

## SUPPORTING INFORMATION

### A bipolar functionality of Q/N-rich proteins: Lsm4 amyloid causes clearance of yeast prions

Keita Oishi, Hiroshi Kurahashi, Chan-Gi Park,  
Yasushi Sako and Yoshikazu Nakamura\*

#### Supporting Materials

Table S1. PCR Primers.

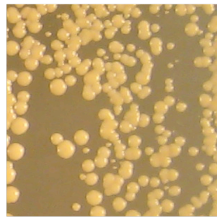
Primer	Sequence (5'-to-3')
P1	GGGGATCCATGCTACCTTTATATCTTTTAAC
P2	CCCCTCGAGTTAAAATTCGACCTTTTGTG
P3	CCCGAGCTCGATTTCTCGAATAAATC
P4	TTTGTGCGACTCCGCTTCCTCCTCCGCTTCCTCCTCAAATTCGACCTTTTGTGG
P5	CCCGTCGACATGGCCTCCTCCGAGGACG
P6	GGGCTCGAGTTAGGCGCCGGTGGAGTGG
P7	GGGCTCGAGTTACTTGACCTTGTCAATTATATTATC
P8	CCCCTCGAGTTAGTAGTATCTTTTATGCC
P9	CCCCTCGAGTTAGCCGTTATTATTATTTTC
P10	CCCCTCGAGTTAGTTACTGCTGTTGCTGTTG
P11	CCCCTCGAGTTAACCTAAACCATTGTTC
P12	CCCGGATCCATGCAGCAAATTAACCTCCAAC
P13	CCCGGATCCAGCGTAATCTGGAACATCGTATGGGTACATACTAGTTCTAGAATCCG
P14	CCCCATATGCTACCTTTATATCTTTTAAC
P15	CCCGGATCCATGCCTCAAAGAAGTTTAAGG
P16	GGGCTCGAGTCAATCTTCTTCATCGTCAG
P17	CCCGGATCCATGACATCAGTTCAAAAAC
P18	GGGCTCGAGTTACATAAGCGTACAACAAAAC
P19	CCCGGATCCATGGATTTCTTTAATTTG
P20	GGGCTCGAGTCATTCAAATTTGGTTAGG

P21 CCCGGATCCATGAACGTTTGCAAGTTG  
P22 GGGCTCGAGTTAGTAATATCTATCGCTTTG  
P23 CCCGGATCCATGATGAATAACAACGGC  
P24 GGGCTCGAGTCATTCACCACGCAATGC  
P25 CCCGGATCCATGGAGACCAGTTCTTTTG  
P26 GGGCTCGAGTTACCATAGATTCTTCTTG  
P27 CCCGGATCCATGAATCCGGGCGGTGAAC  
P28 GGGCTCGAGTTAGTCGTAGTTTTTC  
P29 CCCGGATCCATGATGAATAACAACGGC  
P30 GGGCTCGAGTCATTCACCACGCAATGC  
P31 CCCAGATCTATGTCTGCTTCATTGATTAATC  
P32 GGGCTCGAGTTAAAAGATATTATTAAC  
P33 CCCGGATCCATGTCCATGCCATAGCAAG  
P34 GGGCTCGAGTTAGCAACAACCTAATTTTTTG  
P35 CCCAGATCTATGTTTGGAGTTAGCCGTG  
P36 GGGCTCGAGTCAGGTCTGCTCTGCAGCG

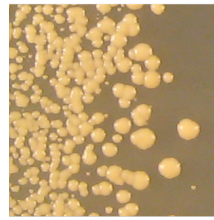
---

## Supporting Figures

A

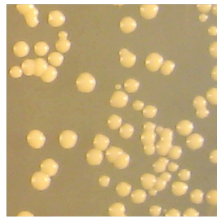


NPK50 [*PSI*<sup>+</sup>]  
+ empty vector

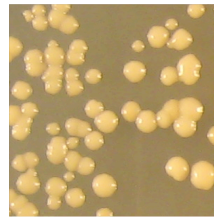


NPK50 [*PSI*<sup>+</sup>]  
+ pLSM4

B



NPK302 [*URE3*]  
+ empty vector

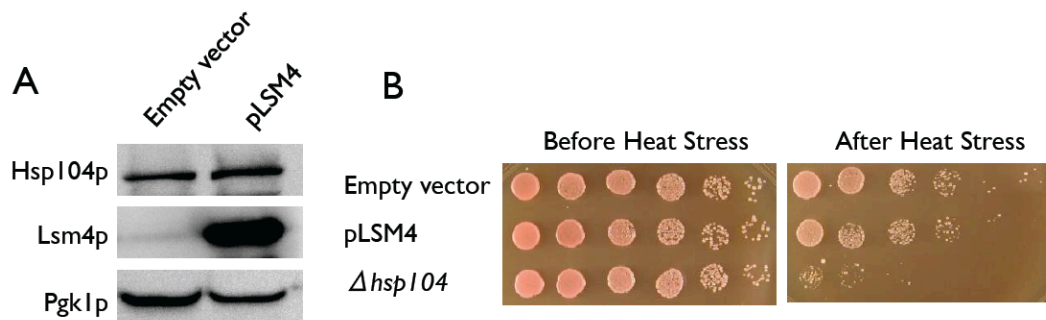


NPK302 [*URE3*]  
+ pLSM4

**Fig. S1.** Absence of the growth inhibition effect in [*PSI*<sup>+</sup>] or [*URE3*] cells harboring an *LSM4* overexpressing plasmid on SC-leu selective media.

A. Absence of the growth inhibition effect in [*PSI*<sup>+</sup>] cells. [*PSI*<sup>+</sup>][*rnq*<sup>-</sup>] cells (NPK50) were transformed with pRS425 (an empty vector) or pRS425GPDp-*LSM4* (a multicopy *LEU2*<sup>+</sup> plasmid expressing *LSM4* under the control of the *GPD* promoter; denoted by pLSM4). Transformants were subsequently incubated on SC-leu media for 3 days.

B. Absence of the growth inhibition effect in [*URE3*] cells. [*URE3*][*rnq*<sup>-</sup>] cells (NPK302) were subjected to the same experiment as A.



**Fig. S2.** Cellular abundance and thermo-tolerance activity of Hsp104 are unaffected by *LSM4* expression.

A. Cellular abundance of Hsp104 on *LSM4* overexpression. *LSM4* was overexpressed from pRS425GPDp-*LSM4* (under the *GPD* promoter) in NPK302 [*URE3*][*rnq*<sup>-</sup>] strain. Due to the low amount of lysate loaded, the Lsm4 protein expressed from the genome (see the lane of “Empty vector”) was narrowly observed. Immunoblotting was carried out using anti-Hsp104 antibody, anti-Lsm4 antibody and anti-Pgk1 antibody.

B. Thermotolerance of *LSM4*-overexpressing cells. Cultures in the mid-log phase were incubated at 37°C for 1 hour, and then heat-treated at 50°C for 20 min. Survival rates were visualized by 5-fold serial dilutions on YPD plate with untreated controls. Used strains were NPK301 ([*ure-o*][*rnq*<sup>-</sup>]) carrying an empty vector (top) or pRS425GPDp-*LSM4* (middle), and NPK377 (NPK301-derivative carrying *Δhsp104*; bottom).