Title:	Role of host membrane regulators in salmonella typhimurium pathogenesis
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Abstract:	Salmonella Typhimurium is an intracellular pathogen which invades epithelial cells and macrophages through its bacterial effector proteins and reorganizes the host cell endomembrane machinery to establish its replicative niche (Salmonella containing Vacuole-SCV). It also acquires membranes and nutrients from host late endocytic compartments. With the help of SPI-2(Salmonella pathogenesis island) effectors, highly dynamic tubular filamentous structures emanate from the SCV called Salmonella Induced Filaments (SIFs), which are essential for its survival as Salmonella uses them to acquire nutrients for its replication. Endoplasmic Reticulum (ER) is a very important organelle in the cell which performs a variety of functions like protein and lipid synthesis, and calcium storage. It maintains physical contacts with all the organelles in the cell with multiple contact Sites (MCS) that enables ER to regulate various aspects of organelle dynamics, including fission, maturation and positioning. ER-Lysosomal MCS are formed with ER tubules and lysosomes and are very essential in many physiological processes including lipid transfer, calcium exchange, receptor tyrosine kinase signaling, lipid droplet formation, autophagosome formation. Salmonella and its effect on the endomembrane biology has been under study for more than 3 decades now and inter-organelle crosstalk is a very new topic to be studied under the context of Salmonella infections. So, in this study I look into Reticulon-4a (Rtn4a), an ER curvature stabilizing protein and its role in Salmonella pathogenesis. We have found that Salmonella replication is inhibited under Rtn4a Knockdown condition, notably, the number of Salmonella-induced filaments remained similar but their progression seemed impaired. We also found that LAMP1 positive tubulation was substantially less around 3 hours post-infection in Rtn4a Knockdown condition.
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