

<b>Fiscal Year:</b>	FY 2024	<b>Task Last Updated:</b> FY 04/08/2024	
<b>PI Name:</b>	Turner, Russell T Ph.D.		
<b>Project Title:</b>	Time Course for Re-Adaptation of Thermoregulation and Bone Following Spaceflight		
<b>Division Name:</b>	Space Biology		
<b>Program/Discipline:</b>			
<b>Program/Discipline-- Element/Subdiscipline:</b>			
<b>Joint Agency Name:</b>		<b>TechPort:</b>	No
<b>Human Research Program Elements:</b>	None		
<b>Human Research Program Risks:</b>	None		
<b>Space Biology Element:</b>	(1) Cell & Molecular Biology (2) Animal Biology: Vertebrate		
<b>Space Biology Cross-Element Discipline:</b>	(1) Musculoskeletal Biology		
<b>Space Biology Special Category:</b>	None		
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<b>Zip Code:</b>	97331-8558	<b>Congressional District:</b>	4
<b>Comments:</b>			
<b>Project Type:</b>	Flight	<b>Solicitation / Funding Source:</b>	2018 Space Biology NNH18ZTT002N:Russian Bion-M2 Mission
<b>Start Date:</b>	04/01/2020	<b>End Date:</b>	04/21/2025
<b>No. of Post Docs:</b>	0	<b>No. of PhD Degrees:</b>	0
<b>No. of PhD Candidates:</b>	0	<b>No. of Master' Degrees:</b>	0
<b>No. of Master's Candidates:</b>	0	<b>No. of Bachelor's Degrees:</b>	0
<b>No. of Bachelor's Candidates:</b>	0	<b>Monitoring Center:</b>	NASA ARC
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<b>Flight Program:</b>	Bion-M2		
<b>Flight Assignment:</b>	NOTE: End date corrected to 04/21/2025 per F. Hernandez/ARC (Ed., 2/12/25). NOTE: End date changed to 04/20/2025 per F. Hernandez/ARC (Ed., 6/14/24).		
<b>Key Personnel Changes/Previous PI:</b>			
<b>COI Name (Institution):</b>	Branscum, Adam Ph.D. ( Oregon State University ) Iwaniec, Urszula Ph.D. ( Oregon State University ) Wong, Carmen Ph.D. ( Oregon State University )		
<b>Grant/Contract No.:</b>	80NSSC20K0998		
<b>Performance Goal No.:</b>			
<b>Performance Goal Text:</b>			

	<p>Spaceflight results in increased fat infiltration into bone marrow, decreased hematopoiesis, and bone loss. The mechanisms leading to these detrimental changes are incompletely understood. We recently demonstrated increased non-shivering thermogenesis in brown adipose tissue (BAT) in mice sacrificed aboard International Space Station (ISS) following exposure to microgravity for 37 days. Thermo-regulation is critically important to maintain core body temperature within a narrow range. The sympathetic and sensory nervous systems regulate nonshivering thermogenesis in BAT. Chronic increases in non-shivering thermogenesis in response to increased sympathetic outflow from the hypothalamus lead to replacement of hematopoietic tissue in bone marrow with white adipose tissue (WAT) as well as bone loss. Based on these observations, we propose the novel hypothesis that increased non-shivering thermogenesis induced by elevated sympathetic signaling contributes to increased bone marrow adiposity, decreased hematopoiesis, and bone loss in mice during spaceflight.</p> <p>If increased non-shivering thermogenesis in mice plays a role in spaceflight-induced infiltration of bone marrow by fat and bone loss, there should be a strong post-landing association between restoration of normal thermoregulation and bone recovery. The rodent experiment design for the BION-M2 mission would provide an excellent platform for comprehensive analysis of relevant pathways regulating thermoregulation and bone metabolism and how these change during spaceflight and post-flight re-adaptation. To test our hypothesis we propose four Specific Aims.</p> <p>Specific Aim 1. Hypothalamus: Determine the effects of spaceflight and re-adaptation on expression of genes related to sympathetic outflow.</p> <p>Specific Aim 2. Brown adipose tissue (BAT): Determine the effects of spaceflight and re-adaptation on BAT histology and expression of genes related to non-shivering thermogenesis.</p> <p>Specific Aim 3. White adipose tissue (WAT): Determine the effects of spaceflight and re-adaptation on WAT histology and expression of genes related to WAT turnover.</p> <p>Specific Aim 4. Marrow adipose tissue (MAT): Determine the effects of spaceflight and re-adaptation on bone and MAT histology, lipid composition of bone marrow, and expression of genes related to bone cell and adipocyte turnover, and hematopoiesis. We will also characterize associations between changes in MAT, bone cells, and bone microarchitecture. We anticipate increased non-shivering thermogenesis in BAT as well as bone loss following spaceflight. Increased thermogenesis will be associated with increased sympathetic outflow from hypothalamus and WAT turnover. Bone loss will be associated with increased MAT, increased osteoclasts, and decreased hematopoiesis. During re-adaptation, we anticipate normalization of thermogenesis to occur prior to normalization of bone metabolism. Our analyses of histology, lipid composition of MAT, gene expression, and bone microarchitecture will provide insight into the specific pathways by which altered thermogenesis influences bone metabolism during spaceflight and during re-adaptation.</p> <p>The proposed research is relevant to NASA because it will contribute to a mechanistic understanding of bone loss during spaceflight.</p>
<b>Task Description:</b>	
<b>Rationale for HRP Directed Research:</b>	
<b>Research Impact/Earth Benefits:</b>	<p>We will investigate the time course for re-adaptation of thermoregulation following spaceflight in mice. The flight has not yet occurred so it is premature to know research impact/Earth benefits. However, we believe that this research will provide important information regarding how organisms re-adapt to Earth following being exposed to a particularly harsh orbital environment.</p>
<b>Task Progress:</b>	<p>NOTE: Per F. Hernandez/ARC, there is no additional progress to submit for this reporting period. The NASA Space Biology Program has indicated that the project is presently on hold (Ed., 4/8/2024).</p>
<b>Bibliography Type:</b>	Description: (Last Updated: 06/16/2025)