iMedPub Journals

Acta Rheumatologica

2020
Vol.7 No.3

Strain-Specific Interactions of *Listeria monocytogenes* with the Autophagy System in Host Cell

John H Brumell

Department of Molecular Genetics, University of Toronto, Toronto, Canada Email:john.brumell@sickkids.ca

Listeria monocytogenes is an intracellular bacterial pathogen that can replicate in the cytosol of host cells. These bacteria undergo actin-based motility in the cytosol via expression of ActA, which recruits host actin-regulatory proteins to the bacterial surface. *L. monocytogenes* is thought to evade killing by autophagy using ActA-dependent mechanisms. ActA-independent mechanisms of autophagy evasion have also been proposed, but remain poorly understood. Here we examined autophagy of non-motile (Δ*actA*) mutants of *L. monocytogenes* strains 10403S and EGD-e, two commonly studied strains of this pathogen. The Δ*actA* mutants displayed accumulation of ubiquitinated proteins and p62/SQSTM1 on their surface. However, only strain EGD-e Δ*actA* displayed colocalization with the autophagy marker LC3 at 8 hours post infection. A bacteriostatic agent (chloramphenicol) was required for LC3 recruitment to 10403S Δ*actA*, suggesting that these bacteria produce a factor for autophagy evasion. Internalin K was proposed to block autophagy of *L. monocytogenes* in the cytosol of host cells. However, deletion of *inlK* in either the wild-type or Δ*actA* background of strain 10403S had no impact on autophagy evasion by bacteria, indicating it does not play an essential role in evading autophagy. Replication of Δ*actA* mutants of strain EGD-e and 10403S was comparable to their parent wild-type strain in macrophages. Thus, Δ*actA* mutants of *L. monocytogenes* can block killing by autophagy at a step downstream of protein ubiquitination and, in the case of strain EGD-e, downstream of LC3 recruitment to bacteria. Our findings highlight the strain-specific differences in the mechanisms that *L. monocytogenes* uses to evade killing by autophagy in host cells.L. monocytogenes was first portrayed by E.G.D. Murray in 1924 dependent on six instances of unexpected demise in youthful bunnies, and distributed a portrayal with his associates in 1926 .[14] Murray alluded to the living being as Bacterium monocytogenes before Harvey Pirie changed the variety name to Listeria in 1940.[15] Although clinical depictions of L. monocytogenes disease in the two creatures and people were distributed during the 1920s, it was not perceived as a noteworthy reason for neonatal contamination, sepsis, and meningitis until 1952 in East Germany.[16] Listeriosis in grown-ups was later connected with patients living with traded off insusceptible frameworks, for example, people taking immunosuppressant medications and corticosteroids for malignancies or organ transfers, and those with HIV infection. L. monocytogenes was not distinguished as a reason for foodborne sickness until 1981, notwithstanding. An episode of listeriosis in Halifax, Nova Scotia, including 41 cases and 18 passings, generally in pregnant ladies and children, was epidemiologically connected to the utilization of coleslaw containing cabbage that had been tainted with L. monocytogenes-defiled sheep manure.[18] Since at that point, various instances of foodborne listeriosis have been accounted for, and L. monocytogenes is currently broadly perceived as a significant danger in the food business.

The infective portion of L. monocytogenes differs with the strain and with the helplessness of the person in question. From cases contracted through crude or as far as anyone knows purified milk, one may securely accept that, in helpless people, less than 1,000 absolute living beings may cause sickness. L. monocytogenes may attack the gastrointestinal epithelium. When the bacterium enters the host's monocytes, macrophages, or polymorphonuclear leukocytes, it becomes bloodborne (sepsis) and can develop. Its quality intracellularly in phagocytic cells likewise allows admittance to the cerebrum and presumably transplacental movement to the embryo in pregnant ladies. This cycle is known as the "Trojan Horse instrument". The pathogenesis of L. monocytogenes focuses on its capacity to endure and increase in phagocytic host cells. It appears to be that Listeria initially advanced to attack layers of the digestive organs, as an intracellular contamination, and built up a compound instrument to do as such. This includes a bacterial protein internalin (InlA/InlB), which connects to a protein on the intestinal cell film "cadherin" and permits the microbes to attack the cells through a zipper system. These grip particles are likewise to be found in two other surprisingly intense boundaries in people — the blood-cerebrum obstruction and the fetal–placental hindrance, and this may clarify the obvious fondness that L. monocytogenes has for causing meningitis and influencing children in utero. Once inside the cell, L. monocytogenes quickly ferments the lumen of the vacuole conformed to it during cell section to initiate listeriolysin O, a cholesterol-subordinate cytolysin fit for upsetting the vacuolar layer. This liberates the microorganism and gives it admittance to the cytosol of the cell, where it proceeds with its pathogenesis.[27] Motility in the intracellular space is given by actin gathering instigating protein, which permits the microscopic organisms to utilize the host cell's actin polymerization apparatus to polymerize the cytoskeleton to give a "help" to the bacterial cell so it can move in the phone. A similar instrument additionally permits the microbes to head out from cell to cell.

The term microbe came into utilization in the 1880s.[1][2] Typically, the term is utilized to depict an irresistible microorganism or specialist, for example, an infection, bacterium, protozoan, prion, viroid, or fungus.[3][4][5] Small creatures, for example, specific sorts of worms and bug hatchlings, can likewise deliver illness. In any case, these creatures are ordinarily, in like manner speech, alluded to as parasites instead of microorganisms. The logical investigation of minute life forms, including tiny pathogenic creatures, is called microbiology, while the investigation of sickness that may incorporate these microorganisms is called pathology. Parasitology, in the interim, is the logical investigation of parasites and the living beings that have them.

This work is partly presented at 13th world congress on Rheumatology, Orthopedics And Sports Medicine