Advances in Cancer Prevention
ISSN: 2472-0429

Lung Cancer Progression is not always Halted by Voluntary Exercise

Prakseema Baxla*

Zurich Center for Integrative Human Physiology (ZIHP), University of Zurich, Switzerland

Abstract

Lung cancer remains a formidable global health challenge, characterized by diverse histological subtypes and varying clinical trajectories. This review comprehensively explores the intricate molecular and cellular mechanisms underlying the progression of lung cancer, encompassing non-small cell lung cancer (NSCLC) and small cell lung cancer (SCLC). By dissecting the key drivers of tumor initiation, metastasis, and treatment resistance, this analysis sheds light on potential therapeutic targets and strategies for improved patient outcomes. The evolution of lung cancer involves a complex interplay of genetic alterations, epigenetic modifcations, and dysregulated signaling pathways. Driver mutations, including alterations in EGFR, ALK, ROS1, and BRAF, delineate subgroups within NSCLC, driving tumor initiation and progression. Additionally, genomic instability, immune evasion, and angiogenic signaling contribute to the malignant phenotype.

Metastasis, a hallmark of advanced lung cancer, involves a cascade of events infuenced by tumor microenvironment components, including immune cells, fbroblasts, and extracellular matrix elements. The elucidation of molecular mediators and signaling pathways governing metastatic dissemination provides opportunities for targeted intervention and the development of novel therapeutics. Furthermore, the emergence of treatment resistance poses a signifcant clinical challenge. Molecular mechanisms such as target gene amplifcation, activation of bypass pathways, and acquired mutations contribute to therapeutic resistance in both NSCLC and SCLC. Understanding these resistance mechanisms is imperative for the design of combination therapies and the development of next-generation treatment strategies.

Immunotherapy, particularly immune checkpoint inhibitors, has revolutionized the treatment landscape for lung cancer. However, patient selection and the identifcation of predictive biomarkers remain critical for optimizing immunotherapeutic outcomes. Additionally, the integration of targeted therapies, immunotherapies, and conventional treatments in a multimodal approach holds promise for overcoming resistance and improving long-term survival. In conclusion, LQGLYLGXDOVDHFWHGEWKLVGHYDVWDWLQJGLVHDVH

*Corresponding author: Prakssema Baxla, Zurich Center for Integrative Human Physiology (ZIHP), University of Zurich, Switzerland, E-mail: pb.prakseema@baxla.com

Received: 04-Sep-2023, Manuscript No. acp-23-114216; **Editor assigned:** 06-Sep-2023, PreQC No. acp-23-114216 (PQ); **Reviewed:** 20-Sep-2023, QC No. acp-23-114216; **Revised:** 23-Sep-2023, Manuscript No. acp-23-114216 (R); **Published:** 30-Sep-2023; DOI: 10.4172/2472-0429.1000183

Citation: Baxla P (2023) Lung Cancer Progression is not always Halted by Voluntary Exercise. Adv Cancer Prev 7: 183.

complex interplay of genetic, epigenetic, and environmental factors.

In small cell lung cancer, which accounts for the majority of cases,

is characterized by diverse driver mutations, including alterations in

genes such as EGFR, ALK, ROS1, and BRAF. These genetic aberrations

confer distinct molecular subtypes, each with its own trajectory of

progression and potential targeted therapeutic strategies.

Actual activity safeguards against the turn of events and movement of a wide range of disease types. Mechanistically, exercise alters the

tumor microenvironment by targeting four physiological mechanisms:

(1) the oxygenation and vascularization of the tumor, (2) anaerobic

cancer metabolism, (3) the production of myokines in activated

muscles, and (4) the activation of immune cells that suppress tumor

ly that two or more of these pathways are activated

simultaneously during exercise and wo

and development of tumors. For instanc

practicing muscles or the arrival of the

from adrenal organs initiates cytotoxic,

or regular executioner cells, individua

discharge cytokines (myokines) that d

crosstalk or enact growth suppressive

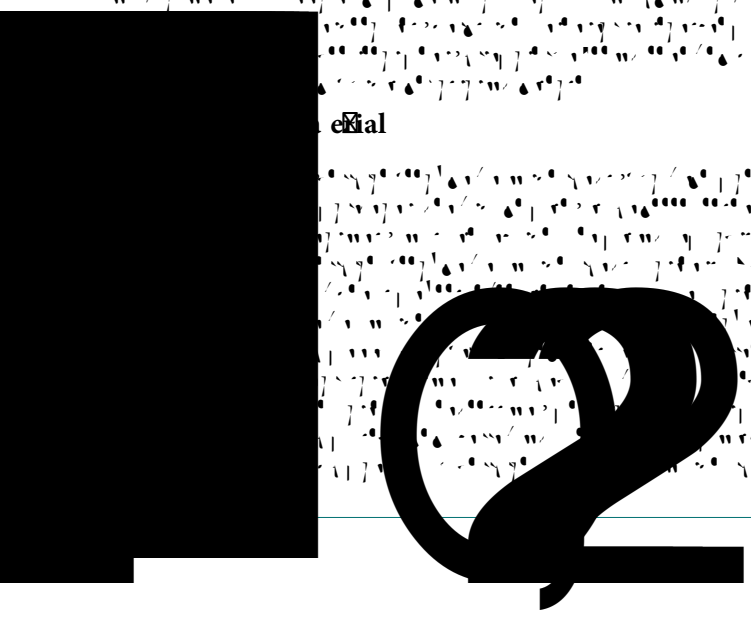
including musclin, irisin, BARC, interleu

factor, have been shown to directly

instance, they can target the metabolis

proliferation, prevent metastasis, or i

myokines, for example, interleukin, min



Re l and Di c ion

Ackn ledgemen

Con ic of In e

References

1. Baralt L,Weitz TA (2012) The Komen–planned parenthood controversy: Bringing the politics of breast cancer advocacy to the forefront. Womens Health Issues 22: 509-512.
2. Bob Roehr (2012) Charity's decision to cut funding to Planned Parenthood sparks controversy. BMJ 344: e870.
3. Fontana RS, Sanderson DR, Woolner LB, Taylor WF, Miller WE, et al. (1986) Lung cancer screening: the Mayo program. J Occup Med US 28:746-750.
4. McKinney SM, Sieniek M, Godbole V, Godwin J, Antropova N, et al. (2020). International evaluation of an AI system for breast cancer screening. Nature 577: 89-94.
5. Secretan BL, Loomis D, Straif K (2015) Breast-cancer screening-viewpoint of the IARC Working Group. N Engl J Med 373: 1479.
6. Hamashima C, Shibuya D, Yamazaki H, Inoue K, Fukao A, et al. (2008) The Japanese guidelines for gastric cancer screening. Jpn J Clin Oncol 38: 259-267.
7. Sabatino SA, White MC, Thompson TD, Klabunde NC (2015) Cancer screening test use: United States, 2013. MMWR Morb Mortal Wkly Rep 64: 464-8.
8. White A, Thompson TD, White MC, Sabatino SA, Moor JD, et al. (2017) Cancer Screening Test Use - United States, 2015. MMWR Morb Mortal Wkly Rep 66: 201-206.
9. Horner-Johnson W, Dobbertin K, Andresen EM, Iezzoni LI, et al. (2014) Breast and cervical cancer screening disparities associated with disability severity. Womens Health Issues 24: e147-53.
10. Horner-Johnson W, Dobbertin K, Iezzoni LI (2015) Disparities in receipt of breast and cervical cancer screening for rural women age 18 to 64 with disabilities. Womens Health Issues 25: 246-53.