Genetic snapshots of the Rhizobium species NGR234 genome

ABSTRACT

By'skimming' the genome of Rhizobium sp. NGR234, researchers gain new insights into the fine structure and evolution of its replicons, as well as the role of abiotic functions in the complex that forms part of the genetic makeup of an organism. Despite the fact that most likely coding sequences were partitioned into functional classes, similar to Bacillus subtilis, NGR234 had more functions related to transposable elements.

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 spirochaplasia, 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 cessnin and desmin can colocalize with each other. During co-immunopreciptation 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

According to earlier investigations on FMR proteins, the FXR1 protein is primarily cytoplasmic, but undifferentiated cells in various tissues of human foetuses and mouse embryonic stem cells occasionally exhibit nuclear localization. By examining the model system of C2C4 myoblasts that can be manipulated in vitro to differentiate into mytubes, we provide strong evidence that FXR1P isoforms are actually stored in the nucleus as well as in other forms. We propose that this nucligen-cytoplasmic partitioning of

FYR1,P may be controlled by factors regulating cell differentiation. Moreover, we also postulate that FMRP isoforms, which have been kept secreted until now because of the scarcity of available antibodies, may play a nuclear role in mRNA maturation during specific phases of neuronal differentiation and plasticity. To sum up, the model system presented here is a potent resource for ongoing research on the structure-function relationships among various FMR family members, as the role of FXR1P and FYR2P in Fragile X Mental Retardation is yet to be determined.