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

DECLARATION OF RONG LI, PhD

I, Rong Li, declare under penalty of perjury:

1. I currently hold several positions at the University of Texas Health Science Center at San Antonio (UTHSCSA), including: Professor, Department of Molecular Medicine/Institute of Biotechnology since 2007; Co-Leader, Cancer Development & Progression, Cancer Therapy & Research Center 2008; and Member, Executive Committee, Cancer Therapy & Research Center since 2009.

2. In 1985 I received a B.S. from Fudan University, Shanghai, PR China in genetics. In 1991 I received a Ph.D. from the University of California, Berkeley in molecular biology. From 1991 to 1993 I was a postdoctoral research fellow in DNA tumor viruses at the University of California, Berkeley. From 1994 to 1996 I was a postdoctoral research fellow in cell cycle control at Cold Spring Harbor Laboratories. I have served as both an assistant and an associate professor of biochemistry and molecular genetics at the University of Virginia. 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 1999, shortly after Myriad's publication of the full-length *BRCA1* sequence. I have published 22 peer-reviewed articles reporting my research on the BRCA genes in research collaborations with over 50 different scientists around the world. I was aware that aspects of the BRCA genes are covered by Myriad's patent rights.

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

(a) We discovered and/or further elucidated several aspects of *BRCA1* itself, including: BRCA1 alters the local chromatin structure and stimulates chromosomal DNA replication (which in turn suggests increasing chromatin accessibility may be an important mechanism used by other transcription factors to facilitate multiple nuclear processes) (ref. 22.); a new activation domain in BRCA1 that may contribute to the heterogeneous risk of BRCA1 mutations in different tissues. (ref. 20); BRCA1's role in processing

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

the replication protein A-associated intermediates during double-stranded break repair (ref. 17); BRCA1's mechanisms for sensing xenobiotics and regulating AhR-dependent stress responses to these xenobiotics could play a role in BRCA1's tumor suppressing ability (ref. 3); and decreased BRCA1 confers tamoxifen resistance in breast cancer cells by altering estrogen receptor-coregulator interactions (ref. 1).

(b) We have helped in elucidating why BRCA1 deficiency leads disproportionately to breast and ovarian cancers. For example: BRCA1's coordinating role in gene expression may ensure the appropriate quantity and quality of the mature transcripts for certain breast and ovarian cancer-related genes, as well as the genetic integrity of the breast and ovary tissues (ref. 12); BRCA1 deficiency in epithelial and certain nonepithelial cells may result in combined effects of aberrant estrogen biosynthesis and compromised DNA repair capability, which in turn may lead to specific cancers in the breast and ovary (ref. 11); and in addition to its well-characterized activities in breast epithelial cells, a role of BRCA1 in modulation of estrogen biosynthesis in ASCs [adipose stromal cells] may also contribute to its tissue-specific tumor suppressor function (ref. 9).

(c) We developed a novel chromatin unfolding assay that can aid in the characterization of deleterious mutations in the C-terminal transactivation domain of BRCA1 and may provide more reliable presymptomatic risk assessment. (Ref. 18.)

(d) We discovered and/or further elucidated the physiological relationship between BRCA1 and COBRA1. For example: A novel cofactor of BRCA1 (COBRA1) is recruited to the chromosome site by the first BRCT

repeat of BRCA1, and is itself sufficient to induce chromatin unfolding (ref. 19); COBRA1 causes RNA polymerase II (RNAPII) to pause at the promoter-proximal region, which may be important to control the duration and magnitude of a rapid and reversible hormonal response (ref. 14); COBRA1 is a novel oncogene in upper gastrointestinal adenocarcinomas (ref. 10); COBRA1 plays a critical role in the regulation of clustered gene expression at preferred chromosomal domains in breast cancer cells (ref. 7); COBRA1 may coordinate multiple steps in ligand-dependent gene expression, which in turn ensures both the quantity and quality of hormone-stimulated gene products (ref. 6); COBRA1 and BRCA1, significant overlap of COBRA1- and BRCA1-regulated genes, may engage in common gene regulatory pathways to suppress breast cancer progression (ref. 5); and a lack of COBRA1 expression in breast carcinoma may serve as a useful indicator for poor prognosis (ref. 4).

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 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.

Rong Li, 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