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Could Ribosomal Heterogeneity Contribute to Bone Marrow Failure?

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It has long been thought that ribosomes; the complex ribonucleoprotein particles responsible for mRNA translation, are identical in composition and function in every cell. Recent work has however challenged this notion as evidence for heterogeneity of ribosomes in different tissues accumulates. Diamond Blackfan anemia (DBA) is perhaps the best studied ribosomopathy wherein mutations in about 11 different ribosomal proteins (RPs) lead to bone marrow failure. Additionally, DBA patients often suffer from tissue-specific defects such as cleft palate, craniofacial and limb abnormalities, heart defects, growth retardation and a predisposition to cancer. The observation that different RP mutations are associated with different defects, for instance, *RPL5* mutations are associated with cleft palate while *RPL11* mutations with a lack of craniofacial defects, suggests that these ribosomal proteins may have unique functions in different tissues. Additionally, evidence that RP mutations can lead to specific defects in the translation of selected mRNAs is also mounting. For example, ablation of Rpl38 in mice led to the specific reduction of mRNA translation of a set of Homeobox genes during embryonic development. Additionally, haploinsufficiency of *RPS19* in mouse erythroblasts led to the reduction in translation of a subset of erythroid specific mRNAs. Based on these observations, it is tempting to speculate that individual RPs may play a more significant and specific role in the translational control of subsets of mRNAs in specific tissues than previously thought. Haploinsufficiency of specific RPs may thus contribute to disease by altering the quality and/or quantity of ribosomes in a tissue specific manner leading to aberrant translation of a subset of mRNAs. The idea that tissue-specific ribosomes could contribute to the pathobiology of DBA is an intriguing one which warrants further scrutiny. This would pave the way to gaining a better understanding of the molecular mechanisms underlying bone marrow failure associated with DBA, and perhaps lead to the development of new therapies for this and other bone marrow failure syndromes.

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