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# Insights of Psychotropic Drug Effects on Ribosomal Genes and Protein Synthesis

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## Introduction

The pathophysiology of schizophrenia (SCZ) is still being studied. Increased copy number of ribosomal RNAs has been reported in SCZ patients, which is consistent with increased expression of ribosomal genes, a well-documented feature of SCZ. Animal model studies also point to ribosomal involvement in SCZ. Overexpression of SH3 and multiple ankyrin repeat domains 3 (SHANK3), a gene associated with schizophrenia pathogenesis, for example, resulted in mania-like behavior and an enrichment of ribosome-related genes, according to The Kyoto Encyclopedia of Genes and Genomes (KEGG) gene library. The ribosome and its associated genes may be involved in the pathological processes of neuropsychiatric disorders [1-3] such as schizophrenia.

Ribosomes translate mRNAs into proteins, and their function is an indicator of cell morphology and structure integrity. Ribosomes in eukaryotic cells are made up of a small (40S) and a large (60S) subunit that assembles over the mRNAs. The ribosome's small subunit anchors the mRNA, allowing a sequence of three nucleotides (a codon) to be presented to a specific tRNA carrying an amino acid at the aminoacyl site (A site). The ribosome's large subunit connects each amino acid to form a polypeptide chain at the peptidyl site (P site), while the empty tRNA is ejected from the ribosome at the exit site (E site).

Mitochondrial dysfunction has been linked to the pathophysiology of a number of psychiatric disorders, including SCZ, bipolar disorder (BD), and major depressive disorder [4,5] and is associated with involvement in energy metabolism and redox mechanisms. However, little is known about mitochondrial ribosomes and their potential role in neuropsychiatric disorders. Similar to cytosolic ribosomes, mitochondrial ribosomes are made up of two subunits (28S and 39S), but they are found in the inner mitochondrial membrane rather than the cytosol. They are in charge of translating mitochondrial mRNAs, which encode mitochondrial membrane proteins and energy-producing enzymes.

## Description

Multiple studies show that abnormal protein translation is linked to the pathophysiology of SCZ (reviewed by), possibly as a result of decreased synaptic plasticity and thus neurotransmission. BD is linked to a stress response in the endoplasmic reticulum, which contains some ribosomes. As a first-line treatment for BD, lithium has been shown to reverse protein synthesis dysfunction by inhibiting the phosphorylation of eukaryotic elongation factor-2 (eEF2), an essential regulator of mRNA translation. Furthermore, in rodent neurons, ribosome biogenesis and protein composition may be influenced by location and cellular environment, resulting in the production of 'specialized' ribosomes with exceptional protein translation capacity.

This demonstrates the importance of ribosomes in the remote remodelling and repair of neurons along dendrites and axons. In the context of neuropsychiatric disorders, this machinery may be impaired. Reduced protein translation is implicated in SCZ and other psychiatric disorders such as BD and major depressive disorder, according to the findings. Several studies have also found altered expression of genes involved in the regulation of protein translation at the genetic level. In human brain biopsies, for example, more pronounced transcriptome alterations in pathways involved in protein synthesis and translation initiation in dorsolateral prefrontal cortex pyramidal cells were associated with the diagnosis of SCZ.

## Conclusion

Together, the ribosome and protein translation could be used to develop new treatments for SCZ and other neuropsychiatric disorders. To investigate this possibility, we chose some commonly used psychotropic drugs with different molecular mechanisms of action and investigated their transcriptional effects on the expression of genes involved in protein translation, including ribosomal genes, in an in vitro model of human neurons. The effects on protein synthesis were also investigated. Given the link between ribosomal dysregulation and SCZ, we hypothesized that psychotropic drugs would alter ribosomal gene expression and protein synthesis rates.

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