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Listeria monocytogenes: A virulence tailored to cross barriers

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Listeria monocytogenes is an invasive pathogen capable to cross physiological barriers like blood brain (BBB) and fetoplacental barrier (FPB) leading to severe infection of the central nervous system and the fetus during pregnancy respectively. This pathogen is a facultative intracellular microorganism, capable of invading most host cells and each step of intracellular parasitism is dependent upon the production of virulence factors controlled by a single pleiotropic regulatory activator, PrfA.

To understand the underlying molecular mechanisms involved during invasive process of the brain and the fetal infection we studied the role of virulence factors by using a genetic approach, and *in vitro* human cell lines and *in vivo* animal models.

We first demonstrated that in vitro, the virulence factor listeriolysin–O (LLO) acts as a pro-inflammatory protein to induce an NF-kB dependent activation of endothelial cells. Then by using transgenic murine model, brain endothelial activation occurs also *in vivo*, and is absolutely required to induce brain parenchymal invasion by facilitating the adhesion of *L. monocytogenes*-infected bone marrow derived myeloid cells on the endothelium of the BBB.

In contrast to endothelial cells, trophoblastic cells of the FPB are highly susceptible to be infected, including by avirulent bacteria. This suggests that placental cells provide a protective niche for bacterial survival. However, the crossing of the FPB requires the virulence factor ActA, which mediates cell-to-cell spreading and plays a major role to induce fetal infection.

Thus, the virulence factors required for intracellular life cycle of the bacteria are also exploited by *L. monocytogenes* in the crossing strategies of physiological barrier during infectious process.

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