## UNITED STATES DISTRICT COURT FOR THE SOUTHERN DISTRICT OF NEW YORK

ASSOCIATION FOR MOLECULAR PATHOLOGY;
AMERICAN COLLEGE OF MEDICAL GENETICS;
AMERICAN SOCIETY FOR CLINICAL
PATHOLOGY; COLLEGE OF AMERICAN
PATHOLOGISTS; HAIG KAZAZIAN, MD; ARUPA
GANGULY, PhD; WENDY CHUNG, MD, PhD;
HARRY OSTRER, MD; DAVID LEDBETTER, PhD;
STEPHEN WARREN, PhD; ELLEN MATLOFF, M.S.;
ELSA REICH, M.S.; BREAST CANCER ACTION;
BOSTON WOMEN'S HEALTH BOOK COLLECTIVE;
LISBETH CERIANI; RUNI LIMARY; GENAE
GIRARD; PATRICE FORTUNE; VICKY
THOMASON; KATHLEEN RAKER,

No. 09 Civ. 4515 (RWS)

**ECF** Case

Plaintiffs,

## -againsţ-

UNITED STATES PATENT AND TRADEMARK OFFICE; MYRIAD GENETICS; LORRIS BETZ, ROGER BOYER, JACK BRITTAIN, ARNOLD B. COMBE, RAYMOND GESTELAND, JAMES U. JENSEN, JOHN KENDALL MORRIS, THOMAS PARKS, DAVID W. PERSHING, and MICHAEL K. YOUNG, in their official capacity as Directors of the University of Utah Research Foundation,

Detenuants.																												
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I, Jeffrey D. Parvin, declare under penalty of perjury:

1. I currently hold several positions at The Ohio State University, including: Interim-Chair, Department of Biomedical Informatics since 2008; Director, Biomedical Informatics Shared Resource of the Ohio State University Comprehensive Cancer Center; Professor, Department of Biomedical Informatics since 2007; Louis Levy Professor for Cancer since 2007; and Adjunct Professor, Department of Molecular Virology, Immunology and Medical Genetics since 2008.

- 2. In 1982 I received a B.S. from Haverford College in chemistry. In 1987 I received a Ph.D. from the Mount Sinai School of Medicine in microbiology. In 1989 I received an M.D. also from Mount Sinai School of Medicine. From 1989-1994 I was a postdoctoral research fellow in biology at the Center for Cancer Research at Massachusetts Institute of Technology. I have served as both an assistant and an associate professor of pathology at the Harvard Medical School. A copy of my curriculum vitae and a listing of my BRCA1-related publications are attached as Exhibits 1 & 2, respectively.
- 3. I began researching the *BRCA1* gene in 1997, shortly after Myriad's publication of the full-length *BRCA1* sequence. I have published 21 peer-reviewed articles reporting my research on the BRCA genes in research collaborations with over 80 different scientists around the world. My most current *BRCA1* research has yielded another paper in press to be published in early 2010 and yet another awaiting review. I have also published 12 articles reviewing the BRCA research conducted by others. I was aware that Myriad patents cover aspects of the BRCA genes.
- 4. During my *BRCA1* research I have helped elucidate various physiological roles and properties of the protein encoded by the *BRCA1* gene. For example:
  - (a) BRCA1 plays a role in DNA damage repair and the cell cycle (e.g., BRCA1 and BARD1 heterodimerization is stabilized via domains not previously thought to interact and BARD1 acts in both ubiquitination-

<sup>&</sup>lt;sup>1</sup> "Ref. \_" below refers to the indicated publication listed in the attached Exhibit 2.

dependent and ubiquitination-independent ways to influence the role of BRCA1 in DNA repair). (Refs. 21 & 23.)

- (b) BRCA1 is an RNA polymerase II holoenzyme-bound protein involved in both transcription activation and repression (e.g., the BRCA1 domain responsible for transcription regulation). (Refs. 1, 4, 7, 16, & 17.)
- (c) Defective BRCA1 plays a role in cancer genesis via disregulated centrosome duplication, including the specific ubiquitination domains involved in such disregulation. (Refs. 13, 14, 18, 19, & 20.)
- (d) Multiple distinct protein complexes comprising BRCA1 mediate the multiple processes with which BRCA1 is involved (rather than one supercomplex). (Ref. 6.)
  - (e) BRCA1 is involved in histone modification. (Ref. 22.)
- (f) Despite previous focus on BRCA1's conserved amino- and carboxy termini, an internal ubiquitin ligase domain of BRCA1 mediates a potent growth suppressive effect. (Ref. 9.)
- (g) My BRCA1 research has even helped elucidate the functions of other transcription activation proteins based on their interaction with BRCA1 protein. (Ref. 5.)
- 5. This research involved, *inter alia*, plasmids containing *BRCA1* cDNA, heterologous cells expressing BRCA1 protein, primers designed to PCR amplify *BRCA1* cDNA, and antibodies against BRCA1 protein.
- 6. Until asked to prepare this declaration, I had never been contacted by Myriad Genetics, Inc. Neither the patents at issue in this suit nor Myriad Genetics, Inc., have in any way hampered my research.

I declare, pursuant to 28 U.S.C. §1746, under penalty of perjury under the laws of the United States, that the foregoing is true and correct to the best of my knowledge and belief.

Jeffrey D. Parvin, MD, Ph.D

Executed on 12/08, 2009

## **CERTIFICATE OF SERVICE**

This is to certify that on December 23, 2009, a true and correct copy of the foregoing document has been served on all counsel of record via the court's ECF system.

/s/ Brian M. Poissant Brian M. Poissant