OPEN ACCESS Freely available online

Journal of Clinical and Cellular Immunology

Short Communication

A Brief Note on Tissue-Resident Memory Cells

Georg Gasteiger*

Department of System of Immunology, University of Würzburg, Germany

ABSTRACT

Tissue-resident memory T (TRM) cells provide as a first line of defense against infections that enter through barrier locations. TRM cells in the lungs protect against infections, although they decline faster than TRM cells in other tissues. Because there isn't a stable TRM population in the lung parenchyma, infections with pathogens like influenza and respiratory syncytial virus (RSV) can reoccur throughout life. Intranasal (IN) vaccination with a murine cytomegalovirus (MCMV) vector expressing the RSV M protein (MCMV-M) has been demonstrated to produce large numbers of $CD8^+$ TRM cells that accumulate over time and mediate early viral clearance. We evaluated the inflationary $CD8^+$ T cell population generated by MCMV-M vaccination with a traditional $CD8^+$ T cell population and further findings. Vaccination with MCMV-M2 resulted in a fast diminishing population of M2-specific $CD8^+$ TRM cells, similar to the M2-specific $CD8^+$ TRM cell population evoked by RSV infection. MCMV-M and MCMV-M2 administration, in contrast to the natural immune dominance profile, did not suppress the M-specific $CD8^+$ T cell response, implying that progressive expansion was driven by continuous antigen presentation, regardless of the competitive or regulatory effects of M2-specific $CD8^+$ T cells.

Keywords: M protein; Immune-dominance; Tissue-resident memory; MCMV-M

DESCRIPTION

Tissue-resident memory T (TRM) cells protect barrier tissues from entering pathogens by killing infected cells directly and activating other immune effector cell populations into the tissue. The migration pattern, function, and phenotype of TRM cells in numerous anatomical regions have been extensively studied in recent years. TRM cells have been shown to be diverse, with different needs for localization and maintenance in tissue types. TRM cells have been found to mediate immunological protection against RSV as well as heterosubtypic cross-protection against influenza virus in the lungs. TRM cells are also important for cancer immune protection. TRM cells, in particular, have been found to improve the efficacy of intranasal cancer vaccinations in orthotropic head and neck tumor models in mice. The presence of TRM cells in malignant lung tumors was also linked to human survival. TRM cells in the lungs, on the other hand, tend to diminish over time, possibly reflecting a harsher and more dynamic environment than in other barrier tissues. This gradual depletion of TRM cells most likely explains why RSV and influenza virus infections repeat throughout life. Vaccination techniques aiming at maintaining high TRM cell counts in the lungs may thereby improve immunity to respiratory infections and diseases [1-5].

In some tissues, cytomegalovirus (CMV) has been shown to elicit large numbers of TRM cells. The persistent nature of CMV leads to a unique phenomenon among memory $CD8^+$ T cells, which has been well characterized in mouse models using murine cytomegalovirus (MCMV). MCMV infection generates two distinct populations of memory $CD8^+$ T cells, known as conventional and inflationary memory $CD8^+$ T cells. Conventional $CD8^+$ T cell populations expand during acute infection and subsequently contract, whereas inflationary $CD8^+$ T cell populations accrue over time in the effector memory (EM) compartment, even if they do not predominate in the early phase. CMV vectors' ability to cause memory inflation could explain why they've showed promise as vaccine candidates, guarding against a variety of diseases and infectious pathogens while also acting as effective immune-contraception.

Correspondence to: Georg Gasteiger Department of System of Immunology, University of Würzburg, Germany, E-mail: georggasteiger@gmail.com

Received: 03- Feb-2022, Manuscript No. JCCI-22-16063; **Editor assigned:** 07- Feb-2022, PreQC No. JCCI-22-16063 (PQ); **Reviewed:** 21-Feb-2022, QC No. JCCI-22-16063; **Revised:** 28-Feb-2022, Manuscript No. JCCI-22-16063 (R); **Published:** 7-March-2022, DOI:10.35248/2155-9899.22.13.653.

Citation: Gasteiger G (2022) A Brief Note on Tissue-Resident Memory Cell. J Clin Cell Immunol. 13:653.

A variety of factors determine whether a given epitope elicits traditional or inflationary $CD8^+$ T cell populations. The source protein must be transcribed during latency for inflationary memory responses, a trait that is mostly determined by genome position. Furthermore, because antigen presentation occurs mostly on the surface of non-hematopoietic cells, which lack immune-proteasomes, the generated epitope may require processing by constitutive proteasomes. Given the observation that high-avidity clonotypes are preferentially selected for inflation during MCMV infection, interclonal competition may potentially play a role. In the case of human CMV infection, similar findings have been described. Other possible contributors include epitope-dependent co-stimulation requirements and $CD4^+$ T cell assistance.

CONCLUSION

Even in well-defined mouse models, memory inflation is difficult to anticipate, but a thorough understanding of the process is essential for the development of successful vaccinations that deliver protective antigens composited by CMV. The protocol was approved by the Animal Care and Use Committee of the Vaccine Research Center, National Institute of Allergy and Infectious Diseases, National Institutes of Health. The Association for Assessment and Accreditation of Laboratory Animal Care International completely accredited. All applicable federal and National Institutes of Health rules and regulations were strictly followed during the animal procedures.

REFERENCES

1. Masopust D, Vezys V, Marzo AL, Lefrancois L. Preferential localization of effector memory cells in nonlymphoid tissue. Science. 2001; 291(5512):2413–2417.
2. Gebhardt T, Wakim LM, Eidsmo L, Reading PC, Heath WR, Carbone FR,et al. Memory T cells in nonlymphoid tissue that provide enhanced local immunity during infection with herpes simplex virus. Nat Immunol .2009; 10(5):524–530.
3. Ariotti S, Hogenbirk MA, Dijkgraaf FE, Visser LL, Hoekstra ME, Song JY, et al. T cell memory. Skin-resident memory CD8 (+) T cells trigger a state of tissue-wide pathogen alert. Science.2014; 346(620): 5:101–105.
4. Shane HL, Klonowski KD. Every breath you take: the impact of environment on resident memory CD8 T cells in the lung. Front Immunol. 2014;5(8):320.
5. Schenkel JM, Masopust D. Tissue-resident memory T cells. Immunity.2014; 41(6):886–897.