OMB No. 3145-0279 (Expiration Date 10/31/2026)

NIH BIOGRAPHICAL SKETCH COMMON FORM

Name: Brossay, Laurent

Persistent Identifier (PID) of the Senior/Key Person: https://orcid.org/0000-0002-7497-8488

Position Title: Professor

Organization and Location: Brown University, Providence, RI, United States

PROFESSIONAL PREPARATION

INSTITUTION AND LOCATION	DEGREE	Start Date	Completion Date	FIELD OF STUDY
La Jolla Institute for Allergy & Immunology, San Diego, CA, United States	Postdoctoral Fellow	05/1997	12/1999	Immunology
UCLA, Los Angeles, CA, United States	Postdoctoral Fellow	07/1994	04/1997	Immunology
Laval University, Quebec City, Quebec, Canada	DOCTOR OF PHILOSOPHY	01/1990	06/1994	Immunology
Rennes University, Rennes, Not Applicable, N/A, France	MASTER OF SCIENCE	09/1986	06/1988	Immunology
Rennes University, Rennes, Not Applicable, N/A, France	BACHELOR OF SCIENCE	09/1983	06/1986	Biochemistry

Appointments and Positions

2000 - present Professor, Brown University, Providence, RI, United States
2011 - 2021 Chair Molecular Microbiology & Immunology Department, Brown University, Providence, RI, USA
2009 - 2011 Acting Chair Molecular Microbiology & Immunology Department, Brown University, Providence, RI, USA
2006 - 2014 Associate Professor, Brown University, Providence, RI, USA
2000 - 2006 Assistant Professor, Brown University, Providence, RI, USA

Products

Products Closely Related to the Proposed Project

1. Reilly SP, Smith ML, Borys SM, Fugère C, Demers D, Hogan MJ, Zemmour D, Brossay L. Unconventional CD8(+) T cell surveillance of cytomegalovirus via Qa-1/HLA-E-restricted epitope recognition. Sci Adv. 2025 Dec 19;11(51):eaea8707. PubMed Central PMCID: PMC12716397.
2. Mundy MA, Demers D, Brossay L. Lung NK cells are sufficient to control viral dissemination during respiratory MCMV infection. J Immunol. 2025 Jun 1;214(6):1310-1320. PubMed Central PMCID: PMC12207078.
3. Tata A, Dodard G, Fugère C, Leget C, Ors M, Rossi B, Vivier E, Brossay L. Combination blockade of KLRG1 and PD-1 promotes immune control of local and disseminated cancers. Oncoimmunology. 2021 Jun 15;10(1):1933808. PubMed Central PMCID: PMC8208121.
4. Myers JA, Jordon ARD, Borys SM, Dinh K, Brossay L. E-cadherin expression promotes tumor growth via KLRG1-dependent pathways. J Immunol. 2026 Apr 15;215(4) PubMed Central PMCID: PMC13108842.
5. Borys SM, Reilly SP, Magill I, Zemmour D, Brossay L. NK cells restrain cytotoxic CD8(+) T cells in the submandibular gland via PD-1-PD-L1. Sci Immunol. 2024 Dec 20;9(102):eadl2967. PubMed Central PMCID: PMC12099074.

Other Significant Products Highlighting Contributions to Science

1. Dodard G, Tata A, Erick TK, Jaime D, Miah SMS, Quatrini L, Escalière B, Ugolini S, Vivier E, Brossay L. Inflammation-Induced Lactate Leads to Rapid Loss of Hepatic Tissue-Resident NK Cells. Cell Rep. 2020 Jul 7;32(1):107855. PubMed Central PMCID: PMC7383148.
2. Hogan MJ, Maheshwari N, Begg BE, Nicastri A, Hedgepeth EJ, Muramatsu H, Pardi N, Miller MA, Reilly SP, Brossay L,

Lynch KW, Ternette N, Eisenlohr LC. Cryptic MHC-E epitope from influenza elicits a potent cytolytic T cell response. Nat Immunol. 2023 Nov;24(11):1933-1946. PubMed Central PMCID: PMC12116205.

3. Tessmer MS, Reilly EC, Brossay L. Salivary gland NK cells are phenotypically and functionally unique. PLoS Pathog. 2011 Jan 13;7(1):e1001254. PubMed Central PMCID: PMC3020929.
4. Wesley JD, Tessmer MS, Chaukos D, Brossay L. NK cell-like behavior of Valpha14i NK T cells during MCMV infection. PLoS Pathog. 2008 Jul 18;4(7):e1000106. PubMed Central PMCID: PMC2442879.
5. Anderson CK, Reilly EC, Lee AY, Brossay L. Qa-1-Restricted CD8(+) T Cells Can Compensate for the Absence of Conventional T Cells during Viral Infection. Cell Rep. 2019 Apr 9;27(2):537-548.e5. PubMed Central PMCID: PMC6472915.

Certification:

I certify that the information provided is current, accurate, and complete. This includes, but is not limited to, information related to current, pending, and other support (both foreign and domestic) as defined in 42 U.S.C. § 6605.

In accordance with Section 10632 of the CHIPS and Science Act of 2022 (42 U.S.C. § 19232), each individual identified as a senior/key person must certify that they are not a party to a malign foreign talent recruitment program.

Research Security Training Requirement for Federal Award Personnel: In accordance with Section 10634 of the CHIPS and Science Act of 2022 (42 U.S.C. § 19234), each individual identified as a senior/key person must certify that they have completed the requisite research security training that meets the requirements specified in Item 2 of Important Notice No. 149 within 12 months prior to proposal submission.

Misrepresentations and/or omissions may be subject to prosecution and liability pursuant to, but not limited to, 18 U.S.C. §§287, 1001, 1031 and 31 U.S.C. §§3729-3733 and 3802.

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NIH BIOGRAPHICAL SKETCH SUPPLEMENT

Name: Brossay, Laurent

Persistent Identifier (PID) of the Senior/Key Person: https://orcid.org/0000-0002-7497-8488

Position Title: Professor

Organization and Location: Brown University, Providence, RI, United States

Personal Statement

My laboratory is interested in understanding the molecular mechanisms controlling the development and activation of both Natural Killer (NK) and non-classical T cells. We investigate the contribution of these innate-like cells during the immune response to both infections, autoimmunity, and cancer. During the last 25 years, we have generated several tools, including an anti-CD1d antibody, an anti-KLRG1 antibody, the KLRG1 tetramer, and immunodeficient animals such as Qa-1-deficient mice and KLRG1-deficient mice. Most reagents are shared freely and/or donated to Jackson lab or NIH tetramer facility
I have served on numerous NIH study sections (~30) and I was a member of the Immunity and Host Defense study section. I also served as a Section Editor and Associate Editor for the Journal of Immunology and I am currently an Associate Editor for Frontiers in NK Cell Biology. I have been continuously funded by the NIH since 2000.
I was the chair of the Molecular Microbiology & Immunology Department from 2009 to 2021. As a chair, I recruited 6 tenure-track Assistant Professors. They have been awarded NIH RO1s, NIH R21s, NIH K99s, DARPA awards, DOD discovery awards, SEARLE award, and a multitude of local grants. Most of them have been project leaders on NIH COBRE grants. Papers published by these junior members are Immunity, Nature Immunology, Cell Host and Microbes, PloSPathogens etc. Finally, I am the mentor for several successful Assistant Professors from other departments.
I have graduated 14 graduate students, and I am currently training two, several of whom won presentation and/or poster awards at national or international conferences such as the American Association of Immunologist Conferences or Keystone conferences. Six graduate students from my laboratory were awarded an NIH F31 fellowship. Most of the graduate students I trained have published papers in top-tier journals as a first author. Overall, I am committed to mentoring my trainees and helping them to become successful and visible immunologists. I have been a trainer for the Brown Respiratory Research Training Program since 2017.

Honors

2015	American Association of Immunologist Laboratory Travel Award, AAI
2013	American Association of Immunologist Laboratory Travel Award, AAI
2004	American Association of Immunologist Junior Faculty Travel Award, AAI

Contributions to Science

1. NK Cell Response and Non-Classical T Cell Response during MCMV Infection:
   We first characterized KLRG1 as a marker of recently activated NK and CD8+ T cells. This is now widely accepted, and this marker is used in many critical studies, which aim to understand the development of memory CD8+ T cells. We then demonstrated that in response to MCMV, NK cells follow a kinetic parallel to CD8+ T cells. In this system, we showed that NK cells undergo accelerated phenotypic maturation, expand, and contract. This work has been in part the foundation of a novel concept called memory NK cells, developed by several investigators. We also identified Qa-1 as a restricting element for non-classical CD8+ T cells during MCMV infection. These cells can compensate for the absence of conventional T cells and protect against MCMV-induced lethality. We also identified the peptides presented by Qa-1 and characterized the TCR repertoire of the MCMV-specific non-classical CD8+ T cells.

2. Identification of the KLRG1 Ligands and Role of KLRG1 as a Checkpoint Inhibitor:
   Using a reporter cell line and the KLRG1 tetramer, we identified E, and N-cadherin as ligands for KLRG1. Our group also demonstrated a robust synergistic effect of ICI therapies targeting KLRG1 and Programmed Cell Death Protein 1 (PD-1) using a model of E-cadherin+ melanoma in vivo. The KLRG1 tetramer has been made available to the scientific community at the NIH tetramer facility. The KLRG1-deficient animals have been donated to Jackson Laboratories.

3. NK Cell-Like Behavior of iNKT Cell during Infection:

iNKT cells are unique T cells that can respond within minutes to specific antigens, mostly agonists such as α-GalCer. In the absence of a specific antigen, iNKT cells have been shown to contribute directly or indirectly to the immune response to several pathogens, leading to a second model of iNKT cell activation, which involves both TCR engagement and inflammatory cytokines. We hypothesized that in some cases, such as viral infection, iNKT cells may not need TCR engagement to be activated. Using the MCMV system, we demonstrated that iNKT cell activation is mostly TCR-independent. Therefore, iNKT cells essentially behave like NK cells during MCMV infection. These findings were confirmed by others using TCR reporter mice. In fact, the current view of iNKT cell activation during pathogen infection has shifted to a more important role of cytokine-mediated activation than TCR-mediated activation during infections.

4. Unique Population of NK cells and T cells in the Salivary Glands:
   NK cells are critical effector cells during the immune response to viral infections, yet despite their presence in the salivary glands, several viruses persist in this organ for several weeks to months. We demonstrated that salivary gland NK cells exhibit a unique phenotype characterized by reduced effector functions. New findings in the laboratory indicate that this mucosal tissue harbors several subsets of NK cell-like cells. We are currently testing the hypothesis that although NK cells cannot clear viral infections in this organ, the NK cell response is sufficient to prevent viral reactivation while limiting tissue injury. We also recently demonstrated that NK cells regulate CD8+ T cell number and frequency in the salivary glands.

5. Role of Phosphatases during NK Cell and NKT Cell Development:
   NK cell and NKT cell inhibitory receptors associate predominantly with SHP-1 or SHP-2, which are thought to dephosphorylate key players of cellular activation. Using mice deficient for SHP-1, SHP-2, or SHIP1, we have defined the roles of these phosphatases during NK and iNKT cell development.

Certification:

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