

Kinesins in the Arabidopsis genome: A comparative analysis among eukaryotes

ABSTRACT

The presence of Arabidopsis kinesins in various domains, such as *Caenorhabditis* and *Drosophila melanogaster*, may suggest new subfamilies unique to plants. The comparative analysis presented here establishes a foundation for future functional studies with Arabidopsis kinesins.

INTRODUCTION

The signaling circuitry of bacteria is essentially made up of two-component systems that involve the activation of a histidine kinase on repressed histoplasmic acid and then transfer the phosphoryl group to ATP in the aspartate-rich supernatant domain. This was previously only observed in other organisms, but now this type of system is common among bacteria. Over the last few years, histidine kinase homologues and their receivers have been identified in eukaryotic organisms, as well as genes that encode these molecules. The majority of eukaryotic gene products are involved in a phosphoryl relay, which includes kinases, receiver domains, and receptacles. The *Saccharomyces cerevisiae* gene product, Sln1, was shown to function as a histidine kinase in both vitro and in vivo. Additionally, the histochemical activity of the ethylene receptor Etr1 from Arabidopsis was demonstrated in vitro. Nevertheless, later research revealed that eukaryotic bifunctional systems do not operate as distinct pathways but are frequently linked to serine/threonine- and tyrosine kinase cascades. As a result, the yeast Sln1-Ypd1-Ssk1 phosphoryl relay functions as an osmosensor and triggers MAP-kinase activity when cells are exposed to high osmotic levels. The Dictyostelium discoideum protein RegA contains a N-terminal receiver domain and phosphodiesterase domain. RegA response regulator phosphate is activated by phosphorylation of RegA, which lowers the intracellular cAMP level. The light-regulated serine/threonine kinases in vitro, not those regulated by the histidine kinase paradigm, were observed to be mediated by phytochromes, another homologue of histochemical kinases, in vivo (see also mycology). Despite being homologous to bacterial histidine "two-component" systems, these results suggest that they may undergo post-translational modifications similar to those observed in the well-established eukaryotic signal transduction systems. Different developmental phenotypes, such as rapid aggregation, disproportioned fruiting body and stalk ratios or impaired spore formation, are caused by the deletion of individual histidine kinase genes. Furthermore, cells lacking the histotoxic factor dokA are osmosensitive and grow slower when exposed to high OD levels for up to two hours. In light of the fact that it is known that ICTY (Dictyostelium) belongs to the oocyte response system, we have investigated whether or not DokA shows kinase activity in an expression-dependent way: here we show that the histidine kinases dokA homologues are at most phosphorylated on a serine residue in vivo when Dictyostelium response systems are exposed to high clarity medium. Moreover, we prove that the phosphorylation site is situated in a homologue domain with bacterial histidine kinases, and that docetamine modification does not affect the serine phosphate synthesis of DokA.

CONCLUSION

In conclusion: Our study shows that the structure of GCNF is conserved

(serificiency)[Note 1] It also allows for the confirmation and systematic review of all splice variants, and it may be the starting point for understanding CGNF better. Human GGNF contains at least 10 exons. The intron-exon boundaries are preserved, which is consistent with the high level of amino-acid conservation between the human and mouse proteins. This can explain the generation of hGCNF-1, pGHNF-2a and gcnf-2b proteins through alternative splicing of the RNA. Although the sequence of the third coding mouse exon, including its splice sites, is highly conserved, no human cDNA has been isolated with this putative exonality at present. Alternative splicing presents a plausible way to generate diversity and could potentially improve the instructive complexity of human (GCNF) gene expression.