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Journal of Experimental Medicine (*JEM*) is pleased to present a special collection of recent and exciting advances in immunology, celebrating *JEM*'s long commitment to publishing outstanding basic as well as translational research. The collection covers *JEM*'s broad scope, from immunodeficiencies leading to inflammatory diseases, persistent IFNγ-induced memory in human macrophages, immunology at mucosal sites from nasal-associated lymphoid tissue to the intestine, transcriptomic control of $CD8^+$ T cell responses in cancer, novel regulators of plasma cell differentiation, fibrosis, and central tolerance maintenance. If you enjoy this collection, we encourage you to scan the QR codes to view the articles online and sign up for email alerts to receive the latest research. If you are interested in submitting work in this field to *JEM*, we encourage you to contact our Editorial office via jem@rockefeller.edu or +1 212-327-8575.

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On the cover: The subepithelial dome of nasal-associated lymphoid tissue of a CX_3CR1-GFP mouse adoptively transferred with $Rosa26^{tdTomato/+}$ $B1\text{-}8^{hi}$ B cells and $CD45.1^+$ OT-II T cells, imaged using two-photon laser scanning microscopy 5 days after intranasal NP-OVA + MPLA immunization. Collagen is labeled blue. Image © 2026 Liu et al.
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See page 5.

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Activated PI3Kδ rewires Th2 differentiation

While inputs regulating CD4⁺ T helper (Th) cell differentiation are well defined, the integration of downstream signaling with transcriptional and epigenetic programs that define Th lineage identity remains incompletely resolved.

PI3K signaling is a critical regulator of T cell function; activating mutations affecting PI3Kδ result in an immunodeficiency with multiple T cell defects. Using mice expressing activated PI3Kδ, we found aberrant expression of proinflammatory Th1 signature genes under Th2-inducing conditions, both *in vivo* and *in vitro*. This dysregulation was driven by a PI3Kδ-IL-2-Foxo1 signaling amplification loop, fueling Foxo1 inactivation, loss of Th2 lineage restriction, and extensive epigenetic reprogramming.

Surprisingly, ablation of *Fasl*, a Foxo1-repressed gene, normalized both Th2 differentiation and TCR signaling. BioID and imaging revealed Fas interactions with TCR signaling components, which were supported by Fas-mediated potentiation of TCR signaling that could occur in the absence of FADD.

Our results highlight Fas-FasL signaling as a critical intermediate in phenotypes driven by activated PI3Kδ, thereby linking two key pathways of immune dysregulation.

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Photo credit: R. Germain

ORIGINAL PAPER

Golec, D.P., P.H. Gazzinelli-Guimaraes, D. Chauss, K. Yu, H. Nagashima, A.C. Cruz, T. Hill, S. Ganesan, J.L. Cannons, J.K. Perry, L. Nivelo, I. Joshi, N. Pereira, F.M.S. Oliveira, Y. Zheng, M. Jean Pierre, K.M. Druey, J.B. Lack, E.V. Dang, T.B. Nutman, A.V. Villarino, J.J. O'Shea, B. Afzali, and P.L. Schwartzberg. 2026. A PI3Kδ-Foxo1-FasL signaling amplification loop rewires CD4⁺ T cell signaling and differentiation. *J. Exp. Med.* 223 (4): e20252154. https://doi.org/10.1084/jem.20252154

Trapped IFNγ provides memory to human macrophages

Macrophages, as key sentinel cells of the innate immune system, can retain memory of prior stimulus exposure. IFNγ plays a central role in maintaining trained immunity *in vivo* and induces potent innate immune training of macrophages. Such training is associated with the formation of *de novo* enhancers that alter gene expression responses to subsequent stimuli. However, how such enhancers are maintained after cytokine exposure remains unclear.

We report that the mechanism underlying durable IFNγ-induced enhancers is not cell intrinsic. IFNγ-exposed macrophages continue to exhibit JAK/STAT signaling days after cytokine removal. Blocking IFNγ signaling with a JAK inhibitor or anti-IFNγ neutralizing antibodies after cytokine removal is sufficient to reverse IFNγ-induced enhancers and erase the potentiated state of the treated macrophages.

Our findings suggest that epigenetic changes in macrophages do not inherently encode innate immune memory or a "potentiated" macrophage state, but in fact are themselves dependent on ongoing signaling from cytokines sequestered at the cell surface. Thus innate immune memory, in at least some physiological scenarios, may be better thought of as a tissue-emergent property than a cell-intrinsic epigenomic property.

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ORIGINAL PAPER

Gorin, A., S. Niu, N. Harriott, V. Koduvayur, Q.J. Cheng, and A. Hoffmann. 2026. IFNγ-induced memory in human macrophages is sustained by the durability of cytokine signaling itself. *J. Exp. Med.* 223 (4): e20250976. https://doi.org/10.1084/jem.20250976

CCR6 and BCR affinity regulate nasal IgA responses

Antibody-mediated immune responses in mucosal tissues are critical for defending against pathogens while maintaining homeostasis with commensals. Nasal vaccination aims to induce local protection in the upper airway mucosa. Although B cell–driven immunity is well characterized in gut-associated lymphoid tissues such as Peyer's patches and mesenteric lymph nodes, much less is known about analogous processes in the upper airways.

We show that B cell receptor (BCR) affinity and CCR6 regulate germinal center (GC) seeding and class-switch recombination (CSR) to IgA in nasal-associated lymphoid tissue (NALT) following nasal vaccination. B cells bearing low-affinity BCRs failed to upregulate CCR6 and did not support T follicular helper cell differentiation or seed GCs in the NALT. CCR6-deficient B cells were unable to migrate to the NALT subepithelial dome or undergo IgA CSR and seed GC effectively in response to nasal vaccination or commensal bacteria signals.

Thus, effective targeting of B cell clones to induce CCR6 expression is essential for nasal vaccine design.

ORIGINAL PAPER
Liu, J., L. Stoler-Barak, and Z. Shulman. 2026. Nasal germinal centers and IgA class-switch recombination depend on CCR6 and B cell receptor affinity. *J. Exp. Med.* 223 (4): e20251901. https://doi.org/10.1084/jem.20251901

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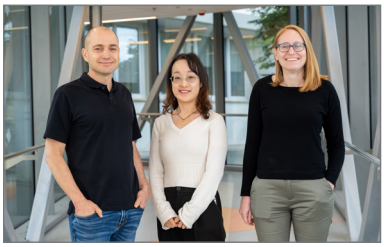

Left to right: Shulman, Liu, Stoler-Barak

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Zbtb32 enhances CD8 T cell antitumor responses

In the tumor microenvironment (TME), "exhausted" CD8⁺ T cells are classified into progenitor (Tpex) and terminally exhausted (Ttex) populations. Tpex cells, critically regulated by zinc finger and BTB domain containing 27 (Zbtb27)/Bcl6 transcription factor, could be reinvigorated during immune checkpoint blockade (ICB) therapy, while Ttex cells, characterized by stronger proliferation and cytotoxicity, play an indispensable role in tumor control. However, the mechanisms governing the differentiation into Ttex and their function remain not well understood.

In this study, we identified that Zbtb32, highly expressed in CD8⁺ Ttex subset, is crucial for CD8⁺ T cells within tumors. Zbtb32, regulated by CD28 signaling, promotes the differentiation of CD8⁺ T cells into Ttex subset, enhancing their cytotoxicity, proliferation, and anti-tumor capability. Importantly, we found a competitive DNA binding between Zbtb32 and Bcl6, especially in regulation of Id2 expression.

Thus, our findings demonstrate the pivotal role of Zbtb32 in CD8⁺ T cell anti-tumor function, with implications in cancer immunotherapy.

ORIGINAL PAPER
Pan, B., Q. Sun, R. Li, J. Feng, J. Hao, B. Xie, X. Zhao, Z. Zhao, P. Wei, Q. Lan, S. Xie, T. Xie, Y. Chen, K. Wei, X. Zhong, H. Qi, L. Ni, and C. Dong. 2026. Zbtb32 promotes CD8⁺ T cell differentiation and function in cancer. *J. Exp. Med.* 223 (4): e20250005. https://doi.org/10.1084/jem.20250005

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An *in vivo* CRISPR/Cas9 screen for B cell regulators

Immune responses to pathogens lead to the generation of plasma cells through a complex interplay of B cells with their microenvironment in lymphoid organs. To identify new regulators of B cell activation and plasmablast differentiation in the context of the splenic microenvironment, we established an *in vivo* system for pooled sgRNA CRISPR/Cas9 screens in immunized mice.

To improve the infection efficiency of naïve B cells, we generated *Cd23*-Cre *Rosa26*$^{LSL\text{-}EcoR/+}$ mice exhibiting increased expression of the ecotropic lentivirus receptor EcoR on naïve B cells. Upon adoptive B cell transfer and immunization of recipient mice, 379 sgRNAs, targeting genes with high expression in plasma cells, were analyzed for their effects on plasmablast generation. Gene hits, encoding 23 positive and 18 negative regulators of B cell activation, plasmablast differentiation, or homeostasis, were uniquely identified in these *in vivo* screens. Validated genes encoded proteins involved in cell adhesion, signal transduction, protein folding, iron transport, and enzymatic processes.

Hence, our *in vivo* screening system identified novel regulators controlling B cell–mediated immune responses.

ORIGINAL PAPER
Calderón, L., M. Schäfer, M. Rončević, R. Rauschmeier, M. Jaritz, T.A. Schwickert, Q. Sun, A. Pauli, J. Zuber, and M. Busslinger. 2026. *In vivo* CRISPR/Cas9 screens identify new regulators of B cell activation and plasma cell differentiation. *J. Exp. Med.* 223 (3): e20250594. https://doi.org/10.1084/jem.20250594

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Tet2-deficient macrophages promote liver fibrosis

Clonal hematopoiesis driven by *Tet2* deficiency in myeloid cells (*Tet2*$^{\Delta Mye}$) is prevalent in elderly individuals; however, the role of *Tet2*$^{\Delta Mye}$ in liver fibrosis pathogenesis remains elusive.

In this study, we demonstrated that *Tet2*-deficient monocyte-derived macrophages (MDMs) promoted cellular expansion and elevated C–C motif chemokine ligand 2/8 (Ccl2/8) secretion by stabilizing their mRNAs through 5-hydroxymethylcytosine-mediated alterations in RNA–protein interactions. These chemokines engaged with the upregulated C–C motif chemokine receptor (Ccr2/3) on *Tet2*$^{-/-}$ monocytes, forming a positive feedback loop that amplified pro-inflammatory MDMs (pMDMs) accumulation in liver. *Tet2*$^{-/-}$ pMDMs activated hepatic stellate cells through IL-6, driving extracellular matrix deposition and fibrotic progression.

Pharmacological inhibition of Ccl2/Ccl8 with Bindarit attenuated MDMs accumulation and liver fibrosis, whereas combined therapy with Bindarit and IL-6 neutralization synergistically suppressed liver fibrosis in *Tet2*$^{\Delta Mye}$ mice and aged chimeric models recapitulating *Tet2*$^{\Delta Mye}$-related myeloid hematopoiesis. These findings present the mechanism by which *Tet2*$^{\Delta Mye}$ aggravates liver fibrosis and highlight MDMs depletion plus IL-6 neutralization as a promising therapy for liver fibrosis in patients with *Tet2*$^{\Delta Mye}$-related myeloid hematopoiesis.

ORIGINAL PAPER
Feng, J., B. Wu, Y. Li, P. Sun, Q. Liu, Q. Xiao, J.-B. Cai, Y. Zheng, H. Chen, Y. Xu, Y. Liu, G.-M. Shi, L. Tan, and Y.G. Shi. 2026. *Tet2* deficiency–induced expansion of monocyte-derived macrophages promotes liver fibrosis. *J. Exp. Med.* 223 (2): e20251114. https://doi.org/10.1084/jem.20251114

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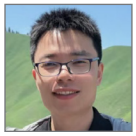

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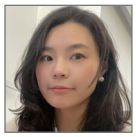

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Fibroblast diversity within human GALT

Gut-associated lymphoid tissues (GALT) represent major sites of adaptive immune priming in the intestine, yet our understanding of human GALT diversity and function remains limited.

We used single-cell RNA sequencing, flow cytometry, and confocal laser microscopy to map the fibroblast (FB) landscape of human GALT, including that of Peyer's patches (PP), mucosal isolated lymphoid follicles (M-ILF), and submucosal ILF (SM-ILF). We identify CD24 as a marker that distinguishes GALT from other intestinal FB and demonstrate that CD24$^+$ FB consist of distinct subsets that locate within discrete niches. We show that the composition and transcriptional profile of M-ILF and SM-ILF FB differs with SM-ILF FB appearing more focused at providing T cell support.

Finally, we find the transcription profile of PP T zone reticular cells to be altered in Crohn's disease and that cells with a GALT FB-like profile can be detected in other chronic inflammatory diseases. Collectively, our findings provide an important framework for understanding GALT diversity and function.

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ORIGINAL PAPER
Mörbe, U.M., F.V. Junghus, G. Nos, P.B. Jørgensen, M.J. Ensmenger, V.A. Väänänen, M.D. Wewer, G.R. Madsen, L.B. Riis, H.L. Jakobsen, L.R. Olsen, S. Brunak, O.H. Nielsen, and W.W. Agace. 2026. Fibroblast diversity within human gut-associated lymphoid tissues. *J. Exp. Med.* 223 (3): e20250471.
https://doi.org/10.1084/jem.20250471

Claudin 1 is required for central tolerance

Central tolerance, which relies on the presentation of self-antigens by medullary thymic epithelial cells (mTECs) and dendritic cells (DCs), prevents autoimmunity by eliminating self-reactive T cells. While mTECs produce self-antigens autonomously, DCs acquire them from mTECs via cooperative antigen transfer (CAT). We previously showed that mTEC and DC subsets exhibit preferential pairing in CAT, providing a rationale for the existence of molecular determinants underpinning this pairing and its outcome.

We compared the transcriptomes of CAT-experienced and CAT-inexperienced DCs and identified Claudin 1 as a molecule involved in CAT and type 1 DC (DC1) maturation. DC1-specific ablation of Claudin 1 resulted in decreased CAT to late mature DC1s and dramatically diminished DC1 maturation. These phenotypes correlated with the displacement of DC1s from mTECs and their decreased expression of MHCII pathway genes. This translated into impaired regulatory T cell selection and clonal deletion, ultimately manifesting in symptoms of multiorgan autoimmunity and shortened lifespan.

Collectively, our results identify thymic DC1-derived Claudin 1 as a regulator of immune tolerance.

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ORIGINAL PAPER
Březina, J., T. Brabec, D. Machač, M. Vobořil, O. Ballek, J. Pačes, V. Sýkora, K. Jančovičová, E. Valter, K. Kováčová, J. Manning, V. Tahtahová, A. Čepková, M. Dobešová, J. Dobeš, J. Kubovčiak, M. Kolář, P. Kašpárek, R. Sedlacek, O. Štepánek, J. Černý, S. Tsukita, B. Malissen, G. Anderson, and D. Filipp. 2026. Claudin 1–mediated positioning of DC1 to mTECs is essential for maintenance of central tolerance. *J. Exp. Med.* 223 (3): e20250970.
https://doi.org/10.1084/jem.20250970

PERSPECTIVE

Next-generation CRISPR screens in immunology

Mapping the causal circuits that shape the phenotypic and functional landscape of immune cells remains a formidable challenge. Recent advances in pooled CRISPR-based screens, coupled with multiplexed single-cell profiling and imaging-based spatial readouts, make this goal increasingly attainable.

In this Perspective, we discuss how CRISPR-based genetic screens will fundamentally transform our understanding of immunobiology. We highlight the applications of state-of-the-art, high-throughput pooled perturbation approaches, including emerging methodologies for bulk, single-cell, and spatial CRISPR screens, to advance our understanding of immunity and in vivo biology. Additionally, we summarize new strategies to address the complexity of combinatorial perturbations to uncover genetic interactions and mechanistic drivers of immunity at unprecedented scale and resolution.

By integrating CRISPR screening data with experimental insights, we advocate a new framework in immunology research that leverages perturbation-driven regulatory effects and networks to discover new therapeutic targets and establish causal systems biology and immunology for advancing immunological knowledge and therapeutic application.

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ORIGINAL PAPER
Shi, H., and H. Chi. 2026. Next-generation CRISPR screens enable causal systems immunology. *J. Exp. Med.* 223 (3): e20241266. https://doi.org/10.1084/jem.20241266

REVIEW

Therapeutic opportunities for NK cells beyond cancer

Natural killer (NK) cells are cytotoxic and cytokine-producing innate lymphocytes with established roles in antiviral and antitumor immunity. In recent years, the biology of NK cells has been exploited in innovative cancer immunotherapies, leading to clinical advances including allogeneic NK cell infusions, chimeric antigen receptor NK cells, and NK cell engager technologies. These studies pave the way to explore how advances in NK cell–based immunotherapies could be leveraged outside of oncology to selectively target pathogenic cells and restore tissue homeostasis in viral infections, neurodegenerative disorders, autoimmunity, and transplantation medicine.

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ORIGINAL PAPER
Cayatte, M., V. Picant, M. Vétizou, and E. Vivier. 2026. Bringing natural killer cells to the clinic: Opportunities beyond cancer. *J. Exp. Med.* 223 (1): e20250612. https://doi.org/10.1084/jem.20250612

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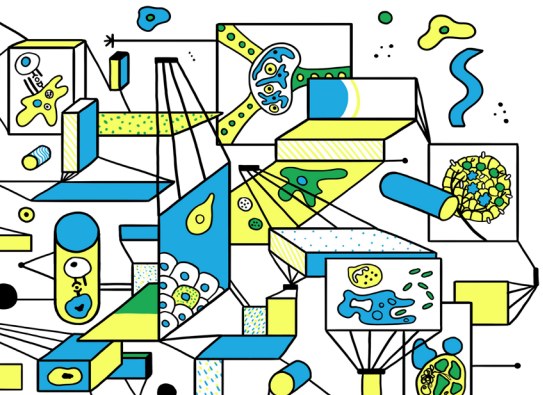

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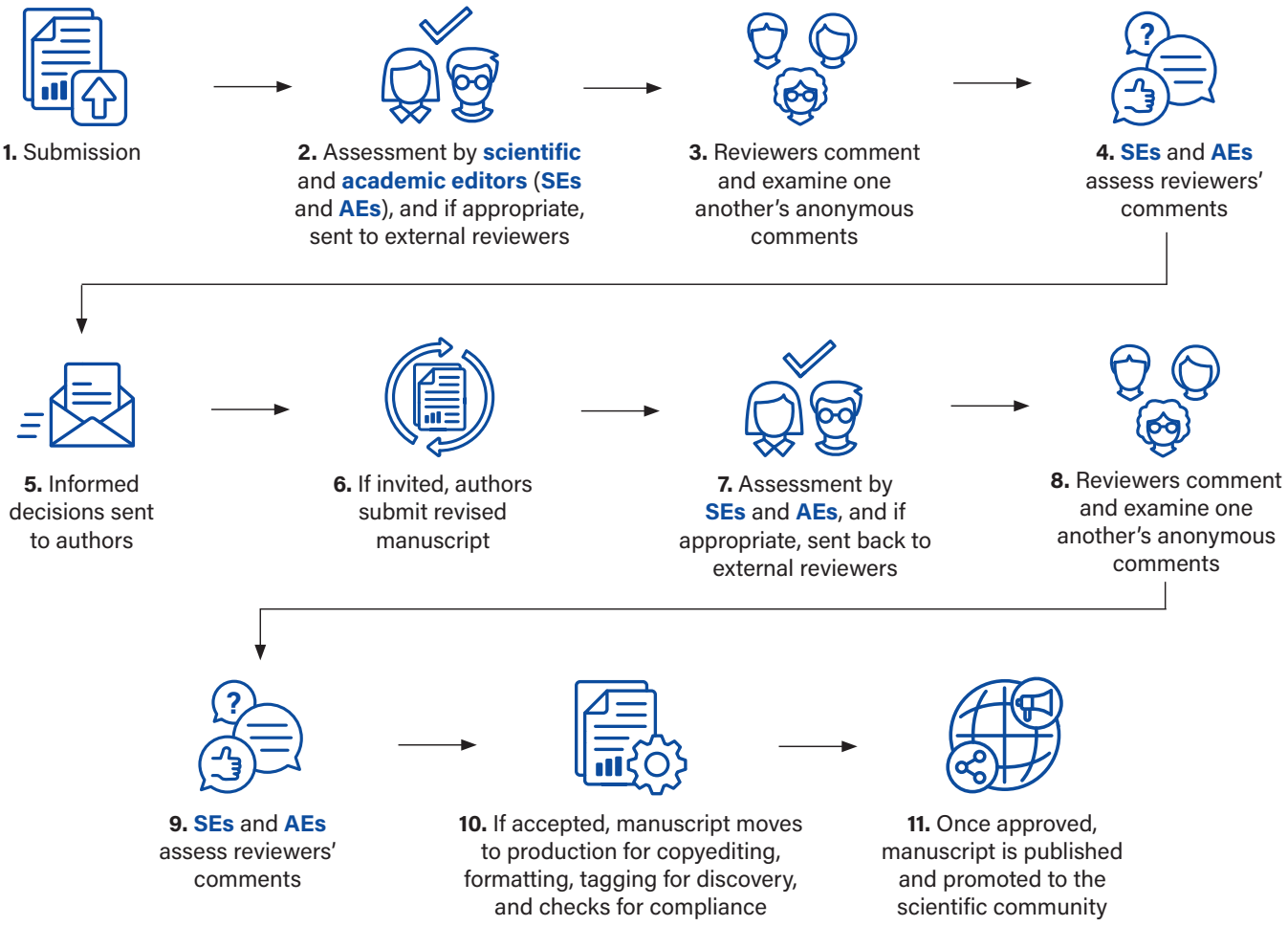

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