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IMMUNOLOGY

Gut migration of innate lymphoid cells

Homing of innate lymphoid cell (ILC) subsets ILC1 and ILC3 to barrier tissues such as the gut has been shown to be dependent on retinoic-acid-induced chemokine and integrin receptor expression. Findings also show that homing of ILC2 is different, as bone-marrow-derived ILC2 progenitors already express gut-homing receptors.

The researchers found that chemokine receptor (CCR)7, CCR9 and α4β7 integrin were expressed in ILCs residing in gastrointestinal tissues and the number of ILCs decreased in the mesenteric lymph nodes, small intestine and colon if these receptors were knocked down in mice.

Retinoic acid is known to induce the expression of receptors required for T-cell migration to gut tissues and so the authors decided to test whether it was also relevant for ILC migration. ILCs were exposed to retinoic acid *in vitro* for 4 days. CCR9 and α4β7 integrin expression was induced and CCR7 suppressed in ILC1 and ILC3, but did not affect receptor expression in ILC2. Vitamin A deficiency in mice was found to impair ILC1 and ILC3 migration to the gut, which also hampered ILC3-related immunity against *Citrobacter rodentium*.

"ILC1 and ILC3 have migration behaviours similar to T cells whereas ILC2 are very different from T cells," remarks corresponding author Chang Kim. ILC2 progenitor cells were found to already express gut-homing receptors and vitamin A deficiency in mice did not affect their ability to migrate to gut tissues.

"Tweaking the migration of ILCs to control their activity and treat IBD and infection in the gut will be interesting future work to undertake," concludes Kim.

Gillian Patman

Original article Kim M. H. *et al.* Retinoic acid differentially regulates the migration of innate lymphoid cell subsets to the gut. *Immunity* doi:10.1016/j.immuni.2015.06.009