## Voracious monocytes

**Immature monocytes develop in the bone marrow, express Gr-1$^{hi}$ and are unable to phagocytose antigens. In the *Journal of Experimental Medicine*, Randolph and colleagues challenge that view. Using fluorescent latex beads, they show that bone marrow–resident Gr-1$^{hi}$ cells acquire antigen indirectly by phagocytosing neutrophils and B cells that acquire the beads in the periphery and then migrate to the bone marrow. After leaving the bone marrow, Gr-1$^{hi}$ monocytes mature to become macrophages and dendritic cells (DCs) and only then present the retained antigens to lymphocytes. These data have implications for how antigen is presented during infection with intracellular bacteria such as listeria, which are known to infect the bone marrow niche.** ***DCB***

***J. Exp. Med.* (22 February 2006) doi:10.1084/jem.20052119**

## Visualizing stem cells

Hematopoietic stem cells (HSCs) have not been visualized *in vivo* because of difficulties of identifying true populations. In the *Proceedings of the National Academy of Science*, Suzuki *et al.* directly visualize HSCs in their bone marrow niche by generating 'knock-in' reporter mice in which green fluorescent protein (GFP) expression is driven by a tissue-restricted *Gata2* promoter. *Gata2*-GFP expression correlates well with phenotypic and functional attributes of HSCs. *In situ* visualization of GFP$^+$ cells in trabecular bone shows discrete GFP$^+$Sca1$^+$ cells in direct contact with osteoblasts near endosteal bone regions. Time-lapse movies show that these cells are mostly immobile in the bone marrow, in contrast to mobile GFP$^-$Sca1$^-$ cells that can be classified as early hematopoietic progenitor cells. These reporter mice not only provide insights into where HSCs reside in the bone marrow but also will be a useful tool for further analysis of HSC biology. *LAD*

*Proc. Natl. Acad. Sci. USA* **103**, 2202–2207 (2006)

## Tolerizing movement

CCR7 is essential for the migration of CD4$^+$ and CD8$^+$ single-positive thymocytes from the thymic cortex to the thymic medulla. In *Immunity*, Takahama and colleagues demonstrate that cortex-to-medulla migration is essential for the establishment of central tolerance. CCR7-deficient thymocytes accumulate in the cortex, from where they can exit the thymus in a sphingosine-1 phosphate receptor–dependent way. Although thymic egress occurs in the absence of CCR7, CCR7-deficient mice demonstrate autoimmune destruction of exocrine glands. Thymic epithelial cells expressing the autoimmune regulator transcription factor (Aire), which is essential for thymocyte exposure to peripheral tissue–restricted antigens, are localized exclusively in the medulla. After positive selection, single-positive thymocytes spend an additional 12 days in the thymus. These results suggest that this 'extended stay' functions at least in part to impose central tolerance by exposing newly generated single-positive thymocytes to peripheral tissue–restricted antigens. *CB*

*Immunity* **24**, 165–177 (2006)

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## Niche-specific memory

Central ($T_{CM}$) and effector ($T_{EM}$) memory T cell subsets were identified during investigations of splenic, lymph node and blood T cells. In the *Journal of Immunology*, Ahmed and colleagues compare gut CD8$^+$ memory T cells with recirculating $T_{CM}$ and $T_{EM}$ cells. Virus-specific gut CD8$^+$ memory T cells differ in surface phenotype, contain more granzyme B, proliferate less and produce less interleukin 2, tumor necrosis factor and interferon-γ than their $T_{CM}$ and $T_{EM}$ counterparts. These disparate characteristics of splenic and gut memory CD8$^+$ T cells are acquired gradually after virus infection but are reversible and depend on persistent localization in the spleen or the gut. These data add a new dimension to the $T_{CM}$-versus-$T_{EM}$ paradigm and suggest that niche-specific factors might ensure that memory T cells are programmed to efficiently respond to niche-specific threats. *CB*

*J. Immunol.* **176**, 2079–2083 (2006)

## NK cells *in situ*

***In vitro* studies have demonstrated functional crosstalk can occur between natural killer (NK) cells and DCs, thereby potentially influencing ensuing immune responses. Whether such interactions occur in more physiological settings remains unclear. In the *Journal of Experimental Medicine*, Germain and colleagues use intravital and confocal imaging of mouse lymph nodes to show that long-lived NK cell–DC interactions form *in situ*. NK cells are found in close proximity to paracortical and medullary DC networks in uninflamed lymph nodes. These NK cells are much less motile than T cells present in the same lymph node. NK cells are rapidly recruited from the bloodstream into draining lymph nodes after infection with *Leishmania major*. These newly immigrant NK cells localize to the paracortical T cell areas of the lymph node, where they also form intimate contacts with DCs and express interferon-γ. These imaging studies show that DC–NK cell communication networks form in physiologically relevant scenarios and affect DC-mediated T cell activation.** ***LAD***

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## Immune privilege

Immune-privileged sites such as the central nervous system (CNS) can be targets of infection and severe disease. In the *Journal of Immunology*, Bergmann and colleagues characterize the mechanism for maintaining CNS immune privilege after vaccination against neurotropic coronavirus infection. Recrudescent coronavirus replication occurs because T cell–mediated immune control is lost. Priming with virus antigen induces robust CD8$^+$ T cell memory in the CNS that effectively clears acutely replicating virus after challenge. Nevertheless, such effective T cell priming cannot prevent viral recrudescence and loss of T cell–mediated control. Virus-specific CD8$^+$ T cells become quiescent because antigen presentation by CNS-resident cells is transient, as resident glial cells are found to rapidly lose expression of both major histocompatibility complex class I and class II molecules. Thus, although vaccination substantially enhances T cell immunity, intrinsic properties of immune privilege sites can abrogate those effector functions. *DCB*

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# Erratum: Research highlight

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In the version of this research highlight initially published, the reference is missing. The reference should be as follows: *Proc. Natl. Acad. Sci. USA* **103**, 2202–2207 (2206). The error has been corrected in the HTML and PDF versions of the research highlight.