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The extra-ribosomal role of ribosomal proteins

Protein synthesis is a highly regulated and coordinated process involving the action of ribosomes and a set of translation factors. Ribosome biogenesis occurs in the nucleolus and requires the action of 80 ribosomal proteins (RPs), 4 ribosomal RNAs (rRNAs), other associated proteins and small nucleolar RNAs. The structure of the ribosomal subunits has identified the role of RPs as RNA chaperones for ribosomal assembly and as endo- and exo-nucleases essential for the maturation of rRNAs. Some play a role in the joining of 40S and 60S subunits during translation initiation. Others interact with tRNA or stabilize the ribosome by encasing the exit groove. Studies have also shown that RPs may have extra-ribosomal functions, ranging from DNA repair to replication, proliferation, apoptosis and chemoresistance. Mutations in RPs in animal models and humans induced a wide variety of phenotypes suggesting a role of RPs beyond the ribosome structure. Thus far, 11 RP mutant mice have been reported exhibiting diverse phenotypes including decrease body size, defective organs and embryonic lethality. Defects in ribosome biogenesis have been linked to many diseases collectively named ribosomopathies. These include myelodysplastic syndromes, due to *RPS14* haploinsufficiency and Diamond-Blackfan anemia, caused by mutations in *RPS19* gene. These abnormalities have shown an increase susceptibility to hematological malignancies. Indeed, RPs has been linked to tumorigenesis in several reports, suggesting a role in promoting transformation. Several RPs are overexpressed upon activation of the oncogene *Myc*. Some are found overexpressed in various human tumors, including prostate and colon cancer, metastatic nasopharyngeal carcinomas, metastatic melanomas and metastatic human breast cancer cells. Some RPs has also been proposed as biomarkers for various cancers, such as colorectal, gastric, prostate cancers and lymph node metastasis. These evidences suggest that RPs could be used as potential targets in cancer therapeutics.

Biography

Zeina Nasr is Assistant Professor, at the Department of Biology in the University of Balamand, Lebanon. She did her PhD from McGill University in the Department of Biochemistry. She has her interest in understanding the molecular aspect of tumor initiation and progression. Her research focuses on studying the effect of translation initiation dysregulation on cancer behavior. She has worked with several cell lines and transgenic mouse models and deciphered important pathways that contribute to cancer initiation and progression to metastasis. She has experience in conducting research and teaching at various institutions. Currently, her work focuses on the extra-ribosomal functions of ribosomal proteins and their effects on tumorigenesis.

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