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apoptotic, anti-proliferative and anti-angiogenic effects, but the precise molecular mechanisms by which it exerts these effects remain unclear. The clinical efficacy of the Sorafenib treatment is very low because the occurrence of resistances is a very common fact. Herein, we show that Sorafenib inhibits global translation initiation in HCC cell lines by polysome profiling analysis. Moreover, we observe a reduction in the S6 ribosomal protein phosphorylation level and demonstrate that this inhibition is driven mainly through the phosphorylation of the Initiation Factor 2 (eIF2), although we do not discard the role of other pathways. Finally, we show that while the global translation is blocked, the expression of selective mRNAs is enhanced upon a Sorafenib treatment. Together, our findings indicate that protein synthesis is a target of Sorafenib. Given the essential role of translation in tumoral transformation, the study of this process in this context could lead to the identification of novel therapeutic targets of HCC.

P-11-013
The presence of a nested alternative open reading frame in the matryoshka gene: translation its mRNA corresponds to the mechanism underlying the ribosome scanning model

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Recently, we discovered the gene that encodes the Kunitz peptidase inhibitor-like protein (KPILP) in plants of the Solanaceae family (which includes the potato, tomato, and tobacco), and that there is little mRNA in the leaves of intact plants (unlike what is observed in the roots). However, mRNA content was sharply increased after abiotic and biotic stress. Unlike the genes that encoding the Kunitz peptidase inhibitor (KPI) in various animals, *KPILP* has no introns, which thereby excludes the possibility that its expression is regulated via alternative splicing. Here, we investigated the mechanism underlying the regulation of *KPILP* in stress conditions and verified our hypothetical model, which proposes that in an intact leaf, the ribosome "ignores" the start codon of the KPILP mRNA because it is within an unfavourable nucleotide context, and instead it prefers to translate the aORF via the mechanism of internal translation initiation. Under stress conditions, aORF translation is suppressed, and translation using the start codon of the maternal mRNA is resumed, which results in an increase in the level of mRNA content in the leaf. We tested this model on a series of vector constructs that we used to alter the nucleotide context of the start codon of the maternal mRNA, the nucleotide sequence preceding the aORF, the aORF start codon and the mRNA sequence. Analysis of the level of accumulation of mRNA encoding the mutant *KPILP* gene confirmed our proposed model, which states that the synthesis of the aORF product determines the degree of mRNA accumulation in the leaf. If the scanning ribosome "prefers" the start codon of the maternal mRNA (due to a favourable context or stress), then the aORF product will not be synthesized, and, therefore, there will be a high level of maternal mRNA in the leaf. This study was performed with the financial support of the Russian Foundation for Basic Research (project No. 17-29-08012).

P-11-015
Whole genome sequencing of sup45 and sup35 nonsense mutants of yeast *Saccharomyces cerevisiae*

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In the yeast *S. cerevisiae* release factors eRF1 and eRF3 are encoded by essential genes *SUP45* and *SUP35* respectively. Previously we showed that nonsense mutations in these genes (*sup45-n* and *sup35-n*) support viability in different genetic backgrounds. It was shown that the viability of strains with these mutations is increased after growth in the absence of wild-type allele. Possibly, additional mutations are selected during the first stage of selection. Thus we aimed to investigate the precise mechanism leading to readthrough of *sup45-n* and *sup35-n* mutations. To achieve this goal we have constructed a complete reference genome assembly of the strain U-1A-D1628 bearing a deletion of the *SUP45* gene and a plasmid pRS316-SUP45 which is an ancestor to the strains harboring *sup45-n* mutations obtained using the plasmid shuffling technique. To create reference genome assembly of this strain we conducted whole-genome sequencing of this strain using the Oxford Nanopore and Illumina technologies. Simultaneously we have constructed the strain U-14-D1690 having a deletion of the *SUP35* gene and a plasmid pRS316-SUP35 which is a close relative of the strain U-1A-D1628. Also now we obtained whole-genome sequencing data of the several dozen *sup45-n* and *sup35-n* mutants. Preliminary analysis of these data showed that no large-scale genomic changes occur upon substitution of the wild-type copy of the genes to its mutant version. The obtained reference genome assembly of the U-1A-D1628 and U-14-D1690 strains, as well as the whole genome sequences of corresponding nonsense mutant strains would allow us to identify the genetic changes that occur during such substitution of wild-type *SUP45*/*SUP35* alleles to the mutant ones. The authors acknowledge the RC "Biobank" of St Petersburg State University. The work was supported by the program 0112-2016-0015 (characterization of mutants) and the grant of the RSF (18-14-00050) (other parts of the work).

P-11-016
Aim23p interacts with the small subunit of yeast mitochondrial ribosome

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Aim23p is a yeast protein found in mitochondria. For many years, its function in the organelles remained unknown. In 2012, using a combination of bioinformatics and genetics, we have shown that Aim23p is initiation factor 3 of mitochondrial translation. However, its action in the protein biosynthesis system is somewhat non-canonical: translations does not stop in absence of Aim23p, it is just misbalanced from the point of view of

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