# Supplemental Data

# The Set1 Methyltransferase

# Opposes Ipl1 Aurora Kinase Functions

# in Chromosome Segregation

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## Supplemental Experimental Procedures

### Yeast Strain Construction

The *SET1, SWD1, SDC1, SPP1, PAF1* and *BRE2* genes were replaced by the *KanMX* cassette by transforming yeast to G418-resistance with a PCR product of *set1::KanMX, swd1::KanMX, sdc1::KanMX, spp1::KanMX, paf1::KanMX or bre2::KanMX* (Guldener et al., 1996) (primer sequences provided upon request). All *set1::KanMX* mutants were confirmed by Southern blotting using a 5' probe specific for sequences upstream of *SET1*. *swd1::KanMX, sdc1::KanMX, spp1::KanMX, paf1::KanMX,* and *bre2::KanMX* mutants were verified by PCR using primers within *KanMX* and flanking the disrupted *SWD1, SDC1, SPP1*, *PAF1*or *BRE2* gene. G418 (Mediatech) was used at 200mg/liter.

The C-terminally tagged HA-Dam1 allele in strain ZK5 was created using a PCR based epitope tagging method (Schneider et al., 1995). A PCR product produced by oligonucleotides Ke111 (GCAAAGAAAACTGAAAAAAAAATAAATACAAGGCCCCCCTTCAGAA GGGAACAAAAGCTGG) and Ke126 (ATATATTTGTGAGGAGGATAATTCTTTG GTTGGGTTGGGCGTAGCTGTAGGGCGAATTGGG) using plasmid pMPY-3XHA as the template was transformed into a diploid strain generated by mating strains KT1112 and KT1113. The transformants were selected on SC-URA3 plates. A URA3 specific oligonucleotide Ke58 (GATGAGTAGCAGCACGTTCC) and a Dam1 specific oligonucleotide Ke114 (CCCACAGAAAGCAGACATTC) were used to genotype first step gene replacement colonies by PCR. Positive colonies were then plated on 5-fluoroorotic acid (5-FOA) containing media and incubated at 30 $^{o}$C. PCR was also employed to genotype the second step gene replacement colonies growing on 5-FOA plates using a HA-tag specific oligonucleotide Ke137 (GTCCGGGACGTCATAGGG) and a Dam1 specific oligonucleotide Ke100 (GACAACAGAGACAAGAGG). Second step positive colonies were sporulated, and following tetrad dissection, spores containing the HA-tagged DAM1 allele were confirmed by immunoblotting whole cell extracts using anti-HA antiserum (Roche).

Strains ZK1, ZK2, ZK3, and ZK4 were derived from strains DBY4962, DBY5301, DBY4962, and DBY 4946 (Chan et al., 1993) by inducing loss of a functional URA3 marker gene by selection on 5-FOA containing media. Additional strains bearing single or combined mutations in *ipl1-2*, *glc7-127,* and *set1Δ* were then derived from strains ZK1, ZK2, ZK3, and ZK4 as shown in Supplemental Table 1.

Strain ZK2 was mated with YPH278, which contains the *SUP11* mini-chromosome used in the chromosome segregation assays. The resulting diploid was sporulated and tetrads were dissected to identify URA$^{+}$, white colonies (named strain IS2) that were temperature sensitive lethal at 37 °C, indicating that they carried the *ipl1-2* allele (confirmed by PCR sequencing). Colonies (named strain WT1) viable at 37 °C were also picked as *IPL1* controls. *SET1* was then deleted in these strains to generate WT1 *set1Δ,* or IS2 *set1Δ* as described above.

The *ipl1-2 H3 K4R* mutant strain was isolated using a random spore approach. CC1077-6C (*ipl1-2*) was mated with JDY1 (H3 K4R) (Briggs et al., 2001) and the resulting diploids were sporulated. Spores were treated with zymolyase and then plated on SC-ura leu trp plates growing at a density of about 300 colonies per plate. After 2-3

days of growth at 25$^{o}$C, Ura$^{+}$ Trp $^{+}$ Leu $^{+}$ colonies were replica plated and tested for viability at 37$^{o}$C for 2 days. The presence of the H3 K4R and *ipl1-2* mutations cells were confirmed by DNA sequencing.

The *ipl1-2 H2B K123R* mutant strain was also isolated using a random spore approach. ZK4 (*ipl1-2*) was mated with either Y131(WT) or Y133 (*H2B123R*), and resulting diploids were sporulated. The zymolyase disrupted spores were plated on SC-Ura, Leu plates as above. After 2 days of growth at 25 $^{0}$C, Ura$^{+}$ Leu$^{+}$ colonies were replica plated and tested for viability at 37 $^{0}$ C for 2 days. Temperature sensitive Ura$^{+}$ Leu$^{+}$ colonies were further tested by plating on 5-FOA media to ensure that the histone plasmid was required for growth, indicating both chromosomal *HTA1-HTB1* and *HTA2-HTB2* alleles were disrupted.

Mutated *DAM1* alleles were introduced into yeast using a two-step gene replacement protocol. The presence of the mutant *dam1* allele in viable colonies was confirmed by DNA sequencing. For those mutant alleles that could not be recovered after the second step gene replacement, first step replacement-positive cells were mated with either ZK4 (*ipl1-2*) or ZK3 (WT). Diploid cells were then grown on 5-FOA plates screened by PCR and DNA sequencing. Heterozygotes were then sporulated and mutant alleles were isolated after tetrad dissection. A similar strategy was used to introduce the *glc7-127* mutation into strains bearing the histone H3 K4R mutation (Zhang et al., 1998), and to create strains bearing the *set1 G951S* mutation. The presence of the *set1 G951S* allele was confirmed by both DNA sequencing and by loss of H3 K4 methylation using an anti H3 di-methyl K4 antibody (Abcam) in immunoblots. To combine *ipl1-2* with the *set1 G951S* mutations, ZK1 (*set1 G951S*) was mated with ZK4 (*ipl1-2*). The resulting diploid cells were then sporulated. WT (ZK9), *set1 G951S* (ZK7), *ipl1-2* (ZK10), *ipl1-2 set1 G951S* (ZK8) spores isolated from one tetrad were used for the plate spot assay shown in Fig 1B. Multiple independent isolates yielded the same results.

The haploid strain carrying both HA-tagged Dam1 and myc-tagged Set1 was generated by mating strain ZK5 with ZK6 strain (Corda et al., 1999). The resulting diploid strain was sporulated and cells containing both tagged proteins were isolated after tetrad dissection.

### Reverse transcriptase PCR (RT-PCR)

Isolation of total RNA from yeast cells was performed as previously described (Cold Spring Harbor Manual in Yeast Genetics). RNA was digested with RNaseA-free DNaseI (Ambion) for 1 hour at 37$^{o}$C. RT-PCR was performed using RT-PCR kit (Perkin-Elmer) according to the manufacturer's protocol.

### Immunoblots

Whole cell extracts were prepared for analysis of histone H3 S10 phosphorylation by immunoblot as described previously (Hsu et al., 2000). Immunoblot analysis was performed using standard SDS-PAGE, and proteins were transferred to Immun-Blot$^{TM}$ PVDF membrane (Bio-RAD). Membranes were blocked for 1 h with Tris-buffered saline/0.05% Tween 20 containing 5% nonfat milk, followed by overnight incubation with antibodies. Rabbit anti-Dam1p antibody (Cheeseman et al., 2001) was used at a dilution of 1:1000, mouse monoclonal anti-myc antibody was used at a dilution of 1:1,000 (Cell Signaling), rabbit anti-histone H3 antibody (Abcam) was used at a dilution of 1:1000, and rabbit anti-phosphorylated Histone H3 S10 antibody was used at a dilution of 1:500 (Cell Signaling). Anti-rabbit or anti-mouse horseradish peroxidase-conjugated secondary antibodies (Amersham Bioscience) were used at a dilution of 1:10,000.

Whole cell extracts were prepared for analysis of Dam1 protein levels and phosphorylation levels as described previously (Cheeseman et al., 2001).

### Mass Spectrometric Analyses of HA-Dam1

HA-Dam1 immunopurified as above was excised from a silver stained SDS polyacrylamide gel, destained, and subjected to cleavage with modified porcine trypsin (sequencing grade, Roche Applied Science). For some experiments, the peptides were further subjected to case elimination and Michael addition of aminoethanethiol to phosphorylated serines prior to analysis (H. Nika, D.H., and R.K., manuscript in preparation). Resulting peptides were analyzed by Nano-LC-MS/MS with online desalting on a system consisting of a Famos® autosampler, Ultimate® Nano-LC module and a Switchos® pre-column switching device (LC-Packings/Dionex Corp., Sunnyvale CA).

Electrospray ion trap mass spectrometry was performed on a linear ion-trap mass spectrometer (LTQ, Thermo-Finnigan, San Jose, CA). Proteins were identified by database searching of the fragment spectra against the NCBI non-redundant protein database using Mascot (Matrix Science, London, UK), and Sequest (ThemoFinnigan, San Jose CA). Modification analysis was further performed using combinations of error-tolerant searching using both search tools as well as manual inspection of spectra.

## Supplemental References


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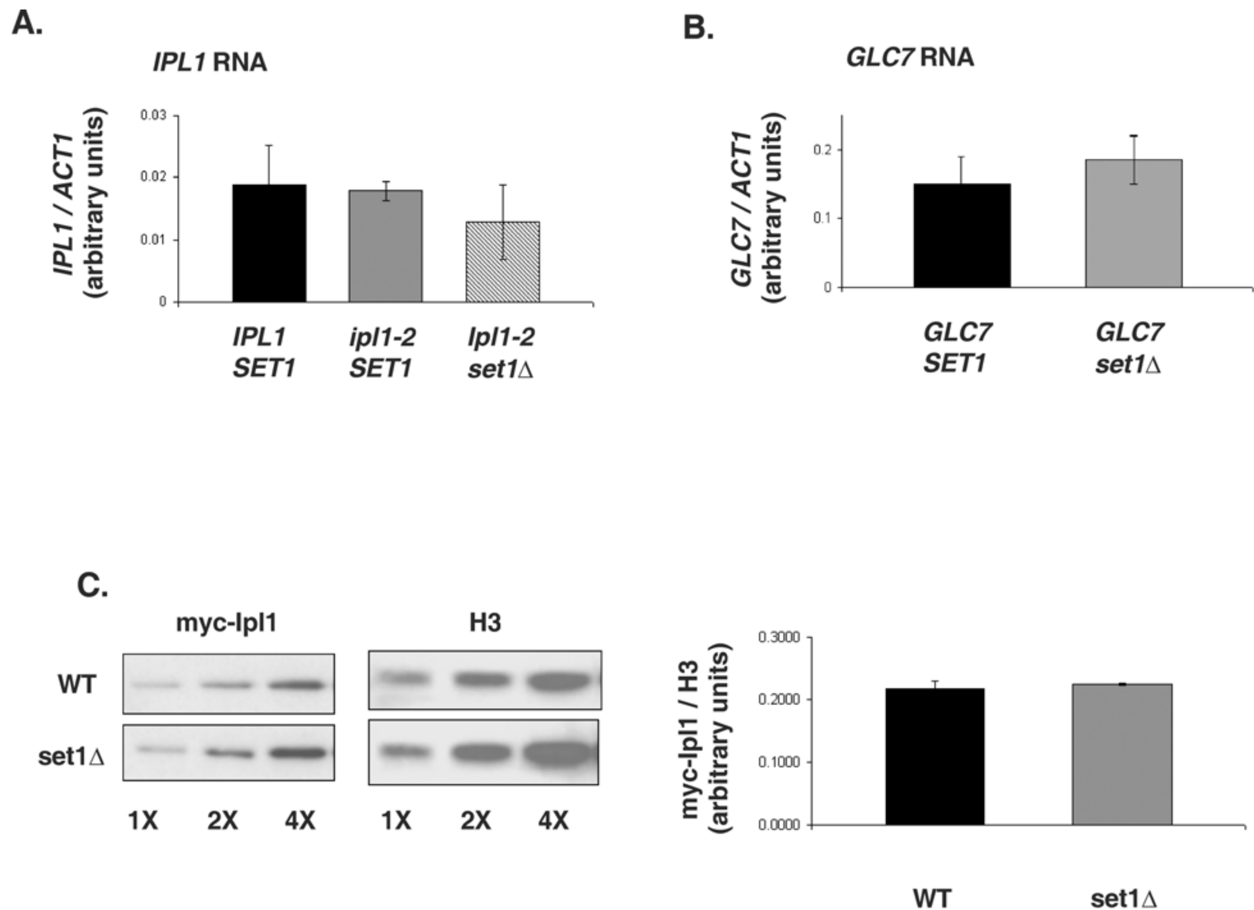
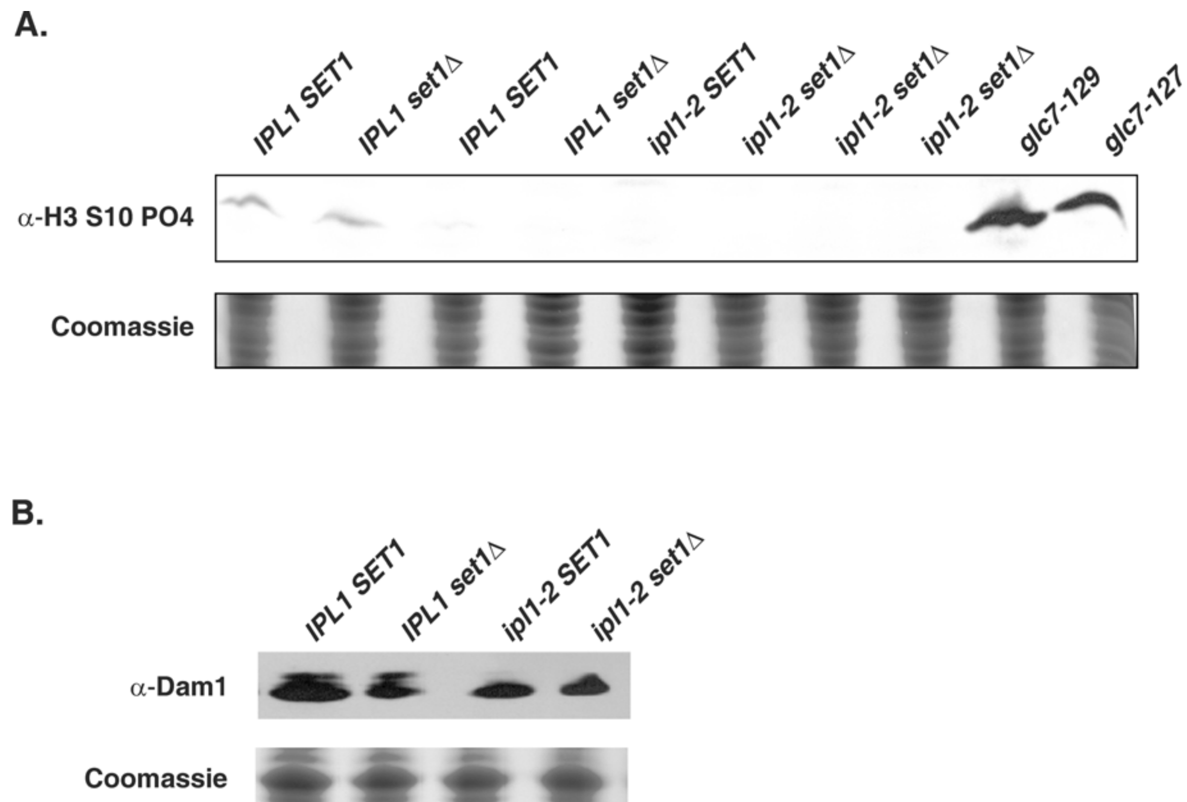
Figure S1. Deletion of *SET1* Does Not Influence the Expression of *IPL1* or *GLC7*



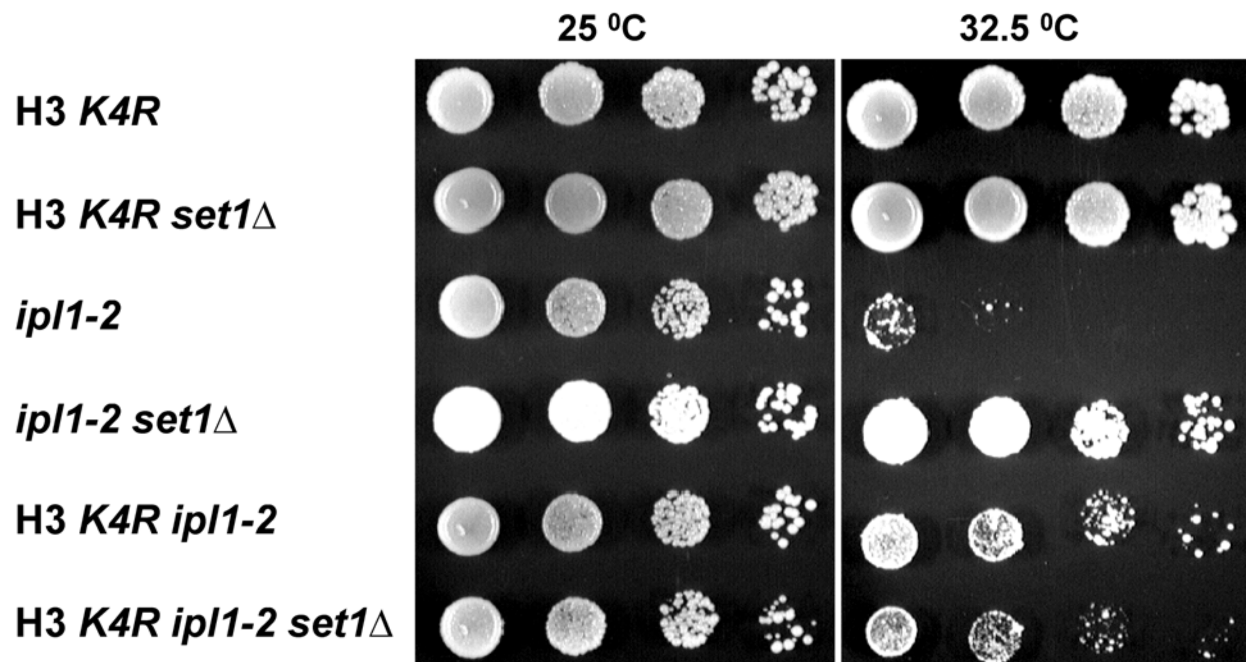

(A and B) *IPL1* or *GLC7* RNA levels were monitored by semi-quantitative RT-PCR. *ACT1* RNA levels were also measured from each of the indicated strains and were used for normalization.
(C) Whole cell extracts from wild type and *set1Δ* strains were serially diluted 2-fold and immunoblotted using anti-myc serum to detect myc-Ipl1 expressed from the native *IPL1* locus (Tanaka et al., 2002). Histone H3 levels were used as an internal protein loading control.

Figure S2. Bulk Phosphorylation of Ipl1/Glc7 Substrates Is Unchanged by Loss of *SET1*



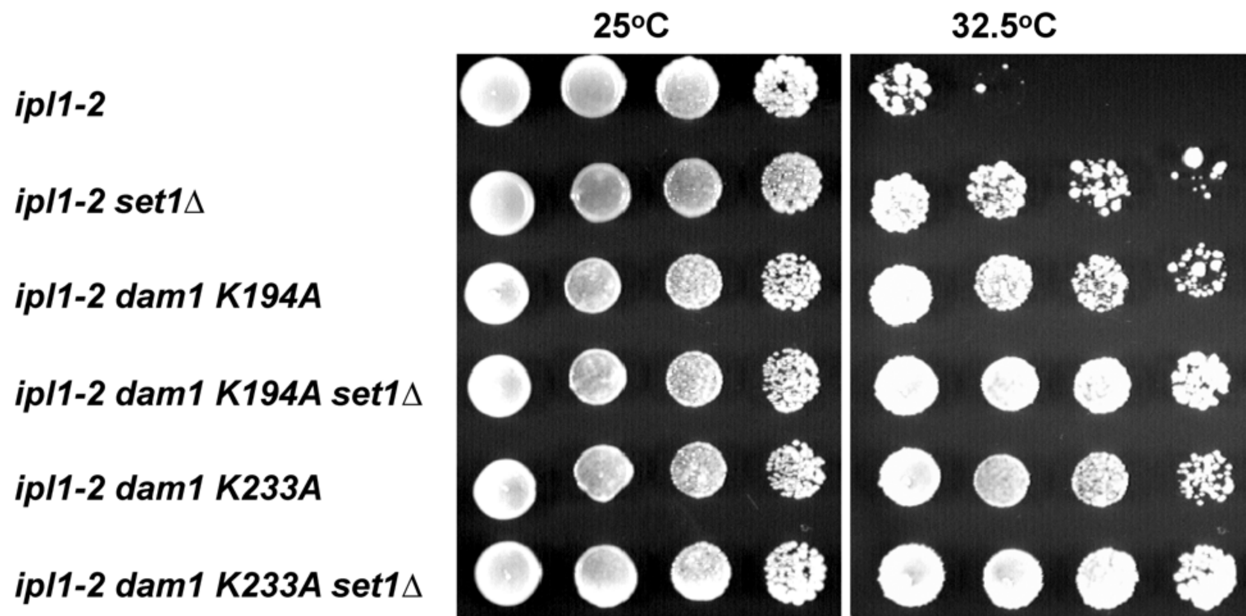

(A) Whole cell extracts from the indicated strains were probed by immunoblot using an anti-phos-S10 H3 antiserum. Histone H3 from both *glc7-127* and *glc7-129* mutants shows increased levels of phosphorylation relative to an isogenic wild type strain. However, no increase in phosphorylation was observed in H3 isolated from *set1Δ* strains relative to wild type strains. In addition, no difference in H3 phosphorylation was observed between *ipl1-2* strains and *ipl1-2 set1Δ* strains.
(B) Whole cell extracts isolated from the indicated strains were probed by immunoblot using an anti-Dam1 antiserum, which detects a cluster of bands corresponding to multiple Dam1 phospho-isoforms (Cheeseman et al., 2002). Although decreased Dam1 phosphorylation is observed in *ipl1-2* cells, no bulk change in Dam1 phosphorylation is observed in *set1Δ* cells, although alterations might occur at specific sites of phosphorylation.

Figure S3. Analysis of *ipl1 set1* H3K4R Triple Mutants




Yeast strains with the indicated genotypes were serially diluted 10-fold, spotted onto YPD medium, and grown at indicated temperatures for 2-3 days.

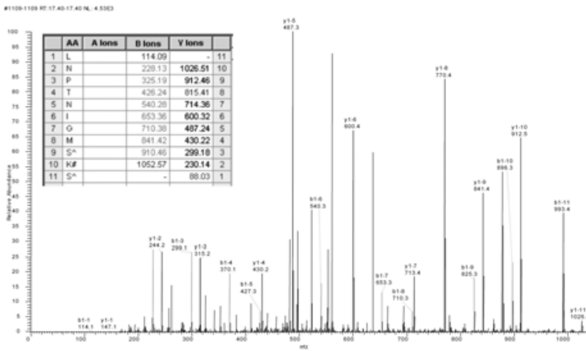
Figure S4. Analysis of *ipl1 set1 dam1* Triple Mutants



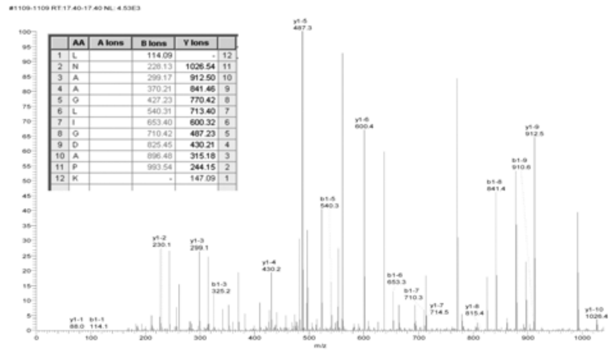

Yeast strains with the indicated genotypes were serially diluted 10-fold, spotted onto YPD medium, and grown at indicated temperatures for 2-3 days.

Figure S5. Mass Spectrometric Analysis of HA-Dam1

Peptide from Dam1




Peptide from LDH




| | MH+ | XCorr | d-Cn | Sp |
|---|---|---|---|---|
| Dam1 | 1139.61 | 2.37 | 0.371 | 1198 |
| LDH | 1139.64 | 3.96 | 0.59 | 2416 |

A representative spectrum is shown that was generated from an SDS gel band corresponding to immunopurified HA-Dam1. The gel band was digested with trypsin followed by Glu-C, absorbed to a C18 ZipTip (Millipore) and derivatized by concurrent base-elimination/Michael addition with aminoethanethiol (described by Nika, Hawke and Kobayashi, 52$^{nd}$ Conference on Mass Spectrometry and Allied Topics, Nashville TN, May 23-27, 2004). A search of the LC-MS/MS experiment with SEQUEST (ThermoFinnigan) with modifications against the Dam1 sequence was used to locate spectra matching to modified peptides. A spectrum was found that matched the Dam1 sequence LNPTNIGMSKS (2 dehydro-Serines and methyl-Lysine) with reasonably high scores and contiguous fragment ions (left panel). A broader search of the data revealed that the same fragment spectrum also matches a possible tryptic peptide from lactate dehydrogenase (LDH; a contaminating protein in the preparation) with even better SEQUEST scores (right hand panel). Based on this evidence we cannot rule out the possibility that both peptides are present in the digest.

Table S1. Yeast Strains Used in This Study

| Name | Genotype |
|---|---|
| CCY 1076-28B | *MATα, lys2, his3, ura3, leu2, trp-1, ura3::URA3-tetO112, leu2:: LEU-tetR-GFP* |
| CCY 1076-28B *Δset1* | *MATα, lys2, his3, ura3, leu2 ,trp-1, ura3::URA3-tetO112, leu2:: LEU-tetR-GFP Δset1::KanMX* |
| CCY 1076-28B *Δpaf1* | *MATα, lys2, his3, ura3, leu2 ,trp-1, ura3::URA3-tetO112, leu2:: LEU-tetR-GFP Δpaf1::KanMX* |
| CCY 1076-28B *Δbre2* | *MATα, lys2, his3, ura3, leu2 ,trp-1, ura3::URA3-tetO112, leu2:: LEU-tetR-GFP Δbre2::KanMX* |
| CCY 1076-28B *Δswd1* | *MATα, lys2, his3, ura3, leu2 ,trp-1, ura3::URA3-tetO112, leu2:: LEU-tetR-GFP Δswd1::KanMX* |
| CCY 1076-28B *Δsdc1* | *MATα, lys2, his3, ura3, leu2 ,trp-1, ura3::URA3-tetO112, leu2:: LEU-tetR-GFP Δsdc1::KanMX* |
| CCY 1076-28B *Δspp1* | *MATα, lys2, his3, ura3, leu2 ,trp-1, ura3::URA3-tetO112, leu2:: LEU-tetR-GFP Δspp1::KanMX* |
| CC 1077-6C | *MATα lys2, his3, ura3, leu2 ,ipl1-2, ura3::URA3-tetO112, leu2:: LEU-tetR-GFP* |
| CC 1077-6C *Δset1* | *MATα, lys2, his3, ura3, leu2, ipl1-2, ura3::URA3-tetO112,leu2:: LEU-tetR-GFP,Δset1:;KanMX* |
| CC 1077-6C *Δpaf1* | *MATα, lys2, his3, ura3, leu2, ipl1-2, ura3::URA3-tetO112,leu2:: LEU-tetR-GFP,Δpaf1:;KanMX* |
| CC 1077-6C *Δbre2* | *MATα, lys2, his3, ura3, leu2, ipl1-2, ura3::URA3-tetO112,leu2:: LEU-tetR-GFP,Δbre2:;KanMX* |
| CC 1077-6C *Δswd1* | *MATα, lys2, his3, ura3, leu2 ,trp-1, ura3::URA3-tetO112, leu2:: LEU-tetR-GFP Δswd1::KanMX* |
| CC 1077-6C *Δsdc1* | *MATα, lys2, his3, ura3, leu2 ,trp-1, ura3::URA3-tetO112, leu2:: LEU-tetR-GFP Δsdc1::KanMX* |
| CC 1077-6C *Δspp1* | *MATα, lys2, his3, ura3, leu2 ,trp-1, ura3::URA3-tetO112, leu2:: LEU-tetR-GFP Δspp1::KanMX* |
| KT1113 | *MATα ,leu2, ura3-52,his3* |
| KT1113 *Δset1* | *MATα, leu2, ura3-52 ,his3, Δset1::KanMX* |
| KT1113 *Δset1 Myc-GLC7* | *MATα, leu2, ura3-52 ,his3, Δset1::KanMX,YCp50-HA-GLC7* |
| KT1640 | *MATα, leu2 ,ura3-52 ,his3, glc7-127* |
| KT1640 Δset1 *Myc-GLC7* | *MATα, leu2, ura3-52, his3, glc7-127, Δset::KanMX,Ycp50-HA-GLC7* |
| KT1112 | *MATa, leu2, ura3-52 ,his3* |
| KT1112 *Δset1* | *MATa, leu2, ura3-52, his3, Δset1::KanMX* |
| T2058 | *MATa, ipl1::IPL1-myc12::URA3, bar1-1 ndc10::NDC10-HA3::TRP1* |
| T2058 *Δset1* | *MATa, ipl1::IPL1-myc12::URA3, bar1-1 ndc10::NDC10-HA3::TRP1, Δset1:KanMX:* |
| ZK1 | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102* |
| ZK7 | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102 Set1 G951S* |
| ZK8 | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102 Set1 G951S ipl1-2* |
| ZK9 | *MATα , his3-d200, ura3-52, lys2-801* |
| ZK10 | *MATα , his3-d200, ura3-52, lys2-801,ipl1-2* |
| *WT1* | *MATα, ade2, his3Δ200, ura3-52, lys2-d101::HIS3::lys2-d102, CFIII (CEN3.L.YPH278) URA3 SUP11* |
| *WT1 Δset1* | *MATα, ade2, his3Δ200, ura3-52, lys2-d101::HIS3::lys2-d102, CFIII (CEN3.L.YPH278) URA3 SUP11 Δset1::KanMX* |
| *IS2* | *MATα, ade2, his3Δ200, ura3-52, lys2-d101::HIS3::lys2-d102,ipl1-2, CFIII (CEN3.L.YPH278) URA3* |

| | |
|---|---|
| | *SUP11* |
| *IS2 Δset1* | *MATα, ade2, his3Δ200, ura3-52, lys2-d101::HIS3::lys2-d102,ipl1-2, CFIII (CEN3.L.YPH278) URA3 SUP11, Δset1::KanMX* |
| ZK1 *dam1 K194A* | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102,dam1 K194A* |
| ZK1 *dam1 S232A* | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102, dam1 S232A* |
| ZK1 *dam1 S234A* | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102,dam1 K234A* |
| ZK1 *dam1 S235A Δset1* | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102,dam1 S235A , Δset1:KanMX* |
| ZK1 *dam1 S235A* | *MATa/α, ADE2/ade2, his3-d200/,his3-d200, ura3-52/ura3-52, lys2-801/ lys2-d101::HIS3::lys2-d102,dam1 S235A/DAM1* |
| ZK1 *dam1 K233A* | *MATa/α, ADE2/ade2, his3-d200/,his3-d200, ura3-52/ura3-52, lys2-801/ lys2-d101::HIS3::lys2- d102,dam1 K233A/DAM1* |
| ZK1 *dam1 K233A S257A* | *MATa/α, ADE2/ade2, his3-d200/,his3-d200, ura3-52/ura3-52, lys2-801/ lys2-d101::HIS3::lys2- d102,dam1 K233A S257A /DAM1* |
| ZK1 *dam1 K233A S257D* | *MATa/α, ADE2/ade2, his3-d200/,his3-d200, ura3-52/ura3-52, lys2-801/ lys2-d101::HIS3::lys2- d102,dam1 K233A S257D/DAM1* |
| ZK1 *dam1 K233A S265A* | *MATa/α, ADE2/ade2, his3-d200/,his3-d200, ura3-52/ura3-52, lys2-801/ lys2-d101::HIS3::lys2- d102,dam1 K233A S265A/DAM1* |
| ZK1 *dam1 K233A S265D* | *MATa/α, ADE2/ade2, his3-d200/,his3-d200, ura3-52/ura3-52, lys2-801/ lys2-d101::HIS3::lys2- d102,dam1 K233A S265D/DAM1* |
| ZK1 *dam1 S235A S257A* | *MATa/α, ADE2/ade2, his3-d200/,his3-d200, ura3-52/ura3-52, lys2-801/ lys2-d101::HIS3::lys2-d102,dam1 S235A S257A/DAM1* |
| ZK1 *dam1 S235A S257D* | *MATa/α, ADE2/ade2, his3-d200/,his3-d200, ura3-52/ura3-52, lys2-801/ lys2-d101::HIS3::lys2-d102,dam1 S235A S257D/DAM1* |
| ZK1*dam1 S235A S265A* | *MATa/α, ADE2/ade2, his3-d200/,his3-d200, ura3-52/ura3-52, lys2-801/ lys2-d101::HIS3::lys2-d102,dam1 S235A S265A/DAM1* |
| ZK1 *dam1 S232D* | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102 ,dam1 S232D* |
| ZK1 *dam1 S234D* | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102, dam1 S234D* |
| ZK1 *dam1 S235D* | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102, dam1 S235D* |
| ZK1 *dam1 K233A S232A* | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102,dam1 K233A S232A* |
| ZK1 *dam1 K233AS234A* | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102,dam1 K233AS234A* |
| ZK1 *dam1 K233AS235A* | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102,dam1 K233A S235A* |
| ZK1 *dam1 K233A S232D* | *MATa/α, ADE2/ade2, his3-d200/,his3-d200, ura3-52/ura3-52, lys2-801/ lys2-d101::HIS3::lys2-d102,dam1 K233A S232D /DAM1* |
| ZK1 *dam1 K233A S234D* | *MATa/α, ADE2/ade2, his3-d200/,his3-d200, ura3-52/ura3-52, lys2-801/ lys2-d101::HIS3::lys2-d102,dam1 K233A S234D /DAM1* |
| ZK1 *dam1 K233AS235D* | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102, dam1 K233A S235D* |
| ZK2 | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102 ipl1-2* |
| ZK2 *Δset1* | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102 ipl1-2, Δset1::KanMX* |
| ZK2 *dam1 K194A* | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102 ipl1-2 ,dam1 K194A* |
| ZK2 *dam1 K194A Δset1* | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102 ipl1-2 ,dam1 K194A, Δset1::KanMX* |
| ZK2 *dam1 K233A* | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102 ipl1-2 ,dam1 K233A* |

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| ZK2 *dam1 K233A Δset1* | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102 ipl1-2 ,dam1 K233A, , Δset1::KanMX* |
| ZK3 | *MATα,lys2-801,his3-d200, ura3-52* |
| ZK4 | *MATα, lys2-801, his3-d200, ura3-52, ipl1-2* |
| ZK1 *glc7-127* | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102, glc7-127* |
| ZK1 *set1Δ* | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102 , Δset1:KanMX:* |
| ZK2 *glc7-127* | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102 glc7-127, ipl1-2* |
| ZK2 *glc7-127 set1Δ* | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102 glc7-127, ipl1-2 , Δset1:KanMX:* |
| ZK1 *K233R* | *MATa ,ade2 ,his3-d200, ura3-52, lys2-d101::HIS3::lys2-d102,dam1 K233R/DAM1* |
| YPH278 | *MATα, ura3-52, lus2-801, ade2-101, his3Δ200, leu2Δ1, CFIII(CEN3.L.YPH278) URA3 SUP11* |
| ZK5 | *MATα ,leu2, ura3-52,his3, dam1::DAM1-HA3* |
| ZK5 *set1Δ* | *MATα ,leu2, ura3-52,his3, dam1::DAM1-HA3, Δset1:KanMX:* |
| ZK5 *K233R* | *MATα ,leu2, ura3-52,his3, dam1::dam1 K233R-HA3,* |
| *Y131* | *MAT a, ade2-1 his3-11 15ura3-1 leu2-3 112trp1, hta1-htb1Δ::LEU2 hta2-hta2Δ, pRS426-HTA1-HTB1* |
| *Y133* | *MAT a, ade2-1 his3-11 15ura3-1 leu2-3 112trp1, hta1-htb1Δ::LEU2 hta2-hta2Δ, pRS426-HTA1-htb1-K123R* |
| *LPY917* | *MATa, ade2-101,trp1Δ1, leu2Δ1,his3Δ-200,lys2-801 TELadh4:;URA3* |
| ZK6 | *MATa, ade2-101,trp1Δ1, leu2Δ1,his3Δ-200,lys2-801 TELadh4:;URA3,set1:;SET1-Myc9::LEU* |
| KT1697 | *MATα ura3-52 his3 trp1 sds22::TRP1 leu2::sds22-6::LEU2* |
| KT1699 | *MATα ura3-52 his3 trp1 sds22::TRP1 leu2::SDS22::LEU2* |