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Host cell actin assembly is necessary and likely to provide the propulsive force for intracellular movement of Listeria monocytogenes

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Listeria monocytogenes is able to escape from the phagolysosome and grow within the host cell cytoplasm. By 3 h after initiation of infection, actin filaments begin to concentrate at one end of the bacterium. Polarization of F-actin is associated with intracellular bacterial movement, long projections of actin filaments forming directly behind the moving bacteria. New actin monomers are added to the region of the projection in proximity to the bacterium. The rate of new actin filament growth correlates closely with the speed of bacterial migration. This actin structure is anchored within the cytoplasm, serving as a fixed platform for directional expansion of the actin filament network. The actin projection progressively lengthens as the bacterium migrates. Cytochalasin blocks both elongation of the projection and bacterial movement but does not result in complete depolymerization of the bacterially induced actin structure, residual actin and alpha-actinin persisting in proximity to one end of the bacterium. Bacteria initially migrate within the cortical cytoplasm but later move to the peripheral membrane, where they form filopodiumlike structures which pivot and undulate in the extracellular medium. In the filopodia, bacteria are occasionally seen to abruptly change direction, turn 180 degrees, and move back into the medullary region of the host cell. All filopodium movement ceases once the bacterium containing the F-actin projection returns to the cortical cytoplasm. These results indicate that host cell actin polymerization is necessary for intracellular migration of listeriae and suggest that directional actin assembly may in fact generate the propulsive force for bacterial and filopodial movement.