

Fiscal Year:	FY 2011	Task Last Updated:	FY 08/06/2010
PI Name:	Wang, Huichen		
Project Title:	Molecular Basis of DNA Repair and Protection from Apoptosis in Neuronal Progenitors Exposed to Space Radiation		
Division Name:	Human Research		
Program/Discipline:	HUMAN RESEARCH		
Program/Discipline--Element/Subdiscipline:			
Joint Agency Name:	TechPort:	No	
Human Research Program Elements:	(1) SR :Space Radiation		
Human Research Program Risks:	(1) BMed :Risk of Adverse Cognitive or Behavioral Conditions and Psychiatric Disorders		
Space Biology Element:	None		
Space Biology Cross-Element Discipline:	None		
Space Biology Special Category:	None		
PI Email:	huwang@pvamu.edu	Fax:	FY
PI Organization Type:	UNIVERSITY	Phone:	936-261-3156
Organization Name:	Prairie View A & M University		
PI Address 1:	P.O. Box 519 MS2230		
PI Address 2:	New Science Bldg 322		
PI Web Page:			
City:	Prairie View	State:	TX
Zip Code:	77446	Congressional District:	10
Comments:	Formerly at Emory University, relocated in September 2014 (Ed., 7/7/15)		
Project Type:	GROUND	Solicitation / Funding Source:	2008 Space Radiobiology NNJ08ZSA001N
Start Date:	10/01/2008	End Date:	09/30/2012
No. of Post Docs:	2	No. of PhD Degrees:	
No. of PhD Candidates:		No. of Master' Degrees:	
No. of Master's Candidates:	1	No. of Bachelor's Degrees:	
No. of Bachelor's Candidates:		Monitoring Center:	NASA JSC
Contact Monitor:	Cucinott1a, Francis	Contact Phone:	281-483-0968
Contact Email:	noaccess@nasa.gov		
Flight Program:			
Flight Assignment:			
Key Personnel Changes/Previous PI:			
COI Name (Institution):	Krzysztof, Reiss (Neurological Cancer Research, Stanley S. Scott Cancer Center, Louisiana State University Health Sciences Center, New Orleans, LA)		
Grant/Contract No.:	NNX08BA08G		
Performance Goal No.:			
Performance Goal Text:			
Task Description:	<p>The health risks to astronauts exposed to space radiation include cognitive deficits and possibly accelerated aging. While the pathogenesis of radiation-induced cognitive dysfunction remains largely uncharacterized, it is thought to include loss of neural progenitors from the brain. Understanding of the molecular and cellular bases underlying neuronal loss and/or dysfunction is absolutely required for the development of counter measures before, during and possibly after space missions. Since experiments in humans are not possible, studies in this direction will benefit from appropriate biological model systems. The neurodegenerative effects of space radiation are likely to derive from DNA damage in the central nervous system (CNS). Therefore, research involving repair of this type of DNA lesions is critical for the development of new neuroprotective countermeasures. In the present proposal, we introduce an in vitro model of neural progenitors (neurospheres), which is derive from the brain of mouse embryo from neurodegenerative transgenic mice to</p>		

	study the detrimental effects of space radiation at the mechanistic level. Using this biological model, we will study DNA damage repair and apoptosis of proliferating and differentiated neural progenitor exposed to low dose of high charge and energy nuclei and protons. The proposed studies will provide novel insights into the molecular and cellular mechanisms underlying CNS risks from space radiation and will help to predict and countermeasure health risks from space radiation particularly with regard to effects on the CNS.
Rationale for HRP Directed Research:	
Research Impact/Earth Benefits:	This proposal will study the mechanism of DNA damage and oxidative stress in neuronal cells induced by high energy particle, iron, and protons, compared to X-ray. This study will provide possible ways to develop accurate quantitative estimates to the risk of the central nervous system (CNS) from galactic cosmic ray (GCR) and solar particles events following long-term space travel.
Task Progress:	<p>We described DNA repair pathways (PARP-1 and ATM) in neural cell survival following exposure to high LET radiation. High energy particles induced larger RBE in cell survival and DNA damage of hippocampal neurons compared to X-ray radiation.</p> <p>PARP and ATM deficiency increased the RBE of cell survival and DNA damage induced by high charge and energy particles.</p> <p>We found that Inhibition of cyclin dependent kinase 5 and glycogen synthase kinase 3 beta could reverse radiation sensitivity in neuronal cells and neurospheres, but enhance radiation sensitivity in glioblastoma cells.</p>
Bibliography Type:	Description: (Last Updated: 11/13/2019)
Abstracts for Journals and Proceedings	<p>Wang M, Tian L, Tang X, Wang H. "Interaction of PARP-1 and CDK5 in DNA damage response in neural cells exposed to space radiation." 21st Annual NASA Space Radiation Investigators' Workshop, Port Jefferson, New York, May 2010.</p> <p>21st Annual NASA Space Radiation Investigators' Workshop, Port Jefferson, New York, May 2010. , May-2010</p>
Articles in Peer-reviewed Journals	<p>Wang H, Liu S, Zhang P, Zhang S, Naidu M, Wang H, Wang Y. "S-phase cells are more sensitive to high-linear energy transfer radiation." Int J Radiat Oncol Biol Phys. 2009 Jul 15;74(4):1236-41. PubMed PMID: 19545789 , Jul-2009</p>
NASA Technical Documents	<p>Cucinotta F, Wang H, Huff JL. "Risk of Acute or Late Central Nervous System Effects from Radiation Exposure." Human Health and Performance Risks of Space Exploration Missions. ed. by J.C. McPhee and J.B. Charles. Houston, Tex. : NASA, 2010. p. 191-212, NASA SP 2009. , May-2009</p>