

## Actin Cytoskeleton and Disease: Disruptions in Cell Function and Pathology

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### DESCRIPTION

The actin cytoskeleton is an essential component of the cellular framework, playing critical roles in maintaining cell shape, enabling movement, facilitating intracellular transport, and coordinating cell division. Composed of actin filaments, this dynamic network is highly regulated by various actin-binding proteins and signaling pathways.

### Function

Actin filaments are key components of the cytoskeleton, the protein network that provides structure and shape to cells. These filaments are highly dynamic, undergoing polymerization and depolymerisation in response to various internal and external cues. Actin filaments form a dense network beneath the plasma membrane, helping to maintain the cell's shape and mechanical integrity. Actin dynamics are important for processes like cell migration, wound healing, and the immune response. Actin polymerization at the leading edge of migrating cells drives the protrusions known as lamellipodia and filopodia. Actin filaments serve as tracks for the transport of organelles, vesicles, and proteins within the cell, in cooperation with motor proteins like myosin. During cell division, actin filaments form the contractile ring that facilitates the separation of daughter cells.

Cancer cells exhibit abnormal regulation of actin dynamics, which contributes to their ability to invade surrounding tissues and metastasize. Actin filament rearrangements allow tumor cells to migrate through tissues, invade the extracellular matrix, and enter the bloodstream or lymphatic system. Actin-binding proteins like cofilin (which severs actin filaments) and Arp2/3 (which nucleates actin branching) play critical roles in this process. Aberrant regulation of these proteins can lead to uncontrolled cell migration and invasion. Alterations in actin also affect cell adhesion, which is essential for the formation of metastatic tumors.

The transition of epithelial cells to mesenchymal-like cells is a key event in tumor progression, and actin plays a central role in this process. The reorganization of actin filaments leads to the

loss of cell-cell adhesion and the gain of migratory and invasive properties, promoting metastasis.

In neurodegenerative diseases such as Alzheimer's Disease (AD), Parkinson's Disease (PD), and Amyotrophic Lateral Sclerosis (ALS), disruptions in actin dynamics contribute to neuronal dysfunction and cell death. The actin cytoskeleton is critical for maintaining the structural integrity of neurons, facilitating synaptic plasticity, and enabling axonal transport. Neurons rely on actin filaments to transport essential cargo, including organelles, vesicles, and signaling molecules. In diseases like Alzheimer's, the accumulation of toxic proteins (e.g., tau tangles or  $\alpha$ -synuclein in PD) can interfere with actin polymerization and motor protein function, leading to defects in axonal transport. This disruption results in neuronal dysfunction and degeneration. In Alzheimer's disease, alterations in actin regulation contribute to impaired synaptic plasticity and cognitive decline. Actin destabilization can also be linked to the formation of amyloid plaques, a hallmark of the disease. Microglial cells, the resident immune cells of the brain, rely on actin filaments to migrate toward sites of injury or infection.

The actin cytoskeleton is essential for the proper function of heart and vascular cells. Mutations in actin or actin-binding proteins like tropomyosin can lead to defective muscle contraction and cardiomyopathies. For example, mutations in actin genes are linked to familial dilated cardiomyopathy, a condition characterized by the weakening of the heart muscle and reduced pumping efficiency. The contraction of vascular smooth muscle cells is driven by actin-myosin interactions, which regulate blood vessel tone and blood pressure.

### CONCLUSION

The actin cytoskeleton is a vital component of cellular architecture and function. Disruptions in actin dynamics can lead to a wide range of diseases, including cancer, neurodegenerative disorders, cardiovascular diseases, and infections. Understanding how actin filaments contribute to disease pathogenesis creates new avenues for therapeutic strategies aimed at targeting actin-regulating pathways.

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