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Opinion

Impact of Nuak Kinase Signalling in Central Nervous System

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INTRODUCTION

A master regulator for cellular homeostasis throughout the body, AMP activated Protein Kinase (AMPK) detects the AMP: ATP ratio inside of cells and serves as a sensor for available cellular energy. Numerous AMPK downstream targets regulate apoptosis, cell growth, proliferation, migration, adhesion, and other processes. Twelve members of the AMPK related protein kinase family share strong sequence similarities with AMPK but lack AMP: ATP ratio sensitive regulatory subunits, which indirectly controls cellular homeostasis. A conserved serine/threonine kinase domain is shared by two members of the novel AMPK related kinase family, NUAK1 (also known as OMPHK1/ARK5) and NUAK2 (also known as OMPHk2/SNARK), which share about 51% of their sequences. The functions of Nuak1 and NUAK2 are best known for enhancing tumour survival, metastasis, cancer cell proliferation, cell survival, and cell adhesion. AMPK, LKB1, and AKT are the regulators of NUAK1 and UAK2, and they all have known functions in the nervous system.

DESCRIPTION

Recent studies show that NUAK1/2 is expressed strongly in the developing brain. Additionally, NUAK1/2 related cell processes in non-neuronal cells include cell migration, proliferation, autophagy, and morphogenesis are crucial for the development of the brain. Scientists are investigating the actions of NUAK kinases in the development of the brain in light of these overlapping responsibilities. According to evidence, Nuak1/2 are crucial for embryonic development because their double deletion causes abnormalities in neural tube closure and embryonic death because the ventral body wall is unable to form. More evidence points to Nuak1/2's involvement in determining neuronal morphology. For the brain to develop functional circuitry, neuronal shape is essential. Large scale changes in brain circuitry can result from minor alterations in axonal and/or dendritic morphology. Neurite formation, the initial stage of defining neuronal morphology, as well as later sequential stages like terminal axon and dendritic branching, have all been related to Nuak1/2. Actin contractility and mitochondrial transport are the key mechanisms by which Nuak1/2 govern these activities mechanistically, however there is some evidence that Nuak1/2 also affect microtubules.

It is not unexpected that *NUAK* genes are altered in developmental disorders including Attention Deficit/Hyperactivity Disorder (ADHD) and Autism Spectrum Disorder (ASD) because neuronal structure and circuitry are frequently disturbed in neurodevelopmental disorders. In addition, many aging-related illnesses are characterised by neuronal shape deterioration and circuitry loss, and Alzheimer's Disease (AD) and other tauopathies have been related to the NUAK kinases. Growing evidence also shows to interactions between the genes, aetiologies, and pathologies that cause ASD and AD, with tauopathy being implicated in several developmental disorders.

CONCLUSION

Since NUAK1/2 kinase inhibitors are currently FDA approved cancer medicines, the NUAK kinases may represent an undiscovered link between these illnesses and hold unrealized therapeutic potential for nervous system problems. The biological mechanisms by which the Nuak kinases control neuronal growth, from neural tube closure to axonal and dendritic maturation and ageing.

AMP activated Protein Kinase (AMPK) detects the AMP: ATP ratio inside of cells and serves as a sensor for available cellular energy. The functions of Nuak1 and Nuak2 are best known for enhancing tumour survival, metastasis, cancer cell proliferation, cell survival, and cell adhesion. Neurite formation, the initial stage of defining neuronal morphology, as well as later sequential stages like terminal axon and dendritic branching, have all been related to Nuak1/2. Growing evidence also shows to interactions between the genes, aetiologies, and pathologies that cause ASD and AD.

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