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Induced Macrophage M2 Polarization by Tumor-derived Exosomal miR-143-3p Leads to Radiation Resistance in Locally Advanced Esophageal Squamous Cell Carcinoma

Jamila Kenin*

Department of Molecular Genetics and Microbiology, Duke University School of Medicine, Durham, NC 27710, USA

Introduction

Esophageal Squamous Cell Carcinoma (ESCC) represents a significant global health burden due to its aggressive nature and often poor prognosis, particularly in locally advanced stages. Recent research has unveiled a critical aspect of tumor microenvironment interactions involving macrophages and tumor-derived exosomes, specifically focusing on the role of miR-143-3p in inducing macrophage polarization towards an M2 phenotype. This phenotypic shift is implicated in promoting radiation resistance, thereby complicating treatment strategies for ESCC. ESCC is a subtype of esophageal cancer characterized by its origin in the squamous cells lining the esophagus. It is prevalent in certain geographic regions such as parts of Asia, where dietary and environmental factors contribute significantly to its incidence. Locally advanced ESCC presents a formidable challenge in clinical management due to its invasive nature and propensity for metastasis, often necessitating multimodal treatment approaches that include radiotherapy [1].

Description

Macrophages are crucial components of the immune system with diverse functions in tissue homeostasis and inflammation. In the context of tumors, macrophages exhibit plasticity and can adopt different activation states, broadly classified as M1 and M2 phenotypes. M1 macrophages are pro-inflammatory and involved in anti-tumor responses, whereas M2 macrophages are immunosuppressive and promote tumor progression by facilitating angiogenesis, tissue remodeling, and immune evasion [2]. Exosomes are extracellular vesicles secreted by various cell types, including cancer cells, and play a vital role in intercellular communication. They contain proteins, lipids, and nucleic acids, including microRNAs (miRNAs), which can modulate gene expression in recipient cells. miRNAs such as miR-143-3p have been identified in tumor-derived exosomes and are implicated in regulating various cellular processes, including immune responses and cancer progression. miR-143-3p has been specifically identified as a key regulator in the crosstalk between ESCC cells and macrophages. It acts by targeting specific genes in macrophages that govern their polarization state, favoring the shift towards an M2 phenotype. This alteration in macrophage polarization is significant as M2 macrophages are known to promote tumor growth, metastasis, and importantly, resistance to radiation therapy [3].

***Address for Correspondence:** Jamila Kenin, Department of Molecular Genetics and Microbiology, Duke University School of Medicine, Durham, NC 27710, USA, E-mail: kenin@edu.com*

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Radiation therapy is a cornerstone of treatment for many solid tumors, including ESCC. However, resistance to radiation remains a major clinical challenge, contributing to treatment failure and disease recurrence. The involvement of M2 macrophages in mediating radiation resistance is multifaceted. M2 macrophages secrete cytokines and growth factors that can promote cancer cell survival and proliferation in the presence of radiation-induced stress. Additionally, they contribute to the establishment of an immunosuppressive microenvironment that shields tumor cells from immune-mediated cytotoxicity induced by radiation [4].

Understanding the molecular mechanisms underlying radiation resistance mediated by tumor-derived exosomal miR-143-3p and M2 macrophages provides insights into potential therapeutic strategies. Targeting miR-143-3p or its downstream effectors in macrophages could potentially reverse M2 polarization, restore anti-tumor immune responses, and enhance the efficacy of radiation therapy in ESCC. Moreover, the identification of biomarkers associated with M2 polarization and radiation resistance could facilitate patient stratification and personalized treatment approaches. Future research directions may include investigating combinatorial therapies that target both the tumor cells and the tumor microenvironment, including M2 macrophages, to overcome radiation resistance effectively. Additionally, exploring the impact of miR-143-3p and other exosomal cargo on other components of the tumor microenvironment could uncover broader implications for cancer biology and therapy [5].

Conclusion

In conclusion, the induction of M2 macrophage polarization by tumor-derived exosomal miR-143-3p represents a critical mechanism contributing to radiation resistance in locally advanced ESCC. This intercellular communication pathway highlights the complex interactions within the tumor microenvironment that influence treatment outcomes. Targeting this pathway holds promise for developing novel therapeutic strategies to enhance the effectiveness of radiation therapy and improve clinical outcomes for patients with ESCC. In conclusion, the convergence of immunoinformatics and mRNA vaccine technology represents a paradigm shift in infectious disease prevention, offering hope for controlling zoonotic threats like Influenza D virus and improving global health security. This intercellular communication underscores the intricate dynamics within the tumor microenvironment that shape treatment responses. Targeting this pathway offers potential for novel therapeutic approaches to augment radiation therapy efficacy and advance clinical outcomes in ESCC patients.

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Conflict of Interest

None.

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