BIOGRAPHICAL SKETCH

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

NAME: Joy, Anna M.

eRA COMMONS USER NAME (credential, e.g., agency login):

POSITION TITLE: Senior Research Scientist

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

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Arizona State University, Tempe, AZ	B.S.	05/1982	Chemistry
Arizona State University, Tempe, AZ	Ph.D.	12/1989	Biochemistry
Barrow Neurological Institute, Phoenix, AZ	Post Doc	03/1990	Cancer Biology

A. Personal Statement My career has been devoted to understanding the role of the PI3K/AKT pathway in tumor initiation, progression and drug response with the goal of developing effective biomarkers and therapeutics. My multidisciplinary background in Chemistry, Cell Biology, the Biology and Clinical issues in Brain tumors and Bioinformatic analysis of Brain Tumor Genomics has prepared me for collaborative, cross disciplinary work that uses innovative approaches to investigate important clinical problems. As the PI on multiple institutional grants and an NIH KO1, I hired and managed employees and volunteers, collaborated effectively and produced peer reviewed publications. This and my extensive experience with cell/molecular biology, experimental design and data analysis has prepared me to direct the genomics lab at PVAMU.

Selected List of Published Works in mybibliography

https://www.ncbi.nlm.nih.gov/sites/myncbi/1lc8Gv-M2tkk6c/bibliography/55327985/public/?sort=date&direction=ascending

B. Positions and Honors

POSITIONS

Senior Research Scientist	Prairie View A & M University, Prairie View TX	2017 – present
Research Assistant Professor	Barrow Neurological Institute, Phoenix AZ	2007 – 2016
Associate Scientist	The Translational Genomics Research Inst. Phoenix, AZ	2002 - 2005

HONORS

Select Poster; Society for NeuroOncology Annual Meeting, Las Vegas NV	2008
Invited Plenary Presentation: Society for Neuro-Oncology	2002
American Association for Brain Tumor Research Fellowship	1994 – 1995

OTHER EXPERIENCE

Member Downtown Biomedical Campus Proteomic Steering Committee	2004 - 2005
Guest Reviewer OncoTargets and Therapy	

Guest Reviewer Medical Science Monitor
Member American Association for Cancer Research
Member Society for Neuro-Oncology

C. Contributions to Science

Glioblastoma is a very aggressive tumor that does not respond well to therapy. Development of effective treatments is impeded by inter-tumor heterogeneity that confuses selection of treatments and reduces resolution of clinical trials. We also lack a detailed mechanistic understanding of oncogene pathway topology, how it varies between tumors and within cells in the same tumor and the effect on therapy resistance. The major thrust of my research is twofold:

(1) *Identify genomic subtypes of Glioblastoma then investigate their oncogene pathway topology and therapeutic sensitivities*. My collaborators and I used high throughput genomic databases to develop an oncogene pathway-based approach to find genomic subtypes of Glioblastoma. A major outcome of this work is the discovery of one AKT subtype and data indicating median survival of these patients can be increased 5 years by use of an alternate, approved chemotherapy drug[1]. This work led to one patent:

PI3K/AKT pathway subgroups in cancer: Methods of using biomarkers for diagnosis and therapy.
US patent application 20120252856, Patent Pending

In unpublished work I used TCGA proteomic data to investigate topology of oncogenic signaling in genomic subtypes. My collaborators and I developed and applied an algorithm that indicates there are differences in PI3K/AKT pathway "wiring" between AKT subtypes. This suggests subtypes will differ in response to PI3K/AKT inhibitors.

(2) *Develop a detailed mechanistic understanding of the PI3K/AKT pathway in Glioblastoma*. I used molecular/cell biological approaches to show the three isoforms of the oncogene, AKT, have different roles and downstream substrates in Glioblastoma[2]. The data unexpectedly indicates AKT3 loss accelerates progression. AKT2 and AKT3 interacted differently with mTORC2 and this may underlie their different roles. Thus pan AKT inhibitors may be detrimental in some tumors and isoform-specific AKT inhibitors may be needed.

D. Additional Information: Research Support and/or Scholastic Performance

K01 NS064952 Joy (PI) $644,100 4/1/2010-3/30/2016
NIH/NINDS: Targeting the PI3K/Akt pathway in High Grade Glioma
Develop pathway based molecular classification of GBM. Find Akt isoform(s) that drive malignancy.
Mentor: Burt Feuerstein

Barrow Neurological Foundation Joy (PI) $25,000 8/1/2012 – 7/31/2013
Are Akt subgroups predictive markers in GBM?
Develop animal models of AKT subgroups to test subgroup-specific therapeutics.

Barrow Neurological Foundation Joy (PI) $25,000 8/1/2011 – 7/31/2012
Do AKT subgroups predict therapeutic response in GBM?
Optimize AKT based molecular classification of GBM.

Barrow Neurological Foundation Joy (PI) $50,000 8/1/2009 – 7/31/2010
Targeting the PI3K/AKT pathway in High Grade Glioma
Investigate the role of AKT isoforms in High Grade Glioma

Barrow Neurological Foundation Joy (PI) $55,337 8/1/2008 – 7/31/2009
Targeting the PI3K/AKT pathway in Astrocytic Tumors.
Investigate the role of AKT isoforms in astrocytic tumors.

R01 NS042262 Berens (PI) $450,000 08/1/2002-07/31/2004
NIH/NINDS: Genetic Pathways of Glioma Invasion
Find genetic pathways driving invasive behavior of malignant glioma.
Role: Co-Investigator

R21 NS043446 Berens (PI) $150,000 9/1/2003 – 8/31/2005
NIH/NINDS: Arrested Migration Fosters Apoptosis of Glioma Cells
Investigate relationship between migration and apoptosis and how to use this to sensitize glioma to cytotoxic therapy.
Role: Co-Investigator

Arizona Biomed. Research. Comm. Moffet (PI) $150,000 7/1/1994-6/30/1997
Role of bFGF and FGF receptors in proliferation of glioma cells
Role: Co-Investigator