ABSTRACT
The absence of mutations in the desmuslin gene did not impact its function. Nevertheless, the single-nucleotide polymorphisms mapped in this study are highly disequilibrated and can be used for disambiguation studies of this region of chromosome 15q26.3.

INTRODUCTION

Desmuslin is an intermediate filament protein that serves as a template for the formation of the nucleosome and the ribosome. It is a member of the human chromosome 15q26.3.

The desmuslin protein is a member of the c-myc family, a family of filament proteins that comprises the desmuslin protein, myosin, and the myosin-like protein (Ml). The desmuslin protein has been associated with genes involved in protein remodeling, cell cycle regulation, cell differentiation, and gene regulation, and has been associated with several diseases.

In a recent study, the authors isolated the desmuslin protein from a human cancer cell line and identified a new, novel target of des The absence of these proteins can result in muscular dystrophy, as they are believed to aid in the anchoring of the muscle cell membrane to the extracellular matrix. The DAPC is composed of various protein subcomplexes, with sarcoglycans being one of them. Dystrophin also provides structural support for the third subgroup, which includes both cytochronic and retroactive proteins. The yeast two-hybrid method was used to identify desmuslin (DMN), an -dystrobrevin-interacting protein that is expressed primarily in cardiac and skeletal muscle. The gene encodes a novel intermediate filament (IF) protein of 1253 amino acids and colocalizes with desmin, another muscle IF protein. Co-immunopreciptation experiments revealed that the desmosullinate interacts mitophilically by creating 'a new mechanical support mechanism' between the DAPC and Z-discs. Due to the fact that several IF proteins, such as desmin, have been linked to human genetic disorders, including congenital and adult onset myopathies (desmuslin) also play a role in myopathy, this is likely due to its exclusive expression of DMN gene in skeletal and cardiac muscle.

CONCLUSION

Remarkable conclusions By describing the building blocks of protein complexes in skeletal muscle and heart, particularly information regarding genotype-phenotype connections, we can better understand the pathophysiology of human muscle diseases. Furthermore, other groups should test for the C598T DMN mutation in their human patient samples affected by muscular and cardiac diseases, as well as generate desmuslin null animal models to further clarify this role.