Response

Is CD103 a good surface marker for in vivo–activated effector/memory Treg cells in patients with chronic inflammation? Unknown yet

We observed that a percentage of CD103+ cells among CD4+FoxP3+ regulatory T (Treg) cells of donor DBA/2 mice was less than 10% before transplantation, and the percentage of CD103+ cells among donor-type CD4+Foxp3+ Treg cells increased to more than 55% in BALB/c recipients with chronic graft-versus-host disease (GVHD) and 70% in recipients without chronic GVHD after hematopoietic cell transplantation (HCT). We also observed that the percentage of CD103+ cells among Foxp3+CD4+ Treg cells in donor C57BL/6 mice was approximately 20% (mean ± SE: 20.7 ± 1.7%, N = 4) before HCT, and the percentage of CD103+ Treg cells increased to around 40% (mean ± SE: 44.3 ± 2.0%, N = 4) in BALB/c recipients with acute GVHD. These results indicate that inflammation, especially chronic inflammation, can augment the expression of CD103 on mouse Foxp3+ Treg cells, although the percentage of CD103+ cells among Foxp3+ Treg cells can differ up to certain levels in different mouse strains or different housing environments, because the percentage of CD103+ Treg cells in our DBA/2 mice is around 10%, but the percentage of our C57BL/6 mice is around 20%; the percentage of CD103+ Treg cells in our C57BL/6 mice is approximately 20%, but the percentage in C57BL/6 mice in Hu¨hn’s reports is approximately 30%.2

It has been well documented that the percentage of CD103+ cells among Foxp3+CD4+ Treg cells in healthy humans is low, around 5%.3,4 However, it is still unknown whether Foxp3+ Treg cells in human HCT recipients express CD103. It has been shown that the percentage of CD103+ cells among CD25+CD4+ T cells was significantly higher than that among CD25--CD4+ T cells in patients with multiple sclerosis.5 In addition, in vitro TGF-beta–induced Treg cells were all CD103+.7 TGF-beta secretion is usually up-regulated in patients with chronic inflammation.8 Therefore, although most of the Foxp3+ Treg cells in normal humans do not express CD103, we cannot exclude the possibility that Foxp3+ Treg cells in patients with chronic inflammation (such as chronic GVHD) will up-regulate CD103 expression. Because it has been reported that effector/memory Foxp3+ Treg cells express CCR6 in mice9 and they express CD39 in multiple sclerosis patients,10 comparison of expression of CD103, CCR6, and CD39 by Foxp3+ Treg cells in patients with chronic inflammation is required to find out which one is best for identifying the Foxp3 Treg cells.

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Acknowledgments: We thank Dr Jochen Hu¨hn at Experimentelle Rheumatologie, Charite Universita¨tsemedizin Berlin for his in-depth discussion, and Dr M. Kleineuwiefeld and colleagues for their comments on differential surface expression of CD103 by murine and human Foxp3+ Treg cells.

This work was supported by a grant from the National Institutes of Health (Bethesda, MD; R01 AI066008 to D. Zeng). Approval for this study was obtained from the City of Hope National Medical Center Institutional Review Board.

Conflict-of-interest disclosure: The authors declare no competing financial interests.

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