NATURE | **LETTER**

An enteric virus can replace the beneficial function of commensal bacteria

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Nature (2014)doi:10.1038/nature13960
Received 05 June 2014
Accepted 13 October 2014
Published online 19 November 2014

Intestinal microbial communities have profound effects on host physiology[1]. Whereas the symbiotic contribution of commensal bacteria is well established, the role of eukaryotic viruses that are present in the gastrointestinal tract under homeostatic conditions is undefined[2, 3]. Here we demonstrate that a common enteric RNA virus can replace the beneficial function of commensal bacteria in the intestine. Murine norovirus (MNV) infection of germ-free or antibiotic-treated mice restored intestinal morphology and lymphocyte function without inducing overt inflammation and disease. The presence of MNV also suppressed an expansion of group 2 innate lymphoid cells observed in the absence of bacteria, and induced transcriptional changes in the intestine associated with immune development and type I interferon (IFN) signalling. Consistent with this observation, the IFN-α receptor was essential for the ability of MNV to compensate for bacterial depletion. Importantly, MNV infection offset the deleterious effect of treatment with antibiotics in models of intestinal injury and pathogenic bacterial infection. These data indicate that eukaryotic viruses have the capacity to support intestinal homeostasis and shape mucosal immunity, similarly to commensal bacteria.