BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors.
Follow this format for each person. **DO NOT EXCEED FOUR PAGES.**

NAME	POSITION TITLE
Weston, Claire Rosemary	CEO
eRA COMMONS USER NAME (credential, e.g., agency login) CWESTON110	

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

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	MM/YY	FIELD OF STUDY
University of Wales, Cardiff, UK	B.Sc. (Hons)	06/97	Biotechnology
Cambridge University, Cambridge, UK	Ph.D.	08/01	Cell Biology
University of Massachusetts Medical School	Postdoctoral	12/06	Cell Biology

A. Personal Statement

This proposal aims to use nanoparticles to develop a multiplex assay for prostate cancer biomarkers. There is a critical need for assays of this type as it is becoming increasingly apparent that patient treatment strategies can be adjusted based on the expression of multiple biomarkers rather than the small number in clinical use today. Multiplexing in a single tissue section allows the expression of multiple biomarkers to be quantified on a cell by cell basis, resulting in powerful data that is not currently available from current practices. I am very motivated to ensure the success of this project as I believe that it will have a positive clinical impact that could benefit a large patient population.

I have considerable relevant expertise in this field; with over 12 years experience developing assays to detect biomarkers in both human and rodent tissue. In particular, I have optimized and validated immunohistochemical, immunofluorescent, and *in situ* hybridization assays that require a range of specialized antigen retrieval techniques, detection methods, multiplexing, and quantification. For example, at Vala Sciences I developed multiplex assays for the quantification of the estrogen receptor, progesterone receptor, and HER2/neu in human breast biopsies and cancer cell lines using quantum dots. The multiplex assay in this proposal builds on the techniques we have already developed in our laboratory, and therefore has a strong technical chance of success.

My career to date has focused on cancer research, starting as a graduate student at Cambridge University in the U.K. During my tenure as a post doctoral fellow at the University of Massachusetts Medical School, under the guidance of Dr. Roger Davis, I enhanced my interest in cancer research and initiated investigations into the role of c-Jun NH2-terminal kinase (JNK) in breast cancer. I am currently the Founder & CEO of Reveal Biosciences where I lead a team of histologists and image analysis experts quantifying biomarker expression in a range of diseased tissues including cancer. Reveal Biosciences provides research services to over 30 biotech, pharmaceutical, academic and government institutions and also participates in translational research to develop and

validate new clinical assays that address critical unmet medical needs. I am very enthusiastic about being part of this translational research and hope that my experience and expertise will allow me to help ensure the success of the proposed project.

B. Positions and Honors

Positions and Employment

1995-1996 Industrial Training Student, Molecular Pathology Unit, GlaxoWellcome, UK
1997-2001 Graduate Student, Cambridge University, UK
2001-2006 Postdoctoral Fellow, University of Massachusetts Medical School
2006-2011 Scientist, PharmaScouts Inc.
2011-2012 Senior Scientist, Vala Sciences, Inc.
2012 – present Founder & CEO, Reveal Biosciences LLC

Other Experience and Professional Memberships

2006-2009 Scientific Editor (Cancer Biology, Cell Biology, Signal Transduction), BioEdit

Honors

1997 Beverton Prize for Best Overall Student (Pure & Applied Biology), University of Wales
1999 First Place, Graduate Poster, Babraham Institute, Cambridge, UK

C. Selected Peer-reviewed Publications

Most relevant to the current application

1. Weston CR, Wong A, Hall JP, Goad ME, Flavell RA, Davis RJ. (2003). JNK initiates a cytokine cascade that causes Pax2 expression and closure of the optic fissure. *Genes Dev. 17(10):1271-80.*
2. Kelkar N, Delmotte MH, Weston CR, Barrett T, Sheppard BJ, Flavell RA, Davis RJ. (2003). Morphogenesis of the telencephalic commissure requires scaffold protein JNK-interacting protein 3 (JIP3). *Proc Natl Acad Sci U S A. 100(17):9843-8.*
3. Weston CR, Wong A, Hall JP, Goad MEP, Flavell RA, Davis RJ. (2004). The c-Jun NH_2-terminal kinase is essential for epidermal growth factor expression during epidermal morphogenesis. *Proc Natl Acad Sci U S A. 101(39):14114-9.*
4. Tran E, Azuma YT, Chen M, Weston C, Davis R, Flavell R. (2006). Inactivation of c-Jun NH2-Terminal Kinase-1 enhances innate IL10 production and dampens autoimmune inflammation in the brain. *Proc Natl Acad Sci U S A. 103(36):13451-6.*
5. Cellurale C, Weston CR, Reilly J, Garlick DS, Jerry DJ, Sluss HK and Davis RJ. (2010). Role of JNK in a Trp53-dependent mouse model of breast cancer. PLoS One. *5(8):e12469*

Additional recent publications of importance to the field (in chronological order)

1. Pell JM, Hill RA, Stewart CE, Weston CR, Flick-Smith HC. (2000). Enhancement of insulin-like growth factor I activity by novel antisera: potential structure/function interactions. *Endocrinology. 141(2):741-51.*
2. Weston CR, Davis RJ. (2001). Signal transduction: signaling specificity- a complex affair. *Science. 292(5526):2439-40.*
3. Weston CR, Davis RJ. (2002). The JNK signal transduction pathway. *Curr Opin Genet Dev. 12(1):14-21.*
4. Weston CR, Lambright DG, Davis RJ. (2002). Signal transduction. MAP kinase signaling specificity. *Science. 296(5577):2345-7.*
5. Garner AP, Weston CR, Todd DE, Balmanno K, Cook SJ. (2002). ΔMEKK3:ER* activation induces a p38 alpha/beta 2-dependent cell cycle arrest at the G2 checkpoint. *Oncogene. 21(53):8089-104.*
6. Weston CR, Balmanno K, Chalmers C, Hadfield K, Molton SA, Ley R, Wagner EF, Cook SJ. (2003). Activation of ERK1/2 by deltaRaf-1:ER* represses Bim expression independently of the JNK or PI3K pathways. *Oncogene. 22(9):1281-93.*
7. Ley R, Balmanno K, Hadfield K, Weston C, Cook SJ. (2003). Activation of the ERK1/2 signaling pathway promotes phosphorylation and proteasome-dependent degradation of the BH3-only protein, Bim. *J Biol Chem. 278(21):18811-6.*
8. Todd DE, Densham RM, Molton SA, Balmanno K, Newson C, Weston CR, Garner AP, Scott L, Cook SJ. (2004). ERK1/2 and p38 cooperate to induce a p21CIP1-dependent G1 cell cycle arrest. *Oncogene. 23(19):3284-95.*
9. Molton SA, Weston C, Balmanno K, Newson C, Todd DE, Garner AP, Cook SJ. (2005). The conditional kinase ΔMEKK1:ER* selectively activates the JNK pathway and protects against serum withdrawal-induced cell death. *Cell Signal. 17(11):1412-22.*
10. Weston CR, Davis RJ. (2007). The JNK signal transduction pathway. *Curr Opin Cell Biol. 19(2):142-9*

D. Research Support

Ongoing Research Support

None