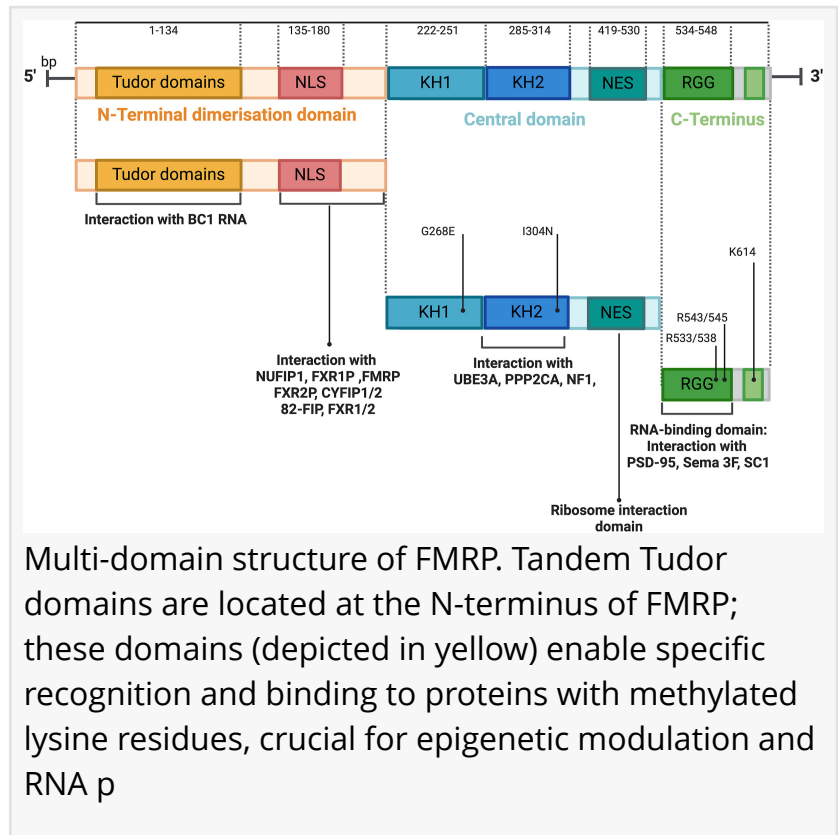


New Insights into Cancer Biology: The Dual Role of FMRP in Tumor Progression and Therapy Resistance

SHANNON, CLARE, IRELAND, April 20, 2025 /EINPresswire.com/ -- A recent review published in *Genes & Diseases* sheds light on the complex and multifaceted role of RNA-binding proteins (RBPs) in cancer progression, with a particular focus on the fragile X mental retardation protein (FMRP). Traditionally recognized for its critical functions in neural development, FMRP is now emerging as a key regulator in cancer biology, influencing tumor growth, metastasis, and therapy resistance. This growing body of knowledge presents a shift in understanding how RNA metabolism can drive oncogenic processes and potentially offer novel diagnostic and therapeutic strategies.



FMRP plays a dual role in cancer, acting both as a tumor suppressor and promoter, depending on the cellular context. Its ability to regulate mRNA stability, translation, and transport positions it as a crucial factor in the intricate network of gene expression that governs cancer progression. Studies indicate that low levels of FMRP are associated with decreased tumor development in some cases, whereas high expression is linked to aggressive cancers, including breast, colorectal, and hepatocellular carcinomas. The variability in FMRP's impact on different tumor types underscores the necessity for a nuanced approach when considering it as a therapeutic target.

One of the most significant findings discussed in the review is FMRP's role in therapy resistance. By interacting with key oncogenic pathways, FMRP contributes to cancer cells' ability to withstand chemotherapy, radiation, and immunotherapy. In colorectal cancer, FMRP stabilizes epidermal growth factor receptor (EGFR) mRNA, enhancing tumor proliferation. Additionally, its involvement in epithelial-mesenchymal transition (EMT) suggests a critical function in tumor

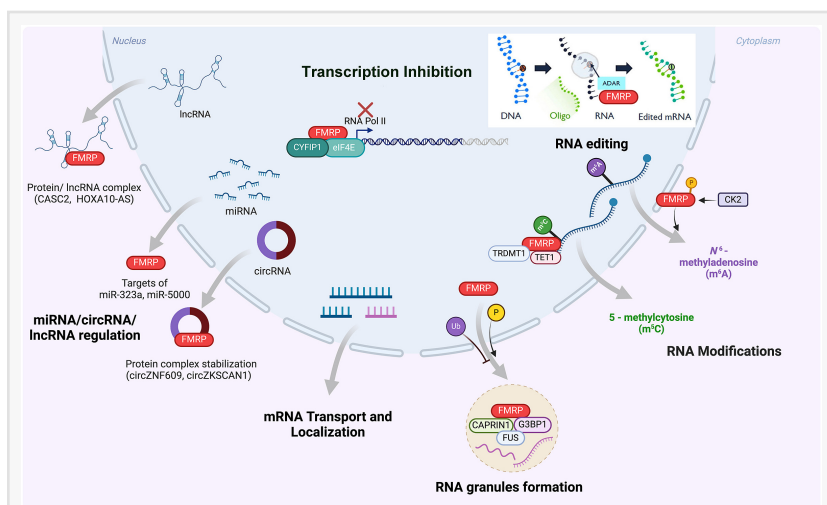
metastasis. Furthermore, its association with immune evasion mechanisms highlights its potential influence on the tumor microenvironment and response to immune checkpoint inhibitors.

The potential of targeting FMRP in cancer treatment is an area of growing interest. By interfering with its ability to regulate oncogenic mRNAs, researchers aim to develop precision medicine strategies that can either inhibit its tumor-promoting functions or harness its tumor-suppressive capabilities. Exploring the post-translational modifications (PTMs) of FMRP and its interplay with other RNA-binding proteins could pave the way for highly specific therapeutic interventions that mitigate its oncogenic effects while preserving essential cellular functions.

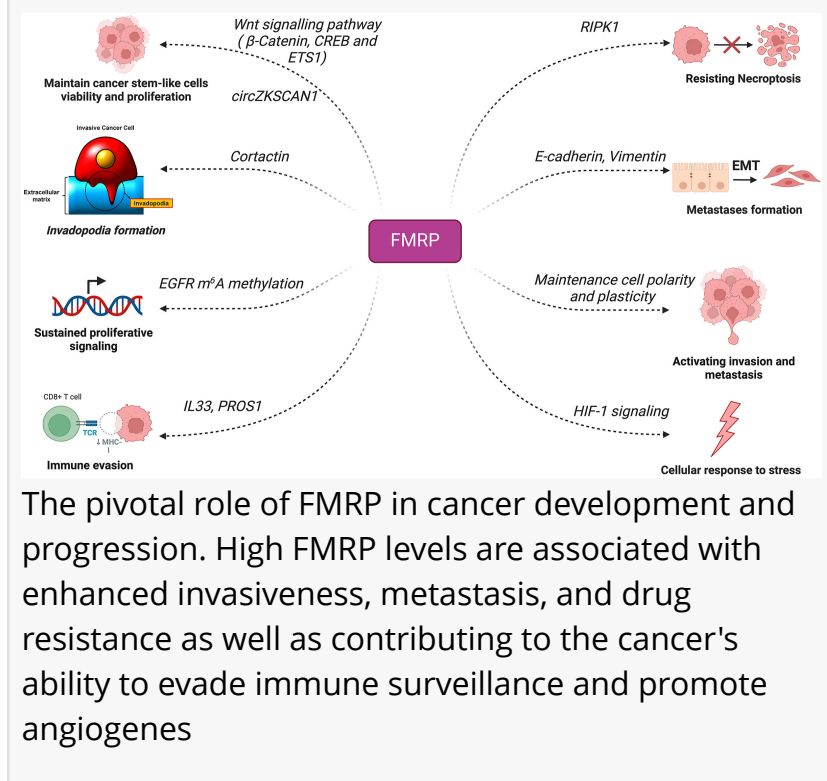
This evolving understanding of FMRP's role in cancer underscores the complexity of RNA metabolism in tumorigenesis. As scientists continue to investigate the intricate balance between FMRP's tumor-suppressive and oncogenic functions, its potential as both a biomarker and therapeutic target is becoming increasingly evident.

#####

Genes & Diseases publishes rigorously peer-reviewed and high quality original articles and authoritative reviews that focus on the molecular bases of human diseases. Emphasis is placed on hypothesis-driven, mechanistic studies relevant to pathogenesis and/or experimental therapeutics of human diseases. The journal has worldwide authorship, and a broad scope in basic and translational biomedical research of molecular biology, molecular genetics, and cell biology, including but not limited to cell proliferation and apoptosis, signal transduction, stem cell biology, developmental biology, gene regulation and epigenetics, cancer biology, immunity



Biological functions of FMRP in RNA biology. The illustrations depict the functional mechanisms of FMRP (depicted in the center) in RNA biology, such as miRNA/cirRNA/lncRNA-mediated regulation, mRNA transport and localization, RNA modification (m5C/5-meth



The pivotal role of FMRP in cancer development and progression. High FMRP levels are associated with enhanced invasiveness, metastasis, and drug resistance as well as contributing to the cancer's ability to evade immune surveillance and promote angiogenesis

and infection, neuroscience, disease-specific animal models, gene and cell-based therapies, and regenerative medicine.

Scopus CiteScore: 7.3

Impact Factor: 6.9

#####

More information: <https://www.keaipublishing.com/en/journals/genes-and-diseases/>

Editorial Board: <https://www.keaipublishing.com/en/journals/genes-and-diseases/editorial-board/>

All issues and articles in press are available online in ScienceDirect

(<https://www.sciencedirect.com/journal/genes-and-diseases>).

Submissions to Genes & Disease may be made using Editorial Manager

(<https://www.editorialmanager.com/gendis/default.aspx>).

Print ISSN: 2352-4820

eISSN: 2352-3042

CN: 50-1221/R

Contact Us: editor@genesndiseases.com

X (formerly Twitter): @GenesNDiseases (<https://x.com/GenesNDiseases>)

#####

Reference

Yunlu Jia, Ruyin Jia, Yongxia Chen, Xuanyi Lin, Nadire Aishan, Han li, Linbo Wang, Xiaochen Zhang, Jian Ruan, The role of RNA binding proteins in cancer biology: A focus on FMRP, Genes & Diseases,

2025, 101493, <https://doi.org/10.1016/j.gendis.2024.101493>

Funding Information:

National Natural Science Foundation of China 82000212

Natural Science Foundation of Zhejiang Province, China LQ21H160022

Medical Health Science and Technology Project of Zhejiang Provincial Health Commission (China) 2024KY989

Genes & Diseases Editorial Office

Genes & Diseases

+86 23 6571 4691

[email us here](#)

This press release can be viewed online at: <https://www.einpresswire.com/article/804853874>

EIN Presswire's priority is source transparency. We do not allow opaque clients, and our editors try to be careful about weeding out false and misleading content. As a user, if you see something we have missed, please do bring it to our attention. Your help is welcome. EIN Presswire, Everyone's Internet News Presswire™, tries to define some of the boundaries that are reasonable

in today's world. Please see our Editorial Guidelines for more information.

© 1995-2025 Newsmatics Inc. All Right Reserved.