

**BIOGRAPHICAL SKETCH**

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NAME: Michael A. Spinner, MD

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

POSITION TITLE: Instructor in Oncology, Stanford Cancer Institute

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)	Start Date MM/YYYY	Completion Date MM/YYYY	FIELD OF STUDY
Emory University, Atlanta, GA	B.A.	08/2005	05/2009	Music (Classical guitar performance)
Vanderbilt University School of Medicine, Nashville, TN	M.D.	07/2009	05/2014	Medicine
Stanford Hospital & Clinics, Stanford, CA	Residency	07/2014	06/2016	Internal Medicine
Stanford Cancer Institute, Stanford, CA	Fellowship	07/2016	06/2020	Hematology/Oncology

**A. Personal Statement**

I am an Instructor in the Division of Oncology at the Stanford Cancer Institute specializing in the management of patients with Hodgkin and non-Hodgkin lymphoma and am engaged in a variety of clinical trials and translational research collaborations related to lymphoma. Under the mentorship of Dr. Ranjana Advani, I have developed particular expertise in Hodgkin lymphoma and have published numerous manuscripts and abstracts on the treatment of Hodgkin lymphoma, including chapters for the ASCO and ASH education programs and for Williams Hematology. I am a co-investigator on multiple trials investigating novel targeted therapies and immunotherapies for Hodgkin and non-Hodgkin lymphomas, both in the frontline setting and for relapsed or refractory disease. My specific research interests include: (1) novel approaches to immunotherapy, for example activating macrophages in the immune microenvironment by targeting the CD47/SIRP $\alpha$  immune checkpoint and exploring potential synergy with PD-1/PD-L1 blockade, and (2) precision oncology, combining genomics, gene expression profiling, and *ex vivo* drug sensitivity screening to inform personalized therapy, particularly for patients with relapsed or refractory disease. Ultimately, my goal is to improve outcomes for patients with Hodgkin and non-Hodgkin lymphomas and to shift treatment paradigms towards well tolerated immunotherapy and targeted therapy approaches.

1. Spinner MA, Advani RH, Connors JM, Azzi J, Diefenbach C. New treatment algorithms in Hodgkin lymphoma: too much or too little? *Am Soc Clin Oncol Educ Book*. 2018;38:626-636.
2. Spinner MA, Advani RH. Risk-adapted therapy for advanced stage Hodgkin lymphoma. *Hematology Am Soc Hematol Educ Program*. 2018;2018(1):200-206.
3. Spinner MA, Varma G, Advani RH. Modern principles in the management of nodular lymphocyte-predominant Hodgkin lymphoma. *Br J Haematol*. 2019;184(1):17-29.
4. Spinner MA, Aleshin A, Santaguida MT, et al. *Ex vivo* drug screening defines novel drug sensitivity patterns for informing personalized therapy in myeloid neoplasms. *Blood Adv*. 2020;4(12):2768-78.

## B. Positions and Honors

### Positions and Employment

2014 - 2016 Internal Medicine Resident, Stanford Hospital & Clinics, Stanford, CA  
2016 - 2020 Hematology/Oncology Fellow, Stanford Cancer Institute, Stanford, CA  
2020 - Instructor in Oncology, Stanford Cancer Institute, Stanford, CA

### Certification and Licensure

2016 - California State Medical License  
2017 - American Board of Internal Medicine, Board Certified  
2020 American Board of Hematology, Board Eligible  
2020 American Board of Medical Oncology, Board Eligible

### Professional Memberships

2014 - Member, American College of Physicians  
2014 - Member, American Society of Hematology  
2016 - Member, American Society of Clinical Oncology

### Honors and Awards

2007 William H. Murdy Natural Science and Mathematics Award, Emory University  
2008 Phi Beta Kappa, Emory University  
2012 - 2013 NIH Medical Research Scholars Program, National Institutes of Health  
2013 - 2014 NIAID Merit Award, for outstanding clinical research leading to the discovery and characterization of GATA2 deficiency, National Institute of Allergy and Infectious Diseases  
2014 Best of *Blood*, first author plenary paper selected as one of the top 15 most outstanding manuscripts in *Blood* in 2014 and cited over 400 times, American Society of Hematology  
2014 Rudolph H. Kampmeier Prize in Clinical Medicine, Vanderbilt University School of Medicine  
2014 David N. Orth Award in Endocrinology, Vanderbilt University School of Medicine  
2014 Alpha Omega Alpha, Vanderbilt University School of Medicine  
2014 Julian Wolfsohn Award for Outstanding Performance in Internal Medicine, Stanford Internal Medicine Residency Program  
2014 Stanford Society of Physician Scholars, Stanford University School of Medicine  
2016 ASH Abstract Achievement Award, American Society of Hematology  
2018 ASH Clinical Research Training Institute, American Society of Hematology  
2020 Scientific Merit Award, Stanford Hematology/Oncology Fellowship Program  
2020 Young Investigator Award, Conquer Cancer Foundation of ASCO

## C. Contributions to Science

I worked with Dr. Steven Holland at the National Institutes of Health (NIH) on characterizing the clinical and pathologic features of a primary immunodeficiency and bone marrow failure disorder caused by heterozygous germline mutations in *GATA2*, which encodes a critical hematopoietic transcription factor. We published a comprehensive clinical characterization of *GATA2* deficiency in a cohort of 57 patients, identified novel genotype-phenotype correlations, and demonstrated that allogeneic hematopoietic cell transplantation (HCT) may be curative. I was the lead author on the manuscript, which was selected as a Plenary Paper in *Blood* and has been cited over 400 times. I also wrote a clinical protocol to prospectively follow patients with *GATA2* deficiency at the NIH to better characterize the natural history of the disease and better determine when to intervene with allogeneic HCT (NCT01905826). In 2014, I received an NIH Merit Award for outstanding clinical research leading to the characterization of *GATA2* deficiency.

1. Spinner MA, Sanchez LA, Hsu AP, et al. *GATA2* deficiency: a protean disorder of hematopoiesis, lymphatics, and immunity. *Blood*. 2014;123(6):809-21.
2. Spinner MA, Sanchez LA, Hsu AP, et al. *GATA2* deficiency: extended clinical phenotype in 57 patients. *J Clin Immunol*. 2013;33(3):672.

3. Spinner MA, Ker JP, Stoudenmire CJ, et al. GATA2 deficiency underlying severe blastomycosis and fatal herpes simplex virus-associated hemophagocytic lymphohistiocytosis. *J Allergy Clin Immunol*. 2016 Feb;137(2):638-40.
4. Spinner MA, Odio C, Calvo KR, et al. Hemophagocytic lymphohistiocytosis associated with NK cell dysfunction and disseminated herpesvirus infection in GATA2 deficiency. *Blood*. 2014;124(21):4978.

I worked with Dr. Robert Lowsky in the Stanford Division of Blood and Marrow Transplantation on several projects related to reducing the risk of graft-versus-host disease (GVHD) and other toxicities following allogeneic HCT. We demonstrated that a nonmyeloablative conditioning regimen of fractionated total lymphoid irradiation and antithymocyte globulin (TLI-ATG) is protective against GVHD with low transplant-related mortality (TRM) and allowed for outpatient allogeneic HCT with most patients never hospitalized within the first 100 days post-transplant. The low risk of GVHD and TRM was observed even among the highest risk subgroups, including patients with partially HLA-mismatched unrelated donors. We recently published our 15-year single-center experience using this regimen in a large cohort of over 600 patients with lymphoid and myeloid malignancies. I have also worked with Dr. Lowsky on multiple trials investigating the safety and efficacy of a CD8+ memory T-cell donor lymphocyte infusion (DLI) for the treatment and prevention of relapse post-transplant. We have now treated over 40 patients with this novel cellular immunotherapy and have demonstrated graft-versus-tumor activity with a lower incidence of acute GVHD compared to historical controls receiving unmanipulated DLI. I am a co-investigator on two clinical trials exploring CD8+ memory T-cell DLI in patients with high-risk lymphoid and myeloid malignancies (NCT02424968, NCT04151706).

1. Spinner MA, Kennedy VE, Tamaresis JS, et al. Nonmyeloablative TLI-ATG conditioning for allogeneic transplantation: mature follow-up from a large single-center cohort. *Blood Adv*. 2019;3(16):2454-64.
2. Spinner MA, Advani RH, Hoppe RT, et al. Allogeneic transplantation using TLI-ATG conditioning for Hodgkin lymphoma after failure of autologous transplantation. *Blood Adv*. 2018;2(13):1547-1550.
3. Spinner MA, Fernández-Viña M, Creary LE, et al. HLA-mismatched unrelated donor transplantation using TLI-ATG conditioning has a low risk of GVHD and potent antitumor activity. *Blood Adv*. 2017;1(17):1347-57.
4. Spinner MA, Muffly LS, Arai S, et al. Consolidative CD8+ memory T-cell donor lymphocyte infusion augments donor chimerism with a low risk of GVHD following hematopoietic cell transplantation. *Blood*. 2017;130:2003.

I have worked with Dr. Ranjana Advani in the Stanford Division of Oncology for the past three years during which time I have developed particular expertise in the management of Hodgkin lymphoma. I have been involved in numerous clinical trials incorporating targeted therapies such as brentuximab vedotin and the immune checkpoint inhibitors in the frontline setting or as first salvage along with trials investigating novel agents such as the anti-CD25 antibody-drug conjugate camidanlumab tesirine for heavily pretreated patients with Hodgkin lymphoma. I have published numerous manuscripts on the management of Hodgkin lymphoma including chapters for the ASCO and ASH education programs and for the 10<sup>th</sup> edition of Williams Hematology. I am an active participant in our weekly Hodgkin Lymphoma Tumor Boards and have collaborated on multiple research projects with colleagues in the Departments of Pathology, Radiation Oncology, and Blood and Marrow Transplantation. I have also been involved in national and international research collaborations on clinical research projects in Hodgkin lymphoma, diffuse large B-cell lymphoma, and peripheral T-cell lymphomas.

1. Spinner MA, Advani RH, Connors JM, Azzi J, Diefenbach C. New treatment algorithms in Hodgkin lymphoma: too much or too little? *Am Soc Clin Oncol Educ Book*. 2018;38:626-636.
2. Spinner MA, Advani RH. Risk-adapted therapy for advanced stage Hodgkin lymphoma. *Hematology Am Soc Hematol Educ Program*. 2018;2018(1):200-206.

3. Spinner MA, Varma G, Advani RH. Modern principles in the management of nodular lymphocyte-predominant Hodgkin lymphoma. *Br J Haematol.* 2019;184(1):17-29.
4. Sica RA, Spinner MA, Tamaresis JT, et al. Improved outcomes for relapsed/refractory classic Hodgkin lymphoma following autologous hematopoietic cell transplantation in the era of novel agents. *Blood.* 2019;134:2022.

I worked with Dr. Peter Greenberg in the Division of Hematology at Stanford and collaborated with a biotech company, Notable Labs (Foster City, CA), evaluating their novel *ex vivo* drug sensitivity screening (DSS) platform in patients with myelodysplastic syndrome (MDS) and related myeloid neoplasms. We partnered with Notable Labs to create a prioritized list of 74 individual drugs and 36 drug combinations to treat patient blood samples or bone marrow aspirate samples *ex vivo* and evaluate blast viability and differentiation, using high-throughput multiparametric flow cytometry to gate on the myeloid blast population. We conducted a pilot and feasibility study in 54 patients with myeloid neoplasms and demonstrated that Notable's *ex vivo* DSS assay can be performed reliably and within a clinically actionable timeframe (median of 15 days following bone marrow biopsy). We found that *ex vivo* DSS defined distinct patient clusters with differential sensitivity to various drug classes, including hypomethylating agents, cytotoxic agents, kinase inhibitors, and other small molecules. Correlating *ex vivo* and *in vivo* responses, we demonstrated that Notable's assay had a positive predictive value of 92%, negative predictive value of 82%, and overall accuracy of 85% in our discovery cohort. Additional patients are being accrued to validate these findings, and ongoing studies are focused on identifying correlations between specific mutations and *ex vivo* drug sensitivity patterns. Future studies are also planned to evaluate Notable's *ex vivo* DSS platform in patients with lymphoid malignancies.

1. Spinner MA, Aleshin A, Santaguida MT, et al. *Ex vivo* drug screening defines novel drug sensitivity patterns for informing personalized therapy in myeloid neoplasms. *Blood Adv.* 2020;4(12):2768-78.
2. Spinner MA, Aleshin A, Santaguida MT, et al. A feasibility study of biologically focused therapy for myelodysplastic syndrome patients refractory to hypomethylating agents. *Blood.* 2019;134:4239.
3. Aleshin A, Santaguida MT, Spinner MA, et al. *Ex vivo* drug response profiling defines novel drug sensitivity patterns for predicting clinical therapeutic responses in myeloid neoplasms. *Blood.* 2018;132:4356.
4. Spinner MA, Schaffert SA, Aleshin A, et al. Correlating clinical and genomic features with *ex vivo* drug sensitivity in patients with myelodysplastic syndrome and related myeloid neoplasms. Submitted for presentation at the 2020 ASH Annual Meeting.

#### **Complete List of Published Work in MyBibliography:**

<https://www.ncbi.nlm.nih.gov/myncbi/1nQKhtsbqoHkk/bibliography/public/>

#### **D. Research Support**

Young Investigator Award, Conquer Cancer Foundation                      Spinner (PI)                      07/2020 - 06/2021

A Phase 2 Study of Magrolimab (anti-CD47 antibody) and Pembrolizumab in Patients with Relapsed or Refractory Classic Hodgkin Lymphoma.

This single-arm phase 2 study will assess the safety and efficacy of a novel immunotherapy combination in patients with relapsed or refractory classic Hodgkin lymphoma after two or more systemic therapies. Twenty-four patients will be accrued at two sites, the Stanford Cancer Institute and the Dana-Farber Cancer Institute. The primary efficacy endpoint is the complete remission (CR) rate, and the investigators hypothesize that the combination of magrolimab and pembrolizumab will increase the CR rate compared to historical controls receiving pembrolizumab alone. The study will also evaluate for potential biomarkers of response including CD47 and PD-L1 expression levels in Hodgkin Reed-Sternberg cells, changes in tumor biopsies after treatment, and changes in circulating tumor DNA levels after treatment.