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Review

Manipulation of host membranes by the bacterial pathogens *Listeria*, *Francisella*, *Shigella* and *Yersinia*

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ABSTRACT

Bacterial pathogens display an impressive arsenal of molecular mechanisms that allow survival in diverse host niches. Subversion of plasma membrane and cytoskeletal functions are common themes associated to infection by both extracellular and intracellular pathogens. Moreover, intracellular pathogens modify the structure/stability of their membrane-bound compartments and escape degradation from phagocytic or autophagic pathways. Here, we review the manipulation of host membranes by *Listeria monocytogenes*, *Francisella tularensis*, *Shigella flexneri* and *Yersinia* spp. These four bacterial model pathogens exemplify generalized strategies as well as specific features observed during bacterial infection processes.

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Abbreviations: ECM, extracellular matrix; FPI, *Francisella* pathogenicity island; LLO, listeriolysin O; OMP, outer membrane protein; PMN, polymorphonuclear neutrophils; ROS, reactive oxygen species; Subsp, subspecies; T3SS, type 3 secretion system; TLR, toll-like receptor; YCV, *Yersinia*-containing vacuole.

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1. Introduction

The eukaryotic cell is a complex environment. Vital functions such as internalization of external solutes, synthesis of novel proteins or genetic information storage are compartmentalized in membrane-bound structures. From the perspective of a pathogen, the eukaryotic cell might be considered as a rich source of nutrients as well as a protected environment in which microbial replication may take place, avoiding contact with extracellular host defenses such as the complement or antibodies. Cells have adapted molecular strategies (e.g. phagocytosis or autophagy) to counteract and destroy intracellular pathogens, which in turn have developed strategies to cope with host cell defenses.

Four major bacterial pathogenesis paradigms are *Listeria monocytogenes*, *Francisella tularensis*, *Shigella flexneri* and *Yersinia* spp. *Listeria monocytogenes* is responsible for a food-borne disease associated with meningitis and abortions. *Francisella tularensis* is the agent of tularemia, responsible for ulcero-glandular and pneumonic severe infections. *Shigella flexneri* is the etiological agent of human bacillary dysentery. *Yersinia pseudotuberculosis* and *Y. enterocolitica* are responsible for enteritis, ileitis, and mesenteric lymphadenitis in humans, and are closely related to *Y. pestis*, the plague agent. Besides their clinical importance, these bacteria are useful paradigms to understand the diverse strategies used by microbial pathogens to subvert host cell functions. While *Listeria* and *Yersinia* invade cultured epithelial cells by interacting with host cell plasma membrane receptors *via* bacterial surface proteins, *Shigella* injects bacterial effectors within the host cell cytoplasm to promote internalization and *Francisella* induces spacious pseudopods to invade macrophages. Once inside epithelial cells, *Yersinia* proliferates in a membrane-bound compartment while *Listeria*, *Shigella* and *Francisella* disrupt their internalization vacuole and escape into the host cell cytoplasm. *In vivo*, *Yersinia* is mainly extracellular. We will review here the major interactions that occur between these important bacterial pathogens and host membranes.

2. Listeria monocytogenes

Listeria monocytogenes is a Gram-positive bacterial pathogen responsible for listeriosis, a disease causing meningitis in the new born and abortions in pregnant women. *Listeria* is now a classical model in the study of bacterial intracellular parasitism [1]. The pathogenic potential of *Listeria* is intimately associated with its capacity to traverse the intestinal, the foeto/placental and the blood/brain barriers, which requires invasion of host cells [2]. Using tissue cultured cells, it has been observed that upon internalization *Listeria* is able to disrupt its membrane-bound compartment and to escape to the host cytoplasm, where it proliferates [3]. *Listeria* is then able to avoid the autophagosomal machinery by polymerizing host cell actin, which drives bacterial cytoplasmic movement and cell-to-cell spread [4] (Fig. 1). Exceptions to this canonical intracellular cycle model have been observed *in vivo*, particularly during the traversal of the intestinal barrier. Indeed, in goblet cells *Listeria* does not escape from the internalization vacuole and is transcytosed to the lamina propia [5]. It may also persist in spacious phagosomes [6].

2.1. Cell invasion mediated by InlA and InlB

Listeria induces its internalization within mammalian cells by using surface invasion molecules that activate host plasma membrane receptors. These proteins belong to the internalin family, whose members are characterized by the presence of N-terminal leucine-rich repeats which drive protein–protein interactions [7]. The prototype internalin (InlA) is a covalently-anchored bacterial cell wall protein that binds the cellular adherens junction molecule E-cadherin [8] and triggers *Listeria* internalization in polarized epithelial cells and tissues, particularly during traversal of the intestinal and the foeto/placental barriers [9,10]. InlB is a second bacterial surface molecule loosely attached to lipoteichoic acids which can interact with the host molecule Met [11], the receptor for the hepatocyte growth factor. *In vitro*, InlB promotes *Listeria* internalization in a wide variety of epithelial cells; *in vivo*, InlB has been shown to cooperate with InlA in crossing the foeto/placental barrier [2] (Fig. 1A).

2.1.1. InlA-invasion pathway

Lipid rafts are cholesterol-rich signaling platforms that allow the clustering of signaling proteins or lipids, and which play a central role in the infectious process of many bacterial pathogens. Interaction between InlA and E-cadherin leads to E-cadherin clustering, a process which is alleviated if lipid rafts are disorganized [12]. This event is followed by E-cadherin cytoplasmic tail phosphorylation and ubiquitylation by Src and Hakai, respectively [13], allowing clathrin recruitment *via* its adaptor Dab2 [14]. Clathrin is a major membrane coat implicated in the endocytosis of many surface receptors, and clathrin-mediated endocytosis is involved in the infectious process of several important viruses [15]. The study of *Listeria* internalization demonstrated that during bacterial entry, clathrin does not function as a classical endocytic coat but instead

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