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SWI/SNF antagonizes SIR heterochromatin to promote transcription of genes expressed during mitotic exit in Saccharomyces cerevisiae
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#### Abstract

: Heterochromatin is a repressive, specialized chromatin structure that is central to eukaryotic transcriptional regulation and genome stability. In the budding yeast, Saccharomyces cerevisiae, heterochromatin formation requires $\operatorname{Sir} 2$ p, $\operatorname{Sir} 3 p$, and $\operatorname{Sir} 4$ p, and these Sir proteins create specialized chromatin structures at telomeres and silent mating type loci. Previously, we reported that the SWI/SNF chromatin remodeling enzyme can evict Sir3 from chromatin fibers in vitro, though whether this activity contributes to the role of SWI/SNF as a transcriptional activator at euchromatic loci is unknown. Here, we characterize genetic interactions between the $\operatorname{SIR}$ genes (SIR2, SIR3, and SIR4) and genes encoding subunits of the chromatin remodelers SWI/SNF and INO80C, as well genes encoding the histone deacetylases Hst3 and Hst4. We find that loss of SIR genes partially rescues the growth defects of swi2, ino 80 , and $h s t 3 / h s t 4$ mutants during replication stress conditions. Interestingly, partial suppression of swi2, ino80, and hst 3 hst 4 mutant phenotypes is due to the pseudo-diploid state of sir mutants, but a significant portion is due to more direct functional interactions. Consistent with this view, transcriptional profiling of strains lacking Swi2 or Sir3 identifies a set of genes whose expression in the M/G1 phase of the cell cycle requires SWI/SNF to antagonize the repressive impact of Sir3.


## INTRODUCTION

Eukaryotic genomes are packaged with positively charged histone proteins to form chromatin. Chromatin can be divided into two functional categories: transcriptionally active euchromatin and transcriptionally silent heterochromatin. In budding yeast, heterochromatic structures are formed at each telomere and the two silent mating type loci (HMR and $H M L$ ). The assembly of heterochromatin domains requires the binding of non-histone proteins to the chromatin fiber, and in yeast these are Sir2, Sir3 and Sir4 (Rusche et al. 2003; Rusché et al. 2002). Sir3 is believed to be the structural component of yeast heterochromatin, whereas Sir2 is a histone deacetylase that functions with Sir4 to target Sir proteins to the proper genomic locations. Deletion of SIR genes leads to expression of both a - and $\alpha$-specific genes in haploids, producing a pseudo-diploid state (Haber 1998). Previous studies have found that this pseudo-diploid state alters the DNA damage response, alleviating the genotoxic stress phenotypes of several rad mutants (Schild 1995; Valencia-Burton et al. 2006).

In addition to heterochromatic regions, Sir3 has also been detected by chromatin immunoprecipitation studies at euchromatic locations, although the functional implications are not understood (Radman-Livaja et al. 2011). Immunofluorescence studies of Sir3 have also revealed that Sir3 forms discrete nuclear puncta for most of the cell cycle, except for a diffuse nuclear staining pattern during mitotic stages (Laroche et al. 2000). Overexpression of Sir3 can lead to the expansion of heterochromatin domains and gene-silencing defects within euchromatin (Taddei et al. 2009; Holmes et al. 1997), indicating that aberrant binding of Sir3 to euchromatic sites can be detrimental.

ATP-dependent chromatin remodeling enzymes are a major contributor to the dynamic nature of chromatin. They modify chromatin structure by mobilizing or disrupting nucleosomes in
an ATP-dependent reaction (Clapier and Cairns 2009). The SWI/SNF chromatin remodeling enzyme is a founding member of this group of enzymes (Smith and Peterson 2005), and subunits of SWI/SNF were first identified in yeast genetic studies as global activators of transcription (Peterson et al. 1992; Laurent et al. 1991). For instance, inactivation of the Swi2 ATPase subunit leads to defects in the transcription of many inducible yeast genes, as well the function of many transcriptional activators. Strains harboring mutations in genes encoding SWI/SNF subunits have severe growth defects, are sensitive to DNA damaging or replication stress agents, and show a defect in mitotic exit (Krebs et al. 2000). Similar to other remodeling enzymes, SWI/SNF can use the energy of ATP hydrolysis to mobilize nucleosomes in cis, or evict nucleosomal H2A/H2B dimers as well as entire histone octamers from DNA. Recently, we also found that SWI/SNF has the novel ability to catalyze the displacement of the Sir3 protein from nucleosomal substrates in vitro. This activity is not shared with other remodeling enzymes, and it requires a direct interaction between the Swi2 subunit and Sir3. This activity appears to be important for cells to contend with replication stress (Manning and Peterson 2014).

In this work, we report genetic interactions between the gene encoding the Swi2 subunit of SWI/SNF and genes encoding the Sir2, Sir3, and Sir4 heterochromatin components. Inactivation of Sir3 alleviated the slow growth phenotype of a swi2 $\Delta$ strain, and partially restored resistance to the replication stress agent, hydroxyurea (HU). Deletion of SIR2 or SIR3 also partially suppressed the replication stress phenotypes caused by loss of the INO80C chromatin remodeling complex as well as the loss of the H 3 lysine 56 -specific histone deacetylases Hst3 and Hst4. Interestingly, in some cases partial suppression of genotoxic stress phenotypes were observed in pseudo-diploid cells, suggesting indirect as well as direct impacts of Sir3 loss. To identify potential transcriptional targets for the SWI/SNF-Sir3 antagonism, we characterized the transcriptional profile of swi2A,
$\operatorname{sir} 3 \Delta$, and swi2 $\Delta \operatorname{sir} 3 \Delta$ strains. A parallel analysis was also performed in SIR 3 and $\operatorname{sir} 3 \Delta$ strains where Swi 2 was conditionally depleted from the nucleus by the anchor away method (Haruki et al. 2008). This latter method circumvented transcriptional defects due to the severe growth phenotype of the swi $2 \Delta$ strain, and together identified a common set of genes where SWI/SNF promotes transcription by antagonizing Sir3.

## MATERIALS AND METHODS:

## Yeast growth media and genetic methods

Yeast were cultured using standard procedures (Rege et al. 2015). For tetrad analysis, at least 30 tetrads were dissected for segregation analysis and growth rates noted.

## List of strains

| Name | Genotype |
| :---: | :---: |
| CY1653 | BY4743; MATa/a ;his3 $31 / h i s 3 \Delta 1$; leu2Д0/leu2ム0; lys2Д0/LYS2; MET15/me ura340/ura3 0 0; swi2::KanMX4/SWI2 sir3A:: HPH $^{R} /$ SIR3 |
| CY1618 | MAT (a) segregant from CY1653, clone 15A, sir3A: $: \mathrm{HPH}$ |
| CY1619 | MAT (a) segregant from CY1653, clone 15B, swi2A $:$ (KANMX and sir $3 \Delta: \because H P$ |
| CY1620 | MAT a segregant from CY1653, clone 15C (wild type) |
| CY1621 | MAT a segregant from CY1653, clone 15D, swi2::KANMX |
| CY1809 | Y40345 MATa tor1-1 fpr1::loxP-LEU2-loxP RPL13A-2x FKBP12:loxP (HH |
| CY1810 | Y40362 MATa tor1-1 fpr1::NAT RPL 13A-2x FKB12::TRP1 SNF2-FRB:kan |
| CY1853 | MAT a sir3D::HYGRO ${ }^{\text {R }}$ in CY1809, clone 1 |
| CY1854 | MAT $\alpha$, sir $3 \Delta:: H Y G R O^{R}$ in CY1810, clone 16 |
| CY1953 | MATa sir2د::HIS in CY1885, clone 12 |


| CY1954 | MATa sir2د: $:$ HIS in CY1810, clone 10 |
| :---: | :---: |
| CY1907 |  ura3A0/ura3 0 0; swi2::KanMX4/SWI2 sir2 $2:: H P H^{R} /$ SIR2 |
| CY1908 |  ura3ム0/ura3 0 0; swi2::KanMX4/SWI2 sir4 $4:: H P H^{R} /$ SIR 4 |
| CY1752 | MATa/a CY927 X CY971; sir3D $:$ :HYGRO ${ }^{\text {R }}$, diploid 2 |
| CY2041 | swi24 in W303, spore 21A dissected from CY1752 |
| CY2042 | sir34 in W303, spore 21B dissected from CY1752 |
| CY2043 | WT in W303, spore 21C dissected from CY1752 |
| CY2044 | swi2d sir34 in W303, spore 21D dissected from CY1752 |
| CY2394 | Y40345 MATa tor1-1 fpr1: :loxP-LEU2-loxP RPL13A-2×FKBP12: 1 loxP (H <br>  |
| CY2395 | Y40345 MATa tor1-1 fpr1::loxP-LEU2-loxP RPL13A-2×FKBP12::loxP (H bar1A::HISG HST3-FRB:kanMX6 hst4A::HPH RPB3-FLAG:NAT |
| CY1838 | Y40345 MATa tor1-1 fpr1::loxP-LEU2-loxP RPL13A-2×FKBP12::loxP (H bar14::HISG INO80-FRB:His3MX6 |
| CY2478 | MATa sir3D $\because$ PHL in CY2395 |
| CY2479 | MATa sir2వ: $:$ PHL in CY2395 |
| CY2186 | MATa sir24: $:$ KanMX in CY1838 |
| CY2162 | MATa sir34: $:$ KanMX in CY1838 |
| CY2190 | MATa sir4D: $:$ KanMX in CY1838 |
| CY2254 | ino80::KanMX sir3::Hygro ${ }^{R}$ clone 1D, segregant from CY 2249 |
| CY2252 | ino80::KanMX clone 1B, segregant from CY2249 |
| CY2487 | MATa nej1泬PHL in CY1810 |
| CY2488 | MATa rme1D: $:$ PHL in CY1810 |
| CY2491 | MATa pst20 $:$ PHLL in CY1810 |
| CY2485 |  |
| CY2486 | MATa rme1 $\triangle: \because$ PHL in CY2395 |


| CY2492 | MATa pst $2 \Delta \because:$ PHL in CY2395 |
| :--- | :--- |
| CY2495 | MATa nej1 $\because:$ PHL in CY1838 |
| CY2496 | MATa rme1 $\triangle:$ PHL in CY1838 |
| CY2490 | MATa pst2 $\because:$ PHL in CY1838 |
| CY2495 | MATa nej1 $\because:$ PHL in CY1809 |
| CY2496 | MATa rme1 $\because:$ PHL in CY1809 |
| CY2489 | MATa pst $2 \Delta \because$ PHL in CY2394 |


| Plasmid |  |
| :--- | :--- |
| CP1212 | pAG25; CEN/ARS w/ NAT cassette. Plasmid \#35121 (Addgene) |
| CP1234 | CEN/ARS SIR3 w/ NAT cassette |
| CP1 | YCp50; CEN/ARS w/ URA3 cassette |
| CP1241 | pJR156 CEN/ARS MATa in YCp50 w/ URA3 cassette (Jasper Rine) |
| CP1242 | pJR157s CEN/ARS MAT in YCp50 w/ URA3 cassette (Jasper Rine) |

115 Microarray sample preparation and analysis:

## Chromatin Immunoprecipitation (ChIP)

Yeast strains were grown in rich media with $2 \%$ glucose at $30^{\circ} \mathrm{C}$ and either DMSO or Rapamycin $(8 \mu \mathrm{~g} / \mathrm{ml}$ final concentration) was added for 60 minutes before fixation with $1.2 \%$ formaldehyde. Cells were quenched with 2.5 M glycine, centrifuged, rinsed with cold water and stored at $-80^{\circ} \mathrm{C}$ until chromatin preparation. Chromatin preparation, immunoprecipitation and DNA extraction were performed as described in (Bennett et al. 2013). The anti-Sir3 antibody ( $1 \mu \mathrm{~L}$ for $100 \mu \mathrm{~L}$ chromatin) was used to immunoprecipitate native Sir3. The anti-H3 antibody, ab1791 from Abcam ( $1 \mu \mathrm{~L}$ for $100 \mu \mathrm{~L}$ chromatin) was used to immunoprecipitate histone H3. The SIR3 gene was Cterminally tagged with a FLAG tag and an anti-FLAG antibody used for immunoprecipitation.

117 Yeast strains were grown in rich media with $2 \%$ glucose at $30^{\circ} \mathrm{C}$ in 50 ml cultures, collected at OD $118=0.8$ for RNA preparation and RNA was extracted using the hot phenol method as described previously (Rege et al. 2015). Samples prepared as described in Welch et al. 2007 were hybridized to Affymetrix Yeast 2.0 arrays from four replicates of swi2 2 and swi2 $\operatorname{sir} 3 \Delta$ strains and analyzed by limma analysis in R (Bioconductor package). Yeast strains were grown in rich media with $2 \%$ glucose at $30^{\circ} \mathrm{C}$ to $\mathrm{OD}=0.6$. and either DMSO or Rapamycin ( $8 \mu \mathrm{~g} / \mathrm{ml}$ final concentration) was added for 60 minutes and pelleted for RNA preparation (Rege et al. 2015). One replicate each of the SWI2-FRB, SWI2-FRB sir3A and sir3A arrays and corresponding WT arrays was used. Total RNA was hybridized on Affymetrix Yeast 2.0 arrays and analyzed using a $\log 2$ fold change cutoff. The raw data files have been deposited on the GEO database (\# in process).

## qRT-PCR

Samples for total RNA were prepared and qRT-PCR was performed as described previously in Manning and Peterson 2014.

## Data Availability Statement

All strains made in this study are available upon request from the Peterson lab. The lists of Group 1_KO genes and Group 1_AA genes are given in Table S1 and Table S2, respectively. The list of Group 1 genes common in the KO and AA datasets are given in Table S3. The lists of Group 2_KO genes and Group 2_AA genes are given in Table S4 and Table S5, respectively. RMA normalized data obtained using GeneSpring Affymetrix Software for all the conditions and replicates are
provided in Tables S5 and S6. Raw microarray .CEL files have been deposited in NCBI's GEO database with the accession number (in process).

## RESULTS:

## The slow growth phenotype of swi2 $\Delta$ is partially rescued by $\operatorname{sir} 3 \Delta$

An isogenic set of wildtype, sir $3 \Delta$, swi2 $\Delta$, and swi $2 \Delta \operatorname{sir} 3 \Delta$ strains was created by tetrad dissection from a swi $2 \Delta / S W I 2 \operatorname{sir} 3 \Delta / S I R 3$ heterozygous diploid. Deletion of SIR3 partially suppresses the growth defect of swi $2 \Delta$ on rich media (Figure 1A), suggesting that these loci genetically interact. Importantly, this suppression segregates with markers for the double mutant after tetrad analysis, eliminating the possibility that a nonspecific, background suppressor causes the growth suppression in swi2 $\operatorname{sir} 3 \Delta$ strains (Figure 1A). We also find that the growth defects of swi2 $\Delta$ are suppressed by $\operatorname{sir} 3 \Delta$ in a different strain background (w303; Figure 1B). In addition to slow growth on glucose media, swi $2 \Delta$ mutants are unable to metabolize alternative carbon sources like raffinose, galactose, glycerol, or ethanol (Abrams et al. 1986; Carlson et al. 1981). Inactivation of Sir3 did not facilitate growth of a swi2 $\Delta$ on raffinose, but limited suppression was observed for growth on media containing galactose, ethanol, or glycerol (Figure 1C). SWI2 is also required for resistance to replication stress, induced by hydroxyurea (HU), and a swi $2 \Delta$ shows a delayed growth rate in this condition (Sharma et al. 2003). Interestingly, deletion of SIR3 partially relieves the HU sensitive phenotype of a swi $2 \Delta$ (Figure 1C). Thus, a subset of swi2 ${ }^{\text {( phenotypes are }}$ alleviated by deletion of SIR3.

To completely eliminate the possibility that a background mutation other than the $\operatorname{sir} 3 \Delta$ segregated with, and caused the growth suppression seen in the double mutant, we transformed the swi2 $\Delta \operatorname{sir} 3 \Delta$ with a plasmid containing $\operatorname{SIR} 3$ expressed from its endogenous promoter. As
expected, complementation with a vector plasmid had no impact on growth, while the SIR3 plasmid slowed the growth of the $\operatorname{swi} 2 \Delta \operatorname{sir} 3 \Delta$ strain (Figure 1D). Given that $\operatorname{sir} 3 \Delta$ suppresses the severe growth defects of swi2 $\Delta$ in multiple strain backgrounds, and that this suppression can be reversed when swi $2 \Delta \operatorname{sir} 3 \Delta$ is complemented by a SIR3 plasmid, these data suggest that SWI/SNF antagonizes Sir3 in vivo.

## Absence of SIR2 does not suppress swi2 $\Delta$ growth defects

Given that SIR3 shows negative genetic interactions with SWI2, we asked whether genes that encode other Sir proteins, Sir2 and Sir4, also showed similar genetic interactions. Sir2 is a histone deacetylase (HDAC) that promotes Sir3 binding to nucleosomes by removing the acetyl group on histone H 4 lysine 16. Sir4 forms a complex with Sir2, and it is believed to play a key role in targeting Sir proteins to telomeres and HM loci (Rusché et al. 2002; Thurtle and Rine 2014). Unlike deletion of SIR3, inactivation of Sir2 did not alleviate the slow growth of the swi2A (Figure S1A, B). In contrast, inactivation of Sir4 suppresses the growth defect of a swi2A mutant (Figure S1C, D). Thus, the studies support genetic interactions between SWI2, SIR3, and SIR4, but not SIR2.

## Comparison of swi2 $\Delta$ alleles with conditional depletion of Swi2

As swi2d null mutants are extremely slow growing, we wanted to establish an alternative approach to interrogate the genetic interactions between SIR genes and SWI2. To this end, the anchor away system was used to conditionally deplete Swi2 from the nucleus (Haruki et al. 2008). The parent strain harbors a FK506 binding protein (FKBP12) tag fused to the C-terminus of an anchor protein, RPL13A. RPL13A is a ribosomal protein that is present in high copy numbers in the cell and
transits from the nucleus to the cytoplasm during ribosome assembly, as shown in Figure 2A. In this parent strain, we tagged the endogenous SWI2 locus at the C-terminus with the FKBP12-rapamycin-binding (FRB) domain. Rapamycin induces formation of a ternary complex between the FKBP12 and FRB domains, and thus, rapidly depletes SWI2-FRB from the nucleus (Figure 2A).

We first compared growth rates of SWI2-FRB strains with or without the SIR3 gene using spot assays. In the presence of DMSO solvent, growth rates of all strains are identical on rich media (Figure 2B), indicating that the SWI2-FRB fusion itself does not impair Swi2 function. In the presence of rapamycin, $S W I 2-F R B$ strains show a decrease in growth rate compared to the WT, consistent with nuclear depletion of Swi2 (Figure 2B). However, the SWI2-FRB strains have a milder growth defect compared to the swi2 $\Delta$ (null) mutant (Figure 1), perhaps due to residual Swi2 present in the nucleus. Similar to the swi2A, depletion of SWI2 also causes HU sensitivity, and this phenotype is partially suppressed by deletion of SIR3, suggesting an important link between Swi 2 and Sir3 during replication stress (Figure 2B). Unlike the case with a deletion allele of SWI2, deletion of SIR2 also partially suppressed the sensitivity of the SWI2-FRB strain (+Rap) to HU, but to a lesser extent than deletion of SIR3 (Figure 2B).

Previous work has shown that SWI2 is required for transcriptional activation of the ribonucleotide reductase (RNR) genes in the presence of HU (Sharma et al. 2003). Consistent with this, we see a large reduction of these transcripts in the swi2 (Figure S1E, F). However, unlike the rescue of growth, the lower levels of RNR transcripts was not restored by the sir $3 \Delta$ following depletion of Swi2. This observation suggests that in HU stress, SWI/SNF may antagonize Sir3 independent of transcription, possibly by assisting replication within SIR heterochromatin.

## Deletion of SIR2 or SIR3 suppresses growth defects of ino80 and hst3/hst 4 mutants

To investigate whether the suppression of growth defects by loss of Sir proteins is unique to SWI/SNF or a more common feature among chromatin modifying enzymes, we determined if deletion of SIR2 and SIR3 suppresses the growth defects caused by nuclear depletion of the histone H3 deacetylases, Hst3 and Hst4 (Hst4 $4 / H S T 3-F R B)$, or the chromatin remodeler, INO80C (INO80-FRB). In the presence of rapamycin, both the Hst4 $/ H S T 3-F R B$ and INO80-FRB strains grow similarly to WT, but they are sensitive to stress conditions, such as when media contains camptothecin (CPT) or HU (Figure 2C, D). Strikingly, deletion of either SIR2 or SIR3 suppresses the growth defects caused by rapamycin-dependent depletion of either Ino80 or Hst3/Hst4 in the presence of HU or CPT (Figure 2C, D). Similarly, deletion of SIR3 also partially suppresses an ino804 strain (Figure S2), confirming the results observed with INO80 anchor away. Thus, in addition to SWI2, the SIR genes also show genetic interactions with HST3/HST4, and INO80.

## Genotoxic stress is partially suppressed by MAT heterozygosity

Our genetic studies suggest that SWI/SNF, INO80C, and Hst3/Hst4 antagonize Sir proteins during replication stress. SIR heterochromatin prevents expression of the silent mating type loci, and consequently, loss of Sir proteins leads to expression of diploid-specific genes and suppression of haploid-specific genes (Rine and Herskowitz 1987; Goutte and Johnson 1988; Herskowitz 1989; Dranginis 1990). This pseudo-diploid state, in which both the MATa and MAT $\alpha$ genes are expressed, is termed the MAT heterozygotic state. MAT heterozygosity has been shown to suppress the DNA repair defects of rad mutants (Valencia-Burton et al. 2006). Indeed, both MAT heterozygosity and sir mutants downregulate NHEJ and preferentially use HR for DNA repair (Valencia-Burton et al. 2006). To determine whether loss of Sir proteins suppresses replication
stress phenotypes in a direct manner or an indirect manner due to pseudo-diploid effects, we investigated the impact of $M A T$ heterozygosity. We introduced an episomal copy of MAT $\alpha$ into the $M A T \mathbf{a}$ anchor away strains to generate $M A T a / M A T \alpha$ haploids. Interestingly, MAT heterozygosity partially suppressed the sensitivity of SWI2-FRB (+ Rap) during HU stress (Figure 3A), though the suppression was less than what was observed by deletion of SIR3 (Figure 2B). In contrast, MAT heterozygosity did not suppress sensitivity of SWI2-FRB (+Rap) to CPT (Figure 3A). The sensitivity of the $h s t 4 \Delta / H S T 3-F R B$ and $I N O 80-F R B$ mutants to CPT, and to a lesser extent HU, was also partially suppressed by MAT heterozygosity (Figure 3B, C). Taken together, the results indicate that the replication stress sensitivity of strains depleted for Swi2, Ino80, or Hst3/Hst4 can be partially suppressed by MAT heterozygosity, either by deletion of SIR genes or by expressing the opposite mating type in haploid strains. However, the extent of suppression by the pseudodiploid state is generally less than what is observed for loss of Sir proteins.

Expression of both MATa and MAT $\alpha$ alters the transcriptional profile by down regulating haploid-specific genes and upregulating diploid-specific genes (Herskowitz 1989). Thus, we sought to investigate which of these pathways are important for suppressing replication stress phenotypes. Previous work has shown that deletion of the haploid-specific gene NEJ1, required for non-homologous end joining (NHEJ), suppressed the growth defect of rad55 DNA damage (Valencia-Burton et al. 2006). To determine whether loss of NHEJ also suppresses the phenotypes due to loss of chromatin modifiers, we deleted NEJ1 in the anchor away strains. Deletion of NEJI did not suppress the growth defects of SNF2-FRB, hst4D/HST3-FRB, or INO80$F R B$ mutants when grown on rapamycin in the presence of HU or CPT (Figure S3A). We next tested the impact of two additional haploid-specific genes, RME1 or PST2, which show genetic interactions with RAD55 and RAD51 during growth on CPT stress (Valencia-Burton et al. 2006).

However, deletion of either RME1 or PST2 did not suppress the HU or CPT stress phenotypes of the SWI2-FRB, hst4D/HST3-FRB, or INO80-FRB (+Rap; Figure S3B, C). Thus, the underlying genetic basis for the MAT-dependent, partial suppression of stress phenotypes due to depletion of SWI/SNF, INO80, and Hst3/Hst4 remains unknown, though it does not appear to be due to loss of NHEJ or activation of meiotic transcriptional programs.

## Loss of Sir3 partially suppresses the transcriptional defects due to loss of SWI/SNF

Since loss of Sir3 has a large impact on phenotypes due to loss of Swi2 than MAT heterozygosity, we entertained the possibility that SWI/SNF may directly antagonize transcriptional repression by Sir3 at a subset of genes. To identify such transcriptional targets, we analyzed RNA profiles of isogenic wild type, swi $2 \Delta$, sir $3 \Delta$, and swi $2 \Delta$ sir $3 \Delta$ strains for 5716 ORFs using DNA microarrays. Consistent with published data, we observed that deletion of SIR 3 mis-regulates genes in the mating type cascade, with almost no other changes (Figure 4A middle) (Lenstra et al. 2011). In contrast, SWI2 regulates 203 genes positively ( $\mathrm{FDR}<0.1$ and LFC $<-0.58$ ) and 488 genes negatively (FDR $<0.1$ and LFC $>0.58$ ) (Figure 4A top). Many genes whose expression is known to be dependent on SWI/SNF, such as SER3, YOR222W, and the acid phosphatase genes, were altered as predicted (Figure 4B) (Sudarsanam et al. 2000). However, these SWI/SNF-dependent genes were unaffected by a deletion of SIR 3 (Figure 4B, third column).

To identify genes that are regulated by both SWI2 and SIR3, we first selected genes that changed significantly in the swi2 $\Delta$ compared to wild type (Figure 4B), and we then performed hierarchical clustering and classified various sub-groups of interest. Genes that decrease significantly (LFC $<-0.58$ and FDR $<0.1$ ) in swi2 2 and are restored to nearly wild type levels in the swi2 $\Delta \operatorname{sir} 3 \Delta$ are defined as Group 1_KO (Table S1). The top gene ontology (GO) term
category enriched in Group 1_KO is ribosome biogenesis/ ribosomal protein coding genes. This suggests that these genes require SWI/SNF to antagonize Sir3 to promote transcription. Indeed, prior studies have reported Sir3 binding to many ribosomal protein genes, using a GAL-SIR3 inducible strain (Radman-Livaja et al. 2011). However, mRNA abundance of genes involved in ribosome biogenesis/ ribosomal proteins strongly anti-correlates with cellular growth rate and may confound our results (Airoldi et al. 2009).

To circumvent potential issues due to growth defects, we also analyzed RNA profiles from the anchor away strains. Transcriptional profiling following Swi2 depletion also identified many previously known SWI/SNF-dependent genes, including YOR222W, SER3, and the acid phosphatase genes (Figure 4C). Notably, genes involved in ribosome biogenesis were not identified in the anchor away datasets, consistent with the possibility that expression of these genes are linked to growth rates. To identify genes that might be co-regulated by Swi2 and Sir3, we again selected genes that changed by 1.5 fold or more after depletion of SWI2-FRB, and performed hierarchical clustering to identify subsets that are co-regulated by sir3 3 . Genes that decrease (LFC $<-0.58$ ) in SWI2-FRB and were restored to nearly wild type levels in the SWI2-FRB sir3 3 were defined as Group 1_AA (Figure 4C; Table S2). The top GO term category enriched in Group 1_AA is ion/ carbohydrate transport and primarily reflects the metabolic defects of SWI2 mutants in carbon source utilization. The overlap between the Group1_AA and Group1_KO sets revealed a very select set of 28 genes that decrease following loss of SWI/SNF but are restored to nearly wildtype levels by inactivation of Sir3 (p-value of $8.7 \times 10^{-9}$; Figure 5C; Hypergeometric test). This common subset of genes, consolidated as Group 1, corresponds to GO term categories of 'cell cycle', 'cytokinesis', and 'lipid metabolism' (Table S3). Notably, this set is enriched for genes
expressed at the end of mitosis, which we previously showed to be SWI/SNF-dependent (Krebs, et al. 2000).

Consistent with previous analyses, we also identified genes whose expression was increased by either the deletion of SWI2 or by Swi2 nuclear depletion (Figure S4). Expression of a subset of these genes was restored to nearly wildtype levels by inactivation of Sir3, and these gene sets were designated Group 2_KO ( $\mathrm{n}=488$; LFC $<-0.58$ and $\mathrm{FDR}<0.1$ ) and Group 2_AA (n= 192; LFC $<-0.58$ and FDR $<0.1$ ). (Figure 4B,C and S4A,B; Table S4 and S5). However, overlap of Group 2_KO and Group 2_AA datasets revealed only 11 common genes (Figure S4C), suggesting that the upregulation of genes by loss of SWI/SNF is primarily due to an indirect effect of slow growth (Holstege et al. 1998b; Sudarsanam et al. 2000).

## Analysis of Sir3 binding at Group 1 target genes

The genetic and transcriptome analyses suggest that SWI/SNF antagonizes Sir3 to promote expression of specific genes. One prediction of this model is that Sir3 may accumulate at such target genes in the absence of SWI/SNF. To test the model, we analyzed Sir3 recruitment by chromatin immunoprecipitation (ChIP) in WT and swi $2 \Delta$ mutants arrested in nocodazole. Nocodazole is a microtubule depolymerizing agent that blocks entry into mitosis and thus, cells accumulate at the G2/M border (Jacobs et al. 1988). Sir3 binding was measured using a native antibody to Sir3, as well as an anti-FLAG antibody in a strain expressing a SIR3-FLAG fusion from its endogenous locus. In both cases, ChIP analyses in the wild type strain demonstrated enrichment for Sir3 at the heterochromatic loci, HMR and TELVI-R. In the absence of Swi2, the occupancy of Sir3 is reduced at telomeres, consistent with a redistribution of Sir3 to ectopic loci
(Figure S5). However, we did not observe significant changes in Sir3 enrichment at selected euchromatic target genes (Figure S5B).

## DISCUSSION:

Establishing a separation between euchromatin and heterochromatin domains is crucial for cell function. The mechanisms that might actively exclude heterochromatin proteins from euchromatin domains remain poorly understood. Previously, we found that the SWI/SNF complex can remove the Sir3 heterochromatin protein from chromatin fibers in vitro, and here, we report genetic evidence that supports a role for SWI/SNF in disrupting the ability of Sir heterochromatin proteins to repress euchromatic gene expression. In particular, this activity of SWI/SNF appears crucial for proper expression of genes expressed at the end of mitosis.

## Genetic interactions between chromatin modifiers and SIR genes

Our in vitro studies indicated that the SWI/SNF chromatin remodeling enzyme was uniquely able to evict the Sir3 heterochromatin protein from chromatin fibers (Manning and Peterson 2014; Sinha et al. 2009). Here, we found that deletion of either SIR3 or SIR 4 alleviated the growth defects of swi2d on media containing glucose, ethanol, or HU, but $\operatorname{sir} 3 \Delta$ and $\operatorname{sir} 4 \Delta \operatorname{did}$ not significantly rescue the severe growth defects of $\operatorname{swi} 2 \Delta$ on alternative carbon sources, such as raffinose or galactose. Likewise, inactivation of Sir proteins also alleviated the growth phenotypes of cells that were depleted of Swi2. Interestingly, the genetic interactions between genes encoding Sir proteins and SWI/SNF were not unique to this particular chromatin remodeler. Deletion of SIR2 and SIR3 also suppressed the growth defects of ino 80 and $h s t 3 / h s t 4$ mutants under genotoxic stress (Figure 2C, D), indicating a potential common mechanism for suppressing replication stress.

How does loss of Sir proteins alleviate the phenotypes of mutants that lack chromatin modifying enzymes? One possibility is that suppression is due to indirect effects caused by the pseudo-diploid state of sir mutant cells. Deletion of SIR genes induces the expression the a1- $\alpha 2$ repressor, generating pseudo-diploid cells. Such haploid cells expressing both MAT genes show greater resistance to radiation and are more recombination proficient than cells expressing only MATa or MATa (Heude and Fabre 1993). We found that pseudo-diploid cells partially suppressed the phenotypes of the $I N O 80-F R B$ and $h s t 4 \Delta / H S T 3-F R B$ strains to a similar level as deletion of SIR genes, especially for growth on CPT (Figure 3B,C), indicating the some genetic interactions between INO80C and Hst3/Hst4 with Sir proteins may be largely indirect. In contrast, although the pseudodiploid state partially suppressed the HU stress phenotype of the $S N F 2-F R B$ strain (Figure 3A), the suppression was much less than that observed after deletion of SIR3 (Figure 2B). This suggests there exists both a direct genetic interaction between SWI/SNF and Sir3, consistent with the ability of SWI/SNF to evict Sir3 in vitro (Manning and Peterson 2014), as well as an indirect interaction caused by MAT heterozygosity.

## Transcriptional profiling of swi2 $2 \Delta$ and $s w i 2 \Delta \operatorname{sir} 3 \Delta$ strains

Our transcriptional profiling data suggest that there may be at least two classes of SWI/SNF-dependent genes - those where SWI/SNF antagonizes Sir proteins, and a second group of genes that may require more "canonical" nucleosome remodeling activities. Our transcriptional profiling results are consistent with this view, as we identified many SWI/SNF-dependent genes whose transcriptional defect was not restored by loss of Sir3 and a separate set of genes where SWI/SNF appears to antagonize Sir3. Furthermore, we recently identified and characterized a separation of function allele of SWI2 (swi2A- $\Delta 10 R$ ) that generates a SWI/SNF complex that has
normal levels of nucleosome remodeling activity but lacks the ability to evict Sir3 from chromatin fibers. (Manning and Peterson 2014).

For genes where SWI/SNF appears to antagonize Sir3, our microarray analyses revealed two categories of genes - the first group includes ribosomal biogenesis and ribosomal protein coding genes and the second include genes involved in cytokinesis and cell division. Notably, the first category of genes are likely to be sensitive to growth rate and thus changes in their expression are most likely due to indirect effects (Airoldi et al. 2009) (Figure 5C). In contrast, genes involved in mitotic exit were identified in RNA profiles from both gene deletion strains and from the anchor away system. This suggest that defects in expression of these genes are likely to be independent of the growth defects of the swi2 deletion strain and may be direct targets where SWI/SNF antagonizes Sir3. These data lend mechanistic insight to previous findings that SWI/SNF promotes expression of genes involved in mitotic exit and that $S W I / S N F$ mutants are defective in exiting mitosis (Krebs et al. 2000).

## Genes expressed during mitosis are dependent on SWI/SNF to antagonize Sir3

Why might Sir3 only impact genes that are expressed during mitosis? Cytological studies have shown that Sir3 localizes to discrete foci during the majority of the cell cycle, reflecting its heterochromatic localization (Laroche et al. 2000). In contrast, Sir3 shows a diffuse, nuclear staining pattern during mitosis, consistent with more promiscuous binding to both euchromatic and heterochromatic sites. However, we do not observe increased Sir3 occupancy at selected gene promoter regions by ChIP qPCR analyses in G2/M arrested cells, compared to asynchronous cell populations (Figure S5B), though a diffuse localization of Sir3 may not be detectable by ChIPqPCR. Likewise, we did not detect changes in euchromatic Sir3 occupancy in the absence of

SWI/SNF, though Sir3 levels were decreased from telomeric regions, consistent with previous reports (Dror and Winston 2004; Manning and Peterson 2014) (Figure S5). Currently, we favor a model in which Sir3 delocalizes from heterochromatic sites during mitosis, leading to the binding of Sir3 to euchromatic regions, perhaps facilitated by the deacetylated state of transcribed gene coding regions. Sir3 may bind in a diffuse manner across euchromatic genes, limiting the detection of Sir3 by ChIP analyses. We envision that SWI/SNF action may be required to remove Sir3, facilitating expression of these cell cycle regulated genes. Notably, this role for SWI/SNF would be distinct from the typical nucleosome remodeling activities of SWI/SNF.

## ACKNOWLEDGEMENTS

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## REFERENCES:

Abrams, E., Neigeborn, L., and Carlson, M. (1986). Molecular analysis of SNF2 and SNF5, genes required for expression of glucose-repressible genes in Saccharomyces cerevisiae. Mol. Cell. Biol. 6, 3643-3651.

Airoldi, E.M., Huttenhower, C., Gresham, D., Lu, C., Caudy, A.A., Dunham, M.J., Broach, J.R., Botstein, D., and Troyanskaya, O.G. (2009). Predicting cellular growth from gene expression signatures. PLoS Comput. Biol. 5, e1000257.

Bennett, G., Papamichos-Chronakis, M., and Peterson, C.L. (2013). DNA repair choice defines a common pathway for recruitment of chromatin regulators. Nat. Commun. 4, 2084.

Carlson, M., Osmond, B. C., \& Botstein, D. (1981). Mutants of yeast defective in sucrose utilization. Genetics, 98, 25-40.

Carmen, A.A., Milne, L., and Grunstein, M. (2002). Acetylation of the yeast histone H4 N terminus regulates its binding to heterochromatin protein SIR3. J Biol Chem 277, 4778-4781.

Carrozza, M.J., Li, B., Florens, L., Suganuma, T., Swanson, S.K., Lee, K.K., Shia, W.-J., Anderson, S., Yates, J., Washburn, M.P., et al. (2005). Histone H3 methylation by Set2 directs deacetylation of coding regions by Rpd3S to suppress spurious intragenic transcription. Cell 123, 581-592.

Clapier, C.R., and Cairns, B.R. (2009). The biology of chromatin remodeling complexes. Annu. Rev. Biochem. 78, 273-304.

Dranginis, A. M. (1990). Binding of yeast al and alpha 2 as a heterodimer to the operator DNA of a haploid-specific gene. Nature 347, 682-685.

Dror, V., and Winston, F. (2004). The Swi / Snf Chromatin Remodeling Complex Is Required for Ribosomal DNA and Telomeric Silencing in Saccharomyces cerevisiae. 24, 8227-8235.

Goutte, C., and A. D. Johnson. (1988). al protein alters the DNA binding specificity of alpha 2 repressor. Cell 52, 875-882.

Haber J.E. (1998). Mating-type gene switching in Saccharomyces cerevisiae. Annu Rev Genet. 1998;32:561-99.

Haruki, H., Nishikawa, J., and Laemmli, U.K. (2008). The anchor-away technique: rapid, conditional establishment of yeast mutant phenotypes. Mol. Cell 31, 925-932.

Herskowitz, I. (1989). A regulatory hierarchy for cell specialization in yeast. Nature 342, 749-757.

Heude, M., \& Fabre, F. (1993). a/alpha-control of DNA repair in the yeast Saccharomyces cerevisiae: genetic and physiological aspects. Genetics 133, 489-498.

Holmes, S.G., Rose, A.B., Steuerle, K., Saez, E., Sayegh, S., Lee, Y.M., and Broach, J.R. (1997). Hyperactivation of the silencing proteins, Sir2 and Sir3, causes chromosome loss. Genetics 145, 605-614.

Holstege, F.C., Jennings, E.G., Wyrick, J.J., Lee, T.I., Hengartner, C.J., Green, M.R., Golub, T.R., Lander, E.S., and Young, R.A. (1998a). Dissecting the regulatory circuitry of a eukaryotic genome. Cell 95, 717-728.

Holstege, F.C.., Jennings, E.G., Wyrick, J.J., Lee, T.I., Hengartner, C.J., Green, M.R., Golub, T.R., Lander, E.S., and Young, R.A. (1998b). Dissecting the Regulatory Circuitry of a Eukaryotic Genome. Cell 95, 717-728.

Jacobs, C.W., Adams, A.E., Szaniszlo, P.J., and Pringle, J.R. (1988). Functions of microtubules in
the Saccharomyces cerevisiae cell cycle. J. Cell Biol. 107, 1409-1426.

Keogh, M.-C., Kurdistani, S.K., Morris, S. a, Ahn, S.H., Podolny, V., Collins, S.R., Schuldiner, M., Chin, K., Punna, T., Thompson, N.J., et al. (2005). Cotranscriptional set2 methylation of histone H3 lysine 36 recruits a repressive Rpd3 complex. Cell 123, 593-605.

Krebs, J.E., Fry, C.J., Samuels, M.L., and Peterson, C.L. (2000). Global role for chromatin remodeling enzymes in mitotic gene expression. Cell 102, 587-598.

Laroche, T., Martin, S.G., Tsai-Pflugfelder, M., and Gasser, S.M. (2000). The dynamics of yeast telomeres and silencing proteins through the cell cycle. J. Struct. Biol. 129, 159-174.

Laurent, B. C., Treitel, M. A., \& Carlson, M. (1991). Functional interdependence of the yeast SNF2, SNF5, and SNF6 proteins in transcriptional activation. Proc Natl Acad Sci U S A. 88, 2687-2691.

Lenstra, T.L., Benschop, J.J., Kim, T., Schulze, J.M., Brabers, N. a C.H., Margaritis, T., van de Pasch, L. a L., van Heesch, S. a a C., Brok, M.O., Groot Koerkamp, M.J. a, et al. (2011). The Specificity and Topology of Chromatin Interaction Pathways in Yeast. Mol. Cell 42, 536-549.

Manning, B.J., and Peterson, C.L. (2014). Direct interactions promote eviction of the Sir3 heterochromatin protein by the SWI/SNF chromatin remodeling enzyme. Proc. Natl. Acad. Sci. U. S. A. 111, 17827-17832.

Peterson, C.L. and Herskowitz, I. (1992). Characterization of the yeast SWI1, SWI2, and SWI3 genes, which encode a global activator of transcription. Cell 68, 573-83.

Radman-Livaja, M., Ruben, G., Weiner, A., Friedman, N., Kamakaka, R., and Rando, O.J. (2011). Dynamics of Sir3 spreading in budding yeast: secondary recruitment sites and euchromatic
localization. EMBO J. 30, 1012-1026.

Rege, M., Subramanian, V., Zhu, C., Hsieh, T. H., Weiner, A., Friedman, N., Clauder-Münster, S., Steinmetz, L. M., Rando, O. J., Boyer, L. A., \& Peterson, C. L. (2015). Chromatin Dynamics and the RNA Exosome Function in Concert to Regulate Transcriptional Homeostasis. Cell Rep. 13, 1610-1622.

Richmond, E., and Peterson, C.L. (1996). Functional analysis of the DNA-stimulated ATPase domain of yeast SWI2/SNF2. Nucleic Acids Res 24, 3685-3692.

Rine, J., \& Herskowitz, I. (1987). Four genes responsible for a position effect on expression from HML and HMR in Saccharomyces cerevisiae. Genetics 116, 9-22.

Rusche, L.N., Kirchmaier, A.L., and Rine, J. (2003). The establishment, inheritance, and function of silenced chromatin in Saccharomyces cerevisiae. Annu Rev Biochem 72, 481-516.

Rusché, L.N., Kirchmaier, A.L., and Rine, J. (2002). Ordered nucleation and spreading of silenced chromatin in Saccharomyces cerevisiae. Mol Biol Cell 13, 2207-2222.

Schild, D. (1995). Suppression of a new allele of the yeast RAD52 gene by overexpression of RAD51, mutations in srs2 and ccr4, or mating-type heterozygosity. Genetics 140, 115-127.

Schwabish, M.A., and Struhl, K. (2007). The Swi/Snf complex is important for histone eviction during transcriptional activation and RNA polymerase II elongation in vivo. Mol Cell Biol 27, 6987-6995.

Sharma, V.M., Li, B., and Reese, J.C. (2003). SWI/SNF-dependent chromatin remodeling of RNR3 requires TAF(II)s and the general transcription machinery. Genes Dev. 17, 502-515.

Sinha, M., Watanabe, S., Johnson, A., Moazed, D., and Peterson, C.L. (2009). Recombinational
repair within heterochromatin requires ATP-dependent chromatin remodeling. Cell 138, 11091121.

Smith, C.L., and Peterson, C.L. (2005). ATP-dependent chromatin remodeling. Curr Top Dev Biol 65, 115-148.

Sudarsanam, P., Iyer, V.R., Brown, P.O., and Winston, F. (2000). Whole-genome expression analysis of snf/swi mutants of Saccharomyces cerevisiae. Proc Natl Acad Sci U S A 97, 33643369.

Taddei, A., Van Houwe, G., Nagai, S., Erb, I., van Nimwegen, E., and Gasser, S.M. (2009). The functional importance of telomere clustering: global changes in gene expression result from SIR factor dispersion. Genome Res 19, 611-625.

Thurtle, D.M., and Rine, J. (2014). The molecular topography of silenced chromatin in Saccharomyces cerevisiae. Genes Dev. 28, 245-258.

Valencia-Burton, M., Oki, M., Johnson, J., Seier, T. A., Kamakaka, R., \& Haber, J. E. (2006). Different mating-type-regulated genes affect the DNA repair defects of Saccharomyces RAD51, RAD52 and RAD55 mutants. Genetics 174, 41-55.

Welch, E., Barton ER, Zhuo J, Tomizawa Y, Friesen WJ, Trifillis P, Paushkin S, Patel M, Trotta CR, Hwang S, et al. (2007). PTC124 targets genetic disorders caused by nonsense mutations. Nature 447, 87-91.

Wilson, C.J., Chao, D.M., Imbalzano, A.N., Schnitzler, G.R., Kingston, R.E., and Young, R.A. (1996). RNA polymerase II holoenzyme contains SWI/SNF regulators involved in chromatin remodeling. Cell 84, 235-244.

## FIGURE LEGENDS:

## Figure 1: swi2 growth defects are partially rescued by deletion of SIR3

A) Tetrad dissection plates of the swi $2 \Delta /$ SWI2 $\operatorname{sir} 3 \Delta / S I R 3$ heterozygous diploid on YEPD plates with the corresponding genotypes marked with symbols listed on the left. A single dissected spore yields an isogenic colony, imaged after 10 days. Relative size of each colony is representative of the growth rate.
B) Spot assay on null mutants dissected from the W303 background. Equal cell numbers were spotted in consecutive ten-fold dilutions on agar plates with $2 \%$ glucose as the carbon source and imaged after 3 days.
C) Spot assay was performed as described in B) with different carbon sources. Raffinose and galactose plates also contain $2 \%$ antimycin to prevent respiratory growth.
D) swi2 $\Delta \operatorname{sir} 3 \Delta$ mutants transformed with a plasmid containing either the vector backbone (left) or with a construct expressing Sir3 from its endogenous promoter (right). Spot assays were performed on individual isolates as described in B ).

Figure 2: Absence of Sir2 or Sir3 partially suppresses phenotypes due to depletion of SWI/SNF, Hst3/Hst4, or INO80C
A) Schematic of the Anchor-away system to induce conditional depletion of nuclear proteins. Strains contain C-terminally tagged versions of the nucleo-cytoplasmic shuttling protein (RPL13AFKBP12; green hook) and the SWI2 gene locus (SWI2-FRB; yellow star) (left panel). Addition of Rapamycin (red dot) facilitates formation of a ternary complex between FKBP12 and FRB, rapidly depleting SWI2-FRB from the nucleus (right panel).
B) Wild type, $\operatorname{sir} 2 \Delta$, and $\operatorname{sir} 3 \Delta$ strains with or without the SWI2-FRB tag were spotted on $2 \%$ glucose media containing either DMSO solvent, $8 \mu \mathrm{~g} / \mathrm{ml}$ rapamycin (RAP) in the presence or absence of 0.1 $M$ hydroxyurea $(\mathrm{HU})$ and then grown for 3 days at $30^{\circ} \mathrm{C}$.
C) Spot assays as in B) for $h s t 4 \Delta / H S T 3-F R B$ in the presence of or absence 0.1 M HU and $5 \mu \mathrm{~g} / \mathrm{mL}$ Camptothecin (CPT).
D) Spot assays as in B) for $I N O 80-F R B$ in the presence or absence of 0.1 M HU and $5 \mu \mathrm{~g} / \mathrm{mL}$ CPT.

Figure 3: MAT heterozygosity partially suppresses replication stress phenotypes due to depletion of chromatin regulators
A) $S N F 2$-FRB tag strains containing either empty vector or a vector expressing MATa were spotted on $2 \%$ glucose media containing either DMSO solvent, $8 \mu \mathrm{~g} / \mathrm{ml}$ rapamycin (RAP) in the presence or absence of $5 \mu \mathrm{~g} / \mathrm{mL}$ Camptothecin (CPT) and 0.1 M HU , and then grown for 3 days at $30^{\circ} \mathrm{C}$. Two transformants were tested.
B) Spot assays as in A) for $h s t 4 \Delta / H S T 3-F R B$ expressing MAT $\alpha$ grown in the presence or absence of $5 \mu \mathrm{~g} / \mathrm{mL}$ CPT and 0.1 M HU .
C) Spot assays as in A) for INO80-FRB expressing MATa grown in the presence or absence of 5 $\mu \mathrm{g} / \mathrm{mL} \mathrm{CPT}$ and 0.1 M HU .

Figure 4: Whole-genome microarray analysis of swi2A and SWI2-AA strains.
A) Volcano plots show transcripts that change significantly in the mutant compared to the wild type (WT) highlighted in blue (p.adj $=\mathrm{FDR}<0.1$ and $\log _{2}$ Fold Change $>0.59$ ).
B) Heatmap of normalized RNA abundance for ORFs that are significantly down-regulated ( $\mathrm{n}=167$ ) and up-regulated $(\mathrm{n}=488)$ in the swi2 $\Delta$ arrays compared to WT. Corresponding values for these genes from swi24 sir34 and sir34 arrays compared to WT are also shown. Group 1_KO are defined as significantly down-regulated in the swi2 $\Delta$ and comparatively de-repressed in swi2 $\operatorname{sir} 3 \Delta$, while Group 2_KO are defined as significantly up-regulated in the swi2s and comparatively reduced in swi2 $\operatorname{sir} 3 \Delta$. Examples of ORFs identified in previous studies that do not change in swi2 $\operatorname{sir} 3 \Delta$ compared to swi2 $\Delta(> \pm 1.5$ fold) are listed along the right.
C) Heatmap of normalized RNA abundance for genes that are down-regulated ( $\mathrm{n}=264$ ) and upregulated ( $\mathrm{n}=193$ ) in the SWI2-FRB compared to WT in the presence of $8 \mu \mathrm{~g} / \mathrm{ml}$ of rapamycin (RAP). Corresponding values for these genes from SWI2-FRB sir34 and sir34 arrays compared to WT are also shown. 'Group $1 \_$AA' and 'Group 2_AA' are defined essentially as described in B). Examples of ORFs identified in previous studies that do not change in swi $2 \Delta \operatorname{sir} 3 \Delta$ compared to swi2 $\Delta(> \pm 1.5$ fold $)$ are listed along the right.

Figure 5: M/G1 expressed genes are regulated by SWI/SNF in a Sir3 dependent manner in both the SWI2 anchor-away and swi2 $\Delta$ strains
A) Heatmap of normalized RNA abundance for Group1_AA ORFs $(\mathrm{n}=263)$ in the SWI2-FRB, SWI2-FRB $\operatorname{sir} 3 \Delta$ and $\operatorname{sir} 3 \Delta$ arrays compared to WT in the presence of $8 \mu \mathrm{~g} / \mathrm{ml}$ of rapamycin (RAP) after hierarchical clustering.
B) Heatmap of normalized RNA abundance for Group1_KO ORFs $(\mathrm{n}=176)$ in the swi24, swi24 $\operatorname{sir} 34$ and $\operatorname{sir} 34$ arrays compared to WT after hierarchical clustering.
C) Venn diagram depicting the overlap of genes from Group 1_AA and Group 1_KO. GO terms specific and common to the knockout (KO) and anchor-away (AA) datasets are shown.
D) RT-qPCR analysis of select Group 1 genes identified from both the knockout and anchor-away datasets sets.

Figure S1: Gene expression and genetic interactions of SIR3, SIR2, and SIR4 with SWI2.
A, B) Absence of SIR2 does not suppress growth defects of swi2A.
C, D) Absence of SIR4 suppresses the growth defects of swi2A.
E) Absence of SIR3 does not impact $R N R$ gene expression and genetic interactions.

Figure S2: ino80 growth defects are partially rescued by deletion of SIR3
A) Tetrad dissection plates of the $i n o 80 \Delta / I N O 80 \operatorname{sir} 3 \Delta / S I R 3$ heterozygous diploid on YEPD plates with the corresponding genotypes marked with symbols listed on the right. A single dissected spore yields an isogenic colony, imaged after 10 days. Relative size of each colony is representative of the growth rate.
B) Spot assay on null mutants dissected from the W303 background. Equal cell numbers were spotted in consecutive ten-fold dilutions on agar plates with $2 \%$ glucose as the carbon source in the presence or absence of $0.05 \mathrm{M} \mathrm{HU}, 0.1 \mathrm{M} \mathrm{HU}, 0.01 \%$ Methyl methanesulfonate (MMS), $0.03 \%$ MMS, $30 \mu \mathrm{~g} / \mathrm{mL}$ CPT, $90 \mu \mathrm{~g} / \mathrm{mL}$ CPT, $2.5 \mu \mathrm{~g} / \mathrm{mL}$ phleomycin, or $5 \mu \mathrm{~g} / \mathrm{mL}$ phleomycin and imaged after 3 days.

Figure S3: Growth defects due to depletion of chromatin regulators are not suppressed by deletion of haploid-specific genes $N E J 1$, RME1, or PST2
A) Wild type and $n e j 1 \Delta$ strains with or without the $S N F 2-F R B$, hst $4 \Delta / H S T 3-F R B$, or $I N O 80-F R B$ were spotted ( $1 / 10$ dilutions) on $2 \%$ glucose media containing either DMSO solvent, $8 \mu \mathrm{~g} / \mathrm{ml}$ rapamycin (RAP) in the presence or absence of 0.1 M HU and $5 \mu \mathrm{~g} / \mathrm{mL}$ Camptothecin (CPT). Cells were then grown for 3 days at $30^{\circ} \mathrm{C}$.
B) Spot assays as in A) for rmeld.
C) Spot assay as in A) for pst $2 \Delta$.

Figure S4: Overlap of Group 2 genes (those repressed by SWI2) between swi24 and SWI2 anchor-away strains
A) Group 2_KO (n=192) heatmap with strains compared to WT anchor away strain. B) Group 2_AA $(\mathrm{n}=488)$ heatmap with null mutants compared to WT.
C) Overlap of the number of genes from Group 2_AA and Group 2_KO and the corresponding GO term categories.

Figure S5: ChIP analysis of Sir3 occupancy
A) Chromatin immunoprecipitation (ChIP) for native Sir3 in nocodazole-arrested (G2/M boundary) cells at two heterochromatic loci in WT, sir $3 \Delta$ and swi2 $2 \Delta$ cells.
B) ChIP for SIR3-FLAG in nocodazole-arrested (G2/M boundary) cells at promoters of SWI2 dependent genes in WT and swi2 $\Delta$ cells.

## SUPPLEMENTARY TABLES

Table S1: List of Group 1_KO genes
Table S2: List of Group 1_AA genes
Table S3: List of Group 1 genes common in the KO and AA datasets
Table S4: List of Group 2_KO genes
Table S5: List of Group 2_AA genes
Table S6: Complete table of RMA values from the KO datasets for all genes
Table S7: Complete table of RMA values from the AA datasets for all genes


## C



Figure 2
A

## B




D


Figure 3
A

|  | 0．001\％DMSO | $8 \mu \mathrm{~g} / \mathrm{mL}$ Rap | $\begin{gathered} \text { DMSO + } \\ 5 \mu \mathrm{~g} / \mathrm{mL} \text { CPT } \end{gathered}$ | $\begin{gathered} \text { Rap + } \\ 5 \mu \mathrm{~g} / \mathrm{mL} \text { CPT } \end{gathered}$ | $\begin{aligned} & \text { DMSO + } \\ & \text { 0.1 M HU } \end{aligned}$ | $\begin{gathered} \text { Rap + } \\ 0.1 \mathrm{M} \mathrm{HU} \end{gathered}$ |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
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|  |  |  |  |  |  |  |
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| $S W I 2-F R B+$ MATa－B | 0 筌安 | ○速 | 人00 |  | 10需 | － |

B

|  | 0．001\％DMSO | $8 \mu \mathrm{~g} / \mathrm{mL}$ Rap |
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| hst4D／HST3－FRB＋vector－A |  | （1）政 |
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| hst4 $/$ HST3－FRB＋MAT $\alpha-\mathrm{B}$ | － | 考 |


Rap＋
0．1 M HU



Figure 4


Figure 5
Rege et al
A


B


D


A
$\frac{\text { swi2 } \Delta}{\text { SWI2 }} \frac{\operatorname{sir} 2 \Delta}{\text { SIR2 }}$


C
$\frac{\operatorname{swi} 2 \Delta}{\text { SWI2 }} \frac{\operatorname{sir} 4 \Delta}{\text { SIR } 4}$


E

RNR3


RNR2


## A

Tetrad Dissection: ino80 $\operatorname{sir} 3 \Delta$


B


Figure S3
Rege et al

A

|  | DMSO | Rap | DMSO＋ $5 \mu \mathrm{~g} / \mathrm{mL}$ CPT | Rap＋ $5 \mu \mathrm{~g} / \mathrm{mL}$ CPT | $\begin{aligned} & \text { DMSO + } \\ & 0.1 \mathrm{M} \mathrm{HU} \end{aligned}$ | $\begin{gathered} \text { Rap + } \\ 0.1 \mathrm{M} \mathrm{HU} \end{gathered}$ |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| wT | O－䜌 $\because$ | 000 和 | O00 \％ | OOO笅 4 | O00 者 | $\bigcirc$ |
| WT nej14 | 000蝺苭 | 000家 ${ }^{\text {c }}$ | 000涨 | － | 00 \％ | 0 |
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| SWI2－FRB nejid | －00䀦： | 00 | －00 事， | －${ }^{3}$ | 000 |  |
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| IN080－FRB nej14 | OOO \％ | $00{ }^{\text {笭 }}$ | 000等 | $\bigcirc$ | 000 \％ | － |

B


Rap＋
$5 \mu \mathrm{~g} / \mathrm{mL}$ CPT


DMSO＋
0．1 M HU


Rap＋ 0．1 M HU


C



 mind swi2D = swi2 null mutant; sir3D = sir3 null mutant, WT = Wildtype

| YORF | NAME | swi2d-WT | 2D | sir3D-WT |
| :---: | :---: | :---: | :---: | :---: |
| YOR107W | YOR107W | -1.736286 | -1.249234 | 0.802684 |
| YORO49C | YORO49C | -1.799513 | -1.528723 | 1.077021 |
| YORO32C | YORO32C | -1.190089 | -1.02068 | 1.545266 |
| YJL116C | YJL116C | -2.189504 | -0.596606 | 2.268662 |
| YIL169C | YIL169C | -0.686039 | -0.35075 | 2.457973 |
| YIL169C | YIL169C | -0.866575 | -0.786447 | 2.38699 |
| YOL155C | YOL155C | -0.771853 | -0.763125 | 2.132386 |
| YLL012W | YLLO12W | -1.095636 | -0.664814 | 0.251892 |
| YMR182W-A | YMR182W | -1.19808 | -0.661026 | 0.069504 |
| YER072W | YeR072W | -1.036205 | -0.931685 | -0.004959 |
| YBR238C | YBR238C | -1.113228 | -1.032084 | 0.050758 |
| YGR079W | YGR079W | -1.056979 | -0.935159 | 0.135865 |
| YNL141W | YNL141W | -1.263049 | -0.889395 | 0.046825 |
| YKL185W | YKL185W | -0.871451 | -0.845494 | -0.158825 |
| YHR049W | YHR049W | -0.982429 | -0.822014 | -0.31239 |
| YER062C | YER062C | -0.938457 | -0.793679 | -0.232057 |
| YOL002C | YOL002C | -0.883949 | -0.611531 | -0.186142 |
| YOR101W | YOR101W | -0.89683 | -0.624431 | -0.017271 |
| YLR008C | YLR008C | -0.812653 | -0.574562 | -0.044313 |
| YFL004W | YFL004W | -0.986432 | -0.698759 | 0.025827 |
| YBR085W | YBR085W | -1.020654 | -0.603666 | -0.067401 |
| YDR384C | YDR384C | -0.95195 | -0.607941 | 0.163866 |
| YJR060W | YJR060W | -0.792775 | -0.641355 | 0.115003 |
| YPR054W | YPR054W | -0.786343 | -0.572491 | 0.119735 |
| YLR300W | YLR300W | -0.593589 | -0.854639 | 0.03546 |
| YERO44C | YER044C | -0.76323 | -0.995545 | 0.009447 |
| YPL092W | YPL092W | -0.784119 | -0.84839 | 0.077845 |
| YMLO75C | YML075C | -0.66478 | -0.742785 | 0.31812 |
| YPL066W | YPL066W | -0.538909 | -0.641721 | 0.178508 |
| YMR030W-A | YMR030W | -0.616896 | -0.454838 | 0.084452 |
| YJL117W | YJL117W | -0.600328 | -0.511951 | 0.060395 |
| YMR032W | YMR032W | -0.641548 | -0.584348 | 0.017399 |
| YER056C | YER056C | -0.658738 | -0.60332 | -0.057556 |
| YHRO22C | YHRO22C | -0.730672 | -0.465317 | 0.488107 |
| YNR009W | YnRoo9w | -0.639524 | -0.539942 | 0.276325 |
| YPL068C | YPL068C | -0.639863 | -0.390321 | 0.311581 |
| YDR044W | YDR044W | -0.787838 | -0.718158 | 0.576189 |
| YPL057C | YPL057C | -1.304416 | -0.453506 | -0.09573 |
| YNL327W | YNL327W | -1.419537 | -0.456658 | -0.035435 |
| YGR272C | YGR272C | -1.318424 | -0.351872 | -0.051133 |
| YJR048W | YJR048W | -1.257396 | -0.326339 | 0.014971 |
| YoL019W | YoL019W | -1.288769 | -0.387384 | 0.11906 |
| YKL099C | YKL099C | -1.150759 | -0.220279 | 0.07291 |
| YHR066W | YHR066W | -1.304707 | -0.185313 | 0.03815 |
| YORO95C | YOR095C | -0.997563 | -0.35532 | 0.050813 |
| Yalo59w | Yalo59w | -0.994499 | -0.398791 | 0.0139 |


| YIL158W | YIL158W | $-1.038477$ | $\begin{aligned} & \text { the authotitun } \\ & -0.298129 \end{aligned}$ | $0.012811$ |
| :---: | :---: | :---: | :---: | :---: |
| YMR239C | YMR239C | -1.079968 | -0.323804 | 0.015705 |
| YDR184C | YDR184C | -1.117233 | -0.405462 | 0.066926 |
| YPL146C | YPL146C | -1.097313 | -0.341373 | 0.089128 |
| YLR073C | YLR073C | -1.124565 | -0.367879 | -0.154846 |
| YHR052W | YHR052W | -1.096203 | -0.388025 | -0.038756 |
| YMR015C | YMRO15C | -1.192328 | -0.529732 | -0.121981 |
| Yelo40w | YEL040W | -1.140605 | -0.537784 | -0.084598 |
| YNLO24C | YNLO24C | -1.124738 | -0.668689 | -0.176781 |
| YOR072W-B | YOR072W. | -1.179767 | -0.501843 | -0.313012 |
| YMR011W | YmR011w | -1.047188 | -0.494915 | -0.209743 |
| YDL179W | YDL179W | -1.4164 | -0.376551 | -0.21954 |
| YGR271C-A | YGR271C- | -1.367881 | -0.301543 | -0.243718 |
| YER124C | YER124C | -1.394713 | -0.365854 | -0.539364 |
| YPL061W | YPL061W | -1.293322 | -0.005912 | -0.610692 |
| YBLO28C | YBLO28C | -1.025202 | -0.206871 | -0.053283 |
| YGL028C | YGL028C | -0.98148 | -0.264637 | -0.118008 |
| YCR020W-B | YCRO20W- | -1.018662 | -0.087625 | -0.049605 |
| YJL011C | YJL011C | -0.792154 | -0.010188 | -0.011872 |
| YPL158C | YPL158C | -0.814284 | 0.01076 | -0.092527 |
| YHR196W | YHR196W | -0.863989 | -0.165838 | -0.124069 |
| YLR287C | YLR287C | -0.782722 | -0.099197 | -0.197116 |
| Yol020w | YoL020w | -0.739805 | -0.203711 | -0.071481 |
| YOR159C | YOR159C | -0.673594 | -0.138845 | -0.077676 |
| YPL227C | YPL227C | -0.680673 | -0.181158 | -0.113845 |
| YLR264W | YLR264W | -0.649254 | -0.204616 | -0.20628 |
| YPL163C | YPL163C | -0.819237 | -0.39144 | -0.165403 |
| YCR043C | YCR043C | -0.828681 | -0.340857 | -0.196241 |
| YBL055C | YBL055C | -0.690262 | -0.333605 | -0.148308 |
| YKL164C | YKL164C | -0.886037 | -0.389714 | -0.091537 |
| YLR285C-A | YLR285C-A | -0.812289 | -0.440132 | -0.07874 |
| YKR061W | YKR061W | -0.855789 | -0.461683 | 0.016426 |
| YNLO75W | YNLO75W | -0.873769 | -0.303583 | 0.06103 |
| YOR119C | YOR119C | -0.88207 | -0.345121 | 0.01433 |
| YOR004W | YOR004W | -0.878897 | -0.356542 | 0.055945 |
| YGR081C | YGR081C | -0.886528 | -0.245544 | -0.064313 |
| YDL063C | YDL063C | -0.8893 | -0.297907 | -0.073846 |
| YML043C | YML043C | -0.904041 | -0.215257 | -0.006759 |
| YNL300W | YNL300W | -0.767885 | -0.357339 | 0.057677 |
| YPL165C | YPL165C | -0.804397 | -0.320086 | 0.053357 |
| YOL144W | YOL144W | -0.742002 | -0.28505 | 0.092757 |
| YML080W | YML080W | -0.808155 | -0.343047 | -0.05663 |
| YNLO34W | YNLO34W | -0.798485 | -0.305995 | -0.046402 |
| YNL119W | YNL119W | -0.738471 | -0.290834 | -0.06127 |
| YDR399W | YDR399W | -0.730769 | -0.433201 | -0.034015 |
| YDLO42C | YDLO42C | -0.852518 | -0.28447 | 0.312551 |
| YJL122W | YJL122W | -0.801952 | -0.352235 | 0.287684 |
| YGR041W | YGR041W | -0.81707 | -0.416311 | 0.221974 |
| YLRO | YiR063W | -0.785169 | -0.351531 | 0.189522 |




|  |  |  |  |  |
| :--- | :--- | :--- | ---: | :--- |
| YHR094C | YHR wa94C | -1.207275 | 0.243913 | 0.274392 |
| YNR067C | YNR067C | -1.430648 | 0.257047 | 0.118234 |
| YDR222W | YDR222W | -1.421418 | 0.408224 | 0.079002 |
| YJR070C | YJR070C | -0.569932 | -0.180888 | 0.142184 |
| YLR409C | YLR409C | -0.618153 | -0.238689 | 0.185317 |
| YLR221C | YLR221C | -0.612013 | -0.171469 | 0.213431 |
| YOR340C | YOR340C | -0.615029 | -0.141354 | 0.225569 |
| YKL166C | YKL166C | -0.69773 | -0.1267 | 0.148319 |
| YJL069C | YJL069C | -0.678246 | -0.180436 | 0.173403 |
| YNL175C | YNL175C | -0.665957 | -0.110683 | 0.207458 |
| YLR099C | YLR099C | -0.403286 | -0.129096 | 0.199719 |
| YDR398W | YDR398W | -0.530144 | -0.079465 | 0.183036 |
| YPL069C | YPL069C | -0.607505 | 0.072517 | 0.178488 |
| YOL007C | YOL007C | -0.626065 | -0.02273 | 0.183157 |
| YOR091W | YOR091W | -0.6291 | -0.014712 | 0.163624 |
| YPR143W | YPR143W | -0.62508 | 0.010663 | 0.175941 |
| YDR087C | YDR087C | -0.654831 | -0.003588 | 0.136556 |
| YIL127C | YIL127C | -0.779948 | -0.014545 | 0.186459 |
| YDR021W | YDR021W | -0.740934 | 0.048523 | 0.200147 |
| YER082C | YER082C | -0.706368 | -0.012994 | 0.202725 |
| YOL010W | YOL010W | -0.638388 | 0.015929 | 0.237123 |
| YDR365C | YDR365C | -0.709428 | 0.021993 | 0.278121 |
| YAL025C | YALO25C | -0.635748 | -0.014059 | 0.325016 |
| YIL104C | YIL104C | -0.628021 | -0.131091 | 0.288274 |
| YBR247C | YBR247C | -0.694492 | -0.127991 | 0.286097 |
| YPL043W | YPL043W | -0.696074 | -0.090589 | 0.270935 |
| YBR267W | YBR267W | -0.668156 | -0.086142 | 0.323763 |
| YBR271W | YBR271W | -0.561659 | -0.021174 | 0.463252 |
| YER028C | YER028C | -0.63341 | 0.196135 | 0.762258 |

 (he author/funder. All rights reserved. No reuse allowed without permission 1810 = SWI2 Anchor away; 1854= SWI2 Anchor away sir3 null mutant; 1853 = sir3 null in Anchor away background; 1809 = Wildtype Anchor away background. _R = with Rapamycin

| YORF | NAME | 1810_R1- | 1854_R1-1809 | 33_R-1809_R |
| :---: | :---: | :---: | :---: | :---: |
| YLR154W-E | null | -1.555192 | -0.351773 | -1.73793 |
| YHR137W | ARO9 | -1.964415 | -0.29048 | -1.25603 |
| YDL037C | BSC1 | -2.301298 | 0.054224 | -1.61956 |
| YFRO32C | RRT5 | -2.357541 | -1.169374 | -1.62064 |
| YDR380W | ARO10 | -2.408232 | -1.078401 | -1.12321 |
| YDR384C | ATO3 | -2.591182 | -1.606145 | -1.25726 |
| YJL012C | VTC4 | -1.730655 | -1.150325 | -0.70305 |
| YPL279C | FEX2 | -1.909143 | -0.916844 | -0.62966 |
| YOR390W | FEX1 | -1.718305 | -0.730397 | -0.87149 |
| YMR189W | GCV2 | -1.884147 | -0.76806 | -0.89462 |
| YJL047C-A | null | -1.877745 | -0.735646 | -1.1494 |
| YNR044W | AGA1 | -1.889627 | -0.851457 | -1.22788 |
| YNL197C | WHI3 | -2.013228 | -0.787274 | -1.16914 |
| YLR342W-A | null | -2.13461 | -1.120897 | -0.62122 |
| YGR041W | BUD9 | -2.429062 | -1.035065 | -0.73259 |
| YKL043W | PHD1 | -2.449638 | -1.059257 | -0.481 |
| YJR147W | HMS2 | -2.521508 | -0.68129 | -0.66571 |
| YKR050W | TRK2 | -1.564904 | -0.905273 | -0.28193 |
| YOR034C | AKR2 | -1.47363 | -0.878702 | -0.31952 |
| YLL066W-B | null | -1.63241 | -0.823486 | -0.21775 |
| YGR233C | PHO81 | -1.485611 | -0.980128 | -0.03703 |
| YDLO42C | SIR2 | -1.420121 | -0.998536 | -0.09364 |
| YKR104W | null | -1.229861 | -0.567416 | 0.079281 |
| YOR066W | MSA1 | -1.373663 | -0.658872 | -0.06609 |
| YFL021W | GAT1 | -1.39381 | -0.739817 | 0.070213 |
| YOL020W | TAT2 | -1.611607 | -0.635395 | -0.02211 |
| YLR285C-A | null | -1.578821 | -0.490248 | 0.013808 |
| YBR296C | PHO89 | -1.466358 | -0.626254 | 0.239036 |
| YGR143W | SKN1 | -1.472033 | -0.868274 | 0.301284 |
| YBR196C-A | null | -1.686044 | -1.024496 | 0.365435 |
| YGR068C | ART5 | -1.308685 | -1.367768 | -0.25619 |
| YPR009W | SUT2 | -1.199259 | -1.578666 | -0.1354 |
| YPL066W | RGL1 | -1.00754 | -1.297962 | -0.02652 |
| YLR278C | null | -1.039162 | -1.355646 | 0.097341 |
| YBR291C | CTP1 | -1.350511 | -1.317544 | 0.568544 |
| YBLO42C | FUI1 | -1.223819 | -0.91923 | 0.308592 |
| YOR342C | null | -1.000736 | -1.138968 | 0.459689 |
| YPR106W | ISR1 | -1.79978 | -0.483187 | -0.2268 |
| YOR084W | LPX1 | -1.799683 | -0.689908 | -0.43065 |
| YOR137C | SIA1 | -1.75522 | -0.752846 | -0.40568 |
| YIR019C | FLO11 | -1.986636 | -0.540304 | -0.32771 |
| YMR266W | RSN1 | -2.049499 | -0.570817 | -0.53056 |
| YDR072C | IPT1 | -1.593271 | 0.019658 | -0.28354 |
| YNR067C | DSE4 | -1.660394 | -0.136176 | -0.36397 |
| YGLO28C | SCW11 | -1.830712 | -0.060328 | -0.32761 |



|  |  |  |  it ${ }_{-0.862954}^{-0.3361}$ |  |
| :---: | :---: | :---: | :---: | :---: |
| YJL193W | null | -1.368008 |  |  |
| YDR143C | SAN1 | -1.293169 | -0.8678 | -0.39477 |
| YPR054W | SMK1 | -1.248613 | -1.072935 | -0.34598 |
| YNL291C | MID1 | -1.077109 | -0.769886 | -0.70305 |
| YDR538W | PAD1 | -1.040836 | -0.710045 | -0.72063 |
| YLR459W | GAB1 | -1.0176 | -0.644804 | -0.73487 |
| YDR404C | RPB7 | -1.108425 | -0.793386 | -0.61565 |
| YHR163W | SOL3 | -1.082184 | -0.831005 | -0.57453 |
| YOL052C | SPE2 | -1.04761 | -0.873968 | -0.6079 |
| YPR121W | THI22 | -1.021577 | -0.693926 | -0.36949 |
| YHR086W-A | null | -1.059386 | -0.784566 | -0.40747 |
| YNR055C | HOL1 | -1.113354 | -0.745037 | -0.50927 |
| YOR321W | PMT3 | -1.080195 | -0.7333 | -0.57796 |
| YKL078W | DHR2 | -1.047444 | -0.702301 | -0.58379 |
| YPL162C | null | -1.091111 | -0.657734 | -0.54618 |
| YBR086C | IST2 | -1.022796 | -0.622193 | -0.57988 |
| YMR274C | RCE1 | -1.413554 | -0.919902 | -0.55519 |
| YCR065W | HCM1 | -1.285848 | -0.962969 | -0.67737 |
| YEL065W | SIT1 | -1.326927 | -0.783409 | -0.60061 |
| YHR086W | NAM8 | -1.318066 | -0.818393 | -0.61668 |
| YGR281W | YOR1 | -1.281143 | -0.81167 | -0.52579 |
| YDL179W | PCL9 | -1.268798 | -0.745332 | -0.50394 |
| YLR141W | RRN5 | -1.303257 | -0.781836 | -0.77922 |
| YGR038W | ORM1 | -1.246203 | -0.704735 | -0.74587 |
| YLL031C | GPI13 | -1.30253 | -0.643208 | -0.68088 |
| YBR175W | SWD3 | -1.260419 | -1.254828 | -0.86015 |
| YER110C | KAP123 | -1.067729 | -1.093829 | -0.8948 |
| YGR131W | FHN1 | -1.038851 | -1.171217 | -0.9338 |
| YBR074W | PFF1 | -1.063322 | -1.206995 | -0.77965 |
| YMR277W | FCP1 | -1.041506 | -0.97864 | -0.84932 |
| YPR052C | NHP6A | -1.022073 | -0.965162 | -0.68902 |
| YLR380W | CSR1 | -1.085019 | -0.869708 | -0.74981 |
| YNL238W | KEX2 | -1.107759 | -0.93974 | -0.74767 |
| YPR128C | ANT1 | -1.14267 | -0.956547 | -0.6306 |
| YPL018W | CTF19 | -1.096172 | -0.982328 | -0.61275 |
| YCL002C | null | -1.095239 | -1.116041 | -0.62248 |
| YOR129C | AFI1 | -1.366272 | -1.293772 | -0.5899 |
| YNLO80C | EOS1 | -1.293655 | -1.163043 | -0.53001 |
| YOR378W | AMF1 | -1.008824 | -0.401119 | -1.10451 |
| YGR146C-A | null | -1.037179 | -0.253796 | -1.17931 |
| YBL112C | null | -1.087553 | -0.294808 | -0.86761 |
| Yelootw | MIT1 | -1.450384 | -0.175074 | -1.11328 |
| YBL032W | HEK2 | -1.428298 | -0.549564 | -1.20662 |
| YMR055C | BUB2 | -1.32715 | -0.832963 | -0.8408 |
| YDL093W | PMT5 | -1.285507 | -0.896378 | -0.84831 |
| YLR381W | CTF3 | -1.309061 | -0.870505 | -0.88433 |
| YNR060W | FRE4 | -1.404624 | -0.994241 | -0.99664 |
| YJR124C | null | -1.421199 | -0.870187 | -0.82313 |
| YPL128C | TBF1 | -1.46835 | -0.971687 | -0.79808 |



| YDL160C | DHH1 | -1.46054 | -0.712223 | 49 |
| :---: | :---: | :---: | :---: | :---: |
| YDR044W | HEM13 | -1.482312 | -0.685862 | -0.96316 |
| YDR414C | ERD1 | -1.115155 | -0.567869 | -1.01918 |
| YOR307C | SLY41 | -1.176199 | -0.6677 | -0.94427 |
| YELO42W | GDA1 | -1.208786 | -0.640769 | -1.01143 |
| YNL283C | WSC2 | -1.176128 | -0.87668 | -0.95097 |
| YOR067C | ALG8 | -1.246612 | -0.771258 | -1.00481 |
| YHR115C | DMA1 | -1.122361 | -0.725427 | -0.90375 |
| YDL048C | STP4 | -1.213949 | 1.304484 | 1.386136 |
| YCL068C | null | -1.523301 | 1.657408 | 1.603099 |
| YFR022W | ROG3 | -1.101124 | 1.820295 | 2.165995 |
| YCR108C | null | -1.06561 | 1.33408 | 2.05838 |
| YPL165C | SET6 | -2.289622 | 1.097893 | 2.20319 |
| YMR182C | RGM1 | -1.899901 | 1.352962 | 1.640581 |
| YDR247W | VHS1 | -1.800171 | 1.391117 | 1.98415 |
| YPL014W | null | -1.910601 | 1.698177 | 2.742215 |
| Yero28C | MIG3 | -1.415682 | 1.989151 | 0.358269 |
| Yol011W | PLB3 | -1.942625 | 0.632071 | 1.215531 |
| YER188C-A | null | -2.006381 | 0.225677 | 0.750596 |
| YER088C | DOT6 | -1.230253 | 0.578579 | 0.870996 |
| YLR120C | YPS1 | -1.208372 | 0.519998 | 0.838073 |
| YBR298C | MAL31 | -1.420194 | 0.582458 | 0.997994 |
| YKL220C | FRE2 | -1.163377 | 0.038322 | 0.585177 |
| YOL136C | PFK27 | -1.330024 | 0.122457 | 0.50754 |
| YBL111C | null | -1.481711 | 0.309395 | 0.461409 |
| YHLO40C | ARN1 | -1.378605 | 0.368258 | 0.793778 |
| YEL063C | CAN1 | -1.266327 | 0.260261 | 0.646736 |
| YOR273C | TPO4 | -1.594325 | 0.143492 | 0.763655 |
| YDR534C | FIT1 | -1.637009 | 0.208779 | 0.328522 |
| YOR381W | FRE3 | -1.59899 | 0.290977 | 0.265652 |
| YCR089W | FIG2 | -1.756856 | 0.166196 | 0.177603 |
| YPL057C | SUR1 | -1.782771 | 0.123331 | 0.541527 |
| YALO40C | CLN3 | -1.861156 | 0.015472 | 0.404578 |
| YLR152C | null | -1.285577 | -0.35867 | 0.317725 |
| YALO60W | BDH1 | -1.328006 | -0.452297 | 0.269904 |
| YBR021W | FUR4 | -1.114172 | -0.330952 | 0.547602 |
| YKL029C | MAE1 | -1.253982 | -0.332381 | 0.555527 |
| YMR272C | SCS7 | -1.212712 | -0.223203 | 0.405308 |
| YOL059W | GPD2 | -1.007211 | -0.056624 | 0.831813 |
| YGL256W | ADH4 | -1.093417 | -0.305503 | 0.710373 |
| YLL066W-B | null | -1.952956 | -0.33443 | 0.509491 |
| YaRO10C | null | -1.799371 | -0.406109 | 0.645434 |
| YHLO16C | DUR3 | -1.741483 | -0.492125 | 0.35648 |
| YBR196C-B | null | -1.369806 | -0.58939 | 0.657573 |
| YCL025C | AGP1 | -1.682185 | -0.331049 | 0.823672 |
| YDR205W | MSC2 | -1.001279 | 0.140402 | -0.1044 |
| YIL006W | YIA6 | -1.039886 | 0.124122 | -0.01684 |
| YLR337C | VRP1 | -1.096899 | 0.188038 | -0.00914 |
| YNL321W | VNX1 | -1.180766 | 0.100233 | -0.12292 |


| YPL190C | $\begin{aligned} & \text { chat in } \\ & \text { NAB } \end{aligned}$ | $\begin{gathered} \text { oy peerrevew) } \\ -1.246992 \end{gathered}$ | $\begin{aligned} 1040 \\ 1.338699 \end{aligned}$ |  |
| :---: | :---: | :---: | :---: | :---: |
| YOR140W | SFL1 | -1.290206 | 0.326112 | -0.15347 |
| YMR069W | NAT4 | -1.221564 | 0.089779 | 0.273154 |
| YKR102W | FLO10 | -1.275704 | 0.194769 | 0.316539 |
| YDR232W | HEM1 | -1.332911 | 0.045818 | 0.348183 |
| YDR246W-A | null | -1.440417 | 0.158604 | 0.091489 |
| YDL140C | RPO21 | -1.31474 | 0.154825 | 0.066087 |
| YBR020W | GAL1 | -1.311369 | 0.086624 | 0.107964 |
| YOR349W | CIN1 | -1.048967 | -0.045259 | 0.035903 |
| YALO20C | ATS1 | -1.068134 | -0.002847 | 0.125437 |
| YGR079W | null | -1.15463 | -0.053631 | 0.260586 |
| YER152C | null | -1.022108 | -0.125026 | 0.246497 |
| YAL063C | FLO9 | -1.067861 | -0.071818 | 0.206601 |
| YKL187C | FAT3 | -1.036015 | 0.178003 | 0.196359 |
| YLR116W | MSL5 | -1.06763 | 0.179945 | 0.243781 |
| YKL198C | PTK1 | -1.096863 | 0.259249 | 0.20616 |
| YDR160W | SSY1 | -1.019812 | 0.148067 | 0.308233 |
| YER060W | FCY21 | -1.029224 | 0.203936 | 0.351275 |
| YGL178W | MPT5 | -1.179571 | 0.479767 | 0.359187 |
| YDR438W | THI74 | -1.258853 | 0.579677 | 0.370009 |
| YLR099C | ICT1 | -1.13923 | 0.600478 | 0.329931 |
| YLR403W | SFP1 | -1.212804 | 0.429344 | 0.237323 |
| YJL094C | KHA1 | -1.017959 | 0.514731 | 0.080093 |
| YMRO08C | PLB1 | -1.103382 | 0.676622 | 0.553909 |
| YGR289C | MAL11 | -1.084481 | 0.696059 | 0.692787 |
| YPL015C | HST2 | -1.078514 | 1.148372 | 0.591381 |
| YHR094C | HXT1 | -1.578098 | 0.964523 | 0.494114 |
| YGR023W | MTL1 | -1.551499 | 0.762923 | 0.690967 |
| YMR291W | TDA1 | -1.2614 | 0.269249 | 1.804324 |
| YGR032W | GSC2 | -1.242827 | 0.236364 | 1.776908 |
| YJL079C | PRY1 | -1.443107 | 0.230725 | 1.836415 |
| YBR067C | TIP1 | -1.191979 | 0.6789 | 2.078076 |
| YHLO26C | null | -1.244398 | 0.708651 | 1.464342 |
| YGR121C | MEP1 | -1.02356 | 0.688905 | 1.588961 |
| YER053C | PIC2 | -1.617607 | 0.204335 | 1.427363 |
| YJL212C | OPT1 | -1.53264 | 0.103232 | 1.427167 |
| YER158C | null | -1.423766 | 0.170882 | 1.213202 |
| YPL036W | PMA2 | -1.151877 | -0.145902 | 1.544101 |
| YLR142W | PUT1 | -1.002157 | 0.184707 | 1.238826 |
| YOR071C | NRT1 | -1.17284 | -0.074057 | 1.26817 |
| YGR260W | TNA1 | -1.147552 | 0.084828 | 1.242491 |
| YFL014W | HSP12 | -1.292276 | 0.237658 | 2.488439 |
| YPL092W | SSU1 | -1.065039 | -0.681846 | 0.960938 |
| YBL005W-A | null | -1.17081 | -0.719291 | 0.961157 |
| YNLO24C | null | -1.26106 | -0.90903 | 1.338866 |
| YNR014W | null | -1.922514 | -0.605679 | 1.777716 |
| YPR160W | GPH1 | -1.099576 | -0.583531 | 2.009052 |
| YOL104C | NDJ1 | -1.647413 | -1.534572 | 1.433746 |
| YDR222W | null | -2.011217 | -1.458479 | 0.856427 |


swi2D = swi2 null mutant; swi2Dsir3D = swi2 and sir3 null mutant, sir3D = sir3 null mutant, WT = Wildtype, 1810 = SWI2 Anchor away; 1854= SWI2 Anchor away sir3 null mutant; 1853 = sir3 null in Anchor away background; 1809 = Wildtype Anchor away background. _R = with Rapamycin

|  |  |  | swi2Dsir3D- |  | 1810_R1- |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| YORF | NAME | swi2D-WT | WT | sir3D-WT | 1809_R | 1854_R1-1809_R | 1853_R-1809_R |
| YBR238C | YBR2381 | -1.113228 | -1.032084 | 0.050758 | -2.643991 | -0.768649 | -0.051082 |
| YER028C | MIG3 | -0.63341 | 0.196135 | 0.762258 | -1.415682 | 1.989151 | 0.358269 |
| YHR094C | HXT1 | -1.207275 | 0.243913 | 0.274392 | -1.578098 | 0.964523 | 0.494114 |
| YKL078W | DHR2 | -1.078741 | -0.409123 | 0.246475 | -1.047444 | -0.702301 | -0.583786 |
| YDL042C | SIR2 | -0.852518 | -0.28447 | 0.312551 | -1.420121 | -0.998536 | -0.093641 |
| YDL179W | PCL9 | -1.4164 | -0.376551 | -0.21954 | -1.268798 | -0.745332 | -0.503944 |
| YDR044W | HEM13 | -0.787838 | -0.718158 | 0.576189 | -1.482312 | -0.685862 | -0.96316 |
| YDR222W | YDR222 | -1.421418 | 0.408224 | 0.079002 | -2.011217 | -1.458479 | 0.856427 |
| YDR384C | ATO3 | -0.95195 | -0.607941 | 0.163866 | -2.591182 | -1.606145 | -1.25726 |
| YGL028C | SCW11 | -0.98148 | -0.264637 | -0.118008 | -1.830712 | -0.060328 | -0.327612 |
| YGR041W | BUD9 | -0.81707 | -0.416311 | 0.221974 | -2.429062 | -1.035065 | -0.732587 |
| YGR079W | YGR079 | -1.05697 | -0.93515 | 0.135865 | -1.15463 | -0.053631 | 0.260586 |
| YLR099C | ICT1 | -0.403286 | -0.129096 | 0.199719 | -1.13923 | 0.600478 | 0.329931 |
| YLR121C | YPS3 | -1.101156 | -0.093144 | 0.564482 | -2.161079 | -0.657103 | 0.534248 |
| YMR011W | HXT2 | -1.047188 | -0.494915 | -0.209743 | -1.933943 | -0.067735 | 0.024425 |
| YNLO24C | EFM6 | -1.124738 | -0.668689 | -0.176781 | -1.26106 | -0.90903 | 1.338866 |
| YNL034W | YNL034 | -0.798485 | -0.305995 | -0.046402 | -1.018354 | -0.190546 | -0.123394 |
| YNL327W | EGT2 | -1.419537 | -0.456658 | -0.035435 | -1.412944 | -0.034828 | -0.171923 |
| YNR067C | DSE4 | -1.430648 | 0.257047 | 0.118234 | -1.660394 | -0.136176 | -0.363971 |
| YOLO20W | TAT2 | -0.739805 | -0.203711 | -0.071481 | -1.611607 | -0.635395 | -0.022112 |
| YOR342C | YOR342 | -0.809384 | -0.059979 | 0.125933 | -1.000736 | -1.138968 | 0.459689 |
| YPL057C | SUR1 | -1.304416 | -0.453506 | -0.09573 | -1.782771 | 0.123331 | 0.541527 |
| YPL066W | RGL1 | -0.538909 | -0.641721 | 0.178508 | -1.00754 | -1.297962 | -0.026519 |
| YPL092W | SSU1 | -0.784119 | -0.84839 | 0.077845 | -1.065039 | -0.681846 | 0.960938 |
| YPL165C | SET6 | -0.804397 | -0.320086 | 0.053357 | -2.289622 | 1.097893 | 2.20319 |
| YPR054W | SMK1 | -0.786343 | -0.572491 | 0.119735 | -1.248613 | -1.072935 | -0.345981 |
| YPR106W | ISR1 | -0.62125 | -0.625154 | -0.222107 | -1.79978 | -0.483187 | -0.226804 |
| YLR285C-A | YLR285( | -0.812289 | -0.440132 | -0.07874 | -1.578821 | -0.490248 | 0.013808 |

 wich was not certifted by peer review) is the author/funder. All rights reserved. No reuse allowed without permission. swi2D = swi2 null mutant; sir3D = sir3 null mutant, WT = Wildtype

| YORF | NAME | swi | i2Dsi | sir3D-WT |
| :---: | :---: | :---: | :---: | :---: |
| YHLO12W | YHLO12W | 1.39651 | 0.363307 | -0.024821 |
| YDL170W | YDL170W | 1.434712 | 0.331249 | 0.178301 |
| YLL060C | YLL060C | 1.345331 | 0.409135 | 0.188886 |
| YOL119C | YoL119C | 1.472612 | 0.061144 | -0.026633 |
| YJR130C | YJR130C | 1.328341 | 0.18232 | 0.025941 |
| YALO28W | YALO28W | 1.252044 | 0.133739 | -0.110437 |
| YER175C | YER175C | 1.426577 | -0.142406 | 0.086255 |
| YNL270C | YNL270C | 1.526947 | -0.30727 | -0.022384 |
| YBR145W | YBR145W | 1.176709 | -0.413801 | 0.091954 |
| YOR289W | YOR289W | 1.303741 | 0.188904 | -0.363791 |
| YJL089W | YJL089W | 1.392584 | 0.290784 | -0.29782 |
| YIL037C | YIL037C | 1.46089 | 0.263056 | -0.534638 |
| YNL194C | YNL194C | 1.510503 | 0.334908 | -0.37898 |
| YCL040W | YCLO40W | 1.474825 | 0.407789 | -0.438249 |
| YKR076W | YKR076W | 1.346451 | 0.424563 | -0.370127 |
| YDR516C | YDR516C | 1.157875 | 0.424397 | -0.411819 |
| YHL044W | YHLO44W | 1.284105 | 0.442617 | -0.568168 |
| YDR309C | YDR309C | 1.169489 | 0.019513 | -0.596586 |
| YGL032C | YGL032C | 1.138942 | -0.080326 | -0.509035 |
| YDL085W | YDL085W | 1.278246 | -0.204536 | -0.537333 |
| YJL103C | YJL103C | 1.095172 | 0.228025 | -0.301721 |
| YMR306W | YMR306W | 1.062371 | 0.272699 | -0.358249 |
| Yol083W | YoL083W | 1.006853 | 0.361934 | -0.320293 |
| YLR270W | YLR270W | 0.941675 | 0.318399 | -0.313685 |
| YAR023C | Yar023C | 0.861997 | 0.295574 | -0.298766 |
| YKL142W | YKL142W | 0.833649 | 0.292383 | -0.429116 |
| YGL104C | YGL104C | 0.867143 | 0.316828 | -0.374073 |
| YAR068W | YAR068W | 0.80976 | -0.118429 | -0.470585 |
| YDLO90C | YDLo90C | 0.790183 | -0.041284 | -0.312308 |
| YHLO22C | YHLO22C | 0.888585 | 0.242201 | -0.507559 |
| YKL217W | YKL217W | 0.882017 | 0.095171 | -0.621602 |
| YGL185C | YGL185C | 0.946109 | 0.881844 | -0.050654 |
| YKR066C | YKR066C | 1.025034 | 0.91132 | -0.026599 |
| YJR103W | YJR103W | 1.067124 | 0.916631 | -0.012947 |
| YPL134C | YPL134C | 1.014002 | 0.862032 | 0.00447 |
| YLR142W | YLR142W | 1.010006 | 0.816799 | 0.045308 |
| YCRO37C | YCRO37C | 1.023084 | 0.81118 | 0.038704 |
| YEL044W | Yelo44W | 0.991116 | 0.832672 | 0.08014 |
| YGL183C | YGL183C | 0.952034 | 0.767311 | -0.026404 |
| YBR230C | YBR230C | 0.892935 | 0.76316 | -0.091499 |
| YLR412C- | YLR412C-A | 0.890019 | 0.81298 | -0.054856 |
| YGL081W | YGL081W | 1.136778 | 0.526974 | 0.151449 |
| YJL155C | YJL155C | 1.155024 | 0.552499 | 0.052828 |
| YLR356W | YLR356W | 1.129004 | 0.560602 | 0.108706 |
| YJL071W | YJLO71W | 1.092489 | 0.56018 | 0.082162 |
| YMR094W | YMR094W | 1.160889 | 0.638316 | 0.075412 |


| YJQioRxivpreprintotorntup YKL086W YKL086W | //doiqrity $9.7101 / 2020$ tritited by peer review) |  | 5; ©his2 2qugipg posted March 25, 2020. The copyright holder for this preprint funder. All rights reserved. No reuse allowed without permission. |
| :---: | :---: | :---: | :---: |
|  | 1.142035 | 0.673786 | 0.124597 |
| YOL084W YOL084W | 1.132696 | 0.61427 | 0.148361 |
| YDL149W YDL149W | 1.185467 | 0.659495 | 0.306894 |
| YILO24C YIL024C | 1.11742 | 0.627506 | 0.220307 |
| YPR036W- YPR036W- | 1.066355 | 0.731882 | 0.263468 |
| YML120C YML120C | 1.088666 | 0.762926 | 0.128583 |
| YDL138W YDL138W | 1.110095 | 0.72298 | 0.164833 |
| YJR036C YJR036C | 1.178944 | 0.7529 | 0.138457 |
| YPL113C YPL113C | 1.171352 | 0.720667 | 0.114903 |
| YIL074C YIL074C | 1.244388 | 0.77317 | 0.168305 |
| YML087C YML087C | 1.237079 | 0.686985 | 0.169487 |
| YDR452W YDR452W | 0.886152 | 0.55767 | 0.04458 |
| YIL046W YIL046W | 0.837093 | 0.584252 | 0.002653 |
| YLR417W YLR417W | 0.825852 | 0.557197 | 0.027376 |
| YLR350W YLR350W | 0.907203 | 0.641615 | 0.020785 |
| YNR058W YNR058W | 0.886758 | 0.633461 | 0.005521 |
| YBR150C YBR150C | 0.91409 | 0.524 | 0.02349 |
| YGL036W YGL036W | 0.890575 | 0.485573 | 0.015498 |
| YOR177C YOR177C | 0.893837 | 0.500689 | -0.037962 |
| YPR026W YPR026W | 0.87614 | 0.456565 | -0.018907 |
| YKL171W YKL171W | 0.837674 | 0.501156 | 0.094123 |
| YKL026C YKL026C | 0.822128 | 0.459803 | 0.057352 |
| YDL024C YDLO24C | 0.793465 | 0.483795 | -0.02712 |
| YLR189C YLR189C | 0.775608 | 0.501842 | -0.030236 |
| YBR001C YBR001C | 0.819745 | 0.525976 | -0.060255 |
| YIL162W YIL162W | 0.813445 | 0.503346 | 0.017028 |
| YDR022C YDR022C | 0.835347 | 0.516279 | -0.019088 |
| YKL107W YKL107W | 0.776387 | 0.59272 | -0.069764 |
| YMR020W YMR020W | 0.7955 | 0.546233 | 0.005497 |
| YMR147W YMR147W | 0.761638 | 0.569792 | -0.019936 |
| YMR139W YMR139W | 0.708373 | 0.514123 | 0.018162 |
| YGR207C YGR207C | 0.709674 | 0.545402 | 0.024355 |
| YGR080W YGR080W | 0.6698 | 0.580932 | 0.029934 |
| YOLO32W YOL032W | 0.740201 | 0.607051 | 0.018664 |
| YGL045W YGL045W | 0.74862 | 0.520836 | 0.126003 |
| YMR114C YMR114C | 0.715137 | 0.581373 | 0.100723 |
| YAL061W YAL061W | 1.080048 | 0.63144 | 0.015344 |
| YFL042C YFL042C | 1.070022 | 0.600394 | -0.016275 |
| YHLO24W YHL024W | 1.045911 | 0.667195 | -0.027581 |
| YKR097W YKR097W | 0.943387 | 0.652423 | -0.043934 |
| YDL027C YDL027C | 0.97885 | 0.647415 | -0.060379 |
| YJL045W YJL045W | 0.959893 | 0.616837 | 0.002423 |
| YML100W YML100W | 1.017937 | 0.622644 | 0.003245 |
| YGR250C YGR250C | 0.936389 | 0.749598 | 0.111166 |
| YFL064C YFL064C | 0.931082 | 0.740061 | 0.099967 |
| YPR155C YPR155C | 0.965036 | 0.741548 | 0.192373 |
| YDR132C YDR132C | 0.969367 | 0.754427 | 0.131802 |
| YMR030W YMR030W | 0.944327 | 0.78541 | 0.146986 |
| YDR358W YDR358W | 1.018333 | 0.653511 | 0.189238 |


| YMiRB30inerepriptplsionteres(which was not YDL238C YDL238C | :/dog $96898101 / 20200$ ertified by peer review) |  | 5; dhis 2 Gisinn posted March 25, 2020. The copyright holder for this rfunder. All rights reserved. No reuse allowed without permission. 0.148495 |
| :---: | :---: | :---: | :---: |
|  |  | 0.649432 |  |
| YKL129C YKL129C | 1.025219 | 0.573397 | 0.178255 |
| YIR014W YIR014W | 1.018121 | 0.564928 | 0.195361 |
| YNL077W YNL077W | 0.979824 | 0.611458 | 0.135164 |
| YCR107W YCR107W | 0.94217 | 0.59097 | 0.159194 |
| YLR260W YLR260W | 0.900669 | 0.607359 | 0.200363 |
| YNR007C YNR007C | 0.953163 | 0.570762 | 0.235159 |
| YBR285W YBR285W | 0.94172 | 0.618527 | 0.232708 |
| YJL070C YJL070C | 0.960937 | 0.508825 | 0.348488 |
| YOR113W YOR113W | 0.963024 | 0.536857 | 0.371851 |
| YCR107W YCR107W | 1.011328 | 0.560049 | 0.300429 |
| YIL107C YIL107C | 0.893802 | 0.522698 | 0.217952 |
| YDL174C YDL174C | 0.913486 | 0.478663 | 0.255499 |
| YFR047C YFR047C | 0.872582 | 0.528302 | 0.138168 |
| YGR097W YGR097W | 0.88382 | 0.501875 | 0.141175 |
| YIR031C YIR031C | 0.803063 | 0.515764 | 0.190921 |
| YOR208W YOR208W | 0.86384 | 0.540229 | 0.311201 |
| YKL124W YKL124W | 0.784504 | 0.516351 | 0.303699 |
| YDR350C YDR350C | 0.799453 | 0.528983 | 0.306911 |
| YIR007W YIR007W | 0.822092 | 0.502518 | 0.274489 |
| YNLO74C YNL074C | 0.917302 | 0.425195 | 0.170422 |
| YMR160W YMR160W | 0.867979 | 0.440628 | 0.187692 |
| YPR081C YPR081C | 0.879047 | 0.386579 | 0.248651 |
| YEL072W YEL072W | 0.900306 | 0.35021 | 0.233605 |
| YFL016C YFL016C | 0.913885 | 0.355887 | 0.209021 |
| YIL154C YIL154C | 0.879745 | 0.374802 | 0.194374 |
| YNL092W YNL092W | 0.800537 | 0.395907 | 0.117566 |
| YGR239C YGR239C | 0.806245 | 0.398141 | 0.114831 |
| YDL054C YDL054C | 0.844388 | 0.377671 | 0.146408 |
| YNL223W YNL223W | 0.871289 | 0.349675 | 0.122003 |
| YGL141W YGL141W | 0.782891 | 0.383586 | 0.187871 |
| YEL059C-A YEL059C-A | 0.764673 | 0.385062 | 0.215634 |
| YAR050W YAR050W | 0.78565 | 0.338906 | 0.218081 |
| YCR063W YCR063W | 0.764622 | 0.39164 | 0.250625 |
| YMR108W YMR108W | 0.831192 | 0.399722 | 0.26184 |
| YJR091C YJR091C | 0.821343 | 0.399253 | 0.272638 |
| YLR128W YLR128W | 0.819945 | 0.363568 | 0.275342 |
| YOLO36W YOLO36W | 0.779427 | 0.430364 | 0.324494 |
| YGR112W YGR112W | 0.779069 | 0.360068 | 0.323016 |
| YBR241C YBR241C | 1.286382 | 0.394891 | -0.056671 |
| YBR169C YBR169C | 1.204758 | 0.444963 | -0.070752 |
| YIL072W YIL072W | 1.174991 | 0.353256 | -0.188291 |
| YOR100C YOR100C | 1.223167 | 0.34738 | -0.226438 |
| YJR025C YJR025C | 1.203687 | 0.27778 | -0.095241 |
| YKL151C YKL151C | 1.120125 | 0.47562 | -0.130729 |
| YOR386W YOR386W | 1.068739 | 0.405265 | -0.187585 |
| YGL227W YGL227W | 1.017435 | 0.512009 | -0.119218 |
| YKLO23W YKL023W | 1.015047 | 0.484131 | -0.127903 |
| YJL037W YJL037W | 1.139261 | 0.529247 | -0.300059 |





| YKR102W YKR102W | 0.595357 | 0.284468 | 0.591786 |
| :---: | :---: | :---: | :---: |
| YOR018W YOR018W | 0.764406 | 0.480334 | 0.561306 |
| YJL100W YJL100W | 0.742634 | 0.433836 | 0.584511 |
| YNL144C YNL144C | 0.705902 | 0.423877 | 0.493434 |
| YFRO22W YFR022W | 0.740657 | 0.402394 | 0.410533 |
| YOR077W YOR077W | 0.690867 | 0.390151 | 0.388449 |
| YNR065C YNR065C | 0.72087 | 0.386812 | 0.325778 |
| YER162C YER162C | 0.733909 | 0.434537 | 0.323612 |
| YOL051W YOL051W | 0.724742 | 0.441366 | 0.335819 |
| YML076C YML076C | 0.670445 | 0.41375 | 0.322985 |
| YGR258C YGR258C | 0.691146 | 0.408519 | 0.334469 |
| YPR002W YPR002W | 0.67846 | 0.467907 | 0.362775 |
| YCR024C YCR024C | 0.612769 | 0.360917 | 0.316836 |
| YDR528W YDR528W | 0.580712 | 0.358707 | 0.381767 |
| YMR253C YMR253C | 0.707862 | 0.277376 | 0.436816 |
| YGR288W YGR288W | 0.680275 | 0.315446 | 0.372509 |
| YGL219C YGL219C | 0.72029 | 0.280519 | 0.332057 |
| YLL063C YLL063C | 0.737355 | 0.305061 | 0.310217 |
| YMR182C YMR182C | 0.56691 | -0.092589 | 0.650821 |
| YJL161W YJL161W | 1.112427 | -0.098702 | -0.090355 |
| YHR018C YHR018C | 1.097463 | -0.010042 | -0.282741 |
| YBR214W YBR214W | 1.048822 | 0.08978 | -0.19885 |
| YLL055W YLL055W | 1.084256 | 0.034793 | -0.084343 |
| YGR019W YGR019W | 0.756911 | 0.03545 | -0.17009 |
| YJL163C YJL163C | 0.742659 | -0.065623 | -0.098911 |
| YBR298C YBR298C | 0.965357 | 0.020371 | -0.242704 |
| YER096W YER096W | 0.903116 | 0.096143 | -0.207056 |
| YAR066W YAR066W | 0.867582 | 0.02989 | -0.215754 |
| YIL116W YIL116W | 0.845978 | -0.160288 | -0.082352 |
| YMR018W YMR018W | 0.907539 | -0.153467 | 0.050713 |
| YBR183W YBR183W | 0.926466 | -0.15967 | -0.026584 |
| YBR132C YBR132C | 0.956762 | -0.269158 | 0.184922 |
| YBR047W YBR047W | 0.916639 | -0.148777 | 0.278189 |
| YNL104C YNL104C | 0.777248 | -0.15216 | 0.191172 |
| YGR053C YGR053C | 0.752639 | -0.138203 | 0.106464 |
| YLR177W YLR177W | 0.904206 | -0.048365 | 0.145183 |
| YML116W YML116W | 0.857739 | -0.136079 | 0.139293 |
| YIR019C YIR019C | 0.647143 | -0.018034 | 0.106158 |
| YOR316C YOR316C | 0.663918 | -0.065329 | 0.16934 |
| YHL006C YHLO06C | 0.724924 | -0.028565 | 0.128995 |
| YOR032W YOR032W. | 0.585149 | -0.072346 | 0.074312 |
| YAL002W YAL002W | 0.593642 | -0.046103 | 0.105006 |
| YDR127W YDR127W | 0.676811 | -0.068427 | 0.026518 |
| YIL164C YIL164C | 0.686601 | -0.036841 | -0.011433 |
| YER066W YER066W | 0.701785 | 0.018448 | -0.028866 |
| YML117W YML117W | 0.727407 | -0.018494 | -0.034254 |
| YMR251W YMR251W | 0.684227 | -0.14025 | -0.001341 |
| YGR070W YGR070W | 0.628337 | -0.222661 | 0.002256 |
| YGR045C YGR045C | 0.605165 | -0.19431 | -0.00647 |


| YIL088C YIL088C | $\begin{aligned} & \text { entitif by pee } \\ & 0.576831 \end{aligned}$ | $\begin{aligned} & \text { w) in autho } \\ & -0.052844 \end{aligned}$ | $\begin{aligned} \text { rffunder. An rig } \\ -0.022622 \end{aligned}$ |
| :---: | :---: | :---: | :---: |
| YBR233W YBR233W | 0.628194 | -0.054141 | -0.004612 |
| YOR173W YOR173W | 0.635113 | -0.081923 | -0.048003 |
| YDR270W YDR270W | 0.535447 | -0.175195 | -0.006447 |
| YOR081C YOR081C | 0.621488 | -0.1019 | -0.15413 |
| YLR034C YLR034C | 0.557415 | -0.117486 | -0.134807 |
| Yalo17W Yaloitw | 0.644572 | -0.083116 | -0.284143 |
| YKRO49C YKRO49C | 0.659206 | -0.169805 | $-0.259712$ |
| YPL257W YPL257W | 0.575563 | -0.290897 | -0.237339 |
| YNROO1C YNROO1C | 0.835871 | 0.09965 | 0.025909 |
| YDR479C YDR479C | 0.758988 | 0.148139 | 0.093184 |
| YOL140W YOL140W | 0.769097 | 0.161215 | 0.067367 |
| YCR069W YCRO69W | 0.648985 | 0.061656 | 0.026119 |
| YAL031C YALO31C | 0.671957 | 0.06073 | 0.013513 |
| YHR198C YHR198C | 0.679071 | 0.140465 | 0.051539 |
| YBR128C YBR128C | 0.649162 | 0.136315 | 0.071388 |
| YMR053C YMR053C | 0.828506 | 0.206417 | -0.09831 |
| YDL216C YDL216C | 0.785739 | 0.188184 | -0.049739 |
| YMRO19W YMR019W | 0.705654 | 0.105397 | -0.030237 |
| YLL041C YLLO41C | 0.704188 | 0.090088 | -0.102415 |
| YHR006W YHR006W | 0.694044 | 0.384871 | 0.078626 |
| YbRO35C Ybro35C | 0.681442 | 0.411983 | 0.066884 |
| YDR436W YDR436W | 0.701215 | 0.499668 | 0.089 |
| YPL230W YPL230W | 0.694019 | 0.486134 | 0.086743 |
| YOR350C YOR350C | 0.697096 | 0.460254 | 0.072359 |
| YMR278W YMR278W | 0.706045 | 0.401434 | 0.02805 |
| YORO59C YORO59C | 0.727356 | 0.41682 | 0.015696 |
| YDL035C YDLO35C | 0.689415 | 0.387173 | -0.040217 |
| YJL141C YJL141C | 0.717176 | 0.374218 | -0.028346 |
| YIL097W YIL097W | 0.761334 | 0.428919 | -0.055043 |
| YPL109C YPL109C | 0.745653 | 0.426795 | -0.017391 |
| YKL065C YKL065C | 0.728626 | 0.478298 | -0.019887 |
| YAR028W Yaro28W | 0.729349 | 0.465076 | -0.023449 |
| YDR191W YDR191W | 0.711605 | 0.465276 | -0.022777 |
| YFL041W-1 YFL041W-ı | 0.733267 | 0.457053 | 0.019081 |
| YELO12W YELO12W | 0.766935 | 0.357536 | 0.046376 |
| YJL048C YJL048C | 0.791764 | 0.395257 | 0.030323 |
| YLR312C-B YLR312C-B | 0.771005 | 0.377709 | 0.00474 |
| YGL181W YGL181W | 0.632044 | 0.475256 | 0.060042 |
| YkL094W YKL094W | 0.573746 | 0.498888 | 0.024055 |
| YNLO14W YNLO14W | 0.601777 | 0.477578 | 0.027808 |
| YOR152C YOR152C | 0.611727 | 0.499225 | 0.013987 |
| YKL104C YKL104C | 0.573801 | 0.358568 | 0.101818 |
| YPL174C YPL174C | 0.603308 | 0.361553 | 0.106251 |
| YCRO26C YCRO26C | 0.590847 | 0.387899 | 0.081455 |
| YLR257W YLR257W | 0.582456 | 0.375314 | 0.091129 |
| YDR383C YDR383C | 0.591796 | 0.444481 | 0.14362 |
| YNL242W YNL242W | 0.624529 | 0.410285 | 0.114073 |
| YDR058C YDRO58C | 0.699754 | 0.509971 | -0.07543 |



|  | entified by pee $0.481823$ | ) is the auth | Chisgqusiby |
| :---: | :---: | :---: | :---: |
| YGL206C YGL206C | 0.542943 | 0.371285 | 0.053108 |
| YFL041W YFL041W | 0.472184 | 0.363991 | 0.036445 |
| YKR034W YKR034W | 0.439557 | 0.262326 | 0.023869 |
| YCR032W YCR032W | 0.390897 | 0.374289 | 0.021102 |
| YGL053W YGL053W | 0.70292 | 0.15773 | -0.309933 |
| YMR196W YMR196W | 0.729957 | 0.139779 | -0.299056 |
| YMR315W YMR315W | 0.581139 | 0.005065 | -0.297006 |
| YER039C YER039C | 0.658019 | 0.03216 | -0.272236 |
| YLR156W YLR156W | 0.687946 | -0.006232 | -0.209862 |
| YBR006W YBR006W | 0.635928 | 0.056828 | -0.193555 |
| YAL001C YALO01C | 0.550178 | 0.105731 | -0.194594 |
| YPR007C YPR007C | 0.548947 | 0.082577 | -0.257611 |
| YBR076W YBR076W | 0.451802 | 0.151175 | -0.313629 |
| YBL091C-A YBL091C-A | 0.623323 | 0.231421 | -0.24542 |
| YJR008W YJR008W | 0.673995 | 0.28283 | -0.275076 |
| YNL006W YNL006W | 0.658405 | 0.28475 | -0.250896 |
| YHR161C YHR161C | 0.707763 | 0.227858 | -0.135645 |
| YCR091W YCR091W | 0.719086 | 0.25168 | -0.164835 |
| YLR211C YLR211C | 0.716051 | 0.299954 | -0.144395 |
| YIL050W YIL050W | 0.75505 | 0.244964 | -0.217685 |
| YAL062W YAL062W | 0.730282 | 0.246295 | -0.229944 |
| YJL185C YJL185C | 0.639832 | 0.228435 | -0.077771 |
| YPR196W YPR196W | 0.610963 | 0.242417 | -0.143186 |
| YPR079W YPR079W | 0.635556 | 0.217538 | -0.132211 |
| YKR089C YKR089C | 0.680805 | 0.183049 | -0.10222 |
| YPR201W YPR201W | 0.696521 | 0.190369 | -0.160795 |
| YGL250W YGL250W | 0.58896 | 0.167738 | -0.17094 |
| YOR136W YOR136W | 0.59268 | 0.370343 | -0.162238 |
| YDL059C YDL059C | 0.650068 | 0.330581 | -0.139622 |
| YKL193C YKL193C | 0.668065 | 0.359615 | -0.161929 |
| YFR024C-f YFR024C-A | 0.659291 | 0.333679 | -0.219687 |
| YDL223C YDL223C | 0.641747 | 0.300869 | -0.170029 |
| YOR347C YOR347C | 0.681784 | 0.389476 | -0.259869 |
| YKL208W YKL208W | 0.565007 | 0.422599 | -0.257768 |
| YKR013W YKR013W | 0.454937 | 0.515898 | -0.181775 |
| YAR035W YAR035W | 0.613453 | 0.393402 | -0.373743 |
| YBR149W YBR149W | 0.623446 | 0.281616 | -0.367106 |
| YGR244C YGR244C | 0.719554 | 0.330664 | -0.392958 |
| YHR138C YHR138C | 0.633966 | 0.45974 | -0.533666 |
| YOR020W• YOR020W. | 0.651711 | 0.525487 | -0.414666 |

 ned peer review) is the authorftunder. All rights reserved. No reuse allowed without permission. 1810 = SWI2 Anchor away; 1854= SWI2 Anchor away sir3 null mutant; 1853 = sir3 null in Anchor away background; 1809 = Wildtype Anchor away background. _R = with Rapamycin

| YORF | NAME | 1810_R1-1809_R | 1854_R1 | 853_R-1809_R |
| :---: | :---: | :---: | :---: | :---: |
| YFR027W | ECO1 | 1.066789 | -0.045791 | -0.669208 |
| YML108W | null | 1.029809 | -0.137468 | -0.538262 |
| YPR148C | null | 1.267408 | -0.077573 | -0.522108 |
| YOL108C | INO4 | 1.16215 | -0.019617 | -0.617004 |
| YDL101C | DUN1 | 1.180908 | 0.008541 | -0.527505 |
| YLR420W | URA4 | 1.285333 | 5.47E-04 | -0.319895 |
| YOR357C | SNX3 | 1.25215 | 0.03339 | -0.412955 |
| YDL135C | RDI1 | 1.203921 | -0.001992 | -0.216348 |
| YMR263W | SAP30 | 1.102979 | -0.021424 | -0.273703 |
| YLR312C | null | 1.01248 | -0.193766 | -0.110078 |
| YDR308C | SRB7 | 1.040916 | -0.11343 | -0.102671 |
| YNR029C | null | 1.050316 | -0.128786 | -0.059821 |
| YMR029C | FAR8 | 1.002667 | -0.058155 | -0.084481 |
| YBR014C | GRX7 | 1.082561 | -0.200944 | -0.265018 |
| YCR082W | AHC2 | 1.088685 | -0.130606 | -0.218864 |
| YDR130C | FIN1 | 1.06606 | -0.17393 | -0.215208 |
| YKL089W | MIF2 | 1.140109 | -0.213645 | -0.15261 |
| YFL034C-A | RPL22B | 1.033232 | -0.217076 | -0.193786 |
| YKR035W- | DID2 | 1.016133 | -0.253712 | -0.24015 |
| Yerozow | CHZ1 | 1.607177 | 0.09653 | -0.370041 |
| YJR022W | LSM8 | 1.538216 | 0.107655 | -0.395975 |
| YPR188C | MLC2 | 1.639887 | -0.114558 | -0.278256 |
| YOR195W | SLK19 | 1.516348 | -0.219008 | -0.286149 |
| YOR194C | TOA1 | 1.286919 | -0.006988 | 0.012911 |
| YGR135W | PRE9 | 1.238083 | 0.063397 | -0.014367 |
| YPR100W | MRPL51 | 1.361305 | 0.102015 | 0.122359 |
| YDR315C | IPK1 | 1.432264 | 0.036723 | 0.137238 |
| YPRO73C | LTP1 | 1.425334 | 0.088795 | 0.090258 |
| YDR079C- | TFB5 | 1.311372 | -0.059416 | 0.088674 |
| YBL090W | MRP21 | 1.31457 | 0.003371 | 0.235119 |
| YIL063C | YRB2 | 1.424641 | 0.128045 | -0.006819 |
| YDL216C | RRI1 | 1.484604 | 0.244172 | -0.107044 |
| YKRO29C | SET3 | 1.084159 | 0.502969 | -0.199273 |
| YNLO50C | null | 1.113443 | 0.55606 | -0.237633 |
| YLR262C | YPT6 | 1.201175 | 0.424257 | -0.27768 |
| YGL244W | RTF1 | 1.064341 | 0.308883 | -0.407579 |
| YER170W | ADK2 | 1.086861 | 0.311351 | -0.253826 |
| YOR094W | ARF3 | 1.03361 | 0.259975 | -0.280725 |
| YDR162C | NBP2 | 1.090693 | 0.510215 | -0.584332 |
| YPL045W | VPS16 | 1.015906 | 0.632306 | -0.45783 |
| YIL138C | TPM2 | 1.682786 | 0.306056 | -0.690793 |
| YDL161W | ENT1 | 1.34342 | 0.389242 | -0.723547 |
| YLR148W | PEP3 | 1.353599 | 0.187139 | -0.569213 |
| YER159C | BUR6 | 1.670063 | 0.437402 | -0.210862 |
| YELO21W | URA3 | 1.632845 | 0.262002 | -0.268966 |

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| YNL122C null | 1.014566 | -0.117233 | 0.197204 |
| :---: | :---: | :---: | :---: |
| YGLO29W CGR1 | 1.054287 | -0.134631 | 0.052386 |
| YMR039C SUB1 | 1.12259 | -0.285114 | 0.368477 |
| YKR060W UTP30 | 1.097246 | -0.373862 | 0.244983 |
| YOR305W RRG7 | 1.281278 | -0.420408 | 0.031223 |
| YJL140W RPB4 | 1.223792 | -0.318465 | 0.080238 |
| YJR057W CDC8 | 1.020569 | -0.374342 | -0.112441 |
| YBR098W MMS4 | 1.162837 | 0.177413 | 0.094098 |
| YMR188C MRPS17 | 1.092885 | 0.22016 | 0.045758 |
| YJL155C FBP26 | 1.093268 | 0.238717 | 0.093247 |
| YMR158W MRPS8 | 1.031525 | 0.153426 | 0.08375 |
| YDR004W RAD57 | 1.060236 | 0.106427 | 0.072368 |
| YKL082C RRP14 | 1.108827 | 0.094592 | 0.006446 |
| YDR494W RSM28 | 1.004573 | 0.313237 | 0.0797 |
| YPR131C NAT3 | 1.011685 | 0.308937 | -0.040734 |
| YJL162C JJJ2 | 1.080659 | 0.360894 | 0.010818 |
| YNL084C END3 | 1.090977 | 0.101192 | -0.099086 |
| YML015C TAF11 | 1.04558 | 0.105481 | -0.175426 |
| YGR029W ERV1 | 1.092301 | 0.227575 | -0.105768 |
| YER048C CAJ1 | 1.047871 | 0.271252 | -0.17278 |
| YOR266W PNT1 | 1.049498 | 0.285775 | -0.183032 |
| YGL230C null | 1.258502 | 0.156629 | -0.213166 |
| YLR051C FCF2 | 1.280758 | 0.207603 | -0.12482 |
| YNR032C-ヶ HUB1 | 1.214463 | 0.103032 | -0.10574 |
| YCL055W KAR4 | 1.211781 | 0.277987 | -0.020767 |
| YDL235C YPD1 | 1.260954 | 0.268571 | -0.070049 |
| YJL085W EX070 | 1.179199 | 0.305385 | -0.089646 |
| YDL099W BUG1 | 1.234792 | 0.341265 | -0.154571 |
| YMR200W ROT1 | 1.204718 | 0.45217 | 0.002839 |
| YJL072C PSF2 | 1.166446 | 0.463231 | -0.036795 |
| YGL058W RAD6 | 1.061177 | 0.473075 | 0.046744 |
| YPL108W null | 1.126847 | 0.43002 | 0.041395 |
| YGR287C IMA1 | 1.13376 | 0.542336 | 0.029656 |
| YGL158W RCK1 | 1.048703 | 0.481122 | -0.036415 |
| YDL002C NHP10 | 1.079376 | 0.467327 | -0.040343 |
| YDR140W MTQ2 | 1.088689 | 0.467984 | -0.007828 |
| YGR240C-/ null | 1.112271 | 0.503602 | -0.062786 |
| YOL023W IFM1 | 1.026897 | 0.570025 | -0.089091 |
| YDR393W SHE9 | 1.033013 | 0.402441 | -0.174446 |
| YPR051W MAK3 | 1.290515 | 0.328365 | 0.088339 |
| YGR196C FYV8 | 1.312014 | 0.364993 | 0.065092 |
| YBR111W-SUS1 | 1.372997 | 0.313199 | 0.013141 |
| YPL052W OAZ1 | 1.1649 | 0.287288 | 0.032563 |
| YKR014C YPT52 | 1.199055 | 0.350467 | 0.081193 |
| YNL306W MRPS18 | 1.20091 | 0.284713 | 0.108797 |
| YPL051W ARL3 | 1.289894 | 0.507288 | 0.138563 |
| YDR056C null | 1.182859 | 0.504021 | 0.170293 |
| YGR215W RSM27 | 1.131787 | 0.178328 | 0.231714 |
| YKL138C MRPL31 | 1.079247 | 0.208727 | 0.219332 |

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| YCR071C IMG2 | 1.111693 | 0.307811 | 0.281549 |
| :---: | :---: | :---: | :---: |
| YLR312W-, MRPL15 | 1.190386 | 0.342826 | 0.324444 |
| YDR337W MRPS28 | 1.09922 | 0.421759 | 0.167552 |
| YPRO20W ATP20 | 1.0745 | 0.346415 | 0.162848 |
| YDL119C null | 1.019403 | 0.384499 | 0.20403 |
| YLR257W null | 1.103853 | 0.045546 | 0.384654 |
| YLR353W BUD8 | 1.16627 | 0.068854 | 0.30876 |
| YBRO35C PDX3 | 1.124083 | 0.174003 | 0.417988 |
| YLLOO9C COX17 | 1.018919 | 0.197931 | 0.401414 |
| YPRO72W NOT5 | 1.064522 | 0.078616 | 0.260964 |
| YKL053C-A MDM35 | 1.025934 | 0.14345 | 0.201852 |
| YBLO38W MRPL16 | 1.09796 | 0.216539 | 0.324062 |
| YOL133W HRT1 | 1.066203 | 0.142261 | 0.354629 |
| YGR168C null | 1.028826 | 0.142986 | 0.296027 |
| YHR207C SET5 | 1.064848 | 0.256471 | 0.286648 |
| YOR166C SWT1 | 1.018944 | 0.250358 | 0.263306 |
| YMR299C DYN3 | 1.445678 | 0.274948 | 0.279285 |
| YHLO22C SPO11 | 1.539086 | 0.169963 | 0.328393 |
| YDL200C MGT1 | 1.517248 | 0.123993 | 0.284844 |
| YJL127C-B null | 1.303283 | 0.155014 | 0.26686 |
| YDR468C TLG1 | 1.333146 | 0.169745 | 0.239176 |
| YDR243C PRP28 | 1.291768 | 0.201127 | 0.195965 |
| YOR148C SPP2 | 1.309947 | 0.185429 | 0.370267 |
| YMR132C JLP2 | 1.343667 | 0.267558 | 0.316044 |
| YDR318W MCM21 | 1.408467 | 0.206356 | 0.359363 |
| YKL137W CMC1 | 1.396391 | 0.152925 | 0.454729 |
| YDR272W GLO2 | 1.406336 | 0.264159 | 0.508817 |
| YOL017W ESC8 | 1.290712 | 0.085347 | 0.530284 |
| YJR082C EAF6 | 1.289898 | 0.193897 | 0.578618 |
| YKL002W DID4 | 1.165457 | 0.127193 | 0.593513 |
| YGR153W null | 2.636502 | 0.65501 | -0.717081 |
| YNLO56W OCA2 | 2.209609 | 0.202457 | 0.243769 |
| YCR020W- HTL1 | 2.43541 | 0.626473 | 0.604439 |
| YLR025W SNF7 | 1.549937 | 0.602071 | -0.037162 |
| YmL062C MFT1 | 1.467596 | 0.50822 | -0.069654 |
| YOR078W BUD21 | 1.433841 | 0.437274 | -0.129684 |
| YMR284W YKU70 | 1.38401 | 0.491141 | -0.018359 |
| YKL160W ELF1 | 1.4777 | 0.386218 | 0.03296 |
| YJL013C MAD3 | 1.470005 | 0.373521 | 0.130819 |
| YDR289C RTT103 | 1.539179 | 0.401045 | 0.156093 |
| YJR011C null | 1.612044 | 0.511631 | 0.185125 |
| YMR197C VTI1 | 1.531006 | 0.565154 | 0.129688 |
| YDR068W DOS2 | 1.553337 | 0.561149 | 0.133572 |
| YDR168W CDC37 | 1.535247 | 0.585925 | 0.192948 |
| YLR170C APS1 | 1.752593 | 0.476661 | 0.012479 |
| YJL179W PFD1 | 1.907616 | 0.404717 | 0.135655 |
| YOL086W-MHF1 | 2.026676 | 0.363643 | 0.046947 |
| YKL138C-A HSK3 | 1.988677 | 0.5422 | -0.05758 |
| YDR371W CTS2 | 1.884904 | 0.719605 | 0.198772 |


YDR357C CNLIC1
YBLO01C ECM
YOR193W PEX2
YHR018C ARG4
YBR253W SRB6
YGLO05C COG
YOR189W IES4
YNLO32W SIW1

| by |  |
| :--- | :--- |
| 1.963634 | 0.278897 |
| 1.897314 |  |

YLR168C UPS2
YDR248C nul
YBR258C SHG1
YDR363W- SEM1
YBLO31W SHE1
YLR298C YHC1
YFR011C MIC19
YJR088C EMC2
YDR163W CWC15
YOR279C RFM1
YBR230W- null
YGL185C null
YIL161W null
Yeloosw Gim4
YOR319W HSH49
YFLO59W SNZ3
YLRO40C AFB1
YHR152W SPO12
YNL166C BNI5
YLR200W YKE2
YIL117C PRM5
YKR095W- PCC1
YPRO46W MCM16
YBR138C null
YFL065C null
YBR233W- DAD3
YDR501W PLM2
YALO34W- MTW1
YKL216W URA1
YOL113W SKM1
YILO08W URM1
YNLO79C TPM1
YNL129W NRK1
YDR378C LSM6
YFL060C SNO3
YLR154C RNH203
YOR216C RUD3
YNL188W KAR1

| 1.897314 | 0.4213 | 0.630408 |
| ---: | ---: | ---: |
| 1.664403 | 0.370363 | 0.792536 |
| 1.744191 | 0.212447 | 0.764307 |
| 1.66342 | 0.413677 | 0.451705 |

$\begin{array}{lll}1.550437 & 0.465454 & 0.355049\end{array}$
$1.532081 \quad 0.3796 \quad 0.363025$
$1.494743 \quad 0.417652 \quad 0.358942$
$1.598847 \quad 0.20454 \quad 0.302982$
$1.683574 \quad 0.18974 \quad 0.364874$
$1.804436 \quad 0.157961 \quad 0.368224$
$\begin{array}{lll}1.711634 & 0.394 & 0.251605\end{array}$
$1.756326 \quad 0.242941 \quad 0.215874$
$1.797467 \quad 0.789333 \quad 0.641127$
$\begin{array}{lll}1.778927 & 0.810115 & 0.444434\end{array}$
$\begin{array}{llr}1.827402 & 0.689866 & 0.51358\end{array}$
$\begin{array}{rrr}1.651898 & 0.668288 & 0.458456 \\ 1.514079 & 0.64749 & 0.516194\end{array}$
$\begin{array}{lll}1.661016 & 0.709246 & 0.584007\end{array}$
$\begin{array}{lll}1.6241 & 0.77675 & 0.646675\end{array}$
$\begin{array}{lll}1.572165 & 0.753236 & 0.577457 \\ 1.548492 & 0.730687 & 0.626362\end{array}$
$1.712134 \quad 0.544794 \quad 0.677793$
$\begin{array}{lll}1.798037 & 0.512656 & 0.610341\end{array}$
$\begin{array}{rrr}1.996371 & -2.293769 & -1.185353 \\ 1.14144 & -0.283468 & -0.833568\end{array}$
$1.037201 \quad-0.219385 \quad-0.741169$
$1.154765-0.007324-0.892606$
$1.086923-0.035356 \quad-0.952209$
$1.031575-0.123828 \quad-1.05424$
$1.311028 \quad-0.342763 \quad-1.06318$
$1.670686 \quad 0.301455 \quad-1.132607$
$1.158689 \quad 0.484584 \quad-1.018472$
$1.797092-0.67985 \quad-0.451534$
$\begin{array}{lll}1.07483 & -0.321768 & -0.580323 \\ 1.072017 & -0.468353 & -0.545572\end{array}$
$1.072017-0.468353 \quad-0.543572$
$1.345975-0.577883-0.551034$
$1.033519-0.587046 \quad-0.721315$
$1.138537 \quad-0.63178 \quad-0.890334$
$1.001356-0.43739 \quad-0.914134$
$1.184104-0.836755-0.805896$
$1.096922-0.892641 \quad-0.563517$
$1.033389-0.973829 \quad-0.694433$
$1.121341 \quad-0.376784 \quad-1.473617$
$1.013053 \quad-0.203636 \quad-1.384204$
$1.113291-0.931413-1.394805$

