BIOGRAPHICAL SKETCH

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NAME: Jeffrey E. Dunn

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POSITION TITLE: Professor of Clinical Neurology and Chief, Division of Neuroimmunology, Stanford

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
	Residency	07/1993	Neurology
University of Washington; Seattle, WA			

A. Personal Statement

I specialize in the diagnosis, treatment and research of Multiple Sclerosis, Neuromyelitis Optica Spectrum Disorders (NMOSD), transverse myelitis and 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 Stanford, California, USA. We are attentive to three core missions of patient care, research and education. 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 200 patients with NMOSD making this one of the largest clinics of its kind in the world. Regional attitude toward clinical research is highly favorable, enabling us to recruit successfully for our work in clinical trials and translational research. We are selective in our trial participation favoring Phase II and Phase III trials of high potential scientific value and rigorously considered methodologic design. We are actively collaborative across disciplines in our translational research. Our Center has developed an IRB approved comprehensive biorepository by which patients are identified and consented by CRAs embedded in clinic on site, yielding human tissue (including whole blood, cerebrospinal fluid, and stool) that is annotated by multi-faceted clinical, radiographic, metabolic and QOL metrics, processed and stored at the Stanford Clinical Trials Unit, available for expedited accession and collaborative curation with our world leading scientists. This architecture 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. Educating the next generation of medical leaders is emphasized. The Stanford University Neurology residency program is now the largest of its kind in the western US, and our Neuroimmunology Fellowship training program has earned a nationally acclaimed reputation for success in mentoring committed subspecialty physician investigators emerging as clinical leaders and investigators of 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
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

C. Contributions to Science

- **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. Submitted to *Science, accepted*

- **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

Analysis of B Cell Subsets in Multiple Sclerosis Patients on Immunomodulatory Therapy Reveals Modulation of CD19+CD24hiCD38hi Cells with Implications for the Diagnosis and Monitoring of MS. Schubert R., Goodyear A., Abraham P., Dunn C., Steinman L.; Senior authors: **Dunn J.** and Axtell R.: American Academy of Neurology Annual meeting abstract # P03.224; San Diego, CA: 19 March 2013

Biomarker panel increases sensitivity for identification of inflammatory MS disease activity beyond NfL. Gawde, S., Agasing, A., Bhatt, N., Tolliver, M., Pardo, G., **Dunn, J.**, Axtell, R. *Manuscript in submission*

- **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. Patients with attenuated humoral response can still mount cellular immune response via acquired T cell immunity as assessed by interferon gamma release assay, showing that effective human immunity can be achieved by different immune pathways working either individually or collectively. This finding could have important implications for how best to assess post-vaccination immune status for vaccines or boosters against other pathogens, and challenges an existing inclination employed by public health professionals to employ IgG antibody response to denote human immune status.

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

- **Discovery of a new disease**

This case report of intracerebral bacillary angiomatosis described a previously unreported medical disease that at the time represented the only fully treatable-curable opportunistic infection of the brain caused by HIV-AIDS. This represented discovery of a new disease and offered hope.

Reference:

Intracerbral Bacillary Angiomatosis in a patient infected with HIV; Spach, D., Panther, L., Thorning, D., **Dunn, J**. Plorde, J.L., Miller, R.; *Annals of Internal Medicine; 1992: 116; 740-743*