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,

Plaintiffs,

-against-

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,

Defendants.

No. 09 Civ. 4515 (RWS)

ECF Case

I, Richard Baer, declare under penalty of perjury:

1. I currently hold the following positions at Columbia University: Professor of Pathology & Cell Biology; Deputy Director, Institute for Cancer Genetics; and Assoc. Dir. for Basic Research, Herbert Irving Comprehensive Cancer Center, Columbia University Medical Center, Irving Cancer Research Center.

2. In 1976 I received a B.A. from Rutgers College in Biological Sciences. In 1981 I received a Ph.D. from Rutgers University in Microbiology. My post-doctoral research at the MRC-Laboratory of Molecular Biology (Cambridge, UK) and my subsequent work as an independent scientist has focused on the molecular biology of cancer. A copy of my *curriculum vitae* and a listing of my *BRCA1*-related publications are attached as Exhibits 1 & 2.

3. I began researching the *BRCA1* gene in 1994, not long after Myriad's landmark publication of the full-length *BRCA1* sequence. In research collaborations with over 90 different scientists since 1994 I have published 27 peer-reviewed articles on *BRCA1*. Of these 27 publications, 24 report my own original research while the other 3 review the voluminous research conducted by myself and others on *BRCA1*. I was aware that aspects of the BRCA genes were covered by Myriad's patent rights.

4. During my *BRCA1* research I have helped elucidate various physiological roles and properties of the *BRCA1* gene and the protein it encodes. For example:[1]

(a) We discovered and/or further elucidated several aspects of *BRCA1* itself, including: BRCA1's participation in DNA damage response (refs. 10, 16, 25); how BRCA1 expression is controlled during the cell-cycle (refs. 9, 19, 25); and how BRCA1 exerts its DNA damage repair effects (ref. 1).

[1] "Ref. _" below refers to the indicated publication listed in the attached Exhibit 2.

(b) We discovered a novel gene, *BARD1*, and discovered that BRCA1 exerts its various effects in the cell in tandem with the BARD1 protein. (Ref. 26.)

(c) We discovered and/or further elucidated several aspects of the BRCA1/BARD1 heterodimer, including: the nuclear co-localization of BARD1 and BRCA1 (ref. 25); BARD1's role in nuclear retention of BRCA1 (ref. 13); the unique pattern of polyubiquitination catalyzed by the BARD1/BRCA1 heterodimer (ref. 11); the critical role BARD1 plays in BRCA1's homologous repair function (refs. 4, 10); the mechanism by which the BARD1/BRCA1 heterodimer mediates RNA processing (ref. 8); and the essential role of the BARD1/BRCA1 heterodimer in normal embryonic development (ref. 12) and in tumor suppression (ref. 2).

(d) We discovered and/or further elucidated several aspects of *BARD1* itself, including: *BARD1*'s genomic structure (ref. 24); the association between *BARD1* mutation and breast, ovarian and endometrial cancers (ref. 24); the importance of BARD1 phosphorylation in DNA damage repair (ref. 7); and the way in which cancer-associated BARD1 mutations give rise to cancer (ref. 4).

(e) We discovered the interaction between the BRCA1 protein and CtIP, a protein involved in DNA damage responses and control of RNA transcription (refs. 22 and 19). We also contributed to studies implicating CtIP in cell cycle checkpoint control (ref. 6) and resection of double-strand DNA breaks (ref. 27).

5. My 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.

Richard Baer

Richard Baer, Ph.D

Executed on December 7, 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