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Recent studies have shown that cytokine IL-6 secreted from monocytes/macrophages promotes acute enteritis by enhancing T-cell function. We have recently extended the preliminary data to show that EGC also are hyperresponsive to enteric stimuli in vitro and that they are a major source of IL-6 in the post-inflamed intestine in vivo. We propose that EGC that become hyperresponsive to enteric stimuli in the post-inflamed intestine contribute to the acute enteritis by enhancing the activation of monocytes/macrophages and promoting the production of proinflammatory cytokines. We propose to define the molecular mechanisms that drive EGC hyperresponsiveness to enteric stimuli by systematically assessing EGC phenotypic, morphological and transcriptional changes after enteric stimulation in vitro and in vivo. Preliminary data show that EGC express the translational regulator LIN28, an important effector of TLR induced proinflammatory responses in macrophages. LIN28 may directly impact

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