

## **Supplemental data:**

Genetic interaction with gene deletion (GID) detailed protocols :

Supplementary Figure 1 describes the GID screening procedure. Colony colors on lead-containing medium are shown in supplementary Figure 2. Annotated map of vectors pRS316-RCF1, pFL36-RCF1, pAG32-ttt, pGID1-RCF1 and pGID2 are shown in supplementary Figure 3.

Contruction of the test strain : The *KAN-MX4* cassette used to delete *HMO1* in the systematic deletion library made in the BY4742 *MAT-α* background was substituted by homologous recombination with an *AscI-EcoRI* digest of the pGID2 plasmid (Figure S2). pGID2 contains the *NAT* gene (Nourseotricin resistance gene from the pAG25 plasmid (5)) under the control of the *MF(ALPHA)2 MAT-α* specific promoter. pGID2 also contains and the tetracycline sensitive tTA transactivator expressed from the *pCMV* promoter (from plasmid the pCM190 plasmid (3)). *HMO1* was cloned in the pGID1-RFC1 plasmid by gateway recombination (Figure S3). pGID1 incorporates the following features: The *MET15* prototrophy/color marker from plasmid pGC3 (2) (allowing to score for the presence of the plasmid based on color on lead-containing medium ; see figure S2), a *URA3* marker (that allows both positive and negative selection for controls), an Hygromycin B resistance cassette (from the pAG32 plasmid; (5)) (6)) and an *ARS-CEN6* cassette from plasmid pFL38 (1). The tetracycline regulatable promoter (from pCM190 (3), consisting of 7 copies of TetO operator sequence upstream of the *CYC1* minimal promoter) is placed just upstream from the *CEN6* sequence. In the presence of the tTA transactivator encoded in pGID2, transcription driven by TetR-VP16 protein (tTA ) inhibits the *CEN6* centromere function. In presence of doxycycline, the plasmid is stabilized by dissociation of TetR-VP16 from TetO sites. This construct results in doxycycline-dependent stability of the plasmid.

Step I: Substitution of the deletion-cassette. Plasmid pGID2 (Figure S2) was *EcoRI-AscI* digested and transformed into strain Y16969 to replace the Kanamycin-resistance-marker by the *NAT*-resistance gene under the control of the *MF(ALPHA)2* alpha-specific promoter (see figure S1). Transformed cells were spread on a Hybond N-filter (Amersham) on YPD containing 10 µg/ml doxycycline (Sigma). After 1 day the filter was transfered onto plates with YPD containing 10 µg/ml doxycycline and 60 µg/ml

Nourseothricin-sulfate (“cloNAT”, Werner Bioagents). Clones were checked by restreaking candidates onto YPD containing 20 µg/ml Doxycycline and 60 µg/ml Nourseothricin-sulfate and at the same time on YPD containing 200 µg/ml G418 sulfate (PAA). A nourseothricin-resistant and Kanamycine (G418)-sensitive clone (YAB41-1a) was chosen.

Step II: Construction of a *MAT $\alpha$  hmo1 $\Delta$  met15- lys2-* strain. YAB41-1a was crossed with BY4741, a segregant was screened for their ability to grow on SC -Lysine and having a dark color (*met15 $\Delta$*  strain) on Pb<sup>2+</sup> containing medium (2). This strain was termed YAB2-2a.

Step III: Transformation of pGID1-HMO1 into YAB2-2a. Transformants were selected on YPD containing 10 µg/ml doxycycline and 200 µg/ml hygromycin (Roche).

Step IV: YAB2-2a containing pGID1-HMO1 was crossed with 42 deletion pools, each pool containing approximately 100 Euroscarf systematic deletion mutants. Crossing was done for 4.5 hours on YPD plates containing doxycycline, and diploids were then streaked on GNA pre-sporulation medium (4) containing doxycycline, hygromycin and G418.

Step V: Sporulation. The 42 crossed pools were resuspended in Sporulation medium (1% Potassium Acetate, 0,005% Zinc Acetate) and incubated for 5 days at 25°C. The temperature was then shifted to 30°C for 3 more days to maximize sporulation efficiency. 200µl of sporulated yeast cultures were spread on a Hybond N filter placed on a 9 cm YPD plates containing 60 µg/ml nourseothricin-sulfate and 100 µg/ml G418. After 2 days, filters were transferred onto 9 cm YPD plates containing Pb<sup>2+</sup> medium, 60 µg/ml nourseothricin-sulfate and 100 µg/ml G418.

White or light beige appearing colonies were restreaked onto filters. Similarly, these filters were kept 2 days on YPD containing 60 µg/ml nourseothricin-sulfate, 100 µg/ml G418 and 10 µg/ml doxycycline, and then transferred onto YPD plates containing the same antibiotics, with plus Pb<sup>2+</sup> medium.

Step VI: White or beige-appearing colonies were isolated on SC –Uracil medium containing doxycycline and SC medium containing 5-FOA. Clones sensitive to 5-FOA were kept.

Step VII: Complementation test. 5-FOA sensitive clones were transformed with pFL36CII and with pFL36CII-HMO1 and scored for *HMO1* dependent growth on 5-FOA containing medium. Clones showing significant *HMO1* growth dependency were scored as genetic interactors.

Step VIII: A PCR was performed with genomic DNA from genetic interactors using a Oligos U1-KD1 (AAGAAGAACCTCAGTGGC) and KU2-D1 (GGATCTTGCCATCCTATG) that amplify the bar codes. The amplicon was sequenced with oligos SKD1 (TCCTAACCTTTTATATTTCTC) and SKU2 (ATGGTATTGATAATCCTGATATG).

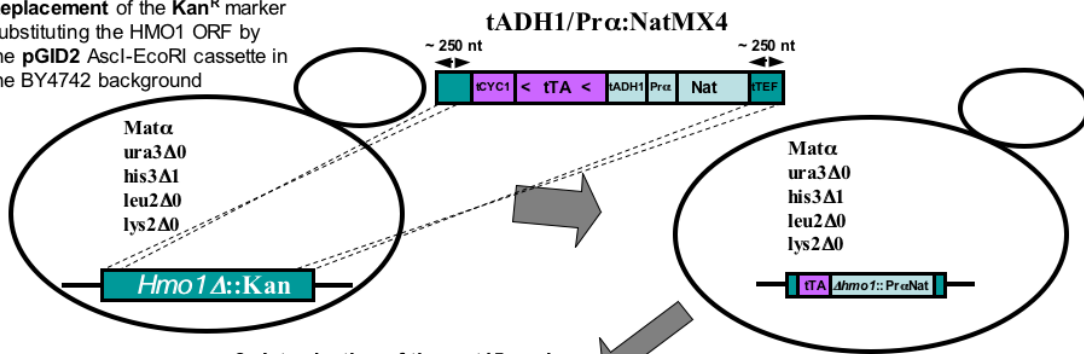
Supplementary Table II: synthetic interactors

NAME	GENE	Overlapping essential genes	Fold	Growth of the double mutant	Functional group	Info SGD, YPD
YNL248C	RPA49		0	1 - synthetic lethal (SL)	1 - transcription	RNA polymerase I subunit A49.
YJR063W	RPA12		0	1 - synthetic lethal (SL)	1 - transcription	RNA polymerase I subunit A12.
YJL075C	Dubious ORF	NET1	1	1 - synthetic lethal (SL)	1 - transcription	Core subunit of the RENT complex, stimulates transcription by RNA polymerase I and regulates nucleolar structure.
YML009C-A	Dubious ORF	SPT5	1	2 - slow growth (ss)	1 - transcription	Protein that forms a complex with Spt4p and acts on transcription elongation.
YJL148W	RPA34		0	2 - slow growth (ss)	1 - transcription	RNA polymerase I subunit A34.5.
YLR234W	TOP3		0	2 - slow growth (ss)	1 - transcription	DNA topoisomerase III, relaxes negatively supercoiled DNA, null mutation is synthetically lethal with <i>rpa49</i> and with <i>rpa43-24</i> mutation
YGL025C	PGD1		2	3 - slightly slower growth (sss)	1 - transcription	Subunit of the Mediator global transcriptional cofactor complex, plays an essential role in basal and activated transcription
YLR055C	SPT8		1	3 - slightly slower growth (sss)	1 - transcription	Subunit of the SAGA transcriptional regulatory complex, required for SAGA-mediated inhibition at some promoters
YGR200C	ELP2		1	3 - slightly slower growth (sss)	1 - transcription 3 - stress response	Part of the RNA polymerase II Elongator, involved in nutrient sensing, null mutant is more sensitive to rapamycin than wild type.
YPL086C	ELP3		1	3 - slightly slower growth (sss)	1 - transcription 3 - stress response	Part of the RNA polymerase II Elongator, involved in nutrient sensing, null mutant is more sensitive to rapamycin than wild type.
YOL121C	RPS19A		5	1 - synthetic lethal (SL)	2 - ribosome	Protein component of the small (40S) ribosomal subunit.
YDR457W	TOM1		2	1 - synthetic lethal (SL)	2 - ribosome	E3 ubiquitin ligase of the hect-domain class, involved in the maturation of ribosomal RNA components .
YPR132W	RPS23B		1	1 - synthetic lethal (SL)	2 - ribosome	Protein component of the small (40S) ribosomal subunit.
YBR048W	RPS11B		1	2 - slow growth (ss)	2 - ribosome	Protein component of the small (40S) ribosomal subunit.
YMR143W	RPS16A		1	2 - slow growth (ss)	2 - ribosome	Protein component of the small (40S) ribosomal subunit.
YBR181C	RPS6B		3	2 - slow growth (ss)	2 - ribosome	Protein component of the small (40S) ribosomal subunit.
YIL133C	RPL16A		2	2 - slow growth (ss)	2 - ribosome	Protein component of the large (60S) ribosomal subunit.
YJR145C	RPS4A		1	3 - slightly slower growth (sss)	2 - ribosome	Protein component of the small (40S) ribosomal subunit.
YNL135C	FPR1		22	1 - synthetic lethal (SL)	3 - stress response	Peptidyl-prolyl cis-trans isomerase (PPIase), binds to rapamycin and inhibits TORC1, null mutant exhibits resistant to rapamycin.
YML016C	PPZ1		1	2 - slow growth (ss)	3 - stress response	Protein serine/threonine phosphatase required for normal osmoregulation, null mutant exhibits increased resistance to rapamycin
YOL001W	PHO80		1	2 - slow growth (ss)	3 - stress response	Cyclin that interacts with Pho85p cyclin-dependent kinase, null mutant is sensitive to rapamycin
YFR021W	ATG18		1	2 - slow growth (ss)	3 - stress response	Protein that plays a role in autophagy, pexophagy, and cytoplasm-to-vacuole targeting, possibly involved in amino acid signaling pathways
YNL119W	NCS2		1	2 - slow growth (ss)	3 - stress response	Involved in nutrient sensing, cytokinesis, and pseudohyphal growth, null mutant is sensitive to rapamycin
YGL227W	VID30		1	2 - slow growth (ss)	3 - stress response	Protein that regulates expression of genes that are involved in nitrogen metabolism, induced by rapamycin, null mutant is more sensitive to rapamycin than wild type.
YPL049C	DIG1		1	3 - slightly slower growth (sss)	3 - stress response	Transcription factor, MAP kinase-associated protein involved in negative regulation of invasive growth.
YLR113W	HOG1		2	3 - slightly slower growth (sss)	3 - stress response	Mitogen-activated protein kinase involved in osmolarity response, the principal component of the high-osmolarity signal transduction pathway.
YOR018W	ROD1		1	3 - slightly slower growth (sss)	3 - stress response	Membrane protein; interact with Rsp5p, an essential hect-type ubiquitin ligase
YDR368W	YPR1		1	3 - slightly slower growth (sss)	3 - stress response	2-methylbutyraldehyde reductase, induced by osmotic and oxidative stress, may play a role in protection from oxidative stress and lipid peroxidation products.
YNL076W	MKS1		3	3 - slightly slower growth (sss)	3 - stress response	Pleiotropic regulatory factor, inhibited by TORC1 through Tap42p, null mutant has increased resistance to rapamycin than wild type.
YPL180W	TCO89		2	3 - slightly slower growth (sss)	3 - stress response	non essential subunit of TORC1 (Tor1p or Tor2p-Kog1p-Lsi8p-Tco89p), null mutant is hypersensitive to rapamycin than wild type.
YJL095W	BCK1		1	3 - slightly slower growth (sss)	3 - stress response	Serine-threonine protein kinase of the MEKK null mutant is sensitive to rapamycin.
YLR371W	ROM2		2	3 - slightly slower growth (sss)	3 - stress response	a GDP-GTP exchange factor for Rho1p that is activated by cell wall defects, only partial rapamycin dependt induction of Slt2p activity relative to wild type.

A

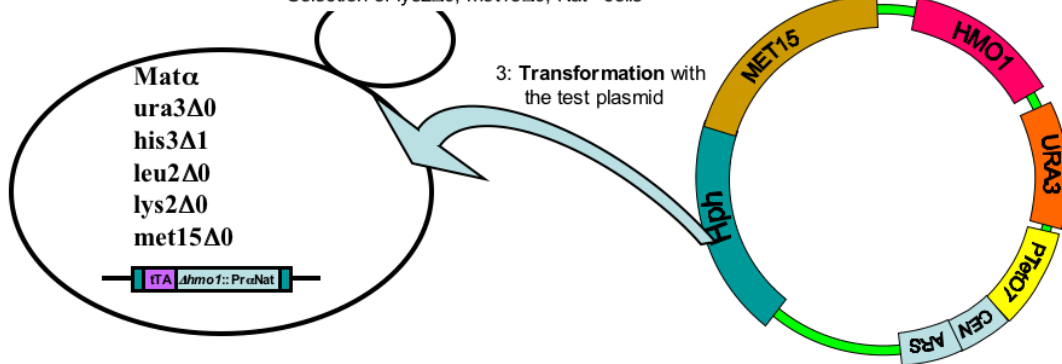
## Construction of the test strain:

- 1: **Replacement** of the Kan<sup>R</sup> marker substituting the HMO1 ORF by the pGID2 AscI-EcoRI cassette in the BY4742 background



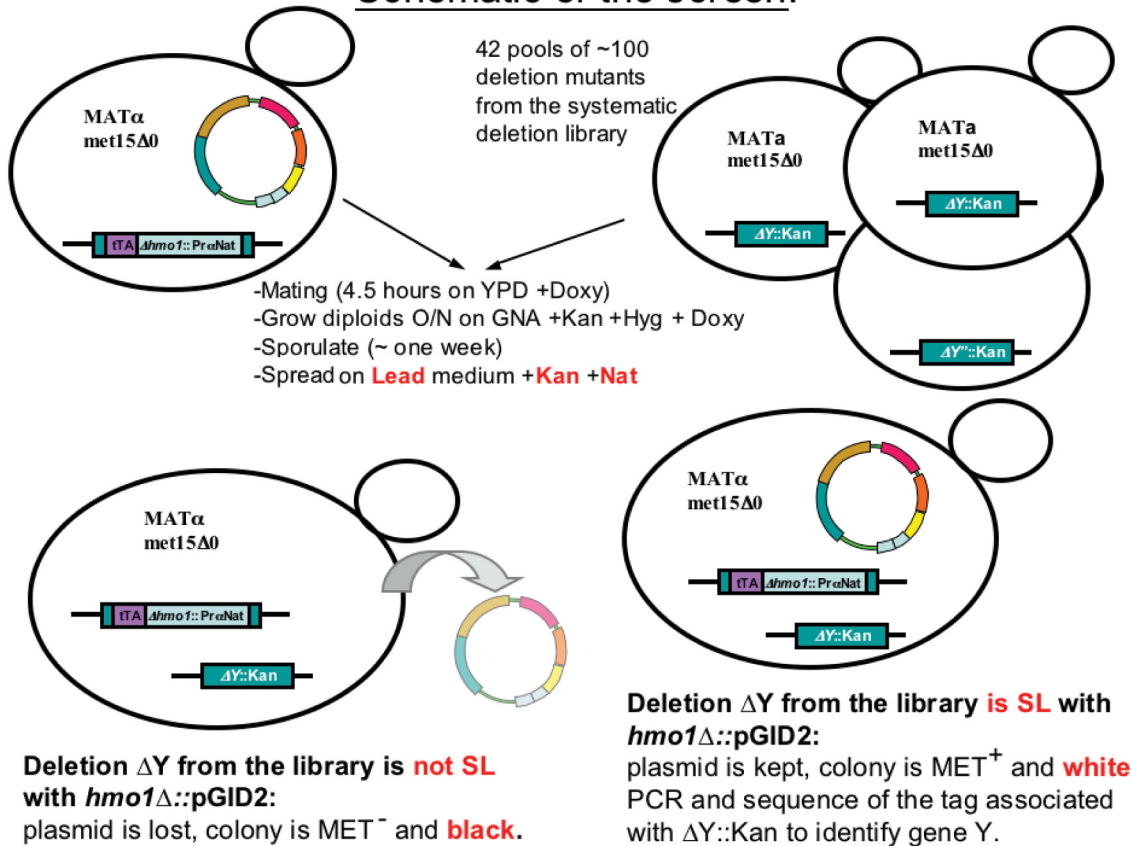
- 2: **Introduction of the met15 marker:**

- Mating with BY4741:  
(MATα; ura3Δ0; his3Δ1; leu2Δ0; met15Δ0)
- In mass sporulation and selection of haploid cells on Nat
- Selection of lys2Δ0; met15Δ0; Nat<sup>R</sup> cells



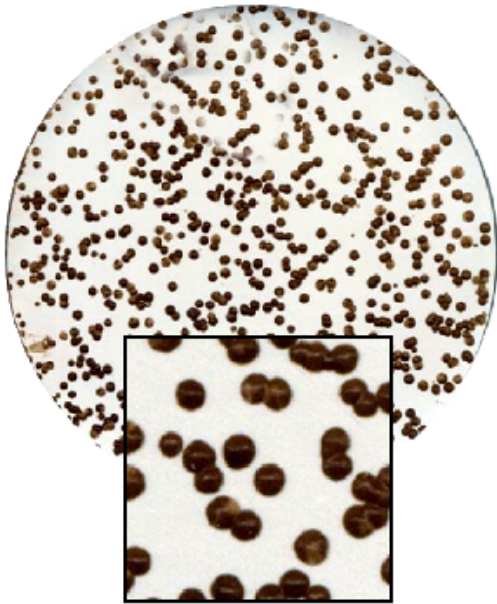
B

## Schematic of the screen:

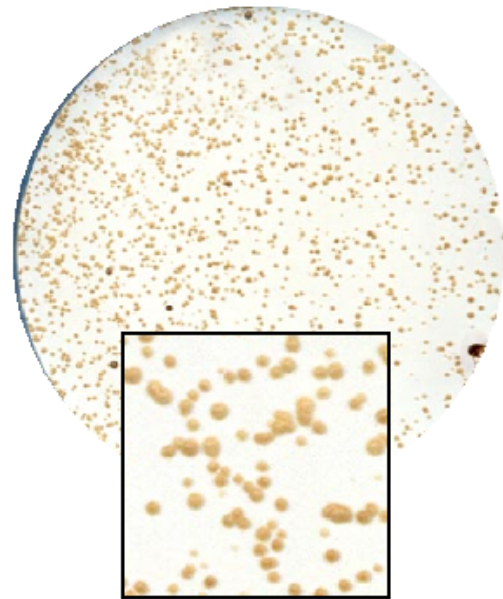


# Result of reconstituted Synthetic Lethal test screen on lead containing medium

non synthetic lethal mutants



synthetic lethal mutants

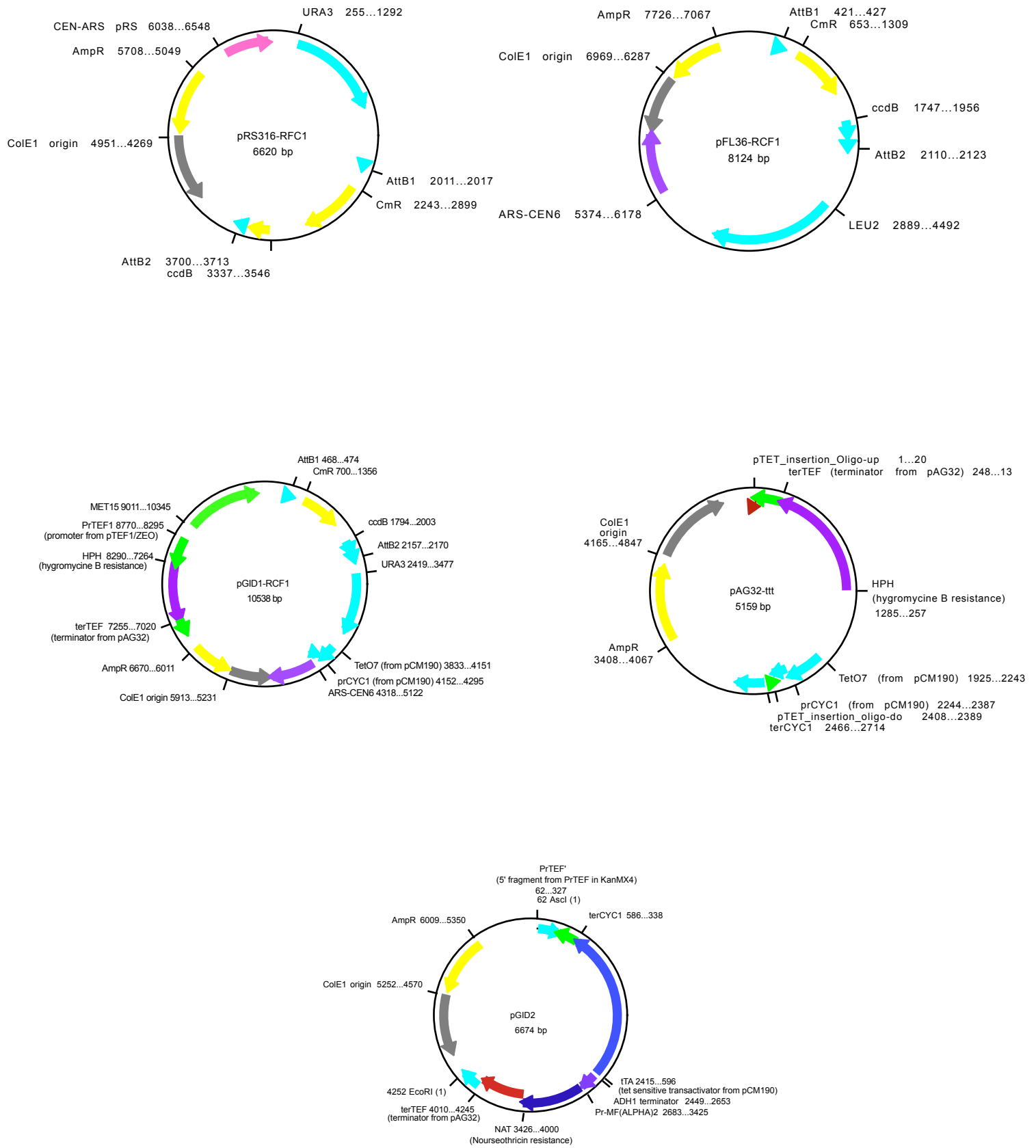


1 out of 10 synthetic lethal mutants

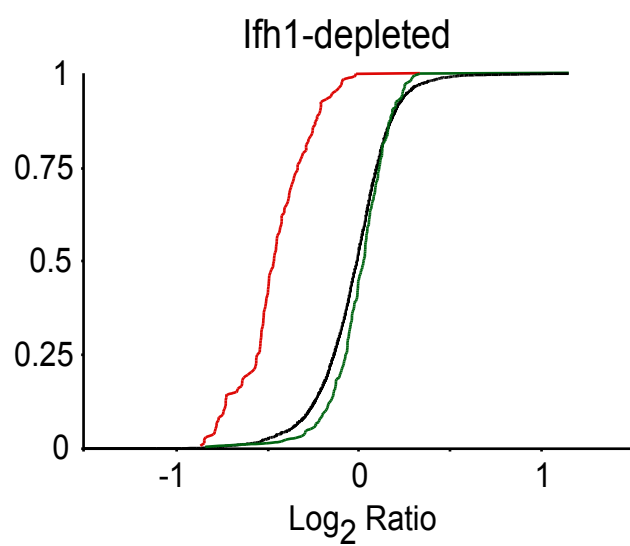
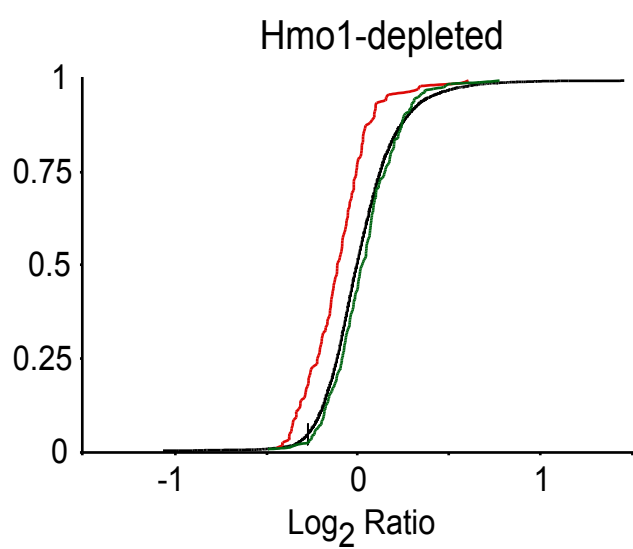


24 white for 286 black colonies

Supplementary figure S2 (Berger et al., 2007)



Supplementary figure S3 (Berger et al., 2007)



Supplementary figure S4 (Berger et al., 2007)



## Supplementary figure legends

Supplementary figure S1. (A) Schematic of the test strain construction steps. (B) Schematic of the GID screening steps.

Supplementary figure S2. Published synthetic lethal (SL) mutants (*vps72Δ*, *cdc73Δ*) were used to assess the GID system. Non SL mutants (*vps72Δ*, *edc3Δ*) were used as a negative control. The VPS72 gene was cloned into pGID1 and transformed into a *vps72Δ* test strain constructed as described for HMO1. The figure illustrates the appearance of colonies combining the non-SL mutants (*vps72Δ*, *edc3Δ*; upper left panel), the SL mutants (*vps72Δ*, *cdc73Δ*; upper right panel) or a 9 to 1 mixture of these two (lower panel). The numbers below the panel indicate the number of white and black colonies counted on the plate. For each panel, a close-up view of a portion of the plate (square field) is shown on top of a complete view of the plate. Dark-brown colonies have lost the pGID1-VPS72 plasmid and are thus *met15Δ*, which confers the dark color to the colony on the lead-containing medium.

Supplementary figure S3. The figure depicts the schematics of the plasmids used in this study. The schematics were drawn using the "ApE" software (<http://www.biology.utah.edu/jorgensen/wayned/ap/>). The reconstituted sequences of these plasmids can be obtained at (<http://www.pasteur.fr/recherche/unites/Gim/supplementary/...>).

Supplementary figure S4. Transcriptome analysis of Hmo1 and Ifh1 depletion. Cumulative distribution of transcript variation of all genes (black line), the 138 ribosomal protein genes (red), or the 236 genes of the Ribi regulon (green) upon Hmo1 or Ifh1 depletion. Total RNA was extracted and reverse transcribed from a strain that was depleted for Hmo1 or for Ifh1 (Ifh1-Depleted). Depletion of Hmo1 or of Ifh1 was performed during 6 hours by adding 5 µg/ml doxycycline to YAB111-1a or to YAB103-1a respectively, using untreated cells as controls.

Supplementary references :

1. **Bonneaud, N., O. Ozier-Kalogeropoulos, G. Li, M. Labouesse, L. Minvielle-Sebastia, and F. Lacroute.** 1991. A family of low and high copy replicative, integrative and single-stranded S. cerevisiae/ E. coli shuttle vectors. *Yeast* **7**:609-615.
2. **Cost, G. J., and J. D. Boeke.** 1996. A useful colony colour phenotype associated with the yeast selectable/counter-selectable marker MET15. *Yeast* **12**:939-41.
3. **Gari, E., L. Piedrafita, M. Aldea, and E. Herrero.** 1997. A set of vectors with a tetracycline-regulatable promoter system for modulated gene expression in *Saccharomyces cerevisiae*. *Yeast* **13**:837-48.
4. **Giaever, G., A. M. Chu, L. Ni, C. Connelly, L. Riles, S. Veronneau, S. Dow, A. Lucau-Danila, K. Anderson, B. Andre, A. P. Arkin, A. Astromoff, M. El-Bakkoury, R. Bangham, R. Benito, S. Brachat, S. Campanaro, M. Curtiss, K. Davis, A. Deutschbauer, K. D. Entian, P. Flaherty, F. Foury, D. J. Garfinkel, M. Gerstein, D. Gotte, U. Guldener, J. H. Hegemann, S. Hempel, Z. Herman, D. F. Jaramillo, D. E. Kelly, S. L. Kelly, P. Kotter, D. LaBonte, D. C. Lamb, N. Lan, H. Liang, H. Liao, L. Liu, C. Luo, M. Lussier, R. Mao, P. Menard, S. L. Ooi, J. L. Revuelta, C. J. Roberts, M. Rose, P. Ross-Macdonald, B. Scherens, G. Schimmack, B. Shafer, D. D. Shoemaker, S. Sookhai-Mahadeo, R. K. Storms, J. N. Strathern, G. Valle, M. Voet, G. Volckaert, C. Y. Wang, T. R. Ward, J. Wilhelmy, E. A. Winzeler, Y. Yang, G. Yen, E. Youngman, K. Yu, H. Bussey, J. D. Boeke, M. Snyder, P. Philippsen, R. W. Davis, and M. Johnston.** 2002. Functional profiling of the *Saccharomyces cerevisiae* genome. *Nature* **418**:387-91.
5. **Goldstein, A. L., and J. H. McCusker.** 1999. Three new dominant drug resistance cassettes for gene disruption in *Saccharomyces cerevisiae*. *Yeast* **15**:1541-53.
6. **Walhout, A. J., G. F. Temple, M. A. Brasch, J. L. Hartley, M. A. Lorson, S. van den Heuvel, and M. Vidal.** 2000. GATEWAY recombinational cloning: application to the cloning of large numbers of open reading frames or ORFeomes. *Methods Enzymol* **328**:575-92.

Name	N°	Sequence
o-F2 HM01	626	GGATCTTCGGAAAAGAAGAAGAAGAAGAAGAGTAAGAAGAAGGACAAATCCAACCTTCTATTCCGGATCCCCGGGTTAATTAA
o-R1 HM01	627	ATTCCCTTTTTTATTATTATATTATTTTGTAGAAAGACAGTAGAGTAATAGTAACGAGTTTGTCCGTCAGAAATTCGAGCTCGTTTAAAC
o-F2 FHL1	2000	TCAACGAGGGTATCACTGAACATAATGTATCCCTTGAGGAAAAACTTCGGATCCCCGGGTTAATTAA
o-R1 FHL1	2002	AAAGATTTATGCTTCTACTTCGAATATGCTAATACTATTATCGAAATTTTGAATTCGAGCTCGTTTAAAC
o-HM01 -200bp am	878	GTGTGGCGGAGAGAAGCTTCCGGAAGGGAA
o-HM01-300bp am	879	CTACTCTGAGAAATATTACCCGAC
o-HM01+300bp av	880	AAAGAACGGCCCCCTGTATATATT
o-Top1 am del M13	881	TCTTTATAGTATTAACACAGCAAAATAAAAAAACTCAAAGGGAGGGCAGAGCTCGAAACTTGAAACGCGTAAAAACGACGCCAGT
o-Top1 av del M13	882	ATAAGATATCTTCCCTAGTAACCCCTAATGCGAACTTGATGCGTGAATGTATTTGCTTCTCCCTATGCTGCGGAAACAGCTATGACCATG
o-Top1-300bp am	883	AAACCTTTTTCACCTCCGGGTAATAC
o-Top1+300bp av	884	AAGGGCATAACATGCGACAAGGGACG
o-HM01+450bp av	885	CAAGGCATACATAGCTAAGAGTT
o-HM01-450bp am	886	GTATGCACATATCCATGTAAGCTAC
o-HM01-DM13 am	887	CCTCAGGGCTGGTCTACTGCCTTATACTCTAGGATGTACATCCTACCACACACAAGCCTGTCACACCGTAAAAACGACGCCAGT
o-HM01-DM13 av	888	TGCTATATTTTATTCCTTTTTTATATTATATTTATTTAGAAAGACAGTAGAGTAATAGTAACGAGTTTGTCCGTCAGGAAACAGCTATGACCATG
o-Hm01 attB1	1529	GGGACAAAGTTTGTACAAAAAAGCAGGCTAGCTATAATTGTTTACCGTATCCATGTG
o-Hm01 attB2	1530	GGGACCACTTTGTACAAAGAAAGCTGGGTGATATTGTAGATCAATATTATGAGCACTC
o-FPR1 up	1547	CACCGGAGGAATTCTGGCGAAAAGTTGG
o-FPR1 do	1548	CTCATCCGTTCCATAGAGGTATCTG
rdNA 1.am	889	TGCGCTATGGTCACCCACTAC
rdNA 1.av	890	GTTCGGCCATATCTACCAGA
rdNA 2.am	891	ATGTGGGAATAAGGTGCATACGAT
rdNA 2.av	892	TTGAAACTACCTCTGCATGCCA
rdNA 3.am	893	CGAGTATTGAGACCATGAGAGTAGCA
rdNA 3.av	894	TCCAAATGAAAATGGCTATCG
rdNA 4.am	895	CCGTTATTGGTAGGAGTGTGGTG
rdNA 4.av	896	ACGGAAATACGCTTCAGAGACC
rdNA 5.am	897	CCGCGGCTTCCGTATT
rdNA 5.av	898	GCCGAGAAAAAATTCAATTTAAGCTA
rdNA 6.am	899	AAAGCAGTTGAAGACAAGTTCGAA
rdNA 6.av	1500	GACTCTCTCCACCGTTTGACG
rdNA 7.am	1501	CTTGCTCAAGATTAAAGCCATGC
rdNA 7.am	1502	ACCACAGTTATACCATGTAGTAAAGGAACT
rdNA 8.am	1503	GCGAACCAAGGACTTTTACTTTGA
rdNA 8.av	1504	AACAAATAGAACCAACGCTCCTATTC
rdNA 9.am	1505	GGTCTGTGATGCCCTTAGACG
rdNA 9.av	1506	AGTTTCAAGATTACCAAGACCTCTC
rdNA 10.am	1507	AATATTAACAACTTCAACAACGGATCTCT
rdNA 10.av	1508	CGATGATTACCGAATTTCTGC
rdNA 11.am	1509	GGTGGTAAATTCATCTAAAGCTAAATATT
rdNA 11.av	1510	CACGTACTTTTCACTCTCTTTTCAAA
rdNA 12.am	1511	TATGAGGTAAGCGAATGATTAGAGG
rdNA 12.av	1512	CACGTTCAATTAGTAACAAGGACTTCT
rdNA 13.am	1513	GGAGGAGTTATCTTTTCTTCTTAACAGCT
rdNA 13.av	1514	AAGGTGCTGGCCTCTTCCA
rdNA 14.am	1515	AAAGAAGACCTGTTGAGCTTGA
rdNA 14.av	1516	GTATTTCACTGGCGCCGAA
rdNA 15.am	1517	GCTACCATCCGCTGGATTATG
rdNA 15.av	1518	GCCTTATTCGTATCCATCTATATTGTGT
rdNA 16.am	1519	ATCATTTGTATACGACTTAGATGTACAACG
rdNA 16.av	1520	AACAAATCAGACAACAAGGCTTAATC
rdNA 17.am	1521	TACGATGAGGATGATAGTGTGTAAGAGTG
rdNA 17.av	1522	TCTCTTTCAACCCATCTTTTGCAA
rdNA 18.am	1523	CTCATTTCTATAGTTAACAGGACATGC
rdNA 18.av	1524	TTCACTTGTCTCTTACATCTTTCTTTGG
rdNA 19.am	1525	TAACAGATATGGAATGGTTGGCG
rdNA 19.av	1526	TGCCGATTAACATATATGATCG
rdNA 20.am	1527	GTACATATCAAGTAGTAGCAACCCAATGAG
rdNA 20.av	1528	ACCATTCGATTACAGAAAAATTCG
RPS5 Prom forw	2035	CTCCCGCCCAGAACGTAAA
RPS5 Prom rev	2036	AGCTTGCAAAAGGAGATTTCCT
RPL16B Prom forw	2037	GTTTCTCAACGCTTCGGCC
RPL16B Prom rev	2038	GCGGGCAGTTTCAGGTTCTA
RPL5 Prom forw	2039	GAAGATTCTTGTTCATGTGATACAGCTT
RPL5 Prom rev	2040	TTTGTCTTTATGGGTGTTTAGGAT
RPS20 Prom forw	2041	GAGAGATTTCCTGAAACTTCTTATTTT
RPS20 Prom rev	2042	GTACAGGTGTGAACCTTGCAAGTAAA
RPL16B forw	2011	CTGCTTGGAACTTTAAAGATCT
RPL16B rev	2012	AAGCTTGAGGAACGACAACCTCTCT
RPS5 forw	2025	TGGTGTGCTAGACGCTCAAGC
RPS5 rev	2026	TCTCTGGCACCAATGGTCAAC
RPS20 forw	2029	GGTCAGTCAAGTACCAACCAAG
RPS20 rev	2030	TTTCCCAACTCTTAGAACCTTCACC
RPL5 forw	2031	TGCTGCCTACTCCCACGAAT
RPL5 rev	2032	GCGATCAACAAACCAAGTACGG
ACT1 forw	1122	ACACTTGTGGTGAACGATAGATGG
ACT1 rev	1123	CCGCTGGATTGGTGGTTCTATC
ptet-IFH1 up	1806	GAAATTGAAGGAAATTCAACAAGGAAGCAAAATAAACAAATAAGGAAAAACAACCGGCAAACTGGAAACAGAACGAAGAATTTCGAGCTCGTTTTCGA
ptet-IFH1 do	1807	AATATTGTCTGSCAGTTTACCAGAATGTGTAATGATGATGCTACTTTTTCGAGGACTTTTTTTCGCTGCCATGGATCCCCCGAATTGATCCG
ptet-KOg1 up	1597	GATTCCCTTTGATTACATTTAGCGAATCCTATTGCATGCAGAGAAGGGTAAAAAGATACATAGAAATTCGAGCTCGTTTTCGA
ptet-KOg1 do	1598	GCTCTTCAAAACCATGTCTCATCCCGTATTGAGTGGCTTTAATGGCTGAGGTCCATAAATCTCCGGCATGGATCCCCCGAATTGATCCG
ptet-HM01 up	1856	CCTCAGGGCTGTCTACTGCCTTATACTCTAGGATGTACATCCTACCACACACAACAGCCTGTCACACCGAATTCGAGCTCGTTTTTCGA
ptet-HM01 do	1857	ATAACTCGAAGAGGGAGGAGACGAGGGAGTCTTTGGCGGACTTCAATTGTACAGAAGGATCTGTAGTCATGGATCCCCCGAATTGATCCG