Spring 2026

Biochemistry and Molecular Biology Brown Bag Series

Divyanshu Aggarwal

"Characterization of the kinetics of solar simulating light vs ultraviolet A/B radiation on cellular response and extracellular vesicle release in human HaCaT keratinocytes"

Tuesday, January 20, 2026

11:00 AM

103 Biological Sciences Building

Lab: Ravi Sahu, Ph.D.

Boonshoft School of Medicine
WRIGHT STATE UNIVERSITY

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

Abstract

Ultraviolet (UV) radiation modulates keratinocyte biology through both cytotoxic and signaling-related mechanisms. However, the differential kinetics of UVA versus UVB in regulating extracellular vesicle (EV) release, as well as the underlying mechanisms, remain incompletely understood. In this study, we examined the effects of UVB, UVA, and solar-simulated light (SSL; emitting both UVA and UVB) on human HaCaT keratinocytes, with a particular focus on platelet-activating factor-receptor (PAFR) signaling. SSL and UVB induced dose- and time-dependent reductions in cell viability, whereas UVA caused minimal cytotoxicity at equivalent doses, indicating that the UVB component of SSL mediates cytotoxic responses. SSL significantly stimulated the release of large EVs (microvesicle particles, MVPs) at early time points and small EVs (exosomes) at later time points. Similar patterns were observed following UVB exposure but not UVA exposure. Pharmacological inhibition of PAFR significantly reduced SSL-induced release of both MVPs and exosomes. Furthermore, inhibition of acid sphingomyelinase (aSMase) or pretreatment with the antioxidant N-acetylcysteine attenuated EV release. Collectively, these findings validate PAFR- and aSMase-mediated signaling as key regulators of UVB-dependent keratinocyte EV release and provide mechanistic insight into how solar radiation influences intercellular communication in the skin.

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Riya Rawal

"Assessments of the effect of benzo[a]pyrene on cellular responses and targeted therapy efficacy in non-small cell lung cancer models"

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Abstract

Non–small cell lung cancer (NSCLC) is a leading cause of cancer-related mortality, driven by invasive behavior and frequent resistance to systemic therapies. Epidermal growth factor receptor tyrosine kinase inhibitors (EGFR-TKIs) benefit patients with EGFR-mutant NSCLC, but their efficacy is often limited by tumor-intrinsic and environmental resistance mechanisms. Benzo[a]pyrene (BaP), a ubiquitous polycyclic aromatic hydrocarbon from tobacco smoke, combustion, and dietary sources, is a known carcinogen; however, its role in modulating therapeutic responses is poorly understood. Studies, including ours, implicate platelet-activating factor-receptor (PAFR) pathway in mediating environmental pollutant– and therapy-induced effects on tumor growth and microvesicle particle (MVP) release. We hypothesized that PAFR activation mediates BaP-induced NSCLC progression and influences EGFR-TKI responses. We assessed the effects of BaP, PAFR agonist CPAF, EGFR-TKIs, and their combinations on cell viability, proliferation, migration, anchorage-independent growth, and MVP secretion. BaP did not alter cell survival but significantly increased migration, growth, colony formation, and MVP release, similar to CPAF, and these effects were blocked by a PAFR antagonist or acid sphingomyelinase inhibitor. Importantly, BaP did not reduce EGFR-TKI efficacy at tested concentrations. These results show that environmental carcinogen modulates NSCLC behavior through PAFR signaling without compromising EGFR-TKI responsiveness, highlighting PAFR as a potential therapeutic target.