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Abrogation of experimental colitis correlates with increased apoptosis in mice deficient for CD44 variant exon 7 (CD44v7)

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Experimental colitis in mice is characterized by infiltration of activated T helper (Th) cells and macrophages into the lamina propria. Particularly, these cells expressed CD44 variant exon 7 (CD44v7)-containing isoforms. Upregulation of CD44v7 isoforms was induced by CD40 ligation, an inflammation-driving interaction between activated Th cells and macrophages. To define the role of CD44v7 in colitis, mice bearing a targeted deletion for exon v7 were generated. In trinitrobenzene sulfonic acid-induced colitis, wild-type mice developed severe signs of persistent inflammation. Mice lacking CD44v7 initially showed unspecific inflammation, then recovered completely. The pathogenic origin was shown to reside in bone marrow-derived CD44v7(+) cells, because adoptive transfer experiments demonstrated an absolute requirement for CD44v7 on hematopoietic cells for maintenance of colitis. Interleukin (IL)-10-deficient mice, which develop a chronic Th1-driven enterocolitis, were crossbred with CD44v6/v7 null mice. In IL-10 x CD44v6/v7 double deficient mice, intestinal inflammation developed only weakly and at an older age. Analysis of cell death in the inflamed lesions revealed that mononuclear cells in the CD44v7 null infiltrates had higher rates of apoptosis than those from wild-type mice. Thus, the region encoded by CD44v7 appears to be essential for survival of effector lymphocytes, resulting in persistence of inflammation.

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