OPEN ACCESS Freely available online

Journal of Cell Signaling

ISSN: 2576-1471

Short Communication

Mitogen Activated Protein Kinase: A Brief Note

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DESCRIPTION

A serine/threonine-specific protein kinase is a kind of mitogen-activated protein kinase (MAPK or MAP kinase) that is selective for the amino acids serine and threonine. MAPKs control how cells respond to different stimuli such as osmotic stress, pro-inflammatory cytokines, heat shock, and mitogens. Proliferation, gene expression, differentiation, mitosis, cell survival, and apoptosis are all regulated by them. MAP kinases are only present in eukaryotes, however, they are found in a wide range of mammals, fungi, and plants, as well as a variety of unicellular eukaryotes.

Types of MAPKs

The activation of most MAPKs is dependent on two phosphorylation events, they have three-tiered pathway, and they have comparable substrate recognition sites. The "classical" MAP kinases are those that are found in the body. However, there are certain ancient outliers from the above-mentioned category that lack dual phosphorylation sites, only form two-tiered pathways, and lack the properties essential for substrate binding by other MAPKs. "Atypical" MAPKs are the name given to these types of MAPKs. It's still uncertain if atypical MAPKs and conventional MAPKs belong to the same family [1].

The MAPK family of kinases in mammals is divided into three subfamilies:

- Extracellular signal-regulated kinases (ERKs)
- c-Jun N-terminal Kinases (JNKs)
- p38 mitogen-activated protein kinases (p38s)

Process of MAPK activation

In their original state, mitogen-activated protein kinases are catalytically inactive. They require, possibly numerous phosphorylation events in their activation loops to become active. Specialized enzymes from the STE protein kinase group are responsible for this. Long-range allostery can be used to cause a conformational change in the structure of a protein in this way. The activation loop of conventional MAP kinases has a TxY (threonine-x-tyrosine) motif which means TEY in mammalian ERK1 and ERK2, TDY in ERK5, TPY in JNKs, TGY in p38 kinases that must be phosphorylated on both the threonine and tyrosine residues to lock the kinase domain in a cascade Phosphorylation of tyrosine generally precedes phosphorylation of threonine in vivo and in vitro, while either residue can be phosphorylated in the absence of the other. Members of the Ste7 protein kinase family, also known as MAP2 kinases, phosphorylate the tandem activation loop (which has been postulated to be either distributive or processive depending on the cellular context). Phosphorylation of MAP2 kinases by a variety of upstream serine-threonine kinases also activates MAP3 kinases. Classic MAPK pathways are multi-tiered, yet rather linear because MAP2 kinases show very limited action on substrates other than its homologous MAPK. These pathways can transport stimuli from the cell membrane, where numerous MAP3Ks are activated to the nucleus or where only MAPKs can enter or to a variety of other subcellular locations [2].

A number of phosphatases are involved in the inactivation of MAPKs. The MAP kinase phosphatases (MKPs), a subclass of dual-specificity phosphatases (DUSPs), are a relatively conserved family of specialized phosphatases. These enzymes can hydrolyze the phosphate from both phosphotyrosine and a phosphothreonine residue, as their name indicates. Some tyrosine phosphatases are also implicated in inactivating MAP kinases since removing either phosphate group dramatically reduces MAPK activity, thus eliminating signaling for example the phosphatases HePTP, STEP, and PTPRR in mammals [3,4].

CONCLUSION

With the new advances in cryo-EM strategies, the cation channel field has acquired another comprehension of ion channel structure–function relationships with never-seen atomic subtleties during the most recent two years. Despite the fact that it has frequently been brought up that the conditions under which cryo-EM is performed are very un-physiological, we accept that the field is drawing nearer to understanding the full gating pattern of both voltage-and ligand-gated channels at the atomistic level as they progress from the resting to the open and the inactivated state, ion channel function is innately unique,

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Received: December 06, 2021; **Accepted**: December 20, 2021; **Published**: December 27, 2021

Citation: Cobb MH (2021) Mitogen Activated Protein Kinase: A Brief Note. J Cell Signal. 6: 262.

and a total comprehension of the systems of gating 44 and permeation and selectivity 45 just as the visualization of ion stream at the atomistic level 44 might be conceivable with the guide of sub-atomic elements simulations. Regardless of whether these constructions will genuinely empower structure-based medication plan and speed up ion station drug disclosure is an alternate question as we would like to think. Medication advancement has numerous perspectives: as a matter of first importance, target validation, which is the reason it is vital to acquire what is regularly named "profound" comprehension of the science of ion channels. As the most recent two years have again illustrated, we are as yet learning new science, as uncovered by the unforeseen job of the Piezo channels in pulse regulation and breathing or the significance of mitochondrial K Na-1.2 channels in energy consumption and fat digestion.

REFERENCES

1. Alexander SP, Striessnig J, Kelly E. The concise guide to pharmacology 2017/18: voltage-gated ion channels. Br J Pharmacol. 2017; 174(Suppl 1):S160–S194.
2. Sun J, Mackinnon R. Cryo-EM Structure of a KCNQ1/CaM Complex reveals insights into congenital long qt syndrome. J Cell 2017; 169(6): 1042–1050.e9.
3. Singh AK, McGoldrick LL, Sobolevsky AI: Structure and gating mechanism of the transient receptor potential channel TRPV3. J Nat Struct Mol Biol. 2018; 25(9):805–13.
4. She J, Guo J, Chen Q. Structural insights into the voltage and phospholipid activation of the mammalian TPC1 channel. J Nature. 2018;556(7699):130–4.