BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors.
Follow this format for each person. **DO NOT EXCEED FIVE PAGES.**

NAME: Jeffrey E. Dunn

eRA COMMONS USER NAME (credential, e.g., agency login): Dunn.Jeffrey

POSITION TITLE: Lily Sarafan Director of Neuroimmunology, Clinical Professor and Chief, Division of Neuroimmunology, Stanford University | Stanford Medicine

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Haverford College; Haverford, PA	BA	12/1983	French Literature
Institute d'Études Françaises; Avignon, France			Linguistics & translation
Temple University Katz School of Medicine; Philadelphia, PA	MD	05/1989	Medicine
University of Washington; Seattle, WA	Residency	07/1993	Neurology

A. Personal Statement

I specialize in the treatment and research of Multiple Sclerosis, Neuromyelitis Optica Spectrum Disorders (NMOSD), and related immune-mediated diseases of the central nervous system. I serve as Chief in the Division of Neuroimmunology within the Department of Neurology & Neurological Sciences at Stanford University in Palo Alto, California, USA. The Stanford Multiple Sclerosis Center is recognized as a Comprehensive Center of Care by the National Multiple Sclerosis Society and is an active Member Center of the Consortium of MS Centers. Together with the Neuroimmunology Clinic at the Stanford Neuroscience Health Center in Palo Alto, California, we serve a clientele of more than 6,000 MS patients with exceptional ethnic diversity and an international referral base. Our Neuromyelitis Optica Clinic cares for more than 300 patients with NMOSD making this one of the largest such clinics of its kind in the world. Regional attitude toward clinical research is highly supportive, enabling us to recruit successfully for our work in clinical trials and translational research. We have created a framework by which annotated human tissue is made available to our world-leading scientists for advanced analysis promoting discovery. This translational construct has yielded some of the more important discoveries within our subspecialty, including our discovery (with Lanz and Robinson) of EBV molecular mimicry as a causal mechanism of MS, and the discovery of KIR CD8+ cells (with Li and Davis) as a novel cell type that modulates autoimmunity. We are a world leader in the use of cellular immune therapy for neuroimmune diseases. Our efforts have been recognized as Runner-Up Science Breakthrough of the Year by the American Association for the Advancement of Science (AAAS) in 2022 (for the relationship of Epstein-Barr virus to MS) and in 2024 (for the first prospective human clinical trial of Chimeric Antigen Receptor CAR-T immunotherapy in MS). Educating the next generation of medical leaders is a highly valued component of our tripartite mission. The Stanford University Neurology residency program is now the largest of its kind in the western US, and our Neuroimmunology and Autoimmune Neurology Fellowship training programs have earned a nationally acclaimed reputation for success in mentoring committed subspecialty physician investigators who have consistently emerged as clinical leaders and investigators of impact and influence.

B. Positions, Scientific Appointments, and Honors

Positions:

1992-1993.	Chief Resident in Neurology, University of Washington; Seattle, WA
1993-1996.	Clinical Instructor in Medicine (Neurology), University of Washington; Seattle, WA
1997-2007.	Assistant Professor of Clinical Neurology, University of Washington; Seattle, WA
2008-2012.	Associate Professor of Clinical Neurology, Stanford University; Stanford, CA
2008-2019.	Neurology Clerkship Director, Stanford University; Stanford, CA
2009-2019	Member, Education Committee, Stanford University Neurology; Stanford, CA
2009-2017	Founder and Director, Neuroimmunology Fellowship Program; Stanford, CA
2011-2014	Chair, MS Education Workgroup, MS Section, American Academy of Neurology
2014-2016	Vice Chair, Executive Committee, MS Section, American Academy of Neurology
2016-2018	Course Director, Stanford Annual CME *Breakthroughs* Conference; SF, CA
2018-2020	Chair, Multiple Sclerosis Section, American Academy of Neurology; St. Paul, MN
2018-2020	Member, National Medical Advisory Board, National MS Society; New York, NY
2008-pres.	Member, Neurology residency ranking and selection committee, Stanford University
2016-pres	Member, Stanford Medicine Teaching & Mentoring Academy; Stanford, CA
2013-pres.	Professor of Clinical Neurology, Stanford University; Stanford, CA
2008-pres	Chief, Division of Neuroimmunology, Stanford University; Stanford, CA
2022-pres	Lily Sarafan Director of Neuroimmunology, Stanford University; Stanford, CA

Scientific Appointments:

2019-pres	Faculty, Wu Tsai Neurosciences Institute, Stanford University; Stanford, CA
2020-pres	Member, Stanford Institute for Immunity, Transplantation & Infection; Stanford, CA
2022-pres	Affiliated faculty, ChEM-H Innovation Hub, Stanford University; Stanford, CA
2022-present	Ordinary Member, International Society of Neuroimmunology
2008-pres	Fellow, American Academy of Neurology; St. Paul, MN

Honors:

2008-2009	Lysia Forno Award for Outstanding Departmental Teaching; Stanford Neurology
2008-2017	Neurology Clerkship Student Teaching Award, inclusive; Stanford University
2009-2010	Excellence in Teaching Award; Stanford University School of Medicine
2011-2012	Arthur L. Bloomfield Award for Excellence in Teaching Clinical Medicine; Stanford SOM
2011-2012	Highest percentage recruitment award, AUPN; Minneapolis, MN
2013-2014	Henry J. Kaiser Family Foundation Award for Excellence in Clinical Instruction
2010-pres	Castle Connolly “Top Doctors” 10 consecutive year award
2016	Special Commendation, California State Senate; Senator Wolk 3rd district
2017-2018	Arthur L. Bloomfield Award for Excellence in Clinical Instruction; Stanford SOM
2019-2020	HealthCare Partner of the Year Award, National MS Society; Santa Clara, CA
2019-pres	Eponymous designation, Fishers-Dunn Award for best resident teaching of students
2019-2020	AB Baker Teacher Recognition Award, American Academy of Neurology
2020-2021	Neurology Clerkship Teaching Award, Stanford Department of Neurology
2021	Oscar Salvatierra Award for Exceptional Service to Medical Students and SOM
2022	Science Breakthrough of the Year Runner-Up, EBV-MS: AAAS
2023	Certificate of Special Congressional Recognition, US Congress (CA-04)
2025	Kaiser Family Foundation Award for Excellence in Clinical Teaching
2025	Osler Excellence in Bedside Medicine Endowed Lectureship: Johns Hopkins
2025	Science Breakthrough of the Year Runner-Up, CAR-T in MS: AAAS

C. Contributions to Science

- **Cellular Immune Therapy for the Treatment of Multiple Sclerosis**

Chimeric Antigen Receptor Immunotherapy (CAR-T) has demonstrated great therapeutic benefit in some types of lymphomatous malignancy. With the single chain variable fragment CD-19, it is hypothesized that CAR-T therapy may have great therapeutic potential in autoimmunity including human neuroimmune diseases. Our group was the first to test CAR-T/CD-19 immunotherapy in a prospective Phase 1 Trial for progressive phenotypes of Multiple Sclerosis. We continue to expand clinical assessments in human subjects with plans to advance to Phase 2 trials this coming year. Our early focus in primary outcomes has been in feasibility and safety, but we have observed remarkable early clinical benefit including eradication of unique CSF oligoclonal bands in treated subjects with this therapy. Our work has been invited for presentation at ACTRIMS, ECTRIMS, AAAS and the American Academy of Neurology Annual Meetings in 2025.

Reference:

Chimeric Antigen Receptor T Cell Immunotherapy for Progressive Phenotypes of Multiple Sclerosis: early results from a Phase 1, Open Label Single Center Study of an Autologous Fully Humanized Anti-CD-19 CAR-T. **Jeffrey Dunn**, Anna Tomczak, Kristin Galetta, Garrett Timmons, Emma Martinez, Tobias Lanz, Monali Manohar, Bita Sahaf, Everett Meyer, Robert Lowsky. European Committee for the Treatment and Research of Multiple Sclerosis, 41st *ECTRIMS* Congress. Abstract # 2978; abstract category 4 Therapy-Immunomodulation. Barcelona, Spain: 23 Sept 2025

- **Collaborative discovery of molecular mimicry to Epstein-Barr virus as a causal pathway of MS**

Multiple Sclerosis afflicts more than one million men and women in the US and millions worldwide, but its cause has remained unknown for centuries. Unraveling the cause of Multiple Sclerosis is a requisite step toward cure or eradication. Working in collaboration with immunology researchers Lanz and Robinson at Stanford, our group identified, collected, and processed cerebrospinal fluid (CSF) samples from patients who met confirmed McDonald criteria for a definite diagnosis of MS and provided the acquired samples according to process and accession requirements. CSF derived antibody was identified by single cell sequencing of the paired B cell repertoire of MS blood and CSF, followed by protein microarray-based testing of recombinantly expressed CSF derived antibodies against MS associated viruses, employing sequence analysis, affinity measurements and crystal structuring. Results identified an antibody recognition cross reaction between the Epstein Barr virus transcription factor EBNA1 and the CNS protein GlialCAM. This finding imputes molecular mimicry as a mechanism by which Multiple Sclerosis may be caused in humans who have acquired EBV infection, which is known to be generally ubiquitous. The finding would explain why only a small minority of EBV infected persons develop MS, and suggests a pathway by which more rigorous and effective immunotherapy can be pursued and tested. Our findings were published in the journal *Nature*

Reference:

The B Cell Repertoire in Multiple Sclerosis Reveals Molecular Mimicry between EBV EBNA1 and GlialCAM. Tobias V. Lanz, R. Camille Brewer, Peggy P. Ho, Kevin M. Jude, Daniel Fernandez, Ricardo A. Fernandes, Alejandro Gomez, Gabriel Nadj, Ryan D. Schubert, Bianca Teegen, **Jeffrey E. Dunn**, Christopher B. Lock, Lucas B. Kipp, Victoria C. Cotham, Beatrix M. Ueberheide, Blake T. Aftab, Michael R. Wilson, Rachael J.M. Bashford-Rogers, Michael Platten, Raymond A. Sobel, Christopher K. Garcia, Lawrence Steinman, William H. Robinson. 2021-06-09836C. *Nature* DOI 10.1038/s41586-022-04432-7 *:* 10 March 2022

- **Collaborative discovery of human KIR+ CD8+ T cells in modulating autoimmunity**

In a collaborative effort with Dr. Jin Li and Dr. Mark Davis at Stanford, our Neuroimmunology group identified, collected, annotated and processed whole blood and plasma from men and women with confirmed Multiple Sclerosis and provided these to the Davis lab where advanced T cell immune-analysis was performed. The lab had previously identified that KIR CD8+ pathogenic T cells appeared to modulate autoimmunity favorably in

cases of celiac disease, and was able to extend this finding also to COVID and to Multiple Sclerosis. The finding serves to identify a novel T lymphocyte not previously known to have this immunomodulatory effect in autoimmune disease, opening up an opportunity for immune modulation at the clinical level with focus on T cell dynamics. This research has been submitted and accepted to the journal *Science*.

Reference:

Human KIR+ CD8+ T cells target pathogenic T cells in celiac disease and are active in other types of autoimmunity. Li, J., Zaslavsky, M., Sikora, M., Christophersen, A., Chiou, SH, Chen, L., Ji, X., Wilhemy, J., McSween, A., Van Unen, V., Palanski, B., Bhamidpati, K., Pai, J., Kipp, L., **Dunn, J.,** Hauser, S., Oksenberg, J., Satpathy, A., Robinson, W., Steinmetz, L., Khosla, C., Nadea, K., Utz, PJ, Sollid, L., Fernandez-Baker, N., Saligrama, N., Davis, M. *Science*, 15 Apr 2022, Vol. 376 (6590). Doi:10.1126/science.abi9591

- **Patent for biomarker of MS disease responsiveness to immunotherapy**

While there has been a burgeoning expansion of disease modifying therapies (DMT) that have shown statistical significance in reducing MS relapse rate, reducing MRI disease activity over a defined trial epoch, and in some cases reducing the rate of cumulative neurologic disability from MS, the individual patient response to prescription DMTs remains variable, heterogeneous and unpredictable. A biomarker that could satisfy Prentice criteria for disease status prediction could substantially augment DMT response rates and optimize neurologic outcomes. Other than neurofilament light chain, there has been no widely accepted biomarker for MS disease status. Collaborating with Drs. Axtell and Shubert, we identified a biomarker of patient responsiveness to prescription disease modifying therapy considered sufficiently unique and of sufficient potential to earn a patent from the US Patent Trade Office, subsequently licensed to a biotechnology interest for development via the Stanford Office of Technology Licensing.

References:

United States Patent & Trademark Office patent # 10054588; issue date 21 Aug 2018: “marker for determination of patient responsiveness.”

Interferon beta treatment requires B-cells for efficacy in neuro-autoimmunity. Schubert R., Hu Y., Kumar G., Szeto S., Abraham P., Winder, J., Guthridge JM, Pardo G., **Dunn J.,** Steinman L., Axtell R.C. *Journal of Immunology*, 2015; 194 (5): 2110-2116

- **Attenuated immunogenicity to COVID vaccination in MS patients on immunotherapy**

Multiple Sclerosis is a disease for which immunomodulatory and immunosuppressive treatments of different mechanisms of action (MOA) have been shown effective. FDA-approved prescription DMTs have been monitored for in-trial and post-marketing side effects with attention to infection risk and malignancy. Immunization effects of DMT have not been systematically studied. The need for such investigation became obvious with the advent of the COVID pandemic and widespread vaccination. Testing for post-vaccination immune status has been assessed by measuring immunoglobulin IgG to the spike protein of the SARS-CoV-2 coronavirus. Our group studied humoral and cellular responses to COVID vaccination in MS patients treated with a range of DMT MOA. We found the use of CD20+ specific monoclonal antibody therapies and S1P modulators are associated with attenuated post-vaccination immunoglobulin response, while use of other MOA of DMT yield normal IgG response.

Reference:

Invited platform and poster presentation: Adaptive B cell and T cell responses to CoV-2 vaccination in patients with Multiple Sclerosis on disease modifying immunotherapy. Tomczak, A., Sumera, J., Joseph, Y., Wu, D., McDonald, J., Sattarnezhad, N., Kipp, L., Lock, C., Hay, M., and **Dunn, J.** : *ACTRIMS*. 22 Feb 2022. West Palm Beach, Florida