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Role of CX_3CR1^+ cell in the protection of the intestinal mucosa

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During infection intestinal $CX3CR1^+$ cells can either extend transepithelial cellular processes to sample luminal bacteria or, very early after infection migrate into the intestinal lumen to capture bacteria. However, up to date, the biological relevance of the intraluminal migration of $CX3CR1^+$ cells remained to be determined. We addressed this by using a combination of mouse strains differing in their ability to carry out CX3CR1-mediated sampling and intraluminal migration. We observed that, the number of S. Typhimurium traversing the epithelium did not differ between sampling-competent/migration-competent C57BL/6 and sampling-deficient/migration-competent Balb/c mice. By contrast, in sampling-deficient/migration-deficient $CX3CR1^{-/-}$ mice the numbers of S. Typhimurium penetrating the epithelium were significantly higher. However, in these mice the number of invading S. Typhimurium was significantly reduced after the adoptive transfer of $CX3CR1^+$ cells directly into the intestinal lumen, consistent with intraluminal $CX3CR1^+$ cells preventing S. Typhimurium from infecting the host. This interpretation was also supported by a higher bacterial faecal load in $CX3CR1^{+/gfp}$ compared to $CX3CR1^{gfp/gfp}$ mice following oral infection. Furthermore, by using real time in vivo imaging we observed that $CX3CR1^+$ cells migrated into the lumen moving through paracellular channels within the epithelium. Also, we reported that the absence of CX3CR1-mediated sampling did not affect antibody responses to a non-invasive S. Typhimurium strain that specifically targeted the CX3CR1-mediated entry route. These data showed that the rapidly deployed $CX3CR1^+$ cell-based mechanism of immune-exclusion is a defence mechanism against pathogens that complements the mucous and secretory (s)IgA antibody-mediated system in the protection of intestinal mucosal surface.