Autophagic Flux Modulation in Oncogenic Stress: A Case Study Using Advanced Live-Cell Imaging Techniques in Triple-Negative Breast Cancer Cells

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Abstract. This study explores the dynamics of autophagic flux modulation in response to oncogenic stress within triple-negative breast cancer (TNBC) cells. Utilizing advanced live-cell imaging techniques, we observed significant alterations in autophagic activity, providing insights into cellular survival mechanisms. Our findings indicate that TNBC cells exhibit a unique autophagic response, potentially contributing to chemoresistance. Understanding these mechanisms paves the way for developing targeted therapies to overcome drug resistance in aggressive breast cancer subtypes.

Keywords: Autophagic Flux, Oncogenic Stress, Live-Cell Imaging, Triple-Negative Breast Cancer, Chemoresistance, Targeted Therapy, Cellular Homeostasis, Cancer Pathophysiology, Drug Resistance

Introduction

Autophagy, a catabolic process involving the degradation of cellular components, is a critical mechanism in maintaining cellular homeostasis. In cancer cells, particularly those in aggressive subtypes like triple-negative breast cancer (TNBC), autophagy can play a dual role, either promoting survival in stressful environments or leading to cell death. The global burden of TNBC remains substantial, posing significant challenges to effective treatment due to its aggressive nature and lack of hormone receptor targets. Oncogenic stress, a common phenomenon in cancer pathophysiology, results in cellular environments that induce autophagic responses, potentially contributing to chemoresistance. This study employs state-of-the-art live-cell imaging techniques to investigate the modulation of

autophagic flux in TNBC cells under oncogenic stress. By delineating the relationship between autophagic activity and cell survival, we aim to enhance the understanding of TNBC pathology and explore novel therapeutic strategies. The paper is structured to first address the background and significance of autophagy in cancer, followed by an in-depth analysis of our methodology and results, and concluding with potential implications for treatment strategies.

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