Genome Sequence of *Saccharomyces cerevisiae* Double-Stranded RNA Virus L-A-28

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Killer phenotypes among *Saccharomyces cerevisiae* were discovered and linked to cytoplasm-persisting double-stranded RNAs (dsRNAs) more than four decades ago (1). Exhaustive efforts to analyze genetic determinants behind this phenomenon led to the discovery of the interaction between two viruses belonging to the *Totiviridae* family—toxin-coding M and helper L-A—which enable the killing property. Recently, the virus Klus has been added to the group of well-described viruses responsible for the K1, K2, and K28 killer phenotypes (2). Despite the established general virus propagation scheme, evolutionary connections between the L-A and M viruses remain unresolved. In particular, cross-maintenance of dsRNAs in different killer type strains is largely unexplored, even though data on the persistence of certain combinations have begun to emerge (3).

Of the four established *S. cerevisiae* killer types, K28 clearly stands out for its unique mode of entering the target cell by endocytosis and induction of apoptosis after traveling to the nucleus (4–6). Given the importance of L-A viruses in the expression and maintenance of all four known killer branches, L-A-1 representing one and L-A-2-lus with L-A-2 the other. Given the outstanding mode of action for the K28 toxin, the sequence of L-A-28 is surprisingly similar to that of other viruses of the same family; this finding provides an important evolutionary tie between L-A viruses in killer strains of *S. cerevisiae*, suggesting a common ancestor for all viruses in the Totivirus genera. The complete genome sequence of the L-A-28 virus thus completes the set for *Totiviridae* viruses involved in the expression and maintenance of all four known types of M dsRNA viruses behind the yeast killer phenotypes that have been discovered in baking yeast so far.

**Nucleotide sequence accession number.** The complete genome of *Saccharomyces cerevisiae* dsRNA virus L-A-28 has been deposited in GenBank under the accession number KU845301.

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**REFERENCES**

1. Bevan EA, Herring AJ, Mitchell DJ. 1973. Preliminary characterization of two species of dsRNA in yeast and their relationship to the "killer" character. Nature 245:81–86. http://dx.doi.org/10.1038/245081b0.
2. Rodríguez-Cousíño N, Maqueda M, Ambroña J, Zamora E, Esteban R, Ramírez M. 2011. A new wine *Saccharomyces cerevisiae* dsRNA virus (Klus), encoded by a double-stranded RNA virus, with broad antifungal activity is evolutionarily related to a chromosomal host gene. Appl Environ Microbiol 77:1822–1832. http://dx.doi.org/10.1128/AEM.02501-10.
3. Rodríguez-Cousíño N, Gómez P, Esteban R. 2013. L-A-lus, a new variant of the L-A totivirus found in wine yeasts with Klus killer toxin-encoding Mlus double-stranded RNA: possible role of killer toxin-encoding satellite RNAs in the evolution of their helper viruses.
4. Schmitt MJ, Tipper DJ. 1990. K28, a unique double-stranded RNA killer virus of Saccharomyces cerevisiae. Mol Cell Biol 10:4807–4815. http://dx.doi.org/10.1128/MCB.10.9.4807.

5. Eisfeld K, Riffer F, Mentges J, Schmitt MJ. 2000. Endocytotic uptake and retrograde transport of a virally encoded killer toxin in yeast. Mol Microbiol 37:926–940. http://dx.doi.org/10.1046/j.1365-2958.2000.02063.x.

6. Schmitt MJ, Klavehn P, Wang J, Schönig I, Tipper DJ. 1996. Cell cycle studies on the mode of action of yeast K28 killer toxin. Microbiology 142:2655–2662. http://dx.doi.org/10.1099/00221287-142-9-2655.

7. Pfeiffer P, Radler F. 1984. Comparison of the killer toxin of several yeasts and the purification of a toxin of type K2. Arch Microbiol 137:357–361. http://dx.doi.org/10.1007/BF00410734.

8. Potgieter AC, Page NA, Liebenberg J, Wright IM, Landt O, van Dijk AA. 2009. Improved strategies for sequence-independent amplification and sequencing of viral double-stranded RNA genomes. J Gen Virol 90:1423–1432. http://dx.doi.org/10.1099/vir.0.009381-0.