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СБОРНИК ТЕЗИСОВ

BOOK OF ABSTRACTS

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DIFFERENTIAL EFFECTS OF MOLECULAR CHAPERONES AND PROTEIN-SORTING FACTORS ON YEAST PRIONS

Barbitoff Y.A.[1,], Matveenko A.G.[1,2], Moskalenko S.E.[2], Zemlyanko O.M.[1], Jay-Garcia L.M.[3], Newnam G.P.[3], Patel A.[3], Chernoff Y.O.[1,3], Zhouravleva G.A.[1]

[1]*St. Petersburg State University, Russia, Saint-Petersburg, 199034, Universitetskaya emb. 7/9*

[2]*Vavilov Institute of General Genetics of Russian Academy of Sciences, Russia, Saint-Petersburg, 199034, Universitetskaya emb. 7/9*

[3]*Georgia Institute of Technology, USA, Georgia, Atlanta, GA 30332-2000, 950 Atlantic Drive, Engineered Biosystems Bldg.*

barbitoff@bk.ru

Prions of baker's yeast *Saccharomyces cerevisiae* are heritable genetic determinants represented by self-perpeturating protein aggregates. These protein aggregates propagate due to tight interaction with the cellular protein quality control (PQC) system, *i.e.* molecular chaperones and protein-sorting factors. In this study we systematically assessed the effects of different PQC components on the propagation of the two most studied yeast prions, [*PSI*$^+$] and [*URE3*].

We show that, contrary to a common view, overexpression of the major chaperone involved in maintenance of yeast prions, *HSP104*, curees both [*PSI*$^+$] and [*URE3*]. On the other hand, overproduction of the other major protein involved in yeast prion life cycle, Hsp70-Ssa1, does not exert any observable effect on any of the prions. We also show that the two major chaperones of the Hsp40 group, Sis1 and Ydj1, exhibit differential effects on [*PSI*$^+$] and [*URE3*]. We demonstrate that overproduction of Ydj1, but not Sis1, cures [*URE3*] but enhances phenotypic manifestation of [*PSI*$^+$]. On the other hand, we show that relocalization of the other major cytosolic Hsp40 Sis1 into the nucleus affect yeast prion propagation similarly to the overexpression of *YDJ1*. We discover that such changes in the intracellular balance of Hsp40 occur upon overexpression of the protein-sorting factor Cur1, which also differentially affects yeast prion propagation. We link such differential effects of different chaperones to their varying ability to facilitate two distinct activities of the Hsp104 complex, *i.e.* fibril fragmentation and malpartition of prion seeds. Our results indicate that Sis1 is strictly required for fragmentation of the [*URE3*] fibrils, while binding of Sis1 to the prion aggregates of [*PSI*$^+$] mostly stimulates the anti-prion malpartition process. Hence, when Sis1 is downregulated or relocalized to the nucleus, such changes impair [*URE3*] propagation while not affecting or even increasing transmission of the [*PSI*$^+$] prion seeds.

Our results highlight complex interactions between different protein aggregates and molecular chaperones in the eukaryotic cells, and emphasize the importance of intracellular chaperone balance for prion propagation.

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