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Review

Oncogenic Signaling Pathways in Tumor Progression: An Update on PI3K/AKT/MTOR and MAPK axis

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Abstract:
Oncogenic signaling pathways play a crucial role in the initiation, progression, and metastasis of various cancers. Among these, the PI3K/AKT/mTOR and MAPK signaling axes are frequently implicated in tumorigenesis and are considered key drivers of tumor cell survival, proliferation, and resistance to therapy. The PI3K/AKT/mTOR pathway regulates cellular processes such as growth, metabolism, and survival, while the MAPK pathway, comprising multiple kinases like ERK, JNK, and p38, is involved in cell differentiation, migration, and stress responses. Dysregulation of these pathways often results in uncontrolled cellular proliferation and metastasis. Recent advances in understanding the molecular mechanisms underlying these signaling pathways have led to the development of targeted therapies aimed at inhibiting specific components within these pathways. This paper provides a comprehensive update on the role of the PI3K/AKT/mTOR and MAPK axes in tumor progression, focusing on their molecular interactions, functional implications in cancer, and the emerging therapeutic strategies targeting these pathways. Understanding these signaling networks is critical for developing more effective treatments for cancer patients.

Keywords: Oncogenic signaling, PI3K/AKT/mTOR pathway, MAPK pathway, tumor progression, cancer therapy, molecular mechanisms, targeted therapies, cellular proliferation, metastasis, kinase inhibitors.

1.1 Introduction:

Cancer remains one of the leading causes of morbidity and mortality worldwide, characterized by uncontrolled cell growth, resistance to apoptosis, and the ability to invade surrounding tissues and metastasize to distant organs. The progression of cancer is driven by complex alterations in cellular signaling networks that govern essential processes such as cell cycle regulation, survival, differentiation, metabolism, and migration. Among the most critical signaling pathways involved in tumorigenesis are the PI3K/AKT/mTOR and MAPK axes, which have emerged as key players in promoting tumor initiation, progression, and resistance to therapies.(1)

The PI3K/AKT/mTOR pathway is frequently altered in a wide variety of cancers, driving processes like cell proliferation, growth, and survival by regulating metabolic pathways and inhibiting apoptosis. Dysregulation of this pathway is often caused by mutations, amplifications, or deletions in genes encoding the pathway's components, leading to constitutive activation. Similarly, the MAPK pathway, which encompasses several downstream signaling cascades such as the ERK, JNK, and p38 pathways, plays a vital role in controlling cellular responses to extracellular signals, including those involved in stress, inflammation, and growth factors. Aberrant activation of MAPK signaling, through mutations or overexpression of upstream components, contributes to cancer progression by promoting cell division, survival, and metastasis.(2)

These signaling pathways not only drive the progression of many cancers but also confer resistance to conventional therapies such as

chemotherapy and radiation. Targeting the components of the PI3K/AKT/mTOR and MAPK pathways has therefore become a focus of therapeutic development. However, the development of effective targeted therapies has been challenging due to the redundancy and complexity of these signaling networks.

This review provides an updated overview of the PI3K/AKT/mTOR and MAPK signaling pathways, their role in cancer biology, and the therapeutic strategies aimed at targeting these pathways. By deepening our understanding of these crucial signaling axes, we aim to highlight their potential as therapeutic targets and improve the clinical management of cancer patients.

1.2 Cancer as a Major Global Health Challenge:

Cancer remains one of the leading causes of death worldwide, affecting millions of people across different age groups and populations. With an increasing incidence rate, cancer has become a significant public health issue, placing a heavy burden on healthcare systems globally.(3) The World Health Organization (WHO) estimates that nearly one in six deaths is attributed to cancer, and the number of new cases is expected to rise as populations age and lifestyle factors such as smoking, diet, and physical inactivity continue to influence cancer rates. Early detection, improved treatment strategies, and better understanding of cancer biology have contributed to some progress, yet many cancers remain difficult to treat and cure. The complexity of cancer's molecular mechanisms, coupled with challenges such as metastasis and resistance to therapies, underscores the need for continuous research into its causes and more effective treatment options.(4)

1.3 The Role of Signaling Pathways in Cancer Progression:

Signaling pathways are essential for the regulation of cell growth, survival, differentiation, and metabolism. In normal cells, these pathways are tightly regulated to ensure proper cellular function. However, in cancer cells, aberrant activation or inactivation of specific signaling pathways can lead to uncontrolled cell proliferation, survival, and metastasis, contributing to tumor progression.(5) Dysregulation of signaling pathways often results from mutations in genes encoding for key components such as receptors, kinases, and tumor suppressors. These abnormalities disrupt the delicate balance between cell division and apoptosis, leading to sustained tumor growth and resistance to chemotherapy or other treatment modalities. Key oncogenic signaling pathways, such as PI3K/AKT/mTOR and MAPK, have been shown to be frequently altered in a wide variety of cancers, making them critical drivers of cancer progression and potential therapeutic targets.(6)

1.4 Overview of Oncogenic Signaling Pathways:

Oncogenic signaling pathways are complex networks of proteins and enzymes that control cellular processes involved in cancer initiation and progression. These pathways are activated by external signals like growth factors, hormones, and extracellular matrix components, which bind to cell surface receptors and trigger intracellular signaling cascades.(7) In cancer, these pathways become dysregulated, either through mutations in their components or abnormal activation by other factors, leading to enhanced tumor cell growth, survival, and spread. The PI3K/AKT/mTOR and MAPK pathways are among the most commonly dysregulated signaling networks in cancer cells. These pathways mediate various cellular functions, including cell proliferation, survival, metabolism, and migration, all of which are vital for cancer progression. A better understanding of these signaling pathways offers valuable insight into the molecular mechanisms driving cancer and provides opportunities for targeted therapeutic interventions aimed at disrupting these aberrant signaling circuits.(8)

1.5 Introduction to PI3K/AKT/mTOR Signaling Pathway:

The PI3K/AKT/mTOR signaling pathway is one of the most frequently altered pathways in cancer and plays a critical role in regulating cell growth, survival, metabolism, and protein synthesis. This pathway is initiated when receptor tyrosine kinases (RTKs) on the cell surface are activated by extracellular growth factors. Upon activation, PI3K (phosphoinositide 3-kinase) is recruited to the plasma membrane, where it catalyzes the conversion of phosphatidylinositol-4,5-bisphosphate (PIP2) to phosphatidylinositol-3,4,5-trisphosphate (PIP3). PIP3 then activates AKT, a serine/threonine kinase, which in turn phosphorylates a wide array of downstream targets involved in cell survival, metabolism, and growth. The mTOR (mechanistic target of rapamycin) complex, activated by AKT, controls protein synthesis and cell growth.(9) Dysregulation of this pathway, through mutations or

amplifications of PI3K, AKT, or mTOR, leads to uncontrolled cell proliferation and survival, often promoting tumorigenesis. Because of its pivotal role in cancer, the PI3K/AKT/mTOR pathway has become a target for therapeutic strategies aimed at inhibiting its components to control tumor growth and sensitizing tumors to chemotherapy.(10)

1.6 Mechanisms of PI3K/AKT/mTOR Pathway in Cancer:

The PI3K/AKT/mTOR pathway plays a central role in regulating key cellular processes such as growth, metabolism, survival, and protein synthesis. In normal cells, this pathway is activated by external signals, such as growth factors binding to receptor tyrosine kinases (RTKs) on the cell surface.(21) However, in cancer cells, various mutations, amplifications, or loss of tumor suppressor genes often lead to the aberrant activation of this pathway, contributing to uncontrolled cell proliferation, survival, and metastasis. PI3K, when activated by RTKs, generates PIP3 (phosphatidylinositol-3,4,5-trisphosphate), which recruits and activates AKT (also known as protein kinase B). AKT, in turn, phosphorylates numerous downstream targets that promote cell survival by inhibiting pro-apoptotic proteins, stimulating cell cycle progression, and enhancing glucose metabolism for tumor growth. The mTOR complex, which is activated by AKT, regulates protein synthesis and cellular growth through the phosphorylation of key components like S6K and 4E-BP1. Dysregulation of this pathway can result in resistance to therapies and contribute to the metastatic potential of tumors, making it a critical target for therapeutic intervention in cancer treatment.(22)

1.7 The MAPK Signaling Pathway and Its Role in Tumorigenesis:

The MAPK (Mitogen-Activated Protein Kinase) signaling pathway is essential for regulating cell proliferation, differentiation, migration, and survival, making it a crucial player in tumorigenesis. This pathway consists of a cascade of protein kinases, including the ERK (extracellular signal-regulated kinase), JNK (c-Jun N-terminal kinase), and p38 MAPK. Upon stimulation by growth factors, cytokines, or stress signals, RTKs or G-protein-coupled receptors (GPCRs) activate small GTPases such as Ras, which then trigger a series of phosphorylation events through MAP3K (MAP kinase kinase kinase) and MAP2K (MAP kinase kinase) to activate MAPKs.(23) The ERK pathway is particularly associated with cell proliferation and survival, while JNK and p38 MAPK are involved in stress responses and apoptosis. In cancer, the MAPK pathway is frequently dysregulated, often due to mutations in Ras, BRAF, or other upstream components. This dysregulation leads to constitutive activation of MAPK signaling, contributing to uncontrolled cell division, survival, migration, and invasion, all of which are hallmarks of cancer. As such, targeting the MAPK pathway has become a prominent focus for cancer therapy, though challenges such as pathway redundancy and resistance to inhibitors remain significant obstacles.(24)

1.8 Activation of the PI3K/AKT/mTOR Pathway in Cancer Cells:

In cancer cells, the activation of the PI3K/AKT/mTOR pathway is often the result of genetic alterations that lead to the loss of regulation, allowing for uncontrolled cellular processes. Mutations in receptor tyrosine kinases (RTKs), such as EGFR (epidermal growth factor receptor), or mutations in downstream components like PI3K, AKT, or mTOR itself, can result in constitutive activation of this pathway.(25) This pathway can also be activated by mutations in the tumor suppressor PTEN (phosphatase and tensin homolog), which normally acts to inhibit PIP3 levels, thus preventing the activation of AKT. Once activated, AKT phosphorylates a wide array of targets involved in promoting cell survival, inhibiting apoptosis, and facilitating cell cycle progression. Additionally, the mTOR complex, regulated by AKT, controls protein synthesis, cellular metabolism, and autophagy, all of which are critical for sustaining tumor growth. In cancer, hyperactivation of this pathway results in increased cell proliferation, enhanced tumor cell survival, resistance to chemotherapy, and the ability to invade surrounding tissues. The dysregulated PI3K/AKT/mTOR pathway thus plays a pivotal role in cancer progression and metastasis, making it a promising target for therapeutic strategies aimed at inhibiting its components.(26)

1.9 Dysregulation of the MAPK Pathway in Cancer Progression:

The MAPK pathway is often dysregulated in cancer, contributing to the transformation of normal cells into malignant ones. Dysregulation can occur through mutations in various components of the pathway, such as the Ras or BRAF oncogenes,

which are frequently found in cancers like melanoma, colorectal, and lung cancer. These mutations lead to the constitutive activation of the MAPK pathway, driving unchecked cell proliferation, survival, and migration. Ras mutations are particularly common in many human cancers, resulting in persistent activation of downstream kinases like MEK and ERK. This uncontrolled activation of the MAPK cascade leads to altered cellular responses, including the promotion of tumorigenic behavior such as increased cell division, resistance to apoptosis, and enhanced motility, all of which contribute to cancer progression.(27) The MAPK pathway also plays a critical role in the metastatic potential of tumors, as it regulates the expression of genes involved in epithelial-to-mesenchymal transition (EMT), a process by which tumor cells acquire invasive properties. Moreover, crosstalk between the MAPK and PI3K/AKT/mTOR pathways further amplifies oncogenic signaling, creating a robust network that supports cancer cell survival and expansion. Targeting the MAPK pathway holds great promise for therapeutic intervention in cancers driven by these mutations, though challenges remain in overcoming resistance mechanisms.(28)

1.10 Cross-Talk Between PI3K/AKT/mTOR and MAPK Pathways:

The PI3K/AKT/mTOR and MAPK signaling pathways are often interconnected, forming a complex network that regulates various cellular functions. Cross-talk between these pathways can amplify or modulate cellular responses to external stimuli and influence cancer progression. For instance, both pathways can be activated simultaneously by growth factors such as epidermal growth factor (EGF) and insulin-like growth factor (IGF), which engage both receptor tyrosine kinases and G-protein-coupled receptors (GPCRs). (29)The activation of one pathway can enhance the signaling of the other, leading to synergistic effects that promote cell proliferation, survival, and migration. For example, AKT activation can enhance the activity of the MAPK pathway through the phosphorylation of certain components, such as Raf or MEK. Conversely, MAPK activation can upregulate the PI3K/AKT/mTOR pathway by increasing the expression of RTKs or by modifying the activity of PI3K. This cross-talk ensures that cells can respond dynamically to various environmental cues, but when dysregulated, it can lead to aberrant growth, metastasis, and therapy resistance in cancer cells. Understanding the interactions between these two pathways is essential for developing therapeutic strategies that target both simultaneously to overcome resistance mechanisms and improve treatment outcomes.(30)

1.11 Impact of PI3K/AKT/mTOR on Cellular Metabolism and Growth:

The PI3K/AKT/mTOR pathway plays a fundamental role in regulating cellular metabolism and growth, making it critical for cancer progression. Activation of this pathway promotes anabolic processes such as protein synthesis, lipid biosynthesis, and nucleotide production, all of which are essential for cell division and tumor growth. AKT, a central component of the pathway, regulates key metabolic enzymes that control glucose uptake, glycolysis, and oxidative phosphorylation. (11)In cancer cells, the PI3K/AKT/mTOR pathway promotes a shift toward aerobic glycolysis (the Warburg effect), where cells preferentially use glucose to generate energy, even in the presence of oxygen. This metabolic reprogramming supports the rapid energy demands of tumor cells. Furthermore, mTOR regulates the synthesis of proteins involved in cell growth, including ribosomal proteins and translation factors, thus directly impacting cellular growth and proliferation. By controlling nutrient sensing and metabolism, the PI3K/AKT/mTOR pathway ensures that tumor cells can adapt to nutrient-poor environments, resist metabolic stress, and continue to proliferate, making it an attractive target for cancer therapy aimed at disrupting cellular metabolism and limiting tumor growth.(12)

1.12 MAPK Pathway in Cellular Migration and Invasion:

The MAPK pathway, particularly through the ERK signaling branch, plays a critical role in cellular migration and invasion, processes that are key to cancer metastasis. Upon activation by growth factors or other external signals, the MAPK pathway leads to the activation of transcription factors like c-Myc and AP-1, which regulate the expression of genes involved in cytoskeletal rearrangement and cell motility.(13) Additionally, the MAPK pathway regulates the production of matrix metalloproteinases (MMPs), enzymes that degrade the extracellular matrix (ECM) and facilitate tumor cell invasion into surrounding tissues. This invasive behavior is one of the hallmarks of cancer and contributes to tumor spread to distant organs.

Moreover, the MAPK pathway also promotes epithelial-to-mesenchymal transition (EMT), a process by which epithelial cells lose their cell-cell adhesion and gain mesenchymal characteristics, enhancing their migratory and invasive potential. In sum, the MAPK pathway orchestrates multiple mechanisms that enable cancer cells to detach from the primary tumor, invade surrounding tissues, and eventually spread to distant sites, making it a central player in tumor metastasis.(14)

1.13 PI3K/AKT/mTOR and MAPK Pathways in Tumor Metastasis:

Both the PI3K/AKT/mTOR and MAPK signaling pathways are critically involved in tumor metastasis, the process by which cancer cells spread from the primary tumor to distant organs. These pathways regulate a variety of processes that support metastasis, including cell survival, migration, invasion, and the ability to colonize distant tissues. The PI3K/AKT/mTOR pathway helps tumor cells survive in hostile microenvironments by promoting metabolic reprogramming and resistance to apoptosis.(15) It also contributes to the remodeling of the extracellular matrix and enhances the invasive potential of cancer cells. On the other hand, the MAPK pathway facilitates cellular migration, invasion, and EMT, which are essential for the dissemination of cancer cells from the primary site. Additionally, the cross-talk between the PI3K/AKT/mTOR and MAPK pathways further amplifies these metastatic processes, creating a robust signaling network that promotes tumor cell survival and spread. Together, the dysregulation of these pathways enhances the metastatic potential of cancer, making them key targets for therapies designed to prevent or limit metastasis in cancer patients.(16)

1.14 Therapeutic Targeting of PI3K/AKT/mTOR and MAPK Pathways:

Given the critical roles of the PI3K/AKT/mTOR and MAPK pathways in cancer progression, they have become attractive targets for therapeutic intervention. Inhibitors targeting components of these pathways are being developed to block tumor cell growth, survival, and metastasis. For the PI3K/AKT/mTOR pathway, a range of small molecule inhibitors, including PI3K inhibitors (e.g., idelalisib), AKT inhibitors (e.g., ipatasertib), and mTOR inhibitors (e.g., rapamycin and its analogs), have shown promise in preclinical and clinical studies. These therapies aim to block aberrant signaling in tumor cells and reverse resistance to traditional treatments like chemotherapy.(17) Similarly, targeted therapies aimed at inhibiting the MAPK pathway, particularly in tumors with mutations in Ras or BRAF, such as BRAF inhibitors (e.g., vemurafenib) and MEK inhibitors (e.g., trametinib), have shown significant clinical benefits in certain cancers, particularly melanoma. However, despite these advances, resistance to these therapies remains a major challenge, often due to feedback loops, pathway redundancy, and cross-talk between the PI3K/AKT/mTOR and MAPK pathways. Combination therapies targeting both pathways are being explored to overcome resistance and improve therapeutic outcomes in cancer treatment.(18)

1.15 Challenges in Targeting PI3K/AKT/mTOR and MAPK Pathways:

Despite the potential of targeting the PI3K/AKT/mTOR and MAPK pathways in cancer therapy, several challenges exist. One of the primary obstacles is the development of resistance, which can arise through various mechanisms, such as mutations in the target proteins, activation of compensatory signaling pathways, or alterations in downstream effectors. For example, prolonged inhibition of one pathway can lead to the activation of alternative survival pathways, including upregulation of the other pathway (PI3K/AKT/mTOR or MAPK), creating a feedback loop that diminishes the effectiveness of monotherapy. Another challenge is the redundancy and complexity of these signaling networks, which can make it difficult to selectively target specific components without affecting normal cellular processes.(19) Moreover, the tumor microenvironment, which can alter the sensitivity of cancer cells to targeted inhibitors, poses an additional layer of complexity. Off-target effects, toxicity, and the inability to effectively target certain mutations also contribute to the limitations of current therapies. To overcome these challenges, combination therapies, personalized medicine approaches, and the development of next-generation inhibitors that target multiple components of these pathways are being actively researched to improve clinical outcomes for cancer patients.(20)

Signaling Pathway	Role in Cancer Progression	Targeted Therapeutic Strategies

PI3K/AKT/mTOR	Regulates cell growth, survival, metabolism, and protein synthesis. Aberrant activation leads to uncontrolled cell proliferation and survival.	PI3K inhibitors, AKT inhibitors, mTOR inhibitors (e.g., rapamycin).
MAPK	Regulates cell proliferation, migration, and invasion. Dysregulation through mutations contributes to metastasis and resistance.	MEK inhibitors, BRAF inhibitors (e.g., vemurafenib), ERK inhibitors.
Cross-Talk Between PI3K/AKT/mTOR and MAPK	Both pathways influence each other, amplifying oncogenic signaling and promoting tumor cell survival and resistance to therapy.	Combination therapies targeting both PI3K/AKT/mTOR and MAPK pathways.
PI3K/AKT/mTOR in Tumor Metastasis	Promotes cell survival and migration, facilitating tumor growth in distant organs. Targeting components of the pathway can block metastatic potential.	mTOR inhibitors and AKT inhibitors can potentially block metastasis by reversing metabolic advantages.
MAPK in Tumor Metastasis	Increases cell migration and invasion, often through activation of EMT and MMPs. Dysregulation enhances metastatic spread of tumors.	MEK inhibitors (trametinib) and ERK inhibitors for metastatic cancers.
Therapeutic Targeting of PI3K/AKT/mTOR	Inhibitors targeting PI3K/AKT/mTOR can block tumor growth by reversing metabolic reprogramming and reducing resistance to apoptosis.	PI3K/AKT/mTOR inhibitors for reversing growth signals and enhancing chemotherapy effectiveness.
Therapeutic Targeting of MAPK	MAPK inhibitors target mutated components (e.g., Ras, BRAF), leading to decreased tumor proliferation and survival, especially in melanoma and other cancers.	BRAF and MEK inhibitors to prevent Ras-MAPK pathway activation, often in combination for more effective results.

CONCLUSION:

The PI3K/AKT/mTOR and MAPK signaling pathways are integral to the regulation of various cellular functions, and their dysregulation is a hallmark of cancer progression. Both pathways play pivotal roles in driving tumorigenesis, metastasis, and resistance to therapies by promoting uncontrolled cell growth, survival, migration, and invasion. The cross-talk between these pathways further amplifies their oncogenic effects, making them critical drivers of tumor progression. Targeting these pathways through specific inhibitors has emerged as a promising therapeutic strategy for cancer treatment. However, challenges such as pathway redundancy, compensatory signaling, and therapy resistance limit the success of current treatments. The complexity of these networks necessitates the development of combination therapies, where inhibitors of both pathways can work synergistically to overcome resistance and improve treatment efficacy. As our understanding of the molecular mechanisms underlying these pathways deepens, novel strategies to target these signaling axes more effectively will likely offer new avenues for more personalized and effective cancer therapies. The continuous exploration of the PI3K/AKT/mTOR and MAPK pathways in cancer research is crucial to advancing our ability to combat this complex and devastating disease.

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