Signaling pathways: Disease, targets, and intervention.

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Introduction

This review details various signal transduction pathways frequently dysregulated in cancer, such as PI3K/Akt/mTOR, MAPK/ERK, JAK/STAT, and Wnt/β-catenin. It explores current therapeutic strategies, including small molecule inhibitors and biological agents, that target these pathways to inhibit tumor growth, metastasis, and enhance sensitivity to other treatments. The article emphasizes the complexity of these networks and the potential for combination therapies to overcome resistance.[1]

This article provides a comprehensive overview of the signaling pathways involved in neuroinflammation, a critical process in neurodegenerative diseases. It discusses how activation of specific receptors and downstream cascades, like NF-κB, MAPK, and inflammasome pathways, contributes to the inflammatory response in the brain. The review highlights emerging therapeutic strategies that modulate these signal transduction mechanisms to mitigate neuroinflammation and protect neuronal health.[2]

This review delves into the mTOR signaling pathway, a central hub integrating nutrient availability, growth factors, and stress signals to regulate cell growth, proliferation, and metabolism. It outlines the structure and function of mTORC1 and mTORC2 complexes, their upstream activators, and downstream effectors. The article also discusses the implications of mTOR dysregulation in various diseases, including cancer and metabolic disorders, and highlights therapeutic targeting strategies.[3]

This article reviews the latest developments in understanding G protein-coupled receptor (GPCR) signal transduction, emphasizing their roles as crucial drug targets. It covers the diverse mechanisms of GPCR activation, G protein coupling, and β-arrestin mediated signaling, along with their intricate regulation. The review discusses how these insights are being leveraged for the rational design of new drugs, including allosteric modulators and biased agonists, to achieve more precise therapeutic outcomes with fewer side effects.[4]

This review explores the complex signal transduction networks in plants that enable them to perceive and respond to various environmental stresses, such as drought, salinity, and extreme temperatures. It covers key signaling molecules like phytohormones (ABA, ethylene, auxins), reactive oxygen species (ROS), and calcium, and discusses their roles in activating downstream transcription factors and gene expression changes. Understanding these pathways is crucial for developing stress-tolerant crops.[5]

This article examines the intricate signal transduction pathways that govern immune cell activation, differentiation, and function. It delves into the roles of T cell receptor (TCR), B cell receptor (BCR), and cytokine receptor signaling, highlighting how these pathways coordinate immune responses. The review emphasizes the therapeutic potential of targeting specific signaling molecules, like kinases and phosphatases, to modulate immune function in autoimmune diseases, inflammation, and cancer immunotherapy.[6]

This review discusses the complex signal transduction networks that initiate and maintain cellular senescence, a state of irreversible cell cycle arrest associated with aging and various diseases. It details key pathways such as p53/p21, p16/Rb, and DNA damage response (DDR), along with the senescence-associated secretory phenotype (SASP) and its signaling components. The article explores how targeting these pathways holds promise for developing senolytic and senomorphic therapies to combat age-related pathologies.[7]

This article provides an in-depth review of the Notch signaling pathway, a highly conserved cell-to-cell communication system critical for embryonic development, tissue homeostasis, and cell fate decisions. It outlines the canonical Notch activation mechanism involving ligand-receptor interaction, proteolytic cleavage, and nuclear translocation of the intracellular domain. The review also discusses the dysregulation of Notch signaling in various diseases, including cancer and developmental disorders, and highlights its potential as a therapeutic target.[8]

This review explores the fundamental principles of cytokine signal transduction, focusing on pathways like JAK/STAT, MAPK, and PI3K/Akt that mediate the diverse biological effects of cytokines. It discusses how cytokine-receptor interactions trigger specific intracellular cascades, leading to gene expression changes and modulation of cellular functions in immunity, inflammation, and hematopoiesis. The article also highlights the clinical relevance

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of targeting these pathways for treating inflammatory diseases, autoimmune disorders, and cancer.[9]

This review focuses on the signal transduction pathways activated in response to endoplasmic reticulum (ER) stress, a cellular condition resulting from accumulation of misfolded proteins in the ER lumen. It elaborates on the unfolded protein response (UPR) branches, including PERK, IRE1, and ATF6 pathways, and their roles in restoring ER homeostasis or initiating apoptosis if stress is prolonged. The article connects ER stress signaling to the pathogenesis of various diseases, such as neurodegeneration, metabolic disorders, and cancer, suggesting therapeutic opportunities.[10]

Conclusion

Signal transduction pathways are fundamental biological mechanisms that govern diverse cellular processes and physiological responses across living systems. In human health, their dysregulation is frequently implicated in critical diseases. For example, pathways like PI3K/Akt/mTOR, MAPK/ERK, JAK/STAT, and Wnt/β-catenin are often aberrantly activated in cancer, making them prime targets for small molecule inhibitors and biological agents aimed at controlling tumor growth and metastasis. Neuroinflammation, a significant factor in neurodegenerative conditions, involves specific cascades such as NF-κB and MAPK, and modulation of these pathways offers new therapeutic avenues. The mTOR pathway, a master regulator of cell growth and metabolism, is also central, with its dysfunction linked to cancer and metabolic disorders. G protein-coupled receptors (GPCRs) represent crucial drug targets, where advances in understanding their signaling mechanisms lead to the design of more precise drugs with fewer side effects. Beyond human diseases, signal transduction is vital for plant responses to environmental stresses like drought and salinity, involving molecules such as phytohormones and reactive oxygen species. Immune cell function, including activation and differentiation, relies heavily on pathways like T cell receptor, B cell receptor, and cytokine signaling, which are increasingly targeted for autoimmune diseases and cancer immunotherapy. Aging-related pathologies and diseases are also tied to cellular senescence, a state maintained by networks like p53/p21 and p16/Rb, presenting opportunities for senolytic therapies. The Notch pathway plays a conserved role in development and tissue homeostasis, while endoplasmic reticulum (ER) stress activates the Unfolded Protein Response (UPR) in response to misfolded proteins, impacting neurodegeneration and metabolic disorders. Collectively, these studies emphasize the complexity of signaling networks and their immense potential as targets for therapeutic intervention across a broad spectrum of conditions.

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