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P-26-015
A transcriptome-wide approach to identifying RNA targets of the Prader-Willi locus snoRNAs

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Prader-Willi syndrome (PWS) is the most common known genetic cause of life-threatening obesity in children. It is a complex genetic disorder with implications for the metabolic, endocrine, neurologic and behavioral impairments. The PWS results from lack of expression of paternally expressed genes from 15q11.2-q13 genomic region. In a close up, a deletion of paternally expressed SNORD116 gene cluster (a group of C/D-box snoRNAs located in PWS region) was shown to be the primary genetic determinant of the PWS phenotype. Since molecular mechanism instigating PWS still remain unknown, there is an urgent need to study their identification. SNORD116 (HBII-85) family consists of 29 homologues snoRNAs (small nucleolar RNAs), which display no apparent sequence complementary to canonical RNA targets and are considered as orphan snoRNAs with yet unknown function. Still, ectopic overexpression of SNORD116 in HEK293T cell line, endogenously not expressing this gene, resulted in the changed expression of more than 200 protein coding genes. This implies on SNORD116 cluster to play a regulatory role in mRNA stability. Whether the SNORD116 RNAs play a direct role in that or indirect by regulating transacting factors remains to be resolved. Thus we study the function of SNORD116 snoRNAs by primarily identifying its interacting RNA targets. In our SNORD116 overexpressing cell models, the combination of transient interaction capture via psoralen crosslinking and subsequent enrichment of cross-linked RNAs, followed by high-throughput sequencing are allowing us to efficiently detect interacting events. We are using the PARIS protocol with our own modifications that support targeted approach. NTERA-2 cell line, with high endogenous expression of SNORD116, was used for hybrid library construction and for identification of SNORD116-RNA hybrids.

Proteins: structure, disorder and dynamics

P-27-001
Amino acid substitutions in essential domain of eRF3 are lethal in combination with [PSI+] prion in yeast *Saccharomyces cerevisiae*

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The essential *SUP35* gene of yeast *S. cerevisiae* encodes the translation termination factor eRF3 (Sup35p). The Sup35p consists of three domains: C-domain is essential for translation termination and possess GTF- and eRF1-binding sites. N-domain is responsible for Sup35p aggregation and [PSI^+] prion formation. M-linker is involved in the maintenance of [PSI^+]. Both *sup35* mutations and [PSI^+] cause reduction of the translation termination fidelity and lead to the nonsense suppression. It was shown that *sup35* mutations affecting the N-domain have an influence on [PSI^+] appearance and maintaining. In this work, we have studied three *sup35-m* (missense) mutations located inside the C-domain of Sup35p and affected its GTF-binding motifs. We have shown using semi-denaturing detergent agarose gel electrophoresis and fluorescence microscopy that [PSI^+] aggregates disappear after replacement of wild type *SUP35* by mutant allele. To check whether mutant Sup35p is able to aggregate we purified corresponding proteins from *E. coli* and investigate their amyloidogenicity *in vitro*. Products of *sup35-m* alleles form high-molecular weight aggregates. We assume that investigated substitutions extremely affect structure and aggregation speed, and due to this, cells cannot maintain aggregates of corresponding proteins. Significant defects in the translation termination process may be another reason of incompatibility of prion and *sup35-m*. Both [PSI^+] and *sup35-m* are strong suppressors, which working together can severely reduce cell viability. We concluded that the incompatibility of mutations that lead to amino acid substitutions in the Sup35p nonpyrogenic C-domain is associated with the alterations in [PSI^+] structure or/and translation termination process. The work was supported by RSF grant 18-14-00050. Part of experimental work was done in the resource centre of SPBU "Centre for Molecular and Cell Technologies".

P-27-002
The contribution of N- and C-terminal regions to chaperone function and oligomerization of small heat shock protein IbpA from *Acholeplasma laidlawii*

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Small heat shock proteins (sHSPs) are ubiquitous molecular chaperones that prevent the irreversible denaturation of proteins under the heat shock conditions. Well studied sHSPs IbpA and IbpB from *Escherichia coli* work in strong cooperation and are necessary for bacterial survival at high temperatures. *Acholeplasma laidlawii*, the only one free-living bacterium of Mollicutes, carries only one gene encoding the sHSP protein IbpA (*Al*IbpA). Here we report the role of the N- and C-termini of *A. laidlawii* IbpA for its oligomerization and chaperone function. Independently on the temperature, a protein forms heterogeneous mixture of globular and fibrillar structures with ratio of 1:6, while the removal of either 12 or 25 N-terminal amino acids lead to the formation of only fibrillar structures. Since in *E. coli* the IbpB blocks fibrils formation by IbpA, we suggest that the N-terminus of *Al*IbpA carries inhibitory motif which complements the lack of IbpB and is responsible for globular structure formation. By contrast, the ΔN12 and ΔN25 *Al*IbpA retained the chaperone functions on the insulin, leaving the question regarding the functional role of N-terminus opened. The deletion of the C-terminal conserved LEL motif, which is shown to be required for oligomerization of *E. coli* IbpA, or its substitution to SEP, impaired the temperature stability of *Al*IbpA and abrogated the chaperone function, while the protein remained presumably in globular state. By contrast, only N- and C-terminally truncated protein containing pure α-crystalline domain was unable to interact with substrates while formed a huge oligomeric conglomerates, probably, because of α-crystalline self-oligomerization. These data suggest non-trivial properties of *Al*IbpA, which seems to combine functions of both IbpA and IbpB-like proteins and recognizes the substrate proteins via both N- and C-termini. This research was funded by the Russian Science Foundation (project No. 17-74-20065).