

THE INTERFACE BETWEEN CHRONOLOGICAL AGING AND KILLER MAINTENANCE IN *SACCHAROMYCES* YEASTS

Martynas Rojus Bartkus, Bazilė Ravoitytė, Ramunė Stanevičienė, Elena Servienė

Laboratory of Genetics, Institute of Botany, Nature Research Centre, Akademijos g. 2, 08412, Vilnius, Lithuania
martynas-rojus.bartkus@stud.vgtu.lt

Aging is a process affecting every cell of any living organism. Chronological life span (CLS) is the length of time that a non-dividing yeast cell survives. CLS studies is a model of aging of non-dividing cells of higher eukaryotes. Domesticated yeast *Saccharomyces cerevisiae* has been widely investigated in various fields of research, including aging [1]. *Saccharomyces paradoxus* is a closest relative of *S. cerevisiae* widespread in nature, which has been investigated in a less extent.

S. cerevisiae and *S. paradoxus* are known to possess *Totiviridae* double-stranded RNA (dsRNA) viruses, namely, L-A and satellite M [2]. M type dsRNA encodes a toxin and thus provides a killer phenotype and self-immunity to the host cell. Host cell and dsRNA virus are highly interconnected. Virus propagation and killer toxin synthesis require functions of various cellular proteins and components. To our knowledge, there is no published data on connection between cell aging and the dsRNA conferred killer phenotype in *Saccharomyces* yeast.

In this work, CLSs of *S. paradoxus* AML-15-66 and *S. cerevisiae* M437 killer strains, harbouring different dsRNA viruses [3,4], were compared. Colony forming units (CFUs) assay was used to evaluate yeast survival. Killing assays were performed to depict killer and non-killer cells in an aging population. Double-stranded RNA content of non-killer cells was investigated. Aging experiments in buffered and unbuffered growth medium were performed. Insights into the interface between aging and the killer yeast population is important for understanding adaptability of dsRNA viruses and their possible roles in cell survival upon different aging conditions.

[1] V. D. Longo, G. S. Shadel, M. Kaeberlein, B. Kennedy, Replicative and chronological aging in *Saccharomyces cerevisiae*. *Cell Metab.* 16(1):18–31 (2012).

[2] N. Rodríguez-Cousiño, P. Gómez, R. Esteban, Variation and distribution of L-A helper Totiviruses in *Saccharomyces sensu stricto* yeasts producing different killer toxins. *Toxins (Basel)*, 9(10), pii: E313 (2017).

[3] I. Vepšaitė-Monstavičė, J. Lukša, A. Konovalovas, D. Ežerskytė, R. Stanevičienė, Ž. Strazdaitė-Žilienė, S. Serva, E. Servienė *Saccharomyces paradoxus* K66 killer system evidences expanded assortment of helper and satellite viruses. *Viruses*, 10(10): 564 (2018).

[4] J. Lukša, B. Ravoitytė, A. Konovalovas, L. Aitmanaitė, A. Butenko, V. Yurchenko, S. Serva, E. Servienė, Different metabolic pathways are involved in response of *Saccharomyces cerevisiae* to L-A and M viruses, *Toxins (Basel)*. 9(8): 233 (2017).