



**FALL 2023**

**Biochemistry and Molecular Biology  
Brown Bag Series**

**Gerone Sta Ana**

**4<sup>th</sup> Year Undergraduate Student**

*“Impacts of the benzo[a]pyrene, ultraviolet B, and solar simulated light radiation exposure on microvesicle particle generation from murine skin”*

**Tuesday, November 21, 2023**

**11:00 AM**

**135 Oelman Hall**

**Lab: Ravi Sahu, Ph.D.**



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<https://science-math.wright.edu/biochemistry-and-molecular-biology>

## **Abstract:**

### **Impacts of the benzo[a]pyrene, ultraviolet B, and solar simulated light radiation exposure on microvesicle particle generation from murine skin.**

The human skin is the first layer of defense against external and environmental insults that increase the potential for developing skin cancer. Human skin is constantly exposed to two major environmental insults, namely solar UV radiation also known as simulated solar light (SSL), a known carcinogen, and pollutants such as benzo[a]pyrene (BaP), a carcinogen present in smoke originating from forest fires, cigarettes, and burnt food, etc. Due to the prevalence of these hazards in daily life, it is important to understand the effects of BaP, SSL or ultraviolet B (UVB) exposure on skin tissue, and the mechanisms by which BaP and UVB/SSL mediate their effects in order to design relevant strategies to mitigate their effects. Epidemiological studies indicate that increased exposure to these environmental hazards simultaneously can augment the risk of contracting human malignancies such as skin, mucosal, and lung cancers. Our previous studies demonstrated that exposure to UVB radiation induces the generation of microvesicle particles (MVPs), nanosized extracellular vesicles from human and murine skin in a dose-dependent manner. Notably, these MVPs carry a potent phospholipid mediator, Platelet-activating factor-receptor (PAFR) agonists. Given that PAFR agonists play crucial roles in mediating pro-oxidative stressors, including UVB and therapeutic agents-induced systemic immunosuppression and augmentation of experimental cancer growth, the current studies sought to determine the impacts of topical exposures to BaP, UVB, and SSL on MVP release from PAFR-expressing murine skin. The data demonstrated that these environmental hazards induce increased MVP release in a dose-dependent manner. While studies to determine the combined effects of BaP+UVB and BaP+SSL as well as the involvement of the PAFR and MVP are ongoing in the WT, PAFR-deficient and acid sphingomyelinase (aSMase) enzyme deficient mice, the current studies are in agreement with the assumption that PAFR agonists-laden MVP release is one of the possible mechanisms in mediating these hazards-induced effects.