains even being avirulent (Lindbäck *et al.*, 2010; Roche, S.M. *et al.*, 2003; Vivant *et al.*, 2013), it can be a source of contamination in a wide range of raw and processed food (Valderrama & Cutter, 2013). This micro-organism is able to withstand and grow under a wide range of environmental stresses including pH (4.3–9.6), temperature (1–45 °C), salt (up to 10 % NaCl) and water activity (A_w down to 0.93). Furthermore, *L. monocytogenes* adheres to and colonizes abiotic surfaces, which contributes to its persistence in processing food chains (Carpentier & Cerf, 2011). Cell surface proteins are the major adhesion factors contributing to surface colonization in *L. monocytogenes* (Renier *et al.*, 2011). According to the most recent proteogenomic analyses based on the secretome concept (Desvaux *et al.*, 2009; Renier *et al.*, 2012), 58 secreted proteins were predicted to be located in the cell wall, of which 43 correspond to LPXTG proteins covalently attached to the cell wall and 15 correspond to GW (five), WXL (four), LysM (five) and PGBD1 (one) proteins attached to the cell wall by non-covalent interactions. Furthermore, numerous proteins are predicted to be located at the cytoplasmic membrane, namely 74 lipoproteins and 686 integrated membrane proteins (IMPs), in addition to cell surface supramolecular protein structures, namely the pseudo-pilus and flagellum. Surprisingly, only a few of these proteins and their role in adhesion to abiotic surfaces have been characterized to date. In Fig. 4, the most important adhesion molecules of *L. monocytogenes* are depicted.

Flagella

L. monocytogenes has four to six peritrichous flagella per cell and their expression is regulated by temperature (Peel *et al.*, 1988). The role of flagella in surface attachment of *L. monocytogenes* was first demonstrated by non-flagellated *fla* mutants, which were impaired in initial adhesion to stainless steel (Vatanyoopaisarn *et al.*, 2000). Several transposon mutagenesis studies supported the finding that mutations in flagella synthesis affect biofilm formation (Chang *et al.*, 2012; Kumar *et al.*, 2009; Ouyang *et al.*, 2012). In addition to temperature, pH and salinity were also shown to have an influence on flagella biosynthesis and, consequently, *L. monocytogenes* adhesion (Caly *et al.*, 2009; Tresse *et al.*, 2006, 2009). However, the absence of flagella only delayed biofilm formation but did not affect the final levels of adherent bacteria observed after longer periods of time (Vatanyoopaisarn *et al.*, 2000).

A mutant strain of *L. monocytogenes* 10403S that expresses flagella but lacks flagellar motility did not adhere to or invade human epithelial cells more efficiently than unflagellated listerial cells (O'Neil & Marquis, 2006). Likewise, non-motile flagellated listerial cells showed a similar defect in biofilm formation as an unflagellated mutant (Lemon *et al.*, 2007). The stimulating role of flagella in adhesion, invasion and biofilm formation is thus caused by motility, probably by increasing the likelihood of encountering a surface and overcoming the repelling electrostatic forces, and not by flagella acting as surface adhesins per se. The swarming over swimming motility hypothesis could further explain the importance of flagella as motility determinants rather than adhesins in biofilm formation (Renier *et al.*, 2011).

While those studies on *L. monocytogenes* agree on the positive influence of flagella on biofilm formation under static conditions in microtitre plates, the opposite appears to be the case under dynamic conditions. Although initial adhesion of both unflagellated and non-motile *L. monocytogenes* mutants was reduced under static conditions, the same mutants were hyper-biofilm formers when grown in flow cells (Todhanakasem & Young, 2008). The effect of flagella-driven motility on *L. monocytogenes* biofilms is therefore more complex than anticipated. All-in-all, flagella clearly affect adhesion and biofilm formation in *L. monocytogenes* but the promotion or inhibition of these processes seems to depend on the environmental conditions, such as hydrodynamics. The effects of different parameters such as growth conditions (e.g. pH, temperature, rich/minimum medium, nutrient or hydrodynamic regime) should probably be more closely considered to discriminate their respective and relative contribution. Interestingly, the flagellin FlaA is the first and only surface protein reported to be glycosylated in *L. monocytogenes* (Schirm *et al.*, 2004); the importance of *β*-*O*-linked

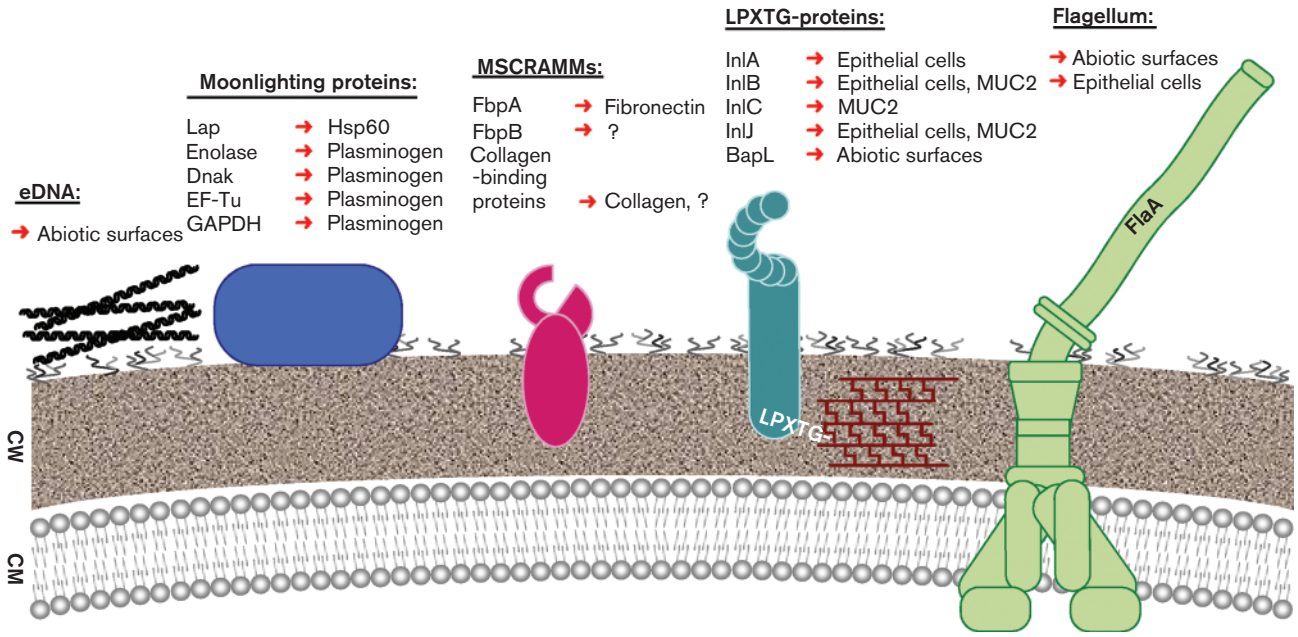

Fig. 4. Schematic drawing of the cell envelope of *L. monocytogenes* (CM, cytoplasmic membrane; CW, peptidoglycan cell wall) with symbolized bacterial adhesion molecules including their receptors (?, unknown receptors). The different bacterial adhesion molecule categories are symbolized and described in the text. The structures depicted do not necessarily reflect the real macromolecule structures.

N-acetylglucosamine glycosylation in listerial colonization has yet to be studied.

Biofilm-associated protein L (BapL)

A homologue of the biofilm-associated protein (Bap) was recently identified in *L. monocytogenes* and was named BapL (Lmo0435) (Jordan *et al.*, 2008). Among the 43 LPXTG proteins identified to date in *L. monocytogenes*, BapL is the only one characterized as playing a role in adhesion to abiotic surfaces. Indeed, an isogenic mutant of *lmo0435* showed a significant reduction in adhesion in comparison with wild-type *L. monocytogenes* 10403S. However, only four of 17 *L. monocytogenes* clinical and food isolates tested possessed the gene encoding this protein. Furthermore, several BapL-negative strains showed higher adherence levels than BapL-positive strains. Collectively, these data suggest that in *L. monocytogenes*, BapL is neither an essential factor influencing adhesion to surfaces nor is it required for virulence *in vivo* (Jordan *et al.*, 2008). In marked contrast to all other bacteria possessing a Bap homologue (Lasa & Penadés, 2006), the role of BapL in the course of sessile development could not be established as a reduced level of adhesion did not prevent the formation of a biofilm by the *bapL* mutant. This questions whether Lmo0435 is a functional protein and/or its actual relationship with the Bap family; such aspects would undoubtedly require further in-depth investigations.

Other protein determinants

The genome of *L. monocytogenes* encodes a family of proteins called internalins (Bierne *et al.*, 2007). Based on the presence of leucine-rich repeat (LRR) domains, i.e. exhibiting a Sec-dependent N-terminal signal peptide, 35 proteins were identified from the available genomes of different *L. monocytogenes* strains (Bierne *et al.*, 2007). Internalins can be discriminated into three classes: (i) covalent cell wall anchoring to peptidoglycan via the LPXTG motif, which represents the majority of internalins; (ii) non-covalent attachment to the cell surface via cell wall-binding domains such as GW or WXL motifs; and (iii) extracellular (Bierne *et al.*, 2007). Among the characterized LPXTG internalins, InlA, InlB and InlJ were demonstrated to play a role in adhesion to different types of eukaryotic host cells (Lecuit *et al.*, 1997; Sabet *et al.*, 2008). The two best-characterized internalins participating in *Listeria* invasion are InlA and InlB, which promote bacterial internalization into mammalian epithelial cells that express the surface proteins E-cadherin and tyrosine kinase Met, respectively (Mengaud *et al.*., 1996; Shen *et al.*, 2000). Interestingly, some internalins contain a mucin-binding domain (MucBP) (Bierne *et al.*, 2007). For InlB, InlC and InlJ, it was shown that the LRR was sufficient to bind to the mucin of type II (MUC2) but not to MUC1 (Lindén *et al.*, 2008). In fact, mucin glycoproteins constitute the protective mucus layer lining the gastrointestinal tract, where MUC2 comprises most of the mucus layer, whereas MUC1

is a cell surface mucin. To date, the role of internalins in adhesion to abiotic surfaces or biofilm formation has not been investigated.

Besides LPXTG proteins, none of the other surface proteins mentioned above (i.e. lipoproteins, IMPs and GW, WXL, LysM and PGBD1 proteins) have been investigated with respect to their contribution to adhesion and biofilm formation in *L. monocytogenes*. In addition to flagella, the genome of *L. monocytogenes* encodes another cell surface supramolecular protein structure secreted and assembled by the fimbrilin-protein exporter (FPE) (Desvaux & Hébraud, 2006). Homologues of these proteins form a pseudo-pilus in *Bacillus subtilis* (Chen & Dubnau, 2004; Chen *et al.*, 2006) and complete type IV pili in *Streptococcus pneumoniae* (Laurenceau *et al.*, 2013). In *L. monocytogenes*, expression and involvement of this structure in adhesion remains an intriguing and open question (Renier *et al.*, 2011). Furthermore, several MSCRAMM proteins were identified in *L. monocytogenes* by proteogenomic analysis (Chagnot *et al.*, 2012), including two fibronectin-binding proteins (FbpA and FbpB) and seven collagen-binding proteins (Renier *et al.*, 2013). Of these, only FbpA was shown to bind to immobilized human fibronectin (Dramsi *et al.*, 2004); the remaining proteins have never been functionally characterized with respect to their potential to bind to ECM components, especially fibronectin and collagen, and to their role in adhesion to abiotic surfaces and/or colonization of food matrices, especially meat products.

Moonlighting proteins are defined as multifunctional proteins that perform multiple autonomous and often generally completely unrelated functions, especially when present at different subcellular locations (Wang *et al.*, 2013). Many of the currently known moonlighting proteins are highly conserved, especially glycolytic enzymes and chaperones (Henderson & Martin, 2011). In *L. monocytogenes*, the primarily cytoplasmic proteins enolase (Lmo2455), DnaK (Lmo1473), EF-Tu (Lmo2653) and glyceraldehyde-3-phosphate dehydrogenase (GAPDH; Lmo2459) were shown to be present on the bacterial cell surface and to bind human plasminogen (Schaumburg *et al.*, 2004). *Listeria* adhesion protein (Lap) is one of the most fascinating moonlighting proteins. It was initially characterized as a key surface adhesin and allows bacterial adhesion to intestinal epithelial cells (Santiago *et al.*, 1999). Later, this protein was identified as an alcohol acetaldehyde dehydrogenase. On the surface of human host cells, Lap binds with high affinity to human chaperone Hsp60 (Wampler *et al.*, 2004). The involvement of moonlighting proteins in adhesion and biofilm formation in *L. monocytogenes* remains to be investigated but might be of particular importance.

Exopolymers

A striking feature of biofilm formation in *L. monocytogenes* is the absence of a dense exopolymeric matrix as observed in most other microbial biofilms (Renier *et al.*, 2011). In general, the extracellular biofilm matrix is a complex mixture of different exopolysaccharides as well as eDNA and/or polyglutamate. Using ruthenium red staining, carbohydrate compounds could be visualized on the surface of *L. monocytogenes* cells (Borucki *et al.*, 2003). However, this method could not discriminate between the presence of exopolysaccharides and other glycosylated molecules such as peptidoglycan, teichoic acids or proteins, and these data were not considered conclusive by the authors themselves. This is further supported by the fact that isolation and characterization of exopolysaccharides has not been convincingly reported for *L. monocytogenes* in the last decade. This is also supported by the absence of genes encoding known biosynthetic pathways for exopolysaccharides in the genome of sequenced *L. monocytogenes* strains (Renier *et al.*, 2011). Interestingly, several groups have reported the presence of fibre-like structures between listerial cells and a surface or other bacterial cells (Borucki *et al.*, 2003; Hefford *et al.*, 2005; Marsh *et al.*, 2003; Renier *et al.*, 2011; Zameer *et al.*, 2010); some authors suggested that massive shrinkage of the exopolymeric materials resulting from complete dehydration in the course of sample processing for electron microscopy might lead to the presence of these thin fibres. In regard to their appearance and distribution on the bacterial cell surface, these data were recently reinterpreted and it was proposed that these structures could actually be pili (Renier *et al.*, 2011). This question would, of course, require further in-depth investigations.

L. monocytogenes is amongst the bacteria for which the presence of eDNA in the biofilm matrix has been demonstrated (Harmsen *et al.*, 2010; Okshevsky & Meyer, 2013). The presence of DNase I inhibited initial adhesion of *L. monocytogenes* EGDe to glass slides (Harmsen *et al.*, 2010). Moreover, eDNA was isolated from the supernatants of the inoculum and was shown to be of chromosomal origin. In chemically defined medium, the addition of DNase I at early time points inhibited biofilm formation and dispersed biofilms of a number of isolates from different sources and to some extent also exerted the same effects when added at later time points (Harmsen *et al.*, 2010). An effect of DNase treatment on biofilm formation by *L. monocytogenes* was also shown by other authors (Kadam *et al.*, 2013). Interestingly, an insertion mutant in the *lmo1386* gene encoding a putative DNA translocase was impaired in biofilm formation (Chang *et al.*, 2013). With respect to biofilm formation ability, *L. monocytogenes* is subjected to high strain variability, which is further exacerbated by the environmental conditions (Lianou & Koutsoumanis, 2013). All-in-all, much remains to be learned about the contribution of exopolymer(s) to *L. monocytogenes* adhesion and sessile development along the food chain.

Food safety impact

Regarding food contamination and safety, extracytoplasmic proteins appear the most important determinants of adhesion in *L. monocytogenes*, although eDNA can also play

a role in the early stages of biofilm formation under certain environmental conditions. However, their exact and respective contributions in contamination of the food-processing environment and/or food matrices have not been demonstrated to date and the mechanisms and proteins involved in the colonization process remain to be characterized.

Conclusions and remarks

The ability of bacteria to adhere to surfaces or bind various compounds provides them with a range of physiological and survival advantages such as: biofilm formation and long-term persistence; increased resistance to physical stressors and chemical compounds; tissue colonization, infection and internalization; and evasion of the immune response through interaction with components of the immune system. Bacterial adhesion is a multi-factorial process involving a range of factors such as environmental conditions, host–pathogen interactions and physico-chemical properties of both inert and bacterial cell surfaces. Specific structures present on the bacterial cell surface play a key role in adhesion. These can act as surface adhesins with varying degrees of affinity to particular substrates or can modulate cell surface properties in such a way that promotes bacterial adhesion. In many bacteria, various surface determinants such as fimbriae/pili, flagella, LPSs, exopolysaccharides and numerous surface proteins may be involved in adhesion. A range of adhesins specifically recognize different host receptors, such as various structures on the surface of eukaryotic cells (e.g. integrins), blood proteins and ECM proteins, but could also be involved in adherence to abiotic surfaces and biofilm formation. Therefore, understanding the mechanisms of bacterial adhesion would help in the development of new strategies targeting molecular structures involved in attachment. This would in turn facilitate new approaches for the control of bacterial adhesion in terms of prevention of both bacterial contamination and infection.

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