Motility allows S. Typhimurium to benefit from the mucosal defence.

The mammalian intestine is colonized by a dense bacterial community, called microbiota. The microbiota shields from intestinal infection (colonization resistance). Recently, we have shown that enteropathogenic Salmonella spp. can exploit inflammation to compete with the intestinal microbiota. The mechanisms explaining the enhanced pathogen growth in the inflamed intestine are elusive. Here, we analysed the function of bacterial flagella in the inflamed intestine using a mouse model for acute Salmonella Typhimurium enterocolitis. Mutations affecting flagellar assembly (Fla(-)) and chemotaxis (Che(-)) impaired the pathogen's fitness in the inflamed intestine, but not in the normal gut. This was attributable to a localized source of high-energy nutrients (e.g. galactose-containing glyco-conjugates, mucin) released as an element of the mucosal defence. Motility allows Salmonella Typhimurium to benefit from these nutrients and utilize them for enhanced growth. Thus, nutrient availability contributes to enhanced pathogen growth in the inflamed intestine. Strategies interfering with bacterial motility or nutrient availability might offer starting points for therapeutic approaches.