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Salmonella transforms follicle-associated epithelial cells into antigen sampling M cells to promote intestinal invasion

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As the preferred cell type to translocate across the gut epithelium, *S. Typhimurium* targets antigen-sampling microfold (M) cells, which represent a small proportion of the specialized follicular associated epithelium (FAE) overlying mucosa-associated lymphoid tissues. Although M cell numbers have been documented to increase during Salmonella infection, the molecular mechanism underlying this increase is unclear. Using *in vitro* and *in vivo* infection models we demonstrate that the *S. Typhimurium* type IIIeffector protein SopB induces an epithelial-mesenchymal transition (EMT) of FAE enterocytes into M cells. This cellular trans-differentiation depends on activation of the Wnt/β-catenin signaling leading to induction of both the growth factor receptor RANK as well as its ligand RANKL. The autocrine activation of RelB expressing FAE enterocytes by RANKL/RANK induces EMT regulator Slug that marks epithelial trans-differentiation into M cells. This study demonstrates a novel host-pathogen interaction in which the pathogen transforms primed epithelial cells to promote host colonisation and invasion.

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