BIOGRAPHICAL SKETCH

Provide the following information for all key personnel.
Follow the sample format for each person found in **Biosketch Sample**. **DO NOT EXCEED FOUR PAGES.**

NAME	**POSITION TITLE**
Brian D. Poole, PhD	Assistant Professor Dept. of Microbiology and Molecular Biology Brigham Young University

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)*

INSTITUTION AND LOCATION	**DEGREE** *(if applicable)*	**YEAR(s)**	**FIELD OF STUDY**
Brigham Young University	B.S.	1993-1999	Microbiology
Pennsylvania State University	PhD	1999-2004	Immunology/virology
Oklahoma Medical Research Foundation	(Postdoc)	2004-2008	Immunology/virology

A. Positions and Honors.

Trustees Scholar, Brigham Young University 1993-1999
Life Science Consortium Fellow, Pennsylvania State University, 1999-2001
Arthritis Foundation trainee award 2002, 2003
Research Assistant, Pennsylvania State University, 2002-2003
Writer for "Science Lions" Column in the Pennsylvania State University Newspaper *The Daily Collegian*, 2002-2003
Intercollege Graduate Student Outreach Award, Pennsylvania State University, 2004
Associate Research Scientist, Oklahoma Medical Research Foundation, 2004-2008
Member Post-doctoral committee, Oklahoma Medical Research Foundation, 2005-2008
Assistant Professor, Brigham Young University, 2008-present

B. Selected peer-reviewed publications (in chronological order).

Poole, Brian D; Karetnyi, Yuory; and Naides, Stanley J. Parvovirus B19-Induced Apoptosis of Hepatocytes. Journal of Virology 2004 78(14):7775-7783.

McClain, Micah; **Poole, Brian D**.; Bruner, Benjamin; Kaufman, Kenneth; Harley, John, and James, Judith A. An altered immune response to Epstein-Barr virus nuclear antigen-1 (EBNA-1) in pediatric systemic lupus erythematosus. Arthritis & Rheumatism, 2005 Dec 29;54(1):360-368

Poole, Brian D.; Scofield, R. Hal; Harley, John B.; and James, Judith A. Epstein-Barr Virus and Molecular Mimicry in Systemic Lupus Erythematosus. Autoimmunity 2006 Feb;39(1):63-70.

Poole, Brian D.; Zhou, Jing, Grote, Amy; Schiffenbauer, Adam; and Naides, Stanley. Apoptosis of Liver-Derived Cells Induced by Parvovirus B19 Nonstructural Protein. Journal of Virology, 2006 April; 80(8):4114-4121.

Poole, Brian D. and James, Judith A. Infection and Autoimmunity. *In* Systemic Lupus Erythematosus, a Companion to Rheumatology. George C. Tsokos, Caroline Gordon, And Josef S. Smolen, Editors. Elsevier Press, Philadelphia, PA. Jan 2007 pp143-155.

Poole, Brian D., Gross, Timothy, Maier, Shannon, Harley, John B., James, Judith A. Lupus-like Autoantibody development in rabbits and mice after immunization with EBNA-1 fragments. Journal of Autoimmunity 2009 (4):362-71.

Poole, Brian D.,Schneider, Rebecca I.; Guthridge, Joel M.; Velte, Cathy; Reichlin, Morris; Harley, John B.; James, Judith A. Early Targets of nRNP Humoral Autoimmunity in Human Systemic Lupus Erythematosus. Arthritis and Rheumatism 2009 60(3):848-59.

Poole, Brian D.; Templeton, Amanda K.; Guthridge, Joel M.; Brown, Eric J.; Harley, John B.; James, Judith A. Aberrant Epstein-Barr viral infection in systemic lupus erythematosus. Autoimmun Rev. 2009 8(4):337-42.

C. Research Support.

Current

Brian D. Poole (PI) Mentoring Environment Grant. 02/01/2009-/01/31/2011This award is to provide an environment for undergraduate research at Brigham Young University.

Completed

Brian D. Poole (PI). Functional manifestations of SLE Risk Haplotypes on EBV infection and Related Stimuli NIH 5 P20 RR020143-05.07/2008-08/2008. This award is to study the role of the IRF 5 risk haplotype on Epstein-Barr virus infection and gene expression in the development of systemic lupus erythematosus. The grant was only utilized for one month because of a change in institution and position.

Brian D. Poole (PI). Molecular Basis of Immunity NIH T32 AI007633-07. 09/2006-08/2008. This award is to study the role of Toll-like receptors in lupus. I was responsible for designing the experiments, carrying out the research, analyzing the results and supervising support personnel who worked on this project.

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D. Research interests

Systemic lupus erythematosus is a common, debilitating autoimmune disease in which components of the body's own cells are targeted and attacked by the immune system. Lupus is ninefold more prevalent in women than in men, often being diagnosed during the childbearing years. The origins of the disease are still unclear, but it is known that genetic, environmental, and hormonal factors are all involved in causing lupus. We are studying the interactions of genetic risk factor and the putative environmental factor Epstein-Barr virus (EBV). Considerable evidence indicates that EBV infection is associated with lupus. The major question with this finding is that nearly everyone has been infected with EBV, while very few of those people will go on to develop lupus. The genetic background of the person may play a key role in deciding the outcome of the infection. Although there is a strong genetic component to lupus, not much is known about how genes influence the development of the disease. We are focusing on lupus-associated variations in the IRF5 gene. The IRF5 protein is a key intermediary in the response to viral infection, and interacts with EBV during the course of the EBV infection. We hypothesize that the modifications to the IRF5 gene associated with lupus cause aberrant activation of EBV and heightened response to EBV infection, potentially leading to the altered immune response to EBV seen in lupus patients and people at risk for lupus. Through studying these interactions, we hope to identify markers for people who are at high risk for lupus, and early indicators that autoimmunity is developing. Early intervention in the disease has shown promise in slowing its effects.