The role of surface glycoconjugates in Leishmania pathogenicity
S.M. Beverley
Dept. of Molecular Microbiology, Washington University School of Medicine, St. Louis MO 63110, USA

Cell trafficking alterations caused by the H. pylori VacA toxin
M. de Bernard, W. Dundon, C. Montecucco, M. Moschioni, E. Papini, R. Rappuoli and M. Zoratti
Centro CNR Bimembrane and Dipartimento di Scienze Biomediche, Universita di Padova and CHIRON-Vaccines, Siena, Italy

Peroxisome Biogenesis and Human Peroxisomal Disorders
Yukio Fujiki
Department of Biology, Kyushu University, Fukuoka 812-8581; CREST, JST, Tokyo 107-0013, Japan

Molecular interactions and signaling during the infection by the intracellular pathogen
Pascale Cossart
Unité des Interactions Bactéries-Cellules Institut Pasteur 28 rue du Docteur Roux 75724 Paris cedex 15

Listeria monocytogenes is a bacterial pathogen responsible for food borne infections. Interestingly, this bacterium crosses silently the intestinal barrier. Via the lymph and the blood, infection then proceeds to the liver and spleen and ultimately to the brain and the placenta, resulting in meningitis, septicemias and abortions. In infected tissues, Listeria monocytogenes is intracellular due to its capacity to survive in phagocytic cells and to invade and replicate into non-phagocytic cells. Our laboratory is interested in understanding how this organism enters into non phagocytic cells.

Our goal is to identify the bacterial factors involved, their cellular ligands and the signaling events that trigger actin rearrangements and membrane curvature, culminating in internalisation. Two bacterial proteins play a key role in this process: internalin and InlB which are both Leucine rich repeats (LRR) proteins but have different types of association to the bacterial cell surface. Two receptors have been identified on the mammalian cell: E-cadherin, an adhesion molecule, for internalin and gC1qR, the receptor for C1q, the first component of the complement cascade, for InlB. After bacterial cell contact, a sequential activation of tyrosine kinase(s), PI3 kinase and PLC-γ takes place. How these events orchestrate to mediate entry is currently investigated.

Peroxisomes are ubiquitous intracellular organelles that are found in organisms ranging from yeasts to human beings. Peroxisomes function in a wide variety of metabolic pathways, including the catabolism of very-long chain fatty acids by β-oxidation and the biosynthesis of plasmalogen-type glycerolipids. The functional significance of human peroxisomes is highlighted by fatal human genetic diseases, called peroxisome biogenesis disorders such as Zellweger syndrome. Genetic heterogeneity has been evidenced in subjects with peroxisomopathies and abortions. In infected tissues, Listeria monocytogenes is intracellular due to its capacity to survive in phagocytic cells and to invade and replicate into non-phagocytic cells. Our goal is to identify the bacterial factors involved, their cellular ligands and the signaling events that trigger actin rearrangements and membrane curvature, culminating in internalisation. Two bacterial proteins play a key role in this process: internalin and InlB which are both Leucine rich repeats (LRR) proteins but have different types of association to the bacterial cell surface. Two receptors have been identified on the mammalian cell: E-cadherin, an adhesion molecule, for internalin and gC1qR, the receptor for C1q, the first component of the complement cascade, for InlB. After bacterial cell contact, a sequential activation of tyrosine kinase(s), PI3 kinase and PLC-γ takes place. How these events orchestrate to mediate entry is currently investigated.

These various cell alterations induced by VacA are interpreted in terms of a strategy of the long-term survival of Helicobacter pylori in the sub-mucous apical niche of the stomach mucosa where it is preferentially found.

© 2000 Biochemical Society