Artykuł przeglądowy *Review*

Role of type 1 fimbriae in the pathogenesis of *Salmonella* infections: twenty five years of personal research

MACIEJ UGORSKI

Department of Biochemistry and Molecular Biology, Faculty of Veterinary Medicine, Wroclaw University of Environmental and Life Sciences, Norwida 31, 50-375 Wrocław, Poland

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Ugorski M.

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Summary

Type 1 fimbriae are surface structures produced by all *Salmonella* serovars. It is generally accepted that these appendages play an important role in early stages of *Salmonella* infections, being responsible for the attachment and colonization of gut mucosa by the pathogens. Nevertheless, despite many years of research, their exact role in the pathogenesis of infections caused by these bacilli remains far from fully understood. The same applies to the role of the FimH adhesin of type 1 fimbriae, which directly contributes to their adhesive properties. In this review article, I summarize the major findings on FimH adhesins from different *Salmonella* serovars (Enteritidis, Gallinarum, Choleraesuis, Dublin, and Abortusovus), the result of 25 years of research on this topic. Our studies focused on the adhesive properties of these adhesins, including their interactions with specific ligands and the identification of cellular receptors, as well as their role in *Salmonella* host specificity.

Keywords: *Salmonella*, type 1 fimbriae, FimH adhesion, infection

Type 1 fimbriae are adhesive appendages prevalently expressed on the surface of many species of *Enterobacteriaceae*, including *Salmonella*. They are filamentous structures, taking the form of rigid, hollow fibers with a diameter of approximately 7 nm and a length not exceeding 1.5 µm (1, 34, 43). A bacterial cell produces between 200 and 300 such fimbriae on its surface. The shaft of the fimbria is composed of FimA protein subunits (Fig. 1), whose molecules form a right-handed helical structure. The FimA protein shows low amino acid sequence homology among different species of *Enterobacteriaceae* (46). At the tip of the fimbria, the adhesin FimH is located, which directly participates in binding to carbohydrate structures rich in D-mannose residues. In the case of *Salmonella*, the FimH protein is connected to the fimbrial shaft through the FimF protein. Despite being collectively classified as mannose-sensitive (MS) lectins, FimH proteins from different species of enterobacteria display distinct specificities toward particular carbohydrate structures (19, 20, 49). Although knowledge regarding the natural ligands for FimH adhesins remains limited, it is believed that they preferentially bind mannose-rich *N*-glycans of glycoproteins, which are commonly found in the membranes of eukaryotic cells (49).

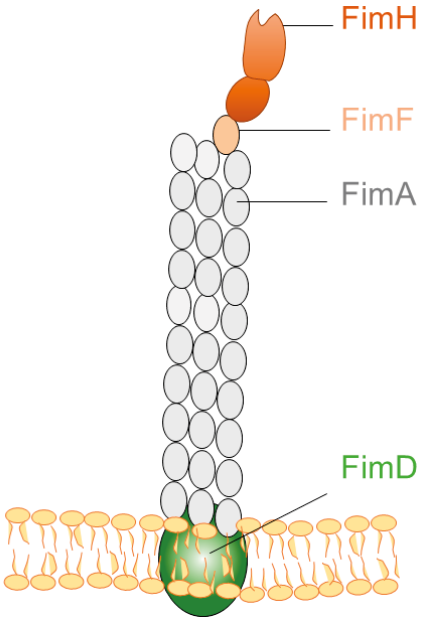

Fig. 1. Schematic representation of the structure of *Salmonella* type 1 fimbriae
Explanations: FimH – lectin-like protein that directly participates in binding to high-mannose *N*-glycans; FimF – protein that connects FimH with the fimbrial shaft; FimA – a major structural protein that participates in forming the fimbrial shaft; FimD – an usher outer membrane protein.

The first information about the expression of type 1 fimbriae by *Salmonella* was published in 1958 by Duguid and Gillies (13). Subsequent studies revealed that type 1 fimbriae are expressed in more than 80% of 1453 clinical isolates, representing 149 serovars (11). Taking into account the surface localization of these organelles, as well as their morphology and agglutination properties, it has been suggested from the very beginning that they are responsible for the adhesion of *Salmonella* to host cells and participate in the early stages of infection by these pathogens. Indeed, early studies using isogenic strains differing in their level of fimbriae expression demonstrated that, in the case of serovars such as *Salmonella enterica* subsp. *enterica* sv. Typhimurium (*S.* Typhimurium), California (*S.* California), and Enteritidis (*S.* Enteritidis), the presence of type 1 fimbriae was essential for their adhesion to various cells and tissues (3, 4, 8, 11, 17, 37, 39, 44, 45, 52). Using, as mentioned above, natural mutants of *S.* Typhimurium and *S.* Enteritidis to orally infect mice, it was demonstrated that type 1 fimbriae play a significant role in natural infections caused by these pathogens (4, 12, 53, 54). Fimbriated strains exhibited significantly higher virulence compared to mutants lacking fimbriae. However, it was determined that type 1 fimbriae are not absolutely essential for infection in animals by these bacilli; rather, they facilitate the process and prolong the carrier state. With the advancement of recombinant DNA techniques, studies on the role of type 1 fimbriae in adhesion began to utilize deletion mutants generated through insertional inactivation of genes within the *fim* operon. These studies confirmed the crucial role of these fimbriae in the interaction of *S.* Typhimurium and *S.* Enteritidis with host cells (2, 7, 9, 18, 26, 28). Deletion mutants were also studied to reveal the significance of type 1 fimbria expression in the pathogenesis of *Salmonella* infections in mice. Surprisingly, in contrast to results obtained with natural mutants/variants, deletion mutants of *S.* Typhimurium exhibited higher virulence compared to the non-fimbriated parent strains (38, 56). More detailed studies on this topic, involving *S*. Enteritidis and 19-day-old rats revealed that type 1 fimbriae are an important virulence factor, but only in the early stages of infection (42). In the later stages, this phenotypic trait becomes disadvantageous for the bacteria, as in experiments involving administration of both wild-type and mutant strains, the fimbriated strain was preferentially eliminated from the gastrointestinal tract. Several studies have used chickens for experimental purposes because *S*. Enteritidis plays an important role in poultry infections. These studies showed that in the case of chickens type 1 fimbriae do not appear to play a major role in the pathogenesis of these bacteria (1, 47), although another report suggests their possible involvement in increased colonization of the liver and spleen in 1-day-old chicks during the early stages of *S.* Enteritidis infection (10).

FimH adhesins and their role in *Salmonella* adhesion and pathogenicity: an *in vitro* study

The first evidence of the crucial role of the FimH adhesin in the binding of *Salmonella* to host cells was provided by Hancox et al. (26). These studies demonstrated that a mutant strain of *S.* Typhimurium lacking the ability to express the FimH protein loses its ability to bind to HeLa and HEp-2 cells. In addition to cells of epithelial origin (6, 24), the FimH adhesin plays an important role in the binding of *S.* Typhimurium to M cells and dendritic cells. In the case of the former, the likely ligands for the adhesin are the N-glycosylated chains of glycoprotein 2 (GP2), which is involved in transcytosis and in enabling the bacteria to cross the intestinal barrier (27). In the case of the latter, FimH adhesin is involved in the internalization of bacteria (25). However, despite the key role of the FimH protein in the adhesive properties of type 1 fimbriae, and consequently in the binding of *Salmonella* to host cells, research has been limited only to the Typhimurium serovar. Therefore, we have decided to expand research on FimH adhesins to include *Salmonella* serovars that are important from the epidemiological standpoint of poultry and livestock infections. Taking into account that the Enteritidis serovar is one of the most frequent causes of foodborne zoonosis in humans (15, 16), we first cloned and sequenced the *fimH* gene of *S.* Enteritidis. We demonstrated a very high degree of homology – approximately 97% – between the newly cloned *fimH* gene of *S*. Enteritidis and the *fimH* genes of *S*. Typhimurium (32). This finding revealed that the FimH adhesin expressed by various *Salmonella* serovars are allelic variants similar to the *Escherichia coli* FimH adhesins (51). This was further confirmed when the *fimH* genes of the following *Salmonella enterica* subsp. *enetrica* serovars: Gallinarum (*S*. Gallinarum), Abortusovis (*S*. Abortusovis), Choleraesuis (*S*. Choleraesuis), and Dublin (*S*. Dublin) were cloned and sequenced by us (22, 33).

Despite the high homology (98%-99%) of *fimH* genes, which results in only minor changes to the amino acid sequence of FimH proteins, these differences can drastically affect the binding and, consequently, the biological properties of type 1 fimbrial FimH adhesins. Unlike most *Salmonella* serovars, *S. enterica* subsp. *enterica* sv. Gallinarum biovar Gallinarum (*S*. Gallinarum) and *S. enterica* subsp. *enterica* sv. Gallinarum biovar Pullorum (*S*. Pullorum) produce type 1 fimbriae that do not agglutinate erythrocytes and bind to animal cells in a mannose-independent manner (11, 33). For this reason, these mannose-resistant (MR) fimbriae were originally described as type 2 fimbriae (11, 43). We have shown that the loss of the ability to bind to high-mannose *N*-glycans results from a single mutation in which the threonine found in FimH adhesins from *S*. Enteritidis and *S*. Typhimurium, which bind well to glycoproteins carrying high-mannose

oligosaccharides, is replaced by isoleucine at position 78 (33). Significantly, we were able to demonstrate that the mutated FimH proteins from biovars Gallinarum and Pullorum, with threonine at position 78 instead of isoleucine, as seen in the wild-type FimH, bound well glycoproteins carrying high-mannose oligosaccharides. It should be noted that in this study we used recombinant proteins produced in *E. coli* for the first time to analyze the biological properties of FimH adhesins, instead of using whole bacteria or purified type 1 fimbriae.

Allelic variants of the FimH protein are found not only at the serovar level, but also within the same serovar of *Salmonella*. This was first demonstrated for *S*. Typhimurium strains LB5010 and SL1344, which differ by two amino acid residues: alanine versus glycine at position 61, and serine versus phenylalanine at position 118 (6). Notably, these amino acid differences affected the biological properties of the adhesins. *S*. Typhimurium LB5010, which expresses FimH with an alanine at position 61 and a serine at position 118, adhered to HEp-2 cells in significantly higher numbers than *S*. Typhimurium SL1344, which expresses FimH with a glycine at position 61 and a phenylalanine at position 118. Based on these findings, the former strain was called the „high-adhesive" variant and the latter strain was called the „low-adhesive" variant. Unlike *S*. Typhimurium, our studies on *S*. Enteritidis showed that all analyzed human and avian isolates were the low-adhesion variant (29). As an extension of these studies, we used site-directed mutagenesis to mimic situations found in *S*. Typhimurium and obtain high-adhesion variant of *S*. Enteritidis FimH protein with an alanine at position 61 and a serine at position 118 (21). Using recombinant protein, we demonstrated the critical role of the substitution of phenylalanine for serine at position 118 in the binding properties of *S*. Enteritidis FimH, and identified Ser118 as the primary determinant of the high-adhesion phenotype of type 1 fimbriae from *S*. Enteritidis. These studies also demonstrated, for the first time, that the functional differences observed in whole fimbriated bacteria could be reproduced at the level of the purified adhesin. Using recombinant forms of the FimH protein, we clearly showed that the adhesive properties of type 1 fimbriae are solely determined by structural differences in FimH proteins. This contradicts previous suggestions that the fimbrial shaft influences these properties (14, 55).

Recent studies using chicken enterocytes and a macrophage-like cell line have highlighted the importance of the FimH adhesin in the pathogenesis of *S*. Enteritidis infection (40). Based on these findings, we propose that FimH from type 1 fimbriae significantly impacts the extent of host cell invasion by *Salmonella* because it facilitates direct, stable contact between bacteria and host cells through type 1 fimbrial-mediated adhesion. This allows for more efficient T3SS-1-mediated invasion.

Most pathogenic serovars of *Salmonella enterica* can infect a variety of animals. For instance, *S*. Enteritidis causes disease in poultry, humans, rodents, pigs, and cattle. A few serovars, called host-adapted serovars, have a narrow host range. *S*. Dublin and *S*. Choleraesuis, for instance, primarily infect cattle and swine, respectively, and occasionally infect humans and mice (50). Some serovars are host-restricted and are generally associated with one species of animal. *S*. Gallinarum, for example, infects only poultry (5), while *S*. Abortusovis infects only sheep (43). Our comparative studies of allelic variants of the FimH adhesin from several *Salmonella* serovars – Enteritidis, Typhimurium, Abortusovis, Choleraesuis, and Dublin – suggest that type 1 fimbriae may play a role in *Salmonella* host specificity. Western blot analysis revealed that the recombinant FimH adhesin from *S*. Enteritidis bound to a 130-kDa surface membrane glycoprotein when analyzed with lysates from enterocyte cell lines derived from sheep, pigs, and cattle (22). In contrast, FimH adhesins from *S*. Abortusovis, *S*. Choleraesuis, and *S*. Dublin bound to a 55-kDa surface membrane glycoprotein present in each cell line. It should be noted that *S*. Enteritidis FimH is a low-adhesion variant, while the FimH adhesins from *S*. Abortusovis, *S*. Choleraesuis, and *S*. Dublin are high-adhesion variants. The differential binding of FimH proteins from host-unrestricted and host-adapted *Salmonella* to enterocyte glycoproteins was confirmed using mutant FimH proteins obtained *via* site-directed mutagenesis. The low-adhesive variant of FimH from *S*. Choleraesuis, which has the Leu57Pro mutation, lost the ability to bind to the 130-kDa protein band. Instead, it interacted with a glycoprotein of approximately 55-kDa. Conversely, the high-adhesive variant of FimH from *S*. Enteritidis with the Asn101Ser mutation did not bind to the 130-kDa receptor but interacted with the 55-kDa glycoprotein ligand. The importance of type 1 fimbriae for *Salmonella* host specificity is further supported by the identification of calreticulin, expressed by pig intestinal cells, as a receptor for the *S*. Choleraesuis FimH adhesin (23).

FimH adhesins and their role in *Salmonella* adhesion and pathogenicity: an *in vivo* study

Building on the aforementioned studies, the significant role of type 1 fimbrial FimH adhesins in *S*. Enteritidis infection was confirmed through *in vivo* experiments using a mouse model (36). In this study, mice were infected orally with wild-type *S*. Enteritidis and its mutant strain, *S*. Enteritidis fimH::kan, which has a knockout of the *fimH* gene. Using bioluminescence imaging, it was found that (1) bioluminescent signals, and therefore bacteria, were detected much earlier in mice infected with the mutant *S*. Enteritidis strain lacking type 1 fimbriae than in mice infected with the wild-type strain, and (2) mice infected with the non-fimbriated mutant strain of *S*. Enteritidis had

significantly shorter infection-free times than animals inoculated with the fimbriated wild-type strain. Based on these results, we propose that *S*. Enteritidis and other non-typhoidal serovars that express type 1 fimbriae are less virulent and have less potential for systemic dissemination than serovars that lack these appendages or carry nonfunctional FimH adhesins (e.g., *S*. Gallinarum). In the same studies, we also showed that type 1 fimbriae significantly affect cytokine expression and secretion levels in mouse enterocytes, suggesting their contribution to the induction of intestinal inflammation during *Salmonella* invasion, which creates an ideal environment for the growth of this pathogen.

Type 1 fimbriae and veterinary practice

As mentioned above, *S*. Enteritidis is one of the main causes of foodborne gastroenteritis in humans, making it an important public health and economic problem. Therefore, the eradication of poultry *Salmonella* infection is a serious problem in veterinary practice that can be solved with a proper vaccination program. However, due to the intracellular nature of *Salmonella* infections, developing an effective vaccine remains a challenge. Due to their localization and structure, fimbriae are an excellent target for the host immune system. Therefore, we studied the immunological properties of the FimA (major fimbrial protein of SEF21/type 1 fimbriae), AgfA (major fimbrial protein of SEF17), and SefA (major fimbrial protein of SEF14 fimbriae) proteins of *S*. Enteritidis with the intention of using them as components of a subunit vaccine. Our studies revealed that the recombinant SefA and FimA proteins elicited a strong humoral and cellular response in immunized hens, unlike the AgfA protein (31, 35). However, despite high immunogenicity, the immunization of hens with SefA and FimA proteins showed only a weak protective effect, as evidenced by the lack of significant differences in the colonization of the duodenum, cecum, liver, and spleen by *S.* Enteritidis between vaccinated and unvaccinated birds (35).

Unlike *S*. Enteritidis, *S*. Gallinarum biovars Gallinarum and Pullorum only infect poultry (see above). However, due to significant economic losses, they remain important pathogens in Africa and Asia. These biovars cause different diseases, such as fowl typhoid and pullorum disease. Therefore, their proper diagnosis is important but challenging. In light of these difficulties, we found that *fimH* genes from *S*. Gallinarum and *S.* Pullorum differ by a single-nucleotide polymorphism (SNP) at position 544 bp from the start codon of the *fimH* open reading frame. This allowed us to propose a new DNA-based method to discriminate between these two biovars (30). Using polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) with the restriction enzyme SacI, we demonstrated that PCR amplicons of the *fimH* gene from *S*. Gallinarum and *S*. Pullorum can be clearly distinguished, enabling clear discrimination between the two biovars.

Concluding remarks

Over the course of more than 25 years of research performed in the Department of Biochemistry and Molecular Biology at the Faculty of Veterinary Medicine at the Wrocław University of Environmental and Life Sciences, this line of study has resulted in a series of publications that have significantly contributed to our understanding of the role of type 1 fimbriae in the pathogenesis of *Salmonella* infections. This is evidenced by the numerous citations of these publications in the international scientific literature. Among the most notable achievements was the demonstration that FimH adhesins of various *Salmonella enterica* serovars represent allelic variants with a very high degree of amino acid sequence homology, ranging from 97% to 98%. Of great significance, even single amino acid substitutions can substantially alter their biological activity. A particularly striking example is the FimH protein of *S. Gallinarum*, in which the substitution of threonine with isoleucine at position 78 results in a loss of the ability to bind sugar chains rich in D-mannose.

In reflecting on this period of scientific activity, it is also a great pleasure to acknowledge the invaluable contributions of all my collaborators, without whom none of these studies would have been possible.

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Author's address: prof. dr hab. Maciej Ugorski, Department of Biochemistry and Molecular Biology, Faculty of Veterinary Medicine, Wroclaw University of Environmental and Life Sciences, Norwida 31, 50-375 Wrocław, Poland; e-mail: maciej.ugorski@upwr.edu.pl