The immune system uses multiple mechanisms to maintain a relatively constant number of lymphocytes. The expansion of antigen-specific lymphocytes during the immune response to infection results in a large increase in the cellularity of the secondary lymphoid organ (1, 2), which is normally followed by activation-induced cell death (3, 4). On the other hand, T lymphocytes spontaneously divide when the hosts are lymphopenic (5–7). Lymphopenia is found in newborn animals (8) and in those exposed to chemotherapy (9) or irradiation (5–7). Because the latter event is viewed as the host attempts to restore the lymphocyte cellularity, it is often referred to as homeostatic proliferation.

Homeostatic proliferation is similar to antigen-driven proliferation in its requirement for MHC–TCR interaction (5, 10). However, these two types of T cell proliferation differ in several important ways. First, homeostatic proliferation is polyclonal and results in the preservation of the TCR repertoire (11–13), whereas antigen-driven proliferation results in the clonal expansion of T cells that are specific for the antigens involved (1, 2). Second, homeostatic proliferation and antigen-driven proliferation use distinct costimulatory pathways. For instance, although B7–CD28 interaction has a major impact on antigen-driven proliferation (14–16), it is dispensable for homeostatic proliferation (17). Likewise, CD40–CD40L and 4-1BB–4-1BBL interactions are also not required for homeostatic proliferation (17). On the other hand, we have recently reported that CD24 expression on T cells is essential for homeostatic proliferation (18), although the targeted mutation of CD24 did not impair T cell priming in the lymphoid organ (19, 20). Thus, a lack of CD24 in the non-T host cells bypassed the requirement for T cell expression of CD24 in homeostatic proliferation in the WT host. Our data demonstrate that CD24 expressed on the DCs limits T cell response to homeostatic cue and prevents fatal damage associated with uncontrolled homeostatic proliferation.

In response to a lymphopenic cue, T lymphocytes undergo a slow-paced homeostatic proliferation in an attempt to restore T cell cellularity. The molecular interaction that maintains the pace of homeostatic proliferation is unknown. In this study, we report that in lymphopenic CD24-deficient mice, T cells launch a massive proliferation that results in the rapid death of the recipient mice. The dividing T cells have phenotypes similar to those activated by cognate antigens. The rapid homeostatic proliferation is caused by a lack of CD24 on dendritic cells (DCs). Interestingly, although CD24 expression in T cells is required for optimal homeostatic proliferation in the wild-type (WT) host, mice lacking CD24 on all cell types still mount higher homeostatic proliferation than the WT mice. Thus, a lack of CD24 in the non-T host cells bypassed the requirement for T cell expression of CD24 in homeostatic proliferation in the WT host. Our data demonstrate that CD24 expressed on the DCs limits T cell response to homeostatic cue and prevents fatal damage associated with uncontrolled homeostatic proliferation.

The online version of this article contains supplemental material.

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The online version of this article contains supplemental material.
signal. In this study, we report a serendipitous observation that in the lymphopenic CD24-deficient host, the T cells undergo massive homeostatic proliferation, leading to the rapid death of the recipients. The uncontrolled proliferation is caused by the superior stimulatory activity of DCs generated from the CD24-deficient mice. Our results reveal a vital inhibitory checkpoint that controls the pace of homeostatic proliferation.

RESULTS
Uncontrolled homeostatic proliferation of syngeneic T cells in the lymphopenic CD24-deficient hosts
To study the contribution of CD24 on the host APC to homeostatic proliferation, we transferred CFSE-labeled naive T cells from Thy1.1 congenic mice into sublethally irradiated wild-type (WT) B6 mice and CD24−/− B6 mice. The spleens were harvested from the recipients 4 d after adoptive transfer. Cell division was measured by CFSE dilution by flow cytometry. As shown in Fig. 1 a, the transferred CD4 T cells from the WT B6 recipients underwent twice divisions. At the same time, CD8 T cells divided four times. On the other hand, both populations of the transferred T cells in the CD24−/− mice underwent much more accelerated (more than seven) divisions. We then performed a kinetic study to substantiate this surprising observation. On day 1, the T cells in both recipients maintained the same level of CFSE fluorescence density (Fig. 1 b). However, on day 2, when little change happened to the transferred T cells in the WT B6 recipients, the T cells in CD24−/− recipients already started dividing. By day 3, in WT B6 mice, when T cells barely divided, a substantial fraction of T cells had already divided five times in CD24−/− recipients. Thus, although the same amount of naive T cells was transferred into both groups, T cells started to divide much earlier and faster in the CD24−/− recipients. Nevertheless, we have not observed an increased accumulation of T cells in the CD24-deficient host (Fig. S1, available at http://www.jem.org/cgi/content/full/jem.20052293/DC1).

Although these studies raised the intriguing possibility that CD24 on the host cells may serve as a key regulator for the rate of homeostatic proliferation, several caveats deserve consideration. First, despite the fact that the CD24−/− mice were produced with embryonic stem cells from C57BL/6 mice (23), it remains possible that the two hosts are not histocompatible. To test this possibility, we transplanted the skin grafts from the CD24−/− mice into CD24−/− mice specifically affects homeostatic proliferation, we labeled CD24−/− recipients with CD4−/− CD8−/− mice. Thus, the results demonstrate that in the absence of lymphopenic cue, CD24 deficiency in the host cells was unable to induce the proliferation of T cells from congenic mice.

Our extensive analyses have demonstrated that CD24 deficiency does not substantially impact the priming of T cells (19, 20, 24). To test whether CD24 deficiency in the host cells specifically affects homeostatic proliferation, we labeled
OT-1 T cells with CFSE and injected them into the host that had been immunized with the specific ovalbumin peptide. As shown in Fig. 2, essentially identical proliferation was observed in the WT and CD24-deficient mice. Again, the OT-1 cells did not proliferate in unimmunized mice. Collectively, our data demonstrate that CD24 deficiency in the host selectively enhanced homeostatic T cell proliferation. Therefore, CD24 on the APC served as a suppressor of homeostatic T cell proliferation.

In the CD24-deficient mice, cells undergoing homeostatic proliferation displayed markers of T cells induced by cognate antigen

Previous studies have demonstrated that T cells undergoing homeostatic proliferation have the markers of central memory cells; namely, they express high levels of CD44 but generally fail to down-regulate CD62L (6, 7). Moreover, T cells that underwent antigen-driven proliferation expressed high levels of CD25 and CD69, whereas those that underwent homeostatic proliferation under normal circumstances lacked these two markers (25). As shown in Fig. 3 a, few cells isolated from WT recipients expressed CD25. However, close to 50% of T cells that were recovered from the CD24−/− host expressed CD25. Regardless of the CD24 status in the host, T cells acquired memory cell phenotype with a high level of CD44 expression (Fig. 3 b, top), which is consistent with previous studies (6, 7). However, the majority of T cells from CD24−/− recipients but not those from the WT mice had down-regulated 62L (Fig. 3 c). The down-regulation of CD62L was more pronounced at 2 wk after adoptive transfer, when an overwhelming majority of activated T cells displaced a CD44hiCD62L− phenotype. Thus, removing CD24 from the lymphopenic host converts a weak homeostatic cue into a strong stimulation similar to antigen-driven proliferation.

Superior stimulatory activity of the CD24−/− DCs

CD24 is expressed in both hematopoietic and nonhematopoietic cells (26). To determine whether CD24 expressed on

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**Figure 2.** CD24 deficiency does not facilitate antigen-induced proliferation of OT-1 cells. Purified OT-1 cells (10⁶/mouse) were adoptively transferred into either naive or immunized mice 1 d after immunization with 10 μg SIINFEKL peptide in CFA. 3 d after adoptive transfer, the spleen cells and lymph node cells were harvested for flow cytometry. The transgenic T cells were enriched by gating on Vβ5+ and Vα2+ CD8 T cells. Data shown are histograms depicting the distribution of CFSE intensity among the Vα2+Vβ5+CD8+ T cells and are representative of two independent experiments. The CFSE− cells are of donor origin. Similar patterns were observed in six WT and four CD24−/− recipients in which the donor cells could be clearly identified.

**Figure 3.** Distinct phenotypes of T cells undergoing homeostatic proliferation in WT and CD24−/− hosts. (a) Expression of CD25 markers in the WT (top) and CD24-deficient host (bottom). (b and c) CD44 and CD62L expression on cells undergoing homeostatic proliferation. Data shown are FACS profiles of donor cells at days 4 (b) and 14 (c) after adoptive transfer. Numbers in the quadrants indicate percentages of cells. The cell surface phenotypes have been reproduced in >10 experiments.
the hematopoietic cells is responsible for pacing homeostatic proliferation, we transferred WT and CD24−/− bone marrow into lethally irradiated CD24−/− mice. After 4 wk, we transferred CFSE-labeled purified naive T cells from Thy1.1 congenic mice into both recipients, which had been sublethally irradiated right before T cell transfer. We harvested spleens from both groups and analyzed CFSE dilution in the donor T cells (Fig. 4a). As shown in Fig. 4b, in the CD24−/− bone marrow–reconstituted recipients, both CD4 and CD8 T cells had undergone massive proliferation over the 4-d period as expected. In contrast, much less proliferation was observed in the chimera mice reconstituted with WT bone marrow cells. Thus, the expression of CD24 on bone marrow–derived cells is both necessary and sufficient to control the pace of homeostatic proliferation.

It has been established that both mouse and human DCs can induce T cell homeostatic proliferation in vitro when the DC number is substantially higher than T cells (27). To determine whether a lack of CD24 on the DCs promotes their ability to induce homeostatic proliferation, we generated DCs from both WT and CD24−/− bone marrow and cultured CFSE-labeled purified naive Thy1.1 congenic T cells with the DCs at a suboptimal 1:2 ratio. After 5 d, we analyzed the CFSE dilution to determine T cell proliferation. As shown in Fig. 4c, T cells cultured with WT B6 bone marrow–derived DCs had barely divided, whereas those cocultured with CD24−/− DCs divided more than one to five times. Thus, CD24-deficient DCs had a superior stimulatory activity for homeostatic T cell proliferation. Careful analysis of the expression of MHC and costimulatory molecules in the two populations of DCs revealed little difference (Fig. S2, available at http://www.jem.org/cgi/content/full/jem.20052293/DC1). In contrast, the coculture of syngeneic macrophages (Fig. 4d) and B cells (Fig. 4e) with naive T cells failed to induce proliferation regardless of whether or not the CD24 gene had been inactivated.

To confirm that CD24 deficiency is sufficient to confer superior homeostatic proliferation in vivo, we adoptively transferred naive T cells together with WT and CD24−/− DCs into lymphopenic WT hosts and monitored T cell proliferation 4 d later. As shown in Fig. 4f, the injection of CD24−/− but not WT DCs led to the strong homeostatic proliferation of T cells. Collectively, our data demonstrate that CD24 expressed on DCs controls the pace of homeostatic proliferation in lymphopenic hosts.

**Fatal destruction of the CD24-deficient host associated with mass T cell proliferation**

Surprisingly, ~60% of the CD24−/− recipients died within 2 wk after T cell transfer, whereas all of the WT recipients remained healthy (Fig. 5). The death was caused by adoptively transferred T cells, as the control mice that received no T cells were healthy. We have systematically examined the host for histological signs of acute graft versus host diseases. As shown in Fig. S3 (available at http://www.jem.org/cgi/content/full/jem.20052293/DC1), no inflammation was observed in the gut or other organs of the CD24-deficient host. Thus, the destruction of the recipient is pathologically distinct from graft versus host diseases. Histology of the spleen suggests extensive hemolysis in the red blood cells in the moribund mice (unpublished data). The high fatality and rapid onset demonstrate the vital role of CD24-mediated regulation of homeostatic proliferation.
Our data demonstrate that WT T cells adoptively transferred into CD24-deficient mice underwent massive homeostatic proliferation that ultimately led to the death of the majority of the recipients. The death was associated with massive T cell activation but not infiltrations of organs, which is inconsistent with the activation of organ-specific T cells. Although this may be associated with so-called “cytokine storm,” our analysis did not reveal an obvious rise in the percentage of T cells capable of producing TNF-α, a cytokine frequently associated with cytokine storm (Fig. S4, available at http://www.jem.org/cgi/content/full/jem.20052293/DC1). Therefore, the death was unlikely caused by excessive TNF production. Histological analysis of the spleen revealed extensive hemosiderin deposits in red pulps, which is indicative of a cytokine storm, our analysis did not reveal an obvious rise in the percentage of T cells capable of producing TNF-α, a cytokine frequently associated with cytokine storm (Fig. S4, available at http://www.jem.org/cgi/content/full/jem.20052293/DC1). Therefore, the death was unlikely caused by excessive TNF production. Histological analysis of the spleen revealed extensive hemosiderin deposits in red pulps, which is indicative of a cytokine storm, our analysis did not reveal an obvious rise in the percentage of T cells capable of producing TNF-α, a cytokine frequently associated with cytokine storm (Fig. S4, available at http://www.jem.org/cgi/content/full/jem.20052293/DC1). Therefore, the death was unlikely caused by excessive TNF production. Histological analysis of the spleen revealed extensive hemosiderin deposits in red pulps, which is indicative of a cytokine storm.
of hemolysis in moribund mice (unpublished data), although its cause and significance remains to be determined.

In addition to the adoptive transfer model, we also observed a substantially increased homeostatic proliferation in CD24-deficient mice after CD4 and CD8 T cells were deleted by a single injection of a low dose of anti-CD4 + anti-CD8 antibodies. These results are noteworthy for three reasons. First, because this assay involved no adoptive transfer, it helped to further rule out the possibility that increased proliferation was caused by histoincompatibility between donor and recipient cells even though the CD24-deficient mice were produced in embryonic stem cells derived from C57BL/6 mice. Second, because the hosts were not irradiated, the increased proliferation was not associated with changes in the irradiated recipient. Third, although CD24 expression in T cells was essential for optimal homeostatic proliferation in the WT host, such a requirement can be bypassed by a lack of CD24 on host APCs. Thus, negative regulation mediated by CD24 on APCs plays a dominant role over the positive regulatory role for CD24 on T cells.

We have presented several lines of evidence that the lack of CD24 in the DCs explains the massive homeostatic proliferation. Thus, the CD24-mediated suppression of homeostatic proliferation is mediated by a lack of CD24 on host APCs. We have demonstrated that a targeted mutation of CD24 exacerbated immune deficiency associated with the targeted mutation of CD28 (19, 20), which revealed CD24 as a redundant costimulatory molecule for antigen-driven proliferation. In contrast, CD24−/− DCs have a drastically stronger activity in inducing homeostatic proliferation. The apparently opposite function of CD24 in the two processes highlights the distinct requirements of T cell response to antigen and homeostatic cue. In this context, it is worth noting that anti-CD24 antibody 20C9, which blocks the costimulatory function of CD24, has no effect on the DC-mediated costimulatory function (unpublished data). It is likely that distinct structures on CD24 are involved in T cell costimulation and in homeostatic proliferation.

CD24 is a highly glycosylated cell surface molecule that is anchored through a glycosylphosphatidylinositol tail. Mouse CD24 consists of 27 amino acids that include eight potential O-linked glycosylation sites and three potential N-linked glycosylation sites (30). The heterogeneity of CD24, which is presumably caused by differential glycosylation, has made it difficult to identify the counter receptor involved in pacing homeostatic proliferation. In spite of this, our work provides the first evidence that the pace of T cell homeostatic proliferation is actively maintained by CD24 expressed on host APCs to avoid deleterious consequences associated with the uncontrolled polyclonal activation of T cells.

MATERIALS AND METHODS

Mice and antibodies. C57BL/6/J and BALB/c mice were purchased from Charles River Laboratories through a contract with the National Cancer Institute. Transgenic mice OT-1 that express TCR specific for ovalbumin peptide SIINFEKL and B6.Thy1.1 mice were purchased from The Jackson Laboratory. Mice with the targeted mutation of CD24 were produced using embryonic stem cells from C57BL/6 mice as described previously (23). All of the mice were maintained under specific pathogen-free conditions in the animal facility at the Ohio State University. All studies involving animals...
were approved by the Ohio State University Institutional Laboratory Animal Care and Use Committee.

All conjugated antibodies used were purchased from either eBioscience (Thy1.1, CD80, CD86, CD44, and CD62L) or BD Biosciences (CD4, CD8, H-2K^d, I-A^b, CD25, CD40, Annexin V, IFN-γ, TNF-α, and isotype control).

**Analysis of T cell division in vivo.** T lymphocytes were purified from pooled spleen and lymph node cells by negative selection. In brief, pools of spleen and lymph node cells were incubated with a cocktail of antibodies specific for CD11b (Mac-1), Fc receptor (2.4G2), B220, and CD11c. The Dynal beads coated with goat anti-rat IgG were used to negatively select T cells. The purity of the cells was checked by flow cytometry to be >95%. The purified T cells congenic in the Thy1 locus (B6 and Thy1.1*) were labeled with CFSE and injected intravenously into irradiated (600 R) recipient mice that were WT or CD24^−/− (B6 and Thy1.2*) at a dose of 5 × 10^6/mouse. At given times after adoptive transfer, spleen cells were harvested and analyzed for the intensity of CFSE dye and other cell surface markers.

**Bone marrow reconstitution.** Bone marrow cells from WT B6 mice and CD24^−/− mice were transferred back to 1,000 rad-irradiated CD24^−/− mice. 4 wk later, CD24 expression was detected in both groups to determine the reconstitution efficiency.

**DCs cultured from bone marrow.** Bone marrow cells from WT B6 mice or CD24^−/− mice were cultured with 10% RPMI and recombinant mouse GM-CSF as described previously (31). LPS was added into the culture after day 10 at a final concentration of 1 μg/ml for 2 d. The nonadherent cells harvested from the culture were mature DCs.

**T cell–DC coculture.** For suspension cultures, CFSE-labeled Thy1.1^+ T cells (usually 500,000/well) were mixed with given numbers of matured DCs in 24-well plates in RPMI 1640 medium. Cells were cultured at 37°C in a humidified 5% CO2 incubator for 5 d before analysis.

**T cell depletion and recovery in vivo.** WT or CD24^−/− mice were injected with 100 μg anti-CD4 (GK1.5) and 100 μg anti-CD8 (2.4.3) antibody. The mice were bled, and percentages of CD4 and CD8 T cells in the peripheral blood were determined by flow cytometry. 7 d after the antibody injection, some of the mice were given three injections of BrdU (1 mg/injection i.p.) with 12-h intervals. 12 h after the last BrdU injection, spleen cells were harvested for flow cytometric analysis.

**Flow cytometry.** The cell surface markers, including CD4, CD8, CD24, CD25, CD44, CD62L, and Thy1.1, were analyzed by three- or four-color flow cytometry using fluorochrome-conjugated monoclonal antibodies purchased from BD Biosciences. To assess intracellular cytokine production, spleens were harvested. The splenocytes were stimulated with PMA for 4 h and stained for cell surface markers CD4 and CD8 followed by intracellular staining for IFN-γ/TNF-α and/or isotype control using the CytoFix/ CytoPerm kit (BD Biosciences).

**TREC assay for recent thymic emigrants.** Copy numbers of the γTREC were used to determine the frequency of recent thymic emigrant (26). In brief, total DNA was purified from total splenocytes with the DNA Easy kit (QIAGEN). Real-time PCR was performed using the Quantitative PCR kit (Invitrogen). The primers used were as follows: mRrec:primers 5′-GGGACACAGCAGCGTGTG-3′, δRrec: primer 5′-GCGAGTTTTTGTAAAGGTGCTCA-3′, mRrec-ψREN:forward probe 5′-FAM-CACAAAGCAGCTGACCCGTGCA-TAMRA-3′, and CD8β forward primer 5′-CAGGACCCCGAAGCAAGACT-3′, reverse primer 5′-CAGATTTCCACATACAAATACTTGTTG-3′, and probe 5′-FAM-TGATTTCTGAGCTGGTTCT-TAMRA-3′. The number of TREC/million T cells was calculated by: 2 × (number of TREC/number of CD8β × percentage of T cells) × 10^6.

**Online supplemental material.** Fig. S1 shows that despite a major difference in the rate of homeostatic proliferation, the numbers of donor T cells were comparable in WT and CD24^−/− recipients. Fig. S2 shows that CD24 deficiency does not alter the expression of MHC and costimulatory molecules on DCs. Fig. S3 shows the histology analysis of healthy WT mice and moribund CD24-deficient mice. No inflammation was observed in the moribund mice. Fig. S4 shows the high and comparable percentages of TNF-α and IFN-γ-producing cells in WT and CD24^−/− recipients. Online supplemental material is available at http://www.jem.org/cgi/content/full/jem.20052293/DC1.

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