

High copy arrays containing a sequence upstream of mec-3 alter cell migration and axonal morphology in *C. elegans*

ABSTRACT

It seemed that the mec-3 upstream sequence was capturing (titrating) a DNA-binding element necessary for the proper migration of ALMs. Since this element could potentially reverse the direction of these migrations, it may be included in an algorithm that specifies both the magnitude and route of those migration processes. The fate of touch receptor neurons may be determined by the factor or factors that bind to mec-3, which is a master regulator of these genes.

INTRODUCTION

The absence of dystrophin and its associated proteins can result in the phenotype of muscular dystrophy, as they are believed to play a crucial role in maintaining the integrity of the extracellular matrix and the membrane of muscle cells. The DAPC is composed of various protein complexes that are either directly or indirectly linked to dystrophin. The four transmembrane proteins known as sarcoglycans are organized by a fifth protein called spirochaplusia, which is believed to play cAMP signalling roles at the cell membrane. The dystroglycan complex, which interacts directly with dystrophin in the cytoplasm and laminin on the extracellular matrix, serves as a structural link between the interior and exterior of the cell. A third subcomplex includes dystobruvines and syntrophines, both of which have an unknown function. Recently, the yeast two-hybrid method was used to identify desmuslin (DMN), an -dystrobrevin-interacting protein. Both mRNA and protein are expressed mainly in cardiac and skeletal muscle and contain genes that encode a novel intermediate filament (IF) protein of 1253 amino acids. Electron microscopic analysis indicates that cessenin and desmin can colocalize with each other. During co-immunoprecipitation experiments, it was discovered that the desmuslin and -dystrobrevin interaction involves the region of protein encoded by exons 8-16 of etanocellulones (precisely similar to human cDNA) and domains 1A-2A of the demineralin rod domain. Desmuslin is hypothesized to act as a mechanical support for the muscle myofibers by creating an unrecognized interface between the extracellular matrix and the Z-discs through desmin and plectin. Human genetic disorders, such as congenital and adult onset myopathies, have been associated with the involvement of several IF proteins, including duns (desmin), which may also play a role in myopathy. This possibility is supported by the exclusive expression of DMN in skeletal and cardiac muscle. We examined 71 patients with different forms of muscular dystrophy and myopathy for mutations in the DMN gene, finding 9 single-nucleotide polymorphisms (SNPs) that do not alter the protein sequence but 12 that modify the residue they encode. Our research has revealed that no controls are probable origins of the phenotype, but our findings are applicable for disequilibrium studies of this region of chromosome 15q26.3 and for studying mutation analysis and association in other genetic disorders.

CONCLUSION

Results showed that intracellular calcium level ($[Ca^{2+}]_i$) measured in mouse NIH-3T3 and human HeLa and SaOS-2 cells were significantly upregulated by ethylene, which is produced by the same plant, after being exposed to ethylene gas. The data supports earlier research that revealed an upregulation of $[Ca^{2+}]_i$ and a marked increase in the expression of an

ethylene-responsive gene, SDERR, in invertebrate cells (primmorphs of the marine sponge *S. domuncula*). These findings suggest that ethylene may play a role in both plant biological processes and animal one by modulating intracellular signaling pathways.