Spring 2026

Biochemistry and Molecular Biology Brown Bag Series

Dipinkumar P U

"Exploring the Potential of Repurposing Loratadine, Rupatadine and Diphenhydramine in Non-Small Cell Lung Cancer"

Tuesday, January 27, 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

Non–small cell lung cancer (NSCLC) is the leading cause of lung cancer–related mortality worldwide, with therapeutic resistance remaining a major challenge. This resistance is often driven by genetic mutations in key signaling receptors and exacerbated by chronic inflammation within the tumor microenvironment. Emerging evidence suggests that repurposing anti-inflammatory and antihistaminergic drugs may offer therapeutic benefit. As epidermal growth factor receptor (EGFR) mutations are among the most common oncogenic alterations in NSCLC, this study examined the role of platelet-activating factor-receptor (PAFR) signaling in the effects of loratadine, rupatadine, and diphenhydramine, and evaluated their cytotoxic activity alone or in combination with EGFR tyrosine kinase inhibitors (gefitinib and erlotinib) in NSCLC cell lines. All three drugs reduced cell growth in a dose- and time-dependent manner and inhibited cell migration, effects that were reversed by PAFR activation. Co-treatment with a PAFR antagonist or EGFR-tyrosine kinase inhibitors (TKIs) enhanced cytotoxicity compared with monotherapy. These findings highlight the potential of repurposing antihistaminergic drugs and support multi-targeted therapeutic strategies to overcome drug resistance in NSCLC.

Keywords: Non-small cell lung cancer; Drug repurposing; Antihistaminergic drugs; Platelet-activating factor-receptor; Epidermal growth factor receptor; Tyrosine kinase inhibitors.

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Mittalkumari Shivsinh Chauhan

"Mechanisms of arsenic compounds-mediated inhibition of lung cancer growth and the efficacy of targeted therapies"

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Abstract

Repurposed agents are also being explored in combination with targeted therapies to improve treatment efficacy. However, the effects of repurposed agents such as arsenic compounds on epidermal growth factor receptor (EGFR)–based tyrosine kinase inhibitors (TKIs) in lung cancer remain poorly understood. Our previous studies identified a role for platelet-activating factor receptor (PAFR) signaling and PAFR-driven microvesicle particle (MVP) release in modulating tumor growth and therapeutic response. In the current study, we determined the role of PAFR and MVP pathways in arsenic compound–mediated modulation of EGFR-TKI efficacy in non-small cell lung cancer (NSCLC) models. PAFR-expressing NSCLC cells were treated with arsenic compounds, alone or in combination with EGFR-TKIs, and assessed for viability, cytotoxicity, migration, proliferation, colony formation and MVP release. We observed that arsenic treatment reduced cell viability and migration in a dose- and time-dependent manner and enhanced the antitumor effects of gefitinib and erlotinib. PAFR activation attenuated arsenic-induced inhibition of cell migration, while arsenic exposure increased MVP release in a PAFR- and acid sphingomyelinase–dependent manner. These findings indicate that arsenic compounds suppress tumorigenic properties while concurrently stimulating MVP secretion, likely as a stress response, and highlight the importance of PAFR–MVP signaling in regulating arsenic efficacy in NSCLC.