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Signaling of Brain Neurons Generation

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Introduction

The GTP-binding "ON" state of RAS GTPases in the cell is a crucial switch for controlling brain activities. The subcellular membrane localization of rat sarcoma (RAS) and RAS homolog protein enriched in brain (RHEB) GTPases involved in this switch are discussed, as well as their role in triggering specific signalling pathways that regulate synaptic connectivity, axonal growth, differentiation, migration, cytoskeletal dynamics, neural protection, and apoptosis. Neuronal H-RAS activation appears to have a protective effect in neurodegenerative disorders, according to cellular and animal studies. Recent optogenetic experiments have revealed new information about the spatiotemporal features of the RAS/Mitogen Activated Protein Kinase (MAPK) or Phosphoinositide-3 kinase (PI3K) pathways.

Description

We explore magnetic guidance of re-growing axons as a supplementary strategy to optogenetic regulation of cellular communication in deep brain areas, which needs light penetration across enormous distances of absorbing tissue. Dopaminergic neuronal cell bodies in the substantia nigra deteriorate in Parkinson's disease. The incapacity of neuronal axons to navigate across a significant distance from the grafted site into striatal target regions must be taken into account in current human trials of stem cell-derived dopaminergic neurons. Grafting dopaminergic precursor neurons into the degenerating substantia nigra is described as a novel method for guiding axonal growth by activating GTPase signalling via protein-functionalized intracellular magnetic nanoparticles that respond to external magnets.

More than 150 human members make up the RAS superfamily of tiny guanosine triphosphatases (GTPases). RAS, RHO, RAB, RAN, and ARF are the five primary subclasses based on functional and sequence similarities [1,2]. RAS signalling is strictly regulated in general, and disruptions in RAS signalling result in the creation of malignant tumours. RAS genes were discovered as retroviral oncogenes of the Harvey [3] and Kirsten [4] rat sarcoma (H-RAS, K-RAS) viruses in the 1960s. The discovery of constitutively active RAS mutations in human malignancies, in particular, ushered forth a new era in RAS research [5].

RAS Homolog Protein Enriched in Brain (RHEB) belongs to the RAS subclass, which is highly conserved in organisms ranging from yeast to humans. Two separate RHEB genes have yet to be discovered in mammals. RHEB1 (from now on RHEB) and RHEB2 (also RHEBL1) gene products share 54 percent identity and 74 percent similarity, suggesting that these proteins perform similar tasks. RHEB is found in a variety of human tissues, but RHEB2 is mostly found in the brain, particularly in the cerebral cortex, occipital pole, frontal, and temporal lobes. RHEB was first found in the rat brain as an immediate-early gene, whose cellular level is rapidly elevated in an N-methyl-d-aspartate (NMDA)-dependent way by high frequency-induced synaptic activity.

Conclusion

The RAS and RHEB GTPases are compared as switch proteins that control major brain activities such neuronal survival and regeneration, synaptic connection, growth, differentiation, migration, and cytoskeletal integrity in this study.

We will focus on the importance of subcellular membrane localization after reviewing structural key elements of proteins involved in this switch function and their regulation of downstream intracellular signalling, culminating in recent aspects of non-invasive optogenetic remote controlling of GTPase signalling after exposing light to brain neurons. Finally, we look at how magnetic guiding cues in neuronal axons incorporating functionalized intracellular paramagnetic nanoparticles can be used to manipulate intracellular RAS signalling pathways. The potential constraints of these unique therapeutic techniques as well as their new therapeutic vistas are critically examined.

References

1. Wennerberg, Krister, Kent L. Rossman, and Channing J. Der. "The Ras superfamily at a glance." *J Cell Sci* 118 (2005): 843-846.
2. Rojas, Ana Maria, Gloria Fuentes, Antonio Rausell, and Alfonso Valencia. "The Ras protein superfamily: Evolutionary tree and role of conserved amino acids." *J Cell Biol* 196 (2012): 189-201.
3. Harvey, J.J. “An unidentified virus which causes the rapid production of tumours in mice.” *Nature* 204 (1964): 1104-1105.
4. Kirsten, W.H, and L.A. Mayer. “Morphologic responses to a murine erythroblastosis virus.” *J Natl Cancer Inst* 39 (1967): 311-335.
5. Malumbres, Marcos, and Mariano Barbacid. "RAS oncogenes: The first 30 years." *Nat Rev Cancer* 3 (2003): 459-465.

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