Role of Telomerase in Vascular Function and Exploration of Mitochondrial Dynamics: A Novel Approach to Treatment of Vascular Dysfunction

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Coronary Artery Disease (CAD), an aging related detriment, is the most common cause of mortality in the world. In addition, chemotherapy jeopardizes cardiovascular integrity and was shown to be interconnected with CAD via RNAseq. Telomerase, an anti-aging related enzyme, is a nuclear derived telomere regulator and was hypothesized to mitigate the vascular effects of aforementioned ailments. Following creation of a specialized videomicrocopy apparatus, global activation of telomerase resulted in restored vascular function amidst CAD and chemotherapy. Separate from its conventional nuclear role, telomerase was shown to translocate to the mitochondria and preserve mitochondrial function. As global overexpression of telomerase is known to be oncogenic, a novel, clinically applicable mitochondrial telomerase decoy peptide was created in order to preserve therapeutic effects with minimal oncogenic activity. The decoy peptide restored vascular function and preserved cellular integrity amidst CAD, chemotherapy and vascular stressors. In order to accelerate telomerase-related drug development, a unique computational method using a developed at-home computational cluster was developed. Moreover, an ultra precise mechanistic mathematical model of the mitochondrial dynamics was created in order to simulate and predict novel mitochondrial telomerase interactions as well as further explore mitochondrial systems. This multidisciplinary study presents a revolutionary mechanism to combat CAD and preserve vascular function amidst chemotherapy. Additionally, the developed platform technologies have implications with many other diseases, representing a promising advancement to the biomedical field.

Awards Won:

Dudley R. Herschbach SIYSS Award

First Award of $5,000

Intel ISEF Best of Category Award of $5,000