

Fiscal Year:	FY 2011	Task Last Updated:	FY 01/18/2011
PI Name:	Kucik, Dennis F. M.D., Ph.D.		
Project Title:	Mechanisms, early events, and dose dependence of radiation-induced atherosclerosis		
Division Name:	Human Research		
Program/Discipline:	HUMAN RESEARCH		
Program/Discipline--Element/Subdiscipline:	HUMAN RESEARCH--Radiation health		
Joint Agency Name:	TechPort:	No	
Human Research Program Elements:	(1) SR: Space Radiation		
Human Research Program Risks:	(1) Cardiovascular: Risk of Cardiovascular Adaptations Contributing to Adverse Mission Performance and Health Outcomes		
Space Biology Element:	None		
Space Biology Cross-Element Discipline:	None		
Space Biology Special Category:	None		
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Zip Code:	35205-4831	Congressional District:	7
Comments:			
Project Type:	GROUND	Solicitation / Funding Source:	2009 Space Radiobiology NNJ09ZSA001N
Start Date:	02/01/2011	End Date:	01/31/2014
No. of Post Docs:	No. of PhD Degrees:		
No. of PhD Candidates:	No. of Master' Degrees:		
No. of Master's Candidates:	No. of Bachelor's Degrees:		
No. of Bachelor's Candidates:	Monitoring Center: NASA JSC		
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Flight Program:			
Flight Assignment:			
Key Personnel Changes/Previous PI:			
COI Name (Institution):			
Grant/Contract No.:	NNX11AC61G		
Performance Goal No.:			
Performance Goal Text:	<p>Radiation causes vascular inflammation, which is a known risk factor for atherosclerosis. Epidemiological studies have shown that radiation from many sources, including cancer treatments, atomic bombs, and excessive occupational exposure all increase the risk for atherosclerosis. Previous studies, using gamma radiation, have demonstrated that radiation causes increased white blood cell (WBC) adhesion to the blood vessel wall, an essential early event in atherosclerotic plaque formation. What is not known is whether the cosmic radiation astronauts will be exposed to on missions to the moon and Mars will similarly increase the risk for atherosclerosis.</p> <p>In our current project, we established that X-ray, ⁵⁶Fe (iron ion) and proton irradiation of blood vessel cells increase adhesiveness of the vessel wall, and that X-rays and ⁵⁶Fe accelerate development of atherosclerosis in a mouse model (results of proton experiments are pending). The molecular mechanism for this, however, is not yet known. In addition, it</p>		

Task Description:	<p>remains to be determined how fractionation of doses and irradiation of other tissues affect the dose dependence of both cell adhesion and development of atherosclerosis.</p> <p>With the hypothesis that radiation in general and cosmic radiation in particular directly alter the adhesive properties of vascular endothelium, and resultant vascular inflammation accelerates atherosclerosis, we propose to systematically investigate mechanisms of radiation effects on vascular cells, using both isolated cells and whole mice, to better predict risk and to provide the basis to develop possible future countermeasures. Our specific aims are:</p> <p>Aim 1: Determine whether atherogenic effects of radiation are limited to local effects on vascular endothelium, or if other systems contribute to disease progression and/or modify dose dependence.</p> <p>Aim 2: Determine the molecular mechanism of acute activation of leukocyte-endothelial cell adhesion in response to radiation.</p> <p>Aim 3: Determine how fractionation of doses affects dose-dependence of progression rates, latency periods, and surrogate endpoints.</p>
Rationale for HRP Directed Research:	
Research Impact/Earth Benefits:	
Task Progress:	New project for FY2011.
Bibliography Type:	Description: (Last Updated: 04/12/2018)