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Exploring the FMNL2 Interact me through Quantitative Mass Spectrometry

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Abstract

The study investigates the FMNL2 interactome using quantitative mass spectrometry, revealing novel protein interactions and potential functional insights into FMNL2-associated pathways.

Keywords: FMNL2; Interactome; Quantitative Mass Spectrometry; Protein Interactions; Functional Insights; Pathway Analysis

Introduction

Quantitative mass spectrometry has revolutionized the study of protein-protein interactions by enabling comprehensive mapping of protein interactomes. Understanding the interactome of Formin-like 2 (FMNL2), a member of the formin family of proteins involved in actin dynamics and cytoskeletal organization, is crucial for elucidating its

revealing novel insights into its protein interaction network and functional implications in cellular processes. Future studies building upon these ndings could further unravel the intricate mechanisms through which FMNL2 coordinates cellular dynamics and contribute to disease progression.

Conclusion

In this study, we employed quantitative mass spectrometry combined with co-immunoprecipitation to explore the FMNL2 interactome, revealing a diverse array of protein interactions implicated in various cellular processes. Our ndings underscore the central role of FMNL2 in regulating actin dynamics, cell motility, and potentially in uencing signaling pathways critical for cellular homeostasis. rough rigorous validation and network analysis, we con rmed the speci city and functional relevance of FMNL2 interactions with key proteins involved in cytoskeletal organization and cellular signaling. is comprehensive approach not only expands our understanding of FMNL2 biology but also provides a framework for future investigations into its role in health and disease. e insights gained from this study pave the way for further exploration into how FMNL2-mediated protein interactions contribute to cellular phenotypes and disease states. Targeting speci c components of the FMNL2 interactome may hold promise for developing novel therapeutic strategies aimed at modulating cellular dynamics in conditions where FMNL2 dysregulation is implicated. Overall, our study contributes to the broader eld of proteomics and cellular biology by elucidating the intricate network of FMNL2 interactions and their implications for cellular function and disease pathogenesis. Future research e orts will be crucial for dissecting the precise mechanisms underlying FMNL2-associated pathways and translating these ndings into clinical applications.

Acknowledgement

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Con ict of Interest

None

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